Development of a Classification System for Periodontal Diseases and Conditions

Gary C. Armitage*

* University of California, San Francisco, California.

Classification systems are necessary in order to provide a framework in which to scientifically study the etiology, pathogenesis, and treatment of diseases in an orderly fashion. In addition, such systems give clinicians a way to organize the health care needs of their patients. The last time scientists and clinicians in the field of periodontology and related areas agreed upon a classification system for periodontal diseases was in 1989 at the World Workshop in Clinical Periodontics.[1] Subsequently, a simpler classification was agreed upon at the 1st European Workshop in Periodontology.[2] These classification systems have been widely used by clinicians and research scientists throughout the world. Unfortunately, the 1989 classification had many shortcomings including: 1) considerable overlap in disease categories, 2) absence of a gingival disease component, 3) inappropriate emphasis on age of onset of disease and rates of progression, and 4) inadequate or unclear classification criteria. The 1993 European classification lacked the detail necessary for adequate characterization of the broad spectrum of periodontal diseases encountered in clinical practice. The need for a revised classification system for periodontal diseases was emphasized during the 1996 World Workshop in Periodontics.[3] In 1997 the American Academy of Periodontology responded to this need and formed a committee to plan and organize an international workshop to revise the classification system for periodontal diseases. The proceedings in this volume are the result of this reclassification effort. The process involved development by the Organizing Committee of an outline for a new classification and identification of individuals to write state-of-the-science reviews for each of the items on the outline. The reviewers were encouraged to depart from the preliminary outline if there were data to support any modifications. On October 30–November 2, 1999, the International Workshop for a Classification of Periodontal Diseases and Conditions was held and a new classification was agreed upon (Fig. 1). This paper summarizes how the new classification for periodontal diseases and conditions presented in this volume differs from the classification system developed at the 1989 World Workshop in Clinical Periodontics.[1] In addition, an analysis of the rationale is provided for each of the modifications and changes. *Ann Periodontol 1999;4:1-6.*

KEY WORDS

Periodontal diseases/classification; gingival diseases/classification.

CHANGES IN THE CLASSIFICATION SYSTEM FOR PERIODONTAL DISEASES

Addition of a Section on "Gingival Diseases"

As mentioned above, the 1989 classification did not include a section on gingival diseases. This has been remedied by the development of a detailed classification of gingival diseases and lesions that are either dental plaque-induced (pages 18-19) or not primarily associated with dental plaque (pages 30-31). An important feature of the section on dental plaque-induced diseases is acknowledgment that the clinical expression of gingivitis can be substantially modified by: 1) systemic factors such as perturbations in the endocrine system, 2) medications, and 3) malnutrition. The section on non-plaque induced gingival lesions includes a wide range of disorders that affect the gingiva. Many of these disorders are frequently encountered in clinical practice.

Replacement of "Adult Periodontitis" With "Chronic Periodontitis"

From the outset, the term "Adult Periodontitis" created a diagnostic dilemma for clinicians. Epidemiologic data and clinical experience suggest that the form of periodontitis commonly found in adults can also be seen in adolescents.[4] If this is true, how can non-adults (e.g., adolescents) with this type of periodontitis be said to have "adult periodontitis?" Clearly, the age-dependent nature of the adult periodontitis designation created problems. Therefore, workshop participants concluded that it would be more

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 gonorrhea*-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 candidosis
      b. linear gingival erythema
      c. histoplasmosis
      d. other
    4. Gingival lesions of genetic origin
      a. hereditary gingival fibromatosis
      b. other
    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 (NOS)

Figure 1.
Classification of periodontal diseases and conditions.
* Can occur on a periodontium with no attachment loss or on a periodontium with attachment loss that is not progressing.

accurate to adopt a nonspecific term such as "Chronic Periodontitis" to characterize this constellation of destructive periodontal diseases.

A great deal of discussion centered around what words should be used to replace the Adult Periodontitis term. Substitute terminology such as "Periodontitis—Common Form" and "Type II Periodontitis" were considered and eventually rejected by the majority of the group. The term "Chronic Periodontitis" was criticized by some participants, since "chronic" might be interpreted as "noncurable" by some people. Nevertheless, "Chronic Periodontitis" was eventually agreed upon as long as it was understood that it did not imply that this disease was nonresponsive to treatment.

Traditionally, this form of periodontitis has been characterized as a slowly progressive disease.[5] Indeed, data from many sources confirm that patients with this form of periodontitis usually exhibit slow rates of progression.[6,7] However, there are also data indicating that some patients may experience short periods of rapid progression.[8,9] Therefore, workshop participants concluded that rates of progression should not be used to exclude people from receiving the diagnosis of Chronic Periodontitis.

Replacement of "Early-Onset Periodontitis" With "Aggressive Periodontitis"

The term "Early-Onset Periodontitis" (EOP) was used in the 1989 AAP and 1993 European classifications as a collective designation for a group of dissimilar destructive periodontal diseases that affected young patients (i.e., prepubertal, juvenile, and rapidly pro-

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)
    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 periodontic-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

Figure 1. (Continued)
† 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 or 2 mm CAL, Moderate = 3 or 4 mm CAL, and Severe = ≥5 mm CAL.

gressive periodontitis). It was logically assumed that these diseases all had an early onset because they affected young people. Unfortunately, the "early onset" designation implies that one has temporal knowledge of when the disease started. However, in clinical practice and most other situations this is rarely the case. In addition, there is considerable uncertainty about arbitrarily setting an upper age limit for patients with so-called early-onset periodontitis. For example, how does one classify the type of periodontal disease in a 21-year-old patient with the classical incisor-first molar pattern of Localized Juvenile Periodontitis (LJP)? Since the patient is not a juvenile, should the age of the patient be ignored and the disease classified as LJP anyway? This type of problem stems from the age-dependent nature of the 1989 classification system. A similar problem arises when the 1989 classification is applied to a 21-year-old patient with generalized periodontal destruction. Does such a patient have "Rapidly Progressing Periodontitis" (RPP) or "Generalized Juvenile Periodontitis" (GJP)? It can be argued that neither designation is acceptable. The diagnosis of RPP may not be appropriate since the rate of progression is not known, and the GJP designation is unacceptable because the patient is no longer a juvenile.

Because of these problems, workshop participants decided that it was wise to discard classification terminologies that were age-dependent or required knowledge of rates of progression. Accordingly, highly destructive forms of periodontitis formerly considered under the umbrella of "Early-Onset Periodontitis" were renamed using the term "Aggressive Periodontitis." In general, patients who meet the clinical criteria for LJP or GJP are now said to have "Localized Aggressive Periodontitis" or "Generalized Aggressive Periodontitis," respectively. In the consensus report for "Aggres-

sive Periodontitis" (page 53), workshop participants have listed some characteristics that should be helpful in distinguishing between localized and generalized forms of this group of periodontal diseases. Since these features have not been universally used in the older literature to place patients in the LJP or GJP categories, it would be inappropriate to assume that there will be a consistent one-to-one relationship in transferring information from the old classification system to the new. For example, some patients formerly classified as having GJP in the older literature might appropriately be placed in either the Chronic Periodontitis or Generalized Aggressive Periodontitis categories in the new classification system, depending on a variety of primary and secondary characteristics.

The Rapidly Progressive Periodontitis (RPP) designation has been discarded. Patients who were formerly classified as having RPP will, depending on a variety of other clinical criteria, be assigned to either the "Generalized Aggressive Periodontitis" or "Chronic Periodontitis" categories. It should be emphasized that patients with rapidly progressive forms of periodontitis exist. They do not, however, represent a homogeneous group.

The 1989 classification contained a category termed "Prepubertal Periodontitis" which had localized and generalized forms. The category was originally developed to accomodate those rare situations in which children with primary teeth had severe periodontal destruction. It is now known that most of the patients who have been given the diagnosis of generalized prepubertal periodontitis actually had one of a variety of systemic conditions that interfere with resistance to bacterial infections. Such conditions include leukocyte adherence deficiency,[10,11] congenital primary immunodeficiency,[12] hypophosphatasia,[13] chronic neutrophil defects,[14,15] or cyclic neutropenia.[16] Under the new classification system, such patients would be placed under the heading of "Periodontitis as a Manifestation of Systemic Diseases" (page 64).

Workshop participants agreed that prepubescent children who have periodontal destruction without any modifying systemic conditions would, depending on a variety of secondary features, fit under the categories of "Chronic Periodontitis" or "Aggressive Periodontitis" in the new classification. The idea that periodontitis has its beginnings in childhood is supported by retrospective epidemiologic data suggesting that localized radiographic bone loss can be detected around the primary dentition of some children.[17-19] In addition, generalized periodontitis has also been reported in young children without any detectable underlying systemic disease.[20] The concept that periodontitis develops at an early age is strengthened by data from many epidemiologic studies demonstrating that periodontal attachment loss can be found around the permanent teeth of adolescents.[21-31]

Elimination of a Separate Disease Category for "Refractory Periodontitis"

In the 1989 classification, a separate disease category was devoted to Refractory Periodontitis. This heterogeneous group of periodontal diseases refers to instances in which there is a continuing progression of periodontitis in spite of excellent patient compliance and the provision of periodontal therapy that succeeds in most patients. Because of the diversity of clinical conditions and treatments under which periodontal therapy fails to arrest the progression of periodontitis, workshop participants were of the opinion that "Refractory Periodontitis" is not a single disease entity. Indeed, it was considered possible that a small percentage of cases of all forms of periodontitis might be nonresponsive to treatment. Therefore the group concluded that, rather than a single disease category, the "refractory" designation could be applied to all forms of periodontitis in the new classification system (e.g., refractory chronic periodontitis, refractory aggressive periodontitis, etc.). It is recommended that future studies of these patients describe as fully as possible the population under investigation to minimize heterogeneity of the study sample.

Clarification of the Designation "Periodontitis as a Manifestation of Systemic Diseases"

In the 1989 classification, one of the disease categories was "Periodontitis Associated With Systemic Disease." In general, this category has been retained in the new classification since it is clear that destructive periodontal disease can be a manifestation of certain systemic diseases. The Consensus Report for this portion of the workshop (page 64) contains of list of systemic diseases in which periodontitis is a frequent manifestation. It should be noted that diabetes mellitus is not on this list. In the collective view of workshop participants, diabetes can be a significant modifier of all forms of periodontitis but there are insufficient data to conclude that there is a specific diabetes mellitus-associated form of periodontitis. For example, the presence of uncontrolled diabetes mellitus can alter the clinical course and expression of chronic and aggressive forms of periodontitis. Similarly, the new classification does not contain a separate disease category for the effects of cigarette smoking on periodontitis. Smoking was considered to be a significant modifier of multiple forms of periodontitis.

One of the apparent inconsistencies in the new system is inclusion in the "Dental Plaque-Induced Gingival Diseases" (pages 18-19) portion of the classification a list of gingival diseases that can be modified by systemic factors. On this list is "diabetes mellitus-associated gingivitis." How can one justify inclusion of a diabetes mellitus-associated gingivitis category and purposely exclude a parallel periodontitis category?

The reason for this decision was that plaque-induced gingivitis was considered a single entity by the workshop participants. This is not the case for periodontitis, where there are clearly different clinical forms. It would have been possible to include in the new classification additional subcategories such as "diabetes mellitus-associated chronic periodontitis" and "diabetes mellitus-associated aggressive periodontitis." However, the group decided that this would be unnecessarily complicated and not yet justified by supporting data.

Replacement of "Necrotizing Ulcerative Periodontitis" With "Necrotizing Periodontal Diseases"

Workshop participants acknowledged that necrotizing ulcerative gingivitis (NUG) and necrotizing ulcerative periodontitis (NUP) are clinically identifiable conditions. However, the group was less certain about the relationship between NUG and NUP. Are these clinical conditions part of a single disease process or are they truly separate diseases? Since there are insufficient data to resolve these issues, the group decided to place both clinical conditions under the single category of "Necrotizing Periodontal Diseases." If future studies show that NUG and NUP are fundamentally different diseases, then they can be separated in subsequent revisions of the classification.

One of the potential problems with inclusion of "Necrotizing Periodontal Diseases" as a separate category is that both NUG and NUP might be manifestations of underlying systemic problems such as HIV infection. If this is true, then it might be more appropriate to place these conditions under manifestations of systemic diseases. The reason that this was not done is that there are many factors, other than systemic diseases, that appear to predispose to the development of NUG or NUP such as emotional stress and cigarette smoking. Since our understanding of these clinical conditions is far from complete, it was concluded that for the time being they should be included under a single and separate category in the new classification.

Addition of a Category on "Periodontal Abscess"

The 1989 classification did not include a section on periodontal abscesses. This has been remedied by the addition of a simple classification (page 83) primarily based on location (i.e., gingival, periodontal, pericoronal) of these commonly encountered lesions. It could be argued that periodontal abscesses are part of the clinical course of many forms of periodontitis and formation of a separate disease category is not justified. However, in the view of workshop participants, since periodontal abscesses present special diagnostic and treatment challenges they deserve to be classified apart from other periodontal diseases.

Addition of a Category on "Periodontic-Endodontic Lesions"

The 1989 classification did not include a section on the connection between periodontitis and endodontic lesions. Therefore a simple classification dealing with this area has been added (page 90).

Addition of a Category on "Developmental or Acquired Deformities and Conditions"

Although the deformities and conditions listed in this section of the classification are not separate diseases, they are important modifiers of the susceptibility to periodontal diseases or can dramatically influence outcomes of treatment. In addition, since periodontists are routinely called upon to treat many of these conditions they have been given a place in the new classification (page 101).

FUTURE REVISIONS TO THE CLASSIFICATION

The classification of periodontal diseases and conditions in this volume should provide a workable framework upon which to study and develop effective treatments for this complex group of infections. It is anticipated that as we learn more about the etiology and pathogenesis of periodontal diseases, future revisions to the classification will be needed. All classification systems have inconsistencies or inaccuracies. The present effort is no exception. Nevertheless, the current classification represents the consensus of an international group of experts and it is hoped that the system will be useful to the profession and public we serve.

REFERENCES

1. The American Academy of Periodontology. *Proceedings of the World Workshop in Clinical Periodontics*. Chicago: The American Academy of Periodontology; 1989:I/23-I/24.
2. Attström R, van der Velden U. Consensus report (epidemiology). In: Lang NP, Karring T, eds. *Proceedings of the 1st European Workshop on Periodontics, 1993*. London: Quintessence; 1994;120-126.
3. Armitage GC. Periodontal diseases: Diagnosis. *Ann Periodontol* 1996;1:37-215.
4. Papapanou PN. Periodontal diseases: Epidemiology. *Ann Periodontol* 1996;1:1-36.
5. Brown LJ, Löe H. Prevalence, extent, severity and progression of periodontal disease. *Periodontol 2000* 1993; 2:57-71.
6. Löe H, Anerud A, Boysen H, Morrison E. Natural history of periodontal disease in man. Rapid, moderate and no loss of attachment in Sri Lankan laborers 14 to 46 years of age. *J Clin Periodontol* 1986;13:431-440.
7. Papapanou PN, Wennström JL, Gröndahl K. A 10-year retrospective study of periodontal disease progression. *J Clin Periodontol* 1989;16:403-411.
8. Socransky SS, Haffajee AD, Goodson JM, Lindhe J. New concepts of destructive periodontal disease. *J Clin Periodontol* 1984;11:21-32.
9. Jeffcoat MK, Reddy MS. Progression of probing attachment loss in adult periodontitis. *J Periodontol* 1991;

62:185-189.
10. Waldrop TC, Anderson DC, Hallmon WW, Schmalstieg FC, Jacobs RL. Periodontal manifestations of the heritable Mac-1, LFA-1, deficiency syndrome. *J Periodontol* 1987;58:400-416.
11. Meyle J. Leukocyte adhesion deficiency and prepubertal periodontitis. *Periodontol 2000* 1994;6:26-36.
12. Batista EL Jr, Novaes AB Jr, Calvano LM, et al. Necrotizing ulcerative periodontitis associated with severe congenital immunodeficiency in a prepubescent subject: clinical findings and response to intravenous immunoglobulin treatment. *J Clin Periodontol* 1999;26:499-504.
13. Plagmann H-C, Kocher T, Kuhrau N, Caliebe A. Periodontal manifestation of hypophosphatasia. A family case report. *J Clin Periodontol* 1994;21:710-716.
14. Dougherty N, Gataletto MA. Oral sequelae of chronic neutrophil defects: case report of a child with glycogen storage disease type 1b. *Pediatric Dent* 1995;17:224-229.
15. Kamma JJ, Lygidakis NA, Nakou M. Subgingival microflora and treatment in prepubertal periodontitis associated with chronic idiopathic neutropenia. *J Clin Periodontol* 1998;25:759-765.
16. Prichard JF, Ferguson DM, Windmiller J, Hurt WC. Prepubertal periodontitis affecting the deciduous dentition and permanent dentition in a patient with cyclic neutropenia. A case report and discussion. *J Periodontol* 1984;55:114-122.
17. Sweeney EA, Alcoforado GAP, Nyman S, Slots J. Prevalence and microbiology of localized prepubertal periodontitis. *Oral Microbiol Immunol* 1987;2:65-70.
18. Bimstein E, Delaney JE, Sweeney EA. Radiographic assessment of the alevolar bone in children and adolescents. *Pediatric Dent* 1988;10:199-204.
19. Sjödin B, Matsson L. Marginal bone loss in the primary dentition. A survey of 7–9-year-old children in Sweden. *J Clin Periodontol* 1994;21:313-319.
20. Bimstein E, Sela MN, Shapira L. Clinical and microbial considerations for the treatment of an extended kindred with seven cases of prepubertal periodontitis: a 2-year follow-up. *Pediatric Dent* 1997;19:396-403.
21. Lennon MA, Davies RM. Prevalence and distribution of alveolar bone loss in a population of 15-year-old schoolchildren. *J Clin Periodontol* 1974;1:175-182.
22. Aass AM, Albandar J, Aasenden R, Tollefsen T, Gjermo P. Variation in prevalence of radiographic alveolar bone loss in subgroups of 14-year-old schoolchildren in Oslo. *J Clin Periodontol* 1988;15:130-133.
23. van der Velden U, Abbas F, van Steenbergen TJM, et al. Prevalence of periodontal breakdown in adolescents and presence of *Actinobacillus actinomycetemcomitans* in subjects with attachment loss. *J Periodontol* 1989; 60:604-610.
24. Källestål C, Matsson L, Holm A-K. Periodontal conditions in a group of Swedish adolescents. (I). A descriptive epidemiologic study. *J Clin Periodontol* 1990;17:601-608.
25. Källestål C, Matsson L. Marginal bone loss in 16-year-old Swedish adolescents in 1975 and 1988. *J Clin Periodontol* 1991;18:740-743.
26. Bhat M. Periodontal health of 14–17-year-old US schoolchildren. *J Public Health Dent* 1991;51:5-11.
27. Cappelli DP, Ebersole JL, Kornman KS. Early-onset periodontitis in Hispanic-American adolescents associated with *A. actinomycetemcomitans*. *Community Dent Oral Epidemiol* 1994;22:116-121.
28. Aass AM, Tollefsen T, Gjermo P. A cohort study of radiographic alveolar bone loss during adolescence. *J Clin Periodontol* 1994;21:133-138.
29. Clerehugh V, Worthington HV, Lennon MA, Chandler R. Site progression of loss of attachment over 5 years in 14- to 19-year-old adolescents. *J Clin Periodontol* 1995; 22:15-21.
30. Albandar JM, Brown LJ, Brunelle JA, Löe H. Gingival state and dental calculus in early-onset periodontitis. *J Periodontol* 1996;67:953-959.
31. Timmerman MF, van der Weijden GA, Armand S, et al. Untreated periodontal disease in Indonesian adolescents. Clinical and microbiological baseline data. *J Clin Periodontol* 1998;25:215-224.

Send reprint requests to: Dr. Gary C. Armitage, University of California San Francisco, School of Dentistry, 521 Parnassus Ave., C-628, Box 0650, San Francisco, CA 94143-0650. Fax: 415/502-4990; e-mail: garmit@itsa.ucsf.edu