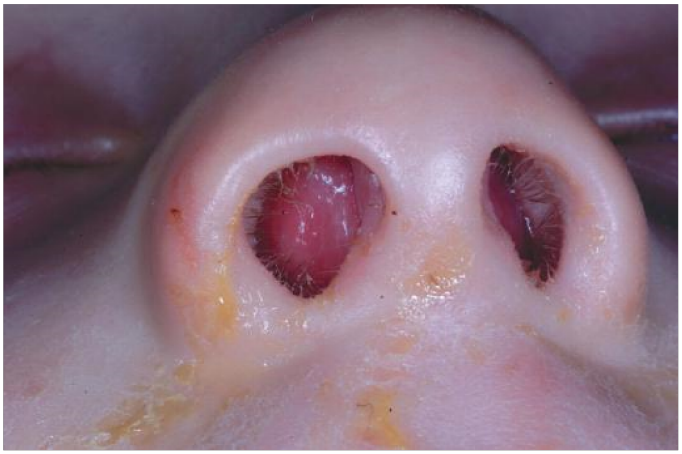


FIGURE 244-14. Trauma patient with nasal septal hematoma in the right nares. [Photo contributed by Lawrence B. Stack, MD. Reproduced with permission from Knoop K, Stack L, Storrow A, Thurman RJ: *Atlas of Emergency Medicine*, 3rd ed. © 2010, McGraw-Hill, New York.]

teeth to evaluate for extension of disease. Subacute and chronic sinusitis persist for 4 weeks or more.

DIAGNOSIS

The diagnosis of uncomplicated **acute rhinosinusitis** is clinical.[37] Plain sinus radiographs or CT scans are not needed. The symptoms of acute rhinosinusitis are similar to a common cold or viral upper respiratory infection, but symptom duration ranges from 7 days to up to 12 weeks.[37] CT scans are helpful to diagnose complications in a toxic patient or to evaluate for intracranial extension. The differential diagnosis of rhinosinusitis includes migraine headache, craniofacial neoplasm, foreign body retention, and dental caries.

TREATMENT

The treatment for **acute uncomplicated rhinosinusitis** is generally supportive. Nasal saline irrigation alone, or in conjunction with other adjunctive measures like nasal decongestants, may decrease symptom severity.[34] Restrict the use of topical decongestants like oxymetazoline to approximately 3 days to avoid rebound mucosal congestion or edema (rhinitis medicamentosa). Topical (intranasal spray) corticosteroids may shorten the duration of illness.[38]

In a 2014 Cochrane database systematic review analyzing the efficacy of antibiotic therapy, the authors concluded that antibiotics may provide a small treatment effect in patients with symptoms of rhinosinusitis lasting >7 days.[33] However, because 80% of patients treated with placebo also improved within 2 weeks, it is unclear whether the treatment effect is clinically significant. In general, **antibiotics should be reserved for patients with purulent nasal secretions and severe symptoms for ≥7 days.** If antibiotics are prescribed, amoxicillin is recommended as first-line therapy for most adults,[37] at a dose of 500 milligrams PO three times per day. Patients with penicillin allergies may receive macrolide antibiotics or trimethoprim-sulfamethoxazole.[37] For patients who have received antibiotics within the past 4 to 6 weeks, consider a fluoroquinolone or high-dose amoxicillin-clavulanate.[34] Use caution in selection of antibiotics in patients who are on oral anticoagulation.[19] In the aforementioned Cochrane review, comparisons between different classes of antibiotics showed no significant difference.[33] Follow-up with a primary care provider is advised.

Patients with **subacute, chronic or recurrent rhinosinusitis** should be evaluated for conditions that modify management, such as allergy, cystic fibrosis, or immunocompromise. Outpatient noncontrasted CT of the sinuses can evaluate for invasion of neighboring tissues and neoplasms.[34] Bacterial cultures may be helpful to tailor therapy in outpatients who are at risk for multidrug-resistant organisms. ENT follow-up is advised.[37,39]

COMPLICATIONS

Complications of rhinosinusitis are mostly related to extension of the infection beyond usual anatomic boundaries. Meningitis, cavernous sinus thrombosis, and intracranial abscesses are rare but important complications associated with contiguous spread of sinus disease. Up to 75% of cases of orbital cellulitis, which can lead to blindness through venous congestion and ischemia of the optic nerve, are attributable to disease of the sinuses.[37] Frontal sinusitis can lead to osteomyelitis of the frontal bone with a doughy swelling of the forehead called *Pott's puffy tumor*, and can also be associated with an extradural or subdural empyema. In general, patients with these deeper infections usually appear systemically ill or have focal neurologic signs and require admission and IV antibiotics.

REFERENCES

The complete reference list is available online at www.TintinalliEM.com.

CHAPTER 245

Oral and Dental Emergencies

Ronald W. Beaudreau

ORAL AND DENTAL ANATOMY

The normal adult dentition consists of 32 permanent teeth. The adult dentition has four types of teeth: 8 incisors, 4 canines, 8 premolars, and 12 molars. The primary or deciduous dentition consists of 20 teeth of three types: 8 incisors, 4 canines, and 8 molars. **Figure 245-1** shows the

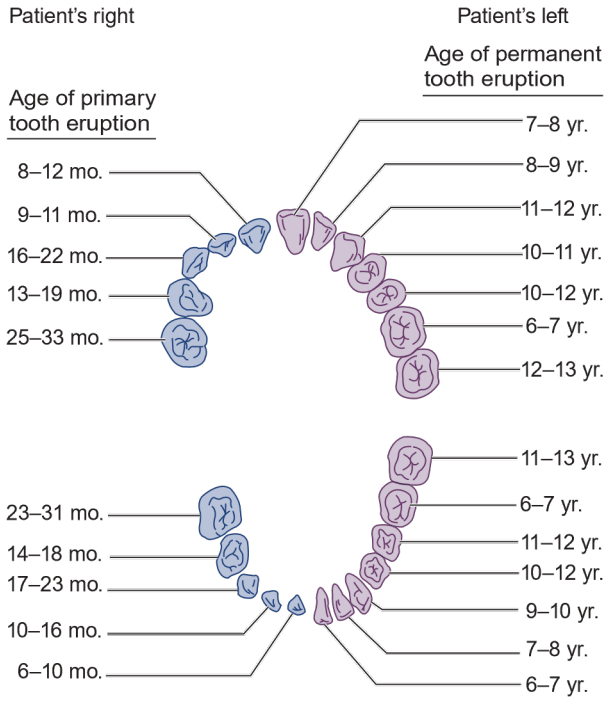


FIGURE 245-1. Normal eruptive patterns of the primary and permanent dentition. mo. = month; yr. = years.

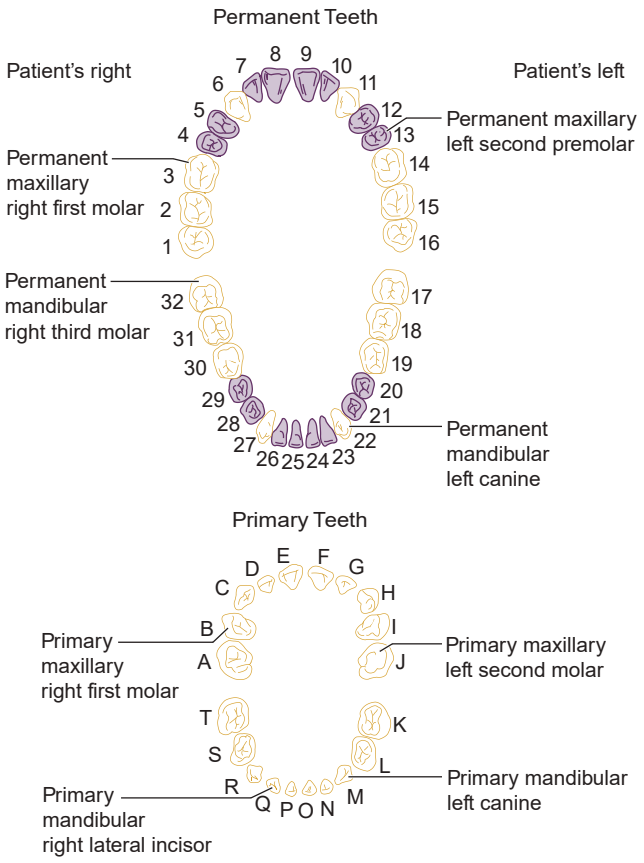


FIGURE 245-2. Identification of teeth.

eruptive pattern of both the primary and permanent dentition. **Figure 245-2** illustrates the most commonly used tooth numbering system; however, description of the tooth type and location is also appropriate.

ANATOMY OF THE TEETH

A tooth consists largely of *dentin,* which surrounds the *pulp*, the tooth's neurovascular supply (**Figure 245-3**). Dentin is a homogeneous material produced by pulpal odontoblasts throughout life. Dentin is deposited as a system of microtubules filled with odontoblastic processes and extracellular fluid. The *crown*, or the visible portion of tooth, consists of a thick *enamel* layer overlying the dentin. Enamel, the hardest substance in the human body, consists largely of hydroxyapatite and is produced by ameloblasts before eruption of the tooth into the mouth. The *root* portion of the tooth extends into the alveolar bone and is covered with a thin layer of *cementum.*

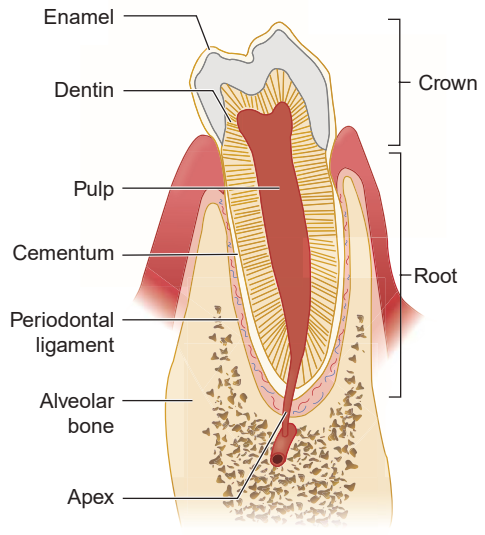


FIGURE 245-3. The dental anatomic unit and attachment apparatus.

THE NORMAL PERIODONTIUM

The periodontium, or attachment apparatus, is essential for maintaining the integrity of the dentoalveolar unit. The attachment apparatus consists of a gingival component and a periodontal component. The gingival component includes the junctional epithelium, gingival tissue, and gingival fibers and primarily functions to maintain the integrity of the periodontal component. The periodontal component includes the periodontal ligament, alveolar bone, and cementum of the root of the tooth and forms the majority of the attachment apparatus. Disease states such as gingivitis and periodontal disease weaken and destroy the attachment apparatus, resulting in tooth mobility and tooth loss.[1]

Gingival tissue is keratinized stratified squamous epithelium. It can be divided into the free gingival margin and the attached gingiva. The free gingiva is the portion that forms the 2- to 3-mm-deep *gingival sulcus* in the disease-free state. The attached gingiva adheres firmly to the underlying alveolar bone. The nonkeratinized alveolar mucosa extends from the attached gingiva to the vestibule and floor of the mouth. The mucosal tissue of the cheeks, lips, and floor of the mouth is also comprised of nonkeratinized squamous epithelium.

OROFACIAL PAIN

Table 245-1 lists common causes of orofacial pain. Pain of dental origin may be diffuse in nature, presenting as a headache, sinus pain, eye pain, or jaw or neck pain, or may be localized to a single tooth. Remember to consider myocardial infarction as a cause of jaw pain.

Examine the soft tissue using a tongue depressor to expand your view looking specifically at the inner lips, the buccal and labial mucosa, the hard and soft palate, and the tongue and floor of the mouth. Ask the patient to extend the tongue, and gently grasp it with a piece of dry gauze, further extending it to the right and to the left, to expose each base. The floor of the mouth should be palpated with a finger in the mouth and one externally under the chin to check for a mass or lesion. Examine the teeth visually, and then gently percuss the suspected teeth with a firm clean object to determine if a specific tooth is the source of pain. After trauma, test for mobility and tenderness with gentle pressure and percussion. Assure that all the teeth occlude as normal for the patient. The degree of opening of the mouth should be evaluated. Palpate for potential fractures of the mandible of maxilla. Finally, the temporomandibular joint should be evaluated by placing your index fingers in the ears and feeling for crepitance or popping while the mandible is fully opened and closed.

PAIN OF ODONTOGENIC ORIGIN

Tooth Eruption and Pericoronitis Discomfort is commonly associated with the eruption of primary or deciduous teeth in infants. Irritability, drooling, and decreased intake are commonly associated findings. Approximately 11% to 12% of teething infants will have a mildly elevated temperature when examined in a doctor's office, but a cause-and-effect relationship between teething and fever has never been demonstrated.[2] Eruption of permanent teeth, especially third molars, or *wisdom teeth,* may cause pain. Gingival irritation and inflammation associated with tooth eruption are common and must be distinguished from *pericoronitis.* Pericoronitis is inflammation of the *operculum*, or the gingival tissue, overlying the occlusal surface of an erupting tooth. Impaction of food and debris beneath the operculum results in a severe inflammatory response. Without intervention, this progressive inflammatory process

TABLE 245-1 Differential Diagnosis of Orofacial Pain

Nontraumatic Causes of Potential Dental Pain	
Odontogenic origin	
Tooth eruption	Cracked tooth syndrome
Pericoronitis	Periradicular periodontitis
Dental caries	Periapical abscess/facial space infection
Dentinal sensitivity/cervical erosion	Postextraction discomfort
Reversible pulpitis	Postextraction alveolar osteitis
Irreversible pulpitis	Post-restorative pain
Periodontal pathology	
Gingivitis	Periodontal abscess
Periodontal disease	Acute necrotizing gingivostomatitis
Gingival abscess	Peri-implantitis
Neurogenic/neurophysiologic syndromes	
Trigeminal neuralgia	Bell's palsy
Other cranial neuralgias	Temporomandibular disorder
Nondental infections	
Oral candidiasis	Hand-foot-and-mouth disease
Herpes simplex types 1 and 2	Sexually transmitted infections
Varicella-zoster, primary and secondary	Herpangina
Mumps	Sinusitis
Sialadenitis	Parotitis
Malignancies	
Squamous cell carcinoma	Leukemia
Kaposi's sarcoma	Melanoma
Lymphoma	Graft-versus-host disease
Other etiologies	
Aphthous ulcers	Pyogenic granuloma
Traumatic ulcers	Lichen planus
Stomatitis and mucositis	Cicatricial pemphigoid
Uremia	Pemphigus vulgaris
Vitamin deficiency	Erythema multiforme
Radiation/chemotherapy related	Crohn's disease
Benign migratory glossitis	Behçet's syndrome
Orofacial Trauma	
Dental fractures	Alveolar ridge fractures
Subtle enamel cracks/infractions	Facial bone fractures
Dental crown and/or root fractures	Oral soft tissue lacerations
Dental luxations and avulsions	

will result in localized infection. Because of the close proximity of the masticator space (comprised of the masseteric space, pterygomandibular space, and the superficial and deep temporalis space) to third molars, this infection can cause trismus. If the infection spreads into the connecting parapharyngeal spaces, it can be life threatening. Treatment of mild to moderate pericoronitis without associated systemic symptoms consists of appropriate antibiotic therapy such as penicillin VK, 500 milligrams PO four times a day, or clindamycin, 300 milligrams PO four times a day; local irrigation of food and debris from underneath the operculum; saline mouth rinses; and analgesia with nonsteroidal anti-inflammatory drugs (NSAIDs) and opiates as appropriate. More severe cases may require IV antibiotics and admission. If pericoronitis is related to trauma from an opposing tooth during mastication, as is frequently the case with third molars, antibiotics and extraction of the opposing tooth will bring marked relief within 24 hours. For outpatient management, referral to a general dentist or an oral and maxillofacial surgeon within 24 to 48 hours is appropriate.[3]

Dental Caries and Pulpitis *Dental caries* is the loss of integrity of the tooth enamel from hydroxyapatite dissolution by prolonged exposure to the acidic metabolic by-products of plaque bacteria. Caries most commonly occurs in areas where plaque accumulates such as pits and fissures of the occlusal surface, interproximally, and along the gingival margins. When a sufficient breach of enamel integrity occurs and the dentin is involved, caries spreads along dentinal microtubules. Direct communication between the oral environment and the vital dental pulp is established, and sensitivity to cold or sweet stimulus may result.

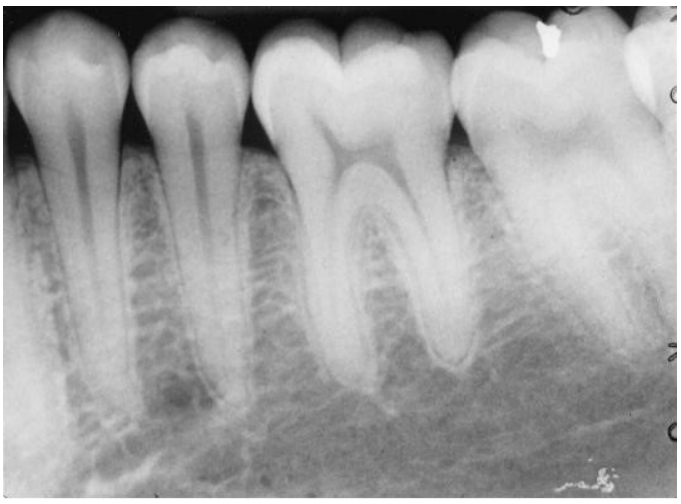
The pulpal inflammatory process is initially reversible, but with continued stimuli, the pulp's ability to respond and repair is compromised. *Irreversible pulpitis* can be distinguished from *reversible pulpitis* by the duration of symptoms. In reversible pulpitis, the duration of pain is short, lasting seconds, as compared with irreversible pulpitis, in which the pain may last for minutes to hours. The most common stimulus is heat or cold, although sweet or sour stimuli also can elicit pain. Spontaneous tooth pain usually represents *pulpal necrosis* and is treated with analgesia; penicillin VK, 500 milligrams PO four times a day, or clindamycin in penicillin-allergic patients; and referral to a general dentist. Antibiotics for dental pain are controversial; two systematic reviews have concluded there is there is insufficient evidence to determine whether antibiotics for irreversible pulpitis reduce pain[4,5] if there is no obvious infection. The use of local anesthetics as discussed in this chapter's section on dental local anesthesia techniques can greatly reduce symptoms and should be considered for short-term pain management. The definitive treatment for irreversible pulpitis and pulpal necrosis is root canal therapy or dental extraction.

Cracked Tooth Syndrome *Cracked tooth syndrome* is an incomplete fracture of a tooth that may extend into the vital pulp. Molars are most commonly affected. The patient experiences sharp pain on chewing that resolves when chewing ceases. Cold and sweet stimuli may also evoke pain. NSAIDs are frequently effective at temporarily controlling pain.[3] The patient should avoid chewing on the affected side and see a dentist for definitive treatment.

Periradicular Periodontitis Acute *periradicular periodontitis* is the extension of pulp disease, inflammation, or necrosis into the tissues surrounding the root and apex of the tooth (deepest portion of the tooth socket). Occasionally it can be due to occlusal trauma. Periradicular lesions appear as a slight widening of the periodontal ligament space, a thinning of the lamina dura, or a radiolucent area associated with the root apex on a periapical dental radiograph. A Panorex is rarely useful for identification of all but the most extensive periradicular lesions, but can be important in identifying other painful osseous pathology (**Figure 245-4**).

Pain on percussion of the suspected tooth with a light metal instrument, such as a handle of a dental mirror, helps to identify the offending tooth. Radiographically and clinically indistinguishable from periradicular periodontitis, a *periapical abscess* by definition contains a collection of pus. A small swelling of the gingiva with a draining fistula adjacent to the affected tooth is known as a *parulis*, and can help identify the involved tooth (**Figure 245-5**). If a dental abscess erodes through the cortical bone but does not drain spontaneously, then subperiosteal extension results in intraoral or facial swelling and fluctuance that should be incised and drained. Treat dental abscesses or other periapical lesions with penicillin VK, 500 milligrams PO four times a day, or clindamycin, 300 milligrams PO four times a day, and analgesia with an NSAID or opiate. Refer to a dentist for definitive treatment.

Facial Space Infections Spread of odontogenic infections into the various facial spaces is relatively common. Buccal extension of a periapical infection of the mandibular teeth will involve the buccinator space. Maxillary labial extension of infection primarily will involve the infraorbital space. Perforation through the lingual cortical bone of mandibular molars, particularly the second and third molars, usually occurs below the mylohyoid ridge and involves the submandibular space. Lingual spread of periapical infections associated with mandibular anterior teeth will affect the lingual space. The submandibular space and lingual space communicate with each other at the posterior border of the mylohyoid muscle. Cellulitis of bilateral submandibular spaces and the lingual space is called **Ludwig's angina** and is potentially life threatening. As



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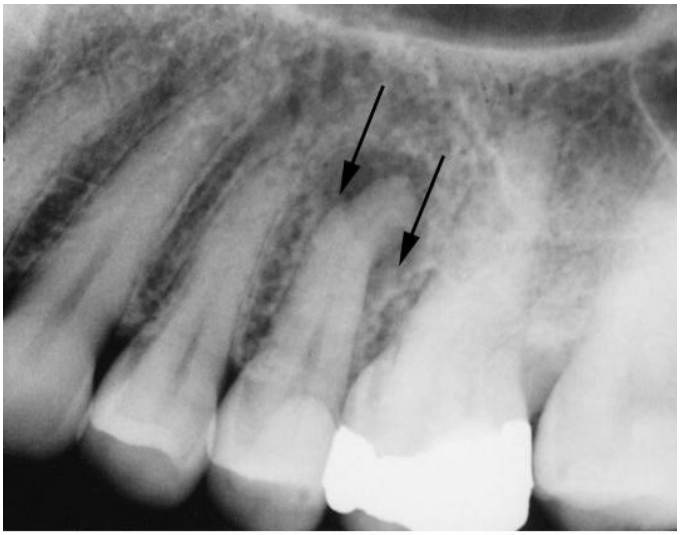


B

FIGURE 245-4. The radiographic appearance of a healthy tooth with a normal periodontal ligament space and distinct lamina dura (**A**) compared with the radiographic appearance of periapical radiolucency (*arrows*) consistent with periradicular periodontitis, a periapical abscess, or periradicular cyst (**B**). [Image used with permission of Gary M. Beaudreau.]

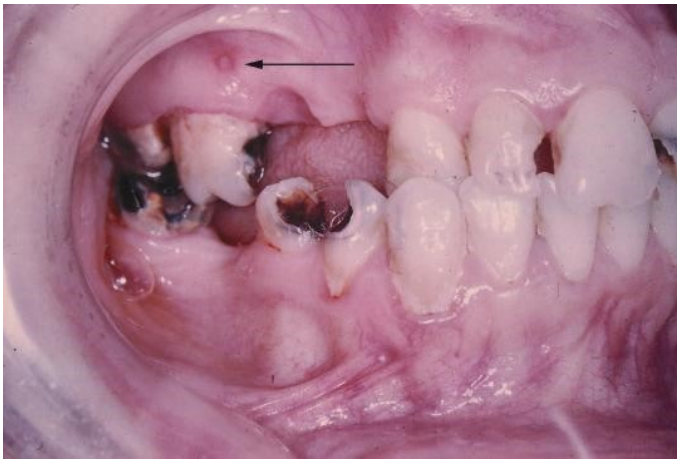


FIGURE 245-5. A parulis (*arrow*) superior to the maxillary molar. [Image used with permission of David E. Beaudreau.]

these spaces and the masticator space communicate directly with the parapharyngeal space, airway compromise is the immediate concern. For detailed discussion, see chapters 243, "Face and Jaw Emergencies" and 246, "Neck and Upper Airway."

Infection of the infraorbital space may have a potentially devastating outcome if retrograde spread through the ophthalmic veins occurs and the cavernous sinus becomes involved. **Cavernous sinus thrombosis** presents as an infraorbital or periorbital cellulitis with rapidly developing meningeal signs, sepsis, and coma. Early recognition and treatment with a high-dose IV antibiotic, as above, are essential in decreasing morbidity and mortality.

Postextraction Pain Immediate postoperative pain is most commonly related to the trauma of surgery. Postoperative edema, such as with extraction of third molars, peaks within the first 24 to 48 hours and is best managed with ice packs, elevation of the head of the bed to 30 degrees, NSAIDs, and oral narcotics. Trismus, common immediately after extraction, can result from direct injury to the temporomandibular joint, injury to the muscles of mastication during administration of the inferior alveolar nerve block or during the surgery, and, most commonly, normal perioperative inflammation. Trismus peaks in the first 24 hours and usually decreases thereafter unless infection develops. Progressively worsening trismus is concerning for a postoperative infection.

Postextraction Alveolar Osteitis (Dry Socket) *Postextraction alveolar osteitis*, or **dry socket**, usually occurs on the second or third postoperative day and is associated with exquisite oral pain. Total or partial displacement of the clot from the socket or fibrinolytic dissolution of the clot results in exposure of the alveolar bone and initiates a localized osteomyelitis of the exposed bone. Risk factors for developing postextraction alveolar osteitis include smoking, preexisting pericoronitis or periodontal disease, a traumatic extraction, a prior history of alveolar osteitis, and hormone replacement therapy.[6] The incidence of postextraction alveolar osteitis is 1% to 5% of all extractions but is considerably higher (up to 30%) among impacted third molar extractions.[6,7]

Treatment is gentle irrigation of the socket with warmed normal saline or chlorhexidine 0.12% oral rinse.[6-9] Local dental anesthesia (see the section on dental local anesthesia techniques) or topical anesthesia may be needed. Management of pain with NSAIDs or opiate medication is necessary. Antibiotic therapy with penicillin VK, 500 milligrams PO four times a day, or clindamycin, 300 milligrams PO four times a day, is reserved for the most severe cases. Refer for dental follow-up.[6-9]

Postextraction Bleeding Postextraction bleeding is not uncommon. Displacement of the clot may result in recurrent or continued bleeding. Generally, firm pressure applied to the extraction site is adequate to control bleeding. This is best accomplished by folding a 2 × 2-inch gauze pad and placing it over the extraction site and applying firm pressure by clenching with the opposing teeth. Pressure must be held firmly, not a chewing action, for 20 minutes or until hemostasis is complete. If direct pressure is not successful, then apply an absorbable gelatin sponge (Gelfoam®, Pfizer Inc., New York, NY), microfibrillar collagen (Avitene®, Davol, Inc., Warwick, RI), or regenerated cellulose (Surgicel®, Ethicon, Inc., Somerville, NJ) into the socket to provide a matrix for clot formation. Sutures can be used for holding such agents in place or to loosely close the gingiva over the socket. **Do not suture the gingiva tightly because this may cause necrosis of the gingival flap.** If this is not successful, careful injection of the soft tissue surrounding the extraction with lidocaine with epinephrine may control the bleeding. Careful cautery with silver nitrate can also be useful. If these methods are unsuccessful, then oral and maxillofacial surgical consultation is necessary.

Postrestorative Pain Postrestorative pain can result from normal trauma from mechanical instrumentation of the tooth or direct exposure of the pulpal tissue during instrumentation. Pain associated primarily with mastication may be the result of improper occlusion of the new dental restoration or filling. After endodontic therapy, buildup of pressure in the pulpal chamber can cause severe pain. Provide NSAIDs or narcotic analgesia and refer to the patient's dentist. Temporary prolonged pain relief can also be obtained using 0.5% bupivacaine with epinephrine and the appropriate dental anesthetic block as discussed in the dental local anesthesia technique section of this chapter. Follow-up with the dentist the next day then should be possible.

Orthodontic Appliances The most common emergency is a broken or bent wire that is irritating or lacerating the cheek or lip. This wire needs to be bent back away from soft tissue. This can easily be accomplished with dental instruments, or something soft like a pencil eraser can be used to gently bend the wire. Cutting the wire is generally not indicated as it makes the end sharper. The broken portion of the wire can be removed in its entirety by removing the rubber ligatures from each orthodontic bracket, but this generally is not necessary. The patient should follow up as soon as possible with the orthodontist.

PERIODONTAL PATHOLOGY

Periodontal Disease Periodontal disease is a continuum of disease that begins with gingival inflammation and bleeding, or gingivitis, and can progress to destruction of the periodontal attachment apparatus, deepening of the normal gingival sulcus, periodontal pocket formation, bone loss, tooth mobility, and ultimately loss of teeth.[1] Besides oral hygiene, many factors including hormonal variations, medications, and systemic disease can also influence periodontal health.

Periodontal disease usually progresses painlessly but may present as swollen gingival tissue or gingival bleeding. Treatment is directed at slowing or arresting the progression of disease primarily by the removal of plaque and its by-products.[1] Antibiotics may play a role in treatment. Referral to a dentist for definitive treatment is indicated because the treatment involves extensive dental cleaning, instruction and improvement in oral hygiene, and in some cases, periodontal surgery.

Gingival and Periodontal Abscess A *gingival abscess* is an acutely painful swelling confined to the margin of the gingiva or interdental papilla. It usually rapidly enlarges over 24 to 48 hours, and purulent exudate can frequently be expressed from the orifice. The most common etiology is the entrapment of foreign matter such as a popcorn kernel, piece of meat, toothbrush bristle, or piece of food in the gingiva. Treatment includes identifying and removing the embedded foreign body and irrigating with normal saline. Continued home irrigation is beneficial, and symptoms resolve quickly.[3]

When plaque and debris are entrapped in the periodontal pocket, a *periodontal abscess* may form, resulting in severe pain. Small periodontal abscesses respond to local therapy with warm saline rinses and antibiotics such as penicillin VK, 500 milligrams PO four times a day, or clindamycin, 300 milligrams PO four times a day. Larger periodontal abscesses require incision and drainage. Chlorhexidine 0.12% mouth rinses twice daily are useful in the short term. Provide analgesia with NSAIDs or narcotics as indicated.[3]

Acute Necrotizing Ulcerative Gingivitis *Acute necrotizing ulcerative gingivitis* is an aggressively destructive process (**Figure 245-6**). Also known as *Vincent's disease* or *trench mouth*, it is part of a spectrum of disease ranging from localized ulceration of the gingiva to often fatal noma, in which localized ulceration and necrosis spread to the adjacent tissues of the cheeks, lips, and underlying facial bones.[10] The diagnostic triad includes pain, ulcerated or "punched out" interdental papillae, and gingival bleeding. Secondary signs include fetid breath, pseudomembrane formation, "wooden teeth" feeling, foul metallic taste, tooth mobility, lymphadenopathy, fever, and malaise.[3,10]

The differential diagnosis for acute necrotizing ulcerative gingivitis is quite extensive, but herpes gingivostomatitis is the most difficult to differentiate. Herpes gingivostomatitis usually has smaller vesicular eruptions, less bleeding, more systemic signs, and lack of interdental papilla involvement.[10]

The cause is still poorly understood. Acute necrotizing ulcerative gingivitis appears to be an opportunistic infection in a host with lowered resistance. Anaerobic bacteria such as *Treponema*, *Selenomonas*, *Fusobacterium*, and *Prevotella* invade otherwise healthy tissue, resulting in an aggressively destructive disease process.[10,11] The most important predisposing factor is human immunodeficiency virus infection. A previous episode of necrotizing gingivitis is the second most important predisposing factor. Other contributing factors include poor oral hygiene, unusual emotional stress, poor diet and malnutrition, inadequate sleep, Caucasian descent, age <21 years old, poor socioeconomic status, recent illness, alcohol use, tobacco use, acatalasia, and various infections such as malaria, measles, and intestinal parasites.[10,11]

Treatment consists primarily of bacterial control. Chlorhexidine 0.12% oral rinses twice a day, professional debridement and scaling, and adjunctive antibiotic therapy with metronidazole, 500 milligrams PO three times a day, are the mainstay of treatment. Reduction in pain can be expected within 24 hours of institution of this regimen. Identification and resolution of the predisposing factors and supportive therapy with a soft diet rich in protein, vitamins, and fluids are important in establishing and maintaining a disease-free state.[10,11]

Peri-Implantitis Osseointegrated dental implants have become common over the last 30 years, allowing for a dental implant to replace a tooth. However, as with any procedure, complications do occur, and problems related to implants may present to the ED. Pathologic changes around an implant are all given the general term of *peri-implant disease.* Patients who present with *peri-implantitis* present with a similar presentation to that of a periodontal abscess and require similar treatment. Gentle removal of the plaque and debris from around the implant and irrigation with normal saline or 0.12% chlorhexidine solution should be done. Antibiotic treatment with metronidazole, 500 milligrams PO three times a day for 10 days, or amoxicillin, 500 milligrams PO three times a day for 10 days, is indicated. Give analgesia as needed, and refer to a dentist for definitive care.[3]

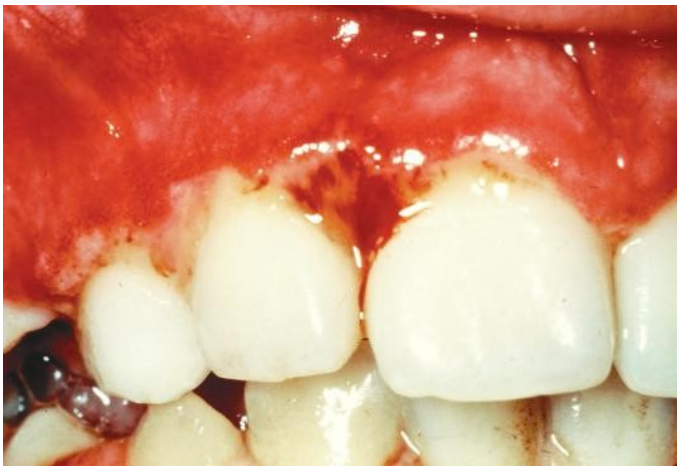


FIGURE 245-6. Acute necrotizing ulcerative gingivitis. [Image used with permission of Philip J. Hanes.]

NEUROGENIC AND NEUROPHYSIOLOGIC SYNDROMES

Craniofacial Neuralgias *Trigeminal neuralgia* is the most common of the craniofacial neuralgias. Other significantly less common neuralgias of the craniofacial region include *glossopharyngeal neuralgia*, *vagal neuralgia*, and *superior laryngeal neuralgia* involving the respective nerve distributions. Post–herpes zoster–related neuralgia is also a cause of acute facial pain and may become chronic in nature. See chapter 165, "Headache" for further discussion.

Bell's Palsy (Idiopathic Facial Nerve Palsy) *Bell's palsy* is a peripheral unilateral weakness of the facial nerve of unknown etiology. As part of the differential diagnosis for orofacial pain, patients with a facial nerve palsy related to herpes zoster may present with nonspecific facial pain prior to the onset of weakness or the onset of any visible vesicles. In such cases, this diagnosis must be considered. See chapter 243 for a more extensive discussion on facial nerve palsy.

Temporomandibular Disorder *Temporomandibular disorder* is a common cause of facial pain and headache representing a group of signs and symptoms that involve the muscles of mastication or the temporomandibular joint. See chapter 243 for a more extensive discussion.

SOFT TISSUE LESIONS OF THE ORAL CAVITY

APHTHOUS STOMATITIS

Aphthous stomatitis, or ulceration, is one of the most common oral lesions, affecting 20% of the normal population (**Figure 245-7**). The cause appears to be a cell-mediated immune response to a yet unidentified triggering agent. Multiple factors predispose to aphthous ulcer formation: local trauma, stress, poor sleep, a hormonal imbalance, smoking, and certain foods such as chocolate, coffee, peanuts, cereals, almonds, strawberries, cheese, tomatoes, and gluten. Aphthous ulceration involves the nonkeratinized epithelium, especially the labial and buccal mucosa, and begins as an erythematous macule that ulcerates and forms a central fibropurulent eschar. Aphthous stomatitis occurs in a major and minor form. *Minor aphthae* usually measure from 2 to 3 mm to several centimeters in diameter, are painful, and frequently are multiple. They usually resolve spontaneously in 10 to 14 days. *Major aphthae* have larger, deeper ulcers that take significantly longer to heal. A third form, called *herpetiform aphthae*, has up to 100 ulcers, each 1 to 2 mm in diameter. They tend to coalesce, creating much larger ulcers that require 10 to 14 days to heal. Treatment is symptomatic but, in severe cases, may consist of topical corticosteroids such as Orobase® (Colgate Oral Pharmaceuticals, Canton, MA) or 0.01% dexamethasone elixir as a mouth rinse. Resolution typically occurs quickly after onset of therapy. Major aphthae are more resistant to therapy and may require intralesional steroid injection or systemic steroid therapy.[12,13]

HERPES ZOSTER AND OTHER INFECTIONS

Herpes zoster (see chapter 153, "Serious Viral Infections") frequently occurs along the distribution of the trigeminal nerve. Herpes zoster typically begins as a 1- to 4-day prodrome of exquisite pain in the area innervated by the affected nerve and may be mistaken for a simple headache or toothache. Vesicular eruptions characteristically occur unilaterally, do not cross the midline, and last 7 to 10 days. Isolated intraoral lesions can occur but are not common. Involvement of the ophthalmic branch of the trigeminal nerve requires urgent ophthalmologic consultation.

Other common infections such herpes simplex type 1 and 2, herpangina, hand-foot-and-mouth disease, and varicella-zoster cause painful ulcerative lesions of the oral cavity and perioral region. These conditions are adequately discussed in chapter 121, "Mouth and Throat Disorders in Infants and Children." Many sexually transmitted infections can affect the oral cavity. In general, the appearance of oral lesions is similar in appearance to that of their genital counterpart. Treatment of sexually transmitted diseases of the oral cavity is the same as for genital involvement. See chapter 149, "Sexually Transmitted Infections," for detailed discussion.

TRAUMATIC ULCERS

Traumatic ulcers are a result of direct trauma to epithelial tissue. Common sources of trauma include rough or jagged edges on teeth or restorations, ill-fitting dentures, oral hygiene mishaps, and burns to the hard or soft palate secondary to hot foods. Removal of persistent sources of trauma is essential; otherwise, treatment is palliative.

MEDICATION-RELATED SOFT TISSUE ABNORMALITIES

Gingival hyperplasia is associated with many commonly used medications (**Figure 245-8**). Approximately 50% of patients on phenytoin will develop significant gingival hyperplasia. Many other medications, such as cyclosporine and calcium channel blockers, especially nifedipine, cause gingival hyperplasia. Concomitant use of two such medications results in accelerated gingival proliferation. Enlargement begins in the interdental papillae. The clinical and histologic characteristics of gingival hyperplasia related to phenytoin, cyclosporine, and calcium channel blockers appear to be identical. The clinical appearance of the gingival tissue depends on oral hygiene and secondary inflammation. In the absence of inflammation, gingival proliferation results in dense tissue, normal in coloration, with a smooth, stippled, or granular texture. Inflammation causes edematous changes and an erythematous coloration. Inflamed tissue bleeds readily. Histologically, an increase in collagen fibers, fibroblasts, and glycosaminoglycans is seen. Epithelial acanthosis also occurs. Although the cause of drug-related gingival hyperplasia is unclear, poor oral hygiene clearly increases its likelihood and severity. Treatment includes fastidious oral hygiene to slow the hyperplasia and gingivectomy in advanced cases.

Many other medications are known to cause abnormalities of the oral mucosa or dental structures. Allergic mucositis, erythema multiforme, and fixed drug-type reactions are examples. Xerostomia and associated mucosal alterations are a side effect of many medications such as anticholinergics, antidepressants, and antihistamines.[1] Stomatitis and mucosal ulcerations from chemotherapeutic agents are also common.

LESIONS OF THE TONGUE

Many systemic conditions and local stimuli affect the appearance of the tongue. Many systemic conditions, various vitamins deficiencies, and iron-deficiency anemia cause atrophy of the filiform papillae, resulting in a smooth erythematous appearance. Occurrence of ectopic thyroid tissue on the midline posterior portion of the tongue is called a *lingual thyroid* and is a common finding. Some common conditions affecting the tongue are discussed below.

Benign Migratory Glossitis Geographic tongue, or benign migratory glossitis, is a common benign finding on oral examination, occurring in

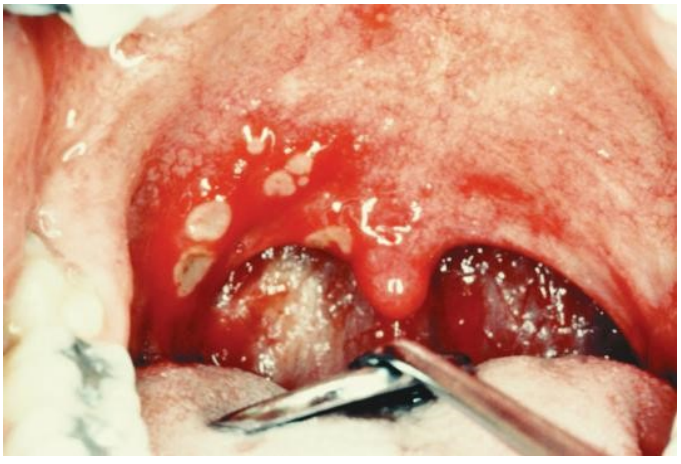


FIGURE 245-7. Aphthous stomatitis. [Image used with permission of Baldev Singh.]

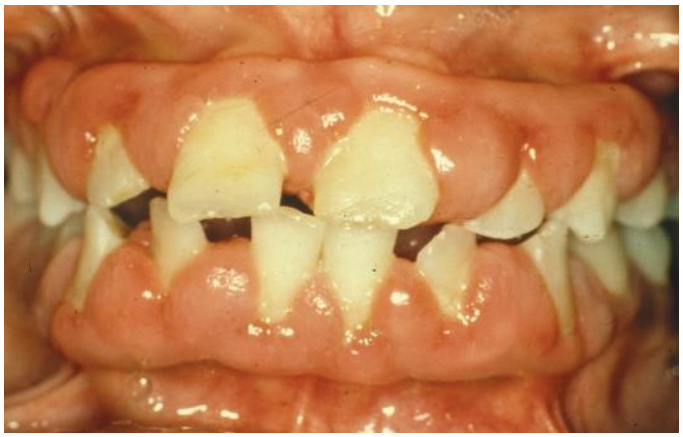


FIGURE 245-8. Gingival hyperplasia (overgrowth) secondary to phenytoin (Dilantin). [Used with permission of Philip J. Hanes.]

1% to 3% of the population. Females are affected twice as often as males. The typically multiple, well-demarcated zones of erythema on the tongue are caused by atrophy of the filiform papillae. The lesions concentrate on the tip and lateral borders of the tongue and heal in several days, only to quickly reappear in other areas. These lesions usually are asymptomatic; however, a burning sensation or sensitivity to hot or spicy foods has been described. The cause is unknown, but fluctuations with stress and menstrual cycle occur. Generally, treatment is not indicated because this entity is benign. Reassurance of patients is usually sufficient. In patients in whom discomfort is a major factor, oral topical steroids such as fluocinonide gel applied several times daily may provide relief.[14]

Strawberry Tongue Strawberry tongue is associated with erythrogenic, toxin-producing *Streptococcus pyogenes*. Clinically, the tongue has prominent red spots on a white-coated background. Microscopically, the fungiform papillae are hyperemic with a smooth glossy surface. Treatment is with antibiotics directed at group A streptococci.

LEUKOPLAKIA AND ERYTHROPLAKIA

Leukoplakia is a white patch or plaque that cannot be scraped off and cannot be classified as any other disease. Leukoplakia is the most common oral precancer; however, only 2% to 4% of leukoplakic lesions show dysplastic changes. The cause is unknown, but tobacco, alcohol, ultraviolet radiation, candidiasis, human papillomavirus, tertiary syphilis, and trauma have all been implicated. The most common intraoral site involved is the buccal mucosa. Other sites of involvement include the hard and soft palates, maxillary gingiva, and lip mucosa. Biopsy is mandatory for all persistent leukoplakic lesions. Leukoplakic lesions of the floor of the mouth, tongue, and vermilion border are most likely associated with malignancy. Lesions demonstrating dysplastic changes warrant removal.[15]

Erythroplakia is defined as a red patch that similarly cannot be clinically or pathologically characterized as any other disease. Although erythroplakia is far less common than leukoplakia, it has a greater potential for dysplastic changes.

ORAL CANCER

Oral cancer accounts for 2% to 4% of the cancers in the United States. More than 90% of all oral malignancies are squamous cell carcinoma (**Figure 245-9**).[15] Lymphomas, Kaposi's sarcoma, and melanoma comprise most of the remainder. Several intrinsic and extrinsic etiologic factors for oral squamous cell carcinoma have been identified. Extrinsic factors include tobacco use, especially chewing tobacco or snuff; excessive alcohol consumption; and sunlight exposure. Intrinsic factors include general malnutrition and chronic iron-deficiency anemia. Oral candidiasis, especially the hyperplastic form, immunosuppressive states such as human immunodeficiency virus infection, and oncogenic viruses such as human papillomavirus, herpes simplex virus, and various adenoviruses and retroviruses may play some role in the etiology of oral cancer.

Oral squamous cell carcinoma has four common morphologic presentations. It can be exophytic, with an irregular surface, or ulcerative, with irregular depressions and rolled borders. Malignant leukoplakic and erythroplakic lesions are believed to represent squamous cell carcinomas that have yet to form a mass or ulcerate.

The most common site involved in oral cancer is the tongue, particularly the posterolateral border, accounting for 50% of the oral cancers in the United States.[15] Cancer of the floor of the mouth accounts for nearly 35%. Cancer of the lips is common and usually secondary to sunlight exposure. Oral cancer is generally painless, and patients are often unaware of the presence of a mass until it is advanced. Oral cancer is usually firm, may bleed from ulceration, and have a history of poor healing. There may be associated firm lymphadenopathy. Early diagnosis is the key to successful treatment of oral squamous cell carcinoma. All ulcers, erythroplakic lesions, and leukoplakic lesions of the oral cavity that do not respond to palliative treatment in 10 to 14 days warrant biopsy. Treatment depends on site of involvement and staging of disease.

DENTOALVEOLAR TRAUMA

Management of dentoalveolar trauma depends on the extent of tooth and alveolar involvement, the degree of development of the apex of the tooth, and the age of the patient. In injuries in younger patients, especially those who are <12 years of age, the pulp of anterior teeth is quite large, and dental fractures involving the pulp are common. Fortunately, in this age group, the apex of the root also is usually incompletely formed, allowing for a greater pulpal regenerative capability. As one ages, more dentin is formed. Thus, in older patients, the pulp chamber may be very small and pulpal exposure highly unlikely. Involvement of the root of the tooth compromises the attachment apparatus and makes it difficult to restore the tooth to function.

DENTAL FRACTURES

The International Association of Dental Traumatology system divides dental trauma into eight categories: enamel infraction, enamel fracture, enamel-dentin fracture, enamel-dentin-pulp fracture, crown-root fracture without pulp exposure, crown-root fracture with pulp exposure, root fracture, and alveolar bone fracture (**Figure 245-10**). The International Association of Dental Traumatology has developed guidelines to aid dentists and other healthcare professionals in the management of each these categories.[16]

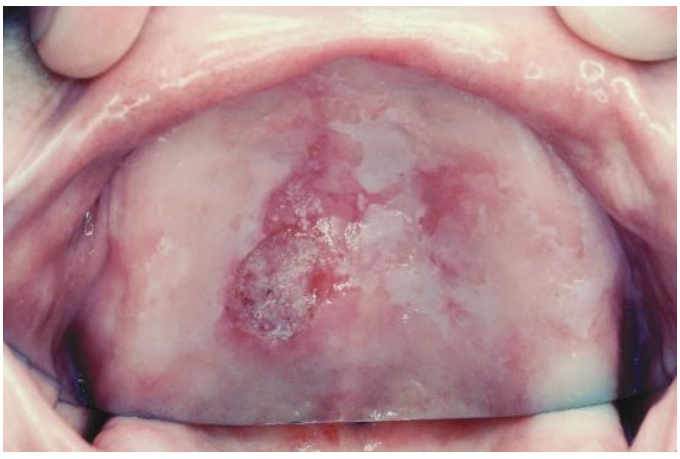


FIGURE 245-9. Oral squamous cell carcinoma of the hard palate. [Image used with permission of H. Anthony Neal.]

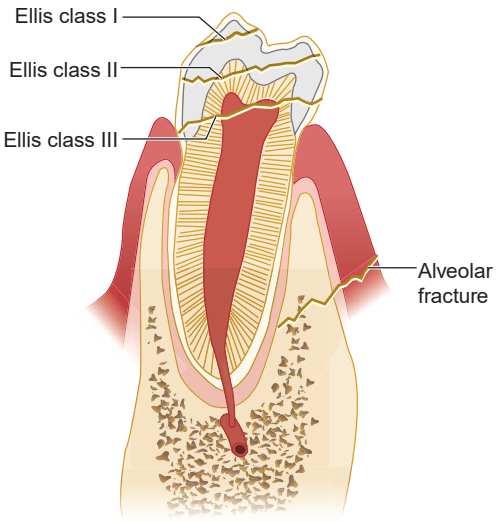
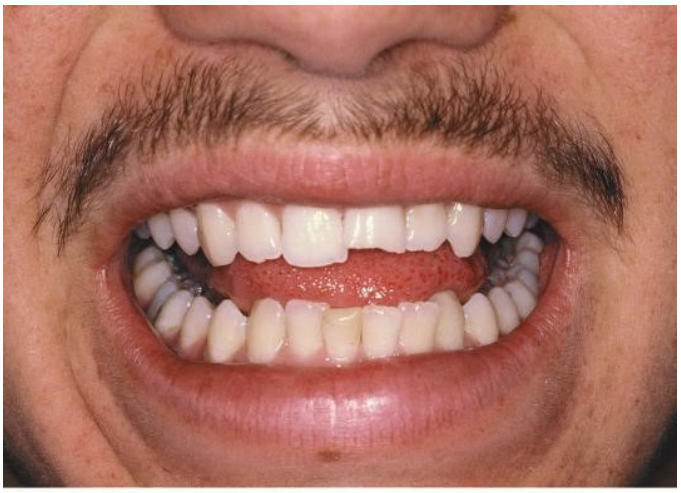


FIGURE 245-10. The International Association of Dental Traumatology classification of dental fractures.

The goal of the emergency treatment of a fractured tooth is maintaining pulpal vitality and completion of the formation of the root and apex of the tooth. The proximity of the fracture to the pulp and the length of time before treatment, as well as other associated injuries, are most important in determining outcome. Treatment is aimed at sealing the dentinal tubules and creating a barrier between the dental pulp and the oral environment. In properly treated uncomplicated dental fractures, 1% to 3% of the affected pulps undergo necrosis. Because pulpal necrosis is a process, it can occur at any time after trauma, and serial follow-up with a dentist is recommended.[16]

A fracture of the crown of the tooth can involve any part of the enamel, dentin, and pulp of the tooth. *Infraction* is the least serious type dental injury and essentially is a crack in the enamel of the tooth without loss of structure. This can be obvious or quite subtle. No emergency treatment is needed.

Enamel fractures do not extend into the dentin of the tooth (**Figure 245-11**). Generally, no emergency treatment is indicated, except to smooth sharp corners that may irritate the tongue or mucosa. If the enamel fragment is recovered and kept moist, the patient's dentist can bond it back in place. If the fragment was not recovered, soft tissue radiographs of any oral lacerations are necessary to rule out foreign bodies. Referral to a general dentist for aesthetic repair depends on the degree of cosmetic concern of the patient.[16]



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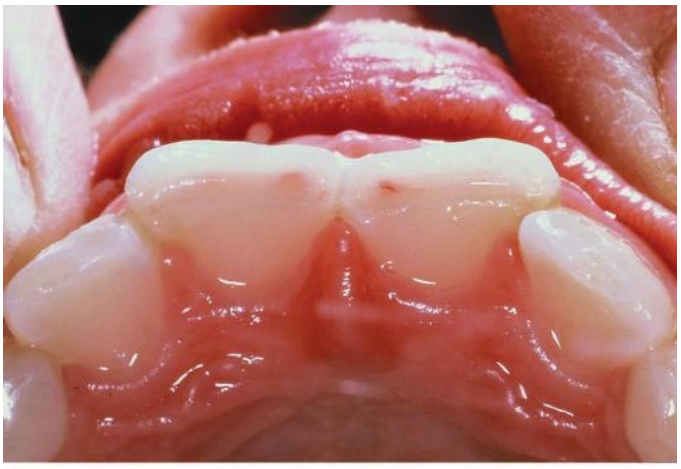

B

FIGURE 245-11. International Association of Dental Traumatology classification for fractures of teeth (clinical). **A.** Enamel fracture. **B.** Enamel-dentin fracture of the central incisor on the left in the photograph (notice the pink blush of the pulp through the thin layer of dentin), and an enamel-dentin-pulp fracture of the central incisor on the right in the photograph. [Image used with permission of Felicity K. Hardwick.]

Enamel-dentin fractures involve the dentin of the tooth and require intervention (Figure 245-11). These fractures account for 70% of tooth fractures. Generally, patients experience sensitivity to hot or cold stimuli as well as air passing over the exposed surface during breathing. The enamel-dentin fracture can be identified both by the patient's symptoms and visualization of exposed dentin, which is a creamy yellow color compared with the whiter enamel. Because dentin is microtubular in structure, communication with the oral environment or desiccation from mouth breathing initiates an inflammatory response in the dental pulp. The thickness of remaining dentin determines the rate of pulpal contamination. Greater than 2 mm of remaining dentin is felt to offer some protection to the pulpal tissue. A delay in treatment of more than 24 to 48 hours increases the likelihood of pulpal necrosis. The ED goal is the identification of a fracture. If definitive treatment cannot be assured in 1 to 2 days, then cover the exposed dentin to decrease likelihood of pulpal injury. This is best achieved using glass ionomer dental cement that is easily mixed according to the manufacturer's instructions and carefully applied to the dried exposed dentin (DenTemp□, Majestic Drug Co., South Fallsburg, NY; and others). If the dentin layer is less than 0.5 mm and the pulp can be seen as a pink area without bleeding, then first place a thin layer of calcium hydroxide base (Dycal□, Dentsply International, York, PA) followed by glass ionomer, as described above, to further protect the dental pulp. Referral to a dentist for definitive treatment is important.[16]

In *enamel-dentin-pulp* fractures, exposure of the pulp has occurred (Figure 245-11). On wiping the fractured surface dry with sterile gauze, blood originating from the pulp of the tooth is easily identified. After carefully controlling pulpal bleeding with sterile gauze or a cotton pellet, cover the exposed pulp with a calcium hydroxide base (Dycal□, Dentsply International, York, PA), and then cover this and the remaining exposed dentin with glass ionomer cement as in enamel-dentin fractures until urgent dental evaluation can occur. If the pulpal exposure is extremely small, placing a calcium hydroxide base and glass ionomer is adequate until dental evaluation. For all but the smallest pulpal exposures, definitive treatment is some kind of endodontic or root canal therapy. Oral analgesics should be prescribed and topical analgesics avoided.[16]

Crown-root fractures and *root fractures* are an uncommon consequence of dental trauma. The coronal segment of the tooth may be displaced or simply mobile. Tenderness to percussion is usual. With any dental trauma, careful attention must be paid to identifying fractures of the root, as they can be clinically obscure and dental radiographs from several angles may be necessary to identify these fractures. This, however, is beyond the scope of most EDs. Crown-root fractures may or may not involve the pulp. Emergency treatment consists of stabilizing the coronal segment until definitive treatment can be arranged. In isolated root fracture, the pulp is always involved. Healing of stabilized root fractures has been reported; thus, current recommendations are to reposition the coronal segment to its original position, confirm that position by radiograph, if available, and then stabilize with a flexible splint as described below for luxation injuries. Dental follow-up within 24 to 48 is important, as splinting for a minimum of 4 weeks is required. In the ED, where oral surgical or dental consultation may not be readily available, extraction of an extremely mobile coronal segment of the tooth may be required to prevent possible aspiration. If less than one third of the root is involved, a dentist can perform root canal therapy, and restoration of the tooth may be possible.[16,17]

LUXATION INJURIES

The same forces that cause dental fractures may result in loosening of a tooth from the attachment apparatus. Careful evaluation of the teeth for tenderness, malpositioning, or mobility must be performed. Luxations account for nearly 50% of injuries to teeth. There are six types of luxations: concussion, subluxation, extrusive luxation, lateral luxation, intrusive luxation, and avulsion.[16]

Concussion is injury to the supporting structures of a tooth with clinical tenderness to percussion but no mobility. *Subluxation* is injury resulting in mobility without clinical or radiographic evidence of dislodgment of the tooth. *Extrusive luxation* is partial or total disruption

of the periodontal ligament resulting in a partial dislodgment of a tooth from the alveolar bone. *Lateral luxation* is displacement of a tooth labially (toward the lip) or lingually (toward the tongue) with concomitant fracture of the alveolar bone. *Intrusive luxation* is displacement of a tooth into its socket with associated periodontal ligament damage and alveolar bone contusion and fracture. Treatment of luxations depends on the tooth involved, the severity of injury, and the presence of associated root fracture and/or significant associated alveolar fracture.[16]

Concussions A concussive injury to a tooth is minor. The degree of tenderness to percussion determines the treatment. Stabilizing the tooth by splinting it to adjacent teeth is not indicated. Management of pain with NSAIDs, soft diet, and referral to a dentist to confirm the diagnosis and exclude more severe injury is the most appropriate course of action for the emergency physician.[16]

Luxations *Subluxation* represents a more significant injury and is associated with a higher incidence of subsequent pulpal necrosis. Clinically, tooth mobility and some bleeding along the gingiva may be noted. A subluxed tooth generally does not require splinting.[16]

An *extrusive luxation* requires repositioning the tooth to its original position and splinting to stabilize the tooth during healing. Ideally, dental radiographs should be obtained prior to repositioning the tooth to ensure that there is not a fracture of the root of the tooth, but in most EDs, this may not be possible, and repositioning and stabilizing should be attempted regardless. Repositioning the tooth may require local anesthesia. Firm, gentle pressure usually will reposition the tooth. If a clot has formed apical to the tooth, then more aggressive manipulation may be required. A flexible wire splint placed by a dentist provides ideal stabilization.[16] In the ED, a temporary splint with a noneugenol zinc oxide periodontal dressing (Coe-Pak□, GC America Inc., Alsip, IL; or ZONE Periopak□, DUX Dental, Oxnard, CA) (**Figure 245-12**) is acceptable. Avoid excess material placement, especially on the occlusal surface, because interference in occlusion will place stress on the tooth during mastication. Other treatment options include wire splinting, bondable reinforcement ribbon, calcium hydroxide paste, and light cured composite.[18] The patient should see a dentist or oral and maxillofacial surgeon within 24 hours.

A *lateral luxation* represents a more extensive injury and is associated with fracture of the surrounding alveolar bone. Repositioning of the tooth is generally more difficult. It usually can be accomplished by manipulating the displaced tooth with the thumb and forefinger. Once the apex has been dislodged from its locked-in position labially, apically directed axial pressure will reposition the tooth. Intra-arch stabilization is necessary for a minimum of 4 weeks. Temporary splinting with a periodontal dressing is acceptable if a minimal associated alveolar fracture occurred. Otherwise, splinting by an oral and maxillofacial surgeon or general dentist in the ED is mandatory.[16]

Intrusive luxations are the most serious because significant damage to the alveolar socket and periodontal ligament occurs. Root resorption is common as a result of damage to the periodontal ligament. Recommended treatment is allowing the tooth to erupt on its own or to orthodontically extrude the tooth if no eruption is noted by 3 weeks.[16]

All patients who sustain luxation injuries should be instructed to maintain a soft diet for at least 2 weeks. Meticulous oral hygiene is essential. Twice daily rinsing with chlorhexidine 0.12% mouth rinse is helpful. Referral to a dentist for close follow-up is indicated.

Avulsions

Tooth Replantation and Care at the Scene **Total displacement of a tooth from its socket, or *avulsion*, represents up to 16% of all dental injuries and is a true dental emergency.[19]** Replantation at the scene is the treatment of choice because the long-term prognosis for the replanted tooth is highly time dependent. Ideally, the patient or a healthcare provider at the scene should perform this procedure. Handling only the crown portion

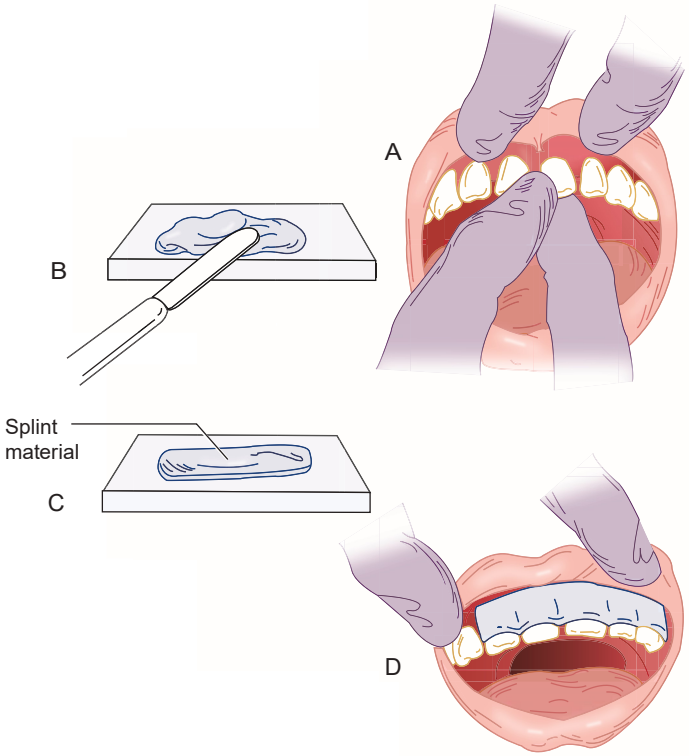

FIGURE 245-12. Temporary stabilization of a replanted or repositioned tooth. **A.** Tooth is repositioned back into its original position in the socket. **B.** Splint material is mixed thoroughly. **C.** Splint material is shaped and made ready for application. **D.** Packing is molded over repositioned tooth and two adjacent teeth to each side.

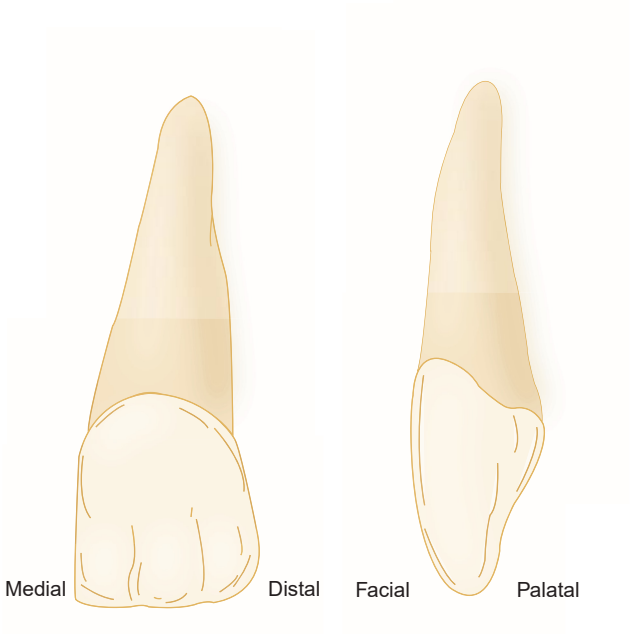

FIGURE 245-13. Illustration of a maxillary left central incisor. Note that the part of the tooth facing medially comes to more of a right angle at the incisal edge (biting edge) than occurs distally. The facial portion of the tooth is more convex.

of the tooth, gently rinse the tooth for a maximum of 10 seconds with sterile normal saline or tap water to remove debris. Then replace it immediately into the socket. Anterior teeth are most commonly affected, and **Figure 245-13** illustrates the morphology of the maxillary central incisor to assist replantation in the proper orientation. Early improper replantation holds a higher success rate for tooth salvage than delayed replantation resulting from waiting for arrival at the ED or for an oral and maxillofacial surgeon.[19]

If immediate replantation is not possible, or if the risk of aspiration is high, such as in a child or a patient with a decreased level of consciousness, then transport the tooth with the patient to the ED. Acceptable transport media include isotonic solutions such as Hank's balanced salt solution, sterile saline, milk, and saliva. Commercial preparations of Hank's balanced salt solution such as Save-A-Tooth® (Phoenix-Lazerus, Inc., Pottstown, PA) and EMT Tooth Saver® (Smart Practice, Phoenix, AZ) are available and come with a useful transport container as part of the system. If the avulsed tooth was not recovered, obtain radiographs to ensure that the tooth was not aspirated.[19]

Survival of the periodontal ligament fibers that remain attached to the root of an avulsed tooth is key to successful replantation. Milk is an acceptable storage medium because of its osmolarity and essential concentration of calcium and magnesium ions. Hank's balanced salt solution, a pH-balanced cell culture medium, is the best transport medium and maintains periodontal ligament cell viability for up to 4 to 6 hours. Hank's solution can help to restore cell viability in a tooth that has been avulsed longer than 20 minutes.[19]

Tooth Replantation in the ED In the ED, before replantation, rinse the tooth root clean of dirt and debris with sterile saline or, preferably, Hank's balanced salt solution. Do not scrub the root of the tooth, and do not disrupt existing periodontal fibers. Handle only the crown of the tooth. If an avulsed tooth with an open apex has been dry for <20 minutes, then the prognosis for reestablishing a vital pulp is good. If the apex is completely closed and cannot be opened with saline irrigation, then revitalization is not possible. If the tooth has been dry from 20 to 60 minutes regardless of apices, soak the tooth in a physiologic solution while preparing to replant the tooth. Physiologic solution decreases the chance of ankylosis (fixation of the tooth to the underlying bone). For an avulsed tooth that has been dry for >60 minutes, the periodontal cells are dead, and the goal is to maintain alveolar bone contour and aesthetics; however, ankylosis, root resorption, and eventual tooth loss are the expected outcome. **Table 245-2** provides specific recommendations.[19]

Preparation of the dental socket plays little role in the success or failure of the replanted tooth. Prepare the socket by carefully removing the clot and gently irrigating with sterile normal saline. Avoid socket manipulation if possible. However, any fracture of the socket wall should be carefully repositioned with an appropriate instrument. Local anesthesia is usually required. Replantation is accomplished with firm pressure.

TABLE 245-2 Specific Recommendations for Replantation of Avulsed Teeth

Clinical Scenario	Treatment
Open apex	1. Gently irrigate the tooth root clean with sterile saline.
Moist tooth stored in acceptable media, and/or <60 min extra oral dry time	2. If available, cover root with minocycline hydrochloride microspheres (Arestin™, OraPharma, Inc.) or soak for 5 min in doxycycline solution (doxycycline, 1 milligram/20 mL saline). 3. Administer local anesthesia. 4. Remove coagulum from the socket with a stream of saline. Examine the socket. If there is a fracture of the socket wall, reposition it with an appropriate instrument. 5. Firmly replant tooth and verify the tooth position clinically and radiographically, if possible. 6. Flexible splint for up to 2 wk.
Open apex	1. Remove from tooth attached necrotic soft tissue carefully with gauze.
Extra oral dry time >60 min or other reason suggesting nonviable cells	2. If available, immerse tooth in 2% stannous fluoride solution for 20 min. 3. Administer local anesthesia. 4. Remove coagulum from the socket with a stream of saline. Examine the socket. If there is a fracture of the socket wall, reposition it with an appropriate instrument. 5. Firmly replant tooth and verify the tooth's position clinically and radiographically, if possible. 6. Flexible splint for up to 4 wk.
Closed apex	1. Gently irrigate the tooth root clean with sterile saline.
Moist tooth stored in acceptable media, and/or <60 min extra oral dry time	2. Administer local anesthesia. 3. Remove coagulum from the socket with a stream of saline. Examine the socket. If there is a fracture of the socket wall, reposition it with an appropriate instrument. 4. Firmly replant tooth and verify the tooth's position clinically and radiographically, if possible 5. Flexible splint for up to 2 wk.
Closed apex	1. Remove from tooth attached necrotic soft tissue carefully with gauze.
Extra oral dry time >60 min or other reason suggesting nonviable cells	2. If available, immerse tooth in 2% stannous fluoride solution for 20 min. 3. Administer local anesthesia. 4. Remove coagulum from the socket with a stream of saline. Examine the socket. If there is a fracture of the socket wall, reposition it with an appropriate instrument. 5. Firmly replant tooth and verify the tooth's position clinically and radiographically, if possible. 6. Flexible splint for up to 4 wk.

Having the patient bite on gauze until more permanent stabilization can be arranged is acceptable. Some form of stabilization of the tooth in the ED such as a periodontal dressing (Figure 245-12) is necessary until follow-up with an oral and maxillofacial surgeon.[18,19]

All patients need antibiotics. Doxycycline, 100 milligrams PO twice a day, is the preferred antibiotic choice. For children <12 years old, penicillin VK PO four times a day (12.5 milligrams/kg/dose) is acceptable. Tetanus prophylaxis is necessary if the tooth has been contaminated by soil and the tetanus status is uncertain. Posttreatment instructions for most dental trauma are essentially the same. Patient should be instructed to maintain a soft diet for 2 weeks, brush carefully with a soft toothbrush after each meal, and use chlorhexidine 0.12% mouth rinse twice a day.[19]

Sequelae of Luxation Injuries Posttraumatic sequelae are variable. Pulp canal obliteration, pulpal necrosis, internal and external resorption of the root, and ankylosis may occur. The severity of luxation or avulsion is the most important determining factor in sequela occurrence. Transient apical breakdown occurs with all type of luxations but is especially common with extrusive and lateral luxations and avulsions. More than 50% of extrusively luxated teeth undergo pulpal necrosis within 1.5 years of the traumatic event. Close dental follow-up is essential for early identification of these sequelae.

Significant force must occur to dislodge or fracture teeth; consequently, associated **alveolar ridge fracture** is common. Care to ensure the integrity of the maxilla and mandible is also important. Stabilization of repositioned alveolar segments and associated teeth is essential for optimal results. This is best accomplished with flexible fixation placed by a general dentist or oral surgeon. Stabilization is maintained for up to 4 weeks depending on the severity of the involvement of alveolar bone. With significant alveolar ridge fracture, segments may require intermaxillary stabilization for up to 6 weeks in order to ensure adequate healing.[19]

Luxation Injuries of Primary Teeth Avulsion or luxation injuries of primary teeth are treated differently from those of permanent teeth. In patients age 6 to 12 years old, dentition is mixed, so it is very important to distinguish primary from permanent teeth. Avulsed primary teeth are never replanted. Most luxation injuries in children require no treatment and heal spontaneously. However, severe luxations of primary teeth generally require extraction of the tooth. Repositioning or replanting primary teeth risks injuring the underlying permanent teeth and thus is avoided. Intruded primary teeth are generally left alone to re-erupt into normal position. Because of the risk of damage to the permanent dentition, unless there is occlusal interference or the risk of aspiration, a dentist should manage most complicated luxation injuries of primary teeth. Referral to a general dentist for follow-up is essential to ensure optimal long-term outcome.[20]

SOFT TISSUE TRAUMA

ORAL CAVITY MUCOSAL LACERATIONS

Traumatic injuries to the soft tissue of the oral cavity are common and can involve any of the soft tissues of the mouth. Appropriate treatment remains an area of controversy. Generally, because of the vascularity of the oral tissue, lacerations of the mouth heal quickly. See the "Intraoral Mucosal Laceration" section of chapter 42, "Face and Scalp Lacerations" for management recommendations. Lacerations involving the cheek or buccal mucosa must be examined carefully for involvement of Stensen's duct, which drains the parotid salivary gland (the duct opens into the mouth opposite the upper second molar). If Stensen's duct is compromised, repair by an oral and maxillofacial surgeon or otorhinolaryngologist is indicated. Likewise, lacerations to the floor of the mouth require careful evaluation for involvement of Wharton's duct of the submandibular salivary glands.

LIP LACERATIONS

Lip lacerations are a potential cosmetic problem, so careful closure is essential. (see chapter 42 for management recommendations).[21]

FRENULUM LACERATIONS

Laceration of the maxillary labial frenulum, unless unusually large, does not require repair. These lacerations can be very painful, so provide adequate analgesia. Because of the vascularity of adjacent tissue, lacerations to the lingual frenulum of the tongue usually do need to be repaired. An absorbable suture such as 4-0 chromic gut or Vicryl□ is appropriate.

TONGUE LACERATIONS

Lacerations of the tongue require special consideration because bleeding and delayed swelling can compromise the airway. **Simple linear lacerations less than 1 cm involving the central portion of dorsal surface of the tongue and that do not gape open heal well without repair.** Except for the most extensive tongue lacerations, suturing does not necessarily improve outcome or reduce morbidity. All lacerations that bisect the tongue require repair. Partial amputations can be successfully replanted with the appropriate microsurgical techniques.[21]

Local anesthesia can be obtained by local infiltration or topically by placing 4% lidocaine-soaked gauze for 5 minutes on the laceration. Bilateral lingual nerve blocks can be used for lacerations of the anterior two thirds of the tongue that cross the midline. Repair of the tongue, especially in children, presents a special challenge, and many adjuncts such as a dental bite block or a Molt mouth prop can be helpful in keeping the mouth open. A piece of 4 × 4 gauze can be used to grasp the tongue, or a surgical towel clamps or a large caliber suture such as 0-silk or nylon placed through the anterior portion of the anesthetized tongue can facilitate retraction and control the tongue.[21]

When laceration repair is warranted, absorbable sutures such as 4-0 chromic gut or Vicryl should be used. Sutures should be placed so as to include the muscular layer and the superficial mucosal layers of the tongue. Wound edges should be approximated and closed very loosely to allow for swelling of the tongue, which can be significant. Placing a closed hemostat between the suture and the tongue while tying can help prevent overtightening of the suture. The constant motion of the tongue quickly unties sutures in the mouth so sutures should be tied with at least four square knots. If feasible, the sutures can also be placed so that the knots are buried into the wound. All patients should be instructed rinse several times daily with a saline or chlorhexidine 0.12% mouth rinse.[21]

DENTAL LOCAL ANESTHESIA TECHNIQUES

Competence in dental local anesthesia is a useful skill for the emergency provider. The maxillary teeth can be anesthetized using local infiltrative or supraperiosteal techniques, which are easily learned by reading.

ANATOMY

The trigeminal nerve is the largest cranial nerve, and although it has important motor function, it is primarily a sensory nerve (**Figure 245-14**). It divides into three main braches: the ophthalmic nerve, maxillary nerve, and mandibular nerve. The maxillary nerve provides sensory innervation to the maxilla and associated structures including the maxillary teeth and gingiva and oral mucous membranes. The third division, or mandibular nerve, is the largest of the three branches. The mandibular nerve has three main divisions. The first is the long buccal nerve that provides sensory innervation to the mucosa of cheek and buccal gingiva of the mandibular molars. The second is the lingual nerve that runs superficial to the internal pterygoid muscle and innervates the anterior two thirds of the tongue, lingual gingiva, and floor of the mouth. Finally, the largest branch is the inferior alveolar nerve, which accompanies the inferior alveolar vein and artery in a neurovascular bundle passing between the ramus of the mandible and the sphenomandibular ligament to enter the mandibular canal. It divides at the region of the premolars with the mental nerve exiting the mental foramen to innervate the soft tissue of the lip and chin, and the incisal nerve continuing within the mandibular canal to innervate the teeth and gingiva.

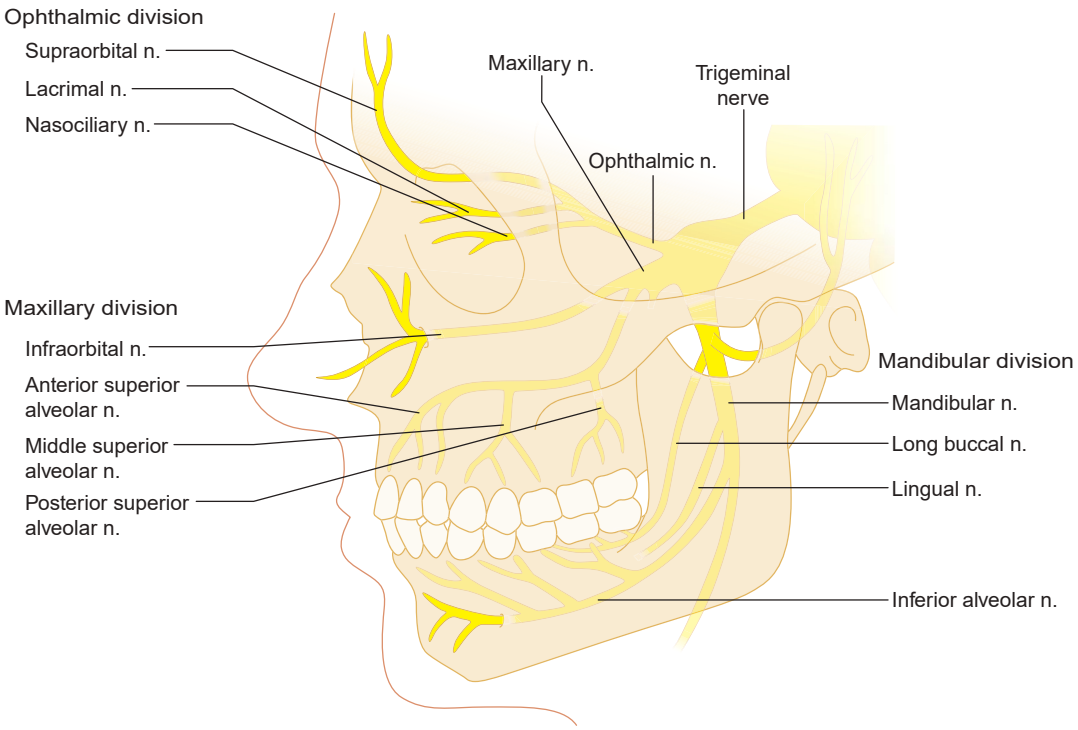

FIGURE 245-14. The trigeminal nerve and its three main divisions.

EQUIPMENT

Specialized equipment is useful when giving dental injections, especially the inferior alveolar nerve block. Equipment includes:

1. A monojet aspirating dental syringe
2. Dental cartridges of anesthetic solution
3. Disposable 27-gauge, 32-mm needle
4. Topical anesthetic, if possible

If an aspirating dental syringe is not available any 3-mm aspirating syringe can be used. With larger syringes, the inferior alveolar nerve block is difficult because the syringe barrel interferes with the proper positioning of the needle.

Position the patient in a dental chair or stretcher in a semi-reclined position with the head firmly against the headrest. Anticipate sudden movement of the patient. An overhead light is essential because dental injections require good visibility and control at all times to ensure safety of both the patient and the physician.

MAXILLARY INJECTIONS

Supraperiosteal Infiltration Maxillary cortical bone is thin and porous enough to allow the diffusion of anesthetic solution to reach the apex of the root and effectively anesthetize the tooth. **Figure 245-15** illustrates the supraperiosteal infiltration technique. The upper lip or cheek, depending on which tooth you are anesthetizing, is pulled up and taut. With the bevel facing toward the bone, the needle is inserted at the height of the buccal fold adjacent to the tooth. The needle should be directed along the long axis of the tooth and inserted to height of or slightly above the apex of the tooth. In most cases, the needle only needs to be inserted a few millimeters. Aspirate, and if negative, then slowly inject 0.5 to 1 mL of anesthetic solution. It is important to remember that the root of the canine is significantly longer than that of other teeth and that, in general, the roots of teeth are inclined in a distal direction. The goal is to deposit the anesthesia at or above the apex of the desired tooth.[22-24]

Palatal Injection
Supraperiosteal infiltration of the maxillary teeth provides adequate anesthesia for pain control for a toothache, but not for procedures such as management of a dry socket or avulsed tooth. This may also require anesthesia of the palatal tissue, which can simply be accomplished by local infiltrative anesthesia. With a fine-gauge needle, puncture the palatal mucosa about half way up the palate adjacent to the target tooth (**Figure 245-16**). Inject 0.1 mL of anesthetic solution. Blanching of the surrounding tissue is common, and this injection is usually quite uncomfortable. If a larger area of anesthesia is required, anesthesia of the anterior portion of the palate can be obtained by injecting anesthesia over the incisive foramen. The landmark is the incisive papilla. Anesthesia to the unilateral posterior portion of the palate can be obtained by injecting a small amount of anesthesia over the greater palatine foramen.[22-24]

MANDIBULAR INJECTIONS

In contrast to the maxilla where the cortex of overlying bone is relatively thin and infiltrative anesthesia is usually effective, the mandibular boney cortex is relatively thick and the inferior alveolar nerve block is usually required to gain adequate anesthesia of the mandibular molars and premolars. A mental nerve block, as described in chapter 36, "Local and Regional Anesthesia," can provide anesthesia for the premolars, canines, and incisors, as well as soft tissue of the lip and chin, when properly administered. The bone adjacent to the canines and incisors is thinner and more amenable to infiltrative anesthesia. Also, soft tissue anesthesia of the tongue can be obtained using the basics of the inferior alveolar nerve block. Injection of the long buccal nerve may be necessary for incision and drainage of dental abscesses in the mandibular molar region, and this technique will be described.

Inferior Alveolar Nerve Block (Direct Technique) The patient should be instructed to open the mouth widely to ensure good visualization of anatomic landmarks (**Figure 245-17**). First, the physician needs to palpate the greatest depth of the anterior border of the ramus or coronoid notch with the index finger or thumb. Then with that

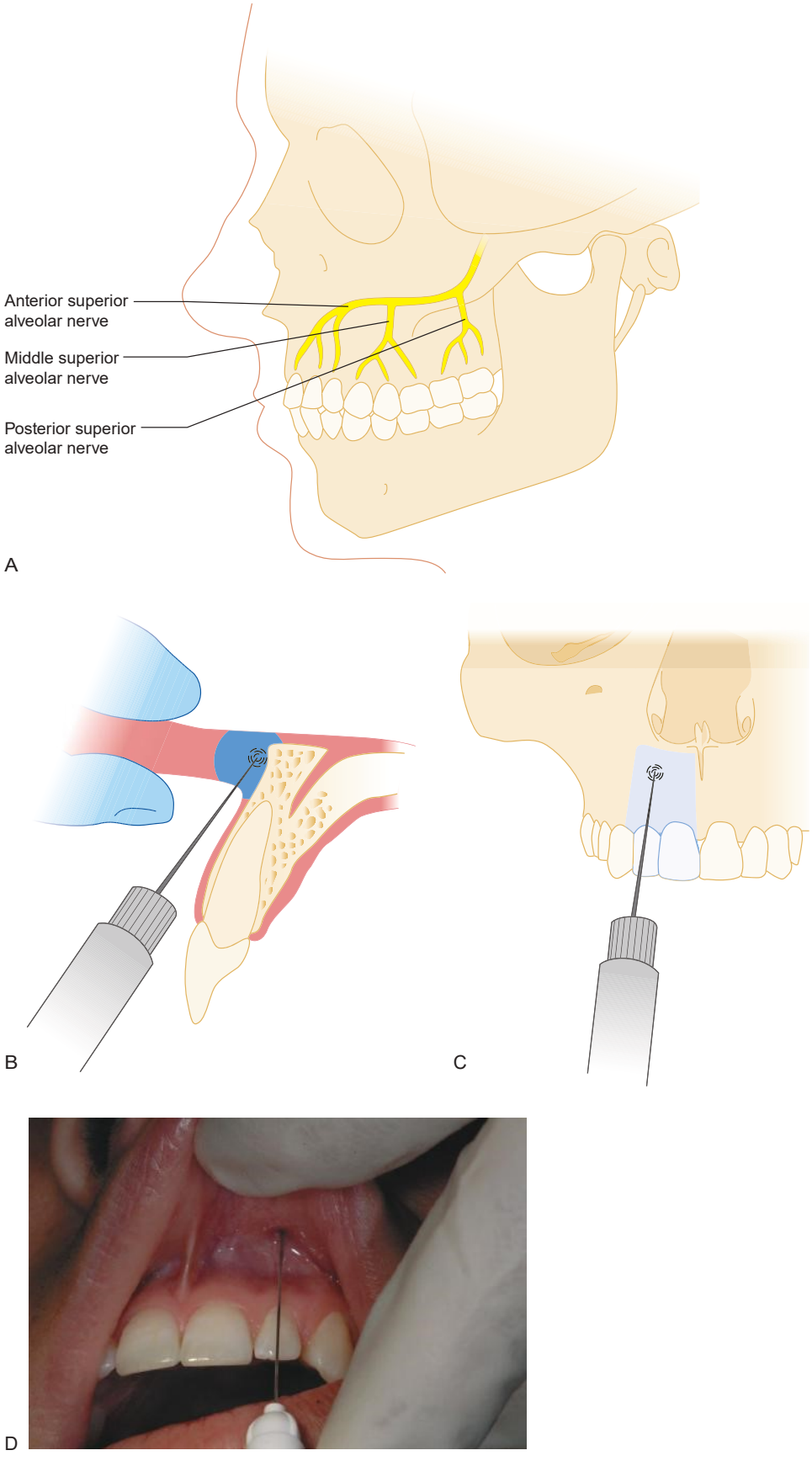

FIGURE 245-15. The supraperiosteal infiltration. **A.** The maxillary nerve and innervation of the maxillary teeth by the posterior superior alveolar nerve, the middle superior alveolar nerve, and the anterior superior alveolar nerve. **B.** With the lip or cheek pulled taught, the needle is inserted at the height of the buccal fold. Note: the 2-3 concentric circles of dotted lines around the needle tip indicate the area where the anesthesia is deposited. **C.** The needle is directed along the long axis of the tooth, and anesthesia is deposited just superior to the area of the root apex. **D.** Clinical photograph depicting a supraperiosteal infiltration of the maxillary lateral incisor.

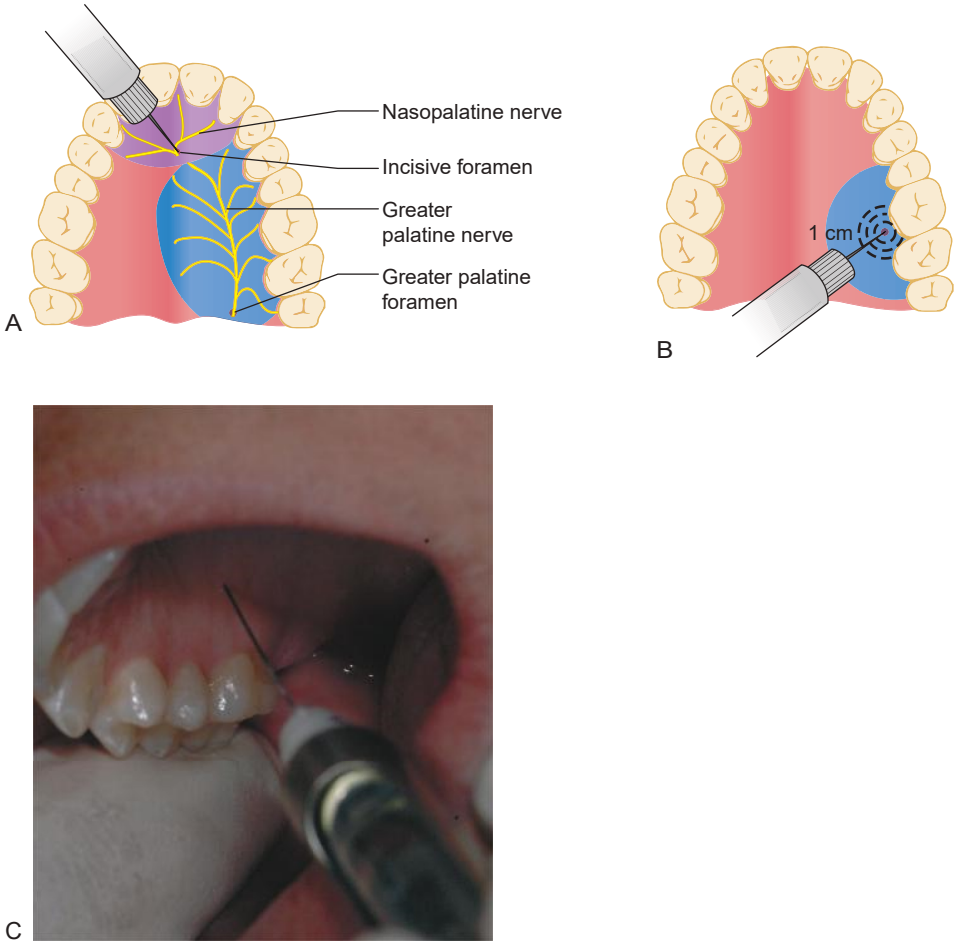

FIGURE 245-16. Palatal Infiltration. **A.** The innervation to the soft tissue of the hard palate. The posterior portion of the palate is innervated by bilateral greater palatine nerves after they each exit their respective greater palatine foramens (see blue shaded area). The anterior portion of the hard palate is innervated by the nasopalatine nerve after exiting the incisive foramen. Injection over the incisive foramen (shown) will result in anesthesia to the purple shaded area. **B.** A fine-gauge needle is inserted into the palatal tissue about 1 cm below the gingival margin, and a small amount of anesthesia is injected. Blanching of the palatal tissue surrounding the injection site is noted. This will provide anesthesia to the tissue adjacent to area injected as shown. A similar injection over the area of the greater palatine foramen or the incisive foramen will provide anesthesia to their respective zones. **C.** Clinical photograph depicting a palatal infiltration.

finger or thumb, the tissue is retracted toward the cheek revealing the pterygotemporal depression that is between the raised ridge of mucosa (pterygomandibular raphe) medially and coronoid notch laterally. With the syringe directed from the opposite premolar area, the needle is inserted into the pterygomandibular depression at a point 1 to 1.5 cm above the occlusal plane. This is approximately at the level of index finger or thumb. The needle is then slowly advanced until bone is contacted (about 20 to 25 mm). Once bone has been contacted, the needle should be withdrawn about 1 to 2 mm and then aspirated. If no blood is aspirated, then about 1.5 mL of anesthetic solution should be slowly injected. The needle then should be withdrawn about half of the distance, and the remainder of the dental carpule injected. This will ensure the anesthesia of the lingual nerve.[22-24] With experienced operators, the direct technique has about a 20% to 25% failure rate. Most commonly, failure is due to using too low of a point of injection. This places the anesthesia below the sphenomandibular ligament, which impedes its flow toward the inferior alveolar nerve. Repositioning the syringe higher above the occlusal plane will usually result in a successful injection.

Lingual Nerve Block If only lingual nerve anesthesia is desired, then the above technique can be followed except the needle need only be initially inserted about 10 mm, aspirated, and then the anesthetic agent instilled. This will provide anesthesia to the ipsilateral half of the anterior two thirds of the tongue (**Figure 245-18**).[22-24]

Long Buccal Injection If buccal soft tissue anesthesia is required, then this injection should be used. The needle is inserted into the mucosa in the buccal vestibule adjacent to the second or third mandibular molars (Figure 245-18). [22-24]

Injection Complications The normal vasovagal symptoms including syncope that can occur with any injection may occur during or after a dental injection. Flushing and elevation of the heart rate can also occur. Mild to severe allergic reactions are possible. Due to the close proximity of the inferior alveolar artery and vein and the pterygoid plexus, a potential complication of inferior alveolar nerve block is traumatic hematoma formation. These are usually self-limiting and treated with ice to the face but can be quite uncomfortable and an aesthetic issue for more than a week. Due to the superficial nature of the lingual nerve relative to the inferior alveolar nerve, inadvertent injury to the lingual nerve with subsequent temporary or permanent paresthesia can occur. This not only can affect sensation to the anterior two thirds of the affected side but also may affect taste because taste fibers run with the lingual nerve. Postinjection trismus, due to local trauma to the muscles of mastication, can occasionally occur. Needle breakage, although uncommon, has been reported, especially with improper technique such as bending the needle prior to use or redirecting the needle while injecting. Finally, unintentional injection too posteriorly may result in injection into the parotid region and can cause temporary facial nerve palsy.[24]

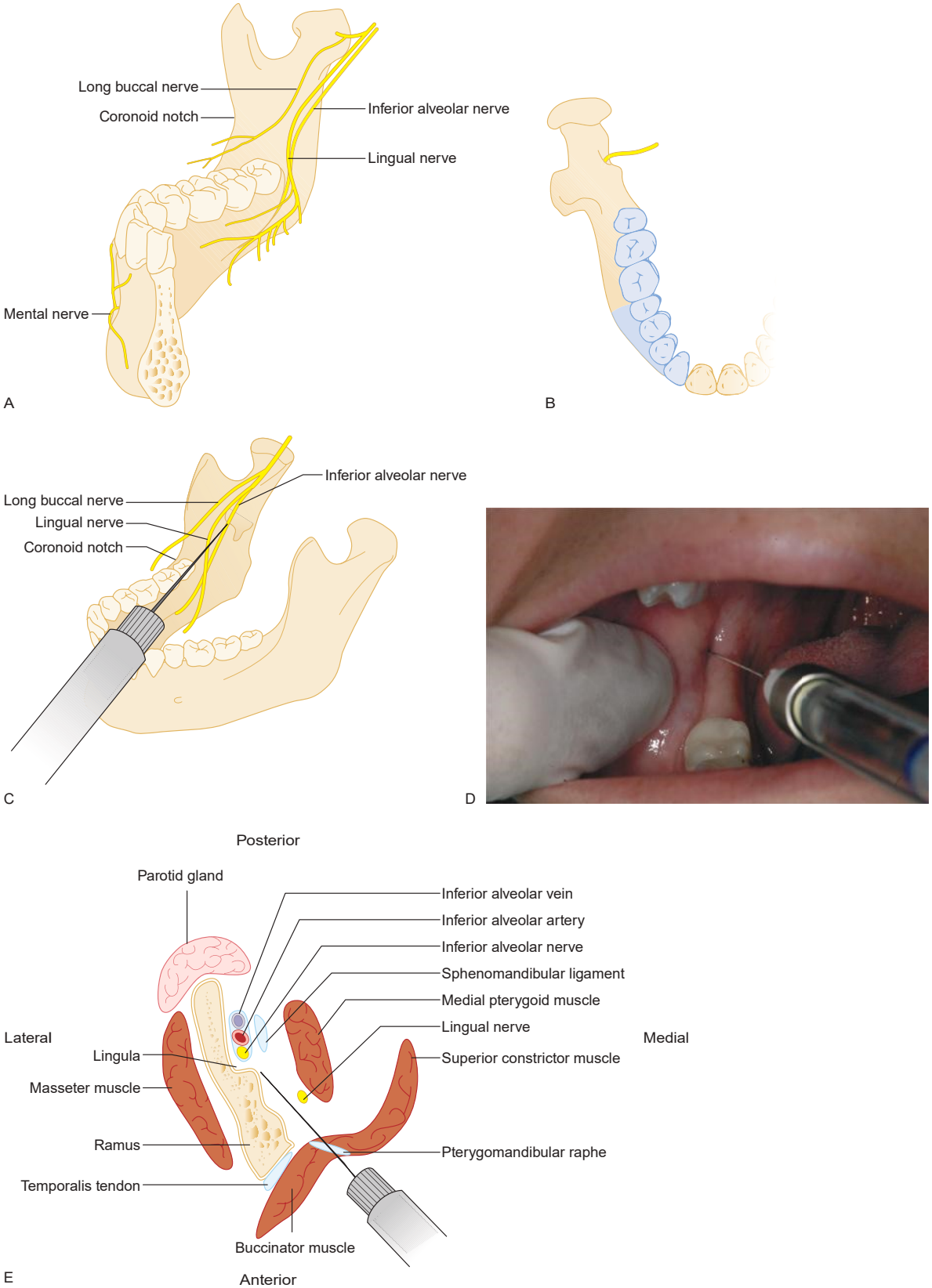

FIGURE 245-17. Inferior alveolar nerve block (direct technique). **A.** Shows the anatomy of the inferior alveolar nerve as it enters the mandibular foramen in the pterygomandibular space. The lingual nerve lies superficial and medial to the inferior alveolar nerve. The coronoid notch is noted. **B.** The area of anesthesia obtained with a successful inferior alveolar nerve block. Usually the lingual nerve is also blocked, which provides anesthesia to the floor of the mouth, lingual gingiva, and the anterior two thirds of the tongue. **C.** The syringe should be directed from the contralateral premolar area about 1 to 1.5 cm above the mandibular plane. It is inserted about 20 to 25 mm until bone is touched in an area above the lingula. The needle should then be withdrawn 1 to 2 mm and aspirated for blood before injecting anesthesia. The sphenomandibular ligament attaches to the lingula and prevents the anesthesia from reaching the inferior alveolar nerve if the injection is too low. **D.** Clinical photograph of the direct technique. Note the point of injection in the pterygomandibular depression just lateral to the pterygomandibular raphe. **E.** A diagram of a transverse section of the pterygomandibular fossa at the level of an inferior alveolar nerve injection. Note that the needle passes through the buccinator muscle to an area just superior to the lingula.

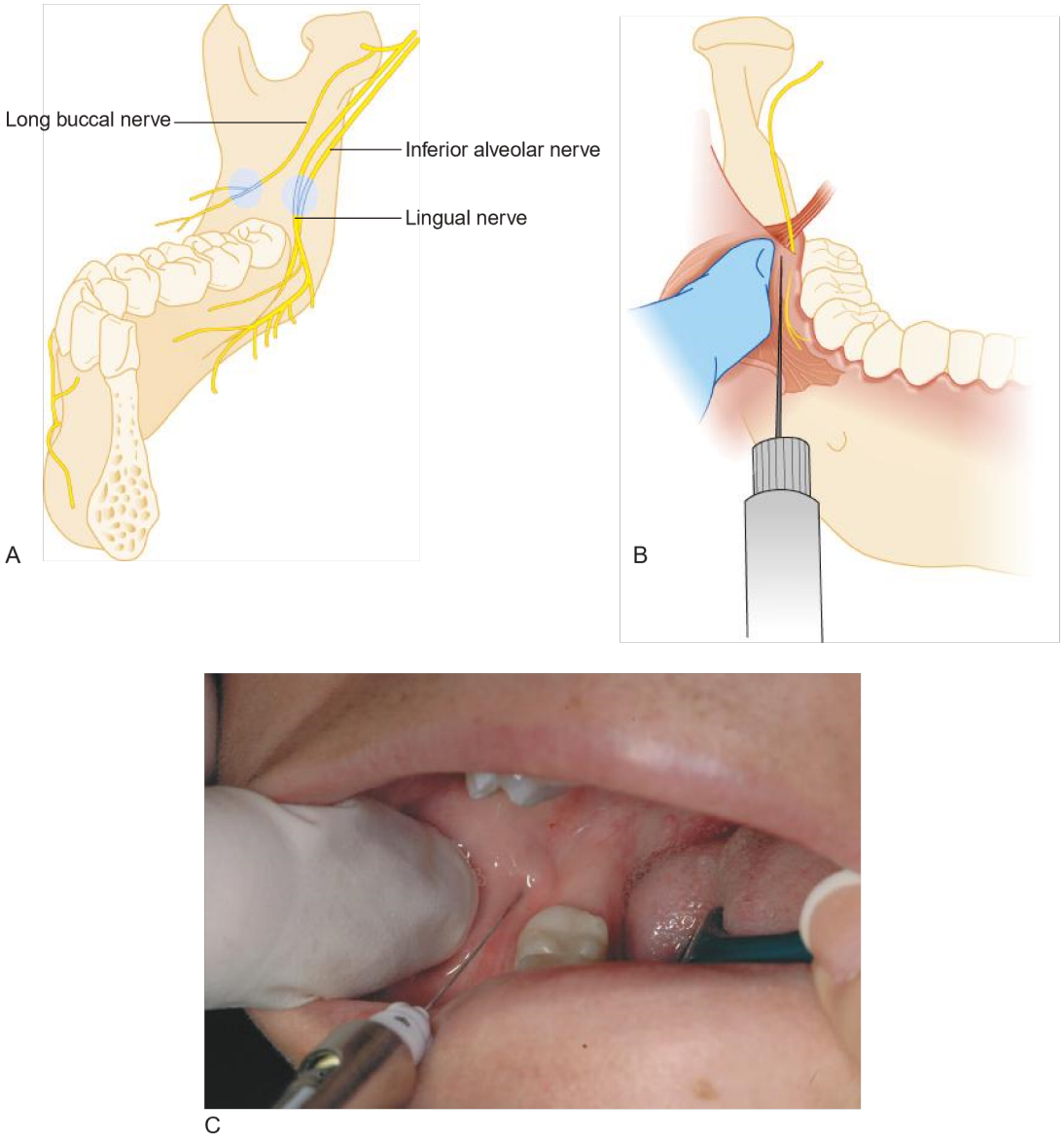

FIGURE 245-18. The lingual nerve block and the long buccal nerve block. **A.** The shaded areas represent the area in which anesthesia should be deposited for their respective blocks. The lingual nerve runs superficial and medial to the inferior alveolar nerve and can be easily anesthetized as part of the inferior alveolar nerve block. The lingual nerve block can be performed by depositing anesthesia about half the depth of the inferior alveolar nerve block. **B.** A diagrammatic representation of the long buccal nerve block. The long buccal nerve requires a separate injection of a small quantity of anesthetic just lateral to the molars in the buccal mucosa. **C.** Clinical photograph of the long buccal nerve block.

REFERENCES

The complete reference list is available online at www.TintinalliEM.com.

CHAPTER 246

Neck and Upper Airway

Nicholas D. Hartman

This chapter reviews infectious and noninfectious conditions that can obstruct the upper airway. These disorders must be recognized quickly because early airway management may be lifesaving. Neck trauma is discussed in the chapter 260, "Trauma to the Neck," and angioedema is discussed in the chapter 14, "Anaphylaxis, Allergies, and Angioedema."

NECK AND UPPER AIRWAY INFECTIONS

PHARYNGITIS/TONSILLITIS

Viruses account for the majority of cases of pharyngitis or tonsillitis. Acute viral pharyngitis is most commonly caused by rhinovirus but can be caused by multiple other viral agents (**Table 246-1**).[1]

VIRAL PHARYNGITIS

Viral pharyngitis generally displays a vesicular or petechial pattern on the soft palate and tonsils and is associated with rhinorrhea. However, in

TABLE 246-1 Microbial Causes of Acute Pharyngitis

Pathogen	Syndrome/Disease	Estimated % of Cases*
Viral		
Rhinovirus (100 types, 1 subtype)	Common cold	15–20
Coronavirus (3+ types)	Common cold	>5
Adenovirus (types 3, 4, 7, 14, 21)	Pharyngoconjunctival fever, acute respiratory disease	6
Herpes simplex virus (type 1, 2)	Gingivitis, stomatitis, pharyngitis	4
Parainfluenza virus (types 1–4)	Common cold, croup	2
Influenza virus (types A, B)	Influenza	2
Respiratory syncytial virus	Bronchiolitis, pharyngitis	1–2
Coxsackievirus A (types 2, 4, 5, 6, 8, 10)	Herpangina	<1
Epstein-Barr virus	Infectious mononucleosis	<1
Cytomegalovirus	Infectious mononucleosis	<1
Human immunodeficiency virus type 1	Acute retroviral syndrome	<1
Bacterial		
Streptococcus pyogenes (GABHS)	Pharyngitis, tonsillitis, scarlet fever	10–22
Fusobacterium necrophorum	Pharyngitis, tonsillitis, Lemierre's syndrome	5–10
Streptococcus dysgalactiae subspecies *equisimilis* (formerly group C β-hemolytic streptococci)	Pharyngitis, tonsillitis	3–5
Neisseria gonorrhoeae	Pharyngitis	<1
Corynebacterium diphtheriae	Diphtheria	<1
Arcanobacterium haemolyticum	Pharyngitis	<1
Chlamydial		
Chlamydia pneumonia	Pneumonia, bronchitis, pharyngitis	<1
Mycoplasmal		
Mycoplasma pneumonia	Pneumonia, bronchitis, pharyngitis	<1

Abbreviation: GABHS = group A β-hemolytic *Streptococcus*.
*Estimates of percentage of all cases of pharyngitis due to the indicated organism.

patients with nonstreptococcal pharyngitis (mostly viral), 16% have tonsillar exudate, 55% have cervical adenopathy, and 64% lack cough.[2] Most cases of viral pharyngitis require no specific diagnostic testing. There are three notable exceptions where testing may be indicated: suspected influenza, infectious mononucleosis, and acute retroviral syndrome. See Centers for Disease Control and Prevention influenza Web site for testing and treatment recommendations (http://www.cdc.gov/flu/). Infectious mononucleosis, influenza herpesvirus, and cytomegalovirus infections are discussed in the chapter 153, "Serious Viral Infections." The acute retroviral syndrome of early human immunodeficiency virus infection can also mimic mononucleosis. Symptoms of pharyngitis develop 2 to 4 weeks after exposure and resolve within 2 weeks. See the chapter 154, "Human Immunodeficiency Virus Infection" for recommendations on testing and treatment. Non–human immunodeficiency virus, noninfluenza viral pharyngitis should be treated symptomatically with oral hydration, antipyretics, analgesics, and rest. Patients unable to tolerate oral fluids or who become dehydrated should be given IV fluids.

BACTERIAL PHARYNGITIS

Group A β-Hemolytic *Streptococcus* **Group A β-hemolytic *Streptococcus* (GABHS) pharyngitis,** Lancefield group A species of *Streptococcus pyogenes*, is responsible for 5% to 15% of pharyngitis in adults.[1,2] After an incubation period of 2 to 5 days, patients develop the sudden onset of sore throat, painful swallowing, chills, and fever. Headache, nausea, and vomiting are common. Signs and symptoms of GABHS pharyngitis include marked erythema of the tonsils and tonsillar pillars (found in 62% of cases); tonsillar exudate (32%); and enlarged, tender cervical lymph nodes (76%).[2] A 2012 epidemiologic study found that only 6% of GABHS cases had fever and 28% had cough.[2] Patients may have uvular edema, myalgias, and malaise but are less likely to have rhinorrhea or conjunctivitis compared to viral pharyngitis.

Uvula edema, sometimes referred to as *Quincke's edema*, can be associated with upper airway infections such as GABHS pharyngitis, peritonsillar abscess, or epiglottitis. It can also be idiopathic. If it is an isolated finding and symptoms are uncomfortable to the patient, dexamethasone, 4 milligrams IV or PO, can be given as a single dose in the ED.

The original Centor criteria listed four clinical indicators of GABHS pharyngitis: (1) tonsillar exudates, (2) tender anterior cervical adenopathy, (3) absence of cough, and (4) history of fever.[2] The Centers for Disease Control and Prevention reversed its prior recommendation for empiric treatment based on clinical findings in 2012 in concert with the Infectious Diseases Society of America.[3] The Centers for Disease Control and Prevention and Infectious Diseases Society of America recommend using two or more Centor criteria as a threshold for selecting patients for rapid strep testing and treating only those with positive tests.[3,4] Guidelines do not recommend throat cultures in adult patients with one or fewer Centor criteria or routine throat culture for those with negative rapid strep tests, unless considering other bacterial pathogens[3]

Untreated, GABHS infection lasts 7 to 10 days. Antibiotic therapy of GABHS hastens resolution by 1 to 2 days if initiated within 2 to 3 days of symptom onset and prevents suppurative complications and rheumatic fever, although not glomerulonephritis.[3] **GABHS has never been resistant to penicillin, so penicillin remains the recommended first-line drug for GABHS.**[5,6] Adults should receive a single IM dose of 1.2 million units of benzathine penicillin G, 500 milligrams of penicillin VK PO two times daily for 10 days, or amoxicillin 500 milligrams PO two times daily or 1000 milligrams one time daily. A first-generation cephalosporin antibiotic or clindamycin may be used for penicillin-allergic patients.[5] A single dose of PO or IM dexamethasone in immunocompetent adults with moderate to severe pharyngitis can achieve an earlier onset of pain relief and a shorter duration of pain.[7]

Other Causes of Bacterial Pharyngitis Several other bacteria can cause pharyngitis, although these infections are less common (Table 246-1). ***S. dysgalactiae* subspecies *equisimilis***, previously known as β-hemolytic groups C and G streptococci, are important pathogens causing pharyngitis, skin infections, and more serious infections such as meningitis or toxic shock syndrome in the elderly or immunocompromised.[8] *S. dysgalactiae* subspecies *equisimilis* frequently colonizes the upper respiratory tract (60% who are culture positive are asymptomatic),[8] so distinguishing acute infection from a carrier state may be difficult;[9] treatment is recommend for patients with acute symptoms.[8] *S. dysgalactiae* subspecies *equisimilis* pharyngitis is almost uniformly susceptible to penicillin.[8] Clindamycin and fluoroquinolones are alternatives.[8]

Fusobacterium necrophorum, a gram-negative anaerobe,[10] is the causative agent in **Lemierre's syndrome**, a complication of pharyngitis causing suppurative thrombophlebitis of the internal jugular vein, with or without bacteremia and septic emboli. Suspect *F. necrophorum* in adolescents or young adults with worsening symptoms and neck swelling.[11] Treatment is with penicillin, clindamycin, or third-generation cephalosporins; *F. necrophorum* resistance to macrolides is high.[11]

Gonococcal pharyngitis is usually associated with genital infection and is treated by the same antibiotics. **Diphtheria** is caused by *Corynebacterium diphtheriae* and is rare in well-immunized populations. It is characterized by a slow onset of mild to moderate pharyngeal discomfort and low-grade fever. On physical examination, a gray membrane is

seen adherent to the tonsillar or pharyngeal surface and may extend to the uvula, soft palate, pharynx, and larynx. Treatment is with diphtheria antitoxin and metronidazole to prevent transmission to others.

PERITONSILLAR ABSCESS

A peritonsillar abscess is a collection of purulent material between the tonsillar capsule, the superior constrictor, and palatopharyngeus muscles. Risk factors include periodontal disease, smoking, chronic tonsillitis, multiple trials of antibiotics, and previous peritonsillar abscess.[12] Peritonsillar abscess develops primarily in adolescents and young adults without seasonal variation as previously thought.[12,13] Although peritonsillar abscesses are typically polymicrobial infections, in patients 15 to 24 years of age, *Fusobacterium necrophorum* has been the most common organism in many communities.[13,14]

CLINICAL FEATURES AND DIAGNOSIS

Patients with peritonsillar abscess (adolescents and adults) appear ill and present with sore throat (99%), fever (54%), malaise, odynophagia, dysphagia, and/or otalgia.[15] Physical signs include inferior and medial displacement of the infected tonsil(s) (46%), contralateral deflection of the swollen uvula (43%), tender cervical lymphadenopathy (41%), trismus (32%), muffled voice ("hot potato voice"), palatal edema, and dehydration[15] (**Figure 246-1**). The differential diagnosis of a peritonsillar abscess includes peritonsillar cellulitis, mononucleosis, lymphoma, herpes simplex tonsillitis, retropharyngeal abscess, neoplasm, and internal carotid artery aneurysm. In peritonsillar cellulitis, erythema and edema of the tonsillar pillar and soft palate are evident, but pus has not yet formed. Diagnosis of a peritonsillar abscess is often made by history and physical examination alone. When the diagnosis is in question, intraoral US has a sensitivity of 89% to 95% with a specificity of 79% to 100% for peritonsillar abscess.[16] CT scan with contrast is indicated if there is concern for spread beyond the peritonsillar space or lateral neck space complications.[16]

TREATMENT

Treatment options include drainage of the abscess by needle aspiration, incision and drainage, or, rarely, immediate tonsillectomy. Choice of treatment depends on clinical symptoms, degree of patient cooperation, history of previous tonsil disease, and healthcare personnel experience. There is no difference in outcome when comparing needle aspiration with incision and drainage.[16] Abscess tonsillectomy ("quinsy tonsillectomy") should only be considered when patients have strong indication for tonsillectomy, such as sleep apnea, recurrent tonsillitis, or recurrent peritonsillar abscess.[16] Needle aspiration is minimally invasive, less painful than incision and drainage, and may be performed by general or specialized medical personnel. Approximately 90% of patients will be treated effectively after a single needle aspiration.[16]

Needle aspiration should be performed by an individual trained in the technique. First, apply lidocaine spray or gel or benzocaine-tetracaine spray to the overlying mucosa. Then inject 1 to 2 mL of lidocaine with epinephrine into the mucosa of the anterior tonsillar pillar using a 25-gauge needle **The drainage needle should penetrate no more than 1 cm because the internal carotid artery usually lies laterally and posterior to the posterior edge of the tonsil.** The plastic sheath of the needle can be cut 1 cm from its tip to serve as a guard. If the internal carotid artery lies more medial and anterior, it can usually be palpated in this area. Once adequate anesthesia is achieved, introduce an 18-gauge needle just lateral to the tonsil, approximately halfway between the base of the uvula and the maxillary alveolar ridge, until the abscess cavity is encountered and pus is aspirated. Often, multiple aspirations may be required to find the abscess. If not done previously, a contrast CT scan of the neck is recommended when the results of needle aspiration are negative and a parapharyngeal or retropharyngeal space process is suspected.

Initial therapy should include a 10-day course of antimicrobials effective against group A *Streptococcus* and oral anaerobes (including *F. necrophorum*). Proven agents are penicillin VK plus metronidazole[15] or clindamycin for penicillin-allergic patients. Toxic patients or patients unable to take medicine PO should receive piperacillin-tazobactam, 3.375 grams IV, or similar agent. Single IV use of high-dose steroid (methylprednisolone, 125 milligrams, or dexamethasone, 10 milligrams) in addition to antibiotics and drainage improves severity and duration of pain.[15,17] Provide follow-up within 24 to 36 hours of aspiration, with instructions to return to the ED if worse. If the patient is not improving, consider repeating the aspiration, otolaryngologic consultation for incision and drainage or tonsillectomy, or obtaining a CT scan to confirm or reject the diagnosis. Complications of a peritonsillar abscess include airway obstruction, rupture of the abscess with aspiration of the contents, hemorrhage secondary to erosion of carotid sheath, retropharyngeal abscess, mediastinitis, and poststreptococcal sequelae.

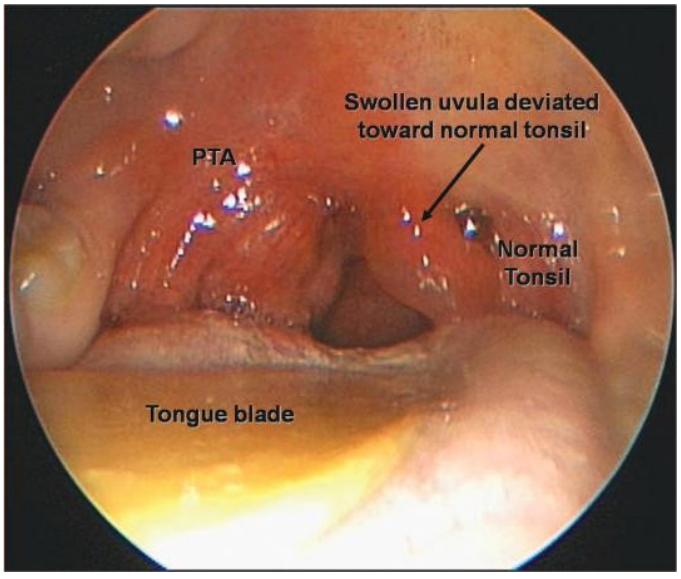

FIGURE 246-1. Right peritonsillar abscess (PTA) displacing right tonsil medially and the uvula toward the normal left tonsil. Abscess is between the right tonsil and the superior constrictor muscles.

ADULT EPIGLOTTITIS □ SUPRAGLOTTITIS□

Epiglottitis is an inflammatory condition, usually infectious, primarily of the epiglottis but often including the entire supraglottic region (many prefer the term *supraglottitis*). It can lead to rapid airway obstruction. Prior to the introduction of a conjugate vaccine for *Haemophilus influenzae* type b in the 1980s, most cases of epiglottitis affected children age 1 to 5 years. In the postvaccine era, the dramatic decline in pediatric cases has confined the disease primarily to adults, with an estimated mean age of 45 years.[18,19] Most cases in adults are caused by *Streptococcus* species, *Staphylococcus* species, viruses, and fungi, although most frequently, no organism can be isolated.[19] Risk factors for mortality in patients with epiglottitis are advanced age and male sex.[20]

CLINICAL FEATURES AND DIAGNOSIS

Symptoms are typically a 1- to 2-day history of worsening dysphagia, odynophagia, and dyspnea, particularly in the supine position. The clinical triad of the "three Ds" (drooling, dysphagia, and distress) is a classic but infrequent presentation. Other symptoms are fever, tachycardia, cervical adenopathy, and anterior neck tenderness with pain on gentle palpation of the larynx and upper trachea. Stridor is primarily inspiratory. Patients often position themselves sitting up, leaning forward, mouth open, head extended, and panting.

Diagnosis is clinical and confirmed by radiographs or transnasal fiberoptic laryngoscopy. Lateral cervical soft tissue radiographs demonstrate obliteration of the vallecula, swelling of the aryepiglottic folds,

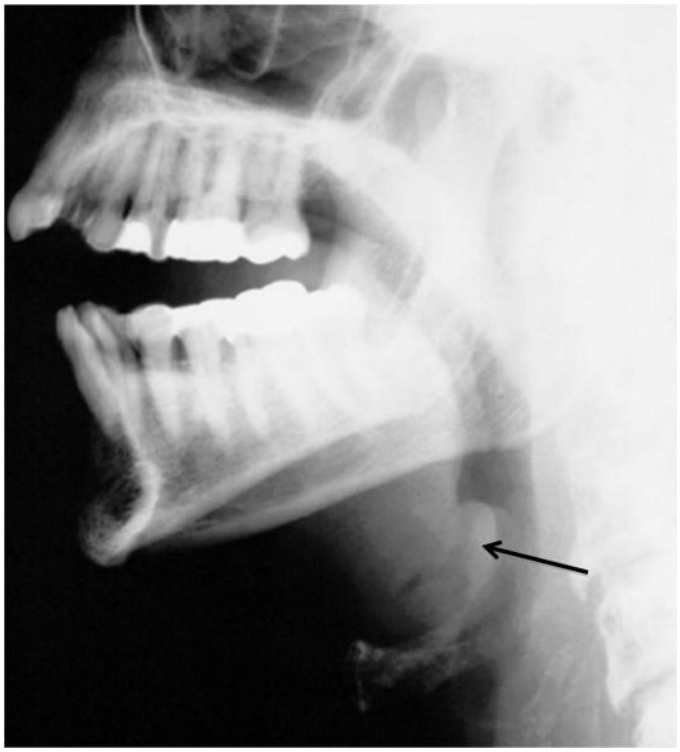

FIGURE 246-2. Acute epiglottitis. *Arrow* points to thickened epiglottis resembling a thumb print on a soft tissue lateral radiograph.

edema of the prevertebral and retropharyngeal soft tissues, and ballooning of the hypopharynx (**Figure 246-2**). The epiglottis appears enlarged and thumb-shaped. Direct laryngoscopy examination can confirm the diagnosis in adults if necessary but should be done carefully to avoid sudden, unpredictable airway obstruction. **Patients with worsening dyspnea in the supine position should *not* be sent to the CT scanner; CT of the neck is not needed to make the diagnosis.**

TREATMENT

Obtain immediate otolaryngologic consultation for suspected epiglottitis. Be prepared to establish a definitive airway. Patients should not be left unattended, and they should remain sitting up. Initial treatment consists of supplemental humidified oxygen, IV hydration, cardiac monitoring, pulse oximetry, and IV antibiotics. Humidification and hydration can help decrease the risk for sudden airway blockage. Steroids are often given to decrease airway inflammation and edema (methylprednisolone, 125 milligrams IV).

In adults, the need for intubation usually can be determined by transnasal fiberoptic examination of the supraglottis. Intubation is generally accomplished by "awake" fiberoptic intubation in the operating room, with preparations for immediate awake tracheostomy or cricothyrotomy. In cases of airway obstruction in the ED, be prepared for a very difficult intubation secondary to the swollen, distorted anatomy. In the case of intubation failure, the last resorts for preserving the airway are cricothyrotomy and needle cricothyrotomy.

Current antibiotic recommendations are cefotaxime 50 milligrams/kg IV every 8 hours plus vancomycin 15 milligrams/kg every 12 hours.[21] Alternative antibiotics include ampicillin-sulbactam, ceftriaxone, or piperacillin-tazobactam.[21] Respiratory fluoroquinolones are an option for patients with severe penicillin allergies.

RETROPHARYNGEAL ABSCESS

The retropharyngeal space is a potential space anterior to the prevertebral fascia that extends from the base of the skull to the tracheal bifurcation. In adults, a retropharyngeal abscess is usually due to intraoral procedures, trauma, foreign bodies such as a fishbone, or extension from odontogenic infection.[22] Cultures from retropharyngeal abscesses are usually polymicrobial: group A β-hemolytic streptococci, *Staphylococcus aureus* (including methicillin-resistant *S. aureus*), *H. influenzae*, and *Bacteroides*, *Peptostreptococcus*, and *Fusobacterium* species.

CLINICAL FEATURES AND DIAGNOSIS

The most common symptoms in adults are sore throat, dysphagia, neck pain, and less commonly, stridor. In addition, patients may also have complaints of cervical lymphadenopathy, poor oral intake, muffled voice, and respiratory distress. Visible neck swelling is not common.

A lateral soft tissue radiograph of the neck taken during inspiration with moderate cervical extension can demonstrate thickening and protrusion of the retropharyngeal wall, classically with 5 to 7 cm of prevertebral widening at the second cervical vertebra.[23] However, contrast-enhanced CT scan of the neck is the test of choice for diagnosis of a retropharyngeal abscess.[24] Early CT findings may reflect reactive, nonsuppurative edema, mild fat stranding with discernible tissue planes, linear fluid, minimal mass effect, and no associated enhancement. Necrotic nodes with central low attenuation and ring enhancement reflect an abscess (**Figure 246-3**). A patient with airway distress should not be sent unobserved for CT scanning.

TREATMENT

Obtain immediate otolaryngologic consultation. Provide IV hydration and antibiotic treatment with either clindamycin or cefoxitin IV; alternatively, piperacillin-tazobactam or ampicillin-sulbactam may be used.[21] Although a few patients with small abscess cavities may be managed with IV antibiotics alone, most patients will require surgical intervention. Catastrophic complications from retropharyngeal abscess include extension of

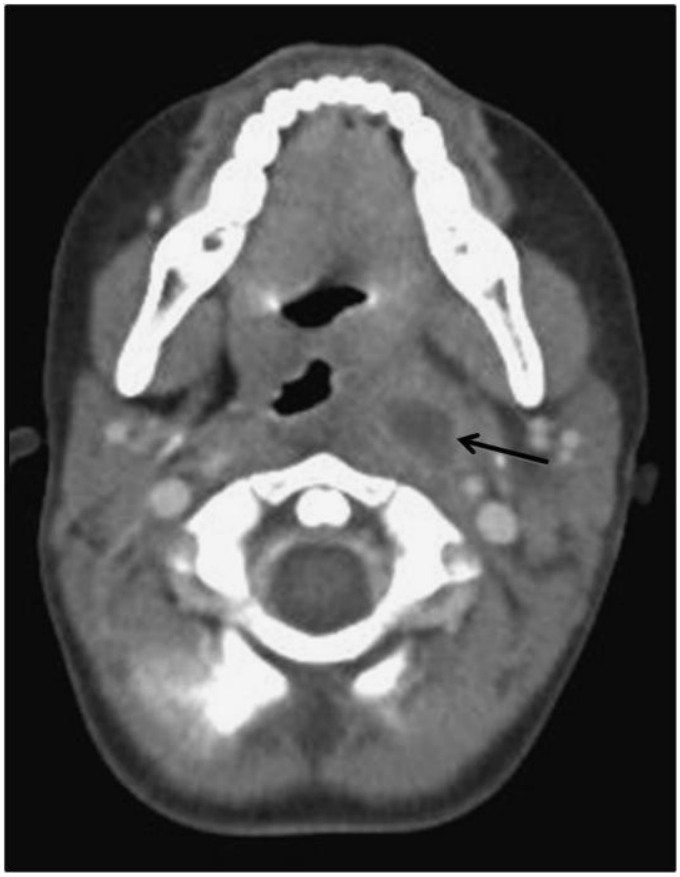

FIGURE 246-3. Contrasted CT of a left retropharyngeal abscess (*arrow*).

the infection into the mediastinum and upper airway asphyxia from direct pressure or aspiration after sudden rupture of the abscess.[25]

ODONTOGENIC ABSCESS

Odontogenic infections can arise from an infected tooth or after a tooth extraction. Development of the infection varies from <1 day to up to 1 to 3 weeks after the onset of tooth pain and may occur despite oral antibiotics. Odontogenic infections are polymicrobial; the most common bacteria are *Streptococci viridians*, *Peptostreptococcus*, *Prevotella*, and staphylococci.[26] **Most deep neck infections originate from an odontogenic source, usually the mandibular teeth.** Dental abscesses may spread into the parapharyngeal and retropharyngeal spaces. Presenting features include neck mass, trismus, fever, leukocytosis, dysphagia, and dyspnea. Potential complications include necrotizing fasciitis, descending necrotizing mediastinitis, orbital infections, and hematogenous dissemination to distant organs.

CLINICAL FEATURES

See chapter 245,"Oral and Dental Emergencies" for management of dental infections isolated to the mandible or maxilla. Soft tissue extension from odontogenic infection ranges from diffuse cellulitis to abscess formation in labial or buccal gingiva. In some cases, intraoral or dentocutaneous fistula formation may occur. Fascial layers of the head and neck produce planes or potential spaces for infectious spread. Infections associated with maxillary teeth tend to spread into potential spaces in the face. Infections of maxillary molars tend to involve the masticator space, which can extend into the parapharyngeal space and downward into the neck and mediastinum. Infections of anterior mandibular teeth tend to spread into the neck. Infections of anterior teeth, bicuspids, and first molars of the mandible tend to enter the sublingual space, with edema of the floor of the mouth with little extraoral swelling. Involvement of the submandibular space is typically the result of second and third mandibular molar infections.

DIAGNOSIS AND TREATMENT

Superficial odontogenic abscesses can be diagnosed with US at the bedside.[27] For diagnosis of suspected deep space infections, contrast-enhanced CT scan is recommended to identify the need for surgical management.[27] Treatment of odontogenic infections includes appropriate antibiotic therapy (aerobic and anaerobic coverage) and surgical drainage of abscesses. Penicillin VK and amoxicillin remain appropriate options for outpatient treatment; amoxicillin-clavulanate, clindamycin, cefuroxime, and levofloxacin are second-line choices.[28,29] Patients with deep-neck infections require IV antibiotics; ampicillin-sulbactam with clindamycin and ciprofloxacin is one recommended regimen. Other useful agents include piperacillin-tazobactam, imipenem-cilastatin, and ertapenem.

COMPLICATIONS

Ludwig's angina is infection of the submental, sublingual, and submandibular spaces. Patients usually present with poor dental hygiene, dysphagia, and odynophagia. Clinical examination reveals trismus and edema of the entire upper neck and floor of mouth. Infection progresses rapidly and can posteriorly displace the tongue, causing airway compromise. Definitive airway management should be considered early in the course, including awake fiberoptic intubation or awake tracheostomy.[30] Stridor, difficulty managing secretions, and cyanosis are late signs and require emergent airway management. Systemic antibiotics are not a substitute for definitive airway management because it may take >1 week for edema resolution with antibiotic therapy.

Patients with **necrotizing infections** are critically ill, with overlying skin discoloration, crepitus of the subcutaneous tissue, and systemic signs, including fever, tachycardia, hypotension, and confusion. CT reveals subcutaneous emphysema, deep tissue gas, and pockets of suppuration (**Figure 246-4**). Aerobic and anaerobic cultures are necessary for identification of causative organisms. Therapy of necrotizing fasciitis is immediate surgery with fasciotomy with wide local debridement and broad-spectrum IV antibiotics. Mediastinal extension places the patient at risk for great vessel erosion, retroperitoneal extension, pleural abscess, pericardial effusion, and sepsis; mortality ranges from 10% to 40%.[31] Tracheostomy should be performed if airway obstruction develops. Surgery can be lifesaving, and immediate surgical consultation is required for this rapidly progressing disease.[31]

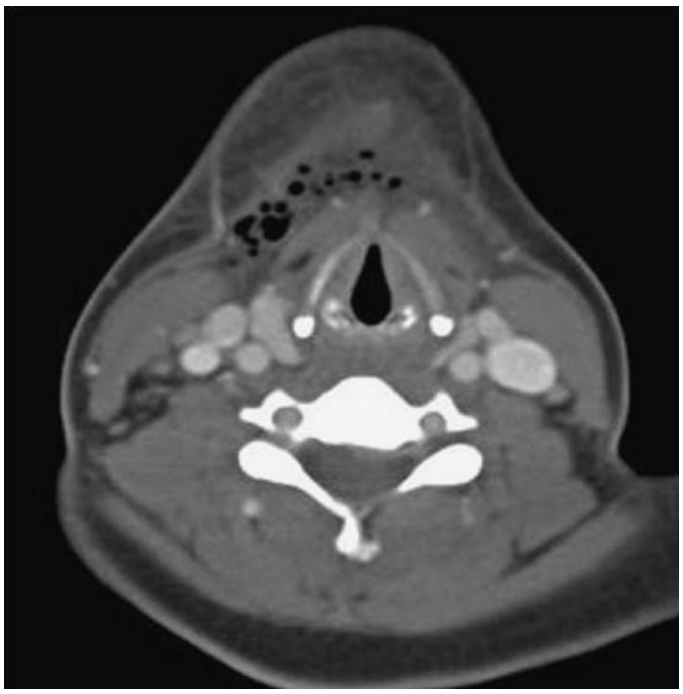

FIGURE 246-4. CT demonstrating necrotizing fasciitis with gas in the deep tissue of the anterior neck.

NECK AND UPPER AIRWAY MASSES

CLINICAL FEATURES

Neck masses (**Figures 246-5 and 246-6**) can result from congenital, infectious, glandular, or neoplastic disorders. Enlargement may lead to airway compromise, dehydration secondary to dysphagia and odynophagia, or secondary infected. Age of the patient and characteristics including location of the mass may aid in the diagnosis (**Tables 246-2 and 246-3**).[32] Neck masses in children are discussed in the chapter 122, "Neck Masses in Infants and Children," in the Pediatrics section. **In adults >40 years old, up to 80% of lateral neck masses persistent for >6 weeks are malignant.[33]**

DIAGNOSIS AND MANAGEMENT

The urgency for evaluation of a neck mass depends on patient acuity.[33] Patients with airway compromise or significant dysphagia and odynophagia should be evaluated by flexible nasopharyngolaryngoscopy **before** CT scan. CT scan will delineate the extent of the mass and likely will be required for surgical intervention. If no airway compromise or dehydration is present, the patient should follow up with primary care for outpatient imaging and further evaluation. The final diagnosis for a neck or upper airway mass will not be made in the ED. All neck masses should have follow-up for diagnosis and treatment.

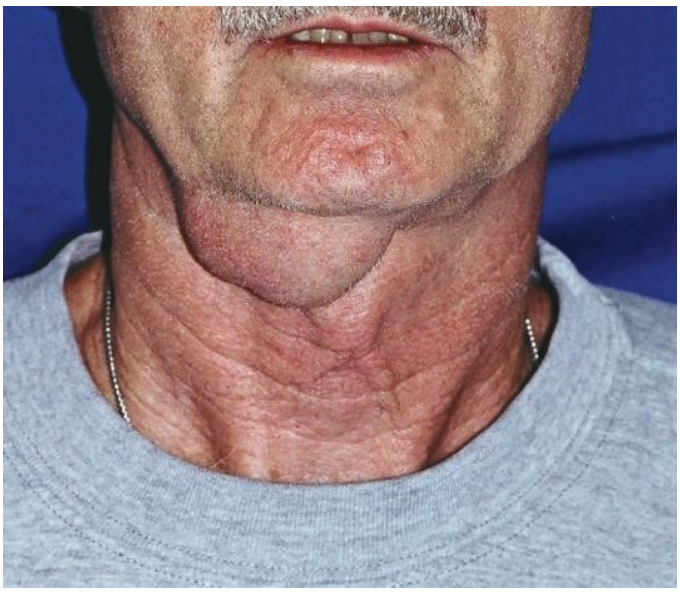

FIGURE 246-5. Right plunging ranula presenting as a painless ballotable submandibular mass.

Empiric antibiotic therapy should be initiated for inflammatory lymph nodes, usually with cephalexin, 250 to 500 milligrams PO three to four times daily; amoxicillin, 250 to 500 milligrams PO three times daily; or clindamycin, 300 milligrams three to four times daily. Resolution is expected in 2 weeks for those lesions that are due to infection alone. Empiric therapy of sialoadenitis should include staphylococcal coverage most commonly, clindamycin[34] (see chapter 118, "Neck Masses in Children").

TABLE 246-2 Neck Masses in Young and Older Adults

Young Adult	Adult
Reactive lymphadenopathy	Metastatic aerodigestive tract carcinoma
Mononucleosis	Salivary gland infection or neoplasm
Lymphoma	
Branchial cleft cyst	Lymphoma
Thyroglossal duct cyst	Thyroid disorder
	Tuberculosis

POSTTONSILLECTOMY BLEEDING

Tonsillectomy is the second most common reason for care in pediatric hospitals in the United States.[35] Postoperative bleeding is a well-known complication of tonsillectomy that can, rarely, lead to death from airway obstruction or hemorrhagic shock. Rate of secondary hemorrhage varies according to the method used for the procedure. The incidence of posttonsillectomy bleeding ranges from 1% to 8.8%, with approximately half requiring surgical intervention for control of bleeding.[36]

Although bleeding can be seen within 24 hours of surgery, most significant hemorrhage occurs between postoperative days 5 and 10. There is a significantly higher incidence of bleeding in patients between 21 and 30 years of age, as well as those over age 70.[36] Posttonsillectomy bleeding can be fatal and requires prompt intervention with control of the airway. An otolaryngologist should be consulted early.

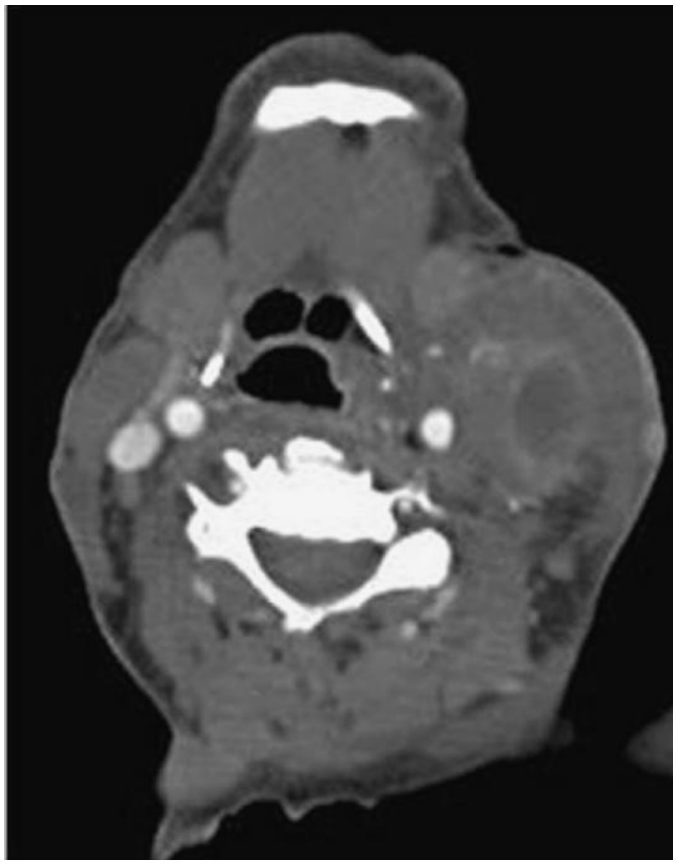

FIGURE 246-6. Hypopharyngeal squamous cell carcinoma metastatic to left cervical lymph nodes. Note the thrombosis of the left jugular vein with displacement of the airway to the right.

TABLE 246-3 Common Causes of Neck Masses in Adults

Disorder	Physical Finding	Pathology	Management
Ranula	Sublingual area swelling	Mucus retention cyst due to ductal obstruction of the sublingual gland	Surgical excision
Laryngeal papillomas	Sessile, warty-appearing lesions on the soft palate or tonsillar pillars	Human papillomavirus type 6 or 11 infection	Surgical excision
Palatine torus	Boney smooth painless mass of the hard palate	Exostoses of the palate	No treatment needed in most cases
Mandibular torus	Boney smooth painless growth of the mandible under the tongue	Exostoses of the mandible	No treatment needed in most cases
Branchial cleft cysts	Painless, fluctuant masses close to the angle of the mandible	Incomplete obliteration of the branchial apparatus during development	Antibiotics if infected, surgical excision
Thyroglossal duct cysts	Soft, mobile, subhyoid bone midline mass	Remnant of the thyroid anlage	Antibiotics if infected, surgical excision
Lymphoma	Multiple, rubbery low-neck masses, night sweats, fever, malaise	Malignant process	Biopsy, referral to ENT and oncology
Acute retroviral syndrome	Generalized adenopathy, unprotected sex by history	Human immunodeficiency virus infection	Antiretroviral medication
Squamous cell carcinoma	Firm, possibly fixed cervical lymph node	Oral lesion metastatic to cervical node	Biopsy, referral to ENT and oncology
Parotid tumors	Nonpainful masses under or anterior to the ear	Benign or malignant process	Biopsy, referral to ENT and oncology as needed
Sialoadenitis	Tender swelling in area of parotid, submandibular, or sublingual salivary gland	Salivary gland infection	Antibiotics, salivary stimulants, also see chapter 122, "Neck Masses in Children"
Thyroid enlargement	Diffuse nodular thyroid enlargement or solitary nodular thyroid	Benign or malignant process	See chapters 228, "Hypothyroidism," and 229, "Hyperthyroidism"

Abbreviation: ENT = otolaryngology.

TREATMENT

Keep the patient NPO (nothing by mouth) and sitting upright, monitor with pulse oximetry, and maintain IV access. Obtain a CBC and coagulation studies, and type and cross-match blood. Examine the oropharynx to see if bleeding can be visualized. A grayish-white eschar is normal following a tonsillectomy. **Apply direct pressure to the bleeding tonsillar bed using a tonsillar pack or a 4×4 gauze on a long clamp, moistened with either thrombin or lidocaine and epinephrine.** To prevent loss of the pack into the airway, place a suture through the pack and tape the suture to the face. Place pressure on the lateral pharyngeal wall, avoiding midline manipulation, to decrease stimulation of the gag reflex. Massive bleeding is rare, but when it occurs, intubation may be the only means of protecting the airway. This is always difficult, with oropharyngeal edema from recent surgery and blood obscuring visualization of the cords. Plans should be made for an emergent cricothyrotomy prior to attempting intubation.

Pressure alone can be adequate for control of posttonsillectomy hemorrhage until the otolaryngologist arrives. Alternatively, if a bleeding site can be visualized, bleeding may be cauterized with silver nitrate after local infiltration with 1% lidocaine with epinephrine. Otolaryngologic consultation in the ED is always needed because patients may have a second or even third posttonsillectomy hemorrhage,[37] and surgery or endovascular embolization may be necessary for definitive control.[38]

REFERENCES

The complete reference list is available online at www.TintinalliEM.com.

CHAPTER 247

Complications of Airway Devices

John P. Gaillard

TRACHEOSTOMY TUBES AND CANNULAS

A tracheostomy is an opening between cartilaginous rings in the trachea and the skin, with a tracheostomy tube placed into the stoma to facilitate ventilation. Tracheostomy is usually performed by an otolaryngologist as an elective or semi-elective procedure and is not an emergency procedure. Most tracheostomies are performed on chronically ill patients requiring prolonged mechanical ventilation.

There are many types of tracheostomy tubes available, including those made of plastic, silicone, nylon, and metal. Most hospitals stock only a few types of tracheostomy tubes, and one must be familiar with the types available. Tracheostomy tubes vary in diameter, total length, the length before and after the curve, and the presence or absence of a cuff (**Figure 247-1**). The size of the tracheostomy tube is usually defined by the inner diameter, ranging in adults from 5 to 10 mm and in pediatric patients from 2.5 to 6.5 mm. Most pediatric and adult tracheostomy tubes have a 15-mm standard respiratory connection that may be used with ventilator tubing or a bag-valve device.

Fenestrated tracheostomy tubes have an opening along the dorsal surface of the body of the tube. The fenestration allows the passage of air through the tracheostomy tube to the vocal cords so the patient can speak. Irritation from the fenestration may promote growth of granulation tissue, which may extend into the fenestration, leading to bleeding, obstruction, and difficulty removing the tracheostomy tube. If any difficulty is encountered removing a fenestrated tracheostomy tube, obtain surgical or ear, nose, and throat consultation.

Most adult tracheostomy tubes have a removable inner cannula, which allows secretions to be cleared from the lumen without removing the entire tube from the trachea. In assessing an adult tracheostomy patient, remove and examine the inner cannula for crusting or obstruction. Both disposable and reusable inner cannulas may be cleaned by using a small brush dipped in a solution of hydrogen peroxide and then