

Risk Assessment and Management of Periodontal Diseases and Pathologies in Pediatric Dental Patients

Adopted

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Abstract

This best practice supports clinicians in assessing risk for and clinical decision making in the management of periodontal diseases and pathologies in pediatric dental patients. This document highlights principles of periodontal disease diagnosis, risk assessment, and therapies to be applied to pediatric dental patients with special considerations for individuals with special health care needs when indicated. Recommendations on the management of contributing factors and conditions that increase the risk of periodontal disease and pathologies, as well as treatment considerations on the use of adjunctive antibiotics and surgical therapies, are reviewed. Special attention is focused on care coordination, collaborations, and referral of care to specialists. In cases where the published data regarding periodontal diseases and pathologies among children and adolescents was limited, recommendations were extrapolated from evidenced-based literature among adult patients, as well as on the consensus opinions of the working group.

This document was developed through a collaborative effort of the American Academy of Pediatric Dentistry Councils on Clinical Affairs and Scientific Affairs to offer information and guidance regarding risk assessment and management of periodontal diseases and pathologies in pediatric dental patients.

KEYWORDS: PERIODONTAL DISEASE, PERIODONTAL-RISK ASSESSMENT, PERIODONTAL THERAPY, CHILD, ADOLESCENT, ANTIBIOTIC THERAPY

Purpose

The American Academy of Pediatric Dentistry (AAPD) recognizes the importance of periodontal health and its effect on the well-being of pediatric patients, including those with special health care needs (SHCN). Periodontal-risk assessment (PRA) and management protocols are essential elements of contemporary clinical care for pediatric dental patients. These recommendations are intended to assist practitioners in assessing risk for and clinical decision making in the management of periodontal diseases and pathologies in pediatric dental patients.

Methods

This best practice document was developed utilizing the resources and expertise of AAPD members and an expert consultant in periodontics operating through the Council on Clinical Affairs. Literature searches of PubMed®/MEDLINE and Google Scholar databases were conducted using the terms: periodontitis as a manifestation of systemic diseases, necrotizing periodontitis, aggressive periodontitis, localized periodontitis; fields: all; limits: within the last 10 years, human, English, clinical study, clinical trial, comparative study, multicenter study, observational study, randomized clinical trial, meta-analysis, and systematic reviews. The search returned 1,222 articles that matched the criteria. The articles were evaluated by title and/or abstract and relevance to dental care for children and adolescents. When data did not appear sufficient or were inconclusive, recommendations were based upon expert

and/or consensus opinion by experienced researchers and clinicians.

Background

A periodontal examination and risk assessment are important parts of the routine dental examination of pediatric dental patients. The gingival and periodontal tissues in the primary, mixed, and permanent dentition are subject to morphological changes due to normal patterns of oral growth and development. Gingivitis occurs in half of the population by age of four or five years and peaks nearly to 100 percent at puberty.^{1,2} Distinguishing normal physiological changes during growth and development from gingival and periodontal diseases helps prevent erroneous diagnoses and unnecessary treatment. Maintenance and restoration of gingival and periodontal health during childhood and adolescence will facilitate healthy gingival and periodontal health at older ages.

ABBREVIATIONS

AAPD: American Academy of Pediatric Dentistry. **BoP:** Bleeding on probing. **CAL:** Clinical attachment loss. **CEJ:** Cementoenamel junction. **CHX:** Chlorhexidine. **MM:** Millimeter. **NSAIDs:** Non-steroidal anti-inflammatory drugs. **PDL:** Periodontal ligament. **PPD:** Periodontal pocket depth. **PRA:** Periodontal-risk assessment. **SHCN:** Special health care needs. **SIB:** Self-injury behavior. **SRP:** Scaling and root planning. **TDI:** Traumatic dental injuries.

Recommendations

Diagnostic phase

The diagnostic criteria for gingivitis are based on clinical features, taking into consideration the presence of plaque and that the inflammatory response to plaque is an age-dependent phenomenon. Three distinct forms of periodontal disease have been defined as: (1) periodontitis (single category grouping the two forms of the disease formerly recognized as aggressive or chronic); (2) necrotizing periodontitis; and (3) periodontitis as a manifestation of systemic conditions.³ Early diagnosis ensures more promising treatment outcomes and effective periodic maintenance protocols.⁴

Periodontal-risk assessment (PRA)

In health care, risk is defined as the probability that an individual will develop a disease during a specific time period.^{5,6}

Risk factors are defined as characteristics of individuals that increase their probability to developing the disease.^{5,6} Risk factors for periodontal disease are complex and may be biological, environmental (social), and behavioral.⁶ PRA identifies risk factors that place individuals at an increased risk of developing gingival and periodontal diseases and pathologies, as well as factors that influence the progression of the disease. PRA can improve clinical decision making and allow the implementation of individualized treatment planning and proactive targeted interventions.⁷ Evidenced-based PRA tools have been developed based on studies conducted among adult patients.⁸ Due to the limited literature regarding PRA among children and adolescents, factors associated with elevated risk were extrapolated from evidence from adult patients (Tables 1 and 2).^{5,9-13}

Table 1. Factors Associated with the Development and Progression of Periodontal Diseases and Pathologies for Patient <13 Years Old

Factors	High risk	Moderate risk	Low risk
<i>Biological factors</i>			
Systemic conditions/genetic susceptibility (e.g., family history of aggressive periodontitis) and syndromes ^α	Yes		
Immunosuppressive or radiation therapy		Yes	
Medication(s) known to affect the periodontal tissues		Yes	
History of traumatic injury to the periodontal apparatus (e.g., avulsion, luxation)		Yes	
Traumatic gingival/oral mucosal lesions		Yes	
Nutritional deficiencies		Yes	
<i>Social and behavioral factors</i>			
Socioeconomic stability (e.g., adequate health literacy, regular dental care)			Yes
Adequate daily at-home oral hygiene either performed or supervised by caregiver			Yes
Tobacco or marijuana smoking/smokeless tobacco use	Yes		
<i>Clinical and radiographic factors</i>			
Adequate attached gingiva and normal frenum attachments			Yes
Tooth-related factors contributing to plaque retention		Yes	
Physical barriers for proper oral hygiene		Yes	
Generalized gingivitis (≥30% of teeth affected)		Yes	
Disproportional gingival inflammation in relation to age, amount of plaque accumulation, or oral and systemic developmental changes	Yes		
Presence of calculus	Subgingival	Supragingival	None
Bleeding on probing	Yes		
Periodontal probing depths >3 millimeter	Yes		
Chronic pericoronitis		Yes	
Abnormal tooth mobility	Yes		
Furcation involvement	Yes		
Radiographic alveolar bone loss	Yes		
Tooth loss due to periodontitis	Yes		

Circling those conditions that apply to a specific patient helps the practitioner and caregiver understand the factors that contribute to the development and progression of periodontal diseases and pathologies. Clinical judgment may justify the use of one or more factors in determining the overall risk.

Overall assessment of the patient's risk: High Moderate Low

^α Most common examples include, but are not limited to, agranulocytosis, Chédiak-Higashi syndrome, cyclic neutropenia, diabetes, Ehlers-Danlos syndrome, human immunodeficiency virus infection, hypophosphatasia, idiopathic immune disorders, Langerhans cell histiocytosis, leukemia, leukocyte adherence deficiency, osteoporosis, neutropenia, trisomy 21, Papillon Lefèvre syndrome, plasminogen deficiency, and respiratory diseases.

Prognosis and treatment planning

Determination of the prognosis follows the diagnostic phase and is a dynamic process to be re-evaluated at all therapeutic phases (i.e., systemic, behavioral, nonsurgical, surgical, maintenance). Prognosis, based on the probability of disease progression and clinical parameters, can be categorized as favorable, questionable, unfavorable, and hopeless.¹⁴

The treatment plan is formulated after completing a comprehensive examination, establishing a diagnosis, determining

the prognosis, and identifying the individual needs and desires of the patient and caregiver. It addresses immediate, intermediate, and long-term goals to arrest or slow down the periodontal disease progression. Initial treatment plans may be subject to modifications based on unforeseen developments during care.¹⁵ Other important considerations include emergency treatment for pain or infections, need for exodontia, and esthetic demands.¹⁵

Table 2. Factors Associated with the Development and Progression of Periodontal Diseases and Pathologies for Patient ≥13 Years Old

Factors	High risk	Moderate risk	Low risk
<i>Biological factors</i>			
Systemic conditions/genetic susceptibility (e.g., family history of aggressive periodontitis) and syndromes ^α	Yes		
Immunosuppressive or radiation therapy		Yes	
Medication(s) known to affect the periodontal tissues		Yes	
History of traumatic injury to the periodontal apparatus (e.g., avulsion, luxation)		Yes	
Traumatic gingival/oral mucosal lesions		Yes	
Nutritional deficiencies		Yes	
Mental health disorders (e.g., stress, depression)		Yes	
Pregnancy		Yes	
<i>Social and behavioral factors</i>			
Socioeconomic stability (e.g., adequate health literacy, regular dental care)			Yes
Adequate daily at-home oral hygiene			Yes
Tobacco or marijuana smoking/smokeless tobacco use	Yes		
Drug abuse (e.g., crack cocaine, methamphetamine)	Yes		
Intraoral/perioral piercing and oral jewelry/accessories		Yes	
Individuals with special health care needs living in supported community (group) homes		Yes	
<i>Clinical and radiographic factors</i>			
Adequate attached gingiva and normal frenum attachments			Yes
Adequate plaque biofilm control			Yes
Tooth-related factors contributing to plaque retention		Yes	
Physical barriers for proper oral hygiene		Yes	
Generalized gingivitis (≥30% of teeth affected)		Yes	
Disproportional gingival inflammation in relation to age, amount of plaque accumulation, or oral and systemic developmental changes	Yes		
Presence of calculus	Subgingival	Supragingival	None
Bleeding on probing (% of sites)	>25	10 to 25	0 to 9
Periodontal probing depths (mm)	>5	3.5 to 5	<3.5
Chronic pericoronitis		Yes	
Abnormal tooth mobility	Yes		
Furcation involvement	Yes		
Radiographic alveolar bone loss over 25% of sites	Yes		
Tooth loss due to periodontitis	Yes		

Circling those conditions that apply to a specific patient helps the practitioner and caregiver understand the factors that contribute to the development and progression of periodontal diseases and pathologies. Clinical judgment may justify the use of one or more factors in determining the overall risk.

Overall assessment of the patient's risk: High Moderate Low

^α Most common examples include, but are not limited to, agranulocytosis, Chédiak-Higashi syndrome, cyclic neutropenia, diabetes, Ehlers-Danlos syndrome, human immunodeficiency virus infection, hypophosphatasia, idiopathic immune disorders, Langerhans cell histiocytosis, leukemia, leukocyte adherence deficiency, osteoporosis, neutropenia, trisomy 21, Papillon Lefèvre syndrome, plasminogen deficiency, and respiratory diseases.

General considerations

- A periodontal assessment includes a discussion of the chief complaint, detailed medical, dental, and social history reviews, extra- and intraoral examinations, radiographs, and periodontal probing as indicated. Further investigations (e.g., genetic, microbiological, gingival biopsy, and biochemical tests) may be needed on an individual basis to differentiate types of periodontal diseases.
- Bleeding on probing (BoP) in primary teeth during early childhood, even at a low number of sites, is indicative of high susceptibility to periodontal diseases, due to the age-dependent reactivity of the gingival tissues to plaque.^{16,17}
- Probing assessments may be initiated after the eruption of the first permanent molars and incisors and only if tolerated by the child. Pseudopockets (greater than three millimeters [mm]) may be present around partially and newly erupted teeth.¹⁸ Probing assessment on primary teeth is required before the eruption of the first permanent molars and incisors when clinical and radiographic findings indicate the presence of periodontal diseases.
- Assessing for generalized (i.e., involving 30 or more percent of the teeth) gingivitis may be performed for patients unable to undergo probing due to age, anxiety, or SHCN.¹⁹
- Alveolar bone loss in the primary dentition indicates increased susceptibility to periodontal disease.²⁰⁻²²
- Good quality bitewing radiographs are necessary for diagnosing alveolar bone loss.²²⁻²⁴ While bitewing radiographs are useful with assessing abnormal molar mobility,^{21,22,24,25} periapical radiographs may help rule out any other associated pathology (e.g., root resorption). For abnormal anterior tooth mobility, periapical radiographs are the most appropriate images.²⁶
- 1 ± 0.5 mm distance from the most coronal portion of the alveolar bone crest to the cemento-enamel junction (CEJ) is considered a normal alveolar bone height in the primary dentition,^{20,22,27} while a distance of more than two mm is considered to represent bone loss²⁰. A distance of more than two mm may be considered normal when the bone is adjacent to exfoliating primary teeth or erupting permanent teeth.²⁸
- Two-mm distance (on average, varying between 1.0 ± 3.0 mm) from the most coronal portion of the alveolar bone crest to the CEJ is considered a normal alveolar bone height in the permanent dentition.²⁴

Recommendations

- For patients in the primary dentition, a visual assessment of the gingiva should be part of every comprehensive examination. All dental radiographs should be examined for evidence of caries, alveolar bone loss, developmental anomalies, and other pathologies.
- A simplified basic periodontal examination is recommended for individuals aged seven to 17 years.¹⁸ After the eruption of the first permanent molars and incisors,

six index teeth (the first permanent molars, the permanent maxillary right central incisor, and the permanent mandibular left central incisor) are assessed for: (1) BoP; (2) presence of calculus; (3) plaque retention factors; (4) periodontal pocket depth (PPD); (5) furcation involvement; and (6) recession.

- PRA, based on a child's age and biological, social/behavioral, and clinical/radiographic factors, should be a routine component of new and periodic oral examinations.
- Practitioners may use the estimated risk level to establish a periodicity and intensity of diagnostic, counseling, and therapeutic interventions (Table 3).
- The treatment plan should be used to establish the methods and sequence of delivering periodontal treatment and include:
 - periodontal procedures to be performed;
 - medical consultation or referral for treatment when indicated;
 - consideration of diagnostic testing that may include genetic, microbiological, gingival biopsy, or biochemical tests or monitoring during the course of periodontal therapy;
 - consideration of adjunctive restorative, prosthetic, orthodontic, and/or endodontic consultation or treatment;
 - consideration of chemotherapeutic and antibiotic agents for adjunctive treatment;
 - provision for re-evaluation during and after periodontal or dental implant therapy; and
 - periodontal maintenance program.

Behavioral phase

The success of both prevention and treatment of periodontal diseases and conditions relies significantly on the ability of the patient/caregivers to comply with requested oral hygiene and dietary practices (e.g., brushing, flossing, adequate nutrition) and to change behaviors regarding harmful risk factors (e.g., smoking, drug use). Psychological models and theories of motivation (e.g., health belief model, motivational interviewing, self-determination theory) may be used to help patients adopt healthier behaviors.^{29,30}

Nutrition

The role of nutrition and, more specifically, the relevance of vitamins on periodontal health³¹⁻³³ are thought to be related to the effect on inflammation. Persistent lack of vitamin C, an essential nutrient for collagen synthesis, in the diet has been associated with more severe periodontitis.³⁴ This deficiency, known as scurvy, manifests with gingival bleeding and swelling, proceeds to tooth loss, and can result in death.

Systematic reviews show a positive association between periodontal disease and obesity in children and adolescents.³⁵⁻³⁷

Table 3. Example of Management Pathways for Periodontal Diseases and Pathologies

Risk category	Diagnostics	Counseling						Nonsurgical therapy						Surgical therapy
		Twice daily brushing and daily flossing	Healthy diet and nutrition	Injury ^α prevention	Tobacco use and drug misuse ^β	Use of oral hygiene adjuncts ^γ	Compliance with medical care and/or periodontal treatment or maintenance	Oral prophylaxis: supragingival plaque and calculus removal	Debridement, scaling and root planning	Systemic antibiotics and/or use of adjunctive topical anti-microbials	Management of plaque retentive factors ^α	Monitor previous traumatic injuries to the periodontal apparatus	Management of oral conditions and side effects from therapies, medications, infections, gingival injuries, etc.	
Low risk	<ul style="list-style-type: none"> - Recall every six to 12 months - Radiographs every 12 to 24 months 	Yes	Yes	Yes	Prevention	Yes	Compliance with medical care and/or periodontal treatment or maintenance	Every six to 12 months						
Moderate risk	<ul style="list-style-type: none"> - Recall every six months - Radiographs every six to 12 months - Monitoring of systemic conditions by laboratory analysis and consultation with medical specialists, if indicated 	Yes	Yes	Yes	Prevention or cessation	Yes	Yes	Every six months	Yes	Yes	Yes	Yes	Yes	Yes
High risk	<ul style="list-style-type: none"> - Recall every three months - Radiographs every six months 	Yes	Yes	Yes	Prevention or cessation	Yes	Yes	Every two-four months depending on disease severity and disease response to treatment	Yes	Yes	Yes	Yes	Yes	Yes

^α Plaque retentive factors include, but are not limited to, caries lesions, enamel defects, dental anatomical anomalies, malposed teeth, defective restorations, inadequate contoured crowns, orthodontic appliances, dental prostheses.

^β Prevention of injuries resultant of accidents, piercings, habits.

^γ Oral hygiene adjuncts include, but are not limited to, powered toothbrushes, interdental brushes, or oral irrigation; chemical antiplaque and anticalculus agents.

Smoking and substance misuse

The association between smoking and drug use and periodontal diseases is clear.³⁸⁻⁴³ Compelling evidence supports the significant benefits of tobacco use prevention and cessation on the periodontal and oral health in general, across all ages.⁴⁴⁻⁴⁶

Recommendations

Dental professionals should utilize psychological theories of motivation to help patients adopt healthier behaviors and counsel their pediatric patients and parents on:

- the role of diet in the development and progression of periodontal conditions;
- the harms of all tobacco products to help prevent or cease tobacco use; and
- the serious health consequences of drug misuse, as well as refer to an appropriate provider for cessation when the habit is identified.

Informed consent

Informed consent is essential in the delivery of healthcare. As part of the informed consent process, the clinician shares information and answers questions about the patient's oral health conditions and the nature, risks, and benefits of recommended and alternative treatments, including no treatment. For periodontal conditions, the discussion would also include the need for maintenance treatment due to the possibility of disease recurrence or progression. Written consent is advisable as it may decrease the liability from miscommunication, especially if risks, complications, or possibility of failure are expected with the proposed therapy. Referral is indicated when treatment needs are beyond the treating dentist's scope of practice. Patients should also be informed if referrals to other specialists are needed.⁴⁷

Nonsurgical periodontal therapy (phase I)

The major goal of phase I therapy is to control the factors responsible for periodontal inflammation; this involves educating the patient in the removal of bacterial plaque biofilm. Phase I therapy also includes scaling, root planing, and other therapies such as caries control, replacement of defective restorations, occlusal therapy, orthodontic tooth movement, and cessation of confounding habits such as tobacco use.^{7,48}

Management of bacterial plaque biofilm and calculus

Controlling gingival inflammation is the primary preventive strategy for periodontitis, as well as the secondary preventive strategy for recurrence of periodontitis.⁴⁹ A systematic review demonstrated antiplaque effectiveness for toothpastes containing stannous fluoride or chlorhexidine (CHX).^{50,51} Toothpastes containing pyrophosphates reduce the formation of new supragingival calculus,^{1,52} but no improvements have been reported in gingival inflammation and subgingival calculus. Mouthrinses with antiplaque agents significantly improve gingival inflammation and plaque levels when compared to toothpastes with such agents.⁵⁰ The use of 0.12 percent CHX

gluconate can help improve dental plaque, gingival bleeding, and gingival inflammation indices.⁵³⁻⁵⁸ Adverse effects of use (e.g., alteration in taste sensation; unpleasant taste; calculus formation; brown staining of teeth, tongue, and restorations) compromise patient acceptance^{50,51,59,60} and are most common when used for four weeks or longer^{56,57}. Rinses have higher antiplaque efficacy than sprays.⁵⁹ The CHX-containing mouthrinse may be applied via toothbrush for patients unable to spit or at risk of aspirating the agents. Different proposed regimens of CHX include: (1) once or twice a day for one week every month; and (2) once or twice a day for two weeks every three months.⁵⁵⁻⁵⁸ Preferred active agent, patient preference, economic cost, compliance, and adverse effects influence selection of a delivery system.⁵⁰ Although CHX allergy is extremely rare, prolonged exposure to CHX may lead to contact sensitization, allergic contact dermatitis or stomatitis, or even anaphylactic shock when used during surgery.⁶¹⁻⁶³

Oral prophylaxis along with scaling and root planing (SRP) are the basis of professional mechanical plaque control.^{21,48,51,64} Oral prophylaxis removes supragingival plaque and calculus via hand or powered instruments. Subgingival instrumentation, considered the gold standard of periodontal treatment, is divided into three procedures: (1) debridement (removal of subgingival plaque); (2) scaling (removal of supra- and subgingival plaque, calculus, and stains); and (3) root planing (removal of cementum or surface dentin that is rough, impregnated with calculus, or contaminated with toxins or microorganisms).⁴⁸ Supra- and subgingival instrumentation is an important component of initial and recall dental appointments. When comparing subgingival instrumentation modes, hand instruments (e.g., curettes) remove a significantly greater amount of calculus and leave a smoother root surface than ultrasonic scalers.⁵¹ On the other hand, ultrasonic devices cause less soft tissue trauma, require a shorter treatment time, and are less technique and operator sensitive.⁵¹

Recommendations

- Dental professionals should provide oral self-care instructions that are individualized and include appropriate adjuncts.
- For adolescents and individuals with SHCN who exhibit poor oral hygiene, clinicians should consider the use of chemical antiplaque agents in mouthrinses or incorporated into fluoridated toothpastes to control plaque accumulation and gingival inflammation, along with instituting more frequent recall appointments.
- Because plaque or biofilm and calculus serve as physical barriers for proper home oral hygiene execution, a dental prophylaxis and SRP should be performed at both initial and recall dental appointments when necessary.
- Use of ultrasonic devices and mouthrinses may be contraindicated for patients who are unable to expectorate and at risk for aspiration.

Management of local factors for periodontal disease and pathologies

In addition to plaque or biofilm and calculus, other local factors can contribute to plaque retention and physical barriers for proper oral hygiene execution increasing the risk of periodontal disease and pathology initiation and progression among pediatric patients.^{21,48,64-68}

Caries lesions. Caries prevention and adequate restoration of dental caries lesions are of great importance for the periodontal health of pediatric patients. Gingival inflammation is highly associated with dental caries and dependent on the degree of tooth destruction, the presence of bacteria in the biofilm, and host response.²¹ Gingivitis and interproximal alveolar bone loss have been observed in young children with severe caries.^{69,70} The alveolar bone loss occurs with extensive interproximal caries due to food impaction and biofilm retention in the interdental area.⁷⁰ Due to the dysbiotic nature of the caries-association microbiome, temporary or permanent restorations remove the reservoir of bacteria in these lesions helping to maximize the healing of the periodontal tissues.⁴⁸ Restorations with adequate proximal contour will promote healing of alveolar bone defects.⁷⁰

Defective restorations. The use of minimally-invasive restorative dentistry, when clinical conditions allow, can help avoid negative effects of restorations on the periodontal tissues. Gingivitis and clinical attachment loss (CAL) have been associated with defective restorations and crowns (i.e., subgingival restorations, margin discrepancies, overhanging restorations).⁴⁸ In addition, a study among 354 children aged six to nine years revealed radiographic interproximal alveolar bone loss adjacent to proximal surfaces in the primary molar area in 30.8 percent of the sites without an adequate amalgam restoration and 25.8 percent of the sites with inadequate crown restoration.⁷⁰ Inadequately contoured stainless steel crowns and residues of set cement remaining in contact with the gingival sulcus also may cause gingival inflammation and abnormal bone resorption.^{69,70} If meticulous oral hygiene is not maintained, interproximal lesions of posterior teeth treated with caries-arresting agents (e.g., silver diamine fluoride, silver nitrate) but not restored are capable of food impaction that can potentially cause severe gingival inflammation, bleeding, and patient discomfort.⁶⁷ Arrested cavitated lesions may benefit from receiving a restoration in order to prevent food impaction or caries lesion progression.⁷¹

Malocclusion and orthodontic appliances. An increased risk for periodontal disease has been associated with malocclusion, especially in cases of severe anterior dental crowding and gingivitis among children and adolescents wearing orthodontic appliances.^{64,65,72} Gingival overgrowth, recession, and invagination are among the most cited soft tissues changes during orthodontic treatment.⁶⁵ Due to dental plaque accumulation around appliances, patients undergoing orthodontic treatment

with deficient oral hygiene are at higher risk of developing gingival inflammation, white spot lesions, and dental caries. Inflammatory changes associated with puberty gingivitis may be exacerbated in adolescent patients undergoing orthodontic treatment.⁶⁸

Dental enamel defects and other dental anomalies. Children and adolescents with dental defects (e.g., enamel hypoplasia, amelogenesis imperfecta) may present with less ideal oral hygiene due to the sensitivity associated with the condition. Desensitizing toothpastes containing remineralization compounds, fluoride varnishes, and toothbrushes with soft bristles may minimize the sensitivity and, consequently, allow better oral hygiene.^{21,73}

Many teeth with dental defects are prone to fractures close to the gingival margin; crown-lengthening surgery is sometimes necessary to facilitate placement of restorations with cleansable margins.²¹ Other dental anomalies, such as enamel projections, enamel pearls, proximal and palatogingival grooves, and fused and supernumerary teeth, may impact periodontal health. Some of these anomalies, for instance, are associated with gingivitis and CAL due to the impediment of proper oral hygiene or mucogingival problems as a consequence of developmental aberrations in eruption and deficiencies in the thickness of the periodontium.^{64,68}

Recommendations.

- Clinicians should consider restoring open, arrested cavitated lesions when food impaction causes gingival inflammation, bleeding, or patient discomfort.
- Defective or failing restorations should be corrected by smoothing rough surfaces, removing overhangs with burs and/or hand instruments, or replacement.^{48,64}
- When placing preformed crowns, well-adapted restorations (i.e., contoured, well-fitted, and crimped) are recommended to maintain the health of the periodontium.
- Because orthodontic appliances often hinder brushing and flossing, clinicians should:
 - consider more frequent recall appointments and prophylaxis depending on home oral hygiene compliance and degree of periodontal inflammation, and
 - consider suspension of the orthodontic treatment if the patient is not able to maintain proper oral hygiene.
- In cases of sensitivity associated with dental defects, desensitizing toothpastes, fluoride varnishes, toothbrushes with soft bristles, and sealing the enamel of the teeth should be considered.

Topical antimicrobial adjuncts and systemic antibiotics

Topical (local) agents, available as fibers, gels, chips, microspheres, and solutions, are delivered directly inside the periodontal pocket and present fewer side effects than systemic agents.^{51,74-76} Compared to systemic agents, they utilize a smaller total dosage and provide higher localized concentration

of the drug, but lack the capability to reach different oral surfaces and saliva.^{51,74-76} Although systematic reviews have reported that adjunctive local antibiotics improve PPD and CAL in short-term studies and PPD in long-term studies, their use is controversial due to high cost and small magnitude of clinically-relevant benefits.^{75,76} Local antibiotic therapies have been used more commonly during the maintenance phase to treat remaining and isolated recurrent pockets.⁷⁵

SRP is effective in improving clinical parameters (e.g., BoP, PPD, CAL) for most patients with periodontitis, but not those with advanced periodontitis and deep periodontal pockets.^{51,76-78} Several clinical trials, systematic reviews, and meta-analyses support the adjunctive effect of systemic antibiotics to improve the outcomes of SRP during both non-surgical and surgical therapies.^{75,76,78-83} Systemic antibiotic therapy will be most effective if the disruption of subgingival biofilm by SRP occurs immediately before or during the antibiotic therapy.^{51,79} Stand-alone antibiotic therapy, however, is not effective in the treatment of periodontal disease.^{51,77}

Systemic antibiotics are indicated when patients exhibit moderate periodontitis with three to four mm of CAL and PPD of less than five mm.⁸² Younger patients with periodontitis characterized by rapid attachment and bone loss^{51,76-78,83}, patients with necrotizing periodontitis^{77,78}, and those with periodontitis as a manifestation of systemic conditions^{51,78,84-86} may benefit significantly from adjunctive antibiotic therapies in combination with SRP. Several factors (e.g., patient's clinical parameters, health history, dental history, drug allergy, medication compliance, personal/parental preferences, adverse effects, bacterial resistance, treatment response in primary versus permanent dentitions) influence the decision to use topical or systemic antibiotic adjuncts to SRP.^{74,79,80,87,88}

Systemic antibiotics have the advantage of reaching all oral surfaces and fluids, as well as the potential to reach periodontal pathogens that ultimately invade the host's tissues.^{76,83} In addition, antibiotic therapy may reduce bacterial endotoxins helping to minimize the local inflammatory response.^{89,90} Disadvantages of systemic administration include adverse drug effects (e.g., gastrointestinal symptoms, allergic reaction), poor patient compliance, and, very importantly, development of bacterial resistance due to indiscriminate use.^{76,83} When compared to SRP alone, the combination of amoxicillin and metronidazole (and, to a lesser degree, azithromycin and metronidazole) as an adjunctive therapy has shown to reduce the number of major periodontopathogenic bacteria, significantly improve CAL gain, and promote higher percentage of pocket closure, as well as reduce BoP, PPD, and frequency of pockets of greater than four mm.^{75,76,79-83,91-93} Regimen durations of one to two weeks have been cited in the literature with respective advantages and disadvantages.^{51,79} For patients allergic to penicillin, antibiotic regimen using metronidazole alone is an alternative treatment.⁹³ Additionally, azithromycin is effective against periodontal pathogens with positive immunomodulatory properties and has been proven effective in treating aggressive periodontitis in young patients⁹⁴ as well

as adults⁹⁵. Azithromycin is one of the safest antibiotics for patients allergic to the penicillins, but there are risks of cardiac complications including cardiotoxicity.^{96,97} Cardiac risk in pediatric patients seems to be due to an increased risk of QT prolongation associated with higher dosage levels⁹⁸, and caution should be exercised in patients with cardiac risk factors. *The Reference Manual of Pediatric Dentistry* includes information on recommended antibiotic dosage for children and adolescents, as well as for adults, available at https://www.aapd.org/globalassets/r_usefulmeds.pdf.⁹⁹ Having the child drink a small cup of grape soda immediately after ingesting liquid antimicrobials may help mask the unpleasant smell and taste of the medication and increase compliance with the antibiotic regimen.¹⁰⁰

Recommendations:

- Stand-alone antibiotic therapy is not recommended in the treatment of periodontal disease.
- Adjunctive antibiotic therapy to SRP should be considered for patients with advanced or aggressive periodontal disease.
- When adjunctive antibiotic therapy to SRP is indicated, the decision to use topical or systemic antibiotics should be carefully evaluated and based on patient's general health status, periodontal disease severity, compliance, and response to SRP.

Re-evaluation (determining success or lack of success of nonsurgical therapy)

After procedures of phase I (e.g., debridement, scaling, root planing, caries control, correction of defective restorations) are completed, the periodontal tissues will go through a process of healing that may take four or more weeks to occur.⁴⁸ Transient tissue sensitivity is often observed during the healing process and usually diminished with good home plaque or biofilm control.⁴⁸ Re-evaluation findings help determine the need for any further nonsurgical therapy procedure or periodontal surgery.⁴⁸

Recommendations:

- Components of re-evaluation appointments should include probing the periodontal tissues, examining all related anatomic structures, reinforcing home care regimens, and discussing existing harmful habits with a goal of cessation.
- The frequency of supportive periodontal therapy must be individualized and based on the patient's symptoms, clinical and radiographic findings, risk factors, initial severity of the disease, as well as residual diseased sites at the end of the active periodontal treatment in relation to the patient's age, treatment outcome, caries risk, and plaque or biofilm control.

Systemic phase

The Reference Manual of Pediatric Dentistry includes information on several genetic and nongenetic systemic diseases and pathologies associated with manifestations on periodontal

tissues.¹⁰¹ General characteristics, diagnostic criteria, clinical and radiographic findings, as well as treatment considerations are presented for some of the conditions observed in pediatric patients.

Recommendations

- Clinicians should consider systemic diseases and conditions that can affect the periodontal attachment apparatus or the course of periodontal diseases in order to achieve accurate diagnoses and plan treatment.^{84,102}
- Consultation with the patient's medical care provider may be necessary for management of at-risk patients.^{84,102}

Special management considerations

Respiratory diseases affecting the periodontium

Health of the periodontium depends on saliva's mechanical cleansing and antimicrobial properties. Respiratory diseases, either directly (e.g., mouth breathing) or through side effects (e.g., xerostomia) of therapeutic agents, may alter salivary flow.^{103,104} Nasopharyngeal obstruction from adenoid and tonsillar hypertrophy, as well as significant neuromuscular weakness with a history of snoring, can also affect periodontal health.¹⁰³ Depending on the individual oral/dental needs of patients with respiratory diseases, the pediatric dentist plays an important role in early diagnosis of general and oral health problems associated with respiratory diseases, care management, and establishment of a multidisciplinary approach that may include, but is not limited to, orthodontists, primary care providers, otolaryngologists, and speech pathologists.¹⁰³ Regular dental check-ups with oral hygiene instructions for proper home plaque control, mouth rinsing after medications, and use of fluoridated toothpaste are important preventive regimens to reduce the risk of periodontal disease and dental caries among patients with respiratory diseases.¹⁰³

Recommendations:

- Clinicians should carefully evaluate the patient's health history and medications in order to identify respiratory conditions and medications that impact salivary flow and dental and periodontal health.
- If airway obstruction is determined to affect periodontal health, an evaluation by an otolaryngologist is recommended.
- Clinicians should consider a multidisciplinary approach, referral, and/or care coordination for patients with general and/or oral health problems associated with respiratory diseases.

Oral conditions related to immunosuppressive or radiation therapies

Patients undergoing immunosuppressive or radiation therapies may present with periodontal problems associated with treatment. Gingival bleeding, soft tissue necrosis, salivary gland dysfunction, opportunistic infections (e.g., candidiasis, herpes simplex virus), and oral graft-versus-host disease are among the many acute and long-term complications associated with these therapies.¹⁰⁵⁻¹⁰⁹ Special attention should be given to

partially-erupted molars that may be at risk for pericoronitis.^{107,108} When definitive periodontal therapy cannot be rendered, extraction of hopeless periodontally-involved teeth is the treatment of choice.¹⁰⁷⁻¹⁰⁹ A periodontal assessment and appropriate therapy are indicated before patients undergoing cancer treatment receive bisphosphonates.¹⁰⁹ Refer to AAPD's *Dental Management of Pediatric Patients Receiving Immunosuppressive Therapy and/or Head and Neck Radiation*¹⁰⁹ for additional information on managing periodontal considerations in these circumstances."

Recommendations:

- Clinicians should work closely with the patient and his caregivers, as well as with his multidisciplinary health care team, to ensure that any medically-necessary dental treatment is integrated, coordinated, and delivered in a timely and safe manner before, during, and after immunosuppression or radiation therapy.¹⁰⁵

Drug-influenced gingival enlargements

Drug-influenced gingival enlargements have been associated with three types of medications: anticonvulsants (e.g., phenytoin, sodium valproate), calcium channel blockers (e.g., verapamil, diltiazem), and immunosuppressants (e.g., cyclosporine).^{19,111,112} In most cases, the gingival enlargement is induced by the combination of the drugs (i.e., fibrotic aspect) and the bacterial biofilm (i.e., inflammatory aspect).¹¹¹ Treatment options may include: (1) possible drug discontinuation or change; (2) biofilm control by means of home oral hygiene, use of antimicrobial agents (e.g., CHX), frequent professional cleaning and SRP, removal of plaque-retentive areas (e.g., faulty restorations); and (3) surgical removal of enlarged gingiva (e.g., gingivectomy using a scalpel or laser-assisted therapy, flap surgery, or electrosurgery).^{111,112}

Periodontal flap surgery to manage gingival enlargements is favored over gingivectomy in terms of minimizing the amount of tissue and time recurrences.¹¹¹ However, in general, gingivectomy is indicated for small areas of gingival enlargement (i.e., up to six teeth) where there is no evidence of CAL or the need for osseous surgery; while flap surgery is indicated for larger areas (i.e., more than six teeth) with evidence of CAL or the need for osseous surgery.¹¹¹ Antibiotic therapy as an adjunctive antimicrobial and anti-inflammatory agent has been proposed as another step in the management of gingival enlargements.^{111,112}

Recommendations:

- Clinicians should understand the etiology of gingival enlargements before considering the best management approach.
- Biofilm control, SRP, and timely evaluation of the initial treatment response should occur before considering surgical therapy.

Oral soft-tissue and tooth-supporting structure injuries

Orofacial trauma can result in extraoral and intraoral soft tissue injuries such as lacerations, contusions, abrasions, and

avulsions.^{113,114} Traumatic dental injuries (TDI) almost always involve the periodontal tissues which may undergo ischemia, crushing, or loss.^{21,66} Injuries to the periodontal ligament (PDL) may range from minor lacerations with dental concussion, tearing of the fibers with subluxation, to partial or complete separation with luxation or avulsion, and loosening and displacement of the tooth can occur.^{115,116} When foreign bodies (e.g., gravel, tooth fragment) may be embedded within the injured soft tissues, clinical inspection is supplemented by a soft-tissue radiograph.¹¹³ Removal of foreign bodies is necessary to avoid tissue infection, scarring, or tattooing.^{117,118} Cleansing, debridement, hemostasis, and closure are the major steps in managing soft tissue injuries with the goals to maintain tissue vascularity, enhance healing, and prevent tissue devitalization, as well as to minimize the risk of gingival recession and bone/root exposure.¹¹⁸ Reapproximated soft tissue wounds are sutured using the minimal number of small-diameter sutures.^{117,118} Because determining which wounds are tetanus prone is not possible, need for tetanus prophylaxis is based on the patient's current immunization status.¹¹⁹ A decision for antibiotic prophylaxis is based on the severity and contamination status of the tissue injury.¹²⁰

Splinting stabilizes traumatized teeth with the goals to optimize PDL reattachment and healing and to protect the teeth against further insult.^{121,122} Characteristics of an ideal splint for mobile traumatized teeth include being passive, flexible, and non-irritating to surrounding soft tissues as well as allowing for physiological tooth mobility and proper oral hygiene.^{121,122} Alveolar bone fractures require a more rigid splint with longer splinting time.¹²³

The risk of PDL healing complications is very low for concussion, subluxation, and extrusive and lateral luxation injuries and significantly more for TDI involving multiple teeth and teeth with full root development.^{115,116} The most common complications are "repair-related resorption (surface resorption), infection-related resorption (inflammatory resorption), ankylosis-related resorption (replacement resorption), marginal bone loss, and tooth loss".¹¹⁶ Ankylosis-related root resorption is an expected outcome in replanted teeth, especially with an extra-alveolar dry time longer than 60 minutes or transport medium other than one capable of maximizing the vitality of the PDL cells (e.g., milk, Hanks' Balanced Salt Solution).^{123,124}

Recommendations:

- Management of orofacial soft tissue injuries should include cleansing, debridement, establishing hemostasis, and closure of wounds in a manner that maintains tissue vascularity, enhances healing, and prevents tissue devitalization.
- The clinician should determine the need for tetanus prophylaxis based on the patient's current immunization status. When immunization status is in doubt, evaluation by a physician within 48 hours is indicated.^{117,120,123,125}

- A decision for antibiotic prophylaxis should be based on the severity and contamination status of the tissue injury.^{117,120} Because the PDL of an avulsed tooth may have been contaminated by oral or environmental bacteria, systemic prophylactic antibiotics are recommended following tooth replantation.¹²³
- Depending on the extent of the injury suffered by the periodontium, collaboration between the primary care dentist and a periodontologist may be needed to allow effective and successful clinical outcomes following dentoalveolar trauma.

Infections of bacterial, fungal, and viral origins

The gingiva may demonstrate a variety of lesions that are not caused by plaque and usually do not resolve after plaque removal.³ Infections of bacterial (e.g., necrotizing gingivitis), fungal (e.g., candidiasis), and viral (e.g., primary herpetic gingivostomatitis, recurrent intraoral herpes simplex infection) origins are some examples of nonplaque-induced gingival lesions observed in the pediatric population.⁶⁸ Successful treatment of infectious lesions requires clinicians to perform a thorough medical history appraisal, assessment of local and systemic contributing factors, and comprehensive oral examination aimed to achieve appropriate diagnoses and treatment plan. Elimination or reduction of all local and systemic risk factors that contribute to the infection initiation or progression is needed for treatment completeness, followed by close monitoring to assess treatment effectiveness, patient compliance, and risk of recurrence.

Recommendations:

- Initial therapy should focus on alleviating acute symptoms of pain and distress. This could include oral analgesics to control fever, malaise, and pain, as well as fluids to prevent dehydration.
- Antimicrobial therapy should be considered when an infection is not self-limiting or if there are frequent recurrences.

Traumatic gingival and oral mucosa lesions

Traumatic lesions can be accidental, iatrogenic, or self-inflicted and are physical (e.g., oral piercing, aggressive toothbrushing), chemical (e.g., dental materials, topical cocaine), or thermal (e.g., overheated foods and drinks) in nature.^{126,127} The appearance of the lesion (e.g., acute ulcerations vs chronic gingival defects) and a detailed history are crucial in achieving a diagnosis. Self-injury behavior (SIB) has been reported among individuals with psychiatric illnesses (e.g., personality disorders, bipolar disorder, major depression, anxiety disorders, obsessive-compulsive disorder) and congenital insensitivity to pain (e.g., familial dysautonomia), as well as a variety of developmental and intellectual disabilities (e.g., autism).¹²⁸ Gingival picking/scratching is among the most common oral SIB.¹²⁷⁻¹³² Management of self-inflicted traumatic lesions may be complicated due to lack of patient's compliance. The patient's primary care provider may help

rule out any medical reasons for SIB (e.g., otitis media, infection, pneumonia) or specific genetic disorders (e.g., Lesch-Nyhan syndrome) or determine comorbid psychiatric conditions. An approach that includes medical and behavioral specialists may be indicated. Periodontal plastic surgery (e.g., placing a graft to create or widen the attached keratinized tissue)¹³³ may be necessary for permanent gingival defects.^{127,129,131}

Recommendations:

- Management of traumatic oral lesions requires removal of the offending agent and symptomatic therapy.
- Treatment of SIB should be individualized; diagnosis and treatment of the underlying mechanism comprise the most successful approach.¹³²
- Behavior modification, pharmacotherapy, immobilization devices, oral appliances to control harmful habits, and/or psychological or psychiatric support may be beneficial.^{128,132}
- Re-evaluation and monitoring management approaches should occur while treating self-inflicted traumatic lesions.

Pericoronitis

Pericoronitis refers to an inflammatory lesion developed when food debris and bacteria are present beneath the excess flap of soft tissue surrounding partially-erupted teeth, most frequently involving mandibular third molars.¹³⁴ The pericoronal flap of soft tissue may be chronic without any symptoms; however, when acute, patients may experience severe pain, mouth opening restriction, gingival abscess, cellulitis, fever, lymphadenopathy, and presence or risk for systemic complications.¹³⁵ A rare complication is Ludwig's angina, a life-threatening condition that occurs when infection spreads to submandibular, sublingual, and submental spaces thereby compromising the patient's airway.¹³⁵ The first course of treatment for acute pericoronitis is management of infection and pain.^{134,135} Non-steroidal anti-inflammatory drugs (NSAIDs) are the analgesics of choice since the control of inflammation helps to control acute pain.¹³⁶ Patient compliance for home oral hygiene is also key for treatment success.¹³⁵ Once acute symptoms resolve, decisions can be made regarding the need for further treatment (e.g., pericoronal tissue surgery or tooth extraction).^{134,135}

Recommendations:

- Management during the acute phase should consist of^{134,135}:
 - debridement and irrigation of the pericoronal area,
 - drainage of purulence to relieve pressure,
 - occlusion evaluation to determine the need to reduce soft tissue or adjust occlusion of opposing tooth,
 - pain control using NSAIDs,
 - antibiotics if the infection is not localized or there are systemic signs and symptoms, and
 - home care plan to include oral cleaning, warm saline rinses, antiseptic agents (e.g., CHX), and sufficient fluid intake.

- After the acute phase, practitioners should^{134,135} evaluate prognosis and likelihood that the tooth involved will either erupt without complications or continue to pose a risk for pericoronitis recurrence and decide to either remove the pericoronal flap (if not removed during the acute phase) or extract the tooth to prevent recurrence.
- Ludwig's angina requires early recognition, immediate intervention (e.g., early and aggressive antibiotic therapy, surgical drainage, nutrition, hydration), and close monitoring. Due to the threat of rapid airway compromise, emergency referral to an otolaryngologist or an oral and maxillofacial surgeon should occur without delay.¹³⁷

Considerations for treatment, coordination and/or referral of care with a periodontist

Most pediatric patients will attain periodontal disease control with nonsurgical therapy and not require further surgical intervention. When PPD are greater than five mm, referral to a periodontal specialist may be indicated. Periodontal surgery may improve tooth support through pocket reduction, bone augmentation, and regeneration procedures.⁴⁸ Other considerations for referral include: (1) extent of the disease (generalized or localized periodontal involvement); (2) presence of short-rooted teeth; (3) teeth hypermobility; (4) difficulty in SRP deep pockets and furcations; (5) possibility of damage to the developing permanent successor tooth; (6) restorability and importance of particular teeth for reconstruction; (7) lack of resolution of inflammation after thorough plaque or biofilm removal and excellent SRP; (8) presence of systemic diseases and other conditions that compromise the host response; and (9) very importantly for the pediatric population, the age of the patient.⁴⁸ Younger patients, both systemically healthy and compromised, with extensive CAL are more likely to have aggressive forms of periodontitis that can be rapidly destructive necessitating timely advanced therapy. Early loss of primary teeth and bone loss visible on posterior bitewing radiographs are important indicators of aggressive forms of periodontitis that require further follow-up and/or referral.¹³⁸ The possibility of an underlying systemic disease cannot be discarded.

The treatment for periodontitis as a manifestation of systemic conditions is dependent on the systemic disorder. Two fundamental treatment differences exist: (1) patients for whom the systemic disease and a conservative periodontal treatment approach do not represent grave danger to life; and (2) patients for whom the systemic disease (e.g., hypophosphatasia, leukocyte adhesion deficiency syndrome, neutropenia) and a conservative periodontal treatment approach may represent grave danger to life. Managing the periodontal diseases in these children, even when extractions of primary teeth at an early age is the treatment of choice, is crucial since such systemic diseases may endanger the children's lives.¹³⁹⁻¹⁴²

In terms of coordination and referral of care with a periodontist, important considerations include^{143,144}:

- the primary care dentist will be working closely with the medical team, and all pertinent patient information needs to be available to the periodontist to determine the necessity of advanced periodontal therapies;
- the level and frequency of communication between the primary care dentist and the periodontist will be more than is required for healthy patients. Timely communication before and after each diagnostic and surgical appointment is essential; and
- the types and levels of behavioral and pharmacologic pain and anxiety control available in the periodontal office may not be ideal for the young patient. Seeing the patient together may help meet these needs.

Recommendations

- The treatment of periodontitis as a manifestation of systemic disease where a conservative periodontal treatment approach may represent grave danger to the child's life should include communication with the pediatrician or medical specialist, as well as a periodontist, to consider the risk and benefit of conservative periodontal treatment versus tooth extractions. Extraction may be the best treatment with a continuing periodontal infection causing severe destruction of bone and developing permanent teeth and endangering the child's life.
- The treatment of periodontitis as a manifestation of systemic disease where a conservative periodontal treatment approach does not represent grave danger to the child's life should include:
 - communication with the child's pediatrician or medical specialist about the systemic condition, its diagnosis based on the oral, laboratory and systemic findings, as well as coordination of systemic and periodontal treatments;
 - consultation, coordination, and/or referral of care with a periodontist if beyond the scope of pediatric dentistry practice;
 - nutritional evaluation and counseling;
 - assessment of traumatic gingival lesions, harmful habits, and self-injurious behavior;
 - oral prophylaxis, SRP, and individualized patient oral hygiene instruction;
 - consideration of chemical adjunctive antiplaque and anticalculus agents;
 - management of risk factors (e.g., caries lesions, defective restorations, dental trauma);
 - consideration of topical antimicrobial adjuncts and systemic antibiotics;
 - consideration of periodontal surgery for severe gingival or periodontal diseases; and
 - recall appointments based on each individual compliance and treatment achievements.

Surgical therapy (phase II)

Periodontal surgical therapy, which includes “plastic, aesthetic, resective, and regenerative procedures, becomes necessary when access for root therapy is required or correction of anatomic or morphologic defects is necessary”.¹³³ Placement of dental implants can also be part of phase II therapy. The main goals of surgical therapy are to improve prognosis of the teeth and their replacements, as well as improve aesthetics.¹³³ During this phase, the role of the primary care dentist is to provide treatment or refer/coordinate the care with a periodontal specialist when the needed treatment exceeds the practitioner's scope of practice. Prior to any surgical therapy, clinicians should provide the patient an opportunity to have questions answered and obtain written informed consent to proceed with the therapy proposed. Following are some surgical therapy considerations.

Pocket reduction surgery

The primary goal of surgical pocket reduction is to create access for professional SRP and reduce PPD.^{51,133} It is especially useful for areas with bony defects and/or with furcation involvement¹³³ and best limited for pockets depths greater than five mm⁵¹. If successful, surgery will enable the patient to perform adequate home cleaning and maintain long-term periodontal health. The most common pocket reduction surgical procedures are resective (e.g., gingivectomy, flaps) and regenerative (e.g., flaps with graphs or membranes).¹³³

Resective surgery

- **Gingivectomy.** The indication for gingivectomy in the treatment of periodontal disease is to remove the soft tissue of the pocket wall in order to create visibility and access for complete SRP. In combination with gingivoplasty (i.e., recontouring of the gingiva), gingivectomy can achieve a favorable environment for soft-tissue healing and physiological gingival contour.^{133,145} The two main advantages of gingivectomy are the ease and simplicity of this surgical procedure.¹¹¹ Due to secondary wound closure, gingivectomy procedures cause more post-operative discomfort and bleeding when compared to periodontal flap surgeries.¹¹¹ With advances in flap surgeries, gingivectomy is less utilized¹³³ but remains beneficial in the treatment of gingival enlargements and suprabony pockets when the pocket wall is firm and fibrous.^{145,146} Gingivectomy is not indicated in cases when access to bone is required, the keratinized tissue zone is narrow, aesthetics is a concern, and risk for postoperative bleeding is increased.^{111,146}
- **Flap surgery.** Periodontal flap surgery, the most widely used procedure for pocket therapy, provides great access for SRP, periodontal regeneration, and gingival and osseous resections¹³³ in moderate and deep posterior pockets. Due to esthetic concerns, nonsurgical periodontal treatment in the anterior maxillary area is preferred; however, surgery is indicated when better

visualization and SRP access are needed.^{111,133} In addition, flap surgery allows primary closure improving both wound healing and patients' postsurgical discomfort.^{133,145} Conversely, the periodontal flap approach is more technically difficult compared to gingivectomy.¹¹¹

Regenerative surgery

Periodontal regeneration aims to restore the lost periodontal tissues and their respective functions by the formation of new alveolar bone, cementum, and PDL.¹⁴⁷⁻¹⁵⁰ In addition to managing intrabony and furcation defects resultant of periodontal diseases,¹⁴⁹ regeneration may correct undesirable outcomes associated with resective surgical techniques such as loss of CAL and soft tissue recession.¹⁵¹ In cases of hopeless teeth, regeneration therapy is less costly when compared to extractions and dental implants.¹⁵² Several regeneration therapies including guided tissue regeneration and bone grafts (e.g., autogenous, allogenic, xenogenic, synthetic or alloplastic) have been studied.¹⁴⁸⁻¹⁵¹ Systematic and meta-analysis reviews have shown periodontal regeneration in intrabony defects results in shallower residual PPD and greater CAL gain than flap surgeries.^{150,151} In addition, a combination of regenerative approaches appears to be more effective when compared to regenerative monotherapies.¹⁵¹ Disadvantages of regenerative therapies include their technically-demanding surgical procedures and dependence on patients' compliance with home oral hygiene and professional maintenance care, as well as the need for longitudinal randomized clinical trials to provide more evidence regarding their long-term benefits.¹⁴⁹⁻¹⁵¹

Laser therapy

Lasers have been used successfully in several periodontal therapies such as gingivectomy/gingivoplasty, frenectomy, drug-induced gingival overgrowth reshaping, crown lengthening and exposure, depigmentation, and management of excess tissue in gummy smile and pericoronitis.^{153,154} Advantages associated with the use of lasers include better visualization during the surgical procedure due to hemostasis and coagulation, easier use than scalpels, reduced need of sutures, wound detoxification, enhanced healing, better patient acceptance, and postoperative pain control.¹⁵⁴⁻¹⁵⁷ Laser-assisted new attachment procedure (LANAP) has shown to initiate regeneration and improve clinical outcomes in the nonsurgical treatment of moderate to advanced periodontitis, as either a monotherapy or as an adjunct to SRP^{154,155}, due to its benefits of detoxification, calculus removal, minimally invasive access for SRP, and killing of periodontal pathogens.¹⁵⁴⁻¹⁵⁶ However, more data is needed to support the use of lasers as adjuncts to resective and regenerative therapies.^{155,156} The greatest risk associated with lasers is unintentional tissue necrosis due to excessive temperatures.¹⁵⁴ The use of laser in labial frenectomies has shown to be superior to scalpel regarding postoperative pain and discomfort during speech and mastication¹⁵⁷, while its use for lingual frenotomies has not shown to be superior to other techniques¹⁵⁸.

Extractions of teeth due to periodontal reasons

Extraction of periodontally-compromised teeth may be the best management for some patients. Important considerations include previous unsuccessful therapies, dental implants as an alternative, cost-effectiveness of periodontal procedures, as well as the patient's systemic health, compliance, and finances.^{148-150,152} For pediatric patients, extraction of primary teeth may be indicated if the periodontal lesion approximates the developing permanent successor, endangering the dental development.

Dental implants

The placement of dental implants in younger patients requires a carefully coordinated and multidisciplinary team approach. In general, conservative treatment is indicated for growing patients with missing teeth. Important considerations include:

- the number of missing teeth along with soft and hard tissue anatomy,
- growth and development,
- systemic conditions and psychological and behavioral maturity¹⁵⁹, and
- alternative therapies such as orthodontic and prosthetic treatments.

Assessment of growth and development is key to successful outcomes for dental implants in pediatric patients. Early placement of implants in the growing patient can result in rotation of the dental implant and infra-occlusion as the adjacent teeth continue to erupt and the jaw grows.¹⁵⁹ Patients vary considerably in their growth patterns, and individual patients may have periods of rapid and slower growth.¹⁶⁰ Thus, chronological age is not a good indicator of completion of growth. In contrast, skeletal maturation, assessed by cephalometric analysis or hand wrist radiographs, is a good determinant.¹⁶¹ While age is not the determining factor for when implants are appropriate and the evidence from long-term studies is still evolving, case reports give some indication of success.^{161,162} A general recommendation exists for the age of 15 in girls and 17 for boys for implants in the maxillary anterior region.^{143,161,162}

Recommendations

- If PPD inhibits subgingival access or anatomic/morphologic defects require correction, the clinician should inform the patient of the need for and benefits/risks of periodontal surgical therapy, as well as treatment alternatives.
- Extraction of periodontally-compromised teeth may be the best management for some patients.
- Clinicians should consider referral to a specialist when the surgical interventions are beyond their scope of practice.
- Determination for advisability and timing of implant placement must be based on the specific circumstances of the individual patient. The patient's stage of growth and development is critical to treatment success.

Maintenance phase

The long-term success of periodontal therapy outcomes is highly associated with the quality of recall maintenance.^{51,163} Following are some considerations of the maintenance therapy phase:

- determination of recall procedures (i.e., prophylaxis, periodontal maintenance).
- determination of recall interval based on risk factors and history of disease.
- use of antimicrobial adjuncts during maintenance.
- individualized home care reinforcement.
- decision to when re-enter phase I or phase II therapy.

A classic study¹⁶⁴ assessing the efficacy of a maintenance care program demonstrated that patients placed on a three-month recall maintained excellent oral hygiene parameters and stable periodontal attachment levels for two to six years following periodontal therapy, while the nonrecall control group demonstrated significant periodontal attachment loss. A 30-year outcome report¹⁶⁵ from this study¹⁶⁴ demonstrated that patients placed on an individualized maintenance program with a three- to 12-month recall interval maintained stable periodontal conditions for 30 years. A review¹⁶³ assessing predefined periodontal recall intervals conducive to periodontal health and stability concluded that evidence supports a two- to four-month recall interval for patients affected by moderate to advanced periodontal disease. Moreover, evidence supports a maintenance therapy program with at least 12-month interval recalls for patients who are periodontally healthy, are stable periodontally, or have mild forms of periodontitis.¹⁶³

Recommendations

- Clinicians should educate their patients and caregivers about the importance of supportive periodontal therapy to prevent disease relapse and provide individualized periodontal supportive care when needed.
- Every two to four months and at least every 12-month interval recalls are recommended for patients with higher and lower periodontal disease risk, respectively.

References

1. Perry DA, Takei HH, Do JH. Plaque biofilm control for the periodontal patient. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019:511-20.
2. Stenberg WV. Periodontal problems in children and adolescents. In: Nowak AJ, Christensen JR, Mabry TR, Townsend JA, Wells MH, eds. *Pediatric Dentistry Infancy Through Adolescence*. 6th ed. Philadelphia, Pa.: Elsevier; 2019:371-8.
3. Caton JG, Armitage G, Berglundh T, et al. A new classification scheme for periodontal and peri-implant diseases and conditions—Introduction and key changes from the 1999 classification. *J Periodontol* 2018;89(Suppl 1): S1-S8.
4. Alrayyes S, Hart TC. Periodontal disease in children. *Dis Mon* 2011;57(4):184-91.
5. Bouchard P, Carra MC, Boillot A, Mora F, Rangé H. Risk factors in periodontology: A conceptual framework. *J Clin Periodontol* 2017;44(2):125-31.
6. Elangovan S, Novak KF, Novak MJ. Clinical risk assessment. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019: 410-12.
7. Douglass CW. Risk assessment and management of periodontal disease. *J Am Dent Assoc* 2006;137(Suppl): 27S-32S.
8. Chapple ILC. Risk assessment in periodontal care: The principles. In: Chapple ILC, Papapanou P, eds. *Risk Assessment in Oral Health: A Concise Guide for Clinical application*. Switzerland, Ga.: Springer International; 2020:77-88.
9. Lang NP, Suvan JE, Tonetti MS. Risk factor assessment tools for the prevention of periodontitis progression a systematic review. *J Clin Periodontol* 2015;42(Suppl 16): S59-70.
10. Sai Sujai GV, Triveni VS, Barath S, Harikishan G. Periodontal risk calculator versus periodontal risk assessment. *J Pharm Bioallied Sci* 2015;7(Suppl 2):S656-9.
11. Mullins JM, Even JB, White JM. Periodontal management by risk assessment: A pragmatic approach. *J Evid Based Dent Pract* 2016;16(Suppl):91-8.
12. Trombelli L, Minenna L, Toselli L, et al. Prognostic value of a simplified method for periodontal risk assessment during supportive periodontal therapy. *J Clin Periodontol* 2017;44(1):51-7.
13. Petsos H, Arendt S, Eickholz P, Nickles K, Dannewitz B. Comparison of two different periodontal risk assessment methods with regard to their agreement: Periodontal risk assessment versus periodontal risk calculator. *J Clin Periodontol* 2020;47(8):921-32.
14. Kwok V, Caton JG. Commentary: Prognosis revisited: A system for assigning periodontal prognosis. *J Periodontol* 2007;78(11):2063-71.
15. Do JH, Takei HH, Carranza FA. The treatment plan. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019:426-30.
16. Bimstein E, Eidelman E. Morphological changes in the attached and keratinized gingiva and gingival sulcus in the mixed dentition period. A 5-year longitudinal study. *J Clin Periodontol* 1988;15(3):175-9.
17. Bimstein E, Huja PE, Ebersole JL. The potential lifespan impact of gingivitis and periodontitis in children. *J Clin Pediatr Dent* 2013;38(2):95-9.
18. Cole E, Ray-Chaudhuri A, Vaidyanathan M, Johnson J, Sood S. Simplified basic periodontal examination (BPE) in children and adolescents: A guide for general dental practitioners. *Dent Update* 2014;41(4):328-37.

References continued on the next page.

19. Murakami S, Mealey BL, Mariotti A, Chapple ILC. Dental plaque-induced gingival conditions. *J Periodontol* 2018;89(Suppl 1):S17-S27.
20. Sjödin B, Matsson L. Marginal bone loss in the primary dentition. A survey of 7-9-year-old children in Sweden. *J Clin Periodontol* 1994;21(5):313-9.
21. Drummond BK, Brosnan MG, Leichter JW. Management of periodontal health in children: Pediatric dentistry and periodontology interface. *Periodontol 2000* 2017; 74(1):158-67.
22. Bimstein E, Soskolne AW. A radiographic study of interproximal alveolar bone crest between the primary molars in children. *J Dent Child* 1988;55:348-50.
23. Bimstein E, Treasure ET, Williams SM, Dever JG. Alveolar bone loss in 5-year-old New Zealand children: Its prevalence and relationship to caries prevalence, socio-economic status and ethnic origin. *J Clin Periodontol* 1994;21(7): 447-50.
24. Al Jamal G, Al-Batayneh OB, Hamamy D. The alveolar bone height of the primary and first permanent molars in healthy 6- to 9-year-old Jordanian children. *Int J Paediatr Dent* 2011;21(2):151-9.
25. Bimstein E. Radiographic description of the distribution of aggressive periodontitis in primary teeth. *J Clin Pediatr Dent* 2018;42(2):91-4.
26. Tugnait A, Clerhugh V, Hirschmann PN. The usefulness of radiographs in diagnosis and management of periodontal diseases: A review. *J Dent* 2000;28(4):219-26.
27. Needleman HL, Ku TC, Nelson L, Allred E, Seow WK. Alveolar bone height of primary and first permanent molars in healthy seven- to nine-year-old children. *J Dent Child* 1997;64:188-96.
28. Bimstein E, Matsson L. Growth and development considerations in the diagnosis of gingivitis and periodontitis in children. *Pediatr Dent* 1999;21(3):186-91.
29. Renz ANPJ, Newton JT. Changing the behavior of patients with periodontitis. *Periodontol 2000* 2009;51: 252-68.
30. Chang CP, Barker JC, Hoeft KS, Guerra C, Chung LH, Burke NJ. Importance of content and format of oral health instruction to low-income Mexican immigrant parents: A qualitative study. *Pediatr Dent* 2018;40(1): 30-6.
31. Najeeb S, Zafar MS, Khurshid Z, Zohaib S, Almas K. The role of nutrition in periodontal health: An update. *Nutrients* 2016;8(9):530.
32. Valera-López A, Navarro-Hortal MD, Giamperi F, Bullón O, Battino M, Quiles JL. Nutraceuticals in periodontal health: A systematic review on the role of vitamins in periodontal health maintenance. *Molecules* 2018;23(5): 1226.
33. Cagetti MG, Wolf TG, Tennert C, Camoni N, Lingstrom P, Campus G. The role of vitamins in oral health. A systematic review and meta-analysis. *Int J Environ Res Public Health* 2020;17:938.
34. Luo PP, Xu HS, Chen YW, Wu SP. Periodontal disease severity is associated with micronutrient intake. *Aust Den J* 2018;63(2):193-201.
35. Li L, Wong HM, Sun L, Wen YF, McGrath CP. Anthropometric measurements and periodontal diseases in children and adolescents: A systematic review and meta-analysis. *Adv Nutr* 2015;6(6):828-41.
36. Martens L, Smet SD, Yusof MYPM, Rajasekharan S. Association between overweight/obesity and periodontal disease in children and adolescents: A systematic review and meta-analysis. *Eur Arch Paediatr Dent* 2017;18(2): 69-82.
37. Klokkevold PR, Mealey BL. Influence of systemic conditions. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019: 208-24.
38. Goultshin J, Cohen HD, Donchin M, Brayer L, Soskolne WA. Association of smoking with periodontal treatment needs. *J Periodontol* 1990;61(6):364-7.
39. Akef J, Weine FS, Weisman DP. The role of smoking in the progression of periodontal disease: A literature review. *Compendium* 1992;13(6):526, 528-31.
40. Machuca G, Rosales I, Lacalle JR, et al. Effect of cigarette smoking on periodontal status of healthy young adults. *J Periodontol* 2000;71(1):73-8.
41. Bergström J, Eliasson S, Dock J. Exposure to tobacco smoking and periodontal health. *J Clin Periodontol* 2000; 27(1):61-8.
42. Antoniazzi RP, Zanatta FB, Rösing CK, Feldens CA. Association among periodontitis and the use of crack cocaine and other illicit drugs. *J Periodontol* 2016;87(12): 1396-405.
43. Shariff JA, Ahluwalia KP, Papapanou PN. Relationship between frequent recreational cannabis (marijuana and hashish) use and periodontitis in adults in the United States: National Health and Nutrition Examination Survey 2011 to 2012. *J Periodontol* 2017;88(3):273-80.
44. Shibly O, Cummings KM, Zambon JJ. Resolution of oral lesions after tobacco cessation. *J Periodontol* 2008; 79(9):1797-801.
45. Warnakulasuriya S, Dietrich T, Bornstein MM, et al. Oral health risks of tobacco use and effect of cessation. *Int Dent J* 2010;60(1):7-30.
46. Chaffee BW, Couch ET, Ryder MI. The tobacco-using periodontal patient: Role of the dental practitioner in tobacco cessation and periodontal disease management. *Periodontol 2000* 2016;71(1):52-64.
47. Klokkevold PR, Takei HH, Carranza FA. General principles of periodontal surgery. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019:599-608.
48. Takei HH. Phase I periodontal therapy. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019:506-10.

49. Trombelli L, Farina R, Silva CO, Tatakis DN. Plaque induced gingivitis: Case definition and diagnostic considerations. *J Periodontol* 2018;89(Suppl 1):S46-S73.
50. Chapple IL, Van der Weijden F, Doerfer C, et al. Primary prevention of periodontitis: Managing gingivitis. *J Clin Periodontol* 2015;42(Suppl 16):S71-6.
51. Graziani F, Karapetsa D, Alonso B, Herrera D. Non-surgical and surgical treatment of periodontitis: How many options for one disease? *Periodontol 2000* 2017;75(1):152-88.
52. Sambunjak D, Nickerson JW, Poklepovic T, et al. Flossing for the management of periodontal diseases and dental caries in adults. *Cochrane Database Syst Rev* 2011;(12):CD008829.
53. Montiel-Company JM, Almerich-Silla JM. Efficacy of two antiplaque and antigingivitis treatments in a group of young mentally retarded patients. *Med Oral* 2002;7:136-43.
54. Clavero J, Baca P, Junco P, Gonzalez MP. Effects of 0.2% chlorhexidine spray applied once or twice daily on plaque accumulation and gingival inflammation in a geriatric population. *J Clin Periodontol* 2003;30(9):773-7.
55. Featherstone JD, White JM, Hoover CI, et al. A randomized clinical trial of anticaries therapies targeted according to risk assessment (caries management by risk assessment). *Caries Res* 2012;46(2):118-29.
56. Varoni E, Tarce M, Lodi G, Carrassi A. Chlorhexidine (CHX) in dentistry: State of the art. *Minerva Stomatol* 2012;61(9):399-419.
57. James P, Worthington HV, Parnell C, et al. Chlorhexidine mouthrinse as an adjunctive treatment for gingival health. *Cochrane Database Syst Rev* 2017;3(3):CD008676.
58. Featherstone JDB, Chaffee BW. The evidence for caries management by risk assessment (CAMBRA®). *Adv Dent Res* 2018;29(1):9-14.
59. Zhang J, Ab Malik N, McGrath C, Lam O. The effect of antiseptic oral sprays on dental plaque and gingival inflammation: A systematic review and meta-analysis. *Int J Dent Hyg* 2019;17(1):16-2.
60. Quirynen M, Laleman I, de Geest S, de Hous C, Dekeyser C, Teughels W. Breath malodor. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019:521-30.
61. Liippo J, Kousa P, Lammintausta K. The relevance of chlorhexidine contact allergy. *Contact Dermatitis* 2011;64(4):229-34.
62. Pemberton NN, Gibson J. Chlorhexidine and hypersensitivity reactions in dentistry. *Br Dent J* 2012;213(11):547-50.
63. Chiewchalernsri C, Sompornrattanaphan M, Wongsa C, Thongngarm T. Chlorhexidine allergy: Current challenges and future prospects. *J Asthma Allergy* 2020;9(13):127-33.
64. Clerehugh V, Tugnait A. Diagnosis and management of periodontal diseases in children and adolescents. *Periodontol 2000* 2001;26:146-68.
65. Gorbunkova A, Pagni G, Brizhak A, Farronato G, Rasperini G. Impact of orthodontic treatment on periodontal tissues: A narrative review of multidisciplinary literature. *Int J Dent* 2016;2016:4723589.
66. Yu CY, Abbott PV. Responses of the pulp, periradicular, and soft tissues following trauma to the permanent teeth. *Aust Dent J* 2016;61(Suppl 1):39-58.
67. Kanellis MJ, Owais AI, Warren JJ, et al. Managing caries in the primary dentition with silver nitrate: Lessons learned from a clinical trial. *J Calif Dent Assoc* 2018;46(1):37-44.
68. Silva DR, Law CS, Duperon DF, Carranza FA. Gingival disease in childhood. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019:277-86.
69. Bimstein E. Frequency of alveolar bone loss adjacent to proximal caries in the primary molars and healing due to restoration of the teeth. *Pediatr Dent* 1992;14(1):30-3.
70. Bimstein E, Zaidenberg R, Soskolne AW. Alveolar bone loss and restorative dentistry in the primary molars. *J Clin Pediatr Dent* 1996;21(1):51-4.
71. Ng MW, Sulyanto R. Chronic disease management of caries in children and the role of silver diamine fluoride. *J Calif Dent Assoc* 2018;46(1):23-34.
72. Bernhardt O, Krey KF, Daboul A, et al. New insights in the link between malocclusion and periodontal disease. *J Clin Periodontol* 2019;46(2):144-59.
73. Elhennawy K, Schwendicke F. Managing molar-incisor hypomineralization: A systematic review. *J Dent* 2016;55:16-24.
74. Kinane DF, Stathopoulou PG, Papapanou PN. Periodontal diseases. *Nat Rev Dis Primers* 2017;3:17038.
75. Herrera D, Matesanz P, Martín C, Oud V, Feres M, Teughels W. Adjunctive effect of locally delivered antimicrobials in periodontitis therapy: A systematic review and meta-analysis. *J Clin Periodontol* 2020;47(Suppl 22):239-56.
76. Feres M, Figueiredo LC, Soares GM, Faveri M. Systemic antibiotics in the treatment of periodontitis. *Periodontol 2000* 2015;67(1):131-86.
77. Drisko CL. Periodontal debridement: Still the treatment of choice. *J Evid Based Dent Pract* 2014;14(Suppl):33-41.e1.
78. Dar-Odeh N, Fadel HT, Abu-Hammad S, Abdeljawad R, Abu-Hammad OA. Antibiotic prescribing for orofacial infections in the paediatric outpatient: A review. *Antibiotics (Basel)* 2018;7(2):38.
79. Keestra JA, Grosjean I, Coucke W, Quirynen M, Teughels WJ. Non-surgical periodontal therapy with systemic antibiotics in patients with untreated chronic periodontitis: A systematic review and meta-analysis. *Periodontol Res* 2015;50(3):294-314.

References continued on the next page.

80. Miller KA, Branco-de-Almeida LS, Wolf S, et al. Long-term clinical response to treatment and maintenance of localized aggressive periodontitis: A cohort study. *J Clin Periodontol* 2017;44(2):158-68.
81. Nibali L, Koidou VP, Hamborg T, Donos N. Empirical or microbiologically guided systemic antimicrobials as adjuncts to non-surgical periodontal therapy? A systematic review. *J Clin Periodontol* 2019;46(10):999-1012.
82. Pretzl B, Sälzer S, Ehmke B, et al. Administration of systemic antibiotics during non-surgical periodontal therapy –A consensus report. *Clin Oral Investig* 2019;23(7):3073-85.
83. Teughels W, Feres M, Oud V, Martín C, Matesanz P, Herrera D. Adjunctive effect of systemic antimicrobials in periodontitis therapy: A systematic review and meta-analysis. *J Clin Periodontol* 2020;47(Suppl 22):257-81.
84. Albandar JM, Susin C, Hughes FJ. Manifestations of systemic diseases and conditions that affect the periodontal attachment apparatus: Case definitions and diagnostic considerations. *J Periodontol* 2018;89(Suppl 1):S183-S203.
85. Giannetti L, Roberto Apponi R, Dello Diago AM, Jafferany M, Goldust M, Sadoughifar R. Papillon- Lefèvre syndrome: Oral aspects and treatment. *Dermatol Ther* 2020;33(3):e13336.
86. Moghaddasi M, Ghassemi M, Shekari Yazdi M, Habibi SAH, Mohebi N, Goodarzi A. The first case report of Haim Munk disease with neurological manifestations and literature review. *Clin Case Rep* 2021;9(9):e04802.
87. Merchant SN, Vovk A, Kalash D, et al. Localized aggressive periodontitis treatment response in primary and permanent dentitions. *J Periodontol* 2014;85(12):1722-9.
88. Montenegro SCL, Retamal-Valdes B, Bueno-Silva B, et al. Do patients with aggressive and chronic periodontitis exhibit specific differences in the subgingival microbial composition? A systematic review. *J Periodontol* 2020;91(11):1503-20.
89. Kalash D, Vovk A, Huang H, Aukhil I, Wallet SM, Shad-dox LM. Influence of periodontal therapy on systemic lipopolysaccharides in children with localized aggressive periodontitis. *Pediatr Dent* 2015;37(5):35-40.
90. Allin N, Cruz-Almeida Y, Velsko I, et al. Inflammatory response influences treatment of localized aggressive periodontitis. *J Dent Res* 2016;95(6):635-41.
91. Sgolastra F, Petrucci A, Gatto R, Monaco A. Effectiveness of systemic amoxicillin/metronidazole as an adjunctive therapy to full-mouth scaling and root planing in the treatment of aggressive periodontitis: A systematic review and meta-analysis. *J Periodontol* 2012;83(6):731-43.
92. Sgolastra F, Severino M, Petrucci A, Gatto R, Monaco A. Effectiveness of metronidazole as an adjunct to scaling and root planing in the treatment of chronic periodontitis: A systematic review and meta-analysis. *J Periodontol* 2014;49(1):10-9.
93. Rabelo CC, Feres M, Gonçalves C, et al. Systemic antibiotics in the treatment of aggressive periodontitis. A systematic review and a Bayesian Network meta-analysis. *J Clin Periodontol* 2015;42(7):647-57.
94. Haas AN, de Castro GD, Moreno T, et al. Azithromycin as an adjunctive treatment of aggressive periodontitis: 12-months randomized clinical trial. *J Clin Periodontol* 2008;35(8):696-704.
95. Zhang Z, Zheng Y, Bian X. Clinical effect of azithromycin as an adjunct to non-surgical treatment of chronic periodontitis: A meta-analysis of randomized controlled clinical trials. *J Periodontol* 2016;51(3):275-83.
96. Araújo L, Demoly P. Macrolides allergy. *Curr Pharm Des* 2008;14(27):2840-62.
97. Bartold PM, du Bois AH, Gannon S, Haynes DR, Hirsch RS. Antibacterial and immunomodulatory properties of azithromycin treatment implications for periodontitis. *Inflammopharmacology* 2013;21(4):321-38.
98. Zeng L, Xu P, Choonara I, et al. Safety of azithromycin in pediatrics: A systematic review and meta-analysis. *Eur J Clin Pharmacol* 2020;76(12):1709-21.
99. American Academy of Pediatric Dentistry. Useful medications for oral conditions. *The Reference Manual of Pediatric Dentistry*. Chicago, Ill.: American Academy of Pediatric Dentistry; 2022:628-35.
100. Marek CL, Timmons SR. Antimicrobials in pediatric dentistry. In: Nowak AJ, Christensen JR, Mabry TR, Townsend JA, Wells MH, eds. *Pediatric Dentistry Infancy Through Adolescence*. 6th ed. Philadelphia, Pa.: Elsevier; 2019:128-41.
101. American Academy of Pediatric Dentistry. Systemic diseases and syndromes that affect the periodontium. *The Reference Manual of Pediatric Dentistry*. Chicago, Ill.: American Academy of Pediatric Dentistry; 2022:609-11.
102. Jepsen S, Caton JG, Albandar JM, et al. Periodontal manifestations of systemic diseases and developmental and acquired conditions: Consensus report of workgroup 3 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Periodontol* 2018;89(Suppl 1):S237-S248.
103. Ballikaya E, Dogan BG, Onay O, Tekcicek MU. Oral health status of children with mouth breathing due to adenotonsillar hypertrophy. *Int J Pediatr Otorhinolaryngol* 2018;113:11-5.
104. Moraschini V, Calasans-Maia JA, Calasans-Maia MD. Association between asthma and periodontal disease: A systematic review and meta-analysis. *J Periodontol* 2018;89(4):440-55.
105. Epstein JB, Thariat J, Bensadou R, et al. Oral complications of cancer and cancer therapy: From cancer treatment to survivorship. *CA Cancer J Clin* 2012;62(6):400-22.
106. da Fonseca M. Childhood cancer. In: Nowak AJ, Casamassimo PS, eds. *The Handbook of Pediatric Dentistry*. 5th ed. Chicago, Ill.: American Academy of Pediatric Dentistry; 2018:361-9.
107. da Fonseca M. Oral and dental care of local and systemic diseases. In: Nowak AJ, Christensen JR, Mabry TR, Townsend JA, Wells MH, eds. *Pediatric Dentistry Infancy*

- Through Adolescence. 6th ed. Philadelphia, Pa.: Elsevier; 2019:66-76.
108. Hong CH, da Fonseca M. Considerations in the pediatric population with cancer. *Dent Clin North Am* 2008; 52(1):155-8.
 109. American Academy of Pediatric Dentistry. Dental management of pediatric patients receiving immunosuppressive therapy and/or head and neck radiation. *The Reference Manual of Pediatric Dentistry*. Chicago, Ill.: American Academy of Pediatric Dentistry; 2022:507-16.
 110. Hong CHL, Hu S, Haverman T, et al. A systematic review of dental disease management in cancer patients. *Support Care Cancer* 2018;26(1):155-74.
 111. Camargo PM, Pirih FQ, Takei HH, Carranza FA. Treatment of gingival enlargement. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019:628-35.
 112. Mawardi H, Alsubhi A, Salem N, et al. Management of medication-induced gingival hyperplasia: A systematic review. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2021;131(1):62-72.
 113. Bourguignon C, Cohenca N, Lauridsen E, et al. International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 1. Fractures and luxations. *Dent Traumatol* 2020;36(4):314-30.
 114. Levin L, Day PF, Hicks L, et al. International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: General introduction. *Dent Traumatol* 2020;36(4):309-13.
 115. Hermann NV, Lauridsen E, Ahrensburg SS, Gerds TA, Andreasen JO (A). Periodontal healing complications following concussion and subluxation injuries in the permanent dentition: A longitudinal cohort study. *Dent Traumatol* 2012;28(5):386-93.
 116. Hermann NV, Lauridsen E, Ahrensburg SS, Gerds TA, Andreasen JO. Periodontal healing complications following extrusive and lateral luxation in the permanent dentition: A longitudinal cohort study. *Dent Traumatol* 2012;28(5):394-402.
 117. Andersson L, Andreasen JO. Soft tissue injuries. In: Andreasen JO, Andreasen FM, Andersson L, eds. *Textbook and Color Atlas of Traumatic Injuries to the Teeth*. 5th ed. Copenhagen, Denmark: Wiley-Blackwell; 2018:626-44.
 118. Elias H, Baur DA. Management of trauma to supporting dental structures. *Dent Clin North Am* 2009;53(4): 675-89.
 119. Rhee P, Nunley MK, Demetriades D, Velmahos G, Doucet JJ. Tetanus and trauma: A review and recommendations. *J Trauma* 2005;58(5):1082-8.
 120. Day PF, Duggal M, Nazzal H. Interventions for treating traumatised permanent front teeth: Avulsed (knocked out) and replanted. *Cochrane Database Syst Rev* 2019; 2(2):CD006542.
 121. Goswami M, Eranhikkal A. Management of traumatic dental injuries using different types of splints: A case series. *Int J Clin Pediatr Dent* 2020;13(2):199-202.
 122. Sobczak-Zagalska H, Emerich K. Best splinting methods in case of dental injury: A literature review. *J Clin Pediatr Dent* 2020;44(2):71-8.
 123. Fouad AF, Abbott PV, Tsilingaridis G, et al. International Association of Dental Traumatology guidelines for the management of traumatic dental injuries: 2. Avulsion of permanent teeth. *Dent Traumatol* 2020;36(4):331-42.
 124. Khinda VIS, Kaur G, Brar GS, Kallar S, Khurana H. Clinical and practical implications of storage media used for tooth avulsion. *Int J Clin Pediatr Dent* 2017;10(2): 158-65.
 125. Callison C, Nguyen H. Tetanus Prophylaxis. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021. PMID: 32644434.
 126. Holmstrup P, Plemons J, Meyle J. Non-plaque-induced gingival diseases. *J Periodontol* 2018;89(Suppl 1):S28-S45.
 127. Rawal SY, Claman LJ, Kalmar JR, Tatakis DN. Traumatic lesions of the gingiva: A case series. *J Periodontol* 2004; 75(5):762-9.
 128. Romer M, Dougherty NJ. Oral self-injurious behaviors in patients with developmental disabilities. *Dent Clin North Am* 2009;53(2):339-50.
 129. Krejci CB. Self-inflicted gingival injury due to habitual fingernail biting. *J Periodontol* 2000;71(6):1029-31.
 130. Medina AC, Sogbe R, Gómez-Rey AM, Mata M. Factitial oral lesions in an autistic paediatric patient. *Int J Paediatr Dent* 2003;13(2):130-7.
 131. Dilsiz A, Aydin T. Self-inflicted gingival injury due to habitual fingernail scratching: A case report with a 1-year follow up. *Eur J Dent* 2009;3(2):150-4.
 132. Malaga EG, Aguilera EMM, Eaton C, Ameerally P. Management of self-harm injuries in the maxillofacial region: A report of 2 cases and review of the literature. *Oral Maxillofac Surg* 2016;74(6):1198.e1-9.
 133. Takei HH. Phase II periodontal therapy. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019:585-9
 134. Klokkevold PR, Carranza FA. Treatment of acute gingival disease. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019: 488-92.
 135. Schmidt J, Kunderova M, Pilbauerova N, Kapitan M. A review of evidence-based recommendations for pericoronitis management and a systematic review of antibiotic prescribing for pericoronitis among dentists: Inappropriate pericoronitis treatment is a critical factor of antibiotic overuse in dentistry. *Int J Environ Res Public Health* 2021;18(13):6796.
 136. Moussa N, Ogle OE. Acute pain management. *Oral Maxillofac Surg Clin North Am* 2022;34(1):35-47.
 137. Bridwell R, Gottlieb M, Koyfman A, Long B. Diagnosis and management of Ludwig's angina: An evidence-based review. *Am J Emerg Med* 2021;41:1-5.

References continued on the next page.

138. Devi A, Narwal A, Bharti A, Kumar V. Premature loss of primary teeth with gingival erythema: An alert to dentist. *J Oral Maxillofac Pathol* 2015;19(2):271.
139. Delcourt-Debruyne EM, Boutigny HR, Hildebrand HF. Features of severe periodontal disease in a teenager with Chédiak-Higashi syndrome. *J Periodontol* 2000;71(5):816-24.
140. Lozano ML, Rivera J, Sánchez-Guiu I, Vicente V. Towards the targeted management of Chédiak-Higashi syndrome. *Orphanet J Rare Dis* 2014;9:132.
141. Thumbigere Math V, Rebouças P, Giovani PA, et al. Periodontitis in Chédiak-Higashi Syndrome: An altered immunoinflammatory response. *JDR Clin Trans Res* 2018;3(1):35-46.
142. Ajitkumar A, Yarrarapu SNS, Ramphul K. Chediak Higashi Syndrome. 2021 Oct 12. In: StatPearls [Internet]. Treasure Island, Fla.: StatPearls Publishing; 2021. Available at: "https://www.ncbi.nlm.nih.gov/books/NBK507881/". Accessed September 28, 2022.
143. Kraut RA. Dental implants for children: Creating smiles for children without teeth. *Pract Periodontics Aesthet Dent* 996;8(9):909-13.
144. Kraut R. Implants for children. In: Babbush CA, Hahn JA, Krauser JT, Rosenlicht JL, eds. *Dental Implants-E-Book: The Art and Science*. 2nd ed. Maryland Heights, Mo.: Saunders Elsevier; 2010:389-402.
145. Deas DE, Moritz AJ, Sagun RS Jr, Gruwell SF, Powell CA. Scaling and root planing vs. conservative surgery in the treatment of chronic periodontitis. *Periodontol* 2000 2016;71(1):128-39.
146. Do JH, Takei HH, Whang M, Shin K. Periodontal surgical therapy. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019:609-27.
147. Reynolds MA, Kao RT, Camargo PM, et al. Periodontal regeneration – intrabony defects: A consensus report from the AAP Regeneration Workshop. *J Periodontol* 2015;86 (2 Suppl):S105-7.
148. Larsson L, Decker AM, Nibali L, Pilipchuk SP, Berglundh T, Giannobile WV. Regenerative medicine for periodontal and peri-implant diseases. *J Dent Res* 2016;95(3):255-66.
149. Kao RT, Takei HH, Cochran DL. Periodontal regeneration and reconstructive surgery. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019:642-52.
150. Aimetti M, Fratini A, Manavella V, et al. Pocket resolution in regenerative treatment of intrabony defects with papilla preservation techniques: A systematic review and meta-analysis of randomized clinical trials. *J Clin Periodontol* 2021;48(6):843-58.
151. Stavropoulos A, Bertl K, Spinelli LM, Sculean A, Cortellini P, Tonetti M. Medium- and long-term clinical benefits of periodontal regenerative/reconstructive procedures in intrabony defects: Systematic review and network meta-analysis of randomized controlled clinical studies. *J Clin Periodontol* 2021;48(3):410-30.
152. Cortellini P, Stalpers G, Mollo A, Tonetti MS. Periodontal regeneration versus extraction and dental implant or prosthetic replacement of teeth severely compromised by attachment loss to the apex: A randomized controlled clinical trial reporting 10-year outcomes, survival analysis and mean cumulative cost of recurrence. *J Clin Periodontol* 2020;47(6):768-76.
153. Mossaad AM, Abdelrahman MA, Kotb AM, Alolayan AB, Elsayed SA. Gummy smile management using diode laser gingivectomy versus botulinum toxin injection: A prospective study. *Ann Maxillofac Surg* 2021;11(1):70-4.
154. Klokkevold PR, Butler B, Kao RT. Lasers in periodontal and peri-implant therapy. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, eds. *Newman and Carranza's Clinical Periodontology*. 13th ed. Philadelphia, Pa.: Elsevier; 2019:688-95.
155. Jha A, Gupta V, Adinarayan R. LANAP, periodontics and beyond: A review. *J Lasers Med Sci* 2018;9(2):76-81.
156. Behdin S, Monje A, Lin GH, Edwards B, Othman A, Wang HL. Effectiveness of laser application for periodontal surgical therapy: Systematic review and meta-analysis. *J Periodontol* 2015;86(12):1352-63.
157. Protásio ACR, Galvão EL, Falci SGM. Laser techniques or scalpel incision for labial frenectomy: A meta-analysis. *J Maxillofac Oral Surg* 2019;18(4):490-9.
158. Messner AH, Walsh J, Rosenfeld RM, et al. Clinical consensus statement: Ankyloglossia in children. *Otolaryngol Head Neck Surg* 2020;162(5):597-611.
159. Bohner L, Hanisch M, Kleinheinz J, Jung S. Dental implants in growing patients: A systematic review. *Br J Oral Maxillofac Surg* 2019;57(5):397-406.
160. Gross EL, Nowak AJ. The dynamics of change. In: Nowak AJ, Christensen JR, Mabry TR, Townsend JA, Wells MH, eds. *Pediatric Dentistry Infancy Through Adolescence*. 6th ed. Philadelphia, Pa.: Elsevier; 2019:181-99.
161. Kamatham R, Avisá P, Vinnakota DN, Nuvvula S. Adverse effects of implants in children and adolescents: A systematic review. *J Clin Pediatr Dent* 2019;43(2):69-77.
162. Lambert F, Botilde G, Lecloux G, Rompen E. Effectiveness of temporary implants in teenage patients: A prospective clinical trial. *Clin Oral Implants Res* 2017;28(9):1152-57.
163. Trombelli L, Simonelli A, Franceschetti G, Maietti E, Farina R. What periodontal recall interval is supported by evidence? *Periodontol* 2000 2020;84(1):124-33.
164. Axelsson P, Lindhe J. The significance of maintenance care in the treatment of periodontal disease. *J Clin Periodontol* 1981;8(4):281-94.
165. Axelsson P, Nyström B, Lindhe J. The long-term effect of a plaque control program on tooth mortality, caries and periodontal disease in adults. Results after 30 years of maintenance. *J Clin Periodontol* 2004;31(9):749-57.