

Diagnosis and classification of periodontal disease

J Highfield*

**Discipline of Periodontics, Faculty of Dentistry, The University of Sydney, New South Wales.*

ABSTRACT

Periodontal diseases have been recognized and treated for at least 5000 years. Clinicians have recognized for many years that there are apparent differences in the presentation of periodontal diseases and have attempted to classify these diseases. Systems of classifications of disease have arisen allowing clinicians to develop structures which can be used to identify diseases in relation to aetiology, pathogenesis and treatment. It allows us to organize effective treatment of our patients' diseases. Once a disease has been diagnosed and classified, the aetiology of the condition and appropriate evidence-based treatment is suggested to the clinician. Common systems of classification also allow effective communication between health care professionals using a common language. Early attempts at classification were made on the basis of the clinical characteristics of the diseases or on theories of their aetiology. These attempts were unsupported by any evidence base. As scientific knowledge expanded, conventional pathology formed the basis of classification. More recently, this has been followed by systems of classification based upon our knowledge of the various periodontal infections and the host response to them. Classification of periodontal diseases has, however, proved problematic. Over much of the last century clinicians and researchers have grappled with the problem and have assembled periodically to review or develop the classification of the various forms of periodontal disease as research has expanded our knowledge of these diseases. This has resulted in frequent revisions and changes. A classification, however, should not be regarded as a permanent structure. It must be adaptable to change and evolve with the development of new knowledge. It is expected that systems of classification will change over time. This review examines the past and present classifications of the periodontal diseases.

Keywords: Diagnosis, classification, periodontal diseases, gingivitis, periodontitis.

Abbreviations and acronyms: AST = aspartate aminotransferase; CAL = clinical loss of attachment; GCF = gingival crevicular fluid; LJP = localized juvenile periodontitis; NUG = necrotizing ulcerative gingivitis; NUP = necrotizing ulcerative periodontitis.

INTRODUCTION

Periodontal disease is a disease, or more likely a number of diseases of the periodontal tissues that results in attachment loss and destruction of alveolar bone. The natural history of periodontal disease, in some but not all patients, results in tooth loss.¹ Periodontal disease, however, encompasses a wider spectrum of diseases than just periodontitis and the recognition of these diseases requires a diagnosis be made.

Diagnosis is the recognition of the presence of a disease.² Clinical diagnosis of periodontal disease is made by the recognition of various signs and symptoms in the periodontal tissues which herald a departure from health. The diagnosis of periodontal disease demands a firm knowledge of what constitutes periodontal health. The healthy periodontium,³ of which only the gingival tissues may be directly observed, is described as being stippled, pale pink or coral pink, in

the Caucasian (Fig 1), with various degrees of pigmentation in other races. It is tightly adapted to the underlying tissues, with a knife edge margin where it abuts the tooth. The gingival margin is located, in the absence of pathology, at the cemento-enamel junction. It displays a scalloped edge configuration highest interdentally, where it constitutes the interdental papilla and lowest buccally and lingually. There is a gingival crevice where it abuts the tooth which in health is 1–3 mm deep. There is an absence of bleeding from the crevice on gentle probing. The crevice in health will show a small amount of interstitial fluid, gingival crevicular fluid.⁴ The lateral wall of the crevice constitutes the free gingival margin. From the most apical extent of the free gingival to the mucogingival junction is the attached gingiva which varies in width from 1 to 9 mm and has a stippled surface. It is an immobile tissue tightly bound down to the bone as a mucoperiosteum and is a keratinized mucosa well suited to resist injury. Apical from the mucogingival junction



Fig 1. Healthy gingiva. Pale pink, stippled, knife edge margin and tightly adapted.

and continuous with the lining mucosa of the mouth is the alveolar mucosa, which is freely mobile and surmounted by a non-keratinized epithelium. It is generally thought that alveolar mucosa functions poorly as a marginal tissue and areas where there is lack of attached gingiva may constitute mucogingival problems. Departures from this concept of the healthy periodontium may suggest the presence of disease. The relevant changes which are diagnostic of the presence of disease will be discussed later in this paper.

Systems of classifications of disease have arisen to allow clinicians to develop a structure which can be used to identify diseases in relation to aetiology, pathogenesis and treatment. It allows us to organize effective treatment of our patient's disease. Once a disease has been diagnosed and classified, the aetiology of the condition and appropriate evidence-based treatment is suggested to the clinician. Classification systems also allow the clinicians and researchers everywhere to communicate with a common language.

The most commonly accepted systems of classification of periodontal disease have been those of the American Academy of Periodontology (AAP). Over much of the last century, the Academy has struggled to identify and classify the various forms of periodontal disease as research has expanded knowledge. This has led to frequent revisions and changes but has created some confusion. A classification, however, should not be regarded as a permanent structure. It must be adaptable to change and evolve with the development of new knowledge. It must be expected that systems of classification will change over time, however much this may be confusing and perhaps annoying to practitioners.

This article is a review of the historical and contemporary attempts to classify periodontal diseases.

Historical perspective

Recognition and treatment of periodontal disease can be traced back to antiquity. Descriptions of treatment

are found in ancient Egyptian and Chinese writings and would suggest that periodontal diseases were recognized possibly 5000 years ago.⁵ The first modern writings were by Abu I Quasim, also known as Abucussis of Cordova Spain in the 10th century.⁶ More recently, descriptions of treatment of periodontal disease were made by Pierre Fauchard⁷ who published the first dental textbook, "The Surgeon Dentist" in 1728 and John Hunter⁸ who published "The Natural History of the Human Teeth" in 1771 and "A Practical Treatise on the Diseases of the Teeth" in 1778. Although there was already a considerable body of writings on dentistry, possibly in excess of 450 treatises in existence before this one,⁹ Hunter provided the scientific foundations of modern dentistry. The presence of bacteria around the teeth was recognized by Von Leeuwenhoek^{5,10} in the 17th century. He described what he saw as "animicules" and related them by inference to disease. He was also probably the first person to recognize the protective effect on bacteria of the properties of biofilms when he described the effect of vinegar upon the animicules *in vivo* and *in vitro*. His observations were not remarked upon and the bacterial aetiology of periodontitis was not accepted until the latter part of the 19th century following the seminal work on the germ theory of disease of Pasteur, Koch and Lister.¹¹ Adolph Witzel¹² (1847–1906) appears to be the first individual to identify bacteria as the cause of periodontal disease but the first true oral microbiologist was WD Miller¹³ (1853–1907). Until then it was widely accepted that periodontal disease was largely related to systemic factors. The importance of local factors was, however, recognized by many practitioners. John W Riggs¹⁴ (1811–1885), a leading authority on the treatment of periodontal disease, and who incidentally was Mark Twain's periodontist, clearly recognized the importance of local irritants in the aetiology of periodontal disease. Although he published little he lectured extensively, if controversially, on the treatment of periodontal disease with the emphasis on the removal of local factors rather than on systemic factors. For many years, particularly in America, periodontitis was known as "Riggs' disease".

In the 19th century, little was understood about the aetiology and pathogenesis of periodontal diseases. Classification was made on the basis of their clinical characteristics or theories on their aetiology and was unsupported by any evidence base. The term "pyorrhea alveolaris" was introduced early in the 19th century to describe periodontitis and literally meant "pus oozing out of the alveolus". This suggested that the bone in periodontitis was infected. This, unfortunately, was incorrect but influenced treatment of periodontitis for many years toward the removal of infected marginal bone via flap surgery.

Early attempts at classification reflect what Armitage¹⁵ has termed the clinical characteristics paradigm which was in vogue from 1870 to 1920. He describes three major paradigms of understanding which have had a major influence on our attempts at classifying periodontal diseases: the clinical characteristics paradigm, the classic pathology paradigm and the infection/host response paradigm. From 1920 to 1970 the major influence was the classic pathology paradigm, and from 1970 to the present day the infection/host response paradigm.

It had long been recognized clinically that there appeared to be different types of periodontal disease and various attempts were made to define them. Gottlieb in 1921 attempted to split up the broad field of pyorrhea alveolaris into schmutz pyorrhea or filth pyorrhea, paradontal pyorrhea where there is deep seated disease within the gingival crevice that hygiene measures do not alleviate, diffuse atrophy of the alveolar bone and accelerated eruption.¹⁶ Fish¹⁷ described pyorrhea simplex where there is gradual equal deepening of the sulcus and pyorrhea profunda where isolated deep pockets existed with little general deepening of the sulcus around most teeth. Stillman and McCall¹⁸ argued for the terms, gingivitis, ulatrophia, alveoloclasia, and pericementoclasia for disease processes attacking primarily the gingival tissues, the pericementum or the alveolar bone. Box¹⁹ divided chronic periodontitis into complex and simplex, and forcefully ascribed a prominent role for occlusal trauma in the aetiology of complex and, interestingly, cites in support of his theory an article published in the *Dental Science Journal of Australia* in 1926 by R Morse Withycombe, the first periodontist to practise in Sydney, Australia.

Almost every periodontist of note seemed to have his individual terminology. There was little agreement or coordination until 1942 when Orban²⁰ proposed a classification scheme based on the principles of basic pathology. This was accepted by the American Academy of Periodontology (AAP) and gained wide acceptance. Periodontal disease was classified into broad groups: inflammatory, dystrophic and traumatic disturbances. Periodontitis was classified into simplex and complex. This was an attempt to classify the differences in the presentation of periodontitis seen clinically. Simplex was secondary to gingivitis and characterized by bone loss, pockets, abscess formation and calculus deposits. Complex was secondary to periodontosis, which he considered a degenerative disease, having similar aetiological factors to periodontitis and little or no calculus.

The terms simplex and complex gained fairly wide acceptance. Definitions, however, varied. MacPhee and Cowley²¹ defined simplex as being characterized by pocket formation of regular depth throughout the

mouth and a pattern of horizontal bone loss. Complex was characterized by advanced tissue destruction relative to the age of the patient, pocket formation of irregular depth around the mouth and irregular vertical bone loss. Complex also inferred that the disease was not a simple response to local irritants but suggested a co-factor in the aetiology, such as systemic factors or local co-factors such as occlusal trauma. Many texts, however, continued to classify inflammatory periodontal disease simply as gingivitis and periodontitis.^{22,23}

The AAP further addressed the issue of classification in the 1966 World Workshop in Periodontics.²⁴ The term chronic marginal periodontitis was accepted but the workshop failed to produce a definite system of classification for periodontitis. No agreement could be reached on the existence of periodontosis as a separate disease and the suggestion of Loe that periodontosis be called periodontitis complex was not supported. Emslie suggested further research be undertaken into periodontosis. The outcome of the workshop resulted in only one form of periodontitis, chronic marginal, being recognized. In 1977, however, the term juvenile periodontitis, which had largely replaced the term periodontosis, was accepted by the AAP. The Academy then recognized two distinct forms of periodontitis.

From 1970 to the present, Armitage¹⁵ stated that the infection/host response paradigm has been dominant. This has led to the development of the concept that periodontitis comprises a spectrum of related but distinct diseases that differ in aetiology, natural history progression and response to treatment. In 1982, Page and Schroeder²⁵ stated they could identify at least five distinctly different forms of periodontitis in humans. They subclassified marginal periodontitis as adult periodontitis and rapidly progressive periodontitis of which they felt there may be at least several types. They designated the forms of periodontitis as prepubertal, juvenile, rapidly progressive, adult and acute necrotizing ulcerative gingivo-periodontitis. In November 1986, the AAP adopted a new classification which embraced these groups as follows:

- I. Juvenile periodontitis
 - A. Prepubertal periodontitis
 - B. Localized juvenile periodontitis
 - C. Generalized juvenile periodontitis
- II. Adult periodontitis
- III. Necrotizing ulcerative gingivo-periodontitis
- IV. Refractory periodontitis

A further workshop convened by the AAP at Princeton in 1989²⁶ amended the classification further. This remained the generally accepted classification for the next 10 years. The main features of the revised classification were:

- 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

This classification was based upon:

- 1. Presence/absence of clinically detectable inflammation.
- 2. Extent and pattern of attachment loss.
- 3. Patient's age at onset.
- 4. Rate of progression.
- 5. Presence/absence of miscellaneous signs and symptoms, including pain, ulceration and amount of observable plaque and calculus.

There were, however, voices of dissent at the workshop regarding this process. Hubert Newman, for example, argued that all periodontal diseases could be classified along the lines of conventional pathology as had been done previously.

A similar but simplified classification focusing upon adult, early onset and necrotizing ulcerative periodontitis was produced by the First European Workshop on Periodontics in 1993.²⁷

Classification – the current situation

The 1989 classification and the simplified European classification gained widespread acceptance and use throughout the world. Over time various problems with the application of the classifications were observed and criticisms arose. As observed by Armitage,²⁸ the criticisms largely related to the emphasis on age of onset and rates of progression in the classification which was felt to be inappropriate. "Early onset" implies we have knowledge of when the disease started and "rapidly progressive" implies knowledge of the rate of progression which in many cases we do not have. It was also observed that there was often considerable overlap of disease categories, difficulty in fitting some patients into any of the categories and the classification criteria were frequently found to be unclear or inadequate. It was also observed that a gingival component to the classification was absent. A further World Workshop in Periodontics held by the AAP in 1996²⁹ did not produce a new classification. Reasons stated were "considerable overlap among disease categories, certain patients do not fit in any category and many microbiological and host response features are shared by multiple disease categories".

These concerns were further addressed and the classification was revised in 1999 when the International Workshop for Classification of Periodontal Diseases and Conditions was convened.³⁰ This resulted in the introduction of a gingival disease category. Adult periodontitis was replaced by chronic periodontitis and early onset periodontitis was replaced by aggressive periodontitis. Periodontitis associated with systemic disease was redefined as periodontitis as a manifestation of systemic disease and the new category necrotizing periodontal diseases incorporated both necrotizing gingivitis and necrotizing periodontitis. Separate categories for abscesses of the periodontium, periodontitis associated with endodontic lesions and development or acquired conditions were added. Refractory periodontitis was removed as a disease category.

The 1999 AAP Classification²⁸ (Fig 2) is encyclopaedic. It is very complete, detailed and complex and perhaps does not lend itself for use in its entirety on a daily basis by practitioners. A more convenient and simplified summary is:

- I. Gingival diseases
 - A. Plaque induced
 - B. Non-plaque induced
- II. Chronic periodontitis
 - A. Localized
 - B. Generalized
- III. Aggressive periodontitis
 - A. Localized
 - B. Generalized
- IV. Periodontitis as a manifestation of systemic disease
- V. Necrotizing periodontal diseases
- VI. Abscesses of the periodontium
- VII. Periodontitis associated with endodontic lesions
- VIII. Developmental or acquired deformities and conditions.

Gingival conditions

This is an important inclusion in the classification. Gingival lesions are classified into two broad categories. Plaque induced and non-plaque induced. Dental plaque induced lesions (gingivitis) may be purely plaque related with or without local contributing factors (Fig 3) or may be modified by systemic factors, medications (Fig 4) or by malnutrition. It should be noted that, although by definition gingivitis has been traditionally described as being associated with a periodontium where there has been no loss of attachment, it is possible for gingivitis to occur on a periodontium with a reduced attachment level which is stable and not experiencing progressive loss of attachment. Non-plaque induced gingival lesions encompass those caused by specific bacterial, fungal or viral infections, genetic origin, systemic conditions (dermatological conditions, allergic reactions), foreign

(a)

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| <p>I. Gingival Diseases</p> <p>A. Dental plaque-induced gingival diseases*</p> <ol style="list-style-type: none"> 1. Gingivitis associated with dental plaque only <ol style="list-style-type: none"> a. without other local contributing factors b. with local contributing factors (See VIII A) 2. Gingival diseases modified by systemic factors <ol style="list-style-type: none"> a. associated with the endocrine system <ol style="list-style-type: none"> 1) puberty-associated gingivitis 2) menstrual cycle-associated gingivitis 3) pregnancy-associated <ol style="list-style-type: none"> a) gingivitis b) pyogenic granuloma 4) diabetes mellitus-associated gingivitis b. associated with blood dyscrasias <ol style="list-style-type: none"> 1) leukemia-associated gingivitis 2) other 3. Gingival diseases modified by medications <ol style="list-style-type: none"> a. drug-influenced gingival diseases <ol style="list-style-type: none"> 1) drug-influenced gingival enlargements 2) drug-influenced gingivitis <ol style="list-style-type: none"> a) oral contraceptive-associated gingivitis b) other 4. Gingival diseases modified by malnutrition <ol style="list-style-type: none"> a. ascorbic acid-deficiency gingivitis b. other <p>B. Non-plaque-induced gingival lesions</p> <ol style="list-style-type: none"> 1. Gingival diseases of specific bacterial origin <ol style="list-style-type: none"> a. <i>Neisseria gonorrhea</i>-associated lesions b. <i>Treponema pallidum</i>-associated lesions c. streptococcal species-associated lesions d. other 2. Gingival diseases of viral origin <ol style="list-style-type: none"> a. herpesvirus infections <ol style="list-style-type: none"> 1) primary herpetic gingivostomatitis 2) recurrent oral herpes 3) varicella-zoster infections b. other | <ol style="list-style-type: none"> 3. Gingival diseases of fungal origin <ol style="list-style-type: none"> a. <i>Candida</i>-species infections <ol style="list-style-type: none"> 1) generalized gingival candidosis b. linear gingival erythema c. histoplasmosis d. other 4. Gingival lesions of genetic origin <ol style="list-style-type: none"> a. hereditary gingival fibromatosis b. other 5. Gingival manifestations of systemic conditions <ol style="list-style-type: none"> a. mucocutaneous disorders <ol style="list-style-type: none"> 1) lichen planus 2) pemphigoid 3) pemphigus vulgaris 4) erythema multiforme 5) lupus erythematosus 6) drug-induced 7) other b. allergic reactions <ol style="list-style-type: none"> 1) dental restorative materials <ol style="list-style-type: none"> a) mercury b) nickel c) acrylic d) other 2) reactions attributable to <ol style="list-style-type: none"> a) toothpastes/dentifrices b) mouthrinses/mouthwashes c) chewing gum additives d) foods and additives 3) other 6. Traumatic lesions (factitious, iatrogenic, accidental) <ol style="list-style-type: none"> a. chemical injury b. physical injury c. thermal injury 7. Foreign body reactions 8. Not otherwise specified (NOS) |
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(b)

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| <p>II. Chronic Periodontitis†</p> <ol style="list-style-type: none"> A. Localized B. Generalized <p>III. Aggressive Periodontitis†</p> <ol style="list-style-type: none"> A. Localized B. Generalized <p>IV. Periodontitis as a Manifestation of Systemic Diseases</p> <ol style="list-style-type: none"> A. Associated with hematological disorders <ol style="list-style-type: none"> 1. Acquired neutropenia 2. Leukemias 3. Other B. Associated with genetic disorders <ol style="list-style-type: none"> 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) <p>V. Necrotizing Periodontal Diseases</p> <ol style="list-style-type: none"> A. Necrotizing ulcerative gingivitis (NUG) B. Necrotizing ulcerative periodontitis (NUP) <p>VI. Abscesses of the Periodontium</p> <ol style="list-style-type: none"> A. Gingival abscess B. Periodontal abscess C. Pericoronal abscess | <p>VII. Periodontitis Associated With Endodontic Lesions</p> <ol style="list-style-type: none"> A. Combined periodontic-endodontic lesions <p>VIII. Developmental or Acquired Deformities and Conditions</p> <ol style="list-style-type: none"> A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis <ol style="list-style-type: none"> 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 <ol style="list-style-type: none"> 1. Gingival/soft tissue recession <ol style="list-style-type: none"> 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 <ol style="list-style-type: none"> 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 <ol style="list-style-type: none"> 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 <ol style="list-style-type: none"> 1. Primary occlusal trauma 2. Secondary occlusal trauma |
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Fig 2. Classification of periodontal diseases and conditions. The American Academy of Periodontology. (Reproduced with permission from the American Academy of Periodontology). *Can occur on a periodontium with no attachment loss or on a periodontium with attachment loss that is not progressing. †Can be further classified on the basis of extent and severity. As a general guide, extent can be characterized as localized = \leq of sites involved and generalized = $>$ 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 = \geq mm CAL.



Fig 3. Gingivitis. (a) Marginal gingivitis with erythema, oedema and loss of stippling. Note the composite veneers on the maxillary central incisors. (b) Severe oedematous gingivitis with marked erythema and oedema spreading over the adjacent attached gingiva.

body reactions, trauma lesions and a catch all, not otherwise specified, for forms of gingivitis that do not fit neatly into any of the other areas. Figure 5 illustrates a dermatological disease, lichen planus which is common, may occur in the mouth without skin lesions and is frequently confused and misdiagnosed as plaque induced gingivitis. Note the severe erythema of the attached gingiva and the presence of cheek lesions and an ulcer opposite the second molar.

Chronic periodontitis

“Chronic periodontitis”, in the 1999 classification, has replaced the term “adult periodontitis”. It was felt that adult periodontitis was an inappropriate term as, although it was the most common form of periodontitis in adults, it could also be seen in adolescents and occasionally children. The term “chronic” was chosen as it was felt to be non-specific and not age dependant and thus less restrictive. It may be either localized or generalized, depending upon the number of sites affected. Localized periodontitis is described as 30 per cent or less of sites affected and generalized periodontitis being more than 30 per cent of sites affected.

Features of chronic periodontitis listed in the 1999 International Workshop³¹ are:

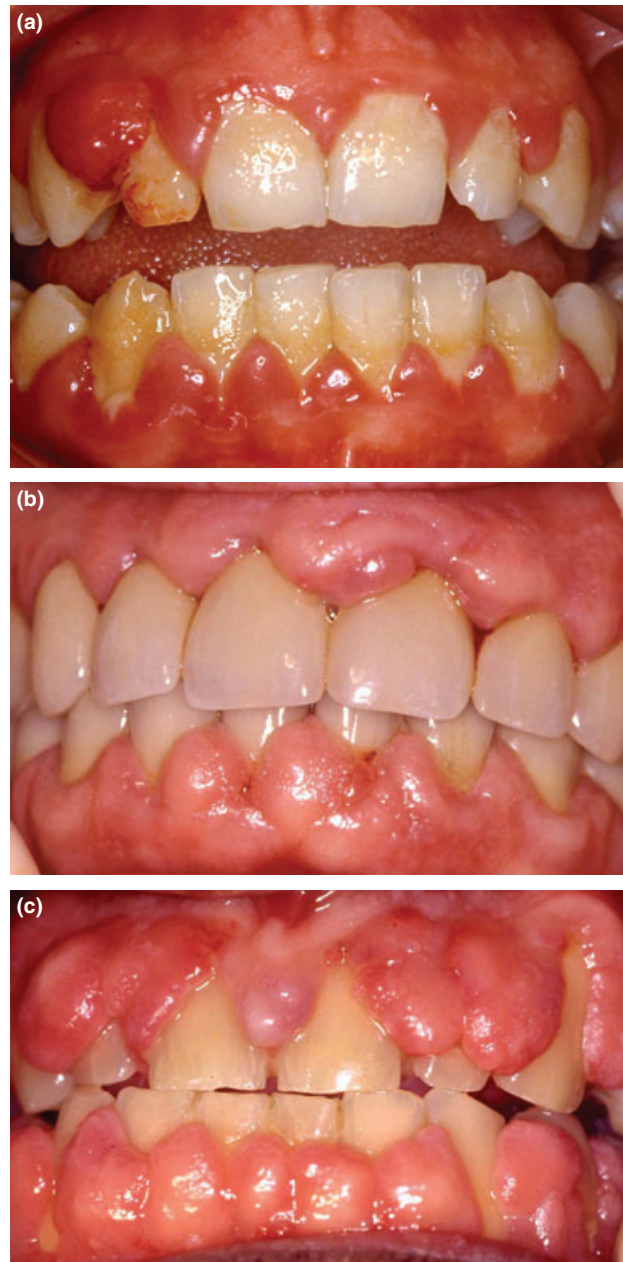


Fig 4. Gingivitis modified by hormonal effects and medications. (a) By the hormonal effects of pregnancy. (b) and (c) By medications. (b) By an antihypertensive drug, nifedipine, which is a calcium antagonist. (c) By cyclosporine, an immunosuppressant use to prevent organ graft rejection.

- most prevalent in adults, but can occur in children and adolescents;
- amount of destruction is consistent with the presence of local factors;
- subgingival calculus is a frequent finding;
- associated with a variable microbial pattern;
- slow to moderate rate of progression, but may have periods of rapid progression;
- can be further classified on the basis of extent and severity;

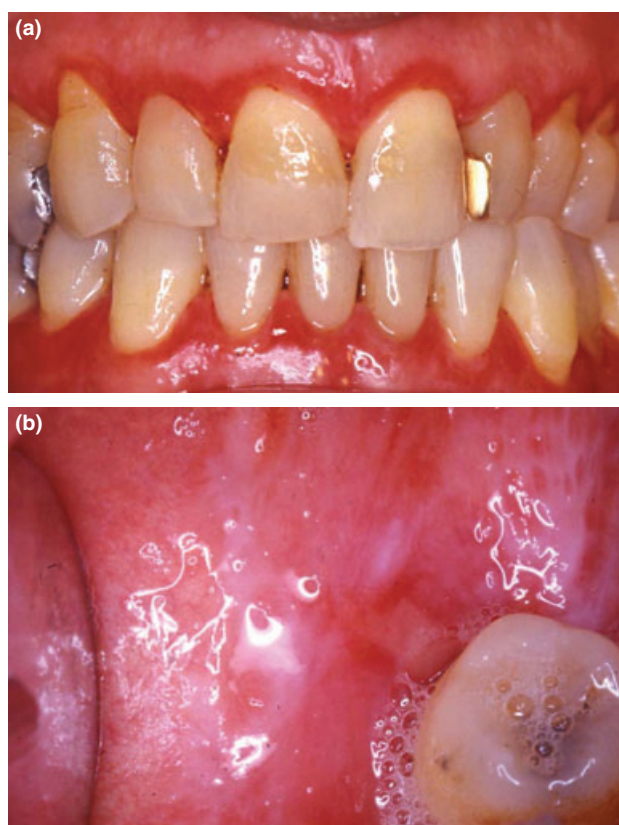


Fig 5. Oral lichen planus. (a) Note the intense erythema of the marginal and adjacent attached gingiva that has not responded to plaque control measures. (b) Shows typical cheek lesions with white plaques with an ulcerative lesion adjacent to the second molar, typical of oral lichen planus.

- can be associated with local predisposing factors (e.g., tooth-related or iatrogenic factors);
- may be modified by and/or associated with systemic diseases (e.g., diabetes mellitus, HIV infection);
- can be modified by factors other than systemic disease such as cigarette smoking and emotional stress.

The workshop also produced the following working definition and features of chronic periodontitis: “An infectious disease resulting in inflammation within the supporting structures of teeth, progressive attachment and bone loss. It is characterized by pocket formation and/or gingival recession. It is recognized as the most frequently occurring form of periodontitis. Its onset may be at any age but is most commonly detected in adults. The prevalence and severity of the disease increases with age. It may affect a variable number of teeth and has variable rates of progression.”

As a guide, severity of the disease has traditionally been characterized as being slight or early where bone loss is in the coronal third of the root, moderate where bone loss is in the middle third of the root and advanced when in the apical third of the root length. The workshop categorized a general guide for severity on the basis of clinical loss of attachment (CAL) as



Fig 6a and b. Generalized chronic periodontitis in a 55-year-old female. Note the generalized recession, plaque, calculus and anterior migration with opening of a diastema. The radiograph shows generalized advanced bone loss which is both horizontal and vertical.

follows: slight = 1–2 mm CAL; moderate = 3 to 4 mm CAL; and severe = 5 mm CAL.

Signs of inflammation are often variable depending upon the patient’s plaque control. As the disease progresses mobility and migration of teeth, which may be individual or segmental, may occur. Figure 6 shows a generalized advanced chronic periodontitis in a 55-year-old female. Note the lack of marked inflammation, presence of gingival recession, calculus and anterior migration with the opening of a diastema. The radiograph shows advanced bone loss which is both horizontal and vertical.

Aggressive periodontitis

Aggressive periodontitis replaces the category “early onset periodontitis” which in the 1989 AAP and 1993 European classifications embraced a number of diseases affecting young patients. It was felt that the use of this new term addressed the clinical characteristics of the disease while avoiding the controversial age barrier. While generally patients in this category would be under the age of 30, it is recognized that older patients may also experience periods of more rapid attachment loss. It also avoids the dilemma frequently confronted using the 1989 classification as to where to place certain patients. For example, deciding whether a young adult with generalized periodontal destruction has rapidly progressive periodontitis or generalized

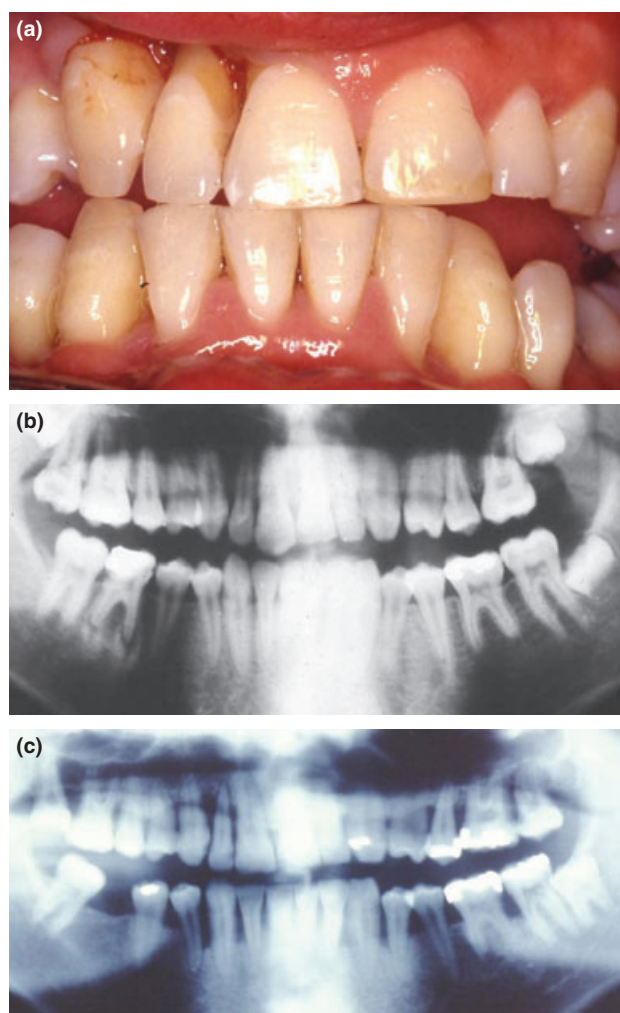


Fig 7. Rapidly progressive periodontitis (generalized aggressive periodontitis). (a) Clinical photo of male aged 21 years showing anterior gingival recession reflecting attachment loss. Inflammation is not marked as the patient had received some treatment and the disease was in remission. (b) Radiograph at age 16 showing normal bone levels. (c) Radiograph at 21 showing advanced bone destruction illustrating rapid destruction over five years.

juvenile periodontitis under the 1989 classification highlights the problem with age dependency and rates of progression. A diagnosis of generalized juvenile periodontitis, which has persisted into adulthood, requires knowledge of the time of onset. Alternatively, the condition may be a rapidly progressive periodontitis. However, making this diagnosis requires knowledge of rates of progression and time of onset which can only be gained from previous records, which in many cases may or may not be available (Fig 7). Further, is it appropriate to classify it as a juvenile periodontitis when the patient is now an adult? Should the classification be changed to something else and, if so, what? It may not fit into any of our categories. One purpose of a classification system is to provide a framework in which to undertake orderly treatment of a disease. In this instance the classification system is

contributing to confusion and is not suggesting appropriate treatment. Should we be carrying out treatment appropriate for a rapidly progressive disease or treatment for a generalized juvenile periodontitis which may no longer be rapidly progressive? Localized juvenile periodontitis has a circumpubertal onset and progresses very rapidly for a number of years then frequently goes into remission,³² becoming more generalized and, as Suzuki³³ suggests, clinically similar to adult (chronic) periodontitis. Thus, we may be treating what under the 1999 classification is a chronic periodontitis. The new classification system attempts to avoid these problems by simplifying the diagnosis. The treatment dilemma, however, may still exist with the new classification when we have imperfect knowledge of the disease history.

The workshop decided to discard all classifications that were age dependant or where knowledge of rates of progression was required. Patients previously classified as having rapidly progressive periodontitis were classified as having either generalized aggressive periodontitis or chronic periodontitis, depending upon their clinical characteristics. The term localized juvenile periodontitis was replaced by localized aggressive periodontitis, and prepubertal periodontitis was removed as a separate disease category. In most patients given the classification of generalized prepubertal periodontitis, the periodontitis was found to be a manifestation of a systemic disease. Those prepubescent children with periodontitis without any modifying systemic conditions would fit under the chronic or aggressive disease categories.

Common features of localized and generalized forms of aggressive periodontitis listed in the 1999 workshop³⁴ are:

- except for the presence of periodontitis, patients are otherwise clinically healthy;
- rapid attachment loss and bone destruction;
- familial aggregation.

Secondary features that are generally, but not universally, present are:

- amounts of microbial deposits are inconsistent with the severity of periodontal tissue destruction;
- elevated proportions of *Actinobacillus actinomycetemcomitans* (*Aggregatibacter actinomycetemcomitans*) and, in some populations, *Porphyromonas gingivalis* may be elevated;
- phagocyte abnormalities;
- hyper-responsive macrophage phenotype, including elevated levels of PGE₂ and IL-1 β ;
- progression of attachment loss and bone loss may be self arresting.

Further specific features were identified:

Localized aggressive (Fig 8)

- circumpubertal onset;
- localized first molar/incisor presentation with interproximal attachment loss on at least two permanent teeth, one of which is a first molar, and involving no

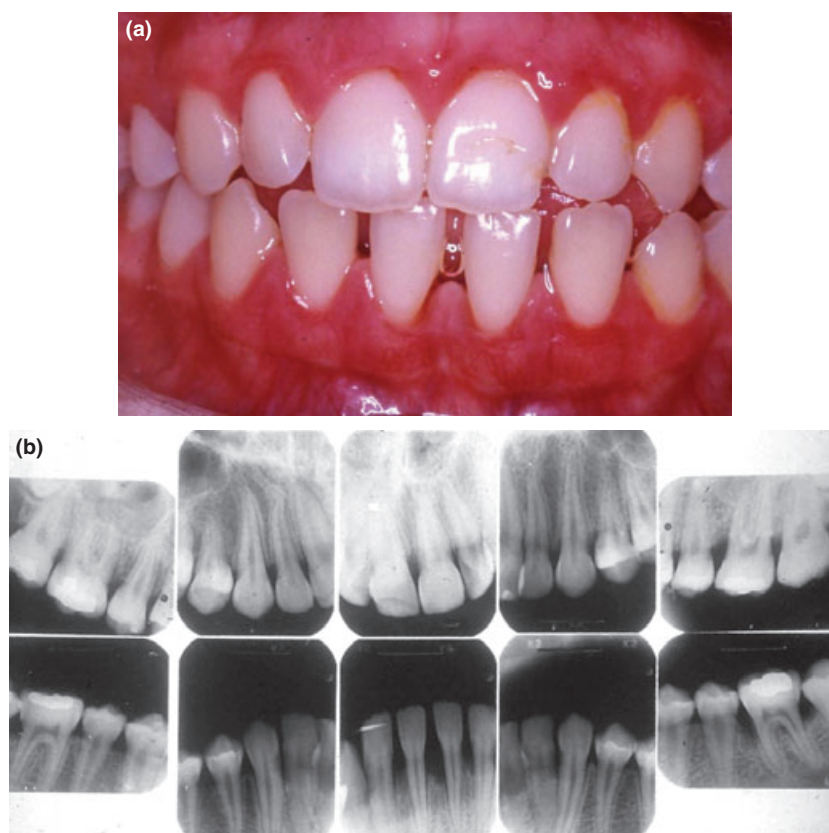


Fig 8a and b. Localized aggressive periodontitis (localized juvenile periodontitis) in a 13-year-old (circumpubertal) otherwise healthy female. The gingival tissues show marked gingivitis which is not typical; the radiographs show bone loss in the first molar and incisor teeth.

more than two teeth other than first molars and incisors;

- robust serum antibody response to infecting agents.
- Generalized aggressive (Fig 9)
- usually affecting persons under 30 years of age, but patients may be older;
 - poor serum antibody response to infecting agents;
 - pronounced episodic nature of the destruction of attachment and alveolar bone;
 - generalized interproximal attachment loss affecting at least three permanent teeth other than first molars and incisors.

The workshop noted that factors which modify risk, such as cigarette smoking, stress, drugs or sex hormones, which affect the course of all types of periodontal disease, may be added to these primary descriptors to further describe the type of disease being managed or studied.

Periodontitis as a manifestation of systemic disease

This category has been redefined to include only those diseases where the periodontal disease is a manifestation of the disease process and excludes those which act as modifiers of all types of periodontal disease. This includes various haematological disorders such as

acquired neutropenia and leukaemia; various genetic disorders such as familial and cyclic neutropenia (Fig 10), Down syndrome (Fig 11), leukocyte adhesion deficiency syndrome, Papillon-Lefèvre syndrome, Chediak-Higashi syndrome, histiocytosis syndromes, hypophosphatasia and others.³⁵ Other diseases such as HIV and diabetes (Fig 12) were considered to be modifiers of both chronic and aggressive periodontitis. It was concluded that there was insufficient evidence that there is a specific periodontitis associated with these diseases.²⁸

Necrotizing periodontal disease

The workshop recognized that necrotizing ulcerative gingivitis (NUG) and necrotizing ulcerative periodontitis (NUP) were clinically distinguishable disease entities but were unsure as to whether they were part of the same disease process or were two distinct diseases. They concluded that there was insufficient data to resolve this problem, thus they decided to place both conditions in the one category of “necrotizing periodontal disease”.³⁶ Certainly clinical observation would suggest that they are part of a continuum with initial infections perhaps showing little or no clinically recognizable attachment loss despite soft tissue destruction of the papillary

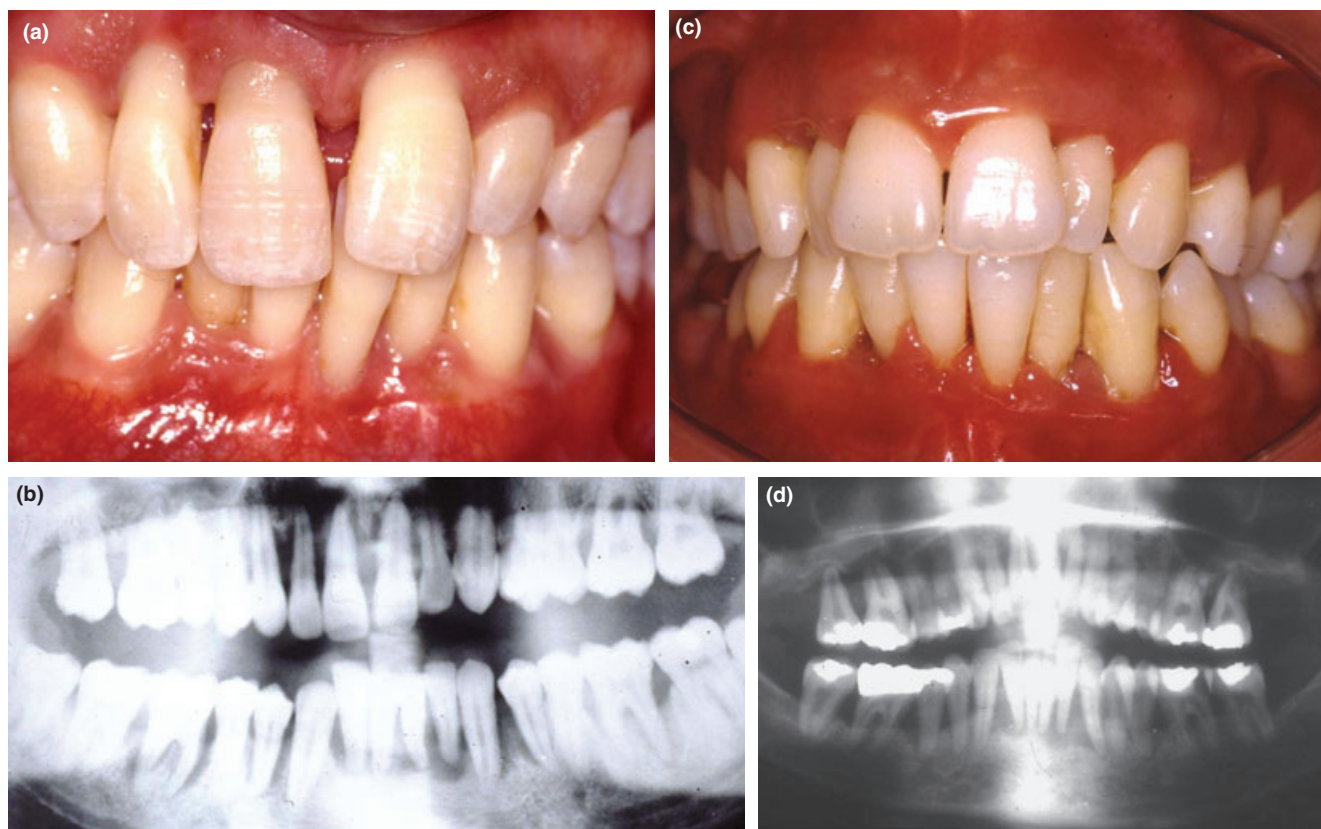


Fig 9. Aggressive periodontitis. (a) and (b) Generalized aggressive periodontitis in an otherwise healthy 21-year-old male. The clinical photograph shows some gingival inflammation with recession and anterior migration of teeth, the radiograph shows severe generalized bone destruction. The patient had no symptoms until he noticed his anterior teeth were loose. (c) and (d) A more typical generalized aggressive periodontitis in a 29-year-old female displaying very erythematous and oedematous gingiva with severe bone loss evidenced on the radiograph.

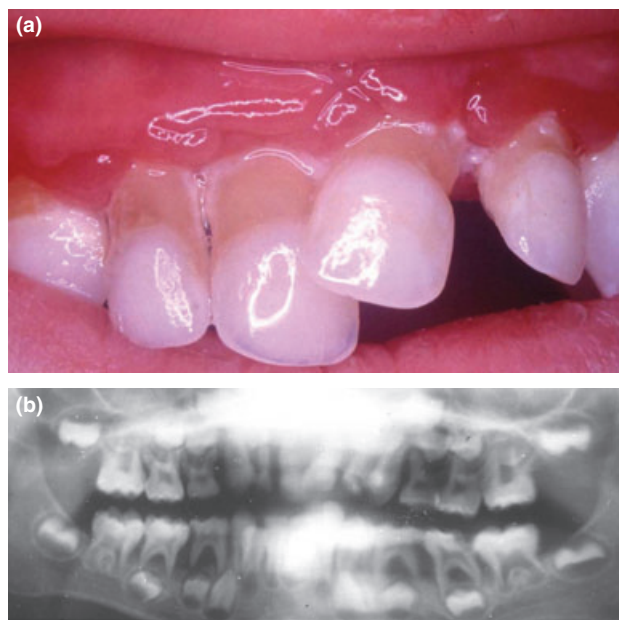


Fig 10a and b. Cyclic neutropenia. A 5-year-old boy. Note the very inflamed, erythematous and oedematous gingiva, anterior migration, the advanced premature exfoliation that is apparent and the generalized severe bone loss on the radiograph.

tissue. However, with recurrent infections, to which these patients are prone, the attachment loss becomes more apparent. Figure 13 shows necrotizing ulcerative gingivitis in a 21-year-old law student taken during his final year examinations. This patient was under stress, was smoking tobacco heavily and had a history of “bleeding gums” (pre-existing gingivitis). These are common predisposing factors for necrotizing periodontal disease. He complained of painful gingival tissues, of sudden onset. Note the marked gingival erythema and oedema, loss of papillary tissue with punched out crater ulcers and spread of the ulceration to the marginal tissue with obvious bleeding. Necrotizing ulcerative gingivitis tends to be recurrent if predisposing factors remain and progresses to necrotizing ulcerative periodontitis, in some cases causing severe destruction of the periodontal tissues (Fig 14). This condition is commonly associated also with HIV infection (Fig 12) and because of this there has been some discussion as to whether it should be included under manifestation of systemic diseases. However, it is likely that HIV acts as a predisposing factor by lowering host resistance.

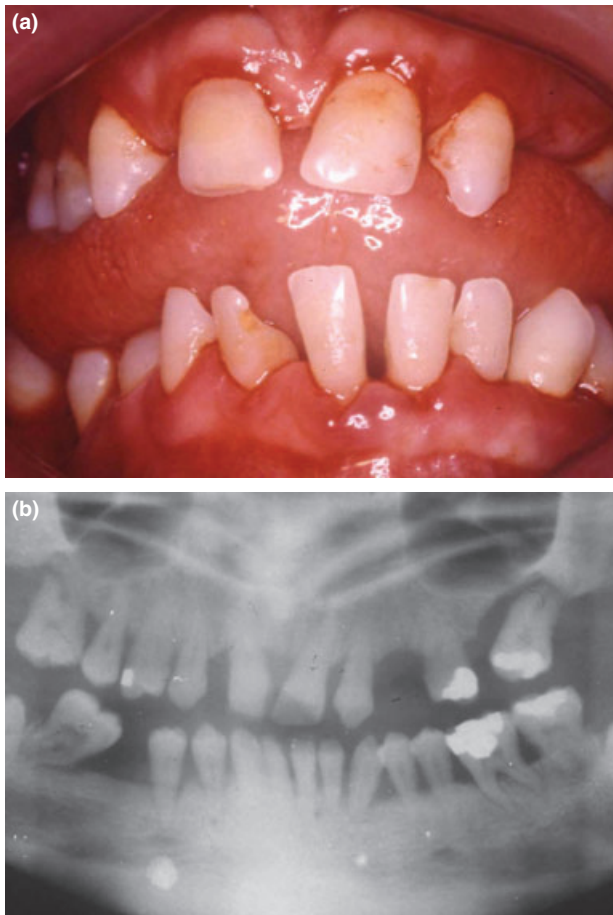


Fig 11a and b. Down syndrome. Note the gingival inflammation, splayed teeth and macroglossia. The radiograph shows severe bone loss.

Periodontal abscess

Periodontal abscess was added as a separate category. It was felt that, although periodontal abscess formation is a clinical feature of both chronic and aggressive periodontitis, it presents as a distinct clinical entity that requires specific diagnosis and treatment and thus deserves a separate classification. In most cases periodontal abscess formation reflects the acute exacerbation of a pre-existing periodontal pocket (Fig 15). It may be more correctly called an acute periodontitis. Abscesses may occur because of other causes such as foreign body impaction, trauma, including occlusal trauma causing vertical and horizontal root fractures or cemental tears. They can present a diagnostic dilemma, so perhaps a separate classification is justified.

Periodontal–endodontic lesions

The workshop included a single category, the “combined lesion” where an endodontic lesion is draining through a pre-existing periodontal pocket.

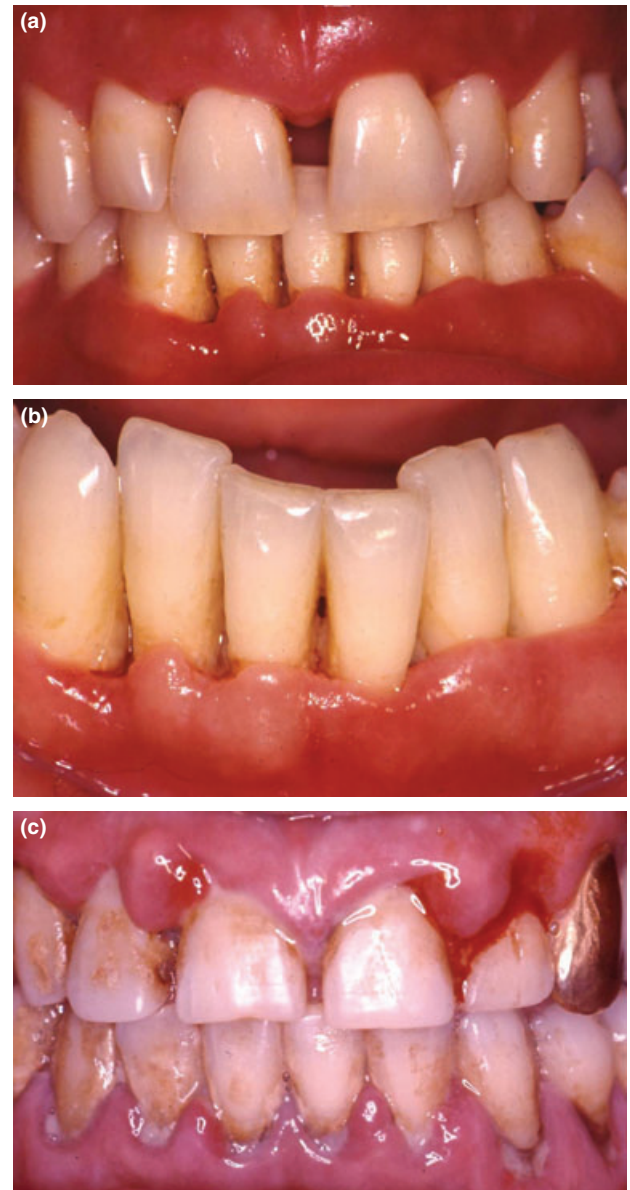


Fig 12. Periodontitis modified by systemic disease. (a) and (b) Severe necrotizing ulcerative periodontitis in a patient with HIV infection (previously classified as HIV associated periodontitis). Note the severe interproximal gingival destruction, recession and necrotizing lesions. (c) A patient with advanced periodontitis modified by poorly controlled insulin dependant diabetes.

Developmental or acquired deformities and conditions

This category appears to have been added for completeness. It includes localized tooth related factors and mucogingival conditions which may modify or predispose to disease. Many of these conditions do not constitute disease entities in their own rights but they modify and alter susceptibility to disease. Therefore, it is debatable whether they should be included in a disease classification. Occlusal trauma, primary and secondary is included. Primary occlusal trauma, where an injury to the attachment or tooth is the result of



Fig 13. Necrotizing periodontal disease. Necrotizing ulcerative gingivitis. A 21-year-old male, under stress and a smoker. Complaints of pain of sudden onset, fetor oris and gingival bleeding. Note the papillary and gingival ulcers, marked bleeding and presence of a pseudomembrane at the margin, the result of necrosis.



Fig 14. Necrotizing periodontitis. Note the tissue destruction that has occurred following recurrent episodes of infection. The disease is not self limiting and will continue if untreated with periods of remission and exacerbation.

excessive occlusal forces, is recognized as a condition. However, secondary occlusal trauma, where normal forces cause a traumatic lesion in a periodontium with reduced support is a debatable and perhaps doubtful phenomena. Conditions such as recession, excessive gingival display and ridge deformities which may require treatment for reasons other than treatment of disease, are also included and this is easier to justify.

Diagnosis

Traditionally, the diagnosis of the presence of periodontal diseases is made on the basis of evaluation of clinical signs and symptoms and may be supported by evidence from radiographs. Gingival changes including colour, contour, texture alterations and the presence of bleeding on probing from the gingival tissues allow the diagnosis of plaque induced gingival diseases. Non-plaque induced gingival diseases may necessitate other investigations such as histopathology, microbiology or



Fig 15a and b. Acute periodontal abscess. Patient complains of pain of sudden onset and gingival swelling. Note the well circumscribed swelling on the attached gingiva with points of fluctuation. The radiograph shows pre-existing periodontal marginal bone loss on the mesial of 11, indicative of a periodontal pocket and an apical rarefaction that is unrelated to the periodontal abscess but which confuses the differential diagnosis.

serology to effect a diagnosis. Periodontitis is diagnosed by the presence of gingival changes as may be evidenced for gingivitis plus the presence of reduced resistance of the tissues to periodontal probing with a deeper gingival sulcus or “pocket” which reflects loss of periodontal attachment.³⁷ It is important to recognize that “pockets” may have a horizontal as well as a vertical dimension, thus the clinician in carrying out their probing for attachment loss must be careful to

evaluate furcation involvements. The detection of attachment loss in furcations demands a sound knowledge of tooth and furcation anatomy, particularly the sites of the furcation openings on multi-rooted teeth. Tooth mobility and migration must also be assessed. It is, however, important to realize that mobility is not by itself diagnostic of periodontitis and may be the result of occlusal trauma as may be migration of teeth which may be segmental or single tooth migration. Mobility and migration solely related to periodontitis are usually late symptoms of the disease and are possibly of more importance in assessing prognosis and in treatment planning. Family history and factors which modify risk, such as cigarette smoking, stress, drugs or sex hormones, which affect the course of all types of periodontal disease need to be assessed and added to these primary descriptors to further describe the type of disease being diagnosed. Radiographs provide a secondary diagnostic tool and may demonstrate the presence of marginal bone loss, thus confirming the attachment loss. The role of radiographs in diagnosis will be addressed in another article in this supplement.⁵⁵ It is generally agreed that the healthy gingival crevice can range from 1 mm to 3 mm. In health, the distance from the cemento-enamel junction to the alveolar bone crest is also variable and has a range of 1 mm to 3 mm. It must, however, be understood that attachment loss by itself does not constitute periodontitis which is an inflammatory lesion in the periodontal tissues and that health can exist in the presence of severe attachment loss and recession. Thus, a healthy periodontium can exist at different levels along the root as happens after successful treatment.

The periodontal probe remains the primary diagnostic tool and is used to detect the presence of periodontal pockets as measured from the gingival margin to the base of the crevice and loss of attachment as measured from the cemento-enamel junction to the base of the crevice. Measurements recorded by the probe, however, are not in fact the actual pocket depth or attachment level but the distance from a fixed reference point to where the probe tip penetrates the tissues. This measurement will depend upon the probing pressure used, the tine size of the probe tip, angulation of the probe, the presence of subgingival deposits and, most importantly, the presence or absence of inflammation in the tissues. Thus, clinical attachment level and probing depth changes recorded during treatment may not reflect a true change in fibre attachment levels but merely changes in the depth of penetration of the probe into the tissues caused by change in the above factors.³⁸

Once a diagnosis of disease has been made, the disease may be classified according to the criteria of the classification system. This process of diagnosis, while it may be valid for diagnosis of periodontal disease for clinical management in dental practice presents problems when trying to determine what constitutes

periodontal disease in order to undertake clinical studies.³⁹ The problem is that our diagnosis is made upon an assessment of the destruction caused by the disease and not by an assessment of the presence of a destructive disease process within the periodontal tissues using the means usually used in assessing other diseases in medicine, such as the identification through biochemical markers, identification of responsible microbes or identification by histopathology. As Caton⁴⁰ says "periodontitis by definition is inflammation of the supporting structures of the teeth – usually a progressive destructive change leading to loss of bone and periodontal ligament. Periodontal disease activity refers to the stage of the disease characterized by loss of supporting bone and tissue attachment. This implies the natural history of periodontal disease is marked by periods of active destruction and relative quiescence, even though the periodontal tissues remain relatively inflamed". This may not appear on the surface to be of great consequence in clinical practice when confronted with a patient with obvious periodontal inflammation, attachment loss and probing depth. However, it does pose problems in recognizing the presence or absence of a destructive disease process in the tissues particularly when monitoring treatments and in designing research studies. Does a patient who has been treated with non-surgical periodontal treatment and now has a number of sites with residual probing depths that bleed on probing still have a periodontitis that requires further active therapy or surgical treatment, or is the condition stable and the disease in remission? Similarly, do patients recruited into a research study on the basis of certain criteria such as more than 30 per cent of teeth affected with pockets of ≥ 4 mm all have active periodontitis, and are the results of these studies valid and comparable? This may partly explain the confusing and often contradictory results of published research on the same topic in the literature. Problems in using different criteria for defining periodontal disease were illustrated in a recent article by Manau *et al.*,⁴¹ where the authors reanalysed their original data on the relationship between periodontitis and pregnancy using 14 different periodontitis definitions obtained from other publications. They found that the periodontal disease definition used influenced the association found between the presence of periodontitis and the association with adverse outcomes. Six of the 14 definitions of periodontitis resulted in a statistically significant association of periodontitis with adverse pregnancy outcomes while the other eight found no significant association.

Our diagnostic tools, as Preshaw²⁵ says, are "crude". He further states "we do not (yet) have the luxury of accurate, precise and reproducible indicators of periodontal disease". Much research has been done to determine accurate markers or indicators of disease.^{29,40} In addition to traditional diagnostic

procedures, assessments of inflammation and damage to the periodontal tissues, these indicators include biochemical mediators as markers, microbiological diagnostic procedures, immunological methods and genetic methods. Despite extensive research into biochemical and microbiological markers, several of which have been developed into commercial products, little of value to the clinician has evolved. One product which was marketed for a brief time used the ability of three putative periodontal pathogens, *B. gingivalis*, *B. forsythus* and *Trepenoma denticola*, to hydrolyze the synthetic trypsin substrate, N-benzoyl-DL-arginine-2-naphthylamide (BANA) forming a colour reaction to detect the presence of these putative periodontal pathogens in the subgingival plaque.⁴² Another detected the presence of aspartate aminotransferase (AST) in the gingival fluid.⁴³ AST is an enzyme present in all cells and released in abundance on cell death. In medicine it is used as a non-specific marker of cell death and routinely used as a diagnostic indicator of myocardial infarction. Gingival crevicular fluid (GCF) levels of AST have been shown to increase with inflammation and there is evidence that AST levels in GCF correlate with disease activity as assessed by probing attachment loss and with severe gingivitis.⁴⁴ The problem with the test was in distinguishing between severe gingivitis and disease activity. These tests added little to our diagnostic ability and their place in clinical practice was unclear. There was uncertainty about their ability to reliably distinguish between sites that were progressing and sites that were inflamed but not progressing. Periodontal pathogens are found in sites with and without disease activity and also in population groups resistant to periodontitis.⁴⁵ AST is found at sites with gingivitis, non-progressing and progressing lesions thus the ability of the test to discriminate is questionable.⁴⁶ Some microbiologic investigations such as DNA probes and monoclonal antibody investigations may be of use particularly in the treatment of refractory forms of periodontitis.

Therefore, despite enormous research there has been no major breakthrough in diagnosis of periodontal diseases. Although there have been major advances in our understanding of periodontal diseases, we still must rely upon our traditional diagnostic procedures, assessment of inflammation and assessment of damage to the periodontal tissues. Signs of inflammation indicate that the tissues are no longer healthy but cannot distinguish between gingivitis and periodontitis. Signs of inflammation must be evaluated in combination with evidence of attachment loss and probing depth. Inflamed periodontal tissues may exhibit some or all of the cardinal signs of inflammation, rubor (redness), tumor (swelling), calor (heat), dolor (pain) and functio laesa (loss of function), although the two last signs usually occur late in the disease process. In periodontal disease we may also diagnose bleeding on probing, suppuration, and

increased gingival fluid flow. Bleeding on probing and suppuration are generally used by clinicians as presumptive indicators of disease activity. Bleeding on probing, while a reliable indicator of gingival inflammation,⁴⁷ correlates poorly with disease activity and is a poor predictor of the progression of periodontitis.^{48,49} A good diagnostic test should have high sensitivity, the ability to detect true negatives (unlikely to be negative if someone has the disease), and high specificity, the ability to detect true positives (unlikely to be positive if someone truly does not have the disease). Sensitivity, however, often comes at the expense of specificity and vice versa. Bleeding on probing has low specificity for predicting disease activity, however sensitivity is apparently high.⁴⁰ The use of bleeding on probing as a primary diagnostic tool means that we may be treating sites which are inactive. Conversely, the absence of bleeding on probing is used by clinicians as an indicator of periodontal stability.⁵⁰ Suppuration has a high specificity for further disease progression in populations with a high prevalence of periodontitis.^{29,51} One would suspect that most practitioners would find the presence of suppuration disturbing.

Statistically significant increase in probing attachment level is the gold standard for the measurement of periodontal disease activity at a site.⁵² This can only be reliably done using histological evidence of periodontal breakdown. Clinically, it must be done by longitudinal monitoring of sites with probing measurements. Practically, this is difficult given the errors inherent in periodontal probing detailed above and the difficulty of detecting the cemento-enamel junction or some other feature as a fixed reference point. When using manual probes, changes in measurements of between 2 mm to 3 mm must occur before we can be certain that disease progression has occurred and the change is not the error in the system. Obviously, this is clinically unacceptable. As Caton⁴⁰ states "this favours specificity over sensitivity and standards may have to be lowered to reduce false negatives in patient management". Most practitioners I suspect would treat a site where lesser amounts of attachment loss or increased probing depth are detected.

CONCLUSIONS

Despite intensive research efforts to develop new technologies to improve diagnostic ability, traditional diagnostic procedures based upon clinical signs of inflammation, probing depths and clinical attachment loss still form the basis upon which periodontal diagnosis is made. Absence of gingival inflammation and shallow probing depths have a strong negative predictive values for periodontal stability.^{48,53} The clinician should strive to achieve this endpoint in the treatment of our patients.

Diagnosis of the disease also involves classification. Systems of classification are continually evolving. Classification of periodontal diseases is difficult and all classification systems produced to date have their imperfections and their critics. Van der Velden⁵⁴ argued that the 1999 reclassification was not helpful and suggested a classification based on age, which appears to be a simplified version of the 1989 AAP classification, and further classified on the basis of extent, severity and clinical characteristics. This classification allows the possibility of making an accurate clinical diagnosis for any patient with periodontitis. Milward and Chapple,⁵⁶ while recognizing the 1999 World Workshop attempted to produce a classification from an evidence-based perspective, criticized the 1999 classification as being unnecessarily complex and not suited for routine general dental practice. Lopez and Baelum⁵⁷ argue from an epidemiological point of view that there is little justification for the use of complicated classification systems and favour an approach based upon simple clinical attachment loss measurements such as ≥ 3 mm etc., a minimalist approach that does not try to differentiate between different types of periodontitis.

Why is classification of periodontal disease so difficult and so controversial? The answer lies in the heterogeneity of the clinical presentation and our lack of understanding of the true nature of the differences between the different clinical presentations of the disease. We attempt to classify using evidence based upon the different infections represented and on the host response. However, in most cases our knowledge is incomplete or confused. Much of the certitude that was felt in the 1980s that we had reached a point where we could truly distinguish between the different disease presentations in a scientific manner, has largely evaporated. It is of interest that localized juvenile periodontitis (LJP), one disease which has long been recognized as being a distinct or separate disease entity on the basis of clinical characteristics, microbiological factors and host response, the features of which are well documented, has been discarded as a distinct disease entity and has been reclassified as localized aggressive periodontitis. This apparently was done to conform to the predetermined criteria of the classification and in particular the removal of the emphasis on age. However, I have yet to see an LJP in anyone other than a juvenile and the circumpubertal age of onset is an integral part of the disease description and its deletion does not appear to have been a progressive step. We should also consider that perhaps there is just one periodontitis with different clinical expressions in different hosts.

Armitage,¹⁵ in a thoughtful article on classification, stated that "the classification system proposed by the '1999 International Workshop for a Classification of Periodontal Diseases and Conditions' has corrected some of the problems associated with the previous

system that had been in use since 1989. Nevertheless, the new system is far from perfect and will need to be modified once there are sufficient new data to justify revisions". He is of the opinion that current disease designations such as "chronic periodontitis" are constellations of polymicrobial and polygenic infections whose clinical expression is altered by important environmental and host modifying conditions.

It would seem that we are trying to classify diseases of which we do not have sufficient knowledge. The present classification of periodontitis looks surprisingly like a return to simplex and complex. Until we have greater understanding of the aetiology, the bacteria associated with different periodontal infections and the pathogenesis and genetics of periodontal diseases, it is very likely that we will see further reclassifications at regular intervals.

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Address for correspondence:
Adjunct Associate Professor John Highfield
Discipline of Periodontics
Faculty of Dentistry
The University of Sydney
2 Chalmers Street
Sydney NSW 2010
Email: johnh@dentistry.usyd.edu.au

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