A Chronological Classification of Periodontal Disease: A Review

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Abstract

The subject of classification may be considered to be dull by some people, even contentious by others, but it does afford us a means to an end. The evolution of the history allows us to see how our predecessors thought and how periodontal disease has extended itself as an important entity into many aspects of the medical field. A good classification system allows us to understand the complexity of the disease that we are attempting to treat. This is a subject that Drs. I Glickman, I Weinmann, B Orban and the 1987 and 1999 American Academy of Periodontology have tried to teach. This paper presents a review of both historical and modern classification systems for periodontal disease.

1. Kantorowicz - 1924

I. Inflammatory disease

A. Paradentitis

II. Dystrophic disease with little inflammation

A. Presenile atrophy
B. Dystrophy from occlusal trauma
C. Dystrophy from lack of occlusion
D. Diffuse atrophy

2. McCall and Box - 1925

I. Gingivitis

A. Acute
B. Chronic

II. Periodontitis

A. Acute
B. Chronic

III. Periodontitis simplex (exogenous factors)

IV. Periodontitis complex (or rarefying pericementitis fibrosa - endogenous factors)

3. Simonton - 1927

I. Chemobacterial

A. Paradentitis

II. Systemic

A. Paradentitis
B. Diffuse atrophy

4. Haupl and Lang - 1927

I. Paradentitis

A. Marginal paradentitis (etiology includes mechanical, thermal, chemical and infectious factors as well as functional disturbances, tooth malformation, systemic disturbances, etc.
B. Superficial marginal paradentitis
1. Epithelial changes - regressive and progres sive

5. Gottlieb - 1928

I. Schmutzpyorrhea

II. Degenerative or atrophic

III. Diffuse alveolar atrophy (systemic/metabolic)

IV. Paradental pyorrhea

6. Becks - 1929/1931

I. Paradentitis

A. Simple
B. Secondary

II. Paradentosis

A. Presenile atrophy
B. Paradentosis due to trauma
C. Paradentosis due to lack of occlusion
D. Diffuse alveolar atrophy
E. Paradentosis secondary to paradontitis

III. Paradentoma

7. Jaccard - 1930/1933

I. Inflammatory complex

A. Pure gingivitis
B. Preparadontal gingivitis
C. Inflammatory paradentosis

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II. Osteopathic dystrophic complex

A. Dystrophic paradentosis
B. Presenile atrophy
C. Senile atrophy

8. Roy - 1935

I. Alveolar pyorrhea, characterized by precocious senile alveolar resorption due to upset in general constitution of the individual

II. Formation of thp ocket

III. Connective tissue changes

A. Subepithelial
B. Supraepithelial

IV. Changes in paradental bone

V. Marginal paradentitis profunda

VI. Apical paradentitis

9. Robinson - 1935

I. Clinicalyp es qf aradentosis

A. Ortho pyorrhea (classical form)
B. Hypertrophic pyorrhea (no alveolar resorption and common in young people)
C. Pyorrhea with pockets - common, hyperemic, ischemic
D. Pyorrhea without pockets – no gingivitis, juvenile atrophy, osteoporosis, deformatory pyorrhea
E. Rubro pyorrhea - uniform redness of the gingiva and marked tooth attrition seen
F. Senile pyorrhea - a physiological alveolar resorption complicated by peridental inflammation

Classification systems vary to reflect current changes in our knowledge about periodontal infections. In the early days Kantorowicz, Simonton, Box and McCall, Haupi and Lang, and Gottlieb's classification all were very simple but adequate for the time (1924-1928). It was not until 1929-1933, as Becks and Jaccard began to approach the more scientific type of classification, that we begin to see a change in thinking as far as their knowledge of the disease process would permit.

10. Weski - 1937

I. Paradentitis (gingivitis) - hypertrophic, simple, ulcerative

II. Paradentosis p artial (true form of paradentosis) and total (alveolar atrophy)

III. Paradentoma - epulis (localized form), elephantiasis gingivae (generalized form)

Weski's classification was a reversal to the 1924-1928 thinking by keeping it simple.

11. Isadore Weinmann - 1934-1957

I. Marginal gingivitis

A. Non-suppurative
   1. Due to calculus
   2. Faulty dentistry
   3. Pregnancy - transitory nature
   4. Blood dyscrasias
   5. Cardiac disease - especially the right side of the heart
   6. Renal disturbance
   7. Metallic poisoning
   8. Diabetes
   9. Scorbutus
   10. Avitaminosis
   11. Food allergy
B. Suppurative - may result from long-term chronicity of any of the preceding causes. It may occur suddenly in debilitated individuals if the tissues are overwhelmed by pyogenic microorganisms.

II. Gingival recession

A. Crescents - due to traumatic occlusion in its early stages, usually fibrous in nature.
B. Clefts - usually follow crescents
C. Festoons - usually due to toothbrush trauma
D. Atrophic - physiologic due to age, or pathologic due to disease

III. Hypertrophic gingivitis

A. Physical irritation
B. Post-nasal obstruction (mouth breathers)
C. Blood dyscrasias (particularly leukemia)
D. Pregnancy
E. Scorbutus
F. Endocrine disturbance
G. Prolonged fever
H. Metabolic disturbance

Hypertrophies are of two types. There are hard, dense nodular masses (fibrosis), as seen in mouth breathers; or soft, spongy, jelly-like masses made up of poorly organized granulation tissues and many faulty blood vessels, as seen in pregnancy or advanced scorbutus.

IV. Ulcero-membranous gingivitis

A. Vincent's infection
B. Tuberculosis ulcers
C. Aphthous ulcers
D. Noma

V. Alveolar atrophy (true alveolaclasia)

A. Normal or physiological due to age (senile arteriosclerosis even of slight degree)
B. Pathologic

1. Local causes
   a. Faulty occlusion, food impaction, toothpicks, pipes, etc., loss of contacts, faulty dentistry
   b. Due to involvement from inflammatory changes in the soft tissues
2. Systemic causes
   a. Diabetes
   b. Syphilis
   c. Dysfunction of the endocrines or any meta bolic disturbance

Finally, in 1934, Weinmann's classification began a more modern era and showed the true magnitude of periodontal disease, beginning with gingival diseases such as gingivitis (both non-suppurative and suppurative), gingival recession, hypertrophic gingivitis and ulceromembranous gingivitis. The category of non-suppurative gingivitis lists many medical areas that are repeated again in hypertrophic gingivitis. This was followed by alveolar atrophy (periodontitis) covering faulty occlusion, habit neuroses and systemic causes that are also mentioned under gingivitis. The extent of periodontitis coverage was limited by the knowledge of the time.

12. Thoma and Goldman - 1937

I. Inflammatory conditions

A. Gingivitis - marginal, hypertrophic, ulcerative
B. Marginal paradontitis

II. Degenerative conditions

A. Paradontosis (bone destruction affecting other periodontal structures)
B. Atrophy
   1. Gingival recession
   2. Presenile atrophy
   3. Disuse atrophy
   4. Atrophy due to abnormal occlusal trauma
C. Syndrome of paradentitis and paradentosis

13. Fish - 1944/1952

I. Gingivitis

A. Acute ulcerative
B. Subacute marginal
C. Chronic marginal
D. Traumatic

II. Pyorrhea

A. Pyorrhea simplex/profunda
B. Senile alveolar resorption
C. Neoplasia
D. Odontoclasma
E. Cementoma
F. Fibrous epulis

14. Hine and Hine - 1944

I. Inflammation

A. Gingivitis (local calculus, faulty restorations, poor contact areas, drugs)
B. Systemic - nutritional deficiencies, blood dyscrasias, etc.
C. Periodontitis simplex (more severe irritation than gingivitis)
D. Specific entities (tuberculosis, syphilis, radiation)

II. Atrophy or degeneration

A. Gingival recession
   1. Trauma
   2. Senile
   3. Disuse
   4. Idiopathic
B. Periodontitis complex
C. Periodontitis - systemic disturbances
   1. Degeneration of connecting fibers in periodontal membrane
   2. Bone resorption
D. Hypertrophy
   1. Gingival hypertrophy may accompany gingivitis
   2. Trauma
   3. Senile
   4. Disuse
   5. Idiopathic

15. Orban - 1942/1949

I. Inflammatory conditions

A. Gingivitis
   1. Localized to the free margins of the gingiva, swelling, and shallow pockets
   2. Acute or chronic according to duration
   3. Ulcerative or purulent according to symptoms
   4. Local (extrinsic) infections (physical or chemical)/systemic (intrinsic)
   5. Dietary deficiency, endocrine disturbances

II. Degenerative conditions

A. Gingivosis - systemic etiology, degeneration of connective tissue
B. Periodontosis - degeneration of collagenous fibers of the periodontal membrane; irregular bone resorption; primarily systemic etiology-inherited inferiority of the dental organ. Early, no inflammation; later, deep pockets with periodontitis

III. Atrophic conditions

A. Periodontal atrophy—bone recession
B. Precocious aging
C. Disuse - loss of normal function
D. Trauma - toothbrush/orthodontia
E. Periodontal traumatism - pressure

F. Necrosis and its consequences
1. Primary (overstress/bruxism)
2. Secondary (loss of supporting tissues)

16. Hulin - 1949

I. Inflammatory processes

A. Paradontitis
1. Exogenous gingivitis
a. Tartar
b. Bacteria
2. Endogenous gingivitis
a. Avitaminosis
b. Intoxication

II. Degenerative processes

A. Paradontosis
1. Precocious senile atrophy
2. Juvenile paradontosis
3. Senile paradontosis
4. Pyorrhetic paradontosis
5. Traumatic paradontolysis
B. Paradontomes
1. Epulis/gingival elephantiasis

17. Held - 1949

I. True paradontopathia

A. Gingivitis
B. Paradontolysis
1. Paradontitis
2. Periodontal atrophy
3. Symptomatic paradontopathia - avitaminosis, blood dyscrasias, etc.
C. Enlargement conditions
1. Epulis, elephantiasis gingivae

18. Pucci - 1950

I. Marginal paradentitis

A. Incipient
B. Hypertrophic
C. Desquamative
D. Localized
E. Advanced

II. Paradentosis

A. Atrophic
B. Constitutional
C. Horizontal alveolar atrophy with marginal paradentitis
1. Pure form
2. Complicated form
3. Alveolar decalcification
D. Physiologic alveolar atrophy
1. Horizontal-precocious senile
2. Accelerated passive eruption

19. Miller - 1950

I. Gingivitis - acute, subacute, etc.

II. Periodontal abscess

A. Parodonta
B. Pericemental
C. Periapical

III. Alveolaclasia - bone resorption

A. Nutritional deficiencies
B. Endocrinopathic

IV. Pericementoclasia - pocket formation

V. Ulatrophia - ischemic, calcic, afunctional, traumatic

20. Lyons - 1951

I. Inflammatory

A. Gingivitis
1. Acute
2. Simple, purulent, necrotizing
B. Chronic
1. Simple, purulent, necrotizing
2. Hyperplastic, desquamative, pigmented
C. Periodontitis
1. Simplex
2. Complex
D. Retrogressive periodontosis
1. Atrophic periodontosis, senile, presenile, hyperfunctional. Periodontosis gravis includes deficiency diseases, endocrinopathies, systemic toxicities, blood dyscrasias and metabolic diseases

II. Neoplastic

A. Benign
1. Fibroma
2. Elephantiasis gingivae
B. Malignant

21. Kerr - 1951

I. Gingivitis - simple, infective, hormonal, atrophic, herpetic gingivostomatitis

II. Periodontitis

III. Periodontosis

IV. Traumatism

22. Goldman - 1956

I. Inflammation

A. Gingivitis
B. Marginal periodontitis

II. Dystrophy

A. Disease atrophy
B. Occlusal traumatism
C. Gingivosis
D. Periodontosis

23. McCall - 1956

I. Primary (process originates in the periodontium

A. Productive processes (periodontal hyperplasia)
B. Destructive processes (periodontitis)
C. Degenerative processes (periodontosis)

II. Secondary (process originates outside the periodontium)

A. Diseases and non-pathological conditions having specific periodontal effects
B. Diseases having non-specific periodontal effects
C. Neoplasms

24. American Academy of Periodontology - 1957

I. Inflammation

A. Gingivitis
B. Periodontitis
   1. Primary (simplex)
   2. Secondary (complex)
C. Dystrophy
   1. Occlusal traumatism
   2. Periodontal disuse atrophy
   3. Gingivosis
   4. Periodontosis

25. Robinson - 1959

I. Gingivitis

II. Periodontitis

III. Periodontosis

IV. Atrophy

A. Hypertrophy and hyperplasia
B. Traumatism

26. Carranza - 1959

I. Inflammatory periodontal syndrome

A. Superficial
B. Deep

II. Traumatic periodontal syndrome

A. Compensated
B. Uncompensated

III. Combined periodontal syndrome

A. Compensated
B. Uncompensated

27. Ray - 1962

I. Inflammatory

A. Gingivitis
B. Periodontitis

II. Degenerative

A. Gingivosis
B. Periodontosis
C. Trauma
D. Atrophy

III. Proliferative

A. Gingival hyperplasia
B. Periodontal neoplasms

From 1937-1962, the classifications tended to be shorter in an attempt to reduce their complexity. However, the classification systems of Thoma and Goldman, Fish, Hine and Hine, Orban, Hulin, Held, Puci, Lyons, Miller, Kerr, Goldman, McCall, The American Academy of Periodontology, Robinson, Carranza, and Ray all fall short of being thorough. All the proposed classification systems for this 25-year period did not reflect any significant improvement.

28. Glickman - 1964

I. Classification of gingival disease

A. Uncomplicated gingivitis
   1. Chronic marginal gingivitis
   2. Acute necrotizing ulcerative gingivitis (NUG)
   3. Acute herpetic gingivostomatitis and other viral infections
   4. Allergic gingivitis
   5. Nonspecific gingivitis
   6. TB and syphilis
   7. Moniliasis and other fungal infections
   8. Pyostomatitis vegetans
B. Combined gingivitis
   1. Dermatoses
   2. Chronic desquamative gingivitis (gingivosis)
   3. Chronic menopausal gingivostomatitis (senile atrophic gingivitis)
   4. Benign mucous membrane pemphigoid
C. Conditioned gingivitis
   1. Gingivitis in pregnancy and puberty
   2. Gingivitis in vitamin C deficiency
   3. Gingivitis in leukemia

D. Gingival enlargement
   1. Inflammatory
   2. Noninflammatory hyperplastic
   3. Combined
   4. Conditioned
   5. Neoplastic
   6. Developmental

E. Recession
   1. Gingival atrophy

II. Classification of periodontal disease

A. Periodontitis
   1. Simple periodontitis
   2. Compound periodontitis

B. Periodontosis
   1. Early
   2. Advanced

C. Trauma from occlusion

D. Periodontal atrophy
   1. Presenile atrophy
   2. Disuse atrophy

Glickman's classification scheme was an attempt to support that of Weinmann in 1934 and to steer the ship in the correct direction, so to speak, but his classification of periodontitis failed to categorize osseous problems even though the use of hip marrow grafting was already common practice. Also, the gingival disease portion of Glickman's classification had a lot of overlap with systemic disease.

29. Drum - 1975

I. Drum felt that "all persons afflicted by periodontal diseases exert parafunctions" that may be classified as:

A. Physically motivated parafunctions - neurotic phenomena such as bruxism, bruxomania, nail biting, thumb sucking, etc.

B. Stress-motivated parafunctions - non-neurotic phenomena such as stress caused by combat or severe pain, or that suffered by athletes, truck drivers, workers on high-rise buildings/bridges, etc.

C. Habitual parafunctions - tailors, seamstresses, bootmakers, upholsterers, wind instrument players, chewers of pens/pencils, etc.

D. Endogenous parafunctions - muscle spasms caused by diseases such as tetanus, meningitis, epilepsy, etc.
E. Hyper-compensating parafunctions - exaggeration of normal compensating motions triggered by occlusal interferences and other disturbances in the mouth

II. These depend upon the following factors:

A. Force of the parafunctions

B. Directions of that force

C. Duration of the parafunctions

D. Intervals between periods of parafunctions

E. Configuration of the roots

F. Configurations of the alveolar bone

G. Position of teeth

Drum was attempting to revitalize some older theories that most, if not all, periodontal problems could be corrected by occlusal equilibration. A better explanation of the aforementioned may be better understood through the review and the application of Frost's Laws of Bone Formation, and reference to Sorrin's classification of habitual contributions to periodontal disease and its revision by Weinmann.

30. American Academy of Periodontology - 1987

I. Gingival disease

A. Gingivitis
   1. Nonspecific (plaque, bacteria, and their products)
   2. Acute necrotizing ulcerative gingivitis (ANUG)

B. Manifestations of systemic diseases and hormonal disturbances
   1. Acute herpetic gingivostomatitis
   2. Blood dyscrasias
   3. Leukemias
   4. Autoimmune diseases (pemphigus)
   5. Diabetes
   6. Sex hormones

C. Drug-associated - gingival inflammation and/or enlargement (dilantin hyperplasia)

D. Miscellaneous gingival changes associated with various etiologies
   1. Atrophy
   2. Cyst formation
   3. Hyperplasia
   4. Neoplasia
   5. Infection
   6. Irritation
   7. Trauma

II. Mucogingival conditions

A. Recession

B. Frena

C. Muscle attachments

D. Minimal attached gingivae

III. Periodontitis

A. Adult periodontitis
   1. Slight
   2. Moderate
   3. Advanced
   4. Refractory
B. Juvenile periodontitis (JP)
   1. Prepubertal (generalized or local)
   2. Generalized (GJP)
   3. Localized (LJP) - thought to be associated with
      a. *Aggregatibacter actinomycetemcomitans*
      b. Heredity
      c. Abnormalities in white blood cell functions

   (predilection for central incisors and first molars)
C. Periodontal abscess

IV. Pathology associated with occlusion (associated with temporo-mandibular joint disorders)

A. Trauma from occlusion
B. Bruxism

V. Other conditions of the attachment apparatus and all pathologic processes of the periodontium, including:

A. Infection
B. Abrasion
C. Trauma
D. Cystic changes
E. Degenerative changes
F. Neoplastic changes

The 1987 Academy of Periodontology Classification refined and better organized the systems expressed by Weinmann (1934) and Glickman (1964). This was the first time that drug-associated gingival disease was recognized, but the list was still far short on this subject as well as the classification of systemic disease and endocrine involvement. The periodontitis section was broader in depth of coverage than all of its predecessors.

31. American Academy of Periodontology - 1999

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)

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

It appears that throughout history we went from short to lengthy to moderate to longer classifications as we realized the multiple complications of pure periodontitis to the role of systemic factors in the periodontal disease process and the role of periodontal disease in contributing to medical problems. Now we are considering the enormity of periodontal disease as it relates to the patients' overall health. While most of the classifications listed are for pure historical comparison, the only classifications that are of significant importance are those of Dr. I. Weinmann (1934-1957), Dr. I. Glickman (1964), and The American Academy of Periodontology (1987 and 1999). These all show the value of a good and thorough classification.

But, in the 1999 classification the following are points of disagreement; (1) the word "other" is used too freely; (2) under "drug-influenced gingival diseases" it does not mention the effects of alcohol, cocaine, heroin, crack and heart medications (alpha and beta blockers, Mevacor, and Lipitor) that are well documented in the literature as causing increased plaque formation and stimulating gingival overgrowth; (3) there is no discussion of temperomandibular joint (TMJ) problems and stress as aggravating factors in periodontal disease; (4) there is no mention of biochemical mediators of gingival crevicular fluid and their effects on periodontal tissues (such as cytokines IL-1α, IL-1β, IL-6, IL-8, IL-10, TNF-α, MMP_3, PGE_2, etc.); (5) the section on occlusal trauma does not in our opinion adequately cover the magnitude of the pathology associated with occlusion, malocclusion, and the habit neuroses that alter occlusion and contribute to TMJ malfunction; (6) we still believe in the breakdown of "adult periodontitis" over "chronic periodontitis" (especially to the lay person to whom chronic means non-curable and because it is non-curable the patient(s) would wonder why they should bother to undergo treatment); and (7) with juvenile (early onset) periodontitis, which is age-related instead of "aggressive periodontitis" which means "rapidly progressing

periodontitis" (RPP) independent of age despite totally adequate periodontal therapy. These are two separate entities and should be recognized as such; (8) there is still considerable overlap in disease categories. For example, in aggressive periodontitis we have two categories that involve localized aggressive periodontitis (LAP) and generalized aggressive periodontitis (GAP). Both forms share the following features: rapid loss of attachment and bone, patients are clinically healthy except for the presence of periodontitis, and familial aggregation. LAP patients have a pronounced serum antibody response, whereas GAP patients have a poor antibody response to the infecting agents; and (9) NUP involves a great deal of alveolar bone and attachment loss, whereas in NUG loss of clinical attachment is generally absent.

It is our sincere hope that this paper be received by the professional community as an effort to enliven a rather mundane but necessary subject, as well as attempting to make the classification of periodontal disease a more realistic, current concept as we become aware of more medical problems that are influenced by periodontal diseases and more periodontal problems that are affected by systemic diseases. The classification of periodontal disease should be kept as simple and thorough as possible. This classification can be added to more simply than past attempts, without semantics becoming a major stumbling block. A recommendation for the next classification is that wherever possible the insertion of the periodontal codes, the ADA codes, and the International codes be included. For some reason, since the 1960's the addition of code numbers has not been attempted. This would help to facilitate insurance claims and office procedures as well as not allowing the insurance companies a back door to denying claims. Perhaps we should add a section devoted entirely to implantology and surgeries connected to improving implant procedures, since so many periodontal practices are now doing 80% implantology and 20% pure periodontics.

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