Understanding periodontitis in adolescents: Historical background
Rodrigo López

Abstract

Different schools of thought had influenced the understanding of periodontitis among adolescents. A systematic literature search on this topic illustrates that studies available were characterized by an enormous variation in the methods used to address the question as to whether periodontitis among adolescents differ from clinical entities among adults. Disease classifications have changed numerous times during the last two decades and it is not clear how these new classification systems reflect improved knowledge regarding the nature of destructive periodontal diseases. This paper gives a brief review on the historical background of the periodontitis in adolescents.

Key Words: Adolescence; Periodontal Diseases; Periodontitis

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Current definition and classification of periodontitis reflect the lines of thinking of generations of clinicians and scientists. (1) The signs and symptoms from the periodontium were discovered from more than 2,000 years ago, but more comprehensive written accounts did not appear until the 18th century. Nevertheless, it is clear from the treatises of both Pierre Fauchard (2) and John Hunter (3) that tooth loss and tooth mobility associated with loss of “the gums, alveoli and teeth” characterized symptoms that could receive a disease name. (2)

John Hunter described two main conditions, one entailing bone loss as “a wasting of the alveolar processes, which are in many people gradually absorbed, and taken into the system” while the other was described as “a filling up of the socket at the bottom, whereby the tooth is gradually pushed out”. (3) It is also remarkable that he made an explicit point about the testability of disease characteristics against norms, even though they were ill defined. He stated “though the wasting of the alveoli at their mouths, and the filling up at their bottom, are to be considered diseases, when they happen early in life; yet it would appear to be only on account of a natural effect taking place too soon; for the same thing is very common in old age”. (3)

Despite both Hunter and Fauchard used nominalistic disease concepts, examples are available in their essays of a phraseology, which can be understood as representing an essentialistic disease concept. Hence, Hunter mentioned “the teeth made loose by the disease” and Fauchard noted that “diseases which are common to the gums … are the ones which destroy the teeth most frequently”. (2, 3) Both examples illustrate the predetermined idea that diseases can be active in producing signs and symptoms and illustrates how simply colloquial speech translates into an essentialistic disease concept, if due care is not applied. Not only did Hunter describe two diseases “of the alveolar processes”, he also suggested that they could have different causes. Hence, he mentioned that “these diseases arise often from visible causes” in which case treatment could be effective, but also suggested that an intractable form exist: “when the disease does not arise from a constitutional cause, which may be removed, but from a disposition in the parts themselves, I have seen little relief given by them … astringents”. (3) So, Hunter laid the basis for a controversy, which remained central to periodontology for almost two centuries: Inflammatory or degenerative?

For many decades after the essays of Fauchard and Hunter, the main dispute in periodontology concerned the causes of alveolar pyorrhea, a term created and widely used to describe periodontal signs and symptoms. During this period two different schools of thought can be identified: The European school of constitutional/systemic causes, according to which “pyorrhea may and does exist independently of foreign deposits” and the North American school of local causes, according to which “pyorrhea” resulted from “a sequence of local causes... chiefly... salivary calculus”. (4) However, towards the end of the 19th century another three schools of thought began to crystallize, mainly because of new methods became available. New histological techniques, dental radiology and microbiology were contributory to the addition of the school of atrophy and degeneration; the school of occlusal trauma; and the school of infectious causes to the list of schools of thought in the field of periodontology. (4)

Especially, the school of atrophy and degeneration was to gain a pronounced influence on the understanding of periodontitis, mainly as it concerns young individuals.
Gottlieb set out to answer the question: Does alveolar bone loss precede gingival ulceration and inflammation or is ulceration and inflammation the primary event, which secondarily brings about alveolar bone loss? Based on histological studies of jaw specimens from deceased persons, most of whom were rather young - faithful to their European origins - concluded that bone atrophy was the first and causal factor for alveolar pyorrhea. More specifically, Fleischmann & Gottlieb held that epithelial proliferation and pocket formation were not primary events, but occurred after atrophic or constitutional processes in the bone. They also alleged that gingival inflammation was an essentially unconnected feature, which might be observed only if a concomitant external irritation had imposed injury to the epithelial surface. If such inflammation was allowed to progress, suppuration might result.

The idea of the existence of a form of periodontitis that is special to young subjects is often attributed to Gottlieb. However, it is worth mentioning that nowhere did Gottlieb directly link his histological observations on diffuse atrophy with the young age of the persons providing the histological samples studied. Rather, the link seems to be indirect, from Gottlieb's statement that pyorrhea with marginal atrophy can be excluded as a diagnosis when the destruction affects young subjects with outstanding oral hygiene. It is thus clear that pyorrhea with marginal atrophy could be excluded in case of a young person with good oral hygiene, which by default meant that the diagnosis had to be diffuse atrophy. This shows that Gottlieb understood pyorrhea with marginal atrophy as a condition, which would not develop unless large amounts of deposits were also present. This understanding is quite similar to that formulated decades later by Baer who argued periodontosis as a distinct disease on the grounds that "the amount of destruction is not commensurate with the amount of local irritants present".

The concept of the existence of a form of periodontal disease that is singular to young individuals is better accredited to Wannenmacher. He described the features of periodontitis in four cases. In first case, the periodontal symptoms occurred concomitant with "keratoma hereditaria palmare et plantare", whereas no systemic afflictions. He diagnosed the other three cases as "Paradentitis marginalis progressiva" and made a point that inflammation and secretion were obvious features. He noticed that a preponderance of bone lesions residing in the incisor and first molar area and concluded that systemic diseases were rarely associated. Later he modified his standpoint, and concluded that a local lack of resistance is a prerequisite for the bone loss to occur. The combination of these observations with Gottlieb's concept of diffuse atrophy explains why periodontitis in children and adolescents was to be considered a degenerative disease for many decades. It is noteworthy, however, that nowhere did Wannenmacher state that this supposedly degenerative disease was an entity specific to young subjects. The reality is that the concept of a distinct periodontal disease entity happening in young subjects did not really change until the concept of a degenerative etiology was abandoned.

The name of the condition described by Gottlieb, diffuse atrophy, did not endure long time. In 1931, Becks made a division between paradentitis, a disease "which originates in the gum tissue in the form of gingivitis" and genuine paradentosis that "originates in the bony alveolus, perhaps in the form of an osteopathy". In 1942, Orban & Weinmann embraced this nomenclature, using the anglicized term periodontosis to designate this "non-inflammatory disease", and afterward for many decades periodontosis was used to refer to non-inflammatory degenerative changes of the periodontal tissues. Thus, periodontosis was considered a distinct disease entity, definitely different from periodontitis which was considered the sequela of gingivitis to the deeper periodontal tissues, and therefore of inflammatory source.

Although some were prepared to admit that inflammation was a dominating feature of late stages of periodontosis, they nevertheless maintained that the "early development of the condition [periodontosis], however, is of a degenerative nature and inflammation is a secondary occurrence". Based on a literature review covering the period from Gottlieb's 1920 paper, the 1951 Workshop on Periodontal Disease concluded that "It appears from the evidence to be found in the literature that periodontosis does exist as an entity ...".

As the 1960's were approached, adult periodontitis was considered to occur almost universally; and to progress at a slow continuous rate, thus increasing in severity with increasing age. Moreover, a firm belief had been established that gingivitis is the initial lesion to periodontitis. In the early 1960's, the well-known experimental gingivitis studies of Löe and coworkers established that accretion of dental plaque results in gingivitis and that gingivitis can be treated by means of plaque removal. These results were seen to deliver the final proof of the relationship between poor oral hygiene and periodontitis.
This concept, later called the “nonspecific theory in microbial etiology of inflammatory periodontal diseases” gained broad acceptance and the evidence for the existence of degenerative lesions was gradually questioned. Already the committee report of the 1951 Workshop on Periodontal Disease had acknowledged regarding periodontosis that “It is true, when the patient seeks treatment, that inflammation of the periodontal tissues is a dominating feature”.(10)

At the time of the 1966 Workshop in Periodontics it was agreed that the term periodontosis should be eradicated from the periodontal nomenclature, due to the lack of evidence to support the concept of degeneration. Even so, the committee of the meeting maintained the idea that a clinical entity different from adult periodontitis may occur in adolescents and young adults.(11)

In 1967, the term juvenile periodontitis was introduced by Butler to label early periodontal destruction mainly localized to the permanent first molars. Hence, Butler dismissed the term periodontosis arguing that the suffix “-osis” implied a degenerative source for which there was no evidence.(12) Baer took offset in the 1949 definition of periodontosis as a “degenerative noninflammatory destruction of the periodontium” and raised some worries about the definition.(7) His point was to support the concept of a distinct disease entity of degenerative origin, and he described a set of features, which he considered to distinguish this entity from periodontosis (Table 1).

<table>
<thead>
<tr>
<th>Feature</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset</td>
<td>During the circumpubertal period.</td>
</tr>
<tr>
<td>Affects more females</td>
<td>Than males.</td>
</tr>
<tr>
<td>Familial background</td>
<td>Present.</td>
</tr>
<tr>
<td>Lack of relationship</td>
<td>Local etiologic factors.</td>
</tr>
<tr>
<td>Arc-shaped loss of alveolar bone</td>
<td>From distal of 2nd bicusp to mesial of 2nd molar.</td>
</tr>
<tr>
<td>Rapid loss of alveolar bone</td>
<td>More than one tooth of the permanent dentition.</td>
</tr>
<tr>
<td>Primary teeth unaffected</td>
<td></td>
</tr>
</tbody>
</table>

Table 1. Features of periodontosis according to Baer.(7)

Baer defined periodontosis as a “...disease of the periodontium occurring in an otherwise healthy adolescent, which is characterized by a rapid loss of alveolar bone about more than one tooth of the permanent dentition”, and stated that “the amount of destruction is not commensurate with the amount of local irritants present”.(7) Thereby, Baer’s arguments effectively re-established periodontosis, not only as a degenerative disease, but also as a particular disease entity essentially confined to adolescence. Consequently, many epidemiological studies based on Baer’s definition have excluded subjects with systemic diseases, and/or large amounts of local deposits from the pool of eligible subjects.(13-15)

While the concept of a particular periodontal disease entity found in young subjects has survived until present day, the re-invoked notion of degeneration as the etiological factor did not survive for long. Hence, during the 1970’s, the belief in the existence of periodontosis as a distinct disease entity of degenerative etiology was gradually abandoned. Novel microbiological and histological methods were used to investigate periodontal lesions, and one set of evidence against the periodontosis concept was provided by Waerhaug who examined teeth extracted from supposed cases of periodontosis. He observed a “very high degree of congruence between the subgingival plaque front and the line of lost attachment on all the teeth”, and concluded that microorganisms played a role in these cases. His study on the effect of plaque control in similar patients, and reported that the cases responded positively to “total” plaque control, while “incomplete” plaque control resulted in “extremely rapid bone loss” and “eventually extraction”. These studies showed that subgingival dental plaque does play a role as a cause of periodontosis and Waerhaug therefore suggested the replacement of the name periodontosis with highly destructive juvenile periodontitis when referring to this disease.(16)

Another set of evidence against the degenerative etiology of periodontosis was delivered by investigators who conducted microbiological studies using innovative subgingival plaque sampling techniques, and culture methods. These investigators reported the presence of certain capnophilic gram-negative microorganisms, and anaerobic rods in cases of periodontosis/juvenile periodontitis, indicating that “some or all of these organisms play a significant role in the initiation and or progression of the pathologic process in periodontosis”.(17)

Ranney suggested that the qualitative differences in the microflora of the subgingival dental plaque might well explain the apparent lack of relation between the amount of tissue destruction and the amount of dental plaque characteristic of periodontosis / juvenile periodontitis.(17) Socransky took this idea further when he suggested that different forms of periodontal disease had specific microbial etiologies: “Current information... indicates that there are a number of periodontal diseases, with rather specific local bacterial etiologies and host parasite interactions that lead to different clinical
manifestations". (18) The microbiological observations were coupled with the suggestion made by Lehner et al. and later reported by others that juvenile periodontitis might also differ from adult periodontitis by the presence of a selective cell-mediated immunodeficiency. (18, 19) This, may be what led Waerhaug to suggest that an "unfavorable systemic factor" is present in juvenile periodontitis patients, resulting in a "specific imbalance in the host/parasite equilibrium which makes it possible for the subgingival plaque to make its way toward the apex". (16)

Taken together these studies effectively eliminated the idea of a degenerative etiology of juvenile periodontitis, and it is interesting to note that Baer distanced himself from the idea of a degenerative disease entity when he stated that "The suffix 'osis' has never etymologically meant 'degenerative' and does not reflect the etiology of a disease. Thus, what is needed is not necessarily a change in the term but an updated definition of the term". These comments illustrate a pronounced change of the focus of the debate, which turned towards the distinguishing features and defining characteristics for juvenile periodontitis. (7)

From the perspective of periodontitis in children and adolescents, the 1980's and 1990's have been characterized by many tries to integrate the increasing body of knowledge about the microbiology and immunology of periodontitis with the clinical features to 'advance' definitions and classifications. Hence, during the 1980's and 1990's at least 10 different systems for classification of periodontitis have been suggested. As previously discussed, the syndromic nature of periodontitis makes it impossible to express the 'true nature' of periodontitis by means of new classifications that incorporate the latest knowledge, since no diagnostic truth exists. New classifications merely correspond to changes in the syndrome definitions. (20) Page suggested a division between prepubertal, juvenile, rapidly progressive, and adult periodontitis (Table 2) on the basis of age at onset, intra-oral distribution of the lesions, rate of progression, as well as neutrophil functionality and microbial characteristics. (20)

| 1. Prepubertal periodontitis      |
| 2. Juvenile periodontitis         |
| 3. Rapidly progressive periodontitis |
| 4. Adult periodontitis            |
| 5. Acute necrotizing ulcerative gingivo-periodontitis |

Table 2. Classification of periodontitis Page & Schroeder. (20)

The classification embraced by the American Academy of Periodontology in 1986 (Table 3) did not acknowledge the existence of an entity named rapidly progressive periodontitis and held that prepubertal periodontitis was a sub-class within the major category of juvenile periodontitis. The major factors considered characteristic for the different disease categories were age at onset, intra-oral distribution and morphology of the lesions, functional defects in neutrophils or monocytes, rate of progression, and the response to treatment. (21)

| 1. Juvenile periodontitis         |
| 2. Adult periodontitis            |
| 3. Necrotizing ulcerative gingivo-periodontitis |
| 4. Refractory periodontitis       |

Table 3. The 1986 AAP classification of periodontitis. (21)

In 1988, Johnson et al. proposed a classification (Table 4), in which clinical features, such as age, intra-oral distribution and rate of progression, formed the sole basis for the distinction between the entities. (22)

| 1. Childhood periodontitis        |
| 2. Juvenile periodontitis         |
| 3. Post juvenile periodontitis    |
| 4. Adult onset periodontitis      |
| 5. Periodontitis associated with systemic diseases |
| 6. Traumatic periodontitis        |
| 7. Iatrogenic periodontitis       |

Table 4. Classification of periodontitis by Johnson et al. (22)

Also in 1988, Suzuki proposed a new classification (Table 5), which constituted an amendment to the classification outlined by Page and Schroeder. Suzuki argued that "Additional clinical observations in our laboratories during investigations on the mode of inheritance of juvenile and rapidly progressive periodontitis have suggested that further qualifications can be made for these forms", and proposed a subdivision of rapidly progressive periodontitis into type A and type B, based on factors such as age, microbial deposits present and the autologous mixed lymphocyte reaction. Furthermore, the term post-juvenile periodontitis was intended to delineate a slow-progression-type of juvenile periodontitis. (23)

| 1. Adult periodontitis            |
| 2. Rapidly progressive periodontitis Type A |
| 3. Rapidly progressive periodontitis Type B |
| 4. Juvenile periodontitis         |
| 5. Post juvenile periodontitis    |
| 6. Prepubertal periodontitis      |

Table 5. Classification of periodontitis by Suzuki (23)

The 1989 World Workshop in Clinical Periodontics recommended a modification of the 1986 AAP classification (Table 6). The main changes were the use of the term early onset periodontitis to cover prepubertal, juvenile as well as rapidly progressive periodontitis, the existence of which was thus recognized, and the introduction
of periodontitis associated with systemic disease as a different category meant to encompass periodontitis among individuals presenting with systemic diseases that “appear to predispose the individuals who have them to periodontitis”.

1. Adult periodontitis
2. Early-onset periodontitis
   a. Prepubertal periodontitis
   b. Generalized, Localized
   c. Rapidly progressive periodontitis
3. Periodontitis associated with systemic disease
4. Necrotizing ulcerative periodontitis
5. Refractory periodontitis

Table 6. Classification of periodontitis by the AAP.(24)

Shortly after the 1989 World Workshop in Clinical Periodontics, Topić proposed a classification (Table 7) which essentially tried to amalgamate all previous classifications. (25)

1. Acute conditions
   a. Periodontitis
   i. Periodontal abscess, ii. Pericoronitis
2. Chronic conditions
   a. Periodontitis
   i. Simplex (adult)
   ii. Complex
   iii. Generalized forms
      - Rapidly progressive type A or B
      - Prepubertal
   b. Localized forms
      i. Juvenile, ii. Post-juvenile, iii. Prepubertal
   c. Symptomatic

Table 7. Classification of periodontitis by Topić.(25)

In 1993, Ranney proposed that four major groups, adult periodontitis, early onset periodontitis, necrotizing ulcerative periodontitis and periodontal abscess, be used as the headings for a rather large number of sub-categories, primarily determined by systemic factors (Table 8).

1. Adult periodontitis
   a. Non-aggravated
   b. Systemically aggravated
      - Neutropenias, Leukemias, Lazy leukocyte syndrome, AIDS, Diabetes mellitus, Crohn’s disease, Addison’s disease
2. Early onset periodontitis
   a. Localized early onset periodontitis
      - Neutrophil abnormality
   b. Generalized early onset periodontitis
      - Neutrophil abnormality, Immuno deficient
3. Early-onset periodontitis related to systemic disease
   a. Leukocyte adhesion deficiency, Neutropenias, Papillon-Lefèvre syndrome, Leukemias, Hypophosphatasia, AIDS, Trisomy 21
   b. Chédiak-Higashi syndrome
   c. Diabetes mellitus type I, Histioctysis X
   d. Ehlers-Danlos syndrome (Type VII)
4. Early-onset periodontitis, systemic determinants unknown
5. Necrotizing ulcerative periodontitis
   a. Systemic determinants unknown
   b. Related to HIV
   c. Related to nutrition
6. Periodontal abscess

Table 8. Classification of periodontitis by Ranney.(26)

This rather complex classification system had a pronounced difference with the classification system recommended in 1994, in the European Workshop on Periodontology. (27) This classification (Table 9) considered only three categories, early onset, adult and necrotizing periodontitis. The simplicity of the classification was inspired in the “extensive overlap between the different categories” in previous classifications, just as the “need for assumptions concerning previous disease progression” and “the necessity for detailed information on the quality of treatment provided previously and the patient’s response to this therapy” inherent in previous classifications were considered disadvantageous.

1. Early onset periodontitis
2. Adult periodontitis
3. Necrotizing periodontitis

Table 9. Classification of periodontitis recommended in the European Workshop on Periodontology.(27)

In recognition of the large number of classifications proposed over a very short time, the American Academy of Periodontology commissioned a series of state-of-the-art-reviews on the body of scientific knowledge regarding periodontal diseases for the purpose of developing a new and fully satisfactory classification system for these conditions. The resulting new classification system encompasses many categories and sub-categories, and Table 10 shows only the main categories as they pertain to periodontitis. (27)

1. Chronic Periodontitis
   a. Localized, b. Generalized
2. Aggressive periodontitis
   a. Localized, b. Generalized
3. Periodontitis as a manifestation of systemic diseases
   a. Associated with hematological disorders
      - Acquired neutropenia,
      - Leukemias,
      - Other
   b. Associated with genetic disorders
      - Familial and cyclic neutropenia
      - Down syndrome
      - Papillon-Lefèvre syndrome
      - Chédiak-Higashi syndrome
      - Histioctysis syndrome
      - Glycogen storage disease
      - Infantile genetic agranulocytosis
      - Cohen syndrome
      - Ehlers-Danlos syndrome (IV and VII)
      - Hypophosphatasia
      - Other
   c. Not otherwise specified (NOS)
4. Necrotizing periodontal diseases
   a. Necrotizing ulcerative gingivitis
   b. Necrotizing ulcerative periodontitis
5. Periodontitis associated with endodontic lesions
   a. Combined periodontic-endodontic lesions

Table 10. Classification proposed in the International Workshop for a Classification of Periodontal Diseases and Conditions.(27)
The category periodontitis associated to systemic diseases was retained as periodontitis as a manifestation of systemic diseases. The changes with regard to earlier classifications were founded in the recommendation that “any classification system for the various forms of periodontitis should not be based on the age of the patient at the time of presentation, but should instead be based on clinical, radiographic, historical and laboratory findings.”. This classification system is the one currently adopted by the American Academy of Periodontology.(28)

Evidently, all the classifications presented above originated in the essentialistic disease concept. It is thus characteristic that in none of them the different forms of periodontitis have been defined in terms that are testable against an observable norm, which would be consistent with a nominalistic disease concept. Hence, the distinction between localized and generalized forms of periodontitis, between early onset and adult periodontitis, between chronic and aggressive periodontitis is usually described in rather vague and imprecise terms, chiefly referring to typical examples of the categories. Thereby, the defining criteria for the various groups remain ambiguous.

The general devotion to an essentialistic disease concept as regards periodontitis may have hindered scientific progress. Van der Velden’s proposal for a classification system represents the first potentially useful system for classifying the periodontitis syndrome.(11) His proposal from the point of view of essentialism and nominalism; but contended that previous classifications suffer from the central problem that they are “susceptible to multiple interpretations”. Hence, the proposed system for classifying periodontitis is based on the assessment of four dimensions, extent, severity, age, and clinical characteristics.(Table 11)

| 1. Extent | Incidental, Localized, Semi-generalized, Generalized |
| 2. Severity | Minor, Moderate, Severe |
| 3. Age | Prepubertal, Juvenile, Post-adolescent, Adult |
| 4. Clinical characteristics | Necrotizing, Rapidly progressive, Refractory |

Table 11. Classification of periodontitis by Van der Velden(11)

Each of the dimensions has a number of sub-categories defined in terms that are testable against an observable norm. As an example, the descriptor localized may be used when 2-7 permanent teeth are affected; the term juvenile covers the age range from 13-20 years of age; and rapidly progressive periodontitis refers to “documented rapid breakdown... i.e. patients showing at affected sites a progression of ≥ 1 mm interproximal attachment/bone loss per year.”(11)

Lastly, the primary signs to look for when diagnosing persons are confined to clinical attachment loss or radiographic bone loss. Unlike all previous classification systems, the one offered by Van der Velden is characterized by being exhaustive as well as exclusive. Thus, use of this classification system avoids all the difficulties inherent in previous classifications of ill-defined and overlapping categories. Furthermore, Van der Velden’s classification system can accommodate the concept of disease as a continuum thereby avoiding “the thorny issues of case definition as well as the number and types of sites to assess”.(11)

As might be predicted, the proponents of periodontal disease classification systems that are based on essentialistic disease concepts have reacted very negatively to Van der Velden’s proposal. Even though they spot that “any attempt to group the entire constellation of a periodontal disease into an orderly and widely accepted classification system is fraught with difficulty, and inevitably considerable controversy. No matter how carefully the classification is developed, and how much thought and time are invested in the process, choices need to be made between equally unsatisfactory alternatives”, they do not vacillate to express their dislike of Van der Velden’s proposal. Hence, Van der Velden’s suggestion is classified as no more than “a return to the domination of the ‘Clinical Characteristics’ paradigm that reigned from approximately 1870 to 1920 when we knew little about the nature of periodontal diseases!”(29)

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