Classification of Periodontal Diseases in Infants, Children, Adolescents, and Individuals with Special Health Care Needs

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Abstract

This best practice familiarizes clinicians with new classifications of periodontal and peri-implant diseases/conditions to improve their diagnoses. Three major determinants of periodontal health include microbiological determinants (e.g., plaque and biofilm), host determinants, and environmental determinants (e.g., smoking, medications, stress, and nutrition). Gingival diseases are categorized as dental plaque biofilm-induced gingivitis or non-dental-plaque-induced gingival diseases. Periodontal disease can be grouped as periodontitis, necrotizing periodontitis, and periodontitis as a manifestation of systemic conditions. An assessment of the periodontal status of pediatric patients should be part of a routine dental visit and oral examination. Bleeding on probing remains the best parameter to monitor gingival health or inflammation longitudinally, and the practice of probing should be initiated once permanent first molars are fully erupted and the child is cooperative. While destructive periodontal disease may be uncommon among children and adolescents, nearly half of all children will experience gingivitis in their later preschool years, and nearly all will by puberty.

This document was developed through a collaborative effort of the American Academy of Pediatric Dentistry Councils on Clinical Affairs and Scientific Affairs to offer updated information and guidance regarding the classification of periodontal diseases in infants, children, adolescents, and individuals with special health care needs.

KEYWORDS: BLEEDING ON PROBING; GINGIVAL; GINGIVAL DISEASES; ORAL DIAGNOSES; PERIODONTAL DISEASES; PERIODONTITIS

Purpose

The American Academy of Pediatric Dentistry (**AAPD**) recognizes that although the prevalence of destructive forms of periodontal disease is low among children and adolescents, this population can develop several forms of periodontal diseases and conditions most frequently associated with an underlying systemic or immunologic disorder.[1-4] In addition, current and early studies show that gingivitis occurs in half of the population by age of four or five years and peaks nearly to 100 percent at puberty.[3] The prevalence of gingivitis can be similar to or greater than dental caries during childhood.[1] Nevertheless, when compared to dental caries, gingivitis in children has received much less attention in understanding the long-term impact that chronic inflammation of the periodontal tissues in childhood may have on overall health of the periodontium throughout life.[1] Therefore, it is critical that pediatric dental patients receive a periodontal assessment as part of their routine dental visits. Early diagnosis ensures the greatest opportunity for successful treatment, primarily by reducing etiological factors, establishing appropriate therapeutic measures, and developing an effective periodic maintenance protocol.[2]

In 2017, the American Academy of Periodontology and the European Federation of Periodontology co-sponsored the World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions. The objective of the workshop was to update the previous disease classification established at the 1999 International Workshop for Classification of Periodontal Diseases and Conditions.[5] One of the major highlights included the recategorization of three forms of periodontitis, the development of a multidimensional staging and grading system for periodontitis, and the new classification for peri-implant diseases and conditions.[6]

The intent of this best practices document is to present an abbreviated overview of the new classification of periodontal and peri-implant diseases and conditions, including gingivitis. In addition, this document aims to emphasize the key role dentists have in diagnosing, treating and/or referring pediatric

ABBREVIATIONS

AAPD: American Academy of Pediatric Dentistry. **ADA:** American Dental Association. **BoP:** Bleeding on probing. **CAL:** Clinical attachment loss. **EPL:** Endodontic-periodontal lesions. **FDA:** Food and Drug Administration. **GH:** Gingival health. **ICD:** International Statistical Classification of Diseases and Related Health Problems. **HIV/AIDS:** Human immunodeficiency virus and acquired immune deficiency syndrome. **mm:** millimeters. **PA:** Periodontal abscess. **PPD:** Periodontal probing depth. **RBL:** Radiographic bone loss. **WHO:** World Health Organization.

Table 1. 2017 WORLD WORKSHOP ON THE CLASSIFICATION OF PERIODONTAL AND PERI-IMPLANT DISEASES AND CONDITIONS (Adapted from Caton et al.[6])

Periodontal Diseases and Conditions

Periodontal Health, Gingival Diseases and Conditions
Chapple et al. 2018 Rept
Trombelli et al. 2018 Case Definitions

Periodontal Health, Gingival Diseases and Conditions
Lang & Bartold 2018

1. Clinical gingival health on an intact periodontium
2. Clinical gingival health on a reduced periodontium
   a. Stable periodontitis patient
   b. Non-periodontitis patient

Gingivitis – Dental Biofilm-induced
Murakami et al. 2018

1. Associated with dental biofilm alone
2. Mediated by systemic or local risk factors
3. Drug-influenced gingival enlargement

Gingival Diseases – Non-dental-biofilm-induced
Holmstrup et al. 2018

1. Genetic/developmental disorders
2. Specific infections
3. Inflammatory and immune conditions
4. Reactive processes
5. Neoplasms
6. Endocrine, nutritional & metabolic diseases
7. Traumatic lesions
8. Gingival pigmentation

Periodontitis
Papapanou et al. 2018 Consensus Rept
Jepsen et al. 2018 Consensus Rept
Tonetti et al. 2018 Case Definitions

Necrotizing Periodontal Diseases
Herrera et al. 2018

1. Necrotizing gingivitis
2. Necrotizing periodontitis
3. Necrotizing stomatitis

Periodontitis as Manifestations of Systemic Diseases
Jepsen et al. 2018 Consensus Rept
Albandar et al. 2018

Classification of these conditions should be based on the primary systemic disease according to the International Statistical Classification of Diseases and Related Health Problems (ICD) codes

Periodontitis
Fine et al. 2018
Needleman et al. 2018
Billings et al. 2018

1. Stages: Based on severity and complexity of management
   Stage I: Initial periodontitis
   Stage II: Moderate periodontitis
   Stage III: Severe periodontitis with potential for additional tooth loss
   Stage IV: Severe periodontitis with potential for loss of the dentition
2. Extent and distribution: localized; generalized; molar-incisor distribution
3. Grades: Evidence or risk of rapid progression, anticipated treatment response
   a. Grade A: Slow rate
   b. Grade B: Moderate rate of progression
   c. Grade C: Rapid rate of progression

Other Conditions Affecting the Periodontium
Jepsen et al. 2018 Consensus Rept
Papapanou et al. 2018 Consensus Rept

Systemic Diseases or Conditions Affecting the Periodontal Supporting Tissues
Albandar et al. 2018

Periodontal Abscesses and Endodontic-Periodontal Lesions
Papapanou et al. 2018
Herrera et al. 2018

Mucogingival Deformities and Conditions
Cortellini & Bissada 2018

1. Gingival phenotype
2. Gingival/soft tissue recession
3. Lack of gingiva
4. Decreased vestibular depth
5. Aberrant frenum/muscle position
6. Gingival excess
7. Abnormal color
8. Condition of the exposed root surface

Traumatic Occlusal Forces
Fan & Caton 2018

1. Primary occlusal trauma
2. Secondary occlusal trauma
3. Orthodontic forces

Tooth and Prosthesis-related Factors
Ercoli & Caton 2018

1. Localized tooth-related factors
2. Localized dental prostheses-related factors

Periodontal Diseases and Conditions (Adapted from Berglundh and Armitage et al.[9])

Peri-implant Health
Araujo & Lindhe 2018

Peri-implant Mucositis
Heitz-Mayfield & Salvi 2018

Peri-implantitis
Schwarz et al. 2018

Peri-implant Soft and Hard Tissue Deficiencies
Hammerle & Tarnow 2018

patients and those medically compromised or with special health care needs affected by periodontal problems. A comprehensive review of the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions including the rationale, criteria, and implementation of the new classifications, is available in the June 2018 *Journal of Periodontology* (Table 1).[6-28]

Methods

This document presents an abbreviated overview of the new classification of periodontal and peri-implant diseases and conditions.[6-28] In addition to reviewing the proceeding papers from the 2017 World Workshop, an electronic search was conducted using PubMed®/MEDLINE using the terms: periodontal health AND children, periodontal health AND adolescents, gingival disease AND children, gingival disease AND adolescents, periodontal disease AND children, periodontal disease AND adolescents, gingivitis AND prevalence, periodontitis AND prevalence, gingival disease AND prevalence, periodontal disease AND prevalence, dental plaque AND children, dental plaque AND adolescents; fields: all; limits: within the last 10 years, humans, English, and clinical trials. From this search, 1588 articles matched these criteria and were evaluated by title and/or abstract. Information from 61 papers for review was chosen from this list and from references within selected articles. When data did not appear sufficient or were inconclusive, recommendations were based upon expert and/or consensus opinion by experienced researchers and clinicians.

Background

Periodontal health, gingival diseases and conditions

Periodontal health

The World Health Organization (**WHO**) defines health as "a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity".[29] Following this framework, periodontal health is defined as the absence of clinical inflammation associated with gingivitis,

Table 2. CLASSIFICATION GINGIVAL HEALTH AND GINGIVAL DISEASE AND CONDITIONS (Adapted from Chapple et al.[11])

Periodontal Diseases and Conditions

Periodontal Health

1. Clinical health on an intact periodontium
2. Clinical gingival health on a reduced periodontium
   a. Stable periodontitis patient
   b. Non-periodontitis patient

Gingivitis—Dental Plaque-induced

1. Associated with biofilm alone
2. Mediated by systemic or local risk factors
   a. Systemic risk factors (modifying factors)
      – Smoking
      – Hyperglycemia
      – Nutritional factors
      – Pharmacological agents (prescription, non-prescription, and recreational)
      – Sex steroid hormones (puberty, menstrual cycle, pregnancy, and oral contraceptives)
      – Hematological conditions
   b. Local risk factors (predisposing factors)
      – Dental plaque biofilm retention factors (e.g., prominent restoration margins)
      – Oral dryness
3. Drug-influenced gingival enlargement

Gingival Disease—Non-dental-plaque-induced

1. Genetic/developmental disorders (e.g., hereditary gingival fibromatosis)
2. Specific infections
   a. Bacterial origin
   b. Viral origin
   c. Fungal origin
3. Inflammatory and immune conditions
   a. Hypersensitivity reactions
   b. Autoimmune diseases of skin and mucous membranes
   c. Granulomatous inflammatory lesions (e.g., orofacial granulomatosis)
4. Reactive processes (e.g., epulides)
5. Neoplasms
   a. Premalignancy
   b. Malignancy
6. Endocrine, nutritional and metabolic diseases (e.g., vitamin deficiencies)
7. Traumatic lesions
   a. Physical/mechanical trauma
   b. Chemical (toxic) burn
   c. Thermal insults
8. Gingival pigmentation
   a. Melanoplakia
   b. Smoker's melanosis
   c. Drug-induced pigmentation (antimalarials, minocycline)
   d. Amalgam tattoo

periodontitis, or any other periodontal conditions, and may include patients who have had a history of successfully treated gingivitis or periodontitis, or other periodontal conditions, and who have been and are able to maintain their dentition without signs of clinical gingival inflammation.[11] According to the WHO health framework,[29] the absence of inflammatory periodontal disease allows an individual to function normally and avoid the consequences (mental or physical) associated to present or past disease.[11]

Assessing periodontal health is important to establish a common reference point for diagnosing disease and determining therapy outcomes by practitioners.[11,21] Four levels of periodontal health have been proposed, depending on whether (1) the periodontium (attachment and bone level) is structurally and clinically sound or reduced, (2) the ability to control local and systemic modifying factors, as well as (3) the relative treatment outcomes. These levels are: (1) pristine periodontal health, characterized by total absence of clinical inflammation, and physiological immune surveillance on a periodontium with normal support; (2) clinical periodontal health, characterized by an absence or minimal levels of clinical inflammation in a periodontium with normal support; (3) periodontal disease stability, characterized as a state in which the periodontitis has been successfully treated and clinical signs of the disease do not appear to worsen in extent or severity despite the presence of a reduced periodontium; and (4) periodontal disease remission/control, characterized as a period in the course of disease when symptoms become less severe but may not be fully resolved with a reduced periodontium (Table 2).[6,21] It should be noted that "pristine periodontal health" characterized by no attachment loss, no bleeding on probing (**BoP**), no sulcular probing greater than three millimeters (**mm**) in the permanent dentition and no redness, clinical swelling/edema or pus is a rare entity, especially among adults.[21] Therefore, minimal levels of clinical inflammation observed in "clinical periodontal health" is compatible with a patient classified as periodontally healthy.

Monitoring gingival health or inflammation is best documented by the parameter of BoP since it is considered the primary parameter to set thresholds for gingivitis and the most reliable for monitoring patients longitudinally in clinical practice.[6,21] Clinicians are encouraged to start probing regularly when the first permanent molars are fully erupted and the child is able to cooperate for this procedure in order to establish a baseline, detect early signs of periodontal disease, and prevent its progression. Probing prior to the eruption of the first permanent molars is encouraged in the presence or suspicion of any clinical and/or radiographic signs of periodontal disease. While probing, clinicians should rule out the presence of pseudopockets associated, for example, with tooth exfoliation or partially erupted teeth. For patients with special health care needs receiving dental treatment under sedation and/or general anesthesia, clinicians are encouraged to take this opportunity and perform the periodontal probing. The probing force should not exceed 0.25 Newton (light probing) in order to rule out the confounding issue of BoP induced by too much pressure, as well as unnecessary bleeding resulting from trauma.[21] When probing positioning and pressure into the sulcus/pocket are performed correctly, the patient should not feel discomfort. With regards to periodontal probing depth (**PPD**), there is strong evidence that deep pockets are not necessarily consistent with disease. Deep pockets may remain stable and uninflamed, especially in cases where patients receive long term careful supportive periodontal care and are referred to as "healthy pockets". PPD or probing attachment levels alone should not be used as evidence of gingival health or disease; rather, they should be considered in conjunction with other important clinical parameters such as BoP, as well as modifying and predisposing factors. Radiographic assessment is a critical component of clinical assessment of the periodontal tissues. Radiographically, a normal, anatomically-intact periodontium would present an intact lamina dura, no evidence of bone loss in furcation areas, and a two mm distance (on average, varying between 1.0 and 3.0 mm) from the most coronal portion of the alveolar bone crest to the cemento-enamel junction. While analyzing dental radiographs of children, it is important that clinicians not follow only on diagnosing interproximal caries lesions, but also evaluate the periodontal status, especially as the child grows older. Tooth mobility is not recommended as a clinical parameter of either periodontal health or disease status.[21]

Important differences between periodontal disease stability and periodontal disease remission/control are the ability to control for modifying factors and the therapeutic response. Stability is characterized by minimal inflammation (less than 10 percent in BoP sites), optimal therapeutic response (no probing depths greater than four mm), and lack of progressive periodontaldestruction while controlling for risk factors. Remission/control is characterized by a significant decrease in inflammation, some improvement in other clinical parameters, and stabilization of disease progression. Stability is the major treatment goal for periodontitis; however, remission/control may be the more realistically achievable therapeutic goal when it is not possible to fully control for modifying factors.[11,19,22,28]

There are three major determinants of clinical periodontal health. These include:

1. microbiological determinants
   a. supragingival plaque; and
   b. subgingival biofilm compositions.
2. host determinants
   a. local predisposing factors
      i. periodontal pockets;
      ii. dental restorations;
      iii. root anatomy;
      iv. tooth position; and
      v. crowding.
   b. systemic modifying factors
      i. host immune function;
      ii. systemic health; and
      iii. genetics.

3. environment determinants
   a. smoking;
   b. medications;
   c. stress; and
   d. nutrition.

In order to attain or maintain clinical periodontal health, clinicians should not underestimate predisposing and modifying factors for each patient and should recognize when these factors can be fully controlled or not. Predisposing factors are any agent or condition that contributes to the accumulation of dental plaque (e.g., tooth anatomy, tooth position, restorations), while modifying factors are any agent or condition that alters the way in which an individual responds to subgingival plaque accumulation (e.g., smoking, systemic conditions, medications). Many factors are determined controllable (e.g., removal of overhangs, smoking cessation, good diabetes control) while others are not (e.g., genetics, immune status, use of critical medications).[21]

Gingival health

Gingival health (**GH**) is usually associated with an inflammatory infiltrate and host response in relatively stable equilibrium.[21] GH in a patient with intact periodontium is diagnosed by (1) no probing attachment loss, (2) no radiographic bone loss (**RBL**), (3) less than three mm of PPD, and (4) less than 10 percent BoP.[11] GH can be restored following treatment of gingivitis and periodontitis. The diagnostic criteria for GH in a patient followingtreatment of gingivitis are the same as those just mentioned. These same clinical features also are observed on a reduced periodontium following successful treatment of periodontitis. A patient with a current GH status who has a history of successfully treated and stable periodontitis remains at an increased risk of recurrent periodontitis; therefore, the patient should be monitored closely to ensure optimal disease management.

Gingival diseases and conditions

Gingivitis is a reversible disease characterized by an inflammation of the gingiva that does not result in clinical attachment loss (**CAL**).[30] Gingivitis is highly prevalent among children and adolescents[11,21] and a necessary prerequisite for the development of periodontitis and progressive connective tissue attachment and bone loss.[6,22,28] Controlling gingival inflammation is considered the primary preventive strategy for periodontitis, as well as the secondary preventive strategy for recurrence of periodontitis. Even though there is a predilection of attachment loss to occur at inflamed sites of the gingiva, not all affected areas are destined to progress to periodontitis. This is because the interrelationship between health, gingivitis, and periodontitis is highly dependent on the host's susceptibility and immune-inflammatory response. Nevertheless, clinicians must understand their crucial role in ongoing management of gingivitis for their patients of all ages with and/or without a history of periodontal disease. There are broadly two categories of gingival disease and conditions: dental plaque biofilm-induced gingivitis and non-dental-plaque-induced gingival disease.

Dental plaque biofilm-induced gingivitis

During the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions, revisions of the 1999 classification system[5] for dental plaque-induced gingival diseases included four components: (1) description of the extent and severity of the gingival inflammation; (2) description of the extent and severity of gingival enlargements; (3) a reduction in gingival disease taxonomy; and (4) discussion of whether mild localized gingivitis should be considered a disease or variant of health.[22] These four components are addressed in this review.

Dental plaque biofilm-induced gingivitis usually is regarded as a localized inflammation initiated by microbial biofilm accumulation on teeth and considered one of the most common human inflammatory diseases (Table 2).[6,19] When dental plaque is not removed, gingivitis may initiate as a result of loss of symbiosis between the biofilm and the host's immune-inflammatory response. The common features of plaque-induced

Table 3. DIAGNOSTIC LOOK-UP TABLE FOR GINGIVAL HEALTH OR DENTAL PLAQUE-INDUCED GINGIVITIS IN CLINICAL PRACTICE (Adapted from Chapple et al.[11])

Intact periodontium	**Health**	**Gingivitis**
Probing attachment loss	No	No
Probing pocket depths (assuming no pseudopockets)	≤3 mm	≤3 mm
Bleeding on probing	<10%	Yes (≥10%)
Radiological bone loss	No	No
Reduced periodontium Non-periodontitis patient	**Health**	**Gingivitis**
Probing attachment loss	Yes	Yes
Probing pocket depths (all sites & assuming no pseudopockets)	≤3 mm	≤3 mm
Bleeding on probing	<10%	Yes (≥10%)
Radiological bone loss	Possible	Possible
Successfully treated stable periodontitis patient	**Health**	**Gingivitis in a patient with a history of periodontitis**
Probing attachment loss	Yes	Yes
Probing pocket depths (all sites & assuming no pseudopockets)	≤4 mm (no site ≥4 mm with BoP)	≤3 mm
Bleeding on probing	<10%	Yes (≥10%)
Radiological bone loss	Yes	Yes

J Periodontol 2018;89(Supp 1):S74-S84. John Wiley and Sons.
Available at: *"https://aap.onlinelibrary.wiley.com/doi/full/10.1002/JPER.17-0719"*.

gingivitis include (1) clinical signs and symptoms of inflammation confined to the free and attached gingiva that do not extend to the periodontal attachment (cementum, periodontal ligament and alveolar bone); (2) reversibility of the inflammation achieved by biofilm removal at and apical to the gingiva margin; (3) presence of a high bacterial plaque burden needed to initiate the inflammation; and (4) stable attachment levels on a periodontium, which may or may not have experienced a loss of attachment or alveolar bone (Table 3).[11,22,28] The diagnostic criteria for gingivitis is based on clinical features. Radiographs and probing attachment level analysis should not be used to diagnose gingivitis since they usually do not indicate loss of supporting structures. Clinical signs of inflammation include erythema, edema, heat, and loss of function. Clinical signs of gingivitis include swelling (loss of knife-edged gingival margin and blunting of papillae), redness, and bleeding and discomfort on gentle probing. Patient symptoms may include bleeding gums, metallic/altered taste, pain/soreness, halitosis, difficulty eating, appearance of swollen red gums, and reduced oral health-related quality of life.[11]Although there are no objective clinical criteria for defining gingivitis severity, the extent of gingivitis (referred as mild, moderate, and severe) can be used as a patient communication tool. The definitions of mild, moderate, and severe gingivitis continue to be a matter of professional opinion. Practitioners may define gingivitis as percentages of BoP sites (e.g., mild = < 10 percent, moderate = 10-30 percent, severe = > 30 percent sites) or based on grading (e.g., Grade 1 to 5 in 20 percent quintiles for percent sites BoP).[10] The gingival index by Löe[31] also can be used to describe intensity of gingival inflammation as mild (area with a minor change in color and little change in the texture of the tissue), moderate (area with glazing, redness, edema, enlargement, and bleeding upon probing), and severe (area of overt redness and edema with a tendency toward bleeding when touched rather than probed). Lastly, the extent or the number of gingival sites exhibiting gingival inflammation can be described as either localized (< 30 percent of the teeth are affected) or generalized (≥ 30 percent of the teeth are affected).[22]

As mentioned above, one revision from the 1999 classification system[5] was the proposal to introduce the term incipient gingivitis "where, by definition, only a few sites are affected by mild inflammation, expressed as mild redness and/or a delayed and broken line of bleeding rather than edema or an immediate unbroken line of bleeding on probing. Incipient gingivitis may be regarded as a condition that is part of a spectrum of 'clinical health,' but may rapidly become localized gingivitis if untreated."[22]

The severity, extent, and progression of plaque-induced gingivitis at specific sites or at the entire mouth vary between individuals and can be influenced by local (predisposing) and systemic (modifying) factors. Local oral factors that exacerbate plaque-induced gingivitis are those that can influence the initiation or progression of gingival inflammation by facilitating accumulation of bacterial plaque at a specific site, inhibiting daily mechanical plaque removal, and/or creating a biological niche that encourages increased plaque accumulation. Examples of plaque-induced gingivitis exacerbated by plaque biofilm retention are prominent subgingival restoration margins and certain tooth anatomies that contribute with plaque accumulation increasing the risk for gingivitis and, consequently, compromising the gingival health. Oral dryness is a clinical condition frequently associated with xerostomia, which in turn is a symptom caused by a decrease in the salivary flow (hyposalivation). Hyposalivation interferes with plaque removal, thereby increasing the risk of caries, halitosis, and gingival inflammation among other oral conditions. Xerostomia may occur as a side effect of medications such as antidepressants, antihistamines, decongestants, and antihypertensive medications. In addition, health diseases/conditions such as Sjögren's syndrome, anxiety, and poorly controlled diabetes may cause xerostomia due to hyposalivation.[11,22]

Systemic risk factors can modify the host immune inflammatory response in the presence of dental plaque biofilm resulting in exaggerated inflammatory response. Examples of systemic conditions include: (1) sex steroid hormones (e.g., puberty, pregnancy, menstrual cycle, oral contraceptives); (2) hyperglycemia; (3) leukemia; (4) malnutrition; and (5) smoking.[11,22]

Elevations in sex steroid hormones, especially, during puberty and pregnancy may modify the gingival inflammatory response and result in an exaggerated gingival inflammation in the presence of even relatively small amounts of plaque. Other factors that predispose to gingivitis in both male and female adolescents are dental caries, mouth breathing, dental crowding, and eruption of teeth. As for the use of oral contraceptives, exaggerated gingival inflammatory response to plaque is not reported in current, lower-dosage formulations as previously was observed with first generation high-dose oral contraceptives.[32-34] Although modest gingival inflammation changes have been reported during ovulation,[35-37] most women with gingival inflammation associated with menstrual cycles will present with nondetectable clinical signs of the condition.[38-40]

Hyperglycemia, hematologic malignancies (e.g., leukemia), and nutritional deficiencies also are significant systemic conditions that can negatively affect the gingival tissues. Increased incidence of chronic gingivitis and risk of periodontitis among children with poorly controlled Type 1 diabetes mellitus have been reported.[41-43] The severity of gingival inflammation may be more associated with the level of glycemic control rather than the quality of plaque control.[36-40] Hyperglycemia can alter the immune system and have a negative direct effect on periodontal cells and neutrophil activity, as well as have an indirect adverse effect by stimulating immune system cells to release inflammatory cytokines.[44,45] Early diagnosis of periodontal problems among children and adolescents with poorly controlled diabetes through periodic periodontal screenings, as well as prevention of periodontal diseases among this population, is of fundamental importance. It is worth mentioning that, in addition to gingivitis and periodontitis, xerostomia and candida

infections also are associated with diabetes.[45] Certain hematologic malignancies (e.g., leukemia) are associated with signs of excess gingival inflammation inconsistent with levels of dental plaque biofilm accumulation. Oral manifestations include gingival enlargement/bleeding, petechiae, oral ulcerations/infections, and cervical lymphadenopathy. Signs of gingival inflammation include swollen, glazed, and spongy tissues that are red to deep purple in appearance.[11,22,46,47] These oral manifestations may be either the result of direct gingiva infiltration of leukemic cells or thrombocytopenia and/or clotting-factor deficiencies. Both gingival bleeding and hyperplasia have been reported as initial oral signs and symptoms of patients with acute and chronic leukemias.[22,46,47] Through periodic clinical examinations, dentists have an opportunity for early diagnosis of such malignant diseases, as well as timely referral and, subsequently, increased chances for improved patient treatment outcomes.

The literature lacks information regarding the exact role of nutrition in the initiation and/or progression of periodontal diseases. However, the role of vitamin C (ascorbic acid) in supporting periodontal tissues due to its essential function in collagen synthesis is well-documented.[10,19] Vitamin C deficiency, or scurvy, compromises antioxidant micronutrient defenses to oxidative stress and collagen synthesis leading to weakened capillary blood vessels, consequently increasing the predisposition to gingival bleeding.[48] Nevertheless, gingival inflammation due to vitamin C deficiency may be difficult to detect clinically and indistinguishable from plaque-induced gingivitis.[22] Scurvy may occur in certain populations of pediatric interest such as infants and children from low socioeconomic families.[22]

One major change in the 2017 classification of dental plaque-induced gingival diseases was to simplify the system for the clinician and condense the catalog to include only conditions affecting the gingiva that could be clinically identified. Therefore, terms previously used such as menstrual cycle-associated gingivitis, oral contraceptive–associated gingivitis, and ascorbic acid-associated gingivitis were eliminated from the classification system because signs of these conditions were not clinically evident to the dentist.[11]

Smoking is a major lifestyle and behavioral risk factor for periodontitis mostly attributed to alterations in the microflora and/or host response.[11,22] Increased pocket depth measurements, attachment loss, and alveolar bone loss are more prevalent in smokers than nonsmokers.[49] Tobacco use is no longer classified as a habit but as a dependence to nicotine and a chronic relapsing medical disorder.[50] Smoking and smokeless tobacco use almost always are initiated and established in adolescence.[51-57] The most common tobacco products used by middle school and high school students are reported to be e-cigarettes, cigarettes, cigars, smokeless tobacco, hookahs, pipe tobacco, and bidis (unfiltered cigarettes from India).[52] However, the exposure to cannabis (marijuana) among children and adolescents has increased in the United States due to its legalization in many states.[55] Frequent cannabis use has been associated with deeper probing depths, more CAL, and increased risk of severe periodontitis.[55] Periodontitis, visible plaque, and gingival bleeding also have been reported among crack cocaine users.[56] Clinical signs associated with smokeless tobacco may include increased gingival recession and attachment loss, particularly at the sites adjacent to mucosal lesion associated with the habit.[55] Health professionals who treat adolescents and young adults should be aware of the signs of tobacco use and be able to provide counseling (or referral to an appropriate provider) regarding the serious health consequences of tobacco and drug use, as well as use brief interventions for encouragement, support, and positive reinforcement for cessation when the habit is identified.

Drug-influenced gingival enlargements occur as a side effect in patients treated with anticonvulsant drugs (e.g., phenytoin, sodium valproate), certain calcium channel–blocking drugs (e.g., nifedipine, verapamil, diltiazem, amlodipine, felodipine), immune-regulating drugs (e.g., cyclosporine), and high-dose oral contraceptives.[11,57] For drug-influenced gingival conditions to occur, the presence of plaque bacteria is needed. The onset of this condition may occur within three months of the drug use,[11] but not all individuals taking these medications are susceptible and will develop gingival overgrowth. Reports show that approximately half of the people who take phenytoin, nifedipine, or cyclosporin are affected with this condition.[57] A major consideration during the 2017 workshop was to select an easy and appropriate clinical assessment to define the extent and severity of the drug-influenced overgrowth. The extent of gingival enlargements was defined as either localized (enlargement limited to the gingiva in relation to a single tooth or group of teeth) or generalized (enlargement involves the gingiva throughout the mouth).[22] Mild gingival enlargement involves enlargement of the gingival papilla; moderate gingival enlargement involves enlargement of the gingival papilla and marginal gingiva; and severe gingival enlargement involves enlargement of the gingival papilla, gingival margin, and attached gingiva.[22] Drug-influenced gingival enlargement is not associated with attachment loss or tooth mortality.

Non-dental-plaque-induced gingival diseases

The gingiva and oral tissues may demonstrate a variety of gingival lesions that are not caused by plaque and usually do not resolve after plaque removal (Table 2).[6] However, the severity of the clinical manifestations of these lesions often is dependent upon plaque accumulation and subsequent gingival inflammation. These lesions may be manifestations of a systemic condition or medical disorder. They also may represent pathologic changes confined to the gingiva. Because oral health and systemic health are strongly interrelated, it is important that dentists and other health care providers collaborate to adequately diagnose, educate the patient about his condition, treatment plan, treat, or refer to a specialist for treatment. The current classification of non-dental-plaque-induced gingival conditions is based on the etiology of the lesions. These include: genetic/developmental disorders (e.g., hereditary gingival fibromatosis); specific infections of bacterial (e.g., necrotizing periodtal diseases, Streptococcal

gingivitis), viral (e.g., hand-foot-and-mouth disease, primary herpetic gingivostomatitis), and fungal (e.g., candidiasis) origins; inflammatory and immune conditions and lesions (e.g., hypersensitivity reactions, autoimmune disease of skin and mucous membranes); reactive processes (e.g., epulides); premalignant neoplasms (e.g., leukoplakia); malignant neoplasms (e.g., leukemia, lymphoma); traumatic lesions (e.g., physical, chemical, thermal insults); endocrine, nutritional, and metabolic diseases (e.g., vitamin deficiencies); and gingival pigmentation (e.g., amalgam tattoo). The major difference between the 1999 and 2017 classifications is the development of a more comprehensive nomenclature of non-plaque induced gingival diseases and conditions based on the primary etiology, as well as the inclusion of the International Statistical Classification of Diseases and Related Health Problems (**ICD**)–10 diagnostic codes (e.g., ICD–10 code for primary herpetic gingivostomatitis is B00.2).[6,11,19] Several of these conditions may occur in pediatric patients, as well as in those with special health care needs; therefore, they are of great interest to pediatric dentists. For a comprehensive review on this topic, the reader is encouraged to review the position paper on non-dental-plaque-induced gingival diseases by Holmstrup et al.[19] and the workshop consensus report by Chapple et al.[11]

Classification of periodontal diseases

The new classification of periodontal disease proposed in the 2017 workshop defines three distinct forms: (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. The new periodontitis classification was further characterized based on a multi-dimensional staging and grading framework system. The former indicates the disease severity and complex management, while the latter estimates the rate and likelihood of the disease progression and/or response to standard periodontal therapy taking into consideration the patient's biological features.[6,24,26] An individual case of periodontitis should be further defined using a simple matrix that describes the stage and grade of the disease[24] as seen in Table 4.

Periodontitis

Currently, evidence is insufficient to support the notion that chronic and aggressive periodontitis are two pathophysiologically distinct diseases. Due to concerns from clinicians, researchers, educators, and epidemiologists regarding their ability to properly distinguish between chronic and aggressive periodontitis, the 2017 World Workshop members proposed grouping these two previously forms of periodontitis into a single category simply referred to as periodontitis.[24,27] The clinical entity previously referred to as aggressive periodontitis due to its rapid rate of progression is now categorized as Grade C periodontitis and represents the extreme end of a continuum of disease rates.

Periodontitis is a multifactorial, microbially-associated, host-mediated inflammatory disease characterized by progressive destruction of the periodontal attachment apparatus. Loss of periodontal tissue support is the primary feature of periodontitis, which is detected as CAL by circumferential assessment of erupted teeth using a standardized periodontal probe with reference to the cemento-enamel junction. Clinically, a patient is characterized as a periodontitis case if: (1) interdental CAL is detectable at two or more nonadjacent teeth; or (2) buccal or oral CAL three or more mm with pocketing greater than three mm is detectable at two or more teeth. Furthermore, the CAL cannot be attributed to nonperiodontal causes such as: (1) gingival recession of traumatic origin; (2) dental caries extending in the cervical area of the tooth; (3) the presence of CAL on the distal aspect of a second molar and associated with malposition or extraction of a third molar; (4) an endodontic lesion draining through the marginal periodontium; and (5) the occurrence of a vertical root fracture.[24,27]

In the context of the 2017 World Workshop, three clearly different forms of periodontitis have been identified based on pathophysiology. Differential diagnosis is based on the history and the specific signs and symptoms of necrotizing periodontitis and the presence or absence of an uncommon systemic disease that definitively modify the host immune response.[6,24,27]

Evidence supports necrotizing periodontitis as a separate disease entity based on (1) distinct pathophysiology characterized by prominent bacterial invasion and ulceration of epithelium; (2) rapid and full thickness destruction of the marginal soft tissue resulting in characteristic soft and hard tissue defects; (3) obvious symptoms; and (4) faster resolution in response to specific antimicrobial treatment.[27] This painful and infectious condition should be diagnosed primarily based on its typical clinical features, which includes necrosis and ulceration in the interdental papilla, gingival bleeding, pseudomembrane formation, and halitosis.[18,24] In severe cases, bone sequestrum also may occur.[58] Pain and halitosis are observed less often among children, while systemic conditions such as fever, adenopathy, and sialorrhea (hypersalivation) are observed more frequently.[18,59] Necrotizing periodontal diseases are strongly associated with impairment of the host immune system. Predisposing factors include inadequate oral hygiene, chronic gingivitis, human immunodeficiency virus and acquired immune deficiency syndrome (**HIV/AIDS**), malnutrition, tobacco/alcohol consumption, psychological stress, and insufficient sleep among others.[24] Among children, higher risk of necrotizing periodontitis is observed in those with severe malnutrition, extreme living conditions (e.g., substandard accommodations, limited access to potable water, poor sanitary disposal system), and disease resultant from severe viral infections (e.g., HIV/AIDS, measles, chicken pox, malaria).[18,24] Although the prevalence of necrotizing periodontitis is low, it is a severe condition leading to very rapid tissue destruction that can be life-threating among compromised children.[18] For a more in-depth review of necrotizing periodontitis, readers are directed to the positional papers by Herrera et al.[18] and Tonetti et al.,[27] as well as to the consensus report by Papapanou et al.[24]

Systemic disease is defined as a disease that affects multiple organs and tissues or that affects the body as a whole.[60] Several systemic disorders and conditions can affect the course of periodontal diseases or have a negative impact on the periodontal attachment apparatus independently of dental biofilm-induced inflammation.[7,20] For some cases, the periodontal problems may be among the first signs of the disease. These disorders or conditions are grouped as periodontitis as a manifestation of systemic disease, and classification should be based on and follow the classification of the primary systemic disease according to the respective ICD codes.[6] Moreover, they can be grouped into broad categories such as genetic disorders that affect the host immune response (e.g., Down syndrome, Papillon Lefèvre, histiocytosis) or affect the connective tissues (e.g., Ehlers-Danlos syndrome, systemic lupus erythematosus); metabolic and endocrine disorders (e.g., hypophosphatasia, hypophosphatemic rickets); inflammatory conditions (e.g., epidermolysis bullosa acquisita, inflammatory bowel disease); as well as other systemic disorders (e.g., obesity, emotional stress and depression, diabetes mellitus, Langerhans cell histiocytosis, neoplasms). For a more comprehensive review of classifications, case definitions and diagnostic considerations, the reader is encouraged to read the positional paper and consensus report by Albandar et al.[7] and Jepsen et al.,[20] respectively.

The remaining clinical cases of periodontitis that do not present with the local characteristics of necrotizing periodontitis or the systemic characteristics of a rare immune disorder with a secondary manifestation of periodontitis should be diagnosed as periodontitis and be further characterized using the staging and grading system that describes clinical presentation,[6,7,18,20,24,27] (Table 4).

The concept of staging is adopted from the field of oncology that classifies staging of tumors based on baseline clinical observations of size or extent and whether it has metastasized or not.[61] Understanding the stage of the periodontal disease helps the clinician communicate with the patient the current severity and extent of the disease (localized or generalized), assess the complexities of disease management, develop a prognosis, and design an individualized treatment plan for the patient. Staging is determined by a number of variables such as PPD, CAL, amount and percentage of bone loss, presence and extent of angular bony defects and furcation involvement, tooth mobility, and tooth loss due to periodontitis.[27] Staging involves four categories: Stage I (initial periodontitis), Stage II (moderate periodontitis), Stage III (severe periodontitis – potential for tooth loss), and Stage IV (advanced periodontitis – potential for loss of dentition). Grading assesses the future risk of the periodontitis progression and anticipated treatment outcomes but also estimates the positive or negative impact that periodontitis and its treatment have on the overall health status of the patient. Grading also allows the clinician to incorporate the individual patient risk factors (e.g., smoking, uncontrolled Type 2 diabetes) into the diagnosis, which may influence the comprehensive case management. Grading includes three levels: Grade A (low risk of progression), Grade B (moderate risk of progression), and Grade C (high risk of progression). Table 4 shows the framework for staging and grading of periodontitis, as well as the criteria for periodontitis stage and grade, respectively.[27] Table 5 presents the three steps to staging and grading a patient with periodontitis.[27] For a more comprehensive description of staging and grading of periodontitis, the reader is encouraged to review an outcome workshop paper by Tonetti et al.[27] and the workshop consensus report by Papapanou et al.[24]

Other conditions affecting the periodontium

Peridontal abscesses and endodontic-periodontal lesions

Both periodontal abscesses (**PA**) and endodontic-periodontal lesions (**EPL**) share similar characteristics that differentiate them from other periodontal conditions. These include pain and discomfort requiring immediate emergency treatment, rapid onset and destruction of periodontal tissues, negative effect on the prognosis of the affected tooth, and possible severe systemic consequences.

PA are defined as acute lesions characterized by localized accumulation of pus within the gingival wall of the periodontal pocket, initiated by either bacterial invasion or foreign body impaction.[18,24] The most prominent sign associated with PA is the presence of an ovoid elevation in the gingiva along the lateral part of the root. Other signs and symptoms may include pain, tenderness and swelling of the gingiva, bleeding and suppuration on probing, deep periodontal pocket, bone loss observed radiographically, and increased tooth mobility.[18,24] Facial swelling, elevated body temperature, malaise, regional lymphadenopathy, or increased blood leukocytes are less commonly observed.[18] Etiologic factors such as pulp necrosis, periodontal infections, pericoronitis, trauma, surgery, or foreign body impaction may explain the development of PA. PA can develop in both periodontitis and nonperiodontitis patients. Of interest to pediatric dentists, PA can occur in healthy sites due to impaction of foreign bodies (e.g., dental floss, orthodontic elastic, popcorn hulls), harmful habits (e.g., nail biting, clenching), inadequate orthodontic forces, gingival enlargement, and alterations of the root surface (e.g., invaginated tooth, alterations, enamel pearls, iatrogenic perforations, vertical root fracture, external root resorption).

EPL are pathological communications between the endodontic and periodontal tissues at a given tooth that occur in either an acute or a chronic form and are classified according to the signs and symptoms that have direct impact on their prognosis and treatment (e.g., presence or absence of fractures and perforations, presence or absence of periodontitis, the extent of periodontal destruction around the affected teeth). The primary signs associated with EPL are deep periodontal pockets reaching or close to the apex and/or negative or altered response to pulp vitality tests. Other signs and symptoms may include radiographic evidence of bone loss in the apical or furcation region, spontaneous pain or pain on palpation and percussion, purulent exudate or suppuration, tooth mobility, sinus tract/fistula, and crown and/or gingival color alterations.[18,24]

Table 4. PERIODONTITIS STAGING AND GRADING (Adapted from Tonetti et al.[27])

Framework for periodontitis staging and grading		**Disease Severity and Complexity of Management**			
		Stage I: Initial periodontitis	**Stage II:** Moderate periodontitis	**Stage III:** Severe periodontitis with potential for additional tooth loss	**Stage IV:** Advanced periodontitis with extensive tooth loss and potential for loss of dentition
Evidence or risk of rapid progression, anticipated treatment response, and effects on systemic health	Grade A	**Individual Stage and Grade Assignment**			
	Grade B				
	Grade C				

Periodontitis stage		**Stage I**	**Stage II**	**Stage III**	**Stage IV**
Severity	Interdental CAL at site of greatest loss	1 to 2 mm	3 to 4 mm	≥5 mm	≥5 mm
	Radiographic bone loss	Coronal third (<15%)	Coronal third (<15% to 33%)	Extending to mid-third of root and beyond	Extending to mid-third of root and beyond
	Tooth loss	No tooth loss due to periodontitis		Tooth loss due to periodontitis of ≤4 teeth	Tooth loss due to periodontitis of ≥5 teeth
Complexity	Local	Maximum probing depth ≤4 mm Mostly horizontal bone loss	Maximum probing depth ≤5 mm Mostly horizontal bone loss	In addition to stage II complexity: – Probing depth ≥6 mm – Vertical bone loss ≤3 mm – Furcation involvement Class II or III – Moderate ridge defect	In addition to stage III complexity: Need for complex rehabilitation due to: – Masticatory dysfunction – Secondary occlusal trauma (tooth mobility degree ≥2) – Severe ridge defect – Bite collapse, drifting, flaring – Less than 20 remaining teeth (10 opposing pairs)
Extent and distribution	Add to stage as descriptor	For each stage, describe extent as localized (<30% of teeth involved), generalized, or molar/incisor pattern			

Periodontitis grade			**Grade A: Slow rate of progression**	**Grade B: Moderate rate of progression**	**Grade C: Rapid rate of progression**
Primary criteria	Direct evidence of progression	Longitudinal data (RBL or CAL)	Evidence of no loss over 5 years	<2 mm over 5 years	≥2 mm over 5 years
	Indirect evidence of progression	% bone loss/age	<0.25	0.25 to 1.0	>1.0
		Case phenotype	Heavy biofilm deposits with low levels of destruction	Destruction commensurate with biofilm deposits	Destruction exceeds expectation given biofilm deposits; specific clinical patterns suggestive of periods of rapid progression and/or early onset disease (e.g., molar/incisor pattern; lack of expected response to standard bacterial control therapies)
Grade modifiers	Risk factors	Smoking	Non-smoker	Smoker <10 cigarettes/day	Smoker ≥10 cigarettes/day
		Diabetes	Normoglycemic/no diagnosis of diabetes	HbAlc <7.0% in patients with diabetes	HbAlc ≥7.0% in patients with diabetes

Signs observed in EPL associated with traumatic and/or iatrogenic factors may include root perforation, fracture/cracking or external root resorption, commonly associated with the presence of an abscess accompanied by pain. In periodontitis patients, EPL usually presents low and chronic progression without evident symptoms. For further review on the classification, pathophysiology, microbiology, and histopathology of both PA and EPL, readers are directed to the positional paper by Herrera et al.[18] and the consensus report by Papapanou et al.[24]

Mucogingival deformities and conditions

Normal mucogingival condition is defined as the absence of pathosis such as gingival recession, gingivitis, and periodontitis. Mucogingival deformities, including gingival recession, are a group of conditions that affect a large number of patients, are observed more frequently in adults, and have a tendency to increase with age independent of the patient/s oral hygiene status. Recession is defined as an apical shift of the gingival margin caused by different conditions and pathologies that is associated with CAL in any surface (buccal/lingual/interproximal) of the teeth.[20] Although, gingival thickness has been referenced in the literature as gingival biotype, the 2017 World Workshop group strongly suggested the adoption of the term periodontal phenotype, which is determined by gingival phenotype (gingival thickness, keratinized tissue width) and bone morphotype (thickness of the buccal bone plate). Periodontal phenotype can be assessed by measuring the gingival thickness through the use of a periodontal probe. The phenotype is classified as thin when a periodontal probe inserted into the sulcus is visible through the tissue, indicating the tissue is one mm or less in thickness. If the probe is not visible through the tissue, indicating the tissue is greater than one mm thick, it is classified as a thick phenotype.[20] The development and progression of gingival recession is not associated with increased tooth mortality. However, this condition often is associated with patient esthetic concerns, dentinal hypersensitivity and carious/noncarious cervical lesions on the exposed root surface.[12,20] While lack of keratinized tissue is a predisposing factor for gingival recession and inflammation, periodontal health can be maintained despite the lack of keratinized tissues in most patients with optimal home care and professional maintenance. Conversely, patients with thin periodontal phenotypes, with inadequate oral hygiene, and requiring cervical restorative and/or orthodontic treatment are at an increased risk for gingival recession.[12,20] Monitoring specific gingival recession sites is considered a proper approach in the absence of any pathosis. However, mucogingival surgical interventions may be necessary in the presence of esthetic concerns, dentin hypersensitivity, cervical lesions, thin gingival biotypes and mucogingival deformities.

Table 5. THREE STEPS TO STAGING AND GRADING A PATIENT WITH PERIODONTITIS (Adapted from Tonetti et al.[27])

Step 1 Initial Case Overview to Assess Disease	Screen: • Full mouth probing • Full mouth radiographs • Missing teeth Mild to moderate periodontitis will typically be either Stage I or Stage II Severe to very severe periodontitis will typically be either Stage III or g IV
Step 2 Establish Stage	For mild to moderate periodontitis (typically Stage I or Stage II): • Confirm clinical attachment loss (CAL) • Rule out non-periodontitis causes of CAL (e.g., cervical restorations or caries, root fractures, CAL due to traumatic causes) • Determine maximum CAL or RBL • Conform RBL patterns For moderate to severe periodontitis (typically Stage III or Stage IV): • Determine maximum CAL or RBL • Confirm RBL patterns • Assess tooth loss due to periodontitis • Evaluate case complexity factors (e.g., severe CAL frequency, surgical challenges)
Step 3 Establish Grade	• Calculate RBL (% of root length x 100) divided by age • Assess risk factors (e.g., smoking, diabetes) • Measure response to scaling and root planning and plaque control • Assess expected rate of bone loss • Conduct detailed risk assessment • Account for medical and systemic inflammatory considerations

J Periodontol 2018;89(Supp 1):S159-S172. John Wiley and Sons.
Available at: "*https://aap.onlinelibrary.wiley.com/doi/full/10.1002/JPER.18-0006*".

Traumatic occlusal forces and occlusal trauma

Traumatic occlusal force is defined as "any occlusal force that causes an injury to the teeth and/or the periodontal attachment apparatus."[20] It may be indicated by one or more of the following: fremitus (visible tooth movement upon occlusal force), tooth mobility, thermal sensitivity, excessive occlusal wear, tooth migration, discomfort/pain on chewing, fractured teeth, radiographically widened periodontal ligament space, root resorption, and hypercementosis.[20] Occlusal trauma is a lesion in the periodontal ligament, cementum, and adjacent bone caused by traumatic occlusal forces. It may be indicated by one or more of the following: progressive tooth mobility, fremitus, radiographically widened periodontal ligament space, tooth migration, discomfort/pain on chewing, and root resorption.[20] Traumatic occlusal forces and occlusal trauma can be classified as: (1) primary occlusal trauma; (2) secondary occlusal trauma; and (3) orthodontic forces. Primary and secondary occlusal trauma have been defined as injuries resulting in tissue changes from traumatic occlusal forces, the former when applied to a tooth or teeth with normal periodontal support and the latter when applied to a tooth or teeth with reduced support.[20]

There is either little or no evidence that traumatic occlusal forces can cause periodontal attachment loss, inflammation of the periodontal ligament, noncarious cervical lesions, abfraction, or gingival recession.[14,20] Traumatic occlusal forces lead to adaptive mobility in teeth with normal support and are not progressive, while in teeth with reduced support, they lead to progressive mobility usually requiring splinting. Although, there is evidence that traumatic occlusal forces may be associated periodontitis, there is no evidence that these forces can accelerate the progression of periodontitis in humans.[20] Moreover, there is insufficient clinical evidence regarding the impact that elimination of traumatic occlusal forces may have on the response to periodontal therapies. With regards to orthodontic forces, observational studies suggest that orthodontic treatment has minimal adverse effects to the periodontal supporting apparatus, especially in patients with good plaque control and healthy periodontium.[14,20] However, noncontrolled orthodontic forces can have adverse effects such as pulpal disorders as well as root and alveolar bone resorptions.

Dental prostheses and tooth-related factors

Several conditions associated with the fabrication and presence of dental restorations and fixed prostheses, placement of orthodontic appliances, as well as tooth-related factors may facilitate the development of gingivitis and periodontitis, especially in individuals with poor compliance with home care plaque control and attendance to periodic maintenance visits.[13,20]

Tooth anatomic factors (e.g., cervical enamel projections, enamel pearls, developmental grooves), root proximity, abnormalities and traumatic dental injuries potentially altering the local anatomy of both hard and soft tissues, as well as tooth relationships in the dental arch and with the opposing dentition, are associated with dental plaque-biofilm induced gingivitis and periodontitis. Placement of restoration margins infringing within the junctional epithelium and supracrestal connective tissue attachment (biological width) also can be associated with gingival inflammation and, potentially, recession. Tooth-supported and/or tooth-retained restorations and their design, fabrication, delivery, and materials often have been associated with plaque retention and loss of periodontal supporting tissues. However, optimal restoration margins located within the gingival sulcus do not cause gingivitis if patients are compliant with self-performed plaque control and periodic maintenance care.[13,20]

The available evidence does not support that optimal removable and fixed dental prostheses are associated with periodontitis when patients perform adequate plaque control and attend maintenance appointments. However, there is evidence to suggest that removable dental prostheses can serve as plaque retentive factors and be associated with gingivitis/periodontitis, increased mobility and gingival recession in patients with poor compliance.[20] Moreover, there is evidence to suggest that design, fabrication, delivery, and materials used for fixed dental prostheses procedures can be associated with plaque retention, gingival recession, and loss of supporting periodontal tissues.[13,20]

Lastly, it is important to point out that dental materials, including commonly used appliances (e.g., stainless steel crowns, space maintainers, orthodontic appliances) may be associated with hypersensitivity reactions observed clinically as localized inflammation. If the hypersensitivity does not resolve with adequate measures of plaque control, additional treatment may be required, including removal of material or appliance. However, it appears that adequate periodontal assessment and treatment, appropriate instructions, and motivation in self-performed plaque control and compliance to periodic maintenance protocols are the most important factors to limit or avoid the potential negative effects on the periodontium caused by fixed and removable prostheses when hypersensitivity reactions are not suspected.[13]

Peri-implant diseases and conditions

The 2017 World Workshop members developed a new classification for peri-implant health, peri-implant mucositis and peri-implantitis. The case definitions were developed based on a review of the evidence applicable for diagnostic considerations for use by clinicians for both individual case management and population studies.[6,25] Because the majority of pediatric dentists are not the ones responsible for the placement of osseointegrated dental implants, the reader is encouraged to review the positional paper by Renvert et al.[25] and the consensus report by Berglundh et al.[9] for more comprehensive information about the rationale, criteria, and implementation of the new classification. Nevertheless, it is important that all clinicians are able to diagnose potential problems, complications, and failures associated with dental implants in order to either provide proper treatment or refer the patient to a specialist. Case definitions and clinical criteria of these conditions are presented below.

Peri-implant health
Clinically, peri-implant health is characterized by an absence of visual signs of inflammation such as redness, swelling, and profuse BoP, as well as an absence of further additional bone loss following initial healing. Peri-implant health can occur around implants with normal or reduced bone support.[6,25]

Peri-implant mucositis
Peri-implant mucositis is characterized by visual signs of inflammation such as redness, swelling, and line or drop of bleeding within 30 seconds following probing, combined with no additional bone loss following initial healing. There is strong evidence that peri-implant mucositis is caused by plaque, while very limited evidence for nonplaque-induced peri-implant mucositis. Peri-implant mucositis can be reversed with dental plaque removal measures.[6,25]

Peri-implantitis
Peri-implantitis is defined as a plaque-associated pathologic condition occurring in the tissue around dental implants, characterized by signs of inflammation in the peri-implant mucosa, radiographic evidence of bone loss following initial healing, increasing probing depth as compared to probing depth values after the implant placement, and subsequent progressive loss of supporting bone. In the absence of baseline radiographs, radiographic bone level three or more mm in combination with BoP and probing depths six or more mm is indicative of peri-implantitis. Peri-implantitis is preceded by peri-implant mucositis.[6,25]

Recommendations

1. Periodontal disease in children is of great interest in pediatric dentistry and a problem that should not be ignored. Therefore, it is critical that pediatric dental patients receive a periodontal assessment as part of their initial and periodic dental examinations. Early diagnosis of periodontal diseases ensures the greatest opportunity for successful treatment, primarily by reducing etiological factors, establishing appropriate therapeutic measures, and developing an effective periodic maintenance protocol.
2. Pediatric dentists are often the front line in diagnosing periodontal conditions in children and adolescents and in great position to treat or refer and coordinate, collaborate, and/or organize the patient care activities between two or more health care providers to ensure that the appropriate treatment is delivered in a timely fashion. Therefore, clinicians should become familiarized with the current classification of periodontal diseases and conditions, including gingivitis, in order to properly diagnose patients affected by these problems.
3. Monitoring gingival health or inflammation is best documented by the parameter of bleeding on probing since it is considered the primary parameter to set thresholds for gingivitis and the most reliable for monitoring patients longitudinally in clinical practice. Clinicians are encouraged to start probing regularly when the first permanent molars are fully erupted and the child is able to cooperate for this procedure in order to establish a baseline, detect early signs of periodontal disease, and prevent disease progression.
4. Probing prior to the eruption of the first permanent molars is encouraged in the presence or suspicion of any clinical and/or radiographic signs of periodontal disease. For patients with special health care needs receiving dental treatment under sedation and/or general anesthesia, clinicians are encouraged to utilize this opportunity to perform the periodontal probing.

The intent of this document was to present an abbreviated overview of the proceeding papers from the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions. Major highlights from the 2017 workshop included the recategorization of three forms of periodontitis, the development of a multidimensional staging and grading system for periodontitis, and the new classification for peri-implant diseases and conditions. A best practice document on periodontal disease therapies will be available in a future publication of *The Reference Manual of Pediatric Dentistry*.

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