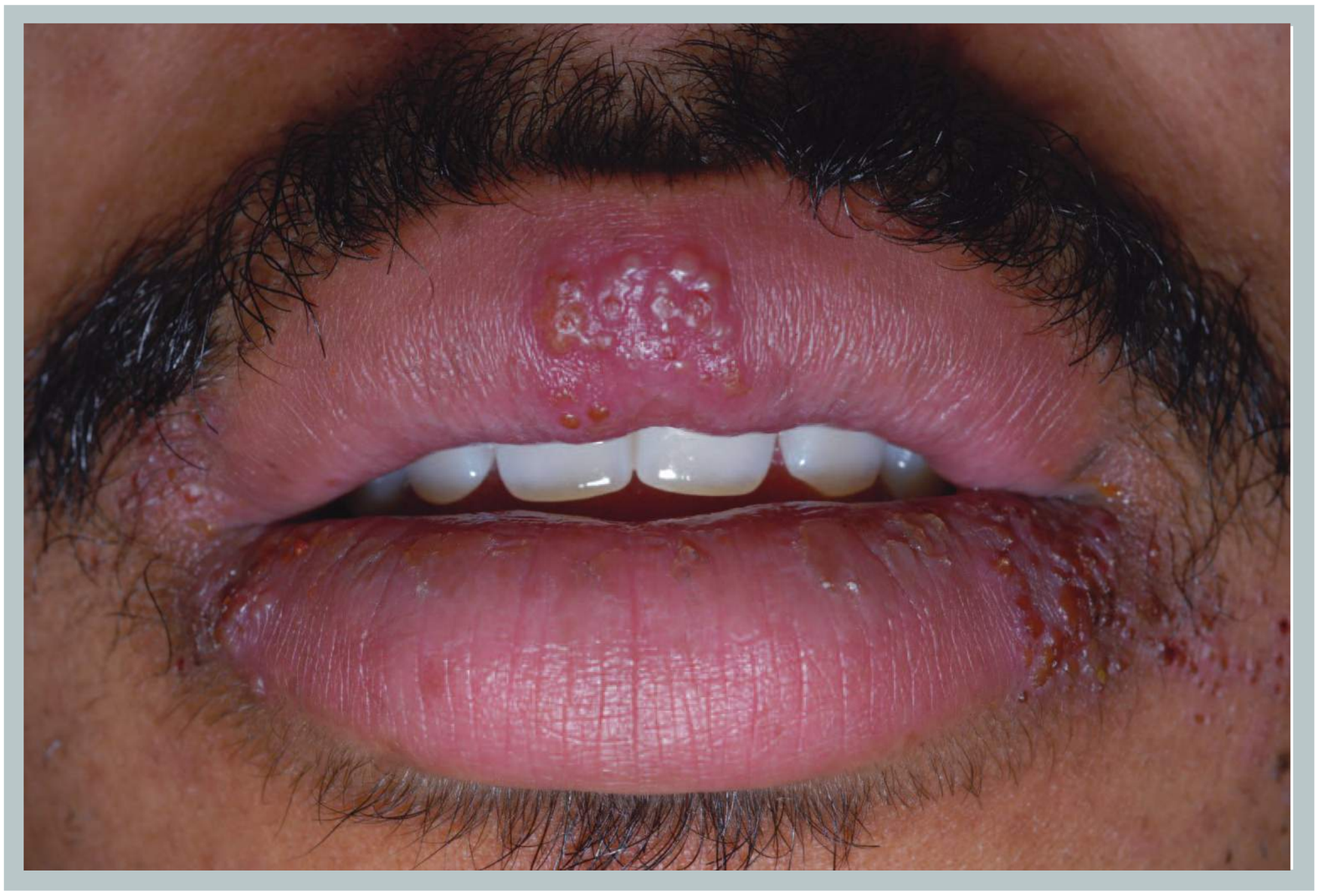
Chapter 6

ORAL CONDITIONS

Edward C. Jauch
James W. Howell

Scan QR code or go to www.ematlas.com for video related to chapter



Herpes Labialis. Note the extensive painful ulcerations on the patient's upper lip and corners of the mouth. A prodromal period of fever, malaise, and cervical adenopathy may herald the onset of these painful ulcerations. (Photo contributor: R. Jason Thurman, MD.)

The authors acknowledge Brent E. Gottesman, MD, Edwin D. Turner, MD, and Sara-Jo Gahm, MD, for portions of this chapter written for the previous editions of this book.

TOOTH SUBLUXATION

Clinical Summary

Tooth subluxation, the loosening of a tooth in its alveolar socket, is most commonly secondary to trauma; however, infection and periodontal disease may also produce subluxation. Gingival lacerations and alveolar fractures are associated with dental subluxations. Gentle pressure to the teeth with a tongue blade or fingertip may produce movement, mild displacement, or blood along the crevice of the gingiva, all signs of subluxation. Dental impaction and alveolar ridge fracture should be considered and ruled out clinically or radiographically.

Management and Disposition

1. Primary teeth: If the subluxated tooth is forced into close proximity to the underlying permanent tooth, follow-up for extraction is indicated. Otherwise, the patient should be instructed to follow a soft diet for 1 to 2 weeks, allowing the tooth to reimplant spontaneously.
2. Permanent teeth: Unstable teeth should be temporarily immobilized using gauze packing, a figure-eight suture around the tooth and an adjacent tooth, aluminum foil, or a special periodontal dressing, and the patient referred for dental follow-up.

Pearls

1. Any evidence of tooth mobility following trauma is a subluxation by definition.
2. Always consider an associated underlying alveolar or occult root fracture.

FIGURE 6.1 ■ **Tooth Subluxation.** Note the presence of blood along the crevice of the gingival margin of both central incisors—an indication of subluxation following trauma. Mild displacement of the subluxated teeth is noted. (Photo contributor: James F. Steiner, DDS.)

FIGURE 6.2 ■ **Tooth Subluxation.** Tooth subluxations can be quite subtle, as in this case of a slightly subluxed tooth #9 missed on initial examination. A careful dental examination is essential in patients with oral trauma. (Photo contributor: Kevin J. Knoop, MD, MS.)

TOOTH IMPACTION (INTRUSIVE LUXATION)

Clinical Summary

Impacted or intruded teeth result when a tooth is forced deeper into the alveolar socket or surrounding tissues as a result of trauma. The tooth appears shorter than its contralateral partner. An impacted tooth may be partially visible or completely hidden by the gingiva and buried in the alveolar process. Completely impacted teeth may erroneously be considered avulsed until a radiograph demonstrates the intruded position. The apex of a completely impacted permanent central incisor may be driven through the alveolar bone into the floor of the nares, causing epistaxis. Associated injuries may include alveolar fractures, dental crown or root fractures, and oral mucosal or gingival lacerations. Pulp necrosis occurs in 15% to 50% of cases.

Management and Disposition

Impacted primary teeth usually re-erupt and reposition spontaneously within 1 to 3 months. Any intruded primary tooth whose apex is displaced toward or impacts on the follicle of its permanent successor requires dental follow-up for extraction and monitoring clinically and radiographically for 1 year. Permanent teeth do not re-erupt. Surgical reduction is indicated to prevent complications such as external root resorption and loss of supporting bone. Orthodontic repositioning and splinting is generally carried out over 3 to 4 weeks.

Pearls

1. An undiagnosed impacted tooth is predisposed to infection and may have a poor cosmetic result.
2. The maxillary incisors are the most commonly impacted teeth.

FIGURE 6.3 ■ **Tooth Intrusion.** This impaction injury with multiple anterior maxillary tooth involvement shows various degrees of tooth impaction. Also note the complete absence of a central incisor. This may indicate a complete intrusion into the alveolar socket or an avulsion of the tooth. Radiographic studies are required when a tooth's location is in question. (Photo contributor: James F. Steiner, DDS.)

TOOTH AVULSION

Clinical Summary

Avulsion is the total displacement of a tooth from its socket. There is usually a history of trauma; however, infectious etiologies may result in complete disruption of the periodontal ligament from the affected tooth. Various degrees of bleeding from the socket and surrounding gingiva may be noted and there may be underlying alveolar fracture depending on the mechanism of injury. Prompt inquiry into the location of any unaccountable tooth is indicated. Radiographic evaluation to rule out aspiration, soft tissue entrapment, impaction, or dentoalveolar fracture is required when teeth are missing.

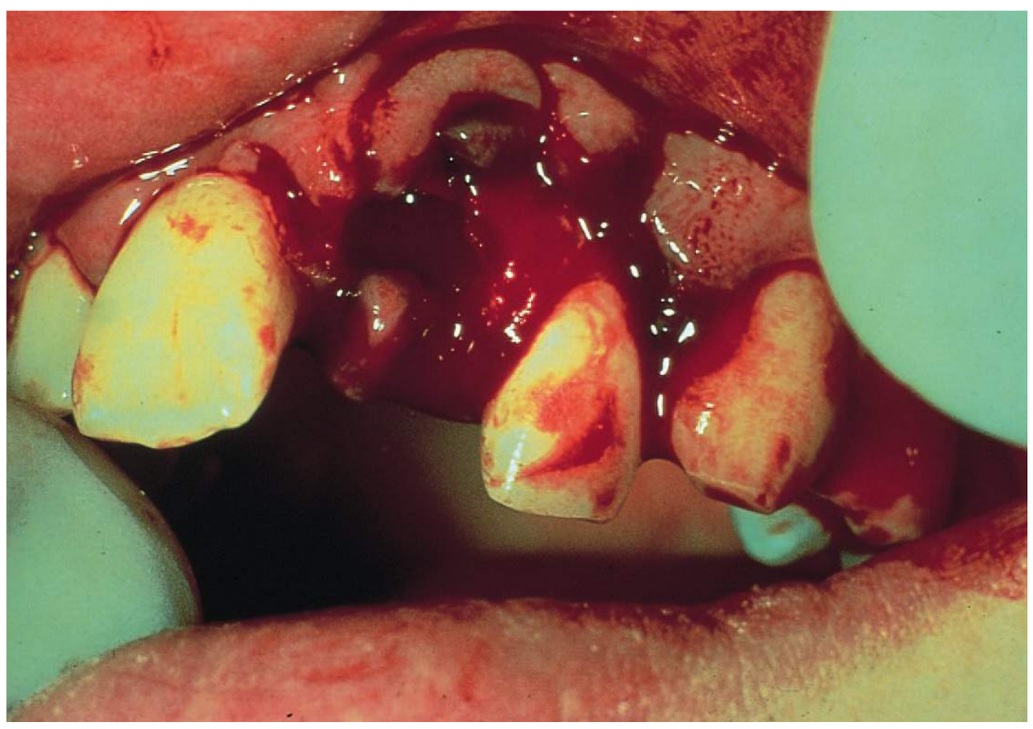


FIGURE 6.4 ■ **Tooth Avulsion.** Avulsion injury with angulation and displacement of teeth from the alveolar socket. (Photo contributor: James F. Steiner, DDS.)

Management and Disposition

Successful reimplantation decreases by 1% for every minute the tooth is out of its socket. Permanent teeth should be replaced in their sockets as soon as possible. Successful reimplantation depends on the survival of periodontal ligament fibers; the tooth should be rinsed with saline, but not scrubbed, with care not to handle the root while replacing it in the socket. Emergent dental consultation, tetanus prophylaxis, and antibiotics targeting mouth flora are indicated. If not replaced, the avulsed tooth should be stored in the mouth of the patient or parent, or in a container of milk. Normal saline and commercial preservatives are reasonable alternatives, but tap water should not be used. Primary teeth are not reimplanted; reimplanted teeth may interfere with eruptions of permanent teeth because of ankylosis and fusion to the bone. Follow-up should be obtained for possible orthodontia until the permanent tooth erupts.

FIGURE 6.5 ■ **Tooth Avulsion.** Significant avulsion injury of tooth #8 in a patient with direct oral trauma. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Primary teeth should not be reimplanted.
2. Successful reimplantation occurs best within the first 30 minutes.
3. Storage and transport media in decreasing order for preserving tooth viability include balanced salt solution or a tissue culture medium, chilled low-fat milk, saline, and saliva.

FIGURE 6.6 ■ **Tooth Avulsion Reimplanted.** Reimplantation of the avulsed tooth in Fig. 6.5 was performed by an oral surgeon with the use of an arch bar to secure the tooth. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Anatomically, each tooth has a crown and root portion. Externally, the crown is covered with white enamel and the root portion with cementum. The cementoenamel junction (cervical line) is where the crown and root meet. The yellow-to-tan dentin is the second innermost layer and comprises the bulk of the tooth. The red-to-pink pulp tissue is located in the center of the tooth and includes the tooth's neurovascular supply. The Ellis classification system is commonly used to describe tooth fractures above the cervical line in anterior teeth:

Ellis class I: Involves only the enamel.

Ellis class II: Involves the enamel plus exposure of the dentin. The patient may complain of temperature sensitivity.

Ellis class III: Fracture extends into the pulp. A pink or bloody discoloration on the fracture surface is diagnostic. The patient may have severe pain but may also have no pain due to loss of tooth nerve function.

Tooth fractures of the dental root may also occur below the cementoenamel junction and are commonly missed on initial evaluation. Bleeding may be observed at the gingival crevice with associated tooth tenderness on percussion. Radiographic evaluation may aid in differentiating these conditions.

Management and Disposition

Ellis class I: Pain control and referral for rough tooth edge and cosmetic management are indicated.

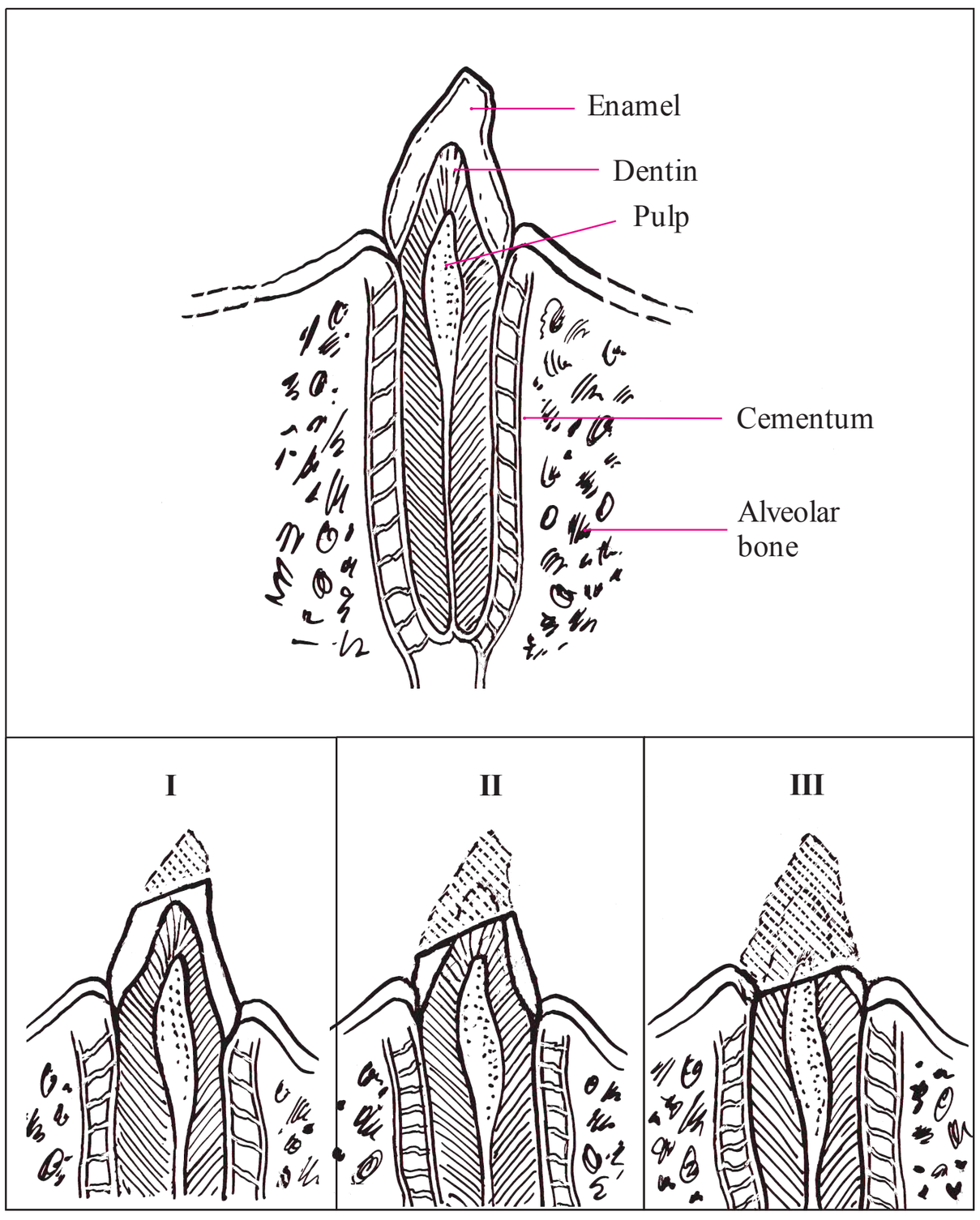


FIGURE 6.7 ■ **Tooth Fractures.** Enamel, dentin, and pulp are the anatomic landmarks used in the Ellis classification of tooth fractures.

Ellis class II: Patients under 12 years of age have less dentin than older patients and are at risk for pulp infection. They should have a calcium hydroxide dressing placed, covered with gauze or aluminum foil, and seen by a dentist within 24 hours. Older patients should see a dentist within 24 to 48 hours.

Ellis class III: This is considered a dental emergency, and dental consultation within 24 to 48 hours is indicated. Delay in treatment may result in abscess formation.

Root fractures: Early reduction, immobilization/splinting, and dental referral within 24 to 48 hours are indicated. Most teeth sustaining root fractures maintain pulp viability.

Pearls

1. Check for tooth mobility on initial examination to aid in differentiating mobility involving the entire tooth from involvement of only the fractured segment.

FIGURE 6.8 ■ **Ellis Class I Tooth Fracture.** Note the fracture of the left upper central incisor. The sole involvement of the enamel is consistent with an Ellis class I fracture. (Photo contributor: James F. Steiner, DDS.)

FIGURE 6.9 ■ **Ellis Class II Tooth Fractures.** Bilateral maxillary central incisor injuries with exposed enamel and dentin consistent with an Ellis class II fracture. (Photo contributor: James F. Steiner, DDS.)

FIGURE 6.10 ■ **Ellis Class III Tooth Fracture.** A fracture demonstrating blood at the exposed dental pulp. This sign is pathognomonic for an Ellis class III fracture. (Photo contributor: Kevin J. Knoop, MD, MS.)

FIGURE 6.11 ■ **Multiple Ellis Fractures.** Multiple Ellis fractures resulting from blunt trauma: Ellis I fractures are seen in teeth 7 and 11, Ellis II in teeth 8 and 10, and Ellis III in tooth 9. (Photo contributor: Rosie Korman, MD.)

Clinical Summary

The alveolus is the tooth-bearing segment of the mandible and maxilla. Fracture of the alveolar process tends to occur most often in the thinner maxilla. The anterior alveolar processes are at greatest risk for fracture due to more direct exposure to trauma. Both subluxation and avulsion of teeth may be associated with underlying alveolar fractures. Various degrees of tooth mobility and gingival bleeding may occur.

Management and Disposition

Significant cosmetic deformity may result from alveolar bone loss; preservation of viable tissue is important. Gentle direct pressure over the alveolar segment with saline-soaked gauze, avulsed teeth preservation, and tetanus and antibiotic therapy administration are indicated. Oral surgery consultation should be obtained for possible wire stabilization or arch bar fixation.

Pearls

1. Always consider the possibility of an associated cervical spine injury when evaluating patients with facial trauma.
2. Consider aspiration of avulsed teeth.

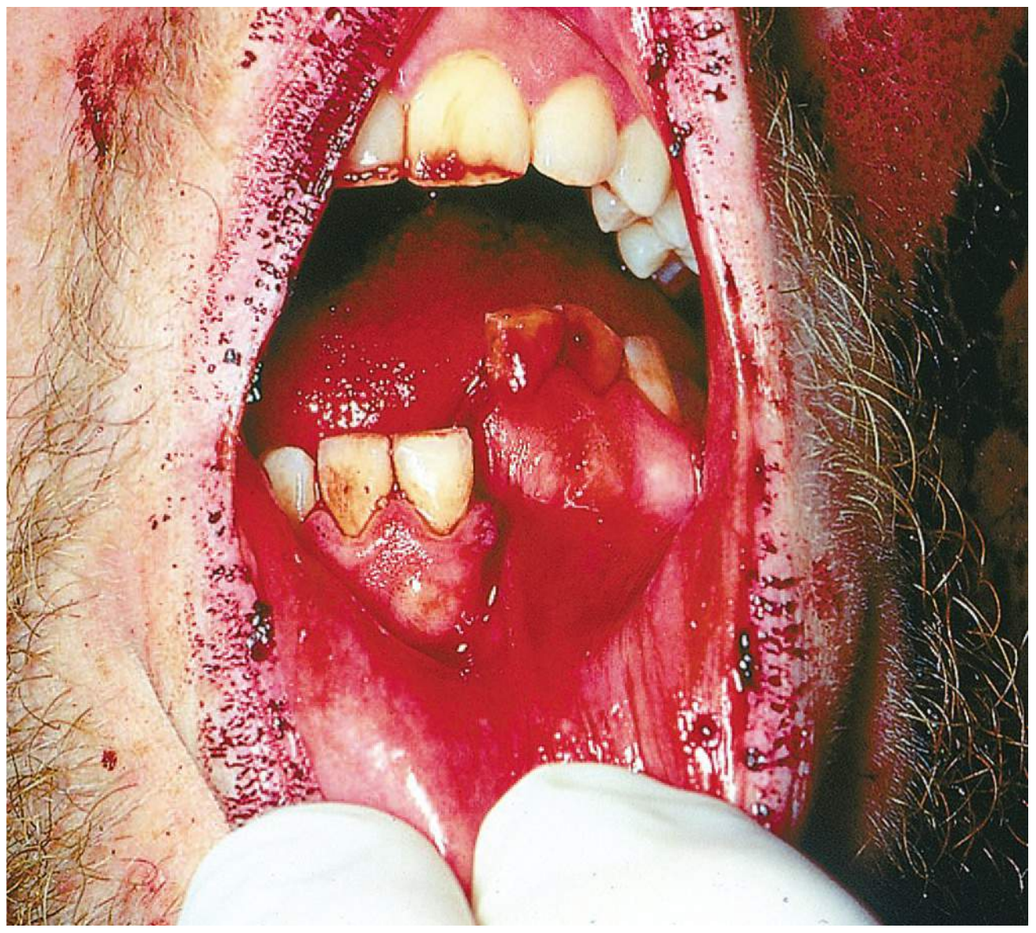


FIGURE 6.12 ■ **Alveolar Ridge Fracture.** Note the exposed alveolar bone segment and associated multiple teeth involvement. Attempts should be made to maximally preserve all viable tissue. (Photo contributor: Alan B. Storrow, MD.)

FIGURE 6.13 ■ **Alveolar Ridge Fracture.** This alveolar ridge fracture was caused by blunt trauma from a steering wheel in a frontal impact motor vehicle collision. (Photo contributor: R. Jason Thurman, MD.)

TEMPORAL MANDIBULAR JOINT DISLOCATION

Clinical Summary

Temporal mandibular joint (TMJ) dislocation generally occurs in predisposed individuals after a vigorous yawn or seizure, or less commonly from direct trauma to the chin while the mouth is open. Dislocation occurs when the mandibular condyles displace forward and become locked anterior to the articular eminence. Masseter muscle spasm contributes to prevention of spontaneous relocation. Weakness of the temporomandibular ligament, an overstretched joint capsule, and a shallow articular eminence are predisposing factors. Patients usually present with an inability to close an open mouth. Other associated symptoms include pain, discomfort, facial swelling near the temporomandibular joint, and difficulty speaking and swallowing. Anterior dislocations are most common; however, posterior dislocation may occur with significant trauma, often in association with basilar skull fractures. Unilateral dislocation results in deviation of the mandible to the unaffected side. TMJ hemarthrosis and dystonic reactions may mimic TMJ dislocations. Mandibular fractures should be considered if there is a history of facial trauma.

FIGURE 6.15 ■ **Bilateral TMJ Dislocation Panorex.** This Panorex image demonstrates bilateral anterior displacement of the mandibular condyles. (Photo contributor: Jake Block, MD.)

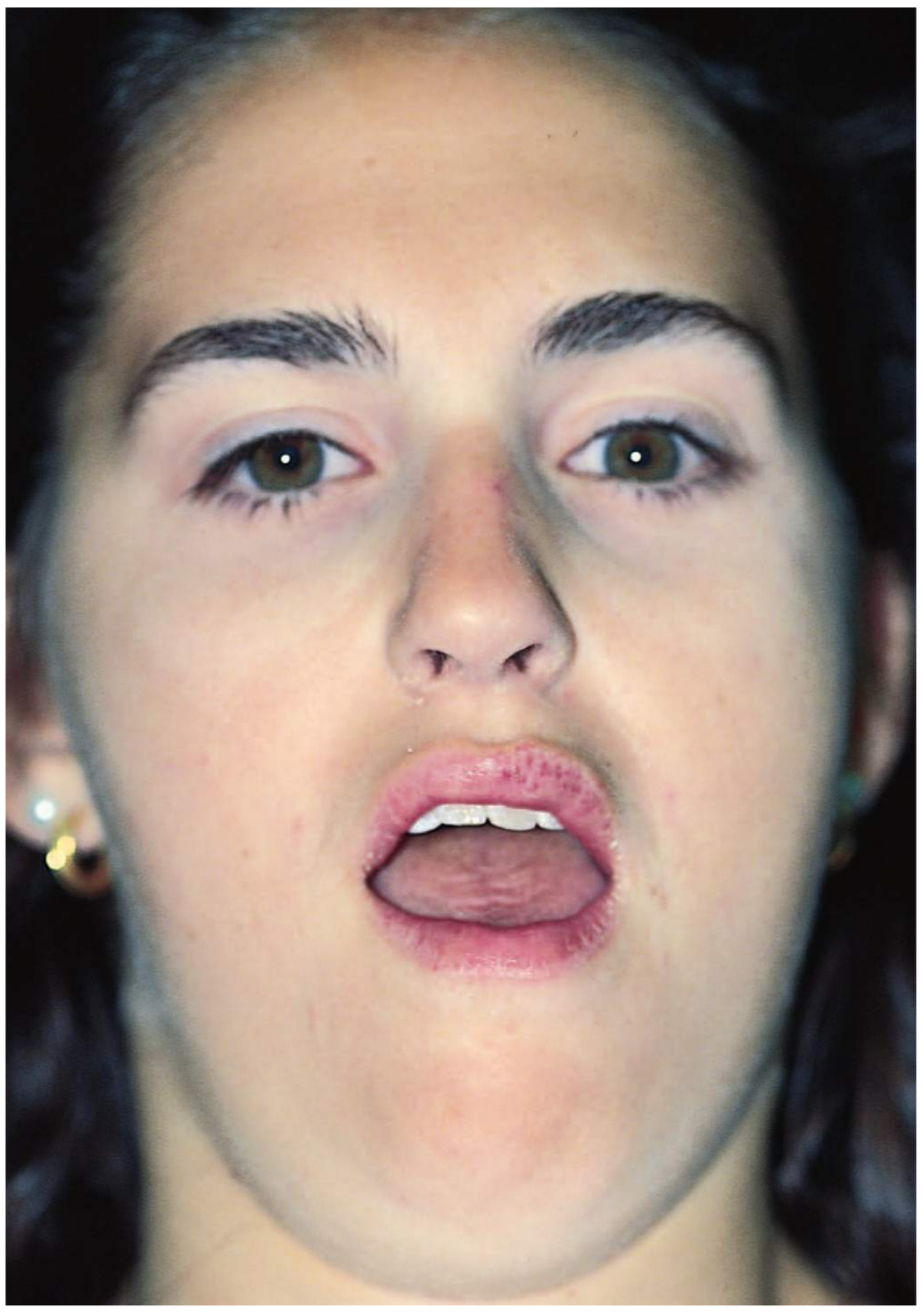


FIGURE 6.14 ■ **TMJ Dislocation (Bilateral).** This patient awoke from sleep with the inability to close her mouth. Note the dry lips and tongue secondary to prolonged exposure. (Photo contributor: Warren K. Russell, MD.)

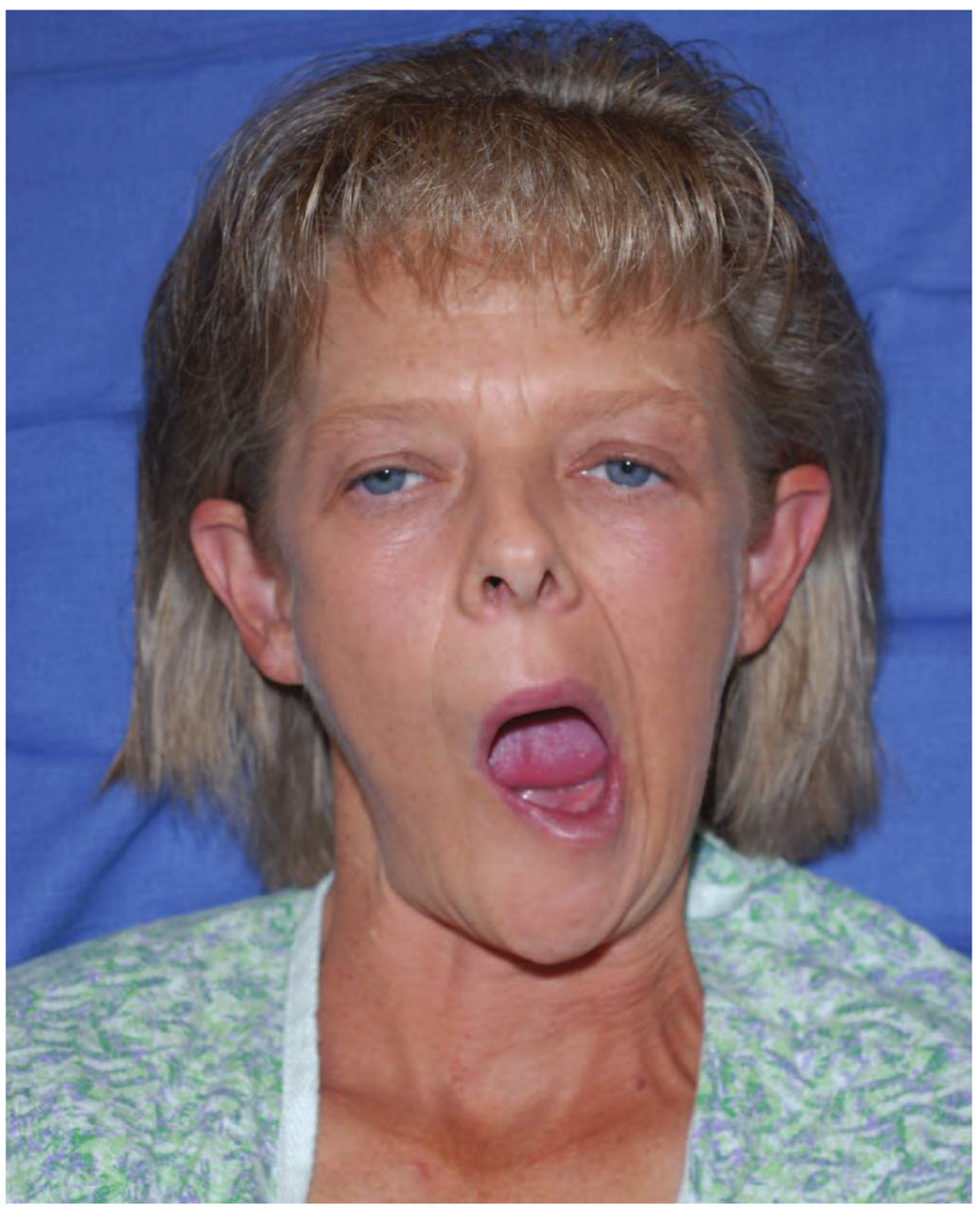


FIGURE 6.16 ■ **TMJ Dislocation, Edentulous Patient (Unilateral).** The mandible is deviated toward the unaffected side. (Photo contributor: R. Jason Thurman, MD.)

Management and Disposition

Acute reduction of pain, muscle spasm, and anxiety is achieved using reassurance, analgesics, and benzodiazepine muscle relaxants. Panorex or TMJ x-ray films (prereduction and postreduction) should be considered to exclude a fracture. A reduction maneuver is performed while facing the sitting patient and grasping the angles of the mandible with both hands. The thumbs are wrapped in gauze for protection and rest on the occlusive surfaces of the molars while downward and backward pressure is steadily applied until the condyle slides back into the articular eminence. Reduction may require some time and force to overcome muscle spasm. Following reduction, instruct the patient to avoid excessively wide mouth opening while eating and yawning for 3 to 4 weeks. Warm compresses to the TMJ, a soft diet for 1 week, and the use of nonsteroidal anti-inflammatory drugs (NSAIDs) are advised. Dental/oral surgery follow-up should be arranged.

FIGURE 6.17 ■ **TMJ Reduction Technique.** Under sedation, the patient in Fig. 6.16 undergoes reduction with inferior-posterior force applied by the physician. The thumbs are not wrapped with gauze in this case as the patient is edentulous. (Photo contributor: R. Jason Thurman, MD.)

Pearls

1. TMJ dysfunction secondary to a neuroleptic or antipsychotic medication–related dystonic reaction is treated with diphenhydramine or benztropine.
2. When trauma is the cause of TMJ dislocation, maintain a high index of suspicion for mandible fractures and cervical spine injuries.
3. A reportedly successful technique for reduction of TMJ dislocation without sedation involves placing a syringe between the posterior molars and having the patient roll the syringe back and forth while gently biting down until reduction is achieved.

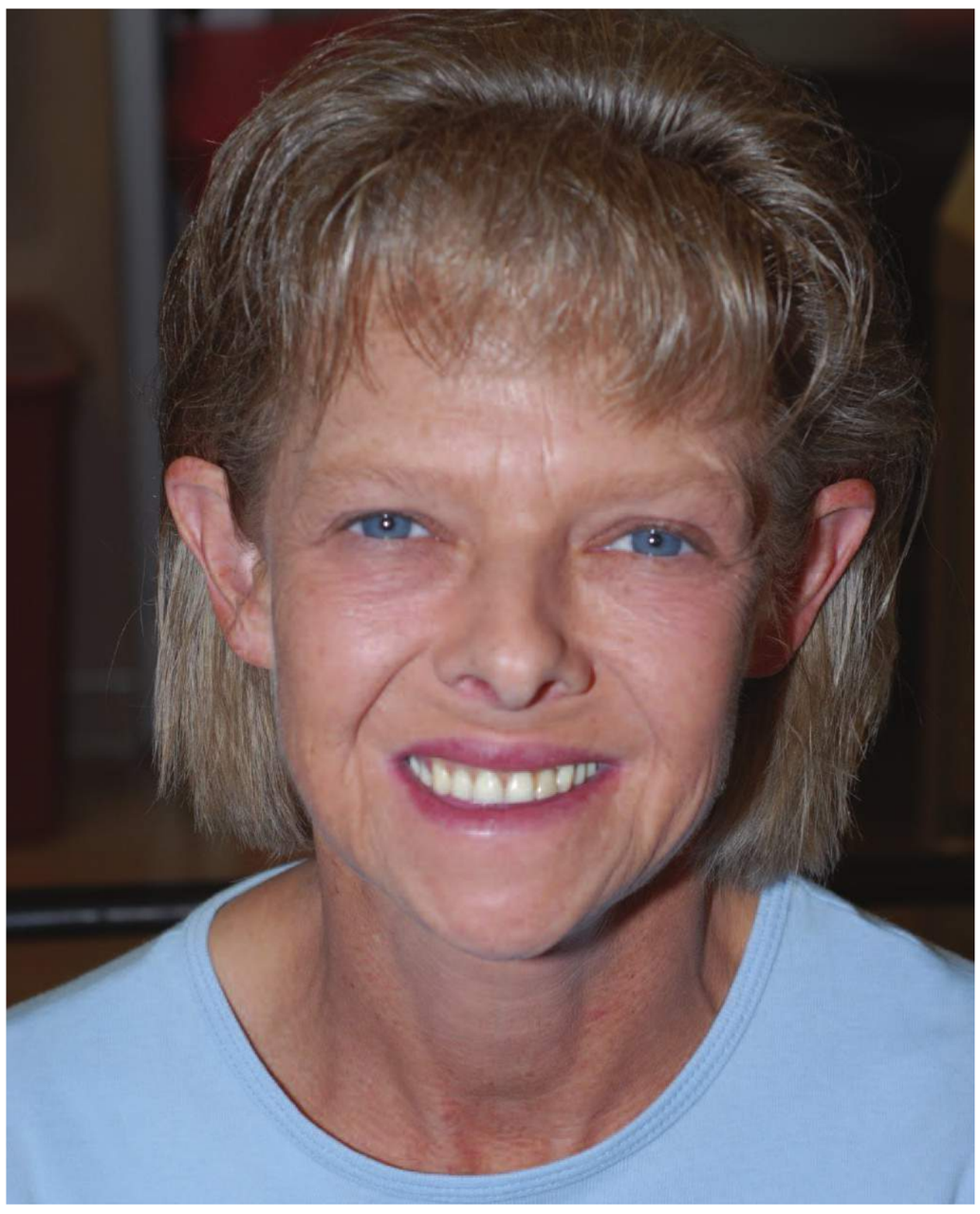


FIGURE 6.18 ■ **TMJ Reduced.** Improved anatomic alignment and a happy patient after reduction of her unilateral TMJ dislocation is accomplished. (Photo contributor: R. Jason Thurman, MD.)

TONGUE LACERATION

Clinical Summary

Injuries to the tongue or mouth floor can cause serious hemorrhage and potential airway compromise and injury to or absence of teeth should be ascertained by inspecting the wound for possibly entrapped dental elements. Dorsal surface tongue lacerations may be associated with a mandibular surface laceration.

Management and Disposition

Most tongue lacerations do not require repair. Lacerations involving the tip or lateral margins or lacerations greater than 1 cm in length that gape widely or actively bleed are best stabilized by a few rapidly absorbable sutures using large bites to include both mucosa and muscle. Anesthesia of the anterior two-thirds of the tongue is obtained by an inferior alveolar/lingual nerve block. Local anesthesia, infiltrated at the site of the wound, may also be used.

Pearls

1. Extensive complex tongue lacerations are at risk for infection and should be prophylactically treated with antibiotics covering oropharyngeal flora.
2. Regional anesthesia for tongue laceration repair avoids distortion of the anatomy prior to repair and is generally better tolerated than direct infiltration of local anesthesia into the tongue.

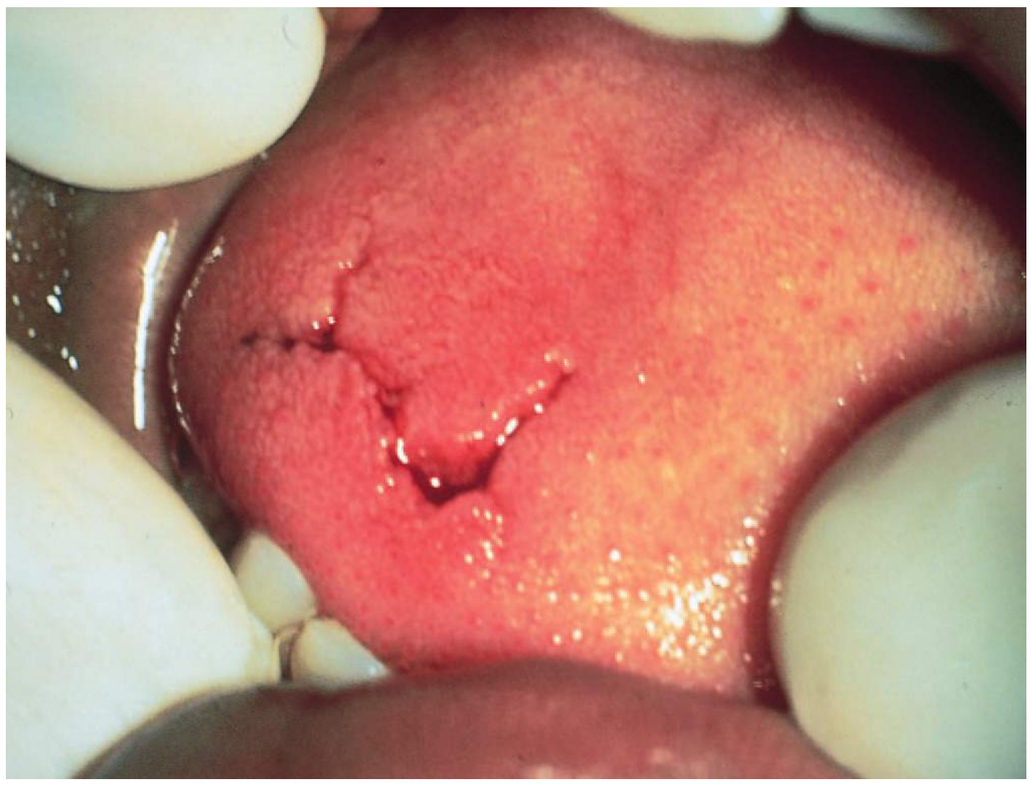


FIGURE 6.20 ■ **Tongue Laceration.** A stellate tongue laceration that does not require suturing is shown. The ventral aspect of the tongue should be examined for additional lacerations sustained from the mandibular teeth. (Photo contributor: James F. Steiner, DDS.)

FIGURE 6.19 ■ **Tongue Laceration.** Due to its length and gaping, this tongue laceration was repaired with absorbable sutures. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 6.21 ■ **Healing Tongue Edge Laceration.** Lacerations of the tip or edge of the tongue should be reapproximated as chronic defects or "forked tongue" may result. (Photo contributor: Lawrence B. Stack, MD.)

VERMILION BORDER LIP LACERATION

Clinical Summary

Anatomically, the vermilion border of the lips represents the transition area from mucosal tissue to skin. Lip lacerations involving the vermilion border present a unique clinical situation, since relatively minor malalignment may produce an unacceptable cosmetic result. An associated underlying gingival or dental injury is a common finding.

Management and Disposition

Accurate vermilion margin reapproximation is the first goal of lip repairs. An unapproximated vermilion margin of 2 mm or greater results in a cosmetic deformity. A regional block of the mental or infraorbital nerve is recommended for anesthesia to avoid additional tissue edema and anatomic distortion produced by local infiltration. Deep or through-and-through lacerations involving the vermilion border should be closed in layers. After closure of the deeper tissue, the first skin suture is always placed at the vermilion border to reestablish the anatomic margin. Using 5-0 or 6-0 nylon, sutures should be placed along the vermilion surface until the moist mucous membrane is encountered. The deep muscular and dermal layer may be closed with 4-0 chromic or Vicryl sutures. Mucosal layers are loosely reapproximated with absorbable suture. The patient should be given wound care instructions and follow-up for wound evaluation and possible suture removal within 3 to 7 days.

Pearls

1. A vermilion border with as little as 2 mm of malalignment may produce a cosmetic defect.
2. Always carefully place the first skin suture at the vermilion border in any lip laceration.

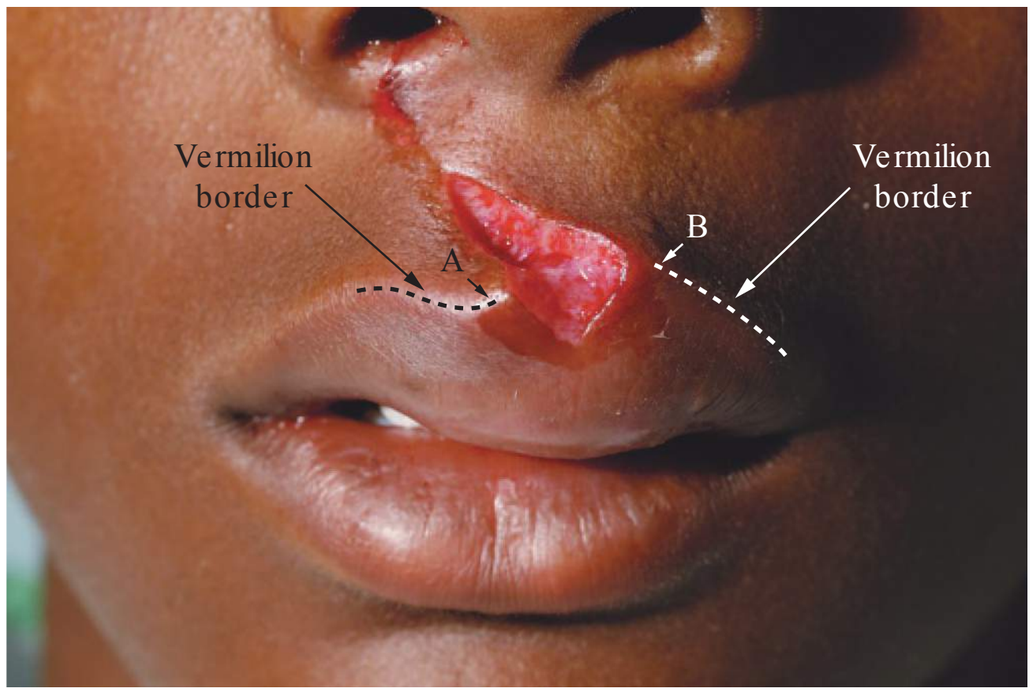


FIGURE 6.22 ■ **Vermilion Border Lip Laceration.** A lip laceration with disruption of the vermilion border. Wound repair begins at the vermilion-skin junction (precise approximation of A to B in this case) for a good cosmetic result. (Photo contributor: Kevin J. Knoop, MD, MS.)

FIGURE 6.23 ■ **Complex Vermilion Border Lip Lacerations.** This patient suffered vermilion border lacerations in two areas from an assault with a broken bottle. Following shaving of the moustache, each vermilion-skin junction was repaired before remaining superficial wound closure was achieved. (Photo contributor: R. Jason Thurman, MD.)

GINGIVAL ABSCESS (PERIODONTAL ABSCESS)

Clinical Summary

Gingival abscesses tend to involve the marginal gingiva and result from entrapment of food and plaque debris and subsequent staphylococcal, streptococcal, anaerobic, or mixed bacterial overgrowth. Localized swelling, erythema, tenderness, and fluctuance in the space between the tooth and gingiva ensue. There may be spontaneous purulent drainage from the gingival margin, or an area of abscess pointing. When the gingival abscess involves the deeper supporting periodontal structures, referred to as a periodontal abscess, the patient may present with a fluctuant vestibular abscess or with a draining sinus that opens onto the gingival surface.

In contrast, periapical abscesses are deep and not obvious on inspection. They usually present as tenderness to percussion or pain with chewing over the involved tooth. A parulis may also simulate a gingival abscess; however, a parulis represents the cutaneous manifestation of a deeper periapical abscess. Unlike a parulis or periapical abscess, gingival abscesses are not usually associated with dental caries or fillings. Pericoronal abscesses tend to involve the gingiva overlying a partially erupted third molar.

Management and Disposition

The initial management is a small incision with drainage and warm saline irrigation. Oral antibiotic therapy, analgesics, and dental follow-up are indicated. The patient's tetanus status should be addressed.

Pearls

1. Patients with gingival abscesses are usually afebrile. Consider more extensive abscess formation and oral disease processes in the febrile toxic-appearing patient.
2. Patients with chronic, deep periodontal abscesses complain of dull, gnawing pain as well as a desire to bite down on and grind the tooth.

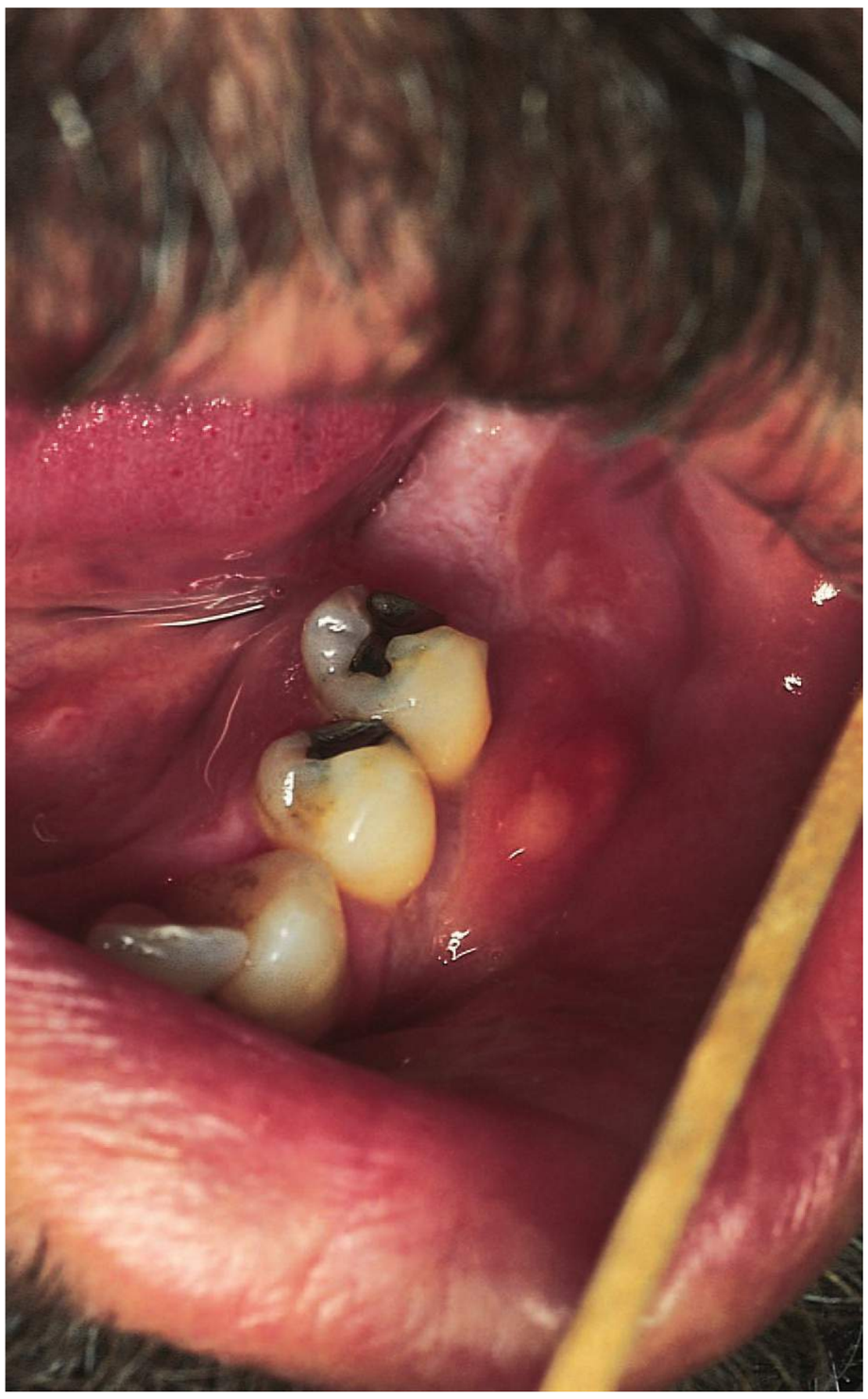


FIGURE 6.24 ■ **Periodontal Abscess.** Localized gingival swelling, erythema, and fluctuance are seen in this periodontal abscess with spontaneous purulent drainage. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Acute pain, swelling, and mild tooth elevation are characteristics of a periapical or dentoalveolar abscess. Exquisite sensitivity to percussion or chewing on the involved tooth is a common sign. The involved tooth may have had dental caries, a filling, or a root canal. Periapical abscesses can enlarge over time and "point," either internally on the lingual or buccal mucosal surfaces, or extraorally with swelling and redness of the overlying skin. Occasionally, these lesions may track up to the alveolar periosteum and gingival surface to form a parulis. Radiographically, these abscesses appear as well-circumscribed areas of radiolucency at the dental apex or along the lateral aspect of the root. Early acute periapical abscesses may not demonstrate any radiographic changes. Both deep periodontal and periapical abscesses may have sinuses draining purulent material onto the gingival surface. If the infection is allowed to progress, it can erode through cortical bone, manifesting itself in a variety of locations. Panorex films, dental radiographs, or a CT scan may aid in the diagnosis.

FIGURE 6.25 ■ **Periapical Abscess.** This periapical abscess points externally to the overlying skin. (Photo contributor: Robin Cotton, MD.)

Management and Disposition

NSAIDs or oral narcotics for pain, tetanus prophylaxis, and antibiotic therapy are indicated. A regional nerve block provides immediate temporary relief. Dental follow-up in 1 to 2 days is recommended for endodontic evaluation or possible extraction of the involved tooth. Incision and drainage along with saline irrigation and prompt referral are indicated for parulis management.

FIGURE 6.26 ■ **"Gumboil" (Parulis).** This lesion is a sinus tract extension of a periapical abscess. It is differentiated from a periodontal abscess by tenderness to tooth percussion. (Photo contributor: Alan B. Storrow, MD.)

Pearls

1. More than one tooth may be involved.
2. Exquisite tenderness and pain on tooth percussion are key features on physical examination and identify the involved tooth.

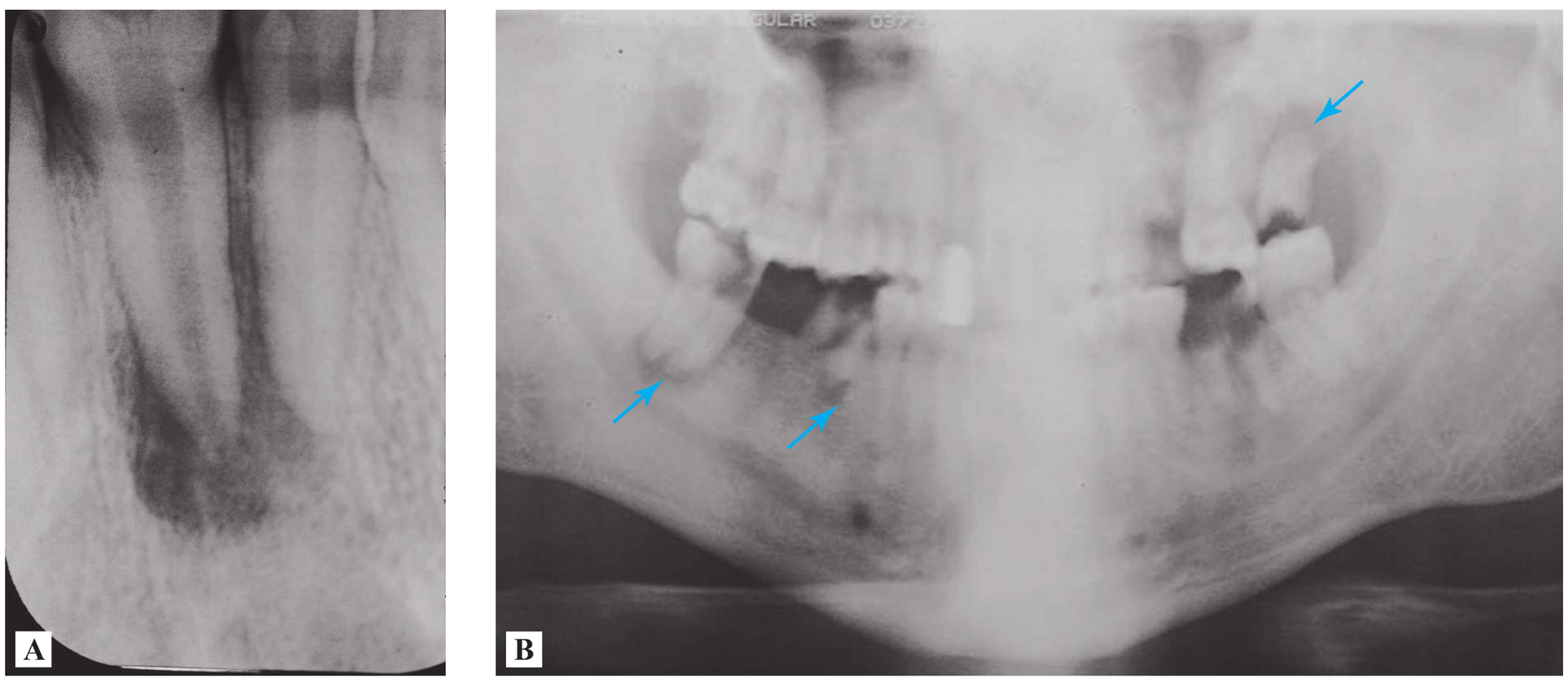


FIGURE 6.27 ■ **Periapical Abscess.** (**A**) Note the well-defined radiolucent area at the apex and lateral root of the tooth in this radiograph. (Photo contributor: James L. Kretzschmar, DDS, MBS.) (**B**) This Panorex film shows lucencies consistent with periapical abscesses, most notably of the right inferior posterior molar tooth (see arrows). (Photo contributor: David P. Kretzschmar, DDS, MS.)

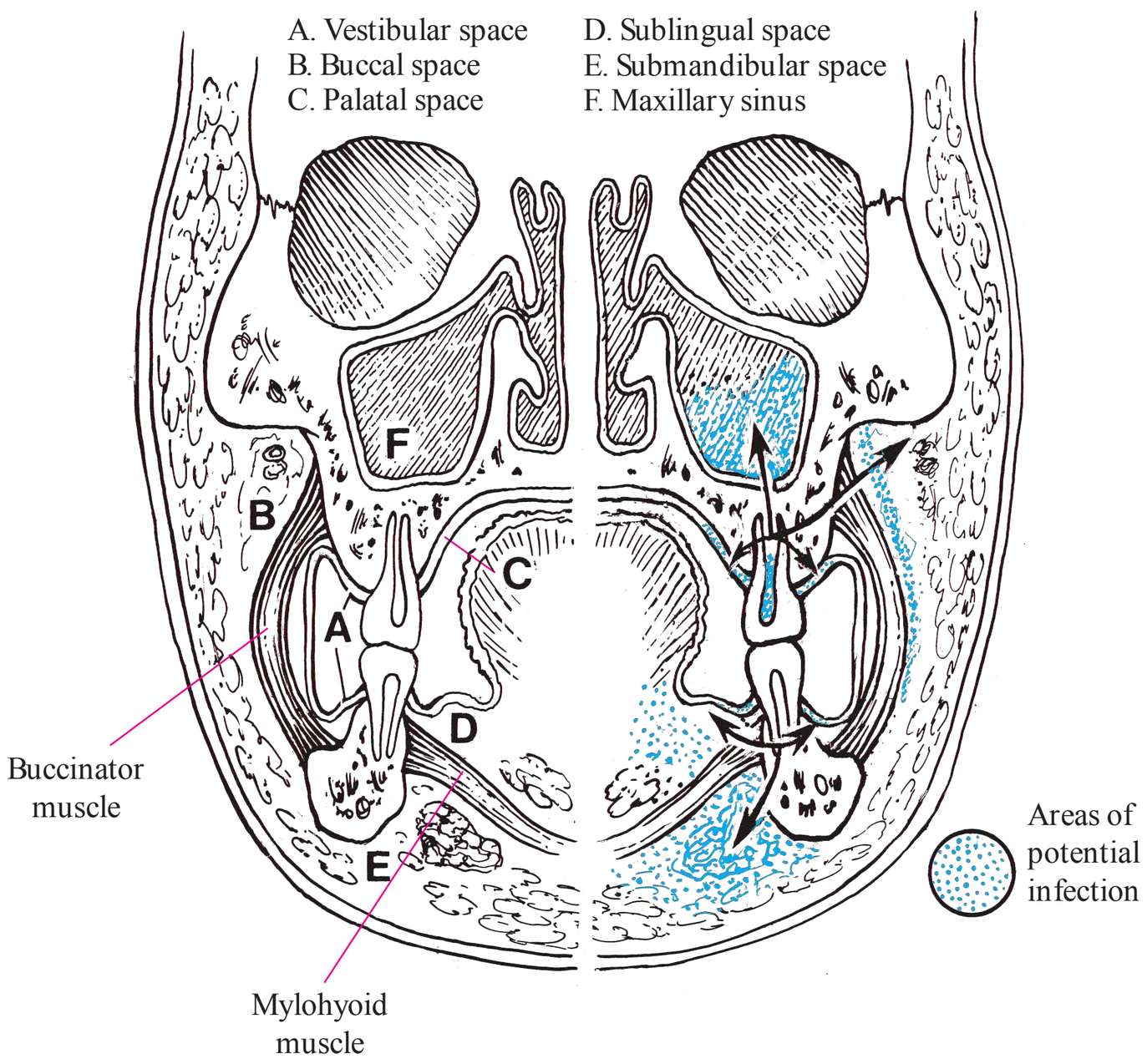


FIGURE 6.28 ■ **Odontogenic Abscesses.** As infection progresses from the pulp at the tooth apex, it erodes through the bone and can express itself in a variety of places. This illustration notes several possible locations or spaces. (Adapted with permission from Cummings C, Schuller D (eds). Otolaryngology Head and Neck Surgery. Chicago: Mosby-Year Book; 1986.)

Clinical Summary

A partially erupted or impacted third molar (wisdom tooth) is the most common site of pericoronitis and pericoronal abscesses. The accumulation of food and debris between the overlying gingival flap and crown of the tooth creates a focus for pericoronitis and subsequent abscess formation. The gingival flap becomes irritated and inflamed, and the tissue is repeatedly traumatized by the opposing molar tooth. The inflamed gingival process may eventually become infected and form an abscess. Foul taste, inability to close the jaw, and fever may occur. Swelling of the cheek and angle of the jaw as well as localized lymphadenopathy is also characteristic. More advanced disease may spread posteriorly to the base of the tongue, oropharyngeal area, and deep cervical spaces with resulting Ludwig angina and peritonsillar abscesses.

Management and Disposition

Superficial incision and drainage of the abscess with warm saline irrigation, analgesia, and antibiotic coverage and referral for possible extraction of the involved teeth are indicated.

Pearls

1. Pericoronitis and abscess formation rarely occur in the pediatric population and tend to be late-adolescent and adult processes; the mandibular third molar is the most commonly involved tooth.
2. Airway compromise is a potential complication with posterior extension of a pericoronal abscess.

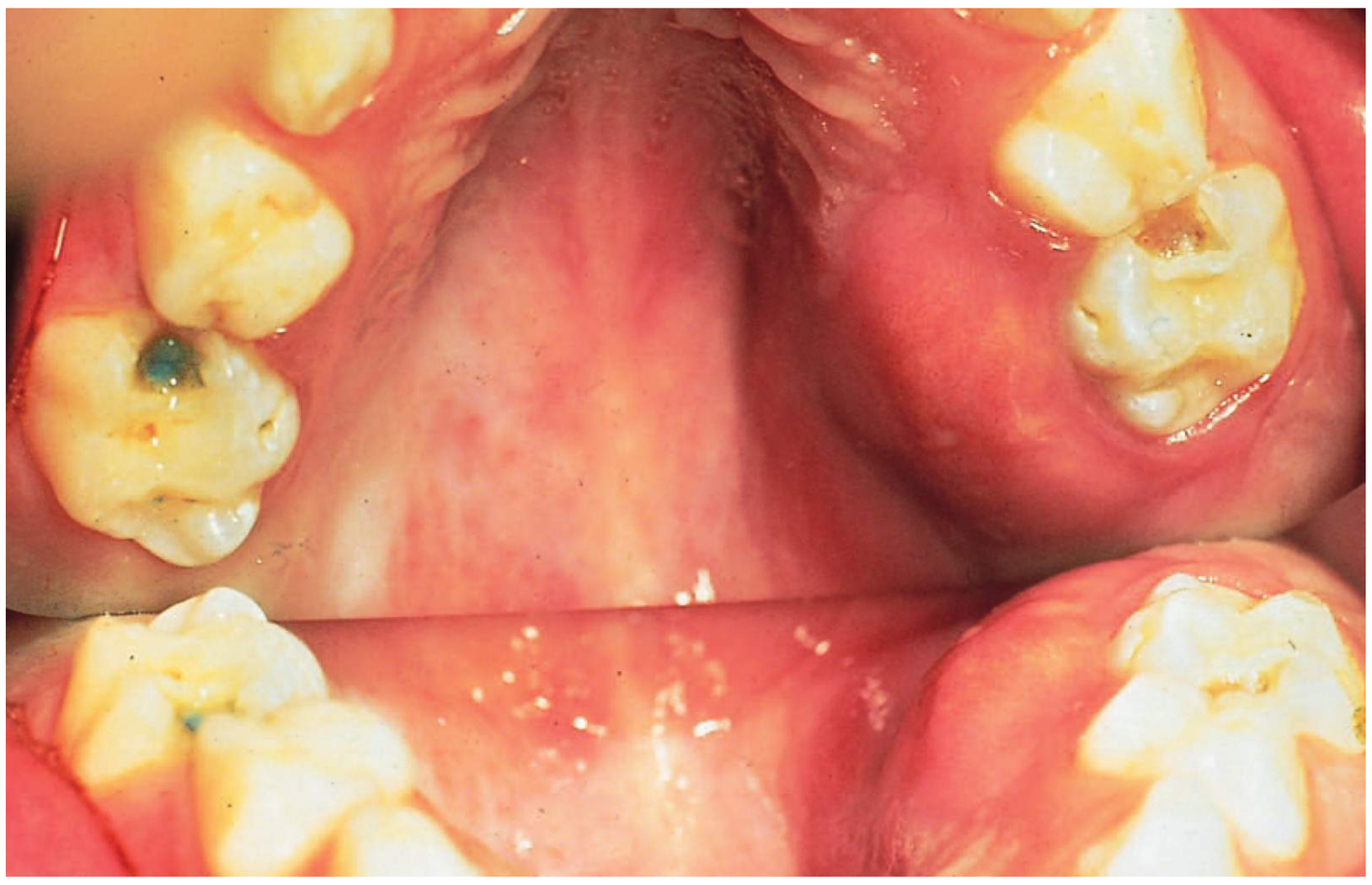


FIGURE 6.29 ■ **Pericoronal Abscess.** Note the inflamed fluctuant gingival tissue approximating the incompletely erupted third molar. (Photo contributor: James F. Steiner, DDS.)

BUCCAL SPACE ABSCESS

Clinical Summary

The buccal space lies between the buccinator muscle and the overlying superficial fascia and skin. The maxillary second and third molars are the usual nidus of buccal space infections, eroding either superiorly through the maxillary alveolar bone or, rarely, inferiorly from the third mandibular molar through the mandibular alveolar bone into the buccal space. Patients usually present with unilateral facial swelling, redness, and tenderness of the cheek. Trismus is generally not present. Parotid gland enlargement due to mumps and suppurative bacterial parotitis should also be considered. The former lacks erythema and warmth of the overlying skin, while the latter is accompanied by trismus and purulent drainage from Stensen duct. Inspection of all the maxillary and third mandibular molar teeth is essential to make the diagnosis. A CT scan may help localize the infection.

Management and Disposition

Broad-spectrum parenteral antibiotic therapy, NSAIDs or oral narcotic analgesics, and dental or oral surgical consultation for endodontic therapy, abscess drainage, and possibly dental extraction are indicated.

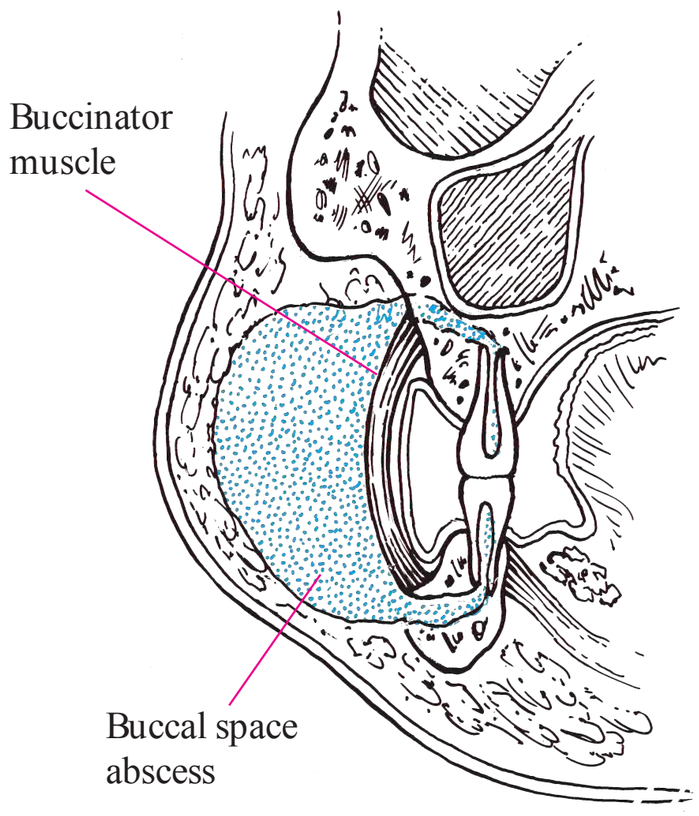


FIGURE 6.30 ■ **Buccal Space Anatomy.** The buccal space lies between the buccinator muscle and the overlying skin and superficial fascia. This potential space may become involved secondary to maxillary or mandibular molar infections. (Adapted with permission from Cummings C, Schuller D (eds). Otolaryngology Head and Neck Surgery. 2nd ed. Chicago: Mosby-Year Book; 1993.)

Pearls

1. Odontogenic infections of the second or third maxillary molars are the most common source for buccal space abscesses.
2. Ovoid cheek swelling with sparing of the nasolabial fold helps to differentiate this from a canine space abscess.

FIGURE 6.31 ■ **Buccal Space Abscess.** Note the ovoid cheek swelling with sparing of the nasolabial fold. This finding, along with accompanying redness and tenderness, helps to identify buccal space abscess formation. (Photo contributor: Michael J. Nowicki, MD.)

FIGURE 6.32 ■ **Buccal Space Abscess.** Note the marked facial asymmetry in this adult patient presenting with a buccal space abscess. (Photo contributor: David Effron, MD.)

Clinical Summary

The canine space lies between the anterior surface of the maxilla and levator labii superioris muscle of the face. Erosion of a maxillary tooth infection through the alveolar bone into the canine space leads to abscess formation, although cutaneous infections from the upper lip and nose are rare sources. Unilateral facial redness, pain, and swelling lateral to the nose with obliteration of the nasolabial fold are characteristic. Severe upper lip and lower eyelid swelling may cause drooling at the corner of the mouth or eye. Maxillofacial CT scan may aid in differentiating these lesions.

Management and Disposition

Parenteral antibiotic therapy including anaerobic coverage is indicated. Dental or oral surgical consultation for incision and drainage is the most definitive treatment for canine space abscesses. Extraction or endodontic treatment of the involved anterior maxillary teeth is usually necessary.

Pearls

1. Loss of the nasolabial fold is characteristic of canine space abscesses.
2. Canine space infections may lead to orbital cellulitis and cavernous sinus thrombosis.
3. Although these patients may drool when significant upper lip swelling is present, they typically do not have trismus, dysphagia, or odynophagia.

FIGURE 6.33 ■ **Canine Space Abscess.** Unilateral facial swelling lateral to the nose with associated redness and the typical loss of the nasolabial fold is shown. The maxillary canine tooth is usually the source of this process. (Photo contributor: Frank Birinyi, MD.)

LUDWIG ANGINA

Clinical Summary

Ludwig angina is defined as rapidly spreading bilateral cellulitis of the submandibular and sublingual spaces with associated tongue elevation. A characteristic painful, brawny induration is present in the involved tissue. The posterior mandibular molars are the usual odontogenic origin for the infection. Streptococcus, Staphylococcus, and Bacteroides species are the most common pathogens. Affected individuals are typically 20 to 60 years old, with a male predominance. Patients are usually febrile and may demonstrate impressive trismus, dysphonia, and odynophagia. Dysphagia and drooling are secondary to tongue displacement and oropharyngeal swelling. Potential airway compromise or spread of infection to the deep cervical layers and the mediastinum is possible.

Management and Disposition

Acute laryngospasm with airway compromise is a potentially life-threatening complication; thorough plans for definitive airway management should be prepared since up to one-third of patients require definitive airway placement. Broad-spectrum aerobic and anaerobic parenteral antibiotic therapy should be initiated. Analgesia should be given as needed. CT or MRI can be used to identify abscess location, but great care should be taken to ensure the patient can tolerate the imaging process. Definitive treatment requires surgical drainage of the abscess. Admission to the intensive care unit is indicated for airway surveillance and management.

Pearls

1. Brawny submandibular induration and tongue elevation are common and characteristic clinical findings.
2. The second mandibular molar is the most common site of origin for Ludwig angina.
3. Acute laryngospasm with sudden total airway obstruction may be precipitated by attempts at oral or nasotracheal intubation.

FIGURE 6.34 ■ **Ludwig Angina.** Note the diffuse submandibular swelling and fullness. Direct palpation of this area would reveal a characteristic brawny induration. Potential airway compromise is a key concern in all patients with Ludwig angina. (Photo contributor: Jeffrey Finkelstein, MD.)

FIGURE 6.35 ■ **Ludwig Angina.** Sagittal view on CT scan demonstrates fluid collection and abscess formation within the floor of the mouth along with superior displacement of the tongue in this patient with Ludwig angina. (Photo contributor: Jake Block, MD.)

PARAPHARYNGEAL SPACE ABSCESS

Clinical Summary

The parapharyngeal space, also known as the lateral pharyngeal or pharyngomaxillary space, is a pyramidal-shaped space with its apex at the hyoid bone and base at the base of the skull. Laterally it is bound by the internal pterygoid muscle and the parotid gland with the superior pharyngeal constrictor muscle acting as the medial border. The posterior aspect of this space is in close proximity with the carotid sheath and cranial nerves IX through XII. Presenting symptoms of parapharyngeal space abscesses include fever, dysphagia, odynophagia, drooling, and ipsilateral otalgia. Unilateral neck and jaw angle facial swelling in association with rigidity and limited neck motion is common. Potentially disastrous complications include cranial neuropathies, jugular vein septic thrombophlebitis, and erosion into the carotid artery. The origin of parapharyngeal abscesses may be from bacterial pharyngitis, sinuses, dentition, or lymphatic spread.

Management and Disposition

Preparations for definitive airway management via endotracheal intubation or surgery are vital. Early recognition and anticipation of other potentially disastrous complications should be considered and managed appropriately. Broad-spectrum antibiotic coverage for mixed aerobic and anaerobic infections should be initiated. Radiologic modalities used to assess parapharyngeal and other deep-space neck infections include contrast-enhanced CT, ultrasound, plain radiography, and MRI. Emergent otolaryngological or oral surgical consultation is warranted for definitive intraoperative incision and drainage of the abscess.

Pearl

1. Suspected oropharyngeal abscesses in association with cranial nerves IX through XII involvement are pathognomonic of parapharyngeal abscesses.

FIGURE 6.36 ■ **Parapharyngeal Space Abscess.** Unilateral facial, jaw angle, and neck swelling is seen in this patient. Nuchal rigidity may also be present. (Photo contributor: Sara-Jo Gahm, MD.)

TRENCH MOUTH (ACUTE NECROTIZING ULCERATIVE GINGIVITIS)

Clinical Summary

Painful, severely edematous interdental papillae are characteristic of acute necrotizing ulcerative gingivitis (ANUG), or trench mouth. Other associated features include the presence of ulcers with an overlying grayish pseudomembrane and a "punched out" appearance. The inflamed gingival tissue is friable, necrotic, and represents an acute destructive disease process of the periodontium. Fever, malaise, and regional lymphadenopathy are commonly associated signs. Patients may also complain of foul breath and a strong metallic taste. Poor oral hygiene, emotional stress, smoking, and immunocompromised states (eg, HIV, steroid use, diabetes) all predispose for ANUG. Anaerobic Fusobacterium and spirochetes are the predominant bacterial organisms involved. The anterior incisor and posterior molar gingival regions are the most commonly affected oral tissues. Acute herpetic, gonococcal, or streptococcal gingivostomatitis, aphthous stomatitis, desquamative gingivitis, and chronic periodontal disease may mimic ANUG.

Management and Disposition

Initial management includes warm saline irrigation, analgesics, topical anesthetics, hydrogen peroxide or chlorhexidine oral rinses, and antibiotic treatment with oropharyngeal coverage. Follow-up within 1 to 2 days is recommended. Patients with more advanced disease may require admission and oral surgical consultation.

Pearls

1. Dramatic relief of symptoms within 24 hours of initiating antibiotics and supportive treatment is characteristic.
2. Periodontal abscesses and underlying alveolar bone destruction are common complications of ANUG and require dental follow-up.
3. Gingivitis is a nontender inflammatory disorder and is not synonymous with ANUG.

FIGURE 6.38 ■ **Acute Necrotizing Ulcerative Gingivitis.** Excessive purulent drainage was associated with this patient's ANUG. (Photo contributor: R. Jason Thurman, MD.)

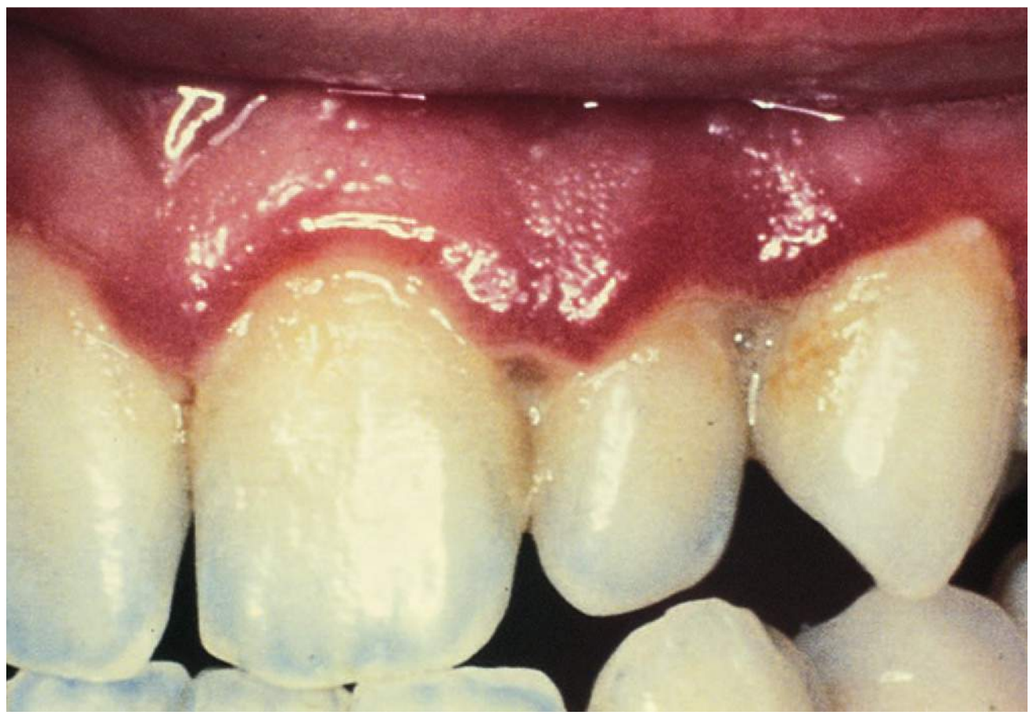


FIGURE 6.37 ■ **Acute Necrotizing Ulcerative Gingivitis.** Note the inflamed, friable, and necrotic gingival tissue. An overlying grayish pseudomembrane or punched out ulcerations of the interdental papillae are pathognomonic. (Photo contributor: David P. Kretzschmar, DDS, MS.)

FIGURE 6.39 ■ **Severe Acute Necrotizing Ulcerative Gingivitis.** Note the extensive gingival necrosis in this advanced case of trench mouth. (Photo contributor: David Effron, MD.)

ACID TOOTH EROSION (BULIMIA)

Clinical Summary

Bulimia nervosa is an eating disorder with significant associated physical complications. It is characterized by binge eating with self-induced vomiting, laxative use, dieting, and exercise to prevent weight gain. Patients with bulimia are at risk for damage to the dental enamel and dentin as a result of repeated episodes of vomiting with chronic exposure to regurgitated acidic gastric contents. The lingual dental surfaces are most commonly affected. In severe cases, all surfaces of the teeth may be affected. Trauma to the oral and esophageal mucosa may also result from induced vomiting. The quantity, buffering capacity, and pH of both the resting and stimulated saliva are found to be reduced, and salivary gland enlargement, most commonly the parotid, may occur as well.

Included in the differential diagnosis of acid tooth erosion are conditions that involve recurrent vomiting. Xerostomia is a condition of excessive mouth dryness (associated with Sjögren syndrome) that can also accelerate the process of enamel loss. Tooth abrasions and erosions may be brought about by the use of chewing tobacco, eating betel nuts (Indian paan), dentifrice use, bruxism, abnormal swallowing, and jaw clenching.

Management and Disposition

The initial emergency department (ED) management of patients with bulimia should address any medical complication of the disorder (hypokalemia, metabolic acidosis, etc). Hospitalization to stabilize medical complications and provide nutritional support may be indicated. Dental treatment should begin with vigorous oral hygiene to prevent further destruction of tooth structures. Regular professional fluoride treatments to cover exposed dentin and cosmetic procedures should be instituted once the patient is adequately stabilized psychologically. A multidisciplinary team approach is necessary and should involve psychiatry, internal medicine, and dental consultation as needed.

Pearls

1. The lingual surfaces of the teeth are the most commonly involved tooth surfaces.
2. Bruxism tends to cause enamel loss from occlusal and incisal dental surfaces.
3. The labial and buccal surfaces of the teeth tend to show enamel loss from repeat or prolonged chemical contact (eg, lemon sucking or tobacco products).

FIGURE 6.40 ■ **Acid Tooth Erosion (Bulimia).** Erosive dentin exposure of the maxillary teeth secondary to chronic vomiting. The involvement of the lingual dental surfaces is characteristic of bulimia. (Photo contributor: David P. Kretzschmar, DDS, MS.)

FIGURE 6.41 ■ **Acid Tooth Erosion (Snuff User).** Note the typical dentin exposure on the buccal dental surfaces resulting from prolonged snuff use and its accompanying acid erosion. (Photo contributor: David P. Kretzschmar, DDS, MS.)

THRUSH (ORAL CANDIDIASIS)

Clinical Summary

White, flaky, curd-like plaques covering the tongue and buccal mucosa with an erythematous base are typical of thrush. These lesions tend to be painless; however, painful inflammatory erosions or ulcers may be noted, particularly in adults. Predisposing factors include antibiotic use, inhaled and oral corticosteroids, radiation to the head and neck, extremes of ages, patients with immunologic deficiencies, and chronic irritation (eg, denture use and xerostomia). Colonization of surface epithelium by Candida occurs due to altered oral microflora. Hairy leukoplakia, lingual lichen planus, flecks of milk or food debris, and liquid antacid adhering to the tongue may be confused with candidiasis. Hairy leukoplakia cannot be removed with a tongue depressor (note Fig. 20.3). This helps differentiate this process from thrush or residue from ingested materials. Microscopic examination of the removed specimen for the presence of hyphae in potassium hydroxide mount will aid in the identification of Candida.

Management and Disposition

Nystatin oral tablets or swish and swallow suspension, fluconazole, or clotrimazole oral troches are therapeutic. Topical analgesic cocktails may provide comfort for patients (eg, liquid antacid with diphenhydramine, viscous lidocaine).

Pearls

1. Thrush is most common in premature infants and immunosuppressed patients.
2. In young adults, thrush may be the first sign of AIDS; a history of HIV risk factors should be elicited.
3. Failure of oral candidiasis to respond to topical antifungal agents may suggest an underlying immune deficiency.

FIGURE 6.42 ■ **Oral Candidiasis (Thrush).** Whitish plaques are seen here on the buccal mucosa. These plaques are easily removed with a tongue blade, differentiating them from lichen planus or leukoplakia. (Photo contributor: James F. Steiner, DDS.)

FIGURE 6.43 ■ **Oral Candidiasis (Thrush).** Extensive thrush is seen on the hard and soft palate of this immunocompromised patient. (Photo contributor: Lawrence B. Stack, MD.)

ORAL HERPES SIMPLEX VIRUS (COLD SORES)

Clinical Summary

Oral herpes simplex may present acutely as a primary gingivostomatitis or as a recurrence. Painful vesicular eruptions on the oral mucosa, tongue, palate, vermilion borders, and gingiva are highly characteristic. A 2- to 3-day prodromal period of malaise, fever, and cervical adenopathy is common. The vesicular lesions rupture to form a tender ulcer with yellow crusting and an erythematous margin. Pain may be severe enough to cause drooling and odynophagia, which can discourage eating and drinking, particularly in children. The disease tends to run its course in a 7- to 10-day period with nonscarring resolution of the lesions. Recurrent herpes labialis may present with an aura of burning, itching, or tingling prior to vesicle formation. Oral trauma, sunburn, stress, and any variety of febrile illnesses can precipitate this condition. Oral erythema multiforme or Stevens-Johnson syndrome, aphthous lesions, oral pemphigus, and hand-foot-mouth (HFM) syndrome are in the differential diagnosis. It should be noted that aphthous ulcers tend to occur on movable oral mucosa and rarely on immovable mucosa (ie, hard palate and gingiva). The vermilion border is a characteristic location for herpes labialis as opposed to aphthous lesions. Posterior oropharyngeal ulcerations with associated hand and foot lesions help to define HFM syndrome.

FIGURE 6.44 ■ **Herpes Simplex Virus (HSV) Stomatitis.** Note the extensive painful ulcerations on the patient's lower lip. A prodromal period of fever, malaise, and cervical adenopathy may herald the onset of these painful ulcerations. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Supportive care with rehydration and pain control is the mainstay of therapy. Temporary pain relief may be achieved with topical analgesics. Viscous lidocaine may be used as an oral rinse, 5 mL every 3 to 4 hours. Oral antiviral agents may be useful in adults with primary infections. Secondary infection of herpetic lesions should be treated with antibiotics.

Pearls

1. Fatal viremia and systemic involvement may occur in infants and children with herpetic gingivostomatitis.
2. Primary acute oral herpetic infection occurs most commonly in children and young adults.
3. Initiation of suppressive therapy with oral antivirals should be considered within the first 72 hours from symptom onset.

FIGURE 6.45 ■ **HSV Stomatitis.** Extensive vesicular lesions along the vermilion border and surrounding tissues are consistent with HSV infection. (Photo contributor: Frank Birinyi, MD.)

APHTHOUS ULCERS (CANKER SORES)

Clinical Summary

Aphthous ulcers are shallow 1 to 15 mm painful mucosal ulcers. A prodromal burning sensation may be noted 2 to 48 hours before an ulcer is noted. The initial lesion is a small white papule that ulcerates and enlarges over 48 to 72 hours. Lesions are typically round or ovoid with a raised yellow border and surrounding erythema. Multiple aphthous ulcers may occur on the lips, tongue, buccal mucosa, floor of the mouth, or soft palate. Spontaneous healing occurs in 7 to 10 days without scarring. The exact etiology is unknown. Deficiencies of vitamin B_{12}, folic acid, and iron as well as viruses have been implicated. Stress, local trauma, and immunocompromised states have all been cited as possible precipitators.

Primary or recurrent herpetic oral lesions may present with an almost identical prodrome and similar appearance. Herpetic lesions, unlike aphthous ones, tend to occur on the gingiva, hard palate, and vermilion border. Oral erythema multiforme may also present similarly to aphthous stomatitis; however, like oral herpes, it may tend to present with multiple vesicles in the early stages. Stevens-Johnson syndrome represents a severe form of erythema multiforme characterized by hemorrhagic anogenital and conjunctival lesions as well as oral lesions. Herpangina results from Coxsackie and echoviruses with oral ulcerations typically involving the posterior pharynx. Oral pemphigus should also be considered. Behçet disease can present with recurrent oral lesions, genital ulcers, and uveitis.

Management and Disposition

Supportive care, rehydration, and pain control constitute the focus of therapy. A topical anesthetic agent such as 2% viscous lidocaine or liquid antihistamine/antacid mix as an oral rinse every 3 to 4 hours is palliative. Use of oral antimicrobial rinses promotes healing. Protective dental paste may be applied every 6 hours to prevent irritation of lesions. Triamcinolone acetonide in an emollient dental paste three to four times daily may reduce pain and promote healing.

Pearls

1. Aphthous ulcers may be associated with Crohn disease.
2. Topical anesthetics may be used as a temporary adjunct in pain relief. Care must be taken to avoid overdose or complications such as methemoglobinemia from overuse.
3. Aphthous ulcers almost never occur on gums or hard palate.

FIGURE 6.46 ■ **Aphthous Ulcer (Single Lesion).** Raised yellow borders with surrounding erythema are typical of aphthous ulcers. (Photo contributor: James F. Steiner, DDS.)

FIGURE 6.47 ■ **Aphthous Ulcerations.** Note the multiple ulcers of various sizes located on the lip and gingival mucosa. These lesions rarely occur on the immobile oral mucosa of the gingiva or hard palate. (Photo contributor: James F. Steiner, DDS.)

STRAWBERRY TONGUE

Clinical Summary

Reddened hypertrophied lingual papillae, called strawberry tongue, are primarily associated with scarlet fever caused by group A streptococcal enterotoxin. The tongue initially appears white with the erythematous papillae sticking through the white exudate. After several days, the white coating is lost and the tongue appears bright red. Other signs of group A streptococcal infection include fever, an exudative pharyngitis, a scarlatiniform rash, and the presence of Pastia lines (petechial linear rash in the skin folds). Kawasaki syndrome may also present with an injected pharynx and an erythematous strawberry-like tongue. It is essential to make the distinction between streptococcal infection and Kawasaki syndrome, since the latter is associated with a high incidence of coronary artery aneurysm if left untreated. Also consider toxic shock syndrome (TSS), in which one-half to three-fourths of patients have pharyngitis with a strawberry-red tongue.

FIGURE 6.48 ■ **Strawberry Tongue.** Note the white exudate with bulging red papillae. The white coating is eventually lost after several days, and the tongue then appears bright red. (Photo contributor: Michael J. Nowicki, MD.)

Management and Disposition

Rapid streptococcal immunoassay testing may help expedite the diagnosis. Pharyngeal cultures are useful for confirming the diagnosis. Antistreptolysin O (ASO) titers can be used for confirmation in the convalescent stage if the diagnosis is in question. Penicillin is the drug of choice for group A streptococcus.

Pearls

1. Strawberry tongue initially appears white in color, with prominent red papillae bulging through the white exudate. After several days, the tongue becomes completely beefy red.

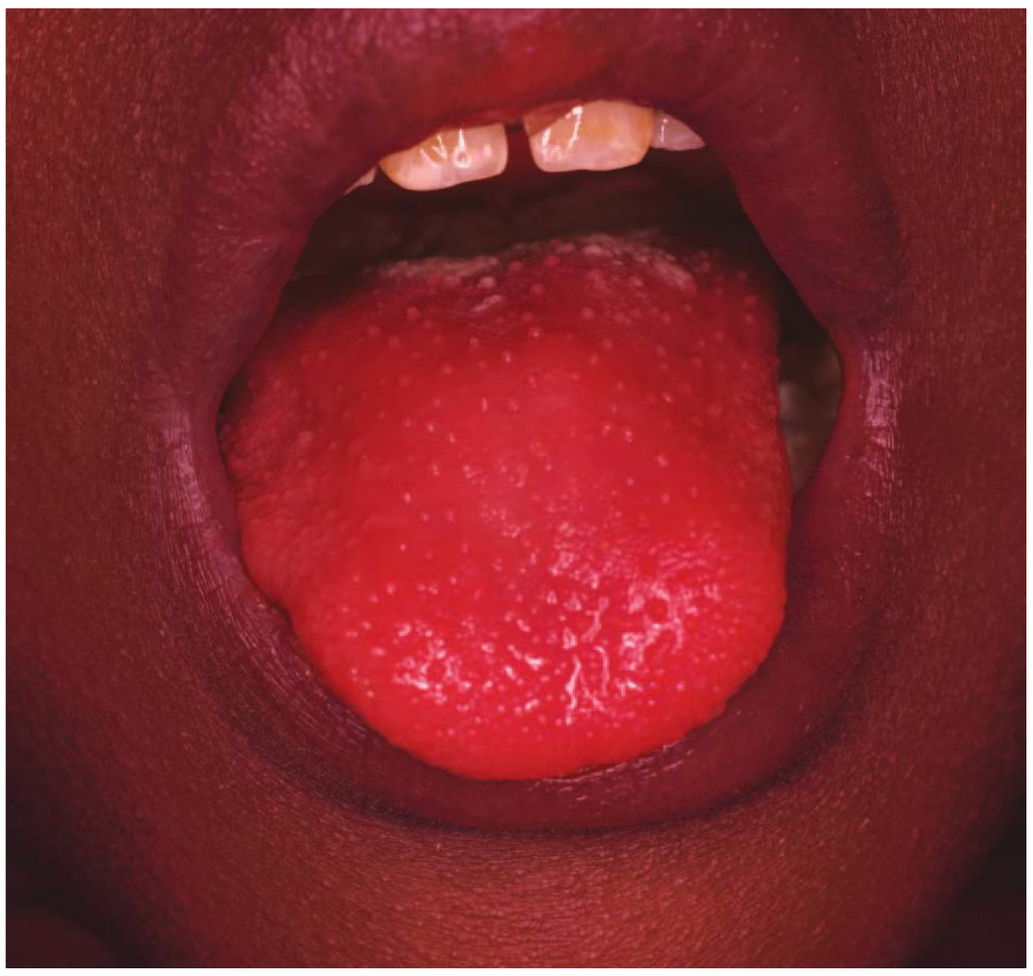


FIGURE 6.49 ■ **Strawberry Tongue.** This patient with scarlet fever demonstrates the bright red appearance of strawberry tongue after most of the white exudate is lost. (Photo contributor: Kevin J. Knoop, MD, MS.)

TORUS PALATINUS

Clinical Summary

Tori are benign nodular overgrowths of cortical bone, occurring in the midline of the palate where the maxilla fuses (palatinus) or on the mandible, typically on the lingual aspect of the molar teeth (mandibularis). Although their physical appearance may be alarming, there is generally no need for concern. Tori are covered by a thin epithelium, which is easily traumatized and ulcerated. These ulcerations tend to heal very slowly because of the poor vascularization of the tori. Torus palatinus, in particular, is slow growing and may occur at any age; however, it is most commonly noted prior to age 30 in adults, and affects women twice as frequently as men. There are a variety of oral conditions that may be confused with mandibular or palatal tori. Gingival fibromatosis, fibroma formation secondary to irritation, granulomas, abscesses, and oral neurofibromatosis located on the palate may resemble torus palatinus. Nodular bony enlargement in the oral cavity may also result from fibrous dysplasia, osteomas, and Paget disease. Oral malignancies may manifest themselves on the palate as primary lesions, though these are rare. Oral radiographs, CT scans, and biopsy may aid in differentiating these conditions.

Management and Disposition

Tori are normal structural variants and do not represent an inflammatory or neoplastic process. They require no treatment unless associated with a complication. Tori may enlarge enough to interfere with eating or speaking, and impair proper fitting of dental prostheses. For some patients, the mere presence of tori may be bothersome and undesirable. Referral is indicated for suspected malignancies or lesions of questionable origin.

Pearl

1. Torus palatinus and mandibularis are nontender and otherwise asymptomatic unless traumatized.

FIGURE 6.50 ■ **Torus Palatinus.** Note the nodular appearance and characteristic central palatal location. (Photo contributor: Kevin J. Knoop, MD, MS.)

FIGURE 6.51 ■ **Torus Palatinus.** Due to location, abrasions and ulcerations can occur on the thin overlying epithelium secondary to trauma. (Photo contributor: R. Jason Thurman, MD.)

Clinical Summary

Gingival hyperplasia, or overgrowth of the gingiva, is a relatively rare condition. Most commonly, gingival hyperplasia is drug-induced, with phenytoin, cyclosporine, and calcium channel blockers most commonly implicated. Gingival hyperplasia may also occur from genetic disorders, myelodysplastic syndromes, granulomatous disease, and neoplastic processes, and may occur in pregnancy or puberty. Generally the condition is asymptomatic, but patients may present to the ED due to bleeding gums, traumatized hyperplastic tissue, dental pain, gingival soreness, or associated periodontal disease.

Management and Disposition

ED treatment is geared toward symptomatic relief and identification of the underlying etiology. History may reveal a causative medication, while laboratory testing should rule out underlying blood dyscrasias. Good dental hygiene and dental follow-up are essential. Removal or reduced dosing of the offending agent may be considered. Gingivoplasty or gingivectomy may be necessary in extreme cases.

Pearls

1. Gingival hyperplasia may occur in up to 50% of patients taking phenytoin.
2. Underlying malignancies, especially leukemias, should be considered in the differential diagnosis of patients presenting to the ED with gingival hyperplasia.

FIGURE 6.52 ■ **Gingival Hyperplasia.** This patient presented to the ED complaining of recurrent gingival trauma and bleeding. (Photo contributor: R. Jason Thurman, MD.)

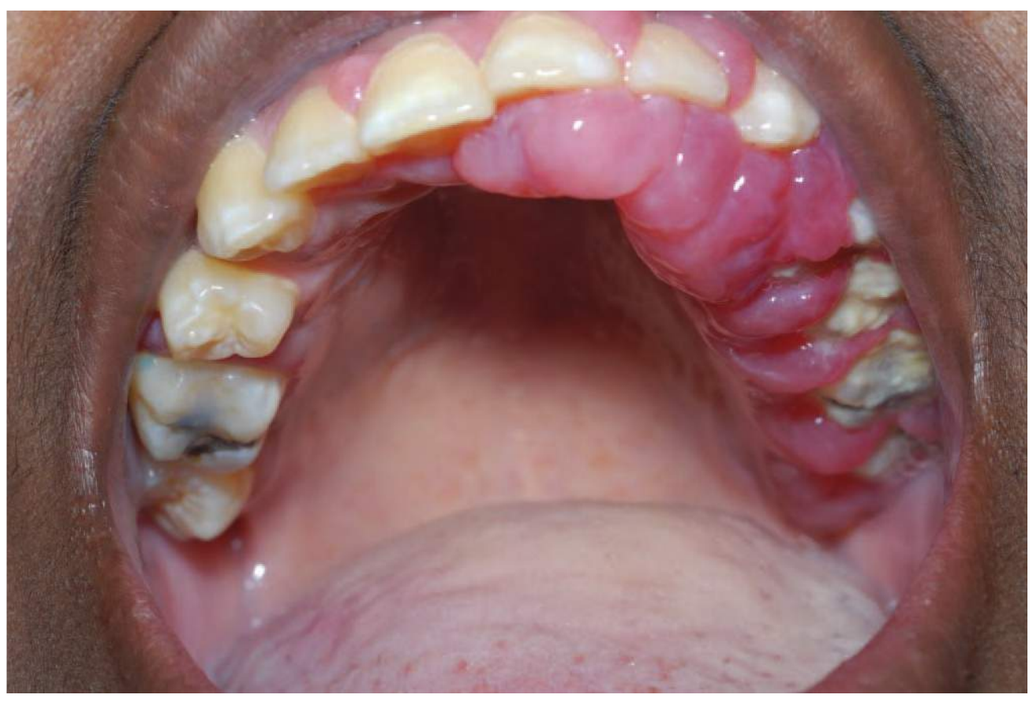


FIGURE 6.53 ■ **Gingival Hyperplasia.** Examination of the oral cavity of the patient in Fig. 6.52 reveals marked hyperplasia of the gingival tissues. (Photo contributor: R. Jason Thurman, MD.)

BLACK HAIRY TONGUE

Clinical Summary

Black hairy tongue (BHT) represents a benign reactive process characterized by hyperplasia and dark pigmentation of the tongue's filiform papillae. The elongated filiform papillae may reach up to 2 cm in length and vary in actual degree of pigmentation from light tan to black. Predisposing factors include excessive smoking, gastroesophageal reflux, poor oral hygiene, chemotherapy, and the use of broad-spectrum oral antibiotics. Pigment from consumed food, beverages, and tobacco products stains the entrapped food debris and desquamated papillary keratin. Some antibiotics may alter normal oral microflora and promote the growth of chromogenic organisms, also contributing to the tongue's discoloration. The darkly pigmented filament-like papillae give the tongue a black, hairy appearance. Males are more often affected than females; this condition very rarely occurs in children. Patients with BHT frequently note alterations of taste perception and fetid breath.

Geographic tongue and orolingual candidiasis may resemble more lightly pigmented forms of BHT. Similarly, dark discoloration of normal tongue papillae may also mimic BHT. This exogenous pigmentation of normal papillae may come from ingested food dyes and certain medications, such as bismuth-containing compounds, ketoconazole, and azidothymidine. The lack of hyperplastic filiform papillae with additional pigmentation of other oral mucosal surfaces may aid in distinguishing these conditions.

Management and Disposition

Improved oral hygiene with gentle tongue brushing and a reduction in the ingestion of exogenous pigment-containing substance represent the cornerstones of treatment. Removal of other predisposing factors (eg, antibiotic withdrawal and smoking cessation) will also promote resolution of this condition. The use of topically applied retinoid preparations and antifungal agents has been advocated for more refractory instances.

Pearls

1. BHT involves the superior aspect of the tongue.
2. The tongue is not always black and can be a tan or yellow color.

FIGURE 6.54 ■ **Black Hairy Tongue.** Hyperplasia of the filiform papillae on the dorsum of the tongue accompanied by deposition of dark pigment is characteristic of black hairy tongue. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

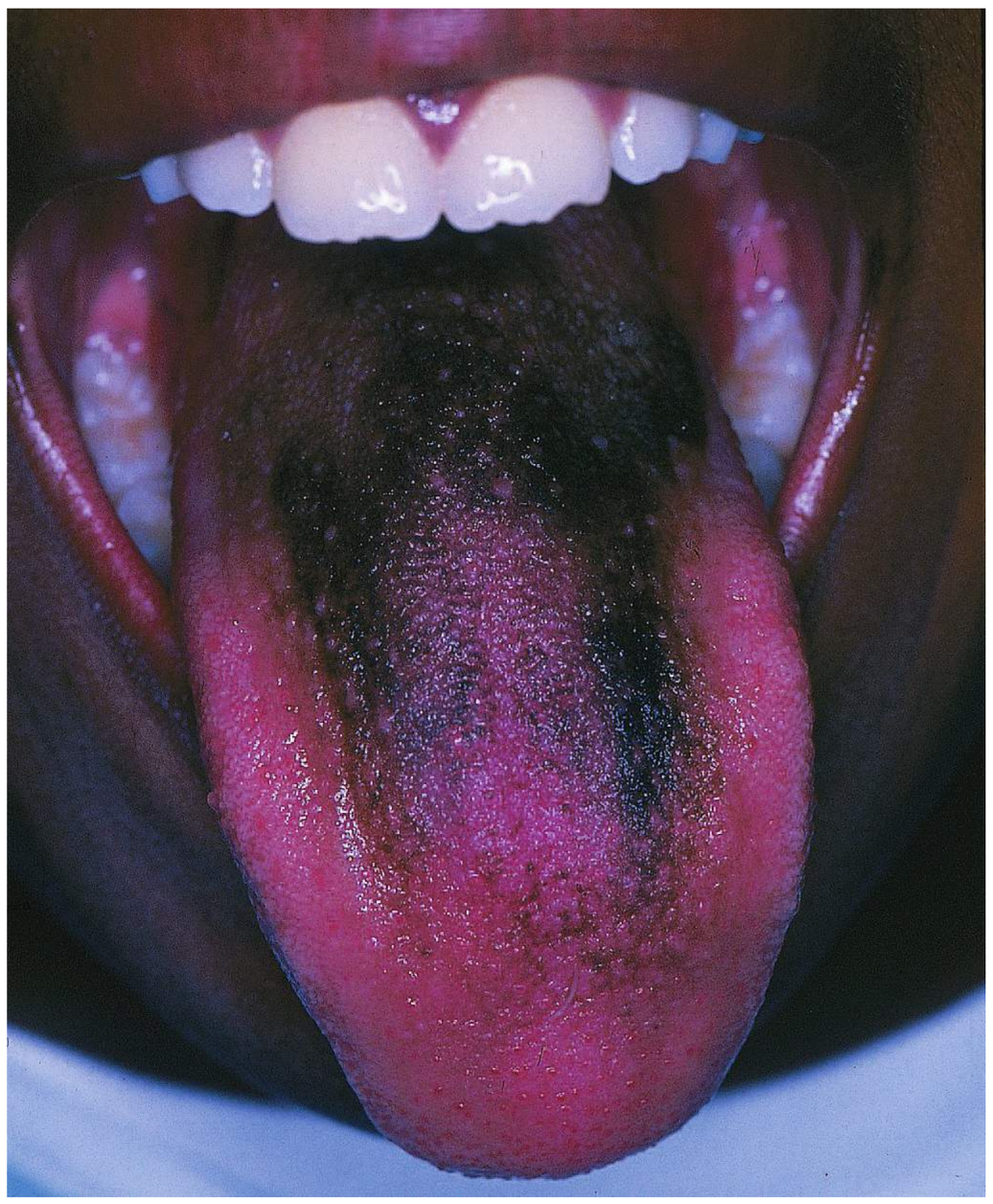


FIGURE 6.55 ■ **Black Hairy Tongue.** Deposition of black pigment secondary to bismuth ingestion. This patient ingested Pepto-Bismol. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Facial piercings are increasingly common. While external piercings, such as ear and eyebrow rings, are visibly apparent, intraoral and extraoral piercing may not be readily visible on initial inspection. An awareness and suspicion for recent lip, labret, tongue, or other oral mucosal piercings should be performed in any trauma patient as they may cause intraoral damage, produce artifact and diminished CT scan quality if not removed, and interfere during intubation. Common nontraumatic complications of intraoral include peri-piercing cellulitis, abscess formation, hemorrhage, penetrating wounds, dental injury, and granuloma formation around a stud. Though infrequent, systemic infection may result. If the ball comes off the post, it may be swallowed or, rarely, aspirated. Playing with tongue rings may result in lingual surface tooth abrasion, erosion, chipping, fracture, and gingival resorption.

Management and Disposition

Piercing should be removed prior to any CT scan if possible. For localized hemorrhage after a recent piercing, direct pressure should be applied. If bleeding persists, consider removal of the piercing. Any infection surrounding a piercing warrants removal of the stud as it will serve as a nidus for continued infection. Chest radiographs are required to evaluate for stud aspiration. Advise patients with chipped teeth to remove the bar or shorten the post.

Pearls

1. Remove piercings prior to cross-sectional imaging.
2. Piercings may act as a nidus for ongoing infection if not removed.
3. Ensure the prompt identification and removal of intraoral and extraoral piercings in any trauma patient potentially requiring emergent airway management, as the presence of jewelry can be a hindrance to successful intubation.

FIGURE 6.57 ■ **Oral Piercing Complication.** This patient suffered a painful injury when the stud of her lip ring came off, resulting in the penetration of her gingiva and inability to open her mouth. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 6.56 ■ **Tongue Piercing Cellulitis.** This patient presented with fever and tongue pain surrounding his piercing site. Note the erythema and swelling of the tongue and sublingual areas extending from the piercing site (central bubble). (Photo contributor: David Effron, MD.)

FIGURE 6.58 ■ **Oral Piercing Complication**. A close-up photograph of the oral piercing complication seen in Fig. 6.57. (Photo contributor: Lawrence B. Stack, MD.)