Growth and development considerations in the diagnosis of gingivitis and periodontitis in children

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Abstract

Increasing information emphasize the relevance of the prevention, early diagnosis and early treatment of periodontal diseases in children. In order to avoid erroneous diagnosis and unnecessary treatments, the pediatric dentist is required to differentiate between pathologic processes and normal changes that take place in the periodontum with age. The present review outlines structural and functional changes of the periodontal structures, the establishment and maturation of the oral microflora and immune defense reactions to periodontal pathogens in children and adolescents. The age-related tendency to develop gingivitis, that is evident in children and adolescents, may be related to changes in the bacterial composition of the dental plaque, the inflammatory cell response, hormonal changes, morphological differences, tooth eruption and shedding. The hormonal influence on the gingival tissues and the composition of the dental plaque are of particular relevance during puberty. Large ranges for the prevalence of attachment loss, periodontitis or destructive periodontal disease in children and adolescents have been reported. The variance in values may be related to population characteristics, method of examination or diagnostic criteria that may include measurements of attachment loss and distances from the cementoenamel junction to the alveolar bone crest, both of which may be either physiological or pathological. The pediatric dentist should be able to diagnose gingival inflammation, attachment loss or distances from the cementoenamel junction to the alveolar crest which are out of proportion to the child's age and the amount of dental plaque. These may be indicative of a high susceptibility to periodontal diseases or reflect systemic conditions that affect the periodontum.(Pediatr Dent 21:186-191, 1999)

The relative lack of information and unawareness of existing information on periodontal health and diseases in children and adolescents may encourage pediatric dentists to utilize data related to the adult periodontum. However, this compromise may be inadequate when biological changes that take place during childhood and adolescence are not taken in consideration (i. e. the structural and functional changes of the periodontal structures during the eruption and exfoliation of teeth,[1-4] the establishment and maturation of the oral microflora,[5-8] and the gradual development of the immune defense system[9]). Therefore, the purpose of this review is to summarize information on these subjects, which is relevant for the prevention, diagnosis, and treatment of gingivitis and periodontitis in children.

Gingivitis

Epidemiological, clinical, and histologic studies indicate that there is an age-related tendency to develop gingivitis. The severity of gingivitis is less intense in children than in adults with similar amounts of dental plaque.[10, 11] Epidemiological studies report a low prevalence of gingivitis during pre-school age, followed by a gradual increase in prevalence reaching a peak around puberty.[12-16] Insignificant signs of gingivitis are noted in pre-school children when oral hygiene is discontinued.[17] There is a positive correlation between age and the dimension of inflamed gingival tissue areas in the human primary dentition.[18] There is no correlation between the amount of plaque and the dimension of inflamed gingival tissue in the human primary dentition.[19]

Although it has been suggested that the age-related tendency to develop gingivitis may be related to differences in the amount of plaque accumulated,[12, 20] results obtained under comparable plaque amounts at different ages indicate that other factors are involved.[10, 11]

Table 1. Differences in Microbial Composition of Dental Plaque at Different Ages

B. melaninogenicus (Bailit et al. 1964)[5]	children < adults
Bacteroides species (Kelstrup 1966)[6]	children < adults
Leptotrichia (Moore et al. 1984)[21]	children > adults
Capnocytophaga (Moore et al. 1984)[21]	children > adults
Selenomonas (Moore et al. 1984)[21]	children > adults
Bacteroides species (Moore et al. 1984)[21]	children > adults
Fusobacterium (Moore et al. 1984)[21]	children < adults
Eubacterium (Moore et al. 1984)[21]	children < adults
Lactobacillus (Moore et al. 1984)[21]	children < adults
Black pigmented *bacteroides* (Delaney et al. 1986)[22]	children < adolescents
Eikenella corrodens (Delaney et al. 1986)[22]	children < adolescents
Capnocytophaga (Wojcicki et al. 1987)[23]	children < adolescents (females)
Eikenella corrodens (Wojcicki et al. 1987)[23]	children < adolescents

Bacterial composition of dental plaque

The microbiota of the oral cavity is dependent on a complex of ecological principles (host characteristics, diet, bacterial adhesion, bacterial transmissibility, etc.) which change as the individual grows and matures.[7] Therefore, significant differences in the presence and proportions of microorganisms in the dental plaque of children, adolescents, and adults are evident (Table 1).[5-8, 21-27] Of special interest to the pediatric dentist are the changes in the dental plaque microbial composition which are related to puberty, and are presented in a subsequent section in this manuscript.

Inflammatory cell response to dental plaque

Periodontal diseases are the expression of the inflammatory reaction of the tissues to microorganisms and their products Their manifestations are dependent on the interaction of several local and systemic factors.[28-38] As these factors change through life, chronically inflamed gingiva in children has the characteristics of an early lesion, while in adults it presents as an established periodontal lesion.[28, 32]

In addition, it has been indicated that some serum antibody levels to periodotopathic bacteria may change with age. Mouton et al.[30] indicated that IgG antibody activity to *Porphyromonas gingivalis (P. gingivalis)* may be found in serum samples from umbilical cords (mean=35.2 ELISA units). These antibodies of maternal origin are short living and only traces are seen in infants aged less than six months, but increase from the primary dentition (mean=7.9 ELISA units) to the mixed dentition period (mean=29.6 ELISA units) and adults above 26 years of age (mean=55.7 ELISA units). IgM levels to *P. gingivalis* follow a different pattern. They are not detected in serum from umbilical cords, but low levels are found in infants less than six months old (mean=1.9 ELISA units), and a small increase takes place until age 12 years (mean=3.7 ELISA units), and this value is similar to the one found in adults (mean=3.4 ELISA units).[30] Tolo and Schenk[33] examined the IgG, IgA, and IgM levels to extracts of six microorganisms (*P. gingivalis*, *Bacteroides ureolyticus*, *Capnocytophaga ochracea*, *Eubacterium sebarreum*, *Fusobacterium nucleatum,* and *Selenomonas sputigena*) in individuals aged 3 to 81 years, and found that in subjects with a healthy periodontum, IgG and IgA scores correlated with age up to age 20. Bimstein and Ebersole[36] examined the IgG and IgM serum levels to ten oral microorganisms, in children and young adults with various degrees of gingivitis. Their findings indicated significant differences between children and adults for all the microorganisms in the IgM values, and for most organisms in the IgG values. When the severity of the disease was also taken in consideration, the differences in serum levels for most organisms were not significant (mostly in the IgM values). This suggests that age had a stronger influence than severity of the disease on the antibody levels. The changes in serum antibody levels to periodontopathic bacteria that are related to puberty are presented in the following section.[36]

Puberty

A peak in the prevalence and severity of gingivitis around the age 9-14 years, which coincide with pre-puberty and puberty, has been reported in several manuscripts.[13, 14, 39, 40] The correlation between the degree of gingival inflammation and parameters describing pubertal maturation (testicular volume and Tanner scores for breast development) suggests that there is hormonal influence on the gingival inflammatory process concomitant to puberty.[22, 40] Moreover, it has been reported that sex hormones may induce endothelial damage and increased vascular permeability,[41] affect the recruitment of leukocytes to inflamed tissue,[42] influence the formation of granulation tissue[43] and facilitate changes in the composition of subgingival flora, which are evidenced by:

- an increase in the proportion of total colony forming units (CFU) of gram negative anaerobes from prepuberty to puberty and the postpubertal period[23]
- an increase in the percentage of CFU of black pigmented Bacteroides species (BPB) in females who are soon to experience menarche[22]
- an increase of total CFU of BPB from prepuberty to puberty[23]
- a higher percentage of CFU of *B. intermedius* at puberty than in prepuberty or postpuberty;[23]
- an increase in the percentage of CFU of *Prevotella intermedia* and *Prevotella nigrescens* from prepuberty to puberty.[25]

While there is consensus related to the increase in CFU and serum antibody levels to periodontal pathogens towards or at puberty, there is conflicting evidence on the levels of BPB after puberty. Some studies indicate an increase in the percentages of CFU of BPB with age after puberty,[5, 6] others describe that the percentages of CFU of BPB species do not correlate with age, were similar, inferior, or nonexistent after puberty.[22, 23, 27]

Additional evidence that the microbiota of dental plaque may react specifically to increased availability of sex hormones in the dental fluid, are the changes related to puberty in the serum antibody titers to periodontopathic bacteria. These include:

- an increase in the IgG antibody titers to *P. gingivalis* from preschool children to early puberty in children with Down's syndrome[38]
- an increase in the IgM antibody titers to *Actinobacillus actinomycetemcomitans* from preschool children to early puberty in children with Down's syndrome[38]
- an increase in the IgG levels to *P. gingivalis* and *A. actinomycetemcomitans* related to puberty gingivitis[24]
- a correlation between serum levels of testosterone in boys and estradiol and progesterone in girls with serum antibody levels of *Prevotella intermedia* and *Prevotella nigrescens*.[25]

An interesting paper reported that six years after puberty, children who had marked puberty gingivitis, when compared to children who had lower degrees of puberty gingivitis, had higher gingival bleeding scores, increased sites with >3 mm attachment loss, and were the only ones with *Spirochetes* and *A. actinomycetemcomitans* in their plaque.[27]

Morphological differences between the gingival tissues of primary and permanent teeth

The inflammatory cell infiltrate resides mostly at the coronal portion of the free gingiva of the permanent dentition while in the primary dentition, the inflammatory lesion occupies a narrower tissue portion along the gingival epithelium.[18, 19, 44, 45] In addition, the junctional epithelium of the primary tooth

Table 2. Reported Prevalences of Periodontitis/Attachment Loss in Children and Adolescents

Author(s)	Age in years	Prevalence	Age of peak prevalence	Method of examination	Country
Jamison (1963)	5-14	25.2%	-	Probing	United States
Kesthelyi et al. (1987)	——-	94%	-	Staining•	Hungary
Saxby (1987)	15-19	0.1	-	Probing+radiographs	United Kingdom
Aass et al. (1988)	14	4.5	-	Radiographs	Norway
Bimstein et al. (1988)	4-17••	0-17.9	7	Radiographs	United States
Bimstein et al. (1993)	3-12	0-17.4	9	Radiographs	Israel
Sjödin et al. (1994)	7-9	2-4.5	9	Radiographs	Sweden
Bimstein et al. (1994)	5	2.1	-	Radiographs	New Zealand
Drummond et al. (1995)	4-14	0-42.9	10	Radiographs	New Zealand
Hansen et al. (1995)	15-17	0-35.7	-	Radiographs	Multinational

• Extracted primary human teeth. •• 18 year old excluded because of small size of sample.

gingiva has been found to be thicker than the one of the permanent tooth, and a thicker junctional epithelium may have a reduced permeability of the epithelial structures to bacterial toxins.[45]

Tooth eruption and exfoliation

The condition, often referred to as eruption gingivitis, may be caused by a greater risk of plaque accumulation in areas of shedding primary teeth and erupting permanent teeth, since oral hygiene may be difficult and even unpleasant to perform.[46] With age, there is an increase in the sulcus depth of the primary dentition,[4] and with the approximation of shedding the epithelial attachment migrates under the resorbing surface aiding in the eventual shedding of the tooth.[1] During tooth eruption, increased permeability of the junctional epithelium has been shown to take place in dogs,[47] and the junctional epithelium in rats may display degenerative changes at the site of fusion with the oral epithelium, indicating a weak point in the epithelial barrier.[48] Furthermore, the long dental epithelial attachment created by the fusion of the oral and dental epithelia, may prompt the formation of a deep pocket,[2] which may act as a niche for pathogenic bacteria.

The primary etiologic factor for gingival reactions associated with tooth eruption and puberty as well as mouth breathing is dental plaque accumulation. A proper oral hygiene regimen is usually sufficient to prevent these undesired gingival reactions. It should be stressed that there is no evidence that the enhanced gingival reactions described above implies an increased risk of developing destructive forms of periodontal disease (periodontitis). However, an increased gingival reaction may create an environment in the gingival sulcus which allows the development of more severe forms of periodontal diseases to occur.

Periodontitis

Periodontitis in children before puberty has been recognized for many years.[49, 50] However, little attention was paid to periodontitis in children with no systemic diseases until the clinical signs of periodontitis were characterized as a disease entity named prepubertal periodontitis.[49] Localized prepubertal periodontitis (LPP) has been described to appear in otherwise healthy children, and generalized prepubertal periodontitis (GPP) in children with a history of delayed umbilical cord separation, delayed wound healing, and as an oral manifestation of serious systemic diseases such as persistent peripheral blood leukocytosis, leukocyte adhesion deficiency, hypophosphatasia, neutropenia, Chediak-Higashy syndrome, leukemia, histiocytosis X, acrodinia, and Papillon Lèfevre syndrome.[51-54] Recently, a case with GPP and no systemic disease was reported;[55] however, there is still a possibility that the child had a systemic disease which was not detected.

A wide range in the values for the prevalence of attachment loss, periodontitis, or destructive periodontal disease in children and adolescents is evident in the literature (Table 2).[56-65] This wide range may be related to population characteristics (caries prevalence, socio-economic status, and ethnic origin), method of examination, and diagnostic criteria. It is possible that normal changes have been considered pathological.

Apical migration of the junctional epithelium

In a study in which the distance from the cemento-enamel junction (CEJ) to the most coronal attachment fibers was measured on extracted stained human primary teeth,[58] the authors con-

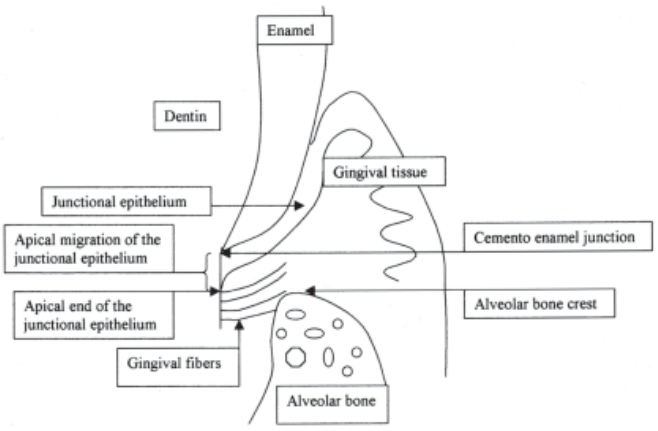

Fig 1. Diagrammatic presentation of the dento-gingival junction.

cluded that periodontitis in the primary dentition is not a rare phenomenon, since attachment loss of 0.26±0.32 mm was found in two-thirds of the tooth surfaces. The attachment loss was significantly larger at the buccal than at the lingual surfaces. Similar findings were found in a histologic study on human extracted primary teeth,[18] in which 0.28±0.06 mm apical migration of the apical end of the junctional epithelium was found in 53% of the sites. The apical migration was significantly larger at the buccal than at the lingual surfaces. The results of both studies may be compared since the most coronal attachment of the gingival fibers is located apical to the apical end of the junctional epithelium (Fig 1).[66] This small attachment loss, or apical migration of the junctional epithelium in the human primary dentition, has no clinical significance and does not necessarily witness the presence of periodontitis, since it may be physiological. Simultaneously to the process of root resorption, due to the normal shedding process, the epithelial attachment may proliferate in an apical direction.[67] In a clinical study,[56] in which the distance between the free gingival margin to the CEJ in primary teeth was measured, the author pointed out that he utilized the expression "destructive periodontal disease" to describe the condition in which the bottom of the gingival sulcus is located apical to the CEJ, a situation which may be a physiologic phenomena which precedes the exfoliation of the primary teeth. Apical migration of the junctional epithelium in primary teeth, may take place without relation to the histological evidence of gingival inflammation.[18, 19] In the primary dentition there is an increase in the length of the epithelial attachment with no change in the histologic sulcus depth, as the attachment encroaches onto the root surface.[18] Apical migration of the junctional epithelium has been related to a physiological apical shift of the dento-gingival junction concomitant to the eruption of human permanent teeth, and and increase in the distance from the cemento-enamel junction to the alveolar bone crest (ABC). [68, 69]

A non-linear increase in the distance from the CEJ to the ABC takes place with age,[70-74] this phenomenon is site specific and may be related to facial growth and attrition.[71, 72] During facial growth, the maxilla and mandible are displaced in an anterior and inferior directions (primary displacement), a "space" is created and bone remodeling takes place with a consequent vertical drift of the teeth.[75] Despite that it has been considered that tooth eruption brings the tooth to a definitive crown height above the gingiva and the bone,[76] the increase in the distance between CEJ and the ABC indicates that in the primary dentition, tooth eruption may take place in a faster rate than ABC deposition. An additional factor that should be taken in consideration is that an increase in the distance between the CEJ to the ABC may be related to adjacent exfoliating primary teeth or erupting permanent teeth.[77]

Various CEJ-ABC distances for different primary teeth and jaws might be considered as cut-off values for the radiographic diagnosis of alveolar bone loss.[73] Still, a 2 mm CEJ-ABC distance may be considered as the borderline for a "healthy" alveolar bone height in most cases. Distances of more than 2 mm may be normal with the approximation of exfoliation, and in primary teeth located adjacent to exfoliating primary teeth or erupting permanent teeth.[70-74, 77] In any case, the clinician should also take into consideration the status of the lamina dura over the ABC when diagnosing periodontitis in the primary dentition, as only complete absence of lamina dura may be indicative of the presence of periodontitis.[74]

Conclusions

The pediatric dentist should be aware of the age dependent reactivity of the gingival tissues to dental plaque, hormonal influence on the gingival tissues, physiologic apical migration of the gingival attachment in the primary dentition, and physiologic increase in the distance from the CEJ to the ABC in the primary dentition.

Considering these variables should enable the pediatric dentist to diagnose gingival inflammation, attachment loss, or CEJ-ABC distances which are out of proportion to age and the amount of dental plaque. These situations may be indicative of a high susceptibility to periodontal diseases or reflect systemic conditions which affect the periodontum.

The relevance of the prevention, early diagnosis, and treatment of periodontal diseases by the pediatric dentist is emphasized by the opportunity to establish a sound foundation for future comprehensive oral health, possibility for relatively simple treatment with an excellent prognosis, the possible connection between gingivitis or periodontitis during childhood and older ages.[27, 78, 79, 80]

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