

Classifying periodontal diseases – a long-standing dilemma

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A long-standing dilemma

Any attempt to group the entire constellation of periodontal diseases 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. Despite this dilemma, in the past hundred years, experts have periodically assembled to develop a new classification system for periodontal diseases, or to refine an existing one (1, 2, 4, 5, 11, 19, 58, 80, 81, 86, 91, 106, 122, 139).

Dominant paradigms in the historical development of classification systems

The development and evolution of classification systems for periodontal diseases have been largely influenced by paradigms that reflect the understanding of the nature of periodontal diseases during a given historical period. Over time, thoughts that guided the classification of periodontal diseases can be placed into three dominant paradigms primarily based on the clinical features of the diseases (~1870–1920), the concepts of classical pathology (~1920–1970), and the infectious etiology of the diseases (~1970–present). Classification systems in the modern era represent a blend of all three paradigms since there is a certain amount of validity to some of the earliest thoughts about the nature of periodontal diseases (2, 4, 5). As classification systems have evolved, newer thoughts about periodontal diseases

have been superimposed on a matrix of older ideas that are still considered to be valid. Only those ideas that are believed to be clearly outmoded or incorrect have been discarded. In a sense, the newest or dominant paradigm rests on a foundation of the still valid components of the older or previous paradigms.

One of the interesting historical features of classification systems is the often intense resistance to their modification. Many people appear to believe that classification systems are rigid and fixed entities that should not be changed. In fact, classification systems should be viewed as dynamic works-in-progress that need to be periodically modified based on current thinking and new knowledge. Unfortunately, it seems that once people learn and accept a given classification, no matter how flawed it may be, they are extremely reluctant to accept revisions to their favorite system of nomenclature. One group of experts on the 1949 Nomenclature Committee of the American Academy of Periodontology (AAP) expressed their frustration with this subject by stating, 'The 1949 Nomenclature Committee is somewhat pessimistic regarding the possibility of this or any similar report receiving immediate and enthusiastic acceptance. Most periodontists have used their own terms for so long that any suggested change is resisted and resented.' (81)

Clinical characteristics paradigm

For the period from approximately 1870 to 1920 very little was known about the etiology and pathogenesis of periodontal diseases. Accordingly, the diseases were classified almost entirely on the basis of their clinical characteristics supplemented by unsubstantiated theories about their cause. At the time, one of the main debates about the nature of periodontal

diseases was whether they were caused by local or systemic factors. Most authors considered these diseases to be primarily caused by local factors (16, 53, 93, 111, 112, 125, 127, 136, 149), whereas some believed that systemic disturbances played a dominant etiological role (32, 97, 114, 115). Many of the advocates for the etiological role of local factors also acknowledged that in some cases both local and systemic factors were important (93, 112, 113, 136). In the late 1800s and early 1900s clinicians used case descriptions and their personal interpretation of what they saw clinically as the primary basis for classifying periodontal diseases (15, 17, 28, 53, 111–113, 125, 127, 136, 137, 149). They expressed their opinions, often with great fervor and conviction, in oral presentations before local and national meetings of dental or medical societies. Their opinions survive in the literature in the form of written abstracts or summaries of the proceedings of these meetings. In many cases the summaries were written not by the presenter of the paper, but by the editor of the proceedings (125, 127). Indeed, John M. Riggs (1811–1875), an American dentist who lectured so widely on the treatment of periodontal diseases that periodontitis was called ‘Riggs’ disease’ by many of his colleagues, rarely published any papers on the subject (89). Riggs’ thoughts and opinions were most often summarized by others (94–96, 127).

Formal papers on the classification of periodontal diseases were rare in the late 1800s and early 1900s. Typical publications on the subject usually represented the opinion of a single person who almost always based the classification on clinical observations and theoretical explanations of causation. A good example is a paper published by C.G. Davis in 1879 (28) who believed that there were three distinct forms of destructive periodontal disease:

- *Gingival recession with minimal or no inflammation.* This was due to ‘... feeble vascular action ...’ and trauma from tooth brushing or other sources.
- *Periodontal destruction secondary to ‘lime deposits.’* ‘The gum retires slowly ... and the alveolar border, deprived of nutrition at the point of pressure, is consentaneously absorbed.’ Davis apparently believed that calculus exerted mechanical pressure on the gingiva causing the alveolar bone to resorb because of lack of nutrition.
- *‘Riggs’ Disease’ the hallmark of which was, ‘... loss of alveolus without loss of gum.’* The perceived problem was a ‘necrosed alveolus’ or death of the periodontal membrane. ‘... we get a disease that

is initiated and continued without any visible mechanical irritant in many cases; and I believe the death of the peridental membrane, depriving the alveolus of nutrition, accounts for the death and disintegration of the bone; or, as is believed by some, among them Dr Waters, of Boston, the alveolus is destroyed by vegetable parasites’.

Similarly, in 1886 G.V. Black (15) published his thoughts on the classification of periodontal diseases based on their clinical characteristics and his understanding of their cause into five separate groups.

- *constitutional gingivitis*; including mercurial gingivitis, potassium iodide gingivitis and scurvy.
- *a painful form of gingivitis.* Black described a clinical condition that resembled what is now termed necrotizing ulcerative gingivitis (NUG), but he never used the term.
- *simple gingivitis.* This was associated with the accumulation of debris that eventually led to ‘calcic inflammation of the peridental membrane.’
- *calcic inflammation of the peridental membrane.* This was associated with ‘salivary’ and/or ‘serumal’ calculus. Usually there was an even or generalized pattern of destruction of alveolar bone. The destruction usually occurred slowly. Black’s description best fits the periodontal disease that is now known as chronic periodontitis.
- *phagedenic pericementitis* (phagedenic = spreading ulcer or necrosis). This condition shared many features with ‘calcic inflammation of the peridental membrane’ but there was an irregular pattern of destruction and not much dental calculus. Destruction of the alveolar bone can occur slowly or rapidly. In a later publication Black replaced the term ‘phagedenic pericementitis’ with ‘chronic suppurative pericementitis’ (17).

The point of these historical examples is to emphasize that little or no scientific evidence was used to support the opinions of the clinicians of the time. As one might expect, the number of theories about what caused periodontal diseases, how they should be classified, and the terminology used to describe them, seem to have approached the number of clinicians who treated patients with these diseases. By 1929 one author estimated that there were ‘... over 350 theories of pyorrhea’ and much confusing terminology (10). It is not surprising then, that no generally accepted terminology or classification system for periodontal diseases was adopted during this era. As a result, in the latter part of the 19th century periodontitis went

under numerous names including: 'pyorrhea alveolaris' (19, 53, 93, 111, 112, 125, 136, 137, 149), 'Riggs' disease' (28, 94–96), 'calic inflammation of the peridental membrane' (15), 'phagedenic pericementitis' (15, 19), and 'chronic suppurative pericementitis' (17). During this period, the dominant term used for destructive periodontal disease was pyorrhea alveolaris.

Classical pathology paradigm (~1920–1970)

As the field of periodontology began to mature scientifically in the first half of the 20th century, many clinical scholars in both Europe and North America began to develop, and argue about, nomenclature and classification systems for periodontal diseases (34, 38, 43, 45, 46, 55, 58, 86, 91, 103, 128, 129, 139). What emerged from this debate was the concept that there were at least two forms of destructive periodontal disease inflammatory and noninflammatory ('degenerative' or 'dystrophic'). It had, of course, been known for a very long time that many periodontal diseases were inflammatory conditions. However, the conclusion that some periodontal diseases were caused by noninflammatory or degenerative processes was a somewhat novel suggestion. This conclusion was primarily based on the over-interpretation of histopathological studies from a group of Viennese investigators led by Gottlieb and Orban. Gottlieb, in particular, had a significant influence on the field when he postulated that certain forms of destructive periodontal disease were due to degenerative changes in the periodontium (42–47). He believed that he had discovered histological evidence of an impairment in the continuous deposition of cementum (i.e. 'cementopathia'). This cemental defect was presumably initiated by the degeneration of the principal fibers of the periodontal ligament that eventually resulted in detachment of connective tissue from the tooth followed by resorption of adjacent bone (45–47). Other authors postulated that in some periodontal diseases there was a degenerative transformation of alveolar bone into fibrous connective tissue (139).

Gottlieb's ideas were probably widely accepted because they appeared to explain the long-standing and perplexing clinical observation that some young patients with relatively clean mouths had massive and localized bone loss with only minimal or no overt signs of gingival inflammation (92, 102, 140, 146). The profession was ready to embrace a plausible etiological explanation for what would eventu-

ally be called localized aggressive periodontitis (4). The impact of Gottlieb's work on classification systems was profound since it suggested that some periodontal diseases were degenerative. As a result, almost all classification systems used from approximately 1920–1970 included disease categories labeled as 'dystrophic', 'atrophic', or 'degenerative' (Fig. 1). Classification systems of the period were dominated by the 'Classical Pathology' paradigm which is based on the 'principles of general pathology' as articulated by Orban et al. (104):

'Periodontal diseases follow the same pattern as do diseases of other organs. There are minor differences which have to be recognized and labeled properly. The basic pathologic tissue changes, however, are the same as those of other organs.' ... According to principles of general pathology, there are three major tissue reactions: inflammatory; dystrophic; neoplastic. Neoplastic changes are not in the therapeutic realm of periodontics.

'Environmental factors, however, dictate the inclusion of a third and different category of pathologic reaction in Periodontology ...' ... pathologic reactions ... produced by occlusal trauma.

Although most classification systems published from approximately 1920 to 1970 included a degenerative disease category (24, 34, 37, 39, 40, 48, 58, 80, 128, 129, 139, 144), at the 1966 World Workshop in Periodontics serious questions were raised about the existence of 'periodontosis' as a distinct disease entity (1). Many in attendance at that meeting recommended that the term be discarded. It was not until the next World Workshop, held in 1977, that convincing arguments were provided that there was no scientific basis for retaining the concept that there were noninflammatory or degenerative forms of destructive periodontal disease (122). Information summarized at that meeting supported the conclusion that 'periodontosis' was actually an infection and 'juvenile periodontitis' should become the preferred term for this group of diseases. Indeed, around 1970 a different paradigm (i.e. the 'Infection/Host Response Paradigm') had begun to dominate thoughts about the nature of periodontal diseases.

In retrospect it is puzzling that Gottlieb's concept of cementopathia was so readily accepted, and for such a long time. Although there has been an occasional report that cemental abnormalities might be associated with some forms of periodontal disease (73, 109), there was never any convincing evidence that Gottlieb's hypothesis was right. It is worth noting, however, that there is a rare condition (i.e. hypophosphatasia) where hypoplasia or absence of cementum is associated with the early loss of de-

Inflammation

- I. Gingivitis (little or no pocket formation; can include ulcerative form – Vincent’s)
 - A. Local (calculus, food impaction, irritating restorations, drug action, etc.)
 - B. Systemic
 - pregnancy
 - diabetes
 - other endocrine dysfunctions
 - tuberculosis
 - syphilis
 - nutritional disturbances
 - drug action
 - allergy
 - hereditary
 - idiopathic, etc.
- II. Periodontitis
 - A. Simplex (secondary to gingivitis) – bone loss, pockets, abscesses can form: cases have calculus.
 - B. Complex (secondary to periodontosis) – etiologic factors similar to periodontitis; cases have little, if any calculus.

Degeneration

- I. Periodontosis (as a rule attacks young girls and older men; often caries immunity)
 - A. Systemic disturbances
 - 1. diabetes
 - 2. endocrine dysfunctions
 - 3. blood dyscrasias
 - 4. nutritional disturbances
 - 5. nervous disorders
 - 6. infectious diseases (acute & chronic)
 - B. Hereditary
 - C. Idiopathic

Atrophy

- I. Periodontal Atrophy (Recession, no inflammation, no pockets; osteoporosis)
 - A. Local trauma (e.g., from toothbrush)
 - B. Presenile
 - C. Senile
 - D. Disuse
 - E. Following inflammation
 - F. Idiopathic

Hypertrophy

- I. Gingival Hypertrophy
 - A. Chronic irritation (see inflammation)
 - B. Drug action (e.g., Dilantin sodium)
 - C. Idiopathic (e.g., gingivoma, elephantiasis, fibromatosis)

Traumatism

- I. Periodontal Traumatism
 - A. Occlusal trauma

*Orban (103) based this classification on a combination of his perceptions of the etiologic, clinical, and pathologic features of the diseases. He grouped them according to the “pathologic” categories of Inflammation, Degeneration, Atrophy, Hypertrophy, and Traumatism. Similar classifications were published by other authors (Coolidge & Hine 1951 (24), Fish 1944 (34), Goldman et al. 1956 (39), Goldman & Cohen 1968 (40), Grant et al. 1968 (48), Hine & Hine 1944 (58), Lyons 1946, (80), Wade 1960 (144)).

Fig. 1. Classification of Periodontal Diseases Following the “Classical Pathology” Paradigm (Orban 1942)* [103]

ciduous teeth (7, 12, 18, 78). Hypophosphatasia is a hereditary disease characterized by low serum levels of tissue-nonspecific alkaline phosphatase, elevated levels of urinary phosphoethanolamine, skeletal abnormalities resembling rickets, and premature loss of anterior deciduous teeth (12, 18, 20, 21, 26, 116, 117, 131). In mild forms of the disease the patient's only complaint may be an unexplained premature loosening of anterior primary teeth. Extensive bone loss can be observed around the affected teeth with no evidence of root resorption (7, 12). On rare occasions, posterior deciduous teeth may be affected; permanent teeth do not usually become involved (70, 116). However, there are a few case reports suggesting that cementum may be absent or thin on the permanent incisors of patients with hypophosphatasia (33, 82, 101). There is also a case report in which the permanent incisors had severe periodontitis (147, 148). However, the patient in this report harbored *Porphyromonas gingivalis* in his subgingival flora, suggesting that something other than hypoplasia of cementum might have contributed to the periodontal destruction.

Infection/host response paradigm (~1970 to present)

Soon after the 1876 publication of Robert Koch (64) in which he provided experimental proof of the germ theory of disease, some dentists began to suggest that periodontal diseases might be caused by bacteria (53, 93, 136). W.D. Miller (93), in particular, was an early proponent of the infectious nature of periodontal diseases:

'In my opinion three factors are to be taken into consideration in every case of pyorrhea alveolaris: (1) predisposing circumstances, (2) local irritation, (3) bacteria.'

'... pyorrhea alveolaris is not caused by any *specific* bacterium, which occurs in every case ..., but various bacteria may participate in it ...'

Miller also recognized that certain systemic conditions (e.g. diabetes, pregnancy) could modify the course of the disease. Although he spent most of his life studying the oral microflora associated with caries and periodontal disease, his work had very little impact on convincing his contemporaries that periodontal diseases were infections (77). He was, however, an early advocate of the 'Infection/Host Response Paradigm' that would come to dominate the field nearly a hundred years later.

Despite an extensive amount of work on the microbiology of periodontal diseases from approxi-

mately 1880 to 1965 very little headway was made in establishing bacterial infections as the foundation upon which periodontal diseases should be classified (133). Part of the reluctance of the profession to accept the idea that most periodontal diseases were infections was an unfortunate preoccupation with the notion that some forms of destructive periodontal diseases were degenerative in nature (i.e. domination of the 'Classical Pathology' paradigm). In addition, microbiological studies revealed that the periodontal microflora was exceedingly complex and no clear group of microorganisms could be causally linked to the diseases. It was not until the classical 'experimental gingivitis' studies published by Harald Löe and his colleagues from 1965 to 1968 that the Infection/Host Response Paradigm began to move in the direction of becoming the dominant paradigm (62, 75, 76, 138). These studies were significant because they provided convincing data that relatively specific changes occurred in the dental plaque flora during the development of gingivitis. The next major discovery in periodontal microbiology was the preliminary demonstration in 1976–1977 of microbial specificity at sites with periodontosis (99, 100). This finding, coupled with the demonstration in 1977–1979 that neutrophils from patients with juvenile periodontitis (periodontosis) had defective chemotactic and phagocytic activities (23, 68), marked the beginning of the dominance of the Infection/Host Response paradigm. Indeed, these seminal findings challenged the validity of the 50-year assumption that degenerative forms of destructive periodontal disease existed. What followed was over two decades of hard work that firmly established that juvenile periodontitis, the new name for periodontosis, was an infection.

The next major landmark in the classification of periodontal diseases emerged from the 1989 World Workshop in Clinical Periodontics where a new classification of periodontitis based on the Infection/Host Response paradigm was suggested (2) (Fig. 2). The classification was a refinement of one that had been proposed by Page & Schroeder in 1982 (106) and a similar one that had been adopted by the AAP in 1986 (2). Five types of destructive periodontal disease were listed: I, Adult Periodontitis; II, Early Onset Periodontitis; III, Periodontitis Associated with Systemic Disease; IV, Necrotizing Ulcerative Periodontitis; and V, Refractory Periodontitis (Fig. 2). This classification, although soundly based in the Infection/Host Response paradigm, depended heavily on the *age* of the affected patients (6, 107, 108) and the *rates of progression* (107). Other important fea-

tures included the acknowledgment that some forms of periodontitis could be significantly modified by host factors (i.e. the category of 'Periodontitis Associated with Systemic Disease') and still other forms did not appear to respond well to conventional therapy (i.e. the 'Refractory Periodontitis' category). At the time the classification was proposed it was recognized that, 'Overlap exists among categories and cases exist that do not clearly fit into any single category' (2). In addition, it was acknowledged that considerable 'heterogeneity' existed within the Refractory Periodontitis category since, '... it includes patients who are unresponsive to any treatment provided - whatever the thoroughness or frequency - as well as patients with recurrent disease at few or many sites. Assignment of refractory cases to other categories may be expected to occur as more information is acquired.' (2) Finally, different forms of periodontitis proposed in the classification shared many microbiologic and host response features, which suggested extensive overlap and heterogeneity among the categories (3).

As a consequence of these problems, the 1989 classification was criticized shortly after it was published and a different system was proposed by Ranney (123, 124). He suggested elimination of the 'Re-

- I. Adult Periodontitis
- II. Early Onset Periodontitis
 - A. Prepubertal Periodontitis
 - 1. Generalized
 - 2. Localized
 - B. Juvenile Periodontitis
 - 1. Generalized
 - 2. Localized
 - C. Rapidly Progressive Periodontitis
- III. Periodontitis Associated with Systemic Disease
- IV. Necrotizing Ulcerative Periodontitis
- V. Refractory Periodontitis

Fig. 2. Classification of Various Forms of Periodontitis Based on the "Infection/Host Response" Paradigm (World Workshop in Clinical Periodontics - 1989) [2]

fractory Periodontitis' category since it was a heterogeneous group and it was impossible to standardize the treatment that necessarily would have to be given prior to making the diagnosis. In addition, he recommended elimination of the 'Periodontitis Associated with Systemic Disease' category since the, '... expression of all forms of periodontitis can be modified by some systemic diseases or abnormalities, it is probably better to consider them in that specific context, rather than treating them as a unique category.' (124) Nevertheless, despite its problems, the classification was adopted by the world community as reflected by its widespread use in the periodontal literature. Its acceptance was facilitated by the ease with which patients could be placed into age-based categories (i.e. adult vs. early onset disease). For example, it was logical to assume that children, adolescents and young adults with extensive periodontal destruction had a different group of diseases (i.e. Early Onset Periodontitis) compared to adults (defined as people ≥ 35 years of age) who had a similar amount of periodontal destruction. This particularly seemed to make sense in the three early onset subcategories of prepubertal, juvenile and rapidly progressive periodontitis. However, it soon became apparent that there were problems with some of the assumptions that had been made.

The disease category of 'Prepubertal Periodontitis' was the first to be seriously questioned. In retrospect, many of the patients in the original publication on this disease category (108) turned out to have either hypophosphatasia (6, 109) or leukocyte adherence deficiency (LAD) (6). Indeed, it is likely that most prepubertal children with severe periodontal destruction affecting the deciduous teeth probably have a systemic disease that increases their susceptibility to bacterial infections such as: LAD (90, 145), congenital primary immunodeficiency (8), chronic neutrophil defects (31, 63) and cyclic neutropenia (118). Such patients should probably have been properly placed under the general category of 'Periodontitis Associated with Systemic Disease'.

Among the other problems with the 1989 classification were firstly, the uncertainty about the proposal that 'Rapidly Progressive Periodontitis' was a single entity, and secondly, the questionable criteria used to determine its presence. For example, do clinicians have to actually document that rapid progression has occurred prior to giving a patient this diagnosis? To be designated as 'rapid', how much progression has to occur and over what time period? Can it be assumed from a single examination that

an adolescent or young adult with massive attachment loss has this disease? How can a clinician distinguish between 'Generalized Juvenile Periodontitis' and 'Rapidly Progressive Periodontitis'? Since there are no definitive answers to these, and other similar questions, the classification lost some of its clinical utility.

The concept that the rate of progression might be a useful criterion upon which to base a disease category may in itself be flawed. The rate at which periodontitis progresses is highly variable and depends on such factors as

- innate and acquired host susceptibility (30, 36, 60).
- composition and quantity of the subgingival flora (27).
- the nature of genetically determined host-bacterial interactions (54, 66).

Almost any form of periodontitis can progress rapidly or slowly depending on the set of circumstances governing the nature of the host-bacterial interactions during a given time period. Indeed, longitudinal studies of patients with untreated Chronic ('Adult') Periodontitis, in which disease progression is usually considered to be 'slow', can undergo bursts of progression during which extensive amounts of attachment loss can occur at localized sites within a short period of time (e.g. 2–3mm within 3 months) (41, 49, 50, 61, 132). Did such sites suddenly develop a different form of periodontal disease (i.e. shift from Chronic Periodontitis to Rapidly Progressive Periodontitis)? This explanation is possible, but unlikely.

The existence of a group of periodontal diseases that would eventually be termed 'Refractory Periodontitis' came from a series of studies of private practice patients who unexpectedly did not respond to treatment (9, 59, 79, 87, 88, 98). The reasons for the unresponsiveness to conventional therapy are not clear, but it is probably due to the emergence of resistant or super-infecting microorganisms, tissue invasion by periodontal pathogens, and innate or acquired alterations or defects in host responses (65, 85).

Whatever the reasons, it has been demonstrated that some patients with periodontitis 'refractory' to treatment harbor enteric rods, staphylococci and *Candida* at unresponsive sites (25, 56, 74, 119–121). Whereas other patients who responded poorly to treatment, or who developed recurrent disease, continued to harbor in the subgingival flora at nonresponding sites elevated levels of *Porphyromonas gin-*

givalis (22), *Prevotella intermedia* (69), *Eikenella corrodens* (69), *Streptococcus intermedius* (84), or microbial complexes consisting of various combinations of *P.gingivalis*, *S.intermedius*, *Treponema denticola*, *Campylobacter rectus*, *Bacteroides forsythus*, *Peptostreptococcus micros* and *Fusobacterium nucleatum* (51, 52). In addition, perturbations in host responses, such as altered neutrophil chemotaxis (83, 105), over-production of certain proinflammatory cytokines (57, 69, 126), and elevated serum (51, 84) or gingival crevicular fluid (GCF) antibody (21) against putative periodontal pathogens, have been reported. The striking feature of the microbiological and host response results in refractory patients is their extensive variability and heterogeneity. The work that has been done on Refractory Periodontitis does not challenge its existence, since there are some people who are clearly unresponsive to conventional treatment. However, studies of such patients appear to indicate that Refractory Periodontitis is not a single entity. As mentioned in the proceedings of the 1989 World Workshop in Clinical Periodontics, it is a heterogeneous grouping (2). In addition, except in the most unusual of circumstances it is very difficult to distinguish between refractory and recurrent periodontal disease (124).

The final major problem with the 1989 classification was its arbitrary and heavy reliance on *age* of the affected patients or *age of onset* of the disease. Indeed, the Adult Periodontitis and Early Onset Periodontitis categories were firmly based on age as a criterion for placing patients into one category or another. In this classification the dividing line between adult and early onset categories was arbitrarily set at 35 years of age (2). Certainly clinical features of a patient's periodontitis were important (e.g. the incisor/first molar involvement in 'Localized Juvenile Periodontitis'), but decisions regarding the final diagnosis depended greatly on the age of the patient. One of the advantages of using the age criterion is that it formally acknowledges the existence of different types of periodontitis in children and adolescents.

There is no question that the patient's age is an important variable in evaluating the nature of an individual's periodontal disease. For example, a 15-year-old patient with multiple sites with 3mm of clinical attachment loss (CAL) has a different kind of periodontal problem compared to a 90-year-old with the same amount of damage. However, when age is used as the single most important determinant in classifying various forms of periodontitis, difficult questions arise. Is it necessary to establish the age of onset of periodontitis before a patient can be cor-

rectly classified or diagnosed? As a child or adolescent with periodontitis gets older, should the periodontal diagnosis change (i.e. with time does Prepubertal Periodontitis become Juvenile Periodontitis which then becomes Rapidly Progressive Periodontitis)? Some might argue that the answer to this question should be 'yes' since it has been reported that two or three of these forms of periodontitis have been observed within highly susceptible families (134, 142). Authors of one of these reports suggested that all three forms of Early Onset Periodontitis might have a common underlying mechanism (134). It can, however, be argued that it is incorrect or simply wrong to use age as the main criterion for assignment of different names to the periodontitis affecting various family members. Indeed, it is just as likely that the subcategories of Early Onset Periodontitis are the *same* disease rather than three separate forms of periodontitis.

Of all the reasons for questioning the use of age as a criterion for classification, the most compelling are data from epidemiological studies indicating that children and adolescents develop attachment loss from a type of periodontitis that clinically resembles that seen in adults (110). In other words, certain children and adolescents develop what is, for all intents and purposes, identical to 'Adult Periodontitis', except that those affected are not adults. Some authors have avoided this obvious nomenclature problem by using the term 'Childhood Periodontitis' (29).

1999 Classification of Periodontal Diseases and Conditions

Problems, inconsistencies, and deficiencies associated with the 1989 classification led many clinicians and investigators to call for a revision of the currently used system. This resulted in a 1999 international workshop on the classification of periodontal diseases (4). One of the goals of this workshop was to correct the problems associated with the 1989 system. There were six major problems with the 1989 classification that needed to be addressed:

- it did not include a gingivitis or gingival disease category.
- the periodontitis categories had nonvalidated age-dependent criteria.
- there was extensive crossover in rates of progression of the different categories of peri-

odontitis. 'Rapidly Progressive Periodontitis' was a heterogeneous category.

- there was extensive overlap in the clinical characteristics of the different categories of periodontitis.
- 'Refractory Periodontitis' was a heterogeneous category.
- 'Prepubertal Periodontitis' was a heterogeneous category.

What emerged was a classification that was even more firmly based on the Infection/Host Response paradigm, but without some of the inherent problems of the 1989 classification (Fig. 3) (4). In reality, the changes could be characterized as a 'course correction' or 'fine-tuning' of the 1989 classification since no massive alterations were made. A badly needed gingivitis or gingival disease category was added. In addition, the heterogeneous disease categories of prepubertal, refractory and rapidly progressive periodontitis were eliminated as distinct or stand-alone entities. The 'refractory' designation remains in the new classification, but not as a single entity. Conceptually, all forms of periodontitis can be unresponsive to treatment. Furthermore, the troublesome criteria of age and rate of progression were removed as a basis for classifying different forms of periodontitis. The reasons for these changes were not arbitrary, but were based on available data and the current understanding of the nature of periodontal infections (4, 35, 67, 71, 72).

As might have been predicted, some clinicians and investigators think that the new classification is nonsense and will not, '... help much in the discussion as to how the various forms of periodontitis should be classified.' (141). Remarkably, one critic has fervently recommended that the classification of periodontitis be based on, '... a combination of a number of clinical symptoms of the disease and the age of the patient.' (141). Indeed, it was suggested that the classification be based on extent and severity of the disease, age, and rate of progression (141). Clearly this would be 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!

A quick comparison of the 1989 and the 1999 classifications could lead to the misconception that all that was done was to arbitrarily change the names of 'Adult Periodontitis' to 'Chronic Periodontitis' and 'Juvenile Periodontitis' to 'Aggressive Periodontitis.' These changes were specifically made to eliminate the nonvalidated age-dependent designations. They were, however, not the most important

Classification of Periodontal Diseases and Conditions Based on the “Infection/Host Response Paradigm” (1999 International Workshop for a Classification of Periodontal Diseases and Conditions) [3]

I. Gingival Diseases

A. Dental Plaque-Induced Gingival Diseases*

1. Gingivitis associated with dental plaque only
 - a. without other local contributing factors
 - b. with local contributing factors (See VIII. A.)
2. Gingival diseases modified by systemic factors
 - a. associated with the endocrine system
 - 1) puberty-associated gingivitis
 - 2) menstrual cycle-associated gingivitis
 - 3) pregnancy-associated
 - a) gingivitis
 - b) pyogenic granuloma
 - 4) diabetes mellitus-associated gingivitis
 - b. associated with blood dyscrasias
 - 1) leukemia-associated gingivitis
 - 2) other
3. Gingival diseases modified by medications
 - a. drug-influenced gingival diseases
 - 1) drug-influenced gingival enlargements
 - 2) drug-influenced gingivitis
 - a) oral contraceptive-associated gingivitis
 - b) other
4. Gingival diseases modified by malnutrition
 - a. ascorbic acid-deficiency gingivitis
 - b. other

B. Non-Plaque Induced Gingival Lesions

1. Gingival diseases of specific bacterial origin
 - a. *Neisseria gonorrhoea*-associated lesions
 - b. *Treponema pallidum*-associated lesions
 - c. streptococcal species-associated lesions
 - d. other
2. Gingival diseases of viral origin
 - a. herpesvirus infections
 - 1) primary herpetic gingivostomatitis
 - 2) recurrent oral herpes
 - 3) varicella-zoster infections
 - b. other
3. Gingival diseases of fungal origin
 - a. *Candida*-species infections
 - 1) generalized gingival candidiasis
 - b. linear gingival erythema
 - c. histoplasmosis
 - d. other
4. Gingival lesions of genetic origin
 - a. hereditary gingival fibromatosis
 - b. other

*Can occur on a periodontium with no attachment loss or on a periodontium with attachment loss that is not progressing.

5. Gingival manifestations of systemic conditions

- a. mucocutaneous disorders
 - 1) lichen planus
 - 2) pemphigoid
 - 3) pemphigus vulgaris
 - 4) erythema multiforme
 - 5) lupus erythematosus
 - 6) drug-induced
 - 7) other
- b. allergic reactions
 - 1) dental restorative materials
 - a) mercury
 - b) nickel
 - c) acrylic
 - d) other
 - 2) reactions attributable to
 - a) toothpastes/dentifrices
 - b) mouthrinses/mouthwashes
 - c) chewing gum additives
 - d) foods and additives
 - 3) other
6. Traumatic lesions (factitious, iatrogenic, accidental)
 - a. chemical injury
 - b. physical injury
 - c. thermal injury
7. Foreign body reactions
8. Not otherwise specified

II. Chronic Periodontitis[†]

- A. Localized
- B. Generalized

III. Aggressive Periodontitis[†]

- A. Localized
- B. Generalized

IV. Periodontitis as a Manifestation of Systemic Diseases

- A. Associated with hematological disorders
 1. Acquired neutropenia
 2. Leukemias
 3. Other
- B. Associated with genetic disorders
 1. Familial and cyclic neutropenia
 2. Down syndrome
 3. Leukocyte adhesion deficiency syndromes
 4. Papillon-Lefèvre syndrome
 5. Chediak-Higashi syndrome
 6. Histiocytosis syndromes
 7. Glycogen storage disease
 8. Infantile genetic agranulocytosis
 9. Cohen syndrome
 10. Ehlers-Danlos syndrome (Types IV and VIII AD)
 11. Hypophosphatasia
 12. Other
- C. Not otherwise specified (NOS)

V. Necrotizing Periodontal Diseases

- A. Necrotizing ulcerative gingivitis (NUG)
- B. Necrotizing ulcerative periodontitis (NUP)

VI. Abscesses of the Periodontium

- A. Gingival abscess
- B. Periodontal abscess
- C. Pericoronal abscess

VII. Periodontitis Associated With Endodontic Lesions

- A. Combined periodontal-endodontic lesions

VIII. Developmental or Acquired Deformities and Conditions

- A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis
 1. tooth anatomic factors
 2. dental restorations/appliances
 3. root fractures
 4. cervical root resorption and cemental tears
- B. Mucogingival deformities and conditions around teeth
 1. gingival/soft tissue recession
 - a. facial or lingual surfaces
 - b. interproximal (papillary)
 2. lack of keratinized gingiva
 3. decreased vestibular depth
 4. aberrant frenum/muscle position
 5. gingival excess
 - a. pseudopocket
 - b. inconsistent gingival margin
 - c. excessive gingival display
 - d. gingival enlargement (See I.A.3. and I.B.4.)
 6. abnormal color
- C. Mucogingival deformities and conditions on edentulous ridges
 1. vertical and/or horizontal ridge deficiency
 2. lack of gingiva/keratinized tissue
 3. gingival/soft tissue enlargement
 4. aberrant frenum/muscle position
 5. decreased vestibular depth
 6. abnormal color
- D. Occlusal trauma
 1. primary occlusal trauma
 2. secondary occlusal trauma

From: Armitage GC. Ann Periodontol 1999; 4:1-6 (Reprinted with permission, American Academy of Periodontology).

[†] Can be further classified on the basis of extent and severity. As a general guide, extent can be characterized as Localized = ≤30% of sites involved and Generalized = >30% of sites involved. Severity can be characterized on the basis of the amount of clinical attachment loss (CAL) as follows: Slight = 1-2 mm CAL, Moderate = 3-4 mm CAL, and Severe = ≥5 mm CAL.

Fig. 3. Classification of Periodontal Diseases and Conditions Based on the “Infection/Host Response Paradigm” (1999 International Workshop for a Classification of Periodontal Diseases and Conditions) [3]

changes. Elimination of the categories of 'Refractory Periodontitis' and 'Rapidly Progressive Periodontitis' was badly needed because of their extraordinary heterogeneity. In addition, elimination of the 'Prepubertal Periodontitis' category was important since existing data do not support the notion that it is a single entity. Some cases of severe periodontitis in children are attributable to the presence of a systemic disease, whereas many cases occur without any modifying systemic conditions (13, 14, 130, 135). Indeed, the data suggest that 'chronic' periodontitis has its beginnings in childhood.

To some clinicians, selection of the term 'chronic' as a replacement for 'adult' to describe the most common form of periodontitis may seem inappropriate since it might be interpreted to mean that the disease is permanent or incurable. These clinicians might argue that since most patients with chronic periodontitis respond favorably to therapy, they should be considered as 'cured.' However, there are no data to support the contention that most patients who have been treated for periodontitis are 'cured' in the sense that the disease and its underlying causes are gone. Although treatment may result in dramatic clinical improvements and reduce the subgingival levels of periodontal pathogens to nondetectable levels (based on cultural data), there are no convincing data to show that the pathogens have been permanently eliminated. Indeed, periodontal infections tend to recur if a rigorous post-treatment maintenance program is not followed.

Future challenges in the classification of periodontal diseases

As we enter the postgenomic era with our increased understanding of the bacteria associated with periodontal infections and the genetic factors controlling host responses to these infections, it would seem that a more mechanistic or etiological classification could be devised. Why could modern classifications of periodontal diseases not be based on the microbiological features of these infections, or on the genetic factors that seem to control the clinical expression of these diseases? The answer is simple. We do not know enough about periodontal infections to take this next step.

It is very likely that 'Chronic Periodontitis' is a constellation of diseases (i.e. it is not a single entity). One of the main problems associated with any

attempt at subclassifying this or other forms of periodontitis, is that these infections are polymicrobial and polygenic. In addition, the clinical expression of these diseases is altered by important environmental and host-modifying conditions (e.g. oral hygiene, smoking, emotional stress, diabetes). It is conceivable that with much more information and the application of sophisticated multivariate analyses, it may eventually be possible to subclassify the multiple forms of 'Chronic Periodontitis' into discrete microorganism/host genetic polymorphism groups such as:

- group A – Set #1 of microorganisms + Set #1 of genetic polymorphisms.
- group B – Set #2 of microorganisms + Set #2 of genetic polymorphisms.
- group C – Set #3 of microorganisms + Set #3 of genetic polymorphisms.
- group D – Set #4 of microorganisms + Set #4 of genetic polymorphisms.

In addition, it will be necessary to superimpose on these 'microorganism/host genetic polymorphism' groupings the effect of environmental or host-modifying factors. It will be necessary to address head on the nagging question, 'When are host-modifying factors (e.g. smoking, diabetes) so important that they should be a principal part of the disease classification?' That is, in an evidence-based classification should there be a 'smoking-induced periodontitis' or a 'diabetic periodontitis?' When do modifying factors become an essential classification characteristic of the disease? The same problems also occur when one addresses the so-called 'Localized Aggressive Periodontitis' and 'Generalized Aggressive' categories. It is likely that these diseases also have multiple forms and are at least as complex as the 'Chronic Periodontitis' group.

There is a tendency for clinicians and investigators to 'jump the gun' and to use etiology- or pathogenesis-based classifications or terms prematurely. For example, it is exceedingly difficult to prove in a given subset of patients that the presence of a known periodontal pathogen in the subgingival flora is actually the cause of the periodontal disease in that group of individuals. Indeed, it has been amply shown that known periodontal pathogens, such as *Actinobacillus actinomycetemcomitans*, can be found in the supra- and subgingival flora of patients without periodontitis (3). Nevertheless, some authors have referred to the existence of an *A. actinomycetemcomitans*-associated periodontitis (143). Al-

though tempting, terms based on assumed etiological or pathogenic associations should be discouraged until there is a body of data to support their use.

Summary and conclusions

In the past 130 years classification systems for periodontal diseases have evolved based on the understanding of the nature of these diseases at the time the classifications were proposed. One consistent feature of the development of classification systems is the guaranteed controversy surrounding any suggested revisions to the previously accepted system of nomenclature. Revisions to existing systems have been largely influenced by three dominant paradigms that reflect thinking at the time the classifications were proposed: the Clinical Characteristics paradigm (~1870–1920), the Classical Pathology paradigm (~1920–70), and the Infection/Host Response paradigm (~1970–present). Although classification systems for periodontal diseases currently in use are firmly based on, and dominated by, the Infection/Host Response paradigm, some features of the older paradigms are still valid and have been retained.

The classification system proposed by the '1999 International Workshop for a Classification of Periodontal Diseases and Conditions' (4) has corrected some of the problems associated with the previous system that had been in use since 1989 (2). Nevertheless, the new system is far from perfect and will need to be modified once there are sufficient new data to justify revisions. Since it is probable that essentially all dentists and periodontists in the world are convinced that most periodontal diseases are infections, it is unlikely that the Infection/Host Response paradigm will be replaced in the near future. It is highly likely that current disease designations, such as 'Chronic Periodontitis', are constellations of polymicrobial and polygenic infections whose clinical expression is profoundly altered by important environmental and host-modifying conditions. Before a classification firmly based on the etiological and pathogenic characteristics of periodontal infections can be devised, numerous fundamental breakthroughs will have to occur in our understanding of host-microbial interactions and the environmental factors that affect them. Until this happens, all classification systems will continue to create a dilemma in that choices will need to be made between equally unsatisfactory alternatives.

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