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Gingival and Periodontal Diseases and Conditions in Children and Adolescents: Consensus Report

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ABSTRACT

Background: The objectives of this Focused Workshop were to update the epidemiology, aetiology, risk factors, diagnosis and management of gingival and periodontal diseases and conditions in children and adolescents, and to explore the applicability of the 2018 Classification in children and adolescents.

Methods: The Workshop discussions were informed by three specifically commissioned systematic reviews covering gingival and periodontal diseases and conditions, in systemically healthy children and adolescents, or in children and adolescents with systemic conditions.

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Results: Over 70 genetic, congenital and acquired systemic conditions that impact the periodontal tissues were identified, with levels of evidence graded as very low, low or moderate. Gingival diseases and conditions in systemically healthy children and adolescents were identified, alongside local predisposing and systemic modifying factors. Periodontitis and other periodontal conditions in the 2018 Classification System also apply to children and adolescents; however, there are challenges with periodontal probing in the primary and mixed dentition.

Conclusions: Periodontal tissues in children and adolescents differ from those in adults and require special consideration, accounting for their stage of development and predisposing and modifying factors unique to younger patients, which may confound accurate diagnosis, prognostication and management. Specific approaches to screening, examination and treatment are necessary for safe and effective management in this patient group.

1 | Introduction

Periodontal diseases in children and adolescents are common; however, the use of multiple different measures to examine and record disease and the historical lack of consistency in the use of case definitions hinder the interpretation and comparison of prevalence and incidence estimates for periodontitis in the literature. Prevalence data for gingivitis are equally challenging to interpret because of variations in indices employed to define gingivitis in studies from different countries across the world, and statistics derived from the use of the community periodontal index (CPI) in two studies vary from as low as 18% to up to 92% (Tsilingaridis et al. 2025). The 2018 International Classification System for Periodontal and Peri-implant Diseases and Conditions presented an opportunity to align case definitions in adults (Caton et al. 2018); it was the first time periodontal health had been classified, and gingivitis had been classified beyond the basic levels of dental biofilm–induced versus non-dental biofilm–induced gingivitis (Chapple et al. 2018). The associated workshop made the decision to capture the risk of periodontitis within the classification system, based upon the historical amount of attachment loss and the rate of disease progression (stage and grade), and also accommodated certain risk factors as grade modifiers. Complexity factors, such as probing depth, were also mapped to the disease stage, and the extent of disease formed the final descriptor that contributed to the classification of periodontitis (Tonetti et al. 2018).

Classification informs diagnosis, but the latter also requires the assessment of levels of inflammation and pocket depths, thus permitting current disease activity status to be determined. A periodontitis patient is designated as one for life, thus capturing their risk of further disease progression. If periodontitis is successfully treated, the patient is regarded as 'stable'. However, when defining their current disease status at a tissue level rather than a case/patient level, such a periodontitis patient may have healthy periodontal tissues on a reduced periodontium. Similarly, if inflammation is present at 10%–30% of sites where pockets are closed (≤4 mm and no 4-mm bleeding sites), such individuals are classified as periodontitis patients with current gingival inflammation; they cannot be deemed gingivitis patients once attachment loss due to periodontitis has occurred. These principles apply also to children and adolescents. However, the 2018 Classification System was designed for adults and not for younger individuals who are in their primary, mixed or even immature adult dentition. There is, therefore, a pressing need to systematically evaluate the epidemiology, aetiology, risk factors, diagnosis and management of gingival and periodontal diseases and conditions in children and adolescents, and to assess the suitability or otherwise of the 2018 Classification for this age group.

In this Focused Workshop between the European Federation of Periodontology (EFP) and the European Academy of Paediatric Dentistry (EAPD), the term 'periodontal disease' was deemed to include not only conditions limited to the gingival tissues but also those affecting the periodontal attachment apparatus and, in its broadest sense, included conditions that were or were not dental biofilm–induced, in both systemically healthy patients and in those with underlying systemic diseases or conditions that impacted upon the periodontium. It was recognised that the periodontal tissues of children, whether in the primary, mixed or early permanent dentition, significantly differ anatomically from those of adults (Bimstein et al. 2001; Bimstein and Matsson 1999). Moreover, the periodontal microbiome changes throughout adolescence, being affected by hormonal changes at puberty, which also modify the inflammatory status of the tissues (Gusberti et al. 1990). Inflammatory changes, along with the lack of full clinical crown height as well as eruption and exfoliation events, can confound measurement of clinical attachment loss (CAL) probing depth and gingival recession in younger people.

The primary teeth emerge in the oral cavity between the ages of 6 months and 2.5 years, then exfoliate and are replaced by the permanent teeth, which start to appear at 6 years, and continue to erupt up to 17 years (sometimes later), with the gingival margin of permanent teeth not stabilising till about 18 years or more. Gingival maturation is also chronological in relation to the time of primary tooth exfoliation and permanent tooth eruption, further complicating the interpretation and value of probing the periodontal tissue sulcus depth and measuring the width of attached gingiva in the primary, mixed and early permanent dentitions. The first permanent molars and incisors would have typically fully erupted with a full clinical crown height at 12 years, being the first permanent teeth to erupt. Interestingly, a longitudinal observational study of 14-year-old adolescents from an area of high deprivation followed volunteers up to 16 years of age and subsequently to 19.6 years and demonstrated slowly progressing CAL occurring by 16 years in 11% of mandibular incisors and almost 30% of upper first molars (Clerehugh et al. 1990). By 19 years, almost 60% of maxillary first molars and 50% of mandibular incisors had 1 mm or more CAL and 9% of maxillary first molars had 2 mm CAL. Presence of sub-gingival calculus increased from 15% at 14 years to 56% at 19 years, and there was a significant association for both subgingival calculus and baseline dental plaque levels at 14 years and subsequent

attachment loss (Clerehugh et al. 1995). This raises the potential for periodontitis cases to develop in adolescence in specific high-risk teeth (first permanent molars and mandibular incisors), which are the same teeth as those impacted by what was traditionally termed localised juvenile (aggressive) periodontitis (LJP). However, the clinical phenotypes are distinct from each other, as in LJP, bone loss was inconsistent with the presence of local plaque retention factors (e.g., calculus), whereas the incipient and progressive CAL described by (Clerehugh et al. 1990; Clerehugh et al. 1995) was slowly progressing and associated with local predisposing factors and plaque accumulation. Dental biofilm–induced periodontitis in pre-pubertal populations appears to be largely associated with syndromes or underlying systemic disease. Where periodontitis is reported to arise in the absence of systemic disease or syndromes, most of those investigations did not test for underlying systemic conditions that could not have been diagnosed using the systemic examinations undertaken, or did not report them, and, therefore, there is no robust evidence that dental biofilm–induced periodontitis occurs in systemically healthy children.

The Workshop grouped gingival and periodontal diseases and conditions into three major categories:

1. Gingival and periodontal diseases and conditions in children and adolescents with underlying systemic diseases and conditions:
   - Genetic and congenital systemic conditions that impact upon periodontal tissues.
   - Acquired systemic conditions that impact upon periodontal tissues.
   - Genetic and congenital conditions that impact upon periodontal disease onset or progression or response to periodontal therapy.
   - Acquired conditions that impact upon periodontal disease onset or progression or response to periodontal therapy.
2. Gingival diseases and conditions in systemically healthy individuals:
   - Dental biofilm–induced gingivitis.
   - Other gingival conditions.
3. Periodontal diseases and conditions in systemically healthy individuals:
   - Dental biofilm–induced periodontitis.
   - Other periodontal conditions.

The 'other' gingival (Tsilingaridis et al. 2025) and periodontal (Molina et al. 2025) conditions in systemically healthy individuals were essentially comprised of the non-dental biofilm–induced conditions from the 2018 Classification System and are largely distinct from the genetic, congenital and acquired conditions that manifest within the periodontal tissues (Eshkol-Yogev et al. 2025).

2 | Objectives

The objectives of this Focused Workshop on gingival and periodontal diseases and conditions in children and adolescents were the following:

- To update the epidemiology, aetiology, risk factors, diagnosis and management of gingival and periodontal diseases and conditions in children and adolescents.
- To compare the findings with those from adult individuals.
- To explore whether the 2018 Classification of Periodontal Diseases and Conditions is also suitable for diseases and conditions that affect children and adolescents.

3 | Methods

The Focused Workshop on 'Gingival and Periodontal Diseases and Conditions in Children and Adolescents' was organised by EFP in collaboration with EAPD. The in-person meeting took place in Madrid (Spain), during 16–17 March 2025. A total of 30 experts examined the current evidence derived from three commissioned systematic reviews on the topic (Eshkol-Yogev et al. 2025; Molina et al. 2025; Tsilingaridis et al. 2025) and debated their findings and implications (Figure 1). This consensus report is jointly published in the Journal of Clinical Periodontology (JCP), the official journal of EFP, and in the European Archives of Paediatric Dentistry, the official journal of the EAPD (Chapple et al. 2026). The article was peer-reviews within the JCP system, with reviewers appointed by both JCP and EAPD.

The scope of the Focused Workshop covered all conditions listed in the 2018 Classification (Table 1), developed during the 2017 World Workshop of the American Academy of Periodontology (AAP) and the EFP, on the Classification of Periodontal and Perri-implant Diseases and Conditions (Caton et al. 2018). Three systematic reviews were commissioned to specifically cover different groups of diseases and conditions.

3.1 | Working Group 1: Gingival and Periodontal Diseases and Conditions in Children and Adolescents With Systemic Diseases

Working Group 1 was chaired by Iain Chapple and Dominique Declerck (Table 2) and focused on diseases and conditions in children and adolescents with systemic diseases. The systematic review was led by Esti Davidovich and Joerg Meyle (Molina et al. 2025).

3.2 | Working Group 2: Gingival Diseases and Conditions in Systemically Healthy Children and Adolescents

Working Group 2 was chaired by Mariano Sanz and Phoebus Madianos (Table 3) and focused on gingivitis and gingival diseases. The systematic review was led by Georgios Tsilingaridis and Rodrigo López (Tsilingaridis et al. 2025).

3.3 | Working Group 3: Periodontitis and Other Periodontal Conditions in Systemically Healthy Children and Adolescents

Working Group 3 was chaired by Sotiria Gizani and David Herrera (Table 4) and focused on periodontitis and other

FIGURE 1 | Photograph of Workshop participants.

periodontal conditions. The systematic review was prepared by Janet Davies and Lior Shapira (Eshkol-Yogev et al. 2025).

4 | Gingival and Periodontal Diseases and Conditions in Children and Adolescents With Systemic Diseases

4.1 | Introduction

Systemic conditions associated with periodontal status in children (< 11 years of age) and adolescents (11 up to but not including 18 years of age) can be broadly categorised into the following:

A. Genetic, developmental or acquired conditions that have an impact upon the periodontal tissues.

B. Congenital and acquired conditions that impact upon periodontal disease onset, progression or response to periodontal therapy.

The term *genetic* refers to conditions that are caused in part or in their entirety by mutations in the genome. For example, cancers are caused by mutations, which are not passed from one generation to the next. Genetic diseases therefore may or may not be hereditary in nature. *Congenital* conditions are those that are present from birth, which may be inherited; therefore, they are hereditary congenital disorders, or they may arise as a result of exposures that impact the foetus and result in a non-hereditary congenital disorder (e.g., enamel defects from maternal illness during pregnancy). *Hereditary* conditions are those that are passed down from one generation to the next and they may have a genetic and/or congenital origin. Throughout this consensus report, the term *periodontal disease* is employed to cover gingival and periodontal conditions, whether they are plaque biofilm–induced or non-plaque-induced conditions.

4.1.1 | Scope of Periodontal Conditions Impacted by Systemic Diseases and Conditions

There is a large number of periodontal diseases and conditions that arise as manifestations of systemic diseases and there are also systemic conditions that may influence the onset or progression of plaque-induced gingivitis or periodontitis, or the nature of the response to periodontal therapy. Indeed, the systematic review by Molina et al. (2025), which formed one of the technical manuscripts for this consensus report, identified over 70 non-dental biofilm–induced conditions/syndromes that impacted the periodontal tissues and 11 systemic conditions that are documented as impacting upon the onset or progression of gingivitis or periodontitis, or the response of those patients exhibiting the systemic disease to periodontal treatment.

The 2018 Classification categorised non-dental biofilm–induced periodontal conditions across three main domains:

1. Non-dental biofilm–induced gingival diseases (eight categories) (Chapple et al. 2018).
2. Systemic disorders having a major impact on the loss of periodontal tissues by influencing periodontal inflammation, for example, Down syndrome (Jepsen et al. 2018) and periodontal Ehlers Danlos syndrome (Malfait et al. 2017).
3. Systemic disorders that cause loss of periodontal tissues independently of periodontitis (e.g., odontogenic tumours, granulomatosis with polyangiitis) (Jepsen et al. 2018).

TABLE 1 | List of conditions within the scope of each Working Group (WG), following the 2018 Classification of Periodontal Diseases and Conditions.

Periodontal health, gingival diseases and conditions			Periodontitis		Other conditions affecting the periodontium				
Periodontal Health and Gingival Health	Gingivitis: dental biofilm-induced	Gingival diseases: non-dental biofilm-induced	Necrotising periodontal diseases	Periodontitis	Periodontitis as manifestation of systemic diseases	Systemic diseases and conditions affecting the periodontal supporting tissues	Periodontal abscesses and Endodontic-Periodontal Lesions	Mucogingival deformities and conditions around teeth	Traumatic occlusal forces	Tooth- and prosthesis-related factors
WG-2 (Tsilingaridis et al. 2025)		WG-1	WG-3 (Eshkol-Yogev et al. 2025)		WG-1 (Molina et al. 2025)			WG-3 (Eshkol-Yogev et al. 2025)		

TABLE 2 | List of participants, with their role, in Working Group 1.

Role	Full name	Country
EFP Chair	Iain Chapple	UK
EAPD Chair	Dominique Declerck	Belgium
EAPD Reviewer	Esti Davidovich	Israel
EFP Reviewer	Joerg Meyle	Germany
EFP Participant	Søren Jepsen	Germany
EFP Participant	Mervi Gursoy	Finland
EAPD Participant	Biniyam Wondimu	Sweden
EAPD Participant	Clara Joseph	France
EFP representative	Nicola West	UK
EAPD representative	Ferranti Wong	UK
Colgate representative	Stephanie Jakumeit	Switzerland

Abbreviations: EAPD, European Academy of Paediatric Dentistry; EFP, European Federation of Periodontology.

4. Other systemic disorders influencing the pathogenesis of periodontal inflammation (e.g., diabetes, obesity, osteoporosis, arthritis) (Jepsen et al. 2018).

Systemic disease involvement is, therefore, mapped across all four categories, but the non-dental biofilm–induced gingival diseases (Chapple et al. 2018) are largely locally infective, immune/inflammatory, neoplastic, traumatic or pigmented lesions, which were dealt with by Working Group 2 of this consensus because of the lack of association with any long-term underlying systemic condition.

In practical terms, it is important that clinicians examine all oral and peri-oral tissues prior to focusing on the periodontium, as signs of systemic conditions may appear elsewhere and support the definitive diagnosis. Moreover, this facilitates the identification of multiple pathologies that may present within the same patient. The neck should also be examined to identify lymphadenopathy, which can indicate whether a lymph node is reactive or whether a more sinister condition is present such as a malignancy. Examination of the eyes, nails, hair and skin also offers key signs to help with a definitive diagnosis. Rarely, where a space-occupying lesion is suspected, examination of the cranial nerve may be indicated. Additional clinical investigations may be required, such as blood tests (e.g., serum alkaline phosphatase for hypophosphatasia), biopsies and additional imaging such as computed tomography (CT), magnetic resonance imaging (MRI) or ultrasound scans. Documentation and monitoring of a condition may be required using plane or 3D digital imaging. Combining the outcomes of such investigations with risk factor assessment (e.g., smoking) may lead to the decision to consult a physician or dental specialist in order to arrive at a definitive diagnosis and determine appropriate care planning.

Here, the suitability of the 2018 Classification system is analysed for children and adolescents. Almost 2760 reports were identified, which varied in quality and level of evidence and ranged from simple case reports or case series to cohort and

TABLE 3 | List of participants, with their role, in Working Group 2.

Role	Full name	Country
EFP Chair	Mariano Sanz	Spain
EFP Chair	Phoebus Madianos	Greece
EAPD Reviewer	Georgios Tsilingaridis	Sweden
EFP Reviewer	Rodrigo López	Norway
EFP Participant	Filippo Graziani	Italy
EFP Participant	Nikos Donos	UK
EAPD Participant	Svante Twetman	Denmark
EAPD Participant	Elisabeth Dursun	France
EFP representative	Moritz Kebschull	UK
EAPD representative	Tina Lambrinaki	Greece
Colgate representative	Zilson Malheiros	UK

Abbreviations: EAPD, European Academy of Paediatric Dentistry; EFP, European Federation of Periodontology.

TABLE 4 | List of participants, with their role, in Working Group 3.

Role	Full name	Country
EFP Chair	David Herrera	Spain
EAPD Chair	Sotiria Gizani	Greece
EAPD Reviewer	Janet Davies	UK
EFP Reviewer	Lior Shapira	Israel
EFP Participant	Valerie Clerehugh	UK
EFP Participant	Elena Figuero	Spain
EAPD Participant	Norbert Kraemer	Germany
EAPD Participant	Cheryl Somani	UK
EFP representative	Monique Danser	The Netherlands
EAPD representative	David J. Manton	The Netherlands

Abbreviations: EAPD, European Academy of Paediatric Dentistry; EFP, European Federation of Periodontology.

cross-sectional studies. The quantity of available information on each condition also varied from one or two reports and/or studies to 567 manuscripts on Papillon-Lefèvre syndrome alone, creating a challenge in assessing the validity and reliability of the reported periodontal characteristics for each systemic condition. Because of the large quantity of information available, hand-searching was not formally undertaken to supplement the systematic search, but where reports or studies were identified by the expert group, they were assessed against the inclusion/exclusion criteria and included where appropriate.

This report aims to provide a minimum core dataset to act as a reference guide for clinicians, alongside a bespoke classification for periodontal diseases in children and adolescents that are associated with systemic conditions. Guidance on management approaches and special considerations in periodontal management protocols is also provided.

4.2 | How Do We Classify Genetic and Congenital Systemic Conditions That Impact Upon the Periodontal Tissues in Children and Adolescents?

Systemic genetic and congenital conditions may impact upon the periodontal tissues at a structural level, via benign or malignant neoplastic processes, by influencing immune-inflammatory pathways or through metabolic functions. Since virtually all of these conditions are rare, the majority of publications comprise case reports, case series as well as cohort and cross-sectional studies, providing a low to very low level of confidence in the consistency of their impact upon periodontal status and the signs and symptoms associated with these conditions (e.g., Molina et al. 2025). It is acknowledged that many of the systemic conditions discussed also manifest in adults and the evidence base may appear broader; however, it is not possible to extrapolate the characteristics seen in adults to children and adolescent patients.

In Table 5A, genetic and congenital conditions are classified based upon a systematic review of the literature, where at least one report exists that describes one or more cases of periodontal involvement that cannot be attributed to plaque-related causes. In Table 5A, the level of available information is classified as very low, low or moderate, as defined in the legend to the table. However, the level does not equate to the quality of the evidence.

4.3 | How Do We Classify Acquired Systemic Conditions That Impact Upon the Periodontal Tissues in Children and Adolescents?

Systemic acquired conditions may manifest in various tissues within the periodontium and, therefore, impact upon periodontal health as a secondary effect. Either individual or multiple periodontal tissues may be affected, and presentations vary accordingly. For example, odontogenic tumours may appear only as a radiographic finding but may also present later in the disease course as a swelling. By contrast, vesiculobullous conditions may present in a limited manner, involving gingiva alone and as blisters or areas of erosion or ulceration. Here, a history of other body sites, siblings or other family members affected may be required from caregivers, and the examination of non-periodontal sites is also necessary.

Similar to systemic genetic and congenital conditions, the level of information available for acquired systemic conditions that impact upon the periodontal tissues is variable and limited to case reports, case series as well as cohort and cross-sectional studies.

In Table 5B, the acquired systemic conditions are classified based upon a systematic review of the literature, where at least one report exists that describes one or more cases of periodontal involvement that cannot be attributed to plaque-related causes. In Table 5B, the level of available information is classified as very low, low or moderate, as defined in the legend to the table. However, the level does not equate to the quality of the evidence.

TABLE 5 | Systemic genetic, congenital and acquired conditions that impact upon periodontal tissues.

A. Genetic & congenital systemic conditions that impact upon periodontal tissues		
1. Structural disorders	***	Cleft lip/palate
	*	Enamel-renal syndrome
	*	Osteosclerotic bone dysplasia
	**	Ehlers Danlos syndrome (Periodontal sub-type)
	*	Hajdu Cheney syndrome
	**	Hypophosphatasia
	**	X-linked hypophosphatemia
	**	Epidermolysis Bullosa: – Simplex (EBS) – Dystrophic (DEB) – Junctional (JEB)
	*	Hereditary sensory and autonomic neuropathy type-4 (CIPA)
	*	Larsen syndrome
	*	Ramon syndrome
	*	Mosaic trisomy-8 syndrome
2. Neoplastic conditions	**	Benign: Hereditary gingival fibromatosis (HGF)
	*	Malignant: Dyskeratosis congenita
3. Immunological-inflammatory diseases	**	Congenital neutropenia
	*	– Kostmann syndrome
	**	Cyclic neutropenia
	*	Chediak–Higashi syndrome
	*	Bardet–Biedl syndrome
	*	Haim–Munk syndrome
	***	Papillon–Lefèvre syndrome
	***	Juvenile idiopathic arthritis
	**	Leukocyte adhesion deficiency
	*	Hyperimmunoglobulinemia Eb (Job's syndrome)
	*	Hypoplasminogenemia
	*	Juvenile dermatomyositis
	*	Common variable immune deficiency
	*	Acatalasemia (Takahara disease)
4. Metabolic & endocrine disorders	*	Glycogen storage disease
	*	Lysosomal storage disease
B. Acquired systemic conditions that impact upon periodontal tissues		
1. Neoplasm		Benign tumours:
	*	– Ameloblastoma
	*	– Epithelial odontogenic tumour
	*	– Calcifying epithelial odontogenic tumour
	*	– Ameloblastic fibroma
	*	– Ameloblastic fibro-odontoma
	*	– Peripheral ameloblastic fibroma
	*	– Central odontogenic fibroma
	*	– Gingival myofibroma
	*	– Cementoblastoma
		Malignant tumours:
	**	– Leukaemia (acute myelomonocytic) (AML)
		– Lymphoma
	*	○ Hodgkin's lymphoma
	*	○ Non-Hodgkin's lymphoma (T-cell & B-cell)
	*	○ Burkitt's lymphoma

(Continues)

TABLE 5 | (Continued)

2. Granulomatous inflammatory-immune conditions	**	Crohn's disease
	*	Orofacial granulomatosis
	*	Granulomatosis with polyangiitis (formerly Wegener's)
	*	Sarcoidosis
3. Acquired immune conditions	***	Human immunodeficiency virus (AIDS)
	***	Langerhans cell histiocytosis (Histiocytosis-X)
4. Atopic (hypersensitivity) conditions	*	Plasma cell gingivitis
5. Autoimmune conditions	*	Pemphigus
	*	Pemphigoid
	*	Lichen planus
	**	Systemic lupus erythematosus (SLE)
	*	Scleroderma
	*	Linear scleroderma (Morphoea)
6. Giant cell lesions	**	Peripheral giant-cell granuloma
	*	Central giant-cell granuloma

Note: ***Moderate level of available information, defined as ≥2 cross-sectional studies + ≥2 case series and several case reports. **Low level of available information, defined as <2 cross-sectional studies + ≥2 case series or several case reports. *Very low level of available information, defined as isolated case reports + <2 case series. #Scoring system internally validated by the group.

TABLE 6 | Systemic diseases/conditions that impact upon onset or progression of dental biofilm–induced periodontal diseases and response to treatment.

A. Congenital conditions that impact upon periodontal disease onset or progression or response to periodontal therapy		
1. Down syndrome	***	
2. Prader–Willi syndrome	*	
B. Acquired conditions that impact upon periodontal disease onset/progression, or response to periodontal therapy		
1. Tobacco exposure	***	
2. Asthma	***	
3. Endocrine and metabolic conditions	***	Diabetes mellitus—type 1
	*	Diabetes mellitus—type 2
	**	Metabolic syndrome
	*	Mauriac syndrome
	***	Obesity
	*	Vitamin C deficiency
4. Mental health	**	Emotional disorders

Note: ***Moderate level of available information defined as ≥2 cross-sectional studies + ≥2 case series and several case reports. **Low level of available information, defined as <2 cross-sectional studies + ≥2 case series or several case reports. *Very low level of available information, defined as isolated case reports + <2 case series. #Scoring system internally validated by the group.

4.4 | How Do We Classify Congenital and Acquired Systemic Conditions in Children and Adolescents That Impact Upon Periodontal Disease Onset, Progression and Response to Periodontal Therapy?

In Table 6, those congenital and acquired systemic conditions in children and adolescents that influence the onset and/or progression of plaque-induced periodontal diseases or may affect response to periodontal therapy are documented. The congenital conditions include syndromes such as Down syndrome where, for example, immune cell and salivary function may be impaired, which can alter susceptibility to and/or the clinical course of gingivitis and periodontitis. Response to therapy may also be negatively impacted.

Acquired conditions include exposures such as tobacco, and medical conditions such as asthma, diabetes and mental health disorders. Type 1 diabetes is more common in children and adolescents as opposed to adults, where type 2 diabetes predominates. Critically important is the level of metabolic control, because well-controlled diabetes patients do not exhibit differences in periodontal disease susceptibility or healing responses following treatment. This provides an example of where a multidisciplinary approach to care is important, as the oral healthcare professional should liaise with the responsible medical team to improve glycaemic control.

Similar to systemic genetic, congenital and acquired conditions, information levels are limited to case reports, case series, cohort studies and cross-sectional studies as well as two randomised controlled trials.

In Table 6, congenital and acquired systemic conditions that impact upon the onset and/or progression of dental biofilm–induced

periodontal diseases, or the response to periodontal therapy, are classified based upon a systematic review of the literature, where at least one report exists that describes one or more cases. In Table 6, the level of available information is classified as very low, low or moderate, as defined in the legend to the table. However, the level does not equate to the quality of the evidence.

Where a multidisciplinary approach may be required, it is indicated in Table 7.

4.5 | How Should Systemic Diseases and Conditions That Impact Upon Periodontal Tissues in Children and Adolescents Be Managed and by Whom?

Management of systemic conditions that present within the periodontal tissues will vary according to the nature of the condition and availability of appropriate expertise. Formal multidisciplinary clinics may be required based on national policies and guidelines. It is important to recognise the role of the responsible family member, guardian or caregiver and the importance of behavioural management, which may also require engagement of behavioural management specialists or psychologists. In general, conditions that have a medical basis are managed medically by the appropriate physician. In all cases, there should be consultation with paediatric dentistry and/or periodontal specialists, or those with additional training in these disciplines. Certain conditions will require surgical management, which may be undertaken by paediatric dental surgeons, periodontal surgeons and/or oral and maxillofacial surgeons. Additional specialities that may need consulting include special-care dentistry, oral medicine and pathology and maxillofacial radiology. A summary overview of the management approaches is provided in Table 7.

4.6 | How Should Gingivitis and Periodontitis Be Managed in Children With Systemic Conditions That Impact Upon Periodontal Disease Onset, Progression or Response to Periodontal Therapy?

In general, gingivitis and periodontitis can be managed in line with the S3-level guideline for stage I–III periodontitis (Sanz et al. 2020) and the S3-level clinical guideline on the management of gingival diseases and conditions (forthcoming in 2026). However, there are key additional considerations that should be taken into account for paediatric and adolescent patients. These considerations are consistent with a personalised approach to care and include the following:

- The growth and development of the younger patient should be considered, which impact their anatomy, physiology, immune system maturation and nutritional status.
- Specific consideration should be given to the management of behaviour and the need and provision for psychological support, not only for the patient but also for their wider support group.
- The role of parents, guardians and caregivers is critical to the success and thus should not be underestimated.
- There may be a need to adjust and modify treatment approaches following regular re-evaluation at key timepoints in their development.
- There may be a need to extract the primary teeth affected by severe forms of periodontitis to prevent contamination of newly erupting, healthy, permanent teeth (Dibart et al. 1998).
- Communication strategies need to be tailored to the individual patient, family members, guardians and caregivers, and will need to change with time.
- Importantly, oral health professionals should work within their level of competence and scope of practice, and where advice or collaboration is required, it should be sought. It should be recognised that the knowledge of the physician on the impact of periodontal conditions upon medical management may be limited, and, again, collaborative decision making will be required.

4.7 | Are There Special Precautions to Be Taken When Managing Gingivitis and Periodontitis in Children With Systemic Conditions (e.g., Antibiotics, Haemostatic Procedures, Medications)

Risks arising during periodontal management in patients with underlying systemic conditions should be identified prior o developing the care plan. These may include the following:

- Risk of infectious complications, especially but not exclusively in those with immunocompromise.
- Risk of haemorrhagic complications, for example, in those with underlying bleeding disorders, liver disease or taking certain medications.
- Risk of unwanted side effects arising from interactions between medications or the effect of certain medications on the prescribed periodontal intervention.
- Risk of triggering or encountering medical emergencies during periodontal management, such as epileptic episodes or asthmatic attacks.
- Risk of impaired wound healing in certain conditions such as epidermolysis bullosa and the Ehlers–Danlos syndrome.

5 | Gingival Diseases and Conditions in Systemically Healthy Children and Adolescents

5.1 | What Are the Main Gingival Diseases and Conditions That Affect Systemically Healthy Children and Adolescents?

Gingival diseases in systemically healthy children and adolescents may exhibit several distinctive features that differ from those observed in adults. Although dental biofilm–induced gingivitis is the most common manifestation of gingival diseases in systemically healthy children and adolescents, the presence of local and systemic determinants can influence these conditions. The main gingival diseases and conditions are discussed below.

TABLE 7 | Core dataset for periodontal diseases and conditions associated with systemic disorders in children and adolescents.

Condition	Aetiology	Periodontal manifestations	Other orofacial manifestations	Recommended management	Impact on onset, progression or response to therapy
SYSTEMIC DISEASES AND CONDITIONS THAT IMPACT UPON PERIODONTAL TISSUES					
1. Genetic and congenital systemic conditions that impact upon periodontal tissues					
1.1 Structural disorders					
Cleft Lip and Palate	Genetic, congenital	↑ GI & ↑ PI ↑ Bone dehiscence and fenestration defects	Clefts in the lip and/or palate, unilateral or bilateral	Requires MDT: management (orthodontics, plastic surgery, maxillofacial surgery) Refer: periodontal care[a]	Indirect effects due to predisposing factors for plaque accumulation.
Enamel–renal syndrome	Genetic	Gingival enlargement Localised periodontitis	Amelogenesis imperfecta Dental abnormalities	Refer: periodontal care[a]	—
Osteosclerotic bone dysplasia (Raine syndrome)	Genetic, congenital	Gingival enlargement Alveolar bone sclerosis Gingival/follicular calcifications Periodontal abscesses	Hypoplastic maxilla Amelogenesis and dentinogenesis imperfecta Periapical abscesses Incomplete root formation Pericoronal radiolucencies	Requires MDT management (paediatrics and respiratory medicine) Refer: periodontal care[a]	—
Ehlers–Danlos syndrome	Genetic	Gingival enlargement Early onset periodontitis Easy bruising and lack of attached gingiva	Hyperlaxity of TMJ	Refer: periodontal care[a] Consult/Refer Physician: (Geneticist, Rheumatology) Haemorrhagic and infective risk Hospital setting is recommended	—
Hajdu Cheney syndrome	Genetic	Alveolar bone resorption Tooth loss	Root resorption	Refer: periodontal care[a]	—
Hypophosphatasia	Genetic, congenital	Root cementum aplasia Alveolar bone loss Premature loss of deciduous teeth; occasionally loss of permanent teeth	Large pulp chambers in deciduous teeth Dental mineralisation impairment	Requires MDT management (metabolic bone specialist, or endocrinologist) Refer: periodontal care[a]	Tooth loss of deciduous dentition is inevitable due to structural defects in cementum
X-linked hypophosphataemia	Genetic	Gingivitis Spontaneous dental abscesses	Dental abnormalities (shortened roots, enlarged pulp chambers, prominent pulp horns) Hypoplastic enamel	Requires MDT management Consult/refer physician: (family doctor for systemic therapy, vit D supplements, monoclonal antibodies) Refer: periodontal care[a]	Systemic therapy improves the periodontal condition

(Continues)

TABLE 7 | (Continued)

Condition	Aetiology	Periodontal manifestations	Other orofacial manifestations	Recommended management	Impact on onset, progression or response to therapy
Epidermolysis Bullosa: – Simplex (EBS) – Dystrophic (DEB) – Junctional (JEB)	Genetic, congenital in certain cases	Gingivitis Gingival blisters and ulcers Gingival atrophy	Enamel hypoplasia Amelogenesis imperfecta Scarring leading to mucosal ankylosis	Requires MDT management (dermatologist, psychologist, geneticist, nutritionalist, podiatrist) Refer: periodontal care[a]	Brushing discomfort may impair adequate oral hygiene and compromise disease progression
Hereditary sensory and autonomic neuropathy type-4 (CIPA)	Genetic, congenital	Lacerations and ulcerations Bone loss Tooth loss by autoextraction and self-mutilation Hypoplastic cementum Dysplastic periodontal ligament	Scarring leading to limited mouth opening Severe dental attrition Tooth luxation	Requires MDT management Consult/refer physician: (paediatrician, neurologist, geneticist) Refer: periodontal care[a]	—
Larsen syndrome	Genetic, congenital	Early-onset periodontitis Bone loss Tooth mobility	Dental abnormalities (hypodontia, microdontia, supernumerary teeth) Cleft lip and/or palate	Requires MDT management Consult/refer physician: (geneticist, orthopaedic, speech therapist) Refer: periodontal care[a]	—
Ramon syndrome	Genetic, congenital	Gingival enlargement	—	Refer: periodontal care[a] May require MDT depending on needs, e.g., epilepsy	—
Mosaic trisomy-8 syndrome	Genetic, congenital	Gingival enlargement	—	Consult/refer physician: (geneticist) Refer: periodontal care[a]	—
1.2 Neoplastic conditions					
Benign: Hereditary gingival fibromatosis (HGF)	Genetic	Gingival enlargement	—	Refer: periodontal care[a]	High recurrence rate
Malignant: Dyskeratosis congenita	Genetic	Gingivitis Periodontitis	Delayed tooth eruption Peg-shaped lateral incisors Blunted roots	Consult/refer physician: (geneticist, haematologist) Refer: periodontal care[a]	—
1.3 Immunological inflammatory diseases					
Congenital neutropenia – Kostmann syndrome	Genetic, congenital	Gingivitis Periodontitis	Mucosal ulcers	Consult/refer physician: (haematologist, immunologist) Refer: periodontal care[a] Infective risk	—

(Continues)

TABLE 7 | (Continued)

Condition	Aetiology	Periodontal manifestations	Other orofacial manifestations	Recommended management	Impact on onset, progression or response to therapy
Cyclic neutropenia	Genetic	Gingivitis Periodontitis Tooth mobility Premature tooth loss	Mucosal ulcers	Consult/refer physician: (gaematologist) Refer: periodontal care[a] Infective risk	—
Chediak–Higashi syndrome	Genetic, congenital or late onset	Periodontitis Gingival ulceration Gingival abscesses Tooth exfoliation	Labial abscesses	Requires MDT management: (haematologist, neurologist, immunologist, ophthalmologist) Refer: periodontal care[a] Infective risk	—
Bardet–Biedl syndrome	Genetic, congenital	Gingival enlargement	Dental abnormalities (hypodontia, crowding, arched palate)	Requires MDT management: (ophthalmologist, geneticist, endocrinologist, dietician, nephrologist, psychologist) Refer: periodontal care[a]	—
Haim–Munk syndrome	Genetic	Early-onset periodontitis affecting primary and permanent dentition	—	Consult/refer physician: (dermatologist) Refer: periodontal care[a] Infective risk	—
Papillon–Lefèvre syndrome	Genetic, congenital	Dyskeratosis of the gingiva Early-onset periodontitis Premature tooth loss	—	Consult/refer physician: (dermatologist) Refer: periodontal care[a]	Unresponsive to treatment
Juvenile idiopathic arthritis	Unknown	Gingivitis	—	Consult/refer physician: (rheumatologist) Refer: periodontal care[a]	—
Leukocyte adhesion deficiency	Genetic, congenital	Gingivitis Periodontitis Premature tooth loss	Abnormal root resorption	Consult/refer physician: (haematologist) Refer: periodontal care[a] Infective risk	—
Hypergammaglobulinemia E	Genetic, congenital	Gingival enlargement Gingivitis Gingival pain Bone loss Tooth mobility	—	Consult/refer physician: (immunologist) Refer: periodontal care[a] Infective risk	—

(Continues)

TABLE 7 | (Continued)

Condition	Aetiology	Periodontal manifestations	Other orofacial manifestations	Recommended management	Impact on onset, progression or response to therapy
Hypoplasminogenaemia (Ligneous gingivitis/ periodontitis—also known as destructive membranous gingivitis/periodontitis)	Genetic	Gingival enlargement with nodular aspect and yellow-whitish membranes Alveolar bone loss Tooth loss	—	Consult/refer physician: (haematologist) Refer: periodontal care[a]	High tendency for recurrence
Juvenile dermatomyositis	Autoimmune	Gingival telangiectasias Gingival erythema	—	Requires MDT management: (rheumatologist, dermatologist) Refer: periodontal care[a]	Improvement in lesions might be expected, but lesions tend to remain
Common variable immune deficiency	Genetic	Gingivitis	Oral ulcers Tonsilitis Candida and herpes virus infections	Consult/refer physician: (haematologist) Refer: periodontal care[a]	—
Acatalasaemia (Takahara disease)	Genetic	Gangrenous gingivitis and periodontitis Bone loss Premature tooth loss	—	Consult/refer physician: (haematologist, paediatrician) Refer: periodontal care[a]	—
1.4 Metabolic and endocrine disorders					
Glycogen storage	Genetic	Gingivitis Periodontitis Gingival enlargement Tooth mobility	Taurodontism Impaired eruption Malocclusions Oral ulcers	Consult/refer physician: (paediatrician, endocrinologist) Refer: periodontal care[a]	—
Lysosomal storage	Genetic	Gingivitis Gingival enlargement	Taurodontism Malocclusions Oral ulcers	Consult/refer physician: (paediatrician, endocrinologist) Refer: periodontal care[a]	—
2. Acquired systemic conditions that impact upon periodontal tissues					
2.1 Neoplasms					
2.1.1 Benign neoplasms					
Ameloblastoma	Unknown	Tumour in the jaw	—	Refer: oral/maxillofacial surgeon	—
Epithelial odontogenic tumour	Unknown	Tumour in the jaw, characterised by duct-like structures with calcifications	—	Refer: oral/maxillofacial surgeon	—

(Continues)

TABLE 7 | (Continued)

Condition	Aetiology	Periodontal manifestations	Other orofacial manifestations	Recommended management	Impact on onset, progression or response to therapy
Peripheral odontogenic tumour with ghost-cell keratinisation	Unknown	Tumour in the jaw, characterised by areas of calcification	—	Refer: oral/maxillofacial surgeon	—
Ameloblastic fibroma	Unknown	Radiolucent intraosseous lesion Might present associated soft-tissue lesion in the gingiva	—	Refer: oral/maxillofacial surgeon	—
Ameloblastic fibro-odontoma	Unknown	Radiolucent intraosseous lesion, including dental hard tissues	—	Refer: oral/maxillofacial surgeon	—
Peripheral ameloblastic fibroma	Unknown	Extraosseous tumour in the gingiva	—	Refer: oral/maxillofacial surgeon	—
Calcifying epithelial odontogenic tumour	Unknown	Radiolucent tumour with variable calcification, and with unilocular or multilocular cystic appearance	—	Refer: oral/maxillofacial surgeon	—
Central and peripheral odontogenic fibroma	Unknown	Radiolucent lesions, presenting either as solitary (COF) or multifocal (POF) images	—	Refer: oral/maxillofacial surgeon	—
Gingival myofibroma	Unknown	Firm, non-ulcerated gingival swelling lesions with no significant radiographic changes	—	Refer: oral/maxillofacial surgeon	—
Cementoblastoma	Unknown	Radiopaque mass attached to a tooth root, and often painful	—	Refer: oral/maxillofacial surgeon	—
2.1.2 Malignant neoplasms					
Leukaemia	Unknown	Gingivitis, mucosal pallor, petechiae	Mucositis, mouth ulcers, xerostomia, herpes or candidiasis	Consult/refer physician: (haematologist, oncologist)	—
Lymphoma (B-cell or Burkitt)	Unknown	Localised swelling, ulceration, pain, tooth mobility. Radiologically lytic radiolucent lesion.	Localised swelling, pain, tooth mobility/ displacement, paraesthesia.	Consult/refer physician: (haematologist, oncologist) Refer: periodontal care[a] Haemorrhage and infection risk Hospital setting is recommended	—

(Continues)

TABLE 7 | (Continued)

Condition	Aetiology	Periodontal manifestations	Other orofacial manifestations	Recommended management	Impact on onset, progression or response to therapy
2.2 Immune alterations					
Granulomatous inflammatory diseases					
– Crohn's disease	Genetic	Fire-red oedema and pain in the gingiva	—	Refer: gastroenterologist Refer: periodontal care[a]	—
– Orofacial granulomatosis	Genetic	Gingival hyperplasia, erythema, swelling, pain and strawberry gingivitis with petechiae	Mucosal ulcers with pseudomembranous covering, purulent secretions and alveolar abscesses	Refer: oral medicine specialist. Refer: periodontal care[a] Infective risk	—
– Granulomatosis with polyangiitis (Wegener's granulomatosis)	Unknown	Gingival hyperplasia, erythema, and strawberry gingivitis with petechiae	Mucosal ulcers with pseudomembranous covering, and purulent secretions	Requires MDT management: (rheumatologist, nephrologist, pulmonologist, neurologist, ENT) Refer: periodontal care[a] Infective risk	Oral manifestations can resolve with immunosuppressive therapy
– Sarcoidosis	Unknown	Firm, non-mobile and non-tender mass Gingival erythema Progressive tooth mobility	—	Requires MDT management: (respiratory, cardiology, neurology, dermatology specialists) Refer: periodontal care[a]	—
Human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS)	Viral infection	Gingivitis Necrotising gingivitis/ periodontitis	Candidiasis, angular cheilitis, parotid enlargement, ulcers and viral lesions (e.g., molluscum contagiosum, herpes zoster)	Consult/Refer Physician: (genitourinary medicine). Refer: periodontal care[a] Infective risk	—
Plasma cell gingivitis	Unknown	Gingival and mucosal erythematous and bleeding lesions, focal and raised gingival enlargement, pseudopocket formation	—	Consult/Refer Physician: (dermatology, clinical immunology) Refer: periodontal care[a]	Lesions tend to be intractable and recalcitrant due to the ubiquitous nature of allergens
Langerhans cell histiocytosis	Unknown	Periodontitis, tooth mobility	Radiolucent osteolytic lesions in the skull and jaw bones	Consult/Refer physician: (haematology, oncology, other specialties if needed) Refer: periodontal care[a]	Poor response to therapy, high tendency for recurrence

(Continues)

TABLE 7 | (Continued)

Condition	Aetiology	Periodontal manifestations	Other orofacial manifestations	Recommended management	Impact on onset, progression or response to therapy
2.3 Autoimmune diseases of skin and mucous membranes					
Pemphigus vulgaris	Autoimmune disease	Mucosal and gingival painful erosions	Painful erosions in tonsils and pharynx Hoarseness and swallowing difficulties	Consult/Refer physician: (dermatology) Refer: periodontal and oral medicine care by specialists	—
Pemphigoid	Autoimmune disease	Mucosal and gingival blistering that evolves into painful erosions, followed by scarring gingivitis, spontaneous bleeding	Painful erosions in pharynx and oesophagus	Consult/refer physician: (dermatology) Refer: periodontal and oral medicine care by specialist	—
Lichen planus	Unknown	Gingival painful erosions	—	Refer: oral medicine Refer: periodontal care[a]	—
Systemic lupus erythematosus	Autoimmune disease	Gingivitis	Temporomandibular joint dysfunction	Consult/refer physician: (rheumatologist) Refer: periodontal care[a]	—
Scleroderma	Autoimmune disease	Fibrotic mucosa Gingivitis PDL space widening	—	Consult/refer physician: (rheumatologist) Refer: periodontal care[a]	—
2.4 Giant cell lesions/ granulomas	Reactive lesions	Firm, lobulated, reddish-purple or bluish mass in the gingiva or mucosa	Tooth displacement Delayed eruption Misalignment	Refer: oral surgeon Refer: periodontal care[a]	Lesions may recur
SYSTEMIC DISEASES AND CONTRIBUTING FACTORS THAT IMPACT UPON THE ONSET/PROGRESSION OF PERIODONTAL DISEASES					
1. Congenital conditions that impact periodontal diseases					
Down syndrome	Genetic, congenital	Gingivitis Periodontitis	—	Refer: periodontal care[a]	—
Prader–Willi syndrome	Genetic, congenital	Gingivitis	—	Refer: periodontal care[a]	—
2. Acquired systemic diseases and contributing factors that impact periodontal diseases					
2.1 Tobacco abuse	Recreational drug	Smokeless tobacco–associated lesions: mucosal wrinkling, discolouring, and thickening	—	Refer: Behaviour change counselling service	—

(Continues)

TABLE 7 | (Continued)

Condition	Aetiology	Periodontal manifestations	Other orofacial manifestations	Recommended management	Impact on onset, progression or response to therapy
2.2 Asthma	Multifactorial	Gingivitis	—	Consult/refer physician: (respiratory medicine) Refer: periodontal care[a]	Consider the impact of inhaled corticosteroids on gingival tissues
2.3 Endocrine, nutritional and metabolic diseases/conditions					
Diabetes mellitus					
– Type 1 diabetes mellitus	Autoimmune	Gingivitis	—	Consult/refer physician: (endocrinologist) Refer: periodontal care[a]	Metabolic control influences the association between type-I DM and gingivitis
– Type 2 diabetes mellitus	Multifactorial	Periodontitis	—	Consult/refer physician: (endocrinologist) Refer: periodontal care[a]	—
Metabolic syndrome	Multifactorial	Gingivitis	—	Consult/refer Physician: (endocrinologist) Refer: periodontal care[a]	—
Mauriac syndrome	Autoimmune	Periodontitis Recession Tooth mobility	—	Consult/refer physician: (endocrinologist) Refer: periodontal care[a]	Mauriac syndrome can revert with good metabolic control
Obesity	Multifactorial	Gingivitis Periodontitis	—	Refer: nutritionist, physician Refer: periodontal care[a]	—
Vitamin deficiencies					
– Vitamin C deficiency (scurvy)	Nutritional deficiency	Gingivitis Gingival enlargement Tooth mobility	—	Refer: nutritionist, family doctor Refer: periodontal care[a]	Vitamin C supplementation reverts the periodontal manifestations
2.4 Mental health/ emotional disorders	Multifactorial	—	Worse perceptions of oral health-related well-being and quality of life	Consult/refer physician: (psychologist, psychiatrist)	—

Abbreviations: DM, diabetes mellitus; GI, gingival index; MDT, multidisciplinary team; PDL, periodontal ligament; PI, plaque index.
[a]Refers to periodontal specialist or other dentists with specific training.

5.1.1 | Dental Biofilm–Induced Gingivitis

Dental biofilm–induced gingivitis is the most common gingival condition, characterised by redness, swelling and bleeding of the gums. In children, this condition is often milder than in adults but can progress rapidly during puberty because of hormonal changes. Depending upon the presence of specific local or systemic determinants, various gingival conditions have been identified, although their pathophysiology is similarly due to dental plaque accumulation:

- Modified by systemic determinants, such as *puberty-associated gingivitis*, which occurs during adolescence (typically ages 11–14 years) due to hormonal fluctuations that increase gingival inflammation and exaggerate the response to plaque and frequently presents with pronounced inflammation and bleeding.
- Modified by local determinants, such as:
  - *Eruption gingivitis*: localised inflammation around erupting primary or permanent teeth. Common during tooth eruption phases.
  - *Tooth developmental anomalies*: conditions such as molar–incisor hypo-mineralisation (MIH) and amelogenesis imperfecta. These can impact oral hygiene and lead to gingival diseases.
  - *Tooth position in the alveolar bone.*
  - *Carious lesions.*
  - *Oral hygiene level.*
  - *Growth and development changes*: involved in transition from primary to mixed to permanent dentitions.
  - *Persisting primary teeth when succeeding permanent teeth are erupting.*

5.1.2 | Gingival Enlargement

Gingival enlargement mainly results from medications, particularly phenytoin, ciclosporine and calcium channel blockers, as well as inherited traits or systemic conditions (see Section 4). It is more common among adolescents.

5.1.3 | Primary Herpetic Gingivostomatitis and Other Infections

These typically affect children and are characterised by painful, widespread gingival inflammation, vesicles and ulcerations.

5.1.4 | Necrotising Gingivitis

Although rare in children from developed countries, malnutrition, extreme living conditions and systemic conditions can predispose them to necrotising gingivitis, which is characterised by painful necrotic gingival papillae (see Section 6).

5.1.5 | Other Conditions

These include epulides, pigmentation and traumatic lesions affecting the gingival tissues.

5.2 | What Is the Epidemiology of Gingival Diseases in Children and Adolescents?

Systematic reviews, identified by the commissioned review (Tsilingaridis et al. 2025), indicate that gingivitis is common in children and adolescents, with occurrence increasing with age. In epidemiological studies involving >1000 participants, the overall estimate for gingivitis was 54%. The high heterogeneity across studies hinders the comparability of estimates across different populations.

5.3 | What Are the Main Characteristics of Dental Biofilm–Induced Gingivitis in Children and Adolescents?

Dental biofilm–induced gingivitis is the most common type of gingival disease in children and adolescents. It is characterised by the following clinical features:

- Red, swollen gingival margins and interdental papillae.
- Bleeding on brushing or probing (from 7 years of age).
- Generally painless.
- No loss of clinical attachment or the alveolar bone.
- Reversible with effective oral hygiene and professional mechanical plaque removal (PMPR). PMPR is defined as the professional removal of dental biofilm, dental calculus and staining supra-gingivally and also in the gingival sulcus (Sanz et al. 2020; Trombelli et al. 2015).

5.3.1 | Age-Specific Characteristics: Primary Dentition

Gingival inflammation is typically less frequent and less severe in younger children than in older ones, possibly due to the greater thickness and height of the gingival tissues and/or the transition from supervised brushing in younger children to unsupervised brushing in adolescents. Moreover, the morphology of teeth contributes to fewer plaque-retentive factors. It usually presents with marginal redness and minimal bleeding.

5.3.2 | Age-Specific Characteristics: Mixed Dentition

Because of the anatomical changes that occur during tooth eruption—specifically the presence of an immature gingival epithelium—there may be increased gingival inflammation and bleeding, deep probing depths and operculitis around emerging permanent teeth.

5.3.3 | Age-Specific Characteristics: Permanent Dentition

Hormonal influences on the response to plaque exposure lead to a higher prevalence and severity of gingivitis during puberty. It typically presents with significant enlargement of the interdental papillae.

5.4 | What Are the Main Differences Between Dental Biofilm–Induced Gingivitis Affecting Adults, Adolescents and Children?

All age groups exhibit similar symptoms, including bleeding, redness and swelling, which result from inadequate oral hygiene and plaque accumulation. However, the underlying determinants often vary based on age-specific factors.

In children and adolescents, the following factors are present:

- Differential exposures to hormonal factors, particularly during puberty.
- An increased likelihood of inflammatory gingival enlargement.
- Changes associated with mouth-breathing in children.
- Oral microbiome changes across different age groups.
- Very rare cases of periodontitis in children in the primary and mixed dentition phases, which are normally due to underlying syndromes or systemic conditions, with a lack of evidence for progression of gingivitis to periodontitis in this age cohort.

In adults, the following consequences may occur:

- If not treated, gingivitis may progress to periodontitis.
- It may be worsened by known risk factors such as diabetes or smoking.
- It may frequently be associated with ageing and stress.

5.5 | What Are the Most Appropriate Tools to Diagnose Gingival Diseases in Children and Adolescents?

Diagnosis should always be conducted by a qualified oral healthcare professional, and diagnostic methods need to be adapted to each child's developmental stage, level of cooperation and specific presenting symptoms.

Various diagnostic tools and methods have been documented in the scientific literature for diagnosing gingival diseases in children and adolescents, including the following:

5.5.1 | Visual Examination

- Evaluation of gingival colour, contour, consistency and texture.
- Evaluation of gingival margins and papillae.
- Bleeding from toothbrushing.

5.5.2 | Bleeding Scores With or Without Periodontal Probing

- Gingival Bleeding Score (dichotomous).
- Papillary Bleeding Score (dichotomous).
- Simplified Gingival Index (SGI), modified for children.

5.5.3 | Plaque Indices

- Plaque-free score.
- Simplified Oral Hygiene Index (OHI-S).
- Plaque Control Record (PCR).

5.5.4 | Radiographic Examinations

Radiographic examinations should be undertaken only when diagnosing caries lesions or when a clear clinical indication exists (e.g., presence of disproportionate gingival inflammation in relation to age).

5.5.5 | Microbiological and/or Supplementary Testing

This may help only in severe or refractory cases or in cases suspected of specific infections or a specific underlying disease.

There is no clear case definition for dental biofilm–induced gingivitis in children and adolescents. Although a threshold of 10% bleeding on probing (BOP) sites has been established for adults in the 2018 Classification of Periodontal Diseases and Conditions to define gingivitis, there is a consensus that this is not suitable for defining gingivitis in children and adolescents. More research is necessary to understand the actual distribution of dental biofilm–induced gingivitis in these populations.

5.6 | What Are the Limitations of Using Periodontal Probes to Diagnose Gingival Diseases in Children and Adolescents?

Periodontal probing has its limitations when diagnosing gingival diseases in children and adolescents for the following reasons.

5.6.1 | Anatomical and Developmental Factors

- *Immature periodontal tissues*: in children, gingival tissues are more vascular and less keratinised, which may result in increased bleeding during probing without necessarily indicating inflammatory disease.
- *Erupting teeth*: partially erupted teeth create pseudopockets that may be mistaken for pathological conditions.
- *Mixed dentition*: the transitional phase between the primary and permanent dentition complicates accurate probe placement and measurement.

5.6.2 | Behavioural Considerations

- *Cooperation issues*: some children may struggle to remain still or to follow instructions during the procedure.

- *Anxiety and fear*: the sensation of probing can be uncomfortable and distressing for younger patients.
- *Pain perception*: children may respond strongly to probing because of limited prior experience.

5.6.3 | Diagnostic Limitations

- *Normal baseline*: the absence of established 'normal' baseline probing depths for various age groups complicates interpretation.
- *Bleeding on probing*: high false-positive rates are thought to be likely in children due to more reactive tissues.
- *Probing pressure*: challenges in sustaining a steady probing force in uncooperative patients.

Until all permanent teeth have fully erupted—typically around 12 years of age—except for the second and third molars, the results of periodontal probing should be interpreted with caution in the diagnostic assessment of gingival diseases, unless there is evidence of bone loss found during radiographic evaluation, or a clear suspicion of periodontal attachment loss is noted during the clinical examination. In younger children, the diagnosis of gingivitis can be based on observable inflammation of the gingival tissues and bleeding during brushing.

5.7 | What Are the Main Local Predisposing Factors for Dental Biofilm–Induced Gingivitis in Children and Adolescents?

Dental plaque biofilm, which contains microorganisms that cause gingival inflammation, is the main local cause of inflammation in children/adolescents. This condition is frequently associated with inadequate oral hygiene practices, which include insufficient or improper brushing and interdental cleaning methods, leading to plaque accumulation, particularly at the gingival margin.

Several local factors are associated with increased plaque accumulation that contribute to gingivitis in children and adolescents:

- *Dental plaque biofilm-retentive factors*: these include orthodontic appliances, open dental caries lesions, overhanging restorations and anatomical issues such as crowded and misaligned teeth and enamel projections can create areas that are difficult to clean.
- *Mouth-breathing*: this frequently occurs in children with allergies or with adenoid hypertrophy, leading to drying of the gingival tissues, reduced plaque removal by saliva and muscle movement and heightened inflammation.
- *Tooth eruption*: during the mixed dentition phase, partially erupted teeth create zones that complicate plaque removal.
- *Tooth exfoliation*: the areas surrounding exfoliating primary teeth often exhibit localised gingival inflammation.
- *Food impaction*: this occurs in the gaps between teeth or in open proximal carious lesions where food becomes trapped, resulting in localised inflammation.
- *Dental calculus*: it consists of calcified deposits that create rough surfaces, promoting further plaque build-up.
- *Tooth developmental anomalies*: these include deep palatal grooves on maxillary incisors and furcation areas on molars, which can serve as sites for plaque retention.

5.8 | What Are the Primary Systemic Modifying Factors for Dental Biofilm–Induced Gingivitis in Healthy Children and Adolescents?

Systemic modifying factors often interact with local factors to enhance plaque accumulation and/or response to plaque accumulation across multiple dimension:

- *Nutrition*: this includes diets high in refined carbohydrates.
- *Hormonal changes during puberty*: increased levels of sex hormones (oestrogen and progesterone) can heighten the gingival inflammatory response to plaque, making adolescents particularly susceptible during this period.
- *Overweight and obesity*: associated with chronic low-grade inflammation that may increase the risk of gingivitis.
- *Environmental factors*: such as exposure to heavy metals, toxins, smoking, vaping and even passive smoke exposure in children, and low socioeconomic status of children and parents.
- *Stress without coping strategies*: can affect the immune response and increase susceptibility to periodontitis in adults, although data is less clear in children.

5.9 | What Are the Key Nutritional Factors Influencing Gingival Diseases in Children and Adolescents?

Nutritional factors can significantly influence the development of gingival diseases in children and adolescents (e.g., severe vitamin C deficiency).

5.9.1 | Macronutrient-Related Factors

- *High consumption of simple/refined sugars*: regularly consuming sugary foods and beverages promotes dental plaque formation and creates an acidic oral environment that supports pathogenic bacteria, which is linked to a higher incidence of gingivitis (as well as dental caries), especially among adolescents.
- *Inadequate protein intake*: during growth, proteins play a crucial role, and a diet lacking sufficient protein can hinder immune function and tissue repair processes, potentially jeopardising the health of periodontal tissues.

5.9.2 | Micronutrient-Related Factors

- *Vitamin C deficiency*: this impairs the synthesis of collagen, essential for gingival health, which can hinder gingival

healing and is also a powerful antioxidant to combat oxidative stress that predisposes individuals to excessive inflammation and can result in bleeding. For cases of severe deficiencies, see Section 4.

- *Vitamin D and calcium deficiency*: this is becoming increasingly common among children who have limited sun exposure and dairy consumption, particularly in those with high levels of skin pigmentation. This condition is linked to decreased bone mineral density, which may compromise the integrity of dental hard tissues and the alveolar bone.
- *Vitamin A deficiency*: if severe, it may compromise the integrity of epithelial tissue, affect the health of all mucous membranes in the oral cavity and potentially reduce resistance to gingival infections.
- *Vitamin B complex deficiencies*: more common in children with restrictive diets, particularly those with deficiencies in folate and B12, it usually manifests as glossitis and gingival inflammation.
- *Iron deficiency*: can predispose to mucosal and gingival ulceration, as iron is an important element for epithelial cell maturation.

5.9.3 | Dietary Patterns and Behaviours

Dietary patterns and behaviours typically linked to socioeconomic environments and low oral health literacy are characterised by the following:

- *Diet rich in ultra-highly processed foods*: often high in simple and refined carbohydrates while low in protective nutrients. Such diets are pro-inflammatory in nature and also lack the naturally cleansing foods such as crisp fruits and vegetables, and are thus associated with increased plaque formation.
- *Irregular eating patterns*: frequent snacking without proper oral hygiene raises exposure to cariogenic foods and disrupts the natural flow patterns of saliva that help cleanse the mouth.
- *Diets high in soft and sticky foods*: reduced mastication decreases the mechanical cleansing of tooth surfaces and is linked to higher plaque accumulation along the gingival margin.
- *Eating disorders*: malnutrition resulting from anorexia or bulimia is more prevalent among adolescents, especially females. It has a severe impact on oral health, causing not only erosion but also damage to the gingival tissues.

5.10 | What Are the Key Preventive Strategies for Managing Dental Biofilm–Induced Gingivitis in Children and Adolescents?

Managing gingival diseases in children and adolescents requires various preventative strategies, such as early diagnosis and appropriate interventions. The most common interventions for primary prevention are discussed below:

- *Proper oral hygiene instruction*: use appropriate age-specific brushing techniques (twice daily with a fluoride toothpaste).
- *Dietary counselling*: limit the intake of free simple sugars while encouraging a balanced diet that is rich in fruits and vegetables.
- *Regular dental check-ups*: in non-risk patients, routine oral health assessments should be conducted at least annually.

5.11 | What Are the Main Treatment Strategies for the Management of Dental Biofilm–Induced Gingivitis in Healthy Children and Adolescents?

The treatment of dental biofilm–induced gingivitis in children and adolescents focuses on controlling dental biofilm build-up and reducing gingival inflammation. Most cases of dental biofilm–induced gingivitis in this age group respond well to improved oral hygiene and PMPR. Severe or persistent cases may require assessment for underlying systemic conditions.

5.11.1 | Professional Dental Care

- PMPR for the elimination of dental plaque and dental calculus.

5.11.2 | Oral Health Education, Motivation, Behavioural Change and Skills Training/Coaching in Oral Hygiene

- Proper brushing technique: tooth brushing twice daily with a fluoride toothpaste.
- Interdental cleaning as appropriate to age.
- Supervision by parents or caregivers for young children.

5.11.3 | Mechanical Dental Biofilm Control

- Manual and powered toothbrushes: the systematic review (Tsilingaridis et al. 2025) reported mixed evidence, with two studies showing no clinically relevant differences between powered and manual toothbrushes, and two more recent clinical trials demonstrating significant reductions in mean gingival indices favouring the use of powered toothbrushes.
- Interdental cleaning suitable for age (dental floss, floss holders, interdental brushes).
- Consider using agents to visualise plaque to facilitate more effective removal.

5.11.4 | Chemical Plaque Control

- Antimicrobial toothpastes should be age-appropriate.
- Antimicrobial mouth rinses should be age-appropriate and alcohol-free (for short-term use in severe or non-responsive cases).

5.11.5 | Contributing Factors

- Improve oral hygiene practices when using orthodontic appliances, as they may need specialised cleaning aids.
- Consider medication side effects that may lead to gingival hyperplasia.
- Provide nutritional counselling to reduce refined sugar intake.

5.11.6 | Follow-Up and Supportive Care

- Reassess gingival health and adjust the treatment plan as necessary.
- Reinforce oral hygiene instructions.

5.12 | What Are the Recommended Specific Behavioural Methods to Improve Oral Hygiene in Children and Adolescents?

Potentially effective strategies to enhance oral hygiene in children and adolescents, according to age, are discussed below.

5.12.1 | In Pre-School Children

Parents, guardians or caregivers should implement toothbrushing and acquire the necessary oral health literacy, guidelines and behavioural techniques.

5.12.2 | In School-Age Children

- In schools, various techniques, such as reward systems, storytelling and teacher supervision, can improve oral health literacy and motivation in children. Consistent brushing times should be integrated into daily routines.
- As children grow, there should be a gradual transition of responsibility from supervised brushing to independent brushing.
- Utilise motivational tools like visual feedback (displaying solutions that emphasise plaque on teeth), set achievable oral hygiene goals with rewards and incorporate positive reinforcement, such as praise and encouragement, instead of criticism.

5.12.3 | In Adolescents (12–17 Years)

Enhance the previously mentioned oral health literacy and behaviour-changing methods, supplemented by additional tools relevant to adolescent behaviour. These tools include increasing self-autonomy, fostering social motivation through peer-based programmes, integrating technology via smartphones or social media and emphasising personal relevance concerning appearance and social confidence.

5.13 | Are there specific oral hygiene methods recommended for children and adolescents?

Oral hygiene practices should be tailored to a child's age and development.

5.13.1 | In Infants (0–1 Year)

Once the first tooth appears, parents, guardians or caregivers should brush their children's teeth (or clean with gauze) at least once a day, preferably before bedtime, using a smear of fluoride toothpaste about the size of a grain of rice.

5.13.2 | In Toddlers (1–3 Years)

Parents must ensure their children brush twice daily with a small soft-bristled toothbrush and a pea-sized amount of fluoride toothpaste (1000 ppm fluoride).

5.13.3 | In Pre-School Children (3–6 Years)

Parents should continue brushing and gradually introduce their child to a toothbrush that is of appropriate size. Brush twice daily using a pea-sized amount of fluoride toothpaste.

5.13.4 | In School-Age Children (Up to but Not Including 12 Years)

Parents should continue supervising their children while brushing and gradually encourage them to brush independently for 2 min twice a day, using an appropriately sized toothbrush and fluoride toothpaste. They should monitor the effectiveness of their children's brushing until around age 12.

5.13.5 | In Adolescents (From 12 to Up to but Not Including 18 Years)

Independently brush for 2 min twice day using a fluoride toothpaste. Depending on the health condition of the gingival tissues, individuals should consider interdental cleaning and the use of mouth rinses as needed (for short-term use in severe cases or, in specific instances, as part of a personalised treatment plan). Support from parents, guardians or caregivers may be required for some adolescents.

5.14 | Is the Use of Antimicrobial Rinses Indicated in Children and Adolescents?

The foundation stone for managing gingivitis is the self-performed mechanical removal of the dental plaque biofilm. Antimicrobial rinses should be prescribed specifically for children and adolescents only when addressing a particular condition and in situations where mechanical plaque control is ineffective. The data on the efficacy of antimicrobial mouth rinses for gingival diseases in children and adolescents are primarily based on

the use of chlorhexidine in various concentrations. Other agents have shown significant results in reducing gingivitis in specific populations, but their external validity is limited.

5.14.1 | Physical/Biological Risks

- Children below the age of 7 or 8 years cannot always control their swallowing reflexes and may swallow the product, potentially causing stomach upset, nausea or toxicity based on the active ingredients.
- *Mucosal irritation*: certain formulations may irritate the sensitive oral tissues of children.
- *Taste aversion*: this can lead to children resisting their use due to strong flavours or sensations.
- *Disruption of the oral microbiome*: prolonged use of antimicrobial rinses may disrupt the natural balance of bacteria in the developing oral cavity and may lead to antimicrobial resistance.

5.14.2 | Other Adverse Effects

- *Alcohol content*: many rinses contain ethanol (up to 26%), which can present risks of toxicity if ingested, leading to oral burning sensations and dry mouth.
- *Chlorhexidine-related risks*: these mainly include temporary taste alterations, tooth staining and increased calculus formation with long-term use. Although rare, allergic reactions to chlorhexidine have been reported.

5.14.3 | Age-Specific Considerations

- In younger children (under 6 years), antimicrobial agents delivered in mouthwash formats are not recommended because of their limited ability to rinse without swallowing, difficulty following proper usage instructions and the developmental stage of their oral tissues.
- In children aged 6–12 years, their use should occur only under strict parental supervision.
- Adolescents are more capable of using them properly, but they still require guidance.

5.15 | What Are the Main Treatment Strategies for the Management of Specific Gingival Conditions?

5.15.1 | Gingivitis Modified by Puberty

Gingivitis modified by puberty is common in adolescents due to hormonal changes that may affect the inflammatory response of the gingival tissues. It is important to note that most puberty-related gingival conditions are temporary and resolve with improved oral hygiene as hormonal fluctuations stabilise. However, professional dental care during this period should be provided and always tailored to the individual patient's needs.

This care is based on guided reinforcement of oral hygiene, which includes regular brushing twice daily with fluoride toothpaste and interdental cleaning in specific areas as needed. It also includes PMPR to eliminate plaque and calculus as well as any other local factors that encourage plaque accumulation.

Regular oral health assessments should be conducted every 6–12 months until gingival inflammation resolves.

In severe cases, consider using antimicrobial mouth rinses or subgingival instrumentation for significant hyperplasia.

In adolescents undergoing orthodontic treatment, additional preventive measures should be implemented.

5.15.2 | Gingival Diseases Associated With Orthodontic Treatment

Orthodontic treatment can sometimes lead to the following gingival conditions in children and adolescents by impairing access to adequate plaque removal: dental biofilm–induced gingivitis, gingival enlargement and gingival recession. It is advised that orthodontic treatment not be started unless these patients have healthy gingival tissues.

The primary preventive strategies during orthodontic therapy involve coaching in oral hygiene to include detailed instructions for proper brushing techniques around brackets and wires, and using special orthodontic brushes, interdental brushes and floss threaders.

The primary professional intervention is PMPR. More frequent dental visits are recommended to evaluate gingival health, typically every 3–4 months instead of every 6 months during orthodontic treatment, tailored to the patient's needs.

In *gingival hyperplasia or enlargement*, the cause is often attributed to plaque accumulation around orthodontic appliances. Therefore, its management focuses on improving oral hygiene, with recommended target plaque scores below 15%–20%, and PMPR. In severe cases, consult the orthodontist to discuss the possibility of halting orthodontic therapy and removing the appliances to reinforce oral hygiene practices. Depending on the specific situation, additional surgical tissue excision may be necessary, either during or after orthodontic treatment. Furthermore, consider using chlorhexidine mouth rinses (0.12%) to reduce inflammation.

In *dental biofilm-induced gingivitis*, the cause is also due to plaque accumulation around orthodontic appliances. Therefore, management should focus on improving oral hygiene and PMPR. In some instances, the use of short-term antimicrobial mouth rinses is advised, as is the potential modification of the orthodontic appliance if specific components are causing irritation.

In *gingival recession*, management should focus on coaching in toothbrushing techniques and evaluating orthodontic forces, as excessive forces may contribute to this condition, particularly in patients with a thin gingival phenotype. Mucogingival surgery may be considered in severe cases, either before or after orthodontic treatment, depending on the patient's age and the type and direction of orthodontic tooth movements. See also Section 6.

Special considerations in managing gingival diseases related to orthodontic treatment include using age-appropriate oral hygiene tools, such as electric toothbrushes, flossers or other devices that enhance brushing compliance around orthodontic appliances. When possible, appliances that minimise gingival irritation should be used. Additionally, in rare cases of documented allergic reactions to nickel-based orthodontic appliances, consult the orthodontist for an appliance change.

If persistent gingival issues arise during orthodontic treatment, it is advisable to pursue interdisciplinary management that includes both the orthodontist and periodontist to effectively address the problems while maintaining essential orthodontic care.

5.15.3 | Medication-Related Gingival Enlargement

Drug-induced gingival enlargement in children and adolescents is primarily associated with three main classes of medications: anticonvulsants/antiepileptics, immunosuppressants and calcium channel blockers used in treating hypertension. Among children and adolescents, phenytoin and ciclosporin tend to cause the most significant gingival changes, with some studies reporting prevalence rates of 50% or higher. Although calcium channel blockers are less frequently prescribed for children, they can still lead to this side effect when used to treat hypertension or certain cardiac disorders.

The severity of gingival enlargement typically varies depending on factors such as medication dosage and duration, concomitant medications, individual susceptibility, the effectiveness of oral hygiene and hormonal changes during puberty that may exacerbate drug-induced effects.

Its management typically involves medication adjustments in consultation with the prescribing physician, enhanced oral hygiene, professional dental care and, in severe cases, surgical intervention. Because this condition requires medication management, a multidisciplinary approach that includes the child's paediatrician, prescribing specialist and paediatric dentist is essential for optimal outcomes.

The first step should always be to consult with the prescribing physician about alternative medications that do not cause gingival enlargement, which is particularly relevant for calcium channel blockers (such as nifedipine), anticonvulsants (such as phenytoin) and immunosuppressants (including ciclosporine), because the overgrowth is a medical side effect and should therefore be managed medically. Subsequently, implement meticulous plaque control measures to lessen the severity of the condition, such as brushing twice daily with a soft toothbrush, using age-appropriate antimicrobial mouthwashes and having regular PMPR every 3–4 months. When gingival enlargement is severe and interferes with function, aesthetics or oral hygiene, consider surgical interventions such as gingivectomy, periodontal flap procedures or laser surgery aimed at removing excess gingival tissue.

In paediatric patients, treatment planning must consider the child's growth and development, the level of co-operation and the psychological impact. Generally, less invasive approaches are preferred, and more frequent follow-up appointments may be necessary.

5.16 | Acute Gingival Infections

The most common acute gingival infections affecting children and adolescents are viral infections. The most significant ones include the following:

- *Herpetic gingivostomatitis*: caused by the herpes simplex virus (HSV-1), this is the most common viral infection affecting the gingival tissues in children. It typically presents with painful, red and swollen gums, fever and vesicles that rupture to form ulcers.
- *Hand, foot and mouth disease* (*HFM*): primarily caused by Coxsackievirus A16, this infection can lead to small, painful ulcers on the gums and characteristic lesions on the hands, feet and inside the mouth.
- *Herpangina*: is caused by Coxsackie viruses A2, 4, 5, 6, 8, 10 and 23 and has similar clinical presentation as HFM.
- *Varicella-zoster virus* (*chickenpox*): while less common in the gingiva specifically, it can still lead to oral manifestations, such as gingival lesions, when children develop chickenpox.
- *Epstein–Barr virus*: associated with infectious mononucleosis, it may lead to gingival bleeding and inflammation in certain cases.
- *HIV-associated gingivitis*: in children infected with HIV, rare cases of gingival candidiasis can arise.
- *Measles* (*and rubella*): can manifest with Koplik spots and inflammation that may impact the gingiva.
- *Human papillomavirus* (*HPV*): can cause oral papilloma, which may sometimes affect the gingival tissues.

Other less frequent non-viral infections include

- *Streptococcal gingivitis*: an uncommon but significant infection caused by group A streptococci. Features include painful, bright red, oedematous gingiva with potential systemic symptoms.
- *Acute pericoronitis*: inflammation around partially emerged teeth (especially third molars), more common in adolescents. It causes pain, swelling and difficulty opening the mouth.
- *Candidiasis* (*oral thrush*): fungal infection presenting as red and/or white patches on gingiva and other oral mucosa, which can become painful if scraped off. Such cases must be investigated for underlying immunosuppression.

In general, acute infections in children and adolescents can be very distressing, especially for young children, due to the pain and discomfort that often lead to decreased fluid intake and an increased risk of dehydration. Treatment primarily focuses on supportive care, including pain management, maintaining hydration and preventing secondary infections. In severe cases of dehydration, hospitalisation for parenteral fluid administration should be considered. An additional factor to consider is preventing infection of family members.

5.17 | Other Gingival Conditions Affecting Children and Adolescents

5.17.1 | Reactive Processes—Epulides (Singular: Epulis)

These are localised, non-neoplastic growths that affect the gingival tissues. Although rare in children, they can manifest as fibrous epulides, which are typically benign reactive lesions resulting from chronic irritation or trauma. Additionally, pyogenic granuloma (a vascular epulis), although usually associated with pregnancy, can also occur during puberty. Management usually involves surgical excision with histopathological examination to confirm the diagnosis. Removing potential irritants is crucial to prevent recurrence. Most epulides have a good prognosis, although some types may recur if not completely removed or if giant cells are present histologically.

5.17.2 | Gingival Pigmentation

Pigmentation affecting gingival tissues can be classified into several categories based on their aetiology and clinical presentation. They are typically physiological, resulting from melanin deposition by melanocytes in the basal layer of the epithelium. This is common in children and adolescents with darker skin tones, although it may also reflect the use of medications (such as antimalarials) and usually presents as a blue-grey to brownish discolouration. Other rare causes may include exposure to heavy metals such as lead or arsenic or pathological pigmentation such as a naevus or oral melanoma, as well as systemic conditions such as Peutz-Jeghers syndrome or neurofibromatosis. Clinical evaluation of gingival pigmentation should involve comprehensive history-taking, visual examination and, occasionally, a biopsy for a definitive diagnosis, particularly for lesions that are irregular, rapidly changing or suspicious.

5.17.3 | Traumatic Lesions

Traumatic lesions affecting gingival tissues typically fall into several categories based on their causes. They are most often due to mechanical trauma (abrasion or laceration following tooth brushing or self-inflicted, e.g., from habits like nail-biting or pencil-biting). Lesions secondary to chemical factors (e.g., reactions to mouthwashes or other drugs) or thermal trauma (e.g., burns) can also appear, although less frequently. Traumatic gingival lesions typically present with pain, bleeding, inflammation and, sometimes, tissue necrosis. The presence of ulcerations, erosions, gingival clefts and gingival recession is common. More rarely, gingival enlargement may occur as a response to chronic trauma. Treatment focuses on eliminating the causative factor/s, supporting tissue healing and preventing recurrence through patient education and adjusted oral hygiene techniques.

6 | Periodontitis and Other Periodontal Conditions in Systemically Healthy Children and Adolescents

The remit of Working Group 3 and the scope of the systematic review commissioned to Davies and Shapira (Eshkol-Yogev et al. 2025) included those periodontal conditions documented in the 2018 Classification (Caton et al. 2018) (Table 8).

6.1 | How Can the Section on Periodontitis and Other Periodontal Diseases and Conditions of the 2018 Classification Be Used in Children and Adolescents up to 18 years and Does It Need Adaptation?

Overall, the section on periodontitis and other periodontal diseases of the 2018 Classification (Caton et al. 2018) can be applied to children and adolescents.

However, the specific features of the primary and mixed dentitions may suggest the need to include specific conditions within the category of periodontitis, along with their case definitions.

6.2 | In the Section on Periodontitis and Other Periodontal Diseases and Conditions, What Are the Proposed Modifications of the 2018 Classification to Be Used in Children and Adolescents up to 18 years?

The following changes/modifications are suggested:

- Include specific type/s of periodontitis for children, with primary and/or mixed dentitions, with its/their case definition/s.
- Include Noma as part of necrotising periodontal diseases in children with a severe and long-term compromised immune system.

6.3 | Are Periodontitis and Other Periodontal Diseases Different (In Terms of Aetiology, Epidemiology, Associated Factors, etc.) in Children and Adolescents up to 18 years, When Compared With Adults?

6.3.1 | Periodontitis

Prevalence. According to the systematic review (Eshkol-Yogev et al. 2025), the criteria used are very heterogeneous, precluding robust conclusions from being made, and with the risk of unwittingly including children with systemic conditions (reportedly systemically healthy). In addition, periodontal evaluation

TABLE 8 | Conditions of the 2018 Classification (Caton et al. 2018) within the scope of the systematic review commissioned for Working Group 3 (Eshkol-Yogev et al. 2025).

Diseases/conditions	Level 1	Level 2	Level 3
Periodontitis	1. Necrotising periodontal diseases	a. Necrotising periodontal diseases in chronically, severely compromised patients	
		b. Necrotising periodontal diseases in temporarily and/or moderately compromised patients	i. Necrotising gingivitis
			ii. Necrotising periodontitis
			iii. Necrotising stomatitis
	2. Periodontitis as a manifestation of systemic diseases (covered by Working Group 1, see Section 4)		
	3. Periodontitis	Stages I–IV	
		Grades A–C	
		Localised, generalised, molar–incisor distribution	
Periodontal manifestations of systemic diseases and developmental and acquired deformities and conditions	1. Systemic diseases and conditions affecting the periodontal supporting tissues (covered by Working Group 1, see Section 4)		
	2. Other periodontal conditions	a. Periodontal abscesses	i. Periodontal abscess in periodontitis patients
			ii. Periodontal abscess in non-periodontitis patients
		b. Endodontic-periodontal lesions	i. Endo-periodontal lesion with root damage
			ii. Endo-periodontal lesion without root damage
	3. Mucogingival deformities and conditions around teeth	a. Gingival phenotype	
		b. Gingival/soft-tissue recession	
		c. Lack of gingiva	
		d. Decreased vestibular depth	
		e. Aberrant frenum/muscle position	
		f. Gingival excess	
		g. Abnormal colour	
		f. Condition of the exposed root surface	
	4. Traumatic occlusal forces	a. Primary occlusal trauma	
		b. Secondary occlusal trauma	
		c. Orthodontic forces	
	5. Prosthesis- and tooth-related factors	a. Localised tooth-related factors	
		b. Localised dental prostheses-related factors	

was generally secondary to other assessments, such as for dental caries.

The majority of studies focusing on the prevalence of periodontitis were conducted in subjects aged 12 years or older from different geographical locations. In most studies, the prevalence of periodontitis was <2% (10 studies), while four additional studies reported higher values (3.00%–30.36%), and still higher values when periodontitis was defined as radiographic bone loss (43%). This may be due to assessments undertaken in specific populations or to methodological issues. It has been reported that Black children have a higher prevalence of periodontitis than White children (1.5% vs. 0.3%, respectively) and that children living in rural areas have higher prevalence than those living in urban areas (0.59%–2.9% vs. 0.36%, respectively).

Risk Factors/Indicators. Evidence, derived mainly from cross-sectional studies, has identified some potential risk factors/indicators:

- *Microbiological factors*: presence of *Aggregatibacter actinomycetemcomitans* JP2 clone has been identified as a risk factor for disease progression in a prospective cohort study. Other bacterial species evaluated were *Porphyromonas gingivalis* fimA, or *Tannerella forsythia*.
- *Local tooth-related factors*: such as a rough tooth surface, anomalies in the cementum, presence of caries lesions and dental calculus and eruption and exfoliation processes.
- *Tobacco smoking*: in nine studies in Finland, smoking tobacco was found to be statistically significantly associated with the prevalence of clinical attachment loss (CAL) ≥ 2 mm (odds ratio [OR] = 4.9 for girls and OR = 5.3 for boys, overall), which could also have been influenced by socioeconomic factors.

Other factors included in the systematic review (Eshkol-Yogev et al. 2025) but for which no significant association was reported were anterior open bite, obstructive sleep apnoea and the presence of fluoridated water.

Different factors may potentially impact on periodontitis onset and progression, including ethnicity, family aggregation (and potential evaluation of siblings), socioeconomic factors and wearing orthodontic appliances, and deserve further evaluation. It is also important to recognise that the occurrence of severe forms of periodontitis in childhood and adolescence is associated with a higher risk of developing the disease in the future, thus requiring meticulous patient follow-up (Bimstein et al. 2013).

Progression. It is unclear whether the progression of disease is faster in children/adolescents when compared with adults. Available evidence suggests that disease progression is faster in individuals aged 16–19 years than in younger individuals aged 14–16 years (Clerehugh et al. 1995). In addition, some factors have been identified that may help predict future disease progression: presence of subgingival calculus, bleeding, plaque and gingival colour change (Clerehugh et al. 1990; Clerehugh et al. 1995).

6.3.2 | Necrotising Periodontal Diseases

Two types of necrotising periodontal diseases are considered (Herrera et al. 2018; Papapanou et al. 2018):

- Necrotising periodontal diseases in chronically severely immunocompromised patients.
- Necrotising periodontal diseases in temporarily and/or moderately compromised patients.

With the exception of unmanaged HIV disease, necrotising periodontal diseases in chronically, severely immunocompromised patients are exclusive to children: children, typically 2–6 years, suffering from severe malnourishment, extreme living conditions or severe infections that may even constitute a severe or even life-threatening situation. This has been found almost exclusively in very defined areas of tropical Africa.

Prevalence. For necrotising periodontal diseases in chronically and severely compromised patients

- Necrotising gingivitis, in Nigeria and Senegal, 0.0%–27.1%.
- Noma, in Nigeria, 0.005%–3.6%.

For necrotising periodontal diseases in temporarily and/or moderately compromised patients, prevalence has been estimated at 6.7% in 12–17-year-olds in Chile (Lopez et al. 2002). Prevalence may be higher in adolescents, increasing with age, because of the presence of potential predisposing factors.

Predisposing Factors. For necrotising periodontal diseases in chronically, severely compromised patients:

- *Malnutrition*: (markedly reduced mean plasma and serum concentrations of retinol, total ascorbic acid, zinc and albumin, or very marked depletion of plasma retinol, zinc and ascorbate; or conversely, significantly increased saliva levels of albumin and cortisol and plasma cortisol concentrations) is a major factor in the development of necrotising gingivitis and Noma, as well as social deprivation and poor oral hygiene.
- *Age*: children aged 3–5 years are the more affected (OR = 3.9, 95% confidence interval [CI] 2.04–7.47) compared with 6–15-year-olds.
- *In addition*: extreme living conditions (living in substandard accommodation, exposure to debilitating childhood diseases, living near livestock, poor oral hygiene, limited access to potable water and poor sanitary disposal of human and animal faecal waste) or severe infections (measles; herpes viruses such as cytomegalovirus, Epstein–Barr virus-1 or herpes simplex virus; chicken pox; malaria and febrile illness) are also relevant predisposing factors.

For necrotising periodontal diseases in temporarily and/or moderately immunocompromised patients, smoking, cannabis use, psychological stress, limited intake of micronutrients and poor quality of sleep may be the most relevant predisposing factors in adolescents.

6.3.3 | Periodontal Abscesses

Periodontal abscesses in periodontitis patients are an infrequent finding in systemically healthy children and adolescents because of the low prevalence of periodontitis.

For periodontitis patients with undiagnosed or poorly controlled diabetes, or other systemic diseases (see Section 4), or taking systemic antimicrobials for non-oral reasons, periodontal abscesses may occur.

Periodontal abscesses in non-periodontitis patients may occur more frequently, especially those associated with foreign body impaction, habits (fingernails), orthodontic factors or gingival overgrowth.

6.3.4 | Mucogingival Conditions and Deformities Around Teeth: Gingival Recession

Prevalence. The criteria employed are very heterogeneous, precluding robust conclusions, with some studies evaluating photographs or dental casts.

Most studies reported 0%–20% prevalence of gingival recession in children and adolescents, although higher values have been reported in incisors in 9-year-old children with anterior crossbites (prevalence = 40%; mean = 1.7 mm; standard deviation SD = 0.08 mm) or in 12–16-year-old adolescents with maxillary canines with a high vestibular eruption (33%), versus 4% in normal eruption. Additionally, an increase in the prevalence of gingival recession with age has been shown. It is important to consider that the final position of the gingival tissue is not stable until 12 years of age, or even older for second permanent molars, and true recession is when the cemento-enamel junction is visible beyond the gingival margin.

Orthodontic therapy has been reported to have a dual effect. On one side, it increases the prevalence of gingival recession (from 0%–10% prevalence before therapy to 13.6%–37.5% after therapy), but it can also reduce the pre-existing recession in incisors with cross-bite (1.7–0.6 mm). It is important to note, however, that the dynamic nature of oral tissue development in paediatric patients may facilitate spontaneous tissue recovery (Eshkol-Yogev et al. 2025).

Predisposing Factors. The following factors have been considered in the papers identified by the systematic reviews:

- Anatomical factors, including high frenal involvement, reduced width of keratinised mucosa and thin gingival phenotype.
- Incorrect brushing technique and high frequency of brushing.
- Oral hygiene and inflammation. Higher plaque scores have been associated with gingival recession. Dental biofilm control during orthodontic therapy is particularly important in individuals with a thin gingival biotype to reduce the risk of inflammation and its consequences, such as a greater risk of gingival recession.
- Smokeless tobacco use (e.g., 'snus').
- Malocclusions, including crossbite, anterior crowding, labial tooth position.
- Ectopic eruption.
- Habits and self-induced gingival recessions, including oral piercings, fingernails, sharp toys or pencils, and so on, which could damage the gingiva.
- Traumatic dental injuries.
- Orthodontic therapy (see the following sections).

Progression. Increased rates of progression of gingival recession have been associated with orthodontic therapy, thin gingival phenotypes and traumatic toothbrushing habits.

Role of Orthodontic Therapy. A higher prevalence of gingival recession has been observed in patients treated with orthodontic therapy. Some orthodontic movements may present a risk for developing gingival recession, such as those in which the root can be moved outside its bony envelope, and this can be observed during and after orthodontic treatment. Follow-up studies indicate that gingival recession risk will continue in the post-orthodontic period, and other factors may also be implicated during this time.

6.3.5 | Other Conditions

Mucogingival Conditions and Deformities Around Teeth: Decreased Vestibular Depth, Aberrant Frenum, Gingival Excess, Abnormal Colour. No information was retrieved on other mucogingival conditions and deformities around teeth, besides gingival recession, although aberrant frenal attachments have been discussed as a predisposing factor because they may limit access to effective oral hygiene. Altered passive eruption and gingival excess may be relevant conditions in children and adolescents. See Section 5 for further information on gingival enlargement.

Endodontic-Periodontal Lesions. No information was retrieved on endodontic-periodontal lesions.

Traumatic Occlusal Forces (Including Orthodontic Forces). No information was retrieved on traumatic occlusal forces.

Prosthesis- and Tooth-Related Factors. The use of ill-adapted stainless steel crowns and restorations on second primary molars has been associated with a poorer periodontal condition in the first permanent molar.

6.4 | Is the Management of Periodontitis and Other Periodontal Diseases Different in Children and Adolescents up to 18 Years, Compared With Adults?

6.4.1 | Periodontitis

Screening and diagnosis. For *clinical assessment*, periodontal probing allows the measurement of bleeding on probing, probing depth, gingival recession and CAL, relative to the position of the cemento-enamel junction (CEJ). Information on periodontal probing should be interpreted similarly to that in adults when teeth are fully erupted. When teeth are not fully erupted, the degree of eruption and other factors may affect the interpretation of the measurement.

Periodontal assessments should be performed at least once a year.

Different approaches are taken (Clerehugh and Kindelan 2021) according to the age and type of dentition (primary dentition, first or second phase of mixed dentition, permanent dentition).

The following simplified periodontal screening examination is to be performed for those aged < 18 years:

- For the age group 0–6 years, no probing except when unusual findings are detected (e.g., tooth mobility).
- For those aged 7–11 years, screening of periodontal tissues may include gentle probing (Clerehugh and Kindelan 2021),

aiming at identifying subgingival and supragingival calculus and bleeding of the first permanent molars and upper right and lower left central incisors (Ainamo index teeth, Ainamo et al. 1984). If periodontal probing is not considered feasible (e.g., because of the child not cooperating), look for other signs including tooth mobility, unusual levels of visual inflammation or accumulation of dental biofilm/dental calculus. If unusual findings are detected, then periodontal probing should be performed to confirm or rule out the presence of periodontitis. This may involve behavioural strategies to acclimatise the child to treatment or referral to a specialist.

- For those aged 12–17 years, periodontal probing may be performed as for adult screening but on the six designated Ainamo teeth (which would have generally fully erupted in this age range). Full-mouth periodontal assessment is indicated if periodontitis is suspected after screening.

For the *role of radiographs*, when taken for other medical and dental indications, bone levels should be assessed. If unusual findings are detected, further periodontal assessment should be performed, including periodontal probing. In cases of localised periodontitis, periapical radiographs are the first choice as a complementary diagnostic tool, while panoramic radiographs are the choice for generalised periodontitis in children and adolescents (Kuhnisch et al. 2020).

Additional factors to be considered: There are different elements to be explored in the medical and dental history:

- *Elimination of the potential for associated systemic conditions*: see Section 4 on referral to paediatrician and timely blood testing.
- *Family history*: when a diagnosis of periodontitis is made in a child or an adolescent, the relatives (parents, siblings) should be assessed periodontally.
- *Evaluation of systemic or environmental risk factors/indicators*: smoking, vaping, diabetes.
- *Evaluation of local risk factors/indicators*: tooth surface, orthodontic appliances.

Future developments may include advanced diagnostic approaches, including those for the initial assessment of increased risk; for example

- Biomarkers.
- Microbiological testing.
- Questionnaires.

With regard to *staging, grading* and *extent*, the first two are valid in fully erupted permanent teeth. Conventional grading may not be useful in children with primary and/or mixed dentitions, and the same is true for staging.

Therapy. Chronological age and dental age need to be considered (ages 0–6, 7–11, ≥ 12 years). In general, current guidelines should be followed (Herrera et al. 2022; Sanz et al. 2020), although the level of cooperation and proximity of the periodontal lesions to the developing permanent teeth should be carefully assessed, as well as the management of behaviour (e.g., need for sedation), the need for involvement of caregivers and the potential for healing of tissues in children.

In *step 1* of care, supragingival biofilm control by the individual (e.g., oral hygiene instructions) needs to be adapted to the patient's age, and caregivers should be involved. Toothbrushing (manual or powered) needs to be supervised until such time that the children can undertake appropriate oral hygiene independently. The same is true for interdental cleaning (considering the absence of contact points in the primary and mixed dentitions). Supragingival biofilm control by the clinician may be performed with hand and/or powered instruments. Step 1 should also include risk factor control (e.g., smoking cessation in adolescents), considering the risks associated with passive smoking and the risk behaviours of the caregivers.

In *step 2*, subgingival instrumentation may be performed with hand and/or powered instruments, usually under local anaesthesia, on a quadrant-wise or full-mouth approach depending on the extent of disease and the cooperation and need for behavioural management. For adjunctive therapies, different adjunctive products have been tested:

- For systemic antimicrobials, very limited evidence is available (mainly case series), and the one randomised control trial identified did not demonstrate additional benefit (doxycycline). Therefore, owing to concerns with public health and antimicrobial resistance, the use of systemic antimicrobials must be avoided or strictly limited to the most severe cases. Here, caution should be exercised with tetracyclines, including doxycycline, in those under 3 years of age because of the risk of tooth staining.
- Local application of antimicrobials (sodium hypochlorite, povidone iodine) or adjunctive use of mouth rinses (chlorhexidine, guava) has also been tested, without evidence to support their use.

Strategic extractions of the involved teeth can also be considered within step 2.

The *re-evaluation following step 2* is performed within 8–12 weeks, to understand whether the desired endpoints have been achieved, using the same endpoints proposed for adults (endpoints for primary/mixed dentition). If they have been achieved, patients should join a supportive periodontal care programme. If not, *step 3* should be considered, with re-instrumentation or periodontal surgery as potential interventions. For the primary and mixed dentitions, surgery should not be performed. In the permanent dentition and with fully erupted teeth, additional healing time (e.g., 8–12 weeks) can be allowed, and careful consideration should be given to implementing re-instrumentation. Specific consideration should be given to intrabony defects with clear regenerative potential (Sanz et al. 2020), for which surgical approaches may be more adequate, while re-instrumentation may impair that regenerative potential. In addition, and only for teeth with hopeless prognosis, the auto-transplant of third molars may be an alternative to consider in 16–17-year-old adolescents.

For secondary prevention (*supportive periodontal care*), interventions are identical to those in step 1, and again it is important to consider patient behaviour, which may be different, and the need to involve parents, guardians and caregivers in prevention, although this need may be reduced in adolescents. It has been established that there is a high risk of recurrence when periodontitis onset is in primary dentition.

6.4.2 | Necrotising Periodontal Diseases

Diagnosis. It is advised to follow the case definitions within the 2018 Classification for necrotising gingivitis, periodontitis and stomatitis (Herrera et al. 2018; Papapanou et al. 2018).

Noma is defined by the WHO (World Health Organisation 2016) as a rapidly progressing, severe gangrenous disease of the mouth and the face, which starts as a soft-tissue lesion (a sore) of the gums and develops into acute necrotising gingivitis that progresses rapidly, destroying the soft tissues and further progressing to involve the hard tissues and skin of the face. Clinical criteria for diagnosis differ according to the stage of the disease, from 0 to 5: respectively simple gingivitis, necrotising gingivitis, oedema, gangrene, scarring and sequelae.

Therapy. For necrotising periodontal diseases in chronically and severely compromised patients, local debridement, antiseptic mouth rinses and systemic antimicrobials have been used for treatment, combined with measures to control nutritional deficiencies, extreme living conditions and co-morbidities. Improvements in oral hygiene may be crucial to avoid recurrence, which may be challenging in a pre-cooperative age group (3–5-year-olds).

For necrotising periodontal diseases in temporarily and/or moderately compromised patients, local debridement with adjunctive antiseptics and control of predisposing factors should be the selected interventions.

Prevention. Secondary prevention is based on controlling predisposing factors, including the underlying periodontal disease, either biofilm-induced gingivitis or periodontitis. Recurrence of disease is frequent (21.4% for necrotising gingivitis, 11.1% for Noma), as previous necrotising periodontal disease is a relevant predisposing factor.

6.4.3 | Periodontal Abscesses

Diagnosis. It is advised to follow the case definitions of the 2018 Classification for periodontal abscesses (Herrera et al. 2018; Papapanou et al. 2018).

Therapy. In the absence of scientific evidence in this age group, the following principles may be followed. If the foreign body is still in place, it should be removed through incision, drainage and debridement. According to expert opinion, pus collections amenable to drainage seem to be less frequent in children than in adults.

In case of periodontal abscess in a young patient with periodontitis, the underlying condition should be managed as for an adult.

6.4.4 | Mucogingival Conditions and Deformities Around Teeth: Gingival Recession

Diagnosis. The diagnosis of gingival recession should be made clinically with a periodontal probe, measuring the distance from the gingival margin (apical to the CEJ) to the CEJ. Most gingival recessions identified in this age group are not associated with clinical attachment loss due to periodontitis. During tooth eruption, the interpretation of clinical findings may be difficult.

Therapy. Mucogingival surgery with recession coverage and/or keratinised tissue gain is not advised in children because of the lack of gingival maturity. In addition, patient cooperation should be considered, including compliance with oral hygiene instructions. Monitoring, along with oral hygiene methods, is necessary to evaluate stability. In adolescence, considerations may be different.

For specific cases, the following may be considered:

- Certain orthodontic tooth movements can help in controlling progression. In cases where teeth are in an anterior cross-bite and recession is present, orthodontic treatment has been shown to arrest the progression.
- Certain orthodontic movements can increase the risk of developing or worsening gingival recession, and therefore preventive mucogingival surgery may be considered, aiming to make the gingival tissues thicker, although some studies have not shown benefits from this approach.
- In cases with a frenal involvement, effective oral hygiene and professional cleaning have been shown to maintain a stable situation.
- Efforts should be made to control negative oral health habits.

6.4.5 | Other Conditions

Mucogingival Conditions and Deformities Around Teeth: Decreased Vestibular Depth, Aberrant Frenum, Gingival Excess, Abnormal Colour. No information was retrieved on other mucogingival conditions and deformities around teeth, apart from gingival recession. Similar principles as those presented in Section 6.4.4 should be followed. Aberrant frenula have been discussed previously.

Endodontic-Periodontal Lesions. No information was retrieved on the diagnosis and treatment of endodontic-periodontal lesions. Therefore, the same principles used for adults should be followed in the fully erupted permanent dentition where the tooth apex is closed. In cases of an open root apex, specific considerations should be made.

Traumatic Occlusal Forces (Including Orthodontic Forces). No information has been retrieved on the diagnosis and treatment of traumatic occlusal forces. Therefore, the same principles used for adults should be followed in the fully erupted permanent dentition.

Prosthesis- and Tooth-Related Factors. Plaque retentive factors, either prosthesis- and/or tooth-related, should be controlled. For example, ill-adapted pre-formed crowns on primary and permanent teeth have been associated with a poorer periodontal condition, and therefore the adaptation of the prosthesis should be improved and/or the crown replaced.

Author Contributions

Iain Chapple, Dominique Declerck, Sotiria Gizani, David Herrera, Phoebus Madianos and Mariano Sanz (listed in alphabetic order here) substantially contributed to the conception and design of the project, to the interpretation of data and to the drafting and critical review of the manuscript. The EFP Focused Workshop participants (listed as co-authors in alphabetic order) significantly contributed by critically reviewing the consensus report and by participating in the workshop discussions. All authors approved the final version of the manuscript.

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Conflicts of Interest

Individual potential conflicts of interest forms were completed by all participants and are available on file at the European Federation of Periodontology. No financial or non-financial interests that were directly related to the work submitted for publication were declared. David Manton is editor in chief of the European Archives of Paediatric Dentistry, and apart from assisting in recommending two suitable reviewers on behalf of the EAPD, with whom he has or had no personal or professional relationship, had no further involvement in the review process.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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