Oral and Maxillofacial Surgery in Dogs and Cats

Frank J. M. Verstraete
Milinda J. Lommer
Boaz Arzi

SECOND EDITION

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Dedications

I dedicate this book to the people from whom I have learned, and thank the two universities that I have been affiliated with in my career, the University of Pretoria in South Africa and the University of California at Davis, who have given me the opportunity to develop my skills and practice veterinary dentistry and oral surgery.

"A mind grows by what it feeds upon—associates, books, teachers, and an atmosphere encouraging to scholarly pursuits." – Owen H. Wangensteen[*]

Frank J. M. Verstraete

For my mother, who has always encouraged me to pursue my highest aspirations. To the numerous individuals who have guided me throughout my education and my life: I continue to strive to be an excellent clinician and an exceptional person, and I am grateful to each of you for your patience and support.

Milinda J. Lommer

My contribution to this book is dedicated to the extraordinary mentors in my past and present that have inspired me and, most importantly, believed in me. You have shaped my professional life and my pursuit of excellence and innovations. To my parents, Yoheved and Israel, my wife, Natasha, and my children, Jonatan and Eden, I am eternally grateful for your love and support.

Boaz Arzi

[*]Wangensteen OH. Credo of a surgeon following the academic line. *J Am Med Assoc* 1961;177:558-563.

Foreword

M. Anthony Pogrel, DDS, MD, FRCS, FACS, Diplomate ABOMS, Professor, Department of Oral and Maxillofacial Surgery, School of Dentistry, University of California, San Francisco, California, USA

It gives me a great pleasure to write this foreword for the second edition of Drs. Verstraete, Lommer, and Arzi's important textbook. Human surgeons and veterinary surgeons live in a symbiotic relationship with each other. In many cases, surgical techniques utilized on humans are first evaluated in an animal model before being transferred to humans. In other cases, surgical techniques evolve in humans before the basic biology of the technique is later evaluated in animal models. On the reverse side of the coin, techniques that have been perfected in humans are often then transferred back to the animal world to become options for animal surgery. This has been very pertinent in the field of oral and maxillofacial surgery, and especially in domestic cats and dogs. Specifically, techniques for fracture fixation, mandibular and maxillary resection and reconstruction, particularly with the use of plates, screws, and bone grafts, are now commonplace in animal surgery, and have evolved from human techniques and utilize the same instrumentation. Restoration of form and function have become more important in domestic animal oral and maxillofacial surgery over the years due to improved prognosis in the management of disease and increased expectations on behalf of both the surgeon and the animal guardian.

Differences, however, still remain with regard to expectations following similar animal and human surgical procedures. In particular, long-term goals are often different, in that in humans we are often looking at 20, 30, and 40 year follow-up results, whereas in cats and dogs a success over a 2 to 3 year period is often all that is desired or required.

This is an important textbook and addition to the literature and, although it is mainly of value to veterinary surgeons, it will also be of value to human surgeons as we continue our close relationship. As the father of a veterinarian trained by Dr. Verstraete, I continue to wish this textbook the best of success.

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Preface

Oral and maxillofacial surgery is an exciting and rewarding field, offering practitioners numerous opportunities to improve their patients' quality of life. Because oral diseases and maxillofacial lesions are often painful and debilitating, treating these conditions can have a huge impact on an animal's well-being. Unfortunately, although oral diseases are prevalent among companion animals, and veterinarians are expected to diagnose and treat them, the anatomy, physiology, and pathology of the oral cavity are discussed only cursorily in veterinary school. This textbook aims to provide students, general practitioners, residents, and specialists with a comprehensive survey of oral and maxillofacial surgery. Each chapter includes the "why" as well as the "how," and the procedures described range from closed, single-rooted tooth extraction to major resection of maxillary and mandibular tumors. It is important to note that the text is not intended to be a "step-by-step" guide to performing these procedures, and that practitioners interested in learning a particular procedure described in the text should pursue additional training with a board-certified specialist before attempting an unfamiliar surgical procedure on a patient.

We also wish to point out that procedures that we feel are ethically questionable (performed for the purpose of pleasing the client or demonstrating the technical prowess of the operator rather than to improve the patient's quality of life) are not included in this book. We specifically refer to orthognathic surgery and dental implants. Orthognathic surgery is complicated, puts the animal at risk of serious complications, and is rarely justifiable. While we aim to restore a comfortable and functional occlusion in our patients, the goal of perfecting a malocclusion through orthognathic surgery is misguided, particularly since malocclusions causing discomfort can typically be treated by orthodontic means or by selective extractions. Regarding the replacement of missing teeth, although dogs have been used extensively in dental implant research, it is very difficult to provide ethical justification for implant surgery in client-owned animals given how well dogs and cats function with missing teeth and the high potential for complications following implant surgery.

The second edition of this textbook was compiled with the contribution of board-certified veterinary surgeons and dentists who are leaders in their fields, as well as experts in human oral pathology and maxillofacial surgery. The information contained is based on reviews of relevant and current literature and the clinical expertise of the authors. We have also added a few new chapters, such as diagnostic imaging, management of temporomandibular joint ankyloses, and regenerative approaches to mandibular reconstruction. As with the first edition, we have attempted to make this edition of the text comprehensive, authoritative, and easy to read. Every effort has been made to ensure that the nomenclature used in this textbook is correct and in agreement with *Nomina Anatomica Veterinaria*. Intentional repetition of some material is present throughout several chapters to allow for complete presentation of the material in the individual chapters and to reinforce basic surgical principles relative to the specific procedures described.

We believe that oral and maxillofacial surgery should be taken seriously by the veterinarian and should not be delegated to staff members. A well-planned and skillfully performed procedure will result in minimal postoperative discomfort, reduced healing time, and rapid return to function for the patient. As veterinarians, our primary goal is to improve our patients' well-being; this can best be achieved by continuing to educate ourselves, to improve our skills, and to share our knowledge with others.

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We are grateful to the contributing authors for sharing their knowledge and passion for the profession, for their generous commitment of time to this project, and for their patience. We would also like to thank the highly professional team at Elsevier who enabled us to bring this edition to completion.

SECTION 1:

Surgical Biology

OUTLINE

CHAPTER 1

Oral soft tissue wound healing

Vivek Shetty, Anh D. Le

Definitions

A *wound*, regardless of the cause of injury, is a disruption of normal tissue continuity and integrity. *Healing* is simply the process of restoring the integrity of the wounded tissue. If the result is tissue that is structurally and functionally the same as the original tissue, then *regeneration* has taken place. However, if tissue integrity is reestablished primarily through the formation of a fibrous, connective-tissue scar, then *repair* has occurred. The nature of the native tissue involved determines whether regeneration or repair will ensue, and the surgeon's expectations should be correspondingly realistic. Whereas a fibrous scar may be normal for dermal healing, it is suboptimal in the case of bone healing.

General considerations

Every injury initiates an orderly, but complex sequence of orchestrated events that reestablish the integrity of the damaged tissue. Despite the body's innate ability to heal, surgical intervention is often used to optimize the healing process and favorably modulate the outcome. Interventions may include adequate debridement of devitalized tissue, removal of diseased tissue or foreign materials, securing adequate hemostasis, and apposing severed tissues with mechanical means until such time the wound is capable of withstanding functional stresses.

From a surgical viewpoint, the nature of wound healing depends upon the site, type of tissue involved, and the surgeon's ability to approximate the wound margins. Healing by *first intention* usually occurs when early primary closure can be achieved by accurately reapproximating the wound margins. Such a wound heals quickly with no separation of the wound edges, and with minimal scar formation. Absent favorable conditions, wound healing is prolonged and occurs through a filling of the tissue defect with granulation and connective tissue. This process is called healing by *second intention* and is frequently encountered following avulsive injury, wound infection, or poor apposition of the wound margins. In instances of infected or contaminated traumatic wounds with severe tissue loss, the surgeon may attempt healing by *third intention*. This is a staged procedure wherein the wound is allowed to granulate and heal by second intention before a delayed primary closure is carried out by bringing together the two surfaces of granulation tissue.

Wound-healing phases

The healing process in different tissues occurs in a cascade of overlapping phases best represented by cutaneous wound healing. Beginning with the inflammatory phase precipitated by the injury, the wound eventually restores itself through sequentially occurring proliferative and remodeling phases. While the rates and patterns of healing depend on a host of local, systemic, and surgical factors, the phases of oral soft-tissue healing are typical for all other tissues as well. In general, wounds in the oral cavity seem to heal faster than wounds to the skin. Oral wounds, despite being exposed to a bacteria-laden, moist, seemingly hostile environment, heal perfectly well and reepithelialize rapidly with minimal or no scar formation.

Inflammatory phase

The wounded area attempts to restore its normal state (homeostasis) immediately following injury. Disrupted blood vessels constrict and thrombose, and the thromboplastin released by the injured cells initiates the coagulation process. The accumulating platelets help form a fibrin clot to control bleeding. Additionally, the injured tissue and platelets begin to release key mediators of wound healing, particularly platelet-derived growth factors (PDGFs) and transforming growth factor β (TGF-β). These chemoattractants recruit inflammatory cells that begin to remove damaged tissue and bacteria from the injured area. Clinical signs include localized edema, pain, redness, and increased warmth at the wound site. Neutrophils are the predominant inflammatory cells during the initial 2 to 3 days following injury but are rapidly outnumbered by macrophages derived from mobilized monocytes. As the primary source of modulating cytokines such as PDGF and vascular endothelial growth factor (VEGF), the macrophages regulate the formation of the granulation tissue that is distinctive of the proliferation phase.[1]

Proliferation phase

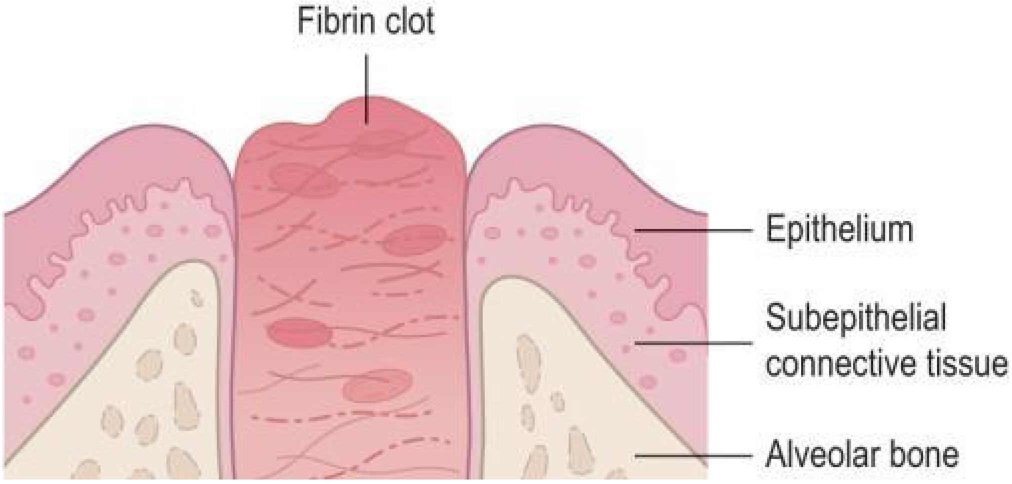
The proliferation phase is a period of intense replication of cells and is characterized by the migration and proliferation of fibroblasts and smooth muscle cells into the wound milieu. The fibroblast is the major cell responsible for the production of collagen and proteoglycans. Fibroblasts interact with their surrounding matrix via receptors known as integrins that regulate the level of collagen gene expression and collagenase induction. Collagen restores the strength and integrity of the repaired tissue, whereas the proteoglycans function as moisture storage. Concurrent with these events is the process of angiogenesis, whereby new blood vessels are formed and lymphatics are recanalized in the healing tissues. This essential process reestablishes transport of the nutrients and oxygen to the local injured site. In a synergistic way, the new capillaries supply nourishment to the developing collagen, while the collagen fibers structurally support the new capillary beds. Epithelial cells originating from hair follicles, sebaceous glands, and margins of the wound edges proliferate and resurface the wound above the basement membrane. In contrast to skin, the process of reepithelialization progresses more rapidly in the oral mucosal wound. The oral epithelial cells migrate directly onto the moist, exposed surface of the fibrin clot instead of under the dry exudate (scab) of the dermis as in dry skin.[2] The rapid reepithelialization limits further insults from the oral cavity environment such as food debris, foreign particles, and microorganisms.

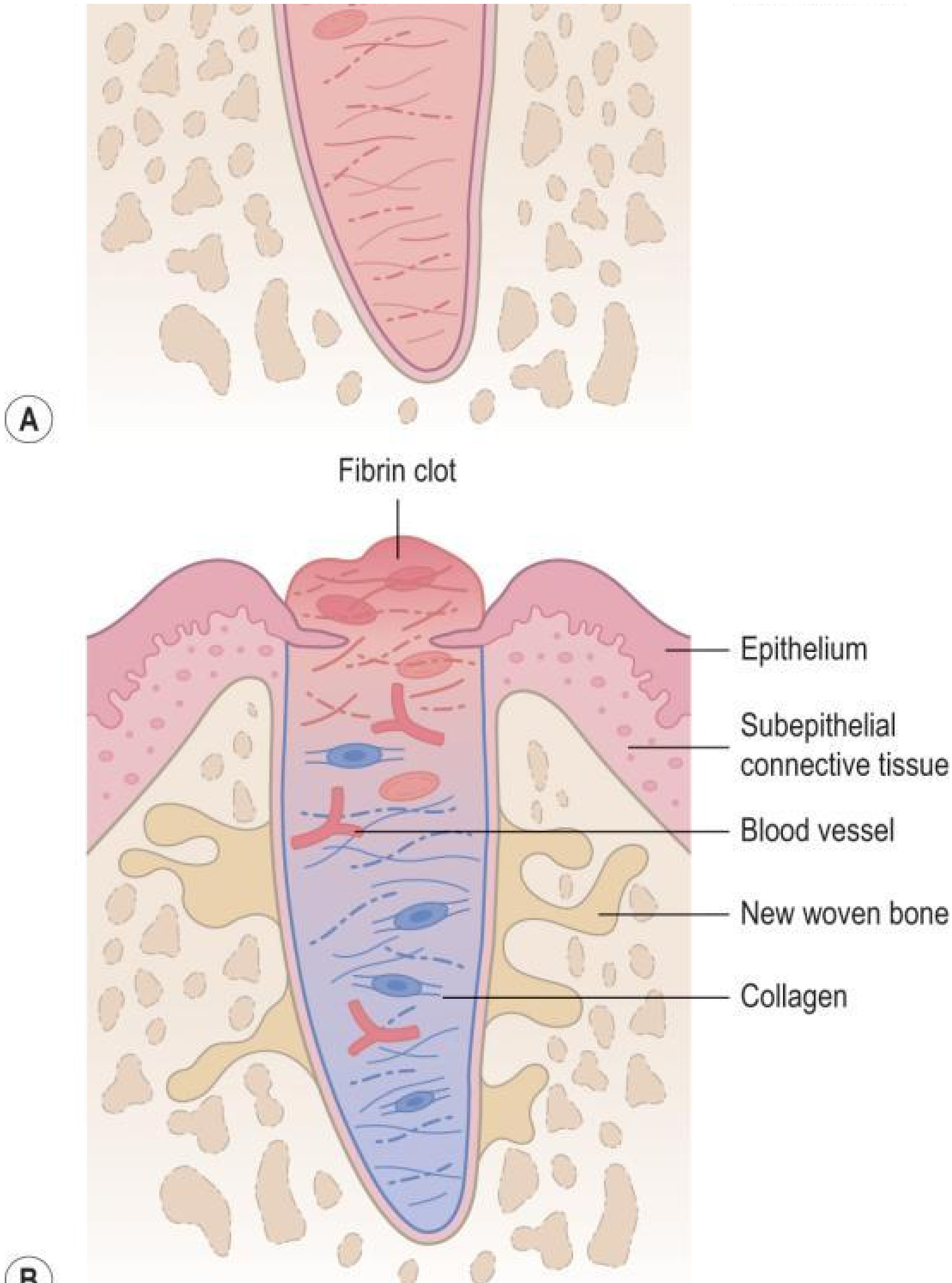
Maturation/remodeling phase

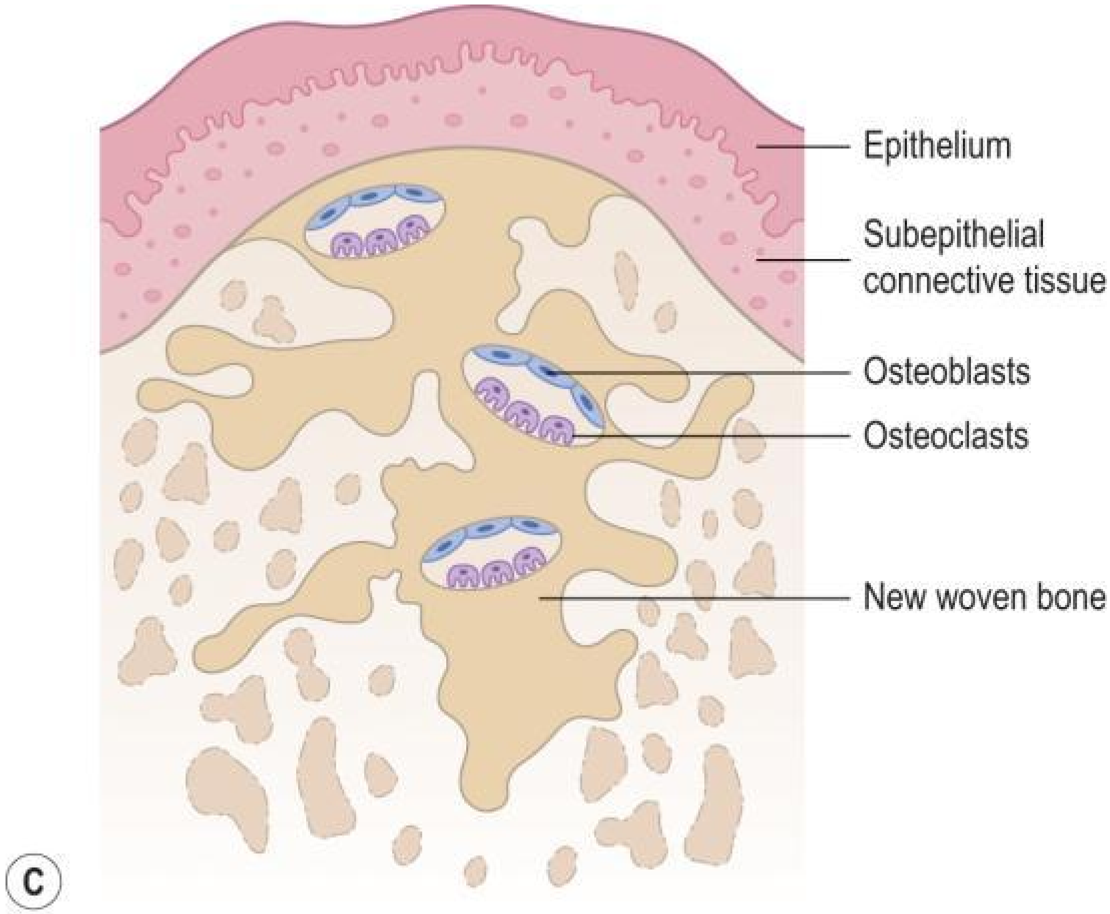
The remodeling phase is the final stage of tissue repair and is distinguished by a continual turnover of collagen molecules as precursor collagen is broken down and new collagen synthesized. The tensile strength of the wound gradually restores as the collagen fibers are realigned and increasingly cross-linked to each other. The maximal tensile strength of a healed wound is reached in 6 to 12 months following injury but never reaches the strength of unwounded tissue. Eventually, active collagen synthesis achieves equilibrium with collagenolysis. However, disruptive processes such as poor oxygen perfusion, lack of nutrients, and wound infection can shift the balance to favor collagen breakdown and wound dehiscence.

Healing of extraction wounds

The healing of a dental extraction wound is a specialized example of healing by second intention (Fig. 1.1).[3] Immediately after the removal of the tooth from the alveolus, 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, 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 (in humans) or margin (in animals) setting the stage for active crestal or marginal resorption. Angiogenesis begins in the remnants of the periodontal ligaments. In the second week, the clot continues to get organized through fibroplasia and neoangiogenesis that begin to penetrate toward the center of the clot. Trabeculae of osteoid slowly extend into the clot from the alveolus and osteoclastic resorption of the cortical margin of the alveolus is more distinct. By the third week, the extraction socket is filled with granulation tissue and poorly calcified bone forms around the periphery of the wound. 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 6 to 8 weeks following tooth extraction. As bone remodeling proceeds, the extraction site becomes less distinct and is inconspicuous after 6 to 8 months.


Fibrin clot
Epithelium
Subepithelial
connective tissue
Alveolar bone


A
Fibrin clot
Epithelium
Subepithelial connective tissue
Blood vessel
New woven bone
Collagen
B



• FIG. 1.1 Healing of an extraction wound after: (**A**) 24–48 hours; (**B**) 1 week; (**C**) 2–4 weeks.

Rarely, 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. Because of 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 alveolus, the infected alveolus remains open or partially covered with hyperplastic epithelium for extended periods.

Factors affecting healing

Infection

Wound infection is the most common cause of impaired wound healing. Though oral wounds are always colonized by bacteria, infection occurs only when the virulence or the number of the bacteria exceeds the ability of local tissue and host defenses to control them. The likelihood of wound infection increases substantially when the bacteria proliferate to levels beyond 10^5 organisms per gram of tissue.[4] Bacteria provoke various degrees of inflammation at the wounded tissue by releasing endotoxins, metalloproteinases, and breakdown products that inhibit the activities of regenerating cells and the scavenger macrophages. In addition to systemic diseases, local factors such as inadequate tissue perfusion and the presence of necrotic tissue or foreign bodies facilitate deterioration of a contaminated wound into an infected wound. The most important factor in minimizing the risk of infection is meticulous surgical technique, including thorough debridement, adequate hemostasis, and elimination of dead space. Proper postoperative care, including stringent wound hygiene and absence of reinjury, further reduces the risk of infection.

Tissue perfusion and oxygenation

Adequate tissue perfusion is critical to the healing process. To a certain degree, hypoxia stimulates the cells to produce angiogenic growth factors. However, severe tissue hypoxia combines with lactic acid produced by bacteria to lower tissue pH and contributes to tissue breakdown or necrosis.[5–7] Wounds in hypoxic tissues are more easily infected and heal poorly as leukocytic, fibroblastic, and epithelial proliferation is depressed by low oxygen concentration. Poor oxygenation also interferes with the synthesis of collagen since oxygen is required for the hydroxylation of lysine and proline.[8] Furthermore, studies have shown that collagen deposition and wound tensile strength are limited by tissue perfusion and oxygen tension.[9]

The impaired healing associated with conditions such as diabetes mellitus, radiation damage, vasculitis, venous stasis, arteriosclerosis, and chronic infection can be largely ascribed to a faulty oxygen delivery system. Ischemic tissues produced by improper surgical techniques are poorly perfused and are excessively prone to infection. Tissue edema, remnants of necrotic tissues, or a systemic perfusion defect, such as hypovolemia, all impair wound healing. It follows that the natural resistance of the wound can also be enhanced by the maintenance of an adequate body fluid volume and satisfactory arterial oxygen tension. The use of hyperbaric oxygen to maintain the wound in a state of hyperoxia is based on this rationale.

Age

In general, oral wound healing is faster in the young animal than in the elderly. The influence of age on wound healing probably results from the general reduction of tissue metabolism that may be manifested by multiple physiologic problems as the animal ages. The major processes that drive soft-tissue healing are diminished or damaged with progressive age. As a result, free oxidative radicals continue to accumulate and are deleterious 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.[10]

Diabetes mellitus

Most of the complications related to diabetes, particularly poor wound healing, can be attributed to the development of diabetic microangiopathy. Local ischemia, secondary to poor oxygen delivery at the tissue level, and small vessel occlusion play an essential role in the pathogenesis and delayed healing of diabetes. Glycosylated hemoglobin has a higher binding affinity to oxygen molecules, which further impairs oxygen delivery to ischemic tissues. In addition, granulocyte function in uncontrolled diabetes is impaired, rendering animals with diabetes more susceptible to wound infection. Poor healing has also been related to the metabolic problems related to hyperglycemia, insulin deficiency, and/or insulin resistance. The wound in the diabetic animal often demonstrates a decreased inflammatory response, fibroblast proliferation, and collagen deposition, resulting in a healing product with reduced tensile strength. A stringent regulation of blood sugar is therefore essential in the diabetic patient undergoing surgery, to optimize the wound healing potential.

Malnutrition

Nutritional deficiencies that produce hypoproteinemia hinder wound healing and impair the immune defense by limiting the availability of the amino acids critical for the synthesis of collagen and other proteins. Methionine, in particular, is a key amino acid in wound healing. It is metabolized to cysteine and plays a vital role in the inflammatory, proliferative, and remodeling phases of wound healing. As long as a state of protein catabolism exists, the wound heals very slowly. Several vitamins and trace minerals play a significant role in wound healing. Vitamin C and iron are essential cofactors for the hydroxylation of lysine and proline during collagen synthesis. Absent adequate compensatory collagen synthesis, scars may dissolve if collagenolytic activity continues unabated. Vitamin A is essential for normal immune function, epithelialization, and proteoglycan synthesis, and healing is impaired when vitamin A is deficient. The B-complex vitamins and cobalt are essential cofactors in the antibody formation, white blood cell function, and bacterial resistance. Vitamin D, thiamin, and riboflavin deficiencies also result in poor repair.

Copper is needed for lysyl amine oxidase, whereas calcium is required for the normal function of granulocyte collagenase and other collagenases at the wound site. Zinc deficiency retards both fibroplasia and reepithelialization.[11] Zinc is required for DNA replication and serves as a coenzyme for DNA-polymerase and reverse-transcriptase. However, pharmacologic overdosing of zinc levels can exert a distinctly detrimental effect on healing by inhibiting macrophage migration and interference with collagen cross-linking.

Current trends in wound care

An increased understanding of the wound-healing processes has generated heightened interest in manipulating the wound microenvironment to facilitate healing. The traditional passive ways of treating wounds are rapidly giving way to approaches that actively modulate wound healing. These approaches include treatments that selectively jump-start the wound into the healing cascade, increase oxygenation and perfusion of the local tissues, or mechanically protect the wound.

Growth factors

Growth factors and cytokines are essential regulators of the healing process and are found to be insufficiently present or dysfunctional in the non-healing wound. Over the years, a variety of topical exogenous recombinant growth factors have been investigated as agents to improve the wound-healing process. These include PDGF, angiogenesis factor, epidermal growth factor (EGF), transforming growth factor (TGF), fibroblast growth factor (bFGF), tumor necrosis factor (TNF), and interleukin-1 (IL-1). 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 recombinant growth factor—recombinant human platelet-derived growth factor-BB form or Becaplermin (Regranex gel: Smith & Nephew, Inc., Fort Worth, TX)—has been approved by the United States Food and Drug Administration for the treatment of cutaneous ulcers, specifically diabetic foot ulcers. The PDGF stimulates cellular growth and migration of granulation tissue, thereby promoting healing. However, Becaplermin is adjunctive to good wound care (GWC) practices, including good initial debridement and infection control. Results from several controlled human clinical trials show that platelet-derived growth factor-BB form gel was effective in healing lower extremity diabetic ulcers and significantly decreased their healing time when compared to the placebo group.[12,13]

More recently, recombinant human keratinocyte growth factor 2 (KGF-2: Repifermin, Human Genome Sciences, Inc., Rockville, MD) has been shown to accelerate wound healing in experimental animal models. It enhances both the formation of granulation tissue in both young and old rabbits and wound closure of the human meshed skin graft explanted on athymic nude rats.[14,15] The safety assessment of the drug showed that KGF-2 was well tolerated in human with no differences in adverse events.[16]

Topical chitosan, a derivative of chitin, is increasingly used in veterinary clinical medicine as a wound-healing accelerator for large open wounds. Chitosan appears to stimulate a rapid infiltration of inflammatory cells and granulation tissue formation along with an increased production of biological mediators and cytokines and fibroblastic proliferation.[17] Typical complications associated with the application of large doses of chitosan (above 50 mg/kg) in dogs include leukocytosis, elevated serum LDH2, LDH3 isoenzymes, and severe hemorrhagic pneumonia.

Despite the promise and excitement, most of the growth factor studies to date have been small and have different endpoints and modes of administration. As such, all data related to exogenously growth factors should be interpreted with caution, and any off-label use of these products for the management of wounds should be used in conjunction with rigorous wound care, including operative debridement to remove impediments to healing.

Hyperbaric oxygen therapy

Hyperbaric oxygen therapy (HBOT) is occasionally used in veterinary medicine to raise tissue oxygen tension to a level that facilitates healing. In HBOT, 100% oxygen is delivered to the patient at pressures between 1.5 and 2.4 atmospheres. This stimulates the growth of fibroblasts and endothelial cells, increases the killing ability of leukocytes, and is lethal for anaerobic bacteria. Multiple studies in HBOT in the treatment of human diabetic patients suggest that HBOT can be an effective adjunct in the management of diabetic wounds.[18] Animal studies suggest that HBOT could be beneficial in the treatment of osteomyelitis and soft tissue infections.[19,20] Adverse effects of HBOT are barotrauma of the ear, seizure, and pulmonary oxygen toxicity.

Skin substitutes

Immediate wound coverage is critical for the acceleration of wound healing. When the surface area is large, wounds can be covered by synthetic and natural dressings. 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 (Epicel: Genzyme Tissue Repair Corp., Cambridge, MA). The second type has only dermal components (AlloDerm Regenerative Tissue Matrix: LifeCell Corp., Woodlands, TX; Dermagraft: Advanced BioHealing, Inc., La Jolla, CA). The third type consists of a bilayer of both dermal and epidermal elements (Apligraf: Organogenesis Inc., Canton, MA; Integra: Johnson & Johnson Medical Integra Life Sciences Corp., Plainsboro, NJ). The chief effect of most skin substitutes 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.

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CHAPTER 2

Maxillofacial bone healing

Randy J. Boudrieau, Franz Härle

Structure of facial bone

The structure of facial bone is determined by its material properties and by its mechanical role. The bone marrow cavity, the cortex, and the spongiosa of the mandible and the midface are similar with respect to their material composition. The major difference is the geometric distribution of the bone. The canine mandible differs from the long bones in that it does not have a medullary canal. Instead, the cortex surrounds the cancellous bone and the mandibular canal, which contains the inferior alveolar neurovascular structures but no hematopoietic cells typical for a medullary canal.[1,2] The structure of the feline mandible closely resembles the canine mandible. The midfacial skeleton differs from the mandible in that it consists of a single, thin lamina of bone. The basic composition of bone is that of a dynamic tissue constantly undergoing resorption and remodeling.[3-5] Bone cell types consist of osteoblasts, osteocytes, and osteoclasts.

Osteoblast

Osteoblasts are derived from pluripotential precursor cells. They produce osteoid, the organic matrix of bone, which is transformed into calcified bone. A layer of 1 μm of osteoid may be produced per day, followed by a maturation phase of 10 days before calcification.

The microscopic structure of the osteoid is differentiated between woven and lamellar bone. In woven bone, the collagen fibrils are randomly orientated and have a felted texture. In lamellar bone, the collagen fibrils are arranged in parallel bundles. Woven bone mineralizes immediately after osteoid deposition. In fracture healing, woven bone is formed first to clinically unite the bone, and then is transformed into lamellar bone.

Osteocyte

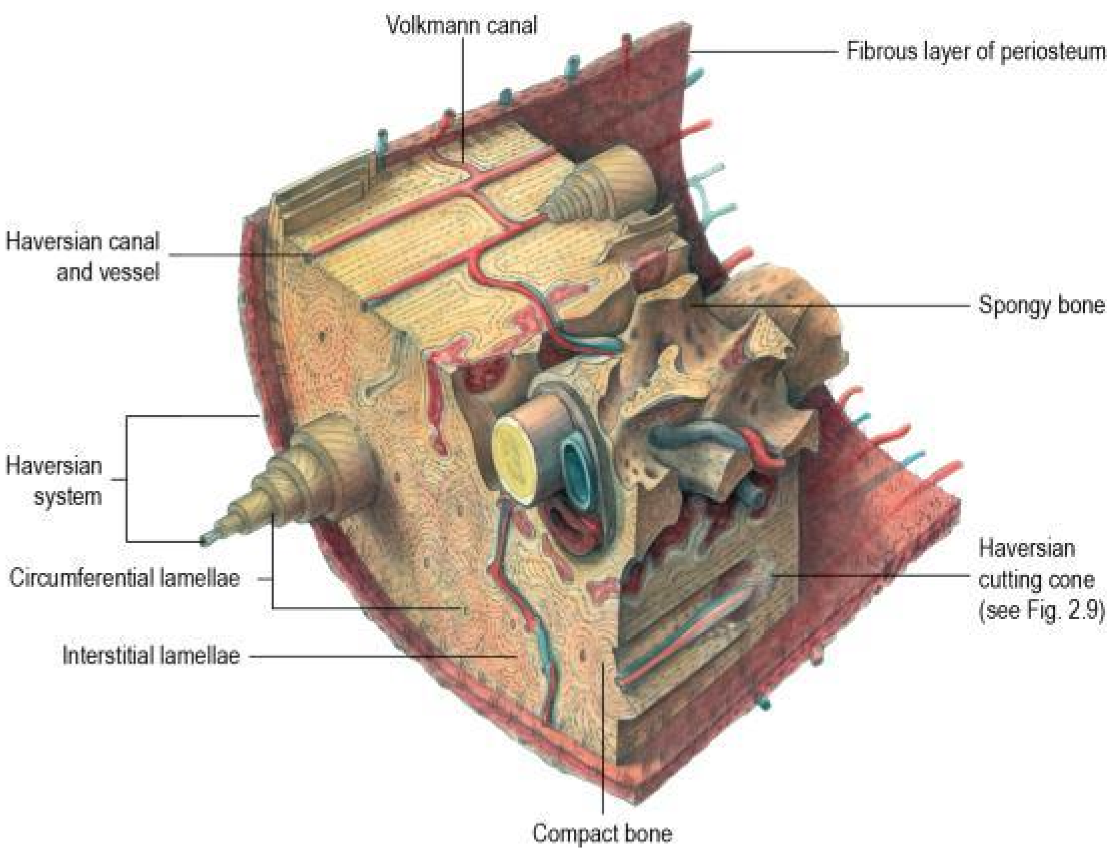
Osteocytes are derived from osteoblasts. Osteocytes essentially are entombed osteoblasts within the mineralized matrix of bone. They transform into osteocytes after they become surrounded within the osteoid. The newly trapped osteocytes are connected with other deeper osteocyte cell layers by long branching processes, which extend into radiating canaliculi for some distance in the mineralized matrix, to the lacunae of neighboring cells. The osteocytes lie between the concentric lamellae.

Osteoclast

Osteoclasts are multinucleated giant cells derived from mononuclear macrophages. Osteoclasts have a specialized role of bone breakdown. These cells are found on the surface of bone in concavities called Howship lacunae. Howship lacunae are small subcellular chambers with a low pH. The pH is maintained by hydrochloric acid, which dissolves the mineral. The organic matrix is degraded by proteases and collagenases, which are thought to be secreted by the osteoclasts. Osteoclasts are capable of resorbing 50–100 μm of bone per day.

Osteon (haversian canal)

Osteons are cylindrical vascular tunnels formed by an osteoclast-rich tissue. They contain pluripotential precursor cells and endosteum known as the cutting cone. The bone removed by the cutting cone is replaced by osteoblast-rich tissue. This is known as the closing cone, which forms concentric layers of lamellar bone that surround the vascular Haversian canal. Volkmann canals also contain nutritional vessels arising from the periosteal and endosteal bone surface, which connect with the Haversian vessels within the osteons.[6] The size of the osteonal transport system is 100 μm, which limits the width of an osteon to approximately 200 μm. The mean width of a lamella is 3–7 μm (Fig. 2.1).



• **FIG. 2.1** Normal bone structure. Source: (From: Härle F. Bone repair and fracture healing. In: Härle F, Champy M, Terry BC, eds. *Atlas of craniomaxillofacial osteosynthesis: miniplates, microplates, and screws*. Stuttgart: Thieme; 1999: 8–14. Reprinted with permission.)

The three most important conditions necessary for bone formation and bone mineralization are pluripotential precursor cells, ample blood supply, and mechanical stability.[7]

Pluripotential precursor cells

In any wound, the cascade of events following an injury is similar: inflammatory response, removal of organic debris, proliferation of fibroblasts and endothelial cells, and the formation of granulation tissue. Bone healing, in addition to these events, must include the removal of necrotic bone and subsequent proliferation of specifically committed osteogenic precursor cells, with the maturation of granulation tissue to callus, fibrocartilage, and, finally, perfectly remodeled bone. This is the typical scenario of classical (or indirect) bone healing, which is discussed in more detail below.

Within the initial hematoma that forms as a result of the fracture, the initial fibrin network acts as a scaffold for the subsequent fibrous connective tissue deposition and the migration of a variety of cell types to the region (e.g., mesenchymal stem cells), which coincides with the upregulation of angiogenic factors that also stimulates a rich vascular invasion into the area.[8] The key point is that the fracture hematoma is an important structure where the local milieu of cytokines and progenitor cells migrate and develop within the forming matrix, which is the start of, and controls, the early phase of bone healing.[9] Hematoma maturation is considered the beginning of the healing process and should be preserved, as its removal can cause a delay of cell differentiation and then subsequent bone formation.[9] This is especially important with bridging comminuted fractures using relative stability, where the fracture hematoma should not be washed away with an open approach, which has alternatively been described as the "open, but do not touch" philosophy with bridging fixation. With anatomic reconstruction and absolute stability, the fracture hematoma may be removed to allow for observation and manipulation of the reduction; the trade-off here is the goal of mechanical stability attained through perfect anatomic reconstruction versus the previous preference for the biology. Understanding the biology and biomechanics that relate to the treatment by these different methods must be rooted in understanding their different effects on the bone-healing process.

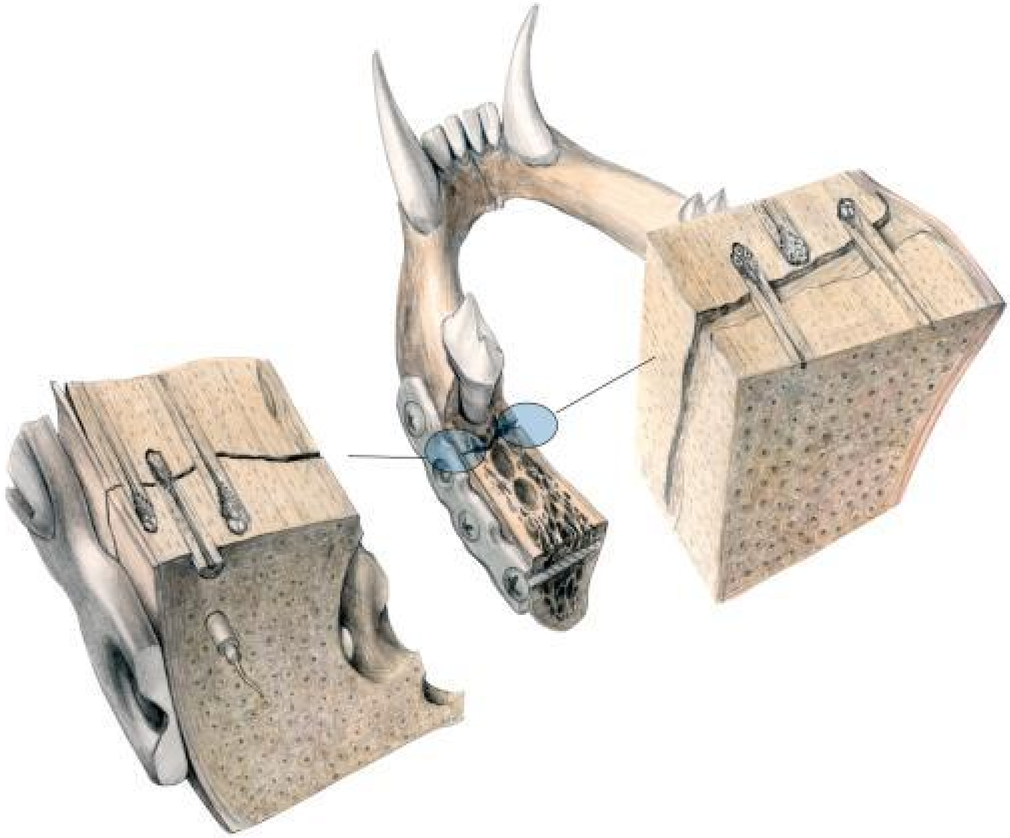
Blood supply

The blood supply to the bones of the mandible is similar to that of the long bones, other than the absence of a true medullary canal and absence of a separate medullary circulation (no hemopoietic elements).[1,10] The inferior alveolar artery performs its function as the nutrient artery to the mandibular body supplying the cortical bone, and additionally supplies the teeth via branching dental arteries, with interdental and interradicular arteries supplying the alveolar bone and periodontal ligament.[1,10] Arterioles supplying the cortex of bone seldom branch, radiating from the nutrient artery, entering the cortex, and supplying the vessels of the Haversian canals; some of these arterioles bypass (traverse) the cortex and anastomose with the periosteal vessels.[2] Periosteal vessels are essentially dormant in the mature animal; however, after injury, all existing (surviving) blood supply becomes locally enhanced, i.e., the normal vascular system increases.[2] The medullary circulation is reestablished by hypertrophy of existing anastomoses and creation of new vascular channels (that occurs over approximately 1 week).[2] In the mandible, when the inferior alveolar artery is blocked, retrograde perfusion is present early, but only in the rostral region; however, after a short interval (within a few days), marked periosteal and symphyseal vascularity and increased medullary perfusion occur.[1,11] Of greater early importance to the healing process is the development of a supplementary blood supply external to the bone; this new blood supply originates from the soft tissues adjacent to the fractured bone and is termed the temporary extraosseous circulation.[2] In the mandible, perforating arterioles from the buccal mucosa maintain the blood supply to the rostral mandible, and in addition, this transient extraosseous circulation becomes established at the fracture site; this vascular supply is transient, and lasts only until the medullary supply returns to its normal dominant position.[2,10,11] Therefore, the health of the surrounding soft tissues in fracture repair cannot be overemphasized, and reestablishing stability is not only important for bone healing, but soft-tissue healing, the latter as a first step in the repair process.

Fracture healing

Fracture healing undergoes the same general processes as with soft-tissue healing: inflammation, removal of organic debris, cellular proliferation, and granulation tissue formation. Fracture healing differs in that there is necrotic bone present, which is difficult to remove, with subsequent proliferation of osteogenic precursor cells, and the transformation of the granulation tissue into callus and, finally, into reconstituted bone. Bone thus is fully reconstituted without the formation of a scar.[3-5] With surgical intervention, stability of the bone fragments favors bone incorporation; on the other hand, persistent instability of the bone fragment favors bone resorption; application of absolute or relative stability with the various fracture repair methods is important as it relates to the concept of strain and its effects on subsequent healing (see Chapter 33).

There are two types of fracture healing, namely indirect and direct bone healing.[3-6] Indirect bone healing occurs via the pluripotential cells located within the cortical and cancellous bone, periosteum, endosteum, and associated soft tissues. Indirect bone healing results from mechanical instability of the fracture, caused by resorption of fracture ends and callus formation. In the case of direct bone healing, the close apposition of the fracture segments provides mechanical stability. Consequently, the osteons of the fracture ends are in direct contact, allowing transverse bridging of the Haversian system with no intervening callus formation (Fig. 2.2). Two different forms of direct bone healing are described: contact healing and gap healing.[6,12] Direct bone healing also constitutes a synergism between contact and gap healing.



• FIG. 2.2 A feline mandibular body fracture with miniplate osteosynthesis on the lateral and aboral (ventral) side of the bone (the bone adjacent to the teeth has been cut away in this diagrammatic representation). Direct bone healing (contact healing) is observed in the buccal cortex of the mandible directly adjacent to (under) the implants (see Fig. 2.9). Direct bone healing (gap healing) is observed in the lingual cortex of the mandible opposite the implants (see Figs. 2.10 and 2.11). Areas with large gaps (>800 μm), or without this degree of rigid fixation, will heal by indirect bone healing.

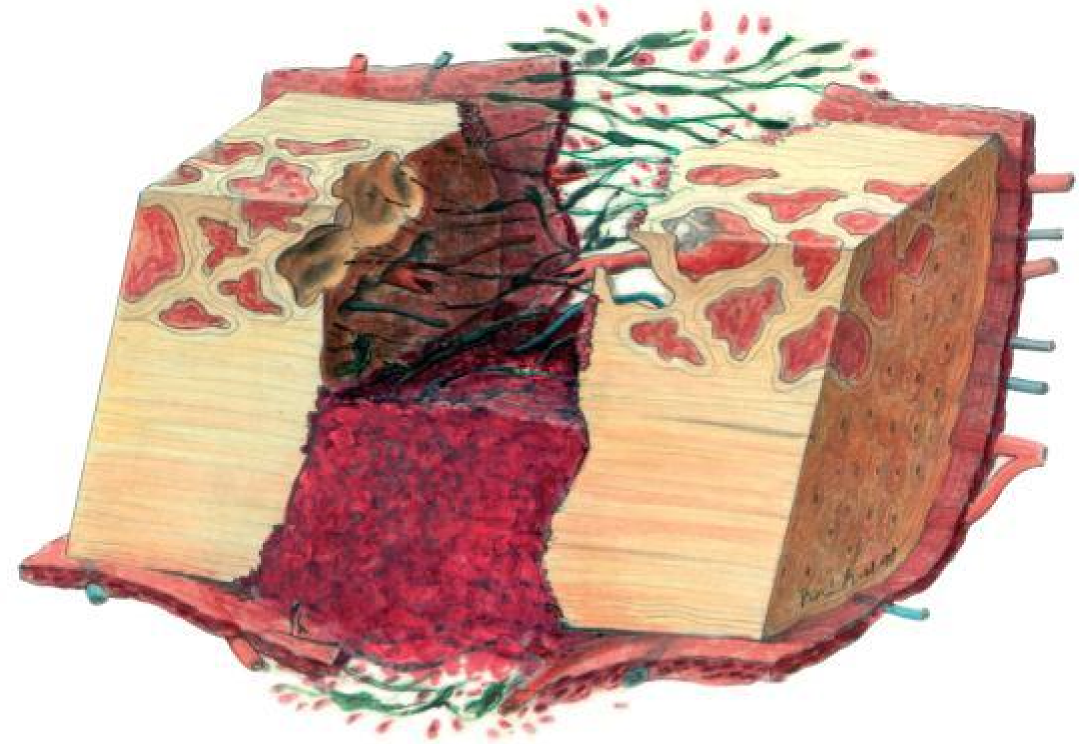
The preferred objective in maxillofacial bone healing is for direct bone healing. Direct bone healing will occur with anatomic reduction and absolutely rigid fixation (absolute stability).[13,14] This form of bone healing results in quicker function, and without the adverse consequences associated with a large callus that occurs with indirect bone healing (relative stability).

The mandible is similar in its bone-healing response to a fracture when compared with other long bones. The bones of the face, on the other hand, consist of a relatively thin lamina, and therefore have a much greater bone surface area per unit volume that is exposed to the soft tissues. This increased proximity to the vascular supply of the soft tissues generally results in rapid healing with few complications.[15-17] There is also a rapid restitution of the vascular supply to the healing bone, which contributes to rapid bone healing.[15]

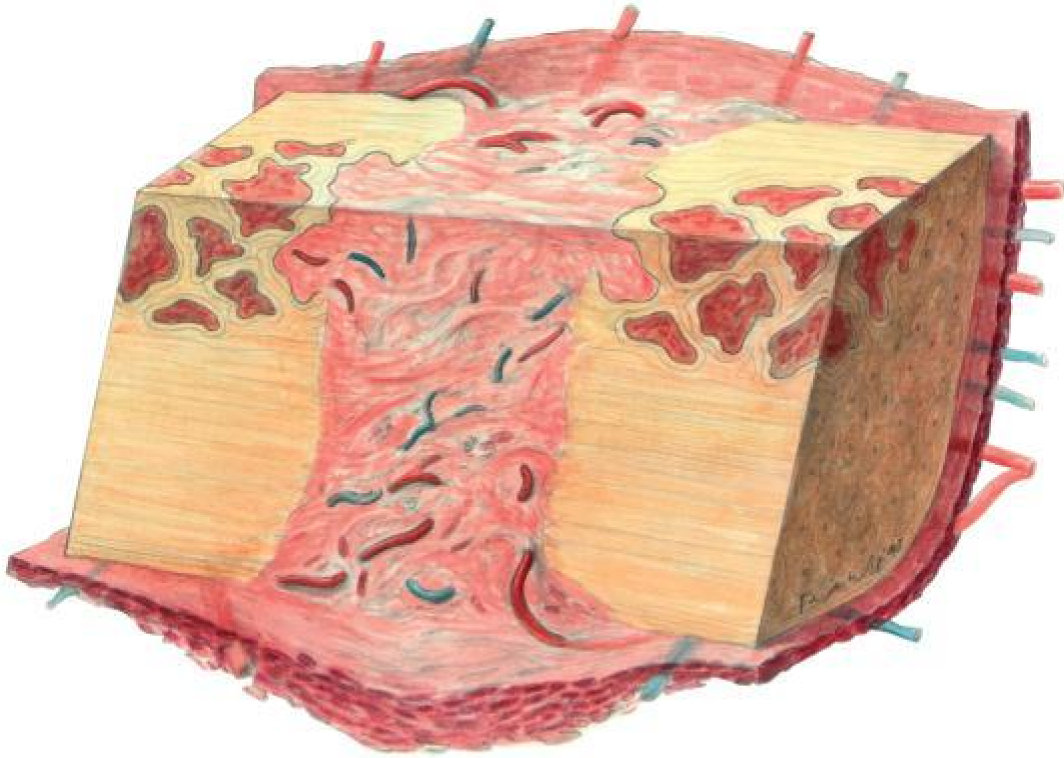
Indirect bone healing

Bone fracture leads to initiation of the inflammatory phase of fracture repair. The fracture causes rupture of blood vessels, torn periosteum, and disruption of the cortex and marrow; contraction and thrombosis of the blood vessels usually limits blood loss. Subsequently, ischemic necrosis of the bone ends occurs within the Haversian and Volkmann canals a few millimeters from the fragment's ends (Fig. 2.3). As a result of this trauma, a protein-rich exudate forms within the fracture site. There is activation of the osteogenic cells of the periosteum and endosteal lining of the Haversian canals and marrow cavity. In addition, a proliferative vascular response ensues (initially a transient extraosseous circulation from the adjacent soft tissues), which also furnishes inflammatory cells and osteogenic progenitor cells to the site. Fibroblastic proliferation occurs within the clot, with accumulation of a cellular infiltrate composed of neutrophils and macrophages. These cells participate in the removal of the debris at the fracture. The osteoprogenitor cells differentiate into fibroblasts and chondroblasts (formation of fibrocartilaginous callus), osteoblasts (formation of new bone), and osteoclasts (resorption of dead bone). These proliferating cells lay down callus, a fibrous matrix of collagen, which thereby forms a bridge across the fracture. A number of contiguous, and overlapping, stages of callus formation can be distinguished. Initially, the formation of periosteal callus leads to a decrease of interfragmentary strain, which is followed by interfragmentary and endosteal callus formation. Granulation tissue invasion replaces the initial hematoma and is transformed into interfragmentary connective tissue (Fig. 2.4) while the ends of the bone fragments are resorbed by osteoclasts (Figs. 2.4 and 2.5).

• **FIG. 2.3** Indirect bone healing: fracture of the bone with rupture of the blood vessels and a hematoma in the surrounding soft tissues. Source: (From: Härle F. Bone repair and fracture healing. In: Härle F, Champy M, Terry BC, eds. *Atlas of craniomaxillofacial osteosynthesis: miniplates, microplates, and screws*. Stuttgart: Thieme; 1999:8–14. Reprinted with permission.)

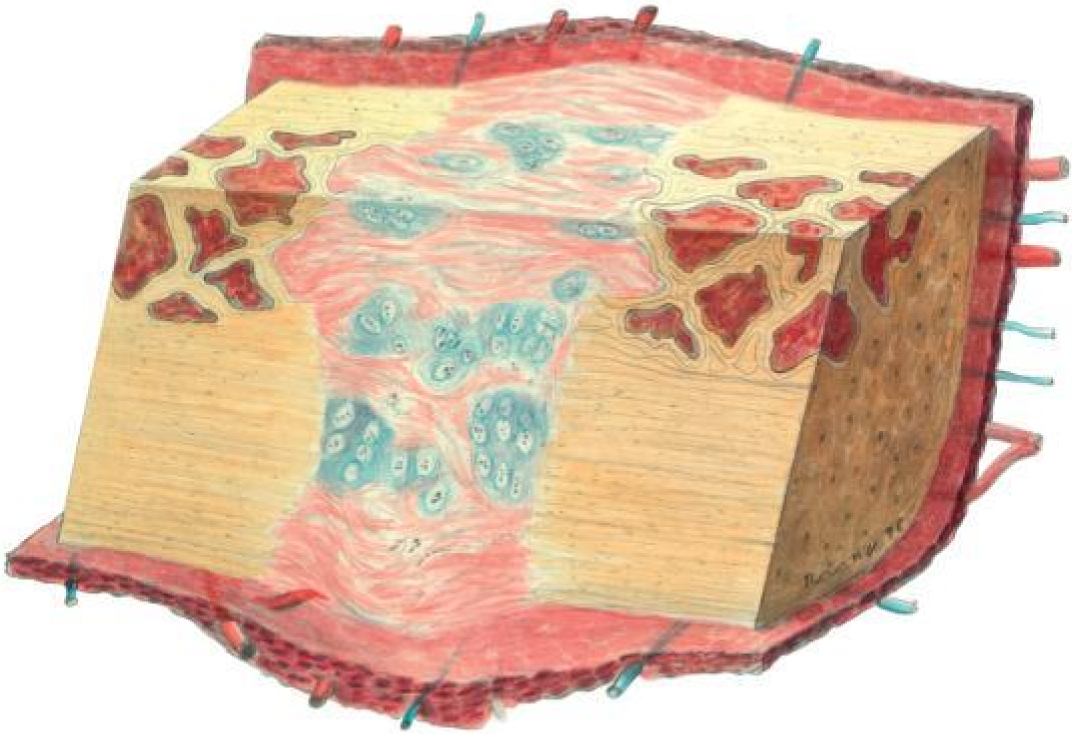


• FIG. 2.4 Indirect bone healing: granulation tissue has replaced the initial hematoma within the fracture site; the necrotic ends of the bone fragments subsequently will be resorbed by osteoclasts. Remaining hematoma is shown only in the ventral part of the fracture site. Source: (From: Härle F. Bone repair and fracture healing. In: Härle F, Champy M, Terry BC, eds. *Atlas of craniomaxillofacial osteosynthesis: miniplates, microplates, and screws*. Stuttgart: Thieme; 1999:8–14. Reprinted with permission.)

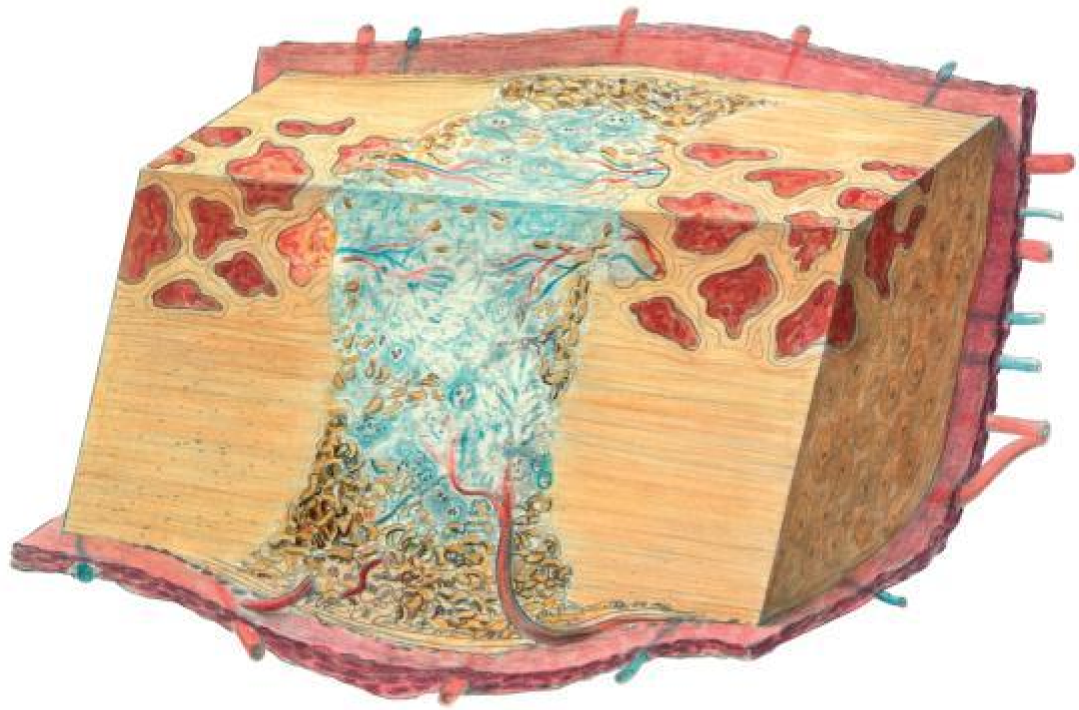


• **FIG. 2.5** Indirect bone healing: the granulation tissue will be remodeled into interfragmentary connective tissue. Source: (From: Härle F. Bone repair and fracture healing. In: Härle F, Champy M, Terry BC, eds. *Atlas of craniomaxillofacial osteosynthesis: miniplates, microplates, and screws*. Stuttgart: Thieme; 1999:8–14. Reprinted with permission.)

The reparative phase of fracture repair consists of consolidation, proliferation, and maturation of this callus. Depending upon the local microenvironment, oxygen tension, pH, tensile, or compressive stresses, the cells within the gap differentiate accordingly. In well-vascularized areas, usually nearest the bone surface, the osteogenic cells transform directly into osteoblasts and lay down a cartilaginous matrix that calcifies directly into bone. In less-well-vascularized areas, the osteogenic cells transform into chondroblasts and form cartilage. The more interfragmentary connective tissue is remodeled into fibrocartilage (Fig. 2.6). Since fibrocartilage is more rigid than fibrous tissue, the interfragmentary tissue becomes stiffer and increases the resistance against motion of the fragments. Subsequently, the fibrocartilage undergoes mineralization. Vascular invasion of fibrocartilage is combined with resorption of mineralized matrix. Calcified fibrocartilage must undergo resorption before osteoblasts can start to produce osteoid as a base for new bone deposition (Fig. 2.7). Initially, the calcified fibrocartilage is replaced by woven bone. After the fracture is bridged by woven bone, stability is obtained and function is possible.[18] This process whereby the cartilage is removed and then replaced with bone is termed endochondral ossification. The increase in this tissue strength, as it is replaced with bone, results in the clinically healed fracture.

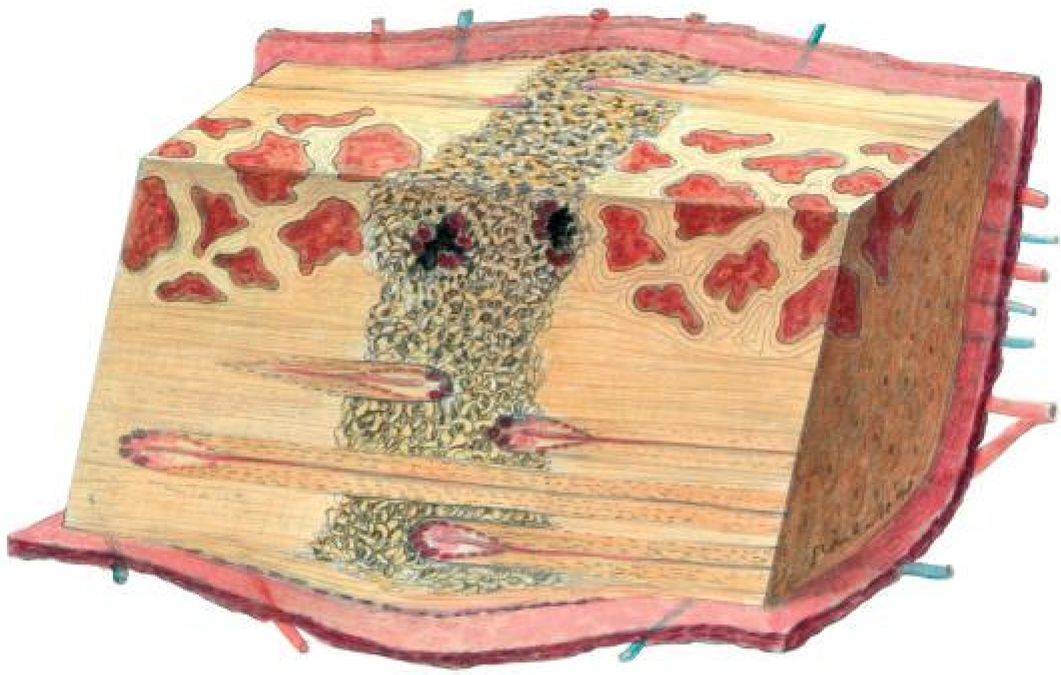


• **FIG. 2.6** Indirect bone healing: the interfragmentary connective tissue will be remodeled into fibrocartilage. Source: (From: Härle F. Bone repair and fracture healing. In: Härle F, Champy M, Terry BC, eds. *Atlas of craniomaxillofacial osteosynthesis: miniplates, microplates, and screws*. Stuttgart: Thieme; 1999:8–14. Reprinted with permission.)



• **FIG. 2.7** Indirect bone healing: calcified fibrocartilage must partially be resorbed before osteoblasts can start to produce osteoid. Source: (From: Härle F. Bone repair and fracture healing. In: Härle F, Champy M, Terry BC, eds. *Atlas of craniomaxillofacial osteosynthesis: miniplates, microplates, and screws*. Stuttgart: Thieme; 1999:8–14. Reprinted with permission.)

The healed fracture undergoes a further increase in strength as the remodeling process restores the bone to its original shape over a prolonged period of time. Redundant bone trabeculae are removed at the same time new structural trabeculae of lamellar bone are formed. This bony deposition in a more advantageous orientation and location is influenced by the stresses within the bone (Wolff's law) to eventually restore the normal pre-fracture bony architecture.[19] This process is termed Haversian remodeling of bone, replacing the woven bone with lamellar bone (Fig. 2.8). It is the identical process whereby bone constantly is remodeled over the animal's lifetime. Haversian remodeling of cortical bone begins with osteoclastic resorption. Several osteoclasts bore a longitudinally oriented cavity (the "cutting cone"). These osteoclasts are followed closely by a vascular loop and osteoprogenitor cells that differentiate into osteoblasts. These cells produce osteoid on the inside wall of the resorption cavity. This osteoid then mineralizes and these successive layers of mineralized osteoid become layers of lamellar bone, leaving a narrow central vascular channel, the Haversian canal.



• **FIG. 2.8** Indirect bone healing: Haversian remodeling begins to reconstruct the lamellar orientation of the bone. Source: (From: Härle F. Bone repair and fracture healing. In: Härle F, Champy M, Terry BC, eds. *Atlas of craniomaxillofacial osteosynthesis: miniplates, microplates, and screws*. Stuttgart: Thieme; 1999:8–14. Reprinted with permission.)

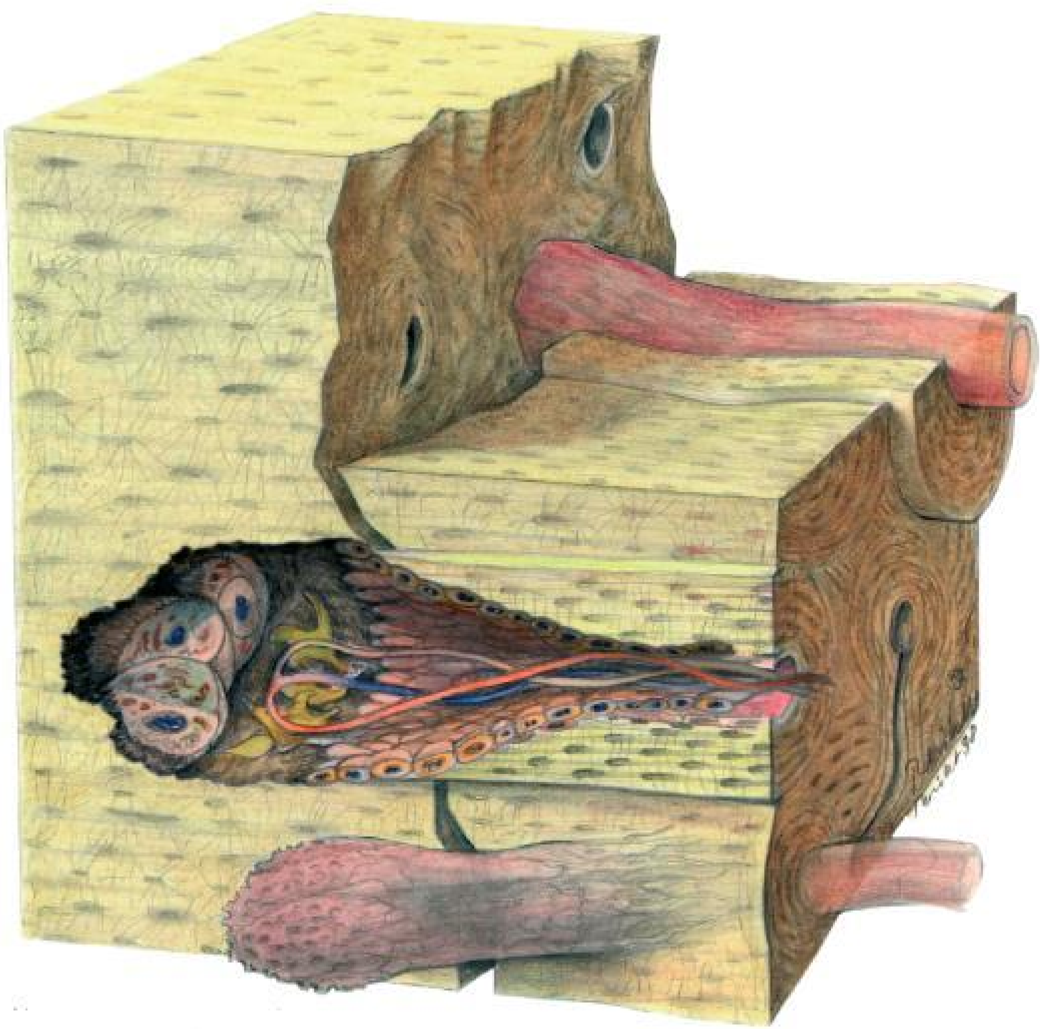
Direct bone healing

Direct bone healing is the process of bone union in which bone is the only type of connective tissue to form between the fracture fragments.[6] Direct bone healing was first described radiographically after perfect anatomic repositioning and stable fixation. Healing of fractures was observed with a lack of callus formation and simultaneous disappearance of the fracture lines. This was originally described as "soudure autogène" (autogenous welding).[20] Callus-free direct bone healing requires what is often called "absolute stability by interfragmentary compression."[21] The biological signal responsible for recruitment of osteogenic progenitor cells, which contributes to callus formation in indirect bone healing, apparently is eliminated with precise reduction and fixation; therefore, direct bone healing occurs with unimpeded cellular function and vascularization via Haversian remodeling.[22] Bone is the only type of connective tissue to form between fracture fragments with direct bone healing; however, the deposition of bone varies, depending upon whether contact or a small gap is present.

If no gap is present, and direct cortical contact is present, contact healing occurs whereby bone union and Haversian remodeling occur simultaneously. If a small gap is present, <800 μm, gap healing occurs whereby bony union and Haversian remodeling are separate sequential processes (with gaps >800 μm, indirect bone healing occurs as previously described).

Contact healing

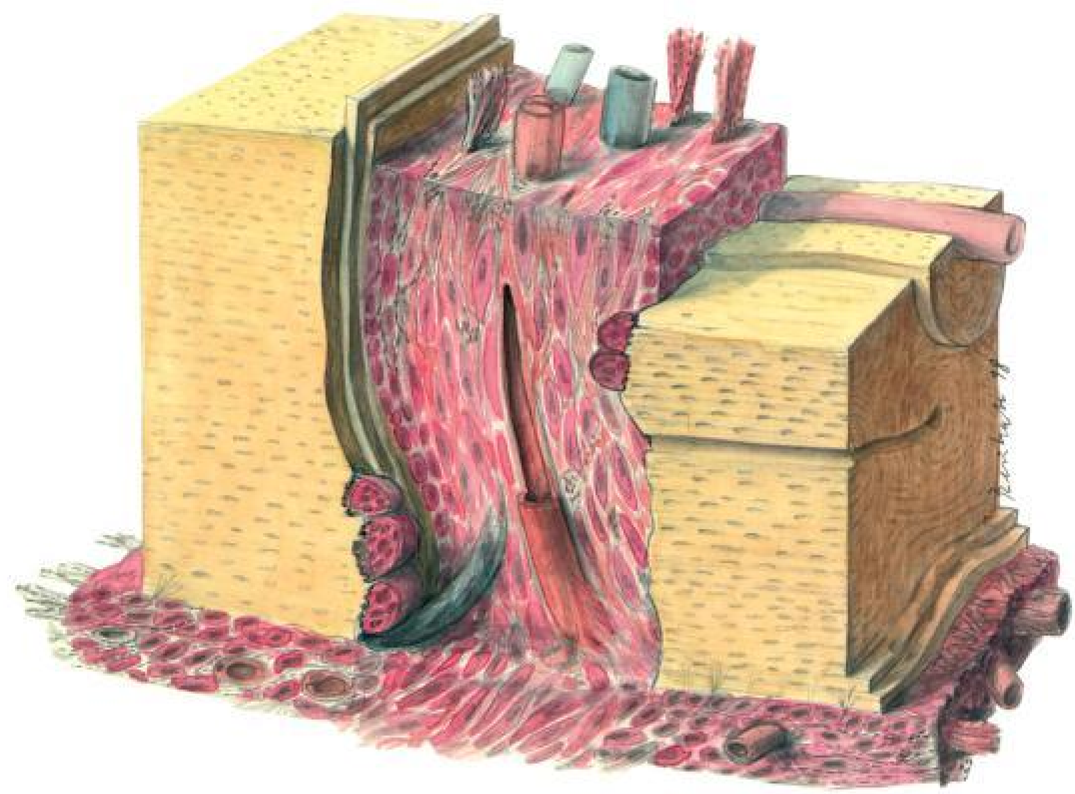
Contact healing of the bone means that healing of the fracture line occurs after stable anatomic repositioning with perfect interfragmentary contact, without the possibility for any cellular or vascular ingrowth.[6,14] Haversian cutting cones are able to cross this interface from one bone fragment to the other by immediate remodeling of the Haversian canal. As previously noted, Haversian canal remodeling is the main mechanism to restore the internal architecture of compact bone (Fig. 2.9). Contact healing takes place after perfect anatomical reduction, osteosynthesis, and absolute mechanical stability. Contact healing generally is observed only directly beneath the fixation, where such direct contact, and interfragmentary compression, can be obtained (see Fig. 2.2).



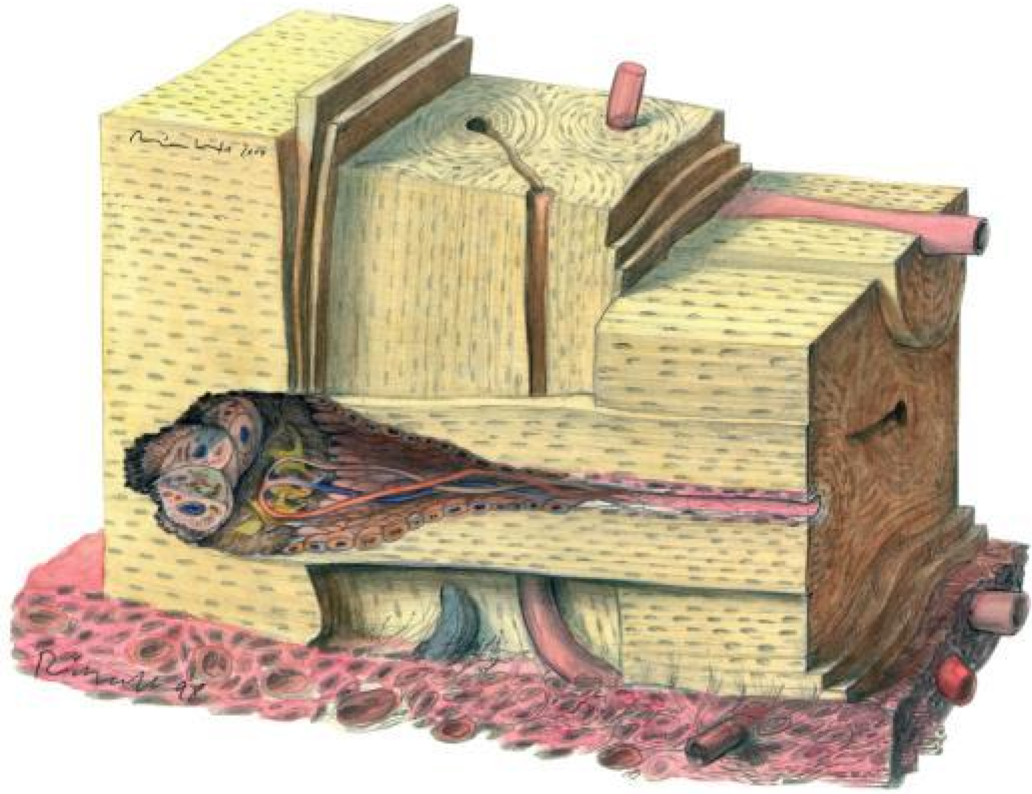
• **FIG. 2.9** Direct bone healing (contact healing): cutting cones cross the interface from one bone fragment to the other by remodeling the Haversian canal (compare with Fig. 2.2, directly adjacent to the implants). Source: (From: Härle F. Bone repair and fracture healing. In: Härle F, Champy M, Terry BC, eds. *Atlas of craniomaxillofacial osteosynthesis: miniplates, microplates, and screws*. Stuttgart: Thieme; 1999:8–14. Reprinted by permission.)

Gap healing

Gap healing takes place in stable or "quiet" gaps with a width of >200 μm osteonal diameter, but <800 μm.[6,14] Ingrowth of vessels and mesenchymal cells starts after surgical stabilization. Osteoblasts deposit osteoid on the fragment ends without osteoclastic resorption. The gaps are filled exclusively with primarily formed, transversely orientated lamellar bone. Replacement is usually completed within 4 to 6 weeks (Fig. 2.10). In the second stage the transversely oriented bone lamellae are replaced by axially oriented osteons, i.e., Haversian remodeling (Fig. 2.11). After approximately 10 weeks, the fracture is replaced by newly reconstructed cortical bone. Gap healing plays an important role in direct bone healing, as small gaps are far more extensive than contact areas. Contact areas, on the other hand, are essential for stabilization by interfragmentary friction as these contact areas protect the gaps against deformation. Gap healing usually may be observed far from the fixation, e.g., the opposite bone cortex, or areas where interfragmentary compression is not obtained (see Fig. 2.2).



• **FIG. 2.10** Direct bone healing (gap healing): osteoblasts deposit osteoid and the gaps are filled with primary formation of transversely oriented lamellar bone (compare with Fig. 2.2, the lingual cortex of the mandible opposite the implants). Source: (From: Härle F. Bone repair and fracture healing. In: Härle F, Champy M, Terry BC, eds. *Atlas of craniomaxillofacial osteosynthesis: miniplates, microplates, and screws*. Stuttgart: Thieme; 1999: 8–14. Reprinted with permission.)



• **FIG. 2.11** Direct bone healing (gap healing): transversely oriented bone lamellae are replaced by axially oriented osteons (Haversian remodeling). Source: (From: Härle F. Bone repair and fracture healing. In: Härle F, Champy M, Terry BC, eds. *Atlas of craniomaxillofacial osteosynthesis: miniplates, microplates, and screws*. Stuttgart: Thieme; 1999:8–14. Reprinted with permission.)

Craniomaxillofacial bone healing

Bone repair and fracture healing to restore original integrity *(restitutio ad integrum)* can be achieved with the use of wire, standard or locking plates (compression or reconstruction), and miniplates, the latter of which provide the optimum environment for direct bone healing. In the mandible, the plates should be placed in biomechanically favorable sites to neutralize the tension forces and torsional movements which cause fracture distraction (see Chapters 27, 32, and 33). Bone plate fixation provides both contact and gap bone healing under conditions of absolute stability. Contact bone healing generally is observed between compressed bone fragments, either adjacent to the plate (cis cortex) or where interfragmentary compression can be applied, e.g., miniplate (see Fig. 2.2 on the bone window in the fracture line on the lateral cortex of the mandible). Gap bone healing generally is observed remotely from the plates, e.g., trans cortex to the plate, e.g., miniplate (see Fig. 2.2 on the bone window in the fracture line on the inner cortex of the mandible).

In the presence of severe comminution, or gaps, bridging fixation is applied with plate fixation in order to maintain three-dimensional anatomic spatial relationships. In this instance, it is more difficult to attain the requisite stability as there is no load-sharing between the plate and bone, and the plate must support all the loads (see Chapters 27 and 33). Therefore, in this situation indirect healing occurs as a result of the relative stability obtained.

Mandibular fracture healing in the dog and cat is similar to healing of long bones.[2,10,11] The obvious primary difference is the presence of the teeth and the mandibular canal. In the dog and cat the tooth roots occupy a considerable volume of the mandible, >40% of the dorsoventral mandibular volume. Because of the presence of the teeth, it is difficult to provide the conditions of rigid stabilization along the alveolar margin, i.e., the tension-band side of the bone, which is the biomechanically preferred location to place implants (see Chapter 27). In humans, implants are placed below the tooth roots and also below the level of the mandibular canal, but remain on the tension-band side of the bone because of the overall large dorsoventral mandibular height.[23,24] Despite the biomechanical advantage of implant (mini- or microplate) location at this level, a second implant (miniplate) may be placed parallel to, and a few millimeters apart from, the tension-band device for added stability. In the dog and cat, the dorsoventral mandibular height is much more limited, and simultaneously has a much larger tooth root volume compared to humans. Despite these anatomic constraints, implants may be placed along the alveolar margin, in this preferential location, between the tooth roots using similar small implants (wire or miniplates; see Chapters 32 and 33). Alternatively, with bridging fixation, a single larger/stronger plate may be placed in lieu of the alveolar miniplate when the tooth to bone height ratio is large (typically in smaller breeds of dogs or cats) or where the overall dorsoventral mandibular height is limited (edentulous mandibles, or toy breed dogs and cats; see Chapter 33). Such "adjustments" to the biomechanical principles can compensate for the degree of relative stability attained, attempting to ensure that the amount of interfragmentary strain stays below the individual tissue strain tolerances.

One principle of application of these implants along the alveolar margin is to avoid penetration of the tooth roots; the implication is that such penetration will lead to tooth death.[25] Despite these recommendations, it is likely that both major (> 50% of screw diameter penetrating the tooth root) and minor (< 50% of screw diameter penetrating the tooth root) contacts with a tooth occur. It is unknown whether significant clinical problems manifest in the dog and cat should this occur; however, studies in humans have shown minimal morbidity in a large series of clinical patients.[26,27]

Due to the large size of the tooth roots in the dog and cat, and the bony area that they occupy, the teeth play an important role in maintaining fracture stability, which directly affects subsequent bone healing. Removal of healthy teeth may increase complications that adversely affect bone healing. Further dissection to excise apical fragments may lead to additional disruption of the blood supply, cause iatrogenic trauma to the adjacent tissues (e.g., further fragment displacement), eliminate available structures for possible fracture fixation, eliminate occlusal landmarks used to realign bone segments to provide for functional occlusion, and create large bony defects that prevent adequate fracture stabilization. In order to enhance the environment for bone healing (and maximize the stability of the fracture repair), healthy teeth that are present within a fracture line generally are not removed unless they cannot be stabilized or unless these teeth are affected by periodontitis.[25]

Additionally, implant placement to avoid the mandibular canal is recommended. This may be difficult in the dog and cat because of the short dorsoventral mandibular height and the large size of the tooth roots. It is possible that this area will be penetrated with implant placement, but with minimal patient morbidity despite the few specific studies to investigate this potential problem.[28] Experimental studies have demonstrated that despite disruption of the nutrient vessel within the mandibular canal (inferior alveolar artery) the teeth remain unaffected, i.e., without evidence of pulpal necrosis or diseased teeth.[28] This is likely due to the rapid reconstitution of the vascular supply from the transient extraosseous circulation (from the surrounding soft tissues) that becomes established shortly after fracture.[1,2]

The midfacial skeleton differs from the mandible in that it consists of a thin lamina of bone, which provides an increased surface area to bone volume ratio as compared with the mandible; consequently, the closer proximity of nutritional vessels provides a superior blood supply to the bone. The result is an increased healing rate of these midfacial bones. A combination of direct and indirect bone healing also occurs in the maxilla, following the principles of miniplate osteosynthesis when applied to this region. However, in the craniomaxillofacial skeleton, interfragmentary compression for direct bone healing is not necessarily required in order to obtain direct bone healing, as demonstrated experimentally and clinically.[29-33] Similar principles of repair applied to human maxillofacial fixation using miniplate osteosynthesis may be applied to the dog and cat (see Chapter 33). All of the considerations with regard to the teeth exist in the maxilla as with the mandible in the dog and cat.

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CHAPTER 3

Use of antibiotics and antiseptics

Eva M. Sarkiala

Definitions

Antimicrobial agents are chemical compounds that kill or inhibit the growth of microorganisms. They may be naturally produced by microorganisms such as fungi (e.g., penicillin) or bacteria (e.g., tetracycline), or they can be synthetically (e.g., sulfonamides and fluoroquinolones) or semisynthetically (e.g., amoxicillin, doxycycline) produced. The term *antibiotic* refers only to natural compounds of microbial origin; however, it is often used as a synonym for antimicrobial agents. Antimicrobial agents are used in animals to treat (therapy) or prevent (prophylaxis) disease.[1]

Antibiotic prophylaxis

Prophylactic antimicrobial therapy is defined as the administration of antimicrobial drugs to a patient to prevent establishment of infection in previously uninfected tissue.[2] The goal of antibiotic prophylaxis is to prevent metastatic bacteremias (i.e., those that originate in one body part and move in blood to infect another body area) and to reduce infections associated with surgery. A prophylactic antibiotic before an invasive dental procedure does not fully prevent bacteremia, but it may decrease its magnitude and persistence.[3] Unfortunately, in some cases antibiotics are used without any indication as “drugs of fear” to prevent claims of negligence.[4,5]

Surgical stress and general anesthesia suppress immune functions in humans, causing granulocytosis and lymphopenia.[6,7] General anesthesia has a transient immunosuppressive effect on canine lymphocytes; T cells are more susceptible to the immunosuppressive effect than B cells.[8] General anesthetics can also affect leukocyte count: in humans, sevoflurane has been reported to increase lymphocyte numbers in the early period during surgery, but decrease them later. However, lymphocyte counts continued to increase during surgery when propofol anesthesia was used.[7] Dental procedures and oral surgery require use of anesthesia for dogs and cats. Does the immunosuppressive effect of anesthesia make veterinary patients more susceptible to infections than humans undergoing the same dental procedures without general anesthesia? This question remains to be answered. The length of anesthesia also affects the risk of infection at the surgery site. A study in dogs reported that in clean orthopedic surgery involving stainless-steel plating systems the risk of infection was increased by ~2% for each minute increase in anesthesia time.[9]

Local anesthesia is recommended and commonly used in veterinary dentistry to reduce the amount of general anesthetic required. Local anesthesia has the advantage of suppressing cortisol and catecholamine levels and reducing muscle breakdown postoperatively. In addition, it has less immunosuppressive effect than general anesthesia.[10]

The American Veterinary Dental College position statement regarding the use of antibiotics in veterinary dentistry recommends the use of systemically administered antibiotics only "for animals that are immune compromised, have underlying systemic disease (such as clinically evident cardiac, hepatic, and renal diseases) and/or when severe oral infection is present."[11]

The following sections may help clinicians develop a specific plan for each patient depending on patient history, health status, and the planned procedure.

Infective endocarditis

Infective endocarditis (IE) is a rare but life-threatening microbial infection of the heart valves or endocardium,[12] previously thought to be induced by bacteremia attributable to dental procedures.[13,14] The incidence of IE is 3–10 per 100,000 persons in most parts of the world and 0.09%–6.6% of dogs presented to a tertiary referral center.[12,15] In cats IE is extremely rare. The median survival time in dogs with aortic or mitral valve IE is 3 days or 476 days, respectively.[12] The most common bacterial causes of IE in dogs in order of frequency include *Staphylococcus* spp. (*aureus*, *intermedius*, coagulase positive, and coagulase negative), *Streptococcus* spp. (*canis*, *bovis*, and β-hemolytic), and *Escherichia coli*. *Bartonella* affects almost exclusively the aortic valve.[12]

The highest risk for endocarditis is reported in human patients with a prosthetic heart valve (20%–30% of all IE incidents).[16]

The most commonly isolated aerobic bacteria in postoperative bacteremia is *Streptococcus viridans*. However, other periodontal pathogenic anaerobic bacteria were found in up to 64% of blood cultures in humans.[17] The prevalence of bacteremia following third molar surgery was 62% at 30 seconds after the first dental extraction and 67% at 15 minutes after finishing the final extraction.[18] However, blood is not sterile before oral manipulations; the incidence of bacteremia is almost as high as a result of activities of daily living (e.g., teeth brushing, flossing, and mastication) as it is after dental procedures.[4,19]

Historically, antimicrobial prophylaxis prior to dental procedures was recommended to prevent IE.[13] Previous recommendations regarding the use of antimicrobial prophylaxis by the American Heart Association (AHA) were based on the assumption that bacteremias induced by dental treatment were the cause of most cases of IE due to *S. viridans*. However, current knowledge suggests that it is far more likely that physiologic bacteremias are responsible for cases caused by *S. viridans,* and antibiotic prophylaxis may prevent few, if any, IE cases in patients undergoing dental treatment.[13,20] Also, adverse effects of antimicrobials may outweigh any benefits in prevention of IE.

Although periodontal disease has been associated with histologic abnormalities in a variety of organs in dogs, including the myocardium and left atrioventricular valves,[21,22] the influence of oral disease or dental procedures on the development of endocarditis in dogs remains controversial. Periodontal pathogens are obligate anaerobic bacteria and are not likely to survive in the highly oxygenated blood of the heart and arterial system. They also lack the surface adhesion factors that are a major factor in the propensity of streptococci and staphylococci to attach to damaged cardiac valves.[4] Most veterinary studies do not support the association between bacterial endocarditis and either dental/oral surgical procedures or oral infection in dogs.[23,24] Furthermore, a study of 76 dogs with and 80 dogs without bacterial endocarditis did not provide evidence of an association between bacterial endocarditis in dogs and oral surgical procedures and oral infection.[24] Myxomatous valve degeneration is the most common cardiac disease in small breed dogs, which also commonly have periodontal disease. However, the most common cardiac disease in dogs with IE is subaortic stenosis. Subaortic stenosis, typically found in large breed dogs, is the only cardiac disease statistically shown to predispose dogs to IE.[12]

The AHA recommends IE prophylaxis for dental procedures only for human patients with underlying cardiac conditions associated with the highest risk of adverse outcome from IE. In 2017, the AHA and American College of Cardiology (ACC) published a focused update to their 2014 guidelines on the use of antibiotics in patients with valvular heart disease. The current guidelines state that use of preventive antibiotics before certain dental procedures is reasonable for patients with prosthetic cardiac valves, prosthetic material used for cardiac valve repair, a history of infective endocarditis, a cardiac transplant with valve regurgitation due to a structurally abnormal valve, and some congenital heart disease.[25] For these patients, prophylaxis is recommended for all dental procedures that involve manipulation of gingival tissue or the periapical region of teeth, or perforation of the oral mucosa, with the following exceptions: local anesthetic injection through noninfected tissue, obtaining dental radiographs, placement of a removable prosthodontic or orthodontic appliance, adjustment of an orthodontic appliance, exfoliation of deciduous teeth, and bleeding from trauma to lips or oral mucosa.[26]

The standard regimen in human antibiotic prophylaxis is a high dose of amoxicillin orally 1 hour, or ampicillin intravenously 30 minutes, preoperatively. For patients with penicillin allergy, clindamycin, cephalexin, cefadroxil, or azithromycin can be used orally, or clindamycin or cefazolin intravenously.[4] Prophylactic perioperative antibiotics such as β-lactam or cephalosporin are indicated in dogs with subaortic stenosis. They may also be indicated in other congenital heart diseases such as pulmonic stenosis or tetralogy of Fallot. Antibiotics should be given 1 hour before a dental or oral surgery procedure and 6 hours after the procedure.[12] There is no evidence that dogs with myxomatous valve degeneration have an increased risk of IE, nor is there evidence of an association between a recent dental procedure and development of IE.[24] Therefore, the use of prophylactic antibiotics prior to dental procedures for dogs with myxomatous valve degeneration is controversial, and further studies are needed.[12]

Prophylactic antibiotic treatment for patients with orthopedic implants

In general, prophylactic antibiotics are not recommended prior to dental procedures for most patients with prosthetic joint implants. For patients with a history of complications associated with joint replacement surgery or who are medically compromised and are undergoing dental procedures that include gingival manipulation or mucosal incision, prophylactic antibiotics should be considered after consulting with the patient and the orthopedic surgeon.[27]

Prophylactic antibiotic treatment for patients with other conditions

Other conditions in humans where antibiotic prophylaxis has been used in association with dental procedures, in the absence of guidelines or recommendations of national medical organizations, are organ transplant recipients, are organ transplant recipients, immunocompromised patients (those with HIV/AIDS, agranulocytosis, cancer, leukopenia, leukemia) when neutrophil count < 1000 mm^3 and/or CD4 count < 200 mm^3, and patients on long-term corticosteroid therapy. Antibiotic prophylaxis may also be indicated in people with poorly controlled type 1 diabetes who require extractions, oral surgery, or periodontal procedures.[20] In patients, especially children, immunocompromised individuals, or during the first 3 years after splenectomy, antibiotic prophylaxis should be considered. Asplenic adult dental patients without risk factors should not routinely receive antibiotic prophylaxis prior to dental procedures.[28]

Antibiotics in oral surgery

The aim of antibiotic prophylaxis in oral and maxillofacial surgery is to prevent infection at the surgical wound site. Characteristics of the surgery, degree of contamination of the surgical field, or the general state of the patient will determine whether antibiotics are indicated. Prophylactic antibiotics may be used in clean-contaminated and contaminated surgeries, and antibiotic treatment is recommended in dirty surgeries.[29]

Noninfected dental extraction, bone grafting, and orthognathic surgery are examples of clean-contaminated oral surgeries. Repair of traumatic wounds with involvement of oral mucosa are considered contaminated surgeries. If antibiotics are not used in contaminated surgeries infection rate may reach 20%–30%. Dirty wounds are those that are infected or result from a trauma in which there has been a delay in treatment, communication with the oral cavity, and possibly the presence of devitalized tissue or foreign bodies. Dirty wounds may have up to 50% infection rate if antibiotics are not used; therefore preoperative and postoperative antibiotics are often used.[2]

In the United States, 17.7% of human oral and maxillofacial surgeons reportedly never use antibiotics to prevent infection, while 59.9% prescribed antibiotics to prevent infection “half of the time,” “often,” or “almost always.” The most frequently used antibiotics are penicillin V, amoxicillin, and clindamycin.[30] Systematic literature reviews in 2014 and 2018 found a lack of randomized controlled trial-based reviews and metaanalyses; uncertainty remains regarding the use of prophylactic antibiotics, which antibiotic to use, and the optimal duration of administration in head and neck and maxillofacial surgery.[31,32]

Dental extractions

Several studies have compared the rate of postoperative complications after third molar tooth extraction surgery in humans with and without the use of preoperative and/or postoperative antibiotics.[33-37] Use of intravenous antibiotics administered prophylactically decreased the frequency of surgical site infections and alveolar osteitis after third molar surgery.[33,36] The longer it took to extract the third molar tooth, the higher the rate of infection (13.8% for long versus 1.6% for short procedures). Also, the difficulty of the procedure increased the rate of infection (with ostectomy, 12.7%, versus without ostectomy, 3.5%).[34] The risk of infection in patients who required ostectomy was 24%, 9%, and 4% with placebo, presurgical dose of amoxicillin-clavulanate, and preemptive 5-day postsurgical treatment with amoxicillin-clavulanate, respectively.[34] A recent study involving 2954 human patients who had at least one third molar tooth removed in a private practice setting by oral and maxillofacial surgeons concluded that any type of antibiotic use versus no antibiotics resulted in inflammatory complications (surgical site infection or alveolar osteitis) in 5.0 and 7.5% of cases, respectively.[37] A Cochrane Review in 2013 concluded that prophylactic antibiotics decrease the risk of infection, dry socket, and pain after third molar tooth extraction and result in an increase in mild and transient adverse effects. However, 12 people needed to be treated with antibiotics to prevent one infection following extraction of impacted wisdom teeth.[38]

The authors of a study involving surgical removal of 528 impacted lower third molar teeth reported that, compared with no antibiotic treatment, postoperative oral antibiotic treatment with amoxicillin-clavulanic acid or clindamycin did not contribute to better wound healing, reduced pain, or increased mouth opening, nor did it prevent inflammatory problems after surgery. They concluded that postoperative oral antibiotic treatment should not be routinely recommended.[35] Scientific veterinary medical studies reporting the indication for antibiotic therapy in association with extractions are not currently available. Based on the data from human studies, however, it can be reasonably assumed that antibiotics are not needed for most veterinary patients undergoing extractions. Veterinarians may consider a perioperative dose of antibiotics for patients who require multiple surgical extractions with ostectomy (i.e., extensive oral surgery), those with severe, generalized periodontitis, and/or patients with medical conditions that might impair their ability to clear the anticipated bacteremia. (See recommendations for patients with other health problems in the "Antibiotic Prophylaxis" section.)

Maxillary and mandibular fractures

Mandibular fractures have a higher rate of infection than maxillary fractures do, and therefore most studies of antibiotic prophylaxis in humans with facial trauma have examined the use of antibiotics for mandibular fractures.[2,39] The highest rate of infection is associated with compound fractures, either open to the oral cavity or with teeth in the line of fracture.[2] The infection rate of mandibular fractures treated with open reduction and internal fixation (ORIF) and with antibiotic prophylaxis is on average 7.6%.[40] Recommendations regarding the use of antibiotics in surgical treatment of mandibular and maxillary fractures vary. Some authors recommend prophylactic perioperative antibiotic administration initiated prior to surgery but not exceeding 24 hours.[41-43] Several studies conclude that postoperative use of antibiotics is often unnecessary in humans and dogs.[41,43-48] Two human studies confirmed that postoperative oral administration of antibiotics (for 5 to 10 days) offered no additional benefit beyond perioperative intravenous antibiotic administration in patients with mandibular fractures.[47,48] Other studies failed to demonstrate any statistically significant benefit with the administration of postoperative antibiotics in patients undergoing open reduction and internal fixation of mandibular fractures.[44,46] Antibiotic therapy had no impact on the development of postoperative complications after repair of mandibular fractures using ORIF alone, maxillomandibular fixation (MMF), or a combination of MMF and ORIF.[45]

In the case of mandibular fractures with communication to the oral cavity, five randomized studies in humans indicate that antibiotics significantly reduce the risk of fracture site infection.[39] Antibiotic prophylaxis is also justified in compound or open fractures and those communicating with paranasal sinuses in humans.[29] When ampicillin/sulbactam or cefazolin was used, the infection rate after ORIF was 0%, and using clindamycin, the infection rate was 19.4%. The authors recommended perioperative use of cefazolin plus metronidazole for humans with ORIF treatment of mandibular fractures.[40]

Maxillectomy and mandibulectomy

Proper surgical technique reduces the likelihood of development of postsurgical infection.[49] Oral soft tissues should not be traumatized, electrocoagulation should not be used, if possible, and irrigation must be used to prevent thermal necrosis of bone during osteotomy, ostectomy, and osteoplasty. Routine periodontal treatment should be performed for dogs and cats prior to major oral tumor surgery. This results in a cleaner surgical field and less inflamed gingival tissue at the time of the surgery.[49]

A review of 547 human patients who had major contaminated oncologic head and neck surgery concluded that perioperative antibiotic administration should be performed, but no evidence exists to support prolonged administration of antibiotics beyond the first 24 hours following surgery.[42] A 1-day antibiotic regimen was as effective as a 3-day regimen in preventing wound or systemic infection in clean-contaminated head and neck cancer surgery without flap reconstruction.[50] Healthy patients undergoing surgery of the salivary glands do not need antibiotic prophylaxis.[29]

Luxated or avulsed teeth

The administration of systemic antibiotics after replantation is questionable. Positive effects upon periodontal and pulpal healing have usually been found in experimental studies.[51] When extracted monkey teeth were treated with topical doxycycline (1 mg/10 mL for 5 minutes) before reimplantation, infection-related root resorption was reduced or prevented.[39] A more recent study concluded that avulsed permanent teeth soaked in doxycycline did not have better pulp survival and periodontal healing when compared with teeth soaked in saline.[52] The 2012 guidelines of the International Association of Dental Traumatology recommend antibiotics for 7 days postoperatively after tooth reimplantation. The first choice is tetracycline, and other options are penicillin or amoxicillin. In immature teeth, topical antibiotics (minocycline or doxycycline, 1 mg per 20 mL of saline for 5-minute soak) may be considered.[51]

Antibiotic use in periodontal surgery

Ideally, scaling and polishing should be a pretreatment procedure in dogs with severe periodontitis and a postscaling examination should be performed a couple of weeks later. As a result, periodontal tissue would have time to heal, and a determination of surgical treatment would not be done on unhealthy tissue. However, in veterinary dentistry, scaling, polishing, and other periodontal treatment are often performed during one anesthetic episode.[53]

Studies in human patients have indicated that the incidence of infection after periodontal surgery is very low in patients treated with or without antibiotics. Therefore, unless there is a medical indication, there is no justification for using prophylactic antibiotic therapy to prevent infection following periodontal surgery.[54,55] Antimicrobial resistance of subgingival bacterial flora in dogs appears to be increasing, as evidenced by the results of two veterinary studies that were published 11 years apart.[56,57] Subgingival microflora in dogs with severe gingivitis or periodontitis were highly susceptible to commonly used antibiotics in the 1995 study.[56] In that study, amoxicillin-clavulanic acid had the highest in-vitro susceptibility (96%) against all canine isolates tested. In a similar study evaluating bacterial isolates from cats with gingivitis, amoxicillin-clavulanic acid had the highest in vitro susceptibility against all subgingival isolates, and both amoxicillin-clavulanic acid and clindamycin were effective against all anaerobes.[58]

In a 2006 study, antimicrobial resistance of subgingival aerobic and anaerobic flora to commonly used antibiotics in dogs with periodontal disease was high.[57] Anaerobic bacteria appeared to be the most susceptible to amoxicillin-clavulanic acid, doxycycline, and erythromycin; aerobic bacteria appeared to be most susceptible to amoxicillin-clavulanic acid, erythromycin, gentamicin, and sulfa-trimethoprim. Although antimicrobial resistance to amoxicillin-clavulanic acid was the lowest of the commonly used antibiotics in dentistry, it was still significant (e.g., *Prevotella intermedia* 33.3%, *Porphyromonas gingivalis*, and *Peptostreptococcus* spp. 25%). *Bacteroides fragilis* was resistant to all antibiotics tested.[57] Susceptibility to clindamycin was not tested in this study, but resistance to metronidazole and doxycycline was high. A more recent study reported that in dogs with or without periodontitis all strains of *Porphyromonas* and *Fusbacterium* were susceptible to all commonly used antibiotics tested (i.e., amoxicillin, ampicillin, amoxicillin/clavulanate, cefoxitin, clindamycin, penicillin G, and tetracycline).[59]

Although amoxicillin may still be advocated as a suitable first-line agent in periodontal therapy in humans, reduced susceptibility of *Prevotella* strains remains a matter of concern with penicillins.[60-62] Studies in humans and dogs conclude that amoxicillin-clavulanic acid, clindamycin, doxycycline, and metronidazole are useful alternatives in combating the anaerobic bacteria involved in dentoalveolar infection and aggressive periodontitis.[59,60]

Locally delivered antibiotics

Subgingival treatment of periodontal pockets with controlled-release tetracycline or minocycline is an option for dogs and cats with periodontitis, and in several studies has resulted in substantial improvement of periodontal health in dogs and humans.[63-66] However, the length of the studies using dogs was only 2 month[65] and 4 months.[64] Some studies have failed to show statistically significant clinical or microbiological results between use of subgingival doxycycline in addition to scaling and root planning (SRP) and SRP alone.[67-69] One study reported a 3-month positive effect on clinical parameters with the adjunctive use of locally delivered doxycycline, but applications repeated annually had no clinical or microbiological effect.[67] Subgingivally delivered, sustained-release doxycycline polymer is registered and marketed for veterinary patients (Doxirobe, Zoetis, Parsippany, NJ, USA) for treatment of periodontal pockets with probing depths 4 mm or deeper after periodontal debridement.[53]

Even at subantimicrobial doses, doxycycline downregulates the activity of matrix metalloproteinases (MMPs), key destructive enzymes in periodontal disease.[70] Therefore, subantimicrobial dose doxycycline (SDD), usually 20 mg BID for 9 months, is sometimes used in the treatment of chronic adult periodontitis. In the treatment of experimentally induced periodontitis in beagle dogs, the clinical periodontal status significantly improved in the treatment group (SDD 2 mg/kg once daily for 8 weeks) compared with the placebo group.[65] Several studies report that SDD as an adjunct to SRP resulted in significantly greater clinical benefits than SRP alone, and appears to be safe and effective in the long-term management of chronic periodontitis.[70-73] In addition, several studies report no evidence of microbiologically significant changes or development of antibiotic resistance in man or dogs treated with SDD.[65,70-72] However, one author states that the clinical relevance of improvements obtained with SDD beyond those obtained with conventional therapy alone is debatable.[74] SDD might be an option to manage some dogs with chronic periodontitis when professional periodontal treatment and dental home care are not effective enough to control the disease.

Side effects of antimicrobial treatment

Risks associated with antibiotics include hypersensitivity reactions (such as anaphylaxis), development of antibiotic-resistant bacteria, superinfections (such as antibiotic-associated colitis and/or diarrhea), pseudomembranous colitis (PMC), photosensitivity, long QT interval, crossreactions with other drugs, and death. The costs involved with the use of antibiotics can be significant as well.[20,75]

Allergic reactions to antibiotics are reported less frequently in veterinary medicine than in human patients.[76-78] Acute anaphylaxis is characterized by one or more of the following signs: hypotension, bronchospasm, angioedema, urticaria, erythema, pruritus, pharyngeal and/or laryngeal edema, vomiting, and colic. Penicillins, cephalosporins and sulfonamides most frequently cause hypersensitivity reactions in animals.[76] In humans, the most common antibiotic capable of inducing diarrhea, colitis, or PMC is amoxicillin, followed by cephalosporins and clindamycin.[75] Reported allergies alter antibiotic therapy in 30% of human patients.[79]

Bacterial resistance to antimicrobials

The occurrence of bacterial resistance corresponds to an increase in an antibiotic's minimum inhibitory concentration (the lowest concentration of the antibiotic capable of inhibiting bacterial growth) for a particular bacterial strain.[80] Bacterial resistance to antimicrobials has been an ongoing challenge for clinicians ever since the discovery of antimicrobial agents. Bacteria have succeeded in developing resistance to all antibacterial agents shortly after they have been marketed.[75,80] Bacteria have developed several mechanisms to neutralize the action of antimicrobial drugs; these are enzymatic drug inactivation, modification/protection of the target site, limited access of antibiotic (altered cell membrane permeability), active drug efflux, use of alternative growth requirements, and overproduction of target sites.[75] Improper use of antibiotics, in terms of either dosage (duration of treatment too long or dose too weak) or indication, is a major factor in the emergence of antibiotic resistance.[80,81] Growing concerns regarding antibiotic resistance caused by overprescribing should therefore be balanced with the potential benefits of prophylactic antibiotic administration.

Bacteria in dental plaque are present in a biofilm.[53,55] Concentrations of antibiotics necessary to inhibit subgingival plaque bacteria in steady-state biofilms have been reported to be 250–1000 times greater than the concentrations needed to inhibit the same strains grown planktonically.[55,82] Bacteria also become progressively more resistant as the biofilm matures.[55,82] Several mechanisms confer greater bacterial resistance in a biofilm: low bacterial metabolic activity within the biofilm limits the assimilation of antibiotics; extracellular matrix of the biofilm limits the diffusion of antimicrobial agents; and extracellular bacterial enzymes involved in deactivating antibiotics are trapped and concentrated in the biofilm.[80] As a result, antibiotic sensitivity tests may be of limited use, given that the behavior of bacteria inside the biofilm is completely different from that observed for the same bacteria in planktonic form.[80,83] Removal of plaque by mechanical debridement (scaling and polishing) is therefore essential prior to considering antimicrobial therapy in the treatment of periodontal disease.[55]

Antimicrobial resistance in animals also poses a risk to humans. Horses, dogs, cats, and exotic animal pets can be reservoirs of resistant bacteria. Companion animals may transfer to people bacteria including methicillin-resistant *Staphylococcus aureus* (MRSA) or *S. pseudintermedius,* extended spectrum β-lactamase (ESBP)-producing *Escherichia coli,* multidrug-resistant (MDR) *Salmonella* serovars, and *Campylobacter jejuni*.[84,85] There is now increasing evidence that MRSA can be transmitted in both directions, from human to animal and from animal to human. Once exposed to MRSA, animals can become colonized, and may serve as reservoirs to transmit the infection to other animals and to humans.[84-91] A study in a university veterinary hospital reports that certain MRSA genotypes are capable of infecting a wide spectrum of small and exotic animals.[92] MRSA isolates from dogs and cats are indistinguishable from human healthcare isolates.[89]

The emergence and dissemination of MDR bacteria is a serious problem in veterinary medicine. Veterinarians should carefully evaluate whether antibiotics are indicated in veterinary dentistry and oral surgery. Antibiotic resistance among bacterial isolates may show differences between countries, geographic regions, and even hospitals. It is important to choose as narrow spectrum an antibiotic as possible in light of the resistance situation in the region.

Oral antiseptics

Antiseptics are substances that prevent or arrest the growth or action of microorganisms on living tissue, either by inhibiting their activity or by destroying them.[93]

Chlorhexidine

Chlorhexidine is a cationic biguanide, available as a solution, gel, or scrubbing agent.[93,94] Chlorhexidine exhibits a broad spectrum of antibacterial activity; it is highly effective against Gram-positive organisms, less so against Gram-negative ones.[93,95] Oral chlorhexidine gel has been shown to inhibit the formation of plaque and reduce the severity of gingivitis in dogs.[96] A study in humans showed that chlorhexidine gel is less effective than oral rinse in plaque inhibition.[97] The cationic nature of chlorhexidine allows it to bind to negatively charged surfaces in the mouth, such as teeth and mucosa.[93,95,96] It is then released over time and provides a continuing bacteriostatic effect.[95] Chlorhexidine does not absorb through the oral tissues.[96]

Chlorhexidine gluconate is commercially available as a 0.12% oral rinse or gel for animals. Before dental treatment and oral surgical procedures, the mouth should be irrigated with an antiseptic solution, such as chlorhexidine.[49,98] Rinsing the mouth preoperatively with chlorhexidine produced significant reductions in bacterial aerosols during ultrasonic scaling in humans[99] and in the number of bacteria entering into the bloodstream in dogs.[100] In the previously mentioned study concluding that postoperative antibiotics do not prevent inflammatory problems after third molar tooth surgery, the mouths of all patients were rinsed with 0.2% chlorhexidine before their extractions, which may have decreased the oral bacterial flora and possibly prevented infection and dry socket.[35]

There are controversial results as to whether chlorhexidine prevents alveolar osteitis (AO) in humans. Irrigation with chlorhexidine for 7 days after the extraction of third molar teeth helped reduce pain and lower the incidence of AO.[101] A metaanalysis by Caso et al. (2005) reported that when postoperative rinses were used, three of five studies showed a significant reduction in AO when the mouth was rinsed with chlorhexidine preoperatively and followed by postoperative rinses for several days.[102] A systematic review paper did not find sufficient evidence supporting the use of chlorhexidine rinse or gel in the prevention of AO.[103]

In veterinary dentistry, chlorhexidine gluconate oral rinse may be used before dental and oral surgical procedures, postoperatively in some patients with severe oral inflammation, or after extractions or other oral surgery. Chlorhexidine may also be used to reduce plaque accumulation on tooth surfaces, since many owners may not be willing or able to brush their pets' teeth. Chlorhexidine mouth rinse maybe more effective than gel when oral hygiene cannot be performed.[97]

Side effects of chlorhexidine in humans include reversible staining of teeth, increased calculus formation, and alteration in taste perception. The incidence of side effects is significantly reduced with 0.12% chlorhexidine gluconate compared with 0.2%.[94]

Zinc ascorbate

Zinc ascorbate is available as an oral gel for animals. Cats seem to tolerate the taste of it better than chlorhexidine. One study reported that it decreases bacterial growth, plaque formation, and gingivitis in cats when applied following a professional teeth cleaning procedure.[104]

Povidone-iodine

Elemental iodine and its derivatives (polyvinylpyrrolidone-iodine complex, PVP-iodine) are among the most broad-spectrum and potent antiseptics available. Povidone-iodine is used as an oral antiseptic in humans. In the treatment of periodontal disease, subgingival 10% povidone-iodine application in addition to mechanical debridement reduces pocket depth and total counts of periodontal pathogens and helps control periodontal disease in man. A recent study concluded that povidone-iodine is even more effective than chlorhexidine in reducing the bacteremia prior to oral surgical procedures.[105] Povidone-iodine has a broad spectrum of antimicrobial action and is less likely to induce development of resistant bacteria and adverse host reactions compared to antibiotics.[106,107]

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CHAPTER 4

Anesthesia and pain management

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Definitions

Pain has been defined by the International Association for the Study of Pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage." One of the notes appended to this states, "The inability to communicate in no way negates the possibility that an individual is experiencing pain and is in need of appropriate pain-relieving treatment." For veterinarians this is an important qualification, since our patients cannot "describe" their pain. A specific definition of animal pain has been given as "an aversive sensory emotional experience representing an awareness by the animal of damage or threat to the integrity of its tissues (note, there may not be any damage): it changes the animal's physiology and behavior to reduce or avoid the damage, to reduce the likelihood of recurrence and to promote recovery; non-functional pain occurs when the intensity or duration of the experience is not appropriate for the damage sustained (especially if none exists) and when physiological and behavioral responses are unsuccessful in alleviating it."[1] This definition addresses both the survival function of pain and chronic pain where the animal has exhausted its ability to deal with the problem and is condemned to suffer. As veterinarians we are dedicated to the comfort of our patients, and so the provision of analgesia is a primary goal in the management of any animal.[2,3]

Hyperalgesia is present when there is an exaggerated response to a stimulus that would normally be noxious. This can either be *primary*, where it is related to changes in sensitivity of the peripheral neurons; or *secondary*, where it is due to changes in central processing of the neuronal input. When a noxious stimulus is applied to tissue, there is a release of various mediators such as histamine, bradykinin, prostaglandins, and various cytokines that reduce the threshold for further stimulation of the nociceptors in the injured area (primary hyperalgesia). Secondary hyperalgesia occurs because the nociceptive input into the spinal cord interacts with adjacent neurons and sensitizes them to further stimuli. This is manifested clinically as a change in nociceptive threshold outside the area of injury.[4] *Allodynia* is defined as "pain due to a stimulus which does not normally provoke pain," and this would apply to the pain associated with hot or cold liquids on exposed dentin as opposed to the lack of pain to such a stimulus on normal enamel.

Principles of pain management

Assessment of pain

Since pain can only be felt by the individual who has been injured, and our patients cannot communicate verbally, our assessment of pain depends on observation of the behavior of the animal and our clinical acumen. It must be assumed that animals that are brought into a strange environment (a veterinary hospital) are anxious and stressed and therefore less likely to tolerate pain. Veterinarians are increasingly using methods to decrease this unwanted anxiety using combinations of behavioral modification (e.g., https://fearfreepets.com/veterinary-professionals/) and anxiolytic or sedative drugs administered before the visit.[5] The second part of being able to assess pain is to know the behavior of the animal. In general terms, cats and dogs tend to respond in different ways: a cat in pain is more likely to crawl to the back of the cage and curl up into a ball whereas a dog is much more likely to vocalize and draw attention to its discomfort. There are breed differences as well as differences related to each individual patient. Animals may show gross signs of being in pain (howling, guarding some particular site, restlessness) or the signs may be more subtle (e.g., adoption of a particular posture, retreating to the back of the cage, a change in temperament—usually becoming more aggressive, failure to interact normally with the caregiver).[6,7] These signs must be assessed in light of the analgesics the animal has already received and the level of consciousness of the patient. There have been no studies in dogs looking at behavior following dental extractions, but one study in cats used an adapted pain scoring system.[8,9]

Efforts to develop a satisfactory scale for the objective assessment of pain in animals are ongoing. The Glasgow University Composite Measure Pain Score[10] may be useful for postoperative pain assessment but was found to be less accurate than a visual analog scale for assessing pain in dogs undergoing radiation therapy[11] due to the initial influence of anxiety in these patients early in the course of treatment. In cats a new scale has been validated and a consensus arrived at for signs of feline pain.[12-14] Facial expression in cats has also been examined in acute pain,[15] and several groups are working on facial expression in dogs.

Regardless of the scale employed, it is best for all hospital employees to use a standardized method for assessing pain, since it is likely that individuals using a particular approach will develop their skills and will be motivated to assess pain routinely.[4,16-18] Such an approach has been embodied in veterinary medicine by the adoption of pain management standards by the American Animal Hospital Association.[19]

Management of pain

Pain is the result of a complex series of interactions in the nervous system. Since there are systems that facilitate and inhibit the passage of nociception to the cerebral cortex, it is possible for a noxious stimulus to be perceived with equanimity or as the most horrible experience. This complexity allows us to intervene to reduce the input, reduce the result of that input, minimize facilitation within the central nervous system (CNS), or block perception.[20]

Reducing the input

Continuous stimulation of C-fiber input tends to produce central sensitization or facilitation to further stimulation, which is largely responsible for secondary hyperalgesia. The simplest way to block the input is to use a local anesthetic such that none of the stimulated neurons deliver their impulses to the CNS. Drugs blocking prostaglandin production (nonsteroidal antiinflammatory drugs; NSAIDs), stimulating opioid receptors, and blocking alpha-1 adrenergic receptors can also affect the input.

Reducing the result of the input

The powerful descending inhibitory systems use opioid, alpha-2 receptors, and serotonin as well as other neurotransmitters, and these decrease what reaches the cerebral cortex. Part of the secondary hyperalgesia is mediated by *N*-methyl-d-aspartate (NMDA) glutamate receptors in the spinal cord. Ketamine is an NMDA antagonist and so may prevent this facilitation.

Minimizing facilitation

The central processing of nociception can be enhanced by pathways descending from the brain to the spinal cord. These are triggered by anxiety, stress, and sleeplessness, and thus any actions to minimize these for the patient may decrease their experience of pain. This could consist of very simple things such as handling the animal gently or could involve the use of drugs to minimize the anxiety and stress (e.g., phenothiazines, benzodiazepines).

Blocking perception

Any anesthetic will make it impossible to feel pain since any feeling requires consciousness. It should be noted that many anesthetics do not alter the input into the CNS from a surgical site and therefore, while the animal may not perceive the pain at the time, facilitation may occur, making the animal more susceptible to pain postoperatively.

The complexity of the system also allows us to try to attack it at several different points at the same time. This is the principle of *balanced analgesia*—using several different drugs to block the perception of pain at various points throughout the system. For example, a balanced analgesia approach might include premedication with a phenothiazine and an opioid, induction with a dissociative drug (NMDA antagonist), administration of a local anesthetic prior to the first incision to block input to the CNS, and postoperative administration of an opioid and an NSAID.

Preoperative concerns

The success of an anesthetic depends on adequate preparation. Dental disease may be a manifestation of systemic disease and may be the cause of some systemic changes.[21] In addition, many patients presented for oral surgical procedures are geriatric, owing to the association between age and the progression of dental disease[22] and the fact that maxillofacial neoplasia is common in older pets. These factors therefore make it imperative to examine the patient carefully and to carry out any ancillary tests to confirm the diagnosis before undertaking the procedure. It is also important to ensure that, if the procedure is elective, the animal should be in the best health possible. For example, if hypothyroidism is diagnosed based on preoperative blood test results, the procedure should be postponed and the patient should be treated with thyroid hormone for sufficient time to counter this metabolic derangement. Hypothyroid animals do not handle stress well, and the outcome can be fatal if they are anesthetized and stressed by a surgical procedure. Old age is not by itself a contraindication to anesthesia but many older patients have concomitant disease, are on other medications, and have poor reserves to combat the effects of the anesthetic drugs, hypothermia, and the stress of the procedure.[23,24] The clinician should be cognizant of the possible interactions between the medications being received and the planned anesthetic drugs. These include direct pharmacological interactions such as the effect of aminoglycosides or organophosphates on the activity of neuromuscular blocking drugs, pharmacokinetic interactions, such as the effects of two highly protein-bound drugs competing for the same binding sites, or pharmacodynamic alterations where one drug increases or decreases the effect of another drug.[25] In modern veterinary practice it is also important to ask the client if the animal is given any herbal therapies or nutritional supplements. Garlic and gingko biloba have been associated with bleeding problems in humans and there may be interactions between these herbal supplements and other drugs, such as NSAIDs, that can affect coagulation.[26] Some of the herbal preparations that have CNS effects (e.g., St. John's wort, valerian) may alter the duration of action of anesthetics. In human medicine it is common for patients not to disclose their use of herbal supplements; it is therefore likely that pet owners do not disclose such use for fear of disapproval by the veterinarian, or because they do not think a "natural remedy" could have any significant impact on the anesthetic.[26] The clinician must therefore ask specific questions and explain why it is important to know of all the substances the animal is receiving. The American Society of Anesthesiologists suggests a two-week withdrawal period for herbal supplements prior to anesthesia in humans.[26]

Before deciding on a course of action with regard to analgesia, it is necessary to consider what the expected level of pain is likely to be for the planned procedure. A young dog that requires dental scaling and polishing is unlikely to need postoperative analgesics. However, if extractions or other oral surgical procedures are planned, administration of analgesics is indicated.

Since one of the approaches to treating pain is to reduce the nociceptive input, techniques that reduce this input before it occurs are often employed. This idea has been termed *preemptive analgesia*. From a biological perspective it makes sense, and the idea has been proven many times in a laboratory setting.[27,28] When it comes to clinical proof, the evidence is less convincing, and meta-analyses of the data available in humans concluded that a preemptive approach has minimal benefit for most treatments used,[29,30] although a recent analysis suggests a benefit of preoperative NSAIDs in human patients with irreversible pulpitis.[31] Drugs commonly used for premedication may provide some preemptive benefit. Phenothiazine tranquillizers, such as acepromazine, reduce the patient's anxiety, and while acepromazine is not an analgesic in its own right, this reduced anxiety may decrease the descending facilitation of pain that can occur when the patient is stressed by the hospital environment. Opioids are analgesics that can reduce the nociceptive input by their effects at both primary and secondary nociceptive neurons.[32] The presence of opioid receptors in peripheral tissues, particularly inflamed tissue, means that opioids may also act to reduce nociceptive input at the tissue level. The cyclohexanones (e.g., ketamine) affect the descending inhibition of pain as well as have a direct effect at the NMDA receptor to prevent wind-up or facilitation. Alpha-2 agonists have analgesic effects in the brain, the spinal cord, and, to some extent, in the periphery (by reducing the release of norepinephrine), and these drugs may also be used preemptively. NSAIDS are used to reduce the release of prostaglandins in peripheral tissues, thus reducing peripheral sensitization of nociceptors, but there is increasing evidence that they also have activity in the CNS as analgesics. In the past, there have been major concerns about the use of NSAIDs in the perioperative period because of concerns about gastric ulceration, renal insufficiency, and platelet dysfunction, but the advent of newer drugs with greater specificity for cyclooxygenase-2 (COX-2) has revolutionized the potential for their use in this context (see later). Finally, local anesthetic techniques applied before the incision will tend to reduce the overall nociceptive input into the CNS and therefore reduce facilitation. *Whether a preemptive approach is chosen or not, the clinician should provide treatment of pain on an ongoing basis after the procedure*.

Induction and maintenance of anesthesia

General anesthetics

The induction technique used should be appropriate for the individual patient. Many practitioners use a single technique (e.g., midazolam/ketamine) and become very proficient with this method. However, despite the safety of some such methods in most patients, there are some where a particular technique may be inappropriate or contraindicated (e.g., the use of ketamine as part of an induction technique was associated with a high mortality rate in cats with hyperthyroidism).[33] If an appropriate technique is not available, or the clinician is unfamiliar with alternative induction agents, consideration should be given to referring the patient to a clinic with the required expertise.

Venous access and intubation

In most cases, venous access with a catheter should be established prior to induction in order to provide fluids and pharmacological support if needed (e.g., antibiotics, positive inotropes, or vasopressors). Patients that are very difficult to handle prior to induction and cannot be catheterized should have a catheter placed as soon as possible after they are anesthetized. The animal should be intubated with a cuffed endotracheal tube and the cuff carefully inflated. Reports of tracheal rupture in cats have been largely associated with dental procedures.[34,35] There has been no specific explanation for this association, but the size of the cuff in relation to the tube may play a role (many of the ruptures in one study were associated with low-pressure/high-volume endotracheal tubes), and the method of inflation may also be a factor. The preferred technique is to connect the tube to the breathing circuit and fill the rebreathing bag with oxygen. A breath is then given manually (to a maximum pressure of 25 cm H_2O), and the anesthetist should listen at the mouth for escaping gas. If there is some gas escaping, a small amount of air is added to the cuff and the procedure repeated until no gas escapes. Once this has been achieved, the vaporizer can be turned on (it is better to turn the vaporizer on after the cuff is sealed, to prevent the escape of inhalant into the room).

In carrying out dental procedures, there is often water and debris in the mouth, which could enter the airway. While an endotracheal tube cuff may prevent gross debris from getting beyond it, it will not prevent all liquid from running down into the distal airway.[36] Hence it is important to position the patient in such a way that fluid does not run into the larynx, to use pharyngeal packing to soak up any liquid, or to use suction in such a way that fluid does not reach this area (see Chapter 7).

If it is known or suspected that fluid has gone down between the endotracheal tube and the larynx, then extubation at the end of the procedure should be performed without deflating the cuff. This should squeeze most of the fluid out of the airway and minimize the risk of aspiration.[37]

If it is necessary to check the patient's occlusion during a procedure and the presence of the endotracheal tube in the mouth will impair this assessment, there are three options:

1. Removal of the tube each time the occlusion needs to be assessed. This is the least desirable option because it leaves the airway unprotected during this time, increases the risk of introducing debris into the trachea and increases the risk of laryngeal trauma if the tube has to be removed and replaced several times.
2. Creation of a pharyngotomy incision and replacement of the endotracheal tube into the larynx via this pharyngeal incision. It is best to carry out this procedure with a wire-reinforced endotracheal tube, which will not kink when curved or bent, in order to minimize the possibility of airway occlusion (see Chapter 61).
3. Placement of a short endotracheal tube with an adapter that fits on the end to lengthen the tube. The endotracheal tube is then positioned such that the proximal end of the tube is in the caudal part of the mouth. The adapter is attached such that the end of this part is just rostral to the incisors. When it is necessary to check the occlusion, the adapter is removed and the mouth can be closed with the short endotracheal tube remaining in the back of the mouth (Fig. 4.1). The animal should be watched carefully when this technique is used because it is possible for the airway to be occluded by the tongue pushing into the end of the tube when the adapter is removed. The adapter can also become disconnected when the mouth is being manipulated, so it must be handled carefully.

• **FIG. 4.1** Endotracheal tube adapter technique allowing the occlusion to be evaluated during the dental procedure. (**A**) Endotracheal tube and adapter connected to the anesthetic machine during the procedure. (**B**) Endotracheal tube disconnected and adapter removed to check the occlusion.

Maintenance of anesthesia

Maintenance of anesthesia with inhalants is the most common method for oral surgical procedures. Currently available inhalants include isoflurane, sevoflurane, and desflurane. The clinical differences between isoflurane, sevoflurane, and desflurane are relatively subtle, with a tendency toward more rapid mask inductions with sevoflurane compared with isoflurane. Desflurane has not reached the veterinary market because it currently requires a vaporizer, which is 4–5 times the cost of the standard units used with the other agents. Nitrous oxide (N_2O)

has been applied widely to reduce the concentration of inhalant used. A concentration of 50–67% is typically used; it is imperative to monitor the concentration of oxygen being delivered to ensure that a hypoxic mixture is not being used; inclusion of pulse oximetry in the monitoring of these patients is also advantageous. The addition of N_2O (compared to an equivalent dose of inhalant alone) has been shown to improve cardiac output in dogs[38] but not in cats.[39] Injectable adjuncts (e.g., opioids) to inhalant anesthetic agents may decrease the nociceptive input during the procedure, and may allow the use of less inhalant. In order to achieve a particular plasma concentration of the drug, it is usually necessary to give a loading dose before the constant rate infusion (CRI). It is generally estimated that it takes five elimination half-lives to reach a steady state, so the need for a loading dose is dependent on this variable. As an example, one report gives the elimination half-life of fentanyl as 46 minutes,[40] so it would take 230 minutes to reach a steady state at any particular infusion rate. Opioids are often used, as they provide good analgesia with minimal effect on the cardiovascular system; these include morphine, hydromorphone, methadone, fentanyl, alfentanil, sufentanil, and remifentanil. These can be given as intermittent boluses (e.g., hydromorphone 0.02–0.05 mg/kg, fentanyl 1–2 μg/kg, sufentanil 0.1–0.2 μg/kg q 15–20 minutes) or as a continuous infusion (fentanyl 10 μg/kg loading dose with 0.1–1 μg/kg/min CRI, sufentanil 1 μg/kg loading dose with 0.01–0.1 μg/kg/min CRI, alfentanil 40 μg/kg loading dose with 1–3 μg/kg/min CRI—halve these doses for cats, remifentanil, no loading dose required, 0.1–1.0 μg/kg/min CRI), with the major disadvantage being the degree of respiratory depression associated with these drugs. If this technique is used, controlled intermittent positive-pressure ventilation should be instituted. These doses of opioids will also cause bradycardia in dogs, which typically responds to administration of atropine (0.02 mg/kg IM) or glycopyrrolate (0.01 mg/kg IM). Lidocaine has also been used as an infusion, and has been reported to result in a 20%–30% reduction in the amount of inhalant needed.[41] This is less likely to produce respiratory depression than the opioids. In the dog, a loading dose of lidocaine at 1–2 mg/kg can be followed by an infusion of 100–120 μg/kg/min. This technique is not useful in the cat, where lidocaine linearly reduces the amount of inhalant needed but also linearly decreases cardiac output.[42,43] Ketamine has also been reported to reduce the amount of inhalant used by 45%–75% in cats and 11%–95% in dogs.[44,45] However, the doses used in these laboratory studies exceed those that would be practical in a clinical setting, and currently a dose of 10–20 μg/kg/min is recommended with a loading dose of 1–2 mg/kg. A combination of an opioid (morphine), lidocaine, and ketamine has become popular, providing a decrease in nociceptive input from multiple mechanisms. Intraoperatively, the lidocaine and ketamine don't seem to add any benefit, in terms of reducing the inhalant dose beyond that provided by the morphine,[46] but continuing this infusion into the postoperative period may provide some advantage. The doses used in the intraoperative study were morphine (3.3 μg/kg/min), lidocaine (50 μg/kg/min), and ketamine (10 μg/kg/min).

Monitoring the patient

Monitoring cardiovascular function

Although the inhalants are not supposed to cause excessive cardiopulmonary depression at regular clinical doses, they may produce profound hypotension in some patients and at higher concentrations.[47-49] Given this disturbing effect on the cardiovascular system, it is important to monitor blood pressure and to be able to treat hypotension if necessary. Several techniques are available to monitor blood pressure; these include Doppler ultrasound and sphygmomanometry, indirect blood pressure measurement via oscillometric or photoacoustic technique, and direct blood pressure measurement using an arterial catheter. The Doppler technique is the least expensive and most robust of these methods, but it requires someone to read the pressures at regular intervals throughout the procedure. Machines that read pressures automatically using an oscillometric or photoacoustic technique are more expensive but can be programmed to read at set time intervals, freeing the clinician to do other things. These devices have not functioned well in small patients, but technological developments are improving their sensitivity, and one unit has been validated for the measurement of blood pressure in cats.[50] The placement of an arterial catheter with the use of direct pressure measurement is generally more accurate than the use of indirect techniques. The placement of such catheters requires practice and is not advocated for the routine measurement of blood pressure in all patients. However, in some patients it is a valuable method because it allows for moment-to-moment analysis of the blood pressure and provides access to the arterial blood for blood gas analysis in the assessment of ventilatory and metabolic acid–base status.

Hypotension is defined as a systolic pressure <90 mmHg or a mean pressure of <70 mmHg. Consideration should be given to treating pressures below these values, and the first step is to assess the depth of anesthesia and turn down the vaporizer as much as possible, given the necessity of providing adequate anesthesia for the procedure. The second step is to increase the rate of fluid administration, usually to the maximum allowed by gravity and the limitations of the fluid administration set and catheter. An initial bolus of 20 mL/kg in dogs and 15 mL/kg in cats of a balanced electrolyte solution is a good starting point to increase the blood volume. However, crystalloid solutions redistribute rapidly and less than 50% of the volume administered will remain in the vascular space.[51] Colloid solutions remain in the vascular space and increase the circulating volume for a longer period than crystalloids. A bolus of hydroxyethyl starch or a polygelatin solution at 5–10 mL/kg would rapidly expand vascular volume and may be more effective than a crystalloid, but it is also much more expensive.[47] If these measures fail to increase the blood pressure into the normal range, then a positive inotrope should be used to try to increase cardiac output and thereby increase perfusion pressure. The most commonly used inotropes in dogs and cats are dopamine, dobutamine, and ephedrine. The first two need to be given as infusions, while the ephedrine is normally given as a bolus. Table 4.1 gives details of how to prepare these solutions and administer them.

TABLE 4.1

Concentrations and Dilutions Used for Infusions of Positive Inotropes in Dogs and Cats

Drug	Concentration	Dilution	Dose	Infusion Rate
Dopamine	40 mg/mL	75 mg (1.825mL)/250mL	5–10 μg/kg/min	1–2 mL/kg/h
Dobutamine	12.5 mg/mL	75 mg (6 mL)/250 mL	2.5–5 μg/kg/min	0.5–1 mL/kg/h
Ephedrine	50 mg/mL	5 mg (0.1 mL)/1 mL	50–100 μg/kg boluses	0.01–0.02 mL/kg per 15–20 minutes

The infusion rates of dopamine and dobutamine should be altered in response to the change in blood pressure. Dopamine, in particular, is known to have highly variable individual pharmacokinetics, so if the initial dose is not working within 5 minutes, it is appropriate to increase it to a higher rate.[52] Arrhythmias or tachycardia probably indicate a relative overdose, in which case the infusions should be decreased or stopped. Repeated doses of ephedrine may be associated with some tachyphylaxis. Another approach to the management of hypotension during inhalant anesthesia is to replace the inhalant with a drug that produces less cardiovascular depression. N_2O is one example and can be given in concentrations of 50%–67%, which should allow a 25%–33% reduction in the amount of inhalant required to maintain an appropriate anesthetic plane. N_2O will often cause a slight increase in blood pressure and may increase respiratory rate in some patients. The concentration of oxygen in the breathing circuit should be monitored to ensure that a hypoxic mixture is not delivered; it is also prudent to monitor arterial hemoglobin saturation with oxygen (SpO_2) using a pulse oximeter.

Central venous pressure

Central venous pressure (CVP) is underutilized as a monitor in veterinary patients despite being simple to measure and providing valuable information about the state of the patient's circulation. A catheter is placed in the jugular vein such that the tip of the catheter is at the entrance to the right atrium. This can then be attached to a water manometer or to a pressure transducer zeroed to the level of the right atrium. CVP is a reflection of vascular volume, intrathoracic pressure, and the ability of the right heart to pump blood forward. In a dog with real or potential congestive heart failure, the CVP will increase if the rate of fluid administration exceeds the ability of the heart to pump the fluids forward. This allows the clinician to adjust the fluid administration to the clinical condition of the animal. Many animals with heart failure receiving diuretics may be relatively dehydrated, but it is very difficult to assess this clinically; CVP measurement allows a more accurate assessment of the patient's ability to tolerate a given fluid volume.

Monitoring respiratory function

Monitoring of respiratory function can be carried out using several methods such as direct observation of the frequency and quality of respiration, breath detection/apnea alerting, respirometry, capnometry, and blood gas analysis. The latter is the gold standard for determining the efficiency of ventilation, while capnometry provides a noninvasive method for the assessment of carbon dioxide elimination. The advantages of capnometry are that it can be applied routinely to all patients and it provides immediate and continuous information. However, there can be significant discrepancies between the end-tidal CO_2 measured by the capnometer and the actual $PaCO_2$, making capnometry a less accurate method than blood gas analysis. Hypoventilation is present when the $PaCO_2$ is above the normal reference point (≈43 mmHg for the dog and 35 mmHg for cats), but it is not common to treat hypoventilation until the animal becomes acidemic and the $PaCO_2$ >65 mmHg. Although positive-pressure ventilation may be performed by hand, a ventilator will provide much more reliable results. A respirometer gives quantitative information on the amount of gas the animal is breathing but does not measure gas exchange, thus making it a less reliable technique than capnometry or blood gas analysis. Breath rate monitors ("apnea alert" monitors) simply indicate whether the animal is breathing and give no quantitative information on the effectiveness of ventilation.

Pulse oximetry is often difficult to use in dental patients because the most reliable site in dogs and cats is the tongue, and the pulse oximeter probe is not a welcome addition to the confined oral space during these procedures. In addition, some pulse oximeters are affected by movement of the site, and will not read if the tongue is being manipulated. Technological improvements in signal processing can account for motion, and newer generations of pulse oximeters may be able to function despite movement of the tongue. If the tongue is not accessible, the probe can be placed on an ear, paw, or tail, but failure rates are much higher in these sites, especially if there is dense pigment of the skin. The measurement of arterial oxygen saturation is also of limited value in patients who are breathing 95–100% oxygen and have no pulmonary pathosis, because the saturation will not change significantly until the PaO_2 decreases to values below 80 mmHg. Since this is a rare event and the technology is prone to artifact, it is likely that the practitioner will be alerted to many false events before a real desaturation occurs. This leads people to believe that the machine is at fault and needs "fixing" when it gives a low reading and not that there is a real problem. The result is that there is a tendency to fiddle with the pulse oximeter, by moving the probe to a different site for example, rather than looking at the patient to see if the number could be real.

Accurate assessment of the anesthetic status of dental patients may also be challenging because the intense work around the head limits the anesthetist's access to the eyes (palpebral response), jaws (muscle tone), and tongue (lingual pulse, blood gas sampling from the lingual vessels). If there is any doubt about the status of the patient, the dental procedure should be discontinued until the animal can be assessed. It is also important to monitor the size of the tongue because it may become swollen due to decreased venous/lymphatic drainage (pharyngeal packs) or due to prolonged manipulation. A swollen tongue may be more prone to injury, and may interfere with ventilation during recovery. Treatment for lingual swelling is symptomatic, using osmotic techniques (sugar applied externally, mannitol IV), diuretics (furosemide IV), and/or corticosteroid injections. These treatments have not been investigated for efficacy in dogs and cats.

Temperature and renal function

Although dental patients do not have a major body cavity exposed, flushing the oral cavity and wetting the head with fluids throughout the procedure increases the likelihood of hypothermia,[53] and it is advisable to make sure the patient is on a heating pad and wrapped up in a blanket or warm air heating blanket. Body temperature should be monitored regularly to ensure that it remains within normal limits.

Many dental patients are old and have some degree of renal insufficiency. It is important that the anesthetic should not cause any further damage to the kidneys. For these patients it is therefore even more crucial to monitor blood pressure, and the addition of CVP monitoring for those patients with concurrent cardiac insufficiency is advisable. The use of drugs to enhance renal perfusion during the procedure is controversial, there being no scientific evidence to suggest either negative or positive effects.

Local anesthetics

Local anesthetics can be used as part of the anesthetic regimen for dental patients. Whenever an invasive procedure is being carried out, the use of a nerve block will decrease the nociceptive input into the CNS, which may have a preemptive analgesic effect. A bigger advantage is that by removing the noxious stimulation, less of the general (inhalant) anesthetic is needed to maintain an adequate anesthetic plane, thus decreasing all the negative effects of these central depressants. Appropriate administration of a long-acting local anesthetic will also delay the need for other analgesic administration[53]; when combined with the need for less general anesthetic, this should allow a more rapid recovery.

Pharmacology

Local anesthetics have a lipophilic and a hydrophilic moiety joined by either an amide or ester linkage. The ester local anesthetics (procaine, tetracaine, benzocaine, and cocaine) are metabolized by cholinesterases in the blood but are rarely used in clinical practice. The amide local anesthetics (which include articaine, bupivacaine, etidocaine, lidocaine, mepivacaine, prilocaine, and ropivacaine) are generally metabolized in the liver and generally have longer half-lives than the esters. Only the most commonly used amide local anesthetic agents will be discussed here; information on the others is summarized in Table 4.2. The particular choice of drug is dependent on desired onset of effect and duration of action. However, the duration of action is dependent on the site of injection—a more vascular site will have a shorter duration of action due to rapid removal of the drug. To counter this, it is common to add a vasoconstrictor to the local anesthetic agent to reduce the rate of absorption and thus prolong the block. Epinephrine is the most commonly used vasoconstrictor; there is no advantage to using concentrations greater than 5 μg/mL (1:200,000), as higher concentrations do not slow the reabsorption of the drug and thus do not prolong the block. This concentration is also less likely to lead to significant absorption and subsequent systemic effects. Even with a consistent site of injection, the duration of action can be highly variable. In four of six dogs, a block with chloroprocaine lasted less than 90 minutes, but in one dog one tooth was desensitized for more than 24 hours.[54] In another study some effect of 0.5% bupivacaine was present in two of eight dogs at 48 hours and when buprenorphine was added to this, the effect persisted in two of eight dogs for 96 hours.[55] The activity of local anesthetics is dependent on the diffusion of the unionized form through the cell membrane, and the amount of the unionized form is pH-dependent. Tissues with a low pH will promote the presence of the ionized molecule, thus decreasing the action of the drug; therefore local anesthetics injected into infected tissue may be ineffective due to the local acidosis.

TABLE 4.2

Local Anesthetics that might be used for Dental Anesthesia

Drug	Trade Names	Concentrations	Onset (minutes)	Duration (hours)
Lidocaine	Xylocaine® Anestacaine™	0.5%–4%	2–5	0.5–3
Bupivacaine	Marcaine® Sensorcaine®	0.25%–0.75%	5–10	2–10
Mepivacaine	Carbocaine®, Isocaine®, Polocaine®, Scandonest™	2%–3% ± levonordefrin	2–4	0.5–3
Prilocaine	Citanest®	4%	2–4	1–2
Etidocaine	Duranest®	1.5% + epinephrine	2–5	2–4
Ropivacaine	Naropin®	0.2%–1%	10–15	2–4
Articaine	Septocaine™	4% + epinephrine	2–5	2–4

Lidocaine is the most commonly used local anesthetic, and it has a rapid onset of action (2–5 minutes) with a duration of 30–120 minutes. In veterinary medicine, it is normally used as a 2% solution, with or without epinephrine. Dental cartridges may contain lidocaine 2% with various concentrations of epinephrine. The maximum dose of lidocaine recommended for dogs and cats is 4 mg/kg, although toxicity is rarely seen at doses <10 mg/kg. Because lidocaine is metabolized in the liver, care should be taken in obese patients or those with hepatic insufficiency. An obese patient will have a smaller hepatic capacity than its weight would suggest. Lidocaine is used as an antiarrhythmic drug, so accidental intravenous administration is not a problem unless a massive dose is given.

Bupivacaine is a longer-acting local anesthetic, often providing a duration of anesthesia 2–3 times as long as lidocaine. It takes about twice as long as lidocaine to reach peak effect. It is supplied in concentrations ranging from 0.25% to 0.75%. Dental cartridges may also contain epinephrine (1:200,000). Bupivacaine is more cardiotoxic than lidocaine, and should not be given at doses exceeding 2 mg/kg. Levobupivacaine (the S-isomer of bupivacaine) has recently been marketed, and is supposed to be less cardiotoxic than the racemic mixture with similar efficacy and duration.[56] Its use has not been evaluated in veterinary dentistry. The information above is a compilation of general facts about these drugs but when applied to dental techniques these "facts" may not apply. In a series of experiments looking at the infraorbital, middle mental, and inferior alveolar blocks in dogs using electrophysiological techniques similar to the ones described by Gross et al.[54] the author has found that:

1. The onset of effect with both lidocaine and bupivacaine is usually within 5 minutes, so there would seem to be little advantage to mixing lidocaine with bupivacaine in order to speed the onset of analgesia.[57] However, where the drug has to diffuse over a distance, onset may be considerably delayed with onset times exceeding 30 minutes in some cases.[58]
2. The duration of effect with lidocaine in the infraorbital canal is 2–3 hours and with bupivacaine it is at least 8 hours and may be >12 hours.[55] An intermediate duration of effect was shown with a mixture of lidocaine and bupivacaine (4–5 hours).[57]

3. The diffusion of local anesthetic into these large nerves may be concentration dependent. This is suggested by block failure in several of the authors' studies,[57,58] and higher concentrations of local anesthetics may prove to be more effective (e.g., 0.75% bupivacaine).
4. The duration of effect is much shorter with the middle mental block than with the infraorbital block (e.g., 1–2 hours with lidocaine and 2–3 hours with bupivacaine).
5. The lateral gingival tissue over the molar teeth for the infraorbital block was largely unaffected and remains sensitive[57] when the local anesthetic was deposited in the canal, but efficacy increased to 70%–80% with injection caudal to the end of the canal using a catheter.[58,59]

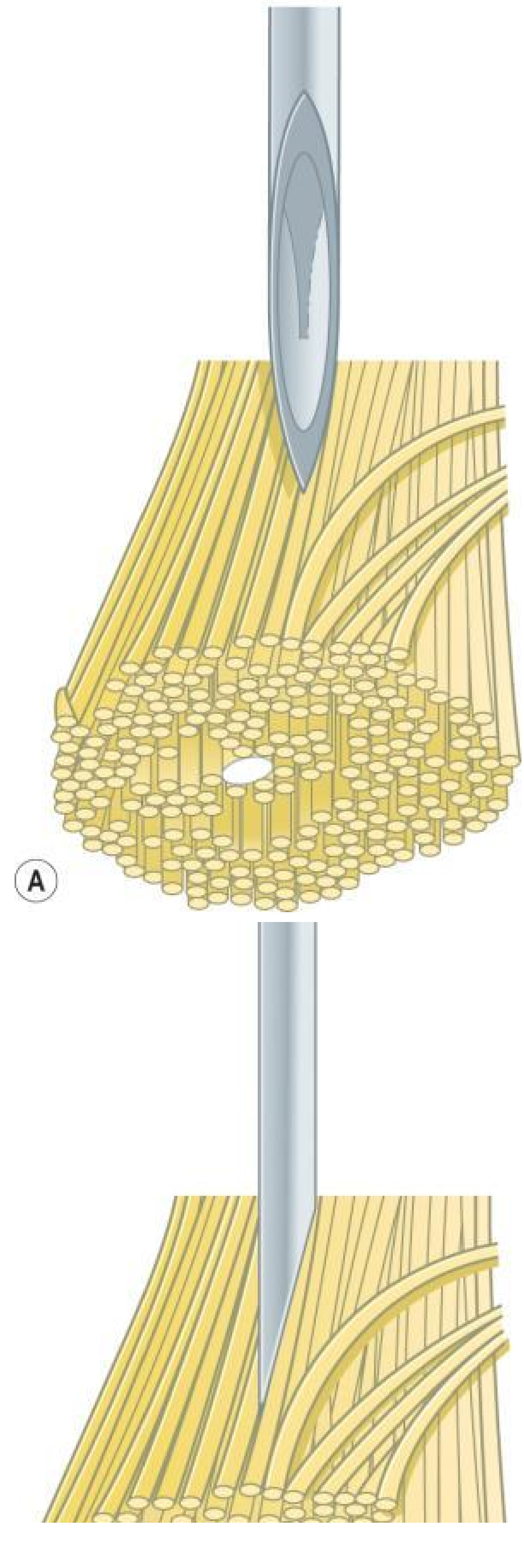
Mepivacaine has a slightly slower onset of action than lidocaine, and a slightly longer duration of action (2–3 hours). It is marginally less toxic than lidocaine. It is supplied as a 2% or 3% solution and is sold with a vasoconstrictor (levonordefrin 1:20,000) in dental cartridges.

Ropivacaine has a relatively rapid time to onset and a duration of action similar to bupivacaine. It is less cardiotoxic than bupivacaine. In humans it has a faster onset than articaine and a longer duration of action when used for maxillary infiltration anesthesia.[60] Concentrations of 0.75%–1% appear to be more effective than 0.5%.[61,62]

Liposome encapsulated bupivacaine or liposomal bupivacaine (Nocita, Exparel) has been licensed for use in infiltration analgesia in dogs. The local anesthetic effect with this product lasts about 72 hours when deposited in tissue, and many studies have shown a benefit from this use of liposomal bupivacaine. In humans the drug appears to decrease pain scores when used following third molar extraction.[63] In humans it is licensed for interscalene brachial plexus block, but there is very little information on whether a nerve block with this product will provide a benefit over regular bupivacaine.[64]

Technique

The greatest concerns when using local anesthetics are hematoma, intravascular injection, and damage to a nerve. The likelihood of damage to a nerve can be minimized by the use of a gentle technique where the needle is not moved very much from side to side once it is in tissue. In conscious human dental patients, penetration of the nerve is accompanied by paresthesia, so the patient can usually warn the clinician that this has occurred. This is not the case if the injection is carried out in the anesthetized patient, so the clinician must be particularly careful about the technique used. Ideally, a fine needle with a short bevel should be used in order to minimize the likelihood of trauma to the nerve. The bevel should be oriented in the same direction as the nerve fibers in order to reduce the chance of cutting through the nerve fibers (Fig. 4.2)[65] but this is of less importance in most dental blocks because the needle is advanced parallel to the nerve. For the infraorbital block, catheters have been used,[54,66] which may decrease the likelihood of damage to the nerves, but there are no definitive data on this potential complication. When a catheter is used it should be long enough to reach the appropriate site and small enough that it will not damage the nerves or blood vessels as it is inserted into the canal. Both 20- and 22-gauge catheters have been used in experimental studies[57,66] and in the clinic. The needle and catheter are inserted into the foramen and then the needle is withdrawn by 3–5 mm. The catheter is advanced as far as is required before the needle is removed, leaving the catheter ready for injection. With this technique it is impossible not to inject a little air, which may be seen on radiographs or a computed tomography (CT) scan.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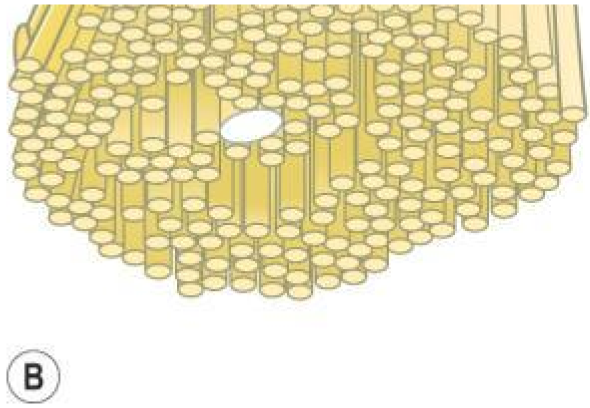


• **FIG. 4.2** Diagram showing the needle (**A**) parallel to and (**B**) perpendicular to the nerve fibers.

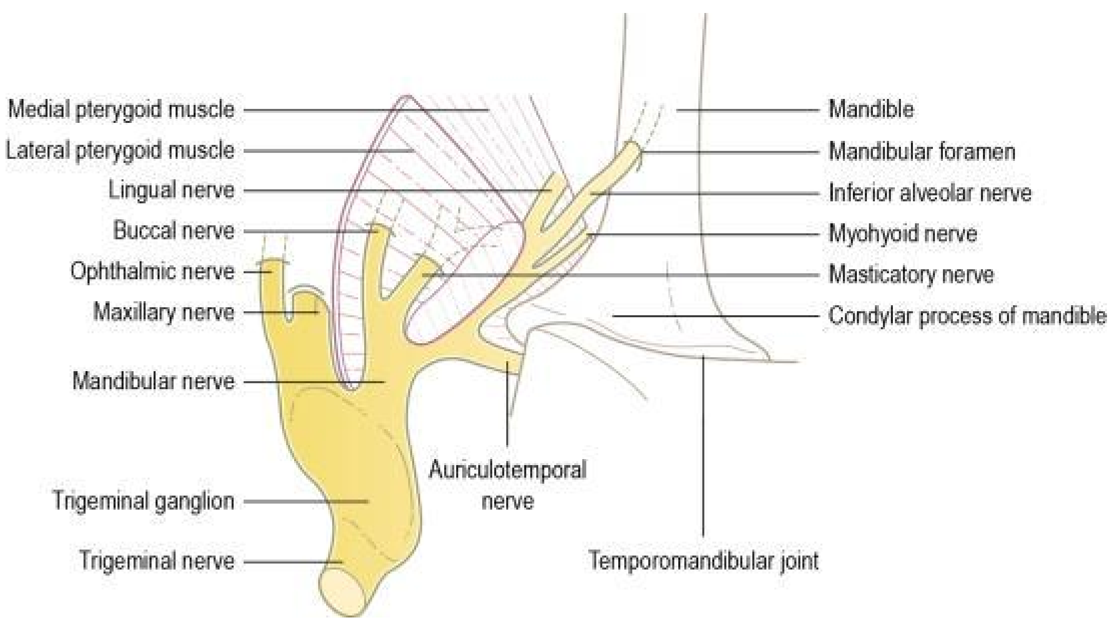
Penetration of a blood vessel is a frequent, minor complication due to the presence of large vessels associated with many of the involved nerves. Again, the use of a fine needle will minimize the risk of hematoma, but the clinician should always be aware of this possibility and apply pressure to the injection site to limit the extent of bleeding, should it occur. Most hematomas resolve without causing any serious sequelae. Of greater concern is the intravascular injection of local anesthetics, because this has been associated with serious, even fatal, outcomes in humans from doses that would not normally be considered dangerous. This may be because inadvertent injection into a facial artery could be associated with retrograde flow into the internal carotid artery, with immediate access to the cerebral circulation. Such reactions have not been described in dogs or cats, but it is wise to be aware of such a possibility. Aspiration, using negative pressure on the syringe, is a simple and effective method for diagnosing vessel penetration and should be carried out before *every* injection of a local anesthetic. Ideally, the needle or catheter should be rotated through at least 90 degrees and a second aspiration carried out, as it is possible to be in a vessel and have a negative aspiration because the bevel of the needle is positioned against the vessel wall. Other minor considerations with the injection of local anesthetics are the possibility of introducing infection into the tissue. This appears to be a rare complication despite the lack of applying disinfectants to the injection site. Allergies have been described to amide local anesthetics in humans, but not in dogs or cats.

The use of regular disposable plastic syringes versus dental syringes is a matter of personal preference. Dental syringes, with their cartridges, were developed to provide a simple, rapid method for loading and delivering the local anesthetic with one hand. The glass cartridges are prefilled with 1.8 mL of local anesthetic solution. They are more expensive than the usual disposable syringes used by most veterinarians and carry few advantages when the patient is under general anesthesia. The size and length of the needle is also a matter of choice. As indicated above, a fine needle is less likely to cause significant trauma, but very fine needles can break off if any stress is placed on them during the injection, so this author usually uses 27-gauge needles.

Neuroanatomy

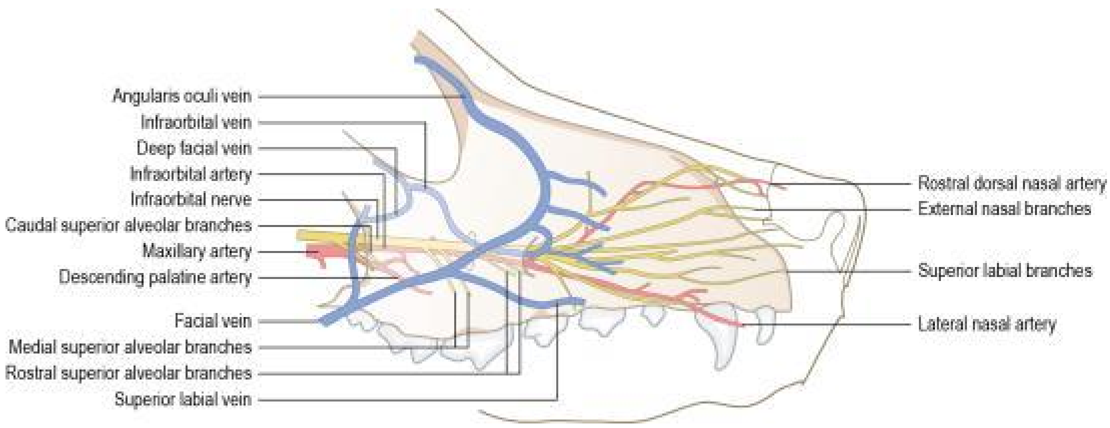
Two branches of the trigeminal nerve provide sensory innervation of the teeth and associated soft tissues: the infraorbital nerve, which is a direct continuation of the maxillary nerve, and the inferior alveolar nerve, which is a branch of the mandibular nerve.

Medial to the condylar process of the mandible, one of the main branches of the mandibular nerve, gives off the auriculotemporal nerve and subsequently divides into three nerves: the lingual nerve (medially), the inferior alveolar nerve, and the mylohyoid nerve (caudally) (Fig. 4.3). The lingual nerve is sensory to the rostral two-thirds of the tongue. In cats it has been shown that the lingual nerve may carry pulpal fibers to the canine tooth.[67] The mylohyoid nerve runs medial to the ramus and body of the mandible and gives off motor branches to the rostral belly of the digastric muscle and the mylohyoid muscle, and two sensory branches to the caudal two-thirds of the intermandibular region. The inferior alveolar nerve enters the mandibular canal through the mandibular foramen, where alveolar sensory branches are given off to the teeth. The local anesthetic agent should be deposited right at the mandibular foramen, in order to affect only the inferior alveolar nerve. If injected further caudally, all three branches may be affected, which may result in numbness of the tongue. Three cutaneous branches exit from the three mental foramina (caudal, middle, and rostral), which are sensory to the lower lip and rostral third of the intermandibular region. It is important to note that the inferior alveolar nerve at the level of the middle mental foramen has already given off its branches supplying the teeth. From there on, the nerve is sensory for the lower lip. Therefore, injecting local anesthetic at the level of the middle mental foramen (sometimes referred to as a "mental block") will not provide anesthesia to the canine or incisor teeth; to achieve anesthesia of these teeth, the needle must be introduced into the middle mental foramen and advanced caudally. This technique is discussed below.



• **FIG. 4.3** Topographical anatomy of the lingual nerve, mandibular nerve, and the mandibular foramen.

The maxillary branch of the trigeminal nerve courses from the foramen rotundum at the base of the skull to the infraorbital foramen. Before it becomes the infraorbital nerve it gives off the pterygopalatine nerve, which then branches into the major palatine nerve that supplies sensory innervation to the hard palate. The maxillary nerve continues as the infraorbital nerve after giving off the caudal nasal nerve. The infraorbital nerve passes over the medial pterygoid muscle before entering the maxillary foramen (Fig. 4.4). Prior to entering the infraorbital canal through the maxillary foramen, the infraorbital nerve gives off a number of caudal superior alveolar branches, which enter the maxillary bone through small alveolar foramina to supply the maxillary molar teeth and possibly the distal root of the fourth premolar tooth. Once within the infraorbital canal, the middle superior alveolar branches are given off on the ventral aspect, mainly to supply the maxillary fourth premolar tooth. Just before the infraorbital nerve exits the infraorbital foramen, the well-defined rostral superior alveolar branches are given off, which enter the alveolar canal to supply the rostral premolar, canine and incisor teeth. The infraorbital nerve, as it exits the infraorbital foramen, has already given off its branches supplying the teeth. From there the nerve is sensory for the upper lip and nose. In order for an infraorbital block to be successful for dental extractions, the drug must reach the respective nerve supply.



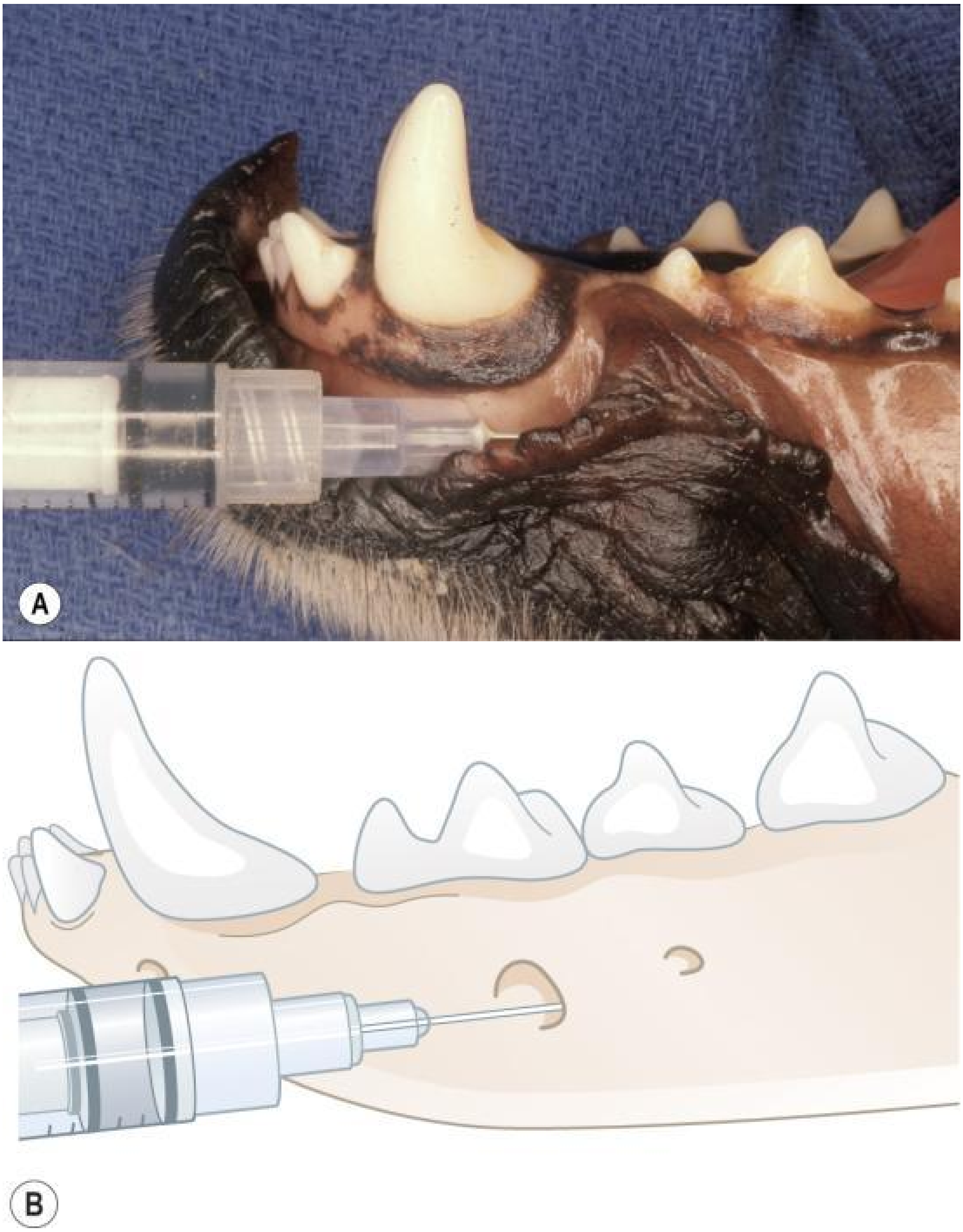
• **FIG. 4.4** Topographical anatomy of the infraorbital nerve and associated blood vessels.

Technique for performing regional intraoral nerve blocks

The volumes of drug mentioned below should be modified by accounting for the maximum dose of the drug that should be used. For example, if a four-quadrant block is planned (using both infraorbital and inferior alveolar nerve blocks) in a 5-kg dog, the total volume used would be 2.12 mL. With bupivacaine (0.5%) this would amount to 10.6 mg, which is slightly over the recommended maximum dose of 2 mg/kg. The volume used for each quadrant could be reduced slightly or the drug diluted up to the recommended volume. In larger animals, the volumes used are unlikely to exceed the maximum recommended dose.

Middle mental nerve block

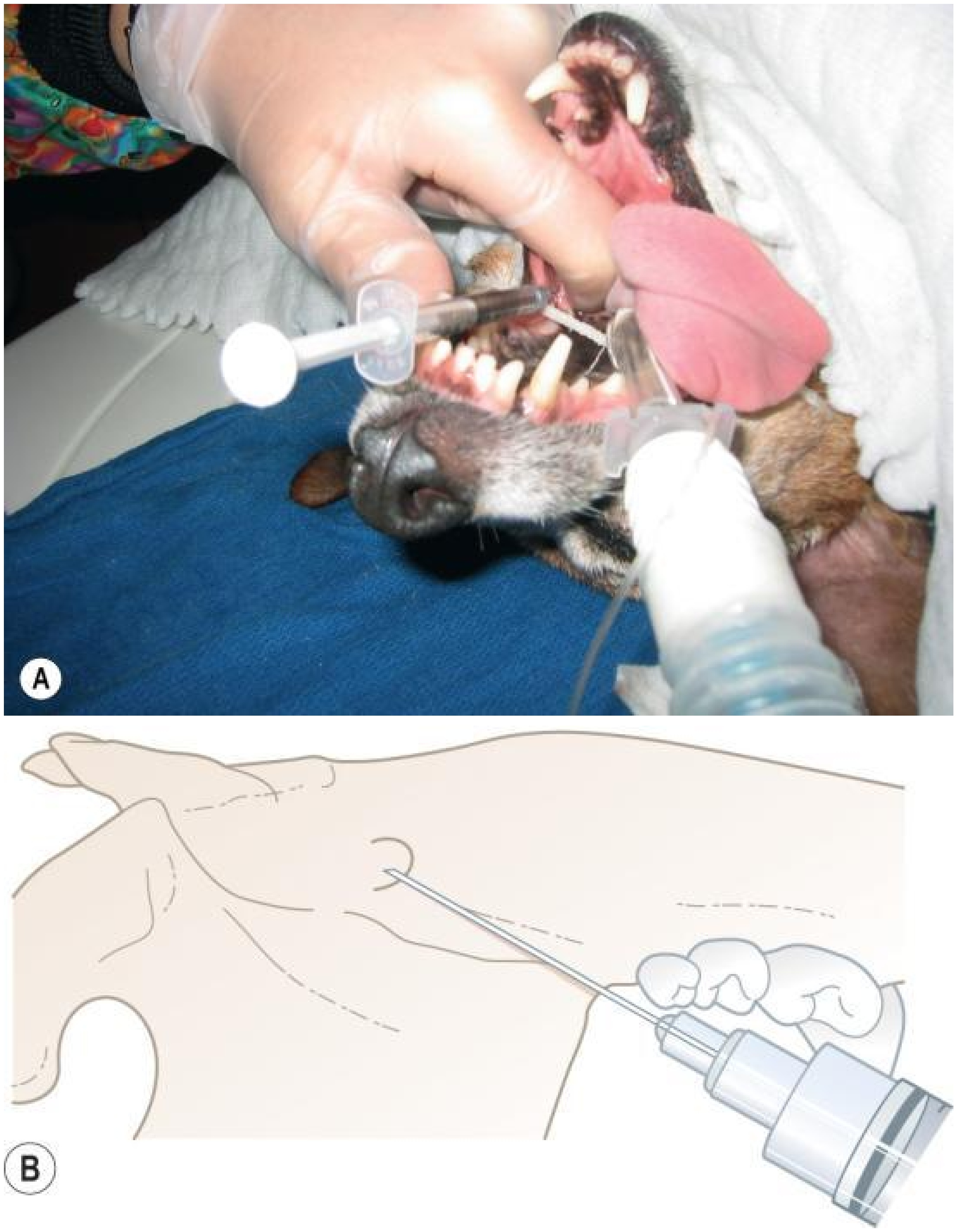
The middle mental nerve is anesthetized for procedures involving the rostral part of the mandible. In one study, anesthesia was established at least as far caudally as the first molar tooth within 10 minutes of injecting 1 mL of 2% chloroprocaine into the middle mental foramen in five out of six dogs.[54] The middle mental foramen is located ventral to the mesial root of the second premolar tooth, medial to the lower lip frenulum (Fig. 4.5). The foramen can be palpated through the frenulum (or the latter can be moved rostrally or caudally) in the ventral third of the vertical distance from the gingival margin to the ventral border of the mandible.[68] Once the landmark has been located, the needle is introduced into the foramen and directed caudally. The canal in which the nerve lies is slightly curved, so the needle may need to be manipulated slightly in order for it to advance far into the canal. In a study trying to define the areas blocked, the authors used a 25-gauge 5/8-inch needle, advanced as far as possible through the middle mental foramen into the mandibular canal, and injected 0.4 mL/m^2 of bupivacaine (about 0.03 mL/kg for the dogs that were used). The authors assessed the block using pressure at the mucocutaneous junction, the mucogingival junction, and by applying a cold stimulus to the teeth. Changes in heart rate and respiratory rate were measured and assessed as evidence of block or no block. The block was only 100% effective to cold stimulation at the third premolar tooth, with <50% efficacy in most sites to the other stimuli.[69] The author has found it possible to advance a 1.5-inch, 27-gauge needle fully into this canal in 20 kg dogs. Injecting this deep into the canal will anesthetize the canine and premolar teeth but does not routinely remove sensitivity from the lateral gingival area over the molar teeth. With the needle in place, the syringe is aspirated before injecting the local anesthetic into the site. Injection should be relatively easy, and there should be no swelling over the site. If it starts to swell as the injection is made, digital pressure should be applied over the area to prevent the drug from escaping into the tissue surrounding the foramen. There are no studies which have defined how much should be injected into this canal. In the study quoted above[54] a volume of 1 mL was used in dogs weighing an average of 20.5 kg, suggesting a dose of about 0.05 mL/kg. It is likely that the volume of the canal is not linearly related to body weight, so this author suggests using a scaled dose of 0.13 $mL/kg^{2/3}$, although even this will not account for the differences in the length of the canal between animals of similar weight but different skull types. For example, it is expected that a lower dose would be used in a 30-kg English bulldog compared with a 30-kg collie. This dose translates to approximately 0.4 mL for a 5-kg dog, 0.8 mL for a 15-kg dog and 1.75 mL for a 50-kg dog. In cats and small dogs it can be difficult to gain access to the canal, so a block of the mental nerve as it egresses from the canal can be achieved by injecting a small volume (0.1–0.2 mL) of anesthetic over the middle mental foramen under the lower lip frenulum if the goal is to anesthetize the soft tissues rostral to this site.



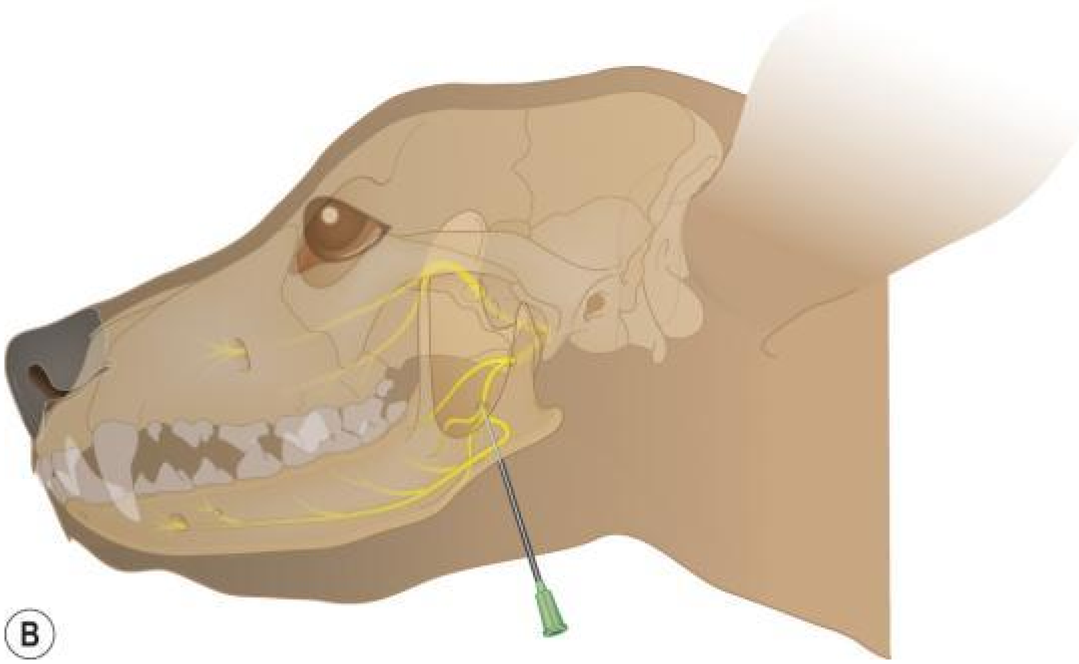
• **FIG. 4.5** Mental nerve block: (**A**) Clinical photograph. (**B**) Line drawing illustrating the topographical anatomy of the middle mental foramen and its relationships with teeth, lower lip frenulum, and mandible.

Inferior alveolar block

Anesthetizing this nerve will remove sensation over the hard and soft tissues of the body of the mandible. There are two approaches to this block: intraoral and extraoral. In both cases the mandible is palpated on the lingual aspect, caudal and ventral to the last molar tooth, in an attempt to locate the mandibular foramen. It is often hard to appreciate the foramen itself, but it is usually possible to feel the neurovascular bundle as a thin, string-like structure as it enters the foramen. With the intraoral technique, the nerve is palpated with one hand while the syringe and needle are held in the other hand (Fig. 4.6). The needle penetrates the mucosa overlying the mandible rostral to the nerve and is advanced caudally until it is next to the nerve. The syringe is aspirated before injecting. In using this technique, the tongue tends to be pushed medially so that there is less likelihood of blocking the lingual nerve at the same time. With the extraoral approach, the landmarks are slightly different in the dog and the cat (Fig. 4.7). In the dog, the deepest part of the depression on the caudal ventral border of the mandible is usually directly over the mandibular foramen if the patient is in dorsal recumbency. In the cat, palpation of the nerve/foramen from inside the mouth becomes the best landmark, since the ventral border of the mandible is relatively flat. Alternatively, the foramen is located approximately halfway on an imaginary line drawn from the last molar tooth to the angle of the mandible in both cats and dogs.[70] The needle should first be advanced perpendicularly on to the most ventral surface of the mandible, then walked medially, advancing towards the foramen as close to the bone as possible. Once in place, the syringe is aspirated before injecting. Volumes of local anesthetic required for this block will be higher than those used for the mental or infraorbital blocks, because the injection is not made into a bony canal. A suggested dose is 0.18m $L/kg^{2/3}$. In the feline study by Gross et al.[71] a volume of 0.25 mL, 2% chloroprocaine, was used with the extraoral approach and there was a failure rate of 50% for blockade of the mandibular first molar tooth but the reasons for these failures were not elucidated. In the author's experiments (unpublished) the extraoral approach without internal palpation failed to provide adequate anesthesia in most subjects.



• **FIG. 4.6** Intraoral technique for administering the inferior alveolar block. (**A**) Clinical photograph. (**B**) Line drawing illustrating the positioning of the needle at the mandibular foramen, with the dog in dorsal recumbency.

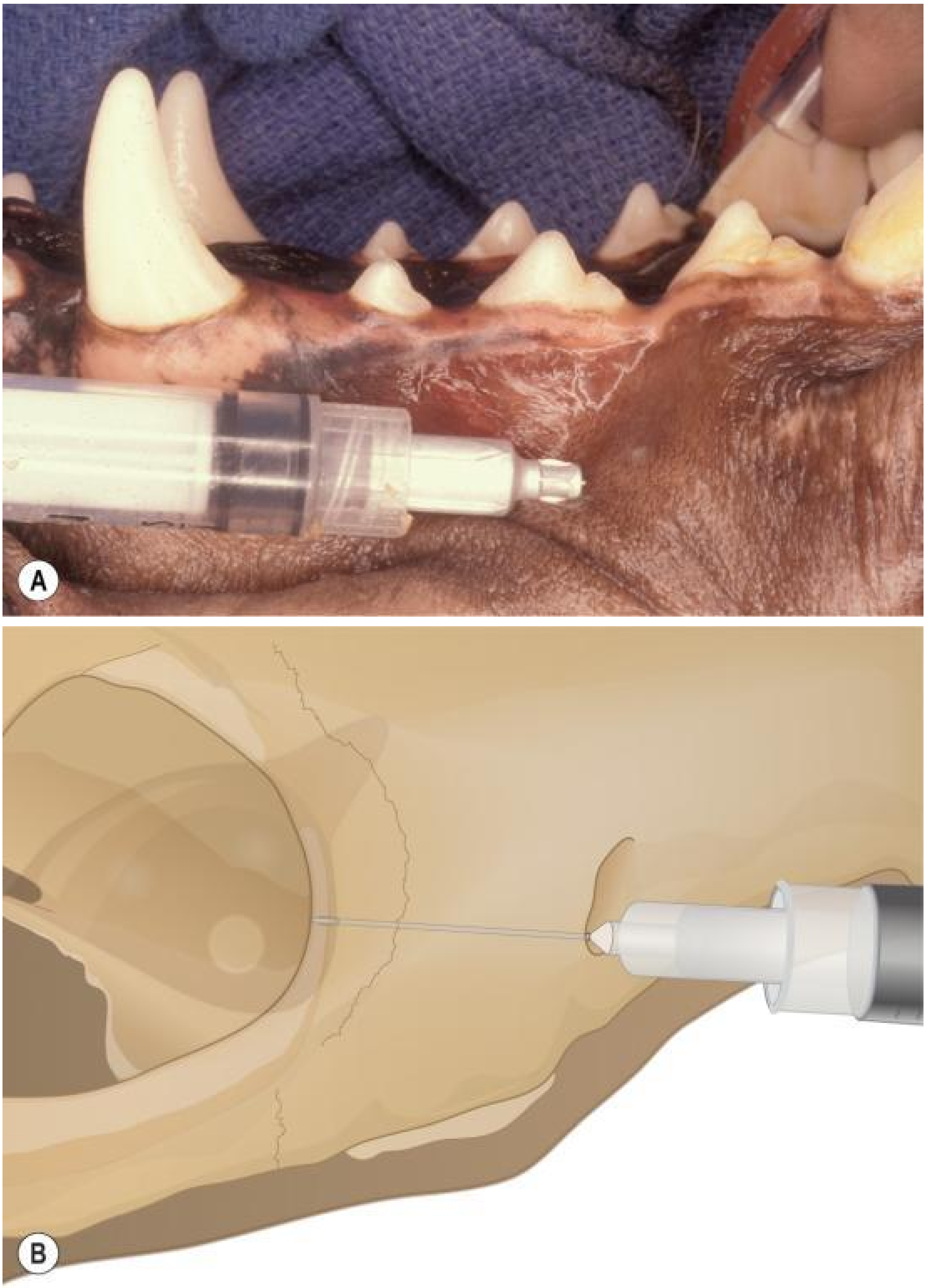


• **FIG. 4.7** Extraoral technique for administering the inferior alveolar block. (**A**) Clinical photograph. (**B**) Line drawing illustrating the positioning of the needle at the mandibular foramen.

There are anecdotal reports of tongue and lip chewing secondary to an inferior alveolar nerve regional block. This has been observed rarely in our practice but may be explained by depositing the local anesthetic too far caudally, thereby blocking the lingual and mylohyoid nerves in addition to the inferior alveolar nerve. In our worst case, a dog with a mandibular fracture that had been stabilized using an intraoral composite splint over the incisor, canine and first premolar teeth, the tongue lacerations were sufficiently damaging that part of the tongue had to be resected. Although the inferior alveolar block may have contributed to this problem, it is not certain that this was the only factor in this injury. Another potential adverse event is systemic toxicity. A cat had profound bradycardia following an inferior alveolar nerve block where the drug was injected near a neoplastic mass. It was suggested that the increased vascularity due to the tumor resulted in rapid uptake of the drug with subsequent toxicity.[72]

Infraorbital block

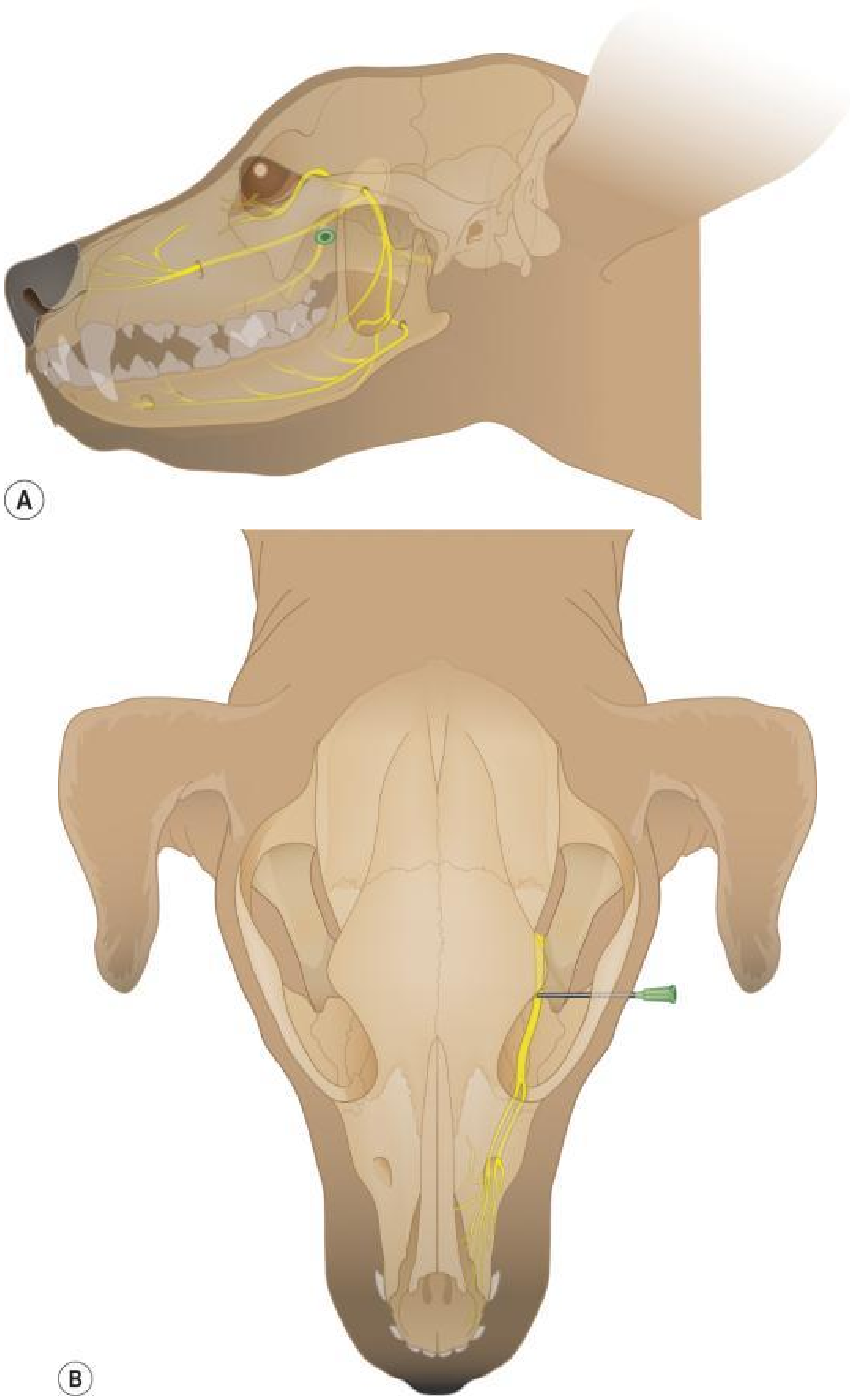
As indicated above, it is necessary to block all of the superior alveolar nerves and the palatine nerve if full desensitization of the maxilla is required for dental extractions, where there is surgical stimulation of the tooth and the gingiva on buccal and lingual surfaces. The approach may be modified if only the incisor or canine teeth are being extracted (cranial superior alveolar nerve). The infraorbital nerve exits from the infraorbital foramen, which is located dorsal to the distal root of the maxillary third premolar tooth. The foramen can easily be palpated from the mucosal surface as a distinct, vertically oriented, elliptical indentation in the maxillary bone. A needle or catheter is introduced into the foramen by advancing it caudally, with the axis of the needle parallel to the bone of the maxilla (Fig. 4.8). The length of needle introduced into the canal can be tailored to the type of block desired. The caudal end of the canal is roughly at a line drawn perpendicularly from the medial canthus of the eye. If the cranial and middle superior alveolar nerves are to be blocked (for the canine and incisor teeth), then advancing the needle to about half to two-thirds the length of the canal will be sufficient. If the caudal superior alveolar nerves are to be blocked (for premolar and molar teeth), then the needle or catheter should be advanced to a point that is perpendicular from the lateral canthus of the eye (i.e., caudal to the caudal end of the infraorbital canal). This will also deposit local anesthetic close to the major palatine nerve to block the palate.[58,66] In cats and brachycephalic dogs the canal may only be a few millimeters in length and if a long needle is used it may end up penetrating the globe of the eye.[73] In dolichocephalic dogs, the canal may be 20–30 mm in length and a longer needle/catheter is required to achieve the desired effect. It is thought that solution injected into the infraorbital canal will spread caudally along the nerve; in the study by Gross et al.[54] the first molar tooth was blocked using this approach, suggesting that the drug can diffuse back to the caudal superior alveolar nerve. However, in our studies the block of the caudal teeth was inconsistent and even with the needle advanced to the end of the canal, the second molar tooth may not be adequately blocked using this technique.[57] If a catheter is used to deposit the drug at the caudal site, it is more likely that the caudal teeth will be blocked.[58] Once the needle/catheter is in place, the plunger of the syringe is pulled back to check the needle placement and, if no blood is aspirated, the solution is injected over 20–30 seconds. Some clinicians will rotate the needle through 90 degrees and reaspirate to ensure that the needle was not just up against the wall of the vessel on the first aspiration (i.e., pulling vessel wall into the end of the catheter rather than blood). Resistance to injection should be absent or minimal. If considerable resistance is detected, the needle may be in the periosteum or the nerve and should be repositioned before continuing. The volume of the solution used for this block is important in order to achieve adequate diffusion. In a cadaver study using dye, it was found that 3 mL of injectate in ~20 kg dogs provided the most consistent staining of the caudal superior alveolar and palatine nerves.[66] In our study we were unable to demonstrate a statistical difference between 1, 2 and 3 mL in ~20 kg dogs.[58] If the drug is injected into the rostral portion of the canal, the volume should be the same as for the mental nerve block—0.11 mL/kg$^{0.67}$; larger volumes may be needed for the caudal block. Some authors advocate using very small doses of the desired anesthetic agent (0.1–0.3 mL), deposited more specifically over the preferred site for the teeth involved by advancing a long needle to that location.[74] This approach may provide a shorter duration of action. The large infraorbital vein and artery also travel through the infraorbital canal, and it is more common to contact one of these vessels when performing this block compared with the others described here.



• **FIG. 4.8** Technique for administering the infraorbital block with the patient in dorsal recumbency. (**A**) Clinical photograph. (**B**) Line drawing illustrating the positioning of the needle in the infraorbital canal.

Maxillary nerve

This nerve is anesthetized when it is difficult to achieve an infraorbital block (e.g., a tumor or abscess over the site) or certainty about anesthetizing the last molar tooth is required. The block can be performed extra- or intraorally and there are at least five different approaches. One landmark for the extraoral block is the notch between the caudal border of the cranial ventral aspect of the zygomatic arch and the maxilla. The needle should be advanced from this point, parallel to the plane of the hard palate and slightly rostrally, until it touches the perpendicular lamina of the palatine bone (Fig. 4.9). It should then be retracted 2–3 mm before aspirating and injecting, as the nerve is located dorsal to the medial pterygoid muscle.[75] Injection into the muscle is unlikely to produce a block.[76] The palatine bone may be very thin at this point, so great care must be taken not to put too much pressure on the bone. Nasal bleeding will be seen if the bone is ruptured. The volume for this block should also be at the higher value used for the extraoral inferior alveolar block (0.18 $mL/kg^{0.67}$). Using this same landmark the needle can be advanced perpendicularly to the axis of the head with the expectation that this will meet the maxillary nerve at a point close to where the pterygopalatine nerve branches from the main nerve.[77] We have seen a greater likelihood of clinically significant hematoma formation with this block compared with the first one, as described using a different approach.[78] It is important with both these blocks that the needle should not be aimed dorsally because this could penetrate the globe with subsequent vision loss.[79] A third direction can be taken from this site and that is to aim caudoventrally to block the maxillary nerve as close to the foramen rotundum as possible. The needle is advanced caudally until it meets the ramus of the mandible. The needle is then "walked" cranially until it passes just rostral to the mandible and is then advanced carefully down to the bone. It is withdrawn 2–4 mm and the injection made at this point. A fourth approach is to start at the ventral orbital rim, passing the needle either through the conjunctiva or through the skin about one-third of the distance from the medial to the lateral canthus of the eye.[76] The globe is gently pushed caudally using the upper eyelid and the needle is advanced a short distance ventrally at right angles to the axis of the hard palate so that it sits directly over the entrance to the infraorbital canal. No clinical studies have been reported for this block but in a cadaver study using new methylene blue injection, 15 out of 17 infraorbital nerves were stained sufficiently to expect a block.[76] In one case the dye was deposited in the oral cavity so it is important not to advance the needle too far. For the intraoral approach the needle is inserted behind the last molar tooth slightly towards the palatine side of the tooth[80] or at the point where the zygomatic arch meets the bone around the last molar.[68] The needle is advanced at a 40 degree[80] or a 90 degree[68] angle with the palate parallel to the teeth and this will intersect with the maxillary nerve before it gives off the caudal superior alveolar branches. Another approach is to use a curved needle to try to place the tip of the needle closer to the entrance to the infraorbital canal. The starting point is the same, but the needle head is rotated as it is being advanced to bring the tip up caudal to the foramen.[78]



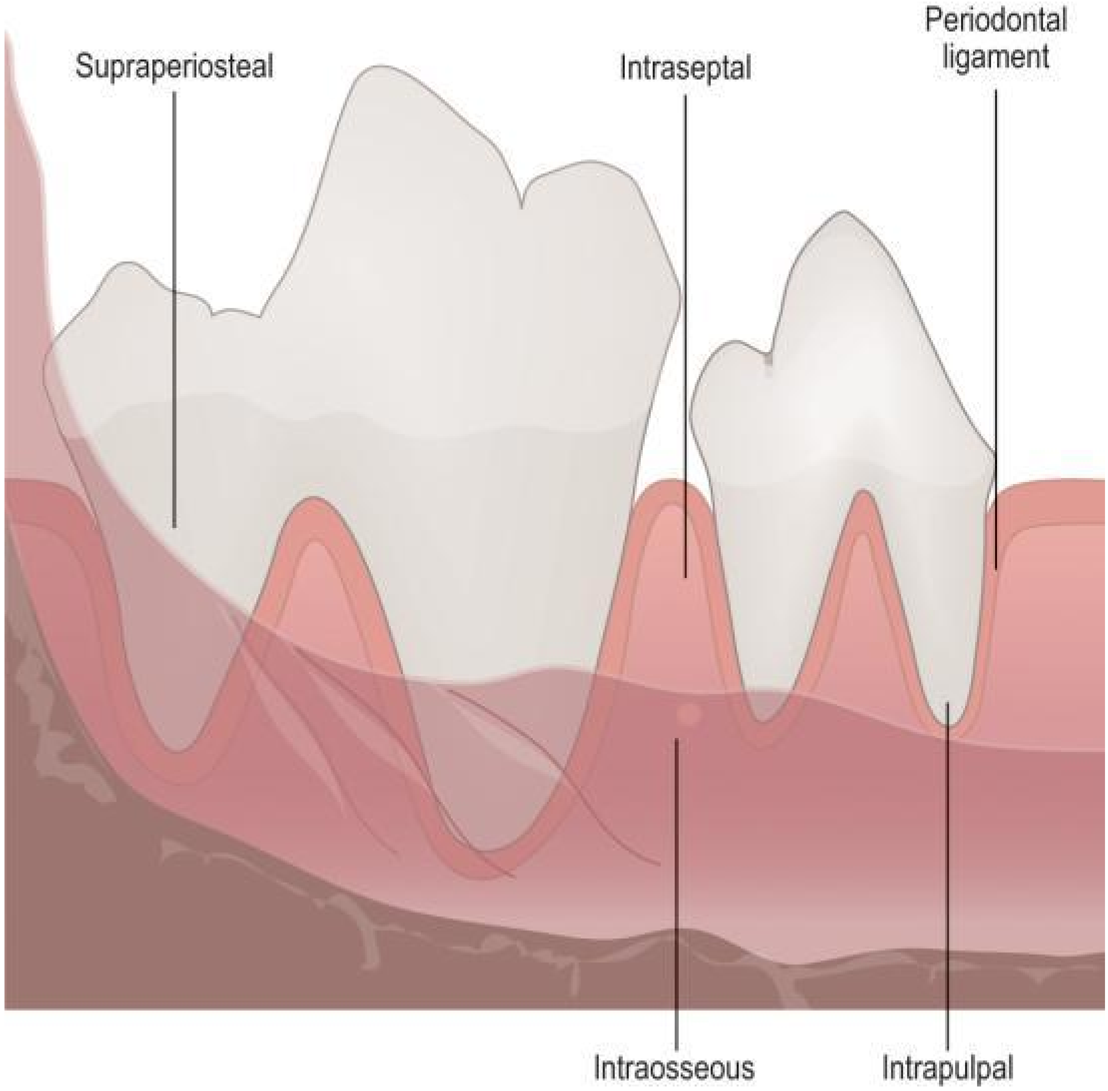
• **FIG. 4.9** (**A, B**) Diagrams showing the needle placement for the lateral extraoral approach to the maxillary nerve block.

Major palatine foramen approach to the maxillary nerve

This approach is sometimes used to anesthetize the maxillary nerve. Because of the thickness of the palatine soft tissue it is difficult to palpate this foramen. It lies at the midpoint between the mesial border of the maxillary first molar tooth and midline. A needle is passed through the major palatine foramen and the injection made into the rostral part of the orbit where the maxillary nerve is located.[52,53] Trauma to the major palatine artery is likely to occur with this approach, in which case severe hemorrhage may result; therefore, this technique is not recommended. Some authors have recommended blocking the palatine nerve as it exits from the canal by depositing 0.1–0.2 mL of 0.5% bupivacaine to provide maximal analgesia of the palate during invasive procedures in this region.[81]

Local infiltration

In humans, five local infiltration techniques are described[82]: supraperiosteal, periodontal ligament, intraseptal, intraosseous, and intrapulpal injection (Fig. 4.10). Supraperiosteal injection involves the injection of the local anesthetic over the periosteum adjacent to the tooth root. This is most effective in young people with less dense bony tissue because it relies on diffusion of the drug through the periosteum and bone to reach the tooth root. It is thought that this will not work for canine and feline mandibles because the bone is too dense.[83] In humans, a volume of 0.6 mL is suggested. Injection into the periodontal ligament is carried out by inserting the needle down the periodontal ligament towards the root of the tooth. It is suggested that the bevel of the needle face the tooth and that an injection of 0.1–0.2 mL of local anesthetic be used. Since this is dense tissue, it can require considerable force to inject the agent into this region (pressures exceeding 17,000 mmHg have been recorded).[84] The high pressures used for such injections may increase osteoclastic activity and cause temporary bone resorption.[85] An injection should be made over each tooth root affected by the procedure. An experiment carried out in young dogs (3–9 months) demonstrated that local anesthetic agent diffused down the paths of least resistance to the vascular channels and intertrabecular spaces, but did not appear to penetrate into the permanent tooth buds.[86] Injection of local anesthetic containing epinephrine (0.1 μg/kg) can be absorbed rapidly by this route, which may result in a decrease in blood pressure.[87] Intraseptal injection requires the placement of the needle in the cortical plate of bone adjacent to the tooth root. The needle is advanced into this plate from the gingiva and 0.3–0.5 mL of local anesthetic deposited. Again, there is a high resistance to injection, and injection of lidocaine with epinephrine can cause up to a 90% reduction in pulpal blood flow using this technique.[88] Intraosseous anesthesia requires the penetration of the bone using a drill and then advancing the needle through this hole into the subcortical bone. This is performed over the tooth of interest using a lateral approach and 0.45–0.6 mL of drug is injected. Intrapulpal injection is only performed when the pulp has been exposed (for example, during an acute complicated crown fracture). A small volume of solution (0.2–0.3 mL) is injected under pressure, directly into the exposed pulp.



• **FIG. 4.10** Supraperiosteal, periodontal ligament, intraseptal, intraosseous (note hole drilled into bone), and intrapulpal injection techniques.

Postoperative care

If an opioid analgesic agent was not employed as a premedicant, it is appropriate to administer an opioid towards the end of any oral surgical procedure. Also, if the procedure has been prolonged it may be appropriate to give a second dose of opioid analgesic to a patient who has received an opioid preoperatively. The intensity of the pain is likely to be at its greatest in the early part of recovery, so an opioid is usually recommended at this stage to cover this period (Table 4.3). Mu agonists, such as morphine, hydromorphone, or methadone, provide more intense analgesia than agonist/antagonists, such as butorphanol, or partial agonists such as buprenorphine; choice of opioid agent should be tailored to the degree of pain expected. The drug should be administered at such a time that it will reach peak effect by the time the animal is recovering. For most opioids, this means 10–15 minutes before the end of the procedure. The exceptions are morphine and buprenorphine, which should be given about 30 minutes before the end of the procedure, because they take longer to reach their maximal effect. Further treatment beyond this initial period will depend on reassessing the animal periodically and administering further analgesics as needed. As indicated later, NSAIDs can be very useful as an adjunct to opioids. Ideally, a technique is chosen which will provide continuous analgesia. This might be a continuous infusion of an opioid, a transdermal administration system or, as indicated earlier, an opioid combined with an NSAID. Since there is a great deal of variation from patient to patient in terms of response to opioids, it is important to reassess the patient frequently to ensure that the amounts and dose intervals used are appropriate. If local anesthetics have been used, the animal should also be monitored for signs of ongoing paralysis or self-mutilation.

TABLE 4.3

Opioid/nonsteroidal Antiinflammatory Doses for Perioperative Pain

Drug	Dose	Species	Route	Duration of Action
Morphine	0.5–1.0 mg/kg	Dog	IM SC	3–4 hours
	0.5 mg/kg loading dose, followed by 0.1–1.0 mg/kg/h	Dog	IM, slow IV IV	Duration of CRI
	0.05–0.1mg/kg	Cat	IM SC	3–4 hours
Meperidine	3–5 mg/kg	Dog/Cat	IM SC	1–2 hours
Methadone	0.1–0.5 mg/kg	Dog/Cat	IM SC	2–4 hours
Hydromorphone	0.05–0.15 mg/kg	Dog Cat	IM IV SC IM SC	2–4 hours
Fentanyl	5 μg/kg + 3–6 μg/kg/h 2–3 μg/kg + 2–3 μg/kg/h	Dog Cat	IV IV	Duration of CRI
Tramadol	1–6 mg/kg	Dog	Oral	6–12 hours
	1–2 mg/kg	Cat	Oral	12 hours
Butorphanol	0.1–0.2 mg/kg	Dog/Cat	IM IV SC	3–4 hours
	0.1–1.0 mg/kg	Dog/Cat	Oral	3–4 hours
Pentazocine	1–3 mg/kg	Dog/Cat	IM IV SC	2–4 hours
Nalbuphine	0.03–0.1 mg/kg	Dog/Cat	IM IV SC	2–4 hours
Buprenorphine	5–30 μg/kg	Dog/Cat	IM IV	8–12 hours
	5–40 μg/kg	Cat	Oral transmucosal	8–12 hours
Buprenorphine 1.8 mg/mL	0.24 mg/kg	Cat	SC only	24 hours
Ketamine	1–2 mg/kg loading dose, followed by 1–2 mg/kg/h	Dog/Cat	IV	Duration of CRI
Carprofen	2–4 mg/kg	Dog/Cat	Oral, IV or SC	6–12 hours
Ketorolac	0.25–0.3 mg/kg	Dog	IM IV	8–12 hours
Ketoprofen	2 mg/kg	Dog/Cat	IM IV SC	12–24 hours
Meloxicam	0.2 mg/kg	Dog/Cat	IV SC	24 hours
Robenacoxib	1 mg/kg oral, 2 mg/kg injectable 2 mg/kg oral and injectable	Cat Dog	Oral, SC, IV Oral, SC, IV	24 hours 24 hours
Grapiprant	2 mg/kg	Dog	Oral	24 hours

CRI, Constant rate infusion

With the new information about NMDA receptors and their role in central facilitation, there have been studies of existing NMDA antagonists such as ketamine and tiletamine for use in postoperative analgesia. Several studies in humans have examined ketamine as an analgesic. At low doses it can provide preemptive analgesia, and provides postoperative analgesia in some cases. There are still concerns over the hallucinogenic effects of the drug, and this has limited its use in humans. It may have a place for providing analgesia in combination with other drugs when they are individually ineffective. Intravenous infusions of ketamine have been used to provide analgesia for dogs and cats[89] (see Table 4.3), usually in combination with opioids.[90] The addition of ketamine to an opioid regimen has provided superior pain relief under some circumstances (e.g., burn patients).[91] Amantadine is a drug that was originally developed as an antiviral compound but it appears to have NMDA antagonist properties as well.[92] One clinical study in dogs with osteoarthritis showed efficacy with this drug at 3–5 mg/kg once daily.[93]

Gabapentin is another drug that is increasingly being used to manage perioperative and postoperative pain, especially chronic and/or neuropathic pain.[94-96] Gabapentin is an analog of gamma-amino butyric acid GABA, but it does not appear to act like GABA in the CNS. It is an antiepileptic drug that can also be used to manage pain in dogs and cats. The mechanism of action is thought to be inhibition of the alpha-2-delta subunits of voltage-gated calcium channels, which are related to signal transduction. Gabapentin is highly bioavailable, minimally protein-bound, and in dogs about 35% of the dose is metabolized in the liver to n-methylgabapentin and the rest is excreted unchanged by the kidneys.[97] The plasma half-life is 3–4 hours in dogs and 2.0–2.5 hours in cats.[98] Although it has been used with success in the perioperative period in humans,[94] veterinary studies with gabapentin have shown minimal benefit.[99,100] Recommended oral doses start at 4–5 mg/kg BID (8–10 mg/kg BID or TID for severely painful patients). Doses in some animals may need to be escalated further (e.g., 60 mg/kg/day) to maintain comfort if the lower doses worked but pain has become more severe. The most common side effect is sedation, and this will usually abate within a few days; the dose can be reduced and then increased again as the animal begins to tolerate the drug. It is important to note that there may be some rebound pain if gabapentin is stopped abruptly, so patients receiving gabapentin should be weaned off the drug with gradually decreasing doses over 2–3 weeks.

Other antiepileptic drugs and antidepressants have also been found to be useful for the management of animal pain. Feline oral pain syndrome (FOPS), observed primarily in Burmese cats, has not responded well to traditional analgesics. The most successful pharmacological treatment of this syndrome has been the use of phenobarbital or diazepam.[101] The tricyclic antidepressants such as imipramine and amitriptyline have been useful adjuncts for neuropathic pain but very little is documented about their use in dogs and cats.[96]

Alpha-2 agonists are very effective analgesics. However, the marked sedation caused by alpha-2 agonists may limit their utility for the management of ongoing pain in small animals.[102] In some cases the degree of sedation provided by these drugs may be desirable. In cats recovering from extensive surgery, it may be difficult to give enough opioid to provide analgesia without promoting further excitation. Dexmedetomidine at 0.5–5 μg/kg will often calm these cats enough for an uneventful recovery. Similarly, dexmedetomidine at low doses (0.5–1.5 μg/kg IV) may provide enough analgesia and sedation to improve the recovery of dogs displaying postoperative excitation or dysphoria.

Use of opioids for the maintenance of analgesia

Oral administration

Opioids are metabolized in the liver, which results in a significant loss of any orally administered opioid agent before it reaches the brain. Hydromorphone has a bioavailability of about 60% when given orally and can be a useful analgesic (0.2–0.6 mg/kg PO q6–8h). Oxycodone is twice as potent as morphine with a 3–6-hour duration in humans. It appears to be effective in dogs at 0.3 mg/kg (increase if required) PO q 6–8h and may be administered with a nonsteroidal antiinflammatory analgesic (personal communication, Kyle. G. Mathews DVM Diplomate ACVS, email, July 2015). Codeine appears to be a poor choice since it has an oral:parenteral ratio of 6.5% in dogs—about one tenth of that in humans.[103] In humans, about 10% of codeine is metabolized to morphine, and it is thought that most of the analgesic action of codeine comes from this conversion. In the dog and cat less than 1.5% is converted to morphine and the intrinsic analgesic action of codeine is thought to be low.[104] However, in a tooth pulp assay in dogs, 4 mg/kg codeine SC was equivalent to about 0.15 mg/kg morphine.[105] In cats a dose of 2 mg/kg orally appeared to have no analgesic effect.[106]

An oral sustained-release morphine has been studied in dogs and found to be absorbed over about 6 hours, with a 20% bioavailability. The terminal elimination half-life for this preparation was 8–10 hours, suggesting that it could be given twice a day at a dose of 2–5 mg/kg. It should be noted, however, that oral administration of the morphine was associated with vomiting in several dogs.[107,108] Methadone has a very low oral bioavailability in dogs, with none detected in plasma after a 1.9 mg/kg oral dose.[109]

Oral butorphanol may be used in dogs at a dose of 0.5-2 mg/kg every 6–8 hours, assuming that the bioavailability is of the order of 20–30%.

Transmucosal administration of buprenorphine has been described in cats and dogs.[110,111] At 20 µg/kg and a standard 0.3 mg/mL formulation, the resulting per-dose volume is very small, and the tasteless solution is well accepted by most patients. The drug is absorbed into the bloodstream through the mucosa, and the reported bioavailability was 32% in cats and 38% in dogs.[110,112] An increase in thermal threshold was present from 30 to 360 minutes after administration in cats.[113] In a study in cats with gingivitis, uptake of buprenorphine administered at 20 µg/kg was rapid, and analgesic effects were noted.[114] Transmucosal administration of 120 µg/kg of a high-concentration formulation of buprenorphine (0.18 mg/mL; Simbadol™, Zoetis Inc., Kalamazoo, MI) increased thermal threshold for about 12 hours with minimal side effects.[115]

Tramadol is being used with increasing frequency in canine and feline patients. This drug and its major metabolite (*O*-desmethyl-tramadol) have mu opioid activity, but also interact with serotonin and alpha-2 adrenergic receptors to produce analgesia. It is a schedule IV drug. The pharmacokinetics of the drug have been examined in the dog[116] and cat.[117,118] It is supplied as a 50-mg tablet and doses range from 1–6 mg/kg BID to QID for the dog and 1–2 mg/kg BID for the cat. Using thermal and mechanical threshold testing, one study was unable to demonstrate significant analgesic effects with tramadol (1 mg/kg) in cats,[119] while a more comprehensive study of thermal thresholds showed a dose-related increase in threshold and duration.[120] With oral administration of 0.5–4 mg/kg, the peak plasma concentration of tramadol occurred between 1 and 3 hours and the peak concentration of *O*-desmethyl-tramadol occurred between 1 and 4 hours. With doses above 2 mg/kg, the increase in thermal threshold lasted for at least 6 hours.[120] In a clinical trial in cats undergoing ovariohysterectomy, tramadol was not much better than placebo but when combined with vedaprofen (an NSAID) it improved pain control.[121] A study in dogs undergoing maxillectomy or mandibulectomy compared tramadol, codeine and ketoprofen and combinations of the opioids with ketoprofen. According to the pain scoring system, the tramadol gave adequate analgesia but there were no significant differences between the treatment groups.[122] In dogs doses of 1 and 4 mg/kg increased thresholds to mechanical but not to thermal stimulation.[123]

Transdermal administration

Highly lipid-soluble drugs such as fentanyl can be absorbed through the skin, resulting in the development of transdermal delivery systems. The original transdermal patches consisted of a drug reservoir containing fentanyl, alcohol gelled together with hydroxyethyl cellulose and an ethylene-vinyl acetate copolymer membrane which controlled the rate of delivery of fentanyl to the skin. The newer patches have the fentanyl in the glue matrix so there is no reservoir. The original patches (Duragesic, Janssen Pharmaceutical Products, Titusville, NJ) are available in five sizes releasing fentanyl at the rate of 12, 25, 50, 75 and 100 μg/h with surface areas of 5.25, 10.5, 21, 31.5 and 42 cm^2, containing 2.1, 4.2, 8.4, 12.6 and 16.8 mg of fentanyl, respectively. The matrix patches (Mylan Pharmaceuticals Inc., Morgantown, WV) are smaller (3.13, 6.25, 12.5, 18.75 and 25 cm^2 for the 12.5, 25, 50, 75 and 100 μg/kg/h patches, respectively).

Fentanyl is a schedule II drug, and as such is subject to Drug Enforcement Agency regulation. Initially licensed for human cancer patients, fentanyl patches are labeled with an explicit warning that they are not for perioperative use. Since there are significant dangers to humans exposed to this drug, it is very important that veterinarians prescribing these patches for outpatients have an understanding of the environment in which the dog or cat is living. A young child could tear a patch off a pet and, by licking the patch or sticking it on himself/herself, be exposed to a fatal dose of fentanyl.[124] Additionally, deaths have been reported following attempts to inject fentanyl extracted from a patch.[125] It is therefore recommended that clients sign an "informed consent" document stating that they understand the risks involved in bringing home a pet with a transdermal fentanyl delivery system in place.

Initial absorption of fentanyl is relatively slow, and it takes approximately 6–24 hours to reach peak plasma concentrations in cats and dogs, respectively.[126-130] Once the peak value is obtained, the plasma concentrations remain fairly constant until removal of the patch (or until the reservoir runs out). In canine studies the patches have been removed at 72 hours, whereas in one feline study the patches were left on until 104 hours, with documented maintenance of plasma concentrations.[131] After removal of the patch, plasma concentrations decay more slowly than after intravenous administration because there is thought to be a reservoir of drug in the dermis which continues to be absorbed. In dogs it takes 2–12 hours for concentrations to decay below a therapeutic value.[126,128,132] Bioavailability of fentanyl delivered transdermally is high (>90%) and there appears to be little degradation of the drug by the skin or its microflora.[133] Several studies have demonstrated the effectiveness of the system as a contribution to postoperative analgesia in humans, but it is not being advocated as a sole approach to pain management.[132,134-136] In humans there have been a number of fatalities caused by heating the patch; this has occurred when a patient had been lying on a heating pad, which increases the rate of absorption of fentanyl from the patch. One study in dogs (22 ± 7 kg) found comparable levels of analgesia after ovariohysterectomy between a fentanyl patch (50 μg/h) and oxymorphone (about 0.05 mg/kg IM as a preanesthetic and at 6, 12, and 18 hours).[137] In two feline studies comparing fentanyl patches versus butorphanol following onychectomy, there appeared to be some benefit in one[127] but minimal difference in the second one.[138] In cats undergoing ovariohysterectomy, the fentanyl patch treatment decreased the cortisol response but did not alter pain scores.[139]

Dose recommendations are:

Cats

12–50 μg/h

Dogs

3–10 kg = 25 μg/h
10–20kg = 50 μg/h
20–30kg = 75 μg/h
>30kg = 100 μg/h

When using the original patches it is extremely important not to cut them. The diffusion of fentanyl into the animal is limited by the membrane on the patch and direct exposure to the gel may cause rapid uptake. If it is necessary to use a smaller area, then the covering on the adhesive surface of the patch could be peeled back and cut so that part of it remains covering the adhesive, and only a half or a quarter of the patch is adhered to the skin.[140] With the matrix patches, it is acceptable to cut them into smaller pieces, as the dose will be proportional to the size of patch applied. A variety of sites have been used for the application of these patches, but all of the above studies were carried out with the patches applied to the lateral thorax or the back of the neck. It is not clear how the uptake of the drug might be affected by sites on the back or the leg although it is likely that these other sites will work well. Before applying these patches, the fur should be clipped and the skin should be gently cleaned with water and allowed to dry. Soap or detergent should not be used, as any residue may prevent the patch from sticking and will increase the likelihood of a skin reaction. If a detergent or alcohol is used these will remove oils from the skin and this may alter the uptake of this lipid-soluble drug. Once the area has been prepared, the patch is applied firmly and held for about 2 minutes. It is preferable to apply a bandage over the patch to decrease the likelihood of the patch being removed by a person or the animal. The bandage should be labeled with the size/dose of the patch and the time and date of application.

An Elizabethan collar (or equivalent) should be considered, as the animal may try to remove the patch, and ingestion of part or all of the patch could lead to a relative overdose.[141]

Subcutaneous administration

Buprenorphine has been produced in a more highly concentrated form (Simbadol, Zoetis Inc., Kalamazoo, MI; 1.8 mg/mL) for subcutaneous use in cats. The idea behind this is that buprenorphine has such high lipid solubility that the drug forms a reservoir in the skin that is slowly released into the circulation, so it is very similar to transdermal administration but with the advantage that its uptake is not affected by having to cross the dermal barrier. A single injection of 0.24 mg/kg provides about 24 hours of analgesia, with onset between 15 minutes and 2 hours.[142] The manufacturer's recommendation is to administer the drug an hour before the procedure and repeat daily for up to 3 days if indicated.

Local administration

Recently, it has been demonstrated that, in addition to CNS opioid receptors, there are opioid receptors on nociceptive nerve terminals which are activated by trauma or inflammation.[111] This knowledge has been put to use for treatment of human dental patients with the demonstration that morphine administered into inflamed tissue surrounding a dental lesion provided better analgesia than when it was not administered or when the same dose of morphine (1 mg) was administered subcutaneously.[143,144] The combination of local anesthetics and opioids has also provided prolonged postoperative analgesia in some instances. In one study, the addition of buprenorphine (7.5 μg/mL) to bupivacaine for intraoral nerve blocks in human patients increased the duration of postoperative analgesia from 8 to 28 hours without apparently prolonging the local anesthetic effect.[145] In Beagle dogs, an effect of bupivacaine was prolonged by the addition of buprenorphine (30 μg added to 0.3 mL 0.5% bupivacaine) with four out of eight and two out of eight dogs still having some block at 48 and 96 hours, respectively.[55] Several opioids have sodium channel blocking properties and buprenorphine is the most potent of these—it has a local anesthetic effect that is about 5 times more potent than bupivacaine[146] so it is not surprising that it can prolong the block with bupivacaine. Other studies using local anesthetics and opioids given submucosally for dental procedures have not shown a difference with the addition of the opioid, but this may be related to dose or site of administration.[147,148]

Nonsteroidal antiinflammatory drugs

These drugs may be useful for perioperative analgesia for more minor procedures but they should be given preoperatively for maximal benefit. The peripheral action has been alluded to earlier and further evidence suggests that there is also a central action.[149,150] The cyclooxygenase enzymes involved in prostaglandin production have now been divided into two isoforms: cyclooxygenase 1 and 2 (COX-1, COX-2). COX-1 is constitutively expressed in many sites including the stomach, kidney and platelets, accounting for the most common side effects of COX-1 inhibitors—gastric ulceration,[151] renal pathosis, and increased bleeding. COX-2 expression is increased during inflammation in many sites in the body but is also constitutively expressed in the kidneys and spinal cord, the latter supporting a role for COX-2 inhibition in acute pain. These discoveries suggest that drugs that have less activity on COX-1, the constitutive form, and more specificity for the induced COX-2 form, would be less likely to cause the side effects described above. An increased tendency to bleed during and after oral surgery is particularly important due to the vascularity of many oral structures. This is particularly relevant with aspirin since it binds irreversibly with platelets.[152] Other COX-1 inhibitors bind reversibly and their effect on platelets decreases as the plasma concentration of the drug decreases, so surgery can be carried out within 24 hours of discontinuing the drug in most cases.

Given this lack of life-threatening side effects, the use of COX-2 inhibitors in perioperative care is becoming more routine. However, the role of COX-2 in renal function suggests that animals that suffer from hypotension during anesthesia may be more at risk of renal pathosis if the drug has been given before the procedure. For this reason, together with the small benefit evident with preoperative administration, many people only administer these drugs postoperatively.

Carprofen

Carprofen is a potent analgesic with antiinflammatory properties but with minimal activity on the cyclooxygenase pathway. Clinical trials on degenerative joint disease[153] and some studies on postoperative analgesia have failed to show any significant incidence of gastric ulceration or gastrointestinal bleeding.[18,154,155] The drug can be given parenterally or orally. The bioavailability after oral administration is >90% and it is highly protein bound. When given intravenously, the terminal elimination half-life is 8–12 hours. After oral administration in tablet form, it reaches its maximum concentration between 1 and 4 hours after administration, with a terminal elimination half-life of 7–9 hours. In postoperative pain studies in dogs, carprofen (4.4 mg/kg IV or SC) provided better and longer-lasting analgesia with less sedation than either papaveretum (morphine mixture) or meperidine.[18,155] One study was able to demonstrate a small preemptive effect of carprofen when given to dogs preoperatively.[154] In cats undergoing ovariohysterectomy given either 4 mg/kg of carprofen or 3.3 mg/kg of meperidine, the carprofen group had similar pain scores to the meperidine group for the first 4 hours after surgery, but lower pain scores 4–24 hours after surgery.[156] In order to avoid the prolonged period to peak effect when given SC, the author usually gives carprofen IV at the end of the procedure.

Ketorolac

Ketorolac has also been shown to be efficacious as a postoperative analgesic in dogs.[157] It is available in a liquid or tablet form and provides analgesic effects for 8–12 hours. The elimination half-life of the drug is 6.5 ± 5 hours.[158] In one study involving shoulder arthrotomy in dogs, ketorolac appeared to be more effective than either butorphanol or oxymorphone.[157] Because gastric ulceration may occur, this drug should be used with caution.

Ketoprofen

When compared with oxymorphone and butorphanol in dogs undergoing orthopedic surgery,[159] ketoprofen was more effective than either of the opioids when used alone, especially in the period 4–12 hours after surgery. Peak plasma concentrations occur very rapidly after an intramuscular injection (7 minutes) and the elimination half-life is 2–3 hours in dogs and cats. Both ketorolac and ketoprofen have been associated with increased intraoperative hemorrhage, so they are often given postoperatively to avoid this problem.

Etodolac

Etodolac is being marketed for use in chronic pain states.[160] Although it is thought to act mainly against COX-2 (COX-1:COX-2 ratios between 1:24 and 1:1000 have been reported) it may still affect platelet function and increase intraoperative bleeding. This drug has an elimination half-life of the order of 8–12 hours and is therefore recommended for once-daily use. Some dogs may become hypoproteinemic with no changes in serum hepatic enzymes.[161] It has been used for perioperative pain management, and provided good analgesia without altering bleeding time or renal function indices.[162]

Meloxicam

Meloxicam is another drug with greater action at COX-2 than COX-1. It has been shown to be effective for both chronic pain[163-165] and for acute surgical pain in dogs.[166-168] It can be given orally or parenterally. Although not Food and Drug Administration (FDA) approved for oral use in cats, the oral form has been shown to be well tolerated by cats[164] and is being used chronically at low doses.[169] It is well absorbed by the oral route and the drug is highly plasma protein-bound.[170] It has a longer elimination half-life (~24 hours) than most comparable drugs and can be given once daily. Its palatability and once-daily administration make it a useful drug for continuing postoperative analgesia.[171]

Robenacoxib

This is a member of the coxib series used in humans (celecoxib, valdecoxib, rofecoxib) which are potent COX-2 inhibitors.[172] Robenacoxib is licensed for use in cats and dogs for 3 days perioperatively and is supplied as palatable yeast tablets (cats), tablets (dogs), or in an injectable form. This drug has a very short plasma half-life (~1 hour) but a much longer half-life in tissue exudate (~40 hours).[173] With oral administration the bioavailability is ~50% with a peak plasma concentration at about 1 hour. Administering the drug with a full meal significantly decreased the bioavailability.[174] It is administered once daily and provides analgesia for about 24 hours.[175,176] In one study it was shown to provide better analgesia than meloxicam in cats undergoing soft tissue surgeries[177] but it was not superior for orthopedic surgery.[178] It has also been used in dogs with similar results to treatment with meloxicam.[179,180]

Deracoxib

This drug comes as a chewable tablet and can be given once daily in dogs, preferably with food. It has a high bioavailability (>90%) and appears to have similar side-effects to other COX-2 inhibitors. It has not been studied in the perioperative period but may be a useful postoperative analgesic in dogs. Reports of gastrointestinal tract perforation in a number of dogs receiving deracoxib illustrates the need to use appropriate doses and not mix these drugs with corticosteroids or other NSAIDs.[181,182]

Firocoxib

This is another member of the coxib series and is a highly selective COX-2 inhibitor. It has been shown to be effective for the treatment of pain associated with osteoarthritis in dogs with fewer side effects than other NSAIDs.[183,184]

Prostaglandin EP4 receptor antagonist (grapiprant)

Prostaglandin E_2 (PGE_2) is one of the major contributors to inflammation in damaged tissue. It activates 4 prostaglandin receptors (EP1-4) and studies in knockout mice have established that the prostaglandin EP4 receptor is the one that contributes most to the cellular processes leading to cytokine production and inflammation.[185] Grapiprant is an EP4 antagonist so it can reverse or mitigate the inflammatory response associated with PGE_2. This drug is currently licensed for use as an analgesic in dogs. There are no trials that have been reported on its use in the perioperative setting to date. Because it acts at the receptor specifically associated with inflammation and does not block other PGE_2 receptors it is unlikely to cause gastric ulceration, platelet dysfunction or renal injury. In trials in dogs it does cause diarrhea and vomiting and may decrease plasma protein and calcium. These changes typically lasted less than two days and were largely self-limiting.[186]

Balanced analgesia

In human and veterinary medicine there is increasing reference to the concept of "balanced analgesia."[187,188] This is similar to the idea of "balanced anesthesia," in which several drugs are used in combination to achieve different aspects of a desirable anesthetic state. In balanced analgesia, combinations of opioids, NSAIDs, NMDA antagonists, and drugs such as gabapentin may have additive or synergistic effects to allow reduced doses of each drug with better pain control for the patient.[121,122] This concept may be expanded to include other mediators of the inflammatory process since many of the latter also sensitize nociceptors.

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CHAPTER 5

Enteral nutritional support

Stanley L. Marks

Therapeutic decision-making

Rationale for enteral nutritional support

Enteral feeding is indicated in patients who cannot ingest adequate amounts of calories but have sufficient gastrointestinal function to allow digestion and absorption of feeding solutions delivered into the gastrointestinal tract via an enteral feeding device. The rationale for prescribing enteral nutrition rather than total parenteral nutrition (TPN) is based on the superior maintenance of intestinal structure and function, safety of administration, and reduced cost of enteral alimentation. Formulating, compounding, administering, and monitoring TPN is both labor-intensive for staff and considerably more expensive for clients than feeding via an enteral feeding tube. In addition, the most important stimulus for mucosal cell proliferation is the direct presence of nutrients in the intestinal lumen. Bowel rest due to starvation or administration of TPN leads to villous atrophy, increased intestinal permeability, and a reduction in intestinal disaccharidase activities.[1,2] Prolonged fasting in the stressed, critically ill patient can lead to intestinal barrier failure and increased permeability to bacteria and endotoxins.

Patient selection for nutritional support

Efforts to assess nutritional status and attempts to decide whether nutritional support is required on the basis of a single biochemical measurement or body weight determination are simplistic and of limited value. Objective methods of assessing nutritional status such as body composition measurement (anthropometry, impedance measurements, dual energy X-ray absorptiometry) are still in their infancy in veterinary medicine, with the result that a subjective global assessment of the patient's nutritional status needs to be performed. This technique is based on easily collected historical information (changes in oral intake, degree of weight loss, and presence of vomiting or diarrhea) and changes found on physical examination (muscle condition score, body condition score and presence of edema or ascites). The reader is directed to the WSAVA Nutritional Assessment Guidelines for further information on nutritional screening of dogs and cats.[3] Although body weight is routinely determined in sick animals, it is important to appreciate its limitations. One cannot equate the appearance of the animal with its state of nourishment because body weight does not differentiate between fat, lean tissue, and extracellular water. Determination of the animal's serum albumin concentration and total lymphocyte count are insensitive determinants of nutritional status because of the large number of disease processes that influence these parameters unrelated to the effects of malnutrition. Nutritional support should be considered for animals demonstrating recent weight loss exceeding 10% of optimal body weight or for those whose oral intake has been or will be interrupted for more than 5 days. Animals with increased nutrient losses from chronic diarrhea or vomiting, wounds, renal disease, or burns should also be considered for nutritional support. Specific maxillofacial indications include long-term mouth closure, less-than-optimal fracture fixation, multiple fractures, and major oral tumor resections. Postoperative inappetence is especially common in cats with maxillofacial trauma or surgical procedures involving the nasal cavity, and these patients greatly benefit from enteral nutritional support.

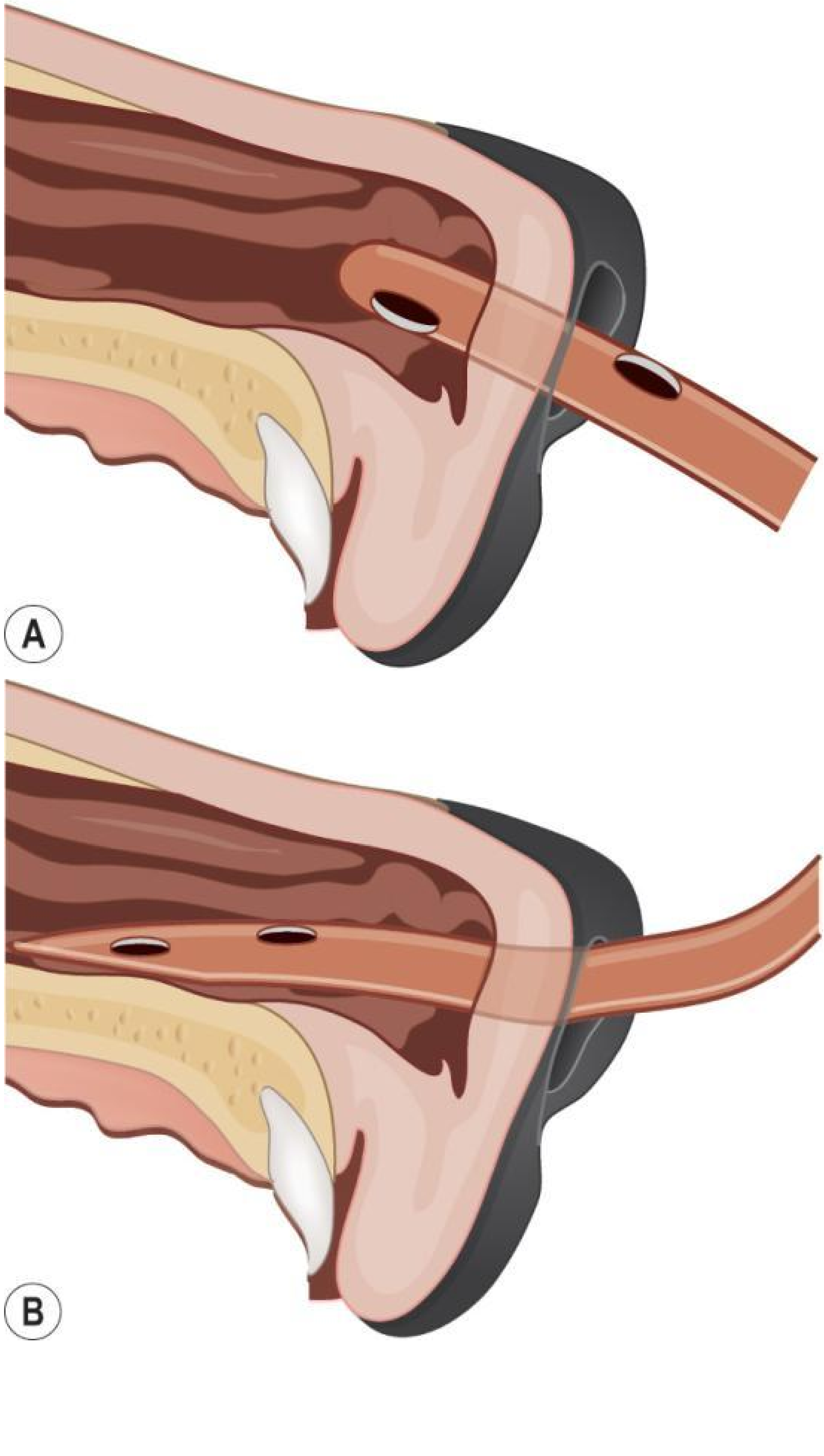
Enteral feeding access devices

Most feeding tubes today are made of polyurethane or silicone. These materials have tended to replace the older polyvinylchloride feeding tubes that tend to stiffen when exposed to digestive juices and are more irritating to patients, necessitating frequent tube replacement. Silicone is softer and more flexible than other tube materials with a greater tendency to stretch and collapse. Polyurethane is stronger than silicone, allowing for a tube of this material to have thinner walls and thus a larger internal diameter, despite the same French size.[4] The flexibility and decreased internal diameter of silicone tubes may lead to clogging or kinking of the tube.[5] Both polyurethane and silicone do not rapidly disintegrate or embrittle in situ, providing a longer "wear." The French (F) unit measures the outer lumen diameter of a tube (one F is equal to 0.33 mm). Tubes that are too flexible may be chilled before placement to increase stiffness.

Nasoesophageal tubes

Nasoesophageal tubes are a simple and efficient choice for the short-term (less than 10 days) nutritional support of most anorectic hospitalized animals that have a normal nasal cavity, pharynx, esophagus, and stomach.[6,7] Nasoesophageal tube feeding is contraindicated in animals that are vomiting, comatose, or lack a gag reflex; it is also contraindicated with maxillary fractures. Polyvinylchloride (Infant Feeding Tube, Argyle Division of Sherwood Medical, St. Louis, MO) or red rubber tubes (Sovereign Feeding Tube, Monoject Division of Sherwood Medical, St. Louis, MO) are the least expensive tubes for dogs and cats, although the polyvinylchloride tubes may harden within 2 weeks of insertion and cause irritation or ulceration of the pharynx or esophagus. Tubes made of polyurethane (MILA International, Inc., Florence, KY) or silicone (Smiths Medical, Minneapolis, MN) are more expensive; however, they are less irritating and more resistant to gastric acid, allowing prolonged usage. An 8–10 F by 109 cm (43 inch) tube (preferably with a guide wire) is suitable for dogs weighing more than 15 kg. A 5–8 F by 56–109 cm (22–43 inch) tube is recommended for dogs weighing less than 15 kg and for cats.

The length of tube to be inserted into the distal esophagus is determined by measuring the distance from the tip of the nose to the eighth or ninth rib. This will help verify the correct placement of the tube in the distal esophagus rather than the stomach and decrease the likelihood of reflux esophagitis.[8] Desensitization of the nasal cavity with four or five drops of 0.5% proparacaine hydrochloride is recommended. The tube tip should be lubricated with a water-soluble lubricant or 5% lidocaine ointment to facilitate passage. The tube is passed by maintaining the animal's head in the normal angle of articulation and gently directing the tip of the tube in a ventromedial direction. The tube should move with minimal resistance through the ventral meatus and nasopharynx and into the esophagus. In dogs, the presence of a small ventral ridge at the proximal end of the nasal passage necessitates directing the tip of the tube dorsally initially to allow passage over the ventral ridge and into the nasal vestibule (Fig. 5.1).[6] Nasoesophageal intubation is more difficult to perform in dogs because of their long, narrow nasal passages and extensive turbinate structures. In the dog, the tube is directed in a ventromedial direction while pushing the external nares dorsally.[9] This maneuver opens the ventral meatus and guides the tube into the oropharynx.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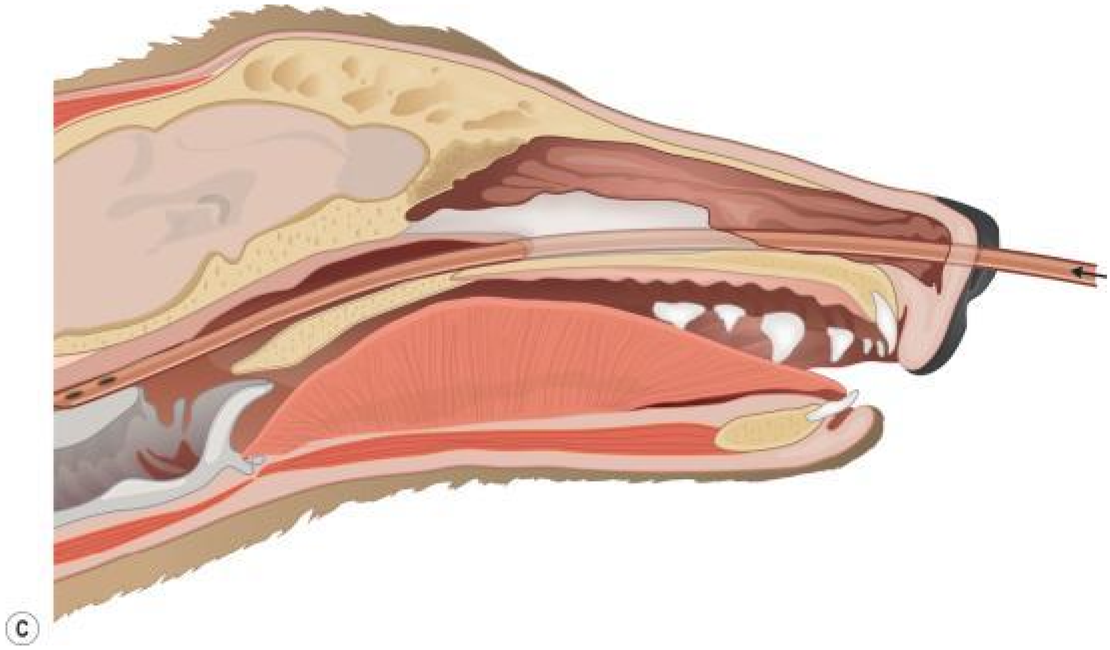
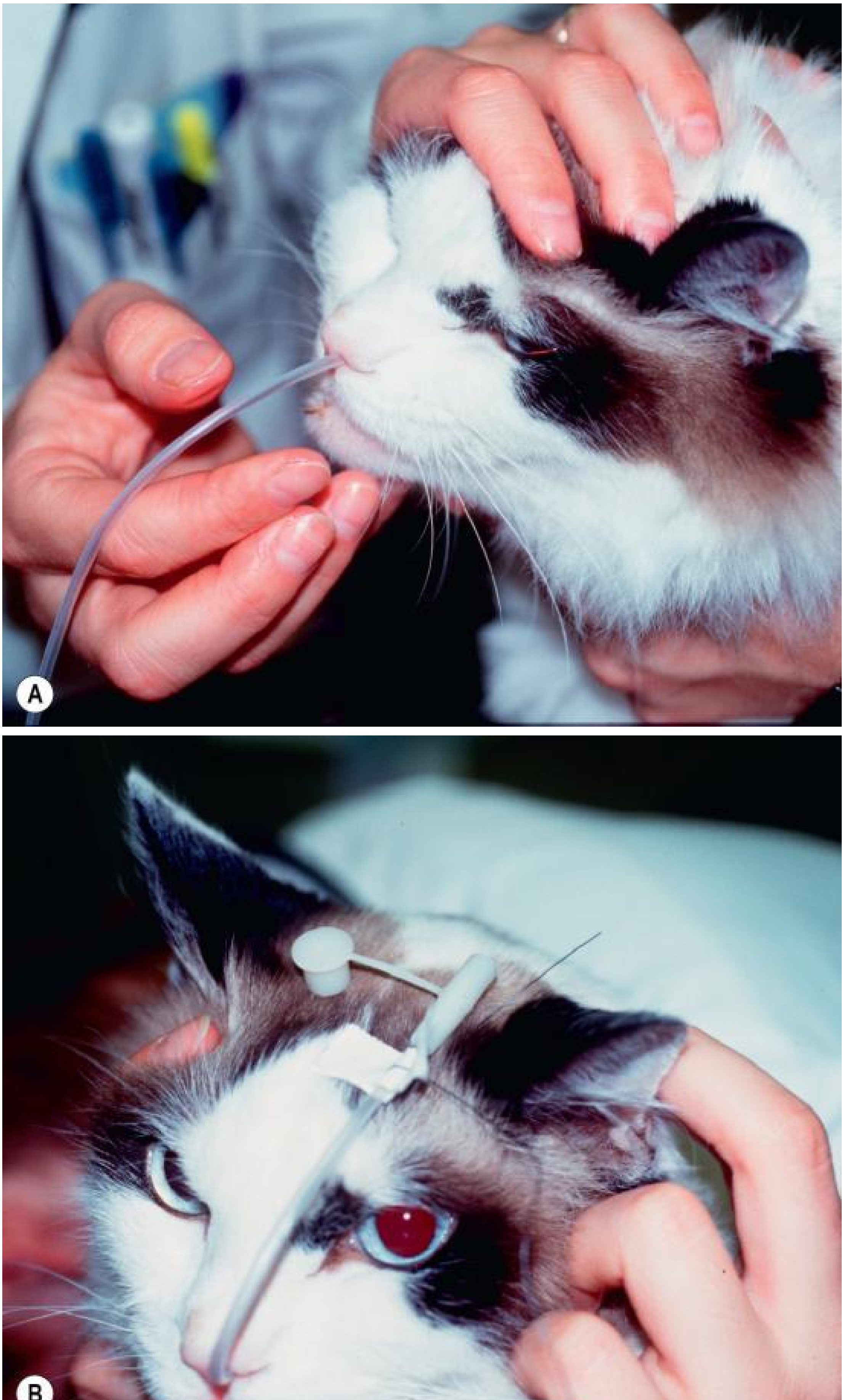


• **FIG. 5.1** Parasagittal section showing stepwise insertion of a nasoesophageal tube through the ventral meatus of a dog: (**A**) The presence of a small ventral ridge at the rostral end of the nasal passage necessitates directing the tip of the tube dorsally to clear the protuberance; (**B**) Once past the protuberance, the tube is aimed medially and ventrally and advanced into the ventral meatus; (**C**) Tube through ventral meatus and nasal pharynx. Source: (From Crowe DT. Clinical use of an indwelling nasogastric tube for enteral nutrition and fluid therapy in the dog and cat. *J Am Anim Hosp Assoc* 1986;22:675–682.)

If the tube is unable to be passed with minimal resistance into the oropharynx, it should be withdrawn and redirected because it could be positioned in the middle meatus with its tip encountering the ethmoid turbinate. Once the tube has been passed to the level of the attached "butterfly" tape, it should be secured as close to the nostril as possible, with either suture material or glue (Superglue, Loctite Corp., Westlake, OH). A second tape tab should be secured to the skin on the dorsal midline between the eyes (Fig. 5.2). An Elizabethan collar is usually required for dogs to prevent inadvertent tube removal; however, most cats do not require such a device. Removal of the tube is facilitated by clipping the hair that is attached to the glue.



• FIG. 5.2 (**A**) The tip of the nasoesophageal tube has been lubricated and passed into the ventral meatus by positioning the animal's head in a normal angle of articulation. (**B**) The nasoesophageal tube can be secured to the skin on the dorsal midline between the eyes with tape "butterflies." A second tape tab should be secured as close to the nostril as possible, with either suture material or glue.

After placement, the tube position is checked by injecting 5–10 mL of air while auscultating the cranial abdomen for borborygmi or by infusing 3–5 mL of sterile saline or water through the tube and observing for a cough response.[6] Confirmation of correct tube placement can also be obtained by obtaining a lateral survey thoracic radiograph and observing the position of the radiopaque tube in the esophagus. The most common complications associated with the use of nasoesophageal tubes include epistaxis, dacrocystitis, rhinitis, tracheal intubation and secondary pneumonia, and vomiting.[6]

A major disadvantage of nasoesophageal feeding tubes is their small diameter, necessitating the use of liquid enteral formulas. Commercially available canned pet foods that are diluted with water will invariably clog the feeding tube. The caloric density of most human and veterinary liquid enteral formulas varies from 1.0 to 1.5 kcal/mL. Diets are fed full strength on continuous (pump infusion) or bolus feeding schedules.

Pharyngostomy tubes

The increasing availability of endoscopic equipment and the advantages of esophagostomy and percutaneous gastrostomy tube placement have resulted in pharyngostomy tubes becoming virtually obsolete. Nevertheless, the introduction of placement modifications has resulted in a dramatic reduction in complications associated with the interference of epiglottic movement and partial laryngeal obstruction.[10,11] The indications for pharyngostomy tube placement are similar to those for nasoesophageal tube placement; however, the procedure requires general anesthesia (see Chapter 61).

Esophagostomy tubes

Esophagostomy feeding tubes are easily inserted, and insertion only requires light general anesthesia with isoflurane or heavy sedation, and intubation with a cuffed endotracheal tube. The technique is minimally invasive and no specialized endoscopic equipment is needed. The patient should be placed in right lateral recumbency, and the left lateral cervical region clipped and aseptically prepared for tube placement.[12-14] A 14- to 20-F red rubber catheter (Cardinal Health, Dublin, OH), silicone catheter (Smiths Medical, Minneapolis, MN), or polyurethane catheter (MILA International, Inc., Florence, KY) should be premeasured from the mid-cervical esophagus to the eighth rib, and marked with a permanent marker to ensure the distal end of the catheter terminates in the distal esophagus.[8] Three basic techniques for placement of a midcervical esophagostomy tube have been described.[12-14]

Technique using curved rochester–carmalt, mixter, or schnidt forceps

Advance the right-angle forceps into the mid-cervical esophagus from the oral cavity. Use the angle of the jaw and the point of the shoulder for landmarks to help ensure that the tip of the forceps can be palpated externally in the mid-cervical region. Push the curved tips of the Rochester–Carmalt forceps laterally at the mid-cervical esophagus, so they can be palpated below the skin. Use a number 11 scalpel blade to make a stab incision through the skin only, exposing the subcutaneous tissue and muscle layers of the esophagus. Be careful to avoid the jugular and maxillofacial veins when selecting the stoma site. Exteriorize the tip of the forceps from the esophageal lumen through the skin incision. Guide the advancing forceps through the esophageal muscle layers and carefully dissect the esophageal mucosa off the tip of the forceps with a scalpel blade. Use the tip of the forceps to grasp the distal end of the feeding tube and draw the tube out of the oral cavity. Secure the distal end of the feeding tube using the forceps to ensure that the tube remains exteriorized while the proximal end of the tube is pulled out of the animal's mouth. Retroflex the proximal tip of the feeding tube and advance it in an aboral direction across the pharynx and down the esophagus, while slowly retracting on the external end of the tube 20–40 mm. A wire guide can be used to facilitate pushing the proximal tip of the feeding tube into the esophagus. The exteriorized portion of the tube will be observed to rotate in a cranial direction as the tube moves down the esophagus, indicating correct placement of the tube in the esophagus. Retention sutures (Chinese finger-trap suture) using 3–0 polypropylene are used to secure the distal end of the tube to the skin. An additional method of securing the tube involves passing a heavy suture on a tapered needle through the skin next to the tube and into the periosteum of the wing of the atlas. Antibiotic ointment and gauze dressing is placed at the incision site, and the tube and entrance site is loosely bandaged with conforming gauze wrap. The correct placement of the tube in the mid- to distal esophagus should be confirmed radiographically. It is important to ensure that the tube does not traverse the lower esophageal sphincter as the tube can cause irritation and predispose the patient to vomiting and gastroesophageal reflux. A recent modification of the abovementioned conventional approach has been described in which the end of the tube does not need to be extracted from the oral cavity and then reinserted.[15] Feeding can be instituted immediately following full recovery of the patient from anesthesia. The tube esophagostomy–skin interface should be examined at least daily during the first week for evidence of infection or leakage of food or saliva. The stoma site can be kept clean with a topical antiseptic solution (1:100 povidone-iodine solution in 0.9% saline). Topical antibiotic creams or ointments are discouraged. The tube can easily be removed once nutritional support is no longer needed by cutting the Chinese finger-trap anchoring suture and pulling the tube. The wound should be allowed to heal by second intention.

Percutaneous feeding tube applicator technique

An alternative tube esophagostomy technique utilizing an Eld percutaneous feeding tube applicator (Jorgensen Laboratories, Loveland, CO) or similar device can be used.[14] The applicator is inserted into the mid-cervical esophagus via the oral cavity. The distal tip is palpated, and an incision is made through the skin and subcutaneous tissue over the tip of the Eld. The trocar is advanced through the esophageal wall and directed through the incision. The distal end of the feeding tube is secured to the eyelet of the trocar with suture material. The Eld device and attached feeding tube are retracted into the esophagus and exteriorized out of the oral cavity. The feeding tube is redirected into the mid-cervical esophagus after inserting a wire stylet into the distal tip of the feeding tube. The tube is secured to the skin as mentioned above.

Percutaneous needle catheter technique

This method incorporates the use of an esophagostomy introduction tube (Van Noort esophagostomy tube set, Smiths Medical, Minneapolis, MN) that is introduced into the mid-cervical esophageal area (Fig. 5.3). The slot in the distal portion of the tube is palpated, and a Peel-away sheath needle (Smiths Medical, Minneapolis, MN) is introduced into the distal portion of the tube. The needle is removed from the sheath, and a 10-F catheter is introduced through the sheath to the distal third of the esophagus. The sheath is peeled away and the esophagostomy introduction tube is carefully removed. The feeding tube is secured as described above. This technique has limitations as the small diameter of the feeding tube (10 F) only allows for the administration of fluids and liquid enteral formulas.

• **FIG. 5.3** An esophagostomy tube set, illustrating the esophagostomy introduction tube, 10-gauge, 50-mm-long needle with Peel-away sheath needle, and a 10-French silicone catheter.

Complications

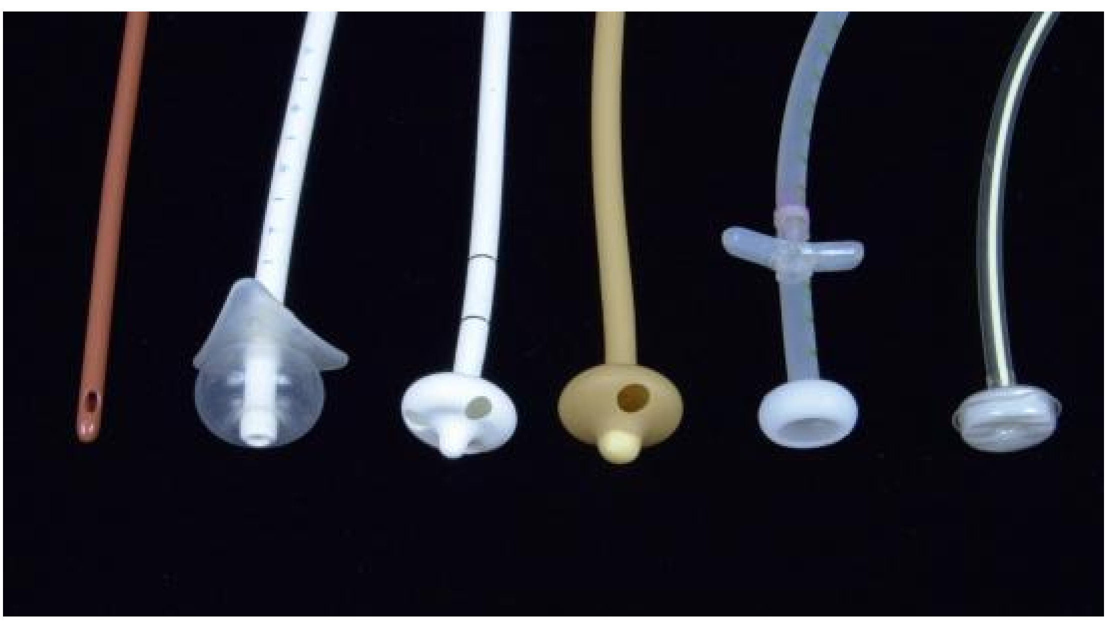
Despite the potential for esophageal scarring and stricture formation, esophageal stricture or a persistent esophagocutaneous fistula has not been observed to develop. The most common minor complication is peristomal inflammation, with peristomal abscessation occurring infrequently.[12-16] Most of the inflammatory reactions are mild and respond to thorough cleansing with topical antibiotics. Other less common complications include regurgitation of the tube into the oral cavity and tube obstruction.[12-16]

Gastrostomy tubes

Gastrostomy tube feeding is indicated for long-term (weeks to months) nutritional support of anorectic or dysphagic animals. Gastrostomy feeding tubes are of comparatively large diameter

(20–24 F), allowing the economic use of blended pet foods and the direct administration of medications. Gastrostomy tube feeding is contraindicated in animals with persistent vomiting, decreased consciousness, or gastrointestinal obstruction. Caution should be exercised in conditions under which the stomach cannot be apposed to the body wall (severe ascites, adhesions, space-occupying lesions).

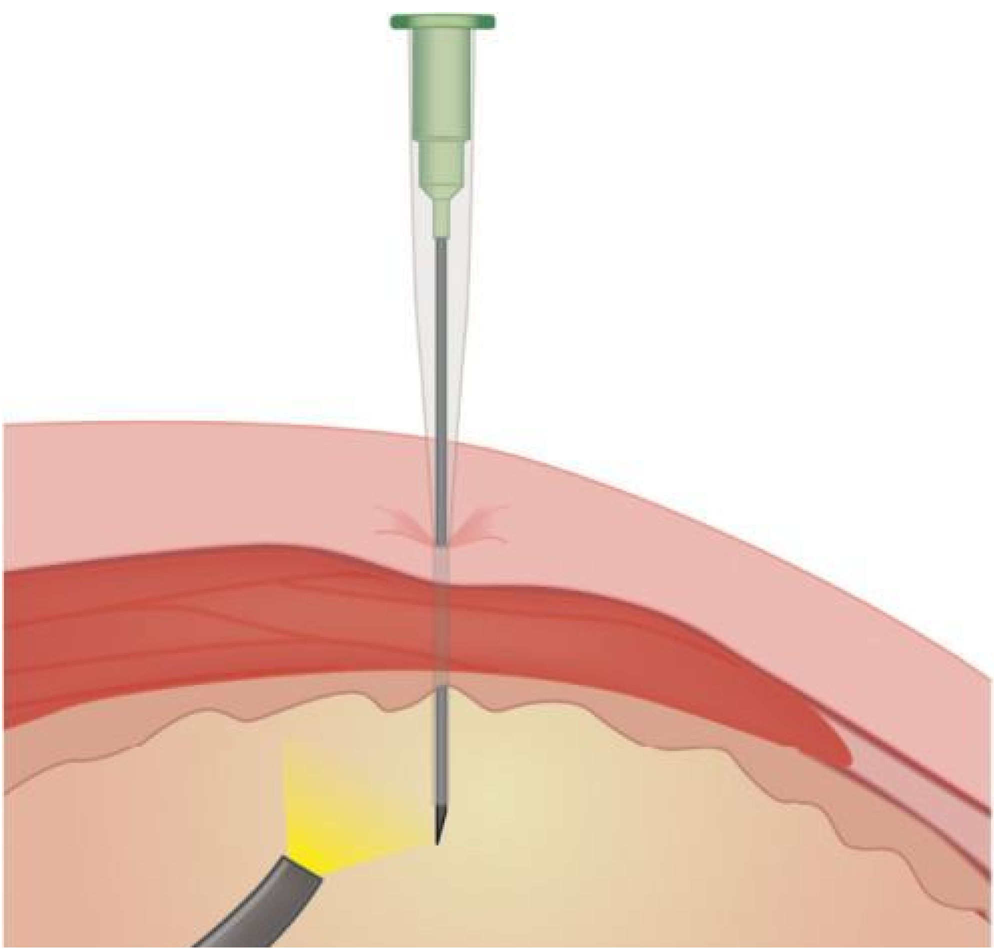
Gastrostomy tubes can be placed percutaneously or during laparotomy. Placement is usually accompanied via a percutaneous endoscopic gastrostomy (PEG) technique or a blind percutaneous gastrostomy (BPG) technique.[17-20] There are a variety of feeding tubes that can be utilized for gastrostomy feeding, including latex (Bard Medical, Covingrton, GA), polyurethane (MILA International, Inc., Florence, KY), and silicon (Smiths Medical, Minneapolis, MN; US Endoscopy, Mentor, OH) tubes with French–Pezzer mushroom, balloon, bumper, or silicone dome tips (Fig. 5.4). One can modify the catheters by cutting off and discarding the flared open end of the catheter and cutting off two 20-mm pieces of tubing (to be used as internal and external flanges) from the same end of the catheter. The end of the catheter opposite the mushroom tip is trimmed to facilitate its introduction into the larger opening of a disposable plastic micropipette. Make a small stab incision through the center of each flange and fit one flange over the cut end of the catheter, sliding it down until it rests against the mushroom tip. The other 20-mm piece of tubing will be used as an external flange that lies against the abdominal wall. It is not recommended to cut the small nipple on the mushroom tip to enhance the flow of food through the tube. Removing the tip of the mushroom compromises the integrity of the mushroom and hinders percutaneous removal of the tube.



• FIG. 5.4 Gastrostomy tubes illustrating the various materials and catheter tips; from left to right: French red rubber catheter, silicone balloon catheter, silicone mushroom catheter, latex mushroom catheter, silicone catheter with dome, polyurethane catheter with bumper.

Percutaneous endoscopic gastrostomy technique

Endoscopic and blind placement of gastrostomy tubes necessitates brief anesthesia. The animal should be placed in right lateral recumbency so that the stomach tube can be placed through the greater curvature of the stomach and the left body wall. Patient preparation for both percutaneous procedures is identical and involves a surgical preparation of the skin caudal to the left costal arch. The endoscope is introduced into the stomach and the stomach is carefully inflated until the abdomen is distended but not drum tight. The left body wall is transilluminated with the endoscope to ensure that the spleen is not positioned between the stomach and body wall. An appropriate site for insertion of the tube is determined by endoscopically monitoring digital palpation of the gastric wall. A small incision is made in the skin with a scalpel blade, and an intravenous catheter (16–18G, 38–51 mm) is stabbed through the body wall into the lumen of the stomach (Fig. 5.5A). The stylet is removed and nylon or polyester suture is threaded through the catheter into the lumen of the stomach. The suture material is grasped with the endoscopic biopsy forceps (Fig. 5.5B), and the endoscope and forceps are carefully withdrawn through the esophagus and out of the mouth. The suture material is secured to the feeding tube and gentle traction is applied to the suture material at its point of exit from the abdominal wall (Fig. 5.5C). The feeding tube is pulled out through the body wall, allowing the mushroom end to draw the stomach wall against the body wall (Fig. 5.5D). The feeding tube is anchored in this position by the external flange placed over the catheter at the skin surface (Fig. 5.5E). The endoscope is then reinserted into the stomach to verify the correct placement of the mushroom against the gastric mucosa. If blanching of the mucosa is observed, less tension should be applied to the tube, otherwise necrosis of the gastric wall may ensue as a result of ischemia. A plastic clamp is placed over the tube and the tube is capped with a Y-port connector. A jacket made from stockinette (3M, Maplewood, MN) is fitted to protect the tube (Fig. 5.5F).

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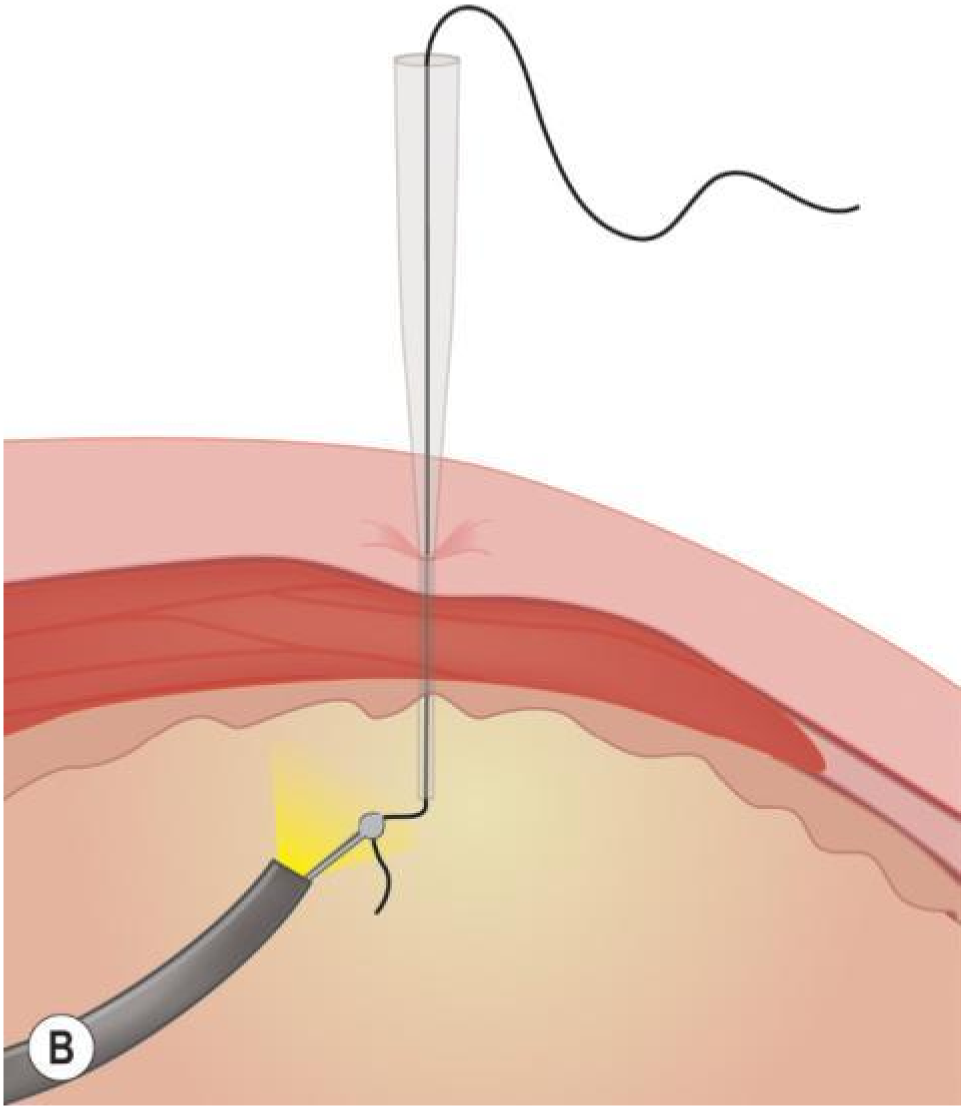
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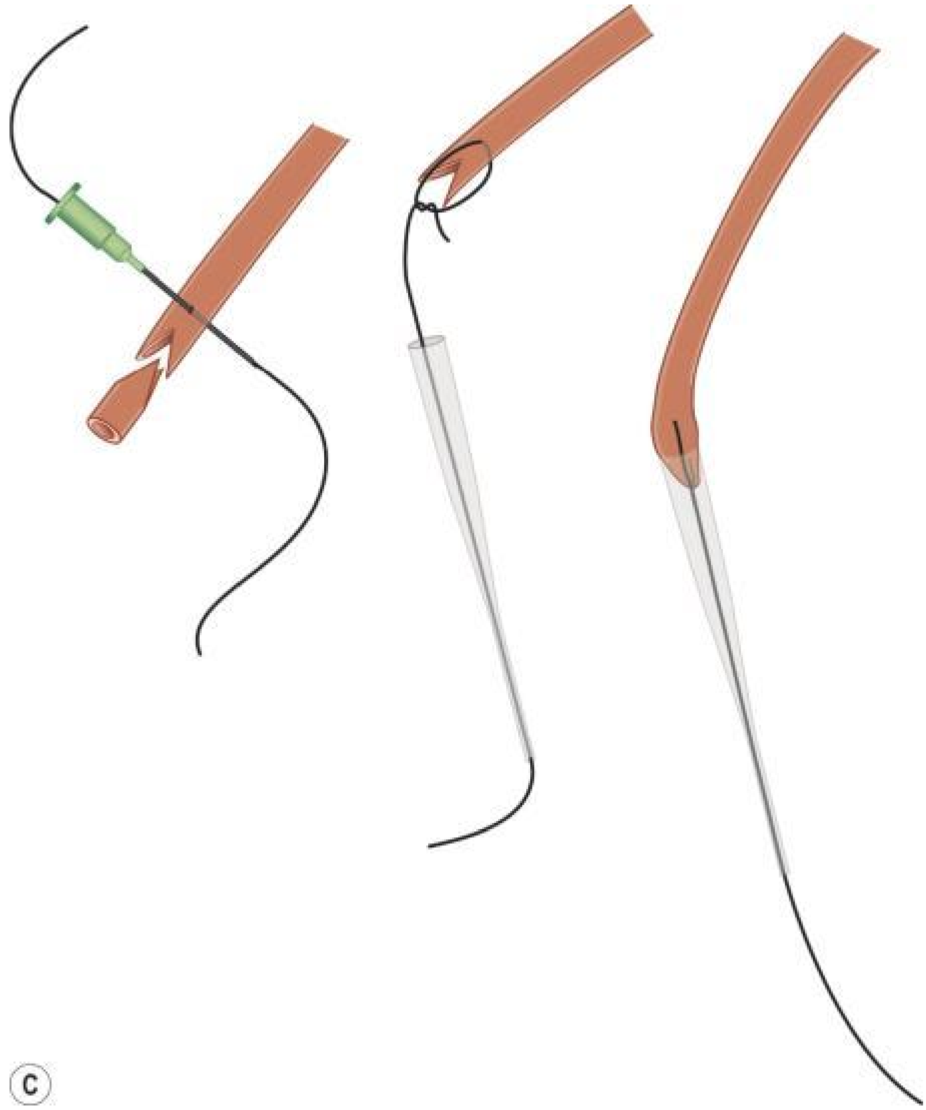

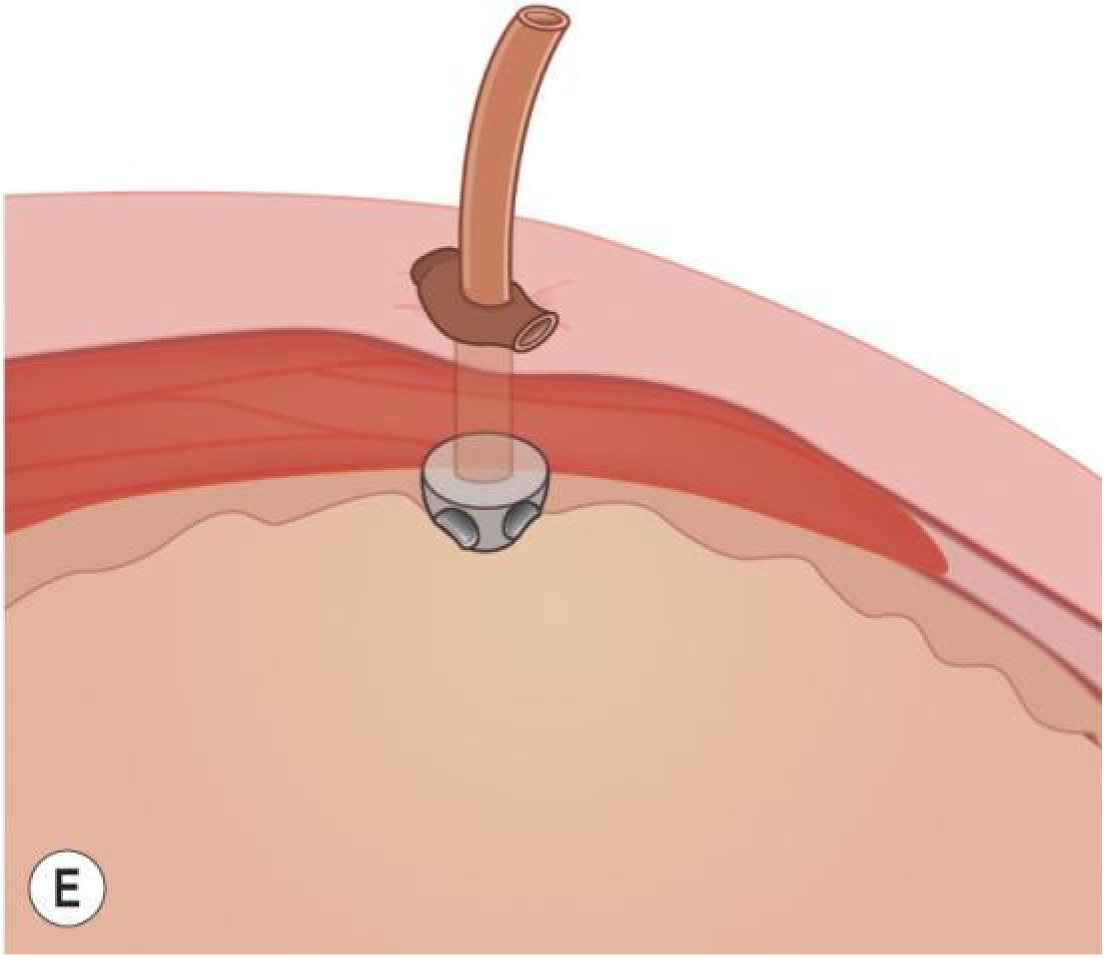
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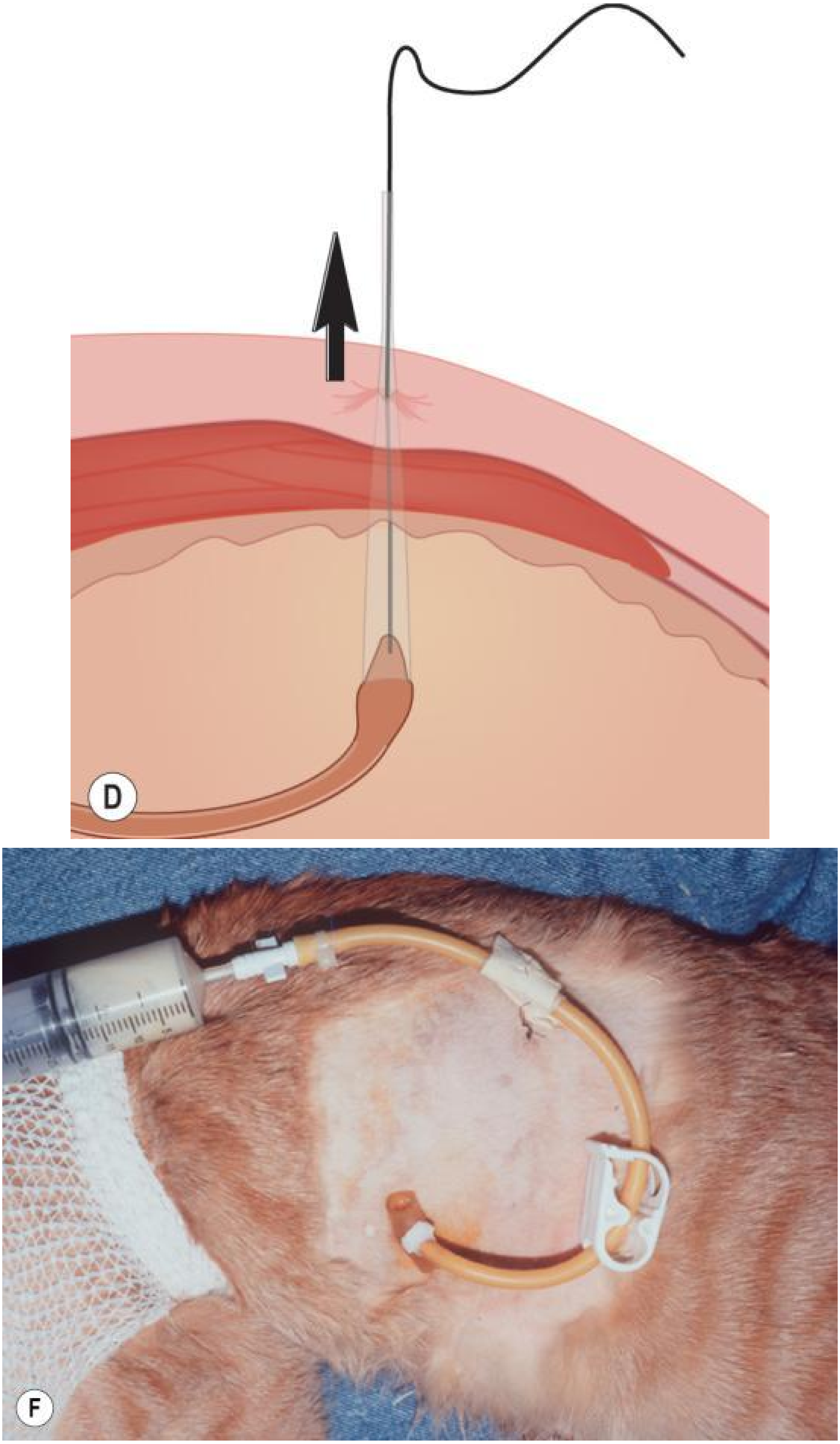


• FIG. 5.5 Percutaneous endoscopic gastrostomy technique. (**A**) With the patient in right lateral recumbency, the endoscope is introduced into the stomach, and the stomach is insufflated with air. The left body wall is transilluminated with the endoscope to ensure that the spleen is not between the stomach and the body wall. A 16- to 18-gauge sheathed catheter is pierced transabdominally into the insufflated stomach lumen. (**B**) The catheter stylet is removed, and nylon suture is advanced through the catheter until it can be grasped with endoscopic retrieval forceps. The nylon suture is pulled out through the mouth as the endoscope is withdrawn. (**C**) The suture material is secured to the feeding tube and water-soluble jelly is applied liberally to the catheter sheath and the mushroom-tip catheter. (**D**) The lubricated catheter is drawn down the esophagus and into the stomach as the assistant applies traction on the suture exiting the abdominal wall. (**E**) The catheter is advanced until the mushroom tip rests gently against the gastric mucosa. Endoscopy should be repeated to confirm the correct position of the mushroom tip. An external flange is fitted down the tube against the skin to prevent the tube from slipping into the stomach. (**F**) Gastrostomy feeding tube in place, with the clamp in the open position. The stockinette jacket is pulled over the gastrostomy tube once feeding is completed.

Complications related to PEG tubes include those associated with placement of the tube (splenic laceration, gastric hemorrhage, and pneumoperitoneum) and delayed complications such as vomiting, aspiration pneumonia, tube extraction, tube migration, and stoma infection.[17,18,21] Splenic laceration can be minimized by insufflating and transilluminating the stomach prior to placement of the needle or catheter into the abdominal wall. In addition, caution should be heeded when placing a PEG tube in an animal receiving corticosteroid treatment. A recent study documented a significantly higher incidence of major severity complications in dogs receiving corticosteroids versus dogs not receiving corticosteroids following placmenent of a PEG tube.[22] The author has recognized a discordant number of large-breed dogs that have had major complications secondary to the stomach falling off the silicone dome at the end of the gastrostomy tube. The stoma appeared normal in all dogs, with the unfortunate consequence that food was introduced intraperitoneally in several dogs. This complication occurred despite the placement of an internal flange between the dome and the gastric mucosa. For this reason, the author recommends that all dogs heavier than 30 kg do not have a PEG procedure, but instead have a gastrostomy tube placed surgically. Alternatively, the gastropexy can be further enhanced with the use of 3-4 T-fasteners (Cope Gastrointestinal Suture Anchor Set, Cook Medical LLC, Bloomington, IN). Minor complications of PEG tube placement include pressure necrosis at the stoma site and cellulitis.[17,18,21]

Blind percutaneous gastrostomy technique

An alternative technique for non-endoscopic and non-surgical gastrostomy tube placement has been described.[19,20] The gastrostomy tube placement device can be prepared with a length of vinyl or stainless steel tubing (diameter 12–25 mm) purchased from a hardware store, or an Eld gastrostomy tube applicator (Jorgensen Laboratories, Loveland, CO) or Cook gastrostomy tube introduction set (Smiths Medical, Minneapolis, MN) can be used. The Eld gastrostomy tube applicator is the only device that utilizes an internal trocar, whereas the Cook gastrostomy tube introduction set contains a wire that is threaded through an introduction needle. The distal tip of a stainless steel tube can be flared and deflected 45 degrees to the long axis of the tube to help displace the lateral body wall. The lubricated tube is passed through the mouth and into the stomach. The tube is advanced until the end of the tube displaces the stomach and lateral abdominal wall. Positioning the animal with its head over the edge of the table and lowering the proximal end of the tube will facilitate identifying the tube tip through the body wall. For the Cook gastrostomy introduction set or similarly prepared device, a percutaneous needle is introduced into the lumen of the introduction tube while the assistant firmly holds the distal tip of the tube between two fingers. A skin nick is made over the end of the tube and a 14-G over-the-needle catheter advanced into the lumen of the tube. Proper positioning of the catheter is confirmed by moving the hub from side to side and feeling the catheter tip strike the inside of the tube. A guide wire prepared from a banjo string is attached to suture material that is 60 cm longer than the stomach tube. The guide wire is threaded through the catheter, into the tube, and out the mouth of the patient. The attached suture is pulled through the tube and cut from the wire at the mouth. The tube and catheter are removed, and the suture is attached to a gastrostomy tube that is secured in an identical fashion to the PEG tube procedure.

The reported complication rate for BPG is similar to that of PEG; however, the risk of penetrating the spleen, stomach, or omentum is greater when the stomach is not insufflated with air prior to positioning the tube against the lateral abdominal wall.[23,24] Contraindications to using the blind technique include severe obesity precluding accurate palpation of the tube against the abdominal wall and esophageal disease. Surgical placement of gastrostomy tubes should be reserved for these patients.

Surgical tube gastrostomy technique

Surgical placement of gastrostomy tubes has been superseded by the percutaneous techniques because of the ease and speed of placement, lower cost, and decreased patient morbidity associated with the non-surgical techniques. A surgical approach is indicated in obese patients, patients with esophageal obstruction, or in situations where the patient requires a laparotomy for reasons other than tube placement.[24] Surgical tube gastrostomy can be performed either via a routine celiotomy, or by a left paracostal flank approach 10–20 mm caudal to the last rib.[25,26] The use of a mushroom-tipped catheter is recommended because the self-retaining tip is more resistant to acid damage than the balloon-tipped urethral catheters (Foley Catheter, Bard Medical, Covington, GA, Covington, GA).

Patient anesthesia and preparation of the left paracostal region is the same as the PEG tube procedure. A 30- to 50-mm skin incision is made just caudal and parallel to the last rib, beginning 20–40 mm below the ventral edge of the epaxial muscles. Blunt dissection is used to separate the external and internal oblique muscles in the direction of their fibers, and the transverse abdominal muscle and peritoneum are incised. The greater curvature of the stomach is located digitally and retracted toward the incision with Allis or Babcock tissue forceps. Location of the stomach can be facilitated by having an assistant insufflate the stomach with air using an orogastric tube.

The stomach is examined to identify the left lateral aspect of the gastric body or the caudal aspect of the fundus for the ostomy site. The stomach is packed off with moistened gauze sponges and temporary stay sutures are placed in the seromuscular layers at the 12 o'clock and 6 o'clock positions. Two full-thickness purse-string sutures are placed around the selected ostomy site. A stab incision is made in the middle of the purse string and the feeding tube is introduced into the stomach. The purse-string sutures are tightened and tied, starting with the inner suture. A layer of omentum is placed around the ostomy site between the stomach and the body wall to help prevent leakage of gastric contents. The stomach is then sutured to the body wall using simple interrupted or continuous 2–0 nylon or polypropylene sutures placed around the ostomy site. The tube may exit through the initial grid incision or via a separate stab incision. Securing of the tube outside the stomach is the same as for the PEG tube procedure.

Enterostomy tubes

Enterostomy tubes are indicated in patients unable to tolerate intragastric or intraduodenal feeding, despite having normal distal small intestine and colon function.[27,28] Specific indications for feeding via jejunostomy tube include gastric outlet obstruction, gastroparesis, recurrent/potential aspiration, proximal small bowel obstruction, and partial gastrectomy.[27] Jejunal tube feeding minimizes the stimulation of pancreatic secretion and is a viable route for patients with severe pancreatitis.[28] Jejunostomy tube feeding is rarely if ever indicated in oral surgery patients, and detailed descriptions of the technique are readily available for the reader if indications for enterostomy tube placement are present.[27,29]

Postoperative care and assessment

Nutrition

Calculation of nutritional requirements

Nutritional support provides substrates for gluconeogenesis and protein synthesis, and provides the energy needed to meet the additional demands of host defense, wound repair, and cell division and growth. An estimate of an animal's nutrient requirements is needed to determine the minimum amount of food necessary to sustain critical physiologic processes (Box 5.1). The resting energy requirement (RER) is the animal's energy requirement at rest in a thermoneutral environment and in a postabsorptive state. A linear formula can be applied to determine the RER of dogs and cats weighing at least 2 kg. Alternatively, one can utilize an allometric formula that can be applied to dogs and cats of all body weights (BW).

$$\text{Linear formula: RER } (\text{kcal/day}) = (30 \times \text{BW}_{\text{kg}}) + 70$$

$$\text{Allometric formula: RER } (\text{kcal/day}) = 70\left(\text{BW}_{\text{kg}}^{0.75}\right)$$

• Box 5.1

Enteral Feeding Worksheet for Dogs and Cats

1. Calculate resting energy requirement (RER):
   Body weight 2 to 45 kg: RER (kcal) = $(30 \times \text{Wt}_{\text{kg}}) + 70$
   Body weight < 2 or > 45 kg: RER = $70(\text{Wt}_{\text{kg}}^{0.75})$
   Body weight = ____ kg; RER = _____ kcal
2. Calculate amount of diet to feed:

Daily amount to feed: (RER) / energy density (kcal/mL or kcal/g)
Daily amount = _____ mL or g

3. Evaluate responses and modify as needed:
   Weight changes often reflect fluid dynamics in the early period following injury. Caloric requirement may need to be increased or decreased depending on animal's metabolic rate and response to nutritional support.

**Animals should be fed their RER initially, and have their body weights, physical examination findings, and ongoing losses carefully evaluated before gradually increasing or decreasing their caloric intake.*
Modified from Marks SL. The principles and practical application of enteral nutrition. Vet Clin North Am Small Anim Pract 1998;28:677–708.

Accurate, direct measurements of energy expenditure in sick or traumatized dogs and cats are not available. Despite the paucity of data on energy requirements of these animals, it is conceivable that the requirements of critically ill animals are less than normal maintenance amounts (MER), but greater than RER. Hospitalized patients should be fed at their calculated RER initially, realizing that their actual energy requirement is likely to change over the course of the disease process through recovery. Use of "fudge factors" extrapolated from the human literature to calculate the energy requirements of critically ill animals is discouraged, particularly in the early phase of nutritional support. Close observation of changes in body weight, physical examination findings (decreased subcutaneous fat stores, muscle wasting, and presence of edema or ascites), and ongoing losses (diarrhea, vomiting, exudative wounds) will help determine whether to increase or decrease the patient's caloric intake.

Diet selection

The type of formula to feed the patient will depend on the selected route of feeding, the functional status of the gastrointestinal tract, and the animal's nutrient requirements. Other factors, such as cost, availability, and ease of use may also be important. Patients fed via nasoesophageal or jejunostomy feeding tubes are limited to receiving liquid enteral formulas that have a caloric density of approximately 1 kcal/mL. When selecting a liquid formula for feeding, one should pay particular attention to the amount of protein in the formula, the type of protein (intact proteins, peptides, and amino acids) and the quality of the protein. Whole egg has the highest biologic value, followed by cow milk, lactalbumin, beef, soy, and casein. Most human liquid formulas contain less than 20% protein calories, precluding their use for the long-term (longer than 2 weeks) feeding of cats. The lower-protein formulas should be supplemented with protein modules such as Promod (Abbott Nutrition, Chicago, IL) at 15–30 g powder per 240 mL can. Almost all human liquid enteral formulas lack taurine, an essential amino acid in cats, necessitating its supplementation (approximately 250 mg taurine per 240 mL can) in this species. High-protein commercial human liquid formulations contain between 25% and 35% protein calories and include Promote (25% of calories from protein; Abbott Nutrition, Chicago, IL) and Impact Advanced Recovery (35% of calories from protein; Nestle HealthCare Nutrition, Bridgewater, NJ).

Polymeric solutions contain macronutrients in the form of isolates of intact protein (casein, lactalbumin, whey, egg white), triglycerides, and carbohydrate polymers. The carbohydrates are usually glucose polymers in the form of starch, and its hydrolysates and the fats are of vegetable origin. The osmolality varies between 300 and 450 mOsm/kg in solutions with a caloric density of 1 kcal/mL; however, the osmolality may reach 650 mOsm/kg in solutions with a greater caloric density. Monomeric solutions contain protein as peptides or amino acids, fat as long-chain triglycerides (LCT) or a mixture of LCT and medium-chain triglycerides (MCT), and carbohydrates as partially hydrolyzed starch maltodextrins and glucose oligosaccharides. These solutions require less digestion and their absorption is more efficient than regular foods or polymeric solutions; however, the partially digested macronutrients contribute to the higher osmolality, which is between 400 and 700 mOsm/kg.

Commercial blended pet food diets are recommended for feeding via pharyngostomy, esophagostomy, or gastrostomy tubes. In select cases, the feeding of a liquid enteral formulation may be indicated (nasoesophageal or jejunostomy tube feeding). There are a number of complete and balanced veterinary enteral formulations (Table 5.1) that contain adequate amounts of protein, taurine, and micronutrients, precluding the need for supplementation in most situations.

TABLE 5.1

Key Information on Selected Veterinary Diets Formulated for Critical Care and Tube Feeding

							CALORIC Distribution (% OF TOTAL KCAL)		
Manufacturer	Product Name	Appropriate for Nasoesophageal Tube	Canine	Feline	Protein Source	Caloric Density (kcal/mL)	Protein	Fat	Carbohydrate
Hill's	Prescription Diet a/d Canine/Feline	No	Yes #	Yes	Pork, chicken, turkey, corn	1.2	33	55	12
Purina	ProPlan Veterinary Diets CN Critical Nutrition	No	Yes *	Yes *	Beef, pork, lamb, chicken, turkey, rice, oat	1.36	28	63	9
Royal Canin	Veterinary Diet Recovery RS	No	Yes **	Yes **	Chicken, pork, beef, egg	1.04	37	59	4
Royal Canin	Feline & Canine Recovery® Liquid	Yes	Yes *	Yes *	Milk protein, soy protein	0.9	32	48	20
Royal Canin	Canine Gastrointestinal Low Fat™ Liquid	Yes	Yes #	No	Milk protein, soy protein	0.9	35	19	46
Royal Canin	Canine Renal Support Liquid	Yes	Yes #	No	Milk protein	1.3	13	51	36
Royal Canin	Feline Renal Support Liquid	Yes	No	Yes #	Milk protein, soy protein	0.9	26	50	24
EmerAid	Intensive Care HDN	Yes	Yes *	No	Casein, hydrolyzed soy protein	1.36–2.26	31	42	27
EmerAid	Intensive Care HDN	Yes	No	Yes *	Casein, hydrolyzed soy protein	1.13–2.41	31	55	14
EmerAid	Canine Sustain HDN	Yes	Yes **	No	Casein, hydrolyzed soy protein	2.23–1.34	26	52	22
EmerAid	Feline Sustain HDN	Yes	No	Yes **	Casein, hydrolyzed soy protein	2.35	29	64	7

#Balanced for intermittent or supplemental feeding only

*Balanced for adult maintenance

**Balanced for growth and adult maintenance

Note: This information is current as of March 2019. Commercial pet diet formulations may change without notice. If particular diet parameters listed herein are important for diet selection, please verify that the formulation has not changed.

Feeding should be delayed for 24 hours after placing a gastrostomy tube, to allow return of gastric motility and allow formation of a fibrin seal. In contrast, feeding can be instituted immediately following pharyngostomy or esophagostomy tube placement once the animal has fully recovered from anesthesia. Diet can be administered as bolus feedings or continuous infusion when feeding via esophagostomy and gastrostomy tube. Improved weight gain and decreased gastroesophageal reflux have been reported in human patients given continuous feedings, although similar studies are lacking in the veterinary literature.[30] If continuous feeding is employed, it should be interrupted every 8 hours to determine the residual volume by applying suction to the feeding tube. If the residual volume is more than twice the volume infused in 1 hour, feeding should be discontinued for 2 hours, and the rate of infusion decreased by 25% to prevent vomiting. Treatment with metoclopramide (1–2 mg/kg/24 hour as a continuous infusion) may be used to enhance gastric emptying and prevent vomiting.[31]

With bolus feeding, the required daily volume of food should be divided into four to six feeds. Patients are usually fed approximately 25% of their caloric requirement on the first day of feeding, with a gradual increase of 25% of the caloric requirement per day. Most patients are able to reach their energy requirement by the fourth or fifth day of feeding. The food should be warmed to room temperature and fed slowly through the tube to prevent vomiting. Flushing of the tube with 15–20 mL of lukewarm water helps prevent clogging. Before each feeding, aspirate the tube with an empty syringe to check for residual food left in the stomach from the previous feeding. If more than half of the last feeding is removed from the stomach, skip the feeding and recheck residual volume at the next feeding.

Removal and replacement

Esophagostomy tube removal

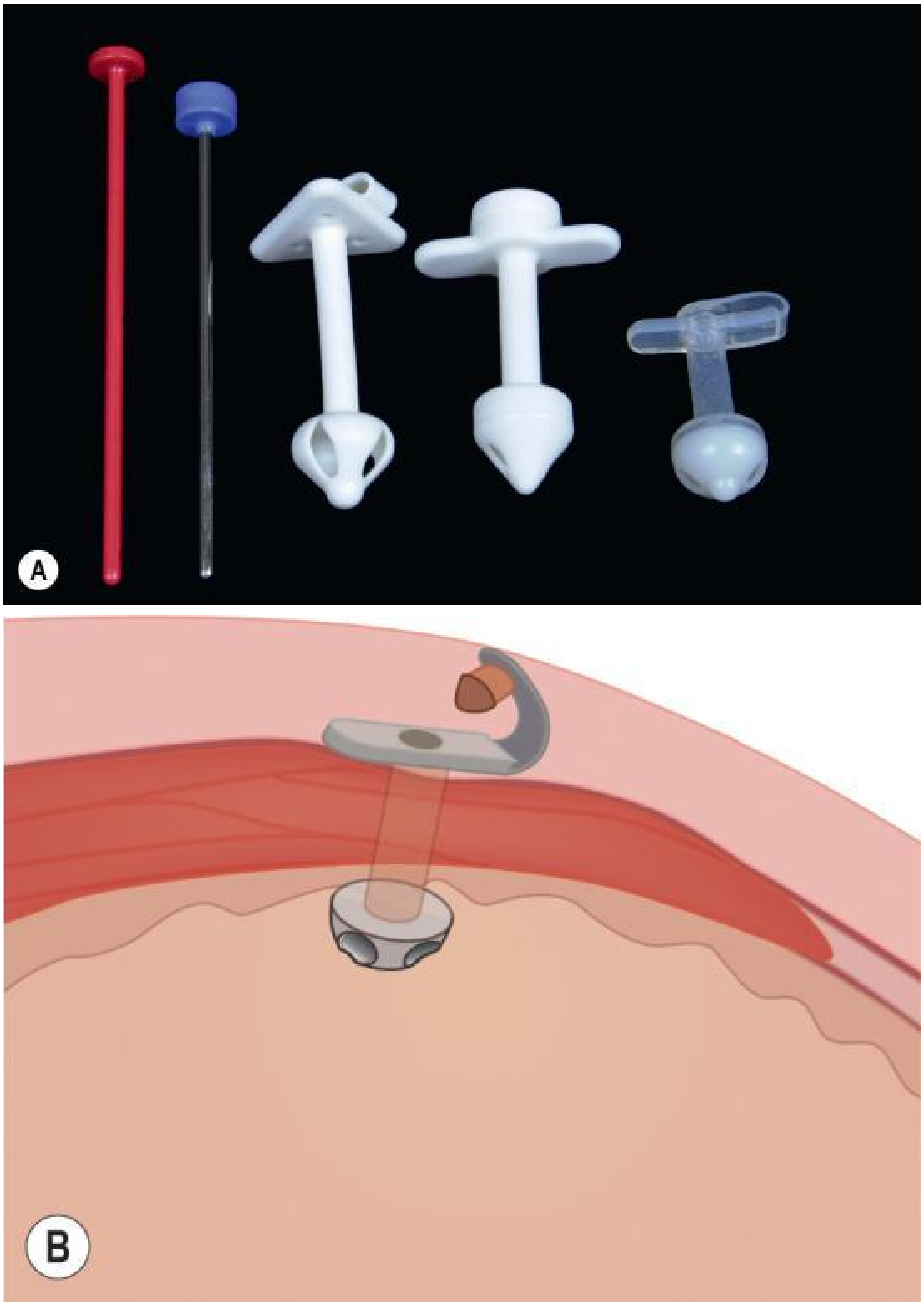
Unlike gastrostomy tubes, an esophagostomy tube can be removed the same day it is placed if necessary without concern for leakage and development of secondary complications. The dressing and sutures are removed while the tube is held in place. The tube is then occluded by kinking and pulled out using gentle traction. The ostomy site should be cleaned, antibiotic ointment applied, and a light dressing placed around the neck. The dressing should be removed in 24 hours, and the ostomy site inspected. The ostomy site should close within 24–36 hours. Skin sutures are not needed for closure of the ostomy site.

Gastrostomy tube removal

For percutaneously placed tubes, it is recommended that the tube be left in place for a minimum of 21 days. Animals receiving immune-suppressive therapy or patients that are severely debilitated may require longer for a peritoneal seal to form. The tube should only be removed when oral food intake is sufficient to meet the patient's caloric requirement. One of two methods of Pezzer tube removal can be applied. The tube can be cut at the body wall and the mushroom tip pushed into the stomach to be passed in the feces. This method is safe in medium to large dogs, because the mushroom and internal flange should be easily passed in the stool. Alternatively, a stylet can be inserted into the tube to flatten the mushroom tip, while exerting firm traction on the tube. This method is recommended for cats and small dogs, because the mushroom can cause intestinal obstruction. Removal of the MILA catheter (MILA International, Inc., Florence, KY) is accomplished by deflating the bumper which occurs once the Y-port adapter is removed. Catheters with a dome (Bard Medical, Covington, GA) are removed by gentle but firm traction on the tube. The gastrocutaneous tract should seal with minimal or no leakage within 24 hours.

Gastrostomy and esophagostomy tube replacement

The PEG tube may malfunction or be prematurely removed by the patient, requiring replacement. If the gastrostomy tube is removed within 14 days of placement (before establishment of the gastrocutaneous tract), a PEG procedure should be performed to evaluate the gastric mucosa and verify correct positioning of the replacement gastrostomy tube. If the tube is inadvertently removed once the gastrocutaneous tract is well healed, one can replace the original catheter with a balloon-type catheter or a low-profile gastrostomy device (LPGD) (Bard Peripheral Vascular Inc., Tempe, AZ) (Fig. 5.6A).[32] Both catheter types do not require an endoscopic procedure or anesthesia for placement. The gastrostomy "button" is a small, flexible silicone device that has a mushroom-like dome at one end and two small wings at the other end that lies flush with the outer abdominal wall (Fig. 5.6B). A one-way antireflux valve prevents reflux of gastric contents through the top of the tube. There are two types of LPGDs: obturated and non-obturated. The obturated device has an enlarged mushroom tip that must be stretched for placement in the stomach by using a special introducer (Fig. 5.6C).[33] The non-obturated tube works like a Foley catheter and does not require forceful entry into the gastrostomy stoma.[33] The length of the gastrocutaneous fistula must be precisely determined to guide correct selection of the appropriate "button" shaft length. This is accomplished with a special stoma measuring device provided with the kit. The main advantages of the LPGD include its durability due to the silicon material, decreased likelihood of inadvertent removal by the patient, and the aesthetically pleasing appearance to the clients.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• **FIG. 5.6** (**A**) Low-profile gastrostomy devices and obturators used for stretching the dome-shaped tip of the device. From left, the Stomate low-profile device (Ross Laboratories, Columbus OH), the Cook low profile device (Cook Medical, Bloomington, IN), and the Button® low-profile device (Bard Peripheral Vascular Inc., Tempe, AZ). (**B**) Low-profile gastrostomy device with small outer wings of the device lying flush against the skin of the abdominal wall. For feedings, the small plastic plug is removed and a feeding adapter is connected to a syringe. (**C**) Correct technique for stretching the dome-shaped tip of the low profile gastrostomy device with an obturator. The dome should not be stretched by passing the obturator through the lumen of the device as it will compromise the integrity of the anti-reflux valve located adjacent to the dome.

Complications

Gastric pressure necrosis

Gastric pressure necrosis can occur from either the mushroom of the PEG tube or flange eroding the mucus layer of the stomach due to excessive tension being exerted on the PEG tube during placement. In addition, overzealous traction of the PEG tube followed by placement of the external flange flush against the skin of the patient can also cause pressure necrosis characterized by redness, swelling, and moistness of the skin. To minimize this problem from occurring, ensure that the PEG tube can be rotated following its placement and leave a 5-mm space between the external flange and the skin.

Feeding tube displacement

This is a relatively common problem, particularly with nasoesophageal tubes. Displacement of the tube can lead to aspiration, diarrhea or, in the case of gastrostomy tubes, peritonitis. Gastrostomy tubes should be marked with tape or a marking pen at the level of the skin to help verify the position of the tube. Detachment of the stomach from the abdominal wall with consequent intraperitoneal leakage of gastric contents can occur in large-breed dogs, and an internal flange should be placed in these animals to minimize dislodgement of the tube.

Tube obstruction

Obstruction of the feeding tube is one of the most common complications of enteral feeding.[34] Most obstructions are secondary to coagulation of formula, although obstruction by tablet fragments, tube kinking, and precipitation of incompatible medications can also result in tube obstruction. Nasoesophageal tubes are prone to obstruction because of their small diameter, and obstruction also occurs up to three times more frequently in patients fed by continuous versus bolus feedings.[35] Sucralfate and antacids have been reported to precipitate with enteral formulas and cause tube obstruction.[35] Several "remedies" have been advocated to relieve tube obstruction. Warm water injected with gentle pressure and suction will relieve most obstructions. For more unyielding obstructions, carbonated water is instilled into the tube and allowed to sit for 1 hour before applying gentle pressure and suction. Pancreatic enzyme infusions and meat tenderizer have also been advocated to dissolve tube obstructions.[34] On rare occasions, the passage of an angiographic wire down the lumen is needed to unclog the tube. Tube obstructions can be minimized by flushing the feeding tube with warm water before and after administering medications or enteral feedings. The tube should also be flushed after checking for gastric residuals, because the acid pH will cause the formula to coagulate in the tube. Elixir forms of medication should be used rather than crushed tablet forms whenever possible. Tablets should be crushed and dissolved in water prior to administration through the feeding tube, if no alternative form of medication is available.

Leakage through ostomy sites

Mild leakage at the stoma site can occur for the first few days following placement of the feeding tube. Persistent leakage may indicate tube dysfunction, peristomal infection, or a stoma site greater than necessary for the tube. Signs of inflammation with or without discharge or fever may indicate infection of the stoma site. This must be differentiated from fasciitis, as a simple wound infection can usually be treated locally with dilute povidone-iodine solution, topical povidone-iodine antibacterial ointments, and more frequent dressing changes. Systemically administered antibiotics are usually reserved for patients with systemic signs of infection.

Aspiration

Pulmonary aspiration is a common complication of enteral feeding, although the actual incidence of this complication is difficult to determine due to the lack of consistency in how aspiration is defined. Risk factors for aspiration include impaired mental status, neurologic injury, absence of a cough or gag reflex, mechanical ventilation, and previous aspiration pneumonia.[36,37] The source of the aspirated material should be identified because withholding gastrostomy feedings or placing a jejunostomy feeding tube in a patient will have no benefit if the patient aspirated oropharyngeal secretions. Although controversial, most authors agree that postpyloric feeding reduces the risk of aspiration.[38] In addition, the use of continuous versus bolus feedings has been shown to induce less gastroesophageal reflux than bolus feedings.[30]

Diarrhea

Diarrhea is the most commonly cited complication associated with tube feeding in human and animal patients, with an incidence ranging from 2.3% to 63%.[39] The clinical implications of enteral feeding-related diarrhea are significant. Severe diarrhea leads to fluid, electrolyte, and nutrient loss and can cause considerable distress to the patient. Diarrhea in tube-fed patients occurs due to multiple factors, including hypoalbuminemia, hyperosmolar or high-fat diets, infected diets, and concomitant antibiotic therapy.[40] The incidence of diarrhea in enterally fed patients taking antibiotics far exceeds the incidence in normally fed patients taking the same antibiotics.[41] Antibiotic-associated diarrhea may arise from overgrowth of enterobacteria *(Klebsiella*, *Proteus*, *Pseudomonas)* or from proliferation of *Clostridium difficile*. Antibiotic administration is also associated with decreased concentrations of fecal short-chain fatty acids, occurring as a result of decreased colonic carbohydrate fermentation.[42]

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SECTION 2:

Surgical Methods

OUTLINE

CHAPTER 6

Diagnostic imaging in oral and maxillofacial surgery

Derek D. Cissell, David Hatcher, Boaz Arzi, Frank J.M. Verstraete

Diagnostic imaging modalities

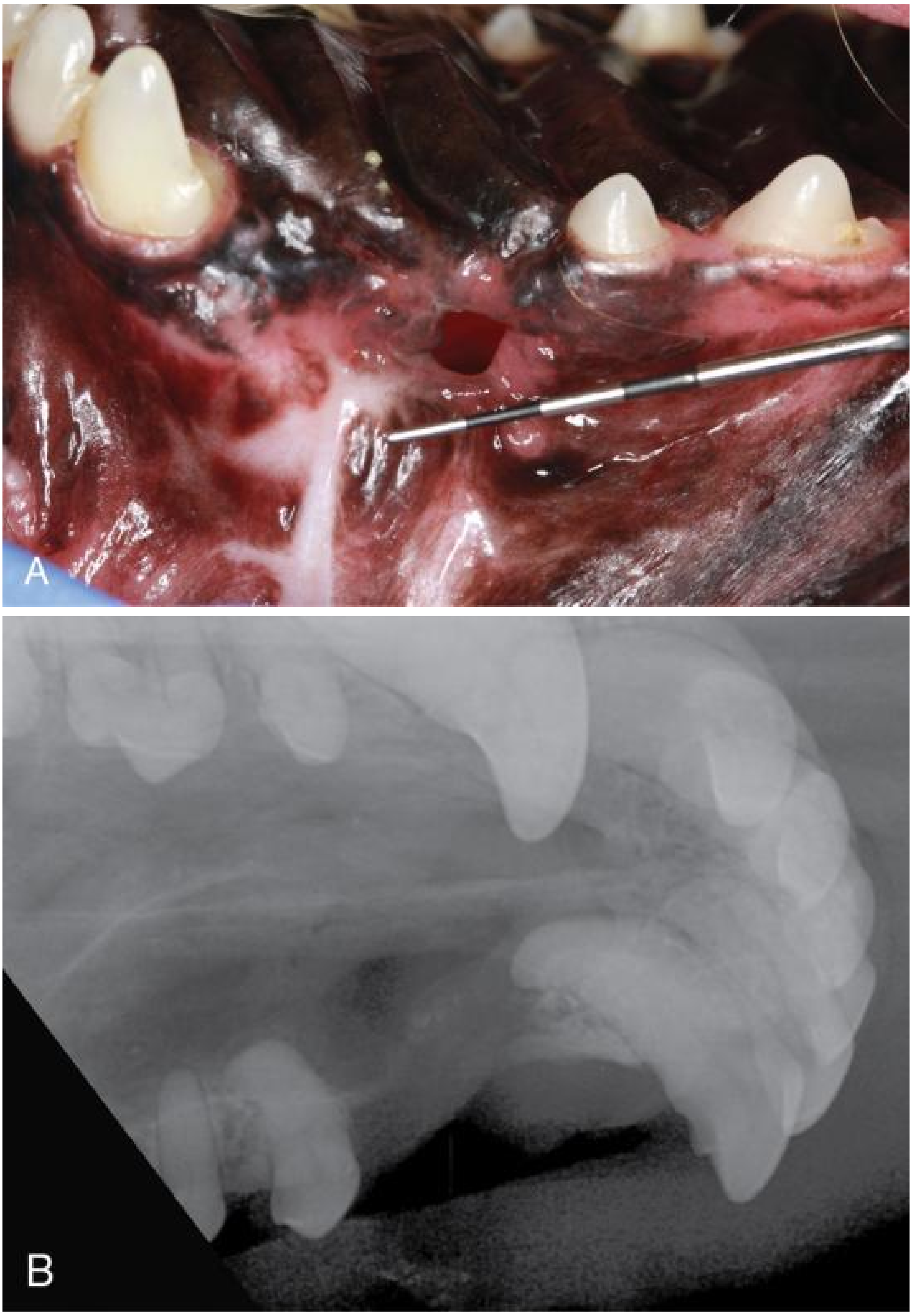
The gold standard for all diagnostic imaging is accuracy and precision of revealing the anatomic truth. Multiple two-dimensional (2D) and three-dimensional (3D) static and dynamic diagnostic imaging methods have been developed, each with its own benchmark on a continuum of revealing the anatomic truth. Many of the diagnostic challenges that routinely occur in clinical practice can benefit from imaging investigations. The art of imaging is choosing the appropriate imaging tool and optimizing that tool in order to fulfill the imaging objectives. Nearly all current imaging employs digital methods; therefore, there are at least two distinct phases in the imaging chain: (1) capture and reconstruction of the anatomy and (2) software display and manipulation of the anatomy.

Conventional radiography

Radiography has been the primary diagnostic imaging modality from the time of its first medical application through to the early 21st century; as of 2006, 2D radiographic and fluoroscopic diagnostic procedures still far outnumbered other imaging modalities in human hospitals.[1] Radiographs rely on differing attenuation of X-rays by materials with varying compositions and densities. The average atomic number and density of air, fat, soft tissue, bone, teeth, and metal create the six classic radiographic opacities distinguishable in a radiograph. The thickness of a given tissue or material, as well as superimposition of multiple objects within the path of the X-ray beam, further influences the absorption of X-rays. Absorption of a large proportion of X-rays by thick objects or highly attenuating materials will cause the corresponding region of the image to appear radiopaque (i.e., light gray or white) in a radiograph. The mineral content and density of bones and teeth create inherently high natural contrast, and radiographs excel at detecting orthopedic and dental pathology and injuries.

Dental radiography

Dental radiographs form an essential part of a comprehensive oral examination, and radiological findings are a key element in decision-making and treatment planning in dental practice. A commonly used approach is to radiograph those areas where one expects to find pathology, based on the visual oral examination and periodontal probing (Fig. 6.1A, B). With this approach, the indications for dental radiography can be summarized as follows: (1) clinical signs of periodontal or endodontal disease; (2) prior to extraction and postextraction; (3) before, during, and after endodontic procedures; (4) clinical staging of oral tumors; (5) dental trauma; and (6) diagnosis of missing teeth.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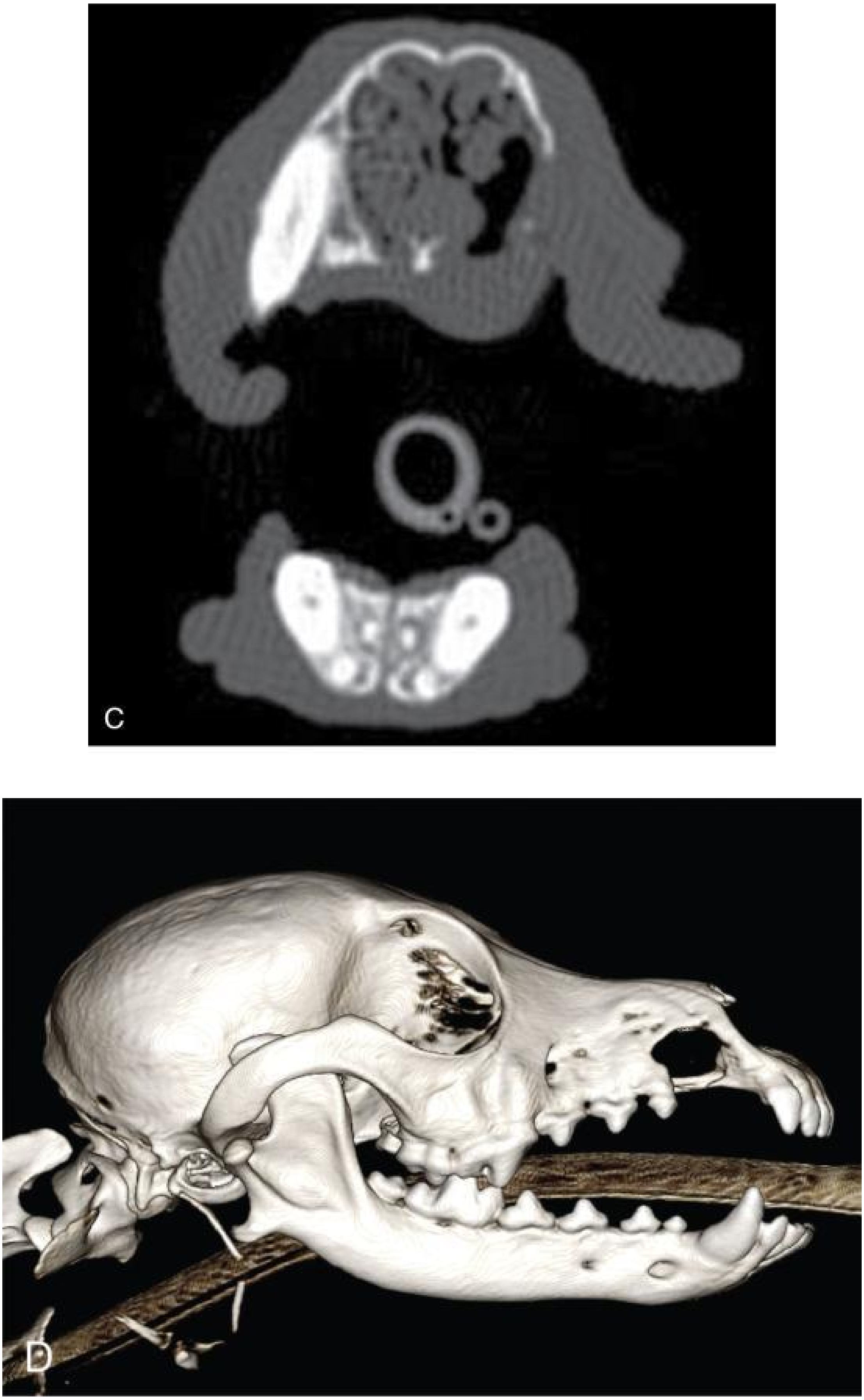


• FIG. 6.1 (**A**) Clinical image of the rostral right maxilla of a 10-year-old miniature dachshund that was presented for evaluation and treatment of an oronasal fistula. (**B**) A dental radiograph of the region shows geographic bone loss in the location of the missing right maxillary canine tooth, as well as the right maxillary third incisor and first premolar teeth. (**C**) Cone-beam computed tomography confirmed that the right maxillary canine tooth is missing and there is regional bone loss of the maxilla, nasal bone, and rostral hard palate. Soft tissue attenuation extends from the oral cavity into the right nasal cavity, with mild destruction of the nasal septum and rostral left and right nasal turbinates. These findings are consistent with oronasal fistula. (**D**) Three-dimensional volume rendering demonstrates the spatial location of the fistula and the extent of the bone defect.

A "full-mouth survey" is defined as a series of radiographs depicting not only the teeth present but also edentulous parts of the jaw bones that are normally tooth-bearing. It is common practice for a full-mouth radiographic survey to be obtained during a patient's first visit to a dentist. This is done for two main reasons: to determine the condition of the teeth and bones and to establish a baseline for future changes. The routine use of full-mouth radiography is well established in human dentistry and, to a lesser extent, in small animal dentistry. For the adult, dentulous, new patient with clinical evidence of generalized dental disease, a full-mouth radiographic examination is considered appropriate.

A study of the clinical value of radiographs in veterinary practice showed that the diagnostic yield of full-mouth radiography in dogs and cats is high.[2,3] In 72.6% of dogs and 86.1% of cats, radiography of areas with clinically evident dental disease provided additional, clinically useful, or essential information. Radiography of areas without clinically evident dental disease revealed incidental findings in 41.7% of dogs and 4.8% of cats and clinically important lesions in 27.8% of dogs and 41.7% of cats. The routine use of full-mouth radiography is therefore justifiable for new canine and feline dental patients.

Direct digital imaging systems have become the standard in dental radiography, replacing radiographic film. The majority of these systems are based on charge-coupled device (CCD) technology. The disadvantages of CCD systems include the restrictions associated with the bulky sensor, connecting wire, limited sensor size (no size #4), and high sensor cost. Systems based on photo-stimulated phosphor (PSP) technology are preferred. Digital imaging based on PSP technology uses reusable imaging plates (including size #4) without cables or sensors, in combination with a conventional dental X-ray unit.

Skull radiography

Despite the advantages of X-ray imaging for evaluating bones and teeth, the complex anatomy of the skull makes acquisition and interpretation of whole skull radiographs challenging. Further, it cannot be overemphasized how important proper positioning and patient restraint are for the acquisition of skull radiographs. Sedation or general anesthesia is necessary in most patients to obtain skull radiographs. Complete radiographic evaluation of the skull requires multiple radiographic projections, depending on the region of suspected pathology. Radiographic projections of the skull should include lateral, ventrodorsal (VD) or dorsoventral (DV), and oblique projections. Lateral projections detect abnormalities along the ventral and dorsal surfaces of the head and skull and provide excellent visualization of the larynx and pharynx but are hindered by superimposition of the right and left halves of the skull. Well-positioned VD and DV projections allow the examiner to use the skull's natural symmetry to aid in the detection of disease. Oblique projections aid in localizing disease to a single maxilla or mandible and help detect lesions at the dorsolateral or ventrolateral aspects of the skull.

Acquiring paired oblique projections from opposite directions (i.e., right dorsal–left ventral and left dorsal–right ventral obliques) helps distinguish normal anatomy from pathology.

Additional projections may be indicated to further evaluate the maxilla, nasal cavities, sinuses, and other regions of the skull. An open-mouth VD projection provides the best possible visualization of the maxillae and nasal cavities using 2D skull radiography. Similarly, an open-mouth DV projection provides excellent visualization of both mandibles without superimposition of the maxillae. Open-mouth VD or DV projections are acquired with a patient in dorsal or ventral recumbency, respectively, while using gauze or tape around the mandibular or maxillary teeth to open the mouth. Angling the X-ray beam slightly rostral to caudal is often necessary for open-mouth projections to minimize superimposition of the maxillae and mandibles. A rostrocaudal projection of the skull provides good visualization of the frontal sinuses and zygomatic arches; this projection can be acquired with a patient in dorsal recumbency using foam wedges or other passive restraint to position the palate perpendicular to the X-ray detector. While the increasing availability of computed tomography (CT) and magnetic resonance imaging (MRI) is largely replacing skull radiographs in veterinary medicine, thorough, well-positioned skull radiographs still play a role when other imaging modalities are not possible.

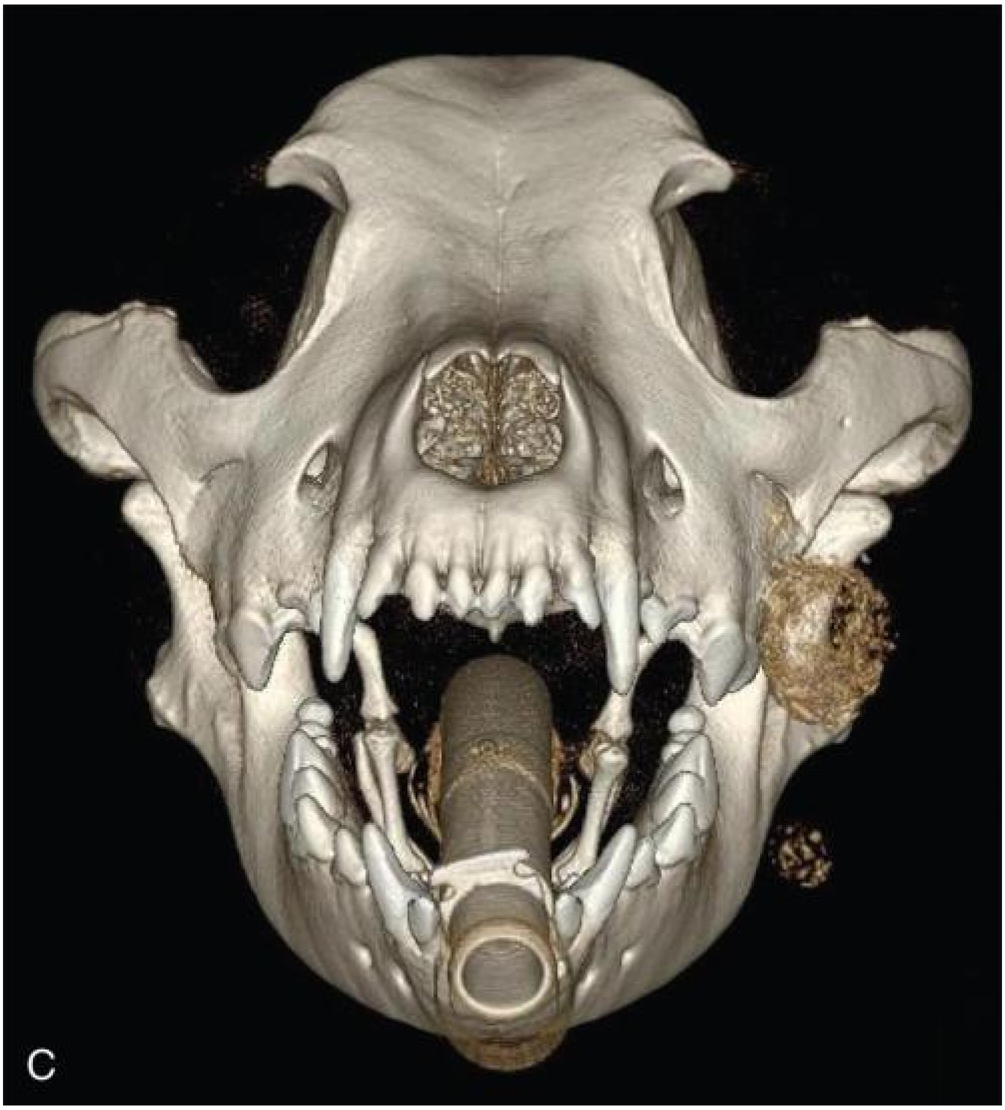
Cross-sectional imaging

Computed tomography

X-ray CT (a.k.a. computer assisted tomography or CAT) has become the gold standard modality for imaging the bones of the skull, nasal cavities, and sinuses. Early CT scanners rotated a thin, fan-shaped X-ray beam and array of detectors around a patient to create a complete cross-sectional image of a slice of anatomy. The patient was then translated a few millimeters relative to the X-ray beam, an image of the next slice was acquired, and this process was repeated to generate a stack of consecutive cross-sectional images that recreated the patient's 3D anatomy. The resulting images had the major advantage of removing superimposition, making CT superior to radiographs for the evaluation of complex body parts. Additionally, the collimated, fan-shaped beam of X-rays markedly improved contrast resolution by reducing detection of scattered X-ray photons. The improved image quality achieved by CT allowed soft-tissue structures, such as lymph nodes, salivary glands, and blood vessels, to be identified even without administration of exogenous contrast media (Fig. 6.2A, B). Technology in modern CT scanners allows "helical" acquisition, in which a patient translates continuously through the path of the collimated X-ray beam to create a 3D data set that is reconstructed into individual slices (see Fig. 6.2C). Additionally, improvements in X-ray detectors and computing technology reduce scan times via acquisition of multiple slices of data simultaneously. Multislice helical CT scanners have become nearly ubiquitous in veterinary medicine.


A


B



• FIG. 6.2 Post–intravenous contrast administration computed tomography images of the head of a 6-year-old American pit bull terrier with a fibroblastic osteosarcoma at the left caudal maxilla (**A, B**). Note the mineral attenuating left maxillary mass and partially mineralized left mandibular lymph node with similar appearance. (**C**) A three-dimensional rendering of the skull demonstrating the spatial location of the mass and the affected (metastatic) left mandibular lymph node.

CT image quality is influenced by parameters related to acquisition of the raw data and by how the raw data are reconstructed into an image and subsequently displayed. CT study protocols may be predefined by the manufacturer or user for imaging specific body regions and applications. An acquisition protocol consists of factors related to X-ray tube exposure, image geometry, and pitch. A user can set the X-ray tube current (mA), which determines the flux of X-ray photons emitted by the tube. Higher mA settings improve image quality by increasing the signal:noise ratio but result in greater radiation dose to the patient and greater heating of the X-ray tube. Modern CT scanners often have an "auto mA" setting that will reduce mA for exposure of thin body parts and increase mA for exposure of thick, highly attenuating regions. The potential difference (kVp) of the CT X-ray tube determines the maximum energy of photons in the X-ray beam. The kVp is typically set much higher (120–130 kV) and has less influence on final image contrast than for diagnostic radiographs. Once CT study protocols are established, exposure factors are typically not modified except to adjust for large patient size or the presence of highly attenuating objects (e.g., metallic implants) or to offset changes in image quality associated with other parameters.

Image geometry is defined by the field of view (FOV), matrix size, and slice thickness, which are critical to realize the optimal possible spatial resolution for a CT study. The FOV should be large enough to include the entire diameter of the body region being imaged. The image matrix divides each slice into discrete elements, each representing a tiny volume of the patient's anatomy. Typical matrix sizes for modern CT images are 512 × 512 or 1024 × 1024. Together, the FOV, matrix size, and slice thickness determine the size of each volume element (a.k.a., voxel) in the patient that will be displayed as individual pixels in the image. A smaller FOV or larger matrix size will result in finer spatial resolution in the x–y plane of the image, whereas slice thickness determines spatial resolution in the z-axis of the image study. Historically, CT scanners have been capable of finer resolution in the x–y plane than in the z-dimension, but current state-of-the-art CT scanners are capable of near isotropic resolution. Importantly, image geometries that achieve the finest possible spatial resolution sacrifice signal:noise ratio and contrast resolution. The goals of an imaging study should be considered when deciding the optimal image geometry.

Pitch is the ratio of the distance translated by the patient during a single rotation of the X-ray tube relative to the image slice thickness. For a single-slice helical CT scanner, acquisition of 1-mm images with a pitch of 1 indicates that the patient and CT bed translate 1 mm for each rotation of the X-ray tube. For multislice helical CT scanners, the number of slices being acquired simultaneously is factored into the definition of pitch; thus, during acquisition of 1-mm images with a pitch of 1 by a 64-slice helical CT scanner, the patient would translate 64 mm during each rotation of the X-ray tube. Default pitch values for imaging protocols are typically close to 1, but pitch >1 can be used to decrease total image acquisition time. Increasing pitch will reduce signal:noise ratio, but the impact on image quality is usually not noticeable for pitch ≤1.5. Decreasing pitch will increase signal:noise ratio at the expense of longer acquisition time. Decreasing pitch may be beneficial to improve image quality and reduce artifacts associated with highly attenuating objects that cannot be removed or repositioned.

Reconstruction of the X-ray data to create a stack of images has a major influence on final image quality. Multiple different reconstruction algorithms (a.k.a. filters or kernels) are typically available for a given CT system, and an operator can choose the algorithm best suited for a particular patient body region and application. In general, different reconstruction algorithms provide varying levels of edge enhancement to improve spatial resolution or smoothing of the image to provide greater contrast resolution. Reconstruction algorithms may be further optimized by the manufacturer for use in specific body regions. It is important for CT users to become familiar with the image reconstruction algorithms available on their system. Greater edge enhancement is useful for imaging of fine details in high-contrast tissues, such as bone and lung. Edge-enhancing algorithms also tend to increase the appearance of image noise and some artifacts. Conversely, smoother algorithms improve contrast resolution for evaluating soft tissues and reduce some artifacts. Most systems allow more than one reconstruction of the same raw data set without reimaging the patient. Two or more reconstructions are desirable when an imaging study calls for optimal evaluation of both hard and soft tissues.

Patient positioning also influences CT image quality. Acquisition of a CT study with the patient off center within the gantry will reduce contrast resolution in the image and increase the patient's absorbed radiation dose. Ensuring that the region of interest is straight, with its long axis perpendicular to the CT gantry, greatly improves ease of image interpretation by acquiring symmetrical transverse images. Ideally, the limbs and any extraneous objects should be positioned such that they are not within the same transverse plane as the region of interest. For example, when imaging the head, the limbs should be flexed or retracted away from the head. Electrocardiography lines, endotracheal tubes with metallic inserts, and other highly attenuating objects should be repositioned, avoided, or removed whenever possible. Lastly, acquiring images with the patient's mouth open or using dry gauze sponges to separate the oral soft tissues can greatly increase conspicuity of oral, pharyngeal, and laryngeal lesions.

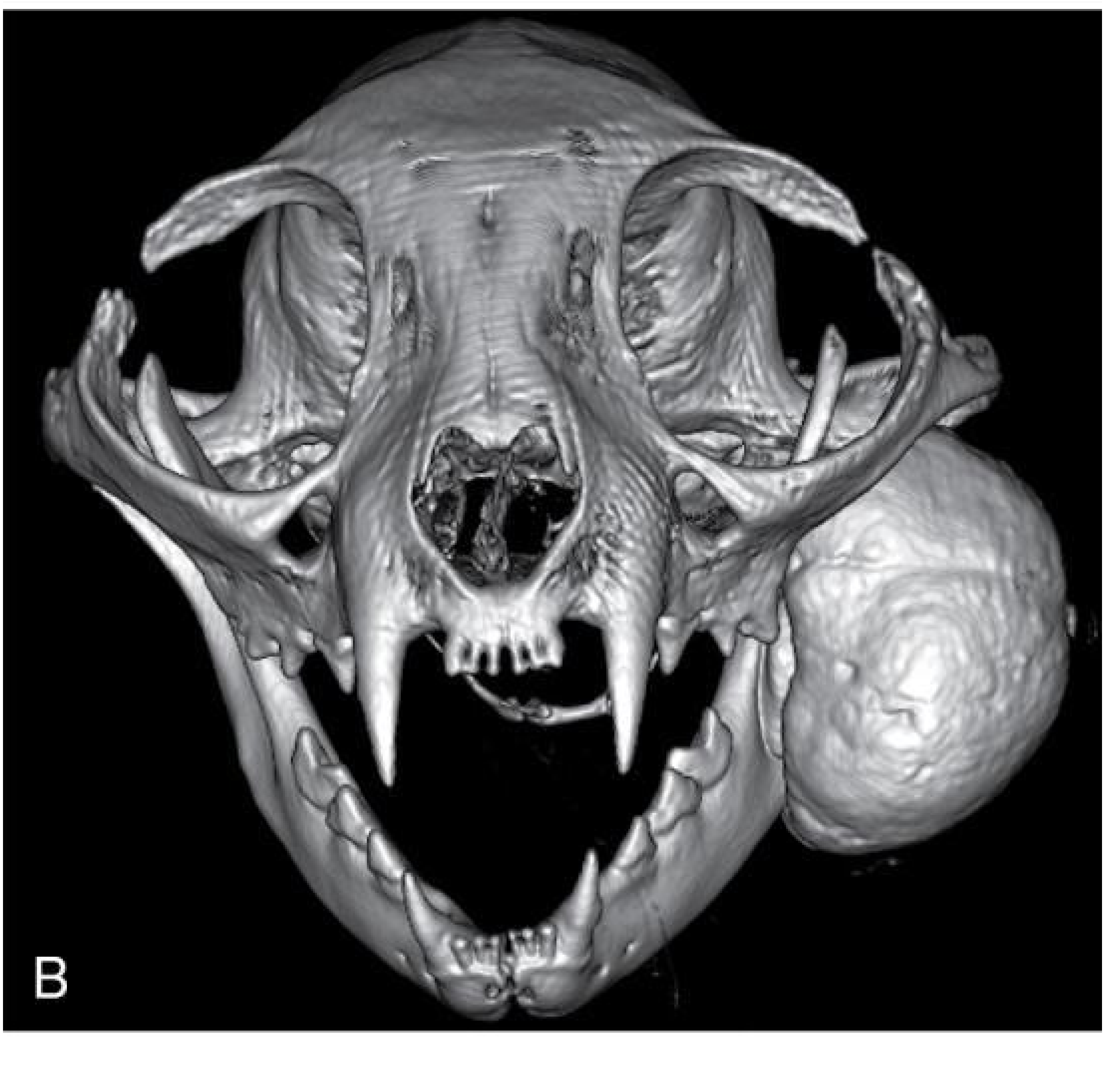
Contrast-enhanced computed tomography

Administration of iodinated contrast media significantly increases the diagnostic value of CT by improving the visibility of certain normal tissues (e.g., blood vessels, lymph nodes, and salivary glands), as well as pathologic lesions associated with neovascularization or high vascular permeability (see Fig. 6.2). Precontrast images should be acquired prior to administration of iodinated contrast media, and contrast-enhanced images should be obtained without moving the patient. Reconstruction of images using an appropriate algorithm is essential, and it may be beneficial to obtain contrast-enhanced images with slice thickness >1 mm to maximize contrast resolution. Otherwise, contrast-enhanced image acquisition should match precontrast images to facilitate comparison between images. Systemic contrast is typically administered intravenously at dosages of 600–880 mg iodine per kg body weight. Adverse reactions to iodinated contrast media are rare, but dehydration, cardiovascular disease, and renal disease represent greater risks for contrast-associated complications. Iodinated contrast media can be ionic or nonionic. Ionic contrast media include salts of diatrizoate, metrizoate, or iothalamate; most are high in osmolality and associated with greater risk of complications.[4] Nonionic contrast media include iopamidol, iohexol, ioxilan, and iopromide, which are lower in osmolality and associated with fewer adverse reactions.[5] In addition to systemic administration, nonionic iodinated contrast media can be used for sialography and dacryocystography and are typically diluted with sterile saline solution to reduce artifacts that may occur with undiluted contrast media.

Cone-beam computed tomography

Cone-beam CT (CBCT) is possibly the most significant of the recent advances in dental imaging for humans and is anticipated to have a similar value for veterinary medicine (see Figs. 6.1C, D, and Fig. 6.3). CBCT machines are compact, relatively inexpensive, and easy to operate.


A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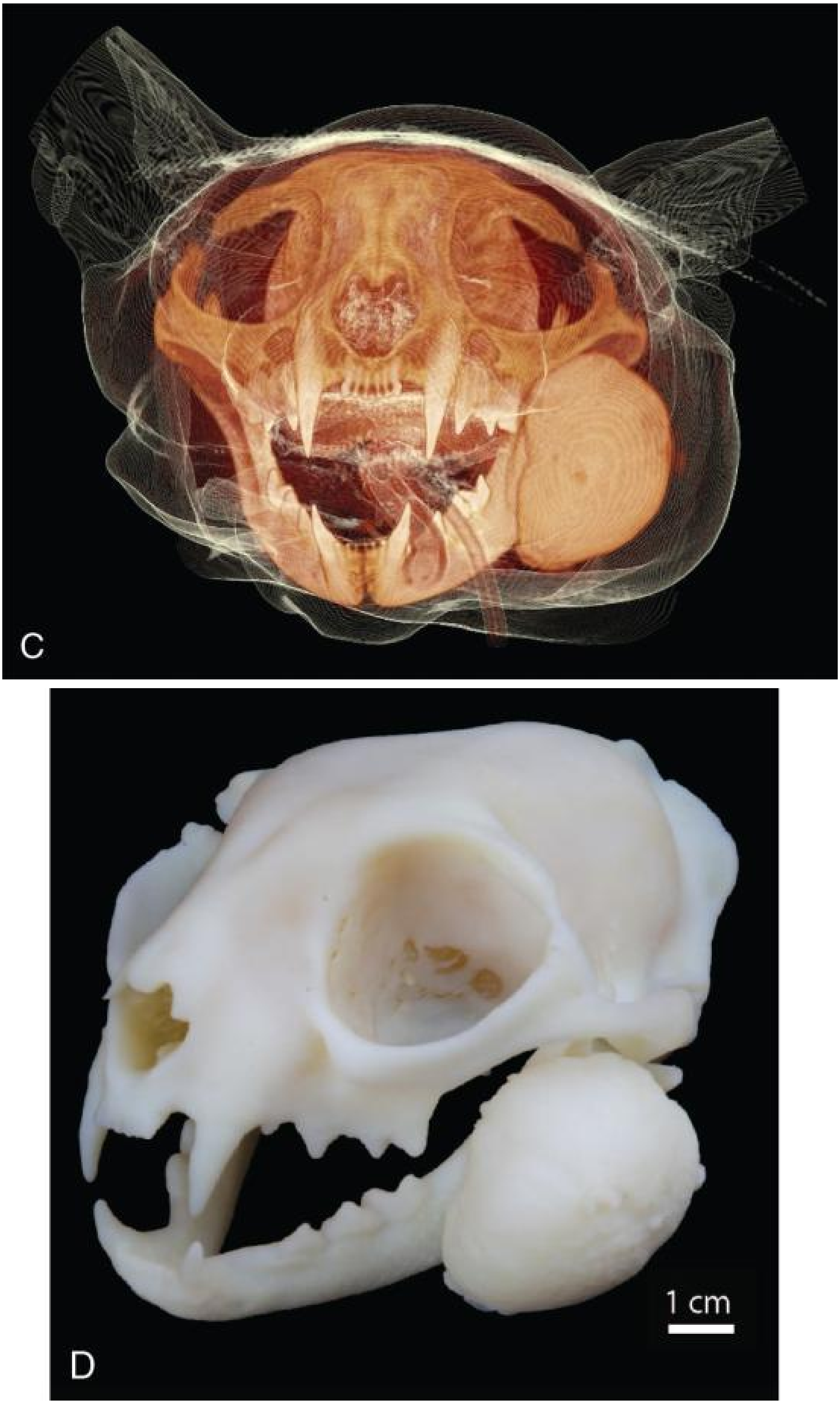


• FIG. 6.3 A cone-beam computed tomography scan of the head of a 7-year-old cat. Note the (**A**) transverse and (**B**) three-dimensional (3D) images denoting a smoothly margined, mineral mass with homogenous density arising from the caudal left mandible consistent with the histological diagnosis of periosteal osteoma. In addition, (**C**) a 3D image showing the bone, tumor, and soft tissues and (**D**) the 3D printed skull are helpful for surgical planning.

There are two phases that occur in the generation of a viewable CBCT scan.[6] The first is patient scanning phase that creates raw data, and the second phase is the primary reconstruction that produces a viewable image volume. During a CBCT scan, a conventional X-ray source (fixed or rotating anode) and a rigidly coupled detector rotate either 180 or 360 degrees, with the rotation center positioned on the anatomic area of interest. The X-rays are emitted and diverge from the source as a sequence of pulses that are directed to the detector. The divergent X-rays are constrained or collimated to conform to the sensor size and shape or the area of interest. Collimation is usually a rectangular shape but occasionally may be circular, producing a pyramid- or cone-shaped beam. The frame rate is referred to as number of X-ray pulses/second (fms), with each pulse generating a 2D image that corresponds with the rotational geometry. The frame rate may vary by manufacturer and machine version and ranges between 8 and 35 fps. The frame rate multiplied by the length of scan (seconds) equals the number of 2D projections acquired during the rotational scan and is referred to as the raw data. The image detectors are generally indirect flat panel detectors (FPDs). The indirect FPD is composed of an outer scintillator layer (usually cesium iodide) covering a photodiode/transistor layer (usually amorphous silicon) arranged in 2D pixel array. Incident X-rays striking the scintillator produce a light that is converted to an electrical signal by the 2D pixel array that is proportional to the attenuated X-ray intensity. The detector 2D pixel size (individual detector elements) is important for determining resolution and the ability to see fine anatomic detail in the reconstructed volume. Other factors such as scintillator type, focal spot size, and reconstructed voxel size also influence resolution. Reconstructed volumes from FPDs have a cylindrical shape and are composed of volume elements (voxels). The voxel attributes include x, y, z dimensions, a 3D coordinate, and gray scale value proportionate to the attenuation produced by the tissue or space it represents. CBCT voxels are generally isotropic and typically range from 70 to 400 microns per side. Most reconstructed volumes are displayed at the 12 or 14 bits dynamic range; 12 bits is 2^{12} or 4096 shades of gray, while 14 bits is 2^{14} or 16,384 shades of gray. Monitors used to display the images are 8 bits and can display only 256 shades of gray. The image viewing software uses a technique of windowing and leveling to manage the full dynamic range. Windowing allows the image data to be scrolled, thus visualizing 8 bits at a time with airway and soft tissues (low attenuation structures) at one end of the attenuation continuum and bone, teeth, and metal (high attenuation structures) at the other end of the continuum. Leveling allows for contrast and brightness adjustments. Windowing and leveling are used to align the vast dynamic range of the image data with the display monitor and the human visual system capabilities.

It is important to recognize that the nature of CBCT results in the detection of more scattered X-ray photons, which deteriorates contrast in the image. Manufacturers are working to improve contrast resolution of CBCT, but currently available systems achieve poorer contrast resolution than conventional CT does.

Three-dimensional image manipulation

Specialized third-party software viewing tools are available for visualizing the maxillofacial anatomy (see Figs. 6.1D, 6.2C, and 6.3B, C). The specialized software is able to optimize viewing angles to satisfy clinical objectives. Orientation of the scanned anatomy is a recommended first step. Orientation allows for a transformation of the Cartesian coordinate system (applied to the scanned anatomy by the CBCT scanner) to a user-defined orientation that is more favorable for viewing maxillofacial anatomy. The viewing options include volume rendering, shaded iso-surface display, multiplanar, oblique sections, and curved planes. The software allows for custom orientation, spacing, and thickness of the displayed planes.[7]

Three-dimensional printing

Advanced mandibular and maxillofacial reconstruction surgery in veterinary medicine is becoming more common and receiving wider acceptance. However, these difficult cases require special preoperative planning due to the region's complex anatomy. The use of 3D imaging and, more recently, 3D printing as surgical planning modalities for mandibular and maxillofacial surgery in dogs and cats was recently introduced.

The use of 3D imaging following CT or CBCT is the standard of care at our institution and is performed by the attending surgeon (see Fig. 6.3D). Several software programs are available for manipulation of DICOM files created by CT or CBCT for volume rendering and 3D imaging. This is routinely indicated for maxillofacial trauma cases[8] as well as for oral tumor cases with bone involvement. It is also indicated for palatal defects, to compare the size and shape of the osseous defect with the soft tissue defect.[9]

Having a 3D model provides the surgeon with the ability to perform precise preoperative planning, practice a virtual osteotomy, and design a patient-specific implant preoperatively.[10] The 3D printing of the affected skull allows for a tangible understanding of the disorder and more precise surgical treatment. Printing 3D models for precise presurgical planning may reduce surgery time and allow for a reduction in overall surgical costs.

Oral and maxillofacial tumors with bone involvement in difficult locations are indications for 3D printing. The 3D printed skulls allow for precise presurgical planning of the ostectomy sites. They are also excellent tools for client and student education.[10]

Patients with complex mandibular and maxillofacial fractures may also benefit from 3D printing.[8,10] The 3D printed skulls can be used for presurgical planning, plate selection, and prebending of the plates, which reduces anesthetic time. For defect nonunion mandibular fractures, the intact mandible can be mirrored for highly accurate prebending of the plate destined for the affected side.[11]

The authors routinely use 3D printing of skulls prior to mandibulectomy and reconstruction.[11–13] The 3D model of the intact mandibles (prior to mandibulectomy) can be used for ostectomy planning and for prebending the plate for the reconstruction.[10]

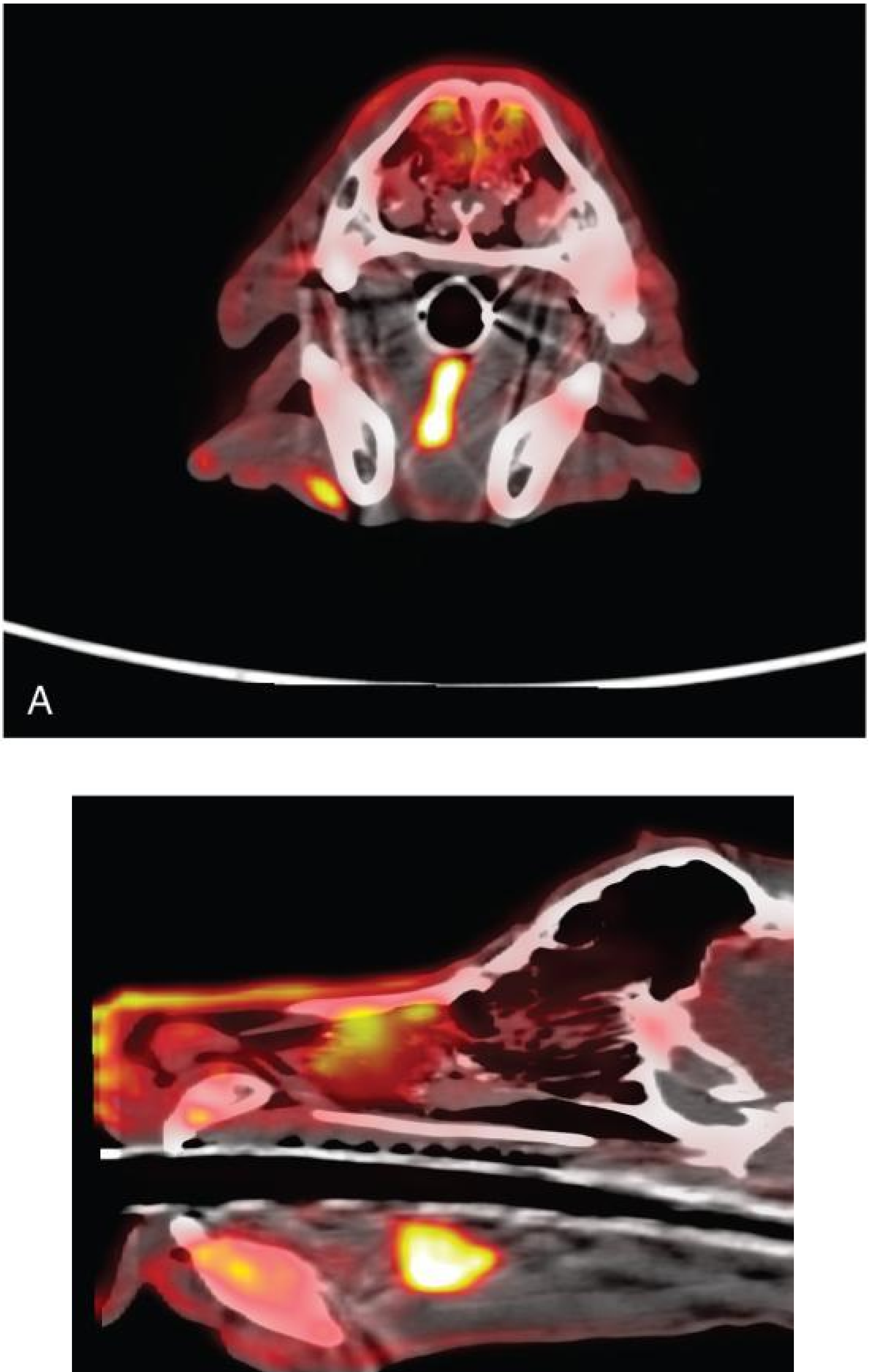
Corrective ostectomies for ankylosis and pseudoankylosis of the temporomandibular joint (TMJ) can be very complex, involving not only the condylar process but also the coronoid process, zygomatic arch, and temporal bone. Precise preoperative planning and practicing a virtual osteotomy is possible with 3D printed models.[10,14]

Magnetic resonance imaging

MRI has become widely available in veterinary medicine and is the gold standard for neuroimaging. MRI provides exceptional soft tissue contrast and is also commonly used in human medicine for imaging the TMJ and other joints. MRI utilizes a strong magnetic field to create an image based on the slight magnetic moment of protons (i.e., hydrogen atoms) and their distribution throughout the body in water and other biomolecules. In addition to the constant strong primary magnetic field, MRI hardware includes gradient coils and a radiofrequency (RF) coil. A carefully timed sequence of gradient and RF coil application, known as a pulse sequence, manipulates the behavior of protons in the magnetic field to generate an image. Multiple different pulse sequences are typically used in the course of imaging a single body region to acquire images with varying contrast and different image planes. Basic pulse sequences include T1-weighted, T2-weighted, and proton density weighted images, each exploiting a different property of tissues related to their composition and microarchitecture. Many other pulse sequences are possible to null signal from fat or low-protein fluid, detect hemorrhage, or even measure water diffusion. The sensitivity of MRI to changes in water content and distribution makes it excellent for detecting inflammation and other soft tissue pathology. Gadolinium-based contrast media can be given intravenously to assess for contrast-enhancing pathology similar to the use of iodinated contrast media for CT. MRI is also uniquely capable of detecting excessive fluid within injured or diseased bone, which may be apparent prior to osseous change visible on CT or radiographs. An MRI study typically takes much longer than CT, and the compromise between spatial resolution, signal:noise ratio, and acquisition time can be limiting when attempting to image small structures in some patients. Nonetheless, MRI is an excellent modality for imaging patients with neurologic deficits, and can be helpful in diagnosing soft tissue or joint pathology.

Positron emission tomography

Positron emission tomography (PET) imaging uses radiopharmaceuticals consisting of a positron emitting radionuclide, typically fluorine-18 (^{18}F), incorporated into a molecule with pharmacodynamic action of biologic interest. The most commonly used PET radiopharmaceuticals are fluorodeoxyglucose (FDG) and sodium fluoride (NaF), which target tissues of increased metabolic activity and increased bone activity, respectively. Many other radiopharmaceuticals are available, primarily used for neurologic, oncologic, or research applications. A radiopharmaceutical accumulates in its target tissue and emits positrons. A positron almost immediately interacts with electrons residing in the tissue, undergoing annihilation and producing two 511 keV gamma rays, which are detected by a ring of gamma-ray detectors. The resulting cross-sectional image represents the relative uptake of the radiopharmaceutical and a map of tissue activity. PET images are extremely sensitive for their intended target but are poorer in spatial resolution than CT, MRI, or ultrasound. CT images are typically acquired immediately before PET imaging, allowing coregistration of PET and CT images to provide improved anatomic localization of tissue activities. PET is frequently used for highly sensitive detection of metastatic disease in patients with known or suspected neoplasia. FDG can also be used to identify sites of inflammation (Fig. 6.4), and NaF is used for detection of bone pathology.


A
B

• **FIG. 6.4** PET-CT scan of the head of a 5-year-old Labrador retriever that exhibited hypersalivation and reluctance to drink water for approximately 1 year with no history of trauma. The (**A**) transverse and (**B**) sagittal images show a midline linear uptake of the soft tissue specific radioisotope (fluorodeoxyglucose) within the midventral tongue. This was confirmed during surgery to be a foreign body.

Ultrasound

Ultrasound uses high-frequency sound waves (typically 2–15 MHz) to create cross-sectional images with contrast based on differences in tissue acoustic impedance. Fluid (especially low protein, acellular fluid) is transmissive of sound waves and appears anechoic or hypoechoic (i.e., black or very dark) in the image. Tissues that reflect sound waves appear hyperechoic (i.e., bright) in the image. Bone and gas are completely reflective of sound waves, appearing as bright lines with acoustic shadows. Ultrasound has very good spatial resolution and soft tissue contrast but is limited by its inability to penetrate bone and gas. In the maxillofacial region, ultrasound is useful for evaluating the regional lymph nodes, salivary glands, and retrobulbar space as well as draining tracts and swelling of unknown origin. Although ultrasound does not penetrate normal bone, changes in the appearance of the bone surface associated with osteomyelitis may be detectable by ultrasound earlier than on radiographs. Ultrasound can also be used to guide needle aspiration or biopsy to accurately obtain a minimally invasive diagnosis.

Protocols

Oral and maxillofacial trauma

CT or CBCT, followed by 3D image manipulation, is the gold standard for evaluation of oral and maxillofacial trauma cases.[8] The diagnostic yield of skull radiographs is much lower but they may be used as an initial screening tool.[15] Dental radiographs are helpful to document concomitant dental trauma. Contrast-enhanced CT or MRI is indicated if brain trauma is suspected. As mentioned before, patients with complex mandibular and maxillofacial fractures may also benefit from 3D printing.[8,10]

Oral and maxillofacial tumors

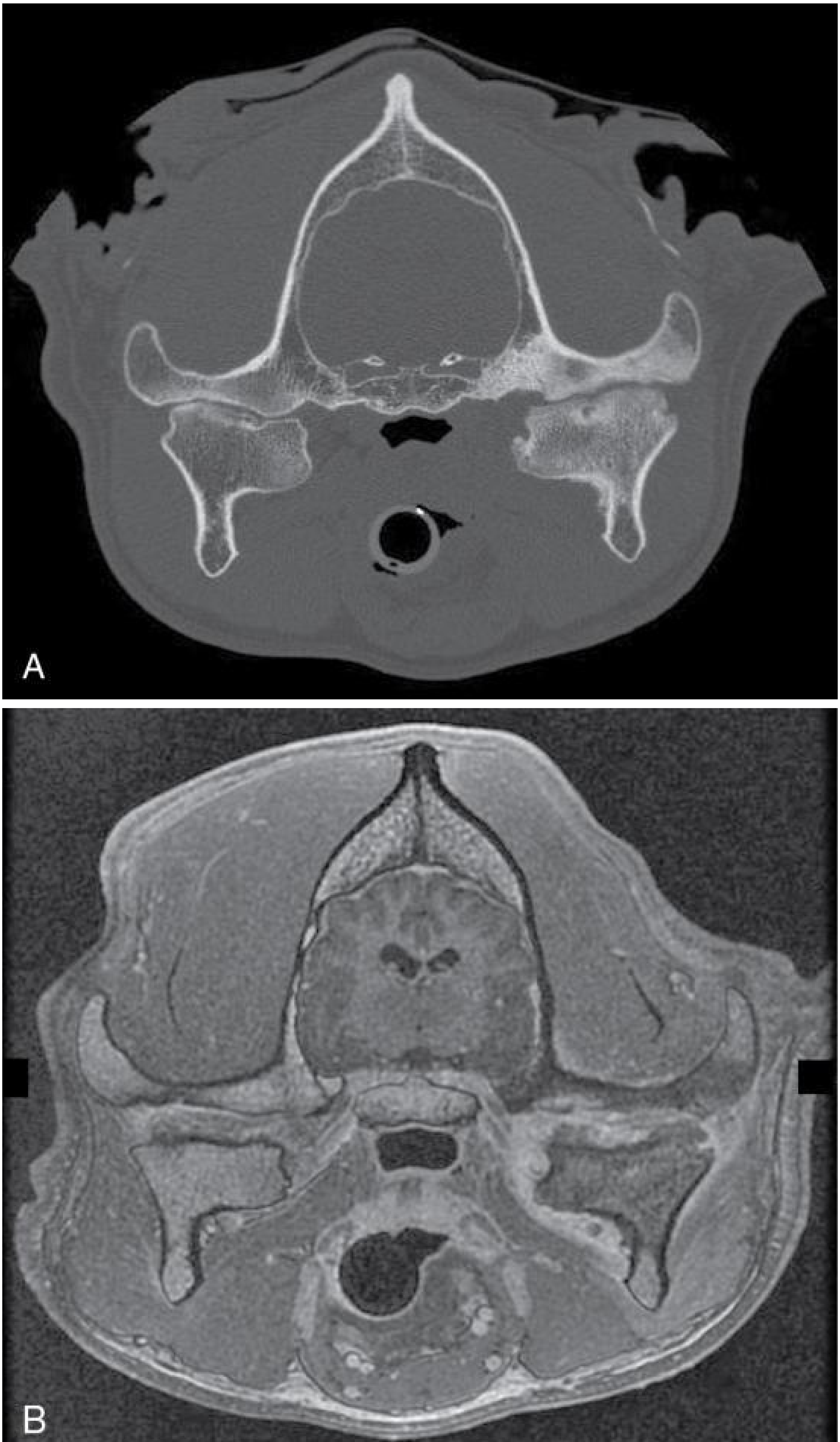
Contrast-enhanced CT is the imaging modality of choice for determining the extent of oral and maxillofacial tumors. The soft tissue component and involvement of bone and adjacent anatomical structures are assessed. The diagnostic imaging findings may also be suggestive of the nature of the tumor (e.g., in case of odontoma or multilobular osteochondrosarcoma). Dental radiographs may be used as a screening tool.[16] Contrast-enhanced CT is routinely used for assessing involvement of the regional lymph nodes and several advanced techniques are under investigation for determining the sentinel lymph node.[17] Ultrasound can be used to guide a fine needle aspirate. The role of diagnostic imaging in clinical oncologic staging is discussed in Chapter 42. Contrast-enhanced CT also plays an important role in radiation treatment planning for oral and maxillofacial tumors.[17]

Palatal defects

In patients with cleft palate and large oronasal fistulas, CT or CBCT followed by 3D image manipulation accurately shows the extent of the bony defect, which typically is larger than the soft tissue defect.[9] It has also been shown that other craniomaxillofacial abnormalities, such as hypoplastic tympanic bullae, are common in dogs with cleft palate,[9] and CT or CBCT imaging can identify these abnormalities as well.

Temporomandibular joint disorders

The objectives of TMJ-disorder imaging are to determine the size, shape, quality, and open and closed spatial relationships of the TMJ components. CBCT or conventional CT is recommended for evaluation of the osseous TMJ components (Fig. 6.5A). Oblique coronal and oblique sagittal serial sections are recommended. The serial sections are oriented to be perpendicular and parallel to the mediolateral long axis of the condylar process. Determination of the orientation angle is achieved by using an axial localizer. MRI can be used to visualize TMJ soft tissues (see Fig. 6.5B).[18] The small size and very thin shape of the canine TMJ disc make specific evaluation of this structure challenging.


A
B

• **FIG. 6.5** (**A**) Computed tomography and (**B**) magnetic resonance image (MRI) of the head of a 7-year-old bull mastiff that was referred for right-sided masticatory muscle atrophy and facial and trigeminal nerve deficits. Note the severe subchondral sclerosis and subchondral lysis of the right temporomandibular joint (TMJ). There is also irregularly margined periosteal reaction surrounding this joint. MRI also demonstrated severe erosive arthritis of the right TMJ, for which the primary differential diagnosis was a septic process with concurrent osteomyelitis. In addition, there was suspected neuritis of the mandibular branch of the trigeminal nerve as a sequela to periarticular inflammation. Secondary atrophy of the right-sided muscles of mastication is also noted.

Maxillofacial soft tissues

An appropriate CT protocol for the evaluation of maxillofacial soft tissues includes acquisition of precontrast images followed by images acquired after intravenous administration of iodinated contrast media. For improved visualization of the palate, oral soft tissues, pharynx, and larynx, the patient should be positioned with its mouth open. The tongue should be positioned away from any known or suspected lesions, and gauze can be used to further separate abutting soft tissues to facilitate evaluation. Precontrast images are often acquired in slices ≤1 mm thick and reconstructed using a fine detail algorithm for the evaluation of the hard tissues. Postcontrast images should be acquired in a thickness of 1–2 mm for large patients and reconstructed using an algorithm appropriate for evaluating soft tissues. Reconstructing the precontrast images with the same slice thickness and algorithm may improve identification of subtle contrast enhancement. Images should be carefully evaluated for symmetry, size, and contrast enhancement of muscles, lymph nodes, salivary glands, and general soft tissues. Bone surfaces should be carefully inspected adjacent to soft tissue masses for potential evidence of bone involvement by neoplastic or infectious lesions. Transverse images are valuable for comparing the right and left sides. For evaluating the retrobulbar space, dorsal plane images are often helpful.[19]

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CHAPTER 7

Instrumentation, aseptic technique, and patient preparation

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Definitions[1-3]

Asepsis: the complete absence of any bacteria, viruses, fungi, molds, or parasites capable of causing infection

Disinfection: the application of a chemical agent to destroy or inhibit the growth of microorganisms on the surface of an instrument or surgical device

Sanitization: the cleansing of an object or area of any dirt or dust, thereby reducing the number of microorganisms present on contaminated surfaces to levels presumed safe by public health standards

Sterilization: the cleansing of an object or instrument of all living microorganisms

Instrumentation

A comprehensive discussion on general surgical and dental instruments and associated basic operative techniques is beyond the scope of this text. Only instruments and aspects of their use that are relevant to oral surgery will be discussed. Extraction instruments are discussed in Chapter 13. In addition, several chapters mention specific instruments used in the techniques described. Personal preference plays an important role in the selection of instruments and this is unavoidably reflected in this and other chapters.

Modified pen grasp

Using a correct instrument grasp can be beneficial in increasing strength and stability, as well as improving hand ergonomics for longevity by preventing musculoskeletal injuries that are common in dentistry. A modified pen grasp is utilized for straight-handled dental instruments including those used for periodontal, endodontic and surgical procedures, and for holding a high-speed handpiece. This grasp allows precise control of the working end, permits a wide range of movement, and facilitates good tactile conduction.[4] The thumb and index finger are placed across from each other, forming a C-shape, and are the only digits that hold the instrument handle. This enables the clinician to roll the instrument when needed. The instrument lies against the side of the middle finger, which creates stability. The ring finger is the fulcrum, which should be placed on a stationary point, such as a nearby tooth. The little finger has no function in the grasp.

Basic oral surgery instruments

Scalpel handles and blades

The most commonly used scalpel handles in oral and maxillofacial surgery are the no. 3, the no. 5, and occasionally the no. 7 handle. Scalpel handle no. 3 is the standard flat scalpel handle used in general surgery, where it typically is held with the fingertip grasp.[5] The flat handle often includes a metric ruler (Fig. 7.1A). In oral surgery it can be held in a pen grasp to make a precise incision.[6] The round scalpel handle no. 5 (Fig. 7.1B) is easier to hold in a modified pen grasp (Fig. 7.2). The round handle allows it to be rotated, which facilitates following the contour of the tooth when making a sulcular incision. This is also possible with the ergonomically designed B3 handle (Fig. 7.1C), also known as the Barron scalpel handle, which has a smooth, tapered octagonal body.[7] The longer, more slender no. 7 handle (Fig. 7.1D) can be used for incisions in deeply situated structures, such as the oropharynx.

• **FIG. 7.1** Scalpel handles used in oral surgery: (**A**) no. 3; (**B**) no. 5; (**C**) no. B3; (**D**) no. 7. Source: (A, C and D courtesy Swann-Morton Limited, Owlerton Green, Sheffield, UK; B courtesy Hu-Friedy Mfg. Inc., Chicago, IL.)

• **FIG. 7.2** A no. 5 scalpel held in the modified pen grasp.

All above-mentioned handles fit the no. 3 blade series, which include blades # 10, # 11, # 15, and # 15c (Fig. 7.3). By far the most commonly used scalpel blade in oral surgery is the # 15.[6] The # 15c is a slender version favored by many periodontists (see Chapter 19).[7,8] Blade # 11 is sharp-pointed, with a straight cutting edge and is used for precision incisions in skin and mucous membrane, such as in cleft lip and palate repair. Blade # 11 can also be used for stab incisions, such as for incising an abscess.[6] The # 10 blade is mainly used, and even then only occasionally, for maxillofacial skin incisions, especially in large dogs. Scalpel blades rapidly become dull during oral surgery, especially when mucoperiosteal incisions are made, and therefore should be replaced as needed in the course of a procedure.

• **FIG. 7.3** Scalpel blades used in oral surgery: (**A**) # 15; (**B**) # 15c; (**C**) # 11; (**D**) # 10. Source: (Courtesy Swann-Morton Limited, Owlerton Green, Sheffield, UK.)

Scissors

Several types of Metzenbaum scissors exist, but the relatively small, curved, blunt-ended version is most commonly used in oral surgery (Fig. 7.4A).[6] Metzenbaum scissors are used for blunt as well as sharp dissection in undermining soft tissue, and for cutting. Iris scissors (Fig. 7.4B) are small, sharp-pointed, straight or curved scissors used for delicate excisions, e.g., for trimming the edges of a mucogingival flap. The Goldman–Fox scissors (Fig. 7.4C) (see Chapter 21) and the double-curved LaGrange scissors (Fig. 7.4D) are variations on the iris scissors that can be used for the same purpose. Scissors incise tissues by a shearing action between the two blades and therefore cause more tissue trauma than scalpel blades. To minimize the crushing of tissues, only sharp scissors should be used. Scissors with tungsten carbide inserts are preferred, as the cutting edges maintain their sharpness longer; instruments with this feature typically have gold-colored handle rings.[9] Scissors are held using the wide-based tripod grasp.[5]

A

B

C

• FIG. 7.4 (**A**) Metzenbaum scissors (curved, blunt); (**B**) Iris scissors (straight); (**C**) Goldman-Fox scissors (curved); (**D**) LaGrange scissors; (**E**) Suture scissors. Source: (Courtesy Hu-Friedy Mfg. Inc., Chicago, IL.)

Scissors used for soft tissue surgery should not be used for cutting sutures.[6] Suture scissors (Fig. 7.4E) that are specifically designed for this purpose or sturdy general surgical scissors should be utilized when cutting suture material.

Tissue forceps

Tissue forceps are hinged instruments that can either be locking or nonlocking. The hinge can either be at the end away from the grasping end, or in the middle, similar to scissors. Tissue forceps that are hinged at the end are also called tissue pliers.

The delicate Adson 1X2 tissue forceps (Fig. 7.5A) is used most commonly in oral surgery for gentle tissue handling, e.g., when stabilizing mucogingival flaps during suturing.[6] The "1X2" refers to the one small sharp point on the one end that fits between the two sharp points on the other end. It causes less tissue trauma compared with the plain Adson forceps and the Adson–Brown forceps, which has multiple teeth. An Adson forceps should be held in a pen grasp rather than the finger grasp to minimize tissue trauma. An Adson forceps is only 120 mm long. If longer forceps are needed, e.g., to reach the caudal part of the oral cavity, a Gerald 1X2 tissue forceps (Fig. 7.5B), which measures 170–180 mm, can be used.[9]

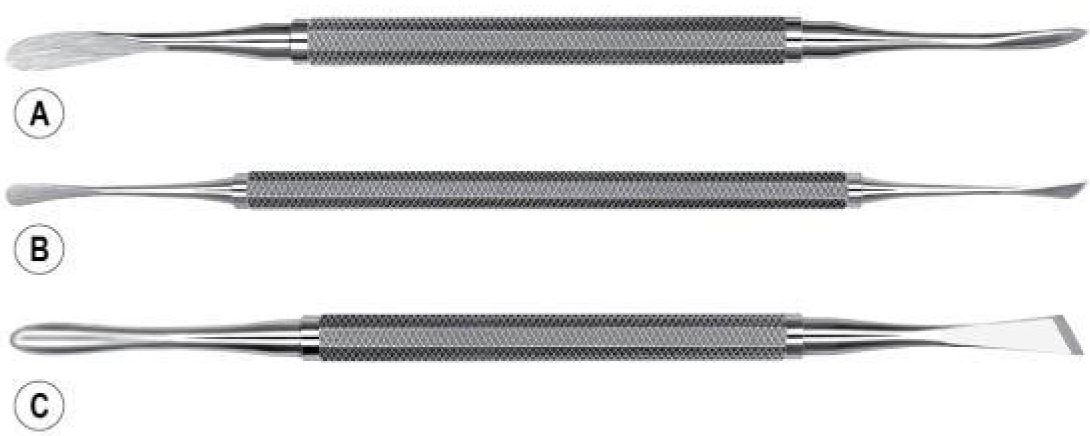
• **FIG. 7.5** (**A**) Adson 1X2 tissue forceps; (**B**) Gerald 1X2 tissue forceps. Source: (Courtesy Hu-Friedy Mfg. Inc., Chicago, IL.)

The Allis tissue forceps (Fig. 7.6) is an example of a locking tissue forceps with a middle hinge. It can only be used on firm tissue that is to be excised, e.g., a large mass of gingival enlargement.[6] It should never be placed on friable tumors, wound edges, or mucogingival flaps because it is very traumatic as a result of its crushing action.

• **FIG. 7.6** Allis tissue forceps. Source: (Courtesy Hu-Friedy Mfg. Inc., Chicago, IL.)

Periosteal elevators

Periosteal elevators (also occasionally referred to as "periosteals") are used to elevate mucoperiosteal flaps.[9] A wide variety of periosteal elevators exists and the choice is often based on personal preference. Many periosteal elevators are double ended. One end is slightly curved and rounded, while the other end typically is pointed or square. The Molt periosteal elevator # 9 (Fig. 7.7A), which is popular both in human and veterinary oral surgery, is a good example. The pointed end of the Molt periosteal elevator is used in a prying motion to elevate the dental papilla between two teeth. However, the pointed end is more applicable to humans than to most animals. In humans and in animals with thick gingiva, the pointed end is gently inserted with the concave side facing the bone in a linear fashion, applying small pushing strokes. In animals with thin or friable gingiva, it is safer to insert the rounded end of the instrument under a small increment of tissue, after which it is gently rotated over 45–90 degrees, lifting the tissue up and sideways. This minimizes the risk of slipping and tearing the gingiva, which can easily occur if the push stroke technique is used. The instrument is held in a modified pen grasp (Fig. 7.8).



• **FIG. 7.7** (**A**) Molt # 9 periosteal elevator; (**B**) # 24G periosteal elevator; (**C**) Mead # 3 periosteal elevator. Source: (Courtesy Hu-Friedy Mfg. Inc., Chicago, IL.)

• **FIG. 7.8** A # 24G periosteal elevator held in the modified pen grasp.

The 24G periosteal elevator (Figs. 7.7B and 7.8) is a delicate instrument, and its rounded end allows mucoperiosteal flaps to be elevated atraumatically, even in very small animals with friable gingiva. The Mead # 3 periosteal elevator (Fig. 7.7C) is a sturdy, double-ended periosteal elevator most suitable for thick attached gingiva and hard palatal mucosa. Its round end is used as described above. The square end is knifelike and is occasionally useful for elevating particularly tightly adhering soft tissues, where it is used with the push stroke technique.

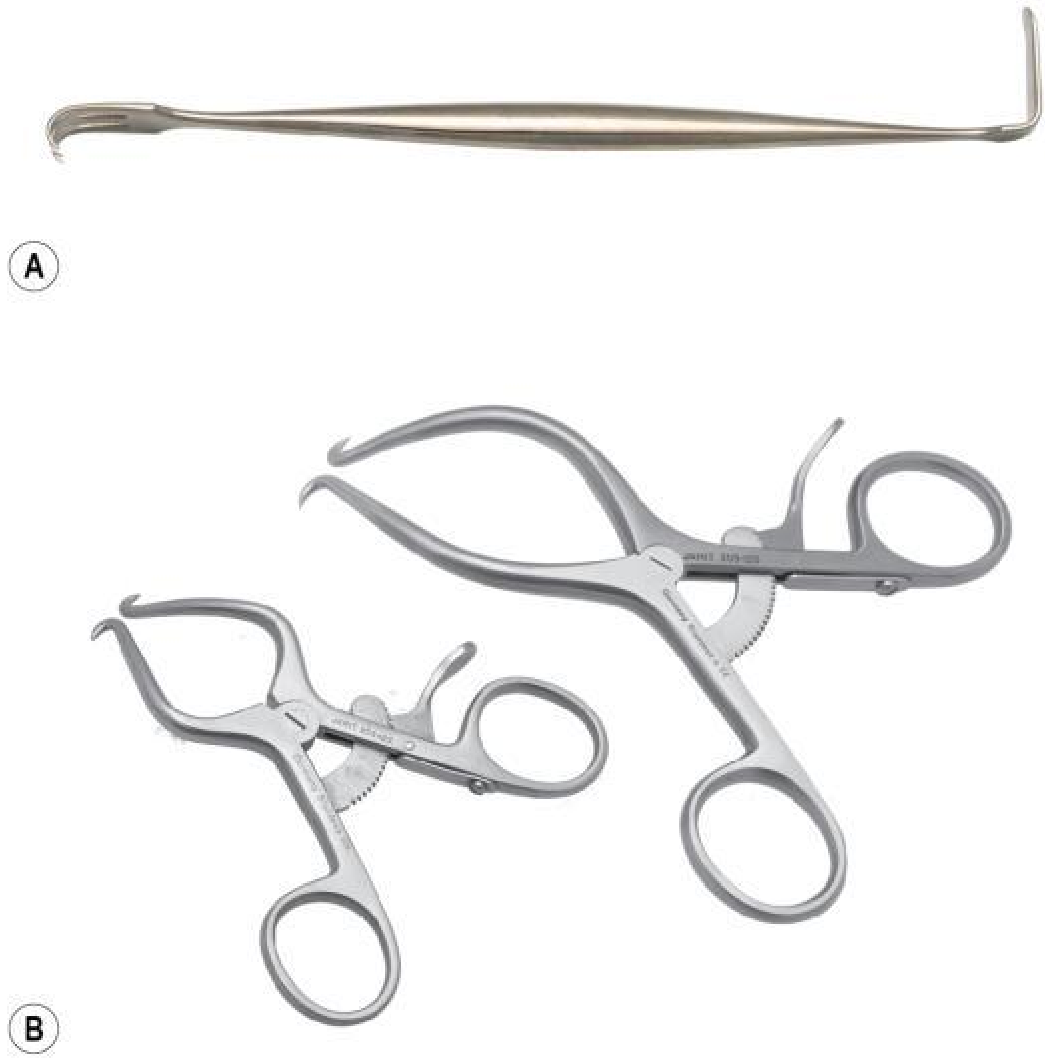
Retractors

Once a mucoperiosteal flap is elevated and bone is exposed, the flap is reflected or retracted in order to protect it during osseous surgery or sectioning of a tooth. The same periosteal elevator used for elevating the flap can be used for this purpose, e.g., a Molt # 9 or # 24G. Periosteal elevators used as retractors are easily scored by burs during this process. If so, these periosteal elevators should no longer be used for lifting flaps but can still be used for retraction. The Seldin retractor (Fig. 7.9A) is an instrument that has evolved from a periosteal elevator to a retractor.[6,9] It is particularly useful for retracting large mucoperiosteal flaps, as in a surgical extraction of a canine tooth.

• **FIG. 7.9** (**A**) Seldin retractor; (**B**) Cawood–Minnesota retractor. Source: (Courtesy Hu-Friedy Mfg. Inc., Chicago, IL.)

Some retractors are specifically made for keeping tongue, lips, and cheeks away from the surgical site. The Cawood–Minnesota retractor (Fig. 7.9B) and the very similar University of Minnesota retractors are typical examples of these. A dental mirror is also occasionally used for this purpose.[6]

During major oral surgery, finger-held retractors, such as the Senn retractor (Fig. 7.10A), are very useful instruments to be held by an assistant. The Senn retractor is a double-ended instrument with three relatively sharp or rounded, bent prongs on the one end and a right-angled finger plate on the other. The sharp-ended version is preferred, as it less likely to slip and is therefore less traumatic.[2] Stay sutures and skin hooks are also commonly used in maxillofacial surgery. If no assistant is available, a self-retaining retractor such as a pediatric Gelpi perineal retractor (Fig. 7.10B) can be used, especially when the surgical site is relatively deep and the access small, such as in a condylectomy of the temporomandibular joint.



• **FIG. 7.10** (**A**) Senn retractor. (**B**) A 89-mm *(left)* and a 140-mm *(right)* pediatric Gelpi perineal retractor.

Needle holders

A needle holder has a short, relatively stout beak, usually with serrated faces, and a locking handle. Fine suture material with small swaged-on needles is generally used in oral surgery (see Chapter 8). The needle holders indicated in oral surgery are therefore delicate, to match the size of the needles. The Halsey needle holder (Fig. 7.11A) is a very versatile needle holder well suited for most intraoral procedures. This needle holder is 130 mm in length. Occasionally, a slightly longer instrument is needed, especially for surgery in the caudal oral cavity of large dogs, and a DeBakey needle holder (Fig. 7.11B), which measures 180 mm but still has a relatively slender beak, would be a good choice. These needle holders are all held with the wide-based tripod grasp (Fig. 7.12). The needle should be grasped at two-thirds of its length away from the tip of the needle.[5,6]

A

B

• FIG. 7.11 (**A**) Halsey needle holder; (**B**) DeBakey needle holder; (**C**) Castroviejo needle holder; (**D**) Olsen-Hegar needle holder. Source: (Courtesy Hu-Friedy Mfg. Inc., Chicago, IL.)

• **FIG. 7.12** A Halsey needle holder held in the wide-based tripod grasp.

In periodontal surgery a Castroviejo needle holder (Fig. 7.11C) is occasionally used.[8] This is a very delicate instrument that can only be used with very small needles. It is held with a pen grasp.[5]

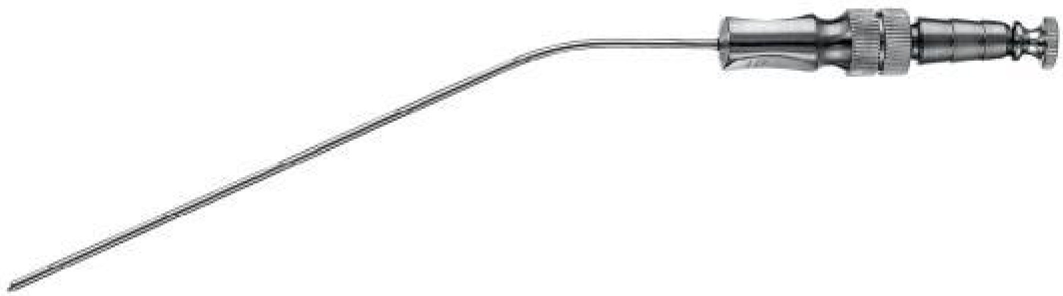
Some surgeons working without assistants prefer a needle holder with built-in scissors such as the Olsen–Hegar needle holder (Fig. 7.11D).

It should be noted that needle holders cannot be used for wire twisting as this would damage the gripping surface; specifically designed ligature twisters should be used for this purpose.[9] Conversely, hemostats are not strong enough to be used as needle holders, and needles can cause permanent damage to these instruments.

Ancillary instruments

Suction tips

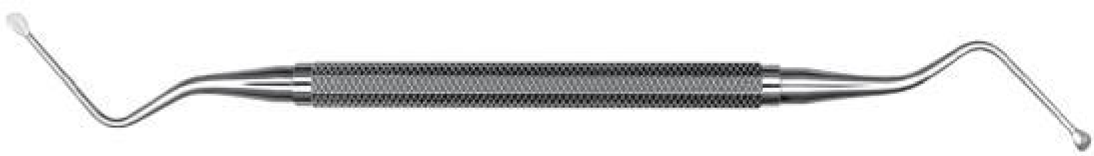
As hemorrhage may be brisk and copious irrigating fluids may be used in oral and maxillofacial surgery, it is imperative to have a well-functioning suctioning device. The Frazier suction tip (typically a # 10) (Fig. 7.13) is the suction tip of choice in oral surgery.[6] It is small and precise and has a decompression opening at the end of the handle. Maximum suction is achieved when the opening is occluded by the index finger of the operator. Suction can be reduced by lifting the finger, which is indicated when working in the vicinity of anatomical structures that could be damaged by the suction. In addition, the Frazier tip comes with a wire stylet that is used for clearing the tip, should it become blocked.



• **FIG. 7.13** Frazier # 10 suction tip. Source: (Courtesy Hu-Friedy Mfg. Inc., Chicago, IL.)

Surgical curette

A small surgical curette is used for removing unwanted soft tissue and debris from a bony surface or defect, such as gently debriding an extraction wound if deemed necessary, or removing soft tissue present between two bone fragments in a longstanding fracture. A Miller curette (Fig. 7.14) is an example of a double-ended, spoon-shaped curette that is very versatile.



• **FIG. 7.14** Miller # 10 surgical curette. Source: (Courtesy Hu-Friedy Mfg. Inc., Chicago, IL.)

Surgical marker pen

A sterile surgical marker pen (Fig. 7.15) is a small but important tool in major oral and maxillofacial surgery. It is used for planning skin or oral mucosa flaps for repair of an oronasal fistula or cleft lip and palate. It is also used for outlining the surgical margins in tumor excision. The use of the marker pen helps the surgeon to plan the incisions more carefully and before tissues are distorted once the first incision is made.

• **FIG. 7.15** Sterile surgical marker pen with ruler and labels. Source: (Courtesy Aspen Surgical Products, Caledonia, MI.)

Instrument organization

It is practical to have standard sets of instruments for various procedures. Open tray systems are commonly used in veterinary dentistry but are incompatible with the aseptic technique desirable in oral surgery. Instrument cassettes greatly facilitate the organization, cleaning, sterilization, and storage of instruments (Fig. 7.16). Cassettes are made from plastic or stainless steel and come in various sizes. Instruments are cleaned, autoclaved, and stored in closed cassettes, which are then placed chairside on a sterile field and opened ready for use. Each cassette should contain a selection of instruments that are likely to be used in a given procedure. Certain instruments are used in essentially every oral and maxillofacial surgery procedure, e.g., scalpel handle, needle holder, Metzenbaum scissors, suture scissors, periosteal elevator, Adson tissue forceps, etc. It is practical to group these in a "basic oral surgery" cassette and have a relatively large number of these available (Table 7.1). In addition, there are cassettes for exodontics (see Chapter 13), periodontal surgery, apicoectomy, etc. Infrequently used instruments can be individually packed in standard autoclavable pouches and stored in an organized fashion to be readily available if needed.

• **FIG. 7.16** An autoclaved instrument cassette in a sterilization bag *(left)* and an unwrapped cassette with the lid removed *(right)* containing a basic oral surgery instrument set (contents are listed in Table 7.1).

TABLE 7.1
UC Davis Basic Oral Surgery Instrument Set

Description	Hu-Friedy (Hu-Friedy Mfg. Inc., Chicago, IL) * Item Number
IMS resin cassette (18 instruments) or Large Signature Series 20 instrument cassette	IMS-1118 IM4209
Adson tissue forceps 1X2	TP5042
Backhaus towel clamps—small (6)	TC3
Cawood-Minnesota retractor	CRM2
Curved baby Allis forceps (2)	TFB
Halsey needle holder	NH5036
Hartman hemostat straight (2)	HHS
Mead periosteal elevator # 3	P3
Metzenbaum scissors—small	S5055
Miller curette # 10	CM10
Periosteal elevator # 24G	P24G
Scalpel handle # 5	10-130-05
Seldin retractor # 23	P23
Suture scissors	S13

*Many other companies have some or all of these or similar instruments.

Power equipment

Rotary instruments

Rotary instruments are used in oral and maxillofacial surgery for sectioning teeth, removing and smoothing alveolar bone, cutting bone, and drilling into bone.

A high-speed handpiece with a tapered diamond bur or a carbide crosscut fissure bur is indicated for sectioning multirooted teeth into single-rooted units (see Chapter 13). An autoclavable straight surgical handpiece, with built-in sterile saline or lactated Ringer solution irrigation and a round carbide or diamond bur, is the instrument of choice for bone removal (Fig. 7.17A).[6,10] The maximum speed of these oral surgery units is 40,000 rpm and the torque is adjustable. A high-speed handpiece with a round diamond or carbide bur is generally used in veterinary dentistry for removing and smoothing bone.[11] This practice is frowned upon in human oral surgery, as the air exhausted from this type of handpiece may be forced into deeper tissue planes and produce tissue emphysema.[6] Emphysematous complications have been documented in humans but not in animals.[12] In addition, most dental units to which the high-speed handpieces are connected use regular tap water or deionized water as irrigation fluid and the tubing is not sterilizable. It has been shown that tap water is harmful to canine fibroblasts in vitro.[13] The microorganisms that may be present in tap water and the biofilm that may form in the waterline can result in a contaminated irrigation fluid, which is cause for concern.[14,15]

A

B

• **FIG. 7.17** (**A**) INTRAsurge 500 oral surgery unit and handpiece (KaVo Dental Corporation, Lake Zurich, IL); (**B**) Lindemann osteotomy bur. Source: (Hu-Friedy Mfg. Inc., Chicago, IL.)

A piezoelectric surgical unit (see Chapter 9) with bone cutting inserts is ideally suited for osteotomy and ostectomy (e.g., mandibulectomy and maxillectomy). A good alternative is an oral surgery unit with either a straight or contra-angle handpiece combined with a Lindemann osteotomy bur (Hu-Friedy Mfg. Inc., Chicago, IL) (Fig. 7.17B).[16] While an oscillating or reciprocating orthopedic saw can be used for this purpose, turbinate damage and transection of the neurovascular bundle in the mandibular canal are almost impossible to avoid with these instruments, and their use cannot be recommended.

Electrosurgery and radiosurgery

Electrosurgery and radiosurgery can be used instead of the scalpel to cut soft tissues, with the goal of reducing hemorrhage.[5] However, both methods have been shown to cause lateral thermal damage to skin, compared with the scalpel.[17] Radiosurgery is associated with less damage than electrosurgery.[17] Scalpel incision of oral mucosa results in faster reepithelialization and greater tissue strength than electrosurgery incisions.[18] When electrosurgery is used to incise oral soft tissues in the vicinity of bone, gingival recession, bone necrosis, and loss of bone height may occur.[19,20] The same occurs when electrosurgery is used in the vicinity of teeth, in addition to trauma to the cementum.[21]

Electrosurgery is not discussed in a recent leading human oral surgery textbook, which is consistent with its infrequent use.[6] There is some limited application in human periodontal surgery, namely for superficial procedures at a safe distance from bone and teeth, e.g., the excision of gingival enlargement and frenoplasty.[22]

There are veterinary practices where electrosurgery and radiosurgery are used.[17] The disadvantages of electrosurgery and radiosurgery with regard to delayed wound healing and hard tissue damage would seem to outweigh the advantage of better hemostasis when used in the oral cavity. A possible indication would be gingivectomy/gingivoplasty in an animal with impaired hemostasis (see Chapter 20). The technical details pertaining to the kind and type of waveform, shape and size of electrode, and speed of the electrode through the tissue should be fully understood if these methods are selected for making incisions.[5,23]

Magnification loupes and headlight

It has become common practice to use magnification loupes in daily clinical practice, not just during surgery but in all aspects of dentistry. Loupes are recommended to those in training, who are encouraged to continue using loupes throughout their careers to help with both ergonomics and enhanced vision. Magnification varies between 2.5 × to 6 ×, and companies offer multiple styles and types of frames and lenses. A headlight can be attached to the frame and helps with increased illumination. The lights are available in a variety of brightness levels and battery options and are either wired or wireless.

Aseptic technique

Aseptic technique in dentistry

Sanitation and proper sterilization techniques have been an integral part of human dental practice for many years, but since the 1980s a number of disease-causing organisms, such as hepatitis B virus and HIV, have made these techniques even more important.[24] Dentists performing oral surgery must adhere to the principles of aseptic techniques for two reasons. First, during most oral surgical procedures, the dentist, assistants, and equipment become contaminated with the patient's blood and saliva, which may contain pathogenic microorganisms. Second, to perform surgery, the dentist must penetrate an epithelial surface, the most important barrier the patient has against infection.[1] An opportunistic portal is thereby created through which a pathogen may enter with sufficient strength and numbers to cause infection.[25]

The use of infection-control techniques, such as barrier precautions and sterilization practices, has been recommended by the American Dental Association (ADA) to reduce exposure and eliminate transmission of pathogenic microorganisms.[26,27] The implementation of many of these techniques by dental professionals is now required by law by the US Occupational Safety and Health Administration (OSHA) as the standard for infection control for dental practice.[26,27] These same techniques should also be considered as recommendations for veterinary practices that provide oral and maxillofacial surgical services.

Surgical infection

Adherence to surgical aseptic technique is crucial to the success of any surgical procedure.[2] Microbial contamination of the surgical site is considered to be a necessary precursor of surgical infections.[28] Based on a review of data from the Centers for Disease Control and Prevention (CDC), surgical infections are the third most frequently reported nosocomial infection among hospitalized patients.[29] Several studies have demonstrated that the overall infection rate of surgical sites of patients in veterinary hospitals is similar to that in human studies.[30,31]

Surgical infections not only hinder the patient's recovery but also cause additional emotional stress as well as financial cost to the client and the attending veterinarian.[31] To ensure that the incidence of postsurgical infections remains as low as possible, it is fundamental that all members of the surgical team perform their duties in a way that minimizes any chance of infection.

Surgical aseptic technique is required to maintain a surgical site as free as possible of all living pathogenic microorganisms.[1,3] The definition of the word *asepsis* does, in fact, mean the complete absence of any bacteria, viruses, fungi, molds, or parasites capable of causing infection.[1,3] The major locations that harbor these microorganisms are the patient, the surgical environment, the surgical materials and instruments, and the operating surgeon and assisting personnel.[3,32] Basic steps of surgical aseptic technique to reduce the risk of infection include: proper preparation of the patient and the surgical personnel, sterilization of all surgical instruments and materials, and maintenance of the surgical room environment.[3]

Achieving asepsis

There are three levels of care used to initiate and maintain an aseptic surgical field: sanitization, disinfection, and sterilization.[24,33] The level of care selected should be based upon the category of each item's intended use.[24] Instruments or equipment that come into contact only with intact skin, such as the headrest and radiographic tube, are classified as noncritical because these surfaces have a low risk of transmitting infection. These items may be cleaned or sanitized with intermediate-level or low-level disinfection, or detergent-and-water washing, depending on the nature of the surface and the degree and nature of the contamination.[25,34]

Instruments, such as mirrors, bite blocks, and cheek retractors, that do not penetrate soft tissue or bone but contact oral tissues or mucous membranes are classified as semi-critical.[25] These devices should be sterilized after each use. If, however, an instrument cannot be sterilized because it may be damaged by high heat, then it should receive, at minimum, high-level disinfection.[25,34]

Surgical and other instruments used to penetrate soft tissue or bone are classified as critical, and should always be sterilized after each use. These items include dental instruments such as forceps, scalpels, bone chisels, scalers, and surgical burs.[25,34]

Sanitization

Sanitization denotes cleansing an object or area of any dirt or dust, thereby reducing the number of microorganisms present on contaminated surfaces to levels presumed safe by public health standards.[1] Sanitization is normally accomplished with the use of water, mechanical action, and detergents or enzymatic products.[35] Thorough cleaning must also precede additional disinfection and sterilization procedures. Failure to remove foreign matter or soil from an object before a disinfection or sterilization process is likely to render the process ineffective.[35] Sanitization is the method used to maintain noncritical instruments and equipment such as the surgical room and table, dental unit, and radiography machine.[25,34]

Disinfection

Disinfection refers to the application of a chemical agent to destroy or inhibit the growth of microorganisms on the surface of an instrument or surgical device. These chemical agents are known as disinfectants and antiseptics.[1] Disinfectants are used to kill microorganisms on inanimate objects that are either too big to be sterilized or cannot be exposed to heat.[1] Antiseptics are applied to kill microorganisms on living tissue, such as the hands and arms of the surgical team or the patient's surgical site. It should be understood that disinfection will not necessarily kill all microorganisms, such as bacterial endospores, but it can significantly reduce their level of contamination.[1]

There are three levels of disinfection: low, intermediate, and high.[24] Low-level disinfection is the least effective disinfection process. It does not kill bacterial endospores or *Mycobacterium tuberculosis* var. *bovis*, a laboratory test microorganism that is used to classify the strength of disinfectant chemicals. But it can kill most bacteria, some viruses, and some fungi. Intermediate-level disinfection is a disinfection process that does kill the *M. tuberculosis* var. *bovis*, but not bacterial endospores.[24] It also kills vegetative bacteria, most viruses, and most fungi.[35] High-level disinfection is a disinfection process that kills some, but not necessarily all, bacterial endospores. This process does, however, kill *M. tuberculosis* var. *bovis*, as well as other bacteria, fungi, and viruses.[24]

Noncritical items, such as countertops and surgical table surfaces, may be cleaned with low-level disinfection by using water and either soap or a disinfectant product registered by the Environment Protection Agency (EPA) for cleaning.[24,34,35] If noncritical items are contaminated with blood, the visible soil should be removed and intermediate-level disinfection applied with a liquid chemical germicide registered with the EPA as a tuberculocidal 'hospital disinfectant' or a fresh solution of sodium hypochlorite (bleach) mixed with water. A ratio of 1/4 (minimum) to 3/4 (maximum) cup of bleach to 1 gallon of water will provide a solution that is strong enough to disinfect.[24]

Ethyl alcohol and isopropyl alcohol are not high-level disinfectants because of their inability to inactivate bacterial spores.[35] Alcohol is not recommended for disinfecting contaminated surfaces because it evaporates quickly and does not allow sufficient contact time for effective antimicrobial action.[24]

To disinfect semi-critical items that may be damaged by heat sterilization, high-level disinfection may be performed by first cleaning the item of visible soil and then soaking the item in a liquid chemical germicide registered with the EPA as a "disinfectant/sterilant" and cleared by the Food and Drug Administration (FDA) for use in sterilizing or disinfecting medical and dental instruments.[24] The FDA requires high-level disinfectants to prove 100% kill of *M. tuberculosis* var. *bovis*.[24] These chemical germicides include 2% glutaraldehyde-based formulations, 6% stabilized hydrogen peroxide, and peracetic acid.[35] Manufacturer instructions for these products must be strictly followed. Attention should be paid to temperature and dilution requirements, contact time, antimicrobial activity spectrum, and reuse life.[26]

Sterilization

Sterilization is the only method proven to rid an object or instrument of all living microorganisms, including highly resistant bacterial endospores.[1,24] It is accomplished by either physical or chemical processes.[35] Steam or chemical vapor under pressure, dry heat, and low-temperature sterilization processes (ethylene oxide gas, plasma sterilization) are the principal sterilizing agents used.[35]

It is crucial that instruments or objects entering any sterile body tissue or the vascular system or breaking the patient's mucosal barrier be kept sterile.[35] Most items should either be purchased as sterile, such as surgical blades and suture materials, or should be heat sterilized.[34,35] If the object is heat sensitive, it may be treated with ethylene oxide gas or other low-temperature sterilization processes.[35]

The ADA and CDC recommend that all instruments classified as critical and semi-critical be sterilized after each use.[34,35] Chemical disinfection is not recommended for any critical or semi-critical dental instrument that can be safely heat sterilized.[34,35] Known as "cold sterilization," immersion of instruments in a chemical sterilant solution is not recommended because the solutions cannot be monitored biologically.[24] In order to remain sterile, instruments sterilized by chemical solutions must be handled aseptically, rinsed in sterile water, dried with sterile towels, and stored in a sterile container.[24,34]

Use of sterilizers

Sterilizers

The most common (and one of the most reliable) means of sterilizing dental and surgical instruments is the utilization of a gravity displacement type of steam sterilizer or autoclave.[1,3,34] An autoclave admits steam into the instrument chamber while forcing cooler air out of an escape valve. When this valve closes, pressure is slowly increased inside the chamber, causing the temperature to rise, killing bacterial spores and heat-resistant viruses.[33] An autoclave is a safe, quick, and relatively inexpensive way to sterilize surgical instruments.[36] However, moist heat tends to dull and rust instrument cutting edges. Hand instruments and burs made of carbon steel will corrode. Use of a rust inhibitor is advised to prevent corrosion.[26]

The operating manual provided with an autoclave should be read and followed to ensure its proper use and working condition. Heat and steam must be able to penetrate the innermost layer of each pack in the allotted time. The usual recommended time of 15–30 minutes at 121.5°C at 1 bar (15 psi) is needed to ensure sterility.[32]

Another type of steam sterilizer is known as a high-speed prevacuum sterilizer. This newer design of autoclave is fitted with a pump which creates a vacuum to remove all air within the instrument chamber before steam enters and is pressurized. Compared to gravity displacement, a prevacuum sterilizer allows faster and more thorough steam penetration throughout the entire load of instruments.[34] The average time would be 4–5 minutes at 132°C.

Sterilization may also be achieved by using an unsaturated chemical vapor system, sometimes referred to as a chemiclave, which involves heating a chemical solution of primarily alcohol with 0.23% formaldehyde in a closed pressurized chamber.[34] This sterilizer is commonly operated for 20 minutes at 132°C at 20–40 psi.

Dry heat sterilizers may be used for materials that might be damaged or corroded by moist heat. However, more time and higher temperatures are generally required to achieve sterility and they may not be as practical for office use.[34] A typical cycle would run for 60 minutes at 160°C to achieve sterility.

The ADA recommends that all handpieces, contra angles, sonic and ultrasonic tips, reusable prophy angles, saliva ejectors, vacuum tips, and air/water syringes be heat sterilized by an autoclave between each patient use.[25-27] Internal surfaces of these devices may become contaminated with patient debris during use. This debris could be expelled into a surgical site during subsequent use in other patients. Surface disinfection by wiping or soaking in liquid chemical germicides is not an acceptable method for completely sterilizing these items.[25] High-speed and low-speed handpieces are manufactured to withstand autoclave sterilization.[25] Manufacturer's instructions for cleaning, lubrication, and sterilization of all items should be followed.

Heat sterilization may be inappropriate for plastics, rubber, and acrylic resin materials with low melting points, as well as for powders and anhydrous oils.[26,36] Alternative sterilizing methods such as ethylene oxide gas should be considered for these items, or suitable disposables could be purchased.[26] Single-use disposable items should be used for one patient only and discarded. These items, such as prophy angles, rubber cups, and saliva ejectors, are neither designed nor intended to be cleaned, disinfected, or sterilized for reuse.[25,34]

Instrument preparation and packs

To prepare instruments for the sterilizer, they should be free of any organic material, oil, or greasy films. An ultrasonic cleaning bath is ideal for this type of preparation.[25,26,34] However, special solutions should be used inside this device to enhance its cleaning activity. These solutions need to be changed daily and instruments completely rinsed off before packing. Instruments may also be soaked in hot water then scrubbed with detergent and rinsed.[26] Heavy-duty utility gloves should be worn to prevent accidental hand injury by sharp instrument edges.[25,26,34] Hinged instruments should be left open and unlocked.[34,37] The instruments may be arranged into cassettes, which are then placed in sterilization bags (see Fig. 7.16) or covered with steam-permeable wrappers and sealed with tape. Sharp tips of hand instruments should be wrapped with gauze to prevent them from perforating bags during processing. Wrapping items before sterilization will prevent air contamination during their storage after processing.[38] Packs should be loosely but evenly distributed within the sterilization chamber to allow circulation of heat and moisture.[37]

Monitoring sterility

There are many internal and external indicator systems available to monitor the effectiveness of autoclave cycles.[3] Chemical indicator strips placed inside packs of instruments will indicate whether steam has penetrated. Sterilization bags and autoclave tape with built-in indicator markings will change color if the proper temperature was reached. However, they do not register how long the temperature was properly maintained.[25,36]

The most reliable method of testing sterilizer cycles is the use of a biological indicator or spore system. Microbiological monitoring is recommended at least once a week with a commercial preparation of *Bacillus stearothermophilus* spores.[34,36] When placed inside the chamber alongside the instrument packs, spores will be killed if the sterilizer is working properly and used correctly. If the spores are not killed, the sterilizer should be checked for proper function, such as temperature, length of cycle, and loading.[36] If subsequent spore tests remain positive, discontinue the use of the sterilizer until it is properly serviced.[34,36]

Storage

Continued sterility of the instruments must be maintained after removal from the sterilizer through adequate handling and storage. After completion of the sterilization cycle, packs should be thoroughly cooled before being handled. The packs should then be inspected to ensure the integrity of the wrappers and bags. Items that have been sterilized properly do not need an expiration date. Sterility is guaranteed until the package is damaged or opened.[37] However, packages may be marked with the date of sterilization to facilitate retrieval of processed items in the event of a sterilization failure.[34] Sterile packages should be stored in a protected environment, such as a closed cabinet, which minimizes damage and handling by personnel. There should be enough space between the packages to prevent crushing and bending.[37]

Routine maintenance of the surgical area

The effective maintenance of a sanitary surgical environment depends on the daily repetition of cleaning and disinfecting procedures. Surfaces such as surgical tables, stands, and floors should be cleaned as debris and spills occur, and after every surgical procedure.[36] Although these surfaces are rarely associated with the transmission of nosocomial infections, it is important to reestablish and maintain a clean environment for the next surgical treatment.[28,32,36]

Cleaning procedures

Floors of the dental surgery room should be vacuumed and then wet mopped using a clean mop head and low-level disinfectant, such as a quaternary ammonium compound.[28] An alternative method to mopping would be spraying disinfectant over the floor and wet vacuuming the excess.[38] Low-level disinfectants may also be applied with clean paper towels to other noncritical areas not directly contaminated with patient debris, such as walls, blinds, and countertops.[25] Surfaces soiled with patient debris or splatter should be disinfected with an intermediate-level disinfectant, such as phenols, iodophors, and chlorine-containing compounds.[25]

Another option to cleaning and disinfecting would be to cover surfaces such as countertops and surgical table tops, dental units, switches, light handles, and radiography unit heads with an impenetrable cover such as plastic, plastic-backed paper, or other commercially available disposable barrier products.[26,34] These covers should be changed between each patient, and the surfaces disinfected at the end of the day.[24,35]

Waterlines

Since 1990, dental unit waterline contamination has been reported and debated among dental professionals. It has been shown that microbial biofilm, a plaque-like substance, forms along the walls of long, narrow-bore dental unit tubing that furnishes cooling and irrigating water to high-speed power instruments and water syringes.[15,34,39] This biofilm then becomes a source of free-floating microorganisms that are released into a patient's oral cavity and tissues.[15,34]

Microorganisms within the biofilm often originate from the public water supply. It is also possible that patient fluids may be retracted into the waterline during treatment, and may exit later into another patient's oral cavity or surgical site. To date, however, scientific evidence indicates there is little risk of significant adverse health effects due to contact with water from a dental unit.[34,40] Furthermore, most dental units on the market today are engineered to prevent retraction of oral fluids.

The CDC and ADA recommend thorough flushing of the dental unit waterlines for a minimum of two minutes at the beginning of each clinic day and for 20–30 seconds after treatment of each patient.[25,34,41] This assists in flushing out patient material that may have entered the lines and may reduce the accumulated loose microbial buildup. However, it does not appear to reduce the adherent biofilm.[34] The CDC and ADA suggest further preventive strategies such as separate water reservoirs independent of the public water supply, chemical disinfection of the water tubing, daily drainage and air purging of the waterlines, avoidance of heating dental unit water, and installation of waterline filters.[34,39,41] As an additional precaution, sterile saline or sterile water should be used as a coolant and irrigant when surgical procedures involving flushing open vascular sites and cutting of bone are performed.[24,25,34,39] Delivery of sterile water can be accomplished with the use of a sterile bulb syringe for surgical site irrigation, and a sterile handpiece with sterilizable dental unit tubing.[34]

Preparation of the surgical team

Guidelines for proper hand scrubbing techniques and surgical attire for operating personnel have been addressed in detail in numerous references.[3,32,42] It is not in the scope of this text to recount these guidelines; however, a few recommendations will be made regarding oral and maxillofacial surgical procedures.

Hand hygiene is the single most important procedure for minimizing microbial contamination and preventing nosocomial infections.[34,36,43,44] This can be accomplished with either handwashing, hand antisepsis, or surgical hand antisepsis, depending on the type of procedure, degree of contamination, and the desired persistence of antimicrobial action on the skin.[44] Handwashing means washing hands with plain, non-antimicrobial soap and water.

Hand antisepsis can be achieved by washing hands with an antimicrobial soap or rubbing hands with an alcohol-based hand rub, also known as a waterless antiseptic agent. The characteristics of a good antimicrobial soap are broad-spectrum, fast-acting, nonirritating, and designed for frequent use. Chlorhexidine gluconate and povidone-iodine are examples of commonly used antiseptic hand products. Vigorously rubbing hands together for 10–15 seconds with 3–5 mL of the product with water will remove dirt and debris and allow sufficient contact time for the antimicrobial agent to work.[42] Hands should be rinsed thoroughly and dried with disposable paper towels.[1,43] Alcohol-based hand rubs, containing 60%–95% ethanol or isopropanol, may be used if hands are not soiled.[34,44] Hands should be rubbed together with an adequate amount of product for 10–15 seconds until dry.

Surgical hand and arm antisepsis should be performed for maxillofacial surgery and other procedures requiring the use of an operating room.[1] A minimum of 2 minutes is needed to properly cleanse the hands and forearms with antimicrobial soap and brushes.[34,42,43] Fingernails should be cleaned as well, using a pick.[1,25] An alternative method to achieve surgical antisepsis would be to prewash hands and forearms thoroughly with a non-antimicrobial soap and completely dry. Follow with an application of an alcohol-based surgical hand scrub product and allow hands and forearms to dry before donning sterile gloves.[34,44] Alcohol-based hand rubs have been found to be rapidly germicidal when applied to the skin but these rubs should include antiseptics to achieve persistent antimicrobial action.[34,44]

Clean nonsterile gloves are considered acceptable for simple (closed, nonsurgical) extractions, although sterile gloves are recommended.[1,45-47] Sterile gloves are required for surgical extractions and major oral and maxillofacial surgery.[1,34] When gloves are torn, cut, or punctured, they should be removed as soon as possible.[34] Hands should be washed thoroughly again and covered with a new set of gloves to complete the surgical procedure.[25] If using an alcohol-based hand rub, hands should be thoroughly dried before gloving, because hands still wet with this product can increase the risk of glove perforation.[34] Although gloves provide a protective barrier to microbes, they are not completely impermeable. Therefore hands should be thoroughly washed again immediately upon removal of gloves.[26,34]

Face masks and safety glasses, or chin-length face shields, are recommended to protect personnel from aerosolized blood, saliva, and potential pathogens, especially during the use of power or high-speed cutting instruments.[25,26,34,43] Head caps are required to cover hair which may shed and fall into the surgical site. Shoe covers and sterile gowns should be added for operating room procedures.

Preparation of the patient

The prevention of infection during surgery is the primary objective of modern aseptic surgery.[3] An animal's own microbial flora is the most common source of bacteria contaminating surgical wounds.[3] Therefore, preoperative use of topical antimicrobial agents and sterile drapes are important infection control measures. Proper positioning of the patient is essential for optimal exposure of and accessibility to the oral surgical site.

Patient positioning

After the patient has been stabilized under anesthesia and necessary radiographs have been obtained, a nonsterile assistant should move the patient into the desired position for the surgery. Care must be taken to maintain the patient's airway and to support the head and neck region. It is most convenient to position the anesthetic machine away from the front of the animal rather than near the head as is routinely done. The oxygen/gas supply tubes are placed alongside or over the animal to the rear.[48] It also is convenient to use a tilt table, so that the end of the table, where the animal's head is positioned, can be further elevated to a comfortable working level for the surgeon. It is essential that the patient's position allow the surgeon maximum viewing and accessibility to the oral surgical site. For maxillofacial fracture repair the head must be securely fastened to the table in order to ensure appropriate landmarks remain referenced to the midline. There are three basic recumbency positions: lateral, dorsal, and sternal. The selection of a position should be based upon the surgical procedure, the condition of the patient, and the surgeon's personal preference.

Lateral recumbency

The most common position preferred by veterinarians performing routine oral surgery is lateral recumbency. The patient is placed on either side, depending on the location of the surgical approach. There is good access to the buccal surfaces of the uppermost teeth and jaws, but there is limited visualization of the palatal and lingual surfaces of the opposite quadrants, making it difficult to see both the buccal and palatal/lingual surfaces of the same tooth. Often, patients must be turned over to the other side to complete procedures. Advantages of lateral recumbency include rapid draining of fluids from water-cooled power instruments, and high-risk patients may be more readily stabilized under anesthesia when in lateral versus dorsal recumbency.

Dorsal recumbency

Dorsal recumbency offers better visualization of the dentition, especially of the maxillary arch. The patient is placed on its back and centrally aligned to the table. Sandbags, body rolls, wedge-shaped blocks, or vacuum-activated positioning bags are used to hold the patient in position. For more secure positioning, the patient's neck should be elevated with a small bag or roll so that the hard palate is level with the table, facilitating fluid drainage (see Fig. 13.8). It is also recommended that the head end of the table be slightly lower. Dorsal recumbency offers direct access to all root positions of the maxillary dentition. It is also the preferred position for rostral maxillectomy procedures and cleft palate repair. It is crucial that the patient's respiratory function and blood pressure be monitored carefully, especially in medically compromised cases.

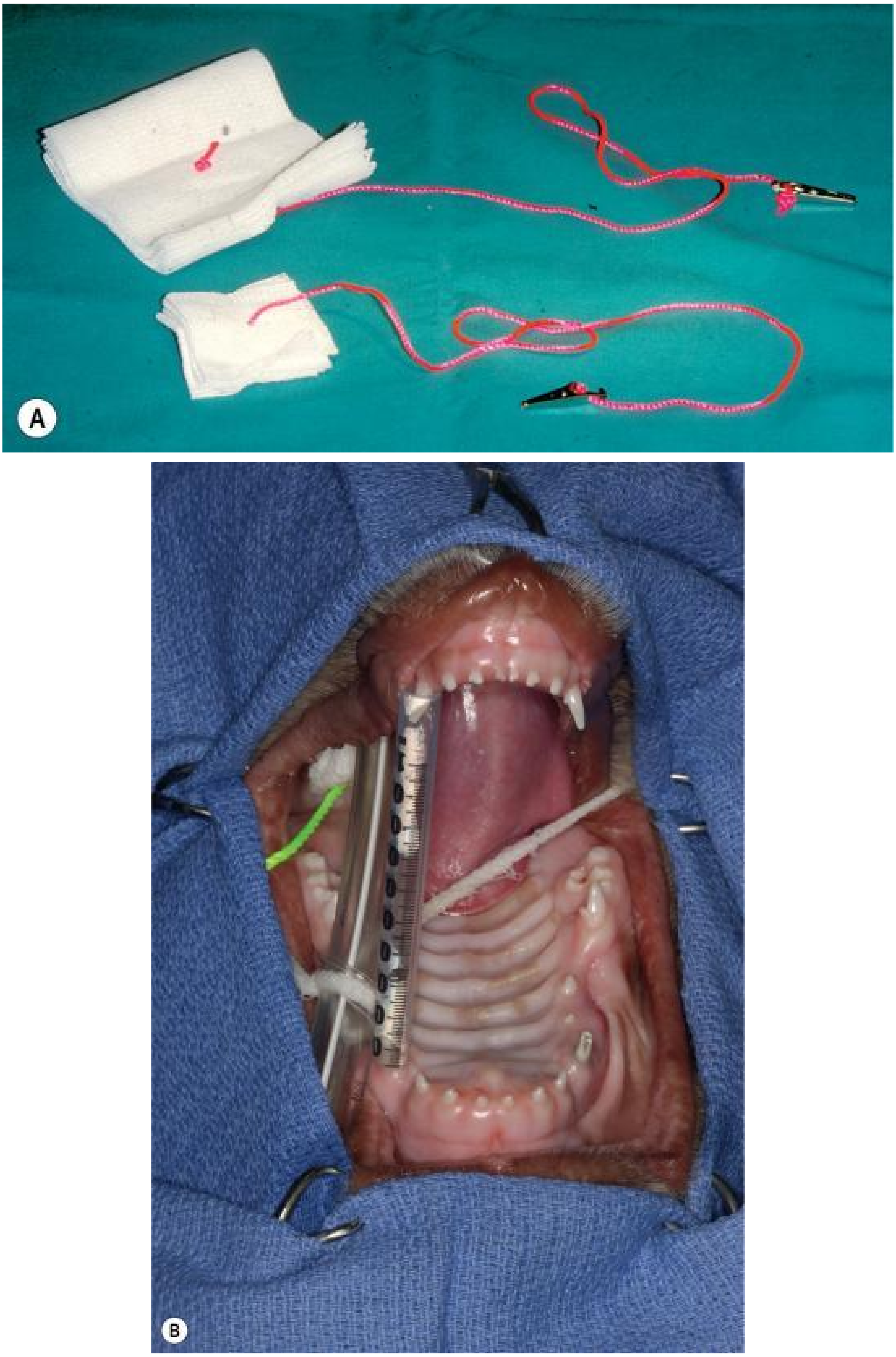
Sternal recumbency

Although not generally used for most oral surgical procedures, sternal recumbency is an excellent position for mandibulectomy procedures. For this surgery the patient is placed in a prone position and stabilized with sandbags or other aids. The maxilla is suspended by perforating the maxillary canines through a long strip of 2-inch adhesive tape. The ends of the tape are then extended and wrapped high around IV poles placed on either side of the patient's head. The head is raised until the mandible is parallel to or slightly lower than the surface of the operating table. For maxillary fracture repair the head is not suspended but fastened to the table by securing the mandible to the table top with waterproof tape.

Preparation of the maxillofacial skin and oral cavity

Pharyngeal pack and speculum

Dental procedures often require the aid of water-cooled power instruments, such as high-speed and surgical handpieces, and sonic and ultrasonic instruments. These devices can flood the patient's oral cavity with fluid. In addition to the use of an endotracheal tube with inflatable cuff during inhalation anesthesia, pharyngeal packs will act as a barrier, preventing liquids or other materials from entering the lungs. Pharyngeal packs may consist of 2–5 exodontia sponges (Exodontia Sponges; Henry Schein, Melville, NY) threaded together on a long, bright-colored string (Fig. 7.18A) The pack is formed into a small roll and placed alongside the endotracheal tube behind the soft palate, with the end of the string attached by means of an alligator clip outside the oral cavity. Pulling on the string will remove the entire pack and prevent it from obstructing the airway after extubation, or from being inadvertently swallowed by the patient upon recovery. The pack must not be so big as to compress the endotracheal tube, interfere with the venous outflow of the oral cavity, or obstruct access to oral structures. Too many sponges excessively packed around the endotracheal tube may result in swelling of the tongue, especially during a prolonged procedure. Packs may need to be changed frequently if they appear to be saturated with liquid.



• FIG. 7.18 (**A**) Pharyngeal pack made from exodontia sponges (Exodontia Sponges; Henry Schein, Melville, NY); (**B**) Dog in dorsal recumbency, draped for minor oral surgery using four drapes secured with Backhaus towel clamps. Note the presence of the mouth speculum.

A mouth speculum or prop may be placed between canine teeth or premolar teeth of the opposing jaws to help hold the jaws apart and improve access to the oral cavity. An excellent and inexpensive mouth speculum can be made by cutting the ends of a plastic 1-mL syringe. The length may be cut according to the size of the patient's opening (Fig. 7.18B). Care should be taken not to strain the masticatory musculature by propping the jaws too wide or for a prolonged time period. The advantages of this type of mouth speculum over conventional metal specula include that it is disposable, small, and unobtrusive. If an animal were to inadvertently wake up during anesthesia and bite on the plastic speculum, there would not be the risk of fracturing any of the canine teeth, which can happen with a metal speculum. In addition, the use of metal specula has been found to occlude the maxillary artery, and has been implicated as a cause of post-anesthesia blindness in cats.[49–53]

Preparation of the oral cavity

Rinsing the oral tissues with an antimicrobial solution prior to oral surgery is an important component of the patient's preparation (see Chapter 3). Antimicrobial solutions have an immediate and lasting bactericidal action within the oral cavity, thereby reducing the risk of surgical wound infections and potential bacteremias.[54-58] Also, it is generally recognized that oral bacteria are aerosolized during dental procedures involving air-turbine handpieces, and sonic and ultrasonic scalers. Preprocedural rinsing has been shown to significantly reduce the level of aerosolized bacteria generated during routine dental procedures, as well as to decrease the number of microorganisms entering the patient's bloodstream.[34,59,60]

One of the most widely used antimicrobials used for oral applications in both animals and humans is chlorhexidine gluconate.[61,62] It exhibits broad-spectrum efficacy, substantivity to tooth surfaces and mucosa, low toxicity, and dental plaque-inhibiting properties.[62] Chlorhexidine is active against a wide range of gram-positive and gram-negative organisms, yeast, fungi, facultative anaerobes, and aerobes. Its action is the result of the adsorption of chlorhexidine onto the cell wall of the microorganism, resulting in a leakage of intracellular components.[63] It may be applied to the teeth and oral tissues with the use of a syringe, brush, swab, or gauze. Chlorhexidine is both light and air sensitive. Therefore it must be kept tightly closed in a light-resistant container until ready for use.

Another oral antimicrobial solution used in dentistry is povidone iodine. Lower levels of bacteremia among patients treated with povidone-iodine solution have been reported over patients treated with chlorhexidine or sterile water.[57] Iodine exhibits rapid antimicrobial action, even at low concentrations. Available as a 10% povidone-iodine solution (1% free iodine), it may be applied with a syringe or swab to intact oral mucous membranes. A 10-fold dilution (1% povidone iodine) is used if a laceration is present.[64,65] Some patients may develop a sensitivity or allergy to this solution, and caution is therefore necessary.[62]

Care must be taken not to accidentally spill or spray these antimicrobial solutions into the eyes or ears of the patient.

Maxillofacial skin preparation

The skin and hair of an animal can harbor a significant amount of microbial activity and are known to be a major source of surgical wound infection.[3] Patient preparation techniques, such as hair removal and skin cleansing of the intended surgical site, will kill surface bacteria, but will not completely sterilize the area of all microbes residing in deeper, inaccessible skin structures.[3] The objective of these techniques, however, is to minimize the risk and the potential for surgical site infections in the postoperative period. All initial skin preparation should be performed outside the operating room.

Clipping is the most recommended technique for hair removal for animals.[3] The clip should be thorough yet gentle to prevent trauma or abrasion to the skin. The surgical site should be clipped immediately prior to surgery after anesthesia has been induced in the animal and before moving to the operating room. A wide area of skin should be clipped around the proposed surgical incision. Long fur growing near the periphery of the clipped area should be cut short enough so that it cannot hang over onto the clipped area.[66] Alternatively, the facial fur can be contained by placing a segment of stockinette bandage material around the head caudal to the angle of the mouth. All loose hair and debris should be removed from the skin, and flushed from the oral cavity.

Chlorhexidine and povidone-iodine products have been recommended as the best preoperative skin preparations because they are broad-spectrum bactericidal agents that rapidly kill accessible skin microbes.[3] When these solutions are used in the maxillofacial region, however, possible complications involving the eyes and ears may arise. A review of five antimicrobial skin preparations for the maxillofacial region in humans concluded that povidone-iodine (10%) solution (not the 7.5% surgical scrub with detergent) was the safest and best-suited product.[67]

Before skin preparation of a patient is initiated, the skin should be free of gross debris and dirt. Ophthalmic lubrication should be applied liberally to the patient's eyes. The patient's skin is then prepared by applying an antimicrobial agent in concentric circles, beginning in the area of the proposed incision. The scrubbing action continues outward until the outer margins of the clipped area are reached. This process is repeated using clean sponges each time until dirt and debris are absent.

After the animal is transported to the operating room, and positioning is satisfactory, the surgical site is sprayed with additional antiseptic. If contamination occurred when moving the animal, it will be necessary to re-scrub the area.[3]

Draping of the patient

The placing of sterile drapes to isolate the oral cavity from the rest of the patient is often a neglected part of aseptic technique during oral surgery. A four-drape system may be used by placing each drape along the mucocutaneous junction of the mandible and maxilla (Fig. 7.18B). The drapes are secured to the patient's skin with towel clamps or staples. For major maxillofacial surgery, an additional sterile fenestrated drape should be placed over the surgical site for a greater degree of asepsis.

Drapes act as a barrier, keeping hair and bacteria from hair follicles and skin from contaminating the mouth, instruments, suture material, or the surgeon's gloves during a procedure.[1–3,42] They also protect the patient's eyes from oral debris.[1] Sterile drapes are available in either reusable cloth or disposable paper. Cloth is generally less expensive but may absorb bacteria when exposed to blood, tissue fluids, and lavage solutions.[3] Cloth drapes are adequate for minor oral surgery. Paper drapes are more expensive, but are water-resistant and are recommended for major oral surgery.

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CHAPTER 8

Suture materials and biomaterials

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General principles

Sutures placed intraorally are exposed to tissues of high vascularity, a constant bathing of saliva, bacteria, fluctuations in temperature and pH, and masticatory trauma.[1] The tissue reaction in the mouth to the placement of sutures is different from other regions of the body, where reactions typically peak between the second and seventh days.[2,3] The physical character of the suture material is the most important consideration in determining intraoral tissue reactivity; in general, monofilament and nonabsorbable sutures are less reactive than multifilament and absorbable suture materials.[2,3] The capillarity of a suture material, the process by which fluid and bacteria are trapped in the interstices of multifilament material, is also an important consideration for the intraoral placement of sutures, as multifilament sutures are more likely to contribute to the wicking of bacteria and oral fluids deep into the wound.[2,4] Suture materials intended for intraoral use should accumulate little or no bacterial plaque.[1] Furthermore, absorbable suture materials are favored over nonabsorbable materials because they enhance patient comfort and eliminate the need for removal. The additional manipulations required during intraoral suture removal may predispose to bacteremia and endocarditis in the high-risk cardiac patient.[5]

The primary objective of dental suturing is to achieve apposition of wound edges to promote optimal healing.[6] Blood and serum that accumulate beneath an inappropriately apposed flap delay the healing process.[6] Sutures function to appose wound edges until the supported tissues have regained sufficient strength to withstand tensile forces. When their strength is no longer needed, the suture material should absorb completely and predictably to prevent additional delays in healing.[1] The ideal time for suture loss specific to the oral tissues has not been clearly defined.[1] It has been suggested, however, that 4 days is the optimum time for suture removal; by 72 hours, a stratified squamous keratinized epithelial layer has formed in the attached gingiva.[7,8] Connective tissue healing with type III and type I collagen occurs by 96 hours.[9,10]

When selecting a suture material, the surgeon should consider the physical properties of the suture material (capillarity, size, tensile strength, absorbability surface characteristics, and tissue reactivity) and the rate of wound healing in the area.[3] All too often, however, the selection of suture material by the surgeon is based on handling properties alone. The minimal requirements for an intraoral suture material are outlined in Box 8.1.

• BOX 8.1

Minimal Physical and Biological Requirements for Intraoral Suture Materials

1. Fast absorption with minimal tissue reactivity
2. Good short-term tensile/knot strength with sutures of small diameter
3. Minimally plaque retentive
4. Low capillarity and fluid absorption
5. Knots with good security and without injury to the soft tissues
6. Low tissue drag
7. High pliability for favorable handling in confined areas

Suture characteristics

Suture materials may be absorbable or nonabsorbable, and monofilament or multifilament (see Box 8.1). Absorbable suture materials have been defined by the United States Pharmacopeia (USP) as strands of collagen or synthetic polymers that are capable of absorption in mammalian tissue. By contrast, nonabsorbable sutures are strands of material that are resistant to absorption. Monofilament suture materials are composed of a single strand of material, and multifilament sutures are braided or woven from multiple strands of material.

Physical properties

Handling characteristics

The handling characteristics of a suture material are intimately associated with the intrinsic stiffness of the material. Suture materials that are more pliable and of smaller diameter have favorable handling characteristics in comparison with stiff suture materials of large diameter.[11] When cut, the sharp ends of stiff suture materials (e.g., stainless steel or nylon) can result in the mechanical irritation of tissues. As a general rule, multifilament sutures have better handling characteristics than monofilament sutures, as do uncoated sutures compared with coated ones. Related to stiffness is elasticity. Most suture materials exhibit limited elongation when exposed to increasing loads. Notable exceptions are polypropylene and polybutester, which are relatively elastic. The surface of uncoated braided materials such as polyglycolic acid is rough and causes considerable friction and trauma when going through tissues.

Capillarity

The degree of bacterial transport along suture filaments is determined by the fluid absorption and capillarity of the suture material.[12] Monofilament sutures withstand contamination better than multifilament sutures, and tissue reactivity is minimized with monofilament suture materials.[13] Multifilament sutures have a fivefold to eightfold higher affinity for bacterial adherence than monofilament nylon sutures.[14]

Tensile strength, knot-pull tensile strength, and knot security

The tensile strength of a suture material refers to the strength required to break an untied portion of suture when force is applied along its length (Table 8.1).[15] Thicker-diameter sutures have greater strength than smaller-diameter sutures, but the thickness also increases the total amount of foreign material in the wound and the severity of associated tissue reaction.[16] The knot is mechanically the weakest link of the suture loop; tension forces are converted into shearing forces at the knot, and the initial tensile strength of a suture is reduced by at least one-third.[17,18]

TABLE 8.1

General Characteristics of Suture Materials used in Veterinary Oral and Maxillofacial Surgery

Suture	Trade Name	Type	Degradation	Tissue Reactivity	Intraoral Survival Time (Days) [a]	Tensile Strength Retention (%)	Complete Absorption (Days)
Catgut (chromic)	Surgical gut [b]Chromic gut [c]	Absorbable Multifilament	Proteolytic enzymes and phagocytosis	Moderate	3–7 [48]; 7–10 [35] (variable, shorter in infected wounds)	Unpredictable	45–60
Polyglycolic acid	Dexon S [c] (uncoated) Dexon II [c] (coated)	Absorbable Multifilament	Hydrolysis	Minimal acute	7–14 [48]; 16–20 [35]	65% (55% sizes 7-0 or smaller) after 14 d; 35% (20% sizes 7-0 or smaller) after 21 d	60–90
Polyglactin 910	Vicryl [b]	Absorbable Multifilament	Hydrolysis	Minimal acute	28 [30]	75% after 14 d	56–70
	Vicryl Rapide [b]		Hydrolysis (phagocytosis)		3 [1]	50% after 5 d; 0% at 14 d	42
	Coated Vicryl Plus [b]		Hydrolysis		28 [30]	75% after 14 d	56–70
Polydioxanone	PDS II [b]PDS Plus [b]	Absorbable Monofilament	Hydrolysis	Slight	>28 [48]	74% after 14 d; 58% after 28 d; 41% after 42 d	180
Polytrimethylene carbonate	Maxon [c]	Absorbable Monofilament	Hydrolysis	Minimal		81% after 14 d; 59% after 28 d; 30% after 42 d	180
Poliglecaprone 25	Monocryl [b]Monocryl Plus [b]	Absorbable Monofilament	Hydrolysis	Minimal acute		50%–60% after 7 d; 20%–30% after 14 d; 0% within 21 d	91–119
Polyamide	Ethilon [b] (monofilament) Nurolon [b] (multifilament) Monosof [c] and Dermalon [c] (monofilament) Surgilon [c] (multifilament)	Nonabsorbable Monofilament/multifilament	Hydrolysis (slow)	Minimal acute		15%–20% loss after 365 d, retains remaining 80% indefinitely	Gradual encapsulation by fibrous tissue
Polybutester	Novafil [c]	Nonabsorbable Monofilament	N/A	Minimal acute		N/A	N/A
Polypropylene	Prolene [b]Surgipro [c] Surgipro II [c]	Nonabsorbable Monofilament	N/A	Minimal acute		N/A	N/A
Hexafluoropropylene-VDF	Pronova [b]	Nonabsorbable Monofilament	N/A	Minimal acute		N/A	N/A
Polyester	Mersilene [b] (uncoated) Ethibond Excel [b] (coated) Ti-Cron [c] Surgidac [c]	Nonabsorbable Multifilament	N/A	Minimal acute		N/A	Gradual encapsulation by fibrous tissue
Stainless steel	Surgical Stainless Steel [b] (monofilament) Surgical Stainless Steel [b] (multifilament) Steel [c] (monofilament) Flexon [c] (multifilament)	Nonabsorbable Monofilament/multifilament	N/A	Minimal acute		N/A	N/A
Silk	Perma-Hand [b] Sofsilk [c]	Nonabsorbable Multifilament	Proteolytic enzymes and phagocytosis	Severe		70% after 14 d; 50% after 30 d	Gradual encapsulation by fibrous tissue

[a]Spontaneous loss

[b]Ethicon, Inc., Somerville, NJ

[c]Medtronic Animal Health, Minneapolis, MN

Knot-pull tensile strength is defined as the force in pounds that is required to break a knotted strand of suture material, whereas knot security refers to the knot-holding capacity of a suture material expressed as a percentage of the tensile strength. The knot-pull tensile strength is related to the diameter of the suture, the type of suture material, and the size of the suture loop.[18,19] Knot security is influenced by the suture diameter, coefficient of friction, and quality of the knot.[18-20]

When induced to failure, secure knots break rather than untie due to slippage of the knot.[21] In general, the use of larger-diameter sutures and the placement of additional throws in the knot will increase the security and reliability of the knot.[16] More specifically, catgut, polyglycolic acid, polyglactin 910, and polypropylene require three throws to ensure knot security; four throws are necessary for polydioxanone and polyamide.[21]

Biologic properties

Suture materials may be derived from natural or synthetic sources. Synthetic sutures account for almost all of the currently used suture materials, and the only naturally derived examples discussed here include catgut and silk (see Table 8.1). Natural and synthetic sutures differ in their mechanism of absorption. Natural sutures are degraded and absorbed by the proteolytic enzymes supplied by macrophages, and synthetic sutures are degraded by hydrolysis in tissue fluid.

Biofilm

Biofilms are complex communities of surface-associated cells enclosed in a polymer matrix containing open water channels that can develop on many different medical devices, including suture materials.[22] Biofilms serve as a reservoir for bacteria. Bacteria growing on the surface of a biofilm have been shown to display a unique phenotype with increased resistance.[23] Within biofilms, bacteria are hidden from the host immune system and are less susceptible to antibiotics.[22] Complete elimination of bacteria from biofilms on suture material may be impossible.[22] The bacteremia created by suture removal and the potential risk of endocarditis has led to the suggestion of antibiotic prophylaxis at the time of suture removal in compromised patients but this is controversial.[24]

Tissue reactivity

All suture materials elicit a foreign body cellular reaction when they are implanted into tissues, but natural sutures exhibit greater tissue reactivity than their synthetic counterparts, and the inflammatory phase of wound healing is prolonged (see Table 8.1). The most inert materials are monofilament synthetics such as nylon and steel. The more material that is implanted in a wound, the greater the tissue reaction, and the knotted portion of the suture loop contains the highest density of foreign material.[25] Suture size contributes more to tissue reaction than extra throws to the knot.[16]

Influence of wound infection

The placement of foreign material in an infected wound can exacerbate or perpetuate infection; therefore, whenever possible, the surgeon should avoid placing sutures in heavily contaminated wounds that require immediate closure.[26] Sutures may potentiate infection by inducing an exudative foreign body response with local tissue autolysis and by physically shielding the bacteria from the host defense.[26] For this reason, multifilament sutures are not recommended for use in contaminated wounds, as the clearance of bacteria from the interstices of multifilament materials is slower than from monofilament materials.[26] Although the physical and chemical structure of a suture material influence the degree of infection, proper surgical technique and wound care also play important roles in minimizing wound infection.[26]

Influence of pH

The pH level in tissues and body fluid varies by location (e.g., gastric juice, 0.9 to 1.5; and pancreatic juice in the duodenum, 7.5 to 8.2), and is influenced by the presence of infection and inflammation. *Proteus,* a urea-splitting bacterium, dissociates urea to ammonia and elevates the pH of urine. The pH of inflamed tissue is generally acidic.[27] The pH of a tissue can affect the retention of tensile strength of suture materials.

The degradation of natural absorbable sutures is accelerated in both acidic and alkaline pH but, in general, only alkaline conditions have a significant effect on synthetic absorbable sutures.[27] Polydioxanone is a notable exception, as it degrades faster in acidic solutions.[28] Silk is the most vulnerable of nonabsorbable sutures to fluctuations in pH, but nylon also exhibits a degradation in tensile strength in acidic environments.[27,28] Polypropylene, a synthetic nonabsorbable suture material, retains its initial tensile strength over a wide range of pH.[28]

Suture materials

Catgut

Plain catgut ("gut") is a natural suture material derived from the submucosa of sheep intestine or the serosa of cattle intestine. Chromic catgut is a modification of plain catgut that is tanned with chromic salts to improve strength and delay dissolution.[29] Gut is absorbed by phagocytosis and is associated with a marked tissue inflammation that can be detrimental to healing. Conversely, tissue inflammation may lead to a more rapid breakdown of catgut. Plain gut has a median survival time of 4 days in the oral cavity, whereas chromic gut retains its strength for 2 to 3 weeks.[26,30] In moist environments such as the oral cavity, the strength of gut is reduced by 20%–30%.[31] Gut is a stiff material that must be moistened in alcohol, and forms knots that can be irritating to the oral tissues.[32,33] Infection rates may increase with the use of gut.[34] The advent of synthetic materials preferable to gut, with less tissue reactivity and more predictable resorption, has almost made catgut obsolete.[25]

Synthetic absorbable suture materials

Polyglycolic acid

Polyglycolic acid (PGA) is a multifilament suture material derived from a homopolymer of glycolic acid (hydroxyacetic acid), and is available uncoated (Dexon S, Medtronic Animal Health, Minneapolis, MN) or coated (Dexon II, Medtronic Animal Health, Minneapolis, MN) with polycaprolate, a copolymer of glycolide and ε-caprolactone. Polyglycolic acid is absorbed by hydrolysis with less associated tissue inflammation than silk or plain or chromic catgut.[35] The median survival time of polyglycolic acid in the oral mucosa is 15 days.[30,35] The initial tensile strength of polyglycolic acid exceeds that of silk and gut, but is decreased appreciably when placed in oral tissue.[36] The handling characteristics of polyglycolic acid are favorable, similar to silk, but its knot security is poor.[37] Polyglycolic acid also has a tendency to cut through friable tissue, which is not a favorable quality for suturing gingival tissues.[21] Polyglycolic acid has been shown to inhibit bacterial transmission due to the release of monomers.[37]

Polyglactin 910

Polyglactin 910 is a braided suture material that is a copolymer of glycolic and lactic acid. The lactic acid provides water repellence, delaying the loss of tensile strength. Vicryl (Ethicon, Inc., Somerville, NJ) is coated with polyglactin 370 and calcium stearate to decrease tissue drag and bacterial adherence. It is absorbed by hydrolysis, and its intraoral survival rate is approximately 28 days.[30] Complete resorption may take up to 40 to 60 days.[38] Because polyglactin 910 persists much longer than is necessary for intraoral wound healing, it is not an ideal suture material for periodontal procedures.[38] The long absorption time of polyglactin 910 also makes it a potential nidus for infection.

Vicryl Rapide (Ethicon, Inc., Somerville, NJ) is an irradiated form of polyglactin 910 that is lost from the skin as early as 10 to 14 days. Intraorally, the median time for suture loss of Vicryl Rapide is 3 days.[1] Gamma radiation alters the molecular structure of polyglactin 910 and enhances its in vivo absorption rate.[39] In contrast to nonirradiated polyglactin, Vicryl Rapide may also be absorbed by phagocytosis.[39] Vicryl Rapide remains in the skin longer than 5 days and is not recommended for facial skin closure.[40] Irradiated polyglactin 910, however, satisfies many of the minimal physical and biological requirements for an intraoral suture outlined in Box 8.1; these include fast absorption, ease of handling, good knot security, and minimal inflammatory reaction of the surrounding tissues.[41] Although Vicryl Rapide is favorable in terms of its enhanced absorption rate, it has a tendency to be more brittle than nonirradiated polyglactin 910 and to break if tugged on suddenly.[41]

Coated Vicryl Plus (Ethicon, Inc., Somerville, NJ) is a recent addition to the Vicryl suture material family that contains the synthetic broad-spectrum antimicrobial agent triclosan (2,2,4′-trichloro-2′-hydroxy-diphenyl ether).[42] Triclosan is an antimicrobial agent found in many personal/oral healthcare products, and is a potent inhibitor of enoyl acyl carrier protein (ACP) reductase, an essential enzyme in bacterial fatty acid biosynthesis.[43] In general, triclosan is considered more effective against gram-positive than gram-negative bacteria species.[43] Coated Vicryl Plus exhibits the same physical and functional handling properties as Coated Vicryl, and the only difference between the two products is the addition of 100–500 ppm of triclosan to the coating.[44] The addition of triclosan to Coated Vicryl was found to affect neither wound healing nor its absorption pattern.[42] Coated Vicryl Plus exhibits in vitro antimicrobial activity against *Staphylococcus aureus* and *Staphylococcus epidermidis*, the most prevalent organisms associated with surgical wound infection, for up to 7 days in an aqueous environment.[45] Triclosan has shown sustained antimicrobial activity during the period of wound reepithelialization within the early postoperative period.[46] The antimicrobial efficacy of triclosan-coated suture material was unhindered when coated with biologic substrates, 20% bovine serum albumin, that mimic the tissue proteins recruited to the margins of a surgical wound.[46] Besides the obvious bactericidal benefit of antibacterial-coated sutures, coated sutures have also demonstrated an ability to significantly reduce bacterial adherence compared to noncoated suture material.[46] The usefulness of Coated Vicryl Plus in the oral cavity is related to triclosan's ability to control oral plaque, and is supported by studies showing the effectiveness of triclosan in oral rinses and dentrifices.[43] Incorporation of antibacterial agents such as triclosan into suture materials, however, is not without controversy; species such as *Pseudomonas aeruginosa* have already been found to be resistant to various antiseptic agents, including triclosan.[46]

Polydioxanone

PDS (Ethicon, Inc., Somerville, NJ) is a polyester of the monomer paradioxanone. PDS is hydrolyzed much slower than other absorbable suture materials and retains 74% of its original tensile strength at 2 weeks, 58% at 4 weeks, and 41% at 6 weeks.[47] In a comparison study of suture materials in the feline oral cavity, PDS was found to be intact at day 28.[48] When used on the gingiva, the hard ends of the suture material may be abrasive to the buccal mucosa that comes into contact with them. PDS is useful in wounds where a long healing time and extended tensile strength are required.[47,48] Although PDS was developed for use as a resorbable suture material, it has also successfully been used as a resorbable alternative to stainless steel wire in transosseous fixation.[49]

Similar to Vicryl Plus, an antibacterial version of PDS, PDS Plus (Ethicon, Inc., Somerville, NJ), has recently been introduced. PDS Plus inhibits bacterial colonization of the suture by *S. aureus*, *S. epidermidis*, *Escherichia coli*, and *Klebsiella pneumoniae*.

Polytrimethylene carbonate

Maxon (Medtronic Animal Health, Minneapolis, MN) is derived from a copolymer of glycolide and trimethylene carbonate. Maxon is very similar to PDS in terms of tensile strength and long tissue retention but is less rigid.[26] Because of its long retention time, Maxon is less desirable as a suture material for routine intraoral use. Maxon retains 81% of its original tensile strength at 2 weeks, 59% at 28 days, and 30% at 42 days.[47] Resorption occurs by hydrolysis between 180 and 210 days.[50]

Poliglecaprone 25

Monocryl (Ethicon, Inc., Somerville, NJ) is an extremely pliable monofilament suture material that is derived from a segmented copolymer of ε-caprolactone and glycolide.[51] The pliability of Monocryl contributes to its excellent handling characteristics.[52] Monocryl loses 20%–30% of its original tensile strength after 2 weeks, and is completely absorbed by hydrolysis in 90 days.[51] Similar to other absorbable sutures that resorb via hydrolysis, Monocryl exhibits minimal tissue reaction that is characterized by macrophages, fibroblasts, lymphocytes, plasma cells, and giant cells.[51,52] Monocryl has superior tensile strength and is less reactive than polyglactin 910.[52] In a recent study involving human patients undergoing dentoalveolar surgery, microbial adherence to Monocryl was shown to be significantly lower than nonresorbable multifilament and monofilament sutures.[53] A triclosan-coated version of Monocryl, Monocryl Plus (Ethicon, Inc., Somerville, NJ), is also available.

Synthetic monofilament nonabsorbable suture materials

Polyamide

Nylon (Ethilon, Ethicon, Inc., Somerville, NJ) is a synthetic polyamide polymer fiber that is a popular suture for cutaneous closure. Nylon is classified as a nonabsorbable suture, but it does exhibit some absorption by hydrolysis and loses 15%–20% of its tensile strength per year.[26,29] The hydrolysis of nylon is influenced by the acidity of the milieu, as acids catalyze the hydrolysis of the amide linkages in nylon.[29] The tissue reactivity of nylon is minimal, but its knot security is poor, and multiple throws are required to properly seat a suture. If used as an intraoral suture material, the sharp cut ends of nylon can be irritating to the patient and injurious to the oral soft tissues.[42] Soaking in alcohol may improve pliability.[26]

Polybutester

Novafil (Medtronic Animal Health, Minneapolis, MN) is a copolymer composed of polyglycol and polybutylene terephthalate. Compared with nylon, Novafil is less stiff and has a lower memory. Novafil also has greater elasticity than either nylon or polypropylene, and the unique feature of being able to stretch 50% of its length under low loads; as a result, Novafil is able to passively accommodate wound edema, reducing suture marks and cutthroughs.[26,54] Although synthetic nonabsorbable sutures are rarely used intraorally, Novafil can be used safely in the mouth with less patient discomfort than nylon.[55]

Polypropylene

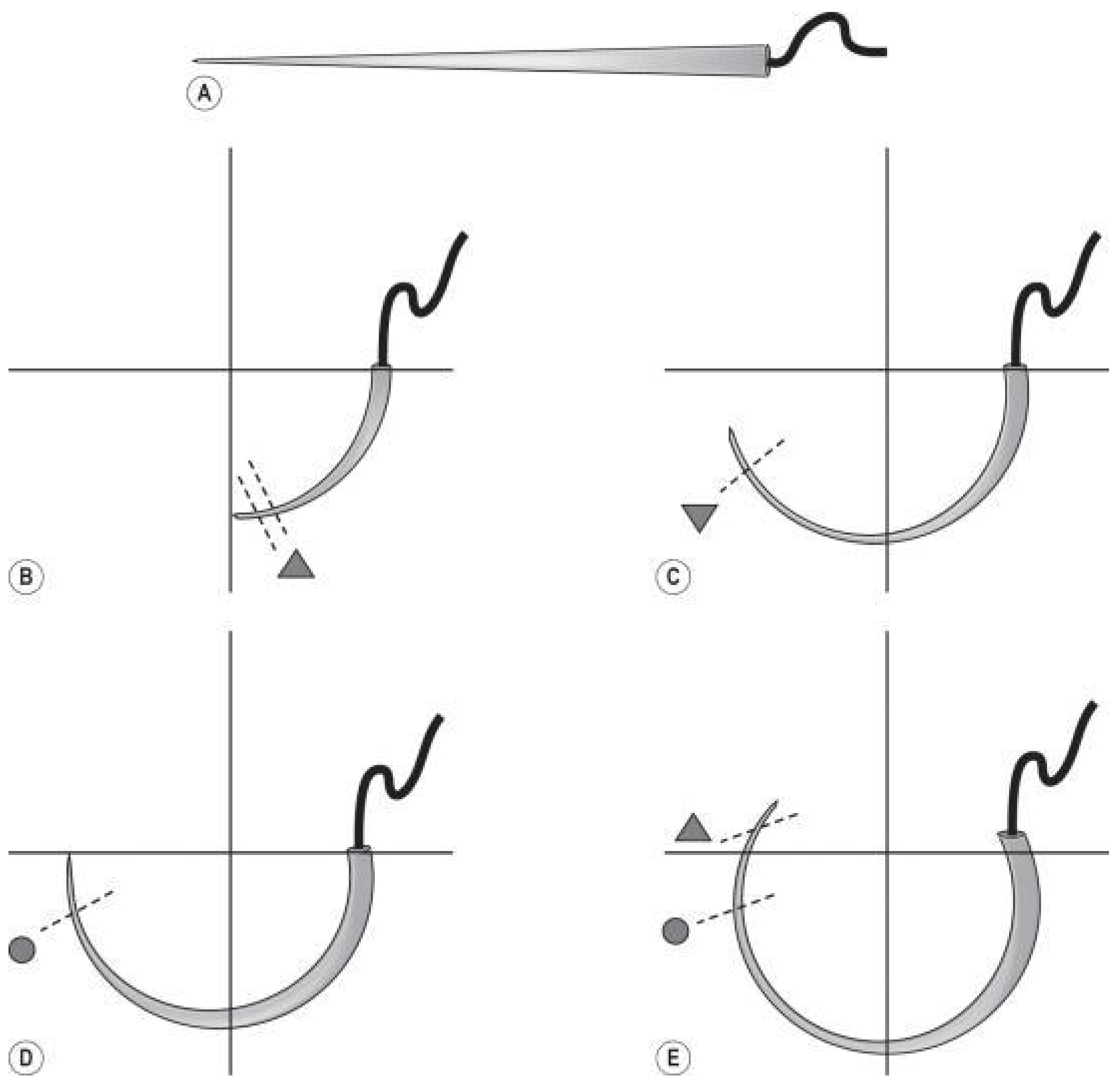
Prolene (Ethicon, Inc., Somerville, NJ) is a synthetic nonabsorbable monofilament that is derived from the polymerization of propylene. The absorption of Prolene is virtually nonexistent. Its tissue reactivity is low, and it is preferable for treating contaminated sites. In comparison with nylon, Prolene has better knot security and elasticity and pulls smoothly through tissues. The plasticity of Prolene sutures accommodates tissue swelling and reduces trauma to the sutured tissue.[26] A newer modification to the Prolene needle, marketed as Ethilon's Hemo-Seal technology, narrows the diameter of the suture attachment to the needle, allowing for smaller needles to be used, which in turn reduces bleeding by allowing for a larger diameter of the suture to fill the needle hole.

Hexafluoropropylene-VDF

Pronova (Ethicon, Inc., Somerville, NJ) is a synthetic nonabsorbable monofilament that is uniquely composed of two polymers: polyvinylidene fluoride homopolymer and polyvinylidene fluoride hexafluoropropylene copolymer. Pronova is comparable to polypropylene in terms of biocompatibility, and its advantages are reduced package memory, easier handling, greater tensile and knot strength, and greater resistance to instrument damage. Pronova has been marketed for use in cardiovascular surgery, ophthalmic surgery, and neurosurgery, but can also be used for general soft tissue wound closure and ligation.

Barbed sutures

In an attempt to reduce the inflammation and tissue ischemia associated with suture knots, as well as eliminate the complication of knot breakage, barbed knotless monofilament sutures have been introduced. Barbed sutures are absorbable or nonabsorbable monofilament sutures that have helically arranged angled (opposite the direction of the needle) barbs that engage the tissue; which maintains wound edge approximation and tension along the suture line, improving the speed of continuous wound closure patterns (Fig. 8.1). The barbs of the suture are designed to flatten down to the core of the suture providing smooth passage of the suture through tissue. Both unidirectional and bidirectional variants are available. The unidirectional variant has a loop at the end of the suture opposite the needle that eliminates the need to tie a knot at the start of the incision; whereas the bidirectional variants have needles at both ends of the suture. Typically, one would select a barbed suture one size larger than the corresponding monofilament suture. A 3-0 knotless, barbed polyglycolic acid and polycaprolactone suture system (Quill Monoderm; Surgical Specialties Corp, Reading, PA) placed in a continuous pattern was shown to decrease oral wound closure time in cats by 41.4% compared with simple interrupted 4-0 poliglecaprone 25 in a split-mouth study.[60] All of the typical suture manufacturers offer barbed equivalents in their suture lines: Stratafix (Ethicon, Sommerville, NJ), Quill (Surgical Specialties Corp., Reading, PA), V-Loc (Medtronic Animal Health, Minneapolis, MN).



• **FIG. 8.2** Shapes of surgical needles and types of needle points. The circles and triangles represent the cross-sectional appearance of the needle points. (**A**) Straight needle; (**B**) conventional-cutting 1/4-circle needle; (**C**) reverse-cutting 3/8-circle needle; (**D**) tapered 1/2-circle needle; (**E**) tapercut 5/8-circle needle.

Synthetic multifilament nonabsorbable suture materials

Polyester

Polyester is a braided suture material that is available uncoated (Mersilene; Ethicon, Inc., Somerville, NJ) or coated (Ethibond Excel; Ethicon, Inc., Somerville, NJ) with polybutilate. The coating minimizes capillarity and allows for tissue passage with less friction, but the coating may crack off after knots are tied, resulting in deposition of foreign material in the wound.[3] Because polyester is braided, its handling characteristics are better than monofilament nylon but, as a result, it is predisposed to greater bacterial adherence. Polyester has low tissue reactivity and is retained within the wound in a fibrous cartilaginous capsule.[56] Due to its high tensile strength and knot security, polyester is an excellent suture for mobile facial areas, tracheal anastomosis, and respiratory tract surgery.[26,56]

Stainless steel

Stainless steel sutures are composed of low-carbon, iron-alloy strands and are available as either a monofilament or a multifilament.[30] Stainless steel has low tissue reactivity and good tensile strength and knot security. Monofilament stainless steel is difficult to handle and is used infrequently as a suture material in periodontal surgery, but is occasionally used around the lips to prevent suture removal in dog and cat patients.[39] Its characteristics are very similar to monofilament synthetic nonabsorbable suture materials, and it is used more commonly for the internal fixation of fractures. Multifilament stainless steel is easier to work with but gives rise to the same problems as multifilament nonabsorbable suture materials, namely wound infection due to capillary action and sinus tract formation when used internally.

Silk

Modern silk sutures are composed of 70% natural silk and 30% extraneous materials (gum, beeswax, and silicone).[32] Silk is classified as a nonabsorbable suture material, but it loses 50% of its original strength within 1 month and is completely absorbed by proteolysis in 2 years.[26] Tensile strength is lost in 1 year.[53] Silk has excellent handling characteristics and knot security and is still occasionally used in oral surgery, but is known to cause substantial tissue inflammation. Host tissue reactions may lead to encapsulation by granulation tissue.[57] Silk has high capillarity and should generally be avoided in contaminated sites.

Suture size

Currently, the common standard for suture size diameter is that issued by the USP. Size is designated by a numerical code, 11-0 being the smallest size available and 7 the largest. The size of the suture material selected should correlate with the tensile strength of the tissue being sutured, and the smallest diameter suture sufficient to adequately appose the wound margins should be used to minimize the trauma incurred from the passage (friction) of the suture through the tissues and the foreign body reaction created by the material that is retained in the wound.[58] Sizes 3-0 and 4-0 sutures are the most commonly used sizes for oral and maxillofacial surgical procedures in humans.[58] In large dogs size 4-0 suture is suitable, whereas size 5-0 suture material is more appropriate in cats and smaller dogs. Some oral surgeons prefer to use 6-0 suture in cats and dogs under 3.0 kg.

Suture needles

The modern suture needle is constructed of corrosion-resistant surgical-grade stainless steel and consists of three basic parts: attachment end (swaged or closed eye), body, and needle point. Needles for use in oromaxillofacial surgery are of the swaged-on variety, where the needle has been permanently crimped onto the suture material, as swaged needles facilitate suture handling and reduce tissue trauma during suturing compared with eyed needles.[58] The body shape of the needle may be straight, curved, or a combination of both (curved-ended straight) (Fig. 8.2). Needles are further divided into cuticular and plastic varieties; the latter is sharpened 36 times and is designed for cosmetic closures.[59]

• **FIG. 8.1** (**A**) Image of a barbed suture. Notice the individual barbs that extend from the core of the suture in a helical pattern, and the looped end that serves as an anchor for the suture. (**B**) A barbed monofilament suture (3-0 Quill Monoderm with reverse-cutting 3/8 circle 18-mm needle; Surgical Specialties Corporation, Wyomissing, PA) in a simple continuous pattern is used to close mandibular premolar tooth extraction sites in a dog. The suture is passed in a reverse direction, across the incision, to finish the suture pattern.

Selection of needle shape is based on accessibility of the area to be sutured and surgeon's preference. For the purposes of oromaxillofacial surgery, the 3/8-circle needle is the most commonly used, as it requires significantly less wrist pronation and supination in comparison with the other commonly used needle in veterinary medicine (1/2 circle). As a general rule, curved needles with a comparatively greater arc (1/2 circle or 5/8 circle) are more suited for suturing small, deep wounds in confined areas (e.g., vestibule or caudal oropharynx).[59] Cutting, tapered, and tapercut are the three basic types of needle points (Fig. 8.2). As their name suggests, cutting needles possess sharp edges (two opposing cutting edges and a third cutting edge along the curvature of the needle) that facilitate penetration through tough keratinized tissues such as the gingiva or skin. Cutting needles may be further subdivided into conventional-cutting and reverse-cutting needles. Although both are triangular in cross-section, the reverse-cutting needle has a flat surface along its inner curvature, preventing the inadvertent cutting of tissue ("cut-out") as the needle is passed through tissue. The reverse-cutting shape also increases the strength of the needle by 32%.[59] Because of the predisposition for cutout with conventional-cutting needles, their use is limited within the oral cavity, and the reverse-cutting needle is the most commonly selected needle for general use in dentistry and oromaxillofacial surgery. Tapered-point needles are round in cross-section and result in less tissue damage than cutting-point needles, but are poorly suited for use in tough tissues, such as gingiva. Tapercut needles combine the cutting action of the reverse-cutting needle at its tip and the round body of the taper for atraumatic passage through delicate tissues. The tapercut needle is recommended for use in mucogingival surgery.

Suture recommendations for specific tissues

Intraoral tissue

Monocryl and Vicryl Rapide in sizes 4-0, 5-0, and 6-0 are currently the most compatible suture materials for intraoral use. For vestibular flap closure following maxillectomy, however, polypropylene has been recommended.[61] Both Monocryl and Vicryl Rapide have rapid absorption times that approximate the optimum time for removal of nonabsorbable suture materials. The benefits of Monocryl include improved tensile strength, knot security, smoothness, and minimal tissue reactivity, previously found only in synthetic monofilament nonabsorbable suture materials. Vicryl Rapide offers the improved handling characteristics of a multifilament, but has a rougher surface and less initial tensile strength and is more expensive and reactive than Monocryl.[54] A simple interrupted pattern is typically used for routine oral wound closures; however, horizontal mattress pattern sutures may be a suitable alternative, in particular when suturing severely inflamed and friable oral tissues, as it allows the needle to be introduced further from the wound edges.[62]

Skin and subcutaneous tissue

The closure of facial skin wounds in the dog or cat is best performed with size 4-0 or 5-0 synthetic monofilament nonabsorbable suture materials such as nylon or polypropylene, whereas in humans, especially in conspicuous areas, size 6-0 suture is typically used to avoid scarring. A simple interrupted suture pattern is recommended for the most cosmetic closure.[26] Skin sutures placed in areas other than the face are traditionally removed at between 3 and 10 days.[59] Facial skin sutures should be removed in 4 to 6 days to prevent epithelialization of the suture track.[59] The subcutaneous tissues should be approximated with size 4-0 synthetic absorbable suture in a simple continuous suture pattern. The use of a continuous suture pattern has the advantage of reducing the quantity of suture buried in the wound.

Surgical adhesive tape

Cutaneous tapes (Steri-Strips Reinforced Adhesive Skin Closures; 3M, St. Paul, MN) are microporous surgical adhesive tapes with a backing of viscous rayon fibers coated with an acrylic copolymer and are available in 1/8-inch, 1/4-inch, and 1/2-inch wide strips.[57] Steri-Strip Antimicrobial Skin Closures (3M, St. Paul, MN) incorporate an iodophor adhesive that provides broad spectrum coverage against common surgical skin infection (*Staphylococcus* spp.) pathogens. The closure of skin wounds with adhesive tape results in excellent wound healing because the skin is not penetrated with a needle, and there is less intrinsic tension on the wound.[26] Approximation of deep tissues cannot be achieved with tape alone, and tape closure is often combined with subcuticular running or interrupted sutures.[26,57] In animals, however, these materials have found little or no application because they are easily removed by the patient. Exudate, skin oils, hair, and moisture negatively affect tape adhesion, result in inadequate adhesion, and are all considered contraindications for the use of tape. Tincture of benzoin can be applied to the skin surface to enhance adhesion.[26]

Surgical staples

Skin clips or staples are useful to expedite closure of scalp or abdominal wounds where cosmesis is less of a concern.[57] Staples maintain wound edges in eversion, but may result in necrosis and edema if used with excessive pressure.[57]

Tissue adhesives

Cyanoacrylates

Cyanoacrylates (PeriAcryl 90; GluStitch Inc., Delta, British Columbia) are liquid adhesives that polymerize in the presence of moisture and have been used for tongue lacerations, extraction wounds, periodontal surgery, to treat dentin hypersensitivity, and to temporarily attach hemostatic dressings to oral soft and hard tissues.[63-65] PeriAcryl 90 is a blend of n-butyl and 2-octyl cyanoacrylates, and is available in a regular viscosity formulation as well as a high viscosity formulation that is nine times as thick as the regular formulation. Cyanoacrylates are well tolerated by oral tissues, promote healing, and shorten surgical times, and they may also have bacteriostatic properties.[65] The butyl and isobutyl cyanoacrylates are the most suitable for use in the oral cavity, and the isobutyl form is the least cytotoxic.[39,66] Butyl or isobutyl cyanoacrylates that are applied to mucogingival tissues are exfoliated in 4 to 7 days; however, only partial phagocytosis of the adhesive occurs when used in deeper tissues, and may result in granuloma formation.[39]

When using cyanoacrylates for the closure of oral wounds, the glue should be applied in multiple thin lines, in layers, and along both sides of the incision. Petroleum jelly can be applied to the glued surface to prevent unwanted adherence.

Fibrin tissue adhesives, patches, and autologous platelet concentrates

Tisseel (Baxter Healthcare Corporation, Deerfield, IL) fibrin sealant is a combination of human fibrinogen and thrombin applied with a sterile delivery device.[67] Within 3 to 5 minutes of delivery, a solid fibrin clot forms that firmly adheres to soft tissues and promotes hemostasis. It is fully absorbed within 10 to 14 days. It has been used extensively in human surgery, including maxillofacial surgery. Applications include mucogingival flap surgery, cancellous bone grafts, cleft palate repair, and oronasal fistula closure.[67,68] It is especially useful for closure of extraction wounds or following cyst enucleation in patients with coagulopathies.[69] Fibrin adhesives do not interfere with wound healing and may in fact promote it.[67,70] Composite fibrin sealant patches (2 × 4-inches) of oxidized regenerated cellulose (ORC) and polyglactin 910 embedded with human fibrinogen and thrombin are also available (Evarrest; Ethicon, Inc., Somerville, NJ); these patches resorb over time in 8 weeks.

Autologous preparations of fibrin sealants may be prepared by centrifugation of a patient's own whole blood or, if larger quantities are desired, from a cryoprecipitate. As a bioadhesive for the repair of oral mucosal defects, autologous fibrin sealants have been shown to be safe and well tolerated in cats.[71] Similar to its xenogenous counterpart, autologous fibrin sealants may be used as a hemostatic, but also serve as a scaffold and a promoter of bone graft healing.[72-74] The substrate provided by the combined interaction of fibronectin, fibrin, and factor XIII encourages the migration and growth of mesenchymal cells, accelerates revascularization, and slows the multiplication of microorganisms.[72] Healing is accelerated with factor XIII, and fibroblast and osteoblast growth are stimulated by fibrin.[72,75,76]

Autologous platelet-rich plasma (PRP) gel is a first-generation modification of autologous fibrin sealants that is formed by mixing PRP from centrifuged autologous whole blood with bovine thrombin and calcium chloride to form a gel.[77] Growth factors are released rapidly from PRP, starting within 10 minutes of platelet activation, with the majority of factors being released within the first hour of activation.[78] This massive release of growth factors from platelets has been theorized to hasten tissue regeneration. Platelet-rich fibrin (PRF) is a second-generation platelet derivative, which is an autologous fibrin matrix containing a high concentration of platelets and leukocytes; it is created using a time-sensitive centrifugation technique pioneered by Choukroun that relies upon natural polymerization in glass centrifuge tubes and does not require use of anticoagulants.[79] Without the addition of anticoagulants, a three-dimensional fibrin scaffold is formed and platelet activation and growth factor release do not occur abruptly as with PRP, but rather occur in a natural fashion, allowing for the steady and slow release of growth factors over the course of 7 to 11 days, in higher numbers than PRP.[80] PRF used at extraction sites was shown to provide good hemostatic properties and improved tissue healing and wound closure compared with extraction sites treated with HemCon Dental Dressing PRO (Tricol Biomedical, Inc., Portland, OR).[81]

Biomaterials for hemostasis

Hemorrhage following flap reflection and exodontia can usually be controlled with aspiration and pressure with moistened gauze, but slow, persistently oozing lesions may require the use of a hemostatic agent. Many hemostatic agents are also used as extraction socket dressings to reduce the volume of the blood clot that is formed and the chance of premature clot dissolution.[82] These agents include bone wax, ferric sulfate, absorbable gelatin sponge, oxidized regenerated cellulose, hemostatic collagen, hemostatic sealants, antifibrinolytics, and polysaccharides.

Bone wax

The present-day formulation for bone wax is a highly purified beeswax that contains isopropyl palmitate as a softening and conditioning agent.[83] The wax functions as a mechanical hemostatic by blocking the vascular openings with plugs of blood and wax.[84] Bone wax is especially useful as a hemostatic during periapical surgery.[83] After hemostasis has been achieved, the bone wax should be removed, as the retained wax will initiate an intense foreign body reaction, characterized by giant cells, plasma cells, and fibrous granulation tissue, that will inhibit osteogenesis.[85,86]

Ferric sulfate

Ferric sulfate (ViscoStat, Ultradent Products, Inc., South Jordan, UT) is a commonly used astringent that arrests capillary bleeding during restorative dentistry and prosthodontic procedures, but may also be used as a pulpotomy medicament, for periradicular and endodontic surgery and, infrequently, to control minor postextraction hemorrhage.[87] The reaction of blood proteins with ferric and sulfate ions creates plugs that block capillary orifices.[87] In dentistry, 15%–20% ferric sulfate is typically used. Ferric sulfate also has an antibacterial effect, most likely due to its low pH and cytotoxicity, but also due to its occlusive effect on capillaries (that is, it blocks bacterial ingress), and is as effective as 0.2% chlorhexidine digluconate.[88] Bone healing may be adversely affected by residual ferric sulfate, and it should therefore be used carefully when controlling osseous hemorrhage.[89]

Absorbable gelatin sponge

Gelfoam (Pfizer Inc., New York, NY) is a porous matrix gelatin sponge prepared from partially hydrolyzed pork skin and intended for use as a hemostatic agent. Gelfoam is provided by the manufacturer in a powder that forms a paste when mixed with a sterile sodium chloride solution. The gelatin sponge is applied directly to the bleeding surface and is absorbed in 4 to 6 weeks.[90] Due to the potential for embolization, Gelfoam should be used with caution in intravascular compartments. The use of Gelfoam in extraction sockets is controversial and has actually been shown to block cancellous bone replacement in dogs.[91] It may also potentiate bacterial growth and serve as a nidus for infection and abscess formation. Cube-sized (1 cm × 1 cm × 1 cm) gelatin sponges (Vetspon; Elanco US Inc., Greenfield, IN) designed for use in extraction sockets are also available.

Oxidized regenerated cellulose

Surgicel (Ethicon, Inc., Somerville, NJ) is an absorbable glucose polymer-based sterile knitted fabric that acts as a matrix for clot formation and as a clot stabilizer.[91] Surgicel is prepared by the oxidation of regenerated cellulose, and its function as a hemostatic agent is dependent upon the bonding of hemoglobin to oxycellulose.[91] Surgicel is also available in a Fibrillar form, which is an easily separated layered construct, and SNoW, which is felt-like; both are as effective as four layers of the original Surgicel. Surgicel is resorbed in 7 to 14 days with minimal inflammation, but can swell by up to 135%, resulting in patient discomfort if used as an extraction site packing material.[92] Surgicel has been marketed to control capillary, venous, and small arterial hemorrhage and, because it does not impede epithelialization, can be used as a surface dressing. Surgicel is also bactericidal (due to a localized reduction in pH) to a wide range of gram-negative and gram-positive aerobes and anaerobes, and has been used successfully as a scaffolding material to fill bony defects (e.g., small to moderate-sized clefts of the palate).[93,94]

Hemostatic collagen

CollaPlug, CollaTape, and CollaPatch (Zimmer Biomet Dental, Palm Beach Gardens, FL) are white, conforming, and non-friable sponge-like collagen products that provide hemostasis primarily by compression (i.e., are held in place for 2 to 5 minutes), but also protect the wound from debris, strengthen the blood clot, and prevent collapse of the tooth socket. These collagen agents are fabricated from bovine deep flexor tendons.[95] Compared with other hemostatic products with a shorter lifespan, these collagen agents may have a resorption pattern as long as 56 days.[95] Avitene (Bard Davol, Inc., Warwick, RI) is an effective microfibrillar collagen hemostatic agent for use on bleeding surfaces and in extraction sockets that is prepared from edible bovine corium as a water-insoluble, partial acid salt of natural collagen.[96] Platelets are attracted, adhere, and aggregate onto the Avitene fibrils. This microfibrillar collagen product is also useful in the face of certain clotting factor deficiencies and heparinization, and has been reported to shorten the reaction time of the intrinsic clotting pathway by 60%.[97] Avitene, however, does not accelerate new bone formation when implanted in bone defects and is more prone to bacterial contamination than oxidized cellulose.[96,98]

Thrombin-based hemostatic agents

Bovine-derived thrombin is available in liquid or powder form as a topical hemostat. Thrombin is an enzyme that is active at the end of the coagulation cascade that converts fibrinogen to fibrin. Thrombin products bypass the need for functional platelets and are useful in thrombocytopenic or thrombocytopathic patients. Since the thrombin is bovine derived, bovine thrombin should be avoided in patients with documented allergic reactions to bovine products.[90] The significance of antibody development to bovine thrombin in veterinary patients is unknown. Synthetic alternatives to bovine thrombin, such as thrombin receptor agonist peptide-6 (TRAP), a synthetic hexapeptide that mimics the effects of thrombin, have been investigated.[99]

Floseal Hemostatic Matrix (Baxter Healthcare Corporation, Deerfield, IL) and Surgiflo (Ethicon, Inc., Somerville, NJ) are both a granular cross-linked collagen-derived matrix that is combined with thrombin to create a flowable gel. In comparison with other topical hemostats (Gelfoam and Surgicel), Floseal granules swell by only 10%–20% upon contact with blood, decreasing patient discomfort when used in extraction sockets. The thrombin that is incorporated with the Floseal granules converts fibrinogen into a fibrin polymer that forms a clot surrounding the collagen-derived matrix. Floseal is completely resorbed by the body in 6 to 8 weeks. In a comparison study of Floseal and Gelfoam plus thrombin for the control of intraoperative hemorrhage, Floseal provided more rapid and effective hemostasis in 93% of first-site applications, compared with 76% for Gelfoam plus thrombin.[100]

Antifibrinolytics (synthetic lysine analogues)

Antifibrinolytic agents increase clot strength, and to a lesser extent decrease clot lysis, by inhibiting plasmin activation through competitive blocking of the lysine binding site of plasminogen. Epsilon aminocaproic acid (EACA) and tranexamic acid (TA), synthetic lysine analogues, are the most commonly used antifibrinolytic agents in dogs. Postoperative use of antifibrinolytic medications has been shown to reduce postoperative hemorrhage in retired racing greyhound dogs with hyperfibrinolysis syndrome.[101] Due to the high percentage of retired racing greyhounds that will likely have delayed postoperative bleeding (26%),[102] perioperative use of EACA or TA is advisable when performing oral surgery in greyhound dogs.

Polysaccharides

Microporous polysaccharide hemosphere (MPH) powder Arista AH (Bard Davol Inc., Warwick, RI) is a hemostatic wound dressing composed of purified cross-linked plant starch (potato) particles ranging in size from 10 to 200 micrometers in diameter.[103] When topically applied to a wound bed, the fluid components of blood (low molecular weight) are absorbed into the controlled-size pores of the particles.[103] The high-molecular-weight solids of blood (platelets, red blood cells, albumin, thrombin, and fibrinogen) are concentrated between and on the surface of the particles to provide a viscous barrier to blood seepage.[103] Manufacturer instructions emphasize the removal of excess dry Arista AH from the surgical site to reduce the possibility of compression necrosis of the surrounding tissues. Hemostasis with MPH can occur as quickly as 30 seconds. MPH particles are degraded by enzymatic hydrolysis within 24 to 48 hours by endogenous alpha amylase.[103]

Another polysaccharide product (HemCon Dental Dressing PRO; Tricol Biomedical, Inc., Portland, OR) is a chitosan dressing derived from the chitin of freeze-dried shrimp exoskeleton. It was modified for oral surgical use from its original function as a combat wound dressing. HemCon Dental Dressing PRO is a sponge-like material that is available in two sizes—10 mm × 12 mm and 1 inch × 3 inch—but can be cut to achieve a custom fit. HemCom Dental Dressing PRO typically dissolves within 48 hours (according to manufacturer instructions, dissolution may take up to 7 days) from the surgical wound, facilitating early hemostasis, while allowing natural wound healing to proceed unimpeded. Chitosan products such as HemCon Dental Dressing PRO are cationic (positively charged) and function primarily through their ability to mechanically seal wounds by attracting red blood cells and platelets, both of which are negatively charged.[104]

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CHAPTER 9

Piezoelectric bone surgery

Philippe Hennet

Definitions

Piezoelectricity: Electricity or electric polarity resulting from pressure and latent heat.
Piezoelectric materials: Crystalline substances that are able to convert mechanical energy into electrical energy, and vice versa.
Piezoelectric bone surgery: Selective cutting of mineralized tissue using a surgical instrument tip that vibrates at a high frequency when electricity is applied to a piezoelectric material.

History[1]

Piezoelectric bone surgery is a recent and innovative technology permitting a selective cut of mineralized tissue while sparing soft tissue. The word "piezo" means "to press, to squeeze" in Greek. Piezoelectricity is a physics property discovered by Pierre and Jacques Curie in 1880 and characterized by the ability of specific materials having a crystalline structure to produce voltage when subjected to mechanical stress. In 1881, Gabriel Lippmann mathematically proved the reverse effect, called converse piezoelectricity, which was experimentally confirmed by the Curie brothers in 1882. This latter effect is the one used to generate vibration of the working tip of dental or surgical instrumentation. Piezoelectric materials can be natural, such as quartz, or manufactured (ceramics). Ceramics are produced by mixing a piezoelectric powder (e.g., lead zirconate titanate) with an organic binder. They can be formed into structural elements with a desired shape (discs, rods, plates, etc.).

In 1948, Alfred Vang proposed the design of a vibratory surgical instrument and filed a patent in 1953.[2] Nevertheless, piezoelectric instruments have been mostly restricted to dental scaling for many years; ultrasonic bone-cutting instruments did not gain popularity until the early 21st century when Tomaso Vercellotti developed the concept of piezoelectric bone surgery. This coincided with the need for more precise osteotomies as the emphasis in maxillofacial surgery extended to involve cortical bone graft collection and surgery of the sinus in preparation for dental implant placement.[3,4]

Technology

The technology is based on converse piezoelectric activity: alternative current applied to piezoactive ceramic discs generates high-frequency vibratory energy.[5] Frequencies of 25–35 kHz are specific for cutting mineralized tissue, whereas soft tissue incisions, such as those performed with the Harmonic piezoelectric scalpel, require frequencies above 50 Hz.[4,6] Similar to a dental scaler, the high-frequency vibration generated by the ceramic pellets contained in the handle of the instruments induces a high-frequency, almost linear reciprocal motion of the metallic tip of the instrument, resulting in up to 300-μm excursions (Figs. 9.1 and 9.2).[6,7] However, the power of the piezosurgical instrument is three to six times higher than that of a regular dental scaler.[8]

• **FIG. 9.1** Piezotome Cube. Source: (Courtesy Satelec/Acteon group, Merignac, France.)

• **FIG. 9.2** Details of the piezoelectric handpiece depicting the six ceramic rings. Source: (Courtesy Satelec/Acteon group, Merignac, France.)

Advantages and limitations

Historically, bone cutting has been performed with manual (osteotomes, bone chisels, rongeurs) or powered (rotary burs, oscillating saws) instruments. Manual bone-cutting instruments require use of high forces, which may lead to bone fracture and soft tissue lacerations that can be accompanied by severe bleeding. Cutting with rotary drills or oscillating saws generates heat and may entrap surrounding soft tissues during osteotomy, leading to severe damage to muscles, nerves, and blood vessels, especially at sites with difficult or limited access. Physical or thermal injury to the bone may result in cell death, lack of regeneration, and bone lysis.[9,10] The degree of thermal injury to the tissues is related to parameters of the instrument (design, diameter, sharpness) and to parameters of bone cutting (speed, feed rate, power, depth of cut, cooling, clearance of bone chips, bone density, and thickness).[9,11-13] Compared with these conventional osteotomy techniques, piezoelectric bone surgery demonstrates many advantages in maxillofacial surgery (Box 9.1).

• Box 9.1

Advantages of Piezoelectric Bone Surgery

Selective cutting of mineralized tissue
Significant reduction of soft tissue trauma
Reduced hemorrhage (cavitation effect)
Excellent visibility within the surgical field, due to minimal bleeding, effective irrigation, and high luminosity LED lights
Precise cutting (limited vibration amplitude and specific design of osteotome tips)
Curvilinear cutting

Lack of thermal damage
Sterile irrigation—steam sterilization of all equipment components

Cutting efficacy

The instrument selectively cuts mineralized tissue a frequencies of 25–35 kHz. When in contact with soft tissue, it vibrates without rupture at the same frequency as the tip of the instrument, dramatically reducing trauma to nerves and vessels.[4,6,14] The cutting power is influenced by bone density, tip characteristics (alloy, shape, sharpness), and working pressure.[4,6] Piezoelectric tips do not require pressure on bone to be effective and, therefore, thermal injuries and bone microfractures are minimized.[3,6,15,16] A cutting rate of 0.25–0.3 mm per second has been demonstrated on bovine femur shafts when piezoelectric units were used at maximum power with irrigation at 50 mL per minute.[10] Though many studies are reporting a longer cutting time with piezoelectric instruments than with rotary burs or oscillating saws,[3,7,15,17-20] especially when the surgeon is learning this technology, a recent metaanalysis comparing piezoelectric surgery with conventional osteotomies for orthognathic surgery found no difference in operative time.[21] In the veterinary field, when performing ventral slot surgery in dogs, the mean operative time with piezoelectric instruments was significantly shorter than with surgical burs (23.4 versus 34.1 minutes).[22]

In contrast to conventional bone surgery with rotary burs or saws where the cutting efficiency is related to the pressure on bone, piezoelectric osteotomy is due to the high-frequency vibration of the tip of the instrument. A moderate axial force close to or a little higher than that used for handwriting has been reported to provide the best results.[10,23-28] Excessive pressure prevents vibration, decreases efficiency, and generates frictional heat.[25] Clinical studies have shown that the optimum force required to cut bone depends on the type of bone (cortical or cancellous), its density (degree of mineralization), and the type of ultrasonic tip used.[10,24-26,29,30] The surgeon must therefore become accustomed to the cutting action of these new instruments and develop a tactile sense of how much pressure is required for different clinical situations.

Safety on vascular and neurological structures

Piezoelectric surgery has been shown to reduce nerve disturbances compared with conventional osteotomy during orthognathic surgery in humans.[21] However, direct application of a piezoelectric surgical tip for 5 seconds on a peripheral nerve with a relatively high working force (1.5 N) has been shown to induce some structural and functional damage to the nerve without dissecting it.[23] The extent of damage was significantly higher with application of increased force on the nerve by the device, but not by activation of the ultrasonic vibration.[23] The efficacy and safety of piezoelectric bone surgery has been assessed in neurosurgery where it has been shown that osteotomy of parietal bone of the cranial vault could be achieved without trauma to the dura mater.[16]

A marked reduction in intraoperative bleeding, up to 30%, is observed during maxillofacial surgery with piezoelectric instruments compared with surgical burs or oscillating saws.[7,8,21] Reduction in blood vessel damage and subsequent blood loss may be due to better visibility, to the sparing effect on soft tissues, and to the cavitation effect associated with ultrasonic instrumentation.[17,31]

Improved postoperative outcome

A significant reduction in postoperative edema, hematoma, trismus, and pain has been observed in humans when mandibular third molar extraction or orthognathic surgery is performed with piezoelectric instruments, compared with an oscillating saw or a rotary bur.[7,32] Under experimental conditions, mean intraosseous temperatures increased between 1.2 and 3.1°C within bovine cortical bone, with the piezoelectric unit producing less heat.[10]

The temperature reached within bone is well below critical levels for intraosseous hyperthermia (40–41°C) and bone necrosis (47°C).[33] The degree of thermal bone damage is directly proportional to the resulting temperature at the cutting site and the period of time the bone is exposed to that temperature.

Speed of osteotomy

Piezoelectric bone surgery is usually considered to be slower than osteotomy with a bur or with an oscillating saw.[3,7,15,18] Nevertheless, constant improvement of this new technology by manufacturers has led to more efficient instruments, which minimizes this problem.[17,18,34] Piezoelectric surgery is associated with an initial learning curve. It takes time to learn how to apply minimal pressure so as to allow the tip to vibrate effectively during the osteotomy.[7,18,34]

Oral and maxillofacial clinical applications

Instrumentation

A variety of piezosurgical units are commercially available. Though there might be differences in efficacy, there are very few studies comparing them.[10] Most manufacturers produce several tips differing in shape, length, and design so as to adapt to specific anatomic regions or types of surgery. The tip may be sharp (scalpels and scrapers), serrated (saw), or diamond coated (Fig. 9.3). Serrated tips are most efficient to cut cortical bone. Diamond-coated tips cut by abrasion, and therefore may be considered less aggressive and safer on thin bone or in the vicinity of soft tissue; they are commonly used for sinus surgery in order to minimize the risk of mucosal tear.[8,29] This would also be a helpful feature to prevent penetration into the nasal cavity when performing apicoectomy of a maxillary canine tooth. Flat sharp tips (scalpel-like) are intermediate and are usually used to scrape cortical bone, to cut bone, or to perform osteoplasty. The tip is firmly screwed to the piezosurgery handpiece using the wrench provided by the manufacturer. Each manufacturer produces its proprietary tips. The handpiece is equipped with a sterile irrigation system and high-luminosity LED lights, which improves visibility at the surgical site even in deep locations. The sterile irrigation system of the piezoelectric handpiece helps clear the surgical field, removing bone chips, dissipating heat due to friction, and improving hemostasis through the cavitation effect.

A

B

C

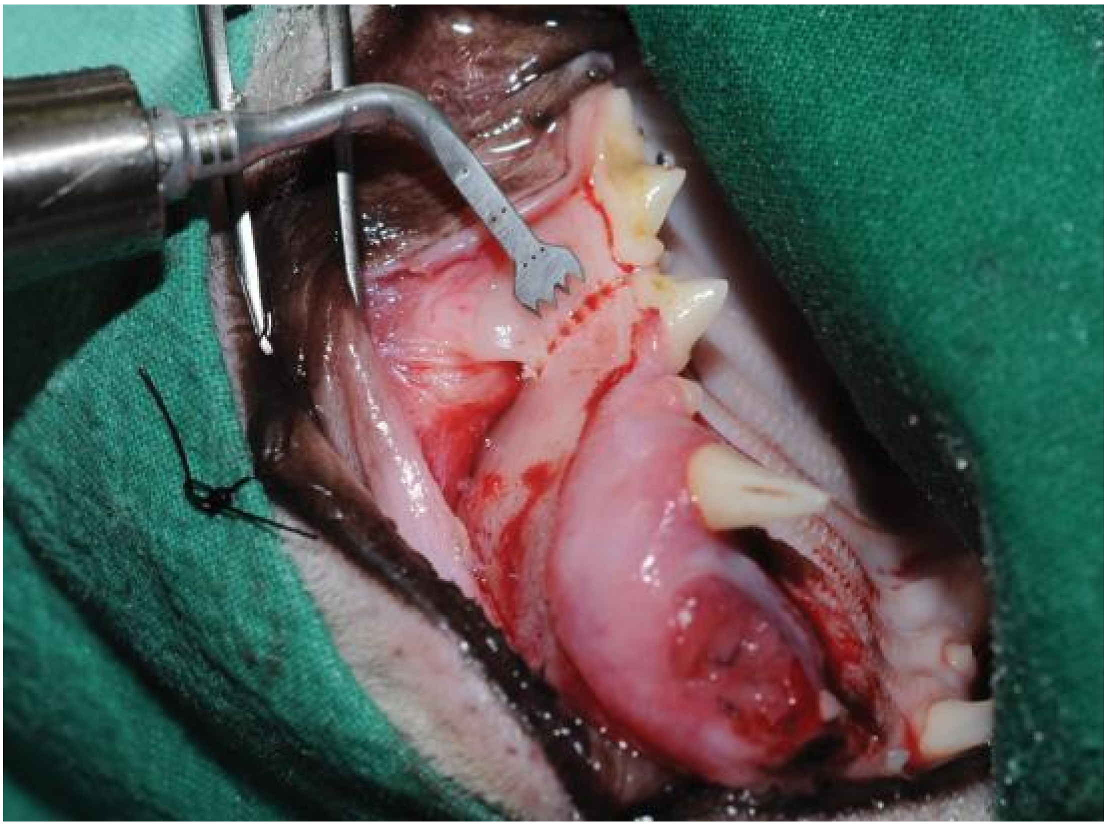
D


SL1
E

• **FIG. 9.3** Specially designed tips for piezoelectric bone surgery. (**A, B**) Serrated tips. (**C, D**) Sharp tips. (**E, F**) Diamond-coated tips. Source: (Courtesy Satelec/Acteon group, Merignac, France.)

Clinical use

The various and versatile tips enable the surgeon to perform osteoplasty and osteotomy procedures on different bone densities as well as to harvest bone by scraping.[35] Because of the thin tips, precise curvilinear osteotomies can be performed, improving the design of bone excision in extensive or reconstructive surgery while sparing surrounding structures. Cutting of dense cortical bone is better performed with a sharp, serrated (saw-like) tip. The tip is held perpendicular to the bone surface, and with copious irrigation (50 mL per second), the surface of the bone is penetrated with the tip in a press-cut action to form a punctuated line (Fig. 9.4) Then, the tip is moved back and forth along this punctuated line to create a groove. In more difficult areas, the bone can be cut to full depth. In deep cuts, care must be taken that the irrigation fluid reaches the tip and intermittent motions are used. The rounded serrated tip may facilitate osteotomy in difficult areas; as the tip cuts within the bone, the sides of the tip are active. The tip should not be angled laterally during the cutting to avoid breakage and must be kept at right angle to the surface. To lessen the risk of thermal damage, continuous motion and pressure on bone should be avoided, especially in deep cuts.[8,29] Intermittent motion applied to the bone with light pressure and copious irrigation helps prevent heat build-up.[33]



• **FIG. 9.4** Press-cut action with a serrated tip to draw a punctuated line delineating the osteotomy site for rostral maxillectomy in a cat. Note that the infraorbital neurovascular bundle is reflected away from the ostectomy site, which directly approaches the infraorbital foramen.

Clinical applications

Piezosurgery in humans was first developed for dental implantology and maxillofacial surgery. More recently, applications for ear-, nose-, and throat surgery, neurosurgery, and orthopedic surgery have been documented in humans.[5,6,8,14] Though its use in veterinary dentistry and oromaxillofacial surgery is becoming more widespread, literature on the subject is sparse.[4,22,36,37]

Dentistry

Exodontics

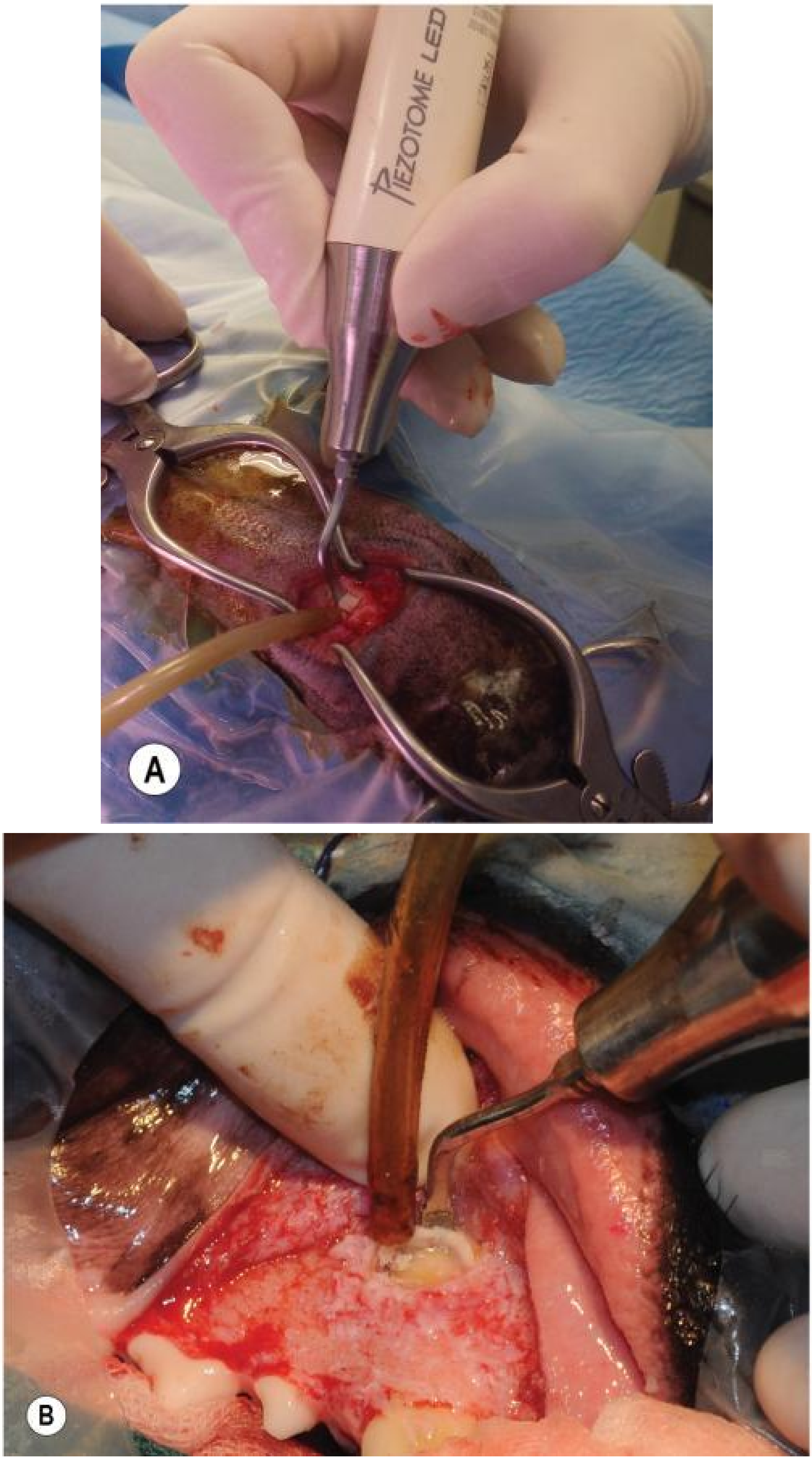
The piezosurgical unit can be used for osteotomy and tooth sectioning in the case of impacted teeth or when performing an extraction with very limited access (restricted mouth opening due to a disease process or an abnormality). In such cases, it is much safer than the use of a rotary bur. For conventional extractions in dogs and cats, it is less clear whether piezosurgery has advantages over traditional techniques.

Periodontal bone surgery

Piezoelectric instruments can be used to perform ostectomy/osteoplasty and to harvest autologous bone graft for reconstructive periodontal bone surgery. Its use in a narrow surgical site is safer than that of rotary burs and allows a precise and more conservative approach. Sharp tips can be used as a scraper and diamond-coated ones are very efficient to perform precise bone contouring (see Fig. 9.3). They can be used with a pen-like motion or with a sweeping motion on the bone surface to cut or reshape bone.

Endodontic surgery

The entire apicoectomy procedure can be safely performed with piezosurgery instruments. Such use has been reviewed in human literature.[38] Osteotomy for apex exposure can be performed either with a serrated tip in thick and dense ventral mandibular bone or with a curved scalpel-like tip on the thinner maxillary alveolar bone (Fig. 9.5A). Once the root tip has been exposed, scalpel-like and diamond-coated tips allow a more conservative osteotomy (see Fig. 9.5B). Root excision can be performed with the serrated tip instead of using a fissure bur, and the cut surface of the resected root can be smoothed with a flat diamond-coated tip. Apical root canal preparation can be further performed with conventional retrograde ultrasonic tips. In such a way, the entire procedure is conducted using piezoelectric ultrasonic instrumentation.



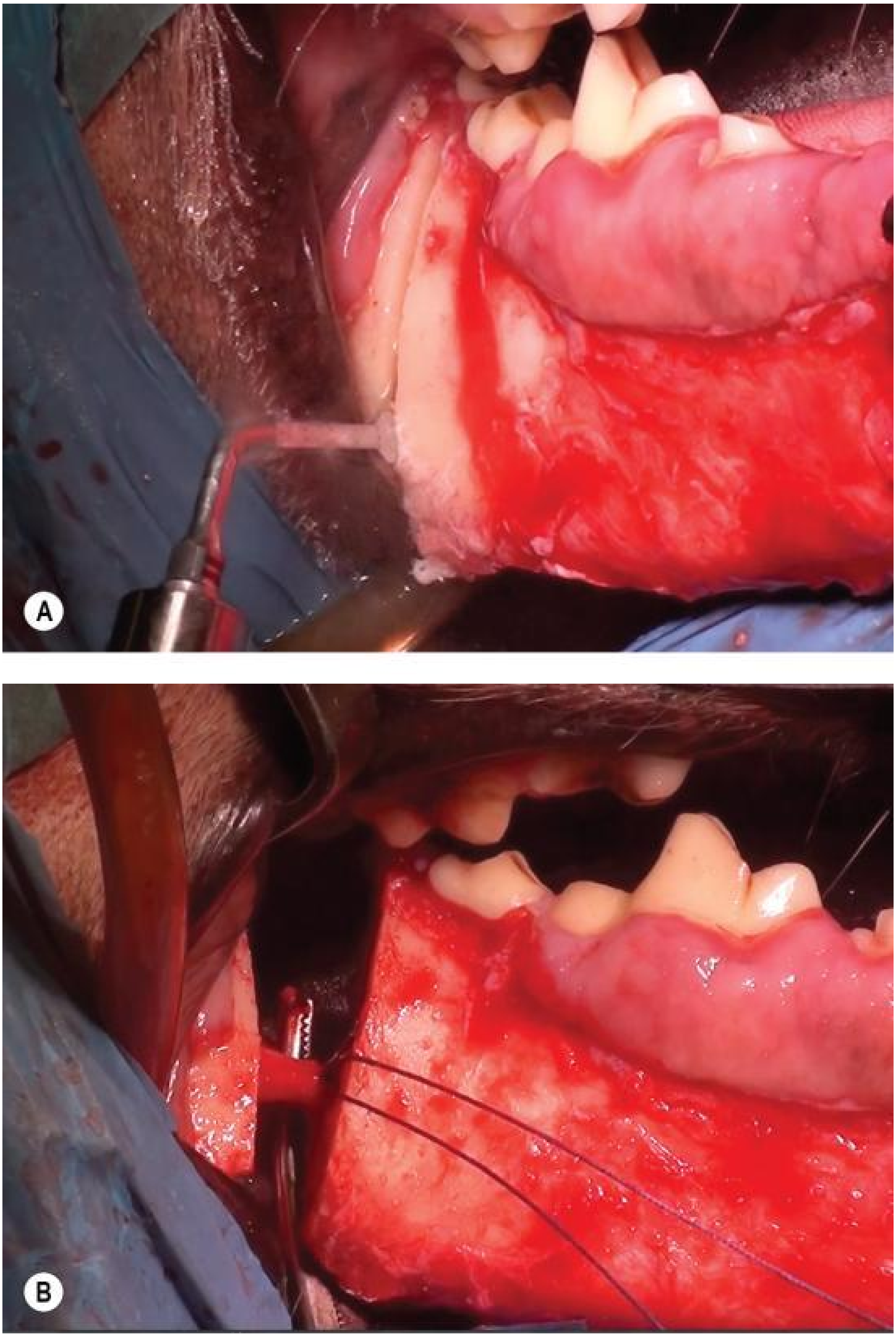
• FIG. 9.5 Endodontic surgery. (**A**) Bone trephination with a serrated tip to expose the apex of a mandibular canine tooth (ventral approach). (**B**)

Osteotomy/osteoplasty with a diamond-coated tip to expose the apex of a maxillary canine tooth.

Oromaxillofacial surgery

Mandibulectomy

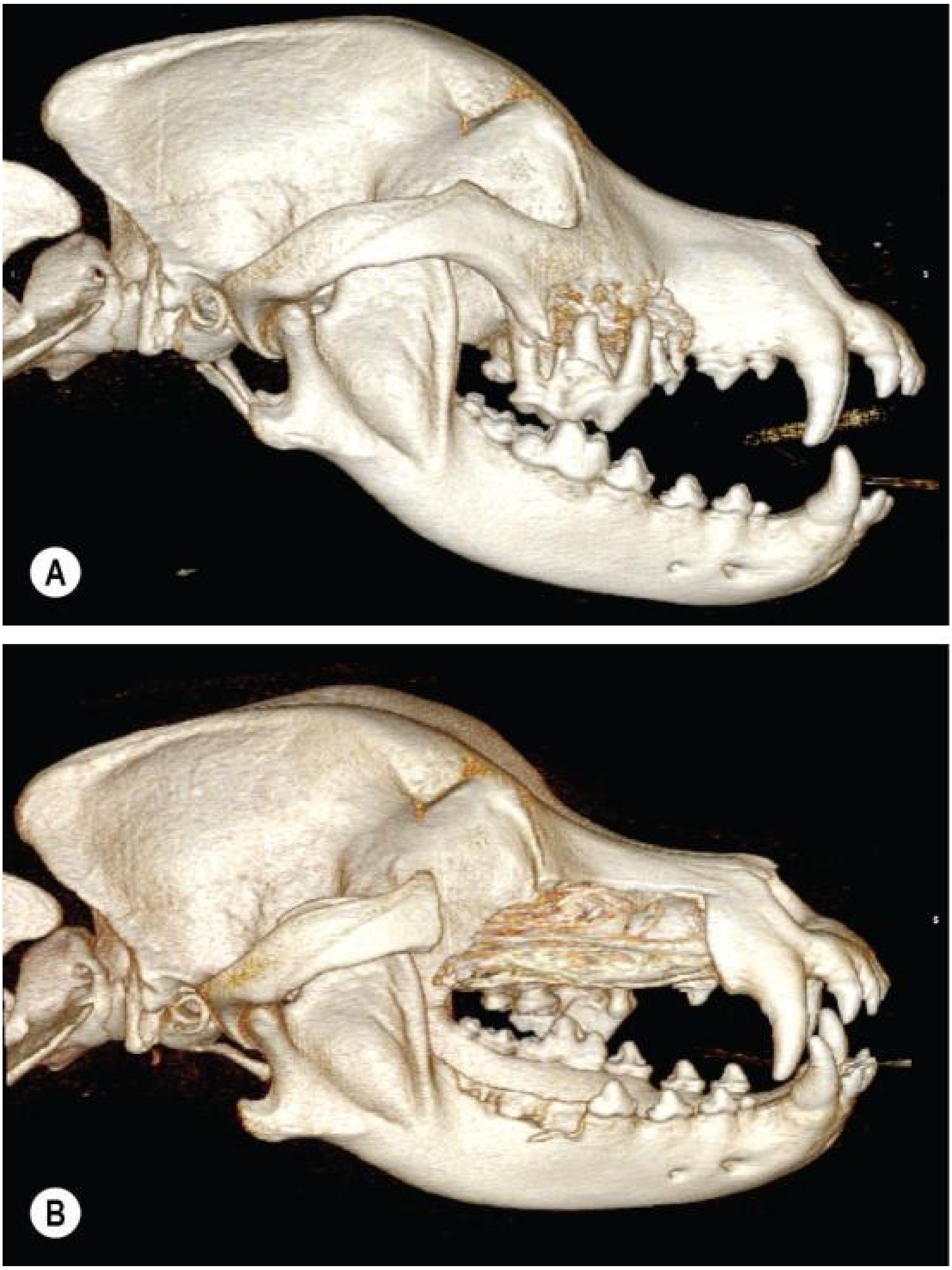
Though considered a little bit slower than rotary burs, piezoelectric surgery is a very precise and safe means of performing mandibular osteotomy without severing the neurovascular bundle within the mandibular canal. As the cortex of mandibular bone is dense and thick, especially in large-breed dogs, the preferred instrument is the serrated tip. To speed up the process, the osteotomy can be initiated with a surgical bur and finished with the piezoelectric tip once the vicinity of the mandibular canal has been reached. Direct contact of the piezoelectric tip with the nerve or vessels does not directly induce damage unless uncontrolled and excessive pressure against these structures is exerted. Once the osteotomy of the mandibular body is performed, the rostral and caudal parts can be separated to expose the blood vessels and nerve, which can be ligated and transected (Fig. 9.6). In the rostral part of the mandible, the ability of the piezoelectric instrumentation to perform precise curvilinear incisions may allow a more conservative approach, with preservation of the caudal part of the symphyseal attachment and/or of the neurovascular bundle exiting the mental foramina.



• **FIG. 9.6** Subtotal mandibulectomy. (**A**) A serrated tip is used to cut the thick cortex of the mandibular body without severing the mandibular neurovascular bundle. (**B**) Identification and ligation of the preserved blood vessels.

Maxillectomy

Maxillectomy is typically associated with more profuse bleeding than mandibulectomy, as the nasal cavity or the orbital area is entered. Because of the complex anatomy and the rich blood supply, caudal maxillectomy is the most challenging jaw surgery (Fig. 9.7). Piezoelectric bone surgery is the technique of choice in these difficult surgical sites. Because there is less risk of soft tissue entrapment and laceration of blood vessels, blood loss is reduced, and visibility of the surgical site is improved. The irrigation and cavitation effects also contribute to hemostasis. The osteotomy line can be precisely adapted to the anatomical features of each patient, which varies considerably with skull conformation.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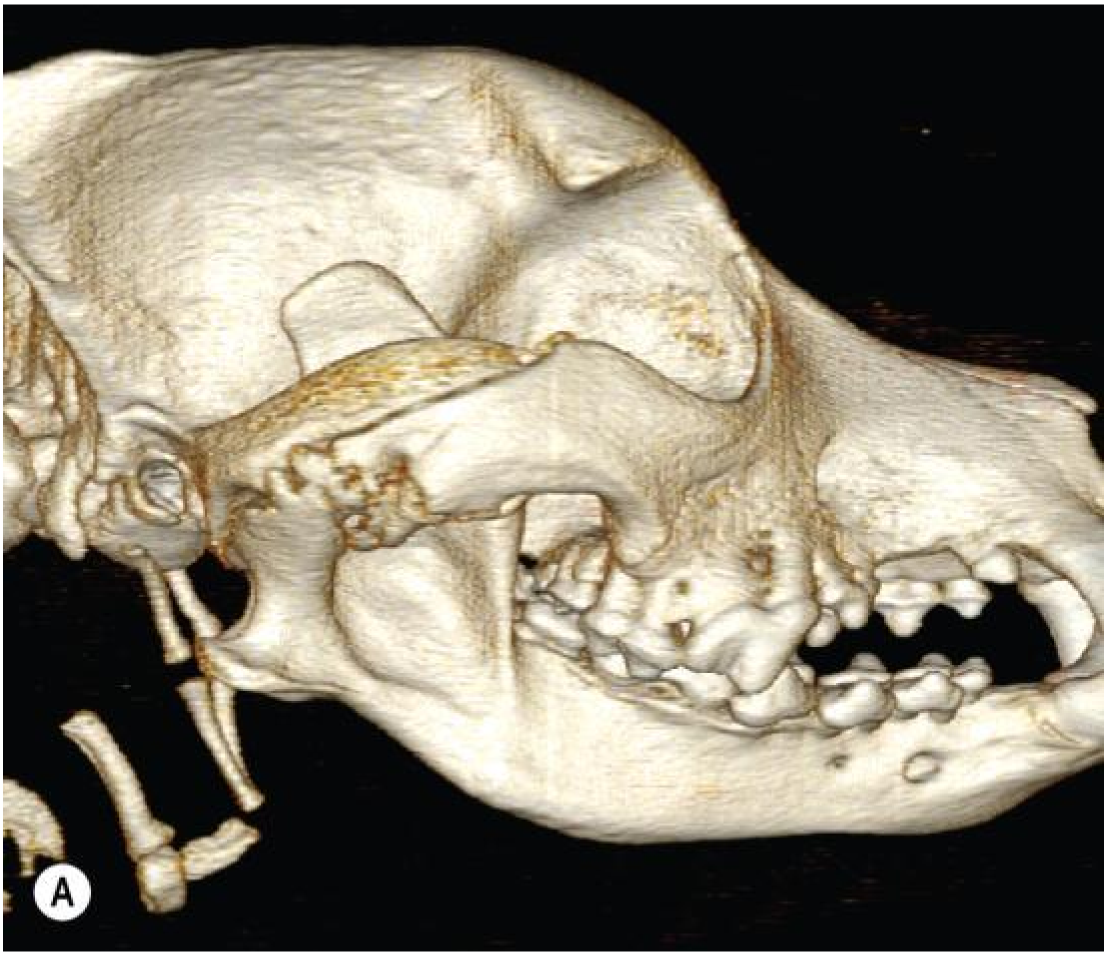
B

• **FIG. 9.7** Tridimensional volume rendering created from computed tomography images of a 9-year-old dog with a maxillary osteosarcoma, before and after maxillectomy performed with a piezoelectric bone surgery unit. (**A**) Preoperative lateral view. (**B**) Lateral and (**C**) ventral views following maxillectomy-orbitectomy with piezoelectric bone-cutting instrumentation.

Temporomandibular joint surgery

Temporomandibular joint (TMJ) surgery is complex, as the surgical site is deep and narrow with a condylar process transversely elongated, poorly accessible and surrounded by a rich blood supply. The maxillary artery is in close proximity to the medial aspect of the joint. The main indication for TMJ surgery is TMJ ankylosis, which may result from maxillofacial trauma, craniomandibular osteopathy, or severe osteoarthritis with osteophyte formation. In patients with TMJ ankylosis, the normal anatomy is often distorted by severe bone remodeling and fusion. Bony fusion may involve the zygomatic arch and the ramus of the mandible in addition to the TMJ proper. In the past, condylectomy has been performed with power (burs or saw) and hand (chisel and rongeurs) instruments. Osteotomy is imprecise with such instrumentation, with a high risk of blood vessel laceration, incomplete removal of the condylar process, and insufficient gap osteotomy. Piezoelectric bone-cutting instruments offer a distinct advantage for TMJ surgery (Fig. 9.8). The LED illumination, continuous irrigation, precise cutting, and preservation of vascular structures allow a more effective and safer surgical procedure. Recently, this advantage was documented in the treatment of TMJ ankylosis in humans.[39] Disorders of the TMJ and their treatment are further discussed in Chapters 36–38.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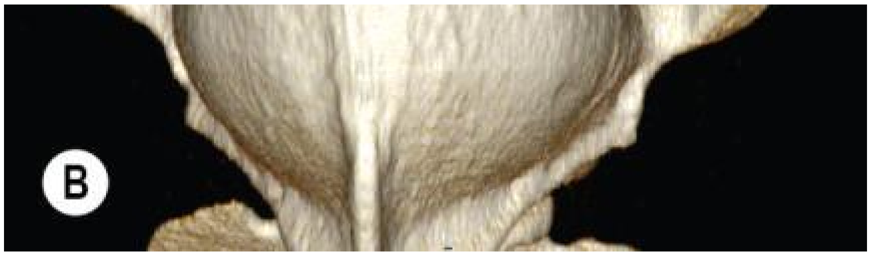
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SAG
C

• FIG. 9.8 (**A**) Lateral and (**B**) ventral views of tridimensional volume renderings created from computed tomography images depicting the severe bony fusion affecting the temporomandibular joint, the ramus, and the zygomatic arch. (**C**) Lateral and (**D**) ventral views of 3D volume renderings showing precise ostectomy of the caudal mandible and zygomatic arch achieved with piezoelectric bone-cutting instrumentation.

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CHAPTER 10

Laser surgery

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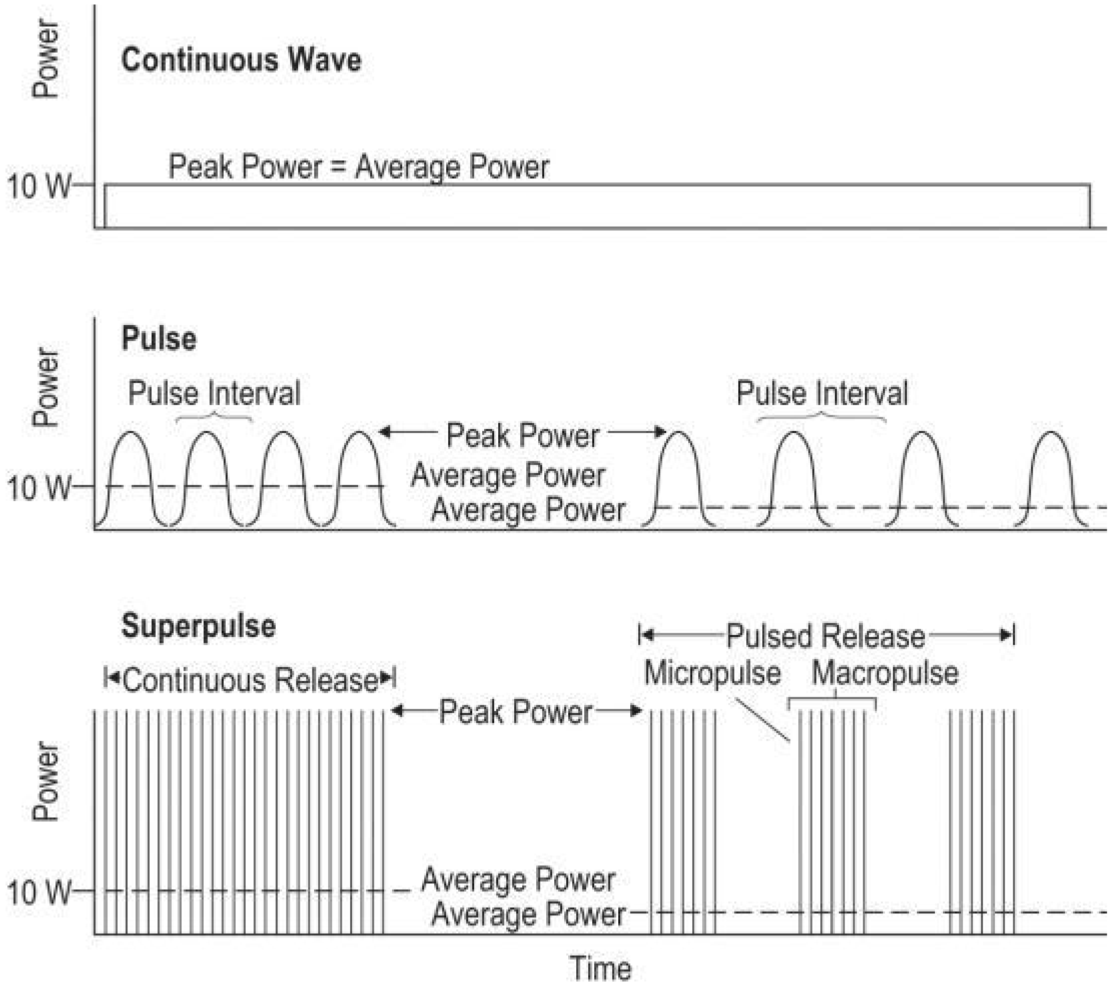
Definitions

The word *laser* is an acronym for light amplification by the stimulated emission of radiation, which is a description of how a laser beam is generated. *Photons* are the form of electromagnetic energy of which light consists. A photon travels in a straight line, undulating in a waveform that is determined by its energy level. The waveform is characterized by the distance between the crests of two successive wave peaks, referred to as the *wavelength*, reported in nanometer (nm) or micrometer (μm). Light is further characterized as being *ultraviolet* (UV) (100–400 nm), *visible* (400–750 nm), or *infrared* (IR) (>750 nm).[1-4]

The light emitted from a laser is *monochromatic* (one wavelength), *coherent* (photons traveling parallel and in phase with each other), and *intense* (photons confined within a beam of small diameter). This differs from incandescent light sources, where the emitted light is polychromatic (multiple wavelengths), noncoherent (photons travel in many directions away from the source), and not intense (photons distributed over relatively large area).[1-3]

Energy is a measure of work and is reported in *joules* (J). *Power* is a measure of the rate that work is taking place and is measured in J/s and reported in *watts* (W). *Energy density* (also referred to as *fluence*) is a measure of the total amount of energy distributed within a defined area, expressed as J/cm^2. *Power density* (also referred to as *irradiance*) is a measure of the rate of work within a defined area (W/cm^2) and is used to describe the concentration of the power output of the laser as delivered within the area of the beam (πr^2) at a specified beam diameter.[5]

When the beam of photons released from a laser is continuously emitted during the period of activation, the beam is delivered in a *continuous wave mode*. When the beam is emitted in successive bursts of energy of programmed duration, the beam is delivered in a *pulsed mode*. Each individual pulse is emitted for a specified duration of time referred to as the *pulse duration* or *pulse width*. When pulses are released in a successive stream, they are emitted in series at a specified time interval referred to as the *pulse interval*, that is, the time between the onset of two successive pulses (Fig. 10.1), or may be specified as the *pulse repetition rate* (pulses/s) or *pulse frequency* (Hz).



• **FIG. 10.1** Continuous wave mode, pulse mode, and superpulse mode (see text). Source: (From Peavy GM. Laser and laser-tissue interaction. *Vet Clin North Am Small Anim Pract.* 2002;32:517–534. With permission.)

During an individual pulse, the energy release starts at zero, increases to a maximum or *peak power*, and then decreases back to zero. During a pulsed laser exposure, energy release occurs during the pulse duration and does not occur between the end of one pulse and the start of the next successive pulse, so the *average power* delivered to tissue over time is the total energy of one pulse divided by the duration of one pulse interval (Fig. 10.1). For a pulse structure where 0.1 J is evenly delivered within a 1 ms pulse with a pulse interval of 4 ms, the peak power of each pulse is 100 W (i.e., 0.1 J/1.0 × 10^{-3} s), while the average power delivered to tissue is 25 W (i.e., 0.1 J/4.0 × 10^{-3} s).

When beam delivery is made in a succession of very short duration, high frequency, high peak power pulses, the delivery may be referred to as a *superpulse mode*. When a succession of very short-duration pulses are delivered for a set time interval, stopped, and then repeated for the same time interval, an individual short pulse is referred to as a *micropulse* and the interval of successive micropulse exposures is referred to as a *macropulse* (Fig. 10.1). A typical superpulse mode would be individual 100 W peak power micropulses of 700 μs each, delivered at a frequency of 200 Hz (200 pulses/s). If the micropulses are released in succession for a period of 1 ms at specified intervals, the micropulse duration is 700 μs and the macropulse duration is 1 ms. The peak power during the laser exposure of each micropulse is 100 W, while the average power delivered during the macropulse is 14 W (i.e., 100 J/s × 700 × 10^{-6} s/micropulse × 200 micropulses/s). If a 1 ms macropulse is delivered every 4 ms, the peak power of each micropulse remains the same (100 W); however, the average power delivered to tissue is 3.5 W (i.e., 14 W × 1 ms/4 ms).

System selection and application

There are four variables that influence both the mechanism of laser–tissue interaction and the resulting effect on tissue. These variables are wavelength, beam intensity, time domain of energy delivery, and tissue handling.[5-13] It is important to understand the principles of these variables not only for the selection of an appropriate laser system but also to understand how variations in the parameters of beam delivery for the selected system will alter the effects of the application of that laser to tissue.

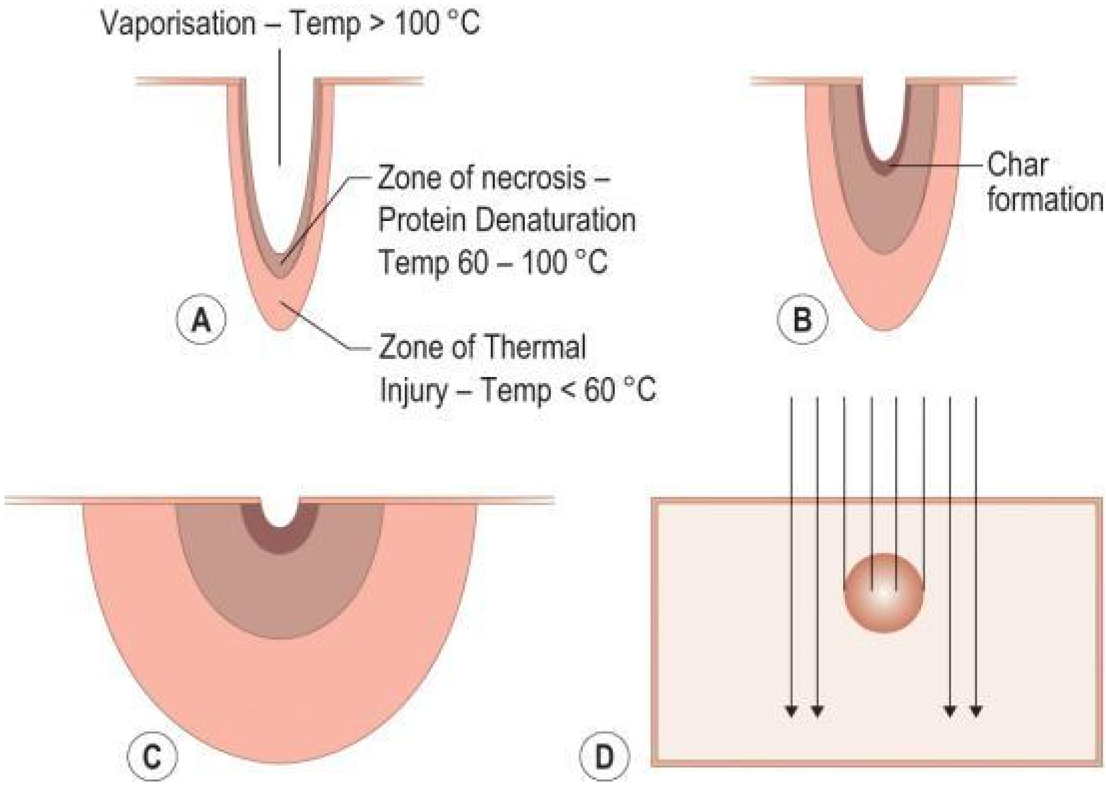
For any medical laser system, the principal operator is responsible by law for applying the principles of laser safety and for the training of all laser users and support staff in the practice. This includes (1) standard operating procedures, (2) biological effects of laser radiation on the eye and skin, (3) hazards presented by reflections and inadvertent misdirection of the laser beam, and (4) control measures. This subject is beyond the scope of this text, but its understanding is an essential requirement prior to using a laser.[14-17]

Laser–tissue interaction

Mechanisms of action

For most surgical laser systems, the mechanism of action for cutting or ablating soft tissue is photothermal (photon energy is absorbed by tissue and transformed into thermal energy that then affects the tissue). Where tissue temperature reaches 60–65°C, proteins are denatured, and that area of tissue no longer remains viable. Where tissue temperature reaches or exceeds 100°C, water in the irradiated area expands, creating pressure within the tissue that produces an explosive vaporization event when the internal pressure exceeds the strength of confinement.[11,13,18]

Where photothermal vaporization has been induced, a crater is created by explosive ejection of tissue. This tissue defect may be surrounded by two to three zones of altered tissue (Fig. 10.2), the presence and size of which will depend upon the factors that influence both photon and thermal confinement.[9,11] A thin layer of carbonization lining the crater wall may be encountered as the first zone of thermal injury. The carbonization represents an area where the tissue temperature greatly exceeded 100°C and tissue composition has been broken down into its elemental parts; however, an explosive vaporization event either has not been induced or has been inadequate to carry away all of the debris.



• **FIG. 10.2** (**A**) When photons of a specific wavelength are absorbed well by a target tissue, they remain confined to a small volume of tissue and rapidly raise the internal tissue temperature to the ablation threshold, minimizing collateral thermal injury caused by photon dispersion. (**B**) and (**C**) When a wavelength is poorly absorbed by the target tissue and photons are dispersed across a large area, or when a well-absorbed wavelength is delivered at a low irradiance that permits diffusion of thermal energy into surrounding tissues before an ablation threshold is reached, shallower areas of vaporization and wider areas of collateral thermal injury are produced. (**D**) Selective photothermolysis uses a wavelength that is transmitted by one tissue to photocoagulate an absorbing tissue located within or under the transmitting tissue. Source: (From Peavy GM. Laser and laser-tissue interaction. *Vet Clin North Am Small Anim Pract* 2002;32:517–534. With permission.)

The next area of adjacent thermal influence is a zone of tissue necrosis, where tissue temperature has been elevated above 60–65°C but has not reached the vaporization threshold. In this zone, protein denaturation has occurred, and while the tissue does retain some structural integrity, it is no longer viable. The third region of collateral thermal injury represents tissue that has been heated, but not high enough or long enough for protein denaturation to occur. This zone of tissue warming will retain viability and will be incorporated in the process of wound healing.

In most cases, one wants to maximize tissue vaporization and minimize collateral thermal injury to achieve the highest efficiency of tissue removal while minimizing injury to adjacent tissue. In some cases, an element of collateral thermal influence is desired, particularly where hemostasis is a consideration. Variations in wavelength selection, beam intensity, time domains of energy delivery, and tissue handling will influence both photon and energy confinement and, thereby, the resulting tissue removal and adjacent tissue injury.

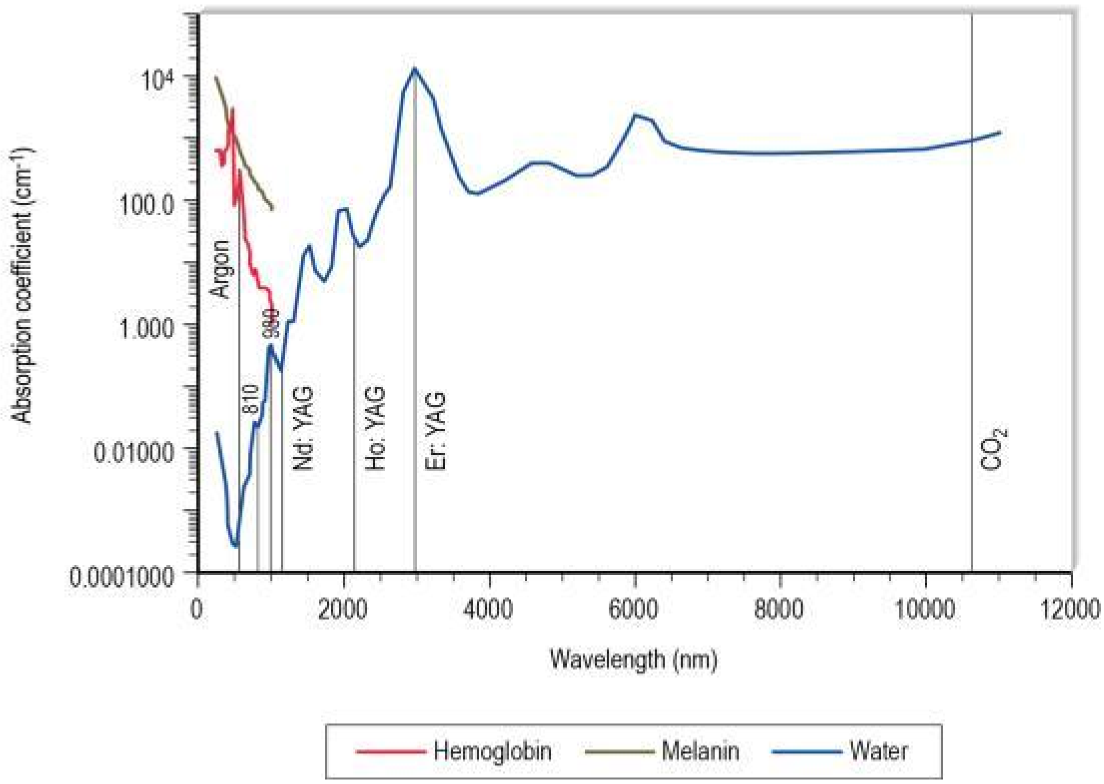
Wavelength selection

There are many different laser sources available, each of which generates a specific wavelength or selection of wavelengths, including excimer (108–353 nm), argon (488 and 514 nm), krypton (647 nm), dye (400–780 nm), semiconductor diode (635–1550 nm), Nd:YAG (1064 nm), Ho:YAG (2120 nm), Er,Cr:YSGG (2780 nm), Er:YAG (2940 nm), and CO_2 (10 600 nm).[4,19,20] Wavelength selection is an important consideration, as different wavelengths can have different effects on the same soft tissue. The wavelength-dependent effects on tissue are influenced by the optical properties of the specific tissue and to what extent those properties reflect, transmit, scatter, and/or absorb the incident wavelength.[11,13]

Maximum ablation efficiency occurs when the incident wavelength is highly absorbed by the target tissue, which maximizes energy concentration by confining the photons and their subsequent conversion to thermal energy within a small volume of tissue. When the incident wavelength is poorly absorbed by tissue, photons are more widely disbursed through the tissue before being absorbed. As a result, the subsequent conversion of photon energy into thermal energy is distributed over a larger volume of tissue, making it more difficult to generate temperatures that reach the ablation threshold, resulting in the creation of small ablation craters and large areas of collateral thermal injury.

The components of soft tissue that most commonly serve as photon-absorbing agents are hemoglobin, melanin, and water. The distribution of photon absorption in tissue is dependent upon the relative absorption of the incident wavelength by these compounds and both the concentration and distribution of each compound in the tissue.

Hemoglobin and melanin are good absorbers of wavelengths in the UV to visible blue-green (400–500 nm) region, with decreasing absorption in the near-IR region. Water is a poor absorber in the visible to near-IR regions but has very good absorption characteristics in the mid- to far-IR regions. While the absorption coefficients for hemoglobin and melanin in the blue-green region are comparable to the absorption coefficients for water in the mid- to far-IR region, the higher concentration of water in soft tissue makes the mid-IR Er:YAG (2940 nm) and the far-IR CO_2 (10,600 nm) lasers more efficient for soft tissue surgery. Near- to mid-IR wavelengths (diode, Nd:YAG, and Ho:YAG) are poorly absorbed by water, allowing them to be delivered through optical fibers, but they are similarly poorly absorbed by tissue (Fig. 10.3).



• **FIG. 10.3** Absorption coefficients for water, hemoglobin, and melanin plotted for wavelengths with the wavelength emission of common laser systems noted. The larger the absorption coefficient value, the better the wavelength is absorbed by the substance. The coefficient values are plotted in log scale as their values range across eight orders of magnitude. Source: (From Peavy GM. Laser and laser-tissue interaction. *Vet Clin North Am Small Anim Pract* 2002;32:517–534. With permission.)

Efficient ablation of dental hard tissues is best achieved by wavelengths that are absorbed well in water and in hydroxyapatite, which are important constituents of enamel, dentine, cementum, and bone. Pure thermal ablation mechanisms are enhanced by thermochemical effects caused by high-pressure water vapor on the weakened hard tissues. Lasers producing this dual effect include the Er:YAG and the Er,Cr:YSGG lasers. Generally, thermal damage to collateral tissues during laser ablation is minimized by the use of a carefully timed and calibrated water-cooling spray. This is important because of the extreme thermal sensitivity of some dental structures such as the dental pulp and periodontal tissues.

Beam intensity

While the power output of the laser is set at a specific level, the surface area of the beam over which the photons are distributed will have a profound effect on the concentration of photons as they reach the tissue, as well as the resulting effect of the beam on the tissue.[5] Power is the selected energy output of the laser (W) and is divided by the area of the beam (πr^2) to calculate the power density ($W/\pi r^2$). Table 10.1 shows the influence on power density of increasing the power output of the laser in comparison to the influence of changing the spot size. The power density of a fixed beam diameter changes linearly as the power output of the laser is adjusted, but when the power output is kept constant and the beam diameter is changed by a factor of 2, the power density is inversely changed by a factor of 4.

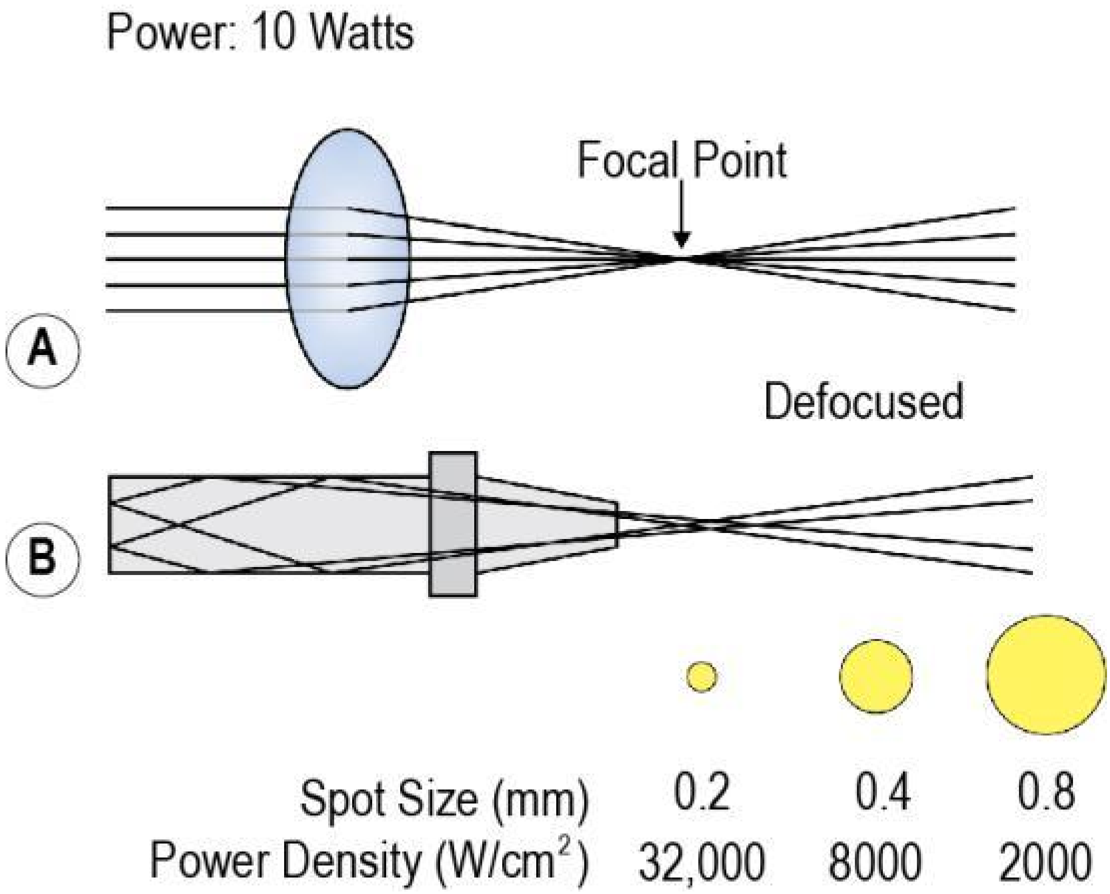
TABLE 10.1

Power Density (W/cm^2) is Determined by the Power Output of the Laser and Spot Size of the Beam (see Text)

Power	Diameter			
	0.2 mm	0.4 mm	0.8 mm	1.0 mm
5 W	16,667	4167	1000	637
10 W	33,333	8333	1992	1274
20 W	66,667	16,667	3984	2547

When using a CO_2 laser, efficient cutting of tissue resulting in minimal collateral thermal injury requires using a power density of at least 4500 W/cm^2.

This is an important concept, which underscores both the necessity to work within the focal point of the beam for maximum efficiency in the cutting of tissue and the versatility in influencing tissue response through changing power density by working in the focused or defocused portion of the beam (Fig. 10.4). Intuitively, it might seem that a change from a 0.4-mm spot diameter to a 0.8-mm spot diameter would have little effect on the overall influence of the beam on tissue. Practically, the surgeon who feels comfortable in using a 0.4-mm spot at 10 W for the incision of skin and switches to a beam delivery device with a 0.8-mm beam diameter would need to increase the power output of the laser to 40 W in order to maintain the same power density.



• **FIG. 10.4** The area over which photons are distributed influences their concentration and the rate at which energy is deposited into tissue. The beam may be delivered (**A**) through a lens that focuses the beam into a spot or (**B**) through a fiber or waveguide, where the beam exits through a tip or fiber end. Source: (From Peavy GM. Laser and laser-tissue interaction. *Vet Clin North Am Small Anim Pract* 2002;32:517–534. With permission.)

The most efficient excision of tissue can be achieved by working at the focal point of the beam. Using the defocused region of the beam by moving the handpiece away from the tissue surface rapidly decreases the power density, permitting superficial tissue ablation, tissue contraction, and/or hemostasis without having to adjust the power setting of the laser.

Time domains of energy delivery

Power density will influence the rate of energy deposition within a volume of tissue and, subsequently, the exposure time required to reach the energy threshold for tissue transformation. Thermal *diffusivity* of a tissue is the speed at which thermal energy will move through that tissue. In a photothermal ablation process, the longer one takes to deposit photon energy into tissue to induce ablation, the more collateral thermal injury will be created as the thermal energy generated at the ablation site has more time to diffuse into adjacent tissue.[8,13]

The time domains of energy delivery to tissue are influenced at two levels, (1) the experience of the surgeon and (2) the mode selection of the laser. In the initial learning phase, the laser surgeon tends to select parameters that result in the use of low power densities (large spot size and/or low power output) that will produce slow cutting at an easily controlled rate. As the surgeon increases in proficiency and confidence, there is comfort in proceeding at a more rapid rate, so the surgeon gravitates to higher power densities (small spot size and high power output). With increasing power densities, ablation proceeds at a more rapid rate, reducing the duration of exposure along the incision line and the time available for thermal diffusion to produce collateral thermal injury.

Further enhancing the surgeon's ability to reduce thermal diffusion, lasers may be equipped with pulsing modes that permit the use of higher powers in a succession of pulses. In this manner, ablation thresholds are reached more rapidly because of the high peak powers, but the interval between pulses permits the application to remain controllable. Additionally, pulse modes allow improved precision and control in the removal of small volumes of tissue.

Basic pulse modes can be found in the 1–500-ms range with pulse frequencies of 1–60 Hz. Further reduction in collateral thermal injury can be achieved by using a succession of high peak power pulses that reach the ablation threshold within a time period that is shorter than the time required for thermal energy to diffuse outside of the irradiated volume of tissue. This is referred to as the *thermal relaxation time* (t_r) of tissue and is both wavelength and thermal diffusivity dependent.[6,9-11,21,22] For the CO_2, laser the t_r values are in the range of 300–700 μs, and for the Er:YAG laser, the t_r approximates 2 μs.[6,21]

The superpulse mode of a CO_2 laser generates high peak power pulses (50–500 W) in a time period that approximates the t_r (500–700 μs) in rapid succession (100–1000 Hz) designed to achieve efficient tissue removal while minimizing collateral thermal injury. Using a CO_2 laser with extremely short pulses and high peak powers, collateral thermal damage in oral soft tissues ranged 15–170 μm. Using the same laser system in the continuous mode setting resulted in collateral damage of approximately 600 μm.[23] For the CO_2 laser it is typical to find reported a zone of 300–600 μm for the CW mode, 100–200 μm for pulses of 2–500 ms, and 50 μm or less for pulse durations of 2–600 μs.[21-25]

To minimize collateral thermal injury and maximize ablation efficiency, enough energy to reach the vaporization threshold of water (2500 J/cm^2) must be delivered into the volume of soft tissue irradiated by the CO_2 laser (μ_a = 794/cm) within the thermal relaxation time of the tissue (t_r = 695 μs). This requires the use of a power density of at least 4500 W/cm^2 (2500 J/cm^3 × 1 cm/794 × 1/695 × 10^{-6} s).[21] While tissue cutting and ablation will occur at lower power densities, they will be accompanied by larger zones of collateral thermal injury because of the longer duration of beam exposure required to achieve tissue vaporization. Table 10.1 outlines power density as a function of power output of the laser and beam diameter at the tissue surface.

Tissue handling

The manipulation of tissue by the surgeon during the application of the laser beam can have a profound influence on the resulting tissue effect. A moderate amount of tension applied across the surgical plane during the laser incision will cause the tissue to separate along the incision line as soon as the tissue is able to part. This moves tissue out of the beam path as it is incised.

Using the separation of the incision line as a visual cue to regulate the speed at which the surgeon moves the beam helps to ensure that the tissue surface is completely incised in one pass. If the skin along an incision line is not incised through its full thickness during the initial pass of the beam, repeat passes will be needed to separate the tissue. Each pass of a manually controlled 0.2–0.4 mm focal point along an incision line is not likely to be centered exactly on the original pass, producing otherwise unnecessary heat generation and increased collateral thermal damage.

When the objective is to ablate (vaporize) a volume of tissue rather than make an incision, a power and spot diameter are selected that will allow for the removal of a wide area of tissue. During the ablation process, as tissue is broken down into its elemental components, it is likely that carbon residue (char) will accumulate on the ablation surface. Char formation may be reduced by employing pulsed modes of beam delivery and/or by passing the beam across the tissue surface at a rapid rate.

The presence of char on the tissue surface will block the entry of the beam into tissue. This results in transfer of thermal energy from the heated char into the tissue, inhibiting ablation efficiency and promoting widespread thermal injury.[9] When the wavelength that is being used is poorly absorbed by tissue, such as a near-IR diode, the presence of char may enhance the ablation process of the laser by acting as a primary absorber, confining photons that would otherwise pass through tissue and transferring the thermal energy into the underlying tissue. However, the tissue effect still occurs as a result of thermal transfer, so wide areas of collateral thermal injury should be expected. When using a CO_2 laser to ablate tissue, it is important to use a moistened gauze sponge or cotton-tipped applicator to remove char from the ablation site. The surgical technique is to alternate ablating and gently wiping away char.

Beam delivery

Transmission of the beam from the laser to the surgery site requires a delivery system that will facilitate directing the beam to the desired location efficiently (minimal loss of intensity), retain good beam quality (even distribution of photons across the beam), and at a clinically relevant intensity (useful focal point diameter). To accomplish this, the laser will be equipped with one of three delivery mechanisms: optical fiber, articulated arm, or waveguide.

Optical fibers

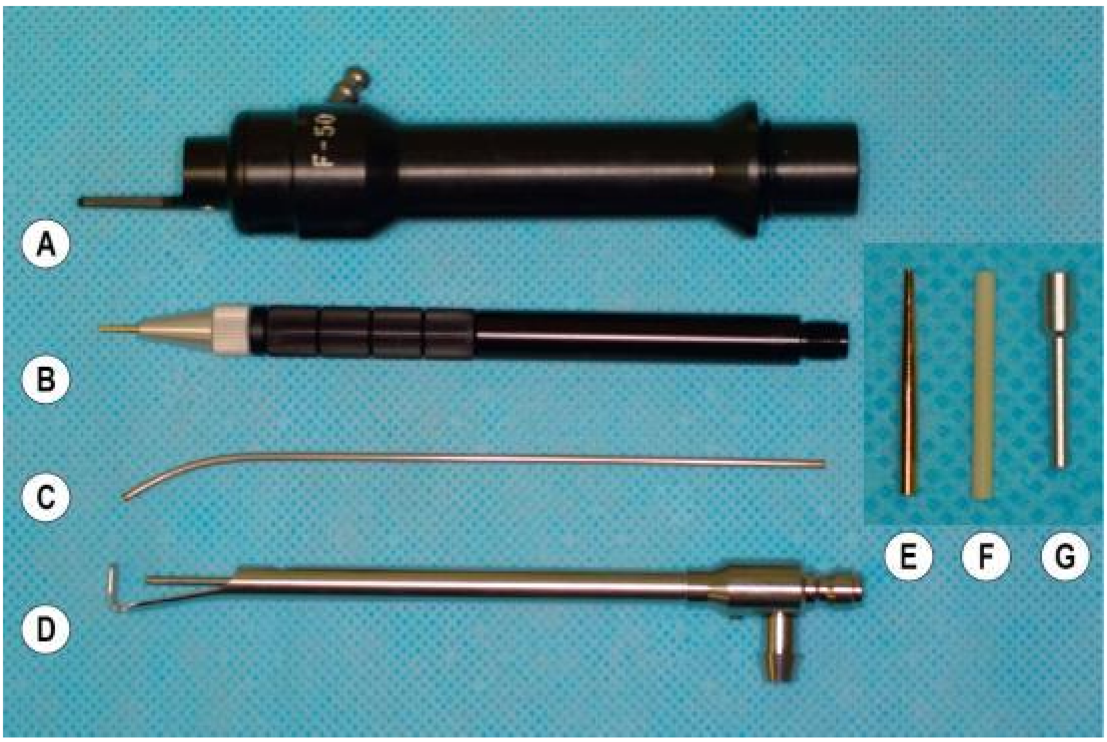
Solid-core, generally silica-based, optical fibers ranging in diameter from 200 to 1000 μm are used with most laser systems that produce wavelengths in the visible to near-IR region. An outer cladding that may be composed of a soft polymer or a hard fluoropolymer covers the silica fiber core. Photons are launched into the fiber at a coupler on the laser and travel through the fiber, exiting through a free cut (cleaved) end, a sculpted tip, or a fixed lens.[20]

The beam generally diverges as it moves from the tip, so the narrowest point of the free beam is at the fiber tip and corresponds to the diameter of the fiber core. The beam can be delivered with the fiber either in contact or noncontact with the tissue. A sculpted tip is generally intended for contact, a fixed lens for noncontact, and a cleaved end for either contact or noncontact applications. When used in contact mode, the fiber is manually drawn through the tissue during irradiation, adding mechanical force to the photothermal mechanism for the incision of tissue.

Wavelengths that are well absorbed by water (longer than 2.0 μm) are not delivered through silica fibers, as the hydroxyl groups of the silica absorb the beam, resulting in poor transmission efficiency with overheating and breaking of the fibers. While fiber delivery facilitates beam application, especially to difficult-to-access places, their use is limited to wavelengths that are less efficient for soft tissue removal than the wavelengths that are absorbed by water. Fiber applications of visible to near-IR wavelengths are associated with more collateral thermal injury than is experienced with the free beam applications of the Er:YAG or CO_2 lasers, except where the laser is able to produce high-intensity, short-pulse beams or the target tissue has a high concentration of an absorbing chromophore such as melanin or hemoglobin.[9,11,12]

Articulated arm

An articulated arm consists of two hollow tubes connected by an elbow joint that rotates around two parallel mirrors, permitting passage of a collimated beam from the laser through the center of the tubes regardless of their position in respect to each other. The terminal end of the arm assembly will have an articulated handpiece attachment containing a focusing lens of fixed focal length and a specified focal point diameter (Fig. 10.5).



• **FIG. 10.5** (**A**) The handpiece of an articulated delivery system contains a lens that focuses the beam into a spot of fixed diameter. (**B**) The waveguide delivery handpiece concentrates the beam as the beam exits through a tip. Waveguide delivery tips may be configured to meet specific beam delivery requirements. Extended length tips like (**C**) the 0.8-mm-diameter 120-mm-long curved tip and (**D**) the laser-assisted uvuloplasty device with 0.8-mm-diameter delivery and backstop permit focused beam delivery at an extended distance from the handpiece, facilitating surgical procedures in difficult-to-access places. Short insertion tips of (**E**) 0.4-mm diameter, (**F**) 0.8-mm diameter, and (**G**) 1.4-mm diameter are most commonly used for general soft tissue surgical applications.

Waveguide

A waveguide is a hollow-core tube with a highly reflective inner surface that attaches to the laser at one end and has either free beam discharge or a handpiece with application tips at the other.[20] Photons traveling through a waveguide exit following their last reflection event from the wall of the waveguide and are traveling a path of divergence from the tip. They are fairly evenly distributed across the beam as their paths of travel cross while exiting the waveguide, but they are found more on the periphery of the beam at an increasing distance from the point of exit (see Fig. 10.4).

For standardization, the specified diameter of a waveguide tip is the width of the beam measured 1 mm distant from the end of the tip. While there is some ability to move from a focal point (1–3 mm from the end of the tip) to a wider, defocused beam diameter by manually retracting the handpiece away from the tissue surface, retention of an even distribution of photons across the beam at increasingly wider beam diameters requires the changing of tip diameters, usually at intervals where the beam diameter reaches a twofold increase from the tip diameter. An advantage of a waveguide delivery system is the variety of tip configurations and angles that can be incorporated into the system, greatly facilitating delivery of the beam to difficult-to-access places that may be problematic for a lens directed beam with a fixed focal length (Fig. 10.5).

Surgical considerations

Surgical approach

When cutting or ablating tissue with a surgical laser, it is important to consider how the beam will be directed and manipulated to avoid inadvertent tissue exposure. Once the beam penetrates through the targeted tissue, it will continue on to the next tissue layer or object in its path. Inadvertent hard tissue exposure at ablative power densities of most clinical lasers will produce charring defects that will remain as permanent etches, and heating of teeth that raises the temperature of dental pulp by 5.5°C can result in permanent damage to vital pulp.[26]

The placement of a beam diffusing, deflecting, or absorbing backstop behind the targeted tissue should be used whenever possible to provide protection to surrounding structures. While backstops are made of a variety of materials, a material that will absorb the selected laser wavelength would be the safest. For the CO_2 laser, water-soaked materials such as gauze sponges, cotton-tipped applicators, or tongue depressors make excellent and inexpensive backstops.

Wound healing

The principles of wound healing, management, and closure are no different for a laser-induced lesion than for any other wound. Although initial collagen formation and healing of soft tissue may be slightly delayed after CO_2 laser surgery, by 3 weeks after surgery, a difference in wound organization and strength is no longer apparent.[27-31] In the long-term, CO_2 and Er:YAG laser wound contraction and scar formation are reduced, leading to improved cosmetic results.

The amount of collateral thermal damage created by the laser application is of considerable importance in determining the rate and quality of wound healing. Immediate and final wound contraction tends to increase with greater initial thermal damage and greater depth of ablation. A thick layer of thermal damage at the margins of a wound, especially if char is produced and not removed prior to wound closure, will tend to delay healing and weaken wound cohesive strength.[27,28,30,32]

At the completion of the procedure, the surgery site and especially any wound margins that will be apposed for primary closure should be gently cleaned to remove any debris, particularly any particles of char. When first learning to use a CO_2 laser, one should anticipate that collateral thermal injury will occur and that primary intention healing will be delayed, but as experience is gained, the healing times will return to those experienced when a scalpel blade is used.

Anesthetic considerations

Laser surgical procedures are not painless and must be performed in conjunction with an appropriate level of anesthesia and patient immobility. Patient airways should be protected from plume inhalation by use of smoke evacuation and, whenever possible, intubation. When using inhalation anesthesia while working in the oral cavity, endotracheal tubes should be protected from errant beam exposure and a smoke evacuation system used for the prevention of the accumulation of ignitable oxygen in the surgical field. Where manual or sedated restraint with use of a local anesthetic may be a consideration, the potential complications associated with patient movement during application of the laser are of particular concern, and appropriate safety precautions need to be taken.

Oral and maxillofacial surgical applications

The following surgical procedures may be performed with standard surgical instrumentation, electro/radiosurgery devices, or lasers, and several may be discussed in other sections of this book. These procedures are selected for discussion here because they may illustrate specific techniques of tissue handling associated with laser surgery or may incorporate modifications of the procedure that are specific to use of a CO_2 laser.

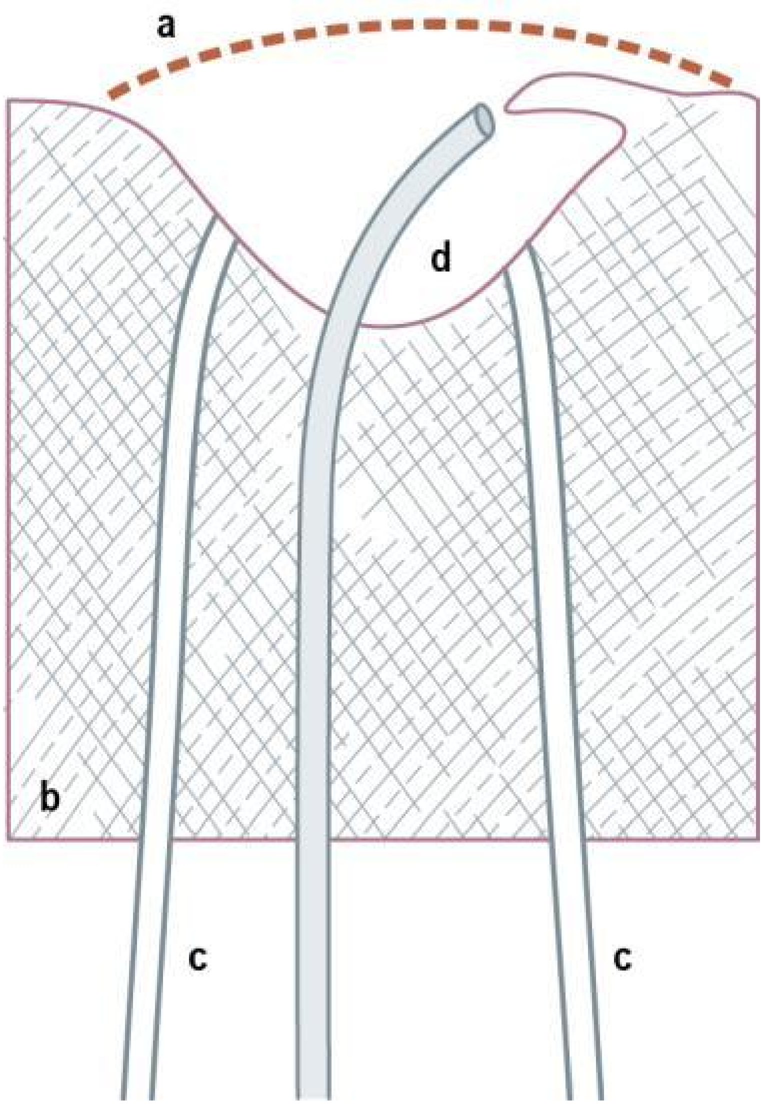
Incision, excision, and ablation can be accomplished with the laser. Changes in power density are controlled by adjustments in beam diameter created by movement of the handpiece to adjust the focal distance during the procedure. A tight focal spot (high power density) is used for incision and excision, while a wider, defocused beam (lower power density) is used for ablation and tissue contraction. All procedures are performed under appropriate general anesthesia and patient preparation and in adherence to routine safety precautions.[14-17]

Correction of overlong soft palate

Elongated soft palate is one component of the brachycephalic airway syndrome. The use of a CO_2 laser in the surgical correction of an elongated soft palate was described by Clark and Sinibaldi in 1994.[33] The approach is similar to conventional surgical correction (see Chapter 60). The incised margin of the soft palate is not sutured following the laser excision of tissue but is left to heal by second intention.[33,34] With use of a laser, additional attention is given to precautions for protection of the airway, using a smoke evacuator for removal of the vaporization plume, and placement of saline-moistened gauze sponges over the tracheal tube and behind the soft palate to protect the tube and pharyngeal tissues from errant beam exposure during excision.

A CO_2 laser with either an articulated arm or waveguide delivery system may be used for this procedure. However, the approach is facilitated and the visual field less encumbered when an extended-length waveguide tip or a laser-assisted uvuloplasty (LAUP) handpiece is used (see Fig. 10.5). Following intravenous induction of general anesthesia, the oropharynx is inspected and the soft palate is marked for excision prior to placement of an endotracheal tube. With the patient in dorsal recumbency and the head supported in a normal position in relation to the neck so that the soft palate and larynx approximate normal alignment with the epiglottis lying under the excess soft palate tissue, the laser beam is directed against the soft palate in a chain of single pulses along the edge of the epiglottis to outline the margins of the segment of soft palate that is to be excised.

Once the excision margin is outlined, a tracheal tube is placed, inhalation anesthesia is administered, and the pharynx is lined by saline-soaked gauze sponges. Tissue forceps, a hook, or stay sutures are used on the margins of the soft palate to pull it forward and apply tension across the surgery site during the excision. The laser beam is passed across the soft palate to make a full-thickness incision that is slightly medial to the premarked outline (Fig. 10.6), which allows for some tissue contraction during the procedure and in healing. Considerations for anesthesia recovery and postoperative care are similar to conventional surgical repair.



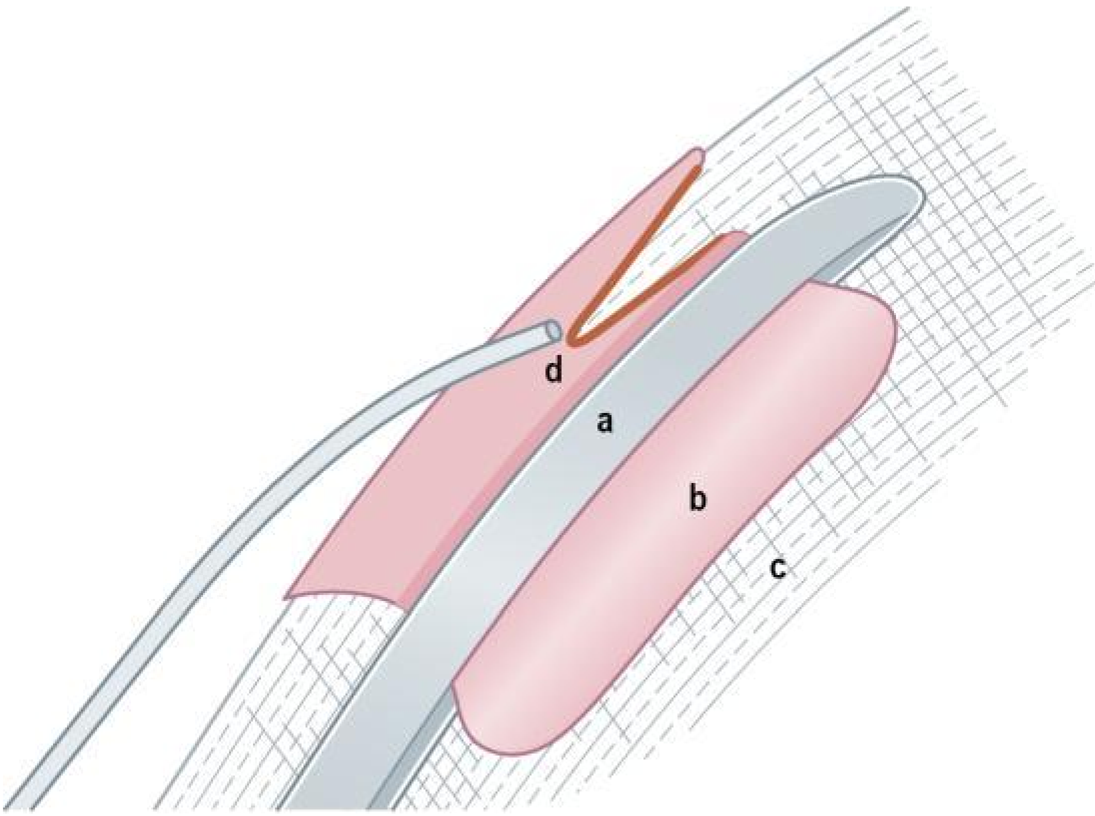
• **FIG. 10.6** Prior to surgical excision, the soft palate is placed over the epiglottis and individual laser pulses are used to outline the margin of the epiglottis on the soft palate *(a)*. The patient is intubated and the end of the soft palate is retracted rostrally over a moistened gauze sponge *(b)* placed to cover the tracheal tube and caudal pharynx. Stay sutures *(c)* and/or Allis forceps are placed on the margin of the soft palate to facilitate the placing of traction across the incision line. The laser beam is used to make a full-thickness incision *(d)* of the soft palate at the marked line.

As some collateral thermal influence on the tissue is desired in order to achieve hemostasis during the excision, a continuous wave mode of delivery is generally preferred over the superpulse mode. The extended waveguide tip and LAUP handpieces usually emit a 0.8-mm diameter beam, which means that the power output of the laser will need to be increased to achieve a cutting efficiency comparable with a 0.2–0.4-mm focal spot.

Tonsillectomy

Tonsillectomy is occasionally indicated in the dog and cat (see Chapter 63), and hemorrhage is a known complication. Use of a CO_2 laser equipped with an extended waveguide tip provides for a quick, easy, and generally bloodless approach for the removal of tonsils.

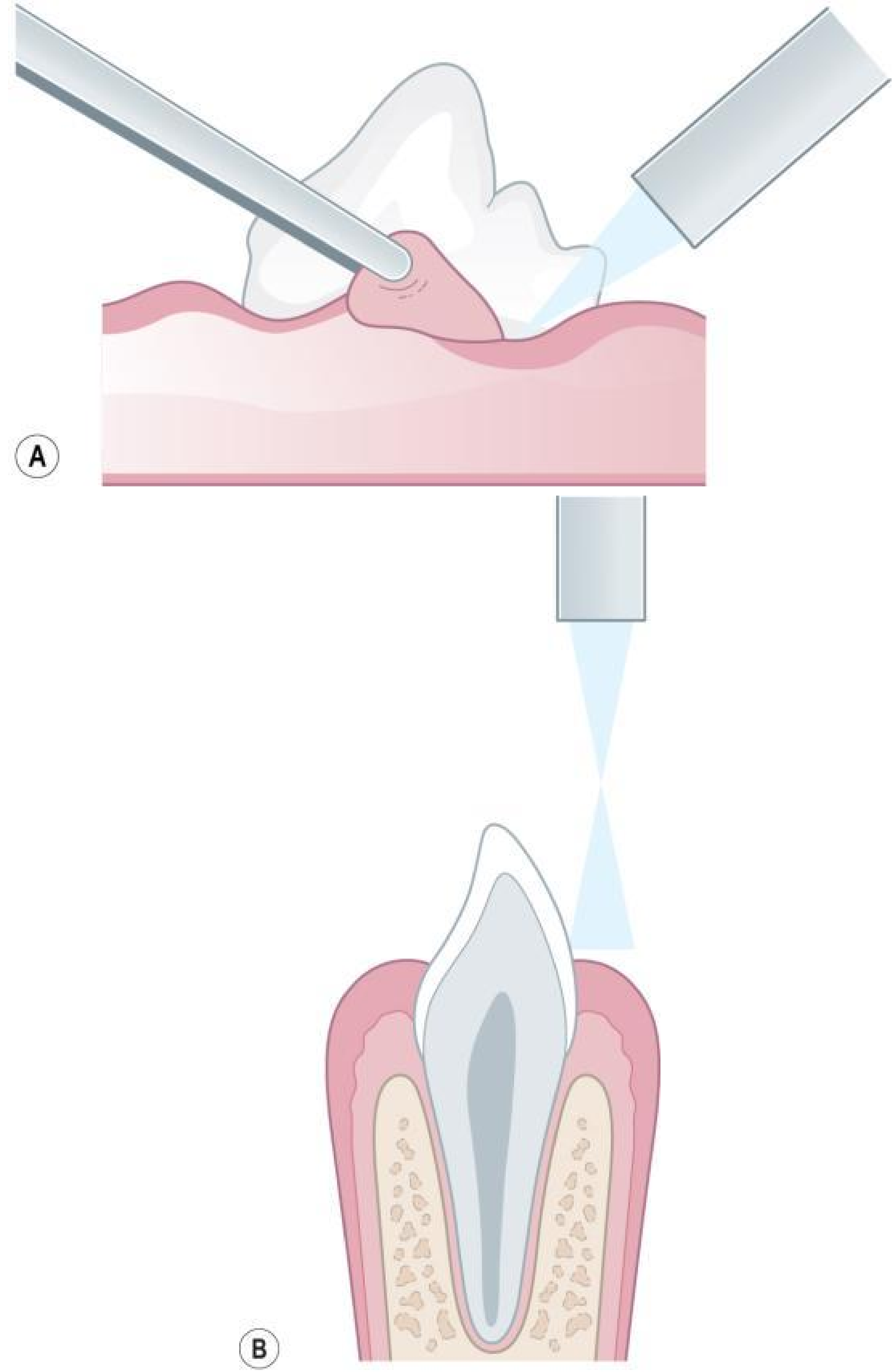
A saline-soaked gauze sponge is placed against the wall of the pharynx, just below the tonsillar fossa, to act as a backstop. A curved forceps is passed behind and clamped across the base of the tonsil and used to extract the tonsil from the fossa, drawing it out and over the gauze sponge. While using the forceps to apply gentle tension to the tonsil, the laser beam is directed across the base of the tonsil, between the forceps and wall of the pharynx, in a caudal to rostral direction. Separation of the tissue is used to gauge the rate of passage of the laser beam so that the tonsil is excised in a single pass of the beam (Fig. 10.7).

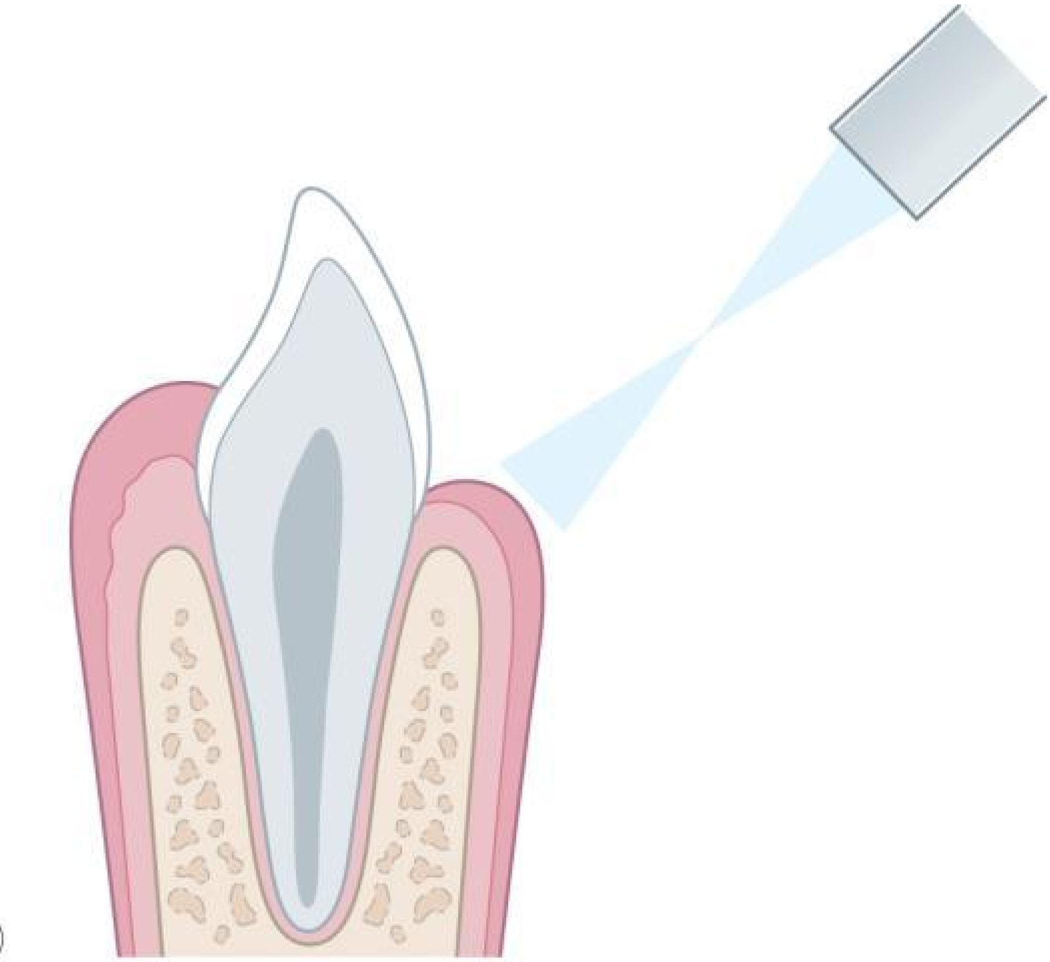


• **FIG. 10.7** Tonsillectomy: curved forceps *(a)* are placed across the base of the tonsil *(b)* and used to exteriorize the tonsil from the fossa over a moistened gauze sponge *(c)*. The laser beam is used to transect the base of the tonsil *(d)*. Either an articulated arm or waveguide CO_2 laser delivery system may be used for this procedure; however, a narrow extended-length delivery tip *(d)* will facilitate visualization of the surgical field.

Gingivectomy/gingivoplasty

The laser provides a tool for achieving hemostasis while removing gingival tissue, with the added advantage of being able to sculpt gingival tissue during removal (see Chapter 20). Care should be taken to protect the enamel surfaces of teeth from direct exposure to the beam. The removal of hyperplastic gingival tissue by a CO_2 laser may be achieved by a combination of excision and ablation techniques. When excising tissue, a margin of the hyperplastic tissue is grasped with thumb forceps while the focused beam is directed in a line parallel to the long axis of the tooth, not perpendicular to the tooth surface (Fig. 10.8A). Vaporization of gingival tissue is achieved by using a defocused beam in a similar manner (Fig. 10.8B). Power density is varied by manually adjusting the focal distance (beam diameter) to control the rate and depth of tissue removal. The beam is moved across the gingival surface in an overlapping, repeating rostral-caudal-rostral pattern, ablating tissue with each pass and removing char as it accumulates on the ablation surface. As the gingival tissue is reduced to the level of the base of the tooth, the handpiece is gradually rotated away from the tooth with each pass in order to sculpt the gingival surface back to a normal conformation (Figs 10.8C and 10.9). Postoperative care is the same as for a routine gingivectomy (see Chapter 20).


A
B



C

• FIG. 10.8 CO_2 laser gingivectomy/gingivoplasty is accomplished by a combination of excision and ablation. (**A**) To excise gingival tissue, the margin is grasped with thumb forceps to apply traction while a focused beam is directed in a plane parallel to the long axis of the tooth. (**B**) Vaporization may be used instead of excision to remove hyperplastic gingival tissue by directing a defocused beam parallel to the long axis of the tooth. (**C**) When using either excision or ablation, once the bulk of the hyperplastic tissue has been removed, the remaining gingival tissue can be recontoured using a defocused beam.

• **FIG. 10.9** (**A**) Gingival hyperplasia with 3–5-mm pseudopockets affecting the mandibular incisors. (**B**) CO_2 laser gingivectomy by defocused beam ablation. (**C**) Healing 11 days and (**D**) 19 days following treatment. Source: (Photographs courtesy Dr. R. Arza.)

Excision of oral mass lesions

Lasers may be used in the treatment of oral mass lesions to remove abnormal tissue by surgical excision, ablation, or photothermal coagulation. The laser is used in accordance with the principles of surgical oncology, with reference to the surgical objective (e.g., cure versus palliation) and surgical margins (see Chapter 48).

In selecting a laser system, consideration should be given to the tissue effect that is desired for the treatment. Where the precise removal of tissue with minimal collateral thermal injury is desired, especially if soft tissue planes are to be preserved or thin layers of tissue vaporized from other structures are to be preserved without injury, the CO_2 or Er:YAG in continuous wave or pulsing modes would be preferred.[35-42] Where coagulation of tissue beyond the margins of surgical excision is desired, or blood vessels 0.5–3.0 mm in diameter are likely to be encountered, a visible or near-IR laser might be selected.[36,39,43-45] Where a large area of tissue coagulation and postsurgical necrosis is intended, similar to cryosurgery or hyperthermia procedures, use of a near-IR wavelength would be the selection of choice.[46-48]

Periodontics

While the CO_2 laser has been the most commonly used laser device for gingival and periodontal applications for many years, a variety of diode lasers in the near-IR range (700–1000 nm) have become available for oral applications. They enjoy widespread use in human dentistry, and devices optimized for veterinary use are also available. Because of strong absorption by the oral soft tissues of these wavelengths of light, such devices are very efficient and effective, ablating quickly at relatively low power levels (ideally with high peak powers and very short pulses). Typically, these produce less heat for greater comfort, faster healing, and minimal trauma to the oral tissues.[49] The diode lasers are typically small (size of a brick) and relatively inexpensive, with purpose-designed, often disposable tips to achieve precise control of hemostasis and tissue cutting for different procedures and tissue biotypes. Additionally, some of these devices include “perio tips” specifically designed for laser bacterial reduction and periodontal pocket treatments.[50,51]

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CHAPTER 11

Microsurgical techniques in maxillofacial surgery

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Introduction

Microvascular free-flaps have fundamentally enhanced the ability to reconstruct complex soft tissue and bony defects in humans since they were first described in 1972.[1] While free-flaps began with conceptually simple fasciocutaneous flaps based on single axial arteries, they have evolved to include myocutaneous, osteocutaneous, and chimeric flaps that incorporate each of these elements.[1-3] In human maxillofacial surgery, these techniques are now routinely used for reconstruction following oncologic ablation and following traumatic injuries that result in significant soft tissue and/or bone loss.

Not long after microvascular reconstruction was introduced to human medicine, these techniques were adopted to veterinary patients, with the first series describing veterinary free-flaps published in 1986.[4] Although this exciting technique has yet to gain widespread adoption within veterinary medicine, a variety of reproducible and dependable fasciocutaneous, myocutaneous, and osteocutaneous flaps have been described, and some tertiary practices have incorporated these techniques into their reconstructive algorithms with excellent flap viability and functional outcomes.[5] This chapter will review veterinary free-flaps and their outcomes and discuss the perioperative management of patients who have undergone these procedures. Veterinary free-flaps and their vascular supply are summarized in Table 11.1.

TABLE 11.1

Veterinary Free-Flaps and Vascular Supply

	Blood Supply
Soft Tissue Free-Flaps	
Superficial cervical	Superficial artery
Trapezius myocutaneous	Prescapular branch of the superficial cervical artery
Myoperitoneal	Cranial abdominal artery
Medial saphenous	Medial saphenous artery
Rectus abdominis	Cranial/caudal epigastric artery
Latissimus dorsi	Thoracodorsal artery
Deep circumflex iliac cutaneous	Deep circumflex iliac artery
Bony Free-Flaps	
Trapezius osteomusculocutaneous	Prescapular branch of the superficial cervical artery
Tibial bone	Saphenous artery
Distal ulnar	Interosseous artery

Soft tissue free-flaps

The first microvascular free-flaps described were soft tissue flaps designed to cover defects too extensive to be covered by locoregional or pedicle flaps. Fasciocutaneous flaps are primarily based on a consistent axial blood supply that can readily be identified and traced proximally to a source arterial pedicle that is of adequate length and caliber to allow for microvascular anastomosis. Soft tissue free-flaps have evolved to include perforator flaps based on septal and myocutaneous dermal perforators and myocutaneous flaps that can be used to host a skin graft or harvested with an overlying skin island.

Superficial cervical free-flap

The superficial cervical flap was the first veterinary free-flap described.[4] It is a fasciocutaneous free-flap based on the superficial cervical artery and vein. This flap was first studied by Miller et al., who performed anatomic studies identifying it as an ideal candidate for a cutaneous flap. The vascular pedicle emerges consistently from the angle formed by the trapezius and omotransversarius muscles with a predictable distal course. Miller and his group performed eight orthotopic microvascular transfers of this flap, of which six survived without complications.[4]

Trapezius myocutaneous free-flap

The anatomic basis for the trapezius myocutaneous free flap was first described in animals by Philibert et al. in 1992.[6] This flap is based on the prescapular branch of the superficial cervical artery. It is harvested with a portion of the trapezius flap and has the potential to cover a relatively wide surface without significant bulk. The donor site results in limited morbidity and there are synergistic muscles that compensate for the loss of trapezius function after harvest. The vascular pedicle is described as having a mean length of 43 mm with a mean vessel diameter of 1.0 mm, making it an excellent candidate for free-flap transfer.

In 2013, Kurach et al. retrospectively described their complications and clinical outcomes with this flap in 20 dogs and a wallaby. The flaps were performed for a variety of indications, including traumatic soft tissue loss, neoplasm excision, osteomyelitis, and palatal reconstruction. All flaps survived with antithrombotic therapy used perioperatively. There were very few postoperative complications, and the severity of the complications that did occur was low.[7]

Myoperitoneal free-flap

In 1996, Degner et al. performed anatomic studies to evaluate the potential for myoperitoneal microvascular free-flaps in dogs. They evaluated the angiosomes of the right deep circumflex iliac artery and the left cranial abdominal artery in canine cadavers. The left cranial abdominal artery showed consistent filling of the vascular bed supplying the cranial half of the transversus abdominis muscle, making it an ideal soft tissue free-flap donor site. The deep circumflex iliac artery, on the other hand, had poor arborization and inadequate vascular perfusion and was therefore deemed not to be a good candidate to support this flap. The myoperitoneal free-flap's ability to support portions of the transversus abdominis muscle makes it ideal for reconstructing a variety of large-volume soft tissue defects. This flap was successfully used by Degner et al. to reconstruct a hard palate defect in an 8-year-old dog.[8]

Medial saphenous free-flap

The medial saphenous free-flap can be harvested as both a fasciocutaneous or myocutaneous free-flap. This flap is based on the cutaneous angiosome of the caudal branch of the saphenous artery as it extends from the most proximal aspect of the medial femoral region to the distal aspect of the medial tibia. The pedicle length and vessel diameter are typically adequate for microvascular anastomosis. When harvested as a fasciocutaneous flap, it conforms well to the wound bed and is well suited for extremity reconstruction. The myocutaneous flap includes the distal half of the caudal head of the sartorius muscle and is ideally suited for defects with substantial soft tissue loss. Degner et al. described their experience with this flap for the reconstruction of extremity and facial soft tissue defects in eight dogs. All of the flaps survived, although two required intervention for venous congestion (revision anastomosis and leech therapy). The use of this flap resulted in a short postoperative hospital stay and low patient morbidity, and the authors advocate for primary consideration of this flap when considering any large soft tissue reconstruction in a dog.[9]

Rectus abdominis myocutaneous free-flap

The rectus abdominis myocutaneous free-flap is used extensively in human medicine for complex soft tissue reconstruction due to its potential to reconstruct large soft tissue defects and its reliable vascular pedicle. It was assessed by Calfee et al. in 2002 with regard to its donor-site morbidity and flap survival in dogs. The authors first performed anatomic studies in six cadaver models, which confirmed the candidacy of this muscle for free-flap transfer. The rectus abdominis muscle in the dog receives its blood supply cranially from the cranial epigastric artery and caudally from the caudal epigastric artery, either of which can be used as the free-flap pedicle. Calfee et al. performed seven heterotopic transplants transferring the rectus muscle via anastomosis of the caudal epigastric artery and vein to the saphenous artery and vein. All the flaps did well in the perioperative period, postmortem examination at 30 days confirmed vascular patency, and there was minimal donor-site morbidity. The authors concluded that the rectus abdominus myocutaneous free-flap in dogs is a good option for repair of complex soft tissue defects.[10]

Miller et al. described their clinical experience with rectus abdominis free-flap for closure of complex wounds in nine dogs. A variety of wounds were addressed, including five distal extremity wounds, three oral palatal defects, and a large postsurgical defect after excision of a hygroma. The caudal epigastric vessel was used as the supplying vessel for these flaps. All flaps survived and there were no major complications with the donor site, with consistently good cosmetic and functional outcomes. This study demonstrates successful incorporation of the rectus abdominis free-flap for complex canine soft tissue reconstruction.[11]

Latissimus dorsi free-flap

The latissimus dorsi flap has been primarily described in cats. This was first described in 1996 by Nicoll et al., who performed anatomic and experimental evaluations of a feline latissimus dorsi muscle flap based on the thoracodorsal artery to assess its potential for use as a free-flap in cats. They performed four heterotopic transplantations and three orthotopic transplantations with flap viabilities of 66% and 100%, respectively. The authors conclude that the latissimus flap is a good candidate for soft tissue reconstruction with low donor site morbidity and adequate long-term anastomotic patency. However, the low rate of viability in the heterotopic transplantations is concerning. In this study, the authors attributed their flap failures to arterial and venous thrombosis possibility, contributed to by small vessel size, kinking, or compression.[12]

Deep circumflex iliac cutaneous free-flap

Jackson et al. have described their research to develop and assess a feline microvascular cutaneous free-flap based on the lateral branch of the deep circumflex iliac artery and vein. They first performed anatomic studies on six feline cadavers to confirm the candidacy of this flap by mapping the angiosome with barium injection and radiographs. Flaps were then elevated in two cats and observed without microvascular harvest, which demonstrated long-term viability of the harvested tissue. The authors then performed 10 free-flaps, which were transferred to the dorsal interscapular region. Six (60%) of these flaps survived for 2 weeks. This study demonstrated the feasibility of this flap for large complex soft tissue reconstruction in cats; however, a higher rate of ischemic complication than that observed in canine free-flaps was again seen. Given the consistently higher rates of failure seen with feline free-flaps, the authors postulated that the length of ischemia time for flap success may be shorter in cats than in other species.[13]

Bone flaps

Bony free-flaps have become an increasingly integral part of the human reconstructive arsenal since they were introduced in 1974.[14] This technique has been incorporated into veterinary medicine, with reports of excellent outcomes.[5] Indications for bone reconstruction in dogs and cats are common, including severely comminuted fractures with bone loss, defect nonunion fractures, postoncologic surgery defects (i.e., mandibulectomy), and chronic osteomyelitis. These types of bone defects are commonly addressed with soft tissue closure or with nonvascularized cancellous and allogenic grafts. However, these techniques often result in functional limitations, and rates of nonvascularized graft failure are stubbornly high. An appealing alternative is utilizing a regenerative approach using recombinant human bone morphogenetic protein (rhBMP)-2 (see Chapter 53).

Trapezius osteomusculocutaneous free-flap

The trapezius myocutaneous free-flap can be harvested to include the central spine and body of the scapula to create an osteomusculocutaneous free flap. This was first described in 1993 by Philibert et al., who performed four orthotopic transplants in dogs and studied bone viability using a variety of techniques. Like the myocutaneous free-flap, the osteomusculocutaneous free-flap is based on the prescapular branch of the superficial cervical artery. Anatomic studies demonstrated that bone viability is maintained primarily along the scapular spine, and the authors recommend that harvest be limited to this portion of the scapula. They also showed that the dogs had minimal gait morbidity after harvest, making this flap an ideal candidate for free-flap reconstruction of bony defects.[15]

Tibial bone free-flap

The tibial bone free-flap was introduced in 2000 by Bebchuk et al. as an excellent option for free-flap reconstruction of canine bony defects. Anatomic studies were performed that demonstrated consistent vascular supply based on the saphenous artery of the medial aspect of the tibia in cadaveric dogs. The tibial bone graft was harvested from the medial aspect of the tibia, with the proximal osteotomy made at the level of the distal protrusion of the tibial tuberosity and the caudal osteotomy made along the insertion of the popliteus muscles. This resulted in an approximately 60-mm-long bone graft with a vascularized skin paddle and vascular pedicle up to 100 mm in length. A tibial free-flap was then performed in seven dogs and was compared to an avascular graft in seven controls. Bone scans and radiographs were performed to evaluate outcomes and a postmortem evaluation was carried out at 60 days. The vascularized bone grafts appeared viable on radiographs and bone scans, and this was confirmed with the postmortem histologic evaluation, while the avascular grafts were grossly and histologically necrotic.[16] The vascularized tibial cortical bone graft appears to provide a dependable source of vascularized bone with acceptable donor site morbidity.

Distal ulnar free-flap

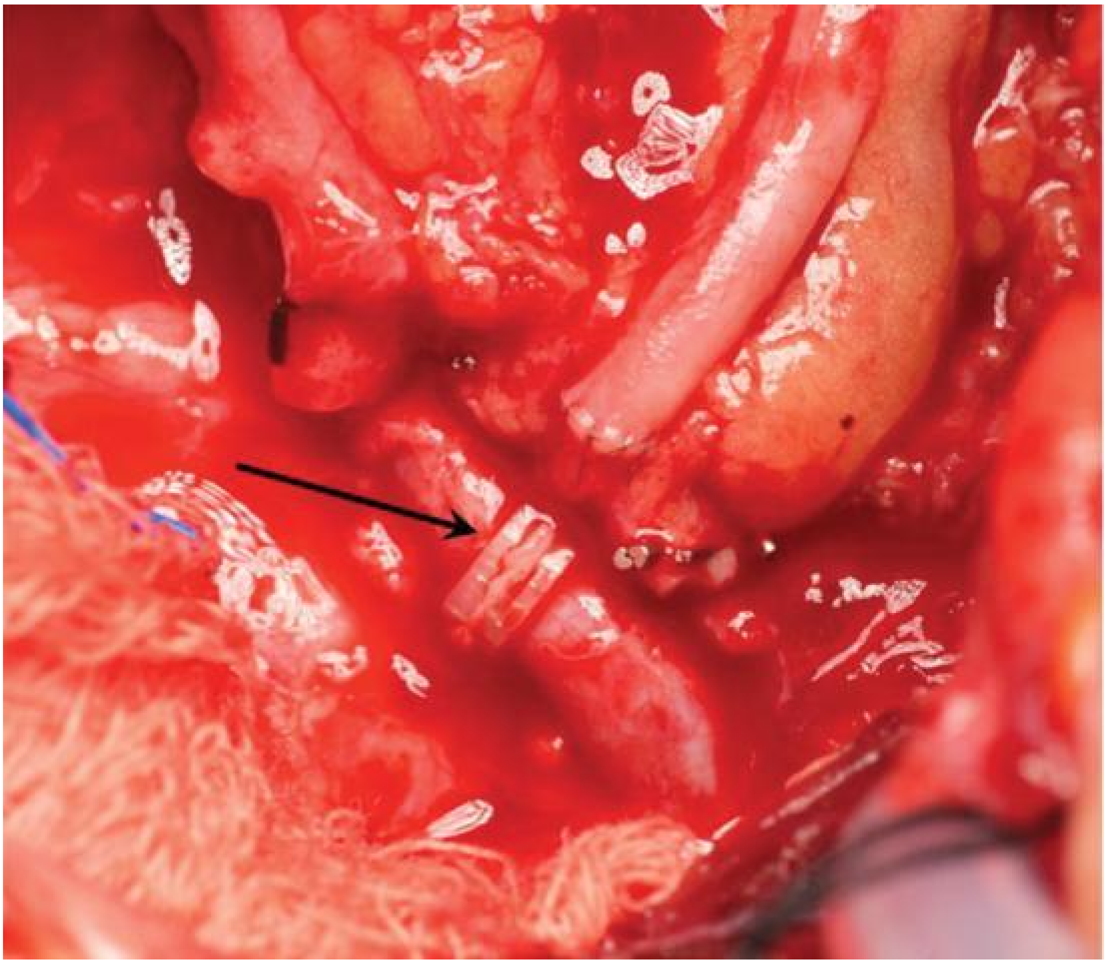
The distal ulna has also been demonstrated as a viable donor site for a canine osteocutaneous free-flap. The ulna is particularly well suited to harvest as it is the lesser of a two-bone system, is relatively dispensable, and, unlike the canine fibula, provides sufficient bone mass to accomplish long bone reconstruction. An anatomic study by Szentimrey et al. on 12 cadaveric dogs demonstrated that the caudal interosseous artery consistently serves as a source artery to the distal ulnar periosteal vasculature and is consistently of adequate size and length for microvascular anastomosis. Despite the entire distal ulnar bone being harvested, the morbidity of ulnar sacrifice was deemed acceptable.[17]

Technical considerations

Intraoperative technique

While harvest techniques are specific to the type of flap and patient species, the microvascular component of the procedures is relatively consistent. Arterial anastomosis is generally performed in an end-to-end fashion, and while a 1:1 vessel diameter ratio is ideal, anastomosis of vessels with diameter ratios of 2:1 and beyond can be reliably performed with technical refinements. The anastomosis is most commonly performed under microscopic visualization with a 9-0 or 10-0 nylon suture with the surgeons standing opposite one another (Fig. 11.1). A double-approximating microvascular clamp can be used to hold the vessels in opposition during suturing. Sutures are typically placed in a simple interrupted fashion, although an opposing 180-degree running suture technique can also be performed. Venous anastomosis is also ideally performed using an end-to-end technique; however, an end-to-side technique can easily be incorporated if there is a deficiency of candidate recipient venous branches. Suture anastomosis of veins in human microvascular surgery has largely been supplanted by use of the microvascular anastomotic coupling system (Synovis Micro Companies Alliance, Birmingham, AL). This system allows rapid and reliable apposition of veins using coupling rings attached to each vein that secure to one another with barbed pins (Fig. 11.2). A variety of intraoperative measures has been described in an effort to reduce the incidence of thrombosis within the vessels. These include the avoidance of vasopressors and the use of osmotic agents and anticoagulants; however, none has been shown to have a measurable protective effect and their use has been largely been abandoned.[18,19]

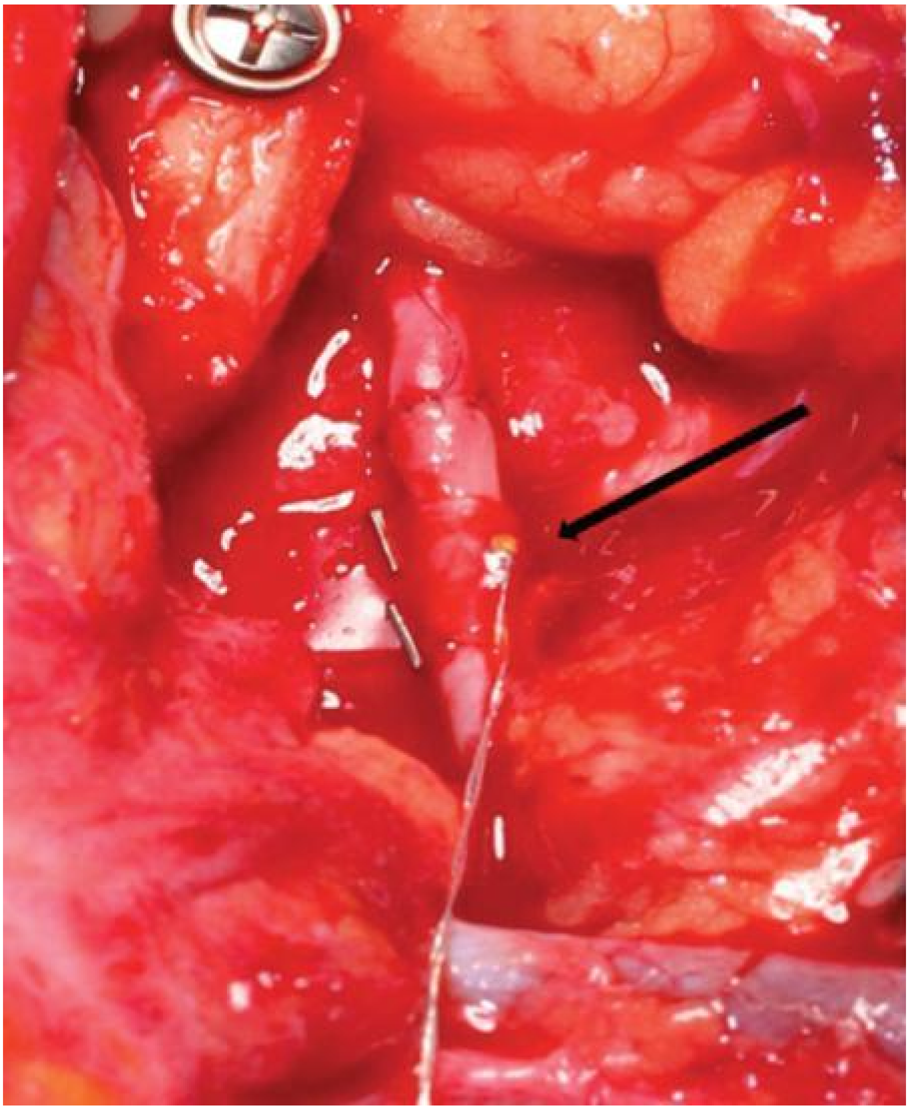
• **FIG. 11.1** Surgeon positioning for microvascular anastomosis.



• **FIG. 11.2** *Black arrow* points to synovis microvascular venous coupler.

Postoperative monitoring

Following free-flap surgery, the standard of postoperative care in human medicine is evolving, with a trend toward deintensification. Most flaps are assessed on an hourly basis for 24 hours after surgery as this time period represents the highest risk for vessel thrombosis. Flap viability is typically assessed both with physical examination via capillary refill or pin-prick and with Doppler confirmation of intact blood flow via pencil Doppler held over the defined course of the vascular pedicle or with an implantable Cook-Swartz Doppler (Cook Medical, Bloomington, IN) wrapped around the vessel of interest (Fig. 11.3). Historically, all free-flap patients were cared for in an intensive care unit. However, there is evidence to suggest that these patients can be adequately cared for outside of the intensive care unit with appropriate nurse training.[20] A number of postoperative care techniques have been described to minimize the risk of postoperative vessel thrombosis, including prolonged anticoagulation, room temperature regulation, and positioning precautions; however, these have not demonstrated improvements in outcomes when subjected to rigorous study.[18]



• **FIG. 11.3** *Black arrow* points to Cook-Swartz implantable Doppler monitoring of an arterial anastomosis.

Conclusions

Microvascular free-flap surgery has dramatically expanded the reconstructive surgeon's ability to restore function and cosmesis following oncologic resection and traumatic injury in human medicine. Although once deemed technically demanding, with standardizations in technique and high-volume training, these free-flaps have evolved into the standard of care and have very high rates of long-term viability (>98%).[19] The field of veterinary microvascular free-flap surgery is close behind: a number of reliable fasciocutaneous, myocutaneous, and osteocutaneous free-flap techniques have been meticulously described, and although these techniques have not yet disseminated widely into clinical practice, several published series of successful outcomes demonstrate that potential. With continued research, technique refinement, and appropriate training, more widespread adoption of microvascular technique may likely benefit the veterinary patients.

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CHAPTER 12

Use of the dog and cat in experimental maxillofacial surgery

M. Anthony Pogrel

The use of dogs and cats as animal models for human research has a long history, which is often arbitrary and not always based on science. In fact, the use of animal models in general has a somewhat checkered history, both scientifically and emotionally. At the present time, attempts are being made to move away from live animal models as much as possible and to move to the use of simulation, biomimetics, tissue cultures, etc. Where animal models need to be used, an attempt has been made to move to smaller animals, including rodents, for studies where size is not an important part of the study.

However, instances still remain where it is required to use an animal that is large enough to render simulation as realistic as possible to obtain realistic results. Primates have always been felt to be the most realistic animal models for human research, but costs have increased such as to make them unusable for anything but the largest studies. Dogs and cats have been used extensively, but their use is decreasing because of noted differences in size, biology, and physiology between dogs and cats and humans, as well as cost issues and psychological issues related to the use of animals that are also domestic pets. Animals such as pigs,[1-3] sheep,[4-6] and goats[7-9] are becoming more frequently used as animal models, in part because of the larger and more realistic size and biological similarities to humans,[3,10-12] and also because there may be less emotion attached to their use.

The laws in the United States regarding the use of dogs and cats for animal testing and research involve both state and federal laws, which have undergone constant refinement since the first law was passed in Massachusetts in 1641 forbidding cruelty to domestic animals.[13] The general public appears to support the use of dogs and cats for medical and scientific research purposes, but the support has dropped to a little over 50%, while in 1950, it was 85%.[13]

The complete genomes for both the dog[14] and the cat[15] have now been sequenced, which potentially will allow them to be utilized in studies involving gene modulation. Currently, the dog and cat are utilized for a number of purposes related to scientific research[16]:

1. The study and modulation of normal disease processes in dogs and cats that in and of themselves mimic similar disease processes in humans.
2. The induction of disease processes in dogs and cats that are similar to diseases in humans.
3. The design, execution, and study of surgical procedures that are planned to study the possibility in humans, and conversely, to study surgeries that are already performed in humans, to investigate the biology and physiology of the procedures and study any possible modification.

The rest of this chapter will discuss these issues.

Dogs

In the maxillofacial region, dogs have a long history of use as an animal model, usually for hard-tissue (bone and teeth) research, but also for soft-tissue studies. The rationale often given for the use of dogs is that their size makes them large enough for realistic surgical procedures to be carried out. In fact, studies have shown that in some respects, they are about the smallest animal that can be used for some realistic studies of human maxillofacial procedures. Their teeth are also of similar size and pulpal pattern to those of humans, and they have similar periodontal ligament attachments as human teeth.[17,18] The bony architecture of the mandible and maxilla is also similar to humans, with Haversian systems (unlike rats), and they are the smallest animals with a similar pattern of bone remodeling to humans.[19,20] Growth, remodeling, structural and chemical properties, and mineralization of bone are similar to that of humans. Age-related bony changes are also similar to humans.[21] They are cooperative animals, and there is often no need for general anesthesia for relatively simple procedures such as obtaining photographs, making measurements, and even procedures such as cleaning teeth or adjusting dental appliances or braces. This can be a considerable advantage in some studies.

Bone turnover

It is generally agreed that the bone turnover time in dogs is faster than that in humans, and that fractures heal more rapidly in dogs than humans.[22] However, the process of fracture healing in dogs is essentially similar to that in humans.[22] Bone turnover time is normally expressed as sigma, which is one complete bone turnover cycle, including both a resorptive phase and an appositional phase.[23-27] This time for the beagle is felt to be two to three times that of a human,[27-32] although other studies have suggested it is around 1.5 times as great.[33] Variations are possible for a number of reasons. It is known that variations occur within the same species of dogs, and certainly, there are variations between species of dogs. There is also a variation depending on age. It must be remembered that most animal experiments involving dogs are carried out on fairly young dogs, usually for financial reasons, whereas equivalent human operations may be carried out on more mature humans. Bone turnover is obviously going to be more rapid in younger animals. There are also variations in bone turnover time for cortical bone versus medullary bone, and this should be taken into account when studies are compared. Studies carried out on animals may therefore not be directly transferred to humans[19] or even to clinical veterinary practice.

The critical size defect has been established for the canine cranium at around 20 mm, and for the canine mandible, it is at 15 mm if the periosteum is removed and 50 mm if it is intact.[34,35] A critical size defect is one which, if created, will not heal spontaneously. Critical size defects are used for research on bone grafting and reconstruction techniques involving a variety of osteoconductive and osteoinductive agents.[36,37]

Facial growth

As we move away from the use of primates in maxillofacial studies, dogs have been examined for their use in a number of studies of facial growth. Losken et al. determined that the mandibular growth pattern of the dog shows similar relative percentage changes in different regions in juvenile through adult stages, as do those of the human being, and suggested that dogs may be an acceptable, inexpensive alternative to primates in certain maxillofacial growth situations.[38] Similarly, dogs have also been studied for their ability to be used as spontaneous animal models of human disease. For example, many dogs suffer from maxillary brachygnathism as a natural phenomenon. In fact, it is even a requirement in the official standards established for certain breeds, including the English toy spaniel, French bulldog, English bulldog, and boxers.[39] Brachycephaly in dogs is a regional manifestation of achondroplasia and occurs naturally in some breeds.[40] Hereditary multiple exostosis has also been recognized to occur in several breeds, including the West Highland white terrier, cairn and Boston terriers, Great Danes, and Labrador retrievers.[36] Data suggest that these animals may make suitable models for investigation of the etiology and treatment of infantile cortical hyperostosis in human infants.[40,41] Dogs have also been used in studies involving orthognathic surgery and tooth viability.[17]

Periodontal disease

The beagle suffers naturally from periodontal disease and periodontal degeneration, and has been widely used in studies of periodontal disease and in evaluations of a variety of surgical and nonsurgical treatments of periodontal disease.[18,42] More recently, the beagle has been used in studies utilizing membrane technology as a method of treating periodontal disease in humans.[43-45] The critical-size periodontal defect has also been established for the beagle.[46]

Postoperative infection rates are extremely low in dogs, and this may also need to be taken into account in studies of healing following certain procedures. Single genomic beagle models have recently been developed, which should lead to more standardization of research data. Soft-tissue studies on dogs have included grafting materials for vestibuloplasty (increasing the amount of gingiva for dentures to rest on).[47]

Implants

Dogs have also been used extensively in studies on various aspects of osteointegrated dental implants for tooth replacement, including osteointegration time, degree of osteointegration, effects of early and late loading, and the effects of orthodontic forces.[48-56] Some of these studies have been combined with the use of growth factors.[43,49] Although these studies have been valuable, it does need to be borne in mind that the bone turnover time is more rapid in dogs than in humans, and thus, the studies may not be directly transferrable to the human situation or even to clinical veterinary practice.

Despite the possible dissimilarities between bone healing in dogs and humans, the canine model has become popular for investigating the management of periodontal disease, both around natural teeth and around implants, and the concept of a barrier membrane to encourage new bone growth. This has led to the concept of a critical-size supraalveolar periodontal defect and the related critical-size supraalveolar periimplant defect in the canine animal model. In the beagle dog model, this critical size defect is approximately 5 mm all around a natural tooth or implant.[50,51]

Distraction

Dogs have also been used to look at various aspects of distraction osteogenesis, which is a more recent technique for slowly moving facial bones by means of gradual distraction following an initial osteotomy.[57-62] Again, many of the parameters utilized in humans have been established from animal research utilizing dogs. However, it needs to be emphasized that the bone turnover rate is faster in dogs than in humans.

Cats

The use of cats as experimental animals in oral and maxillofacial surgery is not found as frequently as dogs, possibly because cats are smaller animals and therefore may not be quite as realistic a model, and also because they are not as cooperative as dogs and therefore may need anesthetizing more often for procedures to be carried out. Nevertheless, cats have been used for a variety of hard- and soft-tissue studies. They have been widely used for orthodontic studies involving tooth movement because the crown-to-root ratio and periodontal ligaments are similar to that of humans and therefore it is felt that the tooth movement obtained orthodontically on a cat may be realistically transferred to the human.[63-66] Studies have shown that the cat may be the most realistic nonprimate model for nasal growth studies to simulate results in humans.[67] In the soft tissues, studies involving the temporal muscle have been carried out on cats, and this appears to be related to the fact that cats have a prominent temporalis muscle, which is particularly amenable to surgical procedures.[-]

In the oral cavity, the cat is utilized for the study of gingivostomatitis since it is a naturally occurring disease model of human chronic oral inflammatory diseases including oral lichen planus, stomatitis, oral Crohn disease, and pemphigus. This prevalent, naturally occurring disease process permits optimization of stem cell dose, route of administration, and cell source, all of which can inform human clinical trials.[16]

Nerve studies

The cat has also been used for studies on the lingual and inferior alveolar nerves, with particular reference to the response of this nerve to injury, and spontaneous and surgically induced regeneration, and also the value of a number of nerve growth factors that may aid reconstruction and regrowth of the branches of the trigeminal nerve.[71-78] The rationale for utilizing the cat for these studies appears to be that there is a long history of its use, and the cat appears to lend itself to carrying out single neuron studies on the branches of the trigeminal nerve, which can be extremely valuable in a number of physiological studies. Injury to the inferior alveolar and lingual nerves occurs in humans as a result of trauma and a variety of surgical procedures, including wisdom tooth removal.[79,80] Loss of sensation, or dysesthesia, causes considerable morbidity and hence the necessity for a reliable animal model.[81]

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SECTION 3:

Exodontics

OUTLINE

CHAPTER 13

Principles of exodontics

Milinda J. Lommer

Definitions

Simple extraction: An extraction not requiring a gingival incision (other than within the sulcus) or sectioning of the tooth. Also called a closed, uncomplicated, or nonsurgical extraction.

Surgical extraction: An extraction that requires a gingival incision, bone removal, and/or sectioning of the tooth. Also referred to as an open or complicated extraction.

Elevation: The process by which the periodontal ligament is fatigued or torn and alveolar bone is expanded to facilitate removal of the tooth from the alveolus. Using an elevator as a lever, the tooth is lifted (elevated) from its alveolus.

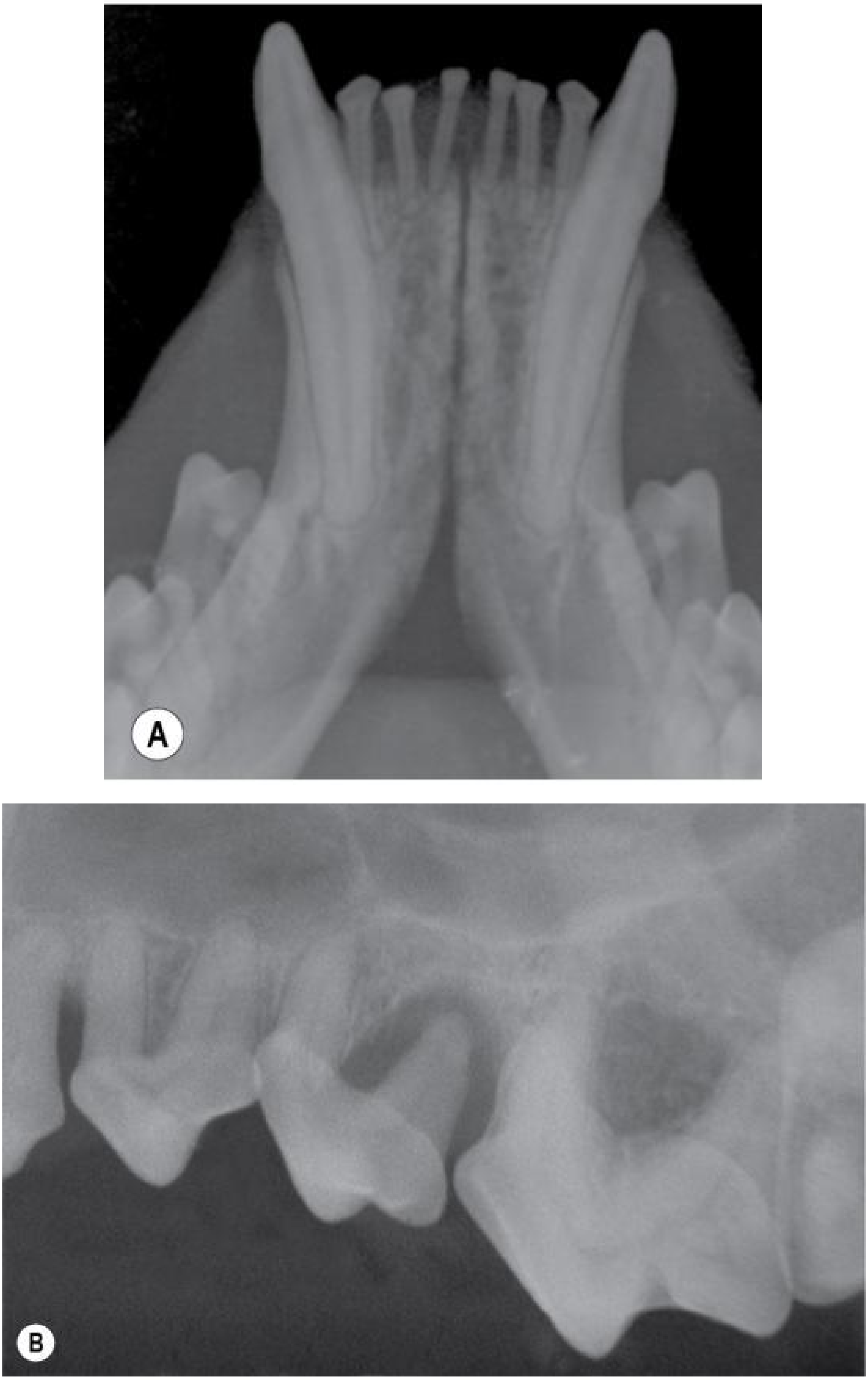
Luxation: The process by which the periodontal ligament is cut or severed to loosen teeth from the surrounding alveolar bone.

Indications for extraction

Periodontitis

Periodontal disease is frequently cited as the most common health problem affecting dogs and cats.[1-5] Because "periodontal disease" includes both gingivitis (inflammation of soft tissue) and periodontitis (dental attachment loss including bone and soft tissue), and large-scale health surveys typically have included only visual inspection of the oral cavity with no probing or dental radiographs, it is difficult to interpret the relevance of these data with regard to the prevalence of periodontal bone loss. However, in one study of 162 dogs randomly selected for oral radiography at necropsy, more than 70% of all dogs (aged 7 months to 14 years) were found to have alveolar bone loss.[6] Both incidence and severity of periodontitis appear to increase with age in dogs[1,6]: whereas fewer than half of dogs aged 7 months to 5 years demonstrated alveolar bone loss, more than 80% of dogs older than 6 years and 95% of dogs aged 12–14 years were affected.[6] Among cats presented for dental treatment, 72% were found to have periodontitis; this percentage did not vary significantly among age groups.[7]

Two distinct patterns of bone loss have been described in both dogs[6] and cats[7]: horizontal bone loss (decreased height of interradicular and interdental alveolar bone; Fig. 13.1A) and vertical bone loss (in which the deepest portion of the pocket is apical to the alveolar margin[8]; Fig. 13.1B). Vertical defects often affect a single root or single tooth and may not be detectable without radiography or probing, while horizontal bone loss frequently exposes furcations of multirooted teeth and is often accompanied by gingival recession.[6,7]



• **FIG. 13.1** (**A**) Horizontal bone loss affecting the mandibular incisor teeth of a cat: the alveolar margin remains perpendicular to the long axis of the roots but is displaced apically. (**B**) Vertical bone loss affecting the distal root of the left maxillary third premolar tooth of a dog: the alveolar margin is no longer perpendicular to the long axis of the root. The result is an infrabony pocket.

Extraction is the treatment of choice for teeth with significant clinical mobility or greater than 50% attachment loss. However, even in cases with less than 50% bone loss, if the furcation of a multirooted tooth is exposed (stage 3 furcation lesion), plaque retention will result in rapid progression of periodontitis at that location.[8] In human patients receiving treatment for periodontitis, furcation involvement is predictive of tooth loss.[9] Therefore, unless the clients are committed to performing meticulous home care, teeth with through-and-through furcation lesions should be extracted.[10] In selected cases and with dedicated clients, advanced techniques such as periodontal surgery and guided tissue regeneration, or hemisection and endodontic therapy, may allow preservation of periodontally compromised teeth.[11-14]

Pulp necrosis

Pulp necrosis may result from direct pulp exposure (e.g., complicated crown fractures), indirect pulp exposure (e.g., uncomplicated crown fractures, attrition, or abrasion), irreversible pulpitis in an otherwise intact tooth (e.g., gray-discolored, intact canine teeth), secondary to malformations, or as a result of severe periodontitis.[15-19] The inevitable sequela of untreated pulp necrosis is inflammation in the tissues surrounding the apex, which can result in destruction of the periapical bone and soft tissues.[20] Therefore, teeth with necrotic pulp should be treated, either by endodontic therapy (root canal treatment) or extraction.

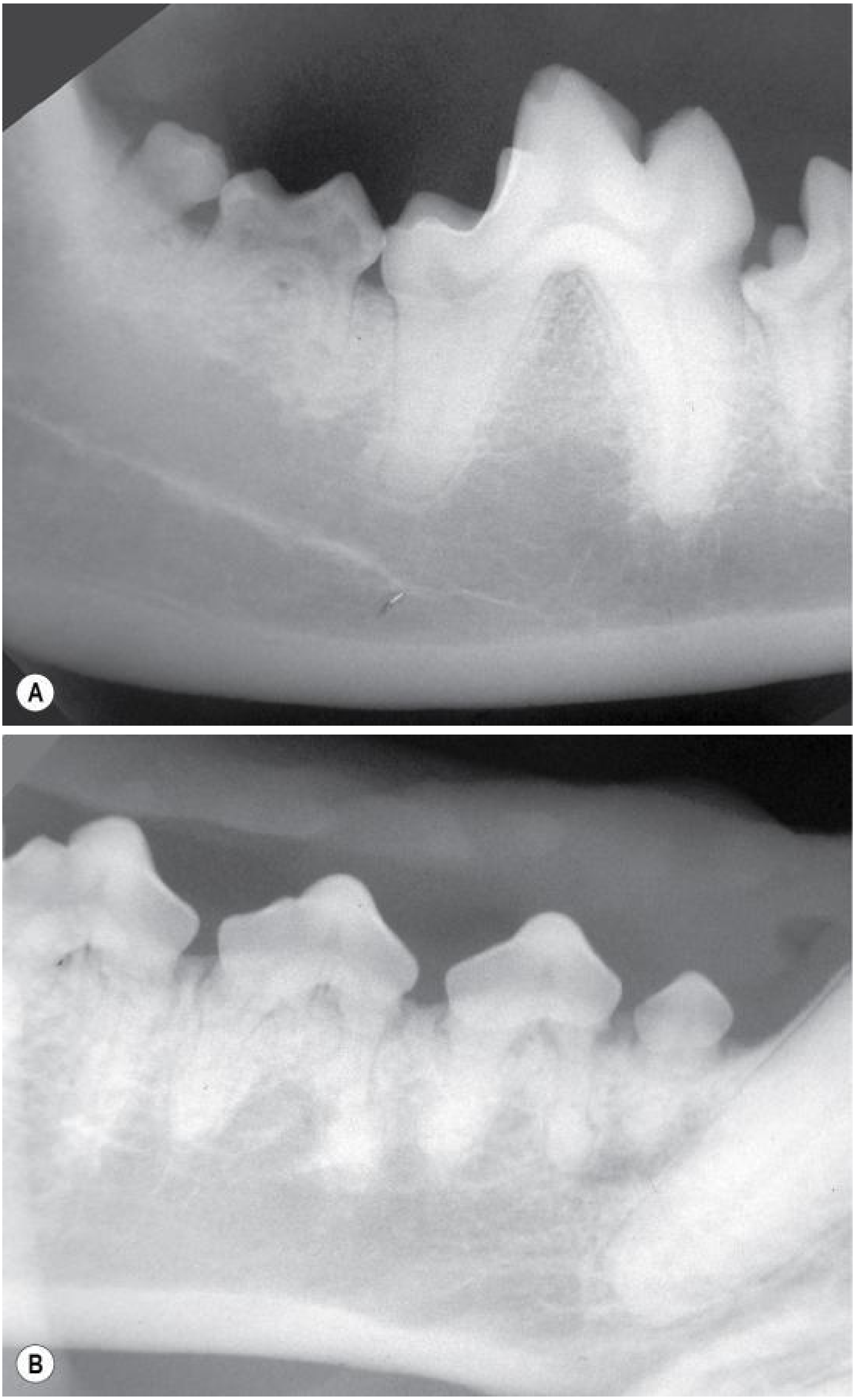
Dental fractures

Fractured teeth may be found in approximately 27% of dogs[21] and nearly 10% of cats presented for veterinary care (231 out of 2431 cats radiographed in the Dentistry and Oral Surgery Service at the University of California–Davis Veterinary Medical Teaching Hospital from 1994 to 2008 had complicated crown fractures). Advances in endodontic therapy for dogs and cats make this a realistic treatment option for many fractured teeth. However, endodontic therapy is not always feasible or advisable for every patient. Deep crown-root fractures extending apical to the alveolar margin will predispose the site to vertical bone loss, and extraction is therefore recommended for these teeth.[22] In addition, the need for additional anesthetic episodes for radiographic follow-up of root canal treatment[23] may preclude endodontic treatment in medically compromised patients, and extraction is advised for teeth with pulp necrosis in this population of patients.

Tooth resorption

Idiopathic tooth resorption is seen in up to two-thirds of domestic cats and increases in frequency with age.[24-27] Because lesions are painful and progressive regardless of attempts to treat conservatively,[28] extraction is the currently recommended treatment for teeth affected by resorption. Specific extraction techniques for teeth with intact roots and those with end-stage root resorption are discussed in Chapter 17.

While less common in dogs, resorption involving both crowns and roots has also been reported (Fig. 13.2A).[29,30] Age-related root resorption, seen in up to 43% of dogs aged 12–14 years,[6] does not affect the crowns, and extraction is generally not indicated for teeth with root resorption in the absence of coronal lesions (Fig. 13.2B).



• **FIG. 13.2** (**A**) Intraoral radiograph of idiopathic tooth resorption of the right mandibular second molar tooth in a dog. (**B**) Intraoral radiograph of age-related external replacement root resorption of the right mandibular premolar teeth in a dog.

Chronic gingivostomatitis

Gingivostomatitis consists of a number of syndromes, with the common presenting clinical symptom of severe inflammation of the gingiva and oral mucosa. Gingivostomatitis a is painful, debilitating condition affecting from 0.7%–12% of cats.[31] The initiating cause is usually not identified and may differ from case to case. Although less common in dogs than in cats, plaque-contact ulcerative stomatitis also occurs in dogs.[32,33]

Medical management has focused on reducing plaque bacteria (by means of professional dental scaling, home care, and antibiotic therapy) and minimizing the immune response (with corticosteroids and other immunomodulatory agents).[34,35] If these measures are not effective in reducing signs of stomatitis, extractions should be considered. Approximately two-thirds of cats[36] and nearly all dogs with gingivostomatitis will improve significantly after extractions.

Fractured deciduous teeth

Because deciduous teeth are long and narrow, and puppies are often very active chewers, fractured deciduous teeth are fairly common in dogs. As with permanent teeth, exposure of the pulp leads to bacterial infection, pulp necrosis, and extension of the infection through the apex and into the surrounding bone. The permanent tooth buds are located in close proximity to the apices of the deciduous teeth, and periapical inflammation from a fractured deciduous tooth may cause enamel hypoplasia or crown malformation in the successor tooth.[22,37] In dogs, the recommended treatment for fractured deciduous teeth is extraction, which, when performed carefully, is unlikely to cause iatrogenic trauma to the developing permanent tooth.[38] To prevent periapical inflammation and subsequent damage to the permanent tooth, a fractured deciduous tooth should be extracted as soon as it is detected.[38]

Persistent deciduous teeth

Persistent deciduous teeth are most common in small-breed dogs but are also seen in cats and large dogs.[38,39] The presence of a persistent deciduous tooth can force the permanent tooth to erupt in an abnormal location, causing malocclusion.[38,40] With time, the close proximity of the permanent and deciduous crowns will lead to rapid plaque accumulation and premature periodontitis. It is recommended that persistent deciduous teeth be extracted as soon as the crowns of the permanent successors are visible above the gingival margin.[38,40] Specific techniques for extraction of deciduous canine teeth are described in Chapter 15.

Malocclusion

Malocclusions can be skeletal (e.g., mandibular brachygnathism, maxillary brachygnathism, wry bite) or dental (e.g., linguoverted mandibular canines, rostral crossbite, and rotated teeth). Both skeletal and dental malocclusions can result in abnormal contact of teeth onto other teeth or soft tissue, which can cause periodontal bone loss, pulp necrosis, root resorption, and oronasal fistula.[38] For malpositioned, structurally and functionally important teeth, such as linguoverted mandibular canine teeth, orthodontic treatment or crown amputation with partial coronal pulpectomy would be preferred to extraction. Because it typically requires multiple anesthetic episodes, orthodontic treatment is neither feasible nor desirable for every patient with a malocclusion. To help prevent long-term problems, extraction should be considered for crowded teeth, rotated teeth, and teeth causing occlusal trauma (Fig. 13.3).

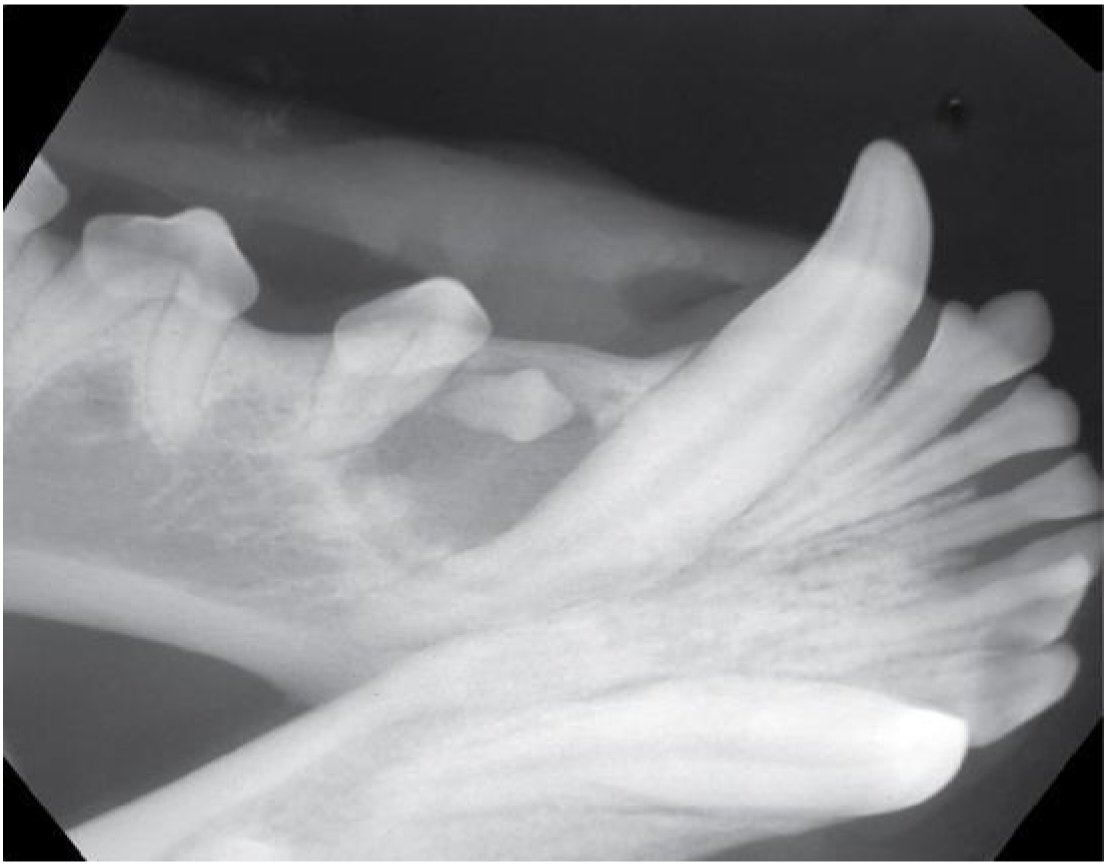
• **FIG. 13.3** Crowded incisor teeth in a dog; the right mandibular second incisor tooth is displaced lingually and should be extracted.

Supernumerary teeth

Supernumerary teeth are reportedly present in 11% of dogs.[41] In dolichocephalic breeds, such as greyhounds (in which there is a reported 36% incidence of supernumerary teeth[42]), no intervention is required. However, in brachycephalic breeds, or whenever a supernumerary tooth causes crowding and/or displacement of other teeth, the supernumerary tooth should be extracted as soon as it is discovered. Failure to do so may lead to plaque retention and premature periodontitis, ultimately resulting in the loss of adjacent teeth in addition to the supernumerary tooth.[38]

Unerupted teeth

Embedded or impacted teeth are frequently associated with dentigerous cyst formation (Fig. 13.4).[43-48] Canine teeth and mandibular first premolar teeth appear to be the most commonly affected teeth. The reduced enamel epithelium, which normally would form the junctional epithelium of an erupted tooth, produces fluid, which accumulates between the epithelium and the unerupted crown, causing expansion of the cyst and destruction of the surrounding bone.[49] Extraction of the embedded tooth and enucleation of the entire cyst lining must be performed to prevent recurrence of dentigerous cysts.



• **FIG. 13.4** Dentigerous cyst surrounding an unerupted mandibular first premolar tooth.

Teeth associated with pathologic lesions

In patients with nonresectable oral tumors, teeth with significant periodontal bone loss, evidence of pulp necrosis, or surrounded by neoplastic tissue should be extracted prior to radiation therapy, to minimize the risk of osteoradionecrosis.[10,50]

Teeth involved in jaw fractures

Severely fractured teeth or periodontally compromised teeth in a mandibular or maxillary fracture line should be extracted to reduce the risk of infection at the fracture site. However, periodontally sound teeth may be retained, as they may contribute to stabilization of the fracture, and manipulation of the fracture fragment during extraction attempts could cause further damage.[10]

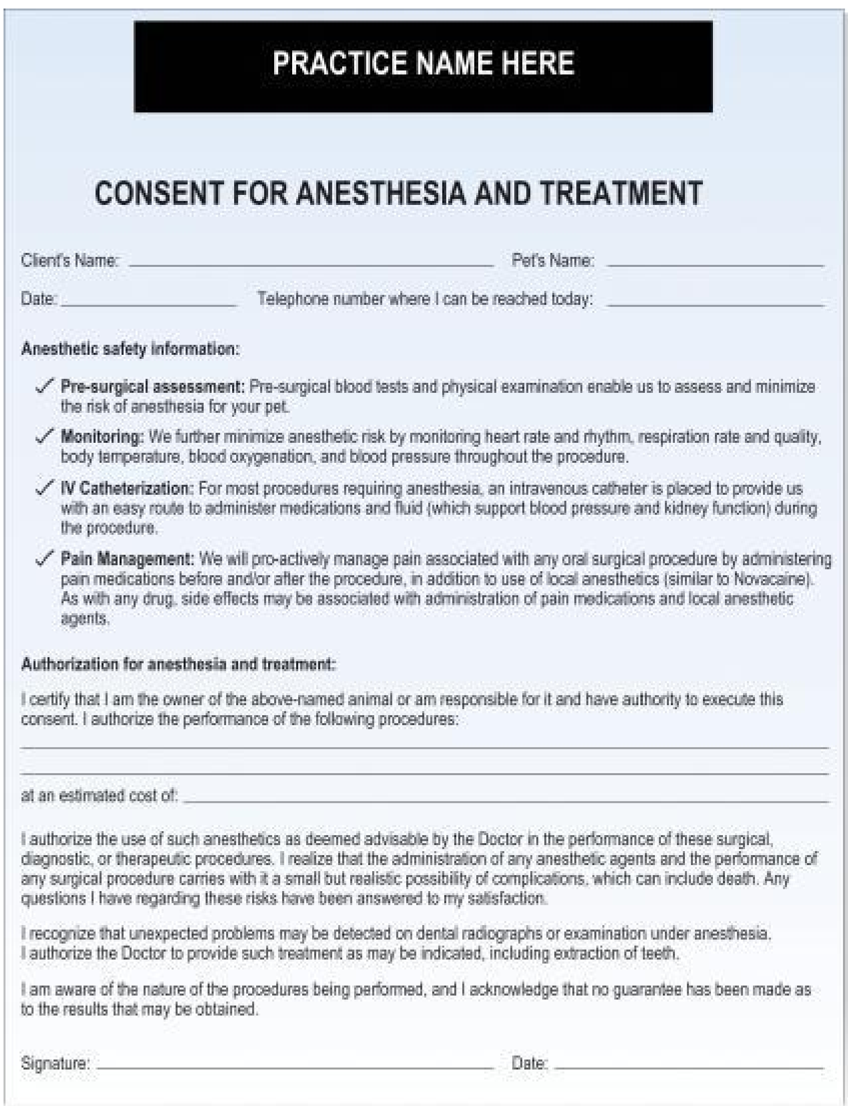
Failed endodontic treatment

Recheck radiographs are required to evaluate whether endodontic treatment has been successful in preventing or resolving periapical inflammation.[23] Although the reported failure rate of root canal treatment in dogs is relatively low at 6%,[23] when endodontic failure does occur, extraction must be presented as the definitive treatment option, alongside conservative therapies such as retreatment or surgical endodontic treatment (apicoectomy).

Contraindications for extraction

General contraindications

If a client has not been informed that extractions might be necessary, every attempt should be made to contact the client for approval prior to extracting teeth. It is prudent to include a paragraph on the anesthesia release form (Fig. 13.5) granting permission to extract teeth in the event that the client cannot be reached.



PRACTICE NAME HERE

CONSENT FOR ANESTHESIA AND TREATMENT

Client's Name: ______________________ Pet's Name: ______________

Date: __________ Telephone number where I can be reached today: ______________

Anesthetic safety information:

- ✓ **Pre-surgical assessment:** Pre-surgical blood tests and physical examination enable us to assess and minimize the risk of anesthesia for your pet.
- ✓ **Monitoring:** We further minimize anesthetic risk by monitoring heart rate and rhythm, respiration rate and quality, body temperature, blood oxygenation, and blood pressure throughout the procedure.
- ✓ **IV Catheterization:** For most procedures requiring anesthesia, an intravenous catheter is placed to provide us with an easy route to administer medications and fluid (which support blood pressure and kidney function) during the procedure.
- ✓ **Pain Management:** We will pro-actively manage pain associated with any oral surgical procedure by administering pain medications before and/or after the procedure, in addition to use of local anesthetics (similar to Novacaine). As with any drug, side effects may be associated with administration of pain medications and local anesthetic agents.

Authorization for anesthesia and treatment:

I certify that I am the owner of the above-named animal or am responsible for it and have authority to execute this consent. I authorize the performance of the following procedures:

____

____

at an estimated cost of: ______________________

I authorize the use of such anesthetics as deemed advisable by the Doctor in the performance of these surgical, diagnostic, or therapeutic procedures. I realize that the administration of any anesthetic agents and the performance of any surgical procedure carries with it a small but realistic possibility of complications, which can include death. Any questions I have regarding these risks have been answered to my satisfaction.

I recognize that unexpected problems may be detected on dental radiographs or examination under anesthesia. I authorize the Doctor to provide such treatment as may be indicated, including extraction of teeth.

I am aware of the nature of the procedures being performed, and I acknowledge that no guarantee has been made as to the results that may be obtained.

Signature: ______________________ Date: ______________

• **FIG. 13.5** Sample anesthesia release form, with a paragraph authorizing extraction of teeth in the event that the client cannot be reached during the procedure.

It should be noted that extracted teeth, unlike other tissues removed from a patient, remain the legal property of the client. It is advisable to clean the extracted teeth (by soaking in hydrogen peroxide or bleach), place them in gauze inside an envelope or plastic vial labeled with the patient's identity and date, and offer them to the client at the time of discharge.

Other legal aspects to consider include the role of veterinary technicians in performing dental extractions. The American Veterinary Dental College "considers the extraction of teeth to be included in the practice of veterinary dentistry. Decision making is the responsibility of the veterinarian, with the consent of the pet owner, when electing to extract teeth. Only veterinarians shall determine which teeth are to be extracted and perform extraction procedures."[51] Many states and provinces specifically prohibit the performance of dental extractions by veterinary technicians.[52,53] In other states, licensed veterinary technicians may be allowed to perform only simple extractions (i.e., those extractions not requiring creating a flap, removal of bone, or sectioning of teeth).[54] There are also states with less well-defined laws, allowing registered veterinary technicians to perform "dental extractions" under the direct supervision of a licensed veterinarian but prohibiting technicians from performing "surgery."[55] The American Veterinary Medical Association maintains a summary report regarding each state's regulations pertaining to technicians and dental procedures.[56] In any case, the legal responsibility for the welfare of the patient is the veterinarian's alone, and the supervising clinician should ensure that veterinary technicians performing dental procedures are properly trained.

Similarly, extractions should not be performed when proper instruments, appropriate equipment (e.g., for creating flaps, removing alveolar bone, and sectioning multirooted teeth), and adequate lighting are not available. The armamentarium for oral surgery is discussed in Chapter 7.

On occasion, veterinarians are asked to extract the canine teeth of an aggressive animal. However, extraction of the canine teeth does not render the animal harmless, and significant damage can be inflicted by premolar and molar teeth. To successfully manage an aggressive patient and prevent injury to family members or other animals, the underlying behavior issue must be addressed through behavior modification and, when indicated, medical management.

Systemic contraindications

Many patients presented for dental care will have systemic disorders, such as renal disease, mitral valve insufficiency, diabetes, hyperthyroidism or hypothyroidism, or hyperadrenocorticism. As long as the disease process is well controlled, general anesthesia and oral surgery are not contraindicated. However, treatment should be deferred in patients with congestive heart failure, uremia, uncontrolled endocrine disorders, severe coagulopathies, or untreated leukemia or lymphoma.[57]

Local contraindications

Extraction of teeth in the field of previous radiation treatment may lead to osteoradionecrosis and should be avoided. Whenever possible, periodontally compromised or fractured teeth should be removed prior to radiation therapy.[57]

It has been suggested that extraction of teeth within a malignant neoplasm may disseminate tumor cells into the bloodstream and hasten metastasis[57]; therefore, teeth embedded in suspected tumors should ideally not be extracted at the time of biopsy, but rather should be removed along with the surrounding tumor at the time of definitive surgical treatment once a diagnosis has been made.[57]

Clinical evaluation of teeth for extraction

Mobility

The mobility of each tooth should be evaluated preoperatively as part of the oral health assessment and charting. Increased mobility may be associated with extensive bone loss, which facilitates extraction and reduces the need for an open approach.[10,57] In the absence of significant periodontitis, increased mobility is likely due to a root fracture, which may necessitate an open approach, and should be verified radiographically before proceeding.

Condition of crown

Teeth with extensive coronal resorption are likely to fracture during extraction attempts. An open approach to extraction and placement of extraction forceps as far apically as possible is advised for teeth with destruction of large portions of the crown.[57]

Radiographic evaluation of teeth for extraction

Preextraction radiographs are a vital part of treatment planning. It is important to determine the configuration of the roots, proximity to adjacent teeth and other structures, and the condition of the surrounding bone.[57]

Configuration of the roots

In a survey of 226 dogs, 23.0% were found to have fused roots, 21.7% had root resorption or ankylosis, 12.8% had hypercementosis, 10.7% had supernumerary roots, 3.5% had root dilacerations (Fig. 13.6), and 3.1% had root fractures.[41] In cats, root resorption was identified in 35.6%.[58] Each of these factors could complicate extraction attempts, and knowledge about root status before extraction planning will facilitate uncomplicated completion of the procedure.

• FIG. 13.6 Dilacerated roots of the right mandibular fourth premolar and first molar teeth.

Proximity to adjacent teeth and other important structures

The relationship of the tooth to be extracted to adjacent erupted or unerupted teeth should be determined radiographically.[57] When bone removal is necessary, it is important to use caution to preserve the attachment of adjacent teeth. When extracting deciduous teeth, gentle technique must be employed to avoid damaging the underlying permanent tooth buds. On the maxilla, the infraorbital artery, vein, and nerve exit the infraorbital foramen dorsal to the distal root of the maxillary third premolar tooth.[59] On the caudal palate, the major palatine artery exits the major palatine foramen (located medial to the fourth premolar tooth) and runs rostrally to the palatine fissure, where it anastomoses with branches of the infraorbital and nasal vessels. The parotid salivary duct opens into the oral cavity through a papilla at the rostral end of a ridge of mucosa, dorsal to the distal root of the maxillary fourth premolar tooth.[59] The zygomatic papilla, through which the major duct from the zygomatic salivary gland exits, is located about 10 mm caudal to the parotid papilla, dorsal to the maxillary first molar tooth.[59] It is possible to damage any of these important structures during attempts to extract the maxillary premolar and molar teeth. Significant differences exist among breeds, making radiographs of each patient imperative prior to commencing extractions.

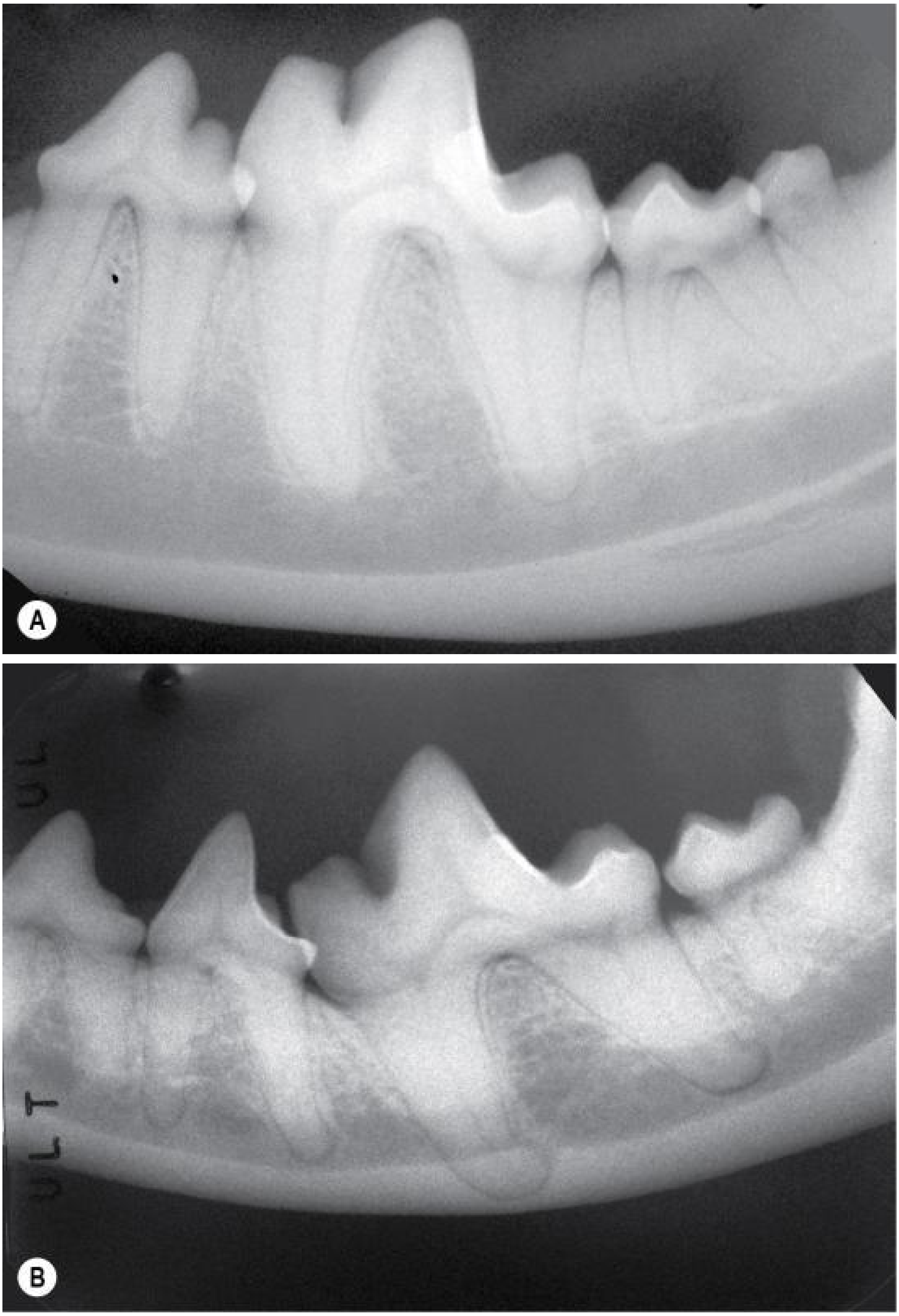
The maxillary recess is a lateral diverticulum of the nasal cavity, the opening of which lies in a transverse plane through the mesial roots of the maxillary fourth premolar tooth.[59] It is possible to dislodge a root into this recess if excessive force is used during extraction attempts. In brachycephalic breeds, the orbit lies directly dorsal to the maxillary fourth premolar, first molar, and second molar teeth. Orbital penetration has been reported during attempts to extract these teeth.[60]

The mandibular canal, containing the inferior alveolar artery, vein, and nerve, is located on the ventral aspect of each mandible. A root or root fragment may be inadvertently displaced into the mandibular canal if inappropriate force is applied during attempts to remove the root.

Condition of the surrounding bone

The density of the bone surrounding the tooth to be extracted may be assessed radiographically; bone that is more radiolucent may be less dense, making extraction easier, while bone with increased opacity may make extraction more difficult.[57] In addition, periapical lucencies may be present around roots of nonvital teeth, and the associated granulomas or cysts should be debrided following removal of the roots.[57]

The proportion of the mandible occupied by the teeth varies with breed; small dogs have proportionally larger mandibular first molar teeth relative to the height of the mandible when compared with large dogs (Fig. 13.7).[61] It is possible to cause iatrogenic fracture of the mandible during attempts to extract mandibular canine or first molar teeth if excessive force is applied or if extensive bone loss is present. The more information the operator has before initiating surgery, the less likely the patient is to suffer such complications, which underscores the importance of preoperative radiographs.



• **FIG. 13.7** Intraoral radiographs of the mandibular premolar and molar teeth of a large (**A**) and small (**B**) dog. Notice the location of the apices of the roots of the first molar tooth in relation to the ventral border of the mandible.

Patient and surgeon preparation

Patient positioning is a matter of operator preference. Lateral recumbency is preferred by some veterinarians, allowing drainage of fluids and good visibility of the buccal surfaces of the uppermost teeth, but providing limited visibility and restricted access to the teeth on the opposite quadrant.[10] The patient must therefore be turned during the procedure to access teeth on the opposite side. Dorsal recumbency allows excellent visualization of all teeth, which is particularly helpful when sectioning the maxillary fourth premolar and first molar teeth, and allows the patient to remain in one position throughout the procedure. To prevent fluid accumulation in the mouth of a patient in dorsal recumbency, a sandbag, "pool noodle" (Fig. 13.8), or rolled towel placed under the neck will help tilt the nose downward. Use of a pharyngeal pack, cuffed endotracheal tube, and suction is also recommended when working with a patient in dorsal recumbency.[10]

• **FIG. 13.8** Use of a "pool noodle" for positioning of the patient's neck while in dorsal recumbency.

The use of a pharyngeal gauze pack is recommended regardless of patient positioning to prevent teeth or fragments of teeth from entering the oropharynx, where they could be aspirated or swallowed during recovery.[57]

Removal of calculus prior to extractions will allow more accurate assessment of the tooth structure and provides a cleaner environment for surgery. Rinsing the oral cavity with a 0.12% chlorhexidine gluconate solution prior to the procedure will reduce bacteremia and aerosolized bacteria.[10,62] Although it is impossible to render the oral cavity a “sterile” environment, aseptic technique should be used for open extractions. Instruments should be sterilized prior to use, and the use of drapes is recommended to prevent calculus, hair, and other debris from contaminating the surgical field.[10]

To prevent contact with aerosolized bacteria and fluid particles, the operator should wear a mask, gloves, and protective eyewear. Because, occasionally, a fragment of tooth or a fractured dental bur will become airborne, hard plastic goggles are recommended instead of simple splash-proof face shields, which may not protect against ocular injury.[10] Long hair should be tied back or preferably covered with a surgical cap.[57]

Instruments and materials

Instruments for creating mucogingival flaps

A # 15 blade is typically used to incise the gingiva and mucosa. The round scalpel handle no. 5 (see Fig. 7.1B) is easier to hold in a modified pen grasp (see Fig. 7.2) than is a flat scalpel handle. The round handle allows it to be rotated, which facilitates following the contour of the tooth when making a sulcular incision. A periosteal elevator such as a # 24G (see Figs 7.7B and 7.8) or Molt # 9 (see Fig. 7.7A) is employed to reflect the gingiva or mucosa from the bone as a single layer with the underlying periosteum.

Tissue retractors

Once a mucoperiosteal flap is elevated and bone is exposed, the flap is reflected or retracted using a periosteal elevator, to protect it during osseous surgery or sectioning of a tooth. The same periosteal elevator used for elevating the flap can be used for this purpose. Some retractors, such as the Cawood-Minnesota retractor (see Fig. 7.9B), are specifically made for keeping tongue, lips, and cheeks away from the surgical site.

Instruments for sectioning teeth and removing alveolar bone

Air-driven dental handpieces are common in veterinary practice, and use of high-speed handpieces with either carbide or diamond burs has largely replaced other methods for sectioning teeth and removing bone. Cross-cut fissure carbide or tapered diamond burs are useful for sectioning multirooted teeth, while round diamond burs are ideal for removal of alveolar bone (Fig. 13.9). Dental handpieces should be held with a modified pen grasp (Fig. 13.10).

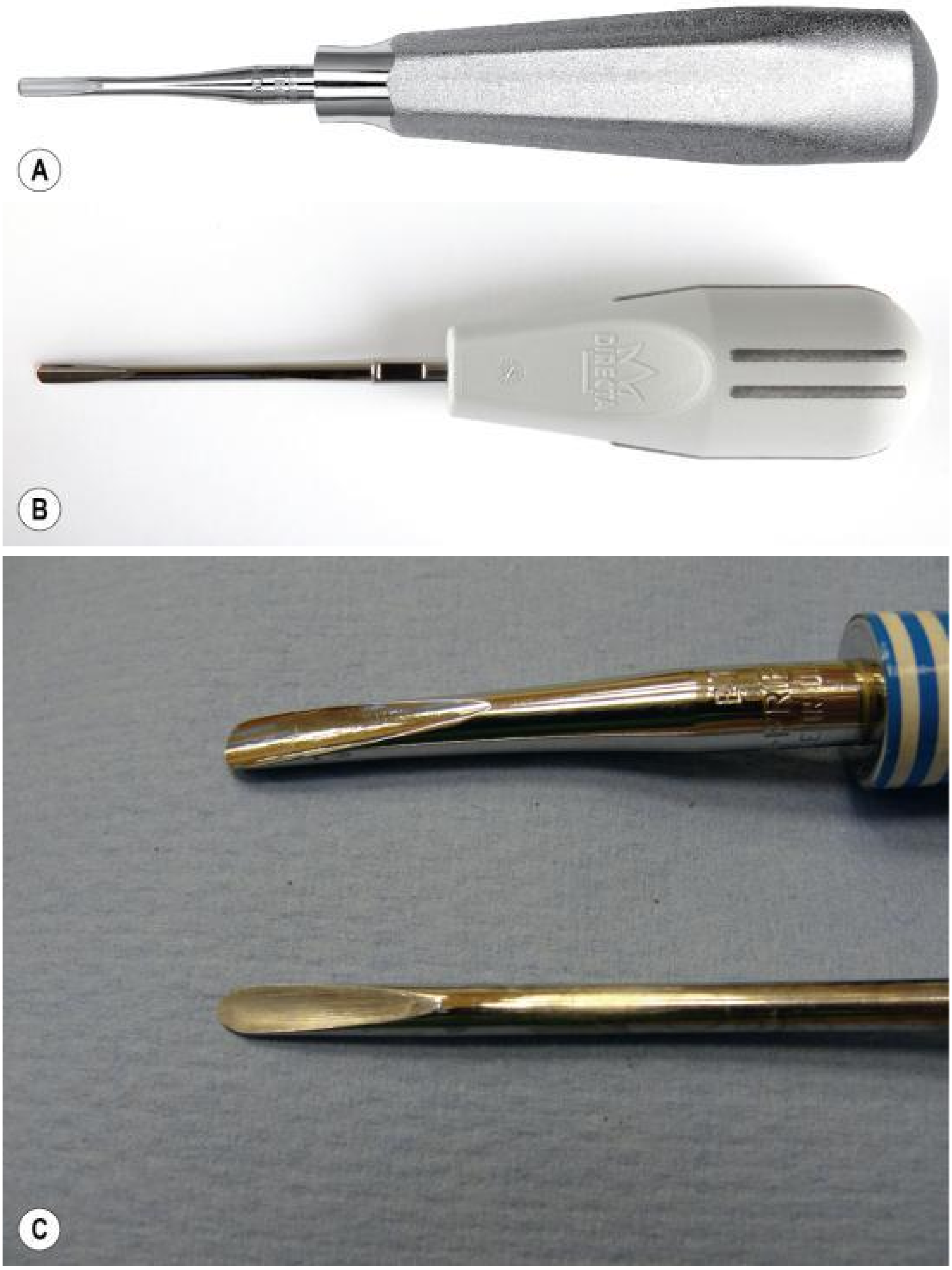
• FIG. 13.9 A sterilizable bur block containing (*left to right*) a cross-cut fissure carbide bur, tapered diamond bur, 1/2-round carbide bur, and small, medium, and large round diamond burs.

• FIG. 13.10 High-speed dental handpiece held in a modified pen grasp.

Dental elevators

One of the most commonly used instruments in dental extractions is the elevator (e.g., Seldin # 304W; Hu-Friedy Mfg. Co., Chicago, IL), which is used to loosen teeth prior to application of the extraction forceps. The elevator is used as a lever, transmitting rotational force from the handle to the blade, to tear Sharpey fibers and to lift the tooth from its alveolus.

Elevators consist of three components: a handle, a shank, and a blade (Fig. 13.11A). The handle is typically made of steel and is of a substantial size, so it may be comfortably held in the palm of the hand and used to apply controlled force. The steel shank connects the handle to the blade and must be strong enough to transmit the force from the handle to the blade. The blade is the working end of the elevator, which is used to transmit force to the tooth or alveolar bone. Its width may vary from 2 to 4 mm. The blade is made of strong steel and has a concave surface on its working side, so that it may be used in the same manner as a shoehorn.[63]



• FIG. 13.11 (**A**) Seldin # 304W elevator. (**B**) Luxator. (**C**) Close-up of blades of elevator *(top)* and luxator *(bottom)*. The flat blade of the luxator is designed to enter the periodontal space to cut the periodontal ligament fibers, while the semicircular shape of the elevator blade is better for leveraging against the tooth to fatigue and tear the periodontal ligament fibers. Source: (A, Courtesy Hu-Friedy, Chicago, IL.)

Some elevator blades have sharp tips, which may be used in a similar manner as luxators, to cut the periodontal ligament rather than fatigue it. When using elevators in this fashion, the blade is placed parallel to the long axis of the root and advanced apically.

Elevators are available with a straight shank and blade, or with the blade offset at an angle from the shank. The angled elevators are designed to facilitate access to the caudal areas of the mouth but must be used with care, as the forces applied to the handle do change direction with the angle of the blade.

Triangular-shaped elevators, such as Cryer elevators (Hu-Friedy Mfg. Co., Chicago, IL), come in pairs (left and right) and are designed for use with a "wheel-and-axle" motion. The tip of the elevator is placed into the alveolus, with the shank on the buccal alveolar bone perpendicular to the root. The sharp tip of the elevator is used to engage the cementum of the root surface, the handle is turned, and the root is thus elevated from the alveolus.[63]

Winged elevators have recently become popular in veterinary dentistry. These elevators have a short shaft and large-diameter handles for improved control and more comfortable use by clinicians with smaller hands.[64] The winged blades, available in 1.5-, 2.5-, 3.5-, and 4.5-mm widths, conform to roots of various circumferences, achieving better purchase on the tooth surface. However, if too much torque is placed on small teeth, root fracture may occur, so care must be employed.

Luxators

The luxator is a sharp instrument with a less concave blade than an elevator (Fig. 13.11B–C). It is used to cut or sever Sharpey fibers within the periodontal ligament and loosen the tooth prior to extraction. The shank and blade are placed parallel to the root surface of the tooth, and the tip of the luxator is pushed into the alveolar socket. Because the luxator is not used as a first-class or wheel-and-axle lever, the handle does not need to transmit rotational forces and is usually made of plastic rather than steel. The shank and blade are made of softer steel than that of an elevator. The thin blade is designed to be resharpened frequently.[65] Luxators (e.g., Ericsson luxators; JS Dental Manufacturing, Inc., Ridgefield, CT) are available in widths of 1–5 mm and with a straight or angled blade. The gouge (e.g., Coupland gouge) is related to the luxator. It has sharp straight-edged blades and is semitubular in shape. Luxators should be held in the palm with the index finger extended toward the tip of the blade (Fig. 13.12). This will minimize trauma to the patient in the event of instrument slippage.

• **FIG. 13.12** Proper grasp of a luxator. The index finger is extended along the shaft toward the tip of the blade to minimize trauma to the patient should the instrument slip during extraction.

Periotomes

Traditional luxation and elevation techniques often involve alveolectomy. An alternative technique involves the use of a periotome, a thin-bladed instrument that is inserted into the periodontal ligament space to sever the collagen fibers and allow extraction of a tooth while preserving the anatomy of the alveolus.[63] There are both handheld and motorized periotomes, with exchangeable or fixed blades. The blade is inserted into the periodontal ligament space and advanced apically approximately 2–3 mm then withdrawn and reinserted into an adjacent site. The process is repeated around the circumference of the tooth, advancing the blade further apically each time, until a depth of approximately two-thirds the distance to the apex is reached. At this point, the tooth can be removed by exerting gentle rotational forces in a coronal direction with extraction forceps. Use of a motorized periotome has been advocated for preserving alveolar bone during extraction prior to immediate dental implant placement,[66,67] and periotome use was associated with shorter surgery times and lower postoperative pain scores than conventional extraction techniques in one investigation.[68]

Root tip picks

There are two types of root tip picks: the apical elevator (Fig. 13.13A) and the root tip “teaser” (Fig. 13.13B). The apical elevator has a handle, shank, and blade similar to a standard dental elevator, but with a smaller diameter handle and a sharper, narrower blade. It may be used as a lever to engage a root fragment and lift it from the alveolus. After drilling a 2–3-mm hole in the root with a bur, the tip of the pick is inserted into the hole and, using the buccal bone as a fulcrum, the root is elevated from the alveolus.[63] These root tip picks can also be used as elevators on very small teeth, such as incisors in the cat. The root tip teaser has a long narrow handle between two angled working ends, which are mirror images of one another. It is a thin, delicate instrument that is used to tease small root tips from their alveoli (see Chapter 16).[63] In contrast to an apical elevator, wheel-and-axle or leverage forces applied to a root tip teaser will damage the instrument.

• **FIG. 13.13** (**A**) Apexo # 301 apical elevator. (**B**) Davis # 11 root tip teaser. Source: (Courtesy Hu-Friedy, Chicago, IL.)

Extraction forceps

Extraction forceps are used after elevation or luxation to grasp the loosened tooth and remove it from the alveolus.[63] The three components of extraction forceps are the handle, hinge, and beak. The handles should be of sufficient size to grasp comfortably; they are usually serrated to prevent slippage and may be straight or curved. The hinge transfers the force applied to the handles to the beak. The beak is designed to adapt to the tooth root at the cemento-enamel junction and should be placed parallel to the long axis of the tooth. It is not designed to grasp the crown of the tooth. Narrow beaks should be used for smaller teeth, and wider beaks, for larger teeth. The more closely the forceps' beaks adapt to the tooth roots, the more efficient will be the extraction.[63] The chance for root fracture increases if the beaks are not properly adapted to the root surface. Extraction forceps should be grasped in the palm, with the index finger between the handles (Fig. 13.14) to avoid generating excessive pressure, which might crush the tooth.

• **FIG. 13.14** Proper grasp of extraction forceps. The index finger is placed between the handles to prevent generation of excessive force, which can lead to crushing of the tooth.

In human dentistry, specific extraction forceps have been designed for each kind of tooth. Many of these forceps are unsuitable for use in the dog and cat. Sharply curved forceps, such as the so-called "lower molar forceps," and forceps with sharp triangular tips are most likely to cause root fractures. Extraction forceps designed for veterinary use are available, but several extraction forceps designed for use in human patients can be successfully used in veterinary practice. For example, so-called "upper anterior forceps" (such as the pedodontic Cryer # 150S [Fig. 13.15A]; Hu-Friedy Mfg. Co., Chicago, IL) have been found to be suitable for use in the dog and cat.[69,70] These forceps have a slightly conical grip and fit most teeth, in spite of the great variation that exists in the size and shape of teeth in dogs and cats. Root forceps, such as the # X49 forceps (Hu-Friedy Mfg. Co., Chicago, IL) (Fig. 13.15), differ from conventional extraction forceps in that they have long, narrow beaks that close completely, making them particularly useful for grasping small root fragments.

• **FIG. 13.15** (A) F-150S Cryer forceps. (B) FX-49 forceps. Source: (Courtesy Hu-Friedy, Chicago, IL.)

Instruments for suturing flaps

Fine suture material with small, swaged-on needles are generally used in oral surgery (see Chapter 8). The needle holders indicated in oral surgery are therefore delicate to match the size of the needles. The Halsey needle holder (see Fig. 7.11A) is a very versatile needle holder well suited for most intraoral procedures. Similarly, the delicate Adson 1X2 tissue forceps (see Fig. 7.5A) is used most commonly in oral surgery for gentle tissue handling. The choice of suture scissors is based on operator preference. Some surgeons working without assistants prefer a needle holder with built-in scissors, such as the Olsen-Hegar needle holder (see Fig. 7.11D).

Treatment planning

Simple versus surgical extraction

Also called a closed, uncomplicated or nonsurgical extraction, a simple extraction is performed without incising the gingiva (other than within the gingival sulcus) or sectioning the tooth. An extraction that requires a gingival incision, bone removal, and/or sectioning of the tooth is a surgical extraction, also known as an open or complicated extraction.

Small, single-rooted teeth are typically extracted in a simple fashion. Maxillary second molar teeth in the dog often have three partly fused roots, so these teeth are also usually extracted with a simple technique. In some cases of severe periodontitis, extensive bone loss may enable simple extraction of a multirooted tooth, but a surgical approach is recommended for most multirooted teeth. Canine teeth should also be extracted with a surgical technique in most cases; simple extraction of a periodontally compromised maxillary canine tooth can lead to formation of a permanent oronasal fistula. Surgical extraction should also be considered for smaller, single-rooted teeth that are ankylosed or have undergone root resorption (e.g., severely abraded incisor teeth with pulp exposure in dogs with a history of frequent chewing).

When determining whether to perform simple or surgical extraction, the clinician must consider clinical findings, such as mobility, and radiographic findings, including root morphology and clinical attachment level. Significant bone loss and the resulting increase in mobility associated with periodontal disease usually leads to simple tooth extraction. On the other hand, if a tooth is being extracted due to fracture or some cause other than periodontal disease and is periodontally healthy, normal mobility is expected, and considerable resistance to extraction may be encountered. Similarly, if radiographs reveal dilaceration or other abnormalities in root morphology, electing a surgical approach initially will save time compared with the effort required to extract a root fragment following an unsuccessful attempt at a simple extraction. Regardless of whether a simple or surgical extraction is elected, the tissues should be handled with care, the alveoli flushed gently to remove debris, and, for all but the smallest simple extraction sites, the gingiva sutured to allow primary-intention healing.

Principles of flap design, development, and management

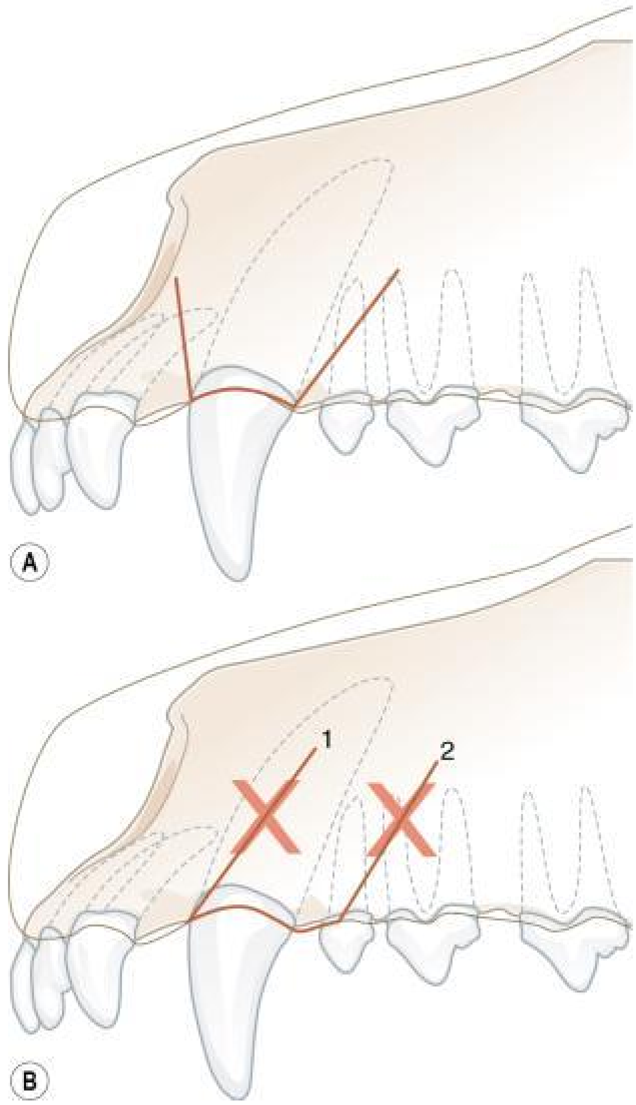
Local flaps are employed for open extractions; the term “local flap” refers to a section of soft tissue (gingiva and/or mucosa and periosteum) that is outlined by a surgical incision, contains its own blood supply, allows access to underlying tissues, can be replaced in its original position, and is expected to heal after being sutured in place.[71] Proper extraction technique requires that the operator have a clear understanding of the principles of design and management of local flaps.

Design parameters for soft-tissue flaps

When designing a flap, several goals must be achieved: the flap must be of sufficient size to allow adequate exposure of the surgical area, the base of the flap must be as wide as or broader than the free margin to preserve its blood supply, and the edges of the flap must lie over intact bone at the conclusion of the procedure.[71] If the incisions are unsupported by sound bone, the flap tends to collapse into the bony defect, which can lead to delayed healing and wound dehiscence.[71]

When designing the flap, it is important to consider adjacent vital structures, such as the infraorbital artery, vein and nerve on the maxilla, and the middle mental neurovascular bundle exiting the middle mental foramen on the mandible.

To minimize tension on the suture line and prevent a defect in the attached gingiva following healing, vertical releasing incisions should be made at line angles of adjacent teeth rather than directly on the buccal aspect of a tooth (Fig. 13.16).



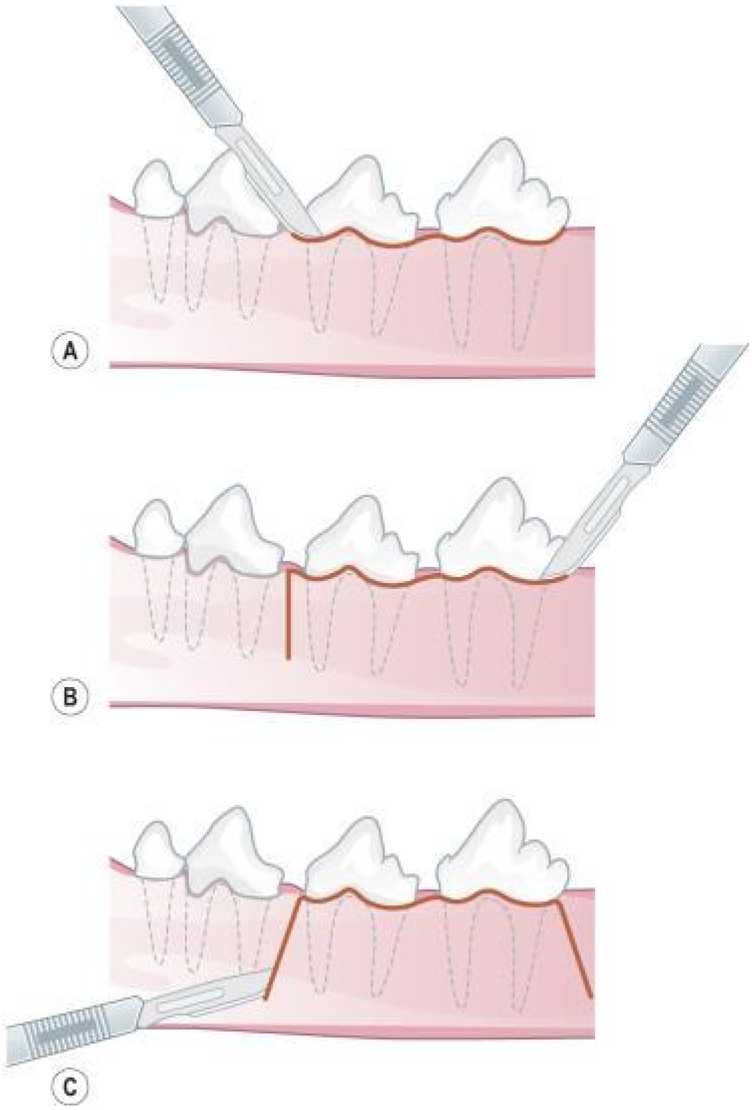
• **FIG. 13.16** (**A**) Correct placement of releasing incisions at mesiobuccal and distobuccal line angles. Incisions are oblique to allow a wide-based flap. (**B**) These two incisions are made incorrectly: (1) crosses the area of bone removal, leaving the incision over an empty space and (2) crosses the attached gingiva directly over the buccal aspect of the adjacent tooth, which will likely result in a gingival defect.

Sulcular incision versus excision of the free gingiva and sulcus epithelium

The purpose of making a sulcular incision is to free the gingival attachment from the tooth to provide increased visualization of and access to the underlying alveolar bone and periodontal ligament space. In cases of periodontitis, the sulcular epithelium is diseased "pocket epithelium," with macrophages, neutrophils, and lymphocytes creating increased distances between epithelial cells and disrupting epithelial basal lamina.[73] Some surgeons prefer to excise the free gingiva (i.e., the 1–2-mm section of gingiva that is not attached to the tooth surface) and the pocket epithelium rather than simply incising the sulcus.[73] While there are no published studies demonstrating that excising the pocket epithelium and free gingiva leads to more rapid healing of extraction sites in dogs or cats with periodontitis, bone regeneration may occur more rapidly in the absence of diseased epithelium. In one study of human dental patients, alveoli of teeth extracted due to advanced periodontitis contained mostly connective tissue for 14 weeks after extraction, with >50% new bone fill noted after 16 weeks, while alveoli of teeth extracted for nonperiodontal reasons showed >50% new bone fill after 8 weeks.[74] Although bone regeneration occurred more slowly in diseased extraction sites than disease-free sites, no long-term differences were observed.

Types of gingival and mucogingival flaps

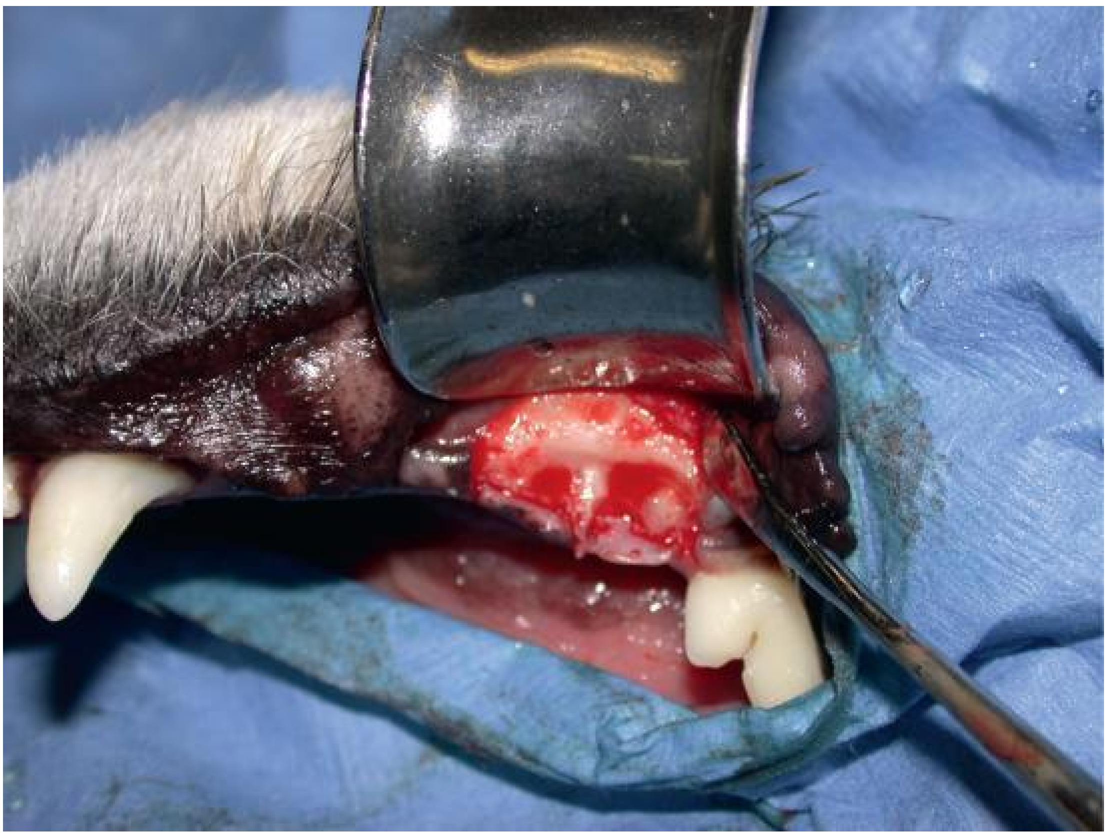
An envelope flap is a gingival flap (i.e., not extending apical to the mucogingival junction) created by making a sulcular incision and elevating some of the attached gingiva on the lingual and buccal aspects, with minimal extension interproximally, and no vertical releasing incisions (Fig. 13.17A). An extended envelope flap is useful for extraction of several adjacent teeth. When more exposure is required or a single tooth is being extracted, a triangle flap is useful. The triangle flap and pedicle flap are mucogingival flaps (i.e., extending apical to the mucogingival junction). Flaps used for extraction procedures are full-thickness flaps that also include the periosteum. A triangle flap is a mucogingival flap consisting of a sulcular incision and one vertical releasing incision, creating a three-cornered flap with corners at the distal extent of the sulcular incision, the coronal aspect of the vertical releasing incision (at the mesial end of the envelope incision) and the apical extent of the vertical releasing incision (Fig. 13.17B).[75] A pedicle flap is a sulcular incision with two vertical releasing incisions, creating a four-cornered flap (Fig. 13.17C); this flap provides the best exposure for removal of buccal alveolar bone for a challenging extraction or retrieval of a fractured root fragment.



• **FIG. 13.17** (**A**) Envelope flap: a single incision is made in the gingival sulcus. To excise pocket epithelium, an internal bevel incision can be made. (**B**) Triangle (three-cornered) flap: a sulcular incision is made, followed by a single vertical releasing incision. (**C**) Pedicle (four-cornered) flap: two vertical releasing incisions are created, providing maximum exposure of the alveolar bone.

Technique for developing a mucogingival flap

To provide proper exposure of the alveolar bone supporting the teeth to be extracted, gingiva, mucosa, and periosteum must be reflected. First, the gingiva is incised, using a # 15 blade on a scalpel handle held in a pen grasp, inserted into the gingival sulcus, and drawn with a continuous, smooth motion.[75] Excising the free gingiva of teeth affected by periodontitis and creating an internal bevel incision to eliminate the pocket epithelium will allow primary-intention healing of healthy gingiva and may allow more rapid bone regeneration in the alveolus.[73] Next, if a vertical releasing incision is to be made, the opposite hand is used to tense the alveolar mucosa and the blade is drawn, beginning at the mesiobuccal line angle of the tooth, in an apical and slightly mesial direction through the mucogingival junction and extending several millimeters into the alveolar mucosa. If desired, a second vertical releasing incision is made beginning at the distobuccal line angle of the tooth and extending apically and slightly distally. Alternatively, this incision may be made interproximally, or at the mesiobuccal line angle of the adjacent tooth. The scalpel blade will dull when pressed against bone, so multiple blades should be employed for patients with multiple extractions.[75] Incisions should extend apical to the mucogingival junction, so that the flap is as long as the root(s) of the tooth. Once the incisions are created, a periosteal elevator is introduced into the sulcus at the mesial aspect and may be turned laterally to pry the gingiva from the underlying bone. The periosteal elevator is then used with pushing and rotating strokes apically and distally to reflect the mucosa and periosteum from the bone (see Chapter 7).[75] After reflection of the flap, the periosteal elevator, a Seldin retractor, or a Cawood-Minnesota retractor (Fig. 13.18) may be used to prevent trauma to the flap during the remainder of the extraction.



• **FIG. 13.18** Use of a Cawood-Minnesota retractor to retract the lip and a periosteal elevator to retract the mucogingival flap during location of a fractured root.

Bone removal (alveolotomy or partial alveolectomy)

After reflection of the flap, removal of the buccal alveolar bone may be performed. Using a round diamond (or carbide) bur in fine, sweeping motions, the buccal alveolar bone is removed beginning at the alveolar margin and moving as far apically as desired. While minimal alveolectomy is required for teeth affected by periodontitis, removal of up to 75% of the buccal alveolar bone will facilitate extraction of teeth with little to no bone loss or ankylosed teeth.

Alveoloplasty

The sharp bone edges present after luxation and/or elevation will delay healing of the gingival flap and may lead to considerable postoperative discomfort. Alveoloplasty (shaping and smoothing of the margins of the alveolus after extractions) is performed with a round diamond bur on a high-speed handpiece, bone rongeurs, or a small file.

Management of the alveolus

Following alveoloplasty, the empty alveolus is cleared of debris.[72] In cases of advanced periodontitis, gentle curettage of the alveolus should be performed to remove pocket epithelium and any remnants of subgingival calculus. Overzealous curettage should be avoided, because disruption of the blood clot within the alveolus will delay healing.[73-77]

Placement of bone grafting materials in the alveolus is advocated by some practitioners as a way to prevent "collapse" of the alveolar margin following extraction of large teeth such as canine teeth and mandibular first molar teeth.[78,79] *Autologous grafts* (i.e., those collected directly from the patient) have historically been the "gold standard" bone replacement material, having osteogenic, osteoinductive, and osteoconductive properties.[80] Collection of a free "crescent graft" from the mandibular ramus has been proposed for immediate use in human extraction sites, with minimal postoperative morbidity.[81] This approach may be adapted for use in dogs (harvesting bone just caudal to the mandibular molars) but may be less suitable for cats, where collection of an adequate sample size may not be feasible.

When a graft is desired but an autologous graft is not practical, an *allograft* (typically freeze-dried or frozen cancellous and/or cortical bone) is an excellent option. Numerous clinical studies in human patients[82-86] and experimental studies in dogs[87,88] have shown production of new bone following placement of freeze-dried bone (FDB) allografts. Demineralization of the FDB exposes the collagen fibrils and associated components of bone matrix, including bone morphogenetic proteins; therefore, demineralized FDB is considered osteoinductive, while nondemineralized FDB is considered osteoconductive only.[89] However, the presence or bioavailability of bone morphogenetic proteins in currently available demineralized freeze-dried bone products remains unquantified. Both canine and feline FDB are commercially available in a mixture of demineralized bone matrix and cancellous chips <0.7 mm in diameter, specifically designed for oral and periodontal use (OsteoAllograft Perio Mix: Veterinary Transplant Services, Inc., Kent, WA).

Alloplasts are synthetic or naturally occurring inert materials that serve primarily to maintain space. As such, they are osteoconductive only. Bioactive ceramics, also called bioglass materials, generally contain oxides of calcium, sodium, phosphorous, and silicone, which reportedly stimulate collagen synthesis and new bone formation. Bioactive ceramics have a broad antimicrobial effect on microorganisms on teeth and implants.[90-92] One bioactive glass (Consil: Nutramax Laboratories, Inc., Edgewood, MD), available in both particulate and putty forms, is marketed specifically for veterinary use; it has been used in extraction site alveolar margin maintenance.[93] While there are no clinical studies in dogs, experimental evidence suggests that bioactive glass is less effective than demineralized FDB allograft in inducing new bone formation in intrabony pockets adjacent to implants in dogs.[88]

Recently, bone matrix impregnated with various growth factors has been proposed as an alternative to more traditional graft materials.[94]

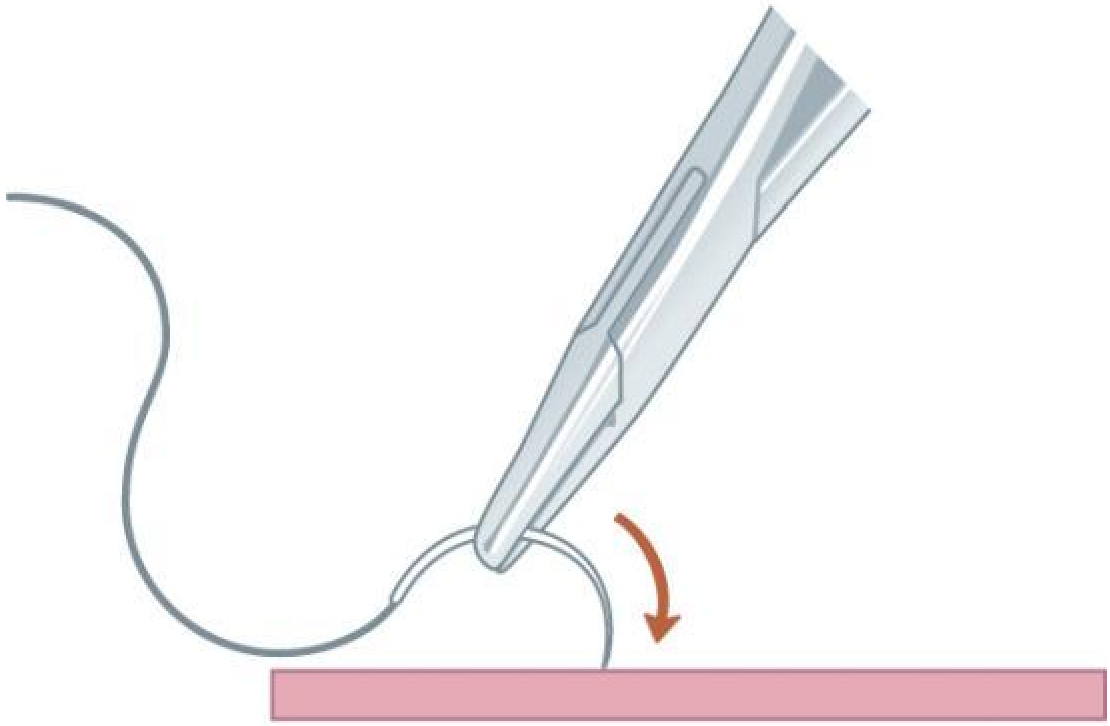
It is important to note that placement of any material in the alveolus may result in postoperative complications, and should be performed in selected cases only after appropriate discussion with clients.

Principles of suturing

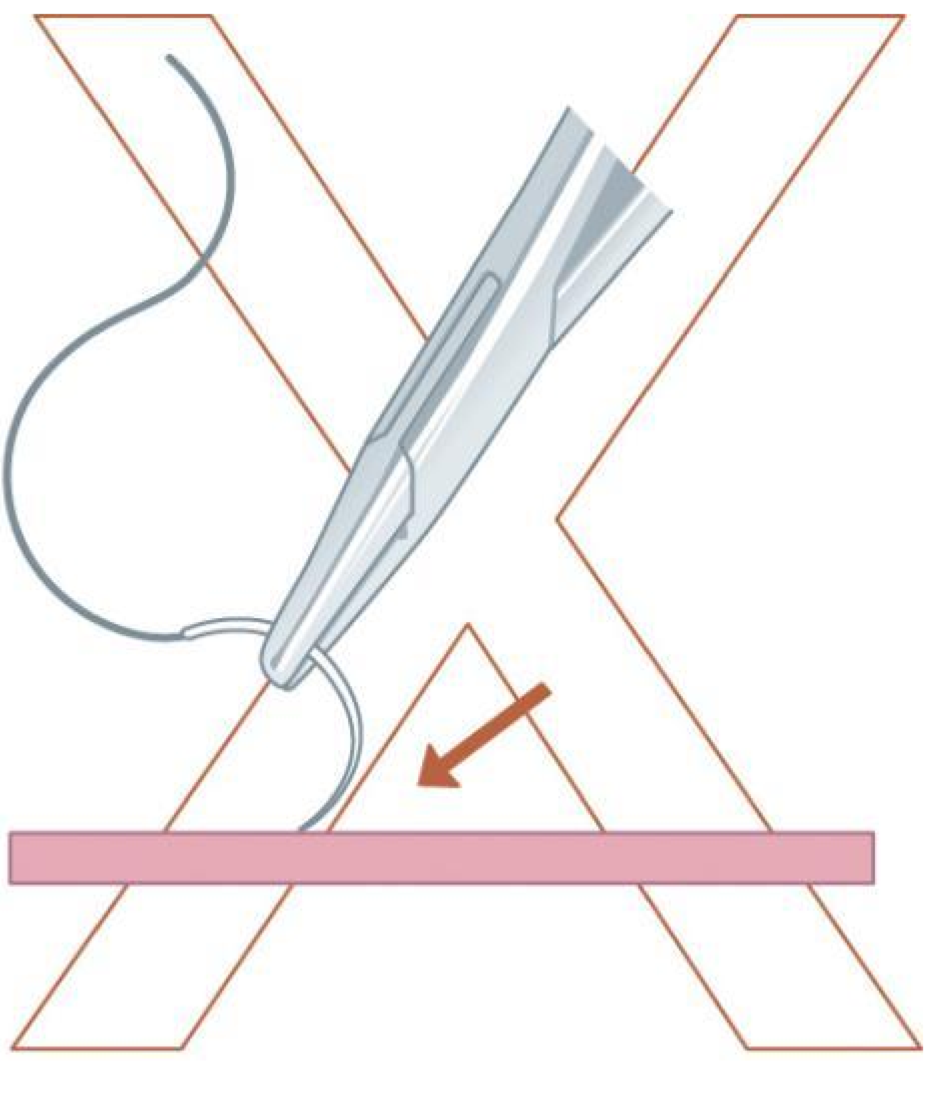
Following completion of the surgical procedure, the extraction sites should be gently lavaged with sterile saline and the flap edges should be apposed without tension. The sharper the incision and the less trauma the wound edges have sustained, the more likely the site is to heal by primary intention. Sutures not only hold the flap edges in place over the underlying bone, they also aid in maintenance of the blood clot, which plays an important role in healing.[75,95] If appropriate suture techniques are not employed, the flap may retract away from the bone or the clot may be dislodged, both of which will result in delayed healing of the extraction site.[75]

The armamentarium for extraction site closure includes a needle holder, tissue forceps, and suture material. A small needle holder, such as a 5-inch Mayo-Hegar or Halsey (see Fig. 7.11), is ideal for cats and small dogs. The needle holder should be held with the thumb and ring finger through the rings and the index finger extended along the length to provide stability and control.[75] The suture needle should be a small diameter, 3/8 to 1/2 circle with a reverse cutting edge, which facilitates passage through the mucoperiosteal and gingival tissue.[75] Selection of suture material is discussed in Chapter 8.

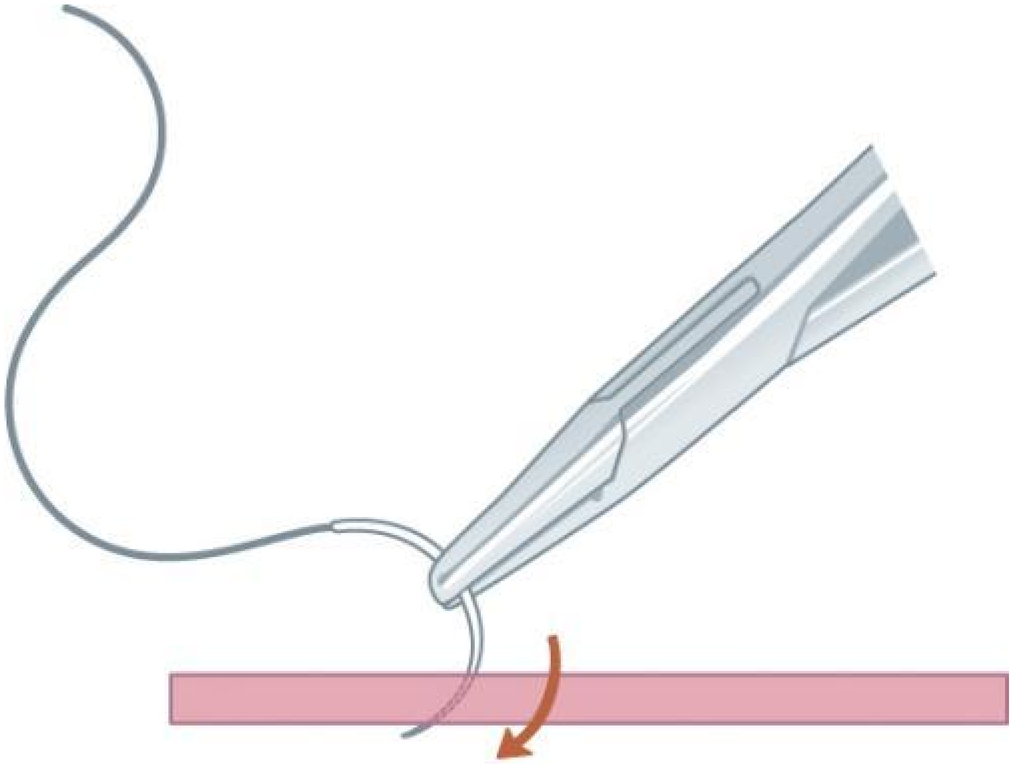
The needle should enter the mucosa at a right angle, to make the smallest possible hole in the tissue; if the needle passes through the mucosa obliquely, the suture will tear through the superficial layers of the flap when the knot is tied, which results in greater injury to the soft tissues.[75] Similarly, if an inadequate bite of tissue is taken, the suture may tear through the edge of the flap. There should be approximately 3 mm of tissue between the suture and the edge of the flap.[75] The needle holder should be turned rather than pushed so it passes easily through the tissue at right angles, minimizing trauma (Fig. 13.19).



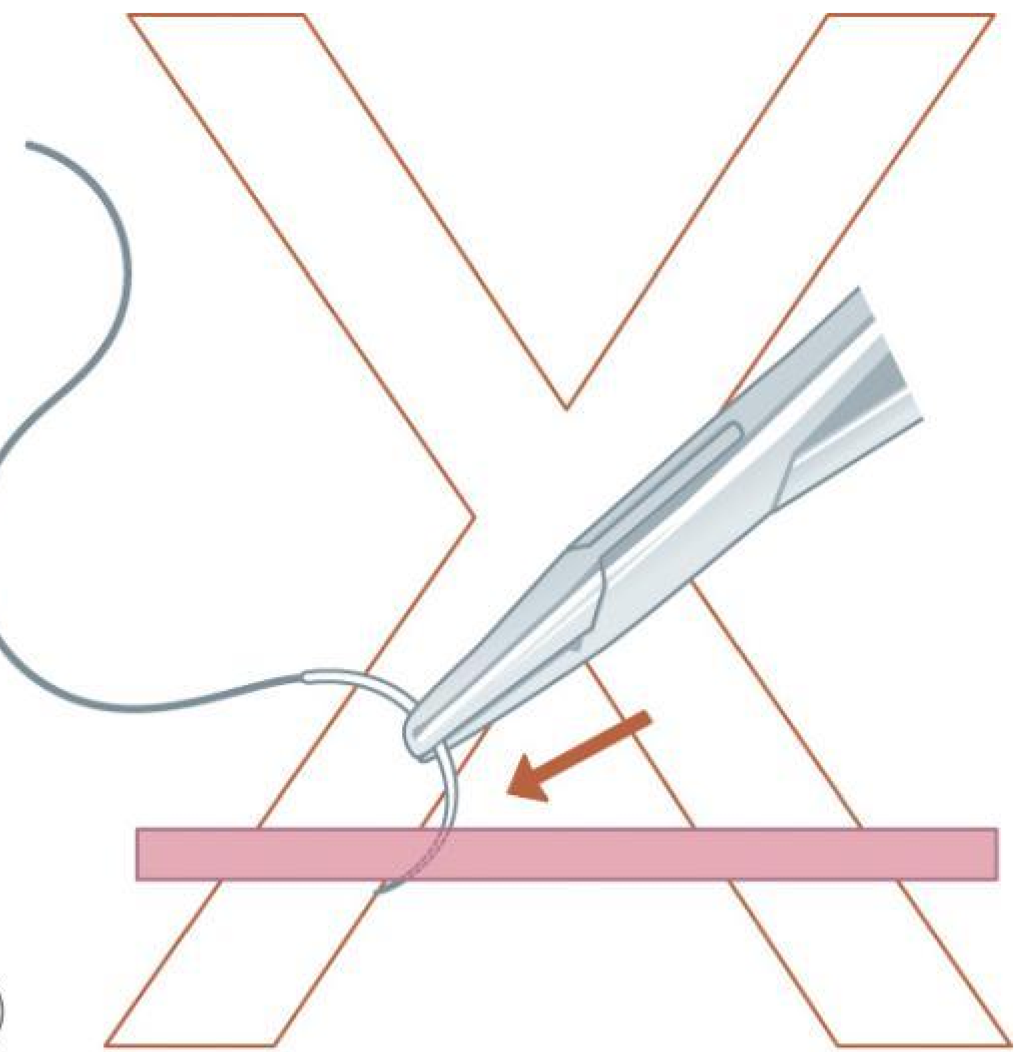
A



C



B



D

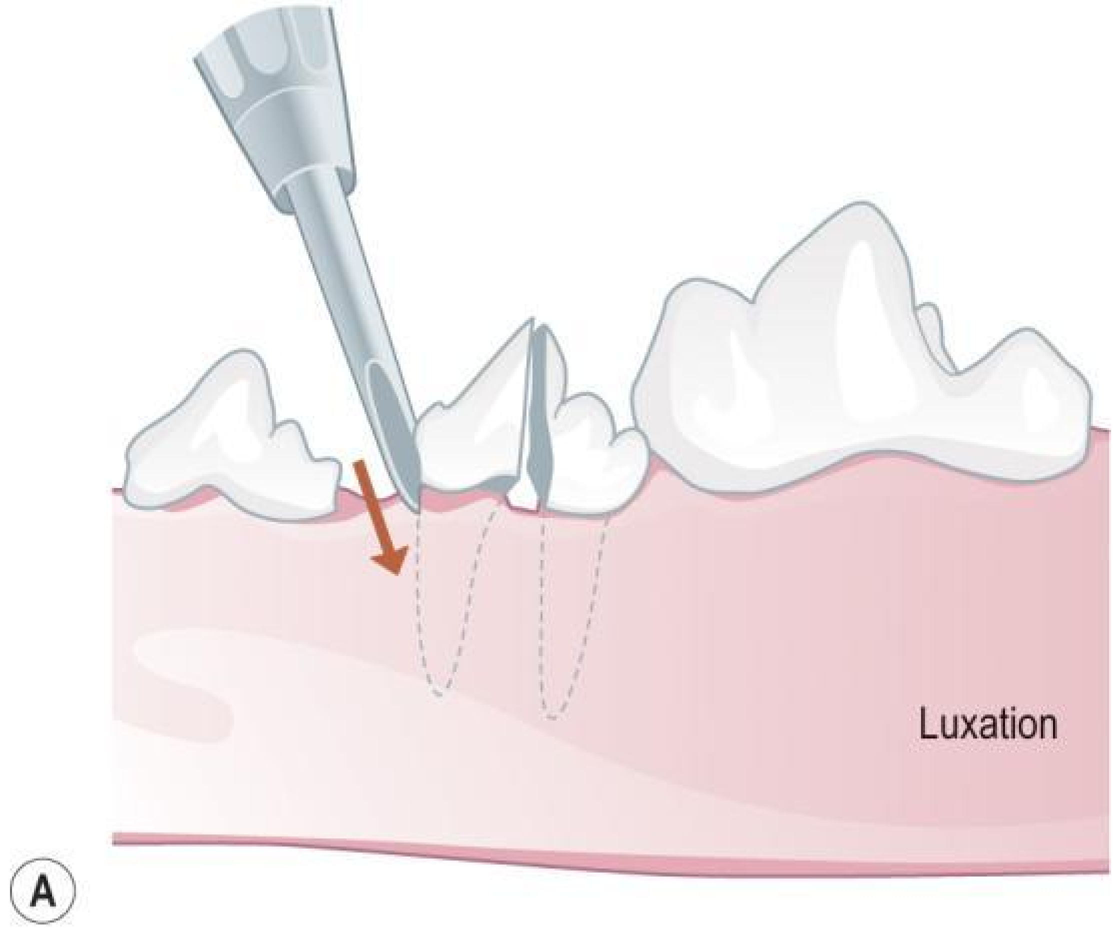
• **FIG. 13.19** (**A**) When passing through the gingiva or mucosa, the needle should enter the tissue at a 90-degree angle. (**B**) The needle holder should be rotated so the needle passes easily through the tissue. (**C**) If the needle enters the gingiva or mucosa at an acute angle and (**D**) is pushed (rather than rotated) through the tissue, tearing of the tissue is likely to occur.

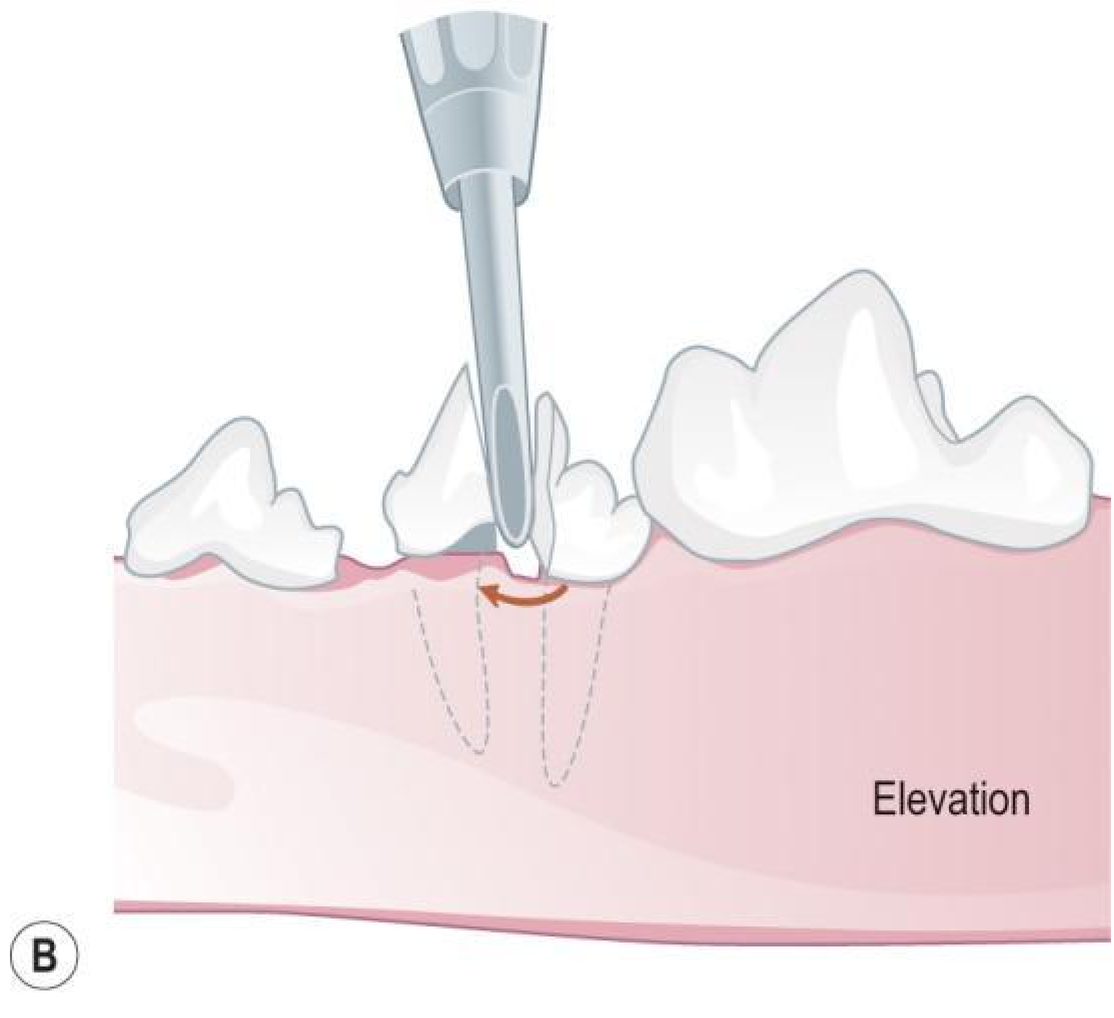
When replacing a triangle or pedicle flap, in general, it is helpful to suture the mucogingival junctions first, to restore the tissue to its presurgical location. The needle should be passed through the flap mucosa first, regrasped with the needle holder, then passed through the attached mucosa. If the two margins of the incision are close together, an experienced surgeon may be able to insert the needle through both sides of the incision in a single pass. However, this can result in tearing of the gingiva or mucosa, so two passes are recommended for most extraction sites.[75]

Once the sutures are passed through both the mobile flap and the immobile mucosa, they are tied with an instrument tie in a simple interrupted pattern, with approximately 2–3 mm between sutures. A simple continuous pattern may be employed for long incisions or in situations where multiple, plaque-retentive knots are undesirable, as in patients with plaque-reactive stomatitis.

Mechanical principles involved in tooth extraction

The removal of tooth roots from their alveoli employs the use of the following mechanical principles: the wedge principle, wheel-and-axle motion, leverage, rotation, and traction. Inserting a luxator into the periodontal ligament space acts as a wedge to expand the alveolar bone while severing the periodontal ligament fibers (Fig. 13.20A). Wheel-and-axle motion is employed when an elevator is inserted perpendicular to and between two roots (allowing the side of the elevator blade to contact a purchase point on one of the roots) and the handle is turned, acting as an axle, while the blade of the elevator acts as a wheel, engaging the root and lifting (elevating) it from the alveolus (Fig. 13.20B).[57] Levers employ a long lever arm and a short effector arm to transmit moderate forces into small movements against significant resistance.[57] Elevators may occasionally be used as levers, engaging the blade in a purchase point on the root and directing the handle downward to lift the tooth from its alveolus. Following luxation or elevation, the beaks of the extraction forceps are placed as apically as possible on the tooth, parallel to the long axis of the root. It is important not to move the forceps side-to-side to “rock” the tooth, as this will create shearing forces near the root apex and will lead to root fracture (Fig. 13.21A). Rather, the beaks of the extraction forceps are placed on the root of the tooth and the tooth is rotated gently to fatigue the remaining periodontal ligament fibers (Fig. 13.21B). Finally, after the periodontal ligament fibers have been fatigued or severed and significant mobility is achieved following luxation, elevation, and rotation, minimal tractional force is applied with extraction forceps to gently deliver a tooth from its alveolus.


Luxation
A


Elevation
B

• **FIG. 13.20** (**A**) Use of a straight luxator as a wedge to displace the root from the alveolar bone. (**B**) Use of an elevator as a wheel-and-axle machine.

• **FIG. 13.21** (**A**) Rotational forces are applied with the beaks of extraction forceps placed as far apically on the tooth as possible. (**B**) If the tooth is "rocked" or moved side-to-side rather than rotated, root fracture results.

Principles and techniques for tooth extraction

A surgical approach to extraction should not be reserved for extreme situations such as a periodontally sound, fractured tooth in an older dog, or considered a salvage procedure for a unsuccessful simple extraction. When correctly performed, a surgical extraction technique may be more conservative, cause less morbidity, and take less time than a simple extraction. Simple extractions requiring application of great force may result in damage to the adjacent soft tissues and removal of large amounts of associated bone, causing significantly more morbidity than a controlled surgical technique.[75]

Whether simple or surgical extraction is elected, the three fundamental requirements for a satisfactory procedure remain constant: (1) adequate visualization of the tooth to be extracted,

(2) an unimpeded pathway for the removal of the tooth, and (3) use of controlled force to luxate or elevate, and remove the tooth.[57] Proper patient positioning, good lighting, irrigation, and suction will improve visibility and make extraction less challenging. Similarly, appropriate sectioning of multirooted teeth and use of well-maintained instruments will allow easier achievement of the stated goals.[10]

For a tooth to be removed from its alveolus, the soft-tissue attachment to the crown must be disrupted, the alveolar bone must be expanded (or removed), and the periodontal ligament fibers must be severed or torn.[57] This is achieved by incising the gingival attachment, removing some alveolar bone with a bur when necessary, and introducing a luxator or elevator into the periodontal ligament space.

To prevent damage to adjacent tissues caused by slippage of the elevator or luxator, it is helpful to extend the index finger along the shaft. In addition, the operator's nondominant hand should be used to stabilize the jaw and the tooth being extracted (Fig. 13.22), relaying tactile information about the tooth's mobility as luxation or elevation proceeds, which helps indicate readiness for placement of extraction forceps and delivery of the tooth from its alveolus.[57] If all of the periodontal ligament fibers are severed, extraction forceps may not even be required to remove the tooth.[10] Techniques for extraction of specific teeth are discussed in detail in subsequent chapters.

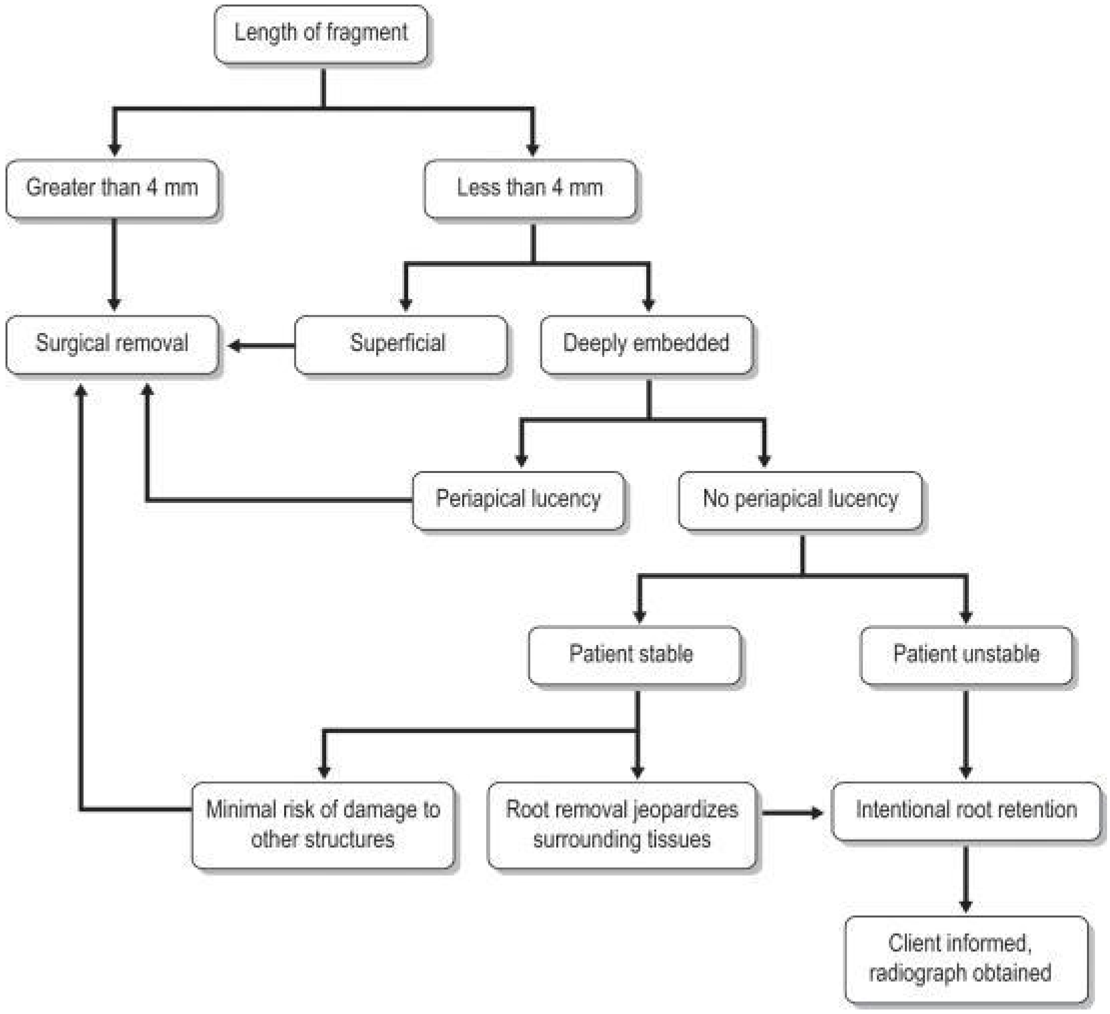
• **FIG. 13.22** Use of the nondominant hand to stabilize the tooth during extraction. This provides tactile feedback regarding mobility of the tooth and readiness for application of extraction forceps.

Removal of root fragments

If a fracture occurs in the coronal half of a root, the remaining fragment should be visible without additional bone removal, and use of a small (1- or 2-mm) luxator will usually allow delivery of the remaining root. However, if the fracture occurs in the apical third of the root, enlargement of the mucoperiosteal flap and removal of additional buccal bone will most likely be required for successful retrieval of the fragment. This is particularly true for dilacerated roots, or bulbous roots with hypercementosis.[75] Excellent light and suction will be required for proper visualization and successful removal of a fractured root fragment. Once the fragment is visualized, a root tip pick or 1-mm luxator is inserted into the periodontal ligament space around the root until the fragment is mobilized and can be teased from the alveolus. When using the luxator or root tip pick, it is important not to apply excessive apical force, which could displace the fragment into other anatomic locations such as the nasal cavity or mandibular canal.[75] A technique has also been described in which a thin, tapered diamond bur is used to create space around the root, allowing introduction of an instrument for removal of the root fragment.[96]

Intentional root retention

Except in cases of end-stage resorption in which the roots have been almost entirely replaced by bone-like material, the goal of any extraction should always be to extract the entire tooth. However, when a root tip has fractured and when attempts to remove it have been unsuccessful, the surgeon may consider leaving the root in place. In some situations, the risks of additional surgery to remove a small root fragment may outweigh the benefits (Fig. 13.23).



• **FIG. 13.23** Flowchart for decision-making for fractured roots.

Three conditions must be met for a tooth root to be left in the alveolus[75]: First, the root fragment must be small, no more than 3–4 mm in length. Second, the root must be deeply embedded in bone and not superficial, to prevent any subsequent bone resorption from exposing the tooth root. Third, the tooth involved must not be infected, and there must be no periapical radiolucency. If these three conditions exist, and if the risk of further surgery is considered to be greater than the benefit, the surgeon may consider leaving the fragment.

The risk of further surgery is considered greater than the benefit in the following situations: (1) removal of the root will require significant destruction of adjacent tissue or extreme removal of surrounding bone; (2) removal of the root jeopardizes vital structures, such as the inferior alveolar nerve; and (3) the patient is not stable under anesthesia, and prolongation of the procedure could be life-threatening.

If the decision has been made to intentionally leave a root fragment in the alveolus, a strict protocol must be observed.[75] First, radiographic documentation of the root tip's presence and position must be obtained and recorded in the patient's record. Second, the client must be informed that, in the surgeon's judgment, leaving the root in place will do less harm to the patient than further surgery. The client should also be instructed to contact the veterinarian immediately if problems develop in the area of the retained root, such as swelling or a draining tract. Finally, the patient should be re-radiographed at regular intervals in the future to determine whether evidence of inflammation is present around the root or to document absence of such evidence.

Intentional root retention for teeth with end-stage resorption is discussed further in Chapter 17.

Postoperative care and assessment

Pain management

Whenever extractions are anticipated, preemptive analgesia should be attained. This may be accomplished by the use of opioids, nonsteroidal antiinflammatory agents, and/or alpha-2 agonists as premedicants, and local anesthetics delivered prior to beginning the extractions. Postoperative analgesia may include nonsteroidal antiinflammatory drugs, nonnarcotic opioids such as tramadol, or a combination of the two, which may provide better antiinflammatory activity than either agent alone.[97] Pain management is discussed in more detail in Chapter 4.

Nutritional support

Following surgical extraction of multirooted teeth, soft food should be offered for 5–7 days. In addition, chew toys and hard treats should be avoided for 7–10 days to prevent disruption of the sutures. A feeding tube may be considered for patients undergoing full-mouth or near full-mouth extractions, but this is rarely necessary.

Oral hygiene

Keeping the teeth and oral cavity clean following extraction results in more rapid healing of surgical wounds.[98] Oral rinses or gels containing known antiseptics such as chlorhexidine gluconate may be used twice daily following meals for the first postoperative week and may result in more rapid healing in veterinary patients with compromised oral hygiene. By the third or fourth day after surgery, it is acceptable for clients to resume gentle brushing of their dog's teeth, avoiding the areas immediately adjacent to the extraction sites until the end of the first postoperative week.

Postoperative assessment

The client should be instructed to observe the patient for lethargy, inappetence, or halitosis, and the patient should be examined 1–2 weeks following the procedure to evaluate healing. For patients who have not had home care previously performed, this postoperative visit provides an opportunity to instruct the client in tooth-brushing techniques and to discuss other home care measures.

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CHAPTER 14

Simple extraction of single-rooted teeth

Milinda J. Lommer, Frank J. M. Verstraete

Definitions

Simple extraction: An extraction not requiring a gingival incision (other than within the sulcus) or sectioning of the tooth. Also called a closed, uncomplicated, or nonsurgical extraction.

Surgical extraction: An extraction that requires a gingival incision, bone removal, and/or sectioning of the tooth. Also referred to as an open or complicated extraction.

Elevation: The process by which the periodontal ligament is fatigued or torn and alveolar bone is expanded to facilitate removal of the tooth from the alveolus. Using an elevator as a lever, the tooth is lifted (elevated) from its alveolus.

Luxation: The process by which the periodontal ligament is cut or severed to loosen teeth from the surrounding alveolar bone.

Preoperative concerns

General health status

For patients with preexisting medical conditions, special precautions may be required to prevent infection, minimize hemorrhage, and prevent exacerbation of the patient's disease state. As discussed in Chapter 3, patients on immunosuppressive drugs or with an immune disorder should receive intravenous antibiotics at the time of the procedure and oral antibiotics afterward. Gentle tissue handling is especially important in diabetic patients, who may experience delayed wound healing, whether extractions are simple or surgical. Although simple extraction of single-rooted teeth is relatively atraumatic, patients with severe thrombocytopenia or clotting factor deficiencies should receive blood or plasma transfusions prior to or during the procedure, as even a simple extraction can lead to excessive hemorrhage in these patients.

Periodontal status

Prior to initiating exodontic treatment, the mobility of the tooth to be extracted should be evaluated. If a tooth is being extracted due to fracture or some cause other than periodontal disease, and is periodontally healthy, normal mobility is expected, and considerable resistance to extraction may be encountered. Severe periodontal disease, conversely, typically results in greater-than-normal mobility and a relatively easy tooth extraction. Periodontal disease of individual incisor teeth is relatively uncommon; it is more common to see horizontal bone loss affecting all or most incisor teeth, particularly mandibular incisor teeth.[1,2] Toy breeds may have mobile mandibular incisor teeth with horizontal bone loss but relatively healthy gingiva.

If a tooth has less-than-normal mobility or radiographs suggest ankylosis or root resorption, a simple approach to extraction is likely to result in root fracture, and a surgical approach should be considered. This is typically the case for severely abraded incisor teeth with pulp exposure in aggressively chewing dogs.

Extraction of incisor teeth may significantly change the appearance of the patient, and clients should be informed of this prior to the procedure.

It is occasionally necessary to extract a small tooth of lesser functional significance to preserve a more important, adjacent tooth. For example, extraction of a periodontally compromised mandibular third molar tooth will allow better access to the second molar tooth, improving the success of home care.

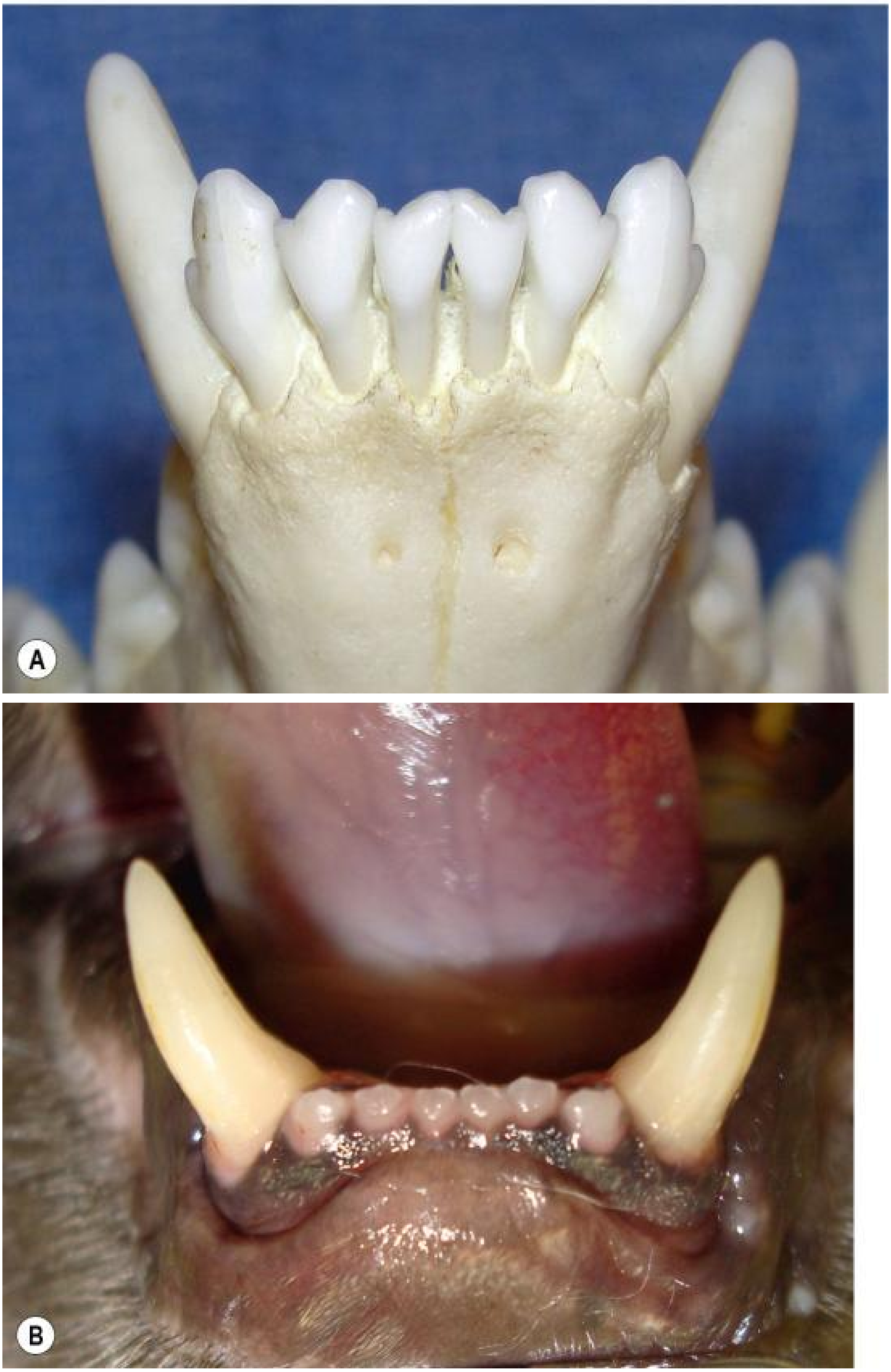
Surgical anatomy

All teeth in dogs and cats are structurally similar, consisting of a pulp cavity surrounded by dentin, which is covered by cementum (on the root) or enamel (on the crown). The crown and root meet at the cementoenamel junction, also known as the "neck" of the tooth. The pulp cavity communicates with the tissues in the periapical region via an apical delta. The tooth-supporting structures include cementum, alveolar bone, gingiva, and periodontal ligament. The periodontal ligament consists primarily of Sharpey fibers (type I collagen), which insert into cementum and bone to secure the tooth in the alveolus.

Permanent teeth

Incisor teeth

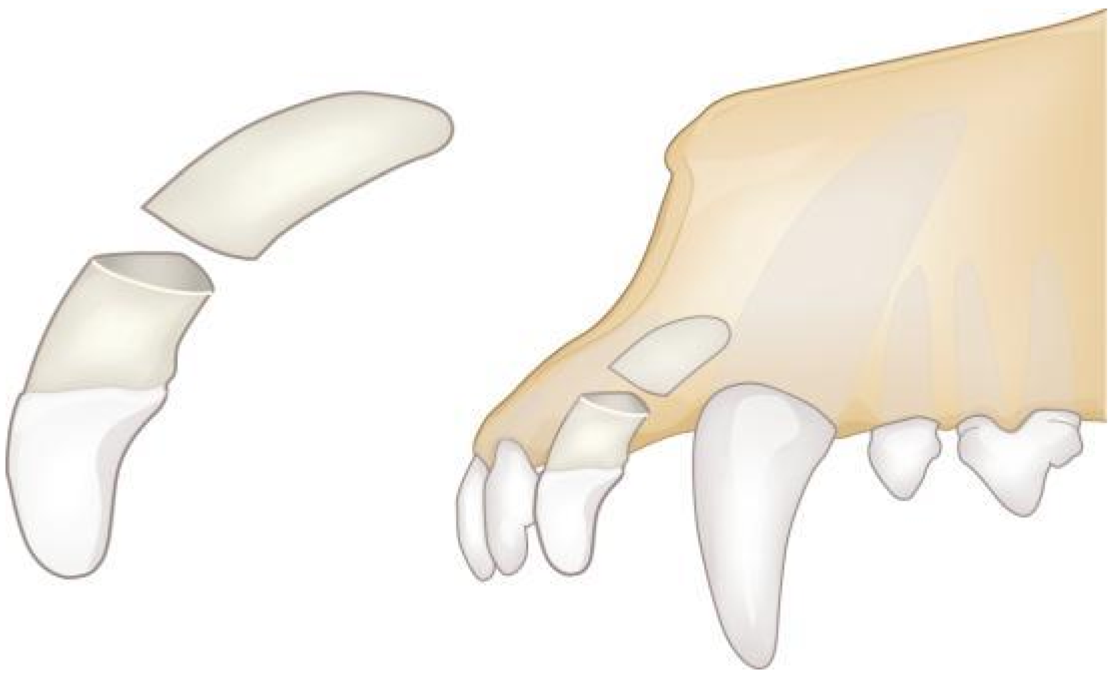
Although upper incisor teeth are rooted in the incisive rather than maxillary bones, the term "maxillary incisor teeth" will be used to differentiate these teeth from the mandibular incisor teeth. Located in the rostral portion of the mouth between the canine teeth, incisors are typically slender, slightly curved, and laterally compressed, so that they are slightly flattened mesiodistally. This is most prominent in the mandibular incisor teeth of the dog (Fig. 14.1A), which are often in very close proximity to one another, with only a thin section of alveolar bone between them. Maxillary and mandibular incisor teeth in the cat are also closely juxtaposed (Fig. 14.1B). It may be difficult to extract one incisor tooth without injuring the adjacent teeth.



• **FIG. 14.1** (**A**) Mandibular incisor teeth in the skull of a dog. (**B**) Mandibular incisor teeth in the cat are in close proximity to one another.

Because the incisor teeth appear small compared with other teeth, it is often assumed that extraction will be uncomplicated. However, the crown:root ratio of incisor teeth in the dog is approximately 1:3; the roots are considerably longer than might be expected. This can make extraction more difficult than originally anticipated, particularly if the teeth are being extracted for reasons other than periodontal disease.

The root of the maxillary third incisor tooth in the dog (Fig. 14.2) has a triangular cross-section, is usually quite curved, and may be of substantial length, often making it the most challenging of the single-rooted teeth to extract (except for canine teeth). The maxillary incisor teeth are separated from the nasal cavity by a relatively thin plate of the incisive bone, and it is possible to penetrate into the nasal cavity during attempts to extract these teeth. In the dog and cat, the incisor crowns have visible cusps, and on the palatal surface of each maxillary incisor tooth is a ridge known as the cingulum, upon which the mandibular incisor teeth rest.[3]



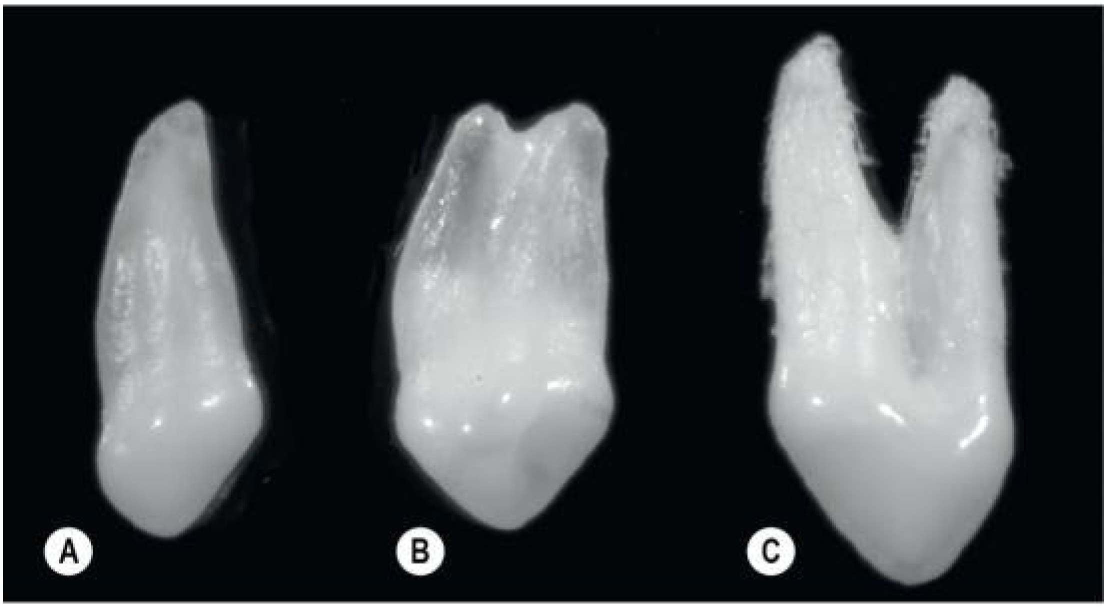
• **FIG. 14.2** Maxillary third incisor tooth in the dog. Note the triangular cross-section and curved root.

First premolar teeth in the dog

These teeth are in close proximity to the canine teeth. They have a pointed crown and a short, conical, tapered root. The mandibular first premolar teeth are often embedded and may give rise to dentigerous cysts (see Fig. 13.4).

Maxillary second premolar teeth in the cat

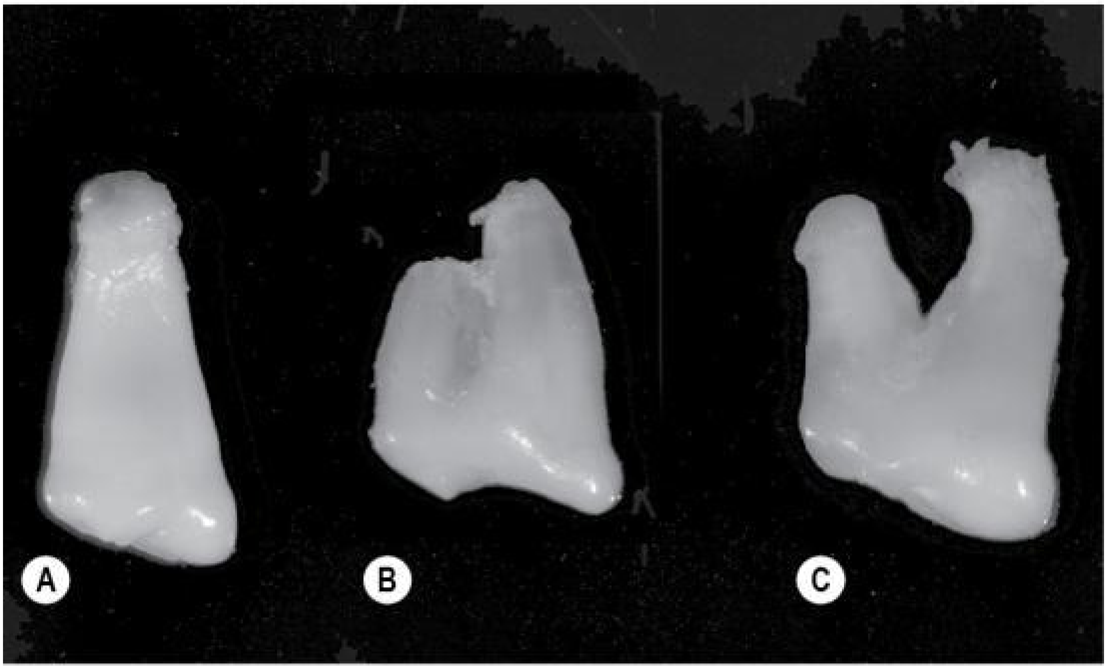
Similar to the first premolar teeth in the dog, a cat's maxillary second premolar tooth has a pointed crown and a short, conical, tapered root. Anatomical variations in the root structure are common; in one study, the frequencies of a single root, a dichotomous root, and two roots were found to be 27.7%, 55.1%, and 9.2%, respectively (Fig. 14.3).[4]



• **FIG. 14.3** Anatomical variations in the root structure of the maxillary second premolar tooth in the cat: (**A**) single root, (**B**) dichotomous root, (**C**) two roots. Source: (From Verstraete FJM, Terpak CH. Anatomical variations in the dentition of the domestic cat. *J Vet Dent.* 1997;14:137–40.)

Maxillary first molar teeth in the cat

Although often classified as two rooted, the maxillary first molar tooth of the cat may also have a single root or two fused roots. In one study, this tooth was found to have a single root in 35.0% of cases and dichotomous roots in 34.7%; only 28.0% of molar teeth had two fully formed roots (Fig. 14.4).[4] Regardless of the conformation, they are typically extracted in a simple fashion without sectioning. The maxillary first molar tooth, rooted in the maxillary bone, is located immediately ventral to the orbit; care must be taken not to penetrate through the bone into the orbit during extraction.



• **FIG. 14.4** Anatomical variations in the root structure of the maxillary first molar tooth in the cat: (**A**) single root, (**B**) dichotomous root, (**C**) two roots. Source: (From Verstraete FJM, Terpak CH. Anatomical variations in the dentition of the domestic cat. *J Vet Dent.* 1997;14:137–140.)

Mandibular third molar teeth in the dog

These teeth have a flattened crown and a short, conical, tapered root.

Deciduous teeth

Deciduous incisor and canine teeth are smaller and more slender than their permanent counterparts; the roots are typically long and narrow, with a wide pulp cavity and very thin dentin walls. The molar teeth and first premolar teeth do not have deciduous precursors.

Therapeutic decision-making

Simple versus surgical extraction

Preoperative radiographs are essential to provide the surgeon with accurate information about the tooth to be extracted and its root(s), adjacent teeth, and surrounding tissues. Even a single-rooted tooth may have a dilaceration (abrupt curve or hook at the root apex), which might complicate a simple extraction. The presence of other anatomical variations, such as dichotomous roots (commonly affecting incisor teeth in dogs), may also affect the decision to perform a simple extraction. External root resorption and ankylosis, common in dogs with severely abraded incisor teeth, are indications for a surgical extraction technique; a wide mucogingival flap exposing the alveolar bone over all six teeth facilitates extraction. (Extensive root resorption, commonly seen with feline tooth resorption, is discussed in Chapter 17.) Previous root canal treatment may result in a more brittle tooth root, and ankylosis may be present; therefore, surgical extraction may be indicated for endodontically treated teeth.[5] Because of their short, conical roots, first premolar and mandibular third molar teeth in the dog rarely require surgical extraction. Conversely, the root of the maxillary third incisor tooth in the dog has a triangular cross-section and is often dramatically curved, making this tooth more suitable for surgical extraction in many cases.

Suturing

Although a simple extraction wound may be left unsutured, placing one to two sutures to approximate the gingival wound edges and secure the blood clot present in the vacated alveolus will allow more rapid healing, by first rather than second intention. In addition, the expanded alveolar bone plates and overlying soft tissues can be approximated by digital compression. The alveolus should be debrided only if necessary. Debris, calculus, tooth, or bone fragments in the alveolus should be removed with gentle curettage or suction. If a periapical lesion is visible on preoperative radiographs but there was no granuloma attached to the apex upon tooth removal, the alveolus should be gently curetted to remove the granuloma or cyst. In the absence of debris or periapical lesions, the alveolus should not be curetted, as the remnants of the periodontal ligament and the bleeding bony walls are in optimum condition to promote rapid healing.[5]

If multiple teeth were extracted and if the gingiva can be apposed without tension, several absorbable sutures should be placed in a simple interrupted pattern. Wound closure is also indicated when the gingiva is inadvertently lacerated. Also, for patients in whom delayed wound healing might be expected or a hemostatic disorder might be present, even a single simple extraction site should be sutured. Suturing may prevent accumulation of debris in the alveolus and may therefore facilitate healing. However, oral soft tissues heal rapidly, and many suture materials remain in the mouth long after healing has occurred. This may be a source of discomfort or irritation to the patient. To avoid this, the smallest-sized suture material should be selected, which will be appropriate for the location. In veterinary dentistry, sizes 4-0 and 5-0 are sufficient to maintain apposition and are less likely to cause irritation to the patient than size 3-0 or larger. Size 6-0 is a good choice for cats and dogs weighing less than 3 kg. Suture material selection is discussed in Chapter 8.

Simple extraction technique

Prior to attempting extraction, calculus should be removed from the tooth to be extracted and adjacent teeth. This allows better adaptation of the instruments to the tooth, and prevents contamination of the alveolus with calculus after the extraction. The mouth should be rinsed with 0.05%–0.12% chlorhexidine gluconate to decrease oral bacterial contamination and reduce the incidence of postoperative infection.[5] Mask, gloves, and eye protection should be worn by the surgeon to prevent injury. When using the elevator, luxator or extraction forceps, the nondominant hand should be used to stabilize the jaw.

Elevation technique

Elevators are used mainly as levers.[5] There are three types of leverage typically employed. A first-class lever involves a fulcrum (typically alveolar bone) between the resistance (tooth) and the applied force (elevator). An elevator or luxator inserted into the periodontal ligament space parallel to the long axis of the root acts as a wedge lever (see Fig. 13.13A). Finally, wheel-and-axle levers are employed when an elevator, after gaining purchase on the tooth surface and positioned perpendicular to the long axis of the root, is rotated such that the force on the tooth is directed outward from the alveolus (see Fig. 13.13B).[6]

There are two primary techniques for using an elevator to extract a single-rooted tooth in a simple fashion. The gingival attachment to the tooth should be severed with a scalpel blade prior to using the elevator. The first technique involves placement of the elevator blade into the periodontal space, parallel to the long axis of the tooth, with the concave surface of the blade against the root. Gentle rotational forces are employed to push the root away from the elevator, tearing the periodontal ligament while expanding the alveolar bone slightly. The rotational movements should be slow and steady; the operator may hold the elevator in the rotated position for several seconds to fatigue the periodontal ligament. In the second technique, the elevator blade is placed at the level of the alveolar margin, perpendicular to the long axis of the tooth, and rotated with the concave surface against the tooth. This is performed with gradually increasing pressure, in several locations around the circumference of the tooth, until the periodontal ligament tears, loosening the root and allowing the tooth to be lifted from the alveolus.[7] Care should be taken not to wedge the elevator against adjacent teeth, as they may be displaced or fractured by the forces applied.

Luxation technique

A luxator is made of softer steel than an elevator and is not designed to bear rotational forces; it is used as a wedge rather than a first-class or wheel-and-axle lever. Therefore, in a simple extraction, the luxator is always used parallel to the root surface of the tooth. After incising the gingival attachment, the blade of the luxator is inserted into the periodontal ligament space and advanced apically, severing the collagen fibers and acting as a wedge (Fig. 14.5A–B). The luxator is repositioned in several locations around the circumference of the tooth, always parallel to the root surface, until the periodontal ligament has been disrupted sufficiently to allow removal of the tooth with extraction forceps. The mesial and distal root surfaces provide the best sites for luxator placement. It is best to avoid positioning the luxator on the buccal or palatal aspect of the tooth, as slippage and subsequent soft tissue damage occur more easily. The luxator should be held with the handle in the palm of the hand and the tip of index finger extended along the shaft, with the fingertip held close to the blade (Fig. 14.5C). This will help prevent soft tissue trauma in the event the luxator accidentally slips over the alveolar bone rather than along the periodontal ligament space.[8]

• **FIG. 14.5** Simple extraction procedure of a maxillary first incisor tooth with the patient in dorsal recumbency: following a sulcular incision (**A**), the luxator is inserted into periodontal space on the distal aspect of the tooth and directed apically (**B**). The luxator is then inserted into the periodontal space on the mesial aspect of the tooth (**C**). The index finger is always extended along the shaft of the luxator, both to protect the patient from injury if slippage occurs and to provide the operator with tactile sense of the tooth's mobility. The luxation process is repeated until significant mobility is achieved, at which time the forceps is placed as far apically as possible and gentle rotational forces are applied for delivery of the tooth (**D**).

When using luxators and elevators, the operator's nondominant hand should be used to retract soft tissues, help stabilize the jaw, and support the alveolar process. In addition to helping prevent iatrogenic trauma to the patient, presence of the nondominant hand provides tactile information to the operator regarding the expansion of the alveolar process during luxation and elevation.[5]

Use of forceps

After the tooth has been loosened with an elevator or luxator, the forceps are placed on the root (not the crown), with the beaks parallel to the long axis of the tooth (Fig. 14.5D). The forceps are held at the end of the handles, to maximize control as well as mechanical advantage. Excessive force on the handles should be avoided, as it may result in crushing of the tooth. The beaks should be placed as far apically as possible, which displaces the center of rotational forces toward the apex of the tooth, decreasing the likelihood of root fracture. The beaks may also act as wedges to expand the alveolar bone slightly.[5] The tooth should not be "rocked" from side to side, as root fracture is likely (see Fig. 13.21A). Instead, gentle rotational forces should be used to disrupt any remaining attachment. The tooth is first rotated clockwise and held for a few seconds in the rotated position, fatiguing and tearing the remaining periodontal ligament fibers (see Fig. 13.21B). The procedure is then repeated in a counterclockwise direction. Tractional forces should not be used until the final portion of the extraction procedure and should not be excessive. The tooth should be delivered, rather than pulled, from the alveolus. This is easily achieved if the periodontal ligament fibers have been completely severed.[7]

Specific teeth

Incisor teeth of the cat and mandibular incisor teeth of the dog

Because the mandibular incisor teeth have flattened mesial and distal surfaces, these surfaces are more suited for placement of an elevator or luxator than the buccal or lingual surfaces. In cats and most dogs, the mandibular incisor teeth are in very close proximity to one another and the likelihood of injury to an adjacent tooth by an elevator is high. Use of the luxation technique may be more appropriate than the elevation technique, because a luxator's thin blade is less likely to contact adjacent teeth than the curved blade of an elevator, and luxators are not used with rotational forces. For cats, use of a 1.3-mm "luxating elevator" (Zoll Dental, Niles, IL) is ideal for extraction of incisor teeth. Alternatively, the small blade of a root tip pick or apical elevator may be used instead of a luxator, taking care not to apply pressure in any direction other than apically.

After incising the gingival soft tissue attachment, the blade of the luxator is inserted into the periodontal space, parallel to the root surface. Gentle pressure is applied to the handle to force the blade apically in the periodontal space, severing the periodontal ligament fibers and expanding the alveolar bone slightly. The index finger of the dominant hand should be placed as close to the end of the blade as possible, to stabilize the blade and prevent slippage and deeper penetration than intended (Fig. 13.2). The nondominant hand should be used to stabilize the tooth and to provide tactile sense of the expanding alveolar bone and increasing mobility of the tooth. The blade is removed and placed in a new location and the process is repeated. This is done on the mesial and distal surfaces until the alveolar bone has expanded enough to allow placement of the extraction forceps on the root surface. In the dog, the extraction forceps may then be used to apply slight buccal and lingual pressure to expand the buccal and lingual alveolar processes. Gentle rotational forces may also be used prior to the final stage, in which minimal tractional forces are employed in an occlusal direction to deliver the tooth from the alveolus. The soft tissues and alveolar processes should be manually compressed to their original location. In the cat, use of the extraction forceps for anything other than very gentle tractional force may result in root fracture and should therefore be avoided.

Maxillary first and second incisor teeth of the dog

While not as pronounced as the mandibular incisors, the maxillary first and second incisor teeth do have somewhat flattened mesial and distal surfaces and therefore may be approached in the same manner as the mandibular incisor teeth. They are often not as closely crowded as the mandibular incisor teeth, so either the elevation or luxation technique may be employed. If the elevation technique is selected, care must be taken not to use the adjacent incisors for leverage when applying rotational pressure. After incising the gingival attachment to the tooth, the elevator or luxator is placed in the periodontal space on either the mesial or distal surface of the tooth, with the blade parallel to the root surface. The blade is directed apically, and if the elevation technique is being used, rotational forces may be applied to further disrupt the periodontal ligament. Once the tooth is mobile, the extraction forceps are applied, with the beaks parallel to the long axis of the root. Slight buccal and lingual pressure may be applied with the forceps. If the roots have no sharp curvatures, rotational forces may be employed as well. Finally, gentle traction is applied in a buccal-occlusal direction to complete the extraction process. The soft tissues and alveolar bone should then be manually compressed.

Maxillary third incisor teeth of the dog

The roots of these teeth have a triangular cross-section, with the mesial-palatal and distal-palatal surfaces being slightly more flattened than the buccal surface, and are typically longer than the roots of the first and second incisor teeth. These factors reduce the effectiveness of rotational forces, which may result in root fracture. Because a luxator or elevator may slip off the buccal surface and cause injury to the soft tissues, the mesial-palatal and distal-palatal surfaces provide the best sites for introduction of a luxator or elevator into the periodontal space. In this fashion, an elevator or luxator may be directed apically to sever the periodontal ligament and slightly expand the alveolar bone. An elevator may also be used perpendicular to the long axis of the tooth, provided the adjacent teeth are not being used for leverage, to engage the cementum and lift the tooth from the alveolus. After luxation or elevation, extraction forceps are placed as far apically as possible, with the beaks parallel to the long axis of the tooth. Buccal pressure may be applied to expand the alveolar bone on that surface. Traction is then applied in a buccal-occlusal direction to remove the tooth from the alveolus, and the soft tissues and alveolar bone are returned to their original positions by manual compression.

Maxillary first premolar and mandibular third molar teeth of the dog and maxillary second premolar teeth of the cat

These teeth have short, conical roots and are therefore most amenable to rotational forces. After incising the gingiva, a 2-mm luxator (JS Dental Manufacturing, Inc., Ridgefield, CT) or 1.3-mm “luxating elevator” (Zoll Dental, Niles, IL) may be introduced into the periodontal space, with the blade parallel to the root surface. The blade may be placed several times around the circumference of the tooth and directed apically to sever the collagen fibers of the periodontal ligament. The extraction forceps are placed with the beaks parallel to the long axis of the tooth and as far apically as possible. Rotational forces may be applied to further disrupt the periodontal ligament prior to application of traction to deliver the tooth.

Maxillary first molar teeth of the cat

Although these teeth may have two divergent or fused roots (Fig. 14.4), they may be extracted in the same manner as a single-rooted tooth. They are typically flat on the mesial and distal surfaces, where a luxator may be introduced parallel to the long axis of the tooth. Care must be taken not to penetrate too deeply, as the orbit is immediately dorsal to the alveolus of the first molar tooth in most cats. After luxation, extraction forceps are used to apply traction in an occlusal direction for completion of the extraction process. Alternatively, an elevator may be placed perpendicular to the long axis of the tooth between the fourth premolar and the first molar teeth and gentle leverage applied to lift the first molar tooth from its alveolus.

Deciduous teeth

In some instances, when the normal root resorption process has taken place, deciduous teeth may be easier to extract than permanent teeth. However, if it is necessary to remove deciduous teeth before substantial root resorption has occurred, extraction must be approached cautiously. The roots of deciduous teeth are very long and delicate and fracture easily. After incising the soft tissue attachment at the gingival margin, a luxator with a narrow (2-mm) blade should be employed to gently penetrate the periodontal ligament space and sever the Sharpey fibers. Alternatively, special, curved elevators with narrow blades have been developed (Fahrenkrug elevator: Shipp's Dental and Specialty Products, Inc., Marana, AZ) for use with curved-rooted teeth such as canines. With either a luxator or an elevator, the tip must always be directed toward the root surface of the deciduous tooth.[6] Because damage to the permanent tooth may occur, leverage against the permanent tooth should not be used, and the elevator or luxator should not be allowed to penetrate deeply on the lingual surface of the deciduous tooth (in the direction of the permanent tooth). Rotational movements should be minimal, to avoid fracturing the fragile roots. Finally, tractional forces should not be applied until substantial mobility has been achieved, and traction should be very gentle. Surgical extraction of deciduous canine teeth is addressed in Chapter 15.

Postextraction care of the alveolus

If severe periodontitis is present, rinsing the alveolus with a 0.05%–0.12% chlorhexidine gluconate solution is advised to reduce bacterial contamination and delayed healing. The alveolus should be debrided if there is obvious debris such as calculus, bone fragments, or tooth fragments in the alveolus or if a periapical granuloma, abscess, or cyst was present. If there was no periapical lesion and there is no debris, curetting the alveolus may cause injury and delay healing. The blood clot should not be disrupted, as it will provide the best conditions for rapid healing.[5] Any expanded alveolar bony plates should be compressed and repositioned to their original position. Suturing may or may not be performed.

Postoperative care and assessment

Use of oral rinses

Rinsing with an antibacterial solution is not necessary following routine extraction of a small number of single-rooted teeth in an otherwise healthy patient. In patients with medical conditions where infection or delayed healing might be anticipated, use of a 0.05%–0.12% chlorhexidine gluconate solution once or twice daily for 3–5 days following the procedure should be considered.

Dietary change

Most patients will prefer a soft food diet for 2–4 days following dental extractions. Commercial canned food is readily available and is a good choice for cats. For dogs normally on a dry food diet, changing to canned food may cause gastrointestinal upset, so it is generally recommended to soak the dry food in warm water prior to feeding in order to soften its consistency.

Recheck examination

A postoperative visit is not required following uncomplicated simple extractions. However, recheck examination provides an opportunity to discuss future oral health recommendations and is an ideal time to instruct the client on oral home care techniques.

Anticipating delayed wound healing

In patients with underlying medical conditions resulting in immune deficiencies, or where severe periodontitis is present preoperatively, extraction sites may not heal as rapidly as in a healthy patient. In these situations, use of broad-spectrum systemic antibiotic and antimicrobial oral rinses, together with a soft food diet for 7–10 days, should be considered. The client should be instructed to observe the patient for lethargy, inappetence, or halitosis, and the patient should ideally be assessed at 5-day intervals following the procedure to evaluate healing.

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CHAPTER 15

Extraction of canine teeth in dogs

Anson J. Tsugawa, Milinda J. Lommer, Frank J.M. Verstraete

Indications for extraction

The canine teeth are the longest teeth in the dog, with large roots nearly three times as long as their crowns.[1] For the dog, not only are they an important functional component of mastication, but structurally, they occupy a large portion of the jaws. As a result of their structural and functional importance, extraction should not be taken lightly or performed without careful consideration of alternative treatments. Iatrogenic mandibular jaw fracture and oronasal fistula are the most serious complications associated with extraction of these teeth; more minor complications include dehiscence, glossoptosis, and maxillary lip entrapment.

Extraction should be performed for medical reasons only. A procedure commonly referred to as “disarming,” in which the canine teeth of an aggressive animal are extracted with the goal of minimizing trauma to humans or other animals, is not an acceptable indication for extraction. Surgical intervention should not be viewed as an alternative to behavioral modification and medical management by a trained animal behaviorist.[2]

Perhaps the most common indication for extraction of canine teeth is severe periodontitis, where the severity of alveolar bone loss has resulted in a mobile tooth or oronasal fistula formation.

Teeth with complicated crown fractures and discolored intact teeth are likely to have nonvital pulp[3] and are candidates for extraction if root canal treatment is not elected. Deep crown–root fractures extending apical to the alveolar margin that are not manageable by advanced periodontal surgery are also an indication for extraction. Traumatically luxated, subluxated, or intruded teeth that are not amenable to splinting or orthodontic extrusion, respectively, are candidates for extraction as well.

Embedded or impacted canine teeth associated with odontogenic cysts should be extracted in the process of enucleating the cyst and its lining.

In puppies with mandibular brachygnathism (class 2 malocclusion), extraction of deciduous canine teeth may be indicated prior to eruption of the permanent canine teeth, particularly if the deciduous mandibular canine teeth are located distal to the deciduous maxillary canine teeth when the mouth is closed, causing a “dental interlock” and preventing independent growth of the maxilla and mandible.[4] Extraction of deciduous teeth to prevent malocclusion of the permanent dentition is referred to as “interceptive orthodontics.”[4,5]

Once the permanent teeth begin to erupt, the deciduous predecessors should exfoliate. Failure of a deciduous tooth to exfoliate, resulting in a persistent deciduous tooth, may cause malocclusion of the permanent teeth and often results in food entrapment, with subsequent periodontal disease.[6] Therefore extraction of persistent deciduous teeth should be performed as soon as the condition is diagnosed.[6]

In certain dental and skeletal malocclusions causing trauma to soft and hard tissues, extraction of the permanent mandibular canine teeth is a therapeutic option for treating occlusal trauma resulting from mandibular drift following mandibulectomy or for addressing persistent malocclusion following jaw fracture repair.

In reference to jaw fractures with canine teeth in the fracture line, unless severely compromised such that they would interfere with healing, the canine teeth should be preserved, as they are a common anchorage surface for many maxillofacial fracture repair techniques.

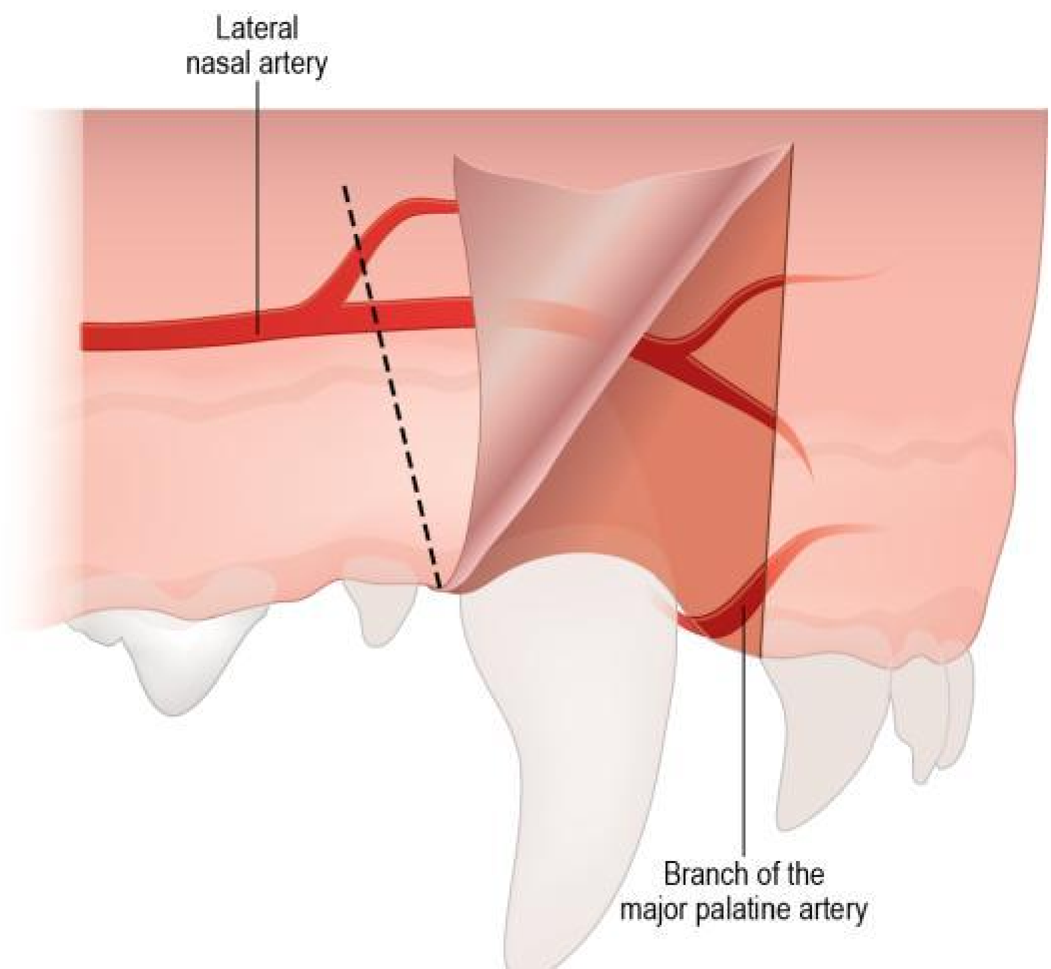
The indications for extraction of canine teeth are numerous and are not limited to the aforementioned list. Due to the structural and functional importance of the canine teeth in the dog, the clinician is reminded to consider alternatives to extractions whenever possible.

Surgical anatomy

Functionally, the canine teeth *(dentes canini)* in the dog are designed for piercing/holding prey and as slashing/tearing weapons in fighting. Comparatively, the canine (cuspid) teeth in humans serve a similar function, for tearing and crushing food, and are also the longest teeth in the human mouth. The canine teeth have roots measuring upwards of 50–60 mm in large-breed dogs.[7] The roots of the canine teeth curve caudally so the apices extend as far as the second premolar teeth.[8] They are pointed at their coronal and apical ends and have a wider midportion at the level of the cemento-enamel junction.[1] In cross-section, canine teeth are linguobuccally compressed and oval in shape.[1]

Maxillary canine tooth

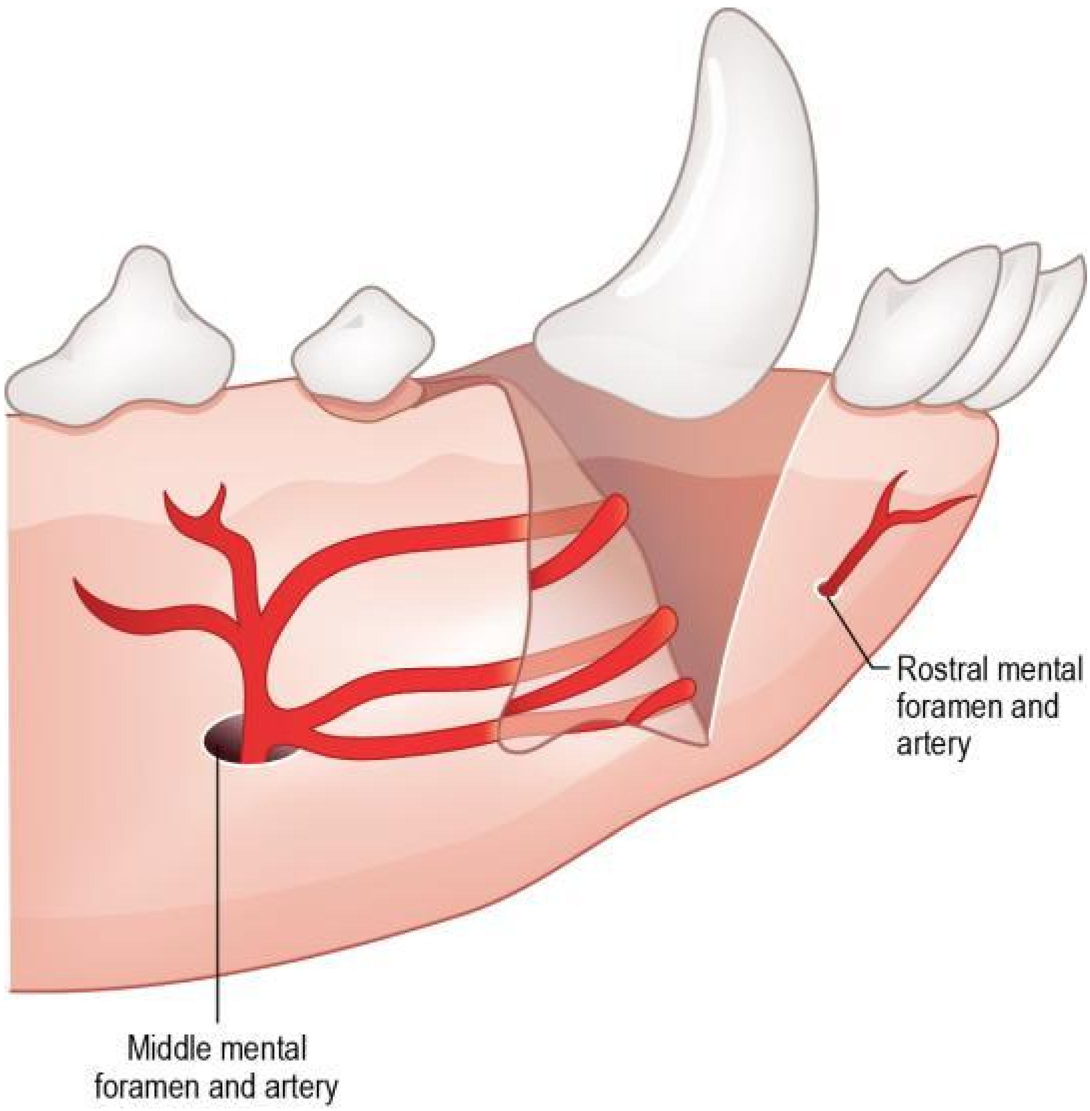
Since the widest section of the canine tooth is usually within the alveolus, a surgical approach to extraction including a full-thickness mucoperiosteal flap is often necessary. Avoidance of local vital structures in the flap design is encouraged, and in the region of the maxillary canine tooth, two structures of importance should be mentioned as sources of hemorrhage if transected: the lateral nasal branches of the infraorbital artery and its anastomosis with the branches of the major palatine artery that runs between the maxillary canine and third incisor teeth. The former is typically transected when a vertical releasing incision is created. In general, if a full-thickness flap design is employed, blunt reflection of the flap down to bone usually carries the majority of large vessels within the reflected flap. If a vertical releasing incision is created mesial to the maxillary canine tooth, it is placed at a level 2–3 mm mesial to the mesial line angle of the tooth to allow the incision line to rest over solid bone upon closure of the flap (see Chapter 13). In the process of creating this interproximal incision, it is likely that a small branch of the major palatine artery that passes through the interdental space between the canine tooth and the third incisor will be transected (Fig. 15.1). Hemorrhage may be brisk but is also well controlled with tamponade and when the flap is sutured closed. Often, the attached gingiva at the mesial corner of the flap, where the palpable jugum slopes down into the interproximal space between the maxillary third incisor and canine teeth, is toughest to pry away; a combination of sharp dissection, making sure the incision is complete, and use of the sharp end of the periosteal elevator facilitates this initial reflection. Once the flap has been reflected to the desired extent to provide the necessary visualization and access to the area, the prominent jugum overlying the maxillary canine tooth root will be clearly visible and accessible for alveolectomy.



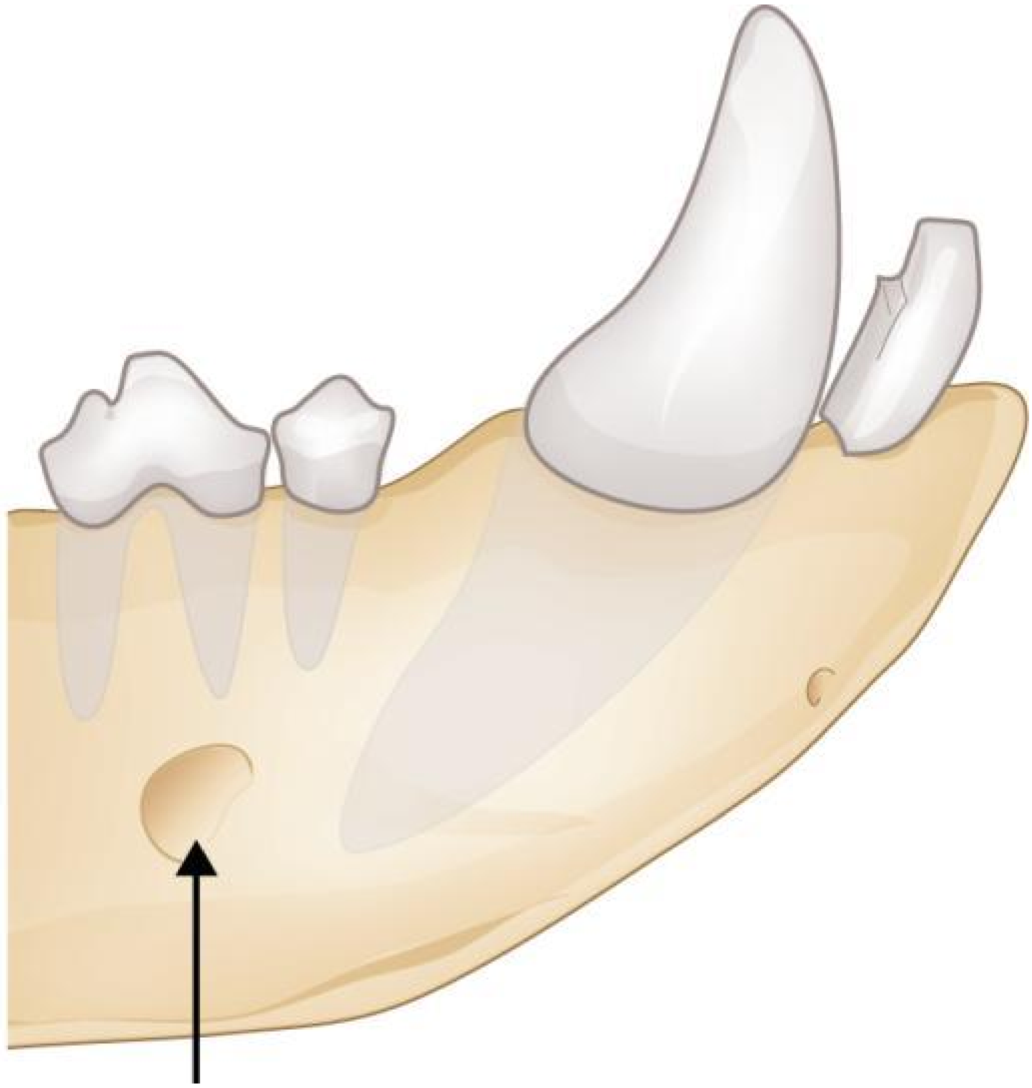
• **FIG. 15.1** Potential sources of hemorrhage encountered during development of a mucoperiosteal pedicle flap for surgical extraction of a maxillary canine tooth: lateral nasal artery; major palatine artery; and location for a distal vertical releasing incision *(dotted line)*.

Mandibular canine tooth

As mentioned above, appropriately planned flaps should avoid local vital structures, and for the mandible, the mental neurovascular structures that exit from the middle mental foramen are readily avoidable with an appropriately planned vertical releasing incision on the mesial aspect (Fig. 15.2). The interproximal space between the mandibular canine and third incisor teeth is minimal compared with the maxilla, and the incision for the vertical release must be carefully placed at line angles to avoid tension upon apposition of the flap, flap dehiscence, and damage to the gingiva of the third incisor tooth. The mandibular canine root is also located lingual and ventral to the mandibular first and second premolar tooth roots, and aggressive alveolectomy or extraction technique may risk damage to these teeth (Fig. 15.3). Contrary to extractions performed elsewhere on the mandible, there is little risk of laceration or damage to the inferior alveolar artery at the level of the mandibular canine alveolus. Anatomically, the root of the mandibular canine tooth does occupy a significant portion of the rostral mandible; conservative alveolectomy is recommended for extraction to avoid excessive weakening of the mandible, symphyseal separation, or worse, fracture of the mandible through the lingual wall of the alveolus.



• FIG. 15.2 The mental neurovascular structures exiting from the middle mental foramen are avoided with a vertical releasing incision on the mesial aspect of the canine tooth.



• **FIG. 15.3** Topographical anatomy of the canine first premolar and second premolar teeth showing the proximity of mandibular canine root to the middle mental foramen *(arrow)*, first premolar tooth, and mesial root of the second premolar tooth.

Deciduous canine teeth

The roots of deciduous canine teeth are long and slender and lie close to the developing crown of the permanent tooth.[5] The deciduous maxillary canine teeth are located distal to their permanent successors, while the deciduous mandibular canines are typically buccal to the permanent canines.

Therapeutic decision-making

Simple or surgical extraction technique

Canine teeth have long and curved roots that are generally difficult to extract by simple (closed, nonsurgical) extraction technique. Unless a canine tooth is severely mobile, surgical (open) technique is indicated. The sequelae of forcibly extracting a canine tooth are numerous and include jaw fracture and oronasal communication. Complications such as iatrogenic mandibular fracture typically occur when nonsurgical exodontic techniques are used when severe periodontal disease is apparent.[9] For deciduous canine teeth, whose roots are slender and may not be entirely resorbed, a surgical technique may be the safest approach to avoid damage to the developing permanent tooth. Even severely mobile canine teeth, which can be delivered with gloved fingers or by simple extraction, must be addressed with surgical flap closure, especially the maxillary canine teeth, where oronasal communication is a concern.

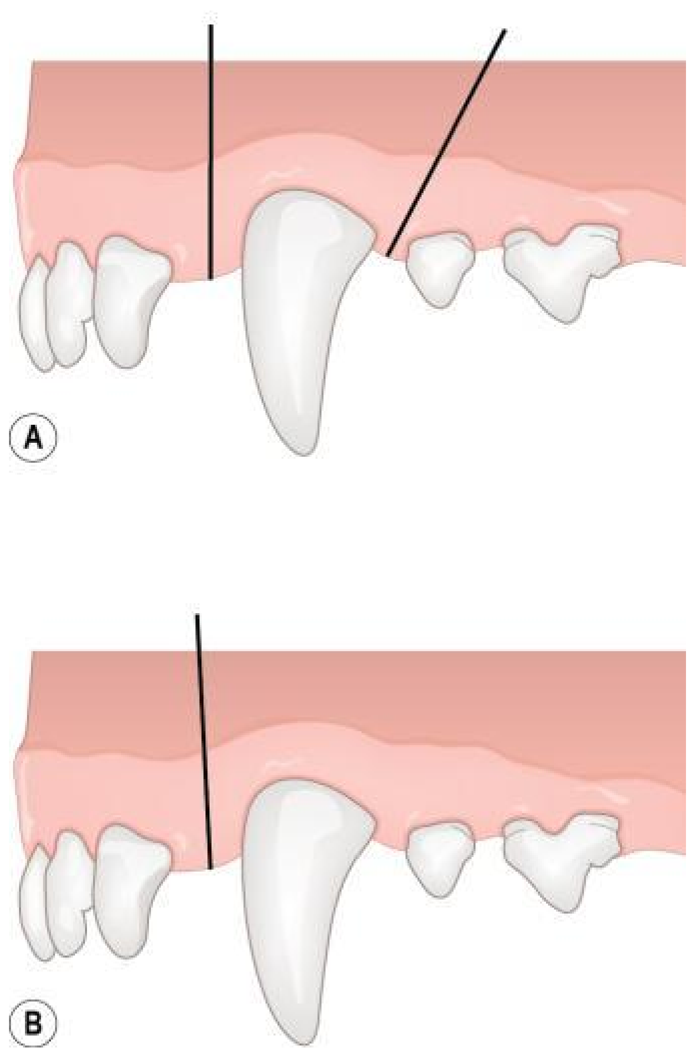
Selection of flap design

Unless there is significant gingival recession and/or axial mobility associated with a canine tooth, an envelope flap will probably not provide the exposure and access necessary for the atraumatic extraction of a canine tooth. Therefore the use of at least one or two vertical releasing incisions, creating three-cornered (triangle) and four-cornered (pedicle) mucoperiosteal flaps, respectively, is recommended.

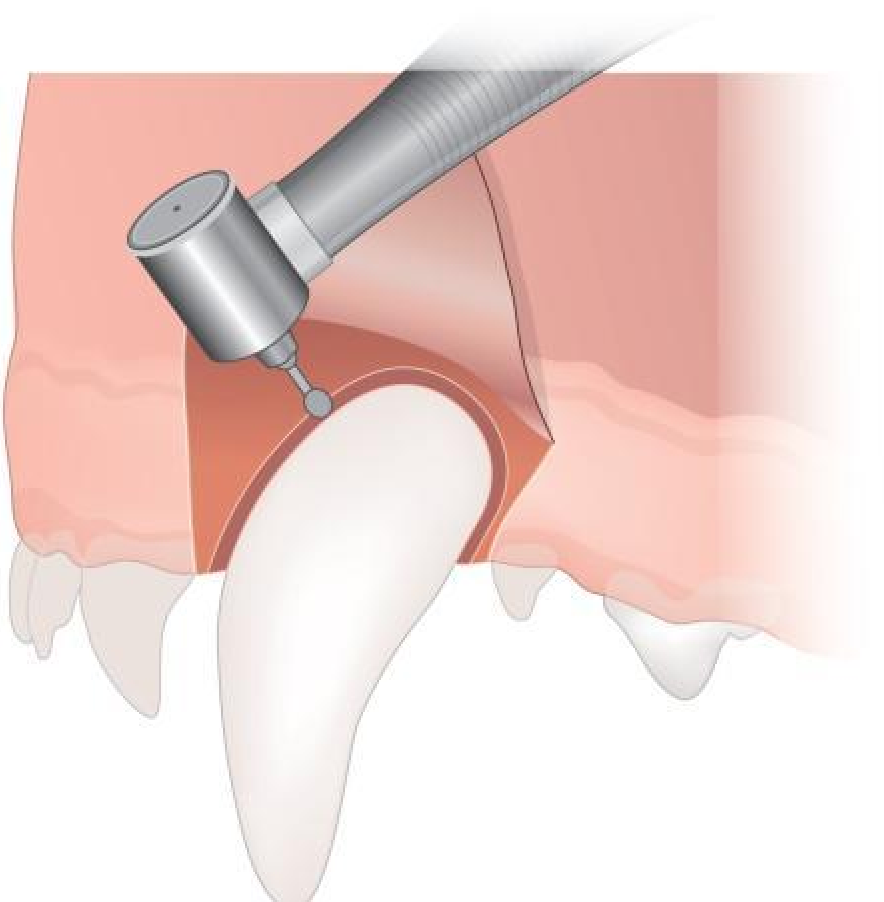
Maxillary canine tooth

The epithelial attachment to the maxillary canine tooth is severed by introducing a #15 scalpel blade down the gingival sulcus and along the entire circumference of the tooth. As an alternative to the sulcular incision, approximately 1 mm of the free gingival margin can be excised before the flap is elevated.[10] In cases where the gingiva is severely inflamed, closure will be simplified by removing the most friable edges of gingiva at the time of the initial incision. The sulcular incision is extended interproximally for 2–3 mm on the mesial and distal aspects. On the buccal side of the tooth, with tension applied to the alveolar mucosa so that the incision can be made cleanly through it, two divergent incisions are made in the gingiva extending into the alveolar mucosa (Fig. 15.4A). The flap should be of sufficient width to allow for the suture line to rest over solid bone and not over the vacated alveolus. Since the root of the maxillary canine tooth arcs caudally as far as the maxillary second premolar tooth, some clinicians advocate placement of the distal vertical releasing incision at the distal line angle of the maxillary second premolar tooth.[11] Alternatively, a single vertical releasing incision can be made, either mesial or distal to the canine tooth, creating a three-cornered or triangle flap (Fig. 15.4B). If an envelope flap is converted into a three-cornered flap, it is preferable to place the vertical releasing incision at the mesial end of the envelope flap.[12] The mucoperiosteal pedicle flap is reflected using blunt dissection with a periosteal elevator. The sharp end of the periosteal elevator is introduced under the free gingival end of the flap and is used to separate the attached gingiva from the underlying bone. Once the free edge of the flap has been raised, the broad end of the periosteal elevator can be used to reflect the flap to the desired amount. A #4 or #6 round bur mounted on a high-speed handpiece is commonly used to perform the buccal alveolectomy, although a bone chisel or rongeurs may also be used. Continuous irrigation should be used when performing the alveolectomy. Regarding the amount of buccal bone that needs to be removed to facilitate extraction, only one-third to one-half of the root length with a width approximately the same as the mesiodistal dimensions of the tooth is usually all that is necessary.[12] Some clinicians advocate the creation of small grooves in the margins of the tooth or channels in the bone along the mesial and distal margins of the root with a round diamond bur to facilitate placement of the elevator or luxator between the periodontal ligament and bone (Fig. 15.5).[7,13,14] This allows the dental elevator or luxator to be inserted more deeply into the periodontal ligament space.

During alveolectomy, a Seldin retractor (see Fig. 7.9A) or Minnesota retractor (see Fig. 7.9B) with pressure applied perpendicular to bone can be used to hold the flap in place without pulling on the soft tissues and inadvertently tearing the flap.[15] Luxation should be started on the mesial and distal aspects of the tooth, gradually moving in an apical direction. Once some progress has been made, the luxator can be introduced on the mesiopalatal aspect. At this point, it is tempting to use the luxator as an elevator, wedging the instrument between the palatal alveolar bone and tooth while applying a significant tipping force to luxate the tooth in a buccal direction. If not done with great care, however, this creates the risk of inadvertently pushing the apex of the tooth into the nasal cavity. Therefore if the whole root cannot be loosened at this stage, additional alveolar bone should be removed. Once the tooth is sufficiently mobile, such that the tooth can almost be delivered with gloved fingers, the extraction forceps can be used in a steady, alternating rotational and hold movement, to tear the final periodontal ligament fibers and deliver the tooth.

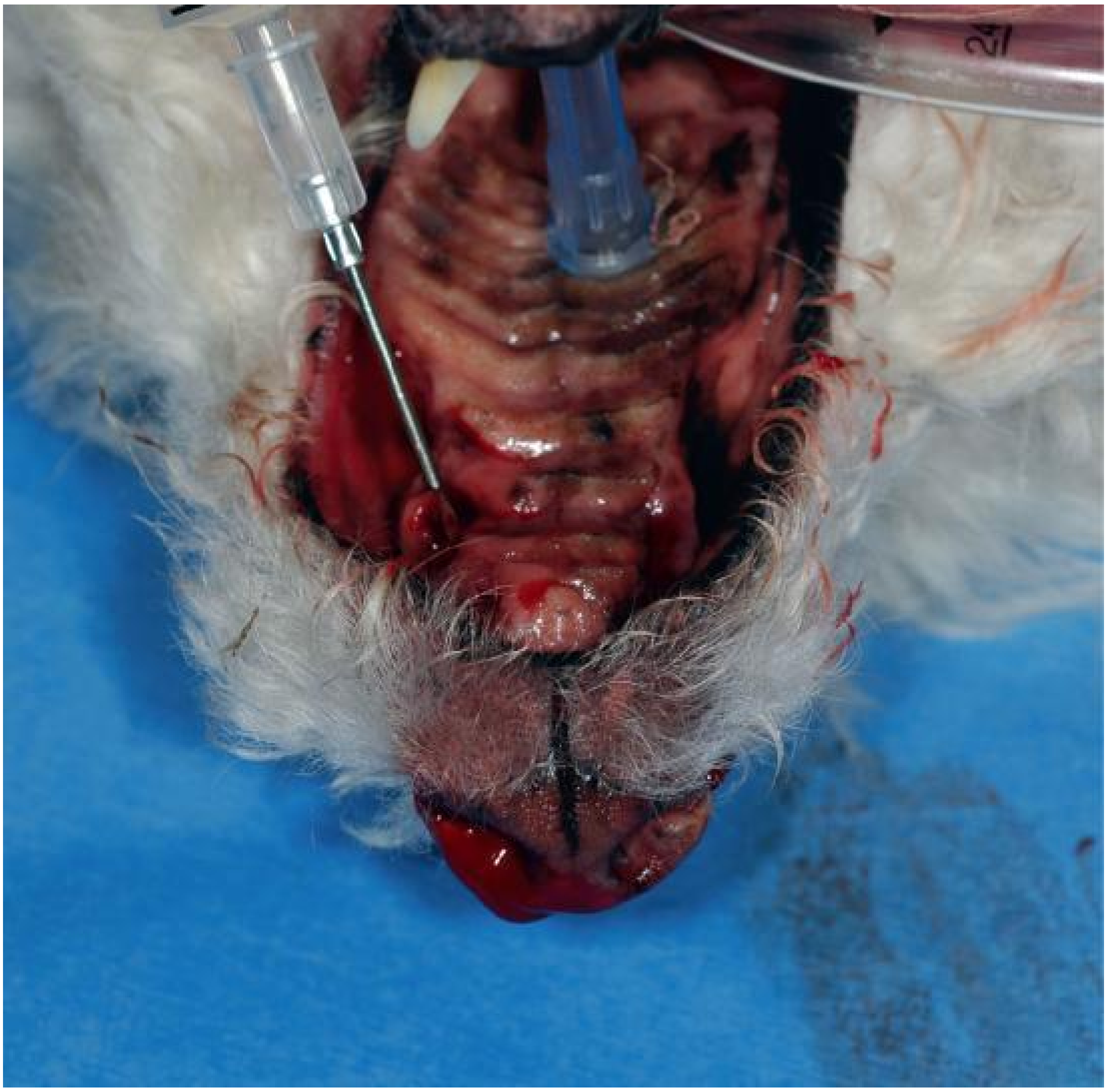


• **FIG. 15.4** Diagrammatic illustrations showing the position of releasing incisions for the various full-thickness mucoperiosteal flap designs for surgical extraction of a maxillary canine tooth: (**A**) pedicle flap; (**B**) triangle flap.



• FIG. 15.5 Channels or troughs between the root and alveolar bone can be made with a round diamond bur on a high-speed handpiece to create space for an elevator or luxator.

After removal of a maxillary canine tooth, it is very important to determine whether communication with the nasal cavity exists or has been created by using a cotton tip applicator to assess the integrity of the alveolar bone. Communication is usually demonstrated by ipsilateral nasal bleeding (Fig. 15.6). This can be caused by avulsion of a part of the alveolar wall during extraction, in which case the avulsed bone will be visible attached to the palatal aspect of the extracted tooth, or by periapical bone destruction due to a chronic inflammatory process. Either case can result in a persistent oronasal fistula if tension-free flap closure is not achieved.



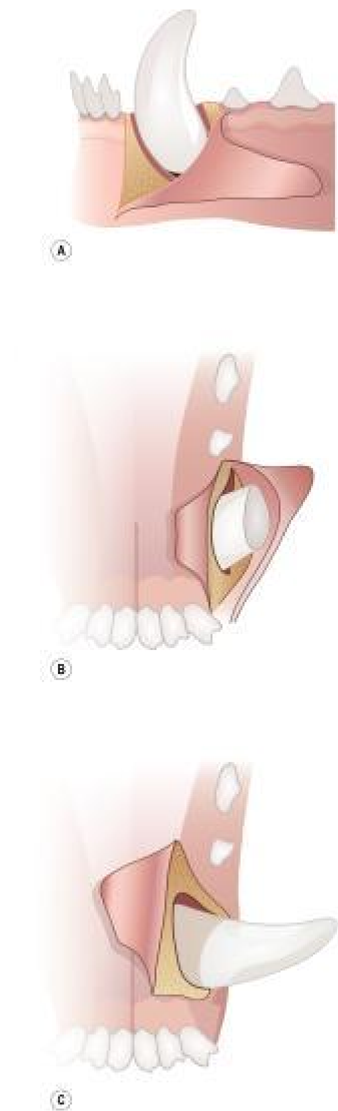
• **FIG. 15.6** Photograph obtained immediately following extraction of both maxillary canine teeth. Oronasal communication is confirmed, as saline flushed through the vacated left canine alveolus reveals blood-tinged fluid exuding through the ipsilateral nostril.

Prior to closure, any palpably sharp bone margins should be smoothed (alveoloplasty) using a round bur on a high-speed handpiece, bone file, or rongeurs. The vacated alveolus and flap should be cleared of any debris (e.g., tooth fragments, bone, hair, or dental calculus) by gentle curettage or irrigation. If neither periapical lesion nor debris is present within the alveolus, the alveolus should not be curetted, as the blood clot present plays an important role in healing. The flap is then eased into its original position and sutured into place using an appropriately sized absorbable monofilament suture material (see Chapter 8 for discussion of suture materials).

Mandibular canine tooth—buccal approach

The mandibular canine tooth can be surgically extracted in a similar fashion; however, the flap design is different because of the presence of the lip frenulum. Following incision of the gingival sulcus with a #15 scalpel blade, an interproximal incision is extended 2–3 mm on the mesial aspect and distally on the alveolar margin and into the gingival sulcus of the mandibular first and second premolar teeth, up to the distal aspect of the latter tooth. A single, caudoventrally directed vertical releasing incision mesial to the lip frenulum is usually all that is necessary;

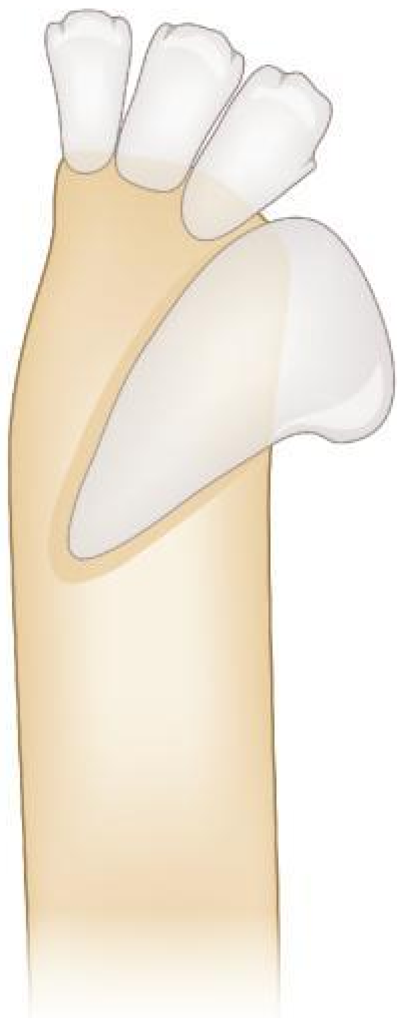
however, a distal vertical releasing incision can be implemented if additional exposure is needed (Fig. 15.7A). The resulting mucoperiosteal triangle flap, including the lip frenulum and associated mental neurovascular bundle, is raised and reflected in a caudoventral direction. The buccal alveolar bone is partly removed and the tooth is delivered as described above. Variations in normal anatomy, with respect to the size and location of the middle mental foramen, may limit the extent of the buccal alveolectomy, when attempting to avoid impinging upon the neurovascular structures that exit the foramen; this is particularly the case for brachycephalic dog breeds. A combination buccal and lingual approach has been described that involves the development of an additional lingual envelope flap and lingual alveolectomy (see Fig. 15.7B).[16] If this approach is used, and to facilitate access to the lingual surface, the crown of the mandibular canine tooth can be amputated just above the cemento-enamel junction of the tooth.[16] Great care should be taken in elevating or luxating this tooth, and the mandible should be firmly supported with the opposite hand.[14] Assessment and recording of the degree of mandibular symphyseal laxity in the medical record before proceeding with extraction of this tooth is a good practice, as a certain degree of laxity is a relatively common finding in small-breed dogs.[14] Particularly with this tooth, the premature application of the forceps may result in a fracture of the lingual surface of the alveolus that communicates with the symphysis.



• **FIG. 15.7** Diagrammatic illustrations depicting the different approaches for surgical extraction of a mandibular canine tooth. (**A**) Buccal approach. (**B**) Combined buccal and lingual approach with coronal amputation of the canine tooth. (**C**) Lingual approach.

Mandibular canine tooth—lingual approach

Fractures of the rostral mandible at the junction of the mandibular body and symphysis or through the mandibular canine tooth alveolus have been described as the primary complication associated with surgical extraction of the mandibular canine teeth. Anatomically, the mandibular canine root is oriented in a lingual direction from the crown and lingual and ventral to the middle mental foramen and roots of the mandibular first and second premolars (Fig. 15.8).[17] Mucoperiosteal flap development and alveolectomy from a lingual direction, commonly referred to as the lingual approach, avoids disruption of the frenulum and of the neurovascular structures of the middle mental foramen and avoids potential iatrogenic damage to the first and second premolar tooth roots (see Fig. 15.7C). In the cat, due to the wider interdental space between the mandibular canine and the mandibular third premolar teeth, and to the more central location of the canine root within the rostral mandible, a lingual approach to extraction is less desirable; an alternative dorsal approach has been described that provides for a significantly shorter closure time compared with the buccal approach.[18] The amount of bone typically removed during a lingual alveolectomy, however, is relatively similar to that of buccal alveolectomy.[17] For the lingual approach, a pedicle flap design is indicated. The mesial and distal vertical releasing incisions of the flap are started at their respective line angles with the mesial release directed in a perpendicular direction, without disruption of the gingiva at the adjacent mandibular third incisor tooth, and the distal release in a caudoventral direction, following the direction of the root. The lingual alveolectomy is performed using a high-speed handpiece and a round carbide or diamond bur. The tooth is extracted using traditional elevation and luxation techniques as described previously. Following the necessary alveoloplasty to contour sharp bone margins, the flap is fully repositioned and sutured to the adjacent tissues.



• **FIG. 15.8** Lingual alveolectomy of the right mandibular canine performed on the skull of a dog demonstrating the lingual orientation of the root in relation to the crown.

Deciduous canine teeth—special considerations

The long, slender roots of deciduous canine teeth, and their proximity to the developing permanent canine teeth, create challenges during extraction of these teeth. Luxators and elevators must be used with great care and should be placed as far from the permanent tooth bud as possible.[6] Root fracture is common; fractured root tips may or may not resorb. Presence of deciduous root tips can impair normal eruption of the permanent tooth, which can result in malocclusion.[5] A surgical approach, consisting of a triangle flap and partial buccal alveolectomy, greatly improves visibility and reduces the likelihood of root fracture.[5,6]

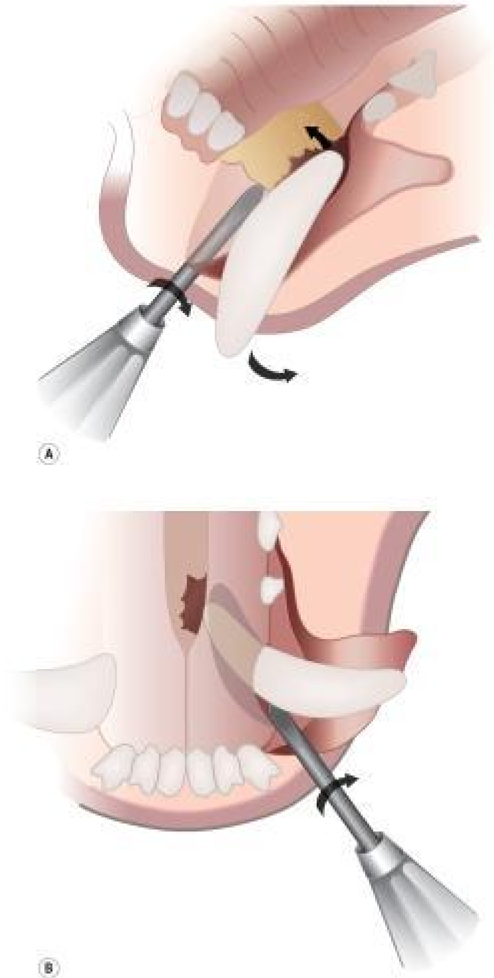
Use of a piezoelectric surgery unit for the luxation of deciduous tooth roots or retrieval of deciduous root tips, as an alternative to traditional rotary and hand instruments, may lessen the chance of iatrogenic trauma, by eliminating potentially damaging leverage forces of hand instruments and heat transfer of rotary instruments onto the developing tooth bud.[19]

Complications

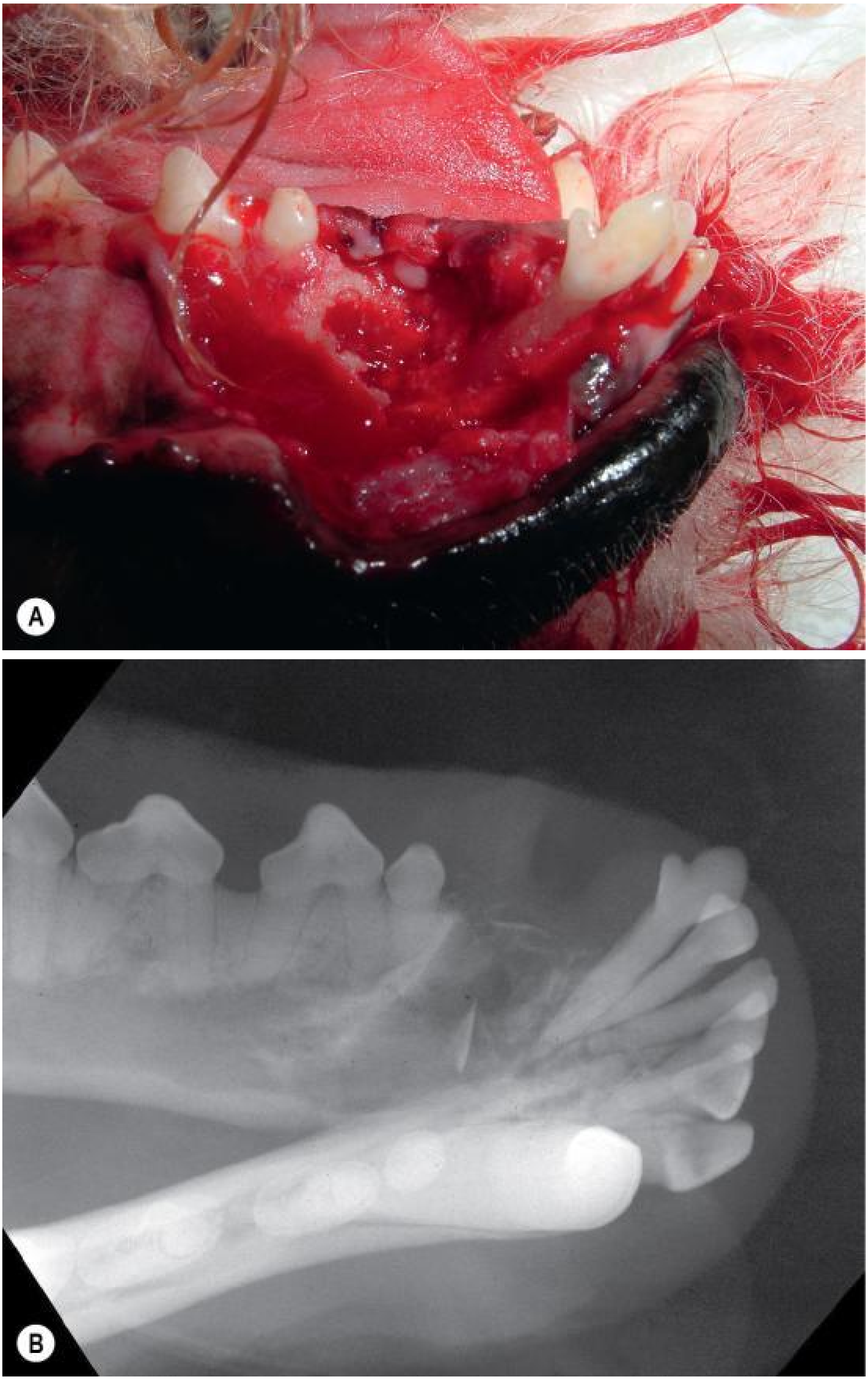
Intraoperative complications

If excessive force is applied with the extraction forceps, large portions of the buccal or lingual cortical bone plate may be unnecessarily included with the extracted root; this is most likely to occur when extracting the maxillary canine tooth. This may also occur with greater frequency during the extraction of persistent deciduous canine teeth. If more exposure is needed, it is far better to raise a soft tissue flap or remove additional bone in a controlled fashion with either a bur or rongeurs.

Perhaps the most advantageous location for positioning a luxator or elevator for application of leverage force is along the mesiopalatal or mesiolingual surface of the canine tooth. Although this leverage sweet spot may be helpful, if applied in excess, the apex of the maxillary canine tooth may be inadvertently displaced in the direction of the nasal cavity (Fig. 15.9A), or that of the mandibular canine tooth through the symphysis (Figs. 15.9B & 15.10). Since the alveolar bone along the palatal aspect of the maxillary canine tooth is normally thin and may already be compromised due to chronic periapical disease, introduction of the apex of the tooth into the nasal cavity is very possible, resulting in oronasal communication and hemorrhage from the ipsilateral nasal cavity. The luxator or elevator may also be traumatically introduced into the nasal cavity. A stop created by grasping the handle of the luxator or elevator with one finger close to the end of the blade may minimize the depth of penetration. Nasal bleeding should resolve in 2–3 days with closure of the extraction socket using an appropriately designed flap.



• **FIG. 15.9** Inappropriately forceful elevation at the mesiopalatal and mesiolingual surfaces of the maxillary and mandibular canine teeth, respectively, can lead to iatrogenic (**A**) oronasal communication and (**B**) mandibular fracture.



• **FIG. 15.10** (**A**) Clinical photograph and (**B**) accompanying intraoral lateral radiographic view of a dog with an iatrogenic fracture through the right mandibular canine tooth alveolus.

The middle mental nerve, which supplies sensation to the regional skin and hair follicles, exits from the middle mental foramen and can be transected with an incorrectly placed distal vertical releasing incision for a mucoperiosteal flap or nicked by a bur during alveolectomy. Being aware of the neurovascular anatomy in the surgical area is the best prevention for nerve injury. In humans, the sensory innervation of these nerves is small, and reinnervation and return of sensation has been reported to occur rapidly.[15]

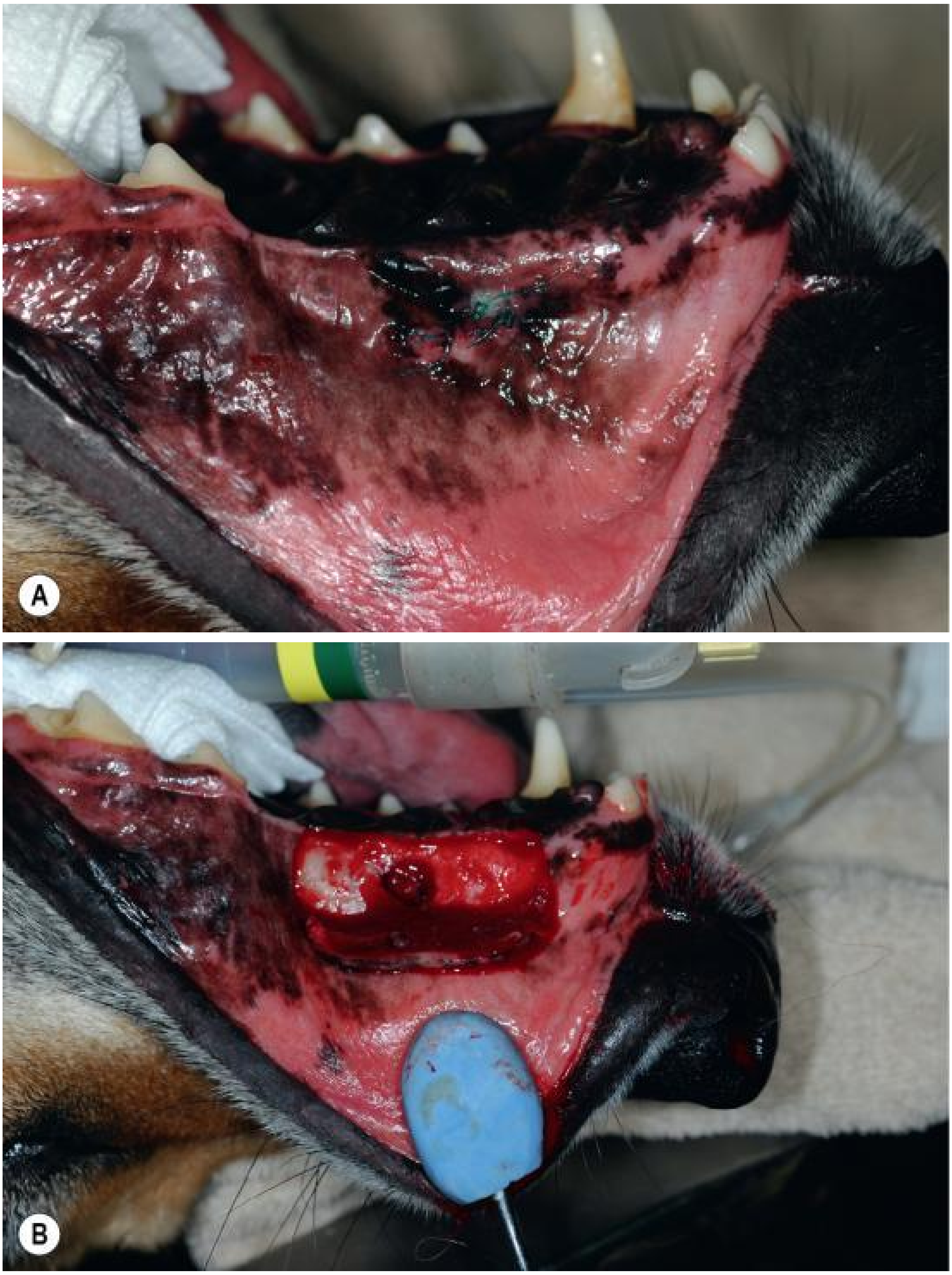
Intraoperative hemorrhage arising from the alveolus or transection of the branch of the major palatine artery that passes between the maxillary third incisor and canine teeth is inevitable; both sources are usually controlled easily and successfully upon closure of the alveolus. Avoidable sources of hemorrhage, such as the middle mental vessels as they exit the middle mental foramen, may not be as readily controlled, as the vessel can be small and retract into the confines of the foramen, requiring additional exposure to directly ligate.

Inadvertent damage to the adjacent teeth and roots by inappropriate use of extraction instruments is most likely to occur during extraction of the mandibular canine tooth. The mandibular third incisor tooth is located very close to the mandibular canine tooth, and the attached gingiva of this tooth can be accidently damaged, or worse, the tooth luxated or partially avulsed. Also due to their proximity, the roots of the mandibular first and second premolar teeth may be exposed or even resected with excessive buccal alveolectomy. Unerupted or developing permanent canine teeth may also be damaged during extraction of persistent deciduous teeth.

Postoperative complications

Oronasal fistula

Oronasal fistula may have been diagnosed prior to extraction of the maxillary canine tooth or identified as an intraoperative complication caused by avulsion of a portion of the alveolar wall during the extraction. Therefore following extraction of a maxillary canine tooth, it is very important to check if a communication with the nasal cavity has been created, as evidenced by ipsilateral nasal bleeding (Fig. 15.11). This complication, if not observed intraoperatively, can largely be avoided with apposition of freshly debrided epithelial edges and precise flap closure. For teeth with previously existing oronasal fistulas, the epithelium lining the palatal aspect of the fistula must be thoroughly debrided prior to suturing the flap. Failure to perform this step will result in recurrence of the fistula. In some cases, the extraction of the teeth adjacent to the fistula, the maxillary third incisor and first premolar teeth, will allow for improved contouring of the surrounding alveolar bone and flap adaptation when suturing the flap.



• **FIG. 15.11** Dog with an oronasal fistula following inappropriate closure of a left maxillary canine tooth extraction site. (**A**) Dehiscence of sutures with oronasal fistula. (**B**) Direct visualization of the oronasal fistula following elevation of a full-thickness mucoperiosteal pedicle flap. Hemorrhage is visible from the ipsilateral nasal passage.

Flap dehiscence

Wound tension and closure of the soft tissue flap without adequate underlying bony foundation are the primary causes of flap dehiscence.[20] Overly tight wound closure, paradoxically, can also predispose to complications.[21] Although second-intention healing will usually suffice for wound dehiscence of extraction sites other than the maxillary canine tooth, if oronasal communication was the reason for extraction in the first place, or if it occurred as an intraoperative complication, a second surgery for revision of the flap may be indicated. A common area of exposed bone following mandibular canine tooth extraction is at the mesial edge of the flap for the mandibular canine closure. A sharp rim of bone along the mesial alveolus, or an abrupt dip from the third incisor tooth into the mandibular canine alveolus, if not removed by alveoloplasty, may contribute to dehiscence in this area. The weight of redundant lip tissue, in brachycephalic dog breeds or other heavy-lipped breeds such as the Newfoundland or Saint Bernard, may cause excessive pull on the suture line and increase the risk of flap dehiscence. Fenestration of the flap may occur in the process of elevating a flap with a periosteal elevator or because of an aberrantly directed rotary bur in an unprotected area of the flap. Both iatrogenic complications can be prevented with improved technique; however, severely inflamed regions of the flap, which are very thin, may also predispose to intra- or postoperative fenestration. The latter is typically of greater concern with the canine teeth of cats with alveolar mucositis and can generally be avoided by careful inspection of the flap, excising the affected area, and appropriately revising the flap design prior to closure. Poorly executed flap closures with inverted edges, in particular where there are deep palatal rugae, and overfilling vacated tooth sockets with graft material, beyond the bony confines of the socket, can also contribute to flap dehiscence.

Maxillary lip entrapment

Excessive buccal alveolectomy with extraction of the maxillary canine tooth in the dog, although more common in the cat, may lead to entrapment of the maxillary lip between the mandibular canine tooth and the palate, a condition that can be very uncomfortable for the patient, ranging from abrasion and ulceration to an actual full-thickness puncture through the lip (Fig. 15.12).[22] Treatment options for this condition include crown-height reduction (with endodontic therapy if the pulp is exposed) or extraction of the offending mandibular canine tooth.[23] Immediate replacement of the tooth with a dental implant, followed by crown restoration, may also be a feasible alternative to prevent collapse of the buccal bone plate.[24]

• **FIG. 15.12** Severe entrapment of the upper lip in a dog following extraction of the right maxillary canine tooth.

Weakening of the mandible

Anatomically, the roots of the mandibular canine teeth provide most of the structure of the rostral mandible. Following extraction of the mandibular canine teeth, especially in small or toy-breed dogs with preexisting infection, the ensuing atrophy of the rostral mandible and symphysis can predispose to pathologic fracture. Placement of an autologous bone graft or allograft (see Chapter 13) may reduce the severity of atrophy and help preserve the integrity of the rostral mandible.

Glossoptosis

At rest, the height of the mandibular canine tooth crowns retains the tongue within the confines of the mouth. Following extraction, the tongue may occasionally droop to the side of the mouth, missing the mandibular canine tooth (Fig. 15.13). In humans, this condition is referred to as glossoptosis, or a downward replacement of the tongue, and is seen as the most common anomaly of the disorder Pierre Robin sequence in infants.[25] Commissurorraphy, a cheiloplasty procedure often performed following total mandibulectomy, can be performed to assist in retaining the tongue within the mouth of dogs experiencing extreme functional impairment (see Chapter 56).[20,26]

• FIG. 15.13 Glossoptosis in a dog following extraction of the left mandibular canine tooth.

Prognosis

The prognosis following appropriately indicated, technically well-performed extraction of canine teeth is excellent. The impairment of the patient following extraction of these important teeth is often more cosmetic than it is functional, and for that reason may be objectionable to the client, but in contrast to the pain and discomfort associated with not extracting a compromised canine tooth, extraction should be considered an acceptable treatment. Complications such as oronasal fistula associated with maxillary canine tooth extraction, if properly managed, can also yield successful results. Other complications, such as mandibular fracture, are certainly more challenging to correct, but when promptly addressed, and when performed without further damage to adjacent teeth, patient morbidity can be kept to a minimum and the results can also be good. Most of the severe complications associated with canine tooth extraction, although treatable, are best avoided in the first place through the use of applied surgical anatomy, proper exodontic technique, and appropriate armamentarium.

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CHAPTER 16

Extraction of multirooted teeth in dogs

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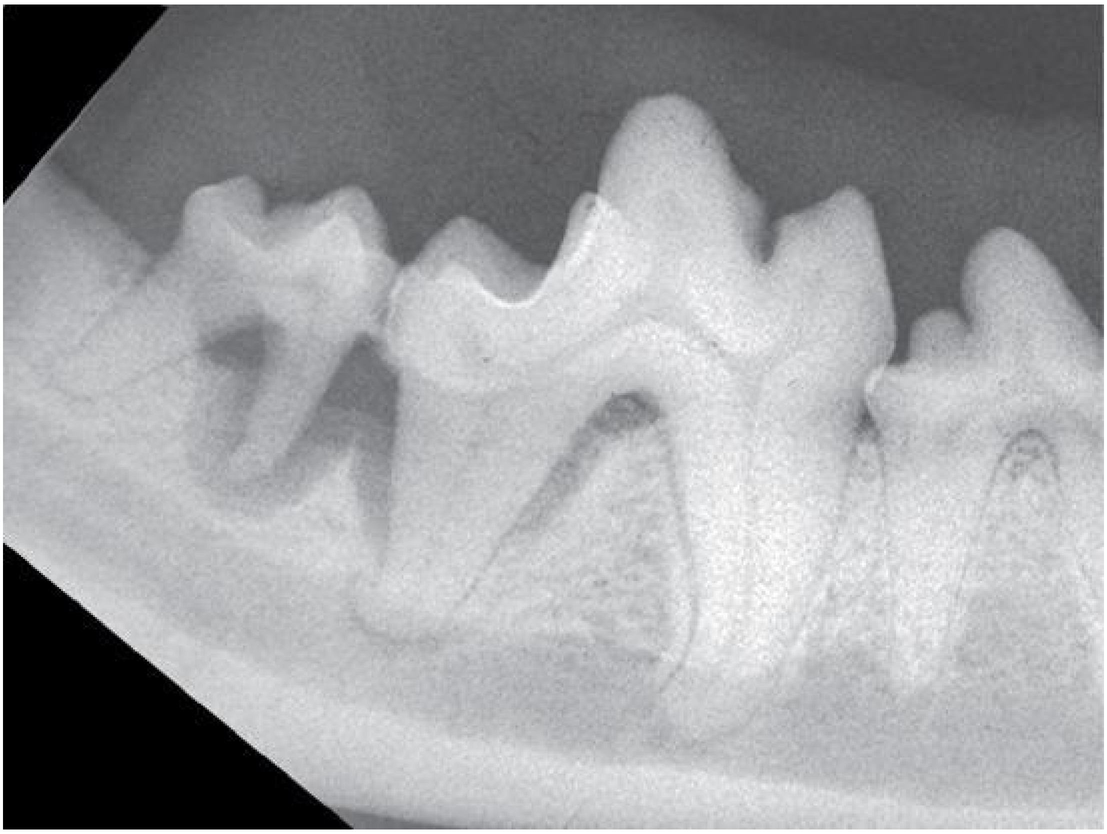
Indications for extraction

Indications for extraction are discussed in Chapter 13. Extraction of teeth affected by severe periodontitis and fractured teeth with pulp exposure (for which endodontic treatment is not feasible) are among the most commonly performed procedures in veterinary practice. Once periodontitis involves the furcation of a multirooted tooth, cleaning of the exposed root surface becomes nearly impossible.[1] In addition, plaque retention in the exposed furcation results in rapid progression of periodontitis.[1] Because of these factors, extraction is typically recommended for teeth with through-and-through (stage 3) furcation exposure.[2] In dogs, it is not uncommon for vertical bone loss to affect only one root of a multirooted tooth or even one aspect of a single root (Fig. 16.1). In this situation, extraction of the periodontally sound root is significantly more challenging than removal of the root with little remaining bone. Advanced treatment options such as periodontal surgery with guided tissue regeneration, or hemisection and root canal treatment, may be alternatives to extraction in selected cases.[1,3,4]



• **FIG. 16.1** Severe vertical bone loss affecting the distal root of the right mandibular first molar tooth and the mesial root of the right mandibular second molar tooth of a dog.

Perhaps the most challenging extractions are those involving fractured teeth with no periodontal bone loss. When possible, endodontic treatment is preferable to extraction of periodontally sound teeth with pulp exposure. If extraction is selected, preoperative radiographs are imperative to establish the proximity of associated vital structures (e.g., the relationship of the maxillary first molar roots to the orbit or of the mandibular first molar roots to the mandibular canal), evaluate root morphology (e.g., presence of dilaceration, root resorption, or supernumerary roots), assess the location of permanent tooth roots when extracting persistent deciduous teeth, and assess the condition of the surrounding bone and periodontal ligament.[5] Evidence of root ankylosis or resorption will often necessitate a wider area of exposure and bone removal.

Preoperative concerns

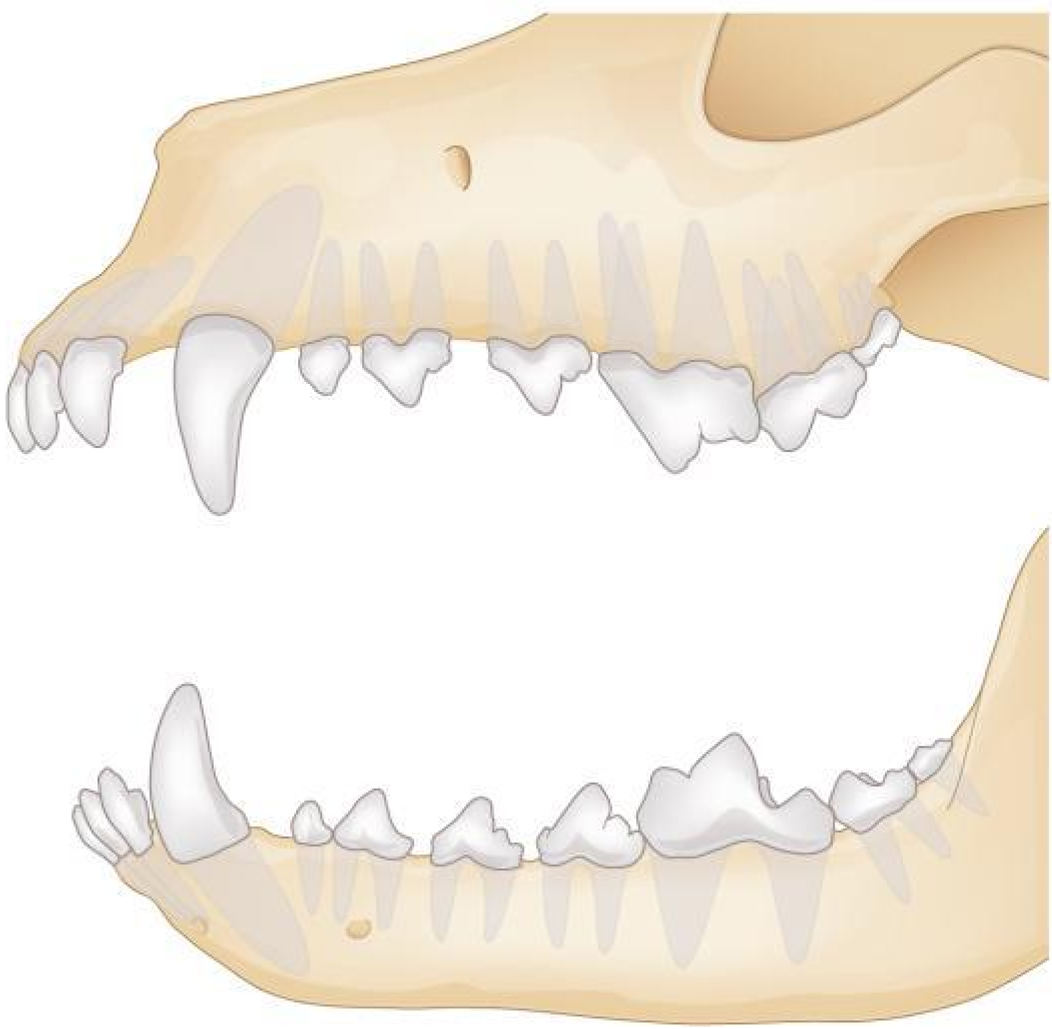
General health status

Patients with severe comorbidities (e.g., end-stage renal failure and uncompensated cardiac disease), uncontrolled endocrinopathies (e.g., poorly regulated diabetes or Addison disease), or untreated coagulopathies and dogs with high-grade tracheal collapse are poor candidates for general anesthesia and extensive oral surgery. While management of the oral disease may improve the patient's quality of life, oral surgery should be deferred until the disease can be brought under reasonable control.[5]

Surgical anatomy (fig. 16.2)

Maxillary premolar and molar teeth

The maxillary second and third premolar teeth have short, pointed crowns and two roots of approximately equal size. Supernumerary roots occur in approximately 10% of dogs and often affect the maxillary third premolar tooth.[6]

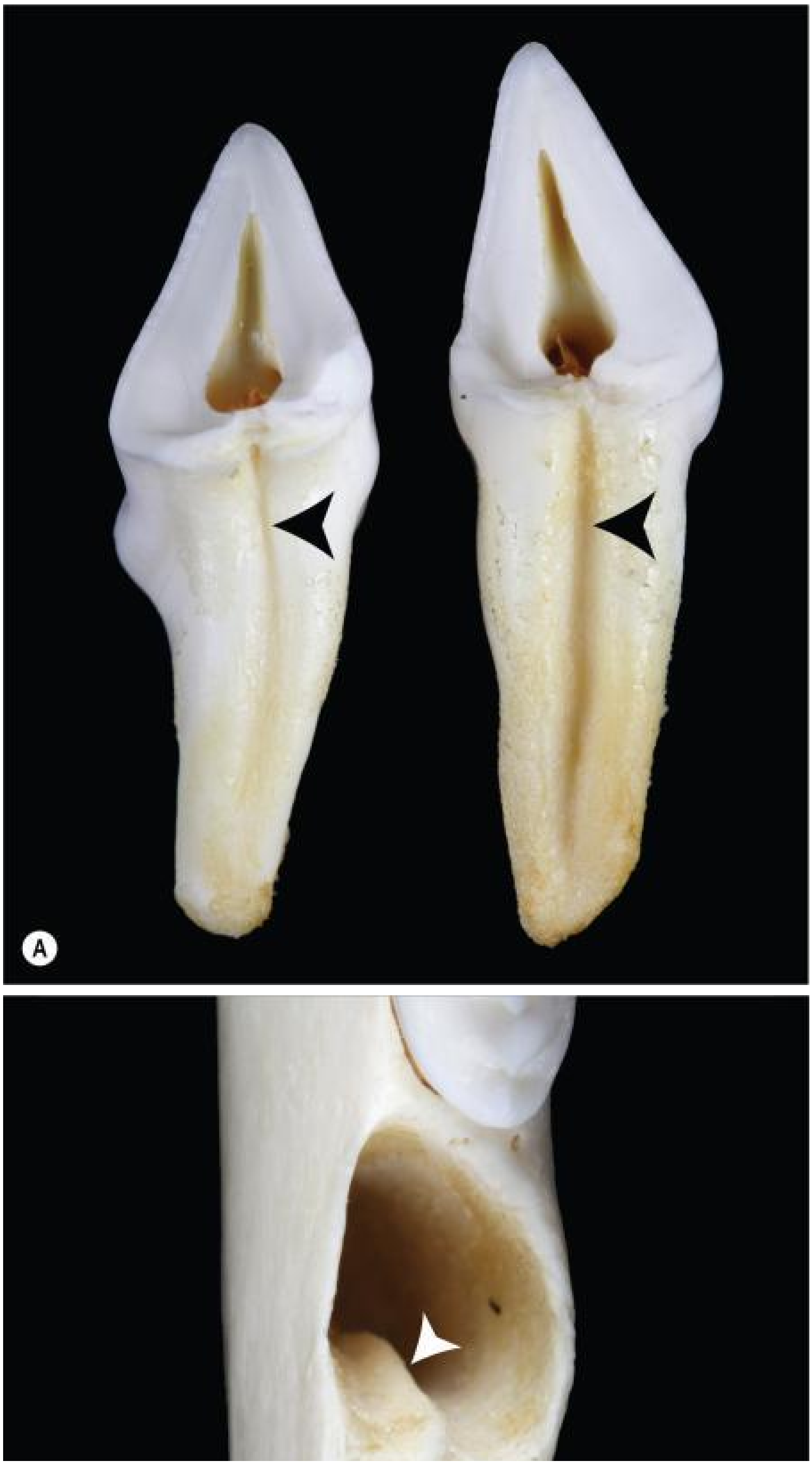


• FIG. 16.2 The dentition of a typical mesocephalic dog.

The maxillary fourth premolar and first and second molar teeth each have three roots. However, the second molar tooth is much smaller than the first, and the roots are relatively short and somewhat fused; as a result, when severely diseased, this tooth is typically extracted without sectioning. The first molar tooth is a large tooth with a short crown: the palatal root is short, conical, and wide relative to the two long and slender buccal roots. The maxillary fourth premolar tooth is a large three-rooted tooth with a pointed crown. The distal root typically has a triangular shape with a broad base at the cementoenamel junction. The mesiobuccal and palatal roots are usually narrow and slender, with a less triangular shape.[7]

Mandibular premolar and molar teeth

The mandibular second, third, and fourth premolar and first and second molar teeth each have two roots. Fused roots are relatively common, usually affecting the second premolar teeth and sometimes the second molar tooth.[6] The roots of the large premolar and molar teeth may have a distinct radicular sulcus, corresponding with an interradicular septum crest or intraalveolar crest: in particular, the mesial root of the mandibular first molar tooth has a prominent radicular sulcus (Fig. 16.3).[2,8]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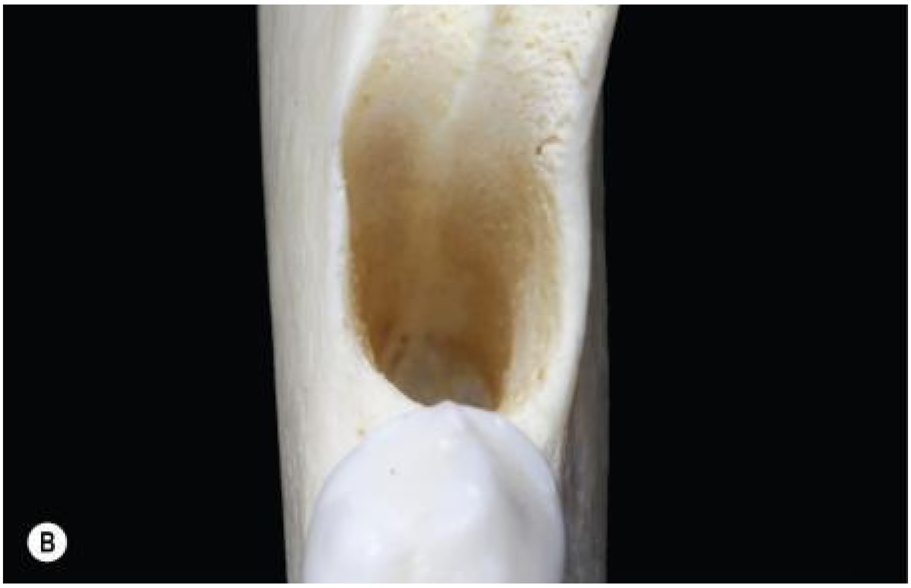


• **FIG. 16.3** (**A**) Radicular sulci *(black arrowheads)* of the sectioned right mandibular first molar tooth (*left*, distal root; *right*, mesial root) of a dog and (**B**) the corresponding interradicular septum *(white arrowhead)* in the vacated alveoli.

Adjacent structures

On the maxilla, the infraorbital artery, vein, and nerve exit the infraorbital foramen dorsal to the distal root of the maxillary third premolar tooth.[9] On the caudal palate, the major palatine artery exits the major palatine foramen (located medial to the fourth premolar tooth) and runs rostrally to the palatine fissure, where it anastomoses with infraorbital and nasal vessels. The parotid salivary duct opens into the oral cavity through a papilla at the rostral end of a fold of mucosa dorsal to the distal root of the maxillary fourth premolar tooth.[7] The zygomatic salivary gland sends one major duct and two to four minor ducts to the caudal oral cavity. The zygomatic papilla, through which the major duct exits, is located about 10 mm caudal to the parotid papilla in a medium-sized dog, dorsal to the maxillary first molar tooth.[7] The maxillary recess *(recessus maxillaris)* is a lateral diverticulum of the nasal cavity, the opening of which lies in a transverse plane through the mesial roots of the maxillary fourth premolar tooth.[9] It is possible to dislodge a root into this recess if excessive force is used during extraction attempts. In brachycephalic breeds, the orbit lies directly dorsal to the maxillary fourth premolar, first molar, and second molar teeth. Orbital penetration has been reported during attempts to extract these teeth.[10]

On the ventral aspect of each mandible is located the mandibular canal, containing the inferior alveolar artery, vein, and nerve. These structures exit the canal through three mental foramina on the rostral mandible. The largest of these is the middle mental foramen, located ventral to the second premolar tooth. When creating mucogingival flaps, care must be taken to avoid inadvertently transecting the mental neurovascular bundle. The proportion of the mandible occupied by the teeth varies with breed; small dogs have disproportionally larger mandibular first molar teeth relative to the height of the mandible when compared with large dogs (see Fig. 13.7).[11] It is possible to cause iatrogenic fracture of the mandible during attempts to extract mandibular first molar teeth if excessive force is applied.

Therapeutic decision-making

Preextraction radiographs of teeth to be removed should be obtained. This will allow confirmation of the diagnosis, visualization of root morphology, identification of root resorption or root ankylosis, and evaluation of the quality of the supporting jaw bone.[2]

Simple or surgical extraction

While a severely mobile multirooted tooth may be a candidate for simple extraction, most multirooted teeth will require surgical extraction, which involves sectioning the tooth, with or without creation of a gingival or mucogingival flap and removal of alveolar bone.

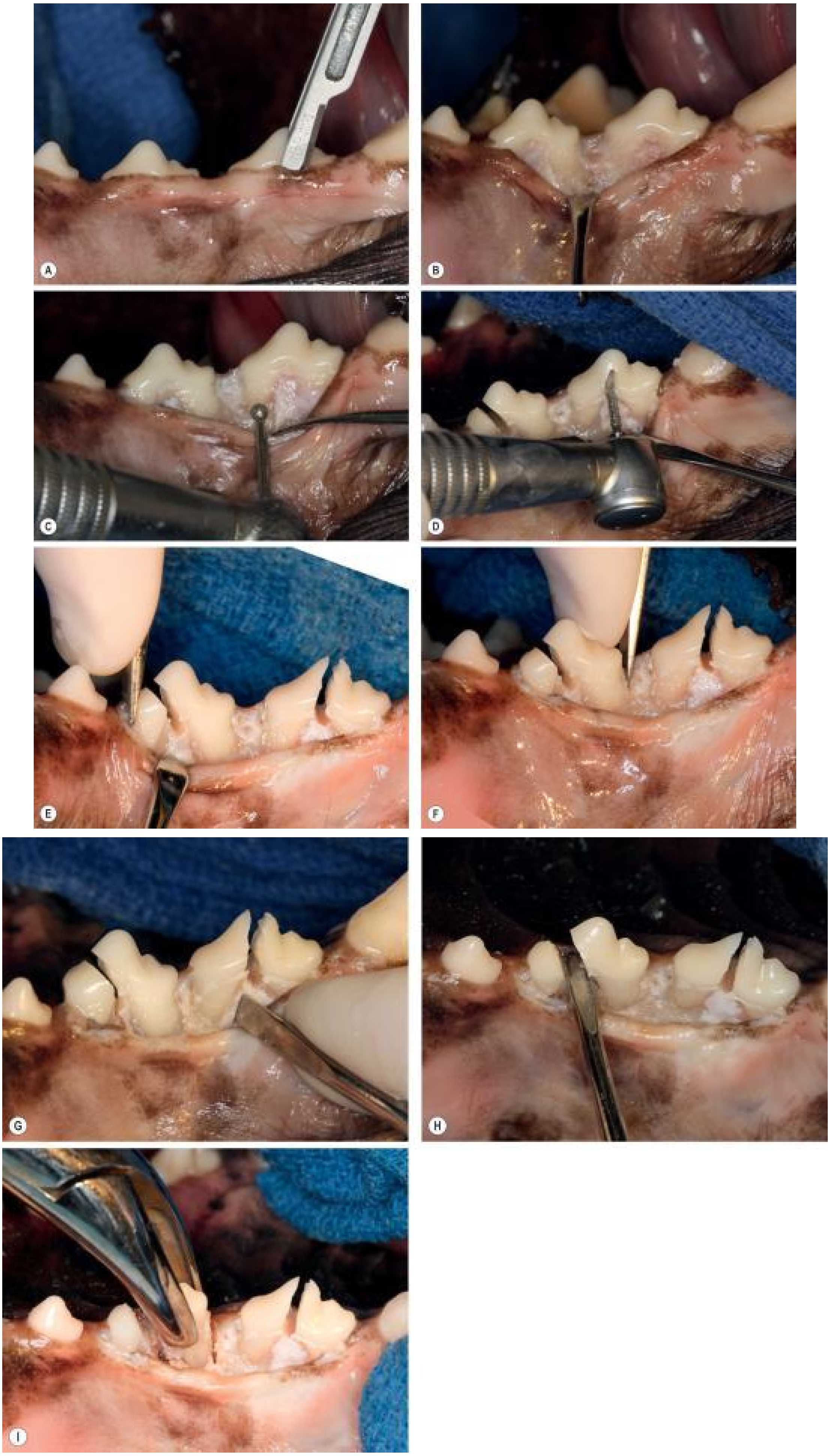
Flap design

The three primary gingival and mucogingival-periosteal flap designs (envelope, triangle, and pedicle—see Chapter 13) are used for surgical extractions of multirooted teeth in dogs. An envelope flap (which is a gingival flap, as the alveolar mucosa is typically not included) may be used for sectioning of mobile multirooted teeth where removal of alveolar bone is not required. Flap design will be discussed in further detail with the description of technique for each tooth type.

Surgical technique

Extraction of maxillary second and third premolar and mandibular second, third, and fourth premolar teeth

For extraction of a single premolar tooth, a triangle mucoperiosteal flap, with a vertical-releasing incision at the mesial interproximal space or line angle of the mesial adjacent tooth, is most useful. When two or more adjacent teeth are being extracted, an envelope flap typically provides adequate visualization and exposure of the buccal alveolar bone (Fig. 16.4A–B). If necessary, 1–3 mm of bone may be removed from the alveolar margin to expose the furcations and visualize the roots prior to sectioning (Fig. 16.4C). A tapered bur on a high-speed handpiece is used to section the teeth, beginning at the furcation and progressing coronally, so each root may be separately extracted (Fig. 16.4D). A luxator is introduced on the mesial (Fig. 16.4E) and distal (Fig. 16.4F) aspects of each root, using gentle pressure and small axial rotations (5–10 degrees) to direct the blade apically in the periodontal space, cutting the periodontal attachment.[2] Alternatively, the luxator may be placed in the periodontal ligament space at a 90-degree angle to the root to sever periodontal ligament fibers (Fig. 16.4G). Once some mobility has been achieved, an elevator may be placed between sectioned roots, and gentle rotational pressure is applied to fatigue the periodontal ligament and lift the root from the alveolus (Fig. 16.4H). When using the elevation technique, care must be employed to avoid generating excessive force, or root fracture will occur. Once each root has demonstrated significant mobility, extraction forceps are placed as far apically as possible. Applying gentle rotational forces and minimal traction with the extraction forceps, each root is delivered from its alveolus (Fig. 16.4I). Premature application of extraction forceps, or use of excessive force with extraction forceps, may result in root fracture.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• **FIG. 16.4** Extraction of a maxillary second and third premolar tooth in a dog. The patient is in dorsal recumbency. (**A**) An envelope flap is created and reflected (**B**) to expose the alveolar bone. (**C**) A round diamond bur on a high-speed handpiece is utilized to remove a small amount of buccal alveolar bone. (**D**) Then a tapered diamond bur is used to section the roots. This may be performed directly through the center of the crown (as was done at the third premolar tooth) or at an angle to minimize the amount of crown the bur travels through (as at the second premolar tooth). Luxation on the mesial (**E**) and distal (**F**) aspects of each root is performed to sever the periodontal ligament fibers. (**G**) The luxator may also be used perpendicular to the long axis of the root. (**H**) Elevation may be employed as an alternative or adjunct to luxation. (**I**) Finally, extraction forceps are placed on each root and rotated gently to free any remaining attachment.

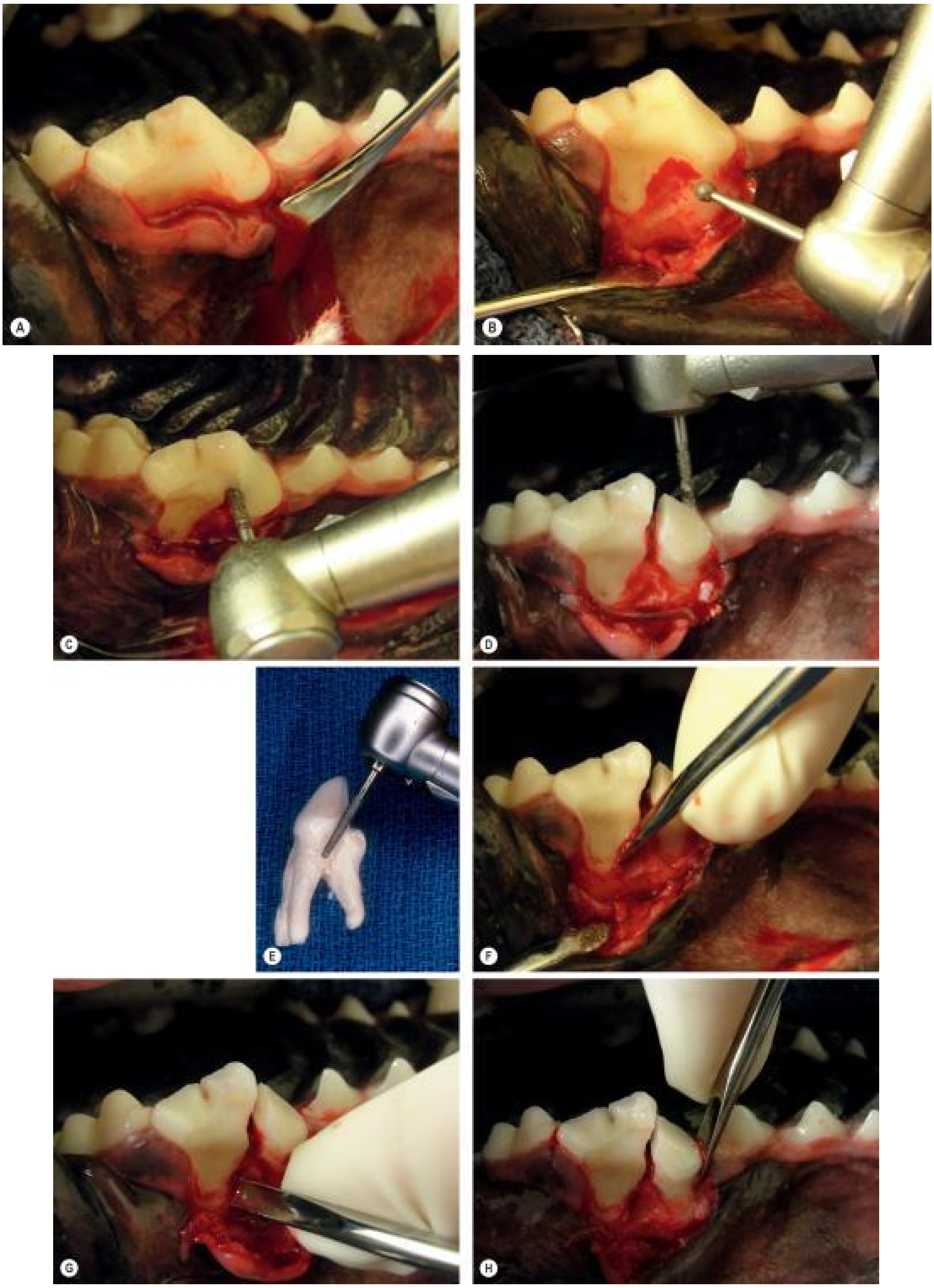
If a root fracture occurs, it is important to have good lighting and visibility before attempting removal of the remaining fragment. If fracture occurred close to the cementoenamel junction and the remaining fragment is large, a 2-mm dental luxator or small elevator may be sufficient to loosen and remove it. Small root tips may be mobilized using 1- or 2-mm luxators, root tip picks, or root tip elevators and then removed with root tip forceps. It may be necessary to extend the mucogingival flap and remove more alveolar bone to obtain access to a small root fragment. Drilling out root tips ("pulverization" or "atomization") can result in damage to the infraorbital or inferior alveolar arteries and nerves, fatal air embolism, or other iatrogenic trauma and is not advisable.[12]

After all roots and root fragments are removed, any expanded alveolar bone is gently compressed back into position, and sharp bony edges are reduced with a round bur. Any granulation tissue present in the alveolus should be gently removed, the empty alveoli are flushed with sterile saline solution, and the mucogingival flap is repositioned and sutured without tension.[2]

Extraction of maxillary fourth premolar teeth

Dorsal recumbency allows much better visualization of the three roots of the caudal maxillary teeth and is therefore preferred to lateral recumbency for extraction of these teeth. A pedicle flap provides optimum visibility for extraction of the maxillary fourth premolar tooth, but a triangle flap may be sufficient for cases where extensive alveolectomy is not required and avoids the salivary papillae. As previously mentioned, the infraorbital blood vessels and nerve exit the infraorbital foramen dorsal to the distal root of the maxillary third premolar tooth. Therefore, when making a vertical releasing incision mesial to the maxillary fourth premolar tooth, care must be taken not to incise through the infraorbital neurovascular bundle. A perpendicular rather than divergent incision is less likely to damage the infraorbital vessels and nerve (Fig. 16.5). If a pedicle flap is selected, the incision at the distal aspect of the tooth should be extended 2–3 mm into the sulcus of the first molar and a vertical incision made, being careful to avoid the parotid and zygomatic papillae.[2] After the incisions are made, the full-thickness mucogingival flap is elevated and reflected to expose the buccal alveolar bone (Fig. 16.6A). Partial alveolectomy may be performed to facilitate exposure of the furcation between the mesiobuccal and distal roots. To facilitate extraction, particularly if the tooth is periodontally healthy or if ankylosis is evident on preoperative radiographs, additional alveolectomy may be performed along the root surfaces (Fig. 16.6B). Following alveolectomy, a tapered diamond or cylindrical carbide bur on a high-speed handpiece is introduced horizontally into the furcation and moved coronally to separate the distal from the mesial root (Fig. 16.6C). After reflecting the gingiva on the mesial aspect of the tooth to better visualize the furcation between the mesiobuccal and palatal roots, the bur is held at a 30-degree angle to the vertical axis of the tooth and moved through the furcation from mesial to distal (Fig. 16.6D–E). To improve the visibility of the furcation and allow for more accurate sectioning through the furcation, some authors recommend removing the mesial cusp of the tooth.[13] Once the tooth has been sectioned into three parts, a luxator may be introduced on the distal and mesial aspects of the two buccal roots (Fig. 16.6F–H). Placement of the luxator on the buccal aspect of the tooth should be avoided, as it is easy to slip out of the periodontal ligament space and across the surface of the alveolar bone, damaging the soft tissues. Horizontal leverage with an elevator placed between the distal and mesiobuccal roots and between the distal root and the adjacent first molar tooth may be employed after luxation has severed some of the periodontal ligament and the roots are somewhat mobile (Fig. 16.6I). It is important not to use excessive force when elevating, as the mesiobuccal root is more slender than the distal root and is likely to fracture if excessive torque is generated. The mesiobuccal root, in addition to comparatively being more slender, may also have root dilaceration, and complete and accurately angled tooth sectioning between the palatal and mesiobuccal roots is critical, especially if axial rotational forces are needed between these roots to deliver the mesiobuccal root.

• FIG. 16.5 A # 15 scalpel blade is employed to make an incision at the mesial-buccal line angle of the left maxillary fourth premolar tooth for creation of a triangle flap.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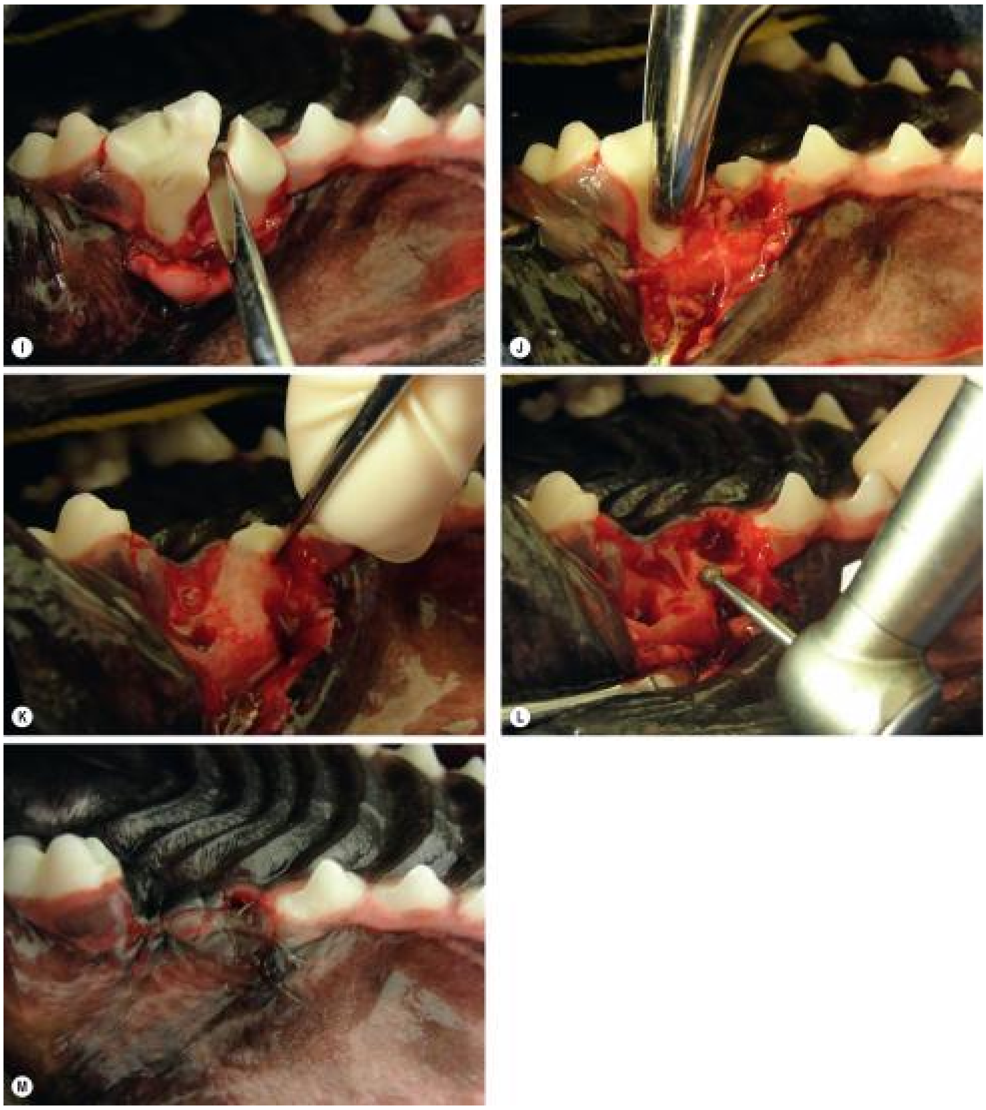


• **FIG. 16.6** Extraction of a maxillary fourth premolar tooth. (**A**) After making the incision, a triangle mucogingival flap is elevated. (**B**) Partial alveolectomy is performed using a round diamond bur. (**C**) A tapered diamond bur is used perpendicular to the vertical axis of the tooth to section the mesiobuccal root from the distal root and (**D**) at a 30-degree angle to the vertical axis of the tooth to section the palatal from the mesiobuccal root. (**E**) This anatomic specimen demonstrates the angle at which the handpiece should be held for optimum sectioning of the palatal from the mesiobuccal root. Although the luxator may be placed parallel to the long axis of the root, (**F**) it may also be helpful to use the luxator in a perpendicular manner, (**G**) to sever the periodontal ligament fibers. After the luxator has been introduced on the mesial and distal aspects of the distal and mesiobuccal roots, (**H**) careful elevation may be performed between the mesiobuccal and distal roots. (**I**) After removal of the mesiobuccal and distal roots, (**J**) the palatal root is easily visualized. (**K**) The luxator is then introduced on the mesial and distal aspects of the palatal root, at an approximately 60-degree angle to the hard palate. (**L**) Alveoloplasty, or removal of sharp bone edges, is performed prior to (**M**) suturing the flap.

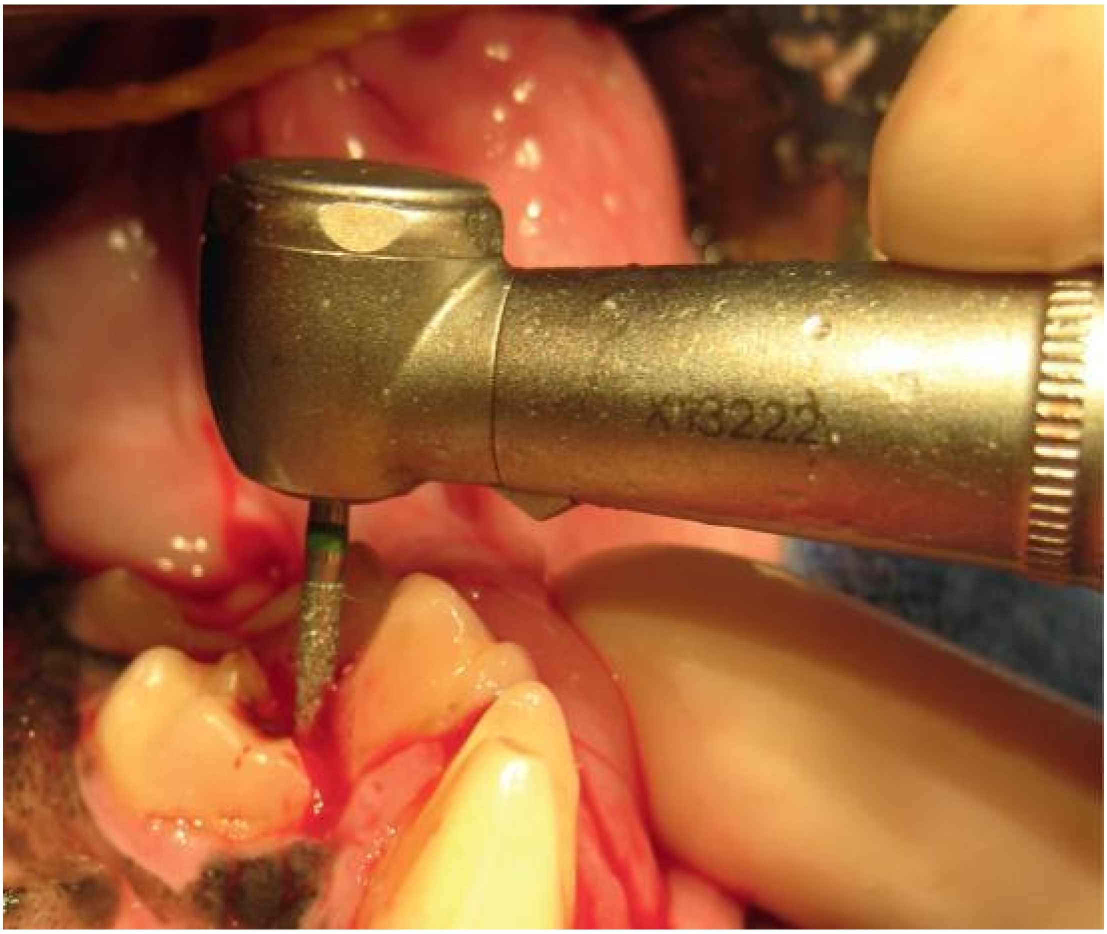
After the distal and mesiobuccal roots have been removed using extraction forceps (Fig. 16.6J), the palatal root is more easily visualized. Removal of some bone from the furcation region between the mesiobuccal and palatal roots will further improve visibility for placement of the luxator. The luxator should be introduced into the periodontal ligament space on the mesial and distal aspects of the root, and at a 60-degree angle to the hard palate (Fig. 16.6K).[2] Elevation is not recommended for this long, slender tooth root because fracture is likely. Once the luxators have been advanced apically and the root is mobile, extraction forceps are employed, with minimal rotational and tractional forces, to deliver the root from the alveolus.

Following removal of all three roots, any rough bony edges are smoothed with a round diamond bur on a high-speed handpiece (Fig. 16.6L), any granulation tissue is gently removed and the alveoli are flushed with saline. The mucogingival flap may be undermined further, and the periosteum may be incised if necessary, to ensure closure without tension. It may be preferable to suture the flap back in its original location (i.e., align the mucogingival junction of the flap with the mucogingival junction of the third premolar tooth, even though this may leave a small space at the palatal root alveolus; Fig. 16.6M) rather than attempting to suture the corner of the flap to the palatal mucosa. This ensures the presence of attached gingiva (rather than mucosa) at the distal aspect of the third premolar tooth.

Extraction of maxillary first molar teeth

Even in the presence of significant periodontal disease, it is advisable to extract the maxillary first molar tooth surgically. The two buccal roots are long and slender when compared with the palatal root and are therefore easily fractured.

With the patient in dorsal recumbency, a sulcular incision is made and extended 1–2 mm mesially. Taking care not to damage the zygomatic papilla, a vertical releasing incision can be made into the mucosa, creating a triangle flap, if necessary. After reflecting the gingiva from the mesial aspect of the tooth, a tapered bur is held vertically and moved from mesial to distal to section the palatal root from the two buccal roots (Fig. 16.7). The palatal root may be extracted first; it is shorter and more conical, making its extraction less challenging. Frequently, this root can be extracted with extraction forceps alone. If an elevation technique is employed using the two mesial roots for leverage, the mesial roots may fracture. Therefore, if extraction forceps alone are insufficient to remove this root, use of luxators rather than elevators is recommended. The buccal roots are separated from one another using a tapered bur. Removal of buccal alveolar bone may facilitate extraction of the two long, narrow buccal roots. Tissue retractors (e.g., Cawood-Minnesota retractor) should be employed to prevent damage to the cheeks from the bur during partial alveolectomy and sectioning of this tooth. As there is risk of orbital penetration due to slippage of the luxator when extracting teeth in this region of the mouth, following sectioning and partial alveolectomy, luxation forces should be directed along the mesial and distal surfaces of the buccal roots, and the luxator should be gripped with an index finger-stop as close to the working end of the instrument as possible. An elevator may be carefully employed once luxation has resulted in significant mobility of the two buccal roots. Minimal tractional and rotational forces should be applied with extraction forceps for final delivery of each buccal root. The buccal bone plate overlying the buccal roots is very thin in this region of the maxilla, and premature use of the extraction forceps may result in fracturing of portions of the buccal bone plate still attached to the root; this is particularly the case with the distobuccal root, where the ligamentous attachment of the masseter muscle attaches to the buccal bone.



• **FIG. 16.7** A tapered diamond bur is used to section the palatal root from the more slender buccal roots of a maxillary first molar tooth.

If a vertical-releasing incision was made, it should be sutured. Otherwise, tension-free closure of the gingiva may not be possible without extensive flap development, and it is acceptable to leave the first molar sockets to heal by second intention.

Extraction of maxillary second molar teeth

The three roots of the maxillary second molar tooth are often fused, making sectioning unnecessary for extraction of the resulting short, conical tooth. In this case, the tooth may be removed intact with an elevator or extraction forceps; however, careful visual inspection should be performed to verify that the tooth was extracted intact. If the roots are not fused, they may be curved and therefore fracture easily.[2] If visibility allows, the tooth may be sectioned as described for the maxillary first molar tooth, and each root removed separately. If root fracture occurs, fragments may be retrieved with a 1-mm luxator or a root tip elevator; often, obtaining a dorsal-ventral intraoral radiographic view of the caudal maxilla will facilitate visualization of retained root tips in this region. Luxators and elevators must be used with extreme caution, as there is often a minimal amount of bone distal to this tooth, and inadvertent dorsal slippage of a luxator can easily lead to orbital trauma or laceration of the minor palatine branch of the maxillary artery (see Fig. 50.1B).

Extraction of mandibular first molar teeth

These are very large teeth, the roots of which are often adjacent to the mandibular canal. Extraction of a periodontally sound mandibular first molar tooth is difficult and may result in iatrogenic fracture. In addition, dilacerated roots may make extraction even more challenging, with an increased risk of root or mandibular fracture (see Fig. 13.6). When possible, conservative therapy (endodontic treatment for a fractured tooth or periodontal surgery and guided tissue regeneration for periodontally compromised teeth) should be offered as an alternative to extraction. If extraction is elected, creation of a triangle mucogingival-periosteal flap and removal of 50%–75% of the buccal alveolar bone will greatly facilitate extraction. When sectioning this tooth, it is suggested to start at the furcation and proceed in a distal-coronal direction, creating an oblique line between the distal and mesial roots, which avoids cutting through the tall central cusp (Fig. 16.8). A combination of luxators and elevators may be employed for extraction of the two large roots. The nonworking hand should be used to cradle and stabilize the ventral aspect of the mandible; this provides the added sensory benefit of being able to readily palpate the stress and strain on the mandible created by luxation and elevation forces. Luxators should be placed on the distal and mesial aspects of each root to avoid soft-tissue trauma due to slippage on the buccal or lingual aspects. After luxators have severed substantial periodontal ligament fibers such that the roots are somewhat mobile, an elevator can be introduced between the two roots, rotated 5–10 degrees, and held for several seconds to fatigue the remaining periodontal ligament and lift the roots from their alveoli. Extraction forceps may then be used with gentle traction to deliver the roots. The radicular sulcus of varying prominence that may be present along the distal surface of the mesial root, and to a lesser extent the mesial surface of the distal root (see Fig. 16.3), creates a tongue-and-groove effect with the interradicular septum, and is particularly prohibitive of the premature application of rotational forces with extraction forceps. If the alveolar bone has been expanded during the extraction, it should be compressed into its original position. Any sharp bony edges should be removed with a round diamond bur on a high-speed handpiece, any granulation tissue present gently removed, and the alveoli flushed with saline prior to apposition and suturing of the mucogingival flap.

• **FIG. 16.8** A tapered diamond bur is used to section the roots of a mandibular first molar tooth.

Complications

Complications of extractions are discussed in Chapter 18. Root fracture is the most common complication when extracting multirooted teeth. As previously discussed, removal of root fragments is most easily achieved following additional removal of alveolar bone and with improved visibility. Magnification loupes, adequate lighting, availability of suction, achieving hemostasis, and the use of delicate root tip picks (e.g., Davis and Heidbrink) and 1- and 2-mm luxators will greatly facilitate removal of fractured root fragments.

Less common complications include iatrogenic mandibular fracture when extracting mandibular first molar teeth in small-breed dogs and orbital penetration during extraction of maxillary fourth premolar or first molar teeth.[10] Proper preoperative assessment, including intraoral radiographs, and careful technique will help prevent these catastrophic complications.

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CHAPTER 17

Special considerations in feline exodontics

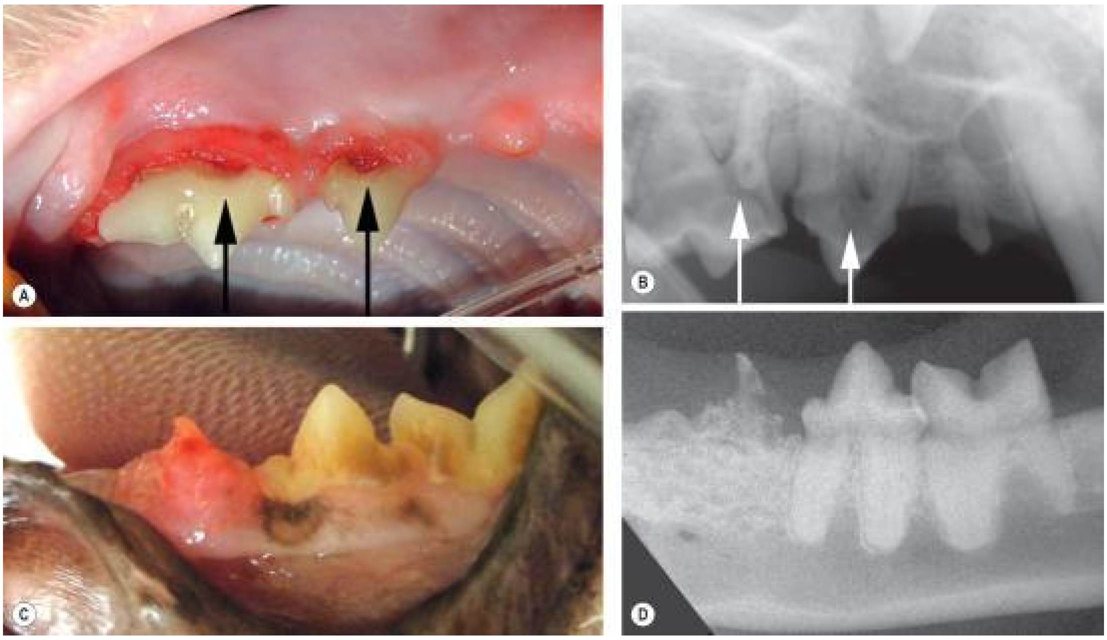
Milinda J. Lommer

Indications for extraction

The most common indications for extraction in cats are periodontal disease, tooth resorption, dental fractures, and chronic gingivostomatitis.

Severe periodontitis is a frequent indication for dental extractions in cats. Once periodontal disease has resulted in severe bone loss such that a tooth is very mobile, extraction is indicated. Also, if bone loss involves the furcation of a multirooted tooth, the furcation becomes a plaque-retentive niche and periodontitis often progresses rapidly, so conserving teeth with furcation exposure is usually not possible without exceptional home care.[1,2]

Tooth resorption (Fig. 17.1A–B) (formerly referred to as odontoclastic resorption lesions, neck lesions, and cervical line lesions) occurs in approximately one-third of the domestic feline population and in up to 60% of cats presented for dental treatment.[3-5] Extraction is currently the treatment of choice for teeth affected by resorption. However, root resorption makes the teeth prone to iatrogenic root fractures, making extraction challenging.[2] Coronectomy (also called crown amputation with intentional root retention) is an option for teeth with extensive root resorption (Fig. 17.1C–D) in the absence of periodontal or endodontic disease.[2,4,6,7]



• **FIG. 17.1** (**A**) Clinical and (**B**) radiographic appearance of type-1 resorption *(arrows)* at the right maxillary third and fourth premolar teeth of a cat. Note also the third root on the third premolar tooth. These teeth should be extracted in toto. (**C**) Clinical and (**D**) radiographic appearance of type-2 resorption at the left mandibular third premolar tooth. This tooth is a good candidate for coronectomy.

Fractured teeth are less common in cats than in dogs, but often go untreated because cats with dental fractures, unlike dogs, rarely present with facial swelling and draining tracts. However, even minor canine cusp tip fractures may result in pulp exposure, with the inevitable sequela of pulp necrosis, periapical inflammation, and external inflammatory root resorption.[4,8] Long-standing, untreated dental fractures may lead to severe sequelae, including chronic rhinitis and ocular disease.[9] Endodontic therapy is a viable treatment option for fractured canine teeth in cats, with a better prognosis when performed before external inflammatory root resorption is detected radiographically.[8] If endodontic therapy is not feasible, extraction is the only treatment that will eliminate the source of chronic inflammation and discomfort. Extraction of a fractured tooth with pulp exposure, even one without radiographically apparent periapical pathosis, is always a better choice than “watching and waiting,” particularly because external signs of endodontal disease are almost always absent in cats.

Although chronic gingivostomatitis is not common,[10] it can be very frustrating to treat. Extraction of all premolar and molar teeth, or even all teeth, is the currently recommended treatment for cats with chronic stomatitis, with approximately two-thirds of cats showing significant improvement or resolution of oral inflammation following extractions.[2,11-13]

Preoperative concerns

General health status

As discussed in previous chapters, patients with preexisting medical conditions may require special precautions to reduce the risk of infection and prevent exacerbation of the patient’s disease state. Intravenous antibiotics are indicated when immunosuppressive conditions are present or delayed wound healing may be expected. Special anesthetic support may be required for patients with cardiomyopathy or renal insufficiency. Gentle tissue handling is particularly important in diabetic cats, which may experience delayed wound healing. Hyperthyroid cats should be rendered euthyroid prior to pursuing elective procedures.

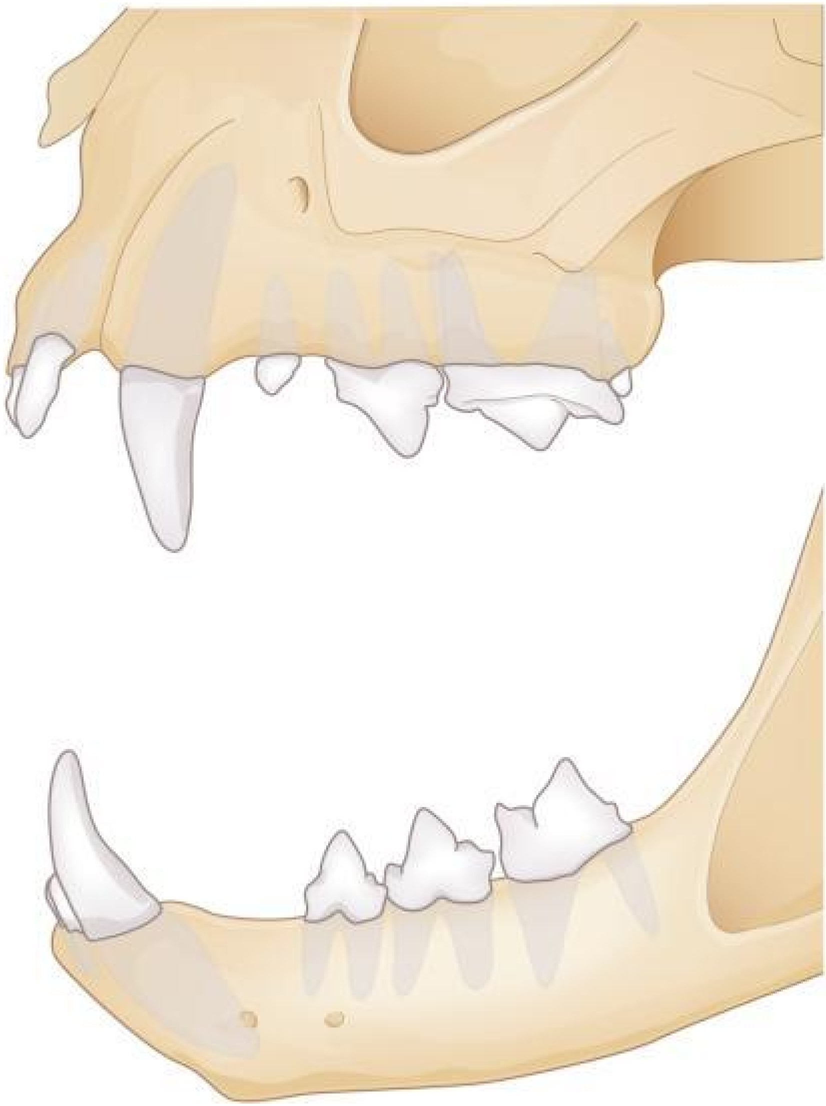
Contraindications for anesthesia and dental treatment include end-stage renal failure with severe uremia, uncontrolled diabetes with ketosis, severe leukopenia, or untreated lymphoma (nonfunctioning white blood cells may result in overwhelming infection), and severe cardiomyopathy.

Viral status

Cats infected with Feline Immunodeficiency Virus (FIV) and/or Feline Leukemia Virus (FeLV) are immunocompromised and should therefore receive intravenous antibiotics at the time of surgery and oral antibiotics afterwards. Extraction sites should be sutured whenever possible to facilitate healing.

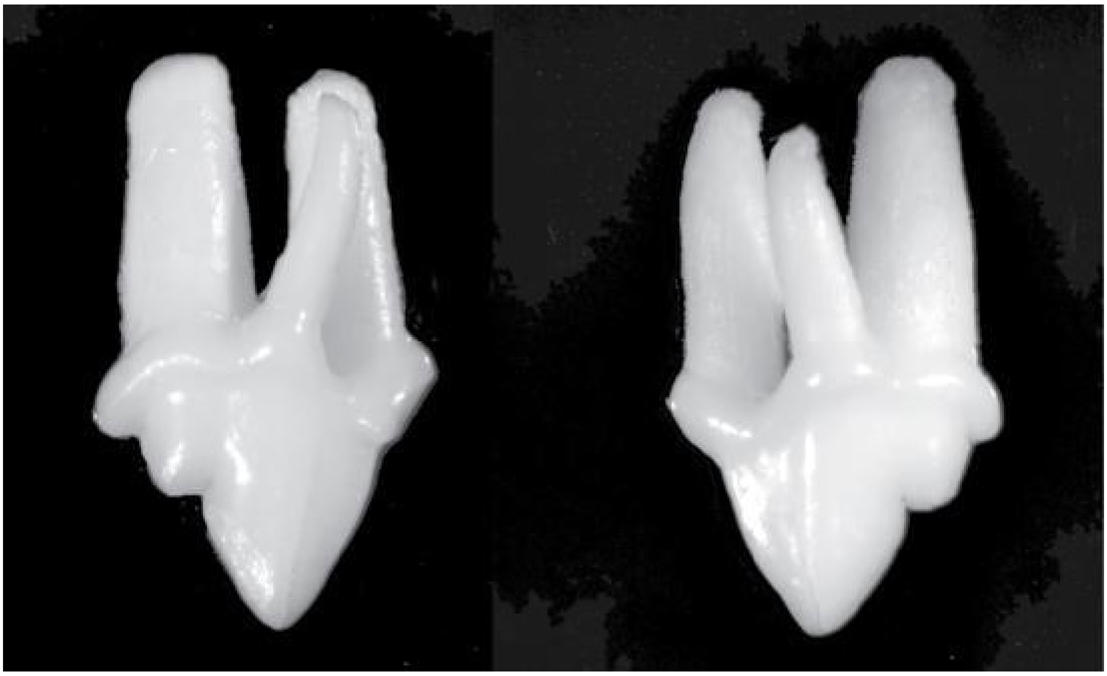
Surgical anatomy

Each incisive bone contains three incisor teeth, and each maxilla contains one canine tooth, three premolar teeth (P2, P3, P4), and a small first molar tooth. Correspondingly, there are three incisor teeth, one canine tooth, two premolar teeth (P3 and P4), and a single molar tooth (M1) in each mandible (Fig. 17.2).



• **FIG. 17.2** Dentition of the cat.

Incisor and canine teeth are single-rooted teeth. A cat's canine teeth, unlike a dog's, are usually not curved significantly, and may have a blunt apex. The incisor teeth are very slender and fragile. The maxillary second premolar tooth is the only single-rooted premolar tooth. However, anatomical variations in the root structure are common: in one study, the frequencies of a single root, a dichotomous root, and two roots were found to be 27.7%, 55.1%, and 9.2%, respectively (Fig. 14.3).[14] The maxillary third premolar tooth is two rooted, although a third root is present in approximately 10% of cases (Fig. 17.3).[14]



• **FIG. 17.3** Bilateral supernumerary roots of the maxillary third premolar tooth. Source: (From Verstraete FJM, Terpak CH. Anatomical variations in the dentition of the domestic cat. *J Vet Dent* 1997;14:137–140.)

The maxillary fourth premolar tooth has three roots. The distal root is much larger than the mesiobuccal and palatal roots, and the furcation is located more mesially rather than in the middle of the tooth. Although often classified as two rooted, the maxillary first molar tooth of the cat may also have a single root or two fused roots (Fig. 14.4). This tooth was found to have a single root in 35.0% of cases and dichotomous roots in 34.7%; only 28.0% of maxillary first molar teeth had two fully formed roots.[14] The mandibular third and fourth premolar teeth are similar in shape and size, and each has two slightly divergent roots. The mandibular first molar tooth has two divergent roots, with the distal root typically much more slender than the mesial root.

The maxillary teeth are separated from the nasal cavity by a relatively thin plate of bone, and it is possible to penetrate into the nasal cavity during attempts to extract maxillary canine and premolar teeth, particularly the palatal root of the fourth premolar tooth. In addition, the maxillary first molar tooth is located immediately ventral to the orbit; care must be taken not to penetrate through the bone into the orbit during extraction or administration of regional anesthetic agents.[15,16]

The roots of the mandibular premolar and molar teeth terminate just dorsal to the mandibular canal, which contains the inferior alveolar artery, vein, and nerve. If excessive force is applied during extraction, it is possible for an instrument or a root fragment to penetrate into the mandibular canal, which may result in severe hemorrhage. Management of a root that has been displaced into the mandibular canal is addressed in Chapter 18.

Special instruments and materials

Instruments suitable for feline exodontics

The examples listed in Table 17.1 represent the author's personal preference; many suitable alternatives are available. The use of magnification loupes is strongly recommended (see

Chapter 7).

TABLE 17.1

Instruments Suitable for Feline Exodontics

Instruments and Suture Material	Comments
Periosteal elevator	The rounded end of a P24G periosteal elevator (P24G; Hu-Friedy Mfg. Co., Chicago, IL) (see Fig. 7.8) is ideal for gently elevating mucogingival-periosteal flaps without tearing the tissues.
Bur	A very narrow, taper diamond bur (Maxima Diamond 850–010 C; Henry Schein, Inc. Melville, NY) is well suited to sectioning cat premolar teeth.
Luxators	Luxators (JS Dental Mfg. Co., Ridgefield, CT) (see Fig. 13.11B) with 1-, 2-, or 3-mm wide blades, straight handles, and sharp blades are used for luxation.
Elevators	An elevator (LTXS 2S and LTXS 3S; Cislak Manufacturing, Inc., Niles, IL) with a narrow tip and a shorter handle is better suited to cats than a large-handled, large-bladed elevator, which can generate excessive rotational force and result in root fracture.
Pedodontic extraction forceps	Small, straight-beaked forceps, such as a Cryer #150S or #150K (F150S and F150K; Hu-Friedy Mfg. Co., Chicago, IL) (see Fig. 13.15A) are suitable for most extractions.
Root tip elevators	Root tip elevators such as the Davis # 11 root tip teaser (ED11; Hu-Friedy Mfg. Co., Chicago, IL) (see Fig. 13.13B) are useful for teasing mobile root fragments from their alveoli; because the end is rounded, it is less likely to cause trauma or penetrate through the alveolus.
Root tip forceps	Root tip forceps, such as FX-49 forceps (FX49; Hu-Friedy Mfg. Co., Chicago, IL) (see Fig. 13.15B), with long, narrow beaks are excellent for retrieving fractured roots.
Small needle holders	Small (4.5" or 5") needle holders, such as a Halsey needle holder (NH-5036; Hu-Friedy Mfg. Co., Chicago, IL) (see Fig. 7.11A), are much easier to maneuver in the small mouth of a cat than the more common 6", 7", or 8" needle holders.
Small surgical curette	A curette, such as a Miller # 10 (CM10; Hu-Friedy Mfg. Co., Chicago, IL) (see Fig. 7.14), is used for removing debris from the alveolus after extraction.
Suture material	Absorbable monofilament suture material (Monocryl; Ethicon, Inc., Somerville, NJ) on a small, reverse-cutting needle, size 5-0, is suitable for cats.

Therapeutic decision-making

Preextraction radiographs of teeth to be removed should be obtained. This will allow evaluation of supporting bone levels or extent of periodontal bone loss, identification of root resorption or ankylosis, and assessment of root morphology.[2,17] As mentioned, anatomical variations are common in cats and, when present, will affect the extraction technique. Furthermore, radiography is particularly important when deciding whether to extract a tooth affected by resorption in toto or by coronectomy. The classification system outlined in Tables 17.2 and 17.3 has been developed by the American Veterinary Dental College and is helpful in determining the appropriate extraction technique.[18]

TABLE 17.2

Classification of Tooth Resorption Based on Clinical Stage

Stage	Definition
TR1	Mild dental hard tissue loss (cementum or cementum and enamel).
TR2	Moderate dental hard tissue loss (cementum or cementum and enamel with loss of dentin that does not extend to the pulp cavity).
TR3	Deep dental hard tissue loss (cementum or cementum and enamel with loss of dentin that extends to the pulp cavity); most of the tooth retains its integrity.
TR4	Extensive dental hard tissue loss (cementum or cementum and enamel with loss of dentin that extends to the pulp cavity); most of the tooth has lost its integrity. **TR4a:** Crown and root are equally affected. **TR4b:** Crown is more severely affected than the root. **TR4c:** Root is more severely affected than the crown.
TR5	Remnants of dental hard tissue are visible only as irregular radiopacities, and gingival covering is complete.

From American Veterinary Dental College Classification of Tooth Resorption. https://www.avdc.org/Nomenclature/Nomen-Teeth.html

TABLE 17.3

Classification of Tooth Resorption Based on Radiographic Appearance

Type	Definition
Type 1	Focal or multifocal radiolucency in a tooth with otherwise normal radiopacity and normal periodontal ligament space.
Type 2	Narrowing or disappearing of the periodontal ligament space in at least some areas, and decreased radiopacity of part of the tooth.
Type 3	Features of both type 1 and type 2 are present in the same tooth. A tooth with this appearance has areas of normal periodontal ligament space and areas of narrow or lost periodontal ligament space. There is focal or multifocal radiolucency in some areas of the tooth and decreased radiopacity in other areas.

From American Veterinary Dental College Classification of Tooth Resorption. https://www.avdc.org/Nomenclature/Nomen-Teeth.html

Extraction versus coronectomy (crown amputation with intentional root retention) for tooth resorption

Variable stages of root resorption result in a high risk of root fracture, making extraction of resorbing teeth especially difficult. Root fractures with retention of an intact apical fragment may result in persistent pain and complications such as osteomyelitis of the alveolar bone and periapical inflammation.[2,19] However, teeth affected by resorption only, in the absence of periodontal disease or preexisting fractures, are very rarely associated with pulpal necrosis or periapical pathology.[4,20] Therefore, coronectomy appears to be an acceptable alternative to extraction in selected cases.[4,6,7] Preextraction radiographs are crucial in case selection: only teeth with advanced root resorption, but without periodontal or endodontal lesions, are good candidates for coronectomy.[2,7]

This technique is especially useful for teeth with extensive resorption where the patient has a high risk of anesthetic complications and expeditious treatment is desirable. Coronectomy should not be performed on fractured teeth or teeth with significant periodontitis, and is not recommended in cats with gingivitis/stomatitis, except where extensive root resorption (stage 4c) is confirmed radiographically.

Postextraction radiographs are indicated in these cases, to document how much root structure was left behind and to confirm the smoothness of the alveolar margin.

Simple versus surgical extraction

The decision to extract a tooth in a simple or surgical fashion should be based on clinical and radiographic findings prior to beginning the extraction procedure (see Chapter 13). Unlike a surgical extraction, a simple extraction does not involve sectioning the tooth, creating a mucogingival flap, or removing alveolar bone.[17] A tooth with significant bone loss and clinical mobility is a good candidate for simple extraction, while a fractured canine tooth with normal alveolar bone levels would be an indication for surgical extraction.

The buccal alveolar bone at the canine teeth is normally up to 2 mm thick. Periodontitis and tooth resorption are often associated with alveolar osteomyelitis, resulting in increased width of this bone (clinically referred to as buccal bone expansion or alveolar bone expansion).[21,22] The presence of alveolar osteomyelitis may make a nonsurgical extraction of a canine tooth possible[22]; however, flap creation, partial alveolectomy, debridement, and alveoloplasty are indicated to allow soft tissue closure over a flat, smooth surface. Following removal of the affected alveolar bone, the ipsilateral mandibular canine tooth may occlude onto the lip.[2] It may therefore be indicated to perform odontoplasty and application of a light-cured dentinal sealer to reduce the height and sharpness of the cusp tip of the ipsilateral mandibular canine tooth to prevent occlusal trauma to the lip following extraction of a maxillary canine tooth.

Flap design

The three primary mucoperiosteal flap designs (envelope, triangle, and pedicle—see Chapter 13) are used for surgical extractions in cats. An envelope flap (which is a gingival flap, as the alveolar mucosa is typically not included) is used for coronectomy of premolar and molar teeth, while a triangle flap allows for tension-free closure following coronectomy of canine teeth. An extended envelope flap, which allows elevation of alveolar mucosa without an alveolar mucosal incision, may be used for the extraction of all premolar and molar teeth in one quadrant.

Full-mouth and premolar–molar extractions

When extensive extractions are planned for treatment of chronic gingivostomatitis, several factors must be taken into consideration. In many cases, the inflammation is less severe at the incisor and canine teeth, so premolar–molar extractions may be elected, with planned conservation of the incisor and canine teeth. Survey dental radiographs prior to extraction are indicated for the reasons mentioned above; they are also useful for determining whether the incisor and canine teeth require extraction for reasons not related to gingival and mucosal inflammation (e.g., the presence of significant periodontal bone loss or tooth resorption). For cases with severe generalized stomatitis, full-mouth extractions may be indicated. In these situations, it may be desirable to stage the procedure, extracting all teeth on one side of the mouth under one anesthetic episode, then completing extractions of the remaining two quadrants 2–3 weeks later. All root fragments must be removed.[23] Pain management for patients undergoing full-mouth or premolar–molar extractions is imperative.[2,23] In addition, nutritional support via a feeding tube (nasoesophageal, pharyngostomy, esophagostomy, or percutaneous gastrostomy tube) should be considered prior to surgery, particularly for patients who are debilitated (see Chapters 4 and 5).[2,23]

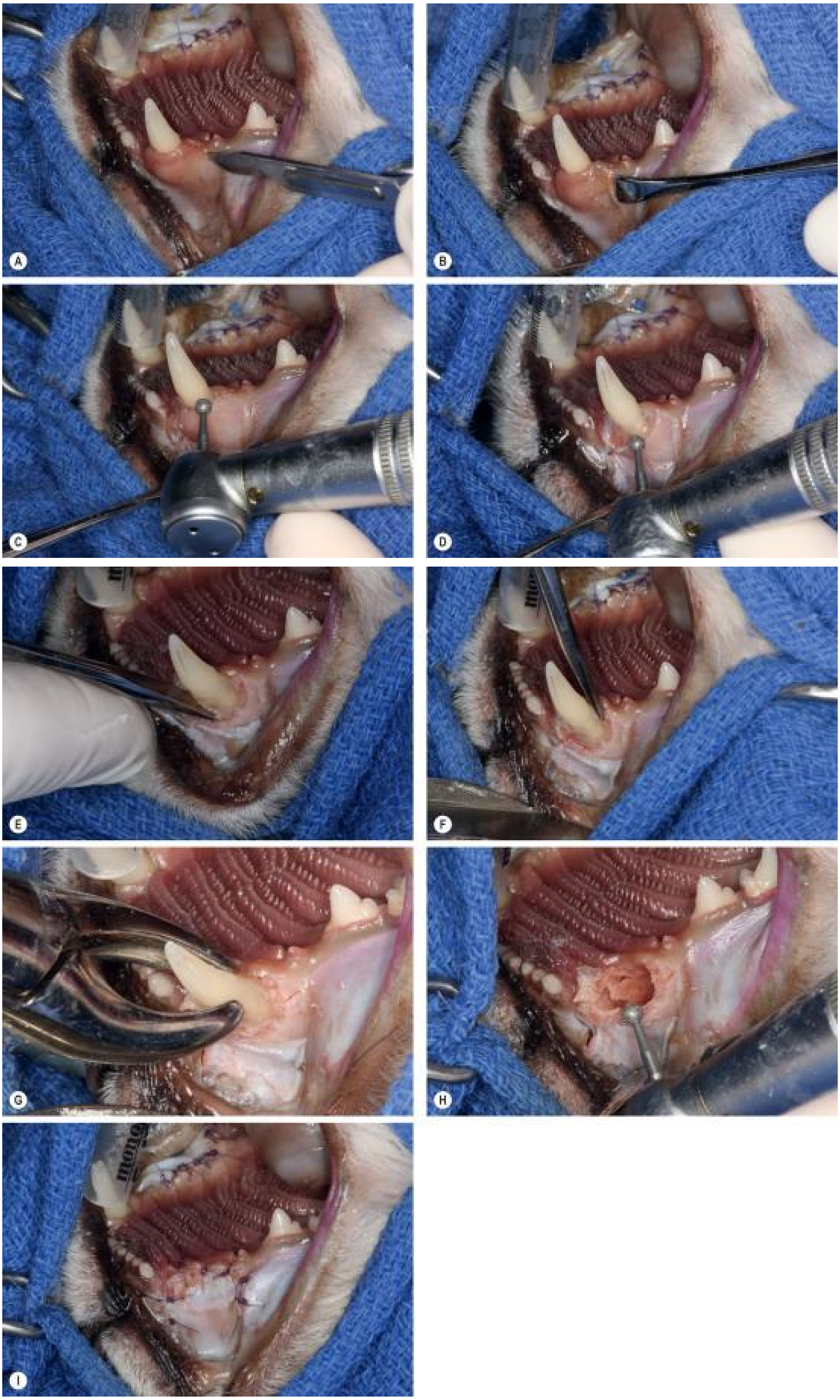
Surgical technique

Extraction of incisor teeth

After incising the gingival epithelial attachment by inserting a scalpel blade into the gingival sulcus circumferentially around the tooth, a 1- or 2-mm luxator is gently inserted into the periodontal space on the mesial side of the incisor tooth. The luxator is advanced using gentle pressure, cutting the periodontal fibers to within two-thirds of the length of the root. The luxator is then applied in a similar fashion to the distal aspect of the root. The tooth should now be dislodged but, if necessary, the process may be repeated on the lingual side or until the entire root circumference is loosened from its periodontal attachment. Completely severing the periodontal attachment minimizes the risk of root fracture and often allows the tooth to be delivered out of the alveolus without the use of forceps.[2] If this is not the case, a root tip elevator, rather than forceps, should be used to elevate the loosened tooth from the alveolus. Unlike the elevation technique, the luxation technique does not use the adjacent alveolar bone or tooth as a fulcrum to lever out the tooth, an important factor considering the proximity of the incisors to one another and the potential for damage to an adjacent tooth during extraction. Suturing is rarely required unless a number of adjacent incisor teeth have been extracted.

Extraction of maxillary canine teeth

After incising the gingival epithelial attachment circumferentially around the tooth and extending the incisions 2–3 mm interproximally, diverging vertical releasing incisions are made through the buccal gingiva and alveolar mucosa at the mesial and distal aspects of the tooth (Fig. 17.4A). A full-thickness mucogingival pedicle flap is elevated (Fig. 17.4B). The flap should be of sufficient width to allow for the suture line to rest over solid bone and not over the vacated alveolus. A round bur on a high-speed dental handpiece is used to remove buccal alveolar bone as needed (Fig. 17.4C–D). A 2- or 3-mm luxator (Fig. 17.4E–F) is introduced on the mesial and distal aspects of the tooth, using firm pressure and small axial rotational movements to direct the blade apically in the periodontal space, severing the periodontal ligament fibers to within about two-thirds the length of the root.[2] Placing the blade on the buccal or palatal aspects of the tooth should be avoided, as slippage may easily occur, resulting in laceration of the soft tissues or penetration into the nasal cavity. After sufficient mobility has been achieved, extraction forceps are placed as far apically as possible on the root (Fig. 17.4G), and gentle rotational and tractional forces are applied to deliver the tooth from the alveolus. If indicated, the alveolus is cleaned of debris with a small surgical curette and flushed. The alveolar margin should be smoothed with a round bur on a high-speed handpiece (Fig. 17.4H), and, if buccal bone expansion is present, additional alveolectomy may be required in order to suture the gingiva without tension (Fig. 17.4I).



• **FIG. 17.4** Technique for extraction of a maxillary canine tooth, demonstrated on a cadaver specimen. (**A**) A pedicle flap is created with a # 15 scalpel blade and (**B**) elevated with a periosteal elevator (**C**) to expose the buccal alveolar bone. (**D**) Buccal alveolectomy is performed using a round diamond bur on a high-speed handpiece. The luxator is placed on the (**E**) mesial and (**F**) distal aspects of the tooth, severing the periodontal ligament. (**G**) When the tooth is mobile, extraction forceps are placed as far apically as possible and gentle rotational forces are applied until the tooth is delivered from the alveolus. (**H**) Alveoloplasty is performed using a round diamond bur on a high-speed handpiece and is followed by wound lavage to remove loose bone particles and debris. (**I**) The flap is sutured with absorbable monofilament suture material in a simple interrupted pattern.

After extraction of a maxillary canine tooth, it is important to verify whether a communication with the nasal cavity is present, either due to severe periodontal bone loss or avulsion of part of the alveolar process during extraction.[2] If a communication is present, special care must be taken to ensure complete closure of the extraction site. This may require removal of additional buccal bone, particularly if buccal bone expansion is present. Following removal of buccal bone and extraction of a maxillary canine tooth, entrapment of the upper lip by the ipsilateral mandibular canine tooth may occur.[2] To minimize lip trauma, odontoplasty and application of a light-cured dentinal sealer may be performed to reduce the crown height of the mandibular canine tooth by 1–2 mm. Preoperative occlusal and lateral radiographs are helpful in determining the location of the pulp cavity in relation to the cusp tip; care must be taken not to expose the pulp during odontoplasty.

Extraction of mandibular canine teeth

After incising the gingival epithelial attachment circumferentially around the tooth, the incision is extended 2–3 mm mesially, and a vertical releasing incision is created at the mesial–buccal line angle of the canine tooth. Rather than creating a distal vertical releasing incision, which would transect the frenulum, an interproximal incision is made on the alveolar margin up to the mesial aspect of the third premolar tooth; this flap design keeps the lower lip frenulum intact (see Chapter 15). Partial buccal alveolectomy is performed as needed, then a 2- or 3-mm luxator is introduced on the mesial and distal aspects of the tooth. Using firm pressure and small axial rotational movements, the blade of the luxator is directed apically in the periodontal space, severing the periodontal ligament fibers to within about two-thirds the length of the root.[2] Placement of the blade on the buccal or lingual aspects of the tooth should be avoided, as this can easily result in instrument slippage and subsequent damage to the buccal or sublingual soft tissues. As with maxillary canine teeth, extraction forceps are placed as far apically as possible on the root after sufficient mobility has been achieved through luxation. Premature application of extraction forceps and excessive rotational forces may lead to fracture through the alveolar process on the lingual aspect of the tooth (see Fig. 18.5), resulting in instability between the mandible and the mandibular symphysis. Therefore, it is prudent to continue using a luxation technique and not to apply the extraction forceps until the canine tooth is mobile enough to remove with only mild tractional forces and minimal rotational forces.

Extensive removal of buccal alveolar bone may facilitate extraction of a canine tooth with evidence of ankylosis. A lingual approach for extraction of mandibular canine teeth has also been described, but was considered less applicable for cats than dogs, based on anatomical differences.[24] A dorsal approach specific for cat mandibular canine teeth has recently been described, involving a single, full-thickness incision in the gingiva of the dorsal alveolar margin overlying the canine tooth root, elevation of the periosteum on both lingual and buccal aspects, and removal of dorsal alveolar bone. This technique was not found to be faster than a standard buccal approach, and analysis with computed tomography revealed a tendency toward more bone loss with the dorsal approach, so a dorsal approach did not appear to offer any advantages over a buccal approach.[25]

Extraction of premolar and molar teeth

The maxillary second premolar and first molar teeth are usually extracted in a nonsurgical fashion, using the luxation technique described above. Because the remaining premolar teeth and the mandibular molar teeth are multirooted, however, a surgical approach is typically indicated, unless severe alveolar bone loss is present, in which case the tooth may be removed intact after its remaining periodontal ligament has been severed. For extraction of a single two-rooted or three-rooted premolar tooth or the mandibular molar tooth, a triangle mucogingival flap, with a vertical releasing incision at the mesial interproximal space, is most useful. When multiple adjacent teeth are being extracted, an envelope flap typically provides adequate visualization and exposure of the buccal alveolar bone. If necessary, 1–3 mm of bone may be removed from the alveolar margin to expose the furcations prior to sectioning. A narrow, tapered bur on a high-speed handpiece is used to section the teeth, beginning at the furcation and progressing coronally, so each root may be separately extracted.

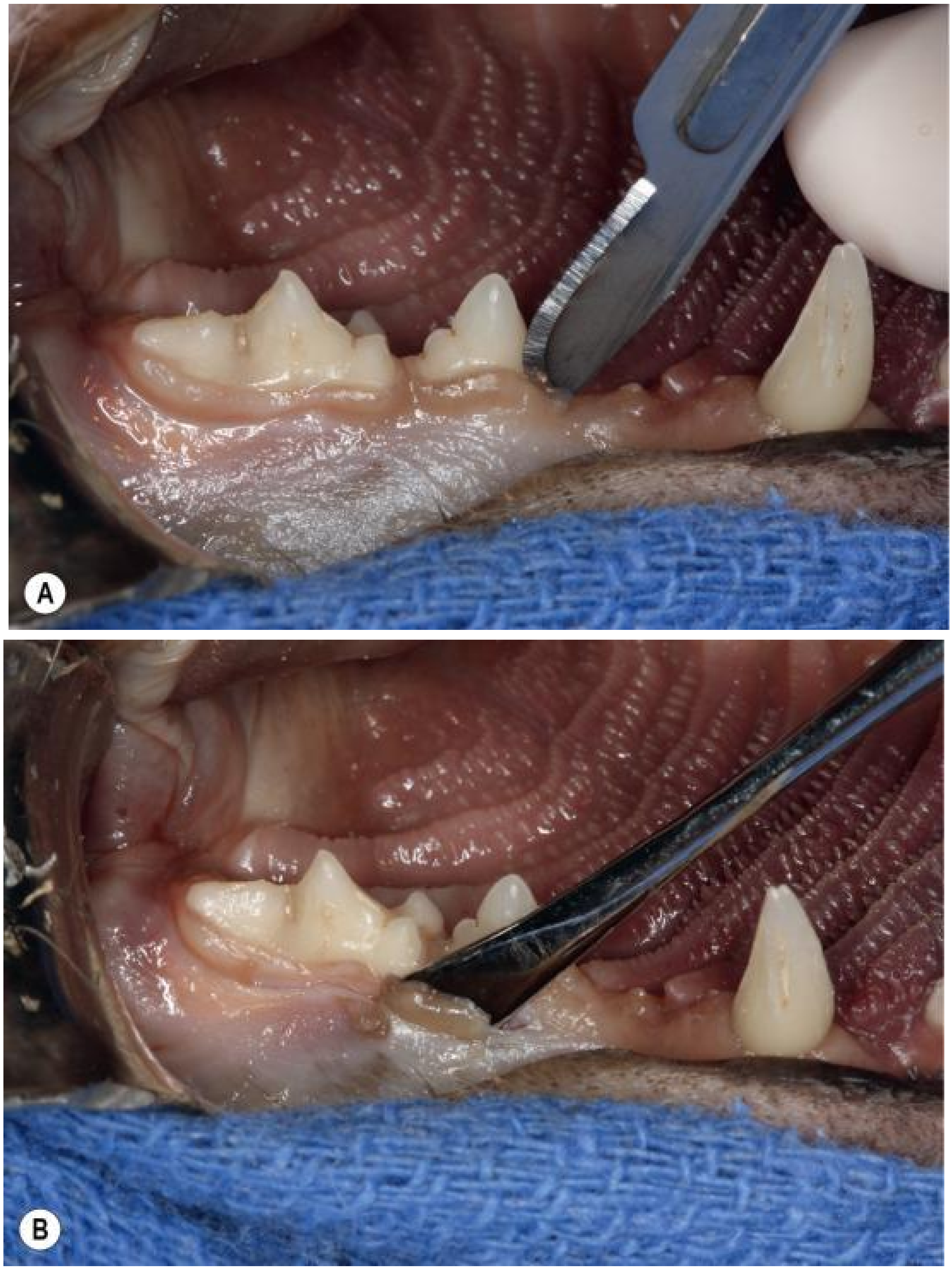
A 2- or 3-mm luxator or luxating elevator is introduced on the mesial and distal aspects of each root, using gentle pressure and small axial rotations (5–10 degrees) to direct the blade apically in the periodontal space, cutting the periodontal attachment.[2] If desired, once some mobility has been achieved, the elevator may be placed horizontally between sectioned roots and gentle rotational pressure applied to fatigue the periodontal ligament and lift the root from the alveolus. When using the elevation technique, root fracture will occur unless extreme caution is exercised to avoid generating excessive force.

For the maxillary fourth premolar tooth, a narrow, tapered diamond bur is used perpendicular to the vertical axis of the tooth, beginning at the furcation and moving coronally, to separate the distal root from the mesiobuccal root. Some veterinarians prefer to then remove the distal root to provide better visualization when sectioning the mesiobuccal root from the palatal root, which is achieved by inserting a narrow, tapered diamond bur into the mesial furcation, at an approximately 30-degree angle to the vertical axis of the tooth (see Fig 16.6). A 2-mm luxator is introduced into the periodontal ligament space on the mesial and distal aspects of the mesiobuccal root, and, if needed, extraction forceps are employed to remove the root after sufficient mobility has been achieved. Following extraction of the mesiobuccal and distal roots, the palatal root is more easily visualized, allowing access with a luxator or elevator. Additional alveolectomy may be required for optimum access to the palatal root.

For the mandibular first molar tooth, the proximity of the fourth premolar tooth may prevent adequate access to the mesial aspect of the mesial root. The luxator may be inserted into the periodontal ligament space along the mesial aspect of the mesial root in a horizontal rather than vertical manner to minimize risk of damaging the fourth premolar tooth.

Premolar–molar and full-mouth extractions

When extracting all premolar and molar teeth in a quadrant, an extended envelope flap provides sufficient exposure for good visualization of the underlying teeth and alveolar bone (Fig. 17.5A–B).[2] If indicated, some of the bone at the alveolar margin may be removed, using a round bur on a high-speed handpiece, to expose the furcation region of multirooted teeth (Fig. 17.5C). A tapered bur is then used to section multirooted teeth into single roots (Fig. 17.5D–E), each of which subsequently may be more easily removed using a combination of luxation and elevation on the mesial and distal aspects of each root (Fig. 17.5F–G). The luxator may be placed vertically (F) or horizontally (G) into the periodontal ligament space to sever the periodontal ligament fibers. However, if an elevator is placed between the sectioned fragments of a multirooted tooth (i.e., in the furcation), excessive rotational forces will result in root fracture. When using horizontal leverage, very gentle forces should be applied. Once each root is sufficiently mobile, extraction forceps are placed as far apically on the root as possible, and very gentle rotational and tractional forces are applied (Fig. 17.5H). The palatal root of the maxillary fourth premolar tooth is more easily visualized after the mesiobuccal and distal roots have been removed. If necessary, additional alveolectomy may be performed to improve visibility of the palatal root and allow proper placement of the luxator blade into the periodontal ligament space (Fig. 17.5I). Following removal, each root tip should be carefully inspected. An intact root tip is rounded and usually has soft tissue attached to it at the apex. If there is any doubt as to whether the entire root has been removed, a dental radiograph should be made to ascertain whether a fragment remains. Unless end-stage root resorption is present, all root fragments must be removed, or inflammation may persist in bone[19] or soft tissues.[26]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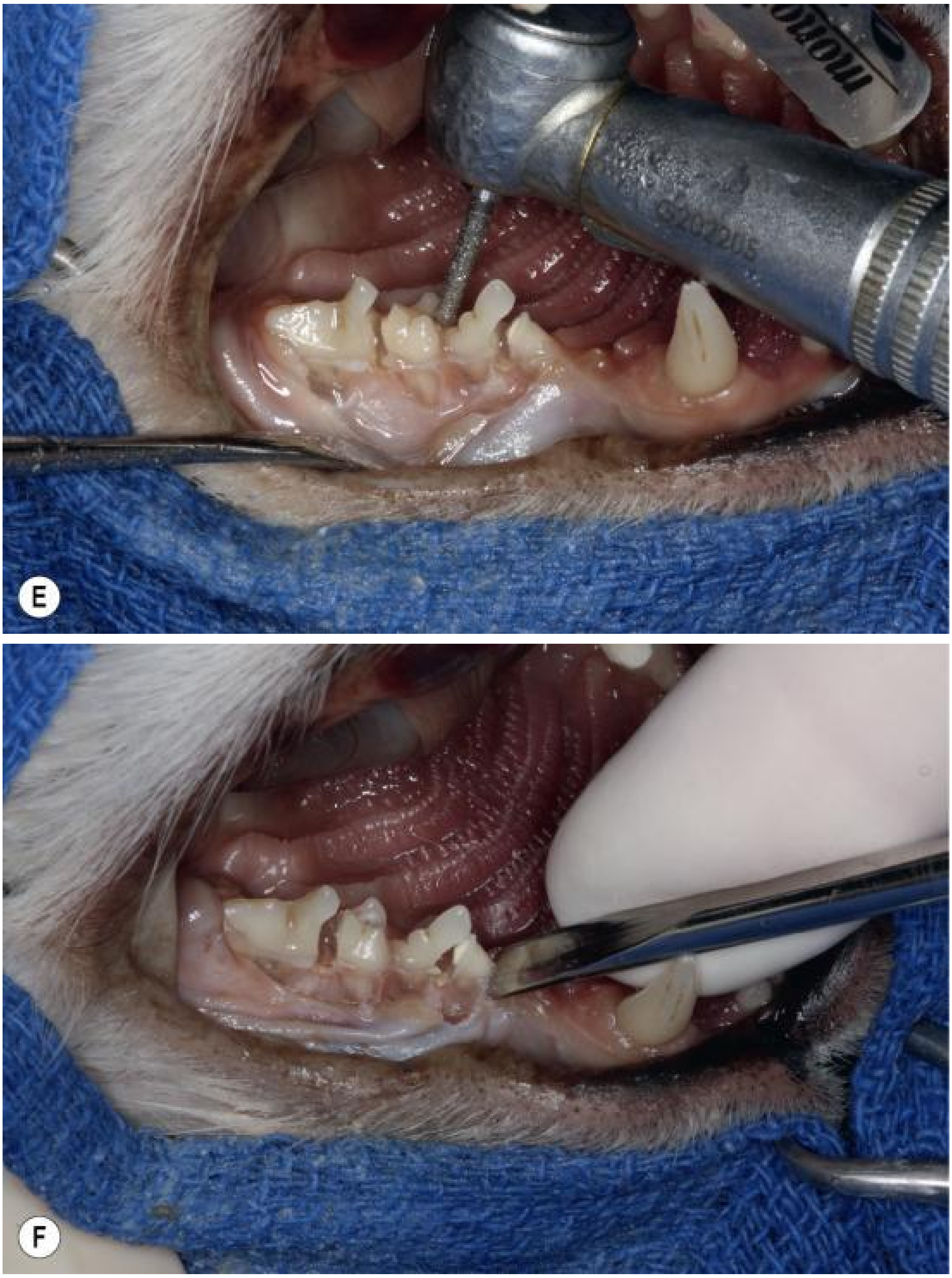
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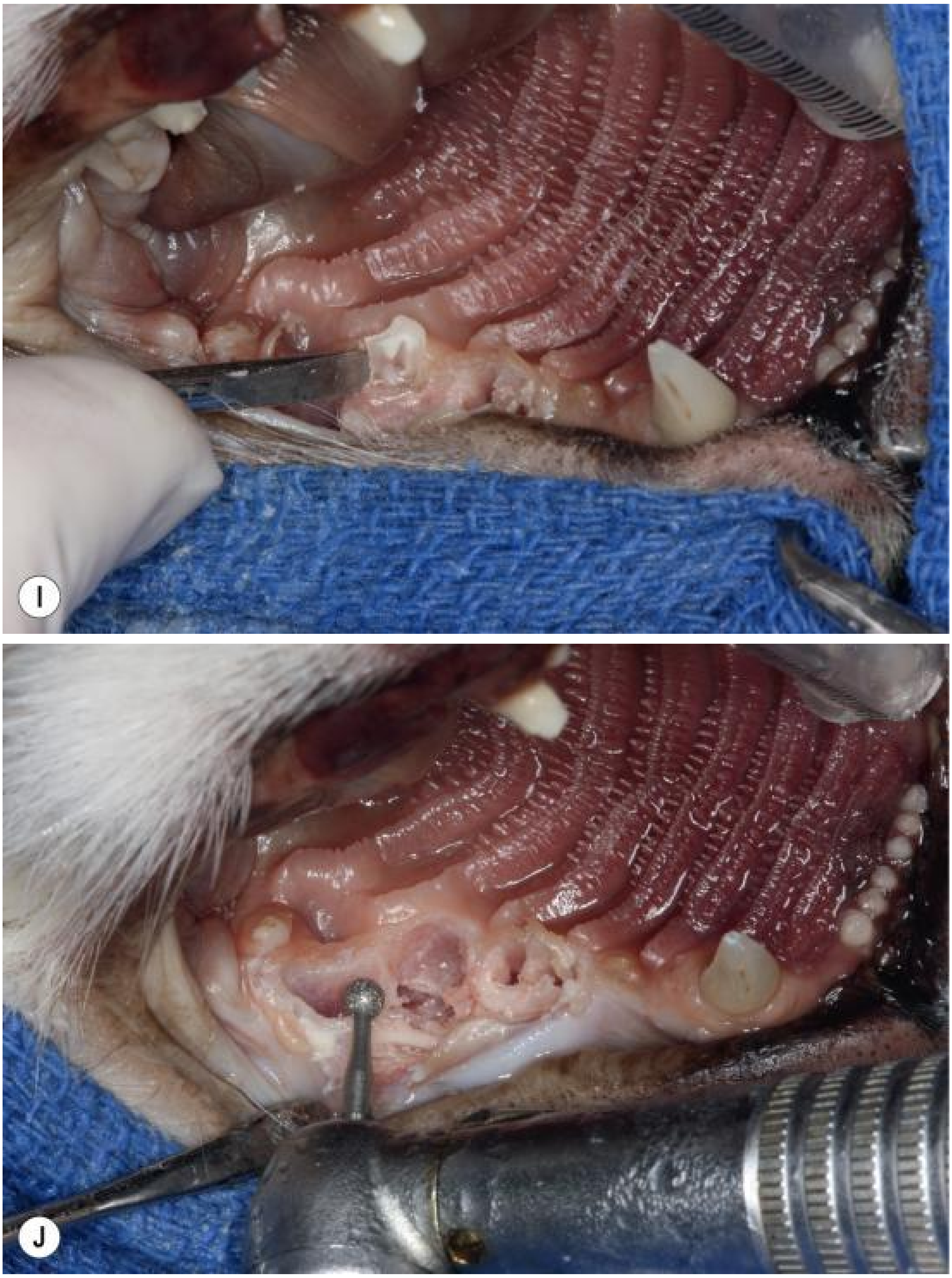
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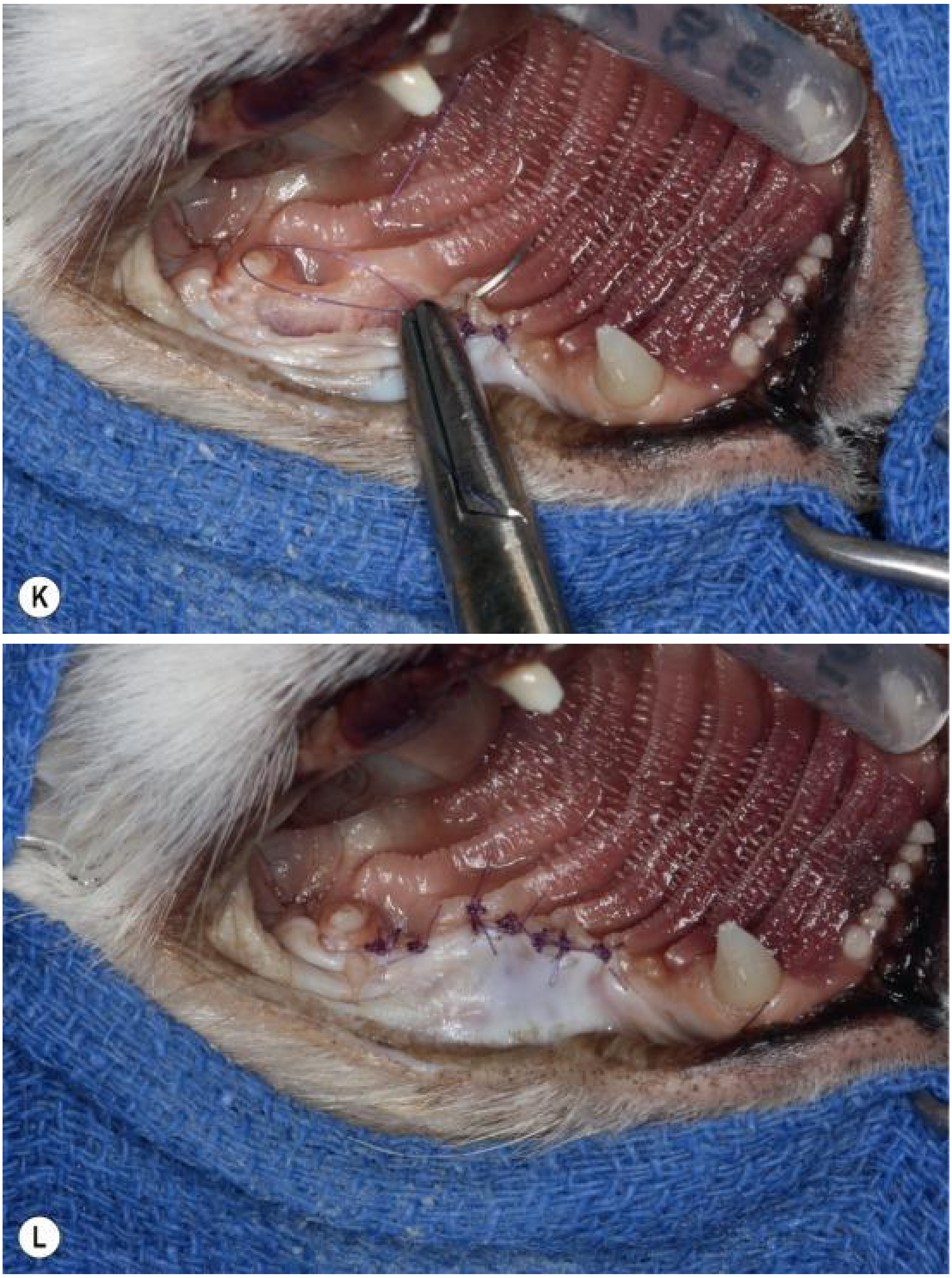


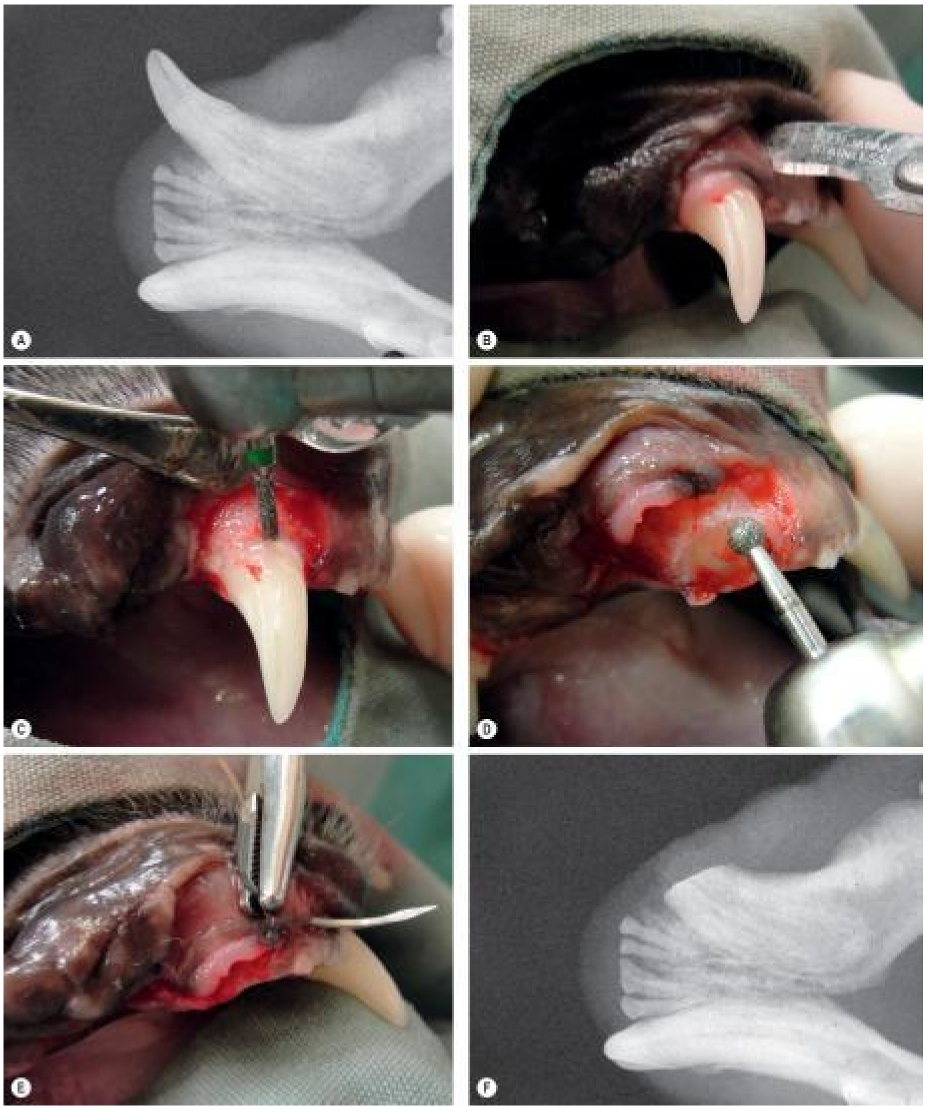
• **FIG. 17.5** Technique for extraction of the left maxillary third and fourth premolar teeth demonstrated on a cadaver specimen. (**A**) An envelope flap is created by inserting the blade into the gingival sulcus, beginning mesial to P3 and extending distally to P4. (**B**) A periosteal elevator is employed to elevate the full-thickness mucogingival flap from the underlying bone. (**C**) Alveolectomy is performed using a round diamond bur on a high-speed handpiece. (**D**) The teeth are sectioned using a tapered diamond bur on a high-speed handpiece. (**E**) The palatal root is separated from the mesiobuccal root using the same tapered diamond bur held almost vertically, at 30 degree angle to the palatal root. (**F, G**) Luxators are placed on the mesial and distal aspects of each root to sever the periodontal ligament. Luxators may be used either parallel (**F**) or perpendicular (**G**) to the root. (**H**) Extraction forceps are placed as far apically as possible on the roots, and gentle tractional and rotation forces are applied until each root is delivered from its alveolus. (**I**) The palatal root of the fourth premolar tooth is more easily visualized following removal of the mesiobuccal and distal roots. Additional alveolectomy will allow optimum visualization for proper placement of the luxator into the periodontal ligament space around the palatal root. (**J**) After all roots have been removed, alveoloplasty is performed with a round diamond bur on a high-speed handpiece to remove any sharp bony edges. (**K**) When suturing the flap, the needle should be passed entirely through the buccal gingiva prior to entering the palatal gingiva. This will minimize tearing of the gingiva. (**L**) The envelope flap has been closed without tension using absorbable monofilament suture material in a simple interrupted pattern.

If a root fracture has occurred, it is important to have good lighting and visibility before attempting removal of the remaining fragment. If fracture occurred close to the cementoenamel junction and the remaining fragment is large, a 1-mm or 2-mm dental luxator or small elevator may be sufficient to loosen and remove it. Small root tips may be mobilized using root tip picks or root tip elevators, then removed with root tip forceps. It may be necessary to extend the mucogingival flap and remove more alveolar bone in order to obtain access to a small root fragment. Attempting to drill out root tips (sometimes referred to as "pulverization" or "atomization") can result in damage to the infraorbital or inferior alveolar arteries and nerves, fatal air embolism,[27] or other iatrogenic trauma, and this technique is not recommended.

After all roots and root fragments are removed, any expanded alveolar bone is gently compressed back into position, and sharp bony edges are reduced with a round bur (Fig. 17.5J). The empty alveoli are lavaged, and the mucogingival flap is repositioned and sutured without tension (Fig. 17.5K–L).[2] Feline gingiva and mucosa may be quite friable; use of fine suture material and gentle tissue handling are recommended.

Coronectomy (crown amputation with intentional root retention)

Preoperative radiographs (Fig. 17.6A) are essential in determining whether a tooth is a good candidate for coronectomy. A gingival or mucogingival flap is elevated (Fig. 17.6B); for premolar and molar teeth, a small envelope flap is suitable. To achieve tension-free closure following coronectomy of a canine tooth, a triangle flap is indicated. Amputation of the crown is performed at the level of the alveolar margin, using a taper or round bur on a high-speed handpiece (Fig. 17.6C). Any sharp bony edges are removed with a round bur on a high-speed handpiece (Fig. 17.6D), the surgical site is irrigated, and the gingiva is sutured over the remaining roots (Fig. 17.6E).[7] Postextraction radiographs (Fig. 17.6F) should routinely be obtained in these cases.



• FIG. 17.6 Technique for coronectomy of a resorbing tooth. (**A**) A lateral radiograph of this left mandibular canine tooth confirms type-2 resorption and no evidence of periodontitis or endodontic disease, making this tooth a good candidate for coronectomy. (**B**) With the patient in dorsal recumbency, a triangle flap is created and elevated. (**C**) Using a tissue retractor to prevent trauma to the flap, a tapered diamond bur is used to amputate the crown at the level of the alveolar margin. (**D**) A small round diamond bur is used to perform alveoloplasty. (**E**) Closure with absorbable monofilament suture material in a simple interrupted pattern. (**F**) A postoperative radiograph confirms that there are no sharp edges of the tooth and documents the condition of the root following the procedure.

Postoperative care and assessment

Pain management

Preemptive analgesia, with use of a balanced anesthesia technique and local or regional anesthesia, will help ensure a more comfortable postoperative period for patients undergoing extractions (see Chapter 4). For pain management at home following routine extractions, buprenorphine may be dispensed. Oral transmucosal (OTM) administration of buprenorphine is relatively easy for clients to perform and well tolerated by cats, but differences in oral pH and swallowing by some cats may affect absorption, and plasma concentrations may not be as predictable after OTM administration as was previously reported.[28,29] Subcutaneous (SC)administration of a high-concentration formulation of buprenorphine (Simbadol; Zoetis, Inc., Parsnippany, NJ) at 0.12–0.24 mg/kg one hour prior to cessation of anesthesia is an option to provide up to 48 hours of postoperative analgesia, though duration of effect is individually variable and therefore somewhat unpredictable.[30] It should be noted that IV or OTM administration of high-concentration buprenorphine resulted in significantly shorter duration of the antinociceptive effect than SC administration,[31] and these routes of administration are not recommended for this formulation.

Fentanyl patches are still a popular choice for postoperative analgesia in cats having more extensive oral surgery, such as full-mouth extractions or maxillofacial fracture repair. However, although blood levels of fentanyl may persist for up to 5 days,[32] there is some controversy regarding the efficacy of fentanyl patches for postoperative pain management following onychectomy.[33-35] Because OTM buprenorphine provides more predictable pain control, the author reserves use of fentanyl patches for those cats who will not allow handling for administration of OTM buprenorphine. If transdermal fentanyl is selected for postoperative analgesia, the patch should be placed 6 to 24 hours prior to surgery, which may also reduce the amount of inhalant required during surgery.[36] Because peak blood levels of fentanyl are not achieved until 6 hours after placement in cats,[37] injectable opioids (pure agonists rather than mixed agonist–antagonists) should be administered postoperatively in the hospital if the patch is placed at the time of surgery (see Chapter 4).

A combination of opiate and nonsteroidal antiinflammatory drugs (NSAIDs) may provide more effective analgesia than either agent alone.[29,38] Robenacoxib is COX-2 selective (compared with meloxicam, which inhibits both COX-1 and COX-2 isoforms),[39] has a neutral effect on glomerular filtration rate in healthy cats,[40] and is effective for controlling postoperative pain when given by SC injection[41] or orally.[42] Oral administration is well tolerated, and was not associated with any toxicologically significant events when compared with placebo.[43]

Nutritional support

Most cats will eat readily following dental extractions, provided excessive tissue trauma has not occurred and analgesia has been adequate. For patients with maxillofacial trauma, or debilitated patients with severe oral inflammation and multiple anticipated extractions, a feeding tube (nasoesophageal, pharyngostomy, esophagostomy, or percutaneous gastrostomy tube) should be placed at the time of the procedure to ensure sufficient caloric intake following surgery (see Chapter 5).

Postoperative assessment

The client should be instructed to observe the patient for lethargy, inappetence, or halitosis, and the patient should be examined 1–2 weeks following the procedure to evaluate healing. For long-term assessment of resorbing teeth on which coronectomy was performed, radiographs should be obtained when the patient is anesthetized for future dental care.

Complications

Complications for extractions are discussed in Chapter 18. Common complications in the cat include root fractures and trauma to adjacent soft and hard tissues. If extraction forceps are employed prematurely or with excessive force, sections of the buccal or lingual alveolar bone may be inadvertently removed with the extracted tooth. If this occurs when extracting a maxillary canine tooth, a communication between the nasal and oral cavities results, which can lead to permanent oronasal fistula formation if proper flap closure is not achieved. Less common complications include dislodging root fragments into the mandibular canal or nasal passages, fracture through the lingual alveolus of a mandibular canine resulting in mandibular symphyseal instability, and orbital penetration.[15] Most complications can be avoided with proper instrumentation and careful technique.

A common postoperative complication is maxillary lip entrapment by the mandibular canine tooth following removal of expanded buccal bone to facilitate closure of a maxillary canine tooth extraction site (Fig. 17.7). As previously mentioned, this can be prevented by removing 1–2 mm of the cusp tip of the mandibular canine tooth.

• **FIG. 17.7** Entrapment of the upper lip by the left mandibular canine tooth following extraction of the left maxillary canine tooth.

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CHAPTER 18

Complications of extractions

Milinda J. Lommer

Definitions

Perioperative complications: undesirable situations that occur during the surgical procedure
Postoperative complications: problems arising after completion of the surgical procedure

Prevention of complications

Common sense dictates that the best and easiest way to manage a complication is to prevent it from happening. Thorough preoperative assessment, treatment planning based on intraoral radiographs, advanced imaging as needed, and realistic evaluation of the operator's skill level will reduce the frequency of unexpected events and undesirable outcomes.[1]

Prior to planning a dental procedure, a thorough review of the patient's medical history and complete physical examination are important. Preoperative diagnostic tests including complete blood count, serum biochemical profile, retroviral testing (in cats), urinalysis, and thoracic radiography are performed as indicated. In addition to reducing the likelihood of anesthesia-related complications (see Chapter 4), comprehensive evaluation of the patient's systemic health prior to the procedure will allow better surgical planning. Patients with systemic diseases, particularly those that cause immunosuppression, may be more likely to experience delayed wound healing (see Chapter 1).

One of the primary ways to prevent surgical complications is to obtain adequate radiographs, including the apices of the roots of teeth to be extracted and the local and regional anatomic structures such as the mandibular canal and maxillary recess.[1] Presence of supernumerary roots, dilacerated roots, or other abnormalities may alter the treatment plan, and the operator may consider a surgical approach rather than nonsurgical extraction in these situations.

It is important for a veterinarian to recognize the extent of his or her training and ability, and not to attempt a surgery that is beyond his or her capability. The veterinarian should recognize that referral to a specialist is warranted if the anticipated procedure is beyond his or her own skill level. Failure to do so will result in suboptimal patient care and is therefore not in the patient's best interest.

Finally, following basic surgical principles such as clear visualization and access to the operative field will help keep complications to a minimum.[1] Clear visualization is achieved with adequate lighting, sufficient soft tissue reflection, and hemostasis. The use of magnification loupes is extremely helpful and recommended. Gentle tissue handling and aseptic technique will reduce the risk of delayed wound healing.

Perioperative complications

Hemorrhage

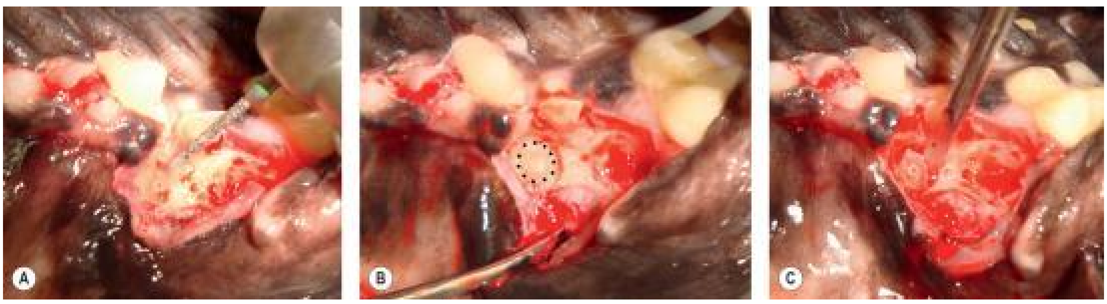
Hemorrhage is expected with any oral surgery, but excessive hemorrhage may occur due to injury to the infraorbital, inferior alveolar, or major palatine arteries. If compression with sterile gauze is insufficient to achieve hemostasis, larger vessels should be ligated with absorbable suture material. In addition, suturing the wound will mechanically obstruct the severed end of the bleeding vessel.[2]

Excessive hemorrhage may occur in patients with clotting disorders such as von Willebrand disease or those receiving fish oil supplements. Fish oil is high in eicosapentaenoic acid, which decreases platelet aggregation and may result in prolonged clotting times.[3-5] Though recent research suggests that coagulation indices are not affected,[6,7] in order to minimize the chance of excessive hemorrhage, it may be advisable to discontinue fish oil supplementation for 2 weeks prior to elective oral surgery. Whole blood or plasma transfusion should be considered for patients with decreased platelet function or hereditary clotting disorders. A thorough evaluation of the patient's medical history and preoperative diagnostic testing will help reduce unexpected bleeding complications in this group of patients.

Root fracture

Root fractures during extraction attempts are a common complication, particularly when an elevation rather than a luxation technique is employed for teeth with resorption or ankylosis, when a multirooted tooth is not sectioned, or when elevation is attempted with insufficient bone removal (alveolectomy). In one retrospective, radiographic study including 74 dogs and 42 cats that had previously undergone extraction of maxillary fourth premolar or mandibular first molar teeth, more than 82% of dogs and nearly 93% of cats were found to have unintentionally retained roots, and of those patients more than half showed evidence of periapical inflammation associated with the retained roots.[8]

If a root fractures during an extraction attempt, it is often, but not always, apparent to the operator based on auditory and tactile stimuli: a cracking sound is typically heard and, when the tooth is removed, the root end is sharp and jagged rather than smooth and rounded.[5] If the fracture occurs toward the coronal aspect of the root, visibility may be sufficient to insert a 1.3-mm "luxating elevator" (Cislak Manufacturing, Inc., Niles, IL) or 1- or 2-mm luxator (JS Dental Manufacturing, Inc., Ridgefield, CT) into the periodontal ligament space and continue the extraction attempt. However, if the root fracture occurs deep within the alveolus, visibility and the ability to place an instrument into the periodontal ligament space are significantly diminished. In these situations, radiography is very helpful in determining the location, size, and morphology of the root fragment. "Blind" attempts to extract a root fragment that cannot be visualized will cause significant damage to the surrounding bone and may result in dislodgement of the root fragment into the mandibular canal, nasal cavity, or maxillary sinus. Instead, additional bone removal should be performed; a combination of alveolectomy and judicious use of the air–water syringe and suction will greatly improve visibility and access.[9] The use of magnification loupes is also extremely helpful in recognizing the root–bone interface. Once the root is visualized, a very small luxator such as a 1.3-mm luxating elevator (Cislak Manufacturing, Inc., Niles, IL), a 1-mm luxator or a 2-mm luxator (JS Dental Manufacturing, Inc., Ridgefield, CT) may be inserted into the periodontal ligament space to sever the remaining attachment and allow retrieval of the fragment. For more difficult cases, such as ankylosed roots, in which it is impossible to insert a luxator into the periodontal ligament space, extraction tips on a piezosurgery unit (see Ch. 9) or a small round (# 1/2) or tapered diamond bur on a high-speed handpiece may be used to create space around the root fragment for placement of the instrument (Fig. 18.1).[9]

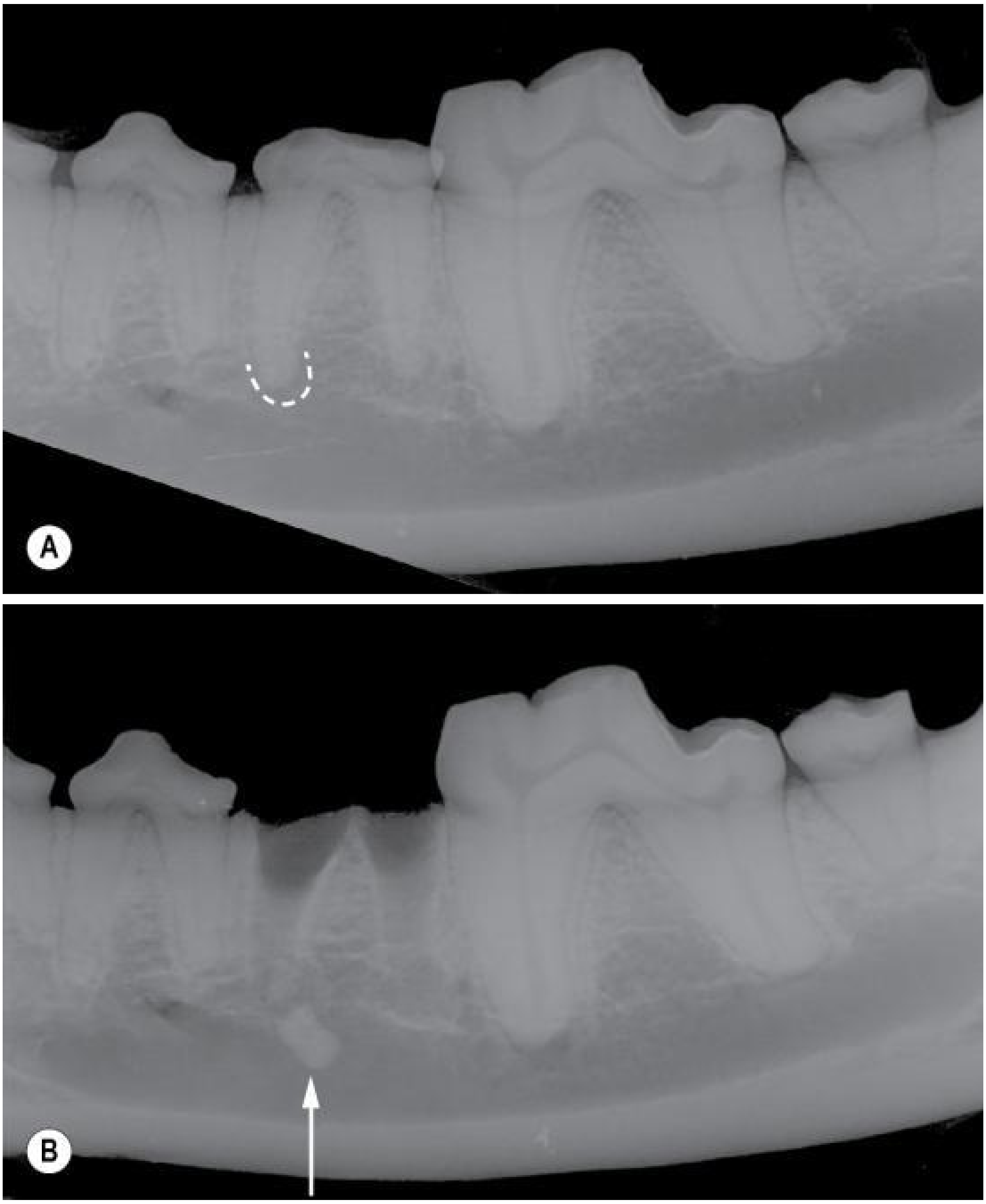


• **FIG. 18.1** Following removal of buccal alveolar bone (**A**), a tapered diamond bur is employed to create a "trough" around the fractured mesiobuccal root fragment *(dotted line)* (**B**) of the right maxillary fourth premolar tooth of a dog. (**C**) This provides room for placement of a narrow-bladed luxator to facilitate removal of the root fragment.

Failure to remove root fragments, particularly those from teeth with preexisting endodontal disease, will lead to continued inflammation in the surrounding tissues, which may result in osteomyelitis, draining tracts, and chronic pain.[10,11] If attempts to remove root fragments are unsuccessful, radiographs should be obtained to document the location and size of the fragments, the client should be informed, and referral to a veterinary dentist should be offered in the interest of providing the best care for the patient.

Displacement of a root or root tip into the mandibular canal, nasal cavity, or maxillary recess

This complication arises when the periapical bone is compromised (Fig. 18.2A) or when excessive apical force is employed during an extraction attempt. In order to avoid displacing a root through its alveolus into the mandibular canal (Fig. 18.2B), nasal cavity, or maxillary recess, the operator should carefully plan each extraction based on radiographic and clinical evaluation. Elevation of a mucogingival flap and adequate bone removal will facilitate extraction and prevent this complication. If a root fragment is displaced but still visible or accessible through the surgical site, the fragment should be carefully removed using a plain Adson forceps or a narrow-beaked root extraction forceps (FX-49 forceps, Hu-Friedy, Chicago, IL).


A
B

• **FIG. 18.2** (**A**) The roots of the mandibular fourth premolar tooth in this dog have periapical radiolucencies *(white dotted line outlines the lucency around the mesial root)*, indicating bone loss. (The lucency ventral to the mesial root of left mandibular first molar tooth is a chevron artifact). (**B**) During the extraction, the mesial root fractured, and attempts to retrieve the apical fragment resulted in displacement into the mandibular canal. Note its more ventral location *(arrow)* compared with the preoperative radiograph. (**C**) After unsuccessful attempts to retrieve the root fragment by removing most of the buccal bone overlying both roots, a window *(arrow)* was created in the buccal alveolar bone more ventrally, allowing retrieval of the root fragment.

If the root tip cannot be removed with the surgical technique immediately after the complication arises, attempts to find the fragment with various instruments must be avoided, as damage to nearby vessels and nerves would be more detrimental than leaving the root in place.[1,2] For a root dislodged into the mandibular canal, an alternative approach may be used to retrieve the fragment. Best performed with a piezoelectric surgical unit (see Chapter 9) to avoid damage to the underlying neurovascular bundle, a circular section of bone may be removed from the buccal aspect of the mandible, creating a window overlying the root fragment (Fig. 18.2C). This allows careful introduction of a small, blunt instrument (e.g., #11 Davis root tip teaser, Hu-Friedy, Chicago, IL) through the window to gently move the root fragment back up through its alveolus and allow retrieval with Adson or root-extracting forceps. Alternatively, if the window is large enough, Adson forceps may be used to grasp the fragment and remove it through the window.

If attempts to retrieve the root fragment are unsuccessful, radiographs should be obtained to document the root's location, and the client should be informed of the complication. If the pet demonstrates signs of pain or paresthesia such as pawing at or rubbing the face following the procedure, referral to an experienced veterinary oral surgeon should be offered.

For a root displacement into the nasal cavity, the client should be advised to observe the patient for nasal discharge and to report persistent sneezing or nasal discharge promptly. Although rare, a root tip in the nasal cavity may become a sequestrum (Fig. 18.3), in which case rhinotomy should be performed to remove the root fragment.

• FIG. 18.3 The root of this cat's right maxillary canine tooth had been dislodged into the nasal cavity during an attempt to extract the tooth 2 years previously. Following rhinotomy to remove the root, the cat's chronic mucopurulent nasal discharge resolved.

Soft tissue injuries

The most common soft tissue injury is tearing of the mucogingival flap during surgical extraction. This usually results from an attempt to be conservative with the flap size and the flap being retracted beyond the tissue's ability to stretch.[1] Tearing may also occur if the tissues are friable and gentle technique is not employed. This complication can largely be avoided by the creation of a large enough mucogingival flap and careful elevation beginning at the attached gingiva. If a tear does occur, the flap should be gently repositioned following the surgery. In most cases, suturing the tear will result in adequate but delayed healing.[1]

Trauma to the cheeks, lips, or sublingual mucosa from a bur may occur if these tissues are not adequately retracted prior to introduction of the high-speed handpiece into the oral cavity. These injuries can largely be avoided with the help of an assistant and the use of tissue retractors such as a Cawood–Minnesota retractor or a tongue depressor.

Puncture wounds of the soft tissues may occur if excessive force is employed during extraction attempts and the luxator or elevator slips. Commonly injured areas are the ventral mandible (e.g., when the luxator slips over the buccal bone during attempts to extract a mandibular canine tooth) and the palate (e.g., when attempting to extract the palatal root of the maxillary fourth premolar tooth). The most severe puncture injuries involve orbital penetration during extraction of maxillary molar teeth, resulting in severe ocular infection and necessitating enucleation.[12,13] This can be avoided by careful positioning of the luxator or elevator between the tooth and bone on the mesial and distal (rather than buccal and lingual) aspects of the tooth and by use of finesse rather than force during extractions.

Fracture of the alveolar process

Fracture of the alveolar process may occur if extraction movements are abrupt and awkward, if extraction forceps are used prematurely, or if there is ankylosis, and part of the alveolar bone (usually buccal) is removed together with the tooth (Fig. 18.4). When the fragment of bone is attached to and removed with the tooth, the remaining bone edges of the empty alveolus are smoothed with a round diamond bur prior to suturing the flap. If the fractured portion of the alveolar bone is separated from the tooth but remains attached to the periosteum, it can be repositioned with the flap and should be stable after the flap is sutured.[2]

• **FIG. 18.4** This extracted mesiobuccal root of a maxillary fourth premolar tooth has a significant amount of buccal bone attached, indicating that the extraction forceps were applied too early in the process and excessive force was used to free the tooth.

Prevention of this complication is achieved by adequate assessment of the tooth and its alveolus via intraoral radiographs and/or cone-beam computed tomography, by controlled removal of alveolar bone (using extraction tips on a piezosurgery unit or a round diamond bur on a dental handpiece with continuous irrigation) prior to luxation or elevation, and by avoiding use of extraction forceps prior to sufficient loosening of the tooth by luxation and/or elevation.[1]

Fracture of an instrument in the tissues

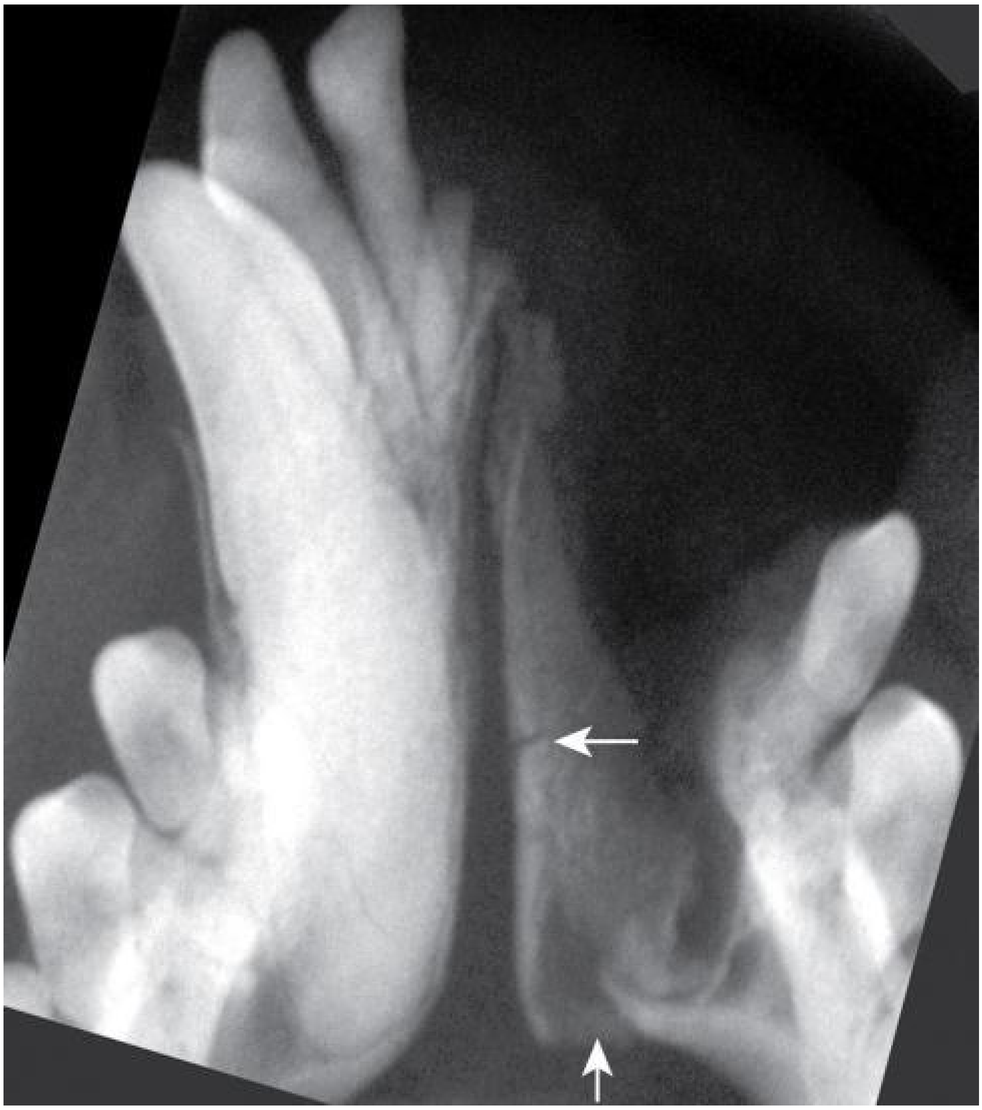
Instrument fracture is uncommon but may result from excessive force during luxation or elevation, from incorrect use of luxators as elevators, or from use of burs for multiple procedures. If the fractured metal fragments are visible, they are carefully removed from the tissues. Radiographs are obtained to confirm complete removal or to identify additional fragments. After radiographic localization, the broken pieces are removed surgically at the same time as the remaining roots.[2]

Fracture of the crown of an adjacent tooth

If adjacent teeth are used as a fulcrum during elevation attempts, fracture of these teeth may occur. If the fracture is superficial and does not involve pulp, the fracture edges are smoothed (odontoplasty), and a light-cured dentinal sealer is applied. If pulp is exposed, the affected tooth must be treated endodontically (with vital pulp therapy or root canal treatment) or extracted. This complication can be avoided by not using adjacent teeth for leverage during extraction attempts.

Mandibular fracture

Anecdotal information suggests that fracture of the mandible during attempts to extract a mandibular canine or first molar tooth is a fairly common occurrence in veterinary practice. At the mandibular canine of small dogs and cats, the lingual alveolar bone is often no more than 1–2 mm thick, and premature use of extraction forceps in a twisting manner results in fracture through the lingual alveolus (Fig. 18.5). The operator is typically aware of the fracture, because following removal of the tooth, there appears to be significant instability of the mandibular symphysis. In actuality, the symphysis is intact, but the fractured lingual alveolar bone just caudal to the symphysis results in a similar feeling of independent movement of both mandibles. Stability can be achieved with a circumferential wire around the rostral mandibles, much like that used for symphyseal separation, but at the level of the mesial aspect of the third premolar tooth (being careful not to injure the sublingual and mandibular salivary ducts when tightening the wire and avoiding slipping of the wire into the empty and fractured alveolus of the mandibular canine tooth). If sufficient teeth are present, the use of an intraoral composite splint may be considered. Placement of a bone graft (e.g., OsteoAllograft, Veterinary Transplant Services, Inc., Kent, WA) is not mandatory but may help speed the healing process.



• **FIG. 18.5** Lingual alveolar fractures (arrows), which occurred during extraction of the left mandibular canine tooth.

When significant periodontal bone loss exists at the mandibular first molar teeth, fracture of the mandibular body may occur during extraction attempts. Preoperative radiographs will help identify patients at risk of this complication and alternatives to extraction, such as periodontal surgery and guided bone regeneration[14] or hemisection and root canal treatment of the remaining root,[15] should be considered. If a fracture occurs, the extraction should be completed and a muzzle applied postoperatively. Once tissues have healed, further fracture treatment planning can be performed. Use of internal fixation techniques and placement of a bone graft (e.g., OsteoAllograft, Veterinary Transplant Services, Inc., Kent, WA) may allow healing, but the client should be informed that the severe preexisting periodontal disease in this location may delay healing. If nonunion occurs, additional extractions and the placement of a plate may be required to achieve stability (see Chapter 53).

Nerve damage

The branches of the trigeminal nerve, including the inferior alveolar nerve, mental nerves, lingual nerve, and infraorbital nerve, are the structures most likely to be damaged during extraction attempts.[1] Nerve injury may occur secondary to local anesthesia, during creation of an incision that extends to the region of the mental or infraorbital foramen, when bone near a nerve is heated excessively due to inadequate irrigation during burring, or during removal of root fragments in or near the mandibular canal. Damage may consist of temporary interruption of nerve conduction (neuropraxia), degeneration of nerve axons (axonotmesis), or permanent interruption of nerve conduction (neurotmesis).[2] Signs of neuropraxia (sensory disturbances such as burning sensation, pins and needles, or numbness) usually resolve within a few days to weeks. Signs of axonotmesis are similar but of longer duration, lasting 6–8 weeks, with the potential for permanent sensory deficits. The most serious nerve damage, neurotmesis, can be produced by ischemia due to prolonged compression, excessive traction on the associated soft tissues, or severance of the nerve fibers, and may result in permanent paresthesia or anesthesia.[2]

If patients exhibit signs of persistent pain postoperatively (hiding, pawing at the face, difficulty prehending or masticating food, or reluctance to groom for more than 1 week after surgery), the extraction site should be examined clinically and radiographically. If there are no root fragments and the soft tissues have healed uneventfully, neuropathic pain should be considered. Although not FDA approved for use in dogs or cats, gabapentin has been used successfully for the management of neuropathic pain in dogs,[16,17] and there is anecdotal information supporting its use in cats.[18,19] Amitriptyline may also be useful for patients who are nonresponsive to gabapentin or for whom three times daily dosing of gabapentin is not feasible.[16,17]

Air embolism

The use of air–water syringes and air-driven dental handpieces, while facilitating sectioning of teeth and removal of alveolar bone and allowing continuous fluid irrigation of the tissues, presents the possibility of introducing air into blood vessels,[20-22] which can be fatal.[23-25] Therefore caution must be exercised when using the air–water syringe or high-speed handpieces, particularly in close proximity to vessels whose integrity may have been compromised by trauma from the extraction procedure.

Postoperative complications

Swelling

Postoperative swelling (Fig. 18.6) may occur secondary to traumatic extraction techniques or excessive compression of tissues resulting in obstruction of lymph vessels.[2] When patient compliance permits, cold compresses may be applied for 10–15 minutes every half hour for the first 4–6 hours after surgery.[2] Administration of nonsteroidal antiinflammatory medications (if not contraindicated based on the patient's medical history and/or perioperative anesthetic complications) may help minimize the severity of the swelling and its associated discomfort.

• **FIG. 18.6** Sublingual swelling following extraction of mandibular premolar and molar teeth in a cat.

Pain

All patients undergoing oral surgery should receive analgesics. As discussed in Chapter 4, administration of analgesics and the use of local anesthetics prior to beginning extractions will help prevent “wind-up” and will decrease the incidence and severity of postoperative pain. In addition, gentle tissue handling, complete tooth extraction, and smoothing of the alveolar bone (alveoloplasty) prior to suturing the gingiva will result in a more comfortable patient than one who has tooth or bone fragments protruding through gingiva, rough bone edges, and extraction sites left open to heal by second intention.

When postoperative pain is evident (demonstrated by inactivity, hiding, pawing at the face, reluctance to groom, difficulty eating, and/or reluctance to eat), it must be addressed. Coadministration of opioids and nonsteroidal antiinflammatory medications should be considered. The use of liquid medications or those that do not require manipulation of the oral cavity (such as transdermal fentanyl patches) may facilitate administration and improve compliance. In severe cases, placement of an esophagostomy tube may be required to allow delivery of medications and to ensure adequate nutrition.

Infection

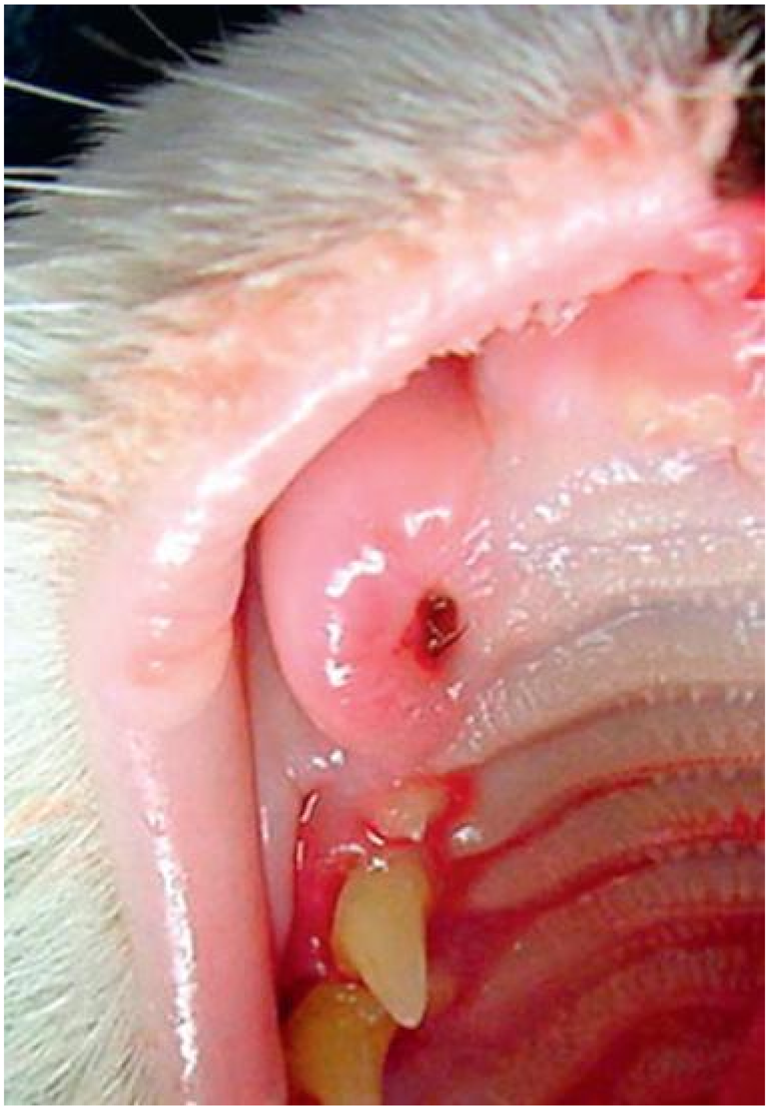
Infection of extraction sites may result from failure to sufficiently reduce oral bacteria by cleaning the teeth before commencing extractions, use of improperly sterilized instruments,[2] and failure to remove debris from surgical sites prior to closure.[1] Patients with severe periodontal disease, active periapical infections, or systemic diseases likely to cause immunosuppression should receive antibiotics intravenously at the time of surgery and, in some cases, postoperatively (by oral administration). The use of antibiotics and antiseptics is discussed in Chapter 3.

Delayed healing or wound dehiscence

There are a number of factors influencing healing of an extraction site. Systemic factors causing delayed healing include diabetes mellitus, hyperadrenocorticism, and chronic corticosteroid administration. Local factors such as presence of infection, sharp alveolar bone edges, or protruding root remnants may also delay or prevent healing. In addition, tension on the gingival flap, caused by insufficient elevation of the periosteum, will lead to dehiscence.[1,2]

Although it has a reported incidence of up to 4% following routine dental extractions in humans,[26] "dry socket," or alveolar osteitis, appears to occur very rarely in dogs[27] and has never been reported in cats. In humans, the suspected etiology is loss of the clot in the alveolus due to increased fibrinolysis (which may result from trauma during the extraction), and estrogens may play a role.[26,28] Although alveolar osteitis is an unlikely complication after extraction, overzealous curettage of the postextraction alveolus should be avoided, and a clot should remain prior to closure of extraction sites.

As mentioned in Chapter 17, the presence of buccal bone expansion may complicate extraction of canine teeth in cats. Expansion of the buccal alveolar bone is typically associated with vertical bone loss,[29] and if nonsurgical extraction of affected canine teeth is performed, healing may be delayed (Fig 18.7). Unless a flap is elevated, the expanded buccal bone removed, and the alveolus debrided of diseased epithelium, the extraction site will heal by second intention, which may take several weeks.



• **FIG. 18.7** Nonsurgical extraction of this cat's right maxillary canine tooth, which had expansion of the buccal alveolar bone due to periodontitis, resulted in delayed healing.

A common cause of nonhealing extraction sites in dogs and cats is neoplasia (Fig. 18.8). Often, teeth found to be "loose" at the time of routine dental cleaning are extracted without preoperative radiographs or dental charting to determine the underlying cause of the increased mobility. When the patient returns with a painful, nonhealing extraction site 1–2 weeks after surgery, biopsy may reveal the true cause of the mobile teeth, which is most often squamous cell carcinoma. Any nonhealing extraction site should be radiographed and biopsied prior to attempts to resuture the wound.

• FIG. 18.8 A nonhealing maxillary canine tooth extraction site in a cat, which was diagnosed as squamous cell carcinoma following biopsy of the soft tissues surrounding the site.

Occlusal trauma

Particularly in cats, entrapment of the upper lip by the mandibular canine tooth may occur following extraction of a maxillary canine tooth (see Fig. 17.7).[30] This can largely be prevented by reducing the crown height of the ipsilateral mandibular canine tooth, as discussed in Chapter 17. Crown height reduction of the mandibular premolars and molar teeth may also be indicated to reduce occlusal trauma to the maxillary soft tissues following extraction of all maxillary premolar and molar teeth in the cat.

Oronasal communication and fistula

Small, dolichocephalic breeds, such as toy and miniature poodles and miniature dachshunds, appear to be predisposed to develop severe vertical bone loss at the palatal aspects of the maxillary canine teeth. A connection between the oral and nasal cavities may result, with the pocket epithelium on the palatal aspect of the maxillary canine tooth becoming continuous with the palatal mucosa on one side of the maxillary bone and the nasal mucosa on the other. If the maxillary canine teeth are nonsurgically extracted, or if the surgical extraction does not include removal of the palatal pocket epithelium, an oronasal fistula may become evident within a few weeks after the procedure (Fig. 18.9). Sneezing after eating or drinking, nasal discharge, and face rubbing may be observed. Chronic exposure of the nasal cavity to oral bacteria will lead to rhinitis, destruction of nasal turbinates, and may result in pneumonia. Therefore oronasal fistulae should be addressed as soon as possible after diagnosis. For small fistulae, single-flap repair[31] may be sufficient for closure. For larger fistulae, double-flap repair[32] or use of an auricular cartilage graft[33-35] should be considered.

• **FIG. 18.9** Persistent oronasal fistula after extraction of the right maxillary canine tooth from a miniature dachshund several months previously.

Alveolar margin recession

Following extraction of large teeth such as canine teeth and mandibular first molar teeth, atrophy of the maxilla or mandible may occur. Bone-grafting techniques may prevent or reduce the severity of recession of the alveolar margin.[36,37] Selection of appropriate materials for placement in the alveolus following extraction is discussed in Chapter 13.

Summary

Most perioperative and postoperative complications can be prevented by careful surgical planning, which includes a thorough review of the patient's medical history, complete physical examination, and comprehensive oral evaluation including preoperative intraoral radiographs. The routine use of postoperative radiographs is recommended. When complications do arise, honest communication with the client and immediate attempts to remedy the situation will allow the continued delivery of optimum patient care.

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SECTION 4:

Periodontal Surgery

OUTLINE

CHAPTER 19

Principles of periodontal surgery

Fraser A. Hale, Cecilia E. Gorrel

Definitions

Attached gingiva: The portion of the gingiva that is apical to the gingival sulcus—this is the gingiva that is physically attached to the cementum and alveolar bone by means of the junctional epithelium and the connective tissue attachment.[1]

Biologic width: The physiologic dimension of the junctional epithelium and connective tissue attachment (see surgical anatomy in Chapter 23 and Fig. 23.1).[2,3]

Cause-related periodontal therapy: Therapy targeting the cause of periodontal disease, i.e., plaque, and consisting of professional periodontal therapy (removal of dental deposits) and establishing proper home care techniques, followed by regular professional maintenance therapy.

Connective tissue attachment: Apical (toward the root tip) to the junctional epithelium and ending at the margin of the alveolar bone, connective tissue fibers from the gingiva insert directly into the cementum of the supraalveolar root to form the connective tissue attachment.[1,2,4]

Free gingiva: The most coronal portion of the gingiva that is not attached to the tooth and that makes up the soft tissue wall of the gingival sulcus (Fig. 19.1).

Gingival recession: The gradual loss of gingival tissue as the free gingival margin migrates apically while the mucogingival junction remains unmoved.[5]

Gingival sulcus: Shallow potential space or groove surrounding each tooth, lined by tooth structure on one side and sulcular epithelium on the other side; normal sulcus depth is 0.5–1 mm in small dogs, 1–3 mm in medium and large dogs, and 0.5–1 mm in cats, with the shallowest sulci around the smallest teeth.

Gingivitis: Plaque-induced inflammation limited to the gingiva.

Infrabony pocket: Periodontal pocket whose base (level of epithelial attachment) is apical to the alveolar margin, and which occurs in conjunction with vertical bone loss.

Junctional epithelium: The unkeratinized, highly permeable epithelium at the bottom of the sulcus, which forms the epithelial attachment to the tooth surface.[1]

New attachment: Formation of new cementum with inserting collagen fibers on a root surface deprived of its periodontal ligament tissue.[6,7]

Periodontal pocket: Increased probing depth due to the apical (towards the root apex) migration of the level of epithelial attachment.

Periodontal probing depth: Distance from the free gingival margin to the base of the sulcus or periodontal pocket, measured in millimeters, with a graduated periodontal probe.[8]

Periodontal surgery: Surgical techniques aimed at removing the predisposing factors and inciting causes of periodontitis and at preserving or regenerating the periodontium.

Periodontitis: Plaque-induced inflammation of the periodontium, resulting in various combinations of progressive gingival recession, destruction of periodontal ligament, and destruction of alveolar bone (attachment loss).

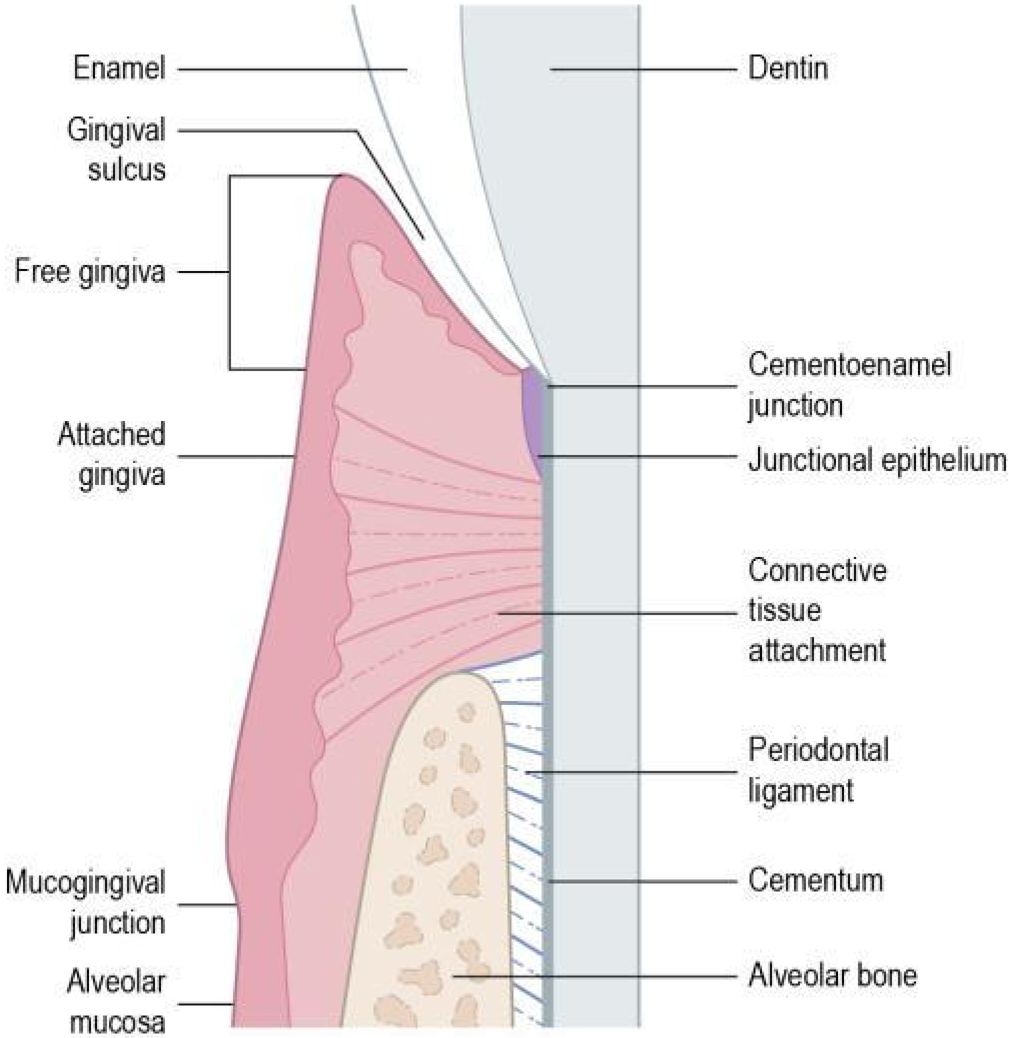
Periodontium: The attachment apparatus of the tooth, consisting of gingiva, periodontal ligament, cementum, and alveolar bone.

Pseudopocket: Increased probing depth due to the coronal (toward the crown tip) migration of the free gingival margin due to gingival enlargement.

Reattachment: The reunion of surrounding soft tissue and a root surface with preserved periodontal ligament tissue.[6,7]

Regeneration: Reformation of cementum, periodontal ligament, and alveolar bone.[7]

Suprabony pocket: Periodontal pocket whose base (level of epithelial attachment) is coronal to the alveolar margin, and which occurs in conjunction with horizontal bone loss.



• **FIG. 19.1** Cross-sectional depiction of the periodontal tissues near the junction of the crown and root of the tooth.

Preoperative concerns

Treatment planning in the management of periodontal disease requires careful consideration of many factors.[5,9,10] These factors can be divided into three broad categories, namely, the client, the patient, and the environment.[10]

Client-related factors

Regardless of the condition presented, it is the client who must present the animal for initial treatment and for follow-up care, provide all postsurgical care and daily home plaque control, and pay for the professional services provided. While the surgeon might have a desire to perform advanced procedures to save periodontally compromised teeth, the client's wishes, expectations, capabilities, and limitations must be considered. If there are any barriers (financial, physical, motivational) that would stand in the way of the postsurgical care necessary for the long-term success of a surgical plan, then an alternative plan, such as extraction, is indicated.[5,10]

Some clients will request that no extractions be performed regardless of the level of disease. The surgeon must ensure that whatever treatment is performed is in the best *medical* interest of the patient. To perform advanced periodontal surgery on a nonsalvageable tooth, or failure to extract teeth with a hopeless prognosis in order to please the client, is not in the patient's best interest and should be considered unethical. It is the surgeon's responsibility to help the client understand the realities of periodontal disease and to obtain informed consent to perform those procedures that will maximize the patient's oral health, even if that means extraction.

Patient-related factors

Periodontal evaluation, surgery, and maintenance therapy all require general anesthesia. If there is reason to suspect that the patient's health will be declining or the anesthetic risk will increase significantly or rapidly, then developing a plan that relies for its success on repeated anesthetics for maintenance therapy would be inappropriate. In such cases, it would be prudent to be more aggressive with extractions.

Systemic or metabolic disease that would interfere with the long-term success of the treatment plan is also a contraindication for periodontal surgery. There is a negative synergy between periodontal disease and diabetes mellitus in that diabetic patients have a higher risk for the development of periodontal disease, and the stress on the body imposed by chronic periodontal disease makes it harder to regulate a diabetic patient.[11-13] There are several other metabolic diseases that have a negative impact on the periodontal prognosis, either on their own or because of the medications required to manage them. These conditions and medications and their effect on the prognosis must be carefully considered. For instance, phenytoin derivatives, calcium channel blockers, and cyclosporine can all result in drug-induced gingival enlargements with the development of pseusdopockets (See Chapter 46). Immunocompromised patients may be less able to fend off the challenges posed by the oral microbiome.

Anatomical factors such as severe dental crowding will have a negative impact on the periodontal prognosis; some of these factors can be ameliorated to improve the prognosis, e.g., by selective dental extractions.[14]

The animal's attitude toward being handled and manipulated is another important factor, as are its dietary needs and habits. Some animals can be quickly trained to accept and enjoy daily tooth brushing. Others will not tolerate this. Fortunately, there is an ever-growing list of safe and effective daily home care products (diets, chews, water additive, sprays, gels, etc.). Readers are encouraged to visit www.vohc.org to see the current list of products that have earned the Veterinary Oral Health Council (VOHC) Seal of Acceptance; products with the seal have valid, independently reviewed in vivo clinical trials demonstrating efficacy in reducing plaque, calculus, or both.[15] It is important to note that no product, strategy, or combination of products and strategies will be completely effective at plaque control; clients must have reasonable expectations for what can be achieved with home dental care. Most clients brush their own teeth twice daily, floss daily, and still seek professional evaluations and maintenance hygiene treatment every 6 months for their own teeth.

If the animal or the owner is unlikely to be sufficiently compliant with any of the various home plaque-control strategies available, extraction would be more appropriate than periodontal surgery.[10]

Environment-related factors

In some cases, periodontal surgery is performed by a veterinary dental specialist to whom the client and patient have been referred by their primary care veterinarian. In a multidoctor general practice, sometimes periodontal surgery is performed by the veterinarian who has the greatest interest and skill in the field of dentistry but is not the patient's primary care practitioner. In either case, the patient and client will likely have future contact with someone other than the person who performed the surgery. The primary care veterinarian must offer the necessary support in the long term and encourage the client to return to the specialist for necessary follow-up assessments and maintenance therapy.

To treat or extract?

The primary objective for the veterinary dental patient is to achieve and maintain a mouth free from pain and infection.[10] While it can be very rewarding for the oral surgeon to return a periodontally diseased tooth to health, the likelihood of achieving and maintaining that objective is often too remote to justify the attempt. Domestic dogs and cats can live very happily in an edentulous state and are far better off with no teeth than with diseased teeth.[10]

In summary, the surgeon must consider the following prior to undertaking advanced periodontal surgery[9,10]:

- What is the nature and extent of disease?
- What is the duration of disease? In periodontal disease, rapid onset carries a poorer prognosis.
- Is the condition localized or generalized? The prognosis for a patient with generalized periodontal disease is not as good as for a patient with overall good periodontal health who has one foreign body–induced lesion, for example.
- What are the causative factors? Are they purely local or are there systemic factors that may be uncontrollable, such as diabetes mellitus or feline immunodeficiency virus (FIV) infection?
- How old is the patient? If the dog is 13 years old, we may need only manage the tooth for a few more years, but if the dog is only 2 years old, we will need to manage the problem for many years.
- To what degree are the roots and furcations involved? Furcation involvement makes both the initial treatment and home care more challenging.[16,17]
- Are there occlusal factors to consider? Overcrowding and rotation of teeth make management of periodontal disease more difficult.
- Generally, advanced periodontal treatment takes longer than extraction, therefore it costs more and increases the risk of anesthetic complications.[18]
- Generally, periodontal surgery is technically more challenging than extraction.
- Is the tooth strategically important enough to justify the investment in time, effort, and money?
- Visualize what the mouth would be like without the tooth, remembering that loss of a tooth leads to resorption of the supporting alveolar bone. Preserving the mandibular canine tooth in a working dog is a higher priority than preserving the maxillary first premolar tooth in a pet dog.

- Does the surgeon have the appropriate equipment and training to treat the tooth properly?

Once the surgeon has completed an assessment of the above issues, a rational, achievable, and medically appropriate treatment plan can be developed. In human patients, periodontal surgery is not the first-line treatment for periodontitis. Cause-related therapy, i.e., removal of dental deposits and establishing proper home care techniques, is performed first, and the need for surgery is evaluated after a reasonable time to allow for healing and improvement in tissue health. If the human patient cannot maintain good oral hygiene, there is no indication for periodontal surgery. Periodontal surgery is a possible adjunct to cause-related therapy, but only if plaque control is optimal.

The need for general anesthesia in veterinary patients often means that the veterinary dentist is required to obtain a diagnosis, provide cause-related therapy, and perform periodontal surgery in one session. Veterinary patients with periodontal disease will often have oral sensitivity and pain, with the result that clients are unable to establish an effective home care program using any mechanical plaque-control measures such as tooth brushing, dental diets, or chews preoperatively.[5] This means that we must sometimes make treatment decisions based on untested assumptions regarding the patient's ability to respond to treatment and the client's ability to establish an effective, daily plaque-control program. Clients must therefore be made aware, and be willing to accept, that the prognosis is based on these assumptions and if reality falls short of expectation, so will the results.

Therapeutic decision-making

Objectives of periodontal surgery

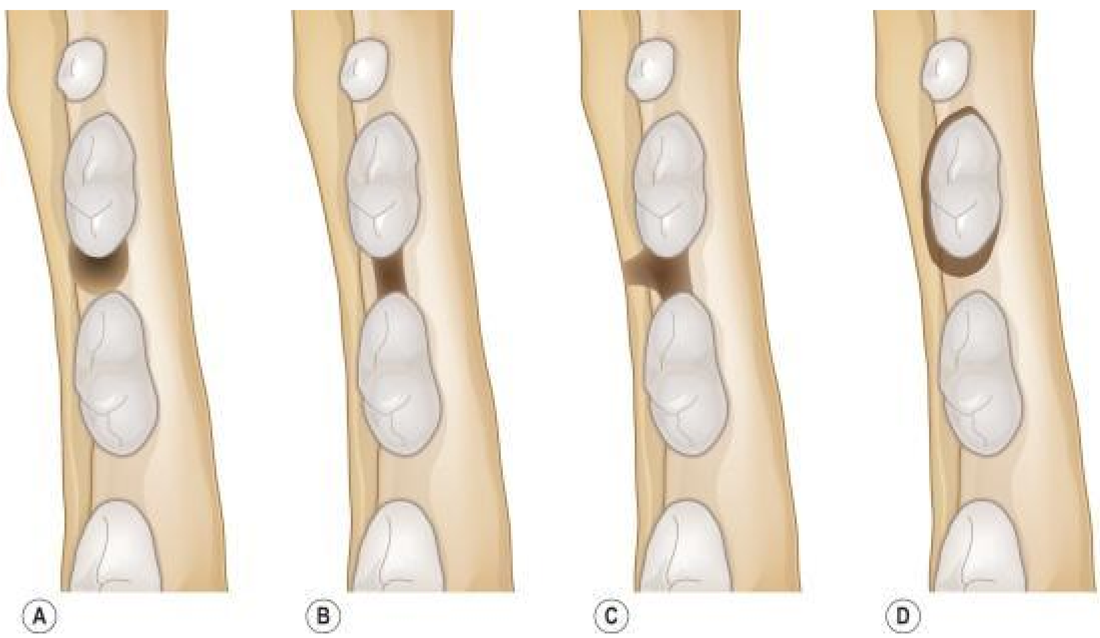
Historically, "pocket elimination" has been the main objective of periodontal surgery.[19] However, increased probing depth does not necessarily indicate periodontal destruction. True periodontal pockets (due to destruction of epithelial and connective tissue attachments, periodontal ligament, and alveolar bone) need to be differentiated from pseudopockets (due to proliferation of the gingiva, e.g., gingival hyperplasia). Moreover, there is no established correlation between probing depth and the presence or absence of active disease.[19] This means that signs other than increased probing depth need to be present to justify surgical therapy. These include clinical signs of inflammation (especially exudation and bleeding on probing to the bottom of the pocket), as well as aberrations of gingival morphology. Finally, since proper plaque control (home care) is a decisive factor for good prognosis, this must be considered prior to surgery.[20,21]

The main objective of periodontal surgery is to contribute to the long-term preservation of the periodontium by facilitating plaque removal and plaque control.[7] Periodontal surgery can serve this purpose by: (1) creating accessibility for professional scaling and root planing and (2) establishing a gingival morphology that facilitates home care. In addition, periodontal surgery may aim to regenerate lost periodontal attachment.

Selection of surgical technique

Many of the technical problems experienced in periodontal surgery stem from the difficulties in accurately assessing prior to anesthesia and surgery the degree and type of periodontal breakdown that has occurred. At the time of surgery, previously undiagnosed defects may be identified, or some defects may be more complex than anticipated. Consequently, a combination of techniques may be required at the same site. As a general rule, techniques that preserve or induce the formation of periodontal tissue should be preferred over those that remove tissue.

Infrabony pockets are described by depth and by the extent of the bony circumference involved (Fig. 19.2). The surrounding alveolar bone is thought of as forming four walls (mesial, vestibular, distal, palatal/lingual). When bone is present around the entire circumference of the pocket, a four-wall defect is present. When bone is missing on one face, a three-wall defect is present. Two-wall and one-wall defects have two and three surfaces of the tooth root without bony support, respectively.[22-24] In general, the more bony walls there are surrounding an infrabony pocket, the greater the potential for the regeneration of lost periodontal tissues following complete removal of all irritants and inflamed soft tissues from the defect.



• **FIG. 19.2** Occlusal views of the right mandible of a dog showing: (**A**) a three-walled bony defect; (**B**) a two-walled bony defect; (**C**) a one-walled bony defect; and (**D**) a cup defect. Source: (From Tsugawa AJ, Verstraete FJM. How to obtain and interpret periodontal radiographs in dogs. *Clin Tech Small Anim Pract*. 2000;15:204–210.)

Gingivectomy/gingivoplasty

Gingivectomy is the excision of gingival tissue, usually to remove the diseased wall of a periodontal pocket (true pocket or pseudopocket).[25] Gingivoplasty is the recontouring of the gingiva to its proper anatomical form without the reduction of periodontal pocket depth (see Chapter 20).[25] This is typically a combined procedure, which can be used in isolation or together with other surgical procedures such as flap surgery.[26] The main indication for gingivectomy or gingivoplasty in veterinary dentistry is in the management of gingival enlargements. Gingivectomy may also be used for type-1 surgical crown-lengthening (see Chapter 23).

Flap operations with or without osseous surgery

Flap procedures can be used in all cases where surgical treatment of periodontal disease is indicated. These procedures can be performed with or without osseous surgery (see Chapters 21 and 22). Flap procedures are particularly useful where periodontal pockets extend beyond the mucogingival junction, the furcation is involved, and/or recontouring of bony lesions is required. The advantages of flap operations include: (1) existing keratinized gingiva is preserved; (2) the operator has increased visualization of the defect and can ensure thorough debridement of dental deposits and inflamed soft tissues; (3) the marginal bone is exposed, whereby the morphology of bony defects can be identified and the proper treatment rendered; (4) the flap can either be replaced at its original position or shifted apically or coronally, thereby making it possible to adjust the gingival margin to the local conditions; and (5) the flap procedure preserves the oral epithelium.

Healing processes for commonly used techniques

In order to understand what periodontal surgery aims to achieve, it is necessary to understand the patterns of wound healing that occur in the periodontium. All wounds heal in three phases: an inflammatory phase, followed by a proliferation phase and then a maturation/remodeling phase (see Chapter 1). The wounds caused by periodontal surgery follow this pattern, with specific variations for each of the commonly used methods (gingivectomy/gingivoplasty versus flap techniques).

Gingivectomy/gingivoplasty

The healing of a gingivectomy/gingivoplasty wound is similar to that of a simple soft-tissue wound, except that there is a tooth in the center of the wound. Following incision with a scalpel blade or gingivectomy knife, the wound is covered by a fibrin clot, and the underlying tissue becomes acutely inflamed with some necrosis.[25] The clot is replaced by granulation tissue, and after 12–24 hours epithelial cells at the margin of the wound begin to migrate across the granulation tissue bed.[26] The epithelial activity reaches its peak by 24–36 hours postincision. Surface epithelialization is generally complete after 5–14 days, but complete epithelial repair and keratinization may take a month.[25] Complete repair of the underlying connective tissue takes about 7 weeks.[25]

Wound healing following electrosurgery/radiosurgery can be affected by various factors such as the frequency of the signal produced by the instrument, the wave form utilized, the power setting utilized, and technique.[27-32] Use of a high-frequency radiosurgical unit (3.0–4.0 MHz) and a fully filtered wave form at an appropriate power setting results in tissue damage and healing similar to cold steel incision.[25,32] Lower-frequency electrosurgical units and other wave forms will result in greater thermal necrosis of the wound and delayed healing. Slow passage of the electrode through the tissues and excessive power settings also increase lateral heat dissipation into tissues, resulting in collateral damage and delayed healing. Electrosurgery is contraindicated in close proximity to bone as it can result in greater gingival recession, bone necrosis, and loss of bone height.[25]

Healing following laser surgery is delayed compared with that following cold steel surgery.[25,33] The risk of collateral damage to the surrounding hard tissues (enamel, cementum, and bone) is also of concern.[33,34]

Flap techniques

The pattern of healing in the absence of vertical bone loss is as follows. There is no open wound after suturing the flap in place. The soft tissue collar around the tooth consists of keratinized gingival epithelium on the oral surface of the flap with its underlying connective tissue in contact with the alveolar bone and root surface. The junctional epithelial attachment and gingival sulcus have been removed during the procedure. Immediately postoperatively, there is a blood clot between the tooth and the flap. The fibrin in the clot functions as a glue between the tooth and flap.[35] As healing starts, both the connective tissue and epithelium are activated. The gingival epithelium will start to grow down between the tooth and the flap to form a new junctional epithelial attachment. Simultaneously, new connective tissue will grow into the fibrin clot trying to gain attachment to the root surface. The situation is now a race between the junctional epithelium growing apically to cover the exposed connective tissue at the root surface and the connective tissue gaining new attachment to the root surface. The result of the race will determine at which level the junctional epithelium's apical attachment will be after healing. Usually, the connective tissue will have time to form some attachment to the root surface coronal to the level of the alveolar bone before the junctional epithelium has grown down. The final stage of the healing process is the reformation of the normal junctional epithelial attachment and gingival sulcus. At 2 weeks, the union of the flap to the tooth is still weak due to the immaturity of the collagen fibers. One month following surgery, the gingival sulcus is fully epithelialized, and a well-defined junctional epithelial attachment is present.[35] Following full-thickness flap surgery, which temporarily denudes bone, there will be superficial bone necrosis resulting in bone loss of about 1 mm (more if the bone is thin).[35]

When vertical bone loss is present, the pattern of healing differs in that first a blood clot forms between the root and the alveolar bone. The blood clot starts to be replaced by connective tissue as fibroblasts become activated, reproduce, and synthesize collagen. New cementum may form on the root surface. Collagen fibers from the periodontal membrane become embedded in the newly formed cementum, forming a new periodontal ligament. Osteoblasts are activated and start laying down bone in the vertical wall of the bony pocket.

The above sequence, i.e., the formation of normal bone and normal periodontal ligament, takes months to occur. The threat to the above events occurring successfully is the apically directed growth of the junctional epithelium trying to cover the "wound" between the tooth and the connective tissue. Various techniques, e.g., guided tissue regeneration, have been advocated to encourage new attachment by preventing epithelial downgrowth.[36,37] Other outcomes, such as external root resorption and ankylosis, are also possible.[7] A more extensive discussion of guided tissue regeneration follows in Chapter 22.

Postoperative care and assessment

With a carefully performed procedure (gentle handling of tissues, ensuring that exposed bone is kept moist, ensuring complete coverage of the alveolar bone when suturing flaps), most patients experience only minimal postoperative discomfort. The pain experienced is limited to the first few days and can be adequately controlled with preemptive and perioperative pain management techniques (see Chapter 4).

Feeding soft food is recommended during the early phase of healing (10–14 days) in order to reduce discomfort and maintain good wound stability. Postoperative plaque control is the most important variable in determining the long-term result of periodontal surgery. Disease recurrence is inevitable, regardless of surgical technique used, if postoperative plaque control is suboptimal.[20,21,38,39] Some advocate twice-daily rinsing with 0.1%–0.2% chlorhexidine gluconate solution.[40] However, it is important that the client not disturb the surgical site(s), as this would not only cause pain for the patient but could also disrupt healing. Therefore, they must be instructed to handle the mouth very gently, if at all, taking care to avoid putting pressure or tension across any suture lines. In the immediate postoperative period (10–14 days), plaque formation may best be retarded by passive, rather than mechanical, methods. This may include the veterinary application of a wax-polymer barrier sealant (Ora-Vet; Merial Ltd., Duluth, GA), or a gingival sulcular sealant (Sanos; Allaccem Inc., San Carlos, CA), or the home use of a VOHC-accepted plaque-retardant water additive (**healthy**mouth; HealthyMouth LLC, Malibu CA).[41] Administration of systemic antibiotics following periodontal surgery appears to be beneficial, resulting in increased attachment levels when compared with periodontal surgery alone, with administration of tetracycline or metronidazole proving to be more beneficial than other antibiotic regimens.[42] However, with ever-increasing concerns regarding antibiotic resistance and responsible antibiotic stewardship, the use of systemic antibiotics in managing periodontal disease is becoming harder to justify. Rather, the administration of a local perioceutic at the surgical site(s) should be considered (Doxirobe; Zoetis, Parsippany, NY, or Clindoral; TriLogic Pharma, Tallassee, AL).

It is important to return to and maintain good mechanical oral hygiene measures as soon as the tissues have healed sufficiently that brushing will not cause pain or disrupt healing, especially since rinsing with chlorhexidine does not prevent subgingival recolonization of plaque and can lead to a nonpermanent but unsightly staining on the crowns of the teeth.

The initial healing should be evaluated after 10–14 days. Following this examination, the client should start gentle brushing of the operated areas using a soft-bristled, nylon toothbrush that has been softened in hot water. Adjunctive use of VOHC-accepted pastes or gels on the brush or water additives or sprays should be encouraged. Diets and treats shown to help reduce plaque accumulation should be considered if there are no medical or dietary contraindications for the patient and if the patient still has sufficient dentition for chewing these consumables.[15] Recheck appointments for conscious examination are scheduled at 2-week intervals to closely monitor plaque control. During this postoperative maintenance phase, adjustments of the methods for optimal home care are made depending on the healing status of the tissues. Depending on compliance, the time interval between visits for supportive care may gradually be increased. The next full periodontal assessment and professional maintenance therapy may be scheduled for between 3 and 12 months postoperatively, depending on a variety of factors. Since complete periodontal healing and regeneration of periodontal tissues takes months, assessing prior to 3 months may underestimate the final result. Conversely, waiting too long before reassessing risks allows time for the reestablishment of the disease process and loss of all gains achieved.

Prognosis

The prognosis for the outcome of periodontal surgery is affected by many factors including: (1) the skill of the surgeon; (2) the habits and diet of the patient; (3) the level of home care (plaque control); and (4) the patient's susceptibility/resistance to periodontal disease. With so many variables, the prognosis is always somewhat uncertain, and the client must be aware of and willing to accept this prior to undertaking the procedure. With careful case selection and treatment planning, followed by establishment and maintenance of proper postoperative plaque-control measures, most surgical treatment techniques will result in conditions that favor the maintenance of a healthy periodontium.

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CHAPTER 20

Gingivectomy and gingivoplasty

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Definitions

Gingivectomy refers to the surgical excision of gingival tissue and is performed to reduce the depth of a suprabony periodontal pocket by removing a portion of the gingival wall of that pocket. *Gingivoplasty* refers to the reshaping of the gingiva to create anatomically normal and physiologically beneficial gingival contours.[1] The two techniques are typically combined.

Preoperative concerns

As the gingival tissues have a rich vascular supply, gingivectomy/gingivoplasty tends to result in considerable intraoperative bleeding. If a clotting profile and platelet count have not already been performed, then a *buccal mucosal bleeding time* (BMBT) test can quickly be done in the anesthetized patient prior to the first incision. This test is performed by employing a BMBT device (Simplate, Organon Teknika, Durham, North Carolina, or Surgicutt, International Technidyne Corp, Edison, New Jersey) or by making a stab incision into the buccal mucosa with a #11 scalpel blade and recording the time it takes for the wound to stop bleeding. Mucosal bleeding times of less than 3–5 minutes are considered normal.[2]

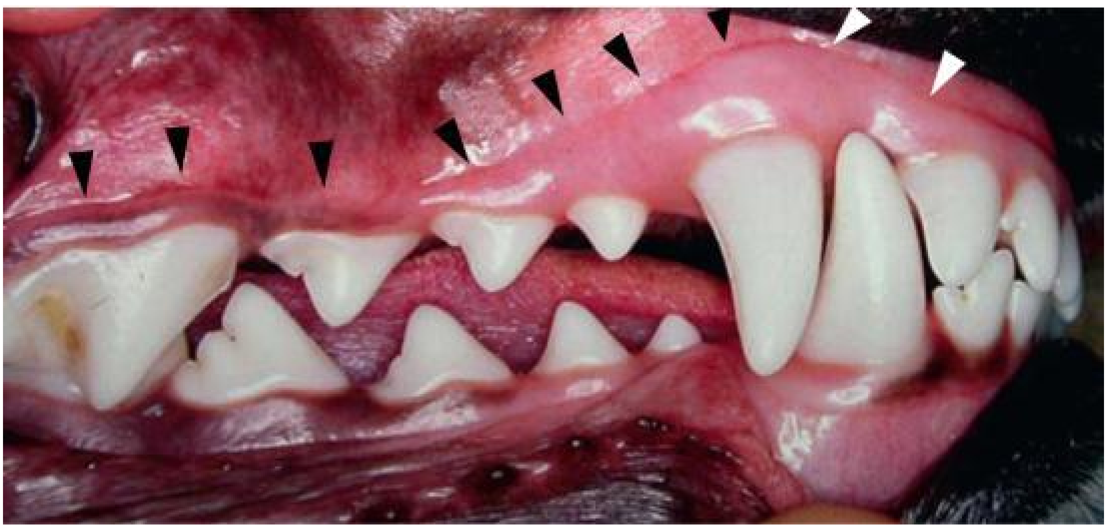
There are several drugs that may predispose to the development of gingival enlargement (see Chapter 46), and so the patient history should be reviewed to see if any of these medications have been administered to the patient.[3] If it is determined that the gingival enlargements are likely drug induced, then efforts to cease administration of that drug should be made. Often, this will result in dramatic improvement and may even make surgery unnecessary. If the patient absolutely requires the causative drug and no substitute can be found, the client must be made aware that the gingival enlargement will almost certainly reoccur following surgery. Maintaining excellent plaque control may reduce or retard the recurrence as it is felt that, while the drug increases the likelihood of developing enlargements, inflammation is still necessary for its development.[3]

Vitamin C deficiency has been reported to cause gingival enlargement. Degeneration of the gingival collagen and alterations in the gingival defenses against plaque result in edema, inflammation, and gingival enlargement.[4]

The literature contains at least one report of a localized gingival enlargement due to disseminated cryptococcosis in a Siamese cat.[5]

Surgical anatomy

The gingiva surrounds the teeth and the marginal parts of the alveolar bone, forming a cuff around each tooth (see Fig. 19.1). It can be divided into the free gingiva, which is closely adapted to, but not attached to, the tooth surface, and the attached gingiva that is attached to the suprabony cementum and the underlying periosteum of the alveolar bone. The attached gingiva is delineated from the oral mucosa by the mucogingival junction (Fig. 20.1), except on the palatal aspect of the maxillary teeth, where it blends imperceptibly with the palatal mucosa (Fig. 20.2).[6,7]



• **FIG. 20.1** Healthy gingiva in a dog showing the mucogingival junction *(arrowheads)*. Regardless of where the free gingival margin may go (coronally with enlargement or apically with recession), the mucogingival junction remains unmoved. It is apparent in this photo that the width of the attached gingiva varies considerably throughout the mouth.

• FIG. 20.2 Palatal view of the right maxillary premolar teeth in a dog showing how the gingiva blends imperceptibly with the palatal mucosa. Note the crown fracture of the fourth premolar tooth necessitating endodontic treatment or extraction.

The gingival tissues in the spaces between the closely spaced teeth (the interproximal spaces) form a gingival papilla (Fig. 20.3).[7,8] This triangularly shaped structure is important in excluding food, hair, and other foreign material from becoming trapped in the interproximal space.[8] Between the facial and lingual/palatal gingival papillae, there will be a valley-like architecture known as the gingival col.[7,8]

• FIG. 20.3 Maxillary incisor teeth of a dog depicting the gingival papillae.

As the free gingiva is not attached to the tooth, there is a shallow potential space between the free gingiva and the tooth known as the gingival sulcus (see Fig. 19.1). The depth of the sulcus can be assessed by gently inserting a graduated periodontal probe until resistance is encountered. This resistance is taken to be the base of the sulcus. The depth from the free gingival margin to the base of the sulcus can thus be measured.

In the periodontally healthy individual, the sulcus depth is 0.5–1 mm in small dogs, 1–3 mm in medium and large dogs, and 0.5–1 mm in cats, with the probing depth being the least around the smaller teeth. In the healthy situation, the sulcus will often be deeper where the band of gingival tissue is wider, and the sulcus will be shallower where the band of gingiva is narrower. For example, the distance from the free gingival margin to the mucogingival junction on the vestibular aspect of the maxillary canine tooth in a large dog may be as much as 15 mm and a probing depth of up to 5 mm would be acceptable. On the other hand, the distance from the free gingival margin to the mucogingival junction over the maxillary fourth premolar tooth in a cat would be approximately 1.5 mm and a probing depth over 0.5 mm would be considered abnormal (Fig. 20.1).

The oral surface of the gingival epithelium contains the four layers typical of epithelia elsewhere, namely the stratum basale, stratum spinosum, stratum granulosum, and stratum corneum.[7,9,10] The stratum corneum may be orthokeratinized or parakeratinized.[7,9,10] Local irritation interferes with keratinization, and healthy gingiva is more keratinized than diseased, irritated gingiva.[7] Nonepithelial cells are also present in the oral gingival epithelium. These include melanocytes and Langerhans cells in the stratum spinosum.[9] The Langerhans cells participate in the local immune response by presenting antigen to and participating in the activation of T-helper lymphocytes.[9]

The gingival sulcus is lined by the oral sulcular epithelium, which is nonkeratinized.[7,10] The sulcular epithelium has the potential to keratinize if the sulcular microflora is totally eliminated or if the epithelium is reflected and exposed to the oral cavity. On the other hand, the outer gingival epithelium will lose its keratinization if brought into contact with the tooth. This is one basis for the theory that local irritants, such as bacterial toxins, may prevent sulcular and oral epithelial keratinization.[7]

Apical to the gingival sulcus is a band of highly permeable epithelium called the junctional epithelium, which forms the epithelial attachment to the tooth.[7] The junctional epithelium is a nonkeratinized stratified squamous epithelium that is derived from remnants of the reduced enamel epithelium during eruption of the tooth. Unlike other oral epithelia, it has only two layers (stratum basale and stratum suprabasale).[11] The junctional epithelium also contains a few leukocytes.[11]

Apical to the junctional epithelium is the connective tissue attachment. Here, fiber bundles of the gingival connective tissue insert into the supra-alveolar cementum.[11] The gingival connective tissue is also firmly attached to the periosteum of the alveolar bone.

Biologic width is defined as the dimensional width of the junctional epithelium and connective tissue attachment (see Fig. 23.1).[12] In human patients, biologic width is typically 2–3 mm. The dimensions in veterinary patients will be quite variable and in proportion to the size of the tooth and its periodontal tissues. Biologic width is commonly discussed in reference to the distance that must be maintained between the alveolar margin and any restoration on a tooth, in order to leave sufficient room for each of the elements of the gingival attachment and a gingival sulcus (the dentogingival complex). When considering gingivectomy/gingivoplasty, biologic width must be respected. If the gingivectomy incision approaches too close to the bone, there will be insufficient room for the establishment of all the elements of the dentogingival complex. The result will be inflammatory resorption of alveolar bone and gradual reestablishment of biologic width in a more apical location.[12]

Instrumentation and equipment

Gingivectomy/gingivoplasty can be performed with cold steel (scalpel blade, gingivectomy knives, and scissors) (Fig. 20.4), diamond or fluted carbide burs in a high-speed dental handpiece (Fig. 20.5), electrosurgery, radio wave radiosurgery (Fig. 20.6), or laser surgery. Each modality has its advantages and disadvantages.[1,13-15] Often, the choice of modality is directed by availability. For example, veterinarians who have invested in laser are inclined to use it for gingivectomy/gingivoplasty; those with a radiosurgical unit are inclined to use that; those with neither are inclined to use cold steel. Used properly and within their limitations, all will perform adequately. Used inappropriately or beyond their limitations, all can cause great harm to the patient. Therefore it is essential that practitioners be very familiar with the technical aspects of the modality they intend to utilize.

• **FIG. 20.4** (**A**) Goldman–Fox periodontal pocket marking forceps (PMGF1). (**B**) Kirkland gingivectomy knife (KK15/16). (**C**) Orban gingivectomy knife (KO1/2). (**D**) Universal 360-degree scalpel handle (K360). Source: (Courtesy Hu-Friedy Mfg. Inc., Chicago, IL.)

• **FIG. 20.5** (**A**) Gingival enlargement at the left maxillary third incisor and canine teeth in a dog. (**B**) An egg-shaped, 12-fluted bur in a high-speed handpiece is used to remove the gingival enlargement and sculpt the gingiva to physiologic height and contour. (**C**) Postoperative appearance. Source: (From Lewis JR, Reiter AM. Management of generalized gingival enlargement in a dog—case report and literature review. *J Veterinary Dent*. With permission.)


ellman
SURGITRON
F.F.P.F. EMC VET.SURG
AC
POWER
RF
SURGERY
ANTENNA
ACTIVE
FULGURATE

A


CUTTING MODE
ENERGIZED
POWER
SELECT
CUT
CUT/COAG
FOOTSWITCH
INDICATOR
COAGULATION MODE
ENERGIZED
SELECT
HEMO
FULGURATE
BIPOLAR
120 IEC
ellman
Surgitron
4.0 Dual RF
FAULT
NEUTRAL
FINGERSWITCH/HANDPIECE
BIPOLAR HANDPIECE
NEUTRAL
ON/OFF

B

• FIG. 20.6 (**A**) Ellman Surgitron EMC, 3.8 MHz radiosurgical unit. (**B**) Ellman Surgitron RF 4.0 MHz unit. (**C**) Electrode tips for use with the Ellman units, from top, C7B triangle loop, B1B round loop, C3B diamond loop and A8B wire tip. Source: (Courtesy Ellman International Inc., Oceanside, NY.)

Cold steel

Cold steel incision is, in many ways, the preferred modality for gingivectomy/gingivoplasty, being listed first in virtually all references of note.[4,4,13–17] It can provide a clean and neat incision with no collateral tissue damage and rapid healing (see Chapter 19). It is inexpensive and readily accessible. However, as the gingival tissues are highly vascular, cold steel incision does result in considerable intraoperative bleeding, often obscuring the field of view and requiring frequent swabbing by an assistant. Also, gingival enlargements are often highly fibrous and may contain osseous metaplasia. This can force the surgeon to apply considerable pressure to incise the tissues, resulting in less control of the cut and the potential to slip and make accidental incisions. Also, cold blades dull quickly when cutting through such tissues and when contacting the underlying dental enamel, so scalpel blades need to be replaced and gingivectomy knives and scissors need to be sharpened frequently during the procedure.

Diamond or fluted carbide bur

While few references make mention of the use of carbide or diamond dental burs in a high-speed handpiece, it is an option to be considered.[4,18] In this technique, diamond or fluted carbide dental burs (in a high-speed handpiece) are used to create the desired incision and bevel (Fig. 20.5).[4] The burs permit good contouring and produce less hemorrhage than cold steel does. Accidental damage of the tooth surface with the bur is a risk when using this technique. Protecting the tooth surface (e.g., with a plastic spatula) is recommended if possible. Ample irrigation is used to prevent thermal damage. The time spent cutting the gingiva around a tooth should be as short as possible.

Electrosurgery and radiosurgery

While these two modalities are often discussed together and even interchangeably, there are significant and important differences between them and they are not interchangeable.

Electrosurgery uses relatively low-frequency electrical energy (0.5–2.9 MHz).[19] It creates heat in the tissue due to the resistance of the tissues to the passing of electrical current through the patient between two points of contact (typically the active handpiece electrode and a grounding plate, which must be in direct physical contact with the patient).[19,20] Since the separation of tissues is achieved through the generation of heat at the electrode contact point, there is lateral dissipation of heat into the tissues beyond the incision line.

Radiosurgery is achieved by passing high-frequency radio wave energy (3.0–4.0 MHz) through the patient between the active electrode and a passive antenna, which need only be in proximity to the patient, not in direct physical contact. The units depicted in the photographs operate at 3.8 and 4.0 MHz, respectively (Fig. 20.6). Tissue resistance to the passage of the radio waves causes ionic agitation in the cells at the active electrode tip. This causes molecular friction and heating of the tissue. The heat comes from molecular friction in the cells, not from the electrode tip itself.[20]

Various studies have demonstrated that high-frequency radio wave radiosurgery, when used properly, results in a smaller zone of thermal necrosis at the incision compared to electrosurgery at lower frequencies and laser.[20-22]

Electrosurgical and radiosurgical units can be configured to produce four different waveforms, although not all units offer all waveform options. The fully filtered waveform provides a pure, continuous flow of current and offers the smoothest incision with the least amount of thermal necrosis and tissue shrinkage, and healing is very similar to a cold incision.[23] Due to the minimal lateral heat transfer of the fully filtered waveform, it can be used in relatively close proximity to bone. It does not provide significant hemostasis (coagulation of small vessels).[24,25]

The fully rectified waveform offers simultaneous cutting and hemostasis by coagulating small vessels. The cost of this hemostasis is a slightly wider zone of thermal damage and more tissue shrinkage. Therefore this waveform should not be used in close proximity to bone or dental hard tissues. It is a suitable waveform for bulk removal of excess gingival tissue, provided the electrode can be kept a few millimeters from bone and tooth.[24,25]

The partially rectified waveform provides an intermittent flow of energy that provides effective hemostasis by coagulating vessels up to 1/16 inch (1.6 mm) in diameter while causing more thermal necrosis and tissue shrinkage. This waveform is not suitable for gingivectomy/gingivoplasty.

The fulguration, or spark-gap waveform, is the most destructive of the four waves, causing considerable thermal necrosis, tissue shrinkage, and scarring. It has no application in gingivectomy/gingivoplasty.[24,25]

In summary, high-frequency radio wave surgery using a fully filtered waveform at an appropriate power setting and with a suitably shaped electrode (Fig. 20.6C) provides a very smooth, pressure-free incision, allowing the surgeon to easily sculpt the tissues to a desirable height and contour, even when approaching very close to the bone. The fully rectified waveform also offers simultaneous hemostasis but, due to a slight increase in lateral heat transfer, should not be used in close proximity to bone. Since one imperative in gingivectomy/gingivoplasty is to ensure the preservation of sufficient gingival tissues (both in height and thickness) to support the periodontal health of the tooth, the incision should not approach the bone and so the fully rectified waveform is quite acceptable.

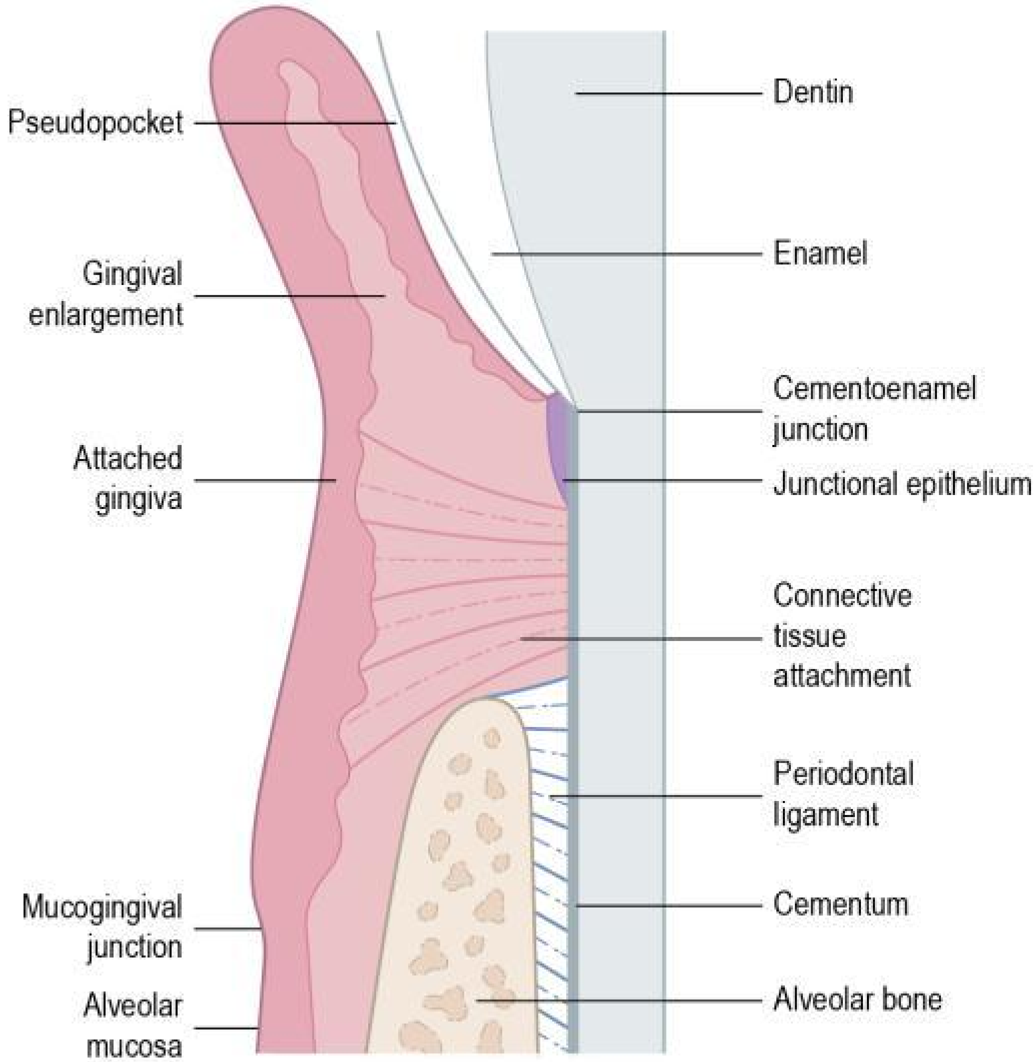
Lasers

Laser technology is evolving at a rapid pace, as are the attitudes toward its suitability for various dental applications. One respected human dental textbook has changed its outlook on the subject considerably from one edition to the next.[26,27] The most common laser in veterinary practice at the time of writing is the CO_2 laser. Compared to cold steel and radio surgery, laser incisions have more thermal necrosis and take longer to heal (see Chapter 19).[4,20-22,24] Laser should not be used close to the bone. There are safety concerns regarding reflected or misdirected beams and risk of damage to surrounding tissues such as enamel and cementum.[14] These factors aside, some surgeons familiar with and skilled in the use of laser technology have used it successfully for gingivectomy/gingivoplasty (see Chapter 10).

Therapeutic decision-making

Gingivectomy/gingivoplasty is used for the reduction of suprabony periodontal pocket depth and reshaping abnormal gingival contours. Gingivectomy is indicated in areas with an abundance of keratinized tissue, and no attachment loss or horizontal bone loss.[28]

In dogs and cats, the main indication for the technique is in the treatment of gingival enlargements such as idiopathic gingival hyperplasia, hereditary gingival fibromatosis, and drug-induced gingival overgrowth, which can be either focal or generalized. Gingival overgrowth and gingival enlargement are terms often used interchangably with hyerplasia, hypertrophy, and fibrosis. Histologically, gingval enlargement due to cyclosporine is characterized by both hypertrophy and hyperplasia, as well as increased extracellular matrix formation,[29] where hereditary gingival fibromatosis and gingival enlargement resulting from antiseizure medications and calcium channel blockers is primarily fibrotic.[30] Despite differing molecular changes depending on the underlying cause, the clinical appearance is similar. In these cases, excess gingival tissue results in the development of periodontal pseudopockets and abnormal gingival contours (see Chapters 19 and 46) (Fig. 20.7). The aim is to remove the gingival wall of the pseudopockets and restore normal gingival architecture. Excision of the entire pseudopocket, leaving a probing depth of 0 mm,31 is recommended in the current human periodontal literature, as during healing, gingival epithelial cell proliferation will result in reestablishment of a normal sulcus depth. However, if the incision is made too close to the alveolar margin, violating biologic width, bone resorption will occur. Therefore, a reasonable goal is to have 0–1 mm probing depth following gingivectomy. In cases of drug-induced gingival overgrowth, withdrawal of the causative drug will often result in dramatic improvement, and surgery may not be required. On the other hand, some residual enlargement may persist and gingivectomy/gingivoplasty may still be indicated.



• **FIG. 20.7** Cross-sectional anatomy of a pseudopocket resulting from gingival enlargement with no periodontal attachment loss.

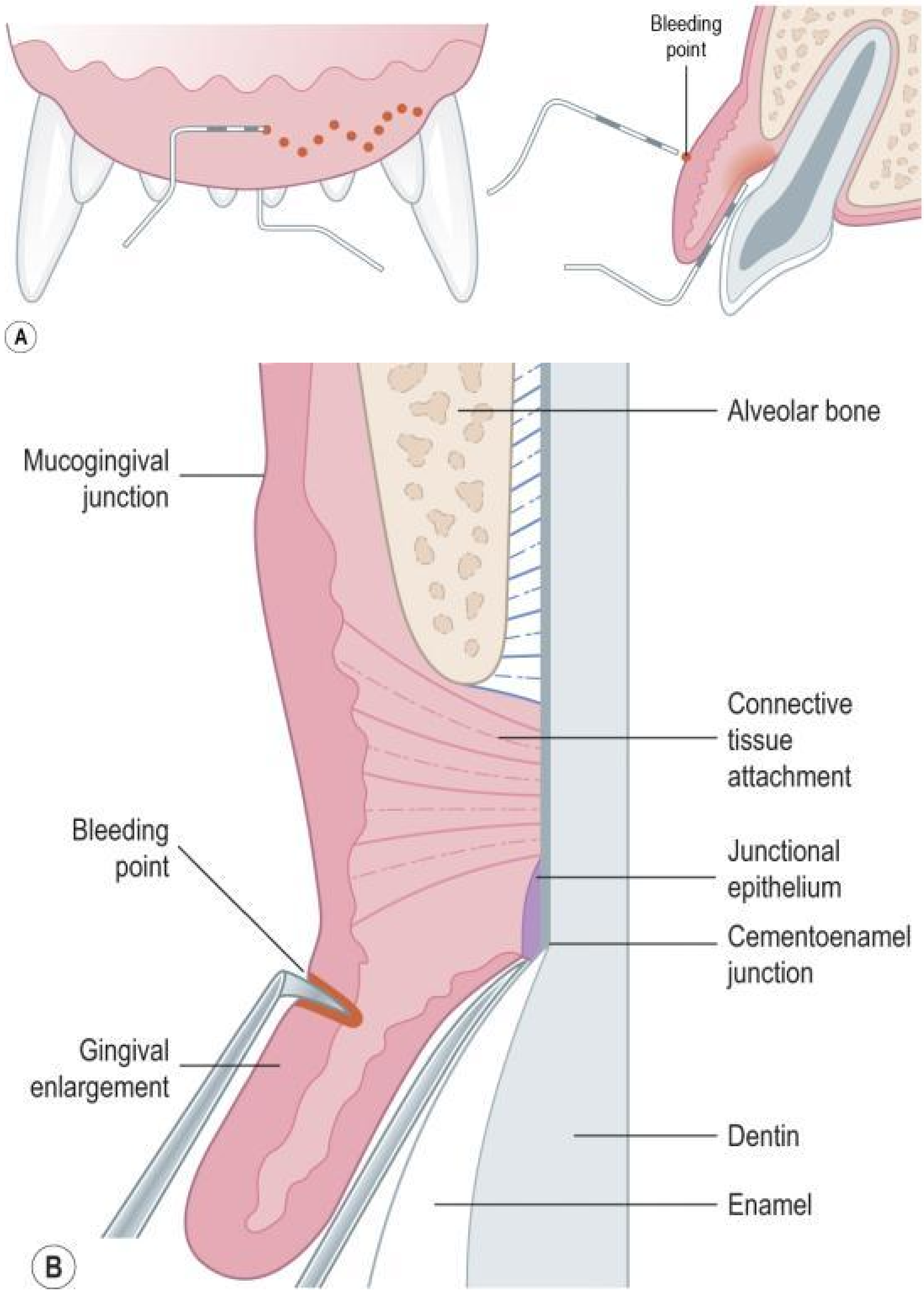
When attempting to reduce the depth of a true periodontal pocket to provide better access for scaling and root planing and home oral hygiene, gingivectomy/gingivoplasty must be applied cautiously, if at all. The goal of periodontal surgery is the long-term preservation of the periodontal tissues.[1,13,15] The periodontal health and future of a tooth rely on the maintenance of a collar of attached gingiva (minimum of 2 mm) around the entire circumference of the tooth.[13] Periodontal disease can result in the loss of gingival tissues through gingival recession. Excising gingiva further reduces the amount of gingiva available to support and protect the tooth. Reduction of the depth of true periodontal pockets should be attained through modalities intended to increase the level of attachment of the gingiva to the tooth (see Chapter 21). Gingivectomy/gingivoplasty should be limited to removing only small amounts of gingiva where an abundance of gingival tissue will remain after the incision, and healing and biologic width will not be compromised and furcations not exposed by the incision.

A third indication for gingivectomy/gingivoplasty is in type 1 surgical crown-lengthening, as outlined in Chapter 23. Again, this should be limited to the removal of only minor amounts of gingival tissue where an abundance will remain postoperatively and where the objectives of the surgery can be met without compromising biologic width or exposing furcations.[12,32]

In some circumstances, clinical and historical findings may make the histologic diagnosis seem obvious. For example, in a boxer dog with generalized gingival enlargements surrounding all teeth symmetrically, a diagnosis of familial fibrous gingival hyperplasia seems very likely. However, as one case report highlighted, making such assumptions can be dangerous. In the boxer dog presented, the tissues submitted for histologic examination reveal three separate diagnoses, namely fibrous gingival hyperplasia, fibrous epulis with osseous metaplasia, and mast cell tumor.[33] Therefore it is important to submit tissues for histopathology.[4,33] Having said that, it is very important to document the location from which each sample came and to keep the samples separate. If five pieces of tissue were submitted in one jar, and four were diagnosed as gingival hyperplasia and one as squamous cell carcinoma with incomplete excision, the location of origin of the malignant mass would be unknown and treatment planning would be impossible.

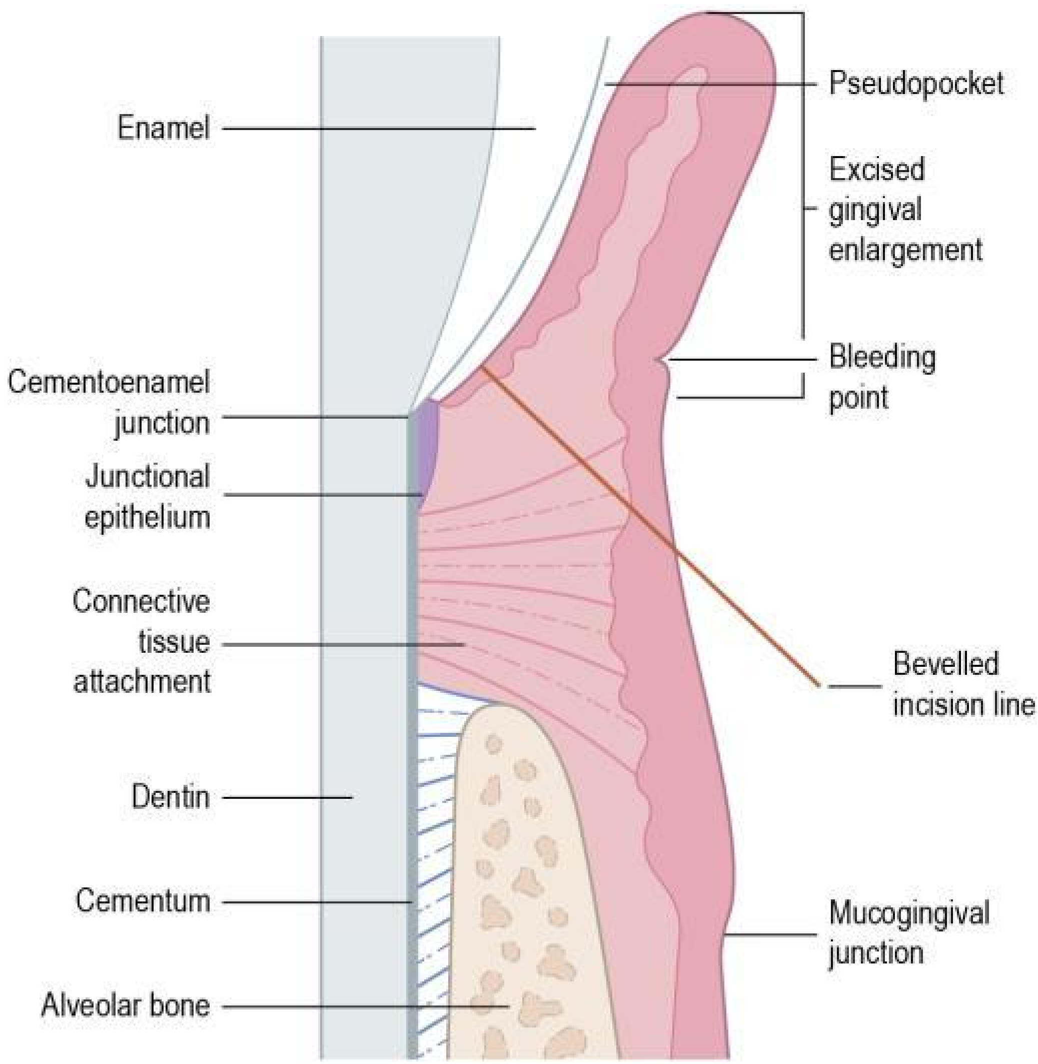
Surgical techniques

Regardless of the modality employed, the surgeon must plan the line of incision carefully. A method often described in the literature is as follows.[1,4,16] Pocket depths are measured with a graduated periodontal probe. The probe is withdrawn from the pocket and held against the outer surface of the gingiva to show the depth of the pocket. The tip of the probe is then turned horizontally and inserted into the gingiva to produce a bleeding point at the level of the bottom of the pocket (Fig. 20.8). Alternatively, pocket-marking forceps (see Fig. 20.4) may be used such that the probing beak of the instrument is placed parallel to the long axis of the tooth and inserted into the pocket. When the bottom of the pocket has been reached, the forceps are closed to pierce the gingiva on the oral surface, thus producing a bleeding point. The process is repeated along the whole circumference of the pocket, producing bleeding points at several points around each tooth, resulting in a dotted line for the surgeon to follow when making the incision (Fig. 20.8).



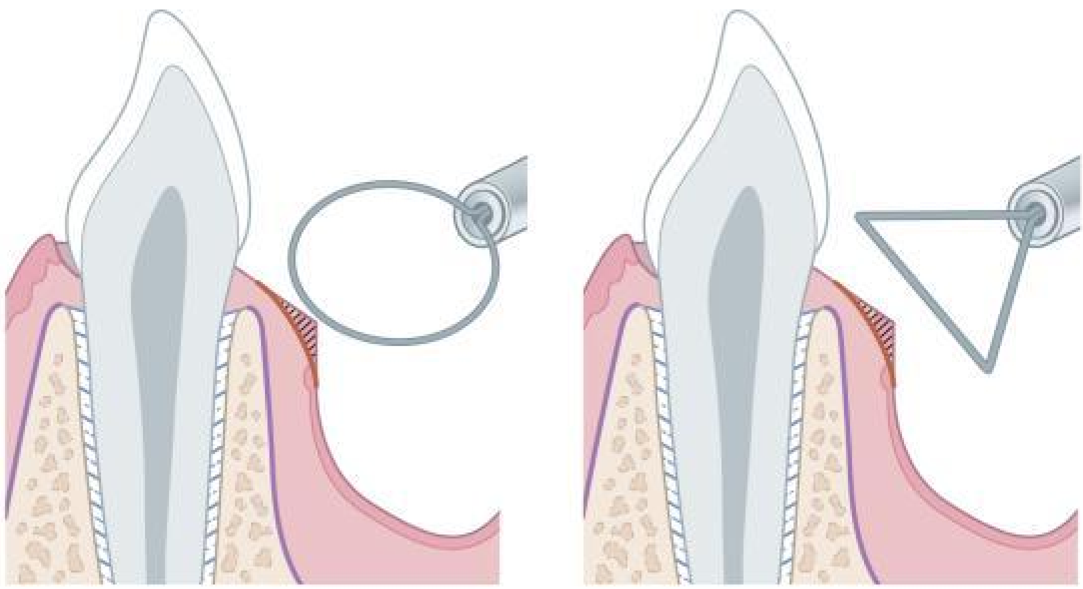
• **FIG. 20.8** The use of a periodontal probe (**A**) and Goldman–Fox periodontal pocket-marking forceps (**B**) to generate a line of bleeding points as a visual reference to guide the surgeon's initial incision.

The above methods are applicable when dealing with uncomplicated pseudopockets and relatively shallow periodontal pockets in areas with abundant attached gingiva. However, when a pseudopocket is combined with a significant true periodontal pocket, the resultant line of bleeding points may be too close to or even apical to the mucogingival junction, as well as apical to the furcations of multirooted teeth. To use these bleeding points as a guide for the line of excision would result in removal of excess gingival tissue and periodontal compromise of the tooth. In such instances, the surgeon should endeavor to keep the newly created free gingival margin 1–2 mm in dogs and coronal to the cementoenamel junction of each tooth (Fig. 20.9). Any clinically significant true pocket that persists is then managed with conservative or regenerative periodontal therapies.



• **FIG. 20.9** The correct angle and placement of the initial incision. The instrument enters the tissue slightly apical to the bleeding points and is angled coronally to meet the tooth coronal to the cemento-enamel junction. In this way, the dentogingival complex is preserved, biologic width is not compromised, and the cross-sectional profile of the gingival tissues is returned to normal. Probing depth following gingivectomy should be 0–1 mm.

When bleeding points are used as the guide, the primary incision, whether by blade, bur, or electrode, is made joining the bleeding points and recreating the scalloped edge of the normal gingival anatomy (see Figs. 20.1 and 20.3).[1,15] The initial, debulking incision starts with the blade or electrode entering the tissue on the oral side of the gingiva just apical to the bleeding points, with the instrument angled coronally as it penetrates the tissues to create a beveled incision (Fig. 20.9). The goal is to reestablish the normal gingival cross-sectional profile and a probing depth of 0–1 mm. Note that if laser, electrosurgery or radiosurgery is used, the probing depth following gingivectomy should be 1–2 mm, as tissue necrosis will occur and subsequently reduce the sulcus depth. Once the primary incision is completed on the buccal and lingual aspects of the teeth, the interproximal soft tissue is separated from the interdental gingiva by a secondary incision using a gingivectomy knife.[4,15] The incised tissues are carefully removed by means of a periodontal curette or a scaler. Remaining tissue tags are removed with a curette or a pair of scissors.[15] Further sculpting of the gingival profile may be accomplished with cold steel, dental burs, laser, or radiosurgery (Fig. 20.10 and see Figs 20.5, 10.8, and 10.9). Hemorrhage is controlled with gauze swabs and digital pressure. The crown and any exposed root surfaces are carefully scaled and polished.



• **FIG. 20.10** Following the initial incision, some final sculpting of contours and anatomy may be achieved by cold steel or by careful use of "hot" technique, shown here as a round or triangle loop in a radiosurgical unit used to plane the surface to the desired shape.

When the incisions are made with a radiosurgery unit, the electrode should be activated at the minimal effective power setting in fully filter (cutting) mode and stroked across the gingiva at the required angle. When using a radiosurgery unit, the incision should be placed approximately 1 mm coronal to the desired final result to allow for the postoperative tissue shrinkage that may occur with this technique. The cut surface should be pink and not bleeding if the setting is correct (Fig. 20.11). Blanched tissue indicates that the setting is too high and should be reduced.[34]

• **FIG. 20.11** (**A**) Preoperative photographs of the left and right maxillary canine and third incisor teeth of a 7-year-old spayed female boxer dog showing conspicuous gingival enlargements with pseudopocket formation. (**B**) Immediate postoperative photographs of these same teeth following radiosurgical gingivectomy/gingivoplasty using a fully filtered wave form and a C7B triangular loop electrode (see Fig. 20.6, *top*). Note the desirable anatomic form (height and profile), the relative lack of hemorrhage, and the lack of a visible char layer.

In veterinary dentistry, gingivectomy/gingivoplasty is usually performed using a combination of the methods. For example, gross debulking of generalized gingival hyperplasia may be done by cold-blade excision, while creating a physiological contour with the correct bevel may be done with a round diamond bur on a high-speed handpiece.

Periodontal dressings to protect the wound surface during healing are not generally used in veterinary practice as dogs and cats are disinclined to tolerate them. However, the open wounds can be painted with several coats of tincture of myrrh and benzoin (Tincture of myrrh and benzoin, Ellman International Inc., Oceanside, NY) as a topical anodyne.

Postoperative care and assessment

The postoperative phase is uncomfortable and analgesics are indicated for the first few days. It is crucial that plaque not be allowed to form on the tooth surfaces, as this will interfere with healing. Animals that have undergone this procedure are unlikely to accept tooth brushing immediately postoperatively, so nonmechanical plaque control is indicated (see Chapter 19).[35,36]

The healing of a gingivectomy/gingivoplasty wound is similar to that of a simple, open soft tissue wound except that there is a tooth in the center of the wound. During the inflammatory phase of healing, the underlying alveolar bone may be slightly resorbed. Superficially, healing is complete when the epithelium reaches the tooth. Epithelialization is generally complete within 14 days after surgery. This epithelium, however, is initially thin and nonkeratinized, with keratinization taking up to a month.[1] The normal gingival anatomy (epithelial attachment, gingival sulcus, and keratinized gingival epithelium) slowly reforms and the connective tissue matures. Complete healing generally takes 7 weeks.[1] However, on clinical examination, the surface of the gingiva may already appear healed after 13–15 days.[37] Optimal plaque control is required for healing. Regeneration of the lost bone does not usually occur during the maturation phase.

Meticulous plaque control by means of daily tooth brushing and/or the use of products that have the Veterinary Oral Health Council Seal of Acceptance for helping to control plaque (www.vohc.org). (see Chapter 19) is necessary to prevent or at least delay recurrence.[1,4,15,38] Regularly scheduled professional periodontal maintenance therapy should be part of the long-term plan.

Complications

With cold incision, complications may include excessive hemorrhage. This may be controlled by the application of direct pressure with saline-soaked gauze or the application of a topical hemostatic agent. Improper planning of the line of incision that infringes on the biologic width or exposes bone directly will result in undesirable tissue loss during healing.

Thermal injury to the teeth and adjacent structures can occur if the gingival resection is performed using electrosurgery, radiosurgery, laser, or high-speed burs.[1,4,24,27] This may result in sloughing of soft tissues and exposure of underlying bone. Use of excessive power with "hot" modalities can result in soft tissue necrosis as well as damage to bone; therefore postoperative sloughing and bone sequestration are further potential complications with "hot" incisions. Thermal damage and subsequent necrosis of the dental pulp are also possible with careless use of laser, radiosurgery, and electrosurgery.

Use of diamond and carbide burs can result in physical damage to the crowns and roots of the teeth if these cutting burs come in contact with the dental hard tissues.

Prognosis

Optimal home care is decisive for the long-term prognosis. In the absence of meticulous plaque control and without cessation of the inciting medication (in cases of drug-induced gingival enlargement), recurrence is common.

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CHAPTER 21

Periodontal flaps and mucogingival surgery

Kevin S. Stepaniuk, Barry B. Staley

Definitions

Apically positioned flap: A periodontal flap moved apical to its original location
Attached gingiva: Gingiva that is firmly attached to the underlying tooth and bone, extending from the free gingival groove to the alveolar mucosa
Coronally positioned flap: A periodontal flap moved coronal to its original location
Dehiscence: an area of tooth root denuded of bone, extending through the alveolar margin
Envelope flap: A full- or split-thickness flap used to access the root surface without vertical releasing incisions and replaced in its original position
Fenestration: An area of tooth root denuded of bone covered only by periosteum and overlying gingiva, with an intact alveolar margin
Free gingival graft: A split-thickness graft of an area of keratinized gingiva transplanted to another site where a bed is prepared; the periosteum, connective tissue and blood supply remain at the donor site, which is left open to heal by second intention
Free (marginal) gingiva: Gingiva that forms the soft tissue wall of the gingival sulcus and is not attached to the tooth surface
Full-thickness flap: A mucogingival-periosteal flap reflected from the alveolar bone, including the periosteum
Mucogingival junction: The junction between the attached gingiva and the alveolar mucosa
Pedicle (sliding or lateral) flap: A periodontal (often split-thickness) flap that remains attached to tissue and its blood supply and is moved in an apical, coronal, mesial, or distal direction away from its base
Periodontal flap: A section of gingiva and/or mucosa that is surgically separated from the underlying tissue and that retains at least one vascular attachment to the donor site
Reverse (or internal) bevel incision: An incision made at the crest of the free gingival margin through the connective tissue to the periosteum covering the alveolar margin
Split/partial-thickness flap: A flap consisting of a portion of the original tissue thickness (e.g., epithelium and a layer of connective tissue), leaving tissue (e.g., connective tissue and/or periosteum) on the bony surface

Subepithelial connective tissue graft (palate): A layer of connective tissue harvested from underneath the palatal epithelium and transplanted to another site where a connective tissue bed has been prepared

Sulcular (or crevicular) incision: An incision made from the base of the pocket, within the periodontal pocket, between the cementum and the epithelial lining of the sulcus, extending through the epithelial attachment to the crest of periodontal ligament-bone interface

Preoperative considerations

Periodontal flap

Appropriate patient and client selection is necessary for a predictable outcome with periodontal surgery. Home care and follow-up treatment are often necessary for success. The design of the periodontal flap will depend on the type of pocket (suprabony versus infrabony) and the subtypes of infrabony defects (one-, two-, or three-wall defects).

Periodontal probing and diagnostic imaging are necessary to diagnose periodontal disease, to determine the types of periodontal pockets, and to develop a treatment plan. Therefore, full-mouth probing of at least six measurements on each tooth (distobuccal, buccal, mesiobuccal, distolingual, lingual, and mesiolingual) and horizontal probing for furcation lesions, combined with a full-mouth series of intraoral radiographs, are necessary to make an accurate diagnosis.[1,2]

Depending on the relative size of the canine patient, identification of pockets greater than 4–5 mm necessitates periodontal treatment. Clinical studies have shown that scaling and root planing do not remove all of the subgingival plaque, and long-term maintenance is therefore compromised.[3] The removal of plaque and contributing factors to the accumulation of the plaque biofilm is necessary for successful periodontal therapy. Periodontal flaps allow visualization of the defect for treatment and removal of inflamed and diseased tissue.

Mucogingival surgery

Mucogingival lesions may be defined as developmental and acquired aberrations in the morphology, position, and/or amount of gingiva surrounding teeth. When considering mucogingival surgery and surgical methods, problems related to the band of keratinized attached gingiva, osseous defects or fenestration/dehiscence, periodontal pockets beyond the mucogingival junction, alveolar bone, tooth position in the dental arch, root exposure, and gingival thickness must be taken into account.

The evaluation and diagnosis of these issues require the use of the periodontal probe and intraoral dental radiographs, with special attention to the remaining attached gingiva and mucogingival tissue associated with the tooth and bone. The normal height and width of keratinized attached gingiva in the dog varies based on breed and location in the oral cavity such as the maxillary fourth premolar teeth, mandibular first molar teeth, and canine teeth.[4-6] Maintaining an attached gingival height of 2 mm is a suggested guideline for maintenance of periodontal health. When assessing a periodontal pocket with the periodontal probe, one must estimate the amount of keratinized attached gingiva by subtracting the pocket depth from the total keratinized tissue observed clinically. Likewise, it is necessary to compare intraoral radiographic findings with probing depths, to confirm that the periodontal probe is not penetrating the apical aspect of the periodontal pocket and penetrating deeper into an infrabony pocket, and to confirm that the periodontal probe is not penetrating past the mucogingival junction.

Surgical anatomy

Periodontal flap and mucogingival surgery

Landmarks

The key anatomical landmarks include cementum, sulcular epithelium, epithelial attachment, gingival connective tissue, free gingival margin, keratinized attached gingiva, mucogingival junction, alveolar mucosa, alveolar bone, periodontal ligament (PDL), and periosteum.

Differences between dogs and cats

There are several differences in dog and cat anatomy. The normal gingival sulcus for a cat is accepted to be 0.5–1 mm, and for the dog <3 mm. The cat oral soft tissues are thinner and more delicate. All periodontal surgery requires delicate tissue handling, meticulous skills, and appropriately sized surgical instruments for the species and patient size.

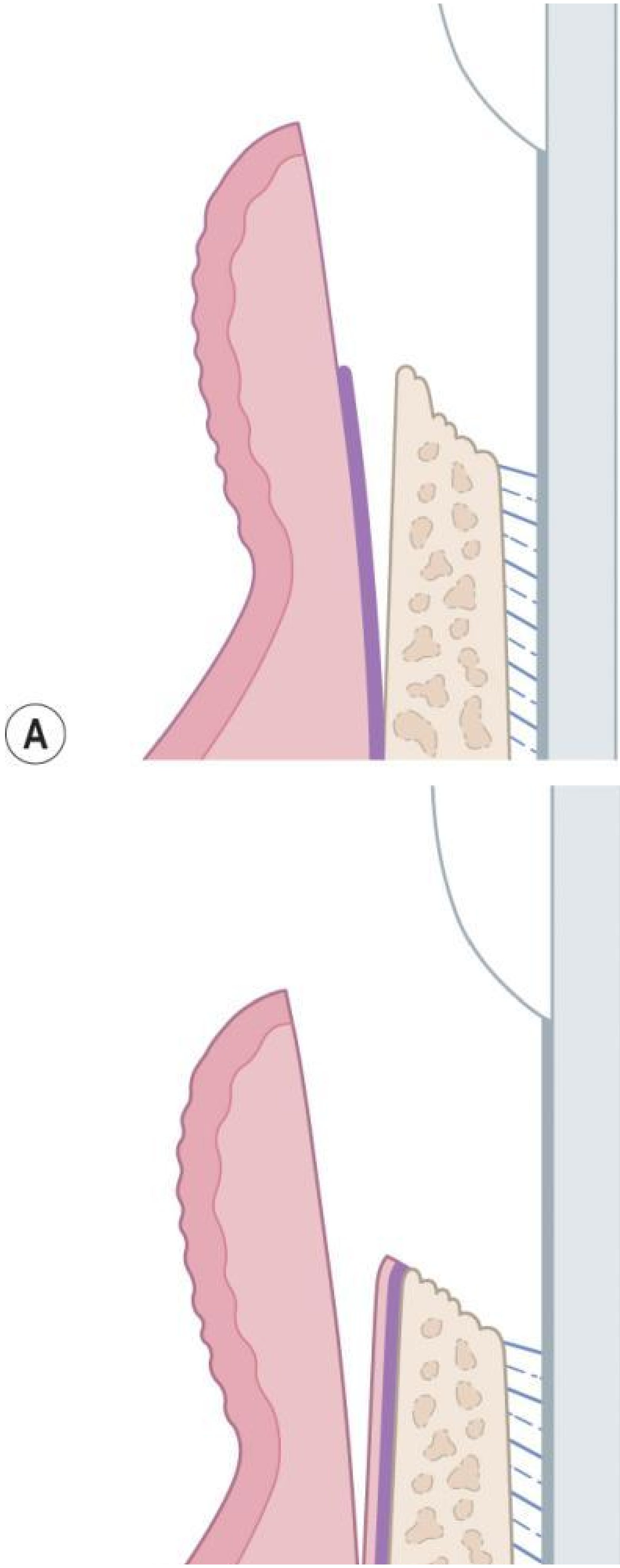
Special instruments

Selection of periodontal surgical instruments is commonly based on the individual surgeon's preference. A selection of instruments for periodontal surgery suitable for use in dogs and cats includes, but is not limited to, periodontal probe, shepherd's hook explorer, universal curettes, Gracey curettes, mini-Gracey curettes, scalpel handle, # 15C scalpel blade, # 11 scalpel blade, beaver blades and handle, Kramer-Nevis interproximal knife, small periosteal elevators, Kramer-Nevis tissue pliers, Bishop Harmon tissue forceps, Ochsenbein chisels, Goldman-Fox scissors, LaGrange scissors, small needle holders with tungsten carbide inserts, delicate tissue retractors, carbide and diamond burs on a high-speed dental handpiece, and small suture material (e.g., 5-0 or 6-0 poliglecaprone 25). In addition to these hand instruments, an ultrasonic scaler designed for periodontal debridement and subgingival use is indicated.

Therapeutic decision-making

Periodontal flaps

There are two types of flaps in periodontal surgery based on inclusion of the periosteum. A full-thickness mucoperiosteal flap includes the periosteum, whereas the split-thickness or partial-thickness flap does not (Fig. 21.1). A combination split/full-thickness flap also exists.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• **FIG. 21.1** (**A**) Full-thickness flap (used with infrabony pockets and apically positioned flaps; the periosteum is included in the reflected flap). (**B**) Split-thickness flap (used with suprabony pockets and apically positioned flaps; the periosteum is left on the bone).

Flaps are also classified by their positional movement and placement. The apically positioned flap is displaced apically from its original position. With the replaced (or nondisplaced) flap, the flap is returned and sutured to its original position. The apically positioned flap is used in procedures involving resection, whereas the replaced flap is used in surgical procedures for tissue regeneration.

A periodontal pocket over 4 mm deep is an area where subgingival plaque can accumulate and continue the inflammatory process, eventual tissue destruction, and periodontal attachment loss. Clinical research in the human dental field has demonstrated that it is difficult or impossible to predictably remove the accretions on a root surface in a pocket that is deeper than 4 mm. The clinician is thus faced with three possibilities if a probing depth greater than 4 mm is present: (1) maintenance as is, with poor long-term results; (2) reduction by subtraction—pocket resectional procedures; or (3) reduction by addition—regeneration of bone-PDL-cementum.[7]

Classification of the type of pocket using the periodontal probe and intraoral radiographic analysis will dictate the flap design. Suprabony pockets with hyperplastic tissue (no bone loss) can be reduced by gingivectomy (see Chapter 20). Suprabony pockets with horizontal bone loss will be approached with split/full/split-thickness flaps and osteoplasty/ostectomy.

Most of the infrabony pockets will be approached via mucoperiosteal flaps to gain access to the tooth surface and alveolar bone to perform osteoplasty and/or regeneration procedures (see Chapter 22).

One-wall and some two-wall bony defects are treated by means of a split-thickness flap and osteoplasty/ostectomy, with the flap apically positioned to the height of the new bone margin. This flap is a split/full/split-thickness flap and will be described in the surgical techniques section to follow. The goal of ostectomy is to make the marginal interproximal bone flat mesial-to-distal and buccal-to-lingual. The interproximal bone should be occlusal to or at approximately the same level as the radicular bone. The opposite is described as reverse architecture and will result in continued soft tissue pocketing. The treatment objective of the parabolic bony architecture will be influenced by the size of the embrasure, because it is more difficult to control soft tissue growth in a narrow interproximal site. The osteoplasty and ostectomy are performed with small round diamond burs, piezosurgery, files, and chisels.

Indications for apically positioned flaps are (1) eliminating pockets (suprabony and one- and some two-walled infrabony defects), (2) preserving keratinized attached gingiva and increasing its width, and (3) establishing gingival morphology to facilitate good hygiene.

Contraindications include (1) some two- and three-walled infrabony defects (suitable for regenerative procedures), (2) multiple deep periodontal pockets, (3) teeth with marked mobility and severe attachment loss, (4) teeth with an unfavorable clinical crown:root ratio, and (5) lack of keratinized free gingiva or attached gingiva to move apically.

Some two- and three-wall osseous defects can be treated by bone regeneration techniques (see Chapter 22). The access flap for these procedures is a full-thickness mucoperiosteal flap.

Indications for a replaced flap and regeneration are (1) to differentiate two- and three-walled defects, (2) to restore lost attachment (bone-PDL-cementum), (3) to avoid gingival recession, and (4) to decrease pocket depth.

Possible concerns include the fact that these are technically demanding procedures, which require a longer anesthetic period. The patient should be a suitable candidate for multiple anesthetic procedures because additional periodontal surgery and follow-up periodontal procedures are often necessary. Client and patient compliance for postoperative plaque control is essential.

Mucogingival surgery

Mucogingival surgical procedures correct or eliminate anatomic, developmental, or traumatic lesions of the gingiva or alveolar mucosa. These are conditions associated with keratinized attached gingiva, shallow vestibules, frenulum interfering with free gingival margins, and oral tissue trauma.

Evaluating the patient for an adequate zone of keratinized attached gingiva is important because the keratinized attached gingiva has dense connective tissue fibers with less vascularization and is therefore more able to resist the initiation and progression of the inflammatory process in periodontal disease. By comparison, the loose nonkeratinized alveolar mucosa is less fibrous and more vascular. This is of particular significance because inflammation extends perivascularly.[7] Most veterinary patients do not receive consistent home care and quarterly recall periodontal cleanings. Any area with plaque build-up in the presence of gingival recession is an area of concern, and mucogingival surgery should be considered an option.

The normal dimensions of keratinized attached tissue and the ideal labial-lingual width of the alveolar process (thickness of the periodontium) have a significant effect on mucogingival problems. Therefore, inadequate keratinized attached gingiva or periodontal dehiscence may predispose a tooth to recession with inflammation or trauma. In the absence of disease, recession may not occur despite the predisposition. Malpositioned and crowded teeth are likely to have inadequate keratinized attached gingiva and dehiscence or fenestration of the alveolar bone.

Indications for mucogingival surgery are (1) to create an adequate zone of keratinized attached gingiva, (2) to establish a strong keratinized attached gingiva that protects the mucogingival complex during mastication of hard food and objects, (3) to restore gingiva in areas of recession, and (4) to cover dehiscence and fenestration. Adequate interdental papillae and interdental alveolar bone adjacent to gingival recession and a sufficient blood supply at the donor site are prerequisites.

Contraindications include (1) an insufficient width and thickness of keratinized tissue at the donor site, (2) gingival recession in an area that is extremely protrusive, (3) deep periodontal pockets and loss of interdental alveolar bone adjacent to the recipient site, (4) a narrow and shallow vestibule, (5) a deep and wide recession area, (6) involvement of juxtaposed multiple teeth, (7) uncontrolled periodontal disease, (8) severe periodontal attachment loss, and (9) poor client/patient plaque control.

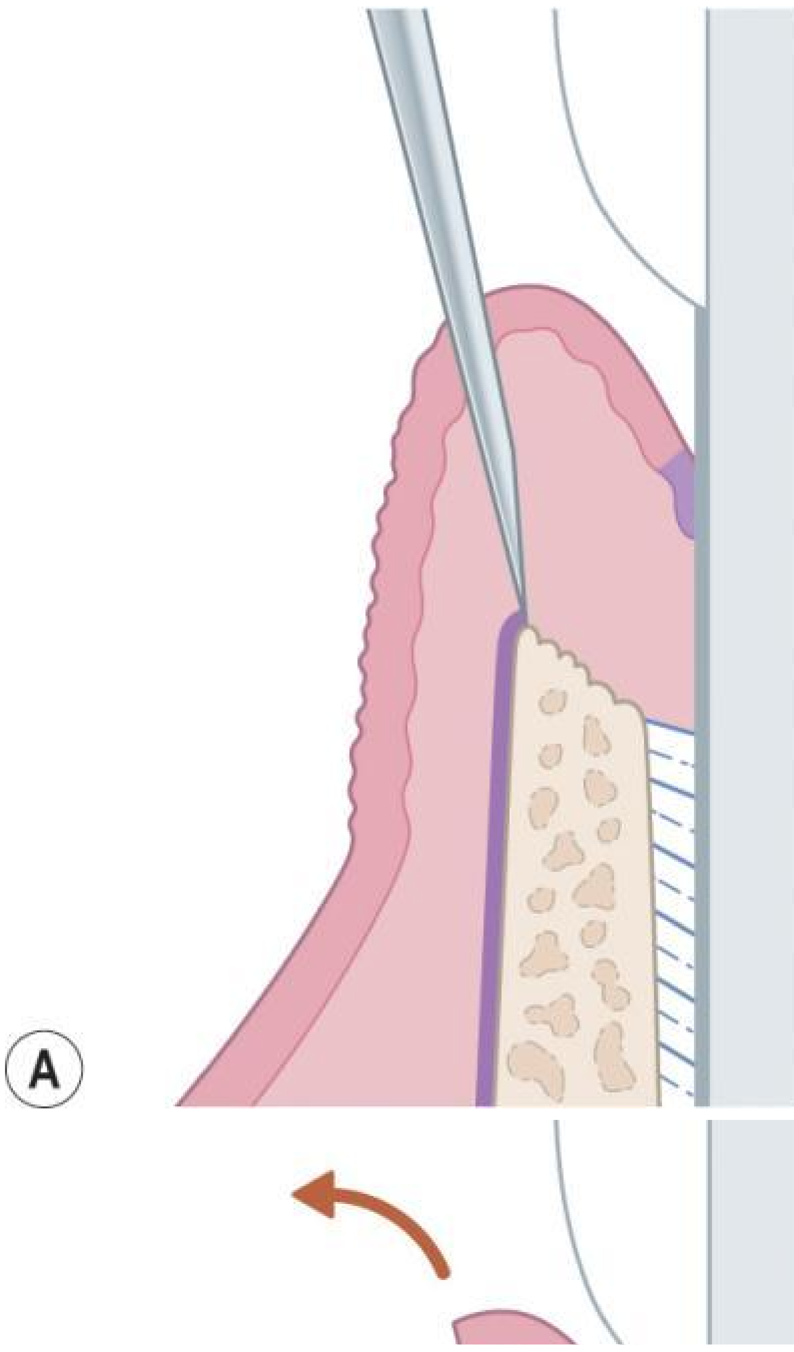
Surgical techniques

Periodontal flap surgery

Apically positioned flap

Initial incision and releasing incisions

The initial incision, using a # 15C scalpel blade in the dog or a beaver blade in the cat, is a reverse-bevel (split-thickness) incision starting at the crest of the free gingival margin. The incision is continued to the margin of the alveolar bone (Fig. 21.2). Vertical incisions are made one tooth mesial and one tooth distal to the defects, making sure the vertical incisions are at the line angles of these teeth.[8] Vertical incisions are not made over furcation areas or in the middle of the interproximal space. If made over the furcation area, the furcation may be exposed; in this case, the tissue must be replaced and not apically repositioned. If the incision is made in the middle of an interproximal space, the soft tissue will tend to fill in the space and recreate a pocket. In the absence of neighboring teeth, the coronal incision is extended for 6–8 mm before the vertical incision is made.



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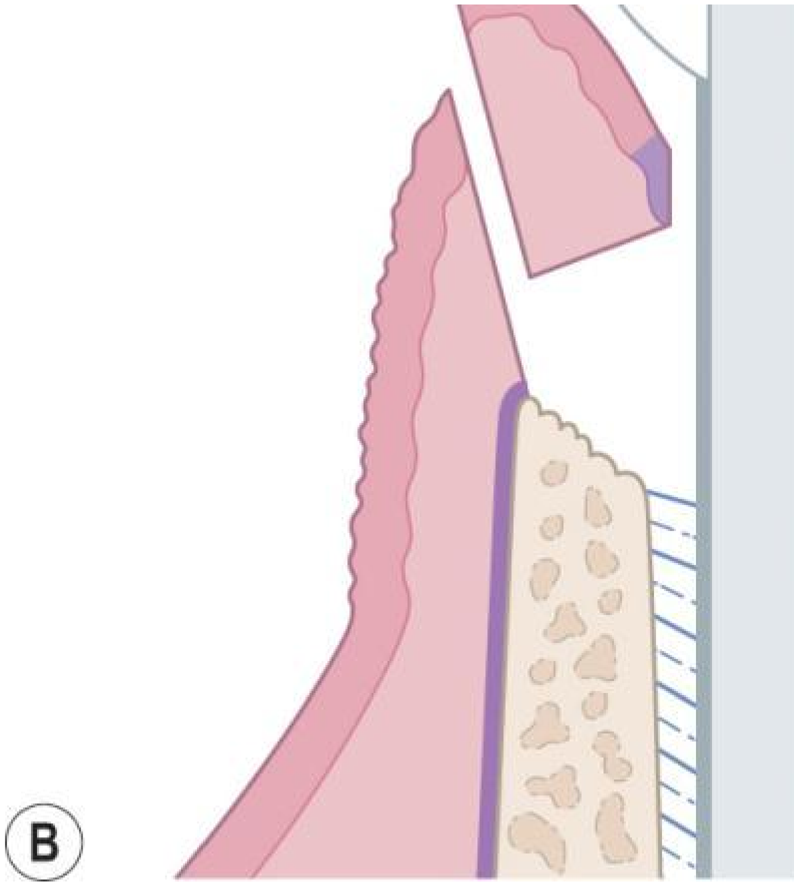
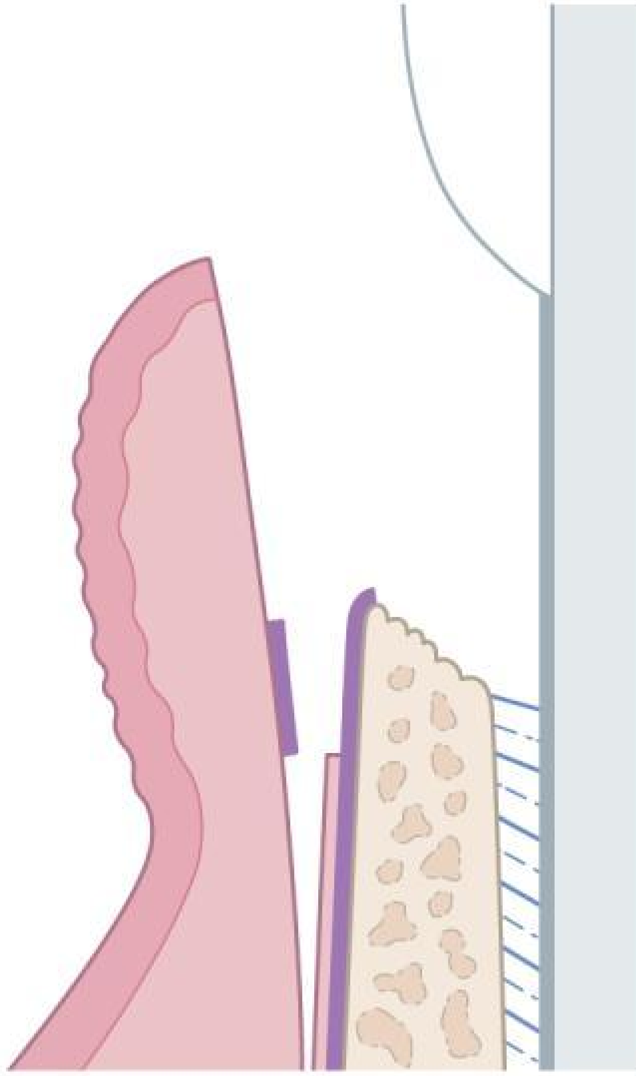


• **FIG. 21.2** (**A**) Reverse-bevel incision (used with split-thickness, apically positioned, coronally positioned, and pedicle flaps). (**B**) Removal of sulcular epithelium.

Elevating the flap

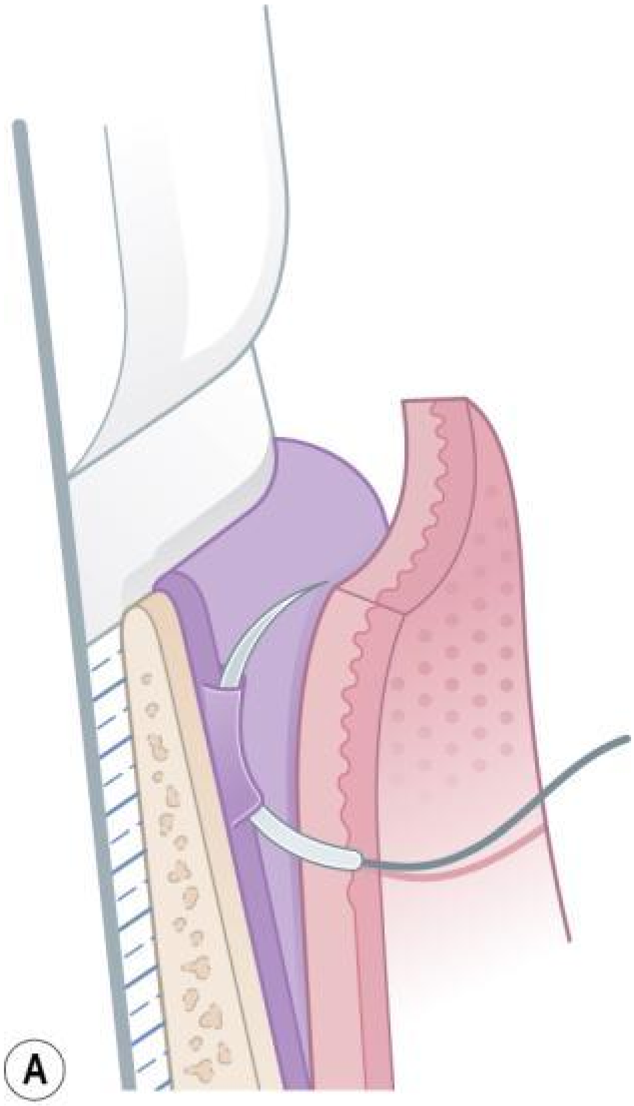
The attached gingiva, including the periosteum (full-thickness), is reflected up to the mucogingival junction using a periosteal elevator. Flap elevation is then continued apically by means of (spilt-thickness) dissection, using a # 15C scalpel blade, in the alveolar mucosa, leaving the periosteum on the bone. This is a split/full/split-thickness flap and is the most commonly used for pocket reduction and access to alveolar bony defects for osteoplasty/ostectomy procedures. Any collar of tissue remaining from the initial reverse-bevel incision is removed with a scaler. The roots are scaled and root planed and any osseous recontouring is performed as necessary. The flap is then apically positioned at the new margin of bone (Fig. 21.3).



• **FIG. 21.3** Split/full/split-thickness flap: the flap is split-thickness through the free gingiva, full-thickness to the mucogingival junction, and split-thickness in the alveolar mucosa.

Suturing of the apically positioned flap

A common suture choice is 5-0 poliglecaprone 25. A periosteal tacking suture (vertical mattress) is used. The needle enters the alveolar mucosa, then in the same motion, the alveolar mucosa is gently sutured to the intact periosteum and the needle rotation is continued to exit in the keratinized tissue near the coronal margin (Fig. 21.4). The suture is then passed through the interproximal space to include the lingual papilla, returned through the same interproximal space, and tied on the buccal surface.[9] This is a positional suture that should not be tied too tightly in the interproximal space. The object is to position the flap at the exact position of the new height of bone after osseous surgery, thereby covering the alveolar margin and apically positioning the flap from its original position (Fig. 21.5). The reason one performs the split dissection in the alveolar mucosa portion of the flap is to be able to tack the flap to the periosteum and to secure the flap during the healing process. After the flap is tacked to the correct apical position, the vertical incisions are sutured with interrupted sutures, always suturing loose tissue to fixed tissue, starting with the coronal keratinized tissue.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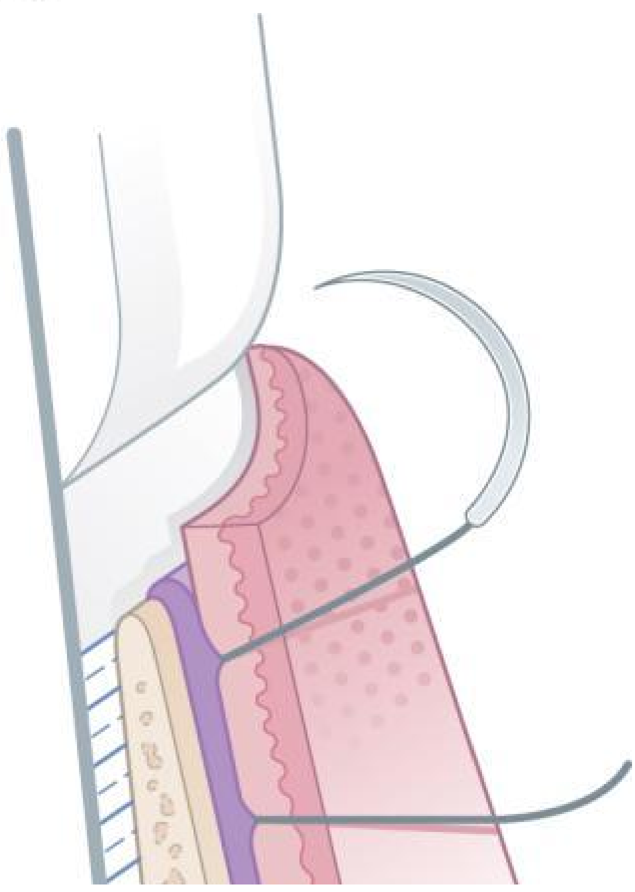

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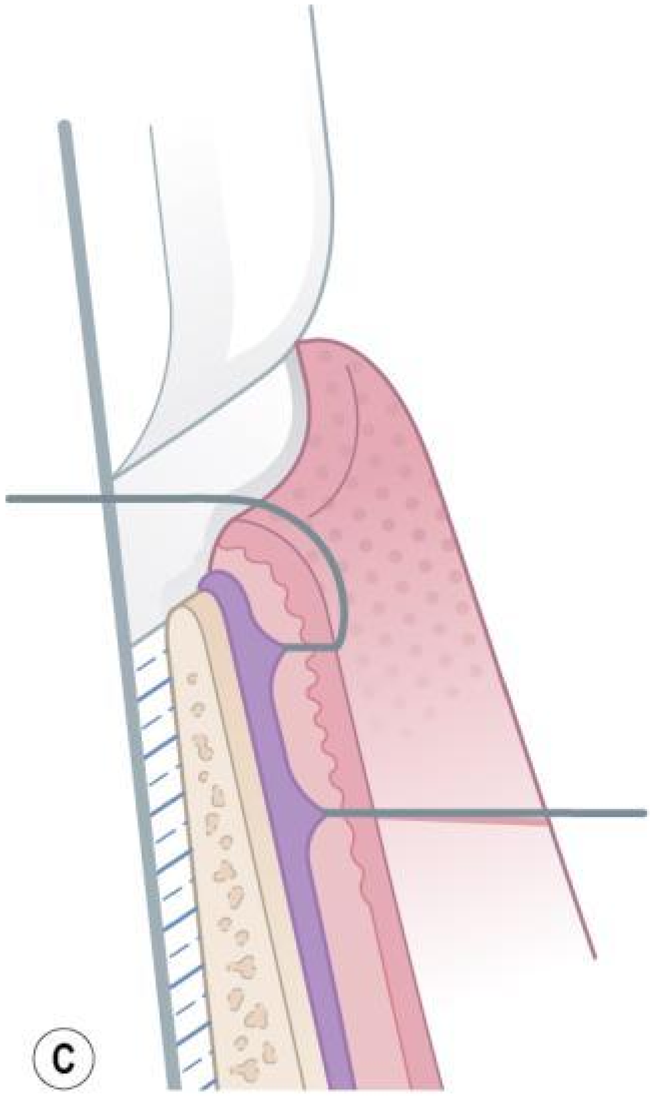
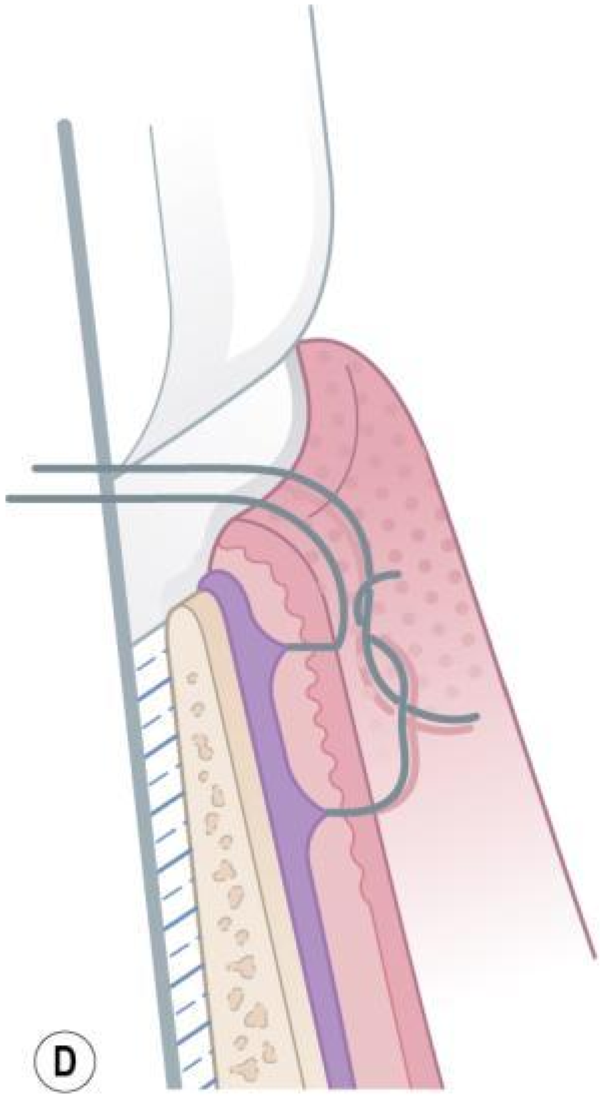
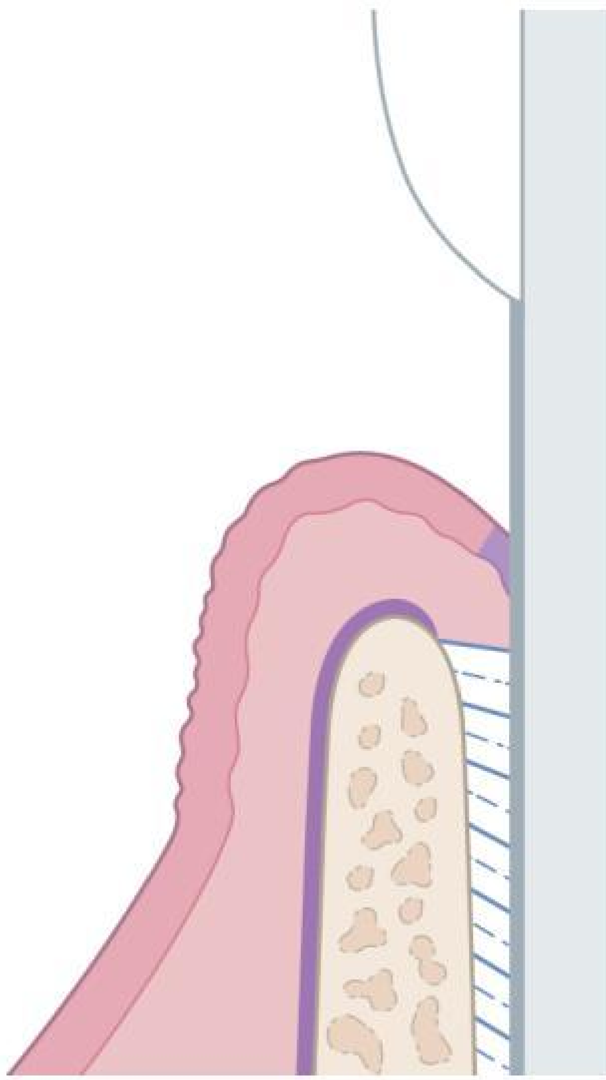




• FIG. 21.4 Suturing of the apically positioned flap: **(A)** Suturing the flap to the periosteum. **(B)** The suture exits through keratinized gingiva. **(C)** The suture is passed through interproximal tissue. **(D)** The suture is passed back through the interproximal tissue and tied on the buccal aspect.

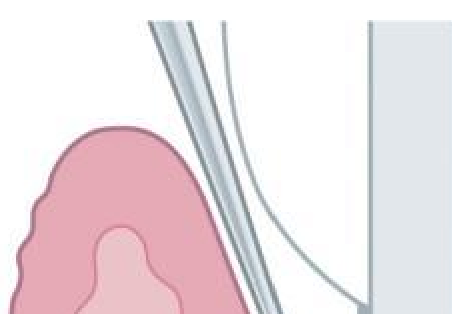


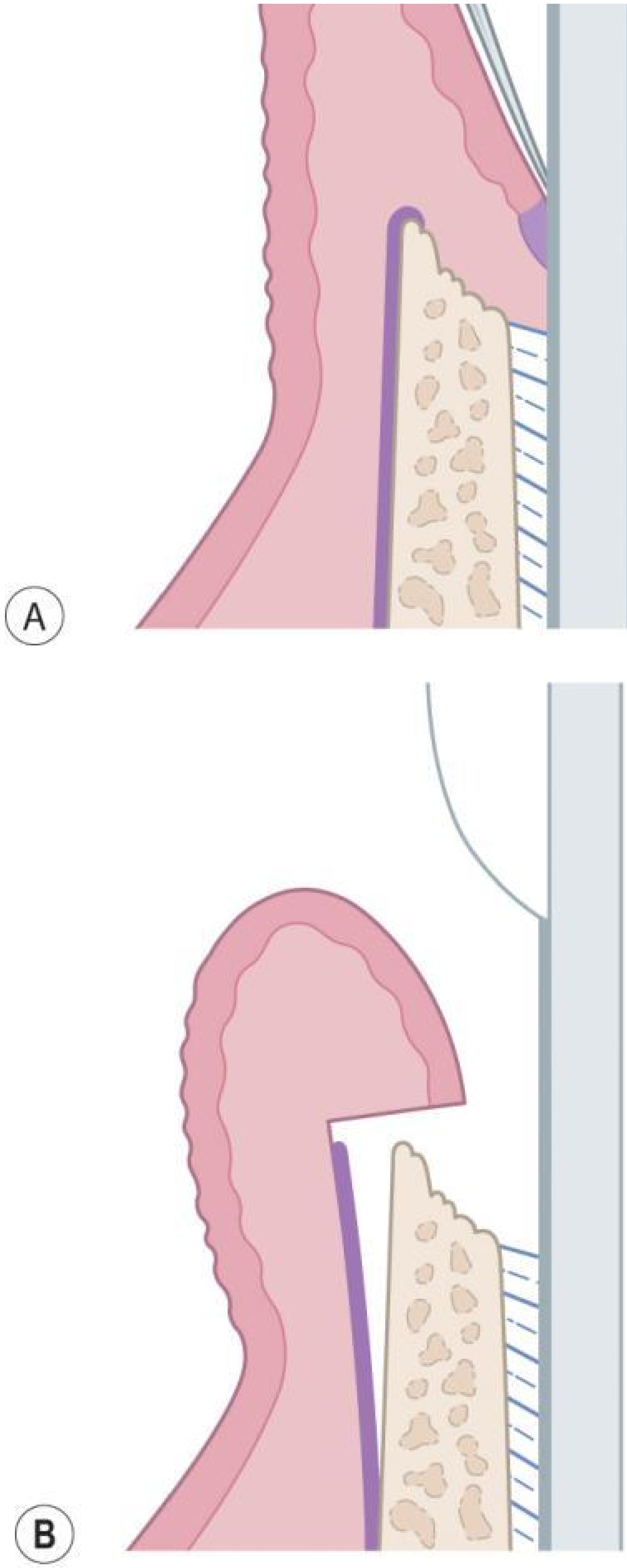
• **FIG. 21.5** Apically positioned flap (repositioned at the new osseous margin).

Full-thickness flap for regeneration procedures

Initial incision and releasing incisions

The initial incision is a sulcular incision within the periodontal pocket between the sulcular epithelium and cementum of the tooth. It extends through the epithelial attachment and to the PDL-bone interface (Fig. 21.6A). Vertical incisions are made one tooth mesial and one tooth distal to the defects, making sure the vertical incisions are at the line angles of the neighboring teeth.



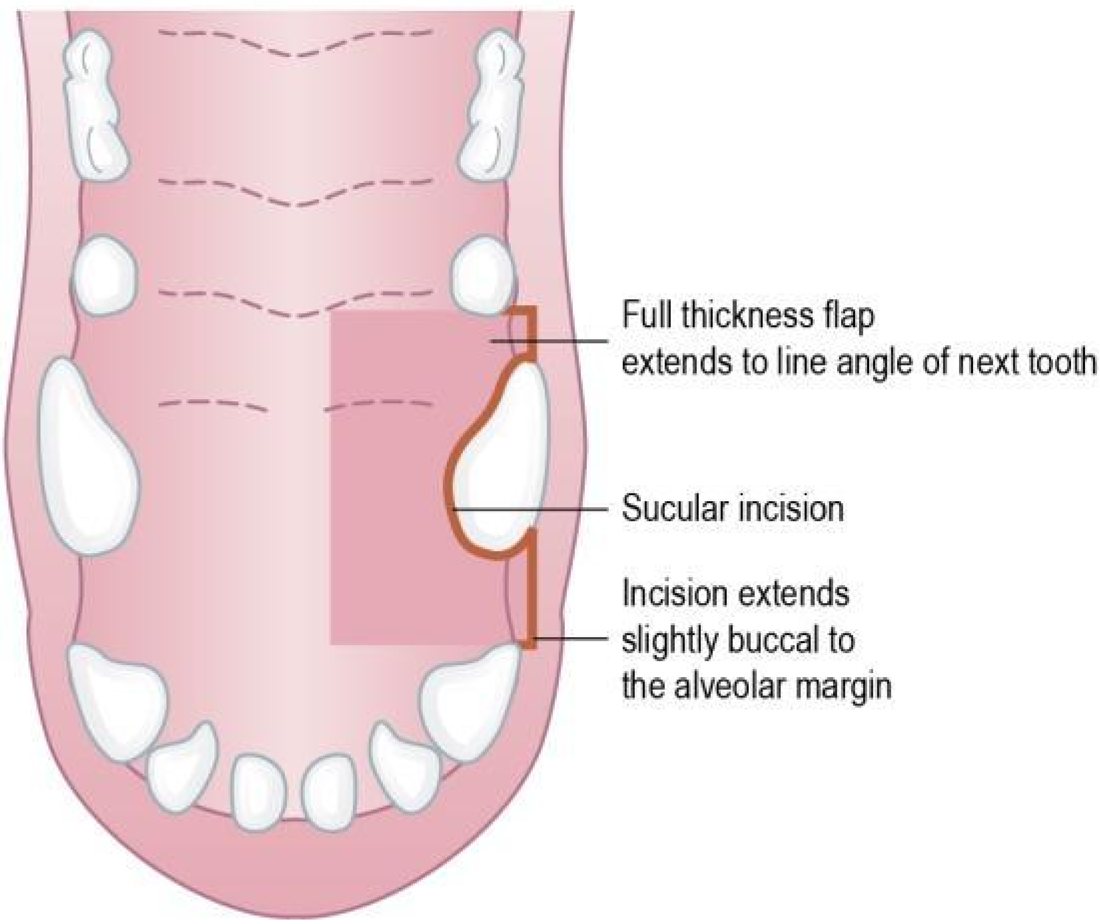


• **FIG. 21.6** (**A**) Sulcular incision (used with full-thickness flaps and three-wall infrabony defects). (**B**) Reflected full-thickness flap with sulcular epithelium.

Elevating the flap

The flap is then reflected, using a periosteal elevator, with the periosteum off the bone (Fig. 21.6B). The root planing and bone grafting procedures are performed and the full-thickness flap is replaced at its original position.

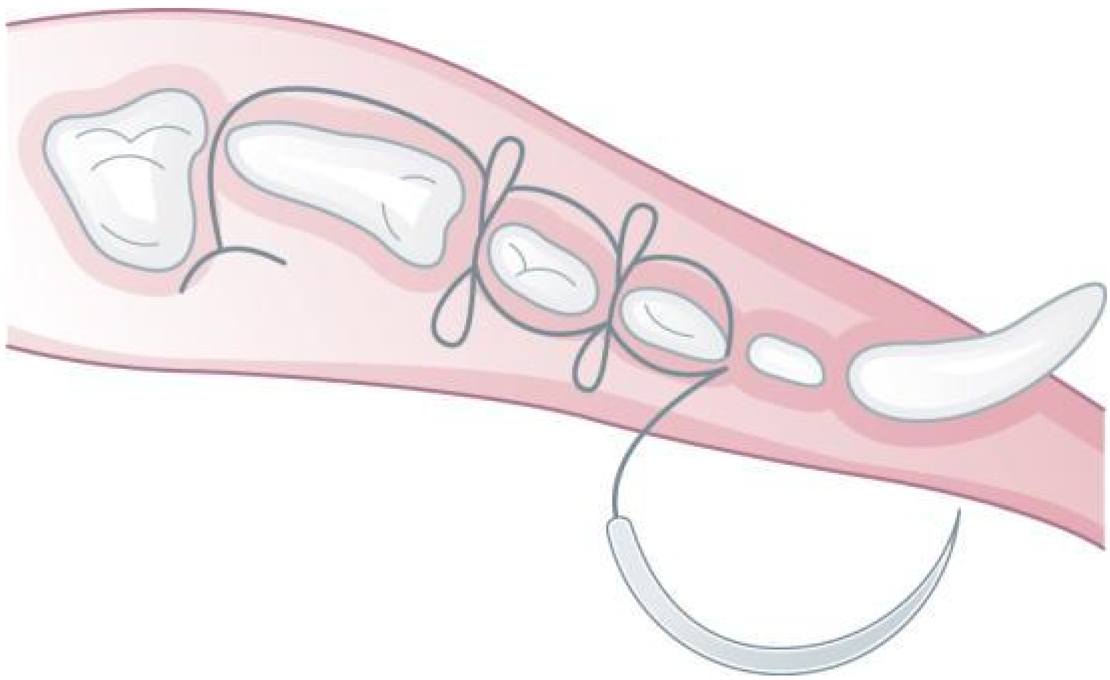
The palatal aspect of the maxillary canine tooth often has a three-walled infrabony defect that is amenable to bone regeneration procedures. A flap design that can be used for this purpose is an extended envelope flap, thereby avoiding the need for releasing incisions in the palatal mucosa (Fig. 21.7).[10,11]



• **FIG. 21.7** Palatal flap (used with three-wall infrabony pockets and bone grafting).

Suturing the full-thickness flap

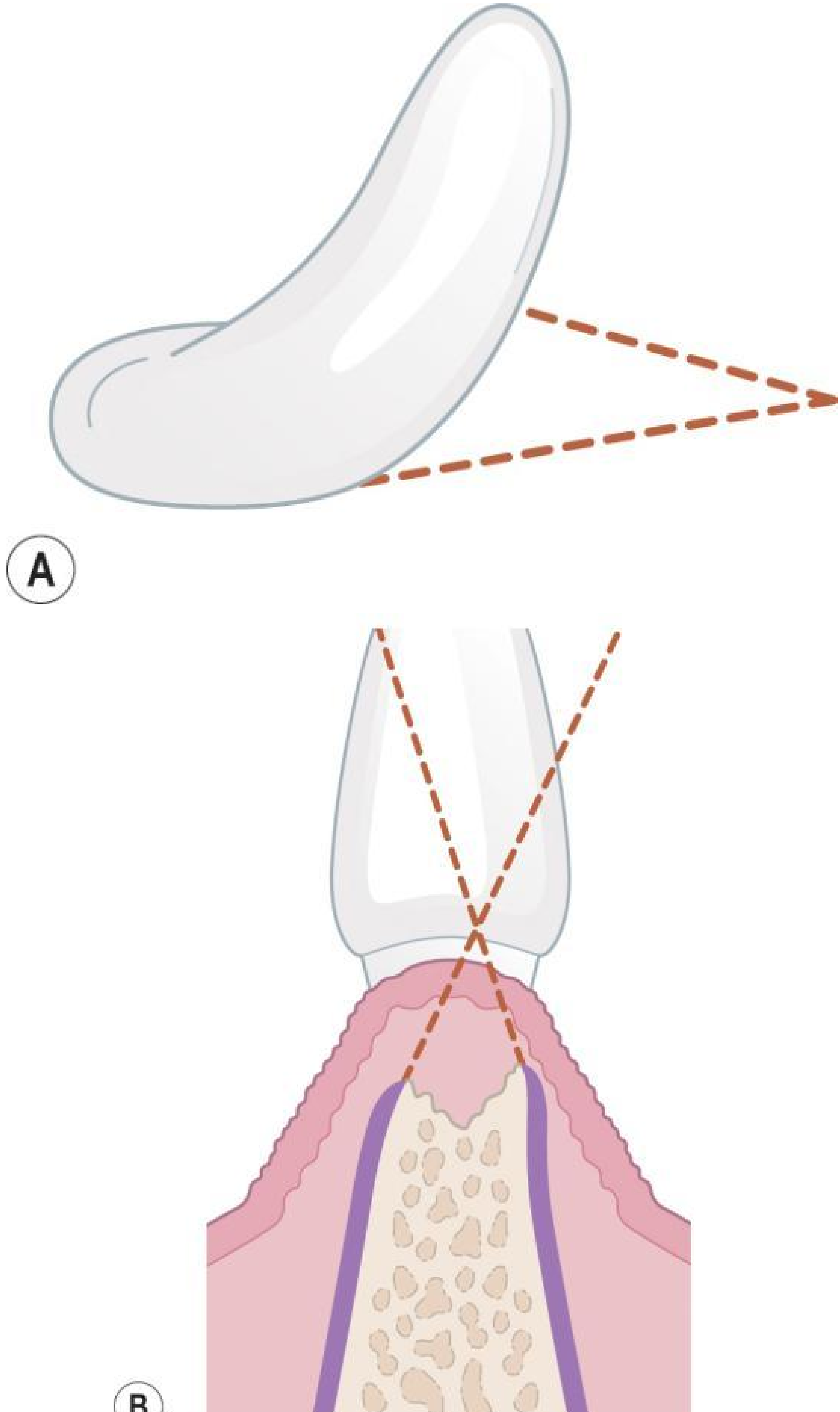
Interrupted sling sutures are used interproximally, picking up the keratinized tissue on the buccal and passing through the interproximal space to suture to the lingual or palatal tissue. The suture is then returned through the interproximal space and tied on the buccal aspect. The vertical releasing incisions are sutured via interrupted sutures, tacking keratinized attached tissue to keratinized attached tissue, and alveolar mucosa to alveolar mucosa. In most periodontal suturing, it is not important to have total primary closure; however, in bone grafting and connective tissue regeneration procedures, the initial sulcular incision tries to preserve as much of the interproximal soft tissue as possible, which is then positioned and sutured to allow primary closure (Fig. 21.8).



• **FIG. 21.8** Continuous sling suture.

Mesial and distal wedges

Full-thickness mesial or distal wedges are used when pockets are on the mesial or distal aspects of a tooth next to an edentulous area. The mesial and distal aspect of canine teeth is where this may be of benefit (Fig. 21.9).[12]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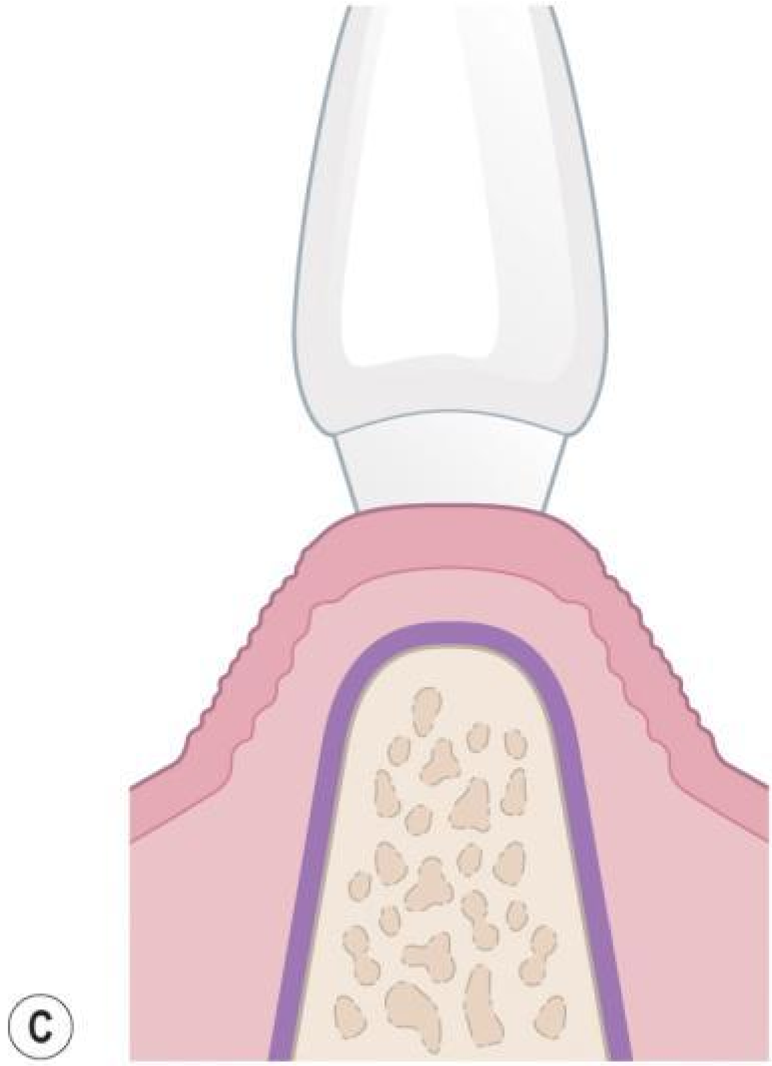


• FIG. 21.9 (**A**) Mesial wedge excision. (**B**) Angled incisions to excise the wedge. (**C**) Tissue apposition following wedge excision, osteoplasty, and apically positioned flap.

Mucogingival surgery

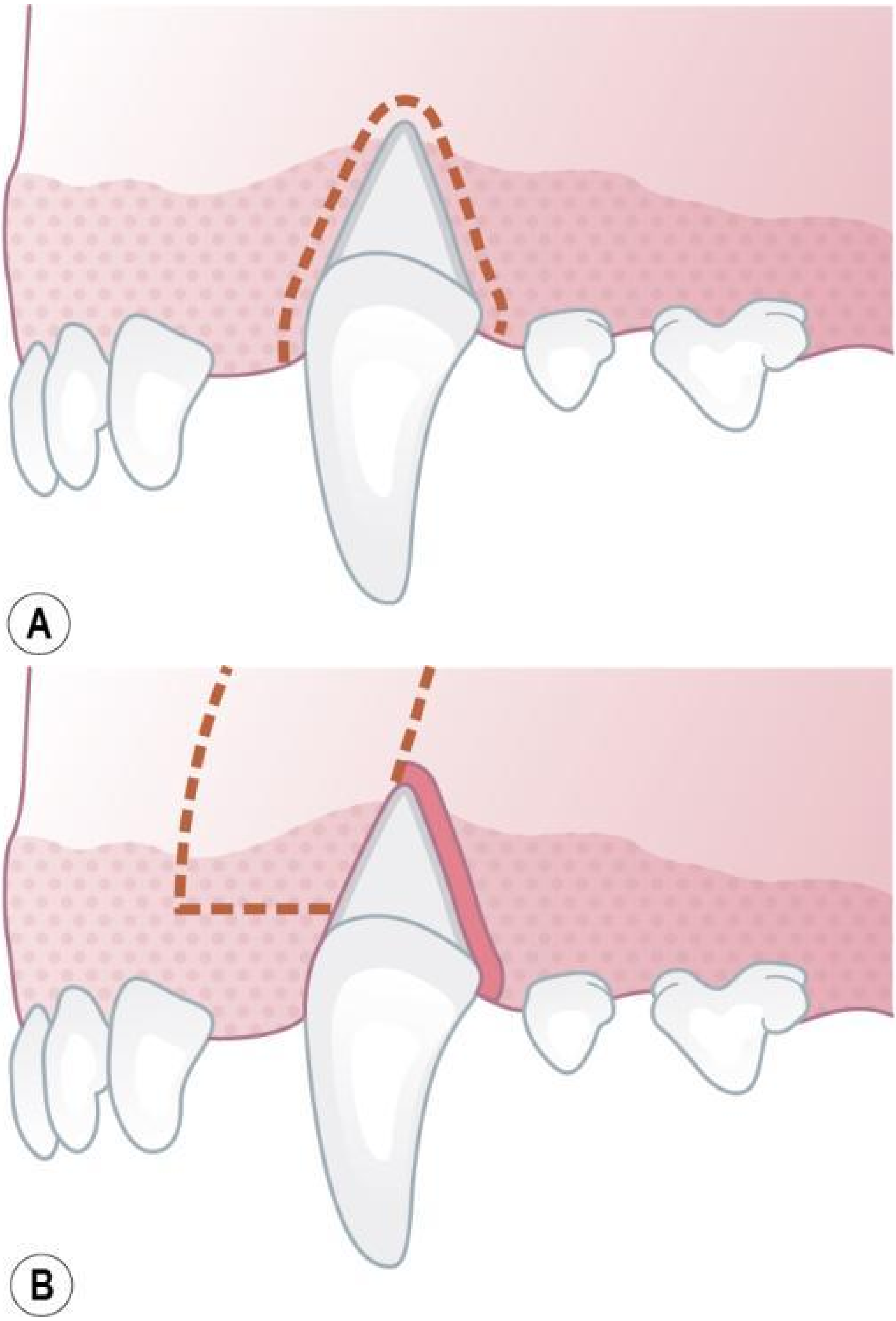
Increasing the zone of keratinized attached tissue in areas of recession or areas with less than 2–5 mm of keratinized attached gingiva (1–2 mm in cats) is the goal of therapy. This can be accomplished by pedicle, free gingival, and subepithelial connective tissue grafting.

Pedicle/sliding/laterally repositioned flap

One indication for a sliding flap is a tooth with recession and an inadequate zone of keratinized attached gingiva (e.g., gingival cleft/festoon), with one or more teeth on either side that have a more than adequate zone of keratinized attached gingiva. The neighboring tooth's keratinized attached gingiva can be used as a donor site, and the tooth with recession, as the recipient site.

Initial incision and releasing incisions

After scaling and polishing, a V-shaped incision is made following the peripheral margins of the free gingiva in the area of recession while preserving the gingiva on the mesial and distal aspects of the tooth (Fig. 21.10A). A wider beveled incision is made on the mesial aspect and an internally beveled incision on the distal aspect. This will create a close adaptation when the pedicle flap is advanced distally to cover the recession. A reverse-bevel incision starting 2 mm from the free gingival margin is made to the bony margin. The reverse-bevel incision is extended to two teeth mesially, approximately 1.5–2 times the width of the defect. Starting the incision 2 mm from the free gingival margin leaves a sufficient collar of keratinized attached gingiva coronal to the flap to protect the donor teeth. The flap should be at least 1.5 times wider than the recipient site.[13-15] The two vertical incisions are angled apically and distally. This will allow less tension on the flap when it is moved in a mesial or distal direction (Fig. 21.10B).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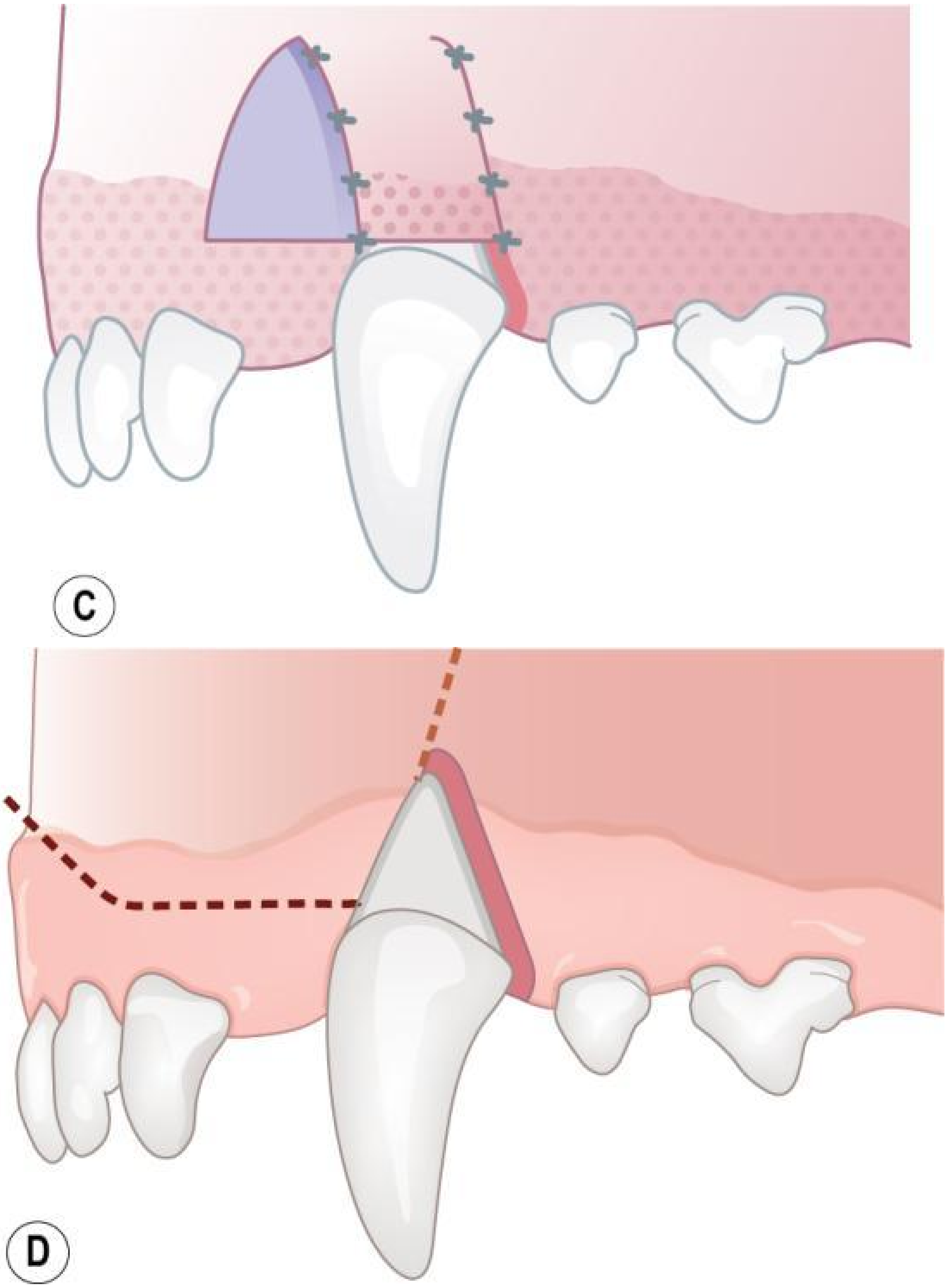
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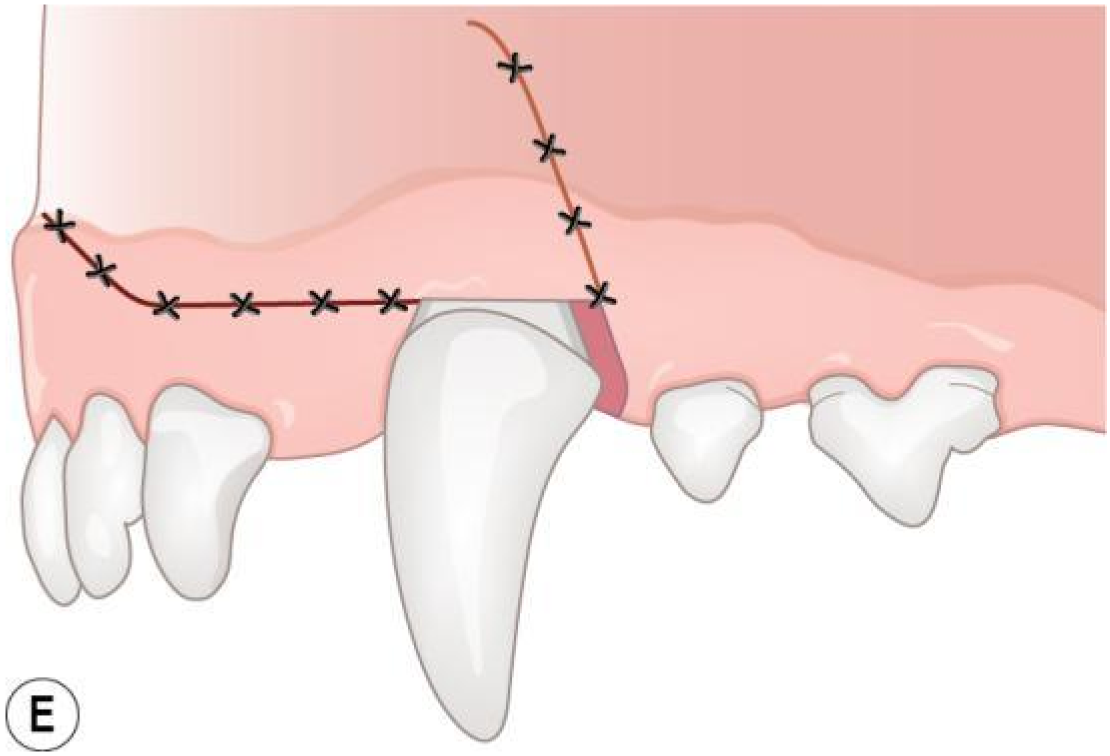


• **FIG. 21.10** Pedicle/sliding/lateral flap: (**A**) Excision of the sulcular epithelium. (**B**) Angled vertical incision toward the sliding direction. (**C**) Tacking sutures to the periosteum. (**D**) Flap design with a diverging incision beyond the mucogingival junction furthest from the defect. (**E**) The divergent incision allows suturing and closure of the donor site.

Elevating the flap

Reflect a full-thickness flap over the tooth adjacent to the recession; this part of the flap will cover the recession area. A split-thickness dissection is made under the flap on the second tooth mesial to the recession area, using a # 15C scalpel blade. This flap is one unit; however, it is full-thickness closest to the recession area and split-thickness in the area of the second tooth mesially. This will leave a periosteal covering at the donor site of the second tooth distally and will aid in the healing if the alveolar mucosa is not sutured closed. In dogs, it is possible to suture the alveolar mucosa closed by initially diverging the incision on the portion of the harvested flap furthest from the defect. The flap is moved distally and covers the area of recession to just over the alveolar margin or a little up the root surface. One should not try to cover the root completely, as the soft tissue will not reattach unless there is immaculate plaque control by the client. The goal is to increase the zone of keratinized attached gingiva over bone.

Suturing of the pedicle/sliding/lateral flap

The pedicle flap is sutured at the coronal mesial points, first trying to match up the mucogingival junction of the mesial tooth to the keratinized attached gingiva of the pedicle flap. The flap is usually placed more apically. The mesial vertical incision is sutured (periosteal tacking) after the mesial and distal coronal interrupted sutures to the interproximal papilla have been placed. Periosteal tacking sutures are placed on the distal vertical aspect of the flap into the exposed periosteum covering the second tooth distal (Fig. 21.10C).

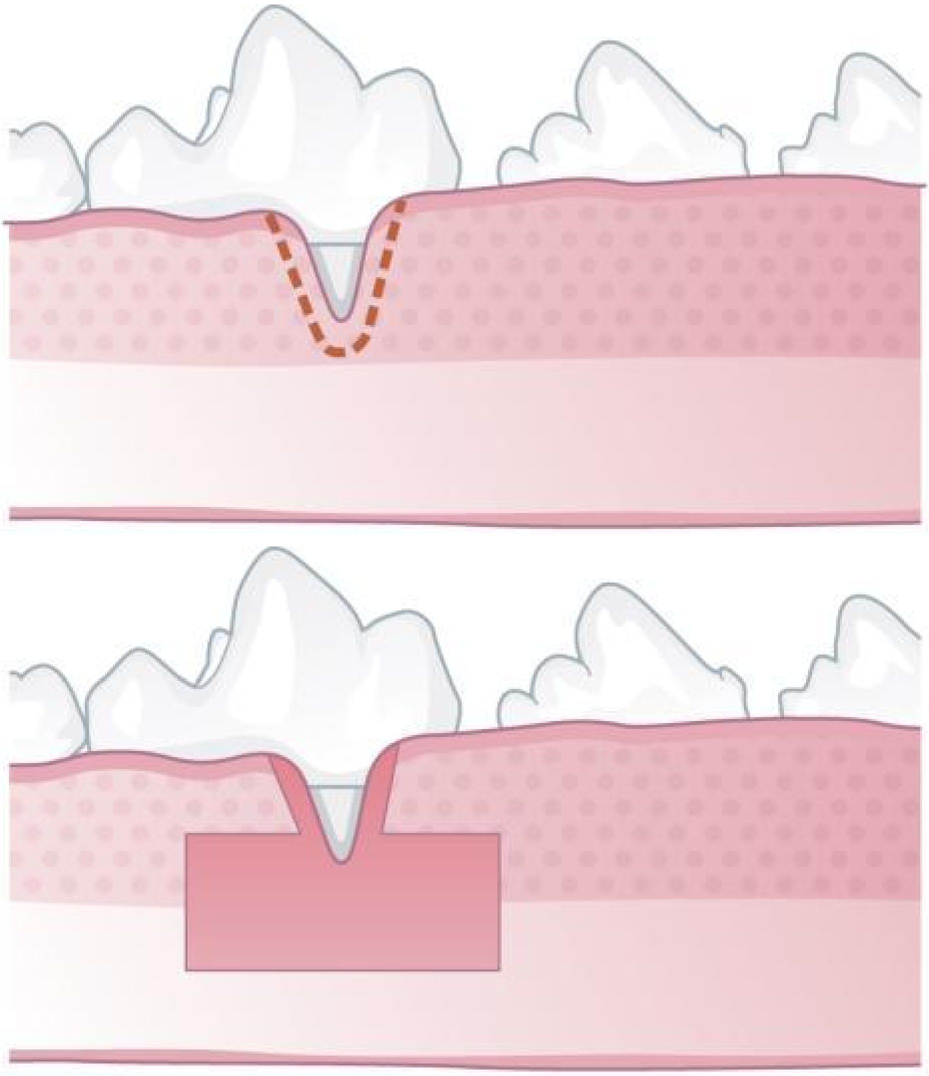
Free gingival graft

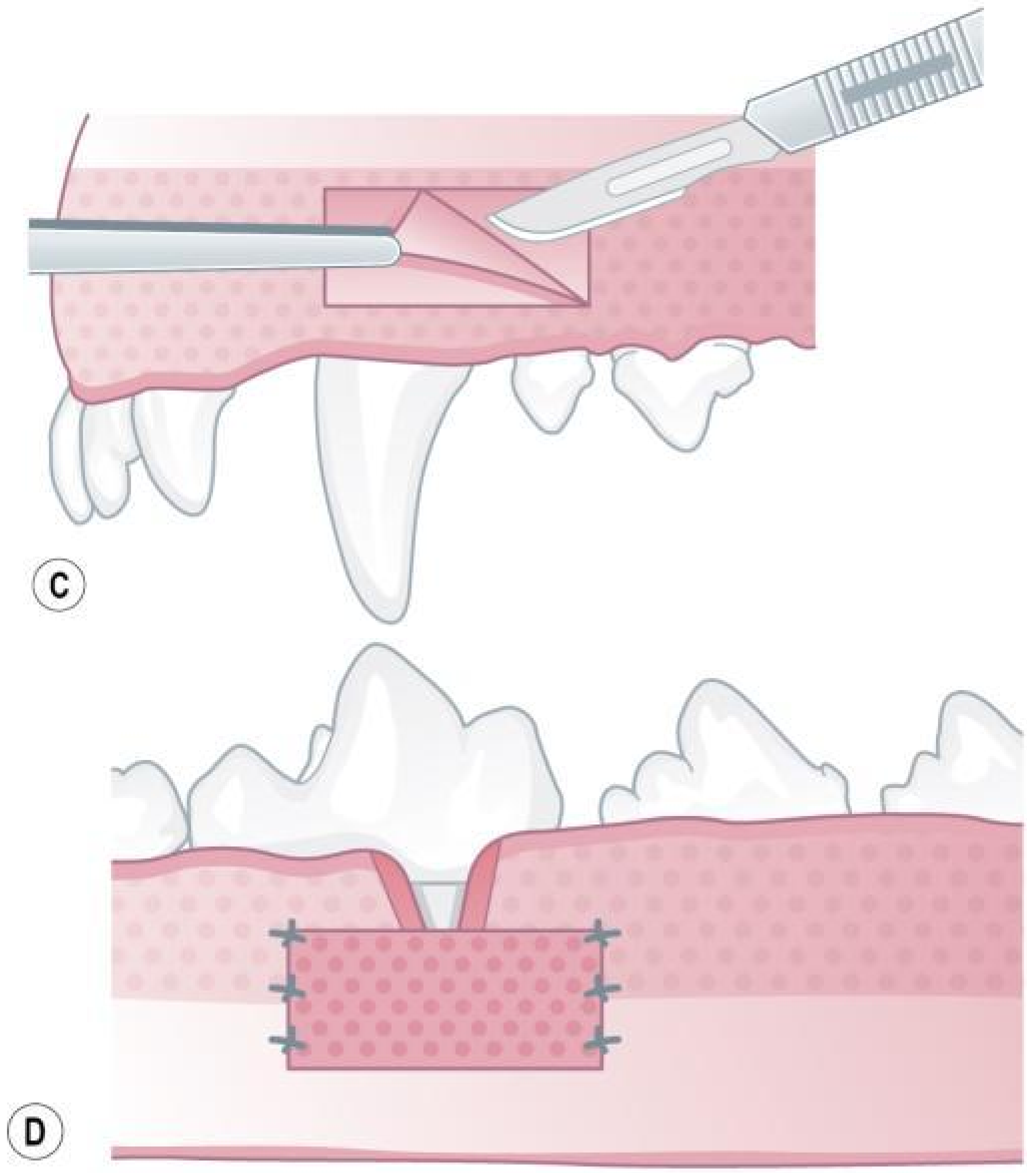
Preparation of the recipient site

At the area of recession that will be the recipient site, a reverse-bevel incision is made around the free gingival margin of the recession to remove the pocket epithelium (Fig. 21.11A). A horizontal incision is made 1–2 mm coronal to the mucogingival junction. Two vertical incisions are made apically and into the alveolar mucosa. A second horizontal incision is made to connect the vertical incisions. The rectangular piece of tissue thus created is removed by split-thickness dissection, leaving the periosteum covering the exposed area. The size of the defect created should be large enough to accommodate and secure the free gingival graft (Fig. 21.11B). Hemorrhage is controlled with wet gauze and pressure.

A

B





• **FIG. 21.11** Free gingival graft: (**A**) Excision of the sulcular epithelium. (**B**) Preparation of the recipient bed. (**C**) Preparation of the donor site. (**D**) Tacking sutures to the connective tissue bed.

Preparation of the donor site

A donor area is selected where there is sufficient keratinized attached gingiva (often the buccal aspect of the maxillary canine tooth). The recipient site is measured and a small piece of aluminum foil template matching the size of the recipient site is made. The template is then placed on the donor site and incisions are drawn around the edges of the foil through the epithelium to the periosteum of the donor area.[16] A corner of the donor epithelium is dissected with a # 15C scalpel blade and the graft removed by split-thickness dissection (Fig. 21.11C).

Suturing the free graft

The graft is sutured to the connective tissue bed using periosteal tacking sutures. The coronal mesial and distal corners are sutured first, followed by the mesial and distal sides. Hemostasis is achieved by gentle pressure using moistened gauze over the grafted area. The graft will fail if a thick blood clot is present under the graft. It is not necessary to suture the graft to the apical alveolar mucosa, as the sutures can get buried, delay healing, and are not needed to retain the graft in position (Fig. 21.11D).

Subepithelial connective tissue graft (palate)

This graft is usually used to cover root surfaces after recession. This is mainly done for cosmetic reasons in humans, so its use in veterinary dentistry is limited.

Preparation of the donor site

The connective tissue is exposed by means of a split-thickness flap on the palate. The subepithelial connective tissue is harvested, leaving the periosteum and the overlying split-thickness flap. The flap is subsequently replaced on the palate ("trap door approach").

Preparation of the recipient site

The recipient site is prepared the same way as the free gingival graft except that the split-thickness alveolar mucosa is not discarded but replaced over the connective tissue graft placed over the area of recession.

Suturing the graft and overlying flap

The connective tissue graft is sutured first (coronal, mesial, and distal) to the recipient periosteum and then the alveolar mucosa flap sutured to the interproximal gingiva. The vertical wound edges are also sutured to the connective tissue and periosteum.

Postoperative care and assessment

Periodontal flap and mucogingival surgery

Decreasing plaque accumulation postoperatively is best achieved by daily oral flushing with 0.05%–0.2% chlorhexidine gluconate solution for 2 weeks. Alternatively, chlorhexidine patches may be used. Appropriate pain medication (see Chapter 4) is prescribed and a soft diet instituted for 2 weeks. Return to routine oral hygiene will aid in the healing process.

Evaluation of the surgical site is crucial and is performed at 7–10 days. Periodontal probing and radiographic evaluation should be done after 6–12 months; this requires general anesthesia and is combined with additional periodontal treatment.

Complications

Periodontal flap

Dehiscence and sloughing of the flap is a serious complication and may happen if the flap is sutured under tension or if the patient is allowed to chew hard or abrasive toys or treats in the first 2 weeks after surgery. A flap may move from the original sutured position; as a result, bone margins may become exposed or the flap may heal coronally to the intended position and recreate a periodontal pocket.

Mucogingival surgery

Bleeding under the graft may cause sloughing of the graft. A graft may be dislodged by the patient's paws or tongue. If placed over thin bone, resorption may occur and the graft lost.

Prognosis

Periodontal flap

The correct diagnosis of pocket type and bony defect is a prerequisite for a successful outcome. Following the selection of the appropriate treatment, skillful surgical technique, including correct flap design and careful suturing, is conducive to a good prognosis. Meticulous home care is crucial and follow-up evaluations are necessary to assess the success or failure of the procedure.

Mucogingival surgery

A good prognosis is expected when treating a narrow and shallow recession, provided the correct diagnosis is made and the procedure is skillfully executed.

Acknowledgments

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CHAPTER 22

Reparative and regenerative periodontal surgery

Nadine Fiani, Milinda J. Lommer

Definitions

Allograft: A graft obtained from genetically dissimilar individual of the same species as the recipient; includes freeze-dried bone and demineralized freeze-dried bone; may be osteoinductive as well as osteoconductive

Alloplast: Synthetic or naturally occurring inert materials implanted into host tissue to occupy space and act as a scaffold for new bone production; includes hydroxyapetite, tricalcium phosphate, polymers, and bioactive glass

Autogenous graft: Obtained from a remote location in the recipient (host); the only osteogenic graft material

Guided tissue regeneration (GTR): Advanced periodontal therapy using barrier membranes with or without graft materials to encourage the growth of periodontal ligament, cementum, and bone cells and exclude unwanted epithelial cells from a healing periodontal pocket

Osteoconductive: Materials that occupy space and act as a scaffold on which new bone can grow

Osteogenic: Materials containing live cells that lay down bone matrix

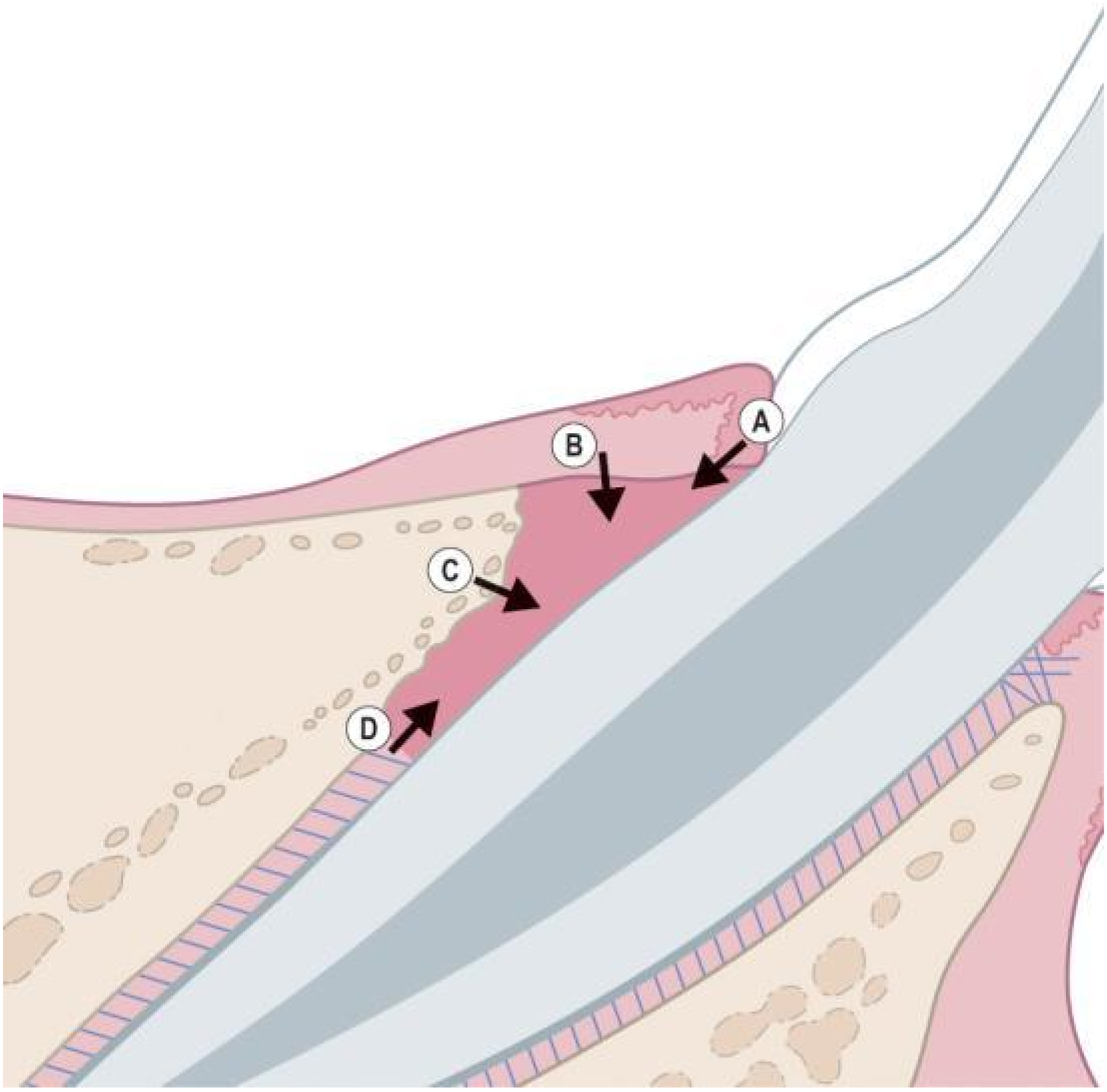
Osteoinductive: Materials containing growth factors or hormones that signal the host to produce new bone

Xenograft: Tissue obtained from an individual of a different species from the host; processing to remove antigenicity renders most of these materials osteoconductive only

Introduction

Periodontal disease is an inflammatory response to infection within the gingival sulcus, as a result of complex interaction between bacteria, their by-products, and the response of various components of the host's own immune system. Periodontitis (loss of attachment) occurs at different rates, depending on many factors.[1] Optimum therapy includes management of the infection, control of the host immune response, and regeneration of diseased or lost portions of the periodontium.[2]

The outcome of periodontal therapy is highly dependent upon the type of tissue that first repopulates the root surface.[3] A healing periodontal pocket is invaded by cells from four different sources: gingival epithelium, gingival connective tissue, alveolar bone, and periodontal ligament (Fig. 22.1).[3]

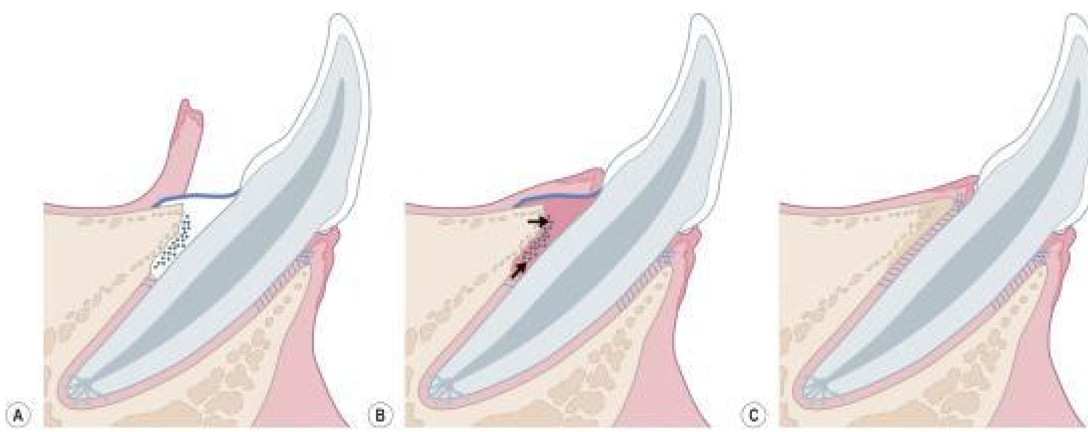


• **FIG. 22.1** Sources of new cells in a healing periodontal pocket. Following curettage and debridement of the pocket epithelium, cells from the following tissues enter the clot: (**A**) gingival epithelium, (**B**) gingival connective tissue, (**C**) alveolar bone, and (**D**) periodontal ligament.

Proliferation of gingival epithelial cells along the root surface before the other tissues reach the area results in a long junctional epithelium, which may be unstable and has a high chance of pocket recurrence.[4] If gingival connective tissue is first to repopulate the root surface, the fibers will be parallel to the root surface and alveolar bone will regenerate with no connection to the cementum.[5] When bone is the first repopulator, the typical response is root resorption and ankylosis. Only when periodontal ligament cells proliferate coronally can new cementum and periodontal ligament formation occur, restoring healthy attachment.[5] Unfortunately, epithelial cells migrate 10 times faster than other periodontal tissue types, so conventional periodontal therapy typically results in reparative healing by the formation of long junctional epithelium rather than regenerative healing, which restores the periodontal ligament, cementum, and bone.[6]

Periodontal *repair* is noted when healing occurs by replacement with epithelial and/or connective tissue that matures into various types of scar tissues, which may or may not be functional.[7] This is often termed *new attachment.* The ideal outcome of periodontal surgery is periodontal *regeneration*, where elimination of the pocket and reconstruction of the periodontal tissues and architecture occur.[7,8] Accurate differentiation between repair and regeneration of the periodontal attachment can only be achieved histologically.[7,9] This is an obvious limitation in clinical patients, leaving periodontal probing and intraoral radiography as the diagnostics of choice for assessing the extent of attachment but not type of attachment.

Because of the aforementioned reasons, periodontal regeneration can be achieved only after removal of junctional and pocket epithelium and prevention of their migration into the healing periodontal space after surgery. Regenerative procedures typically involve the use of barrier membranes with or without bone grafting materials to promote the growth of desired periodontal tissues while excluding unwanted cell types such as gingival epithelial cells (Fig. 22.2).[10]



• FIG. 22.2 (**A**) Guided tissue regeneration involves placement of a barrier membrane with or without bone graft placement. (**B**) This allows the more slowly growing osteoblasts and periodontal ligament fibroblasts to repopulate the pocket, resulting in (**C**) the formation of new bone and periodontal ligament attachment and elimination of the pocket.

Guided tissue regeneration

Guided tissue regeneration (GTR) is an advanced periodontal therapy that attempts to restore tissues (periodontal ligament, cementum, and alveolar bone) lost due to periodontitis.[9,11] GTR has the potential to save teeth that might otherwise be lost due to disease or extraction.

In periodontal GTR, a selective barrier (composed of a barrier membrane with or without a particulate graft) is placed between a properly prepared tooth root surface and the gingival tissues, which are closed over the area with a flap.[12,13] The principle behind this procedure is the mechanical prevention of apical migration of epithelium while maintaining a space for clot stabilization that should then allow for periodontal regeneration.[7,14]

Although, in theory, GTR may appear to be straightforward and simple, failure is likely if there is failure to strictly adhere to the principles of treatment. Success is dependent upon correct selection of site, proper choice of materials, appropriate technique for the site and material, skill of the surgeon, and client commitment to oral hygiene for the patient.[9,14,15]

Patient and client selection

General anesthesia is required both to perform the GTR procedure and to reevaluate the treated area(s) at regular intervals. Guided tissue regeneration is technically more challenging and often more time-consuming than extraction, so the anesthetic period may be prolonged for the initial treatment. In addition, general anesthesia is required for reassessment of the treated area(s) with radiographs and probing. Therefore, patients for whom GTR is considered should be in good general health, without contraindications for repeated anesthetic episodes. The patient must be compliant for visual inspection of the surgical site 2 weeks postoperatively and must receive daily tooth-brushing at home. The client must be dedicated to the preservation of the pet's oral health, willing to perform daily home care, and comfortable with the need for follow-up radiographs under general anesthesia.[9] Further, the client must be informed that the results of GTR are unpredictable, success is not guaranteed, and the tooth or teeth in question may require extraction in the future.[7,14]

Patient preparation

Following intraoral radiographs and prior to diagnostic dental charting, the oral cavity should be disinfected with an agent such as dilute chlorhexidine gluconate, with a concentration not exceeding 0.12%, because at concentrations higher than 0.12%, chlorhexidine inhibits attachment of periodontal cells to the root surface.[16] Further, chlorhexidine solutions are contraindicated for the direct treatment of the root surface and periodontal tissues within the pocket prior to surgery, for even at concentrations as low as 0.0025%, chlorhexidine may have adverse effects on the growth of periodontal cells.[16] Contamination of the surgical field should be minimized by the placement of cotton rolls or rubber dams to restrict salivary or other contaminate incursion.[2]

Site selection

Guided tissue regeneration should be considered for structurally and functionally important teeth, such as canine teeth and mandibular first molar teeth, with infrabony pockets. Currently, clinical indications include stage 2 furcation lesions, two- or three-walled vertical interproximal sites, and three-walled palatal defects of single-rooted teeth.[10,14] Areas with abundant gingival attachment are typically preferred as they facilitate good flap coverage of the barrier. Although there is limited evidence that combination GTR therapy using a membrane and bone graft material may allow some regeneration in suprabony defects,[17] areas with horizontal bone loss (suprabony defects) are generally considered poor candidates for GTR.[12]

Flap creation

Initially, a flap must be created to allow access to the pocket. In designing this flap, the ultimate closure of the site with the incorporation of the barrier must be taken into consideration. Every precaution should be taken to preserve the blood supply of the flap during its creation, elevation, and replacement, as this greatly affects healing and the ultimate success of the procedure. Local anesthesia is recommended; if using a local anesthetic with epinephrine (which prolongs the anesthetic effect and reduces excess bleeding), the epinephrine should not exceed a concentration of 1:100,000 as it may restrict the blood flow to the flap, adversely affecting the flap's vitality.[2] Periodontal flap design is discussed in detail in Chapter 21.

Pocket preparation

Treatment of the pocket must be planned to provide a compartment of adequate dimensions for the development of the desired new bone and periodontal tissues. The pocket must be debrided of all granulation and other undesired soft tissues to expose the alveolar bone walls and root cementum. Treatment of the tooth root of the affected site includes root debridement and subgingival curettage to remove all invading soft tissues, such as epithelial, connective, and granulation tissues from the pocket.[12,18,19] Once exposed, the surfaces of the tooth root may benefit from a surface-conditioning treatment with either citric acid, tetracycline, or Ethylenediaminetetraacetic acid (EDTA) preparations.[8] Root conditioners may aid in attachment of the blood clot and other tissues to the root surface and enhance healing. Citric acid, in particular, has been reported to demineralize the root surface, eliminate bacteria and endotoxins from the root surface, remove the smear layer, and expose the collagen in the cementum; fibrin linkage to the exposed collagen fibers reportedly prevents gingival epithelial cells from migrating over the roots.[8] Not all authors agree which root conditioner is best or exactly when it should be used.[2,20] Therefore, the use of these products should be guided by the barrier manufacturers' recommendations. Osteoplasty (recontouring of the alveolar bone associated with the pocket) may also be performed to improve the site architecture for the barrier placement and flap closure.

Membrane function and combined technique

As previously mentioned, the membrane acts as a barrier that inhibits downgrowth of gingival epithelium and connective tissue into the pocket, preventing these cells from colonizing the root surface.[12] This allows time for the more slowly growing periodontal ligament, cementum, and alveolar bone cells to reproduce and recreate the normal periodontal architecture. Research suggests that barriers need to be in place and reasonably intact for between 42 and 56 days to be effective in GTR.[7,12,14] Various types of absorbable and nonabsorbable membranes are commercially available (see "Membrane Barriers" section).

Although not essential to GTR, many authors advocate the use of a combined technique where different bone graft materials and/or biologic mediators are placed in the defect apical to the membrane.[7,15,17] Depending on the type of particulate used (see "Graft Materials" section), the benefits include space maintenance (stops collapse of the membrane), clot stabilization, osteogenesis, osteoinduction, and/or osteoconduction.[7,21-24]

Membrane barriers

The periodontal membrane should ideally act as a selective barrier that is biocompatibile, integrates well with tissues, prevents ingrowth of cells from outside the membrane, and can be positioned to produce sufficient space for the desired new tissue to proliferate. The membrane should be selected in a configuration that best fits the particular defect, or custom-fitted and seated to the area by trimming with sterile scissors, if allowed by the manufacturer. Sharp corners, wrinkles, folds, and overlapping of the material should be avoided, when possible, to provide a suitable fit for flap closure. Tucking the membrane under the periosteum and placing sutures in the membrane prior to gingival closure will help to stabilize the membrane and help prevent migration due to movement and pressures generated by mastication and tongue motion. Table 22.1 summarizes the factors that influence the outcome of GTR procedures.[10] There are numerous types of membranes, both absorbable and nonabsorbable, that are derived from a variety of natural and synthetic sources.

TABLE 22.1

Factors Influencing the Outcome of Guided Tissue Regeneration Procedures

	Positive Effect on Outcome	Negative Effect on Outcome
Anatomic factors	Deep (≥4 mm) infrabony defect	Shallow infrabony defect
	Narrow defect angle (<45 degrees)	Wide defect angle (>45 degrees)
	Vertical bone loss	Horizontal bone loss
	Three-wall defects	One- or two-wall defects Supragingival defects
	Minimal furcation involvement	Deep furcation involvement
	Adequate tissue thickness (>1.1 mm)	
	Adequate keratinized gingiva (2 mm)	
Patient/client factors	Good oral hygiene/compliance	Poor oral hygiene/compliance
	Systemic health	Systemically compromised patient
Surgery-related factors	Passive flap tension	Excessive flap tension
	Stable wound	Early mechanical disruption
	Aseptic technique	Contamination during surgery
	Primary wound closure	Inadequate wound closure
	Short, minimally traumatic surgery	Prolonged/traumatic surgery
	Proper incision location	Poorly designed incisions
	No membrane exposure	Membrane exposure

Nonabsorbable membranes

Gore-Tex (W.L. Gore & Associates, Inc., Newark, DE), a nonabsorbable expanded polytetrafluoroethylene (ePTFE) membrane, was one of the first commercially available barriers. The membranes are available in various configurations and sizes, in nonreinforced and titanium-reinforced configurations.[14] It possesses adequate stiffness but is supple enough to adapt well over defects. Clinical studies have shown that infrabony defects treated with ePTFE membranes can be expected to achieve 3–5 mm of bone fill and 4–7 mm of clinical attachment level gain.[25-27] Although excellent clinical results have been achieved with nonabsorbable membranes, they are associated with a high frequency of early exposure to the oral environment, compromising their effectiveness.[7,14] Another practical limitation of ePTFE and other nonabsorbable membranes is the necessity for a second anesthetic event in 6–8 weeks to remove the membrane in veterinary patients.[14]

Bioabsorbable membranes

Bioabsorbable membranes have been developed primarily to eliminate the need for a second surgery for membrane removal. Various materials, including collagen, polyglycoside synthetic polymers, calcium sulfate, and demineralized cortical bone, have been used as barrier membranes.[9,28] The clinical efficacy of bioabsorbable membranes relies on their ability to retain their structural integrity for 6–8 weeks of healing before being absorbed.[14]

Collagen barrier membranes, derived primarily from human skin, from porcine skin, and from bovine gastrocnemius tendon, have been used successfully in the treatment of periodontal defects and have gained increased popularity due to their greater biocompatibility.[13,29] As well as preventing epithelial migration, collagen membranes are chemotactic and stimulate proliferation of fibroblasts,[30] provide hemostasis, serve as a scaffold for vascular and tissue ingrowth, and are easily shaped and adapted to cover defects.[14] These membranes are absorbed by macrophage and neutrophil activity. Over a dozen collagen membranes are commercially available, some of which have been modified by cross-linking proteins within the collagen to vary the resorption rate.[10] However, cross-linking may lead to a higher complication rate if the membrane is not absorbed within 6–8 weeks.[31]

Degradable polymer membranes are formed by copolymerization of polylactic acid, polyglycolic acid, and polylactate/polyglycate.[14] These synthetic membranes remain intact for 20 weeks or more before degrading by hydrolysis of ester bonds.[32] The hydrolysis is accompanied by local inflammation. The effect of this on periodontal regeneration is not clear.[33] Polyglactin 910 (Vicryl; Ethicon, Inc., Somerville, NJ) membranes have been used successfully in experiments regenerating periodontal bone in ferrets[34] and in dogs.[35-37] Synthetic polylactic acid liquid membranes have been used in the treatment of experimentally created periodontal defects in dogs, with mixed results.[38-41] A case series of four dog teeth treated with a polymer gel (*N*-methyl-2-pyrrolidone and poly [dl-lactide]) combined with 8.5% doxycycline hyclate (Doxirobe Gel; Zoetis, Parsippany, NJ) suggested that this gel may be used as a barrier in GTR.[42] However, a larger controlled study would be necessary to truly evaluate the efficacy of this treatment modality.

Calcium sulfate barriers provide good adaptation at the defect and are absorbed in about 30 days without an inflammatory reaction.[14] A number of studies in human patients have found these membranes to be similar in efficacy to the collagen membranes.[43,44]

Canine demineralized freeze-dried cortical bone flexible membrane (Ossiflex; Veterinary Transplant Services, Inc., Kent, WA), has been reported to be an effective membrane for GTR in the dog.[9] It should be noted that Ossiflex is the only membrane currently labeled for use in dogs. All other membranes are not approved for clinical use in veterinary patients; therefore, their use requires informed consent from the clients.

Graft materials

Autogenous grafts are those collected directly from the patient. They can be cortical and/or cancellous bone and marrow harvested from intraoral (edentulous jaw) or extraoral (proximal tibia, proximal humerus, etc.) sites.[21,24] These grafts have historically been considered the "gold standard" bone replacement material,[10] and they have osteogenic, osteoinductive, and osteoconductive properties.[21] Despite these obvious advantages, concerns with donor site morbidity, volume of bone required, and unpredictable rate of replacement[24] make autogenous bone grafts of limited practical use in the treatment of periodontal defects in veterinary patients. Interestingly, experimental studies in dogs have failed to show an advantage in periodontal healing when comparing autogenous bone with other materials (coral-derived biomaterial, tricalcium phosphate [TCP], or demineralized freeze-dried canine bone allograft)[45,46]; periodontal regeneration reportedly occurs more predictably with autografts and demineralized freeze-dried allografts than with other materials.[47]

In addition to their osteoconductive properties, *allografts* (typically freeze-dried or frozen cancellous and/or cortical bone) may contain bone morphogenetic proteins (BMPs), which stimulate new bone production (osteoinduction). Numerous clinical studies have evaluated the success of freeze-dried bone allografts in inducing new bone formation in human patients.[48-52] Experimental studies in dogs have shown production of new bone[45,53] and cementum[46,54] following placement of freeze-dried bone allografts. Demineralization of the freeze-dried bone exposes the collagen fibrils and associated components of bone matrix including BMP; therefore, demineralized freeze-dried bone (DFDB) is considered osteoinductive, while nondemineralized freeze-dried bone is considered osteoconductive only.[8,21] Both canine and feline freeze-dried bone are commercially available in a mixture of demineralized bone matrix and cancellous chips, 0.7 mm in diameter, specifically designed for oral and periodontal use (OsteoAllograft Periomix; Veterinary Transplant Services, Inc., Kent, WA). Allografts are appealing in that there is no need for an additional surgical procedure to procure bone from a remote site on the patient.

Alloplasts are synthetic or naturally occurring inert materials that serve primarily to maintain space and largely act as osteoconductive agents.[10]

Calcium phosphate biomaterials include those composed of hydroxyapatite and TCP. They are similar to bone mineral in structure, bioactivity (formation of bone apatite like material), ability to promote cellular function and expression leading to the formation of a strong bone-calcium phosphate interface, and osteoconductivity.[55,56] The original TCPs resorbed quickly (within days to weeks), while hydroxylapatites resorbed slowly, if at all. Currently available products have more controlled resorption rates. Hydroxylapatite, a calcium phosphate mineral component of bone and coral, is available in particulate forms (Calcitite: Calcitek, Inc., Carlsbad, CA; OsteoGen; Impladent, Ltd., Holliswood, NY; SYMBIOS OsteoGraf; Dentsply Sirona, Philadelphia, PA). Tricalcium phosphate is available in particulate (SynthoGraft; Boston, MA) and putty (FormPutty: Theken Spine, Inc., Akron, OH) forms, with controlled resorption rates varying from several days up to 12 months.[57,58]

Calcium sulfate (plaster of Paris) was one of the first synthetic materials used as a bone supplement and for osteoconduction.[59,60] Results in reported studies are mixed,[60-62] and resorption is inconsistent.

Bioactive glass, also called bioglass materials, generally contain oxides of calcium, sodium, phosphorous, and silicone, which stimulate collagen synthesis and new bone formation. Most bioglass materials use silicone oxide as a matrix former. Bioglass is largely considered osteoconductive; however, in vivo studies have suggested that it may have some osteoinductive properties.[55] Bioglass is not replaced by bone, but forms a strong molecular bond between its surface and bone without an intermediate fibrous layer.[56] The minerals are slowly released in an aqueous environment, increasing pH and osmotic pressure at the site, which may slow or prevent resorption of the bioglass. Bioactive ceramics have a broad antimicrobial effect on microorganisms on teeth and implants.[63-65] One bioactive glass (Consil; Nutramax Laboratories, Inc., Edgewood, MD), available in both particulate and putty forms, is marketed specifically for veterinary use; it has been used in extraction site alveolar margin maintenance,[66] to treat bone loss associated with lumpy jaw conditions (actinomycosis),[67] and GTR procedures in dogs.[2,53,68] While there are no veterinary clinical studies in dogs, experimental evidence suggests that bioactive glass is less effective than DFDB allograft in inducing new bone formation in infrabony pockets adjacent to implants in dogs.[53]

The most common *xenograft* is bovine cancellous bone, processed at a high temperature (>1,000°C) to remove all organic matter, eliminating the risk of zoonotic infection and graft rejection. This treatment, however, renders the material osteoconductive only by leaving behind a hydroxyapatite skeleton with a highly porous structure similar to cancellous bone.[56] The resulting inorganic bone mineral has been used experimentally in extraction sites and for GTR in dogs.[69-75] Table 22.2 summarizes available graft materials and their sources.

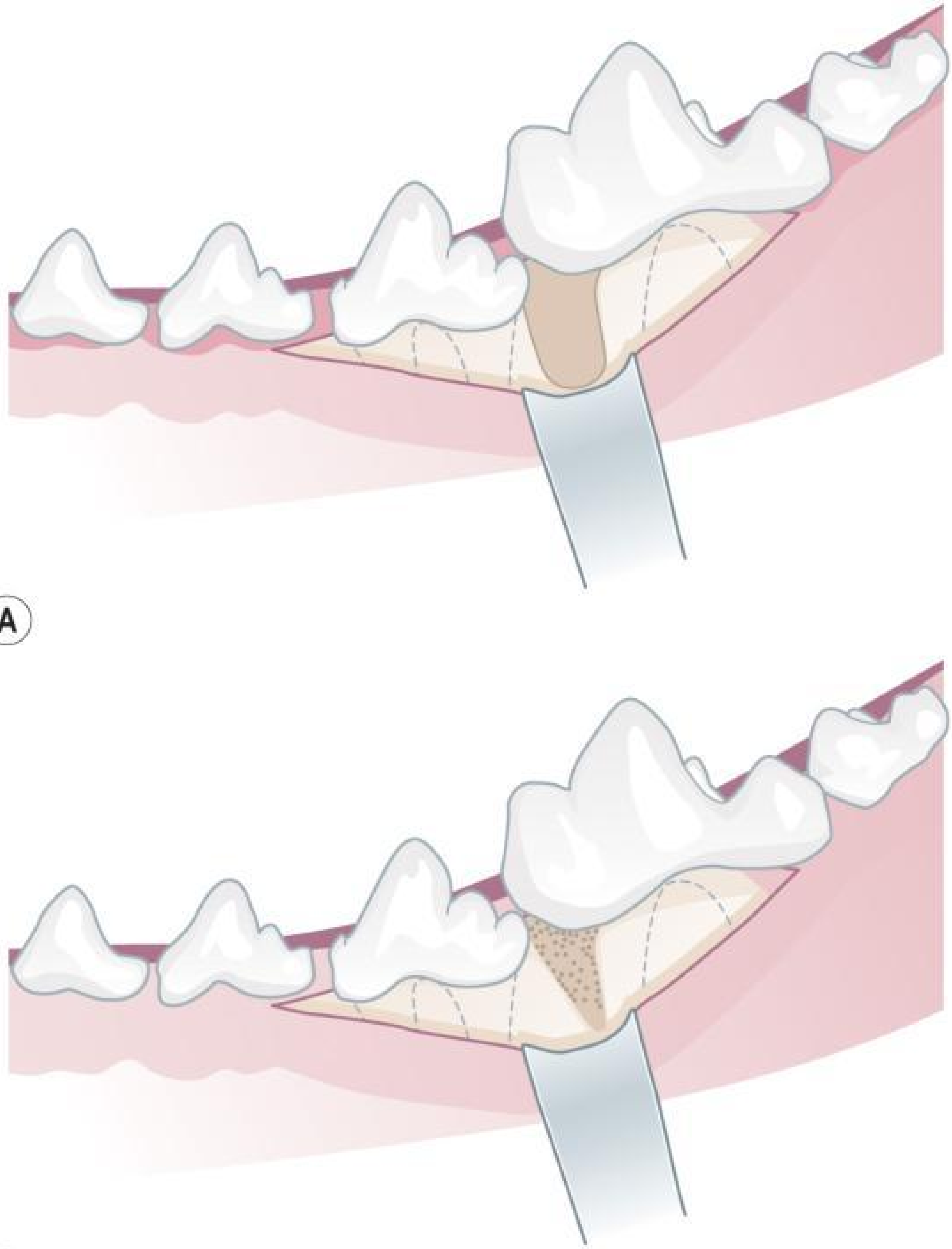
TABLE 22.2

Summary of Bone Grafting Materials and Substitutes

	Autogenous	Allograft	Alloplast	Xenograft
Source	Patient	Same species	Synthetic	Different species
Properties	Osteogenic, osteoinductive, osteoconductive	Osteoinductive, osteoconductive	Osteoconductive	Osteoconductive
Examples	Cancellous bone collected from the proximal humerus, iliac crest, etc.	Demineralized freeze-dried bone and freeze-dried bone, e.g., OsteoAllograft (Veterinary Transplant Services, Inc., Kent, WA)	Bioactive ceramicse.g., Consil (Nutramax Laboratories, Inc., Edgewood, MD)	Bovine cancellous bonee.g., Bio-Oss (Geistlich Biomaterials, Inc., Wolhusen, Switzerland)

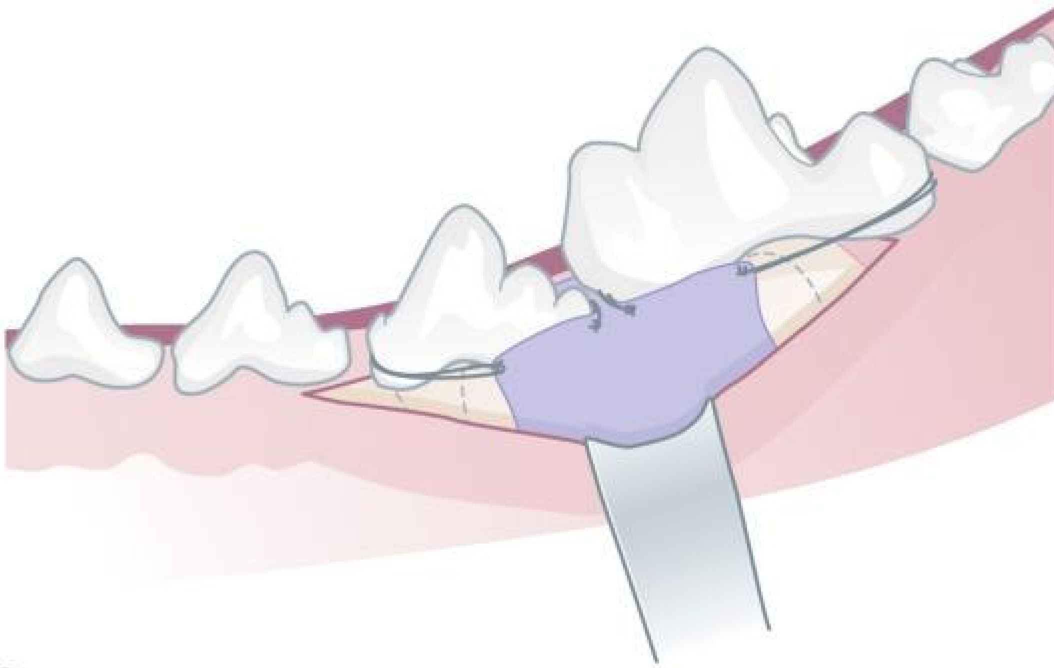
Proposed GTR technique using allograft and absorbable membrane (fig. 22.3)

1. Elevate a periodontal flap (see Chapter 21).
2. Debride the osseous defect, removing all granulation tissue.
3. Remove all root deposits using hand and/or ultrasonic instrumentation.
4. If desired, condition the root surface with EDTA or citric acid.
5. Place the particulate graft material loosely in the osseous defect. Do not pack it down as this will interfere with cell migration.
6. Trim the membrane to the approximate size of the area being treated. The apical border of the material should extend 3–4 mm apical to the margin of the defect and laterally 2–3 mm beyond the defect. The occlusal border of the membrane should lie 2 mm apical to the cementoenamel junction.
7. Suture the membrane tightly around the tooth with a sling suture.
8. Suture the flap back in its original position or slightly coronal to it, in a simple interrupted pattern, making sure that the membrane is completely covered.



A

B



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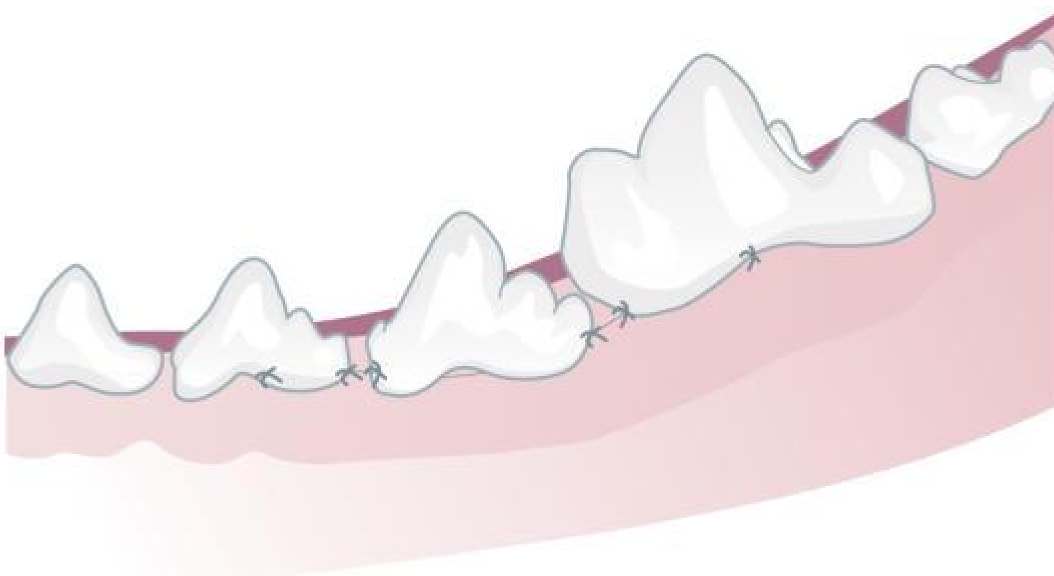

D

• **FIG. 22.3** (**A**) A periodontal flap is elevated. (**B**) After debridement of the pocket, particulate allograft material is placed in the osseous defect. (**C**) The pretrimmed membrane is placed over the graft material and, if needed, sutured with a sling suture; the apical border of the membrane should extend 3–4 mm apical to the margin of the defect and laterally 2–3 mm beyond the defect. (**D**) The flap is replaced and sutured.

Tissue engineering with biologic mediators

Tissue engineering of periodontal defects relies on the manipulation of one or more of three key elements: signaling molecules, scaffold, and cells. There is experimental evidence of the potential of bioactive agents including bone, platelet-rich plasma, enamel matrix derivatives

(EMDs), morphogenetic proteins, various growth factors, mesenchymal stem cells, bone anabolic agents, and gene therapy to regenerate periodontal and dental tissues.[11,76-80]

Currently, three products are commercially available: EMDs, recombinant human platelet-derived growth factor-BB (rhPDGF-BB)/beta calcium phosphate, and synthetic binding protein P-15/anorganic bovine matrix.[7,79]

Enamel matrix derivatives, a mixed peptide combination derived from the immature enamel of juvenile piglets, has been proposed as an alternative to the use of traditional GTR techniques involving membranes and graft materials. Emdogain (Straumann, Andover, MA) is an absorbable gel that contains enamel matrix proteins, primarily amelogenin. Enamel matrix proteins are secreted by Hertwig's epithelial root sheath during tooth development and induce the formation of acellular cementum. Emdogain has been used in experimentally avulsed and replanted teeth in dogs in an attempt to regenerate periodontal ligament and prevent ankylosis, with mixed results.[81,82] Results of clinical trials in humans with infrabony defects have shown comparable results with use of Emdogain and more traditional GTR procedures, although a high degree of heterogeneity was observed among the trials.[83] A recent review concluded that there is robust evidence to support the use of EMD in the treatment of infrabony defects.[79,84] The technique for use of Emdogain has been described by Mellonig.[85]

Recombinant human platelet-derived growth factor (GEM21S" Osteohealth, Shirley, NY) is a powerful mitogenic and chemotactic factor for mesenchymal cells. Several studies in dogs and humans have found rhPDGF to be comparable or superior to other regenerative graft material.[7,79]

P-15 is a polypeptide that mimics the cell-binding domains of Type 1 collagen, which has been shown to increase the rate and extent of cell attachment and migration to root surfaces.[79] The commercially available product, PepGen P-15 (Dentsply International, York, PA), a combination of bovine-derived anorganic bone and a synthetic analog of a 15-amino acid sequence of type I collagen, has been proposed to enhance bone regenerative capacity in periodontal defects.[8] A comprehensive review concluded that adjunctive benefits may be achieved with the use of P-15 when combined with a graft as a carrier.[84]

The future of periodontal regenerative medicine likely lies in tissue engineering: treatments that combine the use of membranes and/or graft materials with stem cells and growth factors.[86-90] As new treatment modalities become commercially available, the clinician should compare the cost with the expected clinical benefit when deciding which treatment approach to use.[91]

Postoperative considerations

In human patients, periodontal dressings are usually placed for protection of the surgical site, but due to the lack of cooperation of veterinary patients and a high rate of rapid failure of these dressings, they are not commonly used in animals.

As with other oral surgical procedures, patients should be offered soft food for several days following GTR, and chew toys and hard treats should be avoided for at least 2 weeks. Appropriate analgesics should be prescribed for 5–7 days. Systemic antibiotics are commonly administered for 10–21 days after periodontal surgery, although a definitive, evidence-based rationale for this approach is lacking,[8] and multiple studies have concluded that there is not enough evidence to support the use of systemic antibiotics following periodontal surgery.[79,92-95]

To avoid disrupting the flap, toothbrushing should not be performed for 7 days following a GTR procedure. Follow-up examination of the surgical site is performed 10–14 days postoperatively; this is also a good time to evaluate the effectiveness of the client's home care efforts. The patient should return for follow-up radiographs and probing in 3–6 months.

Conclusion

Periodontal regeneration is an exciting aspect of advanced periodontal treatment. Patient, client, and periodontal defect selection, as well as surgical skill, is essential; however, success is not always guaranteed. Although there are many scientific advances in this field, clinicians are advised to take care when reviewing experimental regenerative literature as there is a considerable difference between statistical success and clinical success. An attachment gain of 0.5–1 mm may be statistically significant; however, it will mean little clinically. The GTR procedure has good potential; however, many factors play a role in its ultimate clinical success.

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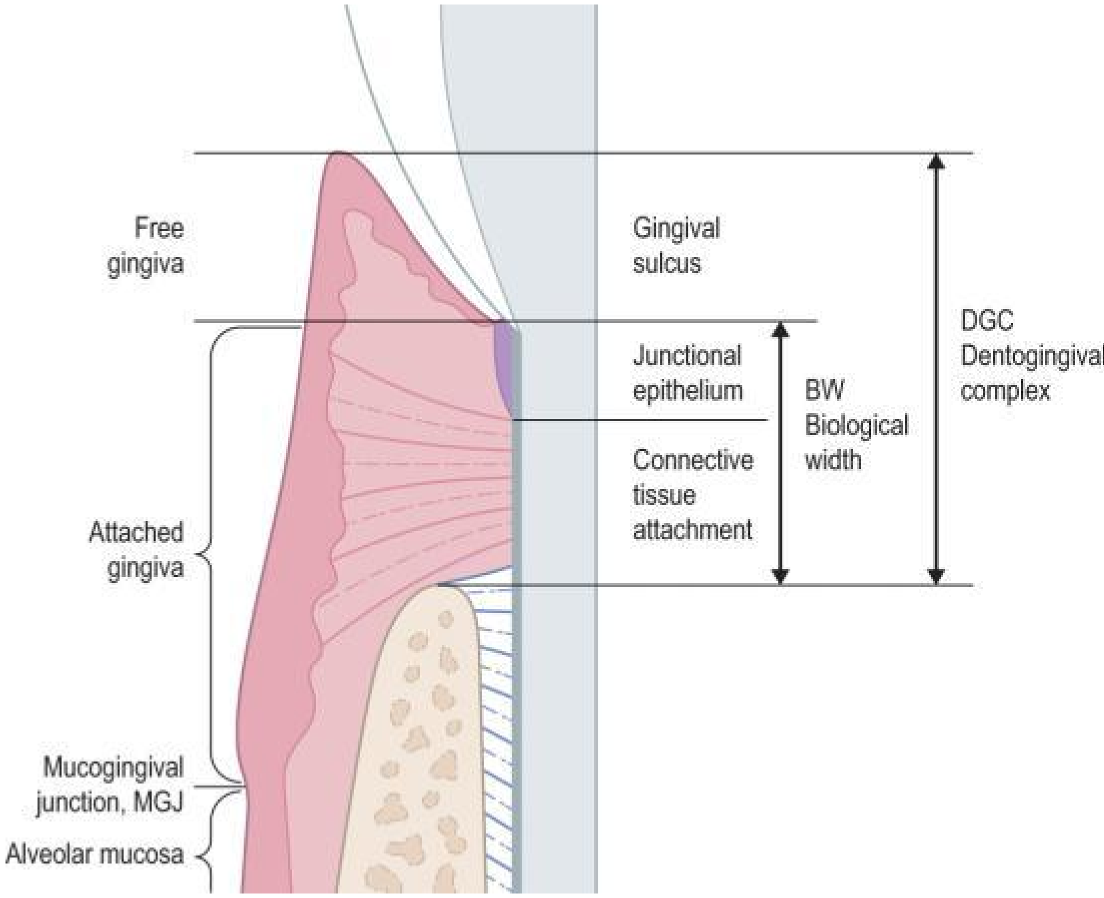
CHAPTER 23

Crown-lengthening

Fraser A. Hale

Definitions

Biologic width: The dimensional width of the junctional epithelium and connective tissue attachment (see "Surgical Anatomy" and Fig. 23.1)[1-3]
Clinical crown: That portion of the natural tooth structure exposed supragingivally
Dentogingival complex: The sum of the widths of the gingival sulcus, junctional epithelium, and connective tissue attachment to root cementum[1]
Emergence profile: The anatomic shape of the tooth as it emerges through the gingiva[4]
Ostectomy: The excision of bone
Osteoplasty: The modification or change of the configuration of bone
Type I crown-lengthening: A gingivectomy to expose more of the tooth[3,5]
Type II crown-lengthening: An apically repositioned flap with osseous contouring to expose more of the tooth[3,5]
Type III crown-lengthening: The forced eruption with an orthodontic device to expose more of the tooth[3,5]



• **FIG. 23.1** The "biologic width" is the sum of the junctional epithelium width + connective tissue attachment width. The dentogingival complex is the sum of the gingival sulcus depth and the biologic width.

Preoperative considerations

Crown-lengthening refers to procedures designed to expose more tooth structure (crown and root) supragingivally. It may be done to expose lesions where they can be managed more effectively or to achieve greater clinical crown surface area for the retention of a prosthetic crown or bridge.[1,6] In the case of an undererupted tooth, crown-lengthening can be used to expose the enamel, to which the gingiva and periodontal ligament will not firmly attach, to eradicate or reduce the periodontal pseudopocket and concomitant pericoronitis.[1,7,8]

Type I crown-lengthening is simple gingivectomy (see Chapter 20). Type III crown-lengthening is an orthodontic procedure (see Chapter 59). Type II crown-lengthening is the surgical repositioning of the alveolar margin and gingiva to expose more of the tooth while maintaining appropriate periodontal relationships, which will be the focus of this chapter.

Crown-lengthening is most easily accomplished on single-rooted teeth such as the canine teeth. For multirooted teeth, the need to avoid exposure of the furcation limits the degree to which the periodontal tissue can be moved apically.

Surgical crown-lengthening is a relatively advanced periodontal surgery, which generates both a risk of failure and a cost to the client. In deciding if the procedure is justified, the surgeon should consider the functional significance of the tooth, the animal's overall periodontal status, and the client's commitment to home care (see Chapter 19).

Indications

Coronal fractures and caries lesions may extend subgingivally. Preservation of the affected tooth depends on restoration of the defect and managing the adjacent periodontal tissues. Gingiva and periodontal ligament will only attach to bone and cementum.[9] Therefore, leaving dentin exposed or placing a restoration deeply subgingivally will result in a deep periodontal pocket. To avoid this, an apically repositioned flap with apical repositioning of the alveolar margin can be used to bring the lesion/restoration supragingivally (or at least suprabony).[9,10] Not only does this make restoring the defect much easier, it also improves access for home care around the restoration and improves the long-term periodontal prognosis for the tooth.[3,6,11]

The placement of a prosthetic crown requires that sufficient clinical crown be available to hold the prosthesis in place.[1,3,12] Crown fractures may result in insufficient natural tooth structure remaining above the gingiva for prosthetic retention. In such cases, crown-lengthening can provide the necessary clinical crown for prosthetic restoration.

Another method for creating additional clinical crown for prosthetic retention is through the use of a post-and-core buildup. Although this can increase the surface area for bonding without periodontal surgery, it can also weaken the tooth and predispose to fractured roots.[13] Therefore, for a tooth subject to strong forces, such as the canine tooth, surgical crown-lengthening, which does not weaken the tooth, is preferred.

Surgical anatomy

Periodontium

The coronal margin of the alveolar process, where the cribriform and cortical plates meet, is the alveolar margin. The alveolar margin is typically 1–2 mm apical to the cementoenamel junction so that there is a band of root cementum residing coronal to the alveolus.[11,14-16]

The free gingival margin is typically 1–2 mm coronal to the cementoenamel junction in dogs and 0.5–1 mm in cats. The gingiva attaches to the cementum of the root that is coronal to the alveolar margin and to the cortical plate of the alveolar process. Immediately apical to the gingival sulcus is the junctional epithelium, which forms a relatively loose attachment to the cementum. Normally, the zone of junctional epithelium will be about 1–2 mm wide. Apical to the junctional epithelium is the more firmly attached connective tissue attachment to root cementum and to the cortical plate. This zone would also typically be 1–2 mm wide.[14,15,17]

The sum of the widths of the gingival sulcus, junctional epithelium, and connective tissue attachment to root cementum is referred to as the *dentogingival complex*, and the sum of the width of the junctional epithelium and connective tissue attachment is known as the *biologic width* (Fig. 23.1).[1,3,6] The normal physiology of the dentogingival complex depends on the preservation of the biologic width. In humans, the sulcus tends to be about 0.7 mm deep, the junctional epithelium just under 1 mm, and the connective tissue attachment just over 1 mm, for a total of about 2.75 mm from free gingival margin to alveolar margin.[6,18] In dogs and cats, the dimensions are much more variable, influenced by patient size and the specific tooth. At the canine tooth of a large-breed dog, the biologic width may be 5 mm or more, whereas around an incisor tooth of a cat, it may be less than 1 mm. Whatever the biologic width for a specific tooth, any periodontal surgery should have as a priority the preservation or recreation of this relationship. Therefore, if the free gingival margin is to be moved 3 mm apically, so must the alveolar margin.[6]

The gingiva's apical margin is the mucogingival junction, which is seen as a distinct line buccally on both the mandible and maxilla and on the lingual side of the mandible. On the maxilla, the palatal gingiva blends imperceptibly with the palatal mucosa (see Figs 20.2 and 20.3).

Healthy gingiva is very firm, dense, and inelastic. The oral mucosa is much thinner, softer, and more pliable/elastic. Therefore, any repositioning of the gingiva typically relies on extending the flap into the more mobile alveolar mucosa.

Topographical anatomy

When elevating the flaps for crown-lengthening of the maxillary canine tooth, it will be necessary to make an incision between the maxillary third incisor tooth and the canine tooth. There is a branch of the major palatine artery that passes through this space to anastomose with the lateral nasal artery.[19,20] This artery will be severed in creation of the flaps, and although it bleeds significantly at first, hemorrhage can typically be controlled with direct pressure.

When elevating the buccal flap at the mandibular canine tooth, care should be taken to not damage the neurovascular structures emerging from the mental foramina, particularly the middle mental foramen, located ventrolateral to the apex of the canine tooth or ventral to the first or second premolar teeth.[19,20]

Crown-lengthening of the maxillary fourth premolar tooth calls for caution to avoid damaging neurovascular elements emerging from the infraorbital foramen, located dorsal to the apex of the distal root of the maxillary third premolar tooth.[19,20] Dorsal to the distal portion of the maxillary fourth premolar tooth is the parotid papilla, and dorsal to the distal aspect of the maxillary first molar tooth is the main zygomatic papilla.[20,21] Care should be taken to avoid severing or ligating either of these salivary ducts.

Special instruments and materials

In addition to the standard set of periodontal instruments (see Chapter 21), bone chisels such as Ochsenbein # 1 (Hu-Friedy, Chicago, IL) and Kramer-Nevins # 1/2 (Hu-Friedy, Chicago, IL) can be used for ostectomy and osteoplasty (Fig. 23.2).

• **FIG. 23.2** Ochsenbein # 1 (Hu-Friedy, Chicago, IL) *(top)* and Kramer-Nevins # 1/2 (Hu-Friedy, Chicago, IL) *(bottom)* bone chisels. Source: (Courtesy Hu-Friedy, Chicago, IL.)

Therapeutic decision-making

Staging

If the crown-lengthening surgery is done in preparation for prosthodontic restoration, the crown will need to be prepared and impressions obtained for the dental laboratory. Crown preparation involves shaping the natural tooth structure to accept the prosthetic covering. An important feature of crown preparation is the placement of the finish line or crown margin. This is the line that marks where the prosthetic crown should end. It is recommended to place the finish line approximately 1 mm supragingivally to reduce the impact of the prosthesis on the periodontium.[1,2,22,23] Another goal is to have the maximum surface area prepared for coverage by the prosthesis to improve retention. Although it might seem advantageous to do the crown preparation and impressions during the same anesthetic episode as the crown-lengthening procedure, this can lead to suboptimal results. Following the surgery, the soft tissues will often shrink and contract during healing, further lengthening the crown.[6] Waiting for 2–4 weeks after surgery can net another millimeter of clinical crown for prosthetic retention. Also, if impressions are obtained immediately following the surgery, the suture material may become trapped in the impression material, making it difficult to remove the impressions without tearing the sutures and damaging the flap margins. Therefore, it is best to do the crown-lengthening procedure at one visit and wait a few weeks for the tissues to heal. Then the crown can be prepared with a finish line placed 1 mm coronal to a stable gingival margin and impressions can be obtained without damage to tissues and sutures.

Prosthetic crown retention

The long-term success of a prosthetic crown largely depends on its retention and resistance, which must be sufficient to withstand the dislodging forces the prosthesis will meet during clinical use. Retention and resistance are influenced by a number of factors, including the total surface area of clinical crown in contact with the luting agent and the shape of the crown preparation.[12] For full-crown coverage of the canine teeth in dogs, a minimum of 6 mm of clinical crown is suggested.[24] Less than that will provide insufficient surface area of tooth-cement-prosthesis interface for retention. Crown fractures may result in less than 6 mm of natural tooth structure remaining above the gingiva. In such cases, crown-lengthening can provide the necessary clinical crown length for restoration.

The shape of the crown preparation is important in that it will determine the angle at which dislodging forces meet the prosthesis and, by extension, the cement film. All luting agents are strongest in compression, intermediate in shear, and weakest in tension. Developing a crown preparation that maximizes the mechanical retention of the prosthesis while minimizing tension forces on the cement film will increase the chances of success. An ideal preparation would have nearly parallel sides with each opposing axial wall tapering coronally at approximately 3 degrees, giving a total convergence angle of 6 degrees.[12] The conical shape of an undamaged canine in a dog typically imposes a far greater degree of taper and some fractures result in even greater taper.[25] Crown buildup with bonded restoratives can reduce this effect, although these represent another interface at which failure under load can occur. If the fracture to the crown is such that a type II crown-lengthening procedure will not result in sufficient clinical crown length and acceptable clinical crown shape for adequate retention and resistance, then a prosthetic crown is likely to fail.

Surgical techniques

Type II crown-lengthening involves raising full-thickness flaps buccally and lingually, ostectomy to move the alveolar margin apically, and apical repositioning of the flaps. When a fracture or caries extends below the preoperative free gingival margin, the new alveolar margin should be placed 2 mm or more apical to the most apical extent of the lesion.[6,10] As gingiva and periodontal ligament will not attach firmly to restorative materials, it is essential to expose some root cementum apical to the restoration for gingival attachment and reestablishment of biologic width. The lesion can then be restored to reestablish the desirable cervical anatomy and emergence profile.[26]

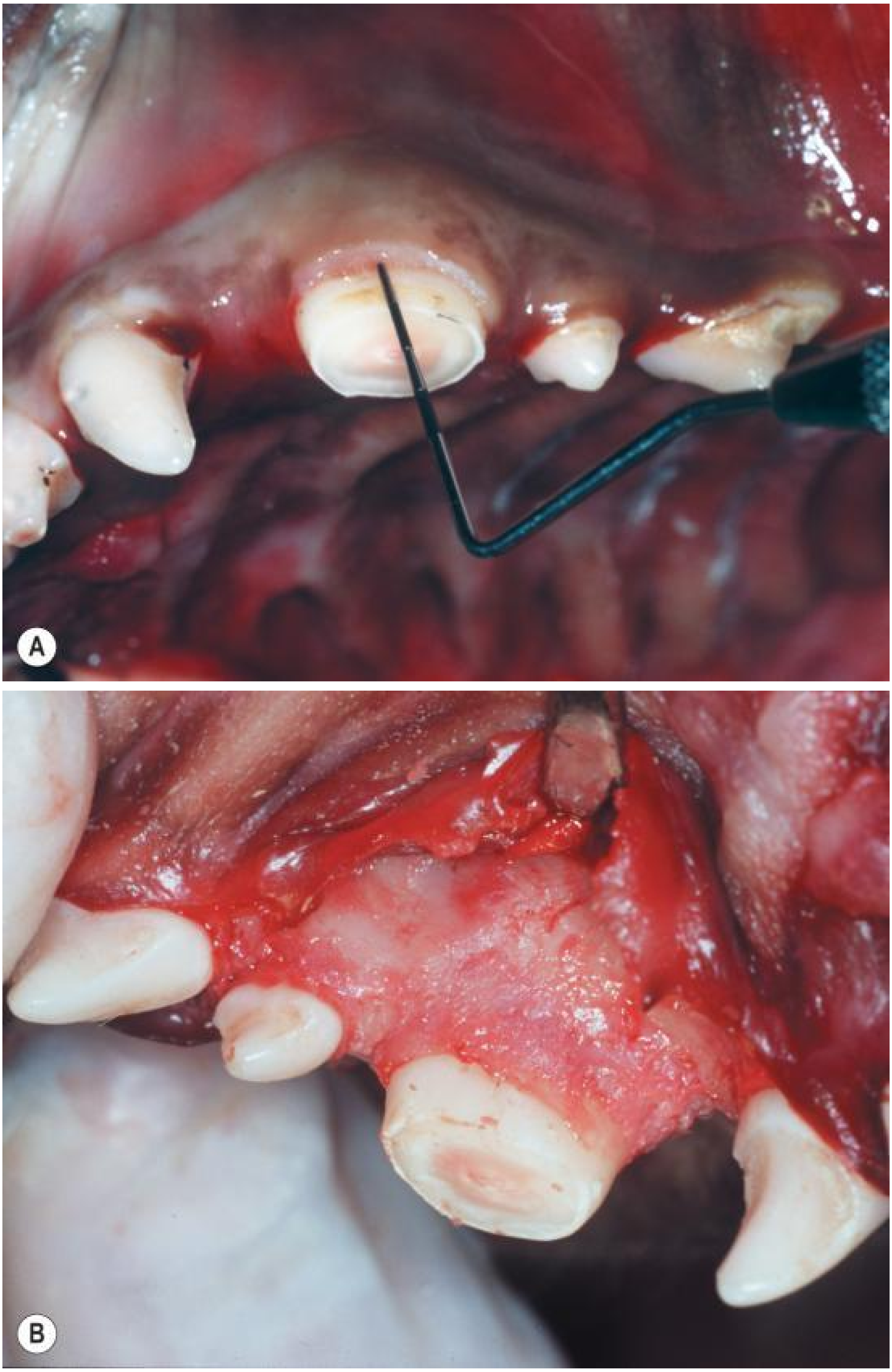
When increasing crown length in preparation for prosthodontic work, the alveolar margin should be a minimum of 3 mm apical to the planned new free gingival margin location, to allow for the reestablishment of biologic width and a gingival sulcus.[6]

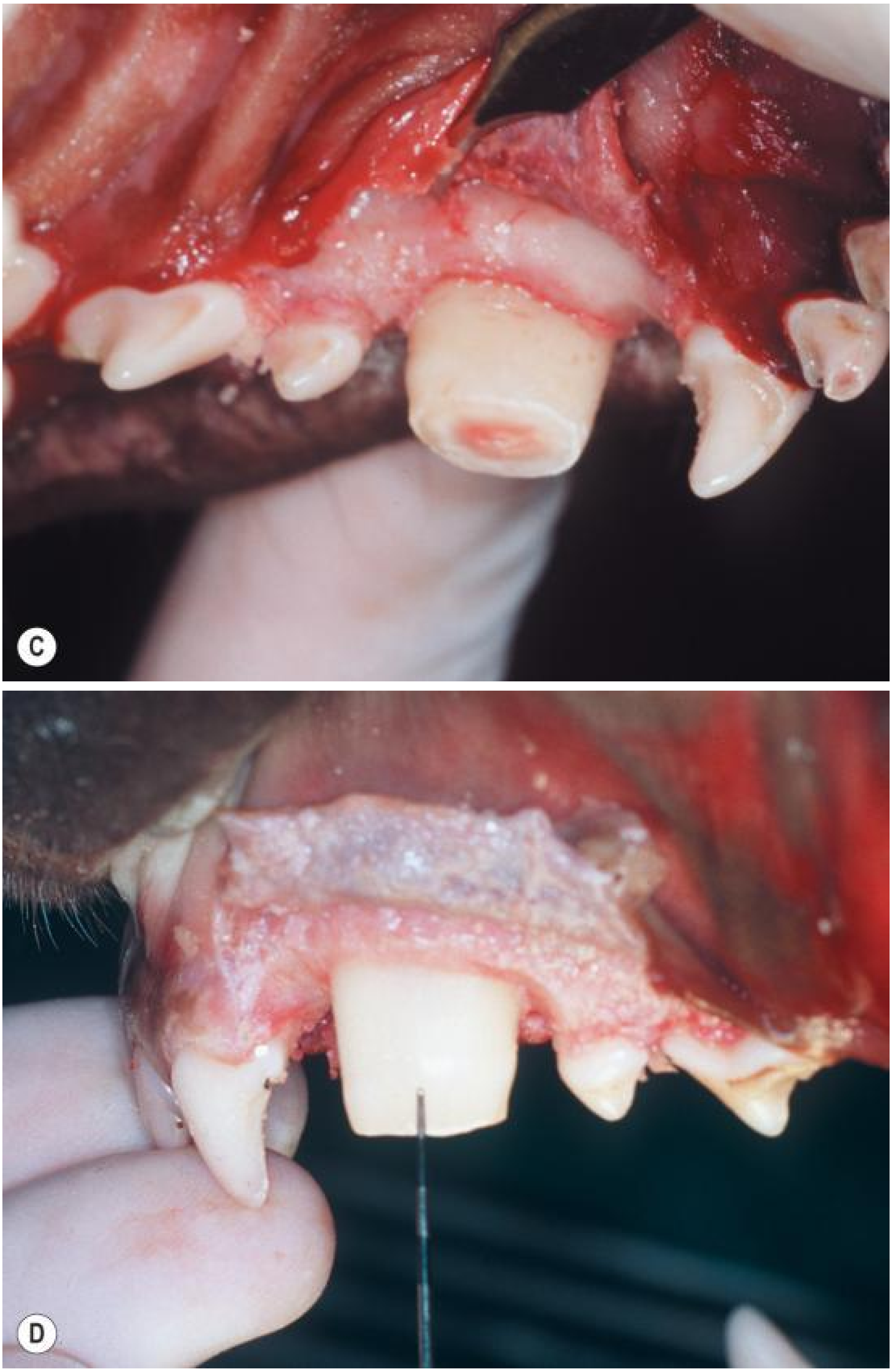
Techniques for maxillary and mandibular canine teeth, and for the maxillary fourth premolar tooth in the dog, will be described. The principles discussed can be applied to other teeth as well.

Maxillary canine tooth[5]

If the crown needs only be lengthened by a few millimeters and there is abundant healthy gingiva, a simple gingivectomy (type I crown-lengthening) may suffice. In considering this approach, it is important to maintain the biologic width.

Type II crown-lengthening starts with the creation of extended envelope flaps buccal and palatal to the tooth (Fig. 23.3). The gingiva is incised from the distal aspect of the third incisor tooth up to the mesial aspect of the second premolar tooth, including sulcular incisions of the canine and first premolar teeth. A full-thickness flap is raised. The buccal flap is reflected dorsally and the palatal flap is retracted toward the midline. If necessary, the envelope flap may be extended mesially or distally, or vertical releasing incisions may be added.

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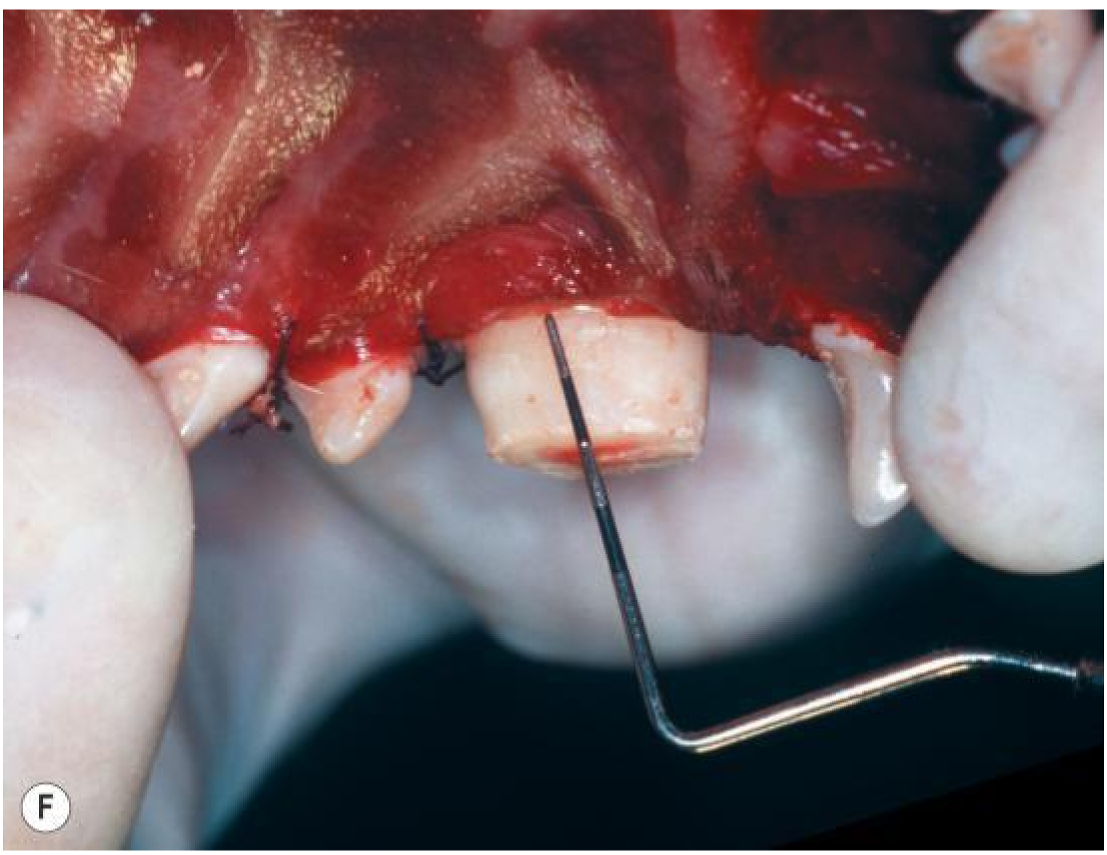


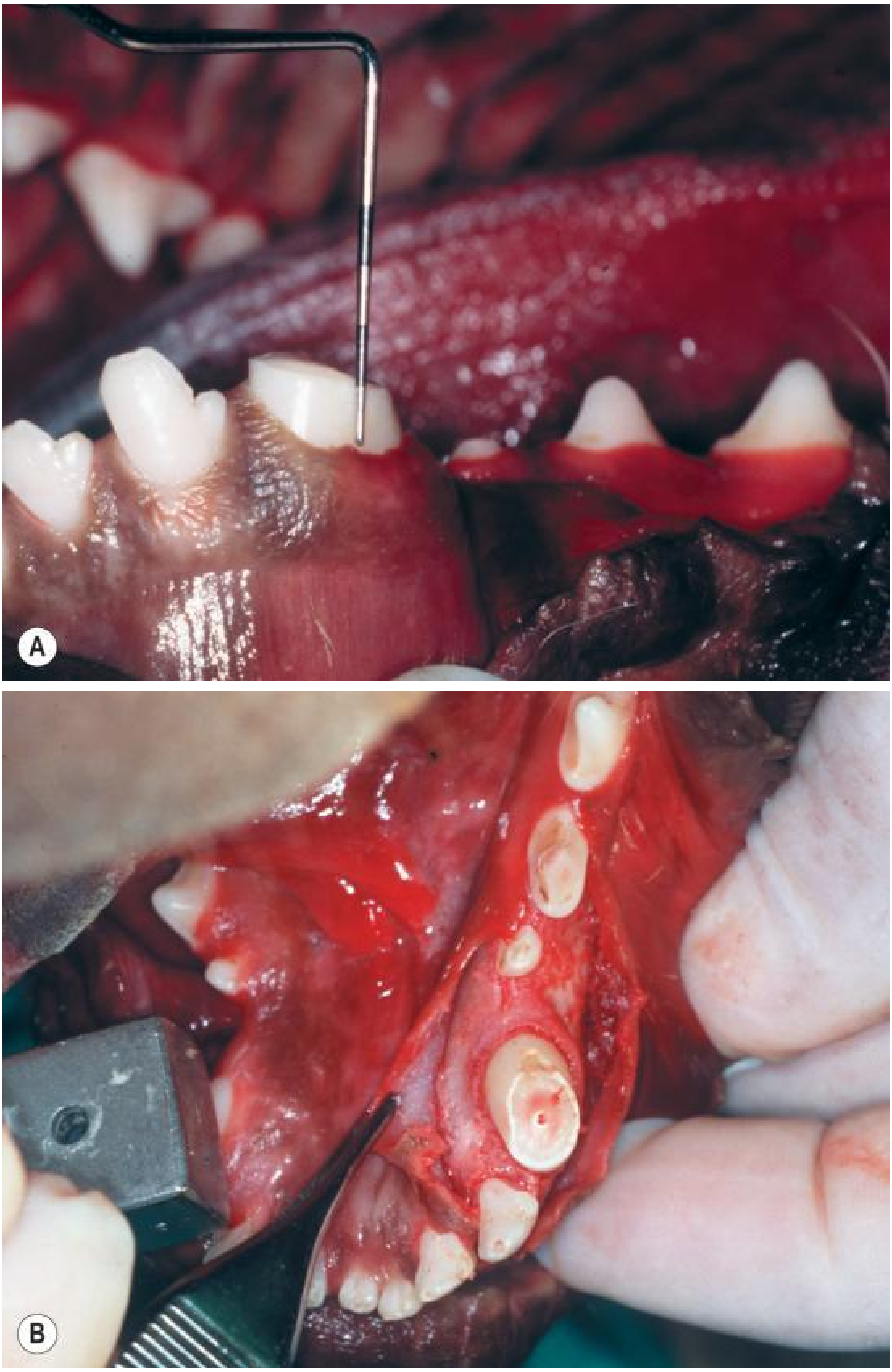
• **FIG. 23.3** (**A**) Simulated crown fracture of the left maxillary canine tooth in a cadaver specimen (lateral recumbency); the periodontal probe indicates a clinical crown length of 3 mm. (**B**) Palatal envelope flap raised, showing the height and shape of the alveolar margin. (**C**) The palatal aspect of the tooth following ostectomy and osteoplasty. (**D**) The buccal aspect of the tooth following ostectomy and osteoplasty, with the periodontal probe indicating the preoperative level of the clinical crown length. (**E**) The buccal and palatal flaps have been repositioned and sutured; the clinical crown length is now 7 mm on the buccal aspect. (**F**) Palatal view showing 6 mm of clinical crown length. Source: (Reproduced with permission from Hale FA. Crown lengthening for mandibular and maxillary canine teeth in the dog. *J Vet Dent*. 2001;18:219–221.)

Bone chisels are used to remove alveolar margin bone. Power rotary instruments should preferably not be used, to avoid damaging the root cementum.[18] However, bulk bone removal far removed from the root surface may be more efficient with well-irrigated rotary instruments. A smooth transition should be created between the surrounding bone and the newly created alveolar margin.[10] With the level of the alveolar bone established, the alveolar margin should be shaped to recreate the thin profile of normal marginal bone. This can be achieved by paring the bone with a well-sharpened periodontal curette. The curette can also be used to plane the exposed root surface in preparation for gingival attachment.

Debris from under the flap should be carefully removed. The flaps are then apposed around the tooth and the amount of crown-lengthening achieved is evaluated. If necessary, the flaps are elevated again and more bone is removed until the goal of the procedure has been met. The buccal and palatal flaps are then sutured where they meet interdentally. As it is not possible to suture the margin of a flap to the adjacent tooth wall, sling sutures may be used to draw the gingiva into close apposition with the buccal and palatal tooth walls.[27] For the palatal flap, the needle passes through the flap near the mesiopalatal line angle, around the tooth on the buccal side to the distopalatal line angle, through the flap, and then back around the buccal aspect to meet itself where it started. As the knot is pulled snug, the palatal flap is drawn tight against the tooth surface. A similar suture can be used to draw the buccal flap against the tooth if needed.

Mandibular canine tooth[5]

A buccal flap is elevated starting with an intrasulcular incision buccally to the third incisor tooth, continuing interproximally, buccally around the canine tooth, through the gingiva along the dorsal margin of the mandible distal to the canine tooth, and buccally around the first premolar tooth (Fig. 23.4). The gingiva between the mandibular canine and first premolar teeth is a fairly narrow band that runs along the dorsal margin of the mandible. Since the gingiva holds a suture well, it is recommended to keep the incision within this narrow strip of gingiva. Once the incision has been made, the flap is reflected ventrally, taking care to identify and protect the neurovascular structures emerging from the mental foramina. A lingual flap is elevated in a similar fashion. If necessary for adequate exposure, the flaps may be extended mesially or distally.

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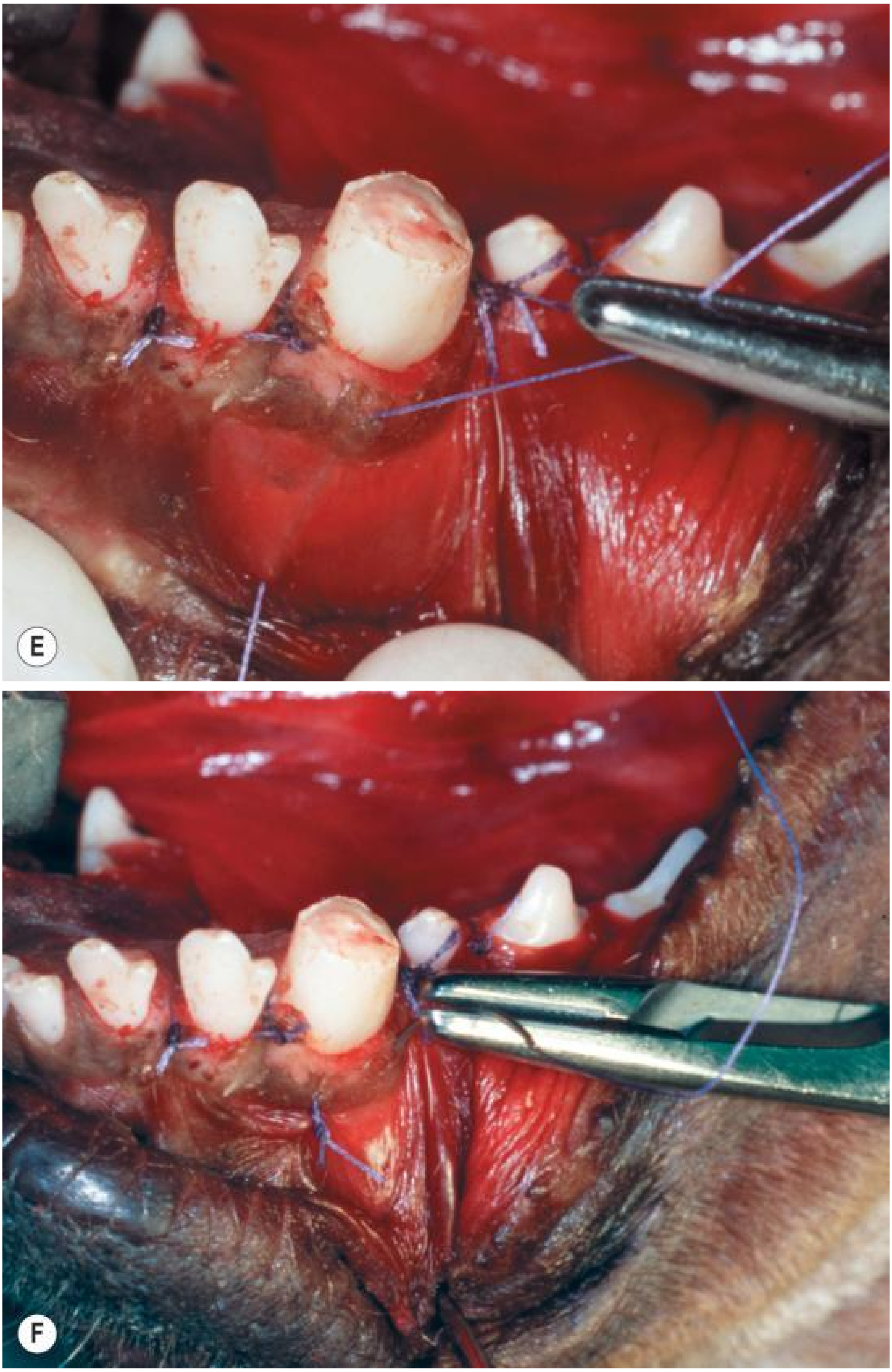

• **FIG. 23.4** (**A**) Simulated crown fracture of the left mandibular canine tooth in a cadaver specimen (lateral recumbency); the periodontal probe indicates a clinical crown length of 3 mm. (**B**) Buccal and lingual flaps have been raised following interproximal and intrasulcular incisions. (**C**) Ostectomy is performed with an Ochsenbein chisel. (**D**) The flaps have been repositioned and sutured; the clinical crown length is now 7 mm on the buccal aspect. (**E**) To prevent the buccal flap from sliding coronally, a tacking suture is placed on the mesial aspect to anchor the gingiva to the ventral mandibular periosteum. (**F**) The mesiobuccal tacking suture has been placed and the distobuccal tacking suture is now placed. Source: (Reproduced with permission from Hale FA. Crown lengthening for mandibular and maxillary canine teeth in the dog. *J Vet Dent*. 2001;18:219–221.)

With the flaps reflected, hand chisels and rotary instruments are used for ostectomy to create a new alveolar margin approximately 3 mm apical to the planned position of the free gingival margin. The bone and tooth surfaces are finished as described earlier.

The proximity of the third incisor tooth on the mesial aspect of the mandibular canine tooth can pose a challenge. At times, achieving the operative goals for salvage of the canine tooth may lead to severe compromise of the periodontal support of the incisor tooth, and extraction of the incisor tooth may be indicated.

Closure is similar to the procedure for the maxillary canine tooth. Some gingivoplasty or gingivectomy may be necessary to allow proper adaptation of the flaps. Since the roots of the third incisor and the canine teeth converge apically, the interdental gingiva may be too wide to fit into the tighter interdental space. The lingual flap may lie against the tooth in its original location despite removal of the underlying bone. In both cases, trimming of the gingiva to improve tissue apposition is indicated.

The buccal flap has a tendency to slide up the crown of the canine tooth toward its original location. To hold the flap in a more apical position during healing, a tuck suture can be employed. The needle is passed through the gingiva of the flap near the distobuccal line angle of the canine tooth, passes below the flap ventrally, engages the periosteum near the ventral border of the mandible, and emerges through the oral mucosa at the base of the vestibule. The suture is then brought back up to the entry point and tied. As the knot is snugged down, the mobile flap will be pulled ventrally toward the immobile ventral periosteum. A second tuck suture can be placed in a similar fashion at the mesiobuccal line angle of the canine tooth. The sutures should be pulled tight enough to place the free gingival margin of the flap at the desired location to achieve sufficient crown-lengthening without compromising biologic width.

Maxillary fourth premolar tooth

Crown-lengthening of the maxillary fourth premolar tooth typically involves an apically repositioned flap on the buccal side only, as the injury is almost always a subgingival slab fracture of the buccal wall. The main limiting factor in crown-lengthening for the maxillary fourth premolar tooth, or any multirooted tooth, is furcation involvement. Careful evaluation of the animal's overall periodontal health and the level of home care anticipated is essential in deciding if salvage or extraction is more appropriate.

In a periodontally sound patient who will receive daily dental home care, some crown-lengthening may be possible, although the buccal flap cannot be apically repositioned such that the furcation would be left exposed.

Given the tight interdental contacts between the maxillary fourth premolar and the adjacent third premolar and first molar teeth, an envelope flap may not provide sufficient exposure or allow sufficient apical repositioning without compromising the adjacent teeth. Therefore, vertical releasing incisions may be made at the distobuccal line angle of the third premolar or the mesiobuccal line angle of the first molar teeth. An intrasulcular incision severs the gingival attachment and the flap is elevated dorsally.

With the flap reflected, the extent of the subgingival damage can be assessed; hairline cracks and fracture fragments must be identified. As previously described, the alveolar margin is reduced with hand instruments to a level 2 mm apical to the extent of the damage. If this cannot be done without going more than a few millimeters apical to the furcation, then extraction is indicated.

With the lesion exposed, any lost root tissue is replaced with restorative materials to reestablish a desirable emergence profile. The flap is then apically repositioned and sutured in place interdentally.

The free gingival margin should be 2 mm coronal to the furcation to allow gingival reattachment; however, it should not be more than 3 mm coronal to the apical extent of the restoration to which gingiva cannot attach or a deep periodontal pocket will be the result. If a releasing incision was made, there will be a step from the unmoved free gingival margin down to the apically repositioned free gingival margin. It is important to ensure that the repositioned gingiva has some contact with the adjacent gingiva such that there can be a contiguous band of gingiva around the teeth. This is another factor that can limit the degree of crown-lengthening that can be achieved.

Postoperative care and assessment

Following surgical crown-lengthening, clients should be instructed to feed only soft food. The patient should be denied access to hard or abrasive chew toys. These measures are intended to protect the flaps from mechanical forces that might disrupt or delay healing.

Toothbrushing should be suspended for 2 weeks, or the operative side of the mouth should be avoided at least. Various nonmechanical plaque control strategies as outlined in Chapter 19 should be employed. An Elizabethan collar should be sent home with the patient to prevent the animal from pawing or rubbing at the mouth and disrupting healing.

After healing but prior to crown preparation, the periodontal health status of the tooth (and adjacent teeth) should be assessed. The tissues should be examined to evaluate the healing and the circumference of the tooth gently probed to evaluate the level and quality of attachment. Any periodontal concerns must be addressed prior to proceeding with the prosthodontic treatment.

Complications

The most significant complication of type II crown-lengthening is dehiscence of the gingival flaps. This can happen if the flaps are under tension or if the animal engages in behavior (chewing and pawing) that stresses the suture line. Should this occur, there will usually be retraction of the flaps away from the surgical site, exposing bone and the periodontal space to contamination. There will also be some loss of gingival tissue.

Treatment of dehiscence begins with reflecting the flaps to expose the underlying tissues for debridement of gross debris and contaminated tissue. The flaps may need to be undermined further to allow tension-free apposition. The flap margins may be covered with epithelium, which will need to be removed to allow apposition of fresh connective tissue margins. Since the initial surgery will have decreased the blood supply to the flaps, delayed healing and dehiscence may occur with subsequent surgery.

Other complications include compromise of the periodontal status of treated or adjacent teeth. Excessive bone removal leading to a poor crown-length:root-length ratio and tooth mobility may necessitate extraction of adjacent teeth.

Gouging of the alveolar bone at the treated tooth can cause the free gingiva to invert instead of properly adapting to the tooth surface. This results in entrapment of food and debris.

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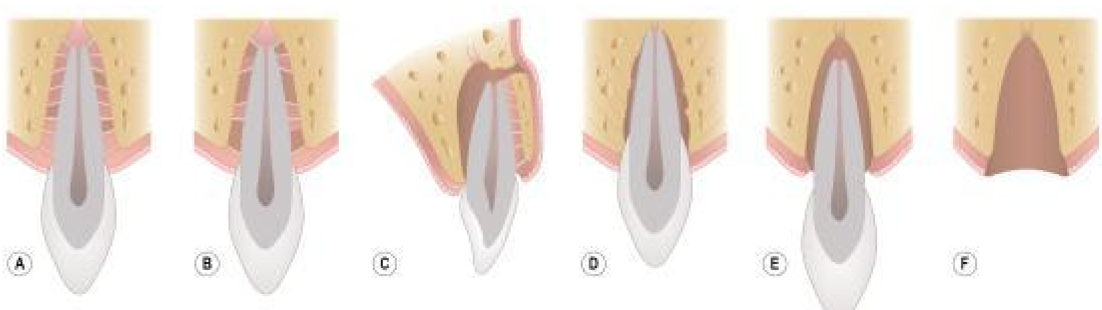
CHAPTER 24

Management of periodontal trauma

Margherita Gracis

Classification and definitions

Traumatic dental injuries (TDIs) or dentoalveolar injuries include injuries to the teeth (i.e., dental fractures) and to their supporting structures (periodontal trauma). Several diagnostic classification systems for TDIs are available. The World Health Organization has adopted a widely used system, based on the work of Andreasen and Andreasen, that includes injuries to the teeth, supporting structures, gingiva, and oral mucosa and including anatomic, therapeutic, and prognostic considerations.[1] Periodontal injuries are those where teeth are not damaged but displaced and are classified as concussion, subluxation, luxation (lateral, intrusive, or extrusive), and avulsion (Fig. 24.1).



• **FIG. 24.1** Periodontal injuries. (**A**) Concussion. (**B**) Subluxation. (**C**) Lateral luxation. (**D**) Intrusion. (**E**) Extrusion. (**F**) Avulsion. Source: (From Fonseca RJ, Walker RV, Betts N, Barber HD, eds. *Oral and Maxillofacial Trauma*. 2nd ed. Philadelphia: WB Saunders, 1997. With permission.)

Concussion

Concussion is an injury of supporting structures of the teeth characterized by marked tenderness to percussion but no abnormal loosening or displacement.[1] In mild cases, discomfort is due to bleeding in the periodontal ligament.[1,2] No radiographic abnormalities are usually visible, except for a slight widening of the periapical periodontal ligament space.[3] In humans, even though the incidence of complications following concussion is very low,[1] it is recommended to radiographically monitor these teeth for pulpal condition for at least 1 year after the trauma.[4] Tooth mobility, in conjunction with other injuries (i.e., coronal fracture), significantly increases the incidence of pulpal necrosis.[2] Furthermore, damage to the gingival tissues may induce root resorption, possibly because of odontoclastic activation.[5]

More severe blunt trauma may also cause pulp hemorrhage and edema. Because of increased pressure within the endodontal system, blood may invade the dentinal tubules, causing secondary intrinsic tooth discoloration (Fig. 24.2). Typically, with time, the crown's color changes from pink to blue, purple, tan, or gray, following the typical degradation of hematic pigments. A clinical study performed in dogs showed that the pulp of intrinsically discolored teeth may actually be necrotic in 92.2% of cases.[6] The diagnosis of endodontal disease was based on radiographic examination and gross visual inspection of the pulp, considering the absence of bleeding during endodontic treatment or exploratory pulpotomy as a sign of pulp necrosis. Importantly, it was shown that not all teeth with necrotic pulp showed radiographic signs of disease.[6] Treatment was therefore recommended for all discolored teeth, irrespective of the radiographic findings.


A

• **FIG. 24.2** Concussion injury of the right maxillary canine tooth in two different dogs, with (**A**) total or (**B**) partial coronal discoloration indicative of pulp injury.

Subluxation

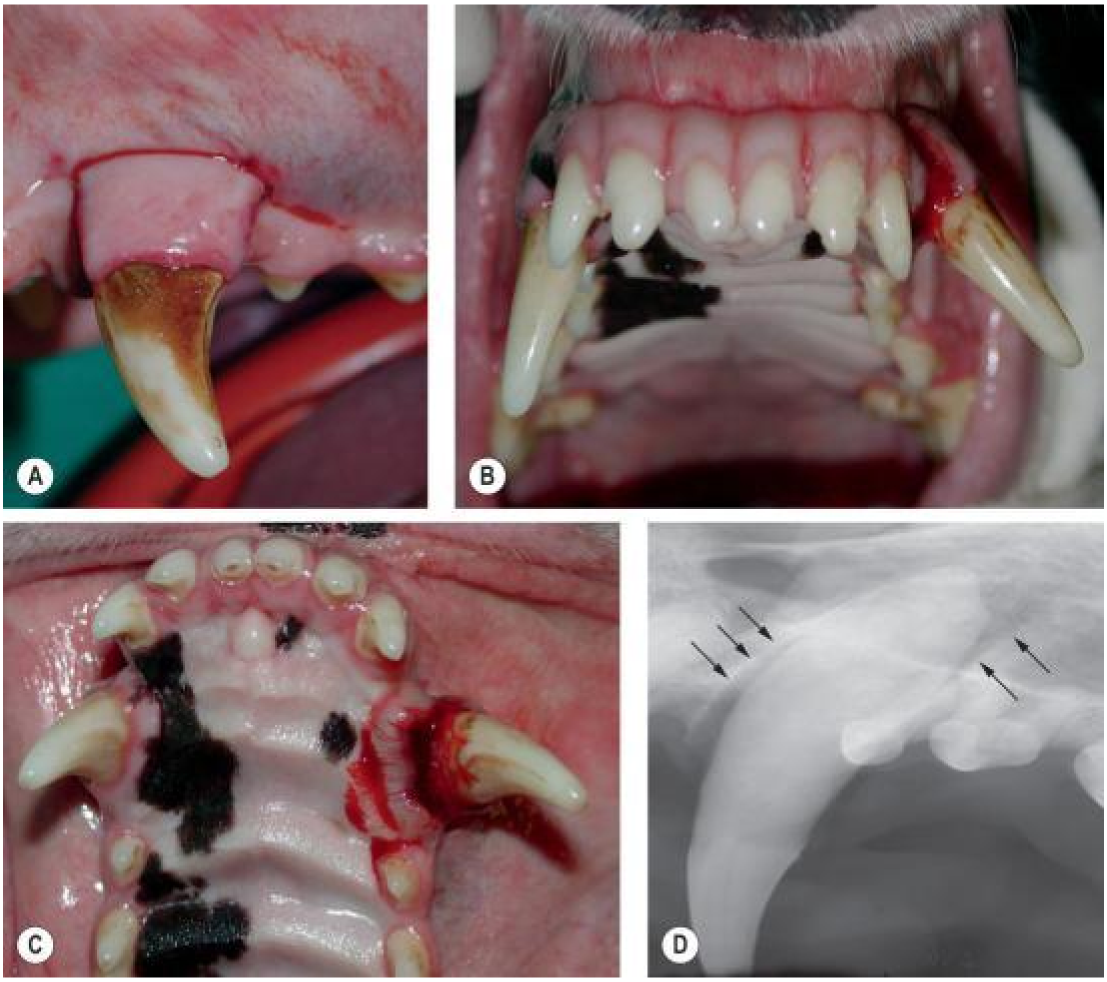
Subluxation is an injury of the periodontal ligament with loosening of the tooth, but without displacement.[1] The tooth may be sensitive to percussion and biting pressure, and there may be some bleeding from the gingival sulcus. A slight widening of the periodontal ligament space may be noted radiographically.[7] A subluxated tooth usually does not require treatment. However, even after minor displacement, the tooth should be monitored clinically and radiographically for late development of complications.[1,2] In human patients showing discomfort, a flexible splint may be used to stabilize the tooth for up to 2 weeks.[4]

Luxation

Luxation is the partial displacement of the tooth in a lateral direction (lateral luxation), into the alveolus (intrusive luxation), or out of the alveolus (extrusive luxation).[1]

Lateral luxation

Lateral luxation is the displacement of a tooth in a horizontal direction and is usually accompanied by comminution or fracture of the alveolus and laceration of mucogingival tissues (Fig. 24.3A–C).[1]

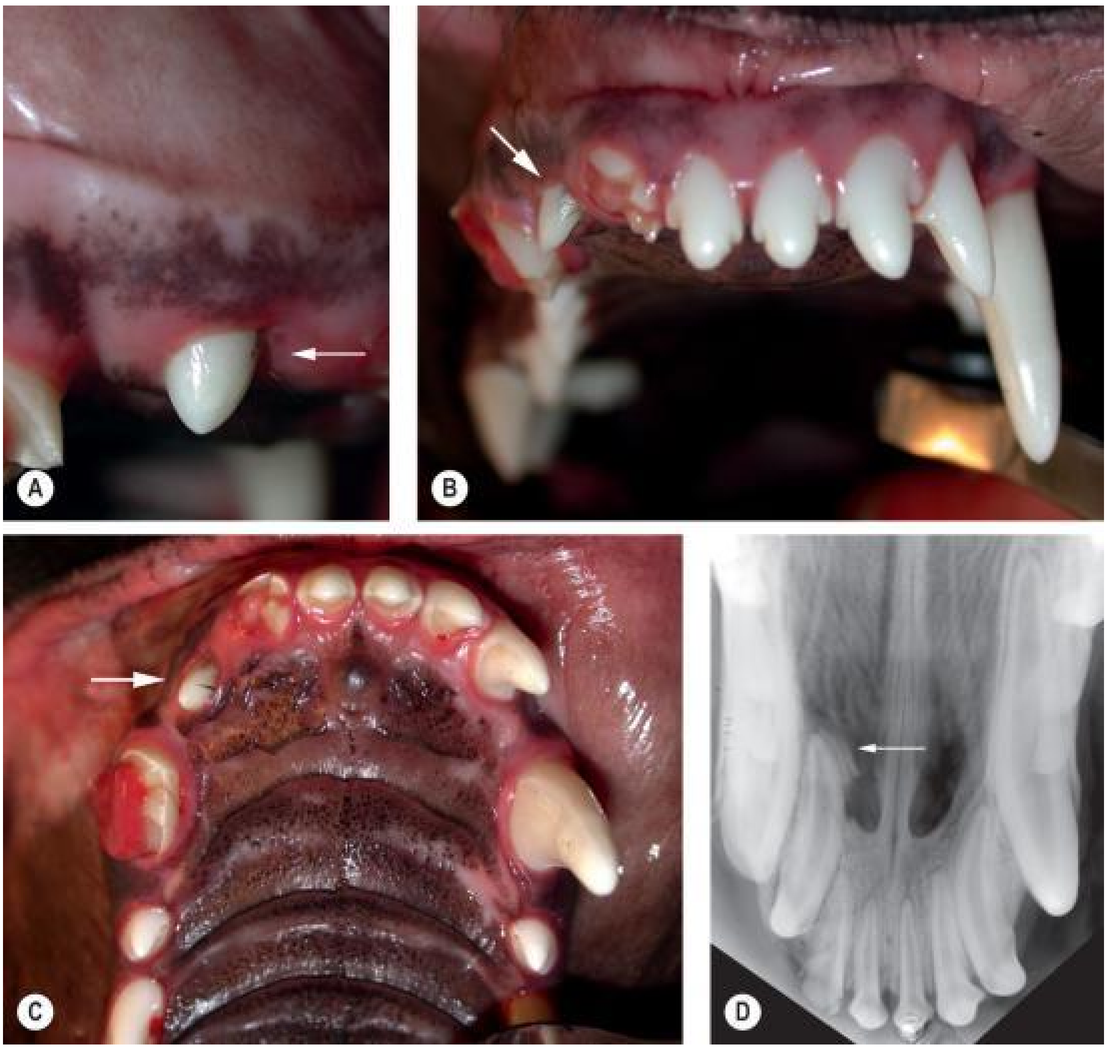


• FIG. 24.3 (**A–C**) Lateral luxation of the left maxillary canine tooth in a 10-year-old dog following a fight with another dog. Note the labial displacement of the tooth and laceration of the soft tissues. (**D**) Intraoral radiographic view. Note the increased distance between the tooth root and the alveolar walls *(arrows)*.

Radiographic signs include widening of the periodontal space and displacement of the lamina dura (Fig. 24.3D). These changes may not be visible, depending on the angle of the X-ray beam.[3,7,8]

Intrusive luxation

Intrusion is the apical displacement of the tooth into the alveolus (Fig. 24.4A–C).[1,9] This type of injury is usually accompanied by fracture of the alveolus and is associated with a very high incidence of complications such as pulp canal obliteration, pulp necrosis, root resorption, ankylosis, and marginal bone loss.[10-12] Clinically, an intruded tooth will be nonmobile. A percussion test produces a dull metallic sound similar to that of ankylosed teeth, which is useful in differentiating the intruded tooth from a tooth that is incompletely erupted.[1] Radiographically, the periapical periodontal ligament space is absent and the lamina dura is discontinuous (Fig. 24.4D).[3,11] Experimental studies in dogs showed that minimally intruded teeth may reerupt spontaneously, particularly if still immature.[13,14] If the injury is severe, the tooth should be returned to its normal position, manually or orthodontically, and splinted to adjacent teeth. The orthodontic movement should be initiated immediately[13] or within the first few days following injury to decrease the incidence of resorption and ankylosis.[1,11,15] Orthodontic movement should be performed slowly, over 3–4 weeks, to minimize the chances of iatrogenic avulsion. In human dentistry, extraction of intruded deciduous teeth is recommended only if there is invasion of the permanent developing bud by the deciduous tooth, or in case of local infection. One experimental study involving dogs demonstrated that there was no difference in permanent tooth alterations following maintenance of the intruded deciduous tooth in the alveolus or extraction.[16]



• **FIG. 24.4** (**A–C**) Intrusive luxation of the right maxillary third incisor tooth *(arrow)* in a 2-year-old dog following a car accident. The second incisor and canine teeth suffered complicated crown-root fractures. (**D**) Intraoral radiograph showing displacement of the right third incisor tooth into the nasal cavity, with fracture of the apical portion of the alveolus *(arrow)*. A processing artifact is present on the crown of the left maxillary first incisor tooth.

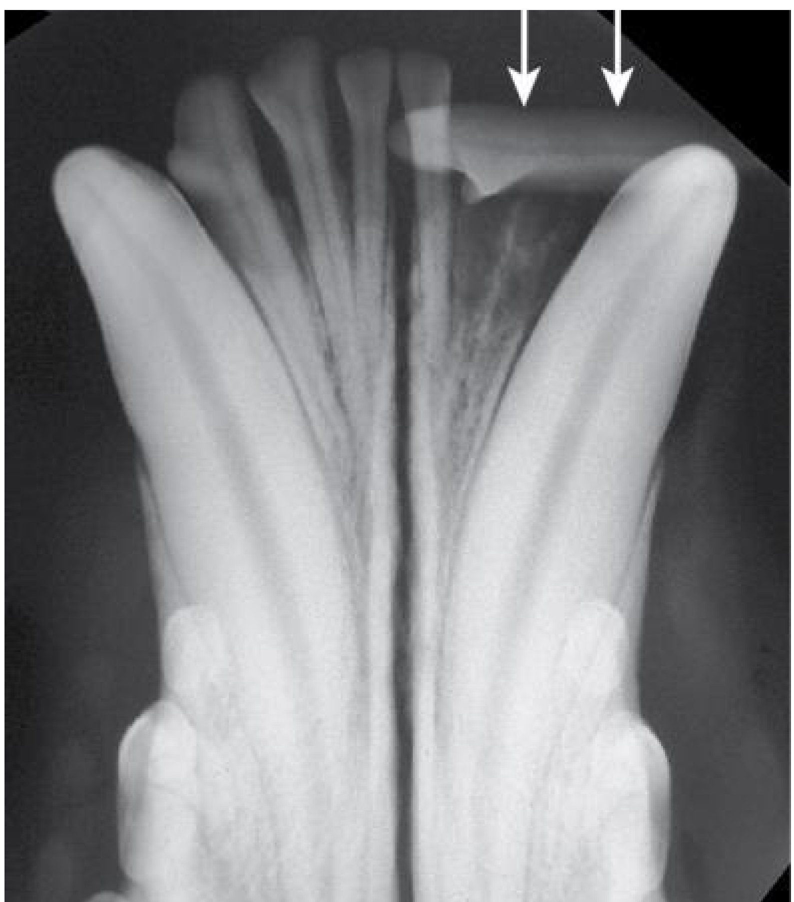
Extrusive luxation

Extrusion is the partial displacement of a tooth out of its alveolus.[1] The tooth may appear elongated and there may be bleeding from the periodontal space. Radiographic examination reveals an increased width of the periodontal ligament space, particularly in the periapical area.[3]

Avulsion (exarticulation)

Avulsion, or exarticulation, is the complete displacement of the tooth from the alveolus (Fig. 24.5A).[1] Following trauma, a radiographic examination should always be performed to differentiate avulsion from severe intrusion or root retention. Radiographically, the tooth is absent and the alveolus appears empty (Fig. 24.5B).[3] Bony fractures may be present.


A


B

• FIG. 24.5 (**A**) Avulsion of the left mandibular second and third incisor teeth in a 3-year-old dog following a car accident. The third incisor tooth is still attached to the lingual gingival tissues; the second incisor tooth has been lost. (**B**) Intraoral radiograph showing the empty alveoli and displaced third incisor tooth (*arrows*).

Epidemiology

The total prevalence of TDIs in canine and feline dental patients was 26.2% in a recent study, with the majority of injuries affecting dogs rather than cats.[17] Periodontal injuries affected mainly young animals (<3 years) and accounted for 17.3% of all TDIs in the study population. Concussion (with tooth discoloration) was the most common periodontal injury (83.1%), followed by avulsion (9.0%), lateral luxation (4.8%), extrusive luxation (1.2%), intrusive luxation (1.2%), and subluxation (0.6%) injuries.[17]

In a different retrospective study of patients treated for maxillofacial injuries, dental displacement injuries accounted for 29.0% of all TDIs.[18]

The teeth most commonly affected in dogs and cats are canine and incisor teeth (up to 79.4% of all avulsed and luxated teeth).[9,17-27]Fights with other animals, car accidents, and falls from a height are among the most frequent causes.[9,19,21-23,27] Energy of impact, resilience of the impacting object and periodontal structures, shape of the impacting object, and direction of the impacting force influence and determine the extent and type of injury (i.e., dental fracture versus dislocation).[1] Following a blow, if the bone is thick and rigid the tooth will fracture because it is more brittle, but if the bone is resilient, the tooth will be displaced.[1] Predisposing factors for avulsion or luxation (as opposed to fracture of the tooth) include immaturity of the periodontal ligament (young age), periodontal disease, and other conditions affecting the root and supporting bone. Iatrogenic dislocation may develop as a possible complication of improperly performed orthodontic treatment.

Preoperative concerns

Tooth luxation and avulsion represent dental emergencies, and the prognosis for affected teeth is dependent on early and correct management. The length of the extraalveolar period and the storage conditions before replantation have been shown to be the most important factors influencing successful healing in the treatment of traumatically exarticulated teeth.[1] Time is an important factor for success of the treatment, as the longer the tooth is out of the alveolus, the poorer the prognosis. Other important factors to be taken into consideration in the treatment of periodontal injuries are the age of the animal, patient and client compliance with aftercare, and integrity of the radicular structure of the traumatized teeth. Periodontal disease of either the teeth involved or the neighboring teeth needed for stabilization predisposes to treatment failure. The presence and extent of maxillofacial fractures and soft-tissue injuries and the presence and severity of any other head, chest, or abdominal injuries need to be addressed before treating dental injuries. When multiple anesthetic procedures are a concern for medical reasons, an affected tooth should rather be extracted. Even if controversy exists about possible indications in favor of a conservative treatment, displaced deciduous teeth should not be replaced/replanted, but either extracted or (in case of intrusive luxation) left to erupt and exfoliate spontaneously, to avoid complications on the development and eruption process of the permanent teeth.[1,28-32]

Special instruments and materials

Instruments and materials needed for splinting include the following:

- Prophy angle, rubber prophy cups, and flour pumice
- Orthodontic or soft stainless steel orthopedic wire, or woven mesh
- Wire-bending and wire-cutting instruments
- Acid-etch gel or solution and brush or ballpoint applicators
- Nonexothermic light-cured or chemically cured composite material, or acrylic resin. Triad (Triad VLC Material; Dentsply-Trubyte, York, PA) is a pink-colored, light-cured, denture base material that contains no methyl methacrylate. It is available in 100 × 100 mm sheets, ropes, and gel format, and is soft and very pliable before being exposed to light. Protemp Garant (3M–Espe, St. Paul, MN) is a self-curing, composite temporary restorative material in an automix delivery system. The system allows easy placement of the viscous material and does not require bonding agent.
- Composite instruments
- Finishing burs

Diagnosis, treatment guidelines, and techniques

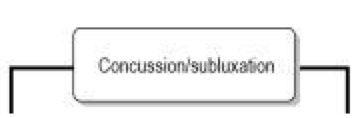
Diagnosis

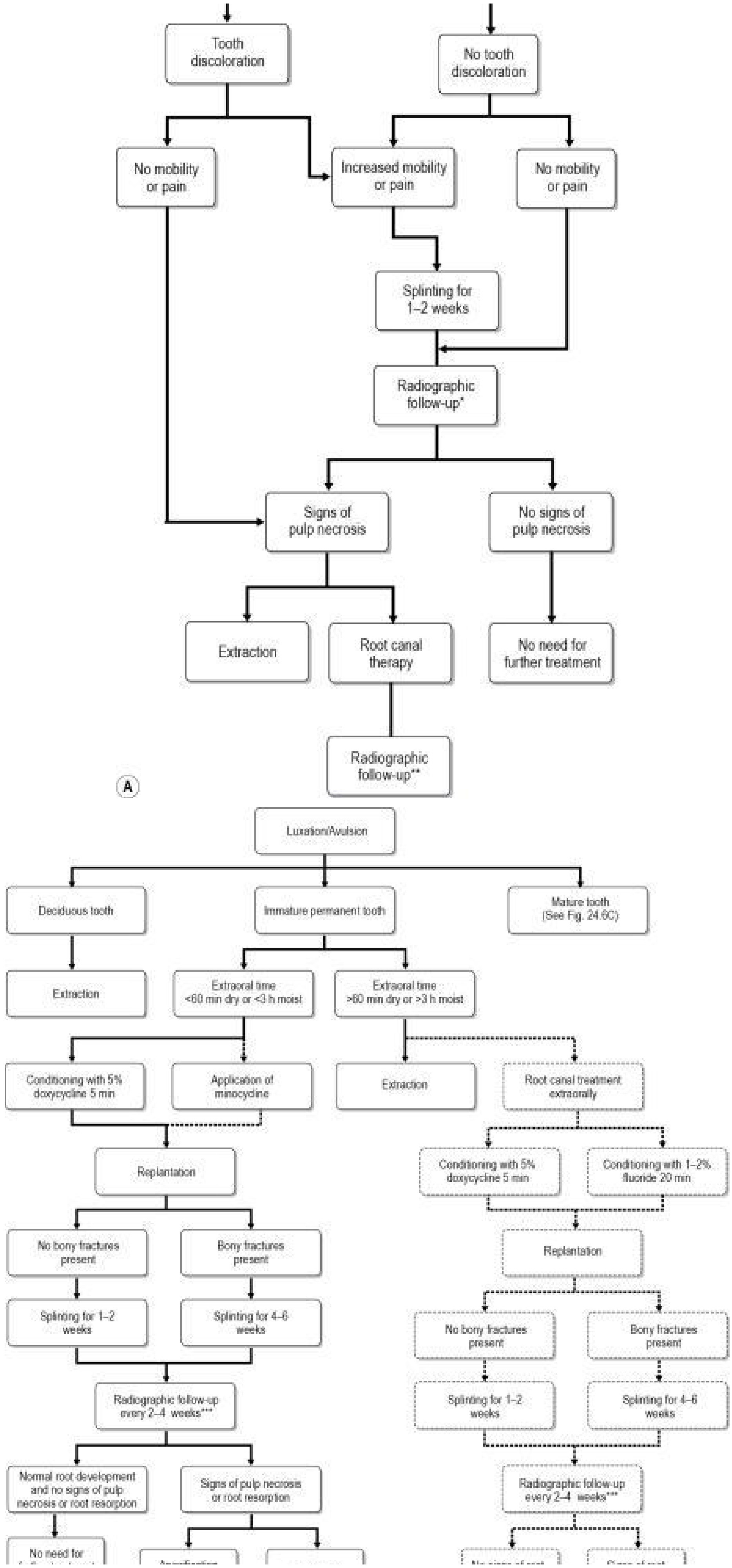
Diagnosis is mainly based on clinical examination and diagnostic imaging. Increased mobility (subluxation and luxation) or decreased mobility (intrusion), soft tissue laceration, gingival bleeding, and dental displacement or loss are among the most common clinical signs. Concomitant injuries, such as crown or root fractures of nearby teeth, are common in humans with dental luxation and subluxation.[1]

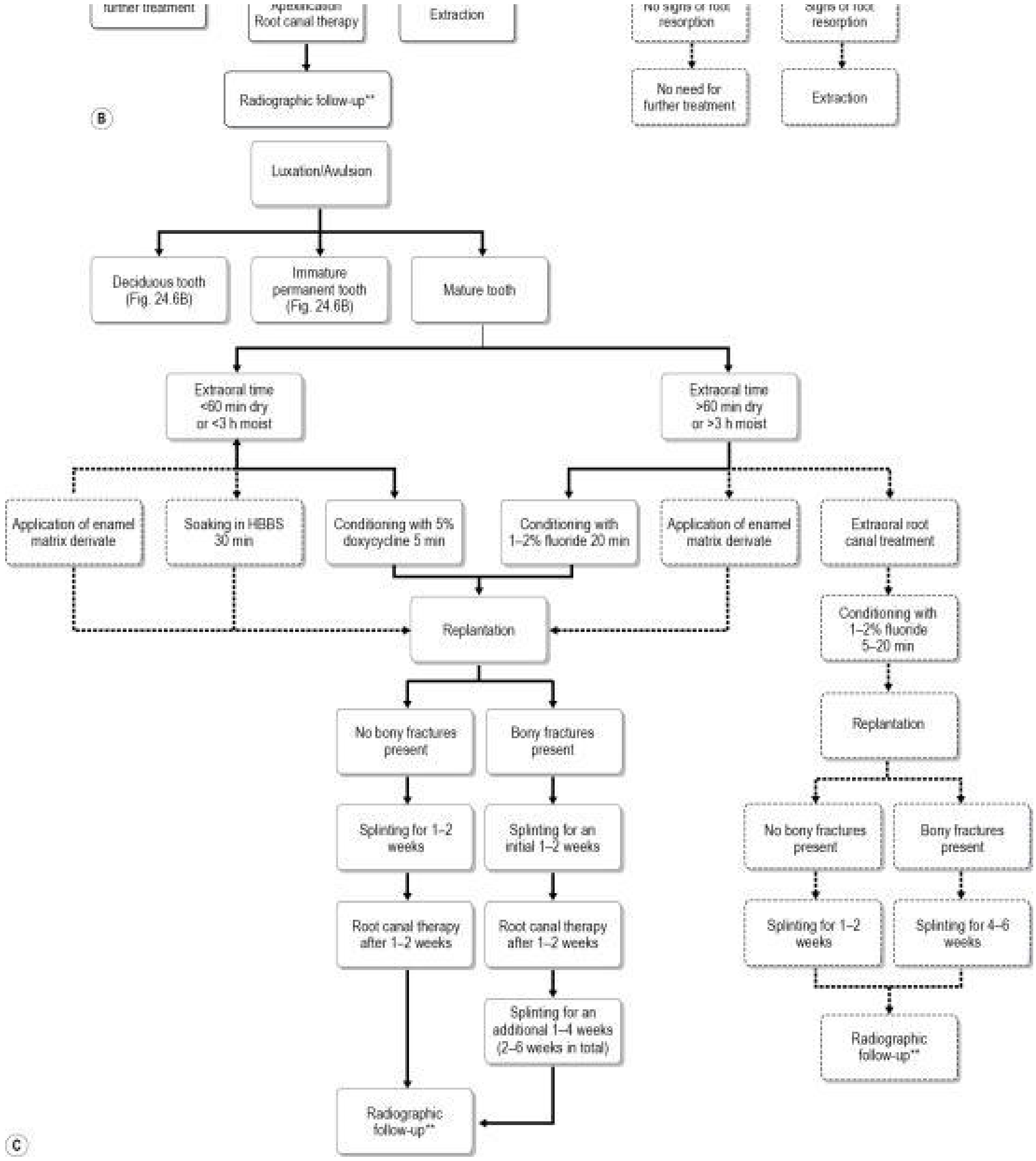
Following facial trauma, all teeth should be tested for abnormal mobility that would suggest displacement or alveolar or jaw fracture. Radiographic and or computed tomographic (CT) examination is necessary to evaluate the degree of dental displacement, presence of preexisting periodontal disease, extent of root development, size of the pulp cavity, presence of root or bony fractures, and presence of tooth fragments and foreign bodies lodged in the soft tissues.[1,2,8] If CT is not available, multiple radiographic views at different angles may be necessary to fully evaluate tooth displacement.[7,33-35]

Treatment guidelines and techniques

Except when crown discoloration develops, concussion and subluxation injuries are rarely diagnosed in veterinary patients. However, it is important to note that pulpal complications may develop even following slight dental trauma and displacement, and clinical and radiographic periodic follow-up examinations should be performed when this type of injury is suspected (Fig. 24.6A).[1]


Concussion/subluxation


Tooth discoloration
No tooth discoloration
No mobility or pain
Increased mobility or pain
No mobility or pain
Splinting for 1–2 weeks
Radiographic follow-up*
Signs of pulp necrosis
No signs of pulp necrosis
Extraction
Root canal therapy
No need for further treatment
Radiographic follow-up**
A
Luxation/Avulsion
Deciduous tooth
Immature permanent tooth
Mature tooth (See Fig. 24.6C)
Extraction
Extraoral time <60 min dry or <3 h moist
Extraoral time >60 min dry or >3 h moist
Conditioning with 5% doxycycline 5 min
Application of minocycline
Extraction
Root canal treatment extraorally
Replantation
Conditioning with 5% doxycycline 5 min
Conditioning with 1–2% fluoride 20 min
No bony fractures present
Bony fractures present
Replantation
Splinting for 1–2 weeks
Splinting for 4–6 weeks
No bony fractures present
Bony fractures present
Radiographic follow-up every 2–4 weeks***
Splinting for 1–2 weeks
Splinting for 4–6 weeks
Normal root development and no signs of pulp necrosis or root resorption
Signs of pulp necrosis or root resorption
Radiographic follow-up every 2–4 weeks***
No need for



• **FIG. 24.6** Algorithmic approaches to treatment options for periodontal trauma in dogs and cats. (**A**) Management of concussion and subluxation injuries. (**B**) Management of luxation and avulsion injuries to deciduous and immature permanent teeth. (**C**) Management of luxation and avulsion injuries to mature permanent teeth. *Broken lines* indicate second choice treatment options.
* Following concussion and subluxation, radiographic follow up should be performed at 1, 3, 6, and 12 months from the time of trauma.
** For both immature and mature teeth, if root canal treatment is performed, radiographic follow-up should be performed after 6 and 12 months and yearly thereafter.
*** For endodontically untreated immature teeth, radiographic follow-up should be performed every 2–4 weeks for 2 months, then after 6 months, 1 year, and yearly for 5 years.

The *Guidelines for Treatment of Avulsed Teeth* of the American Association of Endodontists and the *Guidelines for the Evaluation and Management of Traumatic Dental Injuries* of the International Association of Dental Traumatology (IADT)[4,28,34-37] are applicable with little modification for treatment of displaced teeth in veterinary patients (Fig. 24.6B and C). Differences are mainly due to the need for general anesthesia for dental procedures in animals. Treatment includes repositioning (luxated teeth) or replantation (avulsed teeth), management of bony and soft tissues injuries, and endodontic treatment, as in the majority of cases the blood supply to the pulp is disrupted as a consequence of the trauma.

Storage media for avulsed teeth

Successful replantation of avulsed teeth depends on the existence of viable periodontal fibroblasts that are capable of proliferating over the damaged root areas.[38] Teeth can be replanted without complications if reinserted into the alveolus within 60 minutes, even if stored dry.[36,39,40] Unfortunately, this is rarely possible in veterinary patients. After 60 minutes of extraoral time, the periodontal ligament begins to necrotize, which leads to extensive replacement resorption and ankylosis. If the tooth is placed in a suitable transport medium, however, it can be stored for up to 3 hours before successful replantation.[39] The optimal storage medium should be able to preserve the viability of the injured periodontal ligament cells and should have correct osmolality and pH.[41] Tap water has been demonstrated to be almost as harmful to monkeys' periodontal ligament cells as long-term dry storage because it is hypotonic and results in rapid cell lysis.[38]

Several commercial tissue culture media (e.g., Hank's balanced salt solution [HBSS]; Save-a-Tooth, SmartPractice.com, Phoenix, AZ) have been shown to be effective for storing and transporting avulsed teeth because of their ability to reconstitute periodontal ligament cells and therefore prevent root resorption.[38-48] These media, however, may not be readily available. Low-fat milk has also been shown by studies on human and canine periodontal ligament cells to be an excellent storage medium.[38,39,42,46,49-51] Milk is able to effectively maintain the viability of periodontal ligament cells for up to 6 hours, particularly if held at a cool temperature (4°C).[40,44,49,50] It contains important nutritional substances, such as amino acids, carbohydrates, and vitamins, and it has physiological osmolality and low bacterial content. Milk, however, has no restorative effect when periodontal ligament has already become dehydrated.[49] For teeth that have been kept extraorally for more than 15 minutes or that have been previously stored in milk, it is recommended to soak in HBSS for 30 minutes before replantation.[46] Milk products such as sour milk and yoghurt provide poor conditions for cell survival because of their low pH.[50] A recently published systematic review of the English-language literature on the efficiency of different storage media (including tap water, saliva, coconut water, soymilk, whole bovine milk, saline solution, HBSS, and several other, less readily available liquids) for avulsed teeth in animal models has highlighted that tap water, saliva, or saline solution should not be used to transport avulsed teeth.[52] If a tooth cannot be immediately replanted or stored in a physiologic medium, it should at least be wrapped in plastic or foil to prevent air drying.[53]

Pre-repositoning/pre-replantation treatment of the tooth and alveolus

Luxated and avulsed teeth should be minimally manipulated, and curettage of either the root or the alveolus should be avoided to prevent healthy periodontal ligament from being removed.[33,54] The alveolus should not be air dried, and surgical flaps should be avoided unless bony fragments prevent replantation. Medicaments, disinfectants, or chemicals should never be applied to the root surface.[55] Very gentle irrigation with saline or the careful use of cotton pliers is recommended to remove blood clots and gross debris.[2,43,56] The removal of the blood clot prior to replantation of experimentally avulsed dog teeth resulted in less ankylosis and resorption.[56] A chemotactic stimulus may be released from the fibrin clot if left in place, attracting neutrophils and monocytes that have detrimental effects upon the tissues through the release of lysosomal enzymes.[56]

The application of various medicaments potentially able to delay or prevent root resorption of displaced teeth, including fluoride, sodium alendronate and odanacatib (antiosteoporotic agents), dexamethasone, enamel matrix derivates, fibroblast growth factor 2 (as well as some intracanal steroids), titanium, powdered coconut water, calcium hydroxide, and indomethacin, has been investigated in animal studies.[57-70] However, because of lack of evidence-based data, controversy still exists as to which, if any, medicament is actually effective. Oral administration of nonsteroidal antiinflammatory drugs in dogs has proven ineffective in preventing resorption.[71] A new field of research concerns the regeneration of periodontal ligament-like tissue using cell-based therapy (e.g., autologous periodontal ligament cells and cell sheets) or activation of endogenous repairing system induced by biological cues (e.g., stromal cell-derived factor-1 and bone morphogenetic proteins).[72-76]

When used topically at the time of replantation, doxycycline was shown to decrease the frequency of ankylosis and inflammatory root resorption and increase the frequency of complete pulp revascularization of replanted immature monkeys' teeth.[77] Tetracyclines have also been shown to have antiresorptive properties in addition to antibacterial properties.[78] It may therefore be useful to soak avulsed teeth in a 5% doxycycline solution for 5 minutes to kill the bacteria on the root surface and in the pulp before replantation.[46] This treatment has also been shown to enhance revascularization of immature replanted teeth in dogs.[79] On the other hand, the application of the tetracycline derivative antibiotic minocycline (Arestin; OraPharma Inc., Warminster, PA) to replanted dog teeth after extended dry times did not show any benefit in the attenuation or prevention of external root resorption,[80] but it may be beneficial for pulp revascularization of immature teeth.[81]

After 60 minutes of dry storage, the periodontal ligament cells can be assumed to be necrotic. In this case, or even if the tooth is stored in one of the recommended media longer than the advised time, the periodontal ligament may be mechanically or chemically removed before replantation. In humans, it is currently recommended to immerse avulsed permanent teeth in a 2% sodium fluoride solution for 20 minutes before replantation.[37]

Repositioning and replantation

The terms repositioning and replantation, or reimplantation, refer to the insertion of a tooth in its alveolus following traumatic luxation and avulsion, respectively.[55] Repositioning and replantation differ from the following:

- Intentional replantation: intentional removal of a tooth from its alveolus and reinsertion after endodontic therapy.[82]
- Transplantation: the removal of a tooth or a tooth bud from one alveolus and its insertion into another alveolus of the same person/animal (autotransplantation) or of a different person/animal of the same species (allotransplantation).

- Implantation: the insertion of an artificial tooth into a surgically prepared or a vacated alveolus.

Repositioning and replantation should be performed with great care. The tooth should be handled by the crown and reinserted into the alveolus without effort, minimizing further damage to the periodontal ligament fibers and cementum. The tooth position should be checked radiographically before splinting.

To arrest any hemorrhage and to avoid contamination of the surgical field with dental materials, lacerated soft tissues should be sutured and bony fractures should be stabilized at the time of repositioning and replantation before splinting the displaced teeth. Bone fragments with adequate soft tissue attachment should not be discarded if possible, and the facial and lingual plates should be manually compressed to adhere to the tooth surface.[2,55]

Splinting

A dental splint is defined as "a rigid or flexible device or compound used to support, protect, or immobilize teeth that have been loosened, replanted, fractured, or subjected to certain endodontic procedures."[83] The purpose of splinting is to stabilize the tooth for as long as is required to ensure that there is no further injury and to protect the attachment apparatus in order to allow the periodontal ligament fibers to regenerate.[83]

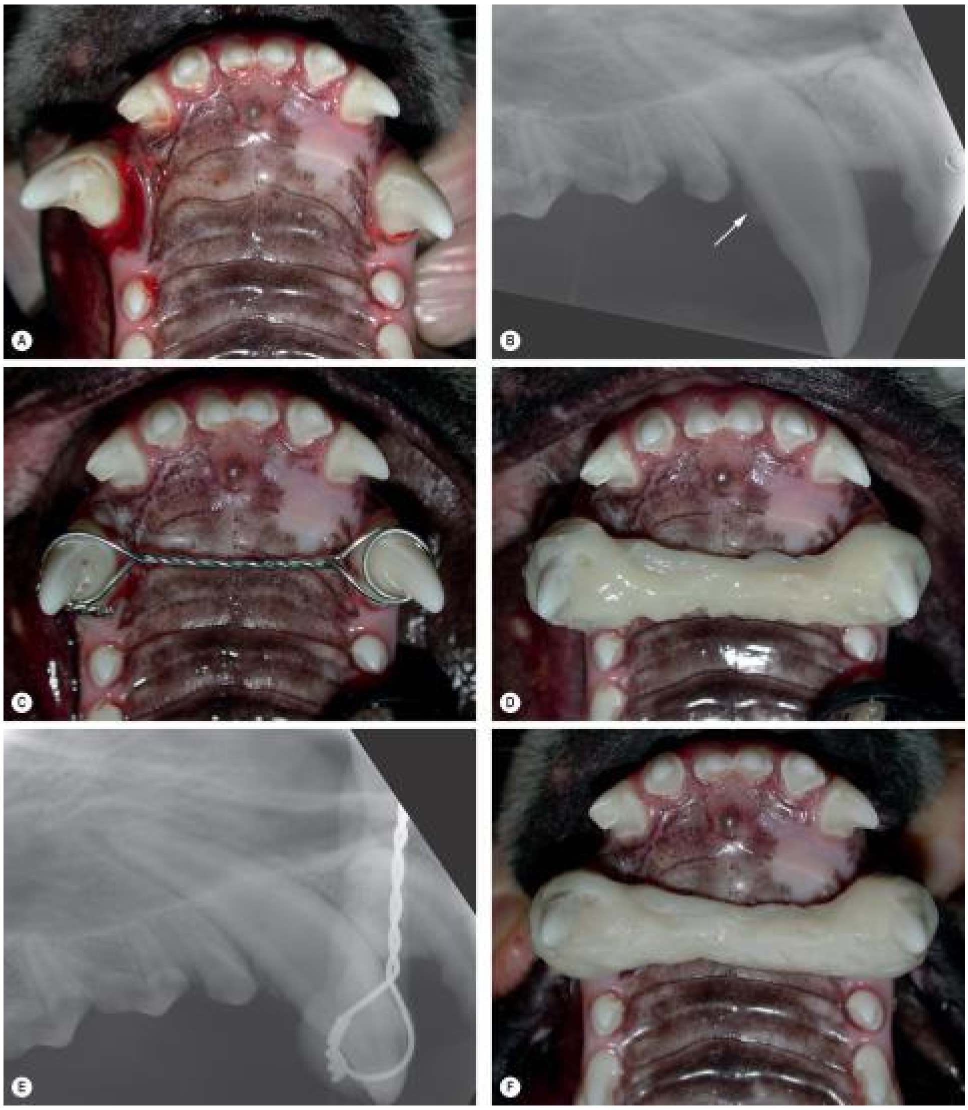
As rigid fixation has been associated with a high degree of ankylosis and root replacement resorption, flexible splints are preferred for the treatment of displaced teeth.[84] It has been shown that the normal masticatory stimulus can prevent and eliminate small external resorption areas on the root surface of replanted teeth, promotes pulpal healing, and is necessary for normal bone remodeling.[83,84]

Animal studies have proven that composite splints (with or without wire) produce adequate lateral support for replanted teeth while allowing slight mobility, which improves periodontal healing.[83,84] These splints are very reliable, easy to apply and remove, well tolerated by dogs, easy to clean for the client, and relatively inexpensive.

The splinting technique involves the following:

- Scaling the teeth to be included in the splint. If polishing is performed, nonfluoride pumice is used, as fluoride interferes with the setting of the composite and resin materials.
- Anchoring the displaced teeth to the adjacent teeth utilizing orthodontic or stainless steel wire (Fig. 24.7A–C). The splint should extend one to three teeth mesially and distally from the displaced ones, or, for displaced canine teeth, the splint should span the hard palate or the symphysis to reach the contralateral tooth (Fig. 24.7C). The size of the wire depends on the size of the dog and its teeth. The wire is shaped with the aid of orthodontic pliers to passively conform to the dental surface and is positioned a few millimeters coronal to the gingival margin to avoid contact with the soft tissue. To prevent occlusal interference, the wire and the bulk of the composite should be placed on the buccal side of the maxillary teeth and on the lingual aspect of the mandibular teeth. In brachycephalic breeds, and other animals with preexisting malocclusion, positioning may be necessarily different.

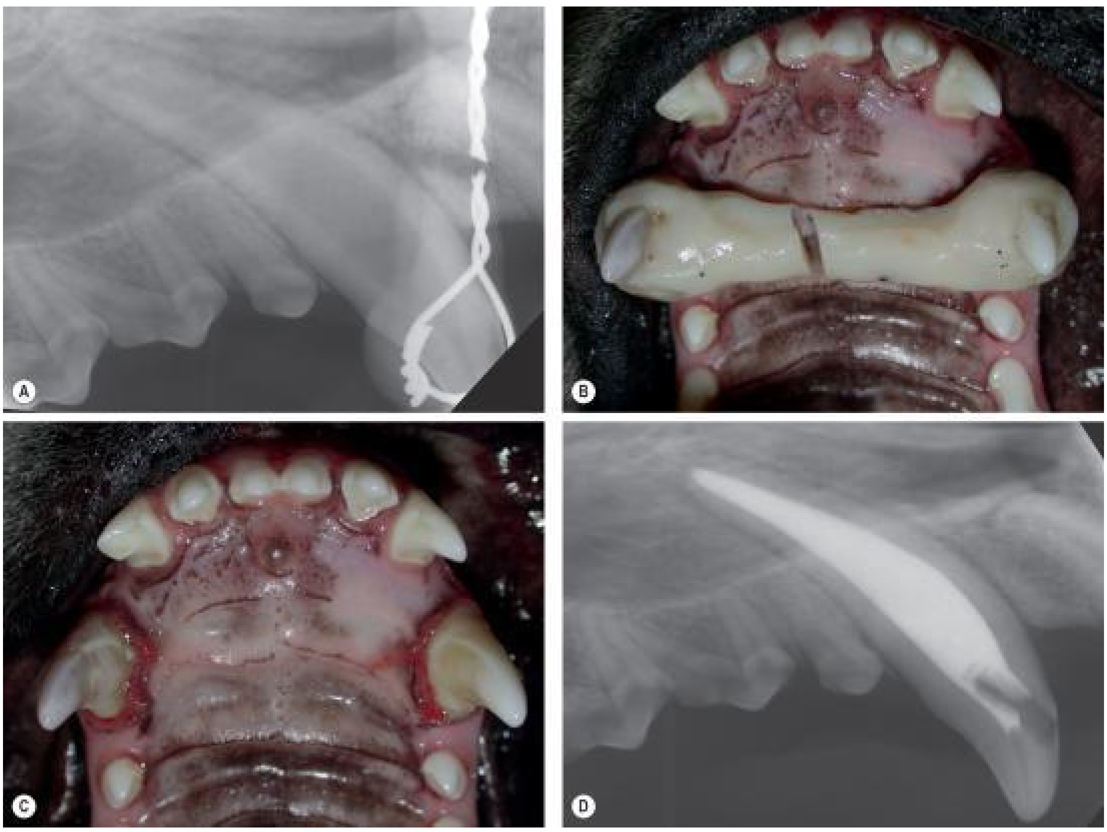
- Acid-etching of the crowns of the teeth to be included in the splint with phosphoric acid etching gel or liquid for 15 seconds. The dental surface should then be rinsed for at least 15 seconds using the three-way syringe to remove the acid-etching material, while protecting the patient's soft tissues. Acid-etching can be avoided if the shape of the teeth included in the splint assures good mechanical retention for the splinting material.
- Applying a bonding material on the teeth and the wires, unless not recommended for the material to be used (e.g., Protemp Garant). Bonding can be avoided if the shape of the teeth included in the splint assures good mechanical retention for the splinting material.
- Applying composite material or acrylic resin to secure the wire (Fig. 24.7D). To easily distinguish the resin from the dental structure at the time of splint removal, a shade distinctly different from the color of the teeth should be used, if available. To avoid inflammation, contact between the acrylic or composite material and the gingiva should be minimized by placing a spacer (e.g., plastic foil) during its application. Also, the splinting material should not extend beyond the mucogingival junction, to avoid mechanical trauma to the alveolar mucosa. Polymerization of light-cured composite is started from the noninjured teeth and finished with the displaced teeth, which can be held in position by digital pressure. After polymerization, the distal ends of the wire may be smoothed with burs.
- Obtaining a radiograph to verify the final position (Fig. 24.7E).
- Trimming and smoothing the splint (Fig. 24.7F).
- Inspecting the occlusion before allowing the patient to recover.



• **FIG. 24.7** (**A**) Lateral luxation of the right maxillary canine tooth in a 14-month-old dog following a fight with another dog. (**B**) Intraoral radiograph showing an increased distance between the tooth root and the alveolar walls, and a small bony fragment attached to the distal surface of the tooth *(arrow)*. (**C**) Wire-splinting of the displaced tooth. (**D**) Composite resin was added to cover the wire to avoid tongue laceration and strengthen the fixation device. (**E**) Intraoral radiograph following replantation showing correct tooth repositioning. (**F**) The composite resin was smoothed.

It is almost impossible to stop dogs from using the splinted teeth, but biting may be kept to a minimum by feeding a soft diet and removing access to chew toys. Proper oral hygiene should be instituted with daily brushing using a soft toothbrush and a 0.1% chlorhexidine gel, or with the use of a water flosser (Waterpik; Water Pik, Inc., Fort Collins, CO). Clinical reexamination of all replanted teeth should be scheduled 1–2 weeks after treatment to check the stability and integrity of the splint, the extent of gingival inflammation, or any undesired orthodontic movements.

The duration and retention of the splint depends on the age of the animal and the extent of the dental and bony lesions. In human patients, the splint is removed in 7–10 days, as prolonged splinting may induce replacement resorption and increase the frequency of pulp necrosis and inflammatory resorption.[1,55,85] If no bony fractures are present, this time is sufficient to secure adequate periodontal healing. The splint should be left in place longer, up to 4–6 weeks, in cases with extensive bony fractures.[1] After radiographically confirming bone and periodontal healing (Fig. 24.8A), the splint is removed by sectioning the splinting material with the appropriate burs on a high-speed handpiece and abundant water-cooling. Particular care should be taken not to damage the tooth surface. If possible, the splint should be initially divided in such a way that tooth mobility can be manually assessed (Fig. 24.8B). If significant mobility is still present, the splint should be repaired and left in place for a longer period. Otherwise, orthodontic bracket removal pliers are used to gently break off the material. After splint removal, the teeth should be polished.



• **FIG. 24.8** The patient in Fig. 24.7 at 4-week follow-up. (**A**) Intraoral radiograph (after dividing the splint) showing periodontal healing. (**B**) Before removal, the splint was divided and tooth mobility was evaluated manually. Note the pink discoloration of the right canine tooth, indicating pulp damage. (**C**) As the tooth was stable, the splint was removed. Mild gingivitis developed underneath the splint but resolved within a few days. (**D**) Intraoral radiograph showing root canal treatment before placement of the final restoration.

Systemic antibiotic treatment

The IADT guidelines recommend the systemic administration of antibiotics after replantation to prevent infection.[34,37] However, although experimental animal studies have shown positive effects upon periodontal and pulp healing,[86] this recommendation is currently under debate, as systematic reviews of the literature have failed to demonstrate the value of systemic antibiotic treatment.[87-89]

If started preoperatively or immediately post-replantation, systemic antibiotic therapy prevents bacterial invasion of the pulp and inflammatory resorption. If, however, antibiotic therapy is instituted some time after replantation, there is no reduction in the incidence of inflammatory root resorption.[41,78] When administered immediately postreplantation, both tetracycline hydrochloride (20 mg/kg PO TID for 7 days) and amoxicillin (22 mg/kg PO BID for 7 days) have been shown to be effective in limiting inflammatory root resorption secondary to pulpal infection in dogs.[86]

Endodontic treatment

As a result of dental displacement, the vascular supply to the pulp is often partially or totally severed, leading to degenerative changes in the pulp tissues. Therefore, standard root canal treatment is generally necessary following repositioning or replantation.[1] Endodontic treatment should be performed when the tooth is in its alveolus.[1] Extraoral filing before replantation always results in ankylosis, due to the fact that root canal treatment prolongs the extraoral period. Furthermore, pressure and damage to the periodontal ligament caused by incorrect handling of the teeth may induce further replacement resorption.[59,85] Only if more than 1 hour has elapsed before replantation and the avulsed tooth has been stored dry during this time can endodontic therapy be performed extraorally.[8,46] The tooth should be washed thoroughly and held by the crown in moist, sterile gauze.[19]

Even though it has been shown that, under experimental conditions, delayed endodontic treatment performed up to 40 days after immediate autogenic transplantation may be effective in minimizing inflammatory root resorption in dogs,[90] generally, endodontic treatment is recommended 7–14 days after replantation, at the time of splint removal.[1] To minimize trauma to the newly formed periodontal ligament fibers, endodontic treatment may be performed before physically removing the dental splint by creating an access hole through the splinting material. Initially, calcium hydroxide is used as filling material.[1] Calcium hydroxide is bactericidal and is apparently able to inhibit root resorption, probably by creating an alkaline environment that stimulates tissue repair while reducing the resorptive process.[91] This treatment, repeated every 3–6 months as the calcium hydroxide dissipates, is followed by permanent filling with gutta-percha approximately 1 year after replantation. In dogs and cats, because of the need for anesthesia for each procedure, short-term treatment with calcium hydroxide or single-stage root canal therapy with gutta-percha at the time of splint removal may be an acceptable treatment option (Fig. 24.8D).

Promising results on the use of a corticosteroid (1% triamcinolone) and antibiotic (3% demeclocycline) containing paste (Ledermix, Lederle Pharmaceuticals, Wolfratshausen, Germany) as a filling material have been shown in a study using a canine dental trauma model.[65] It was demonstrated that intracanal placement of the tested paste immediately following an avulsion injury and before replantation decreases tooth resorption and favors healing.[65] Also, the use of a 1% triamcinolone cream as filling material was shown to be as effective at inhibiting external root resorption as the triamcinolone-demeclocycline paste.[92] These researchers suggested that intracanal placement of corticosteroids should be used as standard treatment protocol at emergency visits for traumatic injuries in which root resorption is predicted. Another canine study using clobetasol or fluocinonide showed similar results.[93] On the other hand, the use of the nonsteroidal antiinflammatory indomethacin as intracanal dressing in avulsed teeth dried for 50 minutes did not show effective inhibition of tooth resorption.[70]

Endodontically treated teeth should be restored using glass-ionomer restoratives (temporary restoration following temporary endodontic treatment) or composite resins (permanent restoration).[36] In both instances, a >4-mm-deep restoration is recommended to minimize the risk of coronal leakage and bacterial contamination.[36]

If the trauma is minimal, such as in a case of mild concussion or subluxation, the pulp may survive, and the tooth should be monitored radiographically at 1, 3, 6, and 12 months to determine whether endodontic treatment is indicated.[1,2]

Finally, in case of immature teeth with an open apex, revascularization of the pulp tissues within 3–4 weeks is possible after immediate replantation.[79,81,94,95] Revascularization is accomplished by in-growth of new cell-rich and well-vascularized connective tissue.[96,97] In these patients, endodontic treatment is delayed and radiographic reexaminations are recommended every 2–4 weeks until continued root development is evident. In case of pulp necrosis or root resorption, temporary endodontic treatment with calcium hydroxide can be performed to achieve apexification before permanent obturation of the canal.

Complications following periodontal trauma and repositioning/replantation

A large amount of information is available, as numerous experimental animal studies have been performed to evaluate the treatment outcome and development of complications following periodontal trauma and repositioning/replantation. These studies, primarily on dogs, have shown that healing of the periodontal ligament starts soon after clot formation within the periodontal space. After replantation, new junctional epithelium is reestablished within 7 days.[98,99] At 2 weeks, new collagen fibers extend from the cementum to the alveolar bone, and periodontal healing is complete in 2–4 weeks.[100] However, complications following dental displacement are common, more so in mature than immature teeth.[31]

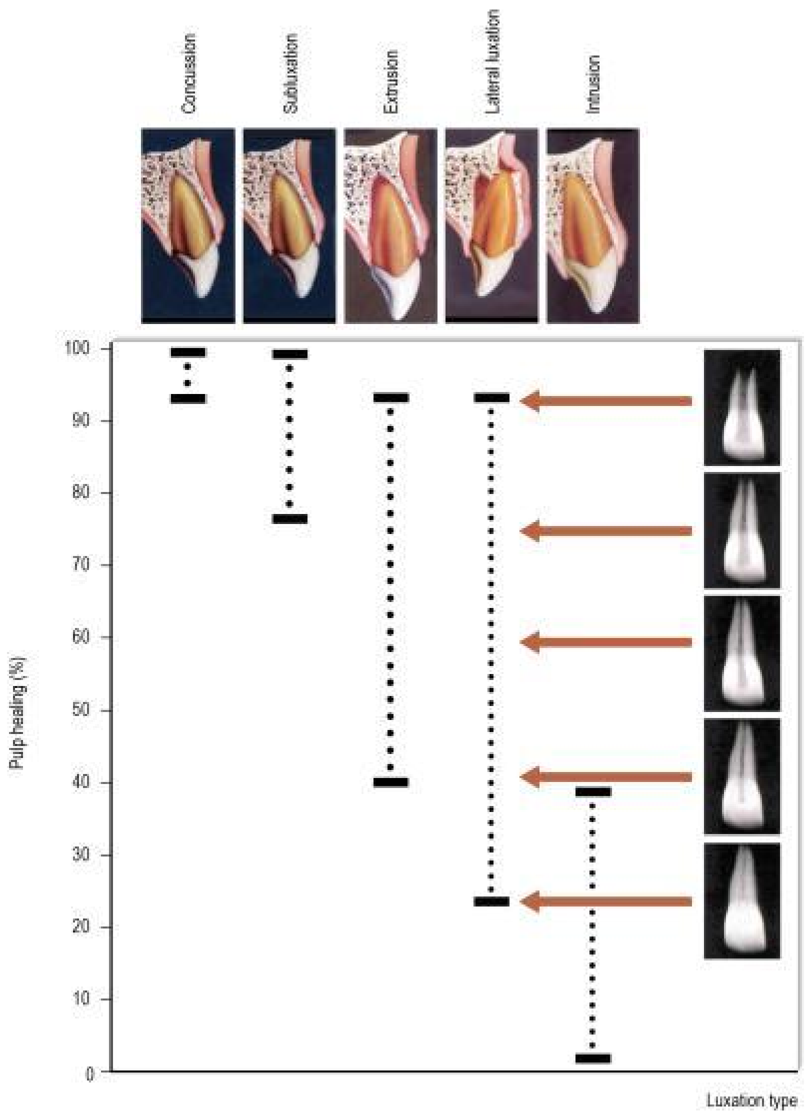
Pulpal healing complications

Coronal discoloration

Discoloration of the crown soon after injury is often a sign of damage to the pulp (Figs. 24.2 and 24.8C). The discoloration may be transient,[101] but more commonly, it is a sign of irreversible pulpitis and pulp necrosis.

Pulp necrosis

The most common complication of severe dental displacement is pulp necrosis.[1,31,85] The necrotic pulp is also often infected as a result of contact with saliva, plaque, or extraoral material through the apex. Whether pulp necrosis will occur following trauma depends on the type of injury and stage of root development (Fig. 24.9).[101] Risk of pulp necrosis is least after concussion and subluxation, and greatest in extrusion, lateral luxation, and intrusion, in that order.[101] Pulp necrosis is more common in teeth with complete rather than incomplete root development.[101]



• **FIG. 24.9** Relationship between luxation diagnosis, stage of root development, and pulpal healing after trauma in human beings. Each bar represents the level of pulp survival 1 year after injury. The upper set of bars represents pulp survival for teeth with incomplete root formation, and the lower set, pulp survival for teeth with complete root formation. Source: (From Andreasen JO, Andreasen FM, Andersson L, eds. *Textbook and Color Atlas of Traumatic Injuries to the Teeth*. 4th ed. Oxford: Blackwell Munksgaard, 2007. With permission.)

Pulp necrosis following dental displacement is diagnosed in humans based on the presence of loss of pulpal sensitivity, crown discoloration, or periapical radiolucency and confirmed by pulp vitality testing.[101] As vitality tests are unreliable in dogs and cats, the diagnosis of pulp necrosis in these patients should be based primarily on radiographic and clinical signs (i.e., coronal discoloration),[6] and endodontic treatment is recommended.

Root canal obliteration

Root canal obliteration should be considered a normal reaction of vital pulp to a moderate trauma. In humans, it is more common following displacement of teeth with incomplete rather than complete root formation, in particular after luxation injuries.[83,85] It is rarely seen after concussion and subluxation, when little damage to the pulp occurs.[101]

Root canal obliteration can usually be diagnosed within the first year after injury.[101] In approximately 1%–16% of human traumatized teeth, it is followed by pulpal necrosis.[1]

Root resorption

Root resorption is a serious complication associated with repositioning and replantation and represents the major cause of failure following treatment of displaced teeth. In humans, it occurs more frequently in avulsed than in luxated teeth.[1] Root resorption progresses more rapidly in younger than older replanted teeth and is negatively influenced by the presence of concomitant fractures of the alveolar bone or by an extended period of splinting.

Root resorption is directly related to an injury to the periodontal tissues. The presence of a vital periodontal ligament has been shown to inhibit invasion of bone cells. In particular, the intermediate cementum is thought to resist root resorption and bone invasion, and its loss as a result of trauma may predispose to resorption.[102] The intermediate cementum cannot be regenerated once it has been damaged; therefore, successful replantation is directly related to the preservation of the periodontal ligament and superficial layer of the root.[41] External root resorption is classified into three types: (1) surface resorption, (2) inflammatory resorption, and (3) replacement resorption.[1] The same tooth may develop a combination of surface resorption, followed by cemental healing and osseous replacement.[80] Internal resorption occurs in only 4% of displaced teeth, mainly after luxation injuries.[31]

External surface resorption

Surface resorption is characterized by small, localized resorption cavities affecting the cementum and the superficial layer of dentin.[1] It is apparently caused by drying of the periodontal ligament and cementum during the extraalveolar time and by pressure directed onto the root surfaces.[83] Surface resorption can be recognized within 1 week after replantation, becomes more prominent at 2 weeks, and may heal spontaneously with reparative cementum within a few weeks.[41] It is usually asymptomatic and cannot be visualized on routine radiographs.[2] When dentin is resorbed, healing may occur with an altered root outline that may become radiographically visible.[103]

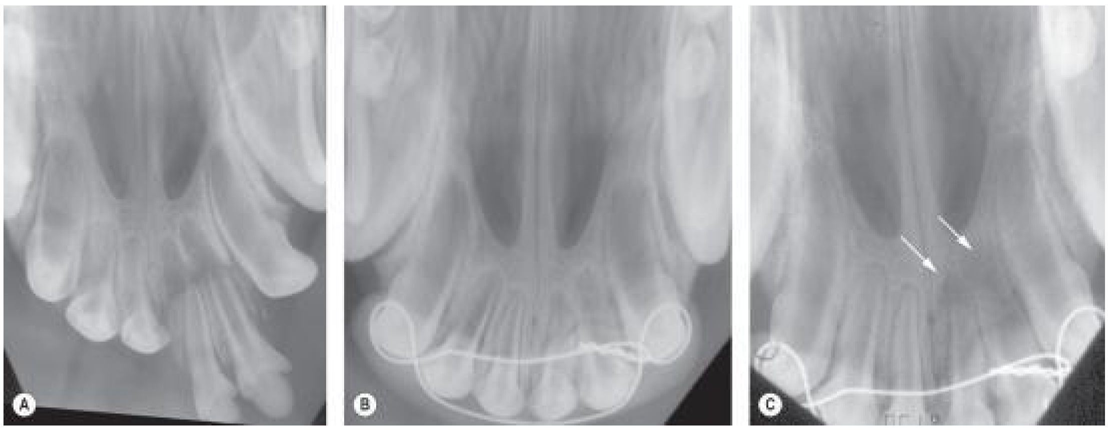
External inflammatory resorption

The development of inflammatory root resorption is directly related to damage of the root surface and periodontium at the time of the traumatic insult and the presence of bacteria within the root canal and dentinal tubules.[1] Under normal circumstances, if the pulp becomes infected, the cemental layer does not allow the toxins from the pulp to reach the periodontal space. After an avulsion, cementum may be damaged and toxins from the necrotic and infected pulp may pass through the dentinal tubules and stimulate an inflammatory response in the corresponding periodontal ligament space. Root substance and bone are then destroyed.

This process is accelerated in the young patient and following an extended extraalveolar period due to increased chances of infection.[1] If inflammatory resorption is not resolved, rapid destruction of the root, progressive tooth mobility, and eventually, tooth loss may occur.[98]

Consequently, control of root canal infection with adequate root canal therapy after replantation is critical for the prevention and/or treatment of inflammatory root resorption. If endodontic treatment is delayed, it is difficult to eliminate resorption once it begins.[2] If the inflammation subsides, ankylosis often results.

Inflammatory resorption consists radiographically of bowl-shaped resorption cavities of the root and radiolucent areas of the adjacent bone.[3] These progressive changes can be seen as early as 2–3 weeks after avulsion (Fig. 24.10).[83,98]



• **FIG. 24.10** (**A**) Radiograph of a 5-month-old beagle with lateral luxation of the left maxillary first and second incisor teeth following playing activity with another dog. (**B**) Radiograph showing correct positioning after replantation and splinting. (**C**) Three-week follow-up radiograph showing lack of root development, apical root resorption, and periapical radiolucency of the replanted teeth *(arrows)*.

External replacement resorption and ankylosis

In external replacement resorption, a large amount of root hard tissue is resorbed and filled with alveolar bone, causing the tooth to be anchored tightly in its alveolus. The clinical signs include reduced motility and a metallic, high percussion sound. Human teeth with less than 10% of ankylosed root surface, though, exhibit normal percussion sound and mobility, and a high-pitched percussion tone has been shown to develop only when at least 20% of the root surface is ankylosed.[40] Radiographically, the distinction between the root and surrounding bone is lost, the periodontal ligament space is absent, and a moth-eaten appearance results.[2,3]

However, visualization is not always possible because of overlapping structures and bone marrow spaces.[40,83] Radiographic signs usually appear after a few weeks from replantation, when the resorption cavities have increased to more than 1–2 mm in diameter.[83]

As long as the protective covering of cementum is intact and periodontal ligament cells are healthy, resorption will not occur.[54] Factors such as extraoral time, use of a suitable storage medium, and the type and duration of splinting are critical for the prevention of replacement resorption.[44] When the periodontal ligament and cementoblasts are damaged, the root is exposed to colonization by osteoclasts, cells of the same hematological origin as osteoclasts that migrate from the adjacent bone marrow into the damaged areas.[1,2]

The ultimate outcome of replacement resorption is destruction of the root, which leads to either infection via the gingival sulcus, crown fracture due to loss of support, or exfoliation of the tooth.[2,40]

The use of agents inhibiting osteoclastic activity has been studied in dogs to evaluate the possibility of decreasing root resorption following displacement and replacement.[64] An enamel matrix derivative (Emdogain; Biora, Malmö, Sweden) has been shown to exhibit a positive effect in regenerating cementum and periodontal ligament fibers in dogs' exarticulated teeth.[60] However, a subsequent study showed inability of the same product to protect the root of reimplanted canine teeth from replacement resorption.[57]

The topical use of recombinant human bone morphogenetic protein-12 (rhBMP-12), a member of the transforming growth factor-b/c gene family, failed to reestablish normal periodontal ligament fibers and to prevent ankylosis and root resorption of dogs' reimplanted teeth.[76]

Fluoride is considered to promote osteoblastic activity or to inhibit osteoclastic activity by reducing the susceptibility of bone and dental hard tissues to resorptive processes.[41]

Loss of marginal bone

Loss of marginal bone support following dental displacement may develop because of mechanical irritation by the splint, plaque accumulation, or directly because of the trauma.[83,85] It increases with the severity of the injury, being very common in cases of intrusion and extrusion.[1,2]

Prognosis

The prognosis of traumatized teeth is generally better in younger patients and in those having intact soft tissues, no root fractures, and maximal bone support.[8] The presence of an intact, viable periodontal ligament is the most important factor in assuring healing without resorption, the critical extra-alveolar time being 60 minutes.[1,37] Healing is also dependent upon the extent of the injury. Severely displaced teeth show a higher incidence of healing complications than subluxated teeth do.[31] Intrusive displacement is second only to avulsion in severity, as root resorption, ankylosis, and pulp necrosis frequently occur.[1] Duration of immobilization has also been shown to influence the prognosis of luxation injuries: the longer the splinting time, the higher the incidence of complications.[85]

It is important to consider that teeth affected by resorption and ankylosis may exfoliate or fracture months or years after the initial injury. Because of the possible development of late complications, radiographic follow-up examinations are recommended in humans for 2–5 years post-replantation.[1,19,36] Further studies are needed to evaluate the incidence and rate of development of late complications in veterinary patients.

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SECTION 5:

Endodontic Surgery

OUTLINE

CHAPTER 25

Principles of endodontic surgery

Santiago Peralta, Edward R. Eisner

Definitions[1-4]

Apicoectomy: Root-end resection of an endodontically treated tooth.

Draining tract: A tract originating from a focus of suppuration, such as a periapical abscess, and discharging pus. Most tracts are lined by chronic inflammatory cells. In long-standing cases, the lining may epithelialize, in which case the term "fistula" may be used.

Endodontic surgery: Endodontic procedure to prevent or treat apical periodontitis by surgical means.

Hemisection: Removing half of the crown (i.e., mesial or distal half) and associated root(s) of a multirooted tooth, combined with appropriate endodontic treatment of the remaining tooth structures.

Partial pulpectomy: Type of endodontic procedure that aims to maintain the vitality of the tooth pulp.

Parulis: A sessile, hyperplastic nodule on the gingiva or at the mucogingival junction at the site where a draining tract of endodontal origin reaches the surface.

Root amputation: Removal of a root of a multirooted tooth, leaving most of the crown intact, and combined with appropriate endodontic treatment of the remaining dental structures.

Nonsurgical root canal treatment: Normograde access to the pulp cavity, followed by chemical and mechanical disinfection, obturation, and restoration of an endodontally diseased tooth; also referred to as standard or conventional root canal treatment.

Root canal treatment, surgical: Surgical approach to the apex of a previously endodontically treated tooth, with subsequent root-end resection (apicoectomy), periapical debridement, and root-end filling.

Introduction

Although nonsurgical root canal treatment is generally sufficient in cases of endodontal disease, certain situations warrant a surgical approach. A surgical root canal treatment is a retrograde procedure that involves elevating a mucoperiosteal flap, removing bone to expose the affected apex, and preparing and filling the apical end of the root canal in a retrograde fashion.[5] Surgical and nonsurgical root canal treatments are complementary and are usually performed by an adequately trained clinician who has access to specialized equipment, instrumentation, and materials.[5-9]

The most common indication for performing a surgical root canal treatment is a failed nonsurgical root canal treatment.[5,9-12] Although the success rate of nonsurgical root canal treatment in dogs and cats is high, a failure rate of up to 6% and 19% has been reported, respectively.[13,14] Other indications for performing a surgical root canal treatment include anatomical barriers (i.e., aberrant anatomy and pulp stones) or irretrievable material (e.g., separated endodontic instruments) that prevent complete normograde debridement and obturation, transverse fracture of the apical third of the root with pulp necrosis, advanced external inflammatory tooth resorption of the apical third of the root secondary to chronic apical periodontitis, and persistent periapical inflammation following nonsurgical root canal treatment.[5,9-11]

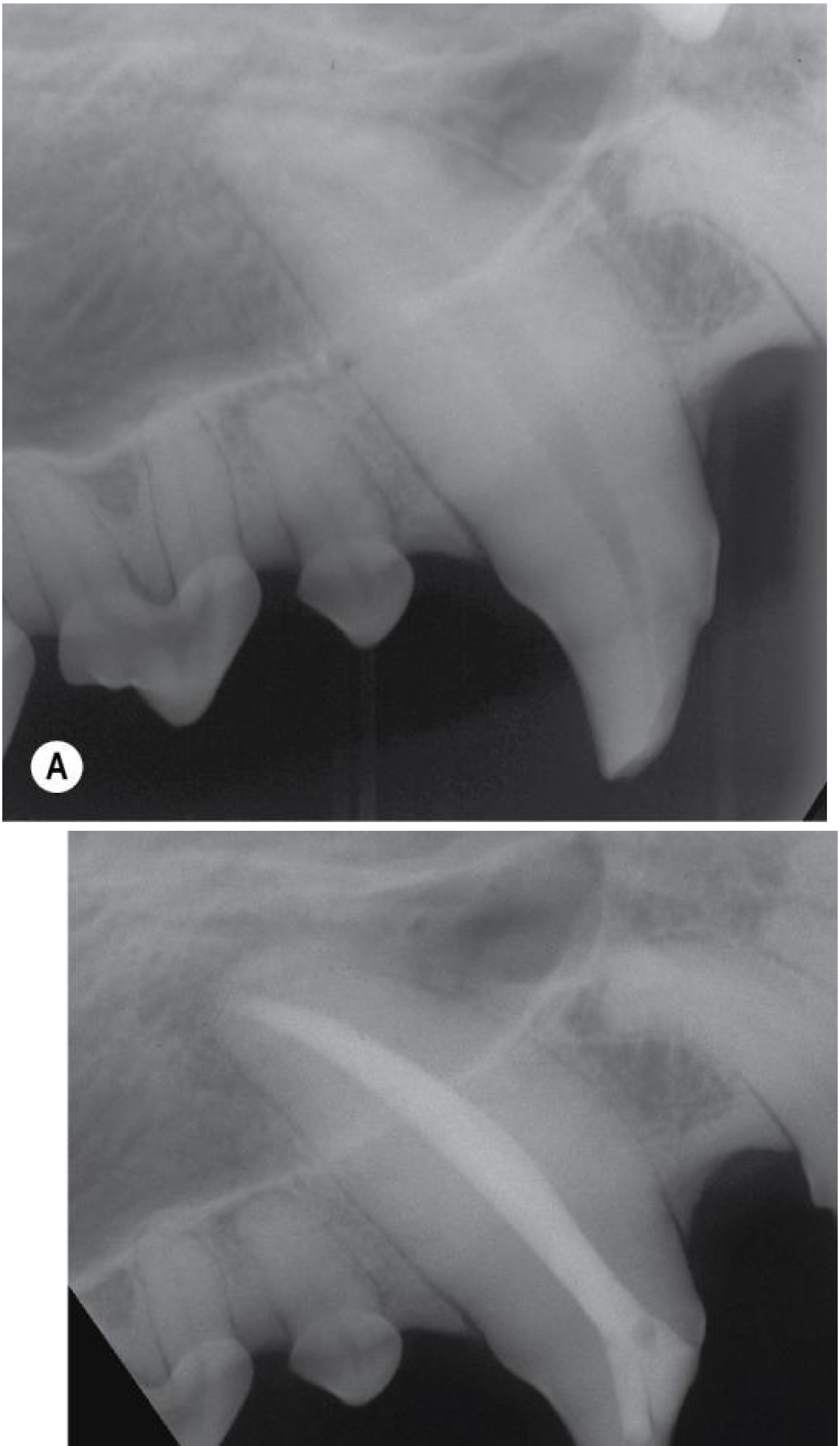
Due to their size and functionality, the teeth most often treated by means of a surgical root canal treatment are the maxillary and mandibular canine teeth, the maxillary fourth premolar tooth, and the mandibular first molar tooth in the dog and the maxillary and mandibular canine teeth in the cat.[5,10,12,15-17] Similar to nonsurgical root canal treatment, the success rate of surgical root canal treatment in dogs appears to be high. In a retrospective case series in 15 dogs, no failed cases were observed after an average of 15.2 months of follow up after surgical root canal treatment was performed.[18] However, the outcome of surgical root canal treatment in cats has not yet been reported in the scientific literature.

Other surgical techniques that involve endodontic intervention are hemisection and root amputation, in which parts of the crown and corresponding root(s) are surgically removed, while the rest of the tooth is preserved. These procedures invariably expose the pulp cavity and dictate appropriate endodontic intervention. They are indicated when part of a tooth cannot be endodontically and/or periodontally treated but preserving the remainder is clinically beneficial.

Therapeutic decision-making

Failed nonsurgical root canal treatment

The treatment options for failed nonsurgical root canal treatment are extraction, nonsurgical retreatment, or surgical root canal treatment.[16] Failure of a nonsurgical root canal treatment is determined based on clinical and radiographic criteria.[13,14] Radiographically, failure is determined if a periapical lucency appears larger at the time of follow up compared to radiographs obtained at the time of initial treatment. A novel periapical lucency that was not present at the time of initial treatment is also a radiographic indicator of root canal failure (Fig. 25.1).


A
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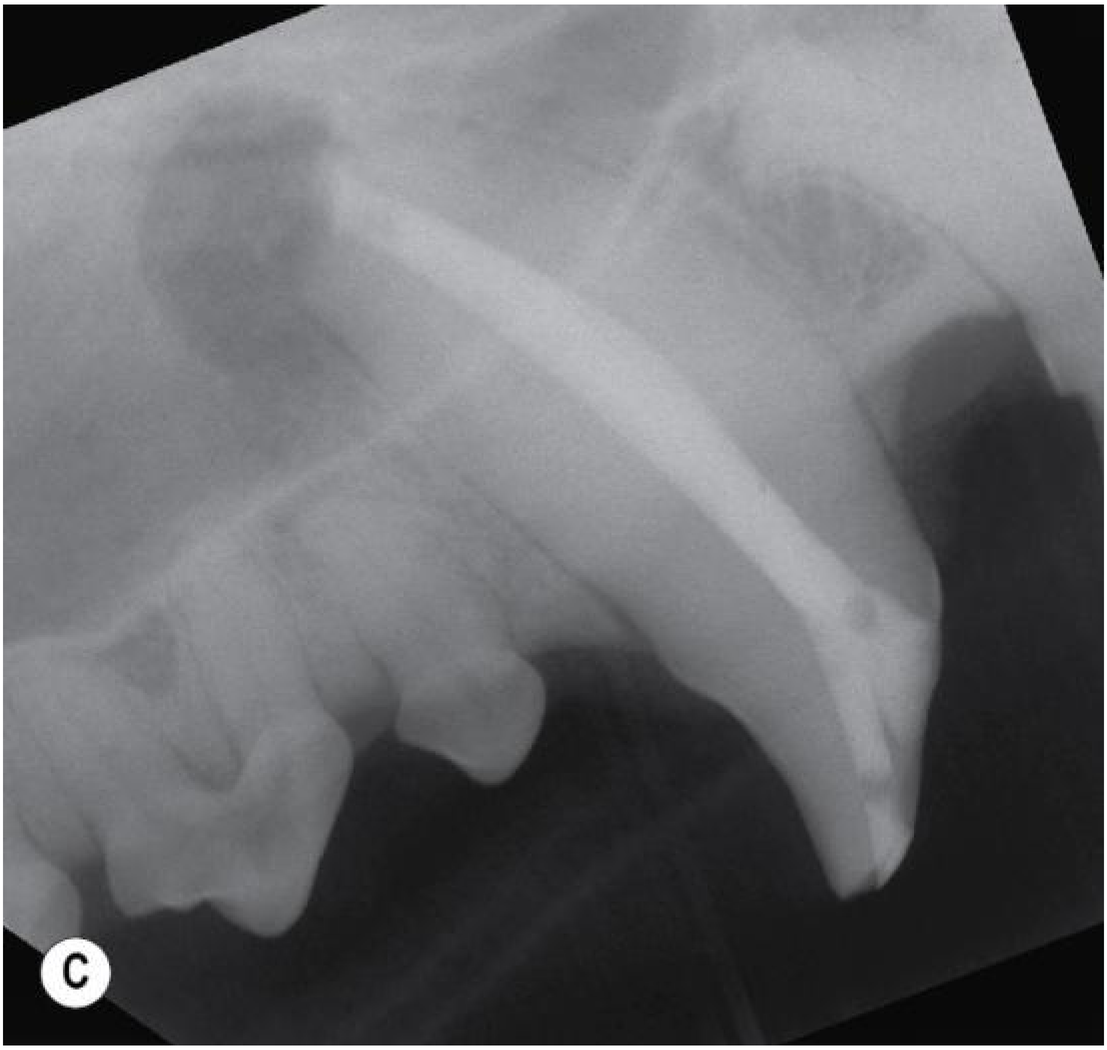
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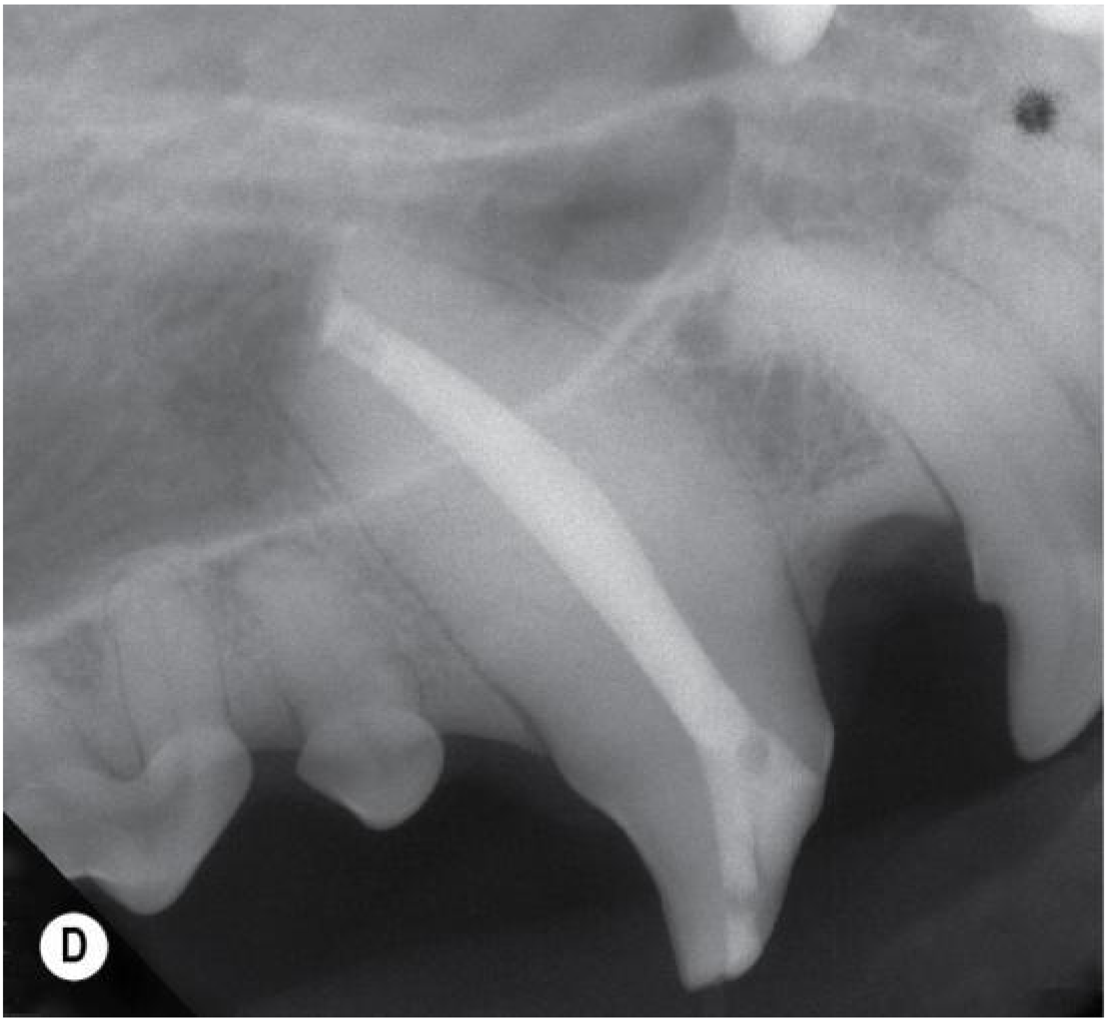
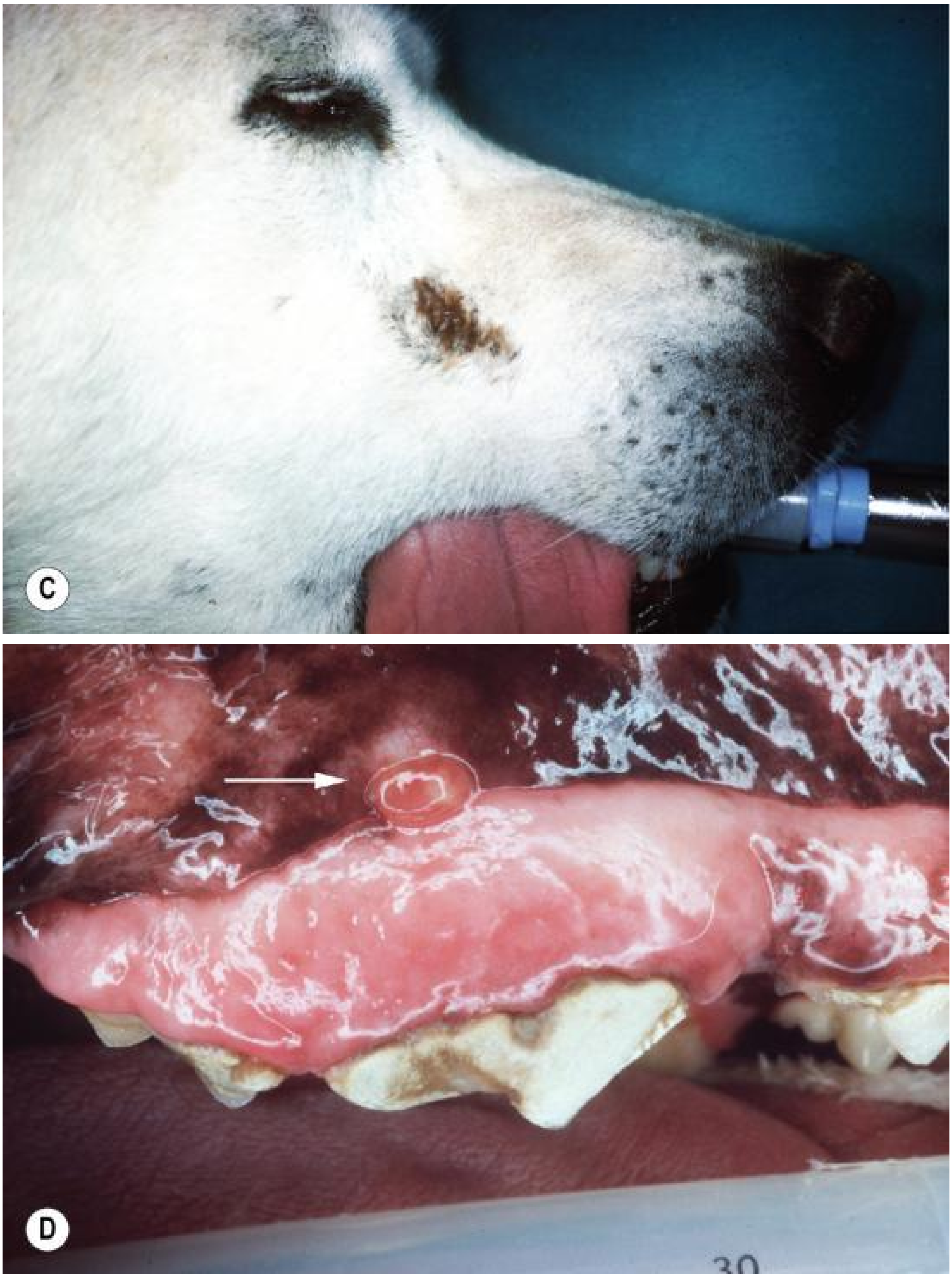


• **FIG. 25.1** Failed nonsurgical root canal treatment in a dog successfully treated by surgical means: (**A**) Lateral radiograph of a fractured right maxillary canine tooth in an adult dog immediately before nonsurgical root canal treatment. No periapical lucency was present radiographically but the pulp was clinically confirmed to be nonvital. (**B**) Follow-up radiograph 6 months after a nonsurgical root canal treatment was performed. Note a novel round, well-defined periapical lucency, consistent with apical periodontitis, indicative of a failed nonsurgical root canal treatment. (**C**) Radiograph of the same tooth immediately after surgical root canal treatment showing the extent of the apicoectomy performed and the root-end filling placed. (**D**) Follow-up 6-month radiograph showing the area of apicoectomy replaced by bone, indicative of a successful surgical root canal treatment.

Clinically, novel, persistent, or worsening signs of odontogenic infection associated with a previously endodontically treated tooth are indicators of failure. The most common clinical signs associated with an odontogenic infection are localized swelling and/or an intra- or extraoral draining tract.[6,10] The anatomical location of these lesions depends on the tooth of origin (Fig. 25.2).


A
B



• FIG. 25.2 Swelling or draining tract indicates the location of the involved root. (**A**) Parulis *(arrow)* at the exit of a draining tract from a periapical abscess of the maxillary canine tooth. (**B**) Cutaneous draining tract originating from the mandibular canine tooth. (**C**) Cutaneous draining tract originating from the mesiobuccal root of the maxillary fourth premolar tooth. (**D**) Parulis *(arrow)* and draining tract originating from the distal root of the maxillary fourth premolar tooth. Source: *(Fig. 25.2C from Verstraete FJM, Tsugawa AJ. Self-Assessment Color Review of Veterinary Dentistry. 2nd ed. Boca Raton: CRC Press - Taylor & Francis Group, 2016.)*

Contrary to extraction, nonsurgical root canal retreatment preserves the tooth and represents an acceptable treatment option in cases of failed nonsurgical root canal treatment. On the other hand, nonsurgical retreatment requires thorough removal of the previously placed restoration and obturation materials, which can be technically challenging and may represent an unacceptably prolonged anesthetic event. In contrast, a surgical root canal treatment is performed without removing the previously placed materials and can usually be completed in a relatively short time.[12] Additionally, a surgical approach allows the inflammatory tissue to be removed, the area debrided, and if indicated, submitted for histopathological analysis.[9] Therefore, surgical root canal treatment should be performed in cases of failed nonsurgical root canal treatment in patients in whom a prolonged anesthesia is contraindicated or if there is concern that the periapical lesion is not inflammatory in origin (i.e., neoplastic).[9,12] As well, failed nonsurgical root canal treatments that do not resolve after nonsurgical retreatment should be either treated surgically or extracted.

Finally, a nonsurgical root canal retreatment may be impractical or impossible if a prosthetic crown is present.[9,19] Removing a prosthetic crown is difficult and can often not be achieved without destroying the prosthesis. Therefore, if a periapical lesion develops on a tooth that was successfully restored using a prosthetic crown, it is justifiable to resort to surgical root canal treatment rather than to perform nonsurgical root canal retreatment.

Hemisection and root amputation

Occasionally, hemisection is an appropriate treatment in a multirooted tooth in which advanced attachment loss (i.e., periodontitis) does not involve all roots, but threatens the tooth with impending ascending endodontal infection. A viable therapeutic option is to remove only the periodontally affected root(s) and overlying crown, so that effective endodontic treatment may be performed on the periodontally sound half of the tooth that remains.[4] This may be recommended if the affected tooth is functionally important (i.e., maxillary fourth premolar and mandibular first molar teeth) or if it is associated with a jaw fracture, and preserving part of the tooth is important for reduction and fixation purposes. At the time of sectioning, if the exposed pulp in the chamber appears healthy, partial pulpectomy may be performed to allow proper restoration while maintaining the vitality of the rest of the pulp. On the other hand, if the pulp appears nonviable, nonsurgical root canal treatment is indicated for the remaining root(s). Nonsurgical root canal treatment may also precede hemisection or root amputation if there is evidence of endodontal disease prior to the procedure.

Root amputation is applicable to cases in which nonsurgical root canal treatment of the maxillary fourth premolar tooth has failed in dogs and surgical root canal treatment is intended. In such cases, the mesiopalatal root and corresponding cusp are removed because the apex is almost impossible to approach without invading other important structures (i.e., nasal cavity and maxillary recess), and the mesiobuccal and distal roots and corresponding cusps are preserved for surgical endodontic purposes. The resultant exposed pulp cavity at the site of amputation should be prepared and restored appropriately. It is generally accepted that amputating this root is a practical alternative, although there are concerns that this may weaken the tooth.[15]

Unresolved apical periodontitis following root canal treatment

The clinical and radiographic signs of apical periodontitis (i.e., pain, draining tract, and periapical lucency) may only partially resolve or may remain unchanged after nonsurgical root canal treatment.[9,12,18] Histologically, apical periodontitis may be due to a periapical granuloma, an abscess, or a cyst. The most common histological form of apical periodontitis is granuloma.[12]

When a granuloma becomes infected, signs of infection (i.e., swelling and intraoral or extraoral draining tracts) ensue. Untreated or unresolved apical periodontitis may become cystic if an epithelial lining forms around the lesion. This is usually the result of chronicity. Although any of the histological forms may fail to resolve after nonsurgical root canal treatment, cysts are the most likely to persist and require debridement and a surgical root canal treatment for resolution.[12] Persistent apical periodontitis may be accompanied by persistent clinical signs (i.e., drainage and pain).

Chronic apical periodontitis and external inflammatory tooth resorption

Chronic apical periodontitis, characterized by periapical bone loss and inflammation and a radiographically detectable periapical lucency, often results in external inflammatory resorption of the apical area of the tooth.[20] Unless the tooth is successfully endodontically treated before advanced tooth resorption has occurred, loss of structural integrity of the apex is a likely outcome. Loss of apical integrity may make adequate instrumentation, disinfection, and obturation difficult during nonsurgical root canal treatment. In such cases, apicoectomy immediately following nonsurgical root canal treatment may be warranted.

Anatomical problems preventing standard normograde root canal treatment

Preoperative intraoral radiographs occasionally reveal anatomical and/or developmental aberrations[21,22] that may make nonsurgical root canal treatment difficult.[9,12,16] These include dilacerated roots, pulp stones, and calcified canals, which may partially or completely obstruct the passage of even the smallest endodontic files from reaching the apex. Although nonsurgical root canal treatment should be attempted first, apicoectomy is indicated if debridement and obturation cannot be satisfactorily achieved.

Transverse fracture of the apical third of the root with pulp necrosis

It is possible for transverse fractures of the apical third of the root to heal and remain asymptomatic. However, if the pulp undergoes necrosis, apical periodontitis may ensue. In this case, either the tooth and fragment should be extracted or a nonsurgical root canal treatment should be performed followed by surgical removal of the apical fragment and retrograde filling.[11]

Endodontic instrument separation and procedural errors

Surgical root canal treatment is effective in salvaging the intraoperative complications of nonsurgical root canal treatment. The most common complications are a separated endodontic file that cannot be retrieved or bypassed in a way that allows thorough disinfection and obturation of the entire root canal, and iatrogenic perforation of the apex due to overinstrumentation.[16]

Similarly, if during attempted nonsurgical retreatment of a failed root canal treatment, the canal becomes obstructed by debris and materials from the initial procedure, and such material interferes with debridement and obturation, surgical root canal treatment may be indicated.

Contraindications for surgical root canal therapy

When retreatment is possible

If the cause of nonsurgical root canal treatment failure is due to poor instrumentation, disinfection, obturation, and/or restoration, nonsurgical retreatment to revise the technical deficiencies should be pursued instead of a surgical approach.

Medical concerns

If possible, surgical treatment should be avoided in patients with hemorrhagic disorders or a history of previous radiation therapy or patients in poor health that pose a significantly increased anesthetic risk that will be associated with the stress of an invasive procedure.[5,9] There are no specific contraindications for endodontic surgery that would not be similar to other types of oral surgical procedures.

Anatomical limitations

Treatment should be avoided if there is high risk of destruction or serious compromise to the mandibular canal, maxillary recess, middle mental foramen, or the major palatine blood vessels, as indicated by the location or extent of disease.[5,9,12] Treatment of short-rooted teeth has a higher likelihood of failure because an unfavorable crown:root ratio is more likely to result in compromised stability. Cases with extensive root damage that would result postoperatively in too short a root may result in subsequent exfoliation due to trauma or inflammatory disease.[5,10] In cases in which there is a fair chance of failure, the treatment of choice may be extraction.

Other dental considerations

Surgical root canal treatment is contraindicated in teeth with advanced periodontitis, in teeth with vertically fractured roots, in small nonessential teeth, and in teeth with advanced external or internal tooth resorption that compromises the integrity of the tooth.[19,23]

When periodontitis is the primary cause of endodontal disease (i.e., type 2 periodontal-endodontal lesions), both the periodontal and the endodontal disease will need to be treated to regain oral health. Cases like this have a higher likelihood of failure, especially in short-rooted teeth, where amputation of a portion of the root will significantly compromise the effectiveness of the root as an anchor for the tooth. In cases where periodontal disease is extensive, adequate debridement may also further weaken the mandible and significantly reduce the chances for successful treatment.[5]

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CHAPTER 26

Endodontic surgical techniques

Santiago Peralta, Edward R. Eisner

Definitions[1-4]

Accessory canal: A small canal communicating from the root canal to the periodontal space, usually in the apical one-third of the tooth root.

Alveolar jugum: The palpable convexity of the buccal alveolar bone overlying a large tooth root.

Apical delta: The diverging branches of the root canal at the apical end of the tooth root typically seen in carnivores.

Apicoectomy: Root-end resection of an endodontically treated tooth.

Draining tract: A tract originating from a focus of suppuration, such as a periapical abscess, and discharging pus. Most tracts are lined by chronic inflammatory cells. In long-standing cases, the lining may epithelialize, in which case the term *fistula* may be used.

Hemisection: Removing half of the crown (i.e., mesial or distal half) and associated root(s) of a multirooted tooth, combined with appropriate endodontic treatment of the remaining tooth structures.

Line angle: An imaginary vertical line forming the intersection of two adjacent vertical dental surfaces; they denote specific positions on a tooth and are important surgical landmarks.

Root amputation: Removal of a root of a multirooted tooth, leaving most of the crown intact, and combined with appropriate endodontic treatment of the remaining dental structures.

Surgical root canal treatment: Surgical approach to the apex of a previously endodontically treated tooth, with subsequent root-end resection (apicoectomy), periapical debridement, and root-end filling.

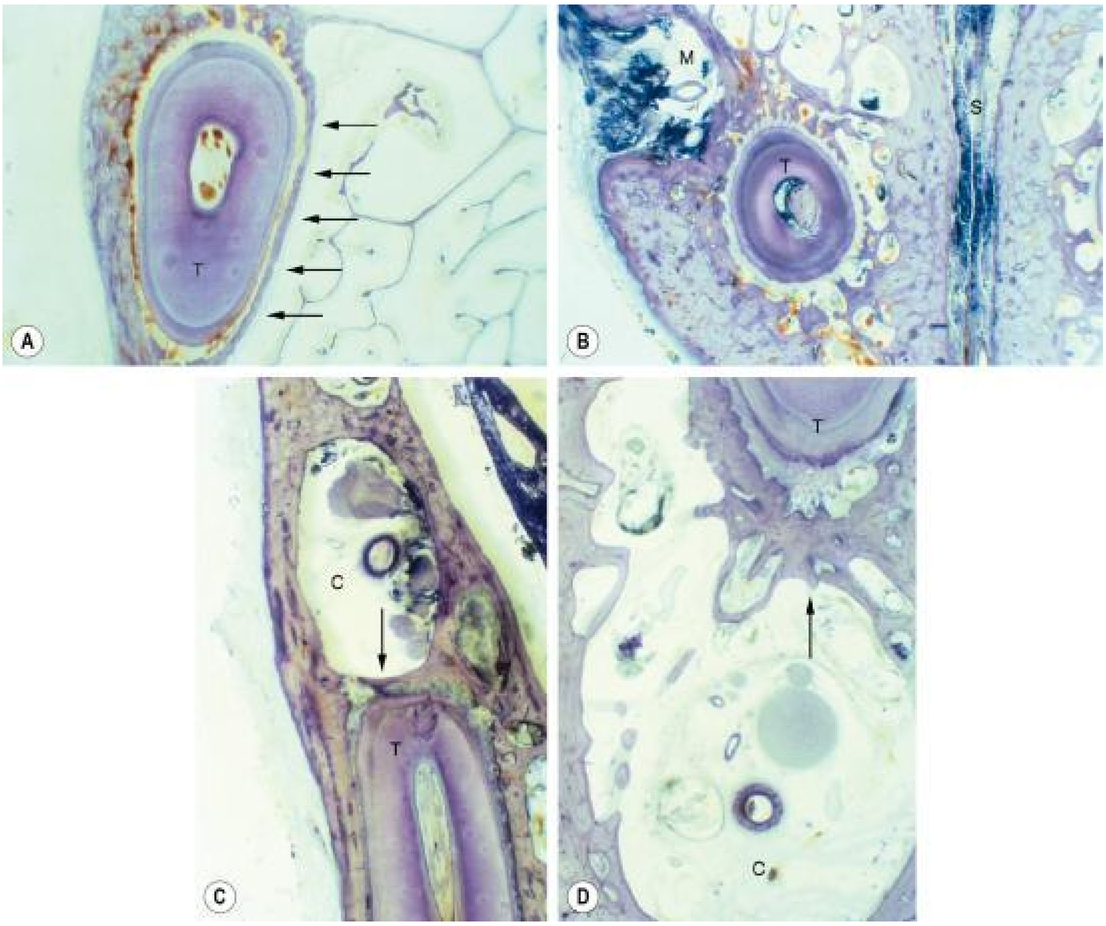
Preoperative concerns

Nonsurgical (conventional) root canal treatment

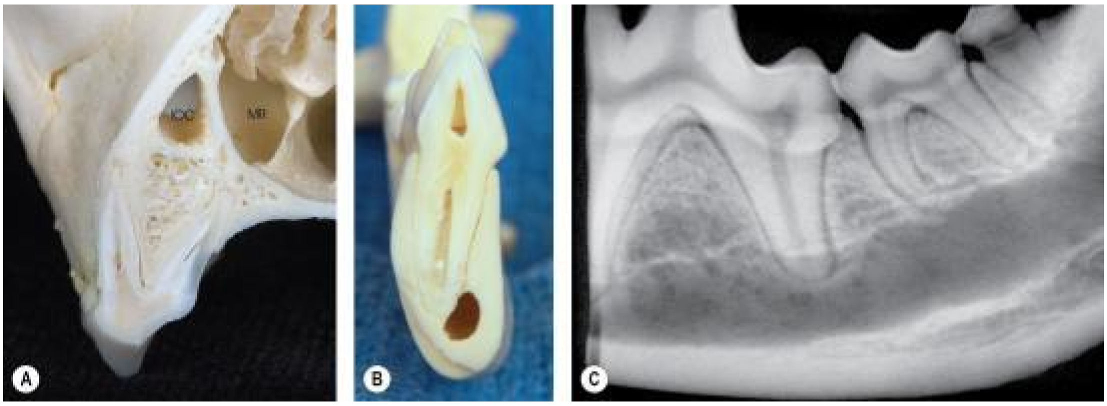
Surgical and nonsurgical root canal treatment are complementary procedures. Nonsurgical root canal treatment is usually performed as a first step.[1,2,4,5] Cases requiring surgical root canal treatment are technically complex and the crucial preoperative consideration is to identify the indications or contraindications (see Chapter 25).

Surgical anatomy and surgical planning

To perform an apicoectomy, the clinician must be familiar with the topographic anatomy of the tooth to be treated, and adjacent structures.[6] When present, swelling or a draining tract will help in identifying the location of the involved root apex and the site of the persistent area of inflammation (see Chapter 25).[7] Evaluation of dental radiographs will also help identify and locate the root apex and lesion. The teeth most frequently in need of surgical root canal therapy are the canine teeth, the maxillary fourth premolar, and the mandibular first molar teeth in dogs, and the canine teeth in cats. The apices of these teeth lie in close proximity to important structures, such as the nasal cavity, as well as the middle mental foramen, infraorbital and mandibular canals, and the corresponding nerves and arteries (Figs. 26.1 and 26.2).[8]



• **FIG. 26.1** Topographical anatomy illustrated by means of histological sections of root tips of a dog's teeth and their adjacent structures. (**A**) Root *(T)* of the maxillary canine tooth—note the thin layer of bone separating the alveolus from the nasal cavity *(arrows)*. (**B**) Root *(T)* of the mandibular canine tooth in close proximity to the middle mental foramen *(M)* and mandibular symphysis *(S)*. (**C**) Mesiobuccal root *(T)* of the maxillary fourth premolar tooth—note the thin layer of bone separating the alveolus from the infraorbital canal *(C) (arrow)*. (**D**) Mesial root *(T)* of the mandibular first molar tooth—note the thin layer of bone separating the alveolus from the mandibular canal *(C) (arrow)*. Source: (From Manfra Marretta S, Eurell JA, Klippert L. Development of a teaching model for surgical endodontic access sites in the dog. *J Vet Dent*. 1994;11:89–93.)

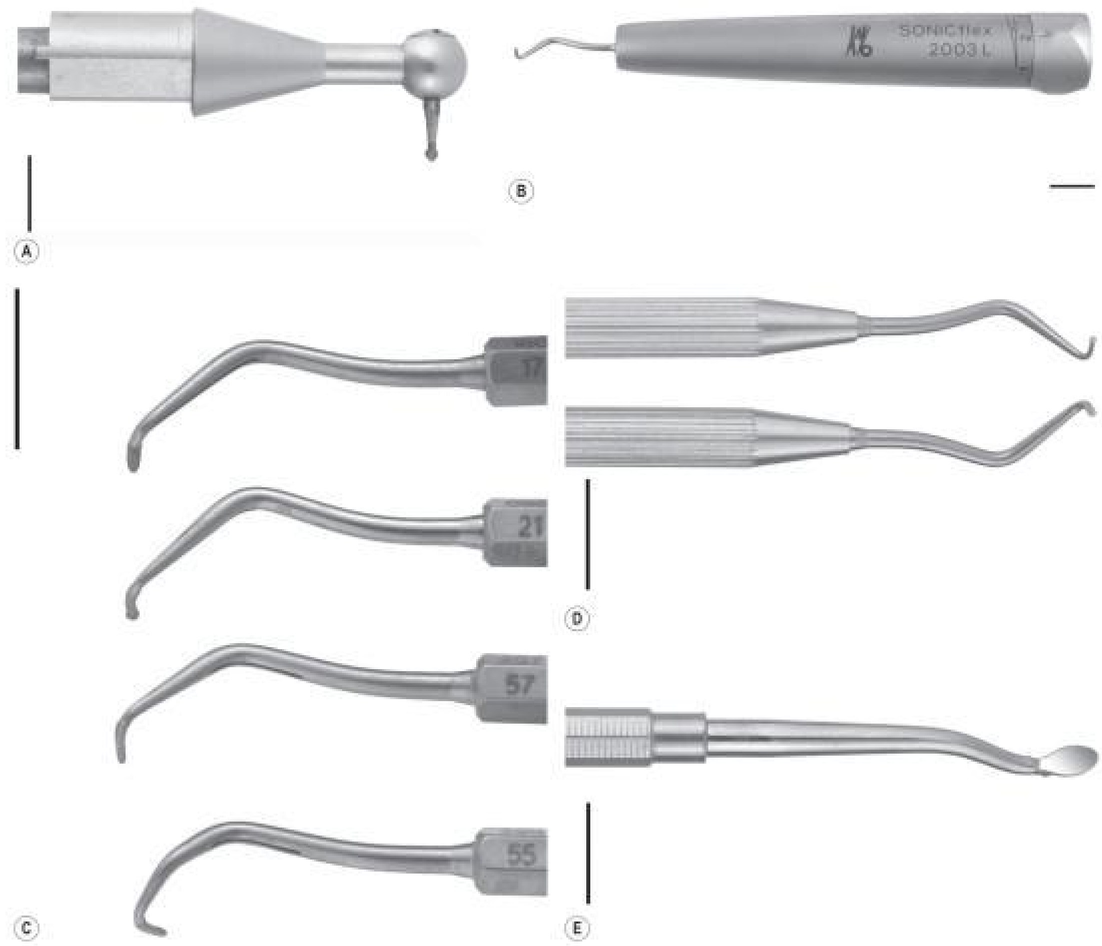


• **FIG. 26.2** Topographical anatomy illustrated by means of osteological specimens and radiograph. (**A**) Transverse section through the mesiobuccal and mesiopalatal roots of the maxillary fourth premolar tooth—note the proximity of the infraorbital canal *(IOC)* and maxillary recess *(MR)*. (**B**) Transverse section through the mesial root of the left mandibular first molar tooth of a small dog—note that the apex of the tooth is located medial to and partly invaginated into the mandibular canal. (**C**) Radiograph of the specimen shown in (B), showing the radiographic illusion of the roots of the first molar tooth seemingly being located within the mandibular canal.

To allow a precise surgical approach, and because anatomical variations are common in dogs and cats,[9,10] surgical planning should ideally be performed using advanced imaging.[1] The two most commonly used advanced imaging modalities for dental and oromaxillofacial applications in small animals are multislice computed tomography and cone-beam computed tomography.[11-13] Although no studies have been performed evaluating the role of advanced imaging in surgical endodontics in small animals, advanced imaging has been shown to impact surgical planning in humans.[14,15] Additionally, the severity and extent of apical destruction may not be readily apparent on dental radiographs, and in some cases, the process may be too severe to warrant additional endodontic intervention and may indicate extraction instead. An example is when the alveolar bone separating the maxillary canine tooth from the nasal cavity is destroyed and an oronasal communication has been established. While radiographs typically do not reveal this, advanced imaging will likely alert the clinician, who may consequently decide to alter the treatment plan. Finally, advanced imaging allows tridimensional image manipulation using specialized software, which may be useful for establishing landmarks for the surgical approach, as well as planning a proper root-end resection angle.[16]

Special instruments and materials

Apart from basic surgical and endodontic instruments, the practice of endodontic surgery requires a specialized armamentarium including ultrasonic or sonic root-end preparation instruments (Fig. 26.3). A selection of specific instruments for endodontic surgery suitable for use in dogs and cats is listed in Table 26.1. Proper magnification of the surgical field by means of an operating microscope or surgical loupes is essential (Fig. 26.4). Although not yet widely available in veterinary clinical settings, the use of an operating microscope for surgical endodontic applications is currently considered part of the standard of care in human dentistry.[17]



• **FIG. 26.3** Instruments for endodontic microsurgery (bar=10 mm). (**A**) Micro handpiece. (**B**) Sonic root-end preparation handpiece. (**C**) Assorted diamond-coated retrotips for the sonic handpiece. (**D**) Retrograde filling pluggers. (**E**) Endodontic micromirror.

• **FIG. 26.4** Surgical root canal treatment using an operating microscope. Modern operating microscopes not only allow excellent control under exceptional lighting and magnification but also promote proper ergonomics allow real-time projection on high-definition monitors for teaching and training purposes and allow the intraoperative capture of high-resolution photos and videos.

TABLE 26.1

Instruments for Apicoectomy and Root-end Preparation in Dogs and Cats.

Description	Details
General	
Surgical loupe	
Small suction tip	Frazier # 6 [a]
Instruments for Approach and Apical Exposure	
Scalpel handle	# 3 [a] or # 5 (round) [a]
Scalpel blade	# 11; # 15C [b]
Periosteal elevator	# 24G [a]
Surgical curette (used as periosteal elevator)	Molt # 2; Molt # 4 [a]
Tissue forceps	Adson 1X2 [a], Adson-Brown 7X7 [a]
Tissue scissors	Goldman-Fox # 16 [a]
Tissue retractors	Senn # 5 [a]
Surgical curette	Lucas # 85 [a]
Excavator (used as a surgical curette)	# 33L [a]
Bone curette	Molt # 5L [a]
Carbide burs	# 2, 701L, and 1558L [c]
Instruments for Apicoectomy and Root-end Preparation	
Micro handpiece	Intralux miniature head; Intralux micro head [d]
Sonic or ultrasonic root-end preparation instruments	Sonicflex Retro [d]
Carbide burs	# 1/2 round; # 33 1/2 inverted cone; # 3 rose [c]
Retrograde carriers	Retro-filling amalgam carrier 1.2 mm (3/64″) or 2 mm (5/64″) [e]
Retrograde filling plugger	# 1; # 2; # 9/10 [a]
Endodontic micromirrors	3-mm round and rectangular [a]
Instruments for closure	
Small needle holder (tungsten carbide inserts)	Hegar-Baumgartner [a]; Halsey (NH-5036) [a]

[a]Hu-Friedy, Chicago, IL.

[b]BD Bard Parker, Franklin Lakes, NJ.

[c]Brasseler USA, Savannah, GA.

[d]KaVo America, Lake Zurich, IL.

[e]Moyco Union Broach, York, PA.

Surgical technique—approach, apicoectomy, and root-end filling

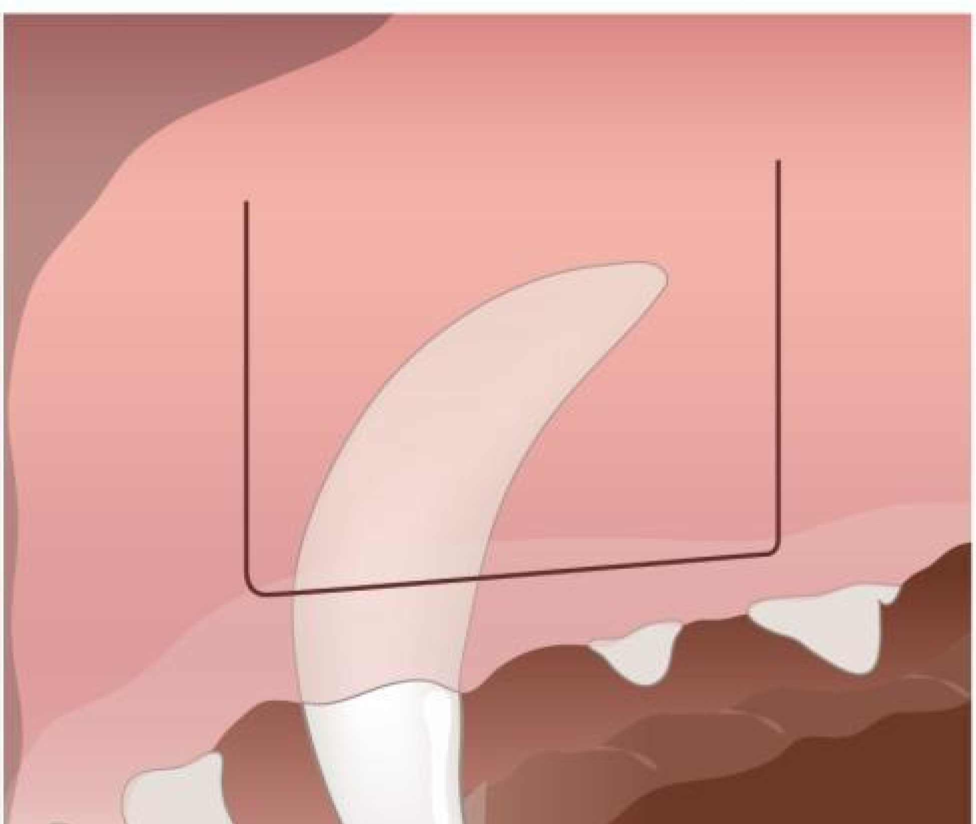
Before performing the procedure on a patient, familiarization with the technique can be gained by assisting an experienced veterinary dentist and by performing the procedure on a laboratory specimen.[6] Aseptic technique is employed.[4] As in nonsurgical root canal treatment, it is important to obtain intraoperative radiographs to evaluate progress so the therapeutic plan can be appropriately adjusted in a timely fashion when necessary.

Flap design

Flap design during surgical root canal treatment should prioritize exposure of the surgical field (i.e., root end and periapex). Since dogs and cats often have a limited amount of attached gingiva, it is not always necessary to include it in the flap. Regardless, the flap must be tension-free, full-thickness, mucogingival, or mucosal; have an adequate blood supply; and expose the apex and periapical tissues.

Semilunar flap

Although other flap designs are common in human endodontic surgery,[1,16] the semilunar flap is the most commonly used in dogs and cats (Figs. 26.5B and 26.7A).[7,18] The exception to this is the mandibular canine tooth, for which a straight rostrocaudal skin incision is made (Fig. 26.6A). The semilunar incision is in the shape of a 3/8 circle and is made entirely in the alveolar mucosa and penetrates to the bone. The apex of the flap is approximately at the apical third of the root(s) and ends apically just dorsal to, and half a tooth mesial and distal of, the apex or apices of the affected maxillary tooth or ventral to the apices of mandibular teeth. The semilunar flap offers a direct but relatively limited exposure.[1,16]



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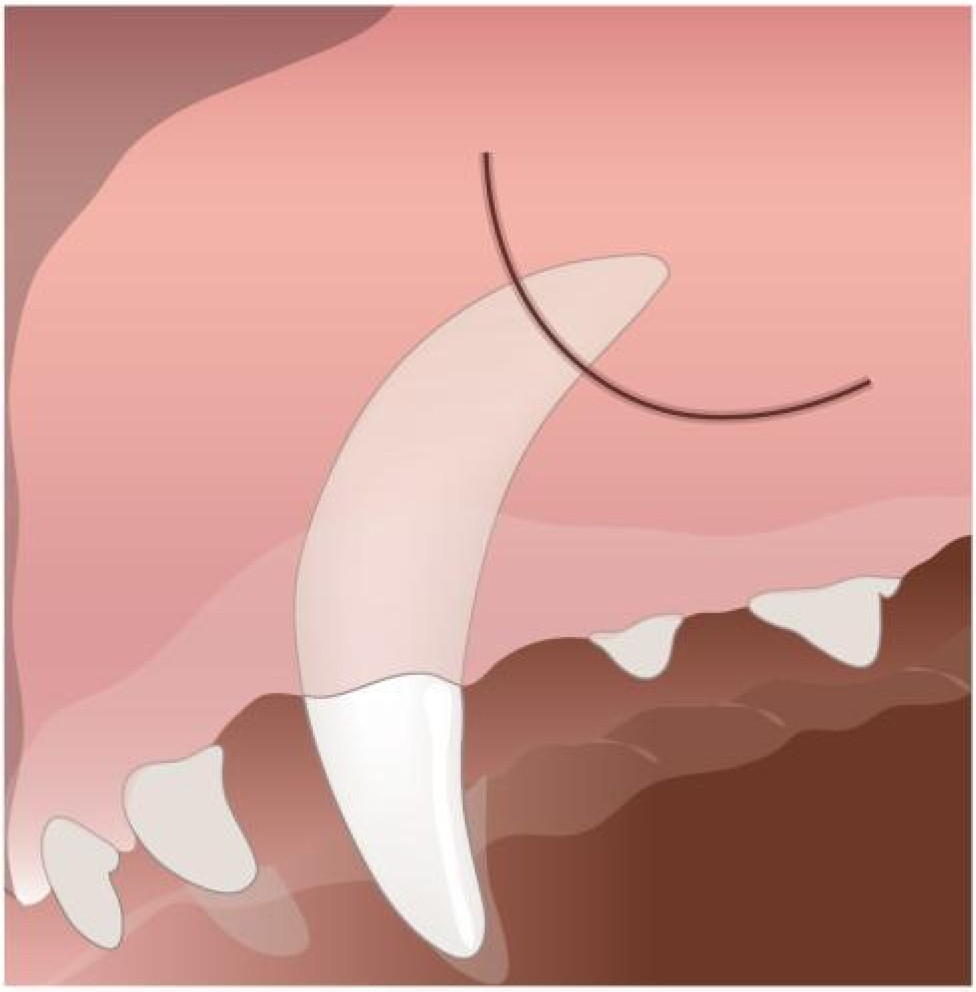

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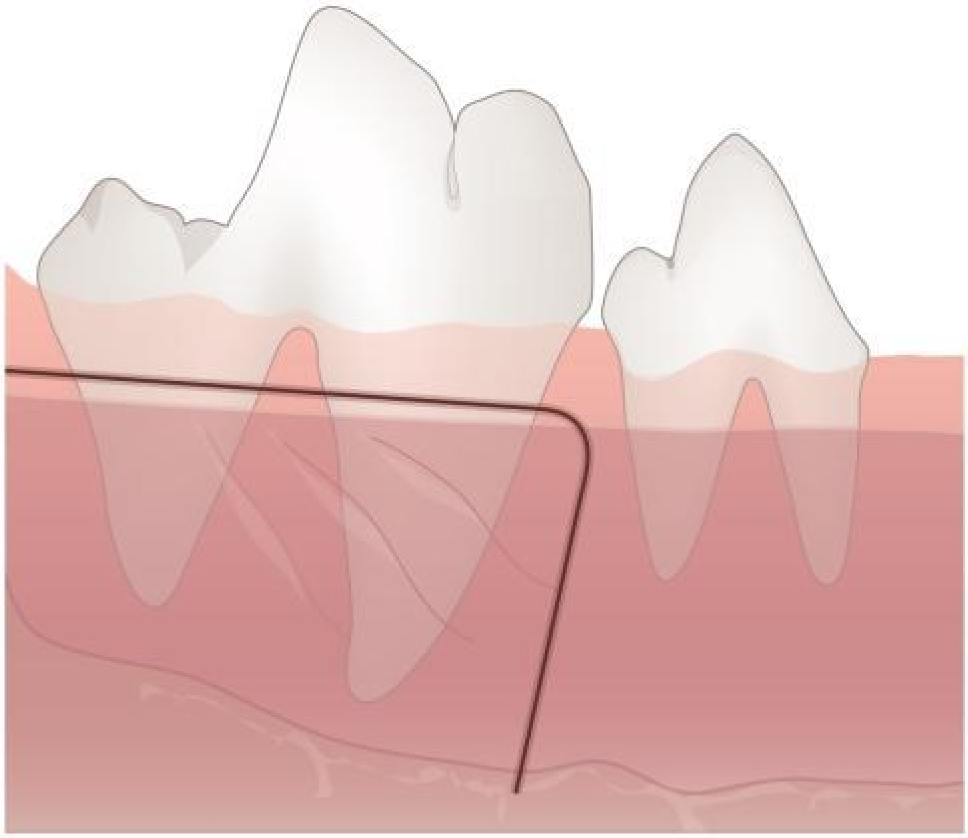

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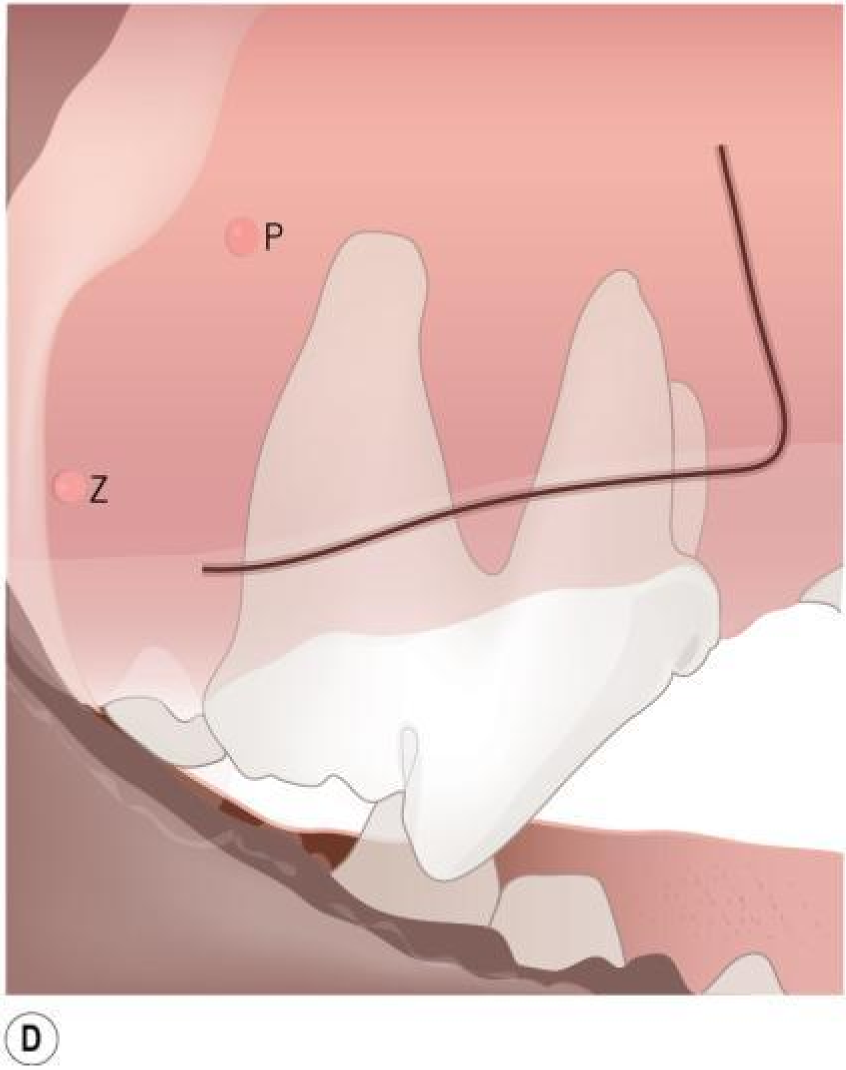
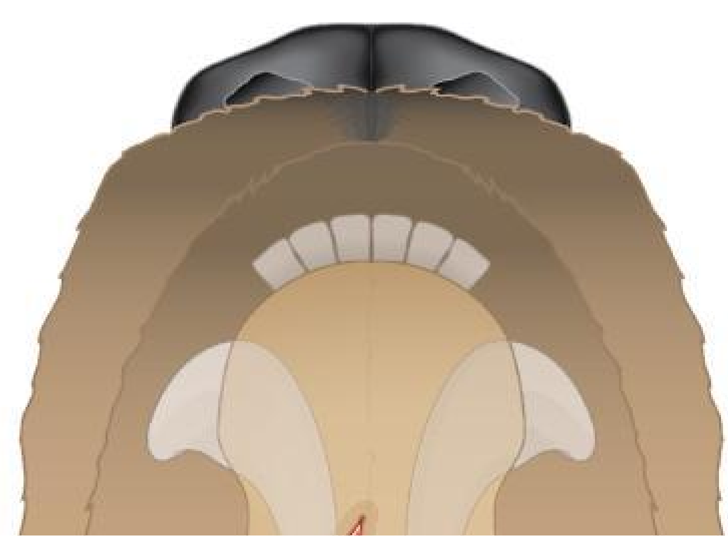


• FIG. 26.5 Flap design for surgical endodontics. (**A**) Pedicle flap with gingival incision for approaching the root of the maxillary canine tooth. (**B**) Semilunar incision into the alveolar mucosa for approaching the root of the maxillary canine tooth. (**C**) Triangle flap with gingival incision for approaching the roots of the mandibular first molar tooth. (**D**) Triangle flap with gingival incision for approaching the roots of the maxillary fourth premolar tooth—note the proximity of the parotid *(P)* and zygomatic papillae *(Z)*.



• FIG. 26.6 Transcutaneous approach to the mandibular canine tooth. (**A**) Line drawing illustrating the landmarks for the soft-tissue incision. (**B**) Intraoperative view of a completed apicoectomy and retrofilling using this approach—note that the apicoectomy was correctly performed near-perpendicular to the long axis of the root.

Triangle (three-cornered) flap

A three-cornered flap is triangular in shape and particularly useful in smaller dog breeds and cats. A full-thickness flap is elevated by making a releasing incision with a # 15C or # 11 scalpel blade, perpendicular to the gingival margin of the mesiobuccal line angle of the tooth to be treated, the mesiobuccal or distobuccal line angle of the tooth mesial to it, or interproximal if this space is wide enough. The vertical releasing incision extends past the mucogingival line and into the alveolar mucosa beyond the level of the apex. The releasing incision is preferably perpendicular to the alveolar margin. The second incision is made, entering through the epithelial attachment of the buccal gingival sulcus and extending from the first incision to a point one-half to one tooth farther than the affected tooth. Alternatively, the sulcular incision is made first, and the vertical releasing incision, second. Instead of a sulcular incision, a submarginal incision can be made into the attached gingiva (Fig. 26.5C and D).[1,16] The latter approach offers the advantage that the gingival contour is not disrupted. Surgical closure is also easier, compared to the sulcular incision technique, as there is tissue available for closure of the horizontal incision of the triangle flap. Using a triangle flap with a sulcular incision in the presence of periodontitis is contraindicated.[1] The full-thickness flap is elevated, beginning in the releasing incision and progressing toward the attached gingiva. One or both incisions may be extended as necessary to permit adequate access and visualization of the apex.

Surgical wound retractors or periosteal elevators used as retractors are important to facilitate good exposure. Most difficulties encountered during flap management result from inadvertent crushing, tearing, or ischemic damage that occurs during incision, elevation, retraction, or suturing.[1,16] Appropriate flap design is essential for the overall success of the procedure.[1]

Pedicle (four-cornered) flap

A pedicle flap design is sometimes required when a larger flap is necessary, as is the case with multirooted teeth and canine teeth in dogs. It essentially is a three-cornered flap that has had an additional vertical releasing incision originating at the attached gingiva on the distobuccal line angle of the tooth involved, the mesiobuccal or distobuccal line angle of the tooth distal to it, or interproximal if this space is wide enough. Similar to a triangle flap, the horizontal incision can be made either in the gingival sulcus or in the attached gingiva (Fig. 26.5A and D).[1,16] For teeth with long roots and/or little attached gingiva, the horizontal incision can be made in the alveolar mucosa. It is currently recommended to make the two vertical releasing incisions parallel,[1,16] although slightly divergent incisions may be necessary in some cases depending on tooth anatomy, to ensure adequate surgical exposure. Slightly rounded corners are preferred in the design of the mucoperiosteal flap.[1,16,19]

Approach and exposure

Maxillary canine tooth in the dog

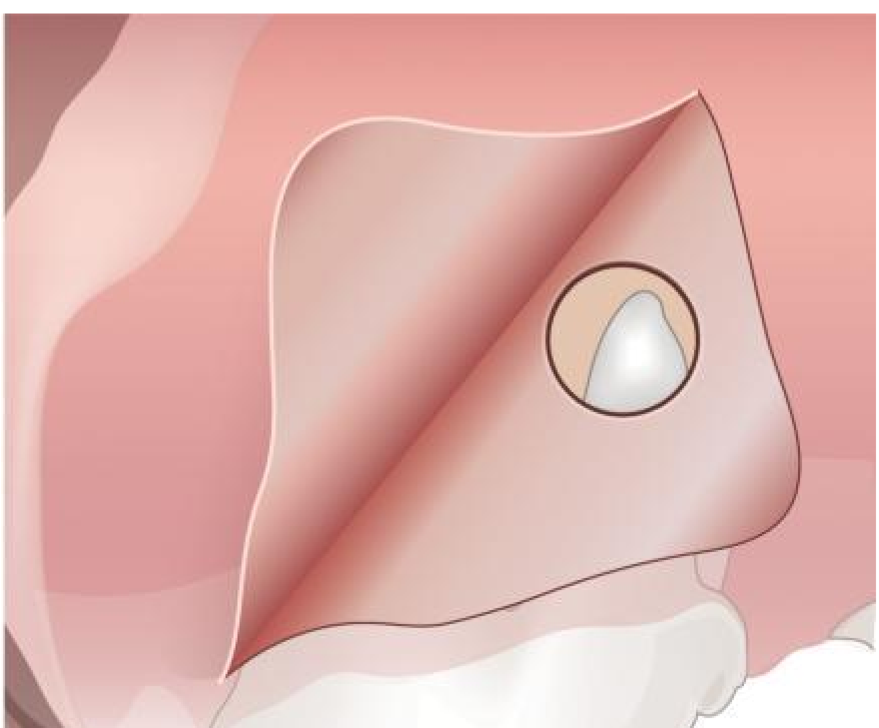
To expose the maxillary canine tooth, a pedicle flap (Fig. 26.5A) or semilunar incision can be made through the alveolar mucosa (Figs. 26.5A and 26.7A). The pedicle or semilunar flap should be wide enough to allow adequate exposure. Both flaps are full-thickness and their base is located dorsally.

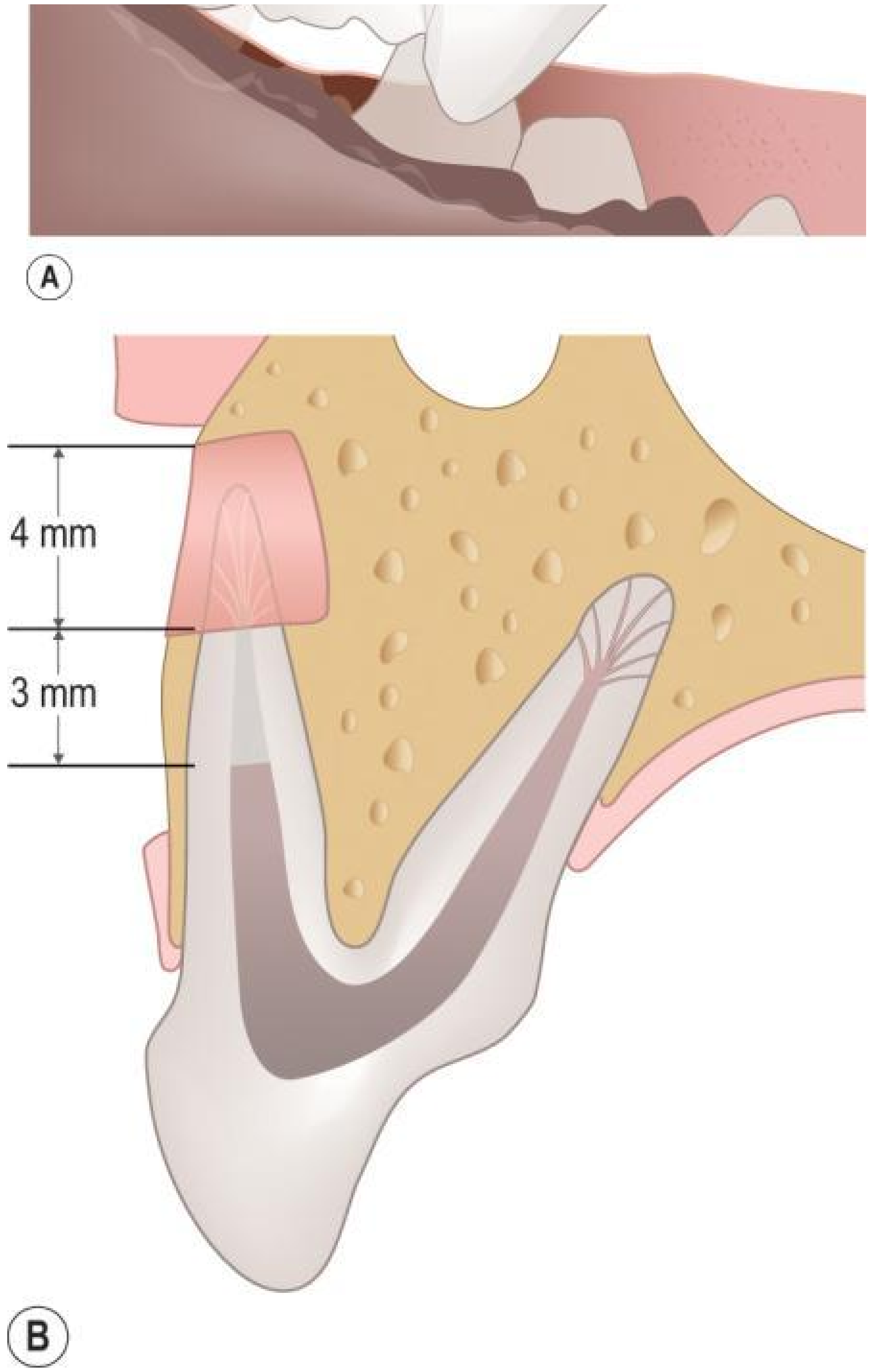
Mandibular canine tooth in the dog

To expose the mandibular canine tooth apex, an extraoral incision is made through the aseptically prepared skin. The incision is directed rostrocaudally on the ventral border of the mandible over the affected site. The apex of the mandibular canine tooth lies just lateral to and slightly rostral to the caudal border of the mandibular symphysis. The incision is extended rostrally and caudally as needed, to permit visualization and access to the periapical tissues (Fig. 26.6B).[4] When present, an osseous fenestration may guide the surgeon to the apex, but generally, it will be necessary to remove a bony window to expose the apex.[16,20]

Maxillary fourth premolar tooth in the dog

To expose the apices of the mesiobuccal and distal roots of the maxillary fourth premolar tooth, a triangle flap with the vertical releasing incision on the mesial aspect of the tooth can be used (Figs. 26.5D and 26.8A and B). Care should be taken to avoid damage to the infraorbital nerve and arteries and corresponding branches, as they emerge through the infraorbital foramen. This flap design may not be ideal if there is alveolar bone loss at the furcation area because it will expose it and potentially result in further loss of attachment. A semilunar incision can also be made through the alveolar mucosa coronal to the root apices. To aid in locating the apices of the mesiobuccal and distal roots of the maxillary fourth premolar tooth, one can visualize that they are located at the dorsal end of an imaginary square, the ventral base of which is the mesiodistal length of the fourth premolar tooth at the gingival margin. The incision should be long enough to adequately visualize the root to be treated. The semilunar mucoperiosteal flap is elevated, with its base located dorsally. The mesiopalatal root of the maxillary fourth premolar tooth cannot be easily accessed surgically. Therefore, amputation of the mesiopalatal root rather than surgical root canal treatment is indicated when failure of conventional root canal treatment is evident at this root.





• FIG. 26.8 Apicoectomy and retrograde filling of the mesiobuccal root of the maxillary fourth premolar tooth. (**A**) Lateral view illustrating the bony window created. (**B**) Transverse view illustrating the near-perpendicular angle of the apicoectomy and the 3-mm retrofilling without undercut.

Mandibular first molar tooth in the dog

The apices of the mandibular first molar tooth can be exposed by making a 3/8 circle, semilunar rostrocaudal incision in the alveolar mucosa, at the level of the apical two-thirds of the two roots. A full-thickness flap, based dorsally to permit enlargement as necessary for increased exposure, is elevated. Adequate exposure with this approach can be difficult to obtain, especially in small brachycephalic breeds. Two alternatives are available to obtain better visualization. A triangle flap with the vertical releasing incision on the mesial aspect of the first molar tooth or the distal aspect of the fourth premolar tooth can be made (Fig. 26.5C). Alternatively, a pedicle flap can be used. An extraoral approach through the skin overlying the ventral border of the mandible has been described, but this approach increases the risk of trauma to the neurovascular structures in the mandibular canal.[6]

Bone removal and debridement

Bone removal is accomplished by removing superficial bone with an irrigated cutting bur such as a # 331L pear-shaped, # 2 round, # 701L tapered crosscut, or # 1558L domed crosscut bur (Fig. 26.7B). Ideally, a surgical handpiece or a high-speed handpiece without air outflow should be used to prevent tissue emphysema (see Chapter 7).[16] Alternatively, a piezoelectric unit with corresponding handpiece and tips can be used for this purpose (see Chapter 9).[21,22] Repeated delicate brush-stroke motions are used, with buccal bone being removed with the side of the bur. This procedure is continued until the unique texture of the tooth root is recognized. A trench is carved around the apex with the bur while exposing at least 5 to 6 mm of root end, being careful that exposure includes peripherally healthy-appearing tissue. It is imperative that the trench penetrates to the medial wall of the alveolus and that the resultant channel created consists visually of healthy-appearing bone. It is important to irrigate during cutting to prevent bone necrosis. The periradicular tissues are debrided, including the removal of any extruded root canal sealant material, with a small cutting bur and curettes (see Table 26.1). Inflammatory tissue is removed as completely as possible before performing the apicoectomy.[16]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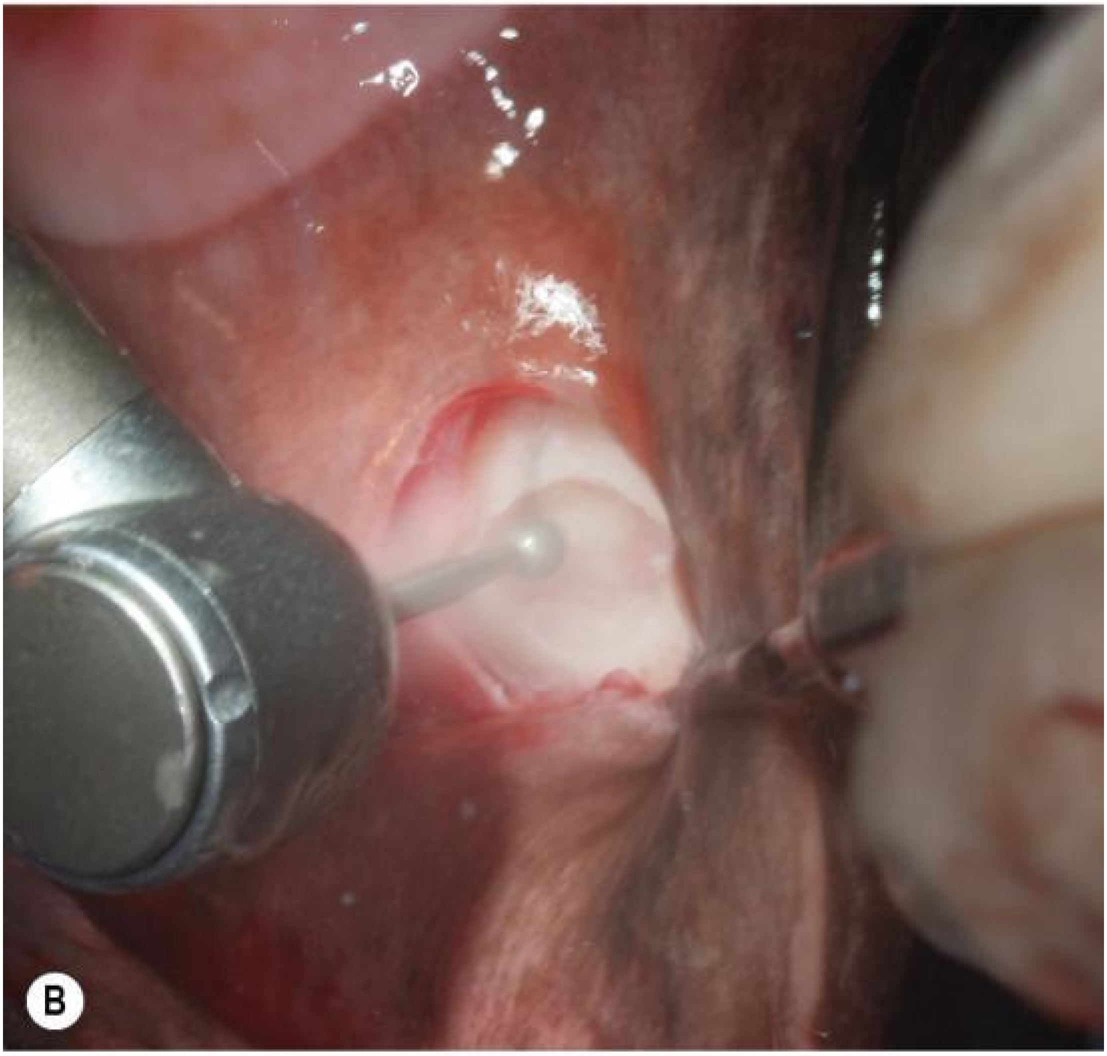

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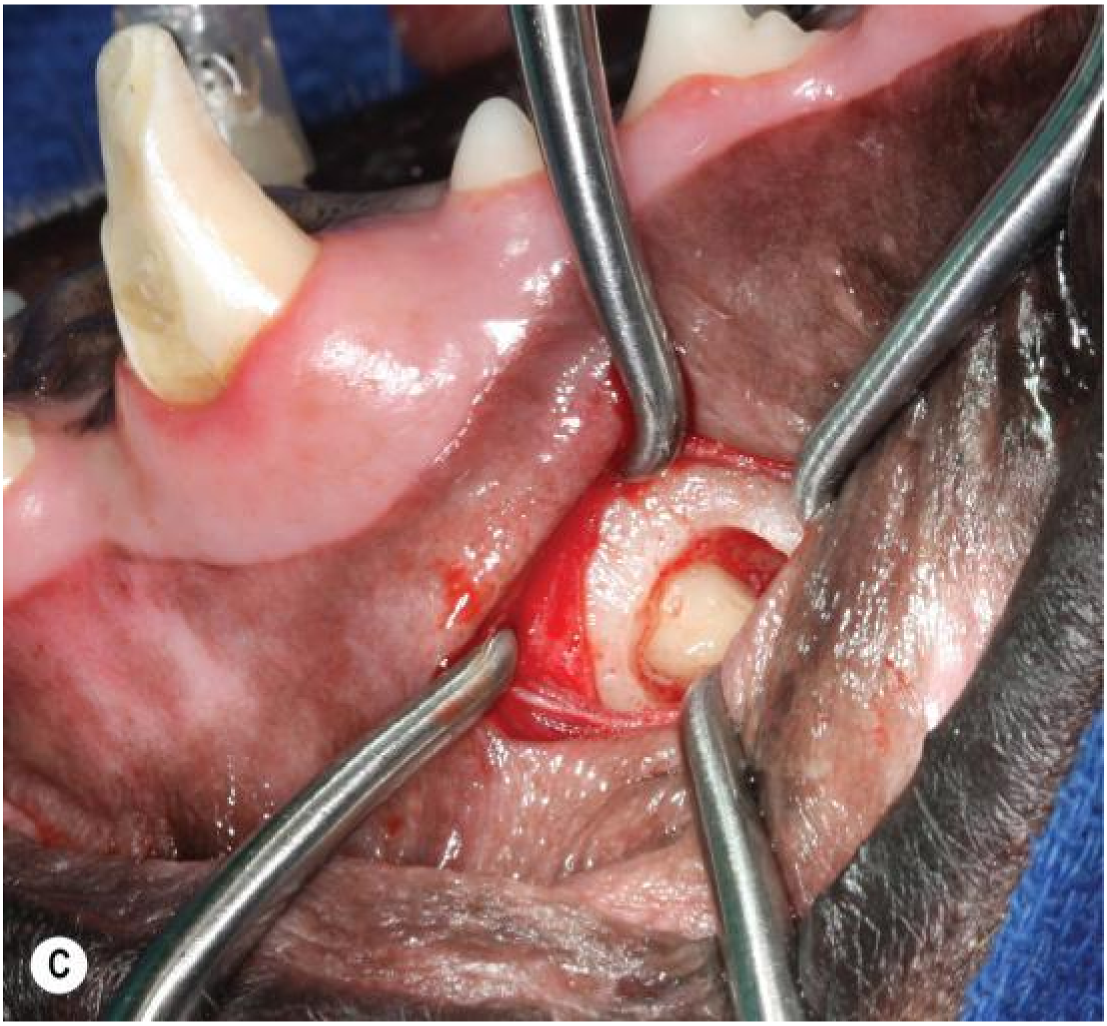
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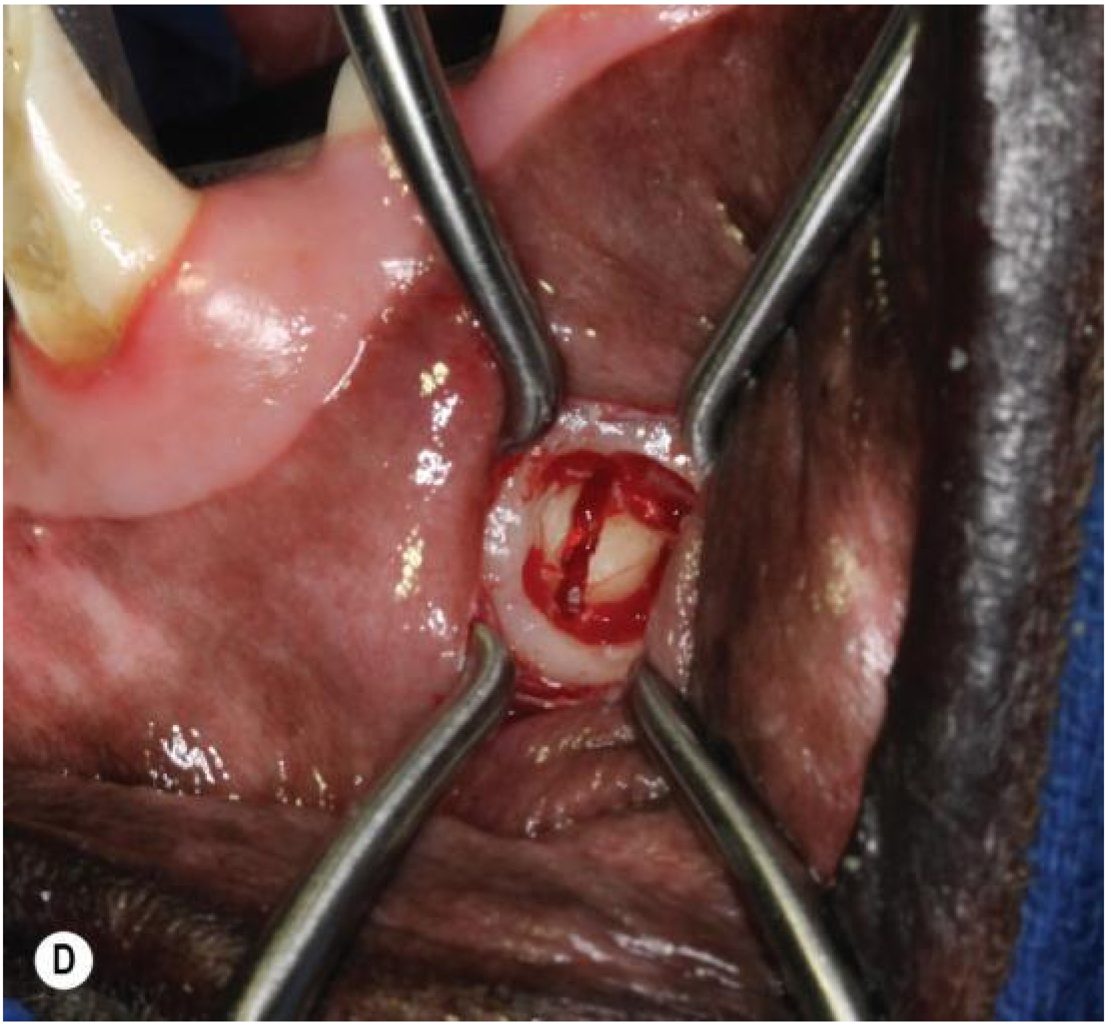
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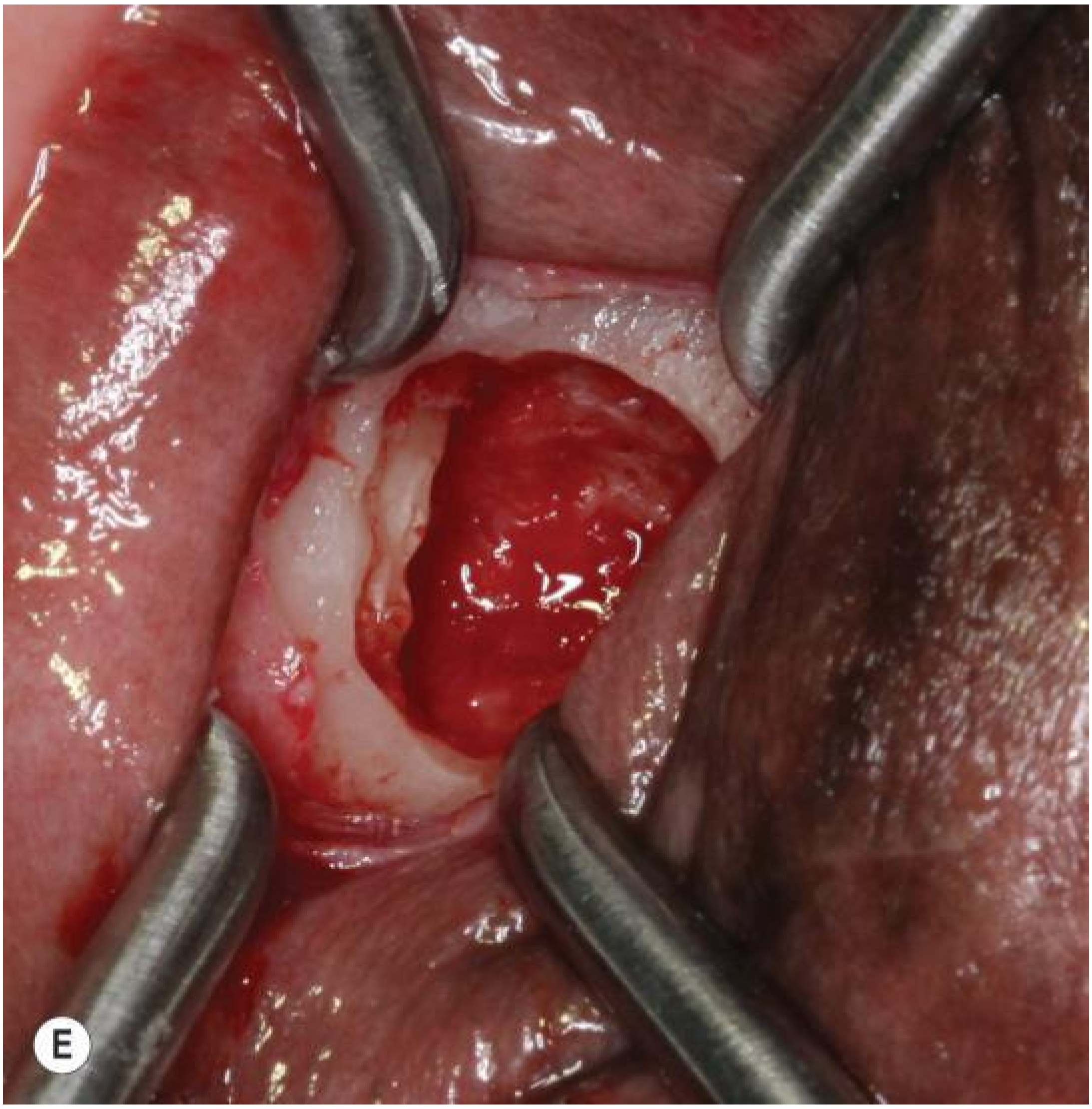


• **FIG. 26.7** Surgical access, root-end resection and retrograde filling of a maxillary canine tooth in a dog. (**A**) Semilunar full-thickness alveolar mucosal incision made over the apical area of the maxillary canine tooth. (**B**) Alveolar bone removal to gain access to the apical area of the tooth, using a round diamond bur on a high-speed handpiece. (**C**) The apical area is exposed and ready for root-end resection. (**D**) The root end has been sectioned in a straight angle to minimize exposure of dentinal tubules. (**E**) Surgical site immediately after apicoectomy.

Apicoectomy

Complete resection of the apex is a very important step during surgical root canal treatment because most endodontic failures occur secondary to apical seal leakage.[1,19] Although the exact resection angle depends on the tooth and root(s) being treated, beveling should be minimized to reduce the number of exposed dentinal tubules and thus reduce the risk of leakage (Figs. 26.7D and E and 26.8).[1,16] The purpose of the resection of the apex is to remove the apical delta, along with any potential lateral canals and any necrotic root that could be a source for residual infection.[20] This is achieved by resecting approximately 3–5 mm of apex. This reduces the chance of leaving residual apical delta or lateral canals on the palatal/lingual wall of the apical end of the remaining root.[1,16] The exact amount of apex to be resected is determined by the visual health of the root, the length of root, and the amount of bone support remaining to maintain a stable tooth subsequent to the procedure.

Once the apicoectomy has been performed, the newly exposed periradicular tissue should be curetted and irrigated with sterile saline to remove inflammatory tissue and debris. When indicated, the periapical tissue that is removed should be submitted for histopathological examination.[23] It should be noted that the diagnostic yield of this practice may be low; therefore, the routine histopathological analysis of resected tissue is not always justifiable.[24] A prudent approach is to submit lesions that are suspicious in nature, based on the history, preoperative evaluation, and intraoperative findings.

Immediately after apicoectomy, visualization of the surgical field is enhanced by using suction equipment. If present, hemorrhage can be controlled by using an appropriate local hemostatic agent (see Chapter 8). Cotton pellets should preferably not be used, as cotton strands left in the surgical site delay healing and result in an inflammatory reaction.[1,20] Bone wax can also be used in apicoectomy procedures to aid in hemostasis, but care should be taken to remove it completely before surgical closure.

Once the root is isolated and hemostasis in the surgical field is achieved, the root-end preparation and filling can be performed.

Root-end preparation and filling

As previously mentioned, most root canal treatment failures are due to apical leakage. Therefore, the retrograde sealing of the canal is very important for therapeutic success. The root-end filling at the apicoectomy site is made so that its outline is parallel to the shape of the root canal, with minimal or no undercut (Figs. 26.7E and 26.9B).[1] Root-end preparation can be performed using small round or inverted-cone burs on a microhead or miniature handpieces. Alternatively, ultrasonic or sonic retrotips can be used for root-end preparation. The apical root canal is prepared to a depth of 3–5 mm, depending on the size of the tooth. Compared with burs, sonic and ultrasonic retrotips are preferred because they remove less tooth structure.[1] Following preparation, the interior canal walls are inspected using a micromirror.

The periapical tissues are air-dried, then packed appropriately to trap any excess root-end filling material and to contain any moisture from bleeding. The filling material is packed into the prepared site in small increments with a 1.2- or 2.0-mm-diameter retro-carrier and then condensed with appropriately sized and shaped condensers, pluggers, or packers. The material is allowed to set according to the manufacturer's directions and smoothed. The marginal integrity of the root-end filling is verified with an explorer because a marginal defect will be an avenue for leakage and failure.

Choice of materials for root-end filling

Many root-end filling materials are available commercially. No single root-end filling material is considered ideal, and all have advantages and disadvantages.[25]

Due to its biocompatibility, mineral trioxide aggregate (MTA) (ProRoot MTA; Dentsply Tulsa Dental, Tulsa, OK) is considered the material of choice if a retentive root-end preparation was achieved.[1,16,17] This material has a high pH, like $Ca(OH)_2$, but is a hard-setting, nonabsorbable compound with cavity adaptation and sealing ability superior to other materials.[26] Its other advantages are that it is the least toxic of all the filling materials, is hydrophilic, and is reasonably opaque. Its main disadvantage is that it has a relatively long setting time and may not be applicable in shallow and/or nonretentive root-end preparations.[17]

Amalgams can also be used for root-end filling. Nonzinc amalgams (Dispersalloy; Dentsply Caulk, Milford, DE) have a higher copper content that results in less marginal deterioration than those with a lower copper content. Products available as comminuted particles (lathe-cut and pulverized particles) are available as both zinc (those containing more than 0.01% Zn) and nonzinc (less than 0.01% Zn) alloys. Even so, studies have shown that amalgam is only 75% effective preventing microleakage due to corrosion.[5] In comparison, Intermediate Restorative Material (IRM) (IRM Intermediate Restorative Material; Dentsply Caulk, Milford, DE) has been used with better results (94%), because it seals well and expands little after setting. Zinc-free amalgam should be chosen because of concerns of precipitation of zinc carbonate into the tissues.[16] Zinc-containing amalgam, if placed in a moist environment, will experience 4% expansion and may result in procedural failure due to creep, restorative expansion or root fracture, or loss of apical seal.[20] The use of amalgam does require that an undercut be made.

IRM is a zinc oxide-eugenol cement reinforced by adding 20% polymethacrylate to the powder.[25] This material has a milder tissue reaction than zinc oxide-eugenol does and it compares favorably with other currently used retrofilling materials; it is easily delivered using a dental syringe delivery system.[4]

Ethoxybenzoic acid cement (SuperEBA; Harry J. Bosworth Co., Skokie, IL) is also a reinforced zinc oxide (60%)-eugenol cement, with 34% aluminum oxide (alumina) added, making it a stronger cement.[1] It is 95% effective against microleakage, is inexpensive, is the strongest and least soluble of the zinc oxide-eugenol formulations, and is user-friendly. It also has excellent antibacterial properties.[27] SuperEBA is readily delivered using a dental syringe delivery system.

Composites may also be used but must be delivered in an absolutely dry field. They can provide as good a seal as the other filling materials mentioned here but are not chosen by most endodontists because they are so technique sensitive.[1]

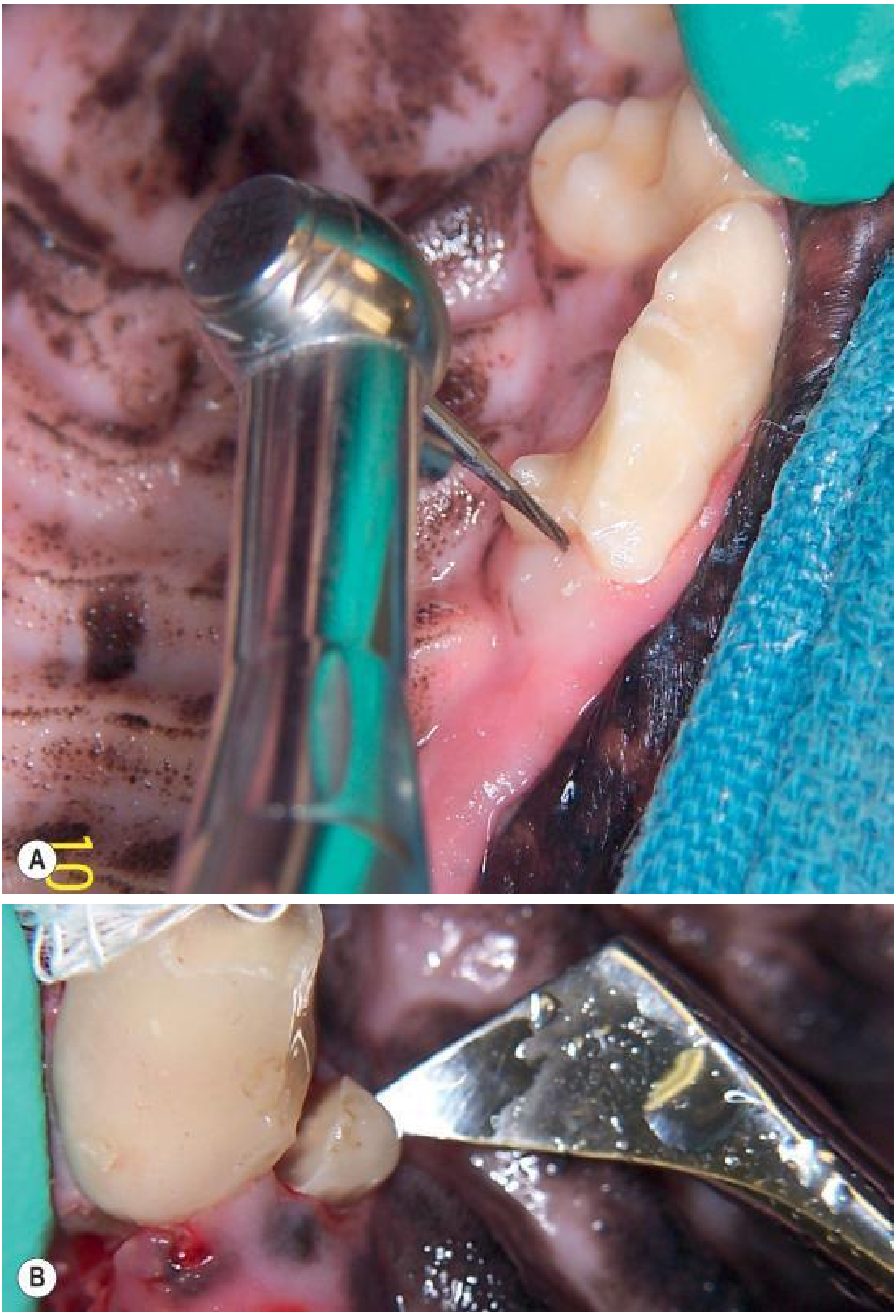
Glass ionomer restoratives have been used, but reports of their efficacy as retrograde restoratives are inconsistent.[28] Ketac Silver (Ketac Silver Aplicap; 3M ESPE Dental Products, St. Paul, MN) was compared to zinc oxide-eugenol and amalgam and was found to be superior in in vitro and in vivo applications.[29,30]

Wound closure

After the apicoectomy, debridement, and restoration have been completed, the surgical site should be irrigated with sterile physiological saline solution to remove debris. The mucoperiosteal flap is sutured with 5-0 or 4-0 absorbable suture material in a simple interrupted pattern. In procedures involving the extraoral approach, the subcutaneous tissues are closed using absorbable suture in a simple continuous or interrupted pattern, and nonabsorbable suture material in a simple-interrupted pattern is used to close the skin (see Chapter 8).

Surgical technique—amputation of the mesiopalatal root of the maxillary fourth premolar tooth

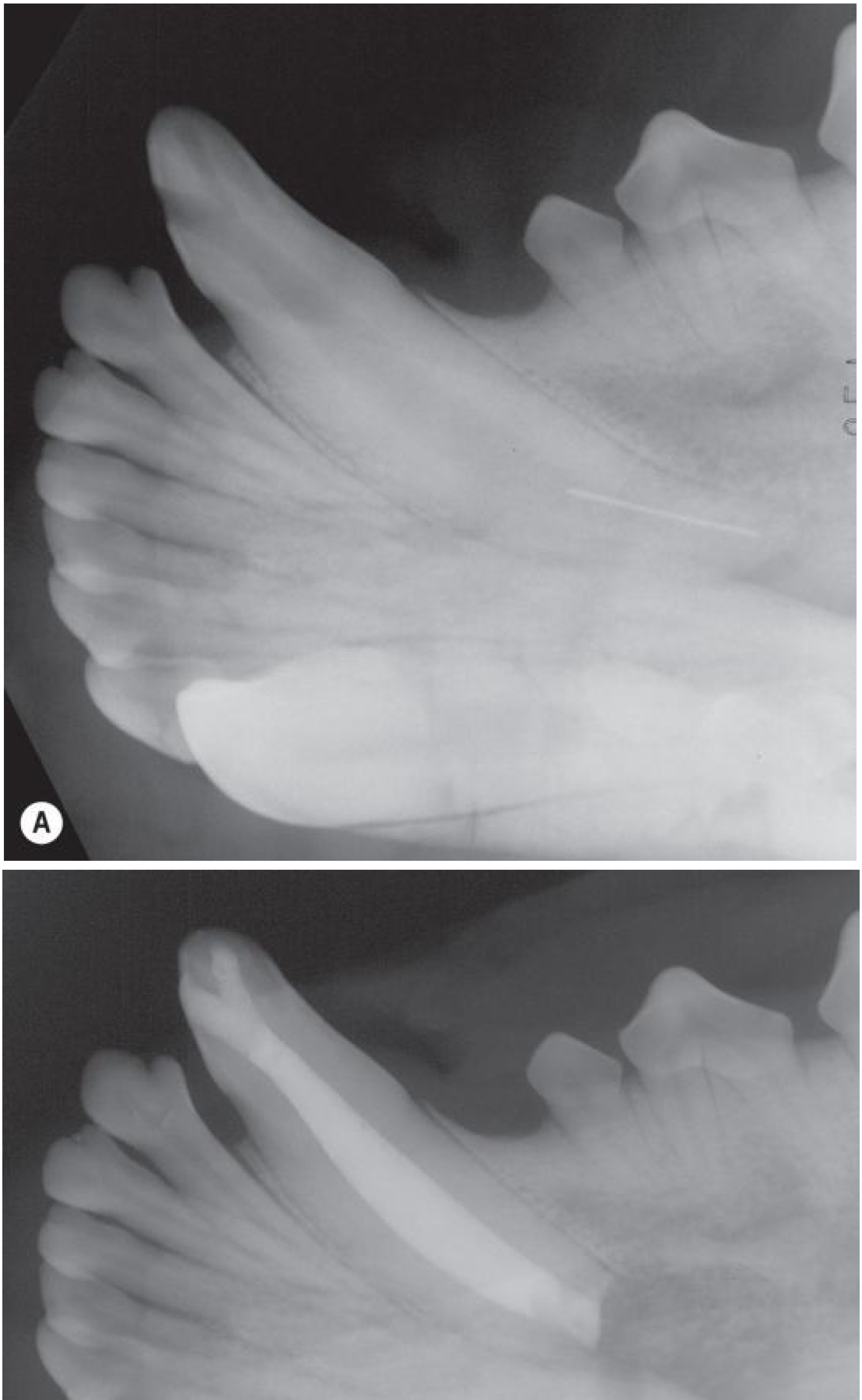
A surgical crosscut bur (e.g., # 1558L or # 701L) or a taper diamond bur is used to section the mesiopalatal root at an oblique, sagittal angle between the mesiopalatal cusp and the mesiopalatal wall of the major cusp of the tooth (Fig. 26.10A). If exposure is limited, a full-thickness palatal mucosal pedicle flap can be raised immediately palatal to the root. After severing its epithelial attachment, the root is removed by elevation or luxation (see Chapter 16) (Fig. 26.10B). The bone periapical to the mesiopalatal root will often be thinned by the inflammatory process and dorsal pressure may cause intrusive luxation of the root into the nasal cavity or maxillary recess.[7] The resultant pulp chamber exposure in the remaining portion of tooth is prepared and restored. If a flap was raised prior to root amputation, it can be slightly rotated to cover the vacated alveolus and to reestablish a gingival collar around the entire tooth.



• **FIG. 26.10** (**A**) The proper angle for resection of the mesiopalatal root of the maxillary fourth premolar tooth, with a # 701L tapered crosscut bur on a high-speed handpiece. (**B**) Use of a Cryer elevator for elevation of the mesiopalatal root.

Postoperative care and assessment

Postoperatively, a radiograph is obtained to verify the root-end filling (Fig. 26.9B). The radiograph is used when comparing follow-up radiographs (see Chapter 25).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• FIG. 26.9 (**A**) File separation occurred during standard root canal treatment on a mandibular canine tooth in a dog, and the file fragment could not be retrieved through the coronal access openings. (**B**) Postoperative radiograph following apicoectomy, removal of the file fragment, normograde root canal treatment, and retrograde filling using glass ionomer; note that there is only a minimal undercut present.

If a draining tract was present, gentle debridement and lavage with 0.9% sterile saline are indicated. In general, a draining tract rapidly resolves following apicoectomy.

Systemic antibiotics are not routinely indicated after surgical endodontic procedures.[1] If an active odontogenic infection associated with the treated tooth was present (i.e., abscess, cellulitis, and osteomyelitis), a course of systemic antibiotics may be indicated (see Chapter 3). The client is instructed to feed only soft food, withhold all hard treats and chew toys, and deny oral play for 2 weeks until the incision has healed. The incision is rechecked after 2 weeks and any remaining sutures may be removed at that stage. A 3-month recheck under general anesthesia and with radiographs is recommended, especially if any swelling persists or drainage is evident. Further radiographic examination should be performed annually therafter.[31]

Complications

Complications related to the surgical approach

Limited surgical exposure may predispose to retention of unnoticed necrotic or infected material that might lead to recurrent infection.

Hemorrhage may be encountered if a major vessel is transected or lacerated. Digital pressure with a moist sterile gauze for a few minutes will usually provide hemostasis. If hemorrhage is persistent, a local hemostatic agent or bone wax will control the bleeding. Bone wax must be removed to reduce the risk of delayed healing or a foreign body reaction.[5]

When using high-speed air-powered handpieces, subcutaneous emphysema may occur occasionally. Use of the equipment should be stopped and the emphysema "milked" manually toward the incision. There is risk of infection when subcutaneous emphysema occurs, and the administration of systemic antibiotics should be considered. Although considered rare, fatal air embolization from air-driven equipment has been reported in humans.[32] High-speed handpieces (Impact Air 45 Handpiece; Palisades Dental, Englewood, NJ) are now available devoid of air at their working end. These newly devised handpieces reduce the risk of tissue emphysema as well as air embolism. The possibility of air embolism with fatal outcome has been reported in an experimental study in the dog.[32]

Swelling and pain are common postoperative concerns. Clients should be advised preoperatively of these possibilities and that the degree of swelling is not proportional to the success of the procedure. Retractor-related bruising is most often the cause for postsurgical swelling. The clinician, being focused on the root-end procedure, is often unaware of this complication until it is too late.

Damage to adjacent anatomical structures

Penetration into the nasal cavity medial to the apex of the maxillary canine tooth can result in epistaxis or rhinitis. The insult will generally heal with the closure of the surgical site.

Perforation into the maxillary recess or nasal cavity may occur during amputation of the mesiopalatal root of the maxillary fourth premolar tooth. If present, exudate in the recess should be drained and the displaced mesiopalatal root should be removed from the recess.

The rostral superior alveolar nerve courses through the infraorbital canal and exits just rostral to the mesiobuccal root of the maxillary fourth premolar tooth in the dog. It may be damaged during instrumentation, where it passes between the mesiobuccal and mesiopalatal roots in small dogs (Fig. 26.2A). The roots of the mandibular premolar and molar teeth, particularly in small breeds, may occupy part of the mandibular canal, and apicoectomy puts the inferior alveolar nerve at risk (Fig. 26.2B). It innervates the mandible, teeth, gingiva, and rostral aspect of the lower lip. Paresthesia may occur if either of the above mentioned nerves is traumatized and infection may spread along the nerve.[16]

Hemorrhage from the cut surface of the alveolar bone may obscure visualization, increasing the risk for iatrogenic damage to an adjacent root or apex, especially in patients where crowded and rotated teeth are present.

Incomplete apicoectomy

Employing a bevel angle greater than 10 degrees in a transverse amputation of the necrotic apex may predispose to leaving a portion of the infected or necrotic apical delta or one or more lateral canals that may happen to be present.[1,16,17] Hemorrhage may interfere with complete amputation of the apex.

Failure of the retrograde filling

The retrograde filling may be dislodged following inadequate root-end preparation or materials placement. Persistent or recurrent infection might occur secondary to inadequate debridement, insufficient apical amputation, or iatrogenic contamination. An incomplete root-end filling predisposes periapical tissues to reinfection.[33]

Filling material inadvertently deposited periapically is a mistake that may be made by inexperienced clinicians. Retreatment may necessary in this case. A postoperative radiograph should be obtained immediately after root-end filling and prior to closure of the surgical site, so that an inadequate procedure can be remedied in a timely manner.

If amalgam is used and amalgam particles are present, they sometimes will be seen later as black "pigmentation" in the mucosa. This phenomenon is referred to as an "amalgam tattoo" and, although unaesthetic, does not pose a problem.

Prognosis

The treatment plan may change intraoperatively; therefore, no guarantee for a successful outcome should be given prior to procedure. However, as long as attention is paid to detail, the tissues are adequately instrumented, and the materials are used in accordance with the manufacturer's instructions, the success rate of surgical root canal therapy can be expected to be very high.

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SECTION 6:

Maxillofacial Trauma Repair

OUTLINE

CHAPTER 27

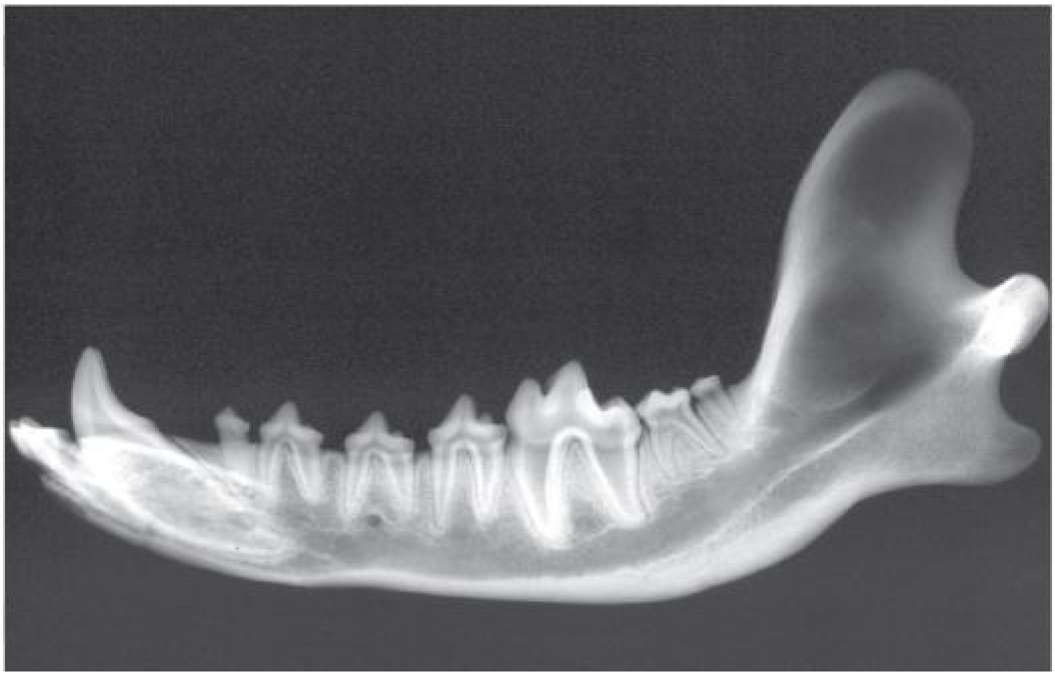
Principles of maxillofacial trauma repair

Randy J. Boudrieau, Boaz Arzi, Frank J. M. Verstraete

Most maxillofacial fractures are of traumatic origin, and there is usually a history of significant blunt or sharp trauma, most often due to motor vehicle impact. Pathologic fractures associated with severe periodontitis, with its associated bone loss, occur most commonly in small-breed dogs, where the teeth occupy a relatively larger portion of the mandible and periodontal disease is more frequent. In these breeds, fractures may occur iatrogenically during routine dental treatment and extractions. Pathologic fractures also may be associated with neoplasia.

Anatomy[1,2]

The mandibles, incisive bones, and maxillae have several unique features complicating fracture management. These bones differ from the rest of the skeleton in that they contain teeth. Most of the dorsal two-thirds of the body of the mandible is occupied by dental roots (Fig. 27.1). The ventral third includes the mandibular canal, containing the inferior alveolar nerve and associated blood vessels. The inferior alveolar nerve provides sensory innervation for the teeth and leaves the bone through three mental foramina as the mental nerves. These nerves are sensory to the soft tissues of the rostral part of the lower jaw. The blood vessels in the mandibular canal are most important, as they supply all of the teeth. Ventral to the mandibular canal there is only a single layer of dense cortical bone.



• **FIG. 27.1** Lateral radiographic view of the mandible of a large-breed dog. The volume that the teeth occupy can readily be appreciated, encompassing approximately two-thirds of the dorsoventral height of the mandible. It should be noted that in small dogs the teeth occupy a greater proportion of the mandible.

The lower jaw consists of two mandibles, joined at the symphysis, which consists of a synchondrosis between the slightly irregular bony surfaces. The symphysis remains a true joint throughout life in the dog and cat. The term *symphyseal separation* is therefore more accurate than symphyseal fracture. The mandible consists of a body (often incorrectly referred to as the horizontal ramus), which is the tooth-bearing part; and a ramus, which is the caudal, non-tooth-bearing part. The body of the mandible can further be divided into an incisive part; and a premolar–molar part. Fractures involving the incisive part are typically discussed together with symphyseal separations, while discussions of fractures of the body of the mandible focus on the premolar–molar part of the mandible. The ramus of the mandible contains three prominent processes: the coronoid process on the dorsal aspect, the condylar process caudally, and the angular process caudoventrally.

The maxillary teeth are located in the maxillary and incisive bones. The term *maxillary fractures* in veterinary surgery often refers to fractures involving the incisive, palatine, zygomatic, lacrimal, frontal, and nasal bones, in addition to the maxillary bone proper. The close proximity of the nasal cavity, maxillary recess, orbit, infraorbital canal, and cranial nerves and associated blood vessels is of great importance in the management of fractures in this area.

General considerations

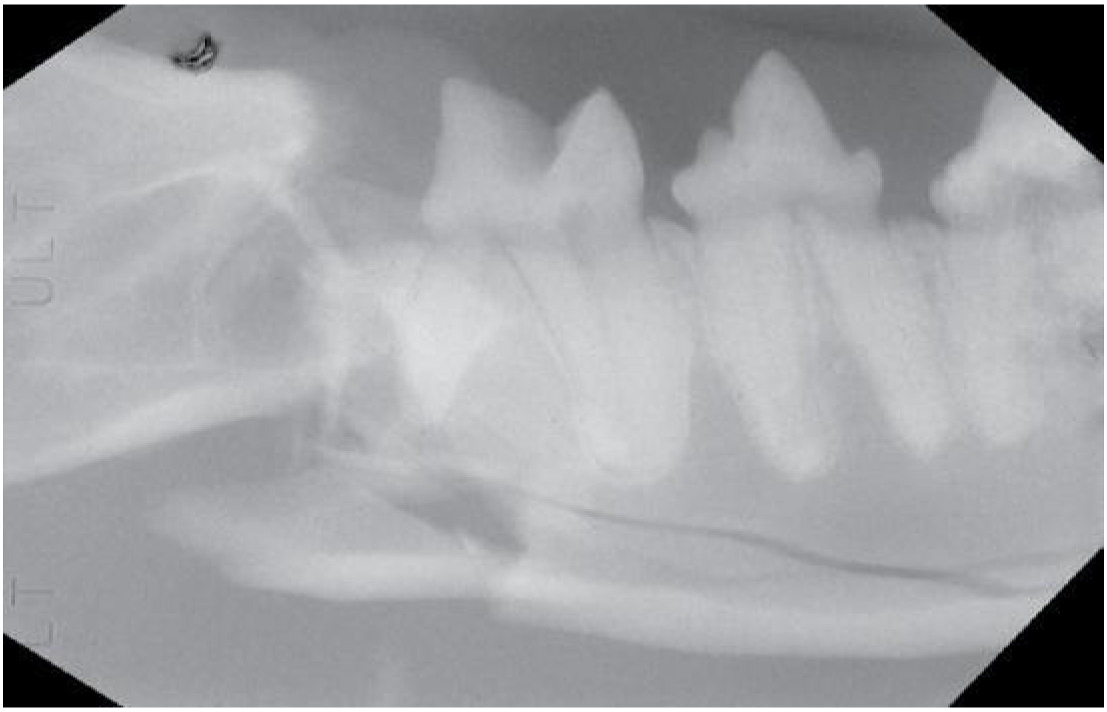
Initial management

A mandibular fracture usually is an obvious lesion; a potential pitfall exists in that other less obvious but equally serious problems may go unnoticed. The diagnosis of a fracture of the mandible usually can be made by inspection and/or palpation. The ventral margins of both mandibles should be gently palpated for asymmetry and discontinuity by placing the fingers in the patient's mouth adjacent to the alveolar margin (Fig. 27.2). Some patients will permit gentle opening of the mouth, which will allow a visual assessment. As most fractures are open to the oral cavity, any discontinuity in the dental row, gingival lacerations, or bony discontinuity usually are easily palpable or visible. Fractures caudal to the teeth, however, are much more difficult to assess on physical examination. The nature and extent of the fracture are best assessed under general anesthesia by gentle palpation and diagnostic imaging.

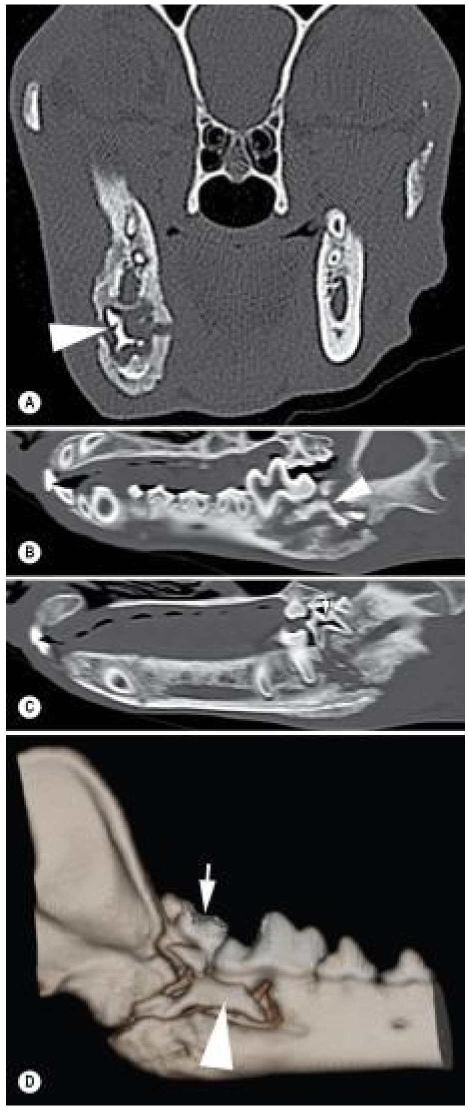
• **FIG. 27.2** **(A)** Cranial and **(B)** lateral photographs of digital palpation of the mandible. **(A)** The index fingers are positioned along the gingival alveolar margin under the lips. **(B)** The mouth may also be gently opened during this palpation to allow a visual evaluation.

Diagnostic imaging is necessary to visualize the fracture site(s) and to diagnose any concomitant dental trauma. Conventional radiography is difficult to interpret due to the many overlying structures of the skull and the bilateral nature of the regional anatomy and is not recommended for the evaluation of oral and maxillofacial trauma (see Chapter 6). Oral radiography using dental film is preferred to conventional skull radiography, but also is limited to the areas of the dental arches. Dental radiographs provide fine detail of the trabecular structure of the bone and document the involvement of teeth in maxillofacial fractures (Fig.

27.3). Fractures involving the ramus or condylar process of the mandible, as well as maxillary fractures, may be observed with standard radiographs, but are best visualized using computed tomography (CT) (Fig. 27.4).

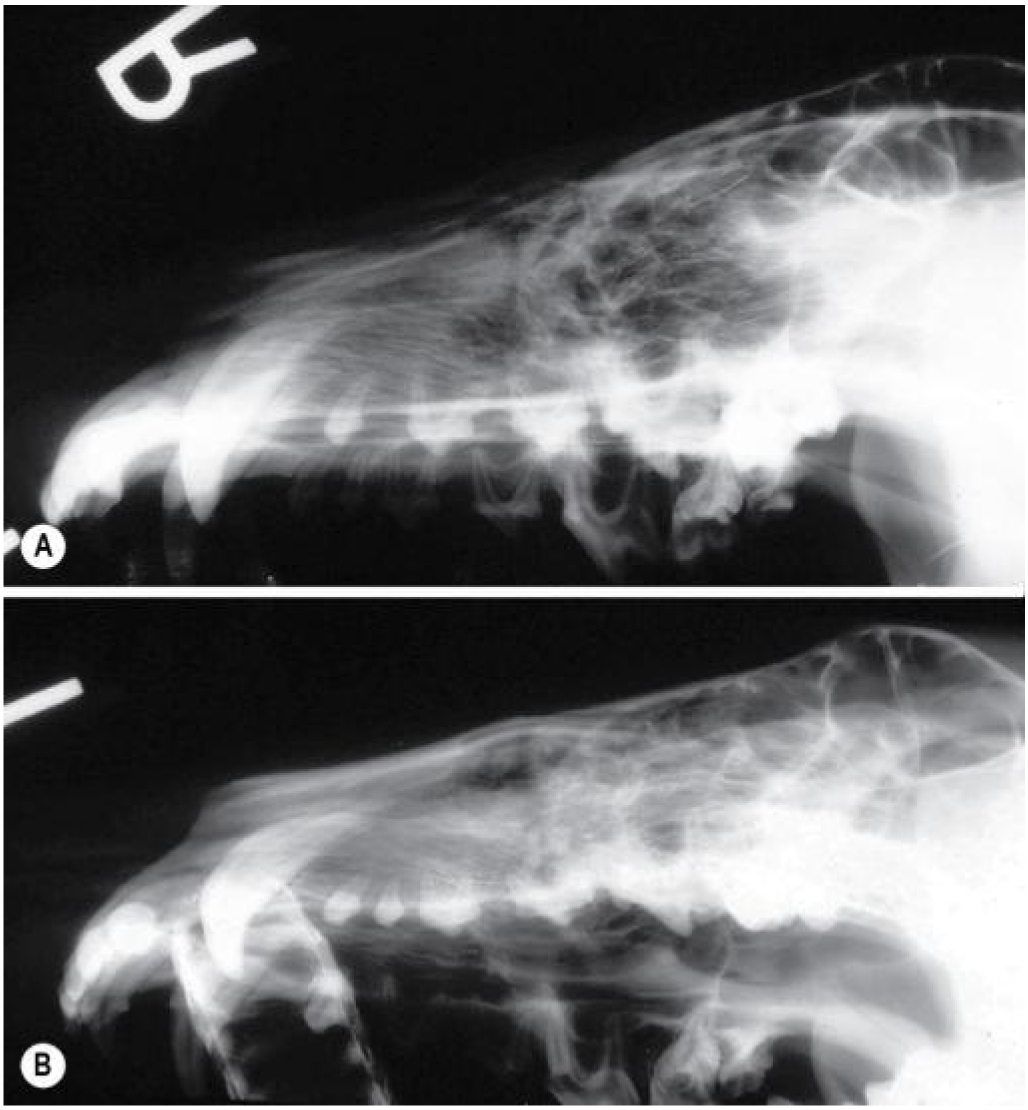


• **FIG. 27.3** Dental radiograph of a mandibular fracture in a cat showing fine trabecular detail and tooth involvement.

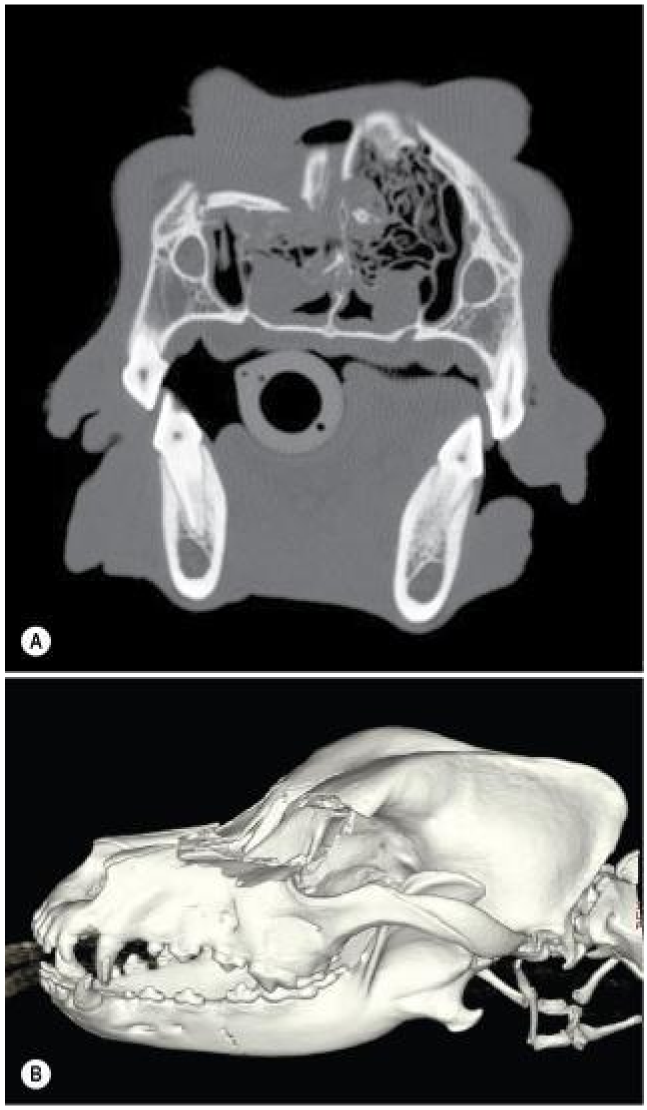


• **FIG. 27.4** Computed tomography (CT) images centered on the region of a fracture nonunion of the angle of the right mandible: **(A)** Transverse single slice; bright density *(arrowhead)* is a sequestrum. **(B)** Sagittal single slice again illustrating the sequestrum *(arrowhead)*. **(C)** Sagittal single slice through the roots of the mandibular second molar tooth *(arrow)*; the fracture traverses through the mesial root and the distal root is not in contact with bone, indicating a loose tooth. **(D)** Three-dimensional reconstruction in the same area demonstrating the fracture nonunion at the angle of the mandible; the reconstruction provides an improved rendering of the overall anatomy but fails to identify the mobile right mandibular second molar tooth *(arrow)* or the sequestrum *(arrowhead)*.

Fractures in the nasal area may be difficult to diagnose as the bone fragments usually are stable (commonly occurring as impaction fractures), and radiographic evidence may be difficult to interpret due to superimposition of the bones and the complex ethmoid and turbinate patterns (Fig. 27.5).[3] Fractures that involve the nasal turbinates may interfere with breathing. Longitudinal fractures along the center of the nose also are commonly observed with very stable position of the fragments (occurring more frequently in cats than in dogs). Swelling, pain on palpation, and nasal bleeding may be signs that maxillary fractures are present. CT is the most useful diagnostic tool utilized if any fractures are suspected (Fig. 27.6). When evaluating maxillary fractures, it is important to recognize upward or lateral deviation of the nose so that proper nasal passage alignment and dental occlusion can be attained with fragment reduction and subsequent fixation. Recognition of mild nasal malalignment can be difficult when coexisting fractures of the mandibles are present, since dental occlusion cannot be used to evaluate the accuracy of reduction. Similarly, fractures of or near the orbit may involve supporting structures of the eye. Due to the absence of circumferential bone, these injuries require special attention to reconstruct the supporting soft tissue structures.



• **FIG. 27.5** **(A)** Lateral and **(B)** lateral-oblique conventional radiographic views of the maxilla in a dog with comminuted maxillary fractures. The location and extent of the fractures are difficult to evaluate due to superimposition of the bones in this area and the overlying complex ethmoid and turbinate patterns.



• **FIG. 27.6** **(A)** Transverse computed tomography (CT) single slice and **(B)** tridimensional reconstruction in the same area demonstrating multiple fractures of the maxilla—including a bilateral depression of the nasal bones and bilateral maxillary fractures. Source: (From Arzi B, Verstraete FJM. Internal fixation of severe maxillofacial fractures in dogs. *Vet Surg* 2015;44(4):437–442, with permission.)

Large bone fragments involving the maxilla or incisive bones also may involve the attached dental structures. Open reduction and internal fixation (ORIF) of these large fragments is recommended to attain appropriate repositioning not only of the nasal cavity but also of the dental arches so as to ensure appropriate occlusion.[4] Fractures through the dental arches elevate and/or tear the gingival mucosa, which also must be addressed after fracture reduction and fixation. It may be necessary to mobilize the labial mucosa to ensure soft tissue closure.

Maxillofacial trauma is most successfully managed by early definitive fracture fixation. In general, oral and maxillofacial trauma should be considered an urgent rather than an emergency indication for surgery. Trauma to the head may also involve traumatic brain injury and/or cervical spine injury. In the face of any neurologic deficits, or uncertainty as to the patient's neurologic status, surgery is delayed in order to better assess/diagnose other life-threatening issues that could put the patient at risk with early anesthesia/surgery. A short surgical delay permits a more thorough evaluation and allows time to coordinate a multidisciplinary approach to patient care. In human maxillofacial trauma, "early" and "late" repairs are defined as <48 or

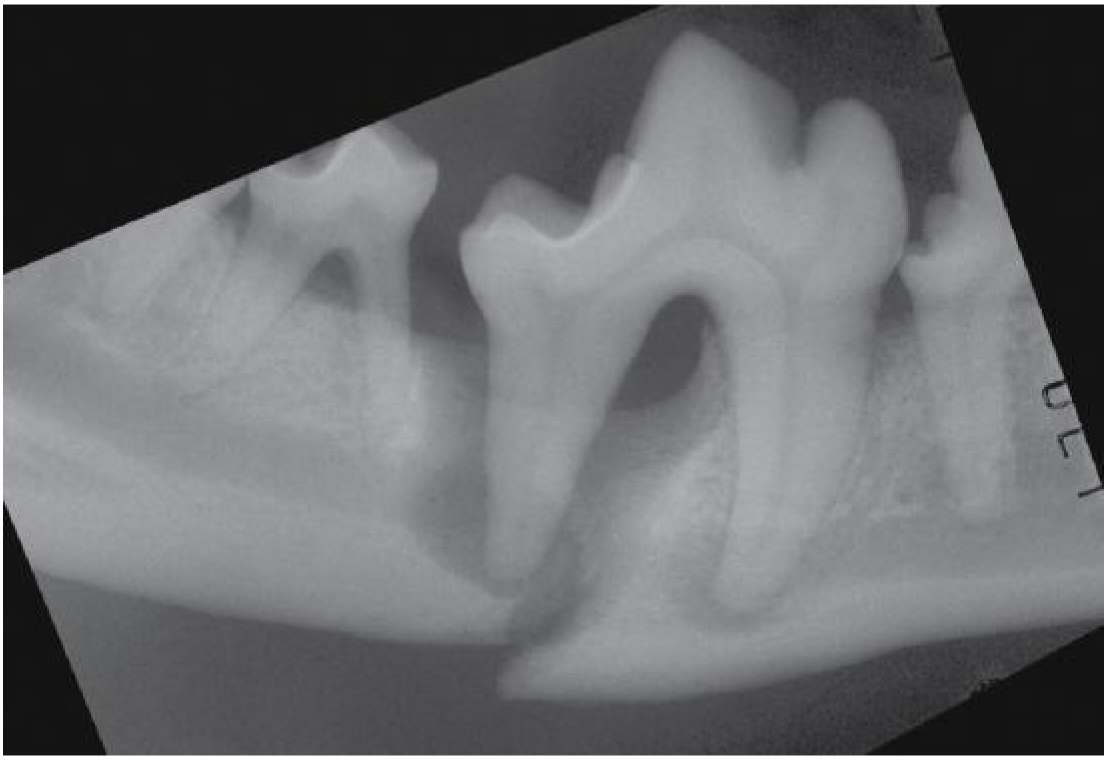
>48 hours from admission to surgery, respectively. Length of hospitalization and rate of complications (e.g., infection) are similar, which further supports such a judicious approach.[4]

Early fracture treatment facilitates the associated soft-tissue management and most quickly restores function.[5–7] Most patients will resume eating quickly if so treated, provided the reduction restores occlusion and the fixation applied provides rigid stability. The patient's cosmetic appearance also is restored. Nutritional support should be considered, as in instances of severe trauma, where the animal may remain inappetent. Those patients in which a definitive procedure is delayed should be managed with temporary bony support (muzzle) and adjunct nutritional supplementation (bypassing the oral cavity). Delay of definitive repair should be avoided, however, as pain control and soft tissue management are very difficult to administer successfully.

Assessment of teeth

Trauma to the teeth frequently occurs with maxillofacial fractures. Fracture of a tooth may expose the pulp cavity, or compromise the blood supply as a result of a bone fracture adjacent to the tooth root. Significant patient morbidity will result if such lesions are not appropriately addressed. Continuous licking, reluctance to chew, resulting in diminished food intake, and sensitivity to heat or cold are all clinical signs consistent with ongoing dental issues.

An increased frequency of complications with fracture healing has been observed when teeth are removed due to their involvement with fractures of the dental alveoli.[8] Removal of intact teeth may increase complications due to disruption of the blood supply and iatrogenic trauma to the adjacent tissues, including the following: further displacement of the fractured bone fragments; elimination of occlusal landmarks useful in realigning bone segments to allow functional occlusion; elimination of available structures for use in the fixation of bone fragments; and creation of a large bony defect adding to the difficulty of reduction and stabilization. Several studies in humans have established that intact teeth remaining in a fracture line did not increase the complication rate or morbidity associated with mandibular fractures.[9,10] Preservation of intact teeth involved within a fracture line in mandibular fractures also has been reported to have a favorable prognosis if optimal reduction and stabilization of the jaw has been achieved.[11] Therefore removal of intact teeth is not advised unless involved teeth are fractured, are affected by periodontal disease or a periapical lesion, or are mobile and cannot be stabilized (Fig. 27.7). Current recommendations by human oral surgeons are to preserve teeth whenever possible in the presence of a fracture.[11–13]



• **FIG. 27.7** Dental radiograph of a mandibular fracture in a dog through the alveolus of the distal root of the first molar tooth with severe periodontal disease and a large periapical lesion.

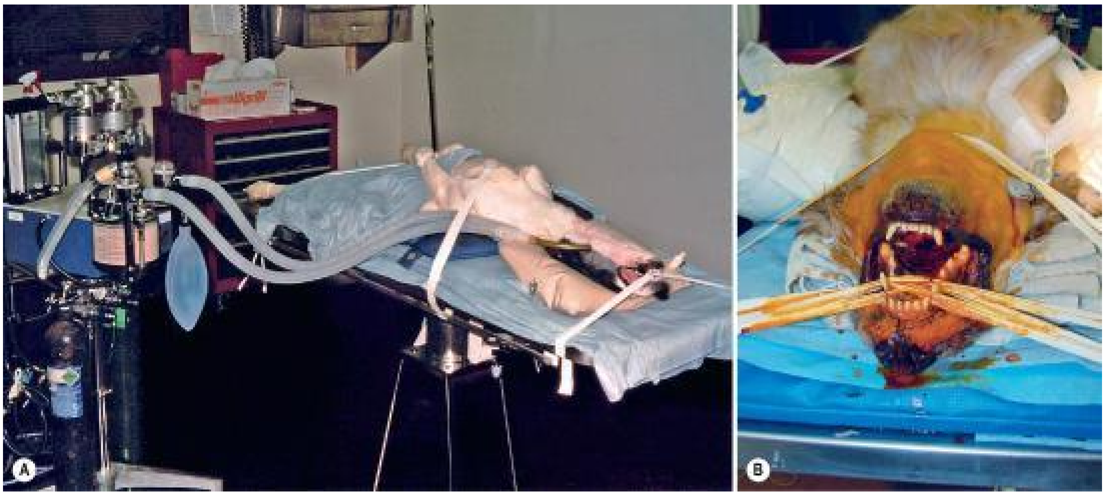
Fracture of the alveolar bone or fracture of a tooth root will usually result in a loose tooth. If only the alveolar bone is fractured and the tooth structure is intact, the preferred treatment is to leave the tooth in place and to stabilize the fracture.[13] Teeth that are avulsed may be reimplanted provided the alveolus remains intact[12,14,15] or can be reestablished with stable fixation.[10,12] Root canal treatment is required in these cases once the tooth is stable, but need not be performed at the time of the initial fracture repair.[12,16] If the fracture involves the tooth, treatment approaches vary. Teeth that are not salvageable and not essential to the stability of the fracture repair should be removed. These extractions most commonly involve the incisor or smaller premolar teeth. Endodontic treatment consists of vital pulp therapy or root canal treatment. The indications for these techniques vary with the severity of the damage to the pulp.[17,18] Vital pulp therapy is designed to treat and preserve vital pulp. If the vitality of the tooth in the fracture area is uncertain, the fracture is treated and the tooth is reevaluated both during and after fracture healing.

Fractures in the presence of periodontal disease further complicate fracture management (see Fig. 27.7). In simple fractures, the recommended therapy involves periodontal treatment and appropriate extractions, especially if the tooth with periodontitis is at the fracture site. Loose teeth should be removed due to the presence of underlying bone disease, as alveolar bone resorption already is present, which inhibits healing. As noted, extraction is indicated if the tooth is affected by periodontitis, implying that the supporting alveolar bone has been damaged and infected. Fracture management usually is difficult in cases with complex fractures and severe osteolysis due to the presence of preexistent periodontal disease, and nonunion and fixation failures are common. Fracture healing is inhibited in the presence of the existing bony destruction as a result of the ongoing osteitis. This poor bone quality does not hold metal implants well, as they frequently pull through the soft, diseased bone. Historically, in order for these patients to retain a functional mandible, the desired outcome was to have fibrous as opposed to bony unions. However, in the presence of bilateral mandibular fractures, there is no inherent stability, and these cases are much more difficult to manage. Therefore any attempt at a bony repair in the face of severe periodontal disease must be performed judiciously. With the advances of internal fixation and regenerative solutions, nonunion mandibular fractures, or for fractures associated with periodontal disease, bony union can be achieved (see Chapter 53).[19] In certain situations with rostral mandibular fractures in the presence of moderate-severe periodontitis, a rostral mandibulectomy may be the only recourse. Regardless, a goal of a fibrous union or a plan for mandibulectomy requires appropriate client education regarding outcome and/or potential complications.

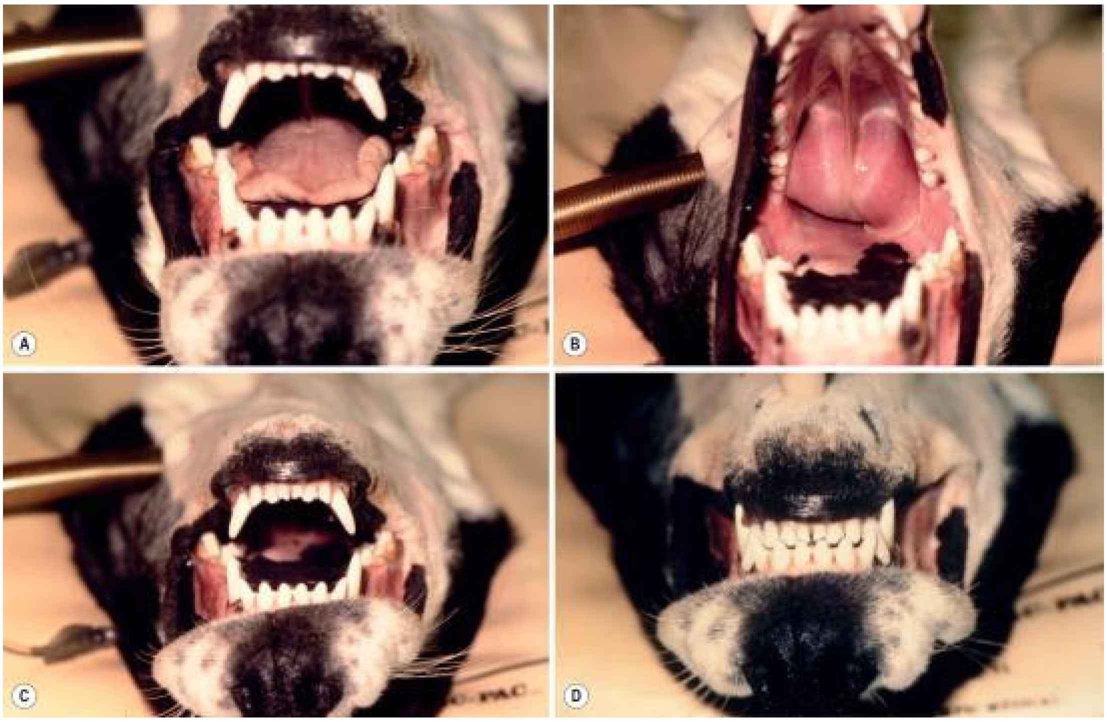
Anesthetic and surgical positioning

Treatment of maxillofacial trauma poses unique problems of appropriate surgical access. Special attention must be given to maintaining an adequate airway, including appropriate tracheal access, and to proper patient positioning so as to secure the head and simultaneously permit unimpeded approach to the bones of the skull and mandible. Routine induction and endotracheal intubation per os for anesthetic maintenance and surgery is performed in the management of simple fractures (large fracture fragments without comminution). Anatomic realignment and reduction of the fracture fragments, rather than dental occlusion, is used to determine the accuracy of surgical reduction in these instances. However, dental occlusion must be used to assess the accuracy of the surgical reduction in cases of severely comminuted fractures or those with bone loss (e.g., gunshot). In these instances, the endotracheal tube must be replaced so as to bypass the mouth (pharyngotomy or transmylohyoid endotracheal intubation) so that the mouth can be fully closed to assess occlusion (see Chapter 61) intraoperatively.

Patient positioning must include consideration for both unimpeded surgical access to the head and anesthetic management that does not interfere with the surgical procedure. Most often, this includes positioning the patient such that the head is at the opposite end of the surgical table from the anesthetic machine (Fig. 27.8A). Additionally, the head should be securely fixed to the table so as to remain stable during surgical manipulation; this can be accomplished by taping the maxilla/mandible to the table with waterproof tape that traverses the upper or lower canine teeth, with the patient in dorsal versus ventral recumbency, respectively (the tape and oral cavity is additionally surgically prepped; see Fig. 27.8B). Finally, the tongue is reflected caudally (into the pharynx) to allow an unobstructed intraoperative assessment of occlusion (Fig. 27.9). Such surgical access and patient positioning complicates anesthetic monitoring, as the routine evaluation of eye position and reflexes and assessment of jaw tone is not possible. Therefore greater reliance is placed on monitoring the heart rate and rhythm, respiratory depth and rate, pulse quality, and blood pressure (see Chapter 4).



• **FIG. 27.8** **(A)** Positioning of a dog with a mandibular fracture in preparation for surgery. The dog is in dorsal recumbency with both forelegs pulled caudally (the forelimb ties also are crossed, which helps stabilize the positioning). The upper jaw is secured near the end of the table with waterproof tape placed over the maxillary canine teeth. The endotracheal tube has bypassed the oral cavity by being placed through a left pharyngotomy. The anesthetic machine is placed at the other (caudal) end of the dog. Sufficient access is obtained by a surgical field that surrounds the entire cranial end of the table. Notice that the tilting mechanism for the table also is under this end of the table so that additional height may be gained by elevating this end of the table. Similar positioning, but in ventral recumbency, is performed for access to the upper jaw. **(B)** Rostral view of a dog with bilateral comminuted mandibular fractures at the junctions of the bodies of the mandibles with the rami. The maxilla has been secured to the table as described in (A) with waterproof tape. In addition to the ventral surface of the mandible, the oral cavity (and the tape) has been prepped for the surgical procedure. Notice that the tongue is not visible (reflected into the pharynx—see Fig. 27.9).



• **FIG. 27.9** Demonstration of tongue position in a dog, dorsal recumbency, with endotracheal intubation through a left pharyngotomy. **(A)** The tongue can be seen resting normally. **(B)** The mouth is opened and the tip of the tongue reflected caudally into the pharynx. **(C)** The tongue can no longer be seen. **(D)** Full closure of the mouth can be obtained in order to assess occlusion; the tongue no longer interferes with this assessment.

Aseptic preparation of the surgical field, including the mouth, is accomplished by routine methods. The eyes must be protected with ophthalmic ointment. In simple fractures, where endotracheal intubation per os is performed, the oral cavity is not included in the draping. In cases where occlusion is to be used to assess the reduction, and the endotracheal tube bypasses the mouth, draping is performed to include full access to the oral cavity. As previously noted, the tongue is reflected back on itself into the pharynx to avoid its interference with intraoperative assessment of dental occlusion (see Fig. 27.8B).

Surgical goals

Proper dental occlusion is the primary objective, which ensures appropriate fracture reduction; however, rigid skeletal fixation is also a necessary adjunct. These two goals are interrelated objectives that cannot be compromised. Malocclusion after fracture reduction and fixation, in addition to adversely affecting function, will result in abnormal leverage against the fixation devices. These abnormal forces likely will result in disruption of the fixation, leading to motion at the fracture site and subsequent loosening of these implants. Bone fragment motion and loose implants will interfere with revascularization and healing, and also contribute to the development of infection. Mandibular fracture stability may be even more important than in other fracture locations because bone fragment motion also will inhibit healing of the oral mucosa. Any gaps in the oral mucosa allow saliva and food particles access to the fracture site, further compromising the healing process and predisposing the area to infection.

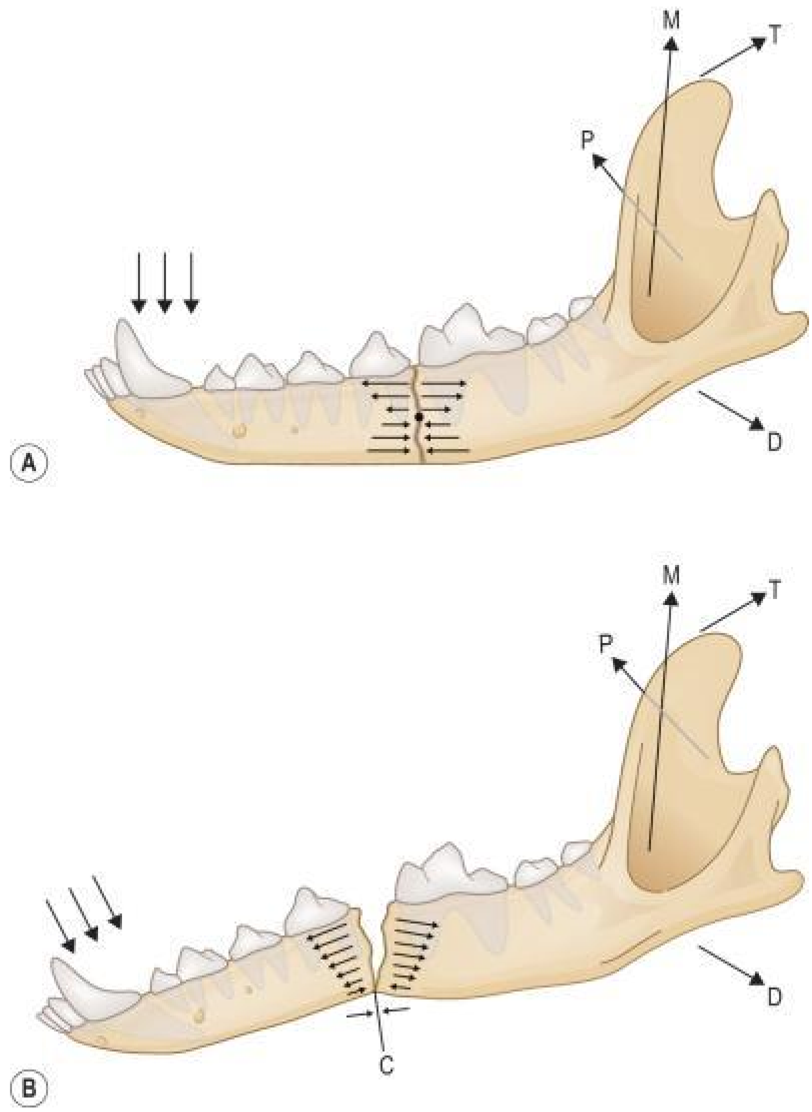
The aims of ORIF are rigid/stable fixation and an early/rapid return to function. These goals are magnified in maxillofacial fracture repair where any failure to attain these objectives results in early complications and difficulty with patient management (i.e., pain, inappetence, infection). Many different techniques and devices are described for this purpose. Uncomplicated healing without infection is obtained only with a full knowledge of the unique biomechanical requirements of this location, and knowledge of both the advantages and limitations of the different methods of fixation. Appropriate reapposition and rigid fixation of bone fragments without further compromise of the blood supply and appropriate management of skin and mucosal lacerations, i.e., judicious soft tissue debridement, create optimal conditions for uncomplicated healing without infection.[20–22] Antibiotic administration is recommended in all cases, as open fractures (which are open to the oral cavity) occur in a majority of these cases and inevitably are contaminated.[23–25] Despite agreement regarding antibiotic use perioperatively, continued postoperative use has been questioned (see Chapter 3).[26]

Biomechanics

The biomechanics of the masticatory system and, in particular, the muscular forces acting on the mandible are of utmost importance in the management of fractures of the mandible. The temporal, masseter, and medial pterygoid muscles are responsible for closing the mouth, and in so doing can generate large occlusal forces. The static force exerted by these muscles also tends to lift the mandible. As the muscular insertions are located on the caudal part of the mandible, this part in particular will be lifted when a fracture of the body of the mandible has occurred; the resultant force exerted by these muscles is in a rostrodorsal direction. The relatively small digastricus muscle, with its relatively small area of insertion at the angle of the jaw, is responsible for opening the mouth. The rostral fragment of a fractured mandible thus displaces in a caudoventral direction. Alternatively, the maxilla is broadly attached to the skull and supports the dental arch with areas of thickened bone (or buttresses) that disperse the forces over a wide area. Application of the fixation devices used for the repair of maxillofacial fractures must include a complete understanding of this functional anatomy. These biomechanical aspects dictate the ideal placement of the various internal fixation devices; furthermore, these factors also play a role in the selection of the most appropriate fixation devices.

Mandible

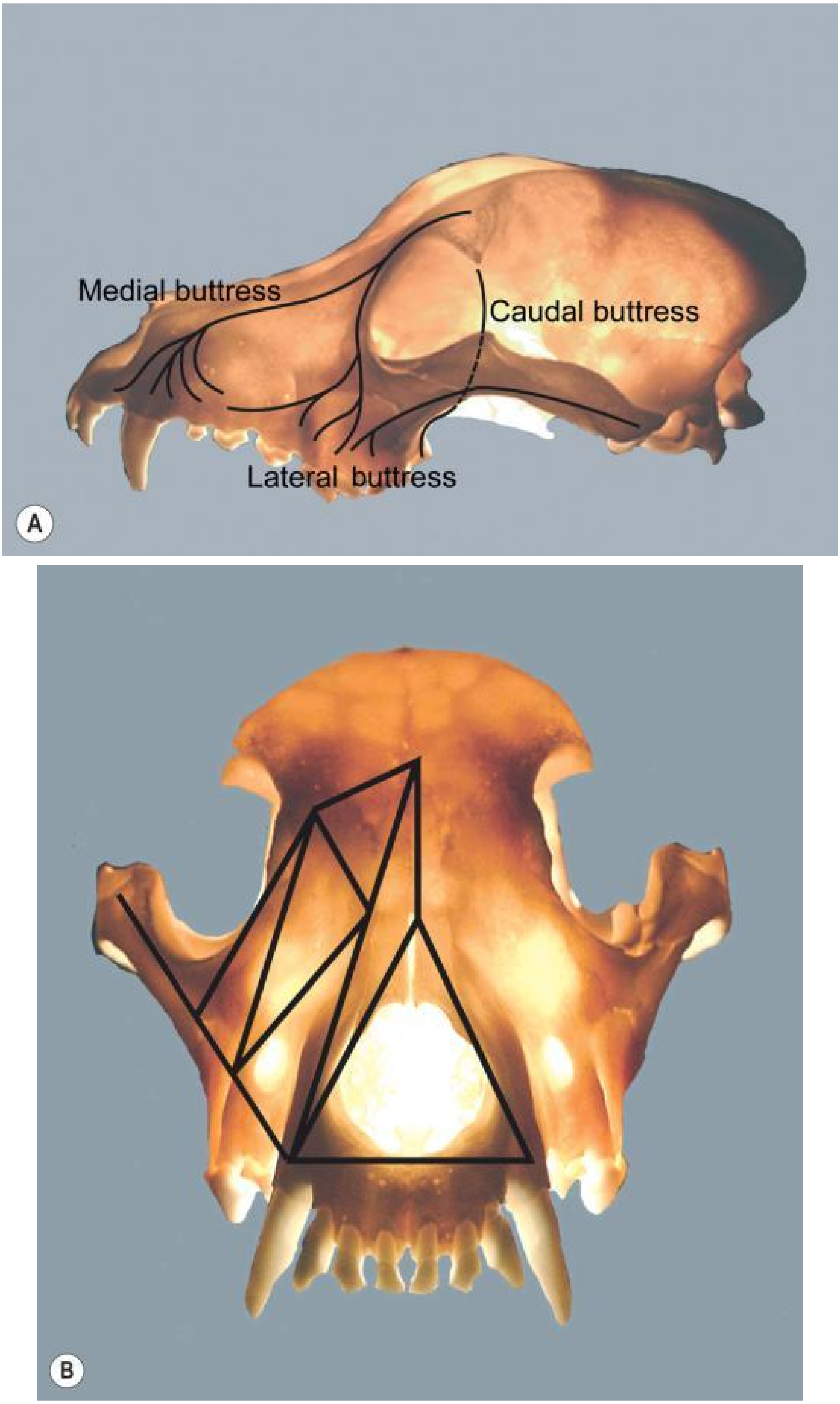
Bending forces are the primary forces acting on the mandible during functional stress (mastication).[27,28] A continuum of tensile to compressive stresses exists from one side of the bone to the other during bending stress.[29] Maximal tensile stresses exist at the oral (alveolar) surface, and maximal compressive stresses exist at the aboral surface.[30] At the ramus, shear forces are maximal, whereas rostrally (symphysis) rotational forces are maximal.[27,28] Distraction of the oral margin therefore will occur after a mandibular fracture and is magnified with any mandibular muscular contraction (Fig. 27.10). The anatomic configuration of a long lever arm with an absence of any further supplemental support is unique to the mandible and must be considered with any fixation method. Therefore application of the fixation should consider both the tension and compression surfaces of the bone. Because fixation devices are strongest in tension (all stresses acting parallel to the longitudinal axis of the implant), they are ideally placed on the tension surface of the bone or, in cases of mandibular fractures, along the alveolar border. The basic biomechanical principle to be considered is tension-band fixation.[31] Depending on the type of fixation to be used, the fixation may not be suitable for application to this location due to interference with tooth roots and neurovascular structures adjacent to the tooth roots. In the dog and cat, the tooth roots encompass approximately two-thirds of the bone adjacent to the alveolar margin, severely limiting the type of implant that can be applied. An alternative approach that may be considered is utilizing a single larger plate along the buccal aspect of the ventral mandibular margin.[32,33]



• **FIG. 27.10** **(A)** Line drawing of the intact mandible demonstrating the normal lines of stress *(small arrows)* through the body of the mandible with muscular contraction and any external force (biting, chewing) applied to the rostral portion of the jaw *(medium arrows)*. **(B)** Bending forces are the primary forces acting on the mandible during functional stress; with a fracture at this level there is distraction along the alveolar surface of the bone; the point *C* shows that the ventral portion of the bone is the only area where compressive stresses exist, if there is contact. (P,M. pterygoideus; M,M. masseter; T,M. temporalis; D,M. digastricus.) Source: (From Boudrieau RJ, Mandibular and maxillofacial fractures. In: Johnston SA and Tobias KM, eds. *Veterinary Surgery: Small Animal* (2nd ed), Elsevier; St. Louis, 2018, with permission.)

Maxilla

The term *maxilla* is used here to refer to the incisive, palatine, zygomatic, lacrimal, frontal, and nasal bones, in addition to the maxillary bone proper. The forces exerted on the mandible are also exerted on the maxilla. However, the distribution of these forces is very different, and it is generally accepted that the maxilla is subject to much less strain. The maxillofacial area can most easily be thought of as an "outer facial frame," which acts as a link between the base of the skull and the occlusal surfaces.[34] The support of the facial region is provided by a series of anatomic buttresses that distribute the masticatory forces to the head. These buttresses exist in the horizontal, vertical, and coronal planes.[34–37] There are three primary buttresses: rostral (medial), lateral, and caudal (Fig. 27.11).[34–37] These buttresses also can be defined anatomically: the rostral (medial) as the nasomaxillary buttress, the lateral as the zygomaticomaxillary buttress, and the caudal as the pterygomaxillary buttress. The anatomic definitions mirror the bones of the skull that comprise these buttresses. The caudal buttress (which is not readily accessible) is composed of the lacrimal, palatine, and pterygoid bones. In the presence of a fracture, the facial frame can be adequately reconstructed with two of the three buttresses: medial and lateral (see later). The incisive bones are not part of the buttresses and therefore may not need to be stabilized, as this area generally does not provide essential support to the skull, or it can effectively be treated by other means (see Chapter 30). Fixation of these areas still may be useful in either providing the support for the incisor teeth or reestablishing the cosmetic appearance of the nasal area when depressed fractures occur.[6] Similarly, maxillary fractures often may not require stabilization unless the buttresses are disrupted.[5] In the latter case, occlusion or support of the orbit is compromised, and fixation (most often miniplate) is ideally suited to stabilize these fractures and reestablish the medial buttress. Likewise, if the lateral buttress is compromised, which often will affect the orbit, plate fixation is ideally suited for stabilization (Chapter 33).[5,6]



• **FIG. 27.11** **(A)** Right lateral and **(B)** cranial view of the skull, which is transilluminated to reveal the buttressing. The lines drawn indicate a pyramidal arrangement (B). The nasomaxillary (medial) buttress is seen rostrally, and the zygomaticomaxillary (lateral) buttress is seen caudally; the pterygomaxillary (caudal) buttress is shown with the dashed line (A). Source: (Modified from Boudrieau RJ. Fractures of the maxilla. In: Johnson AL, Houlton JEF, Vannini R, eds. *AO principles of Fracture Management in the Dog and Cat.* Stuttgart: Georg Thieme Verlag, 2005;116–129.)

An evaluation of the facial support of these buttresses reveals that they are designed as a basic "truss" or "frame," which is a triangle; in three dimensions the basic truss is a tetrahedron (three-sided pyramid)—four triangles that can resist distortion in any direction three-dimensionally.[34] In humans, the vertical buttresses of the midface are clinically most important with regard to the management of midface fractures.[35,36] These buttresses are divided into three principal areas, as previously noted: nasomaxillary (anterior or medial) buttress, zygomaticomaxillary (lateral) buttress, and pterygomaxillary (posterior) buttress.[35–37] These areas all represent thicker bone designed to support the maxilla in humans in the vertical dimension. These buttresses are quite strong in resisting vertically directed stresses; however, they cannot withstand forces of similar magnitudes in the transverse plane. There is no similar detailed description of the maxillary buttresses in the dog or cat; however, a similar anatomic arrangement to the human skull has been suggested (see Fig. 27.11).[5] These trusses can be visualized in the skull as pillars of reinforced bone. In the dog, the medial and caudal buttresses provide similar vertical support as in humans; however, due to the configuration of the skull, these buttresses are better designed to also withstand transverse forces as compared with humans. The lateral buttress primarily functions to withstand forces in the vertical plane. It also further supports the other two buttresses so as to better withstand the increased shearing forces in the premolar–molar region in these species. The lateral buttress in the dog also appears to more effectively neutralize forces in the transverse plane, despite lacking the secondary lateral support adjacent to the orbit (orbital ligament in the dog and cat as compared with bony support in humans, the latter of which also contributes to the lateral buttress). The attachment of the maxilla to the base of the skull therefore appears to have greater bony support in dogs and cats as compared with humans.

Surgical approaches

Treatment planning

Occlusion must be used to determine the accuracy of the fracture reduction in comminuted fractures or fractures with gaps. Simple fractures may be reconstructed anatomically. Performing anatomic reconstruction without relying on occlusal evaluation should be performed with caution, especially in comminuted fractures, as it is not unusual to have a malocclusion despite what appears to be a "perfect" reconstruction on the accessible outer (nonalveolar) bony surface. In most instances the mandible is repaired first, which serves as the "base" for subsequent facial repairs. Ideally, the mandible should be repaired from a caudal to rostral direction. After mandibular reconstruction has been completed, the maxilla is then addressed, concentrating first on the lateral and then on the medial maxillary buttresses as needed. Performing temporary maxillomandibular fixation provides for appropriate occlusal realignment, which serves as a further template for the reconstruction, and also provides some degree of stabilization during application of the definitive fixation. Securing the medial and lateral buttresses in this manner permits exact rostrocaudal positioning of the maxilla and also corrects height and projection. Direct exposure of all fractures in order to ensure accurate anatomic realignment of all bone fragments, and the subsequent appropriate application of implants for proper reconstruction of the entire face, cannot be overemphasized (see Chapter 29).

It may not always be possible to reestablish the buttresses with the placement of implants, due to the degree of comminution or bone loss. In these instances, a reasonable approach to surgical planning and fixation is to consider four factors. First, unstable bone fragments should be secured to surrounding stable bone. Second, reconstruction of the orbit is a priority; therefore fractures that compromise the attachment of the orbital ligament will benefit from internal fixation. Third, fractures affecting the nasofrontal vault often lead to depression of the fragments into the nasal cavity or frontal sinus, which, if left unrepaired, may lead to sequestra formation and chronic nasal discharge. Reconstruction of the nasofrontal vault is therefore also a very important surgical objective. Lastly, as with all craniomaxillofacial fracture repair, restoration of occlusion is always a priority.

Mandibular fractures

Although the fractured bone fragments may be visualized within the mouth due to the extensive lacerations of the mucosa that usually accompany these fractures, separate ventral mandibular approaches are the preferred surgical access for internal fixation (see Chapter 29). The ventral approach to each mandible facilitates exposure and bone fragment manipulation, including the ability to perform an accurate reduction and stabilization.

A separate lateral approach is required to expose the temporomandibular joint (see Chapters 29 and 37). The mandibular ramus dorsal to this joint typically does not need not to be repaired.

Maxillary fractures

As a general rule, approaching fractures through a traumatic wound should be avoided. Skin incisions generally are made directly over the fracture in nasal, maxillary, or frontal areas (see Chapter 29). Dorsal midline incisions may be best to avoid neurovascular structures along the nose and most easily expose the maxillary buttresses (Fig. 27.12).[5,6]

• **FIG. 27.12** A dorsal midline incision has been made to allow full access to the maxillary buttresses in order to apply internal fixation in this dog with multiple comminuted fractures of the nasal and maxillary bones. This approach also was combined with a direct gingival approach next to the alveolar bone to provide additional exposure to the lateral buttresses bilaterally.

Summary

Maxillofacial trauma sufficient to result in fractures generally results in gross and usually severe patient disfigurement and pain, typically results in functional disturbances (i.e., inability to eat and/or drink), and may even cause respiratory issues due to possible upper airway obstruction. These fractures are exceptionally rewarding cases to treat, often resulting in a successful functional outcome within 24 hours after fracture stabilization. Once posttraumatic inflammation and edema resolve, a markedly improved cosmetic appearance follows.

The primary principles of fracture treatment (i.e., restoring occlusion and providing stable fixation to the bone fragments) may be successfully used through an appreciation and proper application of the most appropriate implants and techniques. Knowledge that bending forces (divided into their tensile and compressive components) are the primary forces to be neutralized in mandibular fractures generally indicates the preferred selection and application of fixation devices. Similarly, an understanding of the maxillary buttresses is also of importance for the fixation methods applied to these areas.

Successful treatment is predicated on obtaining a functional and cosmetic result. Anatomic reduction and rigid fixation of fractures that can be reconstructed create optimal conditions for uncomplicated healing. Fractures in which bone loss or severe comminution exists, and which cannot be anatomically reconstructed, must be reduced using dental occlusion as the template for fracture fixation, thereby avoiding postoperative malocclusion. The techniques employed for repairing these cases include bridging fixation (see Chapter 2 and 33).

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CHAPTER 28

Facial soft tissue injuries

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Definitions

Debridement: The excision of damaged and devitalized tissue and foreign material from a wound.

Axial pattern flap: A pedicle flap of skin and subcutaneous tissue that incorporates a direct perforating cutaneous artery and vein into its base.

Rotation flap: A semicircular flap of skin and subcutaneous tissue that moves about a pivot point by a combination of rotation, transposition, and stretching into a defect; it is particularly useful for triangular-shaped wounds.

Single-pedicle advancement flap: A flap of skin and subcutaneous tissue that is mobilized by undermining and advanced into a defect without altering the plane of the pedicle; it is particularly useful for square and rectangular-shaped wounds.

Transposition flap: A flap of skin and subcutaneous tissue, generally rectangular in shape, that is turned on a pivot point to reach an adjacent defect to be covered, which is usually at a right angle to the axis of the flap; it is particularly useful for square and rectangular-shaped wounds.

Preoperative management of facial wounds

Facial soft tissue wounds are typically caused by blunt or penetrating trauma, often with significant shear forces, which can result in contaminated, devitalized, and avulsed tissues in the wound. Other, less common causes of wounds include thermal burns, radiation burns, actinic dermatitides, envenomation, idiosyncratic drug reactions, and idiopathic vasculitides causing sloughing. Management of maxillofacial tumors is covered in Section 9; removal of tumors of the lip or cheek may result in a defect that could be repaired using techniques described in this chapter.

Early administration of prophylactic, systemic, broad-spectrum antibiotics is indicated for all traumatic wounds during initial debridement and lavage. The need for continuing antibiotic therapy will depend on the nature of the wound and the health of the patient. The antibiotic selected should be effective against the bacteria expected in the wound (see Chapter 3).

Upon presentation of a patient with acute trauma to the face, it is critical to evaluate the cardiovascular and neurological status of the animal for concurrent injuries (e.g., concussion, skull fractures, nasal bone fractures, and diaphragmatic hernia). Eyes and ears should also be closely examined (with an otoscope and ophthalmoscope), patency of the nares assessed, and integrity of the hard palate and other intraoral structures determined. Some of these examinations, such as the oropharynx, are more easily accomplished when the animal is sedated or anesthetized for initial wound management.

The goals of wound management are to remove devitalized tissues and foreign material, decrease bacterial contamination, and optimize the wound environment to a stage where it can either undergo a reconstructive effort or heal by second intention. All traumatic wounds are considered contaminated and will become infected if the bacterial inoculum is allowed to invade and colonize live tissues. Thus the earlier wounds are addressed, the lower the chances of infection. Clipping, cleansing, debridement, and liberal lavage should be performed in all facial injuries, typically under general anesthesia. Debris, clots, and nonviable tissue should be meticulously removed, using color and underlying attachment as guides. Tissues that are very dark or very white or that are significantly separated from underlying tissue should be removed. Tissues of questionable viability, such as avulsed lips or red-purple bruised tissue, can be cleansed and left for reevaluation the following day as part of a staged debridement technique, particularly if that tissue may be needed later to close the wound or preserve facial architecture. Due to the excellent blood supply to the region, facial tissues have a remarkable ability to remain viable in the face of insult. Copious wound lavage is best undertaken with sterile isotonic saline, balanced electrolyte solution, or a 0.05% aqueous solution of chlorhexidine gluconate, but tap water can also be used.[1,2] Lavage should be delivered under 7 to 8 psi, a moderate pressure that removes microdebris and decreases bacterial burden without causing trauma. This can be achieved by placing a liter bag of lavage fluid in a pressure cuff, pressurizing it to 300 mm Hg, and delivering the lavage via a standard intravenous administration set and needle. Needles directly attached to syringes may create much higher pressures (17 to 40 psi) and may increase risk of barotrauma.[3,4] The airway is protected during lavage by using a cuffed endotracheal tube and packing a known number of gauze sponges in the pharynx around the tube. The eyes should also be protected, especially if using chlorhexidine.

Wounds in the inflammatory phase of healing will be highly exudative and will require a highly absorbent dressing with frequent changes and wound lavage. Dressing retention on the face can be challenging in dogs and cats. Securing the dressing with several skin staples or loop sutures and a tie-over bandage will help maintain position for several days. An Elizabethan collar is mandatory to prevent self-mutilation. Due to the difficulty in maintaining dressings on the face, and the requirement for repeated anesthesia for dressing changes, reconstruction is often undertaken early, as soon as the wound is considered healthy and converted to a clean-contaminated state. This is often still in the inflammatory phase, within the first week.

Surgical anatomy

The mucosa of the upper lip is more generous than that of the lower lip, which becomes more narrow rostrally. However, the lower lip is easier to mobilize rostrally than the upper lip. The lower lip has a firm attachment (the mandibular frenulum) to the gingiva between the mandibular canine and first premolar tooth, which prevents the lower lip from sagging. It is important to restore this attachment when reconstructing this area. Between the inner mucosa and the skin of the lips and cheeks are two layers of muscles—the superficial orbicularis oris and the deeper buccinator. There are many superficial facial muscles in this area: the levator nasolabialis, caninus, levator labii maxillaris, zygomaticus, sphincter colli profundus-pars palpebralis, frontalis, and platysma. These muscles are critical for facial expression in humans but not as important for dogs and cats. The blood supply to the lips and cheeks (and, to a lesser extent, head) is bountiful. The facial artery divides to supply the cheeks through the superior and inferior labial and the angularis oris arteries. The high-pressure infraorbital artery comes directly off the maxillary artery (the major terminating branch of the external carotid) and supplies a rich arterial network of blood to the upper lip and muzzle. The head is mostly supplied from the branches of the superficial temporal artery, which is the minor terminating branch of the external carotid. There are a half-dozen branches off the superficial temporal that cover most of the head. This excellent arborization and collateral circulation makes it easier to develop flaps with a good chance of surviving.

The facial nerve emerges from the stylomastoid foramen immediately caudal to the external acoustic meatus of the tympanic bulla, runs cranially over the lateral side of the horizontal ear canal, and branches dorsally as the auriculopalpebral nerve to the ear and to the eyelid and cranially as the buccal nerves. The path to the palpebral branches is particularly important to preserve if possible, since these nerves innervate the orbicularis oculi muscle and stimulate blinking.

The surgeon should also be aware of the position of the salivary glands and ducts, especially the sublingual duct and the parotid duct (Chapter 54).

Surgical principles

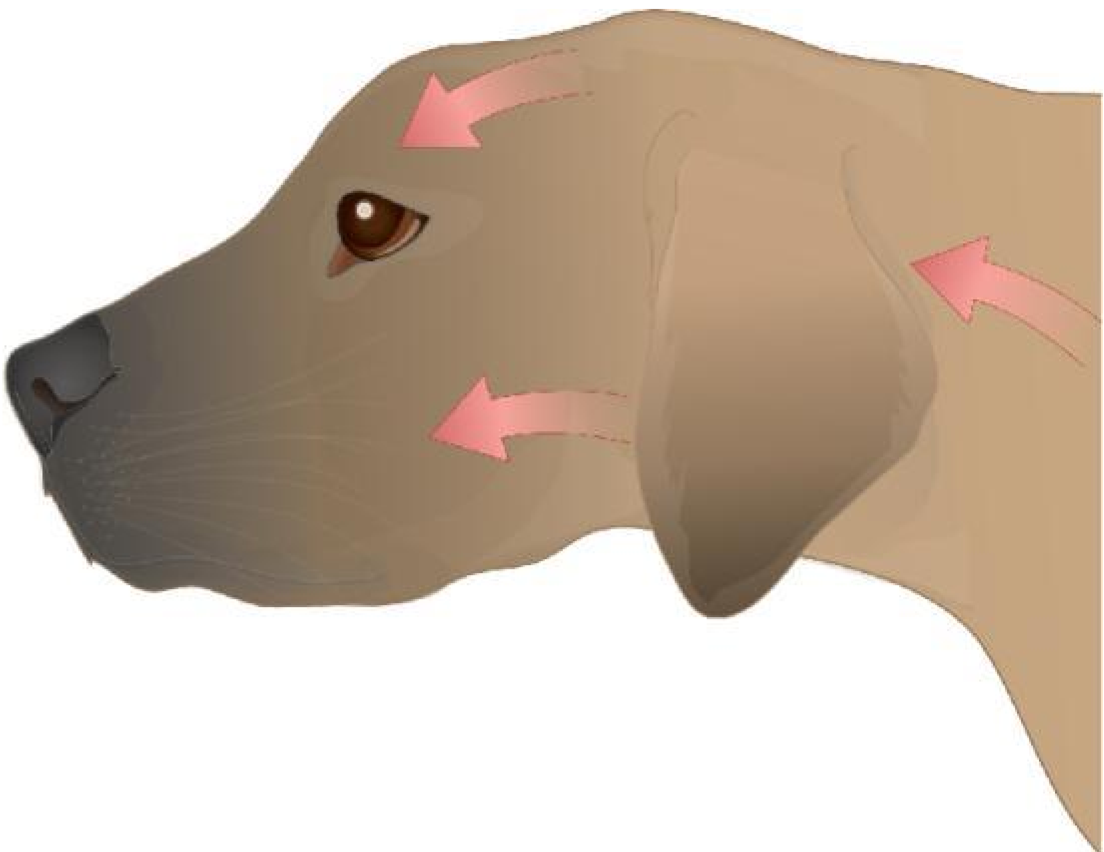
We look at our pets' faces more than other areas, and even if they are not as facially expressive as humans, cosmetic outcome may be of greater concern to the owner compared to other areas of the body. From the animal's perspective, however, *restoration of pain-free function* is probably the most important requirement. To some extent, cosmesis and function are complementary, such as restoring lip continuity—it is beneficial both for function (holding food within the oral cavity) and appearance. Although we need to manage the owner's expectation of the final outcome, most of our patients have the advantages of hirsutism to hide scars and an elongated muzzle that minimizes perception of facial asymmetry.

When repairing facial soft tissue injuries, several principles should be kept in mind.

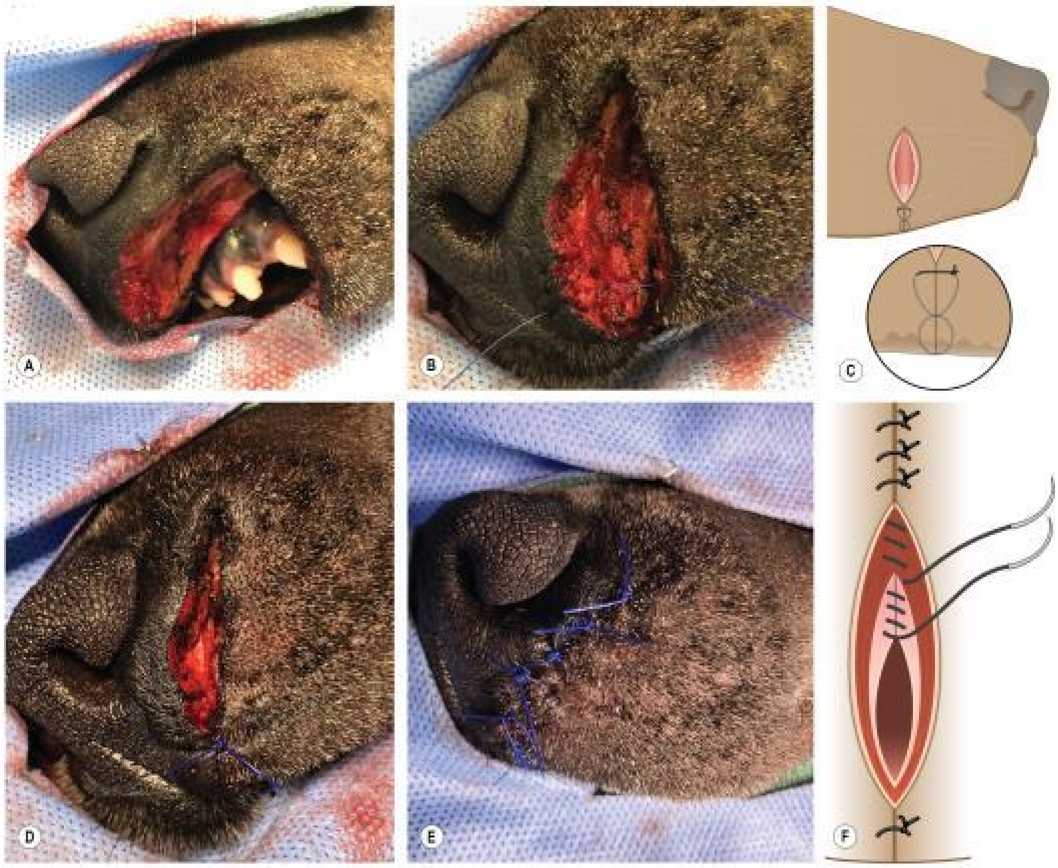
- Due to the sparsity of soft tissue on the rostral area of the face, reconstruction requires moving soft tissue from caudal to rostral (Fig. 28.1).
- Major reconstruction of facial trauma will require strong anchorage to the gingiva and bone. Soft tissues, including viable skin, can be effectively affixed back to the skull and mandibles by transosseous placement of wires and sutures.
- Restoration of lip integrity, including accurate alignment of the lip margin, is critical as the first step of reconstruction and is best achieved with a figure-of-eight suture (Fig. 28.2).
- The superficial muscles of the face should be incorporated into any reconstructive effort, as they will add bulk, protection, and optimize perfusion of the skin. This principle also applies to the superficial temporal muscle of the head and the platysma

muscle of the neck; these muscles contain the deep subdermal plexus and must be included when elevating skin flaps in these regions.

- All closures should be a minimum of two layers—synthetic absorbable continuous suture layer(s) in the subcutaneous/submucosal tissues and interrupted sutures in the skin and mucosa.



• FIG. 28.1 Repair of facial wounds requires moving soft tissue *(arrows)* from caudal, where skin is more abundant *(light area)*, to rostral, where skin is less abundant *(progressively darker shading)*.



• **FIG. 28.2** (**A**) A 'figure-of-eight' placed as the initial suture at this lip margin will ensure accurate alignment. (**B**) "Figure-of-eight" suture being placed according to technique in inset (**C**). (**D**) "Figure-of-eight" suture has been tied. Applying downward pressure to the 'figure-of-eight' suture will even up mild disparity of the wound edges, thus facilitating remaining suture placement. (**E**) Remaining two suture layers (mucosa/submucosa, muscle and skin) have been placed, shown also in inset (**F**). Source: (C and F after Swaim SF, Henderson RA. Small animal wound management. 2nd ed. Baltimore: Williams and Wilkins, 1997; page 192, with permission.)

In addition to the above principles, the following information is also important to keep in mind.

- The surgeon should be aware of the location of the salivary ducts (Chapter 54). When repairing wounds caudal to the maxillary fourth premolar tooth, the parotid papilla should be located and the parotid duct cannulated with a lacrimal needle, an over-the-needle catheter, or a large suture to facilitate preservation of the duct during dissection, if possible. Alternatively, the duct can be moved to a safe spot by transecting it as distally as possible, spatulating the end, and suturing it to a new mucosal incision with 5-0 to 6-0 absorbable material. If it cannot be preserved, it should be ligated proximal to the lesion.[5,6]
- Due to their unstable nature, lips and lids can be challenging to incise accurately. Stabilizing and stretching the lip or lid onto a firm sterile surface (e.g., wooden tongue depressor, folded drape, or Jaeger lid plate) will facilitate clean, accurate cutting.
- Proposed incision lines should always be drawn with a sterile marker, and flap dimensions should be measured with a sterile ruler to ensure they will adequately cover the recipient wound, before commencing surgery.

- In highly mobile areas such as the lip commissures, there can be significant shear forces on the repair. In addition to the layered repairs discussed below, commissure closures can be further reinforced with a stent.
- Mucosal grafts from the sublingual area and inside of the upper lips and the cheeks can be used to reconstruct the nares and vestibule.
- If considerable dead space is created when raising a flap to close a wound, a drain (preferably a closed-suction drain such as a Jackson-Pratt) may be placed under the flap before skin closure. The drain is removed when fluid is serosanguineous and quantity has decreased and plateaued.
- In the event of uncontrollable hemorrhage from the head or face, one or both carotid arteries can be ligated in an attempt to control the bleeding. Ensure that the carotid artery is separated from the vagus nerve during this procedure, as they share a common sheath.

Surgical techniques

Minor lip lacerations

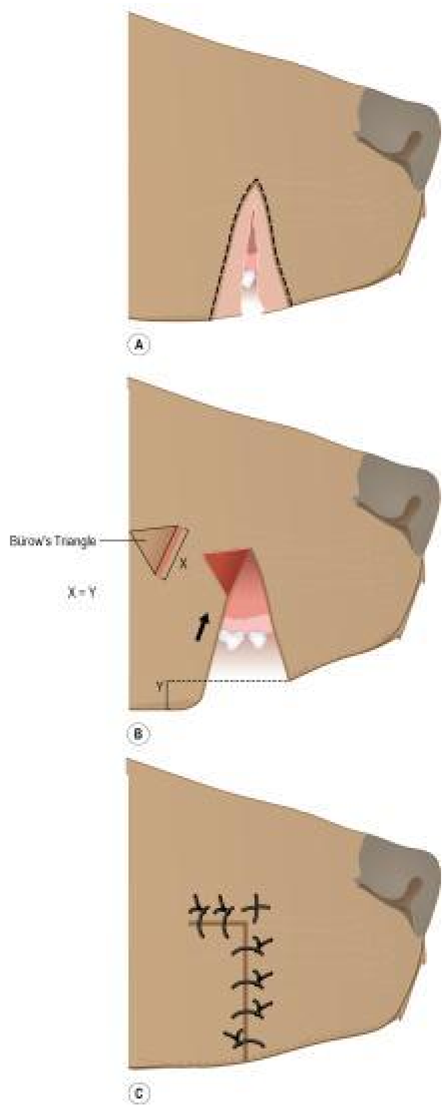
Direct closure

In dogs with large pendulous lips, one-third to one-half of each lip's length may be resected with direct apposition of the wound edges and no resulting deformity or malfunction.[6] Lip lacerations and defects with a minimal amount of tension between the two sides should be sutured in layers, with the number of layers dependent upon the lip thickness. A "figure-of-eight," vertical mattress, or near-far suture should first be placed at the lip margin for perfect alignment (Fig. 28.2). Four layers (mucosa or submucosa, muscle or muscle fascia, subcutis, and skin) may be necessary for closure on a thick lip.[7] Small animals with thin lips may require fewer layers, but a minimum of two layers (mucosa/submucosa and skin) should be used. Once the lip margin integrity has been restored by the first "figure-of-eight" suture, the remaining layers can be apposed, from oral mucosa to skin, using the lip margin suture as a stay suture, applying a little tension to line up the edges evenly. Alternatively, the lip margin suture can be initially kept loose to provide easier access to deep layers, tightening it periodically to make sure alignment is being maintained.

When suturing oral mucosa, care should be taken to avoid mucosal inversion, which can interfere with healing.[6] A submucosal suture pattern will minimize mucosal inversion. Additional suture layers can be placed in the fascial/muscle layer to help align the skin edge and minimize tension on the closure.[5–8] The appropriate types and sizes of suture material for facial surgery are listed in Chapter 8. Interrupted or simple continuous suture patterns can be used in the deeper layers, as deemed appropriate for the patient. A simple interrupted or cruciate pattern is advisable in the skin to decrease the risk of an entire incisional dehiscence if the patient self-mutilates the site. Interrupted fine monofilament stainless steel sutures in the skin may discourage licking at the surgical site around the lips.[7]

Wedge conversion

Lip trauma may result in irregular defects necessitating conversion into a wedge shape. During debridement, shaping the defect into a wedge will facilitate a smooth closure. The base of the wedge is at the lip edge and the apex points away from the lip edge. When debriding wedge-shaped lip defects, avoid creating a narrow portion of lip, as it could compromise perfusion and result in lip edge necrosis and dehiscence. Minor disparity of the wound edges can be minimized by placing mild tension on the lip margin suture during suturing to equalize the wound edges. Significant disparity of the wound edges may be corrected by removing a triangle of tissue (known as a Bürow's triangle) from the longer edge near the wedge apex. Closure of the defect is as stated above for direct closures (Fig. 28.3).[7]



• **FIG. 28.3** Wedge closure of lip defect. (**A**) Lip laceration with a flap to be debrided *(broken line)* to create a wedge-shaped defect. (**B**) Significant disparity of wound edges corrected by removing Bürow's triangle from longer lip edge and shifting tissue *(arrow)* for closure. (**C**) Wound closed.

Y-closure

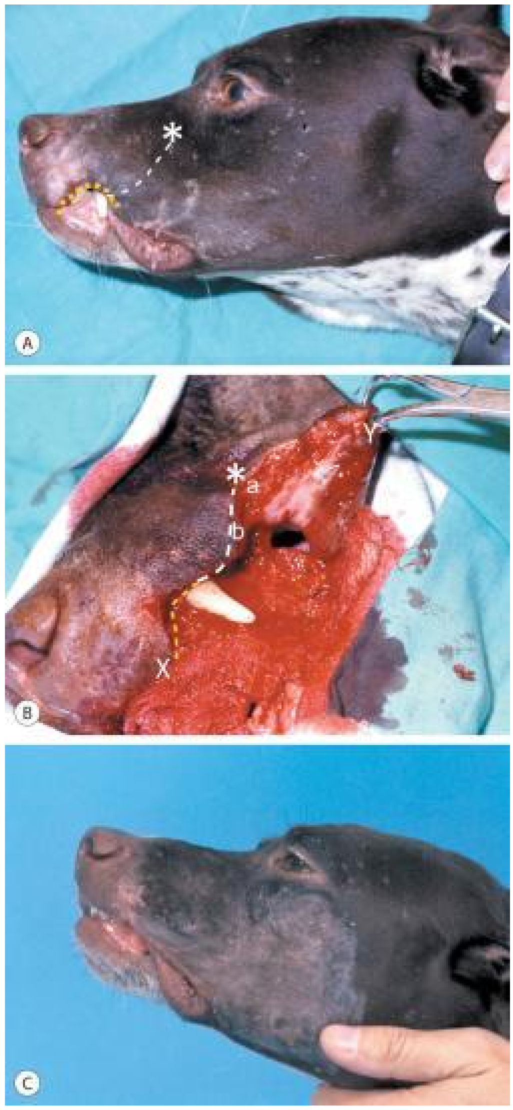
A rectangular lip defect may be closed in a similar manner as a laceration or wedge defect, except that after the initial lip edge-approximating suture is placed, closure of the deeper layers of the lip begins in each of the upper corners of the defect, progressing ventrally, with a resulting Y-shaped closure of the deep tissues and skin (Fig. 49.5).[5,6]

Major lip defects

Because of the size, location, and/or cicatricial tissue around a lip defect, it is often necessary to create various types of flaps for lip reconstruction. In general, all of these flaps entail moving tissue from caudal to rostral for reconstruction.

Rotation flap

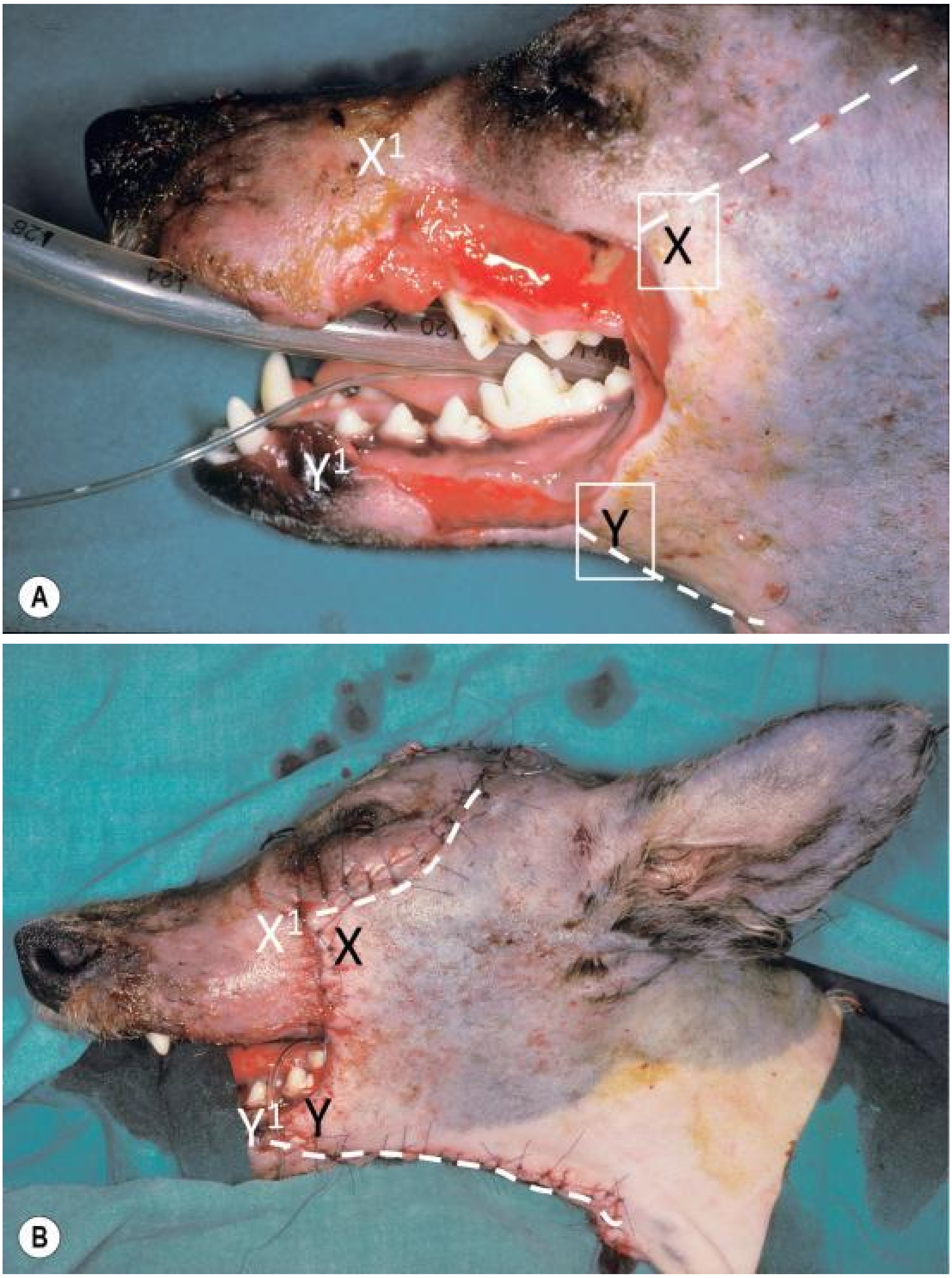
Rotation flaps are useful for large triangular defects in the upper lip.[5,6,8] A full-thickness rotation flap is almost semicircular in shape, with the arc of the flap being up to four times the size of the lip defect. With upper lip defects, the rotation flap is made dorsal to the level of the junction of labial and gingival mucosa. As the incision progresses caudally, the parotid papilla should be identified, and the parotid duct cannulated and preserved, relocated, or ligated as described previously (and in Chapter 54). The flap is rotated rostrally to close the defect. The first suture is pre-placed from the rostroventral tip of the flap to the rostroventral tip of the defect to align the lip edge. There may be too much tension to tie this suture initially, but it serves as an alignment guide as the flap is sutured to the defect. The flap is advanced using “walking” sutures in the strongest available tissue (e.g., subcutaneous, muscle, or fascia layer). The first bite of the first walking suture is placed near the base of the flap. The second bite is taken a short distance rostrally in the dorsal edge of the defect. When tied, the suture advances the flap rostrally a small amount. Subsequent sutures are placed in like fashion to advance the flap into position and minimize tension on the final suture line (Fig. 28.4).



• **FIG. 28.4** Rotation flap to close a rostral upper lip defect. (**A**) Lip defect *(orange dotted line)* with incision line *(white dashed line)* for flap extending dorsocaudal to the level of the junction of labial and gingival mucosa (*). (**B**) Flap incised and reflected. The first suture will be placed between X and Y to align the lip edge. The first bite of the first "walking" suture will be placed at (a) and the second bite of the first "walking" suture will be placed at (b), rostral to the first bite. (**C**) Healed defect, closed by advancing tissues with "walking" sutures.

Single-pedicle labial advancement flap

A full-thickness, single-pedicle, labial advancement flap can be used to reconstruct large rostral square or rectangular defects in the upper lips (Figs. 28.5 and 28.6 and Fig. 49.6).[5–8] These are defects involving one-third to one-half of the lip and may involve transecting the infraorbital vessels and nerve when elevating a large flap.[8] When incising upper lip mucosa, a 5-mm strip of mucosa is left along the gingival border for better suture-holding strength.[7] Initially, a 2.0- to 5.0-cm length of mucosa is incised along the proposed line of the single-pedicle advancement labial flap to determine if adequate tension relief is gained to close the defect without a flap.[7] If not, the mucosal incision is continued through all remaining layers, including the skin, incising parallel to the lip edge from the dorsal limit of the defect to a point caudal to the oral commissure. Subdermal plexus flap guidelines to ensure adequate blood supply state that the long side of the flap should not be more than twice the length of the short side of the flap, although this varies with flap location.[5,8,9] For example, due to the incorporation of the superior labialis artery and vein, this flap can be extended to a length of about three times the width. Identification and preservation of the parotid duct should be done as previously described. Tension bands of tissue can be palpated as the flap is advanced and are carefully divided deep at the flap base, avoiding damage to the flap vasculature. The rostrodorsal corner of the flap can be trimmed for better apposition of this area into the rostral aspect of the labial defect. The lip margin apposition suture is placed, followed by the submucosal suture line and the labial muscle/fascia suture line. A continuous subcutaneous suture layer is placed, followed by interrupted skin sutures.



• FIG. 28.5 A full-thickness single-pedicle advancement labial flap to close a traumatic defect of the lips. (**A**) A large rectangular upper labial defect with a similar smaller lower labial defect. A single-pedicle advancement flap was created by incising along the *white dashed lines*. (The dorsal line deviates more dorsally than typically needed because it was tied in with another part of the repair for this dog). (**B**) After undermining the flap between the white dashed lines, point X was advanced to X^1 and point Y was advanced to Y^1.

• **FIG. 28.6** A bilateral rostral labial defect in a dog is closed with bilateral single-pedicle labial advancement flaps (H-plasty). (**A**) *Dotted line*, drawn parallel to the lip edge, indicates planned incision for left single-pedicle labial advancement flap. (**B**) The dorsal borders of the left and right flaps were incised caudally to a point where the rostral edge of the flaps could be sutured together without tension. An aligning suture was placed rostroventrally to reconstruct the lip margin. (**C**) A mattress suture aligning the dorsal submucosal tissue of both flaps and connecting them to the remaining gingival submucosa is placed. (**D**) Appearance of the repair immediately postoperatively.

A similar labial advancement flap can be used for a lower labial defect, incising the mucosa first in a similarly staged fashion to the upper lip, then the remaining layers if needed to create the necessary flap length. "Walking" sutures, as described for the rotation flap, can also be used in the fascia to advance the flap into position. Mucosal sutures are placed at the level of the mandibular frenulum at the mandibular canine and first premolar teeth to help prevent lip sag (Fig. 49.12).[5]

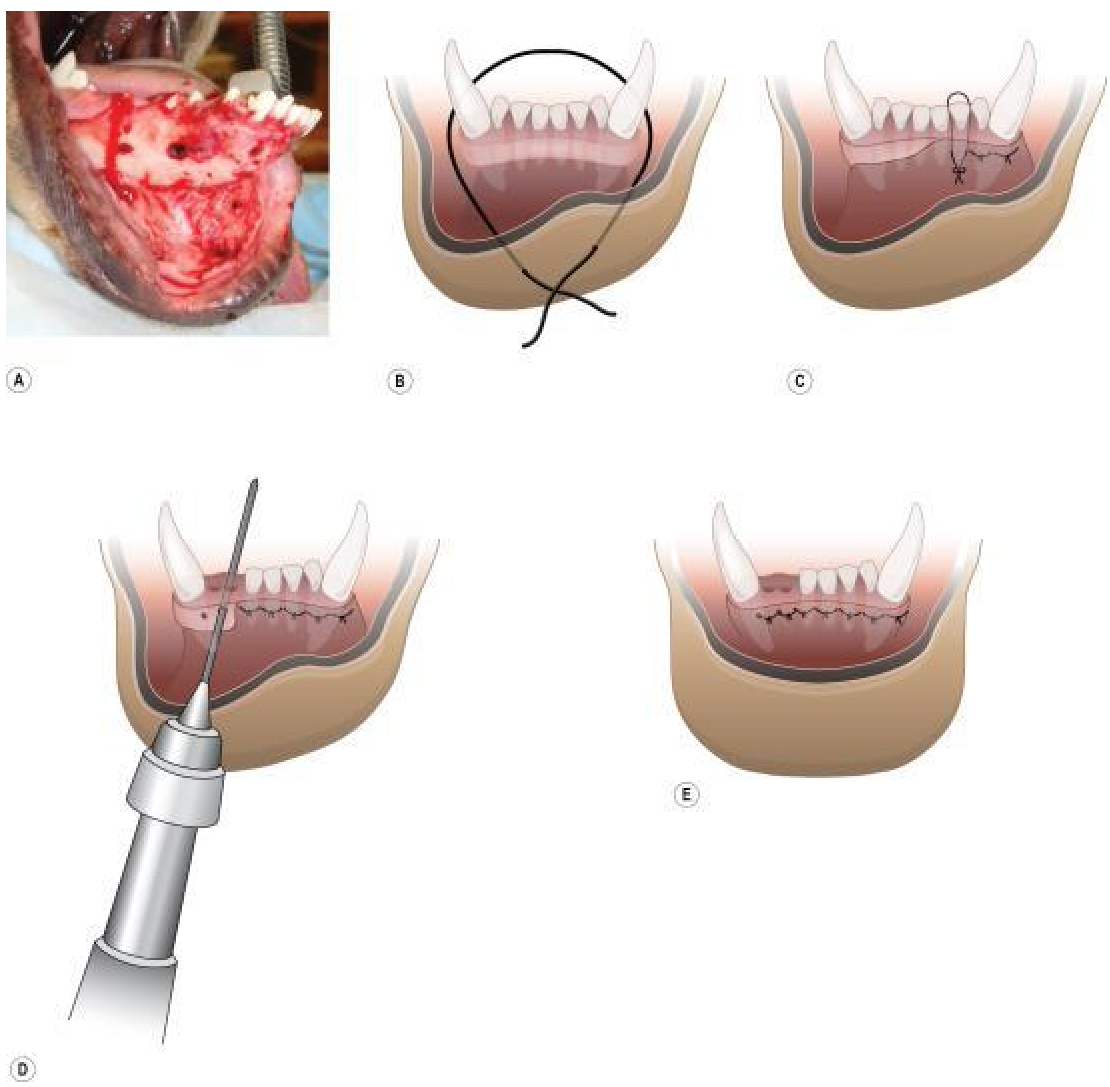
Buccal or commissure rotation flap

For very large upper lip defects, a buccal or commissure rotation flap (lower labial pedicle rotation flap) can be used for correction.[7,8,10] This technique takes advantage of the abundant cheek pouch present in many dogs to create a full-thickness lip flap. The principles of a full-thickness single-pedicle advancement labial flap are followed. However, the base of the flap is more ventral and the labial edge of the flap directly includes the oral commissure (Fig. 49.8). This technique will result in significant rostral advancement of the commissure of the lips.

Avulsion of the lower lip

Avulsion of the lower lip from the mandible caused by shear forces on labial and gingival mucosa as well as subcutaneous tissue can result in separations of varying severities. They range from minor unilateral tissue separation to major bilateral avulsion extending to the oral commissures. Usually the avulsion occurs along the mucogingival junction, leaving only the mucosa and gingiva in the interdental space with which to reattach the lip, and this can present challenges.[6] The degree of tissue separation governs the technique for repair.

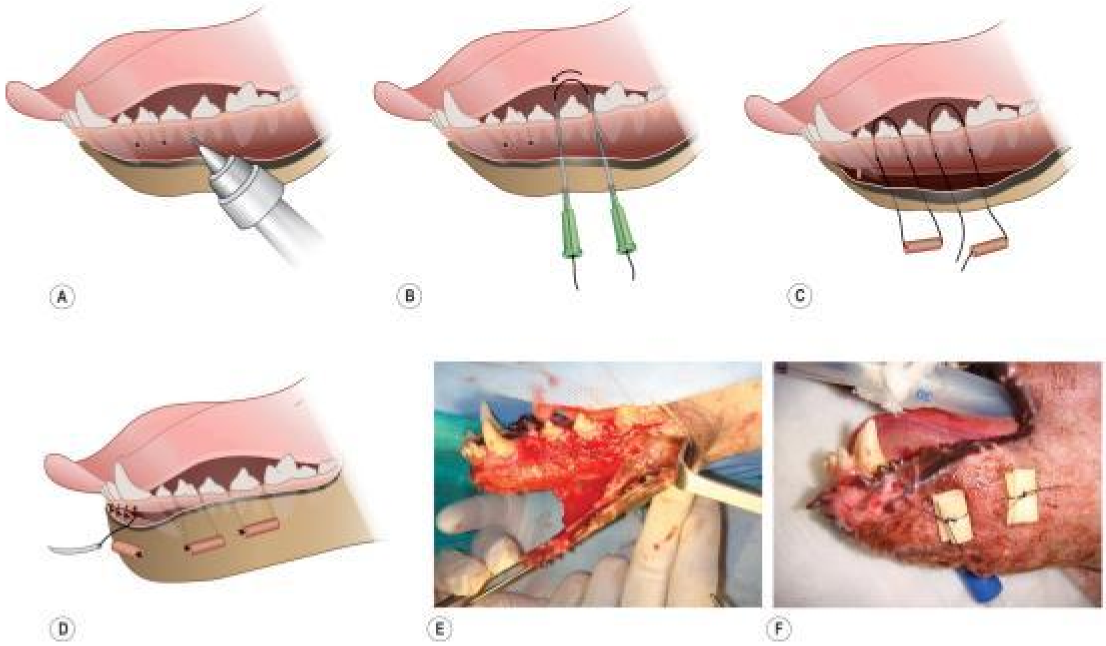
With minor lower labial avulsion, the soft tissue flap can be replaced immediately after cleaning and debridement in many cases. The soft tissues can be kept in contact with the bone using a single, large, monofilament, horizontal mattress suture passed through the skin of the chin and around the canine teeth. This suture is tied fairly loosely on the chin, avoiding further compromise of the blood supply to the tissue flap, while still achieving approximation of the tissues. No additional mucogingival sutures are necessary with this technique (Fig. 28.7A, B).[11]



• **FIG. 28.7** (**A**) Moderate lower labial avulsion. (**B**) Minor avulsion repair with a single, loose horizontal mattress suture to approximate the tissues. (**C**) For moderate avulsions when incisor teeth are still present, the avulsion can be repaired with interrupted horizontal mattress sutures through lip mucosa and looped around incisor teeth. (**D**) Lower labial avulsion with incisor teeth missing will require holes to be drilled in alveolar margin. (**E**) Interrupted sutures through lip mucosa and drill holes to repair avulsion.

Alternatively or additionally, interrupted horizontal mattress sutures can be placed through the mucosa of the lip, looping the suture around the intact incisor teeth by passing the needle as close to the mandible as possible (Fig. 28.7C).[5,7] If incisor teeth are missing, small holes may be drilled into the alveolar margin with a 1.0–1.5-mm Kirschner wire or drill bit or using a narrow, tapered diamond bur on a high-speed dental handpiece. Interrupted sutures are passed through these holes (Fig. 28.7D, E).[5,7] If necessary, a small closed-suction drain can be placed through a stab incision in the labial tissue to prevent fluid accumulation in the dead space.

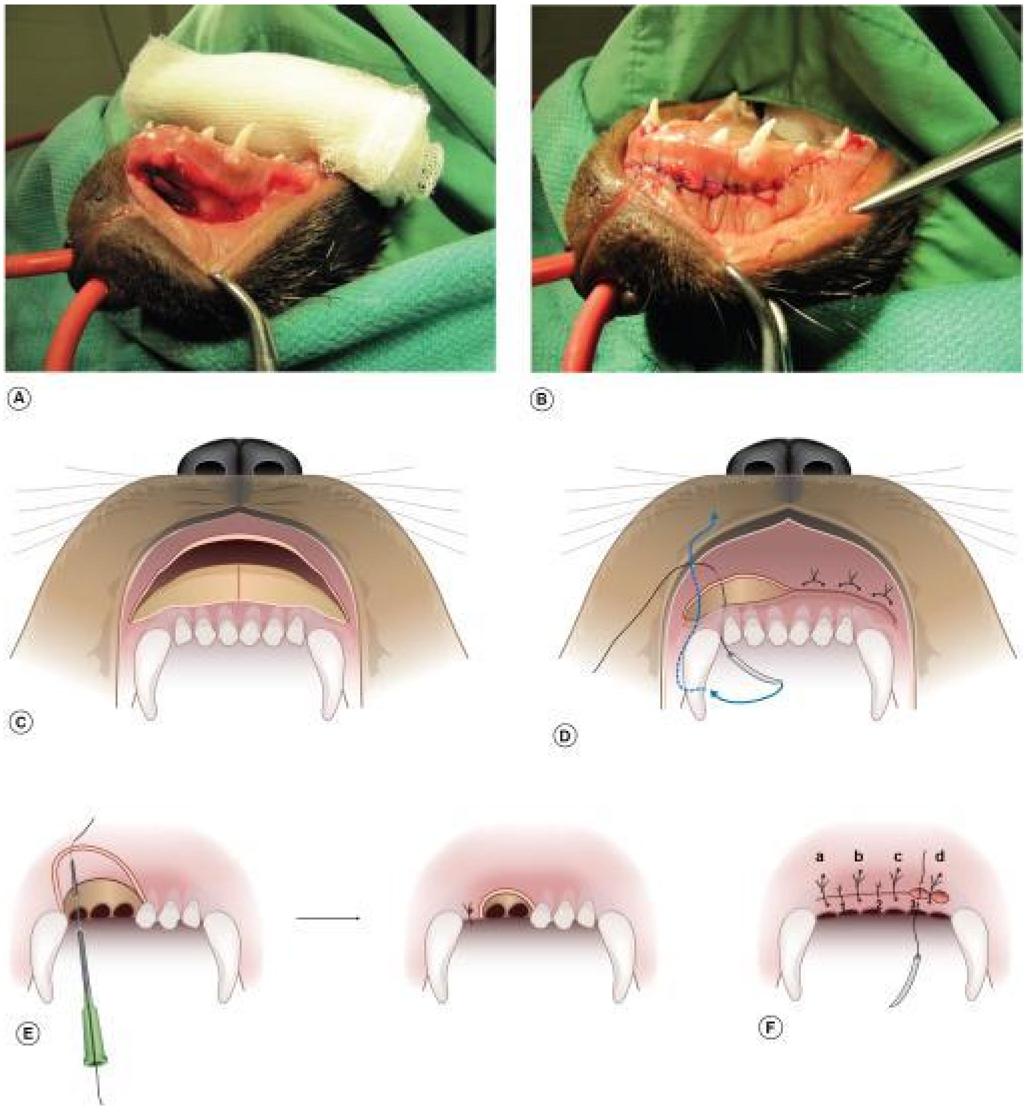
If mandibular fractures or symphyseal separations accompany the labial avulsion, they are typically reduced and stabilized before labial repair (see Chapter 30). Extensive tissue contusion and contamination with lower labial avulsions may preclude immediate closure. During this period of open-wound management, loose sutures can be placed as above to keep the lip from sagging and to help hold dressings in place. Hemoclips on the sutures can be used instead of knots to hold the wound in a "semi-approximated" position; the hemoclips can be removed to allow loosening of the sutures for serial debridement and lavage. Drainage can be provided as described above. With more extensive lower labial avulsion, labial tissue may need to be secured back to the mandible by transosseous tension sutures or wires.[5–7,12] Holes are drilled through the mandible from buccal to lingual with a Kirschner wire or small drill bit while avoiding the teeth roots (Fig. 28.8A). The lip is approximated into normal position, and two 20-gauge hypodermic needles are inserted through the skin at points where the suture will emerge. The needles are placed through adjacent mandibular drill holes to emerge on the medial side of the mandible. One end of a 2-0 monofilament synthetic nonabsorbable suture or 26-gauge wire suture is threaded through the tip of one hypodermic needle until it emerges through the hub of the needle. The same procedure is repeated with the other end of the suture, passing it through the tip of the adjacent needle so the suture midpoint is medial to the mandibles (Fig. 28.8B). The hypodermic needles are removed. The sutures or wires are then loosely tied, with or without a bolster, to approximate the lip soft tissues against the mandible. The gingival and labial mucosa is apposed and sutured (Fig. 28.8D). Closed-suction drainage may be required. With severe lower lip avulsions, be prepared for minor revisional repairs due to minor dehiscence.



• **FIG. 28.8** Extensive lower labial avulsion. (**A**) Drilling holes between teeth for transosseous tension sutures/wires. (**B**) A 20-gauge hypodermic needle passed through the skin and mandible for placing sutures/wire. (**C**) Two transosseous suture/wires placed and Penrose drain in place. (**D**) Transosseous sutures/wires tied through rubber tube stents and interrupted gingivolabial sutures are being placed. (**E**) Suture placement in a clinical case. (**F**) Final appearance in a clinical case.

Avulsion of the upper lip

Upper labial avulsion from the incisive bone is caused by caudodorsal shear force on the nose driving it caudally and dorsally, resulting in disruption of labial or gingival mucosa at the gingival border. With more severe injuries, the nasal cartilage and portions of the upper lip may be avulsed, resulting in an oronasal communication (Fig. 28.9A).[5,7]



• **FIG. 28.9** Upper labial avulsion. (**A**) Minor upper labial avulsion in a puppy, with adequate gingival tissue and minimal exposure of the incisive bone. Nasal patency has been ensured with the temporary insertion of two red rubber catheters, which will be cut short and sutured in place with a fingertrap suture for 5–7 days. (**B**) The tissues have been directly reapposed with absorbable synthetic suture material. A simple interrupted suture pattern is sufficient, as there is less tension on upper labial avulsions than on lower labial avulsions. (**C**) Moderate avulsion with some oronasal communication. (**D**) Avulsion correction with incisor teeth present—interrupted horizontal mattress suture through labial mucosa and gingiva and looped around incisor and canine teeth. (**E**) Minor avulsion with incisor teeth missing. *Left*: A 20-gauge hypodermic needle has been passed through a hole drilled in the incisive bone, and suture has been placed through the mucosal edge and through the needle. *Right*: Needle removed and suture tied. (**F**) Severe upper labial avulsion with all incisor teeth missing—four transosseous sutures *(a–d)* with intervening interrupted mucosal sutures *(1–3)* have been placed.

For a minor upper labial avulsion, tissues may be repaired by simple reapposition if adequate gingival tissue is available, or by anchoring sutures around incisor teeth (Fig. 28.9D).[5,7,13] Absorbable synthetic suture material is used to pass interrupted horizontal mattress sutures through the mucosa, passing the suture through the avulsed labial/gingival mucosa first, then around intact incisor teeth. The needle is passed as close as possible to the incisive bone. The suture is tied with the knot over the avulsed mucosa and not behind the incisor teeth.[5,7]

If incisor teeth are missing, a Kirschner wire can be used to drill holes through the incisive bone.[5,7] Twenty-gauge hypodermic needles are inserted through the holes. After the suture is passed through the mucosal edge, the needle is removed from the suture, which is then threaded through the hypodermic needle. The hypodermic needle is removed from the bone and the suture is tied (Fig. 28.9E).

In cases of severe avulsion of the upper lip where all incisor teeth are missing and there is oronasal communication, four transosseous sutures or wires may be needed across the front of the incisive bone.[7] When the nasal cartilage has been avulsed, care should be used in preplacing sutures in order to align the nostrils properly. Small gaps remaining between transosseous sutures or wires are closed with fine, simple interrupted, synthetic absorbable sutures (Fig. 28.9F).[7] It is critical to ascertain nasal patency in these severe cases, and temporary placement of large nasal catheters for 5 days will facilitate healing and integrity of the rostral nasal passages.

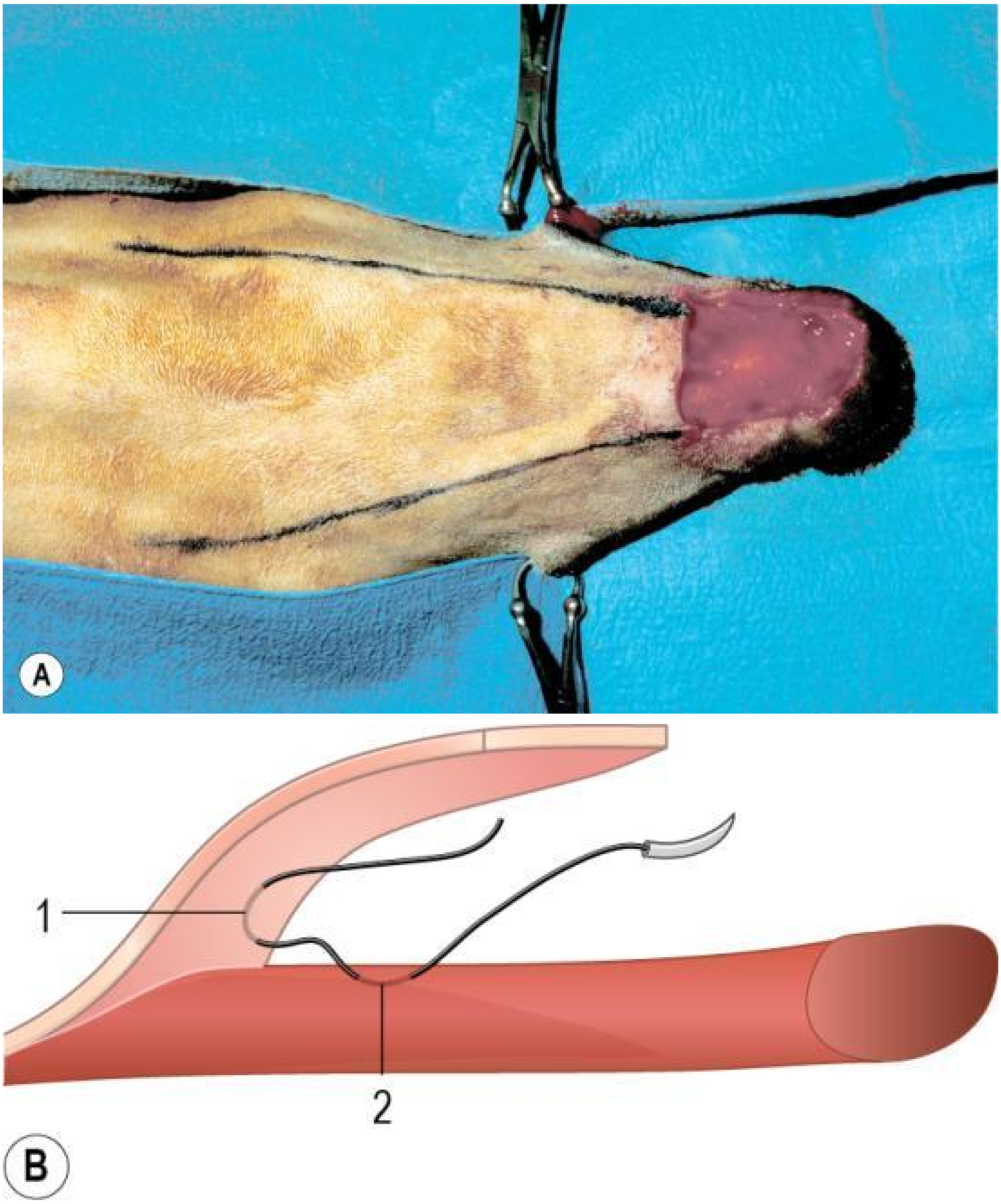
Intermandibular wounds

Pretensioning

Pretensioning, in which skin is placed under a continuous low-grade strain, can be used to recruit skin into wounded areas, such as the intermandibular space, where there is limited skin available for direct closure or flaps. Under a constant load, skin elongates due to realignment of collagen fibers (mechanical creep) and breakage of elastin fibers (stress relaxation). Pretensioning techniques include serially tightening (1) stents anchored by mattress sutures that span the wound, (2) a tie-over bandage, or (3) a loose continuous line of suture.[14] The skin should only be put under a small amount of tension when the tensioning device is first placed, as well as each time it is tightened. Excessive tightening leads to skin necrosis and pull-out of sutures. The stent or suture line is tightened by pulling up on the knot to take up the slack that has developed and placing hemoclips or split-shot (fishing line sinkers) on each arm of the suture below the knot to maintain the tightened position.

Single-pedicle advancement flap

Some skin defects resulting from injury in the rostral intermandibular area can be repaired using a single-pedicle advancement flap from the caudal intermandibular area.[7] With the animal in dorsal recumbency and the head and neck extended, skin incisions are made from the caudal aspect of each side of the defect caudally following the ventral aspect of the bodies of the mandibles (Fig. 28.10). The incisions are made incrementally with undermining of the skin and subcutaneous tissue until a flap of sufficient length has been created to advance and cover the lesion with minimum tension. The length-to-width ratio should not exceed 2:1 for this flap. “Walking” sutures are used to advance the flap into position, preferably on the edges of the incision as described above for the rotation flap. Because excessive use of walking or tacking sutures can compromise blood supply, they should be used judiciously on the deep side of flaps. Once advanced into position, the flap is closed in two to three layers: subcutaneous tissue, cutaneous muscle, and skin.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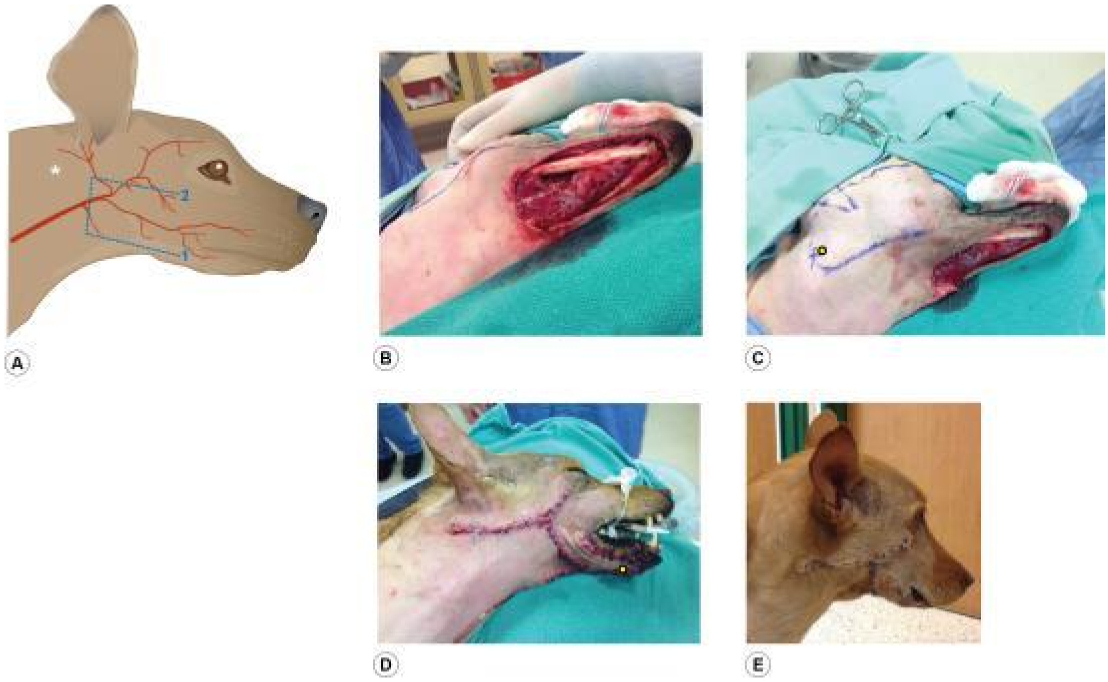
1
2
B

• **FIG. 28.10** Single-pedicle advancement flap for intermandibular wound. (**A**) Skin defect in the rostral intermandibular area with single-pedicle advancement flap drawn following the ventral aspects of mandibular bodies. (**B**) Placement of "walking" sutures to advance flap rostrally, second bite *(2)* is rostral to the first bite *(1)*. (**C**) Flap sutured in place.

Axial pattern flaps

The angularis oris and caudal auricular axial pattern flaps can be used to repair intermandibular wounds (Fig. 28.11).[5,6,8,15–21] Further discussion of these axial pattern flaps may be found in Chapter 52.



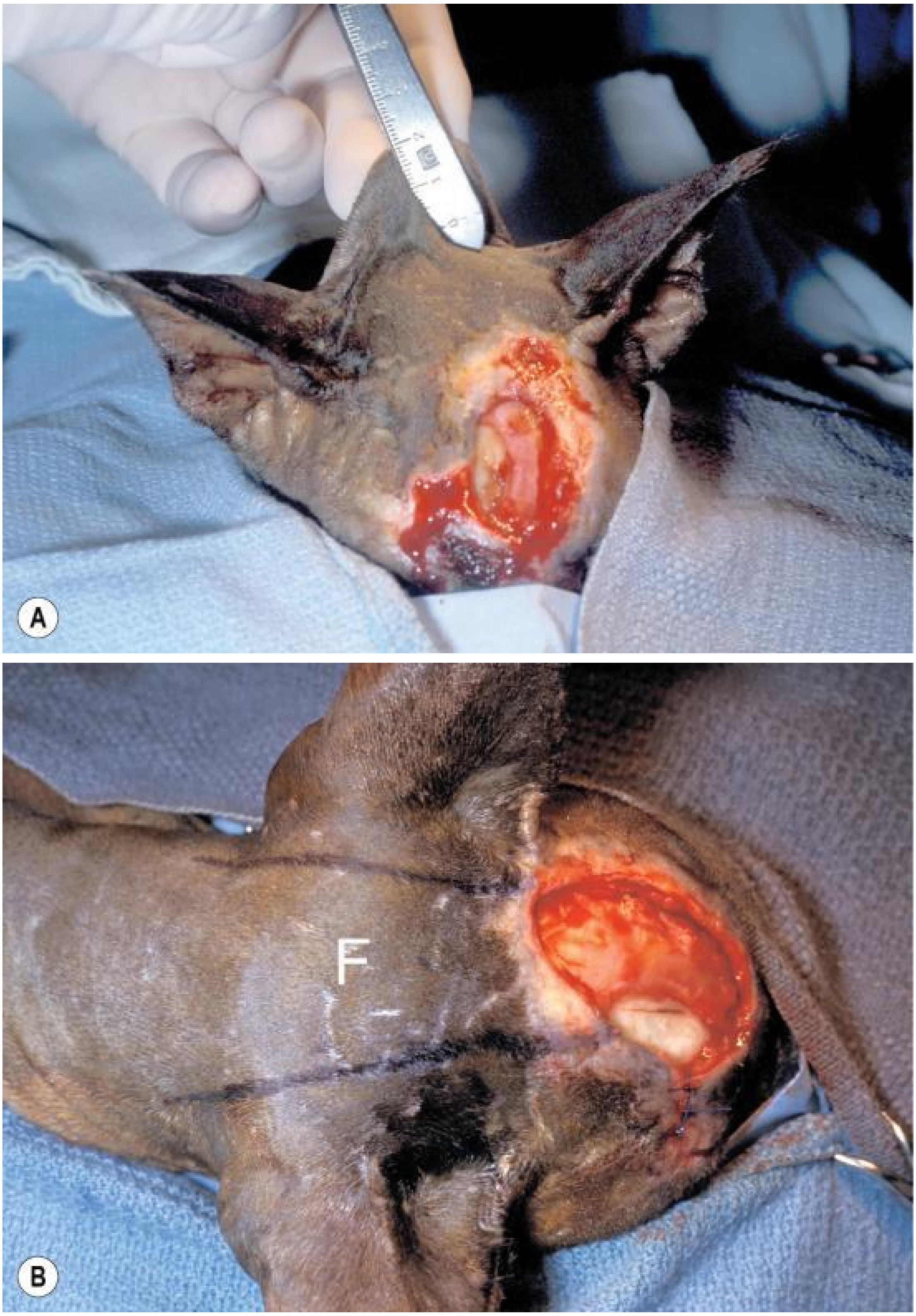
• **FIG. 28.11** Angularis oris axial pattern flap is a caudodorsally oriented flap that can be rotated around its base at the commissure of the lip. The ventral border of the flap is a caudal extension from the body of the mandible *(blue dotted line 1)*. The dorsal border of the flap is the level of the ventral border of the zygomatic arch *(blue dotted line 2)*. The length of the flap should be at the level of the vertical ear canal but can be variably extended to the level of the wing of the atlas *(*)*. The flap can be rotated dorsally to cover the muzzle or ventrally to cover intermandibular defects. (**B**) Intermandibular defect in a dog. (**C**) Landmarks drawn for a left-sided angularis oris flap. (**D**) The flap has been developed, rotated ventrally to cover the defect, and sutured in two layers with a closed-suction drain. (**E**) Two-week postoperative appearance.

Large wounds on the dorsum of the cranium

Wounds on the dorsum of the head can be repaired by single-pedicle advancement flaps, rotation flaps, transposition flaps, or axial pattern flaps (caudal auricular, superficial temporal, or angularis oris).[5–9,17,18,22]

Single-pedicle advancement flap

A single-pedicle advancement flap can be used for wounds in the caudodorsal area of the head and/or craniodorsal cervical area when skin is available on only one side of the defect (usually caudal) (Fig. 28.12).[7,8] Skin surrounding the wound should be manipulated and measured along with measurement of the defect to determine if sufficient skin is present for such a flap. To ensure adequate perfusion to the leading edge of this subdermal plexus flap, the length of the flap should not exceed twice the width of the flap. To create the flap, two parallel or slightly divergent incisions are made from the caudal aspect of the wound using the caudal edge of the wound as the leading edge of the flap. Incisions are made incrementally in the skin and any underlying subcutaneous fascia and panniculus muscle, in conjunction with undermining, until a flap of sufficient length has been created to cover the wound with minimum. "Walking" sutures of synthetic absorbable material can be used on the edges of or underneath the flap to advance the flap over the wound. Skin closure is routine and performed in two layers: subcutaneous and skin.[7]


A
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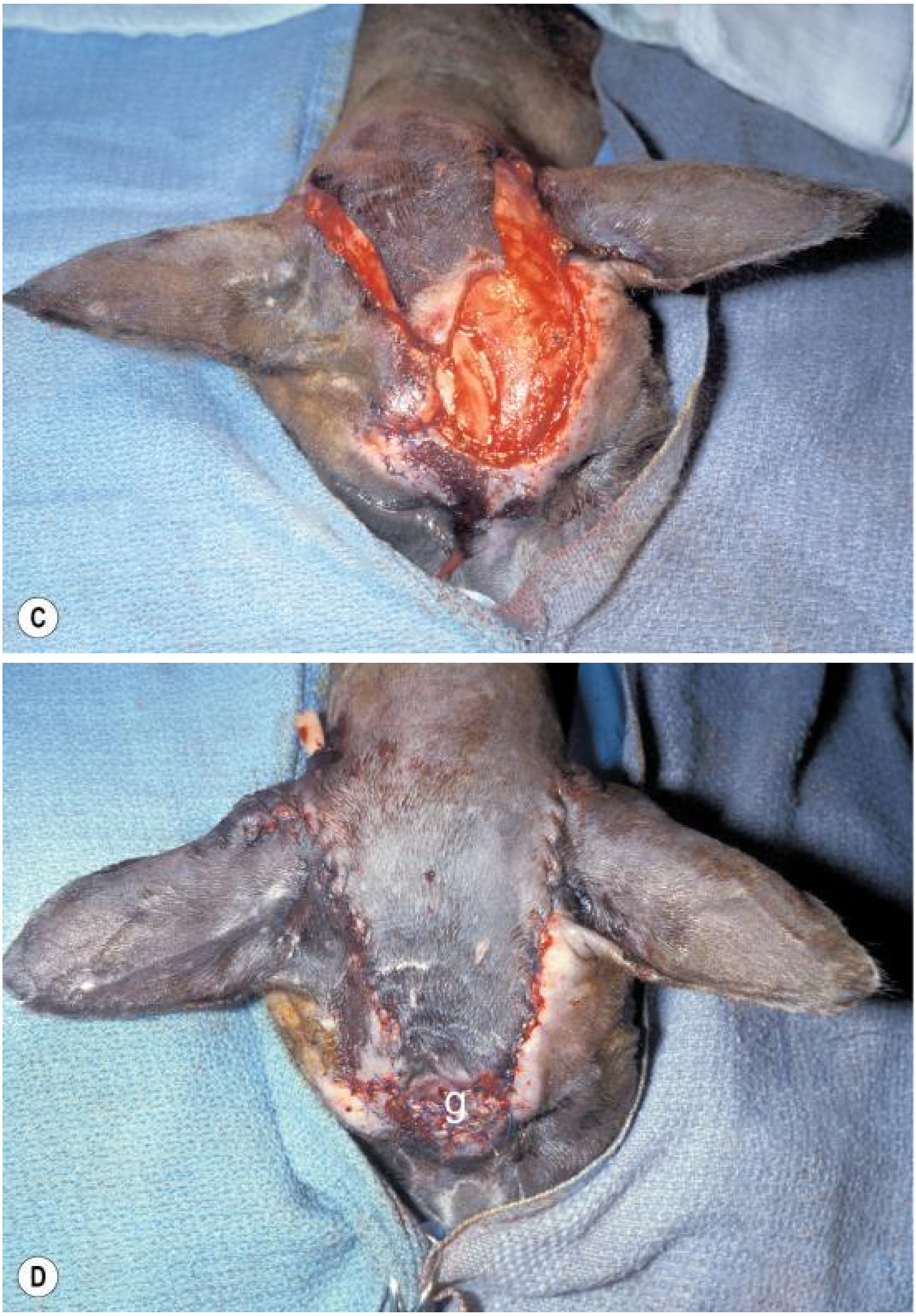

C
D
g

• **FIG. 28.12** Single-pedicle advancement flap to correct a large dorsal cranial wound. (**A**) Large wound on rostrodorsal area of a cat's head. Skin on cranial cervical and caudal cranial area measured for flap creation. (**B**) Flap *(F)* drawn on caudal cranial and cranial cervical skin. (**C**) Flap incised. (**D**) Flap advanced into place. A small mesh graft *(g)* was placed at the rostral-most end of the effect. (**E**) Flap and graft healed. Source: (From Swaim SF, Henderson RA. *Small Animal Wound Management*. 2nd ed. Baltimore: Williams and Wilkins, 1997:250–251, with permission.)

Transposition flaps

If there is concern as to whether a single-pedicle advancement flap will provide the skin needed for wound repair, a transposition flap can be considered. Large flaps can usually be raised from the abundant loose skin in the cervical area. If the flap's base cannot be incorporated into the edge of the defect, a bridging incision can be made between the edge of the wound and base of the flap. Thus the flap base can be completely sutured in place. This avoids tubing the flap base over intact skin. For cosmesis, a strip of skin can be excised along the bridging incision so the flap base lies flat. A drain may be necessary in the donor area because seromas are common due to movement in the cervical area.[6]

Axial pattern flaps

Axial pattern flaps based on the caudal auricular or angularis oris vessels can be used for repair of dorsal cranial wounds.[5,6,8,16–19,22,23] These flaps are described in Chapter 52.

Large frontal and lateral facial wounds

Single-pedicle advancement flaps

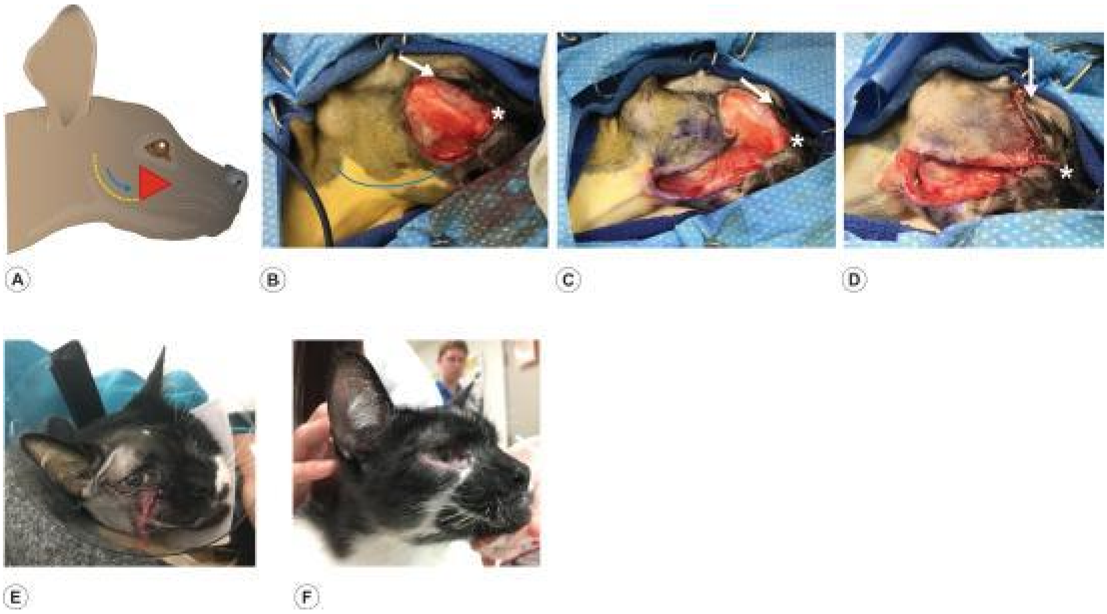
Square or round wounds on the lateral aspect of the face can often be closed with a single-pedicle advancement flap from the caudal aspect, taking advantage of the more abundant skin of the neck (Fig. 28.13). Depending on the amount of skin available, determined by manipulating the periwound skin during planning, two opposing single-pedicle advancement flaps can be developed from the dorsal head and ventral neck (Fig. 28.14). This is called an H-plasty and it provides significant relief of tension on the closure. The length to width ratios for single-pedicle advancement flaps should not exceed 2:1 to maximize flap perfusion.

• **FIG. 28.13** (**A**) Defect on the side of the face from a scorpion bite several weeks earlier. (**B**) Lines demarcating the single-pedicle advancement flap borders have been drawn with a sterile marker, making the flap base a little wider to optimize perfusion of the flap. Additionally, the estimated location of the superficial cervical branch of the omocervical vascular pedicle has also been marked with an X and will be preserved during flap development. (**C**) Single-pedicle advancement flap has been advanced from the neck rostrally into the defect and closed in two layers. Note the drain exiting ventrally.

• **FIG. 28.14** (**A**) A 2-month-old, non-healing envenomation wound from a Brown Recluse spider bite on the left lateral face of a dog. (**B**) Following manipulation of the adjacent skin to assess skin availability and excision of the skin immediately surrounding the wound, two single pedicle advancement flaps were planned (dotted yellow lines), from dorsal and ventral, forming an H-plasty. (**C**) The H-plasty has been developed and advanced to cover the defect. (**D**) Appearance two days postoperatively.

Rotation flaps

A semicircular rotation flap of skin and subcutaneous tissue can be used to repair triangular wounds on the head and face, to include the lateral facial area, caudal bridge of the nose, and especially around the eyes to minimize tension on the eyelids.[7,8] The flap is moved by a combination of rotation, transposition, and some stretching to close triangular wounds that only have skin available for closure on one side of the wound (Fig. 28.15).[5,7] A semicircular flap is drawn on the skin with the leading edge of the flap being one edge of the wound, with the arc of the flap being approximately four times the size of the defect. It should be large enough so there is no tension across it when it is sutured in place. The flap and associated cutaneous muscle and subcutaneous tissue are then raised incrementally with sharp undermining until it can be rotated across the wound without excess tension. Back cuts into the flap are not recommended. The "dog ear" of skin that forms adjacent to the base of the flap on the convex aspect can be removed if it is excessive.[7]

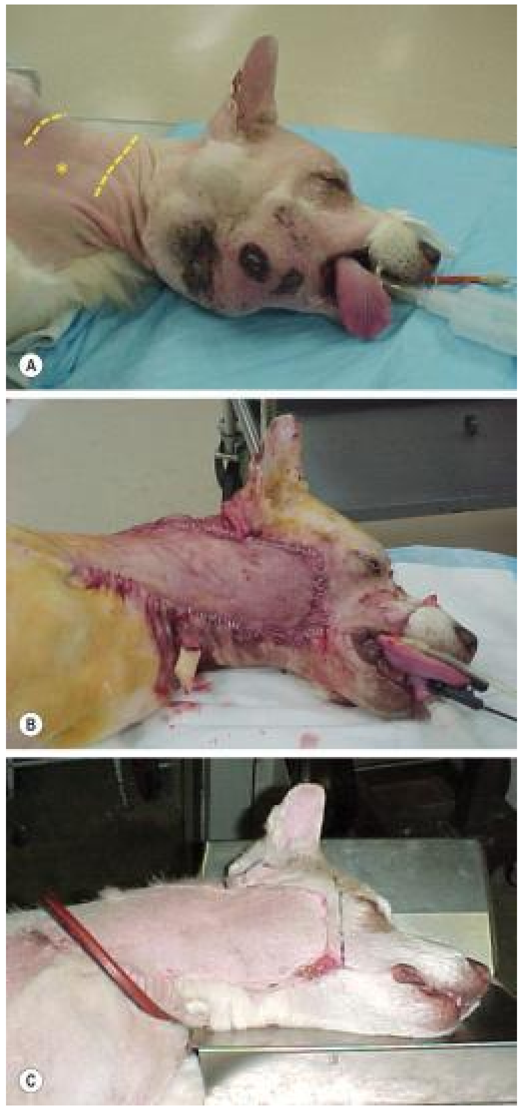


• FIG. 28.15 Rotation flap used to close a triangular defect when skin for closure is only available on one side of the defect. (**A**) Rotation flap to close lesion adjacent to eye. (**B**) Wound involving the lower right eyelid and side of the face in a cat (the eyelid edge is intact). *Arrow* indicates lateral canthus, *asterisk* indicates medial canthus; rostral is to the right. *Dashed line* indicates planned incision for rotation flap. (**C**) Rotation flap partially incised and elevated. (**D**) Dorsal edge of flap sutured in place. (**E**) An Elizabethan collar was placed immediately after surgery to prevent self-trauma. (**F**) Appearance 5 weeks postoperatively.

Axial pattern flaps

There are several flaps that incorporate a direct cutaneous artery and vein that are useful in reconstruction of the face. These include the caudal auricular, superficial temporal, omocervical, and the angularis oris.[a] These flaps have better perfusion, allowing much larger flaps to be developed, compared to their subdermal counterparts. These flaps are also described in Chapter 52.

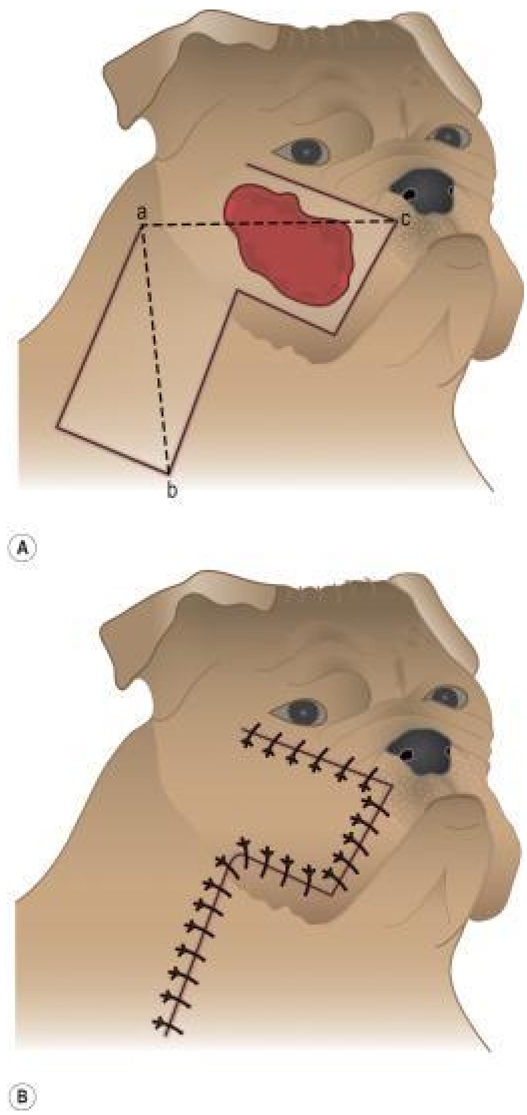
The *omocervical axial pattern flap* can also be used to repair lateral facial wounds (Fig. 28.16), although it does not extend as far rostrally as the angularis oris flap.[5,8] The flap is based on the superficial cervical vessels that exit the musculature cranial to the shoulder at the level of the superficial cervical lymph node and the cranial shoulder depression. The vessels course craniodorsally. Flap boundaries are as follows: ventrally, the acromion of the scapula; caudally, the scapular spine; and cranially, a line parallel to the caudal border, i.e., twice the distance from the scapular spine to the prescapular lymph node. The dorsal midline is a safe distal border. However, it may extend to the contralateral shoulder area, especially in cats, or if the delay phenomenon is utilized.[8,26]



• **FIG. 28.16** (**A**) Myxosarcoma on the lateral aspect of a dog's face. The vascular pedicle is shown with an asterisk, and the proposed incision lines, which extend ventrally from dorsal midline, are shown in dotted lines. (**B**) Immediate postoperative appearance of an omocervical axial pattern (OAP) flap brought in to cover the defect following tumor removal. (**C**) Appearance of the OAP flap 21 days later, before adjunctive radiation therapy.

Transposition flaps

A random-pattern transposition flap taken from the ventrolateral cervical area has been described for repair of a large lateral facial defect on a brachycephalic dog.[27] A dorsally based rectangular flap is designed on the ventrolateral cervical area of such a length that the diagonal across the flap from its pivot point is equal to the diagonal from the pivot point to the far corner of the defect (Fig. 28.17A). This compensates for the loss of effective length of the flap as it is rotated into position.[7] The flap is transposed into position and sutured as described earlier (Fig. 28.17B).



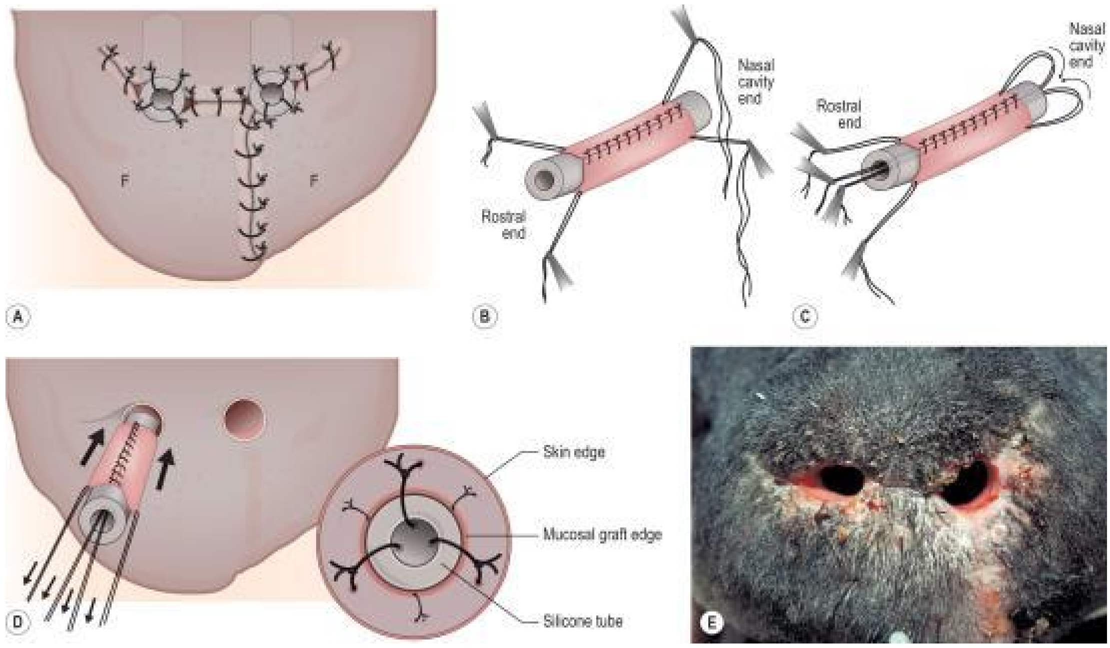
• **FIG. 28.17** Lateral facial wound repair using a random-pattern transposition flap. (**A**) A dorsally based rectangular transposition flap signed on the ventrolateral cervical area. Diagonal across the flap from its pivot point is equal to the diagonal from the pivot point to the far corner of the defect (a–b = a–c). (**B**) Flap in position with donor site closed.

Nasal planum and nares reconstruction

Occasionally it is necessary to reconstruct the nasal plane or nares (see also Chapter 50). Defects of the nasal planum can be covered in a staged procedure using two rotation flaps (Fig. 28.18).[28] This technique results in haired skin where the nasal plane previously existed but provides a nonpainful and tough covering. Other subdermal plexus flaps and full-thickness lip transpositions have also been described.[5,29,30] These planum reconstruction techniques often require individual tailoring and flap modifications to fit the specific defect in each case.

• **FIG. 28.18** Replacement of the dorsal aspect of the nasal planum can be achieved by developing two rotation flaps in a staged fashion. (**A**) Severe actinic dermatitis of the nasal planum. (**B**) The affected planum tissue has been resected and a rotation flap has been developed from the left muzzle. (**C**) The flap has been undermined, rotated into the defect, and sutured in two layers. (**D**) Appearance of the left side of the face 1 month postoperatively, at which time the same flap is performed on the right side. (**E**) Final postoperative appearance after both flaps have healed. The nasal planum is now covered with hirsute skin.

Mucosal grafts can be harvested from under the tongue or from the upper lips or inside the cheeks to reconstruct nares.[5,8,31] For reconstruction of the nares, it is necessary to have some wound tissue in the area where the nares will be reconstructed. This can be in the form of edges of labial flaps that have been used to reconstruct the area or by cutting holes in such a flap that will serve as the "nares" openings. Silicone tubes are placed between the edges of the flaps and back into the nasal passages as the edges are sutured together or into the holes cut in a flap (Fig. 28.19). The tubes are sutured in place by interrupted sutures through the tube and adjacent skin. The tubes are left in place for approximately 2 weeks to allow granulation tissue to form around them. At this time, rectangular mucosal grafts of sufficient width to go around the silicone tube and of sufficient length to traverse the length of the granulation tube that has formed are harvested bilaterally from under the tongue. The grafts may be harvested by hydrodissection.[8] This requires injecting 5 to 10 mL of dilute solution of lidocaine with epinephrine (25 mL of 1% lidocaine/epinephrine per liter of lactated Ringer's solution) submucosally on the ventral tongue surface. This allows easy collection of the graft because of separation of the mucosa from the underlying tissue. The grafts are sutured submucosal side out around the silicone tubes that have been in the nasal cavity. Two doubled traction sutures of synthetic monofilament suture are placed in each end of the graft 180 degrees apart. The two sutures in the end of the graft to be placed in the nasal cavity are threaded back through the end of the tube they are closest to and out the other end of the tube. With traction on all four sutures to keep the graft smooth, each tube with its graft is reinserted in the nasal cavity. The rostral edge of the graft and tube are affixed to the skin at the edge of the "nares" opening with simple interrupted sutures after all traction sutures are removed. Seven days later, the sutures are removed, and the tubes are easily removed, leaving cylindrical "nares" openings with healed mucosal grafts lining the area.



• **FIG. 28.19** (**A**) Silicone tubes placed between labial flaps *(F)* and back into the nasal passages. Tubes are sutured to the skin edges to hold them in place. Mucosal graft preparation for placement. (**B**) Graft sutured, submucosal side out, around silicone tube with two-double-traction sutures 180 degrees apart at each end. (**C**) Sutures at nasal cavity end of graft are passed back through tube. (**D**) Placement of mucosal grafts into granulation tissue-lined "nares." Tube with graft is inserted into "nares" *(large arrows)* with traction on traction sutures *(small arrows)* to hold graft smooth. *(Inset)* Graft and tubes sutured to the skin. (**E**) Mucosal grafts healed in place, lining the "nares." Source: (A–E from Welch JA, Swaim SF. Nasal salvage/reconstruction in a dog following severe trauma. *J Am Anim Hosp Assoc* 2003;39:407–415).

Postoperative care and assessment

Elizabethan collars are mandatory to prevent self-trauma to the lip and facial reconstruction areas. If wounds were infected prior to debridement, systemic antibiotics are indicated. For more extensive flaps, intermittent compresses for 72 hours after surgery may help reduce edema.[1,7,22] Edema is common with axial-pattern flaps. Nonabsorbable skin sutures and transosseous sutures or wires are removed 10 to 14 days postoperatively.

A basket muzzle, nylon muzzle, or tape muzzle may be indicated if opening the mouth a significant distance will put too much tension on the repair site. When creating a tape muzzle, the nonadhesive side of the tape is placed against the skin and the adhesive side is covered with another piece of tape.[32] The muzzle must be designed so that the animal can open its mouth enough to eat (a liquid diet may be needed) and drink, as well as to safely expel any vomitus.

If there is significant intraoral repair, hand feeding the animal canned food shaped into meatballs of a size that can be swallowed whole, without chewing, minimizes the forces exerted in the mouth and decrease the creation of food particles that might stick to sutures and stimulate licking. While typically not needed, an esophageal feeding tube can be placed to completely bypass the oral cavity for a period of time in cases with tenuous or extensive repairs.

Drains are removed when fluid becomes serosanguineous and the quantity has plateaued at a low level.[33] This is usually within 3 days for smaller wounds and within 4 to 5 days for major wounds. Closed-suction drains are preferred over passive drains.

Following lip avulsion, no attempts should be made to examine the surgical site or even to lift the lip to assess mucous membrane color and capillary refill time during the first 7 days unless problems are suspected. If the area is examined, sedation or anesthesia should be used to prevent the animal from resisting examination and damaging the surgical site.[7]

Complications

Proper debridement, lavage, drainage, and tension-free closure are key to achieving good healing and avoiding wound infection, abscessation, and tissue slough. Dehiscence is most likely due to excessive tension and/or tissue necrosis (secondary to the original injury or the tension of the repair). If a nares reconstruction is attempted before there is a solid bed of granulation tissue in place, the tissues may not fully support the graft and the neo-naris may collapse upon inspiration. Dehisced sites should be managed as open wounds at first, delaying surgical revision several days to several weeks until the tissue is healthy and well vascularized, and alternative means to achieve a tension-free closure should then be implemented.

For repairs involving labial or buccal tissues, suturing the mucosa and submucosa together can cause mucosa to invert, which can impair healing. Thus submucosa should be sutured independently, and mucosa should be sutured separately from within the oral cavity.[6,7] When it is necessary to create a new mucocutaneous junction, haired skin may be pulled into the area normally occupied by the labial mucosa, which can result in hair growing in the mouth and becoming matted with saliva. To prevent this, the skin should be trimmed back so that suturing skin to mucosa either results in edge-to-edge apposition at the lip edge or pulls labial mucosa to the outside.

When repairing lip avulsions, it is difficult to suture the gingiva on the buccal surface of the teeth because the gingiva is tightly attached to the underlying bone, resulting in sutures tearing out. Suturing through just the interdental space mucosa may not provide sufficient support to hold the tissues apposed until adequate healing has occurred. Use of swaged taper-cut needles may help prevent sutures from tearing out. However, if major avulsions are not stabilized with transosseous tension sutures, tension on the simple gingival-labial sutures may arise as tissues swell and weight of the lip tissues increases.[5–7,12]

If a flap is designed too short and/or not anchored sufficiently, tension could cause displacement of the tissue to which the flap is sutured. For example, a tight flap may (1) distort the eyelids, predisposing the patient to conjunctivitis or corneal ulceration, (2) pull back the lip, exposing teeth and decreasing retention of food or saliva in the mouth, (3) alter the commissure, compromising ability to open the mouth, or (4) deviate the nasal planum (which usually subsides in 2 to 3 weeks).[5,7,8] Intraoperative assessment of tension should take into consideration the forces that will be exerted when an animal recovers from anesthesia and resumes a normal head and neck flexion posture.

The tip of an axial pattern flap may necrose from lack of perfusion if the flap is too long, the source artery is damaged, or the skin is distorted due to poor patient positioning when the flap is being cut.[5,8,33] If initial measurements indicate that the flap will have to be over-long, another form of reconstruction should be considered, or the flap could be preconditioned using the delay phenomenon.[26] A disadvantage of axial pattern flaps is the difference in type, direction, and length of hair growth in the reconstruction area, especially when moving a caudal auricular or omocervical axial pattern flap from the neck or torso to the face.[19] On long-haired dogs and cats, periodic trimming of their "beard" may be necessary. Particularly when large flaps are used, seroma may result, and drains (preferably closed active suction drains) should be preemptively placed. Aspiration of seromas is not advised due to the risk of seeding infection.

Facial reconstructions are highly susceptible to self-mutilation, even with an Elizabethan collar in place. Recovery from anesthesia should be smooth without any head shaking or thrashing, as these events can cause hemorrhage, undue tension, and dislodge drains. Hemorrhage underneath a mucosal graft can impede graft acceptance. Rubbing of the nose or face along the floor, furniture, or with the paws must be avoided, usually by a combination of physical and chemical restraint. In addition to a large Elizabethan collar, front feet hobbles, and sedation may be required. Due to their location, labial sutures can be easily molested by the animal's tongue, resulting in wound disruption. If wire is used for the transosseous fixation, the sharp ends of the tags help discourage such suture molestation.

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[a] 5,6,8,17,18,20–22,24,25

CHAPTER 29

Surgical approaches for mandibular and maxillofacial trauma repair

Frank J. M. Verstraete, Boaz Arzi, Abraham J. Bezuidenhout

Principles and classification

Principles

An acceptable surgical approach to a bone or joint is the least traumatic yet effective method of exposing the bone or joint, as determined by the topographical anatomy of the region. Major blood vessels, nerves, and salivary glands and ducts must be avoided or retracted.[1] It is therefore essential to be familiar with the normal anatomy. Muscles should be separated by blunt dissection of the intermuscular septa rather than incised or transected. To expose an area of bone that serves as an origin or insertion of a muscle, or where muscle loosely attaches to the bone, one of several techniques may be applicable, depending on the nature of the attachment. In most cases, muscle that adheres to the underlying bone must be elevated subperiosteally. The periosteum is exposed by blunt dissection between two muscle bellies, incised, and elevated using a periosteal elevator. A periosteal elevator designed for oral surgery (see Chapter 7) can be used for this purpose; alternatively, a small orthopedic periosteal elevator is suitable.[1]

The surgeon is encouraged to regain the maximum function of injured skin or mucosa with the least possible deformity and scarring. This may be achieved by evaluating the static and dynamic skin tensions on the surrounding skin.[2] Static tension lines exist within the skin and are oriented in specific but variable directions.

Extraoral approaches

The extraoral or transfacial approaches are most commonly used in veterinary surgery.[1] In order to obtain an optimally healing wound, the extraoral surgical incision should follow the relaxed skin tension line direction within these areas. Skin tension lines are usually oriented perpendicularly to the underlying muscles.[2] Utilization of an extraoral approach is usually performed for open reduction of fractures followed by plating (e.g., mini-plating, locking reconstruction plates), screw applications, and certain wiring techniques. When such an approach is used, the facial neurovascular structures are potentially at risk, and therefore the surgeon must be aware of and consider the relevant topographic anatomy.

Intraoral approaches

Intraoral or transoral approaches are most commonly used in humans, in order to avoid cutaneous scars.[2,3] They are less frequently used in veterinary surgery; when they are used, it is primarily for fractures of the maxilla and incisive bone (see Chapters 32 and 33). The typical intraoral incision lines for exposure of either the maxilla or the mandible are made within the unattached alveolar mucosa 3–4 mm away from the mucogingival junction. To expose the caudal part of the mandible, the incision line is placed directly over the ramus of the mandible.

Approach through a traumatic wound

Making a surgical approach through a traumatic wound should, as a general rule, be avoided.[1] It is occasionally indicated for avulsion of the lip or other oral soft tissues, usually due to vehicular trauma.

In general, the approach should not add unnecessary trauma to that which the injured area has already sustained. The incision may be extended beyond the traumatic wound to allow adequate exposure to better analyze the destruction of anatomic and physiologic functions of the area invaded. In certain instances, a small exposure will force the surgeon to exert excessive pressure when manipulating the wound, which will further injure the muscles and impair circulation to the area.

Anesthetic considerations[4]

A cuffed endotracheal tube and secured pharyngeal pack should be used to prevent aspiration during surgery. Restoring occlusion is the mainstay of proper repair of maxillary and mandibular fractures. When performing the fixation procedure in many patients, particularly in the more complicated cases, a pharyngotomy intubation is done to maintain anesthesia. This technique ensures an open airway while the animal's mouth is closed and the teeth can be occluded during the procedure, ensuring adequate reduction during application of fixation. After the surgery is completed, the endotracheal tube is removed and the pharyngotomy opening is allowed to heal by second intention.

Surgical anatomy

Regional anatomy

In the upper jaw, the incisive and maxillary bones carry teeth and form most of the dorsolateral surface of the face. Alveolar juga of the canine and fourth premolar teeth are features of this surface. The infraorbital foramen, through which the infraorbital nerve and blood vessels emerge, lies dorsal to the septum between the third and fourth premolar teeth. Nasal bones complete the face dorsally, and the lacrimal and zygomatic bones complete the caudal aspect of the face rostral and ventral to the orbit. The temporal process of the zygomatic bone and the zygomatic process of the squamous part of the temporal bone are the only contributors to the zygomatic arch. The orbital ligament connecting the zygomatic process of the frontal bone and the frontal process of the zygomatic bone completes the rim of the orbit caudally.

The body of the mandible has alveolar and ventral margins, and lingual and buccal/labial surfaces. The alveolar border is indented by conical alveoli for the roots of the incisor, canine, premolar, and molar teeth. In mesaticephalic skulls the ventral border is slightly curved (convex), in brachycephalic skulls this convexity is more pronounced, while in dolichocephalic skulls the ventral border is almost straight. Rostrally the buccal surface carries three mental foramina through which the mental nerves and blood vessels emerge. The middle mental foramen is the largest, and lies ventral to the septum between the first two premolar teeth. The position of the caudal foramen is variable; it is generally located at the level of the distal root of the third premolar tooth, but may be rostral or caudal to it. Caudally, at the angle of the mandible, the body meets the ramus, the non-tooth-bearing portion of the mandible. Dorsally the ramus bears two processes: the coronoid process for the attachment of the temporal muscle and a transversely elongated condylar process that articulates with the zygomatic process of the temporal bone to form the temporomandibular joint (TMJ). The two processes are separated from each other by the mandibular incisure. The masticatory branch of the mandibular nerve to the masseter muscle passes from medial to lateral over the incisure. The lateral and medial surfaces of the coronoid process are concave, forming a masseteric fossa laterally and a pterygoid fossa medially. Ventrally, at the angle of the mandible, the ramus bears an angular process for the attachment of the masseter and medial pterygoid muscles. The mandibular foramen for the inferior alveolar nerve and blood vessels lies medially between the base of the coronoid process and the angular process, caudal to the last molar tooth. Left and right mandibles are rostrally united to each other by a slightly flexible, rough-surfaced fibrous joint, the symphysis of the mandible.

A thin fibrocartilaginous disc lies between the condylar process of the mandible and the mandibular fossa of the temporal bone, completely dividing the joint space into dorsal and ventral noncommunicating compartments. The joint capsule attaches to the cartilage and is strengthened by the lateral ligament.

The extensive but thin platysma muscle extends from the dorsum of the neck to the angle of the mouth and ventrally as far as the midline. The mentalis muscle radiates from the alveolar border of the mandible near the third incisors into the lower lip. More caudally, the orbicularis oris muscle extends from the commissural region into the lips near their free borders.

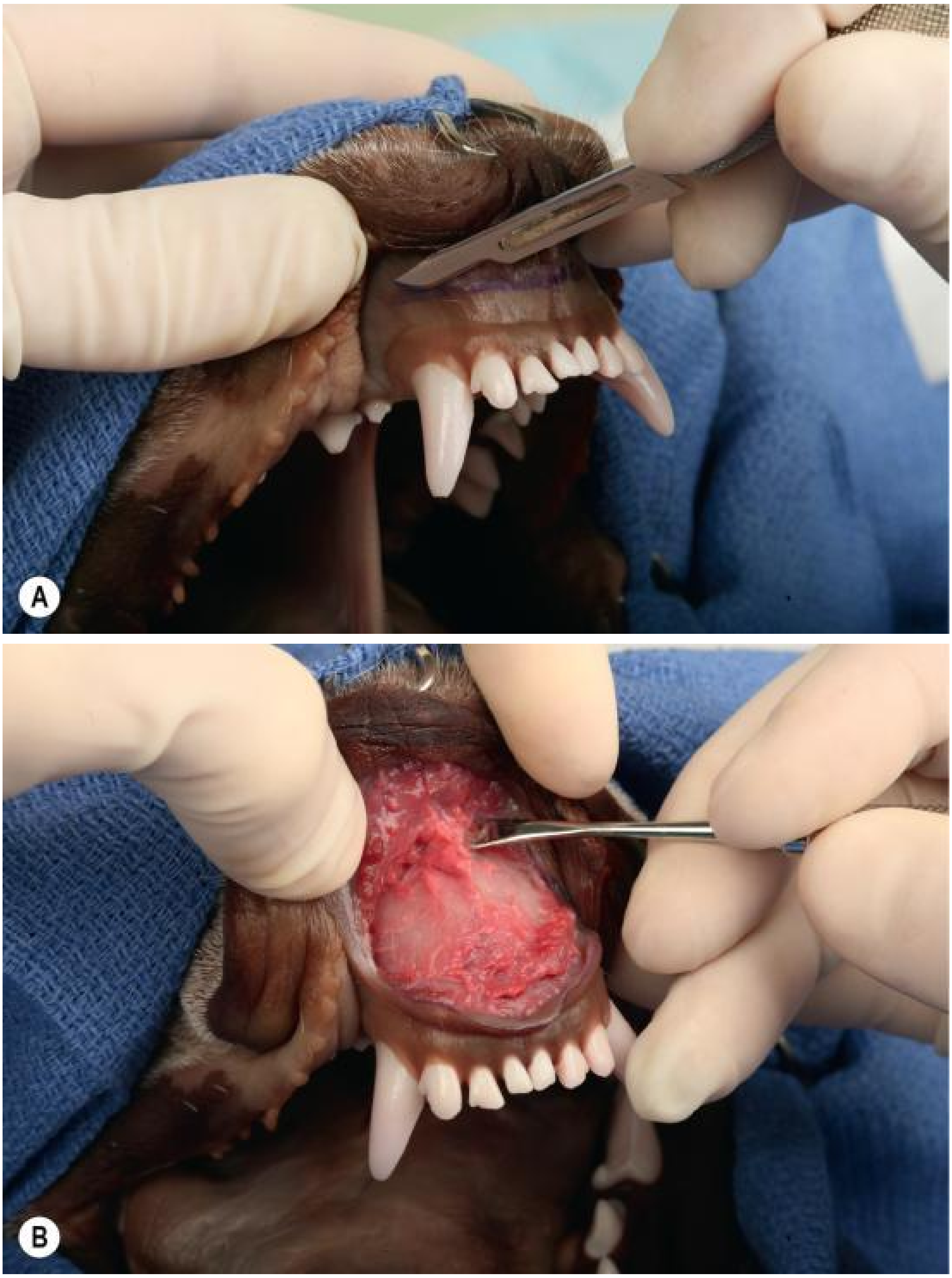
The masseter muscle is attached to the zygomatic arch and the lateral surface of the ramus of the mandible. It projects slightly beyond the ventral and caudal borders of the mandible, with some fibers attaching on the ventromedial surface. The temporal muscle has a broad attachment to the temporal fossa of the cranium and extends to the medial and lateral surfaces of the coronoid process, medially as far distally as the mandibular foramen and laterally as far distally as the ventral ridge of the masseteric fossa. The robust medial pterygoid muscle extends from the lateral surface of the pterygoid, palatine, and sphenoid bones to the medial and caudal surfaces of the angular process of the mandible. The lateral pterygoid muscle extends from the sphenoid bone to the medial surface of the condylar process, just distal to the articular condyle. Other muscles associated with the mandible are the digastric, mylohyoid, geniohyoid, and genioglossus muscles. The mylohyoid muscle is the most superficial, extending from the mylohyoid line of the mandible (along the lingual surface just ventral to the alveoli) to the basihyoid bone, forming a transverse sling for the tongue. The geniohyoid muscle extends along the midline from the caudal edge of the mandibular symphysis to the basihyoid bone; the genioglossus extends from the same area of the symphysis and radiates dorsally into the tongue. The digastric muscle originates from the paracondylar process of the occipital bone and is attached along the ventromedial border of the mandible up to the level of the canine tooth. An indistinct myotendinous intersection separates the caudal and rostral bellies of the muscle.

The parotid, mandibular, and monostomatic sublingual salivary glands lie caudal to the ramus of the mandible. The parotid duct crosses the lateral surface of the masseter muscle, ventral to the dorsal branch of the facial nerve, to open in the vestibule on the parotid papilla located opposite the caudal margin of the upper fourth premolar tooth. The ducts from the mandibular and sublingual salivary glands are closely related to each other throughout their course. The two ducts pass between the masseter and mandible laterally and the digastric muscle medially, then arch rostrally in the intermuscular septum between the genioglossus and mylohyoid muscles. Both ducts open on the sublingual caruncula.

Surgical approaches

Approach to the incisive part and mandibular symphysis

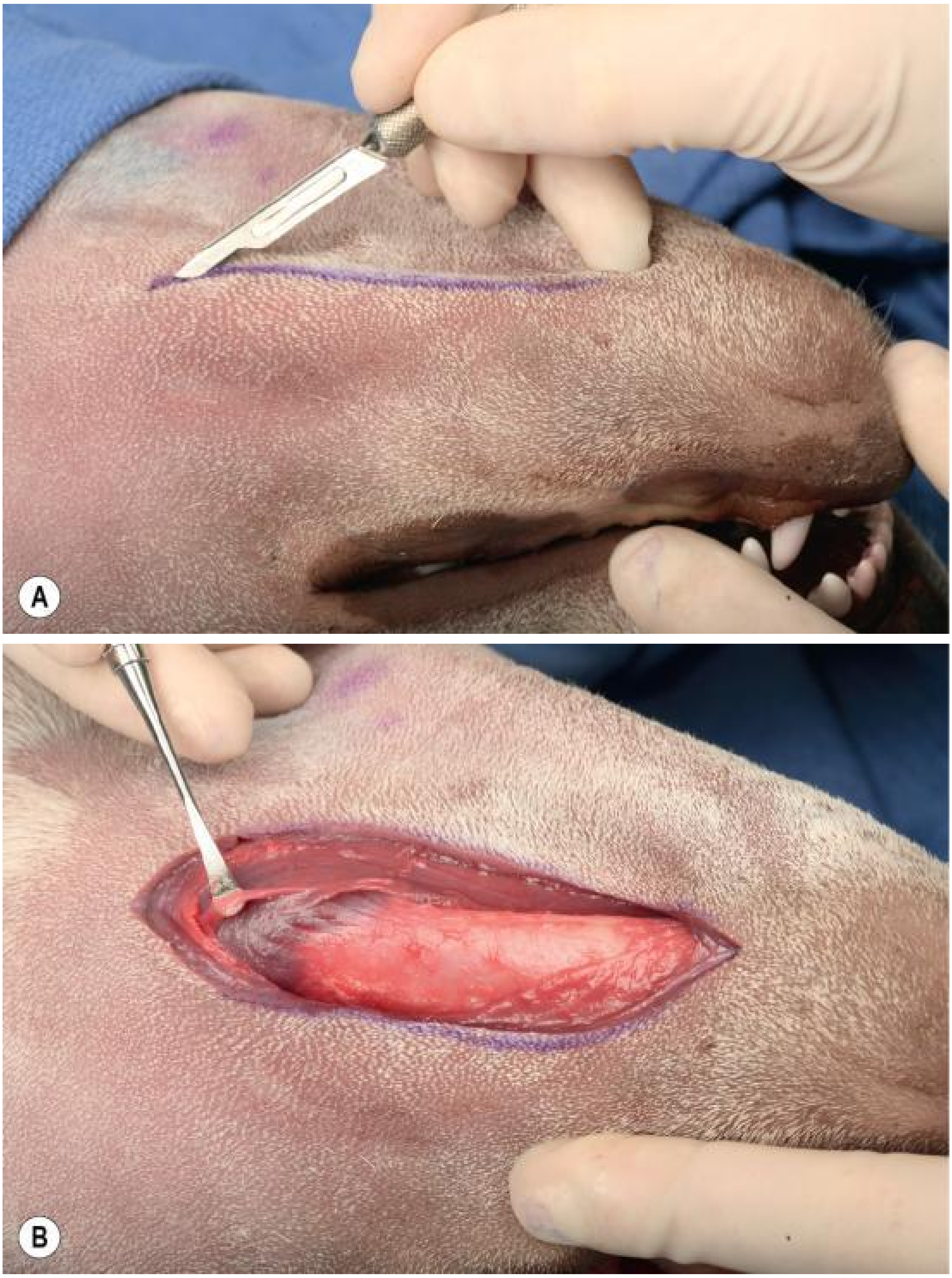
The patient is positioned in dorsal recumbency with the neck extended and supported with a padded area. Forceps may be used to retract the skin of the ventral mandible to better visualize the oral mucosa. The buccal mucosa is incised a minimum of 3–5 mm from the mucogingival junction; the incision extends through the submucosa, muscles, and periosteum (Fig. 29.1).[2,5] Sharp dissection is performed using scalpel or periosteal elevator to incise soft tissues, including the oral mucosa and mentalis and orbicularis oris muscles. Closure is typically performed in two layers. The periosteum and elevated muscles constitute the first layer, and the submucosa and mucosa are closed in a second layer. In certain instances, a single mucosal and submucosal layer is elected.



• FIG. 29.1 Intraoral approach to the incisive part of the body of the mandible and the mandibular symphysis shown in a cadaver head. (**A**) The first incision is made 3–5 mm from the mucogingival junction. (**B**) The incision is then extended through the submucosa, muscles, and periosteum.

Ventral approach to the body of the mandible

The patient is positioned in dorsal recumbency with the neck extended. The skin incision is made medial to the palpable ventral border of the mandible (Fig. 29.2A). If both rostral mandibles have to be exposed, a single midline incision can be made. However, if the entire body of both mandibles needs to be visualized, two separate incisions are recommended. The incised skin is then moved over the prominent ventral border. The subcutaneous fascia, the thin platysma muscle, and the periosteum are then incised with a single full-thickness incision, which ends caudally at the insertion of the rostral belly of the digastricus muscle, thereby avoiding the sublingual branches of the facial artery and vein that cross it (Fig. 29.2B). All soft tissues can then be elevated subperiosteally and retracted medially and laterally, respectively. This includes the mylohyoid muscle on the medial aspect and the insertion of the genioglossus and geniohyoid muscles caudal to the symphysis. Care should be taken as one approaches the mucogingival junction during the elevation and retraction of soft tissues, as perforation can easily occur. It is usually not necessary to elevate the attached gingiva. When exposing the middle and caudal mental foramina, located at the level of the mesial root of the second and third premolar teeth, respectively, care should be taken to retract the associated neurovascular structures.



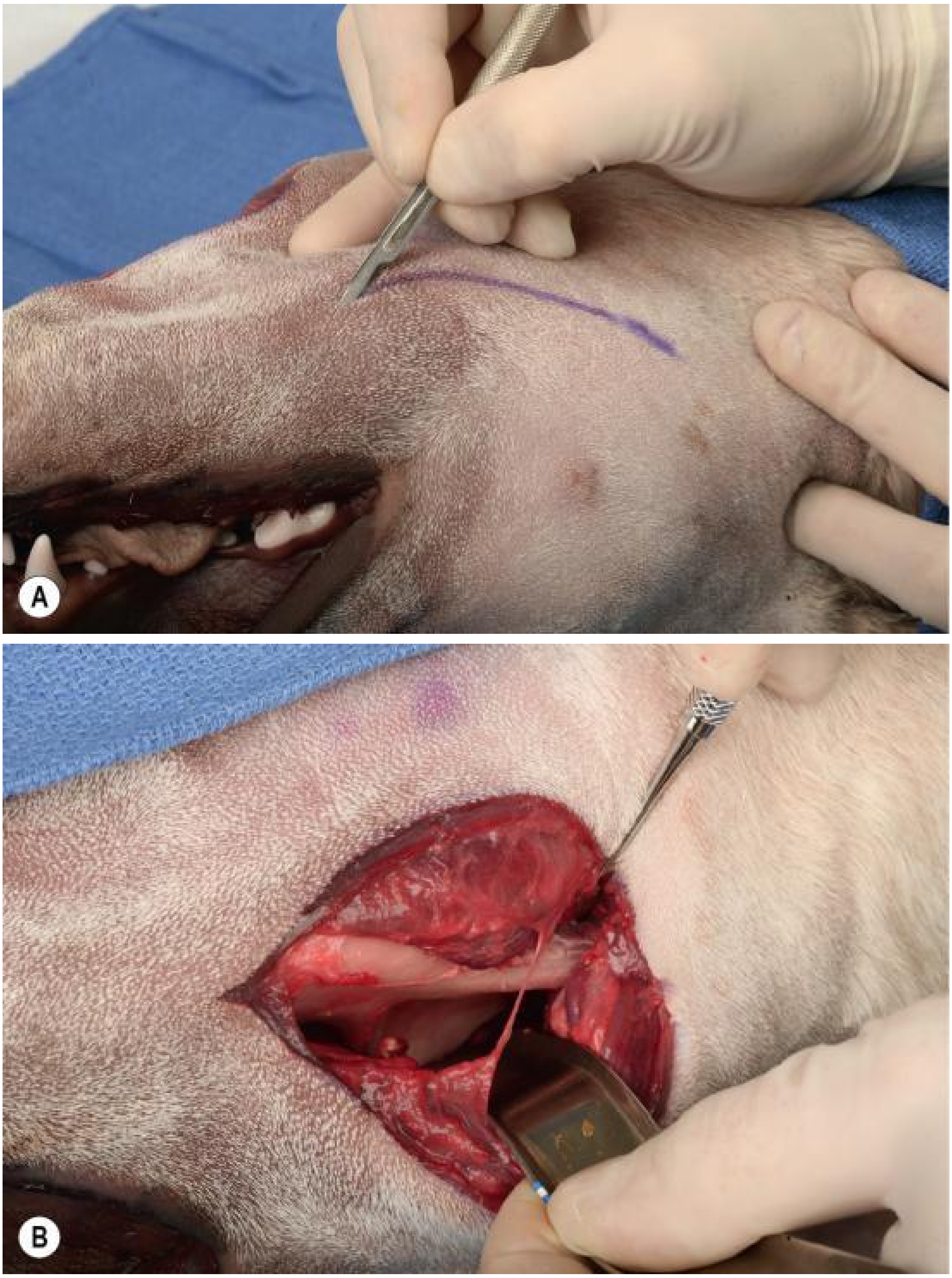
• **FIG. 29.2** Ventral approach to the body of the mandible shown in a cadaver head. (**A**) The skin incision is made medial to the ventral border of the mandible. (**B**) The ventral body of the mandible is exposed, avoiding the sublingual branch of the facial artery and vein.

If the most caudal aspect of the body and the ramus of the mandible need to be exposed, the approach can be extended more caudally.[1]

Closure is usually achieved in three layers. The periosteum and elevated muscles constitute the first layer. The platysma and subcutaneous tissue are closed in a second layer, followed by the skin. The skin suture line is located medial to the ventral border, thereby minimizing the risk of a visible scar. With comminuted fractures, the lacerated oral mucous membrane may require surgical debridement and additional sutures.

Ventral approach to the caudal part of the body and to the ramus of the mandible

The patient is positioned in dorsal recumbency with the neck extended. The skin incision is made medial to the palpable ventral border and angular process of the mandible (Fig. 29.3A). The incised skin is then moved over the prominent ventral border. The subcutaneous fascia and the thin platysma muscle are then incised. The rostral belly of the digastricus muscle on the ventromedial aspect of the mandible and the masseter muscle on the lateral aspect are identified. The intermuscular septum is divided by blunt dissection until the periosteum and insertion of the masseteric fascia are reached. The periosteum is subsequently incised and the two muscles subperiosteally elevated and retracted (Fig. 29.3B).[1] In order to expose the very caudal aspect of the mandible, the insertion of the medial pterygoid muscle has to be elevated. The sublingual artery, a branch of the facial artery and one of the facial veins, which cross the rostral belly of the digastricus muscle in a rostromedial direction, may have to be ligated if they cannot be sufficiently retracted. Care is taken not to damage the facial artery and vein, which are located lateral to the incision.



• FIG. 29.3 Ventral approach to the caudal part of the body and to the ramus of the mandible shown in a cadaver head. (**A**) The skin incision is made medial to the palpable ventral border and angular process of the mandible. (**B**) After incising the subcutaneous fascia and platysma muscles, the intermuscular septum of the masseter muscle and digastricus is separated and retracted. Note the sublingual artery, a branch of the facial artery, and one of the facial veins cross the rostral belly of the digastricus muscle in a rostromedial direction.

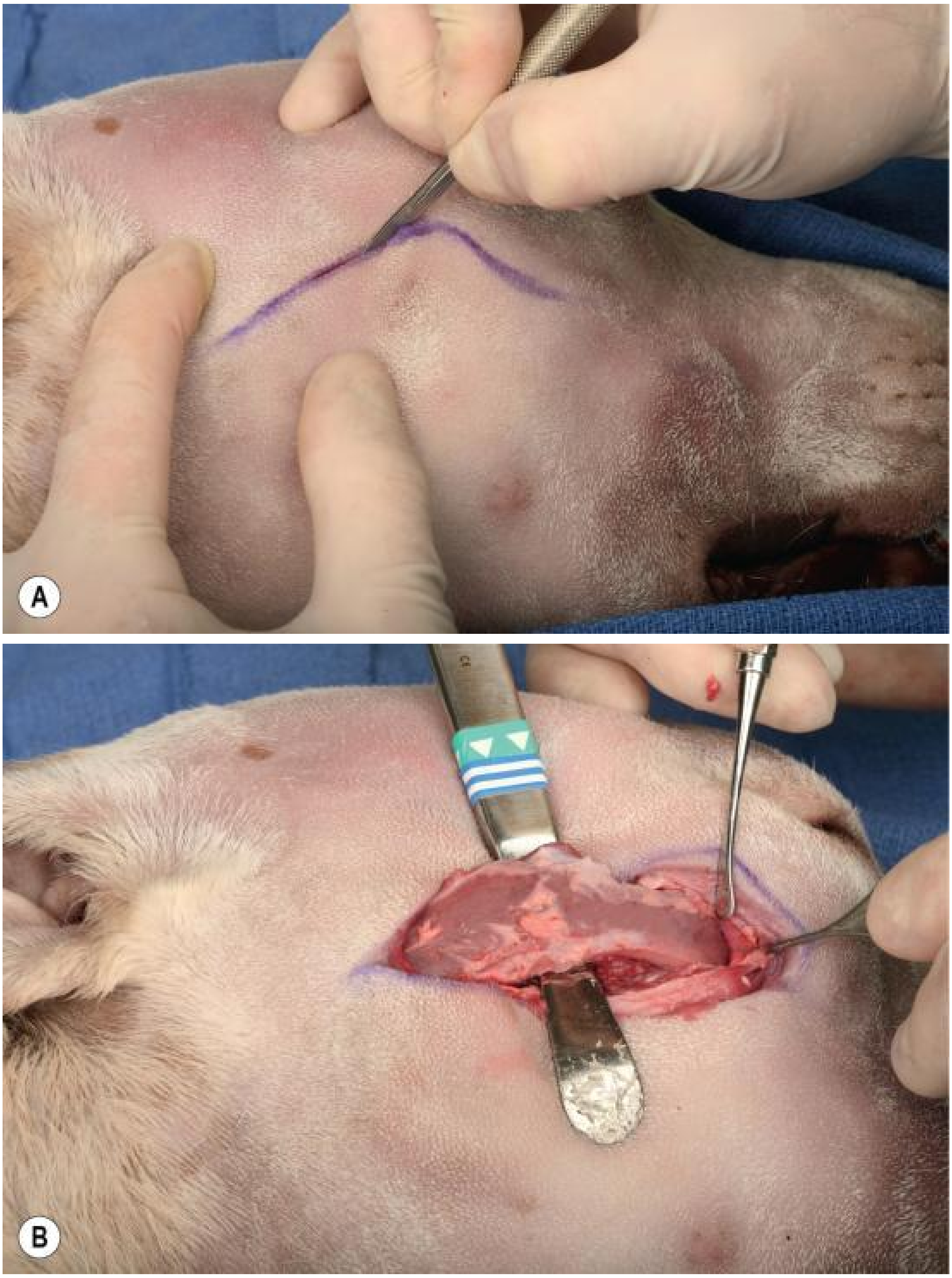
The masseter and temporal muscles can be elevated from the masseteric fossa as far dorsally as the rostrodorsal margin of the coronoid process (often referred to as coronoid crest), to allow miniplate placement in this biomechanically favorable position (see Chapter 33).[6] The exposure on the medial aspect is more limited due to the presence of the mandibular foramen and the neurovascular bundle associated with it.

Closure is usually achieved in three layers. The periosteum and elevated muscles constitute the first layer. The platysma and subcutaneous tissue are closed in a second layer, followed by the skin.

A combination of a ventrolateral approach to the caudal part of the body and to the ramus of the mandible, and a lateral approach to the TMJ, has been described.[4] This requires the subperiosteal elevation of the masseteric muscle from its insertion on the ventral border of the mandible in a dorsal direction, as well as freeing its origin on the zygomatic arch. This can potentially compromise the blood and nerve supply to this muscle. In addition, the two incisions create a relatively narrow strip of skin.

Lateral approach to the zygomatic arch

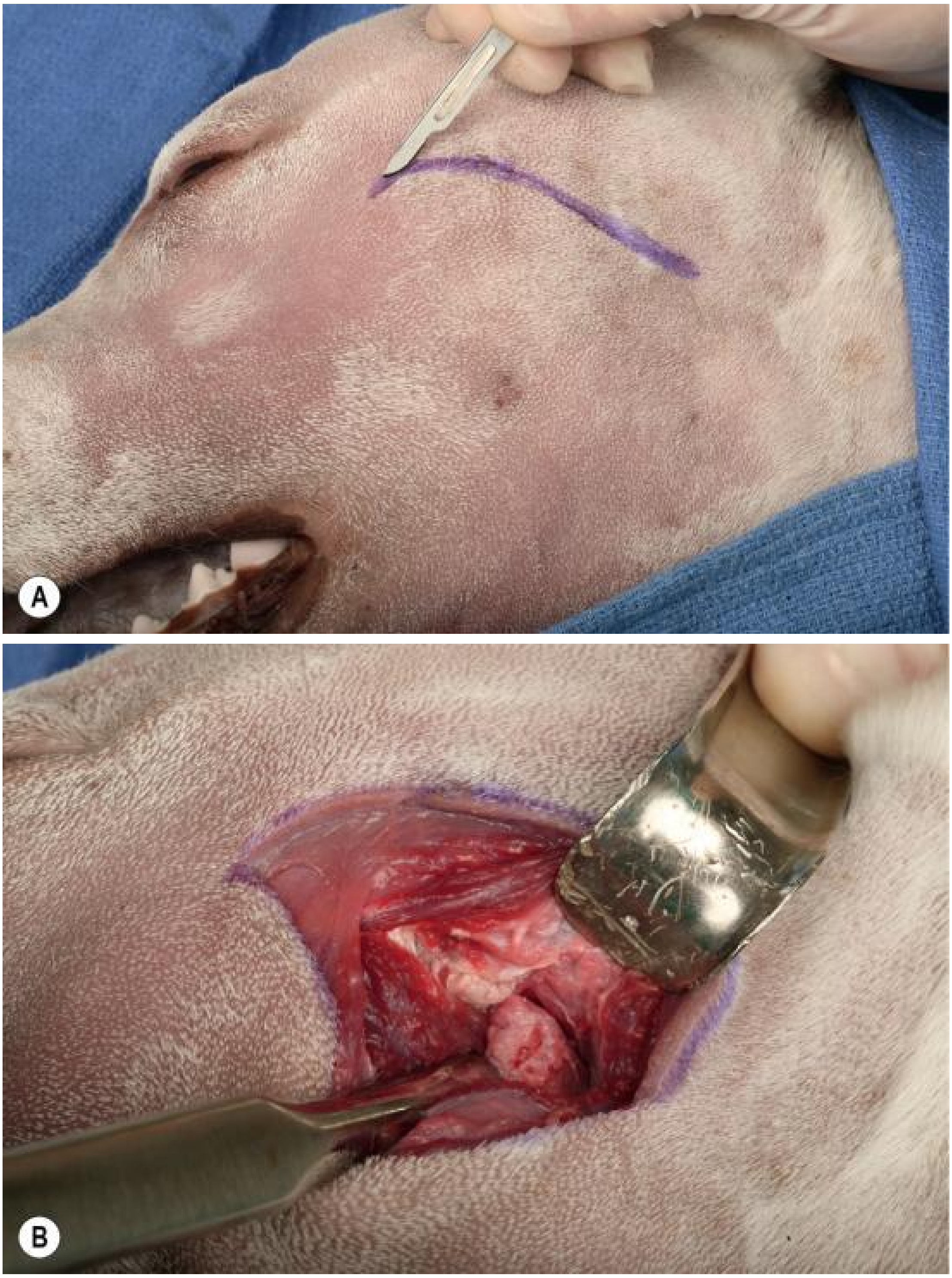
The patient is positioned in lateral recumbency with the neck extended and supported with a padded area. Once the zygomatic arch has been identified by digital palpation, the skin incision follows the ventral or dorsal border of the zygomatic arch and is extended as needed caudally or rostrally (Fig. 29.4A). The periosteum is subsequently incised, and the masseter and temporal muscles are subperiosteally elevated and retracted using scalpel blade and periosteal elevator (Fig. 29.4B). The medial aspect of the zygomatic arch is separated from its muscular attachment using a periosteal elevator. The entire lateral and medial aspects of the zygomatic arch can be easily exposed.[3] Closure is usually achieved in three layers. The periosteum and elevated muscles constitute the first layer. The platysma and subcutaneous tissue are closed in a second layer, followed by the skin.



• **FIG. 29.4** Lateral approach to the zygomatic arch shown in a cadaver head. (**A**) The skin incision follows the ventral or dorsal border of the zygomatic arch. (**B**) Following periosteal incision, the masseter and temporal muscles are elevated and retracted.

Lateral approach to the temporomandibular joint[4]

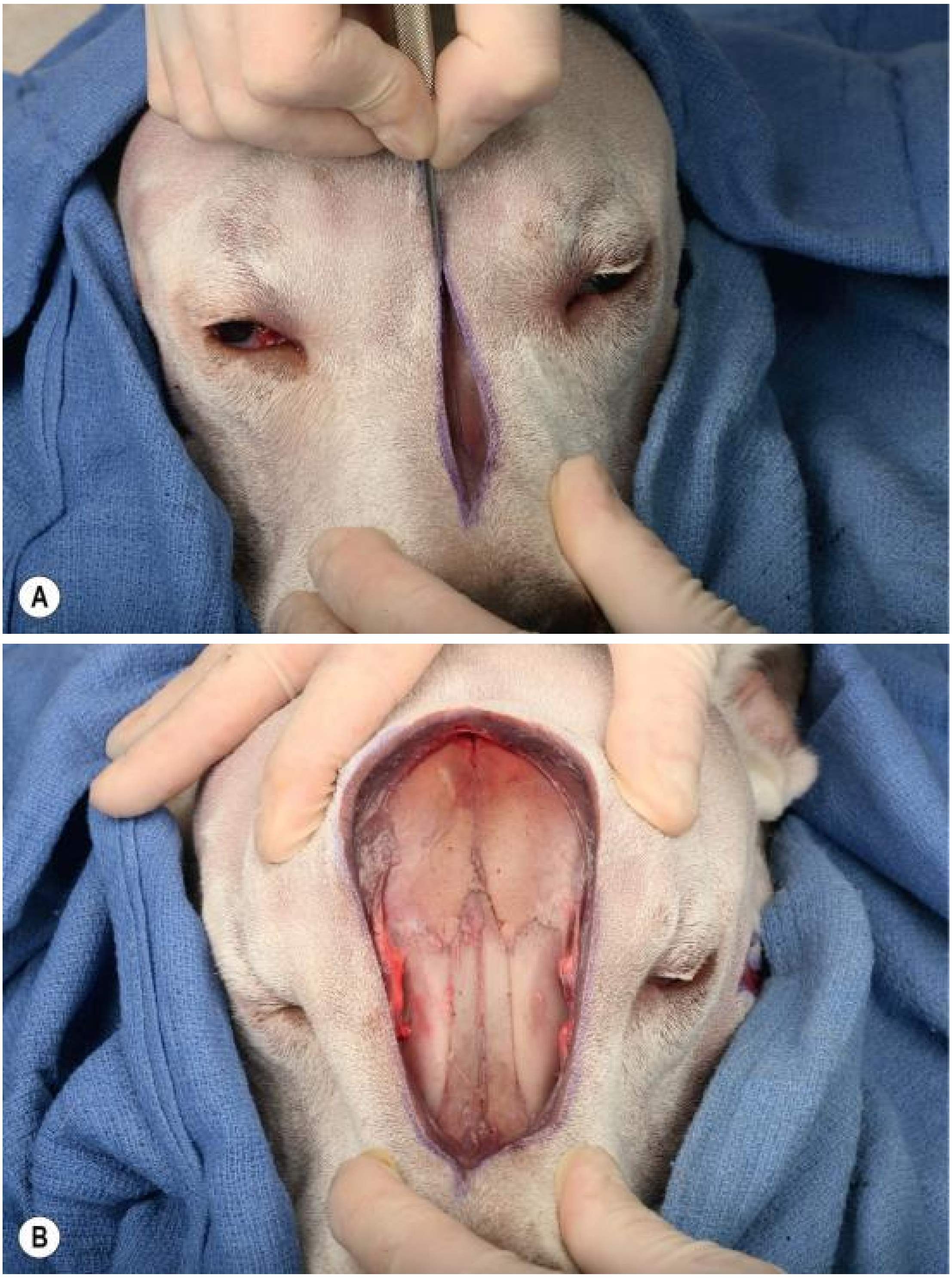
The patient is positioned in lateral recumbency with the neck extended and supported with a padded area. Once the zygomatic arch has been identified by digital palpation, the skin incision follows the ventral border of the zygomatic arch and crosses the TMJ caudally (Fig. 29.5A). The platysma muscle, directly under the skin, is incised on the same line. The origin of the masseter muscle is incised on the ventral border of the zygomatic arch and elevated using a periosteal elevator. The masseter muscle is retracted in a rostroventral direction, avoiding nearby neurovascular structures. The TMJ is identified on the caudal aspect of the zygomatic arch (Fig. 29.5B). If possible (i.e., the jaw is not locked), opening and closing the jaw may help identify the joint. The joint capsule is incised rostrolaterally, and the condylar process is partially visualized by manipulating the mandible. Closure is usually achieved in three layers. The periosteum and elevated muscles constitute the first layer. The platysma and subcutaneous tissue are closed in a second layer, followed by the skin.



• FIG. 29.5 Lateral approach to the temporomandibular joint (TMJ) shown in a cadaver head. (**A**) The skin incision follows the ventral border of the zygomatic arch and crosses the TMJ caudally. (**B**) Following elevation and retraction of the origin of the masseter muscle and incising the fibrous capsule, the TMJ is identified.

Extraoral approach to the maxillae, nasal bones, and frontal sinuses[7]

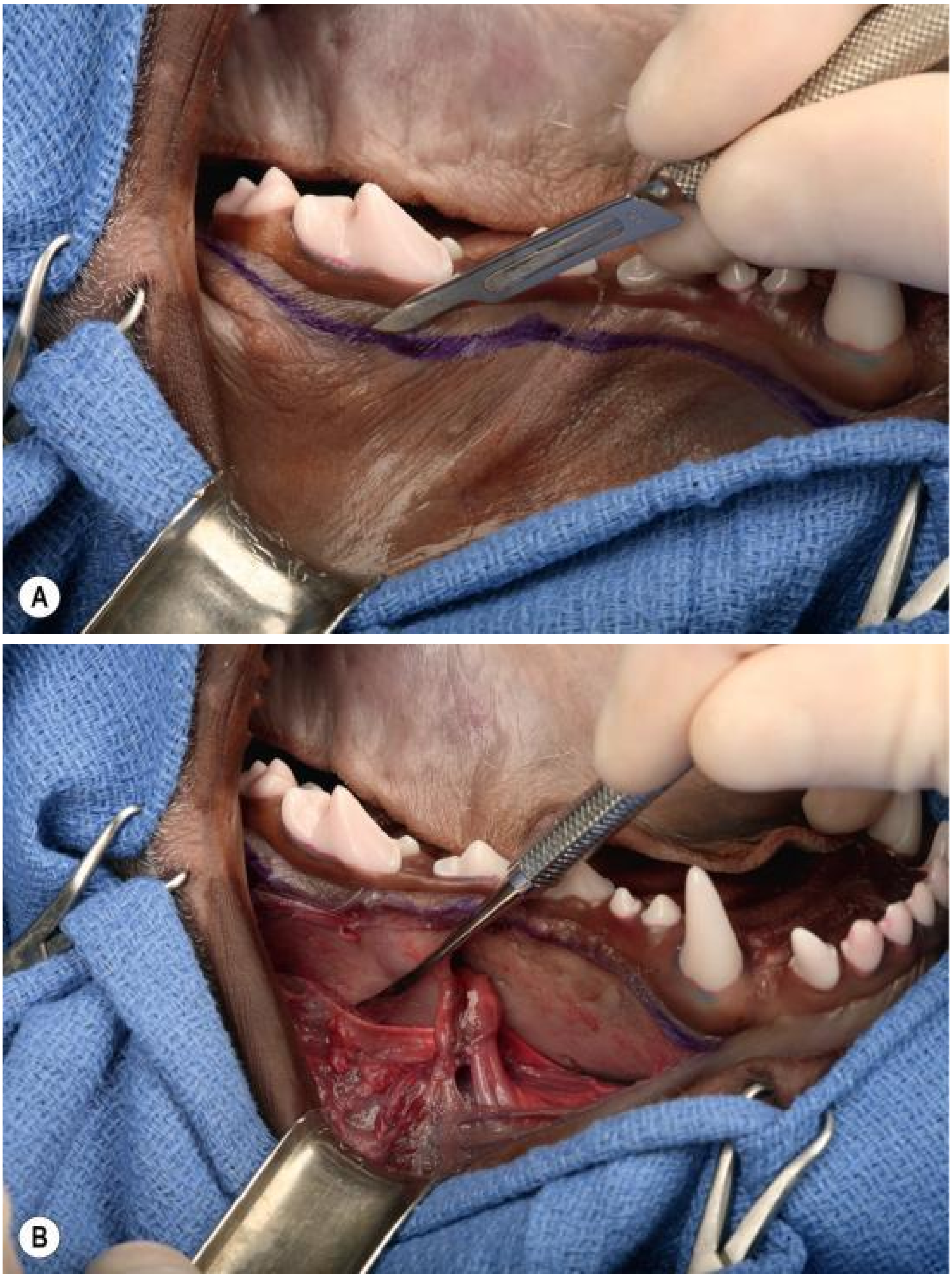
The patient is positioned in sternal recumbency with the head elevated on a padded area. A dorsal midline skin incision is made (Fig. 29.6A). If the fractures are at the maxillae or nasal bones, the incision is made beginning at the caudal end of the incisive bones and extending caudally to the area parallel to the zygomatic process of the frontal bones, midline between the orbits. If the frontal sinus must be exposed, the dorsal midline incision is centered on the location that parallels the zygomatic process of the frontal bone, beginning from midline between the orbits and extending caudally to the rostral end of the sagittal crest. The periosteum is incised on the midline and reflected using a periosteal elevator to expose the bones (Fig. 29.6B). Care should be taken, as the bone plates in these areas are very thin and can be perforated in the fractured area with excessive force. Closure is performed in two layers: the periosteum and connective tissue in one layer and the skin in the second layer. Often, a stent bandage is used to prevent postoperative subcutaneous emphysema.



• FIG. 29.6 Extraoral approach to the maxillae, nasal bones, and frontal sinuses shown in a cadaver head. (**A**) A dorsal midline incision is made depending on the location of the fracture. (**B**) Following periosteal incision, the tissues are retracted to expose the bones.

Intraoral approach to the maxilla[2,3,5]

The patient is positioned in dorsal or lateral recumbency with the neck extended and supported with a padded area. The incision is usually placed 3–5 mm away from the mucogingival junction, leaving the unattached mucosa on the alveolus to facilitate closure (Fig. 29.7A). This tissue is elastic and contracts following incision, although during closure the tissue can be grasped and holds sutures well. Alternatively, a sulcular incision using a scalpel blade and elevation of the attached gingiva and the mucosa with a periosteal elevator, as performed for surgical extractions, could be performed. The incision extends as far caudally and rostrally as needed, depending on the fractured area, to provide exposure.[2,3] Using a periosteal elevator, subperiosteal dissection and elevation proceeds to elevate the mucosa, submucosa, and facial muscles, exposing the underlying tissue. The infraorbital neurovascular bundle is identified by dissecting medially and laterally to the location of the infraorbital canal, working toward the bundle (Fig. 29.7B). The bundle is encountered and the periosteum is dissected completely around the foramen if needed. In general, the infraorbital artery, vein, and nerves should be avoided, as damaging them may cause impairment of blood supply to the fractured area and loss of sensation. If the incision should extend to the caudal maxilla, the facial vein will be encountered as it joins the deep facial vein near the levator nasolabialis muscle. The rostral aspect of the zygomatic bone will be exposed at the level of the maxillary molar teeth. If needed, the masseter muscle attachments can be elevated using a periosteal elevator. Closure is usually achieved in one layer for the submucosa and mucosa in a simple interrupted pattern.



• **FIG. 29.7** Intraoral approach to the maxillae shown in a cadaver head. (**A**) The incision is placed 3–5 mm away from the mucogingival junction and extends caudally and rostrally as needed. (**B**) Subperiosteal dissection using a periosteal elevator exposes the underlying tissues. Note the location of the infraorbital foramen and its associated vasculature, as damage to these structures should be avoided.

Postoperative care and assessment

Postoperative care is dependent on the fracture site and severity as well as on the general health of the patient. However, general guidelines apply to all mandibular and maxillofacial trauma repairs. During the immediate postoperative period, soft food and multimodal pain management are indicated, and mouth play or chewing activity should be avoided. Thereafter the patient should be stimulated to use its jaws actively. Antibiotic therapy may be indicated, depending on the extent of the trauma. Postoperative nutritional support via feeding tube should be considered, especially in cats. If an extraoral approach was used, the skin sutures should be removed after 10–14 days. Absorbable sutures are typically used for an intraoral approach, so suture removal is not necessary in these cases. A 0.05%–0.12% chlorhexidine gluconate solution can be used as an oral antiseptic rinse to keep the wound or incision area clean and to limit the negative effects of food accumulation. As a general rule, maxillofacial fracture repair is evaluated radiographically at 6 weeks postoperatively. Following confirmation of adequate healing, the pet may return to normal eating, playing, and chewing activities.

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CHAPTER 30

Symphyseal separation and fractures involving the incisive region

Ulrike Matis, Roberto Köstlin

Classification and incidence

Mandible

Injury to the incisive region of the mandible may involve the mandibular symphysis and the alveoli of the first incisor, second incisor, and/or third incisor teeth, as well as the canine teeth. In dogs, alveolar fractures and separation of the mandibular symphysis occur with nearly equal frequency, whereas in cats, symphyseal separation is by far the most common injury. The second most frequent injury is fracture of the distal aspect of the alveolus of the canine tooth; in cats, the alveoli of the incisor teeth are seldom involved.

The percentage of mandibular injuries involving the rostral region ranges from 25% to 45% in dogs and from 56% to 78% in cats.[1-9] Symphyseal separations in dogs and cats have been classified based on the amount of tissue damage.[10] In type I lesions no soft tissue laceration is present. Type II lesions are characterized by soft tissue laceration, while in type III lesions major soft tissue trauma, comminution and exposure of bone, and fractured teeth are present. The proportion of open fractures among all fractures in the incisive region has been reported to be 90% in the dog.[2]

Maxilla and incisive bone

In dogs, injury to the rostral maxilla involves mainly alveolar fractures with luxation or subluxation of the incisor and/or canine teeth. In contrast, midline separation of the hard palate is the most common injury in cats, with about a third of all injuries involving the interincisive suture and the remainder the maxillary palatine process and the horizontal part of the palatine bone.[5] Overall, maxillary fractures occur less frequently than mandibular fractures. Of 171 canine and 428 feline jaw fractures treated at a teaching hospital in Munich, only 16% and 37% involved the maxilla, respectively.[3,5]

Concomitant injuries

Concomitant trauma to the skull and brain are common in animals with jaw injuries, particularly maxillary fractures. Ocular injuries may complicate the clinical presentation. Blunt trauma to the thorax as well as limb injuries may also occur. Multiple injuries are particularly common in cats that incur jaw fractures, which occur mainly after falling from a great height; according to one author, as many as 70% of these cats may be affected by multiple injuries.[5] In dogs, the most frequent causes of multiple injuries are being hit by a car and fighting with another dog. Thus, a thorough physical examination is imperative to recognize and treat life-threatening injuries promptly. Thoracic radiographs are of great importance in cats to assess anesthetic risks and should be obtained routinely in trauma patients.

Diagnosis

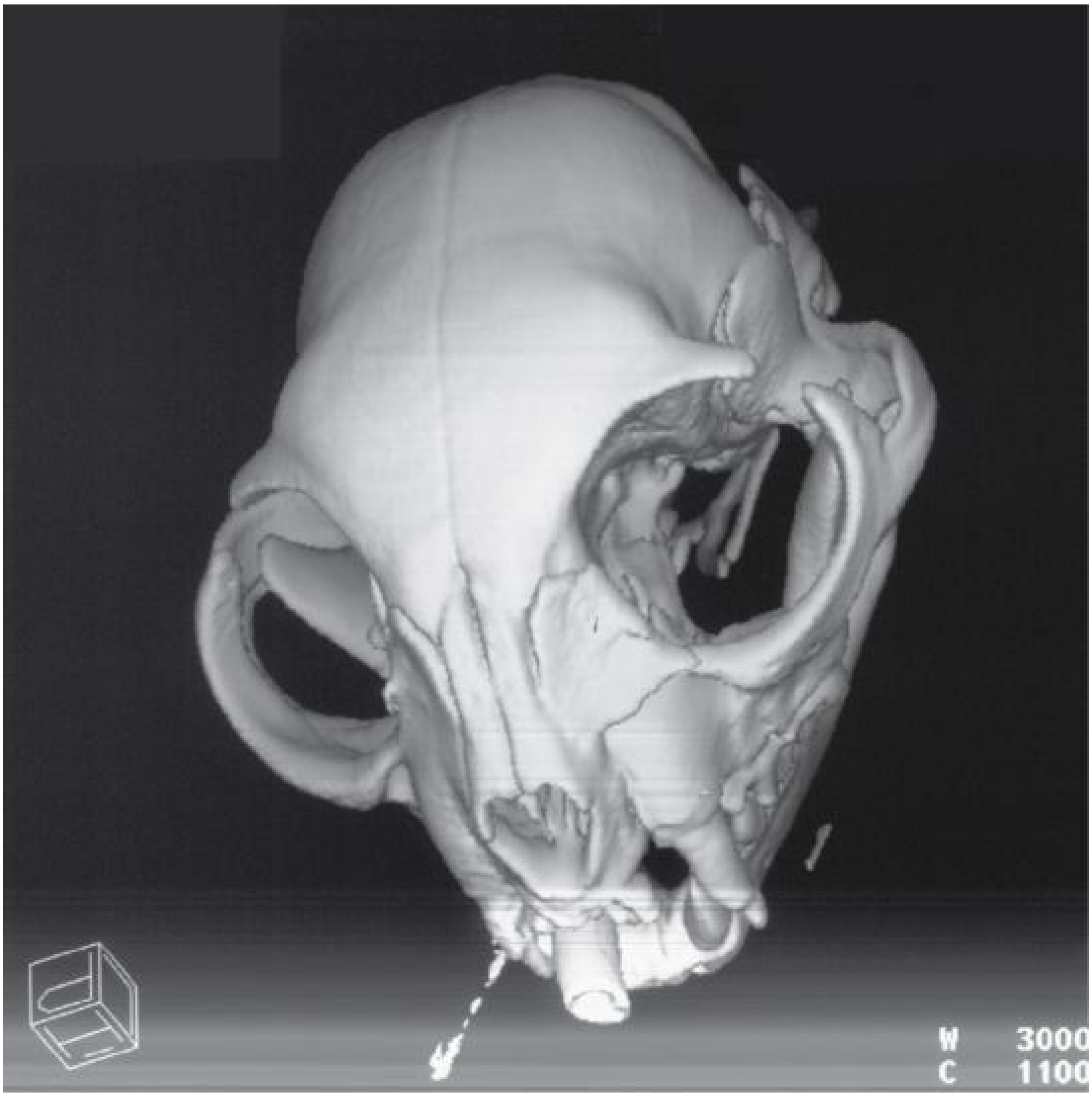
Clinical findings

Characteristic signs of jaw injuries are malocclusion, abnormal movement, and abnormal position of teeth. Nonspecific signs include pain, epistaxis, ptyalism, often with blood-tinged saliva, and anorexia or reluctance to eat. A diagnosis can usually be made based on the clinical signs. However, radiography or other imaging modalities are necessary to determine precisely the course of the fracture(s).

Diagnostic imaging

Correct radiographic views are generally obtained by using the parallel technique; the X-ray beam is at a right angle to the film, which is parallel to the body part being radiographed. Because this is not possible within the oral cavity, the bisecting angle technique may be used to provide a reasonable radiographic image with linear accuracy, although the dimensional accuracy of the parallel technique may be lacking. Both techniques can be used for extraoral views. For images of the incisive region, intraoral projections with the X-ray beam at right angles to the film may also be adequate.

Complex fractures that involve not only the rostral jaw area but also the skull and temporomandibular joint, in particular, are best imaged using conventional or cone-beam computed tomography (CT). Magnetic resonance imaging is useful for the assessment of soft tissue lesions. Three-dimensional reconstruction using transverse CT images provides rapid topographical information, which is needed for establishing a prognosis and treatment plan for patients with multiple fractures (Fig. 30.1). However, fissures and small cracks that are easily seen on transverse CT images may be obscured and therefore missed in three-dimensional reconstructions.



• **FIG. 30.1** Three-dimensional reconstruction of multiple skull fractures in a cat with a fracture of the left maxilla and an impression fracture further caudally in the parietal region. Source: (Reprinted with permission from: Beck W, Hecht S, Matis U. Dreidimensionale Rekonstruktion aus CT-Transversalbildern zur Darstellung komplexer Schädelfrakturen bei der Katze. *Tierärztl Prax*. 2000;28:219–224.)

Anesthetic considerations

Life-threatening injuries must be ruled out and the patient stabilized before anesthesia is carried out (see Chapter 4). Pharyngotomy endotracheal intubation (see Chapters 4 and 61) is typically not required for isolated fractures of the rostral mandible or maxilla. When the jaws must be immobilized in a closed or near-closed position, administration of medications that are known to cause vomiting is contraindicated because of the risk of aspiration. One must also ensure that the patient can breathe adequately. This is particularly important in animals with complex fractures of the maxilla.

Surgical anatomy

Topographical anatomy

The mandibular symphysis joins the bodies of the right and left mandibles and is composed of fibrocartilage and connective tissue, which remain as such for life without being converted to bone *(synchondrosis et sutura intermandibularis)*. The dorsal edge of the incisive region *(pars incisiva)* contains the alveoli of the incisor and canine teeth. The mandibular incisor and canine teeth are closer together than those of the maxilla. This enables the lingual surface of the maxillary incisor teeth to slide past the buccal or labial surface of the mandibular incisor teeth upon jaw closure. However, there are exceptions to the normally secodont occlusion of carnivores; maxillary brachygnathism is common in brachycephalic dog and cat breeds, and a relative maxillary prognathism may be present in dolichocephalic dog breeds (e.g., dachshund, collie, and some of the terrier breeds).

The maxillary incisor teeth are anchored in the alveoli *(alveoli dentales)* of the incisive bone *(os incisivum)*. Its nasal process *(processus nasalis)* forms part of the rostral border of the nasal cavity, and its palatine process *(processus palatinus)*, together with the palatine process of the maxillary bone *(processus palatinus)* and the palatine bone *(os palatinum)* caudally, form the hard palate. The interincisive suture and the median palatine sutures *(sutura palatina mediana)* of the palatine processes of the maxillary bones and of the palatine bones are where these three structures unite at the midline to form a bony plate that serves as the roof of the oral cavity as well as the floor of the nasal cavity.

The roots of the incisor and canine teeth both in the upper and lower jaws are surrounded by only a thin layer of bone. Thus, it can be difficult to secure implants, particularly in the incisive region of the mandible. In the upper jaw, the roots of the incisor teeth are oval and laterally compressed in cross-section and are situated in separated alveoli, which converge medially. In contrast, the smaller incisor teeth of the lower jaw have roots that are straighter and sit in alveoli that may not be fully separated from each other. From mesial to distal, that is from the first to the third incisor teeth, the teeth increase in strength and size in both the mandible and maxilla. When the mouth is closed, the mandibular canine teeth occupy the interproximal space between the maxillary third incisor and canine teeth, and the maxillary third incisor teeth occupy the interproximal space between the third incisor and canine teeth of the lower jaw.

The canine teeth are located between the third incisor and first premolar teeth. The conical and pointed crown of a canine tooth is slightly curved caudally. In adult dogs, the large root of the canine tooth overlays the roots of the first and second premolar teeth. In cats, the incisor teeth are very small and thin, and the dagger-like canine teeth point slightly outward. As in dogs, the mandibular canine teeth of cats are positioned between the maxillary third incisor and canine teeth when the mouth is closed and the occlusion is normal. The maxillary canine teeth, whose tips have a slightly buccal orientation, occlude between the mandibular canine and first premolar teeth.

In addition to establishing correct dental occlusion, care must be taken to preserve the vascular perfusion and innervation of the tissue. The infraorbital artery *(arteria infraorbitalis)*, which is a branch of the maxillary artery *(arteria maxillaris)*, supplies the rostral part of the maxilla. The inferior alveolar artery *(arteria alveolaris inferior)*, also a branch of the maxillary artery, supplies the rostral part of the mandible. The infraorbital nerve *(nervus infraorbitalis)*, which is the largest branch of the maxillary nerve *(nervus maxillaris)*, innervates the maxillary incisor area including the canine teeth, and the mandibular incisive region is innervated by the inferior alveolar nerve *(nervus alveolaris inferior)*, which is a branch of the mandibular nerve *(nervus mandibularis)*. In the mandible, the majority of vessels and nerves exit the mandibular canal through three mental foramina, the most rostral of which is situated below the second incisor tooth. The middle and caudal foramina are located at the level of the second and third premolar teeth in dogs and interproximally between the canine and third premolar teeth and at the level of the third premolar tooth in cats. Provided that these structures are not already damaged by the initial trauma, care must be taken to preserve them during preparation, repositioning, and fixation of the fracture.

Biomechanical considerations

As mentioned before, the roots of the canine and incisor teeth occupy almost the entire rostral region of the mandible and maxilla. Thus, the stabilization technique used must not depend on implants that are anchored in the bone. To obtain maximum stability with small implants, naturally occurring biomechanical forces must be taken advantage of. In humans, the forces exerted on the mandible and maxilla during functional use have been investigated, and it appears that the biomechanical conditions are similar to those in dogs and cats. It is known that the greatest tractional forces occur along the alveolar process of the maxilla and mandible and that, in turn, the greatest compression forces occur along the ventral border of the body of the mandible. The bending moments on the mandible increase from cranial to caudal, whereby not only do tractional and compression forces occur but so do shearing and torsional forces. Torsional forces are particularly prominent rostral to the canine teeth and are greatest in the mandibular symphysis. Thus by following the principles of tension band fixation, stabilization techniques in the rostral region of the jaw should be aimed at preventing the separation of bone fragments in the alveolar region.

Therapeutic decision-making

Conservative management versus surgical treatment

The goal of treatment is restoration of normal function and occlusion, although anatomically perfect positioning of all the teeth may not always be possible. For practical purposes, this goal is achieved provided that unimpaired jaw closure is possible. The extraction of a tooth may be necessary to accomplish this. The choice of conservative management or surgical treatment depends on the degree of malocclusion and the stability of the fracture. Nondisplaced or only mildly displaced fractures that are reasonably stable and allow normal occlusion, as judged by the position of the canine teeth, can be managed conservatively. For example, the majority of traumatic injuries to the hard palate can be left to heal on their own, and other minimally displaced and fairly stable fractures of the maxilla do not require surgical intervention. Details of conservative treatment are described in Chapter 31. Injuries of the rostral mandible usually require surgical repair, which is generally minimally invasive.

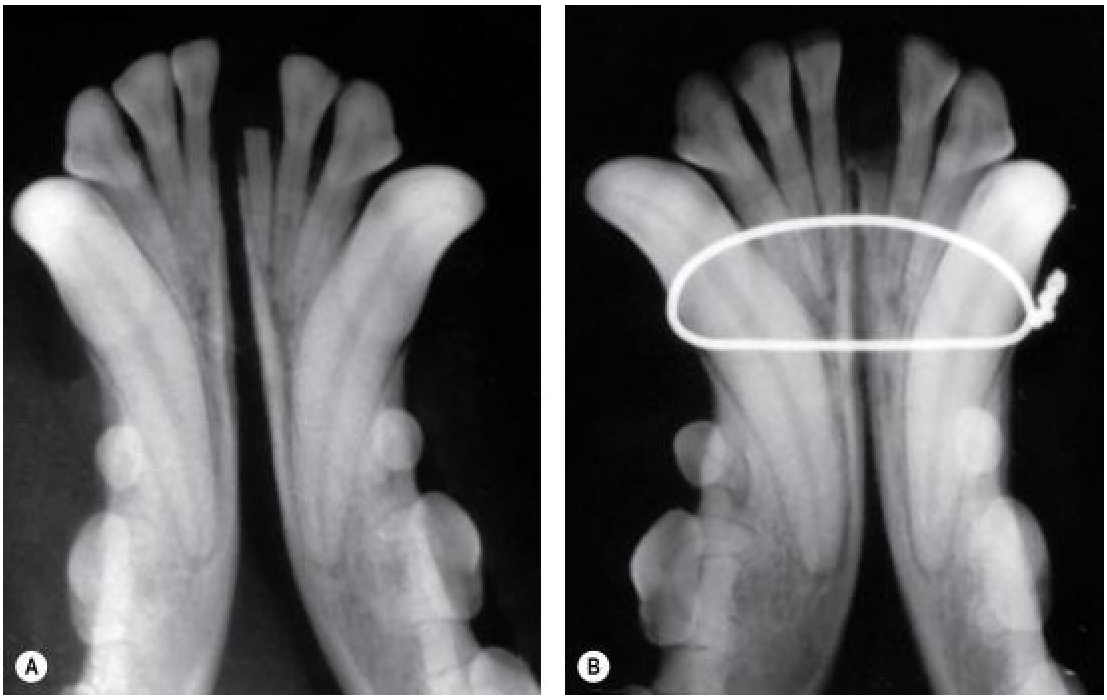
Surgical techniques

Separation of the mandibular symphysis

Mandibular symphysis separation injuries are usually repaired using the circummandibular cerclage wiring technique.

Cerclage wiring

Various modifications of the cerclage wiring technique exist.[11] The authors prefer an intraoral approach. The lower lip is retracted, and a 20G or 18G hypodermic needle is inserted through the junction of the attached gingiva and alveolar mucosa caudal to the canine tooth. It is advanced to the ventral surface of the symphyseal region. A piece of 24G or 22G orthopedic wire is inserted into the needle, which is then withdrawn, leaving the wire pointing in the desired direction. The needle is then inserted through the oral mucosa behind the canine tooth on the opposite side in an identical manner, and the wire is located and inserted into the needle in a retrograde fashion. By advancing the wire and withdrawing the needle, the wire is advanced through the tissue without damaging it. The free ends of the cerclage wire are lightly twisted together caudolateral to one of the canine teeth at the interalveolar margin. After repositioning of the mandibles and confirming proper occlusion, the cerclage wire is tightened. The wire twist is shortened to two or three twists and, observing the direction of the twists, bent and positioned along the gingiva to prevent trauma to the tongue and lower lip (Fig. 30.2). In contrast to the ventral twist technique,[11] this method allows removal of the wire after consolidation of the mandibular symphysis without making a skin incision. For removal, wire cutters are used to cut the cerclage wire on the side opposite the twist at the gingival margin. The twist is then grasped and the wire extracted. In this fashion, the contaminated intraoral section of wire is not pulled through the tissue during extraction. Using an alternative intraoral technique, the ends of a wire are passed through drill holes made below the roots of the third incisor tooth on each side using Kirschner wire directed aborally. The free ends of the wire are then guided around the distal surface of the ipsilateral mandibular canine teeth and twisted together under tension.[12] Care must be taken to avoid damage to the roots of the incisor teeth when this technique is used. The cerclage wire is left in place for 6–12 weeks. In carnivores, the mandibular symphysis is usually not ossified, and therefore consolidation should be assessed by clinical examination rather than by radiography.



• FIG. 30.2 Separation of the mandibular symphysis and fracture of the first incisor tooth in a 3-year-old dachshund: (**A**) before and (**B**) 3 months after extraction of the first incisor tooth and stabilization of the symphyseal separation using circummandibular cerclage wire. Even with clinical resolution of the symphyseal separation, ossification of the mandibular symphysis does not occur.

Composite resin-bonded wire fixation

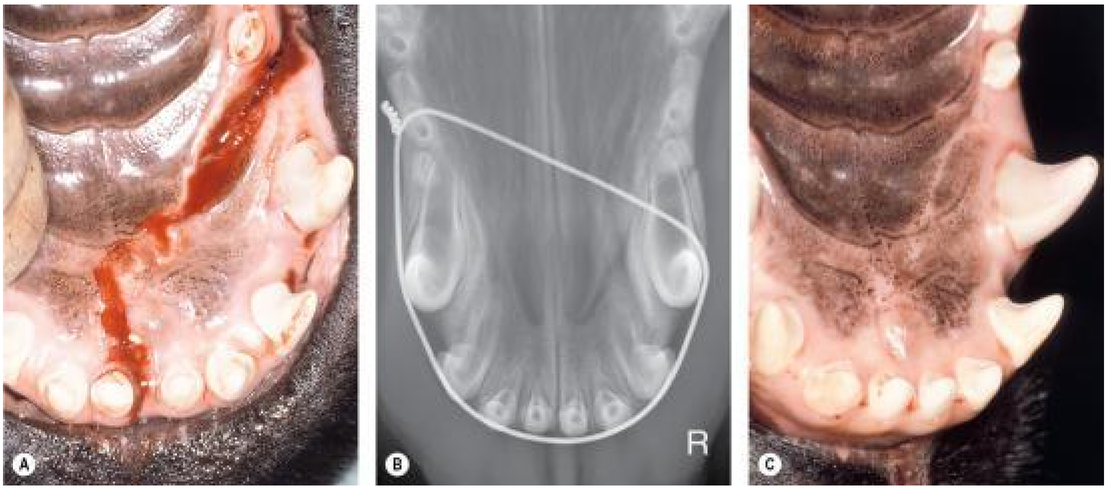
In cases where circummandibular cerclage does not result in normal occlusion because of bone loss or an additional injury, the correct distance between the canine teeth can be maintained by placing a wire between the teeth; the wire is looped around one canine tooth and twisted before being looped around the other canine tooth. The wire is secured to the teeth using composite resin, which is also used to cover the wire and its knot to prevent the wire from slipping and to avoid injury to the oral mucosa by the sharp edges of the knot.[13]

Palatine fractures

Suturing the gingiva is usually adequate for adaptation of bone fragments in relatively stable palatine fractures. For unstable fragments that may result in malocclusion, placement of an intraoral composite and wire splint or internal reduction and fixation using a transfixation pin and locking bone plate and screw systems respectively is recommended.

Cerclage wiring

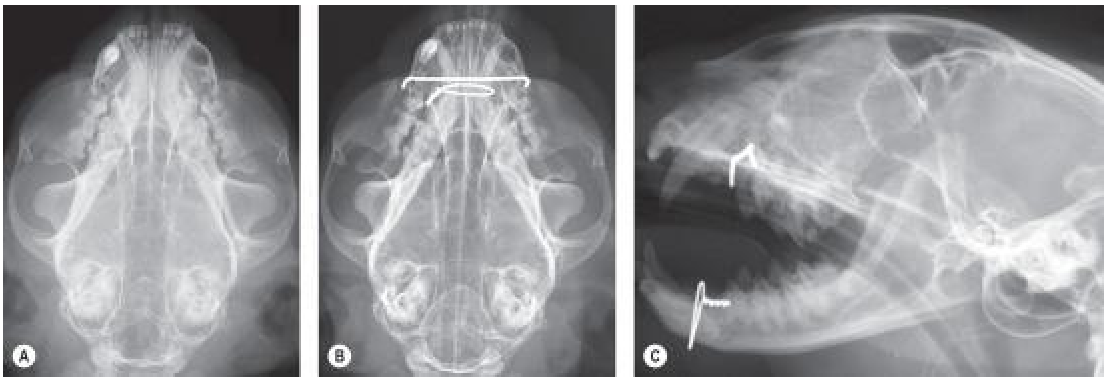
Oblique fractures, in which the isolated bone segment is displaced rostrolabially, can be stabilized by drawing the fragment in a contralateral direction with cerclage wire. After removing blood clots and devitalized tissue, the fragment is reduced with pointed reduction forceps. A cerclage wire is placed over the facial surface of the incisor teeth and/or the buccal surface of the canine teeth and perpendicular to the palatine fracture line over the hard palate. Depending on the direction of the fracture line, the wire is placed caudal to the first premolar tooth on the opposite side or between the roots of the second premolar tooth. The two ends of the wire are then twisted together alongside the first premolar tooth (Fig. 30.3).



• **FIG. 30.3** (**A**) Fracture of the incisive bone and maxillary palatine process in a 1-year-old German shepherd dog; (**B, C**) the fractures were stabilized with cerclage wire placed over the facial surface of the incisor teeth and buccal surface of the canine teeth and between the roots of the contralateral second premolar tooth.

Transfixation pin

Fracture lines that run sagittally in the midline can be stabilized using a Kirschner wire, placed caudal to the canine teeth. After cleaning of the fracture surfaces and compression of the fragments using pointed reduction forceps, the wire is advanced in a transverse direction through the thin palatine bony lamella until it appears on the contralateral side. The free end of the wire is bent to form a hook, and the wire is pulled back until the hook sits tightly against the maxilla. The other end of the wire end is also bent so that it lies tightly against the bone before it is shortened with side cutters (Fig. 30.4). Dislocation of the fragments does not normally occur when the pointed reduction forceps are removed. However, if it does occur, a figure-of-eight cerclage wire is placed around the bent ends of the wire and across the palate. The ends are twisted together laterally so that they do not injure the tongue or lips. Normal occlusion is achieved by closing the jaws during tightening of the cerclage wire.



• **FIG. 30.4** (**A**) Fracture of the median palatine suture and separation of the mandibular symphysis in a 1-year-old cat; (**B**) ventrodorsal and (**C**) laterolateral radiographic views obtained after stabilization using a maxillary transfixation pin and circummandibular cerclage wire.

Plate fixation

Plates are preferred for osteosynthesis of fractures that cannot be adequately stabilized using cerclage wire, transfixation pinning, or intraoral composite and wire splints.[14] In contrast to the mandible, plates can also be used in the incisive region of the upper jaw. Locking bone plate and screws systems are particularly well suited for this purpose because they allow a fixed-angle construct of the plate with screws placed between the tooth roots or proximal to the roots (see Chapter 33).

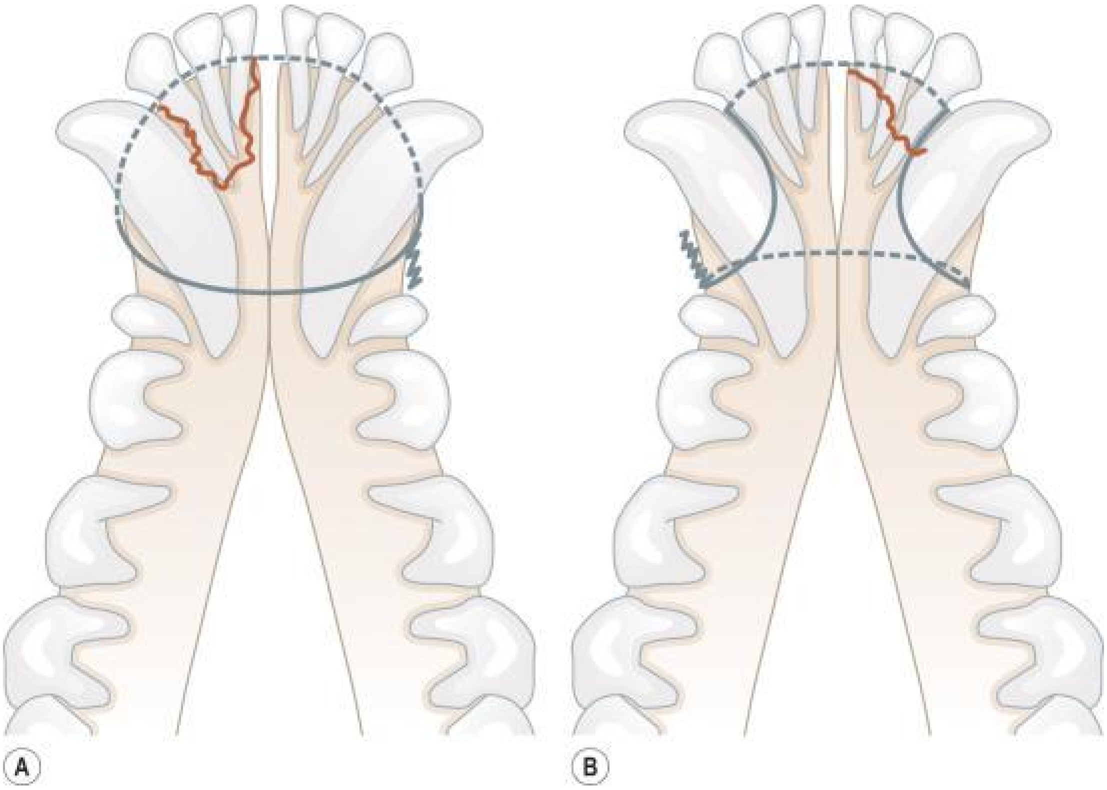
Intraoral acrylic or composite splints

Intraoral splints, which are made of self-hardening acrylic or composite, can be used to stabilize fractures of the hard palate and maxilla. The acrylic or composite can be held in position with transmaxillary wires, the free ends of which are bent to form hooks. More commonly, composite splints can also be attached to the teeth of the maxilla (see later). The use of wire and acrylic or composite splints is addressed in Chapter 31.

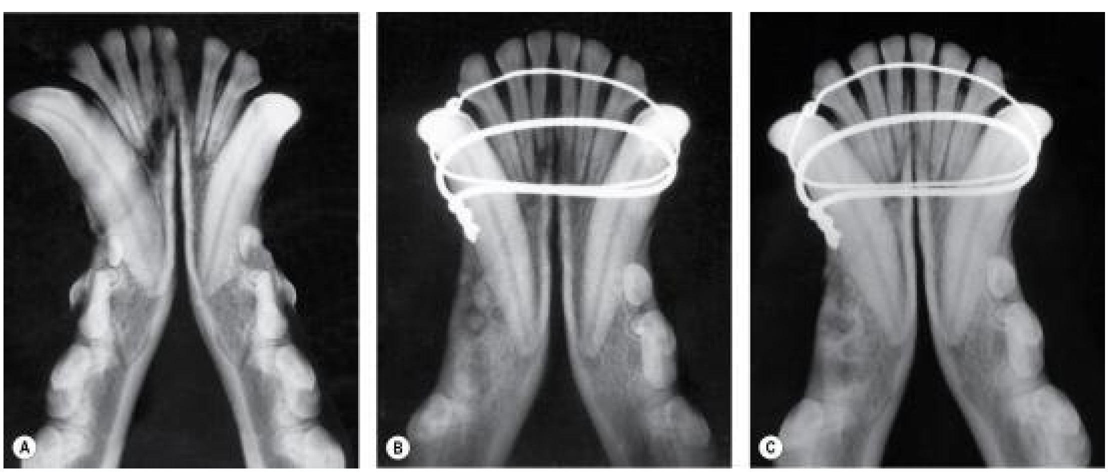
Alveolar process fractures and (sub)luxation of mandibular and maxillary incisor teeth

Cerclage wiring

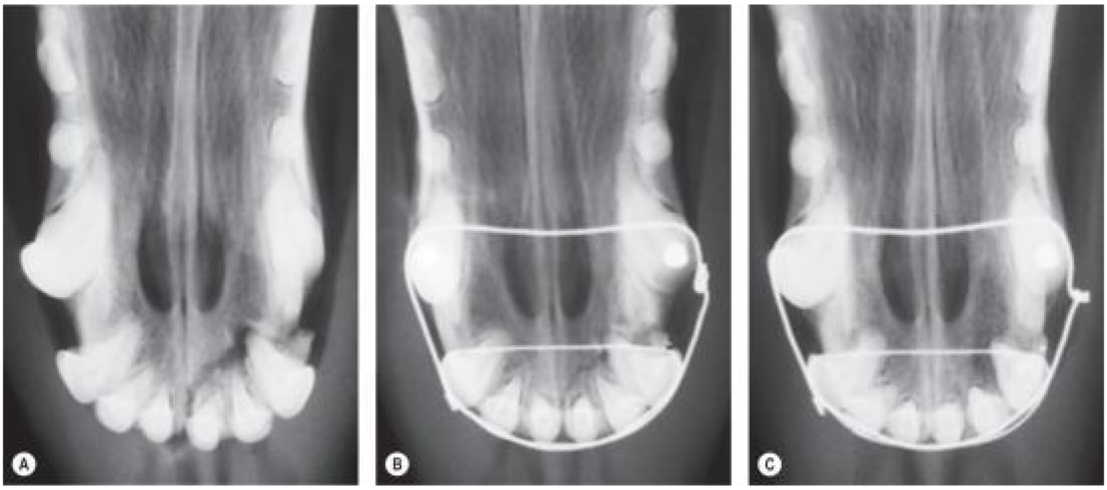
After removal of blood clots and devitalized tissue, the bone fragments and teeth are reduced and stabilized with a cerclage wire. One must be aware of the tendency for the teeth to dislocate. To prevent rostral displacement of teeth when the jaws are closed, the cerclage wire must be placed rostrally on the necks of the incisor teeth and caudally around the canine teeth (Figs 30.5–30.7). To prevent caudal displacement, the wire is placed on the caudal aspect of the neck of the reduced tooth and on the rostral surface of the necks of the normal teeth. The ends of the cerclage wire are twisted together on the labial aspect of the third incisor or canine tooth. Rostral dislocation can also be prevented by placing an acrylic or composite splint on the incisor teeth (Fig. 30.8).



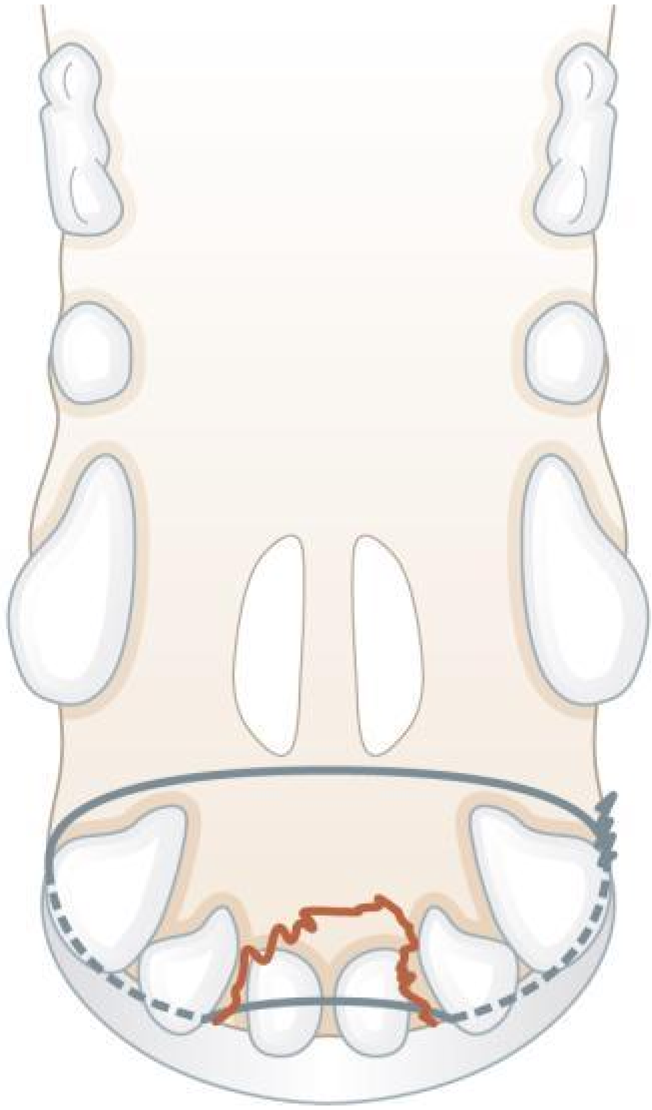
• **FIG. 30.5** Fracture of the alveoli of the first, second, and third incisor teeth with a tendency for the teeth to dislocate rostrally. Stabilization can be achieved using cerclage wire over the facial aspect of the necks of the incisor teeth and (**A**) on the labial and (**B**) lingual surface of the necks of the canine teeth.



• **FIG. 30.6** (**A**) Fracture of the alveoli of the right mandibular canine tooth and incisor teeth and the contralateral first incisor tooth in a 3-year-old dachshund. (**B**) Radiographs obtained immediately after and (**C**) 6 weeks after placement of two cerclage wires on the facial surface of the incisor teeth and distal to the canine teeth.



• **FIG. 30.7** (**A**) Fracture of the maxillary first, second, and third incisor teeth that tended to dislocate rostrally in a 2-year-old Shetland sheepdog. (**B**) Immediately after and (**C**) 2 months after osteosynthesis using two cerclage wires on the facial surface of the necks of the incisor teeth and placed distal to the incisor and canine teeth, respectively.



• **FIG. 30.8** Bilateral fracture of the alveoli of both maxillary first incisor teeth with a tendency to dislocate caudally and rostrally—these fractures can be stabilized with cerclage wire and a composite splint.

Intraoral acrylic or composite splints

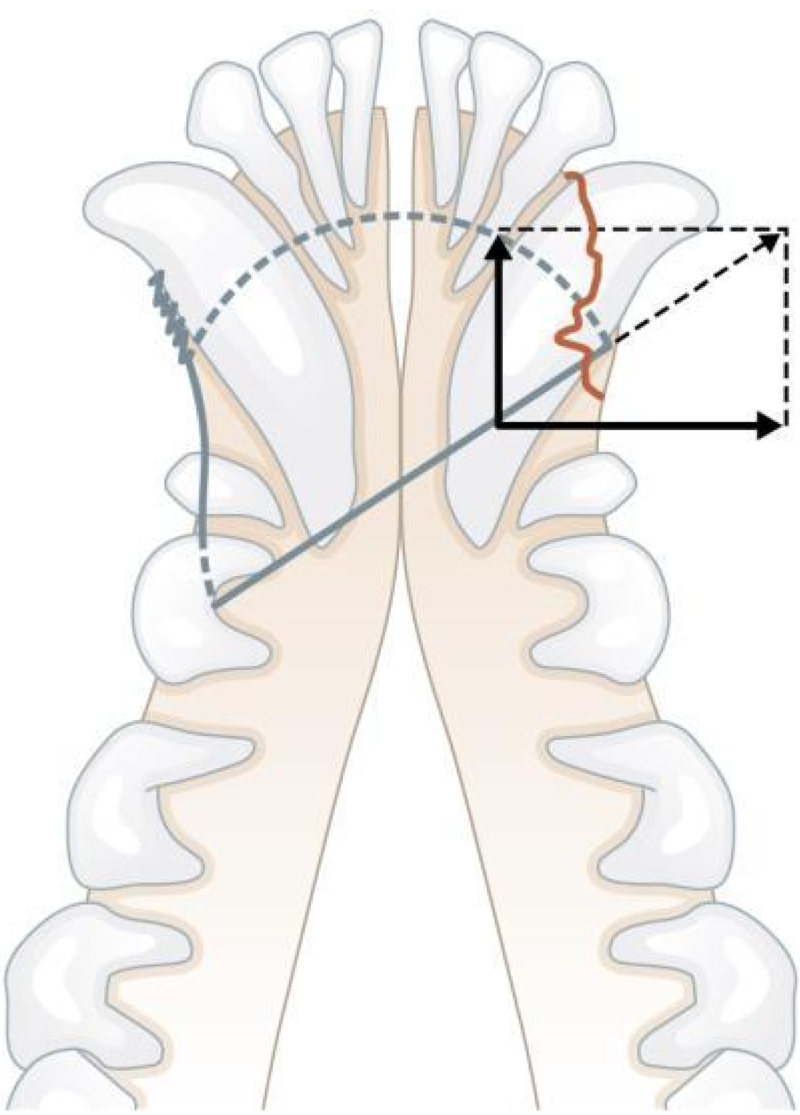
Acrylic or composite splints using the acid-etch technique (alone or in combination with interdental wiring or orthodontic brackets) are available for the repair of alveolar process fractures and (sub)luxation of teeth (see Chapters 24 and 31).[15] Intraoral splints are applied to the labial surface of the teeth in maxillary fractures and to the lingual surface of the teeth in mandibular fractures, if covering the entire crown would cause occlusal interference. An etching gel (~40% phosphoric acid gel) is applied to the enamel surface and, after cleaning and air drying, a bonding agent is applied to the etched surface so that the composite material adheres securely. Some splint materials (e.g., ProTemp Plus, 3M Inc., St. Paul, MN) are used without bonding agents. Depending on the material used, the composite splint either polymerizes on its own or is light cured. During hardening, the fracture fragments must be maintained in reduction with correct dental occlusion. Following light- or chemical-curing, the splint is then smoothed. The strength of the composite splint and the adhesive surface area can be increased by integrating interdental wiring techniques. Brackets and buttons can be used with larger teeth.

After the fracture has healed, the splint is removed by sectioning the acrylic or composite material interdentally with a bur and removing the splint in segments using bond-removing forceps. (Direct Bond Removing Orthodontic Plier, Ormco, Orange, CA). The teeth are selectively polished after removal of the splint.

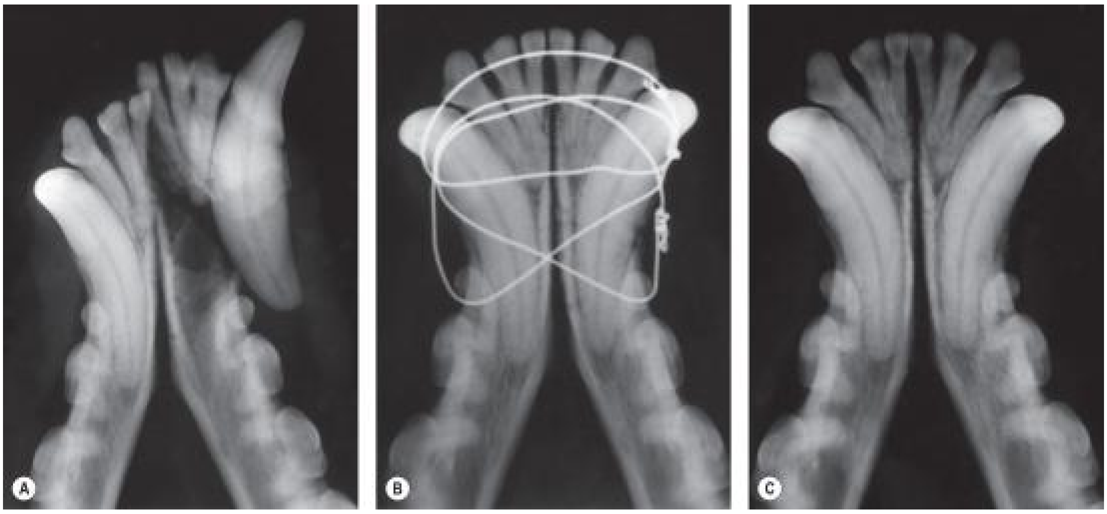
Alveolar process fractures and (sub)luxation of mandibular and maxillary canine teeth

Cerclage wiring

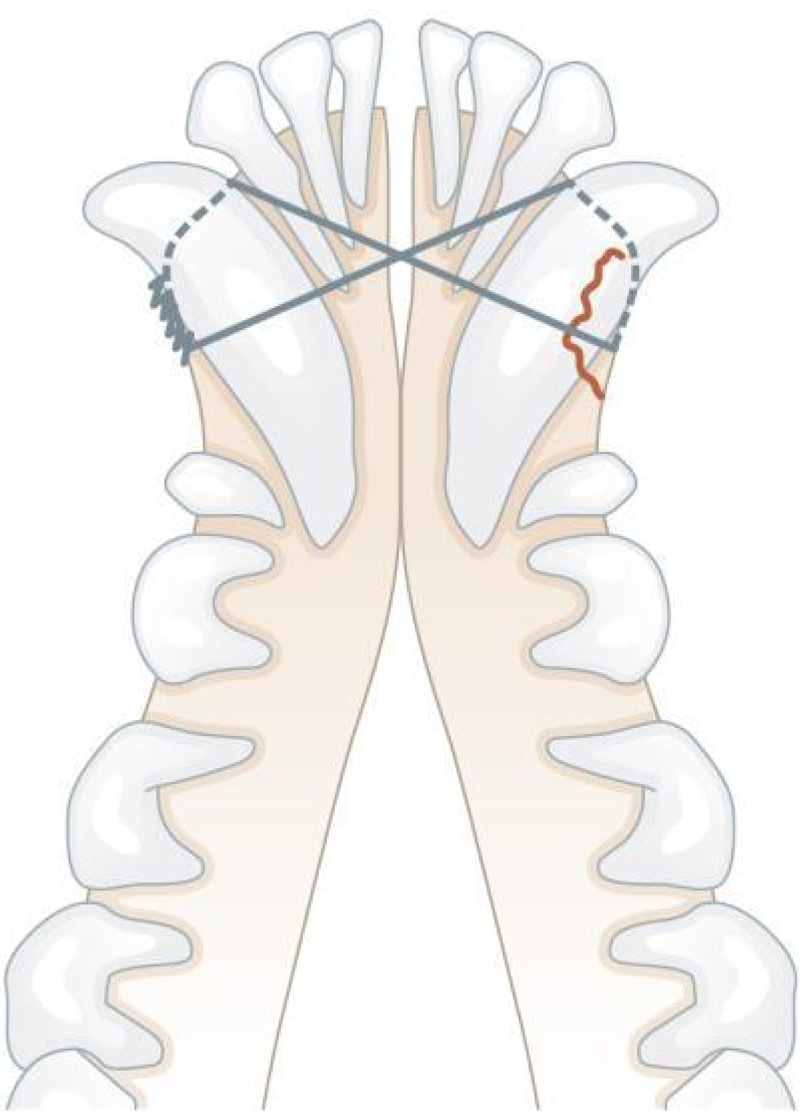
The techniques used for stabilization of subluxated or luxated canine teeth are very similar to those used for the incisor teeth. As with incisor teeth, the tendency for the canine teeth to dislocate must be considered before application of cerclage wire. When the affected tooth tends to move rostrally and labially upon jaw closure, the cerclage wire is placed rostrally around the necks of both canine teeth and around the first premolar tooth or between the roots of the contralateral second premolar tooth. The ends of the wire are twisted together at the alveolar margin of the first premolar tooth (Figs. 30.9 and 30.10). To prevent labial dislocation of a reduced canine tooth, the cerclage wire can be placed in a figure-of-eight around the necks of both canine teeth and the ends twisted together alongside the healthy canine tooth (Fig. 30.11). The different methods of cerclage wiring can be combined when needed. To prevent slippage, the cerclage wire can be secured to the teeth with composite.



• FIG. 30.9 Fracture of the alveolus of a canine tooth with a tendency to dislocate rostrally and laterally (arrows)—stabilization can be achieved with cerclage wire placed around the necks of the canine and incisor teeth and between the roots of the contralateral second premolar tooth.



• **FIG. 30.10** Fracture of the alveoli of the canine tooth and the first, second, and third incisor teeth with luxation of the teeth in a 4-year-old Münsterländer dog. (**A**) Preoperative radiograph; radiographs obtained (**B**) immediately after and (**C**) 4 months after fixation using three cerclage wires.



• **FIG. 30.11** Fracture of the alveolus of a canine tooth with a tendency to dislocate labially—the tooth can be stabilized with a figure-of-eight cerclage wire.

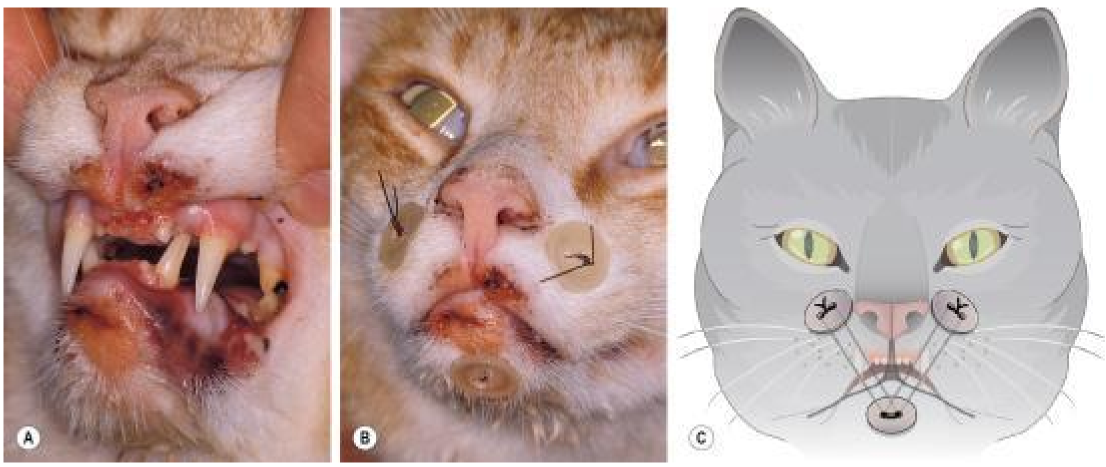
Intraoral acrylic or composite splints

As described for alveolar process fractures, subluxation and luxation of the incisor teeth, intraoral splints (with or without brackets, buttons or interdental wiring) can be used to stabilize a canine tooth when wire alone is inadequate.

Labial reverse suture through buttons

Labial reverse suture through buttons, a minimally invasive method of indirect stabilization, is indicated for caudoversion of the mandibular canine teeth, which is sometimes seen in cats that have fallen from a great height. It can also be used to treat a reduced jaw luxation that is unstable as well as concomitant fractures of the temporomandibular joint of cats.[16-18] The mandible is stabilized with the mouth closed for 8–10 days, which allows consolidation of the injury with correct dental occlusion.

The caudally dislocated canine teeth are reduced to their normal position. Nonabsorbable suture material is then used to close the lips in a mattress pattern. The suture material is placed through buttons to distribute the pressure and is tied loosely. It passes through the lips at the mucogingival junction and is placed adjacent to the canine teeth on both sides of the upper lip and through the midline of the lower lip (Fig. 30.12). Provided that the reduced canine teeth remain fairly stable, the suture can be tied loosely enough to allow the cat to lap up fluid with its tongue. However, if a tighter closure is required, a nasal, pharyngeal, or esophageal feeding tube must be placed (see Chapter 5). A pharyngeal or esophageal feeding tube is preferred in patients with concurrent maxillary fractures. Cooperative cats can be fed fluid food with a syringe via the vestibulum of the lips. The advantage of labial reverse sutures for jaw stabilization is that, unlike the composite technique, the patient loses less saliva, thereby decreasing the risk of an acid–base imbalance.



• **FIG. 30.12** (**A**) This 1-year-old cat has alveolar fractures of both canine teeth with retroversion of the incisive region of the mandible; (**B, C**) the jaws were closed ensuring dental occlusion using a labial reverse suture through buttons. An esophagostomy tube has been placed to provide nutrition while the jaws have been immobilized.

Postoperative care and assessment

Postoperative care consists of feeding wet food and administering antibiotics such as amoxicillin with clavulanic acid, clindamycin, or a cephalosporin for 3–5 days, particularly in patients with open fractures (see Chapter 3). The client is encouraged to have the patient reassessed regularly. In uncomplicated injuries, such as separation of the mandibular symphysis, reevaluation is generally at 6–8 weeks postoperatively, provided that the client can confirm normal food intake and occlusion. Alveolar process fractures that tend to be unstable should be reassessed at shorter intervals. Reexaminations should evaluate not only fracture healing but also whether the client is capable of performing the necessary oral hygiene in the patient. The latter includes routine removal of food particles without jeopardizing the stability of the fixation, particularly in animals with intraoral wires and splints. In uncooperative patients, sedation or a brief anesthesia is recommended to carry out routine oral hygiene procedures. The authors advocate the use of an oral irrigator (Waterpik® Dental Systems, Water Pik Technologies, Newport Beach, CA), set at low water pressure, for cleaning the oral cavity.

Fractures of the alveolar process are often associated with periodontal trauma and devitalization of teeth. Appropriate dental treatment is indicated if dental injuries and complications have occurred (see Chapter 24).[19-21]

Complications

Osteomyelitis is the most serious complication but rarely occurs after injuries of the incisive region of the mandible and maxilla (see Chapter 35). Osseous inflammation that is unresponsive to antibiotics is usually seen in fractures with inadequate fixation. It is important to remember that treating biomechanical deficits with antibiotics is futile. Thus stabilization of jaw fractures is an absolute priority (see Chapter 27). For infections of the jaw bones, the choice of an appropriate antibiotic is discussed in Chapter 3. When the injury in the incisive region fails to heal, resection is possible without major functional impairment. However, incisivectomy and mandibulectomy of the incisive region due to injury are uncommon treatments.[22]

Prognosis

There are numerous reports in the veterinary literature describing surgical methods and their indications, but not many that analyze the results of repair techniques in a representative group of patients. At the Ludwig Maximilians University in Munich, the authors carried out clinical and radiographic evaluations of 56 dogs with fractures of the incisive region and/or the alveolar process that had been stabilized with wire; 52 (93%) had normal jaw function after healing.[1] Of 45 dogs and 49 cats with injuries of the incisive region, 86% and 90%, respectively, had a good outcome.[2,4] The same was true for 46 cats in another study, in which mandibular symphyseal separations and parasymphyseal fractures were treated using an interdental and interfragmentary stabilization technique.[12] We concluded therefore that the majority of rostral jaw fractures can be repaired successfully with a minimum of technical expenditure. For fractures that cannot be adequately stabilized with wire or additional intraoral splint techniques alone, alternative techniques such as maxillary transfixation pinning are available to restore normal jaw function.[23] Partial jaw excision is a rarely used last resort. In the authors' experience, temporary lip closure using a labial reverse suture through buttons has a good prognosis in most feline cases requiring stability to the mandible by the interdigitation of the canine teeth. With this relatively simple procedure, unimpaired jaw function was achieved in 67 of 72 (93%) cats that underwent clinical and radiographic reevaluation postoperatively.[17]

In human medicine, tooth luxation has a very guarded prognosis because of frequent tooth root resorption and crown fractures due to dental ankylosis. There is very little information about this subject in the veterinary literature (see Chapter 24). The authors have had only a few cases that were available for reevaluation over several months. However, it appears that the prognosis for tooth luxation in animals may be better than that suggested in the human literature.

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CHAPTER 31

Maxillofacial fracture repair using noninvasive techniques

Christopher J. Snyder

Definitions

Maxillomandibular fixation (MMF): a fracture repair technique utilizing the patient's normal dental occlusion to approximate fracture fragments, which provides fracture reduction and immobilization to facilitate bone healing.

Interdental wiring: use of orthopedic wire and one of a variety of wiring patterns that involve 22- or 24-gauge wire anchored around and between tooth crowns, using the teeth as anchor points to establish fracture fragment alignment and fixation.

Intraoral composite or acrylic splint: a custom-made, semirigid fixation device that encompasses multiple adjacent teeth and maintains them in a fixed position, thus aligning and stabilizing fracture fragments. Bis-acrylic composites, either chemical- or light-cured, are typically used to fabricate intraoral splints; although methacrylates may be used, their exothermic polymerization (curing) process can cause thermal damage to teeth.[1,2]

Chemical- (self-) cure composite: composite systems that undergo polymerization by mixing an initiator and amine activator, resulting in free radical release and chemical bonding between monomers to form a polymer.

Light-cure composite: composite systems, which undergo polymerization by the induction of free radical formation through a specific wavelength of blue light reacting with a photosensitizing agent and an amine in the composite material.

Macromechanical retention: the process by which composites and/or wire are maintained in place due to the anatomical features of intraoral structures rather than a microscopic interlock of the materials to one another and/or the teeth.

Micromechanical retention: the process by which composite or acrylic products are bonded to the tooth surface, which requires conditioning of the tooth surface to demineralize enamel and/or dentin. This allows monomers of resin bonding agents to enter the resulting microporosities, and polymerization of the resin creates a microscopic interlock between the resin and the tooth surface.

Preoperative concerns

It is easy to develop tunnel vision and focus on the obvious injuries when a patient is presented with maxillofacial trauma. It is a rare occasion in which maxillofacial fractures require immediate repair. Patients should be thoroughly screened for comorbidities, which may increase general anesthesia risk. A thorough general physical examination, neurologic assessment, basic metabolic assessment (by means of complete blood count, chemistry profile and urinalysis), and thoracic radiographs should be performed prior to anesthesia administration. Once the patient has been stabilized, the primary management goal is to return the oral cavity to function and restore normal occlusion.

Most noninvasive fracture repair methods incorporate dental structures into the fixation device. Many patients concomitantly suffer dentoalveolar trauma at the time of maxillofacial trauma[3] or have preexisting periodontal disease that compromises the use of these teeth as structurally stable anchors for stabilization.[4] Thorough assessment of teeth involved with dentoalveolar trauma is important, as treatment of dental injuries may be required to incorporate these teeth into the fixation device. Multirooted teeth with compromised endodontal blood supply may undergo hemisection and endodontic therapy to preserve as much dental structure as possible for incorporation into composite splints or interdental wiring.[4] Endodontally compromised teeth can be temporarily treated with sealants or temporary endodontic therapy, planning for extraction or definitive endodontic therapy following bone healing. Mandibular fractures with teeth requiring treatment that are associated with the fracture line require a longer time to achieve bone healing.[5] Since noninvasive repair techniques involve the attachment of some material to the dental crown, all teeth should be ultrasonically scaled and polished with nonfluoride pumice prior to placement of wires or splints. Polishing with pastes or pumice containing fluoride risks interference between the dental conditioning agents and adherence of composites or acrylics. Extraction of mobile teeth should be considered prior to determination of whether an intraoral repair technique is appropriate, because anchorage for such devices requires two to three healthy teeth on either side of the fracture line. Although a technique has been described for using intraoral composite splints in edentulous patients,[6] open reduction and internal fixation are usually a better choice when teeth are absent. Always follow the manufacturer's instructions before applying dental composites or acrylics to determine if a bonding agent is recommended following etching.

A great deal of anecdotal evidence exists describing successful bone healing in craniomaxillofacial trauma patients using noninvasive repair techniques. The non-weight-bearing nature of the mandible and maxilla, as well as the propensity for injuries to occur in animals less than 12 months of age,[3,7] likely contributes to this success. Appropriate selection for the use of these techniques includes the presence of periodontally healthy teeth on either side of the fracture line, absence of a bony defect, and minimal fracture comminution.

Imaging of traumatized patients is essential for both full appreciation of the extent of damaged structures and to guide veterinarians in long-term follow-up or monitoring recommendations. Computed tomography (CT) scanning has been shown to be more sensitive for identifying maxillofacial injuries compared to skull radiographs.[8] While the identification of additional fractures does not always necessitate intervention and treatment, damage to additional structures is important for long-term monitoring of endodontal health or possible impact on growth and development in juvenile patients.

Surgical anatomy

Noninvasive fracture repair management utilizes methods of closed fracture stabilization and relies on restoration of normal occlusion and radiographic confirmation of fracture fragment apposition for assessment. Biomechanically, the alveolar surface is the tension surface of the mandible,[9] and noninvasive repair methods such as interdental wiring and splinting capitalize on fixation of the tension surface, allowing natural compression of the ventral surface. Anatomical considerations for fracture repair in this fashion include the recognition of the safe locations for placement of noninvasive devices. Dogs and cats have an anisognathous occlusion, with the lower jaw narrower than the upper jaw. Most premolar teeth are not in occlusion, allowing placement of composite on both buccal and lingual surfaces of the crowns, but the relationship between the maxillary and mandibular incisor, canine, and carnassial teeth necessitates careful placement of materials to prevent interference with occlusion. Interference with occlusion risks functional challenges with prehension, could lead to splint failure, and has the potential to cause periodontal disease or traumatic intrusion of occluding teeth. To avoid this, intraoral splints should be placed predominantly on the lingual aspects of the mandibular molar teeth or the buccal surfaces of the caudal maxillary teeth (Fig. 31.1).

• **FIG. 31.1** Intraoral composite splint fabricated for the management of a rostral unilateral maxillary fracture. The splint material is applied to the buccal surface of the maxillary teeth to prevent interference with occlusion.

Consideration of dog and cat dental anatomy is necessary when considering noninvasive repair methods for fracture fixation. Interdental wiring techniques successfully used in humans have been adapted for veterinary patients, despite human techniques relying on dental crowns being tapered at the neck. The pyramidal shape and the absence of a tapered neck of the teeth in dogs and cats create challenges when placing interdental wiring, because tightening the wires results in shifting of the wires toward the cusp tip versus toward the tapered neck as noted in humans. Careful use of an appropriately sized hypodermic needle through the interproximal gingiva can aid in creating anchorage. In some cases, placing a ridge of composite immediately coronal to the interdental wire will help prevent wires from slipping coronally on the tooth. It should be emphasized that creating a groove or notch in the tooth crown to retain interdental wire is never indicated, as it risks causing pulpitis and weakens the tooth, potentially leading to crown fracture and, possibly, failure of the fixation appliance. Since gingival inflammation associated with the use of interdental wires may result in alveolar bone resorption, evaluation for healing and fixation removal should take place at 6 to 8 weeks.[10]

Noninvasive fracture repair methods reduce the risk of iatrogenic trauma to tooth roots and neurovascular structures of the mandible and maxilla commonly associated with open reduction and internal fixation. Especially with caudal injuries, where a majority of mandibular growth occurs after 3 months of age in dogs,[11] noninvasive repair techniques should be considered, when appropriate, to minimize any negative effects on remaining mandibular growth in skeletally immature animals.

Restoration of the patient's pretrauma occlusion is the primary goal of any maxillofacial fracture fixation technique. Endotracheal tubes are traditionally placed via the oral cavity, but this interferes with the ability to close the mouth completely and therefore creates difficulty when assessing occlusion during the procedure. To evaluate occlusion throughout the process of reduction and stabilization, it is necessary to use alternative methods of endotracheal intubation (see Chapter 61). Both pharyngotomy[12] and transmylohyoid[13] intubation techniques permit complete evaluation of dental occlusion, minimize interference by the tube during fracture reduction and stabilization, and eliminate the need for intermittent extubation during the procedure to check occlusion, saving time.

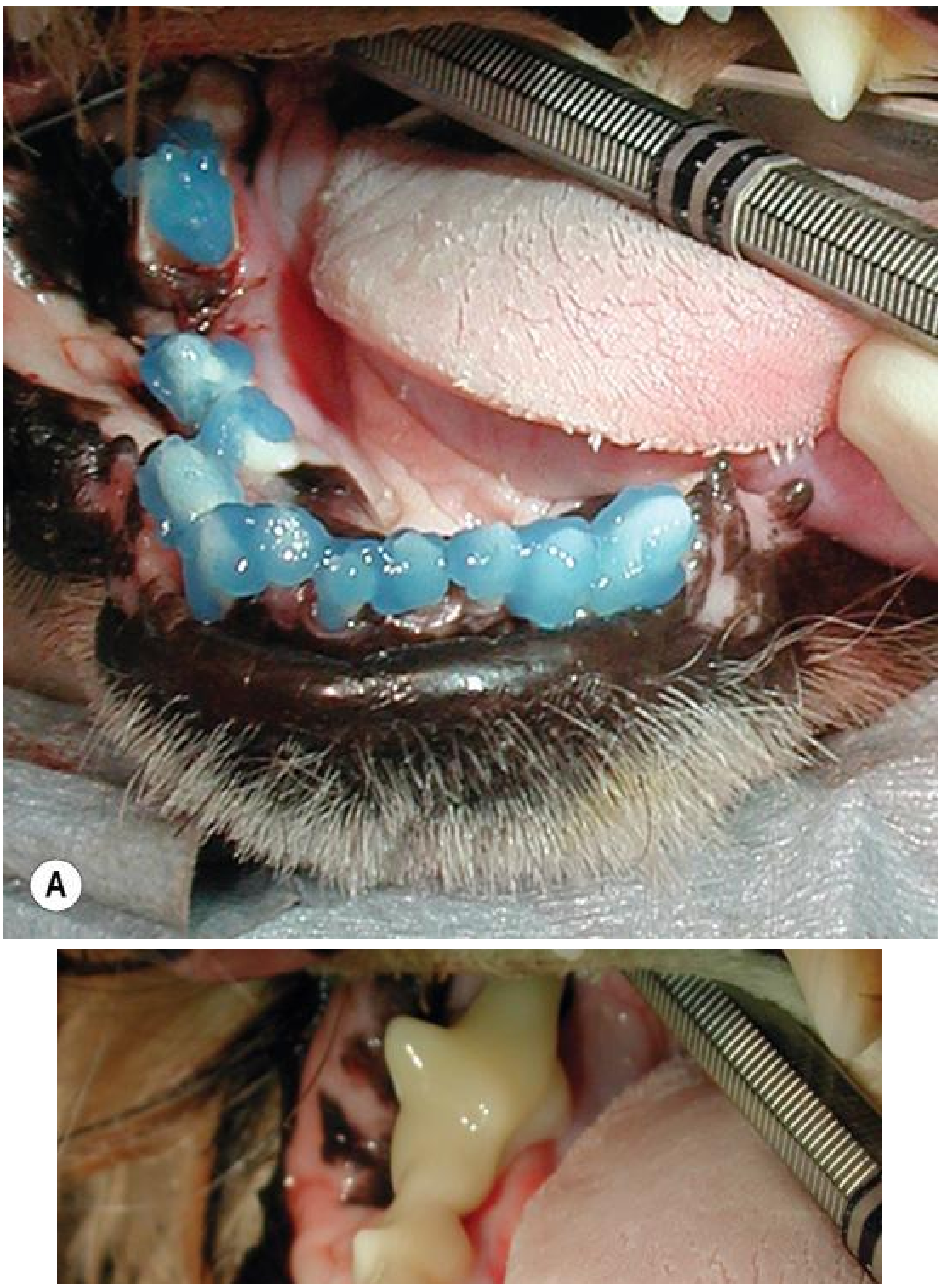
Special instrumentation and materials

Noninvasive fracture management devices range from a simple tape or nylon muzzle to interdental wire and composite splints. The materials required for noninvasive fracture management are generally inexpensive and readily available. They include nylon muzzles and/or 1″ and 2″ bandage tape, orthopedic wire, wire cutters, wire twisting forceps, acid-etchant, dental composite material and an appropriate delivery apparatus (self-cure composites) or curing light (light-cure composites), and an acrylic bur to smooth the surface of the splint after curing.

Commercially available nylon muzzles or custom-designed tape muzzles that limit mouth opening while maintaining the patient's normal occlusion are the simplest and least invasive repair methods but demonstrate the greatest risk for healing complications.[7]

Interdental wiring is commonly performed using 22- or 24-gauge orthopedic wire. Sufficient gauge should be chosen to generate fracture stability, maintain occlusion, and withstand stresses with use. Concern about wire fatigue and breakage is typically mitigated by combining interdental wiring techniques with custom splint fabrication using dental acrylic or composite. Orthopedic wire may also be used for additional points of intraoral anchorage via placement of mandibular cerclage wires to help affix the splint in place in edentulous patients[6] or to provide an additional point of anchorage in patients missing multiple teeth.

Composite splints offer the advantage of custom fabrication of a fixation device that accommodates the patient's anatomical features. Prior to placement of the composite, the tooth surface must be prepared. Sonic or ultrasonic removal of calculus and plaque should be followed by polishing with nonfluoride pumice. Conditioning the tooth surface with an acid-etchant (37% phosphoric acid gel is commonly used) demineralizes the enamel surface, leaving a rough surface of exposed collagen tags. This roughened surface allows the composite to interdigitate with the exposed collagen, creating micromechanical retention (Fig. 31.2).[14] Contamination of the etched tooth surface with blood, water, or saliva between the etchant being rinsed away and composite being applied requires reetching. Teeth demonstrating evidence of dentoalveolar trauma may be sensitive during this step. Use of a dental adhesive applied to the etched surface, but before placement of the composite, has been shown to bond composites more tightly to the tooth surface, which will help prevent detachment of the composite splint but will increase the time necessary for removal and also increases risk for iatrogenic tooth damage during removal.[15] Follow the composite manufacturer's instructions as to whether the placement of bonding agents should be performed before placing composite, as bonding is not recommended for certain materials. In general, the combination of interdental wiring and composite splinting has been recognized to be stronger than either method of fixation alone.[16,17]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• FIG. 31.2 **(A)** Enamel conditioning with 37% phosphoric acid to improve micromechanical retention of composite splint material for management of mandibular fracture. **(B)** Placement of chemical-cure composite material on the lingual surface of mandibular teeth facilitates unencumbered occlusion.

Polymethylmethacrylate compounds were historically used to create intraoral splints, but their exothermic curing process may cause thermal damage to the pulp,[1] and the noxious odor produced during curing is undesirable. Composite materials most commonly used in veterinary dental practice today include light-cure or chemical-cure (self- or cold-curing) bis-acrylic composite materials. Light-cure materials rely on photosensitization of the material and release of free radicals that facilitates addition polymerization. Chemical-cure composite materials polymerize through the mixing of two pastes, one of which contains an activator and the other contains an initiator. The two pastes are typically delivered from a double-barreled cartridge with an automixing tip; mixing results in release of free radicals and initiates polymerization.[18] Light-cure composites are typically more expensive than chemical-cure composites and require a special concentrated 400–500-nm light source to activate the photosensitive initiator.[1] Concentrated frequency of light facilitates a stepwise curing of material, which is limited by surface area and depth of cure, necessitating placement and curing of multiple sections of composite for intraoral splint formation.[18] This is not only time consuming but also allows microscopic voids between sections of composite, which could result in splint failure. Therefore chemical- cure composites offer advantages over light-cure composites for fabrication of intraoral splints. MMF using composite or creation of custom fabricated orthodontic buttons for use of elastics for fracture management are situations where smaller amounts of composite are necessary, and the speed of curing makes light-cure composites preferable to chemical-cure composites.

Therapeutic decision-making and techniques

The advantages of noninvasive repair techniques include not disrupting the fracture hematoma, no surgical trauma to the periosteum in the area of the fracture site, and a decreased risk for iatrogenic exposure of the fracture site to bacterial contamination. Intraoral techniques also eliminate the risk of iatrogenic damage to dental structures or neurovascular structures, commonly associated with creating pilot holes for orthopedic implants. The disadvantages of noninvasive techniques include inadequate reduction and apposition, leading to an absence of primary bone healing, and semirigid fixation risking micromotion, either of which can lead to delayed healing, fibrous union, malunion, or nonunion.[7] Noninvasive repair techniques should not be the first choice for highly comminuted or structurally unstable fractures; open reduction and internal fixation is a better option in these cases (see Chapter 33). If closed reduction of fracture fragments is imperfect, even by just 1–2 mm, the resulting malocclusion can be significant, negatively impacting the patient's ability to function normally following removal of the appliance. Additionally, failure to restore a patient into normal occlusion risks temporomandibular joint (TMJ) degenerative changes due to altered relationships between the articular surfaces, as demonstrated by a study in dogs undergoing mandibulectomy without reconstruction.[19]

When managing juvenile patients with deciduous or mixed dentition, open reduction should be avoided, because in addition to the obvious risk of damage to developing tooth buds, open reduction and internal fixation will disrupt the periosteum, potentially leading to delays in intramembranous ossification and interfering with skeletal growth. These patients are best treated with muzzle coaptation, or, in some cases, intraoral splints with mandibular cerclage wires.

The role that teeth in the fracture line play in stabilizing the fracture repair is a controversial topic. Fractures coursing along tooth roots risk compromising the endodontal health of the tooth, which may result in infection and compromised bone healing. Developing, unerupted teeth in the fracture plane may be left during fracture stabilization and healing but should be monitored radiographically for failure to erupt (which can lead to dentigerous cyst formation), pulpal necrosis, or developmental abnormalities tooth maturation and formation.[20] The decision guiding whether extraction is necessary prior to fracture treatment may impact fixation selection. For example, in a study simulating the loss of the first molar tooth in mandibular fractures, the use of interdental wiring and composite splinting was significantly stronger when the tooth was still present.[17]

Muzzle coaptation

Muzzles may be useful at multiple time points in maxillofacial trauma patient management. In the preoperative period, muzzles may provide support and pain relief while awaiting diagnostics and treatment. Postoperatively, they provide additional support or restricted range of motion if necessary. Muzzles should be changed daily after meals, as this provides an opportunity to clean the face to prevent development of moist dermatitis secondary to accumulation of food debris under the muzzle, which is common when a gruel or other very soft diet is offered.

Muzzle coaptation can be used as a sole form of treatment in the management of fractures in juvenile patients, as it capitalizes on the use of dental occlusion to approximate fracture fragments, and in growing patients, new bone formation occurs rapidly. It is also an option for management of adult dogs when clients have severely limited financial resources, but it should be noted that of all conservative treatment techniques, muzzle coaptation is associated with the greatest risk for healing complications, including malunion and nonunion.[7] Muzzles can be purchased in a variety of sizes, or created with 1″ or 2″ medical tape to provide custom sizing. Muzzle size should be such that the patient is able to open its mouth just enough to exteriorize the tongue to lap gruel and water; limited panting is also desirable. This is often the point where the crowns of the mandibular and maxillary canine teeth overlap by one-third to one-half of the crown height. By design, muzzle coaptation relies on the interdigitation of the mandibular canine teeth into the interdental space between the maxillary canine and third incisor teeth to approximate fracture fragments into a position that will allow healing and restoration of a functional, comfortable occlusion. Custom fabrication of a muzzle with 1″ or 2″ medical tape is accomplished by attaching two pieces of tape together such that there is no exposed adhesive and encircling the muzzle, allowing just enough space for the tongue to protrude. Sidepieces are created in a similar fashion, extending from the muzzle caudally across the face and tied or taped behind the ears. Brachycephalic breeds may benefit from a third piece of tape extending dorsally over the muzzle, between the eyes and attached to the sidepieces behind the head. Commercially available nylon muzzles are a convenient alternative and should be similarly selected for sizes that allow lapping gruel, drinking water, and limited panting. Restricting range of motion for 3 to 5 weeks is recommended to prevent the development of ankylosis, pseudoankylosis, or a permanently restricted range of motion. It is frequently recommended to graduate patients out of a smaller muzzle into one that affords a larger range of motion, without completely allowing the patient to fully open its mouth, for an additional 3–5 weeks. All patients wearing muzzles should be monitored closely to ensure weight is maintained and for appropriate urine and fecal output as a sign of adequate food and water intake. Providing the client with two of the same-sized muzzles permits washing the muzzle, cleaning the patient's face, and drying the fur after meals to prevent development of moist dermatitis.

Interdental wiring techniques

For interdental wiring fixation 22–24-gauge orthopedic wire (depending on patient size) is anchored around the teeth to provide fixation along the biomechanically advantageous tension surface of the mandible. Use of interdental wiring is less invasive and less expensive than open reduction and rigid internal fixation using plates but typically requires two anesthetic episodes for placement and removal. Additional anesthetic episodes may be necessary if the fixation device becomes dislodged or is broken. Although semirigid fixation is not ideal, interdental wiring avoids the need to disrupt the fracture hematoma and does not disrupt the periosteum or otherwise induce surgical trauma associated with more invasive repair techniques. Interdental wiring techniques that utilize a single length of wire typically refer to the wire ends as the "working lead" and "static lead." The static lead refers to the end of the stationary wire that is incorporated into fixation with the working lead, which is anchored around the teeth and to the static lead. Wiring techniques utilizing two separate wires refer to the "primary wire" and "secondary wire."[21]

Whenever possible, any wire twists and wire ends should be placed against the tooth crown on the surface of the tooth least likely to impact occlusion (the buccal surface of maxillary teeth and lingual surface of mandibular teeth.) Wires should be placed at the free gingival margin, or through the gingiva, to prevent slippage in the coronal direction. A larger-gauge needle is used to penetrate the interproximal gingiva to allow wire placement through the gingiva. This will help prevent the device from being displaced coronally. Alternatively, small aliquots of composite restorative material can be used to anchor the wire to the crown of each tooth.

Ivy loop wiring technique

The simplest interdental wiring technique stabilizes the teeth on either side of a fracture line. This wiring technique utilizes a length of wire looped around the distal aspect of the caudal tooth, with the static lead laying against the crown at the free gingival margin of both teeth. The working lead courses along the opposing surface of the crown and at the interdental space, transitions to the side of the static lead, and courses above or below the static lead, looping around that wire and reversing direction back through the interdental space. The working lead then joins the static lead and the two are twisted together along the mesial surface of the tooth rostral to the fracture. The loop of wire at the interdental space is twisted alternately with the wire ends of the rostral tooth to generate compression and stabilization. Once the wires are tightened, the wire ends should be bent along the tooth surface (Fig. 31.3). The limited number of anchorage points for this wiring technique results in a device with limited stability. For fractures caudal to the mandibular first molar tooth, the smaller second and third mandibular molar teeth are not sufficient for anchorage.

• **FIG. 31.3** Photographs of an osteological specimen of the right mandible showing application of the Ivy loop interdental wiring technique with twists placed on the lingual surfaces of the teeth. **(A)** A length of wire is wrapped around the distal aspect of the distal tooth crown with lengths of wire on both the buccal and lingual surfaces. The buccal surface wire (working lead) is fed through the interdental space below the lingual wire (static lead) and is bent around and fed back through the interdental space above the static lead. The ends are twisted upon themselves against the mesial aspect of the mesial tooth crown. **(B)** The twisted ends and **(C)** the working lead loop in the interdental space are alternately twisted to generate compression and bent to the coronal direction.

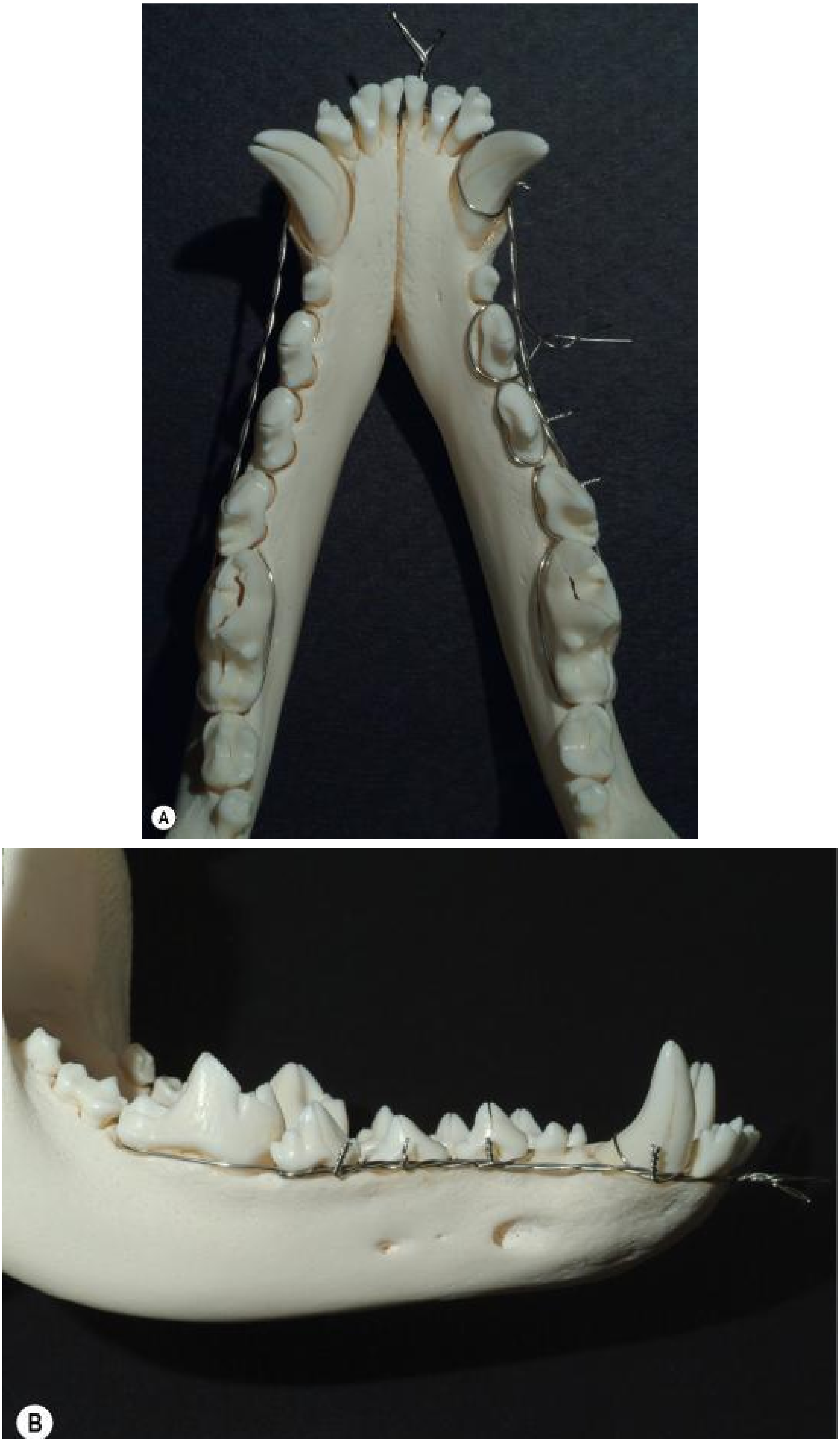
Stout loop wiring technique

The Stout multiple loop wiring technique (Fig. 31.4) utilizes a similar pattern to the Ivy loop technique but incorporates more than one tooth on either side of the fracture line. Utilizing a longer segment of wire to stabilize multiple teeth affords more points of anchorage on either side of the fracture, which is preferred.

• **FIG. 31.4** Stout loop wiring technique is an extension of the Ivy loop wiring technique, incorporating multiple teeth on either side of the fracture, and is demonstrated on an osteological specimen. Following twisting of the wire, all wire twists are bent to lie against the crowns of the teeth and orientated as such to avoid contact with opposing occlusal dentition.

Risdon wiring technique

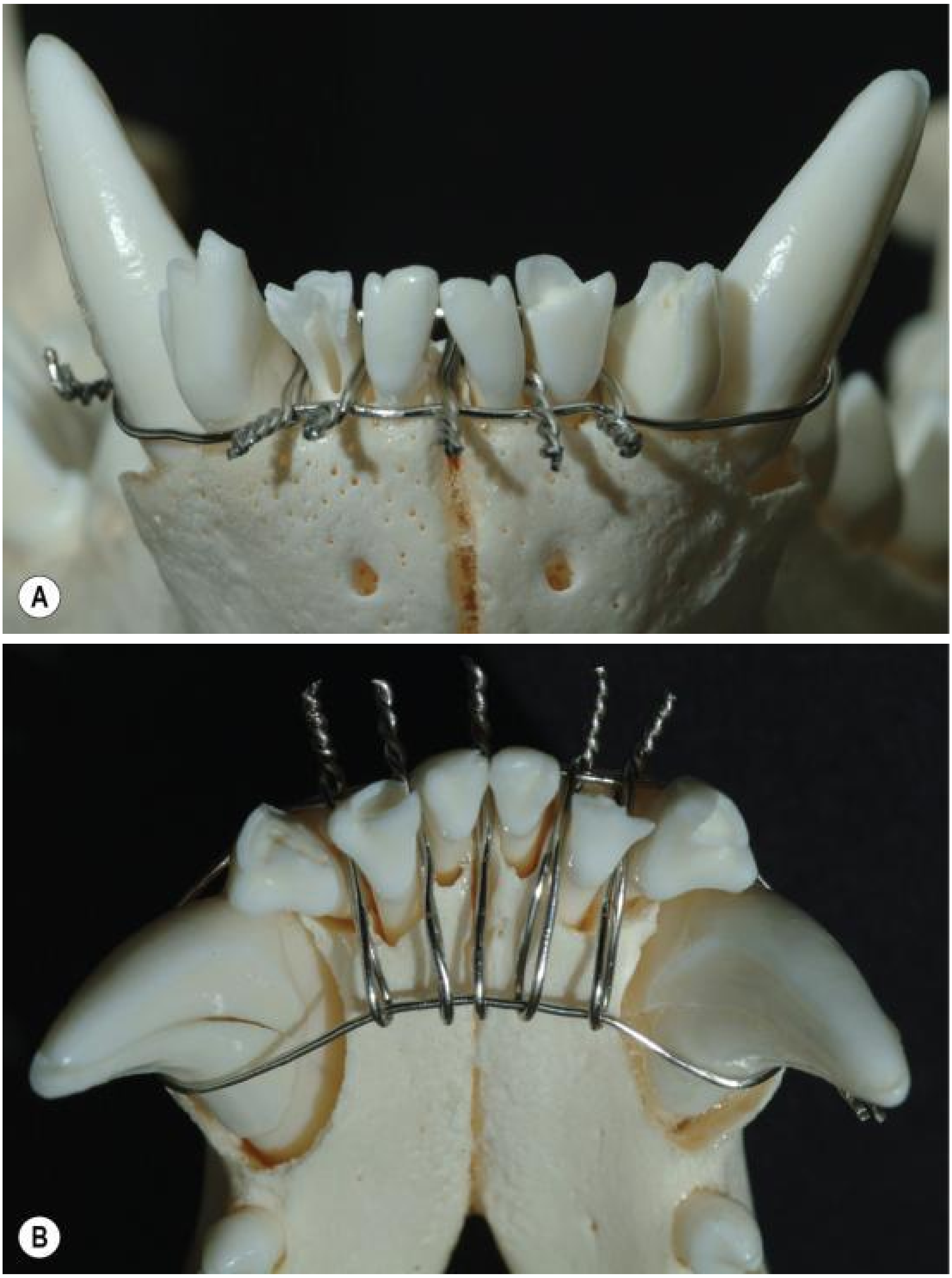
The Risdon wiring technique (Fig. 31.5) incorporates the use of bilateral primary wires, each of which is anchored around a large caudal tooth, typically the mandibular first molar or maxillary fourth premolar tooth. Each primary wire is twisted on itself, and the two primary wires are joined together rostrally. Short segments of wire (secondary wires) are used to wrap around individual teeth and twisted around the primary wire, anchoring the primary wire to multiple points along the dental arch. Application of this wire pattern is especially useful for treatment of rostral mandibular fractures.


A
B

• **FIG. 31.5** Photographs of an osteological specimen showing application of the Risdon wiring technique. **(A)** A wire is placed bilaterally around the caudal anchoring teeth (mandibular first molar teeth), twisted on itself, followed by each primary wire being twisted together in the region of the first incisor teeth. Individual secondary wires are placed interdentally around multiple teeth bilaterally and twisted to include the primary wire. **(B)** Wires are cut and turned to face the crowns of the teeth following tightening.

Essig wiring technique

The Essig wiring technique (Fig. 31.6) uses one long primary wire, anchored distally around a large anchor tooth, with each end following the buccal or lingual surface of multiple teeth on either side of the fracture, and the wire ends are twisted together to encompass a full stretch of teeth in a segment. Multiple short secondary wires are used around smaller teeth to anchor the lingual and buccal stretches of wire into place. This technique may be advantageous for reduction and stabilization of luxated canine teeth with alveolar bone fracture.

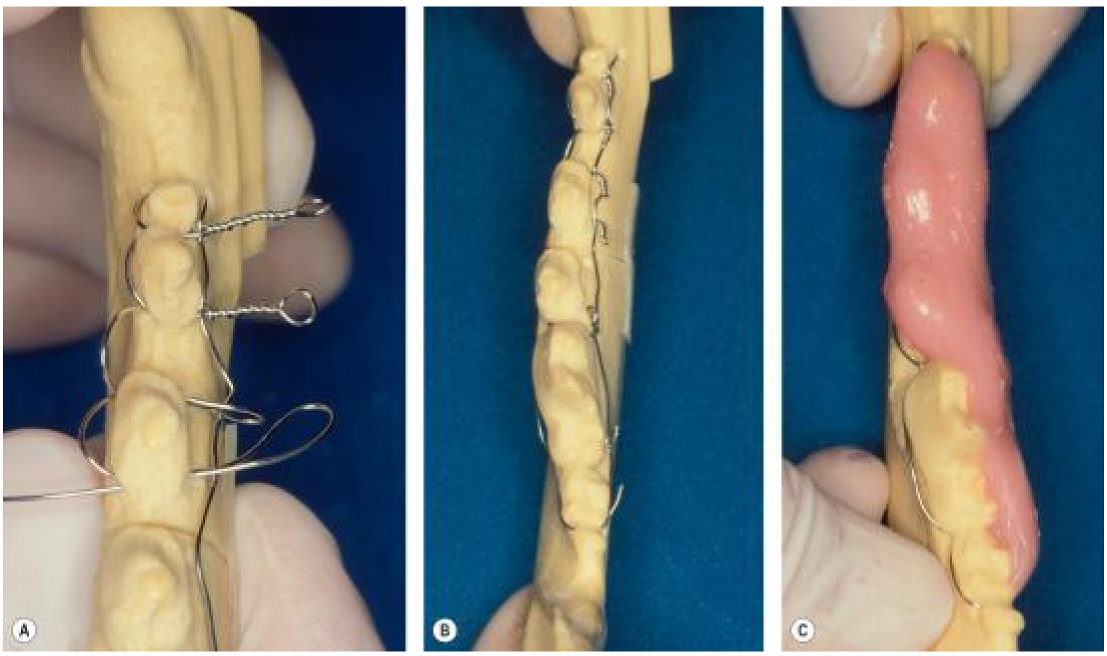


• **FIG. 31.6** Photographs of an osteological specimen showing application of the Essig interdental wiring technique. **(A)** The primary wire is placed around the canine teeth and along the rostral aspect of the incisor teeth. **(B)** Individual secondary wires are placed through the interproximal spaces and around the caudal and rostral aspects of the primary wire. The interproximal wires are tightened to place tension on the primary wire.

Application of intraoral splints

Intraoral splints are considered a “load-sharing” form of fixation and are best applied in cases of transverse or favorable fracture orientation in the mid body or rostral mandible. A commonly employed form of reduction and stabilization is the use of intraoral composite or acrylic splints.

Bis-acryl composite temporary crown and bridge material (ProTemp Plus, 3M Inc., St. Paul, MN) is commonly used to make these splints, which can be fabricated in situ and trimmed and smoothed to accommodate the patient's occlusion. These readily available dental materials can be fabricated to leverage a mixture of micromechanical retention (treatment of the tooth surface to facilitate bonding through acid-etching) and macromechanical retention (resulting from the composite hardening around and between tooth structure and interdental wire) (Fig. 31.7). Ideally, at least two teeth on either side of the fracture line should be available to facilitate stabilization by the interdental wiring and composite splint. The combination of interdental wiring with composite splinting has been shown to be stiffer and stronger than either composite or interdental wiring alone.[16,17]



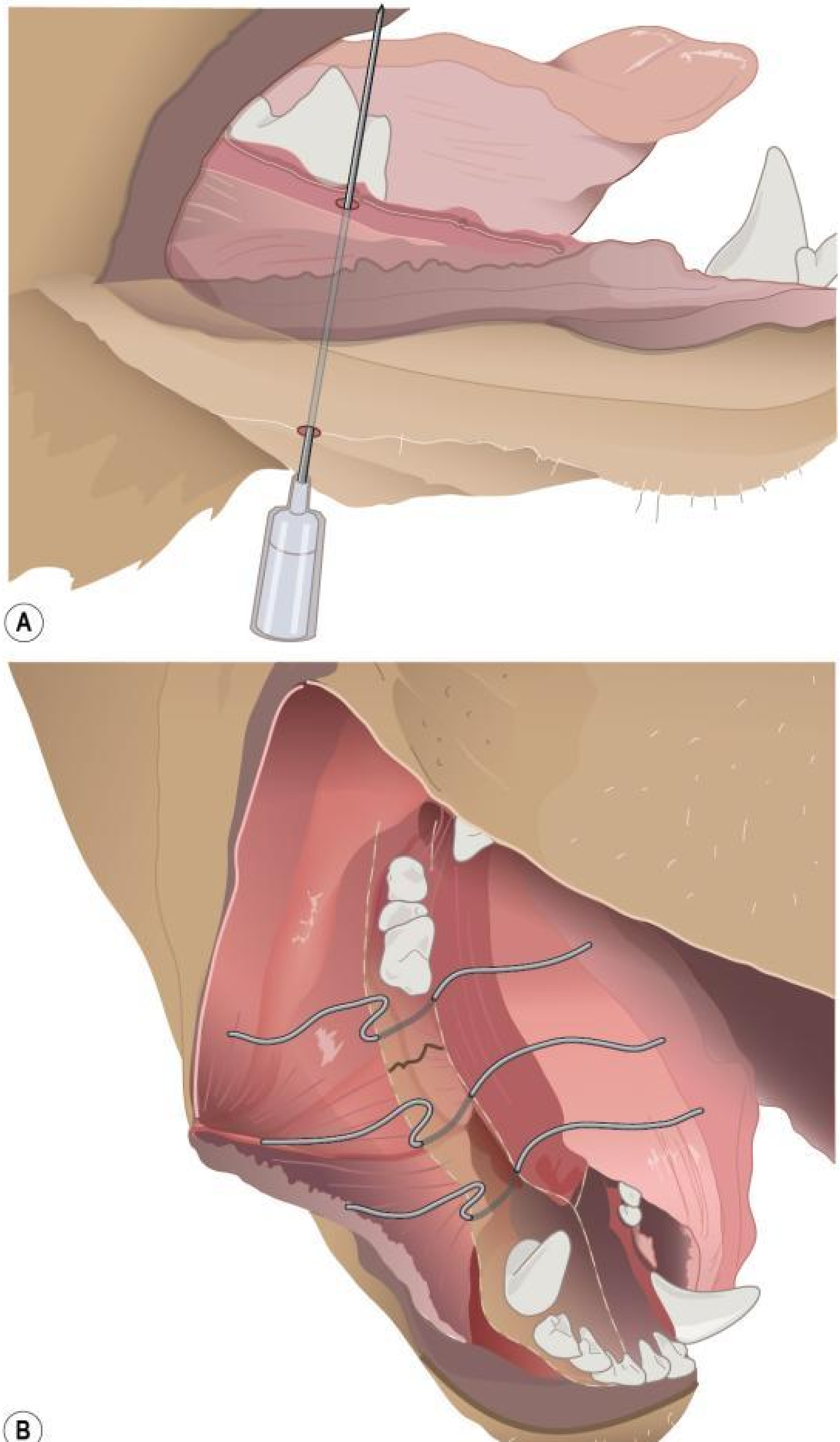
• FIG. 31.7 Photographs showing application of interdental wire and acrylic in a canine mandible model. **(A)** In this example, the wire is placed using a modified Stout loop pattern. **(B)** Wire twists are located on the lingual aspects of the teeth. **(C)** The acrylic is applied on both sides of the premolar teeth and only on the lingual aspects of the mandibular molar teeth in order to avoid occlusal interference.

In a study by Hoffer et al.,[15] it was noted that chemical-cure composites demonstrated similar load to failure, following acid-etching and with or without application of bonding agent. Load to failure was greater overall with light-cure composite resin than chemical-cure composite.[15] Use of light-cure composite for intraoral splints is cost-prohibitive and time consuming, as it requires incremental light curing of very small sections at a time. Dental conditioning and layering chemical-cure composites provide almost limitless opportunity to fabricate a device intraorally. As chemical cure composite sets, contact by oxygen with composite prevents the surface from cross-linking and leaves unlinked monomers.[18] Subsequent layers of chemical cure composite can be applied, and the polymerization reaction can take place with the uncontaminated surface of the cured composite. This oxygen inhibitive layer permits layering of chemical cure composite, while mitigating the concerns with volume-related exothermic heat production, which could damage the pulp.

The patient should be positioned to allow assessment of occlusion during fabrication and customization of the intraoral splint. Use of extraoral intubation techniques (Chapter 61) eliminates the necessity for extubation for the purposes of assessing occlusion. In addition, consideration should be made for patient positioning during composite application and curing. Positioning mandibular fracture patients in sternal recumbency and maxillary fracture patients in dorsal recumbency will optimize the use of gravity during composite delivery. After layering and curing composite or acrylic, the splint can be efficiently trimmed and sculpted with an acrylic bur on a straight handpiece. Care should be taken to ensure that there are no sharp edges of composite and that minimal composite is adjacent to the gingival margin, as gingivitis and periodontitis can occur secondary to presence of a splint at the gingival margin.

Intraoral splints combined with cerclage wires

When managing edentulous patients, or juvenile patients with deciduous or mixed dentition, incorporation of cerclage wires around the mandibles and into the composite splint helps to secure the splint in place without relying on anchorage to teeth (Fig. 31.8). However, with cerclage wires and composite, the exfoliation of deciduous teeth is stalled and eruption of permanent teeth is restricted. Given the short healing time in these patients, normal exfoliation and eruption typically follow removal of the splint and wires.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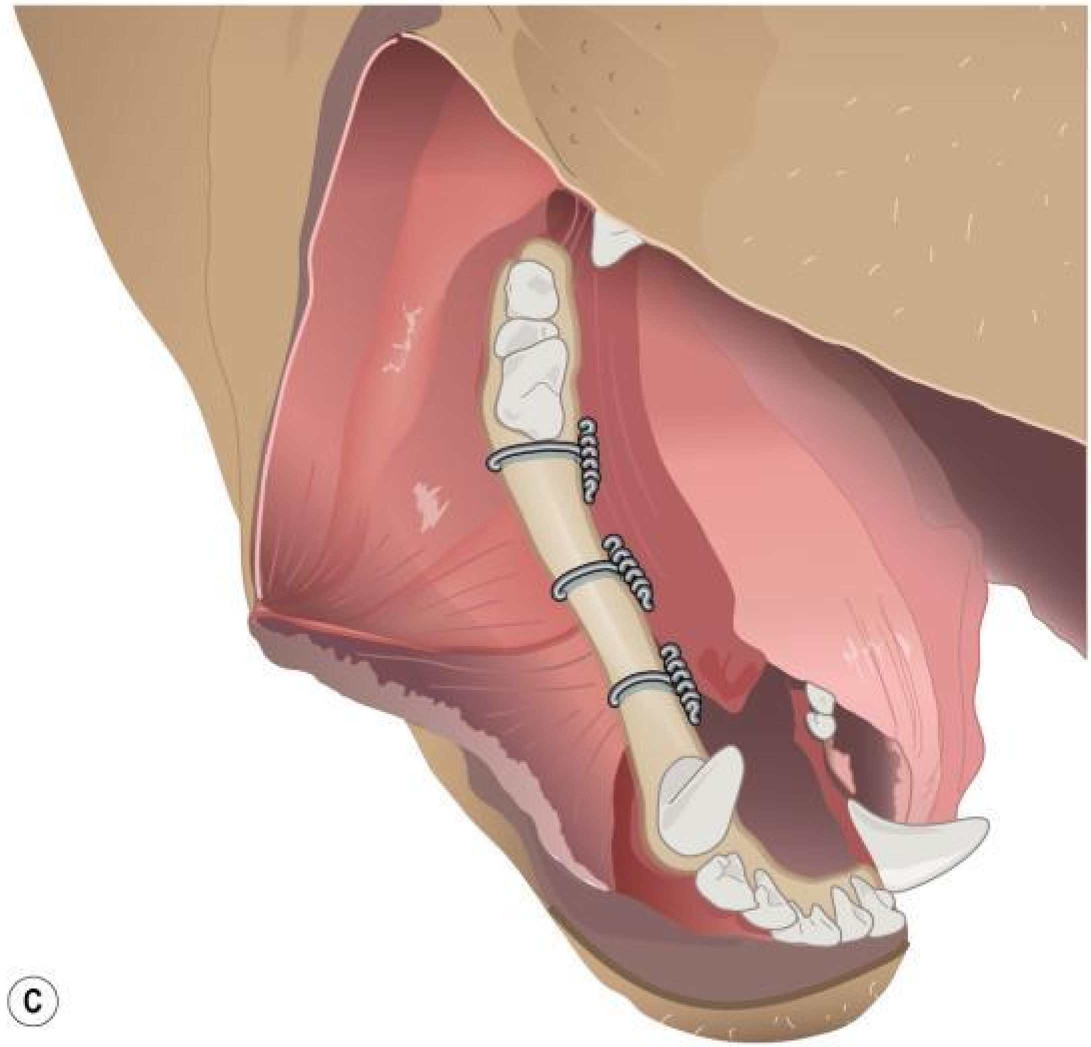

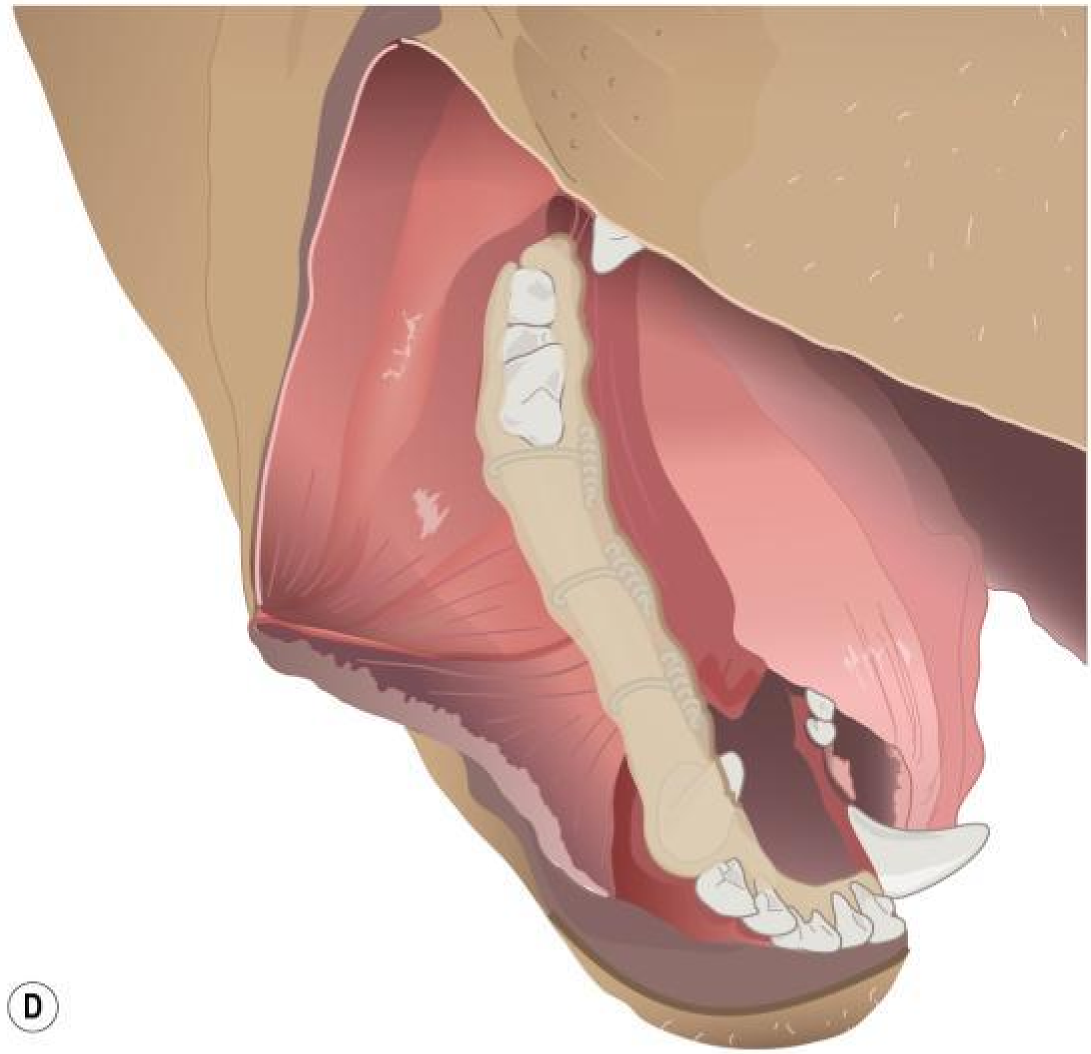

• **FIG. 31.8** **(A)** Incorporation of mandibular cerclage wires involves ventral skin incisions with a combination of sharp and blunt dissection to the ventral margin. An appropriately sized hypodermic needle is passed along the buccal and lingual cortex to facilitate passing of the cerclage wire around the mandible. This illustration demonstrates preplacement of a mandibular cerclage wire. **(B)** Mandibular cerclage wires are placed prior to fabrication of the composite or acrylic splint. The intraoral splint should be anchored in three locations on either side of the fracture by either bonding to tooth structure or secured in place by cerclage wires. **(C)** Once the initial layers of composite or acrylic have cured, cerclage wires are tightened and bent against the composite in a nonocclusal location. **(D)** Final layers of composite or acrylic are placed covering the mandibular cerclage wires and the splint is smoothed.

Patients with multiple missing teeth also benefit from retaining cerclage wires when insufficient anchorage points are available on either side of the fracture. Through simple stab incisions on the ventral mandible, 20-, 22-, or 24-gauge orthopedic wire (depending on patient size) can be directed along the buccal and lingual cortical plates and into the oral cavity at the mucogingival junction. Care should be taken not to entrap soft tissues against mandibular bone. Cerclage wires should be placed prior to application of composite. Following curing of the initial layers of composite, the cerclage wires are tightened, securing the splint against the mandible, and free wire ends are bent against the splint before applying subsequent layers of composite to build the splint up to the final size while covering the free wire ends, preventing them from creating soft tissue damage.

Intraoral splints for salvage treatment of fractures of edentulous mandibles

Edentulous patients can be particularly problematic and challenging for the application of noninvasive fixation devices. Open reduction and internal fixation are usually a better treatment option, but when finances do not allow for surgical treatment, intraoral splints and cerclage wires are an option. Without dental structure present for anchorage, use of composite splints must rely on cerclage wires placed around the ventral mandible and emerging into the oral cavity to stabilize the splint.[6] Following the first layers of composite, the cerclage wires can be tightened in place before additional layers of composite are placed. Sandwiching the cerclage wires into composite and covering the wire twists intraorally will help prevent tissue damage from sharp wire. Composite should be layered on top of cerclage wires, which are placed before splint application and tightened after the initial portion of the splint has been placed and cured. Additional layers of chemical cure composite can then be placed on top of the cerclage wires. Mandibular "J" splints can be fabricated to extend into the incisor tooth region (Fig. 31.9) or "U" splints to extend to the contralateral mandible. By extending the splint into a "J" or "U" shape, stability is afforded to the device to help prevent the splint from displacing in the buccal or lingual direction.

• **FIG. 31.9** A J-shaped intraoral splint can be useful for stabilizing mandibular fractures in the region of the premolar and canine teeth. Spanning the symphysis with composite aids in stabilization of the fracture fragment involving the rostral mandible.

Maxillomandibular fixation using interdental bonding

MMF utilizes alignment and immobilization of the maxillary and mandibular teeth in normal occlusion. This form of fixation is unlikely to be useful in patients with gap defects or comminuted fractures. Alternative forms of this fixation technique involve the use of screws and elastic bands[22] or holes drilled in the maxilla and mandible directly connected by wire.[23,24]

With the refinement and use of dental composites and bonding agents in veterinary patients, immobilization is typically achieved by creating columns of composite connecting each maxillary canine tooth to the ipsilateral mandibular canine tooth (Fig. 31.10).[15,25] Occlusion is maintained in a semiopen mouth position, permitting the patient to pant and to lap water and gruel-consistency food. It is recommended that an esophagostomy tube be placed at the time of fixation to provide supplemental nutrition and/or medications (see Chapter 5). The MMF should be in place for no more than 3 to 5 weeks to prevent risk for development of a functional ankylosis. If 3 to 5 weeks is insufficient for adequate healing, following removal of MMF, the patient can be restricted to soft food and toys are withheld for an additional 3 to 5 weeks to facilitate further bone healing. Alternatively, use of a tape muzzle may permit minimal range of motion while maintaining the jaw relationship in a normal occlusal position with some motion to prevent functional ankylosis.

• FIG. 31.10 Maxillomandibular fixation (MMF) is achieved by applying composite columns connecting the maxillary and mandibular canine teeth. **(A)** The mouth remains in a fixed position, aligning occlusion while permitting panting, lapping food and water, and vomiting if necessary. **(B)** MMF composite bonding is scored and fractured in segments to release fixation.

Use of light-cure composites may be advantageous due to the quick curing capacity of the material. Some variation exists on how much crown surface area is etched in preparation for composite placement. In one investigation of MMF bonding of canine teeth in cats, light-cure composite was significantly stronger (greater load to failure) than chemical-cure composite. The extent of surface area of the crown etched (50% versus 100%) did not yield a difference in bond strength, but increased surfaced area etched was associated with longer removal times and increased incidence of iatrogenic tooth injury.[15] The extent of tooth surface etched prior to composite placement should be weighed against the perceived likelihood that the patient may fracture or detach the appliance.

Use of elastic training/orthodontic anchorage

Fractures of the caudal mandible involving the ramus can be somewhat stable due to attachment of the muscles of mastication. Fractures at this location, including condylar neck fractures, may exhibit slight telescoping of the fracture fragments, resulting in a traumatic occlusion. When considering options for noninvasive management of these injuries, a focus on return-to-normal occlusion may be sufficient to achieve bone healing, which may be a more desirable approach to management than invasive repair. An alternative to MMF includes the use of orthodontic elastics attached to buttons placed on the contralateral side of the mouth to help generate tension when the mouth is open, reducing the likelihood that collapse of the fracture fragments will occur. Orthodontic movement of teeth is not the goal of this technique and, in fact, is an undesirable complication. Application of elastic chains should be such that the elastics are "neutral" (no tension but also not lax) when the mouth is closed (Fig. 31.11). Tension is created when the mouth is open, which helps to resist the tendency for fragments to collapse on the affected side, and helps maintain normal occlusion. An advantage of this technique is that the device can be worn until healing is deemed sufficient, or a similar device can be used to help manage a nonpainful fibrous or nonunion indefinitely in cases that may result in a permanent traumatic occlusion. Orthodontic elastic training can also be considered to aid and maintain reduction of TMJ luxations during the healing period.

• FIG. 31.11 **(A)** Feline patient with a right ramus fracture. Orthodontic elastics have been anchored between the left maxillary fourth premolar tooth and the left mandibular canine tooth to prevent drifting toward the side of the fracture. **(B)**

Elastics are placed in neutral tension when the mouth is closed so that when the mouth is opened, slight tension helps to stabilize the mandible into a normal occlusal position.

Postoperative care and assessment

Postoperative care

Postoperative care recommendation for patients treated with noninvasive repair methods are determined by the type of fixation device employed. Patients with intraoral splints should be restricted from chewing or playing with hard objects. To minimize gingivitis and mucositis due to accumulation of food debris, intraoral splints should be flushed with tap water or a 0.05%–0.12% chlorhexidine gluconate oral rinse after each meal. The use of a water flosser (Waterpik, Water Pik, Inc., Fort Collins, CO) is recommended. Some patients may benefit from an Elizabethan collar to deter them from chewing toys or other objects while the splint is in place. Immediately postoperatively, an Elizabethan collar will also prevent canine or feline patients from traumatizing themselves in attempt to remove an intraoral splint or MMF from their mouth.

Patients treated with muzzle coaptation as the primary method of stabilization risk development of moist dermatitis if not closely monitored (Fig. 31.12). Duplicity of store-bought muzzles affords the opportunity to change muzzles daily, after meals, in order to clean any areas of the face or neck contacted by food. Although an esophagostomy tube is recommended for patients placed in MMF, if a tube is not present and the patient is lapping gruel and water on its own, consumption of food and water should be monitored by daily recording of body weight and observation of eliminations. If intake is adequate, esophageal tube removal can be easily performed without sedation at a follow-up visit.

• **FIG. 31.12** Loose-fitting muzzles (custom-made tape muzzles or commercially manufactured nylon muzzles) must be changed frequently. Due to the nature of the injury, patients are on a gruel-type diet, and if the muzzle is not changed and the face cleaned after each feeding, moist dermatitis may develop.

Appliance removal

All intraoral appliances must be removed to prevent the development of periodontal disease. Any form of stabilization restricting TMJ range of motion should be removed or modified after 3 to 5 weeks to prevent developing degenerative changes of the TMJ. Clients of patients with fixation involving light-cure or chemical-cure composite should be warned about the risk for injury to tooth structure during removal. Care should be taken when sectioning and removing light-cure or chemical-cure composites. Typically, intraoral composite or acrylic splints are scored with a carbide bur on a dental high-speed handpiece and fractured off in small pieces using a bond removing forceps (Direct Bond Removing Orthodontic Plier, Ormco, Orange, CA) (Fig. 31.13). Interdental wires or mandibular cerclage wires can be removed after cutting with wire cutters. An advantage to incorporating the twists inside the oral cavity for mandibular cerclage wires is that surgical exploration is typically not necessary at the time of removal. All of the necessary equipment should be chairside when general anesthesia is induced. Patients with MMF will be unable to be intubated until after the appliance has been partially removed. Anesthesia must be maintained by intravenous route while the appliance is sufficiently removed to allow opening of the mouth and intubation, after which removal of the appliance can be completed once the patient is safely intubated and maintained on inhalant anesthesia.

• **FIG. 31.13** Intraoral splints are removed in segments, following scoring of the splint with a bur on a high-speed handpiece. Bond removing forceps are used to fracture the splint into segments for removal.

Complications

The most common complication with the use of noninvasive techniques is delayed union or nonunion. While the largest advantage to noninvasive repair is not opening the fracture site, the fact that fracture repair is considered semirigid (interdental wiring with composite splinting) or nonrigid (muzzle coaptation and MMF) diminishes the likelihood that primary bone healing can occur. Delayed healing, exuberant callus formation, and fracture nonhealing are all possible.

As with any fracture repair treatment, an absence of visualizing the fracture site results in speculation as to whether the mandibular neurovascular bundle remains intact following injury. Diligent monitoring by the primary care veterinarian should take place to monitor for clinical signs of pulp necrosis in teeth rostral to or around the fracture site. This is primarily indicated by crown discoloration, typically pink, purple, tan, or gray. Intraoral dental radiographs to assess pulp cavity diameter and the presence/absence of periapical radiolucency are indicated to assess pulp vitality. The younger the patient, the earlier that discrepancies can be discerned between pulp cavity diameters.

Teeth in the fracture line should be evaluated at the time of repair to determine whether concerns exist as to the periodontal or endodontal health of the tooth. The goal of fracture treatment does not end with confirmation of bone healing. Endodontal health of teeth in the fracture line or teeth affected by disruption to their alveolar neurovascular structures should be evaluated with serial radiographs 6–12 months apart and be monitored for continued tooth maturation and development.

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CHAPTER 32

Maxillofacial fracture repair using intraosseous wires

Randy J. Boudrieau

Intraosseous wire fixation has historically been the preferred method of maxillofacial fracture fixation in both veterinary and human medicine.[1-4] The rationale for its use was to place a small implant in a location where other modes of fixation were too large or too cumbersome to apply. The basic premise of wire fixation is to use the wire as a rigid suture to reappose and compress the fractured bone fragments together. A prerequisite for the use of wires is that they must be placed along the lines of tension stress, which requires an understanding of the biomechanical stresses encountered. With this mode of fixation, stability is totally dependent on the tension applied to two perfectly apposed fracture fragments, and dynamic compression provided by application of the tension-band principle so as to provide absolute stability. Intraosseous wire fixation therefore is limited to simple, relatively stable fractures, with well-interdigitating fracture fragments. Comminuted fractures and fractures with gaps cannot be addressed with this technique.

Preoperative concerns

Objectives

Proper occlusion and rigid fixation are interrelated objectives; since fractures amenable to intraosseous wiring are relatively simple, they are treated by anatomic realignment and fixation of the bone fragments, which subsequently restores occlusion.[5-7] This anatomic reconstruction provides the necessary base for support of the soft tissues. In comminuted fractures and fractures with gaps, it is not possible to attain this degree of stability with intraosseous wire fixation; therefore other methods need to be selected to provide the appropriate buttress support (see Chapters 31, 33, and 34).

Biomechanical, anatomical, and technical considerations

Lines of stress

The biomechanical principles of the fixation devices used for mandibular and maxillary fracture repair must include an understanding of the functional anatomy (see Chapter 27). Intraosseous wiring techniques rely on the static forces generated by the tension of the wire and by the frictional forces generated between the corresponding bone fragments.[2-4] Therefore adequate stability for healing is provided only with accurate anatomic reduction and sufficient neutralization of two broad, apposing bone fragments.[2,3] Consequently, intraosseous wiring techniques are most successful if all bone fragments can be anatomically repositioned, thereby enabling the bone and implants to share any applied loads.[2-4] Significant comminution or bone loss precludes the ability to obtain precise anatomic apposition of the bone fragments, as it is not possible to achieve continuous interfragmentary compression across all bone fragments.[2-4] Intraosseous wires also only provide two-dimensional stability, as rotation continues to occur around the wires since they are passed through holes of slightly greater diameter than the wire.[2,4]

Because of the small implant size and their use as tight wires to compress two apposing fragments together, it is crucial that the biomechanical limitations of wire fixation be fully understood. Intraosseous wires must be placed along the lines of tension stress in order that the distractive forces of mastication not overwhelm these implants, and yet still effectively neutralize the distractive forces.[8] Application of the tension-band principle takes advantage of the fact that these fixation devices are strongest in tension (all stresses acting parallel to the long axis of the implant).[9]

Mandible

In applying this method of fixation, the tension and compression surfaces of the bone must be considered, as bending forces are the primary distracting forces acting on the mandible that must be neutralized.[10,11] The mandible acts as a long lever arm and resists bending forces with a strong compact layer of bone that lies parallel to the dental trajectory (see Chapter 27).[12] The shape of the trabecular and compact bone is a result of the functional stress of mastication present.[12] Bending moments are most easily neutralized using the tension-band principle.[10,11,13,14] In mandibular fractures, the lines of tensile stress are along the alveolar margin.[7,8] Torsional and shear forces also exist, each most prominent rostrally and at the ramus, respectively. Therefore in addition to addressing the bending forces with the tension-band principle, a second area of fixation also must be considered along the ventral bone margin (see Chapter 27).[8,11,15]

Maxilla

The maxilla has a configuration different from that of the mandible; the bony structures of the maxilla are all buttresses to support the facial frame (see Chapter 27).[12,16,17] Therefore wire fixation is not indicated for most maxillary fractures. Relatively simple fractures that do not involve the buttress support may, however, be treated by this method of fixation provided that there is no potential for bony collapse of the wired fragments. The latter requires that the bone fragments can be "locked" into position when the wires are tightened and that there be no tendency for the reconstructed fragment to collapse on itself. This requirement usually dictates that the bone fragments be quite large.

Anatomical considerations

Bone shape and thickness

The thin bone of the maxilla and mandibular ramus limits the ability to reconstruct the fracture fragments unless they are of substantial size. Multiple small comminuted bone fragments are difficult to fully reconstruct and also attain the desired rigidity. Furthermore, this configuration is likely to collapse on itself as there is insufficient bone stock present to maintain the appropriate bony contours.

Avoiding dental trauma

The biomechanically ideal location of the implants is at the alveolar bone margin. The small wire size enables their placement in the appropriate biomechanically advantageous position while simultaneously avoiding impingement with the teeth.[7] There usually is sufficient space available between the roots of the individual teeth and adjacent teeth below the root trunk to place intraosseous wire fixation into bone without damage to these structures; however, their placement and avoidance of the tooth roots can be challenging (see Chapter 27).

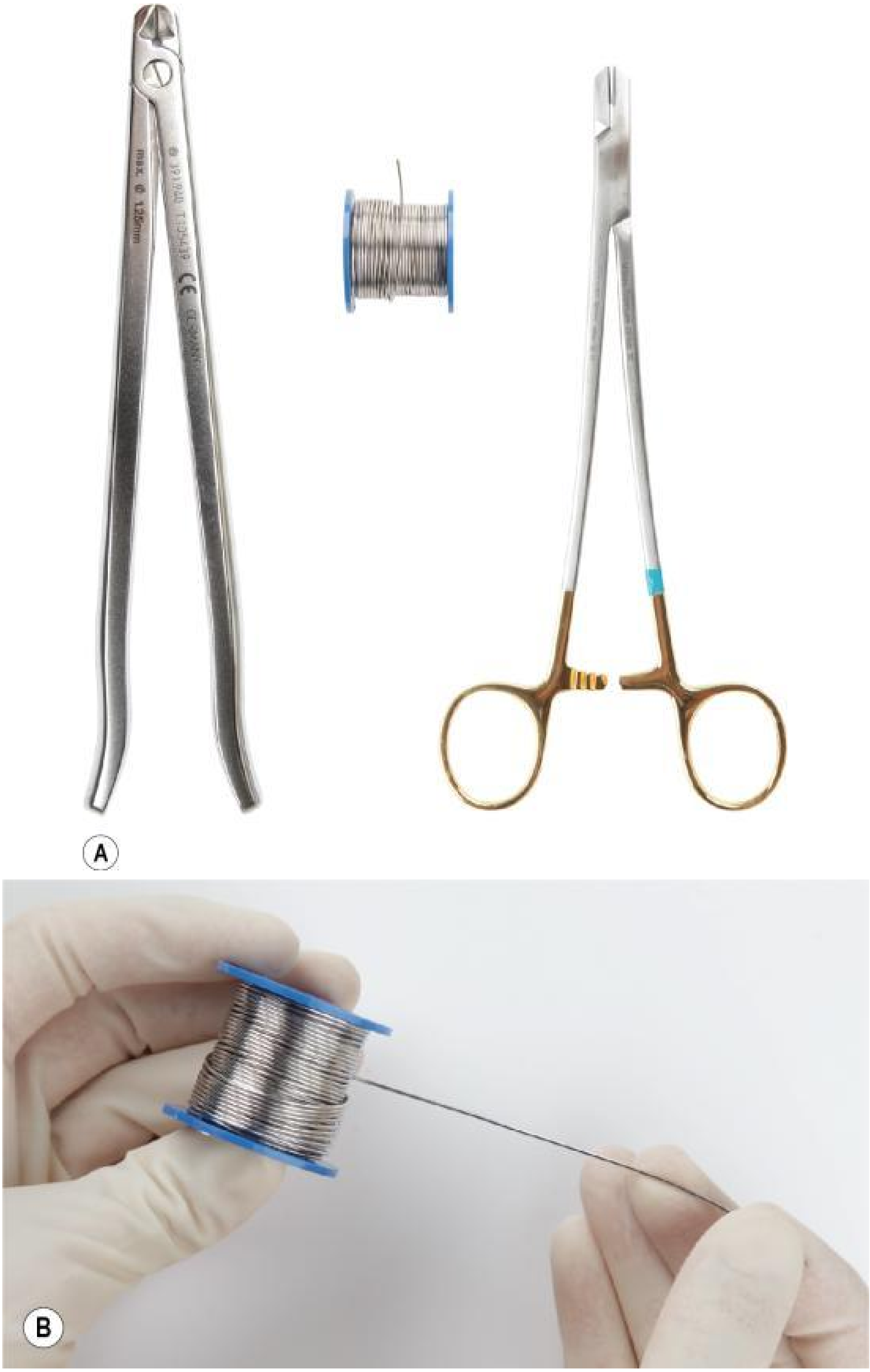
Blood supply and bone healing

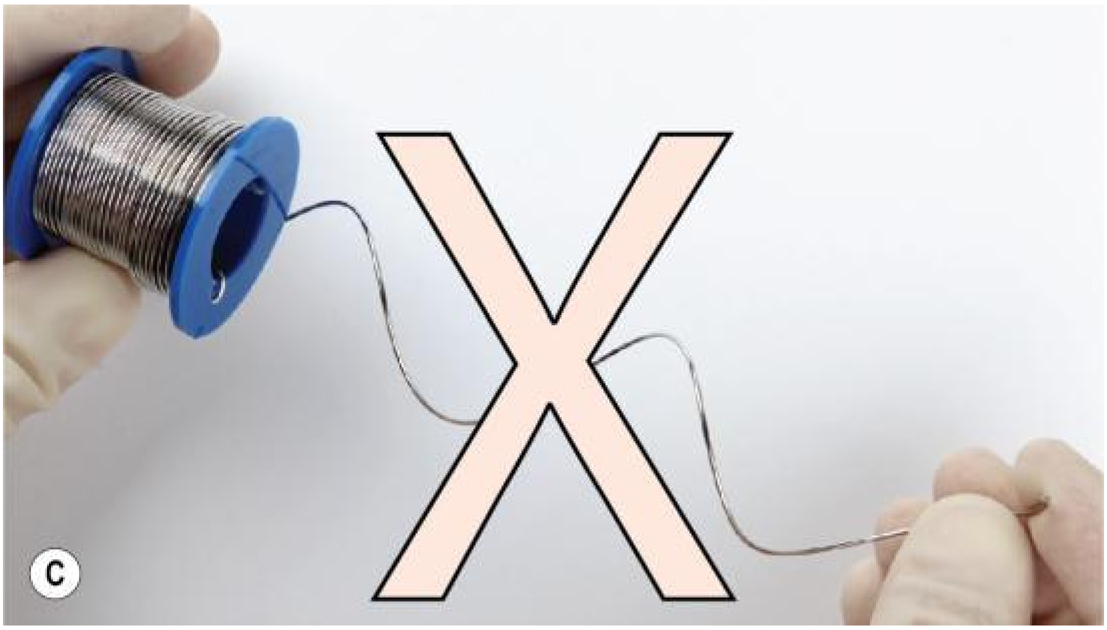
Although craniofacial bone is membranous in origin, bone healing progresses as with any other bone, usually by a combination of direct and indirect bone healing. In the mandible, direct bone healing will occur provided the tension-band principle is successfully applied, i.e., achieves compression across the apposing fracture lines. This can only be accomplished with anatomic reconstruction of the mandibular bone fragments and wire placement along the lines of tensile stress. As such, dynamic compression is generated to obtain absolute stability (the tension-band principle) so as to allow for direct bone healing (see Chapter 2). The mandible is similar in its bone healing response to a fracture when compared with other long bones; however, in the maxilla, despite reapposition of bone, wire fixation does not usually generate sufficient compression to achieve direct bone healing. Because of the maxillary structure of a single, thin lamina of bone, indirect bone healing generally occurs very rapidly.[18-20] The latter is due to the abundant vascular supply to these bones as a result of the large bone surface area per unit volume that is exposed to the soft tissues.

Intraosseous wire fixation

Wire placement

Appropriate anatomic repositioning and tightening of an intraosseous wire are dictated by the wire stiffness and strength. Most orthopedic wire is manufactured with similar quality; however, different manufacturers have wire of somewhat different strength and stiffness (with a different "feel"), and selection is based on personal preference. Orthodontic wire, with its inherent stiffness, is not indicated.[21] Prestretching the wire slightly increases strength and decreases elasticity, which is advantageous for maxillofacial use.[22] Excessive manipulation and kinking of the wire should be avoided, including removing the wire from the spool: unroll the wire rather than pulling it off the end, where it will spiral (Fig. 32.1). Kinked wire is difficult to pass through the drill holes and makes it impossible to maintain even tension.[21] As a general rule, 1.0–1.25-mm wire (18–20 gauge) is used most often; wire <1.0 mm (>20 gauge) is of insufficient strength to maintain the fracture reduction, even in small patients. Although the larger wire is more difficult to pass into the appropriate position, it results in a more secure wire. In relatively stable fractures (see Chapter 27) a single wire across and perpendicular to a fracture and positioned relatively dorsally on the mandible (along the tension-band surface) effectively neutralizes the bending forces.



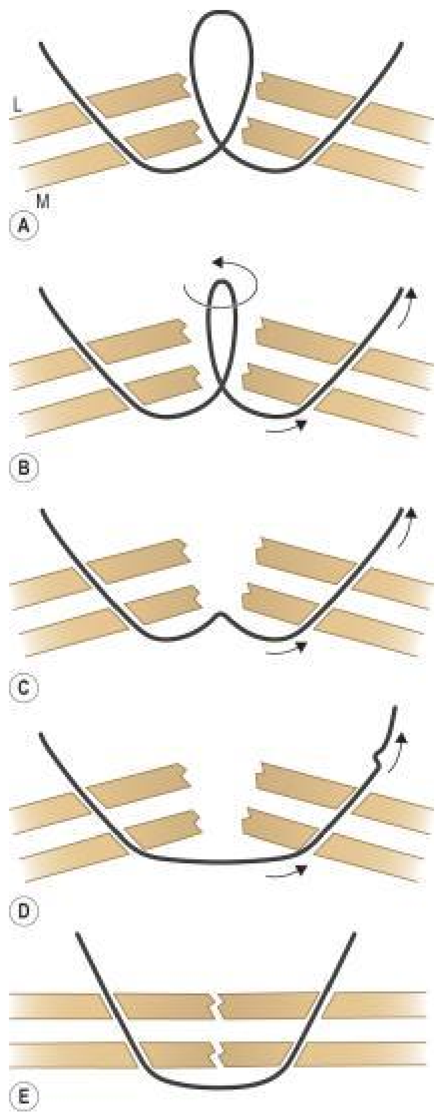


• FIG. 32.1 (**A**) From left to right: wire-cutters, orthopedic wire, and wire twister with a heavy-duty end specific for wire. (**B**) The correct method to remove wire from the spool—by unrolling the wire. Note that a long strand of wire (~8″) is cut from the spool in order to facilitate its application. (**C**) Incorrect removal of wire from the end of the spool, resulting in a spiral wire strand.

The drill holes for the wires are placed such that the wires will cross perpendicular to the fracture line so that, as the wire is tightened, there is no shearing movement when stress is applied. However, this may not always be possible and, in order also to neutralize all shear and rotational forces across the fracture, a second wire in a more ventral position needs to be placed to further secure the fracture. With most fractures the second wire is placed parallel to the first one. With more unstable fracture configurations (e.g., oblique fracture lines), however, the second wire needs to be placed at an angle to the first wire, thus enhancing the stability by using divergent fixation that prevents overriding or rotation of the apposing bone fragments.

Drilling holes for the wire, if performed by hand with trocar-tipped Kirschner wires (K-wires) (0.062″ or 0.045″), allows for more precise placement so as to avoid the teeth (impingement of a tooth root can be palpated when attempting to pass the K-wire). A K-wire can also be used to drill a hole through the mandibular canal, as a smooth K-wire may be less likely to damage the neurovascular structures, compared with a rapidly spinning drill bit that produces greater soft tissue trauma; low-speed drilling with a K-wire is imperative in order to avoid heat necrosis of the bone. The drill holes need to be 5–10 mm away from the fracture in order to prevent an additional fracture, which may result if the holes are drilled too close to the bone edge; furthermore, this placement also diminishes the possibility of pulling the wire through the bone and into the fracture site as the wire is tightened. Conversely, if the drill holes are too far from the fracture line there will be insufficient tension generated and the wires will become loose when placed under stress.

Orienting the drill holes (from the buccal to lingual bone margins) toward the fracture site results in a sloping hole that facilitates both positioning of the orthopedic wire and subsequent tightening. In most mandibular and maxillary fractures, the bone fragments are not very mobile despite obtaining wide surgical exposure of the bone fragments; therefore the orthopedic wire must be passed through drill holes while the bone fragments remain in situ. By sloping the drill hole, the wire may be passed through one fragment, which directs it toward the fracture line, where it is then grasped (Fig. 32.1A). The end of the wire can then be redirected into the second bone fragment by looping it around on itself and passing it through the sloped drill hole in the opposite bone fragment (Fig. 32.2A). After the wire exits this fragment, it is pulled through while simultaneously untwisting the loop in the wire between the two bone fragments (Fig. 32.2B). The area of the loop (and the small kink that develops in the wire) is eliminated from the area that is to be tightened by moving it completely through the drill hole in the second bone fragment (Fig. 32.2C–E).



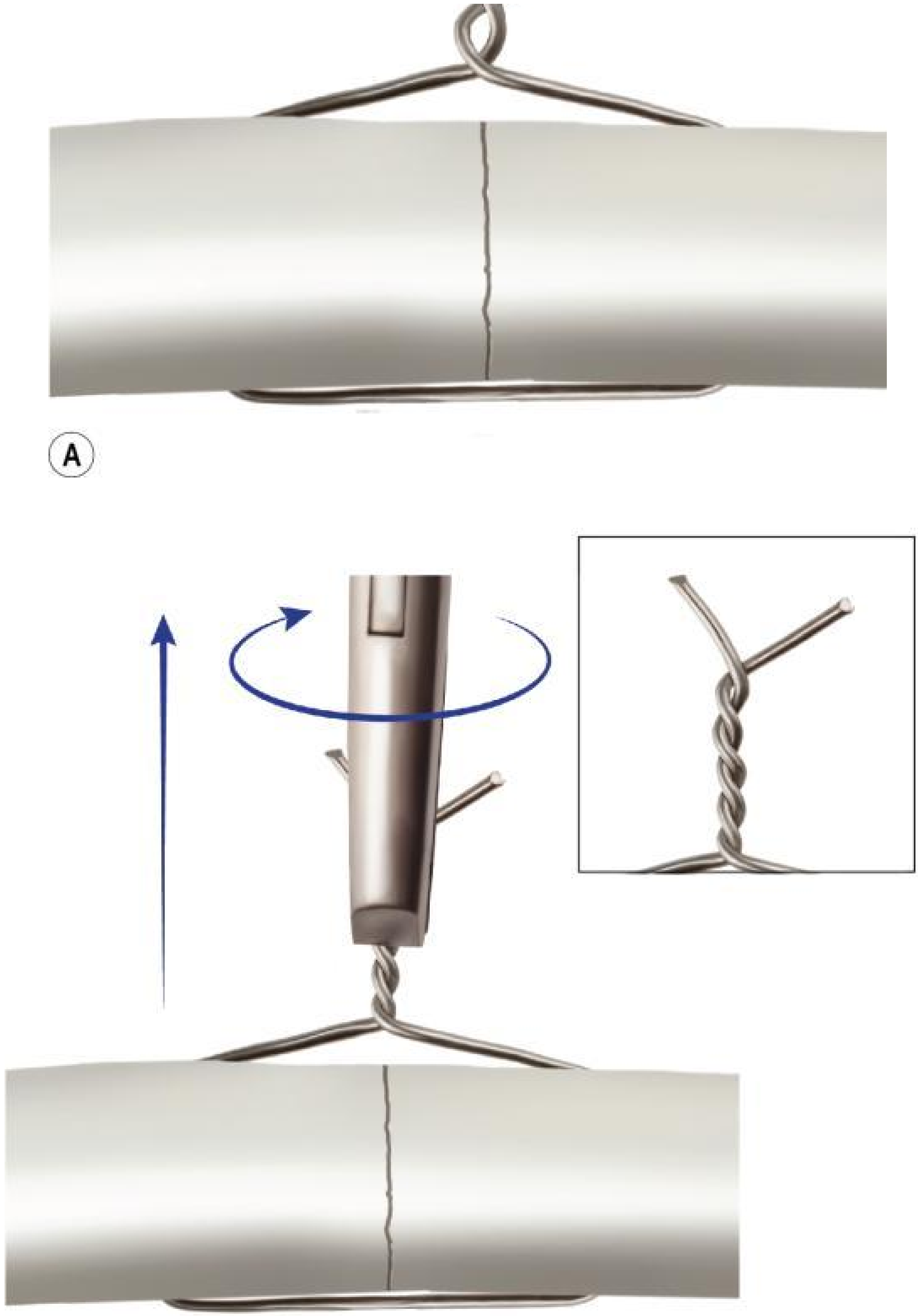
• **FIG. 32.2** (**A**) Orthopedic wire passed through the drill hole in one bone fragment from the lateral *(L)* or buccal to the medial *(M)* or lingual side of the mandible in situ, retrieved, looped, and then passed through the drill hole in the adjoining bone fragment (medial to lateral). (**B**) The wire is pulled through the second bone fragment (reducing the size of the loop) while simultaneously untwisting the loop (**C**), thus avoiding a major kink in the wire. (**D**) The wire then is pulled through the bone fragments a sufficient distance to remove the kinked area from between both bone fragments. (**E**) The bone fragments are reduced as the wire is pulled taut. Source: (From Boudrieau RJ. Mandibular and maxillofacial fractures. In: Johnston SA, Tobias KM, eds. *Veterinary Surgery: Small Animals* (2nd ed), Elsevier, St. Louis; 2018 with permission.)

The technique of passing wires in the mandible can be difficult to perform, especially considering the large size of the wire used: 1.0–1.25-mm wire (18–20 gauge). Passing a small (0.8 mm/0.035″) K-wire into the drilled hole may allow the wire location (drill hole) to be more easily identified within the surgical field. It also is important to use long strands of wire (>20 cm/8″) to facilitate their passage and manipulation. The longer length enables the wire to be pulled through the holes and avoid any kinks or bends that develop with the initial placement within/between the drill holes spanning the fracture; the longer length will also facilitate initial wire hand-tightening. Note that all wires need to be preplaced before commencing tightening. Placing additional wires after a wire has been tightened can create unwanted motion as a result of the manipulation, and often results in previously tightened wire becoming loose.

Wire tightening

Intraosseous wires may be tightened either with a twist knot or a tension loop. The twist method is more easily applied and allows for better control of tightening. The tension loop wire is too easily overtightened with these flat bones with thin cortices, and frequently results in the wire pulling through the bone or in the creation of additional fractures. The hand-twist technique results in a better "feel" for fracture reduction and wire tightening. Regardless of which technique is used, it is essential that the wire be applied tightly to ensure a stable fixation.

Once all of the wires have been passed, the initial wire twist is performed by hand—grasping both free ends of the wires and performing the first one or two twists close to the bone (thus avoiding an excessive number of twists that will impair appropriate wire-twister positioning and grasping of the twist; Fig. 32.3A). Further twisting is performed with a wire twister that is secured within two to three twists from the bone, as it is at this level that the rotational forces of the wire twister are concentrated at the level of the first twist adjacent to the bone (Fig. 32.3B). As the twist knot is tightened, tension must be applied by pulling up on the wire; this tension must be applied equally (even/equal tension) on the two wire strands, which will produce even twists (Fig. 32.3B-D). As twisting continues, the twist must be regrasped with the wire twister to ensure the two to three twist distance to the bone (Fig. 32.3B-D). Holding the twists further away will not result in a tight wire. A tight wire is observed as the twists compress against each other; also, as the wire stretches a dull sheen may be observed. In any event, if the wire is overtightened, it will break and will simply need to be replaced.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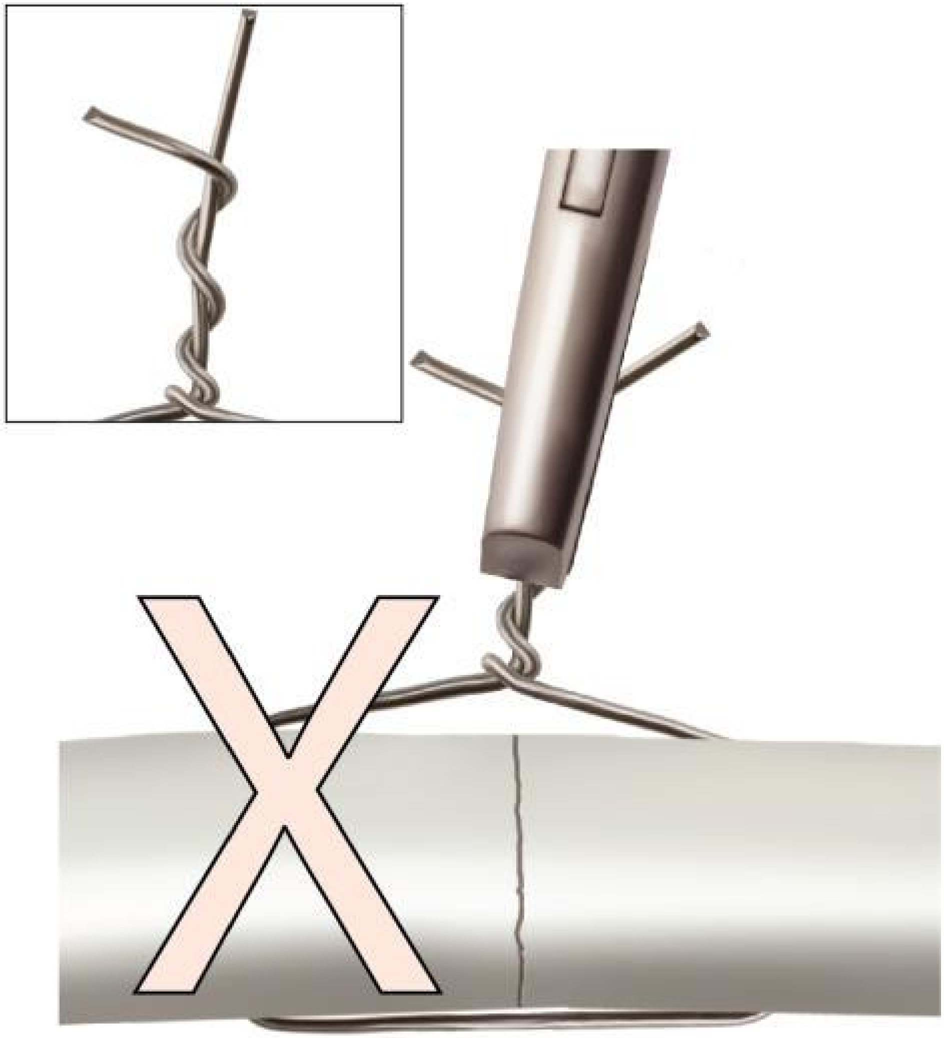

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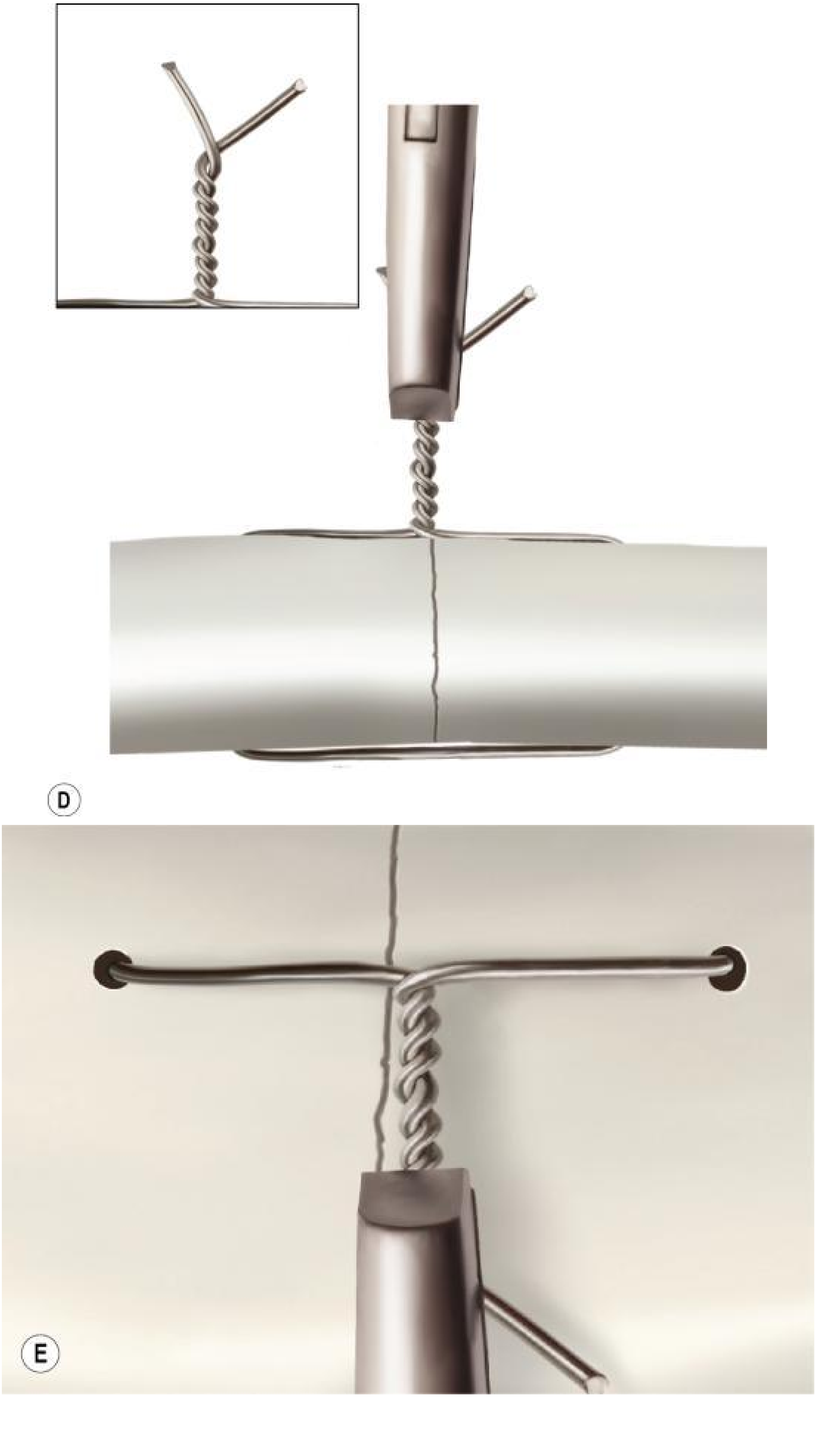
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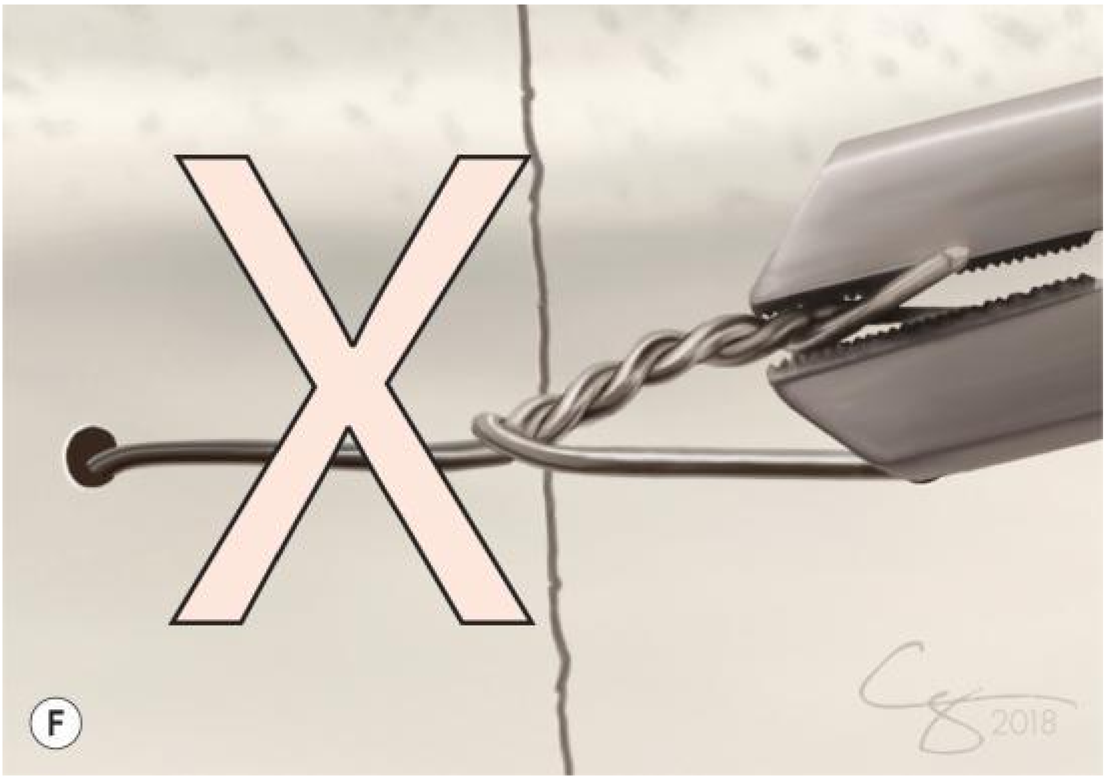
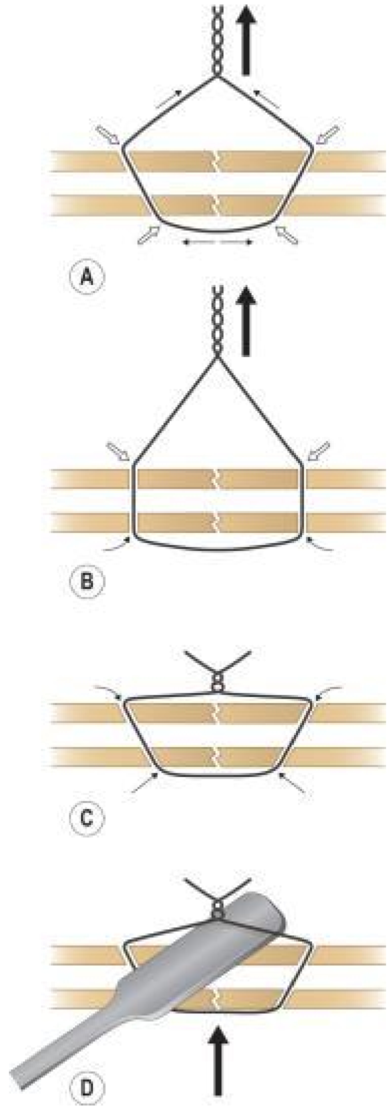


• **FIG. 32.3** Appropriate wire-twisting technique. (**A**) Wire is initially hand twisted so as to start the first one or two twists adjacent to the bone. (**B**) The wire twisters grasp the wire at the level of the first two to three twists, then the wire is twisted with tension applied (evenly with both arms of the twists). Note that as the twisting commences, the wire twisters become located further away from the bone—they must be released and the twist must be regrasped two to three twists from the bone, thus ensuring that the tension generated is close to the bone. Inset: Proper wire twist with equal tension applied to both arms of the wire, resulting in symmetrical wire twist around both arms. (**C**) Improper wire twist with unequal tension applied to the arms of the wire, as a result of pulling up unevenly. Inset: The near arm wraps around the far arm (with the greater tension), resulting in an uneven twist where the far wire arm can simply slide through the opposite wire arm. (**D**) Preparing to bend the wire twist: once the wire is tight (inset), the wire twister regrasps the twist further away from the bone, ~6–8 twists prior to continuing the twist while performing the simultaneous bend (**E**). This repositioning ensures that the wire does not break close to the bone with additional twisting, as the tension generated is now further away from the bone. Note that the wire is bent over perpendicular to the arms of the wire, thus ensuring that the wire remains tight. (**F**) Bending the wire parallel to the wire arm results in wire loosening, in a manner similar to that demonstrated in (**C**).

When tightening the intraosseous wire placed within the mandible, applying even tension ensures that the wire initially slides through the angled drill holes (the obtusely oriented hole on the far side of the bone fragments; see Fig. 32.4A, B); furthermore, the angled drill holes permit the bone fragments to slide into reduction with minimal resistance as the wire is tightened. As the wire twist is completed, the tension is decreased to permit final tightening (Fig. 32.4C). It is important to recognize that the wire will not continue to slide (because of the now acutely oriented wire position on the near side of the fragments). To further ensure that sufficient tension to secure a tight wire is obtained, and the fracture has been firmly compressed, a second instrument is used to temporarily lever under the loop to additionally tighten the wire (Fig. 32.4D), after which the final twisting is completed. It is essential to apply wires tightly in order to compress the bone fragments.



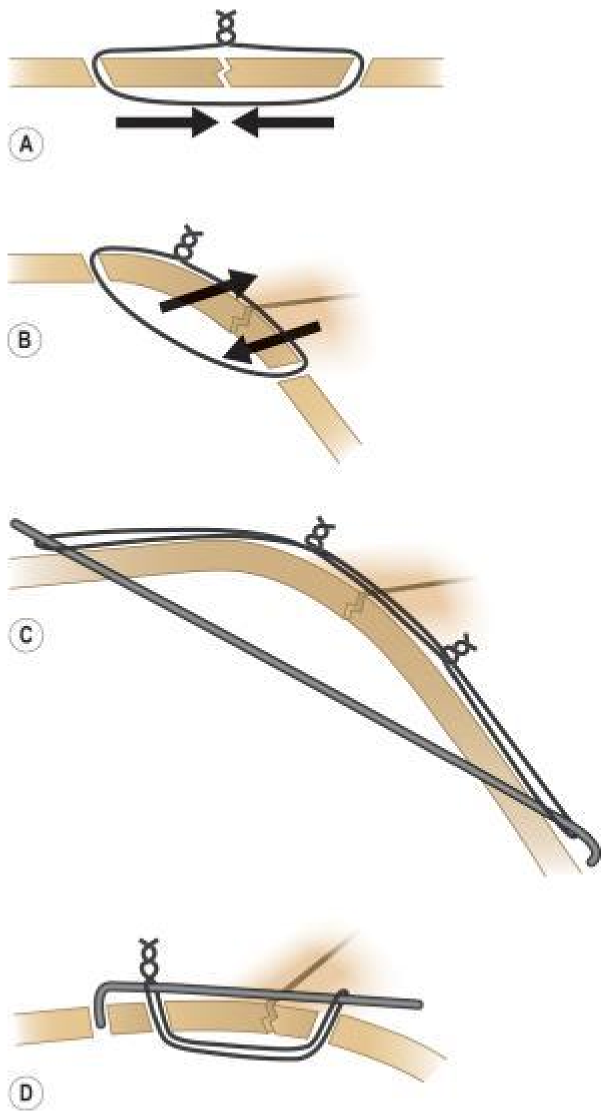
• FIG. 32.4 (**A**) Angled drill holes (toward the fracture line, as observed from the outer (buccal) bone surface) result in obtusely angled corners of the wire at all points *(open arrows)*, thus enabling the wire to slide *(small arrows)* early in the wire-tightening process (*large arrow:* tension applied to the twist). (**B**) If the drill holes are not angled, the angle is too acute for the wire to slide *(curved arrows)* despite appropriate tension applied to the twist, resulting in an inadequately tightened wire. (**C**) As the wire is tightened, and the twist is allowed to approach the bone surface, the angle of wire becomes acute as it exits the bone, and the wire will no longer slide *(curved arrows);* the wire on the opposite bone surface, which maintains the obtusely angled corners *(small arrows)* should be tight at this time. (**D**) Prior to securing the twist, final tightening is ensured by levering a second instrument (e.g., a periosteal elevator) under the wire beneath the twist, ensuring a tight wire on the opposite bone surface *(large arrow)* as the wire will continue to slide through the obtusely angled corners. Source: (From Boudrieau RJ. Mandibular and maxillofacial fractures. In: Johnston SA, Tobias KM, eds. *Veterinary Surgery: Small Animals* (2nd ed). Elsevier, St. Louis; 2018 with permission.)

During the final twisting, to bend the wire twist to lie against the bone, the wire twist is regrasped further away from the bone so as to alleviate the stress concentration closest to the bone, as further twisting or bending will result in a broken wire (Fig. 32.3D). Regrasping the wire further away from the bone allows the wire twisting to be continued as the wire twist is bent over to lie against the bone, thereby preserving tension in the wire. When bending the wire twist, it must be performed perpendicular to both arms of the wire encircling the bone; angling toward either arm of the wire will result in loss of wire tension—and a loose wire (Fig. 32.3E, F). The wire twist is bent over away from the gingival margin and then cut to maintain at least three twists.

Wire patterns

The goal for intraosseous wire application in the mandible is as a simple interrupted pattern, placed along the lines of tensile stress as a tension-band wire. A second stabilization wire is placed parallel to the ventral margin of the mandible. The combined support of these two wires is thus distributed over a greater area of the fracture.

A different situation exists in the maxilla. The thin monocortical bone fragments of the maxilla may be difficult to appose without overriding due to shear forces (Fig. 32.5A, B). Similarly, at areas of bone curvature, there is a greater tendency for the bone fragments to override. In both instances, a K-wire may be needed to secure these fragments so that the shear forces are neutralized. In one technique, a "skewer-pin" technique can be utilized to prevent the overriding (Fig. 32.5C). With this technique, a K-wire is drilled through both bone fragments and a figure-of-eight wire pattern is placed over the exposed ends of the K-wire; the ends of the K-wire are bent over in order to prevent K-wire migration and loss of the figure-of-eight fixation. Both "arms" of the figure-of-eight wire should be twisted to ensure uniform wire tension (large-gauge wire cannot be pulled around sharp bends, e.g., the ends of these K-wires; note this same rationale for the oblique drill holes in the mandible). In an alternative technique, a K-wire may be secured on the outer surface of the bone as an internal "splint" (Fig. 32.5D). The K-wire is secured with orthopedic wire inserted through drill holes in adjacent bone fragments, and tightened over the K-wire applied to the outer surface of the bone spanning the fracture site; K-wire migration can be prevented by bending one end of the K-wire at a 90-degree angle and inserting it into a drill hole in the bone.



• **FIG. 32.5** (**A**) A single intraosseous wire as a rigid suture to reappose and compress *(arrows)* the fractured bone fragments. (**B**) Overriding of the bone fragments *(arrows)* occurs when a wire suture is tightened with an oblique fracture line or on a curved bone surface. (**C**) Placing a K-wire across the fracture site (transfixation pin or "skewer-pin") prevents overriding and bending; the K-wire is secured with an orthopedic wire placed over the ends of the transfixation pin in a figure-of-eight pattern (both "arms" of the figure-of-eight wire are tightened to ensure sufficient tension is generated on both sides of the figure-of-eight). (**D**) A K-wire also may be placed on the outer surface of the bone as a "splint." The orthopedic wire is secured over the K-wire by passing it through two separate drill holes in the bone in this example, and subsequently tightened around the K-wire to secure both bone fragments. Source: (From Boudrieau RJ. Mandibular and maxillofacial fractures. In: Johnston SA, Tobias KM, eds. Veterinary Surgery: Small Animals (2nd ed), Elsevier, St. Louis; 2018 with permission.)

Therapeutic decision-making

Surgical approaches

The specific surgical approaches are discussed in Chapter 29. For intraosseous wiring techniques, the standard ventral approach to the body of the mandible is generally used (separate ventral approaches to each mandible are usually performed to facilitate the manipulation necessary to pass the wires). If necessary, a lateral approach may be used to access the mandibular ramus[23]; however, this is rarely necessary.

The maxilla and incisive bones usually can be adequately approached by an intraoral exposure—simply reflecting the gingiva and alveolar mucosa away from the alveolar bone adjacent to the base of the teeth; alternatively, a dorsal midline approach also may be utilized.

Sequence of repair

The basic principles of fixation can be summarized as follows:

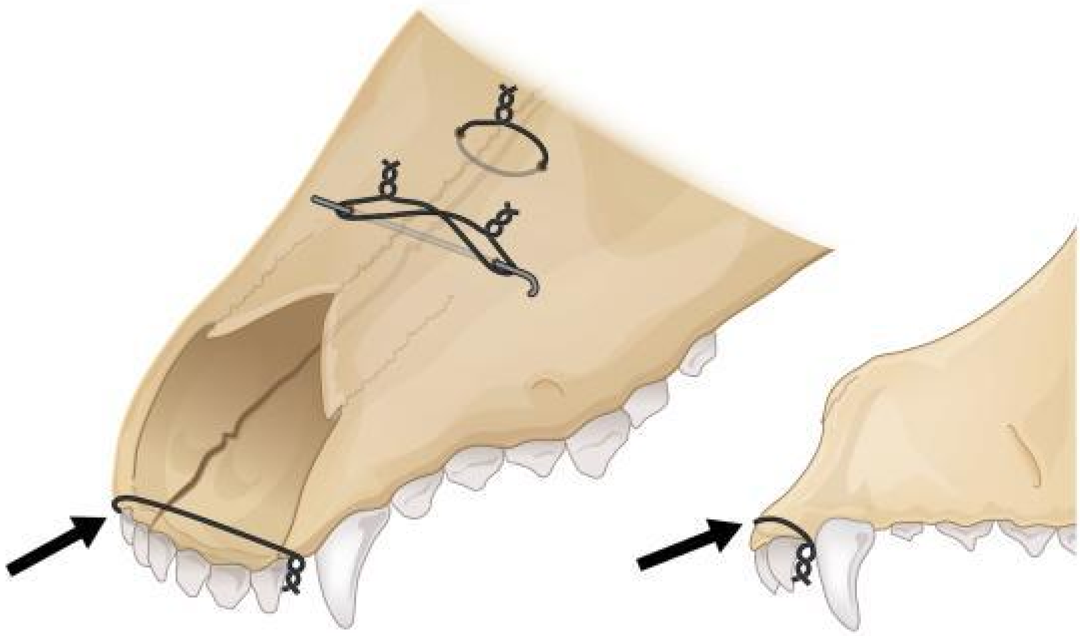
1. Wire placement is planned such that all wires are applied perpendicular to the fracture line whenever possible.
2. A hand chuck and K-wire are preferentially used to place the drill holes, which allows more precise placement, thus avoiding major foramina and the teeth.
3. All drill holes are angled toward the fracture line from lateral (buccal) to medial (lingual) cortex so as to allow the wire to slide more easily along the medial (lingual) cortex and thereby more readily tighten without restriction.
4. All holes are drilled and wires preplaced before final reduction and tightening of all wires.
5. Wires are tightened caudal to rostral. Wires are tightened carefully in order to avoid loosening all previously tightened wires. Symphyseal fractures are wired last.
6. A single 1.0- or 1.25-mm (18- or 20-gauge) tension-band wire usually is adequate to neutralize bending moments; however, a second stabilization wire must be placed a few millimeters away from the tension-band wire, usually parallel to the ventral cortex so as to also neutralize shear and torsion.
7. Wires are twisted evenly with equal tension on each arm that traverses the bone and are bent perpendicular to the wire strand, away from the gingival margin. The wire twist is cut only after it has been bent. All wires are pressed firmly against the bone in order to avoid subsequent penetration of the oral mucosa.

Surgical techniques

Fractures involving the incisive, nasal, and maxillary bones

Intraosseous wire fixation of incisive, nasal, and maxillary fractures is limited because of the thin bone and the difficulty of attaining adequate stability. However, if large fragments of bone are present, this method of fixation may be successful, as the fracture configuration may help maintain the stability and the three-dimensional conformation. Two primary fracture configurations are addressed: longitudinal and transverse (parallel and perpendicular to the sagittal plane, respectively).

It generally is easiest to wire together large right- and left-sided longitudinal bone fragments and then secure the reconstructed fragment to the skull. Midline fractures of the incisive bone are secured by placing a cerclage wire that encircles this bone just caudal to the maxillary incisor teeth and rostral to the canine teeth (Fig. 32.6). For the maxilla, a simple wiring technique (suture) may be performed, although a transfixation pin ("skewer-pin") with a K-wire and a figure-of-eight wire technique usually is more suitable due to the thinness of the bone (Fig. 32.6). This technique is most often performed dorsally.



• **FIG. 32.6** Midline fracture of the incisive bones secured with a full cerclage orthopedic wire placed caudal to the maxillary third incisor teeth *(arrows)*. The twist is bent downward toward the buccal aspect of the third incisor tooth in order to avoid impingement against the mandibular canine tooth when the jaw is closed and to avoid unnecessary trauma to the gingiva. The oblique view of the maxilla demonstrates two methods of securing large left- and right-sided bone fragments on the dorsal surface of the bone: cranially, a K-wire (arrow) is placed as a transfixation pin and secured with a figure-of-eight orthopedic wire around the ends of the pin (see Fig. 32.5); caudally, a simple interrupted suture pattern is used.

Transverse fractures are easier to reduce by first wiring the bone fragments nearest the frontal bone and then successively wiring fragments rostrally (Fig. 32.7). The wires on the dorsum usually are tightened first so as to attain the appropriate realignment of the bone fragments; subsequently, wires that are placed close to the alveolar bone margin (or interdental wires, discussed in Chapter 31) are tightened in order to minimize any tendency for upward displacement of the nose. All wires placed adjacent to the alveolar margin must be placed with attention to the location of the teeth in order to avoid the tooth roots.

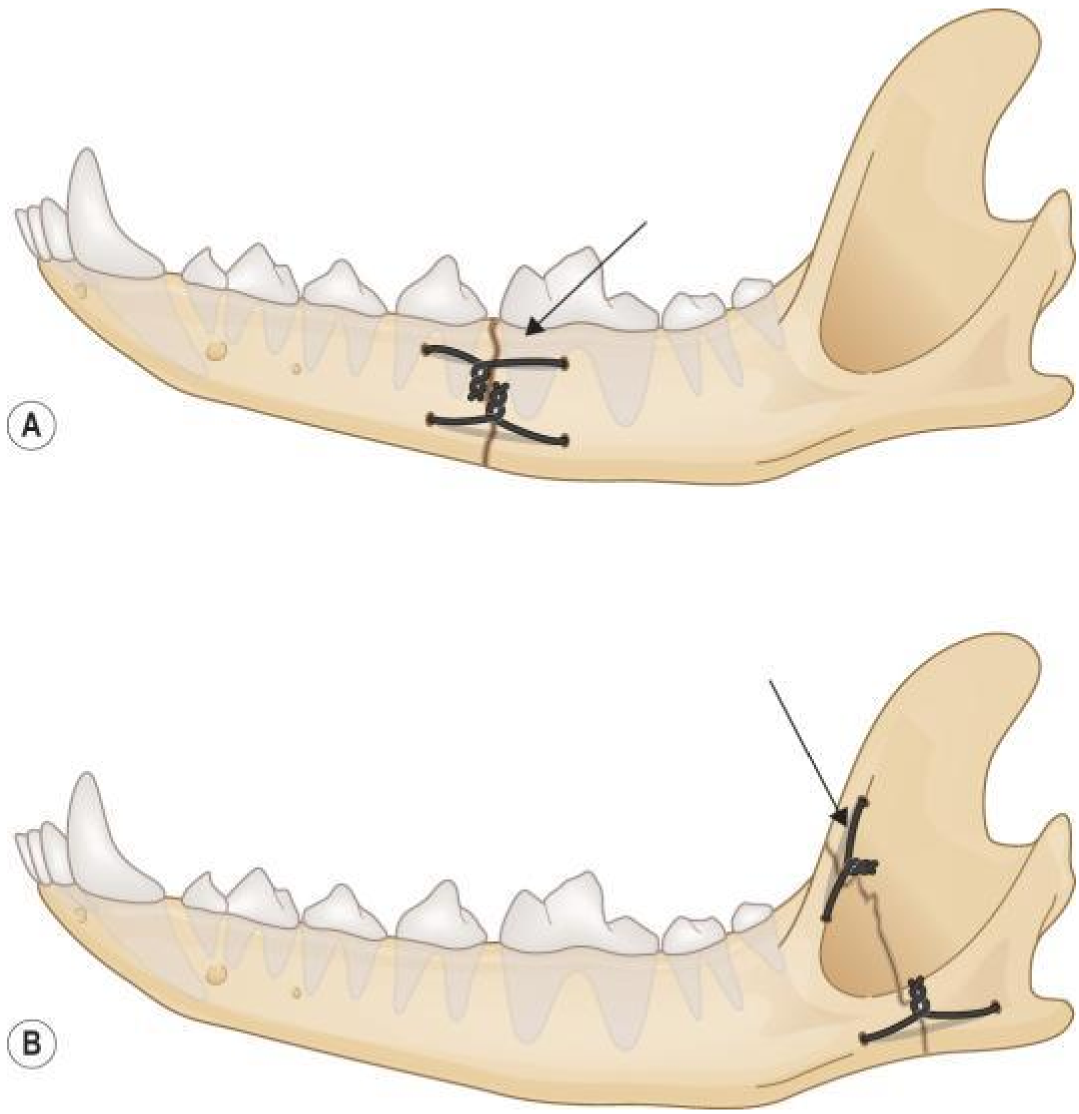
• FIG. 32.7 Oblique view of the maxilla showing reconstruction of multiple transverse fractures using both intraosseous and interdental wires. The most caudal fractures are repaired first, working rostrally. An interdental wire *(large arrow)* is placed around the third and fourth premolar teeth (the wires are placed just below the alveolar surface into the bone in order to prevent them from slipping off the teeth). A similar wire is placed in a figure-of-eight pattern around the canine and first premolar teeth *(small arrow)*. Both of these wires are located along the dental arch to reconstruct the occlusal surface. The remaining wires placed intraosseously as simple interrupted suture patterns, are placed parallel and a few centimeters away in order to further distribute the loads.

Some smaller bone fragments may be held in position by using a K-wire as an internal splint, as previously described. In this manner, the smaller bone fragments can be pulled up to the K-wire that lies on the external surface of the bone. This technique must be used with caution, however, as it provides tenuous stability in most cases. A technique for obtaining much improved stability of these difficult fractures is with miniplate fixation, which permits three-dimensional stability to be obtained (Chapter 33).

Fractures of the mandible

Body of the mandible

Following the ventral approach to the mandible, the bone fragments are visualized. Intraosseous wires are placed such that one wire is placed close to the alveolar margin and a second stabilization wire is placed parallel to the ventral mandibular margin, as described previously (Fig. 32.8A).



• **FIG. 32-8** Simple mandibular fractures in (**A**) the body and (**B**) the ramus. Two wires are placed across each fracture: a tension-band wire *(arrow)* that neutralizes bending forces and a stabilization wire that neutralizes rotational and shear forces. The stability of the repair also is enhanced by distributing the loads over two intraosseous wires. Note that the tension-band wire in A is placed near the alveolar margin and ventral to the furcations of the teeth to avoid damage to the roots.

Ramus of the mandible

Intraosseous wire fixation can be placed identically to that described with mandibular body fractures, using both tension-band and stabilization wires. The tension-band wire is secured along the coronoid crest (ensuring adequate wire purchase in the thicker bone of this region). The stabilization wire then is secured along the ventral mandibular margin, along the condylar crest (Fig. 32.8B). The masseteric fossa is too thin to allow sufficient wire tightening as the wires simply pull through the bone upon tightening.

Because of the thin bone in this region, intraosseous wire fixation must be used with discretion, as three-dimensional stability is difficult to obtain.

Postoperative care and assessment

Activity restriction and diet

If used in the appropriate circumstances of applying a compressive wire suture across two broad (and large) apposing and well-interdigitating bone fragments, intraosseous wire fixation can provide excellent stability of fixation. Patients can eat and drink normally within 24 hours after surgery, limited only by the degree of postoperative swelling that is present. The ability for oral prehension is regained immediately postoperatively. The only limitation to the diet is to use soft food. Chewing on hard objects is not permitted for the first 4–6 weeks postoperatively. Playing with any toys, balls, sticks, etc., which the animal chews, is to be avoided.

Postoperative assessment and wire removal

Maxillofacial fracture repair typically is evaluated radiographically at 6 weeks postoperatively (or sooner if clinical signs indicate possible complications). Once the fracture is considered clinically healed, the wires may be removed, using the same surgical approach; however, they are often left in place. Most buried wires will become covered by bone during the healing process. Erosion of the oral mucosa overlying the wire twists is always a possibility. Wire that penetrates the gingival or alveolar mucosa is removed after healing is complete. Interdental wires always are removed. If there is evidence that the intraosseous wire is loose, it also should be removed.

A simple method of removing wire is to cut it on one side of the twisted knot, grasp the twist with the wire-twisting forceps, and slowly wind the wire onto the twisting forceps. This method will permit even large-diameter wire to be easily removed.

Complications

The most common complication of wire fixation is reduction failure. This complication occurs because of technical failure(s) by the surgeon, either because of inappropriate application of the fixation (most often inaccurate reduction and/or improper wire tightening) or inappropriate case selection (comminuted fractures).

Technical failures

Failure of anatomic reduction

Failure to anatomically reduce the fracture fragments exactly will result in an unstable fixation. In this situation, the bone fragments are not brought into apposition, and therefore there is insufficient contact between the two bone surfaces. When the wires are ineffective in compressing these fragments together, stability is not obtained. Consequently, the fixation becomes loose, resulting in further loss of the marginal reduction obtained. Moreover, the failure to anatomically reduce the bone fragments results in malocclusion. Malocclusion, in addition to adversely affecting function, also will result in fixation failure as abnormal leverage is exerted against the fixation devices that, as noted, are tenuous at best. The abnormal leverage contributes to motion at the fracture site and subsequent loosening of the fixation.

Loose wires will further compromise the ability of the fractures to heal, as the wire will interfere with the initial revascularization to the fracture. It is often said that a loose wire is worse than no wire!

Incorrect wire location

Failure to place the wires in the appropriate biomechanical position predisposes the fixation to failure. The wires must be placed along the tension-band surface of the bone in order to adequately neutralize the large bending forces present in the mandible. Similarly, an additional stabilization wire also is indicated to adequately neutralize the shear and rotational forces in the mandible. Ignoring the biomechanics of the mandible inevitably leads to fixation failure.

Inadequate wire tightening

Inappropriately tightened wires will lead to fixation failure as the wires will not sufficiently compress together the apposing bone fragments. General orthopedic principles of wire tightening must be followed.[24] Twisting with uneven tension on the two strands of wire, continued twisting after adequate tension is achieved, bending the twist over after twisting is completed, and bending the twist back parallel with the wire loop all result in loss of tension or metal fatigue, which will result in premature loosening or breakage of the wire.[24]

Inappropriate case selection

Case selection is crucial to this method of fracture fixation, as three-dimensional stability is only obtained with the help of the fracture configuration. Large bone fragments are necessary; moreover, the fixation is totally dependent on tension, and the resulting compression provided between two perfectly apposed bone fragments. While wiring multiple small bone fragments may be technically feasible, anatomic reduction of each and every fragment is rarely attainable. Similarly, any gaps present in the reduction preclude this method of fixation; attempting to bridge small gaps by spanning wires across a gap to adjacent bone fragments will not be successful, as this form of fixation cannot function as a buttress device.

Wound healing complications

Soft tissue coverage dehiscence

Adequate soft tissue coverage over intraosseous wires is essential to ensure satisfactory healing and prevent infection. In the presence of loose wires, dehiscence will usually occur. Loose implants will interfere with revascularization and also act as a foreign body, both of which predispose the bone to infection.

Infection

Given the open nature of most maxillofacial injuries, intra- and postoperative treatment with a broad-spectrum antibiotic is advisable. For long-standing infected wounds, intraoperative sampling for bacterial culture and susceptibility testing will help ensure appropriate antibiotic selection for postoperative treatment.

Infection will occur, despite appropriate antibiotic therapy, if any bone fragment instability is present. This is a result of implant loosening. Implant failure most commonly results from intraoperative technical failures, improper implant application, or improper case selection.

Delayed/nonunion

A delay or failure of bone healing is once again a result of instability. These are the same factors that predispose the bone (and soft tissues) to infection. Treatment is identical, and has been described previously.

Teeth

Damage to the tooth roots when drilling holes for the wires can result in a compromised blood supply and alternative bacterial access (along the pathway of this drill hole and subsequent implant placement). Pulpal necrosis can thus occur, requiring an endodontic procedure or tooth removal. Any tooth removal must await bone healing, as the creation of a gap along the tension-band bone surface will result in implant loosening and may cause total collapse of the repair.

Sequential evaluation of the repair is essential not only to assess healing but also to identify any potential problems with tooth roots that may be adjacent to the wires. Reevaluation must be concerned with the health of the teeth in addition to the healing of the bone; most often this evaluation is performed with dental radiographs. Iatrogenic dental injury secondary to the surgical fixation technique will continue to cause pain and discomfort, and needs to be addressed once the bone has healed.

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CHAPTER 33

Maxillofacial fracture repair using plates and screws

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Miniplates and locking plates are well suited for many mandibular and maxillary fractures in the dog and cat and are especially useful for fractures located in the mid–mandibular body, caudal mandible, and maxilla. However, because of the small size of miniplates, they do not impart the degree of rigidity that is normally associated with plate and screw fixation. Understanding this point, and the fact that any implant is strongest in tension, generally implies that this implant's use be limited to the lines of tension stress in the mandible. Therefore, in most applications, miniplates are placed in this area using the tension-band principle. Plate fixation, especially using locking plates, additionally provides the ability to obtain bridging support for comminuted fractures or those fractures that have a gap due to bone and/or tooth loss.

Preoperative concerns

Cosmetic appearance versus function

Mandibular and maxillary injuries in dogs and cats may result in severe functional deficiencies and disfigurement. Early repositioning and fixation of the bone fragments and repair of accompanying soft tissue injuries will permit restoration of function and appearance.

Because of the excellent blood supply to the head, vascular compromise to the bone or the soft tissues is unlikely, and healing generally occurs readily. Complications such as infection or delayed healing are unusual unless the injuries are not presented for repair in a timely fashion or repair has been performed with inadequate fracture fixation methods. Neurologic injury may occur, and neurogenic muscle atrophy may result from the severity of the original injury and the superficial location of the nerves of the head.

Consequences of suboptimal or no fixation of maxillofacial fractures include subsequent cavitation, associated soft tissue complications (e.g., exophthalmos, enophthalmos, and facial paralysis), delayed union or nonunion, and sequestrum formation (particularly with fractures involving the frontal or nasal sinuses).[1-3] Without rigid support of the face, contracture of the fibrous connective tissue produced during healing may result in cavitation and deformities of these areas (Fig. 33.1).

• **FIG. 33.1** An 8-year-old St. Bernard was presented for evaluation after having sustained severe maxillofacial trauma as a puppy. The severe facial deformities, including malocclusion, are the result of healing of the facial bone injuries without the appropriate skeletal support. Malunion and nonunion of the historic bone fractures were noted on computed tomography images, primarily on the right side of the face, which resulted in cavitation and deformities to the facial skeleton.

Proper occlusion and rigid fixation are interrelated objectives; treatment of maxillofacial fractures by anatomic realignment and fixation of the bone fragments restores occlusion,[4-6] results in primary bone healing (see Chapter 2), and provides the necessary base for support of the soft tissues. Rigid reconstruction and reestablishment of occlusion will also alleviate pain and discomfort, facilitating rapid healing. In severely comminuted or nonreconstructible fractures, or fractures with gaps, this degree of rigidity is more difficult to achieve, and secondary bone healing results (see Chapter 2); assessment of dental occlusion must be utilized to confirm the accuracy of the surgical reduction.[4-7] Secondary bone healing may result in large callus formation, which could impair function and cause discomfort.

Other head trauma

In general, oral and maxillofacial trauma should be considered an urgent, rather than emergent, indication for surgery. Trauma to the head may involve brain injury and/or cervical spine injury.

In the face of any neurologic deficits, or uncertainty of the patient's neurologic status, surgery is delayed to better assess other life-threatening issues. A short surgical delay permits a more thorough evaluation and allows time to coordinate a multidisciplinary approach to patient care. In human maxillofacial trauma, "early" and "late" repairs are defined as <48 or >48 hours from admission to surgery, respectively. Length of hospitalization and rate of complications (e.g., infection) are similar, which further supports such a judicious approach.[8]

The function and appearance of the eye depend on the support provided by the surrounding bone and soft tissues. Zygomatic arch fractures and fractures of the maxilla at the base of the orbit or zygomatic arch (medial and lateral buttresses) will directly affect both cosmesis and function. Providing the necessary support and stabilization in this area is difficult without the use of a bridging support. Miniplate fixation is best suited for these fractures; additionally, mesh implants may be useful for these areas. The latter are more frequently used in humans due to the full circumferential bone construction of the orbit.[9]

Anesthetic considerations

Patient positioning must allow unimpeded access to the head and face and simultaneously permit an avenue for anesthetic management (see Chapter 7). Dental occlusion must be used to guide the fracture reduction in all but the simplest fractures. Endotracheal intubation therefore is performed via a pharyngotomy or transmylohyoid access to allow assessment of occlusion (see Chapter 61).[6,10,11] Pharyngotomy permits wider access to the mandibles and is generally preferred for ventral mandibular surgical approaches.

When using occlusion to guide orthopedic repair, standard practice in human maxillofacial surgery is to first secure maxillomandibular fixation (MMF), i.e., wiring the jaw closed so as to ensure appropriate occlusal apposition.[12-15] MMF also can be applied in the dog and cat to guide surgical reconstruction. If MMF is not applied, e.g., in simple fractures where anatomic reconstruction is used to guide the surgical reduction, standard orotracheal intubation may be performed, but it is essential that the surgical draping include access to the oral cavity to assess occlusion intraoperatively. The latter assessment remains limited, however, and if any doubt exists with the ability to reconstruct the fracture anatomically, bypassing the oral cavity with the endotracheal tube is performed to enable accurate assessment of occlusion, either temporarily secured by closing the mouth with manual reduction or temporary MMF applied intraoperatively.

Surgical considerations

The skull and mandible in the dog and cat may be divided into areas that are more or less suited for plate fixation. These areas generally are the buttresses of the face (facial frame) and along lines of tensile stress of the mandible, which are particularly suited for miniplates. Locking plate fixation may be used with larger plates or miniplates, including hybrid fixation, which combines conventional and locking plate principles.

Miniplates may be used in most areas and are especially useful in the maxilla and at the junction of the mandibular body and ramus. In the latter area, the rapidly changing bony contours and the thin bone of the masseteric fossa preclude the use of most fixation devices.[16]

Areas of thicker bone along the coronoid crest and ventral margin to the angular process are most suitable for securing implants.[17]

Biomechanics

Lines of stress

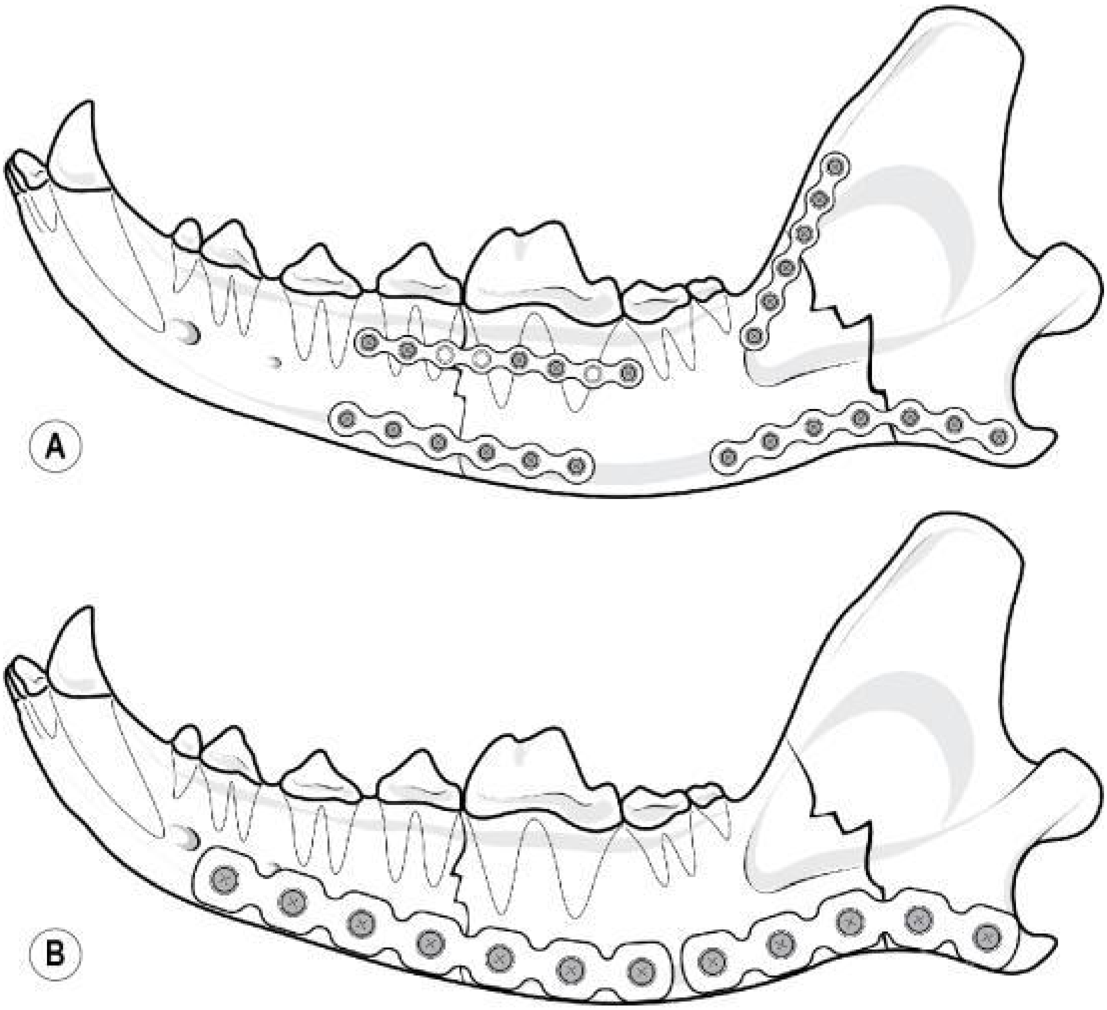
An understanding of functional anatomy (see Chapter 29) is required when selecting fixation devices used for mandibular and maxillary fracture repair. When applying miniplates, it is crucial that their biomechanical limitations be understood. Placement along lines of tension prevents the distractive forces of mastication from overwhelming the implants and allows the implants to effectively neutralize these forces.[18] The tension-band principle reflects the fact that all fixation devices are strongest in tension.[19]

Maxilla

The maxillofacial area is supported by a number of anatomical buttresses that distribute the masticatory forces arising from the mandible (see Chapter 29). These buttresses are present in the rostral/medial, lateral, and caudal planes and make up a basic truss or frame of pillars of reinforced bone.[17,20,21] Reconstruction of such buttresses, which is most readily provided with miniplate fixation, restores three-dimensional (3D) structural support. Because of the small size of these reinforced areas, and the thin bone adjacent to them, small plates that can be contoured to match the bone shape—and screws with a fine thread pitch (so as to obtain purchase in thin bone)—can effectively span these areas and simultaneously provide the requisite support. Locking fixation, however, provides greater implant security in these locations with limited screw purchase, i.e., thin bone.[22]

Mandible

The mandible acts as a long lever arm and resists bending forces with a strong compact layer of bone that lies parallel to the teeth.[21] Application of fixation must consider the tension and compression surfaces of the bone, especially when using smaller implants (miniplates) in larger breeds of dogs (see Chapter 27), as bending forces are the primary distracting forces acting on the mandible that must be neutralized. Bending moments can be addressed using the tension-band principle.[17,23-25] In cases of mandibular fractures, this location is near the alveolar margin, which most effectively neutralizes the bending forces.[18,19] A second device is typically added to further neutralize the shearing and torsional loads that also exist; typically, two plates are applied parallel to and a few millimeters apart from each other—a tension-band device placed on the traction side of the bone (alveolar margin) and a stabilization device placed on the compression side of the bone (buccal aspect of the ventral margin) (Fig. 33.2).[23,26,27]



• **FIG. 33.2** Drawing of a dog mandible depicting two different reconstructible fracture combinations: mandibular body and ramus. **(A)** Both an alveolar tension-band plate and a more ventral stabilization plate are placed on the buccal surface of the mandibular body (note that the alveolar plate is a few millimeters below the gingival attachment in an attempt to avoid the teeth, which provides added opportunity to avoid the tooth roots when placing screws); caudally, the tension-band plate is placed along the coronoid crest, and the stabilization plate is placed along the angular process. Note that the alveolar plate is smaller, with more screw holes per unit length, permitting greater options to place screws so as to avoid the tooth roots. **(B)** Alternatively, in body fractures, a single and much larger plate can be applied at a ventral and buccal position. This single plate technique may also be used on the ramus.

This principle assumes fixation of a reconstructible fracture with absolute stability to maximize the biomechanical advantage; however, this is not always possible due to the presence of teeth, and especially in smaller breeds of dogs where the relative tooth:mandibular height is proportionately greater than in larger breeds. In such cases, an alternative approach may be considered, e.g., utilizing a single larger plate along the buccal aspect of the ventral mandibular margin (Fig. 33.2).[28,29] Additionally, in nonreconstructible fractures, the latter approach may be preferred when applying a locking plate with relative stability.[30] A larger implant, although limited by location, can better neutralize the bending forces that are aligned with the edge of the plate (in-plane bending).[31]

Anatomy

One of the issues with fixation of maxillary fractures, and also fractures in the mandibular ramus, is the lack of adequate bone purchase for most implant systems. This is evident in small animals when attempting to rebuild the maxillary buttresses, where limited space is available to place implants and where bone thickness is approximately 2 mm. Greater bone thickness is present in the mandible, although areas of the ramus (e.g., the masseteric fossa) are generally less than 2 mm thick. If plate application is along the coronoid crest and the condylar process, the masseteric fossa is avoided. However, implant placement along the coronoid crest is limited and complicated by the very rapid changes in bone contour as it joins the mandibular body.

Avoiding dental trauma

The biomechanically ideal location of implants, near the alveolar margin, may not always be achievable, as most fixation devices will interfere with tooth roots and neurovascular structures adjacent to tooth roots. In the dog and cat, these structures comprise a majority of the bone volume along the tooth-bearing portions of the mandible and maxilla. Therefore implants must be placed so as to avoid these structures (i.e., placed on either the more ventral aspect of the buccal mandibular surface or at a level dorsal to the infraorbital foramen on the maxilla), which is not the most biomechanically advantageous position. Miniplates and screws may be applied without interfering with the tooth roots closer to the alveolar margin of the bone.[32] Screw placement, however, is limited to areas between tooth roots and areas where teeth are not present. Screws can be angled away from the tooth roots, but this angle is limited, and a better alternative is to leave the plate hole open; if all plate holes cannot be filled, a longer plate must be utilized in order to achieve a minimum of three screws on either side of the fracture. In reconstructible fractures utilizing the tension-band principle for stability, an alternative solution is to place an intraosseous or interdental wire along the alveolar border and a ventral plate. There are further limitations with rostral mandibular fracture locations, where the large canine tooth roots occupy almost the entire rostral mandible.[32]

Alternatively, a single larger locking plate applied more ventrally on the buccal surface may be used. The principle to be considered in this instance is to maximize the mechanical working distance of the plate across the fracture gap, while also placing a minimum of three locking screws on each side of the fracture.[28,33]

Avoiding iatrogenic damage to tooth roots with any fixation device is an important principle. Any penetration of the tooth root likely will result in pulp necrosis due to interference with its vascular supply.[34,35] Furthermore, screw penetration creates a tract that can provide bacterial access, resulting in infection and, subsequently, a periapical lesion.[35] There are, however, instances wherein a tooth root is penetrated by a fixation device (including intentional fixation in areas with limited access to bone, e.g., region of the canine teeth). In these instances, tooth extraction or root canal treatment must be considered.

Plate fixation features

Although plate fixation provides 3D stability, it is imperative to understand their application under the different circumstances of absolute and relative stability with reconstructible and nonreconstructible fracture fixation methods, respectively. Grasping the concepts of strain theory, and the mechanical and biologic working distances, is essential to the successful implementation of the fixation methods described and the resultant success or failure of the bone healing process. These concepts are beyond the scope of this chapter but can be found in traditional orthopedic texts.[33,36-46]

Implant (screw) purchase

The only limitation regarding screw placement, and its bone purchase, relates to the size of the screw relative to the thickness of the bone. The thickness of the bone must be at least twice the thread pitch distance to develop compression at the fracture site.[43,46-49] The fine thread pitch of the screws (≈1 mm) is such that bone purchase is adequately obtained in any thickness >1 mm for neutralization plate fixation, although a minimum thickness of 2 mm is required to provide sufficient screw purchase to obtain compression plate fixation.[42] Plates are positioned (and contoured) to follow the thicker sections of bone so as to maximize screw purchase. In areas with thin bone, screw purchase may be affected by the inability to generate sufficient compression of the plate against the bone, and in such instances, a locking screw will improve the strength of the fixation as both a fixed-angle construct (where the entire construct shares the loads) and no requirement to compress the plate to the bone.

Screw application

Miniplate screws (2 mm) are self-tapping and have a fine thread pitch (≤1 mm) that provides excellent bone purchase with minimum torque. Newer screws have either a deep hex head or a self-retaining torx recess head (StarDrive, DePuy Synthes, West Chester, PA; Kyon Veterinary Surgical Products, Boston, MA), which simplifies their application (self-retaining in the tip of the screwdriver) and permits proper application of torque (maintains its position without stripping within the recess of the screw head). The self-tapping nature of the screw allows ease of insertion into thin bone and avoids potential enlargement of the screw hole if a tap were used.

Drill holes are usually made of the same diameter as the screw core to obtain a more secure fit at the screw-bone interface. In maxillary fractures, where drilling is limited to a single, thin cortex, a drill stop may be used to prevent the drill bit from overpenetrating, e.g., into a sinus or into the brain with cranial fractures (these depth measurements may be obtained from a preoperative computed tomography [CT] study). Because of the thin nature of the bone in most maxillary applications, precise drilling is essential. Drill speeds should be kept below 1000 rpm and must remain monoaxial, thus ensuring no subsequent adjacent bone necrosis (and later screw loosening) or enlargement of the drill hole.[50,51] Monoaxial drilling is crucial, as any change in the drilling angle will result in an inadequate (and oval) hole in the bone. Enlarged screw holes are the primary reason for stripped screws, a common occurrence in the thin bone of the face. If a screw has been stripped, emergency screws are available (for example, 2.3 mm or 2.4 mm to replace a stripped 2.0-mm screw).

Most screwdriver handles have a rotatory mechanism whereby the screw is turned with only the thumb and index finger and the base is stabilized by the palm of the hand. This ensures a more sensitive adjustment of the rotational force applied to the screw based on the resistance encountered during its placement into the bone. This method of insertion also helps avoid excessive screw tightening, which can result in screw fracture upon insertion (shearing the head) or microfractures within the bone hole, and subsequent screw loosening.

Conventional versus locking plates[52]

Conventional plating relies on the friction generated between the plate and bone. Accurate plate contouring is required to match the anatomic bone contour. In conventional plating, even if the bone fragments are correctly reduced prior to plate application, fracture dislocation can result if the plate does not exactly fit the bone, and as the screws are tightened a loss of reduction may occur (termed *loss of primary reduction*). Furthermore, under an axial load postoperatively, if shear forces are greater than the frictional forces between the plate and bone, loss of fixation may occur due to toggling of the screws ("secondary loss of reduction"). In cases with decreased bone quality, such as osteopenic bone, screws cannot be tightened sufficiently to obtain the compression needed to support the bone; this is also a concern in small dogs and cats in areas where bone thickness is limited. This may cause loosening of the screws and loss of stability, with ultimately a loss of the reduction. Additionally, because the plate is compressed to the bone surface, the periosteum is compressed under the plate area, reducing or even interrupting blood supply to the bone. The result can be delayed bone healing due to temporary osteoporosis underneath the plate. Despite these purported limitations, conventional plating achieves good results in good-quality bone and in fractures conventionally repaired with compression across the fracture surfaces, which achieves direct bone healing (see Chapter 2). Bicortical screw fixation is used with conventional plate fixation, as both cortices are required to achieve sufficient compression so as to both obtain construct stability and prevent pullout, thus maintaining friction between the plate and bone.

With locked plating, screws lock to the plate, forming a fixed-angle construct (a "screw-only" mode of force transfer). Once the locking screws engage the plate, no further tightening is possible. The implant locks the bone segments in their relative positions regardless of the accuracy of the plate contouring or degree of reduction obtained; therefore loss of primary reduction does not occur. Contouring of the plate minimizes the gap between the plate and the bone; however, an exact fit is not necessary for implant stability. By locking the screws to the plate, the axial force is transmitted over the length of the plate, decreasing the risk of a secondary loss of reduction. Finally, locking the screw into the plate does not generate additional compression at the plate-bone interface; therefore the periosteum will be protected if a gap is maintained between the plate and bone, and the blood supply to the bone is preserved. The fixed angle is 90 degrees to the plate surface, which must be drilled correctly; all systems have a specific drill guide that centers the drill bit within the plate hole and ensures the appropriate drill angle. A disadvantage of the fixed-angle construct is that, in most cases, this fixed angle is orthogonal to the surface of the plate; therefore the screw cannot be angled to either ensure bone purchase or avoid undesirable screw position. There are some manufacturers that produce a variable-angle locking screw in an attempt to overcome this issue; however, these designs do not have as secure a locking mechanism (particularly in the smaller implants), resulting in a weaker construct.[53] A fixed-angle construct provides advantages in osteopenic or thin bone or multifragmentary fractures, where traditional screw purchase may become compromised. By the nature of this fixation, plate application is performed without compression of the fracture surfaces, and only indirect bone healing is achieved (see Chapter 2).

Hybrid fixation, whereby both standard and locking screws are utilized, may be employed in specific cases. Standard screws are used to provide compression across a fracture surface (aiming for absolute stability of an anatomic reduction),[54] while locking screws are used to increase the strength of the fixation—primarily in areas with limited or poor bone purchase. Whenever standard screws are applied, accurate plate contouring is essntial. When applying hybrid fixation, standard screws must be placed first to press the plate to the bone—the mechanism required for construct stability. If locking screws are applied first, subsequent standard screws provide no function, as compression at the bone–plate interface is prevented by the presence of the locking screws. With standard screw application as the first step, it must be recognized that the vascularity at the bone–plate interface is no longer preserved; furthermore, subsequent application of locking screws has not been definitively demonstrated to increase the strength of the fixation, unless there is a weakness in the bone purchase (short fragment, thin cortex, osteopenia).[52]

Miniplate design

Miniplates are generally considered to be 2.0-mm plates or smaller. The nomenclature can be confusing, as this refers to the screw size used in many cases. These plates are generally ≲6 mm wide and ≤ 2 mm thick. These are most often ~1.5 mm thick in a low-profile design and are either conventional or locking plates; however, most of the locking plate designs also can accept standard screws. Using standard screws will only permit compression to be applied across the fracture site if the screw-hole in the plate is specifically designed for this function; many locking plates only permit standard screws to be placed in a neutral position.

Titanium implants are the norm for maxillofacial use. The low elasticity (and high deformability) of titanium plates allows ease of adaptation to the contour of the bone in three dimensions (both in-plane and out-of-plane bends and torsional bends).[51] Additionally, the design conformation in the area of the screw holes within the plate, and the connecting bridges between the screw holes, are such that, when bending, the deformation occurs primarily in the region of the connecting bridge without deformation of the screw holes. The screw hole and the screw head also fit together in such a way that the overall low-profile design is maintained.[55]

Plate benders used to contour the plates to the shape of the bone are specific for each miniplate design. Most bending pliers have a design to specifically enable in-plane bends while preserving the conformation of the plate holes.[50,51,55,56] Other instrumentation is similarly designed to protect the holes. Many manufacturers provide screw inserts that are placed in the plate holes during the bending process to preserve the plate hole shape. The latter are, however, limited to the locking plate design (with threads) to secure these inserts.

Mesh implants

Mesh implants are specifically designed for reconstruction of the bony orbit in humans and to support the placement of grafts. Because of their use in the orbit, these mesh implants are manufactured in the lower-profile 1.5-mm system. Their use in veterinary medicine may be limited, as in the dog and cat only the medial wall and part of the roof of the orbit are osseous; the lateral wall and floor are soft tissue. Furthermore, the eye in the dog and cat is supported by two fascial structures: the bulbar sheath and the periorbita, which provides most external support.[57] Mesh implants may, however, have some utility in other areas of the skull and as support for cancellous grafts. The 0.4-mm and 0.6-mm rigid titanium mesh with low-profile neuro screws (Low Profile Neuro Plating System; DePuy Synthes CMF, West Chester, PA) is available either as 100 × 100 mm or 200 × 200 mm squares; the mesh is cuttable and contourable. Screws can be placed from either side of the mesh, are 0.6 mm in diameter, are 3–6 mm in length, and are self-drilling, permitting a one-step insertion.

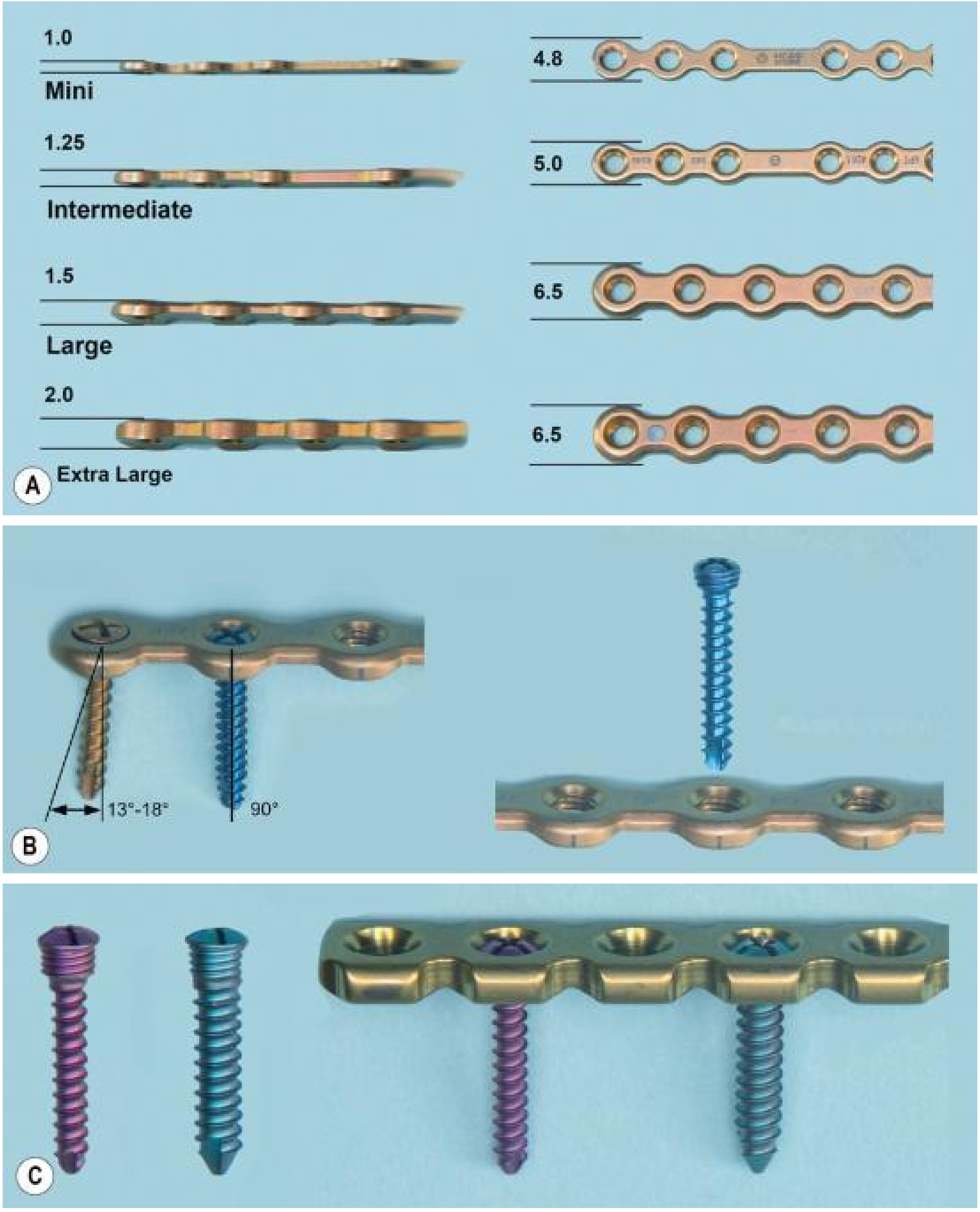
Veterinary plating systems

In veterinary medicine, there are no dedicated maxillofacial implants similar to those described in humans. However, many of the human maxillofacial implants have been used in animals, e.g., DePuy Synthes Maxillofacial, and now also available with DePuy Synthes Vet. There are a number of veterinary manufacturers for smaller implants; while they are not specific to maxillofacial use, appropriate sizing allows their application for maxillary and mandibular fracture repair. A number of manufacturers produce similar implants for maxillofacial use, but two of the most commonly used systems, DePuy Synthes Maxillofacial System (DePuy Synthes Vet, West Chester, PA) and Advanced Locking Plate System (ALPS) (Kyon Veterinary Surgical Products, Boston, MA), will be used to illustrate the principles of their use. It should be noted that different implant systems with alternate designs are not directly equivalent in their construct stiffness and strength; thus their usage needs to be appropriately tailored to the specific implant systems and they should not be used interchangeably.[58,59]

Depuy synthes maxillofacial system [56,60]

These titanium miniplates are offered in various sizes (1.0-mm–2.4-mm systems; these sizes all refer to the screw size used in the plate). The 2.0-mm miniplates are available as either standard or locking plates. The 2.0-mm standard miniplates are 0.85 mm thick, and the straight plates are either 19.3 cm (30 hole) or 10 cm (20 hole), the former with 6.5-mm spacing between the holes and the latter with 5-mm spacing between the holes. These plates are cut to the length desired. Specially designed plate cutters (cutting irons) cut the implant without leaving a bur along the cut edge of the plate. The standard screws have a 1.4-mm core diameter and a 2.0-mm thread diameter, with a 0.6-mm thread pitch. A 2.4-mm screw also is available as an emergency screw. A bone tap is available for thicker bone (>3 mm), where self-tapping may prove to be difficult. The screw head and plate design allow screw placement up to a 20-degree angle relative to the plate surface. True hybrid fixation using compression across the fracture site, to obtain absolute stability, is not possible with these plates due to the round hole plate design.

A 2.0-mm Locking Mandible System also is available. In this system, plates are available in variable thicknesses and widths (Fig. 33.3). Threaded plate holes are present within the plate. The locking screw has double-lead threads beneath the screw head that engage and lock into the threaded plate holes. The screws are available in 5–18-mm lengths, with a thread pitch of 0.75 mm. The locking screws must be centered within the plate hole and inserted perpendicular to the plate surface; locking drill guides are used to ensure that this technique can be properly applied (Fig. 33.4). These plates will also accept standard screws, which may be angled relative to the plate surface. True hybrid fixation is not possible with this plate's round hole design.

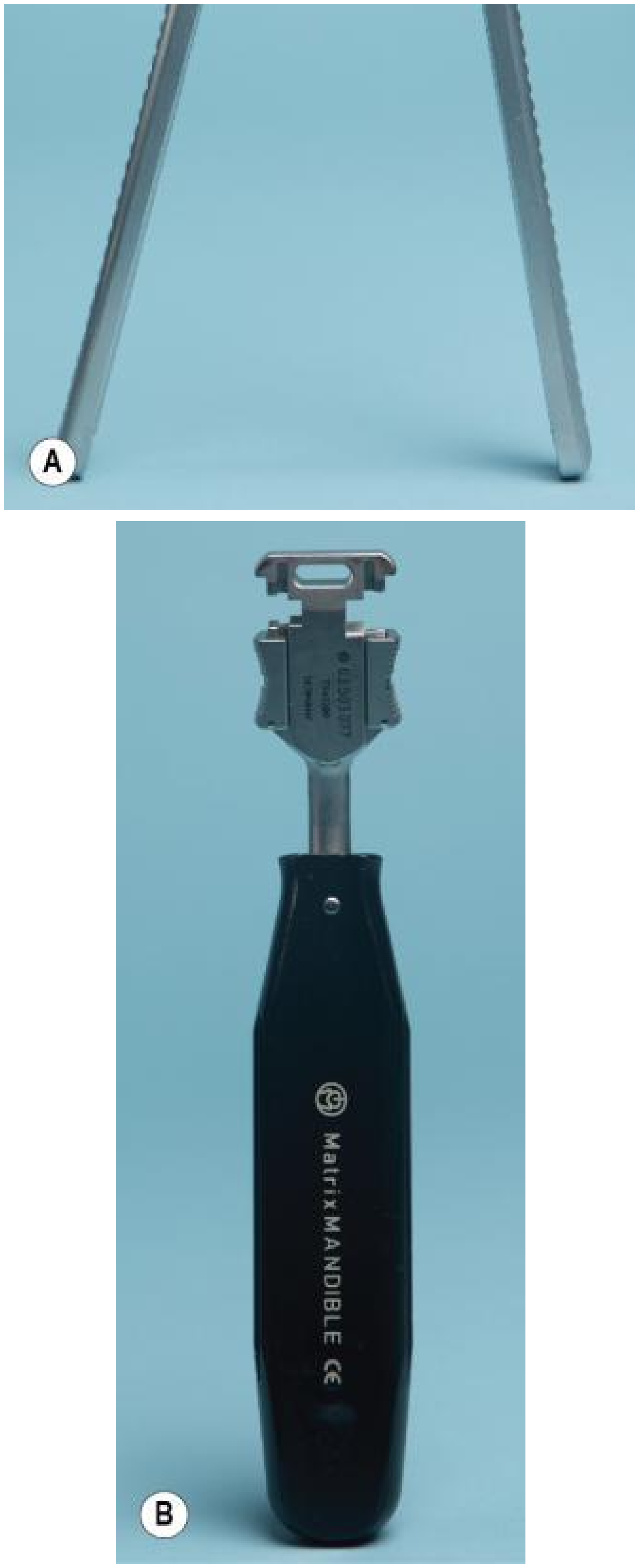


• FIG. 33.3 DePuy Synthes Vet locking plates, which can all be contoured three-dimensionally. **(A)** 2.0-mm miniplates of varying thicknesses, which all use 2.0-mm standard or locking screws. **(B)** A standard or locking screw can be used within the threaded hole of the plate. The standard screw may be angled 13–18 degrees; however, the locking screw must be placed orthogonal to the plate. **(C)** The 2.4-mm locking reconstruction plate; both 2.4-mm and 3.0-mm locking screws can be used.

• **FIG. 33.4** Instrumentation to apply the screws in the 2.0- and 2.4-mm plates. The standard universal drill guide with 1.5-mm and 1.8-mm drill sleeve diameters; the core diameter of the 2.0 mm-screw corresponds to a 1.5-mm drill bit, and the 2.4-mm screw corresponds to the 1.8-mm drill bit. The core diameter of the 2.0 locking screw also corresponds to a 1.5-mm drill bit; locking screw guide screws into the plate, which has threads within the screw hole, in order to center the drill hole within the plate screw hole, and in an orthogonal orientation. Similarly, the 2.4- and 3.0-mm screws have corresponding drill guides with 1.8-mm and 2.4-mm drill guides, respectively. Note that the screws and their respective drill guides are color coordinated.

Specially designed plate bending pliers are used to contour the plate three-dimensionally (Fig. 33.5). Two pliers or bending irons are used together to contour the plate. Each pair of pliers is designed to ensure preservation of the shape of the plate hole and prevent distortion of the plate during bending. The bending irons are also designed to preserve the hole geometry. All in-plane bends must be performed before any out-of-plane bends; if the reverse order is used, the plate will no longer fit within the bending pliers to allow the in-plane bends to be made (Fig. 33.6).


OUT-OF
PLANE
BEND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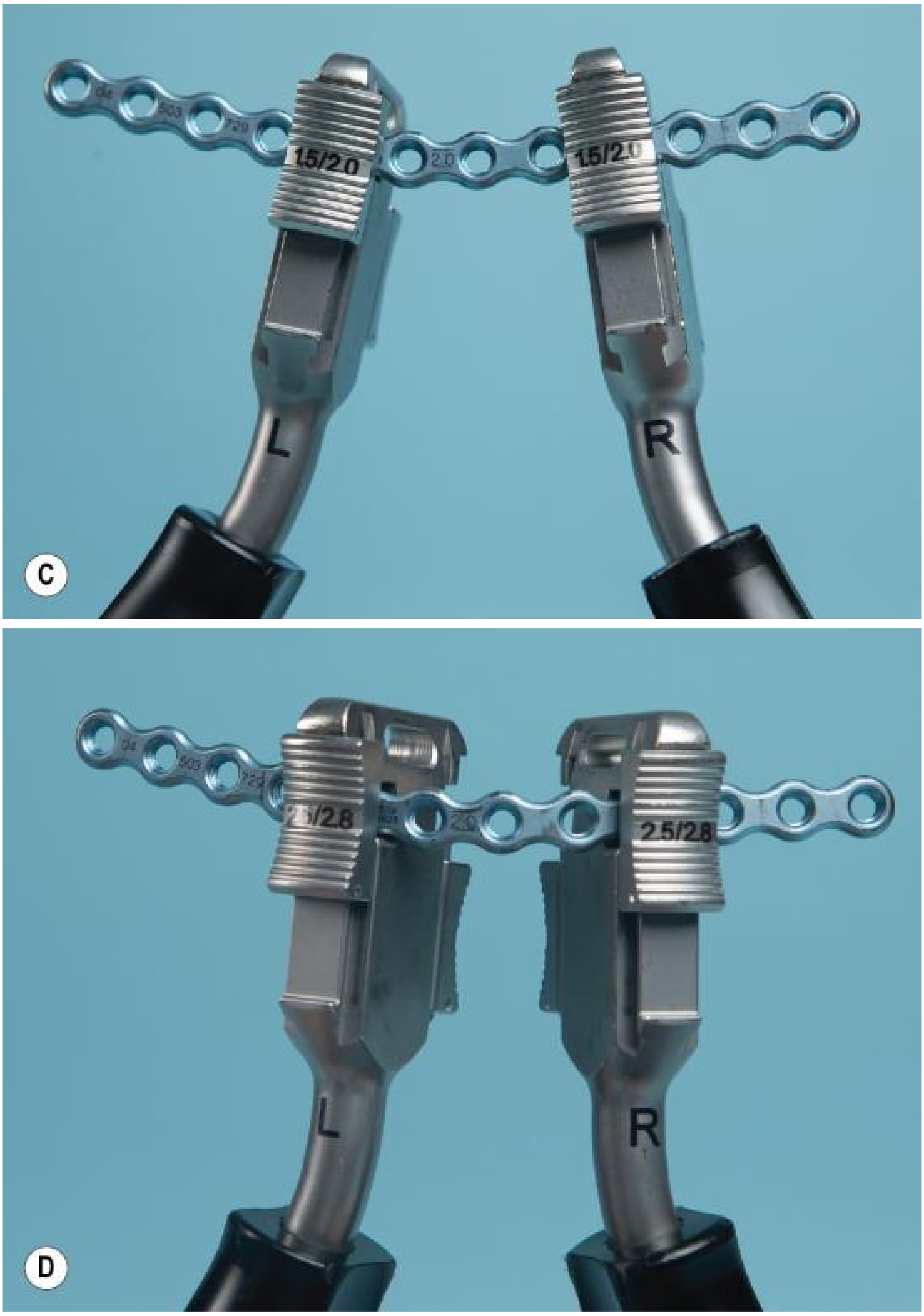
MatrixMANDIBLE
B


1.5/2.0
1.5/2.0
L
R
C
2.5/2.8
L
R
D

• **FIG. 33.5** **(A)** Bending pliers and **(B)** bending irons, both of which are specific to the plate sizes of the 2.0-mm and 2.4-mm plating systems. Two bending irons are illustrated performing **(C)** in-plane, **(D)** out-of-plane, and **(E)** torsional bend.


FIRST
STEP
IN-PLANE BEND

• **FIG. 33.6** **(A)** Bending pliers used to perform an in-plane bend of the 2.0-mm plate. **(B)** The opposite surface of the pliers used to perform the out-of-plane bends. Note that the in-plane bend is performed first.

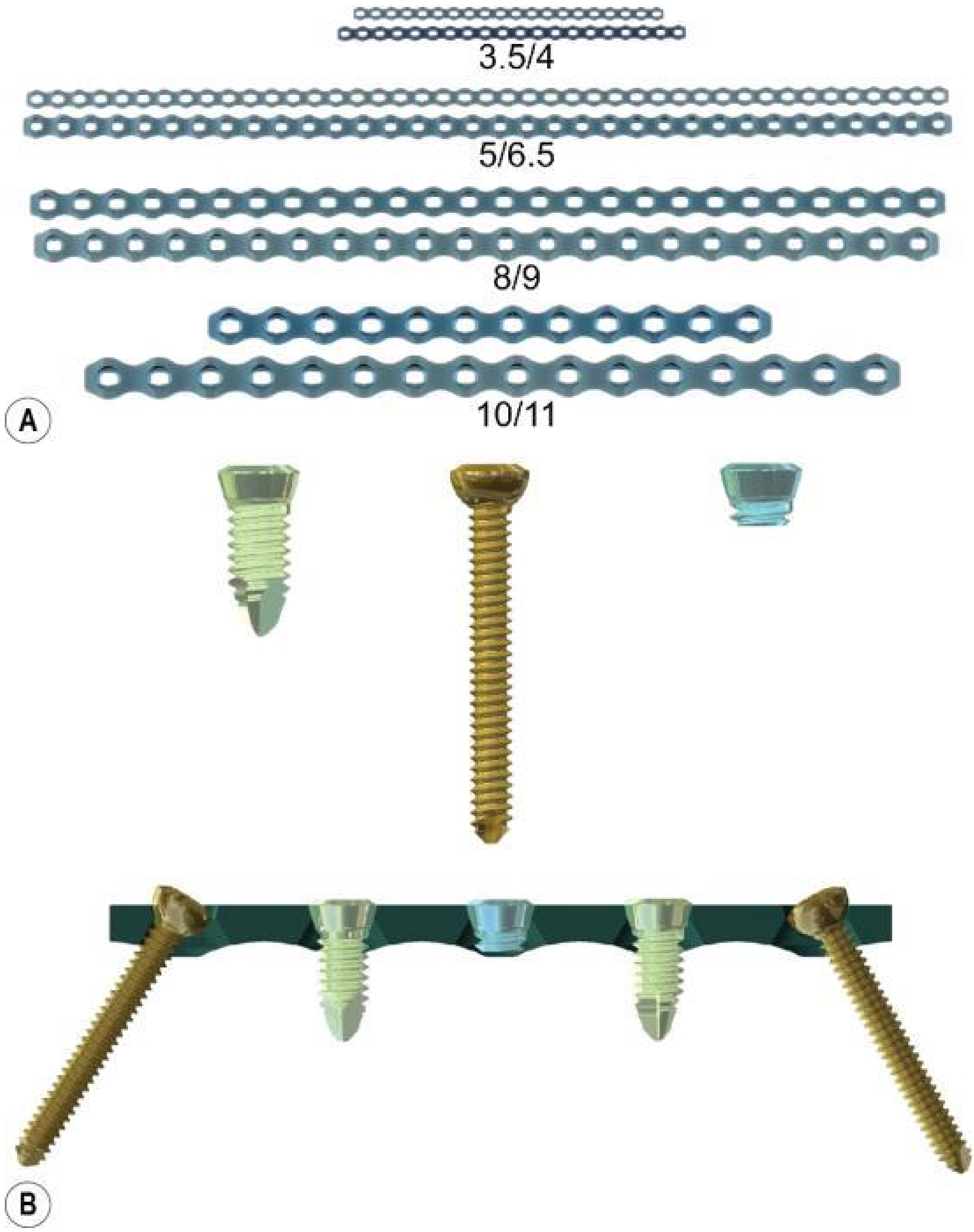
The 2.4 UniLOCK system is a locking reconstruction plate of a low-profile design with the ability to make in-plane bends, which provides fixed-angle stability (Fig. 33.3). Standard 2.4-mm screws (1.8-mm drill bit) or locking screws, either 2.4 mm or 3.0 mm (1.8-mm or 2.4-mm drill bit, respectively), can be used with these plates. These locking screws also have the double-lead thread beneath the screw head that quickly engages and locks into the threaded holes of the plate. Plates can be cut to the appropriate lengths, and because of the reconstruction plate configuration, in-plane bends can be performed, allowing the plate to be contoured three-dimensionally. All bending must occur between the screw holes, or the shape of the hole may become altered, and the screw head will no longer fit in the hole; screw inserts are needed to preserve this shape during contouring. Once again, true hybrid fixation is not possible with this plate's round hole design.

These plates have been used in veterinary mandibular fracture repair and mandibular reconstruction similar to that described in humans. They have been used as an alternative stabilization plate in larger dogs together with an alveolar miniplate; additionally, they have been used as a single implant with mandibular reconstruction.[61,62]

Kyon advanced locking plate system (ALPS)[36,63]

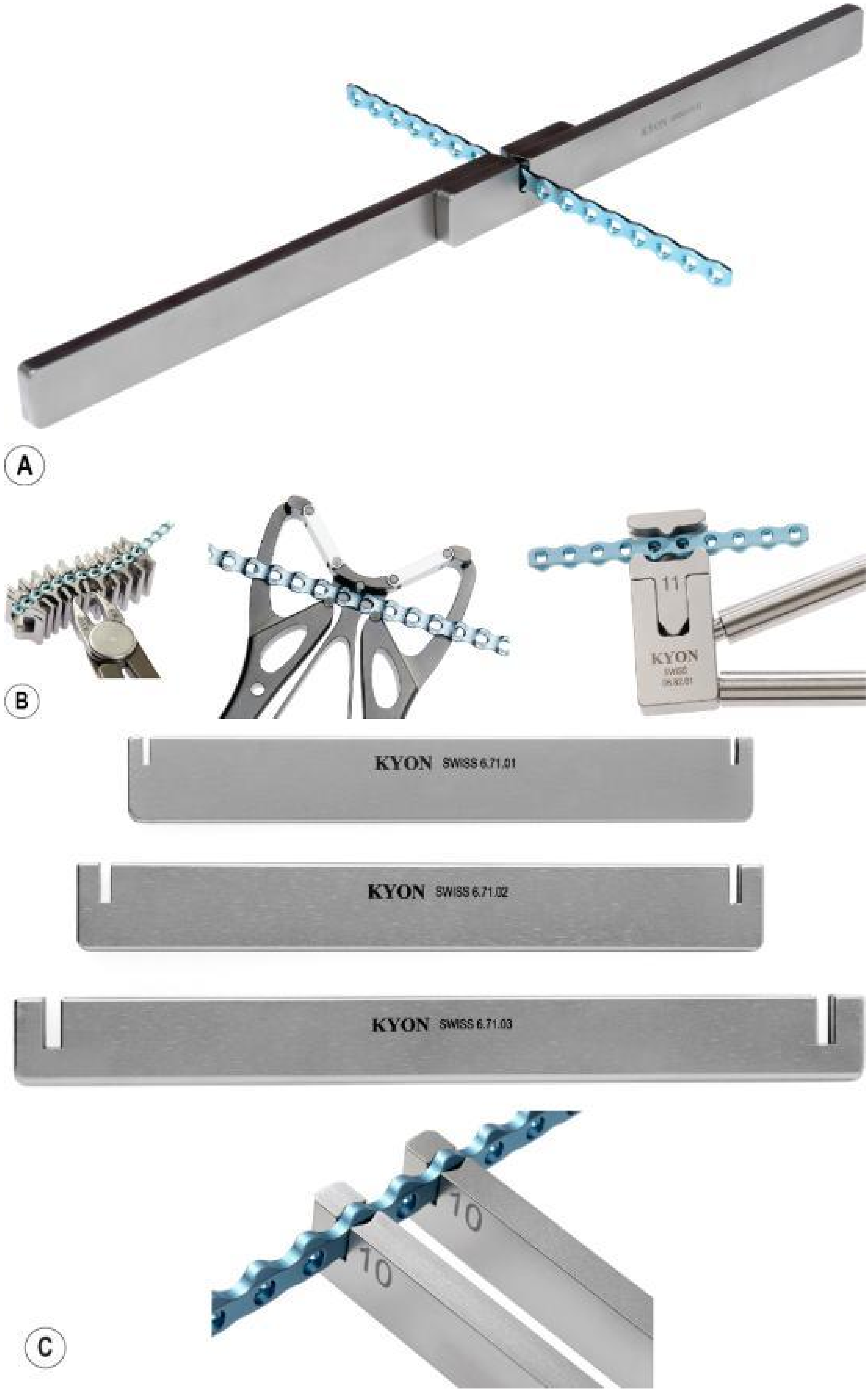
The ALPS is a veterinary implant system designed for general orthopedic trauma. It is a point contact titanium locking plate system, which has a plate configuration that allows in-plane bends to enable 3D contouring. In addition, the screw holes within the plate also allow not only standard screw application (the screw head and plate design allows screw placement up to a 30-degree angle relative to the plate surface) but also the ability to load a screw in order to apply compression across the fracture surface depending on screw placement within the plate hole; as such, absolute stability can therefore be obtained with true hybrid fixation. Locking screw application is monocortical. Experimentally, this plating concept, which preserves a space at the bone–plate interface and limits drilling/screw placement to a single cortex, has been demonstrated to have less vascular compromise and improved bone healing compared to conventional plate fixation.[64,65]

The ALPS plate sizes refer to the width of the plate (in millimeters); for maxillofacial indications, the ALPS 5 and ALPS 6.5 are most often utilized. The screw size is predicated by the plate width: both standard and locking screws (e.g., 1.5-mm standard and 2.4-mm locking screws in the ALPS 5 and 6.5) (Fig. 33.7).

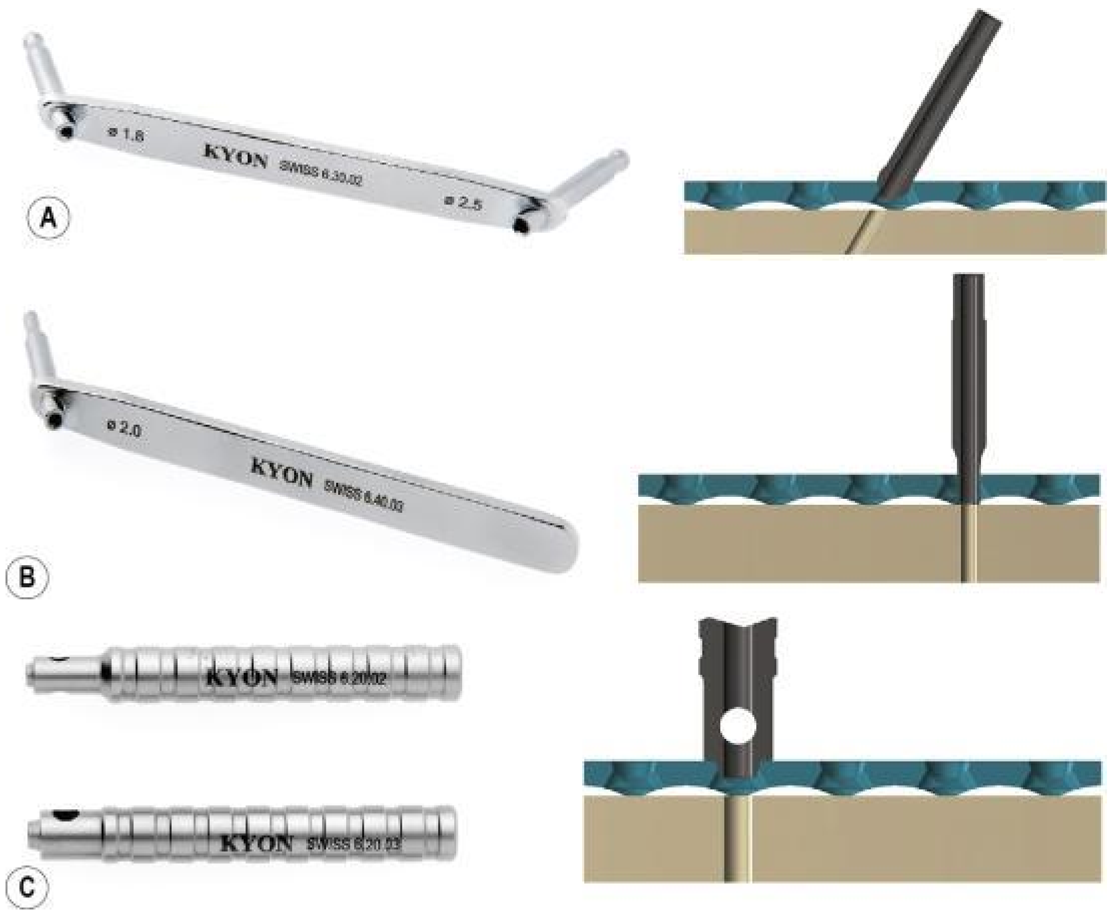


• FIG. 33.7 Kyon Advanced Locking Plate System plates, which can all be contoured three-dimensionally. These point contact plates need to be contoured to match the bone surface and compressed to the bone surface with at least one standard screw in each bone fragment (providing additional torsional stability). **(A)** Various plate sizing (based on plate width); grouping shows the plates with the same screw application. **(B)** A standard or locking screw can be used within the threaded hole of the plate; however, the screw-hole design is oval to permit dynamic compression in order to compress the fracture site in reconstructible fractures. The standard screws may be angled, but the locking screws must be placed orthogonal to the plate. Source: (Courtesy of Kyon Veterinary Surgical Products, Boston, MA, used with permission.)

The concept of plate application is to accurately contour the plate to adapt to the bone surface, thus ensuring point contact. This increases the torsional rigidity of the construct while simultaneously maintaining space for the vascular supply at the bone–plate interface. All plate contouring is performed using specific instrumentation for the plate sizes, which performs the in-plane bends with a four-bar linkage to ensure a smooth bend that does not deform the plate holes (inserts are not required to preserve plate hole conformation, but are available); similarly, the bending irons for the out-of-plane bends grasp the plate at the plate holes, ensuring that the bends occur between holes (Fig. 33.8). Once the plate is contoured, the first two (or more) standard screws are placed on either side of the fracture to secure the plate in the appropriate position against the bone surface. In addition, because of the screw-hole design, standard screw application may also be used to compress the bone fragments at the fracture site to obtain absolute stability in reconstructible fractures (true hybrid fixation) (Fig. 33.9).



• **FIG. 33.8** Kyon Advanced Locking Plate System instrumentation. **(A)** Cutting irons are designed to shear the plates when placed opposite with aligning the shearing edges at the gap between screw holes. **(B)** In-plane bending pliers are designed to preserve plate hole geometry without placing screw inserts; the device used is based on plate size. **(C)** Out-of-plane and torsional bends are performed with bending irons specific to each plate size (each bender can be applied to each plate grouping, i.e., 5/6.5, 8/9, and 10/11, respectively; each end of the bender is marked with the appropriate plate size—see bottom figure); the irons are secured directly over the plate-hole in order to preserve the hole geometry during bending. Source: (Courtesy of Kyon Veterinary Surgical Products, Boston, MA, used with permission.)



• **FIG. 33.9** Instrumentation to apply the screws are all specific to plate sizes. **(A)** Standard drill guide that fits within the center portion of the plate hole; the bulbous end maintains centering when the guide is angled in order to ensure appropriate drilling within the hole (5 degrees transversely, 60 degrees longitudinally). **(B)** The “load” guide (to provide compression/absolute stability at the fracture site in reconstructible fractures) has a tapered end that corresponds to the screw core size, which allows the guide to be placed eccentrically within the oval hole in the plate; screw tightening achieving compression at the fracture site. This system can be used alone as standard fixation but also can be combined with locking screws as hybrid fixation. **(C)** Locking guide, which sits on the surface of the plate (wide flange) to ensure orthogonal drill placement in the center of the hole to engage the threads. Source: (Courtesy of Kyon Veterinary Surgical Products, Boston, MA, used with permission.)

Therapeutic decision-making

Surgical approaches

The specific surgical approaches are discussed in Chapter 29.

Sequence of repair

Simple (reconstructible) fractures are reconstructed anatomically; however, comminuted fractures or fractures with gaps (nonreconstructible fractures) must be bridged using occlusion to determine the accuracy of the reduction, an indication for temporary MMF. In reconstructible fractures, plate fixation of the accessible bone surface with "perfect" reduction (e.g., ventral margin of the buccal mandibular surface) does not always result in a similarly perfect reduction along the lingual or alveolar surface of the bone. Reduction opposite the fracture repair will not always follow, since compression directly under the plate may cause distraction of the opposite cortex and requires "prestressing" of the plate directly over the fracture site.

Reconstruction of any fracture should start with the more straightforward (simple) fracture first and build toward the more difficult (comminuted) fractures (see Chapter 27). In cases of both maxillary and mandibular trauma, the mandible is usually repaired first in both dog and cat, providing a base on which to build the maxillary repair. For the maxilla, the lateral buttresses are repaired first in the dog and cat, followed by the medial buttresses. The caudal buttress need not be addressed, as reconstruction of two of the three buttresses ensures that the remaining buttress will be aligned.

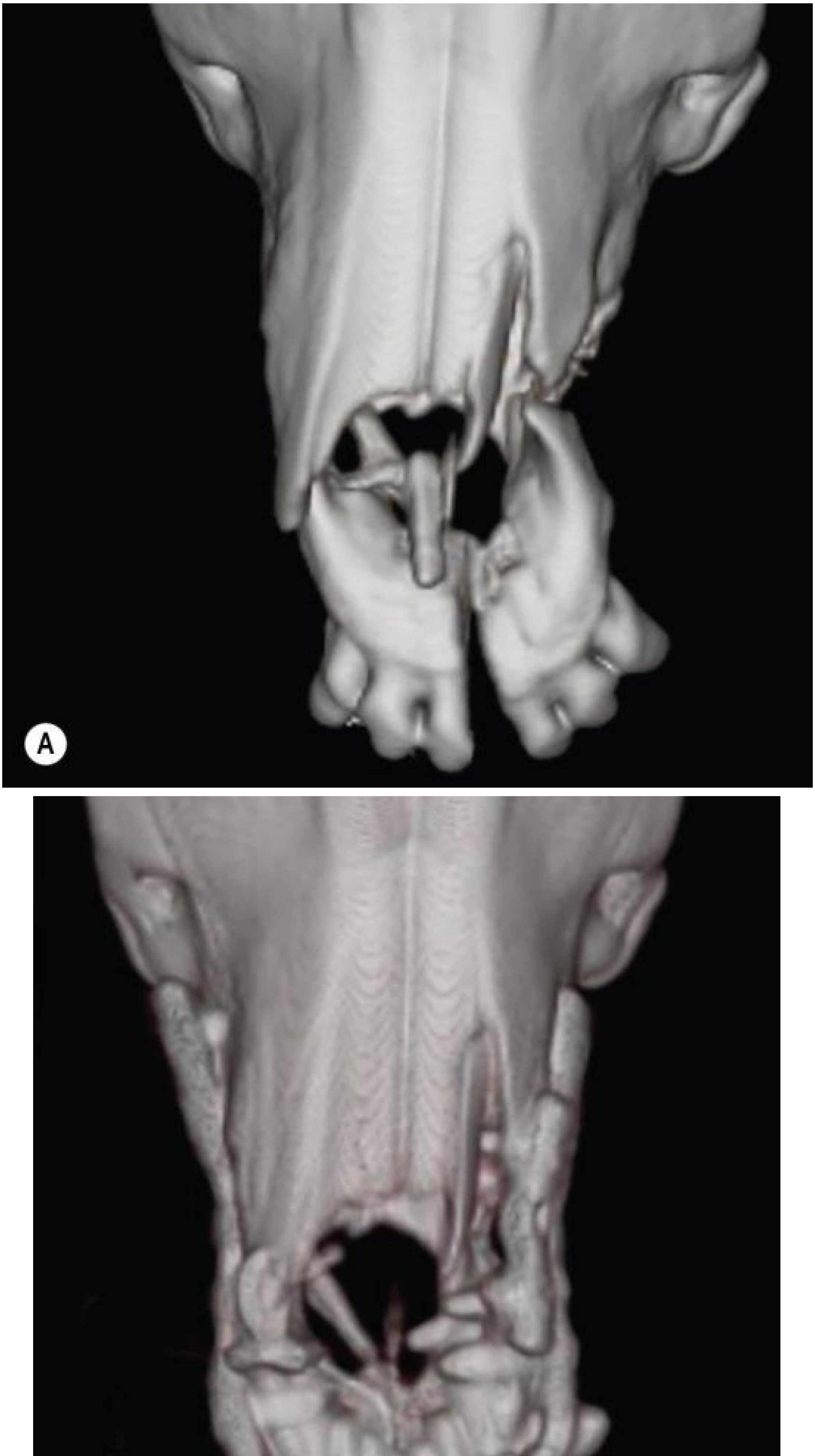
Accurate plate contouring is paramount regardless of the fixation system used. All plates must be accurately bent into the appropriate shape to passively fit the contours of the bone. If the plate is not accurately bent, the underlying bone will be pulled toward the plate when applying standard screws, causing a corresponding shift at the occlusal level. A malocclusion, in addition to adversely affecting function, also may result in fixation failure as abnormal leverage is exerted against the fixation devices. The latter contributes to motion at the fracture site and subsequent loosening of the fixation.

Any concern with regard to the neurovascular bundle within the mandibular canal is not warranted in comminuted fractures of the angle and the body of the mandible, as the fracture has compromised this structure. The vascular supply of the rostral mandible will temporarily be enhanced, as temporary intraosseous circulation from the adjacent soft tissues revascularizes the area; the alveolar circulation will be reconstituted within a few weeks (see Chapter 2). The inferior alveolar nerve, however, is unlikely to recover.

Surgical techniques

Fractures involving the incisive and maxillary bones

Incisive and maxillary bone fractures may or may not interfere with the structural support of the face, but they typically result in an alteration of the occlusion. This is best illustrated where a fracture line traverses the dental arch and results in a step along the occlusal surface. Restoring the line of dentition may be accomplished via miniplate fixation using an intraoral approach—reflecting the alveolar mucosa away from its attachments to the alveolar bone adjacent to the base of the teeth (Fig. 33.10).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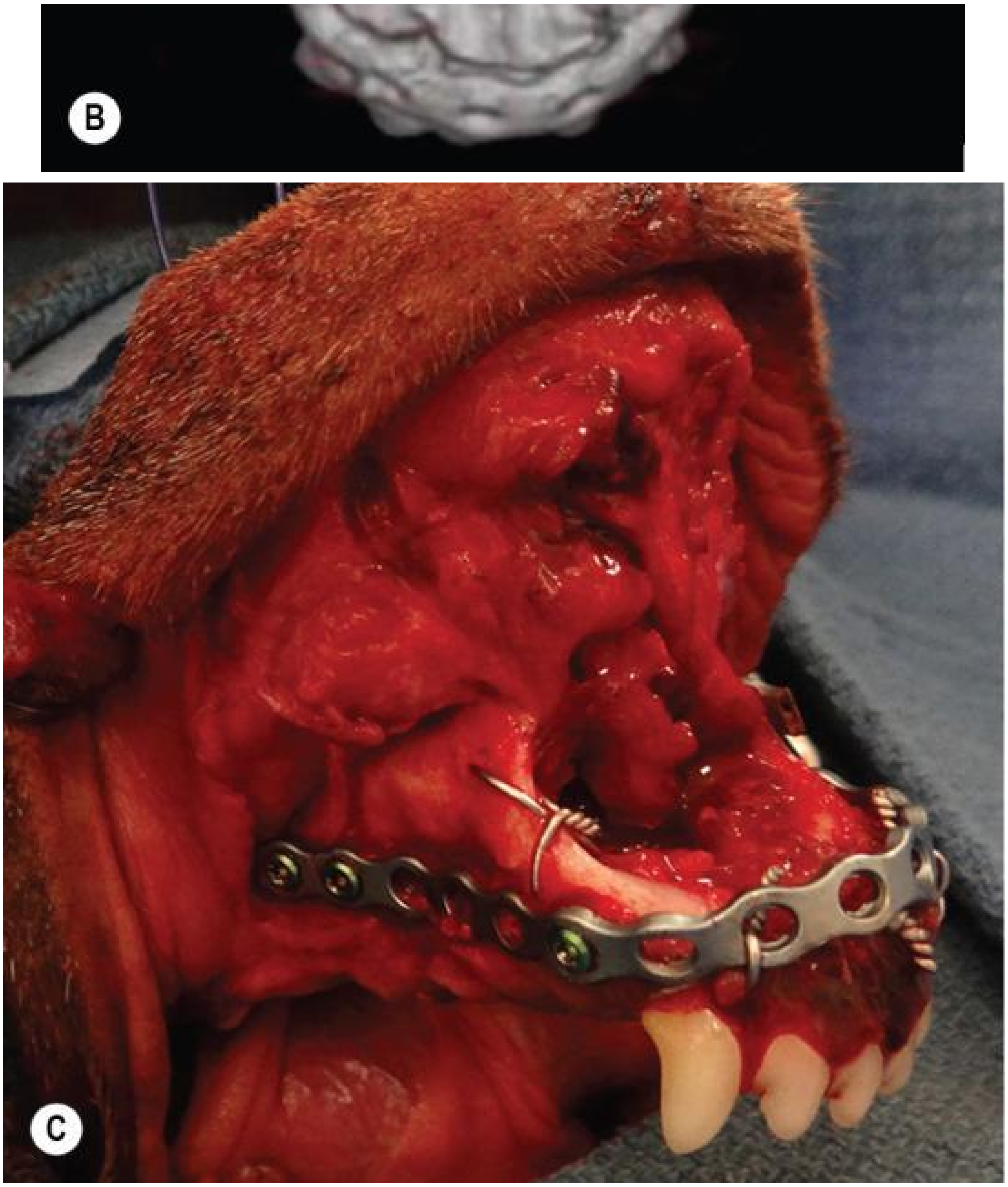


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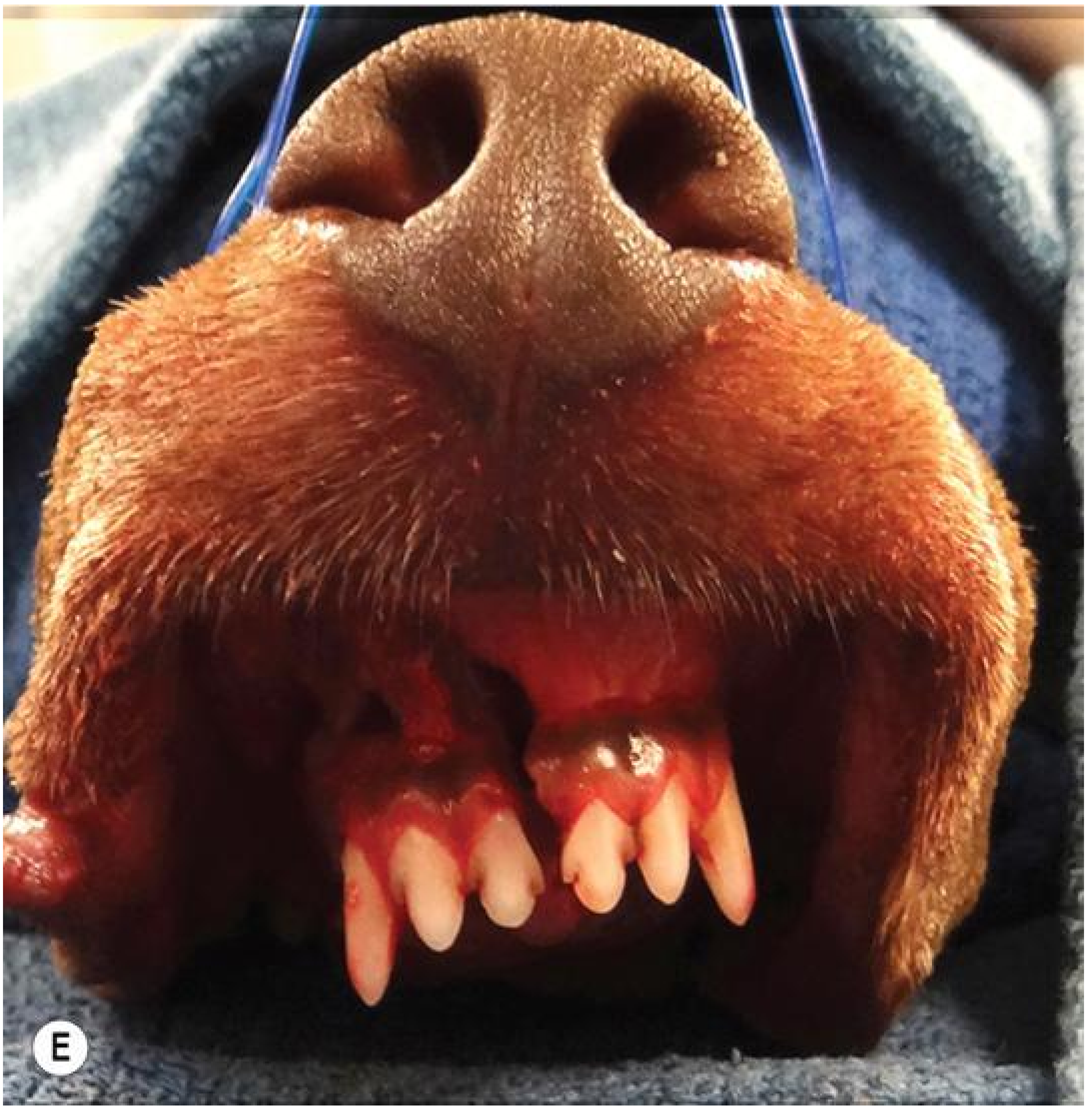
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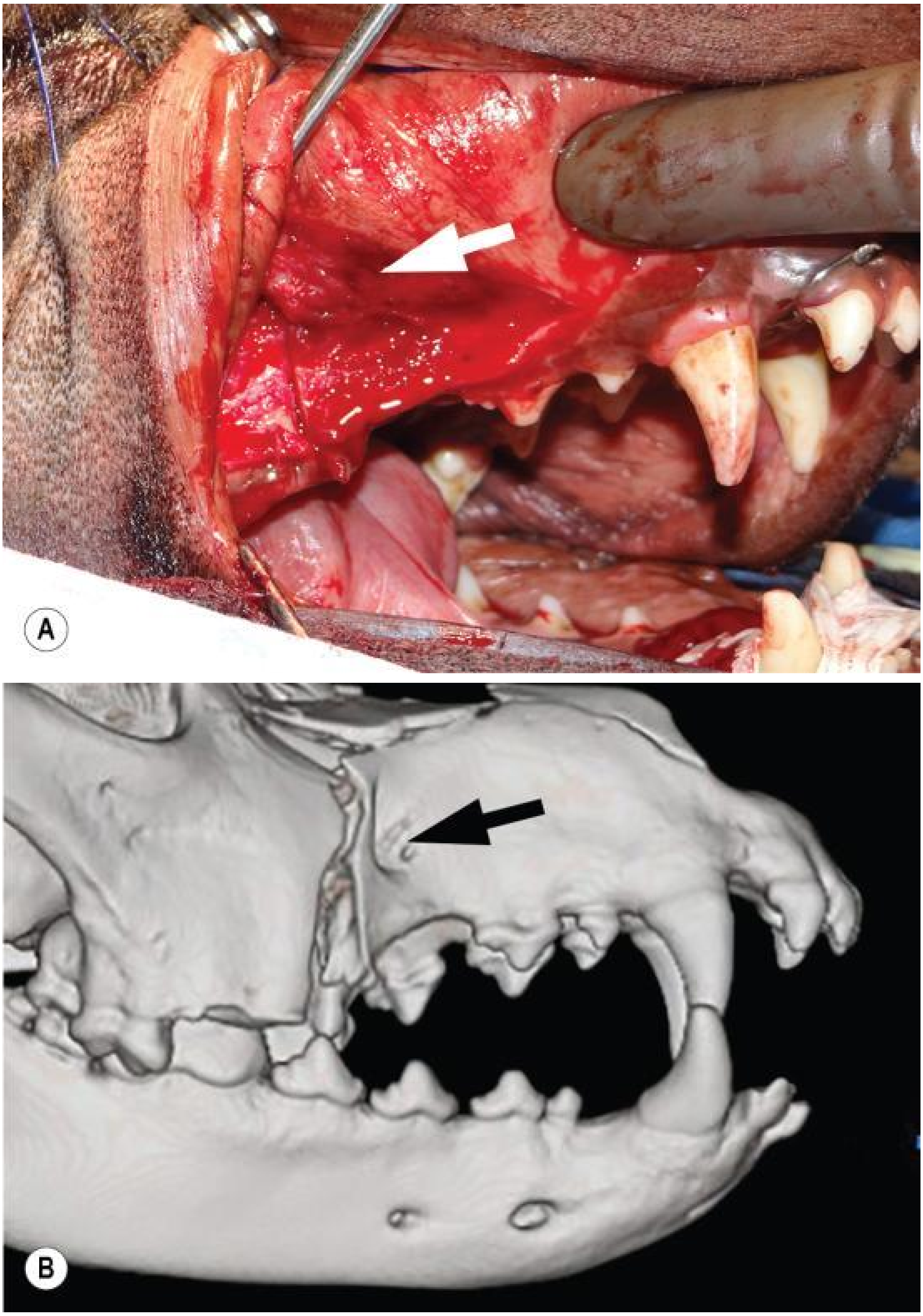
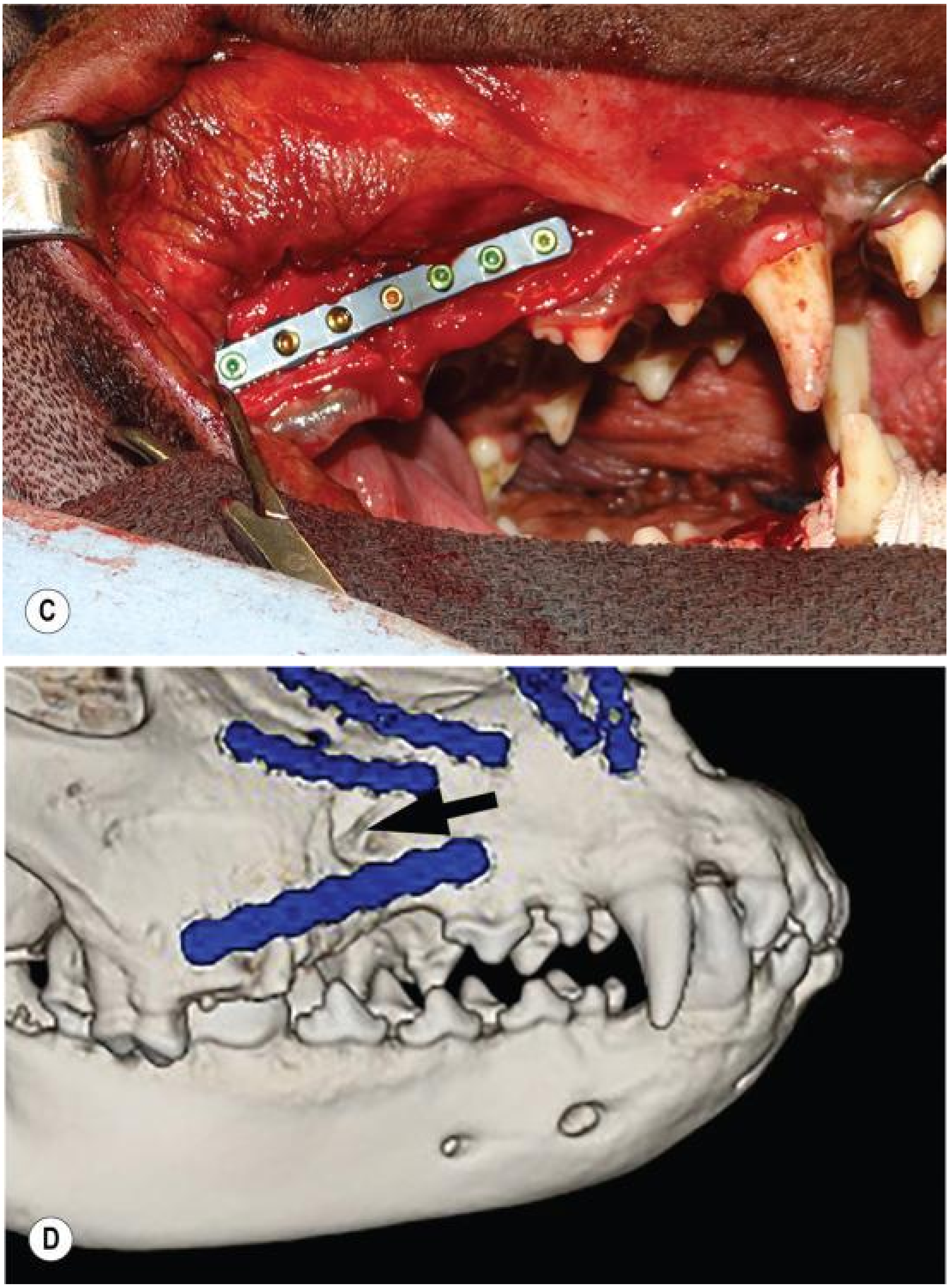
• **FIG. 33.10** Nine-year-old dachshund with maxillofacial trauma due to bite wounds. **(A)** Preoperative and **(B)** postoperative dorsoventral three-dimensional (3D) computed tomography (CT) image, demonstrating the comminuted fractures of the incisive, nasal and rostral maxillary bones, and the bridging fixation with a single Advanced Locking Plate System 5 plate from the maxilla on both sides and around the rostral extent of the incisive bones ventral to the nasal cartilages, with supplemental cerclage wire to secure the plate rostrally. **(C)** Intraoperative photograph and **(D)** corresponding postoperative 3D CT demonstrating the bridging fixation placed. **(E)** Preoperative photograph of the fracture and **(F)** postoperative photograph of occlusion.

Miniplate location for repair of these fractures is typically near the alveolar margin to ensure alignment of the dental arch, with the plate location below the infraorbital foramen of the maxilla laterally; similarly, the plate is kept below the nasal cartilages and applied directly to the incisive bone rostrally (Fig. 33.11). Screws are placed so as to avoid the tooth roots. Alternatively, the plates may be placed further away from the alveolar margin and dorsal to the infraorbital foramen on both sides of the maxilla to avoid the tooth roots; however, a separate plate—or alternate fixation—may be required rostrally. Closure of the alveolar mucosa is not impaired by miniplates due to their low-profile design.

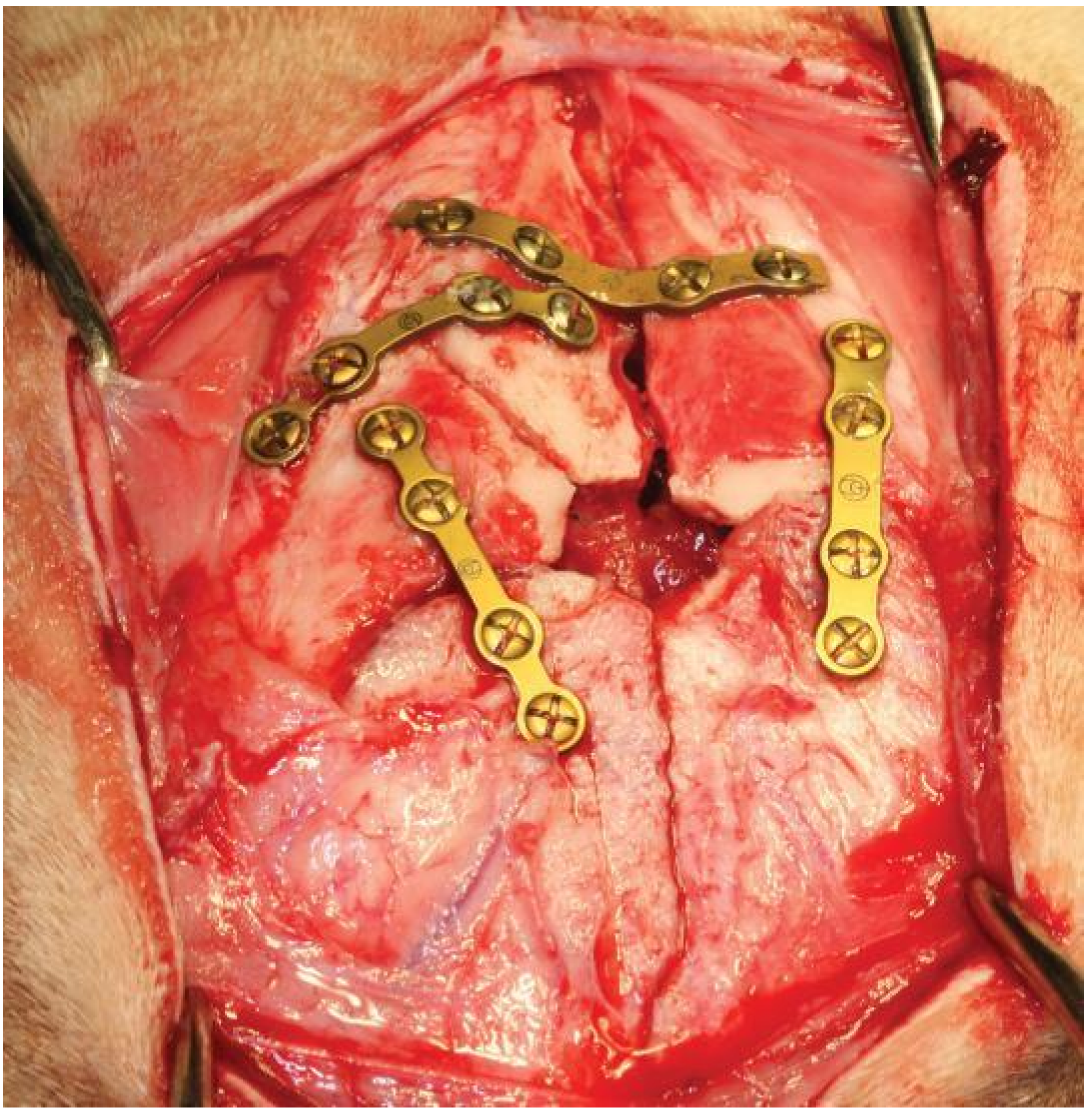




• FIG. 33.11 Four-year-old Labrador retriever that was hit by a car. **(A)** Lateral buttresses. Intraoperative photograph of the maxillary fracture and **(B)** preoperative three-dimensional (3D) computed tomography (CT) demonstrating a vertical fracture through the alveolar margin caudal to the right maxillary third premolar tooth; note the infraorbital foramen *(arrow)*. **(C)** Intraoperative photograph and **(D)** 13-month postoperative 3D CT, **(D)** after placement of Advanced Locking Plate System 6.5 plate parallel to the alveolar margin and below the infraorbital foramen *(arrow)* bridging the fracture site. Note, in **(C)**, the combination of standard screws (gold), which were angled in order to avoid the tooth roots, and locking screws (green). Source: (From Illukka E, Boudrieau RJ. Surgical repair of a severely comminuted maxillary fracture in a dog with a titanium locking plate system. *Vet Comp Orthop Traumatol*. 2014;27:398–404; reprinted with permission.)

Fractures involving the nasal and frontal bones

Although fractures of the nasal and frontal bones rarely compromise facial support, depressed areas will cause a cosmetic defect and may also alter nasal airflow, and miniplate fixation is indicated. A dorsal midline approach, sufficient to expose the entire fracture, is made (see Chapter 29). The miniplates are used as bridging devices to span across the fractured area(s) while secured in intact bone. All small bone fragments also can be secured to these plates, effectively rebuilding the bony contour (Fig. 33.12).



• **FIG. 33.12** Intraoperative photograph of the dorsal aspect of the skull in an 8-month-old golden retriever with comminuted fractures of the frontal bones due to falling off a moving truck. The bone fragments have been elevated and fixed in their relative anatomic positions with four 2.0-mm nonlocking miniplates. Note that in the central area, comminuted small bone fragments were dislodged into the frontal sinus and were too small to be secured and were therefore removed. This central area was left unreconstructed and covered with the soft tissues only. In this area, the bone thickness is relatively robust compared to other areas of the skull, allowing excellent screw purchase. In addition, all forces in these areas of the skull at this level are fairly uniformly distributed, and plate positioning is guided by the ability to secure all bone fragments.

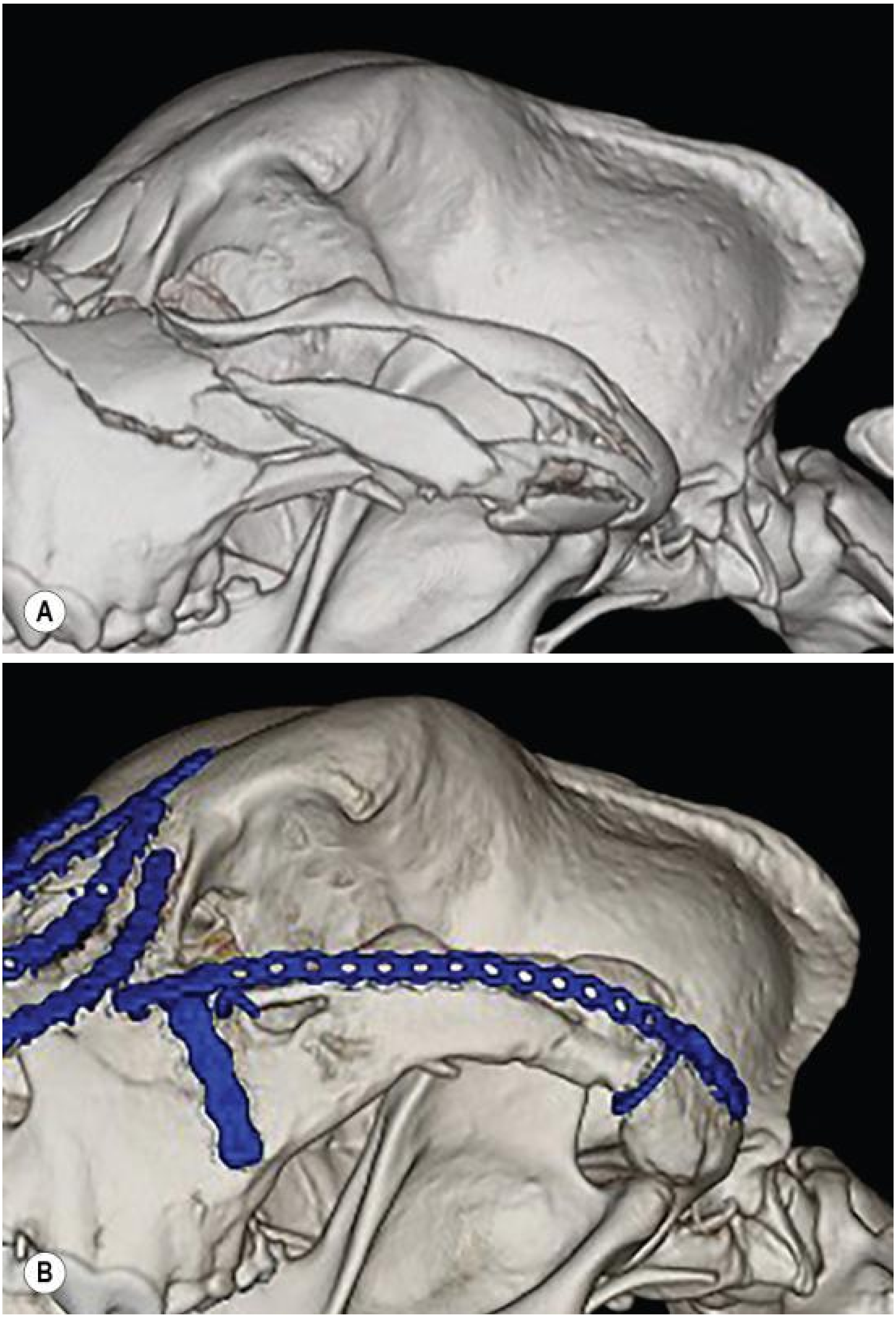
One area of functional importance of the frontal bone is the zygomatic process. This area is the dorsal attachment of the orbital ligament, which serves as the lateral attachment of the lateral palpebral ligament and orbicularis oculi muscle.[57] Loss of support of the zygomatic process of the frontal bone will result in a lack of lateral support of the eye (Fig. 33.13).


A

• **FIG. 33.13** Photographs of a middle-aged Labrador retriever. **(A)** Dorsolateral view of left orbit: comminuted zygomatic and frontal bone fracture (including the zygomatic process) postoperatively, and **(B)** months later, demonstrating contracture and scarring of the soft tissues around the lateral margin of the eye due to a lack of rigid support of the lateral canthal ligament *(arrows)*.

Fractures involving the zygomatic arch

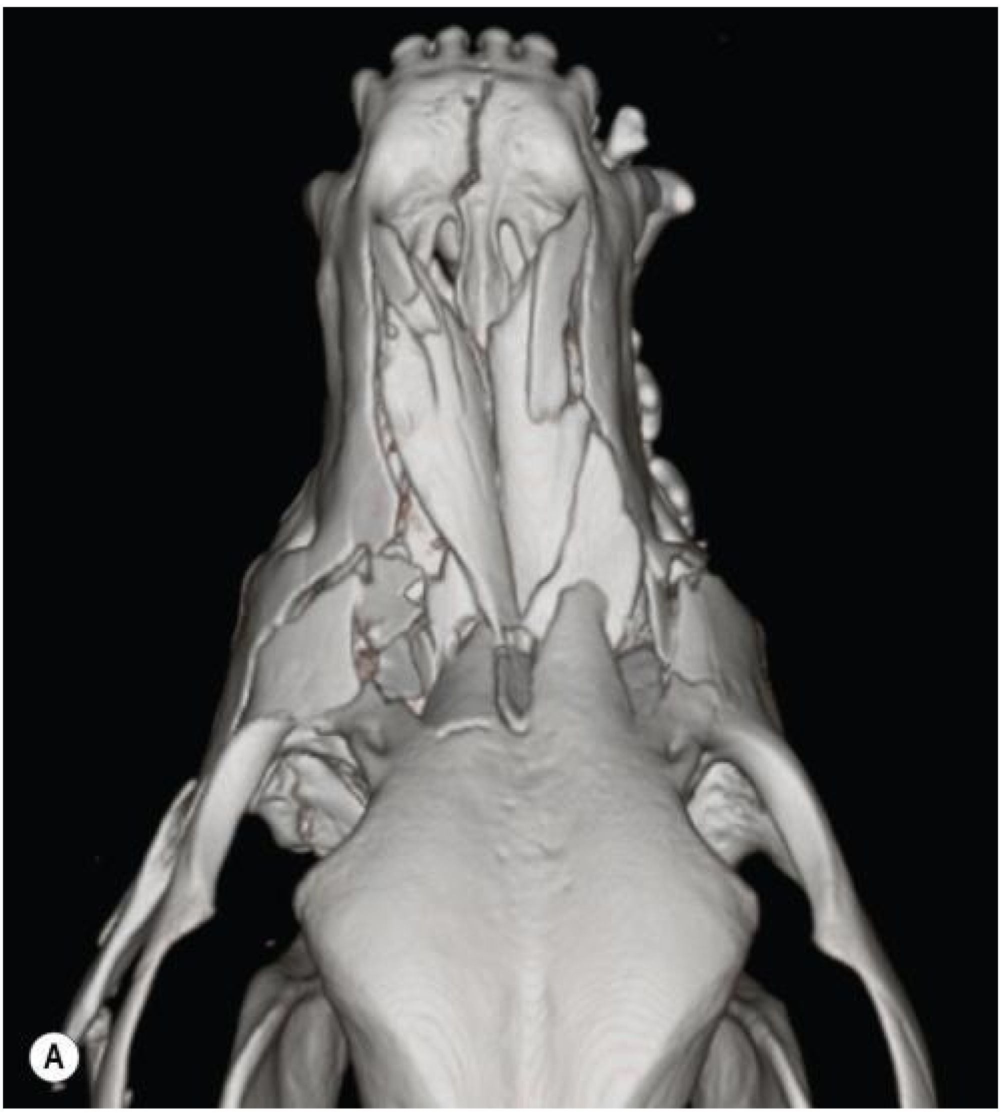
Fractures of the zygomatic bone may be associated with trauma to the eye and orbit. This fracture may result in exophthalmos if the bone fragments have been displaced medially or, alternatively, either a caudal or ventral displacement of the eye if the lateral support provided by this bone is lost. A direct approach to the zygomatic arch is performed. The arch is usually reconstructed with a single plate that spans the entire arch (Fig. 33.14). Caution must be exercised to protect the zygomatic branch of the facial nerve, which courses over the caudodorsal aspect of the zygomatic arch. Repair of fractures at the caudal aspect of the zygomatic arch must be approached with caution due to the proximity of the facial nerve and maxillary artery. It may be more prudent to follow a conservative approach for fractures at this location.



• **FIG. 33.14** Four-year-old Labrador retriever. **(A)** Preoperative three-dimensional (3D) computed tomography (CT) reconstruction of a comminuted left zygomatic arch fracture. **(B)** Follow-up (13-month) 3D CT reconstruction of the healed repair with an Advanced Locking Plate System 5 bridging plate. There was no attempt made to secure the multiple fragments; however, the soft tissues were reapposed around the plate with two sutures. The plate has three screws cranial and two screws caudal; the latter was supplemented with an additional cerclage wire to provide an additional point of fixation for the plate. Source: (From Illukka E, Boudrieau RJ. Surgical repair of a severely comminuted maxillary fracture in a dog with a titanium locking plate system. *Vet Comp Orthop Traumatol*. 2014;27:398–404; reprinted with permission.)

Fractures involving the maxillary (medial and lateral) buttresses

Fractures that cause a disconnect between the skull and the occlusal surfaces of the maxilla are associated with loss of the support of the facial frame, i.e., the medial, lateral, and caudal buttresses.[66] The caudal buttress need not be reconstructed directly, since reconstruction of the medial and lateral buttresses will accurately reposition the caudal buttress. Access to the medial and lateral buttresses can be obtained from a dorsal midline approach, with lateral soft tissue elevation to adequately expose the fractures. If necessary, the lateral buttress may be approached separately, either directly over the rostral aspect of the zygomatic bone or intraorally by elevating the alveolar mucosa dorsal to the fourth premolar tooth. Reconstruction should commence with the lateral buttress to reestablish continuity between the occlusal surfaces and the skull. Further reconstruction and fixation then proceed rostrally (Fig. 33.15).[66]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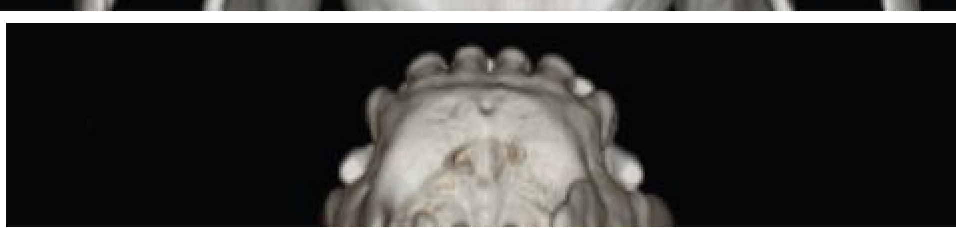

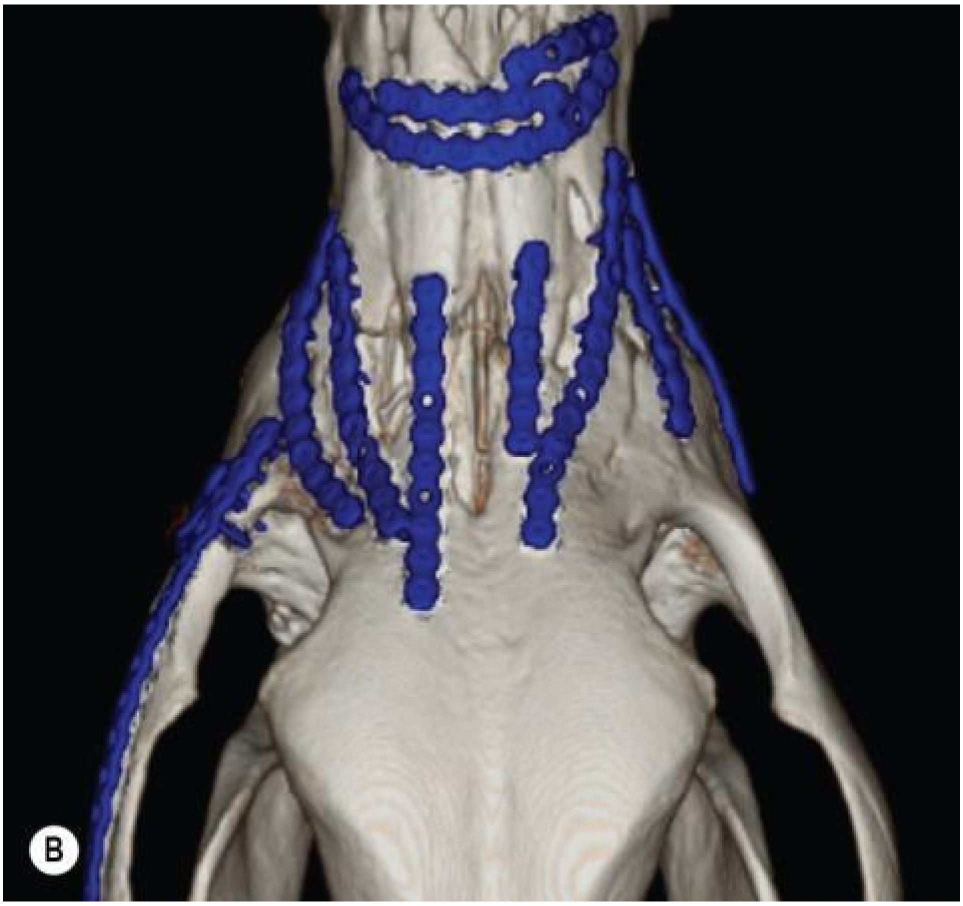
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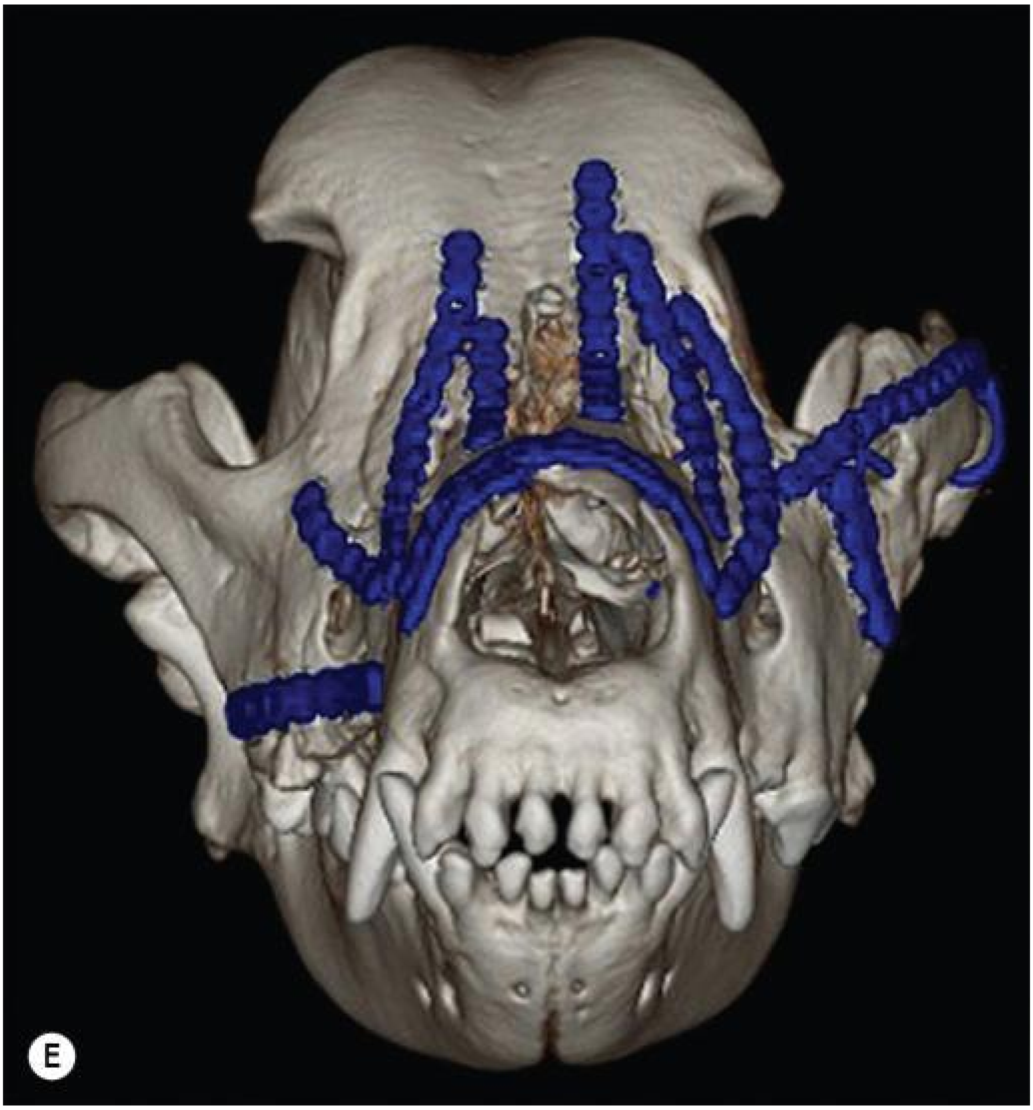
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• **FIG. 33.15** Four-year-old Labrador retriever. **(A)** Preoperative three-dimensional (3D) computed tomography (CT) reconstruction of comminuted nasal and maxillary fractures, dorsoventral view (note that these fractures, combined with the bilateral fractures of the lateral buttresses, have produced a disconnect of the maxilla from the skull). **(B)** Follow-up (13-month) 3D CT reconstruction of the healed repair with multiple Advanced Locking Plate System (ALPS) 5 bridging plates along the medial buttresses, dorsoventral view. **(C)** Intraoperative photograph demonstrating the bridging plate fixation with multiple plates to rebuild the medial buttresses. **(D)** Preoperative 3D CT reconstruction of comminuted nasal and maxillary fractures, rostral view (disconnect of the maxilla from the skull is more obvious on this view). **(E)** Follow-up (13-month) 3D CT reconstruction of the healed repair with multiple ALPS 5 bridging plates along the medial buttresses, rostral view; note that the occlusion has been reestablished. **(F)** Rostral view of the postoperative occlusion. Source: (From Illukka E, Boudrieau RJ. Surgical repair of a severely comminuted maxillary fracture in a dog with a titanium locking plate system. *Vet Comp Orthop Traumatol*. 2014;27:398–404; reprinted with permission.)

Fractures of the mandible

Body of the mandible

A ventral approach to the mandible is used to access mandibular body fractures. Buccal exposure of the mandible is performed apical to the mucogingival junction. Fracture fixation will be dictated by whether the fracture is reconstructible or nonreconstructible, as this will determine whether absolute or relative stability, respectively, can be applied.

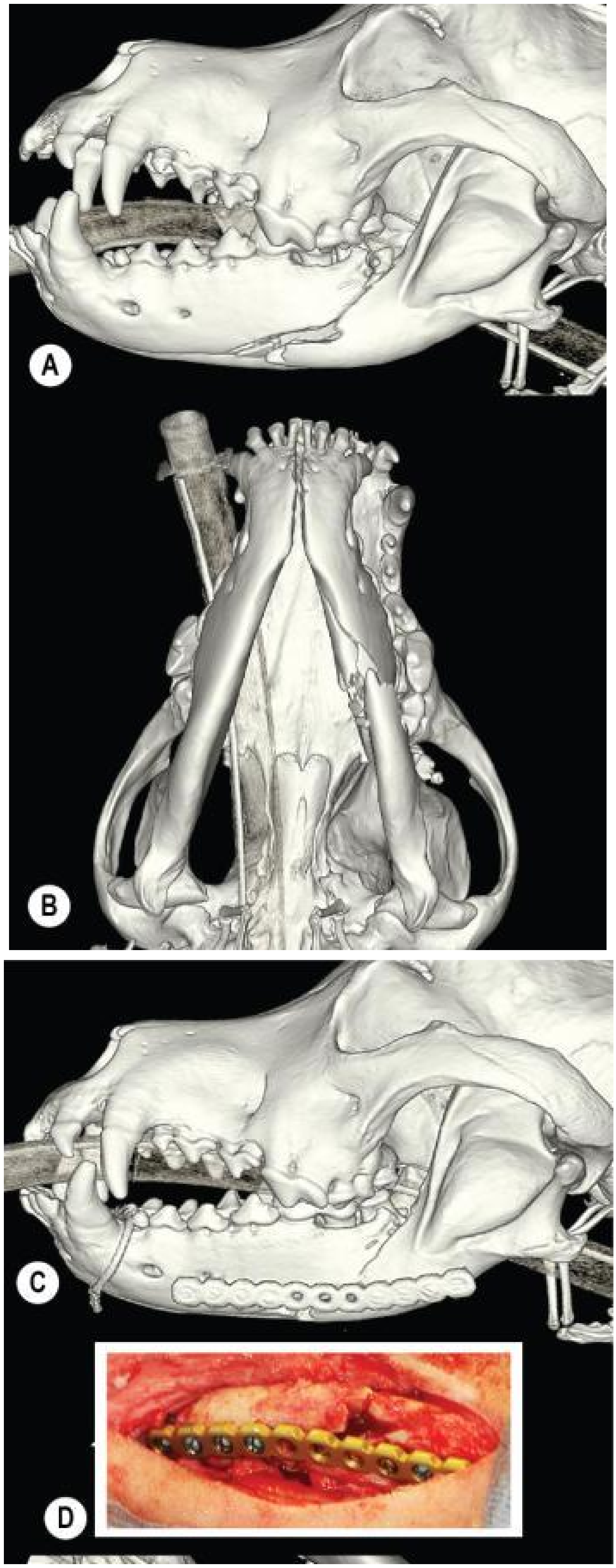
Reconstructible fractures

In a reconstructible fracture, a miniplate can be secured to the bone closer to the alveolar margin; alternatively, in a two-piece fracture, an intraosseous or interdental wire may be applied. This is tension-band fixation, which must be secured first to ensure that the occlusal surface is restored into anatomical alignment under compression. It is only under these conditions that absolute stability can be obtained. As previously described, screws must avoid the tooth roots, and locking screws can be used only in areas where their orthogonal orientation avoids the tooth roots. A second miniplate then is secured parallel to the first miniplate, on the buccal aspect of the ventral mandibular margin (if possible, below the level of the mandibular canal). This plate functions as the stabilization plate. Alternatively, a larger plate may be used as the stabilization plate, especially in medium- to large-breed dogs.[28] The argument in favor of the latter approach is that the masticatory forces of the mandible in animals may be sufficiently great to overwhelm the stability provided by a miniplate at this level, despite the appropriate positioning of a tension-band plate or intraosseous wire along the alveolar margin (parallel to the lines of tensile stress). Because of the mandibular dimensions in this portion of the mandible, and to more simply avoid the tooth roots, applying only the larger plate more ventrally along the buccal aspect may be sufficient, thus avoiding the difficulty of applying the alveolar miniplate and also avoiding potential damage to the tooth roots.[28,29] Success has been documented with both approaches.[4,23,26,27] In the mandibular body, there is sufficient space in all but the smallest dogs, or cats, to place a relatively large plate along the ventral-buccal aspect of the mandible. The greater security provided by the addition of this larger plate may be a practical and sensible alternative to dual miniplate fixation in this area.

Nonreconstructible fractures

Large defects, due to either bone or tooth loss, or comminuted fractures that are nonreconstructible are spanned with bridging plate fixation, providing relative stability. Large gaps may take a long time to heal, even if cancellous bone grafting is performed. Compression fixation can be used in these instances if the fracture gap is spanned by a cortical bone graft (strut).[7] The advantage of a cortical strut is that it is less reliant on the implant as the only means of support. If continuity of bone across the fracture site can be reestablished, the applied shear and bending loads become shared between the bone and the implant, and absolute stability is obtained, resulting in a more stable fixation than if only a bridging device is used.[67] A cancellous graft must be placed at the host-donor interface to ensure healing.[68]

An alternate approach is to bridge the gap with a single large plate, which provides relative stability, recognizing that the implant construct must be of sufficient strength to counteract the forces of mastication, while simultaneously recognizing the need to apply a longer plate with greater screw spacing across the fracture to maximize the mechanical and biologic working distances.[33,37,39,46,69] Applying locking plate fixation across large gaps or areas of comminution can increase the strength and the longevity of the fixation (Fig. 33.16). A second plate, usually a miniplate, may be used to additionally bridge an area of comminution or a gap, placed closer to the alveolar margin. Multiplate fixation will increase the strength of the construct and minimize interfragmentary motion. In some patients, it can be very difficult to apply fixation along the alveolar border as a result of the presence of the teeth; this is especially true in the smaller dog breeds, where the tooth:mandibular height ratio is large.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B
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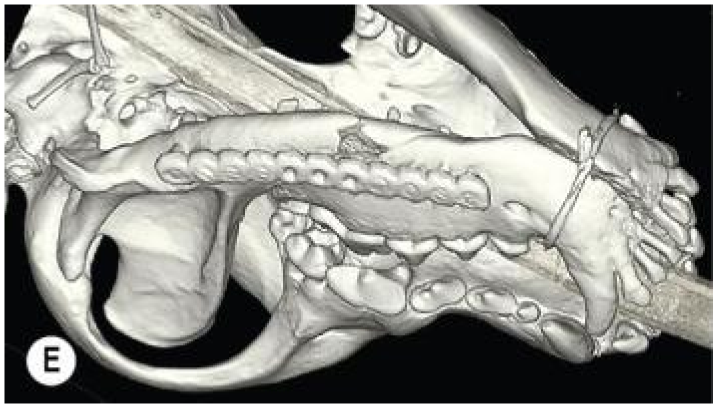


• **FIG. 33.16** One-year-old Labrador retriever with a minimally displaced comminuted left mandibular body fracture and symphyseal separation as a result of having been hit by a car. **(A)** Lateral and **(B)** ventrodorsal three-dimensional (3D) computed tomography (CT) reconstruction demonstrating the fracture obliquity and comminution. **(C)** Postoperative lateral 3D CT reconstruction, **(D)** intraoperative photograph with **(E)** corresponding view of oblique 3D CT reconstruction demonstrating a 2.4-mm locking reconstruction plate placed as bridging plate fixation across this nonreconstructible mandibular fracture. Note that a single plate has been placed on the ventrobuccal surface of the mandible in order to avoid the tooth roots and also the inferior alveolar artery. Notice the long length of the plate with four 3.0-mm locking screws on either side of the fracture and the three open screw-holes at the fracture, demonstrating the principles associated with increasing both biologic and mechanical working distances when using a single plate for fixation.

The addition of a cancellous graft may be necessary to help stimulate bone healing in areas with large fracture gaps. Although this approach is often used in comminuted fractures, it lacks documentation for large gaps (e.g., tumor reconstruction). Use of rhBMP-2 in a compression-resistant matrix has been shown to be a very effective bone substitute in the dog, which stimulates rapid healing across such large gaps (see Chapter 53).[61,62]

Ramus of the mandible

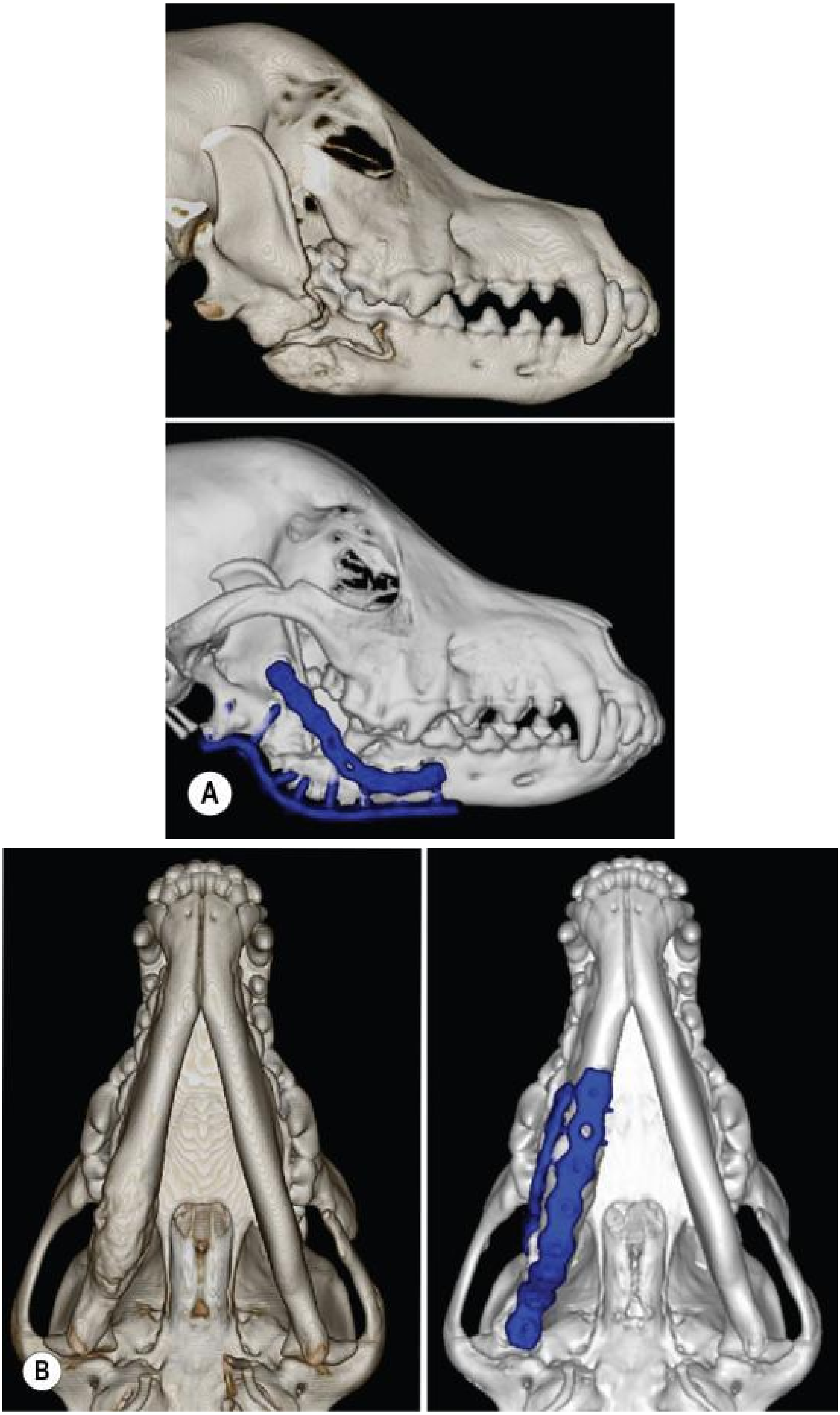
In fractures of the ramus, there is generally limited space available to apply plates with a sufficient number of screws caudal to the fracture location, which is dependent on the amount of intact bone remaining at the level of the angular process.

Reconstructible fractures

As in mandibular body fractures, the tension-band is the first fixation applied to the reduced fracture. At this location, application of a miniplate is easier to perform as opposed to placing an intraosseous wire, which is difficult to pass and tighten at this level. The miniplate is secured along the coronoid crest, thus ensuring adequate screw purchase in the thicker bone of this region. Avoiding the second and third molar teeth is accomplished by placing the rostral extent of this plate ventral to the tooth roots but leaving space for a second, more ventral plate along the buccal surface of the mandible and angular process (immediately ventral to the condylar crest). Alternatively, a larger locking plate may be used along the angular process, as was discussed for mandibular body fractures (Fig. 33.2). An important caveat with plate placement along the coronoid crest is screw length. Perforation of the oral mucosa on the lingual aspect of the mandible may occur if the screws selected are too long. This may also lead to laceration of the tongue. There is minimal soft tissue covering this area of the ramus within the pharynx. In this region, the usual technique to add 1–2 mm to the measured screw length is not used; instead, the screw length equal should be either or subtract 1 mm from this length. The latter method will prevent penetration of the screw into the oral cavity.

Nonreconstructible fractures

Similarly, in nonreconstructible fractures, a bridging plate provides relative stability and can be applied along the buccal surface of the mandible and considered as sole fixation; however, limited caudal plate purchase (fewer than three screws) may be problematic, and additional fixation can be applied along the coronoid crest that will ameliorate any construct weakness under these conditions. A second, small, bridging plate along the alveolar margin can again be applied along the coronoid crest, where the rostral end of the plate is placed ventral to the tooth roots (Fig. 33.17); screw length is critical in this area of placement, and slightly shorter screws are used as discussed previously.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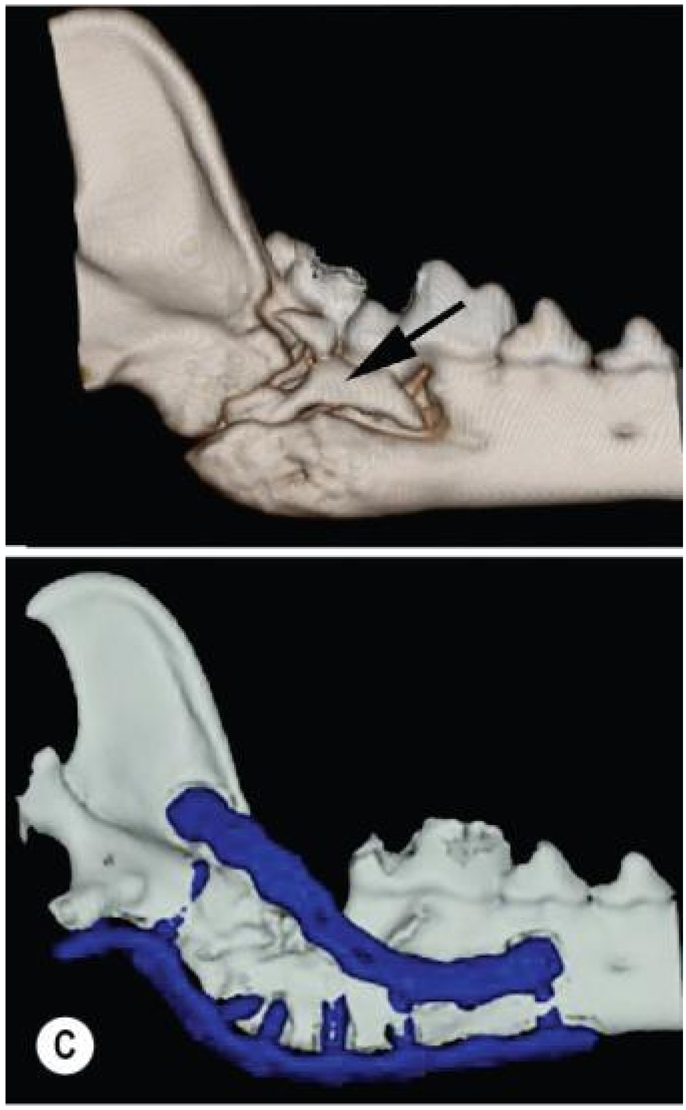


• FIG. 33.17 One-year-old Welsh corgi with a previously comminuted fracture (4 months previously) at the right angle of the mandible, which was presented as an infected nonunion. Preoperative and 8-week postoperative three-dimensional (3D) computed tomography (CT) reconstructions: **(A)** lateral right view of the head. The right mandibular second molar tooth was mobile and was extracted; in addition, the sequestra were removed (also see Fig. 33.18). An Advanced Locking Plate System (ALPS) 6.5 plate was placed along the coronoid crest to bridge the gap along the alveolar surface; an ALPS 8 plate was placed ventrally on the mandible due to limited space and poor bone quality laterally. **(B)** Ventrodorsal views of the head, showing the orthogonal views. **(C)** Isolated views of the caudal right mandible. Note that sequestra are present in the area of the fracture *(arrow)* but are difficult to identify with the 3D reconstruction (compare with Fig. 33.18); healing is well advanced and the bridging plate adjacent to the coronoid crest can be observed spanning the gap at the point of the extracted right mandibular second molar tooth and the associated sequestra.

Additional fixation within the masseteric fossa is unnecessary and of limited value due to the thin bone of this area. Fractures dorsal to a line connecting the occlusal surface with the temporomandibular joint also need not be addressed.

Postoperative care and assessment

Antibiotics

Perioperative and postoperative antibiotic therapy is recommended in the dog[70] and cat,[71] as is the case in humans,[72] since open fractures occur in most of these cases (see Chapter 3).[6] Antibiotic therapy is based on intraoperative cultures and susceptibility testing.

Diet and activity restriction

With rigid skeletal fixation, feeding per os may resume immediately postoperatively. The ability for oral prehension is often regained immediately postoperatively, and patients typically eat and drink normally within 24 hours after surgery, only limited by the degree of pain and/or postoperative swelling that is present. Only soft food (canned food without "chunks" or kibble presoaked with water for several minutes) should be offered. Chewing on hard objects is not permitted for the first 4–6 weeks postoperatively. Playing with any toys, balls, sticks, etc. that the animal chews is to be avoided.

Complications

Malocclusion

Obtaining appropriate dental occlusion is essential. Even small errors will likely become problematic, as function will be adversely affected. This is especially true with plate fixation. Postoperative modification of the reduction/repair is not possible. Attempts to correct occlusion with such treatments as odontoplasty, endodontic procedures, or both, and tooth extraction rarely are effective.[73,74]

Persistent malocclusion, in addition to adversely affecting function, also may result in fixation failure, as abnormal leverage is exerted against the fixation devices. The latter contributes to motion at the fracture site and subsequent loosening of the fixation.

Dehiscence, plate exposure, and infection

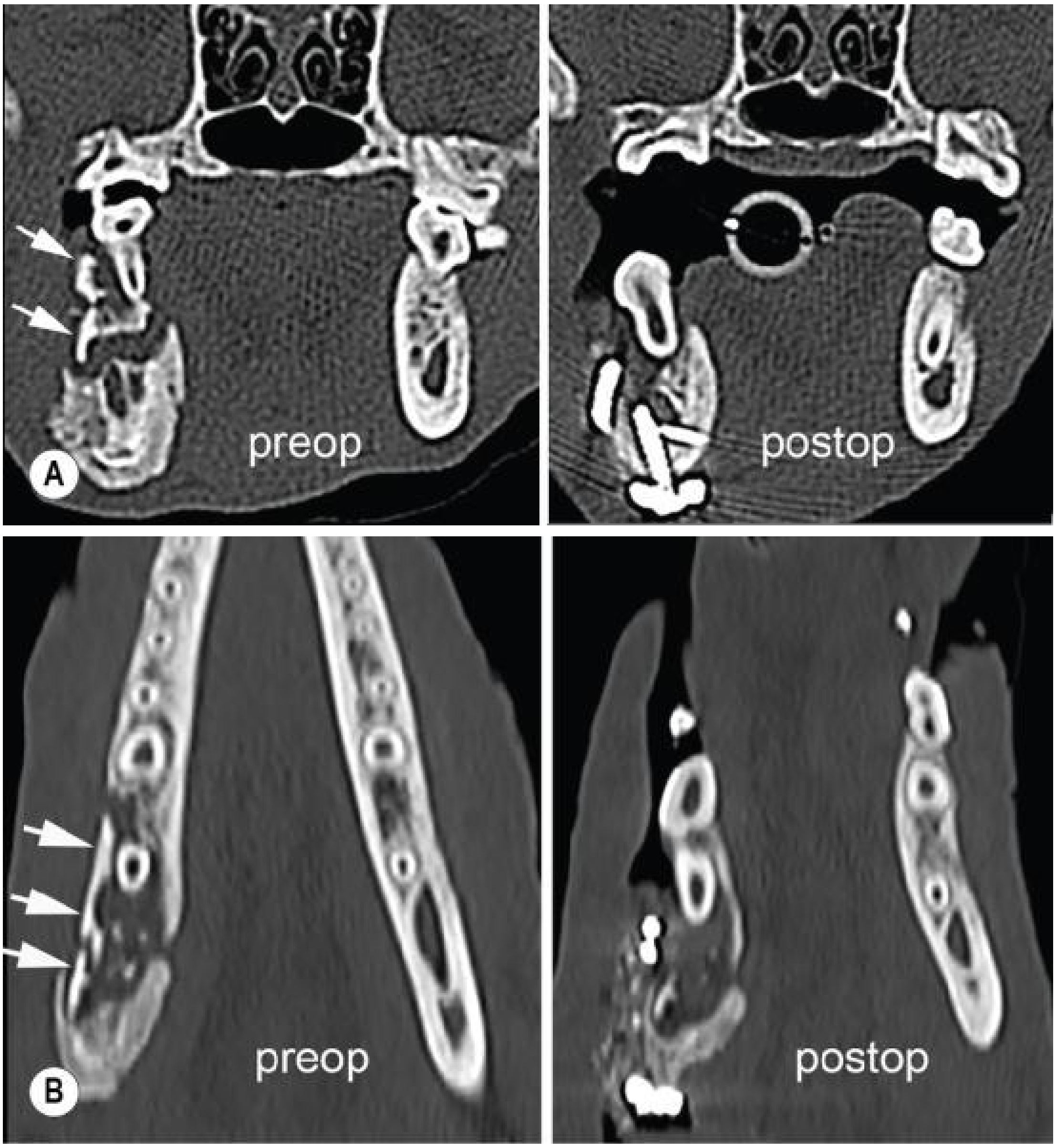
Adequate soft tissue coverage (i.e., soft tissue envelope) over the plates and screws is essential to ensure satisfactory healing and to prevent infection. In the presence of a dehiscence, early closure of the wound to cover the implants is warranted. If insufficient gingiva is present, soft tissue coverage can be obtained using a single pedicle buccal mucosal advancement flap.

In cases with late plate exposure after fracture healing has occurred, the plate(s) should be removed. Because of the small size of miniplates, a portion of the plate may be removed through the area of exposure. Persistent infection, however, may be problematic because of the biofilms present on the remaining portion of the implant, and full implant removal generally is preferred. Regardless, cultures taken directly from bone or implant (not from the drainage) are submitted for bacteriologic cultures, and antibiotic treatment is tailored to these culture results.[75]

In fractures with late plate exposure that have yet to heal, infection often occurs because of construct instability. This is usually secondary to implant loosening, which most commonly is the result of intraoperative technical failures, improper implant application, or malocclusion. In this scenario, debridement and revision of the repair are indicated to attain the requisite stability. Implants are not reused but are exchanged with new implants to again eliminate the bacteria within the implant biofilm and also ensure the requisite stability is obtained.[76,77] In the presence of loose implants, infection is potentiated, and bone is unlikely to heal under these conditions.[78,79]

The conundrum often faced is when infection (either with or without plate exposure) is present with stable implants where the bone has yet to heal, as bone will heal in the presence of infection under stable conditions.[78] Consequently, there often is considerable hesitancy to revise the fixation under stable conditions. However, biofilms are rapidly established, and infection persists and often worsens, leading to severe osteomyelitis; therefore chronic antibiotic therapy in the face of ongoing active drainage is counterproductive.[80] The appropriate treatment is to aggressively debride these areas and exchange the original implants with new implants, identical to that described with unstable implants.[76]

Fractures with infection already established (e.g., infected nonunions) are treated identically as noninfected fractures; however, a source of the infection needs to be identified. In this instance, in addition to the instability that potentiates infection, there may also be a nidus, but in contrast to those cases with implants, the nidus is usually a dead piece of bone (sequestrum). A careful review of adjacent CT slices in more than one anatomical plane will help identify dead bone, as these fragments are denser than the surrounding bone (Fig. 33.18). Necrotic bone fragments need to be removed and fracture stability provided in order for bone to heal as described earlier; furthermore, in most instances, the implants used to stabilize the fracture will likely need to be removed once healing has occurred, as it is not uncommon for bacteria to adhere to the plate surface (biofilm). Antimicrobial therapy is based on bacterial culture and susceptibility testing.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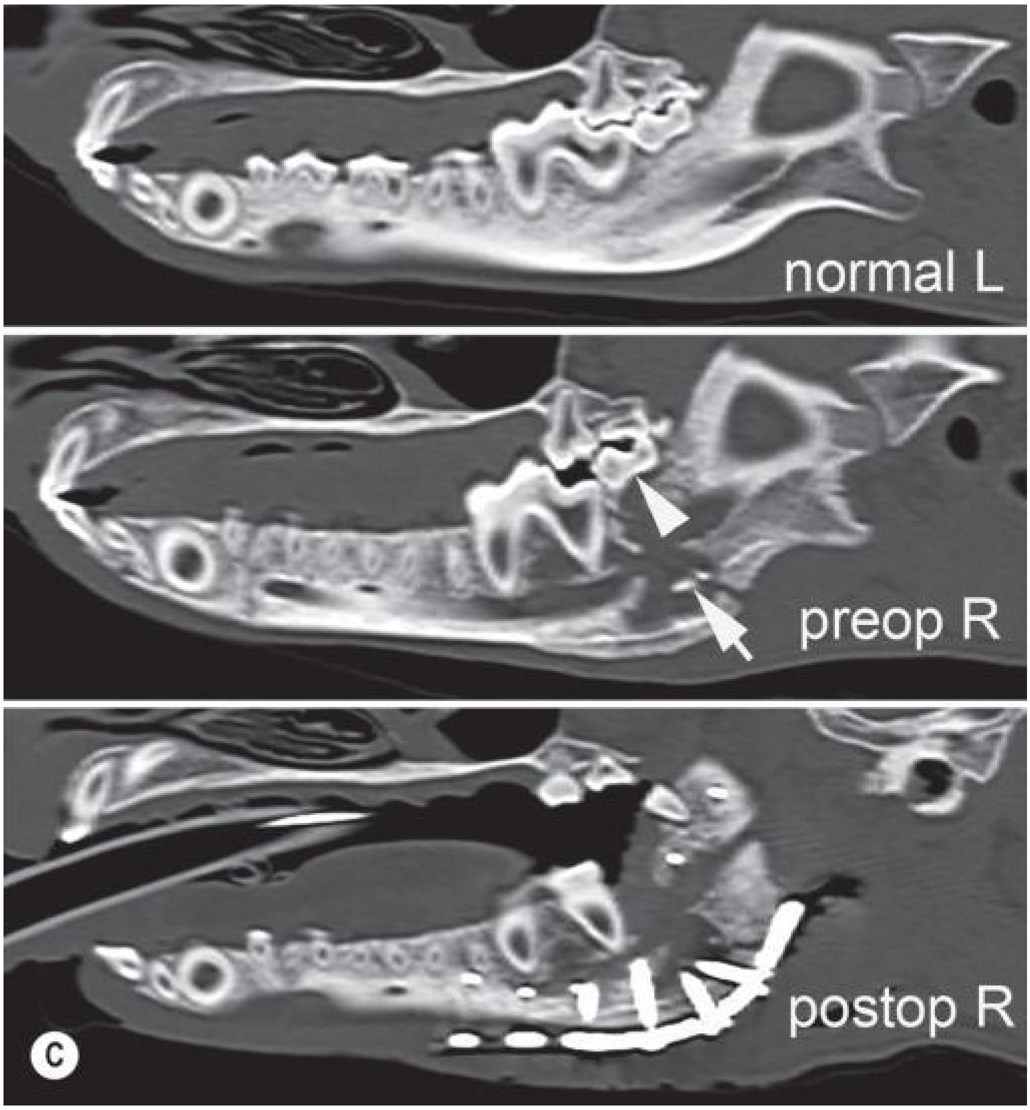

• **FIG. 33.18** One-year-old Welsh corgi, preoperative and postoperative computed tomography (CT) scans; the area of the nonunion is evaluated on frontal **(A)**, coronal **(B)**, and sagittal **(C)** slices, showing that there are areas of increased bone density (sequestra) at the nonunion/fracture site on the lateral/buccal aspect of the mandible *(arrows)*, which are absent on the postoperative views; these were removed intraoperatively. In addition, the exposed root of the right mandibular second molar tooth is evident on the preoperative scan *(arrowhead)*.

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CHAPTER 34

Maxillofacial fracture repair using external skeletal fixation

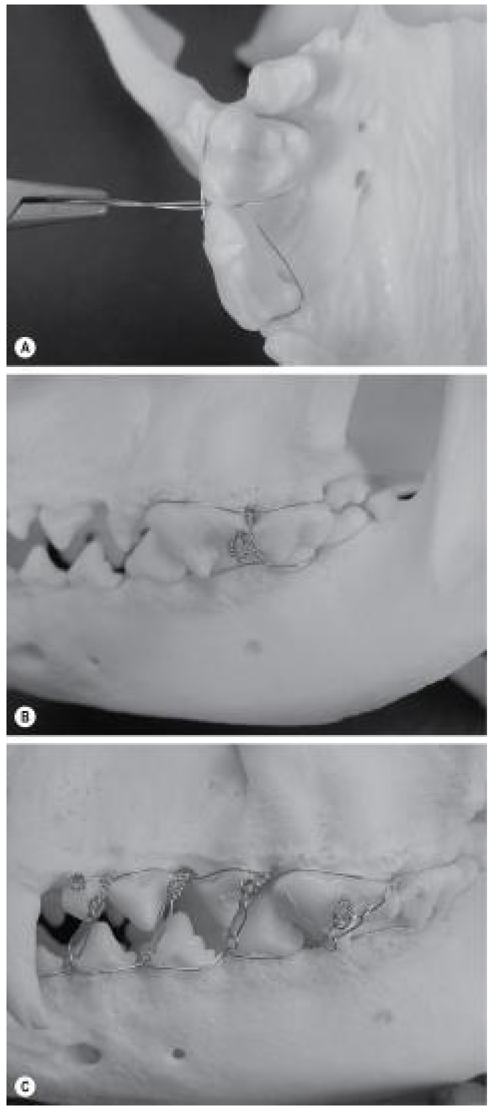
Anson J. Tsugawa, Frank J.M. Verstraete

Definitions

Biplanar: External frame configuration that occupies two planes.
External skeletal fixation: Method of semi-rigid stabilization of bone segments with percutaneously placed fixation pins that are externally connected to one another.
Free-form fixators: Fixator design that consists of full- or half-pins and acrylic columns for connecting bars.
Full-pin: Through-and-through fixation pin that penetrates both skin surfaces and cortices of the bone.
Gunning splint: Indirectly fabricated acrylic or composite intraoral splint secured to the mandible (or maxilla) with circumferential wires.
Half-pin: Fixation pin that penetrates the near skin surface and both cortices of the bone.
Intraoral interdental full-pin: Single intraoral pin anchored to the mandibular canine teeth using a figure-of-eight wire and a bridge of acrylic or composite between the canine and incisor teeth.
Linear fixators: Fixator design that consists of full- or half-pins, connecting bars and clamps.
Type I: Unilateral half-pin frame in a uniplanar or biplanar configuration.
Type II: Bilateral full-pin frame in a uniplanar configuration.
Type III: Bilateral combination half- and full-pin frame in a biplanar configuration.
Uniplanar: External frame configuration that occupies a single plane.

Preoperative concerns

Traditional pharyngotomy or, alternatively, transmylohyoid orotracheal intubation,[1] is the recommended route for intubation to provide uninhibited intraoperative assessment of occlusion and permit maxillomandibular fixation (MMF) if indicated. Temporary MMF is a method of establishing the proper occlusal relationship of teeth before reducing fractures of tooth-bearing bones (Fig. 34.1A–C).[2] Teeth maintain a constant relationship to the bones of the maxilla and mandible, and aligning mandibular or maxillary fracture fragments without establishing pre-injury occlusion will predispose the patient to a suboptimal postoperative occlusion.[3] To obtain the normal occlusion in a dog, the mandibular canine teeth are positioned in the space between the maxillary third incisor and canine teeth, and the crowns of the mandibular fourth premolar teeth are aligned between the maxillary third and fourth premolar teeth.[4] In the dog, MMF is typically achieved through tooth-borne methods of fixation with dental composite bonding of the maxillary and mandibular canine teeth crowns, but may also be performed with bone supported or bone-borne devices such as intermaxillary fixation (IMF) screws placed in the maxillary and mandibular bone that are joined by either wire or elastomeric chains. In humans, traditional Erich arch bars and, more recently, an arch bar-IMF screw hybrid system (SMARTLock™ Hybrid MMF bb™, Stryker CMF, Kalamazoo, MI) are implemented for MMF. External skeletal fixation (ESF) provides semi-rigid stability and patients rarely have to be maintained in postoperative MMF.



• **FIG. 34.1** Wire techniques for maxillomandibular fixation demonstrated on the skull of a dog. Placement of the maxillary wire for an Ernst-type ligature around the fourth premolar and first molar teeth. (**A**) By convention, the wire ends are twisted in a clockwise direction to form a twist knot. (**B**) Completed Ernst-type ligature. (**C**) Stout continuous loop technique. Notice that the wire tails of the completed ligatures are cut short and finished in a "rosette" configuration to minimize trauma to the oral soft tissues.

Surgical anatomy

The fixation pins of an external pin fixator are introduced percutaneously through small stab incisions in the skin and inevitably impale muscle tissue as they traverse a path into the bone. Pin placement, however, should not unnecessarily violate tooth roots and neurovascular structures, and an applied knowledge of the regional cross-sectional anatomy and "safe corridors" in combination with radiographs will facilitate achievement of this goal. This is especially challenging in pediatric patients with unerupted teeth; careful attention must be paid to avoid injuring the tooth buds during surgical reduction. If possible, the neurovascular bundle should be avoided when placing pins in the mandible. In humans, the canal can usually be avoided by placing pins within the inferior border of the mandible. In dogs and cats, the ventral margin of the mandible is usually not of substantial thickness to support pin placement, and as a compromise, pins can be placed dorsal to the mandibular canal between tooth roots. This may be difficult, as the bone that is available for pin insertion is extremely limited in certain areas (see Fig. 27.1).[5] Pins for mandibular fixators are placed in a bicortical fashion with only the pin tip protruding from the opposite cortex. Pin placement in a transmandibular fashion is not recommended because tongue function may be impaired.[6]

In comparison to the bones of the mandible, the maxillary bones are thin, and transmaxillary pin placement is necessary to achieve functional stability with maxillary external fixators. Damage to the nasal conchae and associated hemorrhage are inevitable with this technique. Terminal branches of the facial nerve should be excluded from the skin incisions to avoid paralysis of the upper lip. The infraorbital neurovascular bundle can be readily palpated as it exits its foramen in the maxilla and is easily avoided.

Special instruments and materials

Historical background

ESF of craniofacial fractures was first reported in 1939.[7] Veterinary application of ESF also dates back to the 1930s, and a veterinarian by the name of Stader was the first to introduce a half-pin splint that was widely used on human patients during World War II.[8] Treatment of a comminuted fracture of the mandibular ramus in a dog with a Stader splint was reported in 1947.[9] In the same year, Ehmer developed a veterinary modification of the Anderson splint.[10] The typical veterinary ESF device is uniphasic and is a modification of the Anderson device, consisting of two percutaneously placed pins on either side of the fracture, linked together by a metal bar and connectors. As with many techniques currently used in veterinary oral and maxillofacial surgery, clinical experience with similar medical devices in human medicine usually preceded their use in veterinary medicine. An example of such a device is the biphasic external fixation splint (Joe Hall Morris appliance) for treatment of mandibular fractures that was first introduced in human oral and maxillofacial surgery in 1949, and was later adapted for use in veterinary medicine in the early 1980s.[11–13] Conversely, Becker in the 1950s developed a system consisting of threaded pins, bolts, acrylic, and special instruments that was first used in a variety of domestic animals and shortly thereafter in humans.[14,15] The acrylic connecting bar was secured in place over each pin by means of two nuts.

In the 1970s, the traditional tenets for external fixation were challenged with the introduction of miniplates and screws by Michelet and Champy.[16,17] Many of the comminuted fractures that could previously only be treated by ESF are currently treated by rigid internal fixation (see Chapter 33). In modern day oral and maxillofacial surgery (OMFS) practice, ESF typically only plays a role as an initial bridging technique, to maintain continuity of the defect until resolution of infection and/or stabilization of the soft tissues is achieved and definitive open reduction and internal fixation (ORIF) can be performed.[18] Despite a decline in popularity as a technique for maxillofacial fracture repair, external fixation-type devices are still used for distraction osteogenesis, repair of fractures in other areas of the skeletal system, and ESF has actually experienced a resurgence in popularity due to a trend toward a more “biological” approach to osteosynthesis, where preservation of the fracture hematoma and soft tissues is prioritized.

Components

The basic components of an ESF system include fixation pins, clamps, and connecting bars. The pins are inserted percutaneously on both sides of the fracture and are attached via clamps to an external connecting bar that provides immobilization of the fracture.

Fixation pins

Fixation pins, also referred to as transfixation pins, used for ESF are constructed of implant-grade, hardened stainless steel. Pin shaft diameters vary with the type of ESF system selected. The pin shaft may be threaded or smooth. Threaded pins may be threaded at the end of the shaft (end-threaded) or centrally threaded. Fully threaded or regular Steinmann pins are weak, have a tendency to break, and are not recommended for ESF.[19] The thread pitch can be fine or coarse, fine-pitched for use in cortical bone and coarse-pitched for use in cancellous bone. The profile of a threaded fixation pin refers to whether the threads are machined into the core diameter of the shaft (negative profile) or raised above the core diameter of the pin (positive profile). Due to their reduced core diameter, negative-profile pins are of reduced strength and less resistant to bending forces than positive-profile pins are and should not be used with unstable fractures.[20] The stress focal point of many of the older-design negative-profile pins is at the abrupt threaded–non-threaded junction of the shaft, but newer designs (Duraface®, IMEX Veterinary, Inc., Longview, TX) have a tapered transition from the non-threaded to threaded sections of the pin and do not have this stress riser.[21] Threaded pins have a biomechanical advantage over smooth pins because they are more resistant to axial extraction from the bone.[22,23] Smooth pins must rely on the circumferential forces exerted by the surrounding bone on the pin (radial preload) for retention.[24] The axial extraction resistance of a smooth pin can be improved by increasing the porosity of the pin surface with a coating that encourages fibrous tissue and bone ingrowth.[25] Porous implants may be more prone to infection following contamination, which is a concern when pins are driven through the skin and oral cavity.[25] Pins are also supplied in variety of tip designs. Both fluted and nonfluted tips are available. T-tip pins are a type of nonfluted tip and are the most common tip used in veterinary medicine.[24] T-tip pins produce smaller holes in the bone compared to fluted-tip pins but incur greater tip temperatures during placement because they do not clear bone debris while cutting.[24] Fluted tip designs such as the hollow-ground tip also eliminate the need for predrilling of a pilot hole.[24]

Connecting bars and clamps

Metal (stainless steel or aluminum) clamps and stainless steel, aluminum, titanium, or carbon-fiber composite connecting bars are utilized in the currently available linear ESF systems. Free-form ESF systems use hand-molded acrylic around the external pin ends or acrylic-filled plastic/silicone tubing as substitutes for straight metal bars. The plastic tubing serves as a mold for the acrylic while it is curing. Because of the potential environmental safety hazard associated with the use of acrylics, other materials such as epoxy putty and nontoxic rigid polymers have been investigated as suitable alternatives.[26,27] Although realistically any flexible tubing of appropriate diameter can be used, it has been recommended that the diameter of the free-form connecting bar be two to two and a half times the diameter of the bone being repaired, and three to four times that of the diameter of a metal connecting bar to achieve similar stiffness.[28–30] In studies comparing the mechanical properties of stainless steel and acrylic connecting bars, 19-

mm diameter acrylic columns were found to be stronger than 4.8-mm diameter metal connecting bars.[29,31]

External fixator systems

Linear and free-form ESF frames are used for maxillofacial fracture repair. Linear maxillofacial ESF frames are typically configured in a uniplanar type I or in a modified type II fashion with half-pins. Free-form frames may also be applied in uniplanar, unilateral, or bilateral configurations, but are not classified into types. Half-pins are routinely used in the mandible. Fixation pins for maxillary fractures are placed transmaxillary (through two skin surfaces and four cortices), and are not used as half- or full-pins.

Linear

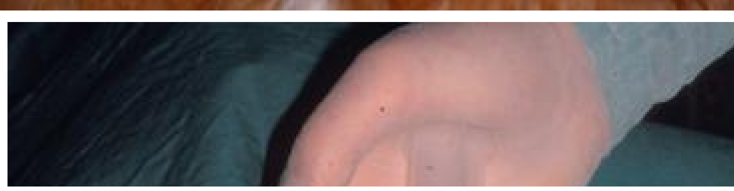
The historical Kirschner-Ehmer (KE) apparatus consists of clamps, connecting bars and fixation pins, and is the most well-known linear ESF system in veterinary surgery. KE clamps and connecting bars are available in three sizes (small, medium, and large), but generally only the small and medium sizes were used in veterinary orthopedics.[4] The large-size clamps and connecting bars are made of aluminum, but the small- and medium-size KE parts are stainless steel.[32] KE fixators were simple to use and affordable, but their stiffness is inferior to that of newer systems.[33] Stiffer ESF devices enhance the speed and quality of fracture healing.[34]

The Securos (Securos TITAN and U-clamp ESF Systems, Securos Veterinary Orthopedics, East Brookfield, MA) and SK® (SK® ESF System, IMEX Veterinary, Inc., Longview, TX) systems are more recent additions to the veterinary ESF market that incorporate the use of modern materials and improved modularity over the traditional KE fixator. In comparison to the KE fixator, both the Securos and SK® systems are more secure at the fixation pin/connecting bar junction, and include larger and stiffer connecting rods for increased biomechanical strength.[35] The Securos system uses fixation pins and connecting rods made of 316L stainless steel, and the SK® system utilizes connecting rods composed of titanium, aluminum, or carbon-fiber composite. The stiffness of the former can be increased by up to 450% with the use of augmentation plates.[36] SK® fixators require a change in the type of connecting rod to adjust fixator stiffness. The clamps for each system are unique and cannot be readily interchanged. Both systems include drill guides that simplify the process of pin placement. The biggest difference between the two systems is that the Securos system gives the operator the option of progressively disassembling the frame (dynamization) to induce adaptive remodeling of the bone and potentially accelerate healing. In the canine tibia osteotomy model, dynamization was shown to have a beneficial effect on fracture healing when performed after 6 weeks.[37]

Free-form

Free-form fixators are a type of external fixator that substitutes the metal connecting bars and clamps of KE-type fixators with acrylic or epoxy putty (Epoxy ESF putty, Jorgensen Laboratories, Loveland, CO) molded into a cylindrical shape over bent pin ends. Embedding the pin ends into the epoxy putty increases the surface area contact and may decrease the chance of pin pull out.[38] As an alternative to bending the pin ends into the epoxy, using half-pins with central knurling and pushing the epoxy around the pins during the curing process may improve the bond between the epoxy and the pin.[38] Acrylic connecting bars can be made by molding doughy acrylic (Formatray™, Kerr, Romulus, MI) over the pin ends (Fig. 34.2A), or by syringing the acrylic (Jet Denture Repair Acrylic, Lang Dental, Wheeling, IL) into a tubular mold pierced over the external pin ends (Fig. 34.2B). Examples of acrylics that can be used include custom impression tray material (Formatray™, Kerr, Romulus, MI), dental acrylic (Jet™ Denture Repair Acrylic, Lang Dental, Wheeling, IL), polymethyl methacrylate bone cement (Simplex P, Stryker, Mahwah, NJ), or hoof acrylic (Technovit, Jorgensen Laboratories, Loveland, CO). Examples of suitable tubular molds include corrugated anesthetic tubing, polyvinyl chloride endotracheal tubes and silicone tubing. Acrylx™ (IMEX Veterinary, Inc., Longview, TX) is a commercially available product that includes a low-odor, methylmethacrylate-based composite resin in a divided cartridge with applicator gun, 6- and 10-mm corrugated plastic tubing and adapter plugs for the tube ends. According to the Acrylx™ product literature, a 1.1-mm pin is the maximum pin diameter for use with the 6-mm tubing, and 2.0 mm is the maximum size for use with the 10-mm tubing. Similar acrylic kits are available from Innovative Animal Products (Rochester, MN) with their acrylic pin external fixation (APEF™) system.


A

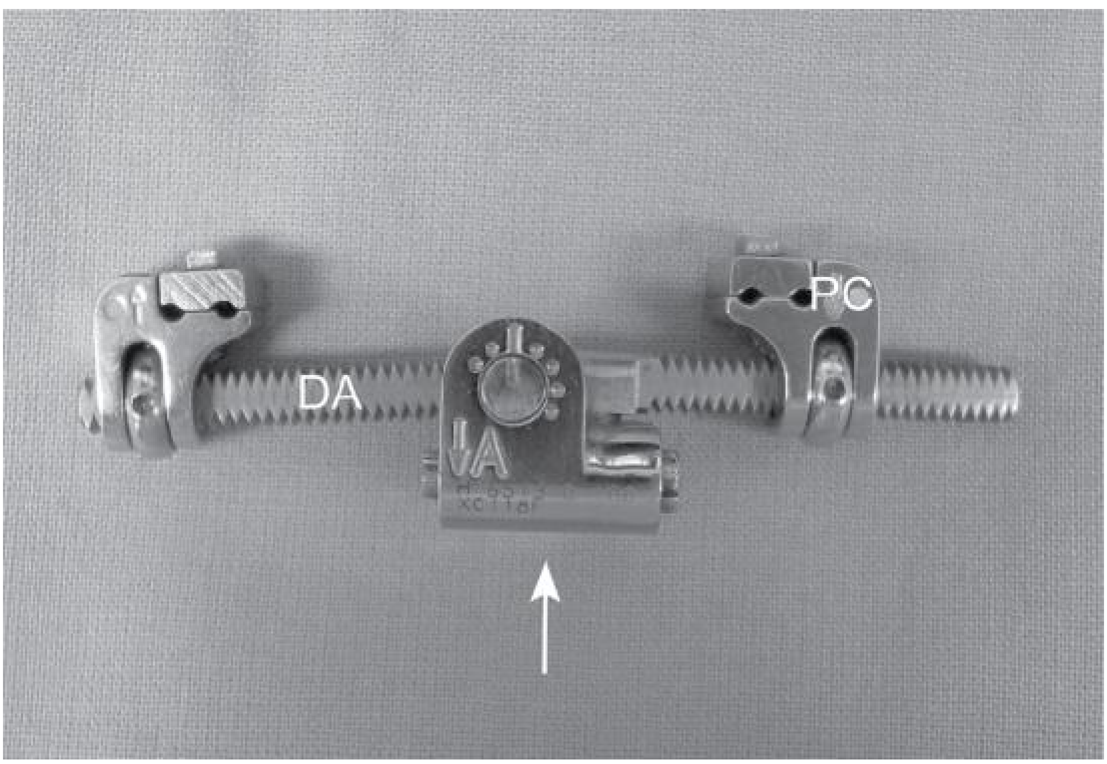


• FIG. 34.2 (**A**) Acrylic external pin fixator with hand-molded acrylic connecting bars for repair of maxillary and mandibular fractures in a dog. (**B**) Acrylic external pin fixator with silicone tubing being used as a tubular mold for the syringed acrylic for repair of bilateral mandibular fractures in a dog. ([A] From Verstraete FJM, ed. *Self-Assessment Color Review of Veterinary Dentistry.* London: Manson Publishing, 1999;99) ([B] From Verstraete FJM. Maxillofacial fractures In: Slatter DH, ed. *Textbook of Small Animal Surgery.* 3rd ed. Philadelphia: WB Saunders; 2003;2190–2207.)

Free-form fixators are lightweight, cost effective, easy to apply, and allow the surgeon to use fixation pins of any diameter and place them in any spatial arrangement without being restricted by clamp size and linearity of the connecting bars.[28] The ability to contour the side bar to the shape of the bone provides increased axial compressive stiffness.[39] The smooth form is devoid of sharp edges and is also more comfortable to the patient.[38] Staged disassembly for dynamization or postoperative adjustments to free-form fixators cannot be performed without cutting the acrylic column with an oscillating saw, and is a recognized disadvantage of this fixator type.[28] The prehension of food can be difficult for patients with bilateral free-form fixators that extend across the rostral midline of the maxilla or mandible.[28]

External fixator systems for distraction osteogenesis

Distraction osteogenesis (DO) is the process of gradual lengthening of bone by traction, and is a surgical procedure that can be used for treating severe deficiencies in maxillary or mandibular length, arch widening, bone transport, airway obstruction, and, less commonly, due to advances in bone grafting and regeneration techniques, reconstruction after mandibular resection and alveolar margin augmentation prior to implant placement.[40] In a recent human study, similar gains in alveolar margin height were achieved with BMP-2 and titanium mesh compared to alveolar DO, prior to implant placement.[41] DO also simultaneously creates adaptive changes in the associated soft tissues and neurovascular structures (a concept referred to as distraction histogenesis), and provides a significant advantage over traditional osteotomy techniques when large skeletal movements are required.[40,42] However, regression is a known problem with this technique and may occur following removal of the DO apparatus; overcorrecting[43] or placement of resorbable plates[44] after removal of the DO apparatus in pediatric patients may lessen this occurrence. The basic components of a DO apparatus for craniofacial distraction resembles a standard linear ESF device, and many of the older-model (extraoral) distraction devices can also be used for fracture fixation (Fig. 34.3). In the interest of reducing the potential for unsightly scars, most of the current DO devices used for human patients are internal devices that are placed subcutaneously or intraorally. The dog has been used extensively by human oral surgeons as a model for DO.[45–49] Although DO is currently uncommonly used in veterinary oromaxillofacial surgery patients, it is another potential area where three-dimensional printing may play a valuable role in treatment planning.[50,51]



• **FIG. 34.3** Extraoral distractor for distraction osteogenesis. Note the similarities to a standard external pin fixator: distractor arm *(DA)*; pin clamp *(PC)*. The distractor body is identified with an *arrow*. The inset screw in the distractor body is turned to make angular adjustments to the distractor. (Distractor courtesy of Dr. SP Bartlett, Hospital of the University of Pennsylvania, Philadelphia, PA.)

Therapeutic decision-making

Indications

Historically, it was believed that the open reduction of comminuted fractures disrupted the tenuous blood supply of the fracture fragments and increased the likelihood of resorption, sequestration, and infection: the "bag-of-bones" aphorism.[52] The technique of external pin fixation avoided direct invasion of the traumatized area, prevented stripping of the periosteum and blood supply to fracture fragments, and was the primary method of treatment for comminuted fractures. The introduction of less invasive surgical approaches and advances in bone-plating systems in human oral surgery have limited the indications for external pin fixators in human patients, and even comminuted fractures are often treated with reconstruction plates and screws.[53,54]

Most maxillofacial fractures are grossly contaminated fractures that are directly exposed to the external environment through the skin, mucosa, or periodontal ligament. Diffuse infection of the bone at the fracture site is a contraindication for open fracture reduction, and is an example where ESF is still the treatment of choice.[53] In these instances, ESF is used as a temporary fixation technique, until the infection is under control and the fracture can be definitively debrided and reconstructed. Despite their limited use in human oral and maxillofacial surgery, ESF devices are still widely used in veterinary medicine, which may be explained by their simplicity and low cost. Currently, the indications for ESF in maxillofacial fracture repair are mainly: grossly infected jaw fractures, pathologic fractures (tumors, odontogenic cysts), fractures of atrophic edentulous jaws, severely comminuted fractures (gunshot wounds) with a tenuous blood supply, and in medically compromised patients (Box 34.1).[53,55] In the case of medically compromised, critically ill patients, ESF offers a simple, temporary means of fixation with minimal operating time.[55] ESF devices are also useful for the treatment of nonunion fractures where bone grafting is necessary, corrective osteotomies, and as a method of interim stabilization before jaw reconstruction.[52] Maxillofacial injuries with continuity defects may require bone grafting. These injuries often have extensive skin and mucosal lacerations. The immediate placement of bone grafts in these contaminated wounds should be avoided because of an increased risk of infection.[56] Grafts are avascular and do not have the ability to fight infection.[57] An ESF device will maintain control of the residual jaw segments until the soft tissues have healed and the defect can be grafted.[56] Mandibular reconstruction plates serve a similar function, are the more current option for treatment, and offer a more elegant solution for repair. When jaw segments are not maintained in anatomic alignment, significant atrophy and fibrosis of the muscles of mastication may occur, making it extremely difficult to realign the fragments at the time of reconstruction.[57]

• BOX 34.1

Indications for External Skeletal Fixation in Oral and Maxillofacial Surgery

1. Infected jaw fractures
2. Severely comminuted jaw fractures
3. Atrophic edentulous jaw fractures
4. Jaw fractures in irradiated patients
5. Nonunion jaw fractures

6. Jaw fractures where there is significant soft tissue loss
7. Control of residual jaw segments until jaw reconstruction
8. Jaw lengthening and bone transport

Biomechanical considerations

Pin–bone interface

Pin design, pin insertion technique, bone quality, osseous response to pin implantation, and loading are factors that affect the integrity of the pin–bone interface.[58] The pin–bone interface is the weakest link of an external pin fixator system.[59] Instability at the pin–bone interface is the most common reason for fixator failure.[58] Motion is detrimental to the pin–bone interface, and can predispose to pin tract sepsis, fracture nonunion, delayed bony union, and increased patient morbidity.[58,60,61] Excessive motion promotes the development of fibrous tissue over bone at the pin–bone interface.[58] Positive-profile threaded pins have greater bone-holding capacity than negative-profile threaded or smooth pins.[58] Although bone damage during pin placement is inevitable, micromechanical and thermal damage to the bone can be minimized by using fluted-tip pins, predrilling pilot holes, and using low-speed drills (300 rpm or less) for pin placement.[58] Bone temperatures adjacent to the pin tract are lower for fluted-tip pins compared to trocar-tip pins.[24] Predrilled pilot holes that approximate the inner diameter of the positive-profile pin improve pin stability and reduce microstructural damage.[62] The use of high-speed drills (700 rpm or greater) for pin placement should be avoided to reduce the danger of thermal bone necrosis.[58]

Pin selection

Threaded pins have greater holding power than smooth pins and may decrease the incidence of the premature pin loosening.[60] Smooth fixation pins will conceivably cause less trauma to neurovascular structures than threaded pins if placed in the mandibular canal.[63] Larger-diameter and shorter pins are less flexible than smaller-diameter, longer pins.[39,64] Pin stiffness is directly related to the fourth power of the pin radius.[65] The largest-diameter pin possible that does not exceed 20%–30% of the bone diameter should be selected.[66]

Pin placement

The patient is positioned in sternal or dorsal recumbency. Local anesthesia can be administered to the selected pin sites. Fixation pins are inserted through longitudinal percutaneous stab incisions. Longitudinal incisions are less likely to transect superficial vessels and nerves.[67] A small hemostat is used to dissect the soft tissues and expose a small area of bone. Pins may be placed exclusively by hand or power drill, or after predrilling a pilot hole with a drill bit 0.1–1 mm smaller than the diameter of the pin.[19] There are disadvantages to each technique: pin tract enlargement from wobbling with hand placement, thermal bone necrosis with power drilling, and the inconvenience of the extra step associated with predrilling. Predrilling of a pilot hole at low speed (150 rpm) followed by hand placement of the pin is advocated in the current veterinary literature.[33,68]

The fixation pins that are located adjacent to the fracture line are commonly referred to as the "mean" pins, and the second or third pins placed in each fragment are often called the "extreme" pins.[69] By convention, the extreme rostral and caudal pins are inserted first. Pins should be placed no closer than 10 mm from the fracture line and approximately 25 mm apart from each other.[55,69] Care should be taken to avoid dental trauma when placing the pins. Temporomandibular joint range of motion should be maintained throughout treatment.[33] A minimum of two pins rostral and caudal to the fracture line is appropriate for most fractures.[52] The number of pins placed in each fragment influences the stiffness of the fixator and load distribution to the pins.[19] The addition of a third pin to each fragment will increase stability by 50% if the pins are aligned in a row, and by greater than 50% if arranged in a triangular configuration.[52] Beyond the addition of a fourth pin per fragment, the mechanical advantage is negligible.[19] Each pin should be placed in a divergent fashion and engage both cortices.[69] Angled pin placement maximizes the surface area of the pin in contact with cortical bone and increases fixator stiffness.[70] The optimum angle for pin insertion is 70 degrees relative to the long axis of the bone.[71] If a biphasic splint is used, the long axes of the screws should diverge by an angle of approximately 5–7 degrees.[69]

Surgical considerations

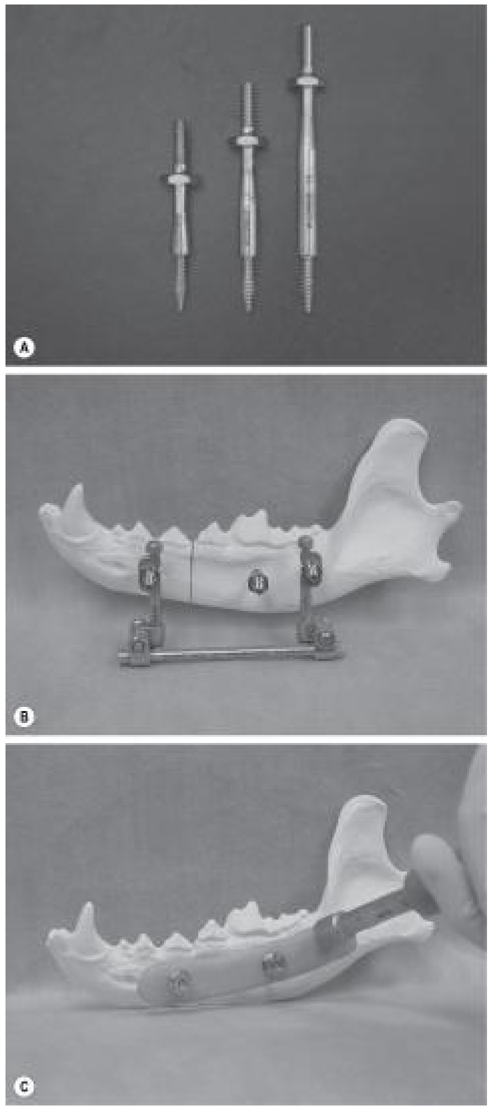
Open versus closed fracture reduction

External pin fixators for maxillofacial fracture repair are preferably placed in a closed fashion. A closed approach will avoid further disruption of the blood supply to the fracture fragments.[72] Closed reduction of a displaced mandibular fracture can often be facilitated by reestablishing the patient's pre-injury occlusal relationship before attempting fixator placement. Similar to the hanging limb technique for reducing long bone fractures, restoring the patient's occlusion will help pull the bone segments into better alignment. Open reduction is occasionally necessary, and may be indicated when there is significant fracture displacement, if debridement is indicated, with autogenous bone grafting, or if the fixator is used as an ancillary device.[33,72]

Clinical applications of external skeletal fixation

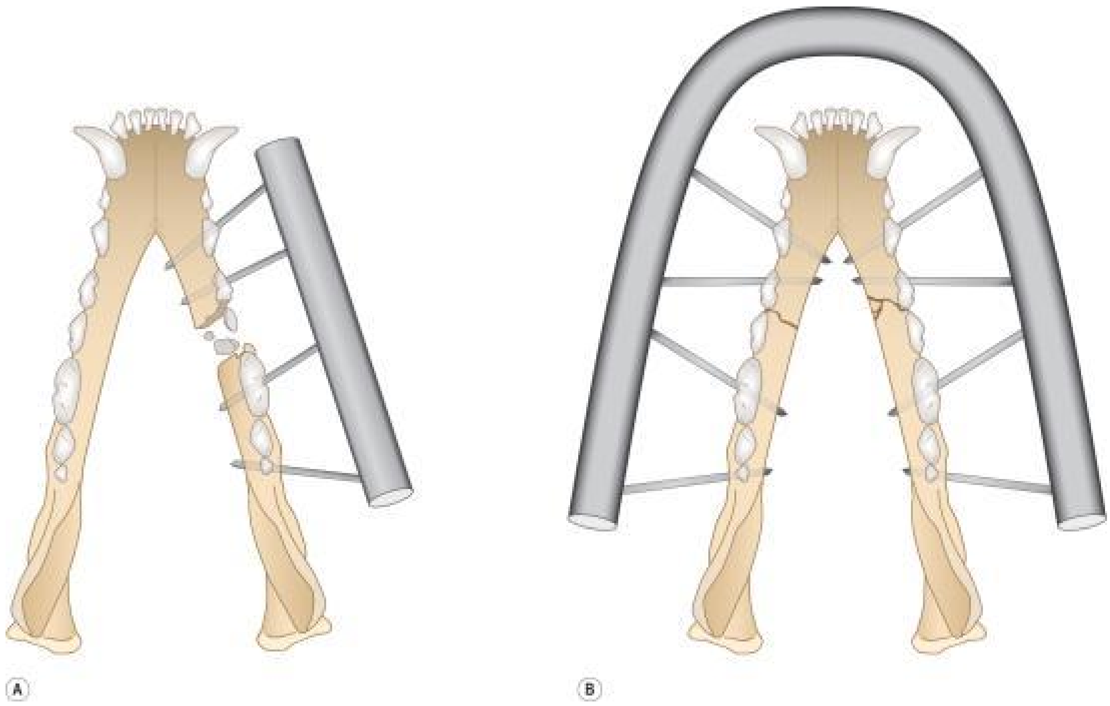
Fractures of the mandible

In human oral and maxillofacial surgery, mandibular fracture repair by external pin fixation is synonymous with the Joe Hall Morris biphasic splint (Bi-Phase External Fixation System, W. Lorenz Surgical, Jacksonville, FL) (Fig. 34.4).[53] The biphasic nature of the splint refers to the two-part procedure required for its installation, fracture reduction with specialized screws and a reduction apparatus, followed by anatomic alignment and fixation with an acrylic bar to connect the screws.[12] The screws for the Morris splint are too long for the mandibles of small dogs and cats, and can only be used in dogs with sufficient mandibular thickness to accommodate the length of screw, generally dogs greater than 12–15 kg.[12]

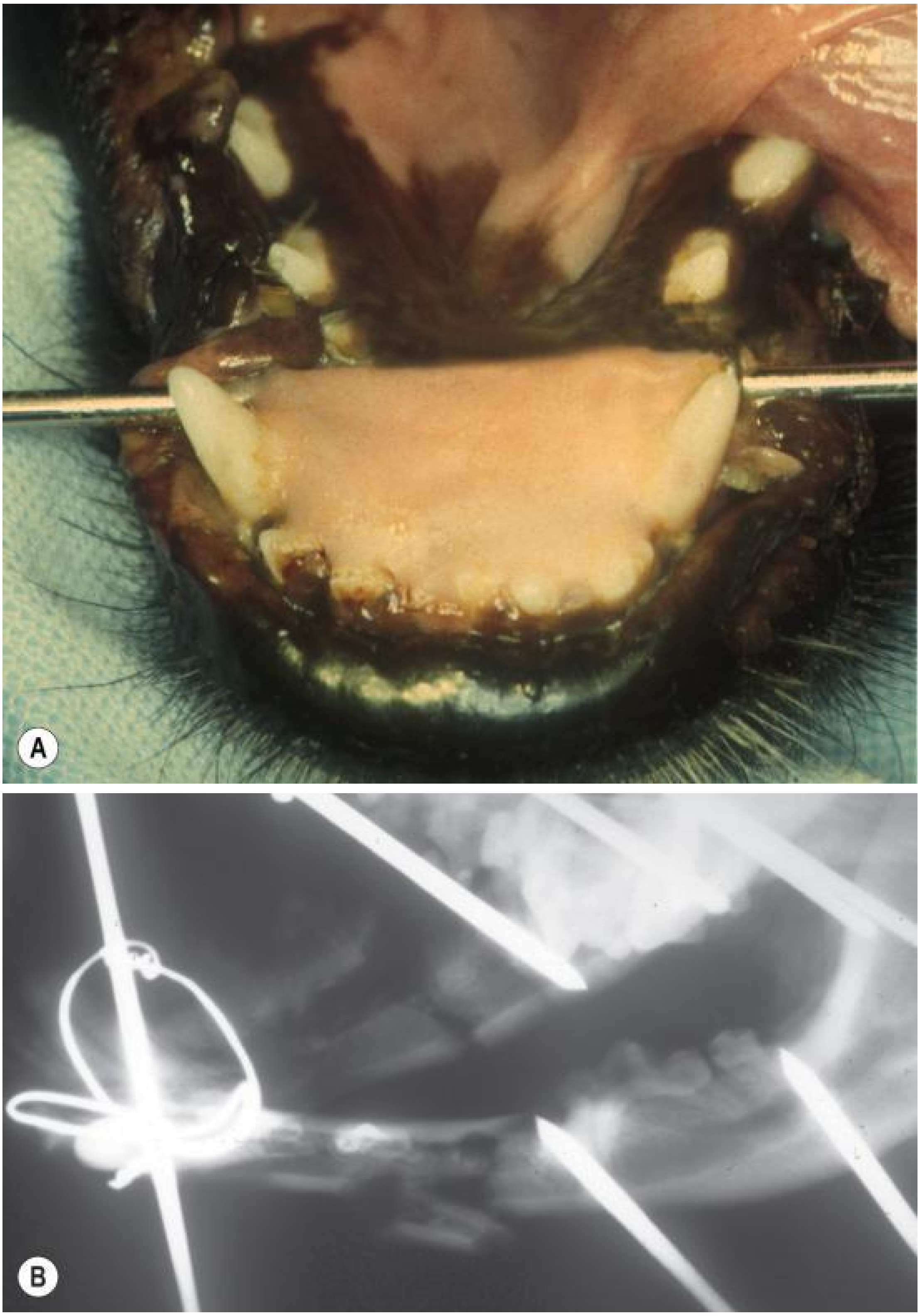


• **FIG. 34.4** Joe Hall Morris biphasic splint (Bi-Phase External Fixation System, W. Lorenz Surgical, Jacksonville, FL) demonstrated on a mandibular model. (**A**) Titanium self-drilling screws (left to right): short (42 mm), medium (51 mm), and long (64 mm). (**B**) Phase I: fracture reduction with primary mechanical splint (screw holding assembly and rod). (**C**) Phase II: secondary splint (acrylic bar) replaces primary mechanical splint.

Mandibular fractures in dogs occur most commonly in the premolar region.[73,74] External pin fixators can be used for both unilateral and bilateral fractures of the body of the mandible (Figs. 34.5 and 34.6A and B).[75] For most mandibular fractures, at least two fixation pins should be inserted on either side of the fracture in the ventral mandibular border or dorsal to the mandibular canal between tooth roots. Although the ventral border of the mandible is the compression surface, the ventral border is the favored location for mandibular pin placement to avoid tooth roots and the mandibular canal; however, as mentioned previously, this is often not feasible due to the insufficient thickness of the bone in this area.[12] External pin fixation of mandibular fractures is largely limited to fractures of the mandibular body.[4] Pin placement can be difficult in caudally located mandibular fractures where bone thickness is inadequate, and mandibular fractures rostral to the third premolar teeth where there is insufficient space for the placement of multiple pins.[4] An alternative technique for rostral mandibular fractures has been described that uses a single intraoral interdental full-pin in lieu of multiple fixation pins.[76] The single intraoral pin is anchored to the mandibular canine teeth using a figure-of-eight wire and a bridge of acrylic between the canine and incisor teeth (Fig. 34.7).[76] The rigidity of this technique has been tested on a mandibular fracture gap model where osteotomies were performed between the third and fourth premolar teeth, and has been shown to be as stiff as fixator configurations containing two or four additional pins in the rostral fracture fragments.[76] The natural dental interlock between the maxilla and mandible resists torsional forces and imparts the necessary additional stability to the fracture repair that would not have been sufficiently addressed with a single intraoral full-pin alone.[76]



• **FIG. 34.5** Line drawings of type I free-form fixators used for the repair of (**A**) a unilateral fracture of the body of the mandible with a substantial missing fragment and (**B**) bilateral fractures of the mandibular bodies.



• **FIG. 34.7** (**A**) Clinical picture and (**B**) accompanying radiograph of the intraoral interdental full-pin technique for repair of rostral mandibular fractures. A medium centrally threaded 3.2-mm pin was secured to the distal aspect of the mandibular canine teeth with a figure-of-eight 18G wire. Polymethyl methacrylate was applied over the wire and between the canine teeth for additional stability. (Photographs courtesy of Dr. MM Smith.)

Another alternative, the Gunning splint, utilizes an indirectly fabricated acrylic or composite intraoral splint, or in humans a patient's dentures, secured to the mandible (or maxilla) with circumferential wires.[77] This type of splint is ideally suited for the repair of mandibular fractures in edentulous patients or those with mixed dentition, either as a sole method of fixation or to augment another form of fixation. In humans, Gunning splints are traditionally combined with MMF or elastics to establish and maintain occlusion.[77]

Fractures of the maxilla

Fractures of the maxilla are less common than mandibular fractures and often are not sufficiently displaced to warrant surgical repair.[63] In contemporary trauma surgery practice, even severely comminuted and unstable maxillary fractures are optimally treated with open reduction and miniplate osteosynthesis. External fixation can be used as an alternative technique to plate osteosynthesis where cost is a factor, but enough maxilla must be available caudal to the fracture to allow placement of a sufficient number of pins.[75] After preinjury occlusion is obtained with one of the previously described temporary MMF techniques, the pins are driven percutaneously through non–tooth-bearing portions of the maxillary bones and across the nasal cavity.[77] Long-term nasal problems from the transmaxillary placement of the pins are usually not significant.[75]

Other

A nonunion of a mandibular fracture can occur when diseased teeth in the fracture line are not extracted prior to fixator placement.[28] Nonunions of mandibular fractures have classically been treated by autogenous cancellous bone grafting, and more recently by regenerative techniques using rhBMP-2,[78] but can also be effectively treated by bone transport with distraction osteogenesis. The dog has been used as a successful model for segmental mandibular regeneration by bone transport, and good long-term results have been reported.[45,46] Bone transport is the gradual movement of a free segment of bone (transport disk) across an osseous defect.[79] A transport disk of no less than 15 mm is created from the caudal bone segment through an extraoral approach. The gingival tissues are not perforated during the corticotomy, and the contents of the mandibular canal are preserved. The pins for the external fixator are placed through separate stab incisions, and a minimum of two pins are placed in the transport disk to achieve rotational stability. After formation of a reparative callus (5–7 days), the disk is transported at a rate of 1 mm per day until it reaches its docking site. Upon completion of bone transport, the transported and targeted bone segments are fused by compression forces.[79] Compression forces applied to the bone segments results in transformational osteogenesis with necrosis of the pathologic tissues, resorption, remodeling, and fusion of the bone segments.[79] Bone transport uses the original mandible as a template and maintains the size and shape of the original mandible in the neomandible.[80] The local soft tissues (gingiva, buccal, and lingual sulcus) are also recreated with bone transport.[80]

Postoperative care and assessment

Aftercare

It is common practice for external pin fixators to be wrapped to prevent injury to the client (e.g., pin impalement), to avoid pin ends snagging on objects in the environment, and to minimize movement between the skin and pins. Clinical movement at the pin site will disrupt the chemotactic gradient used by phagocytes to target bacteria at the pin site, predisposing to bacterial proliferation, sepsis, and chronic drainage at the pin sites.[68] For obvious reasons, external pin fixation devices placed in the maxilla or mandible cannot be similarly wrapped without causing significant irritation to the patient. A reasonable compromise to a circumferential bandage is to place gauze sponges, or alternatively the foam portion of a used surgical brush, inserted between the skin and fixator.[38] The gauze sponges should initially be changed on a daily basis, and the pin sites cleansed daily with a suitable antiseptic scrub (chlorhexidine gluconate or povidone-iodine) to lessen soft tissue inflammation. Antibiotic ointment may also be applied around the pin tracts to reduce infection. Inflammation of the soft tissues surrounding pins has been shown to increase patient morbidity.[67] The frequency of bandage changes can be decreased as the pin sites heal and with development of bacteriostatic granulation tissue at the pin sites. The patient should be rechecked twice weekly for the first 2 weeks, and then weekly until fixator removal, which is usually at 6 weeks.[69]

Diagnostic imaging

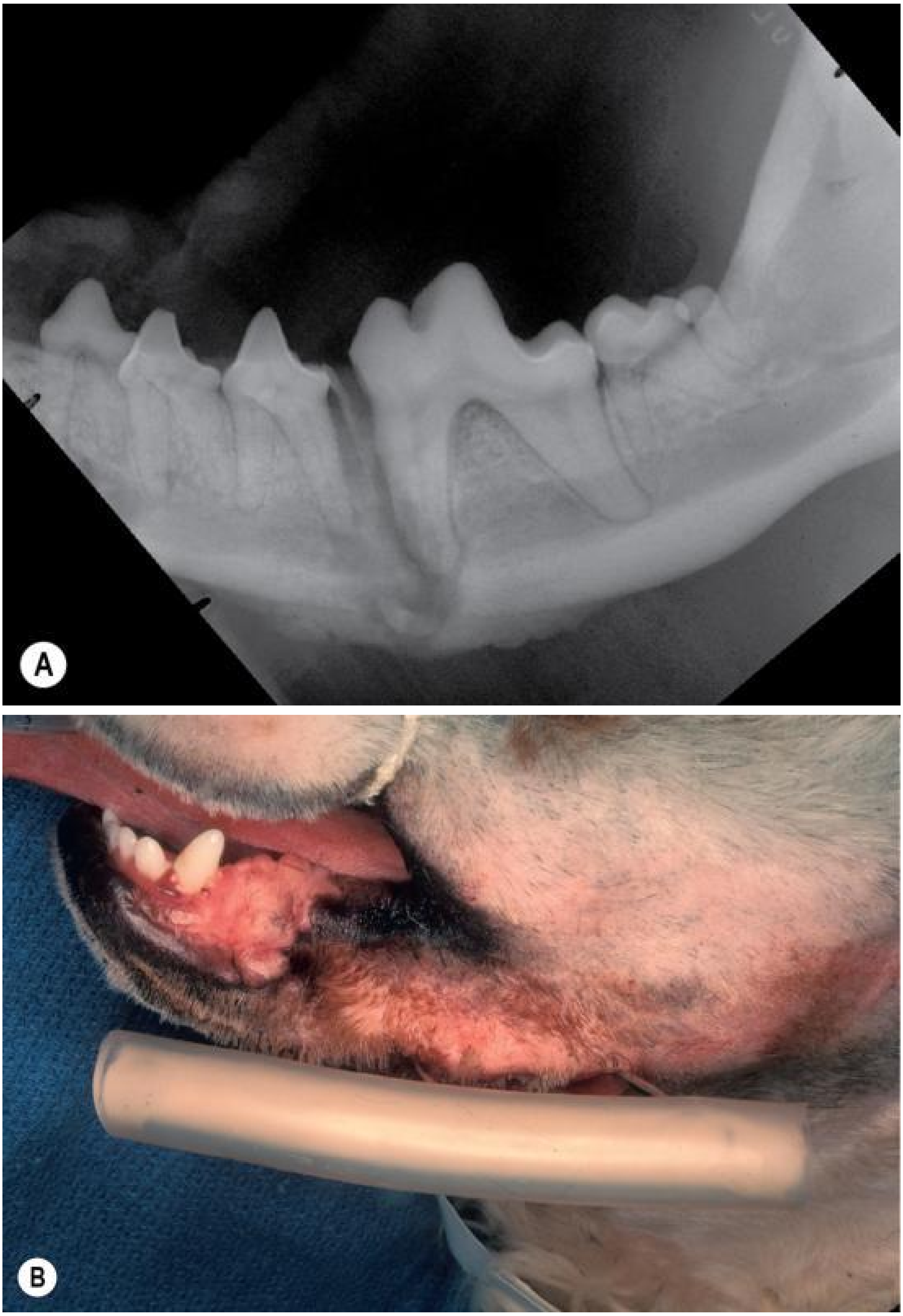
Postoperative radiographs, or computed tomography (CT) when available, should be obtained to document pin placement and fracture reduction. Bone healing is assessed radiographically at 6 weeks, unless clinical signs would indicate possible complications sooner. Follow-up radiographs are best obtained under general anesthesia and should be evaluated from a minimum of two directions if plain films are used, or in the sagittal plane if CT is available.

Fixator removal

External fixators should be removed at the earliest evidence of complete bony union.[81] The recommended time to ESF removal in dogs and cats is 6 weeks.[63] The status of repair and bone consolidation can be manually assessed following careful disassembly of the connecting bar and clamps or after sectioning of the acrylic between the pins adjacent to the fracture line. If healing is inadequate, the connecting bar and clamps can be readily reassembled or rejoined with additional acrylic. At the time of definitive removal, acrylic columns can be notched and cut with an oscillating saw, and the pieces of acrylic attached to the pins can be used as handles for removal of the pins.[31] Alternatively, the pins can be backed out using a pin chuck.[82]

Complications

Due to the severe nature of the fractures treated by ESF, in humans, the complication rate is reportedly very high, upwards of 35%.[55] The most common complication of external pin fixation is infection and necrosis of bone directly adjacent to the pin tract (pin tract osteomyelitis).[83] Radiographically, a ring-shaped zone of osteolysis (ring sequestrum) with variable periosteal new bone formation and osteosclerosis may appear around the pin tract (see Fig. 34.6C and D).[84] A ring sequestrum is best visualized on a radiograph where the primary X-ray beam is directed parallel to the pin tract.[85] Excessive heat build-up or thermal necrosis of the bone at the pin insertion site is a major predisposing factor in the development of pin tract osteomyelitis.[74] The use of power drills for pin placement and cortical placement of the pin may increase the risk of thermal necrosis.[84,86] If a power drill is used, the optimal drill speed to avoid bone necrosis is 500 rpm.[55] Predrilling of pilot holes prior to pin placement decreases microstructural damage and reduces pin loosening and resorption.[38] Thermal damage to the soft tissues and bone can also result from the exothermic reaction of polymethyl methacrylates used for acrylic connecting columns.[87,88] The potential for thermal damage can be minimized by using the smallest-diameter acrylic column (i.e., least amount of acrylic) possible without sacrificing fixator strength and by positioning the acrylic column no less than 10 mm from the patient's body surface during curing of the acrylic.[87,88] The elevation in temperature associated with the exothermic reaction of the acrylic is directly proportional to the diameter of the acrylic column.[38] Increasing the distance between the connecting bar and the skin decreases the axial compression stiffness of the fixator, particularly for unilateral frames, and should be kept to a minimum.[39] If placed too far from the skin, connecting bars can also be cumbersome for the patient, but should be placed far enough away to accommodate for soft tissue edema.[55] The use of saline-soaked gauze sponges between the patient and the acrylic, or a constant saline drip at the pin–acrylic interface during curing of the acrylic can also be used to decrease the malignant transfer of heat to bone and soft tissues, and petroleum jelly–impregnated gauze placed around the pins can be used during the initial healing phase.[31,87] Mobility at the pin site as a result of suboptimal pin placement technique or inadequate patient restriction can also predispose to pin tract infection. Most cases of pin tract osteomyelitis can be treated conservatively with antimicrobial therapy and local wound management, and usually does not adversely affect outcome.[84,89] A ring sequestrum should be surgically removed (see Fig. 34.6D).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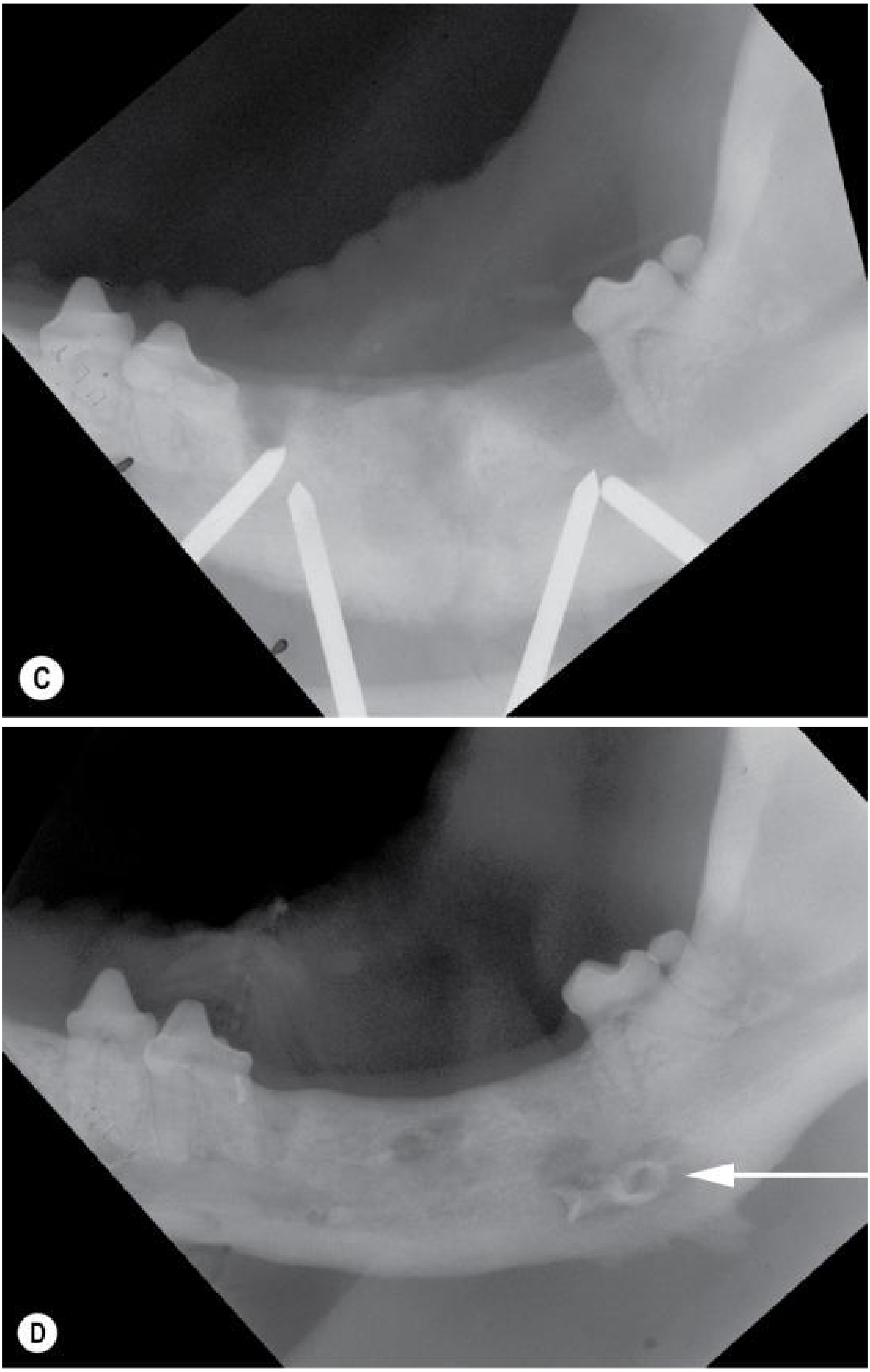

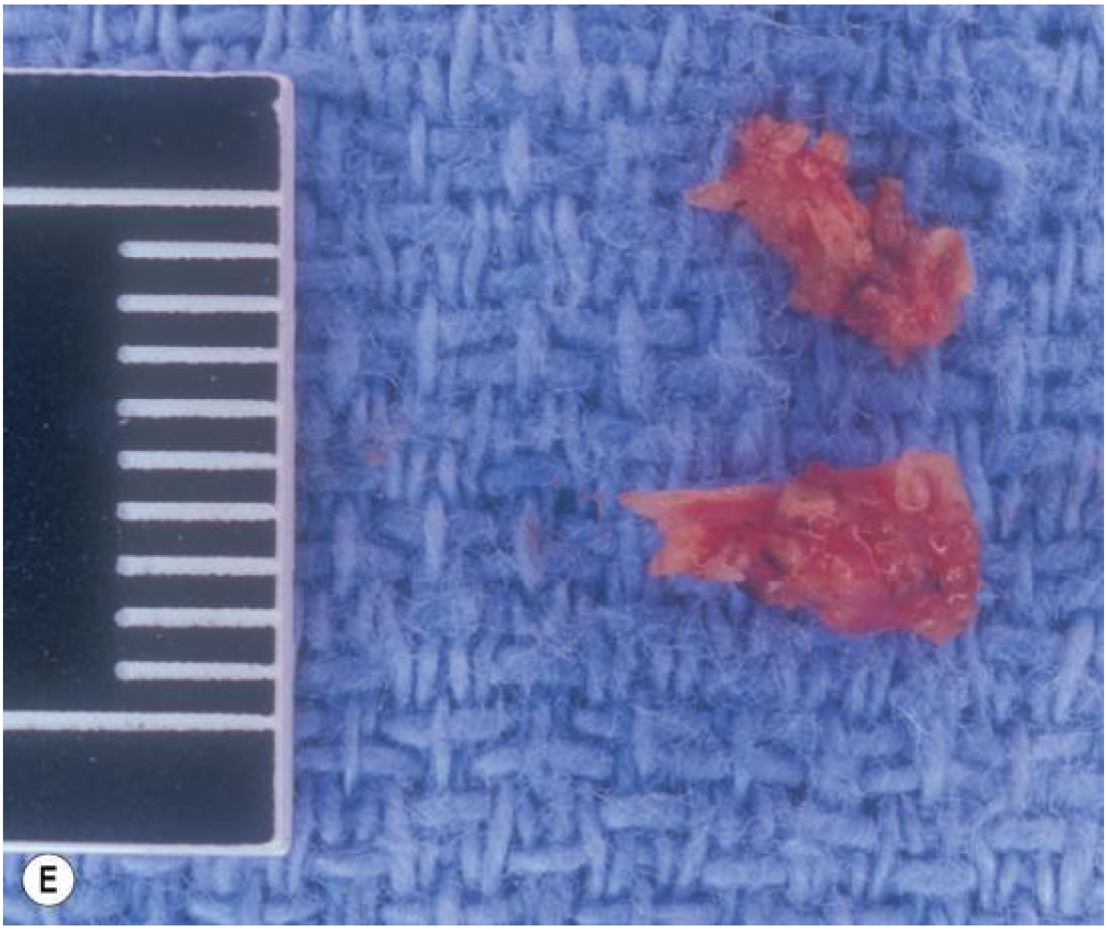

• **FIG. 34.6** Repair of an infected fracture of the body of the right mandible, presented for treatment 2 weeks after the fracture occurred: (**A**) Preoperative radiograph. (**B**) Repair using an acrylic external fixator. (**C**) Two-week follow-up radiograph illustrating pin placement; note that the two teeth involved in the fracture line have been extracted. (**D**) Radiograph obtained 14 weeks postoperatively and following fixator removal, showing a ring sequestrum *(arrow)* at the most caudal pin hole. (**E**) The surgically removed ring sequestrum fragments.

Other potential complications of external pin fixation include pin breakage, pin pull-out, pin loosening, fracture, and secondary dental disease. Pin breakage, pull-out, and loosening are device failures, and are almost always the result of technical error or insufficient patient restriction.[89] Stress points in acrylic connecting bars are created where air has become entrapped in the acrylic mass.[76] Air bubbles can be minimized by maintaining contact of the mixing tip with the extruded acrylic during filling of the tube. Voids also occur within progressively larger-diameter acrylic columns from the heat production and vacuum that is created, a process referred to as vaporization, which increases the porosity of the column and decreases integrity.[38] Therefore although, in general terms, the bending strength of the acrylic column increases with its diameter, there are clearly limitations to simply increasing column diameter size to improve the strength of the fixator.[38] Fractures may develop when fixation pins are placed in osteoporotic bone or when pin selection is inappropriate and exceeds more than 20% of the bone diameter.[84] Tooth roots and their neurovascular supply may be violated during pin placement and can result in periodontal or endodontal complications, nerve injury, and hemorrhage. Tooth impaction may occur if a developing tooth bud is injured.[90] The applied knowledge of cross-sectional anatomy and the use of drill guides and tissue protectors can minimize the risk of iatrogenic injury to nerves and blood vessels.[89] External scarring is unavoidable with this technique.[56]

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CHAPTER 35

Maxillofacial fracture complications

Yoav Bar-Am, Sandra Manfra Marretta

Definitions

Bone sequestrum: A necrotic piece of bone that has become separated from vital bone.
Delayed union: A delayed union is a fracture that takes longer to heal than anticipated when compared with a similar fracture that heals in an uncomplicated way, in a similar patient, treated with a similar technique.[1]
Malocclusion: A condition where opposing teeth or jaws meet in a way that is abnormal for the type of animal, species, or breed.[2]
Malunion: Healed fractures in which anatomic bone alignment was not achieved or maintained during healing.[2]
Nonunion: A nonunion is a fracture that has failed to heal and does not show any further signs of progression toward consolidation.[1]
Oronasal fistula: An abnormal epithelium-lined communication between the oral cavity and the nasal cavity.
Osteomyelitis: An inflammatory process of the bone marrow, cortex, and possibly the periosteum.[3]

Preoperative concerns

Dogs and cats with maxillofacial fracture complications should be evaluated for inadequate nutrition, aspiration pneumonia, secondary rhinitis, and traumatic malocclusion preoperatively.

Therapeutic decision-making

Fracture complications can occur in dogs and cats following traumatic maxillofacial injuries. Accurate preoperative assessment and adherence to basic principles in the management of maxillofacial fractures, and selection of appropriate techniques for repair of these fractures can help minimize complications (see Chapter 27).[3-5]

Diligent postoperative evaluation of maxillofacial fractures is imperative for the early detection of complications to help ensure appropriate and timely management of these complications. Complete evaluation of patients suspected of having maxillofacial fracture complications includes a complete history, physical examination, hematologic and serum biochemical analysis, a thorough oral examination under anesthesia, and radiographic evaluation including dental and thoracic radiographs. Computed tomography (CT) is superior to skull radiographs for evaluation of maxillofacial trauma or complications following fracture repair.[6] Additional imaging modalities may be helpful, including abdominal ultrasound, magnetic resonance imaging (MRI), and scintigraphy.

Maxillofacial fracture complications

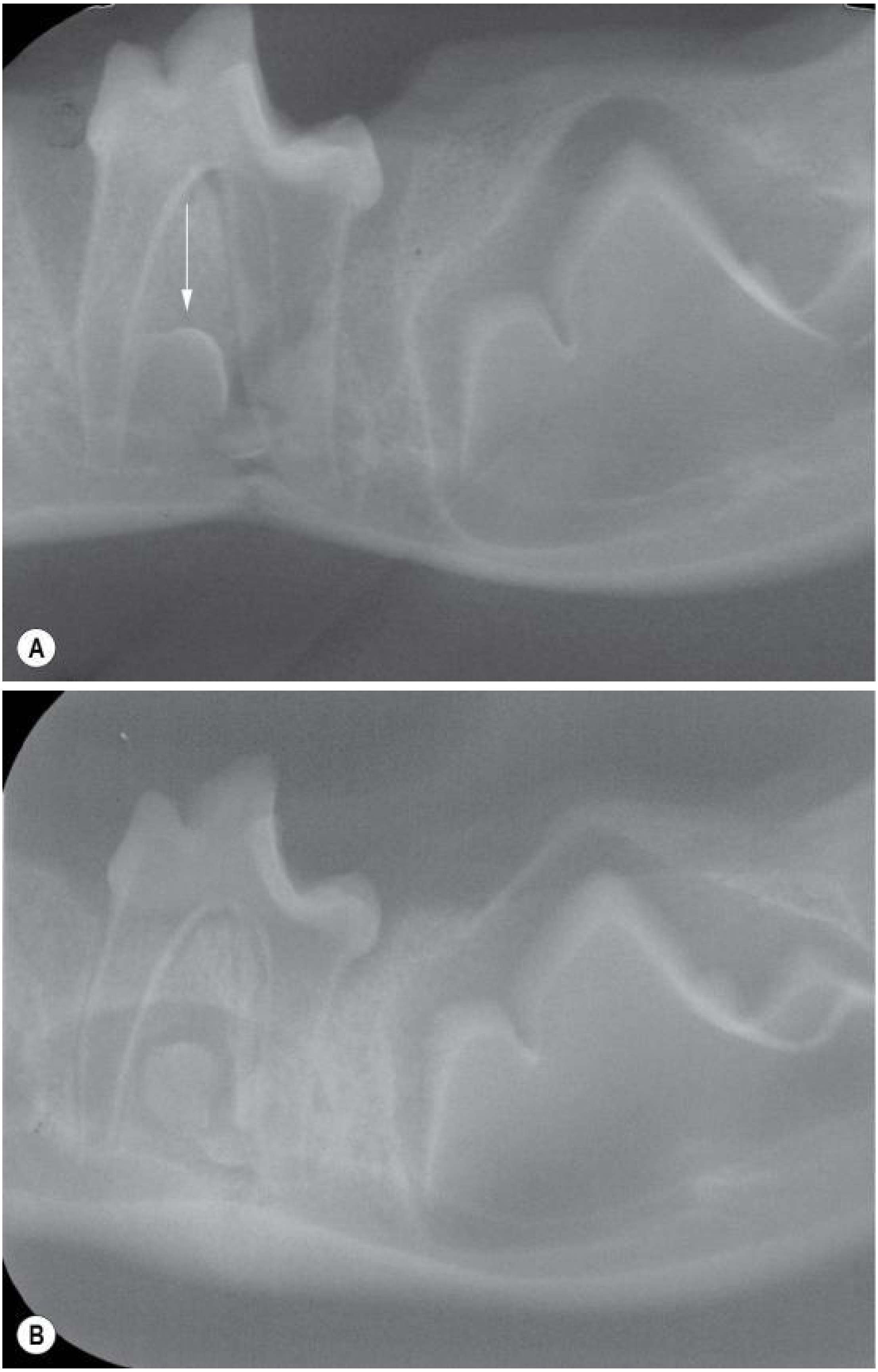
Age-related complications

Juvenile patients

Maxillofacial trauma in juvenile patients involves a unique set of potential complications due to the variety of tissues composing this region and the necessity for synchronous development of the four jaws. Since the jaws grow primarily by intramembranous bone formation, disruption of the periosteum and scarring of soft tissue may cause interference with future growth and development, resulting in facial deformity, asymmetry of dentition, and malocclusions.[7,8]

A conservative and minimally invasive approach rather than an open reduction and stabilization is generally recommended in juvenile patient to minimize these risks. Open reduction of maxillofacial fractures in young patients is generally not required because fractures in these patients are frequently greenstick fractures or are not severely displaced and can be effectively managed with closed reduction; primarily short-term stabilization with tape or nylon muzzles. Furthermore, open reduction in young patients with maxillofacial fractures is more likely to have a negative effect on growth potential because of the additional disruption of the periosteum and soft tissue that is associated with the placement of plates, screws, or wires and is therefore generally not recommended.[7]

Maxillofacial trauma in young dogs and cats can damage developing teeth, which can be associated with a wide variety of potential complications ranging from minor, insignificant issues such as enamel hypoplasia to more significant problems such as failure to erupt, tooth resorption, dentigerous cyst formation, and pulp necrosis.[9] Following maxillofacial trauma in immature dogs and cats, it is important to periodically reevaluate the fracture site for evidence of damage to the developing tooth buds. Periodic radiographic reevaluation of the teeth at, and rostral to, the fracture site can provide important information concerning the status of development. In these cases, radiographs of the contralateral region should also be obtained to help in the detection of potential problems so that appropriate treatment may be instituted as early as possible (Fig. 35.1A–D).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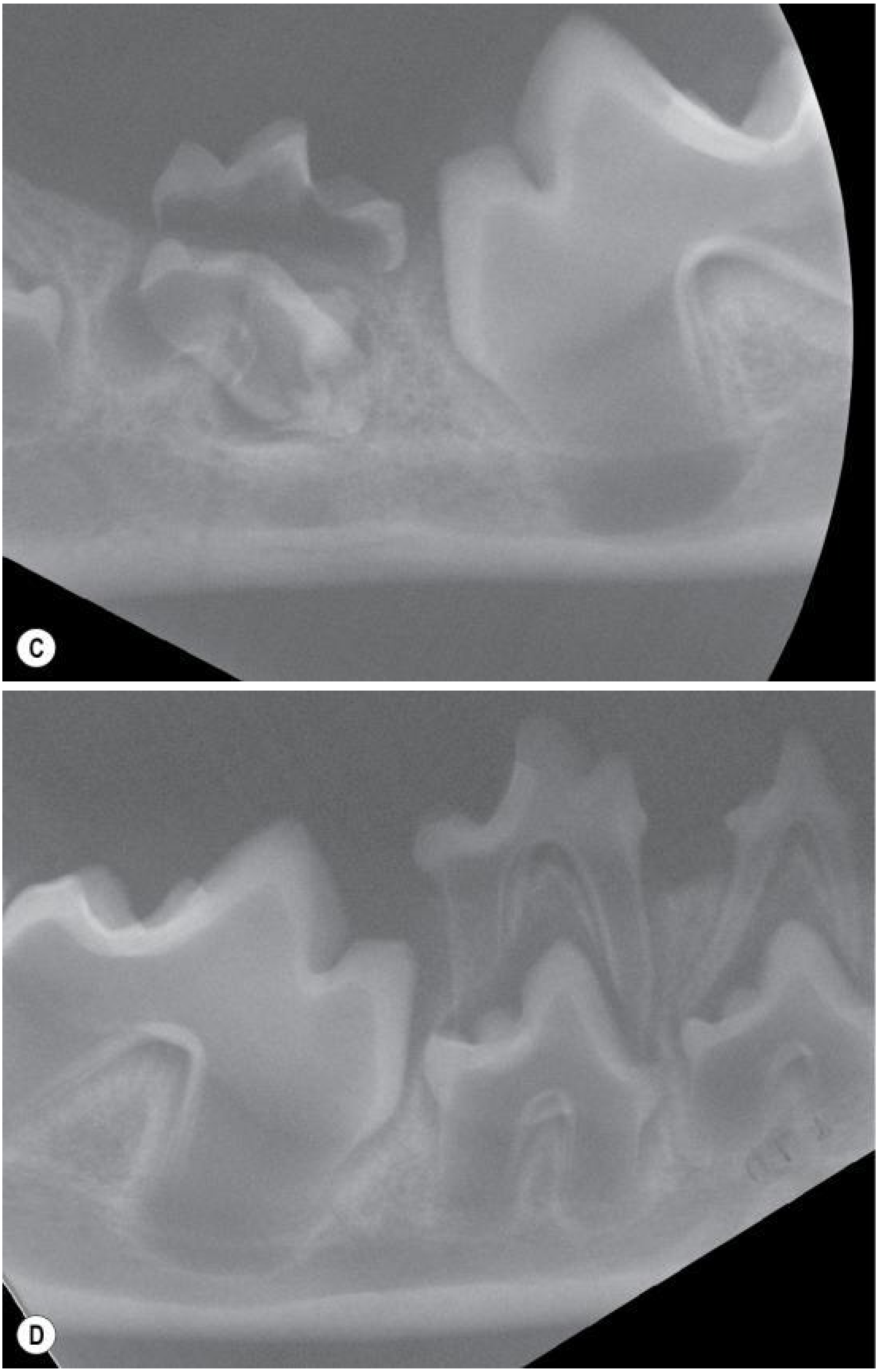


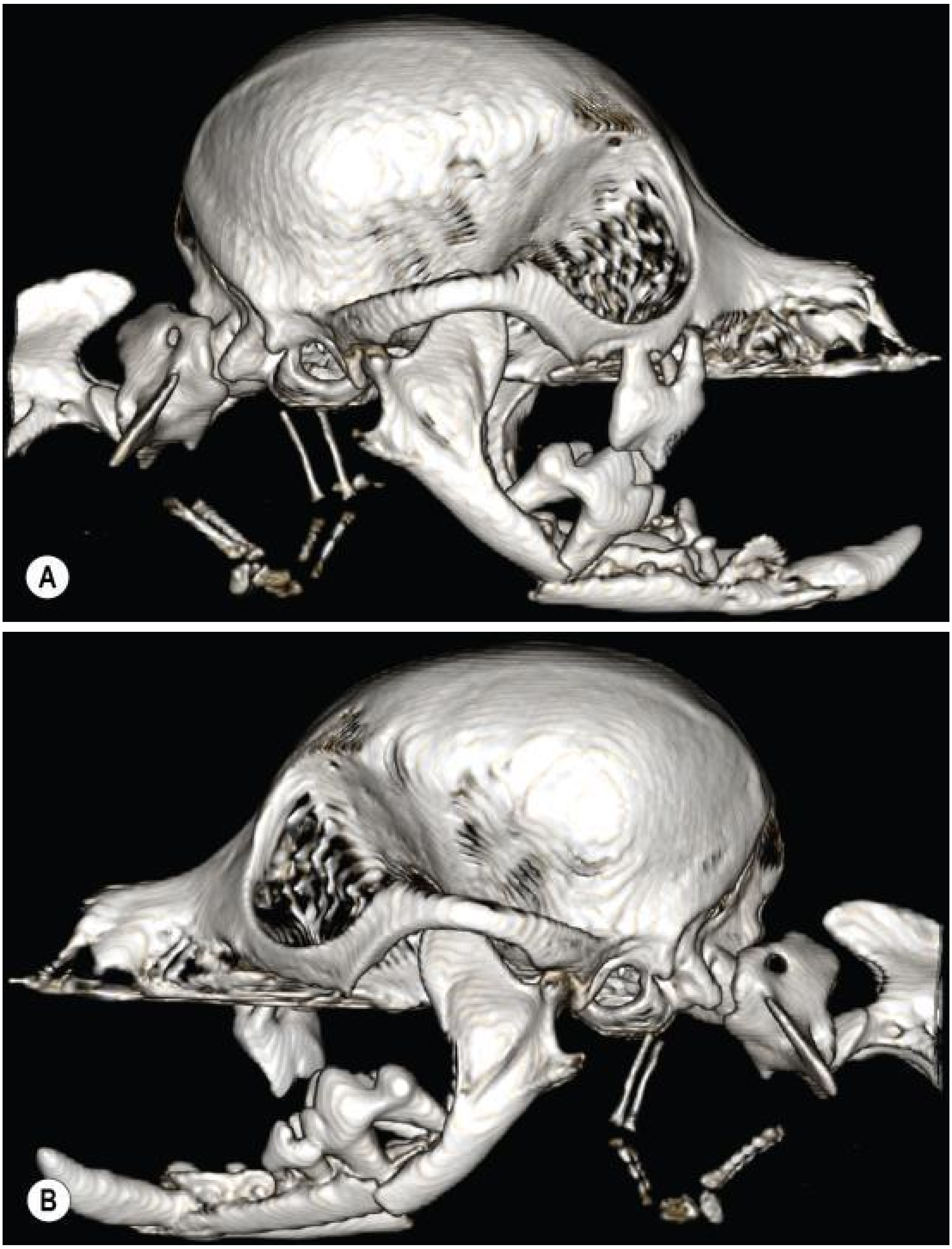
• FIG. 35.1 **(A)** Radiograph of a fractured mandible in the region of the fourth premolar tooth bud *(arrow)* in an 8-week-old puppy. **(B)** Two weeks later there is evidence of new bone formation ventral to the fracture site. **(C)** Two months following the initial traumatic episode a malformed fourth premolar tooth bud can be visualized radiographically. **(D)** The normally developing fourth premolar dental bud is visualized on the contralateral radiograph.

Geriatric patients

Several factors may complicate the management of maxillofacial trauma in geriatric patients.[10] These factors include concurrent systemic diseases, partially or completely edentulous quadrants with associated alveolar bone loss, an increased risk of pathologic fractures secondary to periodontal disease and neoplasia, and an increased incidence of postoperative complications including delayed union or nonunion.

Two major systemic diseases that may complicate the management of maxillofacial trauma in geriatric patients include chronic renal insufficiency and diabetes mellitus. These diseases must be recognized and treated appropriately, and anesthesia protocols must be modified accordingly.

Partially or completely edentulous geriatric small-breed dogs are often affected with severe alveolar bone loss. Pathologic fractures can occur in these patients, most commonly due to severe periodontal bone loss and/or periodontal-endodontal lesions at the mandibular first molar and canine teeth (Fig. 35.2).[8,11] These types of fractures may occur bilaterally following minimal bony stress.[12]



• **FIG. 35.2** **(A)** Right and **(B)** left view of a three-dimensional reconstruction of a computed tomography study of a 14-year-old Chihuahua with bilateral mandibular fractures due to severe periodontal disease at the first molar teeth.

Geriatric patients have an increased risk of delayed union and nonunion following maxillofacial fractures and can be treated similarly to younger patients with these complications. Unsuccessful treatment in these difficult cases may necessitate a salvage procedure such as a partial mandibulectomy.

Complications related to adjacent anatomical structures

Trauma to the inferior alveolar nerve and blood vessels

The anatomic location of the inferior alveolar artery and nerve predisposes these structures to injury during mandibular trauma. Damage to these structures may occur as a result of stretching or laceration during the injury, severance by the jagged edge of a fracture segment, or iatrogenic damage during surgical intervention.[13]

Occasionally, significant hemorrhage may occur as a result of laceration of the inferior alveolar artery, necessitating urgent intervention.

Damage to the inferior alveolar nerve resulting in anesthesia or paresthesia of the lip is a common finding in fractures involving the mandibular body or angle in humans.[14] These sensory deficits would not appear to be clinically significant in animals. A thorough cranial nerve evaluation in maxillofacial trauma patients is indicated to assess their neurological status.

Oronasal fistula

Maxillofacial fractures in which the hard palate is fractured and the integrity of the overlying mucosa is compromised may result in a communication between the oral and nasal cavity (oronasal fistula). Oronasal fistulae may be caused by various types of trauma including dog bites, blunt head trauma, electric shock, gunshot wounds, foreign body perforation, tooth avulsion, and pressure necrosis.[8]

A variety of surgical techniques may be utilized to repair the defect. Small defects in which there is no loss of palatal mucosa may be repaired utilizing a simple interrupted pattern with fine synthetic absorbable monofilament suture material. Large defects can be repaired using a wide variety of techniques including buccal flaps, rotation flaps, advancement flaps, tongue flaps, split palatal U-flaps, and island mucoperiosteal palatal flaps.[8,12,15–17] The technique selected depends on the location of the defect and tissue availability. In general, the technique that provides the largest, tension-free flap is recommended (see Chapter 41).

The most common complication following oronasal fistula repair is dehiscence. Dehiscence will occur if proper healing conditions are not achieved. Suturing the flap under tension is one of the most common reasons for dehiscence. Other reasons are inadequate blood supply to the flap, lack of tissue support, poor debridement, flap design, and tissue handling.[18] In addition, premature return to chewing activity may lead to failure of oronasal fistula repair.

Trauma to the nasal passages and maxillary recesses

The anatomic location of the nasal passages and maxillary recesses predisposes these structures to several potential complications in patients sustaining maxillofacial trauma, with epistaxis and nasal obstruction being frequent observations in patients with maxillary trauma.

The nasal passages and maxillary recesses have a rich blood supply, which often results in epistaxis when this region is traumatized. Nasal hemorrhage secondary to trauma usually resolves spontaneously without therapeutic intervention. Hyperexcitable patients with epistaxis may benefit from the administration of a low dose of a sedative such as acepromazine or dexmedetomidine combined with an opioid analgesic, after careful assessment of the systemic condition of the animal. In rare cases of severe persistent epistaxis, temporary packing of the nasal cavity with petroleum jelly-coated gauze may be necessary.[19] This packing can usually be removed within 2 to 3 days.

Nasal obstruction can be an immediate or long-term complication of trauma to the nasal passages. Acute nasal obstruction in maxillofacial trauma patients may be caused by crushing nasal injuries, hemorrhage, contusion, and edema. Most mesaticephalic and dolichocephalic animals are able to breathe (with some difficulty) through their mouths, even with severe nasal obstruction. By contrast, brachycephalic breeds with acute nasal obstruction often require intensive monitoring and therapeutic intervention. In these patients, supplementation with nasal oxygen, placement in an oxygen chamber, or a temporary tracheostomy may be necessary. Exploration of crushing injuries to the nasal passages to remove fractured turbinates, bone fragments, and devitalized soft tissue has been recommended.[20] Early exploration of these injuries may provide an open airway in a shorter time, reduces infection, eliminates prolonged fetid discharge, and reduces the chance for airway stenosis due to scarring of malaligned tissue.[20] Aggressive exploration in these cases, however, may promote the formation of more scar tissue, resulting in greater upper respiratory stridor long-term.

Persistent unilateral or bilateral nasal mucopurulent or purulent discharge following trauma to this area may result from a bone sequestrum or foreign body such as a lodged tooth or tooth fragment. Exploratory rhinotomy is usually recommended for removal of these objects.

Chronic nasal obstruction following maxillofacial trauma is rare and may be caused by chronic fibrosis of the nasal cavity. Unilateral chronic nasal obstruction is usually well tolerated and does not require treatment in patients that are minimally compromised. Severe bilateral chronic nasal obstruction or nasopharyngeal stenosis may require surgical intervention. Rhinoscopy and CT can help in the localization of the obstruction. Following localization of the lesion, a dorsal or ventral rhinotomy can be utilized to remove obstructing turbinates or nasopharyngeal webs, which may help relieve the chronic airway obstruction. However, the possibility of recurrence of the obstruction is high and may necessitate a permanent tracheostomy in patients with severe recurrent upper respiratory obstruction.[8,21]

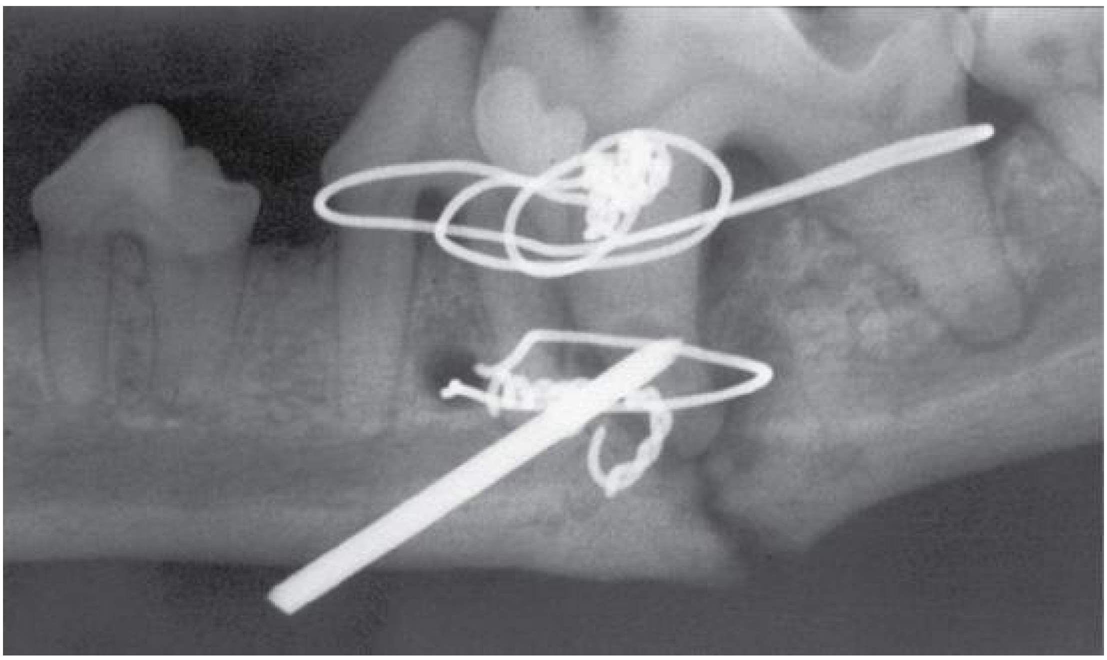
Complications related to implants

Implant exposure, loosening, migration, and implant failure

Implant exposure can occur following internal reduction and fixation of maxillofacial fractures. In an experimental study in which mandibular osteotomies between the third and fourth premolar teeth in dogs were repaired with conventional orthopedic plates and screws, erosion of the alveolar mucosa occurred in 10 of the 15 dogs, with subsequent gingivitis, periodontitis, and severe bone loss.[22] Maxillofacial miniplates are a better choice than orthopedic plates for repair of maxillary and mandibular fractures. In a clinical study in which maxillofacial miniplates were used for repair of mandibular and maxillary fractures in 15 dogs and 3 cats with 11 caudal (junction of the ramus with the mandibular body) and 2 rostral mandibular fractures, 4 maxillary fractures, and 2 zygomatic arch fractures,[23] the only site of soft tissue dehiscence and loss of fixation was at a rostral mandibular fracture, necessitating the performance of a rostral mandibulectomy in that patient. These two studies suggest that there may be an increased risk of implant exposure when bone plates are utilized in the repair of rostral and mid-mandibular body fractures. Implant exposure may also occur on the lingual aspect over sharp screw tips, especially if screws that are slightly too long are used.

When using intraosseous wiring techniques, bending the wire twist over may decrease the tension somewhat, but is usually indicated in order to prevent undue soft tissue irritation and subsequent exposure because of the sharp protruding ends of the wire.

Implant loosening, migration, and failure can occur following internal reduction and fixation of maxillofacial fractures, and typically result from inappropriate implant selection, improper placement, or technical errors. Inadequate tightening of intraosseous wire allows micromotion of the bone fragments and results in resorption of bone (Fig. 35.3).[24] Wires placed in or migrated into a fracture line interfere with the formation of a bridging callus.[24] Fracture healing may occur in the presence of micromotion, but healing will be delayed. When there is significant motion at the fracture site due to implant loosening, migration, or failure, the implant should be tightened or replaced to achieve stability at the fracture site. An external fixation device or an intraoral composite splint may be beneficial in achieving additional stability in these cases.



• **FIG. 35.3** A misplaced pin causing trauma to the mesial root of the first molar tooth. Loose wires have resulted in motion between the bone fragments and resorption of bone. Healing is not occurring because of the diseased tooth in the fracture site and the unstable fixation.

Healing complications

Factors that influence normal healing of maxillofacial fractures include the age of the animal, its systemic health status and immunologic condition, the degree of stability at the fracture site, the size of bony defects at the fracture site, the location of the fracture site, the integrity of soft tissues, the vascular supply at the fracture site, and foreign material located within the fracture site. All of these factors must be evaluated when assessing maxillofacial fracture healing complications. Potential maxillofacial fracture healing complications include delayed union, nonunion, and malunion.

Delayed union and nonunion

A delayed union is a fracture that takes longer to heal than anticipated when compared with a similar fracture that heals in an uncomplicated way, in a similar patient, treated with a similar technique.[1]

A nonunion is a fracture that has failed to heal and does not show any further signs of progression toward consolidation.[1] Every nonunion fracture was a delayed union fracture at one point.

Nonunion fractures have been classified in the Weber-Čech classification as viable (hypervascular) or nonviable (avascular) nonunions.[24,25] Viable nonunions are further subclassified as hypertrophic, slightly hypertrophic, or oligotrophic, depending on the amount of callus present at the fracture site, with hypertrophic nonunions having an abundant hypervascularized callus while oligotrophic nonunions are devoid of visible callus.[24,25] Nonviable nonunions are subclassified as dystrophic, necrotic, defect, or atrophic.[24,25] Dystrophic nonunions have an intermediate fragment that has healed to one fracture fragment but is incapable of bridging the gap to the second major fragment. Necrotic nonunions have avascular or poorly vascularized fragments that eventually die and become sequestra. Defect nonunions are associated with the loss of a large section of bone at the fracture site. Atrophic nonunions may be the final result of the other three types of nonviable nonunions. Oligotrophic, defect, and atrophic nonunions seem to occur most commonly in the mandible.

Causes

There are multiple factors that can negatively influence fracture healing. In general the biological and mechanical environment determine the rate and extent of fracture healing progression.[1]

The major cause of delayed union and nonunion is inadequate fracture stability. Other factors that may contribute to the development of delayed union or nonunion include vascular impairment, large fracture gaps, interposed soft tissues, infection, and inappropriate use of skeletal implants.[24] Teeth in the fracture line can also delay or prevent healing, particularly if they are diseased or loose.[3,26]

Diagnosis

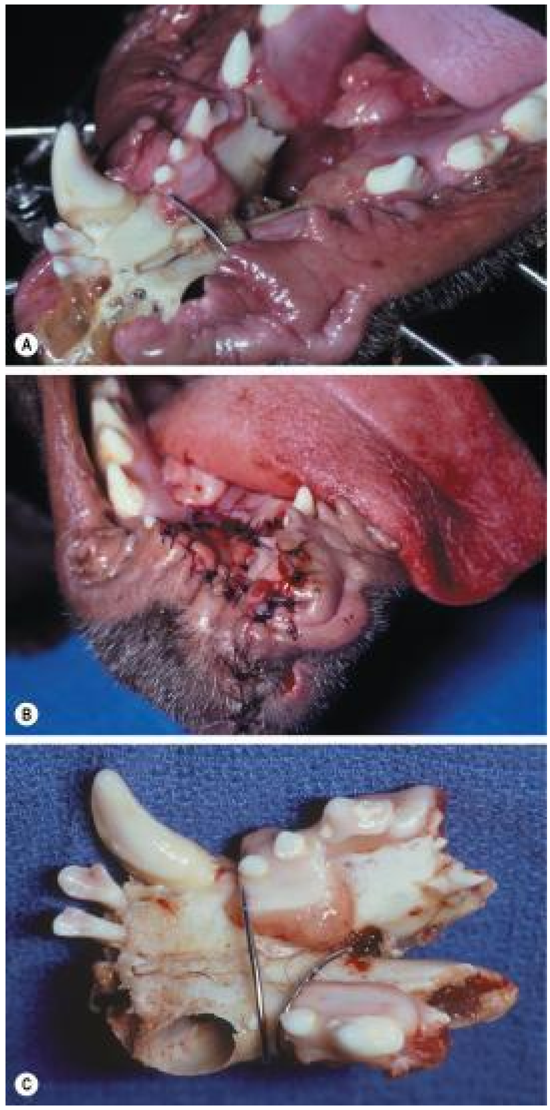
The diagnosis of delayed union requires knowledge of the expected healing rate of the specific bone. Periodic radiographic and clinical examinations are necessary for evaluation of the healing progression. Progressive bone activity, shown by radiographic signs of increasing density of the fracture lines and callus formation, are expected despite the protracted time from fracture repair.

By contrast, nonunion fractures show no radiographic evidence of progression of fracture healing over a period of several months. Radiographic features include a persistent gap at the fracture plane with rounded, well-defined or sclerotic fracture ends. Callus does not bridge the fracture, and there may be displacement of the bone ends. Disuse osteopenia may be noticed.[1]

Treatment

In cases of delayed union, as long as implants remain stable and there are signs of progressive bone activity on sequential radiographs, as evidenced by increasing density of fracture lines, there is no immediate need for additional surgical intervention.[27] When loose or migrating implants are present in delayed union or nonunion, these implants should be removed, the fracture site should be stabilized, and placement of cancellous bone grafts should be considered.[27] Factors other than instability at the fracture site should also be addressed when attempting to prevent or treat delayed union and nonunion.

Vascular impairment can often be prevented by preserving vascularity to the fracture site. Extensive stripping of soft tissue from fracture fragments should be avoided to prevent vascular comprise to the underlying bone. Preservation of the integrity of soft tissue surrounding rostral mandibular fragments is particularly important for bone viability and for the prognosis of fracture union, because revascularization of fracture fragments does not occur across the intraosseous portion of the symphysis, but occurs by formation of a transient extraosseous blood supply.[3,28] When selecting a fixation technique for maxillofacial fracture repair, a technique that is as minimally invasive as possible to achieve appropriate reduction and stabilization should be chosen to reduce the risk of vascular impairment. In cases in which severe vascular impairment results in bone necrosis, thorough debridement of the necrotic bone is necessary. In cases in which the area of bone necrosis is too large, mandibulectomy may be warranted (Fig. 35.4). Large fracture gaps may be managed with external fixation devices or locking plates and placement of a cancellous bone graft in the defect. Alternatively, successful healing of defect nonunions has been reported following internal fixation paired with recombinant human bone morphogenetic protein-2 (rhBMP-2) impregnated in a compression resistant matrix (see Chapter 53).[28,29] Care should be taken not to interpose soft tissue in these fracture gaps, since this will delay or prevent fracture healing. It is also important to remove diseased teeth from a fracture site prior to fixation, since diseased teeth in the fracture site may result in a delayed union or nonunion.[3]



• **FIG. 35.4** **(A)** Bone necrosis of the rostral mandible 6 days following mandibular fracture. **(B)** A rostral mandibulectomy has been performed to remove the necrotic bone. **(C)** The necrotic bone fragment.

Malunion

Malunions are healed fractures in which anatomical bone alignment was not achieved or maintained during healing. Malunion is a significant complication that may be associated with maxillofacial fractures.

Causes

Inadequate stabilization of the fracture, either due to improper technique selection or poor technique execution, is the most common cause of malunion. Failure to completely counteract the forces acting on the fracture or inadequate initial fracture reduction will lead to deformity that will result in malunion.[1,30]

Diagnosis

Malunions are classified as functional if there is a small axial deformity with no functional impairment and no effect on joint function, or nonfunctional if there is more severe deformity and joint function impairment. They can be further described as overriding malunions, where the bone segments have slid beside each other while maintaining axial alignment; or angular malunions, where there is significant deviation of axial alignment.[30] Careful attention to proper dental occlusion during fracture fixation can help prevent malunion. Utilization of pharyngotomy or transmylohyoid endotracheal intubation and temporary maxillomandibular fixation during fracture fixation will help prevent inaccurate reduction and subsequent malunion.

Severe malunion that results in significantly impaired function, including inability to close the mouth or a severe deformity, requires surgical intervention. Treatment of severe malunion may include corrective osteotomy or judicious selective extraction of maloccluding teeth caused by the malunion.

Infectious complications

Wound infection

Wound infection can occur as a complication of maxillofacial fractures. Although the oral cavity is a contaminated environment in which aseptic surgical conditions are difficult to achieve, the rate of wound infection following maxillofacial trauma is significantly less than other areas of the body. The well-vascularized tissue in this area may confer an advantage on the host's ability to prevent an infection in the presence of large numbers of bacteria.[31]

Local wound changes that indicate a developing infection include redness, increased heat, pain, and localized edema.[32] An elevation in body temperature may also be present. Progression of the wound infection results in purulent discharge from the incision site.

Management of wound infections following maxillofacial trauma includes several treatment modalities. Cellulitis should be treated with broad-spectrum antibiotics. Once a localized abscess becomes evident, the wound should be prepared for aseptic surgery and the sutures overlying the infected wound should be removed as needed to provide adequate drainage. Samples should be obtained for aerobic and anaerobic culture and susceptibility testing. In severe wound infections the wound should be thoroughly but judiciously debrided, flushed with sterile saline, and closed. A broad-spectrum antibiotic should be administered and adjusted when results of bacterial culture and susceptibility testing are available.

Osteomyelitis and osteonecrosis

Osteomyelitis or osteonecrosis with bone sequestra following jaw fracture repair may be associated with diseased teeth or root tips, exposed alveolar bone, or operator-induced osseous necrosis.[3]

In humans, the incidence of mandibular osteomyelitis is significantly greater than osteomyelitis of the maxilla following maxillofacial trauma. Clinical experience indicates that this is also true in small animals. The reason for this disparity may be that the endochondral bone of the mandible is structurally similar to the long bones of the body, which are more susceptible to osteomyelitis.[32] The intramembranous bone of the maxilla has less medullary tissue, thinner cortical plates, and a more extensive blood supply than the mandible.[32] These factors all permit an infection in the maxilla to be more easily disseminated into the surrounding tissue than infection in the mandible, thereby helping reduce maxillary osteomyelitis.

Risk factors associated with the development of posttraumatic osteomyelitis following mandibular fractures have been reported in humans (Box 35.1).[32] These risk factors can be applied to small animal patients.

• **BOX 35.1**

Risk Factors for Developing Osteomyelitis

- Ineffective fixation, reduction, and immobilization
- Delay in treatment
- Delay in administration of antibiotic therapy or inappropriate antibiotic therapy
- More than 50% of a tooth root exposed in a fracture
- Premature removal of fixation devices
- Devitalized segments of bone in the fracture site due to excessive periosteal stripping, overzealous use of intraosseous wiring, or overheating of the bone by burs
- Decreased host resistance or increased susceptibility to infection

From Lieblich SE. Infection in the patient with maxillofacial trauma. In: Fonseca RJ, Walker RV, Barber HD, et al, eds. *Oral and Maxillofacial Trauma.* 4th ed. Philadelphia, PA: Elsevier; 2013. p. 790–807.

The treatment of osteomyelitis associated with maxillofacial fractures includes both a surgical and medical approach. The fracture site should be thoroughly assessed for adequate stability. Removal of implants should be considered because they may serve as contamination and biofilm nidi and elicit a foreign body reaction. Further, if implants are not providing adequate stability, they should be removed. External fixation devices and intraoral splints are often helpful in providing stabilization without significant loss of blood supply to the surrounding soft tissues and bone, thus reducing morbidity.

In addition to stabilizing the fracture, it is also important to remove all nonvital tissue, including bone sequestra and diseased or loose teeth (Fig. 34.5). Specimens of infected bone should be obtained for aerobic and anaerobic bacterial culture and susceptibility testing. Broad-spectrum bactericidal antibiotic therapy should be instituted immediately, and can be modified if necessary based on culture results and clinical response. Appropriate antibiotics should be administered for a minimum of 4–6 weeks.

• **FIG. 35.5** Mandibular first molar tooth and bone sequestrum removed from a nonhealing painful mandibular fracture site 6 weeks after repair. Healing occurred rapidly following their removal and stabilization with a tape muzzle.

Complications related to teeth

Several maxillofacial fracture complications are related to the teeth, including malocclusion and dental trauma. Recognition of these complications early in the course of treatment will permit timely management of these problems and decrease the posttraumatic recovery period.

Malocclusion

Malocclusion can be a serious postoperative complication in jaw fracture management. Failure to properly align fracture segments, particularly in caudal mandibular fractures, results in significant deviation of the rostral mandibular segment. In severe cases, the patient may not be able to properly close the mouth because of traumatically occluding maxillary and mandibular premolar and molar teeth. If occlusion is not carefully assessed during fracture fixation, serious malocclusion may not be recognized until the immediate postoperative recovery period. In order to prevent this complication, it is recommended to place a pharyngotomy or transmylohyoid endotracheal tube and perform temporary maxillomandibular fixation (see

Chapter 27). This way the jaws are kept in proper occlusion, and stabilizing the bones while in temporary maxillomandibular fixation will result in good alignment and occlusion.

In less severe cases of malocclusion secondary to maxillofacial fracture repair, there may be traumatic occlusion between a few teeth. These cases may be treated with crown amputation and endodontic therapy or by selective extraction of maloccluding teeth. Selective extraction of maloccluding teeth can permit the patient to close the mouth postoperatively in poorly reduced fracture fixations, but is considered a significant compromise for poor surgical technique.[8]

Dental pathology

Maxillofacial fracture complications that may be related to dental pathology include periodontal, endodontal, and iatrogenic pathologic conditions.

As described above, preexisting severe periodontal disease may predispose geriatric small-breed dogs to pathologic fractures.[3,11] Severe endodontal pathology can potentially predispose patients to pathologic mandibular fractures; however, endodontal pathology of this magnitude is rarely encountered.

Pathologic fractures through severe periodontal or endodontal lesions can be very difficult to treat because of poor bone quality. Traditionally, treatment has consisted of extraction of the diseased tooth, debridement and flushing of the fracture site, placement of a cancellous bone graft in the alveolus of the fracture, closure of the gingiva over the alveolus, and application of a tape muzzle until a fibrous union occurs. The use of an external fixation device or bone plate in conjunction with a cancellous bone graft and antibiotics should be considered very carefully, because poor bone quality in these cases predisposes to implant failure. The use of locking plates and rhBMP-2 impregnated in a compression-esistant matrix may allow better healing than the traditional approach to these difficult cases (see Chapter 53). In all cases of pathologic fracture, a broad-spectrum antibiotic should be administered perioperatively and for several weeks postoperatively.

A salvage procedure involving unilateral or bilateral segmental mandibulectomy with bilateral advancement of the commissures of the lips has been described to manage unilateral or bilateral pathologic fractures through the alveoli of the mandibular first molar teeth.[8] (Fig. 35.6A–F). These procedures should be reserved for severe cases in which bony union is highly unlikely.

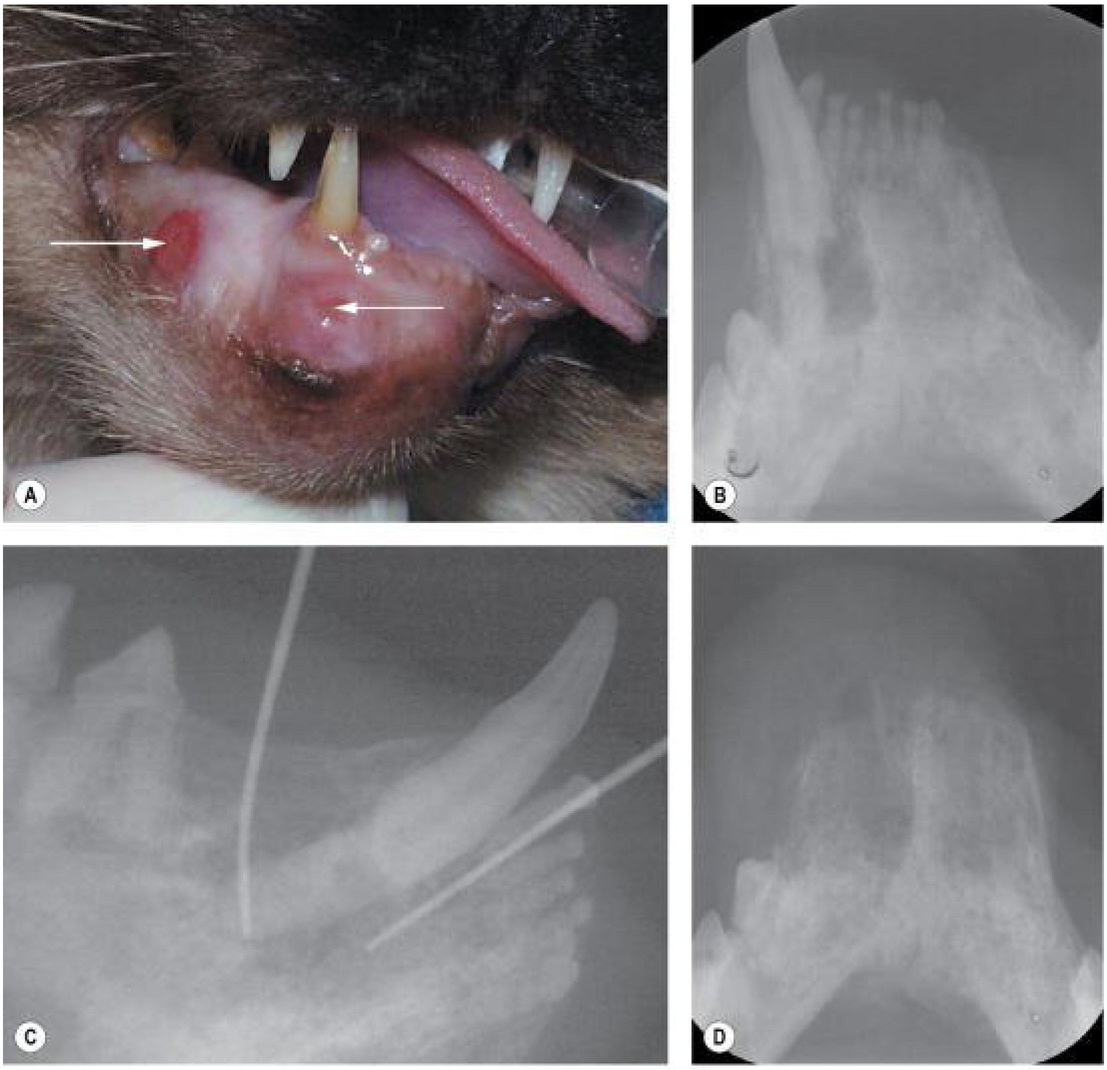
• FIG. 35.6 **(A)** A 14-year-old dog with bilateral mandibular fractures through deep periodontal pockets in the region of the first molar teeth. **(B)** Open fracture site in the region of the extracted first molar tooth. **(C)** The sharp ends of the fracture fragments are removed with a rongeur prior to flushing and closure of the gingiva. **(D)** Bilateral advancement of the commissures of the lips is achieved by excising the lip margins to the level of the canine teeth and suturing the oral mucosa and skin in two layers. **(E)** Postoperative view of bilateral cheiloplasty. **(F)** Lateral view of bilateral cheiloplasty.

Prognostic factors affecting teeth in the line of mandibular fractures have been previously reviewed.[26] The decision to conserve or extract a tooth located in a fracture site should be based on multiple criteria. Timing is a critical factor, and if treatment can be initiated early, the teeth can often be preserved. Removal of teeth from a fracture line can adversely affect the operator's ability to achieve anatomic reduction and fracture stability; therefore teeth should be conserved if possible. Guidelines for the decision-making process concerning the management of teeth in fracture sites have previously been reviewed (see Chapter 27).[26]

The long-term prognosis for teeth affected by maxillofacial fractures depends on the location of the fracture line in relationship to the periodontal ligament. The fracture line with the most guarded long-term dental prognosis is a fracture that extends from the gingival margin along the root surface to the apex of the tooth. This fracture line creates a communication between the apical area of the tooth and the oral cavity. It has been recommended that a tooth associated with this type of fracture be either extracted in toto or hemisectioned, with extraction of the root in the fracture line and endodontic treatment of the remaining root.[26]

Maintenance of healthy teeth in a fracture line can help reduce the frequency of postoperative complications and help facilitate anatomic reduction of the fracture. However, it is important to monitor for evidence of endodontal and periodontal lesions during and following fracture healing. This monitoring process should include periodontal probing and radiographic evaluation. Postoperative complications should be treated with endodontic therapy, periodontal therapy, hemisection and endodontic therapy, or extraction accordingly.

Iatrogenic dental lesions may be associated with maxillofacial fracture repair. These injuries are most often associated with placement of screws during application of bone plates and inappropriately placed pins during the application of external fixation devices (Figs. 35.7 and 35.8). In an experimental study where osteotomies of the body of the mandible in 15 dogs were repaired with plates and screws, 32.5% of the screws were found to have caused pulpal damage, suggesting that it is difficult to place bone plates on the canine mandible without traumatizing the teeth.[22] It is also difficult to place an external fixation device at the rostral mandible without traumatizing the teeth.



• **FIG. 35.7** **(A)** Two draining tracts *(arrows)* that have been present for 7 years following repair of a rostral mandibular fracture. **(B)** Occlusal radiograph showing severe osteolysis at the right mandibular canine tooth. **(C)** Lateral radiograph showing gutta-percha points placed in the draining tracts. **(D)** Postoperative radiograph following extraction of the canine and all incisor teeth; note the moth-eaten appearance of the rostral mandible, suggestive of osteomyelitis.

Trauma to the pulp or periapical tissues with either screws or pins will result in endodontic lesions necessitating removal of the implant and either endodontic therapy or extraction of the iatrogenically traumatized tooth (Fig. 35.8).

• **FIG. 35.8** Teeth extracted from a dog because of a 3-year history of cutaneous draining tracts located ventral to the apices of the canine teeth following repair of a rostral mandibular fracture with an external fixation device. Note the holes in the roots of both mandibular canine teeth.

Complications associated with extensive callus formation

Extensive callus formation following maxillofacial trauma may result in an inability to open the mouth. This inability following maxillofacial fractures may be caused by excessive callus formation around the temporomandibular joint or between the zygomatic arch and the coronoid process of the mandible. CT is required to identify the source of the ankylosis or pseudoankylosis. Adhesions between the zygomatic arch and the coronoid process should be treated with surgical removal of part of the zygomatic arch and/or coronoidectomy (see Chapter 38). Postoperative physical therapy and antiinflammatory drugs may help decrease the recurrence of clinical signs of ankylosis or pseudoankylosis.

Postoperative care and assessment

Diligent postoperative care and assessment are important parts of the management of maxillofacial complications. Maxillofacial trauma patients may develop multiple complications.

Early recognition of these problems by frequent oral examinations, serial dental radiographic studies, and other imaging modalities such as CT will help facilitate early detection of maxillofacial fracture complications.

Immediately following maxillofacial fracture repair, patients should be evaluated for malocclusion. Depending on the severity of the complication, the temperament of the patient, and the compliance of the owners, the use of a tape or nylon muzzle and/or an Elizabethan collar should be considered.

Patients should be monitored for signs of infection in the immediate postoperative period and then as needed, based on clinical signs, since infections secondary to maxillofacial complications can occur. Ideally, maxillofacial fractures should be reevaluated with CT 6 to 8 weeks postoperatively, and later if needed to assess proper healing.

Prognosis

The prognosis for most maxillofacial fracture complications is good to excellent. The prognosis for return to normal function is guarded for geriatric patients with severe bone loss, in patients with large oronasal fistulae, in patients with bilateral nasal obstruction, and in patients with established skeletal malocclusion. Selection and proper application of appropriate fixation techniques can help minimize complications. Early detection of complications and timely administration of appropriate treatment will also improve the prognosis in animals with maxillofacial trauma.

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SECTION 7:

Temporomandibular Joint Surgery

OUTLINE

CHAPTER 36

Temporomandibular joint dysplasia

Boaz Arzi, Gary C. Lantz

Definitions

Dysplasia is an abnormal growth and/or development of tissue.[1] Temporomandibular joint (TMJ) dysplasia is an infrequently reported condition in dogs and cats that results in excessive joint laxity and subluxation or luxation and subsequent lateral and/or rostral shifting of the mandibles, which allows ventrolateral displacement and abnormal contact of the coronoid process with the adjacent zygomatic arch at the ipsilateral side of the TMJ dysplasia. Dysplasia of the TMJ may involve abnormalities of the bone and/or soft tissues.[2] The resulting clinical findings are intermittent episodes of inability to voluntarily close the mouth, known as open-mouth locking, or joint sensitivity without locking episodes.

Background

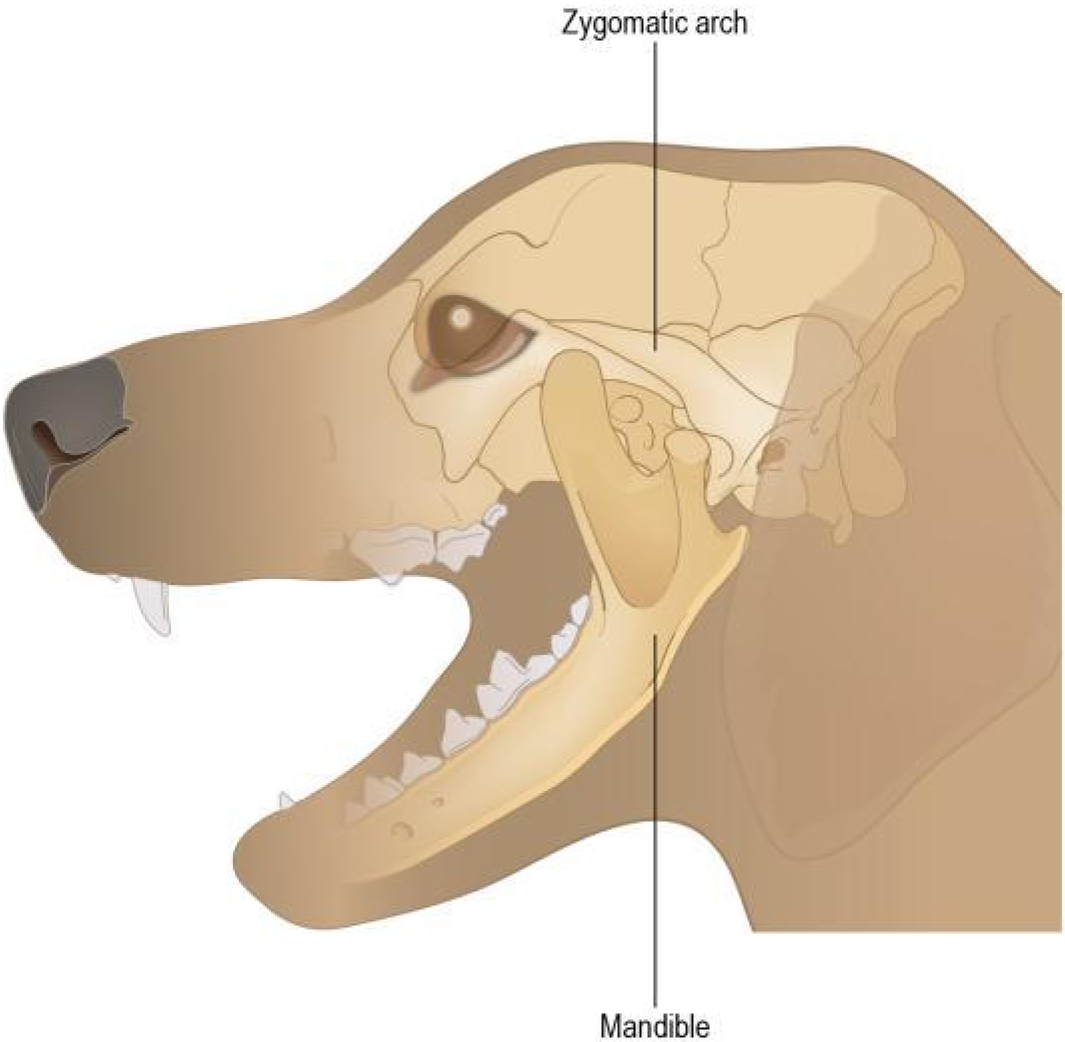
TMJ dysplasia and its surgical correction by partial zygomatic arch resection was originally reported in an Irish setter by Stewart, Baker, and Lee in 1975.[3] Robbins and Grandage reported the condition and similar surgical correction in two basset hounds and proposed a mechanism of open-jaw locking.[4] These and subsequent reports suggest a breed predisposition toward basset hounds, Irish setters, cavalier King Charles spaniels, and cocker spaniels, and the condition has been reported in several cat breeds as well.[2,5,6–11] Furthermore, in dogs, the condition appears to be more prevalent in chondrodystrophic and brachycephalic breeds, which may point to a genetic component in the etiology of TMJ dysplasia. In addition, radiographic evidence of TMJ dysplasia in six related cocker spaniels supported a genetic component.[11] Genetic influence might also play a role in cats with TMJ dysplasia, as most cats reported in the literature had brachycephalic skull conformation.[6] Open-mouth jaw locking has also been reported, possibly resulting from malocclusion-induced TMJ soft tissue laxity, symphyseal laxity, and head trauma.[11,12,13]

Functional anatomy of the temporomandibular joint[14,15,16]

The TMJ is a synovial joint. The head of the mandible *(caput mandibulae)* on the condylar process *(processus condylaris)* articulates with the mandibular fossa *(fossa mandibularis)* of the squamous part of the temporal bone. A fibrocartilaginous articular disk separates the fossa and articular surface of the head of the mandible into two noncommunicating compartments. The disk attaches laterally to the head and medially to the temporal bone by ligamentous extensions. Capsular attachments to the disk are found circumferentially. The lateral aspect of the joint capsule is strengthened by the lateral temporomandibular ligament. This ligament originates from the caudoventral aspect of the zygomatic arch and inserts on the lateral aspect of the condylar process and retroarticular process. Each mandible rotates about a common transverse axis that passes through the center of each condylar process. The long axis of each condylar process is, generally, nearly parallel to the transverse axis. However, TMJ conformation differs between breeds, with small and mid-sized dogs having mandibular fossae that are relatively shallow with less prominent retroarticular processes and irregularly shaped condylar processes.[3,15] However, in large-breed dogs, the TMJ is more congruent with deeper mandibular fossae, prominent retroarticular processes, and a relatively more uniform condylar process. Furthermore, the TMJ shape varies significantly within the breeds and with skull size.[16] The lateral aspect of the mandibular head moves in a rostral and ventral arc during mouth opening. At maximal jaw opening, the joint capsule and lateral ligament are stretched and tense. The masseter, temporalis, and pterygoid muscles hold the condylar process in tight congruity with the fossa. There is a small amount of lateral movement that occurs during mastication, which is limited by the TMJ ligaments and normal occlusal relationship of the caudal maxillary and mandibular teeth.

Pathophysiology

TMJ abnormalities may be unilateral or bilateral, and include a shallow mandibular fossa, flattened mandibular head, abnormal obliquity of the mandibular head and fossa, and joint capsule and ligamentous laxity.[2,17,18] A large angle between the axis of rotation and long axis of the mandibular head suggests an abnormal obliquity of the TMJ, which results in excessive movement of the lateral aspect of the condylar process.[4] Stretching of the lateral ligament and capsule occurs. The joint incongruity and soft tissue laxity results in subluxation of the involved joint and contralateral shifting on the mandibles.[4] The ipsilateral coronoid process contacts the ventral aspect of the adjacent zygomatic arch or moves lateral to the arch. The resulting mechanical interference with the coronoid process motion prevents mouth closure (Fig. 36.1). Variable severity of TMJ dysplasia apparently occurs, which may account for the variable age of onset, frequency, and duration of open-mouth locking episodes, and inconsistent radiographic findings.[2] The lax TMJ may allow rostral displacement of the coronoid process(es) on the same side such that the rostrodorsal aspect of the process contacts the junction area of the zygomatic and maxillary bones when the mouth is opened. In more long-standing situations, due to the abnormal contact of the coronoid process with the zygomatic arch, periosteal reaction forms, mostly at the ventral aspect of the zygomatic arch, further exacerbating the severity and frequency of open-mouth jaw-locking episodes (Fig. 36.2). It is possible that these clinical signs may be due to an abnormally tall coronoid process with or without concurrent TMJ dysplasia.



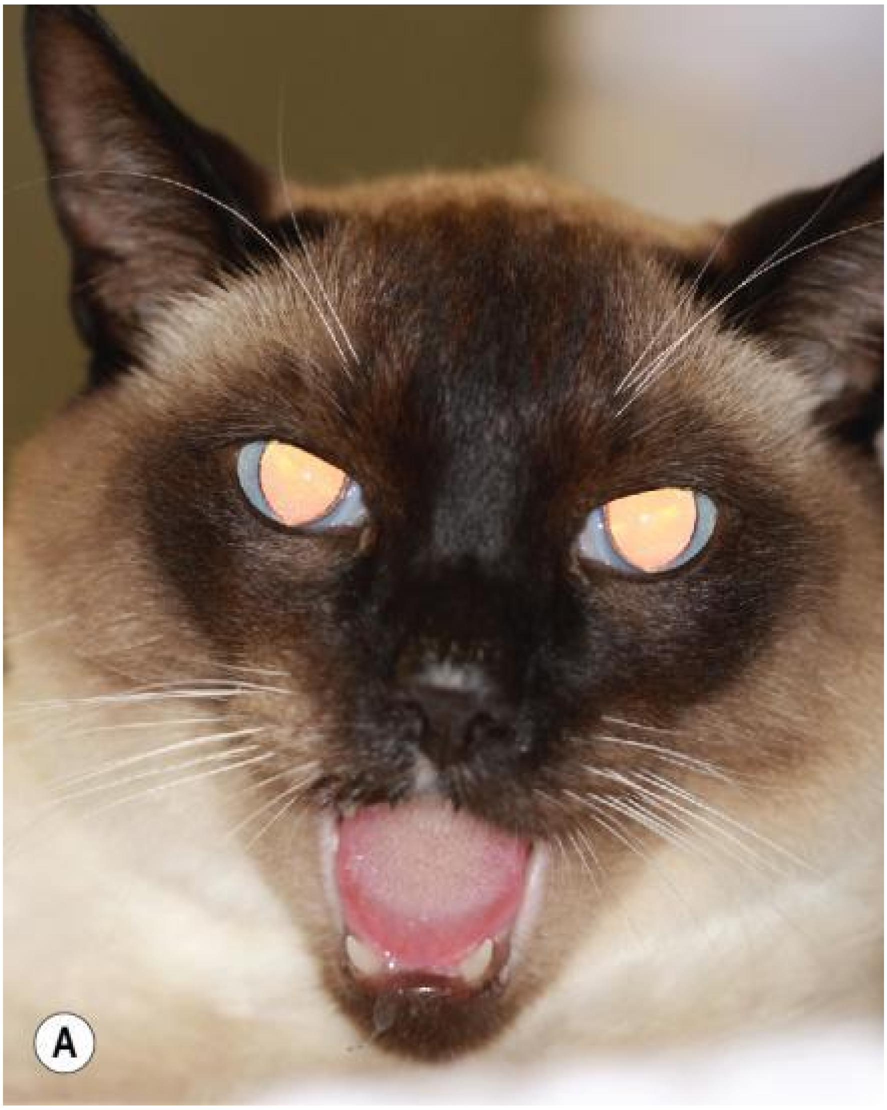
• FIG. 36.1 The coronoid process is displaced lateral to the zygomatic arch as a result of a dysplastic contralateral temporomandibular joint. The lower jaw is locked in an open position.

• FIG. 36.2 Periosteal reaction at the ventral aspect of the zygomatic arch and at the tip of the coronoid process due to long-standing abnormal contact of the coronoid process with the zygomatic arch.

Clinical presentation

Signalment and history

Age of onset ranges from approximately 6 months to 5 years. However, late onset of open-mouth jaw locking secondary to TMJ dysplasia is possible. The initial observation of clinical signs often starts in young adults.[2-12] Observed locking episodes may occur multiple times daily or occasionally over several weeks or months (Fig. 36.3A and B). Most dogs will vocalize, shake the head, paw at the muzzle and rub the muzzle on the ground until the locking is spontaneously reduced. Cats may seek seclusion. Most locking episodes are of short duration, self-correcting by spontaneous reduction. This reduction occurs by opening the mouth wider, thereby allowing the coronoid process to disengage the zygomatic arch. Failure of spontaneous reduction and/or frequent locking episodes results in the client seeking medical care for the animal. If a history of head trauma is reported, the locking may be due to a healed depressed fracture of the zygomatic arch that interferes with the movement of the coronoid process and produces locking episodes that mimic those caused by TMJ dysplasia.[2] Slow, deliberate mastication and a preference change from hard to soft food may be reported, and may be caused by TMJ sensitivity secondary to degenerative joint disease and/or from contact of the rostrodorsal aspect of the coronoid process with the zygomatic and maxillary bone junction area.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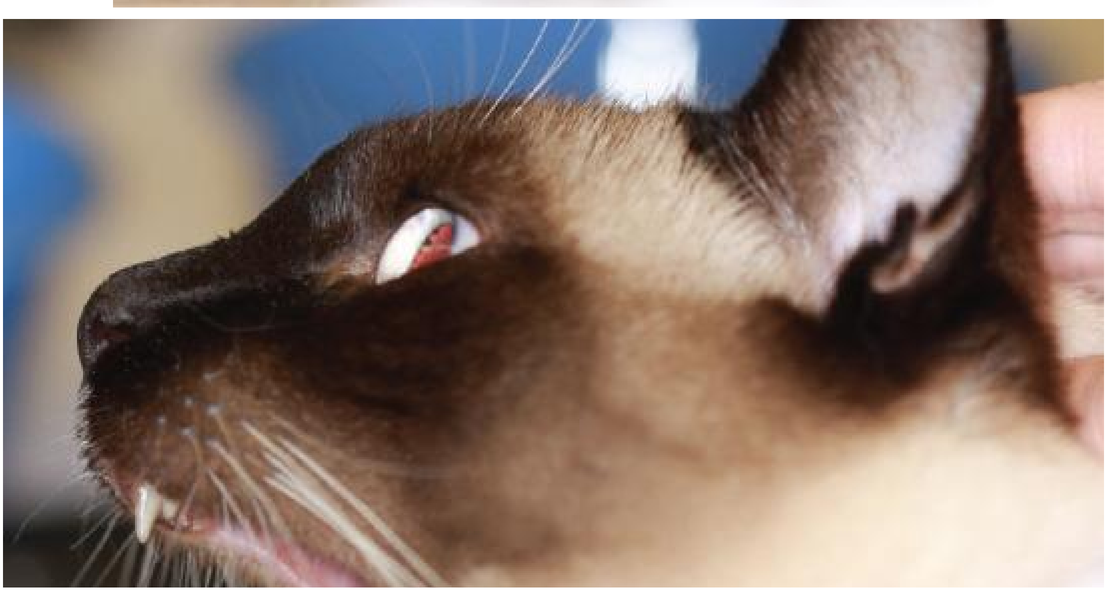


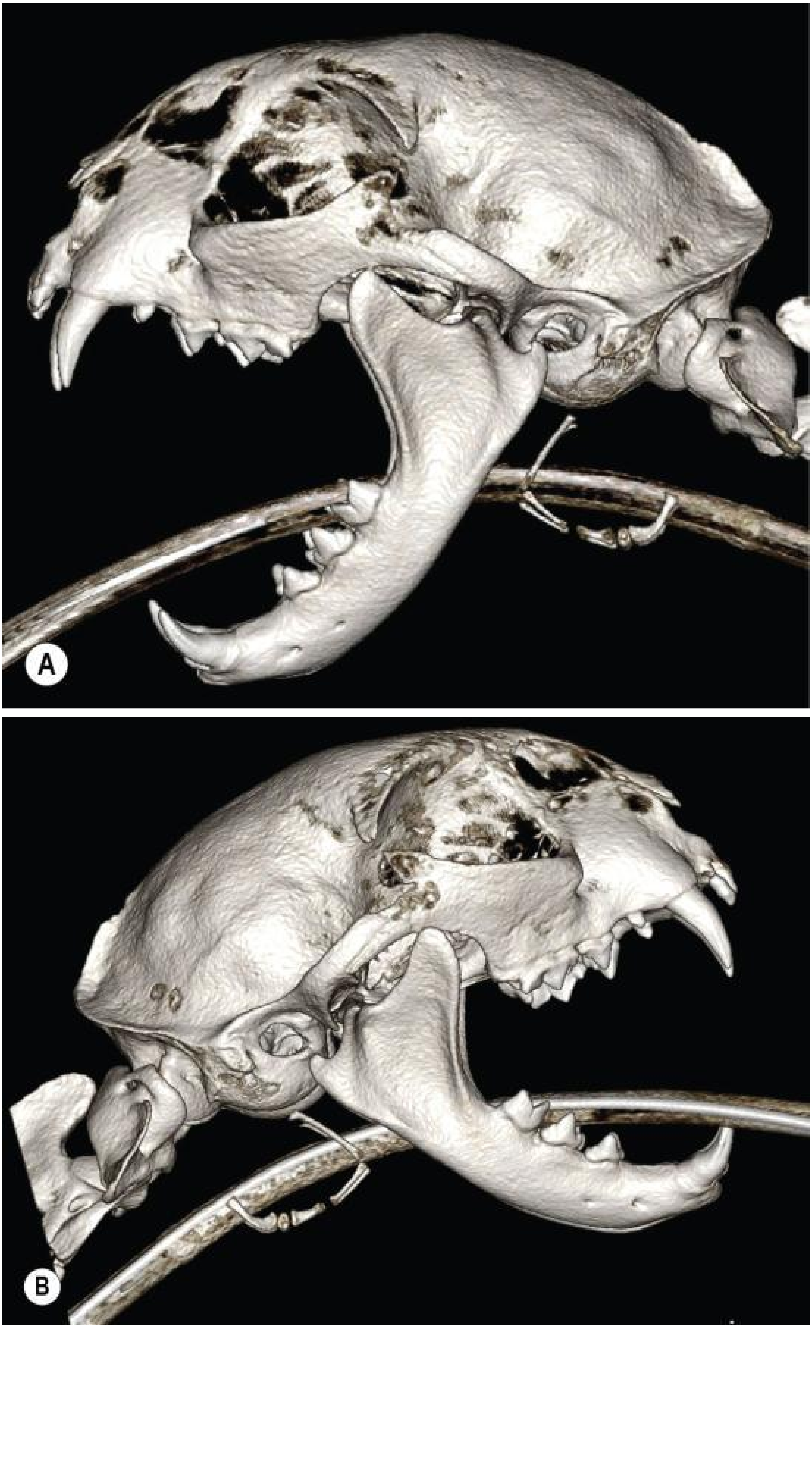
• **FIG. 36.3** The clinical signs observed during the open-mouth locking episodes in a cat in rostral **(A)** and left lateral view **(B)**.

Physical examination

Diagnosis of TMJ dysplasia and the resultant open-mouth jaw locking relies heavily on physical examination and manipulation of the mandibles under general anesthesia. Examination of a patient that has spontaneously reduced the locked jaw may reveal no abnormalities. Resistance to head palpation and manipulation of the lower jaw may be noted. Neurological evaluation of the cranial nerves typically yields no abnormalities. Chronic oral sensitivity may result in visible temporal muscle disuse atrophy.[5] Examination during an open-mouth lock episode shows an open mouth and a patient very resistant to manipulation of the lower jaw. A palpable and usually visible bulge on the side of the face is present when the coronoid process is displaced ventrally or laterally to the zygomatic arch. The lower jaw is shifted toward the side where the lock has occurred and is tilted such that the mandibular body on the locked side is displaced slightly ventrally compared to the opposite mandibular body.

Manipulation of the mandibles

Open-mouth locking can usually be reproduced by manipulation of the lower jaw.[2] After induction of general anesthesia and endotracheal intubation, the patient is placed in sternal recumbency so that both sides can be examined. The mouth is widely opened to see whether the lower jaw tends to shift toward one side. Laterally directed digital pressure is placed on the medial aspect of the right coronoid process and the jaw rotated counterclockwise to lower the right coronoid process ventral to the right zygomatic arch as the mouth is widely opened. The manipulation is then repeated on the left side and with clockwise rotation of the mandible. Clinical experience suggests that successful displacement of the coronoid process to engage the ventral or lateral aspect of the adjacent zygomatic arch and create the lock on one side indicates laxity of the ipsilateral side TMJ. Locking cannot be recreated if joints are normal. Locking may not be recreated in every patient with a history of open-mouth locking episodes. It is important to manipulate the right and left sides even if a unilateral lock was observed in the awake patient, as bilateral TMJ dysplasia may be present that could result in alternating right and left locking episodes (Fig. 36.4). Crepitus secondary to degenerative TMJ disease may be noted during manipulation. Manual reduction of open-mouth locking is accomplished by opening the mouth further, pressing the laterally displaced coronoid process in a medial direction, then closing the mouth. The TMJ laxity is often apparent on manipulation and the coronoid process may be displaced laterally to contact the ventral aspect of the zygomatic arch then slip medially when manipulation is stopped. Neither of these findings is present in normal animals.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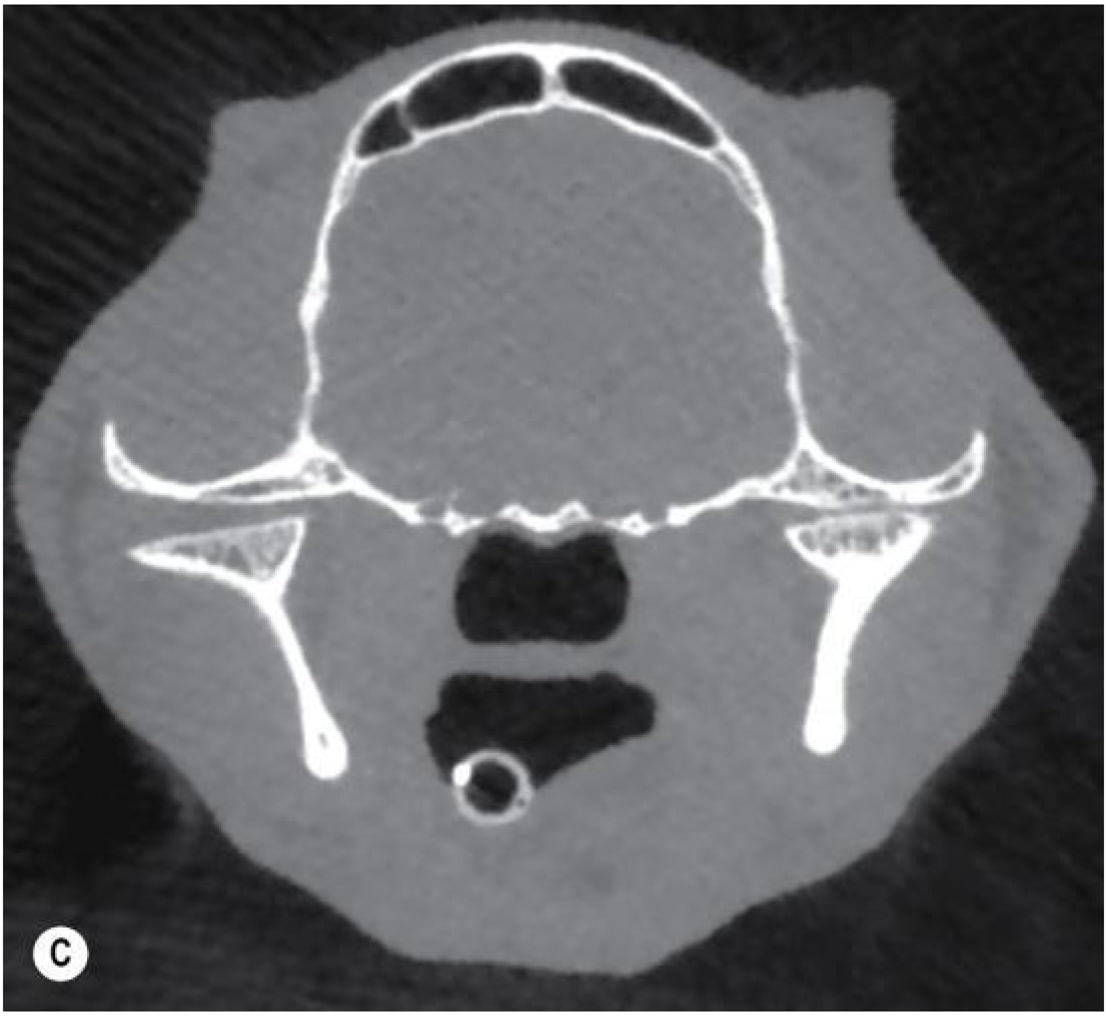

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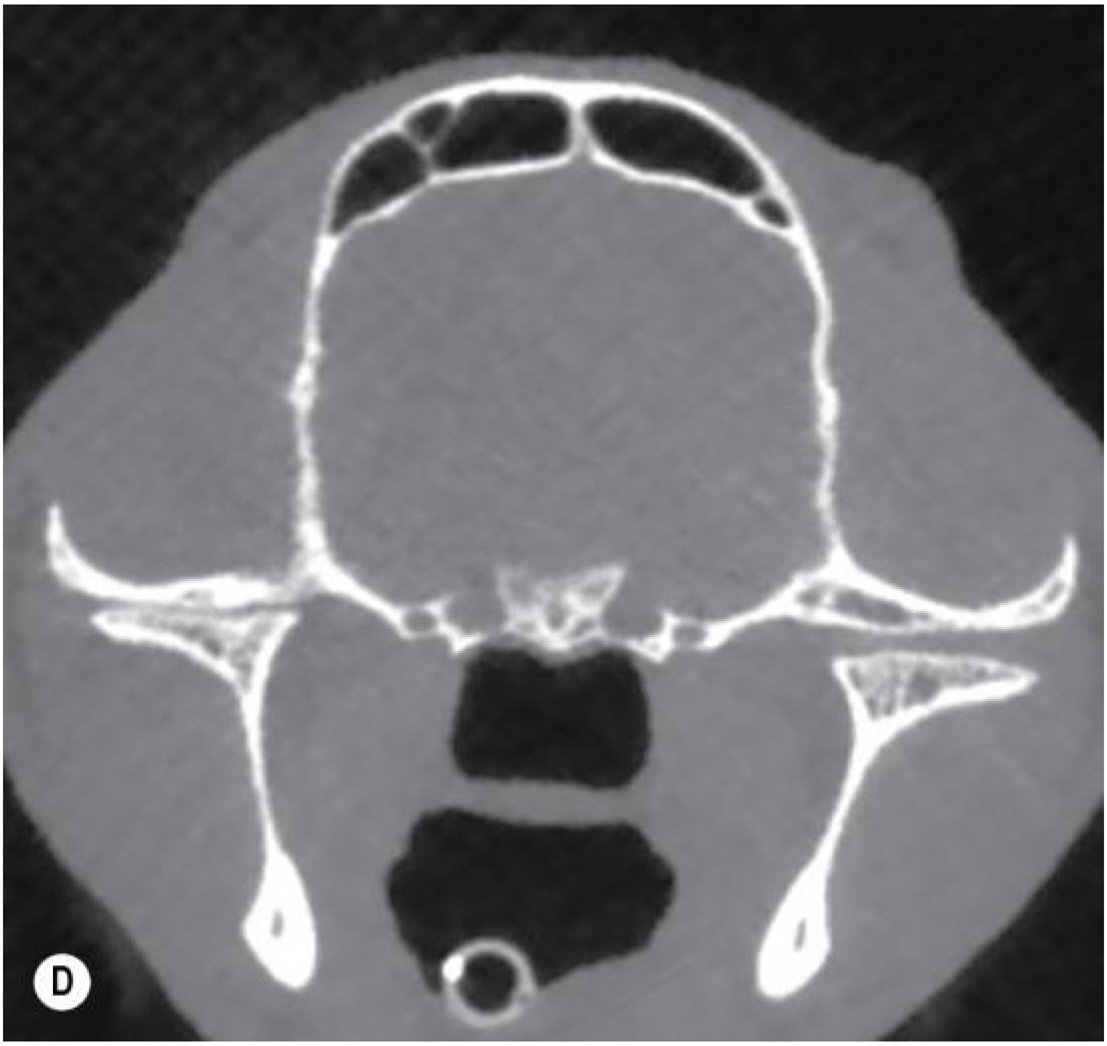


• FIG. 36.4 Three-dimensional **(A, B)** and conventional axial **(C, D)** computed tomography images of a cat. Bilateral temporomandibular joint (TMJ) dysplasia may be present that could result in alternating right and left locking episodes. Note that there is locking of the coronoid process on the zygomatic arch on the left **(A)** and the right **(B)** sides with increased joint space on the contralateral TMJ **(C, D)**.

Diagnostic imaging

Computed tomography and magnetic resonance imaging[6,17–26]

Computed tomography (CT) is currently considered the gold standard diagnostic imaging modality for the diagnosis of TMJ dysplasia and other TMJ disorders (see Chapter 6). [6–9,18–20] In addition, magnetic resonance imaging (MRI) may be used in situations where soft tissue pathology is suspected (e.g., TMJ disc abnormality). Advanced imaging may allow detection of subtle anatomic abnormalities and a comparison between both joints of one patient. The use of CT is of crucial value in evaluation not only of bony lesions but also the spatial position of the TMJ bone components (Fig. 36.5). Furthermore, CT images can be converted into three-dimensional (3D) reconstruction images that further enhance the understanding of the TMJ disorder. This is important, as TMJ disorders frequently include multiple joint abnormalities, such as osteoarthritis, and it is prudent to evaluate all aspects of the disorder rather than focusing on the obvious abnormality.[18] The use of CT also allows assessment of the underlying etiology of the TMJ disorders, which is important for formulating a precise treatment plan. While the CT anatomy of the canine TMJ has been described,[17,21] a recent comprehensive study demonstrated that the TMJ of dogs is diverse in its shape and conformation between breeds and within breeds.[16] The TMJ of dogs within the same breed may exhibit highly variable TMJ shape. Therefore it is of fundamental importance to evaluate TMJ dysplasia by means of CT.

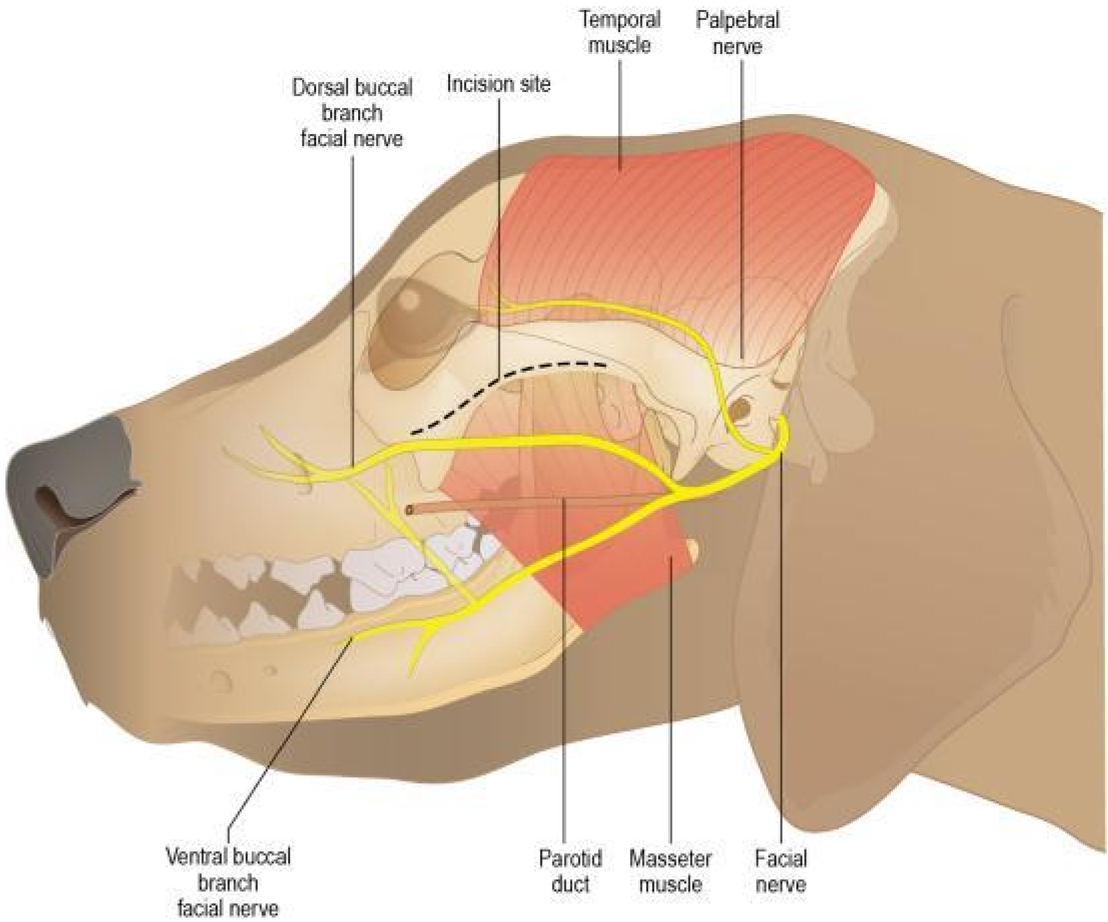
• **FIG. 36.5** Three-dimensional computed tomography (CT) image of a dog with a left side jaw locking. CT is of crucial value in evaluation of the bony lesions and the spatial position of the bone components of the temporomandibular joint.

Radiographic findings

In general, although the radiographic anatomy and patient positioning for TMJ radiographs have been described,[21-24] skull radiography is of limited diagnostic value in TMJ dysplasia and is not recommended for the diagnosis of TMJ disorders in general. However, skull radiography may be used as an initial screening tool.

Surgical anatomy[27]

The function of the zygomatic arch is protection of the eye, origin for the masseter and part of the temporal muscles, and to provide an articulation for the mandible. The zygomatic arch is approached by an incision made along its ventral border. The palpebral and dorsal buccal branches of the facial nerve lie near the dorsal and ventral borders of the zygomatic arch, respectively (Fig. 36.6). The zygomatic and deep sphincter colli muscles, small sensory branches from the mandibular nerve, and small branches of the transverse facial artery and vein cross the incision site. The superficial and middle layers of the masseter muscle originate along the ventral border of the zygomatic arch. The origin of the deep layer of the masseter muscle is intermingled with the lateral aspect of the temporal muscle. The temporal muscle inserts on the medial and lateral aspect of the coronoid process to surround the process. Its lateroventral extent is the ventral aspect of the masseteric fossa. The muscle contacts the pterygoid muscles on the medial aspect of the coronoid process. The body of the masseter muscle lies in the masseteric fossa of the mandibular ramus. The rostral border of the coronoid process, the coronoid crest, is thick cortical bone.



• **FIG. 36.6** The surgical anatomy for partial zygomatic arch excision. Note the location of the zygomatic arch relative to the parotid salivary duct and the ventral and dorsal buccal branches of the facial nerve.

Therapeutic decision-making

Differential diagnoses

Differential diagnoses include conditions that result in acute or chronic inability to completely close the mouth. These include traumatic unilateral or bilateral temporomandibular subluxation or luxation, mandibular fracture, oral foreign body, periodontal disease resulting in extrusion of a tooth with subsequent malocclusion, and trigeminal neuropraxia. In addition, a space-occupying lesion may also result in inability to close the mouth.

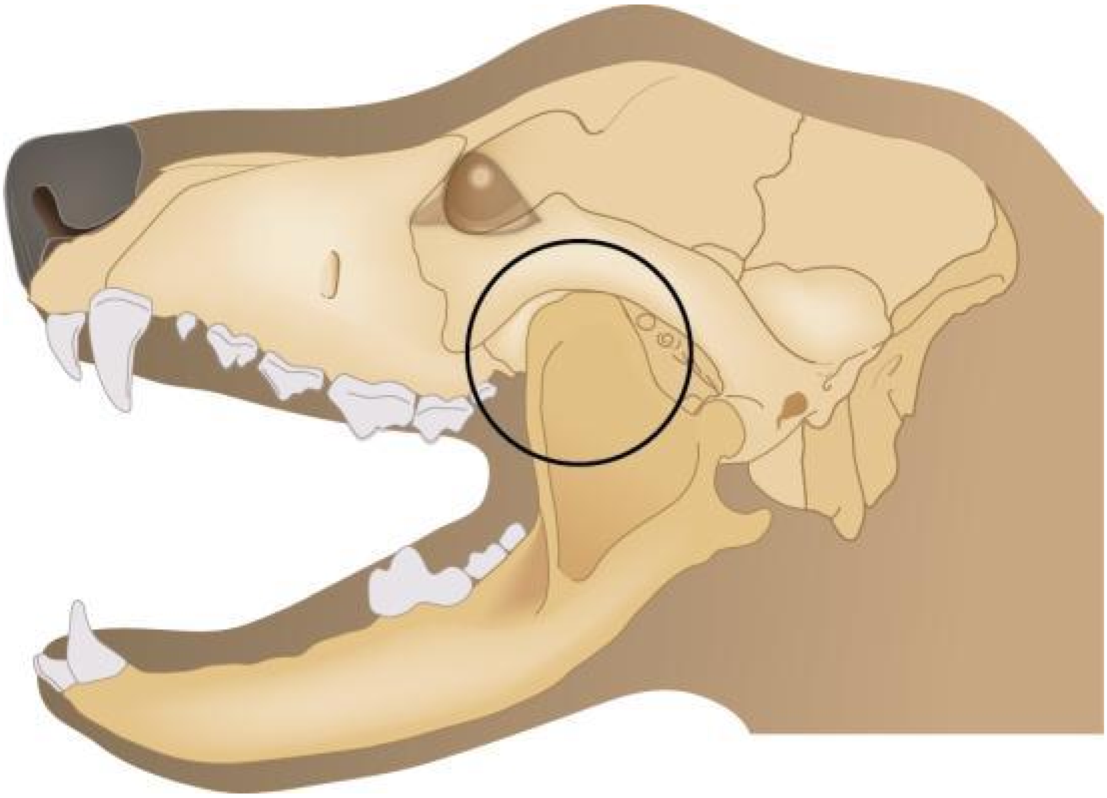
Surgical technique and unilateral or bilateral surgery

The goal of surgical treatment is to prevent further locking episodes rather than to stabilize the TMJ. Partial zygomatic arch resection has been most commonly reported. [2–4,6–9] Mandibular condylectomy, although historically reported,[5] is not recommended, as it is likely to result in further destabilization of the mandibles. Unilateral partial arch resection is considered when (1) the clinical observation of an open-mouth locking episode with a facial bulge is duplicated by jaw manipulation that can only reproduce the same unilateral lock and facial bulge, and (2) history and diagnostic findings support the diagnosis of open-mouth jaw locking and jaw manipulation that can only produce a unilateral lock (with or without a facial bulge). Bilateral surgery should be considered when the history and diagnostic findings support the diagnosis of TMJ dysplasia, and jaw manipulation results in bilateral locking. Imaging using CT with the mouth locked open may help with these decisions. Partial coronoidectomy is indicated for coronoid process height reduction when the history includes documented episodes of open-mouth jaw locking or behavior suggesting oral sensitivity, and diagnostic mandible manipulation confirms contact between the process and junction of the zygomatic and maxillary bones. From a practical perspective, and in order to prevent recurrence, partial zygomatic arch excision together with partial coronoidectomy is recommended for confirmed cases of TMJ dysplasia with open-mouth jaw locking.

Surgical techniques

Partial zygomatic arch excision[3,4]

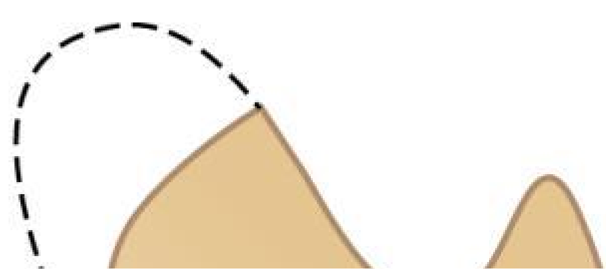
With the patient in lateral recumbency, a skin and subcutaneous tissue incision is made along the rostroventral border of the zygomatic arch for approximately two-thirds of the arch length. Thin fiber bundles of the zygomatic and deep sphincter colli muscles are incised. The mouth is opened and closed by a nonsterile assistant to allow the surgeon to observe the movement of the coronoid process evidenced by lateral displacement of the masseter muscle. Alternatively, the mandibles can be locked prior to surgery to assist the surgeon in localizing the incision. The masseter fascia is incised at the muscle origin along the ventral arch border. The muscle is subperiosteally elevated and ventrally retracted. Mouth opening and closing may be performed as needed to help determine the extent of partial arch excision. A rim (semilunar) ostectomy of the rostral one-half of the arch length and one-half to two-thirds of the arch height is sufficient (Fig. 36.7). Care should be taken to preserve the structures of the orbital ligament. It is recommended that the precision ostectomy be performed using a piezosurgery unit (see Chapter 9) or a surgical handpiece with a bone-cutting bur. Following bone excision, osteoplasty is performed to smooth all the bone edges. Closure is achieved in three layers, with the muscles and fascia closed in one layer, followed by the subcutaneous tissue and skin.

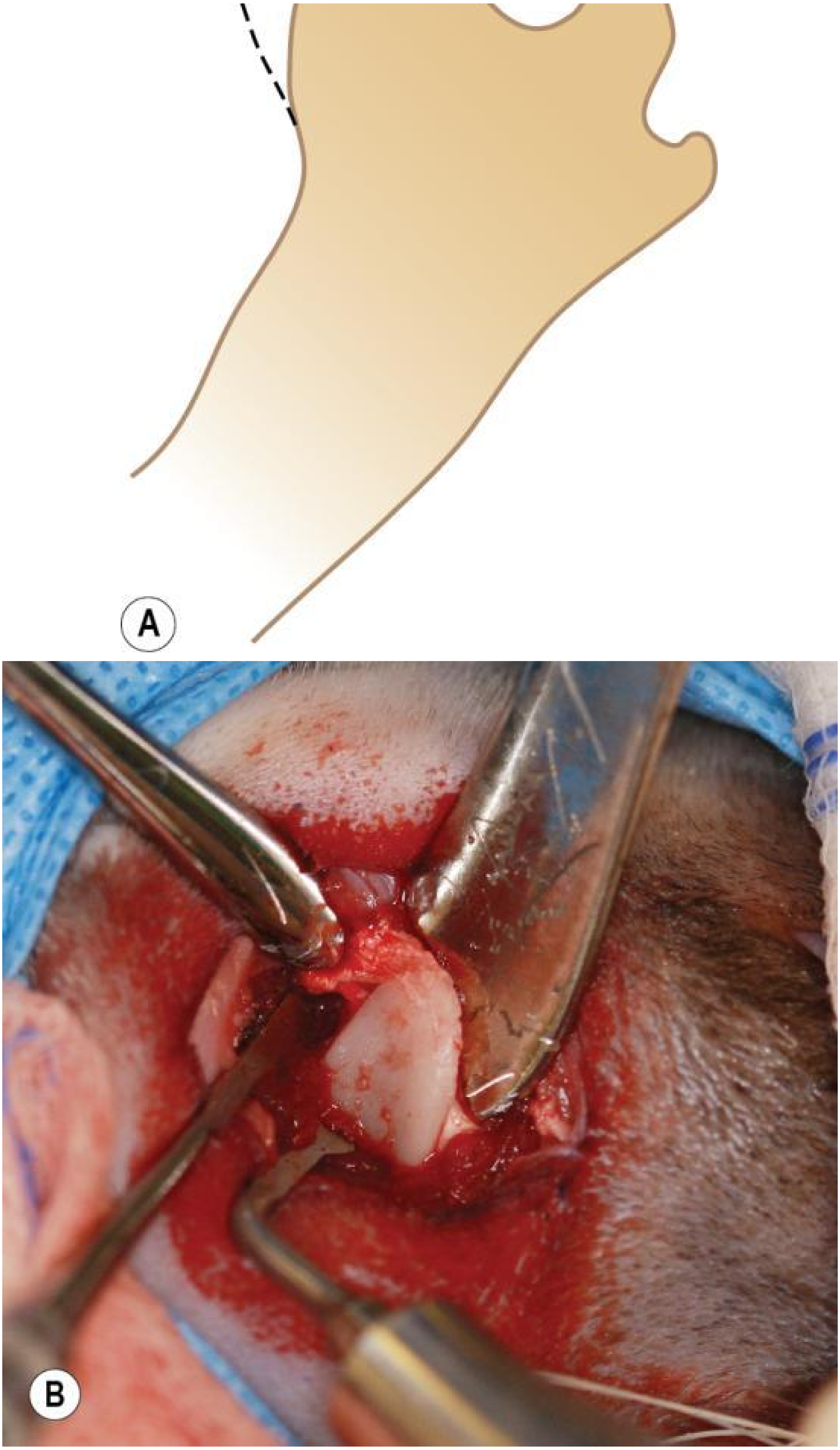


• **FIG. 36.7** The suggested osteotomy lines for the relief of open-mouth jaw locking. A rim (semilunar) ostectomy of the rostral one-half of the arch length and one-half to two-thirds of the arch height is sufficient. Care should be taken to preserve the structures of the orbital ligament. The dorsal one-half to three-quarters of the height of the coronoid process should be removed.

Reduction of the coronoid process height

This procedure is performed in conjunction with partial zygomatic arch ostectomy to provide access to the coronoid process. After partial zygomatic arch ostectomy, the fibers of the deep leaf of the masseter muscle are bluntly separated, the temporal muscle fascia is incised parallel to the coronoid crest, and temporal muscle fibers are longitudinally split immediately lateral to the coronoid crest. The muscles on the lateral aspect are subperiosteally elevated to expose the dorsal one-half to three-quarters of the height of the coronoid process (Fig. 36.8). The fibrous muscle insertion along the coronoid crest is incised and the temporal muscle is subperiosteally elevated from the medial aspect of the coronoid process. Bone excision should be performed with the goal of removing between one-half to three-quarters of the coronoid process height. The bone is cut and carefully separated from any remaining temporal muscle fiber attachments. Following bone excision, osteoplasty is performed to smooth all the bone edges. The temporal fascia is closed and the surgical site is closed as previously described.





• **FIG. 36.8** The extent of coronoid process height reduction is determined in surgery during manipulation of the mandible by a nonsterile assistant. The process is cut and the rostral border tapered in a caudodorsal direction (**A**). Intraoperative image of coronoidectomy in a cat (**B**). Following the elevation of the masseter and temporal muscles subperiosteally, the coronoid process is exposed. Bone excision, preferably using a piezosurgery unit, should aim to remove between one-half to three-quarters of the coronoid process height.

Postoperative care and assessment

Soft food is recommended, and oral activity is limited to eating and drinking for the first 2 postoperative weeks. Thereafter, normal oral activity may resume.

Complications

A small seroma may develop at the surgery site secondary to jaw movement. This usually does not require treatment. If the entire height of the zygomatic arch is excised, some facial asymmetry may result, with the operated side appearing flatter compared to the normal side. Recurrence of locking episodes after a unilateral surgery indicates possible inadequate removal or regrowth of the zygomatic arch, inadequate reduction of coronoid process height, possible undetected bilateral disease, and the need for surgery on the opposite side, or an initial misdiagnosis of the involved side.

Prognosis

The prognosis is generally good after unilateral or bilateral surgery, assuming that enough bone was removed to prevent recurrence. Clients should be informed about the possibility of undetected bilateral disease and the need for additional surgery should locking episodes recur. Long-term results do not indicate further clinical complications associated with the dysplastic temporomandibular joints.[2]

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CHAPTER 37

Fractures and luxations involving the temporomandibular joint

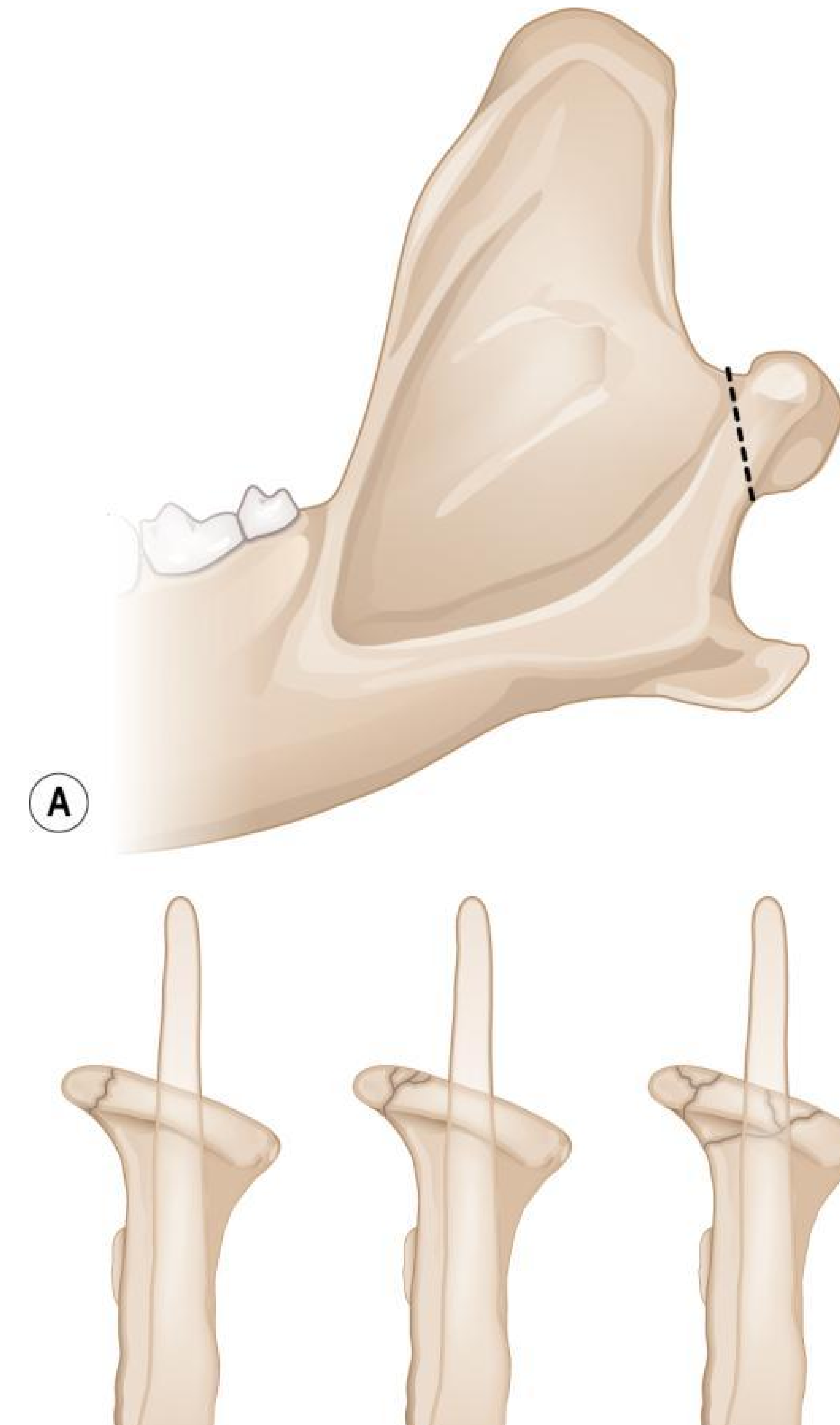
Boaz Arzi, Gary C. Lantz

Definitions

A *dislocation* is a displacement of any part, especially a bone or bony articulation, from its normal position in a joint but the joint components remain within the joint capsule.[1,2] A complete dislocation in which the articular surfaces of a joint are separated is referred to as a *luxation*. A *subluxation* is a partial or incomplete dislocation.

Background

Temporomandibular joint (TMJ) fractures typically occur as a result of trauma and are often seen in combination with other maxillofacial injuries. A classification system made by the Arbeitsgemeinschaft für Osteosynthesefragen Craniomaxillofacial (AO-CMF), modified to accommodate the anatomy of the TMJ region in dogs and cats, can be used to classify TMJ fractures.[3] First, condylar process fractures can be subdivided into mandibular head and condylar process fractures. A condylar process fracture is a fracture line that extends caudal to the mandibular notch (Fig. 37.1A). A mandibular head fracture is defined as fracture line(s) involving the mandibular head. Practically, however, a clear distinction between fractures of the mandibular head and the condylar process may be difficult. In both regions, the complexity of the fracture may be identified as one of three levels: (1) no fragmentation, (2) minor fragmentation, and (3) major fragmentation (Fig. 37.1B).[3] The terms "minor" and "major" refer to fractures with a pattern that preserves or compromises the mandibular head integrity, respectively. With regard to fracture displacement, the vertical apposition of the fragments can be classified as 0 = complete (i.e., full surface contact), 1 = partial (i.e., some surface contact), and 2 = loss or no contact (i.e., complete displacement) (Fig. 37.1C).[3] In cases with several fractures, classification is made according to the worst fracture. Furthermore, mandibular head fractures are typically intraarticular (i.e., involving the fibrocartilaginous articular surface and potentially damage to the TMJ disc). With regard to the mandibular fossa, a similar classification as for the mandibular head can be followed.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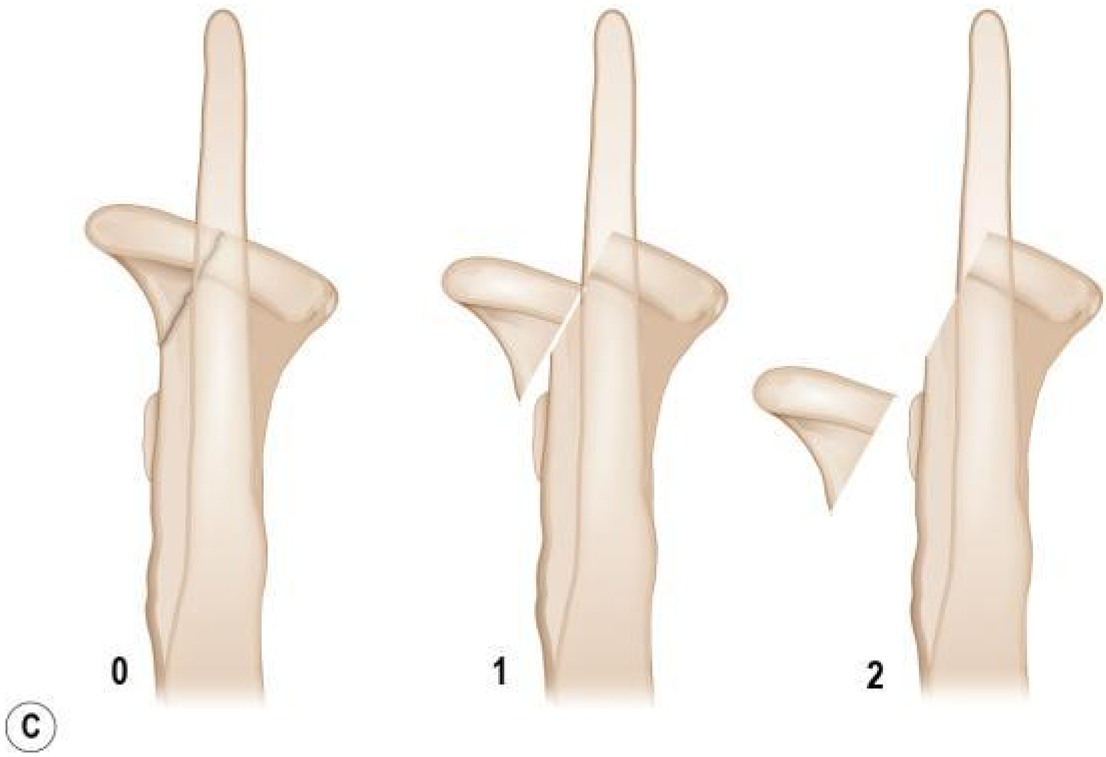


• **FIG. 37.1** A classification system modified from the AO-CMF to accommodate the anatomy of the temporomandibular joint region in dogs and cats. **(A)** A condylar process fracture is a fracture line that extends caudal to the mandibular notch. **(B)** The fracture complexity is classified in one of three levels: (1) no fragmentation, (2) minor fragmentation, and (3) major fragmentation **(C)**. Fracture displacement is classified as 0 = complete (full surface contact), 1 = partial (some surface contact), and 2 = loss or no contact (complete displacement). Source: (After Neff A, Cornelius CP, Rasse M, et al. The comprehensive AOCMF classification system: condylar process fractures—level 3 tutorial. *Craniomaxillofac Trauma Reconstr*. 2014;7[Suppl 1]:S044–S058.)

Clinical presentation

History and physical findings

TMJ fractures and luxations typically occur as a result of trauma.[4-7] They are often seen in combination with other maxillofacial injuries.[8,9] A complete physical examination, including assessment of cranial nerves and the eyes, is performed to search for other injuries caused by the initiating trauma. The most common finding with TMJ subluxation, luxation, or fracture is difficulty or inability to completely open or close the mouth. Subluxation may or may not result in malocclusion, while luxation typically does result in malocclusion. Unilateral rostral luxation results in shifting of the mandible toward the side opposite the luxation. Unilateral caudal luxation results in shifting of the mandible toward the side of luxation. Bilateral rostral luxation results in variable degrees of rostral protrusion of the mandibles. A fracture involving the TMJ may or may not result in malocclusion, depending on the degree of displacement. Fractures of the mandibular ramus and coronoid process commonly cause malocclusion. As in other TMJ disorders, fracture characterization should be made based on computed tomography (CT) imaging.

Oral examination

Initial oral examination without anesthesia is typically limited to evaluation of occlusion and observation of the animal's ability or inability to open and close its mouth. A complete oral examination requires general anesthesia. After anesthetic induction and before intubation, an oral examination is performed to identify and document any occlusion abnormalities, oral wounds, and/or open fractures. The mandibles and maxillae are gently palpated and the mandibles manipulated. Palpable crepitus suggests fracture, and gentle pressure will usually allow mouth closure. Malocclusion and inability to close the mouth by manipulation may suggest TMJ luxation or fractures. The oral examination is combined with diagnostic imaging of the skull, ideally CT, as multiple maxillofacial injuries are often present.[9]

Anesthetic considerations

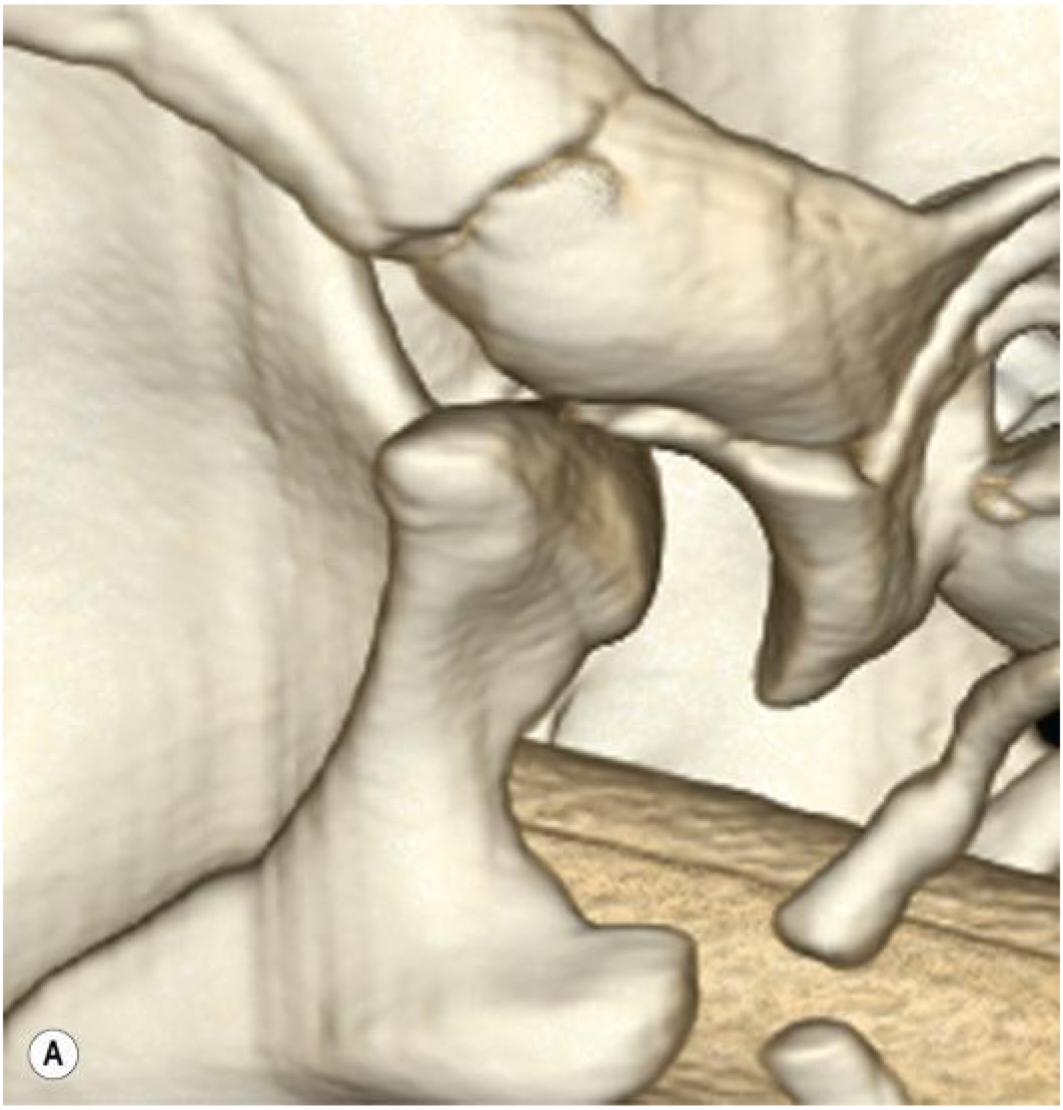
Preanesthesia evaluation

A preanesthesia database should consist of physical examination, complete blood count, serum biochemistry profile, and urinalysis. Thoracic radiographs and abdominal ultrasound should also be obtained for all patients because of the traumatic nature of the condition. The patient is completely evaluated to determine the presence of additional injuries before the induction of anesthesia. After oral examination, CT scanning should be performed and the extent of the injury characterized and assessed. Repair of concurrent mandibular body fractures may require endotracheal tube placement by pharyngotomy to allow intraoperative evaluation of occlusion (see Chapter 61). The pharyngotomy is preferably performed on the side opposite to the involved TMJ. If both TMJs are involved, then a temporary tracheostomy may be needed for intubation.

Diagnostic imaging

Computed tomography and magnetic resonance imaging[9-11]

CT is currently considered the gold-standard diagnostic imaging modality for the diagnosis of TMJ fractures, luxation, and other TMJ disorders (see Chapter 6). Advanced imaging may allow detection of subtle anatomic abnormalities and a comparison between both joints of one patient. CT is of essential value in evaluation not only of bony lesions but also of the spatial position of the TMJ bone components. Furthermore, CT images can be converted to three-dimensional (3D) reconstruction images that further enhance the understanding of the TMJ disorder. CT also allows comprehensive assessment of other maxillofacial injuries that may have occurred during the trauma, which is important for formulating a precise treatment plan. Specifically, a widened joint space may suggest subluxation. With luxation, the condylar process of the mandible is displaced from the mandibular fossa of the temporal bone, most commonly in a rostrodorsal direction (Fig. 37.2).[8] Luxation may be unilateral or bilateral with rostral or caudal displacement. Luxation in a caudal direction is typically associated with a fracture of the retroarticular process.[12-16] Fractures may affect any of the bony joint components.


A

• **FIG. 37.2** Three-dimensional reconstructed computed tomographic images of temporomandibular joint luxation in **(A)** dog and **(B)** cat. Note that luxation typically occurs in rostral and dorsal direction.

Radiographic findings

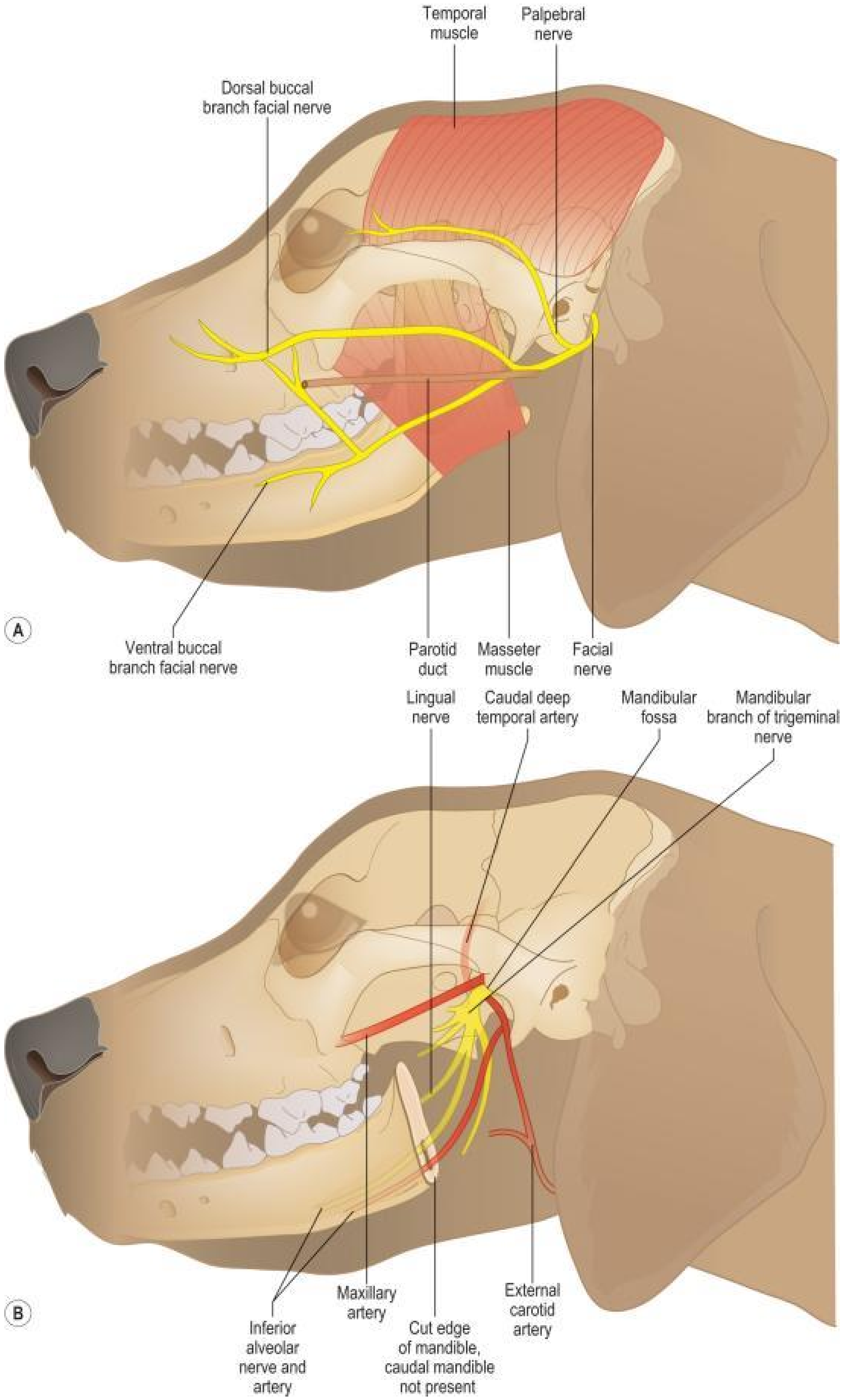
Although radiographic anatomy and patient positioning for TMJ radiographs have been described,[17-21] skull radiography is of limited diagnostic value for TMJ fractures and is not recommended for the diagnosis of TMJ disorders. However, skull radiography may be used as an initial screening tool.

Surgical anatomy[22,23]

The anatomy and function of the TMJ are described in Chapter 36. The head of the mandible on the condylar process articulates in the mandibular fossa of the squamous part of the temporal bone (Fig. 37.3). This synovial joint has a joint capsule that extends between the articular cartilages and fibrocartilaginous disk, separating the articular surfaces into two noncommunicating compartments. The lateral ligament strengthens the lateral aspect of the joint capsule. In comparison with the dog, the feline TMJ has a closer congruity, and the structure of the feline mandibular symphysis allows less independent movement of the mandibles.[17]

• FIG. 37.3 The temporomandibular joint: normal relationship of the mandibular head on the condylar process and the mandibular fossa of the squamous part of the temporal bone.

The lateral aspect of the joint is partially covered by the masseter muscle (Fig. 37.4). The facial nerve is located caudoventral to the joint and its branch, the palpebral nerve, and courses along the dorsal border of the zygomatic arch. The maxillary artery and origin of the inferior alveolar and caudal deep temporal arteries lie ventromedial to the condylar process. The mandibular branch of the trigeminal nerve lies medial to the process. The auriculotemporal nerve passes immediately caudal to the retroarticular process of the temporal bone. The lateral pterygoid muscle inserts on the medial aspect of the condylar process. The mandibular branch of the trigeminal nerve lies immediately rostral to the TMJ.



• **FIG. 37.4** Surgical anatomy (**A**) lateral and (**B**) medial to the temporomandibular joint.

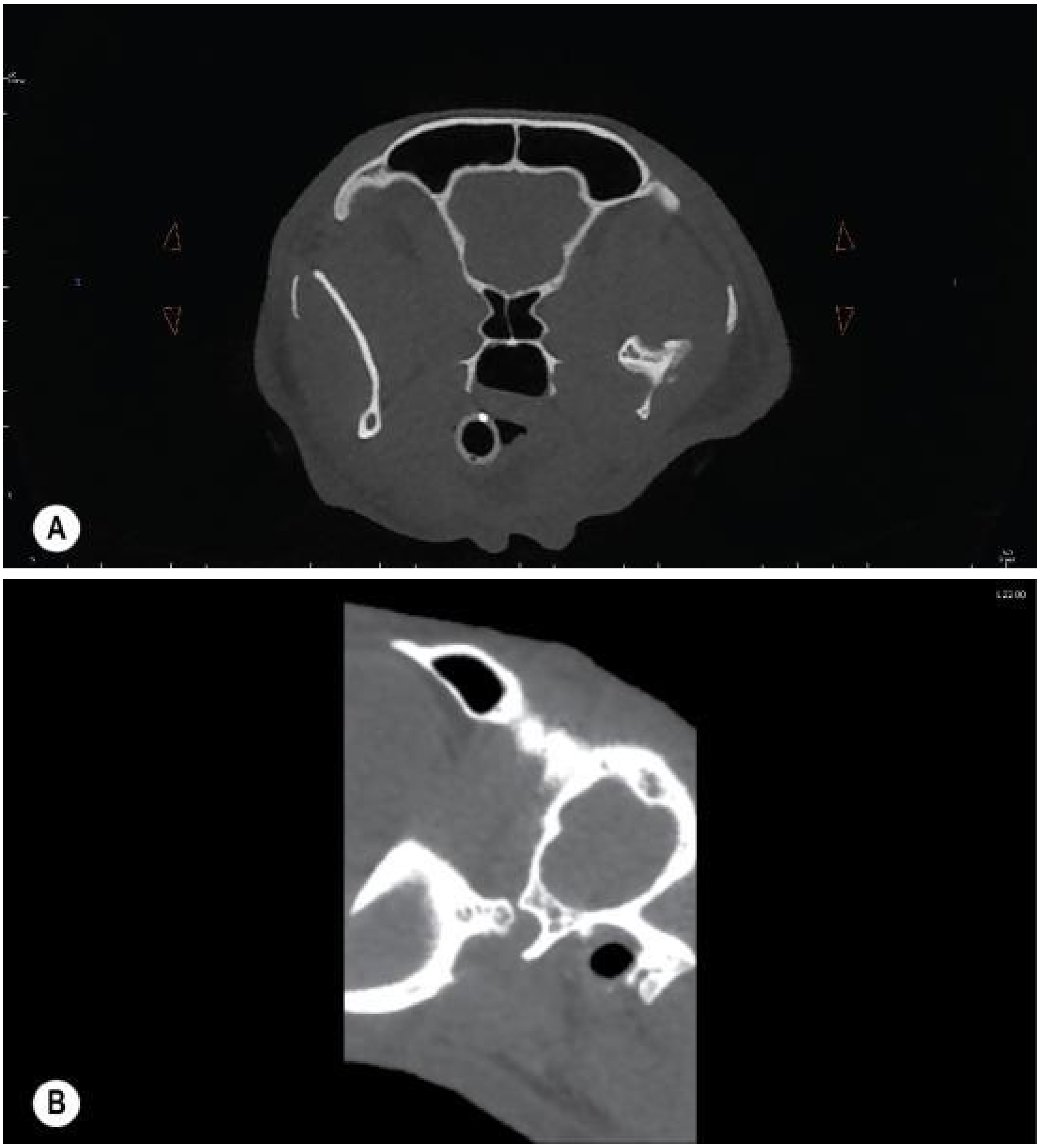
Therapeutic decision-making

Differential diagnoses

Differential diagnoses include conditions that result in acute or chronic inability to completely close or open the mouth. These include traumatic TMJ subluxation or luxation, mandibular fracture, TMJ dysplasia, oral foreign body, periodontal disease resulting in extrusion of a tooth with subsequent malocclusion, and trigeminal neuropraxia.

Closed versus open reduction of subluxations and luxations

Subluxation of the TMJ requires no specific treatment other than soft foods and restriction of oral activity during healing. Reduction of TMJ luxations is attempted using closed techniques. Reduction of a chronic luxation is difficult or impossible due to organized fibrous tissue that fills the joint space or osteoarthritic conditions present in the joint (Fig. 37.5). Therefore closed reduction should be attempted as soon as possible after the injury occurred. Open reduction or condylectomy is considered for nonreducible luxation and recurrent luxation, or for TM joints that exhibit osteoarthritic changes.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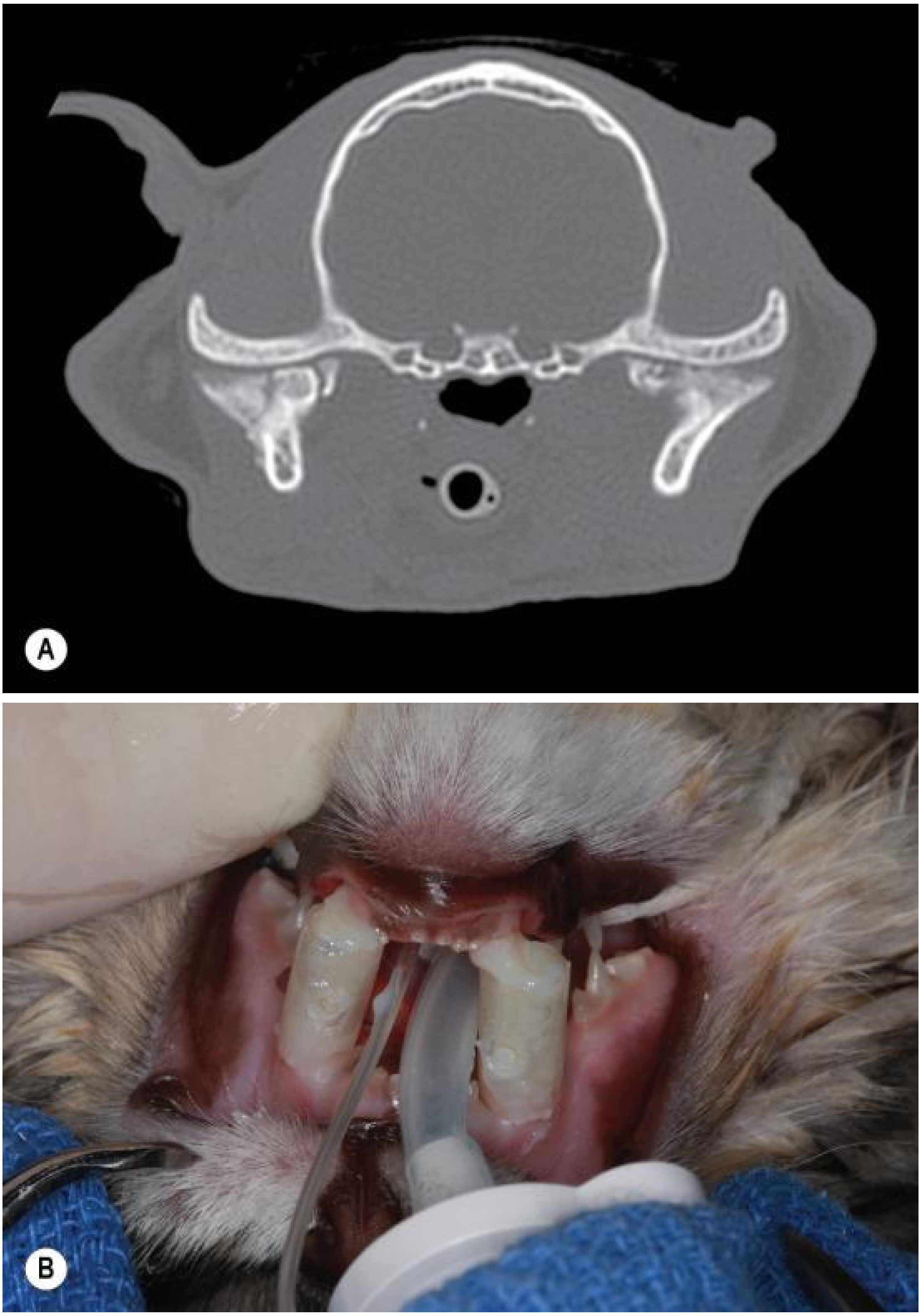
B

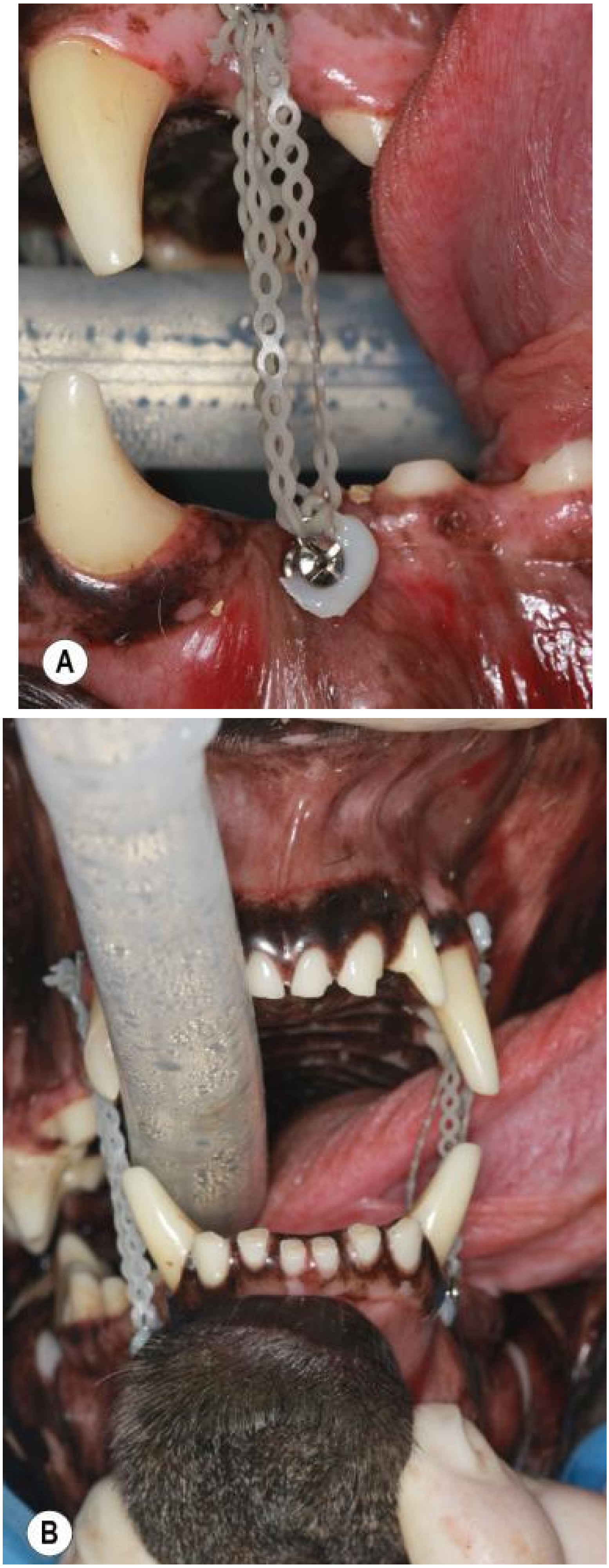
• **FIG. 37.5** Computed tomographic images of luxated temporomandibular joint in a cat. Note the advanced arthritic lesions seen on (**A**) transverse, (**B**) sagittal, and (**C**) three-dimensional images.

Nonsurgical versus surgical treatment of condylar and pericondylar fractures

The goal of managing TMJ fractures is to restore mandibular symmetry, occlusion, and function and to prevent long-term complications. In young dogs and cats, as well as in most adult dogs (and based on the fracture configuration), nonsurgical (i.e., conservative) therapy is the method of choice. There is excellent chance for fracture healing and regeneration of the damaged tissues, as well as continuation of normal development, in young dogs and cats with TMJ fractures. A nonsurgical approach typically involves maxillomandibular fixation (MMF), which can be achieved by one of two methods—(1) *rigid*: a dental composite (i.e., temporization material) that fuses the mandibular and maxillary canine teeth in a partially closed-mouth position, leaving and allowing 10–20 mm of mouth opening (Fig. 37.6); and (2) *elastic*: placement of elastic bands connecting the mandibles to the maxillae functions as a guide for normal occlusion while allowing the jaws to maintain guided minimal mobility. The authors use intermaxillary (IMF) screws and elastic orthodontic chains to manage TMJ fractures in which there is minimal to no malocclusion (Fig. 37.7).



• **FIG. 37.6** (**A**) Bilateral minimally displaced mandibular condylar fractures in a cat. (**B**) Maxillomandibular rigid fixation may be used to stabilize condylar fractures.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• FIG. 37.7 (**A, B**) Elastic therapy using intermaxillary (IMF) screws and elastic orthodontic chain may be used to manage temporomandibular joint fractures in dogs in which there is minimal to no malocclusion.

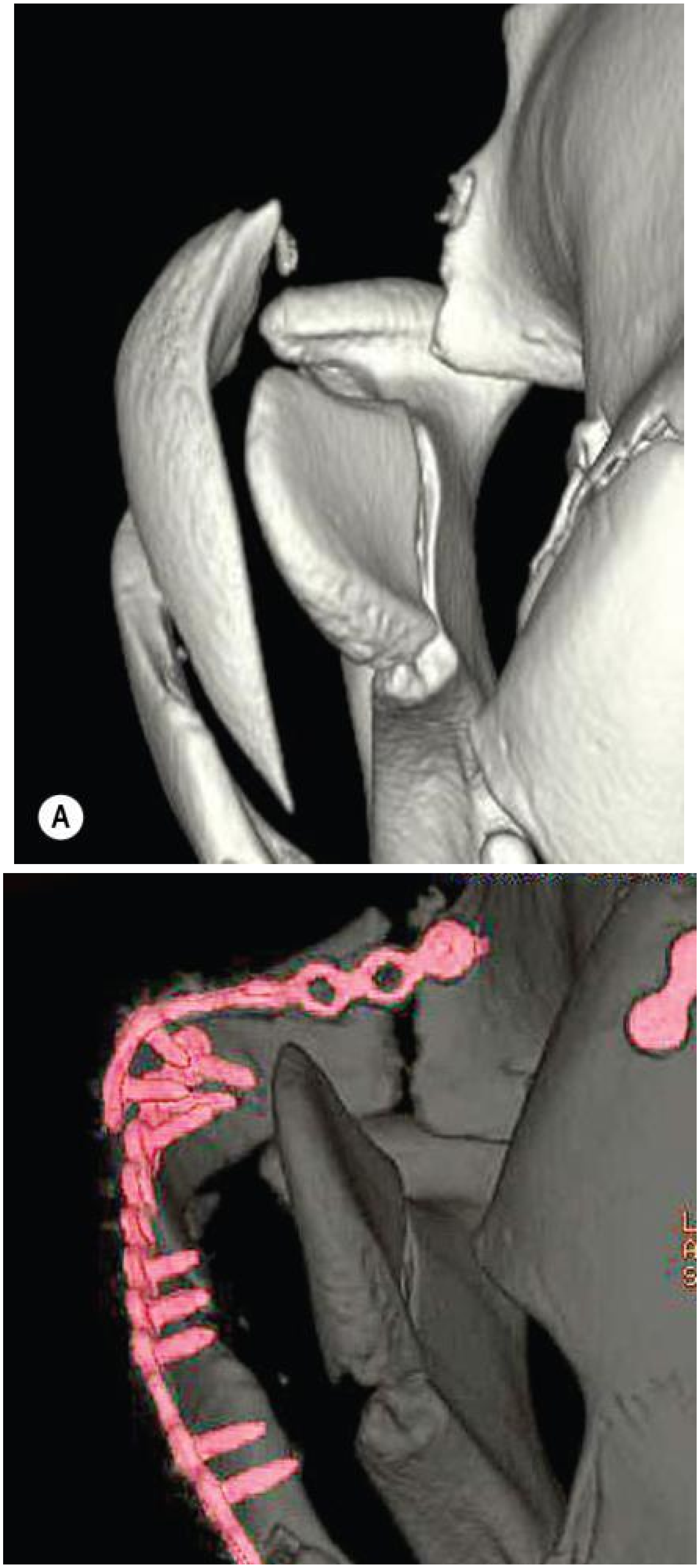
If there is a noticeable moderate to severe malocclusion, then closed reduction and rigid therapy is recommended for a period of 7–14 days in young patients and 2–4 weeks in adults. Once the rigid MMF is removed, elastic therapy can be maintained for two additional weeks.

Disadvantages of rigid therapy include delayed return to normal function, maintaining feeding tube, poor oral hygiene, difficulties in thermoregulation, and potential aspiration.

However, if the fracture is nondisplaced and there is mild or no malocclusion, then elastic (functional) therapy is recommended for a period of 14–21 days. This will allow a more rapid return-to-normal function as compared to rigid therapy, allow the fracture area to receive more blood supply (due to the movement of the joint and muscles surrounding it), and decrease the chance of complications due to aspiration or thermoregulation issues.

Open reduction is recommended only if there are fracture fragments in the joint space preventing opening or closing of the mouth. Condylectomy is not recommended and should be reserved as an extreme measure in case of complete destruction of the TMJ and for fragments that prevent the joint from regaining normal function (Fig 37.8). For severely displaced fractures of the mandibular fossa (squamous part of the temporal bone), reconstruction using miniplates may be indicated (Fig. 37.9).

• FIG. 37.8 Three-dimensional reconstructed computed tomographic image of a complete and severely displaced condylar process fracture in a dog. In this case, the condylar process was surgically removed.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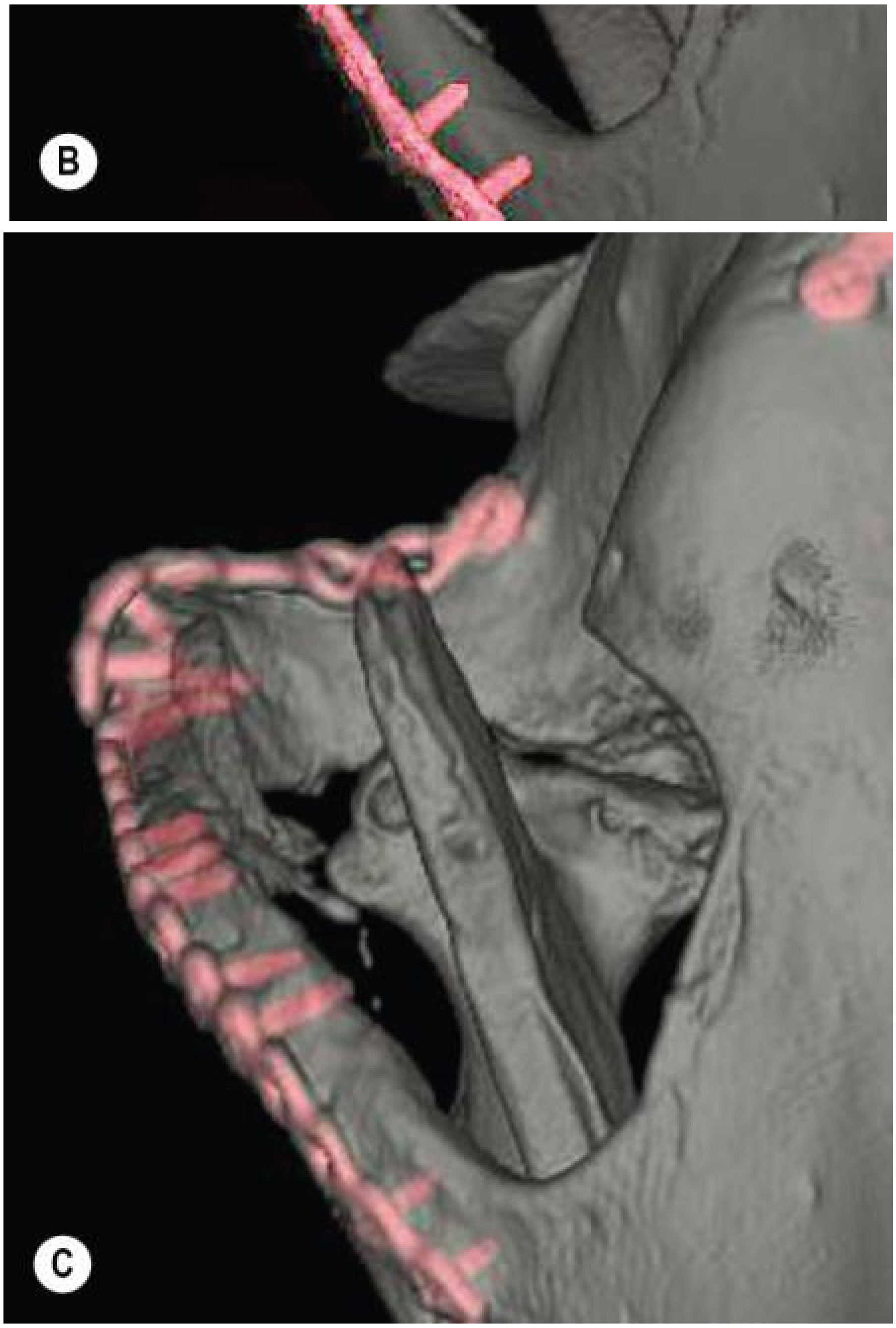
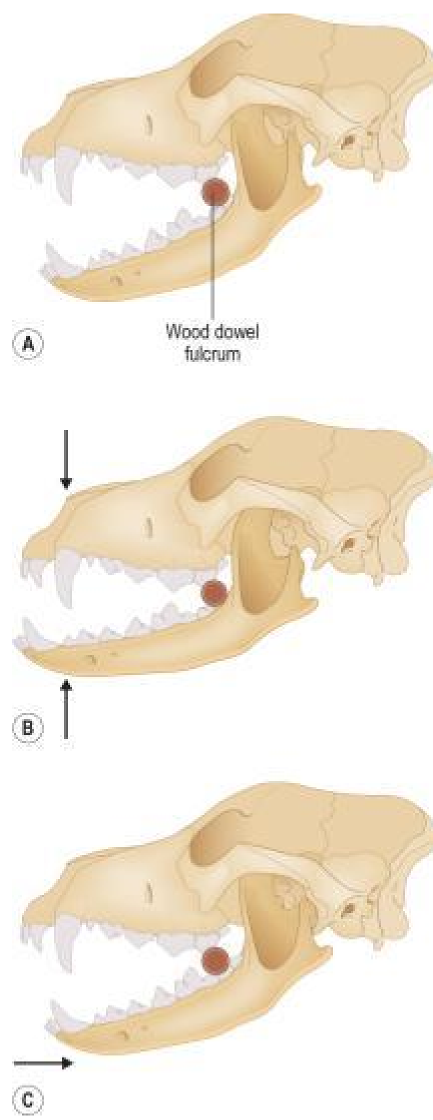


• **FIG. 37.9** Three-dimensional reconstructed computed tomographic images of severe and displaced fractures of the mandibular fossa, the dorsal part of the temporomandibular joint, in a dog (**A**) before, (**B**) immediately after, and (**C**) 2 months following repair using a nonlocking 2.0-mm titanium miniplate. The zygomatic arch fractures have also been reconstructed using a nonlocking titanium miniplate. Regeneration and remodeling of the fractured bone are evident on the recheck image.

Closed reduction and nonsurgical techniques

Closed reduction of luxations

The most common direction of TMJ luxation is rostrodorsal, although this must be confirmed by CT imaging. The patient is anesthetized, intubated, and placed in sternal recumbency. A fulcrum is obtained by placing a pencil (or other similar softer wooden or plastic dowel) transversely across the mandibles at the level of the second and third molar teeth on the same side of the luxation.[5,8,24,25] Larger dogs require using a larger-diameter fulcrum. A fulcrum made of hard material may inadvertently cause tooth cusp tip fracture. The mouth is gently closed while the mandibles are pushed caudally. To help with the reduction, the wooden pencil can be rotated counterclockwise (if the operator is on the left side of the patient). This manipulation moves the displaced mandibular condylar process in a ventral direction. The mandible is shifted caudally toward the side of the luxation to seat the condylar process into the mandibular fossa (Fig. 37.10). The mouth is gently opened and closed and observed for shifting of the jaw toward the side opposite to the luxation, indicating luxation recurrence. Alternatively, especially in cats, reduction can be accomplished by opening the mouth wide and pressing intraorally with one finger over the coronoid process. Reduction will result in return to normal occlusion.



• **FIG. 37.10** Closed reduction of a rostrodorsal temporomandibular luxation. (**A**) A wooden dowel fulcrum is placed transversely across the dental quadrants and the level of the caudal molar teeth. (**B**) The mouth is gently closed, bringing the displaced condylar process to the level of the mandibular fossa. (**C**) The jaw is moved caudally to reduce the luxation.

Following reduction of the TMJ, placement of a tape or nylon muzzle or, preferably, rigid MMF is needed to maintain the reduction.[26,27] A tape or nylon muzzle allows more mandibular movement than does rigid MMF. Success when using a muzzle depends on allowing the patient only minimal mouth opening for eating and drinking and maintaining normal occlusal relationships between the caudal mandibular and maxillary teeth to prevent lateral displacement of the mandible. Postreduction CT imaging is recommended to evaluate the reduction.

Surgical techniques

Open reduction of luxations

For unilateral luxation repair, the patient is positioned in lateral recumbency. For bilateral repair, alternating lateral recumbency will allow the best exposure to the joints. A skin incision is made along the ventral border of the caudal half of the zygomatic arch (see Chapter 27).[23] Incision and retraction of the subcutaneous tissue, platysma, zygomatic, and sphincter colli muscles exposes the ventral border of the zygomatic arch and masseter muscle. The palpebral nerve, parotid duct, and masticatory nerve are avoided. The caudal half of the origin of the masseter muscle is subperiosteally elevated and the muscle retracted in a rostroventral direction. Often, the TMJ capsule has been torn and articular cartilage of the condylar process is visible. If the joint capsule is intact, it is opened with an incision parallel to the zygomatic arch.

Blood clots and tissue debris are removed from the joint space. An intact articular disk may prevent postoperative ankylosis and an uninjured disk should be left in place.[28] However, tearing or folding of the articular disk requires discectomy. The luxation is reduced and capsule remnants sutured together. The masseteric fascia is sutured to the periosteum covering the zygomatic arch. The incision is routinely closed. The mouth is gently closed around the endotracheal tube and MMF rigid fixation is applied for a period of 2–3 weeks, immobilizing the jaws with approximately 10–15 mm of mouth opening measured at the incisor teeth level. Postoperative CT is recommended to ensure proper reduction.

Condylectomy[21,29]

Condylectomy is performed only for exceptional situations such as nonreducible luxation, luxated arthritic TMJ, recurrent luxation, or TMJ fracture with severely displaced bone fragments (see Chapter 37). Displaced fractures involving the condylar process and the mandibular head are usually not reconstructed due to the small size of the fracture fragments. In the case of complete displaced fracture, all bone fragments of the condylar process are excised. Shredded joint capsule is repaired if possible. Osteoplasty is performed using a round diamond bur or piezotome. The articular disk, if undamaged, is left in place. Preservation of the articular disk may reduce fibrous tissue adhesions to the region of the mandibular fossa and may be important for preserving normal postoperative range of motion.

Postoperative care and assessment

Immobilization

The patient must be observed during recovery for signs of inadequate ventilation and vomiting. The surgeon should be present in the recovery area and prepared to remove the MMF if an emergency situation occurs. Whenever rigid or elastic MMF is applied, placement of an esophagostomy tube is recommended and should be performed prior to fixation. Home care consists of free-choice water and feeding soft or gruel-consistency foods. Patients are not to be confined to warm environments, as hyperthermia may result from the inability to pant.

Physiotherapy

Prolonged immobilization of the TMJ should be avoided, as immobilization, combined with healing of traumatized periarticular tissue, may result in limited range of mouth opening and may encourage ankylosis. It is recommended that devices be removed and normal motion allowed by 2–3 weeks for luxations and TMJ fractures.[8,19,24,25] Clinical healing of fractures is determined by physical examination and diagnostic imaging. For TMJ fractures, feeding soft food for the first 2–3 days following removal of MMF should be followed by introduction of large, hard kibble to increase the jaw activity. Clinical healing of fractures involving the TMJ occurs in 10–13 weeks for dogs and 4–8 weeks for cats.[6,7]

Prognosis

Conservative treatment

Results of conservative treatment for luxation and nondisplaced or minimally displaced TMJ fracture are dependent on the individual injury and postoperative patient management. Extensive soft tissue injury and/or inadequate postreduction management may result in recurrent luxation. The lack of appropriate stability or confinement at the fracture site may result in malunion or nonunion. A fibrous union may develop secondary to motion at the fracture site; however, normal mandibular function may still occur. In general, most reduced TMJ luxations and nondisplaced or minimally displaced TMJ fractures heal adequately and provide normal oral function with conservative management.

Complications

Intraoperative complications

Intraoperative hemorrhage and nerve injury may occur during dissection medial to the TMJ with inadvertent injury to large neurovascular structures (e.g., maxillary artery and nerve) in this area. This can be prevented by careful and controlled ostectomy (i.e., piezosurgery) when removing the condylar process.

Postoperative complications

Degenerative joint disease

Trauma to the TMJ that results in luxation or fracture and poor alignment of articular fracture fragments may result in degenerative joint disease (i.e., osteoarthritis). Clinical signs are those of oral pain and may include “deliberate,” slow chewing motion, preference for soft foods, avoidance of chew toys, apparent reduction in appetite with weight loss, and pain on opening and closing the mouth. Crepitus may be found on mandible manipulation of the anesthetized patient. Diagnostic imaging findings may indicate irregularities of the TMJ bone structure. Treatment with nonsteroidal antiinflammatory drugs may reduce the clinical signs.

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CHAPTER 38

Temporomandibular joint ankylosis and pseudoankylosis

Boaz Arzi

Definitions

Ankylosis in the literal sense means "fusion" of body parts.[1] In the context of the temporomandibular joint (TMJ), ankylosis is defined as fibrous or bony fusion of the mandibular head on the condylar process and the mandibular fossa of the squamous part of the temporal bone.[2] Ankylosis of the TMJ can be intraarticular, extraarticular, or both.[1,3] Intraarticular ankylosis involves destruction of the TMJ disc and the articulating fibrocartilaginous surfaces, as well as narrowing of the joint space and flattening of the mandibular head.[4] In contrast, extraarticular ankylosis occurs due to fibrous and/or bony encapsulation of the joint or structures remote from the joint (i.e., the zygomatic arch and the coronoid process of the mandible), with minimal or no intraarticular involvement.[1,4,5] When structures outside the TMJ are affected, the term "false-" or "pseudoankylosis" is used.[3]

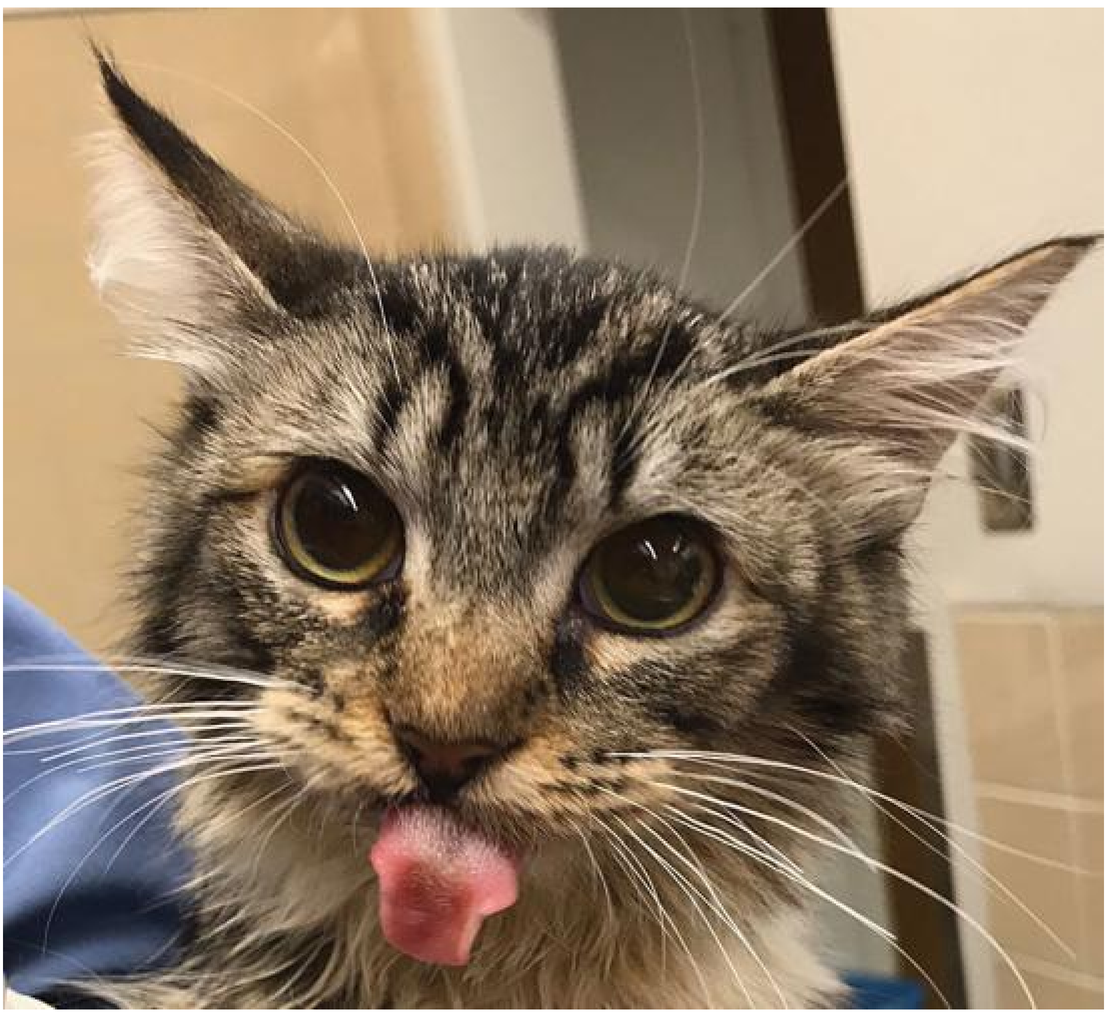
Pathophysiology

Ankylosis and pseudoankylosis are uncommon clinical entities.[3] The most common cause for these disorders is oral and/or maxillofacial trauma.[6] Other possible causes for TMJ ankylosis and pseudoankylosis are infection, neoplasia, and craniomandibular osteopathy.[3] The disorders may occur at any age, but are more commonly seen in young patients. Most cases are unilateral, but bilateral disease is also occasionally seen. When present during skeletal development, TMJ ankylosis may impair facial growth and development.[1,3,7] Histologically, TMJ ankylosis is characterized by an extensive accumulation of dense fibrous tissue and osseous bone formation. There may also be destruction of fibrocartilage and bone with some lymphocytic infiltration.[1]

Clinical presentation

Signalment and history

Commonly, affected patients will be presented with a known history of oral and maxillofacial trauma.[6] However, in patients adopted at later stages of life or those in which the trauma was not noticed by the owners, the given history may not include previous oral or facial injury. Clinically, these dogs and cats are presented with gradually worsening inability to open the mouth.[1,3,5,6] In addition, the mandible may be drifting toward the affected side when opening the mouth. Skeletal and/or dental malocclusion may be present, especially if ankylosis occurred at young age. In severe cases, complete immobilization of the mandibles can be observed with no ability to open the mouth.[1,3,4,8] The tongue may be entrapped, resulting in severely restricted mobility (Fig. 38.1). Pain and discomfort may be present, especially in the early stages of the disorder.



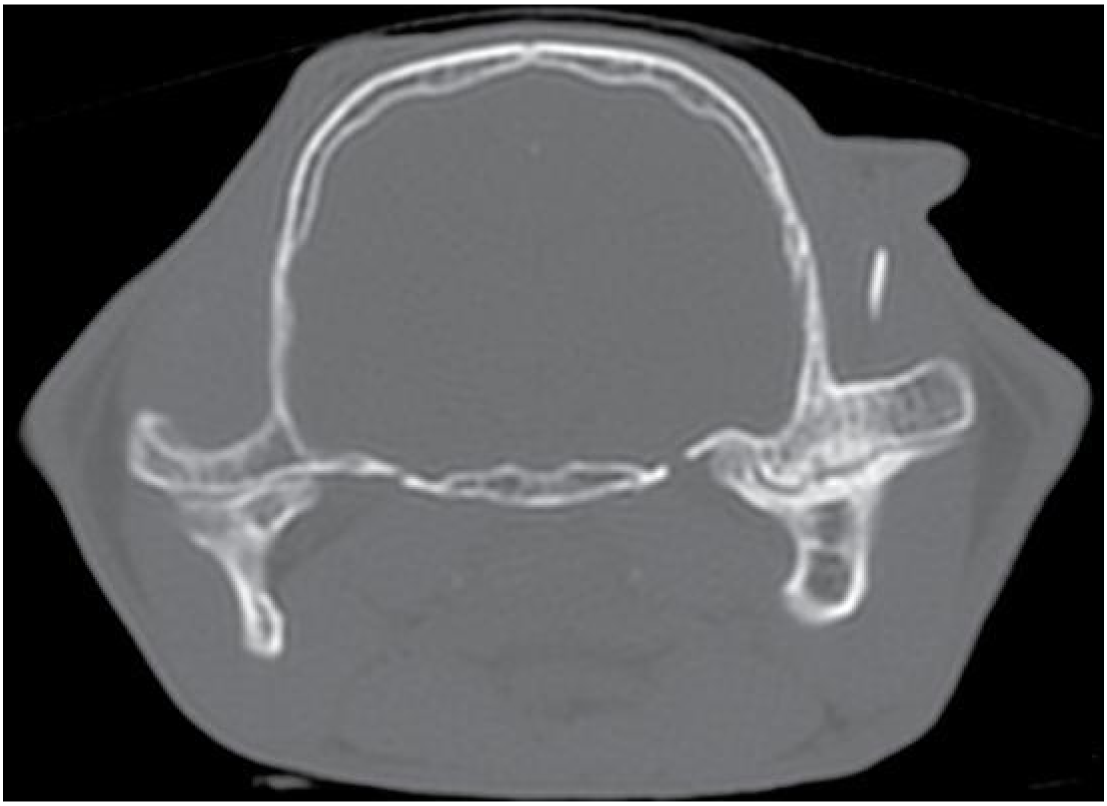
• **FIG. 38.1** Severe bilateral ankylosis in a cat with complete immobilization and tongue entrapment.

Physical examination

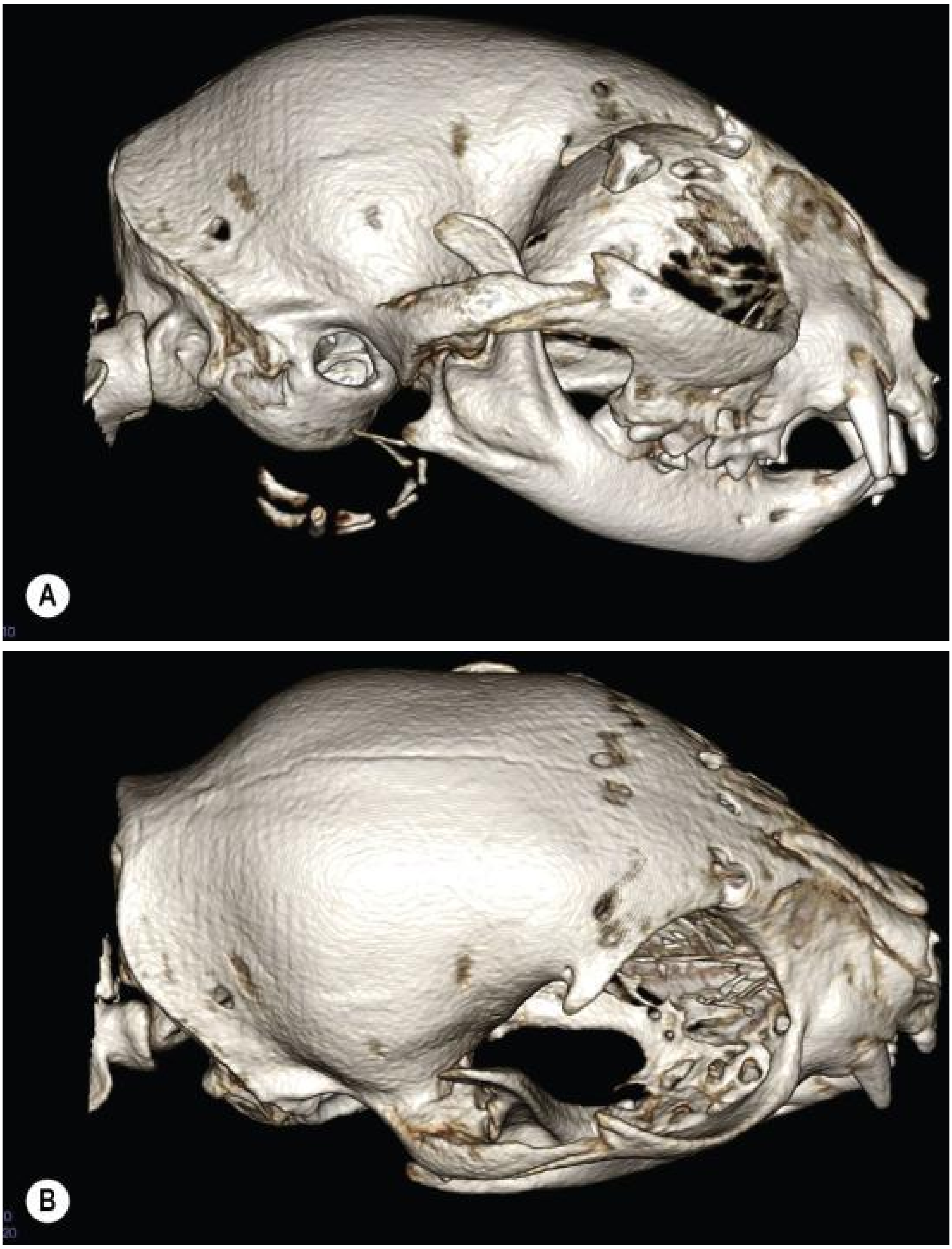
Physical examination may be unrewarding in patients with ankylosis or pseudoankylosis. Palpation of the TMJ region may cause discomfort or pain. Occasionally, a palpable hard or firm mass may be present at the area of ankylosis. Occlusion should be evaluated, as it may demonstrate skeletal abnormalities related to the disorders (i.e., deviation of the mandibular and maxillofacial bones). The integrity of the jaws should be palpated. It is important to assess the mandibular range of motion (ROM), because if ROM is significantly reduced, special consideration should be taken prior to general anesthesia (i.e., tracheostomy or fiberoptic guided intubation).[3]

Diagnostic imaging

Although skull radiographs may be suggestive of TMJ ankylosis, their diagnostic yield for TMJ disorders, particularly for TMJ ankylosis or pseudoankylosis, is poor, and skull radiography is no longer recommended.[2,9] Conventional computed tomography (CT) or cone-beam CT (CBCT) is currently the gold-standard diagnostic modality for TMJ ankylosis and pseudoankylosis.[2,4,5,9] CT findings of TMJ ankylosis may include reduced or absent joint space with irregular enlarged joint margins (Fig. 38.2).[2,4,5] Bone proliferation may completely obliterate the joint space and bridge the condylar process and the squamous part of the temporal bone.[4] Bone proliferation can also fuse the TMJ with the coronoid process and/or the zygomatic arch (Fig. 38.3). In severe cases, bone proliferation may include the base of the skull (Fig. 38.4). Secondary TMJ osteoarthritis is common.[2,4] Pseudoankylosis may be manifested as bone and/or fibrous proliferation and bridging of the zygomatic arch and coronoid process or the maxillary bone and the mandibular bone (Fig. 38.5).[2,3,10] Three-dimensional (3D) volume rendering of the CT images is essential for spatial understanding of the complexity and extent of the ankylosis, and for surgical planning (Fig. 38.6).[2,3] In addition, 3D printing is a powerful tool for surgical planning, resident training, and communicating to the owner the extent of the disorder and the treatment plan.[11]

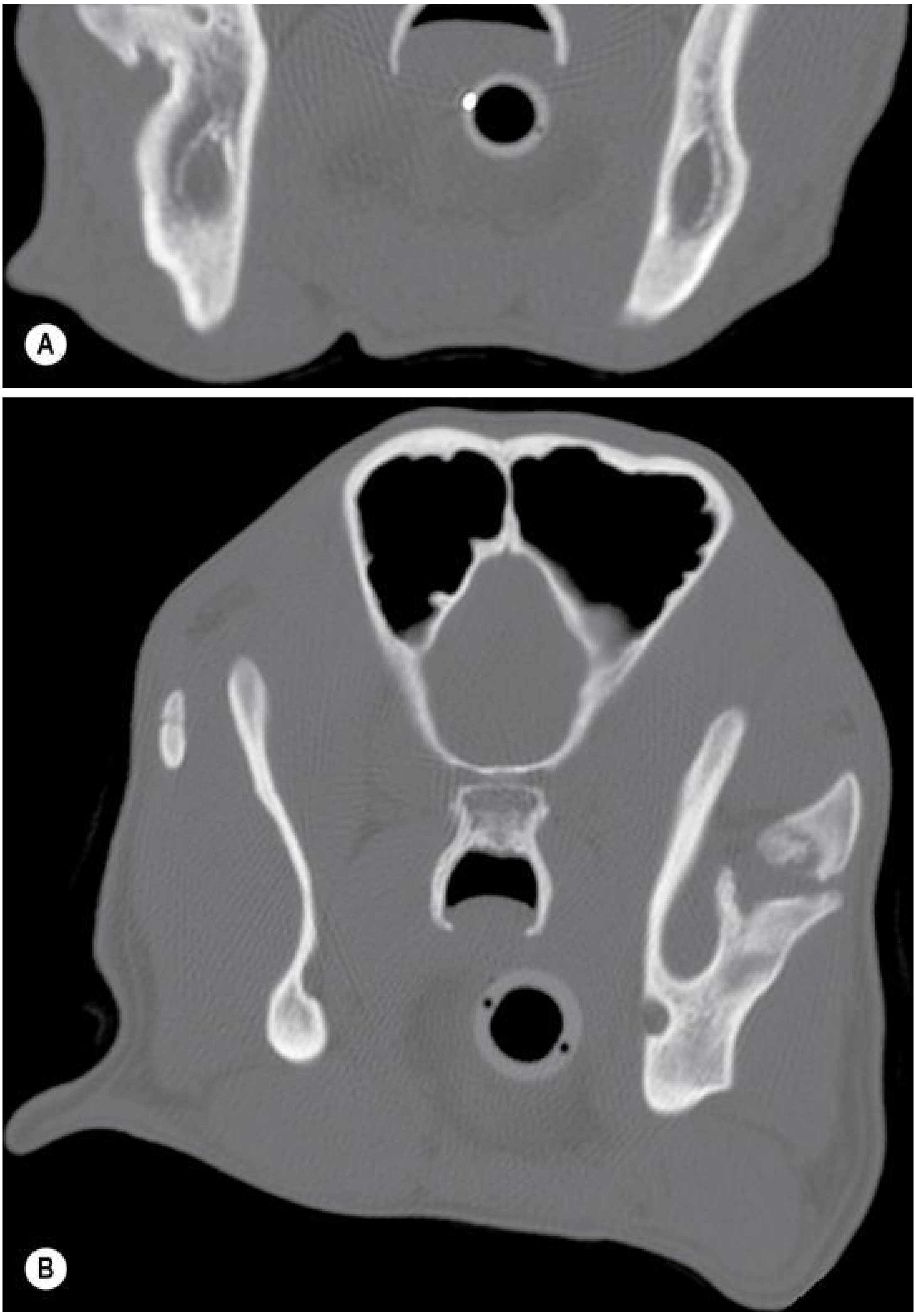


• **FIG. 38.2** Computed tomographic findings of unilateral temporomandibular joint ankylosis demonstrating reduced joint space with irregular, enlarged joint margins.



• FIG. 38.3 (**A**) Three-dimensional reconstructed computed tomographic images of a cat with bilateral temporomandibular (TMJ) ankylosis including fusion of the TMJ with the coronoid process and the zygomatic arch. (**A**) Right lateral view. (**B**) Dorsal lateral view.

• **FIG. 38.4** Computed tomographic axial image depicting a severe unilateral temporomandibular joint ankylosis with bone proliferation incorporating the base of the skull.



• **FIG. 38.5** The most common form of pseudoankylosis is manifested as bone and/or fibrous proliferation that results in bridging of the zygomatic arch and coronoid process. Note that bone proliferation may occur, (**A**) bridging the zygomatic arch and the coronoid process or (**B**) bridging the ventral aspect of the mandible and the zygomatic arch.

• **FIG. 38.6** Three-dimensional volume rendering of computed tomography images of a dog with extensive unilateral temporomandibular joint ankylosis secondary to craniomandibular osteopathy.

Surgical anatomy

The approach to the TMJ is described in detail in Chapter 29. It is important to note that, due to the ankylotic changes, aberrant anatomy may be present. The zygomatic arch forms the origin for the masseter and part of the insertion of the temporal muscles.[1,12] The caudal portion of the zygomatic arch forms the mandibular fossa and participates in the TMJ articulation. The zygomatic arch is approached by an incision made along its ventral border. The palpebral and dorsal buccal branches of the facial nerve lie near the dorsal and ventral borders of the zygomatic arch, respectively. The zygomatic and deep sphincter colli muscles, small sensory branches from the mandibular nerve, and small branches of the transverse facial artery and vein cross the incision site.[13] The superficial and middle layers of the masseter muscle originate along the ventral border of the zygomatic arch. The origin of the deep layer of the masseter is intermingled with the lateral aspect of the temporal muscle.[1,12] The temporal muscle inserts on the medial and lateral aspects of the coronoid process to surround the process. Its lateral ventral extent is the ventral aspect of the masseteric fossa.[13] The muscle contacts the pterygoid muscles on the medial aspect of the coronoid process. The body of the masseter muscle lies in the masseteric fossa of the mandibular ramus. The rostral border of the coronoid process, the coronoid crest, is thick cortical bone. Importantly, the maxillary artery lies just medial to the condylar process of the mandible.

Surgical decision making

Differential diagnoses

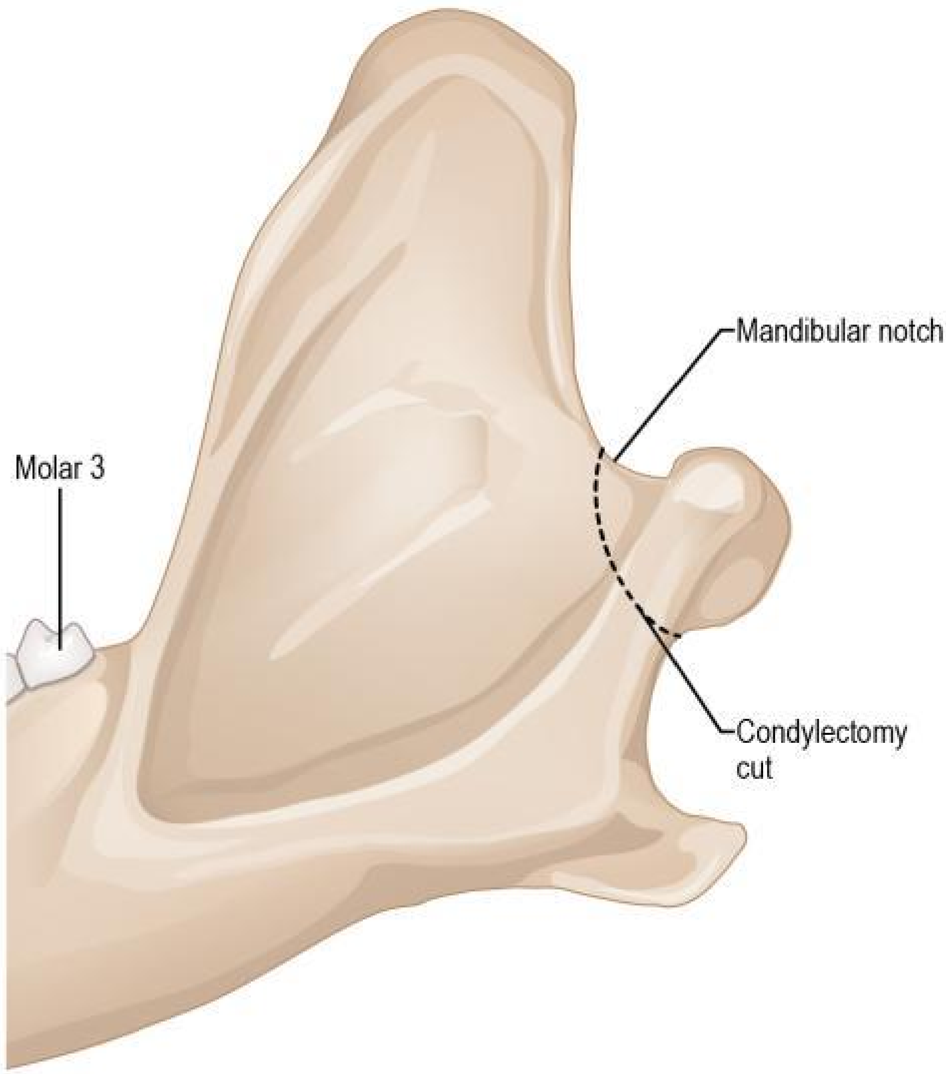
Differential diagnoses include conditions that result in inability to open the mouth. These include traumatic unilateral or bilateral TMJ subluxation or luxation, mandibular fracture, oral foreign body, retrobulbar disease, neoplasia, zygomatic sialocele, and masticatory muscle myositis.

The goal of surgical treatment is to release the ankylosis and prevent recurrence.[1,3] In that regard, the exact surgical plan will depend on the extent of, and the bones that are involved in, the ankylosis. Surgical osteoplasty of the TMJ or the pseudoankylosis is aimed at removal of excessive fibrous and/or bony tissues.[1] The surgeon should be prepared to remove enough bone so that the ankylosis will not recur. This is especially relevant in young patients, as bone growth and turnover rates are higher than those of adults. Therefore, when planning to release TMJ ankylosis, gap arthroplasty is recommended over condylectomy. Repeated forced opening of the jaws ("brisement forcé") combined with corticosteroid treatment is ineffective and not indicated, as reankylosis invariably occurs.[14]

Surgical techniques

Condylectomy

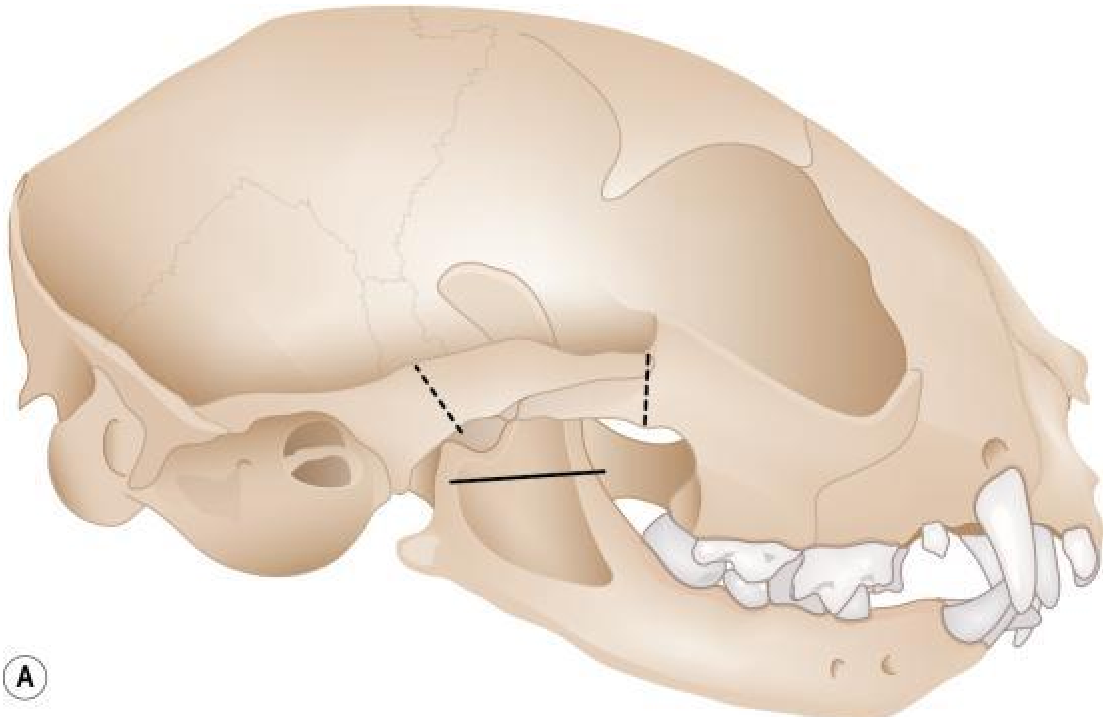
Condylectomy is performed for early TMJ ankylosis or ankylosis without excessive bone proliferation. The ostectomy is made from the mandibular notch in a caudal and ventral direction (Fig. 38.7), in a slightly semilunar configuration in order to ensure complete excision of the condylar process. It is highly recommended that the ostectomy be performed using a piezotome (see Chapter 9) to prevent damage to the maxillary artery that lies just medial to the condylar process. However, a small bone-cutting bur or an osteotome and mallet may be used with care. Due to the risk of damage to the surrounding soft tissues, and difficulty irrigating the area with subsequent risk of necrosis at the ostectomy site, an oscillating saw should not be used to perform condylectomy. After ostectomy, the capsule and remaining attachment of the lateral pterygoid muscle are subperiosteally elevated and removed from the condylar process. Osteoplasty is performed to remove sharp bone edges as needed.



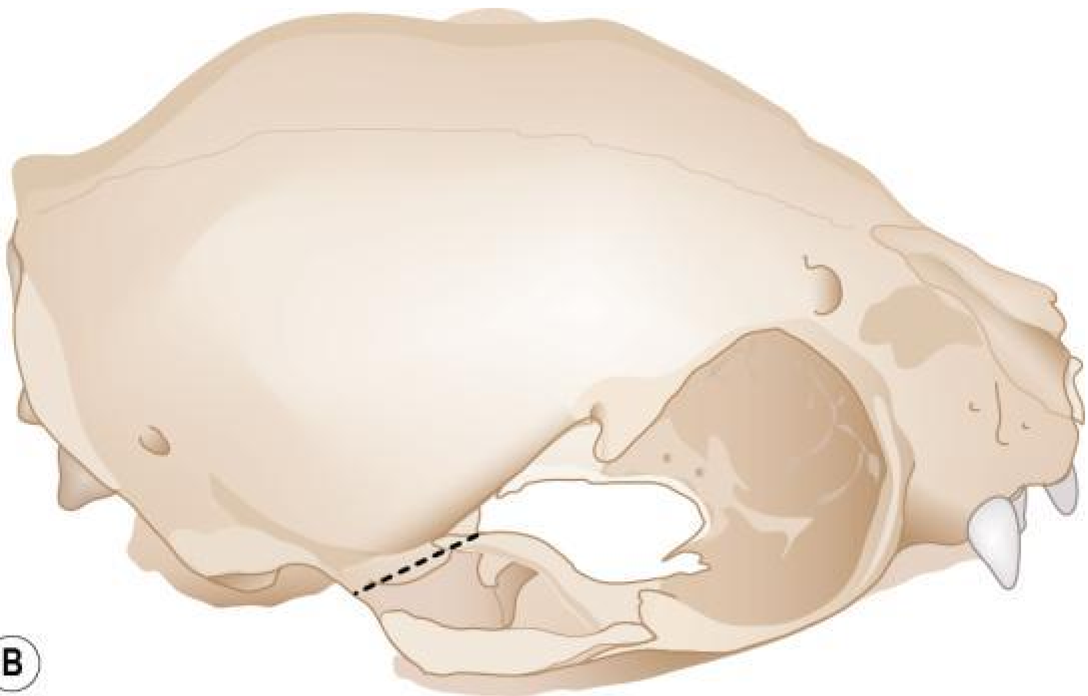
• **FIG. 38.7** The ostectomy for condylectomy is performed from the mandibular notch in a caudal and ventral direction.

Gap arthroplasty

Gap arthroplasty is performed for extensive TMJ ankylosis, or ankylosis in which the excessive bone proliferation involves other bones such as the zygomatic arch, the coronoid process, and/or the temporal bone. When multiple bones are affected, the first step, zygomectomy, is performed by making two osteotomies rostral to the TMJ (Fig. 38.8A) and removing a section of the zygomatic arch. The second step, coronoidectomy (and condylectomy), requires osteotomy ventral to the condylar process extending rostrally in a direct line, avoiding damage to the mandibular foramen. The third step, fossectomy, requires osteotomy at the level of the medial aspect of the mandibular fossa, at the juncture of the zygomatic arch and the cranium (at the level of the retroarticular process) (Fig. 38.8 B and D). As with condylectomy, it is highly recommended that gap arthroplasty be performed using a piezotome. After the ostectomies, osteoplasty is performed to remove sharp bone edges as needed.



A



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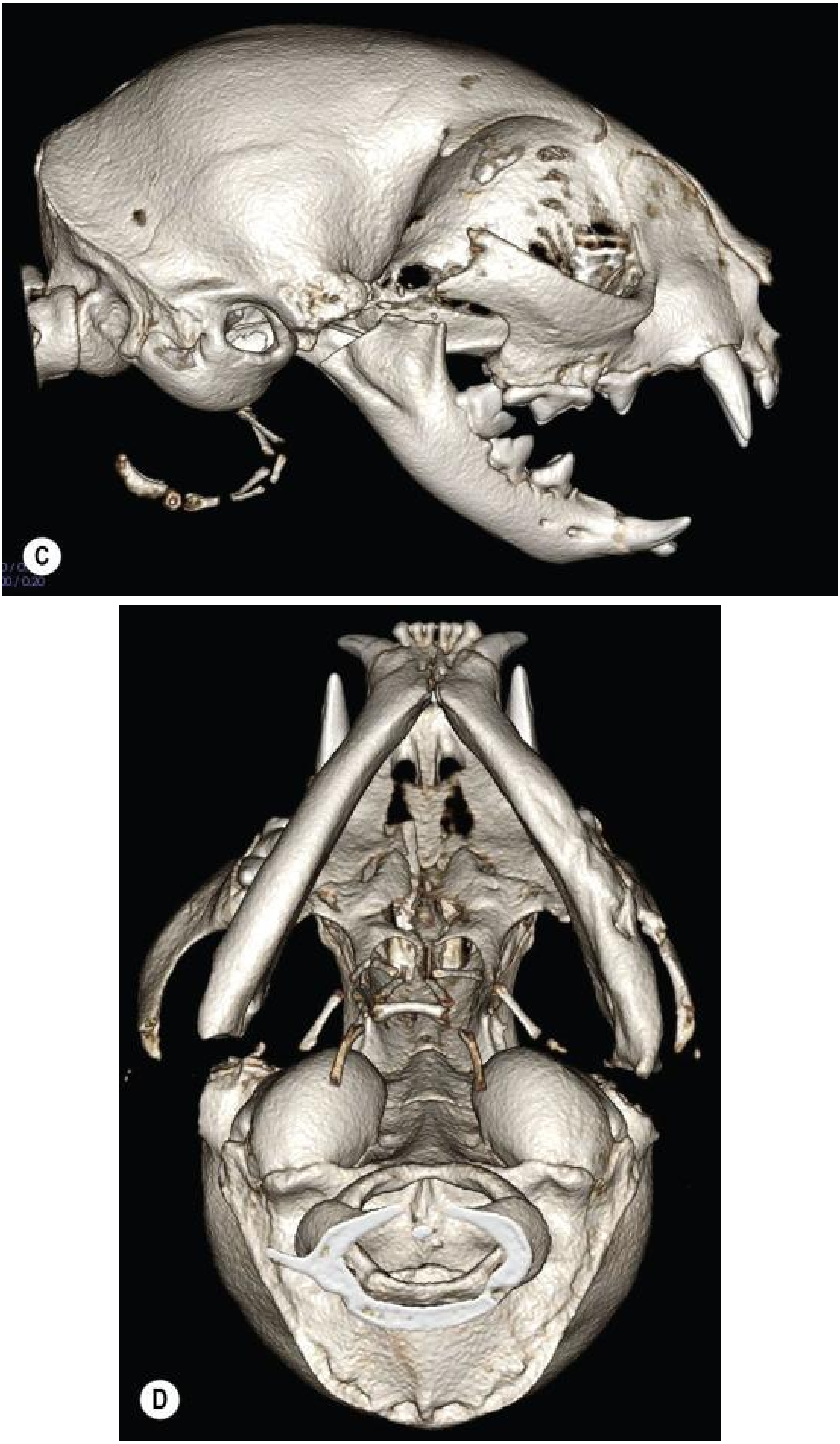


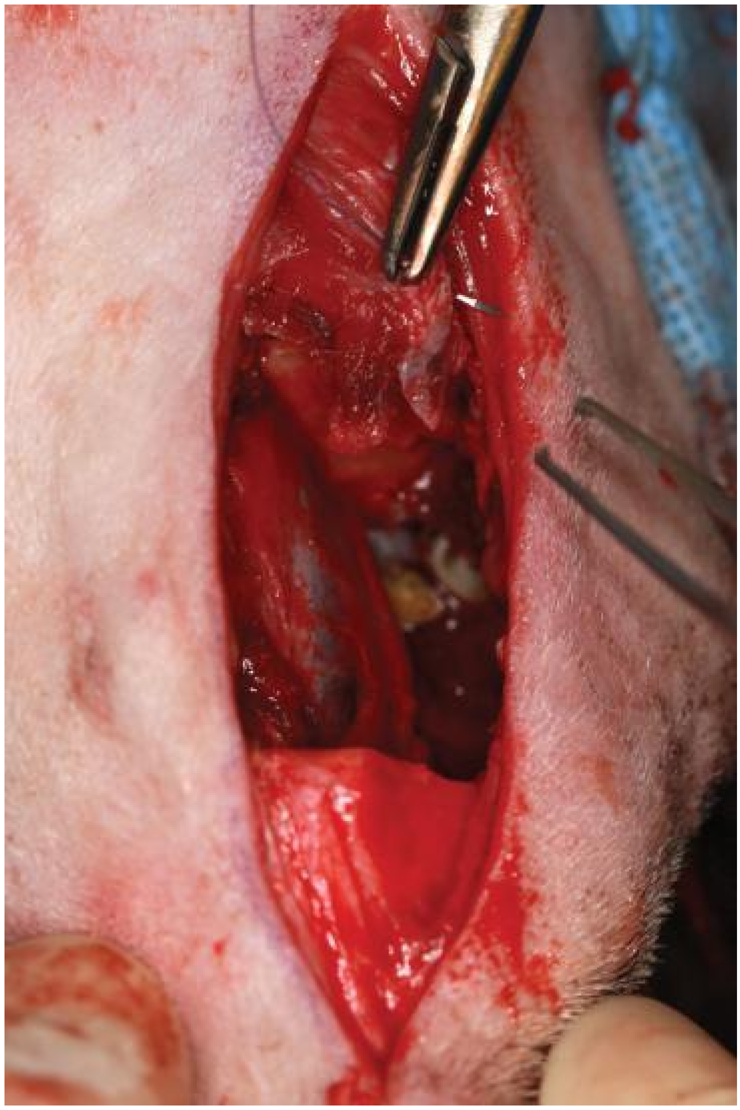
• FIG. 38.8 Gap arthroplasty is performed in three steps: zygomectomy, coronoidectomy, and fossectomy, in that order. (**A**) Two osteotomies of the zygomatic arch are made rostral to the TMJ and just caudal to the frontal process of the zygomatic bone (*dotted lines*) preserving the orbital ligament. The second step, coronoidectomy (including condylectomy), is performed by osteotomy ventral to the condylar process extending rostrally in a direct line, avoiding damage to the mandibular foramen (solid black line). (**B**) Osteotomy at the level of the medial aspect of the mandibular fossa, at the juncture of the zygomatic arch and the cranium (at the level of the retroarticular process), allows removal of the temporal fossa (*dotted line*). (**C**) Lateral and (**D**) ventral three-dimensional volume rendering of postoperative computed tomographic images, demonstrating completed bilateral gap arthroplasty in a cat.

Surgical ostectomy and osteoplasty for pseudoankylosis

Surgical osteoplasty is performed to relieve pseudoankylosis in which the excessive bone or fibrous proliferations involve bones such as the zygomatic arch and the coronoid process or the mandibular and maxillary bones. The most common form of pseudoankylosis is fusion of the zygomatic arch and the coronoid process of the mandible; the condition may be unilateral or bilateral. In this type of pseudoankylosis, the first ostectomy is made at the zygomatic arch rostral to the TMJ and caudal to the frontal process of the zygomatic bone to avoid damaging the orbital ligament. The second osteotomy, to remove the coronoid process, is made at the level of the mandibular notch and extended rostrally in a direct line (i.e., coronoidectomy), avoiding damage to the mandibular foramen. It is highly recommended that the ostectomy be performed using a piezotome to prevent damage to the soft tissues and neurovasculature of the region. However, a small bone-cutting bur may be used with care. Oscillating saws should not be used. After the ostectomy, osteoplasty is performed to remove sharp bone edges as needed.

Salvage mandibulectomy

In situations where TMJ ankylosis is extensive and the bone proliferation extends to include the base of the skull, gap arthroplasty or condylectomy may not be suitable or may present a substantial risk of damage to important adjacent structures (i.e., blood vessels and nerves exiting the brain). In these cases, salvage segmental mandibulectomy may be a reasonable approach. In this procedure, an extraoral approach is utilized to access the mandible and remove a substantial segment, such as amputation of the mandible including the mandibular fourth premolar and first molar teeth (Fig. 38.9). In turn, this will allow the patient to open the mouth, albeit with a mandibular drift, and have an acceptable quality of life.



• FIG. 38.9 For severe temporomandibular joint ankylosis in which gap arthroplasty or condylectomy may not be suitable, or presents a substantial risk of damage to important structures, salvage segmental mandibulectomy may be performed using an extraoral approach.

Complications

Intraoperative complications

Intraoperative hemorrhage and nerve injury may occur during dissection medial to the TMJ with inadvertent injury to large neurovascular structures in this area such as the maxillary artery. This can be prevented by careful and controlled ostectomy when removing the condylar process, preferably using piezosurgery.

Postoperative care

Postoperative care should include a multimodal approach to analgesia, such as a combination of opioids, nonsteroidal antiinflammatory medication, and gabapentin, for a period of 7–10 days. In addition, the patient should be offered soft food for the immediate postoperative period (i.e., first 3–5 days). However, hard food should be gradually offered after the first 3–5 days to encourage joint motion and to help prevent recurrence. The skin sutures should be removed 10–14 days postoperatively.

In addition, if, following gap arthroplasty, condylectomy, or salvage mandibulectomy, dental malocclusion develops, extraction or crown reduction of teeth causing soft tissue trauma should be considered.

Prognosis

The prognosis for surgical correction of TMJ ankylosis and pseudoankylosis is linked to the extent of the surgery. The prognosis is generally good if enough bone was removed and no damage to nerves has occurred.[3,8,10,14]

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SECTION 8:

Palate Surgery

OUTLINE

CHAPTER 39

Biologic basis of cleft palate and palatal surgery

Kevin M. Kelly

Definitions

Apoptosis: programmed cell death; cells die at particular points during the developmental process.

Cleft: an abnormal opening in an anatomical structure resulting from failure of parts to fuse during embryonic development.

Full-thickness flap: a mucoperiosteal flap reflected from the alveolar process and adjacent palatal bone containing the epithelium, connective tissue, as well as the periosteum.

Palatoplasty: a surgical procedure used to repair/reconstruct the palate in a patient with cleft palate.

Primary palate: the lip and palate rostral to the palatine fissures; includes the lip and alveolar process.

Second intention: repair of an open wound through the formation of granulation tissue and epithelialization.

Secondary palate: the structures caudal to the palatine fissures, including the hard palate (excluding the incisive bone) and the soft palate.

Split-thickness flap: a mucosal flap reflected from the alveolar process and adjacent palatal bone containing the epithelium and a layer of connective tissue; the periosteum remains on the donor site.

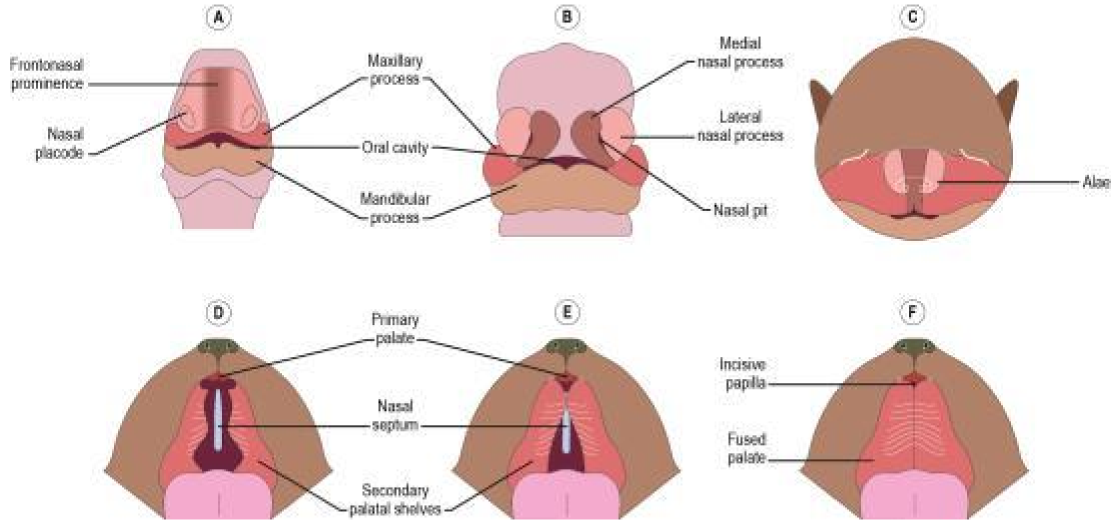
Teratogen: an agent that can cause malformations in an embryo or fetus.

Research in animals has immeasurably improved our knowledge of palatal clefts. Surgical studies using dogs, in particular beagles, have been the basis for understanding and improving the techniques of cleft palate repair. During the author's career, he had the good fortune to have been involved in a number of experimental animal studies designed and performed for the explicit purpose of assessing and improving the surgical procedures used in the treatment of cleft lip and cleft palate in humans.[1-9] The discussion that follows is a compilation of the lessons learned through efforts to improve surgical management.

This chapter is laid out in three parts. The first section describes the normal development of the primary and secondary palate, the anatomical basis for cleft lip and palate, and some of the factors known or suspected to be involved in their etiology. The middle section briefly discusses several of the better-known techniques for cleft palate repair, the philosophy of cleft palate repair, and some of the controversies surrounding surgical correction. The third section reviews what we have learned from surgical studies of cleft palate repair in dogs and cats.

Palatogenesis

In normal palate development (Fig. 39.1), mesenchymal cells from the neural crest migrate to the primitive oral cavity forming the face and the palate in association with the craniopharyngeal ectoderm. Once in situ, local processes induce these cells to differentiate into the bony and soft tissue structures that ultimately form the primary and secondary palate. Although the exact location of the boundary between the primary and secondary palate is controversial[10] and the timing of the appearance of specific structures varies between cats and dogs,[11] the formation of these structures follows similar pathways. The first of the pertinent oral structures to form is the incisive bone. The incisive bone results from the fusion of the paired medial nasal processes. These nasal processes fuse with the maxillary processes beginning at the palatine fissures to form the upper lip, alveolar process, and primary palate. The alveolar process forms through the fusion of the bilateral maxilloincisive suture lines, followed by closure of the rostral nose and the upper lip. The secondary palate forms from the eventual fusion of the palatal processes from the maxillae. In normal palatogenesis, the palatal processes grow vertically down the side of the tongue, then elevate from a vertical to a horizontal position above the dorsum of the tongue. Following elevation, the palatal processes fuse, first with the incisive bone at the maxilloincisive suture and then with each other, forming the median palatine suture. Separation of the oral and nasal cavities is completed by fusion of the dorsal surface of the fused palatal processes (now the hard palate) with the vomer, which also forms a portion of the nasal septum. Once the hard palate is formed, the soft palate and finally the uvula are formed.



• FIG. 39.1 Palatogenesis: graphic representation of formation of the primary and secondary palate in dogs. During development the frontonasal prominence, paired maxillary processes and paired mandibular processes surround the embryonic oral cavity (**A, B**). Paired nasal placodes arise as thicken ectoderm from the frontonasal prominence (**A**). As development progresses, the nasal placodes (**A**) form from the paired medial and lateral nasal processes (**B**) thus establishing the nasal pits (**B**). The lateral nasal processes give rise to the nasal alae (**C**) while the medial nasal processes (**B**) merge with the maxillary processes (**A, B**) to form the flews (upper lip in humans) and the primary palate (**D, E**). The primary palate (**D, E**) gives rise to the incisive bone located rostral to the incisive papilla (**F**). Fusion of the secondary palatal shelves (**D, E**) separates the open oronasal space (identified by the visible nasal septum) (**D, E**) into separate oral and nasal cavities. The hard palate (**F**) forms from fusion of the secondary palate (**D, E**), rostrally, with the primary palate (**D, E**) and, dorsally, with the nasal septum (**D, E**).

Palatal clefts

A cleft is an abnormal fissure in a body structure resulting from failure of parts to fuse during embryonic development.[12] Palatal clefts in dogs and cats, and in a variety of other species, fall under two categories: congenital and induced. Congenital clefts include malformations that spontaneously develop in the uterus without intentional extrinsic intervention. Induced clefts include deformities that are intentionally created using teratogens or surgery. Congenital clefts are further classified as either syndromic or nonsyndromic. Clefts are considered nonsyndromic when they occur with no other physical or developmental anomalies except the cleft lip and/or cleft palate and there is no known exposure to teratogens.

Cleft palate (or *palatoschisis*) involves the structures of the secondary palate. The severity of a palatal cleft can range from a simple fissure involving only a small portion of the caudal soft palate to a complex through-and-through defect to the nasal cavity involving the soft palate, the hard palate caudal to the palatine fissures, and the vomer (in the nasal cavity).

Cleft lip (or *cheiloschisis*) is a defect involving the structures of the primary palate. The cleft is a fissure in the soft tissue that extends from the border of the upper labia at the muzzle and continues dorsally into the upper portion of the lip toward the nostril. An incomplete cleft lip may appear only as a notch or may extend toward but not into the nostril. A complete cleft of the lip will include all the lip and continue into the nostril. A cleft lip may be unilateral, affecting only one side, or bilateral, affecting both sides. A complete cleft lip is usually but not always accompanied by a cleft of the alveolar process. The cleft may consist only of a slight notch in the alveolar process or it may extend completely through the dental arch.

Of the two conditions, cleft lip and cleft palate, cleft palate is the more serious, as communication between the oral and nasal cavities causes difficulties with feeding as well as difficulties with speech in humans. However, cleft lip and cleft palate often occur together; approximately 85% of infants with a bilateral cleft lip and 70% of infants with unilateral cleft lip also have cleft palate.[13] Cleft lip can interfere secondarily with palate closure. For that reason, the presence of a cleft lip, with or without cleft palate, has generally been thought of as one etiologic category.[14] Isolated cleft palate, on the other hand, appears to have a distinct etiology. That distinction having been made, recent epidemiological studies have brought the etiological bases of this classification into question but have not lessened their anatomical/surgical implications.[15]

Congenital clefts

The crucial and defining feature of congenital palatal clefts is the failure of embryonic structures to fuse. If the nasal prominences fail to merge with one side of the maxillary prominence, a unilateral cleft lip will result. If the structures fail to merge on both sides, two fissures are formed, resulting in a bilateral cleft lip. If the palatine processes fail to fuse, a cleft palate is formed. The failure of these structures to fuse, in part or in total, results in the range of anomalies that fall under the heading of palatal clefts.

Fusion occurs between specific epithelial surfaces at crucial times during development. This suggests that localized, cellular changes must occur in areas of fusion prior to their contact.[12,16-22] For example, midline epithelial cells manifest morphological signs of necrosis, probably involved with a programmed lysosomal-mediated autolysis.[19] It has also been suggested that seam degeneration occurs by transformation of basal medial edge epithelial cells to mesenchyme.[20] Another hypothesis is that there is no apoptosis or cellular transformation. Instead, epithelial cells migrate out of the seam and are recruited into, and constitute, epithelium on the oral and nasal aspects of the palate.[21]

Although the precise etiology of congenital palatal clefts is unknown, genetic as well as environmental factors (referred to as a multifactorial etiology) have long been known to be involved.[15,22] Given the complex embryology of the lip and palate, these tissues are affected by a variety of factors. Palatal clefts have been associated with chromosomal aberrations, genetic variations, and environmental teratogens as well as mechanical forces. Indeed, any factor that disrupts the movement and/or fusion of the primary and/or secondary palate will lead to a cleft.[12]

Genetic factors

There is wide agreement that genetic factors are of primary importance in the etiology of clefts. In fact, the search for genes associated with palatal clefts has been an active and productive area of research.[12,15] In humans, variations in the prevalence of palatal clefts among ethnic groups as well as familial recurrences provides strong evidence for genetic differences in susceptibility. Regions of several chromosomes along with several specific loci have been identified as likely candidates for the genes for cleft lip with and without cleft palate.[23] Palatal clefts have been associated with numerous loci in the mammalian species include "Sonic Hedge Hog" (SHH), Msx1, transforming growth factor (TGF)-α, and TGF-β3.[12,15,18,24-27] In addition, a number of specific genes have been identified that alter signaling molecules, transcription factors, or growth hormone in the developing face.[12,13,23] An error in the replication and/or transcription of any of these could easily result in the formation of a cleft. However, to date, no single gene has been identified which explains all or most clefts.[12,15]

Environmental teratogens

Environmental teratogens cause palatal clefts by disrupting the normal developmental process at a crucial stage of development. Teratogens may be included under the general heading of genetics because agents interact with susceptible genotypes to produce the malformations.[28,29] In addition, the timing of the exposure is also critical, as recently demonstrated experimentally.[30] For example, a number of studies in humans have linked cleft lip and cleft palate to medications taken during the first trimester, a crucial period of development for the lips and palate.[13,27] A great variety of chemical and pharmaceutical agents have been identified as causing cleft formation as well as other craniofacial malformations.[30,31]

Mechanical forces

One additional possible mechanism for congenital cleft formation is mechanical interference with the normal tissue movement and fusion. The tongue might obstruct the movement of palatal structures, resulting in a cleft.[32] The extent of the malformation depends on the stage at which the interference occurred. A cleft will result if obstruction restricts the growth of the mesodermal tissue, leaving inadequate tissue for elevation and fusion, or if the tongue is not positioned properly due to a smaller chin, such as in human infants with Pierre Robin syndrome and demonstrated experimentally in mice.[33,34]

Induced clefts

Induced clefts include deformities that are intentionally created in utero using teratogens and surgical defects created either postnatally or in utero.[9,29,31,35] Cleft lip with or without cleft palate is the fourth most common human congenital birth defect and the most common human craniofacial abnormality.[36] Given its prevalence, it is not surprising that great attention has been focused on the problem. Palatal clefts are induced in the process of assessing the teratogenic effects of various agents, to study the embryonic process of cleft formation, and to create experimental surgical models with which to study surgical correction of cleft palate.

Dogs, in particular beagles, have been the preferred experimental model for orofacial surgical research, including studies of soft tissue trauma and bone growth and studies of surgical repair of palatal clefts. Surgical clefts are created by resecting the midpalatal suture along with the adjacent bone and underlying nasal mucoperiosteum. Whether or not the overlying oral mucoperiosteum is resected depends on study design.

Congenital palatal defects

Since 2011, a number of noteworthy publications have addressed the evaluation and management of congenital clefts in dogs[37-40] and/or the establishment of a congenital cleft model in dogs.[41-43] For example, studies of dogs with spontaneous congenital clefts using computerized tomographic images,[37,38] have revealed the defects to be "complex and varied,"[37] reinforcing the need for careful preoperative planning as soft tissue components may obscure the extent of the bony defect.[38] Additional recent surgical innovations include staged closure using full-thickness mucosal flaps with selective extraction of maxillary teeth[39] and, most novel, a demonstration application of three-dimensional (3D) printing for preoperative planning in dogs and cats.[40] Of particular relevance to this discussion has been the attempt to the establishment of a congenital cleft model in dogs.[41-43] First reported in the context of developing a feeding prosthesis for a strain of dog with spontaneous congenital clefts,[41] subsequent publications have documented maxillary growth of the cleft palate puppies[42] and have reported on a preliminary promising results[43] comparing experimental closure using an injection/adhesion technique to traditional two-flap palatoplasty (described below). While attempts to develop a congenital cleft model in dogs may have stalled, the recent evident increase in the number of publications addressing surgical treatment of congenital clefts in dogs suggest a great interest medical management.

Surgical correction

The objective of cleft palate (and cleft lip) repair is the rehabilitation of the individual, which is a complex problem. Two concerns clearly dominate the discussion of the surgical repair of palatal clefts in humans, namely midfacial growth and speech production.

Concerns that cleft palate repair might inhibit the growth of the midface (the maxillofacial skeleton) were first raised by Graber in the 1940s.[44] He found, based on cephalometric studies, that the severity of the midfacial growth deficiencies (specifically, growth deficits in the anteroposterior plane) were associated with the age of the primary palate repair. He observed that children whose palates were repaired at younger ages exhibited greater maxillofacial growth inhibitions than those children whose palates were closed—using an identical procedure—at older ages (5 years of age or older). Noting that palatoplasty is inherently traumatic, he further suggested that the earlier in life that this procedure is performed, the greater its effect on maxillofacial growth.[35]

Similar findings were reported by Herfert in the 1950s, who found that midfacial hypoplasia as well as secondary maxillofacial deformities occurred among patients who underwent primary palatoplasty between 2 and 5 years of age.[45-47] He hypothesized that raising the mucoperiosteal flaps disturbed growth centers on the bony palate. These disruptions, early in life, inhibited the growth of the midface.

Concerns about speech production are crucial to understanding the evolution of cleft palate repair in humans. The techniques developed to close palatal clefts in humans have evolved, in great part, to address concerns about speech production.[48,49]

Palatoplasty in humans

The goal of cleft palate surgery is to create a separation between the oral and nasal cavities. In humans, this goal additionally includes creation of a velopharyngeal valve mechanism crucial for intelligible speech. Three techniques, particularly germane to this discussion (von Langenbeck repair, the V-Y push-back procedure, and two-flap palatoplasty), are briefly described below.

Von langenbeck technique

This technique involves the surgical creation of two bipedicle, full-thickness mucoperiosteal flaps, which remain attached anteriorly and posteriorly.[50,51] The flaps are created by making bilateral incisions palatal to the alveolar margins. The margins of the cleft are incised and the flaps are elevated by undermining between incisions on both sides of the palate; the blood supply from the major palatine arteries is preserved. The flaps are approximated in the medial plane and sutured to cover the palatal defect. The procedure leaves large areas of bone exposed lateral to the mucoperiosteal flaps as well as a raw area on the nasal surface.

V-Y push-back procedure

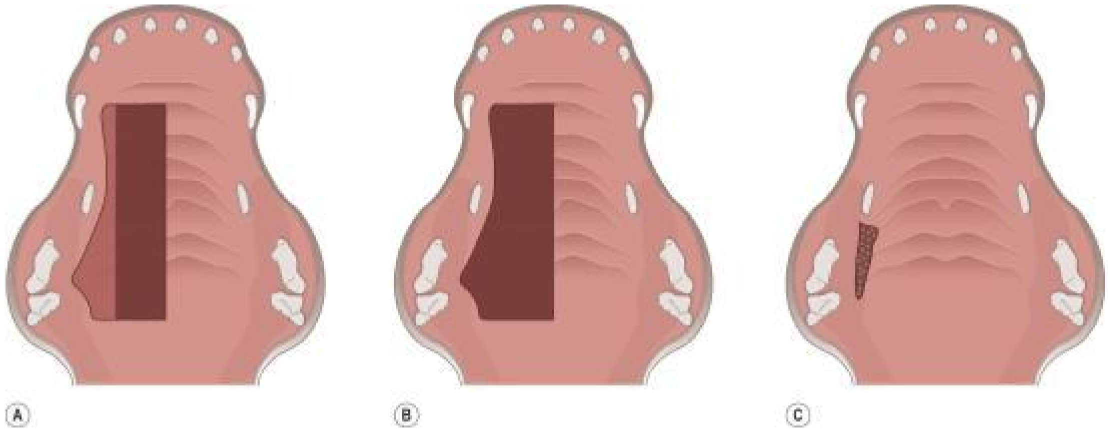
This procedure (and variations known as V-Y retroposition, the Oxford technique, the Wardill–Kilner or the Veau–Wardill–Kilner procedure) involves the surgical preparation of two single-base (unipedicle), full-thickness mucoperiosteal flaps on both sides of the palate. The flaps are elevated from the bony palate, preserving the blood supply from the palatine arteries, and retropositioned (hence, push-back), approximated, and sutured to cover the bony palatal defect and lengthen the soft palate.[52] Lengthening the soft palate, the primary goal of this procedure, is designed to allow for the development of normal speech. However, in the push-back procedures, relatively large areas of denuded bone are left on the lingual surface of the anterior hard palate.

Two-flap palatoplasty

This technique, similar to the V-Y push-back procedure, involves the surgical preparation of two unipedicle, full-thickness mucoperiosteal flaps on both sides of the palate.[53,54] The full-thickness flaps are elevated from the bony palate, taking care to preserve the blood supply from the major palatine arteries. Following elevation, the flaps are positioned anteriorly, approximated, and then sutured to cover the bony palatal defect. The objective of two-flap palatoplasty is optimal coverage of palatal bone.

Experimental research in dogs and cats

The use of dogs to assess the effects of cleft surgery on the growth of the midfacial skeleton dates back to the 1950s and the work of Herfert.[45-47] Herfert, like Graber[44] earlier, had observed midfacial growth deficiencies among his clinical patients. To test his clinical observations, he conducted a series of experimental studies using dogs, using a split-mouth design in which one side of the palate received the surgical treatment while the other side served as the unoperated control.[45-47] In the first study, he removed a narrow strip of mucoperiosteum adjacent to the alveolar margin, thereby denuding and exposing palatal bone.[45] It was found that denudation of a portion of the palatal led to maxillary growth aberrations. In the second experiment, he raised mucoperiosteal flaps that either preserved or severed the blood supply from the palatine artery.[46] He concluded that palatoplasty, mainly the elevation of the mucoperiosteal flaps and the exposure of denuded bone, interfered with facial growth, resulting in maxillofacial deformities, a conclusion later supported by Kremenak, Huffman, and Olin.[55-57] In a series of studies based on Herfert's design, it was found that excision of a strip of mucoperiosteum adjacent to the caudal alveolar process significantly inhibited maxillary growth (Fig. 39.2).[55-57]



• **FIG. 39.2** Schematic representation of three separate surgical procedures performed in studies by Kremenak and coworkers in the College of Dentistry at the University of Iowa.[47] (**A**) The mucoperiosteum is elevated to the midline; the neurovascular bundle is severed. A 4-mm-wide strip of the lateral margin of the flap is resected. (**B**) The mucoperiosteum is elevated to the midline without removal of tissue or interruption of the neurovascular bundle. (**C**) A small incision is made in the caudolateral aspect of the palate to expose and sever the neurovascular bundle.

In contrast to the findings of Herfert, Sarnat reported that elevation and partial removal of mucoperiosteum with severance of the palatal artery and partial resection of the hard palate did not result in grossly apparent growth arrest of the maxilla of the face in rhesus monkeys (*Macaca mulatta*).[58] Similarly, Lynch and Peil found that surgical creation of palatal clefts and partial resection of the vomer in puppies resulted in no significant change in growth patterns, compared with that of control animals.[59]

A major problem with experimental studies conducted by Herfert[45,46] and Kremenak, Huffman, and Olin[55-57] is the split-mouth design. The inherent problem with this approach now seems obvious. Development of one side of the facial skeleton is not independent of the development of the other, and the split-mouth design has therefore been abandoned. Since the 1970s, experimental studies of cleft repair have included one and often two, independent, whole organism controls.

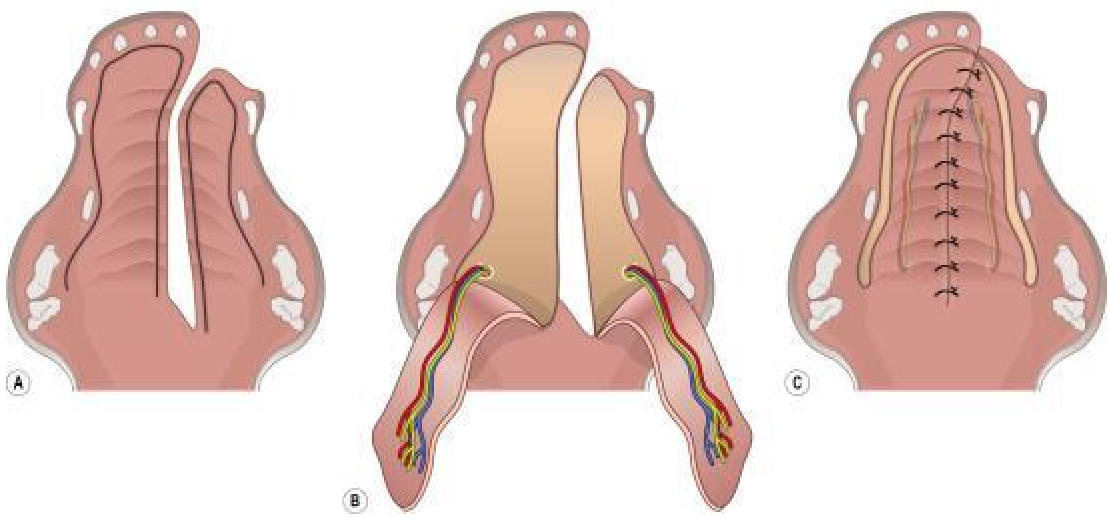
Wound contraction

Subsequent research by Kremenak and his associates focused attention on the physiologic aspects of wound healing in the palatal area.[60-63] Based on a series of experimental studies using beagles, this group postulated a causal relationship between wound contraction in the palatal mucosa and developmental deformities in the maxillofacial skeleton.[63] This hypothesis was first tested by Koopman, who found that covering exposed bone with free graft of buccal mucosa prevented wound contraction.[60] These observations were supported independently by the work of Jonsson and Stenstrom,[64] and Jonsson and Hallmans.[65] Like Koopman, these investigators found that closure of the bony palatal defect with a free skin graft effectively prevented maxillofacial growth aberrations in beagles.[64,65]

Dabelsteen and Kremenak found actin-rich cells in the granulation tissue of palatal wounds, an indication that one of the functions of these cells is contraction.[61] In a later electron-microscopic study of healing palatal wounds in beagles, Squier and Kremenak were able to identify cells with the characteristics of contractile fibroblasts, including bundles of microfilaments and intercellular junctions.[62] These studies suggest that maxillofacial growth inhibitions occur secondarily to wound contraction, a process that may aid with the convergence of wound margins.[63]

Two-flap palatoplasty

A series of experimental studies in beagles carried out by Bardach and his colleagues addressed the various clinical and surgical aspects of cleft lip and cleft palate repair.[1-9,66] Surgically created soft and hard tissue palatal deformities were repaired in combination with or in isolation from cleft lip repair.[1-5] Bony palatal defects were closed using a technique simulating two-flap palatoplasty, the procedure developed by Bardach (Fig. 39.3).[53,54] When appropriate to the research design, straight line closure was employed to repair the tissues of the cleft lip. Craniofacial growth was evaluated using direct cephalometry.



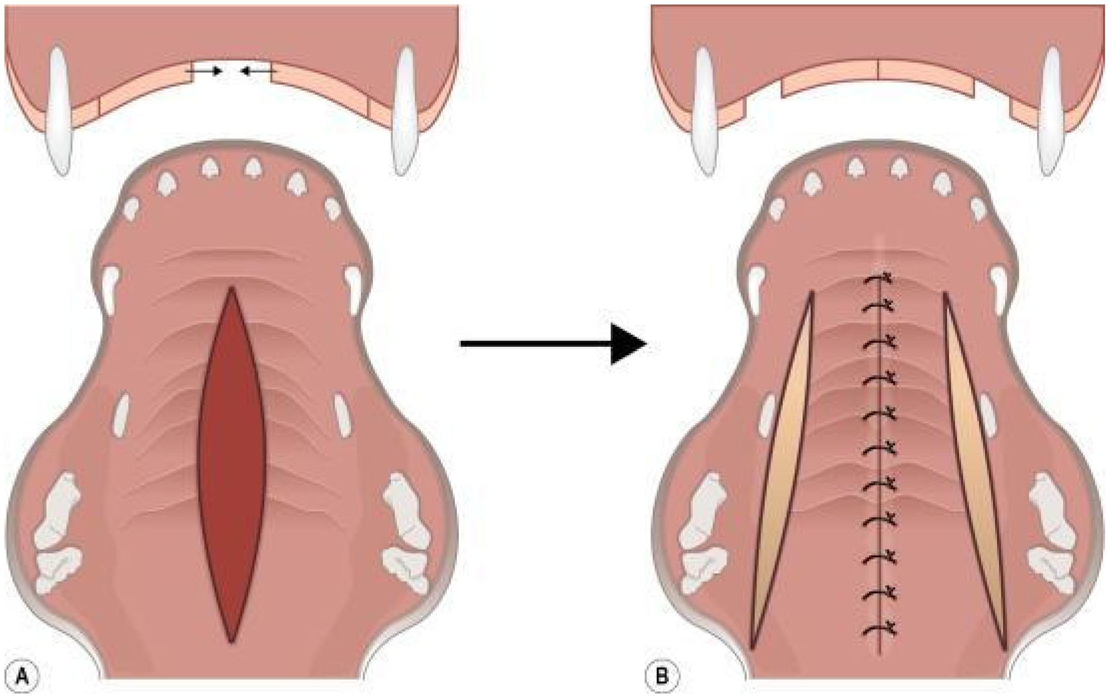
• **FIG. 39.3** Two-flap palatoplasty as performed in studies by Bardach and coworkers in the College of Medicine at the University of Iowa.[6] (**A**) A 5-mm-wide median bony defect is surgically created in the alveolar process and palate. (**B**) Mucoperiosteal flaps are raised lateral to the defect; the neurovascular bundles are preserved. (**C**) The defect is closed by approximating the medial edges of the flaps and suturing them together in midline; the procedure exposes narrow strips of bone lateral to the mucoperiosteal flaps.

This series was designed in part to evaluate the sequence of cleft lip and palate repair. The findings suggested that the commonly accepted human clinical sequence of cleft repair (lip first, palate second) is less detrimental to maxillary growth than repairing the palate first and the lip second or simultaneous closure of both defects.[1,2,4] However, it is important to note that growth aberrations were observed following closure of the lip and palate as well as following closure of the lip alone. This consistent finding lead Bardach to conclude that increased lip pressure, resulting from the lip repair, had to be considered as a factor in secondary growth aberration—equal to if not exceeding the effects of palate repair.[67] In fact, surgical palatal clefts (unilateral defects of the lip, alveolar process and palate, as well as isolated bony palatal clefts) using two-flap palatoplasty did not inhibit overall facial growth.[5,66] Moreover, two-flap palatoplasty appeared to stimulate substantial new bone formation at the site of the defects. In experiments performed to assess the effects of raising mucoperiosteal flaps and exposing palatal bone at the time of palatoplasty, it was found that raising mucoperiosteal flaps is less detrimental to craniofacial growth than leaving large areas of exposed palatal bone.[6]

The study that ultimately concluded the series explored the process of bone formation within the palatal defects.[7] No differences in the overall thickness or density of regenerated bone in surgically recreated bony palatal defects repaired using either mucosal flaps or mucoperiosteal flaps were found.[7] Histologically, a well-differentiated periosteum was present on the maxillas at 4 weeks in both groups of operated dogs. This suggests that maintaining the periosteum at surgical closure is not essential to bone regeneration. We postulated that osteoprogenitor cells, migrating from the undisturbed local periosteum adjacent to the defect, were responsible for new bone growth[7]—a hypothesis now widely accepted as fact.[68]

von langenbeck technique

Surgical closure of palatal clefts using the von Langenbeck technique[50,51] was examined in studies conducted by researchers at the University of Nijmegen in the Netherlands (Fig. 39.4).[69-80] Wijdeveld and coworkers explored age-related effects of palatoplasty in a series of articles.[69-73] In these studies, soft tissue (i.e., nonbony) defects were repaired using the von Langenbeck technique. Overall, narrowing of the dental arch, especially caudal maxillary width, was reported. Palatal surgery did not affect rostrocaudal distances on the palatal mucoperiosteum. In another set of reports, Leenstra et al. compared dentoalveolar development in beagles after palatal repair using the partially split-flap technique and the von Langenbeck method.[74-76] Palatal surgery by the partially split-flap technique resulted in significantly wider transverse maxillary arches than did the von Langenbeck procedure.



• **FIG. 39.4** Simulated von Langenbeck procedure as performed in studies at the University of Nijmegen, the Netherlands.[66] (**A**) A soft tissue defect is created in the midline by incising, elevating, and removing a fusiform mucoperiosteal flap no more than one-quarter of the transverse distance between the fourth premolar teeth; relaxing incisions are made adjacent to the teeth on both sides of the palate from the canine tooth to the caudal molar tooth. (**B**) The bipedicle flaps are elevated from the underlying bone; the neurovascular bundles are preserved. The defect is closed by approximating the medial edges of the flaps and suturing them together in midline. The procedure exposes large areas of bone lateral to the mucoperiosteal flaps.

Recent reports from this group revisit concerns about scarring and wound contraction first raised by Kremenak and his colleagues.[63,77-80] In the first series, the effect of implantation of poly-(L-lactic) acid membranes on dentoalveolar development following palatal surgery was investigated.[77-79] Soft tissue defects created in the medial palate were created in beagles by a standardized elliptical mucoperiosteal flap. The defects were closed using the von Langenbeck technique. Poly-(L-lactic) acid membranes were implanted on the denuded bone. The investigators found that implantation of the membranes did not prevent iatrogenic disturbances of dentoalveolar development.

In a most recent study, the tissue response to three collagen-based and two skin-derived substrates was compared.[80] Histology performed at 3, 10, and 20 days postoperatively showed that all substrates were well tolerated. However, wounds treated with the collagen-based substrates contained fewer myofibroblasts at 20 days. Their findings echo those of previous investigators.[60,65,66] Palatal wounds closed with a dermal substrate healed with fewer indications of scar tissue formation and only a mild inflammatory reaction, which is preferred over the tissue reaction in an untreated wound.

Studies in cats

A series of studies by Freng and Voss are noteworthy mainly for the fact that they were conducted using domestic cats.[81-88] The repair of surgically induced bone cleft defects by single (oral periosteum) and double layer (oral and nasal mucoperiosteum) closure as well as surgically created submucousal clefts in cats were studied.[81-83] Unlike procedures described above, surgical soft tissue repair did not simulate clinical techniques. Instead, the defect was closed by simply replacing the flap that had been raised to expose the bone where the defect would be created. In each study, it was found that significant bone regeneration and reduced transverse maxillary growth occurred, suggesting that soft tissue trauma and the bone bridges that formed in the surgically created palatal defect had restricted maxillary growth. These finding were largely confirmed in the series of studies that followed.[84-88] However, it was concluded that the cat's scissor bite simulated the effect of orthodontic appliances, transferring force from the growing mandibles to the maxillas, thereby accounting for the normal rostrocaudal growth of the operated maxillas as well as the concomitant transverse maxillary hypoplasia.[85-88] Thus, cats were quickly recognized as being inappropriate for cleft surgical research.[9]

Acknowledgments

The author gratefully acknowledges noted plastic surgeon, Janusz Bardach (1919–2002), whose life work and research contributed significantly to this chapter. He began working in Dr. Bardach's laboratory while completing his doctorate and then continued for many years after. What started as a job in an entirely new field, unrelated to his own dissertation, resulted in 14 years of mentoring and investigation, a much longer friendship, and a life-long interest in cranial morphology.

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CHAPTER 40

Orofacial cleft repair

Santiago Peralta, Sandra Manfra Marretta

Definitions

Cleft lip: Congenital fissure affecting only the primary palate (i.e., lip and/or incisive bone). The defects can be complete or incomplete and occur unilaterally or bilaterally.
Cleft palate: Congenital midline fissure affecting only the secondary palate (i.e., hard and soft palate). The defects can be complete or incomplete, and the hard palate defect can be unilateral or bilateral.
Cleft lip and palate: Congenital fissures affecting the primary and secondary palate simultaneously. Multiple anatomical configurations are possible based on laterality and severity of the defects.
Hypoplasia of the soft palate: Congenital fissure parallel to the median plane on the soft palate. The defect can occur unilaterally or bilaterally. When bilateral, a pseudouvula is present.
Orofacial cleft: Any congenital fissure affecting orofacial structures including lip, incisive bone, and hard and/or soft palate.

Preoperative concerns

Depending on the severity and anatomic configuration of the defects, orofacial clefts can cause significant morbidity and mortality. Untreated, affected animals may die of respiratory complications and/or malnourishment. To survive, affected animals may require special and dedicated nursing care, as well as nutritional support. In patients in which tube feeding or bottle feeding is not possible, alternate forms of alimentation should be considered, including esophagostomy and gastrostomy feeding tubes, until the pneumonia and/or severe rhinitis have resolved. For long-term survival, surgical correction of orofacial clefts is indicated. However, surgery should be delayed until the patient is adequately nourished.[1,2]

At initial presentation, common clinical signs associated with orofacial clefts include nasal discharge and increased upper and lower airway sounds, which are usually due to chronic rhinitis and/or aspiration pneumonia.[3] A complete blood cell count, auscultation of the lungs, and three-view radiographs or computed tomography (CT) of the thorax should be performed preoperatively to rule out clinical and subclinical aspiration pneumonia. Empirical systemic antibiotic therapy should be implemented if aspiration pneumonia is confirmed. In selected cases of aspiration pneumonia (i.e., recurring and/or refractory cases), a tracheal wash may be necessary to determine appropriate antibiotic therapy based on culture and sensitivity testing. Nasal bacterial and/or fungal culture and sensitivity testing and lavage of the nasal cavity prior to definitive repair of orofacial clefts may be beneficial.[3,4]

Animals with orofacial clefts should be carefully examined, and CT of the head should be performed to determine whether other concurrent congenital anomalies are present.[5] Concurrent anomalies that have been previously reported in dogs and cats with orofacial clefts include anotia, bifid nose, bifid tongue, polydactyly, malformed tympanic bullae and middle ear disease, encephalocele, hydrocephalus, cranioschisis, microphthalmia, and limb deformities, among others.[5-8] Prior to surgical correction of orofacial clefts, clients should be advised of the possible heritability factor,[9] and recommendations should be made to not use these animals for breeding.

Although significant and uncontrolled blood loss does not usually occur during orofacial cleft repair, coagulation and platelet function tests can be performed prior to surgery at the discretion of the clinician, especially in animals with a known history of coagulopathies, thrombocytopenia, or thrombocytopathy. If indicated, blood typing and cross-matching should be performed before surgery.

Anesthetic considerations

Pediatric patients may be at risk of hypoglycemia perioperatively. Fasting time, anesthetic agents used and doses, parenteral fluid administration, and blood glucose monitoring should be determined and adjusted according to individual patient needs. Rectal temperature should be monitored during surgery; a circulating warm water heating pad and/or a warm-air circulating device should be used perioperatively to help prevent hypothermia.

No guidelines have been defined for the perioperative use of antibiotics during orofacial cleft repair in dogs and cats. The use of perioperative antibiotics during orofacial cleft repair in humans is controversial, and the decision to administer is ultimately clinician dependent.[10] If a decision is made to administer perioperative antibiotics, a single dose of ampicillin/sulbactam is likely sufficient.

A wire-reinforced endotracheal tube is recommended for intubation to help prevent inadvertent occlusion while manipulating it during the surgical procedure. Patients are usually positioned in ventral or dorsal recumbency, with the endotracheal tube firmly secured to the mandible. Pharyngotomy or a transmylohioid endotracheal intubation may be performed to improve the visualization of more caudally located defects, provided the pharyngeal wall is not required for the surgical repair.[11]

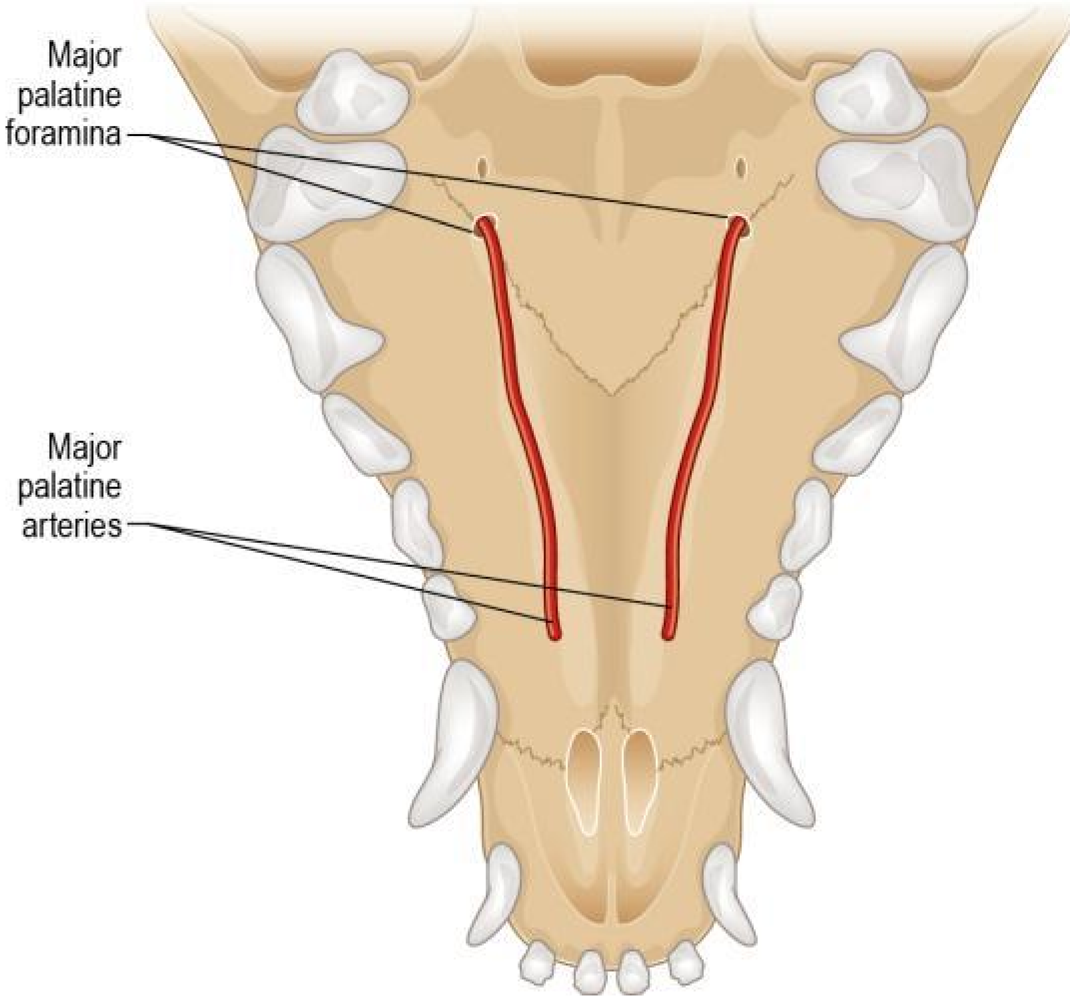
Regional nerve blocks are indicated to achieve a balanced anesthesia and as part of a multimodal analgesic approach. The technique, agent, and dose used depend on the size of the patient, the anatomical location of the defect(s), and the surgeon's preference (see Chapter 4).

The use of mouth gags should be avoided or minimized during surgery, especially in cats. The use of mouth gags may affect blood supply to the brain and has been associated with transient or permanent postoperative neurologic deficits, including central blindness, deafness, and ataxia.[12-14]

Surgical anatomy[15]

The incisive bones, the palatine processes of the palatine and maxillary bones, and the corresponding oral mucosa form the roof of the oral cavity. In general, osseous structures articulate with each other via sutures, which are composed of Sharpey's fibers during development, and eventually fuse during adulthood. The two sutures found on the hard palate are the palatomaxillary, between the palatine and maxillary bones; and the insivomaxillary suture, between the maxillary and incisive bones, in addition to the three midline sutures. Clefts of the primary palate (i.e., cleft lip) affect the lip and/or corresponding alveolar process(es) of the incisive bones. Clefts of the secondary palate (i.e., cleft palate) are associated with midline defects of the hard and soft palate.[16] The palatine fissures, two large openings between the incisive bones and the palatine processes of the maxillae, are typically located medial to the third incisor and canine teeth and can be palpated as soft areas in the rostral aspect of the palate. The caudal aspect of the hard palate is located just caudal to the maxillary second molar teeth, where it merges with the rostral aspect of the soft palate. The caudal aspect of the soft palate normally contacts the epiglottis.

The blood supply to the hard palate of the dog and cat is similar. The right and left major palatine arteries are the main arteries to the mucoperiosteum of the hard palate. The major palatine artery emerges from the major palatine canal at the major palatine foramen and courses rostrally in the shallow palatine sulcus on the surface of the hard palate (Fig. 40.1). The major palatine foramina are usually located medial to the maxillary fourth premolar teeth, although the exact location may vary slightly depending on species (i.e., cat or dog) and skull type and conformation.



• **FIG. 40.1** The major palatine arteries emerge from the major palatine foramen and course rostrally in the palatine sulci.

Special instruments and materials

A Bard-Parker no. 3 long scalpel handle with a #15 scalpel blade can be used to incise skin and/or oral mucosa. A blunt-tipped curved Freer periosteal elevator (Miltex, Lake Success, IL) or a P24G periosteal elevator (Hu-Friedy Instrument Co, Chicago, IL) is ideal for elevating the mucoperiosteal layer during hard palate defect repair. A blunt-tipped elevator combined with judicious elevation of the mucoperiosteum can help prevent inadvertent laceration of the major palatine artery as it emerges from the palatine canal at the major palatine foramen. Longer-length surgical instruments may be useful during repair of soft palate defects. Monofilament absorbable suture materials are recommended for the repair of most palatal defects. Excellent lighting and magnification using surgical loupes are essential during manipulation of the tissues. An operating surgical microscope can be very useful,[17] particularly when repairing defects in small animals (Fig. 40.2).

• FIG. 40.2 Repair of an orofacial cleft in an appropriate surgical environment. The procedure is performed in an operating room in which the surgeon has access to advanced images for anatomical orientation purposes. In this case, the patient was so small that the use of an operating microscope was necessary to enhance lighting and visualization during repair of the soft palate defect.

Under normal circumstances, orofacial cleft repair is performed by mobilizing soft tissues from the vicinity of the defects for closure. The use of grafts and/or membranes (i.e., osseous, collagen, etc.) to replace missing hard tissues, or to support mobilized soft tissues during orofacial repair, has been reported experimentally and clinically.[4,18,19] In humans, autologous cancellous bone grafts are frequently used to recreate normal local hard tissue anatomy of alveolar defects, for later dental restorative and prosthetic purposes (i.e., implant placement).[20] Perfectly recreating normal osseous anatomy is not usually necessary in small animals, as long as the oral and nasal cavities are surgically separated with soft tissues. Moreover, there are no published clinical trials demonstrating the benefits or adverse effects of grafting osseous defects associated with orofacial clefts in small animals. Therefore the routine use of grafting materials for orofacial cleft repair in dogs and cats is not recommended at this time. Advances in tissue engineering, regenerative medicine, and tridimensional printing technologies are expected to make routine fabrication of biocompatible custom-designed grafts for orofacial cleft repair feasible in the future.[21-23]

Therapeutic decision-making

In animals with orofacial clefts, the pertinent history and initial physical and oral examination are extremely important. An assessment must be made concerning the potential for successful repair of the defects and the commitment of the client to the sometimes intensive perioperative care required in these patients. Some orofacial clefts may be too severe to permit successful closure, and clients should be appropriately counseled concerning a poor prognosis. Clients should also be advised that several operations are often required to completely close the defects and that strategically extracting teeth in the vicinity of the defects may be necessary to increase the amount of tissues available for repair.[3,24] If strategic extractions are pursued prior to surgical repair, these are usually performed 4–6 weeks prior to definitive repair to allow healing of extraction sites.[1,3]

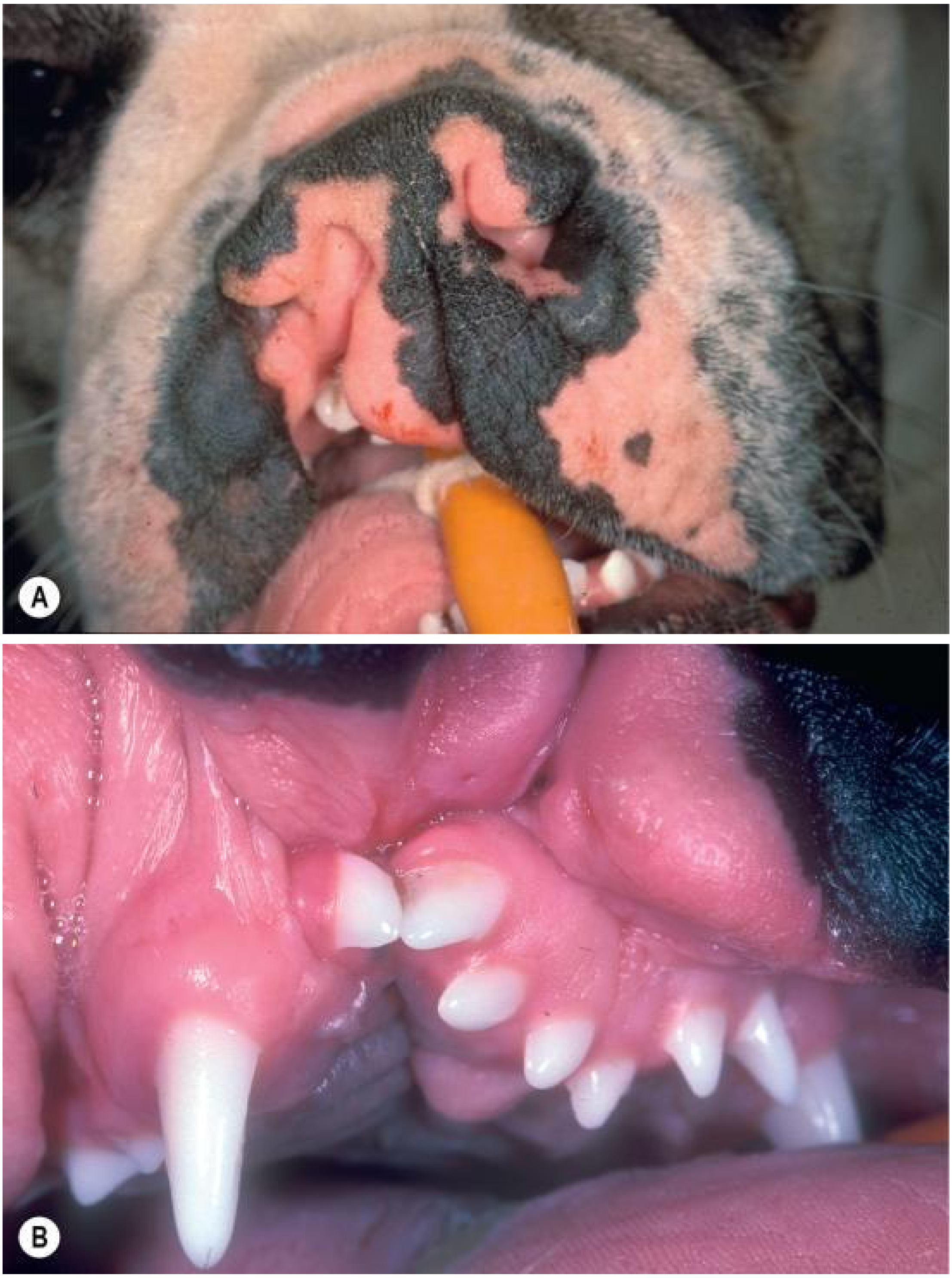
Once the decision is made that surgical defect repair is viable, the surgical technique is planned based on the anatomical configuration and severity of the defects, as revealed by a CT scan of the head. The morphological features of orofacial clefts have been shown to be quite variable and complex.[16] Moreover, the severity of the bony defects is often underestimated based on gross appearance.[5] Because of this, advanced imaging of the head before surgical repair should always be performed. Assessments of the multiplanar reconstructions and of tridimensional rendered images are essential for surgical planning and are likely to influence surgical outcome.[24]

The patient's age at the time of repair is important, and may influence the surgical outcome;[1,24] the timing of surgery should be decided based on the patient's systemic status, and anatomical configuration and severity of the defects. In general, hard and soft palate defects more frequently cause morbidity compared to defects limited to the lip and/or alveolus and thus are generally a higher surgical priority. If the patient has a history of repeated episodes of aspiration pneumonia, surgical repair of the hard and soft palate defects should not be unnecessarily delayed and can be performed as early as 3 months of age, if otherwise medically appropriate. It should be noted that performing surgery at such an early age may alter the maxillofacial growth patterns due to the surgical insult to the tissues and later scarring and retraction.[25] Additionally, if deemed necessary, strategic dental extractions (i.e., for staged repair) would not be technically feasible due to the presence of developing and unerupted permanent dentition.[3] Conversely, if the patient has been otherwise systemically healthy, surgical repair can be delayed until all the permanent dentition has erupted (i.e., 6–8 months of age). The advantages of delaying surgery include that larger areas of tissue are available for defect repair and strategic extractions prior to repair are feasible. Delaying surgery beyond 8 months of age has been associated with a higher rate of oronasal fistula formation after hard palate defect repair[24] and is therefore not recommended.

Surgical techniques

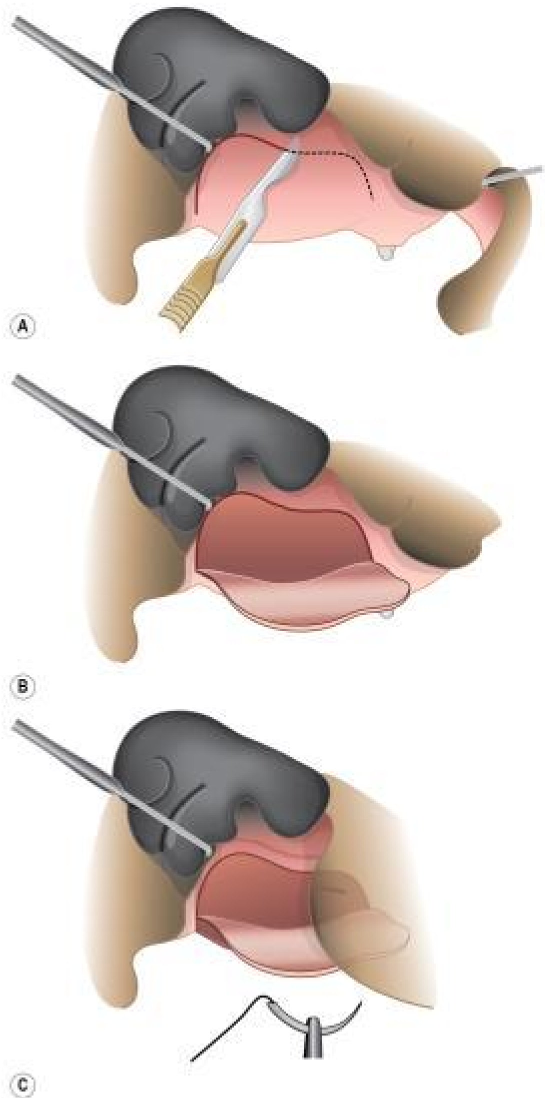
Cleft lip

Cleft lip involves fissures of the incisive bone, typically between the second and third incisor teeth, and/or the lip (Fig. 40.3A). Cleft lip can occur bilaterally and unilaterally. Cleft lip and cleft palate can occur simultaneously (i.e., cleft lip and palate). Due to the embryological sequence of events, unilateral defects of the primary palate tend to occur more frequently on the left side.[16] The defects can be complete or incomplete based on whether the entire anatomical area is affected or only a portion. Regardless, most defects involve a direct communication between the nasal and oral cavities. Surgical repair of these defects involves the establishment of the normal separation between the oral and nasal cavities. Separating the oral and nasal cavities involves reconstructing the floor of the nasal passage and closing the oral defect. In addition, malpositioned, malformed, supernumerary, and/or persistent deciduous incisors adjacent to the alveolar defect(s) are common and may require extraction prior to surgical repair (Fig. 40.3B).[1]



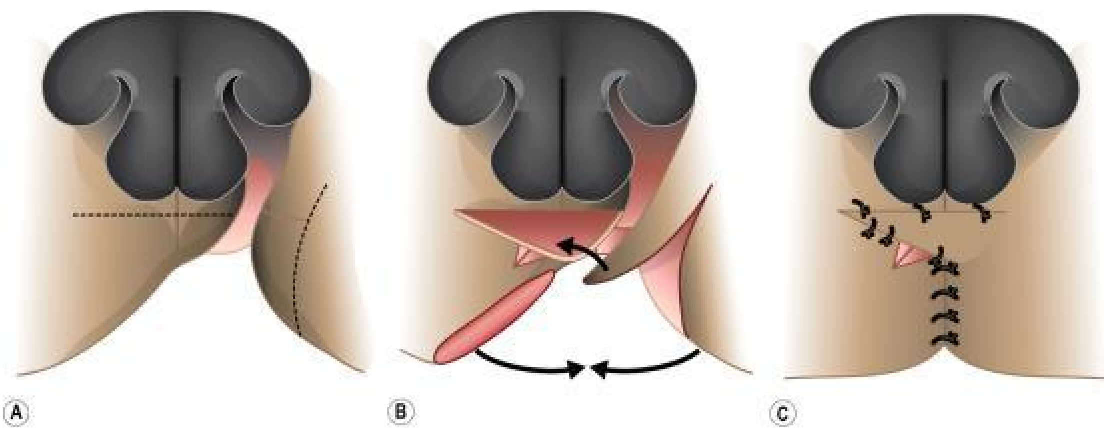
• **FIG. 40.3** (**A**) Unilateral complete cleft lip in a 4-month-old boxer. (**B**) Intraoral view showing the defect; the maloccluding left maxillary second and third incisors should be removed prior to surgical repair. Note the presence of a supernumerary incisor.

For cleft lip repair, the patient can be positioned in ventral or dorsal recumbency, depending on the surgeon's preference. After preparing the site for aseptic surgery, a pedicle flap is created from the medial nasal wall and based at the margin of the medial side of the cleft along the floor of the nares.[1] The flap is then reflected laterally to the lateral lip mucosal side of the defect.[26] An incision is made in the labial mucosa of sufficient length to receive the laterally rotated pedicle flap, which is sutured in position with 5-0 absorbable monofilament suture material in a simple-interrupted pattern (Fig. 40.4A–C).[26]

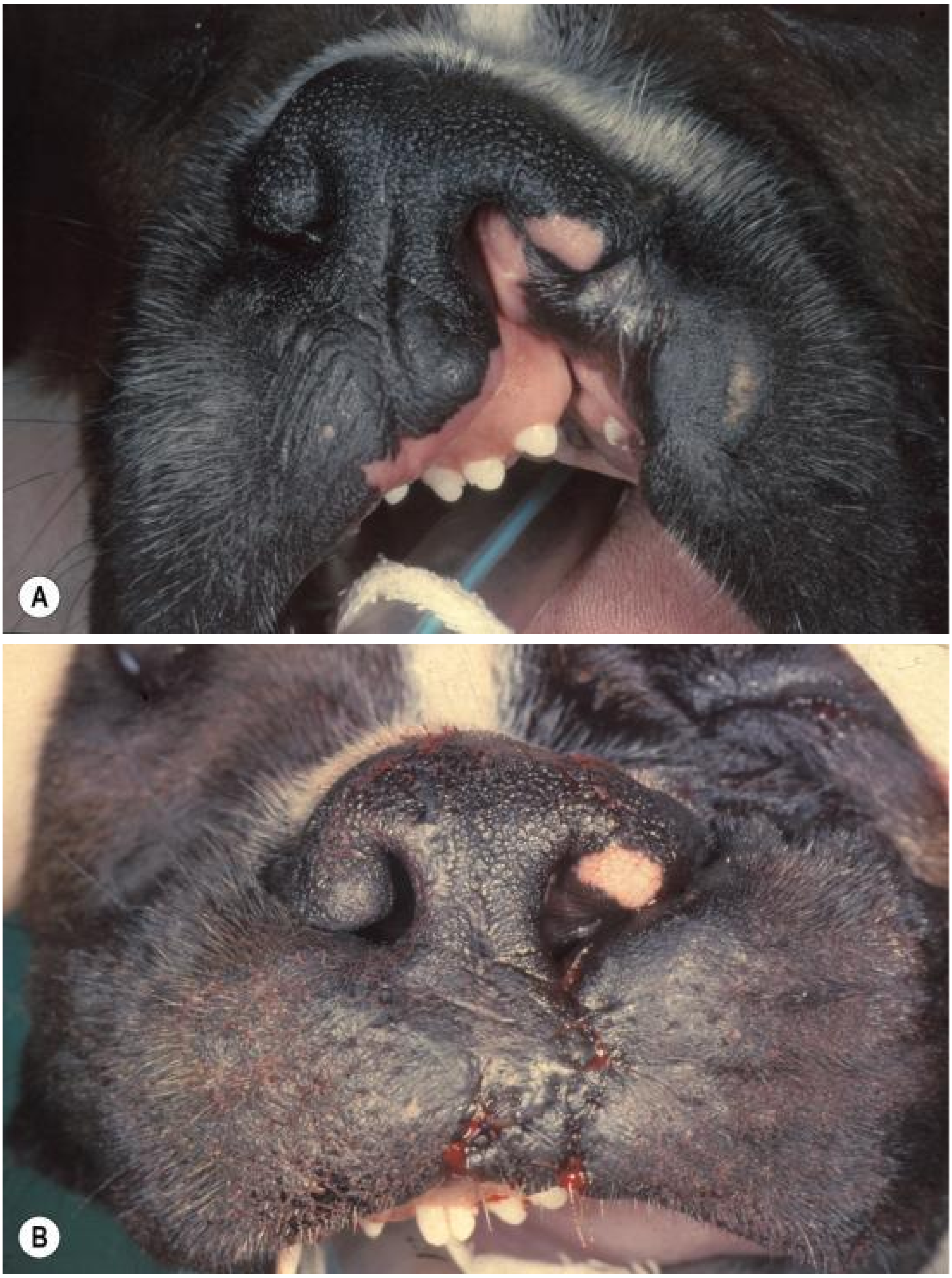


• **FIG. 40.4** A pedicle flap from the medial nasal wall is created (**A**), reflected laterally (**B**), and sutured to a prepared bed of the lateral side (**C**) forming the nasal floor or shelf.

Following the establishment of the nasal floor, reconstruction of the lip is performed. To allow closure, an incision is made in the lateral mucocutaneous margin of the lip defect. This incision is initiated at an imaginary line drawn perpendicular to the most ventral aspect of the philtrum, is extended dorsally along the lateral margin of the defect, and parallels the alar fold to the level of its commissure (Fig. 40.5A). The incision into the lateral defect margin is separated into two layers, a dermal layer and a labial mucosal layer, using blunt and sharp dissection. This permits medial advancement of the midportion of this flap. A second incision is made on the medial side of the defect in the hairy cutaneous lip beginning at the medial defect margin approximately halfway along its vertical course. This incision is directed dorsomedially toward the normal nasal opening to mobilize a rotational flap that is brought across the defect for apposition with the lateral lip margin (Fig. 40.5B). The mucosal surfaces are sutured in a simple-interrupted pattern with 5-0 monofilament absorbable suture material to create a mucosal seal with the knots located on the mucosal surface of the lip. The second or dermal layer of the closure is initiated by placing a suture from the midpoint of the laterally created dermal flap to the dorsal aspect of the medial flap using 4-0 nonabsorbable monofilament suture material (Fig. 40.5C). Additional simple-interrupted sutures are used to complete the dermal layer closure. Various modifications of this technique exist, but the basic principles remain the same (Fig. 40.6A–B).



• FIG. 40.5 The incisions utilized in the repair of a lip defect include a perpendicular incision across the philtrum and a triangular-shaped pedicle flap on the lateral side (**A**). The triangular pedicle flap is rotated across the defect (**B**) and is sutured in place (**C**).



• **FIG. 40.6** Preoperative (**A**) and postoperative (**B**) view of a cleft lip repaired using a modification of the technique illustrated in Fig. 40.5. Important aspects include facial symmetry, a straight philtrum, and a smooth, continuous lip contour.

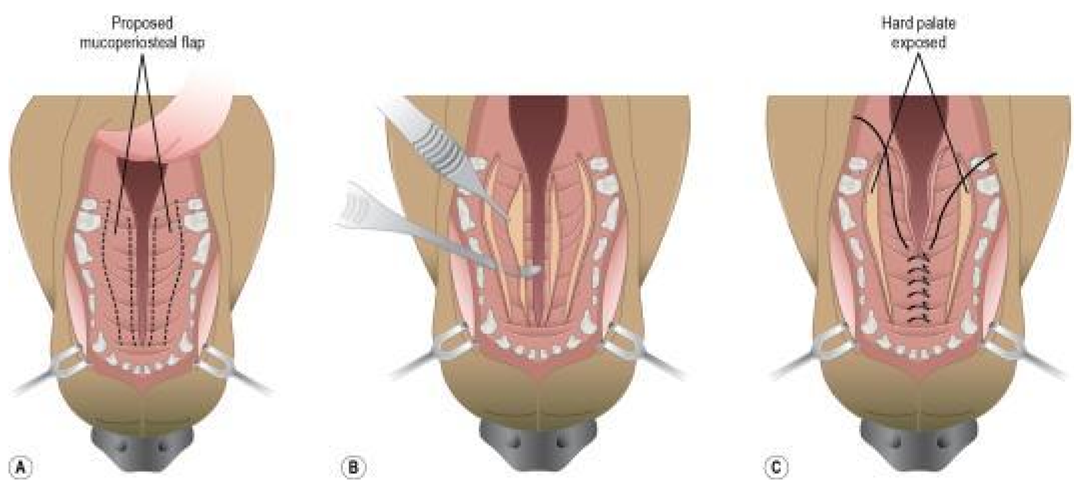
Orthodontic correction of the deviated incisor teeth adjacent to the cleft is rarely performed in veterinary medicine. Extraction of maloccluding incisor teeth is usually indicated 2–4 weeks prior to surgery, especially if the teeth involved are expected to interfere with occlusal function or with the repair of the congenital defects (Fig. 40.3B).[1,3]

Hard palate defect repair

Several techniques have been described in the veterinary literature for the repair of congenital hard palate defects, including the von Langenbeck (also known as medially repositioned double flap) and the overlapping-flap techniques.[24,27,28] Considered the less robust of the two, the von Langenbeck technique is most often applied for relatively narrow hard palate defects. In contrast, the overlapping-flap technique is generally preferred for repair of wider hard palate defects because it is associated with less tension on the suture line. Additionally, the suture line is not located directly over the defect and the area of opposing connective tissue is larger, which results in a stronger repair.

von langenbeck technique

The von Langenbeck technique involves the creation of bilateral releasing incisions approximately 2 mm palatal and parallel to the maxillary teeth. The epithelial margins of the defect are tangentially excised. The mucoperiosteum is undermined bilaterally on both sides of the defect, carefully avoiding transection of the major palatine arteries as they exit the major palatine foramina. The flaps are repositioned medially and sutured over the defect using 4-0 or 5-0 absorbable monofilament suture material in a simple-interrupted pattern (Fig. 40.7A–C). A variation of the von Langenbeck technique is the two-flap palatoplasty (also known as the bipedicle flap technique), in which additional mucoperiosteal incisions are made at the rostral end of the defect (see Chapter 39). This modified technique is useful when tension at the rostral area of the defect is expected or encountered when applying the standard von Langenbeck technique.

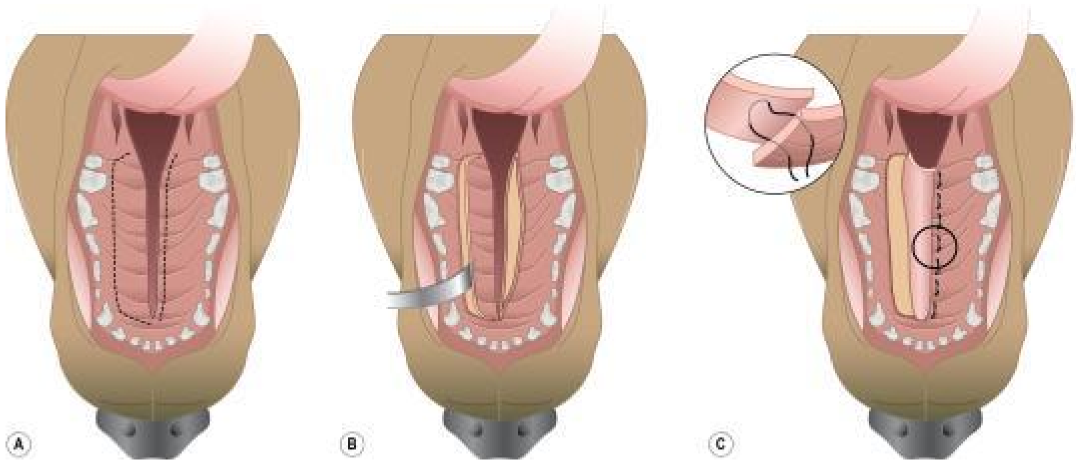


• FIG. 40.7 The von Langenbeck technique: a medially repositioned double flap technique is demonstrated for closure of a hard palate defect utilizing a midline appositional closure.

Before closure of the hard palate mucosa, and when technically feasible based on surgical access and tissue availability, the nasal mucosa can be released from the nasal aspect of the maxilla and/or the septum and closed in a simple-interrupted or continuous pattern.[24] The latter allows closure of the midline defect using two tissue layers, which is considered a more robust repair compared to a single-layer closure.

Overlapping-flap technique

The overlapping-flap technique is initiated by making an incision the length of the palatal defect, 2–3 mm palatal to the maxillary teeth, on one side of the patient's defect. Perpendicular incisions are made at the rostral and caudal ends of this incision, extending to the defect (Fig. 40.8A). The caudal incision should be planned based on advanced imaging so that it is made over the hard palate and not through the soft palate to prevent creation of an oronasal fistula. A blunt-tipped periosteal elevator is used to elevate the mucoperiosteal layer, carefully avoiding the major palatine artery as it exits the major palatine foramen, approximately halfway between the midline and the maxillary fourth premolar tooth. Preserving the minor palatine arteries is ideal but not critical. The flap must be released until it can be reversed, completely freely hinging at the edge of the defect. When elevating this flap, care must be taken not to penetrate the medial edge of the defect where the oral mucosa is confluent with the nasal mucosa.



• **FIG. 40.8** (**A**) The incision sites for utilization of the overlapping-flap technique for repair of hard palate defects are depicted. (**B**) A periosteal elevator is used to elevate the flaps. (**C**) The large flap is reflected 180 degrees and laid beneath the mucosa on the opposite side of the flap and sutured in place with multiple simple-interrupted or horizontal mattress sutures.

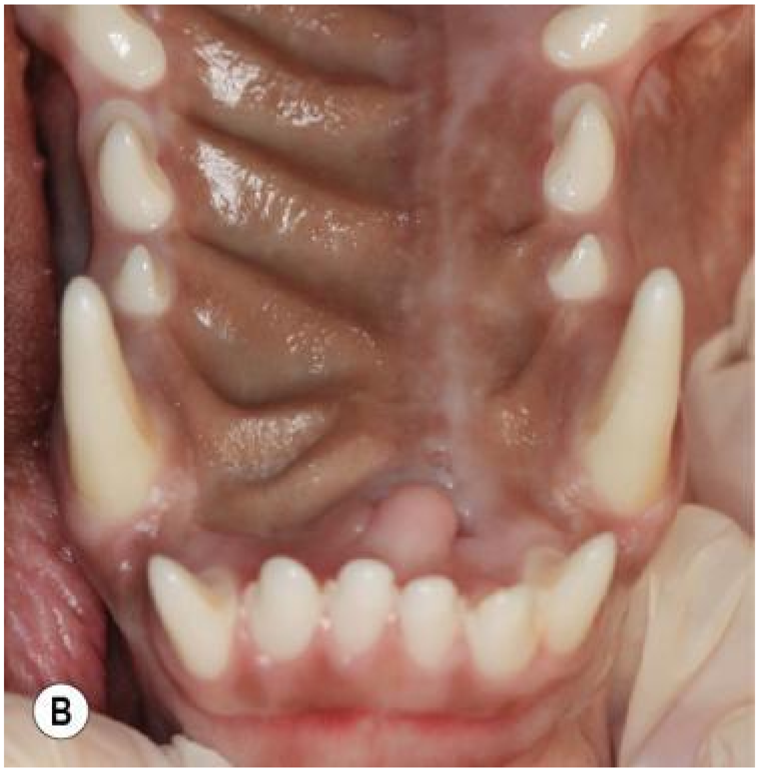
A second incision is made in the mucoperiosteum on the opposing side of the defect along its entire length, perpendicular to the margin, thereby separating the nasal from the oral mucoperiosteum. The oral mucoperiosteum is elevated approximately 8–10 mm away from the defect margin along its entire length (Fig. 40.8B).

The mucoperiosteal flap from the initial side of the defect is hinged or folded over the defect and positioned between the hard palate and the mucoperiosteal flap on the opposing side of the cleft (Fig. 40.8C). The hinged flap should cover the defect and overlap beneath the opposite mucoperiosteal flap approximately 6 mm without tension. In relatively wide defects, a secondary releasing incision 2–3 mm palatal to the dental quadrant may be required at the flap that does not hinge, to permit adequate overlap without tension. The hinged flap is sutured in place using multiple single-interrupted horizontal or vertical mattress sutures using 4-0 or 5-0 monofilament absorbable suture material. These sutures should be preplaced and tagged temporarily with hemostats. Following placement of all sutures, the sutures are tied from caudal to rostral (Figs. 40.8C and 40.9A–B). The defect created by the raised hinged flap is allowed to heal by second intention and usually takes 3–4 weeks to completely granulate and reepithelialize (Fig. 40.10A–B).


A

• **FIG. 40.9** (**A**) Preoperative view of the hard palate defect in an 8-month-old dog 6 weeks after strategic dental extractions were performed to increase the amount of tissues available for defect repair. (**B**) Postoperative view showing a hard palate defect repaired using the overlapping-flap technique; note the intact major palatine artery emerging from its corresponding foramen.


A



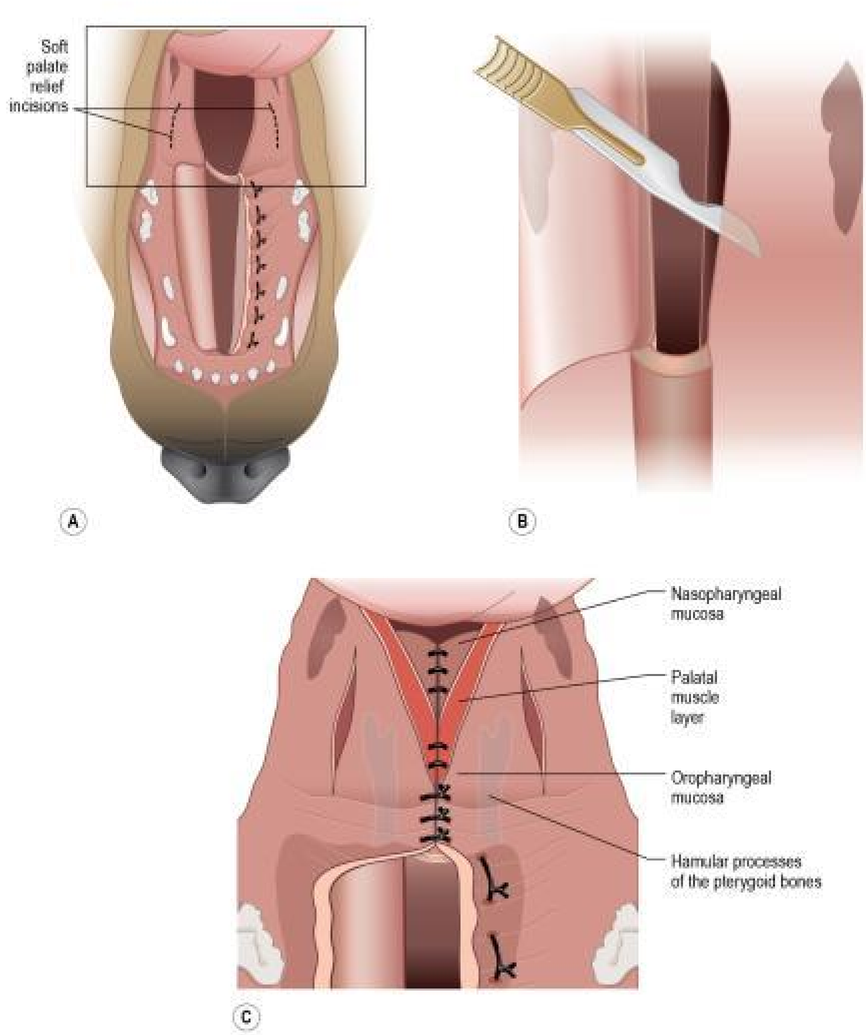
• **FIG. 40.10** (**A**) Appearance 4 weeks following surgical repair of a palatal defect using an overlapping-flap technique in a dog; note the granulation covering the area of previously denuded bone. (**B**) Appearance 12 weeks following surgical repair of the palatal defect; note the completely reepithelialized area of previously denuded bone.

Soft palate defect repair

Midline soft palate defects typically accompany hard palate defects (i.e., cleft palate and cleft lip and palate).[16] Isolated unilateral or bilateral congenital defects of the soft palate (i.e., hypoplasia of the soft palate) occur less commonly and are located lateral to the palatine muscle.[29-31] The etiopathogenetic mechanisms of hypoplasia of the soft palate in dogs has not been investigated, and it likely represents a separate entity compared to cleft lip, cleft palate, and cleft lip and palate.

Double- and triple-layer appositional technique

Relatively narrow midline defects of the soft palate can be corrected utilizing a double- or triple-layer appositional technique as long as no tension is created during closure. These techniques have been previously described.[27,32] The first step is to make an incision along the medial margin of the soft palate defect on each side to the level of the middle or caudal aspect of the palatine tonsils (Fig. 40.11B). Blunt-ended tenotomy scissors (Hu-Friedy Instrument Co., Chicago, IL) or small Metzenbaum scissors are used to bluntly separate the nasal and oral mucosae of the soft palate. Closure of the nasal mucosa should be directed from caudal to rostral utilizing 4-0 or 5-0 monofilament absorbable suture material in a simple-interrupted pattern so that the knots are located in the nasopharynx (Fig. 40.11C). If elected for triple-layer closure, the muscular layer is closed using a simple-continuous pattern with 5-0 or 6-0 monofilament absorbable suture material. Finally, the oral mucosa is apposed using a simple-interrupted pattern with 4-0 or 5-0 monofilament absorbable suture material, with the knots located in the oral cavity. Verification of the proper length of the repaired soft palate is confirmed following temporary extubation of the patient. Ideally, the caudal edge of the repaired soft palate should be located somewhere between the level of the middle to the caudal aspect of the tonsils and just touch the tip of the epiglottis. If tension is present along the suture line, partial-thickness tension-releasing incisions can be made unilaterally or bilaterally, as needed (Fig. 40.11A). These partial-thickness relief incisions, when required, are made in the oral mucosa from the lingual aspect of the last molar to near the level of the tip of the soft palate.

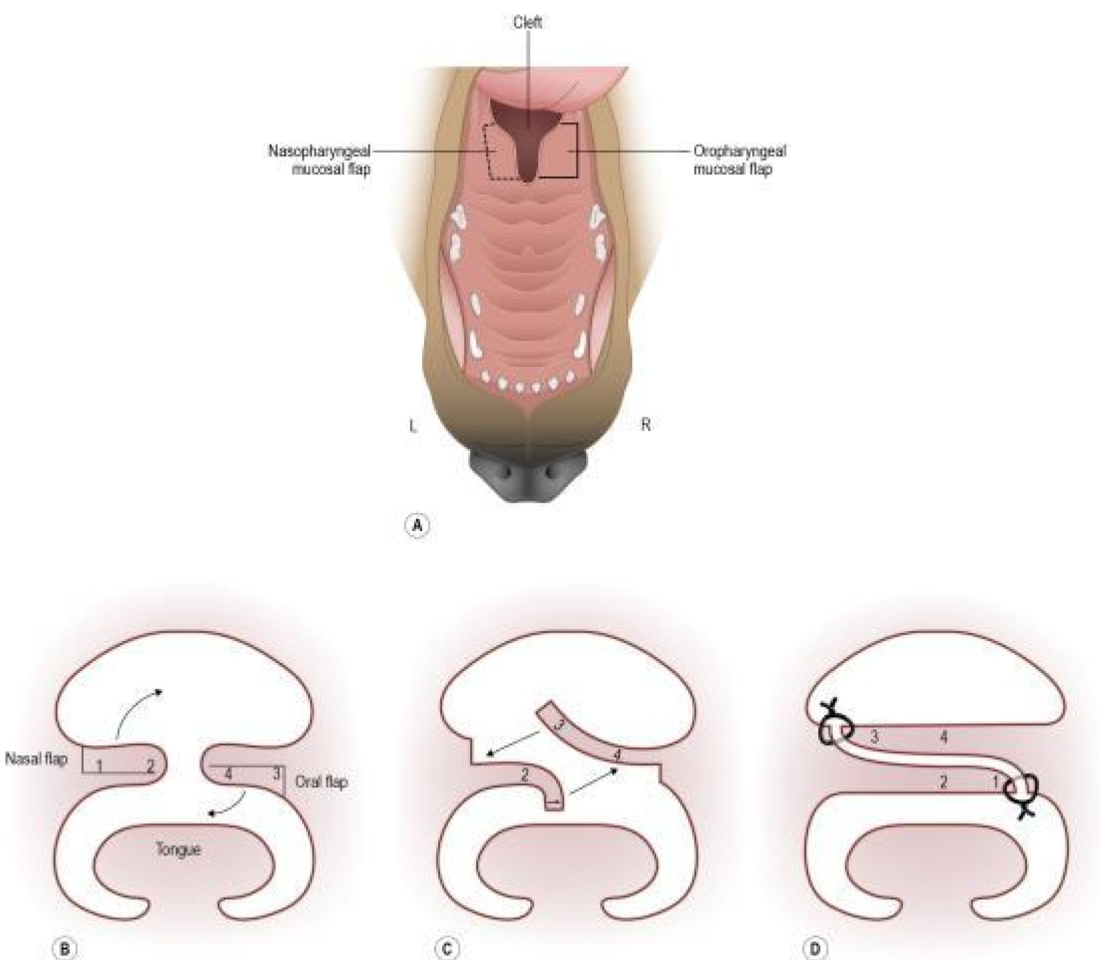


• **FIG. 40.11** (**A**) Closure of midline soft palate defects should begin with partial-thickness relief incisions if needed to prevent tension on the incision line. (**B**) Incisions are made along the edges of the soft palate defect to separate the oral and nasal mucosal layers. (**C**) The palatal defect is closed in two layers, or in three layers if the muscularis layer is sutured separately.

Bilateral overlapping single-pedicle flap technique

An alternative technique has been described for the repair of midline soft palate defects called the bilateral overlapping mucosal single-pedicle flap technique.[33] Surgical correction using this technique is technically more challenging and may not be necessary in most cases. The reported benefits of the bilateral overlapping single-pedicle flap technique over the simple apposition techniques for correction of soft palate defects is that in the overlapping technique, each layer is supported by an underlying layer of intact mucosa, as the suture lines are offset and by placing the suture lines laterally. This technique also minimizes the effect of the pull of the palatine muscles on the incision line.

Repair of soft palate defects utilizing the bilateral overlapping single-pedicle flaps is performed by creating a nasal mucosal flap on one side of the defect and an oral mucosal flap on the other side of the defect, undermining the flaps to develop bilateral single-pedicle flaps with their bases on the edge of the cleft. The nasal mucosal flap is reflected into the oral cavity and the oral mucosal flap is rotated into the nasal cavity. The flaps are sutured in place using a simple-interrupted pattern with 5-0 or 6-0 absorbable monofilament suture material (Fig. 40.12A–C).



• **FIG. 40.12** Bilateral overlapping mucosal single-pedicle flaps for correction of soft palate defects are harvested from the nasal and oral mucosal (**A, B**), which are rotated 180 degrees (**C**) and sutured in position (**D**) to close the defect.

Repair of unilateral hypoplasia of the soft palate

Unilateral defects of the soft palate seem to occur infrequently, although several reports have described its surgical repair.[31,33,34] Dogs with unilateral clefts of the soft palate are presented primarily because of rhinitis.

A relatively simple two-layer technique can be utilized in the closure of unilateral defects of the soft palate where sufficient soft palate tissue exists. This technique is similar to the two-layer closure of midline soft palate defects except for the location of the defect.[31] The patient is placed in dorsal recumbency, the upper jaw is secured to the surgical table, and the lower jaw and endotracheal tube are suspended from a stand. Following surgical preparation of the soft palate, three stay sutures are placed around the defect, one at the rostral border and one at the caudal end of each side of the soft palate defect. Tension is placed on the edges of the defect with the previously placed stay sutures and a small incision is made in the edge of the cleft using a #11 scalpel blade. The margins of the defect are separated into dorsal and ventral components using a blunt-ended tenotomy scissors (Hu-Friedy Instrument Co., Chicago, IL). The defect is closed from caudal to rostral in two layers utilizing a simple continuous pattern using fine absorbable suture material in the nasal mucosa. Suturing of the defect should be accomplished with relatively large bites, and the sutures should be snug but should not strangulate the tissue.[31] The oral mucosa is apposed in a similar manner. The knots for each layer should be placed on the epithelial surface to prevent burying excessive suture material.

Large unilateral defects that are difficult to appose without tension may require a more complex reconstruction technique such as the use of buccal mucosal flaps that have also been recommended for the surgical correction of hypoplastic soft palates.[30]

Bilateral hypoplasia of the soft palate

Animals with hypoplastic soft palate with bilateral defects have significantly shortened soft palates. Oropharyngeal examination in these patients typically reveals a near absence of the soft palate with a small uvula-like projection (i.e., pseudouvula) that extends from the midcaudal aspect of the hard palate (Fig. 40.13A–B).

• **FIG. 40.13** (**A**) Endoscopic view of a normal soft palate in a 6-week-old Shetland sheepdog. (**B**) Endoscopic view of a severely hypoplastic soft palate in a littermate, presented because of bilateral mucopurulent nasal discharge; note the uvula-like projection extending caudally in the middle of the hypoplastic soft palate.

Various recommendations have been made concerning the most appropriate treatment for hypoplasia or congenital absence of the soft palate, ranging from surgical correction to euthanasia.[30,35,36] Normal compensatory mechanisms and proper dietary management, combined with surgery if necessary, may permit these patients to regain some function and in some cases lead a relatively normal life.[36]

Various surgical techniques have been utilized in the treatment of congenital absence of the soft palate. One technique involves the bilateral removal of the tonsils with creation of dorsal and ventral pharyngeal flaps bilaterally, incision of the edges of the uvula-like structure, and suturing of the pharyngeal flaps to the uvula-like structure using a two-layer, simple-interrupted pattern with fine absorbable monofilament suture material.[36]

Buccal mucosal flaps for the correction of hypoplastic soft palate bilateral defects have been recommended.[30] This technique involves the creation of bilateral buccal mucosal flaps based at the palatoglossal fold, at the caudal end of the hard palate. The buccal flaps are carefully undermined to prevent damage to the deep facial vein. The free edge of the soft palate remnant and the mucosa of the pharyngeal walls are incised to create both dorsal (nasal) and ventral (oral) mucosal free edges. The first buccal flap is rotated so that the mucosal surface of the flap faces dorsally to become the floor of the nasopharynx and is sutured rostrally to the nasal mucosal free edge of the soft palate and laterally to the dorsal free mucosal edge of the pharyngeal wall. The second buccal flap on the opposite side is elevated as previously described. This flap is rotated across the defect so that the mucosal surface faces the oral cavity to become the mucosal surface of the oropharynx. The flap is sutured to the oral mucosal free edge of the pharyngeal wall of the pharynx and to the first flap laterally and to the soft palate remnant rostrally along the oral mucosal free edge. The caudal aspects of the two flaps are sutured together. A simple-interrupted pattern using fine absorbable suture material on a cutting needle has been recommended for this procedure.[30]

An alternative free-flap technique for repair of a hypoplastic soft palate using the M. latissimus dorsi and accompanying thoracodorsal artery and vein with microvascular anastomosis has been described.[37]

Postoperative care and assessment

The routine administration of postoperative systemic antibiotics after orofacial cleft repair is controversial and likely unnecessary.[10] Administration of broad-spectrum antibiotics may be indicated postoperatively in cases of severe rhinitis and/or subclinical aspiration pneumonia.

A minimum of one night of hospitalization is recommended postoperatively so that the airway can be monitored, possible complications addressed, and pain appropriately managed. The analgesics used should be determined based on individual needs and systemic status of each patient. Many patients benefit from continuous infusion of single or combined agents. Because the nasal cavity of animals can be filled with blood clots and fluids for several hours after surgery, patients are often uncomfortable and may become dysphoric, especially immediately after anesthetic recovery. Because of this, some patients require sedation.

In most cases, oral intake can begin immediately following complete recovery from anesthesia. Oral intake should begin with a liquid or blended diet for approximately 2 weeks, followed by the slow conversion to a soft diet for an additional 4 weeks. Chew toys and other hard objects should be withheld for 6 weeks. If used, skin sutures should be removed from cleft lip repair sites 10–14 days postoperatively. Surgical sites should be reevaluated at 2 and 6 weeks postoperatively. Sedation or general anesthesia may be required to thoroughly assess healing of the surgical sites, especially in cases in which the soft palate was involved.

The placement of temporary feeding tubes has been recommended in some cases to divert food away from the oral cavity to reduce stress on the surgical site.[38] Placement of a feeding tube should be considered if tension exists on the suture line and tube feeding should be continued for at least 1 week. However, in most cases in which the hard and soft palate defects have been closed with minimal tension, and in all cases of isolated cleft lip repair, alternative methods to routine oral intake are not required.[26]

Complications

The most common complication associated with orofacial cleft repair is oronasal fistula formation due to dehiscence of surgical sites (Fig. 40.14).[24] Dehiscence can be minimized by performing tension-free closures, by gentle intraoperative tissue handling, by preserving adequate blood supply, and, in general, by abiding by sound surgical principles and technique. Although it can occur anywhere along suture lines, the two most common locations for dehiscence and oronasal fistula formation are the rostral area of the hard palate and the junction between the hard and soft palate.[24] Dehiscence of soft palate repair is also possible but occurs less frequently. Revision surgery is indicated in cases of oronasal formation regardless of defect size, especially if the patient's functional status has not been completely restored.

• **FIG. 40.14** Appearance of a hard palate defect 12 weeks following repair using the overlapping-flap technique; note the large area of dehiscence located along the hinging site of the flap.

Repair of oronasal fistulae should be delayed for approximately 6 weeks to allow tissue to heal and epithelialize prior to further surgical manipulation.[3,24]

Prognosis

The reported success rate after congenital hard and soft palate defect repair in dogs is approximately 85%.[24] However, up to 50% of cases require at least one revision surgery due to oronasal fistula formation. Based on a previous study,[24] animals that weigh less than 1 kg at the time of surgery have a poorer outcome compared to dogs weighing more than 1 kg at the time of surgery. This is most likely due to limited surgical access and difficulty manipulating small and delicate tissues. Additionally, patients older than 8 months at the time of surgery and/or with a history of previous failed surgical repair attempts might be at a higher risk of oronasal fistula formation.

The success rate of cleft palate repair in cats or of cleft lip repair in dogs and cats has not been reported but is likely high if surgical principles are followed and adequate technique is used. Animals with soft palate defects will often have persistent clinical signs due to chronic rhinitis, even following surgical intervention. Even if a shelf of soft tissue can be constructed to separate the nasopharynx from the oropharynx, function may not be fully restored. This is because the soft palate functions in synchrony with other muscles of the tongue, pharynx, and larynx during deglutition, which may not regain function after surgery.[37] The mere presence of tissue may not prevent oronasal reflux. However, dietary management, such as choosing foods that remain in a bolus and offering water from an elevated surface or water bottle, may play an important role in the successful management of these cases.[36]

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CHAPTER 41

Acquired palatal defects

Santiago Peralta, Sandra Manfra Marretta

Definitions

Advancement flap: A mucoperiosteal pedicle flap that is advanced along its long axis.

Angularis oris flap: Vestibular mucosal flap harvested immediately caudal to the commissure of the lip, which includes the angularis oris artery.

Double-layer flap: Two separate flaps that are utilized in the closure of a defect in two overlapping layers.

Oronasal fistula: Acquired chronic communication between the oral and nasal cavities, lined by an epithelium.

Split palatal U-flap: Bilateral transposition flaps in which each pedicle is based on the location of the major palatine artery.

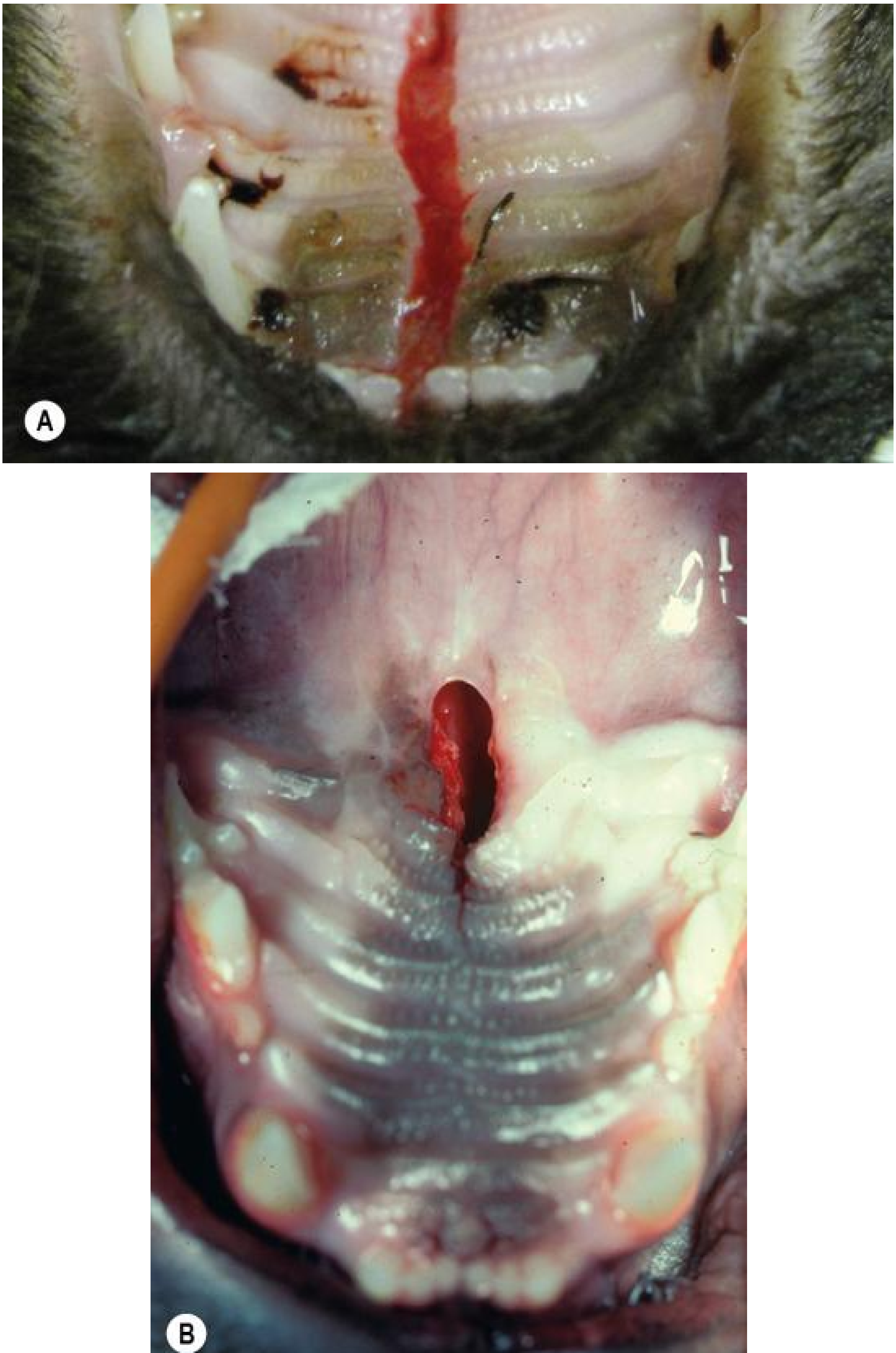
Transposition flap: A mucoperiosteal pedicle flap that is rotated on its basis to cover a defect.

Vestibular mucosal flap: A pedicle mucosal flap with associated underlying connective tissue harvested from the alveolar mucosa and the buccal mucosa of the lip or cheek.

Preoperative concerns

Acquired palatal defects are usually secondary to traumatic injuries (i.e., bite wounds, gunshot wounds, and foreign body penetrations), advanced periodontitis, dehiscence of surgical sites (i.e., after orofacial cleft repair, dental extractions, maxillectomy, etc.), electrical burns, and traumatizing malocclusion (Figs. 41.1 and 41.2).[1,2] Acquired palatal defects can be acute or chronic. Acute defects lack an epithelial lining and in some cases may spontaneously heal. However, epithelialization of the edges frequently occurs and a chronic palatal defect is established. Chronic palatal defects (i.e., lined by an epithelium) are termed oronasal fistulae. The treatment of acquired palatal defects is surgical and usually consists of the mobilization of adjacent soft tissues to cover the defect and isolate the nasal from the oral cavity.

• **FIG. 41.1** Oronasal fistulae in dogs. (**A**) Oronasal fistula following the forceful extraction of the right maxillary canine tooth. (**B**) Palatal defect caused by the prolonged retention of a foreign body wedged between the maxillary fourth premolar teeth, which resulted in pressure necrosis of the underlying palate. (**C**) Bilateral oronasal fistulae and tooth loss due to severe periodontitis of the maxillary canine teeth.



• **FIG. 41.2** Palatal fractures and oronasal fistulae in cats. (**A**) Acute traumatic defect of the hard palate. (**B**) Oronasal fistula resulting from a similar lesion that was left to heal by second intention but failed to do so.

Because acquired palatal defects can cause chronic nasal disease and predispose to aspiration pneumonia, the overall clinical status of the patient should be thoroughly evaluated and appropriately treated prior to surgery. Diagnostic imaging is necessary to assess the nature, location, and extent of the defect(s). Intraoral radiography is essential in cases of acquired palatal defects secondary to severe periodontitis. Computed tomography is indicated to document the extent of the bony defect and in other cases of acquired palatal defects, especially in cases of traumatic origin or when the cause cannot be ascertained based on historical and/or physical examination findings. An incisional biopsy and additional diagnostic tests may be indicated prior to repair if an underlying neoplastic process is suspected.

When the viability of the tissue surrounding the acquired palatal defect cannot be predicted, delay in closure of the defect may be warranted. Alternate forms of alimentation, including esophagostomy and gastrostomy tubes (see Chapter 5), may occasionally be indicated in animals in which surgical repair of the defect must be delayed.

Although significant and uncontrolled blood loss does not usually occur during acquired palatal defect repair, coagulation and platelet function tests can be performed prior to surgery at the discretion of the clinician, especially in animals with a known history of coagulopathies, thrombocytopenia, or thrombocytopathy. If indicated, blood typing and cross-matching should be performed before surgery.

Anesthetic considerations

For acquired palatal defect repair, patients should be anesthetized with an anesthetic protocol based on the historical and physical examination findings, as well as results of any preoperative diagnostic tests of each individual. In cases of acute palatal defects of traumatic origin, a thorough neurologic, ophthalmologic, and respiratory evaluation is warranted to establish the surgical and medical priorities prior to intervening. Repair of palatal defects should be delayed as needed in systemically or neurologically unstable patients. A guarded endotracheal tube is recommended for intubation to prevent inadvertent occlusion during the surgical procedure. Patients should be positioned in either dorsal or lateral recumbency, based on the location of the defect and surgeon's preference, with the endotracheal tube firmly secured to the mandible.

Regional nerve blocks are indicated to achieve a balanced anesthesia and as part of a multimodal analgesic approach. The technique, agent, and dose used depend on the size of the patient, location of the defect(s), and surgeon's preference (see Chapter 4).

The use of mouth gags should be avoided or minimized during surgery, especially in cats. The use of mouth gags may affect blood supply to the brain and has been associated with transient or permanent postoperative neurologic deficits, including central blindness, deafness, and ataxia.[3-5]

Surgical anatomy[6]

The incisive bones, the palatine processes of the maxillary bones, the maxillary process of the palatine bones, and the corresponding oral mucosal tissues form the roof of the oral cavity. In general, osseous structures articulate with each other via sutures, which are composed of Sharpey's fibers during development and eventually fuse during adulthood. There are three midline sutures along the hard palate, which articulate the left and right palatine processes of the incisive, maxillary, and palatine bones. Additionally, the palatomaxillary sutures are found between the palatine and maxillary bones bilaterally, and the incisivomaxillary sutures, between the maxillary and incisive bones bilaterally. The palatine fissures, two large openings between the incisive bones and the palatine processes of the maxillae, are located medial to the third incisors and canine teeth and can be palpated as soft areas in the rostral aspect of the hard palate. The caudal aspect of the hard palate is located just caudal to the maxillary second molar teeth, where it merges with the rostral aspect of the soft palate.

The blood supplies to the hard palate of the dog and cat are similar. The right and left major palatine arteries are the main arteries to the mucoperiosteum of the hard palate. The major palatine artery emerges from the major palatine canal at the major palatine foramen and courses rostrally in the shallow palatine sulcus on the surface of the hard palate (Fig. 40.1). The major palatine foramina are usually located medial to the maxillary fourth premolar teeth, although the exact location may vary slightly depending on species (i.e., cat or dog) and skull type and conformation.

Special instruments and materials

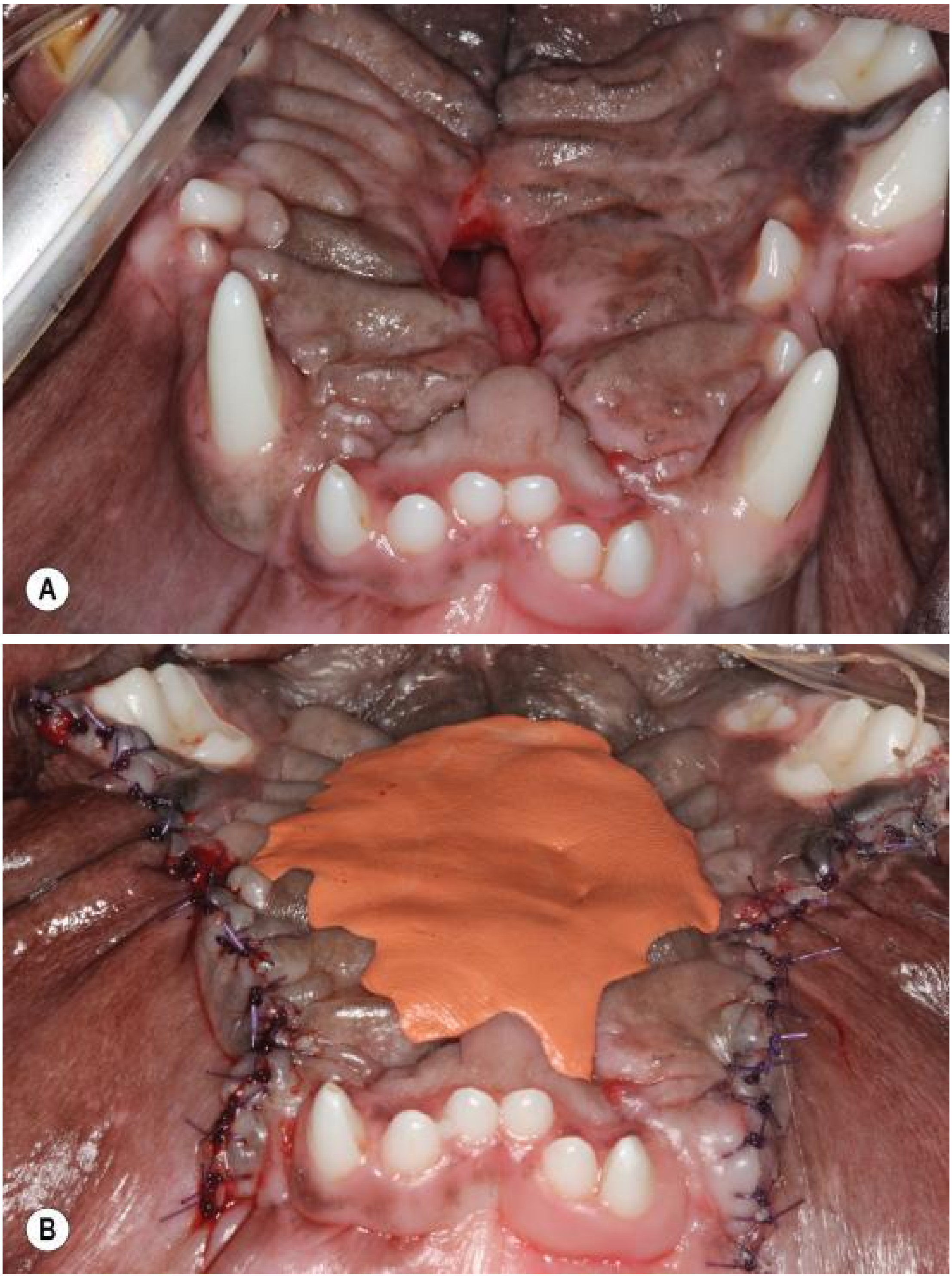
A Bard-Parker no. 3 long scalpel handle with a # 15 scalpel blade can be used to incise the mucosa. When indicated, a P24G periosteal elevator (Hu-Friedy Mfg. Co., Chicago, IL) can be used for elevating the hard palate mucosa, the attached gingiva, or the alveolar mucosa from the underlying bone during flap procedures in small patients. Small round Metzenbaum scissors can be used for blunt dissection of the vestibular mucosal flaps into two layers.

Excellent lighting and magnification (i.e., surgical loupes or operating microscope) are essential during oronasal fistula repair. As in the case of orofacial clefts (see Chapter 40), the routine use of membranes and/or grafting materials is not recommended for oronasal fistula repair in small animals at this time.

Therapeutic decision-making

Prior to surgical repair of acquired palatal defects, it is important to carefully decide what surgical plan is likely to be most successful in a particular type of defect. Therapeutic decision-making in these cases is usually based on the cause, size, and location of the defect(s) and whether or not there has been any prior surgical intervention. Prior unsuccessful surgical tissue manipulation can result in poor tissue quality due to fibrosis and reduced blood supply. Patients with acute defects of traumatic origin often have other clinically significant maxillofacial and/or dentoalveolar injuries that may require simultaneous or delayed repair depending on their nature and severity.[7-9]

Various single-layer surgical techniques can be used to repair acquired palatal defects, including vestibular flaps, transposition flaps, split palatal U-flaps, and island palatal flaps.[1,10,11] Double-layer techniques, including vestibular and reflected palatal flaps, can also be utilized.[12,13] In general, a technique that provides the largest flap with no tension and an adequate blood supply is recommended. Double-layer flaps are considered more robust and are preferred in cases of relatively large oronasal fistulae or in cases in which single-layer closure was unsuccessful. Achieving two-layer closure may require a staged approach. Namely, when the defect(s) are too large relative to tissues available for coverage, strategic dental extractions prior to definitive repair may be indicated. When indicated, strategic dental extractions are usually performed 4–6 weeks prior to definitive surgical repair of the defect(s).[13] The rationale is that by extracting teeth in the vicinity of a defect, larger areas of tissues become available for harvesting, mobilization, and coverage, thus increasing the chance of achieving a tension-free closure (Fig. 41.3). The impact of extracting teeth in dogs and cats is usually considered minimal, once adequate extraction technique is applied. Shortening the period between extractions and defect repair is not recommended because the tissues will not have completely healed. A custom-made temporary obturator made of vinyl polysiloxane (3M Express STD Putty VPS Impression Material) may be useful in some cases, particularly in those that are severely dysfunctional due to the oronasal fistula.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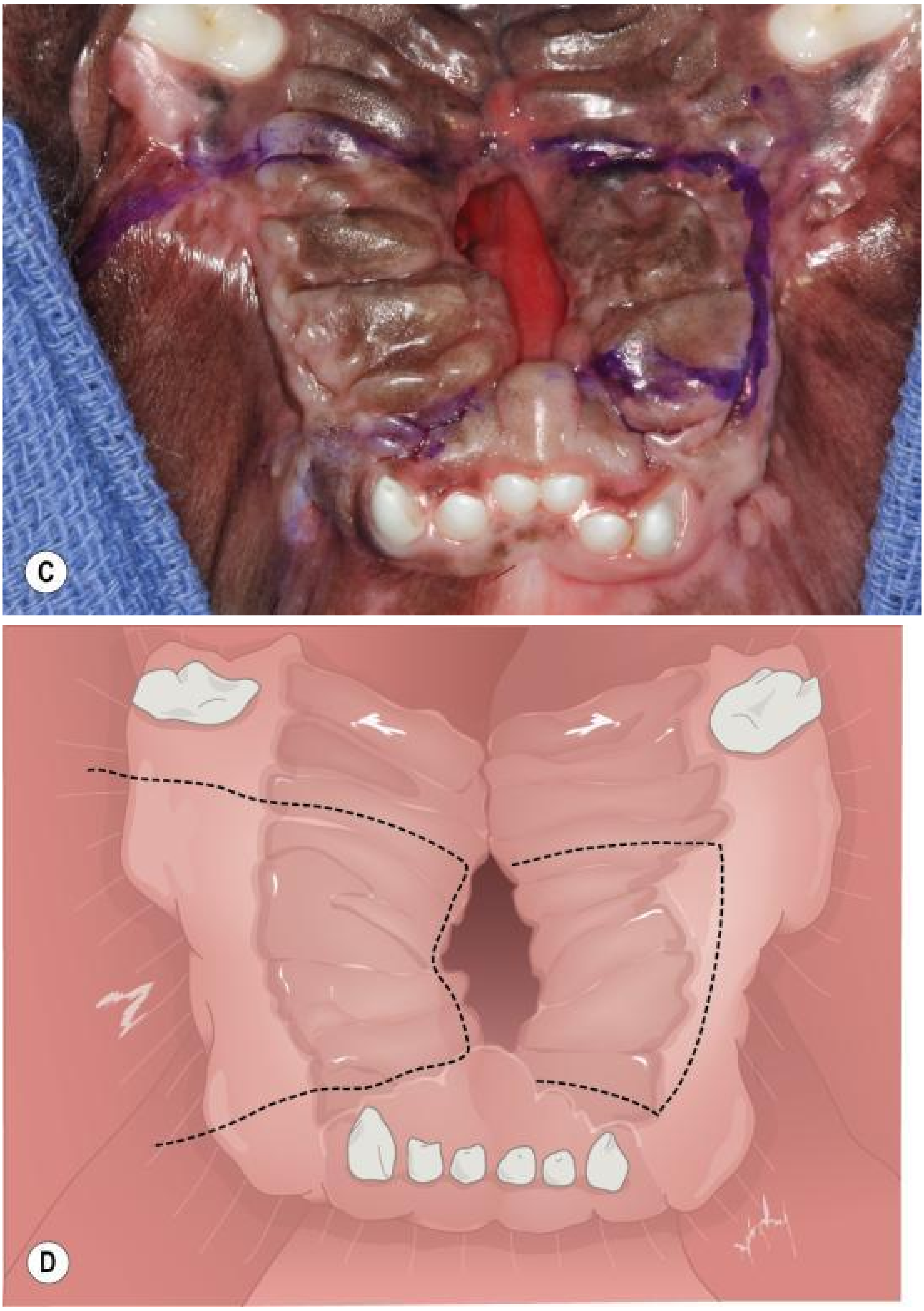
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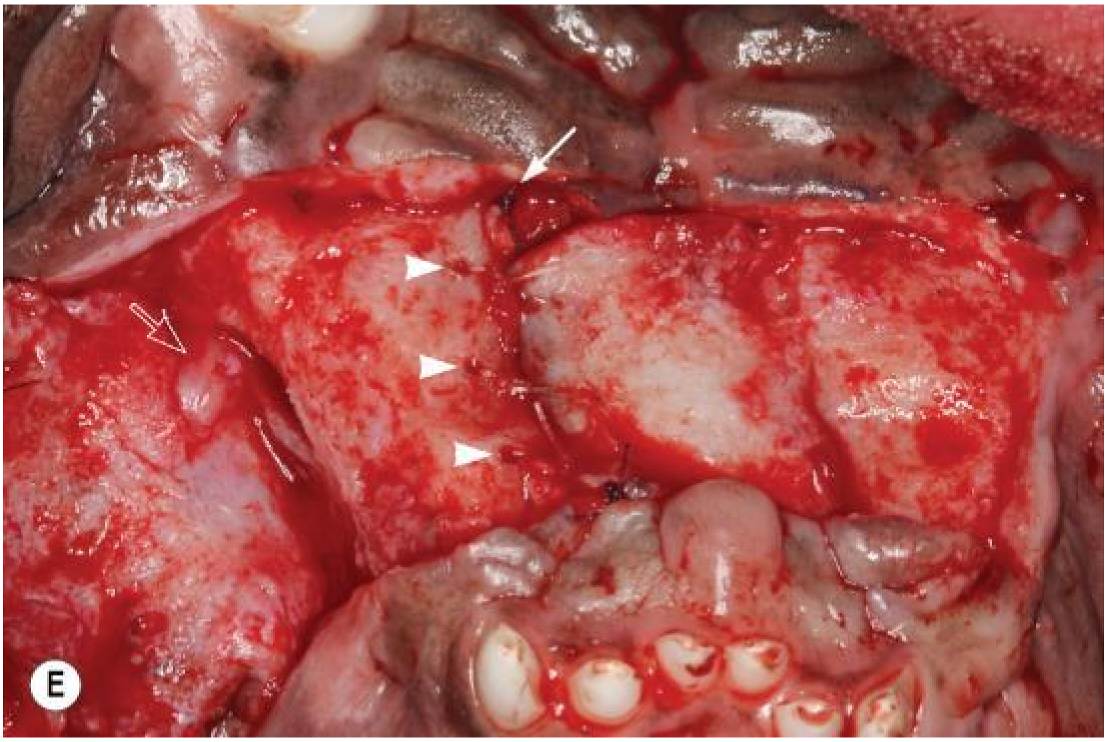
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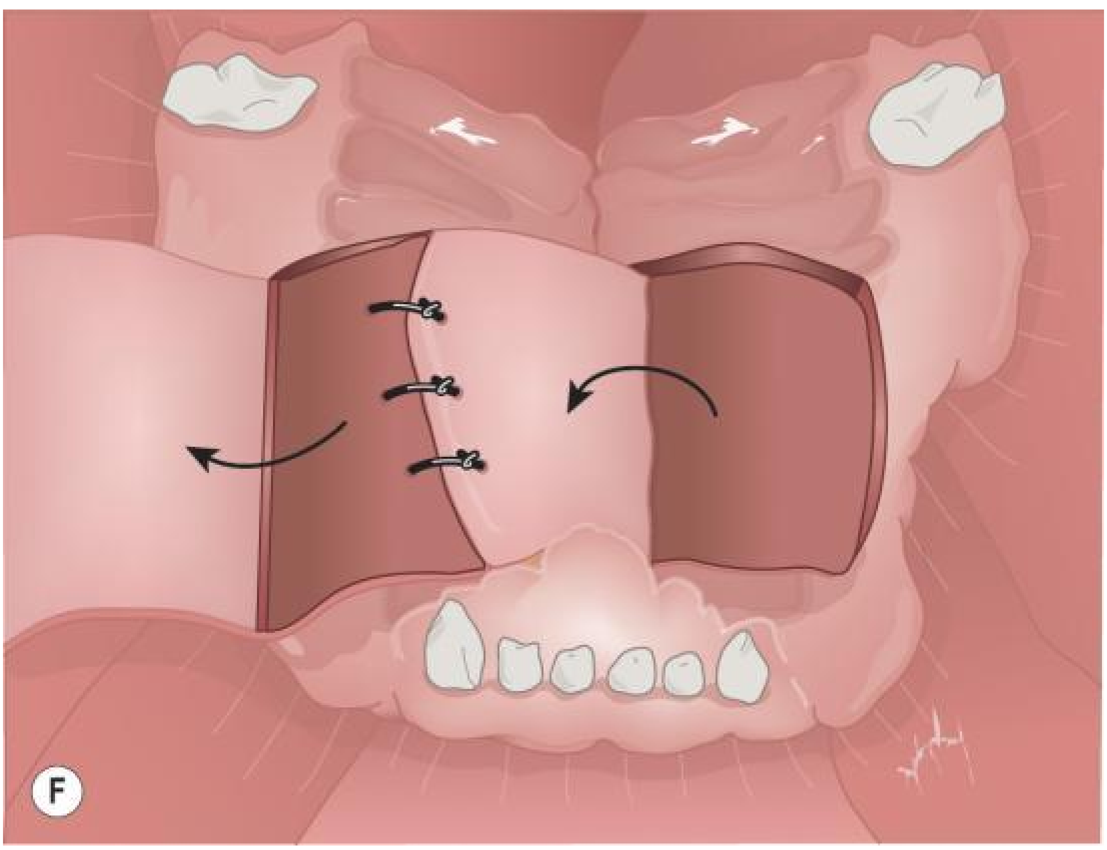
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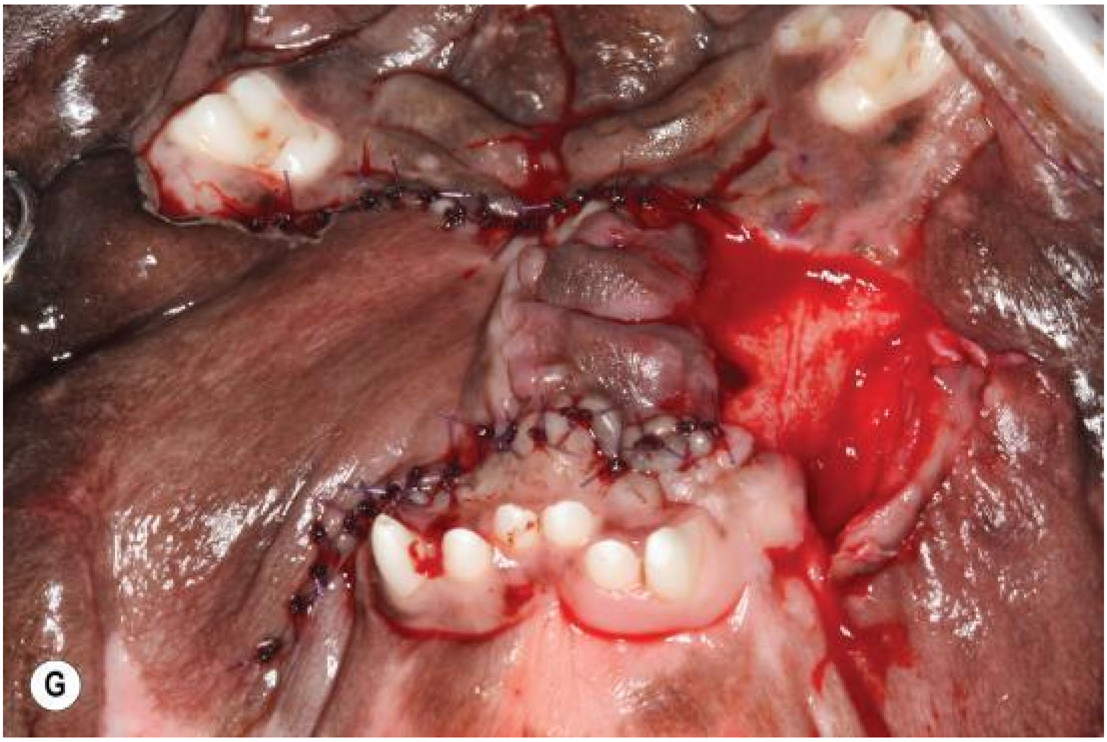
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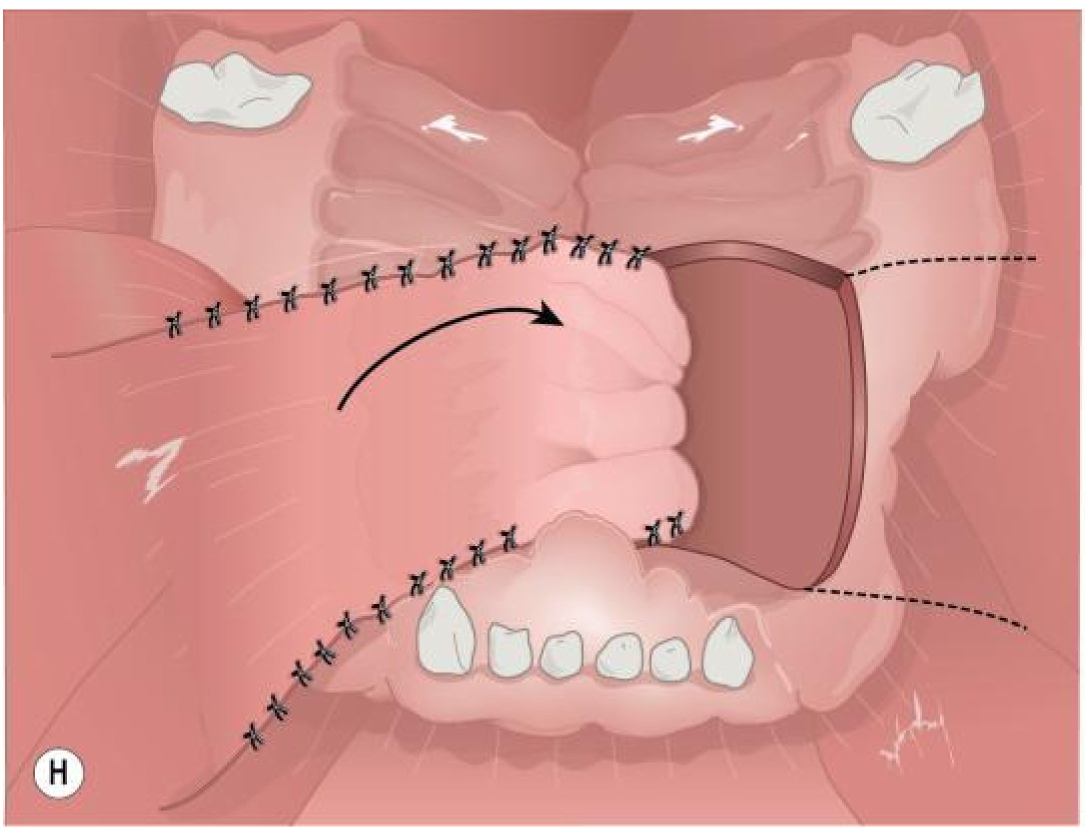
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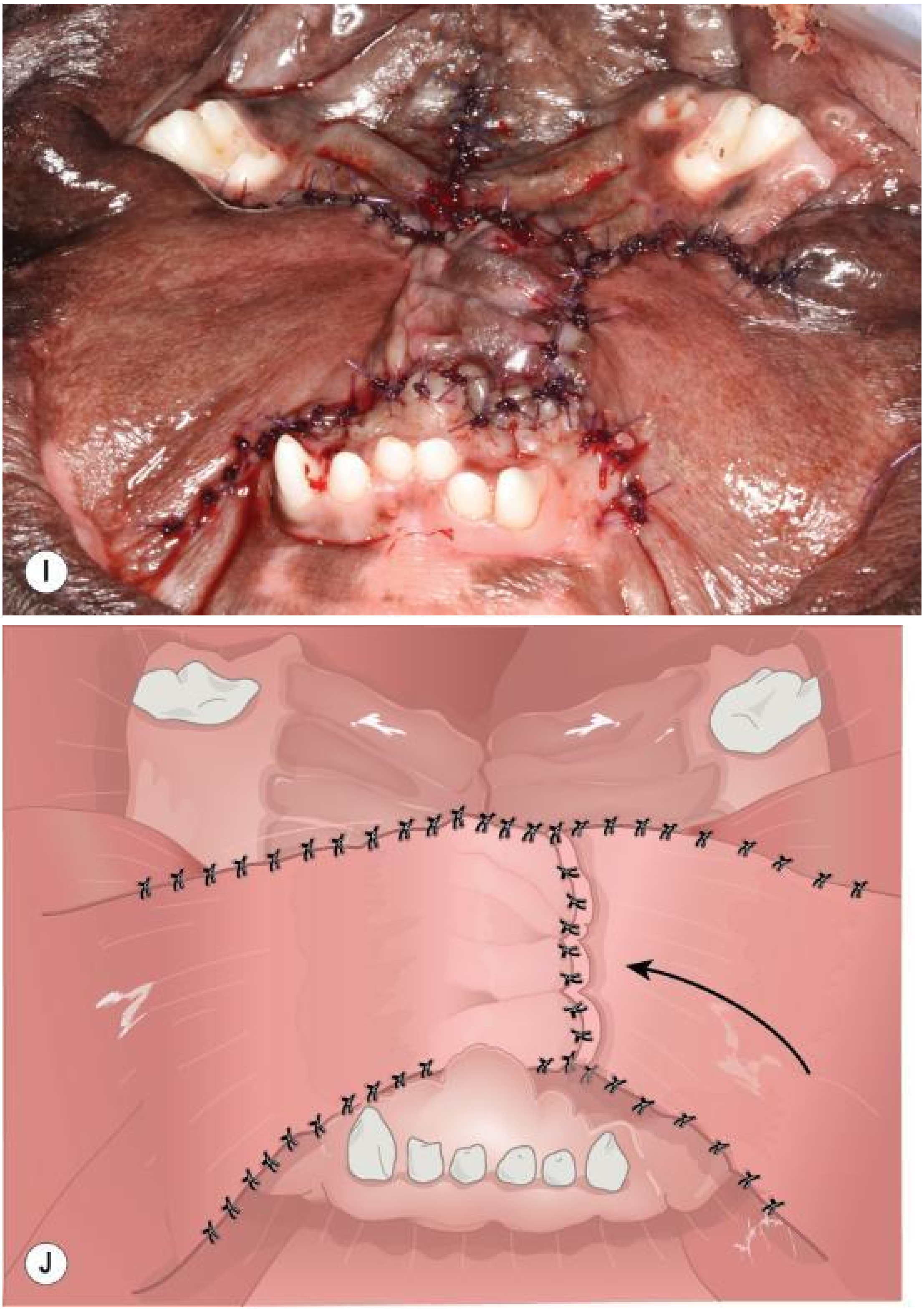
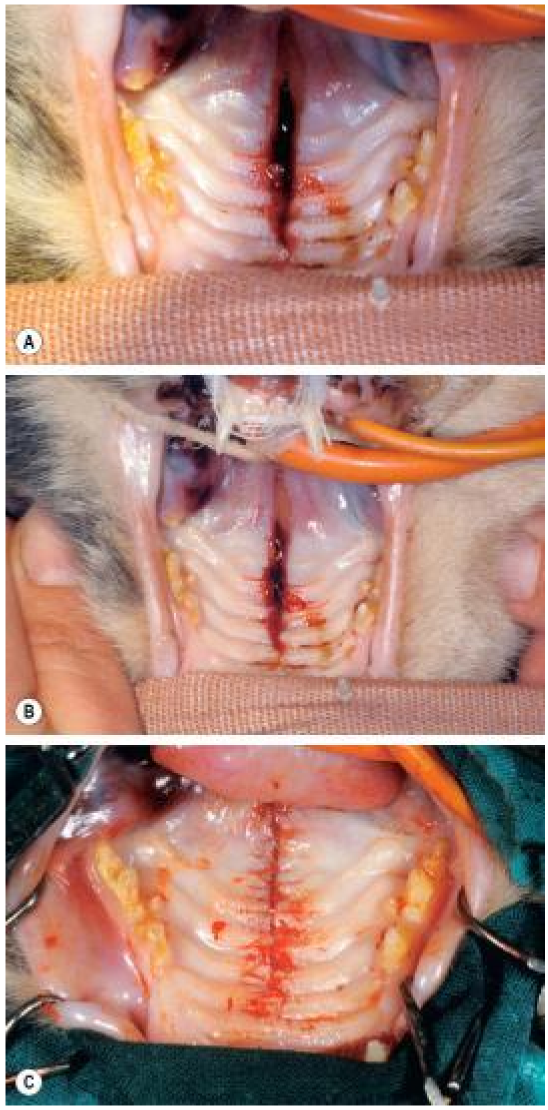


• **FIG. 41.3** Staged approach for palatal defect closure. (**A**) Midline hard palate defect in a dog. The defect is considered too large to close with local single- or double-layer mucoperiosteal flaps based on clinical and computed tomography findings. (**B**) Strategic dental extractions are performed to make additional tissues available for harvesting and mobilization. A temporary obturator can be placed to minimize clinical signs during healing of extraction sites. (**C–D**) Definitive closure is performed when the extraction sites have completely healed, usually 4–6 weeks later. In this case, a flap that hinges at the edge of the defect is planned on the right side, and a mucogingival pedicle flap is planned as a second layer on the left side. (**E–F**) The flap from the right is reversed and secured to the nasal aspect of the opposing palatal bone (white arrowheads). The holes in the bone can be made with a K-wire on a hand chuck. Note that the left major palatine artery was transected in this case (white solid arrow) but the infraorbital artery was preserved (white unfilled arrow) to ensure adequate blood supply to the flap. (**G–H**) The pedicle flap from the left is advanced to cover the submucosal aspect of the reversed flap from the right and is secured in place using monofilament absorbable suture material in a simple interrupted pattern. (**I–J**) A third vestibular mucosal flap is elevated from the right side and is advanced to cover any denuded areas of bone. As the other two flaps, this one is secured using monofilament absorbable suture material in a simple interrupted pattern. Source: (From Peralta S, Nemec A, Fiani N, et al. Staged double-layer closure of palatal defects in 6 dogs. *Vet Surg* 2015;44:423–431. With permission.)

Surgical techniques

Closure of acute midline palatal defect

Acute midline palatal fractures are common in cats that have sustained traumatic injuries, especially after a high-rise fall (Fig. 41.2A and also see Chapter 27).[9] Initially lacking epithelialized edges, some relatively narrow defects may resolve spontaneously without surgical treatment. However, epithelialization of the defect margins can occur, and an oronasal fistula becomes established (Fig. 41.2B). Therefore, provided that the patient is stable, primary closure of acute open midline palatal fractures is recommended to prevent oronasal fistula formation. Preoperative computed tomography of the head to assess the defect and other traumatic injuries is strongly recommended in these cases.[7,9] In relatively narrow acute defects, soft tissue debridement and approximation of the displaced bony structures can be performed by gentle digital pressure, followed by apposition of the palatal mucosal edges in a simple-interrupted pattern using a 5-0 absorbable monofilament suture material (Fig. 41.4).[14] Rinsing and suctioning the nasal cavity prior to closure are indicated, especially if blood clots, debris, or foreign material is present or suspected. In wider acute defects and established oronasal fistula(e) along the midline of the hard palate, lateral releasing incisions and elevation of the palatal mucosa may be necessary to mobilize the tissues toward the midline and achieve tension-free closure. The technique is similar to the von Langenbeck technique used for congenital hard palate defect repair (see Chapter 40). In cases of established oronasal fistula(e) along the midline of the hard palate, the defect edges should be tangentially incised prior to suturing in a simple-interrupted pattern.

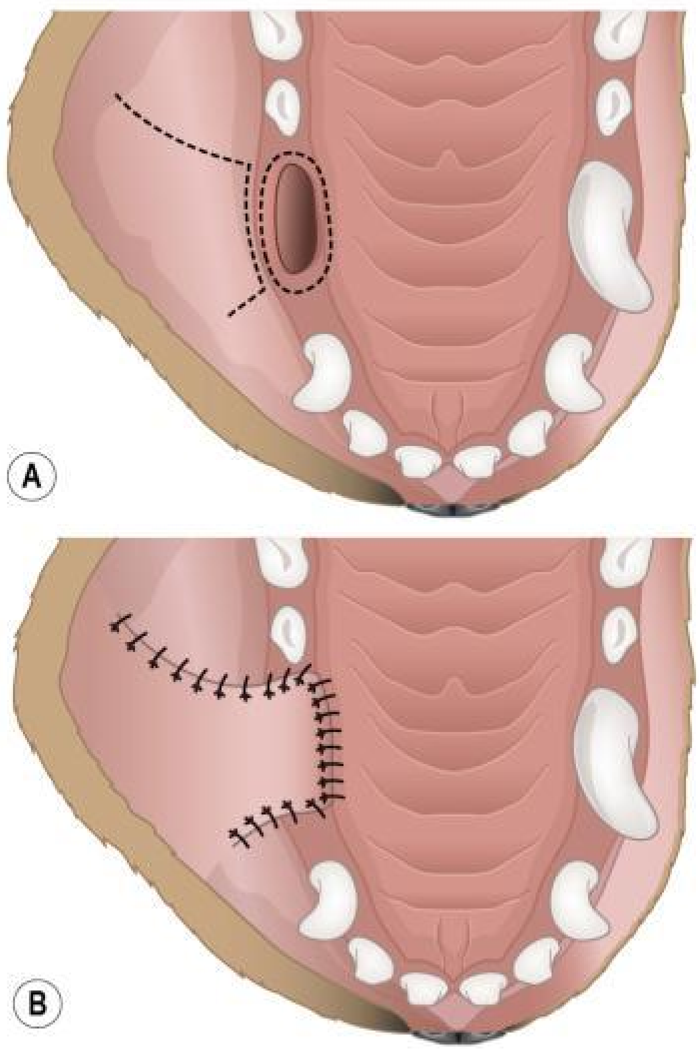


• **FIG. 41.4** Primary closure of an open midline fracture/separation of the hard palate in a cat. (**A**) Soft tissue debridement. (**B**) Approximation of the displaced bony structures by gentle digital pressure. (**C**) Primary closure of the injured palatal mucosa in a simple-interrupted pattern. Source: (**A** and **C** from Verstraete FJM. Maxillofacial fractures. In: Slatter DG (ed). *Textbook of Small Animal Surgery*. 3rd ed. Philadelphia: WB Saunders; 2003:2190–2207.)

Single-layer techniques

Vestibular flaps

Vestibular flaps can be utilized for repair of oronasal fistulae secondary to severe periodontitis and in cases of dehiscence after dental extraction or partial maxillectomy.[15] Prior to closure, the edges of the defect must be thoroughly debrided along the entire epithelial margin. This is accomplished by excising a 2–3-mm rim of tissue from the entire circumference of the defect (Fig. 41.5A). Two divergent incisions are then made in the mucosa beginning at the mesial and distal aspect of the fistula through the attached gingiva, across the mucogingival line, and extending into the alveolar mucosa. A periosteal elevator is used to gently elevate the mucoperiosteal flap from the underlying bone. The inelastic innermost layer of the flap, the periosteal layer, may be incised along the entire base of the flap. This results in a more elastic, mobile flap that can be closed without tension. If tension persists, additional periosteal releasing, extension of the mesial and distal incisions, and additional undermining may be necessary prior to closure. Reduction in the height of the alveolar process with an osteoplasty bur may also be beneficial in reducing tension on the flap. Once a tensionless closure is ensured, the flap is then sutured in place with a fine monofilament absorbable suture material in a simple-interrupted pattern (Fig. 41.5B). The corners of the flap should be sutured first, followed by closure of the palatal and gingival margins. Sutures are then alternately placed in the mesial and distal aspects of the flap, progressing from the gingival margin to the most apical portion of the flap.



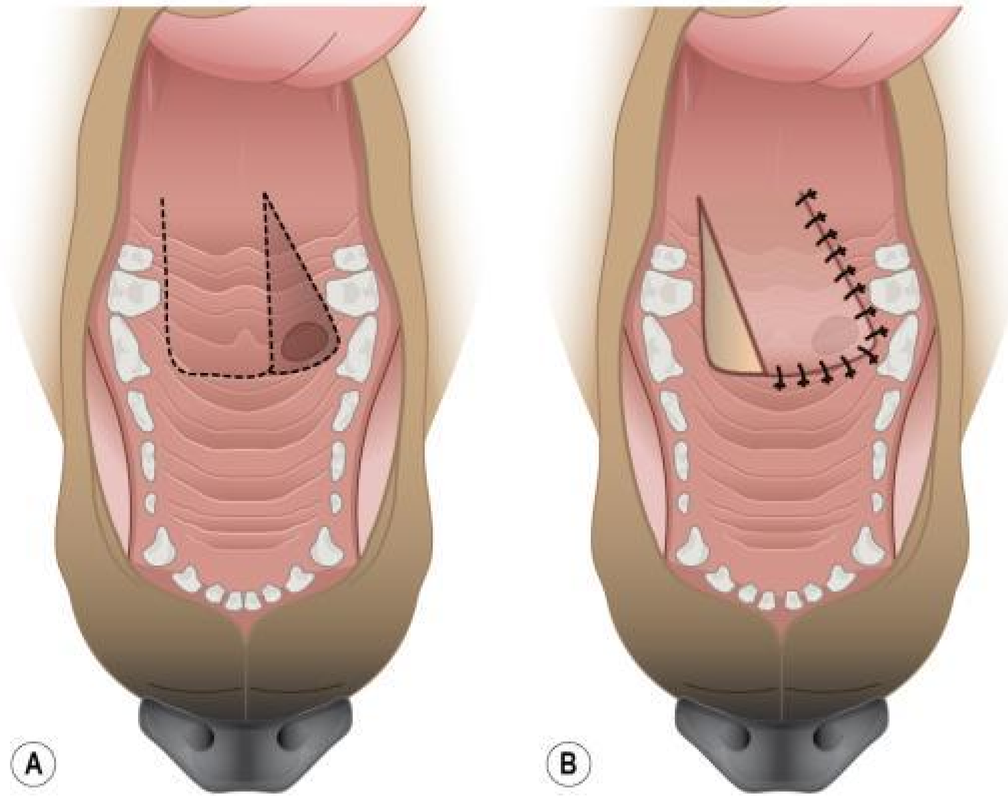
• **FIG. 41.5** Single-layer vestibular flap. (**A**) The rim of mucosa is excised from the margins of the fistula, and the proposed incision line for the vestibular flap is illustrated. (**B**) The vestibular flap is advanced over the defect and sutured in place.

Vestibular flaps that are used to close surgical sites following extensive maxillectomy procedures often require extensive undermining to prevent tension on the incision line. The vestibular mucosal flaps are created by undermining the mucosa and submucosa from the edge of the maxillectomy site toward the mucocutaneous junction of the lip until the flap can be brought into apposition with the subperiosteally elevated edge of the hard palate mucoperiosteum without tension. The vestibular flaps are sutured in position using a two-layer interrupted suture pattern using monofilament absorbable suture material. The initial or deep layer should be placed so that the knots are located in the nasal cavity, and the more superficial layer should be placed so that the knots are located in the oral cavity.

Transposition flaps

Transposition flaps can be used to repair defects located in the hard palate lateral to the midline and rostral to the maxillary fourth premolar tooth.[1,15] The palatal mucoperiosteal tissues contain less elastic tissue, thereby offering very limited pliability when compared to vestibular flaps. However, the palatal mucosa is keratinized and thicker than the unkeratinized buccal mucosa, providing increased strength in palatal flaps. In addition, procedures involving palatal transposition flaps do not affect the depth of the vestibulum.[16]

These flaps should be designed significantly larger than the defect to be covered. Initially, a 2–3-mm rim of tissue is tangentially excised from the entire circumference of the fistula. The transposition flap is then created by making a U-shaped incision with one arm of the U adjacent to the defect, leaving the caudal aspect of base of the flap intact. The removal of a small triangular segment (known as a Bürow's triangle) of mucoperiosteal tissue from the caudal aspect of the lesser curvature of the flap can facilitate adaptation of the flap over the palatal defect.[16] When creating the rostral aspect of the U-shaped flap, the major palatine artery will be encountered and may require ligation rostrally. The flap should be carefully raised using a curved Freer periosteal elevator or a P24G periosteal elevator to avoid traumatizing the major palatine artery. The flap is transposed to cover the defect and sutured in place using fine monofilament absorbable suture material in a simple-interrupted pattern. Because there is no soft tissue to secure the flap on the donor side, it is recommended that small holes be predrilled in the hard palate with a K-wire to allow placement of sutures to secure the flap. The flap is transposed to cover the defect, and sutures are preplaced between the inner layer of the flap and the predrilled holes in the palate while avoiding the major palatine artery. The flap is sutured in place using fine monofilament absorbable suture material in a simple-interrupted pattern. The exposed palatal bone of the donor site is allowed to heal by second intention (Fig. 41.6).



• FIG. 41.6 Transposition flap for repair of hard palatal defects lateral to the midline. (**A**) Shaded area represents proposed site for removal of perifistula palatal mucosa to permit rotation of proposed flap. (**B**) Transposition flap sutured in place over palatal defect.

Split palatal U-flap

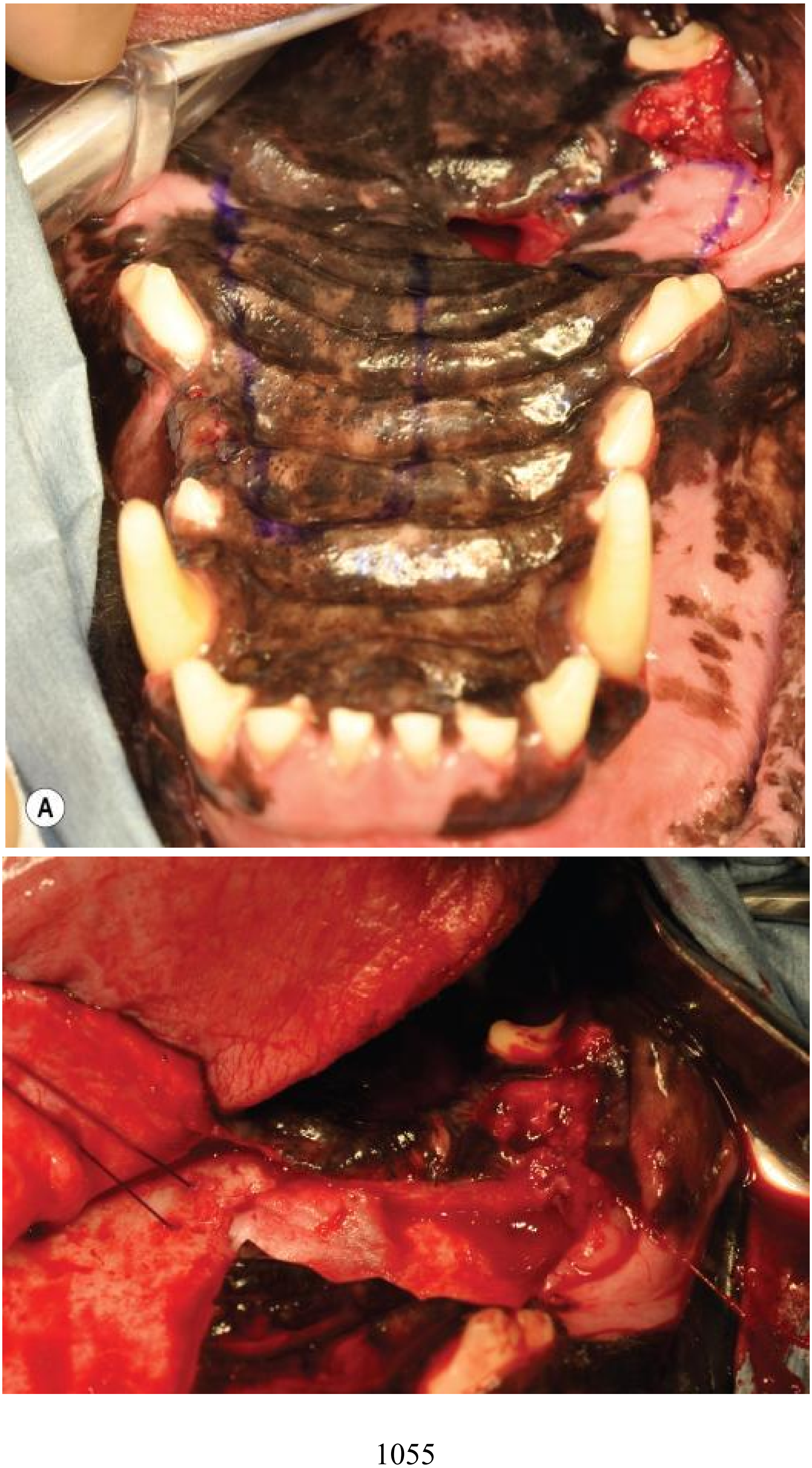
The split palatal U-flap has been described in 1991 for the repair of hard palatal defects that are centrally and caudally located.[17] The technique is infrequently used and no outcome studies are available. This surgical procedure is initiated with tangential excision of the epithelial margin of the defect with a # 15 scalpel blade. A large, full-thickness, U-shaped mucoperiosteal flap is created rostral to the defect (Fig. 41.7B). When creating the rostral aspect of the U-shaped flap, the major palatine arteries will be encountered bilaterally and must be ligated at the rostral aspect of the incision. The flap must be carefully elevated using a curved Freer periosteal elevator or a P24G periosteal elevator to avoid traumatizing the major palatine arteries bilaterally as they course along the palate rostrally after they emerge into the surface of the palate through the major palatine foramina. Gentle intermittent lifting of the flap from the underlying bone during periosteal elevation can help provide visualization of the major palatine arteries and will help prevent inadvertent laceration of these vessels.

• **FIG. 41.7** Split palatal U-flap. (**A**) Central hard palatal defect. (**B**) Proposed incision lines for creation of split U-flap. (**C**) Split U-flap sutured in place over the defect. Note that one flap is shorter than the other to minimize the amount of redundant tissue after securing them in proper position.

Following subperiosteal elevation of the U-shaped flap, the flap is incised along the midline using a # 15 scalpel blade. This divides the large U-flap into two flaps with a major palatine artery entering the base of each flap providing each flap with an excellent blood supply. One side of the hemisected U-flap is rotated 90 degrees over the palatal defect. The medial aspect of the flap is sutured to the caudal aspect of the palatal defect with fine monofilament absorbable suture material in a simple-interrupted pattern. The second side of the hemisected U-flap is rotated 90 degrees and transposed rostral to the previously rotated flap and sutured in place using a simple-interrupted suture pattern (Fig. 41.7C). Based on the surgeon's preference, the flaps can be either similar in size and shape or the one placed rostrally can be slightly longer than the caudal one covering the defect. The latter may help avoid redundancy of tissue and may facilitate adaptation to the area upon closure. Small holes may be predrilled in the palate rostral to the palatal defect using small K-wires in an area of the palate that will be covered by the second flap to help hold the flap in apposition with the hard palate rostrally. This is performed by preplacing sutures through the small predrilled holes in the palate and passing the suture split thickness through the palatal side of the second flap. The exposed bone at the donor site is allowed to heal by second intention and will generally reepithelialize within 1 month.

Double-layer techniques

Various double-layer closure techniques utilizing local tissues have been described for oronasal fistula repair.[2,13,16,18–20] Double-layer flap techniques include two separate flaps. The initial flap is reflected 180 degrees to provide a mucosal surface for the nasal passage and a vascularized support for the oral graft. The second flap is placed over the first flap and provides the oral mucosal surface for the repair. There are unique benefits associated with the use of double-layer and single-layer flaps. The double-layer flaps oppose a much larger area of connective tissue, which results in a stronger repair.[18] The disadvantages of the double-layer flap techniques are that they are more technically challenging to perform and increase the time required to perform the surgical procedure; they may increase donor site morbidity; and they often require strategic dental extractions a few weeks prior to repair if tissue quality and/or availability is a concern (Fig. 41.8).[16]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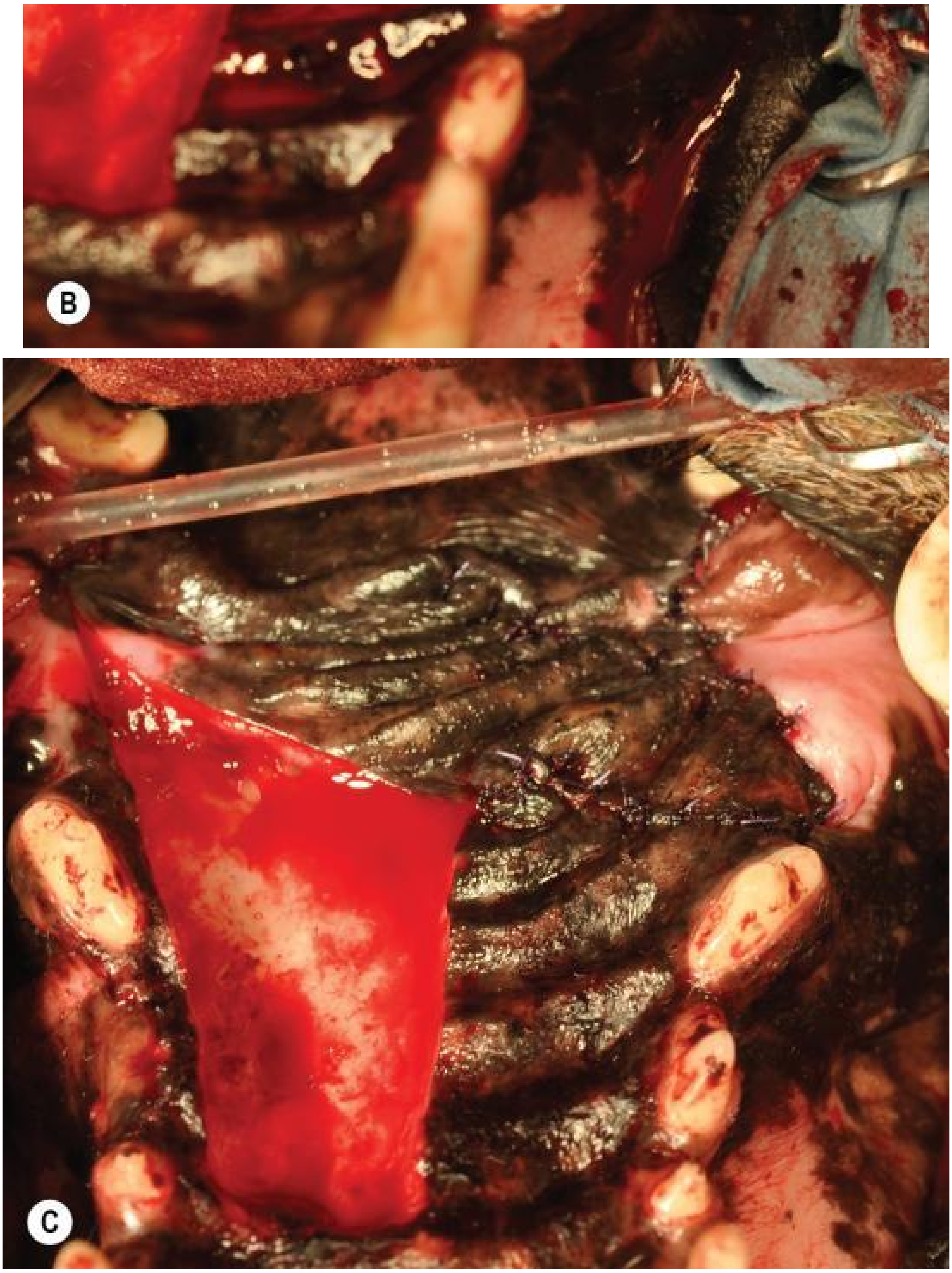
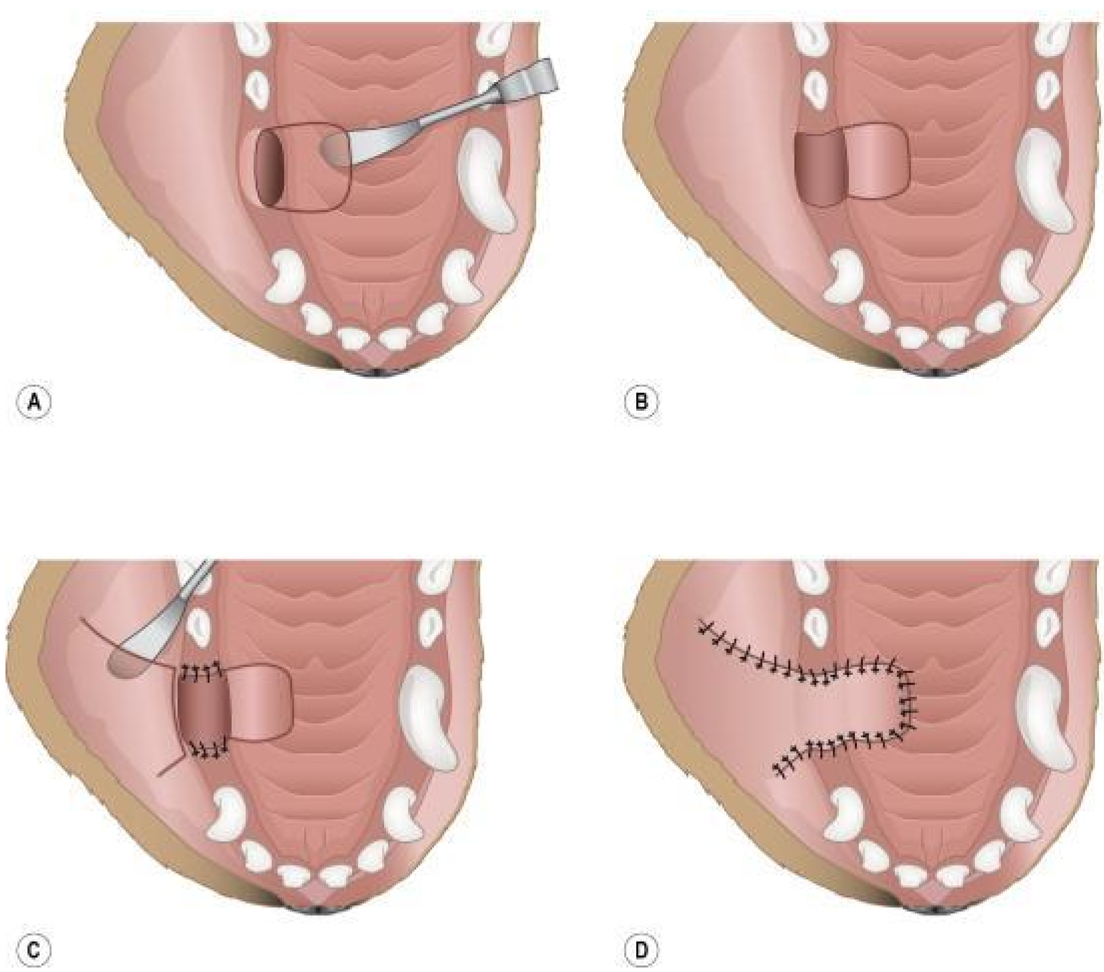
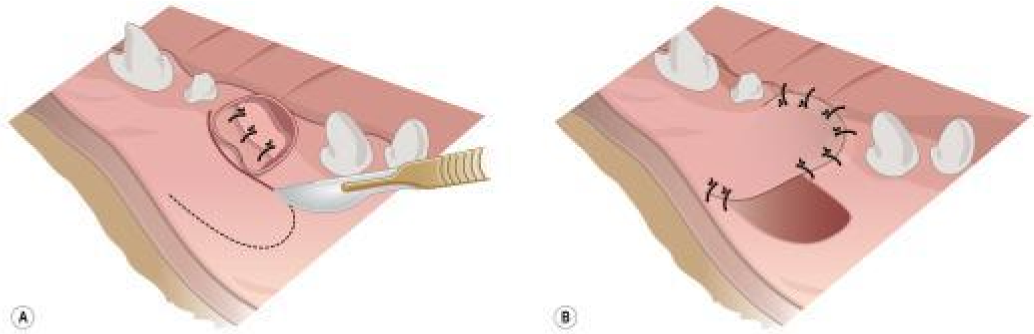


• FIG. 41.8 Double-layer flap. (**A**) Large circular oronasal fistula of traumatic origin on the caudal right hard palate. Note the planned incisions marked with surgical pen. (**B**) A full-thickness hard palate mucosal pedicle flap was elevated and reflected at the edge of the defect and secured to the bone on the opposing side. (**C**) A second pedicle flap was raised on the other side and transposed and secured to cover the initial flap, achieving a two-layer closure.

A double-layer technique can be applied for closure of oronasal fistulae associated with the maxillary canine teeth and/or premolar teeth secondary to severe periodontitis (Fig. 41.9).[2] For this, a flap is raised from the hard palate mucosa palatal to the oronasal fistula by making a U-shaped full-thickness incision. The flap should be of sufficient size to adequately cover the oronasal fistula without tension. The flap is gently elevated to the edge of the fistula, leaving the mucosa of the base of the flap intact. A full-thickness vestibular mucosal flap is then raised opposite the oronasal fistula. The defect is thoroughly debrided, and the epithelial lining is removed, except where the palatal flap hinges. The palatal flap is then hinged and secured to the submucosa of the vestibular flap. Alternatively, small holes in the bone surrounding the defect can be made with a K-wire so that the palatal flap can be secured to it. The palatal flap is sutured in place with 5-0 monofilament absorbable suture in a simple-interrupted pattern. The vestibular flap is then transposed over the palatal flap site and donor site. The vestibular flap is sutured in place with 4-0 monofilament absorbable sutures in a simple-interrupted pattern. If present, tension of the vestibular flap is released by undermining the deepest submucosal layers using a combination of blunt and sharp dissection. Variations and/or modifications of the double-layer techniques can be applied based on the unique characteristics of every defect, the surgical anatomy involved, and surgeon's preference (Fig. 41.10).



• **FIG. 41.9** Double-layer flap. (**A**) The mucosa palatal to the fistula is elevated with a periosteal elevator. (**B**) The palatal flap is hinged over the defect. (**C**) The palatal flap has been sutured in place and the vestibular flap is elevated. (**D**) The vestibular flap is sutured over the palatal flap and donor site.



• **FIG. 41.10** Double-layer flap. (**A**) The perifistula oral mucosa has been elevated and rotated into the defect and sutured in place; the proposed incision line for the vestibular mucosal flap is illustrated. (**B**) The second flap is sutured over the submucosal surface of the perifistula flap.

Miscellaneous techniques

Myoperitoneal microvascular free flaps

Myoperitoneal microvascular free flaps have been reported in the reconstruction of large acquired palatal defects in dogs in which multiple operations using local tissue flaps were unsuccessful.[21,22]

Utilization of a myoperitoneal microvascular free flap involves harvesting of the myoperitoneal flap from the body wall. The myoperitoneal flap may be based on the right cranial abdominal artery and vein and the cranial portion of the transversus abdominis muscle[22] or the caudal epigastric artery and vein and the rectus abdominis muscle.[21] Following isolation of the infraorbital artery and the superior labial vein and preparation of the palatal wound bed by incision of the palatine mucosa along the border of the oronasal fistula, microvascular techniques are utilized to perform the anastomosis. Following anastomosis of the vessels, the edges of the myoperitoneal flap are sutured under the elevated edges of the palatine mucosa using a combination of vest-over-pants sutures and a simple-interrupted pattern. Microvascular techniques require specialized training and access to an operating microscope and highly specialized surgical instruments and materials. Therefore they are currently only feasible in dedicated academic and/or veterinary research environments (see Chapter 11).

Angularis oris flap

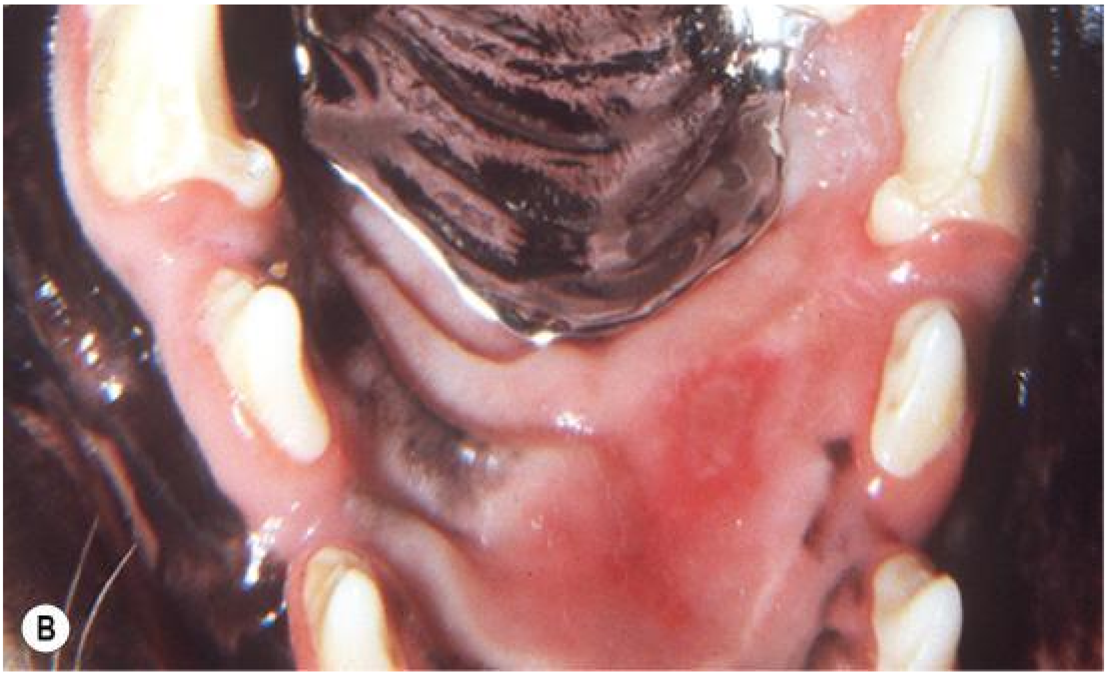
A vestibular flap that incorporates the angularis oris artery has been described in dogs.[23] In this technique, a relatively large area of tissue can be harvested from the vestibular mucosa immediately caudal to the commissure and mobilized to the hard and/or soft palate areas (see Chapter 52). The technique can be applied in cases in which the quality and quantity of available tissues are insufficient based on the size, nature, and/or severity of the oronasal fistula. Although rarely needed, this technique has been applied with relatively good success in cases of large and unfavorable oronasal fistulae of different etiologies.[24,25]

Obturators

Closure of large palatal defects may be difficult to achieve using autogenous tissue. In cases in which repeated attempts to close large palatal defects are unsuccessful, prosthetic appliances may be used to cover these defects.

In humans, various alloplastic materials have been used in the construction of obturators, including gold foil, gold plate, tantalum plate, soft polymethylmethacrylate, and lyophilized collagen.[16] Various types of obturators have been used in the treatment of large acquired palatal defects in dogs and cats, including permanent and removable, acrylic, silastic, and metal obturators (Fig. 41.11).[18,26-29]


A

• **FIG. 41.11** (**A**) Osteoradionecrosis of the hard palate in a dog following radiation therapy of a fibrosarcoma. (**B**) Palliative treatment with a stainless steel obturator following debridement. Source: (Photograph courtesy Dr. G.V. Ling, University of California, Davis, United States)

Direct fabrication of prosthetic acrylic obturators has been described[26]; however, most obturators are fabricated in a dental laboratory, necessitating two anesthetic episodes. During the initial anesthetic episode, an impression of both mandibles is obtained using alginate, and vinyl polysiloxane impressions of the maxilla and the defect are obtained using custom-made impression trays.[27] The impressions, along with clinical photographs of the palatal defect, are submitted to a dental laboratory. Stone models are made and used to construct the obturator. During a second anesthetic episode, the obturator is positioned into the defect and appropriately secured based on the design of the obturator.

Obturators requiring wire components for coronal attachment may not be useful in cats because of the morphologic features of their premolar and molar teeth.[28] A nasal septal button designed for repair of nasal septal perforations in humans has been used to successfully treat a large oronasal fistula in a cat.[28] The intranasal portion of the 30-mm Silastic button (Xomed-Treace, Jacksonville, FL) can be trimmed to fit into the defect. The use of this button should be considered for obturation of 7- to 25-mm oronasal fistulas in small animals that cannot be closed with autogenous tissue.[28]

Postoperative care and assessment

The possible risks and benefits of routine postoperative administration of systemic antibiotics after oronasal fistula repair in small animals have not been evaluated. The routine administration of systemic antibiotics after oral surgical procedures in humans is controversial.[30] Until clear evidence-based guidelines are established for animals, the routine use of postoperative systemic antibiotics after oronasal fistula repair cannot be recommended. Systemic postoperative antibiotics should be prescribed judiciously, limiting it to patients at risk of infection (i.e., immunosuppressed animals).

A minimum of one night of hospitalization is recommended postoperatively so that the airway can be monitored, possible complications addressed, and pain appropriately managed. The analgesics used should be determined based on the individual needs and systemic status of each patient. Many patients benefit from continuous infusion rates of single or combined agents. Because the nasal cavity of animals can be filled with blood clots and fluids for several hours after surgery, patients are often uncomfortable and may become dysphoric, especially immediately after anesthetic recovery. Because of this, some patients require sedation.

In most cases, oral intake can begin immediately following complete recovery from anesthesia. Oral intake should begin with a liquid or blended diet for approximately 2 weeks, followed by the slow conversion to a soft diet for an additional 4 weeks. Chew toys and other hard objects should be withheld for 6 weeks. Surgical sites should be reevaluated at 2 and 6 weeks postoperatively. Sedation or general anesthesia may be required to thoroughly assess healing of the surgical sites, especially in cases in which the soft palate was involved.

Complications

The most common complication associated with oronasal fistula repair is dehiscence. This complication can be minimized by performing tension-free closures, gentle intraoperative tissue handling, maintaining an adequate blood supply to the flap, providing adequate bony support of the suture line, and application of appropriate suturing techniques.

Surgical intervention for treatment of dehiscence following the initial attempt to repair the acquired palatal defect should be delayed for about 6 weeks because tissues are friable following dehiscence. This delay in surgery will help delineate the full extent of dehiscence and permit the tissues to revascularize and regain strength. Oronasal fistula formation following maxillectomies should be biopsied to rule out tumor persistence or recurrence.

Prognosis

The prognosis is very good for most animals following the repair of oronasal fistulae. Most oronasal fistulae can be closed surgically if the client is willing to persist with repeated procedures. Large inoperable defects have a guarded to poor prognosis, however. Utilization of free flaps and obturators can be considered in the management of large inoperable acquired palatal defects. Long-term use of obturators in animals may result in enlargement of the palatal defect secondary to chronic inflammation caused by the obturators and chronic rhinitis because of leakage around the obturators into the nasal cavity.

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SECTION 9:

Management of Maxillofacial Tumors and Cysts

OUTLINE

CHAPTER 42

Clinical staging and biopsy of maxillofacial tumors

Boaz Arzi, Frank J. M. Verstraete

General considerations

Malignant neoplasms of the oral cavity represent approximately 6% of all canine tumors[1]; the incidence is lower in cats.[2] A variety of neoplastic lesions occur, including both odontogenic and nonodontogenic tumor types. Nonneoplastic masses such as gingival hyperplasia and infectious conditions may be confused with oral tumors. Conversely, oral neoplasms may present as nonhealing, ulcerated lesions instead of "typical" prominent masses.[3] Oral tumors frequently go unnoticed by the animal's owner until the tumor reaches an advanced stage of development; it is therefore important to make an accurate assessment of the extent and nature of the condition at the first time of presentation.[4]

If the patient allows, the tumor should be carefully inspected and palpated. The size and location of the tumor, the presence of any ulceration, necrosis, and any abnormal mobility of the teeth are important findings and should be recorded. Fixation of the tumor to underlying tissues suggests bone infiltration; this possibility should be further investigated by diagnostic imaging. The regional lymph nodes are palpated to evaluate their size, shape, consistency, and fixation to underlying tissues. Irregular enlargement, and especially lack of mobility, is highly suggestive of lymph node involvement. However, lymph node metastasis may be present without palpable enlargement. Contrast computed tomography (CT) is very useful for evaluating lymph node involvement. Finally, the patient is thoroughly examined by means of inspection, palpation, auscultation, thoracic diagnostic imaging, and abdominal ultrasound to detect any signs of distant metastasis.

Neoplasms are diseases with their own course, and each tumor in each patient has its own characteristics.[5] The development and course of a tumor are determined by many factors associated with the neoplasm and the host.[5] One of the purposes for the classification and staging of a tumor is planning and evaluation of treatment options. Neoplasms can be classified in several ways, including clinical investigation into the extent of the disease (clinical staging), classification of the host and illness, histologic diagnosis, histologic grading, and prognostic significance. The expected biologic behavior of an oral tumor also depends on the species in which it occurs and the location in the oral cavity.[5] Understanding the biologic behavior enables the clinician to select the method of treatment indicated and to inform the client correctly.

Clinical staging

Due to a historical lack of agreement in categorizing malignancies and their therapeutic results in humans, a clinical staging system was adopted by the International Union Against Cancer.[5] The stage of disease at the time of diagnosis may be a reflection of not only the rate of growth and extension of the tumor but also of the type and tumor-host relationship. The term *stage* does not imply a regular progression from stage 1 to 4; rather, stages are arbitrary divisions often related to prognosis and treatment.[5]

The TNM system was adopted by the World Health Organization–Collaborating Center for Comparative Oncology and is basically the same as the CTNM staging of human cancers.[5,6] These systems are based on the assessments of:

1. The extent of the primary tumor—T
2. The involvement of the regional lymph node—N
3. The absence or presence of distant metastases—M

The addition of numbers to these components (e.g., T_1, T_2, and T_3) indicates the extent of the neoplasm or involvement. Site-specific clinical staging according to the TNM system is shown in Boxes 42.1–42.3.[6]

• BOX 42.1

Clinical Staging of Tumor of the Lips (TNM)[6]

T Primary tumor
- T_{is} Preinvasive carcinoma (carcinoma in situ)
- T_0 No evidence of tumor
- T_1 Tumor <20 mm maximum diameter, superficial or exophytic
- T_2 Tumor <20 mm maximum diameter, with minimal invasion in depth
- T_3 Tumor >20 mm diameter with deep invasion irrespective of size
- T_4 Tumor invading bone

N Regional lymph node (RLN): superficial cervical, medial retropharyngeal, mandibular and parotid
- N_0 No evidence of RLN involvement
- N_1 Movable ipsilateral nodes
- N_{1a} Nodes not considered to contain growth [histologically (−)]
- N_{1b} Nodes considered to contain growth [histologically (+)]
- N_2 Movable contralateral or bilateral nodes
- N_{2a} Nodes not considered to contain growth [histologically (−)]
- N_{2b} Nodes considered to contain growth [histologically (+)]

N_3 Fixed node

M Distant metastasis

M_0 No evidence of distant metastasis

M_1 Distant metastasis (including distant nodes)

• **BOX 42.2**

Clinical Staging of Tumors of the Oral Cavity (TNM)[6]

T Primary tumor

T_{is} Preinvasive carcinoma (carcinoma in situ)

T_0 No evidence of tumor

T_1 Tumor <20 mm maximum diameter

T_{1a} Without bone invasion

T_{1b} With bone invasion

T_2 Tumor 20–40 mm maximum diameter

T_{2a} Without bone invasion

T_{2b} With bone invasion

T_3 Tumor >40 mm diameter

T_{3a} Without bone invasion

T_{3b} With bone invasion

N Regional lymph node (RLN): superficial cervical, medial retropharyngeal, mandibular and parotid

N_0 No evidence of RLN involvement

N_1 Movable ipsilateral nodes

N_{1a} Nodes not considered to contain growth [histologically (−)]

N_{1b} Nodes considered to contain growth [histologically (+)]

N_2 Movable contralateral or bilateral nodes

N_{2a} Nodes not considered to contain growth [histologically (−)]

N_{2b} Nodes considered to contain growth [histologically (+)]

N_3 Fixed node

M Distant metastasis

M_0 No evidence of distant metastasis

M_1 Distant metastasis (including distant nodes)

• **BOX 42.3**

Clinical Staging of Tumors of the Oropharynx (TNM)[6]

T Primary tumor

T_{is} Preinvasive carcinoma (carcinoma in situ)

T_0 No evidence of tumor

T_1 Tumor superficial or exophytic

T_{1a} Without systemic signs

T_{1b} With systemic signs

T_2 Tumor with invasion of tonsil only

T_{2a} Without systemic signs

T_{2b} With systemic signs

T_3 Tumor with invasion of surrounding tissue

T_{3a} Without systemic signs

T_{3b} With systemic signs

N Regional lymph node (RLN): superficial cervical, medial retropharyngeal, mandibular and parotid

N_0 No evidence of RLN involvement

N_1 Movable ipsilateral nodes

N_{1a} Nodes not considered to contain growth [histologically (−)]

N_{1b} Nodes considered to contain growth [histologically (+)]

N_2 Movable contralateral or bilateral nodes

N_{2a} Nodes not considered to contain growth [histologically (−)]

N_{2b} Nodes considered to contain growth [histologically (+)]

N_3 Fixed node

M Distant metastasis

M_0 No evidence of distant metastasis

M_1 Distant metastasis (including distant nodes)

Primary tumor

T_0 means that no primary tumor has been detected. T_1, T_2, and T_3 indicate an increasing degree of extent of the primary tumor. T_x means that it is impossible to fully determine the extent of the primary tumor. T_{is} is reserved for carcinoma in situ (early carcinoma with the absence of invasion of the surrounding tissue).[5] The tumor extent is commonly based on three features: the depth of invasion, surface spread, and size. Tumor size is determined by the amounts of vital and necrotic tumor tissue, secretion product, cystic structures, stroma, and inflammatory reaction. Inflammation can often be found around and in the tumor and may cause a false impression of the tumor size and invasion to adjacent tissues.[5]

Diagnostic imaging of oral tumors

Radiography forms an integral part of assessing the tumor characteristics, in particular the extent of the tumor and the presence of bone involvement.[3] Intraoral dental radiographs are generally indicated in cases of suspected oral neoplasia. Importantly, advanced diagnostic imaging techniques are indicated when oral neoplasia is suspected and/or clinically confirmed.[7] Tumors located in the maxilla necessitate CT to visualize the intranasal and/or periorbital extent of the tumor. In addition, CT is also indicated with most mandibular lesions, to evaluate possible temporomandibular joint (TMJ) involvement and to assess the regional lymph nodes. Regardless of the location, CT is beneficial for evaluating the extent of the mass as well as for surgical planning (see Chapter 6). Magnetic resonance imaging (MRI) and ultrasound can be useful for visualizing tumors with deep soft tissue infiltration and for evaluating lymph nodes.[3]

The radiologic findings associated with oral tumors are often subtle and nonspecific. Careful and systematic evaluation of radiographs, using specific radiologic descriptors, may make it possible to associate different patterns with certain tumor types and/or suggest a benign or malignant (aggressive) lesion.[8,9] However, with the exception of odontomas, the type of tumor cannot be determined solely based on radiographs or CT, and biopsy is always required for definitive diagnosis. It is equally important to match the radiologic findings with the clinical features and location of the tumor and, following biopsy, with the histopathological findings.[3]

Bone involvement may be evidenced by varying degrees of bone resorption and/or new bone formation. It is generally accepted that bone lysis only becomes evident radiographically when more than 40% of the cortical bone has been demineralized; radiographs therefore usually underestimate the extent of the tumor.[1] However, the presence of bone lysis is an indication of advanced bone infiltration, which influences the therapeutic plan.

Regional lymph nodes

The staging of lymph nodes (N) may be assessed by palpation that consists of size, mobility versus fixation, firmness, single versus multiple nodes, ipsilateral-contralateral, and bilateral distribution. The size of the node is important; however, it is not a strong enough criterion to indicate ascending order of progression, because an enlarged lymph node is not necessarily due to metastasis (e.g., reactive lymph node).[5,10] In one study, only 17% of palpably enlarged mandibular lymph nodes in dogs and cats with oral tumors were found to contain metastases.[11] Mobility versus fixation is a more important consideration than size, because loss of mobility may be an indicator of invasion of the lymph node capsule by the neoplasm, which has been found to be associated with a poor prognosis.[10]

Diagnostic imaging of regional lymph nodes using contrast CT is essential. If there is any suspicion of involvement, a fine-needle aspirate or excisional biopsy is indicated.

Regional lymph node filter function has traditionally been assumed to be crucial in the prevention of the systemic spread of malignant cells shed from the primary neoplasm.[12,13] However, in a multitude of clinical studies, prophylactic removal of such regional lymph nodes did not improve the cure rate compared with the observation of these nodes.[14,15] In addition, laboratory studies indicate that lymph node filter function may be neither complete nor effective and that many lymphatic and lymphaticovenous shunts exist that bypass regional lymph nodes and allow both lymphatic and hematogenous dissemination of malignant cells.[12,13] Therefore lymph node metastases should be regarded as indicators but not governors of survival in cancer. The timing of the clinical appearance of regional lymph node metastases and their number are, in most cases, excellent indicators of the biologic behavior of the primary cancer.[12,13]

Distant metastasis

The absence or presence of distant metastases is designated by the letter M, where M_0 indicates absence and M_1 indicates the presence of distant metastasis. Metastatic involvement of the abdominal cavity is best assessed by abdominal ultrasound, which is currently considered the standard of care. Another diagnostic tool that may become available in the future is ultrasound with contrast media. Preliminary research suggests that this modality detects more lesions and may be better at differentiating benign and malignant lesions than ultrasound without contrast.[16-18] CT, with and without contrast, and MRI are excellent alternatives; however, they require general anesthesia, take more time, and are more expensive than ultrasound. Thoracic CT has been found to be significantly more sensitive than thoracic radiographs for detecting soft tissue nodules.[19] Only 9% of CT-detected pulmonary nodules are identified on thoracic radiographs.[19] The lower size threshold is approximately 1 mm to detect pulmonary nodules on CT images and 7–9 mm to reliably detect nodules on radiographs. Therefore thoracic CT should be considered in any patient with neoplasia that has potential for pulmonary metastasis, particularly when accurate characterization of the extent and distribution of pulmonary metastasis affects therapeutic planning.[19]

Histopathologic staging

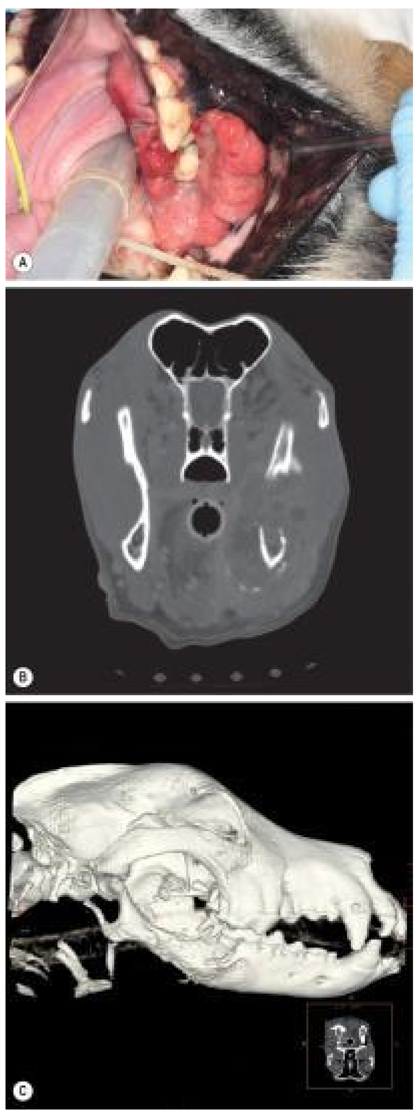
Principles of oral and maxillofacial biopsy

Proper management of a patient with an oral tumor starts with accurate diagnosis.[20] The current gold standard for definitive diagnosis is histopathologic assessment of a tissue biopsy from the lesion. Although biopsy frequently demands relatively few technical skills, the decisions related to the performance of the biopsy require considerable thought and experience.[21] Without appropriate planning and execution, biopsies may lead to adverse effects on patient prognosis and on treatment options. Poorly performed biopsies, poorly placed incisions, and biopsy complications can considerably compromise the local management of bone and soft tissue tumors.[21,22] In addition to accurate histopathologic diagnosis, which depends on the clinician performing an appropriate biopsy and providing adequate clinical information, the diagnosis depends on the pathologist correctly interpreting the biopsy results. To carry out the biopsy appropriately, the surgeon should ensure that adequate diagnostic and staging studies have been performed. These studies include clinical, laboratory, and radiologic assessments to provide the surgeon with knowledge of the extent of the tumor as well as its systemic involvement.[21,23] Since biopsy has potential prognostic and therapeutic consequences, it is recommended that it should be undertaken by the surgeon who plans to carry out the definitive treatment.[21,23]

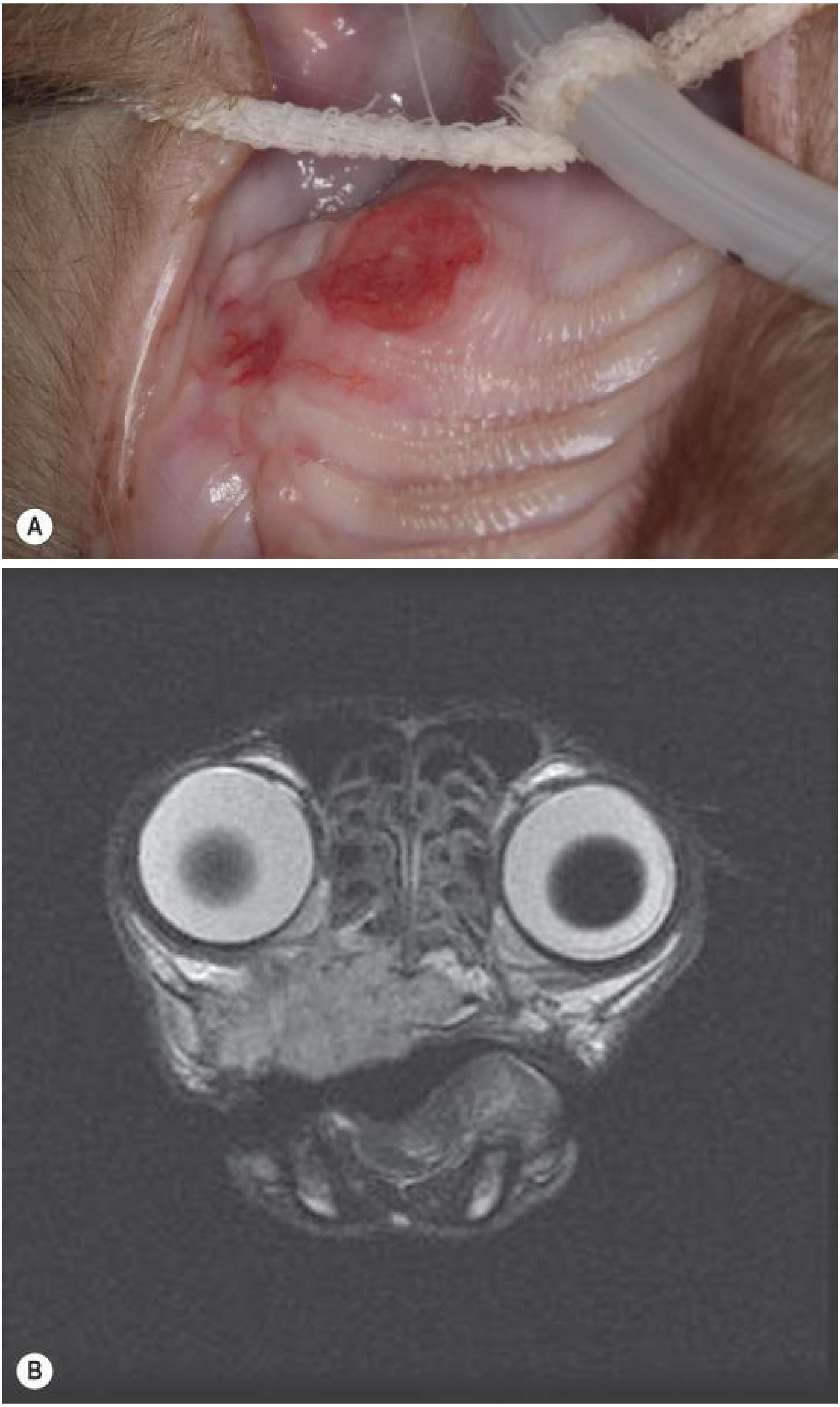
Developing the biopsy strategy

Ideally, the management of oral tumors involves a multidisciplinary approach.[21] In addition to the surgeon, radiologists and pathologists should play a role in the planning of the diagnostic and staging strategy prior to biopsy. Depending on the suspected differential diagnosis, prebiopsy consultation may also include radiation oncologists, medical oncologists, and field-specific surgical specialists.[21] For example, a patient with a clinically extensive maxillary tumor that is not amenable to surgical resection may be prepared for radiation treatment during the diagnostic stages (e.g., molding of a custom-fitted mask for radiotherapy during the same anesthetic episode as CT imaging).

The mainstay of planning the biopsy sites relies on diagnostic imaging. A conventional radiograph of a soft tissue mass does not usually provide any diagnostic information.[21,24] In addition, the maxillofacial region has complex structures and therefore is difficult to accurately evaluate, especially in the presence of oral or maxillofacial tumors. Therefore the gold standard of biopsy planning should include advanced diagnostic imaging such as CT, ideally with contrast (Fig. 42.1), and possibly MRI (Fig. 42.2). In the oral and maxillofacial region, CT is preferred due to its ability to demonstrate the extent of bone involvement in finer detail.[25] However, in selected cases with a soft tissue mass that may invade other structures (e.g., tongue base tumor), MRI may be beneficial.



• **FIG. 42.1** (**A**) Squamous cell carcinoma at the right caudal mandible of a 12-year-old German shepherd dog (with the patient in dorsal recumbency). (**B**) Note the severe bony destruction demonstrated by computed tomography and (**C**) the tridimensionally reconstructed image.



• **FIG. 42.2** (**A**) Squamous cell carcinoma at the hard palate of a 14-year-old cat (with the cat in dorsal recumbency). (**B**) Note the severe infiltration of the tumor into the nasal cavity and the ventral aspect of the left orbit as demonstrated by the magnetic resonance image.

When obtaining CT images, 0.5–1.0-mm CT sections provide more information than 3.0- or 5.0-mm sections do. In addition, three-dimensional reconstruction images from 0.5- or 1.0-mm sections provide a better platform for biopsy and treatment planning. The diagnostic value of CT and MRI is especially beneficial for lesions involving the hard and soft palate. In these locations, incorrect selection of the biopsy site may lead to permanent oronasal communication and associated morbidity. Moreover, CT is also extremely useful in evaluating masses at the caudal mandible, in the area of the coronoid process and the TMJ.[3]

In addition to CT and/or MRI, obtaining dental radiographs of the area involved is recommended, as these will demonstrate the extent of involvement of adjacent teeth and alveolar bone associated with the mass.[3,26,27]

An appropriate biopsy contains tissue that is representative of the most severe or significant change in the lesion. Achieving this involves three key factors: selection of the biopsy site, the procedure used to collect the samples, and proper submission of the biopsy sample.[20] An oral tumor, especially a large mass, often varies in disease severity from one part of the lesion to another. For example, a lesion may have early invasive squamous cell carcinoma in one part and dysplasia in another.[20,28] As a rule, an appropriate biopsy should include tissue from the worst part of the lesion.[20] However, the worst part of a lesion may not be readily apparent from its clinical and radiographic appearance; therefore collection of multiple biopsies is recommended.[20] For example, for a 40-mm lesion, obtaining two biopsies from representative areas or those with different clinical appearance is justified. The biopsies should always remain within the lesion: obtaining adjacent normal-appearing tissue for comparison, as recommended for dermatologic lesions, is contraindicated.[29] The biopsy track also should not transect a normal anatomical musculoskeletal compartment to reach a compartment that is affected by neoplasia, so that it will not be necessary to remove both compartments at the time of the definitive procedure.[21] Also, because infection may be misinterpreted clinically as a tumor, or the reverse, it is often advisable to obtain additional specimens for aerobic, anaerobic, and fungal cultures and sensitivity.[30]

Open and closed biopsy

In a closed biopsy procedure, no incision is required and the tissue specimen is obtained by skin or mucosal puncture, most commonly with a needle.[21] An open biopsy, which requires incision, has been the most common and conventional method. In an open biopsy, the surgeon can obtain a relatively large amount of tissue for diagnosis.[21] The larger tissue specimen may help the pathologist make a more accurate diagnosis. An open biopsy also decreases the likelihood that the surgeon will make a sampling error.[21,23] However, the risk, complications, and consequences of poor biopsy placement increase with open compared to closed biopsy. In addition, open biopsy is more likely to result in hematoma, tumor cell spillage, operative infection, and, if bone is entered, iatrogenic fracture.[21] Nevertheless, open biopsy is the method of choice in oral and maxillofacial biopsies[21] because aspiration of oral tumors, particularly those that derive from connective tissue, usually results in collection of too few cells to provide an accurate diagnosis.[31] Similarly, "impression smears" and scraping the tumor tissue usually do not provide an adequate sample for interpretation.[31] In addition, basic histopathologic diagnostic methods may not be sufficient for determining the diagnosis, and immunohistochemical studies will be needed in many cases to differentiate among many types of malignancies. In these cases, procurement of a large biopsy specimen initially will avoid the need for rebiopsy.

For soft tissue lesions, closed biopsy may reduce the cost of biopsy and the time required for diagnosis.[32] Fine-needle aspiration of soft tissue masses can be carried out with a relatively small needle (0.7 mm in diameter).[21,32,33] The reported diagnostic accuracy in nonoral masses for determining malignancy is 90%.[32,33] However, the accuracy rate is lower for determination of a specific tumor type, or histological grade, because of limited tissue volume obtained and the loss of tissue architecture. Fine-needle aspiration may be useful in the determination of local recurrence of soft tissue tumors and spread to lymph nodes.[21] With the use of the Tru-cut needle biopsy system (Carefusion, Vernon Hills, IL), it is possible to obtain a limited amount of tissue from the lesion with the original architecture preserved.[21] The reported accuracy in nonoral tumors is 96%.[34,35] However, because the amount of tissue retrieved is limited, the disposition of the material must be planned carefully before the tissue is processed, preferably by a pathologist experienced in oral pathology.[21] It should be emphasized that despite the high degrees of accuracy reported (in nonoral lesions), a negative or nondiagnostic closed biopsy should not be interpreted as a reassurance that no tumor exists, as the sample may be from normal adjacent tissue, superficial inflammatory tissue, or from a pseudocapsule surrounding the tumor.[21]

Open biopsy procedures

An open biopsy may be incisional or excisional. Incisional biopsy is most common and is the procedure of choice for open biopsy of most tumors.[21] The decision to perform a primary excision of a mass is complex. As a general rule, if the mass is small, pedunculated, and its complete removal will not affect the treatment options, excisional biopsy can be attempted. However, excisional biopsy of a large or deep mass may cause extensive tumor contamination at the time of biopsy and can subsequently limit treatment options.[21]

Soft tissue biopsy

A number of biopsy techniques are available for soft tissue biopsy, including scalpel, punch biopsy, laser, and electrosurgery.[20] For biopsy of mucosal lesions, the use of laser or electrosurgery should be avoided, as these techniques produce a coagulation artifact that jeopardizes histologic interpretation of the specimen.[20] They are also more traumatic to the mucous membranes and are associated with higher tissue morbidity and suture dehiscence.

Prior to biopsy, administration of local anesthesia is recommended, either as a regional block (such as infraorbital, inferior alveolar, or mental blocks) or local infiltration administered to the area adjacent to the biopsy site (not directly into the biopsy site, to avoid distortion artifacts in the specimen).[20]

Punch biopsy (Fig. 42.3) has been shown to produce fewer artifacts than scalpel biopsy and is considered by the authors to be the method of choice to obtain biopsy specimens from oral soft tissue masses.[36] Ideally, biopsies of the mucosa should be at least 3 mm in diameter.[20] Since biopsies shrink after formalin fixation, punch biopsies 4–6 mm in diameter are recommended.[20] The depth should be at least 2 mm. However, for some malignancies, deeper biopsies such as 4–

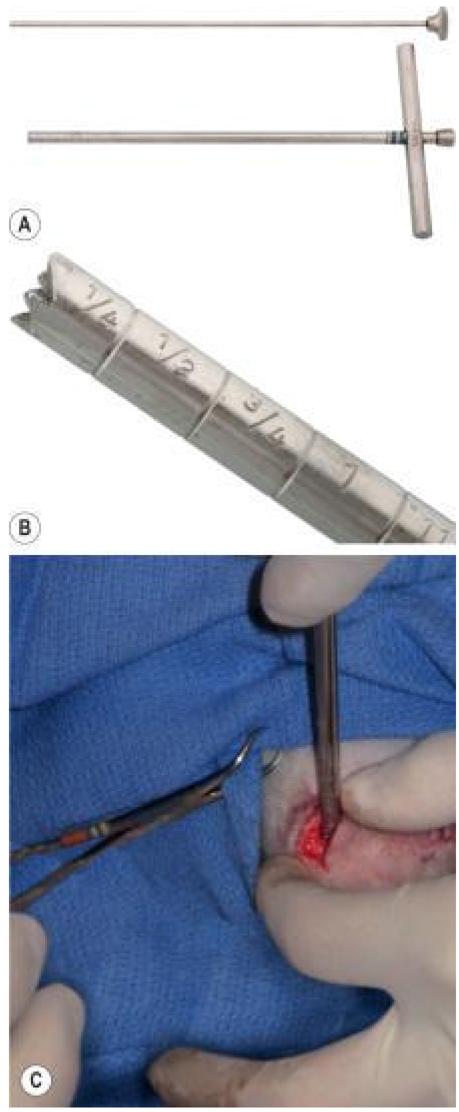
5 mm are needed.[20] The bevel of the cutting edge can be measured and used as a depth guide. The punch should be gently inserted into the lesion with a rotating motion to facilitate cutting the tissue. Once an appropriate depth has been reached, the punch can be gently tilted to the sides to facilitate the removal of the sample ("scooping"). Alternatively, tissue forceps and a scalpel may be used to remove the biopsy sample. If possible, the biopsy site should be sutured with 5–0 or 4–0 poliglecaprone 25 (Monocryl, Ethicon, Guaynabo, Puerto Rico) in a simple interrupted pattern to achieve hemostasis. If suturing is not possible, the biopsy site should be left open, and hemostasis can be achieved by applying digital pressure with sterile wet gauze. In some cases, absorbable hemostatic agents may be used.

• **FIG. 42.3** Biopsy punch instruments used for biopsy of soft tissues in 4-mm, 6-mm, and 8-mm diameter (from bottom to top).

Hard tissue biopsy

When selecting the site for an incisional biopsy of a hard tissue mass, the clinician should assess the radiographs and CT images to locate the least differentiated or least mineralized portion of the mass, because this is usually the most representative portion.[21] The authors use two methods for biopsy of hard tissue mass: an appropriate-size osteotome (e.g., 4 mm, 6 mm, or 8 mm) and mallet (Fig. 42.4) or Michele trephine (Fig. 42.5). Neither technique has been reported to be superior. However, Michele trephine is more prone to produce crushing artifacts of the specimens.

• FIG. 42.4 (**A**) 100-g mallet and osteotomes (8 mm, 6 mm, and 4 mm, from top to bottom, respectively) used for biopsy of hard tissue masses. (**B**) Intraoral biopsy at the right mandible of a dog using 100-g mallet and 4-mm osteotome.



• **FIG. 42.5** **(A)** Michele trephine *(bottom)* and its stylet *(top)* used for biopsy of hard tissue masses. (**B**) The tip of the Michele trephine; note the serrated conformation of the tip and the ¼-inch increments marks. (**C**) Extraoral biopsy at the left mandible using Michele trephine.

Osteotome technique

Once the tissue to be biopsied is identified, the osteotome should be placed at the desired biopsy site. Then, a 100-g mallet is used to drive in the osteotome in gentle, rapid strokes until the desired depth has been achieved. A second incision should be made 5 mm away, at an angle to the first incision in the same manner, creating a wedge-shaped biopsy specimen. Once the two incisions have been made, the surgeon should gently wiggle the osteotome until the sample detaches from the underlying tissue. A tissue forceps can then be used to remove the specimen, which should be in a wedge shape. The depth of the biopsy depends on the size of the mass. Care must be taken not to create an iatrogenic fracture, especially when wiggling the osteotome.

Michele trephine technique[37]

Once the mass is exposed, the trephine is placed at the desired location. It is held with one hand while the other hand helps secure it in place. The trephine is guided downward with slight pressure with a slight twisting action, which leads the trephine into the mass. Once the proper depth has been achieved, the trephine is rocked gently and then removed. The operator can then use the stylet, inserting it into the trephine with gentle pressure, to remove the specimen, which should be in a cylindrical shape.

Tissue handling and submission of the specimen

Hard tissue specimens should be placed immediately in a 10% neutral buffered formalin fixative. Soft tissue samples obtained with a punch biopsy should be placed on a piece of clear paper with the connective tissue surface facing down for 1 minute to ensure that the sample stays flat and to prevent it from curling during fixation, and to orient it properly for histologic examination.[20] The sample is then placed in a 10% neutral buffered formalin fixative. To avoid improper fixation or autolysis, the volume of fixative should be at least 20 times the volume of the sample.[20] No other fixative should replace the formalin. Alcohol, surface disinfectant, local anesthesia solution, or mouth rinse cannot fix the tissue properly for adequate histologic analysis.[20] The biopsy specimen should always be accompanied by pertinent clinical information, including a detailed description of the mass and its radiographic appearance, preferably accompanied by a clinical color image to facilitate the clinical-pathologic correlation.[38] If other imaging was performed, details of those findings should also be included. If multiple lesions were biopsied, each sample should be submitted separately and labeled accordingly. A close rapport between the pathologist and surgeon facilitates communication, streamlining the diagnosis and management of oral lesions.

Frozen sections

The development of the cryostat in the late 1950s led to the use of frozen section biopsy in most human hospitals[39] and some veterinary hospitals. The delay between procurement of a biopsy specimen and receipt of the results is difficult for both the client and the clinician. It is therefore desirable to make a definitive diagnosis from the initial biopsy as quickly as possible and to avoid repeated anesthetic episodes for rebiopsy if the first sample is not diagnostic. Obtaining an intraoperative frozen section may ensure that diagnostic tissue has been obtained, thus avoiding repeat biopsy.[40] Analysis of a frozen section can be of great value to the surgeon, but both the surgeon and pathologist should agree that such a test be used only when interpretation can provide information of immediate or ultimate value in patient management.[39] In addition, the clinician should be aware that tissue examination using frozen samples is much more difficult than when using fixed tissue. Therefore some of the tissue should be saved for permanent and definitive microscopic examination, and the final decision regarding definitive treatment should be made after an assessment of the fixed tissue.[39]

Postoperative considerations

The biopsy of an oral lesion is a relatively minor operation, performed under general anesthesia accompanied by local anesthesia. However, this procedure can be associated with pain and swelling. The human literature describes maximum pain intensity 2 hours following a biopsy and swelling that peaks between 6 and 48 hours after biopsy.[41] There is considerable reduction in pain after the third day post-surgery, and from this point onward, the pain decreases gradually during the course of the week.[41] Therefore it is highly recommended to follow a biopsy procedure with appropriate analgesia for 3–7 days and to recommend a soft diet and avoidance of chew toys or treats for the same time period.

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CHAPTER 43

Clinical-pathologic correlations

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Clinical-pathologic correlations

When historical data and physical signs associated with an oral condition are not clinically diagnostic, microscopic evaluation is generally required. Definitive diagnosis is usually apparent from histopathologic features, although results may occasionally be equivocal and subject to more than one interpretation. Additional special or immunohistochemical stains may help resolve these difficult cases. Importantly, the correlation of clinical and pathologic features can be of considerable benefit. That is, the putative diagnosis should correlate or be consistent with the expected patient age, anatomic site, species, and breed. For example, a diagnosis of canine acanthomatous ameloblastoma would be highly unlikely in a cat, and likewise, a diagnosis of feline inductive odontogenic tumor would be inappropriate for a dog. Among the limited number of oral diseases occurring in dogs and cats, some diseases and lesions seem to be relatively restricted to one species or the other (Table 43.1). Other situations in which clinical-pathologic correlations are important are when the pathologist overinterprets the histopathology (e.g., fibrosarcoma for a focal fibrous hyperplasia or squamous cell carcinoma for acanthomatous ameloblastoma) or underinterprets the histopathology (e.g., peripheral ossifying fibroma for low-grade osteosarcoma). Accurate and complete historical and clinical data can be of immense help to the pathologist in biopsy interpretation. Clinical-pathologic correlation is essential for definitive diagnosis, particularly in difficult and equivocal cases.

TABLE 43.1

Oral Diseases in Dogs and Cats: Summary of Biopsy Specimens From the University of California–Davis Dentistry & Oral Surgery Service (1995–2001)

	Dogs	Cats
Inflammatory/Hyperimmune	**(25%)**	**(53%)**
Gingivitis/gingival hyperplasia	33	10
Ulcerative gingivitis/stomatitis	19	17
Periodontitis	3	2
Mucositis (stomatitis)	21	18
Eosinophilic granuloma	3	2
Osteomyelitis	7	8
Pulpitis	3	0
Hyperplasias	**(31%)**	**(9%)**
Pyogenic granuloma	2	2
Peripheral fibroma/fibrous hyperplasia	59	6
Peripheral ossifying fibroma	45	0
Peripheral giant cell granuloma	3	0
Papilloma	2	1
Lymphoid hyperplasia	3	1
Cysts	**(3%)**	**(2%)**
Periapical cyst/granuloma, abscess	6	1
Dentigerous cyst	2	0
Lymphoepithelial cyst	0	1
Gingival cyst	2	0
Benign Neoplasms	**(4%)**	**(1%)**
Adenoma (salivary)	2	0
Granular cell tumor	3	0
Plasmacytoma	4	0
Lipoma	1	0
Fibromatosis	2	0
Osteoma/osteochondroma	1	1
Benign Odontogenic Tumors	**(13%)**	**(3%)**

	Dogs	Cats
Canine acanthomatous ameloblastoma	39	0
Central ameloblastoma	2	0
Amyloid producing odontogenic tumor	1	2
Myxoma	1	0
Odontoma	5	0
Feline inductive odontogenic tumor	0	1
Malignant Neoplasms	**(25%)**	**(32%)**
Squamous cell carcinoma	21	20
Papillary squamous cell carcinoma	4	0
Verrucous carcinoma	1	1
Fibrosarcoma	16	1
Rhabdomyosarcoma	1	0
Osteosarcoma	8	2
Mast cell tumor	2	3
Giant cell tumor (central)	2	0
Melanoma	26	0
Lymphoma	2	3
Adenocarcinoma	1	1
Metastatic adenocarcinoma/carcinoma	0	2
Sarcoma NOS (not otherwise specified)	5	1
	N = 363	N = 107

The clinician who has a working knowledge of histopathology will be better able to correlate microscopy with clinical features and to ultimately determine definitive diagnosis. This knowledge also gives the clinician an appreciation of underlying disease processes and an edge in clinical interpretation. Cellular and tissue changes are reflected in presenting appearances, giving the knowledgeable clinician, who is aware of these correlations, greater skill in developing a rational differential diagnosis and diagnostic plan. The cases discussed in the following serve to illustrate the importance of clinical correlation, differential diagnosis, and appreciation of histopathology.

Case 1

A 5-year-old golden retriever was presented with a 40 × 50 mm swelling of the incisive part of the mandible (Fig. 43.1A). The lesion was the same color as the surrounding tissue and was covered by intact epithelium. A radiograph showed resorption of subjacent bone (Fig. 43.1B).

The exact duration of the lesion was unknown, but it had recently been rapidly growing. Believed to represent a gingival soft tissue tumor, a clinical differential diagnosis was developed that included connective tissue neoplasm, acanthomatous ameloblastoma, and squamous cell carcinoma. A connective tissue neoplasm was favored because the latter two lesions both characteristically exhibit irregular ulcerated surfaces, even though they can present with similar architectural features in this site (Figs. 43.2 and 43.3). Note that the example of squamous cell carcinoma in Fig. 43.3 lies rostral to a lobulated focal fibrous hyperplasia lesion with intact epithelium.

• **FIG. 43.1** (**A**) Histologically low-grade, biologically high-grade fibrosarcoma of the mandible in a 5-year-old golden retriever. (**B**) Corresponding radiograph shows geographic bone resorption. (**C**) Corresponding photomicrograph shows well-differentiated fibroblasts in a fascicular pattern (hematoxylin and eosin, ×100 and ×250).

• **FIG. 43.2** Canine acanthomatous ameloblastoma of the mandible.

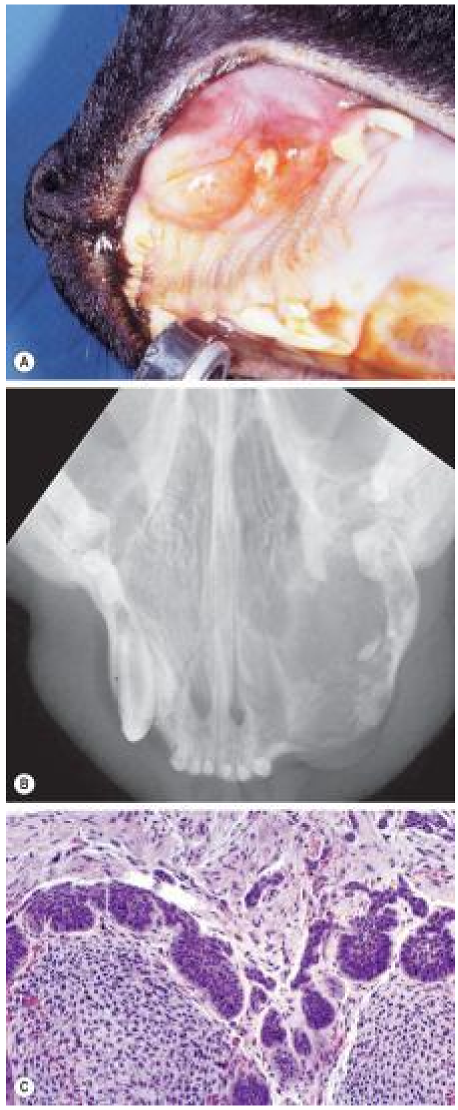
• FIG. 43.3 Squamous cell carcinoma of the incisive bone and rostral maxilla, and focal fibrous hyperplasia on the buccal aspect of the canine tooth. Source: (From Verstraete FJM. Dental pathology and microbiology. In: Slatter DH, ed. *Textbook of Small Animal Surgery*. 2nd ed. Philadelphia: W.B. Saunders; 1993;2316–2326.)

A biopsy of the lesion showed a bland spindle cell proliferation with a modest collagenous background (Fig. 43.1C). The pattern was predominantly haphazard, although some fascicular areas were seen. Few inflammatory cells were present. Spindle cell nuclei were uniform and no mitotic figures were found. A microscopic diagnosis of spindle cell proliferation, consistent with either fibrous hyperplasia or low-grade fibrosarcoma, was made. Clinical-pathologic correlation, including breed, location (mandible), and tumor size, however, favored low-grade fibrosarcoma.[1,2]

As a group, gingival fibrous connective lesions are among the most common tumors in dogs (Table 43.1).[3,4] The histopathology of well-differentiated spindle cell lesions can be particularly deceptive, a feature characteristic of both animal and human tumors. Their bland microscopy can often belie their clinical behavior. Clinically aggressive low-grade fibrosarcomas and fibromatoses can be difficult to separate from some cellular fibrous hyperplasias, making clinical correlation critical.[1] In this particular case, an important aspect of the clinical-pathologic correlation was the awareness that this variant of fibrosarcoma is known to occur in large-breed dogs (mostly golden retrievers).[1] Clinically, this tumor presents as a rapidly enlarging swelling of the jaw, covered by intact epithelium, contrary to the typical oral fibrosarcoma. The histopathological findings of well-differentiated spindle cells and rare or no mitotic figures suggest a fibroma or a well-differentiated fibrosarcoma, which is in contrast with the tumor's rapid growth and invasion. Generally, canine oral fibrosarcomas have a high recurrence rate and a significant metastatic potential.[5] Treatment should be dictated more by biologic behavior than microscopy in these cases. Both surgery and radiation have been used to treat these malignancies, although combination surgery-radiation may produce a longer survival.[2] Chemotherapy may also have role in tumor management.[6]

Case 2

A 1-year-old cat was presented with a lobular submucosal swelling of the left rostral maxilla (Fig. 43.4A, B). The lesion involved the alveolar process and was covered by intact epithelium. Radiographically, a loculated maxillary lucency was evident. The cortex was expanded and discontinuous. The duration of the lesion was unknown. There were no other physical signs or systemic problems. Because of the destructive nature of the lesion, and probable intrabony origin, the differential diagnosis included odontogenic tumor, osteosarcoma, and possibly squamous cell carcinoma. Although the surface of the lesion was intact, squamous cell carcinoma was included because of the destructive nature of the lesion. Typical for this species is that the extent of bone involvement is often much greater than is anticipated from the clinical appearance of the lesion.[7] Also, surface ulceration with squamous cell carcinoma in cats may be very discrete; however, a squamous cell carcinoma in a 1-year-old cat would be most unusual.[7,8]



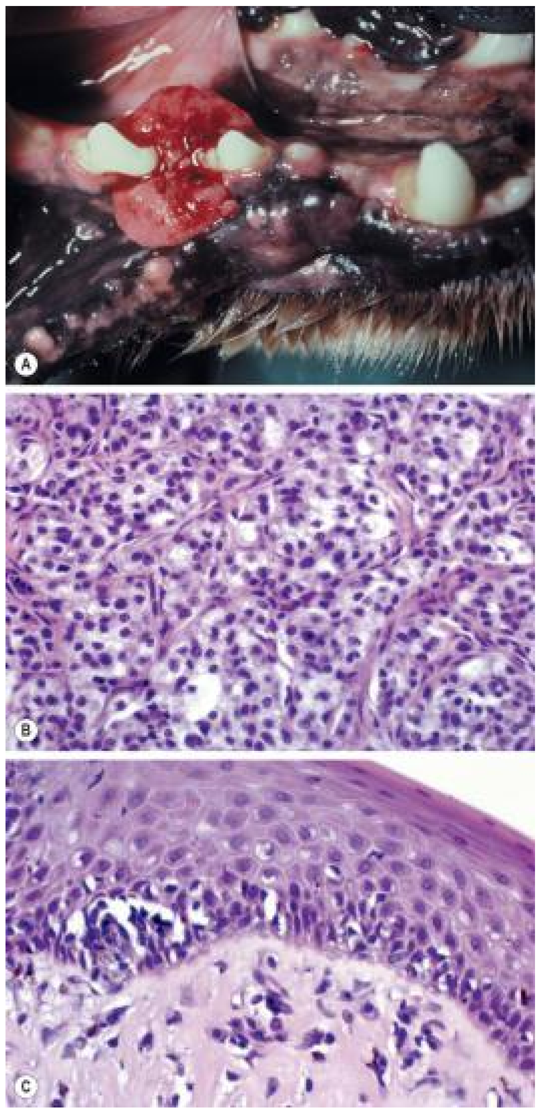
• **FIG. 43.4** **(A)** Feline inductive odontogenic tumor in a 1-year-old cat. **(B)** Corresponding radiograph (see text). **(C)** Surgical specimen shows "budding" odontogenic epithelium surrounding primitive-appearing connective tissue. Note supporting connective tissue (no inductive change) above (hematoxylin and eosin, ×100). Source: (A and B from Verstraete FJM. *Self-Assessment Color Review of Veterinary Dentistry*. Ames: Iowa State University Press; 1999;67.)

On biopsy, a poorly circumscribed, predominantly epithelial odontogenic lesion was evident (Fig. 43.4C). Nests, strands, and a reticulum of odontogenic epithelium with peripheral palisades surrounded focal areas of myxoid connective tissue. A diagnosis of feline inductive odontogenic tumor was made.[9] Ameloblastoma was ruled out because of the prominent myxoid component of the lesion.[10] This diagnosis correlated well with the reported age of the patient, location, and clinical presentation.

The feline inductive odontogenic tumor is a relatively rare mixed epithelial/mesenchymal odontogenic tumor, which is unique to cats under the age of 3 years. Expression of the proliferation marker, Ki-67, has been reported to be extremely low in tissue specimens of this tumor, supporting the benign microscopy.[11] Interestingly, this feline tumor has some resemblance to ameloblastic fibroma in humans. However, the biologic behavior of feline inductive tumor, through its capacity for local infiltration, appears to be more aggressive than the ameloblastic fibroma.[9,12]

Case 3

A 12-year-old Lhasa apso was presented with a gingival mass associated with the right mandibular second and third premolar teeth (Fig. 43.5A). The lesion, which was the same color as the surrounding tissue, bled upon physical examination. Only slight erosion of the subjacent alveolar bone was noted on radiographic examination. Based on apparent origin from gingival soft tissues, clinical differential diagnosis included peripheral (ossifying) fibroma, canine acanthomatous ameloblastoma, squamous cell carcinoma, and other nonodontogenic tumor types.



• **FIG. 43.5** (**A**) Melanoma of the body of the mandible in a 12-year-old dog. (**B**) Corresponding microphotograph shows nested polygonal tumor cells in an area in which no melanin is apparent (hematoxylin and eosin, ×250). (**C**) High-magnification photomicrograph illustrates lateral tumor spread at the epithelial-connective tissue interface in both a nested and single cell pattern (hematoxylin and eosin, ×250).

Biopsy showed submucosa expanded by a dense cellular proliferation of polygonal cells (Fig. 43.5B). Most of the tumor cells had clear cytoplasm, and a few contained melanin. A diagnosis of melanoma was made. Immunohistochemistry using antibodies against Melan A, PLN2, TRP-1, and PRP-2 could be used for histologic confirmation of either melanotic or amelanotic melanomas.[13] Antibodies against HMB-45, MCAM, and anti-S-100 protein are less desirable due low sensitivity or low specificity.[14]

This relatively common oral malignancy of dogs has a poor prognosis. It is seen in older animals, and the gingiva is the most common intraoral site.[15] Poodles have the greatest risk of developing oral melanomas.[16] Histologically, a number of different patterns can be seen (spindle, nested, clear cell, amelanotic). Of note is the occasional appearance of lateral spread at the epithelial connective tissue interface (Fig. 43.5C). In these cases, wide excision is necessary to reduce the risk of recurrence. There are some interesting comparisons between animal and human oral melanomas, even suggesting that dog melanomas may be used as a model for human disease.[16,17] Like dogs, human oral melanomas are typically seen in adults and have a strong predilection for the gingiva and palate. Also, some dog lesions may exhibit a preinvasive lateral spread followed by a vertical growth phase as is typical of many human oral tumors. In both dogs and humans oral melanomas have a much poorer prognosis than do cutaneous lesions.

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CHAPTER 44

Clinical behavior of nonodontogenic tumors

Tracy Gieger, Margaret C. McEntee

General considerations for all tumor types

Incidence

Dogs

In a study evaluating the annual incidence rate of oral tumors in dogs (conducted on insured dogs in the United Kingdom, 1997–98), 112 of 100,000 dogs were affected with oropharyngeal tumors.[1] Specific tumor types included odontogenic tumors ("epulis") (47/100,000 dogs/year), malignant melanoma (MM; 13/100,000 dogs/year), and "other" (52/100,000 dogs/year).[1] In a large case series of dogs with oropharyngeal cancer, MM was the most common tumor, followed by squamous cell carcinoma (SCC) and fibrosarcoma (FS).[2]

Cats

In a retrospective study of feline neoplasms, tumors of the oral cavity represented the third most common site of tumor involvement, after the hemolymphatic system and skin, and 68% of the oral tumors were malignant.[3] In a 10-year survey of tumors in cats, oral tumors represented 10% of all tumors; 89% of the oral tumors were malignant.[4]

Signalment

In an epidemiologic study of oral and pharyngeal tumors in dogs from 1951 to 1962, the only breed at risk was the cocker spaniel, with a significantly higher rate of MM.[5] There was a predominance of males among the dogs with MM (80%) and tonsillar SCC (74%).[5] The specific rate for all types of oral cancer was 41 of 10,000 for males and 24 of 10,000 for females. The average age for all dogs with oropharyngeal tumors was 9.8 years; the oldest mean age was in dogs with MM (10.7 years) and the youngest mean age was in dogs with FS (7.9 years).[5]

In a large series of dogs with oropharyngeal cancer, German shorthaired pointers, Weimaraners, golden retrievers, boxers, and cocker spaniels had a significantly higher risk of developing oral tumors, and dachshunds and beagles had a significantly lower risk.[2] In another study of 361 dogs with malignant oral and pharyngeal tumors, there were 41 different breeds represented, but mixed-breed dogs, cocker spaniels, poodles, and German shepherd dogs accounted for 60% of the cases but only 44% of the hospital population.[6] Smaller breeds tended to develop MM and tonsillar SCC, while the larger breed dogs had more FS and nontonsillar SCC.[6]

Etiology and risk factors

The apparent prevalence of tonsillar SCC in dogs that live in urban industrialized regions as opposed to rural environments may be due to increased exposure to environmental carcinogens. In a study of dogs that were presented to a veterinary hospital in a rural area, there were significantly fewer tonsillar SCC diagnosed than expected.[7]

Canine oral papillomavirus is known to be involved in the development of oral papillomas in dogs and it is thought that there may be progression of viral papillomas to carcinomas in dogs.[8] Papillomavirus has been identified in biopsy samples from dogs with oral SCC.[9] In an investigation of 20 oral SCC in cats, a papillomavirus was detected in one cat, which does not support a causal association between viral infection and development of feline oral SCC.[10]

Cyclooxygenase (COX)-1 and COX-2 overexpression has been demonstrated in both feline[11] and canine[12] oral SCC, suggesting that it may have a role in the pathogenesis of this disease.

There is a higher prevalence of MM in certain purebred dogs that supports the supposition that there is a genetic basis for the development of this tumor. It appears that alterations of tumor suppressor genes and proto-oncogenes with resultant disruption of cell cycle control contributes to both the development and progression of MM in dogs.[13]

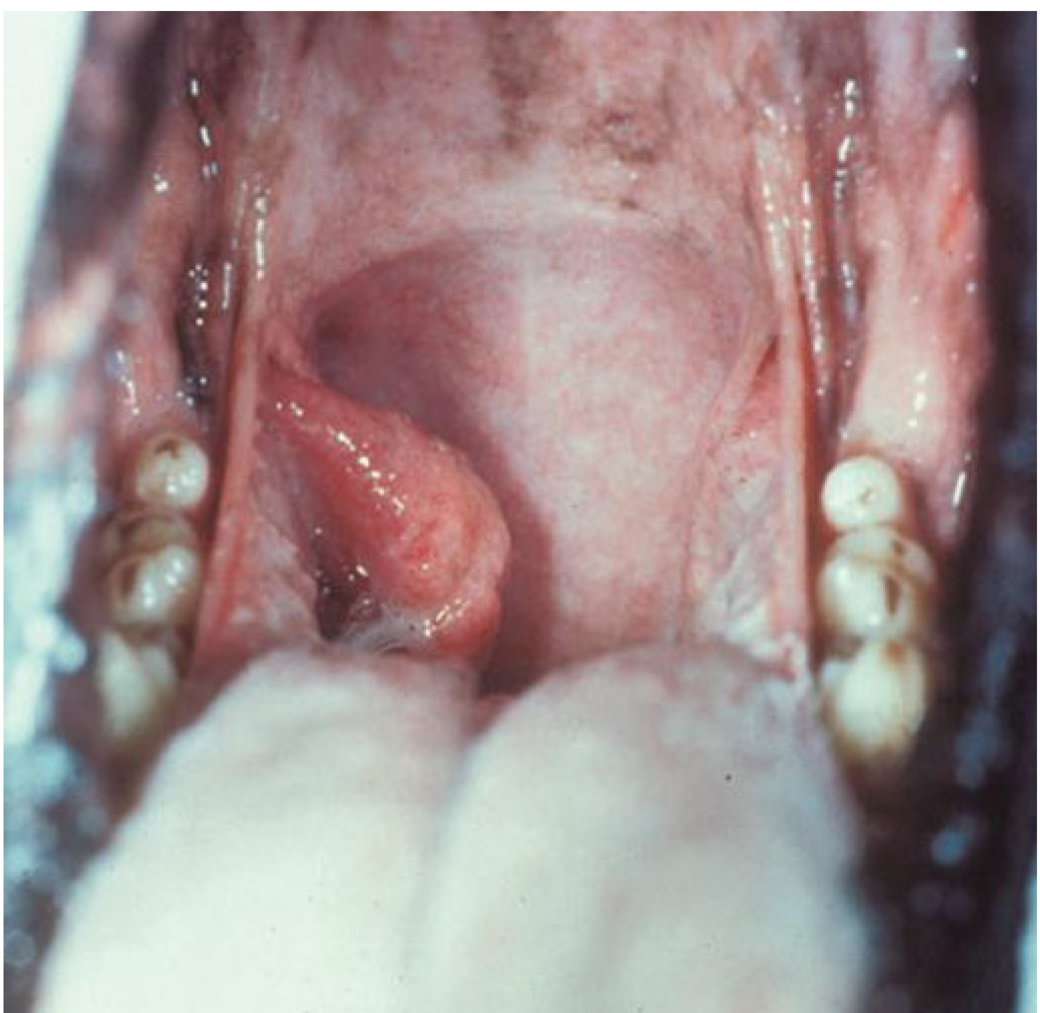
Oral osteosarcoma (OS) has been reported to develop secondary to exposure to ionizing radiation, a known carcinogen.[14,15]

A study in cats with oral SCC identified some environmental and lifestyle risk factors.[16] Cats that wear flea collars had 5 times increased risk of developing oral SCC. Other significant associations included canned food diet (3.6 times increased risk), canned tuna fish diet (>4-fold increase), and exposure to household environmental tobacco smoke (2-fold increase, not significant). Another study further supported the relationship between oral SCC and exposure to household environmental tobacco smoke and furthermore implicated p53 as a potential site for carcinogen-related mutation, with cats exposed to smoke 4.5 times more likely to overexpress p53 based on evaluation of tumor biopsy samples.[17]

Squamous cell carcinoma

In a survey of dogs with oropharyngeal tumors, SCC was the second most frequently diagnosed oral tumor (25%) (Fig. 44.1), and sites of involvement included gingiva, tonsil, oral mucosa, lip, and palate.[2] In another study of dogs with oropharyngeal tumors, 42% were SCC, with most occurring in the tonsils or gingiva.[18] Other sites of involvement included the palate, pharynx, and tongue. The mean age of dogs with gingival SCC was 8.4 years; no sex or breed predilection was noted. The tumors were described as typically reddened, friable, and vascular. The clinical history frequently included tooth extraction with subsequent tumor growth at the extraction site. Of the 22 dogs with gingival SCC, three had ipsilateral mandibular lymphadenopathy suggestive of metastasis and one had pulmonary metastasis.


A



• FIG. 44.1 (**A**) Gingival squamous cell carcinoma affecting the rostral mandible in a dog, which was treated by bilateral rostral mandibulectomy. (**B**) Tonsillar squamous cell carcinoma in a dog.

In a survey of malignant oral and pharyngeal tumors in dogs, the most common tumor was SCC (40%); approximately half were nontonsillar in origin.[6] The median age for dogs with nontonsillar SCC was 9 years (range, 1.2–14 years). One of the 35 dogs that had thoracic radiographs had evidence of pulmonary metastasis. Skull radiographs made in 27 dogs showed evidence of bone invasion in 21 dogs, primarily osteolysis. Of 11 dogs with nontonsillar SCC necropsied, five had regional lymph node (LN) metastasis and four had distant metastasis. The metastatic rate for rostral oral SCC is low and occurs late in the disease course, whereas a high metastatic rate is encountered for tumors in the caudal oral cavity, most notably tonsillar SCC.

Mandibular/maxillary squamous cell carcinoma in dogs

Surgical excision is typically the treatment of choice when deemed feasible based on both physical examination and imaging. In one study, in 21 dogs that were treated with surgery, 94% were alive at 1 year. Tumor location and clinical stage did not affect prognosis.[19] In a report of 24 dogs that underwent partial mandibulectomy for SCC, the median disease-free interval (DFI) was 26 months, with a 91% 1-year survival.[20] Two of the 24 dogs (8%) had local recurrence and metastasis, with survival times of 6 and 18 months. One dog developed only metastasis and had a 33-month survival for an overall metastatic rate of 12.5%. As part of a larger study of dogs undergoing either maxillectomy or mandibulectomy, 19 dogs with oral SCC had a local recurrence rate of 5% and metastatic rate of 11%, and the 1-year survival was 84%.[21]

Radiation therapy (RT) is the treatment of choice for nonresectable canine oral SCC. A retrospective study of dogs with nontonsillar SCC evaluated prognostic factors for survival following orthovoltage RT.[22] Dogs with rostral tumors had significantly longer mean survival (28.1 months) than did dogs with caudal tumors (10 months) or dogs with tumors that extended from rostral to caudal (1.7 months). The mean survival for dogs 6 years old or younger was 39 months versus 9.8 months for dogs older than 6 years. Local tumor recurrence had a negative effect on mean survival, with a survival of 7.1 months in dogs with recurrence versus 28.8 months in dogs without recurrence. In another study of dogs that received RT for oral SCC, the median DFI was 365 days and the median disease-free survival (DFS) was 450 days.[23] The median DFI and DFS were significantly shorter in dogs older than 9 years (210 days and 315 days, respectively) compared with dogs younger than 9 years (470 days and 1080 days, respectively). Progression-free survival (PFS) time and rate were reported for dogs with oral SCC treated with cobalt-60 (^{60}Co) photons.[24] Mean PFS time by stage was 68 months for T1 (<20 mm maximum diameter), 47.7 months for T2 (20–40 mm diameter), and 25.4 months for T3 (>40 mm diameter). Overall, the mean and median PFS times were 51.3 and 36 months, respectively.

Piroxicam has been used for the treatment of dogs with oral SCC and may be useful as an adjunct to RT or if the patient is not a candidate for curative-intent surgery.[25] There has been documented expression of COX-2 in oral SCC in dogs, supporting the use of COX-2 inhibitors therapeutically.[26] The overall response rate was 18% in a group of 17 dogs with measurable disease treated with piroxicam at a dose of 0.3 mg/kg orally once daily.[25] Five dogs (29.4%) had stable disease (SD), with a median time to failure of 102 days (range, 59–858 days).

Chemotherapy has not been uniformly successful in the treatment of canine oral SCC and is not routinely recommended. Cisplatin in combination with piroxicam has been used in a phase I/II trial, with five of nine dogs with oral SCC having a response to therapy; however, renal toxicity precluded routine use of this protocol.[27]

Lingual/sublingual squamous cell carcinoma in dogs

The most common tumor identified in a retrospective study of 57 dogs with tongue tumors was SCC (N = 21; 37%).[28] The tumors typically exhibited bilaterally symmetrical involvement (62%), diffusely infiltrated the full thickness of the tongue (52%), and involved the rostral two-thirds of the tongue (86%). Three of the 21 dogs had regional LN metastasis. Five of the 21 dogs did not receive any treatment and were euthanized within 1 month. Survival times in dogs treated with RT alone or combination therapy ranged from 0.5 to 26 months (median, 4 months). In a study of 1196 dogs with lingual lesions, 54% had neoplasia, with SCC and MM occurring most frequently.[29] The median age of dogs with lingual SCC was 9 years (range, 4–12 years). Females of all breeds and poodles, Labrador retrievers, and Samoyeds were more likely to have SCC than other demographics. In a review of 10 dogs with lingual SCC, three had a white coat color.[30] The most common presenting signs were excessive drooling and oral bleeding. Eight of the 10 dogs received treatment. All eight dogs had surgery with removal of 40%–60% of the tongue, three received mitoxantrone chemotherapy, and two received RT. The metastatic rate was 38%. The prognosis for survival was poor in dogs with high-grade tumors (N = 4; characterized by lack of keratinization, poor differentiation, high mitotic rate, and local invasion); only one of four dogs lived longer than 6 months.

A good functional outcome has been associated with extensive glossectomy in dogs.[31] In a study of 97 dogs treated surgically for lingual tumors, 32% were SCC and 30% were MM. Surgical procedures included marginal excision, subtotal glossectomy, or near-total glossectomy.[32] Surgical complications were rare, but 28% of dogs had tumor recurrence and 19% were known to have developed metastatic disease. The median survival time (MST) for dogs with SCC was 216 days, and for MM, it was 241 days. Dogs with tumors <2 cm had longer survival times, indicating the importance of early detection of oral tumors.

Tonsillar squamous cell carcinoma in dogs

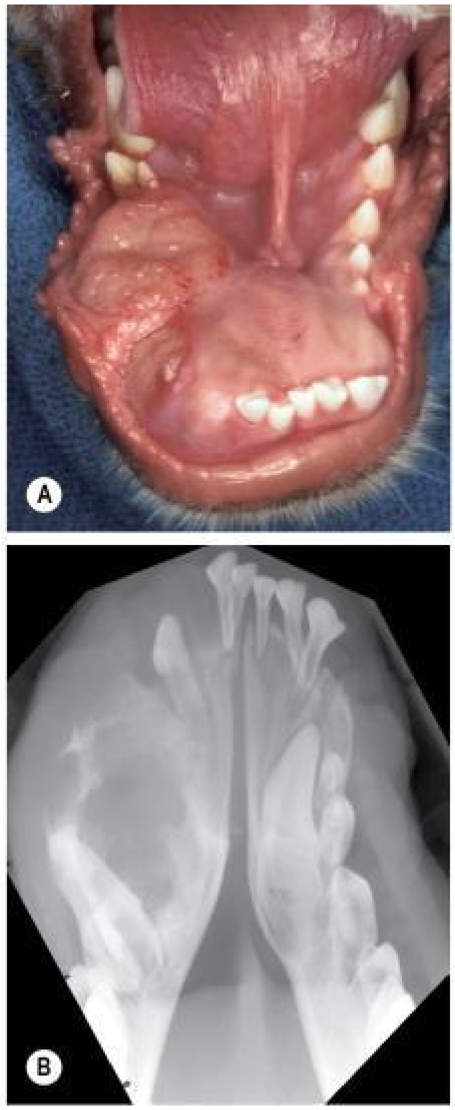
Tonsillar SCC (Fig. 44.1B) has a higher rate of metastasis than gingival tumors do, presumably due to the fact that tonsils are richly supplied with efferent lymphatics that drain to the ipsilateral as well as contralateral mandibular and retropharyngeal LN. Dogs with tonsillar SCC may have enlarged (metastatic) regional LN and silent primary tumors; therefore, in a dog with enlarged mandibular LN with cytology consistent with SCC metastasis, an anesthetized oral examination is indicated to carefully examine and/or biopsy the tonsils.[19] In one study, 28 of 29 dogs (97%) had metastasis to the mandibular LN (ipsilateral and/or contralateral) and/or retropharyngeal LN.[19] Metastasis to the thyroid gland was detected in eight dogs. Seventeen of 29 dogs were necropsied, and other sites of metastasis included lung, liver, spleen, pericardium and heart, ribs, kidney, cranial mediastinal, and pancreatic LN. In 80 dogs (as part of a larger study of dogs with oral tumors) with tonsillar SCC, the median age was 10 years (range, 2.5–17 years).[6] Of the 24 dogs that had thoracic radiographs, 2 (8%) had pulmonary metastasis. Of 48 dogs necropsied, 5 had bilateral tonsillar tumors, 17 had regional LN metastasis, and 20 had distant metastasis.

A retrospective study of canine tonsillar SCC documented the response to surgery and RT.[33] Eight dogs were irradiated with ^{60}Co photons. Survival time ranged from 44 to 631 days (median, 110 days). None of the eight dogs had metastasis past the regional LN at initial presentation, but six of seven dogs evaluated ultimately developed distant metastasis.

Another study evaluated the effect of adjuvant chemotherapy versus combined chemotherapy (doxorubicin and cisplatin) and RT in 22 dogs with advanced stage tonsillar carcinoma.[34] The overall response in 16 dogs treated with adjuvant chemotherapy was poor, with survival times ranging from 30 to 180 days (median, 105 days). The range in survival time for 6 dogs treated with combination chemotherapy and RT was 90–665 days (median, 270 days), and five had a complete response to therapy (CR; no evidence of remaining disease after treatment). Another publication documented treatment including surgical excision followed by coarse fractionated RT (9 Gy once a week for 4 weeks) and carboplatin (intravenous, 300 mg/m^2) in four dogs or carboplatin alone in one dog with tonsillar SCC, resulting in an MST of 211 days, with two dogs alive at 826 and 1628 days.[35]

Papillary squamous cell carcinoma of young dogs

Papillary SCC that may occur in young as well as older dogs may be associated with papillomavirus.[36–39] These tumors typically appear locally aggressive with underlying bone lysis (Fig. 44.2). There have been no reports of metastasis in dogs with papillary SCC. The clinical, histologic, and computed tomography (CT) findings associated with papillary SCC have been described.[39] In that study, the most common location was at the rostral maxilla. Invasion of underlying bone and lymphadenopathy were evident on CT imaging in most dogs. No evidence of metastasis was found on mandibular LN cytology and thoracic radiography. Surgical excision with 1–2-cm margins was complete in all cases, with a mean tumor-free interval of 12.1 months.[39]



• FIG. 44.2 Papillary squamous cell carcinoma in a 5-month-old dog. (**A**) Clinical appearance. (**B**) Radiograph illustrating extensive bony involvement.

Mandibular/maxillary and lingual/sublingual squamous cell carcinoma in cats

In two surveys of oropharyngeal tumors in cats, the most frequently diagnosed tumor was SCC, representing 61%–64% of the oral tumors (Fig. 44.3).[2,4] In a review of 58 cats with tumors involving bone, the most common diagnosis was SCC, including 12 mandibular and 12 maxillary SCC.[40] Radiographically, all cats had bone lysis, and most had new bone formation. In another report of 52 cats with oral SCC, 73% had marked osteolysis on radiographs, with minimal new bone formation, and 46% had more extensive disease based on radiographs than physical examination.[41]

• **FIG. 44.3** (**A**) Squamous cell carcinoma of the maxilla in a cat, clinically presenting as a discrete swelling of the maxilla and superficial ulceration overlying the canine tooth. (**B**) Radiograph of a squamous cell carcinoma of the mandible in a cat illustrating the extensive bony involvement. (**C**) Sublingual squamous cell carcinoma in a cat.

There may not be an appreciable difference in clinical signs at presentation in cats with malignant mandibular tumors as opposed to cats with benign lesions, including dental disease and osteomyelitis. In a study of cats with mandibular swellings, only 50% had a tumor, and osteomyelitis could not be differentiated from cancer based on radiographic appearance.[42] In a study of 18 cats with oral SCC (sublingual/lingual (N = 7), maxilla (N = 5), buccal mucosa (N = 4), mandible (N = 4), pharyngeal (N = 2), soft palate mucosa (N = 1), and lip (N=1), common CT imaging features included marked heterogeneous contrast enhancement, osteolysis, and extension of maxillary masses into the orbit.[43]

In cats with tongue tumors, careful evaluation of the oral cavity with examination under the tongue, as well as palpation of the tongue, will provide information on the location and extent of disease (Fig. 42.3C). Imaging modalities that show soft-tissue changes (ultrasound, CT, or magnetic resonance imaging) are indicated in these cases (see Chapter 6).

Oral SCC in cats is often advanced at presentation, and the prognosis is generally poor despite therapy. Death is usually due to local disease progression and euthanasia. Palliative measures including nonsteroidal antiinflammatory drugs (NSAIDs) and feeding tubes can provide short-term supportive care, but definitive therapy (surgery and RT) is typically unsuccessful in effecting a durable response.

Surgical excision may be indicated in some cases of mandibular or maxillary SCC, especially if lesions are well defined as assessed by physical examination and imaging. Five cats with mandibular SCC that underwent mandibulectomy had an MST of 6 months (range, 4–12 months).[44] The MST for seven cats that underwent surgical excision of oral SCC was 1.5 months.[41] In a study of 42 cats with oral tumors treated with mandibulectomy with a subset also receiving RT and/or chemotherapy, cats with SCC (N = 21) had a significantly shorter survival time than did cats with FS or OS. Cats with mandibular SCC had a disease progression-free and survival rate at 1 year of 51% and 43% and at 2 years of 34% and 43%, respectively.[45]

In a series of 54 cats with oral SCC that received only NSAIDs, antibiotics, steroids, or no treatment, the median survival time was 44 days, with a 1-year survival of 10%. Longer survival occurred in cats that received NSAIDs.[46] Toceranib phosphate is a targeted therapy that may play a role in the management of oral SCC in cats. In one study, use of this drug improved survival times as compared with cats not receiving the drug; however, use of NSAIDs was also allowed in the study. Use of NSAIDs concurrently was associated with a longer survival time. In 23 cats that received toceranib phosphate as part of their treatment, the MST was 123 days as compared with 45 days in cats not treated with this drug.[47]

The use of chemotherapy alone has not shown promise for feline oral SCC. A phase I clinical trial of intravenous carboplatin in 59 cats included 16 cats with oral SCC, of which there were no CR.[48] Eighteen cats with nonresectable oral SCC, including eight cats with lingual tumors, were treated with a lipophilic cisplatin analog; the MST was 60 days and acute toxicity limited clinical utility.[49] Five cats with nonresectable oral SCC were treated every 3 weeks with a combination of doxorubicin and cyclophosphamide; one cat had a partial response (PR) to therapy and survived for 90 days.[50]

RT may be useful in some cases of oral SCC and has been combined with chemotherapy in several studies. Eleven cats with oral SCC (four lingual and seven mandibular or maxillary) were treated with a combination of RT and intravenous mitoxantrone.[51] Of the 11 cats, there were eight CR and one PR. The remission time for the cats with a CR ranged from 28 to 485 days, with a median of 170 days. The PR duration was 60 days. Eight cats with advanced nonresectable oral SCC (sublingual in four, mandibular in four, and maxillary in two) were treated with a combination of low-dose gemcitabine and palliative-intent RT with two CR and four PR, a median duration of remission of 43 days, and an MST of 112 days.[52] Nine cats (four lingual, three mandibular, one tonsillar, and one buccal) treated with an "accelerated" RT protocol (14 3.5 Gy fractions given over 9 days) had an MST of 86 days.[53] Notably, the MST of six cats that had a PR was 60 days and for three cats that had a CR was 298 days. Seven cats with advanced oral SCC were treated with RT; only four of the seven cats completed treatment and survival ranged from 42 to 97 days.[54] Although RT may not prolong survival, it may help to improve the quality of life. In one study of 54 cats treated with a palliative-intent RT protocol (24–40 Gy total dose in three to four weekly fractions), 65% of cat owners reported improved quality of life.[55] The MST of cats treated with RT alone was 113 days and was 80 days in cats also receiving chemotherapy. Cats with sublingual tumors had an MST of 135 days, and it was 80 days for cats with mandibular tumors.

Longer responses to therapy can be seen with combination therapy including wide excision and RT. In a report of seven cats treated with mandibulectomy and postoperative RT, the MST was 14 months.[56] Six of the seven cats were euthanized due to recurrence with a median DFI of 11 months.

In cats with superficial SCC, especially for superficial tongue tumors, Strontium-90 plesiotherapy may be useful. In a published study of two cats treated with this modality, one of the cats survived for over 5 years.[57]

Tonsillar squamous cell carcinoma in cats

Tonsillar SCC is uncommon in cats. Of four cats with tonsillar SCC that underwent partial or complete tumor removal, the survival times were 2–14 weeks and all were euthanized due to local recurrence and metastasis.[58] A study evaluating the use of an accelerated RT protocol (N = 31 cats; 14 3.5 Gy fractions of RT were given over 9 days) with use of carboplatin as a radiosensitizer documented an overall MST of 163 days. In the four cats of this study with tonsillar SCC, the MST had not been reached, indicating long-term survival in this small group of cats.[59]

Malignant melanoma

Malignant melanoma in dogs

MM is the most common or second most common malignant oral tumor in dogs (Fig. 44.4A).[6,18] In a study of 338 oral MM cases, Anatolian sheepdog, cocker spaniel, Gordon setter, mixed breed dogs, chow, golden retriever, Pekingese/poodle cross, and miniature poodle breeds were significantly overrepresented, whereas boxer and German shepherd dogs were underrepresented.[60] Melanomas are typically firm and grayish or brownish black (but can also lack pigment, termed "amelanotic melanoma"), rapidly enlarge, and develop ulceration, hemorrhage, and metastasis (to the regional LN, lungs, and/or other sites) within weeks or months after diagnosis. Males are predisposed, with a male-to-female ratio of 3:1–6:1 and mean age of 10.5–12 years. Melanoma may involve the gingiva, the labial mucosa, palate, buccal mucosa, and tongue.

• **FIG. 44.4** Malignant melanoma at the gingiva and the alveolar bone at the right mandible in a dog.

Melanomas of the lip that arise from mucous membranes typically have a better prognosis than gingival tumors do, but are more aggressive than tumors arising from haired skin. Of 32 dogs with lip MM with histologic features of malignancy, 22 died due to tumor within a year, and 10 were tumor free for at least 1 year.[61] In a report of 64 dogs with 69 histologically well-differentiated melanocytic neoplasms involving the mucous membranes of the lips and oral cavity treated with surgery alone, 61 of 64 (95%) were alive at the end of the study or died of other causes, with a mean survival of 23 months and an MST of 34 months after surgery; the remainder died of tumor-related causes.[62]

Twenty-three of 43 dogs with oral MM in one report were necropsied and 74% had regional LN metastasis and 65% had pulmonary metastasis.[18] Other sites of metastasis reported include brain, meninges, pituitary gland, tonsils, striated muscle, pleura, pericardium, heart, prostate, pancreas, adrenal glands, liver, kidney, spleen, ileum, omentum, mediastinal LN, thyroid, prostate, salivary gland, stomach, testis, and eye.[6,18] In a study of 100 dogs with oral MM, 53% had cytologic or histologic evidence of regional LN metastasis, with 70% having enlarged mandibular LN and 30% having normal sized LN.[63]

There is variable microscopic appearance of MM. There are three main categories based on microscopic features of the predominant cell type, including polygonal (epithelioid), spindle, and mixed (epithelioid and spindle) cells. The distribution by cell type in one study was 21% epithelioid, 34% spindle, and 42% mixed (in another study of 70 oral MMs, 66% were epithelioid, 7% were spindle, and 24% were mixed.[60,64] There was a difference in survival based on the mitotic index, but it was not statistically significant. The most common tumor location was the gingiva, with 53% involving either the labial mucosa or mandibular gingiva. A number of different immunohistochemical stains were used to characterize and identify oral MM, and it was concluded that Melan-A is a specific and sensitive marker for canine MM. In another study, the best prognostic model was based on nuclear atypia.[64]

A retrospective study was done to evaluate a staging system including assessment of tumor size, location, and mitotic index in comparison to the World Health Organization staging system for canine oral MM.[65] Based on this novel staging system, those with tumors smaller than 8 cm^3, tumors located on the rostral mandible or caudal maxilla, and/or tumors having a mitotic index ≤3 had a significantly longer remission duration and survival regardless of the treatment modality. Wide excision effected the longest remission and survival times, and this was independent of whether or not the surgical margins were free of tumor.

Treatment for MM may include surgical excision, RT, chemotherapy, and immunotherapy; due to the locally invasive and highly metastatic nature of this cancer, outcomes are variable.

As part of a larger study of 234 dogs treated with curative-intent surgery for oral tumors, 40 had MM.[66] Most (80%) had evidence of bony lysis on cross-sectional imaging. The local recurrence rate after surgery was 28% and the DFI was 152 days, with only 5% of dogs with MM alive at 1 year. In a study evaluating immunotherapy as an adjunct to surgery, 89 dogs with malignant oral MM were treated by surgical excision alone (N = 47) or surgery and *Corynebacterium parvum* immunotherapy (N = 42).[67] There was no difference in survival between the two treatment groups, although in dogs with advanced stage disease, there was a statistically significant difference between surgery alone and combination therapy. Dogs with stage I disease (tumor diameter <2 cm) had a significantly longer survival regardless of therapy. Stage I dogs had an MST of 511 days, compared to 160 and 168 days for stage II (tumor 2–4 cm in diameter, no other evidence of disease) and stage III (tumor >4 cm or any size with LN metastasis, no distant metastasis) dogs, respectively.

Gene therapy has been evaluated in 26 dogs with MM for the management of canine oral MM.[68] The overall response rate was 46% (12 of 26 dogs had a PR or CR), and in these dogs, a decrease in tumor size usually occurred within 10 weeks. The MST of nine dogs with stage III disease that completed the 12-week induction phase of therapy was 66 weeks, compared to an MST of 15 weeks for stage III dogs in another study treated with surgery alone.[69]

The most recent advancement in the treatment of canine MM has been the development and clinical application of a xenogeneic DNA vaccine that was Food and Drug Administration approved (conditional license) for use in dogs.[70–72] Dogs that gain the greatest benefit from xenogeneic DNA vaccination with tyrosinase family members are those with loco-regionally controlled MM.[71] For 33 dogs with stage II–III disease across the xenogeneic vaccine studies performed, the MST was 569 days.[71] In a study of 69 dogs with oral MM treated with the melanoma vaccine as part of their therapy, the MST was 455 days.[73] Eight of 13 dogs treated with measurable melanoma had a response to therapy, so use of the vaccine could be considered in dogs with nonresectable disease or when RT is not possible.

Response rates to chemotherapy alone in dogs with measurable MM are moderate. In a report of 27 dogs with MM (25 located in the oral cavity) treated with intravenous carboplatin, 12 (44%) had regional LN metastasis and seven (26%) had pulmonary metastasis.[74] There was an overall response rate of 28% (1 CR and 6 PR), with 18 having SD. Eleven dogs with oral MM were treated with a combination of cisplatin and oral piroxicam, with 2 of 11 having a CR to therapy for an overall response rate of 18%.[27] A synergistic response is possible with this drug combination, but nephrotoxicity can be a limitation.

RT is important in the management of MM, especially in dogs where tumors are too large/invasive to be surgically excised. A total of 38 dogs with oral MM were treated with ^{60}Co

photons. The overall mean PFS was 18.8 months (median, 7.9 months).[24] The 1- and 3-year PFS rates were 36.4% and 20.2%, respectively. There was a significant difference in prognosis based on tumor stage with a mean (and median) PFS by stage of 38 months (T1; median, 18.8 months), 11.7 months (T2; median, 6 months), and 12 months (T3; median, 6.7 months). Coarsely fractionated (>4 Gy/fraction) RT protocols are frequently used for treatment of oral MM due to its theoretical ability to repair radiation-induced cellular damage at lower doses/fraction as compared with other cell types. Eighteen dogs with oral MM irradiated with ^{60}Co photons received 8 Gy/fraction on days 0, 7, and 21, for a total dose of 24 Gy.[75] There were nine (53%) CRs and five (29%) PRs. The MST was 7.9 months. Tumor response and survival were not significantly affected by tumor site, stage, or prior surgical excision.

In another study, 36 dogs with oral MM were treated with RT (four fractions of 9 Gy/fraction once weekly [total dose, 36 Gy]); 24 had previous surgery and 34 had macroscopic disease at the time of RT.[76] Responses included a CR in 25 dogs (69%), PR in 9 dogs (25%), and SD in 2 dogs (5.5%). Survival time ranged from 5 to 213 weeks, with an MST of 37 weeks (range, 5–213) in dogs achieving a CR and 20 weeks (range 10–38) in dogs achieving a PR. There was also a significant difference in survival based on tumor size, with an MST of 86 weeks in dogs with tumors <5 cm^3, 16 weeks in dogs with tumors 5–15 cm^3, and 20.5 weeks in dogs with tumors >15 cm^3. A retrospective study of 140 dogs with oral MM compared three different RT protocols (9 Gy × 4 fractions; 10 Gy × 3 fractions; >45 Gy, 2–4 Gy × 12–19 fractions). There was no difference in response based on RT protocol, but on multivariate analysis, rostral tumor location, lack of bone lysis on skull imaging, and microscopic tumor burden were joint predictors of time to first event and survival.[77] The overall MST was 7 months.

The efficacy of a hypofractionated RT protocol in conjunction with platinum-based chemotherapy was evaluated in 39 dogs.[78] The RT protocol was 6 Gy once weekly for six consecutive weeks using either ^{60}Co or a linear accelerator. Sixty minutes prior to each radiation treatment, the dogs received low-dose cisplatin or carboplatin as a radiation sensitizer. Fifteen percent of the dogs had local recurrence, with a median time to recurrence of 139 days. Fifty-one percent of the dogs developed metastasis at a median time of 311 days (range, 24–2163 days). The MST was 363 days. In another study that evaluated a hypofractionated RT protocol (four weekly fractions of 9 Gy) alone (13 dogs) to the primary and any LN metastases or in combination with full dose carboplatin chemotherapy (15 dogs), there was no survival benefit with the addition of chemotherapy or alteration in the number of dogs dying due to metastases.[79] In a study of 111 dogs treated with various RT protocols for oral MM, dogs with stage I disease had longer survival times than other groups did.[80]

Malignant melanoma in cats

Melanomas are uncommon in cats, with ocular and cutaneous sites more common than oral cavity.[81–84] Only 3 of 371 cats with oral tumors were diagnosed with MM (1 palate and 2 gingiva).[4] In a review of 39 cats with MM, the most common site of involvement was ocular, with only four oral tumors.[83] In another study, 29 cats were diagnosed with MM, including 19 ocular, 5 oral, and 5 dermal MM.[84] Four of the cats with oral MM were euthanized at 1–135 days (mean, 61 days) and all had metastasis.

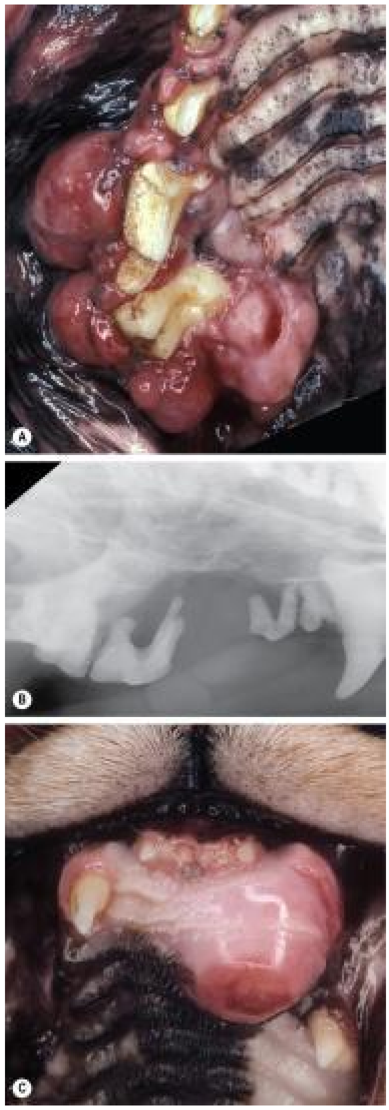
Studies documenting treatment outcomes in cats with MM are scarce. Five cats with oral MM were treated with palliative-intent RT.[85] Three of the five cats had a documented response (one

CR and two PR), but all cats were euthanized due to persistent disease. The MST was 146 days (range, 66–224 days). In a study of 30 cats with nonocular melanomas, 13 had oral cavity tumors, five of which were on the lip.[86] Seven were treated with surgery, and five had local tumor recurrence. The MST for cats with oral tumors was 120 days. The canine melanoma DNA vaccine has been safely administered to cats with a low incidence of adverse effects, but efficacy data are lacking.[87]

Fibrosarcoma

Mandibular/maxillary fibrosarcoma

Based on two surveys totaling 910 dogs with oropharyngeal tumors, the third most common tumor was FS (Fig. 44.5A), representing 18% of oral tumors.[2,6] In a report of 76 dogs with oral FS, the median age was 8 years (range, 0.5–15 years), 25% were less than 5 years of age, and all were large-breed dogs.[6] Sites of involvement for oral FS included gingiva (N = 66), hard palate (N = 5), labial mucosa (N = 3), and one each for soft palate and tongue. Of the 40 dogs that had thoracic radiographs at diagnosis, 4 (10%) had pulmonary metastasis. Of 31 dogs with gingival tumors that had skull radiographs obtained, 21 (68%) had evidence of bone invasion, primarily osteolysis (Fig. 44.5B). Twenty-six of the dogs in this study had necropsies; three had regional LN involvement and six had distant metastasis (lung in six and lung and brain in one).[6]



• **FIG. 44.5** (**A**) Fibrosarcoma of the maxilla in a dog. (**B**) Radiograph of a fibrosarcoma of the maxilla showing extensive osteolysis. (**C**) Fibrosarcoma of the left rostral maxilla and hard palate in a cat.

Histologically low-grade yet biologically high-grade FS of the mandible and maxilla is a distinct histopathologic entity that has been identified in dogs.[88] The histopathologic appearance is compatible with benign fibrous connective tissue, but the biologic behavior indicates otherwise, with 72% of dogs evaluated found to have underlying bone lysis. Additionally, 12% ultimately developed pulmonary metastasis, and 20%, regional LN metastasis. The most common breed appears to be golden retrievers, representing 52% of the dogs in one study. Age at presentation ranged from 3 to 13 years (median, 8 years).[88]

Surgery alone for oral FS results in a relatively high local recurrence rate. In a group of 14 dogs with oral FS that underwent mandibulectomy or maxillectomy, the 1-year survival rate was 50%; 36% had local recurrence and 14% had documented metastasis.[21] In a report of 19 dogs that had mandibulectomy for oral FS, the 1-year survival rate was 50% and the median DFI was 10.6 months.[20] Twelve of the 19 dogs had recurrence and/or metastasis (nine dogs had local recurrence; one dog had metastasis; and two dogs had local recurrence and metastasis).

RT often plays a role in the management of FS since many tumors are deemed nonresectable. In one study, a mean time to local tumor regrowth of 3.9 months after orthovoltage RT was found, with an MST of 6.8 months.[89] Recent reports show increasing promise for the use of RT in the treatment of oral FS. In 31 dogs with macroscopic oral soft tissue sarcoma (STS) treated with either curative intent (N = 20; median total dose, 52.5 Gy) or palliative intent (N = 11; 3 × 8 Gy or 5 × 6 Gy) RT, MSTs of 331 and 310 days, respectively, were achieved.[90]

A report on 28 dogs with oral FS treated with RT reported an overall mean and median PFS of 29.7 and 26.2 months respectively.[24] A poorer prognosis was associated with higher stage. The mean (and median) PFS by T stage was 53 months for T1 (<2 cm) (median, 45 months), 30 months for T2 (2–4 cm) (median, 31 months), and 12.6 months for T3 (>4 cm) (median, 7.1 months). The overall 1- and 3-year PFS rates were 76% and 40%, respectively.

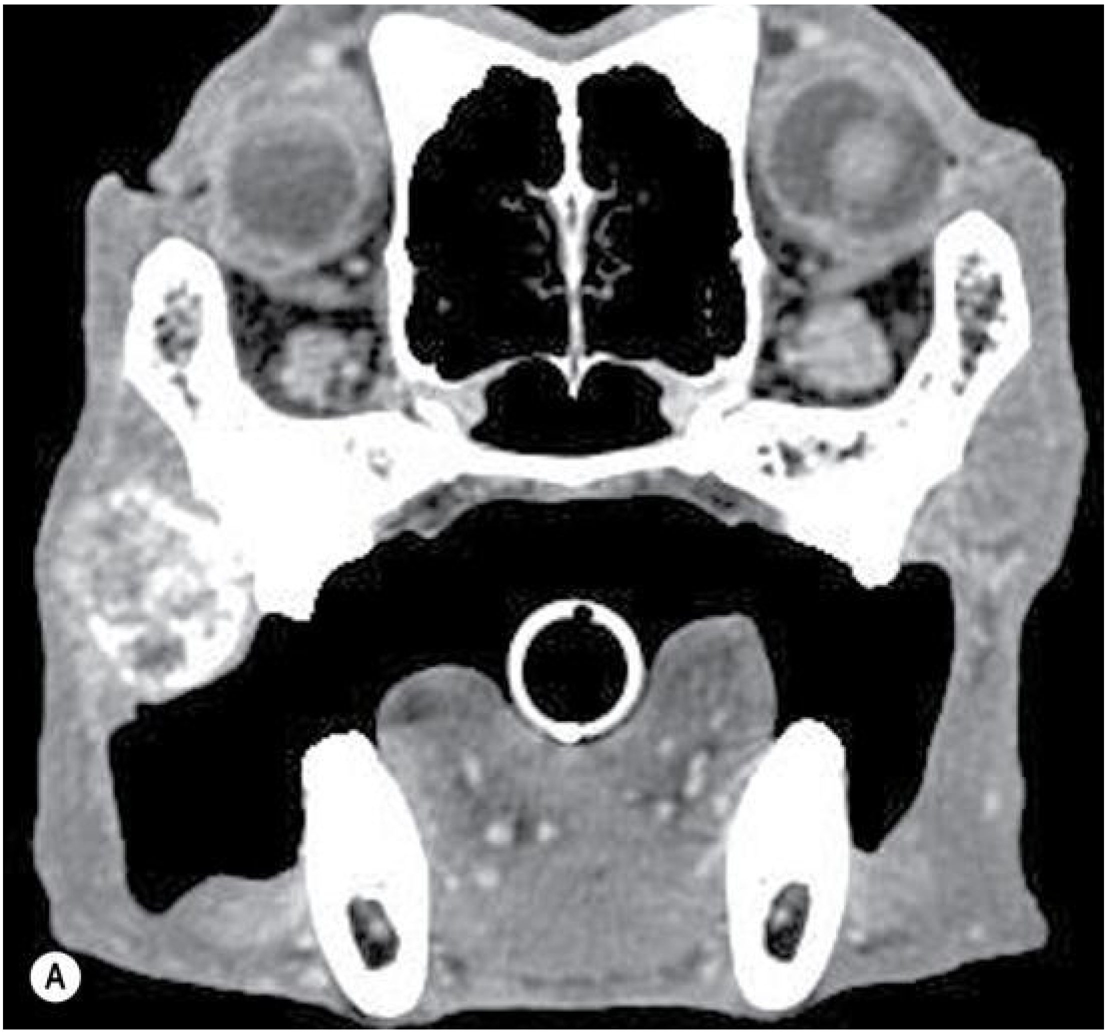
In a report of postoperative RT for STS in 35 dogs, 7 dogs had oral FS.[91] The median survival for dogs with oral STS was significantly less (1.5 years) when compared to STS located in other sites (6.2 years). A retrospective study reported outcome in a cohort of 29 dogs with oral FS treated with surgical excision with or without adjuvant RT. Twenty-one dogs were treated with surgical excision alone and eight dogs with both surgery and RT (total doses ranging from 32 to 48 Gy). The median progression-free interval was >653 days. The median survival time was 743 days. The 1- and 2-year survival rates were 87.7 and 57.8%, respectively. Seven (24.1%) dogs developed local recurrence. Seven dogs (24.1%) developed metastasis.[92] A report retrospectively evaluating 65 dogs with oral FS found that significant predictors of survival were location (dogs with maxillary and mandibular tumors had longer survival than those with palate tumors), stage (stage I did best), type of surgery (wide excision better than conservative surgery), margins, and grade (low grade did best). Significant predictors of PFS were location and radiation protocol (curative intent better than palliative intent). A combination of surgery and RT was the strongest predictor of prolonged median survival and PFS at 505 and 301 days, respectively. Dogs treated with other modalities had an MST of 220 days.[93]

In two surveys of oral tumors in cats, FS represented the second most common oral tumor, representing 13% and 22% of the oral tumors.[2,4] Age of the cats ranged from 1 to 21 years (mean, 10.3 years). The FS arose from the submucosal stroma and were accompanied by local tissue destruction and invasion of skeletal muscle and bone; no predilection site was noted (Fig. 44.5C). Six cats underwent mandibulectomy alone (N = 5) or in combination with chemotherapy (N = 1) for mandibular FS with a 1–2-year survival rate and progression-free rate of 67%.[44]

Bone tumors

Osteosarcoma

OS occurs more commonly in the appendicular than in the axial skeleton.[94–96] Of 394 dogs with bone lesions, the most common tumor was OS (47%), and 27% of the OS involved the axial skeleton, of which 14 (8%) involved the skull (Fig. 44.6A and B).[96] A retrospective study of 116 dogs with axial skeletal OS determined that the most common sites of involvement were the mandible and maxilla.[95] The majority of axial OS occurred in medium- and large-breed dogs, and most of the dogs were middle aged or older. The presence of radiographically evident pulmonary metastasis at the time of diagnosis was uncommon.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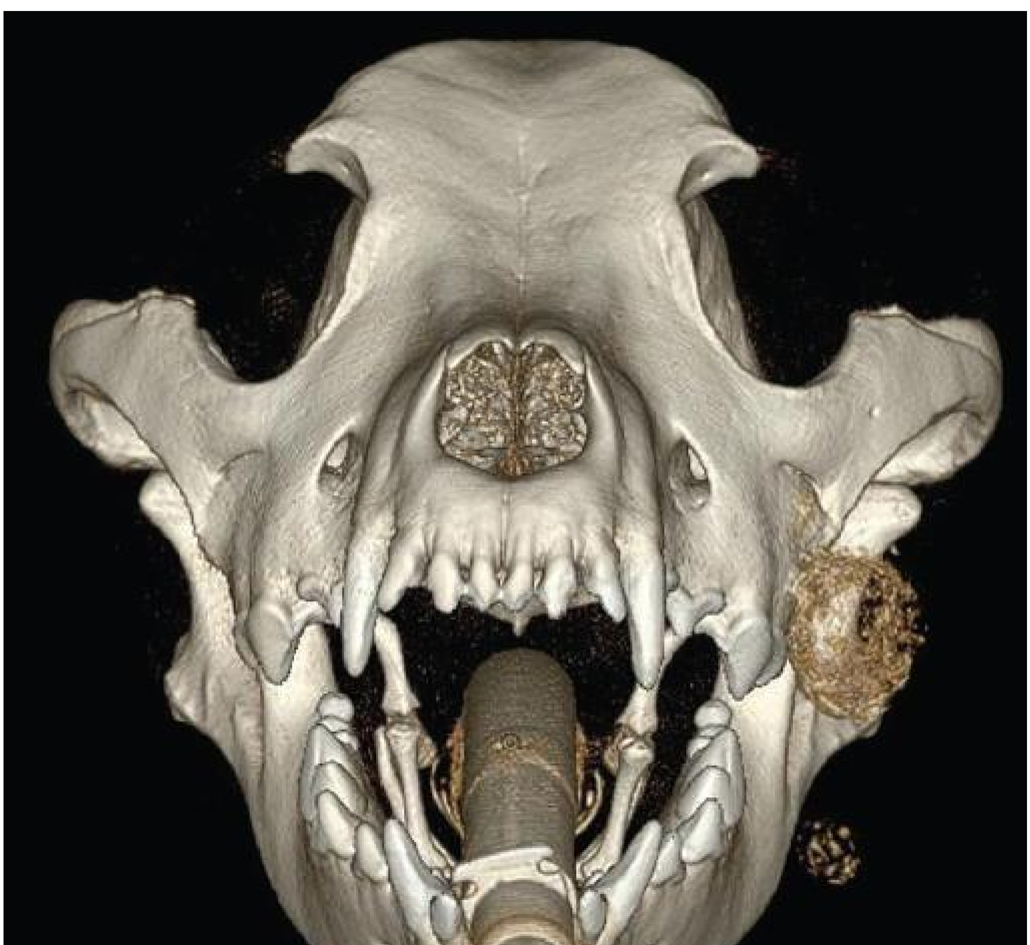


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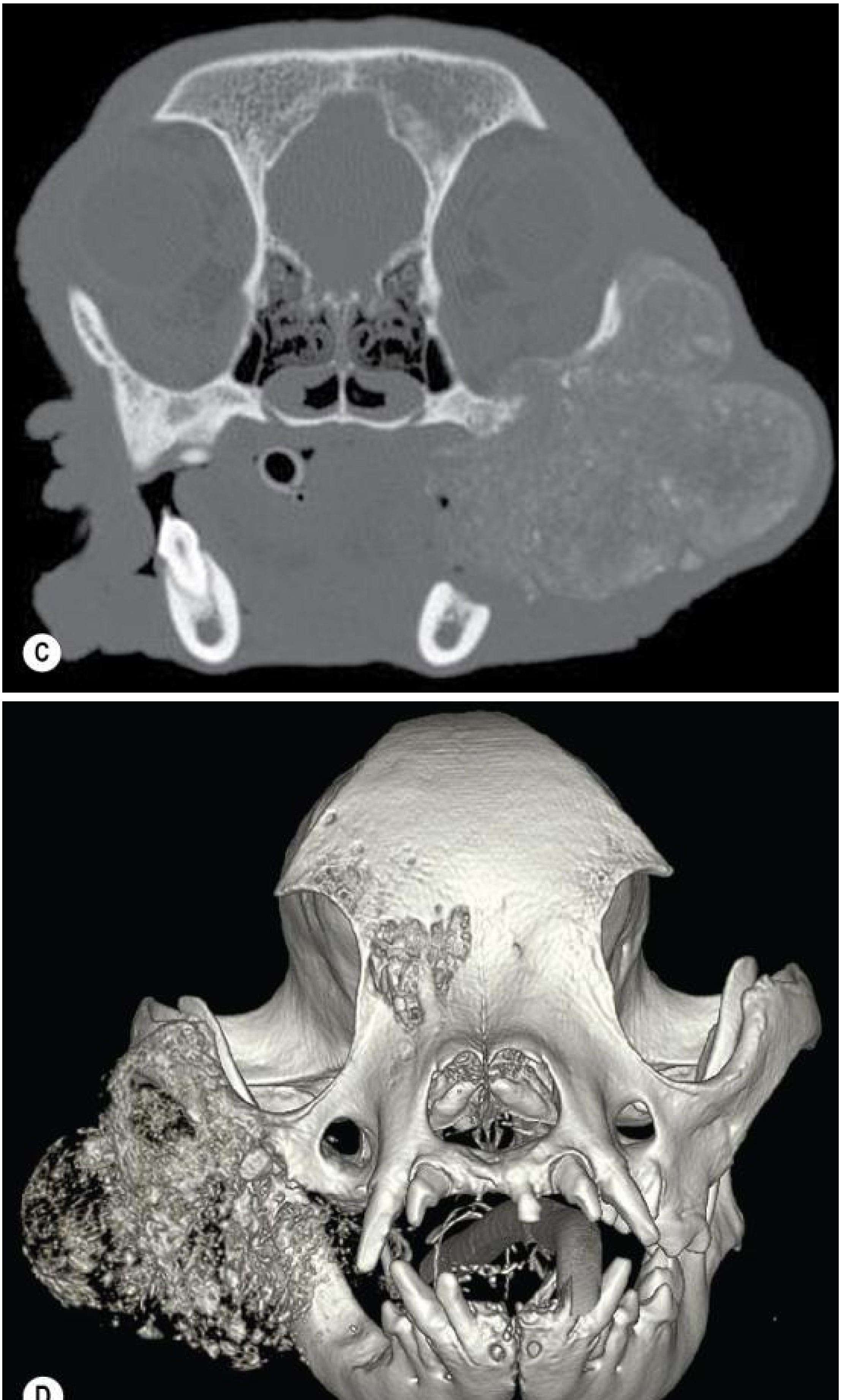

• FIG. 44.6 (**A**) Contrast-enhanced computed tomography (CT) image of an osteosarcoma of the maxilla in a dog. (**B**) Three-dimensional (3D) reconstructed image of the same CT seen in Panel A. Note that the 3D image lacks the soft tissue detail seen in the contrast-enhanced image. (**C**) CT image of multilobular osteochondrosarcoma in a dog. (**D**) Three-dimensional reconstructed image of the same CT seen in Panel C.

A retrospective study of 45 dogs with axial OS included 12 mandibular and seven maxillary tumors.[97] Favorable prognostic variables identified included tumors of the mandible, complete surgical excision, and smaller dogs. The median survival was 223 days for dogs with complete excision and 87 days for dogs with incomplete excision. The MST for 12 dogs with mandibular tumors was 165 days as compared with 105 days for all other sites combined. There was no significant difference in survival based on whether or not the dogs received cisplatin chemotherapy.

In a retrospective study over a 12-year period of 51 dogs with mandibular OS, the mean age was 9.5 years (range, 3–15 years), mean body weight was 27.8 kg (range, 6.4–53.2 kg), and the male-to-female ratio was 1:1.8.[98] Treatment included partial mandibulectomy (N = 32), partial mandibulectomy and chemotherapy (N = 10), partial mandibulectomy and RT (N = 3), partial mandibulectomy, RT, and chemotherapy (N = 4), and RT alone (N = 2). The majority of dogs that were treated with chemotherapy received intravenous cisplatin. The 1-year overall DFS (alive without evidence of local recurrence or known metastasis) rate was 48%, with a median overall DFI of 9.5 months. The overall 1-year survival rate was 59% and was 71% in dogs treated with surgery alone. The MST was 17.6 months. The local recurrence rate postoperatively was 28%, and the metastasis rate was 28%. A retrospective study of 50 dogs with mandibular OS, 21 of which received chemotherapy, demonstrated that 29 dogs (58%) developed metastatic disease.[99] The median metastasis-free interval (MFI) was 627 days, and MST was 525 days. Histological grade and use of adjuvant chemotherapy were prognostic for MFI and MST.

In a retrospective study of 22 large-breed dogs with axial skeleton OS (including four maxillary and three mandibular tumors), the metastatic rate was 46% and the MST for all dogs was 137 days.[100] Dogs treated with a definitive-intent versus a palliative-intent RT protocol experienced a significantly longer survival time of 265 days as opposed to 76 days.

Skull OS in cats is uncommon, and treatment outcomes are not widely documented. In a report of 22 cats with OS, 15 involved the appendicular skeleton and seven involved the axial skeleton, including one on the mandible, one on the maxilla, and two on the rest of the skull.[101] Radiographically, in all seven cats with axial tumors there was a significant amount of periosteal new bone formation. Of the 22 cats, only one cat with an appendicular tumor had documented pulmonary metastasis. The four cats with skull tumors were treated surgically with resultant incomplete excision; one received RT postoperatively and lived for 16 months prior to being euthanized for recurrence; and the remaining three cats with skull OS died within 3 months postoperatively due to recurrence or progression. The MST for cats with axial OS was 5.5 months.

Multilobular osteochondrosarcoma

Multilobular osteochondrosarcoma (MLO) is a relatively uncommon tumor in dogs, rare in cats, and typically involves the flat bones of the cranium.[102–104] MLO has a stippled appearance on radiographs due to the multilobular arrangement of osseous, cartilaginous tissue, or both, surrounded by mesenchymal stroma. The CT and magnetic resonance imaging appearances of

MLO have also been described and provide more detailed information on the extent of the disease for surgical planning (Fig. 44.6C and D).[105,106] In a report of 39 dogs with MLO, sites of involvement included the maxilla (N = 11), mandible (N = 14), calvarium (N = 14), and one each at various other sites.[103] The most common presenting complaint was a firm, fixed mass (54%), and other signs included swelling, pain, exophthalmos, or neurologic signs. Twenty-five dogs were treated with surgery alone and nine were treated with surgery combined with adjuvant therapy, including implantation of cisplatin-impregnated open cell polylactic acid, RT, and intravenous cisplatin. The effect of adjuvant therapy on outcome could not be ascertained. Of the dogs treated, 47% had local tumor recurrence and 56% had documented metastasis. The median time to recurrence was 797 days, median time to metastasis was 542 days, and MST was 797 days (range, 28–1670 days). Four dogs had metastasis at the time of initial presentation. Outcome was affected by histological grade (criteria included borders, size of lobules, organization, mitotic figures, cellular pleomorphism, and presence of necrosis), surgical margins, and tumor location. Dogs with grade II and III tumors had significantly shorter times to metastasis. The local recurrence rate was higher in dogs with high-grade tumors.

Other nonodontogenic tumor types

Undifferentiated malignant tumor of young dogs

Undifferentiated malignant tumor of the oral cavity is a distinct clinical entity that has been identified in young dogs, with only six cases published over a 15-year period.[107] All were less than 2 years of age, with a range of 6 to 22 months. Five of the six dogs had an ill-defined soft gray-tan, hemorrhagic mass involving the maxillary premolar and molar regions, and one dog had involvement of the soft palate. The tumors were locally aggressive and had variable extension of the tumor into the maxillary, nasal and orbital tissues, and in some, into the brain. Necropsy of five dogs revealed diffuse metastasis in four and liver metastasis in one. The most common sites of metastasis were LN, lung, liver, heart, kidney, and brain. Electron microscopy and immunohistochemistry did not reveal any characteristics that would indicate that these tumors were epithelial or mesenchymal in origin, hence the diagnosis of undifferentiated malignant tumor. Surgery was the only treatment attempted with an average survival of 22 days (range, 2–60 days).

Extramedullary plasmacytoma

Extramedullary plasmacytoma (EMP) arises outside of bone, is typically mucocutaneous in location, and occurs most commonly in the mouth (Fig. 44.7A), on the feet or trunk, and in the ears.[108–110] In a report of dogs with EMP, 23% occurred in the oral cavity and locations included the gingiva, tongue, hard palate, and oropharynx.[109] In a report of 75 dogs with mucocutaneous EMP, 17 (23%) were located in the mouth.[108] In a study of 46 dogs with cutaneous EMP, the second most common site of involvement was the lip (24%).[110] Cutaneous EMPs are typically small (mean diameter 8 mm), broad based, spherical or dome shaped, and hairless.

• **FIG. 44.7** (**A**) Extramedullary plasmacytoma of the rostral mandible in a dog. (**B**) Oral lymphoma affecting the lower lips in a dog.

In a retrospective study, 16 of 302 (5%) dogs with oral tumors had EMP, representing 29% of all EMPs over a 10-year time period.[111] Tumor location included lingual (N = 4), rostral mandible (N = 7), maxilla/hard palate (N = 3), buccal surface of upper lip (N = 1), and extensive disease (N = 1). Treatment included none, surgery, chemotherapy, and/or RT. At the time of the study conclusion, five were alive and 11 had died, with an MST of 474 days (range, 37–2906 days).

The primary mode of therapy is surgical excision, and the majority of dogs have long-term local control. The local recurrence rate as well as distant failure is low. It has been noted that gingival tumors have the highest rate of local recurrence (4 of 17 in one study), presumably due to the difficulty in obtaining wide margins, although partial maxillectomy or mandibulectomy may be successful.[109,111] Plasmacytomas are considered to be radiation sensitive, but the outcomes of patients with EMP treated with RT have not been reported.

Oral lymphoma

Epitheliotropic primary oral lymphoma (lymphoma, LSA) in dogs can involve the gingiva and/or lips at the mucocutaneous junction (Fig. 44.7B). In a study of 130 dogs with oropharyngeal tumors, six had LSA.[18] The sites of involvement included tonsils and cervical LNs, membrana nictitans and soft palate, gingiva, buccal mucosa, and pharynx, tongue, and epiglottis. The appearance of oral epitheliotropic T cell lymphoma (ETCL) mimics the appearance of erythema multiforme in dogs.[112] Both diseases appear as stomatitis, and a biopsy is required for confirmation. In three dogs with ETCL, single oral masses or gingival enlargement were the only lesions present.

Surgical treatment of oral cavity LSA is uncommon due to the often-diffuse lesions present. RT and chemotherapy are often used to manage the disease. A study of dogs treated with RT alone (N = 10) and RT plus chemotherapy (N = 4) demonstrated that overall MST was 770 days.[113] Dogs that had a complete response to RT and those without evidence of LN metastasis had longer survival times.

Eleven (2.9%) of 371 cats with oral tumors had oral LSA, and there were also two with tonsillar LSA.[4] The appearance of oral LSA was described as single or multiple raised submucosal masses composed of unencapsulated sheets of neoplastic lymphoid cells. Of the 13 cats with tumors, seven tumors were lymphoblastic and six were lymphocytic. The overlying gingiva was often ulcerated with secondary chronic inflammation and necrosis.

In a report on the use of lomustine in 17 cats with LSA, two cats had oral LSA, one had a PR, and one had progressive disease following therapy.[114] RT alone or in combination with chemotherapy has been used in cats with LSA, with one cat with maxillary LSA having a CR (survival, 47 weeks) and one cat with a mandibular lymphoma having a PR (survival, 9 weeks).[115]

Miscellaneous tumor types

Merkel cell tumor

Neuroendocrine (Merkel) cell tumors of the canine oral cavity are rare.[116] Merkel cells occur in the epidermis in close association with nerve endings and have been reported to occur in the oral mucosa. Merkel cells are part of the neuroendocrine system and function as mechanoreceptors. Although typically dermal in origin, there is a report of four dogs with oral Merkel cell tumors.[116] The dogs ranged in age from 7 to 9 years; three of the tumors were located on the gingiva, one caused lysis of underlying bone, and one was located on the lip. Merkel cell tumors are clinically benign in behavior. Three dogs underwent surgical excision, which was curative, and the fourth dog was treated with RT and was tumor-free for 18 months.

Mast cell tumor

Mast cell tumors (MCTs) are the most common cutaneous tumors of dogs, and rarely, they can arise from the oral mucosa. In 130 dogs with oropharyngeal tumors, there was 1 dog with an MCT of the buccal mucosa.[18]

In a retrospective review of a total of 57 canine tongue tumors, there were five dogs with MCT.[28] The dogs ranged in age from 3 to 10 years. Treatment was mainly surgical. The longest survival time was 16 months, and local disease control was achieved only in one of the five dogs. A retrospective study examined 33 dogs with oral MCT.[117] Fifty-five percent had cytologic evidence of LN metastasis at diagnosis, and those dogs had an MST of 276 days versus "not reached" in dogs without LN metastasis. Dogs with MCT with a mitotic index of >5/10 high-powered fields (HPFs) had a shorter DFI and ST than did those with <5/10 HPFs. Locoregional progression was common in these patients, and dogs with adequate local control of their tumor had an improved outcome (MST 242 days without adequate local control vs. "not reached" if local control was attained via treatment). Treatments varied, but one-third received RT and two-thirds received chemotherapy. The authors concluded that staging for LN metastasis, biopsy of the tumor to determine the mitotic index (MI), and wide excision to attempt to achieve local control of the tumor were important in the management of this disease.

Three oral MCTs were diagnosed out of 371 cats with oral tumors. One cat had an MCT arising from the soft palate, and two had tumors at the mucocutaneous junction of the lips.[4] The ages ranged from 3 to 8 years, with a mean of 5.3 years.

Granular cell tumor (previously granular cell myoblastoma)

Granular cell tumors were previously termed granular cell myoblastomas, but myoblasts were not determined to be the origin of these tumors. The cell type of origin is unknown. In dogs, sites of occurrence included tongue (N = 8), lip (N = 1), and larynx (N = 1).[118] Age ranged from 2.5 to 13 years (mean, 8.9 years). There was no breed or sex predilection. There was no evidence of tumor recurrence or metastasis after surgery. In a retrospective study of 57 dogs with tongue tumors, seven dogs were diagnosed with granular cell myoblastoma.[28] Ages ranged from 7 to 15 years, and there was no apparent sex predilection. One dog had diffuse involvement of the tongue, five tumors occurred on the dorsal, and one on the ventral surface. Six of the seven dogs had early-stage disease (T1aN0M0). Treatment entailed surgery alone. Survival times ranged from 2 to 72 months. One dog died with metastatic disease, and only one dog did not have local tumor control.

Hemangiosarcoma

In a survey of 469 oropharyngeal tumors in dogs, there were 6 hemangiosarcomas (HSAs; 1%).[2] In a compilation of 130 dogs with oropharyngeal tumors, there were two dogs identified with oral HSA of the gingiva.[18] Three out of 57 dogs with lingual tumors were diagnosed with HSA.[28] In another report, 38 of 646 dogs with lingual tumors had HSA, which was the third most common tumor. Border collies were 11.8 times as likely to be diagnosed with this tumor as opposed to other breeds.[29] In a retrospective study of 20 dogs with lingual HSA, most were stage 1 (<2 cm diameter), most (70%) were incidentally discovered during a routine exam or dental cleaning, and most (70%) were on the ventral aspect of the tongue. In 25% of dogs, multiple lesions were identified. Treatments included surgical excision with or without chemotherapy. Median PFS was 524 days and the MST was 553 days. Prognostic factors significantly associated with increased survival included small tumor size and absence of clinical signs of an oral mass at the time of diagnosis. Neither the use of chemotherapy postoperatively nor obtaining tumor-free surgical margins was prognostic for outcome.[119]

In a retrospective study of 395 neoplasms from 372 cats, there were two cats with oral HSA; one involved the gingiva and one involved the palate.[3] In a retrospective study of 53 cats with HSA, two involved the gingiva.[120] Both cats had surgery with incomplete excision and no other adjunctive therapy.

Papillomatosis

Oral papillomas can be solitary but are often multiple cauliflower-like growths that may have frond-like projections (Fig. 44.8).[121–128] This tumor occurs most commonly in young dogs. Canine oral papillomatosis virus is contagious from one dog to another but is species specific. Experimentally induced canine oral papillomas routinely undergo incubation periods of 4–8 weeks, rapid growth and expansion, followed by spontaneous immune-mediated regression within an additional 4–8 weeks.[121] Treatment in clinical cases is usually unnecessary as the lesions will typically regress spontaneously. Excisional surgery, laser surgery, or cryosurgery may be indicated in dogs that have multiple lesions that are causing difficulty eating. However, immune-compromised dogs tend to show persistent and florid lesions that are refractory to therapy.[122–124] Autogenous vaccinations prepared from homogenized papillomas have been found to induce regression of the lesions in a number of species, including dogs.[125–128] Inactivated papilloma extract and recombinant vaccinations consisting of viral coat proteins have been reported as a prophylaxis of canine oral papillomatosis.[126,127] In one case report, a recombinant vaccination was used therapeutically and led to resolution of persistent, florid oral papillomatosis in a young dog.[128]

• **FIG. 44.8** Extensive oral papillomatosis in a dog.

In a recent report, of a total of seven domestic cats that had papillomas; five involved haired skin and two had lingual tumors.[129] The two cats with lingual papillomas were 6 months and 9 years of age. The oral tumors were described as multifocal, small, soft, light-pink, oval, slightly raised flat, sessile lesions, less than 10 mm in diameter on the ventral lingual surface. The tumors were all positive for papillomavirus antigens by immunohistochemistry.

Salivary gland tumors

The histological classification of salivary gland tumors in humans includes adenomas, carcinomas, nonepithelial tumors, and malignant LSA (arising from lymphoid tissue associated with salivary glands).[130] The adenomas include a number of subtypes, the most common being the pleomorphic adenoma, and ductal papilloma and cystadenoma. The carcinomas comprise the different types of adenocarcinoma, ductal carcinoma, SCC, and mucoepidermoid carcinoma. Salivary gland tumors are relatively rare in the dog and cat, and not all types have been recognized. Salivary gland tumors arise most commonly in the major salivary glands but can occur in minor accessory salivary gland tissue present throughout the oral cavity. In a search of 87,392 biopsy records, there were a total of 245 cases (0.3%) in which salivary gland tissue was evaluated from 160 dogs and 85 cats, and 30% of these were malignant neoplasms.[131] Malignant salivary gland tumors included salivary gland adenocarcinoma or carcinoma (36 dogs and 31 cats), salivary adenocarcinoma in dispersed accessory salivary tissue of the oral submucosa and tongue (two dogs and two cats), and malignant mixed salivary gland tumor (three dogs). The mandibular gland was most frequently involved, with the parotid gland second. Benign tumors of the salivary glands included salivary gland adenoma/cystadenoma (two cats) and accessory papillary adenoma (one cat). Clinical signs associated with salivary gland tumors include a cervical mass, dysphagia, anorexia, and pain on opening the mouth. Surgical excision is the treatment of choice, but complete surgical excision is challenging due to the location and normal tissues surrounding salivary gland tumors. Orthovoltage RT was successfully used postoperatively in three dogs with parotid gland adenocarcinomas.[132] Survival time in one dog was 40 months and the other two dogs were alive with no evidence of disease at 12 and 25 months. There is a report on treatment of salivary gland tumors in dogs (N = 24) and cats (N = 30) that utilized various combinations of surgery, chemotherapy, and RT.[133] There were a total of seven dogs and five cats treated with megavoltage RT. The overall median survival time for all dogs was 550 days, and the surgery and chemotherapy group had shorter survival times compared to the surgery alone or surgery and RT groups. Of the 30 cats, nine were Siamese or Siamese-cross, there was a 2:1 predilection for males, and the median age was 12 years (range, 7–22 years). The mandibular gland was most commonly affected (59%), followed by the parotid gland. Various treatment modalities were utilized, with an overall median survival of 516 days, with no difference in survival based on treatment type.

A review of archival material from 35 cats with salivary gland tumors revealed five cats with salivary duct carcinomas.[134] Tumor location in four cats included parotid gland, and minor salivary gland tissue from the oral cavity, soft palate, and cheek. The ages ranged from 7 to 13 years, with a median of 8, and there were four females and one male. Two cats had evidence of metastasis, but complete case information was not available.

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CHAPTER 45

Clinical behavior of odontogenic tumors

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General considerations

Odontogenic tumors (OTs) are unique to the oral cavity and arise from remnants of the embryonic tissues destined to develop into teeth and associated structures.[1-3] They originate from remnants of odontogenic epithelium (rests of Malassez and rests of Serres located within the periodontal ligament stroma and gingiva, respectively), odontogenic mesenchyme, or a combination of the cellular elements that comprise the tooth-forming apparatus.[2,4,5] Clinical behavior ranges from hamartoma-like proliferations to benign and invasive neoplasms.[1,2] Box 45.1 lists OTs according to their behavior and potential for recurrence. Although the etiology and pathogenesis remain unknown, fundamental knowledge of odontogenesis is important to understanding the categorization and biologic behavior of these neoplasms.[5] Histologically, OTs may mimic some stage of the developing tooth bud.[4] There may be soft tissues of the dental organ or dental pulp, or they may contain hard tissue elements of enamel, dentin, and/or cementum.[1,2]

• BOX 45.1

Biologic Classification of Odontogenic Tumors

Benign, little recurrence potential

Odontoma

Benign, some recurrence potential

Peripheral odontogenic fibroma

Benign, locally aggressive

Central ameloblastoma

Canine acanthomatous ameloblastoma
Amyloid-producing odontogenic tumor
Feline inductive odontogenic tumor

Malignant[a]

Ameloblastic fibro-odontoma
Odontogenic myxoma
Ameloblastic carcinoma

[a]*Malignant odontogenic tumors are extremely rare in dogs and cats.*

With the exception of the canine peripheral odontogenic fibroma (POF) (formerly fibromatous epulis) and canine acanthomatous ameloblastoma (CAA) (formerly acanthomatous epulis), OTs are considered uncommon in animals.[4,6] The most detailed information on OTs is obtained from human studies; however, this knowledge is not always transferable to companion animals, and it must be appreciated that incidence, morphology, and biological behavior variations occur between different species.[4,7]

Although there are several studies addressing the overall incidence of oral and odontogenic tumors in animals, there have been no recent additions to the literature.[6,8-14] The actual incidence is unknown because of confusion over nomenclature (tissue of origin) and the fact that many clinicians do not routinely submit specimens for histopathological identification.[4,7,15,16] For example, gingival enlargements are often considered simply as hyperplastic or inflammatory lesions and not submitted for definitive diagnosis.[5,17] A bias is therefore incorporated into studies based on archival material.[15]

Historically, OTs have fallen under the heading of *epulides* (singular *epulis*). The term *epulis* has no specific histopathologic connotation and is a clinical designation for any localized, exophytic swelling on the gingiva.[15,18,19] The term is still commonly utilized in the veterinary literature and is a source of confusion, as several different pathologic entities are collectively categorized as epulides. It is erroneous to consider all epulides as benign and inconsequential. In dogs, many epulides are actually OTs, and their true nature as such should be determined histologically by pathologists with formal training in the histologic characteristics of OTs and cysts.[15] Differential diagnosis of OTs is presented in Box 45.2.

• BOX 45.2

Differential Diagnosis of Odontogenic Tumors

1. Localized gingival enlargements

Reactive lesions
- Focal fibrous hyperplasia
- Pyogenic granuloma
- Peripheral giant cell granuloma

Reactive exostosis

Odontogenic tumors

Peripheral odontogenic fibroma

Canine acanthomatous ameloblastoma

Central ameloblastoma

Odontoma

Odontogenic cysts

Dentigerous cyst

Odontogenic keratocyst (keratocystic odontogenic tumor)

Nonodontogenic tumors (see Chapter 44)

2. Gross swellings with distortion of bone

Odontogenic tumors

Peripheral odontogenic fibroma

Canine acanthomatous ameloblastoma

Central ameloblastoma

Odontoma

Odontogenic cysts

Dentigerous cyst

Canine odontogenic parakeratinized cyst

Odontogenic keratocyst (keratocystic odontogenic tumor)

Non-odontogenic tumors (see Chapter 44)

3. Osteolytic and cystic lesions

Odontogenic tumors

Central ameloblastoma

Amyloid-producing odontogenic tumor

Odontogenic cysts

Radicular cyst

Dentigerous cyst

Canine odontogenic parakeratinized cyst

Odontogenic keratocyst (keratocystic odontogenic tumor)

Currently, OT classification in veterinary medicine utilizes the tissue of origin as the basis for distinguishing between the various types. The three resulting groups are (1) those consisting of odontogenic epithelium *without* odontogenic mesenchyme; (2) tumors arising from odontogenic epithelium and mesenchymal cells, with or without dental hard tissue formation; and (3) those consisting primarily of mesenchyme, with or without odontogenic epithelium.[2,4,20,21] In 2002, a revision was suggested to the World Health Organization's 1992 typing of OTs.[20,22] Inclusion of immunochemistry and molecular biology methodologies was recommended.[20,23] These revisions were adopted in 2005.[24] Although traditional histopathology remains the gold standard for diagnosis, future advancements in OT classification schemes are likely to be based on genetic, molecular, biologic, and immunochemical criteria.[21,25,26]

Epithelial tumors

Ameloblastoma

Ameloblastoma is an epithelial tumor that originates from remnants of the dental organ (rests of Malassez), dental lamina, cells of epithelial origin lining odontogenic cysts, or possibly from basal epithelial cells of the oral mucosa.[6,14,15,17,27] It has been described in many domestic species and occurs in the tooth-bearing areas of the mandible, maxilla, and incisive bone.[4,6,8,14,15,17-19,28] Ameloblastoma does not produce enamel or dentin and so remains a soft tissue tumor. It is considered the least differentiated of the epithelial OTs.[5,6,14] In spite of a benign histological appearance in animals, ameloblastoma typically is a locally invasive, slowly growing neoplasm that does not metastasize.[4,14,18,27] The stimulus for neoplastic transformation of odontogenic epithelium is not completely understood; however, advances in the human field have shown that molecular and genetic factors resulting in the neoplastic transformation are strongly linked to multiple genes associated with mitogen activated protein kinase, sonic hedgehog, and WNT/β-catenin signaling pathways.[29] Hypercalcemia of malignancy has been associated with ameloblastoma in dogs, horses, and humans.[30-32]

Central (or intraosseous) ameloblastoma

Clinical features

The central ameloblastoma is a relatively uncommon OT in dogs.[4,6] It usually manifests as a gross swelling with distortion of bone; however, the presenting complaint may be notable tooth displacement or malocclusion.[2] Because it is usually painless, with slow expansion, patients may be asymptomatic.[4,16,27] Central ameloblastoma most often appears radiographically as an osteolytic, unilocular, or multilocular cystic lesion around tooth roots, with well-defined, sclerotic margins (Fig. 45.1).[2,5,18] Jaw expansion with or without cortical thinning is usually apparent.[2,18] In cats, central ameloblastoma is considered rare and sometimes confused with other, more common types of OTs in this species.[28] Two histologic variants have been reported in cats: a follicular pattern similar to that in humans and a keratinizing pattern similar to that in dogs.[28]

• **FIG. 45.1** Radiographic appearance of a central ameloblastoma in a dog. Source: (From Verstraete FJM. *Self-Assessment Color Review of Veterinary Dentistry*. 1st ed. Ames: Iowa State University Press, 1999.)

Histopathology

The classical histological appearance of an ameloblastoma includes palisading of columnar basal epithelial cells perpendicular to the basement membrane, polarization of basal cell nuclei away from the basement membrane (reverse polarization), and hyperchromatic nuclei. Stellate reticulum may or may not be present focally.[4,6,14,17,33] Ameloblastoma in the dog may have a prominent feature of the central cells undergoing varying degrees of keratinization with subsequent calcification of the keratin.[34] This is referred to as keratinizing ameoloblastoma.[14,34]

Treatment recommendations

Wide surgical excision (mandibulectomy or maxillectomy) is the treatment of choice.[35] In a survey of 12 dogs with central or keratinizing ameloblastomas, local recurrence was noted in two of two patients who were treated with local nonaggressive surgery (curettage only) and three of four patients treated with radiation therapy and for whom follow-up information was available.[34] Conversely, three of three patients who were treated with radical surgical excision remained tumor-free with follow-up of up to 28 months.[34]

Prognosis

Wide surgical excision is considered curative.[14,34]

Canine acanthomatous ameloblastoma

The second, and more common, histologic variant of ameloblastoma found in dogs is the CAA. In fact, one study found CAA to be the most common OT in the dog.[36] In two feline surveys, no acanthomatous ameloblastoma was diagnosed.[8,9] In previous classification schemes, this was called acanthomatous epulis and was distinguished biologically and histologically from other types of epulides by the tendency to infiltrate cancellous bone.[4,7,19,24] The acanthomatous epulis was later recognized as a type of ameloblastoma and termed a "peripheral ameloblastoma."[15] The term *peripheral ameloblastoma* was later replaced by *CAA*, to differentiate it from the peripheral ameloblastoma in humans, which is a noninvasive tumor type.[37] CAA also arises from remnants of odontogenic epithelium located in the gingiva (rests of Serres) in the tooth-bearing areas of the jaws.[19,24] Although CAA is generally accepted as arising from gingival epithelium, it may also arise intraosseously and then break out of bone.[37]

Clinical features

CAA typically appears as an exophytic gingival mass with an irregular surface (Fig. 45.2A). It is considered a benign tumor with invasive properties into surrounding bone. The infiltration of bone distinguishes the CAA from central ameloblastoma.[24] The radiographic pattern is dominated by bony infiltration, alveolar bone resorption, and tooth displacement (Fig. 45.2B).[5,15] On computed tomographic (CT) imaging, intraosseous ameloblastomas were often found to be cystic in structure and appeared subjectively more aggressive than extraosseous masses.[38] The tumor has a predisposition for the rostral mandible (41% of cases) and is found to affect the caudal maxilla least frequently (6% of cases).[36] Golden retrievers, Akitas, cocker spaniels, and Shetland sheepdogs were overrepresented among dogs with CAA.[39]


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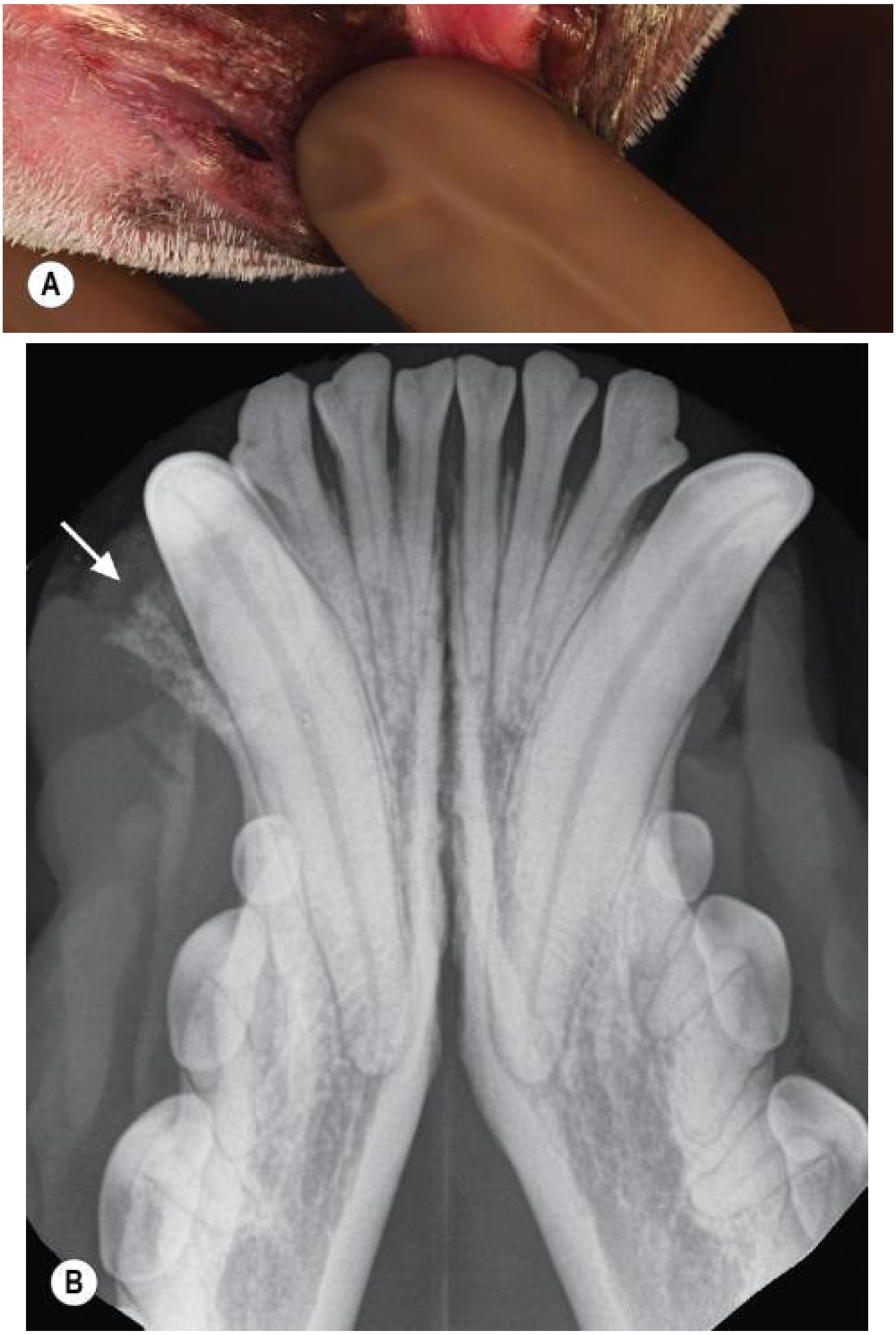


• **FIG. 45.2** (**A**) Clinical and (**B**) radiographic appearance of a canine acanthomatous ameloblastoma in the right mandibular canine tooth of a dog. The *arrow* indicates osseous proliferation and widening of the periodontal ligament space at the buccal aspect.

Histopathology

Histologically, CAA is composed of islands and sheets of mature cells that are clearly squamous epithelium.[15,37] The connective tissue component is typically of low to moderate cellularity; however, the stroma may be variable: similar to the dense connective tissue of the gingiva, fibroblastic connective tissue of the periodontal ligament, or the loose connective tissue of bone marrow.[15,37] The epithelial islands and sheets are each bounded by a row of palisading cells with reverse nuclear polarization, and infiltration in the underlying bone is evident in most cases.[15]

Treatment recommendations

Because CAA is locally aggressive and invades the bone, curative surgical treatment requires en bloc excision of the tumor and at least 1 cm of normal-appearing tissue.[40] This is typically achieved by performing partial mandibulectomy or maxillectomy.[5] Radiation therapy is also an option, and good results have been reported.[41-44] However, there is significant risk of osteoradionecrosis,[44,45] which can lead to permanent oronasal fistula formation. In addition, malignant tumor formation has been reported in up to 18% of previously irradiated sites.[40,43,45] Intralesional bleomycin injections have been reported for the treatment of CAA in a series of seven dogs, with no recurrence noted in the 842-day follow-up period.[46] However, adverse reactions ranging from local swelling to wound formation with bone exposure were reported. Therefore wide surgical excision continues to be the treatment of choice for CAA.[47]

Prognosis

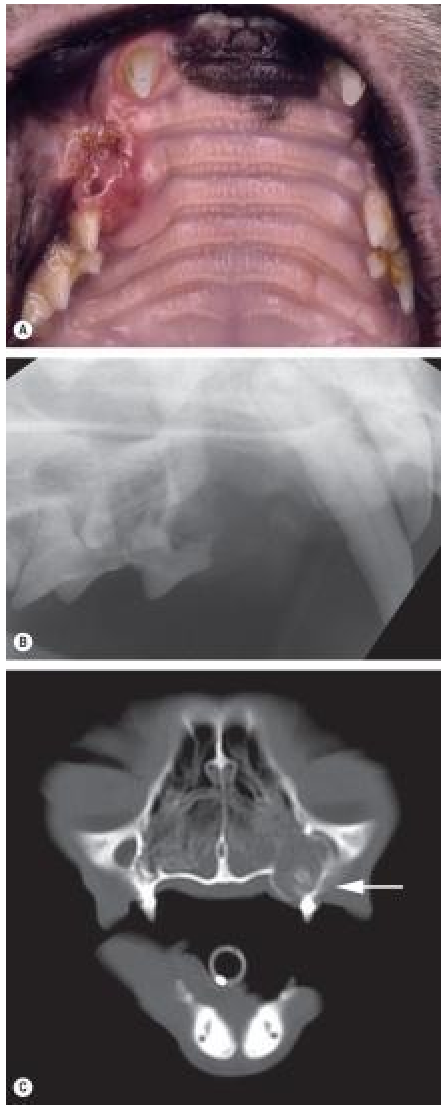
If the tumor is completely excised, the prognosis is excellent. Following wide local excision, one author reported a 1-year survival rate of 100% among 25 dogs,[34] while another study reported a 1-year survival rate of 97% among 42 dogs.[48] In the latter, excision was incomplete in one dog, resulting in local recurrence of the tumor.[48]

Amyloid-producing odontogenic tumor

Amyloid-producing OT (APOT) was previously referred to in the veterinary literature as a calcifying epithelial OT but was later determined not to be equivalent to the human calcifying epithelial OT.[14,49,50] This OT type is rare in dogs and cats and presents in patients between 1 and 13 years.[19,36,49] One study in dogs suggested a female predisposition and a possible predilection for growth in the mandible.[36] Until recently, the histogenesis of APOT was poorly understood; however, it has been shown, through biochemical and immunohistochemical methods, that in both cats and dogs, the tumor originates from ameloblasts.[51,52]

Clinical features

APOT appears as a gingival enlargement that grows by expansion. It is locally invasive but not metastatic.[50] CT imaging of this tumor has been reported to have specific imaging characteristics, including intraosseous location, cortical wall expansion, heterogeneous contrast enhancement, cystic structure, and amorphous mineralization.[38] It often has a cystic appearance on radiographs (Fig. 45.3). A recent publication noted the presence of APOT in the facial skin of three cats.[53] The authors proposed that residual odontogenic epithelial cells are present in the facial skin of cats.



• **FIG. 45.3** (**A**) Clinical, (**B**) radiographic, and (**C**) computed tomographic appearance of an amyloid-producing odontogenic tumor (arrow) in a cat.

Histopathology

Histologically, APOT is a very cellular epithelial tumor. It bears some resemblance to ameloblastoma, with palisading of the basal epithelial cells in some areas; reverse polarizations of nuclei, stellate reticulum, and collagenous matrix are variable focal findings.[19,37] Amyloid (Congo red stain), which tends to calcify, may be present in some parts of the tumor.[19] Any epithelial tumor of the jaws that exhibits amyloid should be suspected of being odontogenic in origin.[4]

Treatment recommendations

Although metastasis has not been observed, local recurrence may occur after incomplete excision.[6,54] Radiation therapy has reportedly been successful in preventing recurrence in one cat after incomplete surgical excision.[55] One author reports no evidence of local recurrence after marginal excision in one dog and two cats, with follow-up ranging from 7 to 19 months.[35] However, for definitive treatment, wide excision is recommended.

Prognosis

Complete excision is considered curative.[56]

Mesenchymal tumors

Peripheral odontogenic fibroma

Many of the tumors previously described as fibromatous and ossifying epulides have been reclassified as POF.[15,19]

Clinical features

POF is a slowly growing, benign neoplasm that is common in the dog and uncommon in the cat.[6,8,9,15,19] It was found to be the second most common OT in dogs after the CAA.[36] POF has a predilection for the rostral maxilla and is more likely to affect castrated dogs.[36] Surface epithelium appears normal, and radiographic features vary according to the presence and amount of mineralized products (Fig. 45.4).[15] Radiographically, these neoplasms result in minimal osseous destruction and vary from soft tissue only to soft tissue with amorphous mineral opacity.[38,57]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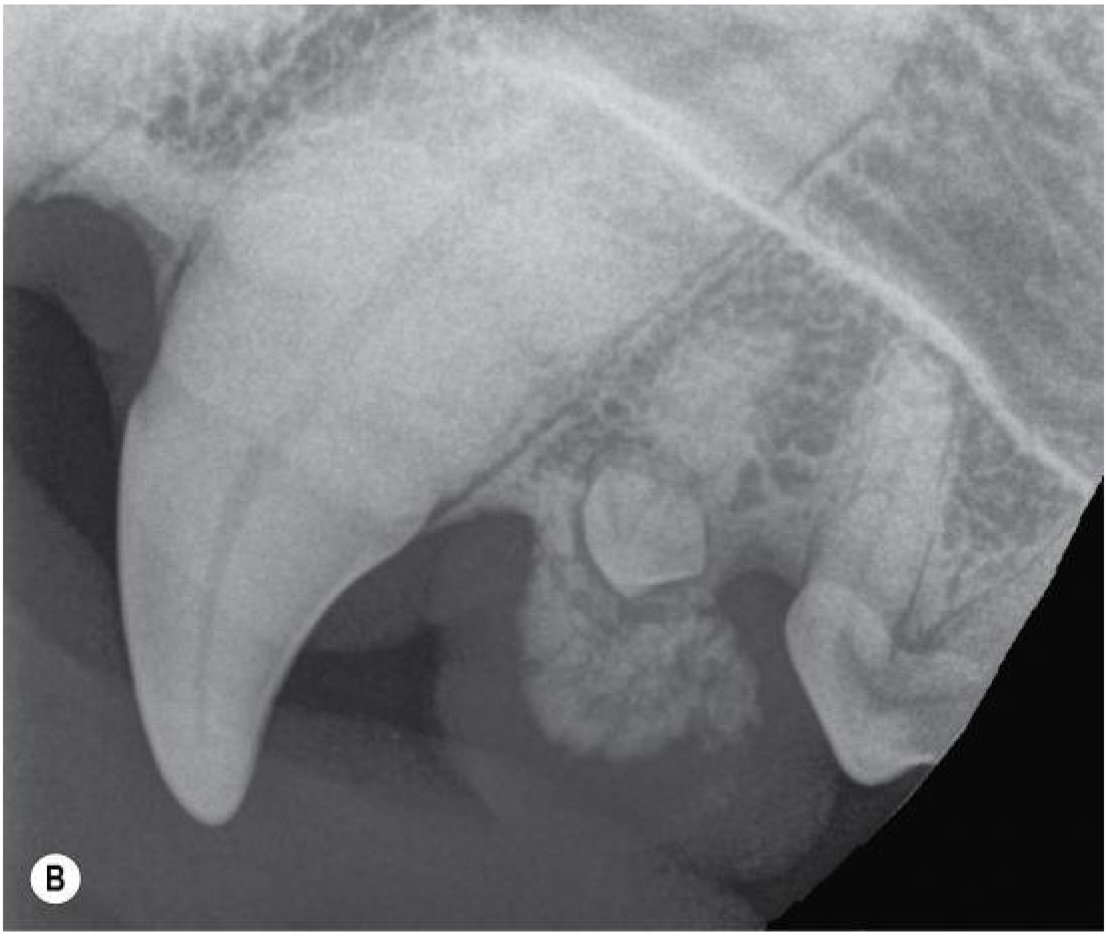
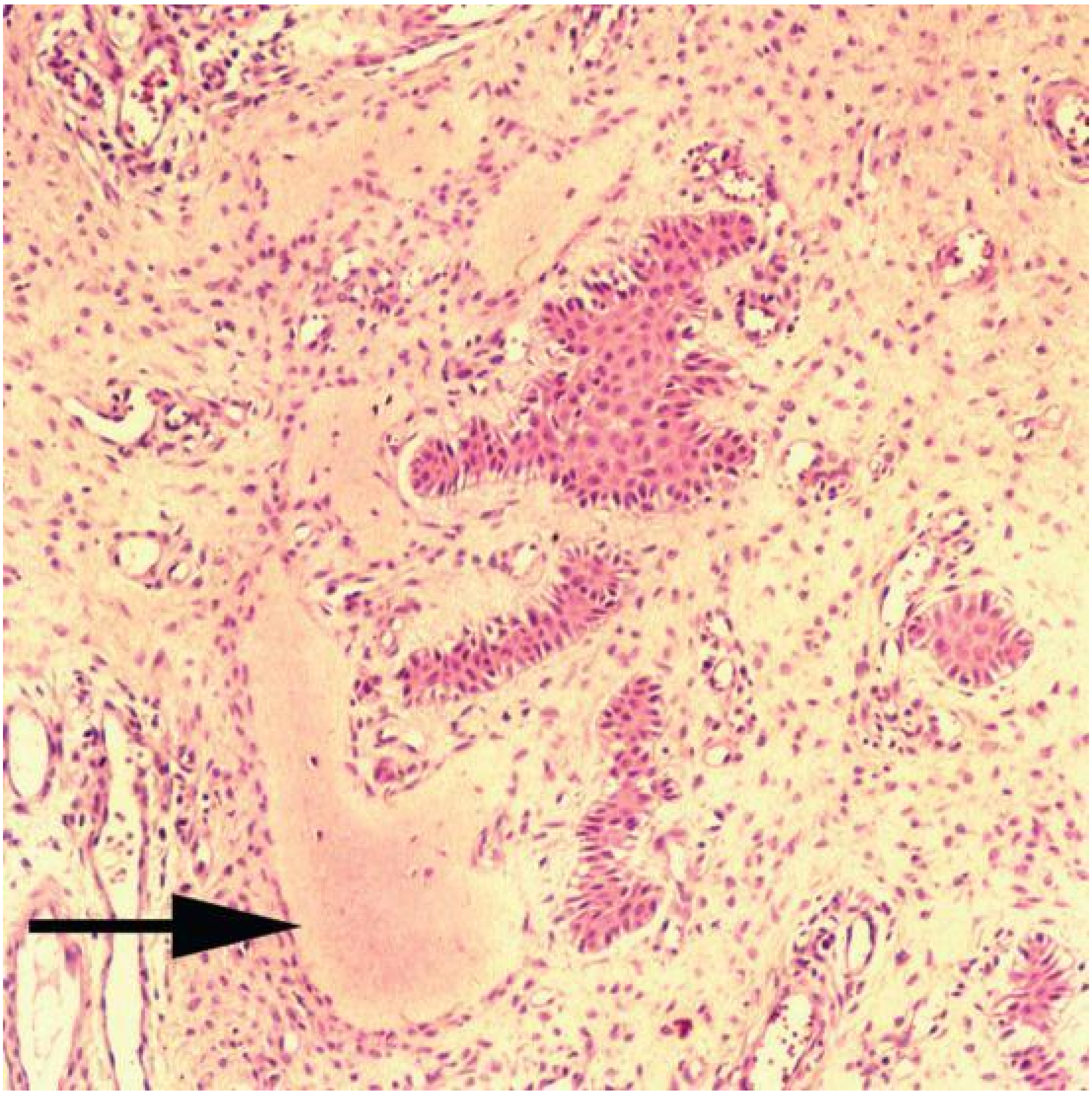


• **FIG. 45.4** (**A**) Clinical and (**B**) radiographic appearance of a pedunculated and partially mineralized peripheral odontogenic fibroma emerging from the level of the left maxillary first premolar tooth in a dog.

Histopathology

Histologically, POF is characterized by a low-grade neoplastic proliferation of fibroblastic connective tissue of variable cellularity in which a variety of bone, osteoid, dentinoid (dentin-like), or even cementum-like material is present (Fig. 45.5). Isolated islands or strands of proliferative odontogenic epithelium are found in close association with foci of hard tissues, suggesting induction.[9,15,19]



• FIG. 45.5 Histopathologic appearance of a peripheral odontogenic fibroma. Note the foci of mineralization, the largest of which is marked with an *arrow*.

Treatment recommendations

Definitive treatment typically requires en bloc excision of the mass and underlying bone. In one survey including 17 dogs with POFs that were marginally excised (i.e., without including the underlying bone), three of the tumors recurred.[17] En bloc excision has been reported to result in tumor-free margins and local cure in two cases that had previously recurred following radiation therapy[58] or marginal excision.[59]

Prognosis

Incomplete excision and local recurrence can result in difficulty with prehension and mastication; one author reported mortality secondary to malnutrition following recurrence of an incompletely excised "ossifying epulis."[60] Complete excision, which may be achieved with en bloc excision of the mass and underlying bone, is considered curative.

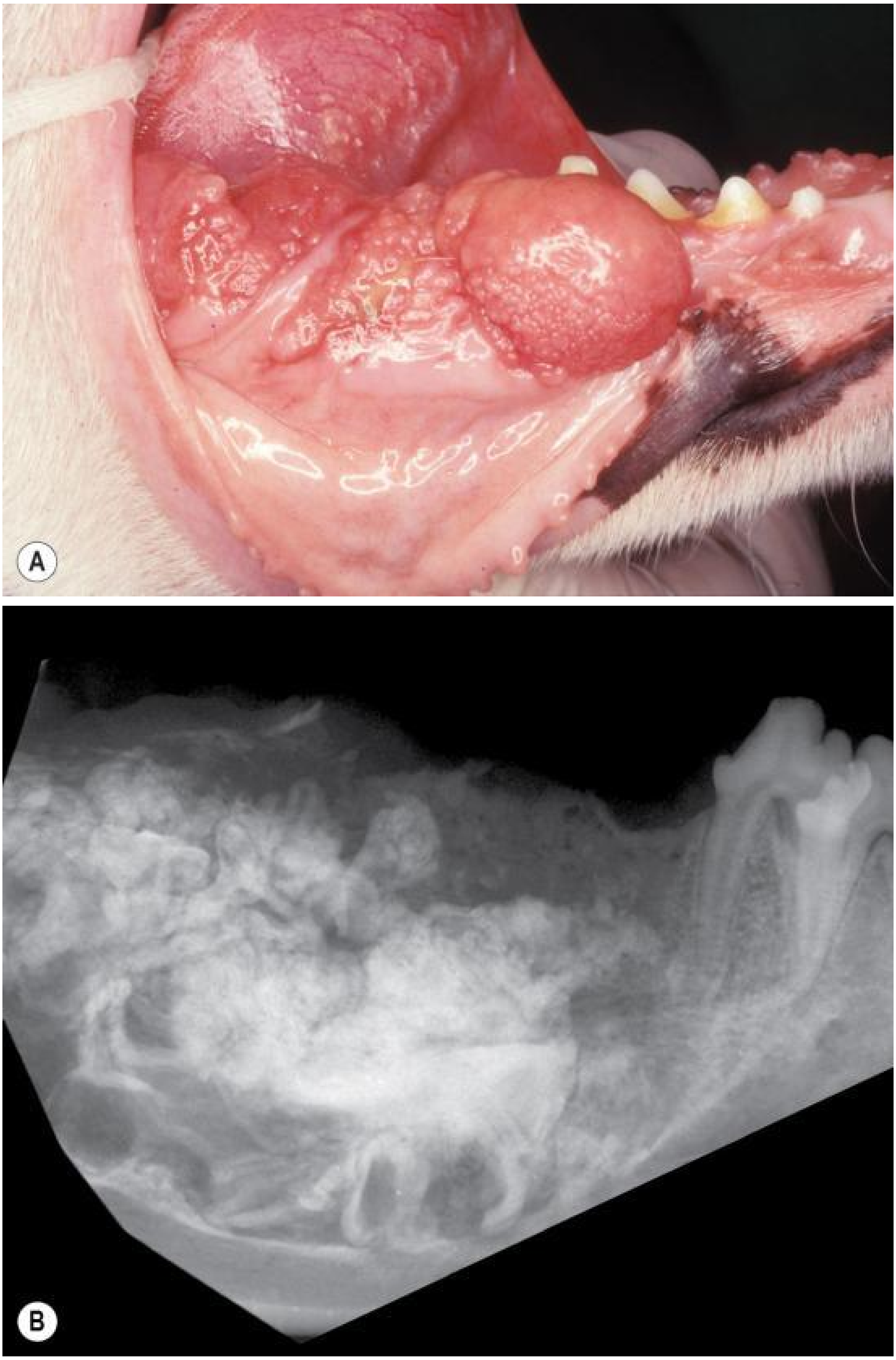
Mixed (mesenchymal and epithelial) tumors

Odontoma

Odontomas are benign inductive tumors diagnosed in young dogs and cats, generally appearing at 6 to 18 months of age.[4,6,14,61-66] They are the most common OT in humans and may be associated with an unerupted tooth, a dentigerous cyst, or attached to an otherwise normal tooth.[4,5] Because odontoma contains well-differentiated cells, it is characterized by the simultaneous occurrence of a composite of soft and hard dental tissues (enamel, dentin, cementum, and dental papilla) of both epithelial and mesenchymal origin.[2,4,6] Odontoma may be considered a nonneoplastic hamartoma.[4] Hamartoma is a tumor-like malformation derived from local cellular hyperactivity and is composed of excessive cells and tissues (native to a part), which arises during development.[67,68]

Clinical features

Odontomas often appear as a nonpainful, expansile enlargement of the maxilla or mandible.[69] The dental tissues in odontomas may or may not exhibit a normal relation to one another. An odontoma in which rudimentary tooth-like structures are present indicates advanced cellular differentiation and is referred to as a *compound* odontoma. An odontoma in which the conglomerate of dental tissues is disorderly, bearing no resemblance to a tooth, is called a *complex* odontoma.[4-6,14] The radiological appearance is typical: either a sharply defined mass of calcified material surrounded by a narrow radiolucent band (complex odontoma) or a variable number of tooth-like structures (compound odontoma) are present.[5] If identified in its early stages, the lesion may be primarily radiolucent with focal areas of opacifying hard dental tissue.[2] Complex odontomas appear as opaque amorphous masses, while compound odontomas appear as numerous tiny tooth-like structures in a focal area (Fig. 45.6).[2] The term *ameloblastic odontoma* is occasionally encountered in the veterinary literature and represents an ameloblastoma with focal differentiation into an odontoma.[4,6,14]



• **FIG. 45.6** (**A**) Clinical and (**B**) radiographic appearance of a complex odontoma in a dog. The radiographic appearance of multiple tooth-like structures is pathognomonic.

Treatment and prognosis

Because odontomas are not true neoplasms, marginal excision or even intracapsular excision to remove the abnormal material may be curative.[69]

Feline inductive odontogenic tumor

Clinical features

This tumor type is unique to young cats (usually 8 to 18 months).[5,9,18,70,71] It was originally described as inductive fibroameloblastoma.[9] Feline inductive OT (FIOT) is a raised, submucosal, soft tissue mass typically located unilaterally on the rostral maxilla (Fig. 45.7).[18,70,71] The tumor may be locally invasive, but metastasis has not been recorded.[5,18]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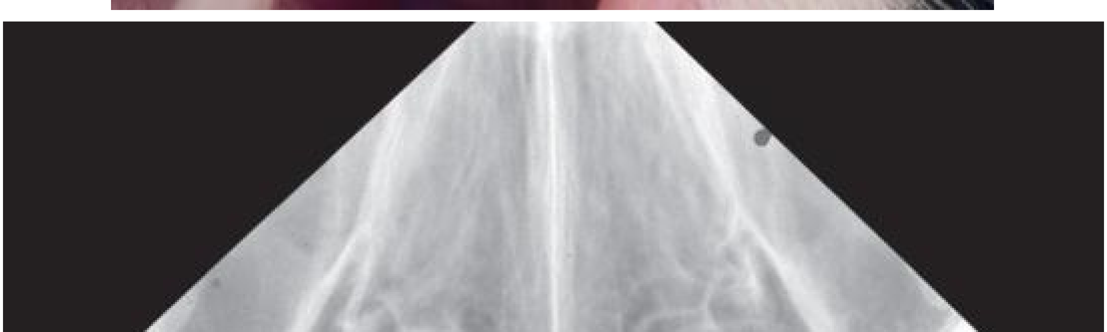


• **FIG. 45.7** (**A**) Clinical and (**B**) radiographic appearance of a feline-inductive odontogenic tumor. Note the absence of the left maxillary canine tooth in the region of the tumor, a hallmark of this tumor type.

Histopathology

This tumor is characterized histologically by multifocal formations of spherical condensations of fibroblastic connective tissue, associated with islands of odontogenic epithelium dispersed throughout the connective tissue stroma.[70,71] The ameloblastic-type epithelial cells may appear arranged around a dental pulp-like stroma of ecto-mesenchymal tissue.[9,70] Although cytological features of FIOT have been described,[72] histopathology remains the definitive method of diagnosis.

Treatment recommendations

Wide surgical excision is the treatment of choice. Radiation therapy has reportedly been successful in preventing recurrence in one cat after incomplete surgical excision.[55]

Prognosis

The biologic behavior of FIOT appears to be similar to that of other locally invasive but benign odontogenic neoplasms: wide surgical excision is considered curative, with local recurrence expected after incomplete excision.[70,71]

Malignant odontogenic tumors

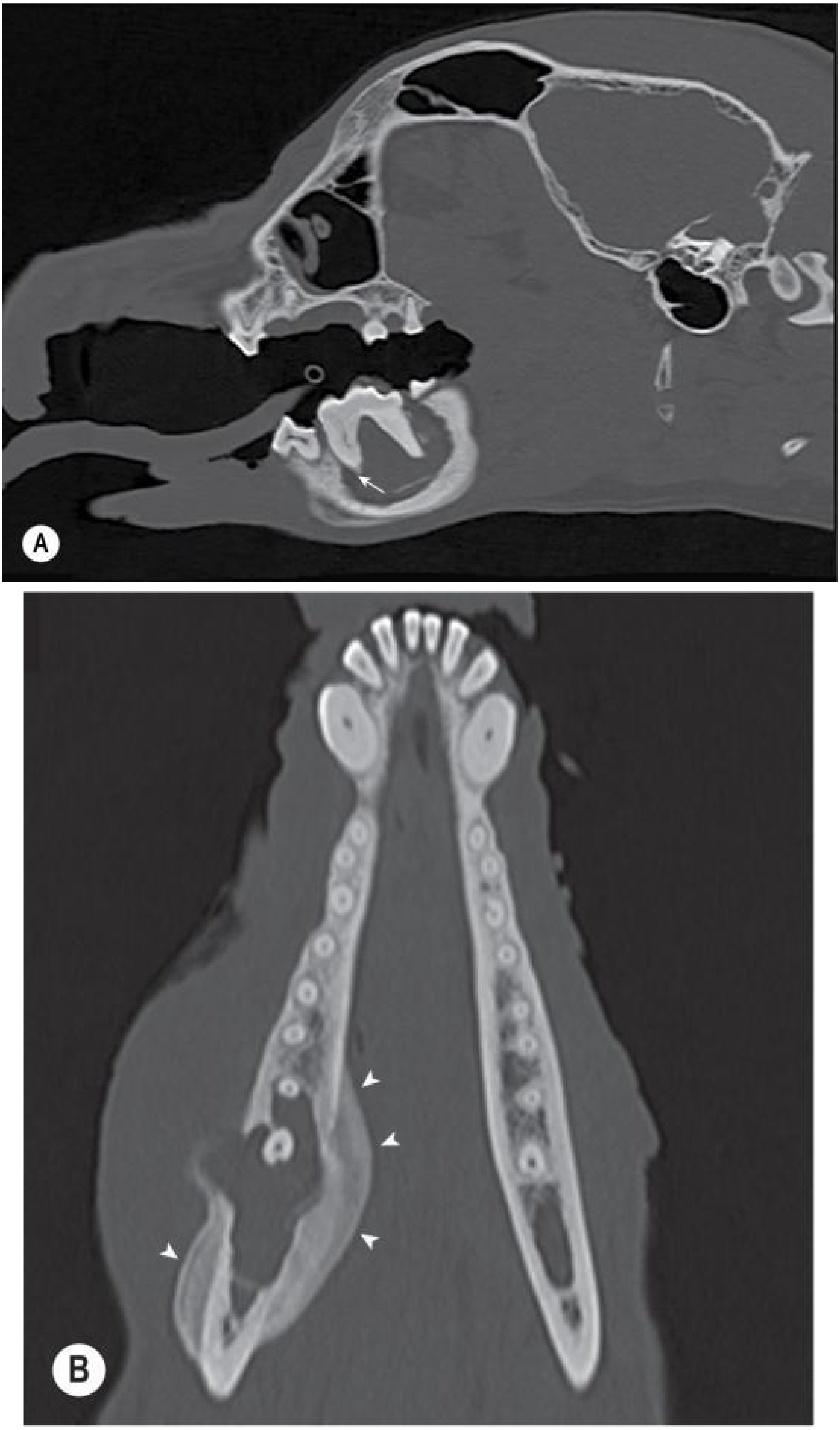
Malignant OTs are considered extremely rare; however, several have been reported in the dog, including ameloblastic fibro-odontoma, odontogenic myxoma, and ameloblastic carcinoma.[73-75] As with nonodontogenic malignant tumors, it is advisable to consult with an oncologist prior to determining a treatment plan (see Chapter 44).

Common radiographic and computed tomographic features of odontogenic tumors

Benign, slow-growing tumors will often result in the displacement of bony cortices and teeth as well as smooth root surface resorption.[76,77] Malignant tumors, which typically grow more rapidly, can result in alveolar bone destruction so that the teeth in the vicinity may appear to be floating.[78] They also cause inflammatory root resorption, which gives a spike-like shape to the roots.

In a study looking at the prevalence and types of tooth resorption in dogs with oral tumors, the authors found that teeth distant from where an OT is located had a higher prevalence of external surface resorption compared with teeth in control dogs.[79] They also noted that dogs with CAA and nonodontogenic (malignant) tumors had a higher prevalence of external inflammatory resorption at teeth both at the tumor site and distant from it. They recommended full-mouth radiography in the comprehensive treatment planning of dogs with oral tumors.[38]

A CT study of OTs found that, as a whole, OTs were often associated with displaced teeth, variable soft tissue attenuation, presence of cyst-like structures, lysis of cortical bone, and enhancement on postcontrast imaging (Fig. 45.8).



• FIG. 45.8 (**A**) Computed tomographic sagittal view and (**B**) coronal view of a central ameloblastoma in the right caudal mandible of a dog. Note the expanded cystic appearance of the lesion, near-total loss of attachment of the first molar tooth, inflammatory root resorption of the mesial root of the mandibular first molar tooth (**A**, *arrow*), and expansion of the lingual and buccal cortices as well as periosteal new bone formation (**B**, *arrowheads*).

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CHAPTER 46

Nonneoplastic proliferative oral lesions

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Gingival enlargement and related lesions

General considerations

There are a number of nonneoplastic, reactive conditions that occur as tumor-like lesions of the gingiva, and result from chronic low-grade irritation. These include gingival hyperplasia, focal fibrous hyperplasia (FFH), pyogenic granuloma, peripheral giant cell granuloma, peripheral ossifying fibroma, and reactive exostosis. The histological classification of these lesions may be confusing, as they represent a wide variety of different cellular elements.[1] Hyperplasia implies that the enlargement is caused by an increase in number of normal cells in normal arrangement.[2,3] In addition, enlargement of the gingiva may also be caused by an excessive accumulation of extracellular matrix proteins. In the latter case, or in the absence of a histopathological diagnosis, the term gingival enlargement is more appropriate.[3–6]

The clinical appearance of the different types of gingival enlargements is not pathognomonic.[1] They generally arise in close proximity to one or more teeth, as a pedunculated or sessile mass, and can be smooth or rough, and frequently ulcerated. They show no predilection for the maxilla or the mandible. Occasionally, gingival enlargement can be associated with displacement of teeth, with or without radiological signs of bone lysis. Due to the inability to distinguish between these lesions based on clinical appearance, hyperplastic lesions on the gingiva must always be biopsied, and should all be biopsied if multiple lesions are present.[7] Differential diagnoses include benign neoplastic lesions of odontogenic origin (see Chapter 45) and various malignant nonodontogenic tumors (see Chapter 44).

Disease entities

Gingival hyperplasia

Gingival hyperplasia is a proliferation of the attached gingiva, often due to a chronic inflammatory response to the bacteria in plaque, food impaction, dental malposition, or dental resorptive lesions.[1,8,9] The histological appearance is characterized by proliferation of the gingival epithelium and ulceration of the sulcular epithelium, in addition to the proliferation of the connective tissue, edema, and neoangiogenesis. In a later phase, the connective tissue proliferation and gingival fibrosis predominate, with less or no edema.[1,4] The gingival hyperplasia slowly progresses, if it is not complicated by trauma or secondary infection. Initially, it involves the marginal contour of the gingiva; subsequently it evolves and can almost completely cover the crowns of the teeth. The slowly growing painless enlargements tend to appear most commonly in areas occupied by teeth, not in edentulous areas.

These lesions can be localized, but often they are generalized, especially in breeds that are believed to have a familial tendency, such as boxer, collie, great Dane, Dalmatian, and Doberman (Fig. 46.1A).[10] Gingival hyperplasia is characterized by a slow development of an enlarged, firm gingival margin, can be symmetrical or localized, and can involve one or more dental quadrants.

• **FIG. 46.1** Gingival hyperplasia and related lesions. (**A**) Generalized gingival hyperplasia in a boxer dog. (**B**) Amlodipine-induced gingival enlargement in a dog treated for hypertension. (**C**) Focal fibrous hyperplasia in a dog.

Drug-induced gingival enlargement

Drug-induced gingival enlargement occurs in humans, dogs and cats (Fig. 46.1B).[1,5,11–15] The term *gingival enlargement* is preferred for all drug-induced gingival lesions previously termed *gingival hyperplasia* or *gingival hypertrophy*.[4–6] These older terms do not accurately reflect the histopathology of these lesions, which is characterized by excessive accumulation of extracellular matrix proteins, such as collagen, or amorphous ground substance.[5] An inflammatory infiltrate dominated by plasma cells and thickened keratinized epithelium is typically present.

The three types of drugs that have been documented to cause gingival enlargement are phenytoin derivatives, calcium channel blockers, and cyclosporine. Phenytoin is an anticonvulsant agent that is seen to cause gingival enlargement in human patients, with a reported incidence of 50%.[15] Two experimental studies in cats and one clinical canine patient with phenytoin-induced gingival enlargement have been documented.[11,12,14] In humans, the extent of enlargement is dependent on the presence of concomitant inflammation and a genetic predisposition to develop lesions. The enlargement is chronic, slow growing, regrows after surgical resection, and usually regresses after the drug is discontinued.[4–6]

Gingival enlargement caused by calcium channel blockers, such as nifedipine and amlodipine, has been documented in the human and veterinary literature.[15–18] As with most drug-related lesions, the presence of the enlarged gingiva makes plaque control difficult, resulting in secondary inflammation that further complicates the condition.[4–6]

Cyclosporine, an immunosuppressive agent, causes slow-growing gingival enlargement in approximately 25% of human patients treated.[5,6] Cyclosporine-induced enlargement is reversible following cessation of drug administration.[1,4–6] Drug-induced lesions are related to the level of oral hygiene and genetic susceptibility of an individual. Excellent oral hygiene greatly reduces, but does not eliminate, the occurrence of the lesions.[5,6] Frequent routine periodontal treatment is indicated for patients with cyclosporine-induced gingival enlargement.[5,6] Chlorhexidine rinses can reduce the severity of gingival enlargement and may prevent recurrence following gingivectomy.[5,6]

Cyclosporine, phenytoin, and calcium channel blockers are all calcium antagonists. Calcium plays an important role in apoptosis (programmed cell death).[3] The gingiva has the highest activity of transglutaminase, a calcium-dependent enzyme involved in apoptosis, when compared with other tissues of the body. Therefore, it is theorized that decreased calcium reduces normal apoptosis and, hence, deregulation of tissue overgrowth contributing to gingival enlargement.[3]

Focal fibrous hyperplasia

FFH is often incorrectly referred to as a fibrous or fibromatous epulis in veterinary medicine. It is a different entity from peripheral odontogenic fibroma, which is a benign neoplastic lesion of odontogenic origin. FFH is a reactive lesion believed to result from irritation caused by plaque and calculus.[19,20] This condition is likely less common than previously thought; in one study, it was found to account for 43.5% of all “epulis”-like lesions in dogs.[7] A more recent study has further classified the histopathology of these lesions, with FFH now representing 16% of epulis-like lesions.[21] The clinical appearance of FFH is not typical. It is usually a sessile lesion, smooth and pink, and neither inflamed nor ulcerated (Fig. 46.1C). It consists of dense fibrous connective tissue covered by stratified squamous epithelium.[1,7,19,22] This lesion was found to have a predilection for the rostral maxilla (57%).[21] If large enough, it can interfere with mastication.

Pyogenic granuloma

Pyogenic granuloma is a reactive lesion occurring at a specific site on the gingiva, as a result of a chronic low-grade irritation. In cats, the most common site is the mucogingival tissues of the mandibular first molar teeth, with traumatic contact of the maxillary fourth premolar tooth as the inciting cause.[23,24] It mainly consists of granulation tissue, rich in blood vessels, with a high-grade proliferation of endothelial cells and scarce collagen fibers. The superficial layers of epithelium are usually ulcerated and inflamed, and calcification has also been seen to occur.[1,4,7,8,19,22,25,26] Pyogenic granuloma is thought to evolve into a peripheral fibroma if left undisturbed, noted by an increase in fibrosis, a reduction of its vascular component and the presence of calcification.[27] Bacteriologic culture often reveals both *Staphylococcus aureus* and *Streptococcus hemolyticus*, which are believed to be contaminants. It is common for the initiating irritation to be a foreign body or small infected wound. With surgical removal of the lesions in conjunction with elimination of irritating local factors, recurrence is uncommon.[28]

Peripheral giant cell granuloma

This is a rare, well-defined lesion, more common in humans and cats than in dogs, characterized by a lobular architecture, where a fibroblastic connective tissue surrounds different areas rich in giant cells, similar to osteoclasts.[19,29] The lesions are covered by hyperkeratinized or parakeratinized epithelium, often ulcerated. Osteoid and bone formation may be present. Clinically, peripheral giant cell granulomas appear as vascular, lobulated gingival masses. A recent review of 2609 canine “epulides” submitted to a pathology service over an 11-year period revealed 26 cases, with a prevalence of 0.99%.[30] There is no breed, sex, or age predilection. Recurrence following marginal excision is common in the cat.[29]

Reactive exostosis

Reactive exostoses are focal lesions, likely of traumatic origin.[1,31] These lesions consist of compact or cancellous bone and appear as proliferations on the alveolar bone. They are of little or no clinical importance but can be confused with an osteoma; thus, biopsy is mandatory.

Therapeutic decision-making

Hyperplastic lesions on the gingiva should always be biopsied because they are often very difficult to diagnose based on clinical appearance. All lesions must be biopsied if multiple lesions are present. Diagnosis of some oral proliferative lesions may be made by contact smear or fine-needle aspirate of the mass; however, in general, an incisional biopsy is recommended for histopathological examination (see Chapter 42). The masses are usually accessible, and a specimen of appropriate size may be obtained with a biopsy punch or other suitable instrument. Diagnostic imaging of the involved jaw is mandatory to determine the presence and the extent of bone involvement (see Chapter 6).[20]

Gingival hyperplasia often results in pseudopocket formation. These should be differentiated from true periodontal pockets, which occur due to attachment loss associated with periodontitis. The base of a pseudopocket is normally located at the level of the cementoenamel junction and the hyperplastic soft tissue forms the walls of the pocket. Pseudopockets are removed by means of gingivectomy and gingivoplasty (see Chapter 20). These are excisional procedures that allow the restoration of the normal anatomy of the gingiva and facilitate oral hygiene.

In the case of drug-induced gingival enlargement, discontinuation of the drug may cause cessation and, possibly, regression of the gingival enlargement.[5,6] If possible, an attempt to substitute with an alternative drug is preferred. If the drug is not replaceable, frequent routine periodontal treatments, as well as meticulous home care, are imperative.[5,6] Topical chlorhexidine reduces plaque build-up and bacterial load, decreasing the inflammatory stimulus for gingival enlargement.[5,6] Systemic or topical folic acid has been demonstrated to decrease gingival hyperplasia.[32] Surgical treatment (gingivectomy/gingivoplasty) is indicated for severe cases and for cases where regression does not occur following discontinuation of the drug.[5,6]

For FFH, pyogenic granuloma and peripheral giant cell granuloma, wide surgical excision is usually curative. A gingivoplasty is performed afterward to reshape the gingival contour.

Postoperative care and assessment

It is generally not necessary to administer antibiotics, unless severe secondary infection is present (see Chapter 3). Chlorhexidine gluconate gel can be applied on the gingiva during the first days postoperatively, while daily tooth-brushing can be instituted as soon as the gingivectomy wounds have epithelialized (see Chapter 20).

Prognosis

Prognosis is usually good, as long as oral hygiene is maintained. Recheck examinations are to be scheduled every 3 to 6 months, combined with routine periodontal treatment as indicated. Long-term recurrence may occur, at all sites or at specific sites. In some cases, extraction of adjacent teeth may be indicated, combined with a repeat gingivectomy.[33] If periosteal reaction or exostosis is present, an osteoplasty and smoothing of the alveolar margin are indicated to prevent recurrence.

Traumatic buccal or sublingual mucosal hyperplasia

Pathophysiology

Self-induced oral trauma to the buccal or sublingual mucosa can result in acutely hemorrhagic mucosal lesions or, more commonly, chronic proliferative lesions (Fig. 46.2). Lesions on the buccal mucosa are frequently confused with calcinosis circumscripta or stomatitis. Sublingual lesions can be caused by excessive barking or panting, with resultant stretching of sublingual tissues over the occlusal surfaces of mandibular teeth and subsequent oral trauma.[34] Sublingual lesions can be confused with eosinophilic granulomas or ranulas. The diagnosis is based on clinical presentation and histopathology.

• **FIG. 46.2** Traumatic buccal mucosal hyperplasia in a dog. Source: (From Verstraete FJM. *Self-assessment Color Review of Veterinary Dentistry*. Ames: Iowa State University Press; 1999. With permission.)

Treatment

Full excision is not necessary unless previously biopsied lesions continue to be traumatized.[35] Excision of traumatized tissues can be performed in a fusiform pattern, ensuring no tissue remains in the occlusal path. Surgical closure is routine, using fine monofilament absorbable material. Prognosis is good if an adequate amount of tissue is excised.[34]

Eosinophilic granuloma complex in the cat

The eosinophilic granuloma complex in the cat consists of three clinical entities: (1) eosinophilic granuloma, (2) eosinophilic ulcer, and (3) eosinophilic plaque. Eosinophilic dermatitis has been described, and some authors have included it with the eosinophilic granuloma complex.[36,37] These lesions have been grouped together because they can occur simultaneously in the same animal and present many common aspects, such as occasional peripheral blood eosinophilia, an eosinophilic tissue infiltrate, and an association with various allergies. Only those lesions that may manifest in the oral cavity will be considered here.

Clinical presentation

Eosinophilic granuloma

An eosinophilic granuloma is a well-circumscribed, raised lesion, yellowish-pink in color, usually localized to the caudal aspect of the rear limb (where it may be linear), and is often bilateral. It may also be seen on the paws, cheek, lip commissure, chin, pinna of the ear, and in the oral cavity. Oral lesions can involve the hard palate, the soft palate, or the base of the tongue (Fig. 46.3A).[38–41] When oral lesions are present, dysphagia or ptyalism is usually seen. Large lesions may interfere with prehension and swallowing of food, and in very severe cases, they may even compromise breathing.

• **FIG. 46.3** Eosinophilic granuloma complex. (**A**) Eosinophilic granuloma of the tongue in a cat. (**B**) Severe eosinophilic ulcers of the upper lips in a cat. (**C**) Focal eosinophilic stomatitis in a Cavalier King Charles spaniel.

Eosinophilic granuloma has not been found to have a breed predilection in cats, but it is more commonly found in young animals (2–6 years), with a twofold higher incidence in females.[40–42] It has been associated with insect bite allergies (e.g., flea and mosquito bites), food allergy, atopy, immunosuppression, bacterial, parasitic (such as mange), and viral (calicivirus) infections.[38,42–48] It has been suggested that some eosinophilic granulomas affecting the oral cavity may be due to embedded insect parts, autoallergens (such as Fel d1 found in cat saliva), or even a heritable eosinophil dysregulation.[49,50] The etiology of oral eosinophilic granuloma is rarely determined and is often considered idiopathic.[45]

The histology of eosinophilic granuloma resembles a foreign body reaction, appearing as granulomatous inflammation with eosinophilic infiltrates surrounding collagen fibers (called flame figures). Reports have revealed that the collagen is not lysed or altered but rather is coated with the contents of degranulated eosinophils.[51,52] These lesions may resolve spontaneously or persist for a prolonged period. Eosinophilic granuloma may be an isolated lesion or manifest as part of the eosinophilic granuloma complex, associated with eosinophilic plaques or deep ulceration (so-called rodent ulcers).[45]

Eosinophilic ulcer

An eosinophilic ulcer is typically a well-circumscribed lesion, with raised edges and necrosis of the superficial layers, most frequently located on the upper lip at the philtrum or adjacent to the maxillary canine tooth (Fig. 46.3B). It can also occur anywhere on the skin or within the oral cavity. It starts as an erosive and exudative lesion, which may evolve into a major ulcer with swelling of the surrounding tissues. The lesions vary in size, depending on their chronicity, and are usually not painful or pruritic.[45] The eosinophilic ulcer has been found in cats of all ages and breeds, but with a higher incidence in middle-aged cats and in females.[42] It may be present as part of the eosinophilic granuloma complex or it may occur as a single lesion. The etiology of the eosinophilic ulcer may be traumatic, as seen in cases where the mandibular canine teeth contact the upper lip with a secondary bacterial infection and fibrosis. A number of eosinophilic ulcers may be secondary to flea allergy.[53]

The histological features of eosinophilic ulcer are often nonspecific. The predominant cell type can vary from a neutrophilic to an eosinophilic infiltrate, and bacterial colonization is common. The transformation of a chronic eosinophilic ulcer into a squamous cell carcinoma has been reported, although this is a rare occurrence.[38]

The differential diagnoses for oral eosinophilic lesions include gingivostomatitis, squamous cell carcinoma, other oral tumor types, trauma, food allergy, and atopy. Biopsy is useful to exclude the possibility of a malignancy. It is important to note that feline patients presenting with eosinophilic granuloma lesions are typically otherwise clinically healthy, and additional work-up is indicated in patients showing systemic signs.

Therapeutic decision-making

Medical treatment

To rule out food allergy and atopy, a restricted-ingredient diet can be used and skin testing performed. Thorough control of ectoparasites is essential. If there is no response to dietary change, or if there is evidence of secondary infection, then antibiotics (cephalexin 22 mg/kg BID) are a therapeutic option. Prolonged (4–6 weeks) treatment may be required before a response is seen.[40,41] In those cases where a skin test has identified allergens that could potentially be responsible for the lesions, it is important to control and, if possible, eliminate exposure, performing desensitization when possible.[38,40,41] This may be very difficult, if not impossible, to achieve.

For the many patients refractory to these measures and in cases where an underlying etiology could not be identified, corticosteroid treatment is indicated (prednisolone 1–2 mg/kg BID, gradually reducing the dosage and progressing to alternate day treatment when a response is seen).[43] Therapy should be continued until the lesions, or at least the signs, have completely disappeared.[45] An alternate therapy may include hydrocortisone aceponate applied to the skin lesions once daily for 7 days.[43] Corticosteroids may have profound side-effects (adrenocortical suppression, polyphagia, polydipsia, polyuria, obesity, and diabetes mellitus). It is best to use the minimum effective dose and avoid unnecessary prolonged or repeated treatment. Therefore, long-acting corticosteroids should be avoided. Treatment with megestrol acetate is also effective but is not recommended because of a high risk of side effects, including diabetes mellitus, mammary tumors, and uterine problems, and should be used only when other options have failed or cannot be used.[54]

Other therapeutic options that have been suggested include ascorbic acid, gold salts, interferon, chlorambucil, cyclosporine, and fatty acids (to improve skin barrier function), as well as intralesional administration of corticosteroids.[55]

Surgical treatment

Before performing any kind of surgery, it is recommended to biopsy the lesion to confirm the diagnosis of an eosinophilic lesion and eliminate other differential diagnoses. Anecdotal reports of lesional regression after returning the oral cavity to a healthy state (such as with extraction of teeth affected by tooth resorption or periodontitis) suggest that periodontal treatment is beneficial. Conventional surgical excision is indicated for very proliferative lesions that interfere with occlusion, breathing, or swallowing. Hemorrhage should be expected. This can be minimized by use of digital pressure and/or hemostatic agents. In general, electrocautery should not be used due to the high probability of collateral damage and increased rate of suture dehiscence. However, if electrocautery is used, caution is needed to avoid thermal injuries to the adjacent tissues. Cautious use of CO_2 laser tissue ablation will also minimize hemorrhage and is anecdotally reported to provide good results (see Chapter 10).[56] In the late 1970s and early 1980s, some authors obtained good results using cryotherapy, with little or no hemorrhage.[57–59]

Postoperative care and assessment

Postoperative pain control is fundamental to help get cats eating as soon as possible. In some cases, it may be necessary to provide postoperative alimentary support via a nasogastric tube. These cases should be hospitalized until they can eat unaided (see Chapter 5).

Complications

Wound dehiscence is a possibility, particularly if the soft palate is involved. Dehiscence is likely if there is excessive tension on the wound margins, when electrocautery has been overused, or if infection is present. Provision of easily swallowed food and daily use of an antiseptic mouth rinse (e.g., 0.12% chlorhexidine gluconate solution) for the first few days are helpful. The other main postoperative complications are hemorrhage, which is usually minor and easy to control, and local recurrence of the disease. Cases should be followed long-term to monitor for recurrence, particularly when the etiology has not been determined.

Prognosis

If the etiology has been determined and eliminated, recurrence is unlikely following appropriate medical or surgical therapy; however, idiopathic eosinophilic lesions tend to recur and require ongoing, sometimes lifelong, treatment. Concurrent diseases, particularly those that contraindicate the use of corticosteroids, worsen the prognosis.

Oral eosinophilic granuloma complex in the dog

Oral eosinophilic granuloma in siberian huskies

The oral eosinophilic granuloma lesions observed in this syndrome are usually found on the tongue: the lateral margins and the frenulum are involved, with raised, firm, yellow-pink lesions that may be ulcerated.[60,61] These lesions can also be seen on the palatal mucosa. Patients with lingual lesions are commonly presented for halitosis and oral discomfort, whereas the palatal mucosal lesions typically do not cause clinical signs.[8] The histopathological findings are similar to those seen in the feline eosinophilic granuloma complex, with granulomatous inflammation rich in eosinophils. The complete blood count reveals a peripheral eosinophilia in 80% of patients.[8] Eosinophilic granuloma is reported in young animals (1 to 7 years of age) and is most common in Siberian huskies, in which it is believed to be a hereditary or familial condition. Recommended treatment is the same as for cats (i.e., corticosteroids or surgical excision). Recurrence may occur.[60,61]

Eosinophilic stomatitis in cavalier king charles spaniels

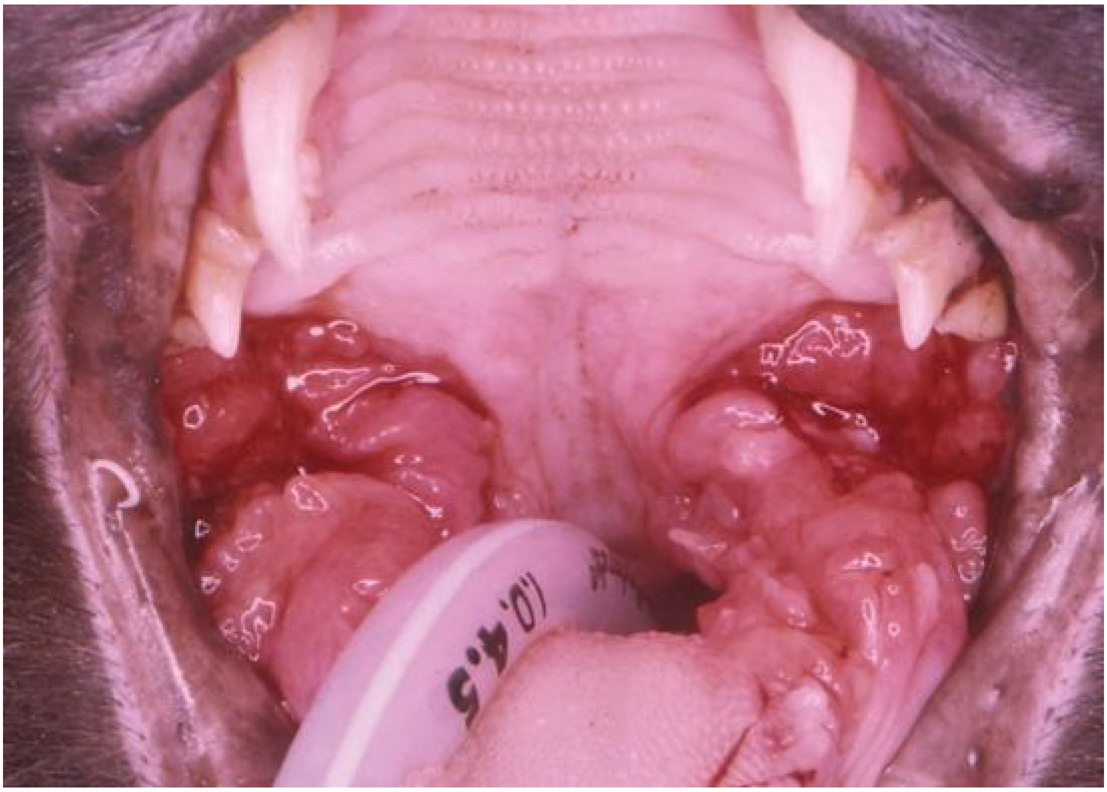
Cavalier King Charles spaniels and other dogs with ulcerative eosinophilic stomatitis are typically presented for dysphagia, pain on opening the mouth, paroxysmal coughing, or anorexia.[62] Oral findings include mucosal ulceration, plaque formation, and granulomatous lesions, although the latter is to a lesser extent than with the earlier mentioned eosinophilic granuloma lesions. Both entities are consistent with the spectrum of lesions found with eosinophilic granuloma complex in the cat (Fig. 46.3C). In very severe cases, oronasal fistula formation may occur.[63] The etiology of the lesions is unknown, although a familiar predisposition is often evident, both in Cavalier King Charles spaniels as well as other affected breeds.[62–65] Peripheral eosinophilia is occasionally seen.[66] A genetic predisposition to eosinophilic syndromes in general may exist, both in Cavalier King Charles spaniels as well as other breeds.[66] Fine-needle aspiration or exfoliative cytology may be performed instead of an incisional biopsy due to friability of the tissues, although histopathology is required for a definitive diagnosis. Care should be taken when performing an incisional biopsy of a lesion on the soft palate, as an oronasal fistula may result from a nonhealing biopsy site. Corticosteroids have been variably effective, although spontaneous regression may occur.[62,64,65] Due to the variability in the response to treatment, prognosis remains guarded.[65]

Chronic gingivostomatitis in the cat

An in-depth discussion of the etiology, pathophysiology, and medical aspects of chronic gingivostomatitis in cats is beyond the scope of this text. Only those aspects that are relevant to case selection for surgical treatment options of this condition are reviewed.

Clinical presentation

Feline chronic gingivostomatitis (FCGS), also called feline lymphocytic-plasmacytic stomatitis, is manifested by chronic inflammation of the gingiva and oral mucosa. No causative agent for this disease has yet been identified. Clinically, patients are presented with varying degrees of inflammation at the gingiva (gingivitis), alveolar and buccal mucosa (mucositis), the area lateral to the palatoglossal folds (often incorrectly referred to as "faucitis"), tongue (glossitis), palate (palatitis), or any combination thereof.[67] The lesions are often symmetrical. It is more common that the soft tissues in the premolar-molar area are affected than the tissues surrounding the incisor and canine teeth (Fig. 46.4).



• **FIG. 46.4** Severe proliferative lesions in a cat with chronic gingivostomatitis.

Affected cats are presented with oral pain and discomfort, dysphagia, ptyalism, and anorexia. Physical examination reveals a painful, inflamed mouth, and variable lymphadenopathy.

Preoperative concerns

A systematic approach should be followed in order to identify concurrent disease processes. Routine laboratory work, including a complete blood count, biochemical profile, and urinalysis, must be performed to rule out oral manifestations of systemic illness (e.g., renal failure and diabetes mellitus). Elevated serum gamma-immunoglobulins are commonly seen with FCGS.

Microorganisms such as *Bartonella henselae*, *Pasteurella multocida*, and calicivirus have been investigated, although a causal relationship has not been established.[68–74]

A detailed oral examination and full-mouth dental radiographs are indicated to diagnose concomitant periodontal disease and resorptive lesions. Root fragments are also commonly found in cats affected by FCGS and are thought to play an important role in perpetuating the disease if left in place (see Chapter 17).[75]

An incisional biopsy is routinely obtained. Histopathological findings typically include a diffuse, dense, mixed lymphocytic and plasmacytic infiltrate in the mucosa and submucosa. In some cases, the infiltrate is predominately plasmacytic, while a variable amount of neutrophils may be present.[76] Superficial ulceration is also common. The main reason for obtaining a biopsy, however, is to exclude the presence of squamous cell carcinoma, which may present as ulcerative lesion rather than a typical exophytic tumor. Conversely, FCGS may result in proliferative lesions that are clinically indistinguishable from squamous cell carcinoma, other tumor types such as fibrosarcoma, or nonneoplastic lesions such as eosinophilic granuloma.

Therapeutic decision-making

The findings of the comprehensive preoperative evaluation and the previous treatment history largely determine the choice of treatment.

Surgical management

The current surgical treatment involves removing periodontally compromised teeth and teeth affected with resorptive lesions and, hence, removing periopathogens. Since daily plaque removal has proven to be difficult and painful, removing the plaque-retentive surfaces by extracting the premolar and molar teeth or full-mouth extractions has shown the most repeatable results and is currently considered the standard of care. It has been demonstrated that following tooth extraction, 28.4% of cats are clinically cured and 30% have substantial clinical improvement if each tooth and its root is completely removed.[77] In the aforementioned study of 95 cats, there was no significant difference in response to extraction of the premolars and molars versus full mouth extractions. The earlier the teeth are extracted and the more steroid-naive the patients are at time of extraction, the better the outcome in most cases.[78]

Medical management for refractory cases is beyond the scope of this text.

Postoperative care and assessment

Preoperative and postoperative pain control is essential to ensure adequate nutritional intake as soon as possible, but in some cases, it is necessary to provide postoperative alimentary support via a nasogastric tube or an esophagostomy tube (see Chapters 4 and 5). These patients should ideally be hospitalized until they can eat on their own.

Prognosis

FCGS is a frustrating condition to treat as no single medical or surgical therapy has demonstrated a 100% response rate. Oral pain and discomfort must be treated. All underlying causes for oral inflammation should be identified and eliminated. Even following full-mouth extractions and the addition of various adjunct medical therapies, some patients (especially those who have already been subjected to many different medical treatments over long periods) fail to respond.

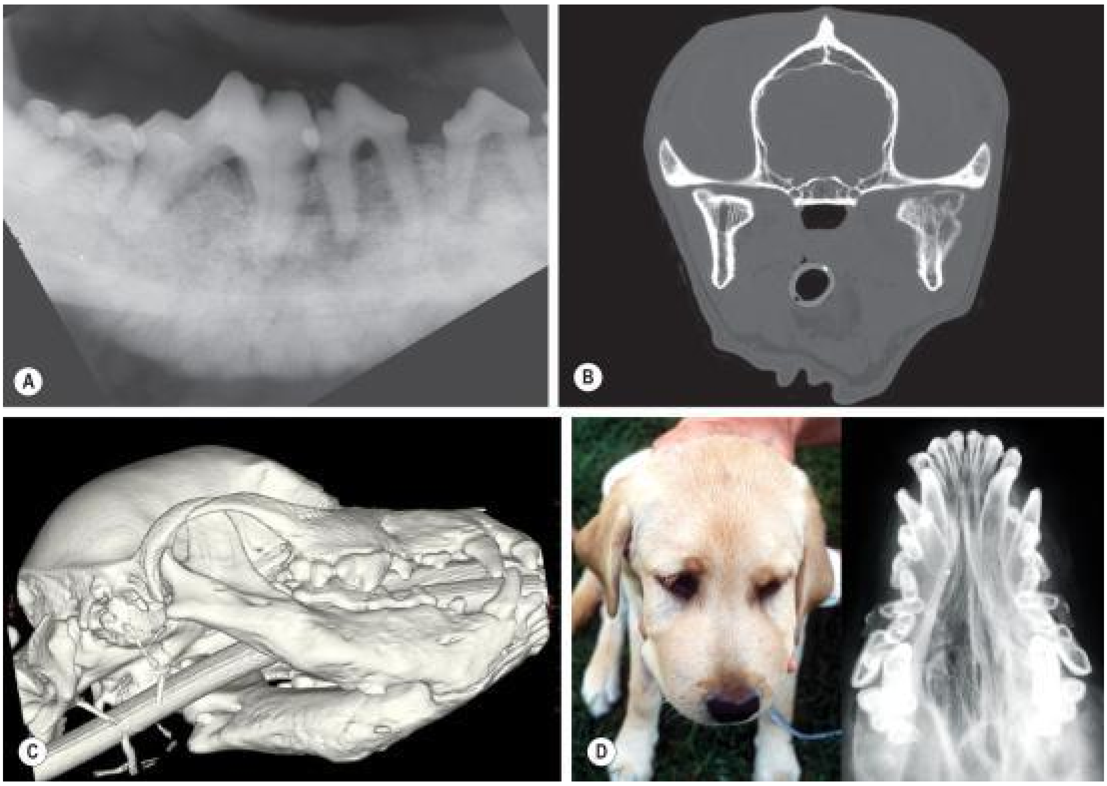
Nontraumatic diseases of the jaws

Disease syndromes

Osteomyelitis

Osteomyelitis can be classified by duration, etiology, and method of infection.[79] Chronic osteomyelitis usually results from inadequate treatment of acute osteomyelitis or may be an insidious process.[80] Osteomyelitis may be bacterial, fungal, or, rarely, viral in origin.[79,80] Most bone infections are of bacterial origin, and approximately half of them are monomicrobial.[79,81] *Staphylococcus* spp. are most commonly isolated. Other aerobic bacteria that may be involved include *Streptococcus* spp., *Escherichia coli*, *Pseudomonas* spp., *Proteus* spp., and *Klebsiella* spp.[79,81] Anaerobes isolated from osteomyelitis lesions include many species that are often found in the oral cavity, namely, *Actinomyces*, *Clostridium*, *Bacteroides*, and *Fusobacterium* spp.[82] Fungal osteomyelitis is often a component of a systemic mycosis.[81,83] The genera that can be involved include *Coccidioides*, *Blastomyces*, *Cryptococcus*, *Histoplasma*, and *Aspergillus*, and some of these are endemic in certain geographic regions.[83]

Osteomyelitis of the jaw most commonly occurs as a complication of trauma and fracture repair. Other sources of infection include extension of soft-tissue infection, including periodontal disease and periapical disease, and hematogenous spread. A low-grade osteomyelitis causing swelling of the mandible is commonly observed in cats and is typically associated with dental disease and retained root tips.[84] This is occasionally also seen in the dog (Fig. 46.5A). Feline patients can also be affected by alveolar bone expansion (i.e., buccal bone expansion). This most classically occurs buccal to the maxillary canines but has been seen to occur at the mandibular canine teeth and at premolar and molar teeth buccal alveolar bone. This alveolar bone expansion occurs in conjunction with periodontal inflammation and frequently with tooth resorption.[85] Fungal osteomyelitis most commonly occurs as a result of hematogenous spread of the causative organism, which gains entrance to the body by inhalation or through the gastrointestinal tract.



• **FIG. 46.5** Nontraumatic diseases of the jaws. (**A**) Radiograph showing osteomyelitis of the mandible associated with severe dental disease in a dog. (**B**) Computed tomography (CT) image showing fungal osteomyelitis of the right condylar process in a dog, who had a similar lesion in the left tibia. (**C**) Tridimensional CT image demonstrating craniomandibular osteopathy affecting both mandibles in a 5-month-old West Highland White terrier. (**D**) Clinical appearance (left) and skull radiograph (right) of an 8-month-old dog with renal secondary hyperparathyroidism Source: (From Verstraete FJM. *Self-assessment Color Review of Veterinary Dentistry*. Ames: Iowa State University Press; 1999. With permission.)

There is no apparent breed disposition for bacterial osteomyelitis, with the possible exception of the foxhound; a series of five related animals with severe osteomyelitis were documented in this breed.[86] Blastomycosis occurs more commonly in coonhounds, pointers, and Weimaraners, whereas a variety of breeds, including the pointer, have been identified to be more at risk for coccidioidomycosis.[83] Cats are more susceptible to histoplasmosis and cryptococcosis than dogs.[83] Osteomyelitis can occur with any of the aforementioned systemic mycoses; involvement of the jaw (Fig. 46.5B) would appear to be rare, although the actual incidence is unknown.[83,87]

Osteomyelitis may present as a firm, warm, and painful swelling of the part of the jaw involved. Draining tracts may be present. Animals with fungal osteomyelitis often have signs of systemic disease, which can range from nonspecific signs like inappetence, weight loss, lymphadenopathy, and fever to respiratory, ocular, cardiac, and nervous symptoms if those systems are involved.[80,83] Complete blood count and biochemical profile may be near normal or reflect the chronic infection and inflammation present. Negative serologic testing for coccidioidomycosis does not rule out the disease, as false-negative serology is common. A comprehensive examination including abdominal ultrasound and thoracic radiographs is indicated if systemic mycosis is suspected.[80,83]

The radiological signs of chronic osteomyelitis of the jaw include new bone production, lysis, and remodeling.[79] The periosteal new bone proliferation varies from smooth, lamellated, or spiculated.[81,88] Endosteal or periosteal scalloping of the cortex, or a moth-eaten pattern of bone destruction, is often evident. The presence of one or more sequestra is typical for osteomyelitis.[81,88]

An incisional biopsy, followed by histopathological evaluation and aerobic and anaerobic microbiological culture, is recommended to determine the exact nature of the lesion.[80] Specimens for bacterial and fungal culture must be collected and transported appropriately.[81]

The principles of treatment of chronic osteomyelitis of bacterial origin include surgical debridement, sequestrectomy, lavage, and administration of appropriate antimicrobial drugs based on culture and sensitivity.[79–81] Open drainage and cancellous bone grafting may be indicated in selected cases.[81] Osteomyelitis associated with dental disease is usually treated by extraction of diseased teeth and root fragments, if present; this is combined with curettage of the affected bone and followed by a therapeutic course of a suitable antibiotic (see Chapter 3). The treatment for fungal osteomyelitis is long-term antifungal therapy with a suitable agent, such as fluconazole; a large percent of animals may require life-long treatment.[80,81]

Craniomandibular osteopathy

Craniomandibular osteopathy (CMO) is an uncommon, noninflammatory, nonneoplastic proliferative bone disease that typically occurs in dogs of 3–6 months of age. It has been reported most often in West Highland white terriers.[89–92] The disease is inherited as an autosomal recessive trait in West Highland white terriers.[93] Affected dogs are typically presented for pain on opening the mouth and inappetence. Other signs include intermittent pyrexia, depression, and palpably enlarged mandibles.[92] The mandibles and tympanic bullae are most commonly affected.[94]

The diagnosis of CMO is based on the characteristic radiographic findings and confirmed by means of histopathological examination of a biopsy. On conventional radiographs or computed tomography, dense osseous proliferations are seen arising from the affected periosteal surfaces (Fig. 46.5C).[92] The histopathological findings include resorption of lamellar bone and replacement with exuberant, coarsely woven, poorly mineralized bony trabeculae, interspersed with fibrous tissue.[92] There are no consistent changes in the complete blood count, biochemical profile, and urinalysis, although the alkaline phosphatase may be elevated.[95]

The disease is usually self-limiting, with bone growth ceasing or even regressing at skeletal maturity.[92] Treatment is symptomatic and supportive and includes pain medication such as nonsteroidal antiinflammatory drugs and opioids as needed. Nutritional support is indicated in severe cases.

The prognosis depends of the degree of bony proliferation at the temporomandibular joints, the angular processes, and the tympanic bullae, which can limit the range of motion of the jaws (see Chapter 38).[92]

Idiopathic calvarial hyperostosis

Idiopathic calvarial hyperostosis is a nonneoplastic proliferative bone disease with clinical and histopathological characteristics similar to CMO.[96,97] It may affect the frontal, temporal, and occipital bones and has to date been described only in young bullmastiff dogs. The disease becomes symptomatic around 6 months of age and most cases are self-limiting, with bone growth ceasing and regressing at skeletal maturity.[97,98]

Mandibular periostitis ossificans

In the nonneoplastic proliferative bone diseases seen in dogs, mandibular periostitis ossificans (PO) is a relatively newly recognized disease entity. Large-breed puppies around 3–5 months of age are presented for swellings centered on the mandibular first molar tooth, around the time of its eruption. These patients are typically not systemically ill and do not experience pain on palpation of the swelling. There are no consistent changes on the complete labwork, but leukocytosis has been recognized. Physical exam reveals swellings that are typically firm ventrally with inconsistent fluctuance, either buccally or lingually. Local lymph node involvement is variable.[99]

The diagnosis of PO is based on the characteristic radiographic finding of a double cortex at the ventral aspect of the mandible. The anatomic ventral margin is separated from the additional, more ventral cortical layer by a radiolucent space containing fluid. The margins of the lesion typically extend from the third or fourth premolar tooth to beyond the third molar tooth. This double cortex formation is not limited to the ventral mandibular margin and can be seen on the lingual bone surfaces in some patients. The histopathological findings include severe, periosteal new bone formation and marked hyperostosis with areas of well-differentiated lamellar bone indicative of chronicity.

Mandibular periostitis is a proliferative bone disease that is usually self-limiting. Treatment includes debridement of fluid-filled lesions and symptomatic use of nonsteroidal antiinflammatory drugs. Antimicrobial therapy should be evidence based, utilized in cases with evidence of systemic (increased white blood cells) or local (necrotic bone) inflammation. Lamellar periosteal bone production at the ventral mandibular border may persist after clinical resolution.[99]

Hyperparathyroidism

Hyperparathyroidism can be primarily due to a functional adenoma or secondary to chronic renal insufficiency or nutritional deficiency.[94,100] Secondary hyperparathyroidism results in resorption of calcium from bone as the body attempts to maintain the calcium homeostasis. Nutritional secondary hyperparathyroidism can occur as a result of diets with a low calcium-to-phosphorous ratio, such as all-meat or organs, which contain adequate phosphorous but insufficient calcium.[100,101] This leads to a transient hypocalcemia, which stimulates the secretion of parathyroid hormone (PTH). In renal secondary hyperparathyroidism, the reabsorption of phosphate is impaired, which results in hyperphosphatemia. This in turn leads to a lower blood calcium concentration, which stimulates parathyroid gland activity.[94] The effect of PTH on bone include osteoclastic bone resorption and osteocyte-mediated release of calcium and phosphorous.[101] The osteoclasts are activated by osteoclast-activating factors released by the osteoblasts in response to PTH.[101] Concurrent with secondary hyperparathyroidism, the synthesis of 1,25-dihydroxyvitamin D in the kidney is also decreased due to the loss of functional nephrons.[94,101] This leads to a decrease in intestinal calcium absorption and impaired mineralization of osteoid. The combined effect of renal secondary hyperparathyroidism and the decreased 1,25-dihydroxyvitamin D is often referred to as *renal osteodystrophy*.[94,102,103]

The resultant bony changes are most likely to become clinically evident in growing dogs but rarely in cats.[94,104,105] The first bone to be affected is the mandible, followed by the maxilla, then the other bones of the skull, and finally the long bones. Before the rest of the skeleton is affected, the bones of the jaws may be severely demineralized and become swollen and malleable. This is evident clinically and is commonly referred to as *rubber jaw* (Fig. 46.5D). The occlusion can be grossly distorted and exfoliation of teeth may occur. Histologically, bone resorption and replacement with fibrous tissue are seen, which are often referred to as *osteitis fibrosa cystica.* In addition, osteomalacia, or poorly mineralized osteoid, may be seen.[106]

The radiological findings are often pathognomonic of the disease. The first radiographic sign is loss of the lamina dura, followed by loss of density of mandibular trabecular and cortical bone (Fig. 46.5D). Teeth may appear almost unsupported by bone.[94] In the cat, decreased density and thin cortices are seen in the long bones and vertebrae.[94]

Treatment depends on the cause of the hyperparathyroidism. For nutritional secondary hyperparathyroidism, the diet should be corrected, in which case remineralization of the bones usually occurs. Treatment of the renal form is based on the medical management of chronic renal failure. The skeletal lesions tend to be more severe with the renal form and therapy may be unrewarding.

Acromegaly

Acromegaly is a chronic endocrine disorder caused by excessive growth hormone (GH) secretion.[107–109] The underlying pathophysiology and clinical presentation of the disease are very different in the dog compared to the cat.

In the dog, acromegaly is rare.[107] The disease mainly occurs in middle-aged, female dogs either spontaneously during metestrus or while treated with progestagens.[107,109] The excess GH is secreted by foci of hyperplastic ductular epithelium of the mammary gland. In cats, the most common cause of acromegaly are GH-secreting tumors of the pituitary gland, similar to what is seen in human patients.[108] The disease is uncommon and mainly affects elderly, male cats. Acromegaly is associated with insulin-resistant diabetes mellitus, especially in the cat. The diagnosis is made by measuring GH concentrations in plasma. Pituitary tumors can be visualized using computed tomography or magnetic resonance imaging.[109]

Acromegaly is associated with enlargement of one or more organs.[108,109] Of importance in maxillofacial surgery is the soft tissue swelling of the face and tongue. The head has a heavy appearance and skin folds may be present. A relative mandibular prognathism and wide spacing of the incisors are often evident.[107–109] Hyperostosis of the skull and periosteal reaction on the mandible has been noted in cats.[108] Soft tissue swelling in the pharyngeal region may give rise to respiratory stridor.[109] The physical changes in cats tend to be less pronounced than in dogs.[109]

The diagnosis of acromegaly is based on clinical findings, plasma GH concentration, and diagnostic imaging. Biopsy of the maxillofacial soft tissues and bone is not indicated.

Acromegaly in the dog is treated by withdrawal of exogenous progestagens and/or ovariohysterectomy. Irradiation is currently the treatment of choice in cats. The maxillofacial soft-tissue features return to normal following treatment; bony changes are permanent but do not require treatment.[109]

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CHAPTER 47

Clinical behavior and management of odontogenic cysts

Frank J. M. Verstraete, Thomas P. Chamberlain

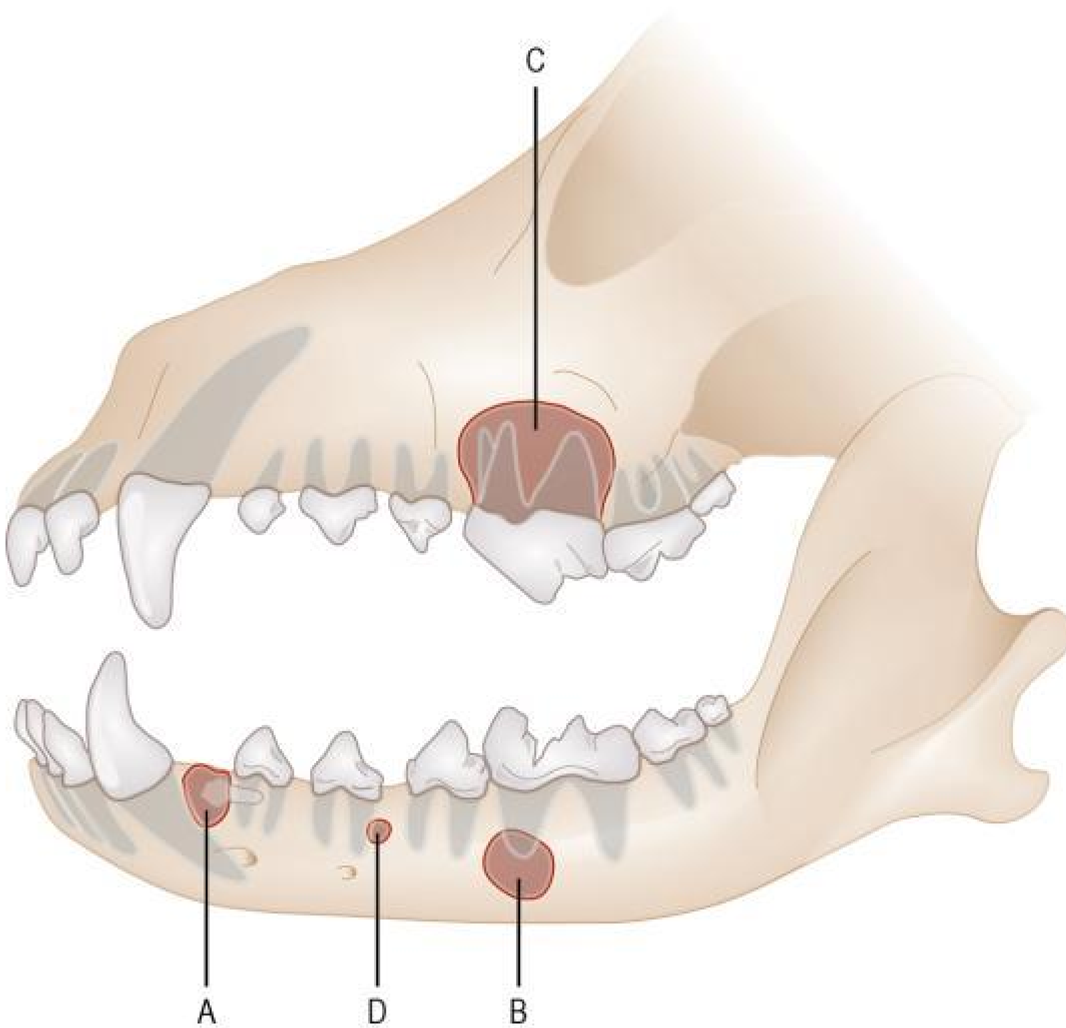
Introduction

Odontogenic cysts (OCs) are epithelium-lined structures that occur in the tooth-bearing areas of both jaws and are considered uncommon in domestic animals.[1-3] OCs reported in dogs and cats include dentigerous cysts (DCs), periapical (or radicular) cysts, lateral periodontal cysts, odontogenic keratocysts (OKCs), and canine odontogenic parakeratinized cysts (COPCs).[1,3-13] In humans, the majority of cystic jaw lesions are discovered incidentally on screening studies.[14] Some diagnostic confusion exists between the study of veterinary and human OCs because unified terminology and classification schemes do not exist.[1,3] OCs arise within islands of remnants of odontogenic epithelium (dental lamina, rests of Malassez) located in the periodontal ligament stroma.[1] Cystic lesions often have multilayered walls of epithelium that resemble ameloblastic epithelium in some areas.[1] Different OCs have variable clinical and biological behaviors.[3,15,16] Adequate treatment planning requires accurate histopathologic diagnosis by pathologists with training in odontogenic tumor and cyst pathology.[3,15,16] Any epithelial tumor may become cystic, as central degeneration of neoplastic epithelium occurs, and the converse is true in that malignant transformation of OC lining has also been reported.[1,12] Histopathology is the main modality for definitive diagnosis of OC, as immunochemistry and molecular diagnostic techniques are not yet clinically used.[17,18]

Dentigerous cyst

Pathophysiology

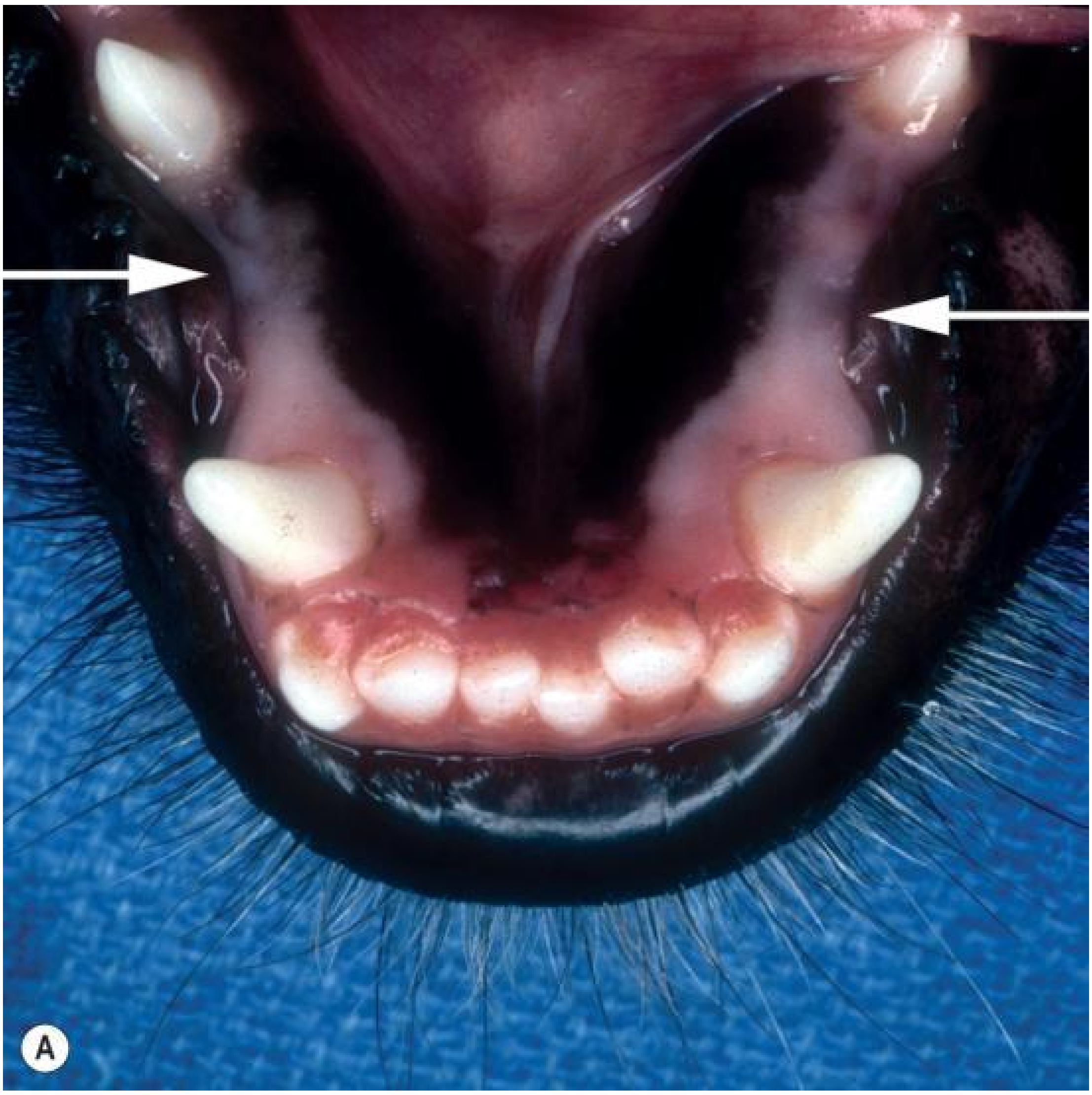
DCs are occasionally seen. They are the second most common type of OC in humans and the most prevalent OC in dogs.[1,3,10-12,18] They are rare in the cat.[11,13] By definition, DCs are associated with unerupted normal or malformed teeth and arise from proliferation of tissue remnants of the enamel organ or reduced enamel epithelium (Fig. 47.1).[3,18,19] As for other OCs, DC expansion occurs by common pathways involving passive osmotic fluid accumulation, proliferation of epithelial cells, and release of mediators of osteoclastic bone resorption.[18,20]



• **FIG. 47.1** Anatomical relationship between odontogenic cysts and teeth. (**A**) Dentigerous cyst. (**B**) Periapical cyst. (**C**) Canine odontogenic parakeratinized cyst. (**D**) Lateral periodontal cyst.

Clinical features

DCs should be a primary consideration for any oral swelling in an edentulous area of either jaw.[3,19,21] Age of initial diagnosis ranges between 6 months and 10 years; however, the highest frequency of DC is discovered between 2 and 3 years of age.[3] Although DCs are identified in a variety of breeds, the brachycephalic breeds are overrepresented.[3,21] The mandibular first premolar teeth are the most commonly affected teeth in dogs; however, DCs associated with unerupted maxillary and mandibular canine teeth are also observed.[3,21] As for humans, the most common location for DC is in the maxillary canine tooth and mandibular third molar tooth, which are the most commonly impacted teeth.[19,22] In the early stages of cyst formation, patients, both humans and dogs, are generally asymptomatic.[21,22] As the cyst enlarges, the mucosa overlying the cyst may appear slightly blue or purple (Fig. 47.2A). Asymmetry with tooth displacement, severe resorption of adjacent teeth, and pain are possible sequela of progressive cyst enlargement.[12,13,18,22] Fluid-filled osseous cyst walls may be palpable. DCs can become large lesions and expand the cortical bone of the maxilla or mandible.[21] Malignant transformation of the epithelial lining cells to primary osseous carcinoma and ameloblastoma has been reported in humans but appears to be extremely rare.[11,18,21] Because DCs are often discovered as incidental findings, the significance of frequent oral examinations and the routine use of full-mouth intraoral radiographs should be emphasized.[3,21]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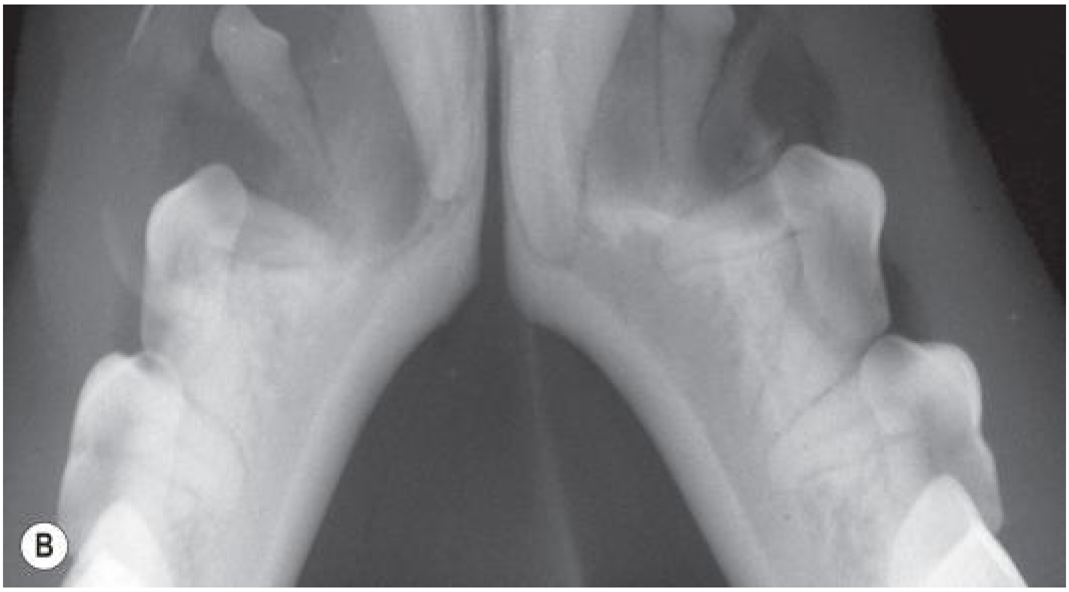


• FIG. 47.2 (**A**) Clinical and (**B**) radiographic appearance of bilateral dentigerous cysts associated with unerupted mandibular first premolar teeth in an 18-month-old Shih Tzu. Note the bluish appearance of the mucosa overlying the cyst cavities (*arrows* in A). Source: (From Verstraete FJM, Tsugawa AJ. *Self-Assessment Color Review of Veterinary Dentistry*. 2nd ed. Boca Raton: CRC Press; 2016.)

The radiologic features of DC are considered nearly pathognomonic (Fig. 47.2B).[3,21] The crown of the unerupted tooth will be enclosed by a radiolucent halo of variable size attached to the cementoenamel junction.[3,12,19,21,22] Although generally structureless, a thin, well-defined, radiopaque margin of cortical bone may be visible surrounding all or part of the radiolucent cyst.[3,13,18,19,21] Cysts are typically unilocular, and the unerupted tooth may be displaced.[18] Resorption of adjacent teeth may also be noted.[3,18,21] The cyst wall is composed of a thin layer of connective tissue and nonkeratinized stratified squamous epithelium of two to six cell layers thick and resembles reduced enamel epithelium.[3,18,22] Occasionally, secondary inflammatory cell infiltrates and epithelial hyperplasia may be observed.[3,18,22]

Management

Treatment involves a surgical approach using a mucogingival-periosteal flap to accomplish removal of the unerupted tooth, complete enucleation of the cyst wall, curettage, osteoplasty, and bone grafting if bone defects are extensive.[3,19,21] Prior to primary surgical closure, careful evaluation of the surgical site is mandatory, and submission of the entire cyst wall for thorough histopathologic examination and definitive diagnosis is necessary.[3,19,21] Incomplete removal of cystic epithelium may result in recurrence of the DC and/or neoplastic transformation.[3,18] Bone grafting may be required if the size of the DC compromises the integrity of the jaw.[3] Because these lesions are often asymptomatic for long periods of time, patients may easily be lost to follow up.[15] Follow up should continue for 2 years or until there is complete reossification of the cyst cavity.[15]

Periapical cyst

Pathophysiology

Although the periapical cyst (PC) or radicular cyst is the most common OC diagnosed in humans, they have been infrequently reported in dogs and cats.[3,5,7–9,21] By definition, PCs are strictly inflammatory cysts associated with a preexisting periapical granuloma of a nonvital tooth.[3,17,18,22,23] Periapical granulomas are the most common finding associated with failed endodontic treatment in humans.[18,22,23] Infected root canal content is the source of bacterial endotoxins and necrotic debris that stimulate a chronic inflammatory response in the periapical area.[24] PCs are believed to develop from endotoxin and immune stimulation of small odontogenic residues (epithelial rests of Malassez) within periapical tissues.[20,22-24] The "true" PC is completely enclosed by epithelium and attached by a cord of epithelium to the root apex, whereas the "pocket" PC has a cavity confluent with the root canal.[25] Cyst formation may represent the formation of a physical barrier that confines necrotic pulp contents to the root canal, thus preventing spread into the surrounding bone and other tissues.[24,26] Once remnant epithelial cells begin proliferating, there are several theories as to how PC formation actually occurs, and this may be genetically programmed.[24] At the center of the developing mass, cells eventually become necrotic. As the result of accumulating protein concentrations, osmotic pressure rises, and fluid ingress occurs as cavitation and passive cyst expansion occurs.[5,18,22,23,26] As the result of cellular degeneration, many proinflammatory cytokines, growth factors, and inflammatory mediators accumulate, promoting osteoclastic bone resorption and cyst expansion.[20,23,27]

Clinical features

A nonvital tooth is a prerequisite for PC formation, although many nonvital teeth are initially asymptomatic.[23] In dogs, few necrotic and/or infected teeth are associated with clinical symptoms recognizable by pet owners.[28] The only evidence of pulp necrosis may be intrinsic tooth staining.[28] In humans, most PCs are asymptomatic and discovered during routine dental radiographic examinations as round-to-ovoid, well-circumscribed, unilocular periapical lucencies that are contiguous with the lamina dura of the involved tooth.[14,23] Depending on the stage at diagnosis, some cysts in dogs may demonstrate relatively wide areas of radiolucency (Fig. 47.3A).[3,5,7] Radiographically, a PC cannot be distinguished from a granuloma.[14,23] With long-standing PC, resorption of the affected tooth as well as adjacent teeth may occur (Fig. 47.3B).[5,14] With periapical granulomas, external inflammatory resorption of the affected tooth may be seen; however, resorption of adjacent teeth would be unexpected.[23] Although computed tomography may have the ability to differentiate between periapical granulomas and cysts, the gold standard for definitive identification of a PC versus a granuloma is histopathologic assessment through serial sections of lesions removed in their entirety.[23,26,29,30] For the sake of academic completeness, the tooth may also be submitted for confirmation of pulp necrosis, if endodontic treatment is not elected.[5]

• **FIG. 47.3** (**A**) Occlusal and (**B**) lateral views of the right mandibular canine tooth. Note the resorption of the roots of the second premolar tooth *(arrow)*. This 9-year-old dog was presented for evaluation of a gingival mass at the right mandibular canine tooth, and the clients had no recollection of root canal treatment having been performed on the left mandibular canine tooth. Extraction was performed, and no bone graft was placed at the client's request. Histopathology confirmed the clinical diagnosis of periapical cyst. (**C**) Six months later, the dog was returned for a follow-up radiograph, which verified complete filling of the cyst cavity with new bone. Source: (From Lommer MJ. Diagnostic imaging in veterinary dental practice. Periapical cyst. *J Am Vet Med Assoc*. 2007;230:997–999.)

PCs are lined by nonkeratinized stratified squamous epithelium of variable thickness, and the underlying supportive connective tissue may be focally or diffusely infiltrated with a population of mixed inflammatory cells.[2,18,22,30,31] Plasma cell infiltrates, multinucleated foreign body-type giant cells, and dystrophic mineralization may also be observed.[23]

Management

As for all teeth with nonvital pulp, management by tooth extraction with curettage of the periapical region or an endodontic procedure should be performed.[23] If endodontic treatment is elected, initial treatment of inflammatory periapical lesions should be with conservative, nonsurgical measures.[26] In periapical pocket cysts, intracanal toxins can be eliminated by nonsurgical endodontic procedures, and these lesions may completely resolve.[20,23,26,29,30] Proliferation of periapical tissues will be inhibited by the removal of bacterial endotoxins, proinflammatory cytokines, inflammatory mediators, and cellular growth factors.[23,26] Although root canal treatment may remove endotoxins from canals associated with true PC, remaining cystic epithelium *(residual cysts)* still contains inflammatory mediators capable of sustaining or enlarging the cyst.[24-26] True PC must be treated by apicoectomy or extraction, because irritants cannot be removed nonsurgically.[31,32] Because many diseased teeth are asymptomatic to the owners for long periods of time, and significant complications of cyst expansion may occur, treatment follow-up should last until the complete ossification of the cyst cavity has occurred, i.e., at least 2 years (Fig 47.3C).[15,23] Failure to remove all of the epithelial cyst lining at the time of tooth extraction and periapical curettage may result in renewed epithelial proliferation and development of a residual cyst (cyst recurrence).[14,24] Any medication that can prevent release of inflammatory mediators and/or proinflammatory cytokines might be able to inhibit proliferation of epithelial cell rests in apical periodontitis, so administration of nonsteroidal antiinflammatory drugs may have beneficial effects in addition to postoperative analgesia.[26]

Odontogenic keratocyst, keratocystic odontogenic tumor, and canine odontogenic parakeratinized cyst

Pathophysiology

Although commonly encountered in humans, true odontogenic keratocyst (OKC) is rare in the dog and cat.[1,3,4,6,17,18] A recent study of OCs in dogs identified 9 out of 41 OCs that had histopathologic features that were suggestive, but not diagnostic, of the human OKC.[3] These were identified as the second most common type of cyst encountered in this species, and the term *canine odontogenic parakeratinized cyst* (COPC) was proposed.[3] The OKC was originally classified as a developmental cyst originating from dental lamina remnants in the maxilla or mandible.[18,22] OKCs are characterized by aggressive and destructive behavior with a marked propensity to recur.[18] Because they often appear as ordinary cysts, they may be underdiagnosed and undertreated, leading to unnecessary recurrences.[33] Increasing evidence supports the theory that OKC development may be gene mutations leading to overexpression of certain proteins.[18] Studies of OC and OKC indicate that cyst expansion and bone resorption are mediated by the common mechanisms of passive osmosis and cellular production of cytokines.[16] Markers of biologic proliferation indicate that the OKC should be considered as a cystic odontogenic tumor rather than merely a developmental cyst, hence the name change to *keratocystic odontogenic tumor* during 2005–2017.[18,22,34,35] However, the authoritative and most recent WHO Classification of Head and Neck Tumors reversed that decision and the OKC is again the recommended term for this well-recognized disease entity in humans, although this issue remains controversial and largely academic.[18,36,37]

Histopathology

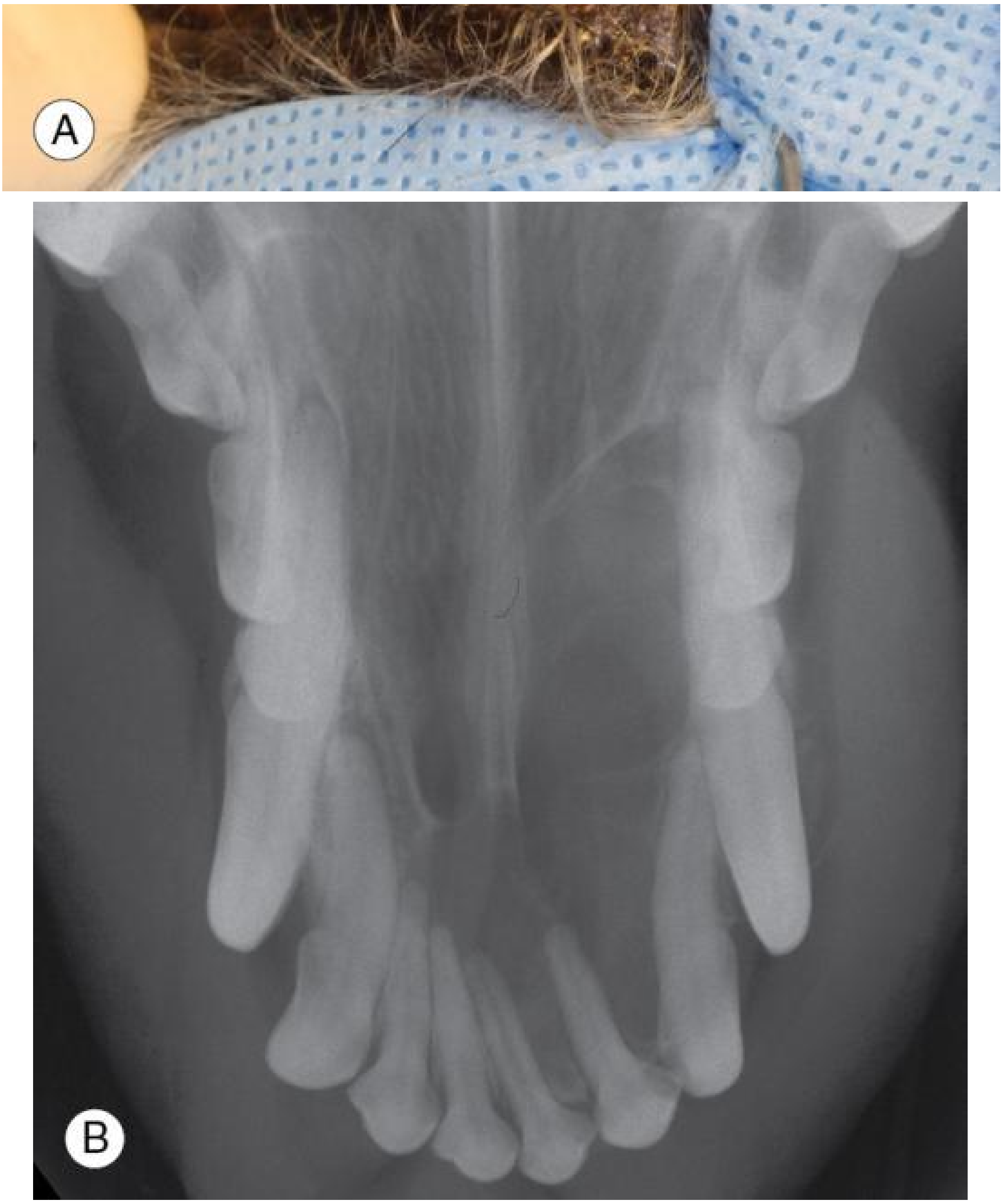
Histopathologic assessment remains the mainstay for diagnosis of OKC.[17,18,35] The microscopic feature of parakeratotic keratinization of the cyst lining is highly characteristic.[16–18,35] The cyst lining is composed of a thin connective tissue periphery supporting a stratified squamous layer of 6–10-cell thickness.[16–18,35] The basal layer consists of well-developed, palisaded, cuboidal, or columnar cells with a higher mitotic index and intensely stained nuclei.[16–18,35] Budding of the basal layer and daughter, microcyst formation may be encountered.[17,18] The luminal surface cells are parakeratinized and refractile and form a corrugated rim.[17,18] The lumen may contain variable amounts of desquamated keratin.[38] If secondary inflammation occurs, an inflammatory cell infiltrate and hyperplasia may ensue, disrupting the characteristic appearance.[17,35] Therefore submitting large areas or the entirety of the cyst for histopathologic assessment is important.[17]

COPC has some histologic features suggestive of OKC, namely a cyst wall consisting of nonkeratinizing stratified squamous epithelium of uniform thickness but with a parakeratotic surface and a flat epithelial-connective tissue interface. COPC, however, lacks the hyperchromatic, palisading basal cell layer and keratinaceous debris in the cyst lumen characteristic of OKC.[3,39]

Clinical features

OKC in humans may occur in any position of either jaw, and multiple cysts may be present; however, they affect most commonly the mandible in an approximate 2:1 ratio.[18] They may be associated with unerupted teeth.[18]

In dogs, all cases of COPC described to date occurred on the maxilla.[3,39] There is no specific age for presentation, and miniature schnauzers are the most commonly represented breed.[3] Patients may be asymptomatic up to the point where facial distortion (Fig. 47.4A) and discomfort become obvious as a result of cyst expansion. COPCs are not associated with unerupted teeth. Extensive maxillary COPC may lead to exophthalmos and palatine, maxillary, and zygomatic bone erosion.[39] Upon surgical exploration, a fluid-filled cystic cavity is identified below the gingiva and mucosa. On diagnostic imaging, COPC, similar to OKC, may present as a well-circumscribed unilocular or multilocular radiolucency with smooth or scalloped margins (Fig. 47.4B–D).[3,14,18,38] Although displacement of adjacent teeth may occur, only rarely is there root resorption.[40]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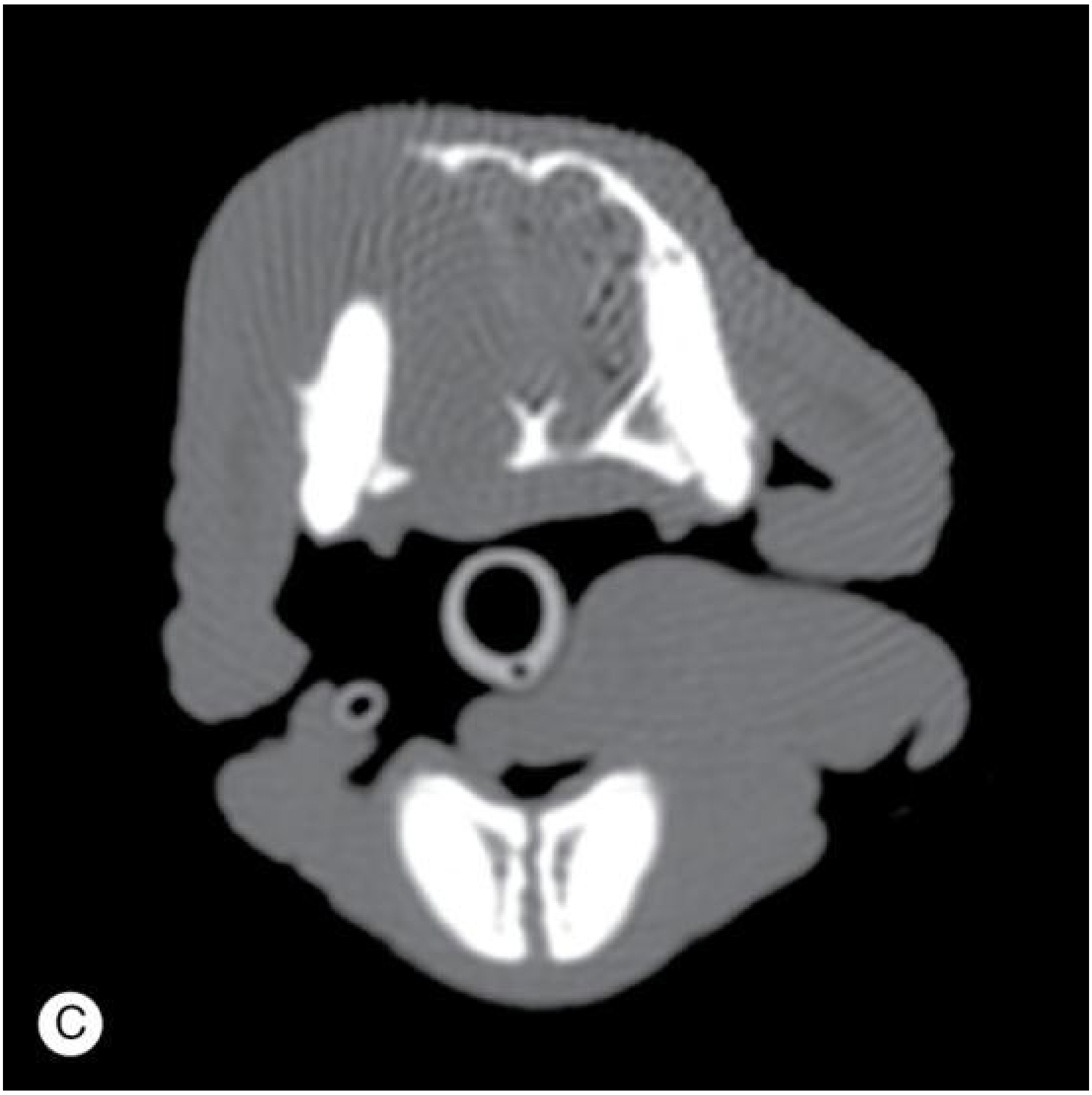
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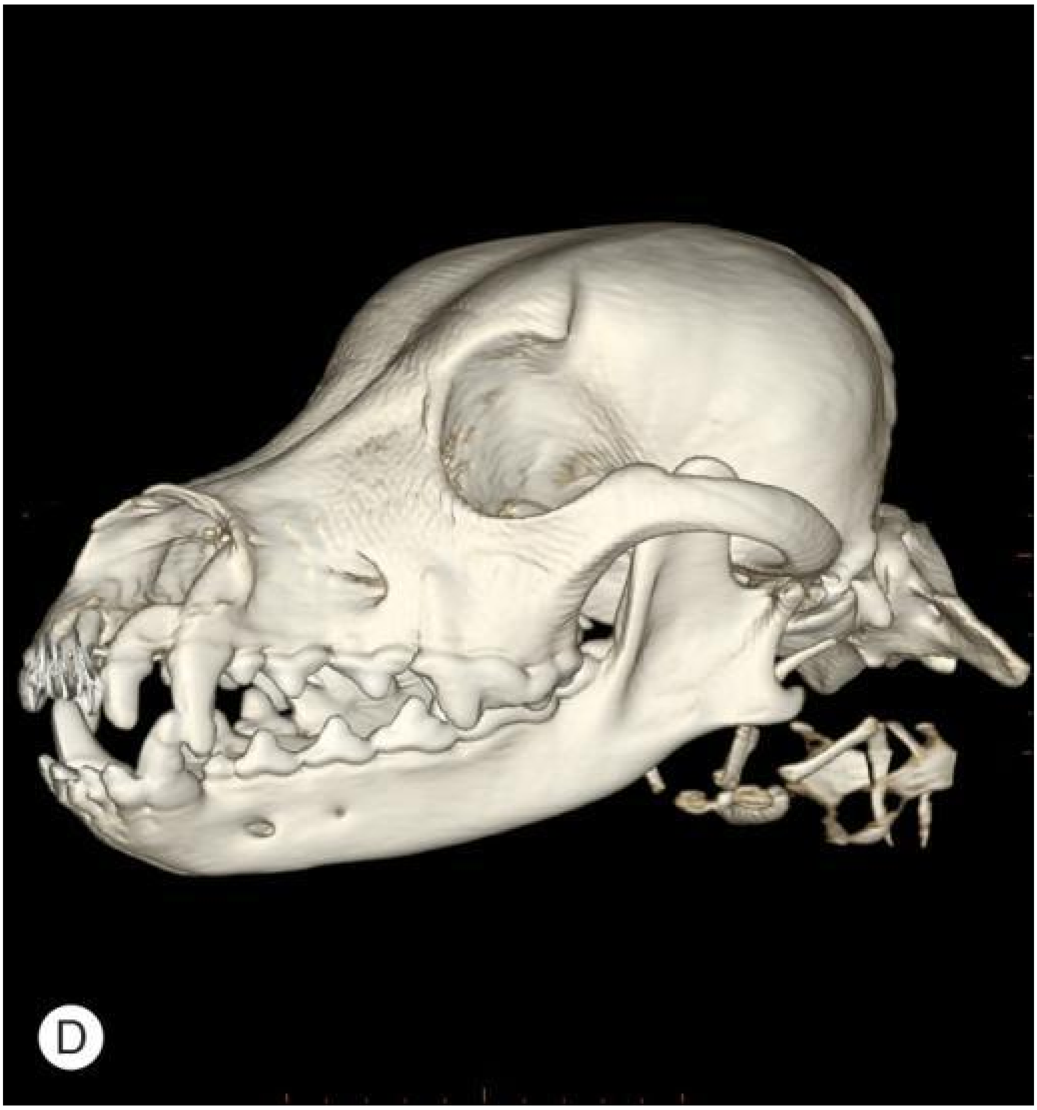

• FIG. 47.4 (**A**) Clinical (the dog is positioned in dorsal recumbency) and (**B**) radiographic appearance of a canine odontogenic parakeratinized cyst involving the rostral portion of the left maxilla in a 4-year-old Miniature Schnauzer, along with a (**C**) cross-sectional computed tomographic image at the level of the lesion and (**D**) three-dimensional reconstruction of the skull. Source: (From Verstraete FJM, Zin BP, Kass PH, et al. Clinical signs and histologic findings in dogs with odontogenic cysts: 41 cases (1995–2010). *J Am Vet Med Assoc*. 2011;239:1470–1476.)

Management

The treatment of OKC in humans remains controversial, as there is no consensus on what is appropriate.[18,41] Treatment goals are focused on prevention of recurrence and minimization of surgical morbidity.[41] Conservative management includes simple enucleation (with or without curettage), marsupialization, or decompression and is associated with a high rate of recurrence.[41] Surgical excision with peripheral osseous curettage or ostectomy is currently the preferred method of treatment in humans.[18] The more aggressive surgical approach is justified by the high recurrence rate, ranging from 10% to 30%, and increasing the longer the follow-up period lasts.[18,41] The friable, thin wall of the cyst may lead to incomplete removal.[18]

In the study involving nine COPC lesions in dogs, treatment was similar to that performed for DCs, namely a partial maxillectomy with thorough curettage of the peripheral tissues; in the long-term follow-up period, recurrence was not observed.[3]

Lateral periodontal cyst

Lateral periodontal cysts are developmental, noninflammatory cysts that occur on the lateral aspect or between the roots of vital erupted teeth.[18,22] They are typically asymptomatic and incidental findings in humans.[18,22] Enucleation or local excision is curative.[18,22]

Two cases have been described in the dog. The first was an incidental finding in a mandibulectomy specimen; mandibulectomy was performed for treatment of a squamous cell carcinoma.[3] The second case was also an incidental finding, with the cyst being located apical to a gingival canine acanthomatous ameloblastoma.[42]

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CHAPTER 48

Principles of oral oncologic surgery

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Introduction

The most common malignant oral tumors of dogs are malignant melanoma, squamous cell carcinoma, fibrosarcoma, and osteosarcoma.[1] In addition, canine acanthomatous ameloblastoma is a common benign but locally invasive neoplasm. Squamous cell carcinoma represents approximately 70% of feline oral tumors, with fibrosarcoma and osteosarcoma occurring less commonly.[2] Prior to 1980, most oral tumors in dogs and cats were treated conservatively, and local recurrence rates were high.[3–7] Because recurrent tumors are more difficult to treat, and because many clients are not inclined to pursue further treatment after an initial failed excision, local recurrence is often a life-threatening event.[3,8] Indeed, as more aggressive surgical approaches have become more common, survival times have improved.[9–13] Therefore, the single most important principle in oral oncologic surgery is that the best chance for local tumor control is at the time of the first attempt at excision.[8]

Whereas benign, well-circumscribed lesions such as cysts and odontomas may be treated conservatively with enucleation or curettage techniques, malignant oral tumors and benign but locally invasive tumors (such as canine acanthomatous ameloblastoma) require more aggressive surgical treatment, typically involving removal of a portion of the maxilla or mandible.[14]

Because of the differences in biologic behavior of different tumor types and the variation in clinical presentation, clinical staging should be performed prior to formulating a treatment plan (see Chapter 42).[15]

Diagnostic imaging

Advanced imaging is essential to determine the extent of the tumor and the presence of bone involvement. Ideally, computed tomography (CT), with and without contrast, is performed prior to biopsy, as part of the clinical staging process. If CT is not available, intraoral dental radiographs are a useful alternative, although it is important to remember that loss of approximately 40% of bone density is required for radiographic detection, so radiographs tend to underestimate bony changes.[1,16] CT more accurately determines the extent of bone involvement and invasion of adjacent structures (such as the nasal cavity and periorbital region for maxillary tumors or the sublingual soft tissues for mandibular tumors).[17] Magnetic resonance imaging and ultrasound can be useful for visualizing tumors with deep soft tissue infiltration and for noninvasive evaluation of regional lymph nodes. Further discussion of diagnostic imaging is detailed in Chapter 6.

Nomenclature

Communication between the veterinary dentist, surgeon, oncologist, and clients is facilitated when the proper terminology is employed. In the veterinary literature, it is common to see terms such as "premaxillectomy" and "hemimandibulectomy," which can lead to confusion. The "premaxilla" is a misnomer for the incisive bone,[18] which contains the incisor teeth. The term "incisivectomy" is therefore more appropriate to describe a surgical procedure involving removal of one or both incisive bones. The term "hemimaxillectomy" is equally confusing, as it is commonly used to describe the surgical excision of one maxilla. Removing all or most of one maxillary bone (typically combined with the excision of all or parts of the incisive and palatine bones) is a "total unilateral maxillectomy." Various types of "partial maxillectomy" can be performed, including a "unilateral rostral maxillectomy" (removal of one incisive bone and the most rostral part of the maxillary bone), "bilateral rostral maxillectomy" (removal of both incisive bones and the most rostral portions of both maxillary bones), "central maxillectomy" (removal of the midportion of the maxilla, containing the rostral premolar teeth), or "caudal maxillectomy" (removal of the portion of the maxilla containing the fourth premolar and/or molar teeth).[14] In addition, for treatment of extensive tumors, bilateral rostral maxillectomy may be combined with a "nasal planectomy" (excision of the soft tissues and cartilages comprising the facial part of the nose),[19,20] while caudal maxillectomy may be combined with an "orbitectomy" (removal of portions of bones that comprise the orbit including the palatine, zygomatic, lacrimal, and frontal bones).[21]

Mandibulectomy procedures are similarly classified according to the part of the mandible that is removed. A "rim excision" is defined as a partial segmental excision, leaving the ventral border of the mandible intact.[15,22–25] Rim excision has also been referred to as "marginal mandibulectomy"[15] and "crescentic osteotomy."[26] The term "marginal mandibulectomy" refers to the excision of the alveolar margin but is confusing as "marginal excision" has a specific connotation in terms of surgical margin (discussed in Surgical Excision, later in this chapter). In this context, a rim excision is a wide excision, not a marginal excision. "Unilateral rostral mandibulectomy" refers to removal of one rostral mandible, containing the three incisor, canine, and first and second premolar teeth. "Bilateral rostral mandibulectomy" (removal of the rostral parts of both mandibles following osteotomy between the second and third premolar teeth) is more commonly performed. If necessary, the osteotomy can be performed as far caudally as between the fourth premolar and first molar teeth.[27] In a "segmental mandibulectomy," part of the body of the mandible is excised, typically including the caudal premolar and one or more molar teeth. A "caudal mandibulectomy" involves excision of the ramus of the mandible, including the condylar and coronoid processes.[14] Although the term "hemimandibulectomy" is often used in the veterinary literature to denote the complete excision of one of the two mandibles, the term "total unilateral mandibulectomy" is more appropriate.

Surgical treatment objectives

Once the histopathologic nature and clinical stage of the tumor have been determined, a decision must be made with regard to surgical treatment. The type and extent of surgical procedure are based on clinical and radiographic findings and the propensity of the specific tumor type for local invasion and metastasis.[28] There are four potential goals of oral oncologic surgery: (1) surgery to cure, (2) surgery for cytoreduction ("debulking"), (3) surgery for local control, and (4) surgery for palliation. The therapeutic goal of a "curative" surgical procedure is to remove the entire lesion and leave no cells that could proliferate and cause persistence of the lesion.[29] Surgical excision is considered curative for benign, locally aggressive tumors such as acanthomatous ameloblastoma. It is also possible to achieve cure of malignant tumors (such as squamous cell carcinomas in dogs) if metastasis has not already occurred at the time of the procedure, if surgical margins are tumor-free, and if the tumor is minimally handled during surgery to avoid possible seeding of the area with neoplastic cells.[30] Cytoreduction, which leaves macroscopic tumor behind, may be performed prior to radiation therapy for large tumors that cannot be completely excised. However, adjuvant treatment is generally less effective when macroscopically visible tumor remains than when residual neoplasia is microscopic.[8] Surgery for local control is typically the goal for patients with malignant oral tumors such as melanoma, where the potential for early metastasis is high and where adjunctive therapy may be performed following surgical excision of the primary tumor. However, some oral cancers cannot be eliminated by any treatment modality. In these cases, surgery for palliation may be undertaken to temporarily relieve pain, improve function, and increase quality of life.[28]

Treatment planning

Following determination of the surgical objective, an appropriate treatment plan can be formulated. Whenever possible, the decision-making process should include a team consisting of a veterinary oral surgeon, medical oncologist, and radiation oncologist,[14] in addition to the primary care veterinarian. The client should be informed of the various treatment options and their associated expectations with regard to the duration and expense of treatment, prognosis, possible complications, and postoperative appearance and function. Postoperative and follow-up photographs of previous patients should be provided to clients to give them a realistic expectation of their own pet's postoperative appearance.[8] For patients undergoing caudal or segmental mandibulectomy, the likelihood of a permanent malocclusion postoperatively and the potential need for long-term management should be discussed, as well as the availability of regenerative reconstructive techniques, which could allow replacement of the missing section of mandible, preventing postoperative malocclusion (see Chapter 53).[31,32]

The use of volume-rendering software to create a three-dimensional (3D) image following a CT scan can be very helpful in treatment planning, and a 3D printed model of the patient's skull is an excellent tool for surgical planning and client communication.[33]

Treatment planning must include the following:

- appropriate anesthesia and analgesia;
- adequate surgical margins;
- patient positioning for optimum visibility;

- prevention of damage to adjacent teeth; if damage occurs, address appropriately;
- preservation of local blood and nerve supply: atraumatic surgical technique and minimal or no use of electrocautery;
- tension-free closure; and
- anticipation and prevention of complications: hemorrhage, infection, dehiscence, dysfunction.

Anesthesia and analgesia

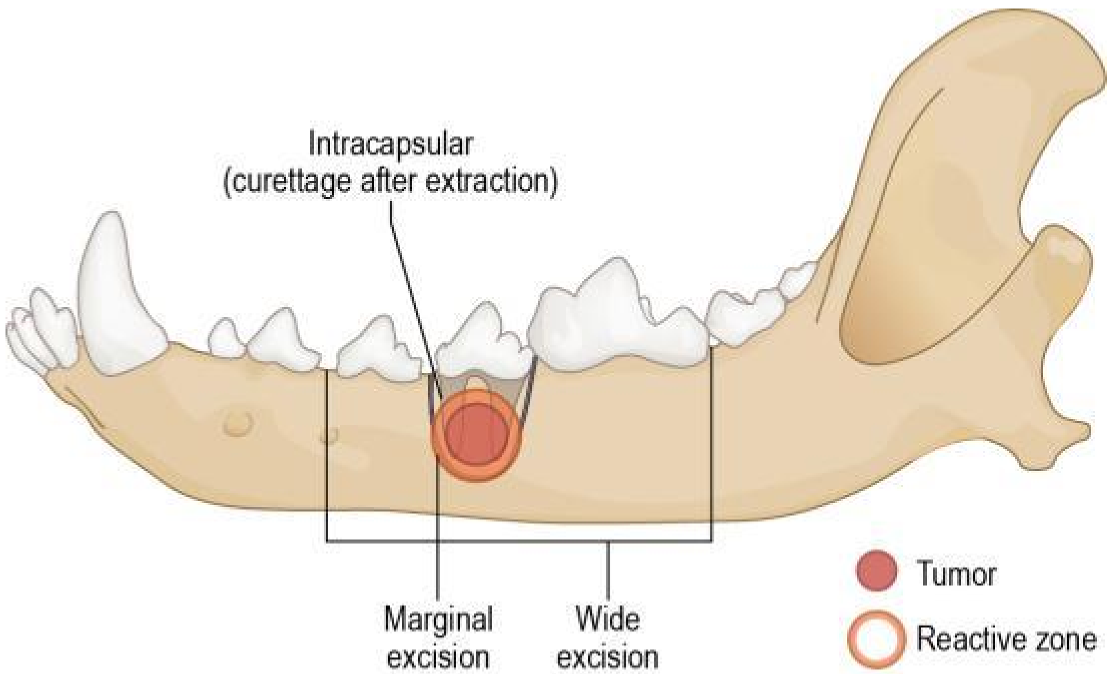
Anesthesia and pain management are discussed in detail in Chapter 4. A multimodal approach to anesthesia and analgesia is indicated for oral oncologic surgery. The perioperative use of opioids,[34–38] nonsteroidal antiinflammatory agents,[39] ketamine infusions,[40–43] local or regional anesthesia,[44–46] and/or alpha-2 adrenergic agonists,[36,47,48] in combination with inhaled anesthetics, should be considered for patients undergoing maxillectomy or mandibulectomy.

Surgical excision

Four types of excision have been described (Fig. 48.1)[8,28,49,50]:

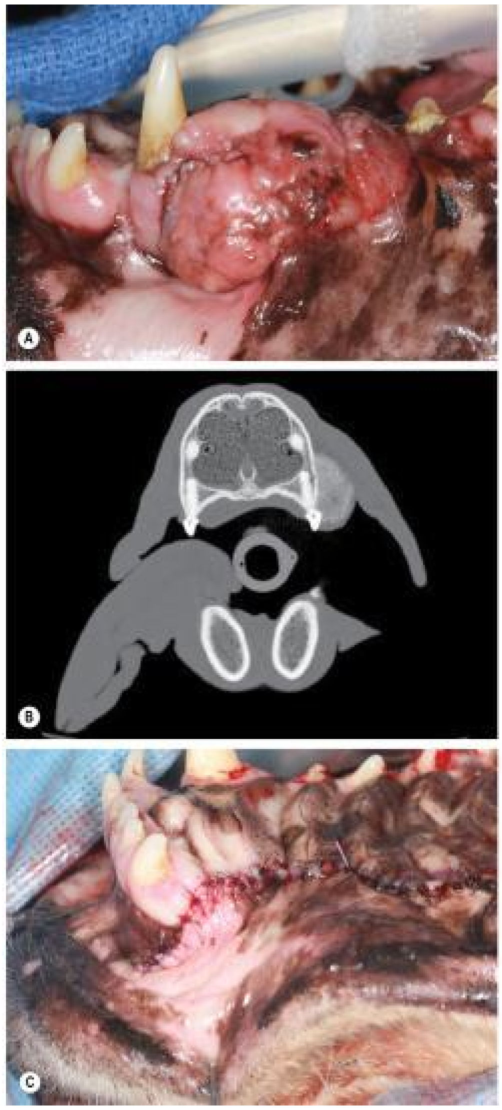
- *Intracapsular excision:* A "debulking" procedure that leaves behind macroscopically visible tumor. Local persistence will happen unless surgery is followed by radiation therapy. This type of excision is acceptable for well-differentiated, benign tumors such as odontomas.[14] Debulking, also called cytoreduction, may also be performed prior to radiation therapy, or as palliative therapy for tumors which are too large to be completely excised (Fig. 48.2).
- *Marginal excision*: Excision immediately outside the pseudocapsule of the tumor, within the reactive zone (which consists mostly of inflammatory cells). Some neoplastic cells may remain visible microscopically. This type of excision may be appropriate for a well-differentiated benign tumor such as peripheral odontogenic fibroma, depending on behavior (e.g., growth), diagnostic imaging findings, and anatomical location (Fig. 48.3).[14] If marginal excision is performed on a malignant tumor, recurrence (or persistence) is likely without adjuvant therapy.
- *Wide excision*: Removal of the tumor, its pseudocapsule, and the reactive zone, with complete margins of normal tissue on all aspects. Partial mandibulectomy and partial maxillectomy (Fig. 48.4) are examples of wide excision. Although the goal with wide excision is local cure, recurrence (or persistence) is still possible. In human oral cancer patients, margins of less than 5 mm were associated with a 26.4% recurrence rate, while 3.4% of tumors removed with margins ≥5 mm recurred.[51] In general, a margin of 10 mm of normal tissue is recommended for most malignant tumors.[8,14,52] However, some aggressively infiltrative tumor types, such as fibrosarcoma, may require much wider margins to achieve surgical cure,[52] and even with a goal of 20 mm margins, curative intent surgery in dogs with oral fibrosarcomas resulted in recurrence in 54.2% of cases.[29] For any oral tumor removed by wide excision, clients should be informed that recurrence, although unlikely, is possible even if margins appear histologically clean.

• *Radical excision* or *resection:* Removal of the entire anatomical structure or compartment containing the tumor. It is questionable whether a total unilateral mandibulectomy meets the definition of a radical excision or whether it is a very wide excision.

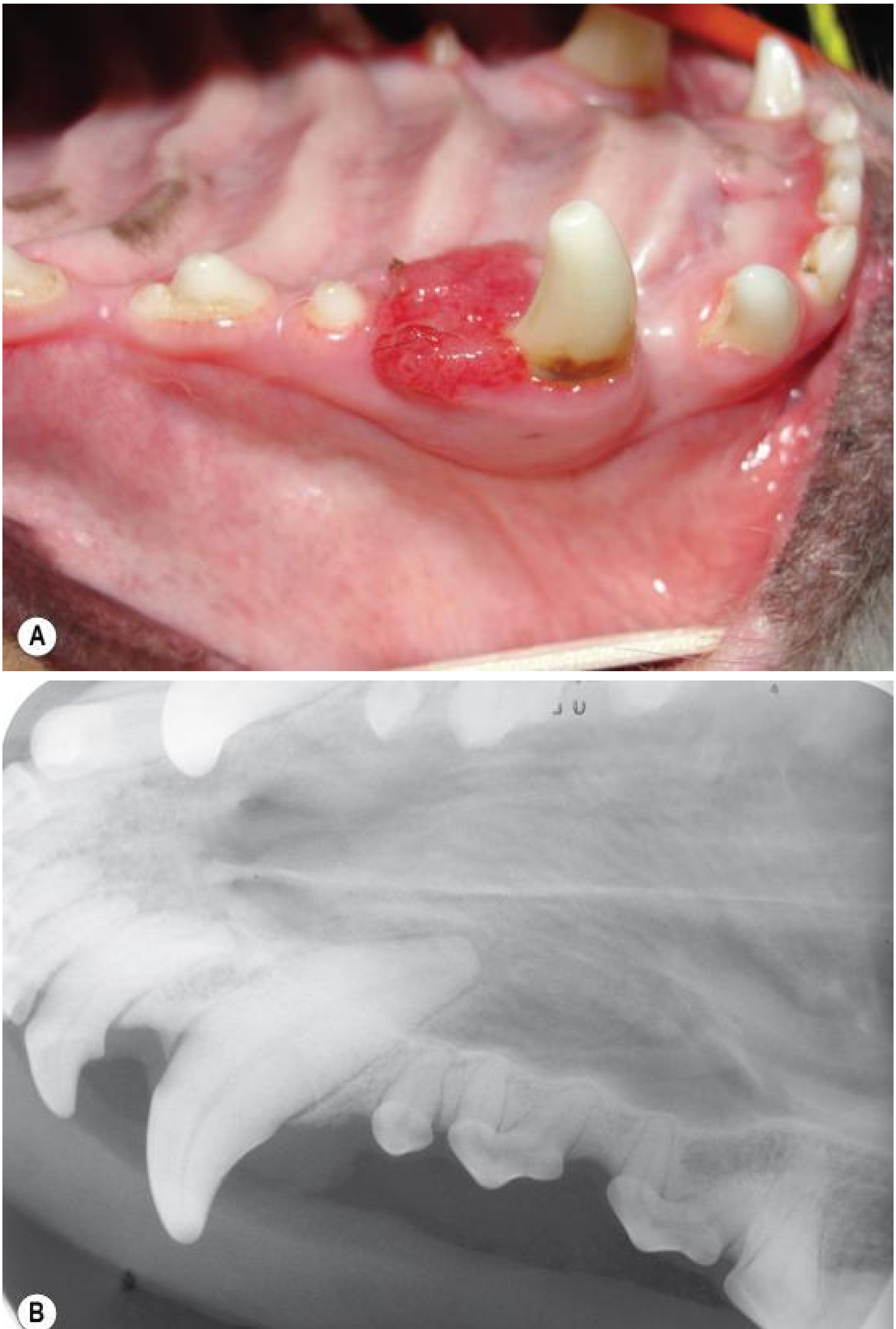


• FIG. 48.1 Diagrammatic representation of surgical resection margins. Intracapsular excision may be appropriate for benign, well-differentiated lesions such as odontoma. Marginal excision, immediately outside the pseudocapsule of the tumor, may be appropriate for a well-differentiated benign tumor such as peripheral odontogenic fibroma.[14] Segmental mandibulectomy is an example of wide excision, which involves removal of the tumor, its pseudocapsule, and the reactive zone, with complete margins of normal tissue on all aspects.

• **FIG. 48.2** (**A**) Clinical and (**B**) radiographic appearance of a malignant melanoma in a 14-year-old dog. (**C**) Debulking (intracapsular excision) was performed for palliative treatment. The patient is positioned in dorsal recumbency.



• **FIG. 48.3** (**A**) Clinical and (**B**) computed tomographic appearance of a slow-growing, ossifying fibroma at the right rostral maxilla of an 12-year-old dog. (**C**) A marginal excision was performed to achieve surgical cure. The patient is positioned in dorsal recumbency.


A
B

• FIG. 48.4 (**A**) Clinical and (**B**) radiographic appearance of an acanthomatous ameloblastoma at the left maxillary canine tooth of a 3-year-old dog. (**C**) Unilateral rostral maxillectomy (wide excision) was performed to achieve surgical cure. The patient is positioned in dorsal recumbency. (**D**) The excised section of maxilla, containing the third incisor, canine, first, and second premolar teeth.

Attention to detail in surgical technique is of great importance in oncologic surgery and the management of oral tumors.[48,49] An oral tumor should be considered an "infective nidus" of neoplastic cells, which may exfoliate into the surgical field and give rise to local recurrence or enter the circulation and cause distant metastasis. During surgery, an oral tumor should therefore be covered using sterile gauze and handled as little as possible; if necessary, stay sutures may be placed in normal marginal tissue to facilitate manipulation. Other important measures include ligating blood vessels as early as possible in the procedure. Once the tumor is excised, a second set of instruments and drapes should be used for the reconstructive stage of the procedure.

Following surgical excision, the margins of the excised tissue should be examined for the presence of neoplastic cells.[14,53] As would be expected, the risk of recurrence or persistence is lower for patients with complete histologic borders.[29] Although sutures may be used to identify the borders of the specimen, using different dyes (Davidson Marking System; Bradley Products Inc., Bloomington, MN) to mark the edges of the specimen enables the pathologist to more accurately identify and orient the sample.[14]

Patient positioning

Lateral recumbency is preferred by some veterinarians for mandibulectomy and maxillectomy procedures.[52] Although lateral recumbency offers good exposure of the buccal surfaces of the uppermost teeth and jaws, it only provides fair visualization of the palate and lingual surfaces of the opposite quadrants. Therefore, dorsal recumbency is recommended for maxillectomy.[14,52,54] For mandibulectomy procedures, the authors prefer sternal recumbency, with the head elevated and the upper jaw suspended from intravenous poles using 2" (5 cm) tape placed at the level of or caudal to the maxillary canine teeth.[14,27] The main hazard of dorsal and sternal recumbency is fluid aspiration. The use of a cuffed endotracheal tube and pharyngeal pack is necessary to prevent aspiration. In addition, having continuous suction available is very helpful. In dorsal recumbency, the neck should be fully extended and the head end of the table slightly lowered. To further improve visibility, use of surgical loupes and a head lamp is beneficial, particularly for surgeries involving the caudal mandible or maxilla.

Management of adjacent teeth

Although maintaining teeth is secondary to achieving adequate surgical margins and tension-free closure, it is important to consider adjacent teeth when planning the osteotomy sites. The excised tissue bloc should preferably include intact alveoli as tumors may infiltrate down the periodontal ligament space.[14] If teeth are transected during osteotomy, the remaining roots must be extracted. Osteotomy in a narrow interdental space may injure a tooth that was not planned to be included in the ostectomy. In these cases, it is preferable to widen the surgical margin and transect or extract the adjacent tooth than to compromise the margin in order to avoid injuring the tooth. For example, when performing rostral mandibulectomy for a tumor involving or approaching the canine tooth, the second premolar tooth should be included in the section to be removed, because performing the osteotomy rostral to the second premolar tooth would transect the root of the canine tooth.

When performing an incisivectomy, great care should be taken not to damage the canine teeth when performing the osteotomies on the distal line angle of the third incisor teeth.[14]

For segmental or caudal mandibulectomies, the blood supply to the teeth in the remaining rostral section is transected. This may result in devitalization of the rostral teeth, necessitating extraction or endodontic treatment.[14]

Preservation of local blood supply

Knowledge of surgical anatomy is essential when planning a mandibulectomy or maxillectomy procedure. While major vessels may necessarily be ligated and transected as part of the surgical procedure, it is important to identify and preserve vessels supplying the mucosal flap and skin. For example, when performing a rostral mandibulectomy, it is usually possible to locate the branches of the mental artery as they exit the middle mental foramen at the level of the second premolar tooth; these vessels may be preserved and included as part of the flap used to cover the remaining mandibular bone. Similarly, branches of the infraorbital artery may be preserved when creating a vestibular mucosal-submucosal flap for closure following partial maxillectomy. Specific techniques for maxillectomy and mandibulectomy are described in Chapters 50 and 51, respectively. Although transection of small vessels leads to persistent, diffuse hemorrhage once the mucosa has been incised, the use of electrocautery should be avoided, as it is associated with delayed wound healing and an increased risk of wound dehiscence.[52,55,56] Similarly, although the use of CO_2 lasers for oral mucosal incisions has been described,[57] research suggests that the inflammatory response in tissues incised with CO_2 laser is greater than that of wounds made with a scalpel or Er,Cr:YSSGG laser[58] and mucosal healing of incisions made with CO_2 lasers is delayed when compared with incisions made with steel blades.[59–61]

Tension-free closure

The defect resulting from removal of a portion of the mandible or maxilla must be reconstructed. This is typically achieved by means of mucosal and submucosal flaps from the adjacent soft tissues.

The tissue from which the vestibular flap originates will depend on the location and extent of the tumor to be resected; detailed surgical techniques for maxillectomy and mandibulectomy are described in Chapters 50 and 51, respectively. The flap should be undermined as needed to enable it to be brought into apposition with the hard palate mucoperiosteum (in the case of maxillectomy) or the sublingual mucosa (in the case of mandibulectomy) without tension.[47] If excision of the tumor precludes closure of the defect with a vestibular mucosal-submucosal flap, an axial pattern flap may be employed (Chapter 52) or a myocutaneous free flap can be transferred to the site with microvascular techniques,[52] which are discussed in Chapter 11.

Anticipation and prevention of complications

Hemorrhage

Hemorrhage is the primary intraoperative complication encountered during mandibulectomy or maxillectomy.[14] Blood typing and cross-matching are indicated, especially before a maxillectomy.[62] Because severe hemorrhage results when one of the main arteries (the inferior alveolar artery during a mandibulectomy and the infraorbital, sphenopalatine, and major palatine or maxillary arteries during a maxillectomy) is transected prior to ligation, every attempt should be made to identify and ligate the arteries prior to transection. A mandibulectomy technique has been described that employs an osteotomy bur (Lindemann bur; Hu-Friedy Mfg. Inc., Chicago, IL) on an oral surgery handpiece (INTRAsurge 300; KaVo America Corp., Lake Zurich, IL) to transect the ventral third of the mandible and make a shallow incision in the bone on the buccal and lingual aspects of the mandible over the mandibular canal without damaging the neurovascular bundle. Following the osteotomy, the mandible is separated, revealing the intact inferior alveolar blood vessels, which are then double-ligated and transected without hemorrhage.[14] Alternatively, piezosurgery can be used for an atraumatic osteotomy, resulting in little soft tissue trauma and a smooth bone finish (see Chapter 9).

During a maxillectomy, if the margin of excision extends beyond the major palatine artery, that artery should be identified and ligated immediately to reduce hemorrhage.[52] Diffuse bleeding may occur following trauma to the nasal turbinates during a maxillectomy and can be a significant source of hemorrhage. Application of pressure, identification and ligation of blood vessels, and placement of absorbable hemostatic agents are all useful methods of controlling hemorrhage.[14] The focal use of electrocoagulation may also be employed. Both the surgeon and the anesthetist should be adequately prepared and skilled to handle extensive blood loss.

In order to estimate blood loss, blood-soaked gauze can be counted and their contents estimated using a visual analog system (Fig. 48.5). The average 10 cm × 10 cm (4″ × 4″) surgical gauze holds 12 mL of blood when saturated and 15 mL when supersaturated.[63] By using a sponge counter bag (Medline Industries, Inc., Northfield, IL) in the operating suite, total blood loss can be quickly estimated.

• **FIG. 48.5** A sponge counting bag (Medline Industries, Inc., Northfield, IL) in the operating suite allows rapid estimation of a patient's blood loss during surgery.

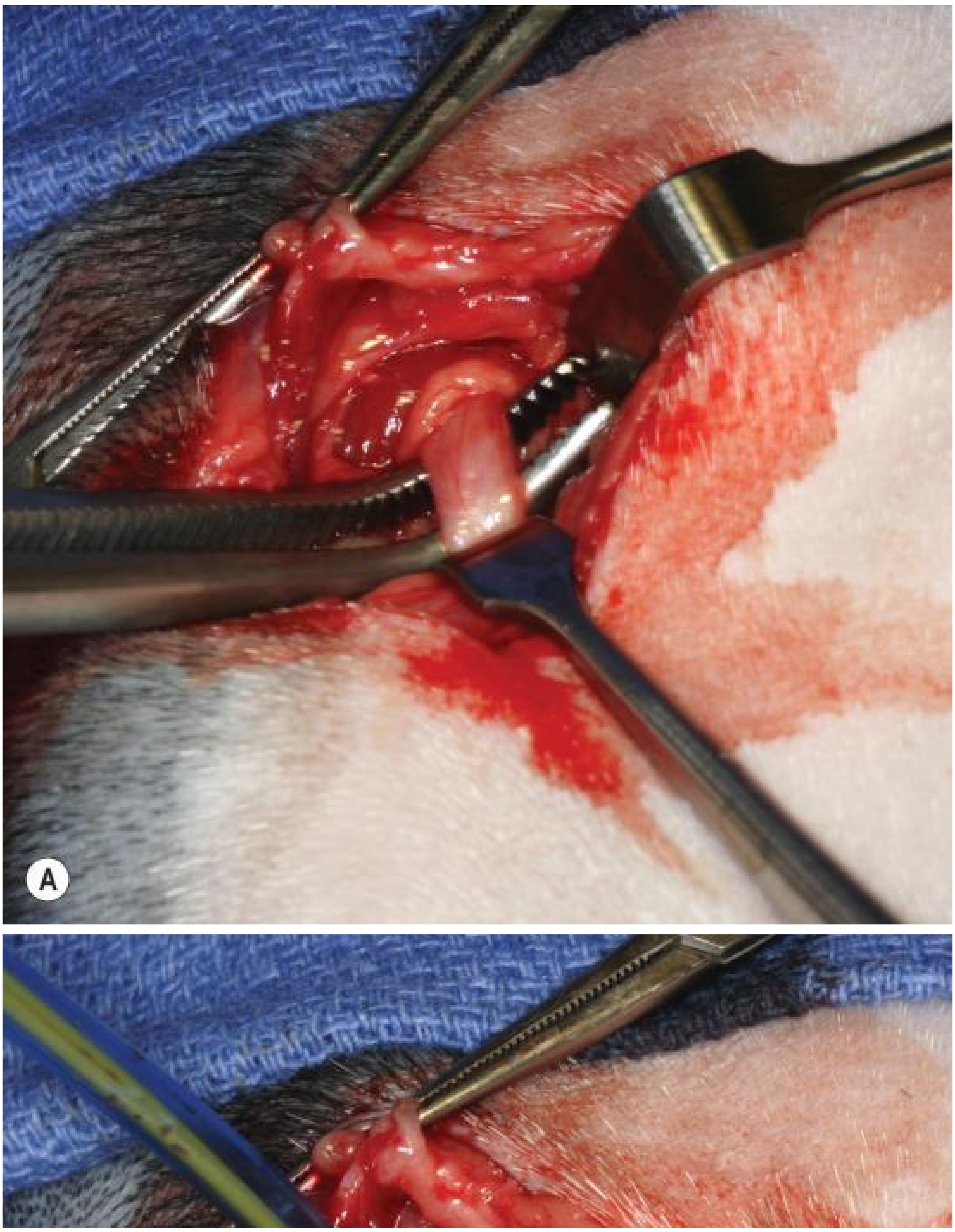
Postoperatively, hemorrhage may occur if a ligature becomes undone or if hemostatic agents become dislodged as blood pressure rises. Hospitalization is indicated to allow careful monitoring for hemorrhage during the first 12–24 hours after surgery.

Common carotid artery ligation

Preoperative, unilateral, or bilateral carotid artery ligation is rarely indicated.[64] However, hemorrhage from the maxillary or the inferior alveolar artery could be life-threatening. In addition, the complicated anatomy at the region of the maxillary artery makes it difficult to access, especially if hemorrhage has occurred, which may render direct maxillary artery ligation impractical.

Temporary or permanent ligation of one or both common carotid arteries can be performed in dogs without detrimental effect due to the rich collateral blood supply from the vertebral artery and maintenance of proper perfusion and cerebral function.[65–68] However, cats lack the collateral circulation seen in dogs, and the use of unilateral common carotid artery ligation should be done with care and avoiding permanent ligation if possible.[66,67]

Approach to the common carotid artery should be done with the dog in dorsal recumbency and the neck extended and supported.[66] A ventral approach is performed and a 5–10-cm skin incision is made just caudal to the larynx, followed by blunt and sharp dissection to separate the sternohyoid muscle, incising the fascia and exposing the trachea. Once the trachea is exposed, the carotid sheath can be observed dorsal and lateral to it (pulse may be observed as well). A curved hemostat is used to isolate and elevate the carotid artery and a Rummel tourniquet is placed to temporarily occlude the artery, allowing the surgeon to identify and occlude the maxillary artery if possible (Fig. 48.6A and B). In a case where the source of the bleeding cannot be identified, the temporary occlusion of the carotid artery by the Rummel tourniquet may be converted into a permanent occlusion using 0 polydioxanone or polyglactin 910 absorbable sutures (Fig. 48.6C).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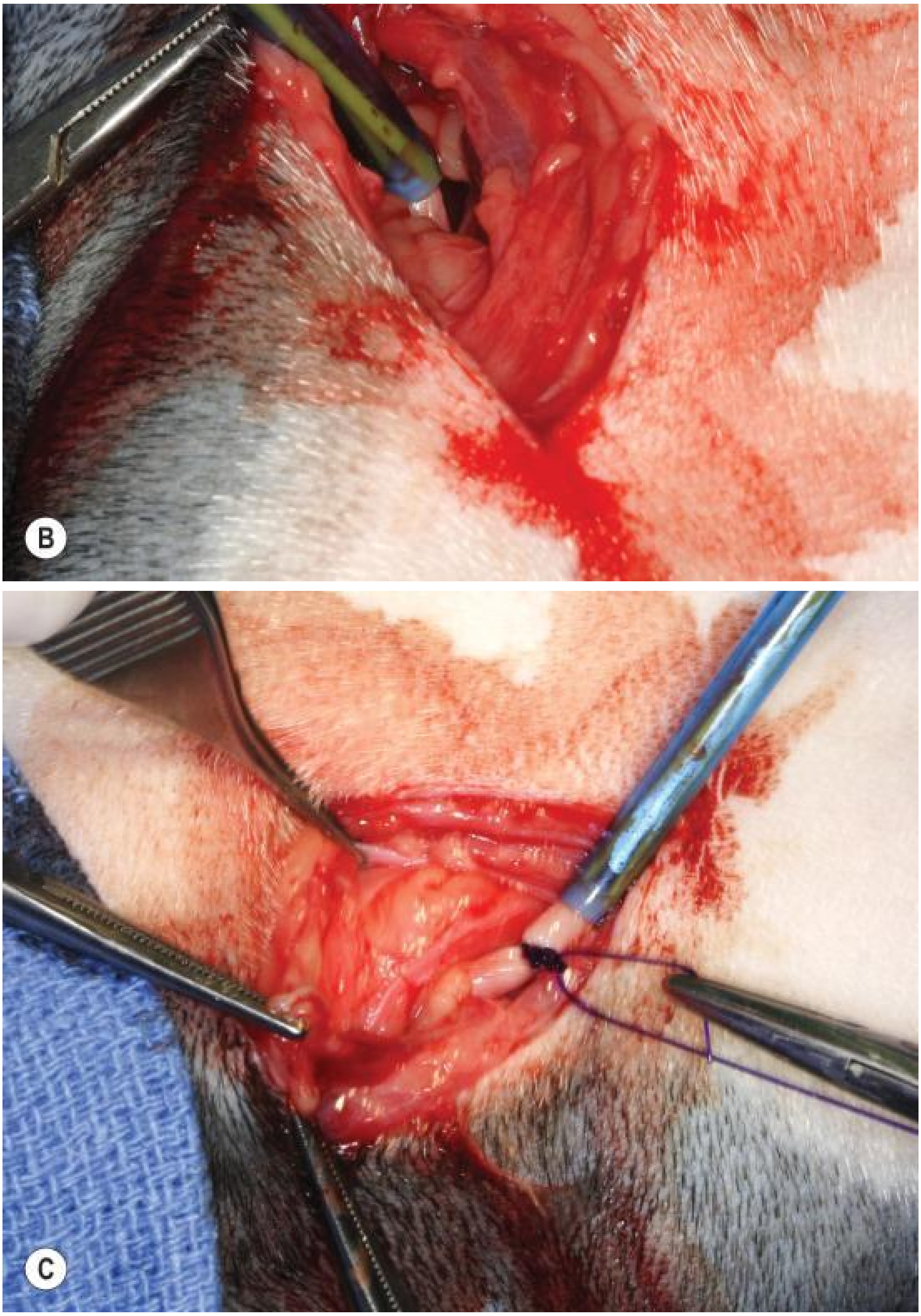

• **FIG. 48.6** Carotid artery ligation in a dog. (**A**) A curved hemostat is used to isolate and elevate the carotid artery. (**B**) A Rummel tourniquet is placed to temporarily occlude the artery, allowing the surgeon to identify and occlude the maxillary artery if possible. (**C**) Converting the temporary occlusion of the carotid artery into a permanent occlusion using polydioxanone or polyglactin 910 absorbable sutures can be performed in a case where the source of the bleeding cannot be identified.

Infection

Due to the excellent blood supply of the oral soft tissues, wound healing in the oral cavity is typically rapid and uncomplicated, and infectious complications are uncommon. However, delayed wound healing and a higher incidence of wound infection should be anticipated in the presence of immunosuppressive systemic illness, following long-term administration of corticosteroids, in animals undergoing chemotherapy, or if the surgical site has previously been irradiated.[14]

Ideally, the teeth will have been scaled and polished during the anesthetic episode for clinical staging, diagnostic imaging, and biopsy.[63] This provides a cleaner surgical field and less inflamed gingival tissues at the time of the maxillectomy or mandibulectomy.[14]

Most oral oncologic surgical procedures involve "clean-contaminated" surgical wounds, and intraoperative antibiotic prophylaxis is warranted.[69] Ampicillin, amoxicillin-clavulanic acid, certain cephalosporins, and clindamycin are appropriate choices.[70–74] The use of antibiotics is discussed in detail in Chapter 3.

Surgical technique plays an important role in preventing infectious complications. In addition to employing aseptic technique, the surgeon must be careful not to traumatize the oral soft tissues, including the wound edges as well as more deeply situated tissues. Electrocoagulation should be ideally avoided or used judiciously.[75] Irrigation must be used during osteotomy, ostectomy, and osteoplasty to prevent thermal necrosis of bone.[14]

Dehiscence

Wound dehiscence is a relatively common complication following maxillectomy, with a reported incidence of 7%–33%; the majority of cases which dehisced were caudal maxillectomies.[10,76,77] As previously mentioned, tension-free closure of the vestibular mucosal-submucosal flaps is essential to proper technique and will largely prevent dehiscence.[52,54] Overzealous use of electrocoagulation may also result in dehiscence.[75]

Although uncommon, wound dehiscence following mandibulectomy may occur at the rostral end of the mandible, especially at the alveolar margin, resulting in bone exposure.[27,52] Small areas of dehiscence may heal by second intention, while larger areas may have to be debrided and closed surgically.[14] Dehiscence of commissurorrhaphy performed concomitantly with unilateral total mandibulectomy may also occur, and is repaired by delayed primary closure.[14]

With the use of regional anesthesia and appropriate postoperative analgesic medications, dogs may want to resume normal oral activities rapidly after maxillectomy or mandibulectomy. For 10 days postoperatively, it is important to restrict access to chew toys and to prevent patients from contaminating the surgical site with dirt or feces. Short leash walks are advised during this time so the pet's activity can be closely monitored. A basket muzzle is useful for dogs with free access to an outdoor area or those that have displayed coprophagic behavior in the past.

Because wound dehiscence may occur as a result of tumor recurrence or persistence, biopsy of the dehisced wound edges is indicated if surgical repair is performed on a dehisced area.[54]

Postoperative dysfunction

Following mandibulectomy in the dog, it is common for the tongue to hang out laterally (unilateral rostral mandibulectomy or total mandibulectomy) or ventrally (bilateral rostral mandibulectomy). The patient's ability to prehend food may be affected, and drooling may be significant; performing a commissurorrhaphy will help to keep the tongue in the mouth and reduce drooling following total unilateral mandibulectomy.[14] Drooling following a bilateral rostral mandibulectomy can be reduced by carefully reconstructing the lower lip in a raised position.[27] If a bilateral rostral mandibulectomy is performed farther caudally than the second premolar tooth, the patient may require assisted feeding for a short time after surgery in order for the tongue to return to normal function.[52,54]

Segmental, caudal, or total unilateral mandibulectomy will generally result in significant postoperative malocclusion. Typically, the intact mandible and the rostral part of the operated mandible, if present, drift toward the side of the resection. If both mandibular canine teeth are still present, they are likely to traumatize the hard palate.[62] Crown height reduction and endodontic treatment of the exposed pulp, or extraction of the mandibular canine tooth or teeth, are indicated to resolve and prevent further ulceration.[62] Alternatively, an "elastic training device" using orthodontic buttons and bands to maintain normal occlusion has been described.[31] If available, the use of regenerative techniques, discussed in detail in Chapter 53, will prevent postoperative malocclusion. Malocclusion can be particularly severe in cats, and temporary maxillomandibular fixation using interdental bonding has been suggested to prevent this complication.[27]

Although bilateral rostral maxillectomy in the dog results in considerable deformity, caused by drooping of the nose, this does not seem to affect breathing through the nostrils.[54] Following maxillectomy, some mandibular teeth can occlude with the vestibular flap and cause mild and transient ulceration[62]; in addition, the ability to pick up objects and retrieve toys and sticks may be impaired, particularly after bilateral rostral maxillectomy.[19]

When premolar and molar teeth are removed as part of a maxillectomy or mandibulectomy, the natural cleaning action of mastication is prevented, resulting in rapid accumulation of plaque and calculus on the teeth of the opposing quadrant. Frequent routine periodontal treatment is therefore indicated.[62]

Cats are much more likely than dogs to display significant morbidity following major oral surgery, with 98% of cats experiencing one or more of the following symptoms in the first 4 weeks following mandibulectomy: tongue protrusion, malocclusion, dehiscence, drooling, pain, and difficulty eating or grooming.[78] More than three-fourths of cats for whom long-term follow up was available displayed one or more of the above symptoms for the duration of their lives, and nearly half (18 of 38 cats with available follow-up beyond 4 weeks postoperatively) developed local tumor recurrence, in spite of adjuvant therapy in several cases.[78]

Client satisfaction following mandibulectomy and maxillectomy in dogs has been reported to be 85% and was proportional to the postoperative survival time.[79] Another important factor was the correct prediction of outcome and possible complications.[79] A similar percentage of clients with cats who had undergone mandibulectomy reported that they would choose the same course of action given the same circumstances, despite the fact that 76% of the cats who survived had adverse effects for the rest of their lives.[78]

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CHAPTER 49

Surgical treatment of tongue, lip, and cheek tumors

Bernard Séguin

Definitions[1]

Cheeks *(buccae)*: The cheeks form the caudal portion of the lateral margins of the vestibular cavity. Dogs possess small cheeks because of the large mouth opening. They are the lateral wall of the vestibule caudal to the commissures of the lips on either side, continuous with the lips.

Lips *(labia oris)*: The lips are the fleshy margins of the opening of the mouth. The upper lip *(labium superius)* is associated with the maxilla and the lower lip *(labium inferius)* is associated with the mandible. The lips form the rostral and lateral margins of the vestibule of the mouth. The upper and lower lips meet at the angles of the mouth *(angulus oris)*, forming the commissures of the lips.

Mouth *(os)*: The mouth in its restrictive definition is limited to the opening between the lips. In a broader sense it designates the entire oral cavity, in which are located the tongue and the teeth. The oral cavity proper *(cavum oris proprium)* is bounded dorsally by the hard palate and the rostral part of the soft palate, laterally and rostrally by the dental arches and teeth, and ventrally by the tongue and the reflected mucosa under the tongue. The vestibule of the mouth *(vestibulum oris)* is the space external to the teeth, gingiva and alveolar mucosa, and internal to lips and cheeks.

Tongue *(lingua)*: The tongue is mostly a large muscle *(m. lingualis proprius)* located on the floor of the oral cavity proper. It aids in prehension, chewing, and swallowing and is the location of the papillae and the taste buds *(caliculi gustatorii)*.

Surgical anatomy

Tongue[1]

The intricate movement of the tongue is made possible by extrinsic and intrinsic muscles. The extrinsic muscles are the *m. styloglossus*, *hyoglossus*, and *genioglossus*. The intrinsic muscle *(m. lingualis proprius)* contains superficial and deep longitudinal, perpendicular, and transverse fibers. The lingual mucosa is thick and heavily keratinized on the dorsal surface of the tongue and on the ventral surface becomes thinner and unkeratinized. Ventrally, it also forms an unpaired, median mucosal fold, the lingual frenulum. The frenulum attaches the body of the tongue to the floor of the oral cavity.

The blood supply to the tongue is primarily through the paired lingual arteries, which are one of the largest branches of the external carotid arteries. The lingual arteries are closer to the ventral surface and midline. There are several anastomoses between the left and right lingual arteries found at the level of the root, body, and tip of the tongue. The lingual veins are in close proximity to their respective lingual arteries.

The tongue is innervated by the lingual nerve, a branch of the trigeminal (V), the *chorda tympani*, a branch of the facial (VII), the glossopharyngeal (IX), and the hypoglossal (XII) nerves. The only nerve responsible for the motor function of the tongue is the hypoglossal nerve. It runs parallel to the lingual artery. All nerves play a role in the sensory function of the tongue such as tactile, pain, thermal, taste, or proprioception.

Lips and cheeks

The lips and cheeks consist of three basic layers: the external layer is the integument, the middle layer consists of muscles and fibroelastic tissue, and the internal layer is the mucosa. The blood supply is provided by the inferior and superior labial arteries bringing blood to the lower and upper lip, respectively. Both are branches of the facial artery, arising from the external carotid. The inferior labial artery anastomoses with the caudal mental and the sublingual arteries. The superior labial artery forms anastomoses with the lateral inferior palpebral, malar, lateral nasal, and masseter arteries. These anastomoses ensure a significant collateral blood supply to the lips.

The lips and cheeks are innervated by the trigeminal and facial nerves. Severing branches of the facial nerve when attempting a tumor excision may lead to droopy lips and drooling, especially in dogs with more pendulous lips. Because the lips do not play a role in the prehension of food in dogs and cats, severing these nerves at the level of the lips or cheeks minimally affects quality of life.

The parotid duct opens, via the parotid papilla, into the vestibular area, at the caudal margin of the maxillary fourth premolar tooth. In some instances, it is necessary to excise this area during tumor removal. The duct can be ligated and the salivary gland will atrophy, or the duct can be preserved and transposed. However, preservation of the duct should not be attempted if it could compromise the completeness of the tumor removal.

Lymphatic drainage[2]

There are three lymph centers in the head: the *lymphocentrum parotideum*, *lymphocentrum mandibulare*, and *lymphocentrum retropharyngeum*. Lymphatic drainage of the tongue, cheek, and lips is mainly to the *lymphocentrums mandibulare* and *retropharyngeum*. Therefore the mandibular and retropharyngeal lymph nodes, as well as the tonsils, are of clinical interest. Lymph vessels of the head and neck can cross over the midline, and therefore nodes on both sides of the head and neck should be evaluated even in the presence of a unilateral lesion. The efferent lymph vessels of the lymphocentrums of the head are part of the afferent vessels of the *lymphocentrum cervicale*. The superficial cervical (occasionally incorrectly termed "prescapular") lymph nodes should also be evaluated when a neoplasm of the head is present.

Therapeutic decision-making

The amount of tissue to be excised depends on the size of the tumor and its histologic type. Performing a biopsy to find out the type of tumor is imperative before attempting definitive excision. As a general rule, benign tumors can be removed by performing a marginal excision, whereas malignant tumors require a wide excision (see Chapter 48). When planning the excision, the primary goal is to achieve a complete excision of the tumor (i.e., "clean"/tumor-free margins). Once the amount of tissue to be removed to achieve a complete excision has been determined, the remaining function needs to be anticipated in light of the reconstructive options. If function is projected to be greatly compromised and to severely affect quality of life, then a different therapeutic option must be sought. Adjuvant or neoadjuvant radiation therapy or chemotherapy may be necessary, and therefore a multidisciplinary approach must always be kept in mind.

Tongue tumors

The tongue plays a much more important role in the prehension of food and water in the cat than in the dog. The tongue is also important for grooming in cats, which is an important function for the quality of life of a cat. Therefore dogs are better at adapting following a partial glossectomy. Malignant tumors that are located on the rostral half of the tongue can be removed by full-thickness partial glossectomy. Tumors located in the caudal aspect of the tongue can be excised if they are superficial, especially if in the dorsal aspect. It has been shown that dogs tolerate 40%–60% of their tongue being excised.[3] Based on a limited number of cases, it appears that dogs can tolerate 75%–100% amputation of their tongue while achieving an acceptable functional outcome and be able to adapt to their disability.[3,4] However, such extensive glossectomy may not always reliably achieve the same prognosis. The cat may only be able to tolerate 50% of its tongue being excised.[5]

Benign tumors, such as granular cell myoblastoma, can be removed with conservative and close margins, i.e., by marginal excision. Malignant tumors require a full-thickness glossectomy, which may be a wedge, transverse, or longitudinal glossectomy. Smaller tumors located near the lateral edge of the tongue a few centimeters away from the tip can be removed with a wedge glossectomy. If the lingual artery is sacrificed, it is possible for the ipsilateral rostral aspect of the tongue to survive because of the existing anastomoses between the paired lingual arteries. If the surgery leaves a significant portion of the rostral aspect of the tongue and the lingual artery was sacrificed, it may be safer to perform a longitudinal glossectomy, as the anastomoses may not be sufficient to maintain the viability of the ipsilateral rostral aspect of the tongue. A longitudinal glossectomy is also indicated for larger tumors that are limited to one side of the tongue. If the tumor invades both sides of the tongue or comes too close to the midline, such that both lingual arteries would be sacrificed during tumor excision, a transverse glossectomy is recommended.

Lip and cheek tumors[6]

In order to achieve a complete excision ("clean"/tumor-free margins), malignant tumors of the lips and cheeks are best removed with a full-thickness excision of the lip or cheek, unless the malignant tumor is strictly cutaneous, in which case the deep margin can be the levator nasolabialis, maxillonasolabialis, orbicularis oris, and buccinator muscles for the upper lip and orbicularis oris and buccinator muscles for the lower lip. However, if there is any doubt as to how deep the tumor infiltrates the lip, a full-thickness excision of the lip is recommended. An en bloc excision of the lip and maxilla or mandible can be done for tumors that involve both the gingiva and the lip. Following tumor excision, the next priority is restoration of function. For the lips and cheeks, this mainly involves preventing drooling and leakage of oral contents. Cosmesis largely depends on the restoration of a smooth lip margin. Selection of the most appropriate method of closure is based on the size and location of the defect, the inherent tissue elasticity of the species, breed, and particular individual patient, as well as the tissues available in the vicinity that can be used in the reconstruction to close the defect. It has been stated that, as a general rule, approximately half of the upper lip (full length being from the philtrum to the commissure) can be removed and the defect closed primarily without resorting to more advanced reconstruction techniques.[7] The cosmetic appearance of the pet can be an important factor in the decision-making for some clients. Providing information to the clients about the prognosis following tumor excision, including the expected quality of life and cosmetic appearance, is crucial. This will lead to greater client satisfaction regarding their decision. Most reconstruction techniques will create some rostral displacement of the commissure of the lips on the ipsilateral side of the tumor excision: the larger the defect, the more noticeable the displacement. The buccal rotation, the labial buccal reconstruction with inverse tubed skin, and the upper or lower labial pedicle rotation flap techniques create the most significant rostral displacement of the commissure.

As a general rule, the simplest technique of excision and lip/cheek reconstruction should always be employed. However, since complete excision is one of the primary goals, the larger the tumor, the larger the defect will be, and therefore the more complex and involved the technique for reconstruction will be.

As a general rule, techniques for the upper lip and lower lip are similar. Smaller tumors can be removed by performing a wedge excision. Larger tumors are removed by performing a rectangular excision. The defect can be closed in a "Y" fashion or, for larger defects, it can be closed by performing a full-thickness labial advancement technique. The lower lip is easier to advance compared with the upper lip, with the mucosa/submucosa being the most significant restricting layers. For tumors that are at the most rostral aspect of the lower lips, i.e., at the level of the incisor teeth, a bilateral labial advancement technique can be performed to close the defect on the rostral midline. For larger defects of the upper or lower lip, a labial pedicle rotation flap technique can be performed. Large defects of the upper lip can be reconstructed with a buccal rotation technique. Excision of the entire upper lip is possible with reconstruction using a buccolabial pedicle flap. Very large defects of the upper lip or cheek, with or without upper lip and/or lower lip involvement, can be reconstructed by using skin flaps. Small gaps in the mucosal surface can heal by second intention. Large mucosal defects are most likely to create scarring, contraction, and distortion of the lips if left to heal by second intention. Therefore the skin flap can be used as a substitute for the oral mucosal lining. Alternatively, an experimental technique has been described where the mucosa from under the tongue is grafted under a proposed skin flap, which will eventually be transferred as a free microvascular skin graft to the cheek/lip area to be reconstructed.[8] The time period between the grafting of the oral mucosa to the underside of a skin flap to the transfer of the skin graft to the facial area can be 2–3 weeks. By that time, the underside of the skin flap will be partially lined with oral mucosa, which will enable reconstruction of large cheek and labial defects.[8]

Dogs with more pendulous upper lips with a tumor that is situated at the dorsal aspect of the lip can be treated with an upper lip pull-down technique. It is important to assure that adequate tissue margins are available before attempting this technique of tumor excision and lip reconstruction. Dogs with malignant tumors extending closer to the alveolar mucosa and gingiva may require a full-thickness lip excision combined with a partial maxillectomy or mandibulectomy.

Surgical techniques—glossectomy

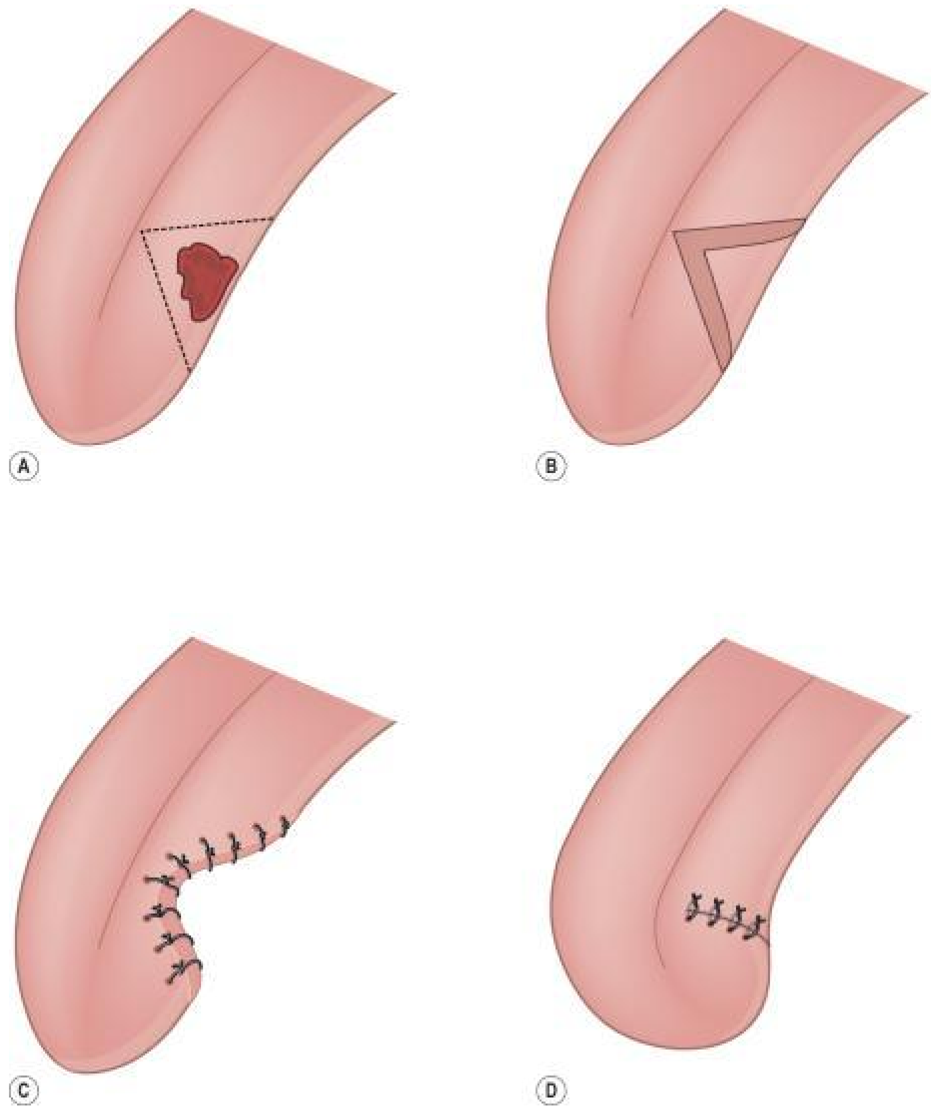
Stay sutures can be placed slightly away from the proposed incision site before the incision is performed. These stay sutures will help manipulate the tongue with minimal use of tissue forceps and hence decrease the trauma to the tongue. Because the tongue is very well vascularized, copious bleeding is usually present during surgery of the tongue. Hemostasis can be achieved by ligation of larger vessels, pressure, or judicious use of electrocoagulation. Through-and-through horizontal mattress sutures can be placed a few millimeters away from the incision to help control hemorrhage. Temporary occlusion of the carotid arteries (see Chapter 48) can be performed before attempting glossectomy to minimize blood loss; however, this is rarely required and blood loss can usually be controlled without having to resort to carotid occlusion. The edges of the incised tongue can be sutured using simple continuous or simple interrupted patterns with fine monofilament absorbable suture.

Marginal excision

A fusiform incision is made around the tumor into the tongue and the tumor is excised by partial-thickness glossectomy. The defect in the tongue is closed with simple interrupted or simple continuous suture pattern by apposition of the lingual mucosa.

Wedge glossectomy (fig. 49.1)

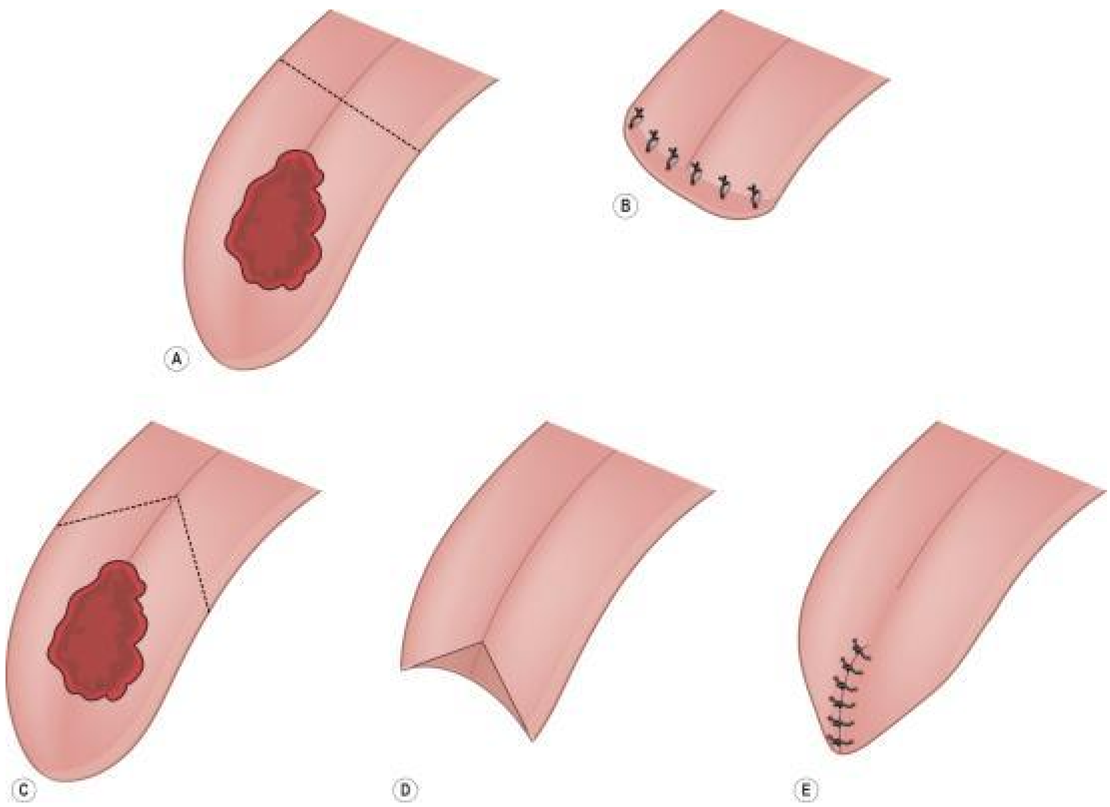
A full-thickness wedge-shape incision is performed around the tumor. The defect can be closed by apposing the ventral mucosa to the dorsal mucosa, thus closing it longitudinally, which will cause a narrowing of the tongue, or it can be closed transversely by suturing the dorsal mucosa together and the ventral mucosa together, which will cause a deviation of the tip of the tongue to the side of the excision.



• **FIG. 49.1** Wedge glossectomy. (**A**) Proposed wedge incision around the lesion. (**B**) A triangular defect is created at the edge of the tongue. (**C**) The incision can be closed by suturing the dorsal mucosa with the ventral mucosa. (**D**) Alternatively, the incision can be closed by suturing the two edges of the ventral mucosa together and the same with the dorsal mucosa.

Transverse glossectomy (fig. 49.2)

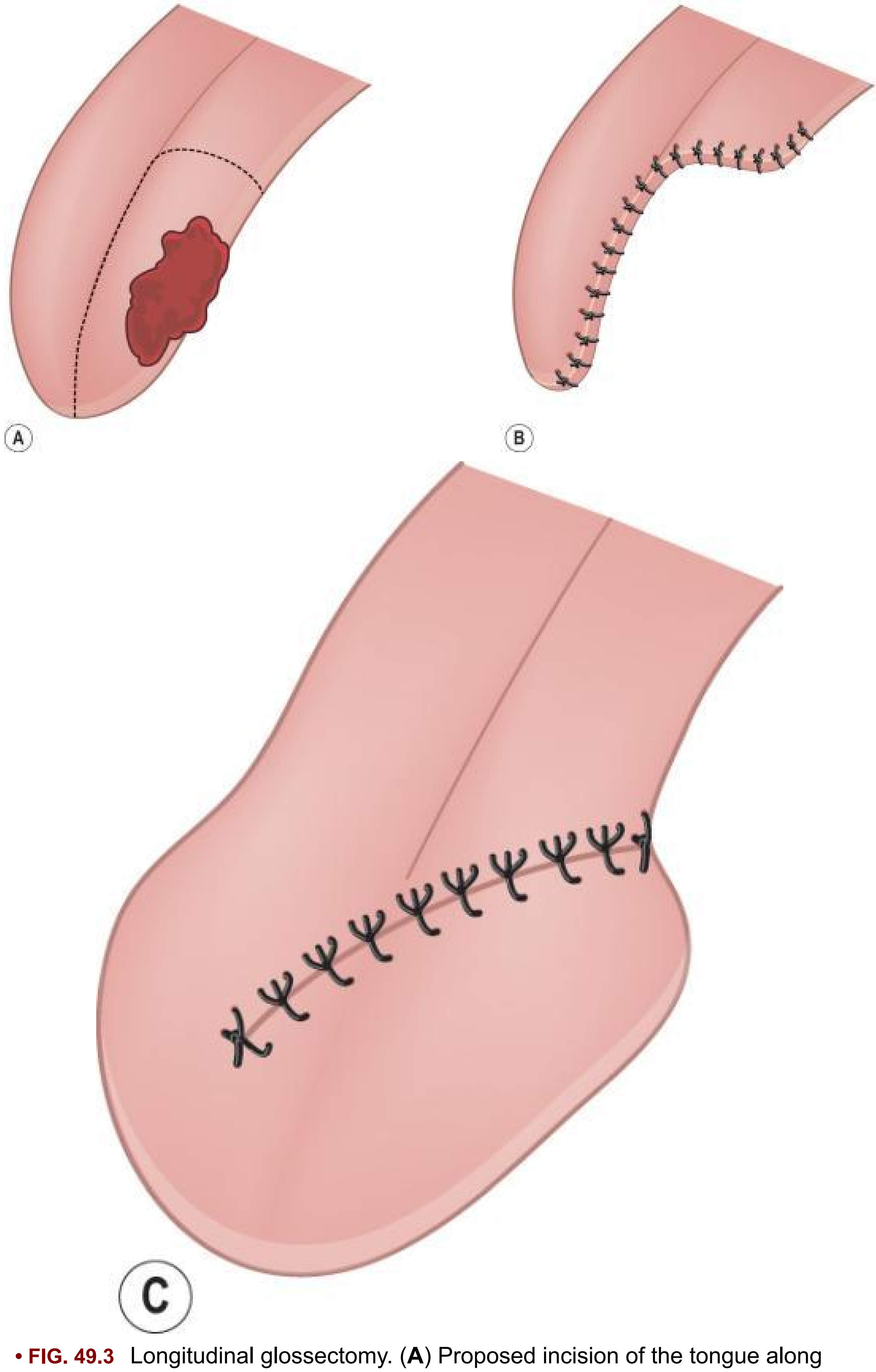
A full-thickness incision is made transversely across the tongue caudal to the tumor. The defect is closed by apposing the dorsal mucosa with the ventral mucosa. Depending on the shape of the tumor, it may be possible to create a V-shaped incision with the apex of the "V" directly caudally on midline. This defect is closed by apposing the dorsal mucosa on either side together and the ventral mucosa as well.



• **FIG. 49.2** Transverse glossectomy. (**A**) Proposed transverse incision of the tongue, caudal to the lesion. (**B**) The incision is closed by suturing the ventral and dorsal mucosae together. (**C**) Alternatively, the incision is made into a "V" with the tip of the "V" being caudal. (**D**) This results in a triangular defect at the rostral aspect of the tongue. (**E**) The incision is closed by suturing the ventral mucosa together and the same with the dorsal mucosa, creating a longitudinal suture line.

Longitudinal glossectomy (fig. 49.3)

A full-thickness longitudinal incision is made along the midline from the tip of the tongue to the caudal extent of the tumor. The incision is then brought to the lateral edge of the tongue, providing an adequate margin of normal tissue. The defect is closed by apposing the ventral and dorsal mucosal layers together along the length of the incision. An alternative way to close the defect is to rotate the remaining tip of the tongue and suture the cut edges of the mucosa dorsally together and ventrally together.[9]



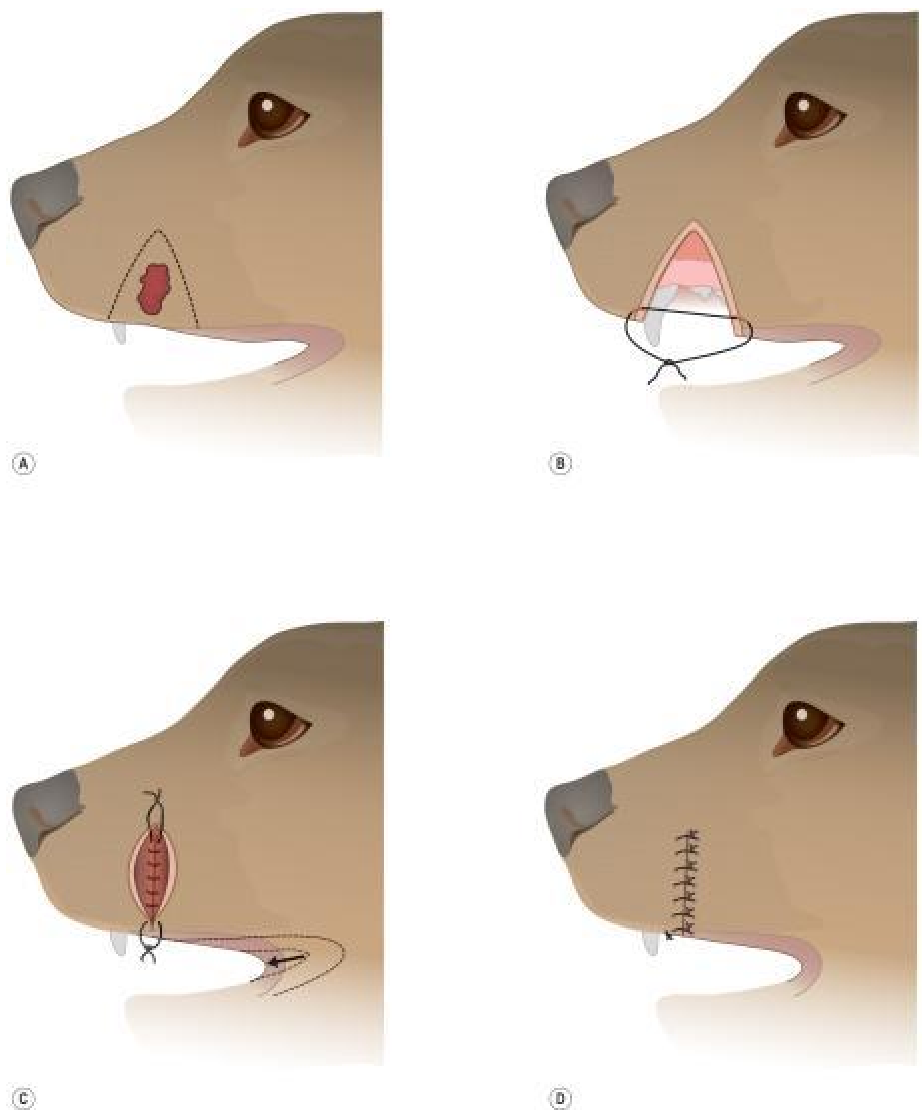
• FIG. 49.3 Longitudinal glossectomy. (**A**) Proposed incision of the tongue along midline and caudal to the lesion. (**B**) The incision is closed by suturing the ventral mucosa and dorsal mucosa together. (**C**) The alternative way to close is to rotate the remaining tip into the defect and suture the mucosa dorsally and ventrally.

Surgical techniques—excision of lip and cheek tumors and reconstruction[6]

Techniques for the upper lip and cheek

Wedge excision (fig. 49.4 and 28.3)

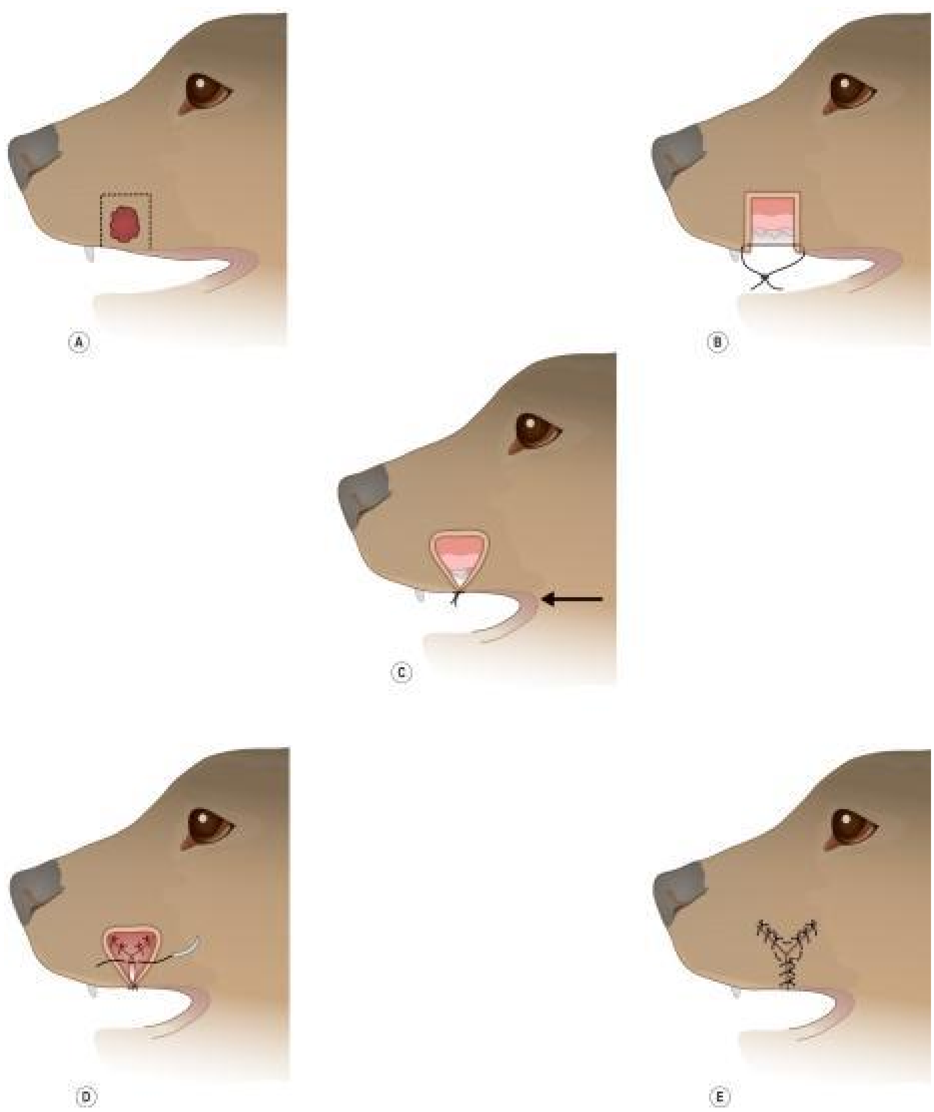
A wedge excision is performed from the lip with the tip away from the labial margin. The defect is closed by apposing the edges starting with the lip margins to ensure proper alignment. A two-layer closure is performed by suturing the mucosa first and then the skin.



• **FIG. 49.4** Wedge excision of lip. (**A**) Proposed full-thickness wedge incision around lesion with tip away from lip margin. (**B**) The defect is closed by apposing the lip margin first to ensure proper alignment. (**C**) A two-layer closure is performed by suturing the mucosa first. The commissure of the lips will move rostrally *(arrow)*. (**D**) The second layer is closed by suturing the skin. Source: (From Pavletic M. *Atlas of Small Animal Wound Management and Reconstructive Surgery*. 3rd ed. 2010. Reproduced with permission of Wiley-Blackwell, Ames, IA.)

Rectangular excision and y-closure (fig. 49.5)

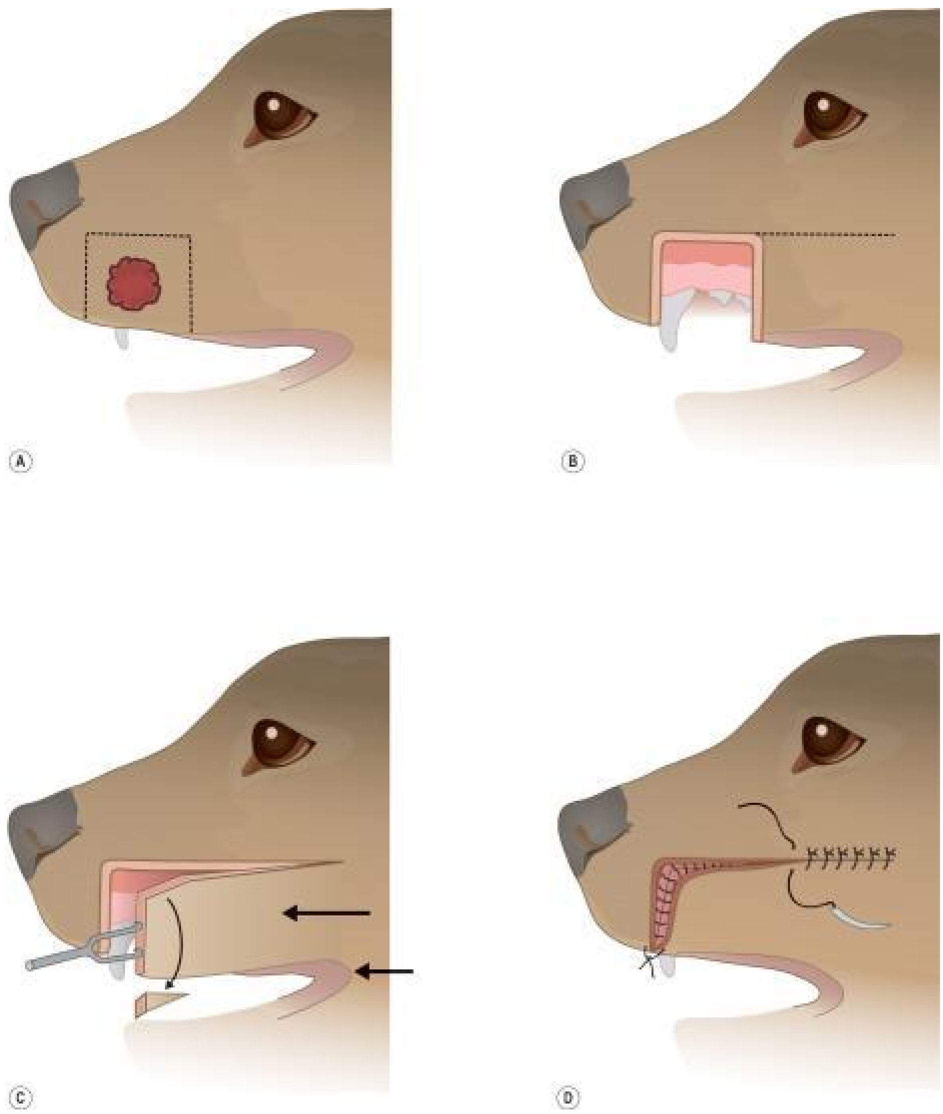
The tumor is removed by performing a rectangular incision around it, as well as removing the labial margin. The defect is closed by apposing the labial margins accurately first. Then the rest of the defect is sutured in a "Y" fashion with a two-layer closure (mucosa and skin).



• **FIG. 49.5** Rectangular excision of lip and "Y" closure. (**A**) Proposed full-thickness rectangular incision around lesion. (**B**) The defect is closed by apposing the lip margin first to ensure proper alignment. (**C**) The commissure of the lips will move rostrally *(arrow)*. (**D**) A two-layer closure is performed in a "Y" fashion, starting with the mucosa. (**E**) The second layer is closed by suturing the skin. Source: (From Pavletic M. *Atlas of Small Animal Wound Management and Reconstructive Surgery*. 3rd ed. 2010. Reproduced with permission of Wiley-Blackwell, Ames, IA.)

Full-thickness labial advancement technique (fig. 49.6)

After removing the tumor with a rectangular incision, the remaining lip caudal to the defect is mobilized by performing a full-thickness incision at the dorsal aspect of the lip, parallel to the teeth. The length of the mobilizing incision depends on the size of the defect needing repair. The flap should stretch over the defect without excessive tension. The labial margins are accurately apposed and the edges of mucosa are reapposed to reconstruct the vestibule. The skin is closed as a second layer. Tension sutures can be used in areas of moderate tension.

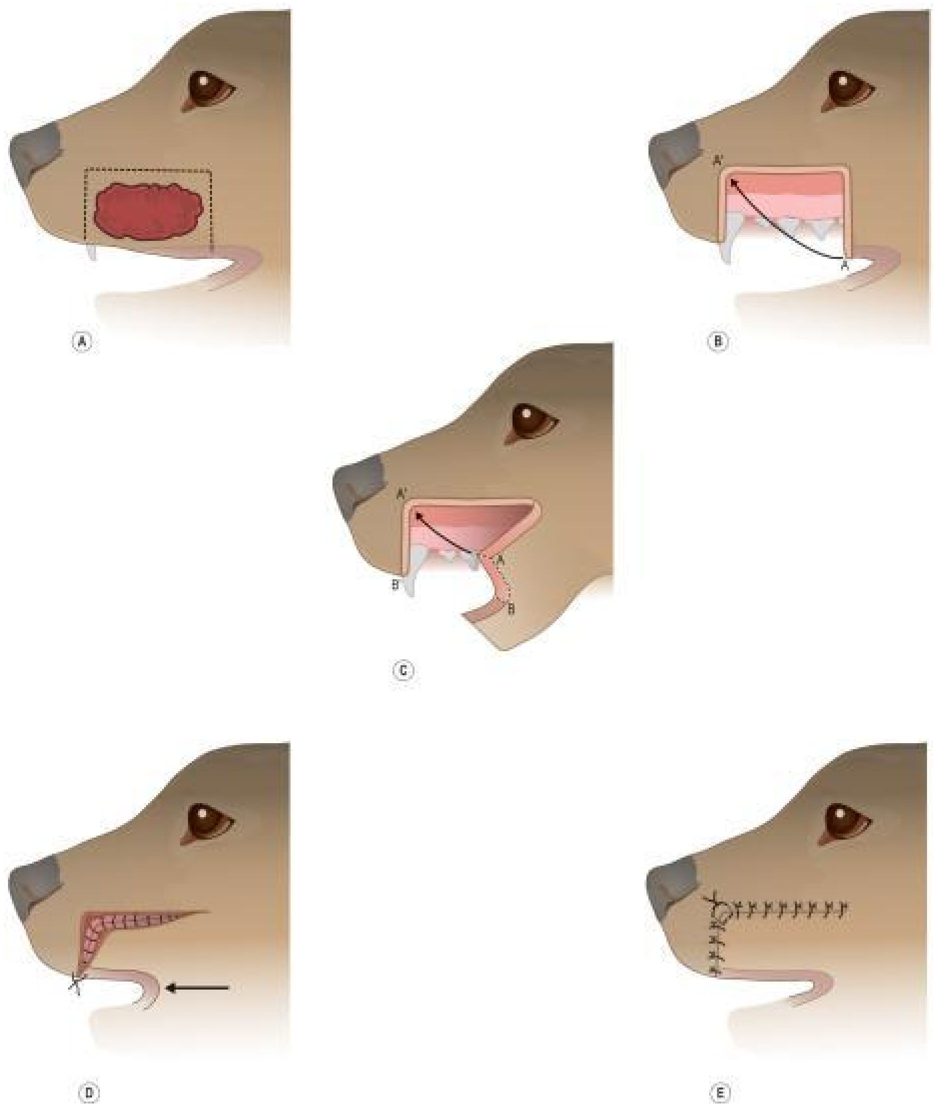


• **FIG. 49.6** Rectangular excision and full-thickness labial advancement technique. (**A**) Proposed full-thickness rectangular incision around lesion. (**B**) A full-thickness incision is performed at the level of the dorsal extent of the excision, the incision being parallel to the dental quadrant or lip margin. The length of the incision will depend on the size of the defect. (**C**) The labial flap is advanced rostrally, which will also cause rostral advancement of the commissure of the lips *(bottom arrow)*. A triangular full-thickness piece of the flap can be removed from the caudorostral aspect of the flap to assure that the lip margin will be leveled to account for the difference in height in the lip. (**D**) The labial flap is sutured to the rostral lip in a two-layer fashion, starting at the lip margin and suturing the mucosa first. Source: (From Pavletic M. *Atlas of Small Animal Wound Management and Reconstructive Surgery*. 3rd ed. 2010. Reproduced with permission of Wiley-Blackwell, Ames, IA.)

Buccal rotation flap (fig. 49.7)

The tumor is removed with a rectangular incision. The lip margin caudal to the rectangular defect is removed for a length equal to the height of the rectangle (i.e., dorsoventral dimension).

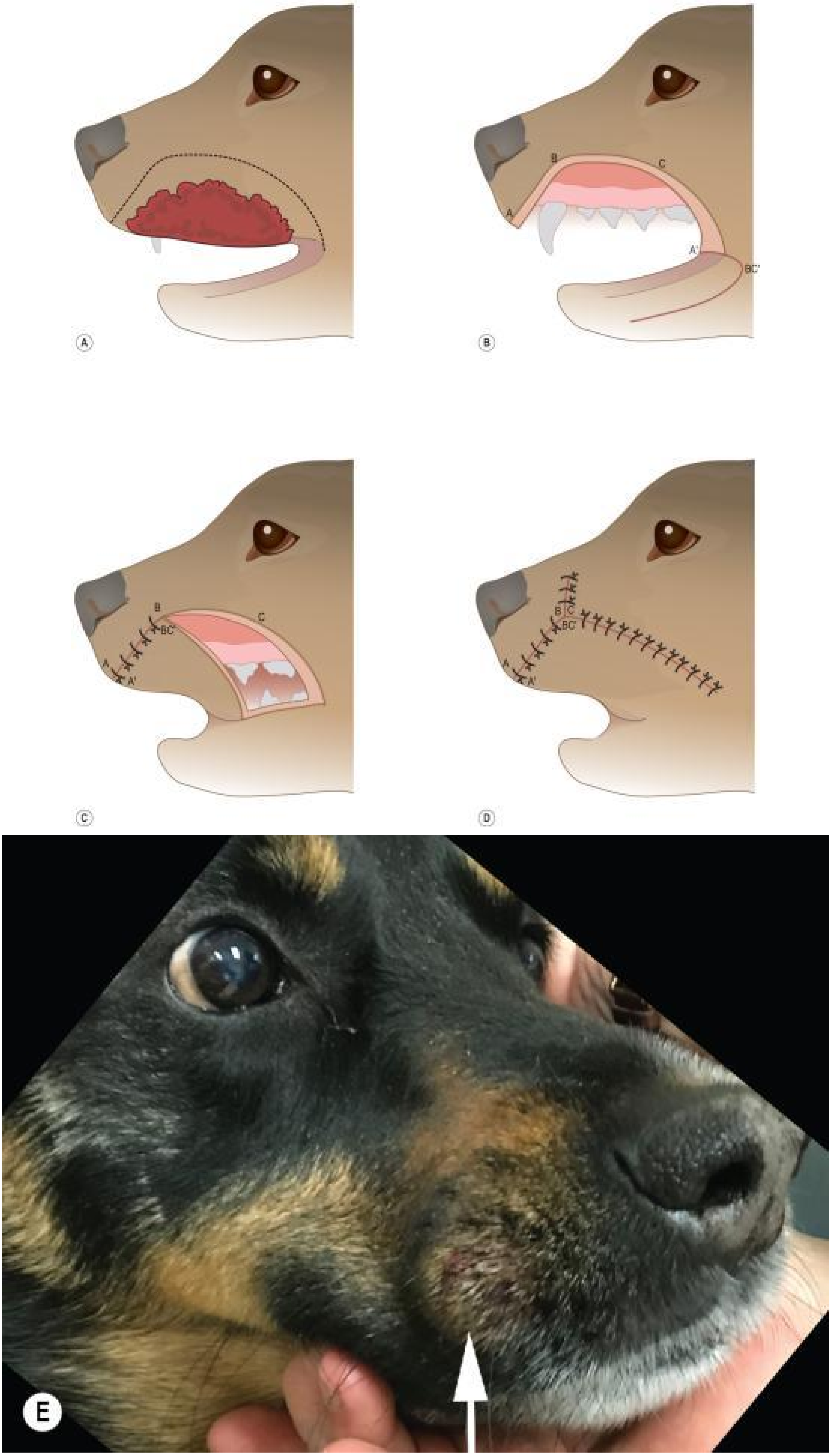
The caudoventral corner of the rectangle is brought into the rostrodorsal corner. A two-layer closure is performed by suturing the mucosal edges together and then the skin edges.



• **FIG. 49.7** Buccal rotation flap. (**A**) Proposed full-thickness rectangular incision around the lesion. (**B**) The caudoventral corner of the defect *(A)* will be apposed to the rostrodorsal corner of the defect *(A')*. (**C**) A segment of the lip margin is removed equal to the height of the defect in the lip. (**D**) The defect is closed in a two-layer fashion by apposing the caudal lip margin *(B)* to the rostral lip margin *(B')* and then suturing the mucosa. This causes the commissure of the lips to move rostrally *(arrow)*. (**E**) The skin is sutured. Tension sutures can be used in areas of moderate tension. Source: (From Pavletic M. *Atlas of Small Animal Wound Management and Reconstructive Surgery*. 3rd ed. 2010. Reproduced with permission of Wiley-Blackwell, Ames, IA.)

Lower labial pedicle rotation flap[8] (fig. 49.8)

The tumor is excised with a wedge or rectangular excision. Approximately 80% to 90% of the lip margin (from philtrum to commissure) can be excised. A full-thickness lower lip flap is created by making an incision parallel to the lip margin and 30–40 mm away from it. The incision begins at the caudal aspect of the upper lip defect and runs several centimeters cranially (long enough to be able to appose it with the rostral edge of the upper lip defect). The flap is rotated after releasing the restrictive soft tissues from the mandible such that the caudal edge of the flap meets the rostral edge of the upper lip defect. The margin of the lower lip flap becomes the margin of the upper lip where the defect was. The buccal mucosa is sutured. Small gaps can be left open to allow wider opening of the jaws. The skin is closed in an inverted "Y" fashion over the defect.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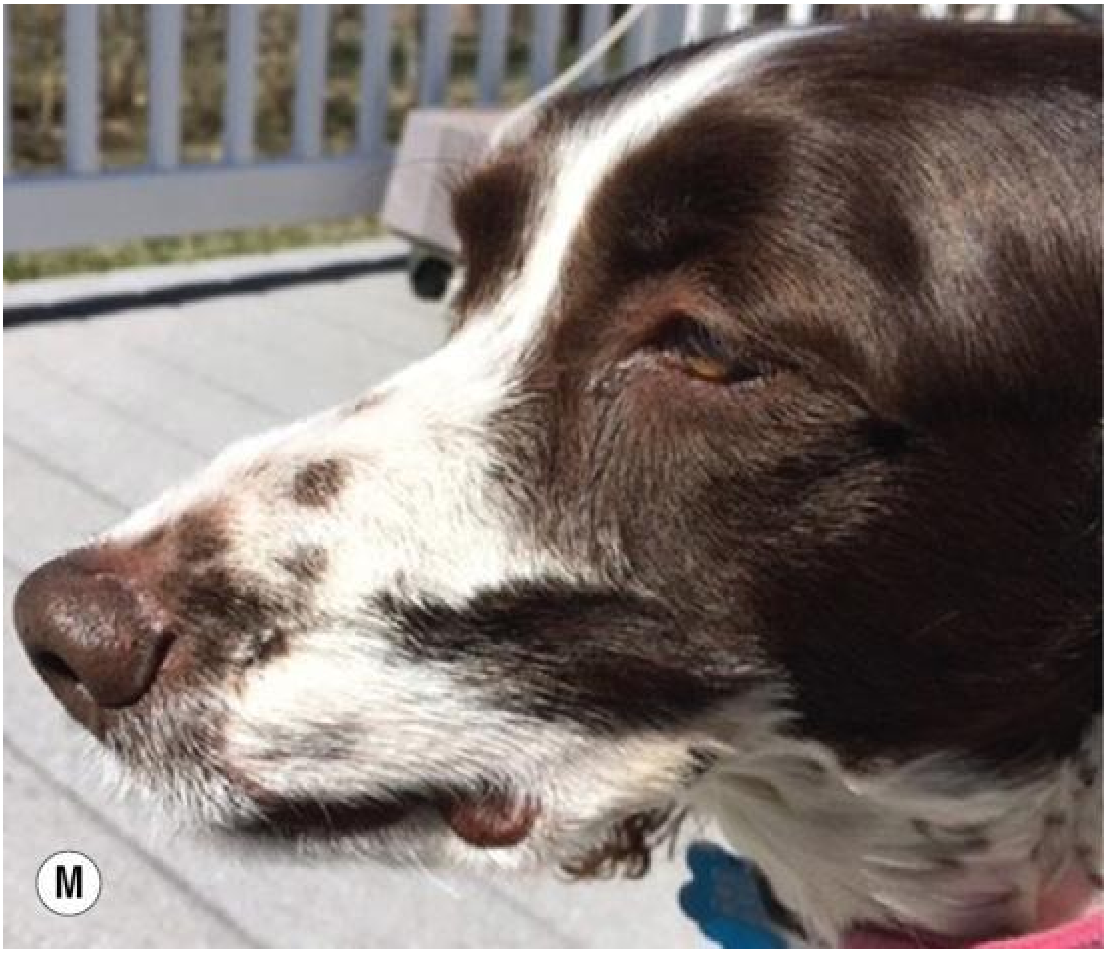
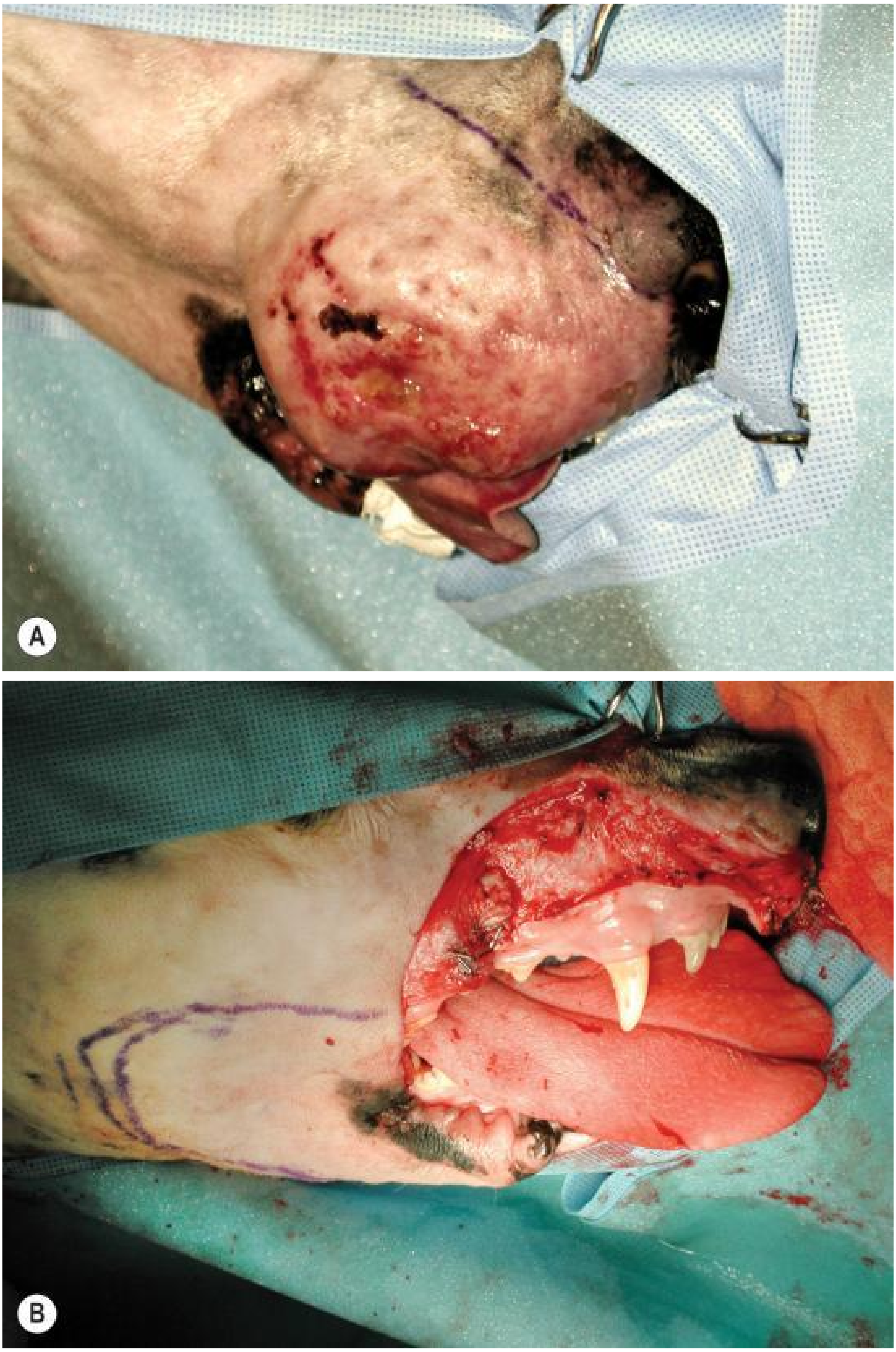


• **FIG. 49.8** Lower labial pedicle rotation flap. (**A**) Proposed full-thickness incision, which is dome-shaped. The defect will reach the commissure caudally. (**B**) A full-thickness labial flap is created with the lower lip. A first incision is made at the commissure, perpendicular to the lower lip margin. The length is equal to the height of the lower lip. A second incision is made parallel to the dental quadrant or lip margin starting at the first incision and running rostrally. The incision is long enough that it is possible to appose the flap to the rostral edge of the upper lip defect. (**C**) The labial flap is rotated and the lip margins are apposed (*A'* to *A*) The width of the flap is sutured to the rostrodorsal aspect of the defect (*BC'* to *B*). (**D**) A point along the dorsal border of the defect caudal to the transposed flap *(C)* is apposed to the flap without excessive tension (*C* to *BC'*). A two-layer closure is performed. Because suturing the buccal mucosa completely can greatly reduce mandibular range of motion, a defect in the mucosa can remain and allowed to heal by second intention. The skin is sutured in a "Y" fashion. This technique creates a significant rostral transposition of the opening of the mouth on the side of the surgery. (**E**) Dog with mast cell tumor of the right upper lip *(arrow)*. (**F**) A wide full-thickness excision has been performed. The planned flap for reconstruction is delineated with purple skin marker. The flap was a combination of upper labial advancement and lower labial pedicle rotation flap. (**G**) The flap has been sutured in its heterotopic position. (**H**) Seven months postoperative. (**I**) A combination lip excision and left unilateral rostral maxillectomy has been performed for a squamous cell carcinoma on the left upper lip in a dog. The planned reconstruction flap (purple marker line) was similar to the one in figure 49.8F. (**J**) Specimen after en bloc excision of the lip and maxillectomy. (**K**) Creation of the flap after incision in the skin and mucosa. (**L**) The flap has been sutured in its heterotopic position. (**M**) One month postoperative.

Buccolabial pedicle flap (fig. 49.9)

The tumor is excised where the entire upper lip is removed in the process. With this technique to reconstruct the upper lip, the lower labial pedicle rotation flap technique is modified by extending the labial flap into the cheek, thereby elongating the flap and allowing larger defects of the upper lip to be reconstructed. To close the lip defect, the flap created from the cheek and lower lip is full thickness such that the deep aspect of the flap is covered with buccal mucosa. An incision is made from the caudal aspect of the defect at the level of the junction between the labial mucosa and gingiva and runs caudally to the level of the most caudal aspect of the vestibule. The height (the ventrodorsal dimension) of the flap is made to be of equal dimension as the height of the excised lip. A ventral incision is made from the caudal incision of the buccal flap extending to the ventral aspect of the lower lip to about half of the length of the lower lip. This creates a buccolabial flap with the pedicle being at the rostral aspect of the flap, ventral to the upper lip, in line with the lower lip. The flap can be made larger and more robust by following the guidelines of the cutaneous angularis oris axial pattern flap to include the angularis oris and superior and inferior labial arteries (see cutaneous angularis oris axial pattern flap, Chapter 52).[10,11] The caudal aspect of the flap in its orthotopic position is rotated rostrally to join the rostral aspect of the upper lip defect. Restrictive soft tissues are released from the mandible to relieve tension on the flap. The incised mucosa of the flap is sutured to the edges of the mucosa of the defect with an absorbable suture material. By apposing the cut edges of mucosa, the buccal cavity is reconstructed. The edges of the skin are apposed where the caudal aspect of the flap (in its orthotopic position) meets with the rostral and dorsal aspects of the upper lip defect, and the ventral border of the flap (in its orthotopic position) meets with the dorsocaudal aspect of the defect. The skin edges are sutured with an absorbable suture material for the subcuticular layer and with nonabsorbable sutures for the dermis. Because the most rostral aspect of the new lip does not have a normal lip margin at the mucocutaneous junction, the skin is sutured to the buccal mucosa of the flap. The defect in the cheek (created by the transfer of the flap) is closed by simply apposing the ventral and dorsal edges together in three layers (mucosa, subcuticular, and dermis).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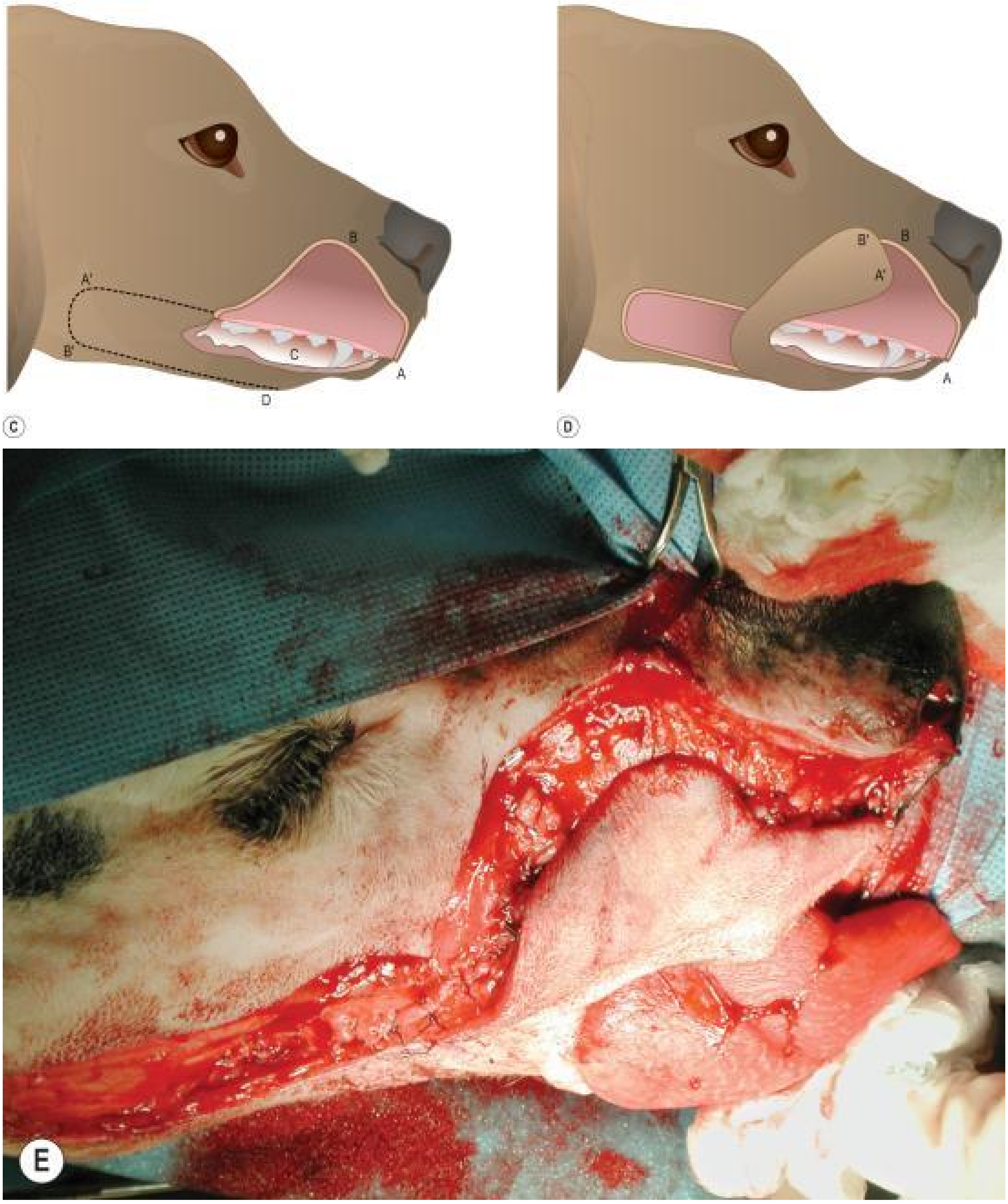

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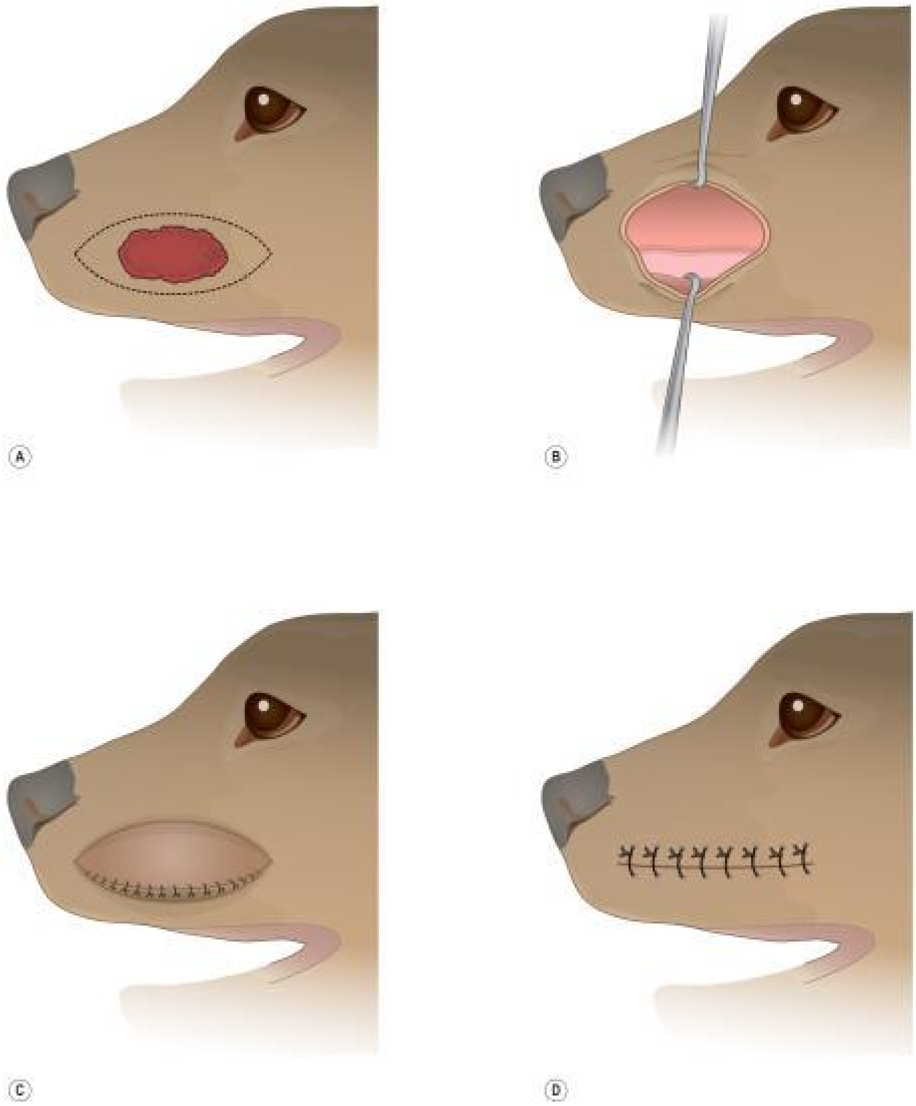
• **FIG. 49.9** Buccolabial pedicle flap. (**A**) A mast cell tumor is present on the right-upper lip before surgical excision. The dog is in left lateral recumbency and the nose is to the right of the figure. (**B**) The tumor has been excised where the entire upper lip was removed in the process. The limits of the proposed flap are demarcated with a surgical skin marker. (**C**) An incision is made from the caudal aspect of the defect at the level of the junction between the labial mucosa and gingiva, and runs caudally to the level of the most caudal aspect of the vestibulum. The height (the ventrodorsal dimension) of the flap is made to be of equal dimension as the height of the excised lip. A ventral incision is made from the caudal incision of the buccal flap extending to the ventral aspect of the lower lip to about half of the length of the lower lip. This creates a buccolabial flap with the pedicle being at the rostral aspect of the flap, ventral to the lower lip. The base of the flap is between points *C* and *D*. In transferring the flap into the defect, point *A'* will meet point *A* and point *B'* will meet with point *B*. (**D**) The caudal aspect of the flap in its orthotopic position is rotated rostrally to join the rostral aspect of the upper lip defect such that point *A'* will meet point *A* and point *B'* will meet with point *B*. Restrictive soft tissues are released from the mandible to relieve tension on the flap. (**E**) The incised mucosa of the flap is sutured to the edges of the mucosa of the defect with an absorbable suture material. By apposing the cut edges of mucosa, the buccal cavity is reconstructed. The defect in the cheek is also closed by apposing the ventral and dorsal edges of the mucosa. (**F**) The edges of the skin are apposed in the same fashion. Because the most rostral aspect of the new lip does not have a normal lip margin at the mucocutaneous junction, the skin is sutured to the buccal mucosa of the flap. (**G**) Photograph of the dog 43 days after surgery. The flap is viable and has healed into its heterotopic position. The tip of the maxillary canine tooth remains uncovered.

Upper labial pull-down technique (fig. 49.10)

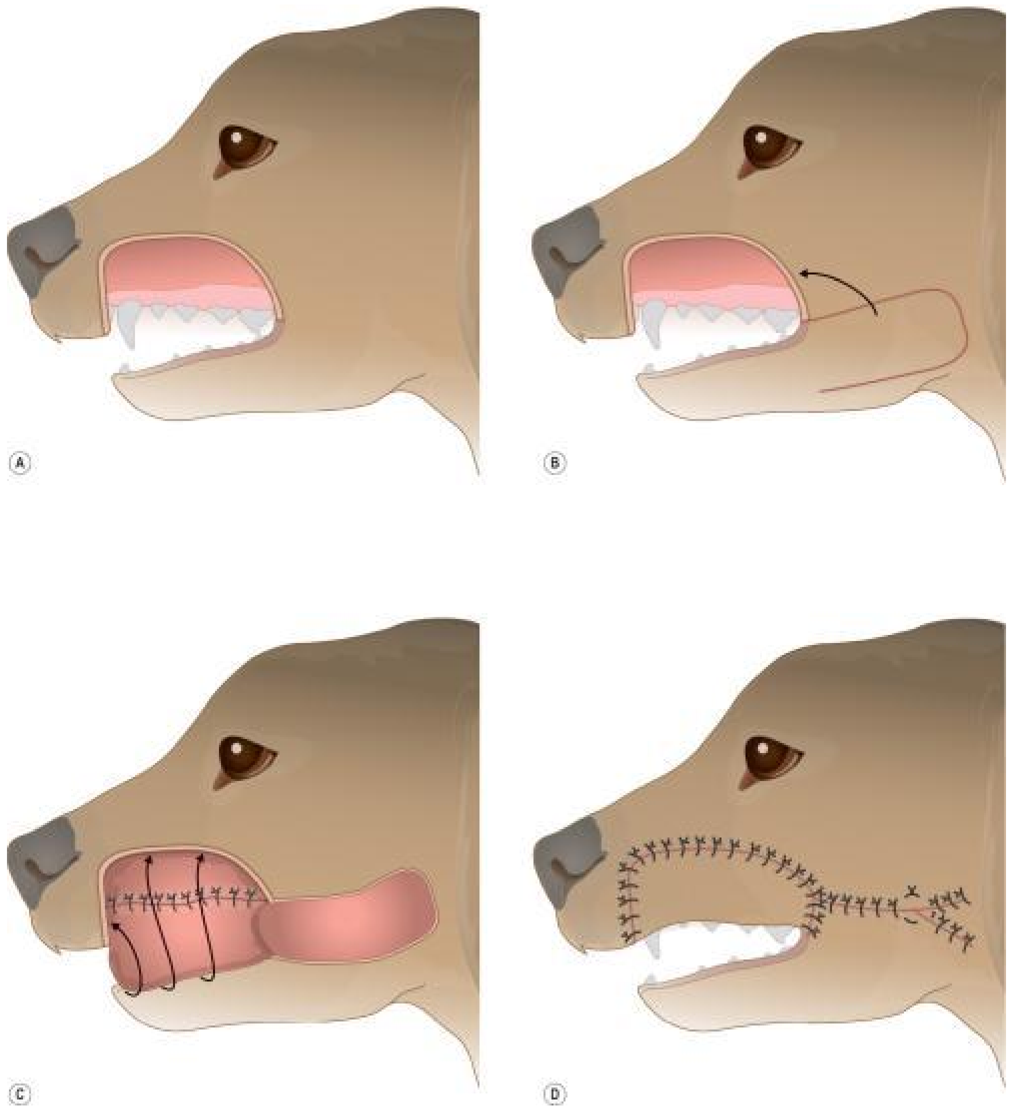
A full-thickness fusiform incision is created around the tumor, preserving the lip margin. The long axis of the defect is parallel to the lip margin. The labial mucosa is sutured to the oral mucosa. The skin defect is closed. This technique can be combined with a maxillectomy if the tumor is very close to the alveolar mucosa and gingiva. However, this technique has limited application for malignant tumors where a wide excision is needed for complete excision.



• **FIG. 49.10** Upper labial pull-down technique. (**A**) Proposed full-thickness fusiform incision around the lesion, preserving the lip margin. The long axis of the incision is parallel to the lip margin. (**B**) It is possible to also remove part of the maxilla to ensure an adequately deep margin when the tumor invades or buttresses against the maxilla. (**C**) A two-layer closure is performed by suturing the labial mucosa to the oral mucosa. (**D**) The closure is completed by suturing the skin. Source: (From Pavletic M. *Atlas of Small Animal Wound Management and Reconstructive Surgery*. 3rd ed. 2010. Reproduced with permission of Wiley-Blackwell, Ames, IA.)

Transposition skin flap (fig. 49.11)

The tumor is excised, creating a defect in the upper lip. A transposition skin flap is created caudal to the defect, ventral to the ear. Skin will be used to replace oral mucosa. The flap should be about twice as wide as the lip excised, since half of the flap is required to replace the mucosal defect. The flap is inverted rostrally to cover the defect such that the skin is inside the mouth. What was the dorsal border of the skin flap is sutured to the oral mucosa. The flap is then folded onto itself and what was the ventral border of the flap as it was created is now sutured to the skin at the dorsal edge of the upper lip defect. The skin defect from the flap bed is also closed. The flap used with this technique can be the cutaneous angularis oris axial pattern flap (see Chapter 52).[10,11]



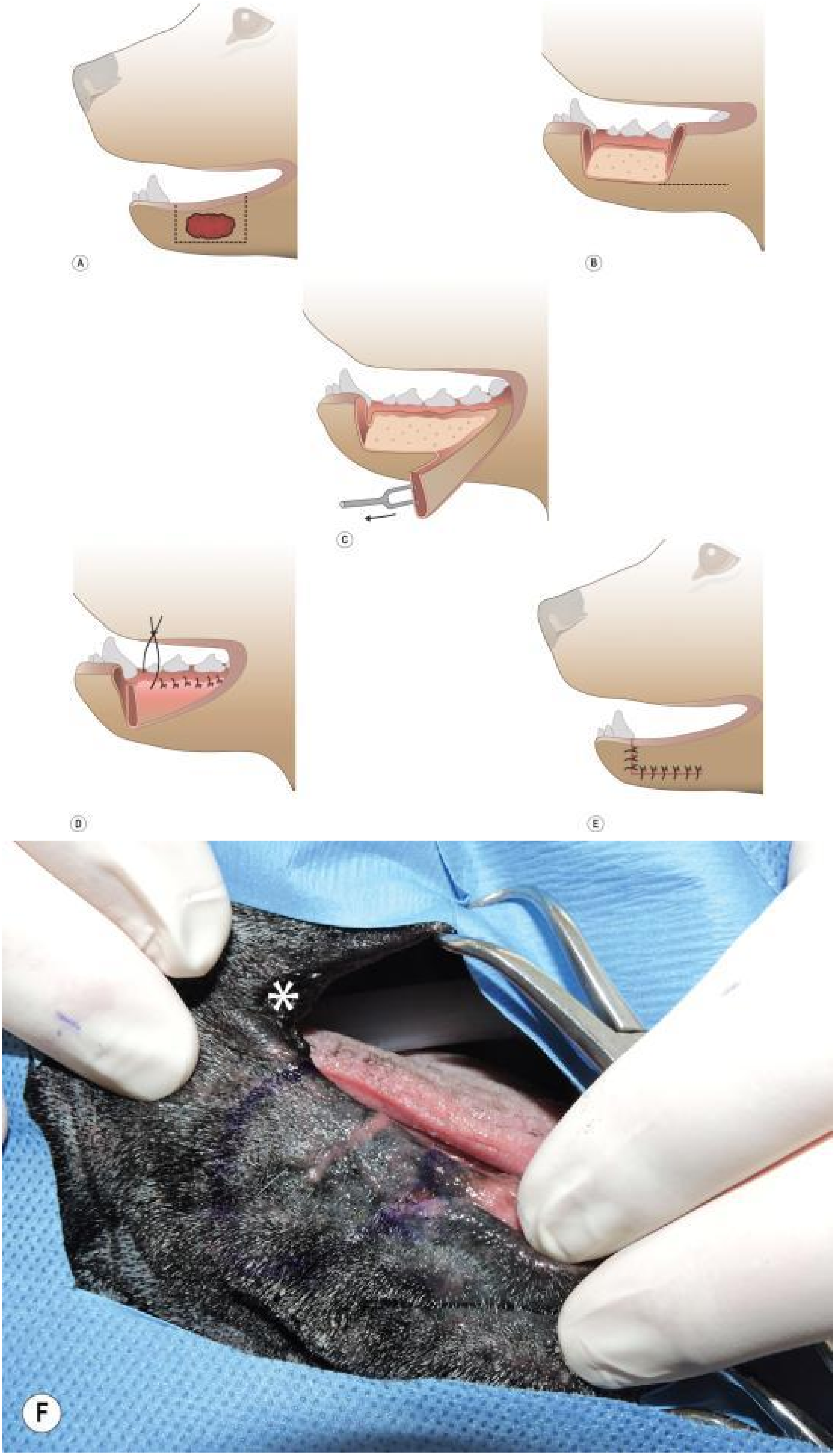
• **FIG. 49.11** Transposition skin flap. (**A**) Proposed full-thickness incision around lesion, removing most of the upper lip. (**B**) A single pedicle skin flap is created caudal to the defect and ventral to the ear. The base of the flap is at its rostral aspect and is aligned with the buccal–labial border. The width of the flap is twice the height of the defect since the flap will be folded in half. (**C**) The flap is folded onto its base rostrally and its dorsal border is sutured to the oral mucosa. The flap is then folded along its long axis such that its ventral border will be sutured to the skin border of the defect dorsally. (**D**) The remaining borders of the flap are sutured to the skin borders of the defect and the donor site of the flap is also sutured. Source: (From Pavletic M. *Atlas of Small Animal Wound Management and Reconstructive Surgery*. 3rd ed. 2010. Reproduced with permission of Wiley-Blackwell, Ames, IA.)

Techniques for the lower lip

The techniques described for the upper lip are also applicable for the lower lip. The full-thickness labial advancement technique and upper labial pedicle rotation flap techniques are reviewed with specific attention to the application to the lower lip.

Full-thickness labial advancement technique (fig. 49.12)

After removing the tumor with a rectangular incision, the remaining lip caudal to the defect is mobilized by performing a full-thickness incision at the ventral aspect of the lip, parallel to the dental quadrant. The length of the mobilizing incision depends on the size of the defect needing repair. The flap should stretch over the defect without excessive tension. The lower lip is easier to advance than the upper lip and consequently often requires a shorter or sometimes no skin incision. It is the mucosa that is the restricting tissue and requires a more substantial releasing incision. The labial margins are accurately apposed and the edges of mucosa are reapposed to reconstruct the vestibule. Care must be taken to recreate the lower lip frenulum between the lip and the oral mucosa at the level of the space between the canine tooth and the first premolar tooth to prevent drooping of the lower lip. The skin is closed as a second layer. Tension sutures can be used in areas of moderate tension.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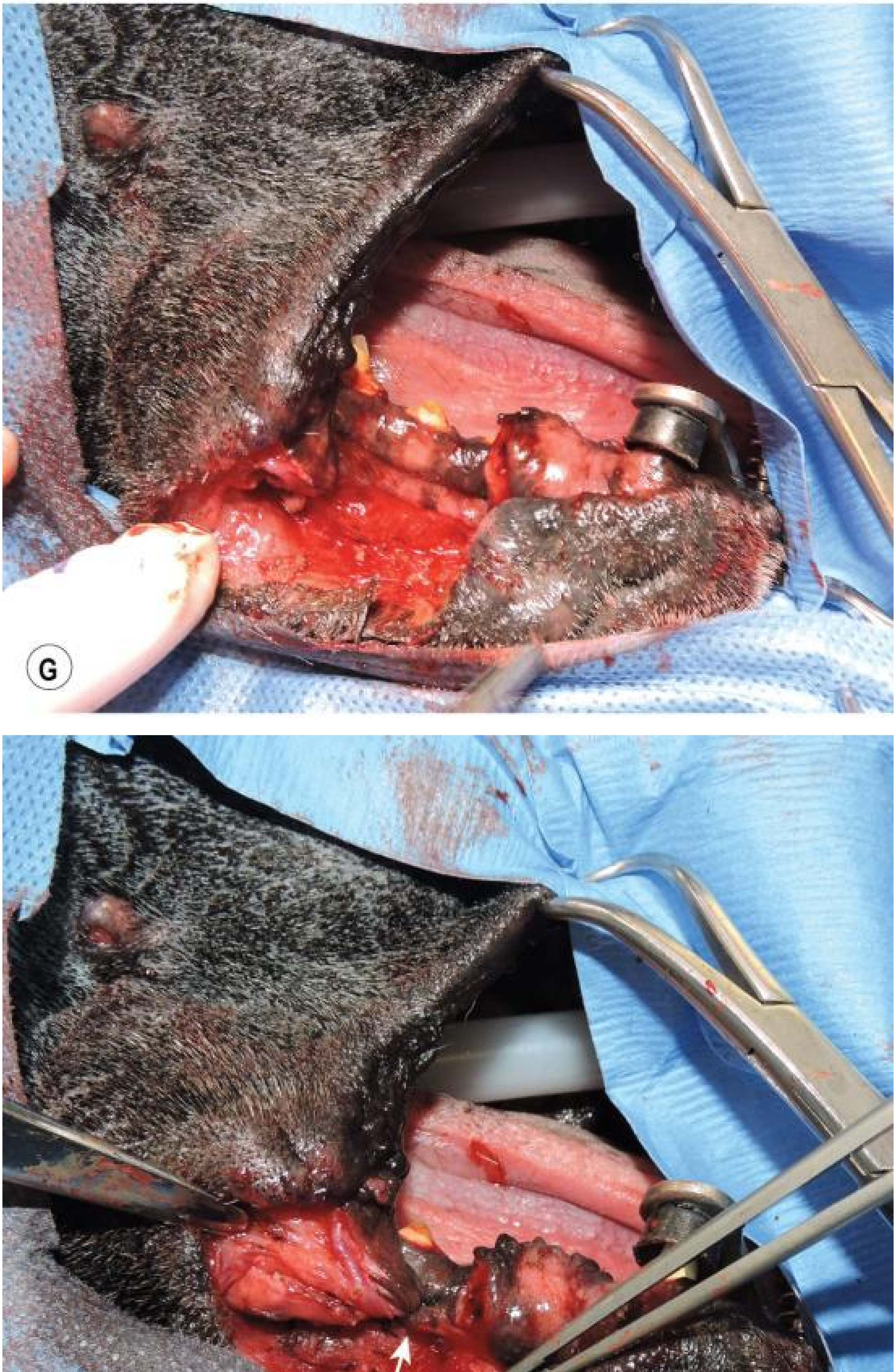
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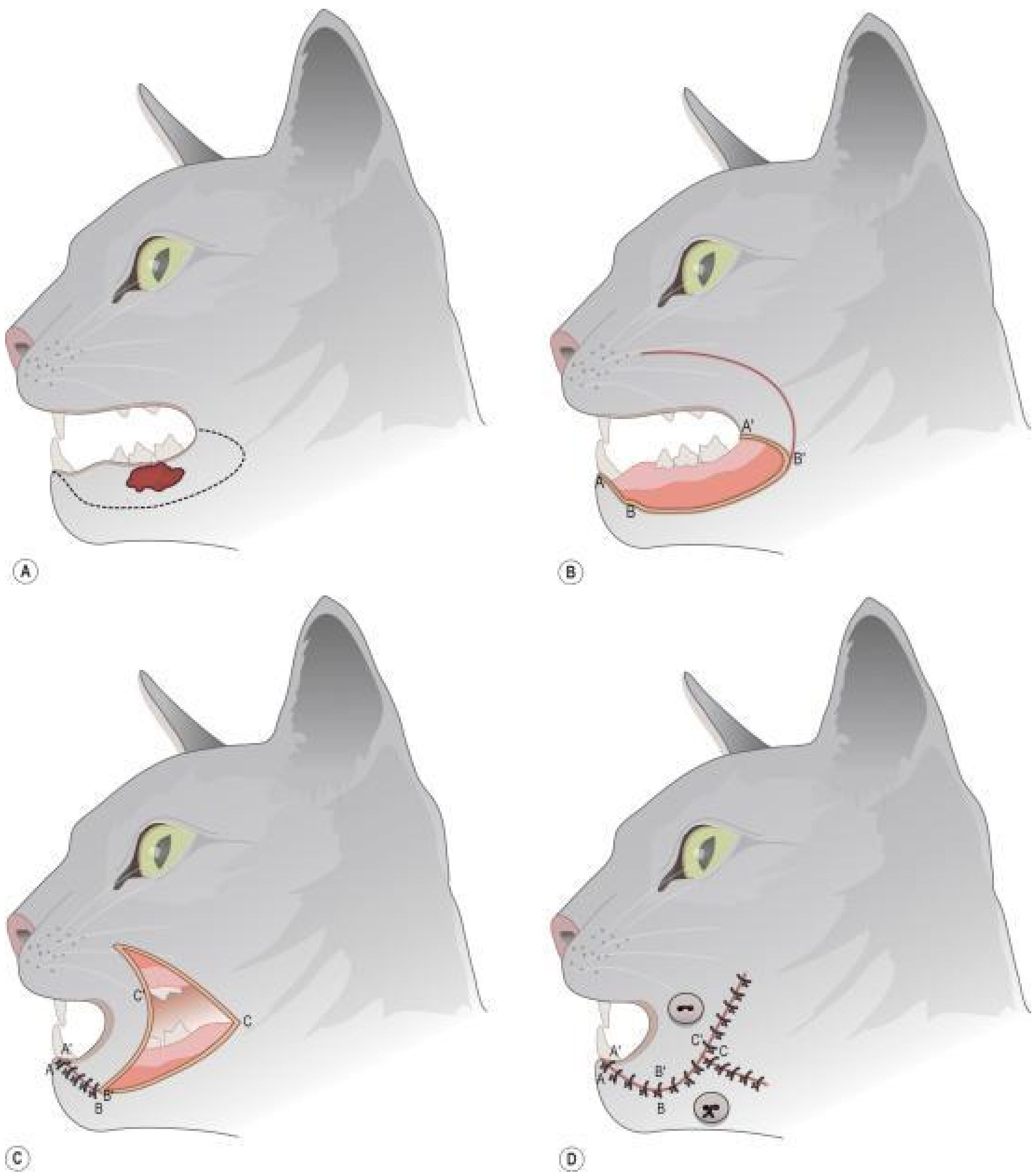
• **FIG. 49.12** Full-thickness labial advancement technique. (**A**) Proposed full-thickness rectangular incision around the lesion. (**B**) A full-thickness incision is performed at the level of the ventral extent of the excision, the incision being parallel to the dental quadrant or lip margin. The length of the incision will depend on the size of the defect. (**C**) Rostral traction on the lower lip helps determine the length of the incision required. (**D**) A two-layer closure is performed by first suturing the labial mucosa to the buccal mucosa. Sutures are placed to recreate the labial frenulum located between the mandibular canine and first premolar teeth. (**E**) The closure is completed by suturing the skin. (From Pavletic M. *Atlas of Small Animal Wound Management and Reconstructive Surgery*. 3rd ed. 2010. Reproduced with permission of Wiley-Blackwell, Ames, IA.) (**F**) Proposed excision of the right lower lip around a previous excisional biopsy site delineated by purple skin marker (the *asterisk* marks the commissure of the lips). (**G**) Full-thickness lip segment has been excised. (**H**) A releasing incision has been performed in the mucosa, along the gum line, both rostral and caudal to the defect. This has allowed advancement of the mucosa to reconstruct the defect. Arrow shows where the mucosal advancement flaps have been sutured together. Because this is the lower lip, which was easily mobilized, a releasing skin incision was not needed.

Upper labial pedicle rotation flap (fig. 49.13)

The tumor is excised with a wedge or rectangular excision. Approximately 80% to 90% of the lip margin (from rostral midline to commissure) can be excised. A full-thickness upper lip flap is created by making an incision parallel to the lip margin for a distance equal to the height of the lower lip. The incision begins at the caudal aspect of the lower lip defect and runs several centimeters rostrally (long enough to be able to appose it with the rostral edge of the lower lip defect). The flap is rotated after releasing the restrictive soft tissues from the maxilla such that the caudal edge of the flap meets the rostral edge of the lower lip defect. The margin of the upper lip flap becomes the margin of the lower lip where the defect was. The buccal mucosa is sutured. Small gaps can be left open to allow wider opening of the jaws. The skin is closed in an inverted "Y" fashion over the defect.



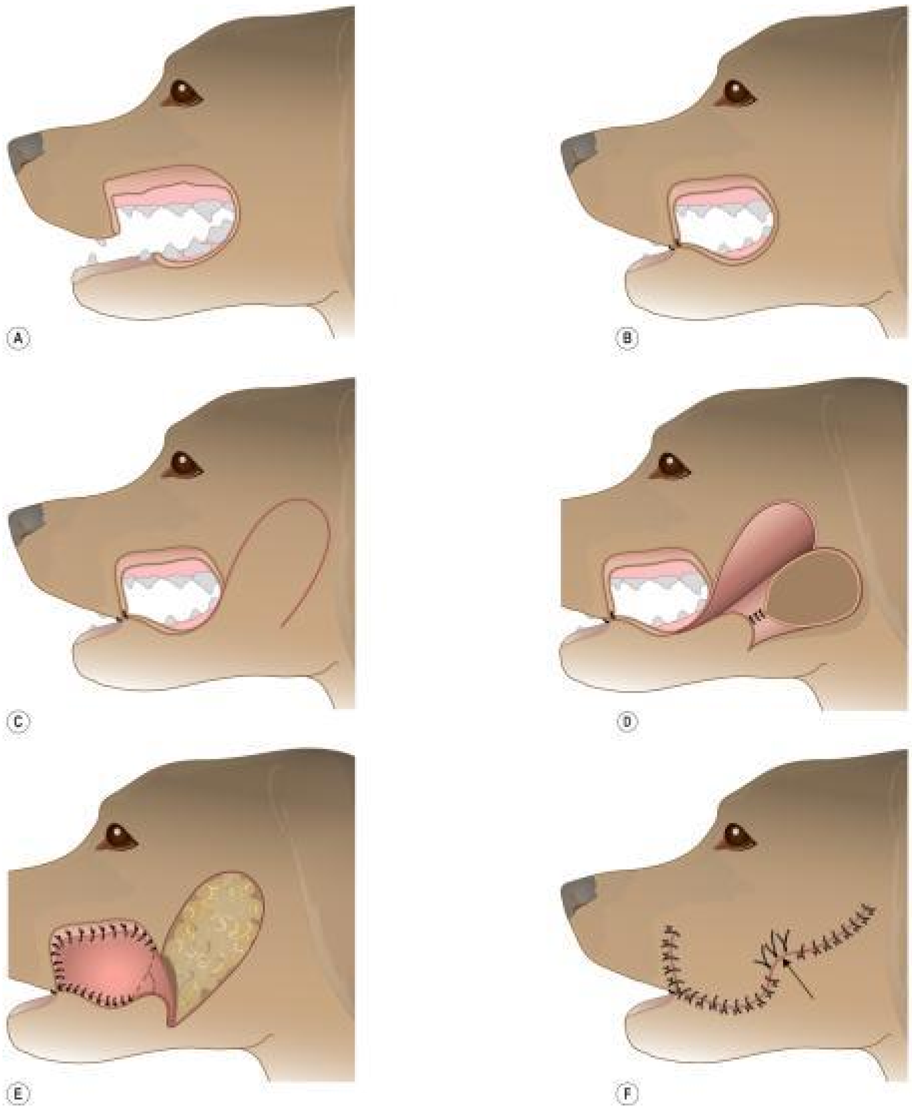
• **FIG. 49.13** Upper labial pedicle rotation flap. (**A**) Proposed dome-shape incision around the lesion. The defect will reach the level of the commissure caudally. (**B**) A full-thickness labial flap is created with the upper lip. A first incision is made at the commissure, perpendicular to the lower lip margin. The length is equal to the height of the lower lip. A second incision is made parallel to the teeth or lip margin starting at the first incision and running rostrally. The incision is long enough that it is possible to appose the flap to the rostral edge of the lower lip defect. (**C**) The labial flap is rotated and the lip margins are apposed (*A'* to *A*). The width of the flap is sutured to the rostroventral aspect of the defect (*B'* to *B*). (**D**) The point at the caudoventral aspect of the defect *(C)* is apposed to the caudal aspect of the flap without excessive tension (*C* to *C'*). A two-layer closure is performed. Because suturing the buccal mucosa completely can greatly reduce mandibular range of motion, a defect in the mucosa can remain and be allowed to heal by second intention. The skin is sutured in a "Y" fashion. This technique creates a significant rostral transposition of the opening of the mouth on the side of the surgery. Buttons may be placed dorsal and ventral to the suture line to relieve some of the tension and help prevent dehiscence by wide opening of the mouth.

Techniques for the lower lip, upper lip, and cheek reconstruction

Following excision of a tumor that creates a defect in the cheek, lower lip and upper lip, such large defects usually will require a skin flap for reconstruction.

Labial buccal reconstruction with inverse tubed skin flap[6] (fig. 49.14)

A full-thickness wide excision of a tumor has been performed. The upper and lower lips are apposed at the rostral end of their respective defect. A transposition flap is created in the skin adjacent to the defect. A portion of the flap is partially tubed in inverse fashion at the base of the flap such that the skin is lining the inside of the tube. The flap is reflected rostrally such that the skin becomes intraoral. The skin edges are sutured to the edges of the gingival mucosa. The skin dorsal and ventral to the defect is advanced over the flap and sutured. The skin adjacent to the defect created from the transposition flap is also advanced and sutured. A narrow opening is left over the short tubed segment. This tubed segment can be excised when healing is complete and neovascularization to the flap is adequate, i.e., 4 to 6 weeks postoperatively.



• **FIG. 49.14** Labial buccal reconstruction with inverse tubed skin flap. (**A**) A full-thickness excision of a lesion requiring removal of the caudal portions of the upper and lower lips and part of the cheek. (**B**) The lower and upper lip margins are apposed. (**C**) A transposition skin flap is created with its base being rostral. The flap is long enough to reach the rostral aspect of the defect and wide enough to reach the dorsal and ventral borders of the defect. (**D**) The rostral part of the flap is tubed. The length of the portion tubed is that part of the flap that will be reflected onto itself so that the skin distal to the tube can replace the buccal mucosal defect. (**E**) The flap is reflected rostrally and the skin edges are sutured to the mucosal edges in a simple interrupted pattern. (**F**) The skin is advanced over the transposition flap, inverse tube segment, and donor bed. There will be a narrow opening of the skin-lined tube *(arrow)*. Source: (From Pavletic M. *Atlas of Small Animal Wound Management and Reconstructive Surgery*. 3rd ed. 2010. Reproduced with permission of Wiley-Blackwell, Ames, IA.)

Free microvascular skin flap grafted with oral mucosa[8]

This technique has been used experimentally in dogs and may prove to be very valuable in certain instances in clinical settings (see Chapter 11).

Complications

Complications following glossectomy

Complications are infrequent and may include hemorrhage, dehiscence, partial tongue paralysis, necrosis of part of the tongue, and inability to eat or drink. Slow hemorrhage in the early postoperative period will usually resolve without any intervention, whereas significant hemorrhage usually requires heavy sedation or anesthesia and applying pressure for 5–10 minutes. If the hemorrhage persists, the sutures need to be removed and the source of bleeding identified so that it can be ligated or electrocoagulated (less desirable). Dehiscence rarely requires intervention. Small defects will heal by second intention. Larger defects may need resuturing. Necrosis of the rostral border of the tongue occurs when the blood supply has been compromised too much. In two dogs, debridement and resuturing were not necessary.[3] If the necrosis leads to anorexia or other systemic signs, surgical intervention would become necessary. Inability to eat or drink in the early postoperative period is probably the most common complication following glossectomy. Intravenous fluid therapy can be used to manage patients that are dehydrated, and a feeding tube (esophagostomy or gastrostomy tube) can be placed in order to feed the patient (see Chapter 5). The feeding tube can be placed under the same anesthesia as the glossectomy in patients that are more likely to have problems in the postoperative period, such as cats or dogs that undergo a large excision (i.e., 50% or more). It can take up to 4 weeks for dogs to be able to eat and drink unassisted following major glossectomy (removal of >75% of the tongue).[4] In one study of glossectomy, all dogs that had long-term follow-up recovered normal prehension.[12] In one study, one dog suffered a complication following a 100% glossectomy, which was ptyalism.[4] Thermoregulation could be a problem in hot and humid environments following significant glossectomy.[13]

Complications following excision of lip and cheek tumors and reconstruction

The most common complication is dehiscence. Most procedures lead to the rostral displacement of the labial commissure, which in turn leads to a decrease in how wide the mouth can be opened. When the animal is eating, opening the jaws can put too much tension on the incision line and cause dehiscence. To prevent dehiscence, tension sutures can be used. Some surgical techniques used to repair a large defect in the upper lip can lead to unilateral deviation of the nasal planum (e.g., full-thickness labial advancement technique) when there is enough tension on the rostral suture line from elastic retraction of the flap. This deviation usually subsides over the next 2 to 3 weeks following surgery. Infection is uncommon in spite of performing surgery in a contaminated site probably due to the rich vascular supply to the area. If the chosen technique left a large mucosal defect, the lip can undergo excessive contraction, which can lead to drooling and leakage of food when eating. The same outcome can occur if the chosen techniques failed to restore adequate lip function. For reconstruction of relatively large defects of the upper lip, it is not uncommon for the tip of the canine tooth to become exposed (Figs. 49.8H and 49.9G).

Prognosis

Prognosis following glossectomy

The prognosis for tongue tumors will vary with the site, type, and grade of tumor.[13] Tumors in the rostral tongue have a better prognosis for any of the following reasons: earlier detection since it is more likely to be seen by the client; the rostral tongue may have less abundant lymphatic and vascular channels as opposed to the caudal tongue; rostral lesions are easier to excise with wide margins.[13] Local recurrence of the tumor occurred in 28% of dogs after glossectomy and 56% of these dogs had an incomplete excision in one study.[12] Dogs with tumors ≥2 cm in diameter have a shorter survival.[12] Dogs with squamous cell carcinoma have a 1-year survival of approximately 50%,[3,13] but in another study the median survival was only 216 days.[12] In one study, no dog with complete excision of the squamous cell carcinoma had local recurrence.[3] The overall metastatic rate for lingual squamous cell carcinoma is 15% to 37.5%.[3,12] Histologic grade appears to be a prognostic factor for survival for dogs with lingual squamous cell carcinoma.[3] For malignant melanoma of the tongue in dogs, the median survival was not reached in one case series and was over 551 days,[14] but was only 241 days in another study.[12] The metastatic rate for lingual malignant melanoma has been reported to be 38%.[12] Hemangiosarcoma accounts for 6% of lingual tumors in dogs.[12] Hemangiosarcomas of the tongue in dogs are typically of low or intermediate grade.[15] Median progression-free survival was 524 days and median overall survival was 553 days in one study.[15] Although complete versus incomplete tumor excision was not prognostic for outcome in that study, all dogs with local recurrence had incomplete margins.[15] Granular cell myoblastoma of the tongue is a highly curable cancer. The prognosis for other tumor types is unknown due to the lack of information.[13] Long-term control of tongue tumors in cats is rarely reported, with a 1-year survival rate for tongue squamous cell carcinoma less than 25%.[13]

Prognosis following excision of lip and cheek tumors and reconstruction

The three most important prognostic factors are histologic type of the tumor (including grade if applicable), status of the surgical margins (i.e., complete versus incomplete excision), and stage. Tumors that are incompletely excised have a higher likelihood of local recurrence, and malignant tumors and tumors that have metastasized carry a worse prognosis. Little information exists regarding outcome and survival following excision of lip and cheek tumors specifically.

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CHAPTER 50

Maxillectomy techniques

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Definitions

Maxillectomy techniques involve removal of various segments of the incisive, nasal, maxilla, palatine, vomer, lacrimal, and zygomatic bones with adjacent neoplastic and normal soft tissues and teeth. The term “premaxillectomy” is often used in the veterinary literature to denote an excision confined to the incisive bone. However, the term “premaxilla” is not accepted veterinary anatomical nomenclature[1]; the term “incisivectomy” is therefore more appropriate. The term “hemimaxillectomy” is equally confusing and incorrect if used to describe the surgical excision of one maxilla. Removing most of one maxillary bone (typically combined with the excision of all or parts of the incisive and palatine bones) is a complete or total, unilateral “maxillectomy”. The term “hemimaxillectomy” could be used to denote the excision of half of one maxillary bone, but the term “partial maxillectomy” is more appropriately used for this. An “orbitectomy” involves removal of portions of bones that comprise the orbit including the maxilla, palatine, zygomatic, lacrimal, and frontal bones.[2]

Closure of the resulting oronasal surgical defects is accomplished using vestibular (i.e., alveolar and buccal) mucosal-submucosal flaps that may be combined with palatal mucoperiosteal flaps. An en-bloc excision is excision “as a whole,”[3] in this case referring to removal of the neoplasm and all soft and hard tissues within the boundaries of the predetermined surgical margins. An oronasal fistula is an abnormal opening connecting the oral cavity with the nasal cavity or maxillary recess.[4] In the dog, the maxillary recess extends approximately from the level of the distal root of the third premolar to the second molar teeth.[5] Alveolar juga are the smooth topographical elevations on the maxilla overlying maxillary tooth roots. Juga of the canine and fourth premolar teeth are the most prominent and easily palpated.[5]

Indications

Maxillectomy techniques are most commonly indicated for excision of malignant and benign oral neoplasms. They are also warranted as salvage treatment of severely comminuted maxillary fractures and treatment of maxillary osteonecrosis.

Background

Maxillectomy for the treatment of oral neoplasms was introduced by Withrow et al.[6] Clinical investigations for treatment of neoplastic[7–15] and nonneoplastic[16] oral conditions, wound healing,[17] and review publications[18–27] have followed, documenting healing and clinical treatment results.[28]

Preoperative concerns

Diagnosis

Whether an oral mass is the primary complaint or an incidental finding during physical examination, an early diagnosis should be aggressively pursued. The biologic behavior and clinical staging using TNM (tumor, node, metastasis) classification of patients with oral tumors have been described (see Chapter 42).[24,29] The initial database must include a complete physical examination including the oropharyngeal region and histopathologic diagnosis of a biopsy of the mass. The biopsy site is planned so it can be excised during the maxillectomy surgery. Appropriate imaging is obtained (see Chapter 6) as part of the clinical staging and surgery planning. Lack of radiographic evidence of bone involvement does not rule out malignancy.[30] Periosteal or bony invasion is assumed with any oral mass that is fixed to bone on palpation. Dental radiographs may help determine invasion along tooth roots in the periodontal ligament spaces. For most maxillary tumors, computed tomography (CT) with and without contrast or magnetic resonance imaging is indicated to determine the feasibility of surgery and to aid preoperative planning. Advanced imaging may help determine if the infraorbital canal or periodontal ligament spaces have been invaded.

Client communications

When clinical staging is completed, treatment options reviewed with the client, and surgery elected, several topics should be discussed with the client. These include (1) intent of surgery (i.e., local cure versus palliation) and prognosis, (2) operative concerns (i.e., blood loss, anesthesia risk), (3) postoperative appearance and oral function, and (4) recommended follow-up evaluation and treatment. The postoperative appearance and functionality of the patient are concerns for most clients. A photograph album including preoperative and postoperative photographs of patients that have had various types of maxillectomy aids this aspect of client communications.

Preoperative management

Antibiotics

A prophylactic course of antibiotics is administered intravenously in most patients. The first injection is administered at the time of anesthetic induction and repeated as needed (see Chapter 3). A therapeutic course is used in debilitated or immunosuppressed or immunocompromised patients. Commonly used intravenous antibiotics for oral and maxillofacial surgery are ampicillin and clindamycin. Oral formulations of amoxicillin-clavulanic acid are used to complete a therapeutic course if indicated.

Oral cavity disinfection

Preoperative periodontal treatment is recommended to reduce the bacterial load and allow a cleaner surgical environment. Elective oral surgery should not be performed in a patient with heavy dental calculus or extensive, active periodontal disease. In the event of severe oral infection secondary to periodontal disease, general oral health is addressed initially and maxillectomy performed in 7 to 10 days when the infection is controlled and the oral tissues are less inflamed. If dental extractions are required, it is prudent to avoid manipulation of the tumor and minimize exfoliation of cells. Rapid growth of the neoplasm may preclude delay in surgery.

Surgical preparation of the oral cavity prior to maxillectomy is routine (see Chapter 7).

Anesthetic considerations

As with any major surgery, a preoperative minimal database should consist of physical examination, complete blood count, serum biochemistry profile, and urinalysis. Coagulation parameters are screened by means of the mucosal bleeding test.[31] Further tests for coagulopathies are indicated if the mucosal bleeding test results are abnormal or if the patient has a history of bleeding tendency. The presence of von Willebrand disease should be evaluated in Doberman pinschers. Extensive excisions have greater blood loss potential. Blood typing and cross-matching are routinely performed. Blood products and colloidal fluids should be available in the event of severe blood loss. Placement of two intravenous and one arterial catheter is recommended.

Preemptive analgesia

A multimodality approach to analgesia administered before the onset of surgery is most effective (see Chapter 4).[32] Typically, the patient receives an opioid and a nonsteroidal anti-inflammatory drug prior to induction of general anesthesia and intubation. Regional blocks are routinely used, except if the tumor is located in the immediate vicinity of the nerve to be blocked. The surgeon should be familiar with the applicable regional anesthesia techniques, mainly the infraorbital block (see Chapter 4). Bilateral blocks may be needed, depending upon the proposed excision.

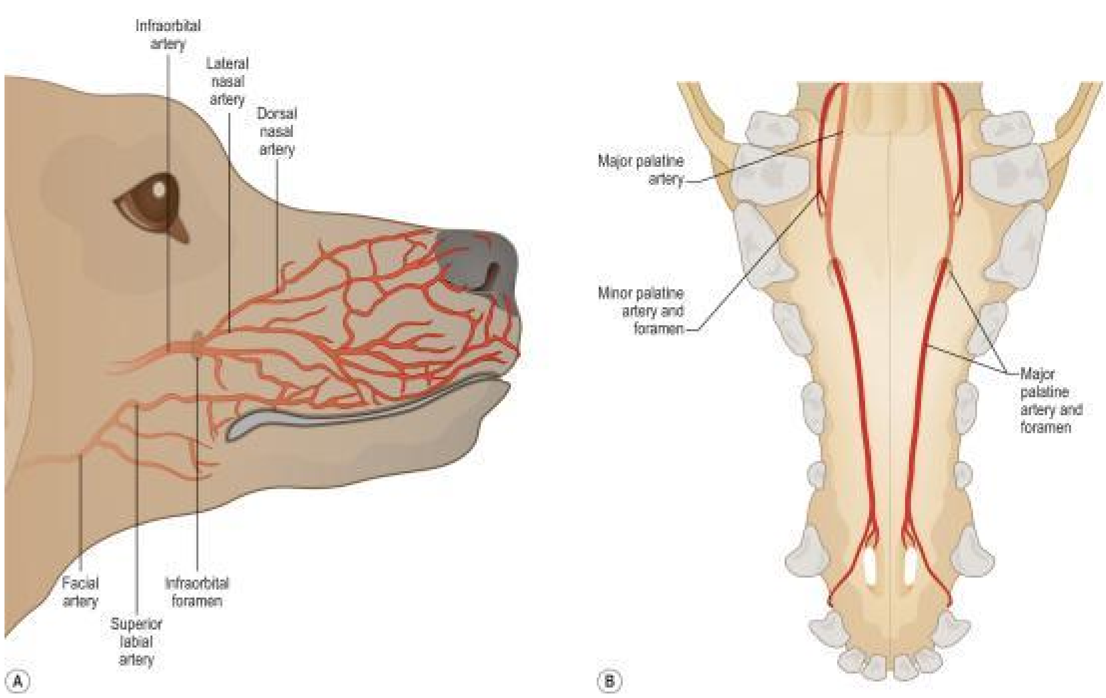
Patient positioning

For the majority of patients, positioning in dorsal recumbency allows good visualization of the surgical field (see Chapter 7). The head is secured into position with a vacuum-charged surgical positioning system (Vac-Pac®; Olympic Medical, Seattle, WA). A mouth gag is positioned away from the surgery site to keep the mouth open. The endotracheal tube is secured to the lower jaw and the tongue is retracted from the surgery site. Alternatively, a pharyngotomy intubation can be performed (see Chapter 61). The table may be slightly tilted to lower the head and provide gravity-dependent drainage for irrigation fluid and blood. Visualization of the surgery site in patients with larger masses extending more dorsolateral on the maxilla can be improved by tilting and rotating the head to allow adequate access to the palate and dorsolateral surface of the maxilla.

After positioning, a pharyngeal pack made of absorbent exodontia sponges (Cotton-filled Exodontia Sponges; Crosstex International Inc., Hauppauge, NY) is placed in the pharynx around the endotracheal tube to prevent blood and irrigation fluids from entering the pharynx, the airway, and the esophagus (see Chapter 7).

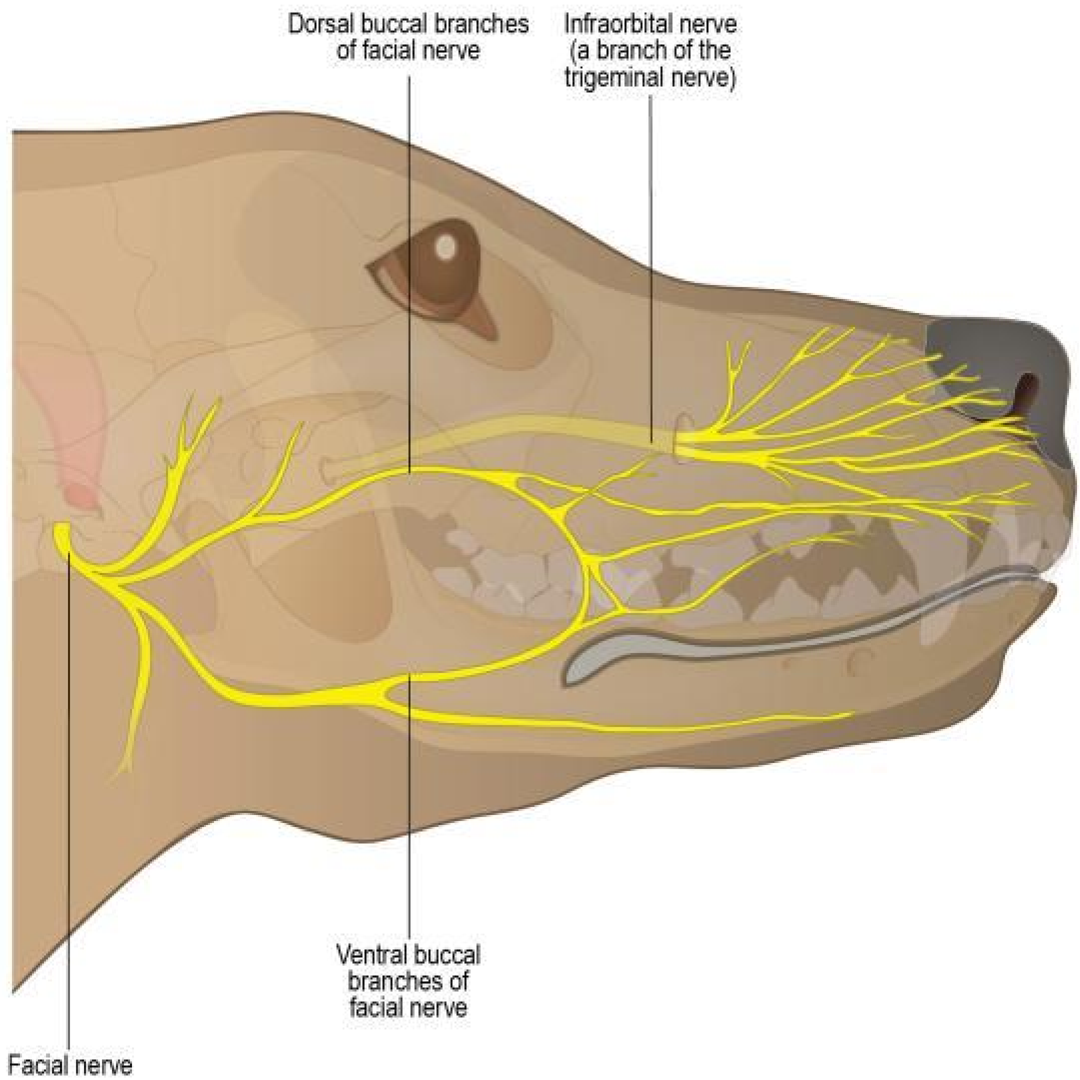
Surgical anatomy[5]

The major and minor palatine, sphenopalatine, and infraorbital arteries originate from the maxillary artery. The malar artery originates from the infraorbital artery. The facial artery originates from the external carotid artery (Fig. 50.1). The greater palatine foramen is located at a level approximately equal to the distal surface of the maxillary fourth premolar tooth and midway between the palate midline and dental arch. The major palatine artery, vein, and nerve exit this foramen and course along the length of the palate in the palatine sulcus. The palatine sulcus is located halfway between the midline and dental arch, making it relatively easy to find and ligate the major palatine vessels. The artery enters the nasal cavity through the palatine fissure as the rostral septal branch of the nasal septum. At the level of the palatine fissure, an arterial branch originates from the major palatine artery and courses on the palate through the interdental space between the canine and third incisor teeth to terminate on the lateral aspect of the incisive bone. This artery may be transected during incisivectomy techniques. The sphenopalatine artery terminates in many branches supplying the nasal cavity, septum, and conchae. The infraorbital artery exits the infraorbital foramen and divides into the lateral nasal and dorsal nasal arteries, which supply the soft tissues of the muzzle. The lateral nasal artery anastomoses with the superior labial branch of the facial artery. These arteries provide the blood supply to the vestibular mucosal–submucosal flaps commonly used to close maxillectomy defects. Terminal branches of the malar artery may be encountered during partial orbitectomy.



• **FIG. 50.1** Blood supply to the upper jaw. (**A**) Lateral aspect. (**B**) Palatal aspect.

The major nerves encountered are the dorsal buccal branch of the facial nerve and the infraorbital nerve (Fig. 50.2). The dorsal buccal nerve provides motor innervation for the muscles of the upper lip. This nerve is normally not injured during maxillectomy and motor control of the muscles is maintained. The infraorbital nerve originates from the maxillary division of the trigeminal nerve and provides sensory innervation to the nose, upper lip, nasal mucosa, and maxillary teeth. Transection of this nerve during maxillectomy creates sensory loss that is typically not clinically relevant. The branches of both nerves are visible during the reflection of a vestibular mucosal–submucosal flap for maxillectomy defect closure.

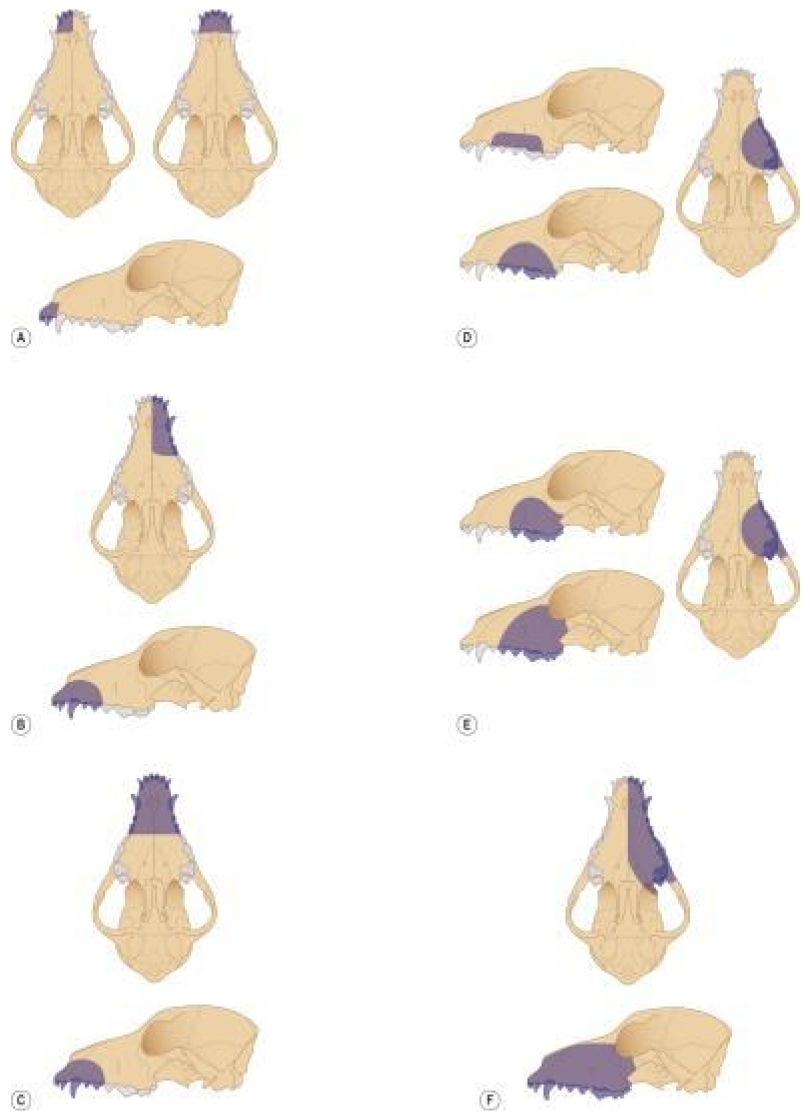


• **FIG. 50.2** Some of the superficial branches of the facial and trigeminal nerves.

Therapeutic decision-making

Types of osseous excisions

The general types of maxillectomy techniques are shown in Fig. 50.3. The extent of any excision is determined by the oral physical examination and diagnostic imaging findings, the histopathological diagnosis, the recommended surgical margins, the intent of the surgery, and the tissue available for defect closure.



• **FIG. 50.3** Major types of maxillectomies *(shaded areas)*. (**A**) Unilateral and bilateral incisivectomy. (**B**) Unilateral rostral maxillectomy. (**C**) Bilateral rostral maxillectomy. (**D**) Central or segmental maxillectomy. (**E**) Caudal maxillectomy. (**F**) Unilateral (complete) maxillectomy.

Surgical technique

General surgical principles

Surgical plan [23–26,33]

The surgical plan for a maxillectomy procedure includes the following important points (1) plan for adequate surgical margins; (2) plan for complete mucosal closure; (3) perform atraumatic surgery and preserve local blood supply; (4) avoid or minimize the use of electrosurgery or laser; (5) harvest large mucosal flaps to avoid suture line tension; and (6) support suture lines with grossly healthy bone if possible.

Surgical margins

Surgical margins are indicated by the biological behavior of the neoplasm as determined from the preoperative histopathological diagnosis, diagnostic imaging findings of bone, and soft-tissue involvement (i.e., CT with and without contrast) and oral physical examination findings (see Chapter 48). The preoperative histopathological diagnosis is essential for definitive surgical planning. In general, a minimum of 10-mm margins should be obtained for all benign and malignant neoplasms except fibrosarcoma, where a minimum margin of 15–20 mm is recommended due to the higher local recurrence rate.[23–26] However, studies evaluating the appropriate surgical margins in dogs and cats are lacking, and these recommendations have not been correlated with clinical outcome. Recently, evaluation of histopathological margins and clinical follow-up in 23 dogs that had mandibulectomy or maxillectomy for the excision of canine acanthomatous ameloblastoma revealed that 34.8% of dogs had complete margins, 43.5% had narrow margins, and 21.7% had incomplete margins. However, despite the variability in the margins obtained, no local recurrence was clinically noted by either the owner or the clinician in any of the patients, including those with incomplete margins.[34] Wider margins can be obtained for malignant neoplasms if a tension-free oral mucosal reconstruction can be obtained. The surgical margins are measured and marked around the circumference of the mass using a sterile surgical marker before starting the surgery. If the neoplasm is found to penetrate cortical bone and enter the nasal cavity, the adjacent turbinates are excised.

Postoperative radiographs of the excised specimen or surgery site may be obtained to document adequate gross surgical margins and the removal of all dental structures associated with the tumor. The entire specimen is sent for histopathological examination to confirm the diagnosis and to assess the bone and soft tissue margins for neoplastic cells. Ideally, the margins of the specimens should be color coded for determination of surgical margins using the Davidson Marking System® (Bradley Products Inc., Bloomington, MN).[35] Finding neoplastic cells on the excision margin implies incomplete removal of local microscopic disease and the need to consider extending the excision site, adjunct radiation, or chemotherapy.[25]

Surgical incision

The incision is made along the measured and marked surgical margins and the incision is taken to the bone. Once incised, the alveolar mucosal margins may retract and result in some distortion of proposed margins. The described incision method maintains accurate surgical margins. In the regions of larger vessels and nerves, the dissection is continued to identify, ligate, and transect the vessels and transect the nerves. Hemorrhage is controlled by ligation and digital pressure and the use of electrocoagulation should either be avoided or be kept to a minimum.[23–26]

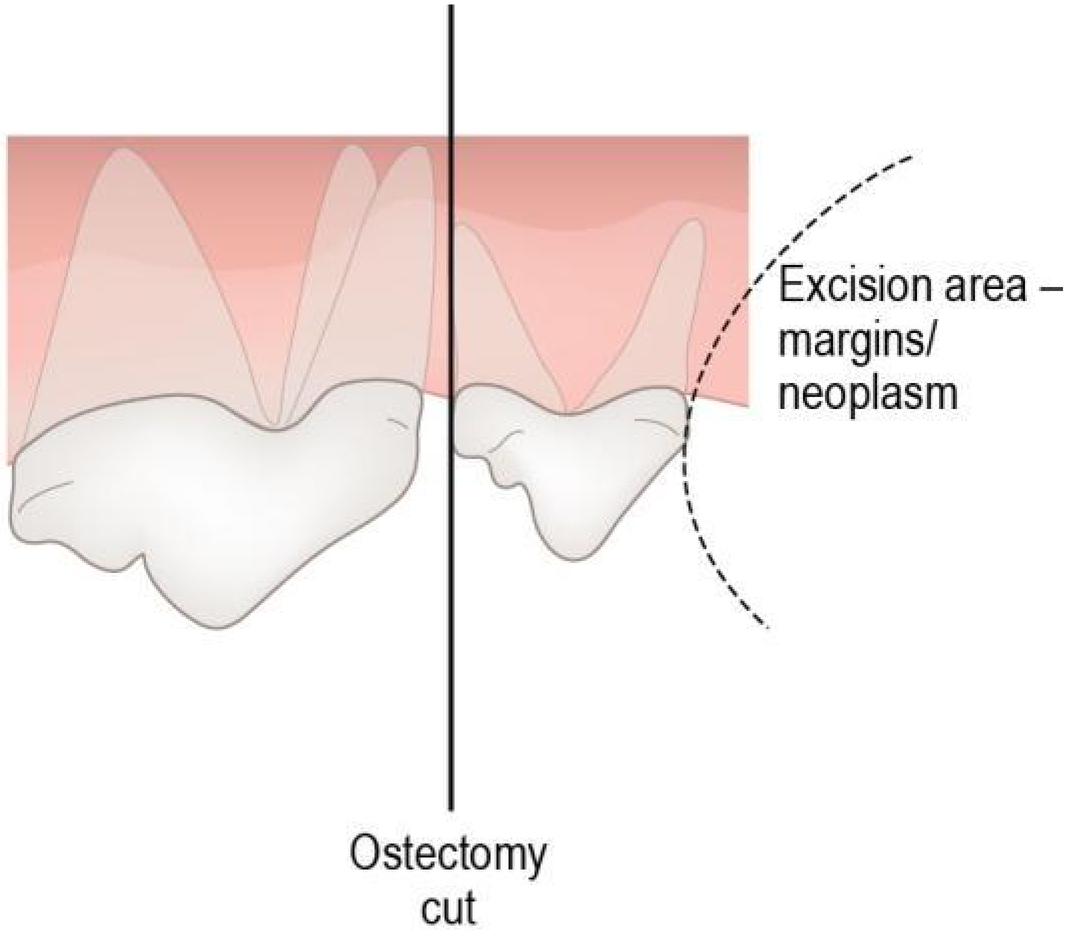
A combined extraoral and intraoral approach has been described for tumors that are located at the caudal maxillas. In this approach, a dorsal skin incision is created first, just lateral to the midline and extending caudally as necessary (i.e., extraoral approach) and followed by an incision through the buccal mucosa (i.e., intraoral approach), effectively creating a bipedicle flap of skin, buccal mucosa, and associated tissues of the lateral aspect of the nasal cavity. This allows visualization of the entire lateral aspect of the maxilla and access to perform the ostectomy.[36]

In addition, for extensive tumors of the rostral maxilla that involve the oral mucosa and the skin, a combined intraoral and extraoral approach that allows a composite excision of tumor has been described.[37] In this approach, an extraoral ellipsoid skin incision is made, dictated by the margins of the lesions as seen grossly and by CT followed by an intraoral incision to result in communication of the two incisions. This allows an access for ostectomy of the rostral maxilla and results in a composite of skin, oral mucosa, associated soft tissues, and bone.

To obtain adequate tumor-free margins, a segment of skin may need to be excised.[25] This is achieved using a rostral-caudal–oriented fusiform excision and linear closure. Rostral-caudal orientation generally parallels the course of most neurovascular structures and reduces iatrogenic injury potential to these structures. Concurrent lip wedge excision may be needed to obtain tumor-free surgical margins.

Management of adjacent teeth

Maintaining teeth is secondary to achieving adequate surgical margins and tension-free closure of the surgical defect. Ostectomy performed at a narrow interdental space may result in injury requiring extraction of a tooth adjacent to the ostectomy site if inadvertent severe periodontal or endodontal injury (pulp cavity exposure) occurred during ostectomy. Ostectomy at the level of a wider interdental space may not result in similar tooth injury. Therefore, to minimize tooth loss, the ostectomy margins are planned to transect a tooth at the margin level (Fig. 50.4). This may require margin extension, which is acceptable. Margins should never be compromised to save teeth. Once the en-bloc tissue mass is excised, the remnant of the transected tooth is extracted. The adjacent teeth and periodontal structures are not injured, leaving a margin of attached and free gingiva adjacent to the ostectomy. The alveolar bone covering the roots of adjacent teeth must remain intact. Careful attention is needed, with additional bone removal around adjacent teeth, as it may be very difficult to visually differentiate bone from dentin. Removal of this alveolar bone results in chronic root exposure to the nasal cavity and will result in periodontal disease, probable endodontal involvement, and abscess formation. Inadvertent root exposure requires extraction of the involved tooth.



• **FIG. 50.4** Performing the ostectomy at the line angle of the tooth on the periphery of the excision will not damage adjacent teeth. If root fragments remain, they must be removed after the excision is completed and the adjacent bone must be smoothed prior to closure.

The apex of the maxillary canine tooth is located dorsal to the maxillary second premolar tooth. Therefore, rostral excisions to ensure complete removal of the alveolus of the canine tooth include the first and second premolar teeth.

Ostectomy and osteoplasty

The soft tissues are subperiosteally elevated away from the ostectomy site. The ostectomy is performed as determined by the premeasured surgical margins. The excision is planned so that all root structures and preferably all intact alveoli of teeth within the surgical margins are part of the en-bloc excision.

Any remaining tooth root fragments from transected teeth at the excision periphery are extracted after the en-bloc tissue segment is removed.

Ostectomy should be performed with care and precision to minimize damage to vital structures such as blood vessels, nerves, the eye and the brain. Therefore, it is highly recommended that the ostectomy be performed using a piezotome (see Chapter 9), especially when ostectomy is performed near the maxillary artery, the major palatine artery, or infraorbital canal. In addition, precision power equipment that cuts bone with burs offers an efficient ostectomy method. Ideally, a surgical handpiece and bone-cutting burs are used with precision and control of the instrument. Ostectomy margins can be cut with rounder borders that facilitate flap closure as opposed to the straight cuts and sharp angles that result when using a saw blade.

Carbide steel bone-cutting burs may leave a roughened edge. Medium-grit diamond tapered cylinder burs produce a smooth cut. A thin osteotome and a mallet may be used with care. The instruments are used with gentle technique to slowly advance the depth of bone cut. Aggressive use may result in fractures of remaining bone and injury to nasal cavity and periocular tissues and the eye. Increased resistance will be found when tooth roots are encountered and may require extraction of the tooth or deviation of the ostectomy course to include the entire tooth. Due to the lack of precision, the risk of damage to the surrounding tissues, and difficulty irrigating the area with subsequent risk of necrosis at the ostectomy site, the use of an oscillating saw is not recommended.

Once the en-bloc tissue segment is removed, osteoplasty is performed. Cut bone margins are smoothed with a round medium- or fine-grit diamond bur or carbide bur. The goal is to remove all sharp bone ridges and bone spikes that may result in ischemic pressure injury and potential perforation of the vestibular mucosal–submucosal flap. The surgery site is irrigated to remove all bone debris before closure.

Flap design

General principles

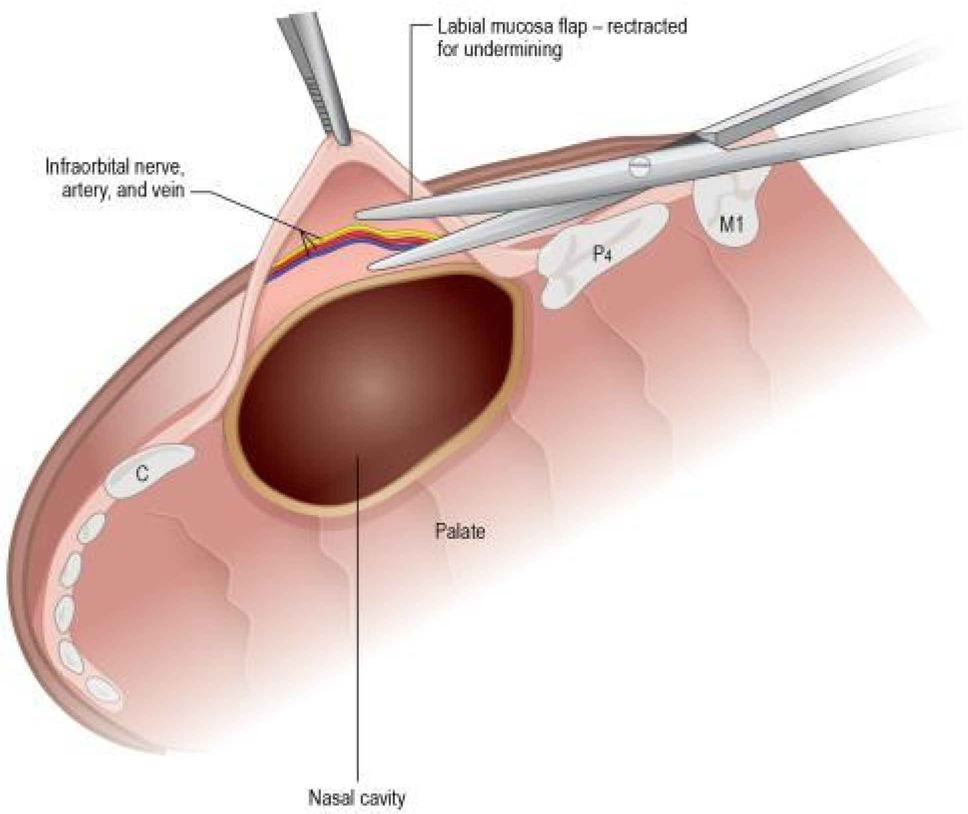
Recommended oral mucosal flap length to base-width ratio is 2:1 in humans[33] and is unknown in the dog and cat. In general, the flap is made slightly larger than necessary to ensure adequate tissue for tension-free closure and should, ideally, not have vertical releasing incisions as flap blood supply may be compromised.[24] If vertical releasing incisions are needed, they are divergent from the flap apex to base to maintain a broad-based flap for blood supply conservation.[33]

The flaps are gently handled. Excessive pulling, crushing, or desiccation may injure flap blood supply.[33] Tissue forceps are not closed tightly to avoid crushing of the tissue and potential marginal ischemic necrosis. Thin tissue flaps may be atraumatically manipulated by placement of stay sutures. Defect closure is most commonly achieved using local mucosal tissues. Advanced closure techniques are discussed in Chapter 52.

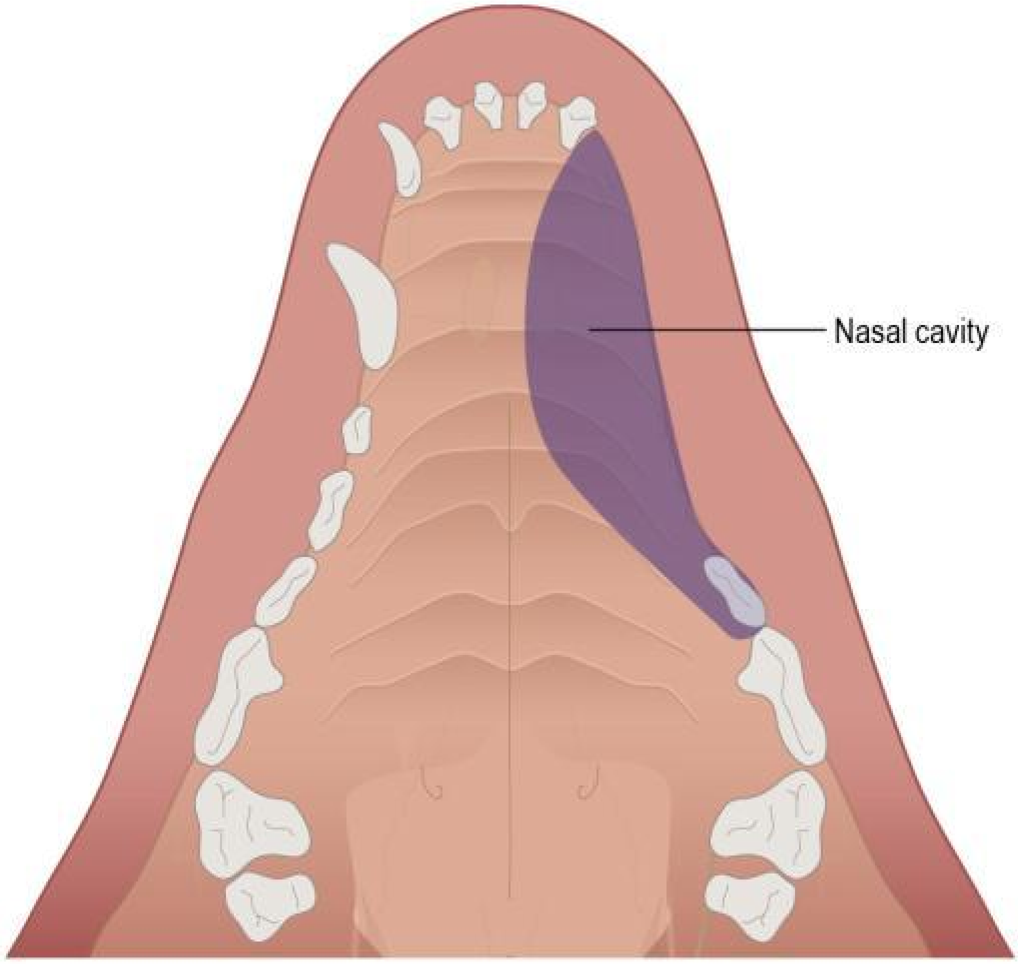
Vestibular mucosal–submucosal flap

The amount of vestibular mucosa that remains for closure is determined by mass size and surgical margins. A lesser amount of remaining vestibular mucosa-submucosa results in a larger concave facial defect with lip margin distortion. A larger flap improves the cosmetic appearance without compromising the surgical margins.

The flap is harvested by dissecting it from the maxillectomy site toward the lip margin.[23–26] The plane of dissection is in the areolar tissue immediately adjacent to the submucosa. As much connective tissue as possible is harvested with the flap. Branches of facial and infraorbital arteries, veins, nerves, and adjacent skeletal muscle tissue are not incorporated into the flap. These structures are visualized during the dissection but remain in original position on the cheek (Fig. 50.5). The rostral and caudal flap ends remain intact, and vertical releasing incisions are optional. Flap elevation is continued until enough tissue is present to allow a tension-free closure. Dissection toward the lip margin and harvesting additional tissue beyond that needed for a tension-free closure may help reduce the degree of postoperative facial concavity. The flap blood supply is from the superior labial branch of the facial artery and lateral nasal branch of the infraorbital artery (see Fig. 50.1).[5] The flap is contoured as needed to fit the surgical defect. Extraction of an additional tooth adjacent to the surgical defect and removal of the alveolar bone to taper the defect margin may aid closure (Fig. 50.6). Further reduction in tension may be obtained by additional flap elevation or by using an alveolar transposition flap and/or a palatal mucoperiosteal flap. The nonepithelialized surface of the vestibular mucosal–submucosal flap that will line the ventral aspect of the nasal cavity after defect closure is covered with nasal epithelium by 1 to 2 weeks postoperatively.[16]



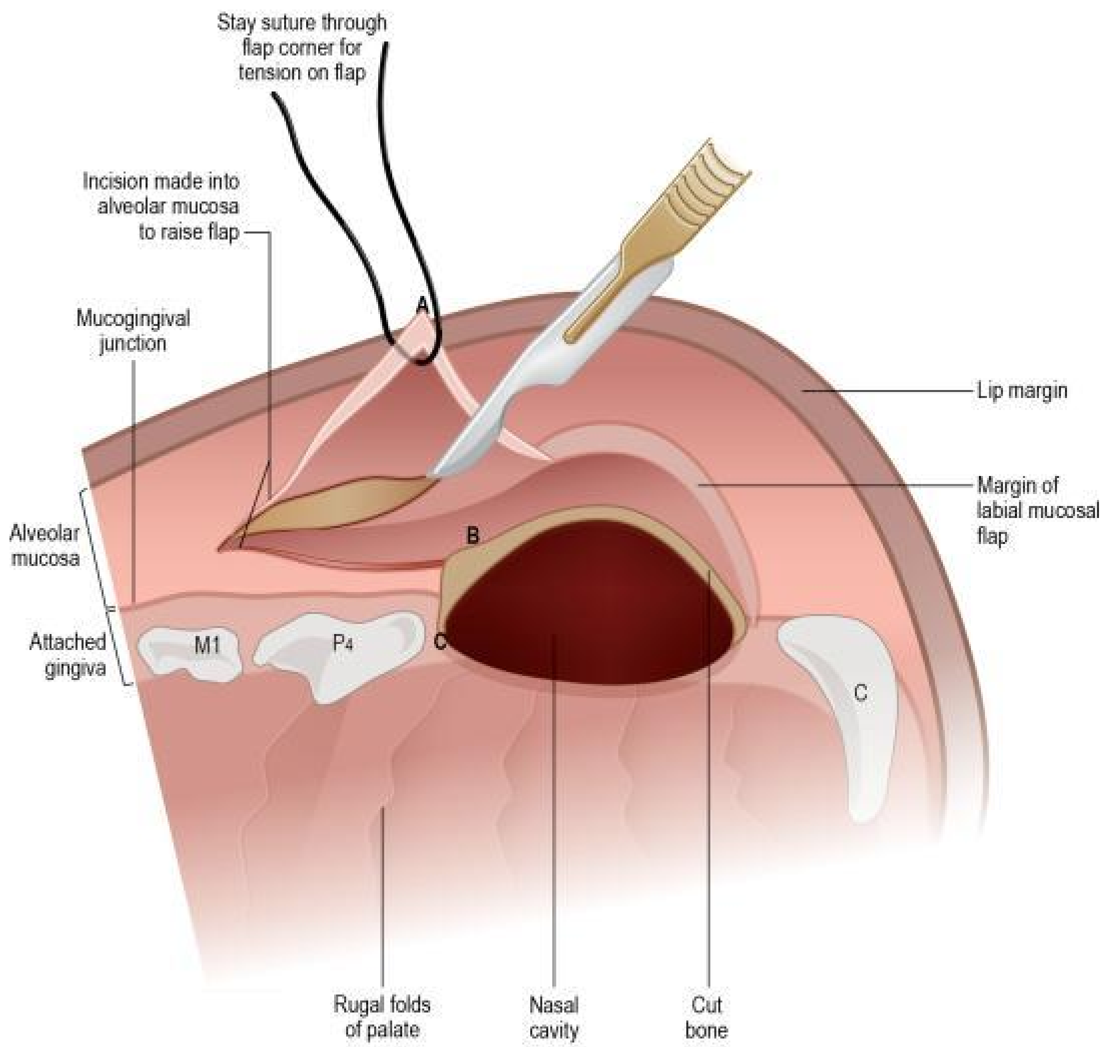
• **FIG. 50.5** The vestibular mucosal–submucosal flap is elevated from the maxillectomy site toward the lip margin. The exposed facial and infraorbital artery and nerve branches and skeletal muscle remain in original position and are not injured.



• **FIG. 50.6** An additional tooth and alveolar bone can be excised, if needed, to facilitate vestibular mucosal–submucosal flap closure.

Alveolar mucosal–periosteal transposition flap

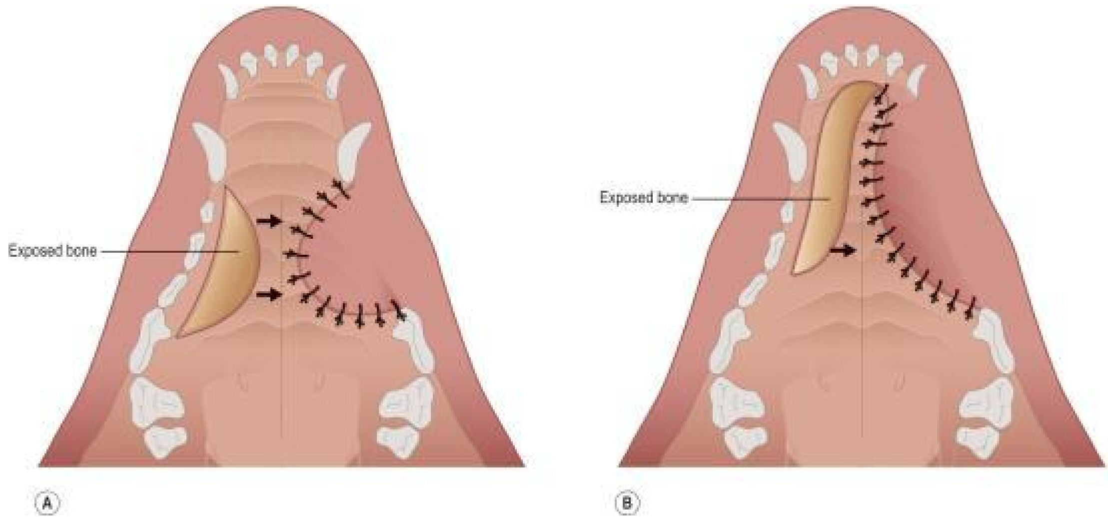
This flap is confluent with the vestibular mucosal–submucosal flap and is used, if needed, to reduce suture line tension that may be found at the defect ends during closure. An alveolar mucosal-periosteal incision is made on one or both ends of the surgical defect (Fig. 50.7). The incision parallels and is dorsal to the mucogingival junction, thereby leaving a 1–2-mm strip of alveolar mucosa attached to the junction for incision closure. The alveolar mucosa is subperiosteally elevated. A stay suture is placed in the corner of the flap and the flap is placed under tension. The periosteum at the level of the flap base is incised in a rostrodorsal oblique angle for caudal-based flaps and in an oblique caudodorsal direction for rostral-based flaps. Care is taken to not incise infraorbital neurovascular structures. If necessary, the periosteal releasing incision is continued parallel to these neurovascular structures. Releasing the periosteum allows a gain in length of this tissue and in effect provides additional tissue for closure around the rostral and caudal aspects of the bone defect.



• **FIG. 50.7** An alveolar mucosa–periosteal advancement flap is elevated. An incision is made dorsal and parallel to the mucogingival groove. The alveolar mucosa is subperiosteally elevated. The flap corner *A* was harvested from *B*. A stay suture is placed in the flap corner and the flap put under mild tension while incising the periosteum. When the periosteum along the base of the flap is incised as shown, the flap length increases. *A* will be sutured to *C*. This technique facilitates closure of the end(s) of the excision site and reduces tension in the area. Note that a rim of alveolar mucosa is left dorsal to the mucogingival groove to facilitate closure of the incision.

Palatal mucoperiosteal flap

This flap may be used to reduce closure tension and, to some degree, reduce uncosmetic deformity. It is considered when a reduced amount of labial mucosa may be present for closure of rostral and central maxillectomies. An incision is made in the mucoperiosteum along the palatal aspect of the dental arch opposite to the surgery site, and the mucoperiosteum, with greater palatine artery and vein, is subperiosteally elevated (Fig. 50.8). In larger patients with thicker mucoperiosteum, a thick split-thickness flap is harvested with a scalpel blade and periosteal elevator in an attempt to leave some periosteal tissue covering donor site bone. The major palatine artery and vein must remain intact. The flap is placed into the new position. The donor site rapidly heals.

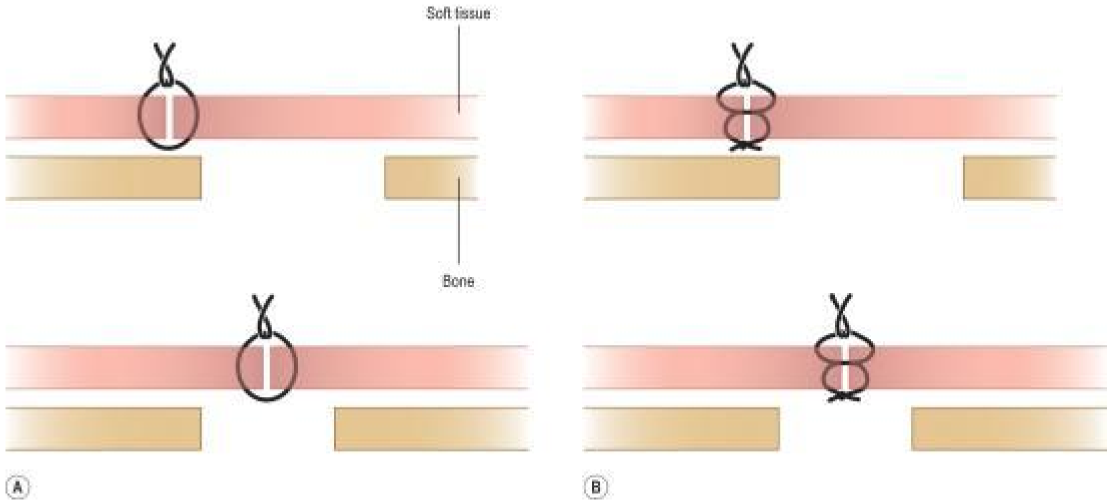


• FIG. 50.8 Variations of a palatal mucoperiosteal flap to help reduce tension and deformity when a limited amount of vestibular mucosal–submucosal flap tissue is available. Use in a central (**A**) and unilateral (**B**) rostral maxillectomy closure. An incision is made parallel to the dental arch. The mucoperiosteal flap with major palatine blood supply is subperiosteally elevated. The exposed donor site rapidly heals.

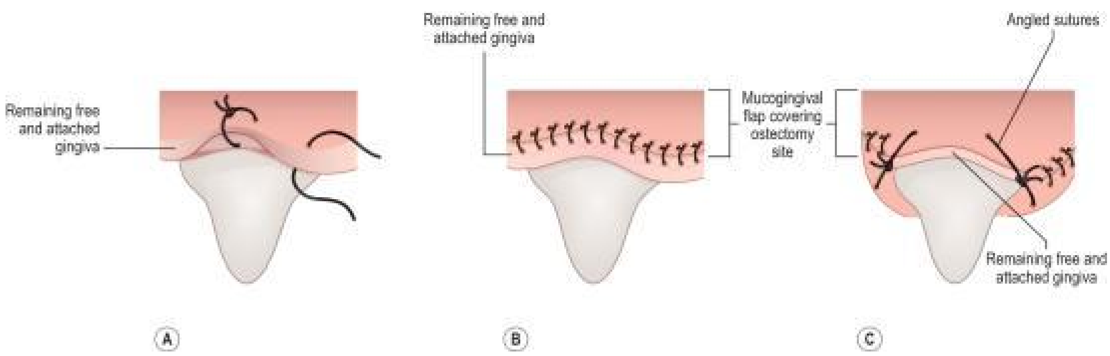
Surgical defect closure

Healing without complication depends largely on accurate tension-free apposition of incised tissue margins (palatal mucoperiosteum and alveolar and labial mucosa-submucosa). Inverted epithelium from poor approximation and suture-line tension delays wound healing. A single-layer mucosal closure using a 4-0 synthetic monofilament absorbable suture material in a simple interrupted fashion is performed and two-layer closure is optional. Ideally, the suture line is supported with healthy bone to help reduce suture line stresses.[33] Bone support is often impossible; however, an attempt should be made to support at least a portion of the suture line. To achieve this, a 1–3-mm wide strip of palatal mucoperiosteum may be excised to leave an exposed bone margin. During closure, the suture line will be over this bone margin.

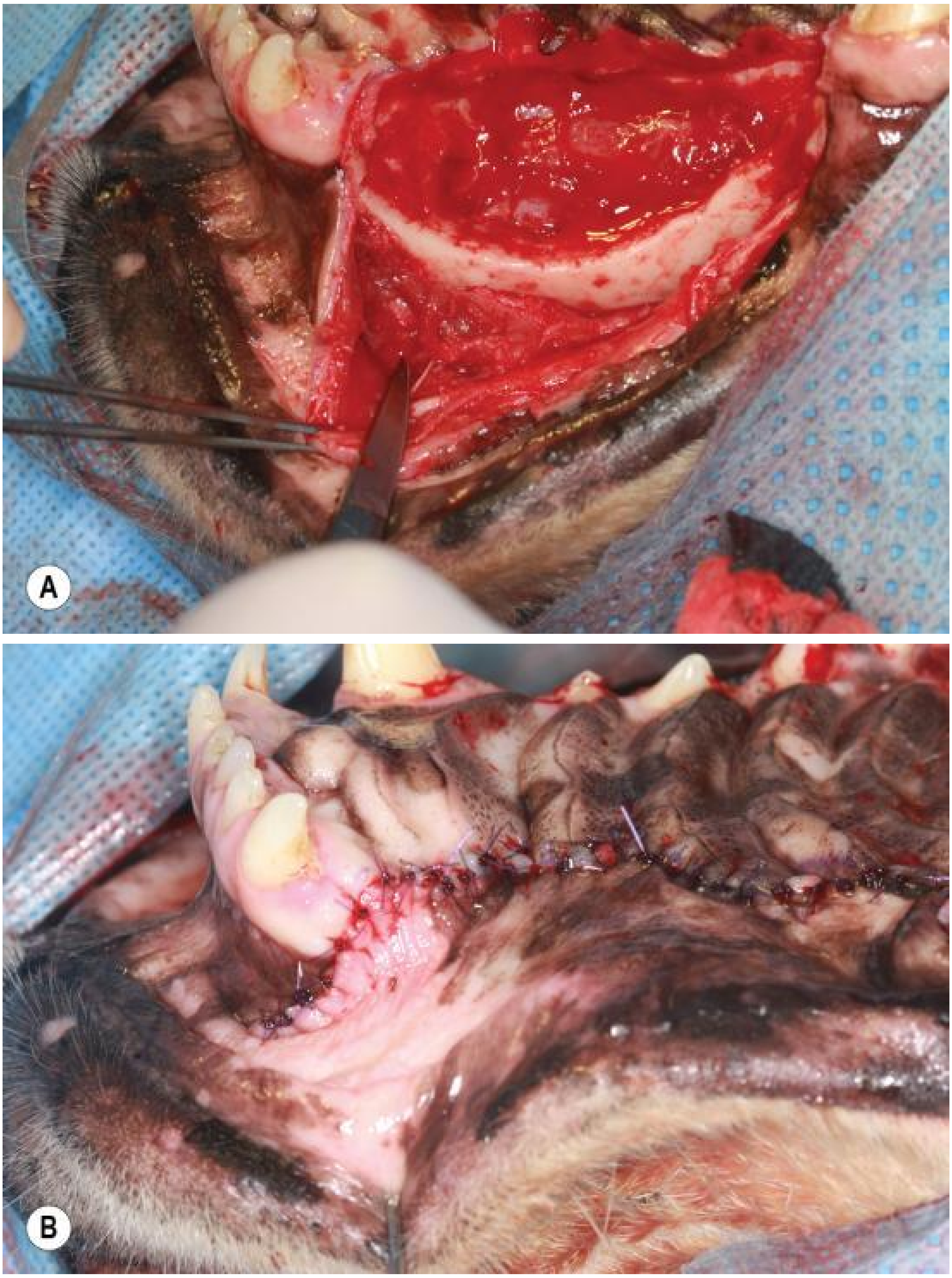
The margin of the palatal mucoperiosteum is subperiosteally elevated to facilitate suture placement. Simple interrupted sutures are always used, with knots placed in the oral cavity. If a two-layer closure is used, a simple interrupted pattern is used for the deep layer and deep bites are taken in the submucosa; the knots are placed on the nasal side. The mucosal layer is apposed with a simple interrupted or simple continuous pattern with knots placed in the oral cavity (Fig. 50.9). A continuous pattern minimizes the number of suture knots and cut ends in the oral cavity that could irritate glossal and labial mucosa and accumulate food debris. The rostral and caudal ends of the surgical defect are closed first, followed by closure of the central portion of the defect. This method, combined with adequate flap tissue, ensures tension-free tissue apposition along the entire closure length by providing an even distribution of vestibular mucosal–submucosal flap tissue. Sutures placed at the alveolar margin adjacent to the intact teeth should not enter the sulcus. Sulcular distortion may lead to focal periodontitis (Fig. 50.10). Figure 50.11 demonstrates a unilateral rostral/central maxillectomy closure using vestibular mucosal–submucosal and alveolar mucosal-periosteal advancement flaps.



• **FIG. 50.9** Cross-section of (**A**) a one-layer and (**B**) two-layer closure with suture lines supported and unsupported by bone.



• **FIG. 50.10** Rostral view of suturing flap tissue to attached and free gingival strip remaining at the teeth adjacent to the excision site. The sulcus is intact. (**A**) Sutures placed into the sulcus will result in eversion of the free gingival margin and compromise of periodontal anatomy. (**B**) A wide segment of attached gingiva allows routine suturing. (**C**) A narrow segment of attached gingiva can be opposed to the flap margin by angled sutures without entering the sulcus.



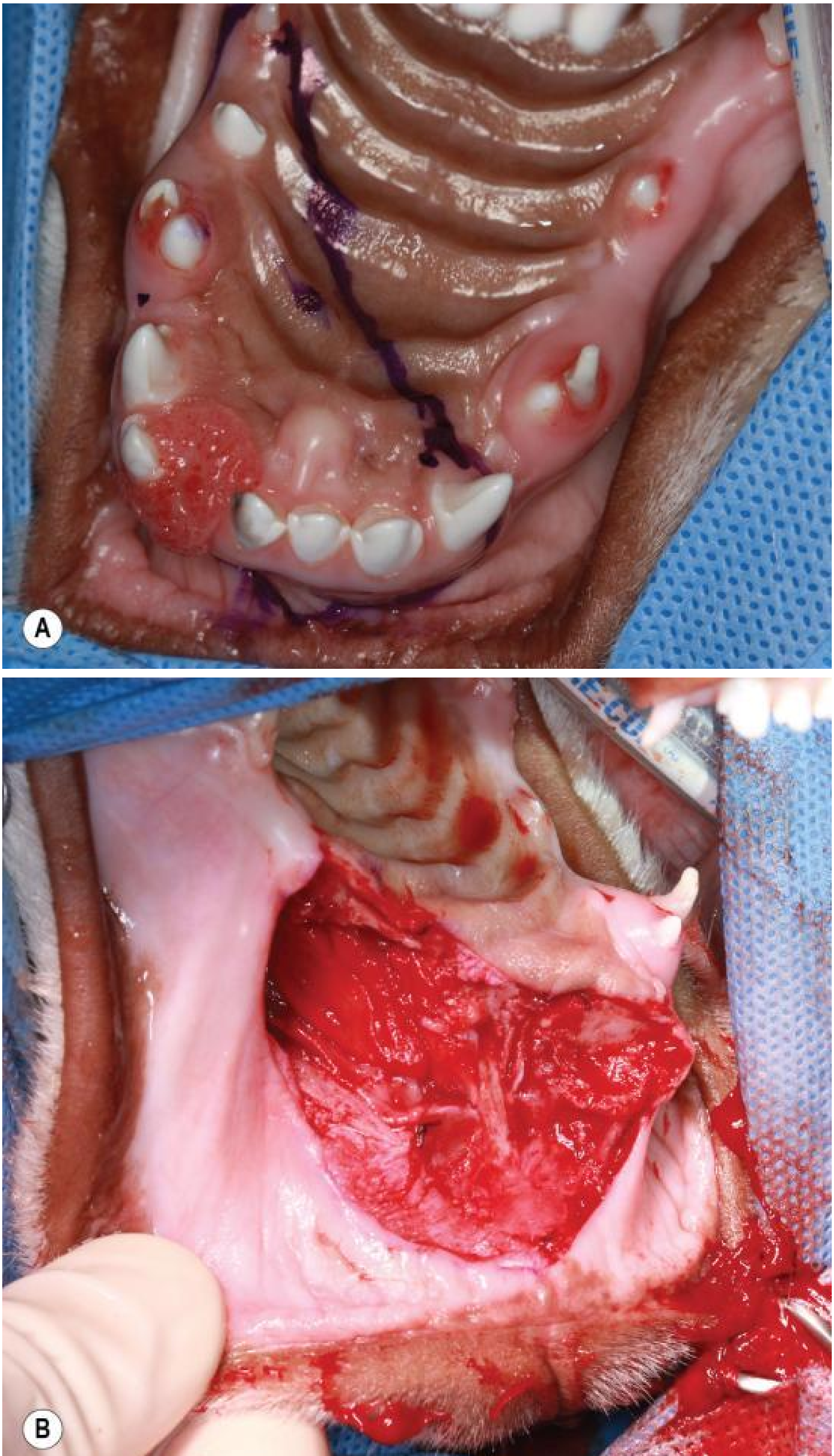
• **FIG. 50.11** (**A**) Soft tissue reconstruction using vestibular mucosal–submucosal and alveolar mucosa–periosteal advancement flaps following an extensive unilateral rostral maxillectomy. (**B**) The flap was sutured in a single layer using a 4-0 poliglecaprone 25 in a simple interrupted fashion.

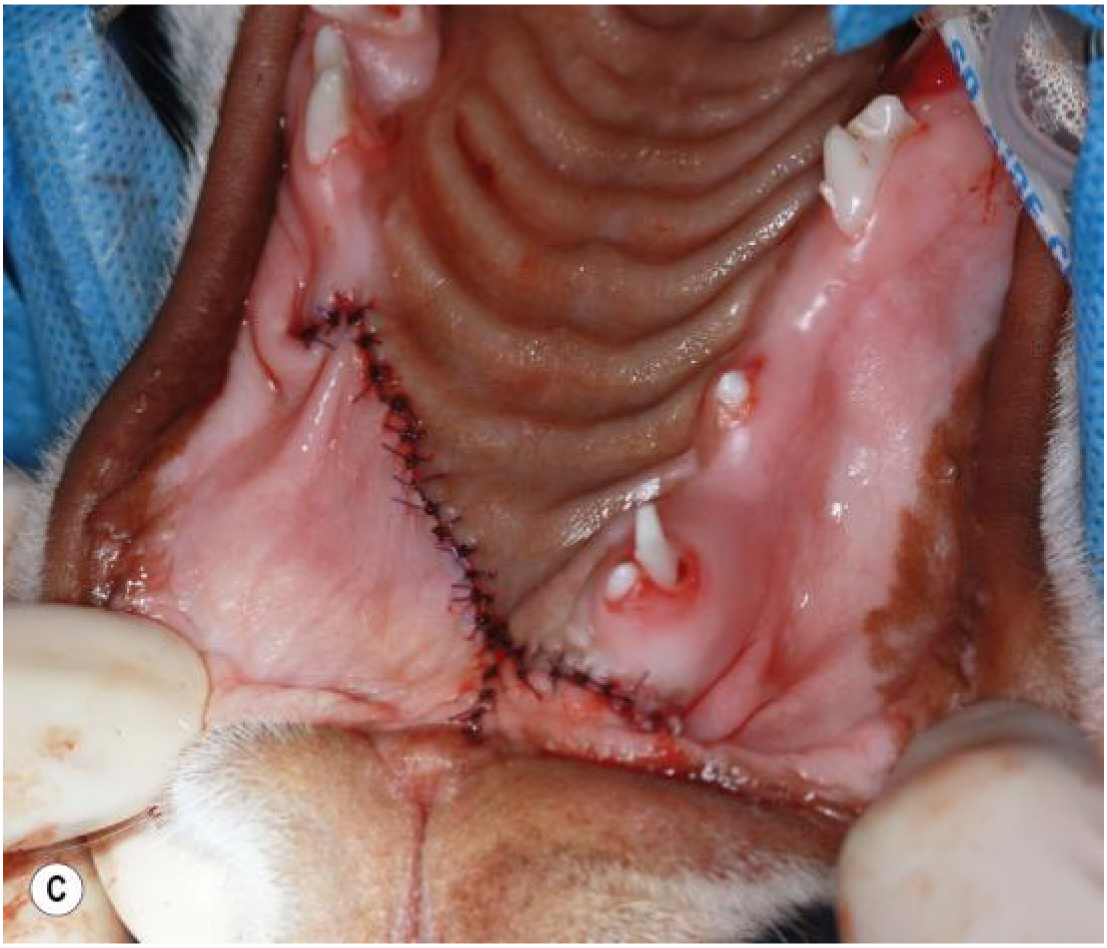
Suture materials are discussed in detail in Chapter 8; preferably, poliglecaprone 25 should be used for maxillectomy closure in a simple interrupted fashion.

Incisivectomy and rostral maxillectomy

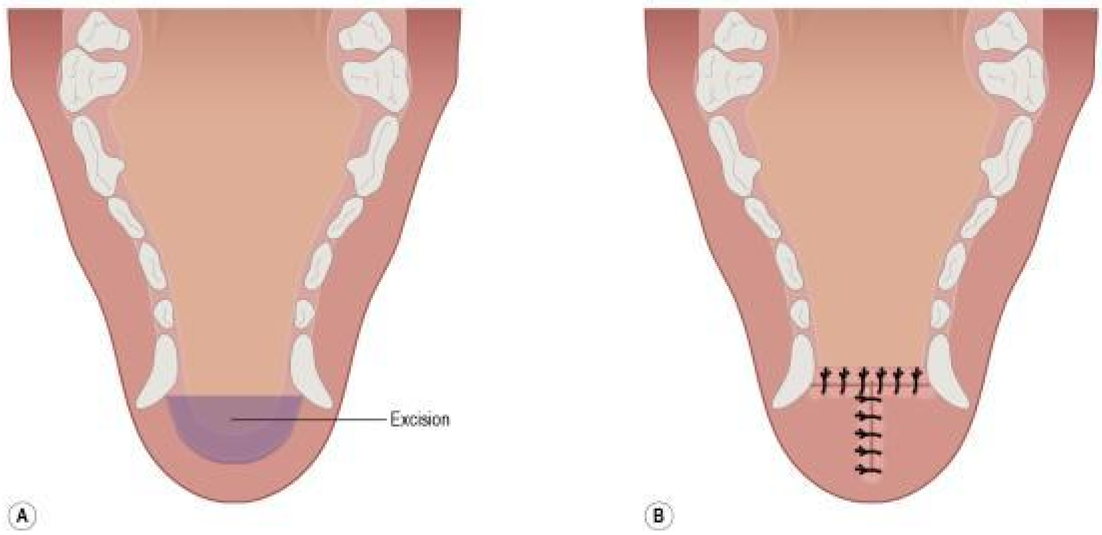
These techniques may be unilateral or bilateral. They may involve the incisor teeth region only (incisivectomy) or involve canine and rostral premolar teeth (rostral maxillectomy).

Incisivectomy often does not enter the nasal cavity but exposes the ventral lateral nasal cartilages. More extensive unilateral or bilateral techniques do create an oronasal communication (Fig 50.12). Vestibular mucosal–submucosal closure often results in a T-shaped suture line when the alveolar process of the incisive bone is excised (Fig. 50.13).


A
B



• FIG. 50.12 (**A**) Papillary squamous cell carcinoma at the incisive region in a puppy. (**B**) The mass extended into the bone and adjacent teeth requiring an extensive incisivectomy and rostral maxillectomy. (**C**) The flap was sutured in a T-shaped single-layer suture line using a 4-0 poliglecaprone 25 in a simple interrupted fashion.

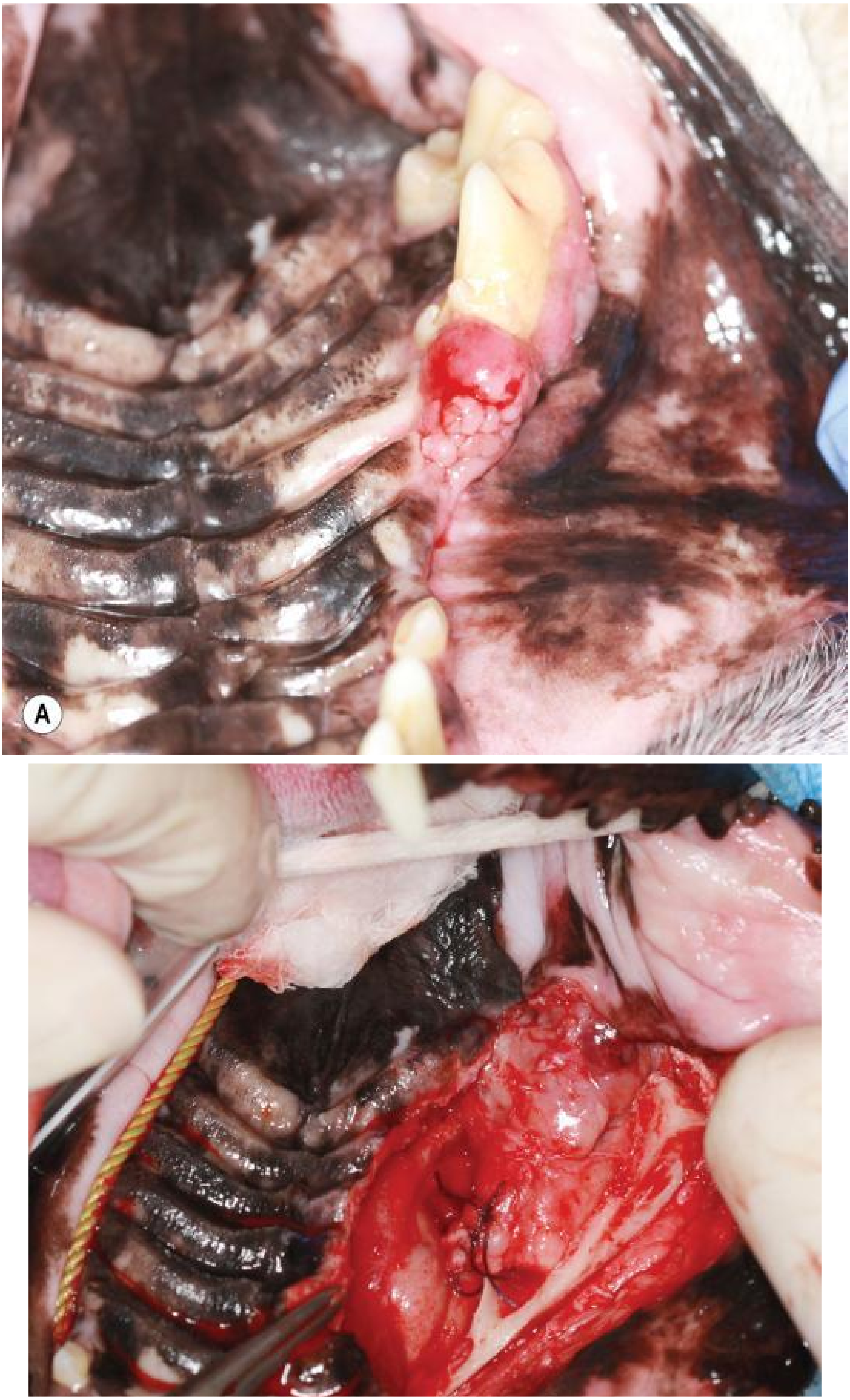


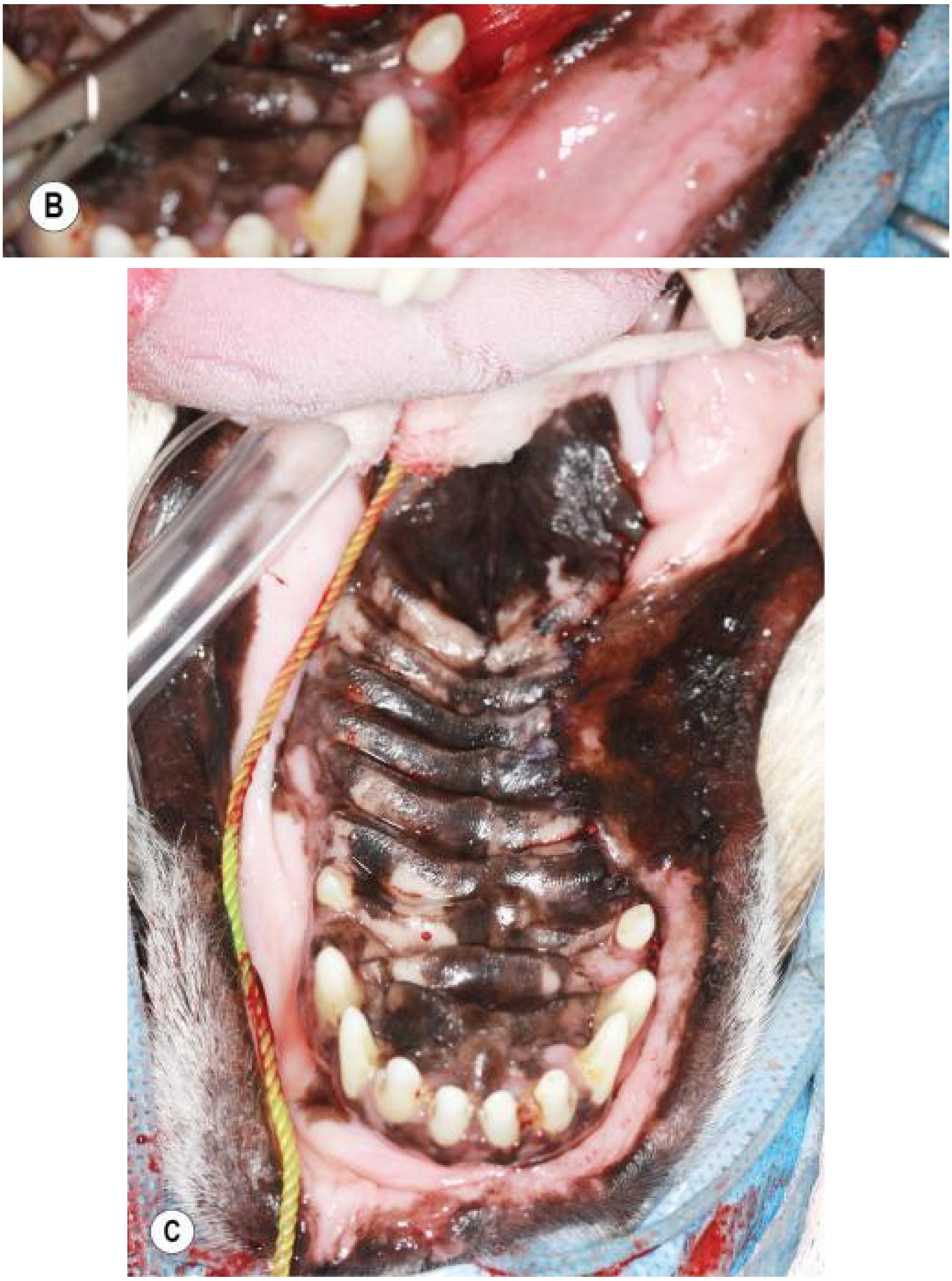
• **FIG. 50.13** (**A**) Excision of the incisive bones. (**B**) T-shaped closure.

Nose droop may occur with incisive bone excision due to removal of the ventral bone support for the nasal cartilages. Lateral deviation may occur secondary to lateral nasal ligament attachment disruption from the dorsal lateral aspect of the incisive bone. Pronounced nasal droop or lateral deviation can be corrected either from the incisivectomy site or from a dorsal approach; the latter approach may provide a better exposure for the correction.[26] A short dorsal midline incision is made over the junction of the nasal cartilages with the nasal and incisive bones and the incision is shifted to the appropriate side. The rostral end of the dorsal lateral aspect of the incisive bone is exposed and a small hole is drilled through the bone. Polypropylene suture is threaded through the bone hole and the suture needle is passed through the dorsal lateral nasal cartilage. The suture is tied at the appropriate tension to correct the deviation. Bilateral sutures are placed to elevate the nose. Long-term success for correction of nose drop following rostral maxillectomy has yet to be reported.

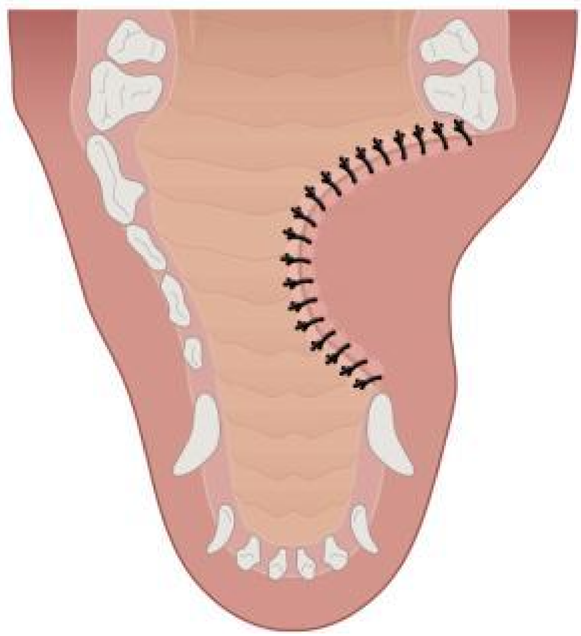
Central and caudal maxillectomy

Central and caudal maxillectomies create a temporary communication between the oral cavity and nasal cavity. Transection of the major palatine artery, vein, and nerve is often required. Careful transection of the palatal mucoperiosteum allows ligation and transection of the vessels at the rostral and caudal extent of the excision (Fig. 50.14). It may be necessary to extend the excision beyond the midline, depending on the size of the tumor; if so, the ability to close the defect will depend on the width of the maxilla and the amount of remaining labial mucosa. Excision of larger masses with appropriate surgical margins will leave less labial tissue for closure of the defect. There is often enough labial mucosa to extend to the midline and sometimes beyond the midline; however, this must be evaluated for each patient (Fig. 50.15).


A

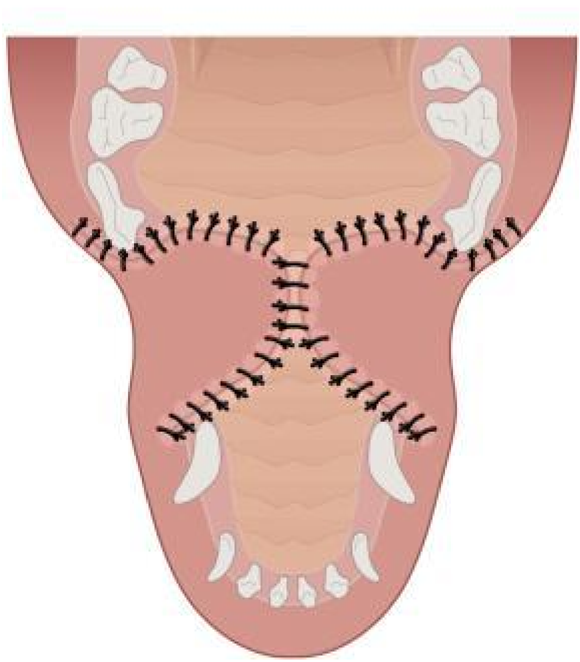


• **FIG. 50.14** (**A**) Malignant melanoma at the right caudal maxilla in a dog. (**B**) The tumor was excised, creating a temporary communication between the oral cavity and nasal cavity. Note the ligature of the infraorbital artery, vein, and nerve as they were transected. (**C**) The flap was sutured in a single layer using a 4-0 poliglecaprone 25 in a simple interrupted fashion.



• **FIG. 50.15** Closure of a large central maxillectomy defect using a vestibular mucosal–submucosal flap. A concave deformity of the face will result.

Another closure option for large maxillary defects is bilateral buccal mucosal-submucosal flaps. A buccal mucosal-submucosal labial flap is harvested from adjacent to the maxillectomy site as described. On the opposite side of the maxilla, the rostral-caudal length of the second flap is determined. A sulcular incision is made on the buccal and palatal side of the teeth at the level of this flap. Buccal-diverging, vertical-releasing incisions are made through the attached gingiva into the alveolar and buccal mucosa. Care is taken to not transect neurovascular structures emerging from the infraorbital foramen. The vestibular flap and palatal mucoperiosteum margins are subperiosteally elevated, the teeth are extracted, and the alveolar margin height is slightly reduced with an osteoplasty bur. Any remaining palatal mucoperiosteum lying between the maxillectomy surgical defect and the second flap is excised or denuded of epithelium with a bur. The free gingiva, including the sulcular epithelium, is excised from the second flap; this flap now consists of attached gingiva, alveolar mucosa, and buccal mucosa-submucosa and periosteum. It is lengthened as needed to meet the original buccal mucosal-submucosal flap from the maxillectomy site for a tension-free closure. Lengthening is accomplished by extension of the vertical-releasing incisions and/or release of the periosteum at the flap base. The defect is closed as described (Fig. 50.16).



• **FIG. 50.16** Closure of a large maxillectomy defect using bilateral vestibular mucosal–submucosal flaps. Creating the flap on the opposite side requires extraction of teeth, reduction in the alveolar bone height, and diverging vertical releasing incisions. A bilateral concave deformity of the face may result.

Orbitectomy[2]

Caudal maxillectomy techniques may require removal of portions of bones that comprise the orbit. Removal of the ventral orbital margin (inferior partial orbitectomy) is most commonly performed for excision of caudal maxillary neoplasia. Surgery involves excision of the rostral zygomatic arch and caudal maxilla and, possibly, lacrimal and palatine bones. Excision of larger more dorsally located tumors require intraoral and dorsal cutaneous incisions to allow access to the tumor circumference as described previously.[36] Partial inferior orbitectomy will not result in displacement of the eyeball as all supporting periorbital soft tissues remain intact. The defect is closed by suturing a vestibular mucosa-submucosa flap to palatal mucoperiosteum and, possibly, to soft palate. More extensive tumor involvement may require excision of the coronoid process of the mandible with associated muscles of mastication. Enucleation is considered if the eye, its neurovascular supply, or a large portion of an eyelid is infiltrated with neoplasia or lies within the desired surgical margins.

When performing a caudal maxillary excision including partial inferior orbitectomy, one may be unable to completely cut all the bone surrounding the tumor. Bone at the level of the rostromedial aspect of the pterygopalatine fossa and the caudomedial wall of the infraorbital canal (portions of palatine and maxilla bones) are difficult to access for ostectomy. In this situation, all bone cuts are made, and this remaining inaccessible area is carefully separated by lateral displacement of the tumor/bone segment. This now isolated tumor/segment is gently manipulated rostrally, the maxillary artery is ligated and divided, and the maxillary nerve is divided as they enter the infraorbital canal. Any visible branches of superior alveolar nerves are divided as they enter accessory canals in the caudal maxilla.

Additional technical considerations

Some partial maxillectomy techniques may require the ostectomy to cross the infraorbital canal or removal of the entire infraorbital canal. The level of the canal may be identified by one of several landmarks, including (1) palpating the lateral bone rim of the infraorbital foramen, which is found dorsal to the distal root of the maxillary third premolar tooth; (2) palpating the alveolar jugum of the maxillary fourth premolar tooth, the dorsal extent of which lies ventrolateral or lateral to the canal; and (3) visually tracing the infraorbital neurovascular structures caudally to the level of the foramen. The canal is opened by ostectomy of the buccal bone plate. The artery and vein are ligated and transected and the nerve is transected. The excision ostectomy may proceed without risk of inadvertently severing these vessels, resulting in profuse hemorrhage. Removal of the entire infraorbital canal, if needed as part of an inferior partial orbitectomy, is accomplished as described earlier. Based on clinical experience, sacrifice of the infraorbital artery does not jeopardize vestibular mucosal–submucosal flap vitality. Assumedly, the blood supply from the superior labial branch of the facial artery and the lateral nasal artery from the contralateral infraorbital artery is sufficient to maintain the flap.

For caudal maxillectomy, the parotid and zygomatic salivary papillae are identified and preserved if possible. If the papillae are included within the excision margins, the salivary ducts are identified and ligated.

The nasolacrimal duct is transected in various maxillectomy techniques. Epiphora generally does not result. Although not specifically studied, the proximal transected end of the duct apparently heals and forms a new ostium for continued tear drainage into the nasal cavity. Healing may occur similarly to various salvage procedures for nasolacrimal duct disruption.[38,39] Stent placement is not required. Surgical trauma to the lacrimal bone and disruption of the lacrimal sac may result in epiphora.

Postoperative care and assessment

Extubation

Immediately after completion of the surgery, all debris, including tissue fragments and blood clots, are removed from the oral cavity. Suction is applied to the pharyngeal area around the gauze packing. The gauze packing is removed and the oropharyngeal area is closely inspected to ensure removal of all debris and fluid. The patient is extubated when it regains consciousness and it is determined that it is able to protect its airway and the airway is patent.

Pain management

Depending on the magnitude of the surgery, most pain is evident in the first 24 to 72 hours postoperatively (see Chapter 4). Signs suggestive of continued pain include tachycardia, hypertension, salivation, pale mucous membranes, and dilated pupils. Vocalization may occur related to pain, anxiety, or fear. The spectrum of behavior can range from lethargy to restlessness, agitation, or delirium. Additional analgesic intervention is needed in the presence of these signs. Concurrent tranquilization will calm the delirious patient. Opioid administration is continued in the first 1 to 3 days postoperatively by intermittent injection or, more effectively, by continuous-rate infusion or a sustained release patch. Residual discomfort (less intense pain related to resolving inflammation) will last longer and is managed with nonsteroidal antiinflammatory drugs. Clinical behavior, including increasing activity level, eating, drinking, and grooming, suggests adequate pain management and patient comfort.

Nutritional support

Multielectrolyte intravenous fluids are continued until normal eating and drinking occur as analgesic medications are being reduced and patients become more alert. Oral water is offered at 24 hours postoperatively. Soft food is then offered. Most patients with maxillectomies will drink and eat at this time provided that higher levels of opioid administration, which may sedate the patient, are no longer needed. Patients that have more extensive surgery may require a longer course of opioids and may not voluntarily eat or drink for 2 to 3 days. Placement of an enteral feeding tube is rarely needed but is recommended as a method of reducing suture line stresses when closure of large defects using bilateral vestibular mucosal–submucosal flaps is performed. Placement of a feeding tube is also considered in the debilitated patient (see Chapter 5).

Postoperative patient appearance[23,26]

Swelling of the surgery site is usually resolved by 3 to 7 days postoperatively. Cosmetic appearance is generally good and depends on the extent of excision. Smaller excisions or larger excisions leaving abundant vestibular mucosa-submucosa for flap harvesting result in unnoticeable or minimal cosmetic abnormalities (Fig. 50.17). Bilateral incisivectomy results in some drooping of the nose and possible caudal retraction of the rostral lip margin. Unilateral incisivectomy may result in slight facial concavity and lip margin elevation. Concurrent lip wedge excision may result in additional lip margin deformity. Unilateral rostral maxillectomy including the canine tooth creates a more pronounced concavity with lip elevation and possible ipsilateral mandibular canine tooth protrusion lateral to the upper lip (Fig. 50.18). Extensive bilateral rostral maxillectomies create relative mandibular prognathism and nose droop (Fig. 50.19). More extensive central and caudal maxillectomies may result in facial concavity with elevation of the lip margin (Fig. 50.20).

• **FIG. 50.17** Frontal view of a dog that underwent a right central maxillectomy. Abundant tissue for creation of a large vestibular mucosal–submucosal flap was available, which resulted in excellent cosmetic results.

• **FIG. 50.18** Mandibular canine tooth protrusion and concavity of the face after unilateral rostral-central maxillectomy.

• **FIG. 50.19** Extensive bilateral rostral maxillectomy created relative mandibular prognathism. The nose droop was corrected by suturing the nose to the rostral aspect of the maxillas.

• **FIG. 50.20** Concavity of the face after extensive unilateral rostral-central maxillectomy.

Homecare and follow-up

Most patients are sent home after 1 to 3 days postoperatively. A soft food diet and removal of chew toys are recommended for 2 to 3 weeks. The client is shown how to administer any oral medications so as not to disrupt the suture line. Medications may be concealed in soft food. If oral manipulation is needed, the manipulation is performed away from the surgery site. The surgery site is examined in 2 weeks and any skin sutures are removed. The surgery site is reexamined at 1 month postoperatively and any residual oral sutures may be removed at this time. Most oral sutures are absorbed or extruded in approximately 3 weeks. A complete physical examination, including the surgical site, is performed at 3-month intervals for 1 year. In the case of malignant neoplasms, three-view thoracic radiographs are obtained at these examinations.

Complications

Intraoperative complications

Hemorrhage is the major intraoperative complication. Extensive blood loss may occur from premature or unintentional transection of the major palatine and infraorbital arteries. A recent study demonstrated that excessive bleeding during surgery was encountered in 53.4% of dogs that underwent maxillectomy, and 42.7% of these cases received intraoperative blood transfusion. These complications were significantly associated with the size of the tumor, the maxillectomy type, and the surgical approach, with dogs treated using a combined extraoral and intraoral approach exhibiting a higher rate of bleeding complications (83% of cases).[40]

Temporary carotid artery ligation before starting the maxillectomy has been described to aid in the control of hemorrhage but is rarely needed (see Chapter 48).[41] Hemorrhage is controlled as it is encountered by digital pressure, ligation, or focal careful use of electrocoagulation. If necessary, a hemostatic agent may be used (see Chapter 8). In the event of brisk hemorrhage, it is essential that the surgeon keep track of the approximate amount of blood loss in gauze sponges (Fig. 48.5) and the suction unit; if such occurs, the physiologic responses of the patient, such as tachycardia and reduced blood pressure, indicate the need for volume replacement.

Postoperative complications

Bilateral surgery may result in temporary occlusion of one or both nasal passages. The patient may experience anxiety in the early postoperative period due, in part, to inability to breathe through the nose. Tranquilization, combined with adequate analgesia, will eliminate the anxiety and allow open-mouth breathing. Sneezing and a small amount of serosanguineous nasal discharge or epistaxis may be present for several days. More radical excision and removal of all turbinates from one side of the nasal cavity may result in permanent serous nasal discharge.[26] A recent study demonstrated that epistaxis occurred in 51.3% of the cases and typically within 48 hours after surgery. Other possible complications that may occur in the first 48 hours after surgery were excessive facial swelling (36.8%), pawing at the face (10.9%), and difficulty eating (11.4%).[40]

Superficial ulceration of the vestibular mucosal–submucosal flap may occur secondary to contact with mandibular teeth. This is generally not a chronic problem, as keratinization of these contact areas increases over time and ulceration ceases in a few weeks.

The ipsilateral mandibular canine tooth may protrude intermittently or continuously to the lateral surface of the upper lip. This is usually a cosmetic problem but occasionally causes ulceration of the labial mucosa and/or skin, and a recent study describes lip trauma to occur in 13.4% of the cases.[40] If indicated, the tooth protrusion is corrected by crown height reduction and partial coronal pulpectomy, direct pulp capping, and restoration.[42,43]

Dehiscence typically occurs in the first 4–5 days postoperatively but may occur between 48 hours and 4 weeks following maxillectomy, and is often a result of technical error, with extrinsic tension on the wound edges being the most important cause.[25] The published occurrence is 11% and may be minimized by following the principles of oral surgery.[40] Inadequate suture placement (small bite depth and wide spacing), suture line tension, the use of electrosurgery, and marginal necrosis of the flap contribute to dehiscence. Repair of a dehiscence that results in an oronasal fistula is usually deferred for 2 to 4 weeks. This delay allows for resolution of flap inflammation and reestablishment of the blood supply of the flap. The fistula is treated by debridement of soft tissue and bone margins and mobilization of adequate mucosal tissues for a tension-free closure. Excised tissues are sent for histopathological evaluation to rule out local tumor persistence as the cause for wound healing failure.

Dehiscence of an incisivectomy that exposes the ventral lateral nasal cartilages usually does not need repair and heals by second intention.

Periodontal abscessation of a tooth adjacent to the surgery site may develop as a result of chronic root exposure to the nasal cavity, with resulting periodontal and possible endodontic disease. This is caused by removal of alveolar bone during the partial maxillectomy. Diagnostic findings may include intraoral swelling at or adjacent to the surgery site, a parulis (an elevated mucosal nodule at the site of an intraoral sinus tract draining a chronic abscess), a periapical radiolucency in the involved area, and oral sensitivity that may improve when treated with antibiotics. Tooth extraction, debridement, and antibiotics based on culture and sensitivity results resolve this complication. Tissues are submitted for histopathologic examination to rule out local neoplasm recurrence as a contributing factor. The overall infection rate following maxillectomy was shown to be 7.9%.[40]

Local tumor recurrence is common with certain tumor types. Suspicious areas noted during recheck examination must be biopsied to differentiate neoplasm recurrence from suture reaction and scar tissue.

Prognosis

The overall prognosis for maxillectomy techniques regarding oral function, operative and postoperative complications, postoperative appearance, and client satisfaction is good to excellent.[15,18–26] This seems directly related to the surgeon's experience with appropriate patient selection, technical performance of the surgery, and preoperative owner communications to set realistic expectations. Less positive results may occur in patients with more aggressive disease and more radical surgery. One study reported an overall 85% client satisfaction with maxillectomy and mandibulectomy procedures. The satisfaction level correlated with the postoperative survival time of the patient.[15]

Local recurrence rates, survival times, and biologic behavior of benign and malignant oral tumors reported in various clinical studies have been reviewed (see also Chapter 44).[25–29] Early recognition, accurate diagnosis, and aggressive, planned excision are essential for optimal results.

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CHAPTER 51

Mandibulectomy techniques

Frank J.M. Verstraete, Boaz Arzi, Gary C. Lantz

Definitions

Mandibulectomy techniques involve removal of various segments of a mandible, one entire mandible, or one mandible with a rostral portion of the other mandible, with adjacent neoplastic and soft tissues. Closure of the surgical defects is accomplished using buccal and alveolar mucosal-submucosal flaps. A *rim excision* or *marginal mandibulectomy* is defined as a partial segmental excision, leaving the ventral border of the mandible intact.[1] The term *hemimandibulectomy* is often used in the veterinary literature to denote the complete excision of one of the two mandibles. However, the term *total mandibulectomy* is more appropriate. The term *hemimandibulectomy* should only be used to denote the excision of half of one mandible, but the term *partial mandibulectomy*, with a reference to which part (rostral, central, or caudal), is more appropriately used for this. Similarly, when referring to the excision of one entire mandible and half of the other mandible, the term *one-and-one-half mandibulectomy* is preferred to *three-quarter mandibulectomy*.

Indications

Mandibulectomy techniques are most commonly indicated for excision of malignant and benign oral neoplasms. They are also used as salvage procedures for mandibular fractures that are severely comminuted or complicated by missing bone fragments or severe periodontitis (see Chapter 35) and mandibular osteonecrosis (see Chapter 58).[2,3]

Background

Mandibulectomy techniques for the treatment of oral neoplasms were introduced by Withrow and Holmberg.[4] Clinical investigation for treatment of neoplastic[5-16] and nonneoplastic oral conditions[2,3] and review publications[16-25] have followed, documenting clinical results. Later, mandibular reconstruction, using regenerative approach, following segmental or bilateral rostral mandibulectomy was introduced (see Chapter 53).[26,27]

Preoperative concerns

Diagnosis

The histopathologic diagnosis of an oral mass must be aggressively pursued and the patient staged using the TNM (tumor, node, metastasis) classification.[22,23] Further details are found in Chapters 42 and 50. Especially relevant to mandibulectomy techniques is to assess how far apically tumors originating on the gingiva have infiltrated the bone. This will largely determine whether a surgically aggressive or more conservative approach is indicated. It is also very important to determine whether and to what extent the root of the tongue is involved; computed tomography (CT) with soft tissue contrast enhancement or nuclear magnetic resonance imaging are especially useful in this respect. CT is also helpful in determining the extent of bone and mandibular canal involvement.

Client communication

Topics to discuss with the client include (1) intent of surgery (local cure or palliation) and prognosis, (2) operative concerns (blood loss and anesthesia risk), (3) postoperative appearance and oral function, and (4) recommended follow-up evaluation and treatment. A collection of preoperative and postoperative photographs of patients that have had various types of partial mandibulectomy is very useful for client communication.

Preoperative management

The reader is referred to Chapter 50 and other chapters for anesthetic considerations, preemptive analgesia, antibiotics, and oral cavity disinfection.

Patient positioning

Dorsal and lateral recumbency

The head is secured into position with a vacuum-charged surgical positioning system (Vac-Pac®, Olympic Medical, Seattle, WA). This device allows the head to be held stable in any position. A mouth gag is positioned away from the surgery site to keep the mouth open. The endotracheal tube is secured to the muzzle and the tongue retracted from the surgery site. For bilateral rostral mandibulectomy, the patient is placed in dorsal recumbency, with the neck flexed to allow access to both oral and cutaneous surfaces of the rostral mandibles. For unilateral rostral or central mandibulectomy, the patient is placed in lateral or dorsal recumbency with the head rotated to facilitate visualization of the surgical field. The head is held parallel with the table top or the rostral end of the muzzle and lower jaw slightly elevated to improve site access. The neck is extended when the patient is in lateral recumbency. For caudal and total mandibulectomy, the patient is placed in lateral recumbency with the neck extended and the rostral end of the muzzle and lower jaw elevated so the surgeon can look into the open mouth.

After positioning, a pharyngeal pack of absorbent exodontia sponges (Cotton-filled Exodontia Sponges, Crosstex International Inc., Hauppauge, NY) is placed in the pharynx around the endotracheal tube.

Sternal recumbency

Sternal recumbency, with the head elevated and the maxilla suspended, is an alternative method of positioning for mandibulectomy procedures. The maxilla is suspended by perforating the maxillary canine teeth through a long strip of adhesive tape. The ends of the tape are then extended and wrapped high around intravenous poles placed on either side of the patient's head. The mandible should be level with or slightly lower than the surface of the surgery table.

Sternal recumbency offers excellent visualization of the mandible and oral cavity. In this position, all mandibulectomy procedures can be performed using an intraoral approach.

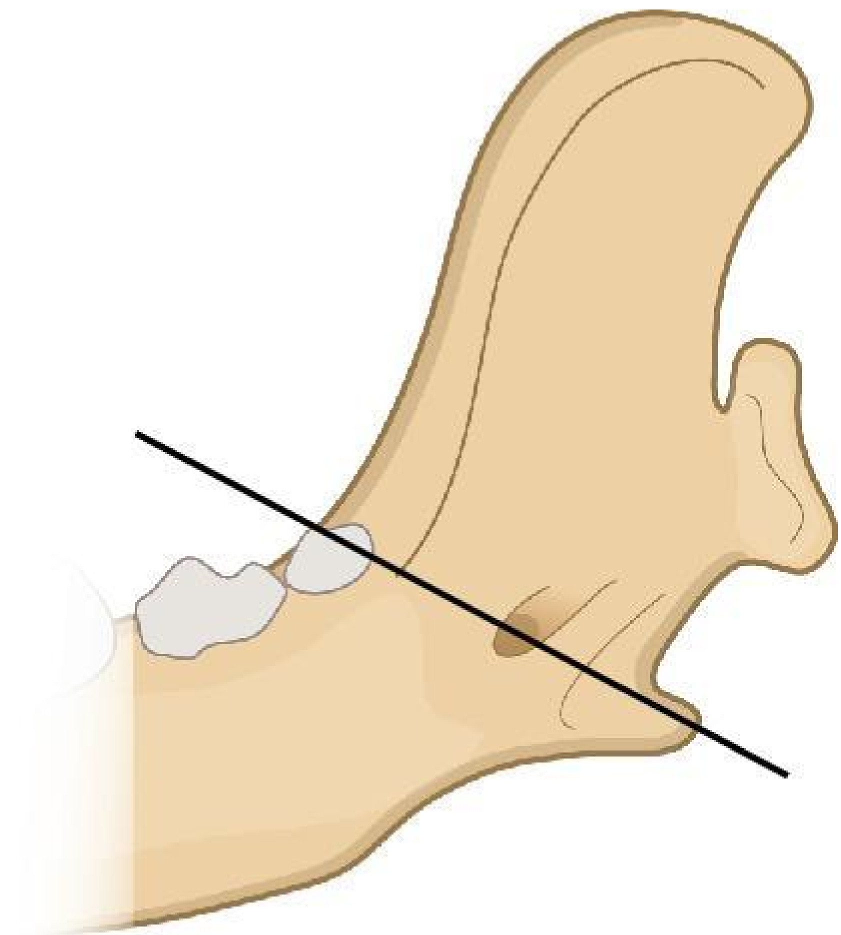
The main hazard of sternal recumbency is fluid aspiration. The use of a cuffed endotracheal tube and pharyngeal pack is necessary to prevent aspiration. Having continuous indwelling suction available is very helpful.

Surgical anatomy[28-30]

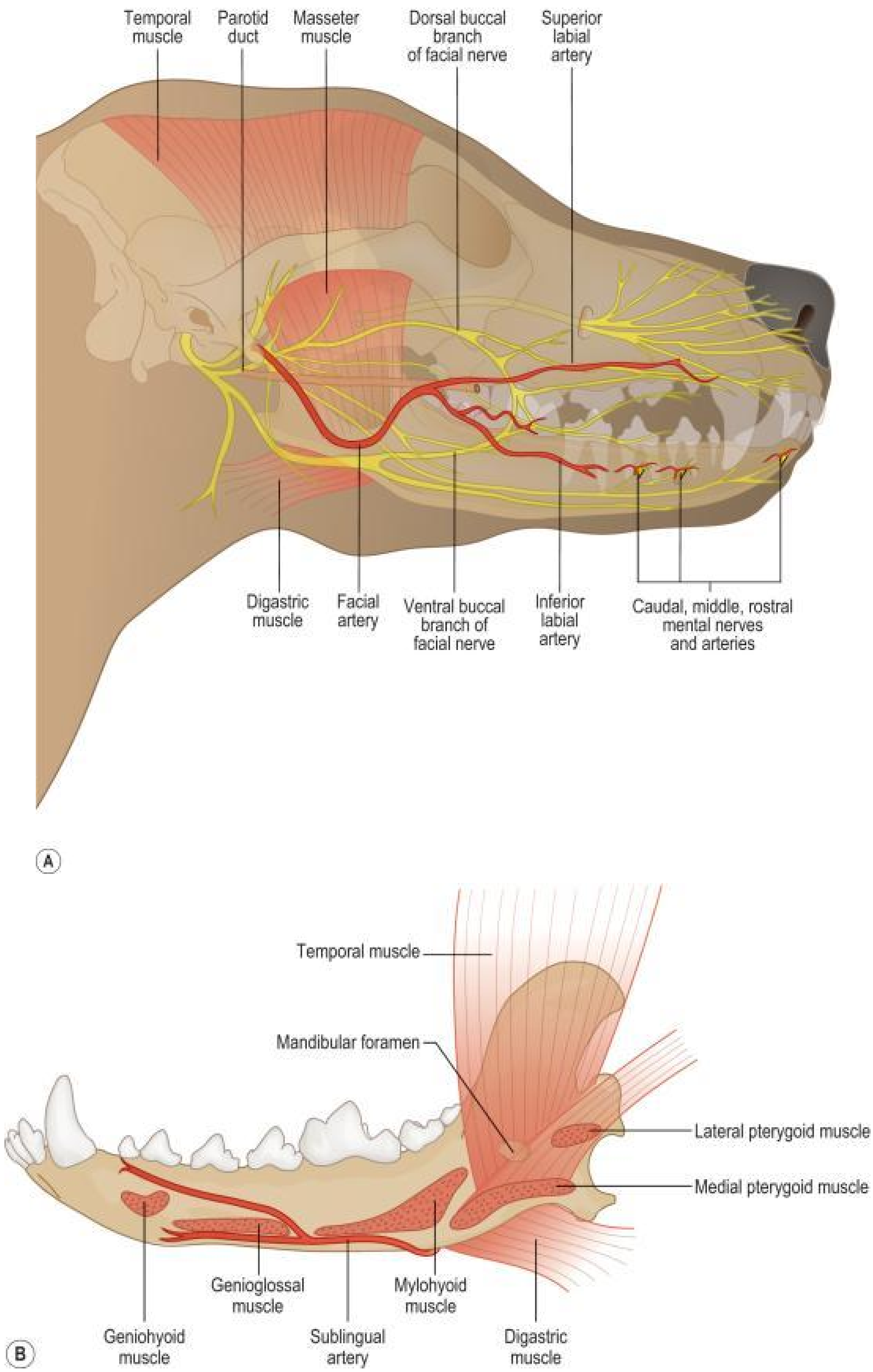
The inferior alveolar artery originates from the maxillary artery. It enters the mandibular foramen and traverses the mandibular canal with numerous arterioles branching to the bone and teeth. There are no direct vascular anastomoses across the fibrocartilaginous mandibular symphysis; however, small extraosseous arterioles span the symphysis in the submucosal tissue. Blood supply to the cortical bone at the symphysis is from endosteal and periosteal surfaces. This extraosseous supply may be important to the viability of a rostral mandibular segment after interruption of the inferior alveolar artery secondary to a caudal or central mandibulectomy. The inferior alveolar artery terminates in the caudal, middle, and rostral mental arteries.

The mandibular canal mainly contains the inferior alveolar artery, vein and nerve, and some loose connective tissue and fat. The nerve is dorsolateral, the artery is in the central area, and vein ventromedial. Bone convexities from the apical alveolar bone of the mandibular teeth border the lingual or dorsal lingual aspect of the canal. The mandibular canal terminates at the mental foramina.

The mandibular foramen is located on the medial aspect of the mandible. It is immediately below and centered on a line from the tip of the angular process to the distal aspect of the third molar tooth in the dog (Fig. 51.1) and the first molar tooth in the cat. Palpation of these landmarks during caudal mandibulectomy aids in the dissection of the inferior alveolar artery and nerve as they enter the foramen. The middle mental foramen in the dog is at the level of the rostral root of the second premolar tooth, immediately distal to the apex of the canine tooth and approximately midway between the alveolar margin and ventral bone margin (Fig. 51.2A). In the cat, this foramen is located at the level of the apex of the canine tooth. The caudal mental foramen is at the level of the third premolar tooth and approximately midway between the alveolar margin and ventral margin in the dog and cat. The rostral mental foramen is generally at the level of the second incisor tooth and a short distance ventral to the alveolar margin. Multiple small foramina may be present.



• **FIG. 51.1** Lingual aspect of mandibular ramus. The mandibular foramen is located in the middle of and immediately ventral to an imaginary line between the palpable landmarks of the angular process and the distal aspect of the mandibular third molar tooth.



• **FIG. 51.2** (**A, B**) Surgical anatomy of arteries, nerves, and muscles pertaining to mandibulectomy techniques.

The apex of the large curved root of the mandibular canine tooth in the dog extends as far as the apex of the distal root of the second premolar tooth. Therefore bone excision to remove the canine tooth alveolus would also result in removal of the first and second premolar teeth. In the cat, the apex of the canine tooth is rostroventral to the mesial root of the third premolar tooth.

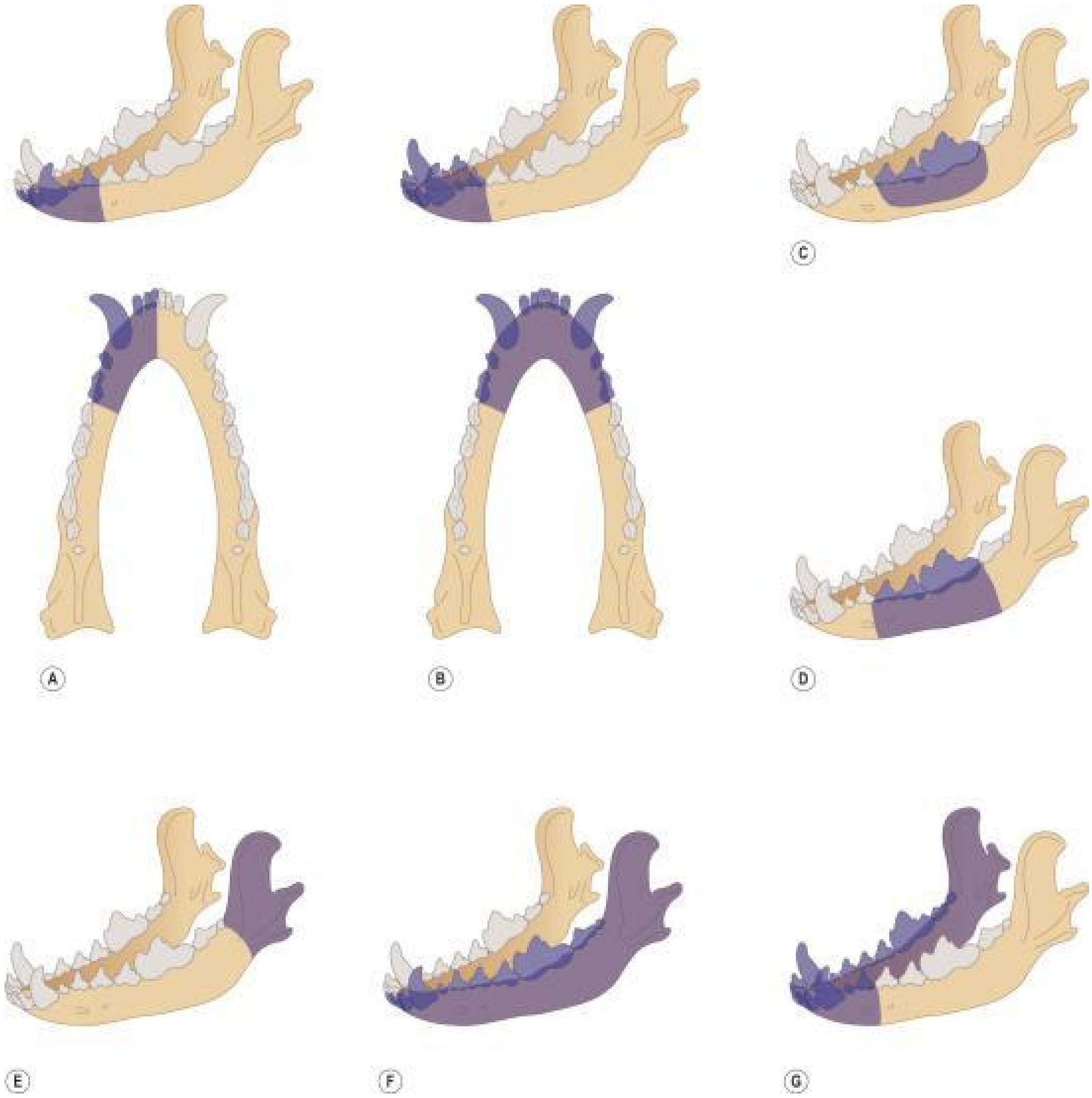
The inferior labial artery originates from the facial artery and supplies the soft tissue of the lower lip. The sublingual artery also originates from the facial artery and courses along the medial aspect of the mandible. The ventral buccal branch of the facial nerve provides motor innervation to the muscles of the lip. The mandibular branch of the trigeminal nerve provides motor innervation to the muscles of mastication and sensory innervation to the lower lips, rostral two-thirds of the tongue, mandibular teeth (as the inferior alveolar nerve), and buccal cavity. The muscles of mastication insert on the ramus of the mandible, and some lingual and hyoid muscles insert on the body of the mandible (Fig. 51.2B).

The mandibular and sublingual salivary ducts are visible through the thin oral mucosa and course lateral to the tongue frenulum to end in the sublingual caruncle located just rostrolateral to the rostral margin of the frenulum.

Therapeutic decision-making

Types of osseous excisions

The most common types of mandibulectomy are shown in Fig. 51.3. The extent of any excision is determined by the oral physical examination and diagnostic imaging findings, the histopathological diagnosis, the recommended surgical margins, the intent of the surgery, and the tissue available for closure of the defect.



• **FIG. 51.3** Examples of mandibulectomy techniques *(shaded areas)*. (**A**) Unilateral rostral mandibulectomy. (**B**) Bilateral rostral mandibulectomy. (**C**) Central or segmental mandibulectomy with preservation of the mandibular canal and ventral margin ("rim excision"). (**D**) Central or segmental mandibulectomy (full thickness). (**E**) Caudal mandibulectomy. (**F**) Total mandibulectomy. (**G**) One-and-a-half mandibulectomy.

Surgical technique

General surgical principles[21-23]

Surgical plan

The surgical plan for a mandibulectomy procedure includes the following important points: (1) plan for adequate surgical margins, (2) plan for complete mucosal closure, (3) perform atraumatic surgery and preserve local blood supply, (4) avoid or minimize the use of electrosurgery or laser, (5) harvest large mucosal flaps to avoid suture line tension, and (6) support suture lines with healthy bone if possible.

Surgical margins

Surgical margins are indicated by the biological behavior of the neoplasm as determined by the preoperative histopathological diagnosis, diagnostic imaging (i.e., dental radiographs and CT with and without contrast), and oral physical examination findings (see Chapter 48). The preoperative histopathological diagnosis is essential for definitive surgical planning. In general, a minimum of 10-mm margin should be obtained for most benign and malignant neoplasms except fibrosarcoma, where a minimum margin of 15–20 mm is recommended due to the higher local recurrence rate.[21-23] For benign tumors, such as peripheral odontogenic fibroma, a margin less than 10 mm may be acceptable in some cases, based on tumor behavior (e.g., growth rate and recurrence), diagnostic imaging findings, and anatomical location. However, studies evaluating the appropriate surgical margins in dogs and cats are lacking, and these recommendations have not been correlated with clinical outcome (see Chapters 48 and 50). Wider margins are obtained for malignant neoplasms if a tension-free oral mucosal reconstruction can be obtained. The surgical margins are measured and marked around the circumference of the mass using a sterile surgical marker before starting the surgery. It is important to remember that the periodontal ligament spaces and mandibular canal may facilitate neoplastic cell infiltration. Therefore if the canine tooth is included in the measured surgical margins, the ostectomy is performed between the second and third premolar teeth (in the dog) to ensure complete removal of the canine tooth alveolus. Invasion of the mandibular canal by a malignancy requires total mandibulectomy.

Postoperative radiographs of the excised specimen or surgery site are made to document adequate gross surgical margins and the removal of all tooth fragments in the excised tissue. The entire specimen is sent for histopathological examination to confirm the diagnosis and to assess the bone and soft tissue margins for neoplastic cells. The margins of the specimens should be color coded for determination of surgical margins using the Davidson Marking System® (Bradley Products Inc., Bloomington, MN).[31] Finding neoplastic cells on the excision margin implies incomplete removal of local microscopic disease and the need to consider extending the excision site, adjunct radiation, or chemotherapy.

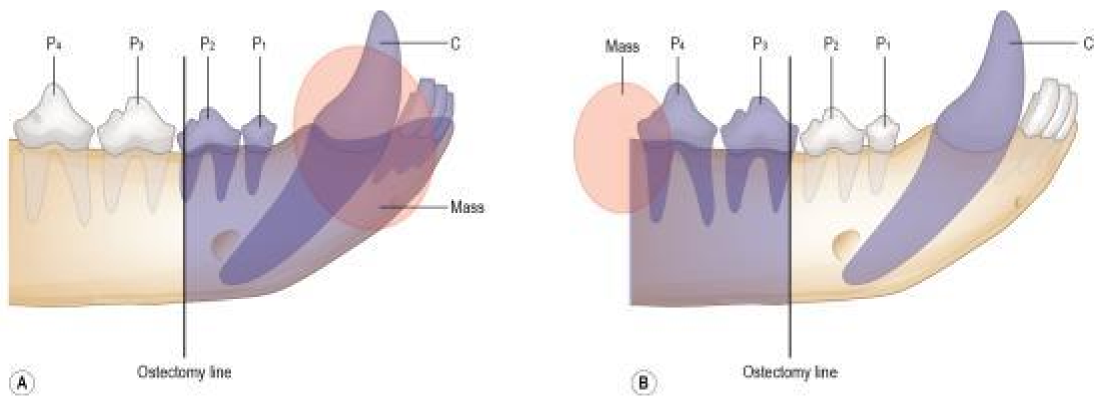
Surgical incision

The incision is made along the measured and marked surgical margins and the incision is full-thickness. Once incised, the alveolar mucosal margins may retract and result in some distortion of the proposed margins. The described incision method maintains accurate surgical margins. In the regions of foramina, the dissection is continued to identify, ligate, and transect the vessels and transect the nerves. Hemorrhage is controlled by ligation, pressure, and, if needed, minimal focal use of electrocoagulation. Depending on surgical margins, muscles attaching to the mandible are subperiosteally elevated or transected.

Concurrent lip wedge excision may be needed to obtain adequate surgical margins. This is true for large neoplasms along the body of the mandible and when a bilateral rostral mandibulectomy is performed. Wedge excision may also be performed during the latter procedure to remove excess skin. Lip excision compromises blood supply to the remaining rostral lip segment; however, viability is maintained by submucosal arterial branches.

Management of adjacent teeth

Maintaining teeth is secondary to achieving adequate surgical margins and tension-free closure of the surgical defect. Ostectomy performed at a narrow interdental space may result in injury requiring extraction of a tooth adjacent to the ostectomy site if inadvertent severe periodontal or endodontal injury (pulp cavity exposure) occurred during ostectomy. Ostectomy at the level of a wider interdental space may not result in similar tooth injury. Therefore to minimize tooth loss, the ostectomy margins are planned to transect a tooth at the margin level (Fig. 51.4). This may require margin extension, which is acceptable. Margins should never be compromised to save teeth. Once the en bloc tissue mass is excised, the remnant of the transected tooth is extracted. The adjacent teeth and periodontal structures are not injured, leaving a margin of attached and free gingiva adjacent to the ostectomy.



• FIG. 51.4 Ostectomy lines for (**A**) rostral mandibulectomy and (**B**) caudal mandibulectomy in relation to adjacent teeth. Transecting a tooth at the time of excision will not injure adjacent teeth; the remaining tooth fragment is removed after the en bloc excision is completed. The specific level of ostectomy is dependent upon measured surgical margins.

In the dog, the apex of each mandibular canine tooth is located slightly ventral to the mandibular second premolar tooth. Therefore rostral excisions to ensure complete removal of the alveolus of the canine tooth include the first and second premolar teeth.

Ostectomy and osteoplasty

The soft tissues are subperiosteally elevated away from the ostectomy site. The ostectomy is performed as determined by the premeasured surgical margins. The excision is planned so that all root structures of teeth within the surgical margins are part of the en bloc excision. Any remaining tooth root tips from transected teeth at the excision periphery are extracted after the en bloc tissue segment is removed.

Power equipment that cuts bone with burs, or less desirably with saw blades, offers the most efficient ostectomy method. The most control is gained when using a surgical handpiece with continuous irrigation and bone cutting burs. Alternatively, piezosurgery can be used (see Chapter 9). Carbide steel bone cutting burs may leave a roughened bone edge. Medium-grit diamond tapered cylinder burs produce a smooth bone cut. Once the en bloc tissue segment is removed, osteoplasty is performed. Cut bone margins are smoothed with a round, medium- or fine-grit diamond bur or carbide bur. The goal is to remove all sharp bone ridges and bone spikes that may result in ischemic pressure injury and potential perforation of the mucosal flaps.

Osteotomy of the ventral third of the mandible is performed last. Ideally, the inferior alveolar artery is isolated, ligated, and transected. In the event of inadvertent transection of the artery during osteotomy, the artery is retrieved and ligated. If the artery cannot be retrieved, an absorbable hemostatic agent is firmly packed into the mandibular canal.

A thin osteotome and mallet are rarely used these days for performing the ostectomy. The instruments are used with gentle technique to slowly advance the depth of bone cut. Aggressive use may result in fractures of remaining bone. The mandibular symphysis is split using an osteotome and mallet.

Flap design

General principles

Recommended oral mucosal flap length–to–base width ratio is 2:1 in humans[32] and is unknown in the dog and cat. In general, the flap is made slightly larger than necessary to ensure adequate tissue for tension-free closure and should, ideally, not have vertical releasing incisions, as flap blood supply may be compromised.[32] If vertical releasing incisions are needed, they are divergent from the flap apex to base to maintain a broad-based flap for blood supply conservation.[32]

The flaps are gently handled. Excessive pulling, crushing, or desiccation may injure flap blood supply.[32] Tissue forceps are not closed tightly, to avoid crushing the tissue and potential marginal ischemic necrosis. Thin tissue flaps may be atraumatically manipulated by placement of two stay sutures. Defect closure is most commonly achieved using local mucosal tissues. Advanced closure techniques are discussed in Chapter 52.

Vestibular mucosal–submucosal flap

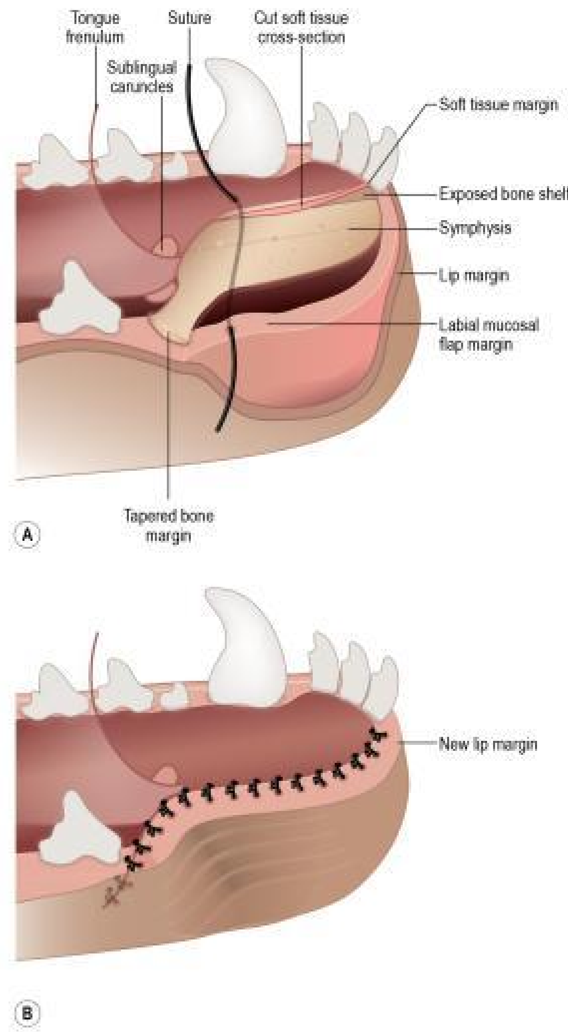
The flap is harvested by dissecting from the mandibulectomy site toward the lip margin. The plane of dissection is below the submucosa and may contain superficial skeletal muscle fibers to ensure a flap of substantial thickness. Branches of the inferior labial artery and vein and the ventral buccal branch of the facial nerve are visualized during flap elevation and remain in their original position. Flap elevation is continued until there is enough tissue for a tension-free closure. The flap blood supply is from the inferior labial artery. The amount of remaining buccal mucosa is determined by the size of the excision. A lesser amount of remaining buccal mucosa results in a larger lingual deviation of the lip and a mandibular concavity.

Surgical defect closure

Reconstruction of an oral mucosal lining is essential and is determined by preoperative planning. Healing without complication depends largely on accurate tension-free apposition of incised soft tissue margins. Inverted epithelium from poor approximation and suture line tension will delay wound healing. A two-layer closure, when possible, is optional.

Closure at the rostral mandible

Separation of the mandible for unilateral rostral or total mandibulectomy is commonly performed at the mandibular symphysis. The vestibular mucosal–submucosal flap is sutured to the mucoperiosteal fibrous tissue at the symphysis. The needle is passed from the intact mucoperiosteum toward the symphysis. A wide bite is taken and the needle is “walked off” the bone at the level of the symphysis. Excellent suture retention is provided, and accurate placement will not tear the tissue. The suture is then passed through the vestibular flap (Fig. 51.5A).

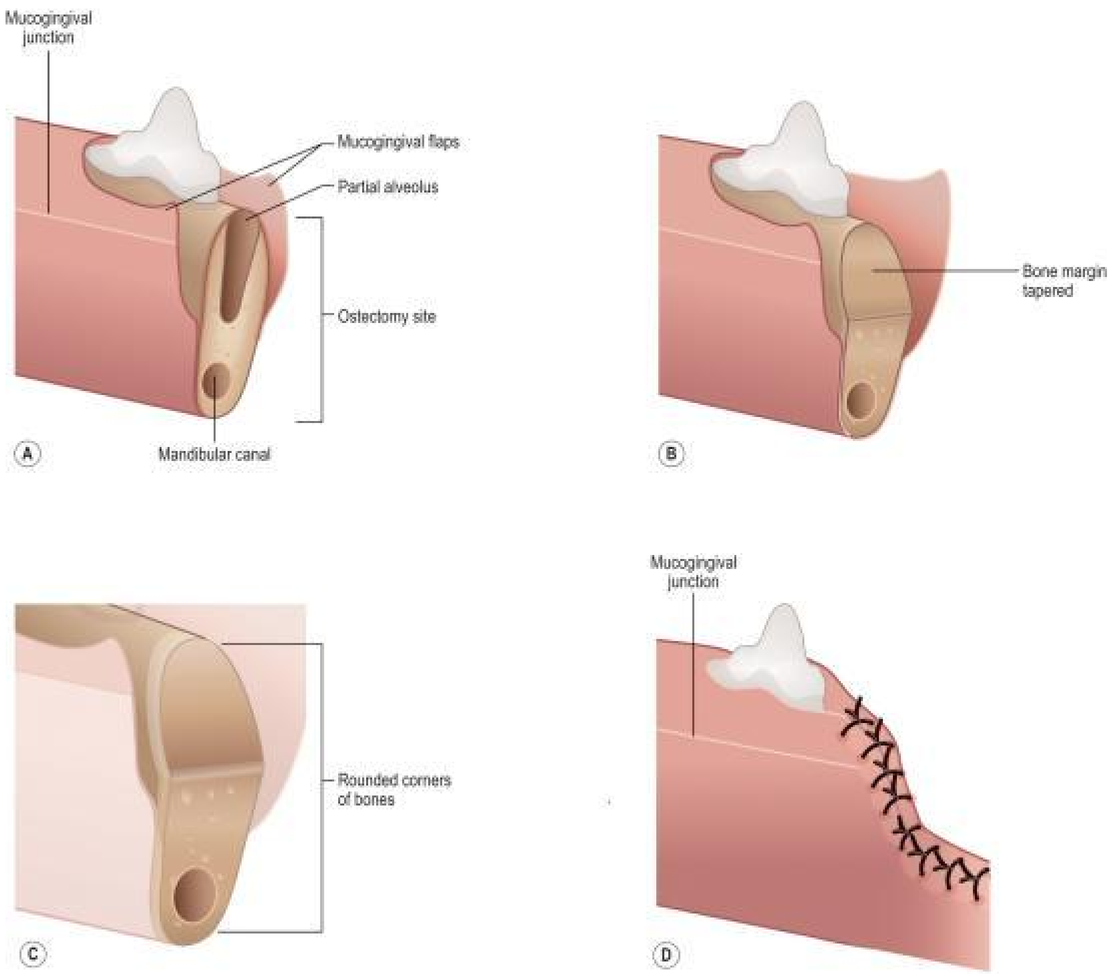


• **FIG. 51.5** Closure of a unilateral rostral mandibulectomy. (**A**) The vestibular mucosal–submucosal flap is sutured to the large amount of mandibular mucoperiosteal fibrous tissue using wide suture bites. (**B**) Closure results in an asymmetrical rostral mandible.

If an ostectomy of the rostral mandible is performed, the mucoperiosteal tissue margin is trimmed back 1–3 mm to expose a bone rim that will support the suture line. The new soft tissue margin is subperiosteally elevated to facilitate suture placement. A one-layer closure is performed and the suture line is supported by bone (Fig. 51.5B).

Closure at the mandibular body

Full-thickness ostectomies of the body of the mandible are made perpendicular to the long axis of the bone. For closure, the ostectomy may be modified to provide tension-free tissue apposition over the cut bone margin (Fig. 51.6).[2] As needed, an alveolar margin incision, combined with a sulcular incision partially around the adjacent intact tooth, is made. The attached gingiva, along with the proximal alveolar mucosa on the buccal and lingual side, is subperiosteally elevated. The corner of the ostectomy at the level of the alveolar margin is tapered 30–60 degrees with a saw blade or bur. Osteoplasty is then performed. The adjacent intact tooth is not injured. The attached gingiva is contoured and then sutured over the tapered area with simple interrupted sutures using appropriate tissue bites. The buccal and alveolar mucosal-submucosal flaps are apposed using simple interrupted sutures. Although this technique provides tension-free tissue apposition, the suture line in the attached gingiva may have stress applied during prehension and mouth closure. This is especially true for bilateral rostral mandibulectomies. The thick keratinized attached gingiva provides excellent suture retention. However, during early healing, all tension is placed on the sutures; therefore slightly larger-gauge sutures may be used for suturing the attached gingiva over the tapered alveolar bone margin.



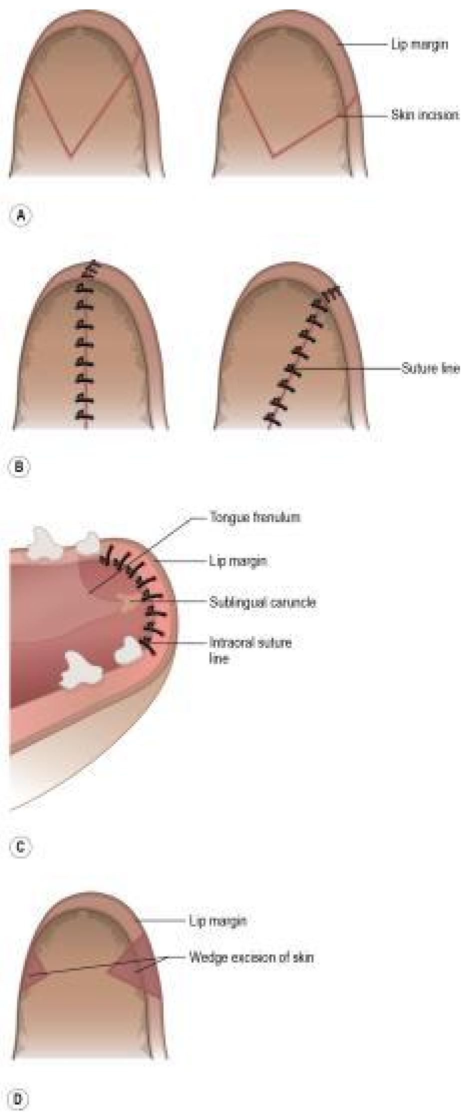
• **FIG. 51.6** Tapering the alveolar bone margin to reduce suture line tension. The adjacent tooth was transected during ostectomy; the root remnant has been extracted leaving a partial alveolus. (**A**) Alveolar margin and sulcular incisions are made, and the attached gingiva and proximal alveolar mucosa are subperiosteally elevated. (**B**) The alveolar margin bone is tapered approximately 30–60 degrees. (**C**) Osteoplasty is performed by rounding the bony edges. (**D**) The attached gingiva and alveolar mucosa is closed over the surgical defect.

Unilateral rostral mandibulectomy

This procedure usually includes removal of the incisor teeth, canine tooth, and first and second premolar teeth in the dog, and incisor and canine tooth in the cat. It may be difficult to obtain an adequate surgical margin on the medial aspect. In this case, instead of splitting the mandibular symphysis, an ostectomy of the rostral aspect of the opposite mandible may be needed. In the dog, the ostectomy generally includes the first and second premolar teeth, as the root of the canine tooth extends up to the level of the distal root of the second premolar tooth. If only incisor teeth are removed, a portion of the mandibular symphysis remains intact. Removal of the incisor part is only rarely indicated due to the proximity of the canine tooth and the need for adequate surgical margins. The labial vestibular mucosal–submucosal flap is sutured to the mucoperiosteum of the remaining rostral mandible (see Fig. 51.5) and alveolar mucosa if the excision extends caudal to the symphysis. Lip wedge excision to remove excess tissue before closure is not needed.

Bilateral rostral mandibulectomy

The surgical margins are determined and the amount of skin left for tension-free closure is estimated. The osteotomy is made at the distal aspect of the second premolar teeth to ensure complete removal of the canine teeth alveoli. The incision is made as a lip wedge excision to include surgical margins and estimated excess skin. For tumors centered at the incisive part and extending facially, a single wide symmetrical wedge of skin is excised on the facial aspect as part of the wide excision (Fig. 51.7). For tumors that occur more caudally or laterally, two skin wedges are removed at the level of the lateral lower labial frenula or a single large asymmetrical skin wedge is removed. Wedge excision is conservatively performed to ensure that an adequate amount of skin is present for tension-free closure. Bilateral full-thickness wedge removal of lip margins sacrifices the inferior labial blood flow to the remaining rostral lip and skin segment. Large bilateral wedge excisions may compromise the remaining random blood supply to this rostral segment. A small portion of the caudal mandibular symphysis may remain intact. Mandibular and sublingual salivary ducts and caruncles are avoided. If the ducts must be transected, as in mandibulectomies at the level of the third or fourth premolar teeth, they are ligated. The mucoperiosteum at the rostral mandible is tightly attached, and sharp dissection may be more efficient than blunt tissue elevation. The rostral mental blood vessels at the level of the apex of the second incisor teeth are ligated or coagulated. When transecting the lateral lower labial frenula, the middle mental blood vessels are encountered and are ligated to prevent unnecessary blood loss. The osteotomy is preferably performed with an osteotomy bur on a surgical handpiece with continuous irrigation in a careful manner to avoid transecting the inferior alveolar blood vessels without prior ligation.



• **FIG. 51.7** Bilateral rostral mandibulectomy. The patient is in dorsal recumbency. (**A**) The wedge incision (either symmetrical, left, or asymmetrical, right) incorporates the surgical margins and estimated excess skin and lip for closure. (**B, C**) Closure results in a linear skin suture line and a transverse oral mucosal suture line. (**D**) Alternative skin and lip closure with bilateral removal of full-thickness labial tissue.

After excision, osteoplasty of the ventral margin is performed using a bur to provide a more normal "chin" contour. If the excision removed the entire symphysis, the ostectomies of each mandibular body are tapered at the alveolar margin (Fig. 51.6) and the attached gingiva is closed. A suture is placed in the lip margin to maintain alignment for cosmetic closure. The oral mucosa is apposed using simple interrupted sutures with knots placed in the oral cavity. The remaining closure is routine.

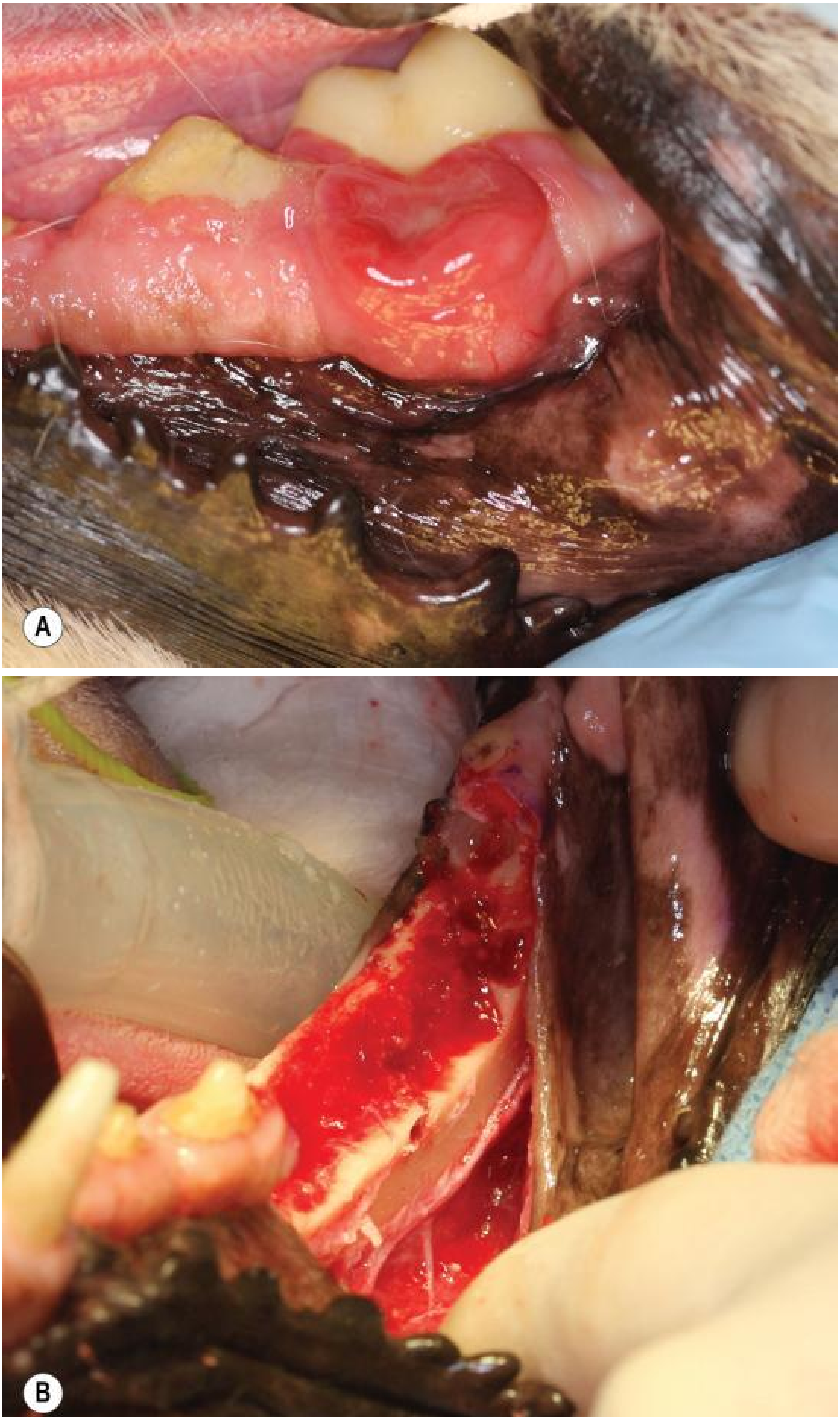
Segmental mandibulectomy

A segment of the mandibular body is excised, leaving behind two ostectomy cuts. Each ostectomy is tapered at the alveolar margin (Fig. 51.6). The attached gingiva and mucosal tissues are sutured. The salivary ducts are avoided when possible. If the ducts are transected, they are ligated.

Segmental mandibulectomy with preservation of the mandibular canal and ventral margin—rim excision[1,33]

In a standard segmental mandibulectomy, the ostectomy involves a full-thickness (dorsoventral) segment of the mandible. However, in the case of smaller neoplasms, adequate surgical margins may be achieved by partial-thickness excision, known as *rim excision*, allowing the mandibular canal and thick ventral cortical bone or a portion of the mandibular symphysis to remain intact (see Fig. 51.3).[1] Postoperative mandibular instability and malocclusion are therefore avoided.

This surgical technique is indicated for excision of minimally invasive mandibular tumors at the level of the premolar and molar teeth in medium to large dogs (Fig. 51.8A). In small-breed dogs, this type of ostectomy is less likely to achieve tumor-free margins and is likely to fracture the mandible or compromise its ventral border.[33]


A
B

• FIG. 51.8 Segmental mandibulectomy with preservation of the mandibular canal and ventral margin—rim excision. The dog is in sternal recumbency. (**A**) Plasmacytoma at the buccal aspect of the left mandibular fourth premolar and first molar teeth. (**B**) A curvilinear rim mandibulectomy was performed, leaving the mandibular canal and ventral mandibular margin intact. (**C**) The remaining attached gingiva and alveolar mucosa were sutured over the bony defect.

Using an intraoral approach to the mandible, buccal and lingual mucosal incisions are made to obtain a 10-mm clean margin beyond neoplastic tissue. Diagnostic imaging is essential to ensure that there is 10 mm between the visible tumor and the mandibular canal. A slightly narrower margin may be considered for very benign tumors, such as peripheral odontogenic fibroma. After subperiosteal soft tissue elevation, a curvilinear rim mandibulectomy is performed using precision osteotomy equipment, i.e., an oral surgery handpiece or piezosurgery, leaving the mandibular canal and ventral mandibular margin intact, followed by osteoplasty (Fig. 51.8B). The remaining attached gingiva and alveolar mucosa are sutured over the bony defect (Fig. 51.8C). Mandibular rim excision, with preservation of the ventral margin and mandibular canal content, preserves the occlusion and leaves the neurovascular structures intact. However, case selection is crucial, and this technique is recommended only for treatment of early odontogenic and malignant lesions of the mandible in medium-to large-breed dogs.[33]

Caudal mandibulectomy

Depending on neoplasm size and location, a full-thickness cheek incision may be made from the commissure and extended caudally as needed. Mucosal incisions are made along the measured surgical margins. However, it is often unnecessary to incise the cheek to achieve surgical margins and have good site exposure, especially when dorsolateral or sternal positioning is used. Using the intraoral approach, a mucosal incision is made over the rostral edge of the ramus then extended in a buccal and lingual direction around the mandibular body along surgical margins. After dissection of the caudal part of the mandibular body and ostectomy, dissection along the ramus is performed. The insertions of the muscles of mastication are subperiosteally elevated or the muscle body is transected, depending on surgical margins. If needed, the masseter is excised as part of the en bloc excision. The digastric and pterygoid muscles are elevated or transected, as determined by surgical margins. The mandibular foramen is located and the inferior alveolar artery is ligated and transected close to the foramen to avoid injury to the maxillary artery. The inferior alveolar nerve is also transected as close as possible to the foramen, to avoid damage to the lingual nerve. The temporalis muscle insertions on the medial and lateral aspects of the ramus and coronoid process are elevated or transected, as determined by the surgical margins. The temporomandibular joint is located by palpation during manipulation of the caudal mandibular en bloc segment. The capsule is incised on the medial aspect. As the mandible is manipulated to aid visualization, adjacent soft tissues are gently "wiped" off the capsule and the capsule incision is extended laterally. The rostral and caudal aspects of the capsule are incised in this manner and the lateral ligament is incised last. It is essential that dissection and joint capsule incision occur immediately adjacent to the condylar process to avoid injury to adjacent vessels and nerves. The tortuous course and close proximity of the maxillary artery on the ventromedial aspect of the joint, and its local branches, make them vulnerable to inadvertent transection. Once the joint is disarticulated, any remaining soft tissue attachments are cut. A local anesthetic "splash" block may be applied to the surgery site. Placement of local hemostatic agents (see Chapter 8) may be needed to help control bleeding from transected muscles. Oral mucosal tissues are opposed using simple interrupted sutures. Closure is routine.

Unilateral (total) mandibulectomy

Penetration of the mandibular canal implies potential spread of neoplasm along the length of the mandible, indicating the need for total mandibulectomy. This procedure can be performed with the patient in lateral recumbency using an incision of the cheek. This may facilitate the caudal dissection, especially in some large patients and in patients with more caudally located masses necessitating excision of the masseter muscle, and transection rather than subperiosteal elevation of other muscles. The procedure can be performed intraorally with the patient in sternal recumbency. The intraoral incision is started as described for caudal mandibulectomy and continued along the predetermined surgical margins. The mandibular symphysis is split using a thin osteotome and mallet, and the body and ramus are dissected free of all tissue attachments, as described earlier for the various partial mandibulectomy techniques. If necessary for surgical margins, the tongue frenulum can be removed without altering tongue function. Soft tissue excision may extend toward the opposite mandible. However, this excision margin is limited by the need for primary mucosal closure. Extensive removal of sublingual muscle may result in impairment of tongue function. The mandible is excised intact.

The commissure may be moved rostrally to minimize drooling and help prevent tongue protrusion (see Chapter 56). Commissurorrhaphy is performed by excision of the lip margin, followed by a three-layer closure. The new commissure is approximately at the level of the mandibular second premolar tooth. A full-thickness horizontal mattress suture may be placed to span between the upper and lower lip immediately rostral to the new commissure. This suture may be tied over buttons or suture bolsters. Its purpose is to restrain mouth opening to keep tension off the commissure suture line and promote normal healing.

Reconstruction following mandibulectomy

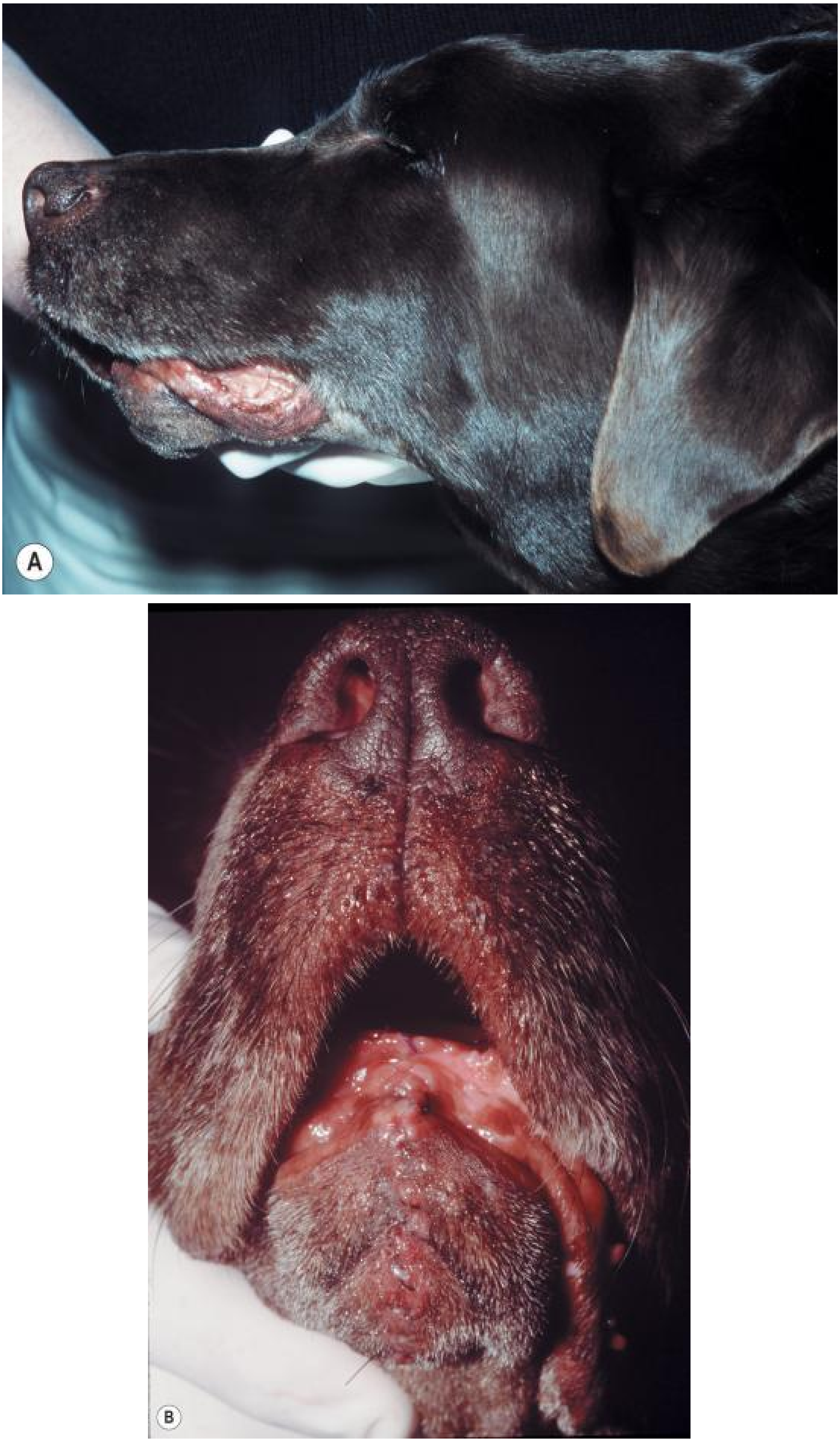
Historically, reconstruction following mandibulectomy has infrequently been performed.[34] An ulnar autograft combined with rigid internal fixation was used to bridge a central mandibulectomy site for benign neoplasm excision, with good results.[35] Stabilization of the remaining lower jaw after bilateral rostral mandibulectomy has been attempted using orthopedic pins,[36] screws,[4,7] and screws and bone graft.[37] A microvascular bone autograft using coccygeal vertebrae has been reported for fracture reconstruction of a central mandibular defect in a dog.[38] Microvascular transfer of bone autografts is the preferred method of mandibular reconstruction in humans.[39] Mandibular reconstruction following segmental and bilateral rostral mandibulectomy in dogs using rhBMP-2 was recently introduced (see Chapter 53) and has since been used by the authors (FV and BA), with excellent results.[26,27]

Postoperative care and assessment

Most aspects of postoperative care, including pain management and nutritional support, are similar to those described for maxillectomy (see Chapter 50).

Postoperative patient appearance[21-23]

Swelling of the surgical site is usually resolved by 3 to 7 days postoperatively. Cosmetic appearances are generally good and depend on the extent of excision. Smaller excisions result in unnoticeable or minimal cosmetic abnormalities. Regrowth of hair will conceal many surgically created facial asymmetries. Unilateral rostral mandibulectomy without canine tooth removal results in no obvious abnormality. When the canine tooth is removed, the tongue hangs out from that side when panting. Bilateral rostral mandibulectomy results in relative mandibular brachygnathism and the most noticeable uncosmetic appearance (Fig. 51.9). The tongue protrudes from the mouth during panting. Drooling may occur. Central mandibulectomy results in a mild facial concavity. Caudal and complete mandibulectomies result in facial concavity and tongue protrusion from the side of surgery during panting (Fig. 51.10). Malocclusion occurs as the remaining mandible shifts toward the operated side.



• **FIG. 51.9** (**A**) Lateral and (**B**) ventrodorsal view of a patient after bilateral rostral mandibulectomy.


A


B

• **FIG. 51.10** (**A**) Lateral and (**B**) ventrodorsal view of a patient after a total mandibulectomy of the right mandible. Lateral tongue protrusion during panting and mandibular drift to the right side are visible.

Homecare and follow-up

Most patients are sent home in 1 to 3 days postoperatively. A soft food diet and removal of chew toys are recommended for 2 to 3 weeks. The client is shown how to administer any oral medications so as not to disrupt the suture line. Elizabethan collar is needed to prevent self-inflicting damage to the surgical site. Medications are concealed in soft food snacks. If oral manipulation is needed, the manipulation is performed away from the surgical site. The surgical site is examined in 2 weeks and any skin sutures are removed. The surgical site is reexamined at 1 month postoperatively and any residual oral sutures are removed at this time. Most oral sutures are extruded in approximately 2 to 3 weeks. A complete physical examination, including the surgical site, is performed at 3-month intervals for 1 year. In case of malignant neoplasms, three-view thoracic radiographs are obtained at these examinations.

Complications[19-23]

Intraoperative complications

Hemorrhage is the major intraoperative complication. It is managed as it is encountered by ligation, pressure, or, in exceptional cases, focal use of electrocoagulation. The risk for excessive bleeding is the highest during ostectomy and during dissection of the caudal mandible. Blood loss during ostectomy is minimized by cutting the bone of the ventral mandible last and isolating and ligating the inferior alveolar artery. In the event of inadvertent transection of the artery, rapid completion of the ostectomy is done and the vessel is retrieved and ligated. If the artery retracts into the canal and cannot be retrieved, the mandibular canal is packed with a suitable hemostatic agent (see Chapter 8). Careful dissection for disarticulation of the temporomandibular joint is required to avoid vascular injury and profuse hemorrhage.

Dehiscence

A small dehiscence over the ostectomy site exposing bone will usually heal by second intention. A larger dehiscence requires debridement and delayed primary closure.

Functional complications

Malocclusion

The contralateral mandible is initially unstable and drifts toward the midline. Elastic training using orthodontic buttons and power chain between the buccal aspect of the maxillary fourth premolar tooth and the lingual aspect of the mandibular canine tooth is a simple method for prevention of mandibular drift but requires good client compliance.[40] Abnormal contact with the remaining mandibular canine tooth may result in palatal or upper lip ulceration. Crown reduction and partial coronal pulpectomy, or extraction of the mandibular canine tooth, is needed to resolve the palatal ulceration.[41] The remaining mandible may occasionally drift away from the midline. When this happens, the mandibular and maxillary teeth meet end on. Clicking is briefly audible until the mandible drifts medially. The unstable mandibular movement is reduced 4–6 weeks after surgery; however, occlusion may remain abnormal. After experimental unilateral rostral mandibulectomy, temporomandibular degenerative joint disease occurred but did not impair the dogs at 3 and 6 months postoperatively.[42]

Prehension

Most dogs prehend and drink normally after partial mandibulectomy. Patients with more extensive excisions practice eating and adapt after a short period of time. Some dogs spill a large amount of food around the food bowl during this period. Food with a consistency similar to ground meat or soaked kibble seems the easiest to prehend. Hand-feeding may be permanently necessary with some patients after a one-and-one-half mandibulectomy. These patients usually adapt to drinking by a combination of lapping and sucking water.

Drooling

Drooling occurs with bilateral rostral mandibulectomies and total mandibulectomies. Drooling often decreases over time. If the new lip margin is immediately rostral to the sublingual caruncles when closing a bilateral rostral mandibulectomy, the mandibular and sublingual salivary ducts may be ligated to reduce drooling potential. Moving the commissure rostrally during closure of a total mandibulectomy usually prevents drooling.

Glossoptosis (tongue protrusion)

Lateral tongue protrusion occurs when a mandibular canine tooth is absent, as in a unilateral rostral mandibulectomy or total mandibulectomy. Ventral tongue protrusion occurs following a bilateral rostral mandibulectomy. The protrusion is most evident when panting. Desiccation of the tongue rarely occurs.

Ranula—sublingual swelling

A ranula may result from surgical trauma to the salivary duct or papilla or postoperative swelling that temporarily impairs flow of saliva from the sublingual caruncle. This condition may occur at 1 to 2 days postoperatively and usually resolves by 5 to 7 days. Treatment is typically not needed.

Dental calculus

The ipsilateral maxillary fourth premolar tooth and molar teeth may accumulate dental calculus more rapidly, as compared to the contralateral side, in patients with central, caudal, and total mandibulectomies. There is an apparent compromise of normal oral cleansing, possibly due to a closer postoperative adaptation of the cheek to the maxillary teeth surfaces. Oral health checks are essential for the life of the patient.

Local tumor recurrence

Local tumor recurrence or persistence may occur if clinical staging and surgical margins were inaccurate, and with certain tumor types, like fibrosarcoma and osteosarcoma. Suspicious areas noted during recheck examinations must be biopsied to differentiate neoplasm recurrence from suture reaction and scar tissue.

Prognosis

The overall prognosis for mandibulectomy techniques regarding oral function, operative and postoperative complications, postoperative appearance, and client satisfaction is good to excellent.[15,21-23] This seems directly related to surgeon experience with appropriate patient selection, technical performance of the surgery, and preoperative client communications to set realistic expectations. Less positive results may occur in patients with more aggressive disease and more radical surgery. One study reported an overall 85% client satisfaction with maxillectomy and mandibulectomy procedures. The satisfaction level correlated with the postoperative survival time of the patient and the accurate prediction of outcome and complications.[15]

The local recurrence rates, survival times, and biologic behavior of benign and malignant oral tumors reported in various clinical studies have been reviewed.[23,24,43] Fibrosarcoma has the highest local recurrence rate (46%), followed by melanoma (25%), osteosarcoma (25%), squamous cell carcinoma (15%), and benign neoplasms (0%–4%).[23,43] Early recognition, accurate diagnosis, and aggressive, planned excision are essential for optimal results.

Other considerations

Extent of mandibulectomy

Ideally, the functions of prehension and mastication for normal alimentation and hydration are maintained. When a one-and-one-half mandibulectomy is performed (see Fig. 51.3), many patients require manual assistance with eating and must be hand-fed food shaped into balls. Drinking is accomplished by a combination of lapping and sucking water. A very extensive mandibulectomy may interfere with the patient's ability to maintain hydration.

Subtotal mandibulectomy

This procedure leaves most of the ramus intact with a normal coronoid process and temporomandibular joint. This avoids the more difficult dissection to remove the caudal aspect of the mandible, thereby reducing surgical trauma. However, the surgeon must be sure that the soft tissue and bone surgical margins are not compromised. The mandibular foramen is located approximately halfway between the coronoid crest and medial aspect of the condylar process. Therefore transection of the mandibular body distal to the third molar tooth will result in a portion of intact mandibular canal being left behind. This is of particular concern when surgery is performed for a tumor that potentially invaded the mandibular canal. It may therefore be considered to make a caudoventrally directed osteotomy, similar to what is illustrated in Fig. 51.1, in order to remove the mandibular canal. Osteoplasty is important before mucosal closure. The normal movement of the intact caudal mandible may result in perforation of the oral mucosa or suture line disruption by sharp bone areas left at the ostectomy site.

Teeth rostral to ostectomy

Central and caudal mandibulectomy transect the inferior alveolar artery and nerve that are the major neurovascular supply to the remaining rostral mandibular teeth.[30] Primate studies of teeth encased in an ostectomized segment of bone that maintains most of its original soft tissue attachments and is stabilized in position with orthopedic wire somewhat mimics the clinical picture of central and caudal mandibulectomy in dogs and in cats in which intact teeth are in a rostrally located bone segment.[44-46] In these studies, it appeared that the periodontal vessels continued to provide blood supply to the dental pulp by anastomosis with apical vessels and through lateral canals.[44-46] This periodontal blood supply originated from the intact soft tissue vessels. The pulp vessel density was initially reduced but was found to increase over time. Ischemic injury was suggested by loss of odontoblasts, inflammation, fibrosis, and foci of pulp necrosis. The pulp vessel density increased over time, and odontoblasts with secondary dentin production and reduced inflammation and necrosis were found. Pulp nerve fiber degeneration occurred. Therefore dental pulp of teeth in isolated segments of bone with intact soft tissues maintains vascularity although ischemia may initially occur. Endodontic intervention is not recommended unless clinically indicated.

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CHAPTER 52

Axial-pattern flaps for maxillofacial reconstruction

Mark M. Smith

Definition

Axial-pattern flap: A pedicle flap of skin and subcutaneous tissue that incorporates a direct cutaneous artery and vein into its base.

Therapeutic decision-making

Animals with no radiographic signs of distant metastasis should be considered for aggressive therapy. The concept of complete local excision of all visible tumor followed by, or concurrent with, chemotherapy or radiation therapy for treatment of presumed micrometastasis has achieved marked acceptance in human oncologic therapy and is being applied in veterinary medicine.[1-4] Surgery is integral to this multimodality treatment plan, especially for large, aggressive neoplasms.[5-8] Tumor excision surgery plus radiation and/or chemotherapy are usually well tolerated by dogs and cats, leaving conservative management for debilitated and geriatric patients.[9]

The goal of surgery for oral neoplasms in small animals is generally curative excision or palliation.[10] The ideal surgical procedure is one that offers the greatest possibility of cure, restores or maintains function, and has an acceptable cosmetic result. Extended dissection from the primary site may improve the incidence of free margins related to surgical excision of direct metastatic pathways, especially for neoplasms of the floor of the mouth. The cranial cervical area may be approached in conjunction with reconstructive surgery. Direct observation of regional lymph nodes allows assessment of gross transcapsular spread of the tumor, which may warrant wider margins for adhered lymph nodes.[11,12]

Cutaneous maxillofacial defects can also result from trauma, complications of radiation therapy, or failure of another reconstructive surgical technique.[13,14] Second-intention wound management of such cutaneous defects is not usually possible based on the inherent mobility of functional areas of the head and an unacceptable cosmetic appearance of the wound during the management period. Further, the result of second-intention healing may contribute to impaired function of the eyelids, nares, or mouth.[15] Surgical techniques have been developed for cutaneous maxillofacial reconstruction that obviate the need to utilize second-intention healing. These surgical treatment methods include the subdermal plexus flap or random-pattern flap, the labial advancement flap, and the buccal rotation technique (see Chapters 28 and 49).[16,17]

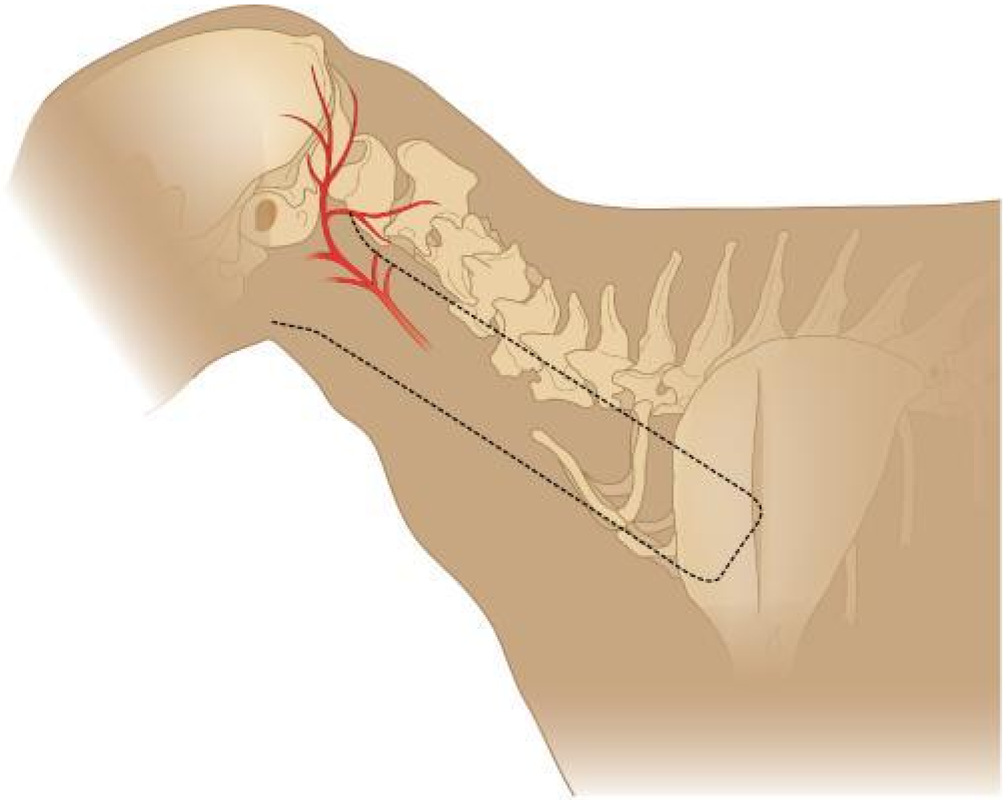
Axial-pattern flaps have been developed in dogs and cats to augment the treatment options for wounds with large skin defects.[16] Axial-pattern flaps have enhanced perfusion compared with subdermal plexus flaps because the former incorporates a direct cutaneous artery and vein at the flap base.[16] This enhanced vascular supply allows formation and transfer of a relatively large area of skin in a single-staged procedure to aid cutaneous reconstruction.[17] Identification and development of an axial-pattern flap for cutaneous maxillofacial reconstruction is of particular importance based on the aforementioned limitations of second-intention healing in this region.

Surgical anatomy and surgical techniques

Caudal auricular axial-pattern flap

Anatomical studies have shown that branches of the CAA provide blood supply to the cranial aspect of the cervical skin in dogs and cats.[18,19] The sternocleidomastoid branches supply skin, platysma, and subcutaneous fat; they eventually anastomose with the superficial cervical artery. These vessels emerge from deeper tissues in the area between the lateral aspect of the wing of the atlas and the vertical ear canal.[20]

Guidelines for flap location were based on these anatomical studies (Fig. 52.1). A palpable depression between the lateral aspect of the wing of the atlas and the vertical ear canal is the cranial border of the flap. The flap base is centered over the lateral aspect of the wing of the atlas. The flap is positioned in the center of the neck within ventral and dorsal lines paralleling the measured flap base width and the same width of measurement centered on the spine of the scapula. Flap dimensions are based on the feasibility of primary wound closure of the donor site and required length to transfer the distal flap to the facial area. Flap length may vary and does not necessarily extend to the spine of the scapula. The surgical plane of dissection is between the cervical neck fascia and the subcutaneous fat, since direct cutaneous arteries are located in the subcutaneous tissues.

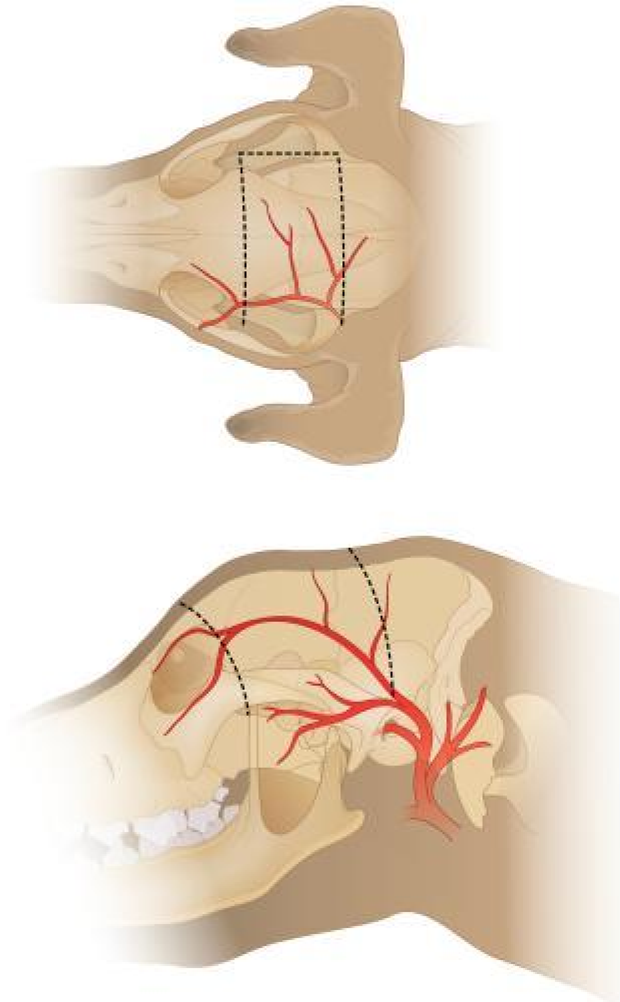


• **FIG. 52.1** Illustration showing location of the caudal auricular axial-pattern flap *(broken lines)* and the sternocleidomastoid branch of the caudal auricular artery in relation to the wing of the atlas and the scapula.

Superficial temporal axial-pattern flap

Branches of the STA provide blood supply to the skin and frontal muscle in the temporal region in dogs and cats.[21-23] The STA originates at the base of the zygomatic arch in a subcutaneous position and extends rostrally along the zygomatic arch, with small branches extending to the skin of the temporal region and the frontalis muscle.

The landmarks for the base of the STA flap are the caudal aspect of the zygomatic arch caudally and the lateral orbital rim rostrally (Fig. 52.2). Flap width is limited by the eye rostrally and the ear caudally and is therefore equivalent to the length of the zygomatic arch. The skin is incised and the thin frontal muscle identified superficial to the fascia of the temporal muscle. The flap is carefully elevated, deep to the frontal muscle, toward the flap base. A small superficial branch of the rostral auricular nerve plexus may be present at the cranial border of the flap over the ipsilateral eye adjacent to the flap base. Incision of this branch and the surrounding subcutaneous tissue enhances flap rotation. Flap length may vary and does not necessarily extend to the contralateral zygomatic arch. In fact, flap length approximating 75% of the distance from one zygomatic arch to the other is associated with reliable complete flap survival.

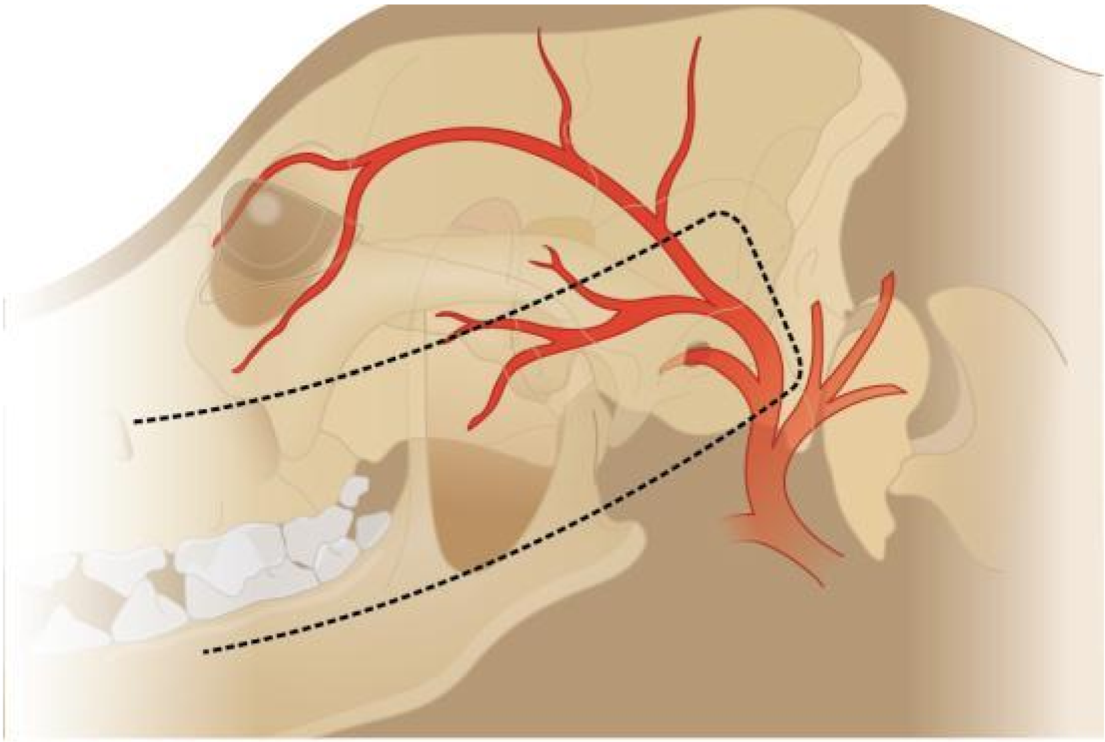


• **FIG. 52.2** Illustration showing the location of the superficial temporal axial pattern flap *(broken lines)* and the cutaneous branch of the superficial temporal artery in relation to the zygomatic arch and orbital rim.

Transection of the small superficial branch of the rostral auricular nerve plexus does not result in functional deficits of the eyelids. Wound dehiscence occurs at areas of flap necrosis. Redundant tissue over the eye due to rotation of flap tissue centrally, and mild tension over the contralateral eye, may occur secondary to primary closure of the donor site. However, in clinical cases, there were no associated clinical problems related to these transient abnormalities.

Angularis oris axial-pattern flap

The angularis oris axial pattern flap (AO) is especially indicated as a surgical procedure option for more rostral maxillofacial defects including the nasal planum.[24-29] The facial artery gives rise to its terminal inferior and superior labial arteries and the angularis oris artery at the caudal aspect of the commissure of the lip.[30] This flap has been described based at the commissure of the lip with its axial pattern vascular supply emanating from the perforating cutaneous branch of the angularis oris artery that courses caudally toward the base of the ear and arborizes with the perforating branches of the transverse facial and masseteric arteries (Fig. 52.3). Maintenance of this branch allows AO development in a caudodorsal direction with rotation to provide reconstruction of rostral facial defects.



• FIG. 52.3 Illustration showing the location of the angularis oris axial pattern flap *(broken lines)* and the cutaneous branch of the angularis oris artery in relation to the zygomatic arch and wing of the atlas. Note the rostral flap base.

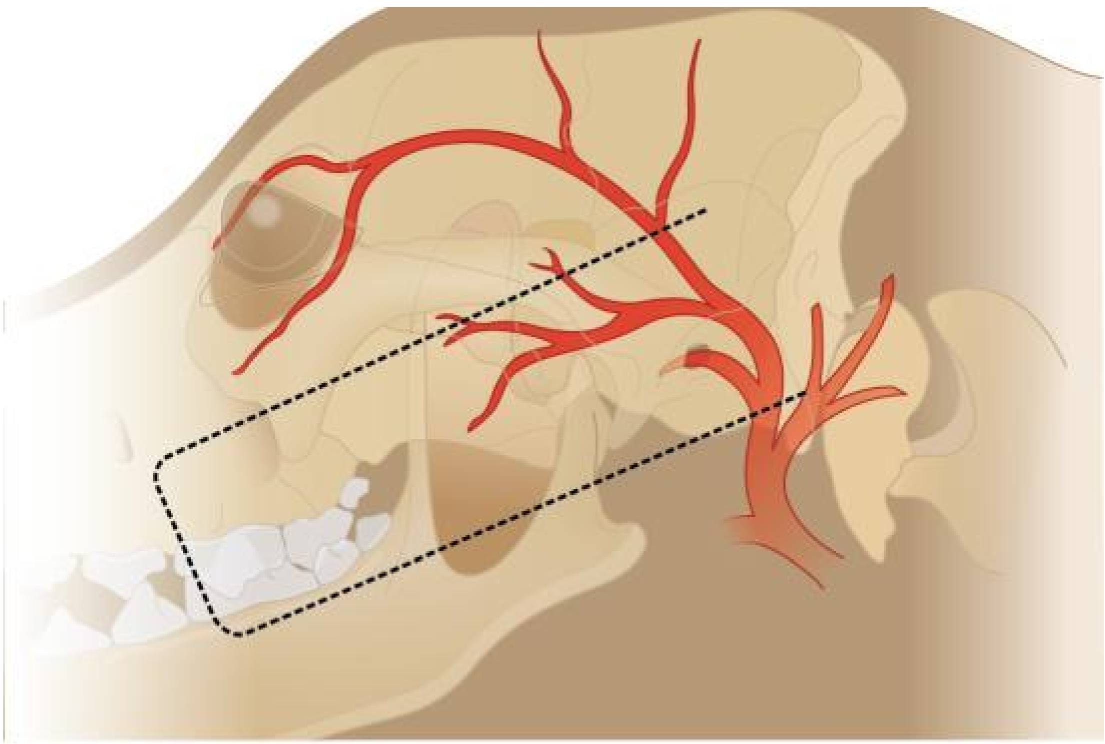
Guidelines for AO development include the dorsal incision along the ventral aspect of the zygomatic arch extending caudally to the level of the vertical ear canal or wing of the atlas, realizing that the longer the flap, the greater the incidence of distal flap necrosis secondary to decreased vascular supply. The ventral incision parallels the dorsal incision at the level of the horizontal ramus of the mandible. Joining these two incisions at the determined caudal location forms the caudal border (Fig. 52.3).

As with all axial pattern flaps, the vascular supply for the AO is located in the subcutaneous tissues necessitating that the plane of dissection be deep to the platysma and sphincter colli muscles, along the fascia of the masseter muscle. Care should be taken to avoid the dorsal buccal branch of the facial nerve and the parotid salivary duct. Branches of the auriculopalpebral and auriculotemporal nerves should be avoided with more caudal dissection in the area of the vertical ear canal and wing of the atlas.

An oral mucosal flap based on the angularis oris artery has also been described for palatal reconstruction.[31-33] A skin incision is made from the lip commissure caudally to the rostral aspect of the masseter muscle parallel to and avoiding the angularis oris artery, the pulse of which can be palpated or the vessel transilluminated with a focal light source in the oral cavity. Retraction of the skin margins exposes the angularis oris artery. Parallel dorsal and ventral mucosal incisions through the buccal mucosa and exposed subcutaneous tissues are made extending to the cranioventral border of the masseter muscle; they form the margins of the flap. This mucosal flap includes the buccal mucosa to the level of the lip commissure.

Transverse facial axial-pattern flap

Vascular supply for the transverse facial axial pattern flap (TFA) and clinical application in one case has been described in the cat.[34] The superficial temporal artery gives rise to the transverse facial artery. It courses in a rostral and slightly ventral direction to communicate with the angularis oris artery.[30] It is indicated for periocular reconstruction, wound closure following orbital exenteration, and midfacial defects.[35] Guidelines for TFA development are similar to those for the AO; however, the flap is based caudally in the area of the vertical ear canal (Fig. 52.4). The rostral, cutaneous component can extend to the commissure of the lip or include the commissure. The caudal flap base allows for the formation and utilization of a composite flap that incorporates the mucocutaneous junction and labial mucosa of the commissure of the lip for periocular reconstruction. The dissection is performed as described for the AO with similar concern to avoid the same anatomic structures.



• **FIG. 52.4** Illustration showing the location of the transverse facial axial pattern flap *(broken lines)* and the cutaneous branch of the transverse facial artery in relation to the zygomatic arch and wing of the atlas. Note the caudal flap base.

Application and surgical closure

These axial-pattern flaps have been developed and transferred immediately to allow extensive reconstruction as a component of definitive surgery. This characteristic is particularly useful in facial reconstruction following very wide excision or extensive trauma, because the transferred flap provides full-thickness skin that is durable in an area of movement (Figs. 52.5–52.8).

• **FIG. 52.5** En bloc excision and partial orbitectomy for fibrosarcoma in a 12-year-old cat. (**A**) Wide surgical margins are included with the lesion. (**B**) The length of the caudal auricular axial-pattern flap (CAA) is measured using umbilical tape. (**C**) The flap is elevated, revealing the direct cutaneous vascular supply. (**D, E**) A bridge incision communicates the donor and recipient sites followed by CAA apposition and donor site wound closure. (**F**) The 6-week postoperative examination indicates complete flap survival with cosmesis similar to enucleation.

• FIG. 52.6 (**A**) Preoperative photograph showing the dorsal view of a 1-year-old Border terrier following maxillofacial trauma. (**B**) The superficial temporal axial pattern flap is elevated. (**C**) The flap is sutured to the defect providing full-thickness skin to cover the wound. (**D**) Follow-up photograph.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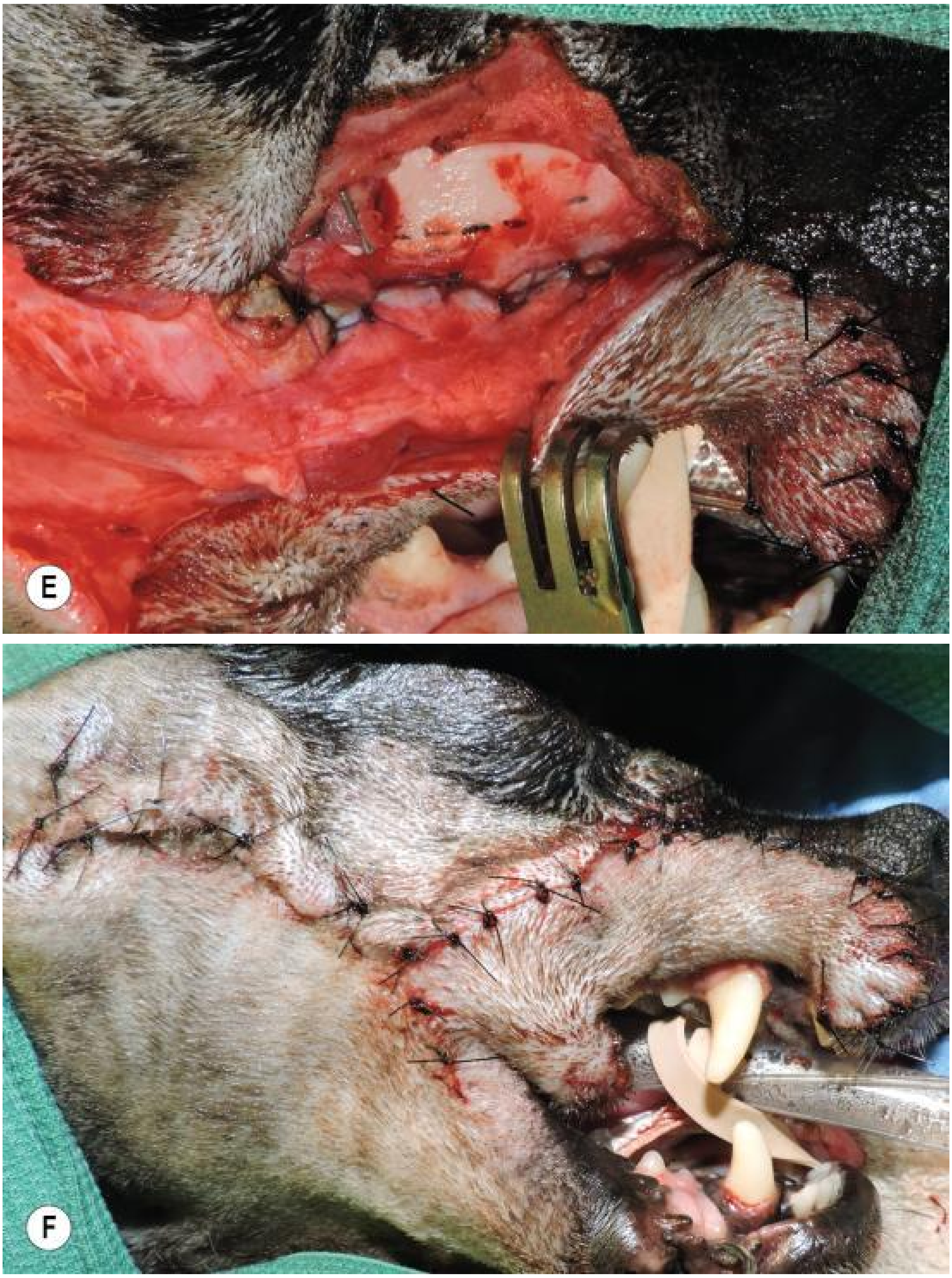
B


C
D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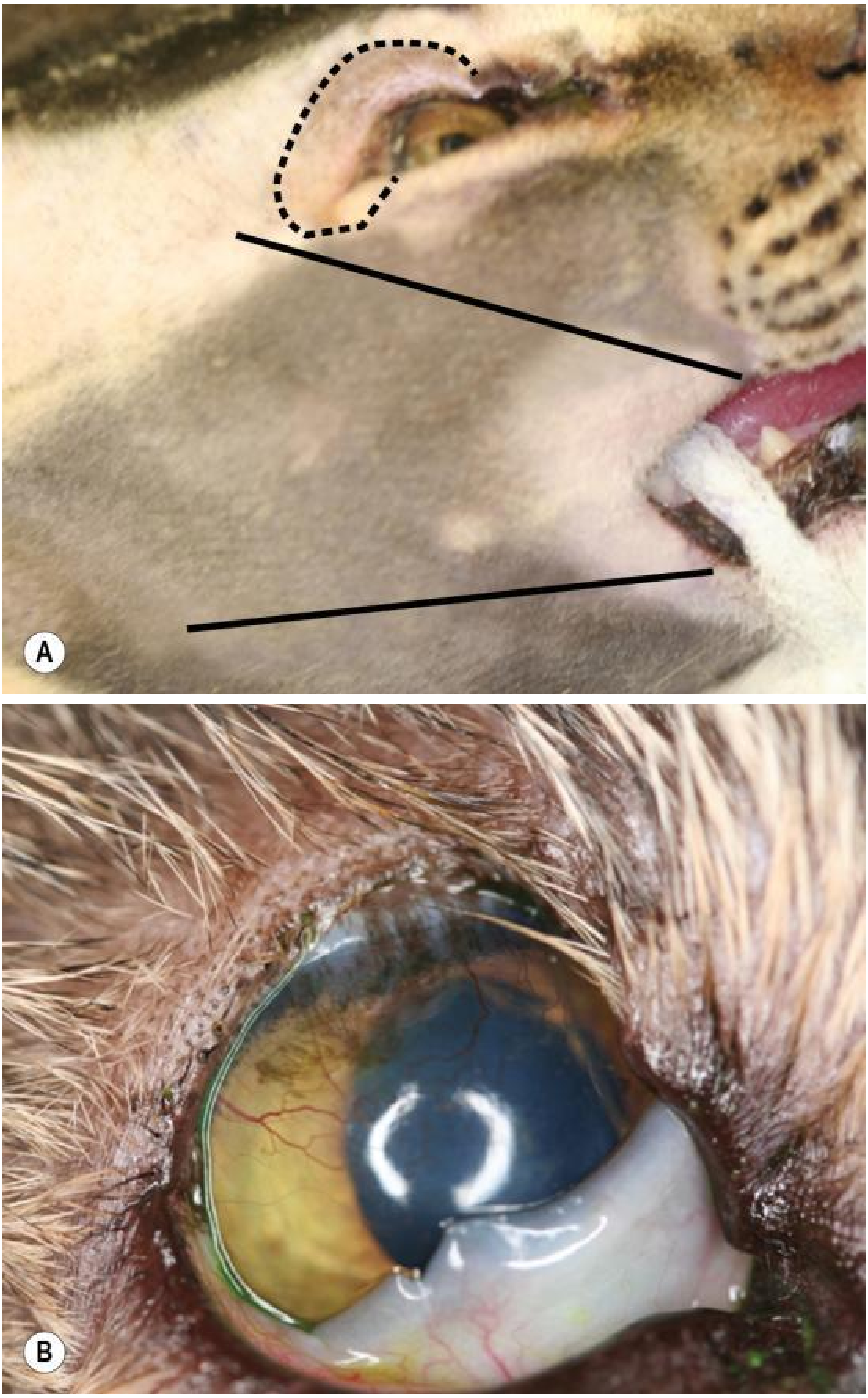
• FIG. 52.7 (**A**) The patient is prepared for surgery and an angularis oris axial pattern flap (AO) planned for maxillofacial reconstruction following surgery to excise (**B**) a right lip malignant melanoma in a 10-year-old mixed-breed dog. (**C**) Following en bloc excision, (**D**) the AO is rotated into position. (**E**) The ventral aspect of the flap is sutured to the oral mucosa juxtaposed with the mucogingival junction (**F**) followed by skin apposition. (**G**) Photograph showing primary healing of the transposed AO 3 weeks postoperatively.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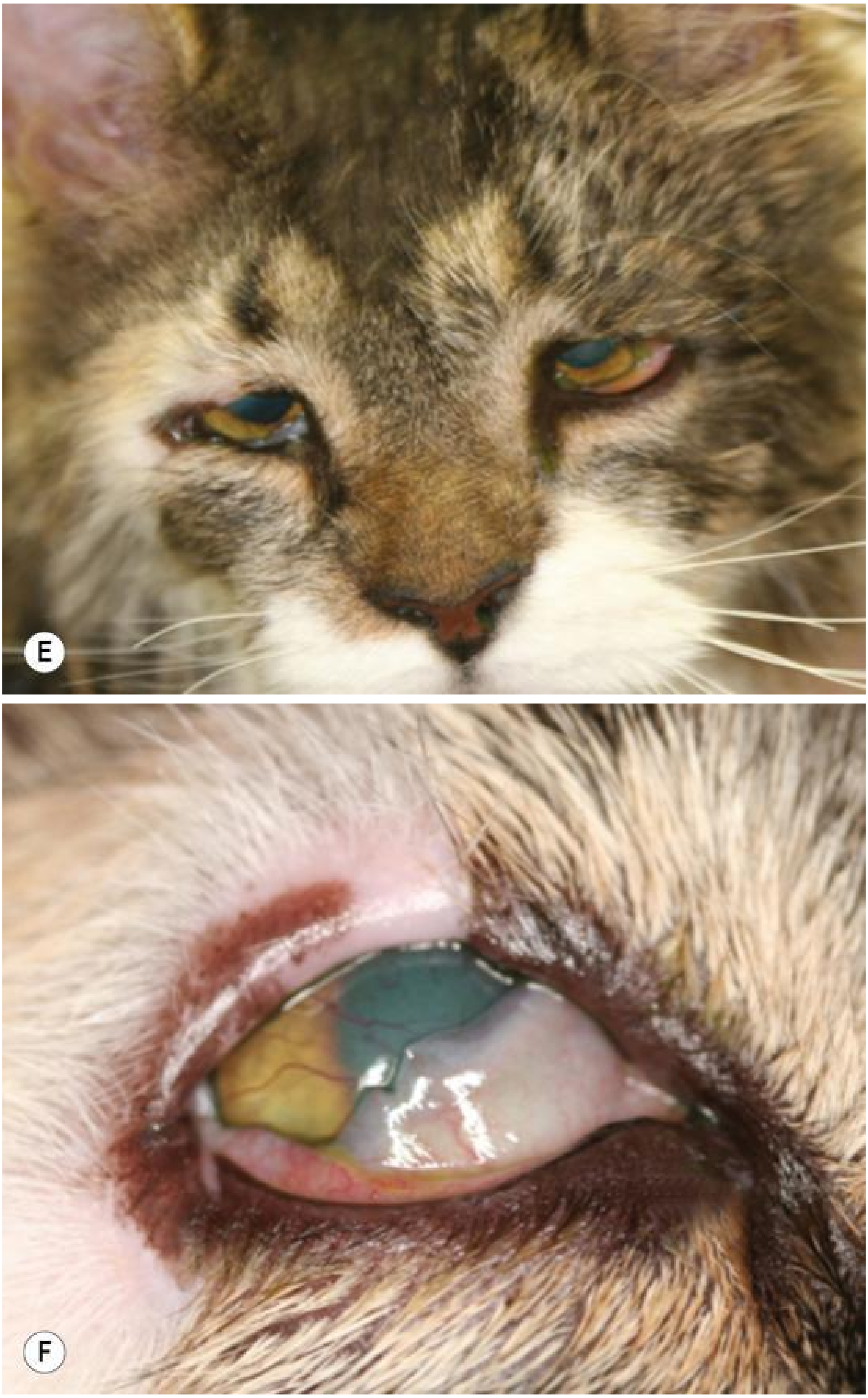


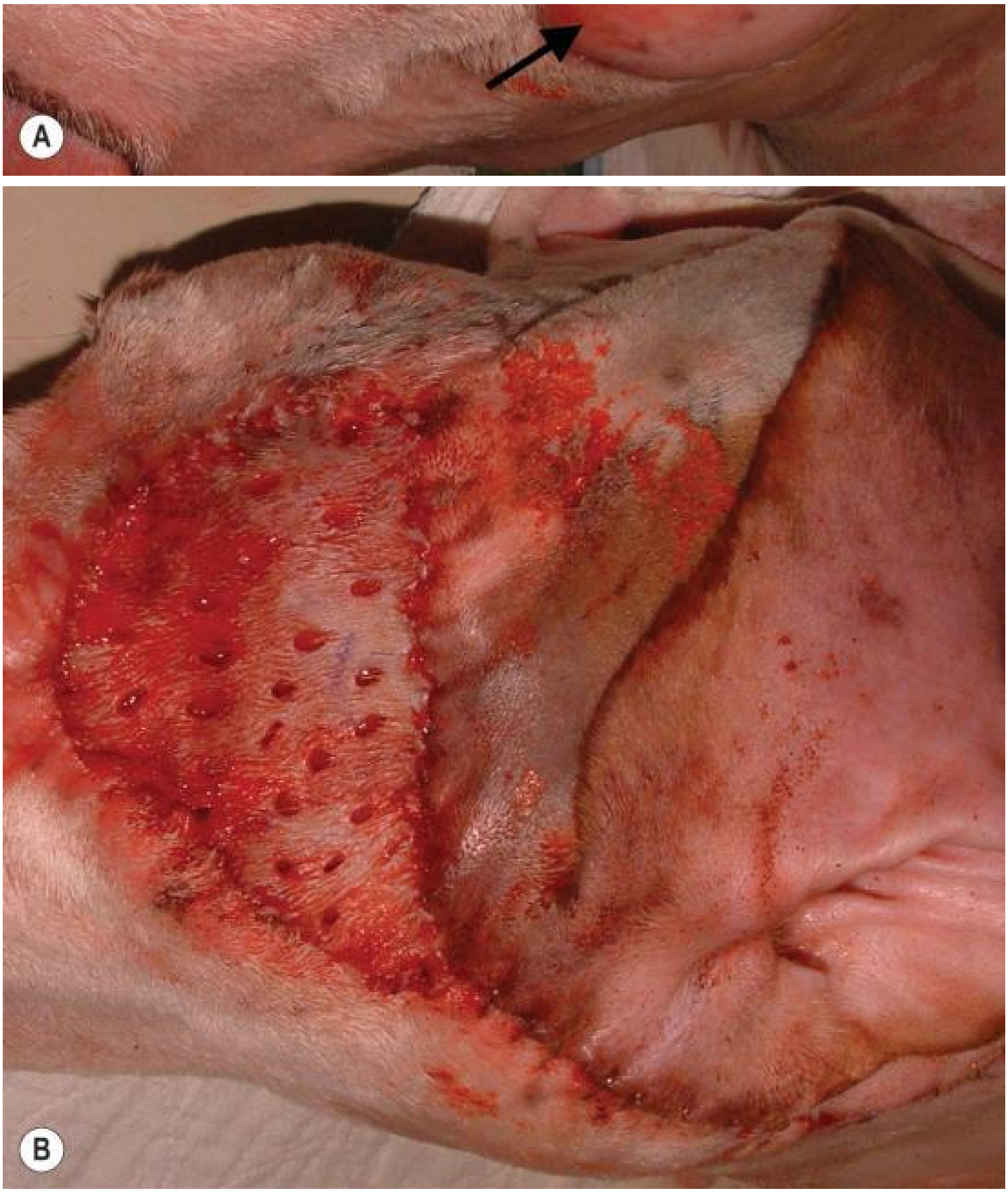
• **FIG. 52.8** (**A**) The patient is prepared for surgery and a transverse facial axial pattern (TFA) flap planned (**B**) for eyelid reconstruction in a 2-year-old domestic short-hair (DSH) cat with eyelid agenesis. (**C**) The TFA is elevated, including the mucocutaneous junction of the lip commissure and (**D**) the required amount of buccal mucosa for eyelid reconstruction. (**E**) The 1-week and (**F**) 6-week postoperative views show flap viability and healing to restore eyelid functional anatomy.

Flaps should be handled with care, and the base of the flap including the vascular pedicle should not be dissected or excessively manipulated, in order to avoid potential direct or indirect (vasospasm) vascular trauma that might negatively impact flap viability. Peripheral continuous or simple interrupted sutures are used to appose skin. Subcutaneous or tacking sutures to decrease dead space are not recommended, as they may occlude direct cutaneous vascular supply. Passive or active drainage systems may be used to prevent seroma formation. They should be placed beneath the flap, exiting through a separate site caudoventral to the flap base, in a position not expected to influence circulation to the flap. A stent bandage may be used to cover the drain exit site.

The donor site is closed in two layers including a subcutaneous–subcuticular pattern with tacking "bites" in the muscular fascia aimed at decreasing dead space. Any wound tension associated with donor site closure should be directed away from the flap, especially the flap base where the direct cutaneous vasculature is located.

Skin grafts

Full- or partial-thickness skin grafts have application for cutaneous defects of the maxillofacial area. Skin grafts are difficult to manage in this location because of the mobility of the area and the requirement for a bandage to promote graft stability and revascularization.[35] A tie-down bandage or closed-suction bandage may facilitate wound stability, leading to a successful outcome (Fig. 52.9).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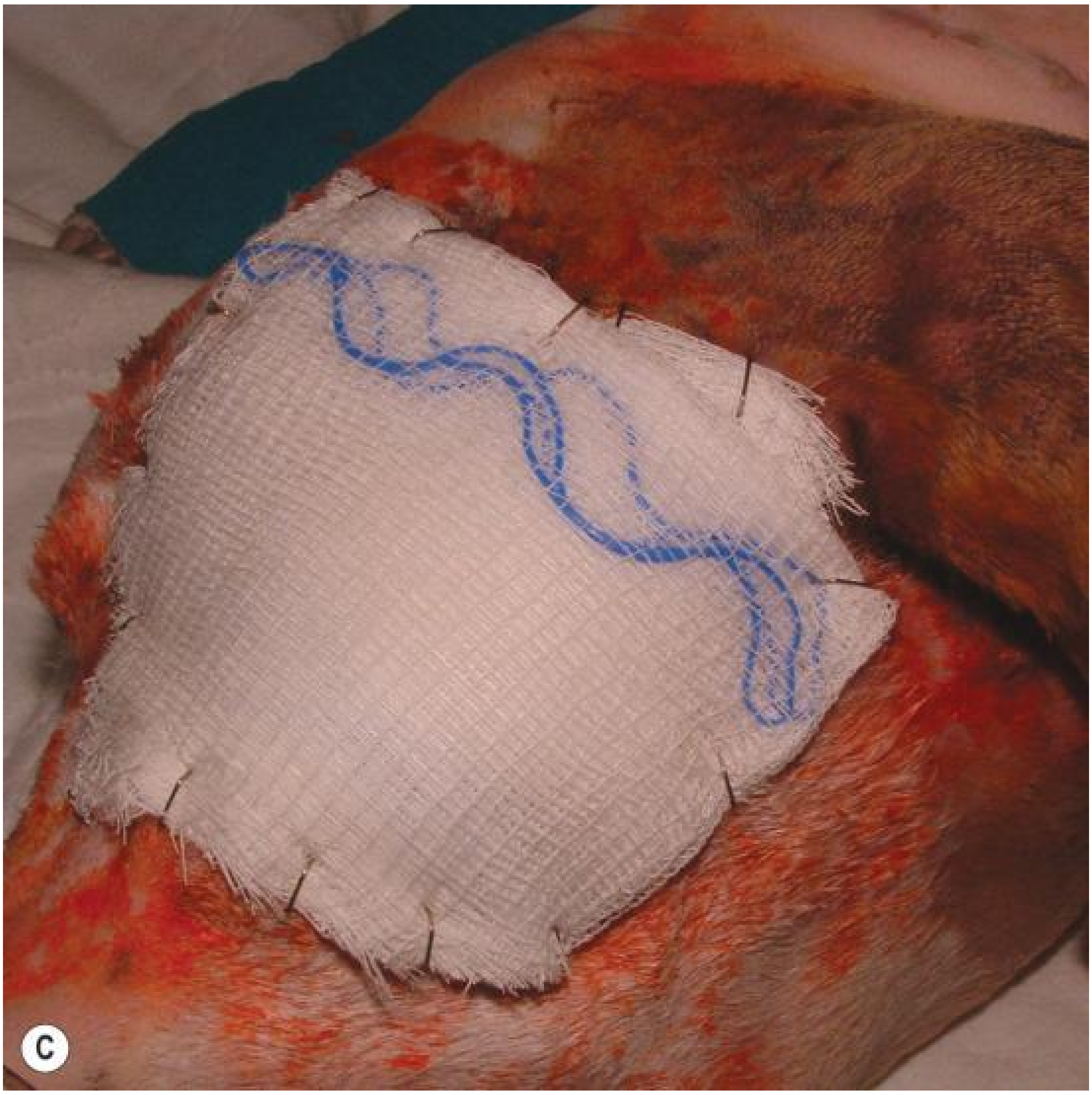
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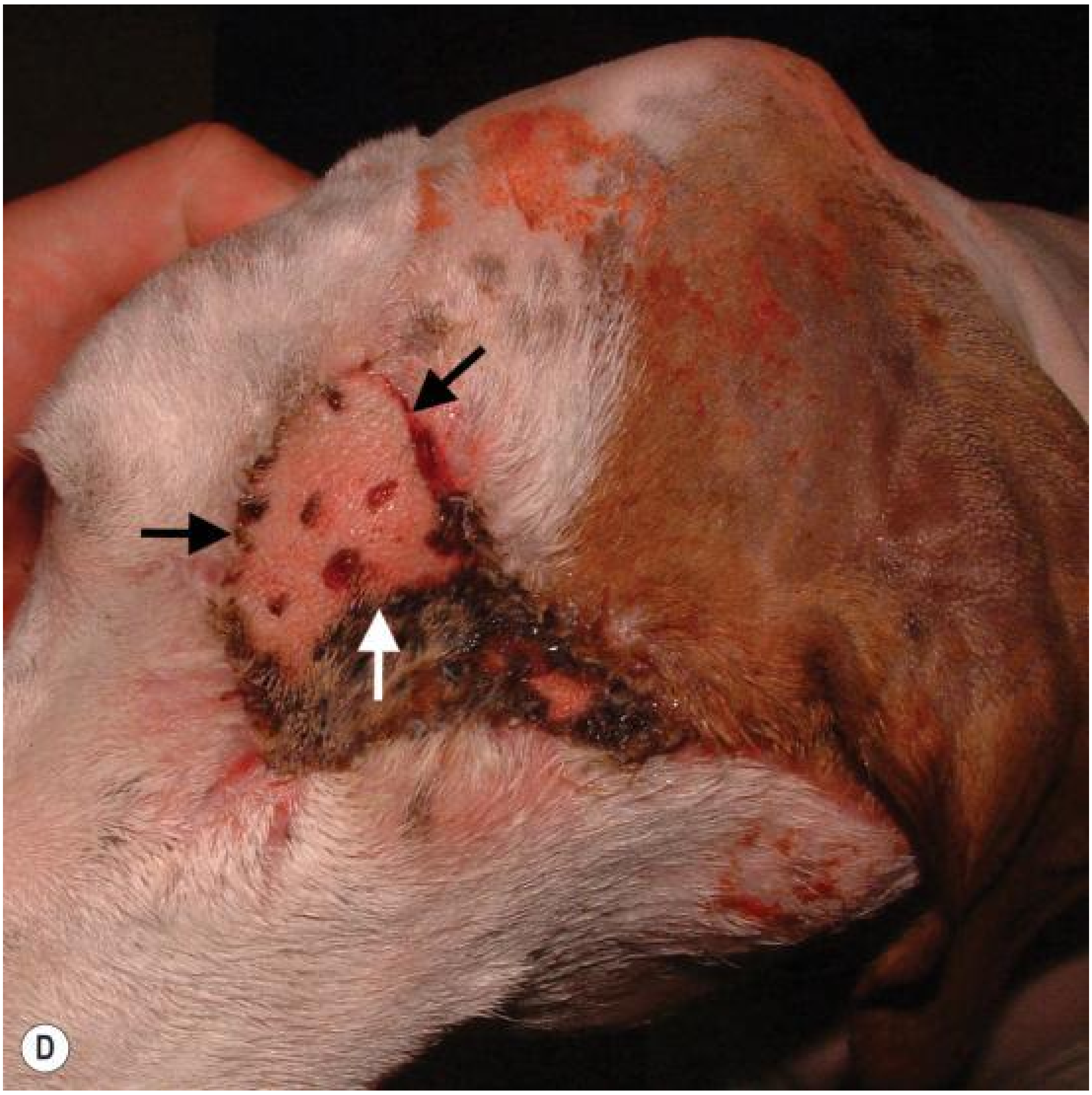


• **FIG. 52.9** (**A**) Skin defect following distal caudal auricular artery (CAA) flap necrosis after orbital exenteration and partial orbitectomy for a malignant neoplasm of the left ocular region. Note the area of granulation tissue and the distal aspect of the remaining CAA flap *(arrow)*. (**B**) A free skin graft was applied to the area. (**C**) A Stent bandage was applied. (**D**) Approximately 60%–70% of the skin graft was viable *(arrows)* 14 days following surgery. (Photographs courtesy of Dr. M. King.)

Postoperative care and assessment

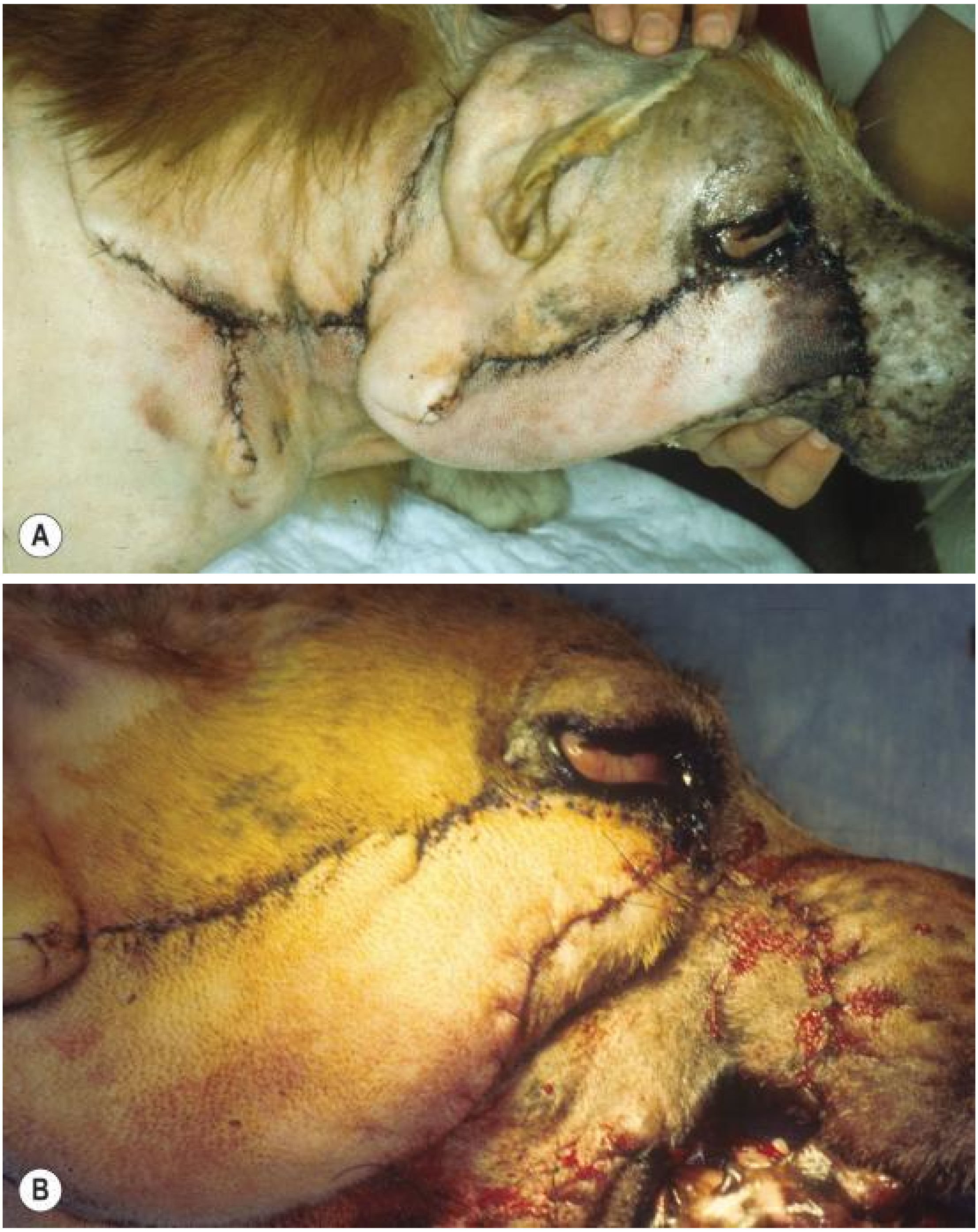
Immediate postoperative care

Skin cleansing of wound drainage and dressing changes are performed daily. Drains are removed by 48 hours postoperatively or when there is minimal drainage. An Elizabethan collar may be indicated to prevent self-mutilation.

Assessment—viability of axial-pattern flaps

The most detrimental complication associated with the use of axial-pattern flaps for wound reconstruction is devitalization of the flap apex, or end. Because of the nature of axial-pattern flap utilization, partial-flap necrosis occurs at an area where the flap is most important. This is particularly problematic in oral and maxillofacial reconstruction where second-intention healing following flap necrosis and wound dehiscence is not a viable management option.

Clinical signs of flap necrosis include a demarcated discolored (black/purple) area, palpably decreased temperature of the discolored area, decreased hair growth in the devitalized area, and wound dehiscence with drainage of a serosanguineous or purulent fluid if there is concurrent infection. The clinician should be deliberate when deciding when to revise the flap. Early intervention may result in even more tension applied to the flap and continued flap necrosis. It is advisable to wait 4–7 days until flap viability is ascertained and the risk of further devitalization is minimal. Another advantage of deliberate management is the improved blood flow to the base of the flap at the time when flap revision is performed (Fig. 52.10). Generally, the revision of a devitalized flap is successful, taking advantage of the increased blood flow and redundant skin present at the base of the flap.



• **FIG. 52.10** (**A**) Postoperative photograph showing skin discoloration demarcating viable and nonviable skin in a caudal auricular axial-pattern flap (CAA) used for maxillofacial reconstruction of a traumatic facial defect. (**B**) The CAA flap revision is performed by elevation and excision of the devitalized area followed by primary wound closure.

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CHAPTER 53

Regenerative techniques in maxillofacial surgery

Boaz Arzi, Frank J.M. Verstraete

Definitions

Regenerative surgery is defined as the application of regenerative materials and techniques using surgical methods to effect clinical therapy for disease or to restore normal function.[1] Although the bony skeleton has a remarkable capacity for spontaneous regeneration, in a setting of skeletal injury beyond a critical size, spontaneous healing does not occur, leading to nonunion and the formation of scar tissue.[2] Skeletal defects resulting from complex trauma, tumor excision, or congenital deformities often require extensive bone reconstruction. However, clinically available sources of bone for skeletal reconstruction are limited by factors including accessibility, cell viability, autologous graft availability, infectious risks from cadaveric materials, and durability of synthetic substitutes.[2] Therefore, the concept of using tissue engineering methods to regenerate bone is appealing, as by manipulating cells, scaffolds, and stimuli, bone tissue will be generated and integrated with the native tissues to restore tissue and lost function.[3]

Bone morphogenetic proteins

Bone morphogenetic proteins (BMPs) were introduced to clinical and research applications by Dr. Marshall Urist's pioneering work on bone regeneration.[4,5] Later, Dr. Hari Reddi proposed that BMPs are responsible for the initiation cascade of developmental events, in which progenitor cells are induced to differentiate into bone cells thus resulting in new bone formation.[6,7] In the following years, recombinant human BMPs (rhBMPs) entered the clinical fields of spinal fusion, fracture healing, and engineering of dental tissues.[8,9]

The BMP is a growth factor family comprised of over 20 distinct ligands; it plays an important role in bone formation and remodeling, and in development and regeneration after tissue damage.[10,11] BMPs are highly potent growth factors, and the therapeutic outcome following the use of rhBMP-2 is critically dependent upon the delivery vehicle, quantity, concentration, and time of application.[12,13] Importantly, the use of rhBMP-2 without a carrier is contraindicated. Due to its high potency, increasing the dose of rhBMP-2 beyond a certain threshold concentration not only does not improve bone quality, but may promote lower quality bone and exuberant bone reaction, and may invoke a detrimental inflammatory response.[14]

Materials

Internal fixation

A detailed description of principles of internal fixation can be found in Chapter 33. A single 2.4-mm titanium locking reconstruction plate (Synthes Maxillofacial, Paoli, PA) is used for medium and large breed dogs. For small breed dogs, a single 2.0-mm locking titanium miniplate (Synthes Maxillofacial, Paoli, PA) is used. Ideally, at least three titanium locking screws should be used at each segment of the defect. However, in very small dogs, the authors have successfully used two locking screws in each bone segment.

rhBMP-2 and scaffold

A compression resistance matrix (CRM) or scaffold made of collagen with embedded granules of hydroxyapatite (HA) and tricalcium phosphate (TCP) (MasterGraft Matrix; Medtronic Inc., Memphis, TN) is used as a carrier for the rhBMP-2.[15] The volume of the defect is measured in three dimensions and a sufficient amount of collagen sponge, enough to provide one-half to three-quarters of the mandibular height and a length 2 mm greater than the defect span, is measured. Ten to fifteen minutes prior to implantation, the CRM is infused with a 0.5-mg/mL solution of rhBMP-2 at a volume corresponding to 50% of the volume of the prepared scaffold. For example, for a scaffold that is 4 cm long, 1 cm wide, and 1 cm high ($4 \times 1 \times 1 cm^3$), the total defect volume is 4 cm^3; thus, 2 mL of the rhBMP-2 solution should be used to soak the CRM. The rhBMP-2 can be prepared in aliquots that are placed in syringes in advance of the surgery and frozen at –80°C. Prior to surgery, the frozen syringe is thawed at room temperature, and rhBMP-2 is infused to the scaffold in a sterile container such as a 50 mL conical centrifuge tube.

Diagnostic imaging

Prior to any regenerative surgery, a precise understanding of the anatomical configuration, bone quality, available bone for internal fixation placement, and size of the defect should be obtained. This means that thin transverse computed tomography (CT) images (i.e., 0.6 mm or smaller sections) should be obtained and reconstructed using a bone filter. Then the Digital Imaging and Communications in Medicine (DICOM) files should be used to create a three-dimensional (3D) image for surgical planning (see Chapter 6). The CT images can also be used for the fabrication of 3D-printed models, which enhance the surgical planning and can be used for contouring the plates prior to surgery. This in turn results in decreased surgery time and ensures that the appropriate plate is available for the surgery.

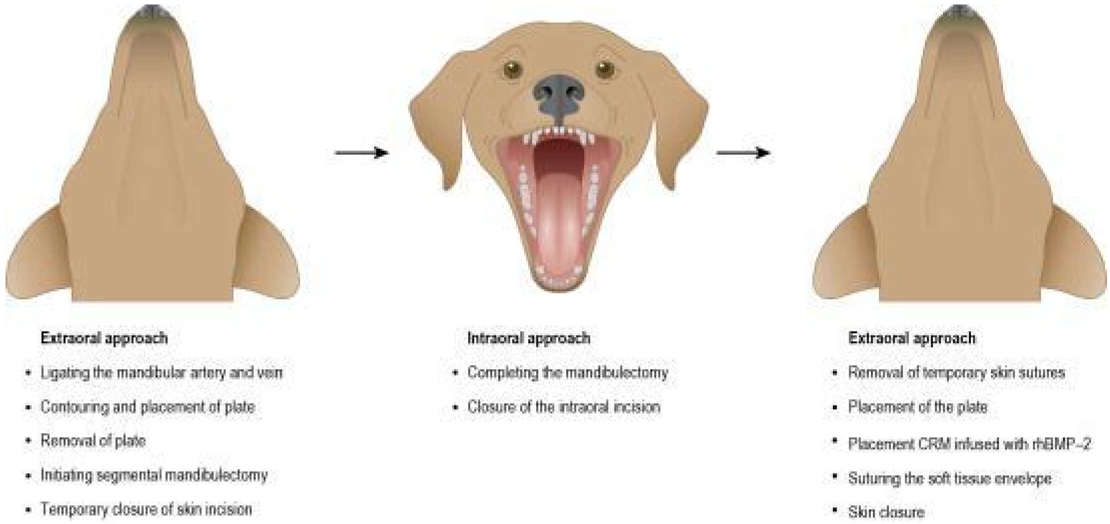
Surgical approach

A detailed step-by-step surgical approach to the mandible is described in Chapter 29. Internal fixation is placed from an extraoral approach as it provides superior access to the entire mandible as compared with an intraoral approach. In addition, an extraoral approach allows access to the inferior alveolar artery and vein in case ligature of these vessels is needed (i.e., to prevent or stop bleeding).

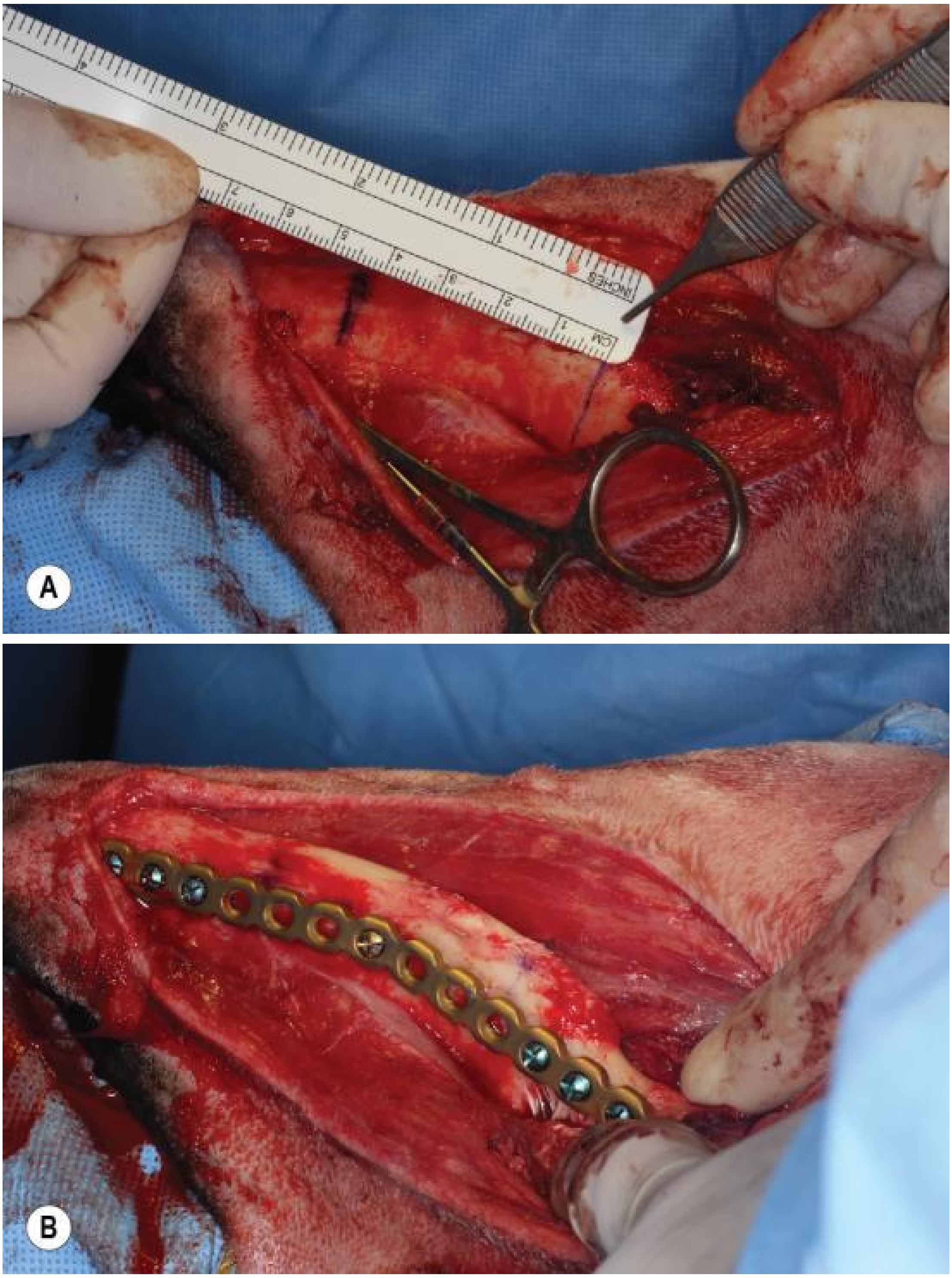
Surgical techniques

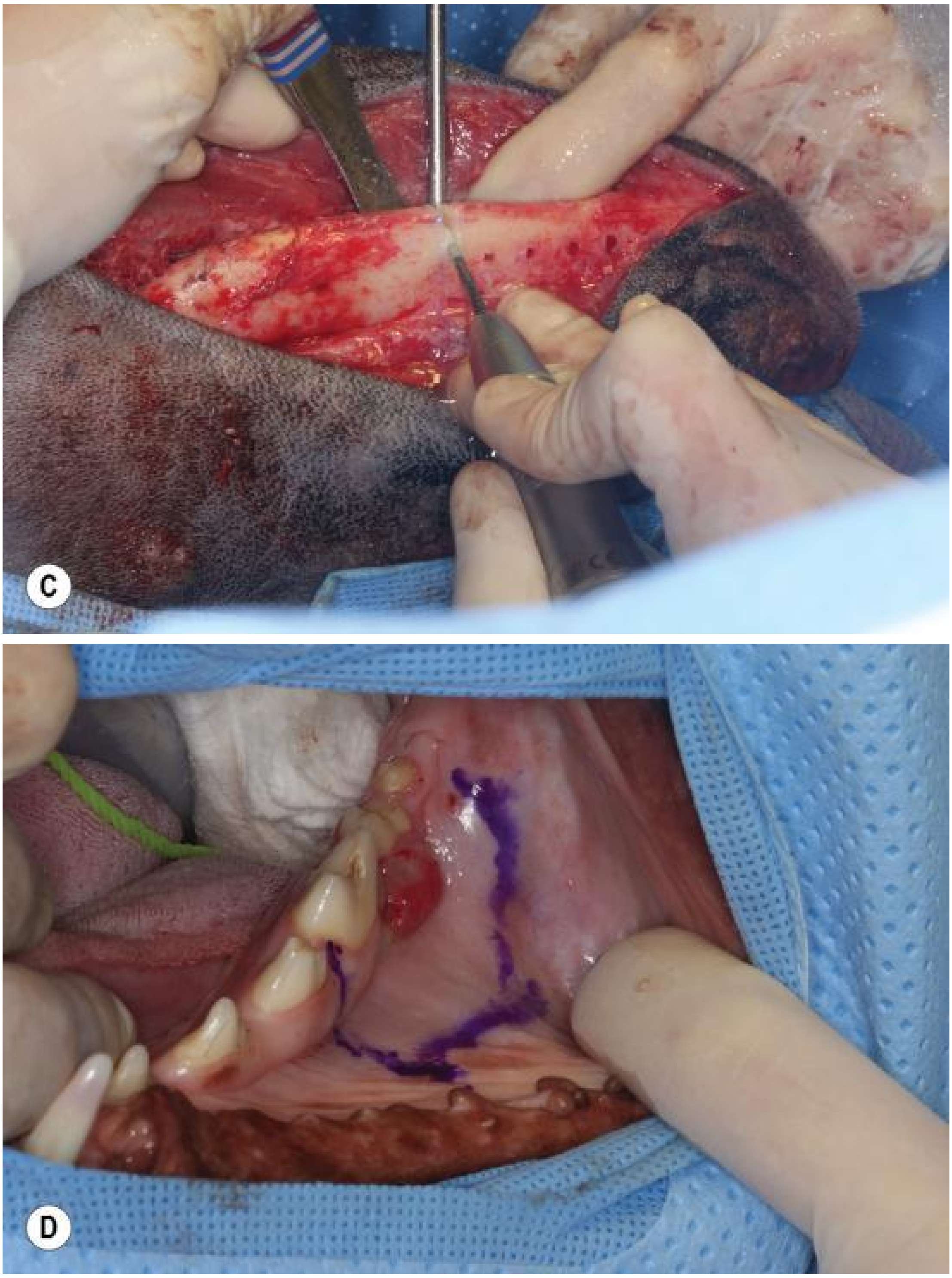
Reconstruction of segmental mandibulectomy[1,16]

A detailed description of segmental mandibulectomy technique can be found in Chapter 51. The mandible can be accessed via extraoral and intraoral approaches (Fig. 53.1). Beginning with an extraoral approach, the mandible is accessed and measured followed by marking of the excision area (Fig. 53.2A). Then, a single titanium 2.4-mm locking plate is contoured prior to mandibulectomy, capturing the normal anatomic contour of the mandible (Fig. 53.2B). The plate is secured to the ventrolateral aspect of the mandible with 3.0-mm titanium locking screws. The plate and screws are removed and the segmental mandibulectomy is initiated extraorally (Fig. 53.2C) with the patient in dorsal recumbency and completed intraorally with the patient in sternal recumbency (Fig. 53.2D). The intraoral part of the mandibulectomy is important for complete removal of the soft tissues and for better handling of tumors as compared with access via the extraoral approach. Excision of the mandible with the aim of appropriate surgical margins is completed, followed by intraoral closure (Fig. 53.2E). The dog is returned to dorsal recumbency, and the plate is returned and secured to the mandible via the extraoral approach. The surgical site is irrigated with sterile saline at this stage, as following CRM implantation, irrigation will no longer be possible (Fig. 53.2F). The rhBMP-2 CRM is implanted in the defect to fit snugly (Fig. 53.2G) and is secured circumferentially with 4-0 poliglecaprone 25 (Monocryl; Ethicon, Somerville, NJ) to prevent migration postimplantation. The surrounding soft tissues are sutured around the plate and CRM to provide a soft tissue envelope. The subcutaneous tissues and skin are closed routinely.



• FIG. 53.1 Reconstruction of segmental mandibulectomy – Diagram of flow of events: in the first stage, the mandible is accessed through an extraoral approach; in the second stage, an intraoral approach; and in the third stage, an extraoral approach. *CRM*, Compression resistant matrix; *rhBMP*, recombinant human bone morphogenetic protein.

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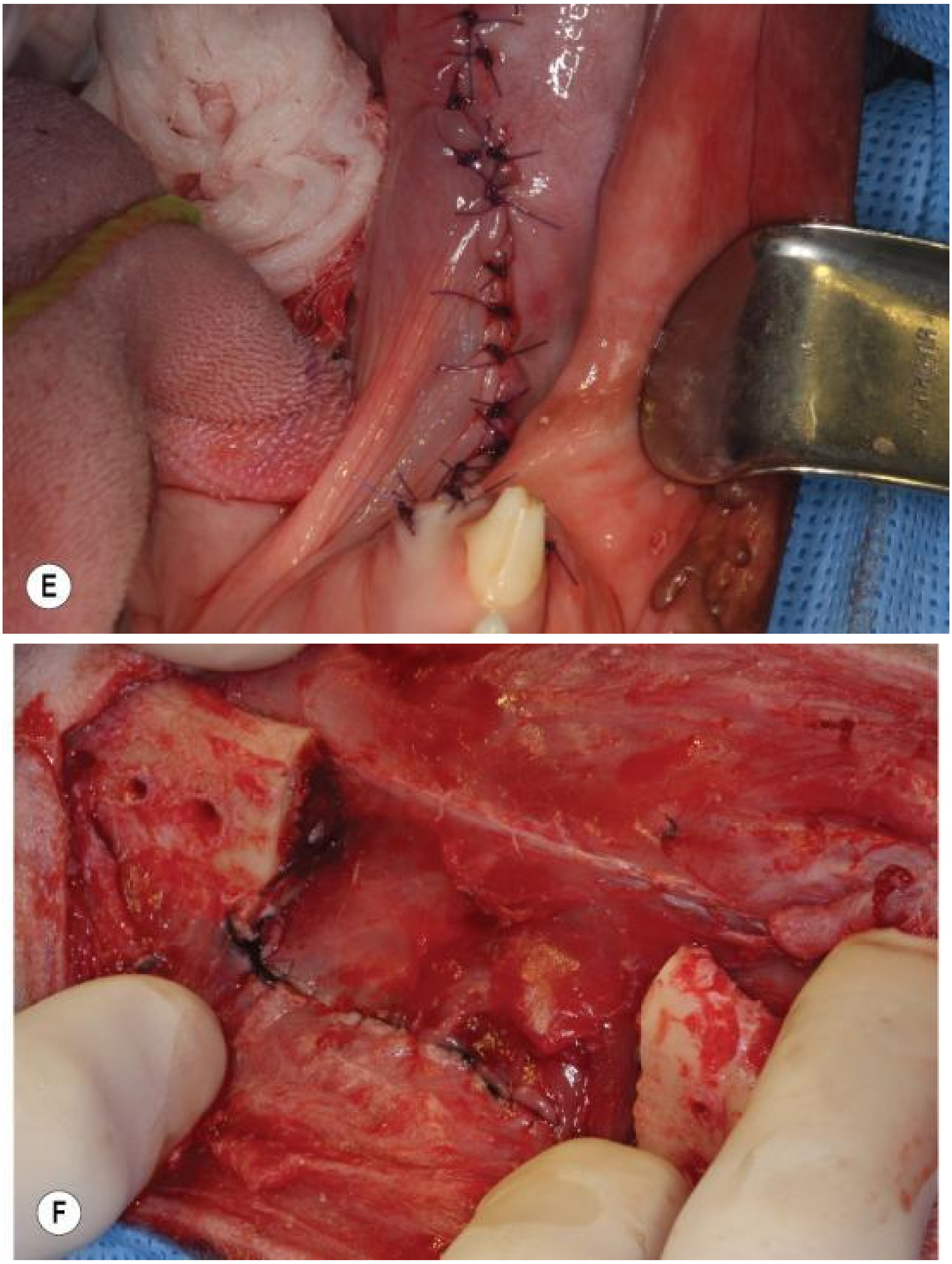
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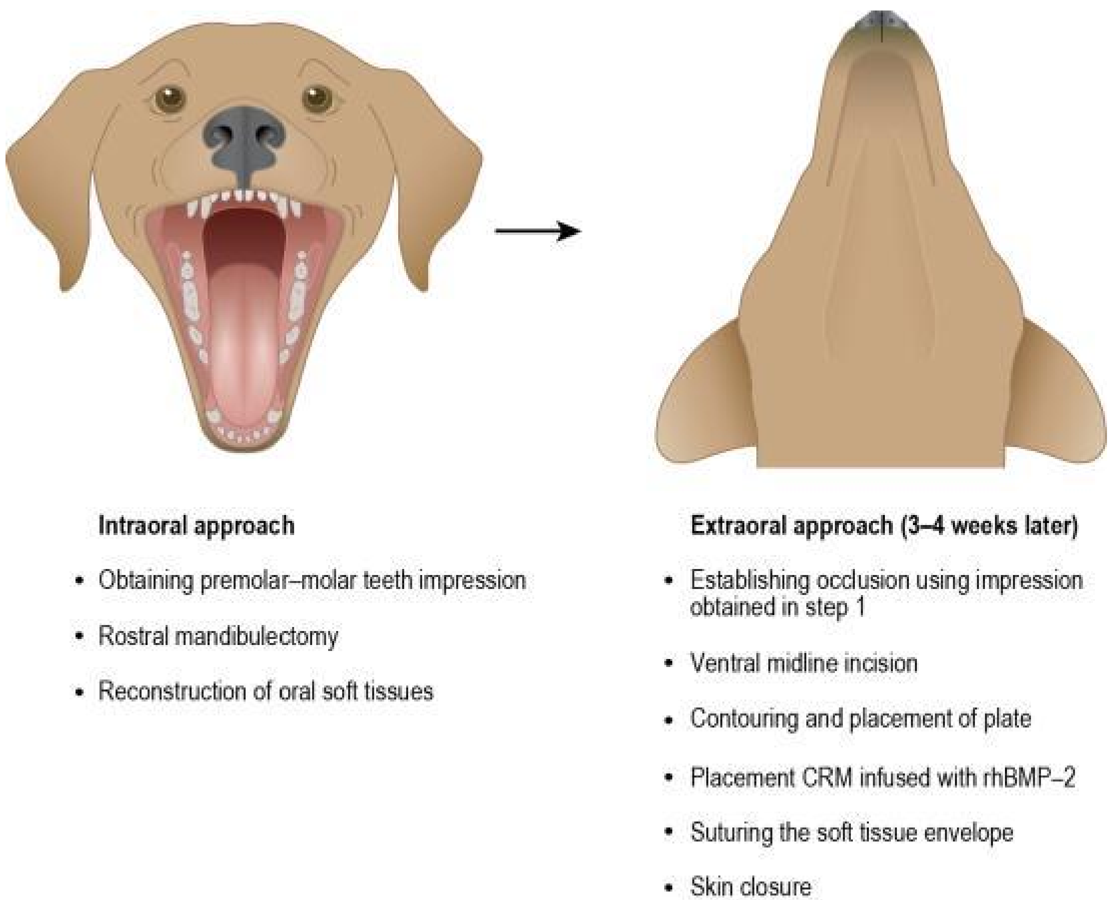
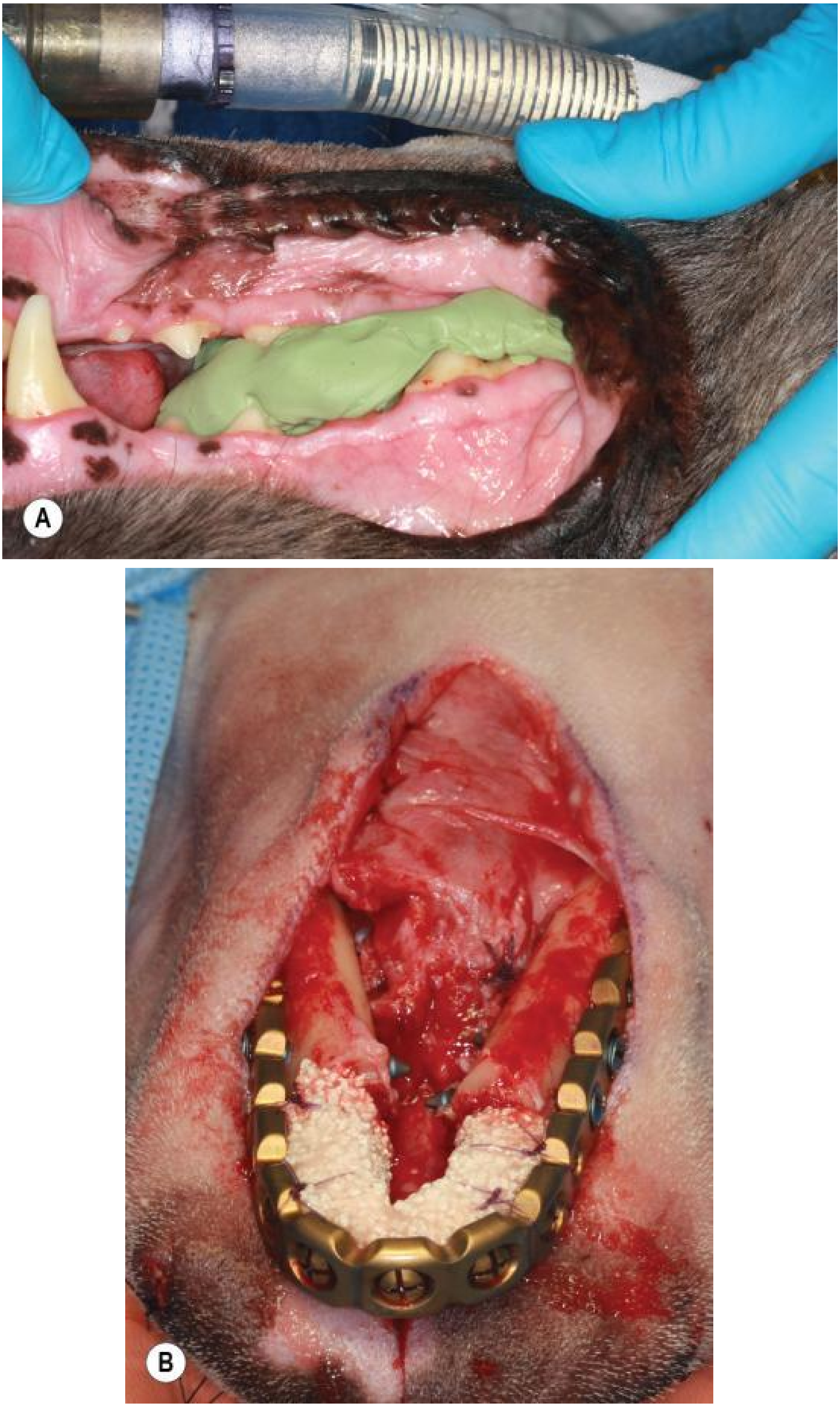
• **FIG. 53.2** (**A**) Following an extraoral approach to the mandibles, the area to be resected is measured and marked. (**B**) A single titanium locking plate is contoured prior to mandibulectomy, capturing the normal anatomic contour of the mandible; the plate is secured with screws and then removed. (**C**) Following the removal of the plate and screws, segmental mandibulectomy is initiated. (**D**) In the second stage of reconstruction of segmental mandibulectomy, an intraoral approach is utilized as it allows better handling of the soft tissues of the tumor as compared with the access via the extraoral approach. (**E**) Following excision of the mandible, intraoral closure in a single layer is performed. (**F**) The surgical site is irrigated with sterile saline and the defect site is ready for plate placement and compression resistant matrix (CRM) implantation. (**G**) The CRM infused with recombinant human bone morphogenetic protein (rhBMP-2) is implanted in the defect to fit snugly.

Reconstruction of rostral mandibulectomy[17]

In general, this procedure is performed in two stages, with the first stage being rostral mandibulectomy and the second stage, reconstruction, occurring 3–4 weeks later (Fig. 53.3). In order to maintain appropriate occlusion during the reconstruction phase, an impression of the caudal oral cavity, distal to the mandibular fourth premolar teeth and extending distally to include the third mandibular molar teeth, is obtained using vinyl polysiloxane impression material putty (Express STD; 3M ESPE, St. Paul, MN). The mandibular area is clipped and surgically prepared for aseptic surgery. A rostral mandibulectomy is performed with the dog in sternal recumbency as described in Chapter 51. Approximately 3–4 weeks following rostral mandibulectomy and after ensuring that the soft tissues have healed properly, the second stage, reconstruction of the rostral mandibles, is performed. Prior to surgery, pharyngotomy intubation is performed as described in Chapter 61. The dog is placed in dorsal recumbency and the previously obtained impression is placed in the mouth to recapture the normal occlusion of the remaining mandibles (Fig. 53.4A). An extraoral approach to both mandibles is made via a single midline incision. Following sharp and blunt dissection, the mandibles are exposed and the plate is contoured, adjusted, and adapted to the bone with bone forceps, then secured with appropriately sized titanium locking screws. Note that the plate can be contoured prior to surgery using a 3D printed model. This reduces surgical time and ensures that the appropriate size plate is available for the reconstruction. Prior to implantation of the rhBMP-2 infused scaffold, the surgical site is irrigated with sterile saline. The infused CRM is then implanted in the defect to fit snugly and is secured circumferentially with 4-0 poliglecaprone 25 (Monocryl; Ethicon, Somerville, NJ) to prevent migration postimplantation (Fig. 53.4B,C). The surrounding soft tissues are sutured around the plate and CRM to provide a soft tissue envelope. The subcutaneous tissues and skin are closed routinely.



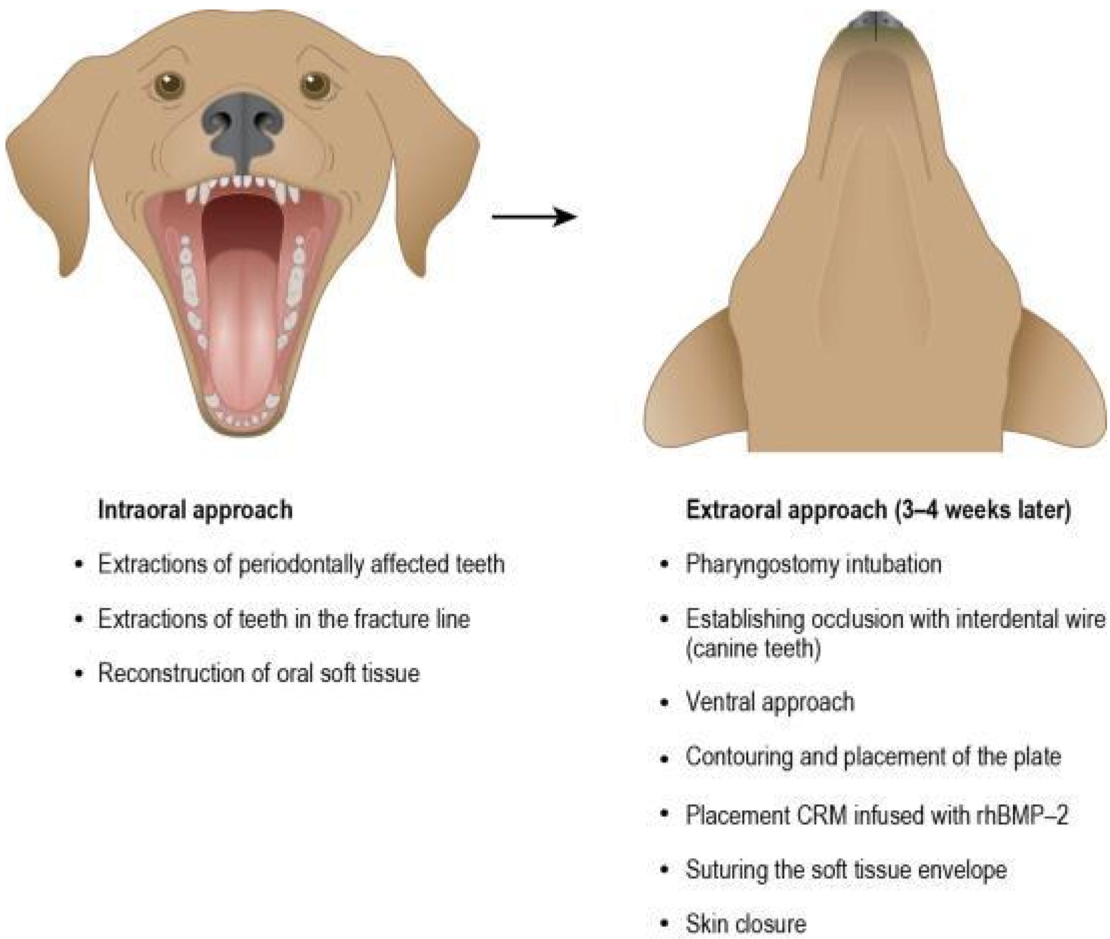
• **FIG. 53.3** Reconstruction of rostral mandibulectomy – Diagram of flow of events: this procedure is performed in two stages with the first stage being rostral mandibulectomy and the second stage, reconstruction, occurring 3–4 weeks later. *CRM*, Compression resistant matrix; *rhBMP*, recombinant human bone morphogenetic protein.



• **FIG. 53.4** (**A**) The polyvinylsiloxane impression that was obtained prior to the rostral mandibulectomy is now placed in the mouth to recapture the normal occlusion of the remaining mandibles. This will help achieve pre-mandibulectomy occlusion following reconstruction. (**B**) The infused compression resistant matrix is implanted in the bone defect at the rostral mandibles and secured circumferentially with 4-0 poliglecaprone 25 to prevent migration postimplantation. (**C**) Postoperative three-dimensional rendering of a computed tomography image of dog following rostral mandibular reconstruction.

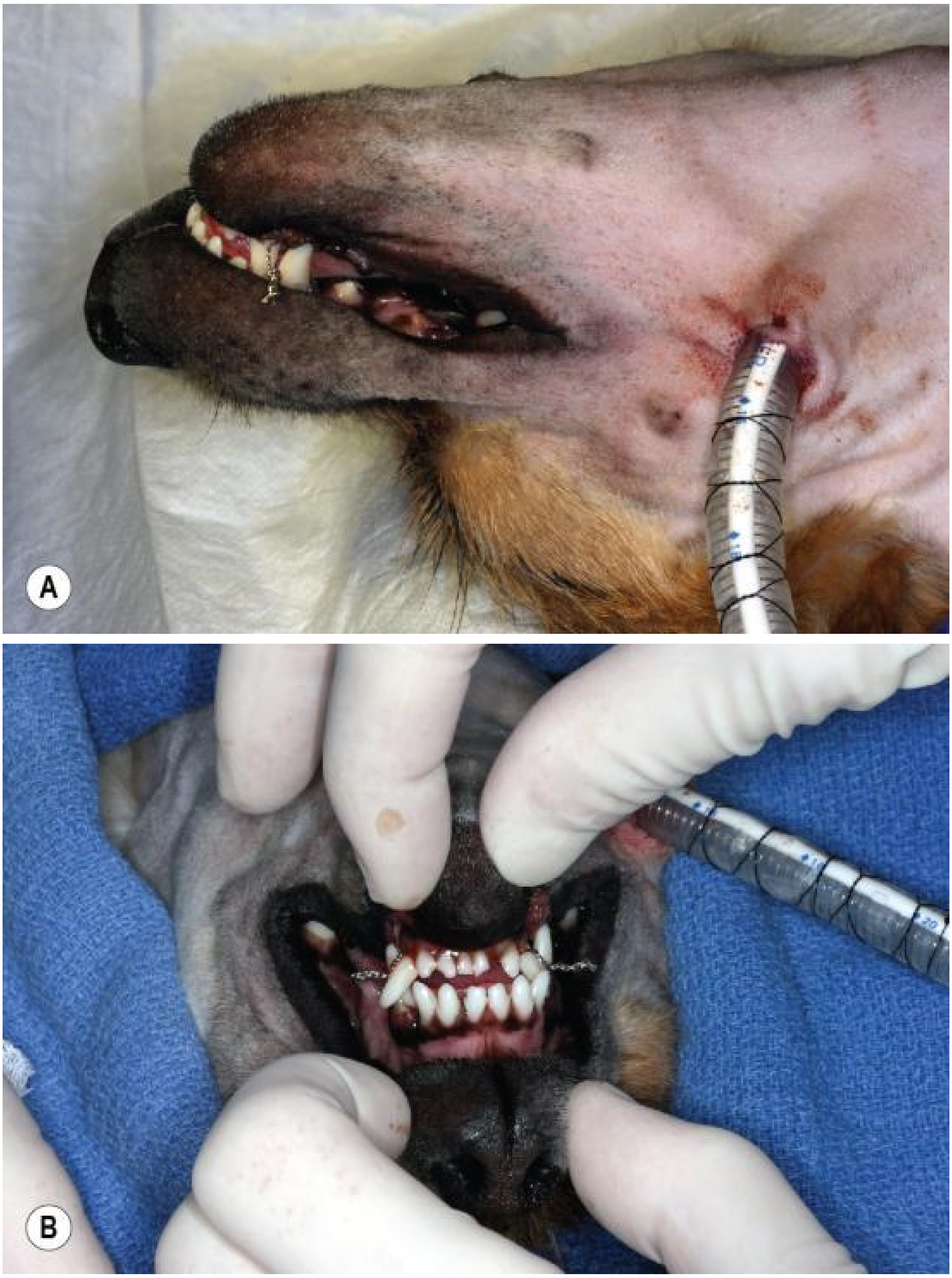
Reconstruction of defect nonunion fractures and delayed reconstruction of segmental mandibulectomy[18]

Prior to reconstruction, it is essential to perform thorough periodontal treatment and extractions of periodontally compromised teeth. The reconstruction phase takes place approximately 3–4 weeks later, after the soft tissues have healed from the dental extractions (Fig. 53.5).



• FIG. 53.5 Reconstruction of defect nonunion fractures – Diagram of flow of events: the procedure is typically performed in two stages. Prior to reconstruction, thorough periodontal treatment and extractions of periodontally compromised teeth is performed. The reconstruction phase takes place approximately 3–4 weeks later, once the soft tissues have healed from the dental extractions. *CRM*, Compression resistant matrix; *rhBMP*, recombinant human bone morphogenetic protein.

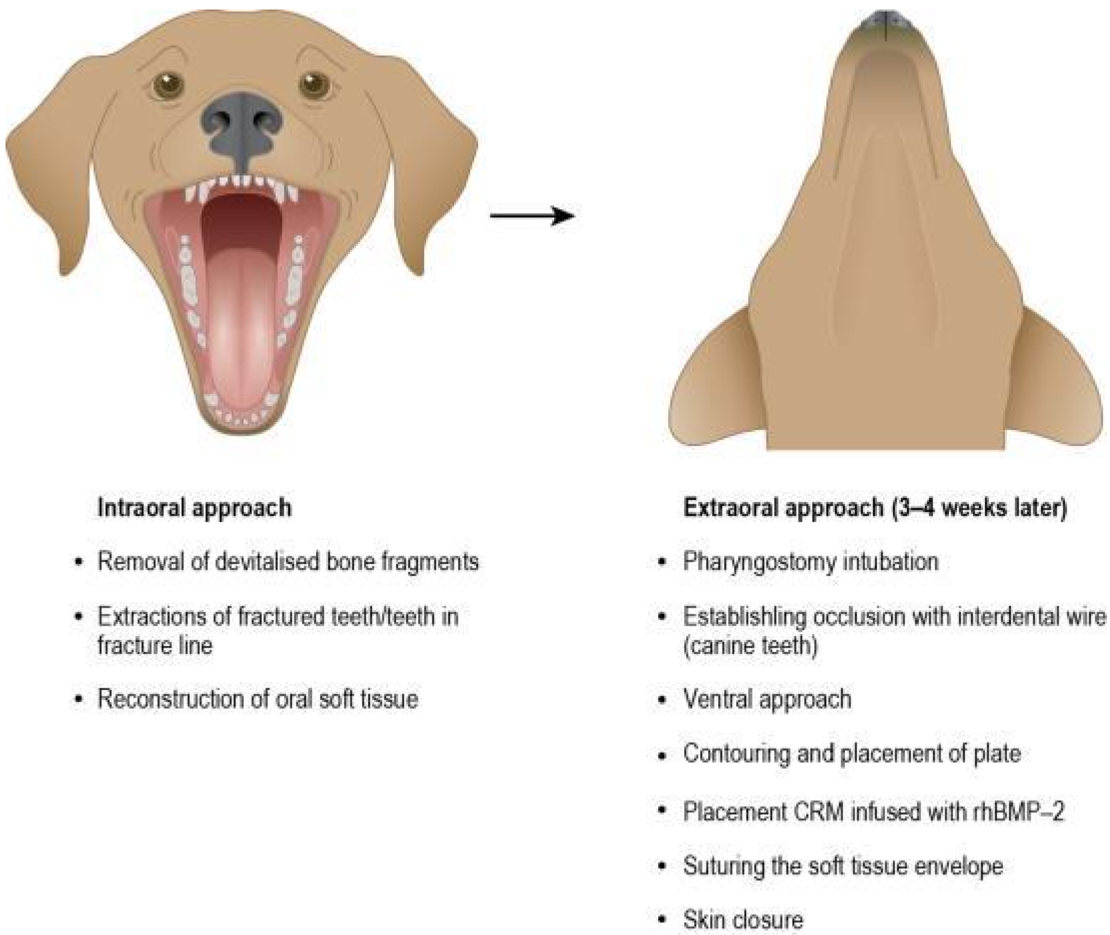
The dog is intubated via pharyngotomy as described in Chapter 61. The mandibles and maxillae should be brought into the desired closed-mouth occlusion, and the mandibular and maxillary canines temporarily wired together using 26- or 28-gauge wire (Fig. 53.6A,B) to secure the jaws. Using an extraoral approach, a single locking titanium 2.0-mm miniplate or 2.4-mm locking reconstruction plate is adapted to the desired anatomical contour of the mandible in a ventrolateral position while avoiding damage to the roots of any remaining teeth. The fracture edges should be debrided of scar tissue. The plate should then be secured to the bone with at least two (small dogs) or three (medium to large dogs) titanium locking screws in each segment of the defect. The surgical area is copiously irrigated with sterile saline. A CRM of appropriate size is cut and infused with rhBMP-2 as described above. The CRM is then implanted in the defect (Fig. 53.6C), and the surrounding soft tissues are sutured around the plate and CRM to provide a soft tissue envelope. The subcutaneous tissues and skin are closed routinely.


A
B

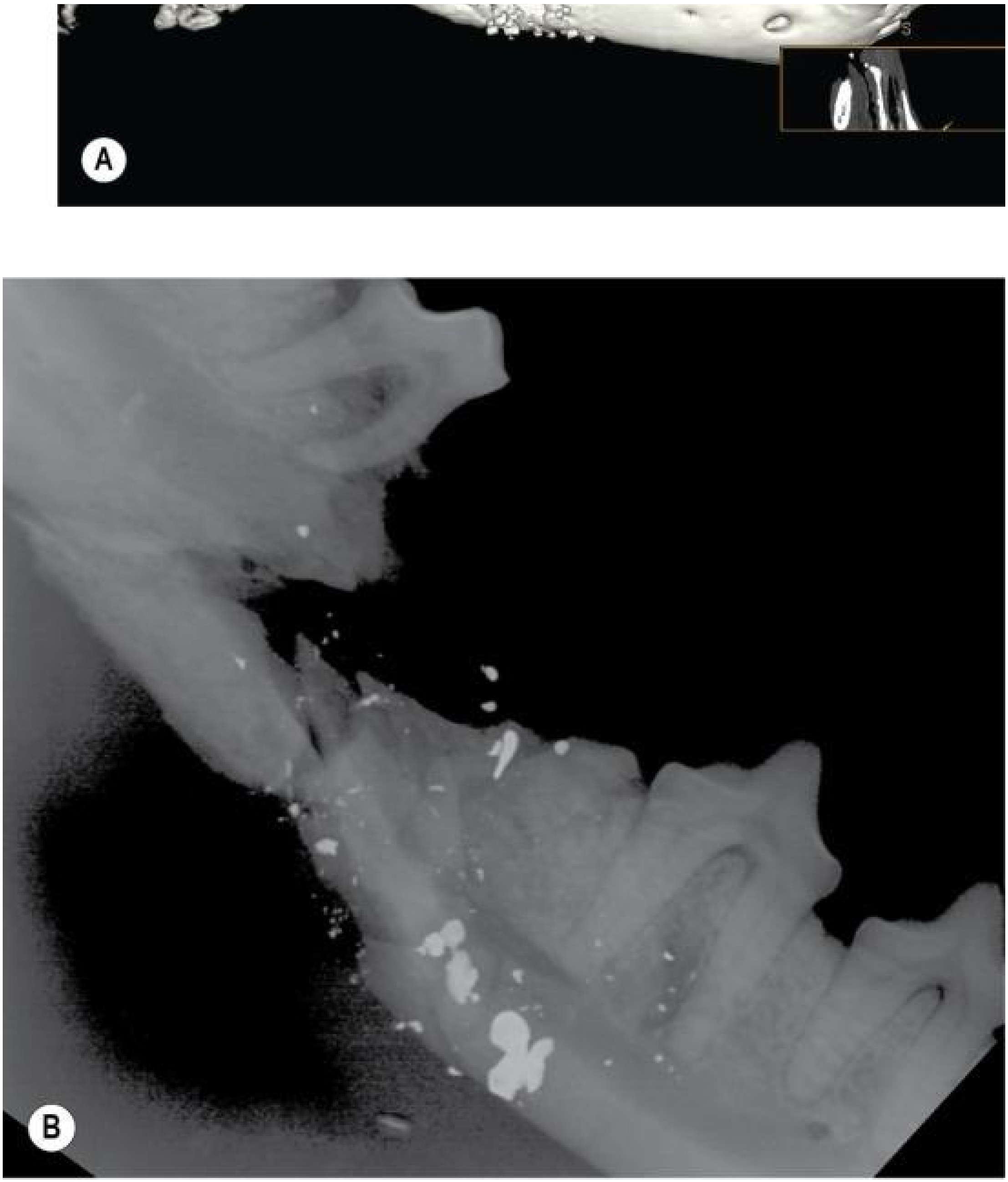
• FIG. 53.6 (**A and B**) This patient has been intubated via pharyngotomy, and (**B**) the mandibles and maxillae were brought into the desired closed-mouth occlusion and wired together using 26- or 28-gauge wire. (**C**) Reconstruction of a defect nonunion in a dog using a 2.0-mm titanium locking plate and compression resistant matrix infused with recombinant human bone morphogenetic protein (rhBMP-2).

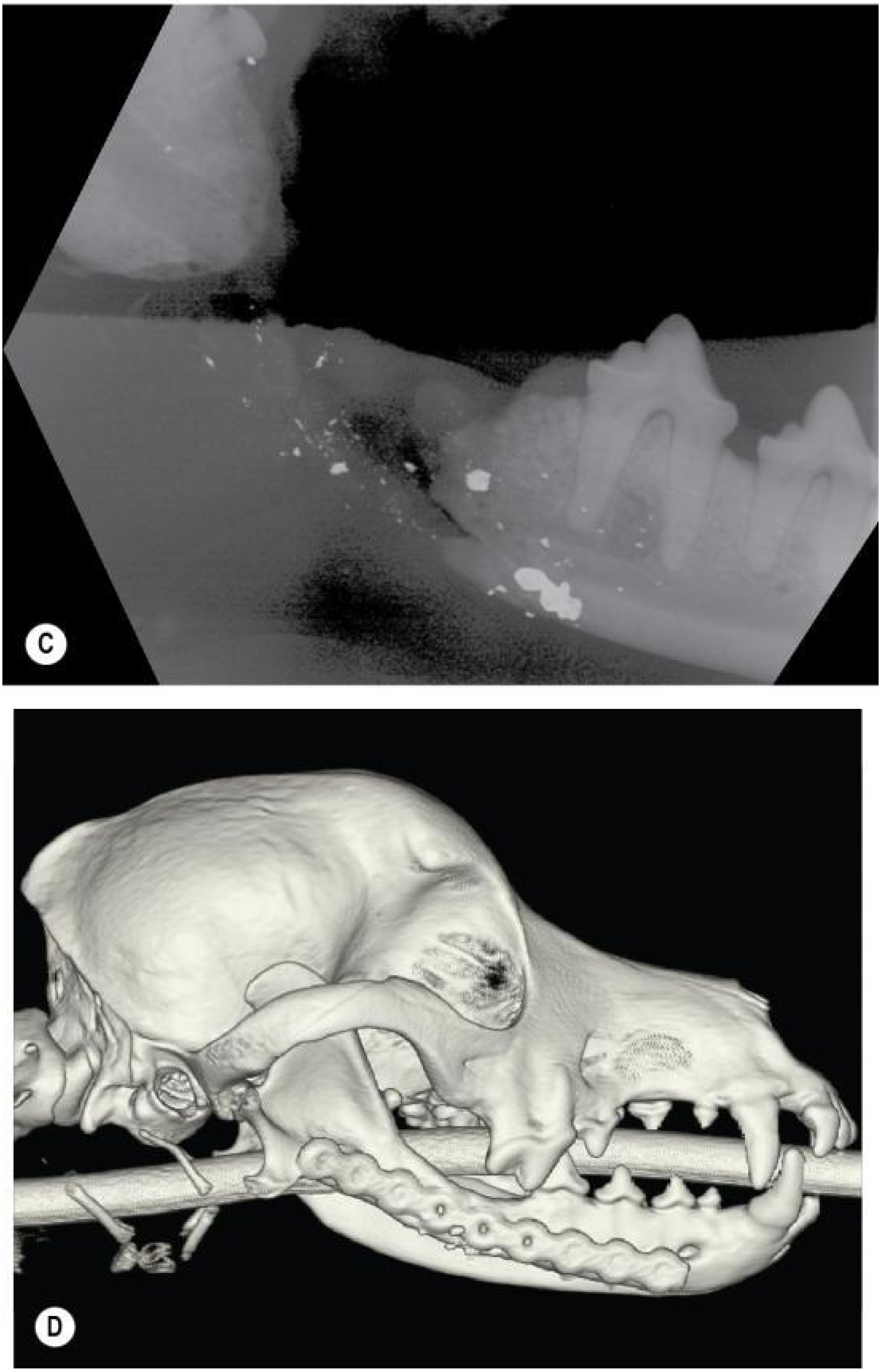
Reconstruction of gunshot-related fractures

Gunshot injuries to the head are likely to cause substantial fractures (Figs. 53.7, 53.8A,B). The first stage in the surgical management of these injuries is to remove devitalized tissues (i.e., bone fragments with no soft tissue attachments) and clean the wound to remove hair and other debris (Fig. 53.8C). Then, the oral mucosa is closed from an intraoral approach, and the skin and subcutaneous tissues are closed from an extraoral approach. The dog is placed in a muzzle (i.e., commercially available muzzle) for 3–4 weeks and returns for regenerative reconstructive surgery once the soft tissues have healed and no infection is present. The fractures are then managed as described in the previous section on reconstruction of defect nonunion fractures (Fig. 53.8D).



• FIG. 53.7 Reconstruction of gunshot-related fractures – Diagram of flow of events: the procedure is typically performed in two stages. In the first stage, devitalized bone fragments and debris are removed and the soft tissues sutured. The reconstruction phase takes place approximately 3–4 weeks later, once the soft tissues have healed. *CRM*, Compression resistant matrix; *rhBMP*, recombinant human bone morphogenetic protein.





• **FIG. 53.8** Three-dimensional (3D) rendering of a computed tomography (CT) image (**A**) and dental radiograph (**B**) of a severely comminuted right caudal mandibular fracture in a small breed dog following a gunshot injury. (**C**) A dental radiograph demonstrating the removal of bone fragments as the first stage in the surgical management of gunshot injury. (**D**) Six-month follow-up CT image in 3D, demonstrating a successful reconstruction of a mandible following gunshot injury in a dog.

Special considerations and complications

Intraoperative hemorrhage may occur during dissection and exposure of the mandible. It is recommended that prior to segmental mandibulectomy and reconstruction (i.e., prior to placing a plate in phase 1 of the procedure), the inferior alveolar artery and veins be ligated or hemoclips placed. In addition, careful and controlled ostectomy using a piezotome (see Chapter 9) will also reduce the likelihood and severity of hemorrhage.

To prevent complications associated with the use of rhBMP-2, precise handling of the proteins and calculation of the dosage is essential. Recombinant human BMP-2 is a very potent protein, and even minor discrepancies in calculations or in infusion of the CRM can result in exuberant bone formation. In addition, once the CRM is placed in the defect, a new surgical pack and new surgical gloves should be used for closure to avoid contaminating the surrounding soft tissues (i.e., touching with gloves or instruments) with rhBMP-2, which may result in ectopic bone formation.

Mild to moderate oozing from the incision sites may be observed for the first 1–3 weeks postoperatively. This typically resolves spontaneously. This is due to transient BMP-induced inflammation that begins on the 3rd day postoperatively, peaks at 1 week, and typically resolves 2–3 weeks postoperatively.[14,19,20]

Another possible complication is plate exposure through the alveolar mucosa.[21,22] This may occur if the plate is placed at or in close proximity to the alveolar margin. To avoid this complication, a single plate should be placed at the ventrolateral aspect of the mandibular border. This approach will also minimize iatrogenic damage to the teeth roots. If plate exposure has occurred, removal of the plate typically results in resolution of the problem, without negative long-term effects.[21,22]

Postoperative care

The patient should receive ampicillin 20 mg/kg IV preoperatively and amoxicillin/clavulanic acid 20 mg/kg orally (Clavamox; Pfizer Animal Health, New York, NY) twice daily for 2 weeks postoperatively. Postoperative care also includes multimodal analgesia (such as opioids and nonsteroidal antiinflammatory medication) for a period of 7–10 days. In addition, the patient should be offered soft food for the immediate postoperative period (i.e., first 7–10 days). However, hard food should gradually be offered after the first 10 days to encourage loading the mandible and encourage bone regeneration and remodeling. The skin sutures are removed 10–14 days postoperatively. The plate should not be removed unless a complication associated with the hardware occurs (e.g., infection, loosening of screws).

Prognosis

The prognosis associated with regenerative reconstructive surgery is good to excellent. Bone regeneration following the use of rhBMP-2 infused on a CRM is a reliable and reproducible technique, allowing a rapid and complete return to function. If exuberant bone reaction occurs, it should be monitored closely for temporomandibular joint involvement but otherwise, the bone remodels within 3–6 months and the exuberant bone reaction disappears.

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SECTION 10:

Salivary Gland Surgery

OUTLINE

CHAPTER 54

Principles of salivary gland surgery

Marijke E. Peeters

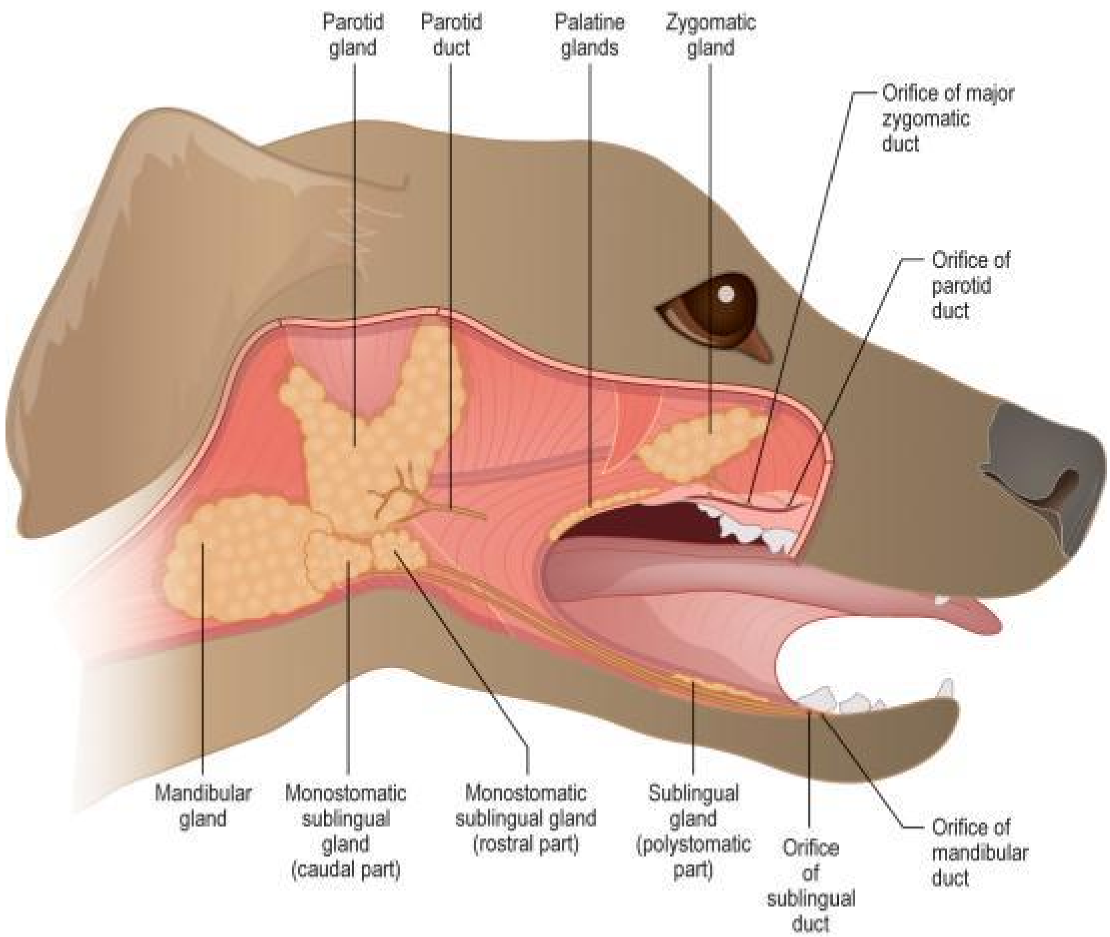
Definitions

Salivary fistula: An epithelialized, nonphysiological communication between a salivary duct and the skin, through which there is a persistent leakage of saliva.
Salivary mucocele: An accumulation of saliva in the subcutaneous or submucosal tissues caused by rupture or perforation of a salivary duct or gland.
Salivary stone (sialolith): A calculus occurring in a salivary duct or gland.
Sialography: A radiographic technique in which a radiopaque contrast agent is infused into the ductal system of a salivary gland prior to diagnostic imaging.

Surgical anatomy

Parotid gland

The V-shaped parotid gland consists of two diverging limbs on each side of the ventral part of the auricular cartilage (Fig. 54.1). Both limbs are connected by the ventrally directed apex.[1,2] The gland is covered by the parotidoauricularis muscle. The rostral border is related to the cranial auricular artery and vein, and the transverse facial artery. Branches of the intermediate auricular blood vessels encircle the caudal border. The deep part of the gland is related to the facial nerve and the maxillary and superficial temporal arteries. The linguofacial vein lies more ventral and superficial than the corresponding nerves and arteries. The parotid lymph node lies deep to the rostral border of the of the rostral limb of the superficial part of the parotid gland.[3]



• **FIG. 54.1** Topographical anatomy of the major salivary glands in the dog.

The parotid duct is formed by two or three smaller ducts, which leave the ventral third of the rostral border of the gland and join on the masseter muscle several millimeters from the gland. The duct runs parallel to the fibers of the masseter muscle and opens into the buccal cavity on a small parotid papilla above the distal aspect of the maxillary fourth premolar tooth. The parotid duct may be localized by cannulation of the papilla with stiff suture material. Accessory parotid glands may be present on one or both sides above the parotid duct. They empty with small ducts into the main parotid duct. The main blood supply of the parotid gland is the parotid artery that branches from the external carotid artery.

Zygomatic gland

The zygomatic gland lies against the ventral part of the periorbita, protected by the rostral part of the zygomatic arch.[1,2] It is the equivalent of the dorsal buccal glands of other mammals. One major and two to four minor ducts drain into the buccal cavity. The papilla of the major duct is located about 10 mm caudal to the parotid papilla dorsal to the first or second molar tooth. The minor ducts all open caudal to the major duct. Blood supply of the zygomatic gland is from the infraorbital artery. The zygomatic gland is related to the maxillary nerve and artery, which run dorsomedial to the gland.

Mandibular and sublingual glands

The mandibular gland is closely connected with the most caudal parts of the monostomatic sublingual gland by a common connective tissue capsule.[1,2] The mandibular gland is located between the linguofacial and maxillary veins, caudal to the angle of the jaw. In the cat the linguofacial vein crosses the gland on its lateral surface.[4] The dorsocranial part may be overlapped by part of the parotid gland. The mandibular lymph nodes lie rostromedial to the mandibular glands. The medial side of the mandibular gland is related to the sternomastoid muscle and tendon, the medial retropharyngeal lymph node and the larynx. More rostrally lie the stylohyoid and digastric muscles. The mandibular duct leaves the medial surface of the mandibular gland and runs medial to the sublingual gland in a cranial direction, closely related to the sublingual duct. Blood supply is from the glandular branch of the facial artery, entering the gland where the mandibular duct leaves it. The monostomatic part of the sublingual gland is related to the cranial part of the mandibular gland and runs rostrally, medial to the body of the mandible, between the masseter and the digastric muscle, reaching the lateral surface of the styloglossus muscle. Rostral to and connected with this part of the gland lies a cluster of lobules, directly under the mucosa of the mouth. These lobules empty into the main sublingual duct through four to six smaller ducts. The main sublingual duct runs with the mandibular duct between the styloglossus muscle medially and the mylohyoid muscle laterally in a rostromedial direction towards a small sublingual caruncle, lateral to the lingual frenulum. The ducts have one common opening or separate openings (in which case the mandibular opening is the most rostral one). The polystomatic sublingual gland consists of 6–12 separate lobules that empty directly into the oral cavity instead of emptying into the sublingual duct. The lobules lie submucosally on both sides of the body of the tongue, rostral to the lingual branch of the mandibular nerve. Blood supply of the monostomatic sublingual gland is from the glandular branch of the facial artery. The sublingual artery (a branch of the lingual artery) supplies the polystomatic sublingual gland.

Molar glands in the cat

The buccal molar gland in the cat is located between the orbicularis oris muscle and the mucous membrane of the lower lip at the angle of the mouth.[4] It empties into the buccal cavity through several small ducts. The lingual molar gland lies in the membranous molar pad just lingual to the mandibular first molar tooth. The gland has multiple small openings into the oral cavity.[5]

Other salivary glands

Other salivary glands are located in the submucosa and muscles of the tongue (lingual glands), the submucosa of the buccal cavity (minor buccal glands), the submucosa of the lips (labial glands), and the submucosa of the ventral side of the soft palate (palatine glands).[6]

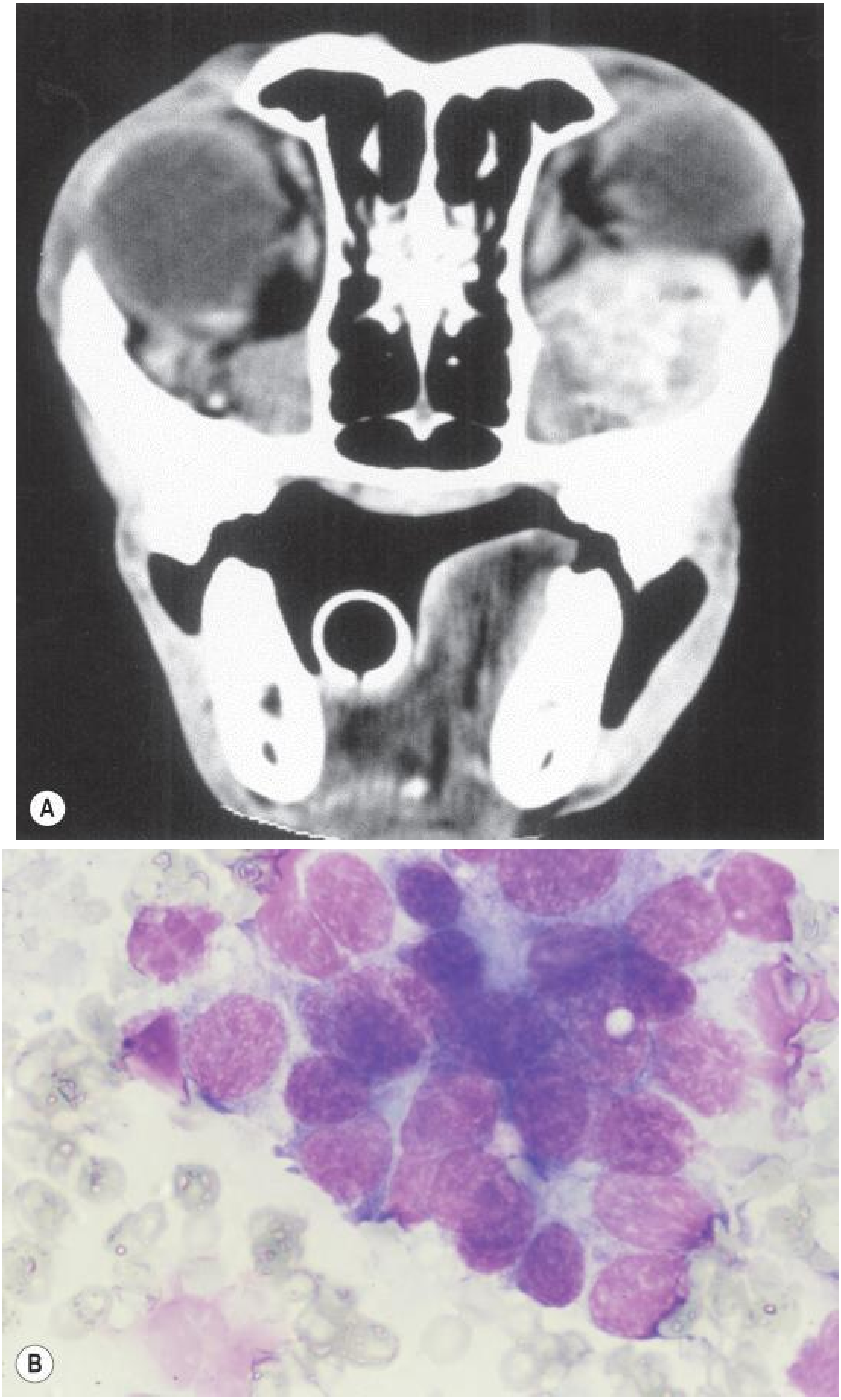
Examination techniques

Diagnostic imaging

Plain radiographs may be diagnostic if a radiopaque foreign body or a salivary calculus is present.[7] Contrast radiography (sialography) is performed under general anesthesia and may be helpful to locate salivary duct tears causing mucocele, to determine which glands should be removed surgically.[8] An iodine-based water-soluble dye (Télébrix 350; Laboratoire Guerbet, France) in a dosage of 1 mL/kg body weight is injected after cannulation of the duct with a 25- or

26-gauge catheter. For cannulation of the parotid duct, the mucosa caudal to the parotid papilla is grasped and pulled rostromedially to straighten the distal portion of the duct. The sublingual duct can only be cannulated when a separate opening is present just caudal to the larger opening of the mandibular duct on the sublingual caruncle.

Computed tomography (CT) and magnetic resonance imaging (MRI) may be useful in the investigation of the extent of salivary gland tumors to determine the feasibility of surgical resection (Fig. 54.2A). CT also shows the location and salivary gland of origin of sialoceles, even if small. Ultrasound is helpful in the differentiation of cystic lesions from inflammation or tumors and as guidance during biopsy procedures.



• **FIG. 54.2** (**A**) Computed tomography image of an adenocarcinoma of the zygomatic salivary gland with craniolateral displacement of the eye in an 11-year-old dog. Source: (Courtesy of Dr. S.A.E.B. Boroffka, Utrecht University, Netherlands.) (**B**) Fine needle aspirate demonstrating the neoplastic cells (May-Grünwald-Giemsa stain; 1000 ×). (Courtesy of Dr. E. Teske, Utrecht University, Netherlands.)

Salivary gland biopsy

Diseased salivary glands can be biopsied to determine the presence or absence of saliva, inflammation, or neoplastic cells (Fig. 54.2B). Indications for obtaining a biopsy include enlarged, painful, or cystic salivary glands.[9–12] Fine needle aspiration (FNA) biopsies are minimally invasive and generally do not require local or general anesthesia. Ultrasound may be helpful to obtain a representative sample of the abnormality. Saliva or cell types of the FNA biopsy can be identified by cytologic examination. Histologic characteristics and invasive growth can only be examined in larger tissue samples obtained by Tru-Cut, punch, or surgical (incisional) biopsy (Tru-Cut biopsy needle; Travenol Laboratories, Inc., Deerfield, IL; Baker Cummons punch biopsy; Key Pharmaceuticals, Inc., Miami, FL). Local or general anesthesia is required. With a Tru-Cut biopsy, a piece of tissue 1–2 mm in width and 10–15 mm long is obtained with a 14- to 18-gauge needle. A closed or open (after incision of the skin) method is used. Punch biopsies will provide a composite of normal and abnormal tissue but are limited in depth. Incisional biopsies are used when less invasive techniques are not diagnostic. After incising the skin, the gland is localized. The capsule, if present, is incised. Unless there is an obvious lesion visible, a lobule of salivary gland tissue is isolated and its duct ligated prior to excision. The surrounding salivary tissue should be handled atraumatically to prevent extravasation of saliva. Care should be taken in removing the biopsy tract during curative resection of malignancies.

Surgical diseases of the salivary glands

Salivary gland and duct injury

Most injuries of the salivary glands and ducts are caused by sharp or blunt trauma to the head or by surgery, and may eventually result in salivary mucocele (see Chapter 55) or salivary fistula. Bite wounds or trauma that is caused by stick penetration injury may result in infected mucoceles that are difficult to differentiate from ordinary abscesses. Injuries resulting in stenosis or blockage of a salivary duct will not cause visible long-term complications.[13]

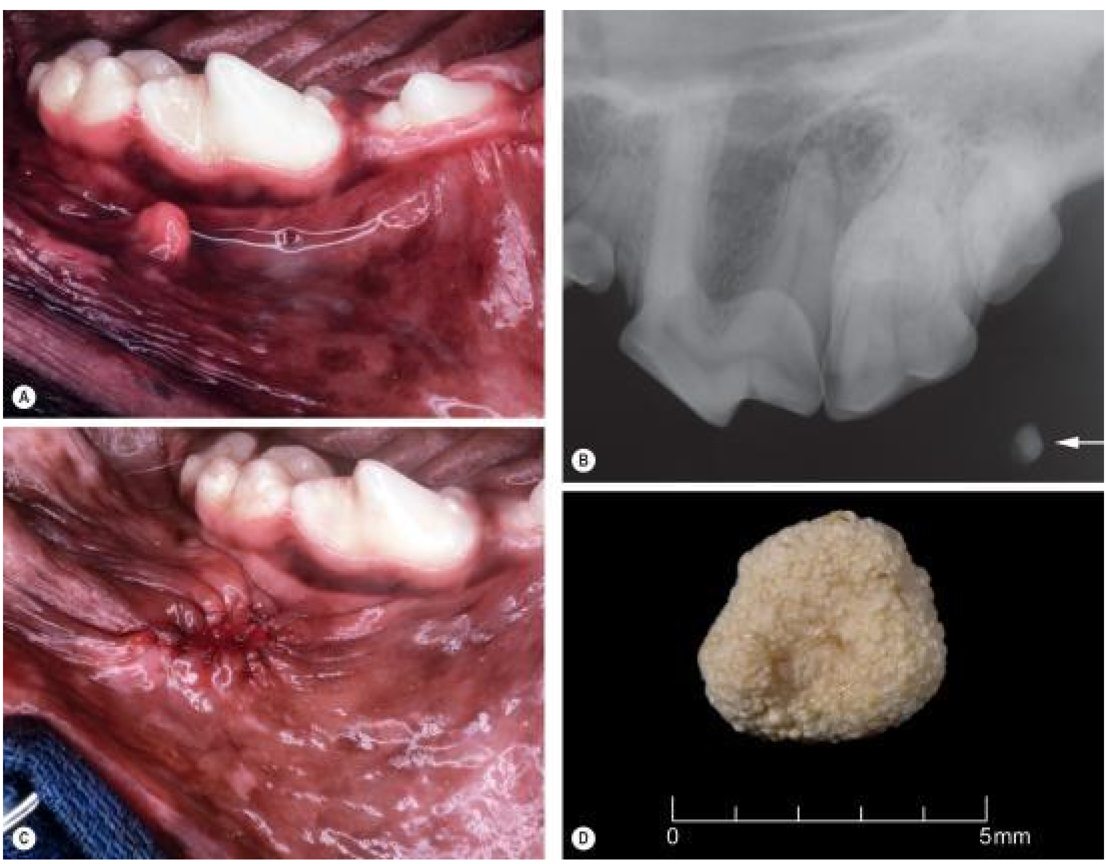
Microsurgical reconstruction of a lacerated duct may be performed with or without stenting. In an experimental study on salivary duct surgery in the dog, it was concluded that a massive scarring process postoperatively interferes with uncomplicated healing. Long-term stenting (14 days or more) was found to increase the success rate of salivary duct surgery.[14]

Salivary fistula

Salivary fistula of the parotid gland may be caused by severing of the parotid duct. The saliva accumulates in the subcutaneous tissues, forming a mucocele, or it finds a new way out through a nonphysiologic opening in the skin, resulting in a permanent fistula.[15] If primary anastomosis or oral reimplantation of the duct is not feasible, ligation of the parotid duct “upstream” is the easiest way to solve the problem.[13,14] The rupture can be localized by sialography.[8] The "upstream" part of the duct is cannulated with a 00 tear duct cannula (Shepard tear duct cannula; Eickemeyer, Hamburg, Germany) or a monofilament nylon suture. The duct is exposed through a lateral skin incision between the buccal nerves on the masseter muscle. The duct is undermined, the cannula or suture is withdrawn, and the duct is double-ligated with monofilament nonabsorbable suture material.[13]

Sialoliths

Sialoliths are calcifications found in salivary glands or ducts (Fig. 54.3).[7,11,15–17] In the dog, they typically occur in the parotid duct and are occasionally caused by an ascending foreign body.[18] Sialoliths may also form within the walls of longstanding mucoceles and in inactive salivary glands.[17,18] Obstruction of a duct may cause a painful swelling of the gland and eventual rupture of the duct.[7] Sialoliths do not cause salivary mucoceles. The diagnosis can be made by plain radiographs or sialography. A sialolith within a duct is removed by incising the duct over the stone through the oral mucosa. By flushing the duct, small remnants of the obstruction are removed. The incision is not sutured.[19]



• **FIG. 54.3** A 6-year-old Labrador retriever presented with a history of recurrent left facial swelling; on examination, the left parotid duct was palpably enlarged. (**A**) A hyperplastic left parotid papilla, caused by a sialolith lodged at the papilla. (The dog is in dorsal recumbency.) (**B**) Radiograph showing the sialolith *(arrow)*. (**C**) Following excision of the hyperplastic papilla and removal of the sialolith, a sialodochostomy was performed. (**D**) Sialolith following removal. Source: (Courtesy Dr. F.J.M. Verstraete, University of California, Davis, CA.)

Salivary gland neoplasia

Salivary gland tumors are rare in the dog and cat, with a reported incidence of 0.17%.[9] Adenocarcinomas of the larger salivary glands as well as carcinomas originating from the salivary tissues in the superficial submucosa of the oral cavity have been described.[10,11,20] Occasionally, malignant tumors of nonsalivary tissue origin occur within the salivary glands.[21–23] The presenting complaint in most cases is a mass being noted by the client, sometimes with halitosis and dysphagia. Tumors of the zygomatic gland may result in exophthalmos.[12] Metastases of malignant salivary neoplasia to lymph nodes, lung, liver, pancreas, heart, adrenal glands, diaphragm, body wall, and bone have been reported.[9,20,24–26] Benign tumors of the salivary glands are very rare in dogs and cats; lipomas and adenomas have been reported.[9,27] The prognosis of malignant salivary neoplasia, in general, is poor, but with early diagnosis and aggressive treatment of the disease with radical surgery and radiation, survival times of over 1 year are achievable in some patients.[12,28] Surgical excision of a salivary gland malignancy should always include biopsy of the draining lymph nodes (parotid, mandibular, and retropharyngeal lymph nodes).

Surgical excision of the mandibular gland

Surgical excision of the mandibular gland is performed through a skin incision caudal to the mandible, avoiding the maxillary and linguofacial veins (see Chapter 55). After incising the platysma muscle, the fibrous tissue of the mandibular gland capsule is identified. Unlike during excision of this gland for the treatment of mucocele, the capsule preferably is not opened if the gland contains malignant neoplasia. Dissection is continued in a rostral direction, where a ligature is placed on normal sublingual gland tissue before removing the neoplastic mandibular gland.

Surgical excision of the parotid gland

Surgical excision of a parotid gland with malignant neoplasia is a surgical challenge because of the diffuse nature of the gland and the presence of the facial nerve. The parotidoauricularis muscle is separated from its vertical ear canal attachment and retracted. The caudal auricular vein is ligated and divided. The parotid gland is dissected beginning at its dorsocaudal angle. Small vessels are ligated or electrocoagulated. Ventrally, the parotid gland is separated from the mandibular salivary gland and the vertical ear canal. The facial nerve, located at the beginning of the horizontal ear canal, is avoided during dissection.

Surgical excision of the zygomatic gland

Surgical excision of the zygomatic gland can be performed using a lateral or a dorsal approach.[29–31] The lateral approach is more commonly used. For the lateral approach the skin over the dorsal rim of the zygomatic arch is incised. The palpebral fascia, retractor anguli oculi lateralis muscle, and orbital ligament are incised at the periosteum and reflected dorsally. The dorsal rim of the zygomatic arch is removed with bone rongeurs or, ideally, a piezoelectric ultrasonic surgical unit (see Chapter 9). The zygomatic gland can be removed by gentle blunt dissection. The zygomatic branch of the buccal artery is ligated.

The dorsal approach is performed through a skin incision over the frontal bone margin of the periorbita. After incising the palpebral fascia at its bone attachment, the orbital ligament can be divided if additional exposure is needed. The tissues are reflected ventrally and the affected gland and periorbital fat are removed by gentle blunt dissection. The lateral superior palpebral artery can be ligated. Temporary dysfunction of the upper eyelid after surgery can be managed with artificial tears. MRI has been found to be helpful for surgical planning in zygomatic salivary gland disease in dogs.[32]

Salivary gland necrosis and sialadenosis

Salivary gland necrosis in dogs causes severe pain on swallowing and palpation, nausea, vomiting, and anorexia. The disease, which mainly affects the mandibular salivary gland, has been described under several different names, namely sialadenitis, salivary gland infarction, salivary gland necrosis, and necrotizing sialometaplasia.[33–37] Ischemic necrosis of the glandular tissues, inflammation, and regenerative hyperplasia of the ductal epithelium are described in most cases. Different etiologic agents have been proposed as the cause of this disease: viral infections, fungi, cervical trauma, limbic epilepsy, and lesions of the esophagus.[11,31–35,38,39] Surgical excision of the affected salivary glands alone does not resolve the clinical signs in most cases.[35,36] Some patients responded well to adjuvant therapy with anticonvulsants.[31,35,37,40] In dogs that were treated successfully for esophageal lesions caused by *Spirocerca lupi*, the salivary signs disappeared.[39]

Sialadenosis has been defined as a condition with bilateral nonpainful enlarged mandibular or zygomatic salivary glands without substantial microscopic lesions. Additional clinical signs consist of retching and gulping. Treatment with phenobarbital results in a rapid response in most cases.[41]

Complications

Postoperative complications after surgery of the salivary glands or ducts may include dysfunction caused by laceration of nerves, mucocele, seroma, and infection. Mucoceles are treated by the complete resection of the affected gland or by marsupialization (see Chapter 55). Persistent seromas may require drainage. Infection is treated with drainage and antibiotics. Complications are prevented if close attention is paid to the anatomy and aseptic technique. The use of a surgical loupe is recommended.

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CHAPTER 55

Surgical treatment of sialoceles

J. Geoffrey Lane

Definitions

Sialocele: An accumulation of saliva that develops within the fascial planes of the throat, the orbit, the oral cavity, or the parotid region after rupture of one of the glands or ducts conducting secretions from a salivary gland to the oral cavity.[1-4] After neoplasia, sialoceles represent the most frequently encountered disorder afflicting the salivary glands of the dog and cat, in which species traumatic ruptures and glandular inflammation are less common. The ultrastructure of the salivary glands of the dog and cat shows them to be “mixed,” meaning that the secretions they produce are partly mucous and partly serous. Thus, the term *salivary mucocele* is not strictly appropriate and will not be used here. The term *salivary cyst* is also occasionally used but this implies that there is a fluid collection enclosed by an epithelial lining, which is not typically the case in animals.

Ranula: A specific presentation of sialocele wherein the leaked saliva gathers at the ventrolateral aspect of the tongue and is simply contained within a layer of oral mucosa. The term derives its name from those species of frog (genus *Rana*) that balloon the sublingual structures intermittently.

Pathogenesis and epidemiology

On the basis of sialographic studies, it has been concluded that the majority of sialoceles in both dogs and cats arise through disruption of one of the small tributary ducts of the monostomatic section of the sublingual salivary gland.[4] The cause of the breach in the wall of these ducts is far from certain. Although it is widely proposed that trauma is responsible, this suggestion is not supported by convincing evidence. In fact, the site where the ductules usually leak lies in a position that is relatively well protected by the mandibles. In addition, experimental studies performed in an attempt to reproduce sialoceles by ligation of the mandibular and sublingual ducts rostrally or caudally, as well as insulting the ducts directly, were unsuccessful in creating sialoceles.[5]

The escaping saliva gathers between the fascial planes and, because it is mildly irritant due to the digestive enzymes it contains, provokes the surrounding connective tissue to form a thin inflammatory capsule—a pseudocystic wall. It is important to appreciate that this capsule is nonsecretory and therefore does not contribute in any way to the contents of the sialocele. Thus surgical removal of the lining will not be helpful to resolve the condition. The logical solution to sialoceles would be to identify and repair the defect in the ductule, but this is impracticable. In practice, extirpation of the glandular tissue that is supplying the saliva into the sialocele offers an effective remedy. This consists of resection of the mandibular gland as well as the sublingual chain (because both lie within a common capsule, it would be difficult to remove one without the other).

Although the etiology of sialoceles remains obscure, many earlier reviews of the condition reported that they arise predominantly in younger dogs.[2,6] However, in a study of 60 cases, no statistically significant age or sex predisposition was recorded,[7] and in a series of sialoceles reviewed by the author, only 78 of 166 dogs (47%) were under 4 years of age.[8] Standard poodles, Labrador retrievers, and dachshunds were overrepresented, and male dogs accounted for almost three-quarters of the group (118 dogs). Two standard poodle littermates and their dam were included in this series. Previous reviews of canine sialoceles have suggested a predisposition in poodle types,[2,6,7] German shepherd dogs,[3,4] and greyhounds.[4] However, most of these earlier case series have been too small for valid statistical analysis. Bilateral development of sialoceles does occur, with a reported frequency ranging from 6.25%[8] to 16%[4] of cases. In one instance a standard poodle developed a right-sided sialocele 4 years after a similar lesion had been treated on the left.[8]

Saliva leaking from the sublingual glands tends to become trapped at one or more of three sites. In order of frequency, these are the ventral aspect of the throat (cervical site) (Fig. 55.1), beneath the sublingual mucosa (ranula) (Fig. 55.2), and in the peritonsillar submucosa (pharyngeal site) (Fig. 55.3) (Table 55.1).[8,9] No correlation has been reported between the site of the defect in the glandular system and the site at which the sialocele forms. However, it seems likely that ranulae develop when the leakage occurs from the more rostral lobules of the sublingual gland, and the surgical remedy should take account of this possibility. In extreme cases, cervical sialoceles may appear as unsightly pouches with loss of hair and thinning of the overlying skin (Fig. 55.4). Nevertheless, spontaneous ulceration of the skin or rupture of the sialocele wall is highly improbable. It is also unusual for overt trauma (such as an oropharyngeal injury due to stick penetration) to rupture the sublingual or mandibular duct and to provoke the formation of a sialocele, although one published case series included five dogs with a reliable history of pharyngeal stick penetration in the period immediately preceding development of a sialocele.[8] The distension of a ranula may produce an obvious deviation of the tongue, and the patient may find it difficult to close the mouth because the teeth tend to occlude onto the sialocele. A common feature of sialoceles arising in the pharyngeal position is that they provoke dyspnea, as the expanding lesions tend to obstruct the airway.[9] This was the case in all eight dogs presented with a sialocele in this location in the author's series.[8] On superficial inspection, lesions at this site might resemble a tonsillar mass.

• **FIG. 55.1** Cervical sialocele in a 2-year-old dog.

• **FIG. 55.2** Sublingual sialocele (ranula) in a 3-year-old cat.

• **FIG. 55.3** Pharyngeal sialocele in a 2-year-old Labrador retriever that was presented with dyspnea and an incorrect diagnosis of tonsillar neoplasia.

• FIG. 55.4 Long-standing cervical sialocele with hair loss and thinning of overlying skin in an Old English sheepdog.

TABLE 55.1

Site of Sialoceles in 166 Dogs. [8]

Site	Number
Cervical	115
Bilateral cervical	9
Sublingual	19
Bilateral sublingual	1
Pharyngeal	5
Cervical and sublingual	14
Cervical and pharyngeal	2
Sublingual and pharyngeal	1

Less frequently, sialoceles arise through leakage of saliva from the ducts of the parotid or zygomatic glands,[10-14] and trauma to the duct system is most commonly responsible. Trauma to the parotid duct or inflammatory reactions adjacent to it may cause stenosis, and initially there will be a palpable distension over the cheek region where the duct dilates. However, there will be a tendency for the glandular tissue to atrophy as pressure increases within the distension, except in the presence of low-grade infection, which stimulates the continued production of saliva. Obstructions of the parotid duct by sialoliths, foreign bodies, and neoplasia have been documented as causes of parotid sialoceles, but it is unclear whether these consisted of grossly dilated parotid ducts or whether there had indeed been leakage of saliva into the adjacent tissue planes.[10,12] The parotid duct lies relatively superficially as it passes over the face of the masseter muscle and is more vulnerable to rupture by an incisive wound than the other major ducts; under these conditions a fistula may develop, with a discharge of saliva directly to the skin surface. Rarely, a sialocele develops from the zygomatic gland to cause orbital swelling or proptosis (Fig. 55.5), and this must be differentiated from a retrobulbar abscess or orbital tumor: a fluctuant swelling may be present beneath the oral mucosa caudal to the last molar tooth. Ultrasonography of the orbit will help make a definitive diagnosis of zygomatic sialocele. Obstructions in the small glandular aggregations in the mouth may give rise to blister-like cystic lesions, particularly around the soft palate (Fig. 55.6).

• **FIG. 55.5** Zygomatic sialocele in a 6-year-old bloodhound. Note that the eye is displaced dorsally by the lesion that has expanded in the floor of the orbit.

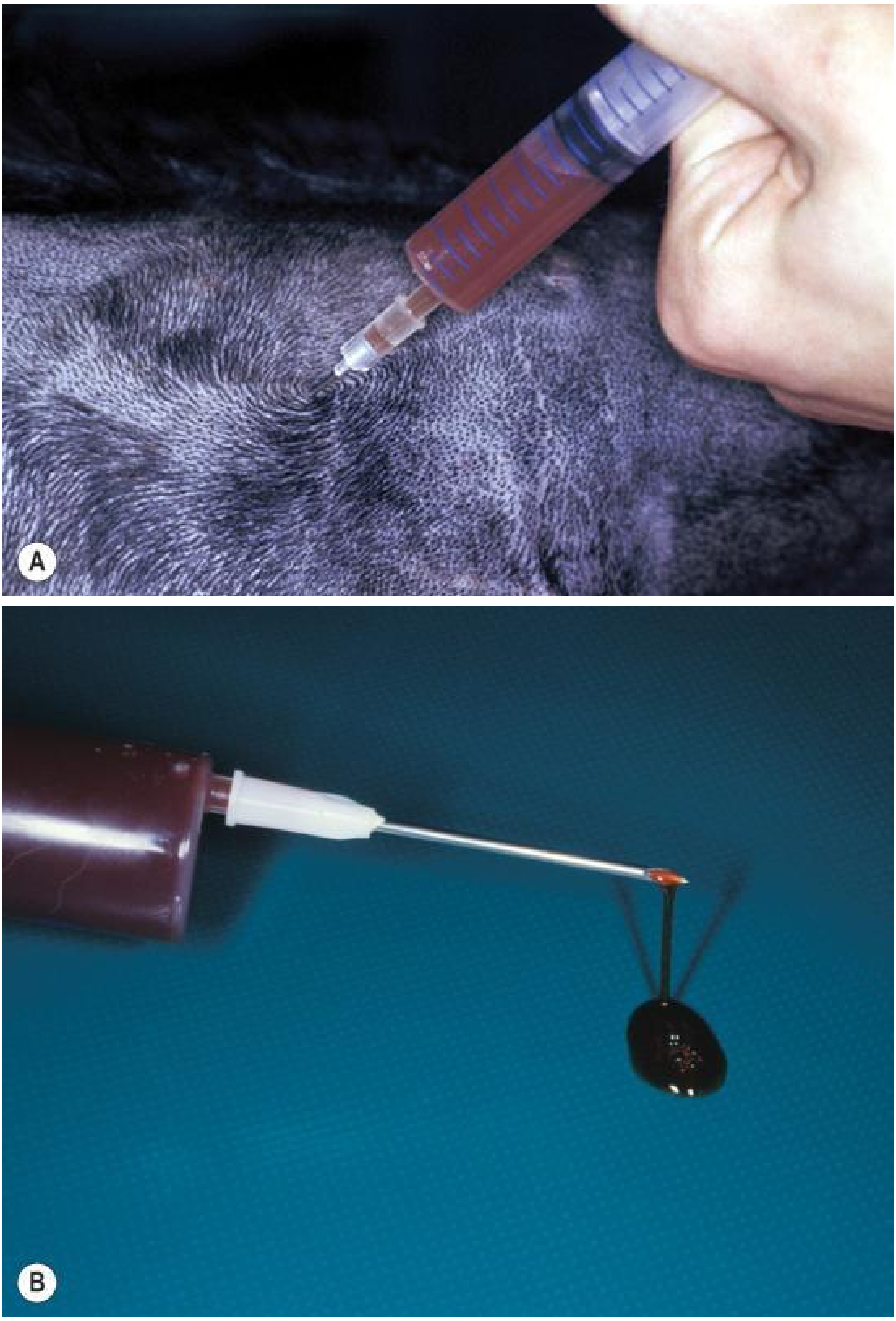
• **FIG. 55.6** Sialocele arising from a minor gland in the palatal submucosa in a French bulldog that was presented with dyspnea.

Sialoceles in cats almost invariably form by leakage of saliva from a ductule of the sublingual gland, causing a sublingual sialocele.[8] However, there has been a single report of a lesion at the pharyngeal site causing obstructive dyspnea,[15] and another case report in which a sialocele from the zygomatic gland arose in the ventral region of the orbit.[16]

Preoperative assessment

Diagnosis of sialocele

The diagnosis of cervical and sublingual sialoceles generally presents no difficulties. The lesions consist of thin-walled, painless, fluctuant swellings. Also, cervical sialoceles can be differentiated from abscesses because the skin overlying a sialocele is never adherent to the underlying lesion unless there has been previous surgical interference. Aspiration of the contents of the sialocele confirms the diagnosis (Fig. 55.7). The stagnant saliva in a sialocele is thick and tenacious; it is difficult to draw through an 18-gauge needle and will form strings when expressed from a syringe. Sometimes it is bloodstained, but so is the pus that might gather in an abscess in this area in the dog. However, pus, while tacky, cannot be described as tenacious. In referral practice, diagnosis by examination of the aspirated fluid can be confused if there has been recent drainage of the sialocele, in which case the vacated space may fill temporarily with serum. The side of origin of a unilateral sialocele in the cervical site is usually obvious in the early stages, but as sialoceles at this site become more pendulous, there is a tendency to migrate toward the ventral midline. Unless the client's memory is dependable, it can be difficult to decide which side is involved. Thus, the major diagnostic challenge with sialoceles can be to determine from which side a cervical lesion has arisen. If this is difficult in the normal standing position, the dilemma may be clarified by placing the patient in dorsal recumbency, after which the swelling should gravitate more toward the side of origin. Sialography (the radiographic study of the salivary glands following the introduction of contrast medium through the duct papillae) may appear to offer the definitive diagnostic technique.[1,4] However, cannulation of the papillae can be very time consuming and frustrating, making this an impractical procedure for general practice. Another alternative is to use contrast computed tomography for accurate localization of the affected salivary gland. The ventral surgical approach to treatment described below affords a simpler means to resolve the dilemma.



• **FIG. 55.7** (**A**) Aspiration of the contents of a cervical sialocele. Note that the stagnant saliva is bloodstained. (**B**) Aspirated saliva is viscous.

Saliva is saturated by calcium hydroxyapatite and calcium salts, which may precipitate within a salivary duct to form sialoliths. Occlusion of a salivary duct by sialolith(s) is one of the proposed causes of sialoceles. However, histologic studies have suggested that sialoliths are not implicated in the majority of sialoceles.[17] On the other hand, stagnant saliva within a sialocele often contains tapioca-like particles of semimineralized aggregates of calcium salts. The discovery of such particles at surgery reinforces the diagnosis of sialocele.

The diagnosis of a parotid or zygomatic sialocele relies on a combination of the location of the lesion, palpation, aspiration of the contents, and supporting imaging techniques including ultrasonography, radiography, and computed tomography (CT).

Therapeutic decision making

Objectives of surgery

The primary purpose of this section is to describe two methods to extirpate the sublingual and mandibular glands, one using the traditional lateral surgical approach and the other using the ventral approach, and to comment on their relative merits and detractions. In a series of 166 dogs with sialoceles referred for surgical correction, the lateral route was used for 120 operations, and the ventral technique was employed 60 times.[8] The apparent disparity in addition arises because there were 10 bilateral cases (6.25%), and in four instances where there was recurrence after excision of the glands by the lateral approach, secondary surgery by the ventral method was used. Cats are far less frequently subject to sialocele formation; over the same time period, only seven feline cases were presented, all of which arose at the sublingual site and were treated successfully, four by the lateral route and three ventrally.

In referral practice it is likely that many patients will already have been subjected to one or more of a variety of techniques attempting to resolve the sialoceles. Drainage, as opposed to diagnostic aspiration of saliva, is the most likely. However, persistence of a sialocele after attempted extirpation of the mandibular and sublingual glands is not an uncommon finding, particularly when the lateral approach to surgery has been employed. Not all such failures can be attributed to the shortcomings of the lateral technique, because some clinicians may be confused in the identification of the salivary glands. Instances have been documented where a complete glandular chain was found on each side after the salivary glands were purported to have been excised. Possibly the local lymph nodes had been removed in error.[8]

Surgical anatomy

General anatomy

The anatomy of the salivary glands is presented in detail in Chapter 54 and is summarized simply here. In the dog and cat there are four pairs of major salivary glands: the parotid, zygomatic, mandibular, and sublingual. In addition to the major glands, salivary tissue is generously distributed in the oral and pharyngeal submucosa. The major section of the sublingual gland, the caudal (monostomatic) portion, shares a common capsule with the mandibular gland and (as the name suggests) drains by a single duct to the sublingual caruncle. Rarely, the distal segment of this duct is united with the mandibular gland duct.[4] A number of small tributary ducts lead from the lobules of the sublingual gland into the monostomatic duct.

Topographical anatomy

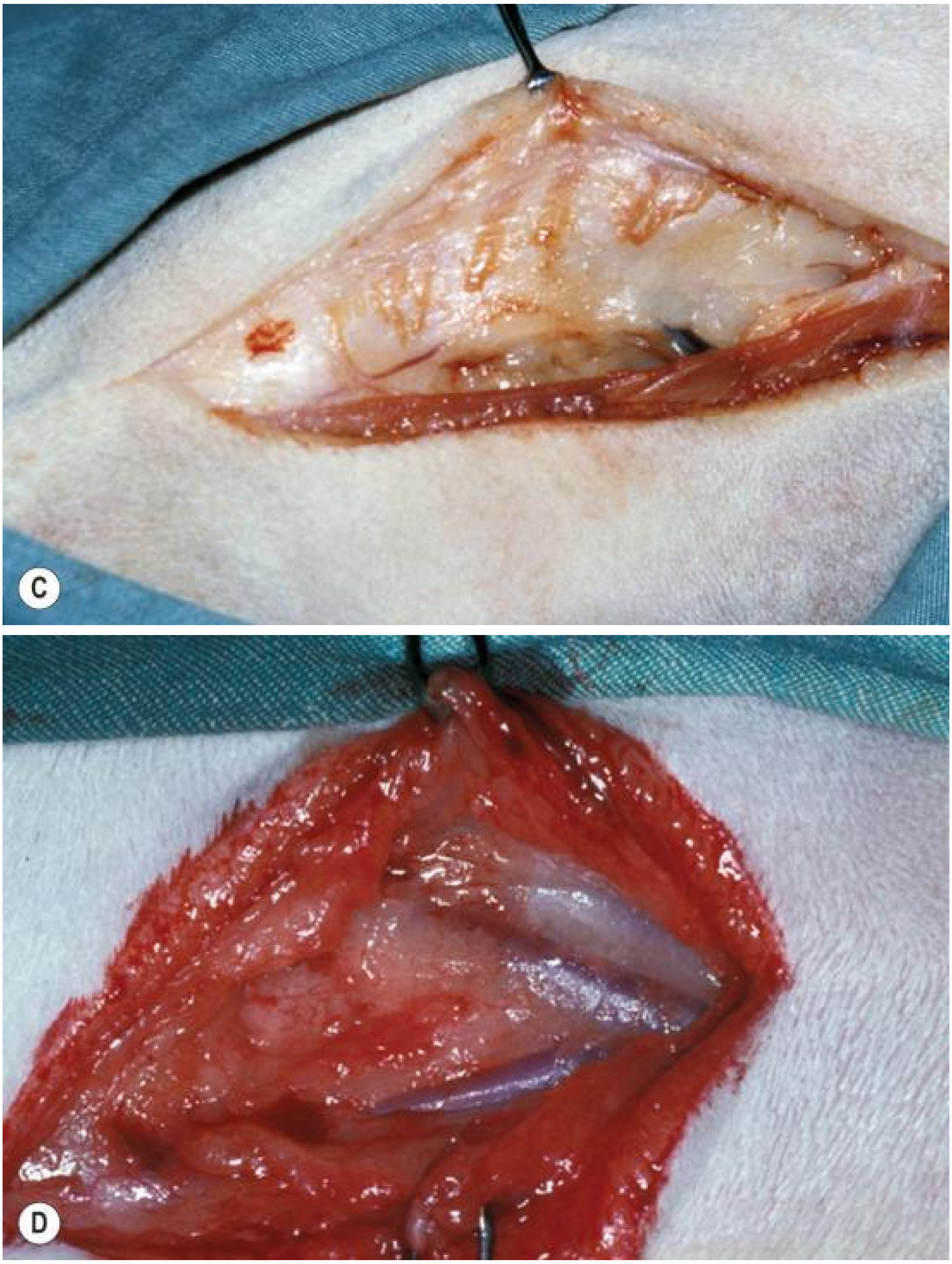
The mandibular gland forms a palpable spherical mass adjacent to the angle of the mandible, in the fork where the linguofacial branch joins the jugular vein. It lies within a tough fibrous capsule that extends rostromedially to envelop the mandibular duct and the sublingual glandular chain and duct. The mandibular gland receives its blood supply through the glandular branch of the facial artery, which enters the capsule at the rostromedial aspect, close to the exit point of the mandibular duct. The mandibular duct, together with the sublingual glandular chain and duct, travels rostrally between the medial pterygoid and digastricus muscles, effectively passing through a tunnel over the dorsal surface of the latter. On leaving the tunnel, the chain passes between the styloglossus and mylohyoid muscles and eventually comes to lie beneath the oral mucosa at the lateral aspect of the tongue. Whichever method of surgical excision is used, communication with the sialocele should be identified during the surgery so that the stagnant saliva within can be drained without creating a separate incision. In rare cases, a sialocele may have become very pendulous, resulting in stretching of the overlying skin to a point where a cosmetic procedure is required to excise the redundant skin. This is more conveniently performed when the ventral approach is used, as it simply entails creating a bi-elliptical rather than a linear incision.

Surgical techniques

Lateral approach to excision of mandibular and sublingual glands[18]

The patient is positioned in lateral recumbency with the affected side uppermost (Fig. 55.8A). The neck is extended over a roll placed behind the underside ear. An incision is made over the mandibular gland, lying in the division between the jugular and linguofacial veins (Fig. 55.8B–D). After clearing the overlying fat and connective tissue, the tough capsule of the mandibular gland is incised in order to shell out the gland within (Fig. 55.8E, F). The dissection continues between the medial pterygoid and digastricus muscles, with gentle tension applied to the glandular chain (Fig. 55.8G, H). Amputation of the chain is made as far rostrally as access between these two muscles will permit (Fig. 55.8I). The surgery concludes with routine closure of the overlying soft tissues and skin.


A
B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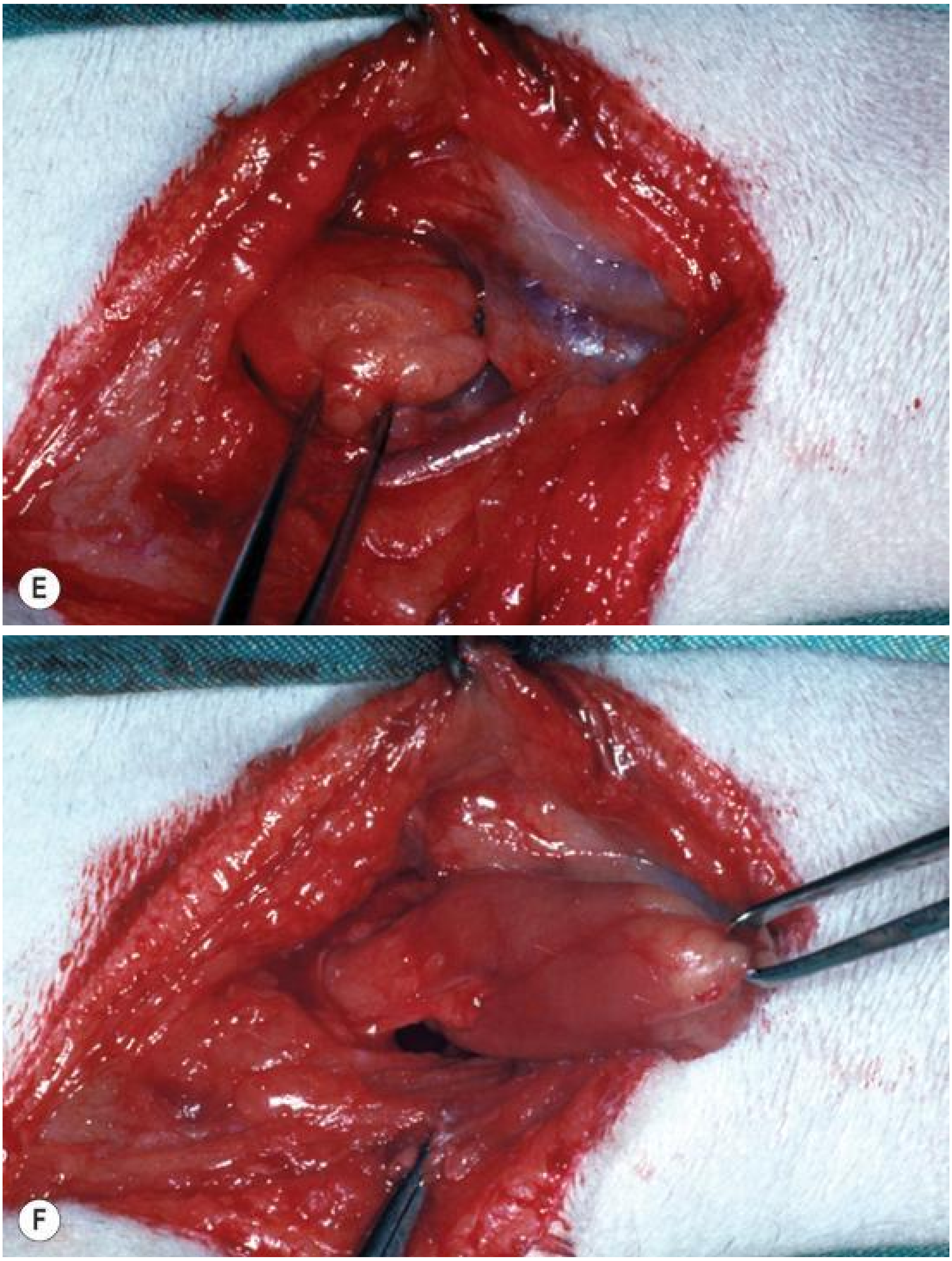
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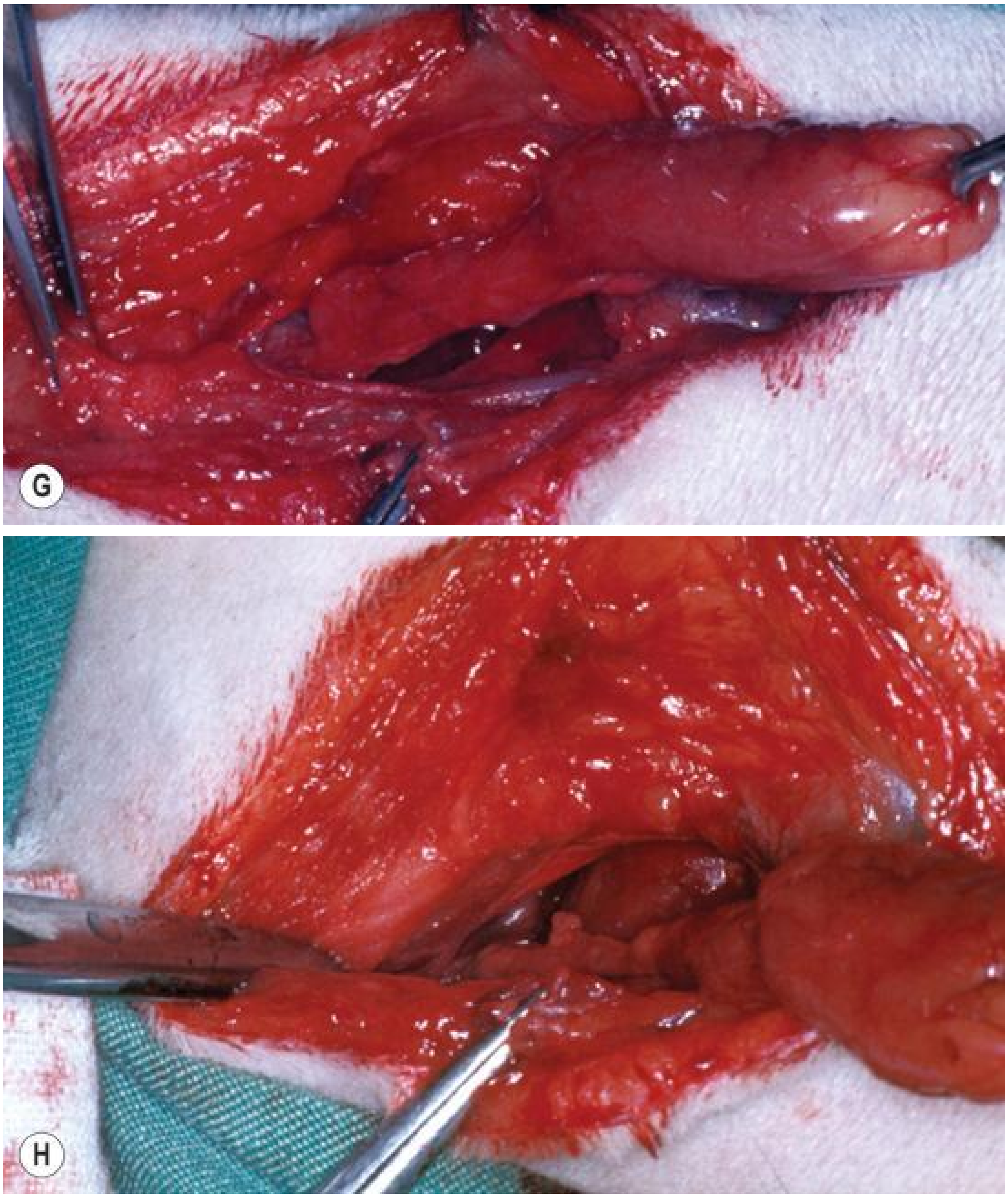
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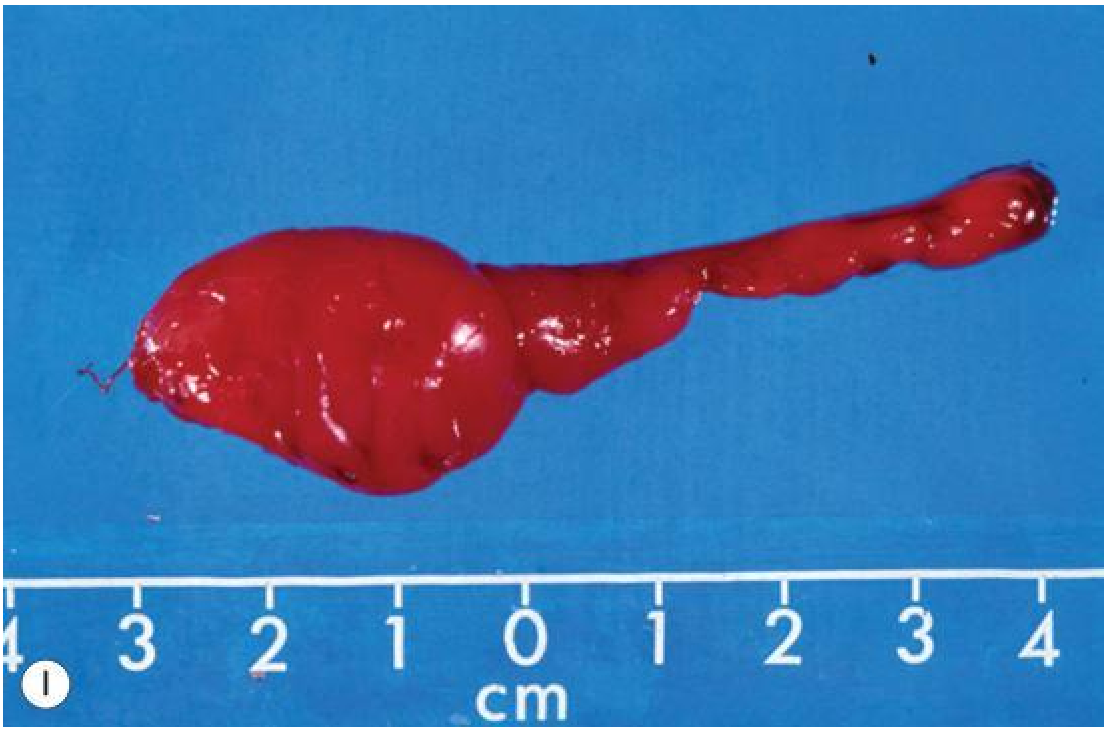
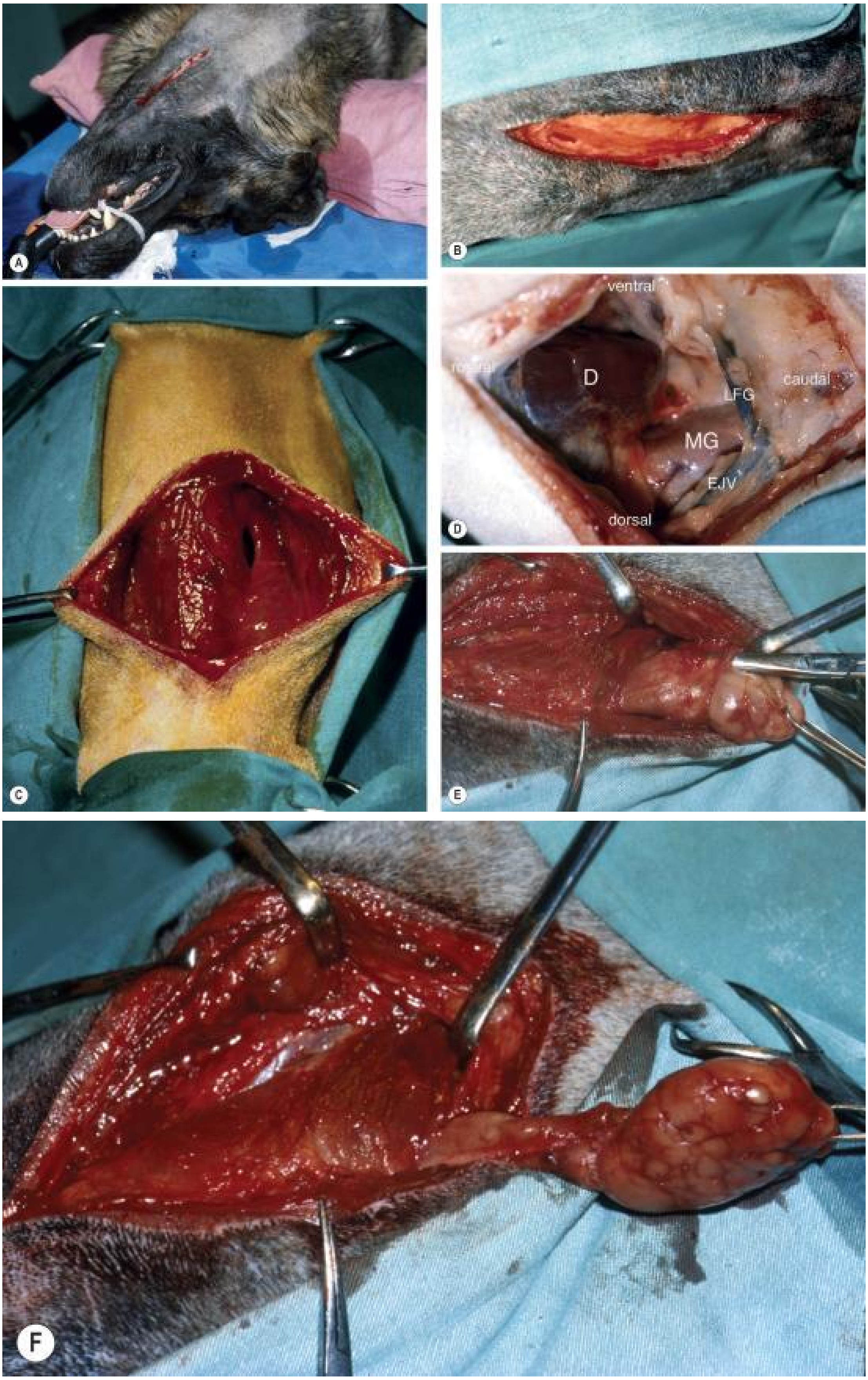


• FIG. 55.8 (**A**) For lateral excision of the mandibular and sublingual salivary glands, the patient is positioned in lateral recumbency with the head extended over a sandbag or roll. (**B**) The skin incision is made directly over the mandibular gland. (**C**)The underlying panniculus muscle is divided. (**D**) The jugular and linguofacial veins are identified. (**E**) The mandibular gland is identified, lying in the division between the veins. (**F**) The mandibular gland is freed from its capsule and drawn laterally. (**G, H**) The chain of the sublingual gland if freed as it passes between the medial pterygoid and digastricus muscles. It is at this stage that the contents of the sialocele may well up into the incision. (**I**) Mandibular and sublingual glandular chain after amputation.

Ventral approach to excision of mandibular and sublingual glands[8,19,20]

The patient is positioned in dorsal recumbency with the neck extended over a sandbag. A longitudinal incision is made medial to the ramus of the mandible and is extended caudally beyond the level of the angular process where the mandibular gland is palpable (Fig. 55.9A, B). A midline incision is used in cases when it is unclear from which side the sialocele has arisen. When the sialocele is in the cervical location, the ventral approach usually necessitates passage through the sialocele itself. This should be regarded as a help and not a hindrance, particularly with midline sialoceles, because tracts will be seen leading from the cyst to the diseased salivary tissue, thereby confirming the side and level of origin (Fig. 55.9C). The dissection is continued through the subcutaneous tissues caudally, and the mandibular gland is identified deep to the bifurcation of the external jugular vein (Fig. 55.9D). The arterial and venous blood supply enters the capsule from the rostromedial aspect; these vessels should be identified and ligated. The glandular tissue is dissected free from its capsule, and the caudal lobules of the sublingual chain are also freed. When this route is used, the dissection rostrally is inhibited by the presence of the digastricus muscle, which crosses the path of the glandular chain (Fig. 55.9E). Thus, at this point, attention is transferred to the rostral portion of the sublingual chain. The line of dissection passes through the tissues between the tongue and the body of the mandible by splitting between the mylohyoid and styloglossus muscles longitudinally. At this level, the sublingual chain is located together with the mandibular duct as it passes dorsal to the digastricus muscle. In cases where previous excision of the glandular chain has been inadequately performed, the stump of residual glandular tissue can be found here. The introduction of a finger or retractor beneath the belly of the digastricus from the caudal aspect helps free the glandular chain and creates a tunnel through which the glandular tissue can be passed to the rostral portion of the surgical field (Fig. 55.9F, G). The complete chain is then amputated cranial to the most rostral glandular lobule. Structures to identify and preserve during the dissection include the hypoglossal and lingual nerves and the lingual artery. A Penrose drain may be used to prevent seroma formation in the dead space created by the surgery. Otherwise, closure of the incision is routine.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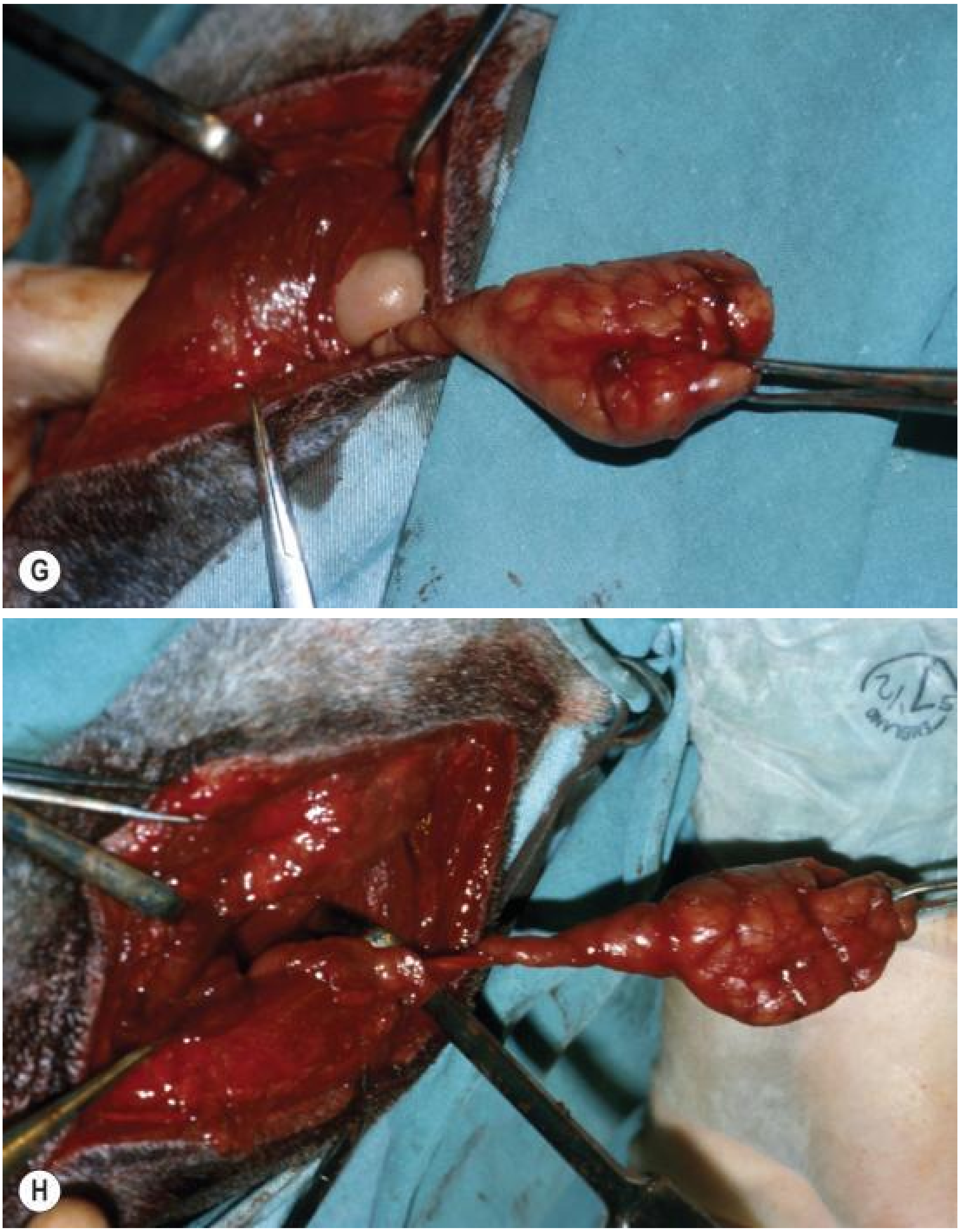


• FIG. 55.9 (**A, B**) For ventral excision of the mandibular and sublingual salivary glands, the dog or cat is positioned in dorsal recumbency with the neck extended. The surgical drapes have been removed to show patient positioning. The skin incision is made medial to the mandible on the affected side or at the midline if the side of origin of the sialocele is uncertain or bilateral. The incision extends caudally to the level of the mandibular gland. (**C**) When the sialocele is incised, a tract can be seen leading to the glandular tissue on the afflicted side. (**D**) Cadaver dissection showing relationship of mandibular gland *(MG)* to the external jugular vein *(EJV)*, linguofacial vein *(LFG)*, and digastricus muscle (**D**). The right side is exposed and the dog is in dorsal recumbency. (**E**) The mandibular gland is separated from its capsule. (**F**) The sublingual glandular chain passes dorsal to the digastricus muscle. (**G**) Blunt dissection with a finger creates a tunnel dorsal to the digastricus muscle through which the chain of glands can be passed. (**H**) The full length of the glandular chain has been freed and lies medial to the digastricus muscle immediately before amputation.

Advantages and disadvantages of lateral versus ventral approaches for sialoadenectomy: Results of two techniques to excise the mandibular and sublingual salivary glands[8,19,20,21]

Although the lateral approach to ablation of the mandibular and sublingual salivary glands is traditionally advocated, the technique has two shortcomings, either or both of which may lead to failure and persistence of the sialocele. The triangular space formed by the linguofacial and jugular veins, combined with the caudal border of the mandible, limits access as the dissection proceeds between the medial pterygoid and digastricus muscles. Thus, residual glandular tissue from the more rostral lobules of the sublingual gland is invariably left behind. If one of these should be the source of the salivary leakage, the sialocele will persist. Alternatively, if the contralateral chain (inaccessible by the lateral approach) is the source of the salivary leakage, the sialocele will persist.

Table 55.2 illustrates the results from one study where sialoceles persisted in a substantial number of dogs when the lateral approach to glandular excision had been attempted before referral. These failures were attributed to one of three causes: (1) the surgery had been performed on the incorrect side; (2) the rostral part of the glandular chain had not been excised on the correct side; or (3) poor anatomical appreciation of the region had led to failure to identify the mandibular gland, and the entire glandular chain remained in situ.[8]

TABLE 55.2

Treatments Used in 166 Dogs With Sialoceles Before Referral. [8]

Previous Surgery	Number
No previous surgery	77
Drainage of sialocele (excludes diagnostic centesis)	65
Excision of sialocele	17
Marsupialization	6
Excision of mandibular and sublingual salivary glands:	21
Once	18
Twice	2
Three times	1

Table 55.3 reproduces the results reported in 120 operations when the lateral method was used. Cases with follow-up intervals of less than 2 months were not included, and the longest period over which data were available was 12.5 years. Although the results were generally favorable, recurrence or persistence of the sialocele was recorded in six cases. Two of these were euthanized without a request for further treatment, but the other four were subjected to secondary surgery by the ventral route. In each case, it was established that residual rostral lobules of the sublingual gland were responsible.

TABLE 55.3

Results of Surgery to Excise Mandibular and Sublingual Glands. [8]

Lateral Approach:	120 Operations
Lost to follow-up	36
Operations with follow-up: 2–185 months	84
Recurred/persisted (including 2 euthanized)	6 (7%)
Resolved, no complication	75 (89%)
Resolved after delayed healing	3 (4%)
Total resolved	78 (92%)

The major detraction of the ventral technique is that it is more tedious to perform than the lateral method. The ventral approach to the surgery has two advantages over the lateral technique: (1) it allows exposure and excision of the entire sublingual chain,[20] and (2) it permits the flexibility to remove the glandular chain on either or both sides through a single incision. The ventral approach provides the only effective option when lateral surgery has already failed, as was the case with 25 of the operations reported. However, in 35 cases of sialocele it was used as the primary surgical technique. The technique was also used uneventfully in three cats.

The results of 60 operations to extirpate the mandibular and sublingual glands by the ventral approach are presented in Table 55.4. There was no record of recurrence or persistence of a sialocele after glandular excision by this method. One dog required a second interference 6 hours after the initial surgery to control hemorrhage from a branch of the lingual artery, and four dogs showed partial wound dehiscence with delayed healing by second intention.

TABLE 55.4

Results of Surgery to Excise Mandibular and Sublingual Glands. [8]

Ventral Approach:	60 Operations
Lost to follow-up	6
Operations with follow-up: 2–122 months	54
Recurred/persisted	0
Resolved, no complication	50 (93%)
Resolved after delayed healing	4 (7%)
Total resolved	54 (100%)

The results of a second study of 41 cases of canine sialocele treated by excision of the salivary glands using the ventral route have been similar: none of the 31 dogs available for follow-up showed recurrence of the sialocele, and seven sustained transient wound complications.[19]

In light of the evidence reviewed above, excision of the mandibular and sublingual glands by the ventral approach should be the primary treatment of choice for sialoceles in the dog. It is also recommended that this be the preferred option to treat the disorder whenever it arises in cats.

Intraoral marsupialization of a ranula

Marsupialization consists of the partial excision of the wall of a ranula followed by coaptation of the lingual mucosa to the lining of the sialocele to form a "pseudofistula," allowing saliva filling the sialocele to drain into the oral cavity.[7] The technique has not been subjected to statistical scrutiny and is probably only of historical interest, having its origins at a time when it was still believed that the lining contributed to the contents of the sialocele. Nevertheless, some success has been reported, especially when nonabsorbable sutures have been used to appose the inflammatory lining of the sialocele to the oral mucosa. However, the recurrence rate with marsupialization is such that removal of the salivary glandular chain offers a greater likelihood of a successful outcome (see also Table 55.2).[7,8]

Parotid sialoadenectomy

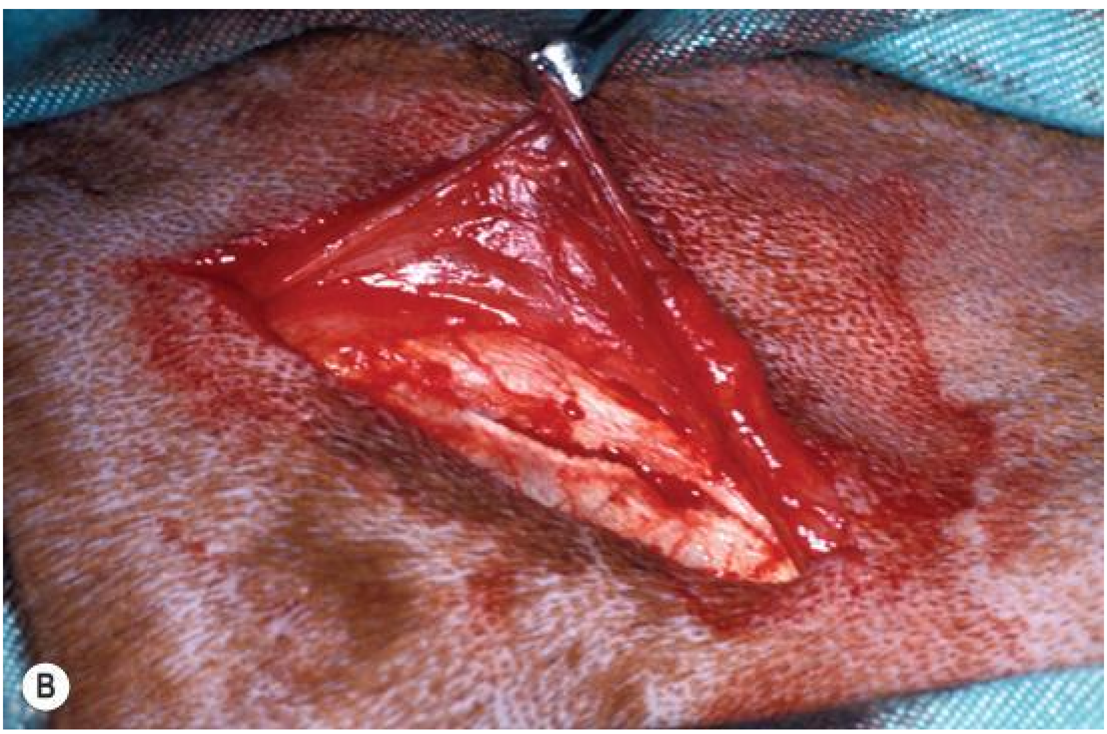
The surgical anatomy of the parotid salivary gland is described in Chapter 54 and, in addition to the treatment of a sialocele, the indications for the removal of the gland may include sialolithiasis, neoplasia, lipomatosis, and trauma.[12] The anatomical proximity of the facial nerve and the branches of the maxillary artery, linguofacial vein, and auricular vessels make this a challenging procedure. A vertical skin incision parallel with the ramus of the mandible is followed by separation of the overlying fascia. The parotidoauricularis muscle is sectioned followed by meticulous dissection of the gland in a caudodorsal to ventrorostral direction. The identification and preservation of the facial nerve as it emerges from the facial canal and passes ventral to the horizontal ear canal is an important aspect of the deeper dissection. Although intraoperative and postoperative complications of parotidectomy are relatively frequent, the overall success rate with long-term resolution of sialoceles arising from this gland is high.[12]

Zygomatic sialoadenectomy

The indications for the removal of the zygomatic salivary gland are very rare and include traumatic rupture, sialocele, and neoplasia. Upon clinical presentation of a patient with proptosis (especially when the globe is displaced dorsally), diagnosis may be made by ultrasonography and fine-needle aspiration. If ultrasonography confirms a fluid-filled structure, and saliva is identified upon aspiration, the diagnosis of zygomatic sialocele is confirmed, while soft tissue echogenicity and a cellular aspirate are more suggestive of neoplasia and are an indication for histopathologic analysis. In addition, oral inspection may reveal a swelling caudal to the maxillary molar teeth, which will fluctuate in the case of a sialocele but will be firmer in the case of a neoplastic disorder.

The zygomatic salivary gland lies in the floor of the orbit and is protected by the overlying arch of the zygomatic bone, which restricts surgical access. The author's preferred method for zygomatic sialoadenectomy is to remove the ventral half of the zygomatic arch after dissecting between the aponeurosis of the masseter muscle and the periosteum (Fig. 55.10A–C). The gland is generally obscured by orbital fat, which is cleared before the gland is withdrawn by gentle traction (Fig. 55.10D). The incision is closed by sutures placed between the periosteum and the masseteric fascia, followed by closure of the subcutis and skin in a routine fashion. The advantage of this technique is cosmetic in that it preserves the facial outline. More generous exposure can be achieved by a full-thickness zygomatic osteotomy.[1]


A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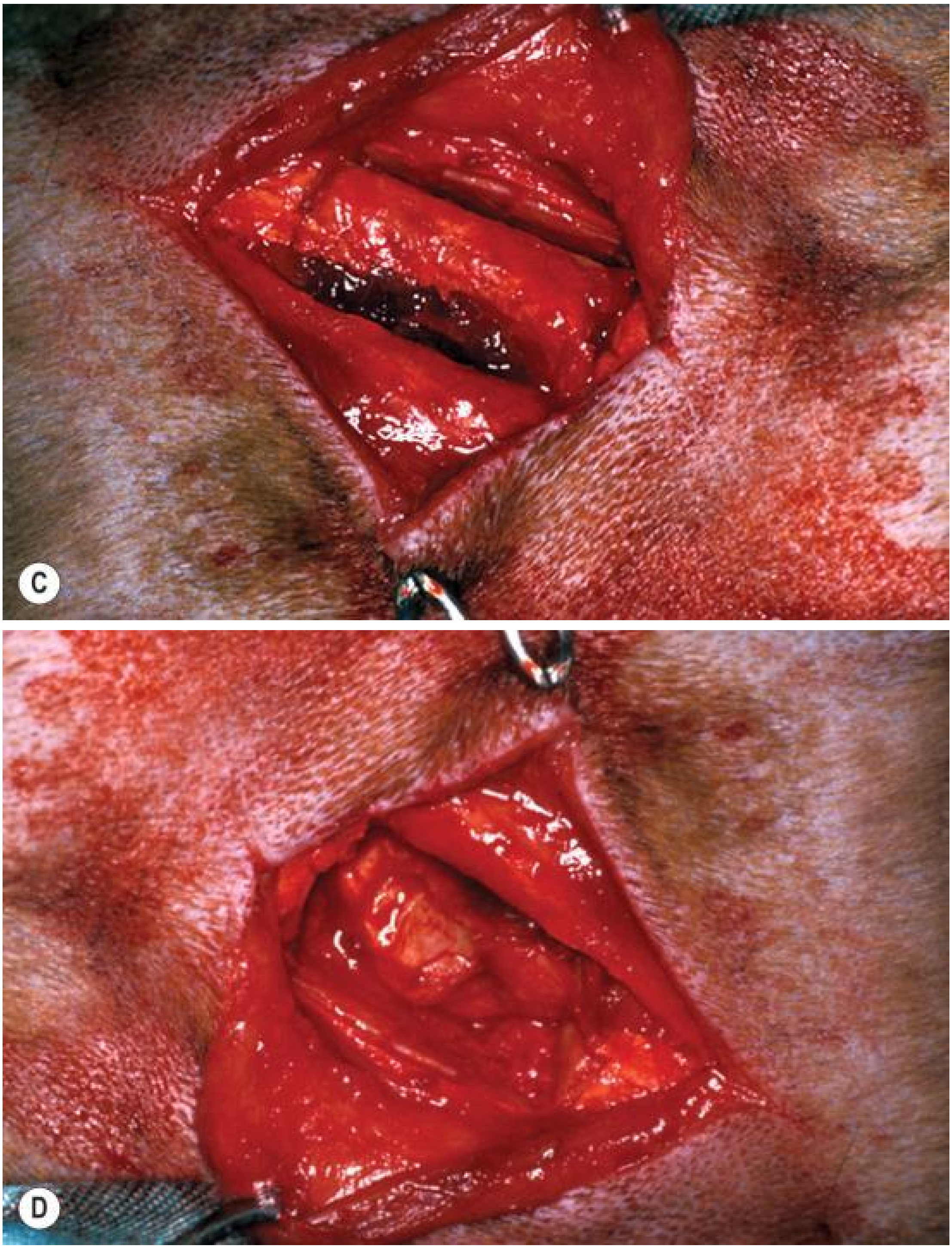

• FIG. 55.10 (**A**) The area ventral to the zygomatic arch has been prepared for surgery. (**B**) The zygomatic arch is exposed at its ventral margin where the aponeurosis of the masseter muscle attaches. (**C**) The ventral section of the zygomatic arch is resected to leave the dorsal arch intact. (**D**) The infraorbital fat has been cleared to expose the underlying salivary gland, which is removed by gentle traction.

Postoperative care, assessment, and complications

Excision of the mandibular and sublingual salivary glands

Mandibular and sublingual sialoadenectomy should be regarded as sterile surgical procedures that do not enter the upper alimentary or respiratory tracts. Thus, normal rigorous aseptic techniques are adopted during preparation for surgery, and prophylactic antibiotic therapy is not required. There are no reports in the literature of a dog or cat that developed deep suppuration after sialoadenectomy, and the author's experience supports this. Short-term incisional dehiscence may result from self-trauma by the patient, but this is sufficiently uncommon that the routine use of a protective plastic collar is not necessary.

In the immediate postoperative period, careful observation is necessary to monitor for tense swelling at the surgical site. Such swelling might arise if hemorrhage from the nutrient vessels of the mandibular salivary gland has not been controlled or if the lingual artery has been inadvertently damaged during surgery. The proximity of the upper respiratory tract renders the patient at risk of asphyxiation if such swelling is not recognized and the cause not addressed promptly.

The dead space previously occupied by the fluid contents of the sialocele is likely to fill with serum even after the Penrose drain has been withdrawn, but the resultant seroma should never become tense or painful. Clients should be advised that some filling at the site is inevitable, and that this does not indicate that the surgery has been unsuccessful. The seroma will normally have regressed by the time the skin sutures are removed 10–14 days postoperatively. Longer-term swelling (weeks or months after a lateral approach to sialoadenectomy) suggests that the sublingual gland has been incompletely excised.

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SECTION 11:

Miscellaneous Maxillofacial Surgery Procedures

OUTLINE

CHAPTER 56

Cheiloplasty

Daniel D. Smeak

Lip fold excision

Dogs with infections involving the lips are typically presented for treatment of severe halitosis or facial pruritus. Skin fold intertrigo is a frictional dermatitis that occurs in areas where two skin surfaces are intimately apposed.[1] If moisture, sebum, glandular secretions, or excretions such as tears, saliva, or urine are present, these areas provide an environment that favors skin maceration and bacterial overgrowth.[1] Although bacteria play a central role in the pathogenesis of fold dermatitis, they rarely invade tissues, so the designation of skin fold intertrigo as a fold pyoderma is technically inaccurate.[1] The surface bacteria act on trapped secretions and sebum and produce breakdown products, which are irritating and odoriferous.[1] Lip fold intertrigo is found caudal to the mandibular canine teeth where the lower lips are contacted by the tips of the maxillary canine teeth (Fig. 56.1). Lip fold excision eliminates these folds so there is no further accumulation of food and saliva to trigger infection.[2]

• **FIG. 56.1** Severe lip fold pyoderma in a dog.

Clinical characteristics

The affected area is usually red, glistening, and ulcerated and, unless the condition is severe, the lesion is confined to a sharply demarcated area within the recesses of the folds. There may be a film of purulent exudate and debris covering the area. On physical examination, the problem is often not obvious until the fold is stretched, revealing the affected area. This condition is most common in dogs with large, pendulous upper lips and prominent lower lip lateral folds, such as St. Bernards, schnauzers, and brachycephalic breeds, but spaniels and setters appear to be particularly predisposed. Food, hair, and saliva collect in these folds, causing inflammation and fetid odors. Since the condition is due to a conformational problem, the course of the disease is chronic and usually only temporarily improved by medical therapy.

Associated conditions

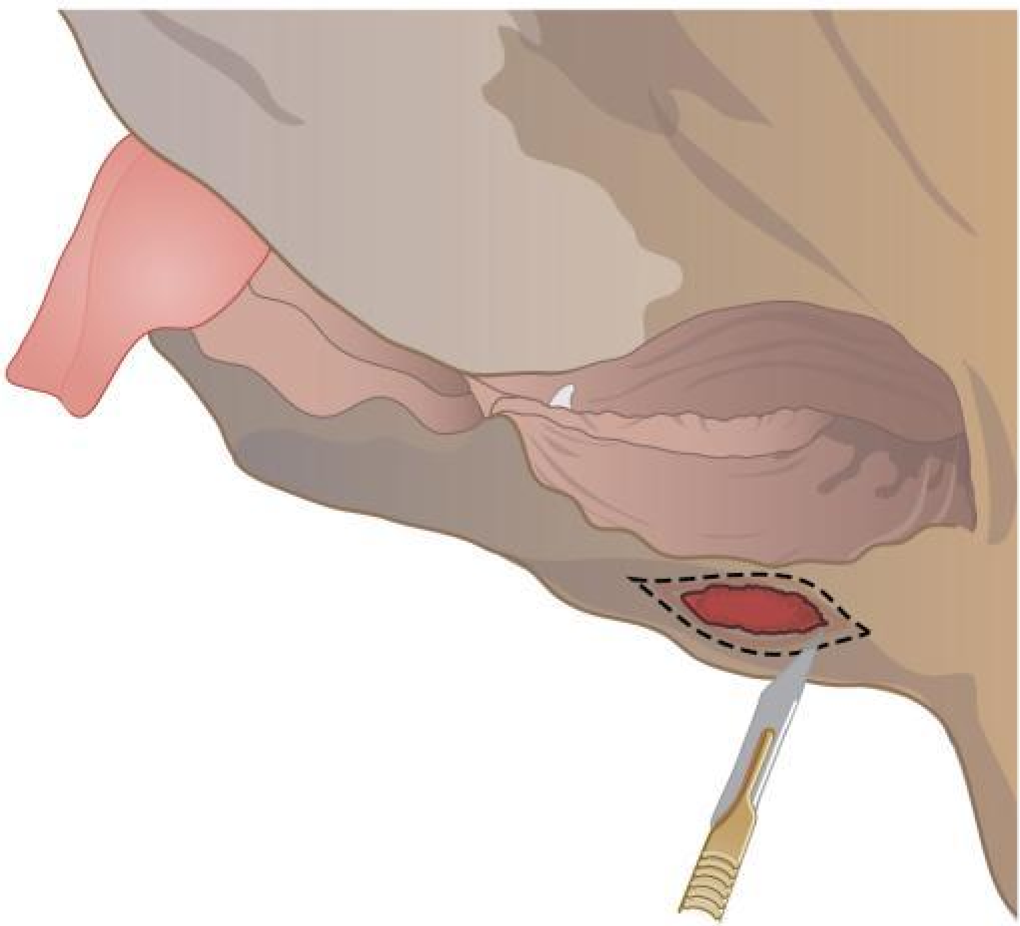
Lip fold pyoderma should be differentiated from other conditions that cause halitosis and/or facial dermatitis such as dental disease, canine acne, juvenile pyoderma, contact or food allergic dermatitis, and other immune-related diseases that cause ulceration of the mucocutaneous areas. If the condition is not limited to the lateral folds, or the lip conformation is not considered abnormal, surgical treatment should not be considered and other dermatological problems should be investigated.

Preoperative considerations

Attempt to improve the local tissue condition before surgery by swabbing the folds daily with 2% acetic/2% boric acid solution wipes (MalAcetic Wet Wipe/Dry Bath, Dermapet, Potomac, MD) or shampoo. Use of topical 2% mupirocin (Bactoderm, Beecham Laboratories, Bristol, TN) or silver sulfadiazine and systemic antibiotics such as clavulanic acid/amoxicillin may be helpful if there is staphylococcal colonization of the folds. Administer a single intravenous dose of a cephalosporin antibiotic just before surgery.

Surgical technique

Place the patient in dorsal recumbency to allow access to both lip folds. Prepare the region for aseptic surgery. Create a fusiform incision, parallel to the body of the mandible, around the affected area to include a rim of normal skin (Fig. 56.2). Elevate the underlying subcutaneous tissue attachments to the outlined skin segment without invading the inner buccal mucosa. Control hemorrhage and close the subcutaneous tissue and skin routinely.



• **FIG. 56.2** The lip fold is excised with a fusiform incision around its margin *(dashed line)*.

Postoperative considerations

Use an Elizabethan collar or basket muzzle to prevent self-inflicted trauma. Continue the medical therapy described above and keep the area clean and dry until suture removal. Healing is complete and permanent following elimination of the fold with surgical excision.[2]

Antidrool cheiloplasty

Antidrool cheiloplasty (ADC) is an elective surgical procedure in which the lower lip is permanently attached in a dorsally suspended position to the upper lip.[3]

Purpose and indications

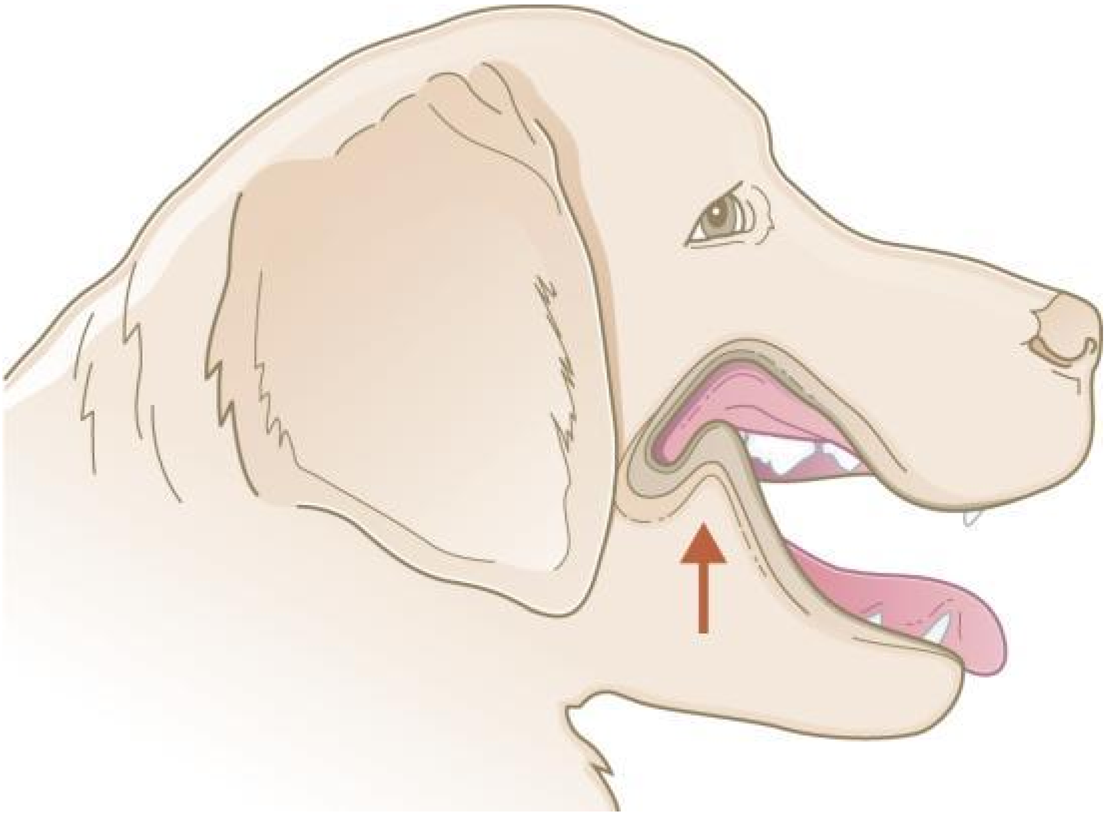
The suspended lower lip reduces the loss of food and saliva from the lateral vestibules of the oral cavity. Indications for ADC include acquired loss of lip motor function from trauma or nerve damage[3] and congenital overabundance and eversion of the caudal lower lip, frequently seen in large- and giant-breed dogs such as the St. Bernard and Newfoundland dog. Most clients seek this procedure because they are tired of cleaning the mess left after their dog eats or drinks.

Preoperative considerations

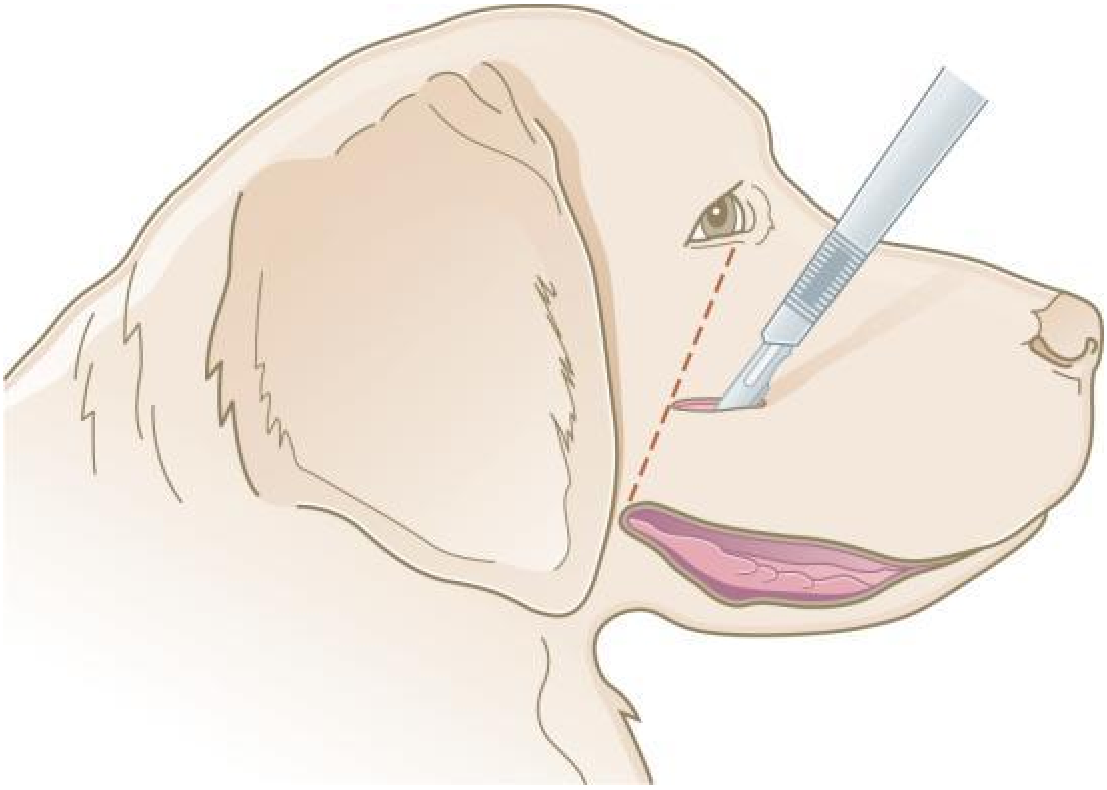
Withhold food from patients for 12 hours before surgery. No prophylactic antibiotics are required. Be sure the endotracheal tube cuff is positioned in the trachea and properly inflated to prevent aspiration of blood during the procedure. Irrigate food and saliva from the oral cavity with water prior to aseptic preparation of the lip. Place the dog in lateral recumbency, prepare the regional skin with povidone–iodine scrub, and daub the buccal mucosa with povidone–iodine solution. Avoid chlorhexidine gluconate antiseptics in or around the face because unintentional ocular contact will result in severe chemosis and conjunctivitis. With the patient in lateral recumbency, stabilize the head and drape the lip region without altering the normal relaxed lip position or the ability to fully open the mouth during the procedure.

Surgical technique

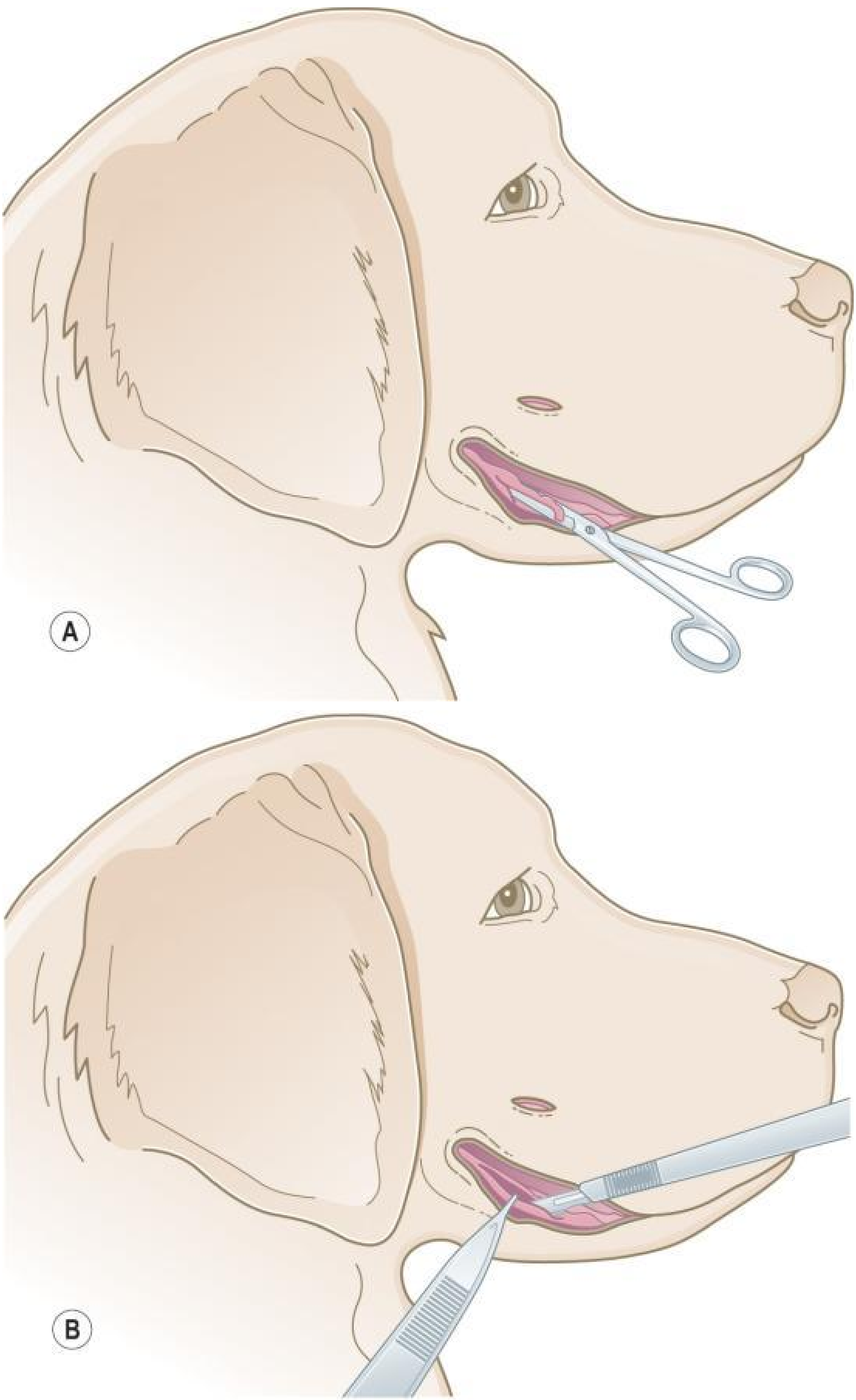
Grasp the irregular lower lip mucocutaneous margin about 20–30 mm rostral to the commissure with an Allis tissue forceps, and elevate the lip dorsally until it is taut when an assistant fully opens the dog's mouth (Fig. 56.3). Mark this spot on the upper lip skin (cheek) with a surgical pen or lightly scratch the skin with a scalpel blade. In most patients this mark roughly corresponds to the level of the distal root of the maxillary fourth premolar tooth. Start a horizontal cheek incision (parallel to the palate), 25–30 mm long, from a point intersecting the marked lip area and an imaginary line drawn from the medial canthus of the eye to the commissure of the lips (Fig. 56.4). Align dissection through deeper cheek tissue directly under the original skin incision, but avoid the superior labial vein coursing just rostrodorsal to this incision. Continue dissection until the buccal mucosa is incised along the limits of the original skin incision, and control hemorrhage before proceeding. Excise a 2-mm mucocutaneous margin of the lower lip with Mayo scissors beginning 20 mm from the commissure and extend the cut rostrally for 25 mm (Fig. 56.5A). Split the cut edge of the lip longitudinally with a scalpel blade to a depth of 7.5–10 mm to create a mucosal and a cutaneous flap (Fig. 56.5B). Pull the flaps through the cheek incision with stay sutures placed at the rostral and caudal aspects of the lower lip incision.



• **FIG. 56.3** Grasp the lower lip, 20–30 mm rostral to the commissure, and elevate it until the lip is taut when the mouth is fully opened. This will mark the dorsal extent of the horizontal cheek incision.

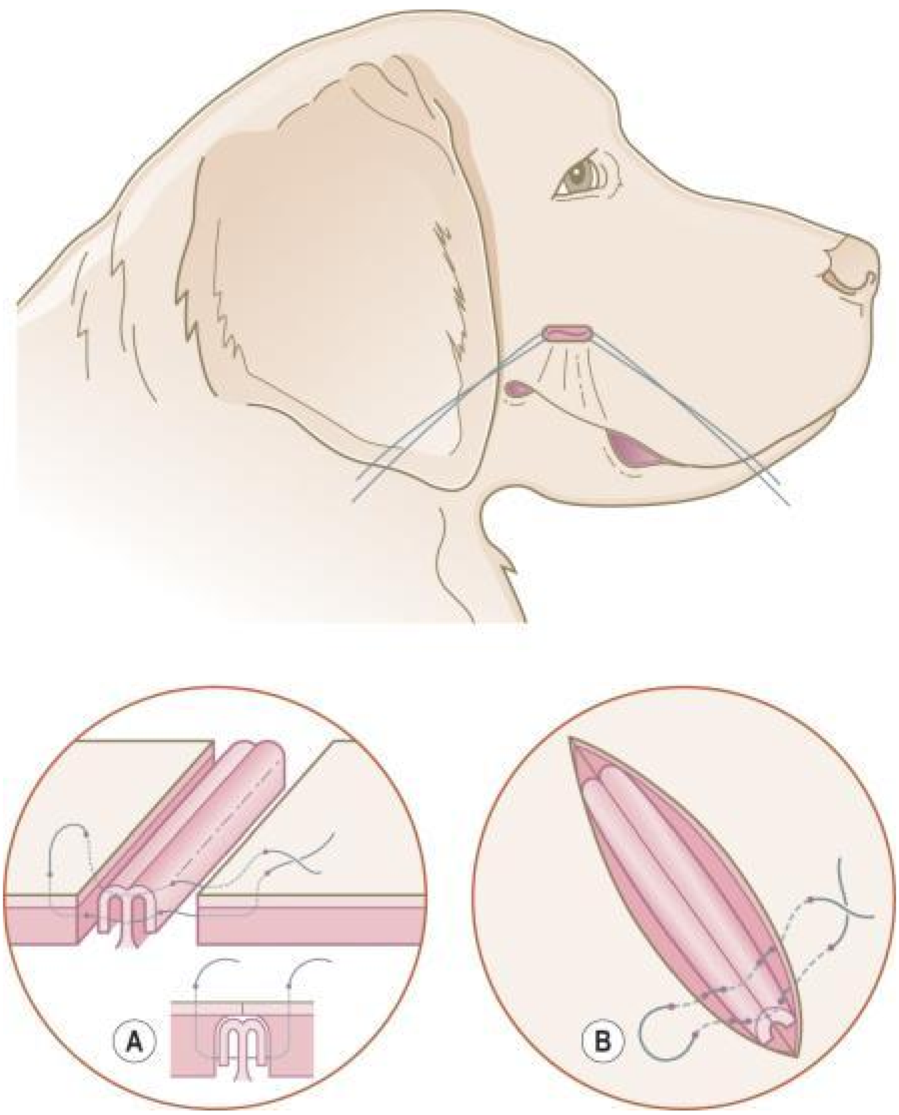


• **FIG. 56.4** Completed full-thickness horizontal cheek incision. The caudal aspect of the incision intersects a line drawn from the medial canthus to the commissure *(dotted line)*.



• FIG. 56.5 (**A**) Excise a 25-mm-long mucocutaneous border from the lower lip. (**B**) Split the incised edge of the lower lip in half to form separate cutaneous and mucosal flaps.

The key goal of the suture pattern described in this procedure is to appose as much raw tissue surface as possible between the cheek incision and lower lip flaps.[3] The mattress pattern must hold the lower lip flaps in eversion within the cheek incision to create a robust and permanent lip adhesion. Appose the everted edges of the lip and cheek with two to three equally spaced and preplaced horizontal mattress sutures using 2-0 or 0 monofilament nonabsorbable suture material. Insert the needle split-thickness through the cheek about 1 cm from the incision. With the flaps everted and elevated from the cheek incision, advance the needle through the raw surface of the facing flap, through the base of the lower lip, out through the opposite flap, and through the cheek skin, again split-thickness. Reverse the needle direction and drive the needle through the lower lip in mirror image to the first needle pass (Fig. 56.6). Tighten each suture just enough to appose the incised edges of the lip and cheek. Moderate swelling occurs after the procedure; this may cause excess intrinsic suture tension, so avoid cinching these mattress sutures too tightly. Check the buccal aspect of the cheek incision to ensure the sutures are correctly placed and lower lip flaps are everted within the upper cheek (Fig. 56.7). When skin is not well apposed, close the cheek incision with simple interrupted 3-0 or 4-0 monofilament nonabsorbable sutures. If bilateral ADCs are planned, undrape and roll the dog, and repeat the procedure on the other side. Bilateral ADCs take approximately 30–45 minutes to complete.



• **FIG. 56.6** (**A**) Preplace three equally spaced horizontal mattress sutures in the lower lip to cause eversion of the flaps within the cheek incision. (**B**) Close-up drawing of suture pattern.

• **FIG. 56.7** Gross appearance of a patient before and after antidrool cheiloplasty. (**A**) Preoperative appearance. (**B**) Following suture removal. (**C**) Appearance of buccal surface at suture removal.

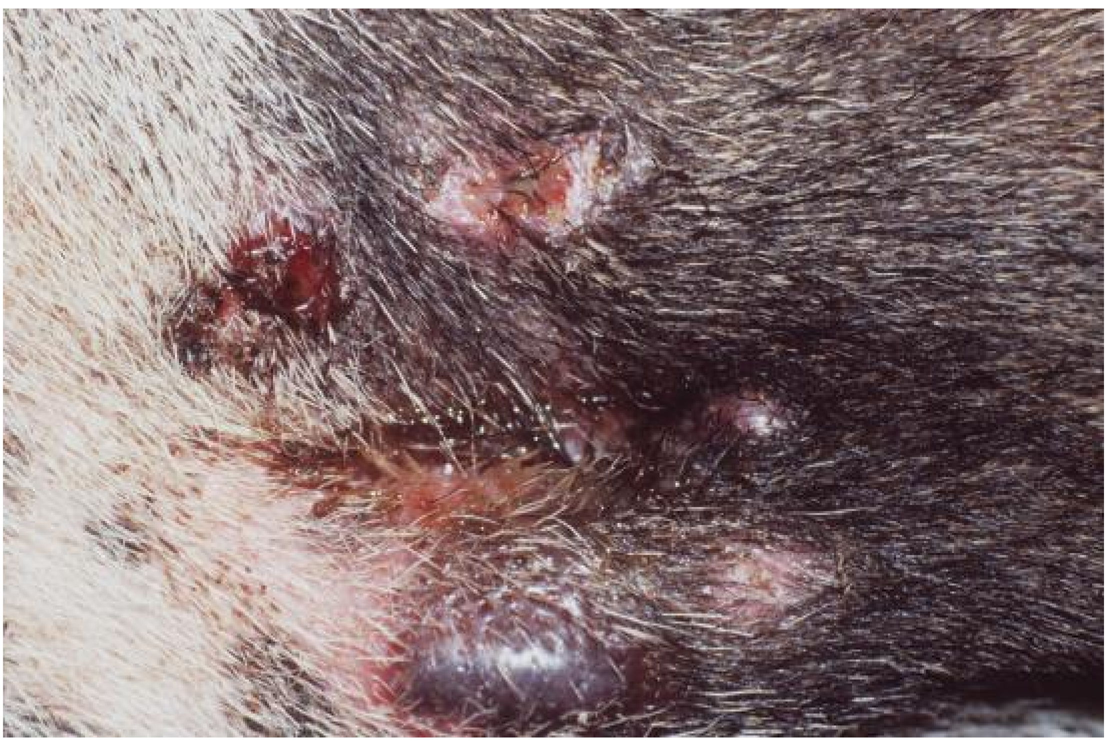
Postoperative considerations

Patients are usually released from the hospital the day following the procedure and can return to their routine diet right away. Irrigation of the oral vestibules after meals with water-filled syringes will remove food debris. Clients should not permit chewable toys or fetching for 2 months after surgery. A short Elizabethan collar should remain on the patient until suture removal to prevent self-mutilation. Sutures are removed and recheck is performed 3 weeks after surgery.

Complications and prognosis

Antidrool cheiloplasty does not appear to cause excessive discomfort after surgery. Dogs are not reluctant to open their mouths the day after surgery and they eat without difficulty. A small indentation or fine incision scar is evident after healing is complete.

Excessive tension on the mattress sutures can cause suture cut-out and abscessation around suture tracks (Fig. 56.8). If this occurs, remove sutures as soon as possible after the mucosal incisions have healed; otherwise, an orocutaneous fistula may occur. Treat the wound open and let it heal by second-intention closure.



• **FIG. 56.8** Suture-induced draining cheek wounds at 3 weeks after surgery.

This procedure is highly successful in reducing drooling associated with congenital lower lip eversion and lip paralysis, but it does not totally eliminate drooling. Advise clients that drooling is reduced to a level similar to dogs with normal lip conformation.[3]

Commissurorraphy

Purpose and indications

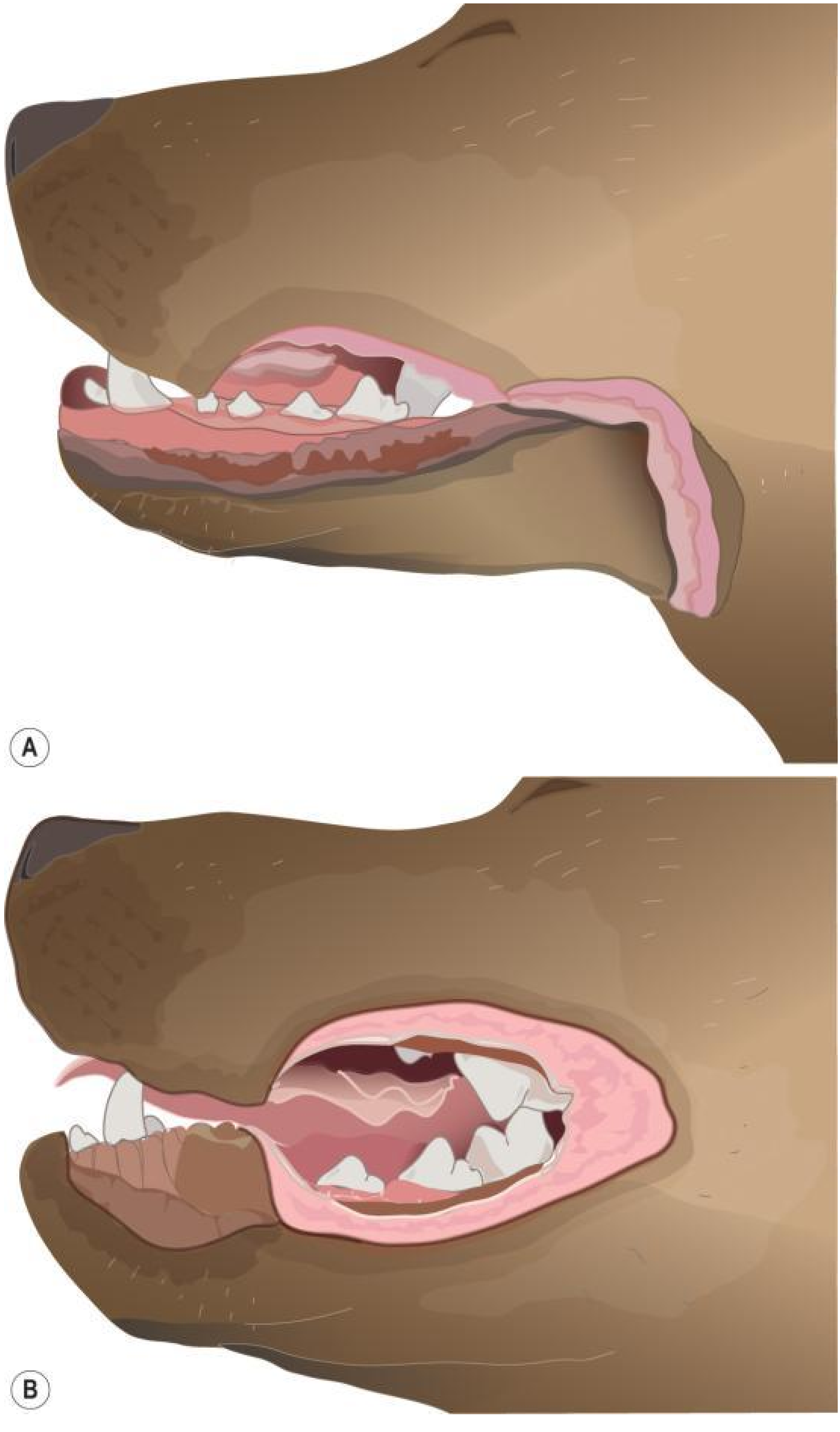
Commissurorraphy, sometimes referred to as lip advancement, is a form of cheiloplasty. It is defined as surgical closure of the lips rostral to the commissure. Lip advancement helps retain the tongue within the oral cavity, helps support the rostral mandible, and may help with food retention and prehension.[4] This surgical procedure is most often performed to provide support for the tongue and improve esthetics after mandibulectomy.[5-9] Bilateral commissurorraphy can be elected in cases undergoing bilateral rostral mandibulectomy, and it has also been described as a salvage procedure to help support bilateral pathological mandibular fractures when bone quality does not allow rigid fixation.[1,10-12]

Preoperative considerations

Withhold food from patients for 12 hours before surgery. No prophylactic antibiotics are required. Be sure the endotracheal tube cuff is positioned in the trachea and properly inflated to help prevent aspiration of blood during the procedure. Irrigate food and saliva from the oral cavity with water prior to aseptic preparation of the lip. Place the dog in lateral recumbency and prepare the regional skin with povidone–iodine scrub and daub the buccal mucosa with povidone–iodine solution. Take extra care using chlorhexidine antiseptics in or around the face, because unintentional contact will result in severe chemosis and conjunctivitis. For unilateral procedures, stabilize the head and drape the lip region without altering the normal relaxed lip position or the ability to fully open the mouth during the procedure. For bilateral procedures, position the head with the nose pointed up, so both sides of the lip margins are accessible.

Surgical technique

The skin surrounding the commissure is clipped routinely and prepared with povidone–iodine antiseptic scrub, and the oral cavity is rinsed with 0.12% chlorhexidine gluconate solution or povidone–iodine solution. Identify and mark the maxillary labia margin adjacent to the maxillary second premolar tooth. Create a full-thickness excision of the lip margin to the commissure with Mayo scissors or scalpel blade. Alternatively, to help preserve lip height, the author sometimes prefers to create a sagittal split of the lip (without removing lip margin) with a # 15 scalpel blade inserted to the depth of approximately 7.5 mm. This effectively separates the skin from the oral mucosa to enable a two-layer closure. In this illustrated series (Fig. 56.9), the lip margin is excised. Essentially, the lip skin, underlying subcutaneous tissue, and mucosa are exposed, then sutured in separate layers to create a permanent adhesion of the commissure. If bilateral commissurorraphy is planned, the procedure is repeated on the opposite side commissure.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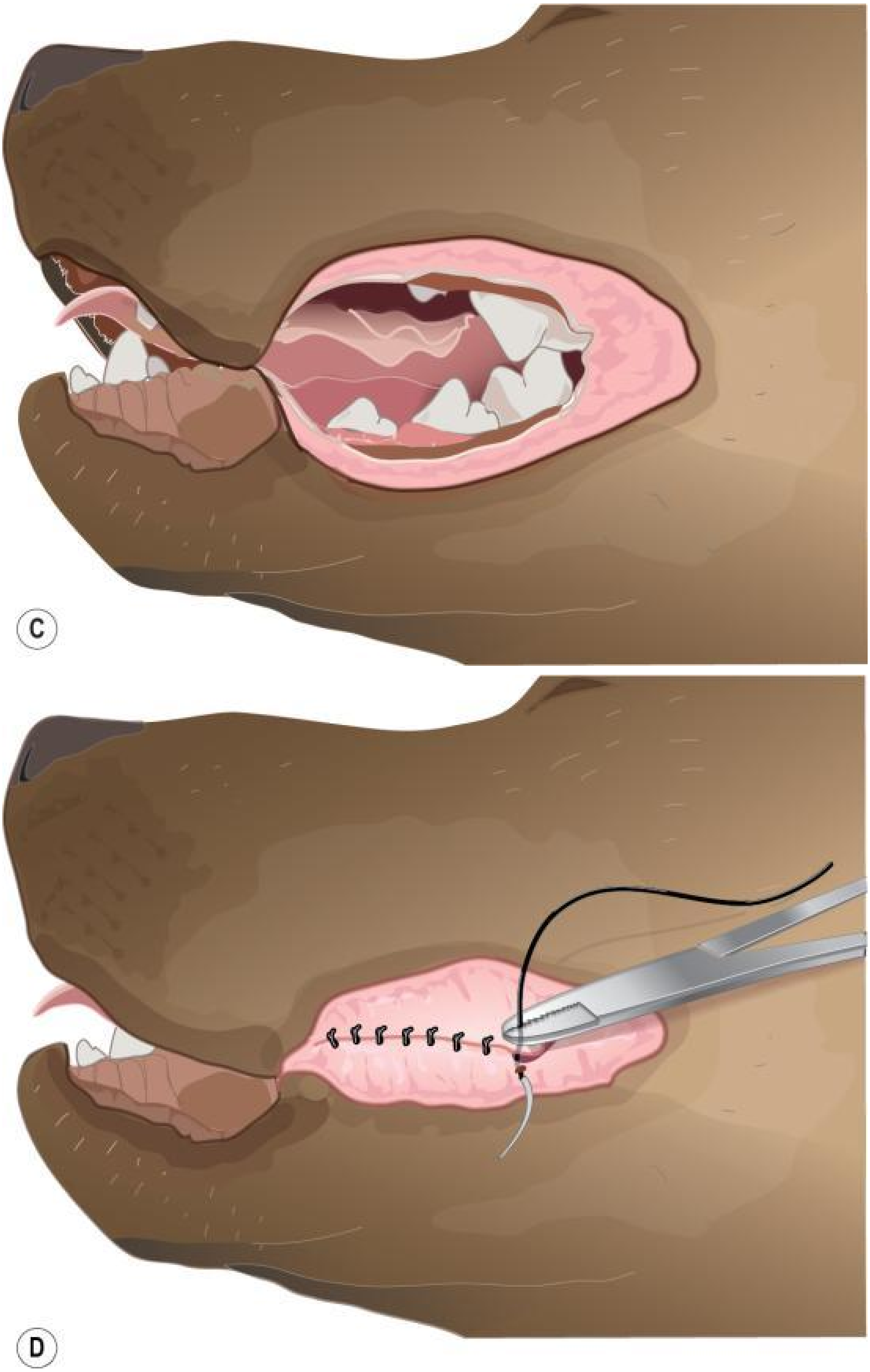
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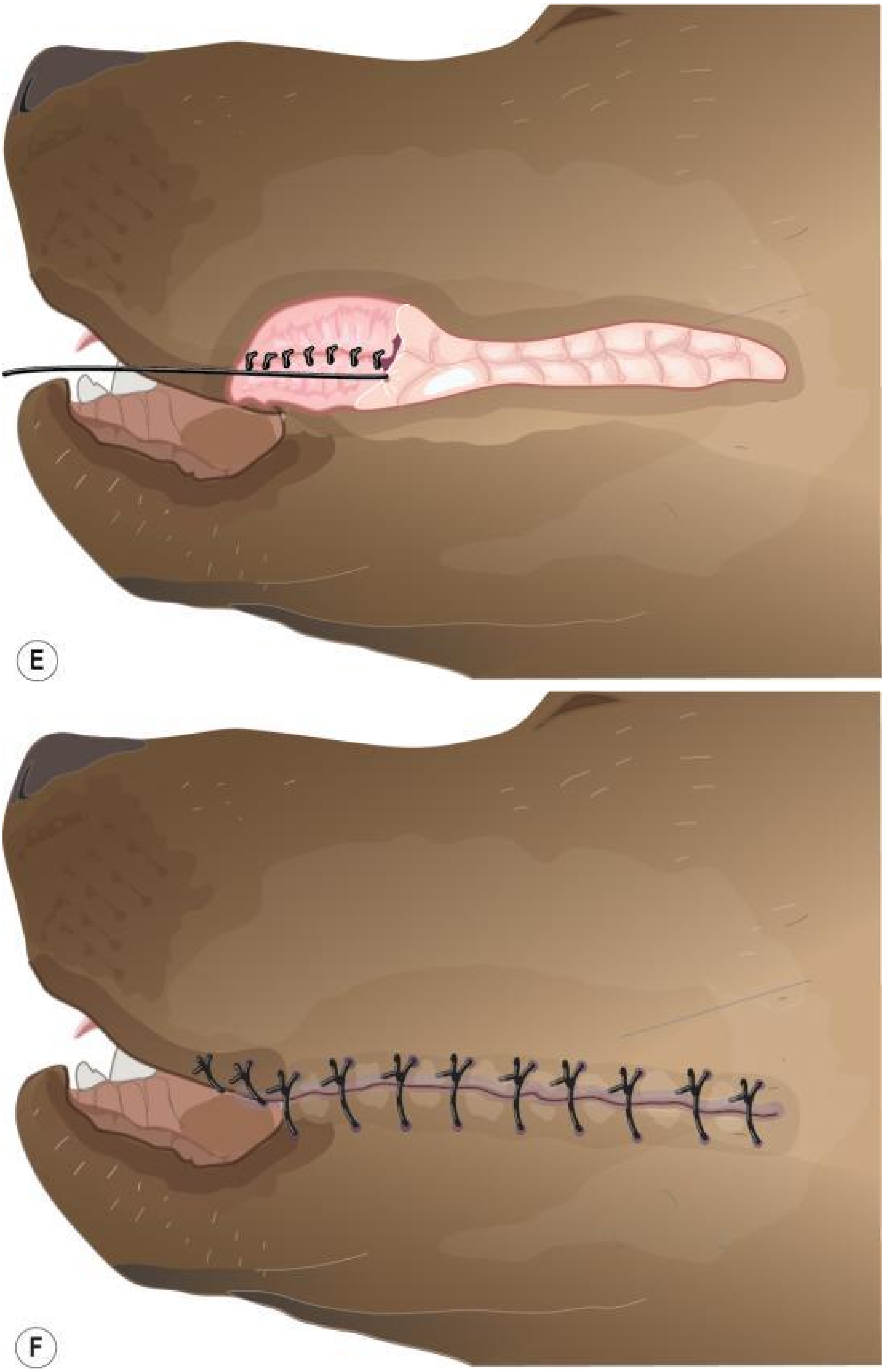

• **FIG. 56.9** Illustration of commissurorraphy technique. (**A**) An incision is made through the margin of the lip at the level of the second maxillary premolar tooth, and excision of the lip margin is continued caudally to the commissure. (**B**) The lip margin excision is continued to include the mandibular labia to the mandibular lip margin adjacent to the mandibular second premolar tooth. (**C**) Rostral edges of the maxillary and mandibular incisions are first aligned with mattress sutures. The author also uses one or two stent sutures (mattress sutures using surgical buttons or plastic tubing stents) placed just rostral to the rostral lip excision margins to hold the upper and lower lip margins together and release tension during mouth opening. (**D**) Appose oral mucosa with monofilament 4-0 poliglecaprone 25 suture in a simple interrupted (shown) or continuous pattern. (**E**) Appose the soft tissue between the oral mucosa and skin with similar suture in a simple continuous pattern. (**F**) The skin is sutured with 4-0 nonabsorbable monofilament suture material in a simple interrupted pattern.

Postoperative considerations

Patients are usually released from the hospital the same day and should be fed a soft food diet after surgery. Irrigation of the oral vestibules after meals with water-filled syringes will remove food debris. Clients should not permit chewable toys or fetching for 2 months after surgery. A short Elizabethan collar should remain on the patient until suture removal to prevent self-mutilation. Sutures are removed and recheck is performed 2 weeks after surgery (Fig 56.10). Sutures used for tension-relieving stents or buttons (Fig. 56.11) may be left in place for up to 3 weeks after surgery to help protect the rostral commissurorraphy from dehiscing.

• FIG. 56.10 Two-week postoperative appearance of a dog showing commissurorraphy performed for a left-sided unilateral total mandibulectomy. Note the majority of the tongue is held within the oral cavity.

• FIG 56.11 Use of surgical buttons to relieve tension on the rostral part of a right commissurorraphy. Source: (From Tobias KM, Johnston SA, eds. *Veterinary Surgery: Small Animal*. 2nd ed. St. Louis: Elsevier Saunders, 2018, with permission.)

Complications and prognosis

The most common complication after this procedure is dehiscence of the rostral margin of the commissurorraphy due to tension. Long-term complications are usually minor and include reduced visibility for oral intubation[4] and restricted access to the caudal dentition for home care by the owners or for future dental procedures.

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CHAPTER 57

Inferior labial frenoplasty and tight-lip syndrome

Steven E. Holmstrom, Peter P. Emily

Definitions

Inferior labial frenulum (or frenum): A fibroelastic band of tissue attaching the lower lip to the gingiva on the distal aspect of the mandibular canine tooth.

Frenectomy: Excision of the frenulum.

Frenotomy: Incision or transection of the frenulum.

Frenoplasty: Correction of an abnormally attached frenulum by surgically repositioning it.

Tight-lip syndrome: A condition seen exclusively in Shar-Pei dogs whereby the lower lips curl lingually and over the mandibular incisor teeth and occasionally the mandibular canine teeth.

Vestibule: The space external to the teeth, gingiva, and alveolar mucosa and internal to the lips and cheeks.

Preoperative concerns

Frenoplasty and tight-lip correction are performed to treat patients for an anatomical defect that is likely of genetic origin. To prevent the retention of these traits in the gene pool, animals who require tight-lip correction or frenoplasty should be spayed or neutered. Clients should be informed that many organizations prohibit showing of animals that have had any type of corrective surgery.

Surgical anatomy

A general description of the anatomy of the lips can be found in Chapter 49.

The inferior labial frenulum is a poorly described structure, consisting of a fibroelastic band of tissue attaching the lower lip to the gingiva on the distal aspect of the mandibular canine tooth. It contains the neurovascular bundle, including the middle mental nerve and blood vessels, emerging from the middle mental foramen. The middle mental foramen is located ventral to the mesial root of the second premolar tooth, while the smaller rostral mental foramen and caudal mental foramen can be found ventral to the second incisor and third premolar teeth, respectively. The skin of the chin is tightly attached to the underlying tissues. The structure of the lower lips has not been described in depth. The margin of the lower lip as far caudally as the canine tooth is hairless over a width of approximately 5 mm. Caudal to the canine teeth the hairless zone increases to 10 mm in width, and conical papillae occur.[1] A well-keratinized area is present where the maxillary canine tooth comes in contact with the lower lip when the mouth is in a fully closed position.

The Shar-Pei is a dog breed of Chinese origin; the literal translation of Shar-Pei is "sand skin." The breed is thought to have been in existence since the Han dynasty, around 200 BCE. Their disposition can be standoffish and skittish toward strangers. Many people develop rashes on their forearms after contact with the Shar-Pei's rough, harsh skin. These dogs have loose skin covering the head and body, small ears, and unusual muzzle shape.

On the rostral aspect of the lower lip there is normally a space (the vestibule) between the lip and the teeth and gingiva. This space is reduced or missing in affected Shar-Pei dogs.

Inferior labial frenoplasty

Clinical presentation

In some dogs, an excessively tight inferior labial frenulum may be present, and the abnormally narrow vestibulum thus created traps food and debris. This results in infection of the periodontium at the distobuccal surface of the mandibular canine teeth. In addition to periodontal disease, this condition may lead to inflammation and infection of the lower lip (cheilitis) lateral and rostral to the inferior labial frenulum.

Therapeutic decision-making

There are three indications for frenoplasty. Patients showing dermatological signs of chronic infection and inflammation of the lower lip are initially treated by medical means; if unsuccessful, they may benefit from frenoplasty by exteriorizing the lip out of the moist environment of the oral cavity. Patients with chronic periodontal disease of the distal portion of the mandibular canine teeth may also benefit from frenoplasty, in combination with conventional periodontal treatment and home care. Finally, inferior labial frenoplasty may be performed as an ancillary procedure in the Shar-Pei dog with tight-lip syndrome (Fig. 57.1).

• **FIG. 57.1** Typical appearance of a tight-lipped Shar-Pei. Note that the lower lip is rolled up and over the mandibular incisor teeth. This causes the patient to bite its lip.

Frenectomy (excision of the frenulum) or simple frenotomy (incision or transection of the frenulum) is rarely, if ever, indicated.

Surgical technique

The lower lip at the attachment of the frenulum is grasped with thumb forceps and held taut. A scalpel is used to cut the frenulum midway between the lip and gingiva in a sagittal direction. The incision is extended with sharp or blunt dissection into the frenulum to relieve the pull of the muscular attachments. Care is taken not to sever the middle mental nerve and blood vessels. The lip will relax laterally when the attachments have been completely severed, and a diamond-shaped cut will be created. The objective of suturing is to prevent the natural adhesion and reattachment of the cut portions of the frenulum. This is accomplished by placing several simple interrupted, absorbable sutures in a transverse direction, bringing the rostral and caudal edges of the incision together, similar to a Heineke-Mikulicz procedure in gastrointestinal surgery.

Postoperative care and assessment

To keep the area clean, the oral cavity and surgical area should be irrigated twice daily for 2 weeks with a 0.05%–0.12% chlorhexidine gluconate solution.

Complications

Hemorrhage may be caused by the inadvertent transection of the mental artery or vein; reattachment of the frenulum may occur if the wound is left unsutured. Chronic cheilitis and localized periodontitis affecting the mandibular canine teeth may persist. Wound dehiscence is uncommon and is generally treated conservatively by wound irrigation, allowing the wound to heal by second intention; further surgical revision may be required if this again results in an abnormally tight frenulum.

Tight-lip correction in the Shar-Pei

Clinical presentation

Because of the reduced space between the lower lip, rostrally, and corresponding teeth and gingiva in affected Shar-Pei dogs, the lower lip can curl up and over the mandibular incisor teeth and sometimes the canine teeth. The lip is traumatized, being crushed between the mandibular and maxillary incisor teeth. Further scarring and contracture aggravate this condition. Patients will have their mandibular lips tightly pressed against, or covering, the incisor teeth (and possibly the canine teeth as well). These teeth may be displaced lingually and predisposed to early periodontal disease. The client may report reluctance to eat, odor, or hemorrhage. The patient may have skin and oral infections and displaced or rotated teeth.

Therapeutic decision-making

Surgical repositioning of the caudoverted rostral portion of the lip off the incisive edges of the incisor teeth reduces the tension between the lip and teeth and increases the depth of the vestibule.

The age of the patient is an important consideration. In young animals, vestibuloplasty is performed to prevent pain and to allow proper growth of the jaw. One technique is to make a releasing incision that separates the lip from its gingival attachment and have the client manipulate the incision daily to prevent the skin from being pulled back to its previous position.[2] An alternative to this would be to increase the space in the vestibule[3-6] by insertion of either an oral mucosa graft or Penrose tubing.

For older patients, where the patient is biting on its lip causing pain and infection (Fig. 57.1), vestibuloplasty is also indicated. In addition, two other techniques are available, which may be performed concurrently. Frenuloplasty can be performed concurrently with tight-lip correction by either vestibuloplasty or the ventral mandibular approach. One technique involves a ventral mandibular incision, freeing of the attachment of the skin/gingiva to the mandible and suturing the gingiva to the mandible so that the lip is no longer in occlusion with the teeth.[4] In patients with excess skin on the chin or when the two previously described techniques are unsuccessful, skin of the chin may be excised.[1]

Antibiotic prophylaxis (see Chapter 3) is generally indicated for these procedures.

Surgical techniques

Vestibuloplasty

The objective of the vestibuloplasty procedure is to increase the amount of vestibule present by separating the lower lip from the chin.

The patient is placed in ventral recumbency with a maxillary support bar (Fig. 57.2A), and the lower lip is retracted to expose the mandibular teeth. A mucosal releasing incision is made at the mucogingival or mucocutaneous junction (Fig. 57.2B, C). The incision should extend from, and include, one inferior labial frenulum to the other (Fig. 57.2D, E). The subcuticular tissues between the skin and mandible are dissected ventrally (Fig. 57.2F). The mucosal flap is secured in position first by a periosteal fenestration made from frenulum to frenulum at right angles to the mandible, in order to prevent reattachment of the lip to its original position. The flap is then repositioned against the prepared mandibular surface, and the ventral portion of the incision is sutured to the mandible (Fig. 57.2G, H), establishing a new, deeper vestibule, in order to prevent reattachment of the mandible to its original position. The lip is inspected to make sure that it is level to the incisal plane and modified if not. The alternative to suturing is to use cyanoacrylate tissue glue. The tissue glue can also be painted on the surgical site for retention of the mucosal flap and to act as a tissue dressing. Initially the lip will hang down as if degloved. Surgery should result in permanent resolution of the condition (Fig. 57.3).


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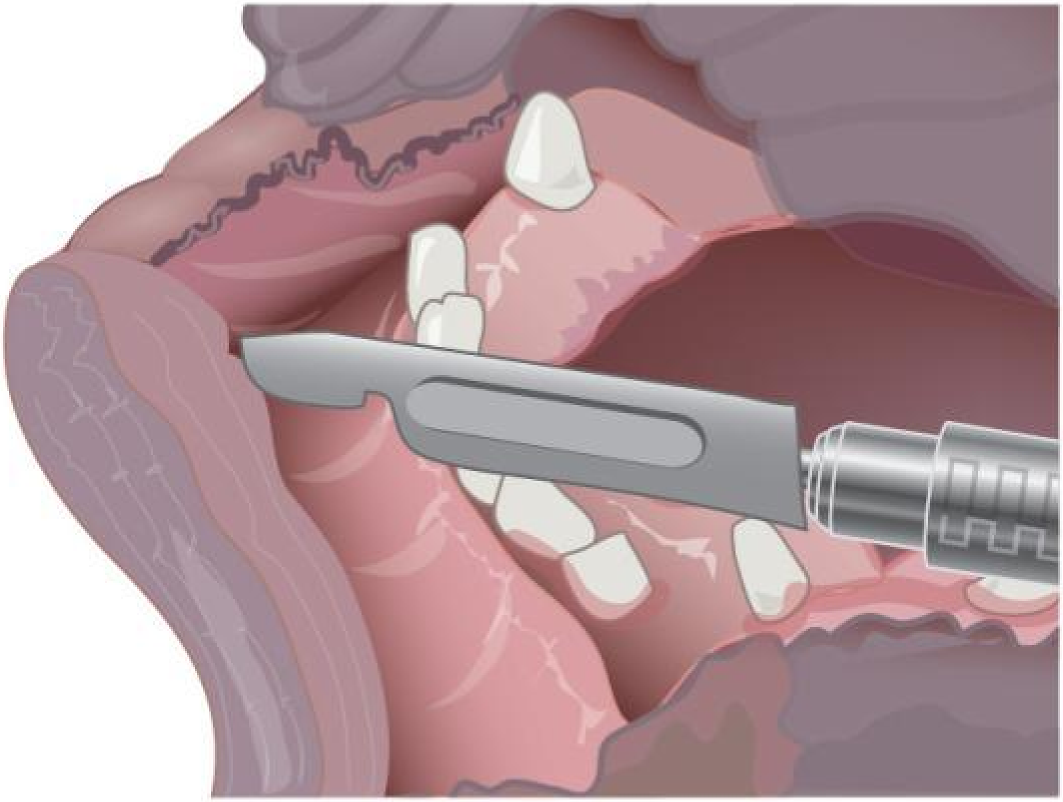

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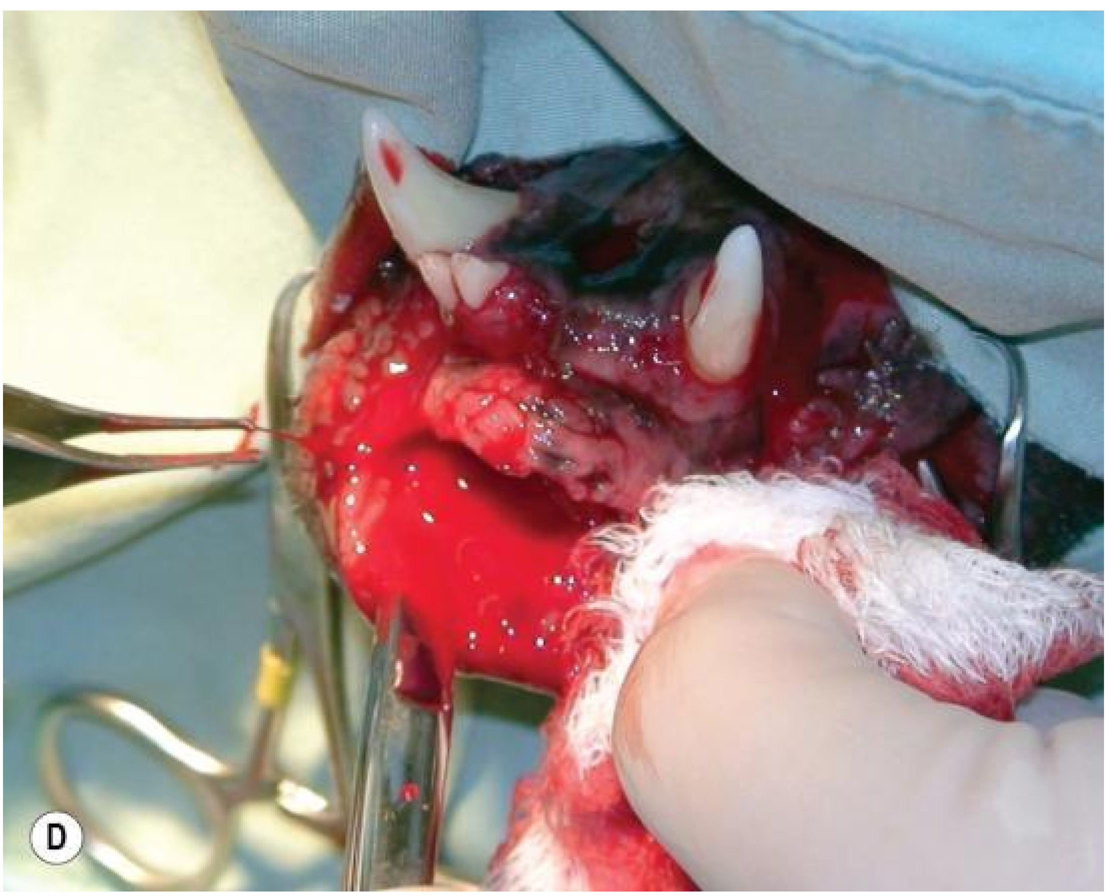

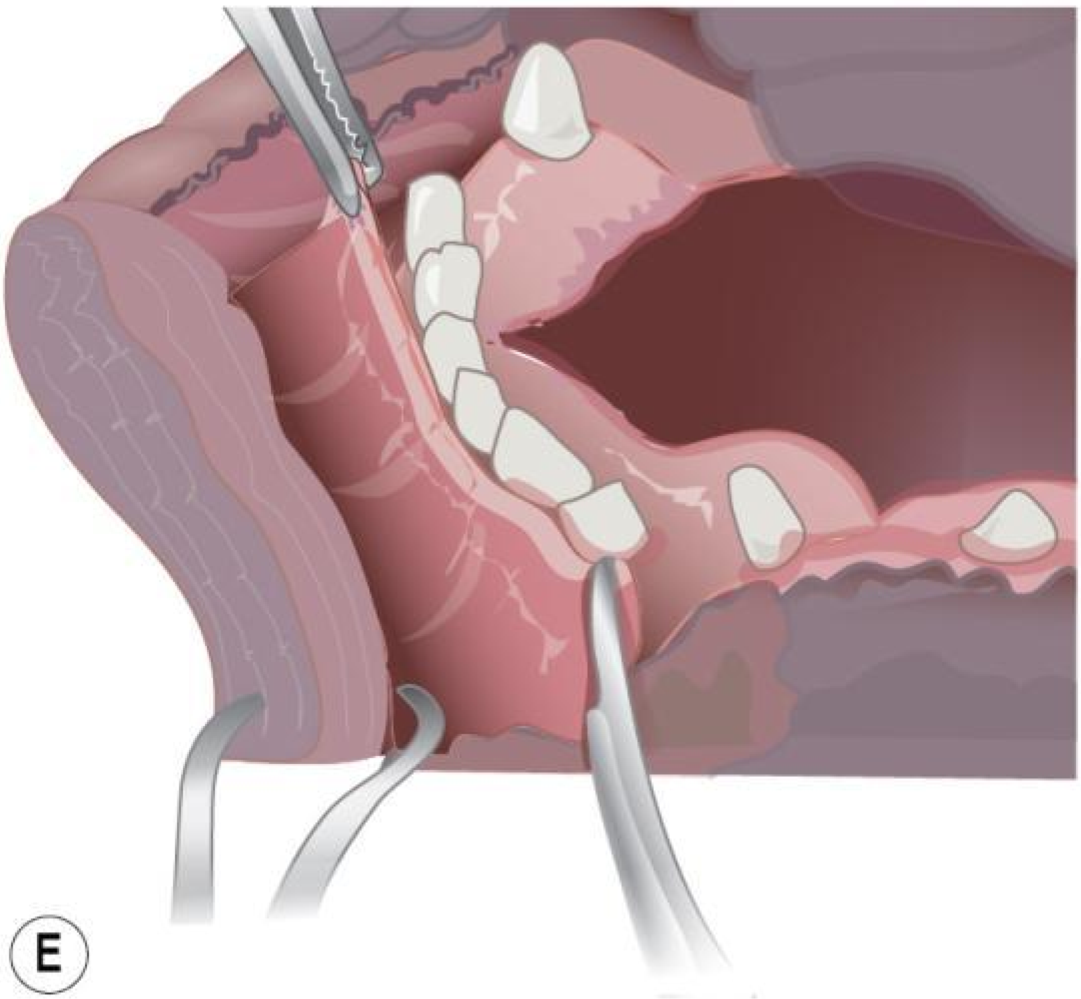
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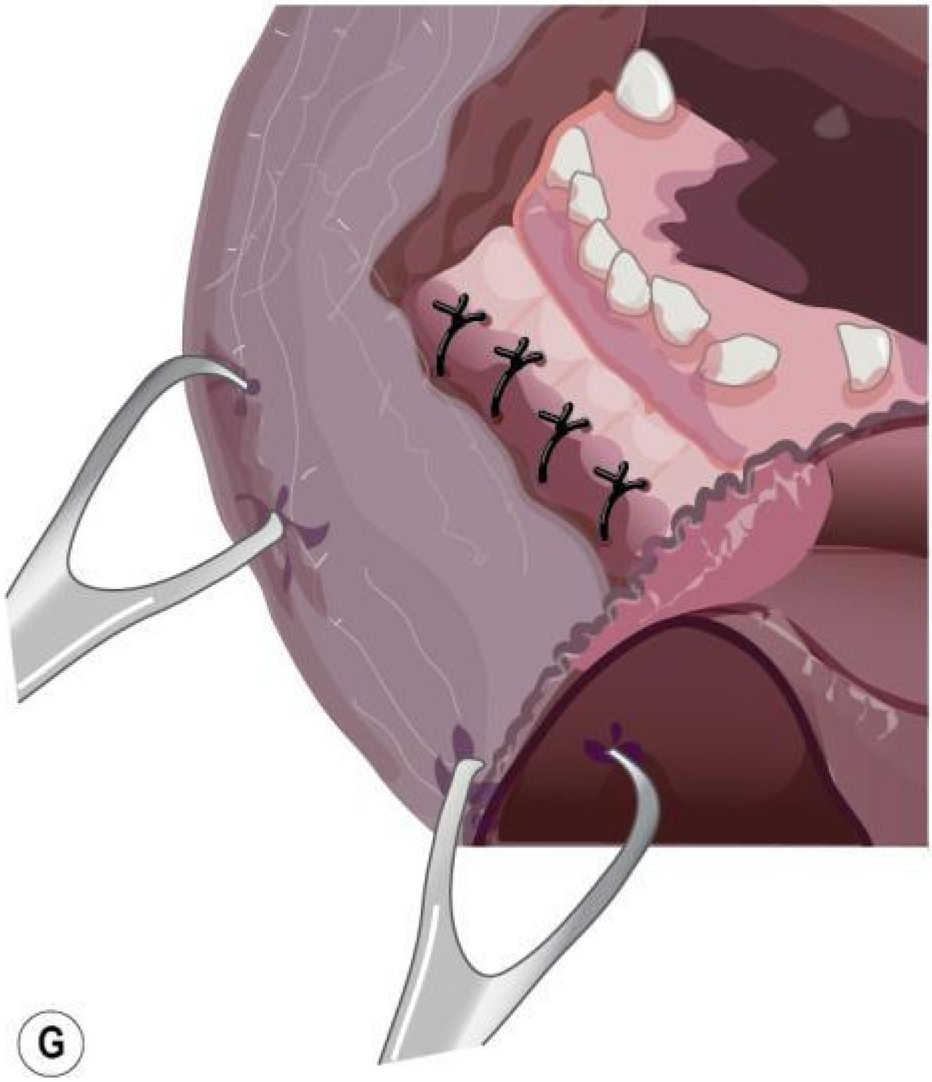

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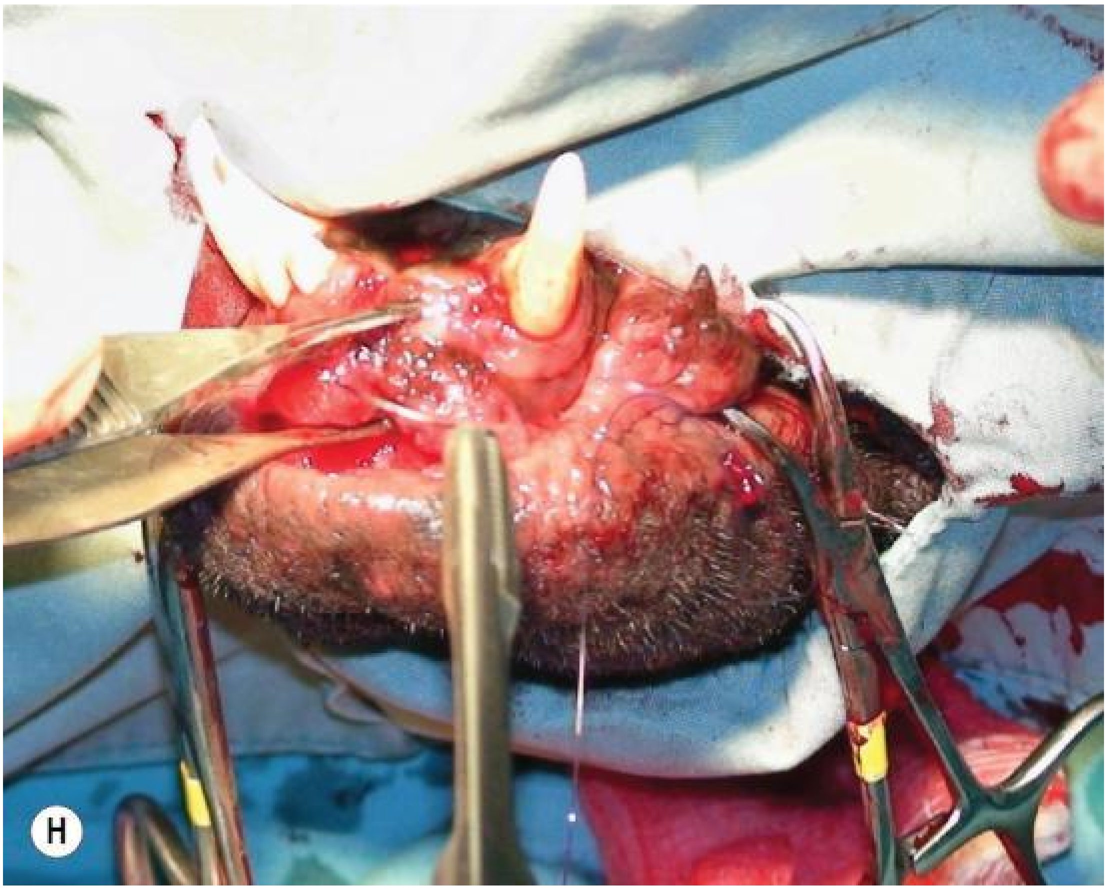

• **FIG. 57.2** (**A**) The patient is positioned in sternal recumbency with a maxillary support bar. (**B, C**) The initial incision is made along the mucocutaneous junction. (**D, E**) The subcuticular tissues are dissected off the mandible ventrally. (**F**) The tissue between the skin and mandible is dissected. (**G, H**) The subcuticular tissue is sutured to the mandible to form an enlarged vestibule.

• **FIG. 57.3** Photograph of a Shar-Pei 2 years after vestibuloplasty. Source: (Photograph courtesy of Dr. Edward R. Eisner)

Ventral mandibular approach

The objective of this approach is to free up the skin covering the mandible and tack the skin so that the lip is pulled ventrally.

An approximately 20-mm incision is made over the mandibular symphysis in a craniocaudal direction. This allows the insertion of sharp-sharp scissors to undermine the skin from the muscle. A suture of 2-0 or 3-0 polydioxanone is passed as far dorsally as possible toward the lip, taking an anchoring bite of subcuticular tissue in order to pull the lip ventrally. The suture is tacked down to the periosteum as far caudally as possible on the mandibular symphysis. The suture is tightened and tied with a reinforced surgeon's knot. Additional sutures, laterally to the right and left, are placed as necessary in order to accomplish the goal of repositioning the skin tissue effectively and cosmetically. The skin is sutured with 4-0 or 5-0 nonabsorbable monofilament suture material.

Skin excision

The objective of this technique is to excise excessive skin on the chin.

The patient is placed in dorsal recumbency. The chin tissues are pinched to approximate the position of the skin when the tissue is excised. It is helpful to use a sterile permanent marking pen to mark the planned incision. If a sterile marking pen is not available, the planned incisions can be marked prior to aseptic preparation. The incision should be symmetrical. A fusiform incision through the skin and subcutaneous tissue is made and the redundant lip tissue is undermined and excised using Metzenbaum scissors. The subcutaneous tissues are sutured with one to three layers of 3-0 absorbable suture material in a continuous pattern. Skin closure is routine, using either an intradermal pattern with absorbable suture material or meticulously placed single-interrupted sutures using 4-0 or 5-0, nonresorbable material in order to avoid scar formation.

Postoperative care and assessment

Vestibuloplasty

The oral cavity and surgical area should be irrigated twice daily with a 0.05%–1.12% chlorhexidine gluconate solution to keep the area clean for 2 weeks. If a Penrose drain is used, it should be removed in 2–3 weeks.

Ventral mandibular approach

The client is advised not to give hard treats or chew toys (soft or hard) or to allow oral play for 3 weeks. Recommendations for daily home oral hygiene are routine. The patient is rechecked in 1 week and the skin sutures are removed in 10 to 14 days.

Skin excision

The patient is rechecked in 1 week and skin sutures are removed in 10 to 14 days.

Complications

Vestibuloplasty

Incomplete fenestration can allow the tissues to join back together in the tight-lip position. An Elizabethan collar may be necessary to prevent self-mutilation and premature dislodgment of the Penrose drain.

Ventral mandibular approach

Breakdown of suture may occur if a sufficient anchor for the suture is not taken or if the patient is not restricted from self-destructive oral behavior. Wound infection may also occur if aseptic technique was not used.

Skin excision

Wound infection and rubbing at the incision with consequent dehiscence are two potential complications of which clients should be made aware.

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CHAPTER 58

Management of maxillofacial osteonecrosis

Ana Nemec, Milinda J. Lommer, Sandra Manfra Marretta

Definitions

Bone sequestrum: A piece of necrotic bone that has become separated from vital bone.
Osteomyelitis: An inflammatory process of the bone marrow, cortex, and possibly the periosteum.
Osteonecrosis of the jaw (ONJ): Exposed nonvital bone in the maxillofacial region that fails to heal after 6 to 8 weeks in patients with no history of maxillofacial radiation.[1]
Osteoradionecrosis of the jaw (ORNJ): ONJ in a previously irradiated field in the absence of persistent or recurrent tumor.[2]
Bisphosphonate/medication-related osteonecrosis of the jaw (BRONJ/MRONJ): ONJ in a patient treated with bisphosphonate or other antiresorptive or antiangiogenic medication that had not had radiation therapy to the craniofacial region.[3-5]

Preoperative concerns

Osteonecrosis is an infrequently diagnosed condition in the maxillofacial region of dogs and cats, which has a significant impact in affected patients. Recognition of maxillofacial osteonecrosis includes evaluation of pertinent clinical history and thorough preoperative evaluation of the patient. Animals with osteonecrosis may be presented with a history of halitosis, severe oral pain, facial swelling, drooling, draining tracts, severe ulcerative mucositis, regional lymphadenopathy, fever, lethargy, reluctance or inability to eat, weight loss, severe purulent nasal discharge, or exophthalmos.[6-8] Preoperative concerns in animals with maxillofacial osteonecrosis include medical stabilization of the patient prior to surgical intervention and evaluation of underlying conditions that may be contributing to the osteonecrosis.

Etiopathogenesis and risk factors

Several causes for osteonecrosis have been identified in humans, including primarily administration of certain systemic medications (bisphosphonates, denosumab, and antiangiogenic medications), (iatrogenic) trauma, radiation therapy, and (dental) infections.[5,9,10] There are also some idiopathic cases, in which the underlying cause is never identified.[9] Similar ONJ has been observed in animals, and in the absence of previous radiation therapy or trauma, it is likely that the pathogenesis of osteonecrosis is multifactorial. The development of nontraumatic osteonecrosis appears to involve vascular compromise, bone and cell death, and/or defective bone repair.[11] Regional endothelial cell dysfunction may be the underlying mechanism, uniting the multiple associated risk factors with a single common pathway.[12]

Dogs of any age, sex, size, and breed can be affected with ONJ,[8] but Scottish terriers may be particularly prone to ONJ, which seems to be associated with chronic stomatitis and periodontitis.[6,8] Recent dental extractions and oral antibiotic use to treat oral/dental disease are commonly reported in dogs with ONJ.[8] Cryptococcal infection has also been described as a possible infectious cause of nontraumatic ONJ in dogs,[13] but other infectious agents should also be considered (Fig. 58.1).

• **FIG. 58.1** (**A**) This beagle was presented with painful rostral maxillary swelling of unknown duration. (**B**) Intraoral examination revealed necrotic bone in the region of the canine tooth with loss of normal mucosal integrity. (**C**) Severe rostral maxillary bone lysis was evident on a maxillary occlusal radiograph. (**D**) An impression smear of an incisional biopsy of the abnormal mucosa revealed budding organisms consistent with blastomycosis.

Osteoradionecrosis (ORNJ) is a possible, although uncommon, long-term complication of radiation therapy (Fig. 58.2). The exact pathogenesis of ORNJ has yet to be determined, but radiation-induced microvascular compromise, inflammation and activation of fibroblasts and imbalance in bone metabolism resulting in bone damage,[14,15] and radiation-induced damage of the microstructure of the oral epithelial cells[16] have been suggested as possible mechanisms in humans. Osteoradionecrosis can develop 3 to 44 months after radiation therapy in dogs and can affect the site of tumor irradiation, the jaw closest to the irradiated mucosal tumor, and also contralateral jaw due to the exit radiation dose.[2] In humans, the risk for ORNJ development in any of these sites is considered to remain for the rest of the patient's life.[14] Osteoradionecrosis is believed to occur in humans above a certain threshold radiation dose and in association with several other risk factors.[2,14,17-19] Similar to humans, surgical procedures or dental extractions in the previously irradiated field are commonly cited as a likely risk of ORNJ development in dogs.[2,20] Although any necessary oral surgery is therefore advised prior to radiation therapy, a recent report in humans suggests that scaling and chlorhexidine use within 2 weeks before radiation therapy is significantly related to development of ORNJ.[21] If oral surgery is needed after radiation therapy in human patients, protocols are proposed for management of these patients; these include chlorhexidine mouth washes, perioperative antibiotics, and treatment as atraumatic as possible performed by experienced clinicians.[22]

• **FIG. 58.2** Osteoradionecrosis in an 11-year-old dachshund that was treated with radiation therapy for nasal squamous cell carcinoma. (**A**) Note the loss of oral mucosa and exposed, necrotic bone. (**B**) A large buccal mucoperiosteal flap has been created. (**C**) Following removal of the underlying necrotic bone and the maxillary first premolar tooth, the buccal mucoperiosteal flap was sutured without tension to close the oronasal fistula. (**D**) Two months postoperatively, extension of the osteoradionecrosis and recurrence of oronasal fistulas at the rostral and caudal aspects of the previous surgical site were evident.

MRONJ seems likely to become a significant problem in companion animals. Bisphosphonate use in veterinary medicine is increasing, especially in treatment of different skeletal conditions.[7,23] Bisphosphonates significantly reduce bone turnover and likely predispose the mandible to necrosis, although even with long-term use of bisphosphonates in dogs, oral exposure of bone may not occur.[24] In humans, dental extraction is a known risk for MRONJ development.[10] Bisphosphonate use (either alone or in combination with corticosteroids) has also been described to result in compromised osseous healing after extractions, profound periosteal reaction, and development of ONJ in some, but not all, dogs. This may be related either to a direct toxic effect of bisphosphonates on bone cells or to the suppressed remodeling rate of the bone, but the effects do not seem to be related to the duration of treatment with bisphosphonates.[4,25-27]

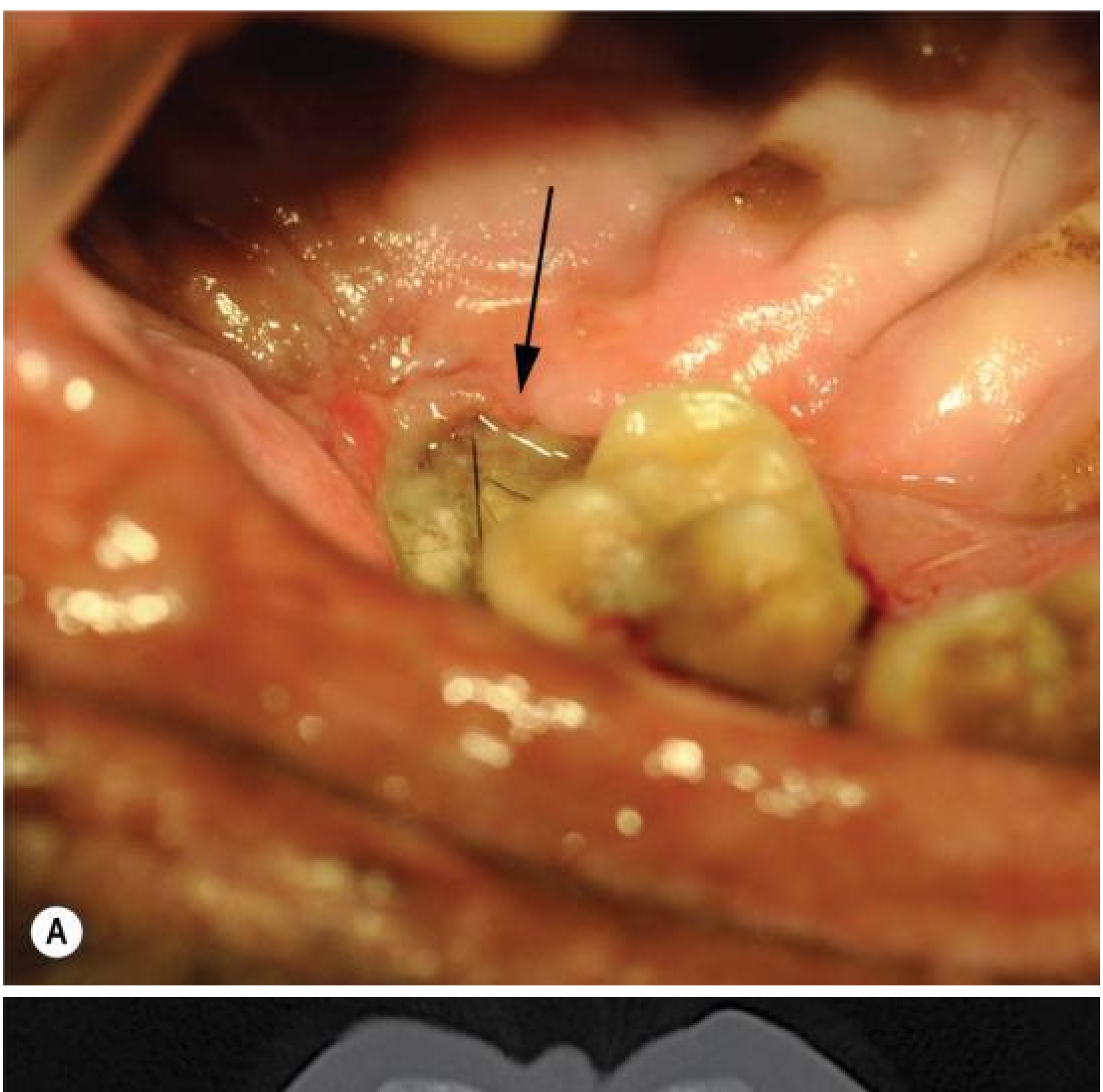
Therapeutic decision-making

Therapeutic decision-making in animals with maxillofacial osteonecrosis is mostly dependent on the etiology, location, and extent of the osteonecrosis. Accurate preoperative assessment and adherence to basic principles in the management of maxillofacial osteonecrosis can help ensure a successful outcome in most cases (depending on the initial cause of the maxillofacial osteonecrosis).

A complete preoperative evaluation of animals with maxillofacial osteonecrosis includes a full history, complete general physical examination on the awake patient, a thorough oral examination on the anesthetized patient, hematologic testing, cytologic/histopathologic examination of the lesion, and diagnostic imaging evaluation including dental and skull radiographs or, preferably, computed tomography (CT) of the head. While criteria for diagnosing and staging of ORNJ[28] and MRONJ[29] have been set in humans, these have yet to be determined in animals.

Hematological and serum biochemistry findings are usually unremarkable in dogs with ONJ or show only mild changes related to chronic inflammation.[8]

Detailed oral examination reveals plaque- and debris-covered exposed necrotic jawbone. Alveolar bone is always affected, but changes commonly also involve the jawbone proper, and variable amounts of granulation tissue may cover the defect. Clinical evaluation often underestimates the extent of the lesions[2,8] (Fig. 58.3).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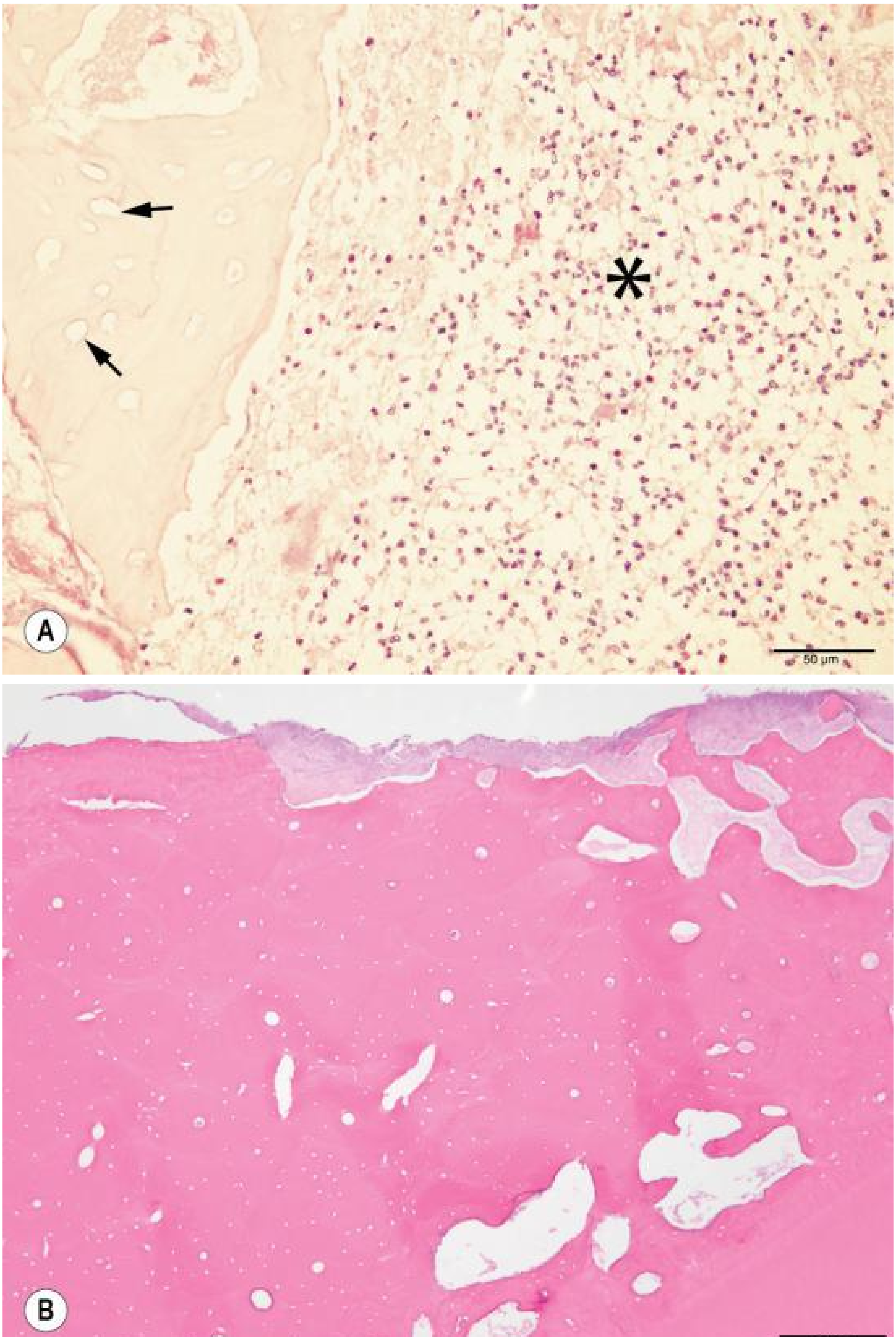
• **FIG. 58.3** (**A**) An osteonecrosis of the jaw site located in the caudal left maxilla *(black arrow)*. Note the missing left maxillary second molar tooth, as well as the plaque and debris covering the exposed necrotic bone. (**B**) An axial slice computed tomography image of the skull reveals an extensive lesion with a large sequestrum at the left caudal maxilla *(white arrows)* that extends into the maxillary recess *(white star)*. Note the associated periosteal reaction *(black arrows)* and total loss of attachment at one of the buccal roots of the left maxillary first molar tooth *(black arrowhead)*. Source: (From Peralta S, Arzi B, Nemec A, et al. Non-radiation-related osteonecrosis of the jaws in dogs: 14 cases (1996–2014). *Front Vet Sci*. 2015;2:7.)

Radiographic signs are usually similar in osteomyelitis, ONJ, ORNJ, and MRONJ. In humans, radiographic signs may even be absent, especially in early-stage lesions.[28] In dogs, osteolytic areas in ONJ and ORNJ cases are usually described to have a moth-eaten or permeative pattern of bone loss.[2,8] The bone may also appear more radiopaque and sclerotic, the definition of the mandibular canal may be lost, and there may be periosteal new bone formation (solid type periosteal reaction).[2,8] Areas of necrosis may appear even more radiopaque than the surrounding bone and may exist as sequestra of varying sizes, surrounded by a radiolucent border. Periosteal reaction is usually more pronounced in ONJ cases with sequestration.[8] Abnormalities associated with teeth in or close to the ONJ lesion are also commonly seen on clinical examination and intraoral radiographs.[8] CT and magnetic resonance imaging are superior to conventional and intraoral radiography in evaluating osteonecrosis, especially of the maxillae[8] (Fig. 58.3) and should be performed as part of the diagnostic workup for patients with osteonecrosis whenever possible.

Biopsy of the lesion(s) is warranted, especially in suspected ORNJ cases, to rule out neoplasia.[2] In humans, osteomyelitis, ORNJ, and BRONJ are described to have a distinct histological picture,[30,31] while in dogs, histopathology usually reveals chronic osteomyelitis associated with bacteria[2,6-8]; the exact role of bacteria is currently unknown, especially in ORNJ and MRONJ cases. Histopathology may also reveal multifocal areas of necrotic bone, where lacunae void of osteoblasts are seen, separated by fibrosis and inflammation.[4,7] Samples obtained from the ORNJ lesions may also show changes associated with hypovascular and hypocellular/fibrotic tissue[2] (Fig. 58.4).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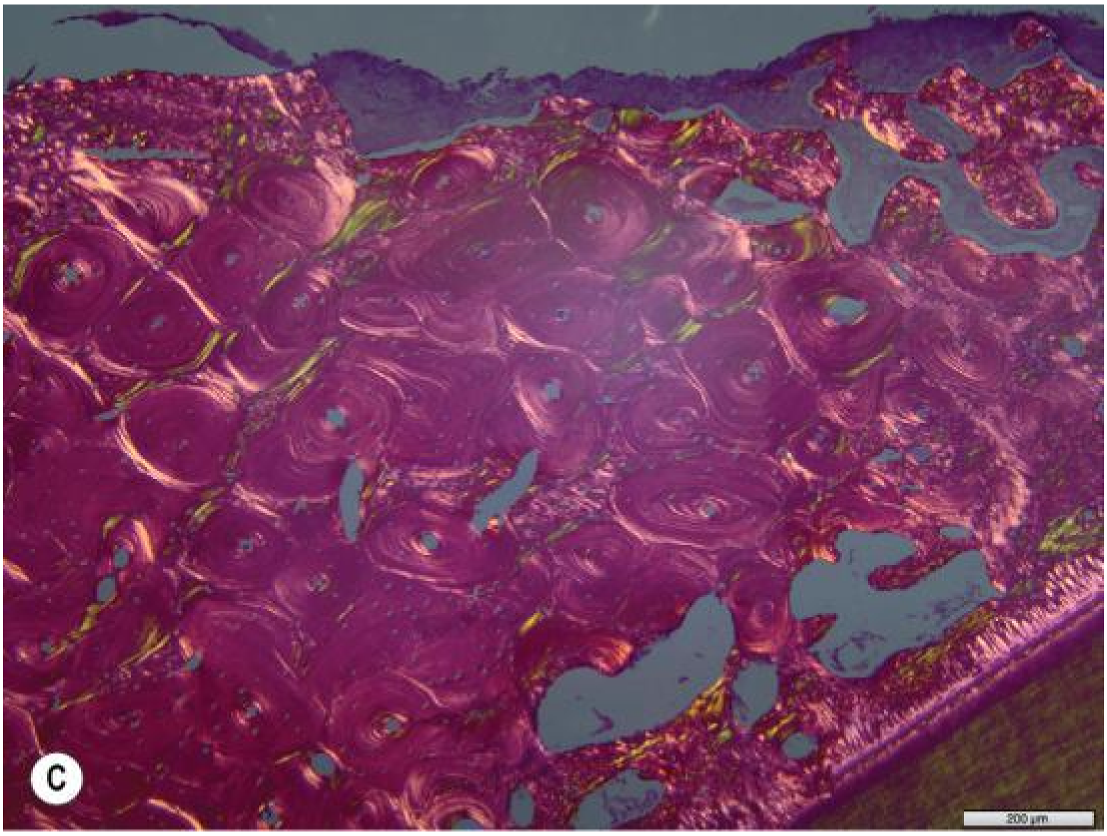


• **FIG. 58.4** (**A**) Histopathology of osteonecrosis of the jaw. The image shows necrotic bone characterized by empty lacunae *(arrows)* and surrounded by a bone marrow space infiltrated with inflammatory cells *(asterisk)*. These findings are consistent with osteomyelitis (H&E, ×40 magnification). Source: (From Peralta S, Arzi B, Nemec A, et al. Non-radiation-related osteonecrosis of the jaws in dogs: 14 cases (1996–2014). *Front Vet Sci.* 2015;2:7.) Regular (**B**) and polarized (**C**) histological image of the specimen from the dog with an osteoradionecrosis of the jaw lesion of the mandible. The alveolar bone comprises primarily lamellar, osteonal bone with limited interstitial woven bone. The bone is necrotic, as evidenced by diffuse, empty osteocyte lacunae, and attached basophilic to eosinophilic biofilm (top of image). Multifocal, irregularly spaced and shaped resorption bays are present within the alveolar bone (clear spaces/osteoporosis). Tooth dentin and a thin rim of cementum are evident at the bottom of the image. These eosinophilic matrices are more evident in the polarized image. A periodontal ligament is absent between the cementum and adjacent alveolar bone, consistent with ankylosis of the tooth. (From Nemec A, Arzi B, Hansen K, et al. Osteonecrosis of the jaws in dogs in previously irradiated fields: 13 cases (1989–2014). *Front Vet Sci.* 2015;2:5)

Biopsied tissue samples should also be submitted for culture and susceptibility[7]; although results usually reveal mixed microbial populations,[8] this information may be valuable for antibiotic selection.

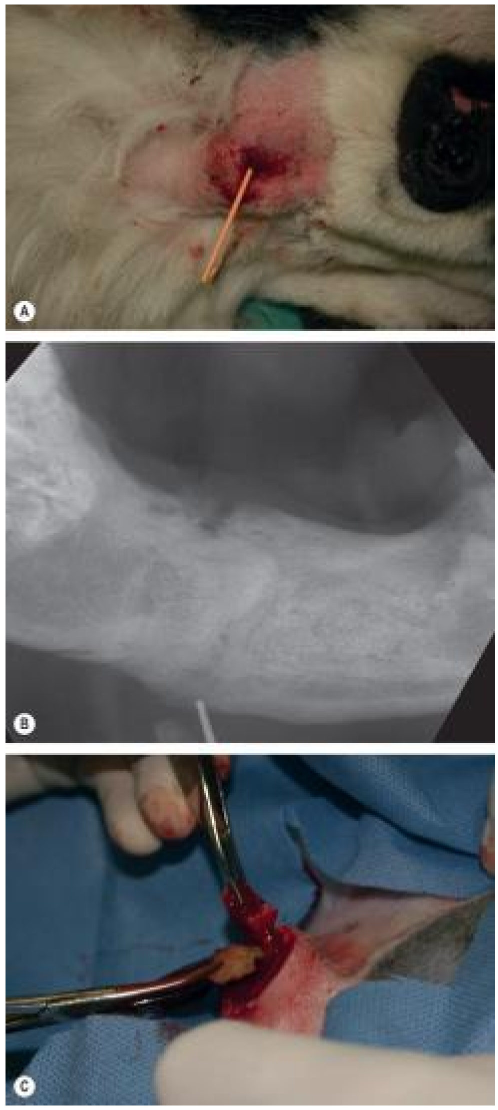
General principles in the surgical management of maxillofacial osteonecrosis

Surgical management is still the most common approach to treatment of ONJ in animals (Figs. 58.2 and 58.5). The initial step in the surgical management of maxillofacial osteonecrosis is the appropriate surgical approach, which typically involves the creation and elevation of a large mucoperiosteal flap surrounding the area of osteonecrosis. In most cases, either a draining tract (mucosal or cutaneous), cleft in the mucosa, or the complete separation of the mucosa from teeth with exposure of underlying necrotic bone is evident. An incision is made around the defect, thereby debriding the edges of the draining tract, cleft, or edges of the mucosa separated from the underlying necrotic bone. Any remaining teeth located in the necrotic bone are extracted. The underlying necrotic bone is removed either en bloc (if not attached to the surrounding vital bone) or debrided using either a round diamond bur on an air-driven high-speed handpiece, a bone-cutting tip on a piezoelectric surgical handpiece, or rongeur until all necrotic bone is removed. Alternatively, partial mandibulectomy or maxillectomy may be performed to remove all abnormal bone. Debridement is adequate when the remaining bone appears normal and bleeds readily. Intraoperatively, tissues are sampled and submitted for cytologic and histopathologic examination. The area is liberally flushed with sterile saline, and the mucoperiosteal flap is closed without tension in a simple interrupted pattern with monofilament absorbable suture material. Skin incisions, when necessary, are closed routinely with a nonabsorbable suture material in a simple-interrupted pattern.[6,8]

Factors to consider in the management of maxillofacial osteonecrosis

Factors to be considered in the management of patients with maxillofacial osteonecrosis include etiology, underlying contributory factors, location, and extent of the maxillofacial osteonecrosis.

Surgical debridement with a course of systemic antibiotics is considered a cornerstone of ONJ treatment, regardless of the inciting cause. Surgical approach, including sequestrectomy and dental extractions when indicated, with careful removal of the necrotic tissues until clinically healthy tissues are encountered, is based on clinical and diagnostic imaging findings. This is most frequently accomplished by creating a mucoperiosteal flap via an intraoral approach, but occasionally an incision through the skin may be necessary depending on the proximity of the sequestrum to the surface of the oral mucosa or skin (Fig. 58.5). En bloc resection of the ONJ site should be considered, if so indicated. Surgical site(s) should be closed with mucoperiosteal flaps. The potential for recurrence of ONJ exists, in either the same location or other sites in the mandible or maxilla, in patients previously treated for idiopathic maxillofacial osteonecrosis.[6,8]



• **FIG. 58.5** (**A**) In this 6-month-old dog that had sustained severe mandibular trauma from a dog bite at 8 weeks of age, a gutta-percha point has been placed in a cutaneous draining tract at the right caudal mandible. (**B**) Dental radiographs of the right mandible revealed a bone sequestrum ventral to the mandible and adjacent to the gutta-percha point. (**C**) An incision was made around the margins of the cutaneous draining tract, and the sequestrum was located and removed.

In ORNJ cases, the surgical treatment becomes more complicated, since the integrity of the surgical site is compromised. In humans, conservative/medical management (pentoxifylline, tocopherol, and doxycycline) has been suggested as a first-line treatment of ORNJ, especially of early-stage lesions.[32,33] Low-intensity ultrasound has been described as potentially useful in improving the healing of previously irradiated bone in dogs.[34] If conservative/medical management fails or surgery is considered the only viable option, the decision on the extent of bone removal is made intraoperatively, based on clinical assessment of bone vitality/osseous bleeding. Oral surgery in animals with osteoradionecrosis, including repair of oronasal fistulas and surgical extractions, may result in dehiscence of the surgical site and progressive extension of the osteonecrosis in spite of aggressive attempts at surgical and medical management (Fig. 58.2). Progression of ORNJ is relatively common in humans and dogs, despite complete removal of necrotic bone in advanced cases.[2,35] The persistence of local neoplasia with associated necrotic bone remains a possibility, as well as the potential for distant metastasis.[2] The value of hyperbaric oxygen use in the prevention and treatment of ORNJ remains controversial.[33]

In humans, surgical debridement of BRONJ/MRONJ lesions is controversial and is suggested as a treatment option only in extreme cases, as it may aggravate the condition. Prevention is considered most important and includes thorough oral/dental evaluation and necessary treatment before bisphosphonate or other medical treatment is initiated.[36] "Drug holidays" should also be considered,[23] especially if BRONJ/MRONJ lesions develop. In cases of BRONJ/MRONJ development, conservative and medical approaches to treatment are described in humans (e.g., use of platelet concentrates, bone morphogenetic proteins, and stem cells).[5,36] Recently, autologous bone marrow mesenchymal stem cells were successfully used as an intra-arterial infusion to treat experimentally induced osteonecrosis of the femoral head in dogs.[37]

Postoperative care and assessment

Broad-spectrum antibiotic therapy, such as amoxicillin-clavulanic acid, metronidazole, or clindamycin, is initiated until bacterial culture and susceptibility results are available. Antibiotic therapy based on the results of bacterial culture and susceptibility testing should be continued for a minimum of 6–8 weeks, together with the use of oral antiseptic rinse.[8] Antifungal therapy may be initiated as indicated.[13] Nonsteroidal antiinflammatory medications and other analgesics are also highly recommended to help manage postoperative swelling and pain.[6,8,13]

Complications

Complications associated with the surgical management of maxillofacial osteonecrosis include local recurrence and progression of the maxillofacial osteonecrosis in spite of aggressive surgical debridement or the development of osteonecrosis in other areas of the maxilla or mandible, especially in dogs presented for idiopathic maxillofacial osteonecrosis. Recurrence of oronasal fistulas from mucoperiosteal flap dehiscence is also a potential complication.

When the cause of the maxillofacial osteonecrosis is associated with trauma or traumatic surgical extractions, recurrence is less likely if all boney sequestra are removed during initial debridement.

When maxillofacial osteonecrosis occurs as a complication of radiation therapy or use of bisphosphonate or other antiresorptive or antiangiogenic medication, the risk of recurrence and progression of local osteonecrosis increases. In these cases, a conservative approach to management may be elected over a surgical approach, or several surgical interventions should be anticipated to control or slow down the process. Following surgical treatment of maxillary osteonecrosis, the incidence of dehiscence and recurrent oronasal fistula is higher in animals previously treated with radiation therapy for maxillary or nasal tumors. This may be associated with progressive radiation osteonecrosis, local tumor recurrence, or both.

Prognosis

The prognosis for animals presented with maxillofacial osteonecrosis largely depends on the initial cause and extent of the osteonecrosis. For cases in which the cause of the maxillofacial osteonecrosis is traumatic, the prognosis is generally excellent following appropriate surgical and medical management.

The prognosis in animals with idiopathic osteonecrosis is fair to good following appropriate surgical and medical management. For cancer patients with osteoradionecrosis, or patients with MRONJ, the prognosis is generally fair to guarded. Progressive disease or extensive lesions may severely impact the quality of life of the patient, with humane euthanasia remaining the only option in some cases.

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CHAPTER 59

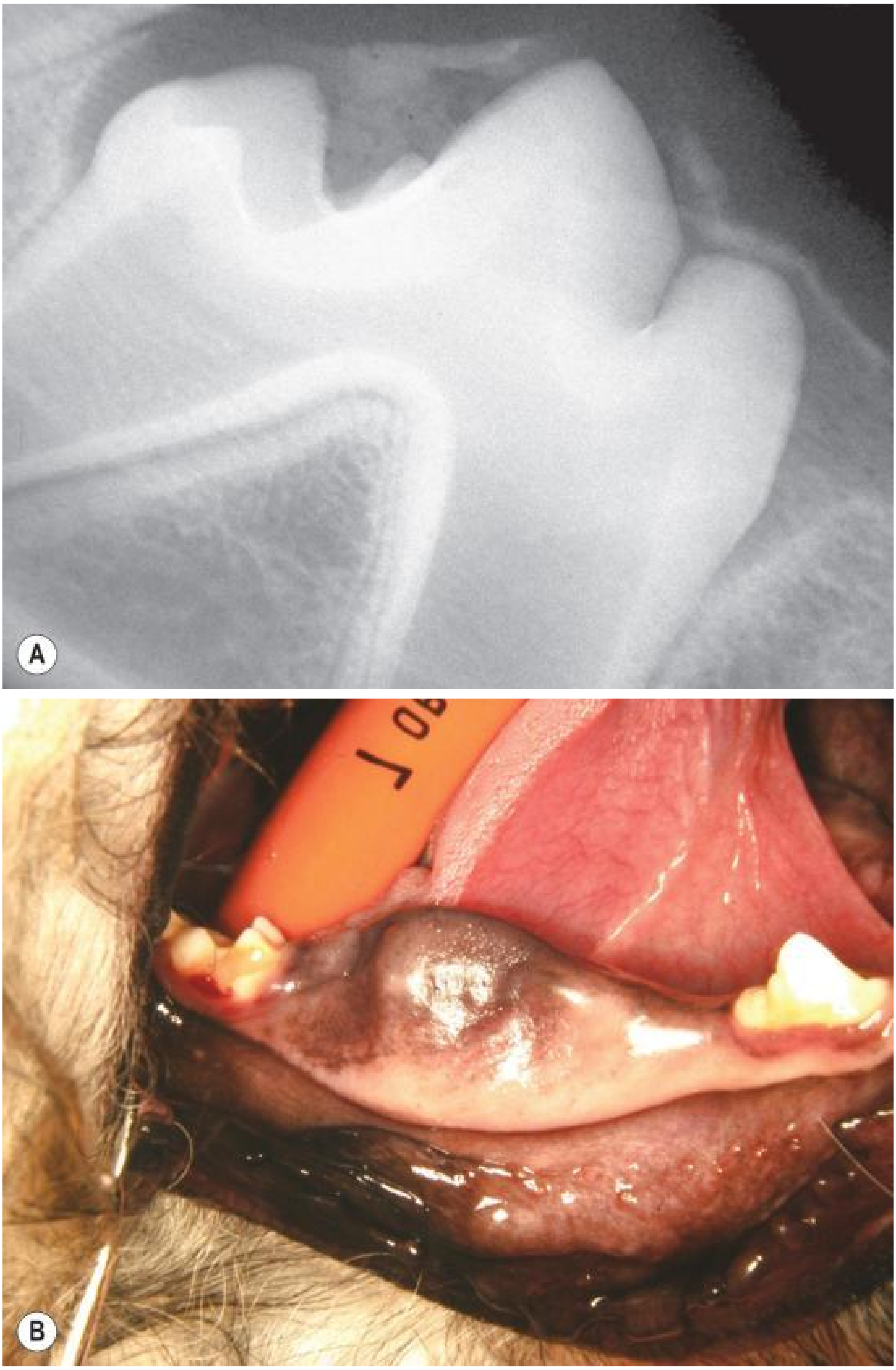
Management of unerupted teeth

Loïc F. J. Legendre

Definitions

Embedded tooth: A tooth that is unerupted, usually because of a lack of eruptive force.[1]
Extrusion: Movement of a tooth in a coronal direction.
Impacted tooth: A tooth that is prevented from erupting by some physical barrier in the eruption path, such as crowding from adjacent teeth.[1,2]
Operculectomy: Excision of the soft tissue overlying an unerupted tooth.
Operculum: Oral mucosa overlying an unerupted tooth.
Pericoronitis: Inflammation of the operculum overlying an unerupted tooth or around the crown of a partially erupted tooth.

The term "unerupted" includes both impacted and embedded teeth. An embedded tooth is unerupted usually because of a lack of eruptive force.[1] An impacted tooth is prevented from erupting by some physical barrier in the eruption path, such as crowding from adjacent teeth.[1,2] Impaction is the most common condition.[2] It may lead to root resorption, root dilaceration, pericoronitis, jaw fracture, and the formation of odontogenic cysts and tumors.[2] A tooth may stop erupting without being blocked by another tooth, but rather by a bone shelf and thickened gingiva not being resorbed properly (Fig. 59.1A).[3]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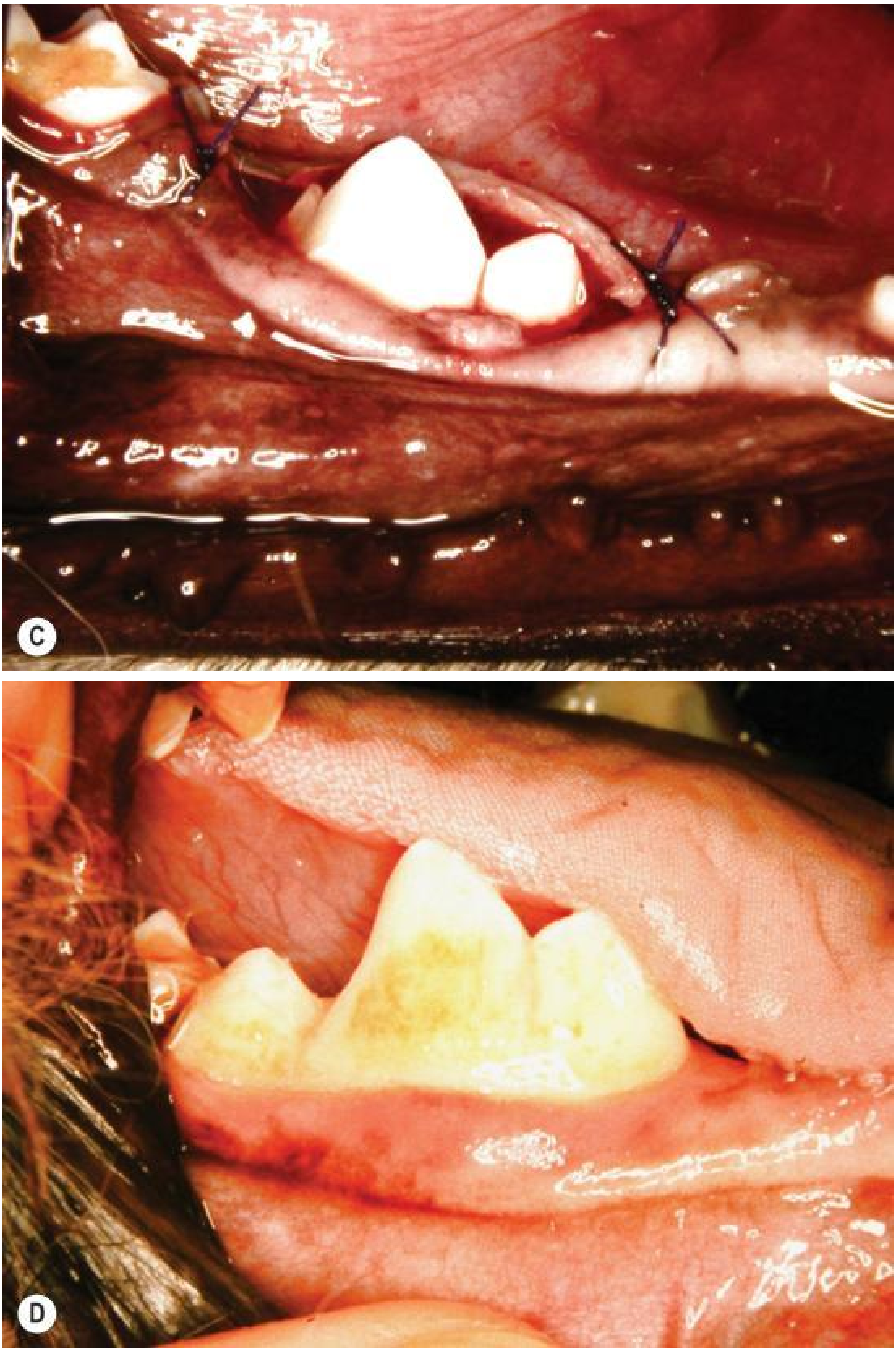


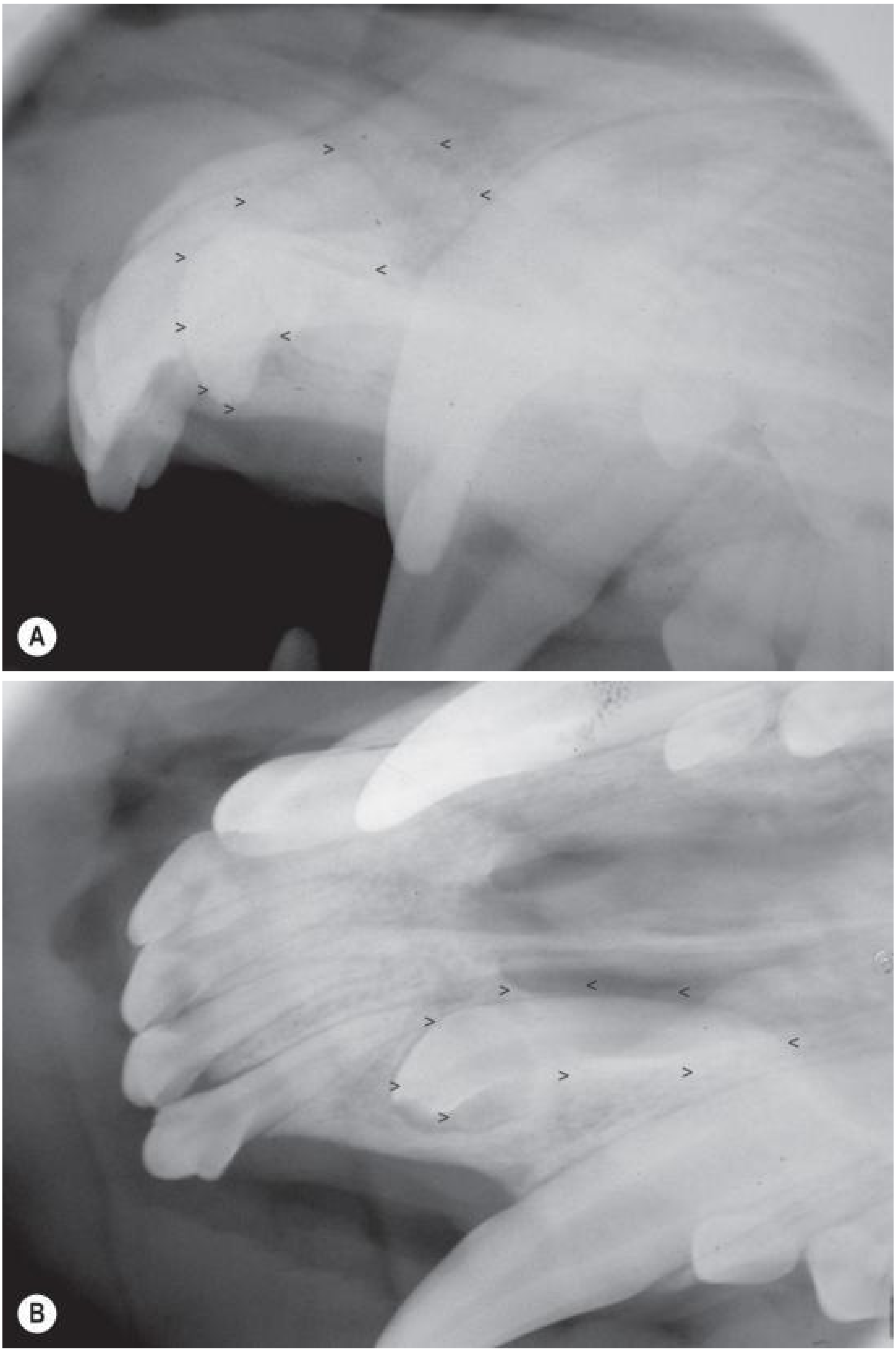
• **FIG. 59.1** (**A**) Radiograph of a right mandibular first molar tooth in a 7-month-old dog impacted under a thin shelf of bone. Clinically, a thick layer of keratinized tissue overlying the tooth was present. (**B**) Right mandibular first molar tooth impacted under a cover of gingiva. (**C**) Two-thirds of the crown of the mandibular right first molar tooth are exposed following gingivectomy. Note that more than 2 mm of attached gingiva is left around the impacted crown. (**D**) The tooth has erupted spontaneously. One month after being exposed, it is in its anatomic position and requires no further treatment. Note that the full crown is now exposed.

Corrective treatment is divided into three steps: surgical exposure, attachment to tooth, and orthodontic movement to bring tooth into position.[4] Surgical exposure typically entails an operculectomy, the surgical removal of the often thickened oral mucosa overlying an unerupted tooth.[2,5,6] The orthodontic movements required to realign an unerupted tooth may be complex, and orthodontic treatment typically includes extrusion, which means that the tooth is slowly moved in an axial direction out of its alveolus.[4]

Surgical anatomy

Location of an unerupted tooth

Preoperative planning and choice of surgical approach are based on the location of the unerupted tooth. It is sometimes difficult to ascertain the exact location of the affected tooth, and preoperative diagnostic imaging is essential. Two radiographic techniques are commonly utilized: the vertical and the horizontal tube shift techniques. These techniques are based on the principle that the movement of an image in relation to a reference object is dependent on the change in angulation of the X-ray beam. With the horizontal shift technique, the X-ray tube is moved mesiodistally on a horizontal plane, moving the affected tooth in the same direction as the tube shift when palatally impacted and in the opposite direction when labially impacted. With the vertical shift technique, the vertical angulation of the X-ray tube is altered. The tooth located farthest from the X-ray beam will be moved in the direction opposite to the beam (Fig. 59.2). Either technique requires at least two radiographs to determine the exact position of the affected tooth.[3,7,8] Recently, cone-beam computed tomography (CBCT) has become more common in veterinary facilities, and in human dentistry CBCT has been used to locate unerupted teeth.[9] It allows the clinician to determine buccolingual positioning in detail, to distinguish and define the extent and depth of root resorption, and to delineate long-axis orientation of unerupted teeth, including root apex location. In addition, three-dimensional imaging contributes to more accurate and less traumatic surgical exposure, as well as to more efficient and directionally appropriate orthodontic traction, than does traditional two-dimensional imaging and thus contributes to faster resolution and better overall tooth prognosis.[9] Accurate localization is crucial for approaching the tooth with minimal damage to the surrounding structures.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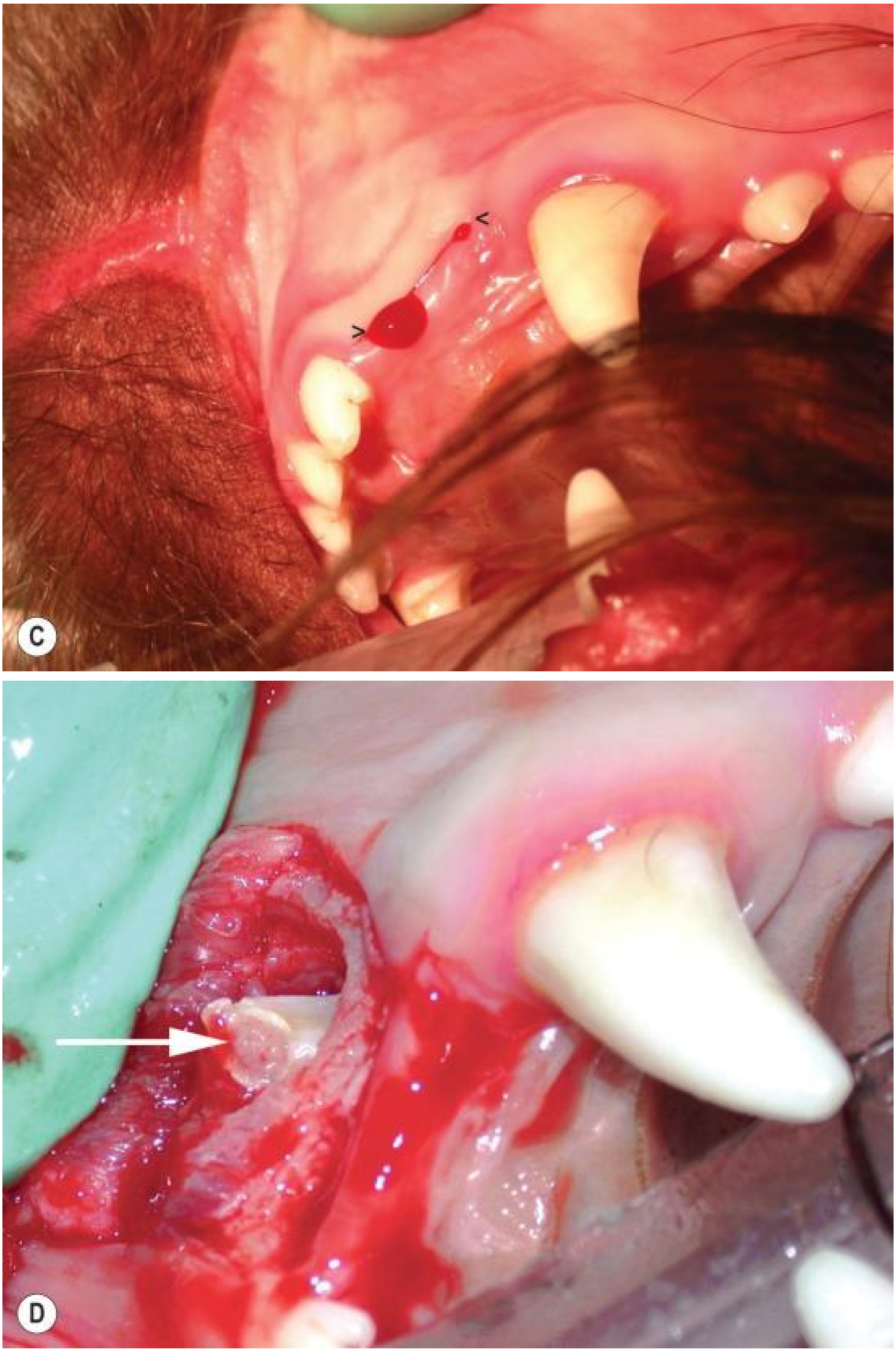
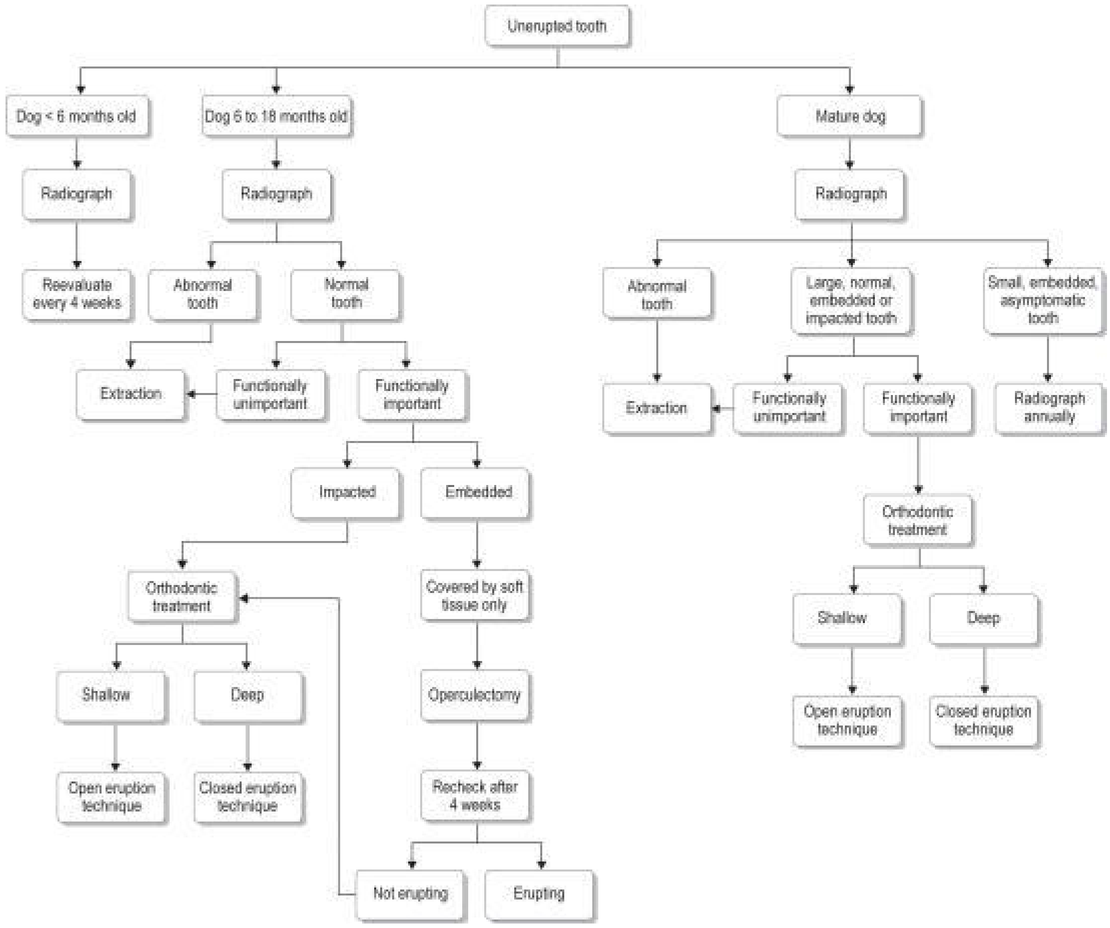


• FIG. 59.2 (**A**) First view of a vertical tube shift technique to localize the impacted incisor tooth. The tube is nearly horizontal in alignment. (**B**) Second view, the tube is moved ventrally and the tooth that is farthest from the tube moves in the same direction. (**C**) An incision is made over the area of the impacted tooth. (**D**) Ostectomy and osteoplasty are performed to expose half to two-thirds of the crown. The *arrow* shows where the attachment will be bonded to the crown to facilitate extrusion of the tooth.

Therapeutic decision-making

Treatment options for unerupted teeth include extraction, intervention to encourage normal eruption, or orthodontic movement of the teeth. The decision as to which option to take depends on several factors. Age is of prime importance. When dealing with deciduous teeth, careful neglect under close observation is indicated only until the adult teeth are due to erupt. Immature impacted teeth will sometimes conveniently erupt unaided once the obstruction is removed. This happens very rarely once root formation is complete.[4] Adult impacted teeth should be treated before complications arise.[2] The type of tooth will also affect the decision; a functionally important tooth warrants more effort to save it, and extrusion would then be recommended. Surgical exposure and orthodontic treatment amount to a lengthy, complex procedure that is rarely undertaken in veterinary dentistry. On the human side, the procedure is approached as a team involving orthodontics and surgery.[10] The presence of complicating factors similarly affects the outcome. Reasons for extracting as opposed to moving teeth are: (1) existing lesions or pain from pericoronitis, periodontitis, periapical abscess, cyst or neoplasm, impingement on and external resorption of adjacent teeth, and inflammation; (2) abnormal, dysplastic teeth; (3) aberrant position, making it impossible to move the affected tooth; and (4) lack of space for the unerupted tooth to move into.[4] Extraction is contraindicated if it carries greater risks than potential benefits, if the medical status of the patient is compromised, and if extraction can result in extensive damage to adjacent structures.[2] Often, secondary lesions are present at time of diagnosis, and the operator needs to be prepared to deal with complications such as cyst or tumor formation that will necessitate an en bloc excision rather than simple extraction. The algorithm in Fig. 59.3 summarizes the correct decision-making process.



• **FIG. 59.3** Algorithm depicting the decision-making process when faced with an unerupted tooth.

Surgical techniques

Surgical exposure

A surgical approach is necessary to expose the crown of the tooth before orthodontically moving it. Various procedures have been described, depending on the depth of the impaction. In cases of shallow impaction when only keratinized tissue is covering the tooth (see Fig. 59.1B), an operculectomy is indicated. Exposure of one-half to two-thirds of the crown is sufficient. A minimum of 2 mm of attached gingiva is left at the base of the treated tooth to prevent gingival recession (see Fig. 59.1C).[3,11] If there is little attached gingiva present, an apically positioned flap procedure is preferred in order to preserve the maximum amount of keratinized tissue. Because the gingiva present is often thicker than normal, a partial-thickness flap is recommended to correct this problem. Once the crown of the tooth is exposed, the flap is sutured in a more apical position.

In cases where the tooth is deeper, an open eruption technique or a closed eruption technique is used. The open eruption technique consists of an apically positioned flap combined with an ostectomy. Half to two-thirds of the crown is exposed before reattaching the flap. Exposing the crown down to the cemento-enamel junction leads to periodontal attachment loss following orthodontic treatment.[12] An attachment device, such as a small button or a hook, is then bonded to the crown to commence the orthodontic part of the treatment.[4] The closed technique is reserved for deep impactions or where an apically positioned flap is impractical. It consists of making an alveolar margin incision over the location of the tooth (see Fig. 59.2C). Vertical incisions are extended mesially and distally to allow exposure of the area without tearing the flap. Bone is removed to expose just enough of the crown to bond an attachment connected to a traction wire (see Fig. 59.2D). Radical removal of bone during the exposure of an impacted tooth is unnecessary and potentially may be harmful in terms of the long-term periodontal health of that tooth.[13] The flap is sutured back in place, and the wire exits through the incision. This ensures that the tooth will erupt through attached gingiva. If the tooth erupts through oral mucosa rather than gingiva, it will be periodontally compromised.[4]

Orthodontic management

Before starting the surgical exposure, the operator should make sure that there is sufficient space for the tooth to be extruded into. If the space does not exist, it must be created prior to exposing the impacted tooth. When the impaction is shallow, an immature tooth may complete its eruption without aid (see Fig. 59.1D).[3,4] Orthodontic treatment may only be necessary to tip it into its correct position. When the impaction is deeper, orthodontic treatment consists of two phases: extrusion and final placement. Traction is applied to the affected tooth using an elastic chain and adjacent teeth as anchor. Sometimes, a rigid arm is created to direct the force on the tooth in the appropriate direction. The arm also increases the range of movement and the consistency of the force applied.[4] Extrusion by means of a small magnet bonded to the crown of the impacted tooth and a larger anchor magnet incorporated into an acrylic appliance has been used in humans.[4,14] Extrusion requires light forces, similar to those used in tipping movements.[4] Once erupted, the tooth is moved or tipped by orthodontic means; once the tooth is in place, a retainer is indicated to prevent relapse.[4]

Extraction of unerupted teeth

A surgical extraction technique is used for the extraction of unerupted teeth. This must be carefully planned, given the fact that the tooth may be covered by a considerable amount of bone, its anatomical location may be abnormal, and its shape may be distorted. A wide pedicle or triangle flap is indicated to obtain adequate exposure. The amount of bone removal should be sufficient to allow atraumatic delivery of the tooth, taking care not to weaken the jaw bone unduly. This may be challenging, especially when extracting an unerupted mandibular canine or first molar tooth. If cystic changes surrounding the unerupted tooth are apparent, the soft tissue lining must be completely removed and submitted for histopathological examination. Bone grafting may be indicated to promote healing of the alveolectomy site.

Postoperative care

Pain management is very important but routine (see Chapter 4). The client is instructed to observe for postoperative signs of swelling, bleeding, discoloration, and inappetence. It is essential for the client to keep the surgical area clean at all times. This is achieved by daily, gentle cleaning of the area using veterinary toothpaste or chlorhexidine rinse and a toothbrush or a cotton swab. Debris, plaque, and calculus deposition will interfere with healing and may result in severe inflammation that can lead to dehiscence and failure of the procedure. The patient is examined at 5 to 7 days postoperatively to ascertain that the flap is healing correctly.

Complications

Three types of complications are encountered: intraoperative, postoperative, and complications associated with the orthodontic treatment. Intraoperative complications include hemorrhage, damage to the unerupted crown, fracture of tooth, and fracture of adjacent bone. Postoperative complications include dehiscence of suture line, flap necrosis, and infection of surgical site. Some complications are specific to the orthodontic treatment. Lack of movement may be due to ankylosis of the unerupted tooth, which should have been diagnosed prior to treatment by gently luxating the tooth following exposure. Trying to move an ankylosed tooth is impossible and would cause the anchor teeth to tip.[3,4,10] Most common are periodontal problems associated with the extruded tooth. Extruded teeth have increased plaque and gingival bleeding indices, greater pocket depths, reduced attached gingival width, increased crown lengths, and reduced bone levels compared to their untreated contralaterals.[15] Unless an adequate band of keratinized tissue is left attached around the base of the crown, the incidence of attachment loss, gingival recession, and inflammation is high. Extrusion and further orthodontic movement have been found to result in impairment of the pulpal blood flow and subsequent necrosis in over 20% of human patients.[16] Endodontic treatment is required to correct this complication. Damage to the orthodontic appliance is also a common complication. Alterations to the design of the appliance may be necessary to prevent repeat breakage. Vertical relapse may occur and an appropriate method and length of retention should be included in the treatment plan.

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SECTION 12:

Oral Approaches to Ear, Nose, and Throat Procedures

OUTLINE

CHAPTER 60

Correction of overlong soft palate

Kyle G. Mathews

Surgical anatomy

The soft palate extends caudally from the hard palate to just beyond the tip of the epiglottis in mesocephalic breeds. It separates the oropharynx ventrally from the nasopharynx dorsally. The elongated soft palate of many brachycephalic dogs may be so long as to reach the vocal folds and thereby partially obstruct airflow (Fig. 60.1). The soft palate is covered with a stratified squamous epithelium ventrally and along its caudal margin, while it is covered by a pseudostratified ciliated columnar epithelium over most of its dorsal surface.[1] Numerous palatine glands open to the ventral surface of the soft palate. The number of glands decreases caudally. The bulk of the soft palate consists of the paired palatine muscles, which are separated at the midline. Dorsal to the palatine muscles and ventral to the nasopharyngeal epithelium there is a palatine aponeurosis, which blends with the terminal fibers of several muscles including the palatopharyngeal, tensor, and levator veli palatini muscles. Arteries feeding the soft palate include the paired minor palatine arteries and, to a lesser extent, the ascending pharyngeal branches which arise from the major palatine arteries. Efferent blood flow occurs via the laterally located paired venous plexi.

• **FIG. 60.1** Preoperative photograph of a pug with elongated soft palate. Oropharyngeal tissues are erythematous and the palatine tonsils are everted from their fossae. Ventral retraction of the epiglottis with a laryngoscope allowed visualization of the caudal tip of the soft palate, which extended to the level of the vocal folds. The laryngeal ventricles were also everted.

Special instruments and materials

All instruments should have handles that are at least 180 mm in length. Kelly hemostats, Metzenbaum scissors or angled (Potts) scissors, DeBakey forceps, long-handled needle holders, head lamp or other supplemental light source, and 3–0 to 5–0 poliglecaprone 25 (Monocryl, Ethicon, Inc. Somerville, NJ) suture are used. If a CO_2 laser is to be used, matted nonreflective instruments and protective eyewear are required (see Chapter 10).

Therapeutic decision-making

Most brachycephalic dogs suffer from some degree of airway obstruction. The English bulldog is the most commonly affected breed.[2] Stenotic nares and everted laryngeal ventricles are commonly present in association with an overlong soft palate.[2-8] This triad of congenital abnormalities is referred to as brachycephalic syndrome. Many brachycephalic dogs, English bulldogs in particular, also have redundant pharyngeal mucosa and a hypoplastic trachea, which exacerbate the problem.[3,9-12] With time, the increased negative pressure that must be generated by the affected animal during inspiration may not only cause laryngeal ventricle eversion but may ultimately result in laryngeal collapse. Laryngeal collapse is the end-stage result of brachycephalic syndrome.[13,14] Weakening of the laryngeal cartilages results in their collapse toward the midline, narrowing of the *rima glottidis* and further airway obstruction. A vicious cycle of obstruction, increased work of breathing, increased inspiratory strain on the laryngeal cartilages, and further collapse ensues. Treatment at this stage may require a permanent tracheostomy with all of the inherent problems associated with a tracheostomy in a loose-skinned dog.[15,16]

Affected animals are typically presented with a history of inspiratory stridor, snoring, and exercise intolerance. Severely affected dogs may have a history of collapse or syncope, and present in respiratory distress, especially during hot and humid weather. They may be cyanotic, acidotic, hyperthermic, have exaggerated thoracic wall movements consistent with airway obstruction, and may develop secondary pulmonary edema.[17,18] Cyanosis is only evident when the PaO_2 is at or less than 60 mmHg.[3,19] Myocardial ischemia may result in arrhythmias. Additionally, some dogs will have vomited as a result of acidosis and stimulation of a gag reflex by the elongated palate.[5] This may compound matters if the vomitus is aspirated. Oxygen therapy, intravenous fluids, diuretics, sedation, and cooling are often needed to stabilize the patient that presents in distress.[3-5,19] Monitoring with an electrocardiogram (ECG), serial blood gas analysis (if available), and pulse-oximetry (SpO_2) should be considered. In severe cases, endotracheal intubation or a temporary tracheostomy may be needed.

Once the patient is stable enough, thoracic radiographs are performed and evaluated for evidence of pulmonary edema and hypovolemia. Tracheal diameter is measured, and other conditions that may result in similar clinical signs, such as laryngeal neoplasia, are investigated.

Chronic hypoxia secondary to brachycephalic syndrome may also result in pulmonary vasoconstriction with resultant pulmonary hypertension, *cor pulmonale* and eventual right heart failure.[3] With these things in mind, owners of brachycephalic dogs should be counseled about warning signs (stridor, etc.) that indicate there is some degree of airway obstruction. The soft palate should be examined at the time of routine neuter or spay to aid in this counseling process. Correction of overlong soft palate (staphylectomy), nasal wedge excision, and excision of everted laryngeal ventricles, if present, should be done before the animal experiences a respiratory crisis.

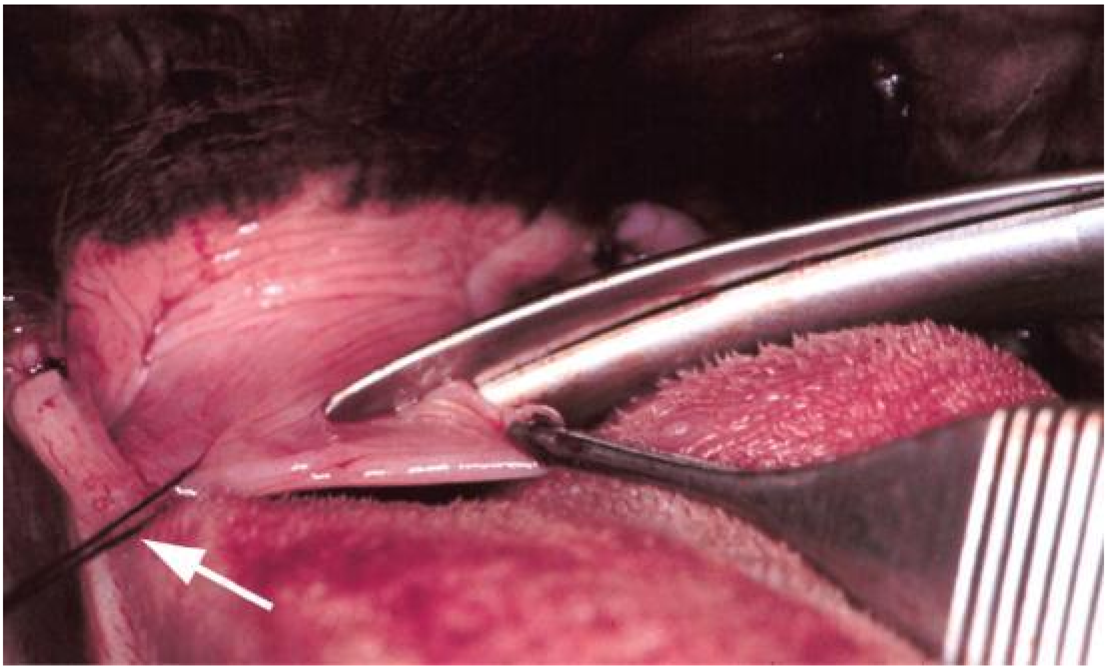
In stable patients, a thorough pharyngeal examination is performed under general anesthesia with the plan to surgically correct an overlong soft palate, if present, during the same anesthetic episode. The patient should be preoxygenated prior to induction. Cefazolin (22 mg/kg, intravenously [IV]) and prednisolone (1 mg/kg, IV) are typically administered at induction. Using a laryngoscope, the pharynx, tonsils, soft palate, and larynx are carefully examined. In affected dogs, the soft palate and larynx are often erythematous and edematous. As previously mentioned, the soft palate extends just beyond the tip of the epiglottis in the normal dog.

Indications for surgery

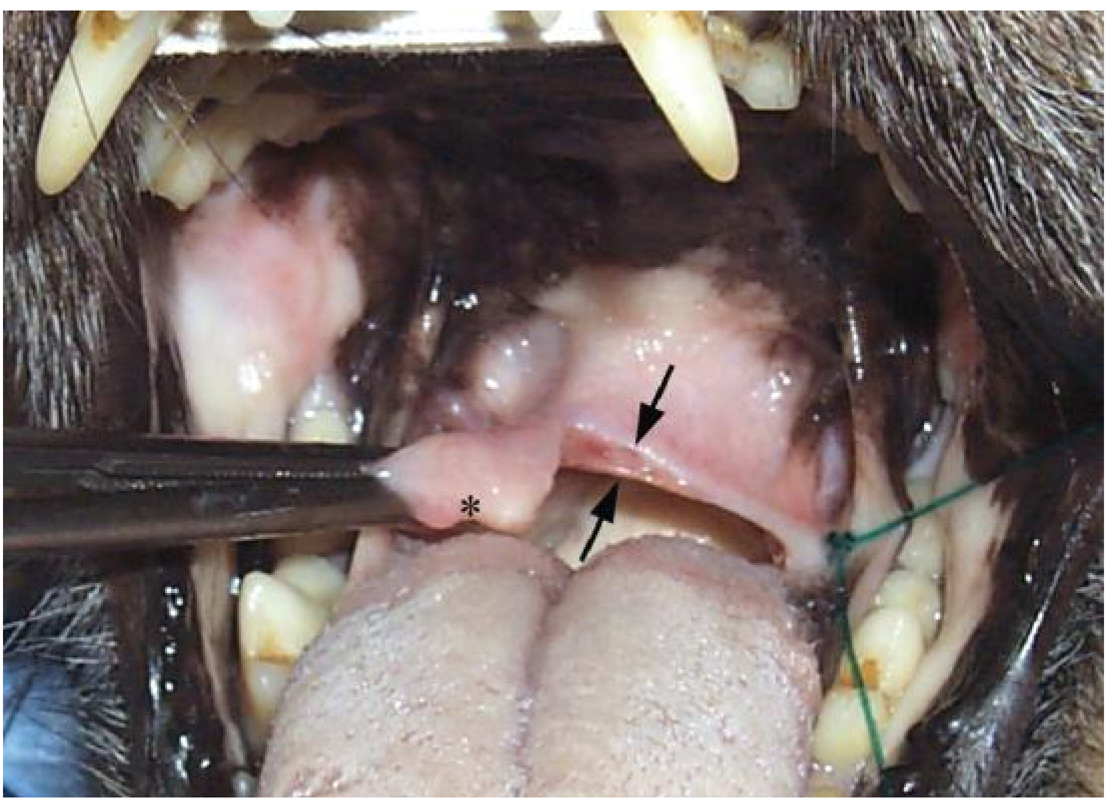
Any brachycephalic dog with inspiratory stridor or exercise intolerance should have its soft palate examined, and if elongated, it should be shortened (staphylectomy). The soft palate may also be thickened due in part to an increase in the collagenous stroma rather than muscular hypertrophy.[20] If so, a folded flap palatoplasty may be considered if palatal thickening is marked.[21] The most appropriate technique (standard staphylectomy versus folded flap) for individual dogs based on standardized imaging and soft palate measurements has not been elucidated with a blinded and randomized prospective clinical trial. Stenotic nares and everted laryngeal ventricles are corrected, if present, and a tonsillectomy performed if the palatine tonsils are significantly enlarged and contributing to airway obstruction (see Chapter 63).[2-7] Simultaneous neutering should also be considered.

Surgical technique

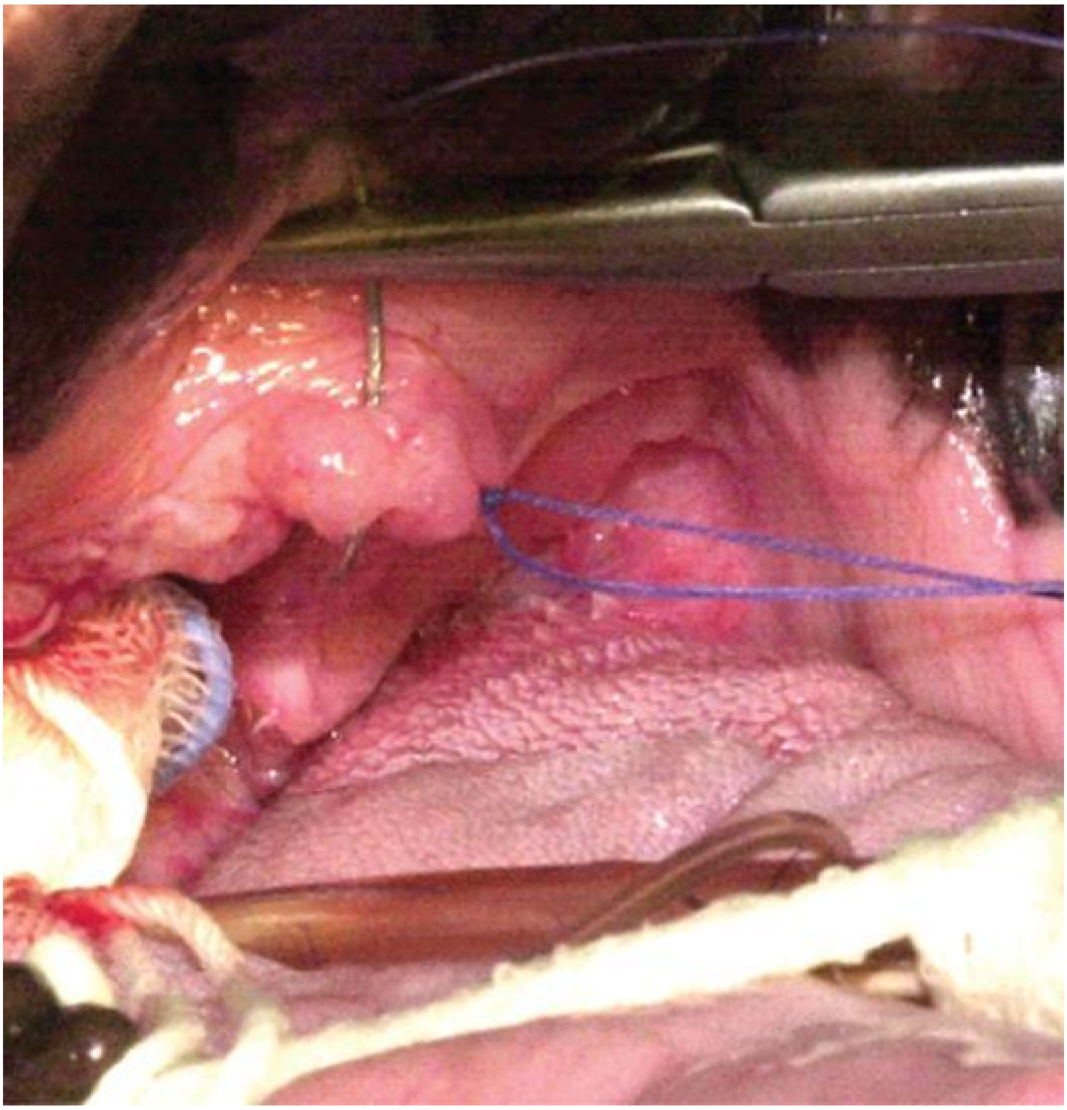
With the dog's maxilla suspended with gauze between two IV poles, a known number of pharyngeal sponges are placed, and the cuff of the endotracheal tube is inflated to prevent aspiration of blood. Adequate lighting is required—a headlamp is preferred. An assistant grasps the dog's tongue with one hand and retracts the epiglottis and endotracheal tube ventrally (if present) with a laryngoscope. Some surgeons prefer to perform this procedure after placing a temporary tracheostomy tube to improve exposure of the soft palate and laryngeal ventricles. The author has found this to be unnecessary in most cases as long as adequate assistance is available. If right-handed, first place a surgical knot in the left lateral edge of the dog's soft palate just caudal to the ipsilateral palatine tonsil. A hemostat is placed on the tag end of the suture, which is left long to aid in the manipulation of the palate (some surgeons prefer to place a stay suture at the palatal margins bilaterally). The assistant places tension on this end of the suture while the surgeon grasps the tip of the soft palate, pulling it ventrally and to the dog's right. This maneuver tenses the soft palate, making it easier to cut. The palate is shortened using sharp dissection with Metzenbaum scissors (Fig. 60.2). A transverse cut is made across one-third of the soft palate near the tip of the epiglottis. By pulling the tip of the soft palate rostrally, the nasopharyngeal mucosa, which may have retracted dorsally, is easy to locate (Fig. 60.3). The oral and nasopharyngeal mucosa of this segment are then sutured together with a simple continuous pattern (e.g., using 4-0 or 5-0 poliglecaprone 25 [Monocryl, Ethicon, Inc. Somerville, NJ]) (Fig. 60.4). This process is repeated in sections to limit hemorrhage. Grasping the mucosa with forceps should be avoided, if possible, to limit swelling. The palatine muscles are not included in the suture. To complete the suture pattern, a second knot is started at the left lateral edge of the soft palate (a mirror image of the right side), a final small attachment of the end of the soft palate is transected, and the knot is finished. All suture ends are then trimmed (Fig. 60.5). Sponges are recounted and the pharynx is suctioned prior to recovery from anesthesia.



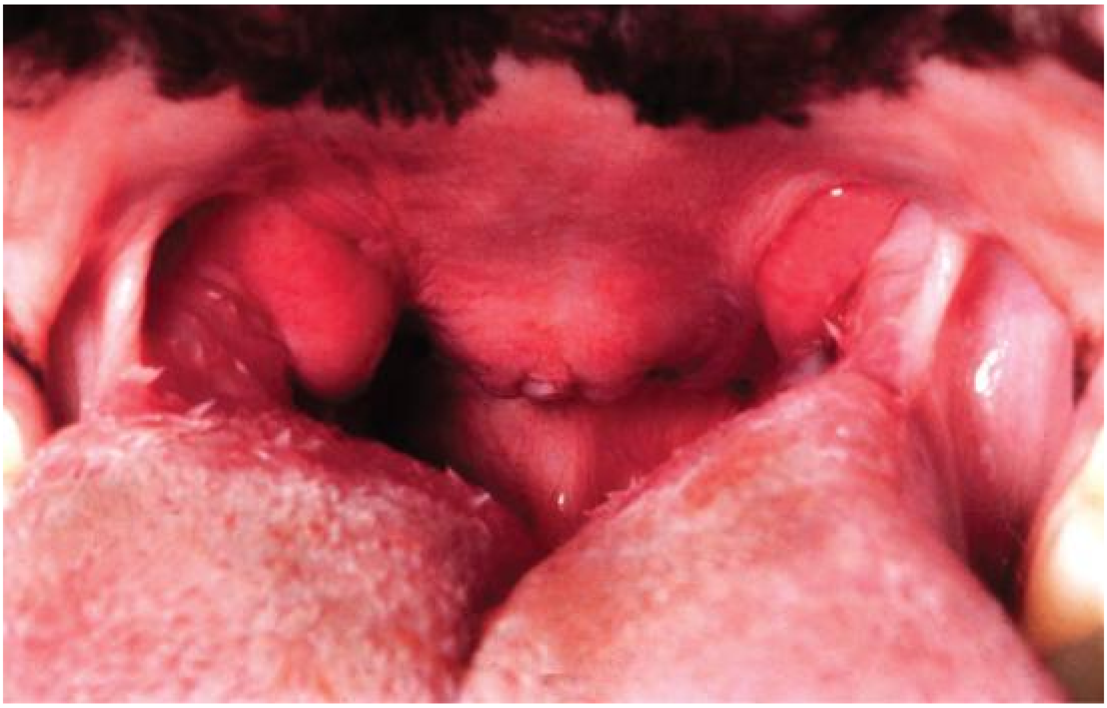
• **FIG. 60.2** The caudal tip of the soft palate is grasped with forceps and retracted ventrally and rostrally. Lateral tension is applied to the caudolateral margins of the soft palate with unilateral or bilateral stay sutures *(arrow)*. Metzenbaum scissors are used to cut transversely across approximately one-third of the soft palate. The nasopharyngeal and oropharyngeal mucosa are sutured together before continuing with the transection.



• **FIG. 60.3** By applying lateral tension to the stay suture, and rostroventral tension to the tip of the soft palate *(asterisk)*, the nasopharyngeal and oropharyngeal mucosa *(arrows)* can be identified.



• **FIG. 60.4** The nasopharyngeal and oropharyngeal mucosa are sutured together with a simple continuous pattern. The palatine muscles are not included in the sutures.



• FIG. 60.5 Following completion of the staphylectomy, the caudal tip of the soft palate should lay just caudal to the tip of the epiglottis. The tip of the epiglottis has been pushed dorsal to the soft palate in this photograph for illustration purposes.

Some surgeons prefer to place a hemostat across the soft palate to crush the tissues and to mark the line of excision so as to prevent overcorrection.[3] Another potential benefit of this technique is decreased hemorrhage, although there is surprisingly little blood lost if performing the cut-and-sew method described above. Electrocautery is not needed and is discouraged. Crushing the soft palate with a hemostat will also likely increase postoperative swelling. It is unclear if this predisposes to clinical difficulties. Others prefer to mark the location of the epiglottis on the soft palate with a sterile marker prior to excision.[5]

Laser staphylectomy is another viable alternative (see Chapter 10).[22-24] Hemorrhage is minimal since the laser seals small blood vessels as it cuts. In one study, surgery time was shorter with CO_2 laser staphylectomy than with a conventional sharp dissection technique. There were few other differences between the two groups in postoperative clinical or histological response.[23] Another study comparing staphylectomies performed with a CO_2 laser, diode laser, or monopolar electrocautery found shorter surgery times and less hemorrhage in the CO_2 treated group.[24] Significant complications occurred in 4 of 20 dogs treated with the diode laser. The use of a laser in the oral cavity requires special precautions to avoid airway fires, and inadvertent injury to other tissues and personnel. The use of CO_2 laser has been compared to a bipolar sealing device (LigaSure, Covidien, Medtronic, Minneapolis, MN) for performing a staphylectomy on normal dogs. Few differences between the two techniques were noted other than a decreased operative time when using the bipolar device.[25] Use of a bipolar sealing device has subsequently been reported in a case series of 22 dogs and was associated with a low number of major complications.[26]

When using the conventional technique, primary repair is important to limit inflammation. Postoperative dysphagia following suture failure and subsequent granulation tissue formation at the edge of the palate is possible. Overshortening the palate prevents the dog from occluding its nasopharynx; the net result of this will be the nasal return of food during swallowing and a severe rhinitis.[7]

Postoperative care and assessment

Despite contamination at the time of surgery, prolonged antibiotic therapy is not indicated. As previously noted, a single dose of corticosteroids is generally given at induction, prior to manipulation of the tissues, to diminish postoperative swelling. Because postoperative laryngeal/pharyngeal edema can result in airway obstruction, continuous monitoring for the first 12–24 hours is highly recommended. If edema is significant at the time of surgery, a temporary tracheostomy tube is placed prior to recovery. Frequent suctioning of the tube will be needed to prevent obstruction.[15,16] The tube is removed when the patient can breathe around it during trial occlusion. Oxygen should be readily available for all dogs undergoing correction for an overlong soft palate. Some prefer to place a nasal oxygen line prior to recovery if there is edema present. Induction drugs and a tracheostomy tube should also be kept nearby so that rapid induction followed by intubation can be performed if needed. Retching and vomiting may occur postoperatively.[3,7] If vomiting occurs, the patient should be evaluated for the possible development of aspiration pneumonia. Soft food is fed for the first 3–7 days.

Prognosis

The prognosis for a full recovery is generally good if brachycephalic syndrome has not resulted in laryngeal collapse or right-sided heart failure. However, owners of affected dogs must be warned about the possibility of postoperative obstruction. English bulldogs may have a worse prognosis than other brachycephalic breeds.[4] They are more likely to have redundant pharyngeal mucosa and a hypoplastic trachea than other breeds. One report indicated good to excellent results in 67% of non-bulldog breeds, and similar results in 45% of English bulldogs.[4] The fact that only 56 of 118 owners returned questionnaires in this study likely affected the results. Still, most feel that English bulldogs are prone to postoperative complications such as aspiration pneumonia. Because of this, preoperative treatment with prokinetic agents such as metoclopramide, and antacids such as famotidine have been suggested.[4] Concurrent esophageal motility disorders, delayed gastric emptying and decreased ability to clear aspirated material from a hypoplastic trachea have been speculated to contribute to an increased postoperative risk of aspiration pneumonia.[4,5] Another report showed that 78% of dogs were improved following surgery.[7] Improvement was more commonly seen in those dogs that had soft palate excision combined with nasal wedge excision. Most dogs can be discharged after a day of monitoring. Those requiring a temporary tracheostomy are usually hospitalized for 3–5 days. Most owners report marked improvement in their dog's energy level and decreased inspiratory noise and effort following surgery.

Recurrence of clinical signs may develop in some dogs. If inspiratory stridor redevelops, a repeat pharyngeal examination should be performed. Possible causes for this include the presence of redundant pharyngeal mucosa, or progressive laryngeal collapse. Soft palate elongation in dogs several years following staphylectomy may also occur; this is presumably due to tissue stretching secondary to the negative pressure associated with partial laryngeal collapse and a hypoplastic trachea.

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CHAPTER 61

Pharyngotomy and pharyngostomy

Yoav Bar-Am, Gary C. Lantz

Definitions

Pharyngostomy: Surgical formation of an artificial opening into the pharynx through which a feeding tube is placed, which will remain in place for a period of time.[1]

Pharyngotomy: Surgical incision into the pharynx performed for placement of an endotracheal or feeding tube[1]; the endotracheal tube is removed immediately after the surgical procedure.

Background

Pharyngotomy for endotracheal tube placement was originally described by Hartsfield et al in 1977.[2] It was specifically developed for improving the surgeon's view of the intraoral operative field and for evaluating dental occlusion during surgery.

Pharyngostomy feeding tube placement had previously been described by Bohning et al in 1970.[3] The original technique was modified by Crowe and Downs to prevent complications of airway obstruction and aspiration pneumonia.[4]

Indications and contraindications

Endotracheal tube

The purpose is to bypass the oral cavity, most commonly for repair of maxillary and mandibular fractures. Placement of the endotracheal tube in this location allows continual intraoperative evaluation of occlusion with the goals of restoring the patient's normal occlusion, thereby returning the patient to normal oral function. An alternative way to allow occlusion evaluation during maxillofacial trauma repair is by performing a transmylohyoid orotracheal intubation.[5] At the time of extubation, a feeding tube could be placed through the same pharyngotomy.[6]

Feeding tube

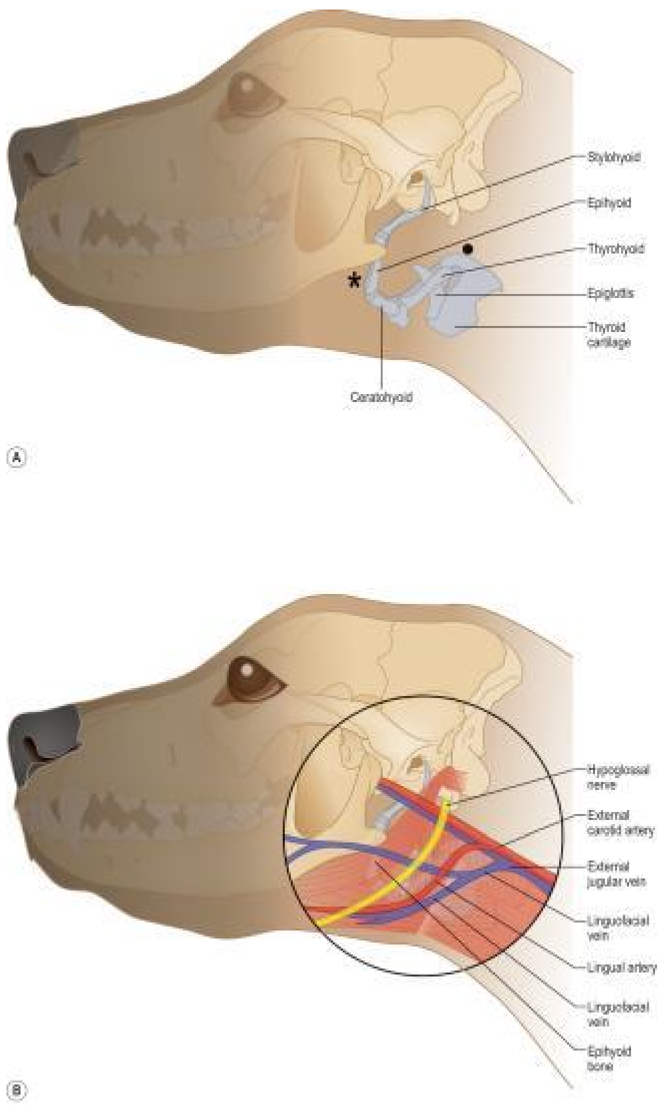
This tube is placed as one method to administer food, water, and medications into the gastrointestinal tract (see Chapter 5).

Contraindications

Tissue inflammation secondary to trauma and infection or the close proximity of neoplasia negates pharyngotomy for placement of either tube. A feeding tube, either via pharyngostomy or esophagostomy, is contraindicated in esophageal disorders such as stricture, esophagitis, neoplasia, and in cases with a recent history of vomiting.

Surgical anatomy[7]

The relevant surgical anatomy is shown in Fig. 61.1. Entry into the pharynx is made behind the caudal pole of the palatine tonsil, immediately ventral to the mandibular salivary gland, dorsolateral to the entrance into the larynx *(aditus laryngis)*, and rostral to the esophageal orifice. The maxillary and linguofacial veins, external carotid artery, vagosympathetic trunk, and the recurrent laryngeal nerve are in close proximity to this pharyngotomy site. Easier access for endotracheal tube placement may be possible by placing the pharyngotomy site immediately rostral to the epihyoid bone as indicated in Fig. 61.1. Placement of a pharyngostomy feeding tube at this site may create epiglottic entrapment with resulting airway obstruction and aspiration pneumonia, and is not recommended.[4]



• **FIG. 61.1** (**A**) Recommended pharyngotomy approaches for feeding tube placement *(black dot)* and endotracheal tube placement *(asterisk)*. Placement of the feeding tube in a more caudal position prevents epiglottic entrapment. The endotracheal tube placed through this pharyngotomy site is removed immediately after surgery. (**B**) Neurovascular anatomy adjacent to the pharyngotomy sites.

Special instruments and materials

Endotracheal tube

A wire-reinforced tube (Aire-cuf, Bivonia Inc., Gary, IN) is recommended to prevent collapse of the tube when bent.[6]

Feeding tube

Generally recommended tube diameters are as follows: 10 to 12 French gauge (F) (3.3 to 4.0 mm) for cats and small dogs; 16 to 18 F (5.3 to 6.0 mm) for animals with a body weight of 10 to 15 kg, and 18 to 20 F (6.0 to 6.7 mm) for larger dogs. The relatively small-sized tube in relation to patient size reduces the risk of injury to anatomic structures during tube insertion and maintenance. Commercial liquid diets or commercial canned foods made into a liquid consistency in a food blender pass through these tubes. Appropriate tube materials include rubber (Sovereign, Kendall Co., Mansfield, MA) and silicone (Cook Veterinary Products, Eight Mile Plains, Queensland, Australia) feeding catheters, and Foley catheters (Foley Catheter, American Hospital Supply Corp., Valencia, CA) (without distension of the bulb). The ends of tubes with side ports are cut off to minimize tube obstruction. Mushroom tips are also cut off.

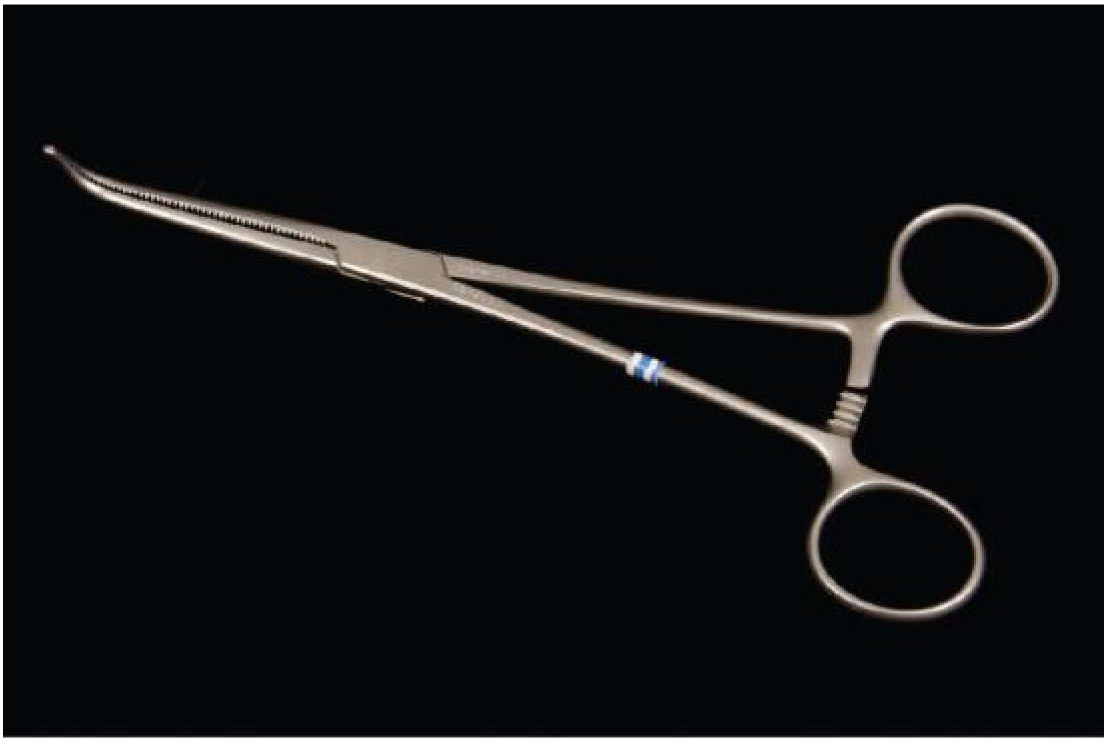
Surgical technique[4,8,9]

Tubes may be placed in the right or left pharyngeal wall according to the surgeon's preference, or as determined by the surgical procedure planned; for example, for caudal mandibular fracture repair the contralateral pharyngeal wall is preferred. The skin surface is prepared for surgery. The external jugular vein is occluded at the thoracic inlet to cause distention of the maxillary and linguofacial veins to confirm their location so as to avoid inadvertent trauma. Once the incision site is determined, the tube should be premeasured and cut to length before placement. A gloved hand is placed into the oral cavity and the hyoid apparatus palpated. The joint between the thyrohyoid bone and thyroid cartilage is found. If an endotracheal tube is to be placed without postoperative conversion to feeding tube placement, then the epihyoid bone is palpated and the tube is placed immediately rostral to this bone. The index finger is flexed and moved laterally to create a visible bulge of the skin at this level.

A small incision is made in the skin and subcutaneous tissue over the index finger parallel to the adjacent veins. Depending on the size of the animal, a curved Kelly or Carmalt forceps (Miltex Surgical Instruments. Tuttlingen, Germany), or a long (203 mm) curved forceps such as Johns Hopkins gallbladder forceps (Miltex Surgical Instruments. Tuttlingen, Germany) is placed in the incision and blunt dissection is used to gently separate tissues until the instrument tips are palpated to rest on the pharyngeal wall at the fingertip. The blunt dissection is performed by opening the instrument tips after each repeated insertion into the tissues. The instrument tips are opened parallel to the adjacent veins. Care is taken to avoid all neurovascular structures.

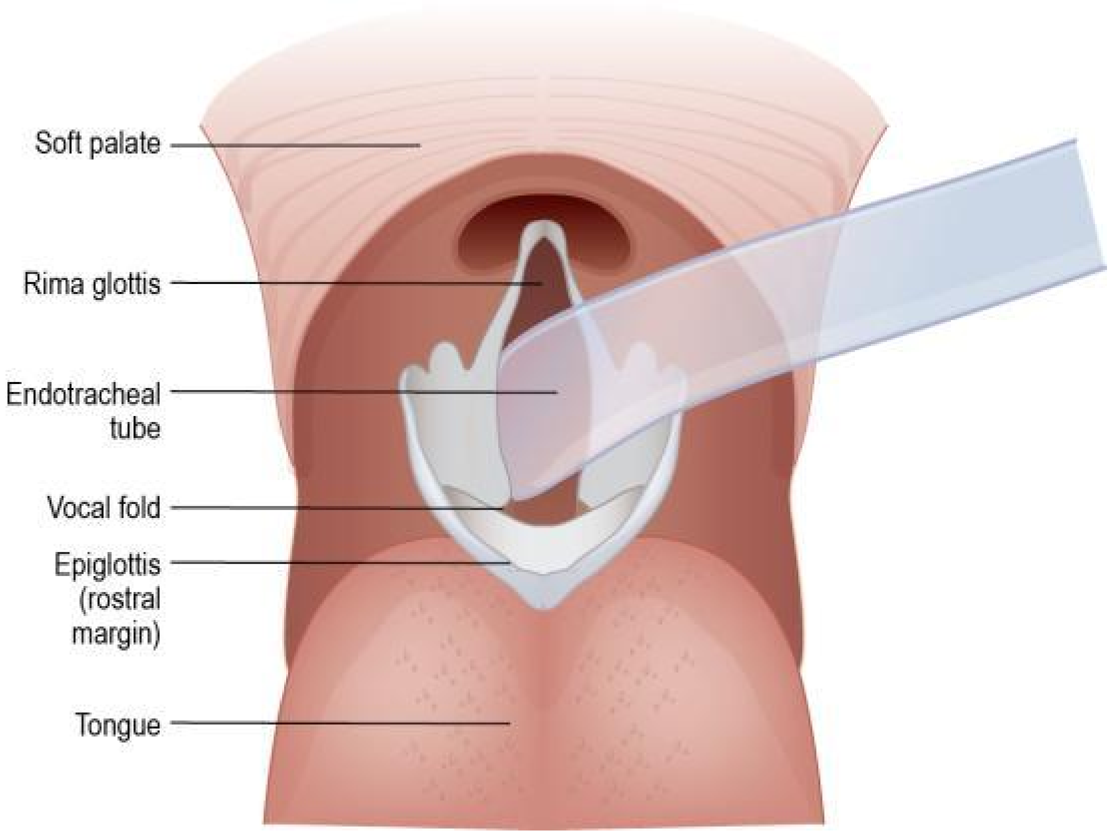
The forceps' tips and index finger are moved medially and the interposed segment of pharyngeal wall is deviated into the pharyngeal lumen. As the pharyngeal wall is viewed via the oral cavity, a small mucosal incision is made in this area and the forceps' tips are advanced into the pharyngeal lumen. An appropriate mucosal opening is made to accommodate the diameter of the tube by stretching the tissue by repeated opening of the forceps jaws. A second pair of forceps is placed in the oral cavity and the tips placed in the open jaws of the first pair. As the first pair of forceps is withdrawn, the second "follows" it into the oral incision and is advanced until its tips are visible at the skin incision. The caudal end of the tube is grasped with the second forceps and the tube drawn through the pharyngeal wall, leaving the rostral portion of the tube exterior to the skin.

Alternatively, the finger used for identifying the landmarks for the skin incision can be replaced by the curved Johns Hopkins gallbladder forceps (Fig. 61.2). The forceps is subsequently pushed in a closed fashion from within the oral cavity through the pharyngeal wall and overlying tissues. As the forceps becomes visible in the skin wound, the instrument tips are opened to separate the subcutaneous tissues and widen the pharyngotomy. The pharyngotomy should be wide enough to draw the tube atraumatically through the opened tissues. The distal end of the tube is grasped with the forceps and pulled through the pharyngeal wall into the pharyngeal lumen.



• **FIG. 61.2** Johns Hopkins gallbladder forceps. Source: (Johnson and Johnson Hospital Services, New Brunswick, NJ.)

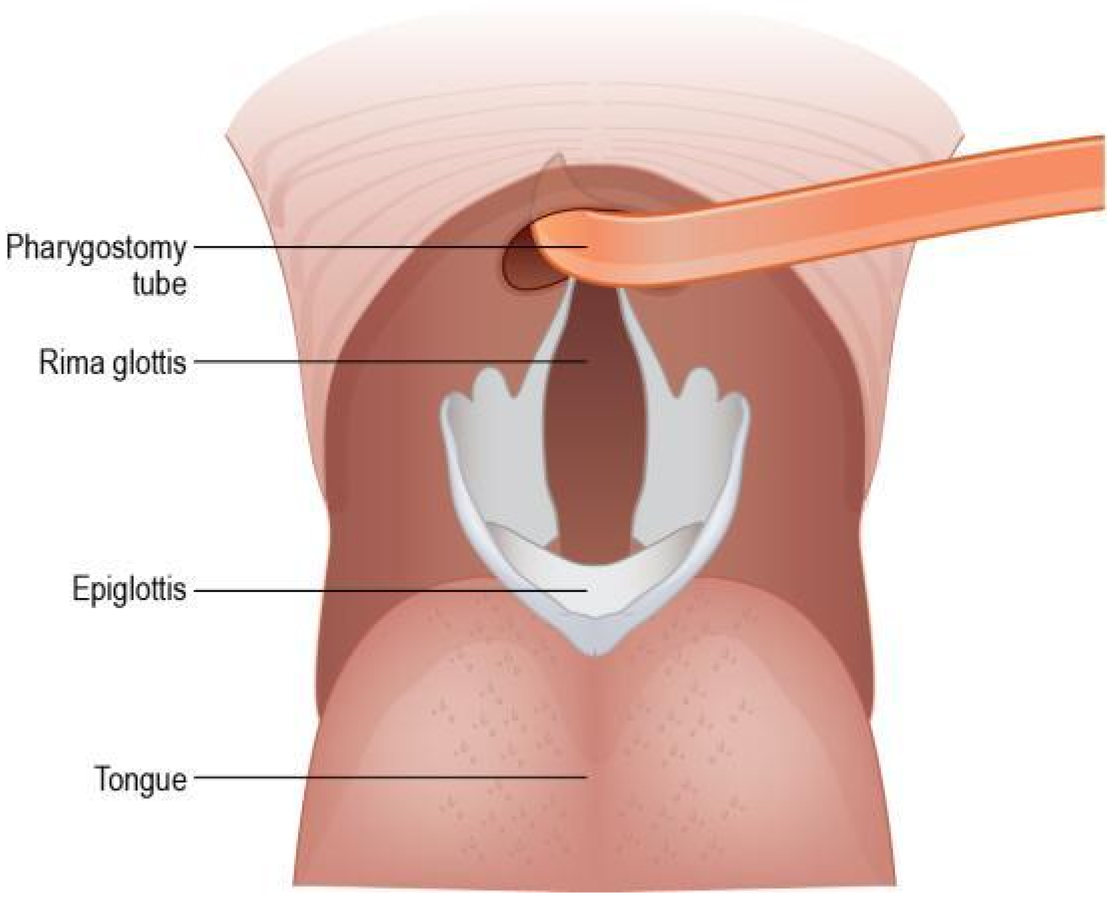
An endotracheal tube is gently guided into the tracheal lumen (Fig. 61.3). The wire-reinforced tubes are fairly stiff and care is taken not to injure mucosa and cartilages of the epiglottis and larynx during tube manipulation. The tube is palpated via the cervical trachea to ensure adequate placement. It is secured into place by a gauze tie around the neck or by means of a tape butterfly attached to the tube and sutured to the skin. The anesthesia hoses can also be taped to the surgery table.



• **FIG. 61.3** Intraoral view showing placement of an endotracheal tube.

In the transmylohyoid orotracheal intubation technique, an incision corresponding to the approximate diameter of the endotracheal tube is made medial to the ventral border of the mandible at the level of the mandibular first molar tooth.[5] The incision is made through the skin and subcutaneous tissue and continued with blunt dissection through the mylohyoid muscle. A second incision is made intraorally through the mucosa overlying the initial extraoral incision to result in a "tunnel" through which the endotracheal tube is passed.[5]

A feeding tube is gently passed into the esophagus and advanced to the mid-thoracic esophagus to the level of the rib at the seventh, eighth, or ninth intercostal space. Obtaining a lateral view, placement verification radiograph, is strongly recommended. Secondary esophageal peristaltic waves will propel nutrient material into the stomach from this point. Placement of the tube at this level will not promote gastric reflux or interfere with esophageal clearance of refluxed gastric acid as may happen if the tube was advanced into the gastric lumen.[10] Laryngeal obstruction and epiglottic entrapment are minimized by passing the tube at the correct level on the pharyngeal wall (Fig. 61.4, and see Fig. 61.1). To secure the tube, a tape butterfly is attached to the tube and sutured to the skin or a purse string suture and a Chinese finger trap suture are placed. The tube should be capped.



• **FIG. 61.4** Intraoral view showing placement of a pharyngostomy tube.

Postoperative care and assessment

Pharyngotomy

The pharyngotomy endotracheal tube is removed immediately after surgery and replaced by a conventional oral endotracheal tube until final extubation is indicated. The pharyngotomy opening is generally left unsutured. Food and water are withheld for 24 hours postoperatively to allow a fibrin "seal" to develop in the tissue tract, meanwhile intravenous fluids are continued. If possible, a light bandage is placed to cover the skin incision. If bandaging is not possible, as is often the case, the skin surface is kept clean and dry as needed. A small amount of serosanguineous drainage may occur for 2 to 4 days and usually resolves spontaneously. If needed, one skin suture may be placed to prevent wide skin gapping during head and neck movement and still allow drainage through an open wound. The wound heals by second intention in 10 to 14 days.

Pharyngostomy

The skin surface around the pharyngostomy tube is cleaned and dried as necessary. Some wound drainage occurs. A neck wrap may be placed around the feeding tube to protect the stoma and the tube. A commercially available specialized collar is available (Kitty Kollar, Jorgensen Laboratories Inc., Loveland, CO) to protect the stoma and the tube and can be changed for hygiene purposes as needed. Eventually, a "cuff" of granulation tissue may be seen between the tube and skin surface. After tube removal, the pharyngocutaneous fistula is not sutured and heals by second intention in 10 to 14 days.

Complications

Hemorrhage is the major intraoperative potential complication. This results from inaccurate placement of the incision and dissection. Observing the distended veins and deviating the index finger to tent the skin laterally before making the incision will displace the neurovascular structures from the incision site. The incision and subsequent blunt dissection must be performed parallel to the veins.

Injury to the mucosal surfaces during excessively traumatic endotracheal tube placement may result in postoperative cough secondary to laryngitis. Fracture of cartilages may result in abnormal epiglottic movement and laryngeal airway obstruction.

Placing a pharyngostomy tube is typically a safe technique but has potential to cause several complications.[4,11] Epiglottic entrapment or reduced movement due to the tube increases the risk of aspiration pneumonia. Larger-diameter tubes may obstruct the *aditus laryngis*. These complications can be minimized by accurate tube placement.[4] The tube is located in an area of normal movement and the exterior part of the tube may contact the side of the head during movement. The presence of the tube may create stress and discomfort for the patient. The tube may become dislodged from the esophagus, resulting in excess tube in the oropharyngeal area or hanging from the mouth. The feeding tube may become occluded with food material. Therefore it is recommended to flush the tube with water after each feeding. Patients may not voluntarily eat with the tube placed, and it may be difficult to assess ability to adequately eat and drink during the course of recovery. Local infection and swelling may occur. Persistent pharyngocutaneous fistula is a potential rare complication that may occur and should be surgically resolved. Larger stiff tubes may cause pressure necrosis.

The advent of improved techniques and instrumentation for placement of esophagostomy and gastrostomy tubes has essentially replaced the use of the pharyngostomy tube for parenteral feeding (see Chapter 5).[12–14]

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CHAPTER 62

Oral approaches to the nasal cavity and nasopharynx

Kyle G. Mathews

Surgical anatomy

The bony portion of the hard palate is made up of the paired palatine processes of the incisive bones rostrally, the palatine processes of the maxillae centrally, and the horizontal laminae of the palatine bones caudally.[1] There is a midline eminence at the caudal border of the hard palate referred to as the caudal nasal spine. The bones of the hard palate are covered with a thick, durable, and vascular mucoperiosteum ventrally, and a pseudostratified ciliated columnar epithelium on the nasal surface. Blood supply to the hard palate is supplied primarily from the paired major palatine arteries, and to a lesser extent from the minor palatine arteries caudally, and the sphenopalatine arteries dorsally. Each major palatine artery runs through a major palatine foramen, between the maxillae and palatine bones, and then rostrally beneath the mucoperiosteum within a groove, called the palatine sulcus, in the ventral surface of the hard palate. Numerous anastomoses exist between these paired arteries. Venous drainage is via a diffuse venous plexus to the maxillary vein. Sensory innervation is supplied to the oral surface of the hard palate by the major palatine branch of the maxillary division of the trigeminal nerve, which runs along with the major palatine artery in the palatine canal. For details of soft palate anatomy, see Chapter 60.

Special instruments and materials

An oral surgery drill unit (see Chapter 7), a piezosurgery unit (see Chapter 9), or an orthopedic nitrogen or battery-powered unit (e.g., Surgairtome, Hall Surgical, Linvatec Corporation, Largo, FL), headlamp or other supplemental light source, electrocautery unit, Lempert rongeurs, curettes, small Gelpi retractors, cold saline, suction, and poliglecaprone 25 suture (Monocryl, Ethicon, Inc. Somerville, NJ) are required. For temporary carotid artery occlusion, umbilical tape, 22 G orthopedic wire, and a 10–12 F red rubber catheter are used.

Therapeutic-decision making

Nasal or nasopharyngeal disease should be suspected in any animal with nasal discharge, sneezing, stertorous respiration, nasal planum ulceration, epistaxis, or changes in phonation. Appropriate diagnostic tests may include nasal and oral (including hard and soft palate) palpation, nasal and thoracic computed tomography (CT) or radiography (less ideal), and rhinoscopy in addition to a complete blood count, serum chemistry panel, fungal titers, bacterial and fungal cultures, and cytology/histopathology.

Indications for surgery

The ventral approach to the nasal cavity can be used as an alternative to dorsal rhinotomy for the removal of foreign bodies that are not retrievable via rhinoscopy and that are located fairly ventrally, to debulk fungal granulomas as an adjunct to topical or systemic antifungal therapy, and potentially as an adjunct to radiation therapy for nasal tumors.[2,3] Subcutaneous emphysema, which can occur following a dorsal rhinotomy, is not a problem with the ventral approach. Clients may consider the ventral approach to be cosmetically more acceptable since the incision is hidden from view. Also, if carotid artery ligation is to be performed, repositioning the animal is not necessary. The decision to approach a nasal lesion ventrally should be based on preoperative radiography, CT or rhinoscopy. Fungal granulomas in the frontal sinuses are more common than those in the ventral nasal cavity, and should be approached via a dorsal sinusotomy. Lesions in the ventral ethmoid turbinates, or rostral to the ethmoid turbinates, can be approached through the hard palate.

Access to the nasopharynx can be gained by incision of the soft palate alone. Fifty-four percent (20 of 37) of dogs with nasopharyngeal disorders in one study were diagnosed with neoplasia.[4] The other dogs in this study had either noninfectious inflammatory tissue or benign cysts obstructing the nasopharynx. Nasopharyngeal granulomas associated with cryptococcal infection have also been reported in both cats and dogs.[5,6] If the granuloma is large, removal with a flexible bronchoscope is usually not possible. Removal of large nasopharyngeal foreign bodies or polyps, and surgical excision of nasopharyngeal stenosis may also require splitting of the soft palate. Nasopharyngeal stenosis is most common in cats and is thought to be acquired secondary to chronic upper respiratory tract infections.[7] The author has seen nasopharyngeal stenosis occur in a dog secondary to vomiting, which presumably resulted in irritation of the nasopharynx. Excision of the stenosis and temporary stenting with a large-diameter nasal Foley catheter resulted in resolution of the problem. Congenital choanal atresia has also been reported in the dog.[8] Successful correction of nasopharyngeal stenosis has been reported following excision with or without a mucosal flap, excision with placement of a nasopharyngeal stent, following balloon dilation, and following placement of a balloon-expandable Stent.[7,9-12]

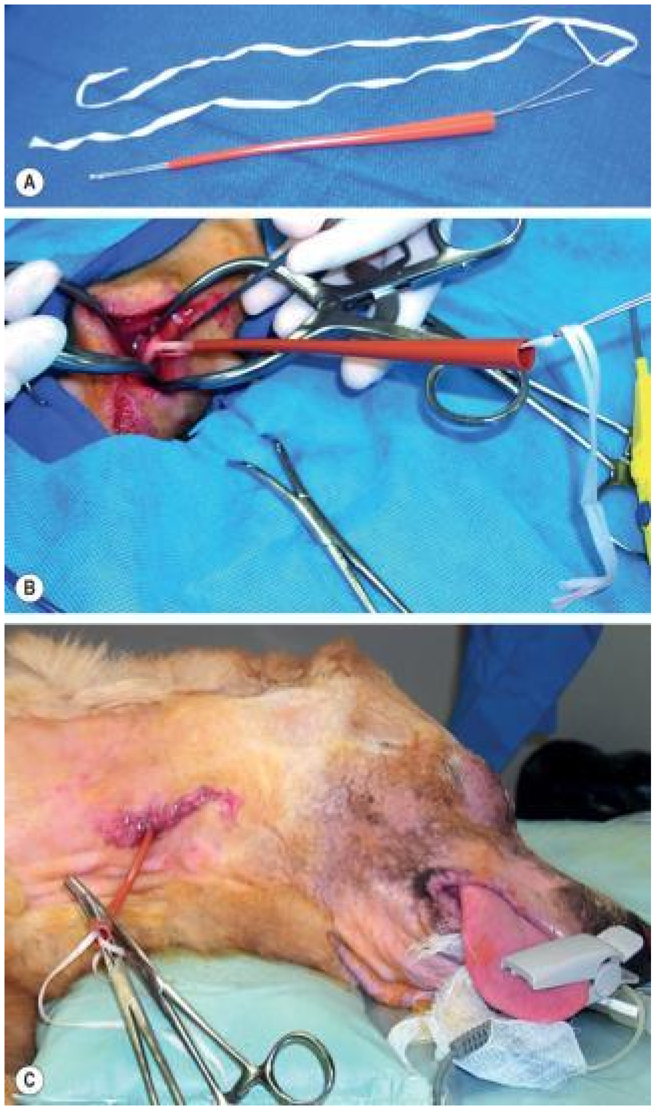
Surgical technique

Control of hemorrhage

Because hemorrhage during ventral approaches to the nasal cavity is expected, coagulation profiles, cross-matching, and the potential need for a transfusion should be considered prior to surgery.

Hemorrhage during a ventral approach to the nasal cavity (ventral rhinotomy) may be diminished by temporary bilateral carotid artery ligation. Because of well-developed collateral circulation originating from the vertebral arteries in dogs, this procedure can be performed with no notable adverse effects. There are at least five described sites of anastomoses between the internal and external carotid arteries in a normal dog, allowing them to survive with no residual effect following long-term ligation of one or both carotid arteries and other major vessels supplying the head.[13-17] Bilateral carotid ligation may result in death in cats; therefore ligation is limited to one side.[3,18] Carotid artery ligation has been shown to decrease lingual arterial pressures in dogs.[19] Similar diminution in blood pressure should occur in the major and minor palatine arteries, since they arise from the maxillary artery which, as with the lingual artery, arises from the common carotid artery. Although the ventral approach through the hard and soft palates will likely be associated with less hemorrhage following carotid artery ligation, it is unclear if the same is true when performing nasal turbinate excision.[1,20]

The common carotid arteries are approached by making a ventral midline incision in the mid-cervical region, or two smaller parasagittal incisions ventral to the jugular groove (see also Chapter 48). The carotid sheath is identified and opened longitudinally with Metzenbaum scissors. Care must be taken to avoid stretching or otherwise damaging the vagosympathetic trunk and internal jugular vein, which also lie within the carotid sheath. Once the carotid artery has been isolated, a Rummel tourniquet is fashioned by passing a strand of moistened 6-mm umbilical tape around the artery (Fig. 62.1A). The two ends of the umbilical tape are drawn through a 60–100-mm section of red-rubber catheter (Fig. 62.1B). This is accomplished by first passing some thin orthopedic wire through the catheter segment. The tourniquet is tightened by pushing the catheter against the artery while maintaining tension on the umbilical tape. A hemostat is placed across the catheter to maintain occlusion of blood flow, and the incision is closed around the tourniquet (Fig. 62.1C). When the oral procedure has been completed, the hemostat, catheter, and umbilical tape are easily removed.



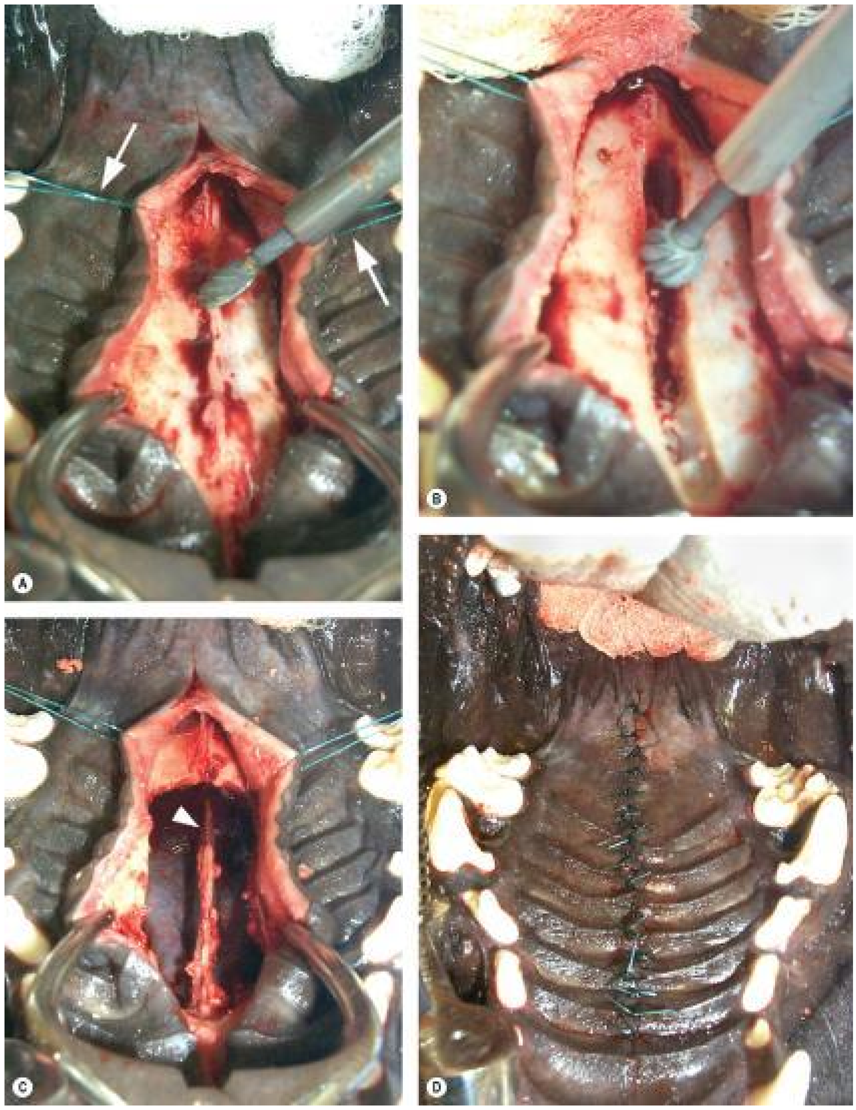
• **FIG. 62.1** Temporary carotid artery occlusion in the dog: (**A**) Supplies needed for the Rummel tourniquet *(see text)*. (**B**) After carefully dissecting the carotid artery out of its sheath, the umbilical tape is passed around the artery. The ends of the umbilical tape are passed through the doubled-over orthopedic wire and pulled through the red-rubber catheter. (**C**) The carotid artery is occluded by pushing the catheter against the artery while applying tension to the umbilical tape. Occlusion is maintained by placing hemostats across the catheter and umbilical tape. The incision is closed around the Rummel tourniquet, which is easily removed following the rhinotomy.

After penetrating the hard palate, further hemorrhage can be expected from the nasal mucosa, which is richly endowed with blood vessels. Lavage of the nasal cavity with cold saline will improve visualization. Several bottles of saline should be refrigerated the day prior to surgery. Instillation of epinephrine (1:100,000) may also improve visualization, but the application of cold saline is usually sufficient. Suction should be used; however, the surgeon must keep track of blood loss through the suction unit. Direct pressure can be obtained by temporarily packing the surgery site with cold saline-soaked sponges before proceeding. Bipolar electrocautery can be used to carefully cauterize individual vessels.

Ventral approach to the rostral nasal passages

Following anesthetic induction, a single dose of antibiotic is given (see Chapter 3). An infraorbital nerve block is performed as outlined in Chapter 4. The patient is placed in dorsal recumbency with the surgical table positioned to form a trough. The thoracic limbs are tied caudally. A rolled towel is placed under the patient's neck for support. If carotid artery ligation is planned, the ventral and lateral cervical area is clipped and aseptically prepared. The palate is maintained in a horizontal position by placing a long strip of tape from the table over the maxillary canine teeth. The mouth is held open by tying or taping the mandible up to an intravenous pole on either side of the surgical table. The endotracheal tube should be elevated along with the mandible away from the hard palate. Gauze sponges are counted and placed in the pharynx, and the endotracheal tube is inflated to prevent aspiration of blood and saline flush during the procedure. The palate is then aseptically prepared for surgery.

Following carotid artery ligation, if performed, a midline incision is made through the mucoperiosteum directly over the area of interest, identified with CT. The mucoperiosteum is elevated from the hard palate with a periosteal elevator. The paired major palatine arteries penetrate the hard palate approximately half- way between the midline and the caudal aspects of the fourth premolar teeth. Lateral mucoperiosteal elevation to the level of the palatine arteries usually achieves adequate exposure. Exposure is maintained by placing small Gelpi retractors into the incision, or by use of several stay sutures placed through the cut edges of the mucoperiosteum (Fig. 62.2A).



• FIG. 62.2 (**A**) With the patient in dorsal recumbency and the hard palate in a horizontal position, a midline incision is made in the palatine mucoperiosteum. The edges of the mucoperiosteum are retracted with Gelpi retractors and stay sutures (*arrows*) to expose the hard palate, which is then penetrated with a rotating bur. Extension of the midline incision caudally through the soft palate may be performed, if necessary. (**B**) The hard palate is removed with a rotating bur over the area of interest. Continuous lavage and suction are required to prevent overheating and improve visibility. (**C**) The nasal cavity is exposed. The vomer *(arrowhead)* and affected turbinates are removed with rongeurs and curettes, if necessary. (**D**) The mucoperiosteum is closed with numerous simple interrupted absorbable sutures. Extra throws are placed on the sutures to prevent premature untying.

A longitudinally oriented trough is then created in the hard palate with a round bur on an oral surgery unit or nitrogen or battery-powered orthopedic drill (Fig. 62.2B). The trough is centered on the midline for bilateral lesions, or can be started to the side of midline for unilateral lesions. Once through the hard palate into the ventral nasal cavity, the trough may be extended in any direction to improve access to the tissues of interest. If irrigation is not provided through the handpiece, cold saline should be continuously dripped on the bur during bone removal to prevent overheating. Continuous suction is used to remove bone debris and improve visibility. Hemorrhage is controlled as outlined above. The vertically oriented vomer and/or nasal septum may be trimmed, if necessary, with Lempert rongeurs (Fig. 62.2C). Diseased tissues, fungal granulomas, and foreign bodies within the nasal cavity are removed with curettes and/or rongeurs.

Once the objectives of the surgery have been achieved, hemorrhage is under control, and the nasal cavity has been thoroughly flushed with saline, the tissues are closed. A single row of simple interrupted monofilament absorbable sutures (e.g., Monocryl, Ethicon, Inc. Somerville, NJ) are placed in the mucoperiosteum overlying the hard palate (Fig. 62.2D). Extra throws are placed on these knots to keep them from untying due to tongue motion. The pharyngeal gauze sponges are recounted and the pharynx is suctioned prior to recovery from anesthesia.

Ventral approach to the caudal nasal passages and nasopharynx

Positioning of the patient is identical to that described for ventral approach to the rostral nasal passages.

A midline incision is made through the mucosa of the soft palate. The caudal edge of the soft palate should be preserved to simplify closure. The incision is continued through the full thickness of the soft palate between the paired palatine muscles, and through the nasopharyngeal mucosa. Exposure to this area is improved by placing stay sutures along the incision margins to retract the tissues laterally. Small Gelpi retractors are also quite helpful. If the nasopharyngeal area of interest is near the caudal edge of the hard palate, the two approaches may be combined. The incision may be extended cranially through the palatine mucoperiosteum. A portion of the caudal hard palate is then removed along the midline.

Once the nasopharyngeal obstruction (stricture, mass, fungal granuloma, foreign body) has been removed, closure of the soft palate is accomplished in two or three layers. Absorbable continuous sutures are placed in the nasopharyngeal mucosa, followed by similar apposition of the palatine muscles. Simple interrupted or continuous monofilament absorbable sutures (e.g., Monocryl, Ethicon, Inc. Somerville, NJ) are placed in the oropharyngeal mucosa. As before, extra throws are generally placed on these knots to keep them from untying due to tongue motion.

Postoperative care and assessment

Most animals will begin eating the day following surgery and can be discharged from the hospital within 1–2 days. A soft diet is fed for 2 weeks. A gastrostomy or esophagostomy tube may need to be placed in some animals. Cats with caudal nasal disease, in particular, may not eat and will require caloric supplementation for several days postoperatively (see Chapter 5). A fentanyl patch should be considered, placed the day prior to surgery if possible, and removed 3–5 days postoperatively (see Chapter 4). Additional analgesia should be provided with an injectable narcotic (e.g., hydromorphone, as needed) while in the hospital. Nonsteroidal antiinflammatory drugs should also be considered (e.g., dogs: carprofen 2 mg/kg, PO, BID for 1 week, then q24 h as needed; cats: ketoprofen 1 mg/kg, PO, q24h for 5 days). The surgical site should be inspected weekly for 2–3 weeks so that areas of dehiscence may be detected early and resutured.

Prognosis

Ventral rhinotomy was performed on 11 cats and 47 dogs in one study.[3] Two cats died in the postoperative period. Both had significant hemorrhage intraoperatively, requiring temporary carotid artery ligation. Decreased blood flow to the brain during surgery likely contributed to their deaths. Persistent mucopurulent or serous nasal discharge occurred in the majority of animals. The cause of postoperative nasal discharge is not understood, although it also occurs following dorsal rhinotomy with turbinectomy. Dehiscence was rarely a problem, with one patient developing an oronasal fistula.

Long-term prognosis depends on the underlying disease process. Most cases of canine and feline fungal rhinitis can be cured with topical and/or systemic antifungal therapy with surgical removal of granulomas, when present.[5,21] Nasopharyngeal cysts, polyps, and stenoses respond favorably to surgical intervention. Nasal and nasopharyngeal neoplasia carries a guarded to poor prognosis. Performing rhinotomy and aggressive surgical debulking prior to orthovoltage radiation for the treatment of nasal carcinomas improved survival times compared to orthovoltage therapy alone in several studies.[22,23] Affected animals may also temporarily benefit from surgical debulking of tumors that impede airflow through the nasal cavity or nasopharynx. Megavoltage therapy of canine nasal carcinoma is commonly performed, and survival times are similar to those reported for combined surgery and orthovoltage therapy.[24-26] Surgical debulking prior to megavoltage therapy is not recommended as it may result in uneven dose distribution within the tumor.[25] The potential benefits of surgical debulking of selected tumors following megavoltage therapy is unclear.[26,27] Stereotactic radiation therapy (SRT) is emerging as the preferred treatment for intranasal tumors. To date, three papers have described outcomes associated with three different SRT delivery techniques with median survival times ranging from 8.5 to 49 months.[28-30] Neither tumor stage nor type was found to be predictive of outcome. SRT appears to be of similar efficacy as compared with conventional full-course megavoltage RT for low-stage tumors with relatively bland histology. Outcomes appear to be improved for dogs with cribriform lysis, and/or aggressive tumor biology (e.g., anaplastic sarcomas and squamous cell carcinoma).

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CHAPTER 63

Tonsillectomy

Kyle G. Mathews

Surgical anatomy

The palatine tonsils are located in the dorsolateral walls of the oropharynx just caudal to the caudolateral borders of the soft palate (palatoglossal folds) and are recessed within the tonsillar fossa in normal dogs.[1] They have no afferent lymphatics, but numerous efferents that empty into the medial retropharyngeal lymph nodes. The lingual artery gives rise to the tonsillar artery, which branches 2–3 times as it enters the base of each tonsil. Numerous small veins leave each tonsil and empty into the palatine venous plexus.

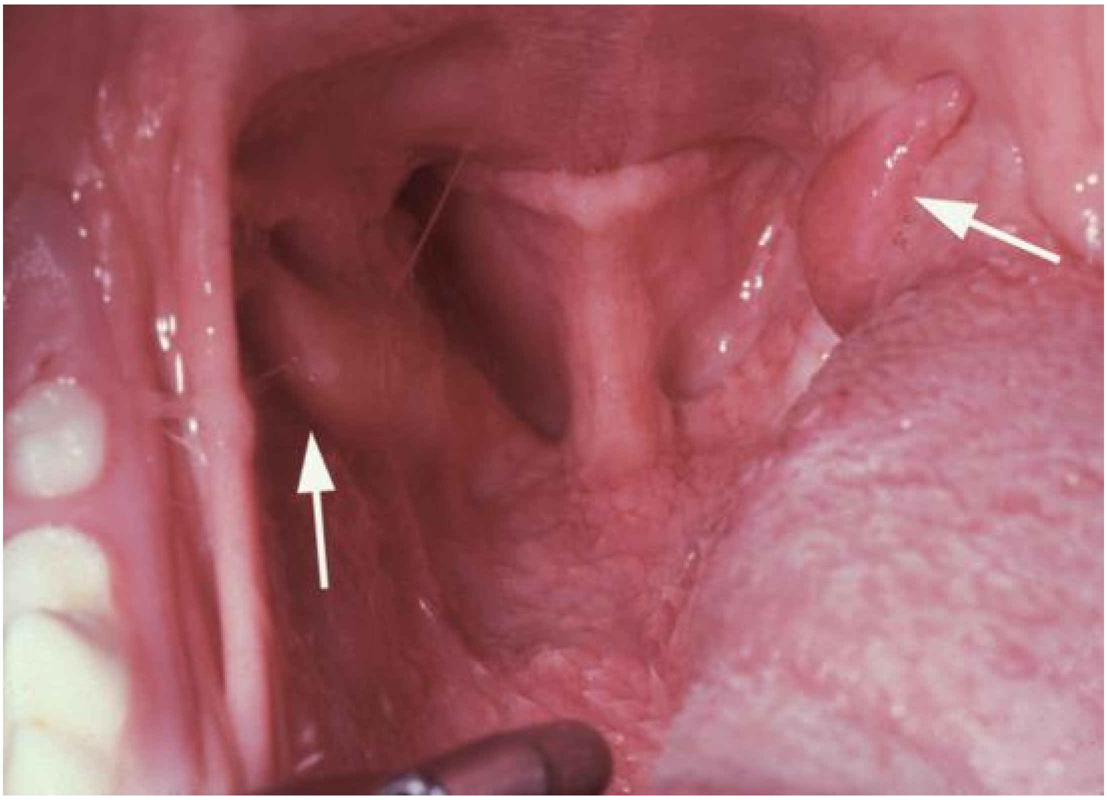
Special instruments and materials

All instruments should have handles that are at least 180 mm in length: Kelly hemostats, right-angled forceps, or hemostatic tonsil forceps, e.g., Schnidt or Sawtell forceps (Sklar, West Chester, PA); Metzenbaum scissors or tonsil scissors, e.g., Dean or Boettcher tonsil scissors (Sklar, West Chester, PA); DeBakey forceps or tonsil forceps, e.g., White or Colver-Coakley forceps (Sklar, West Chester, PA); long-handled needle holders. A headlamp or other supplemental light source is very useful. Recommended suture material is 3–0 to 4–0 poliglecaprone 25 (Monocryl, Ethicon, Inc. Somerville, NJ) or polyglactin 910 (Vicryl, Ethicon, Inc. Somerville, NJ). An electrocautery unit (bipolar preferred) is often needed if a bipolar vessel sealing device (e.g., LigaSure, Covidien, Medtronic, Minneapolis, MN) is not available.

Therapeutic decision-making

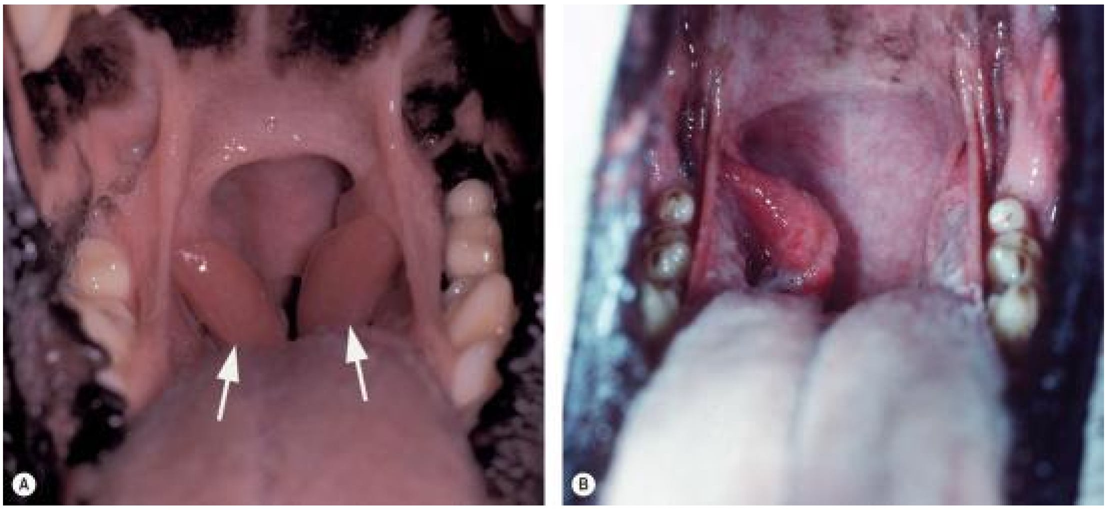
Indications for surgery

Tonsillectomy is indicated in cases of chronic recurrent tonsillitis unresponsive to antibiotic therapy, as an adjunct to radiation therapy for dogs with tonsillar squamous cell carcinoma (SCC), and for removal of benign tonsillar polyps and cysts.[2,3] It may also be required in dogs with brachycephalic syndrome if the tonsils have enlarged to the point where they contribute to airway obstruction.[4] Some surgeons prefer to remove the tonsils in all dogs presented for soft palate and everted laryngeal ventricle resection (Fig. 63.1).[5] The author has not found tonsillectomy to be necessary in the large majority of dogs with this condition. Correction of the airway obstruction due to brachycephalic syndrome presumably results in diminution of negative pressure generated during inhalation, and return of the tonsils to their fossae.



• FIG. 63.1 Bilateral tonsillar enlargement *(arrows)* and eversion from the tonsillar fossae is frequently seen in dogs with brachycephalic syndrome. Resection is only necessary if the enlarged tonsils are contributing to airway obstruction.

Malignant lymphoma may also affect the tonsils, but animals with tonsillar lymphoma tend to have uniform bilateral enlargement (Fig. 63.2A) rather than the irregular, firm, unilateral enlargement seen with tonsillar carcinoma (Fig. 63.2B).[6] In addition, animals with lymphoma generally have multiple other sites of involvement, including distant lymph nodes such as the mediastinal and sternal nodes, seen on thoracic radiography. Dogs with tonsillar carcinoma, which is rare in cats, often have only local pharyngeal and mandibular lymphadenopathy, although they may have lung metastases at diagnosis. Both occur in older animals. The mean age for tonsillar carcinomas is between 8 and 11 years.[6-10] A diagnosis of malignant lymphoma is based on nodal or tonsillar fine needle aspirates and biopsies. Chemotherapy is instituted. Tonsillectomy is not indicated.



• **FIG. 63.2** Tonsillar neoplasia: (**A**) Bilateral tonsillar enlargement *(arrows)* in a dog with malignant lymphoma. (**B**) Unilateral tonsillar squamous cell carcinoma in a dog.

SCC is the most commonly reported tonsillar tumor in dogs.[6-13] Several studies have reported a higher incidence of tonsillar SCC in urban areas, presumably due to increased exposure to environmental carcinogens.[6,7,10,13] This tumor may also occur more frequently in male dogs than in females.[6-9] Tonsillar SCCs are rapidly growing, infiltrative, and rarely resectable. Because the tonsils have multiple efferent lymphatics, and because most tumors go unnoticed until they are large enough to cause clinical symptoms (dysphagia, ptyalism, cough, gagging, anorexia, dyspnea, cervical swelling, voice change), most affected animals have regional lymph node metastasis at the time of diagnosis. Distant metastases to the lungs, liver, and other sites are also commonly reported.[6-8]

Surgery for dogs with tonsillar SCC is limited to debulking prior to radiotherapy. Radical neck dissection to remove all gross tumor and affected nodes is generally not performed in dogs due to the risk to local structures. Biopsy of the ipsilateral and contralateral mandibular lymph nodes, and of the contralateral tonsil, for staging purposes should be considered. One study reported that 8 of 24 dogs had involvement of the contralateral lymph nodes.[6] Contralateral tonsillar involvement has also been reported[7] without gross enlargement of the tonsil. Affected nodes are generally included in the radiation field.

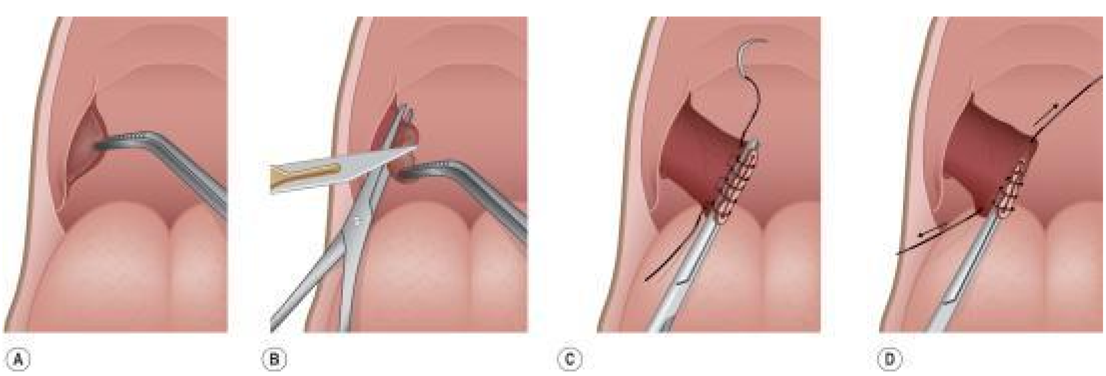
Chronic tonsillitis (bacterial or viral) is a disease of young animals, usually <1 year of age. It is usually a component of viral pharyngitis.[14,15] The tonsils can be 2–3 times their normal size, firm and hyperemic. The animal may be systemically ill with a fever, leukocytosis or leukopenia, and be malnourished and dehydrated. If pharyngitis is present, the animal may hold its neck in an extended position, exhibit pain on palpation, and have difficulty swallowing. Once stabilized with fluid therapy, antibiotics, and nutritional support, tonsillectomy may be considered if prior medical management has not resulted in more than temporary resolution of the problem.

Surgical technique

Although shortening of the soft palate with a CO_2 laser has been reported in dogs, laser tonsillectomy has not. Laser tonsillectomy results in less intraoperative hemorrhage and is commonly and successfully performed in humans.[16,17] The use of lasers for this procedure in dogs would be a logical next step and is described in Chapter 10.

Tonsillectomy in dogs using a bipolar vessel sealing device (LigaSure) has been described and greatly simplifies the procedure while providing effective hemostasis.[18,19]

A variety of conventional tonsillectomy techniques have been reported in dogs. In each case, the animal is placed in sternal recumbency with its head elevated. A tape or gauze sling is created between two intravenous poles. The sling is placed just caudal to the maxillary canine teeth so that the dorsal aspect of the animal's nose is horizontal. A mouth gag is placed as well as a known number of pharyngeal sponges. The cuff of the endotracheal tube is inflated to prevent aspiration of blood. Adequate lighting is required—a headlamp is preferable. While an assistant pulls the tongue out and downward, the surgeon grasps the tonsil with forceps or hemostats and elevates it from its fossa. At this point the tonsil can be pulled through a tonsil snare and the base cauterized.[20] Alternative techniques include transection of the base of the tonsil with Metzenbaum scissors, followed by ligation or bipolar cauterization of tonsillar artery branches and/or digital pressure.[4,21,22] Others prefer to cross-clamp the base of the tonsil with hemostats prior to transection.[5] Continuous sutures are then placed around the hemostats (Parker-Kerr suture pattern) (Fig. 63.3). Upon removal of the hemostat, the suture line is tightened and tied. If individual vessel ligation or cauterization is performed, some surgeons prefer to oversew the fossa with continuous sutures,[4,22,23] while others leave the site open.[20] Transection of the tonsillar base with monopolar cautery has also been suggested, although concerns regarding tissue swelling should limit its use.[24] The use of epinephrine injections (1:5000) into the base of the tonsil followed by digital pressure with epinephrine-soaked sponges has also been reported to control hemorrhage.[25] There is some theoretical concern with injecting epinephrine into a highly vascular region,[15] although complications with this technique have not been reported. In the end, technique is based on personal preference and available materials.



• **FIG. 63.3** Tonsillectomy technique: (**A**) The base of the affected tonsil is clamped with hemostats or right-angled forceps after applying traction to the tonsil. (**B**) The tonsil is resected. (**C**) The base of the tonsil and clamp are oversewn with a Parker-Kerr suture pattern. (**D**) Following clamp removal, the suture line is tightened and tied.

Postoperative care and assessment

Despite contamination at the time of surgery, prolonged antibiotic therapy is rarely indicated unless the animal is systemically affected by bacterial tonsillitis/pharyngitis. If tonsillectomy is to be performed for noninfectious reasons, a single dose of corticosteroids is generally given at induction, prior to manipulation of the tissues, to diminish postoperative swelling. Prior to recovery from anesthesia the pharynx is thoroughly suctioned, and the pharyngeal sponges counted. Patients should be monitored closely for the first 24 hours for evidence of respiratory distress secondary to swelling. An emergency tracheostomy kit and supplemental oxygen should be readily available, if needed. Soft food is fed for the first 3–7 days.

Prognosis

The prognosis for uneventful recovery following tonsillectomy is good for dogs in which it is performed as an adjunct to soft palate resection, and for the treatment of chronic tonsillitis. Postoperative complications such as excessive hemorrhage and infection are uncommon.

Tonsillar SCC carries a poor prognosis even following tonsillectomy and radiation therapy. Many dogs respond to radiation therapy, but because most tonsillar carcinomas have infiltrated local tissues and metastasized by the time of diagnosis, survival is often short.[9,11] Median survival in eight dogs treated with 35–42.5 Gy of megavoltage radiation following tonsillectomy was 110 days.[11] In two other studies, median survival from the time of diagnosis was 211 and 270 days following tonsillectomy, radiotherapy and chemotherapy.[9,26] There is evidence to suggest that some dogs with tonsillar SCC may benefit from the administration of piroxicam, although a large prospective study is needed.[27]

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