OPEN

Dental Topics for Plastic Surgeons, Part Three: Infection and Trauma of the Dentition

Jocelyn C. Zajac, MD,[*] Monica Manrique, MD,[*] Christopher M. Fleury, MD,[*] Jason Marrazzo, DDS,[†] Esperanza Mantilla-Rivas, MD,[*] Joseph H. Talbet, BA,[*] Ashleigh M. Brennan, BS,[*] Marudeen Aivaz, BS,[*] Albert K. Oh, MD,[*] Anupama R. Tate, DMD, MPH,[‡] and Gary F. Rogers, MD, JD[*]

Abstract: Conditions that affect dental and periodontal structures receive sparse coverage in the plastic surgery literature, yet a working knowledge of this subject matter is important in certain areas of clinical practice and a fundamental understanding is part of plastic surgery competency tested on the in-service and written board examinations. This 4-part series written to provide plastic surgeons with a working knowledge of dental topics that may be relevant to their clinical practice. This section, Part III, covers inflammatory and infectious conditions of the dentition and related structures, as well as dentoalveolar trauma.

Key Words: Abscess, dental trauma, dentoalveolar injury, gingivitis, oral trauma, osteomyelitis, periodontal disease, periodontal injury, periodontitis, plastic surgery, pulpal disease, pulpitis

(*J Craniofac Surg* 2021;32: 805–812)

INFECTIOUS CONDITIONS

Pulpal Disease

The dental pulp is non-mineralized dental tissue located in the central part of the tooth, contained within a restricted space surrounded by hard tissue that has a single vascular supply. As such, during infectious or inflammatory processes, the pulp has limited ability to foster an immune response.[1,2] Inflammation of the pulp, or pulpitis (Fig. 1), may be caused by mechanical damage (trauma, iatrogenesis), thermal/chemical exposure, or bacterial infection (via direct extension from overlying caries or hematogenous dissemination).[1] Pulpitis is classified as either reversible or irreversible. A tooth with reversible pulpitis will be mildly more sensitive to noxious stimuli (eg, temperature, electrical, biological, or mechanical stimuli), and recover slightly more slowly when compared to a normal tooth. The pain is often localizable, and the patient may be able to identify the causative tooth. If left untreated, severe bacterial invasion may create irreversible pulpitis and the patient may experience severe, continuous, not localizable pain to the same noxious stimuli listed above. Electrical stimulation, thermal, and mechanical testing may assist with identifying the affected tooth, determining the severity, and assessing the reversibility.[1] Proper identification of the involved tooth with early intervention can prevent progression to irreversible pulpitis and further pulpal necrosis. Removal of the causative agent is the best treatment course for reversible pulpitis, while root canal therapy or tooth extraction are the treatment options for irreversible pulpitis[1,3]

Delays in the treatment of a tooth with irreversible pulpitis may eventually progress to pulpal necrosis, an end-stage pathology. The affected teeth may initially show a pinkish discoloration that can progress to persistent gray with failure to return to normal color. This process may be accompanied by prolonged periapical inflammation, increased tooth sensitivity, tenderness to percussion, and arrested root development. Treatment of primary necrotic teeth

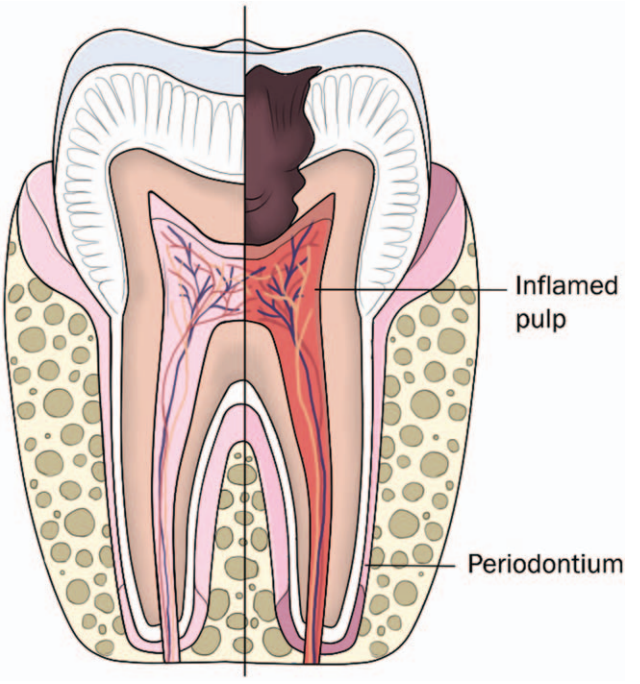

FIGURE 1. Pulpitis.

From the *Division of Plastic Surgery; †Division of Oral Maxillofacial Surgery; and ‡Division of Dentistry, Children's National Hospital, Washington, DC.

Received September 5, 2020.
Accepted for publication September 22, 2020.

Address correspondence and reprint requests to Gary F. Rogers, MD, JD, Division of Plastic Surgery, Children's National Hospital, 111 Michigan Avenue NW, Washington, DC 20010;
E-mail: grogers@childrensnational.org

The authors report no conflicts of interest.

ISSN: 1049-2275
DOI: 10.1097/SCS.0000000000007191

should strive to reduce the impact on the succedaneous permanent tooth. Depending on the condition of the tooth, the patient's age, and the position of the permanent succedaneous tooth, the pediatric dentist may recommend endodontic therapy or extraction of the affected tooth to prevent damage to its permanent successor.[2] Permanent teeth with pulpal necrosis are addressed with endodontic therapy or tooth extraction.[4] If the infective process spreads beyond the tooth structure and into the surrounding bone, it may expand into the soft tissues of the head and neck. Pathophysiologically, the pulp chamber within the tooth acts as a direct conduit to the underlying sterile alveolar bone and soft tissue and may result in periapical disease and associated complications.

Periapical Disease

In the setting of irreversible pulpitis and non-vital/necrotic teeth, the area surrounding the tooth apices may become inflamed with the development of cysts or abscesses. These pathologies are closely related and often transition reversibly from one to another. Apical periodontitis (often referred to as "periapical granuloma," although there is no histologic granuloma formation) (Fig. 2) can be divided into acute and chronic stages. Bacterial contamination in the root can easily spread into the apex of the tooth root. The acute phase is characterized histologically by a neutrophil-predominant infiltrate and osteoclast-driven bone resorption, and clinical may manifest with dull throbbing pain exacerbated by biting or percussion. Radiographs may show bony resorption surrounding the root apex and a characteristic periapical radiolucency. As the process transitions to chronic apical periodontitis, neutrophils are replaced by chronic inflammatory cells. At this stage the symptoms typically diminish, but there may be acute exacerbations. Because of this, chronic periapical granulomas are often discovered incidentally. Treatment focuses on eradicating the offending infection and eliminating the associated toxic products. If the tooth is salvageable, root canal therapy is undertaken. Otherwise, extraction with soft tissue curettage is performed for non-restorable teeth.[1]

Inflammation near the root may stimulate proliferation of the periapical epithelial cells, resulting in formation of a periapical cyst

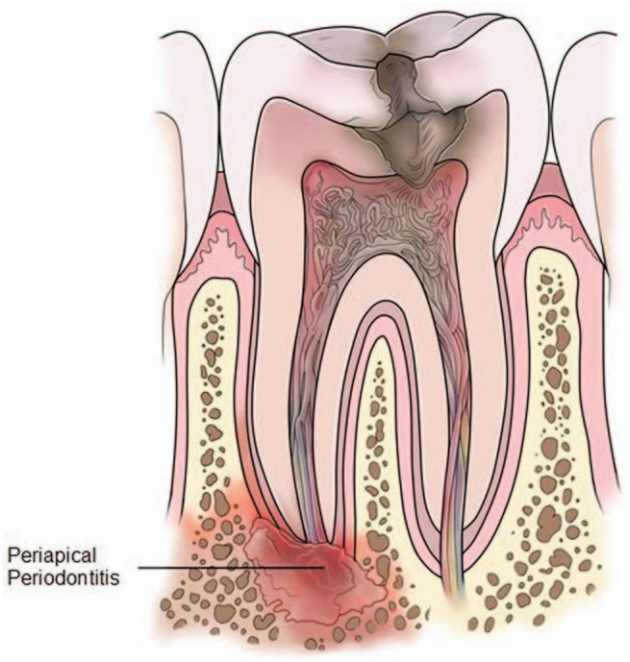

FIGURE 2. Apical periodontitis.

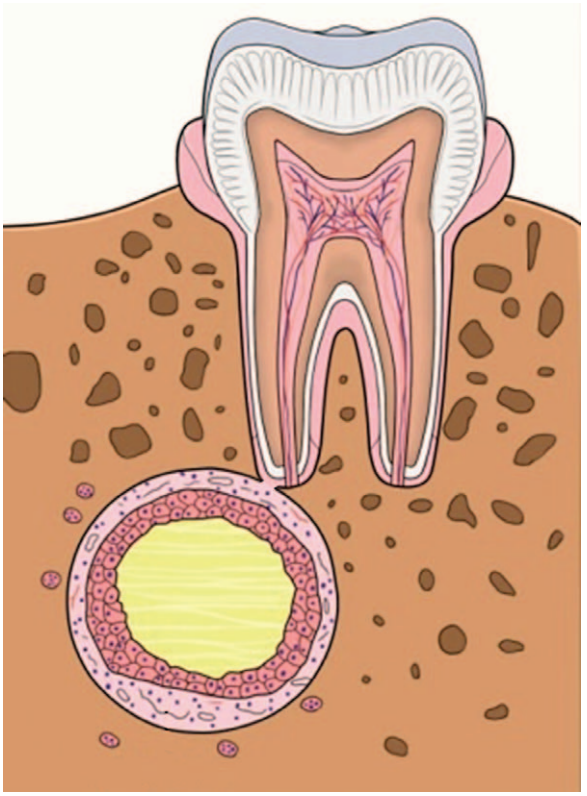

FIGURE 3. Periapical cyst.

(Fig. 3). These cysts are lined with stratified squamous epithelium and filled with protein-rich fluid and cellular debris. They slowly enlarge and are often asymptomatic, although symptoms may arise if inflammation increases or if the cyst grows significantly. Treatment of periapical cysts is the same as that for periapical granulomas. Residual periapical cysts (which persist after removal of the affected tooth) should be excised.[1] Periapical abscesses may develop de novo or from chronic periapical granuloma transformation, and are characterized by collection of inflammatory cells and infected tissue at the apex of a non-vital tooth. If the abscess extends into the alveolus, the patient may experience progressive pain, tenderness, sensitivity to percussion, swelling of adjacent tissue, extrusion of the affected tooth, and systemic signs of infection. Radiographs often demonstrate apical periodontal ligament broadening and an ill-defined radiolucency, but no significant bony defects. Sequelae of an inappropriately treated or untreated abscess include intraoral sinus tract formation, head and neck fascial space abscesses or cellulitis, and osteomyelitis. Management of periapical abscess focuses on drainage, possible tooth extraction with curettage of the cavity, or endodontic therapy if the tooth is thought to be salvageable. Antibiotics are usually unnecessary for treatment of localized abscesses unless there are systemic symptoms, involvement of fascial spaces or osteomyelitis. Intraoral sinus tracts often resolve spontaneously after appropriate treatment of the offending tooth, and require no further intervention.[1]

Complications of Pulpal and Periapical Disease

If a periapical abscess is not drained (either surgically or spontaneously), it can perforate through the cortical bone of the maxilla or mandible and extend along soft tissue fascial planes. The head and neck have multiple fascial spaces delineated by different anatomic structures, including muscles, bone, tendons, ligaments, and so on. Odontogenic infections have the potential to spread into

these spaces and result in superficial or deep space infection. Depending on their anatomic location and surrounding structures, these infections have varying degrees of severity and propensity to cause systemic harm. The most common complication and most frequent cause of death from odontogenic infections is airway compromise. Additional complications may also include, but are not limited to, visual disturbances/blindness, sinusitis, mediastinitis, septic thrombus formation, and systemic sepsis.

Two of the most important and dangerous sequelae of cellulitis arising from odontogenic infections are Ludwig angina and cavernous sinus thrombosis.[1] Ludwig angina is a rapidly progressive infection of the sublingual, submandibular, and submental regions bilaterally. It most often develops from infected mandibular molars and fractures, parapharyngeal space abscesses, oral lacerations, and tongue piercings. Once the submandibular region is involved, the infection can also spread to the retropharyngeal space, potentially extending along that fascial plane into the mediastinum with severe consequences, including mediastinitis, pericarditis, pneumonia, empyema, and death. Clinical features include airway obstruction, mouth and neck pain, restricted neck movement, dysphagia, dysphonia, dysarthria, and copious oral secretions. Fever, chills, leukocytosis, and elevated inflammatory markers may also be seen. Treatment of Ludwig angina centers on airway maintenance and infectious management with surgical intervention and/or intravenous antibiotics. Some clinicians recommend surgical decompression only for patients who are refractory to antibiotic therapy or have developed a localized abscess, while others believe surgery should be utilized in all cases.[1]

Cavernous sinus thrombosis (Fig. 4) refers to septic thrombus development within the of the cavernous sinus, an area located lateral to the sella-turcica and medial to the temporal bone. The cavernous sinus contains the oculomotor nerve (III), trochlear nerve (IV) ophthalmic and maxillary branches of the trigeminal nerve (V1 and V2), abducens nerve (VI), and internal carotid artery; infection and thrombosis of the sinus can affect any of these structures. Odontogenic infections are responsible for 10% of cavernous sinus thrombosis cases. This condition may develop from an anterior pathway initiating at the maxillary anterior teeth, or through a posterior pathway originating from maxillary premolar or molar teeth. Cavernous sinus thrombosis manifests as edematous periorbital enlargement, proptosis, ptosis, induration, and swelling of the adjacent forehead and nose, mydriasis, lacrimation, photophobia, vision loss, and facial pain. As the condition progresses, patients may develop meningitis and localized brain abscesses, altered mental status, and sepsis. Cavernous sinus thrombosis is treated by surgical extraction of infected teeth, drainage of any abscess, and a high-dose antibiotic regimen. Some experts also recommend anticoagulation to prevent further thrombosis and septic emboli.[1,5]

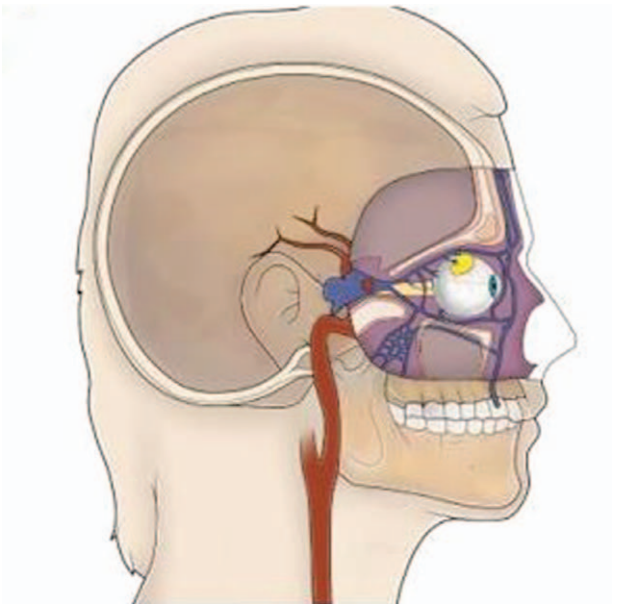

FIGURE 4. Cavernous sinus thrombosis.

Osteomyelitis

Osteomyelitis is defined as inflammation of the bone. It commonly involves the bone marrow, and has a tendency for progression to the cortical bone as well as the periosteum.[6] This term encompasses a broad range of pathologies, the most common of which (and most relevant for plastic surgeons), is suppurative osteomyelitis, which arises as a result of bacterial infection of the bone.[1]

Patients who are immunosuppressed, have chronic disease or decreased bony vascularity are at increased risk of developing osteomyelitis. Suppurative osteomyelitis arises as a result of bacterial infection and is classified as either acute or chronic. The acute phase is characterized by rapid spread of infection accompanied by pain, fever, lymphadenopathy, systemic symptoms, with timeline usually less than 1 month. Conversely, the chronic variant is characterized by the formation of scar and granulation tissue around the infection, with a smoldering clinical course.[1] Plain or panoramic films may demonstrate a nonspecific radiolucency, however, these findings typically lag several weeks behind clinical symptoms as cortical bone involvement is required for radiographic changes to be evident. Once these cortical changes occur, the classic "moth eaten" appearance is often visible. Computed tomography imaging, which is the largely the standard in evaluating maxillofacial pathology, may reveal a sequestrum (segment of dead bone isolated from surrounding bone) and/or and involucrum (sequestrum that has been surrounded by new bone).[6] A computed tomography scan, like a plain film, requires 30% to 50% demineralization of bone before radiographic changes can be appreciated and this may delay diagnosis. On the other hand, magnetic resonance imaging may allow an earlier diagnosis during the acute phase due to changes in the signal attenuation of the marrow. These changes on magnetic resonance imaging are visible before cortical disruption or sequestrum formation is evident.[7] The mainstay of treatment for both acute and chronic suppurative osteomyelitis is surgical drainage of any associated abscess with excision of all dead and infected bone, and culture-directed antimicrobial therapy. Weakened bone may require stabilization, and osseous defects may require reconstruction after the infection has been eradicated.[1,6]

Condensing osteitis and chronic osteomyelitis with proliferative periostitis (or Garre sclerosing osteomyelitis) (Fig. 5) are 2 other relatively common entities associated with a localized inflammatory response in the maxillofacial region. Condensing osteitis, also known as focal sclerosing osteomyelitis, involves areas of bony sclerosis adjacent to apical infections or pulpal necrosis and it is effectively managed by treating the underlying tooth infection. Garre sclerosing osteomyelitis is typically found in the pediatric population and often the results from an odontogenic source of infection or inflammation. This disorder is characterized by the formation of multiple parallel layers of reactive bone due to stimulation of the overlying periosteum. Treatment of the underlying source of inflammation typically leads to resolution and restoration of normal bone after 6 to 12 months. In some instances osseous recontouring may be necessary.[1]

There are other less common variants of osteomyelitis such as diffuse sclerosing osteomyelitis, primary chronic osteomyelitis, and chronic tendoperiostitis. Although an in-depth discussion of these

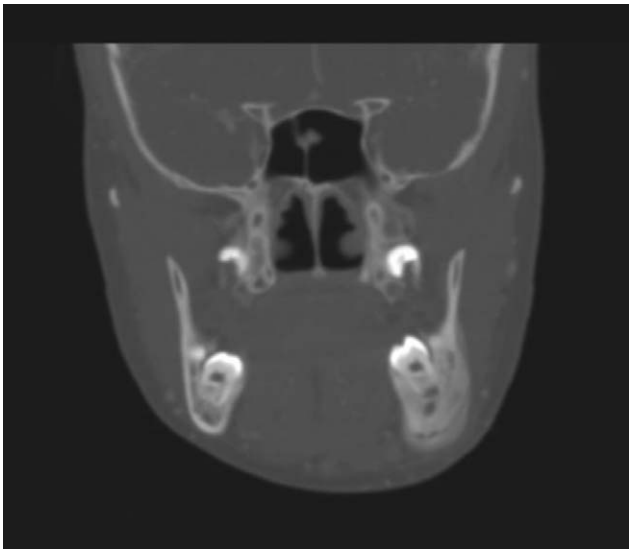

FIGURE 5. Garre osteomyelitis of the left mandible. CT scan showing classic "onion skinning" presentation of redundant layers of reactive cortical bone. CT, computed tomography.

often non-surgical pathologies is beyond the scope of this article, brief descriptions of each are provided. Diffuse sclerosing osteomyelitis results after an unknown stimulus (potentially prior chronic bacterial infection) stimulates sclerosis of the surrounding bone and is treated by eliminating the source of inflammation via tooth extraction or endodontic therapy. The sclerotic appearing areas may resolve after the source is treated or it may remain. The remaining sclerotic areas typically will remain hypovascular and are therefore sensitive to future inflammatory insults. Primary chronic osteomyelitis is a non-suppurative smoldering inflammation without obvious etiology, but may be related to an overactive immune response. It is characterized by recurrent flares of pain and swelling, with radiographs demonstrating diffuse areas of alternating osteolysis and sclerosis; over time, sclerosis predominates. Surgical and antimicrobial treatments have failed to show long-term success. Chronic tendoperiostitis presents with similar symptoms but is thought to be due to chronic overactivity of the muscles of mastication. Radiographs demonstrate areas of radiolucency most commonly localized around the insertion of the masseters, and treatment by decreasing muscle overuse often leads to resolution.[1]

Periodontal Disease

Periodontal disease refers to a group of pathologies that affect the supporting tissues of the teeth, namely the gingiva, periodontal ligament, cementum, and alveolar bone. Local risk factors include malocclusions, traumatic dental injuries, conditions causing plaque retention, and ectopic tooth eruption. General risk factors include genetic, metabolic, and hematological conditions, such as diabetes mellitus and smoking.[2] High levels of progesterone, stress, substance abuse, vitamin C deficiency, medications (calcium channel blockers, phenytoin, cyclosporine), and immune dysfunction may increase susceptibility to periodontal disease. The main management strategy of periodontal disease is optimizing gingival health, which involves eliminating causative factors, improving oral hygiene to reduce plaque, periodic professional plaque removal, and perhaps surgical recontouring of the affected gingiva. Two of the most common types of periodontal diseases are gingivitis and periodontitis.[8]

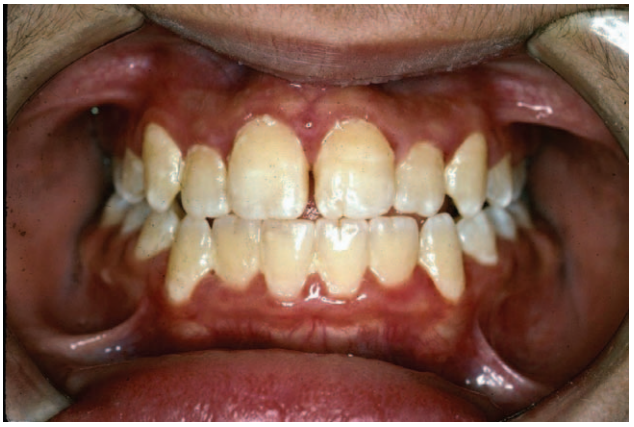

FIGURE 6. Gingivitis.

Gingivitis (Fig. 6) refers to inflammation of the soft tissues surrounding the teeth, excluding the alveolar ridge, periodontal ligament, and cementum.

There are many forms of gingivitis and an in-depth discussion is beyond the scope of this paper. However, there are 2 entities worth mentioning. One of the most severe forms of acute gingivitis is acute necrotizing ulcerative gingivitis (ANUG), also known as Vincent infection or trench mouth (Fig. 7). The most common causative organisms include fusiform bacteria, spirochetes, and gram negative anaerobes such as *Porphyromonas gingivalis*, *Veillonella* species, and *Selenomonas* species. ANUG is associated with smoking, malnutrition, and stress, and characteristically presents with necrosis and ulceration in areas of pre-existing gingivitis, leading to halitosis, pain, spontaneous hemorrhage, and necrosis. If inadequately treated, ANUG will persist in a cyclic pattern of acute symptoms followed by chronic remission and may eventually spread to the alveolar crest, causing necrotizing periodontitis with bone resorption and gingival recession.[8] In very rare cases, children and adults whom are extremely malnourished may develop an

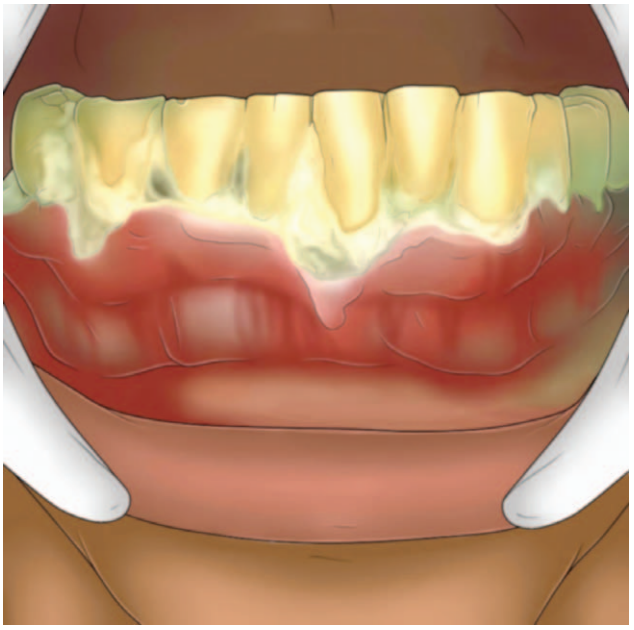

FIGURE 7. Acute necrotizing ulcerative gingivitis (ANUG).

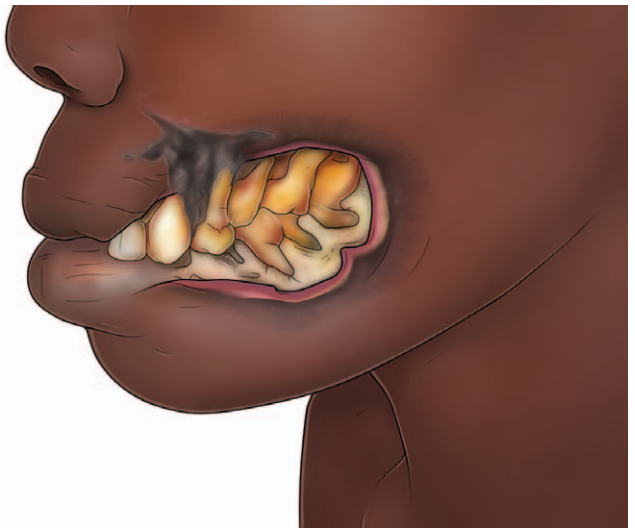

FIGURE 8. Necrotizing ulcerative stomatitis (Noma).

ANUG-like condition called necrotizing ulcerative stomatitis or Noma (Fig. 8). Unlike ANUG that is confined to the periodontium, NOMA may spread to the adjacent soft tissues causing necrosis of the cheeks and/or lips. However, ANUG usually demonstrates quick resolution with appropriate treatment including mechanical debridement, optimal oral hygiene, mouth rinses, and occasionally antimicrobials.[9]

In cases where gingivitis is inadequately treated, the inflammation may spread to the rest of the periodontium including the alveolus, cementum, and periodontal ligament, resulting in periodontitis (Fig. 9). Periodontitis manifests as gingival inflammation with resultant loss of periodontal ligament attachment and alveolar support secondary to apical migration of the crevicular epithelium along root surfaces and alveolar bone resorption, forming periodontal pockets.[8,10] Periodontitis is the leading cause of tooth loss in adults aged 35 and greater, and risk factors parallel those of gingivitis. Generally, it is chronic in nature and follows a cyclic pattern characterized by flares followed by remission; however, a more acute/aggressive periodontitis of both the primary and permanent dentition may also occur. Treatment of periodontitis includes modification of risk factors, improved oral hygiene with plaque removal, antimicrobial therapy in severe cases, and effective treatment of periodontal lesions involving surgical debridement, periodontal surgery or extraction of the involved teeth.[8]

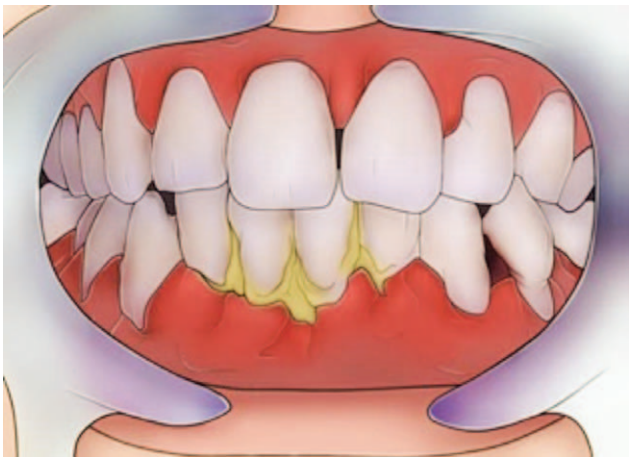

FIGURE 9. Periodontitis.

TRAUMA

Oral injuries can affect several dental structures and, depending of the severity of trauma, may compromise periodontal structures, surrounding soft tissue, or even the jaw. Traumatic injuries are common during childhood and adolescence, and occur most commonly from falls, child abuse, sports injuries or motor vehicle accidents. They are twice as common in boys and mostly affect the anterior teeth. Children with anterior protrusion of the teeth and craniofacial or neuromuscular abnormalities are especially susceptible to dental injuries.[11]

Trauma of the Dentition

Dental trauma can be the result of direct trauma, resulting in injury to the anterior teeth.[11] The literature describes several dental fracture classification systems; nonetheless fracture cataloging based on injury description may constitute a better approach.[12] Part one of this series covers dental anatomy so it will not be reviewed here.[13] For reference, the 4 parts of a normal tooth are shown below (Fig. 10).

Enamel infraction (Fig. 11), or crazing, refers to an incomplete fracture of enamel without loss of tooth structure and may be nonurgently evaluated as an outpatient.

Tooth fractures can be classified as uncomplicated or complicated, depending upon which tooth structures are involved. Uncomplicated fractures (Fig. 12) affect only the hard dental tissue such as the enamel. These types of fractures are treated with covering the exposed dentin to prevent secondary contamination or infection, and restoring aesthetics. These may be accomplished by dental restorative materials or smoothing the area or even replacement of the lost tooth fragment.[11,12]

Complicated fractures (Fig. 13) are those that involve both the enamel and dentin, permitting pulp exposure and presenting with dental bleeding or red, spotted appearance. Teeth with this type of injury are the most likely to develop pulpal necrosis and periapical

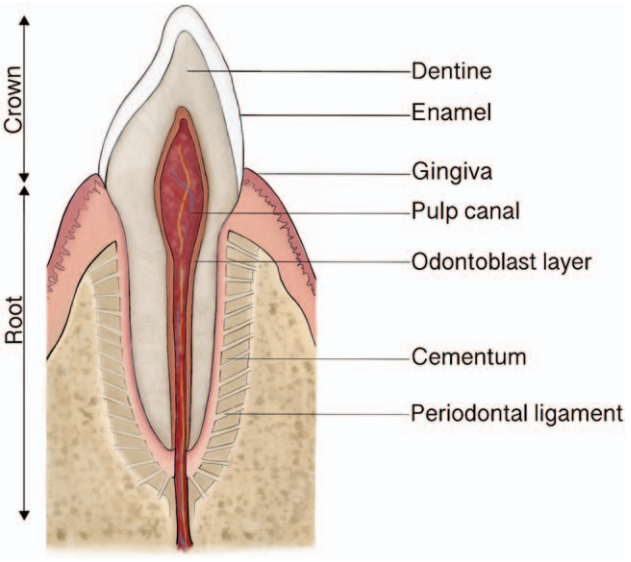

FIGURE 10. Normal tooth anatomy.

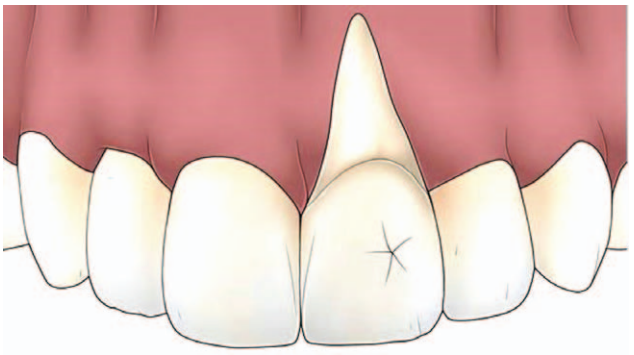

FIGURE 11. Enamel infraction.

abscess. This is a true dental emergency, and immediate assessment should be pursued to preserve the vitality of the underlying pulp and maintain appropriate tooth positioning.[1] Complicated fractures are treated with pulpectomy or pulpotomy. Splinting of the tooth may be needed if there is any involvement of the dental root.[11] Depending on the location of the root fracture they may be treated differently and have varying prognoses.[12]

Trauma of the Alveolar Bone

Periodontal and alveolar bone injury following trauma typically presents as mobile, displaced teeth and may be classified as concussion, subluxation, luxation, and avulsion.[11] Concussions refer to injuries that cause only edema and hemorrhage of the periodontal ligament without its rupture. Affected teeth are neither mobile nor displaced but do exhibit increased sensitivity to percussion. Most tooth concussions require no treatment and resolve completely. Subluxation is a type of injury that causes rupture of the periodontal ligament fibers with mild to moderate horizontal and/or vertical mobility of tooth-supporting structures, but there is no displacement of the teeth themselves. These injuries are usually associated with hemorrhage at the gingival margin.[11] Often times these teeth do not require treatment.[14]

There are different types of tooth luxation; mainly classified based on the direction of displacement. Intrusion or intrusive luxation (Fig. 14) causes a tooth to become pushed up into its socket, sometimes to the point where they are no longer visible clinically. Extrusion or extrusive luxation (Fig. 15) partially displaces a tooth from its socket towards the outside showing a

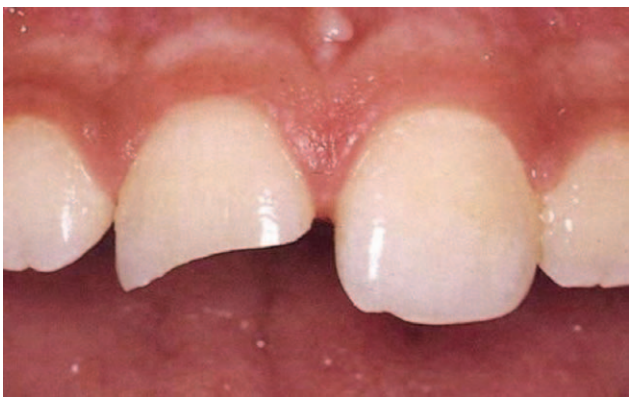

FIGURE 12. Uncomplicated dental fracture.

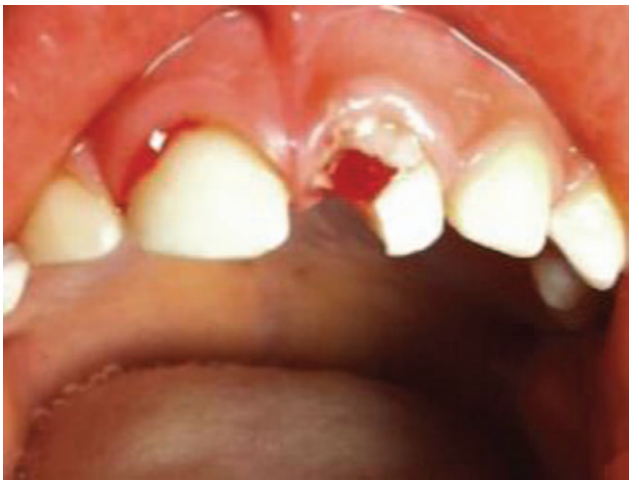

FIGURE 13. Complicated dental fracture.

vertically displaced, elongated, and excessively mobile tooth. Lateral luxation (Fig. 16) also involves partial displacement of a tooth, but horizontal directionality with or without axial displacement is noted, with the affected teeth usually displaced in a palatal-lingual or labial direction. They are typically immobile, and produce a high, metallic sound upon percussion. This pattern of injury is frequently associated with concurrent fracture of the alveolar process. Radiographs are indicated if intrusion, extrusion or lateral luxation injuries are suspected. The specific treatment of each of these varies slightly and is beyond the scope of this article, but is focused on prompt evaluation, repositioning, and fixation. Each of these injuries commonly affect the dental pulp, with the highest incidence of pulpal necrosis occurring with intrusion injuries. It is important to have these teeth monitored regularly by a dentist for tooth necrosis, in which case endodontic therapy is required. If a primary tooth is involved, extraction of the tooth may be the best treatment modality. However, recommendations vary based on the degree and direction of displacement as well as the patients dental age.[11,12,14]

Avulsion (Fig. 17) consists of an injury that causes complete tooth loss.[11] If the tooth is re-implanted appropriately within 1 hour after injury, normal tooth attachment is highly probable. After this period of time, however, periodontal structures will not survive, and root resorption and ankylosis may present. Patients with an avulsed tooth should be instructed to find the tooth, grasp it by the crown without manipulating the root, rinse it under running tap water,

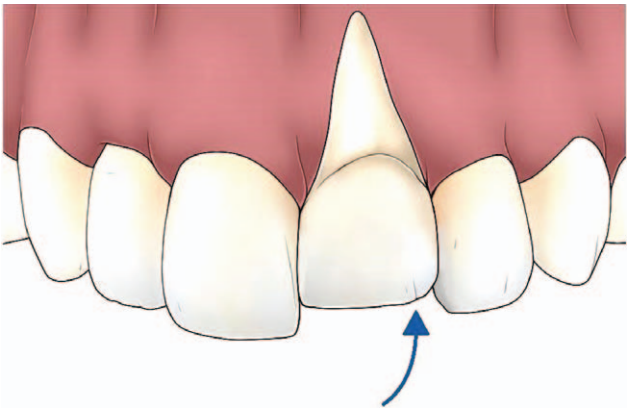

FIGURE 14. Intrusive luxation.

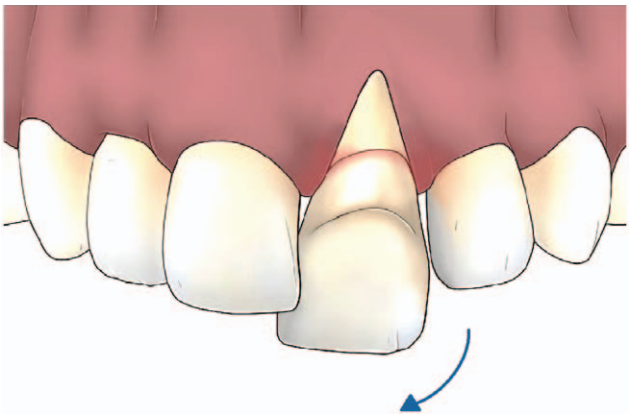

FIGURE 15. Extrusive luxation.

insert the tooth back into the socket or place it into cow's milk or normal saline, and then seek emergency dental treatment. Endodontic therapy will be required after tooth replantation and immobilization.[11,15]

Alveolar fractures (Fig. 18) may be associated with multiple fractured or displaced teeth, but vary in regards to the grade of displacement and mobility of the involved segment. Patients usually present with pain and malocclusion. Radiographs confirm the diagnosis and treatment involves manual reduction and fixation.[12] Care should be taken to preserve as much of the alveolar bone as possible because it is difficult to restore properly.[16]

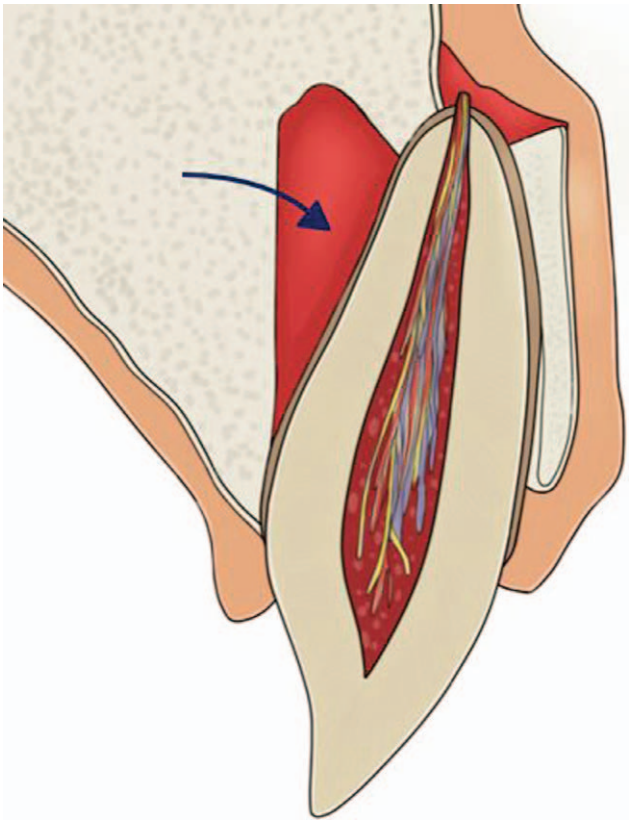

FIGURE 16. Lateral luxation.

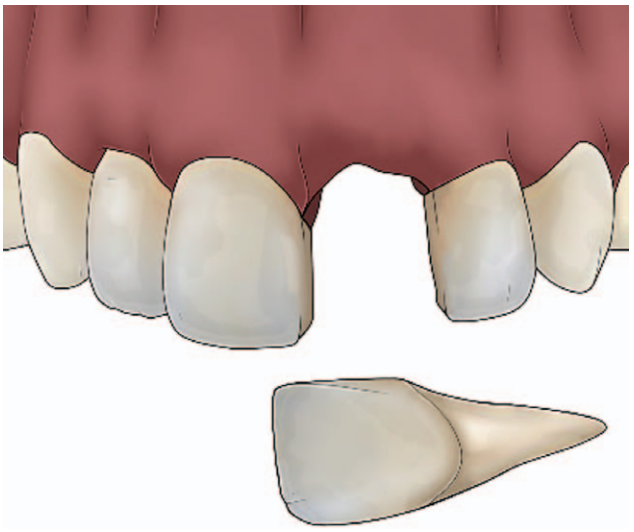

FIGURE 17. Tooth avulsion.

If oral injuries occur during permanent tooth development, the full extent of injuries following trauma cannot be elucidated until the eruption of all permanent teeth occurs. Injuries of the developing teeth may include white or yellow-brown enamel hypomineralization (Fig. 19) and crown dilaceration (Fig. 20), which consists of an abrupt deviation of the crown along its long axis, shifting previously formed hard tissue in the midst of developing soft tissue. Root development anomalies and eruption disturbances may also occur. Treatment options include micro-abrasion, resin or porcelain restoration, conservative measures, surgical exposure with orthodontic realignment, extraction, and splinting.[17]

Dentoalveolar Soft Tissue Trauma

Trauma to the face and perioral region may also cause injury to the dentoalveolar soft tissue. Injuries of the soft tissue may involve the buccal mucosa, gingiva, tongue, and the maxillary or lingual frenulum. Buccal mucosa and gingiva lacerations, if small or superficial, usually heal spontaneously, while lacerations larger than 1 to 2 cm and those involving deeper structures may require additional management. If a large mucosal laceration is present, it is vital to assess the integrity of surrounding underlying structures such as salivary ducts, branches of the trigeminal nerve and – in

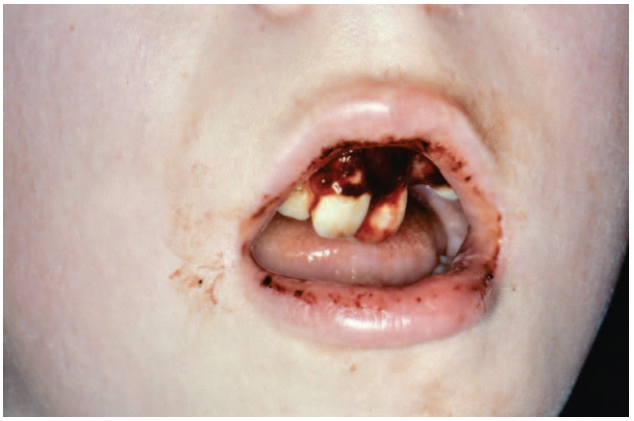

FIGURE 18. Alveolar fracture.

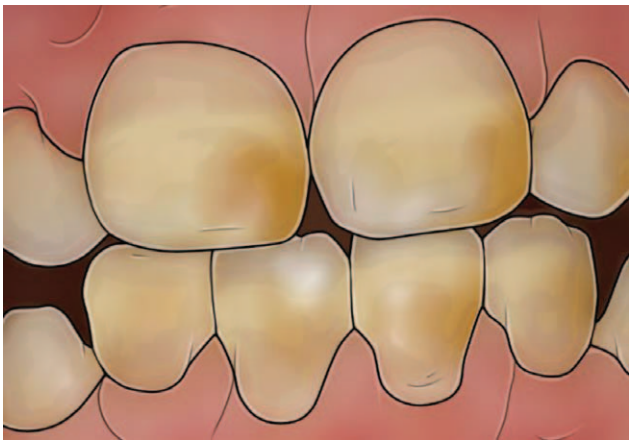

FIGURE 19. Enamel hypomineralization.

instances of a severe cheek laceration – the facial nerve. If these structures are intact, repair should be undertaken with closure of the dead space first by reapproximating underlying structures, followed by mucosal and/or skin closure. Small lesions of the gingiva likely require no treatment. Tongue lacerations that are small or located in the center of the tongue will usually heal completely without suturing. Repair should be undertaken if the wound does not easily self-approximate because it will otherwise epithelize, and create a grooved, bifid or lateral flap appearance. Wounds should also be approximated if they are bleeding extensively, are flap shaped, involve muscle, or are located along the tongue edge. Overall, dentoalveolar soft tissue injuries may be repaired with standard wound care and proper oral hygiene. Currently, there is no standard of care regarding antibiotic therapy.[12]

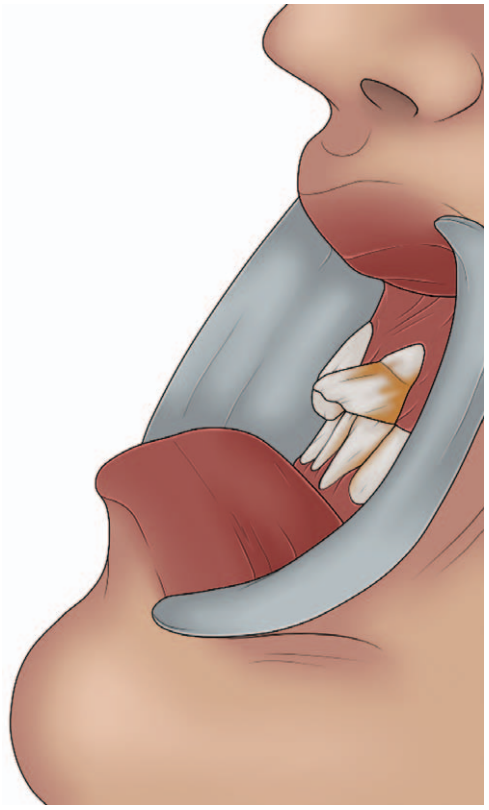

FIGURE 20. Crown dilaceration.

CONCLUSIONS

Inflammatory, infectious, and traumatic processes of the dentoalveolar structures may be first encountered by the plastic surgeon and a working knowledge of these problems is important to guide proper patient care. The overview provided in this series of papers serves as a starting point for plastic surgeons who encounter these conditions to manage or, as warranted, refer to the appropriate dental specialist.

REFERENCES

1. Neville BW, Damm DD, Allen CM, et al. Pulpal and periapical disease. In: Neville BW, Allen CM, Damm DD, Chi AC, eds. *Oral and Maxillofacial Pathology.* 4th ed. St. Louis, MO: Elsevier; 2015: 111–139
2. Goldberg M. Pulp anatomy and characterization of pulp cells. In: Goldberg M, ed. *The Dental Pulp. Biology, Pathology, and Regenerative Therapies.* Paris, France: Springer; 2014:13–34
3. Bjørndal L, Ricucci D. Pulp inflammation: from the reversible pulpitis to pulp necrosis during caries progression. In: Golberg M, ed. *The Dental Pulp: Biology, Pathology, and Regenerative Therapies.* Paris, France: Springer-Verlag; 2014:125–140
4. Saoud TM, Martin G, Chen YHM, et al. Treatment of mature permanent teeth with necrotic pulps and apical periodontitis using regenerative endodontic procedures: a case series. *J Endod* 2016;42:57–65
5. Crighton A, Meechan J. Oral medicine and oral surgery in children. In: Welbury R, Duggal MS, eds. *Paediatric Dentristy.* 5th ed. Oxford, UK: Oxford University Press; 2018:310–340
6. Kushner G, Alpert B. Osteomyelitis and osteoradionecrosis. In: Miloro M, ed. *Peterson's Principles of Oral and Maxillofacial Surgery.* 2nd ed. Hamilton, Canada: BC Decker; 2004:313–322
7. Schuknecht B, Valavanis A, Sailer H. Mandibular osteomyelitis: evaluation and staging in 18 patients, using magnetic resonance imaging, computed tomography and conventional radiographs. *J Cranio-Maxillo-Facial Surg* 1997;25:24–33
8. Neville BW, Damm DD, Allen CM, et al. *Periodontal Diseases Oral Maxillofacial Pathology.* 4th ed. St. Louis, MO: Elsevier; 2015:140–163
9. Leão JC, Gomes VB, Porter S. Ulcerative lesions of the mouth: an update for the general medical practitioner. *Clinics* 2007;62:769–780
10. Heasman P, Waterhouse P. Periodontal diseases in children. In: Welbury R, Duggal MS, Hosey MT, eds. *Paediatric Dentristy.* 5th ed. Oxford, UK: Oxford University Press; 2018:208–226
11. Dhar V. Dental trauma. In: Kliegman RM, St. Geme III JW, Blum NJ, Shah SS, Tasker RC, Wilson KM, eds. *Nelson Textbook of Pediatrics.* 21st ed. Philadelphia, PA: Elsevier; 2019
12. Benko KR. Emergency dental procedures. In: Roberts JR, ed. *Roberts & Hedges' Clinical Procedures in Emergency Medicine.* 6th ed. Philadelphia, PA: Elsevier; 2014:1342–1363
13. Zajac JC, Abbate O, Oh AK, et al. Dental topics for plastic surgeons, part one: normal anatomy, growth and development. *J Craniofac Surg* 2020;31:1168–1173
14. Bourguignon C, Cohenca N, Lauridsen E, et al. International association of dental traumatology guidelines for the management of traumatic dental injuries. Fractures and luxations. *Dent Traumatol* 2020;36:314–330
15. Fouad AF, Abbott PV, Tsilingaridis G, et al. International association of dental traumatology guidelines for the management of traumatic dental injuries: avulsion of permanent teeth. *Dent Traumatol* 2020;36:331–342
16. Mayersak R. Facial trauma. In: Walls R, ed. *Rosen's Emergency Medicine: Concepts and Clinical Practice.* 9th ed. Philadelphia, PA: Elsevier; 2018:330–344
17. Wellbury R, Withworth J, Stone S, et al. Traumatic injurie to the teeth. In: Welbury R, Duggal M, Hosey M, eds. *Paediatric Dentristy.* 5th ed. Oxford, UK: Oxford University Press; 2018:227–256