In order to obtain more knowledge about the causation of periodontal diseases the various forms of the disease have to be classified. In the past 130 years, various classification systems for periodontal diseases based on the understanding of the nature of these diseases at the time the classifications were proposed. 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 (1920-1970), and the infection/host response paradigm (1970-present). The changes in the paradigm have always been followed by the conceptual changes in the classification of periodontal disease. 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.

**Keywords:** Classification, Paradigm, Periodontitis

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**INTRODUCTION**

Advances in science and technology over the last century have greatly expanded our knowledge in the field of periodontology. Even though controversies arise side by side and in controversy, one often ends up where one started, they facilitate our better understanding of periodontal diseases and developing targeted interventions. The current challenge of periodontist is to disentangle the controversies existing in classification of periodontal diseases.

Since decades, there has been a debate among periodontists to classify periodontal diseases. Periodically experts are refining the existing classification to develop a new classification system for periodontal diseases.\(^1\)\(^6\) Classification of periodontal diseases helps in the:

- Development of the framework to study the etiology, pathogenesis, and treatment of diseases.
- Provides the international health care community with a way of communicating in common language.
- Helps to organize and execute treatment strategies for individual patients.

**DOMINANT PARADIGMS IN THE HISTORICAL DEVELOPMENT OF CLASSIFICATION SYSTEMS**

Paradigms that reflect the understanding of the nature of periodontal diseases during a given historical period have largely influenced the evolution of classification systems for periodontal diseases. Classification of periodontal diseases can be placed into three dominant paradigms:

1. Primarily based on the clinical features of the diseases 1870-1920
2. The concepts of classical pathology 1920-1970
3. The infectious etiology of the diseases 1970-present

Since there is a certain amount of validity to some of the primitive thoughts about the nature of periodontal diseases,\(^3\)\(^4\)\(^7\) the classification systems represent a blend of all three paradigms in the current era.

**Clinical Characteristics Paradigm (1870-1920)**

Almost all ancient works refer to the various diseases of the teeth and their supporting tissues but without using any particular terminology. The first specific name for periodontal diseases was introduced by Fauchard in 1723 using the term “scurvy of the gums.”\(^8\) From 1870 to 1920
very little was known about the etiology and pathogenesis of periodontal diseases, the diseases were classified almost entirely on the basis of their clinical characteristics.

In late 1800s and early 1900s details on classification of periodontal diseases were rare in the publications and were based on the previous subject usually represented the opinions, which were in sync with clinical observations and theory of causation. A good example was a paper published by Davis in 1879 who believed that there were three distinct forms of destructive periodontal disease:
1. Gingival recession with minimal or no inflammation
2. Periodontal destruction secondary to “lime deposits.”
3. “Riggs’ Disease” the hallmark of which was, “loss of alveolus without loss of gum.”

Similarly, in 1886 Black published his thoughts by classifying periodontal diseases into five separate groups:
1. Constitutional gingivitis including mercurial gingivitis, potassium iodide gingivitis and scurvy.
2. A painful form of gingivitis: A clinical condition that resembled what is now termed necrotizing ulcerative gingivitis (NUG), but he never used the term.
3. Simple gingivitis: This was associated with the accumulation of debris that eventually led to “calci inflammation of the peridental membrane.”
4. Calci 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.
5. Phagedenicpericementitis: This condition shared many features with “calci 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 “phagedenicpericementitis” with “chronic suppurative pericementitis.”

What emerged from this debate was the concept that there were at least two forms of destructive periodontal disease inflammatory and non-inflammatory (“degenerative” or “dystrophic”).

Gottlieb is generally considered to be the first author who clearly distinguished various forms of periodontal diseases, he postulated that certain forms of destructive periodontal disease were due to degenerative changes in the periodontium. Study carried out by some authors hypothesized that in periodontal diseases there was a degenerative transformation of alveolar bone into fibrous connective tissue. As a result, almost all classification systems used from 1920-1970 included disease categories labeled as “dystrophic,” “atrophic,” or “degenerative.”

In the latter part of the 19th century periodontitis went under numerous names including: “calci inflammation of the peridental membrane,” “chronic suppurative pericementitis,” “pyorrhea alveolaris” and “Riggs disease.” The point of these historical examples is to accentuate that little or no scientific evidence was used to support the opinions of the clinicians of the time.

Classical Pathology Paradigm (1920-1970)
During the first half of the 20th century, many clinical scholars began to develop and argue about the nomenclature and classification systems for periodontal diseases. What emerged from this debate was the concept that there were at least two forms of destructive periodontal disease inflammatory and non-inflammatory (“degenerative” or “dystrophic”).

Gottlieb, 1928
1. Inflammatory
   Schmutz pyorrhea (poor oral hygiene)
2. Degenerative or atrophic
   Diffuse alveolar atrophy
   Paradental pyorrhea

Weski, 1937
1. Paradentitis (gingivitis)
   Hypertrophic
   Simple
   Ulcerative
2. Paradentosis
   Partial atrophic
   Total atrophic
   Paradentoma
   Localized form-epulis
   Generalized form
   Elephantiasis gingivae

Orban, 1942
1. Inflammation
   I. Gingivitis (little or no pocket formation; can include ulcerative from-Vincent’s)
      A. Local (calculus, food impaction, irritating 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.

2. Degeneration
I. Periodontosis (as a rule attacks young girls and older men; often carries immunity)
A. Systemic disturbances
   • Diabetes
   • Endocrine dysfunctions
   • Blood dyscrasias
   • Nutritional disturbances
   • Nervous disorders
   • Infectious diseases (acute and chronic)
B. Hereditary
C. Idiopathic

3. Atrophy
I. Periodontal atrophy (Recession, no inflammation, no pockets; osteoporosis)
A. Local trauma
B. Presenile
C. Senile
D. Disuse
E. Following inflammation
F. Idiopathic

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

5. Traumatism
I. Periodontal traumatism
A. Occlusal trauma

Orban, 1949

1. Inflammatory conditions
I. Gingivitis
A. Acute or chronic according to duration
B. Ulcerative, purulent, etc., according to symptoms
C. Local or systemic according to etiology
   Local (Extrinsic)
   Infectious
   Physical
   Chemical
   Systemic (Intrinsic)
   Dietary deficiency
   Endocrine disturbances
II. Periodontitis
A. Simplex-following gingivitis
B. Complex-following periodontitis

2. Degenerative conditions
I. Gingivosis-systemic etiology
   Degeneration of connective tissue
II. Periodontosis
   Early-no inflammation
   Late-deep pockets with periodontitis
III. Atrophic conditions
   Periodontal atrophy-bone recession
IV. Periodontal traumatism
   Primary - overstress, bruxism, etc.
   Secondary - loss of supporting tissue.
V. Gingival hyperplasia
   Infectious - pyogenic granuloma
   Endocrine dysfunction - pregnancy
   Drugs - dilantin
   Idiopathic

American Academy of Periodontology (AAP), 1957

Inflammation
Gingivitis
Periodontitis

Dystrophy
Occlusal traumatism
Periodontal disuse atrophy

Gingivosis
Periodontosis

Most of the classification systems published between 1920 and 1970 included a degenerative disease category. But serious questions were raised about the existence of “periodontosis” as a distinct disease entity. Many recommended that this term should be discarded. It was not until 1977, that considerable arguments were provided that there was no scientific basis for retaining the concept that there were dystrophic or degenerative forms of destructive periodontal disease. Information potted at that meeting supported the conclusion that “periodontosis” was actually an infection and “juvenile periodontitis” should become the ideal term for this group of diseases.

It was around 1970 that a different paradigm had begun to dominate thoughts about the nature of periodontal diseases.

Infection/Host Response Paradigm (1970-Present)

After the 1876 publication of Koch, in which he provided experimental proof of the germ theory of diseases, some dentists suggested that periodontal diseases were caused by bacteria. Miller was an early proponent of the infectious nature of periodontal diseases. Miller also recognized that certain systemic conditions could modify the course of the disease. It was not until the classical “experimental gingivitis” studies published by Loe et al. from 1965 to 1968 that the infection/host response paradigm began to move in the direction of becoming the dominant paradigm.
In 1976-1977, the next major finding was the preliminary demonstration of microbial specificity at sites with periodontosis.\textsuperscript{30,31} This finding, coupled with the demonstration that neutrophils from patients with juvenile periodontitis had defective chemotactic and phagocytic activities,\textsuperscript{32-34} marked the beginning of the Infection/Host Response paradigm.

\textbf{Prichard, 1972}\textsuperscript{35}
\textit{Diseases affecting the surface or gingiva}
Inflammation without surface destruction
- Marginal gingivitis
- Generalized diffuse gingivitis
- Gingival enlargement

Inflammation with surface destruction
- NUGs
- Herpetic gingivostomatitis
- Desquamative gingivitis
- Oral ulcers

\textbf{Diseases that affect the deeper structures}
Chronic destructive periodontal disease or periodontitis
- Periodontal abscess
- Periodontal traumatism
- Primary traumatism
- Secondary traumatism

\textbf{AAP, 1977}\textsuperscript{36}
Juvenile periodontitis
Chronic marginal periodontitis

\textbf{Grant et al., 1979}\textsuperscript{35}
1. Inflammatory
   - Gingivitis
   - Periodontitis
   - Juvenile periodontitis
2. Traumatic/degenerative
   - Periodontal trauma
   - Gingival recession
   - Alveolar atrophy
3. Systemic/genetic/immunologic
   - Hereditary gingival fibromatosis
   - Chediak-Higashi syndrome
   - Down syndrome
   - Hypophosphotasia
   - Cyclic neutropenia
   - Lazy leukocyte syndrome
   - Diabetes mellitus
   - Juvenile periodontitis
   - Hyperkeratosis palmaris et plantaris

\textbf{Ramfjord and Ash, 1979}\textsuperscript{37}
1. Gingivitis
   a. Simplex
   b. Complex
      - Gingival hyperplasia
      - Necrotizing lesions
   c. Traumatic
2. Gingival atrophy or recession
   - Systemic factors
   - Local causes
3. Trauma from occlusion
4. Periodontitis
   a. Simple
   b. Complex
      - Juvenile, etc.

\textbf{Page and Schroeder, 1982}\textsuperscript{38}
Prepubertal
- Generalized
- Localized

Juvenile
Rapidly progressing periodontitis
- “Adult” type periodontitis

\textbf{AAP, 1986}\textsuperscript{3}
- Juvenile periodontitis
  - Prepubertal
    - Localized juvenile periodontitis
    - Generalized juvenile periodontitis
- Adult periodontitis
- Necrotizing ulcerative gingivo-periodontitis
- Refractory periodontitis

\textbf{Grant et al., 1988}\textsuperscript{38}
Bacterially induced diseases
- Gingivitis
- Periodontitis
  - Adult type
  - Post juvenile
  - Early onset
    - Juvenile
    - Localized
    - Generalized
- Acute NUGs
- Acute abscess
- Pericorinitis

Functionally induced diseases
- Traumatic occlusion
- Disuse atrophy
- Trauma
- Habits, accidents

\textbf{Suzuki, 1988}\textsuperscript{39}
Adult periodontitis
Rapidly progressive periodontitis
Juvenile periodontitis
The next major landmark in the classification of periodontal diseases emerged from 1989 world workshop in clinical periodontics where a new classification based on the infection/host response pattern was proposed. But it was stated that “although the AAP classification was adopted, legitimizing the idea that different forms of periodontal diseases exists, more recently acquired data mandate modification and revisions.”


I. Adult periodontitis
II. Early onset periodontitis
   a. Prepubertal periodontitis
      i. Generalized
      ii. Localized
   b. Juvenile periodontitis
      i. Generalized
      ii. Localized
   c. Rapidly progressive periodontitis
III. Periodontitis associated with systemic disease
    Down Syndrome
    Diabetes type I
    Papillon-Lefevre Syndrome
    AIDS
    Other diseases
IV. Necrotizing ulcerative periodontitis (NUP)
V. Refractory periodontitis

The disease category of “Prepubertal Periodontitis” was the first to be seriously questioned. The uncertainty about the proposal that “Rapidly Progressive Periodontitis” was a single entity, and secondly, the questionable criteria used to determine its presence.

This classification, depended heavily on the age of the affected patients and the rates of progression. Important features of periodontitis could be significantly modified by host factors and still other forms did not appear to respond well to conventional therapy (i.e. the “Refractory Periodontitis” category).

As a result of these problems, the 1989 classification was criticized shortly after it was published and a different system was proposed by Ranney. He suggested elimination of the “Refractory Periodontitis” category since it was a heterogeneous group. 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. Its acceptance was aided by the ease with which patients could be placed into age-based categories.

Ranney (1993)

Gingivitis
1. Gingivitis, plaque bacterial
   Non-aggravated
   Systemically aggravated by sex hormones, drugs, systemic disease
2. NUGs
   Systemic determinants unknown
   Related to HIV
3. Gingivitis, non-plaque
   Associated with skin disease; allergic; infectious

Periodontitis
1. Adult periodontitis
   Non-aggravated
   Systemically aggravated (neutropenias, leukemias, lazy leukocyte syndrome, AIDS, Crohn’s disease, diabetes mellitus, Addison’s disease)
2. Early-onset periodontitis
   a. Localized early-onset periodontitis
      Neutrophil abnormality
   b. Generalized early-onset periodontitis
      Neutrophil abnormality - immunodeficient
   c. Early-onset periodontitis related to systemic disease
      Leukocyte adhesion deficiency, hypophosphatasia, Papillon-Lefevre syndrome, neutropenias, leukemias, Chediak-Higashi syndrome, AIDS, diabetes mellitus type, trisomy 21, histiocytosis X, Ehlers-Danlos syndrome (Type VIII)
   d. Early-onset periodontitis, systemic determinants unknown
3. NUP
   Systemic determinants unknown
   Related to HIV
   Related to nutrition
4. Periodontal abscess

1999 CLASSIFICATION OF PERIODONTAL DISEASES AND CONDITIONS

The 1989 classification led many investigators to call for a revision of the currently used system due to problems, inconsistencies, and deficiencies associated with it. This resulted in a 1999 international workshop on the classification of periodontal diseases. One of the goals of this workshop was to correct five major problems associated with the 1989 system:

- It did not include a gingivitis or gingival disease category.
- The periodontitis categories had non-validated age-dependent criteria.
- There was extensive crossover in rates of progression of the different categories of periodontitis. ‘Rapidly progressive periodontitis’ was a heterogeneous category.
• There was extensive overlap in the clinical characteristics of the different categories of periodontitis.
• ‘Refractory periodontitis’ and ‘Prepubetal periodontitis’ was a heterogeneous category.

So new classification system was proposed by the “1999 International Workshop for a Classification of Periodontal Diseases and Conditions” to correct some of the shortcoming associated with the previous system that had been in use since 1989.

In reality, the changes could be characterized as a “fine-tuning” of the 1989 classification since no immense alterations were made. A badly gingival disease category was added. In addition, the heterogeneous disease categories of prepubertal, refractory and rapidly progressive periodontitis were eliminated. The “refractory” designation remains in the new classification, but not as a single entity.

In addition, the troublesome criteria of age and rate of progression were removed as a basis for classifying periodontitis. Changing the names of “Adult Periodontitis” to “Chronic Periodontitis” and “Juvenile Periodontitis” to “Aggressive Periodontitis” were specifically made to eliminate the non-validated age-dependent designations. In addition, elimination of the “Prepubertal Periodontitis” category was important since existing data do not support the notion that it is a single entity.

1999 INTERNATIONAL WORKSHOP CLASSIFICATION

I. Gingival disease
   A. Dental plaque-induced gingival diseases
      1. Gingivitis associated with dental plaque only
         i. Without other local contributing factors
         ii. With local contributing factors
      2. Gingival diseases modified by systemic factors
         i. 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
         ii. Associated with blood dyscrasias
            1. Leukemia-associated gingivitis
            2. Other
      3. Gingival diseases modified by medications
         i. 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
         i. Ascorbic acid-deficiency gingivitis
         ii. Other
   B. Non-plaque induced gingival lesions
      1. Gingival diseases of specific bacterial origin
         i. Neisseria gonorrhoea-associated lesions
         ii. Treponema pallidum-associated lesions
         iii. Streptococcal species-associated lesions
         iv. Other
      2. Gingival diseases of viral origin
         i. Herpes virus infections
            Primary herpetic gingivostomatitis
            Recurrent oral herpess
            Varicella-zoster infections
         ii. Other
      3. Gingival diseases of fungal origin
         i. Candida-species infections
            Generalized gingival candidiasis
         ii. Linear gingival erythema
         iii. Histoplasmosis
         iv. Other
      4. Gingival lesions of genetic origin
         i. Hereditary gingival fibromatosis
         ii. Other
   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-Lefevre syndrome
   5. Chediak-Higashi syndrome
   6. Histiocytosis syndrome
   7. Glycogen storage disease
   8. Infantile genetic agranulocytosis
   9. Cohen syndrome
  10. Ehlers-Danlos syndrome (Types IV and AD)
  11. Hypophosphatasia
  12. Other
C. Not otherwise specific
V. Necrotizing periodontal diseases:
A. NUGs
B. NUP
V. Abscesses of the periodontium:
A. Gingival abscess
B. Periodontal abscess
C. Pericoronal abscess
VI. Periodontitis associated with endodontic lesions:
A. Combined periodontal-endodontic lesions
VII. 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
   6. Abnormal color
C. Mucogingival deformities and conditions on edentulous ridges
   1. Vertical and/or horizontal ridge deficiency
   2. Lack of gingival/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

The AAP system is far from perfect and should change as soon as enough data are collected by periodontal research to answer many controversies still existing today.

**FUTURE CHALLENGES IN THE CLASSIFICATION OF PERIODONTAL DISEASES**

With our increasing understanding of the bacteria associated with periodontal infections and the genetic factors controlling host responses to these infections are entering the postgenomic era, apparently a more systematic or etiological classification could be devised.

**CONCLUSION**

Though we have made great strides towards the understanding of periodontitis in general, the bitter truth is that we have not hit the target on the true nature of etiopathogenesis. All periodontists are convinced that most periodontal diseases are infections; it is unlikely that the Infection/Host Response paradigm will be replaced. Until then, all classification systems will continue to create a dilemma in which choice will need to be made between equally unsatisfactory alternatives.

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