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Periodontal diagnoses and classification of periodontal diseases

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What is a periodontal diagnosis?

A *periodontal diagnosis* is an important label that clinicians place on a patient's periodontal condition or disease. In the current practice of periodontics it is primarily derived from information obtained from the patient's medical and dental histories combined with findings from a thorough oral examination. The entire constellation of signs and symptoms associated with the disease or condition is taken into account before arriving at a diagnosis. In some instances additional information provided by laboratory tests is useful in the overall decision-making process. Under the best of circumstances a periodontal diagnosis is a clinician's best guess as to what condition or disease the patient has. Assignment of a diagnosis carries with it the implication that the clinician has ruled out other possible diseases that the patient might have had. Since diagnostic deductions (i.e. reasonable guesses) are made on the basis of incomplete knowledge of a patient's actual condition, it is important to realize that the assigned diagnosis might be wrong. Because there is always some uncertainty, experienced clinicians routinely develop a *differential diagnosis* that is a listing of the possible diagnoses of a patient's condition ranked from most likely to least likely. A differential diagnosis is important because it provides the clinician with other diagnostic options if the initial diagnosis subsequently proves to be wrong.

A carefully considered periodontal diagnosis is of major importance in the subsequent management of a patient's periodontal disease. An accurate diagnosis is often a first step toward development of a well-designed and appropriate treatment plan that when implemented leads to resolution of the patient's periodontal infection. An incorrect diagnosis often leads to an ill-conceived treatment approach that ultimately fails to resolve the patient's periodontal problem.

The diagnostic label captures, in a few words, a clinician's entire past experience with a disease or condition. It is a summary term that helps guide the clinician toward answering questions that are important to both the dentist and patient:

- What is the cause of the disease or condition?
- Is referral to another, more experienced, clinician appropriate?
- What will happen if the disease or condition is not treated?
- What are the treatment options?
- What is the best treatment?
- What is the expected outcome of treatment (i.e. prognosis)?
- What are the anticipated side effects of treatment?
- Will the treatment be painful?
- Will the treatment result in esthetic problems?
- How long will treatment take?
- How much will the treatment cost?

Importantly, most patients will want these questions addressed *before* treatment has started. It should be emphasized that from the perspective of many patients a ''healthy'' periodontium is one that is comfortable and free from functional and esthetic problems. Therefore, it is always a good practice to establish a diagnosis and discuss its implications with the patient prior to starting any therapeutic procedures.

Current classification system – what are possible periodontal diagnoses?

Plaque-induced periodontal diseases have traditionally been divided into three general categories: health, gingivitis, or periodontitis. In this context,

the diagnosis of *health* implies that there is an absence of plaque-induced periodontal disease. Plaque-induced *gingivitis* is the presence of gingival inflammation without loss of connective tissue attachment (7). Plaque-induced *periodontitis* is the presence of gingival inflammation at sites where there has been apical migration of the epithelial attachment onto the root surfaces accompanied by loss of connective tissue and alveolar bone (7). In most patients, increased probing depths or the formation of periodontal pockets accompany the development of periodontitis. Plaque-induced gingivitis and periodontitis are, by far, the most frequent of all forms of periodontal disease. They are not, however, the only diagnostic possibilities.

In the 1999 classification system for periodontal diseases and conditions, over 40 different gingival diseases were listed (Table 1)(10). In some of the non-plaque-induced gingival lesions, loss of attachment and destruction of alveolar bone may occur (28, 51, 57, 74, 83, 94). In addition, seven major categories of destructive periodontal diseases were listed:

- Chronic periodontitis (Fig. 1),
- Localized aggressive periodontitis (Fig. 2),
- Generalized aggressive periodontitis (Fig. 3),
- Periodontitis as a manifestation of systemic disease (Fig. 4),
- Necrotizing ulcerative gingivitis/periodontitis (Figs 5 and 6),
- Abscesses of the periodontium (Fig. 7),
- Combined periodontic–endodontic lesions (10).

Periodontitis can be a manifestation of at least 16 systemic diseases. In most of these systemic diseases there is either decreased host resistance to infections or perturbations in gingival connective tissue that increases its susceptibility to inflammation-induced degradation (Table 2).

Table 1. Partial list of possible diagnoses for gingival diseases (1999 Classification) (10)

Dental plaque-induced gingival diseases	
Plaque-induced gingivitis (no other local contributing factors)	(60)
Plaque-induced gingivitis with local contributing factors	(60)
Necrotizing ulcerative gingivitis (NUG)	(23, 80)
Puberty-associated gingivitis	(60, 93)
Menstrual cycle-associated gingivitis	(60)
Pregnancy-associated gingivitis	(54, 60)
Pregnancy-associated pyogenic granuloma	(60, 87)
Diabetes mellitus-associated gingivitis	(34, 60)
Leukemia-associated gingivitis	(60, 99)
Drug-influenced gingival enlargement	(60, 85)
Oral contraceptive-associated gingivitis	(60)
Ascorbic acid-deficiency gingivitis	(19, 60)
Non-plaque-induced gingival lesions	
Neisseria gonorrhea-associated lesions	(41, 83, 86)
Treponema pallidum-associated lesions	(41, 59, 82, 86)
Streptococcal species-associated lesions	(41, 53)
Mycobacterium tuberculosis-associated lesions	(27, 83)
Bacillary angiomatosis	(57)
Primary herpetic gingivostomatitis	(40, 83)
Recurrent oral herpes	(41, 83)
Varicella-zoster infections	(41, 83)
Generalized gingival candidosis	(41, 83)
Linear gingival erythema	(32)
Histoplasmosis	(28, 83)
Hereditary gingival fibromatosis	(33, 42)
Gingival manifestations of	
Lichen planus	(41, 46, 82)
Mucous membrane pemphigoid	(24, 41, 82)
Pemphigus vulgaris	(41, 66, 82)
Erythema multiforme	(14, 41, 58, 82)
Lupus erythematosus	(41, 82)
Linear IgA disease	(20, 46, 82)
Wegener's granulomatosis	(44, 46)
Psoriasis	(100)
Allergic reactions of the gingiva to	
Restorative materials (e.g. mercury, nickel, acrylic)	(41)
Toothpastes	(25)
Mouthrinses	(41)
Chewing gum additives	(49)
Foods and food additives	(41, 84)
Traumatic lesions of the gingiva	
Chemical injury	(4, 48, 67)
Physical injury	(51, 74, 94)
Thermal injury	(41)

Three basic diagnostic questions – components of a periodontal diagnosis

Before arriving at a periodontal diagnosis the clinician must answer three basic questions:

1 What periodontal disease or condition does the patient have?
2 How severe is the problem?
3 Is the disease or condition localized or generalized?

The first of these questions is the most difficult since it requires an assimilation and understanding of all of the information collected during the history-taking process and the clinical examination. In unusual cases where something other than plaque-induced periodontal disease is suspected, laboratory tests, histopathologic examination of biopsy specimens, and a medical consultation may be required

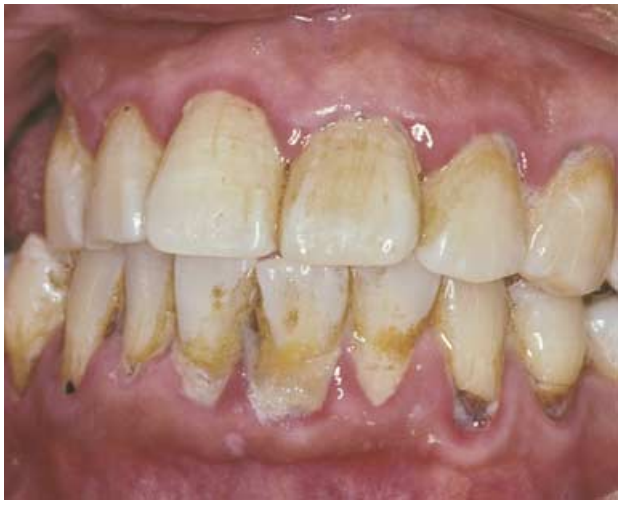

Fig. 1. Chronic periodontitis associated with poor oral hygiene in a medically healthy 45-year-old Japanese male. Inflammation, gingival recession, and attachment loss are most obvious in the mandibular anterior area.

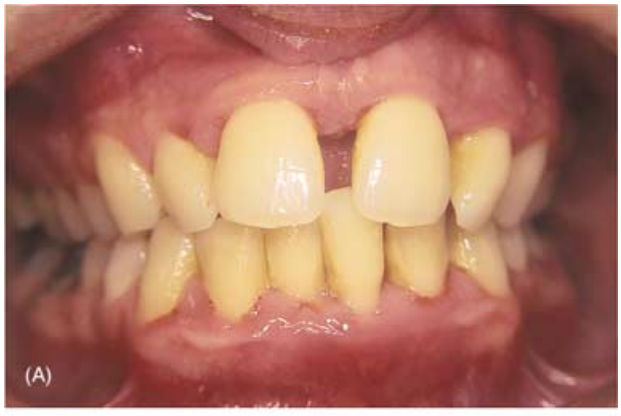

Fig. 2. Advanced localized aggressive periodontitis in a medically healthy 19-year-old Caucasian male. Note that there are minimal amounts of plaque and gingival inflammation (A). Radiographs of the mandibular anterior teeth showing the massive amount of bone loss (B). The periodontal damage was confined to the permanent incisors and first molars.

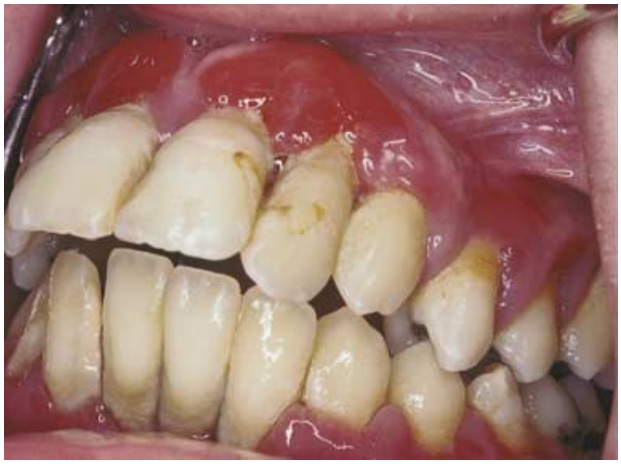

Fig. 3. Generalized aggressive periodontitis in a medically healthy 15-year-old Caucasian male. Note the intense inflammation and heavy deposits of plaque and calculus. Massive bone loss was present around all teeth.

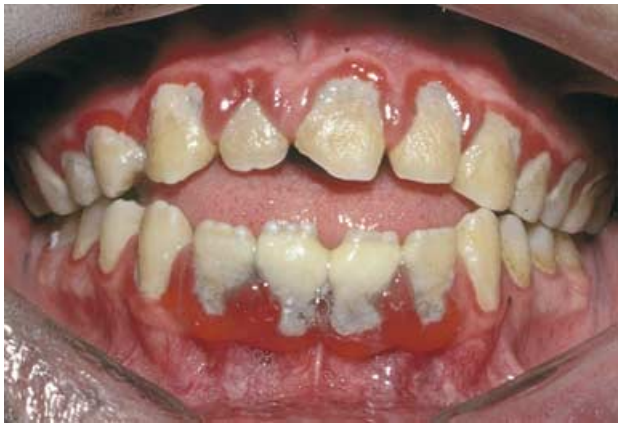

Fig. 4. Periodontitis as a manifestation of systemic disease. Plaque-induced periodontitis aggravated by increased susceptibility to infection in a 24-year-old male with Down's syndrome. Note the intense gingival inflammation.

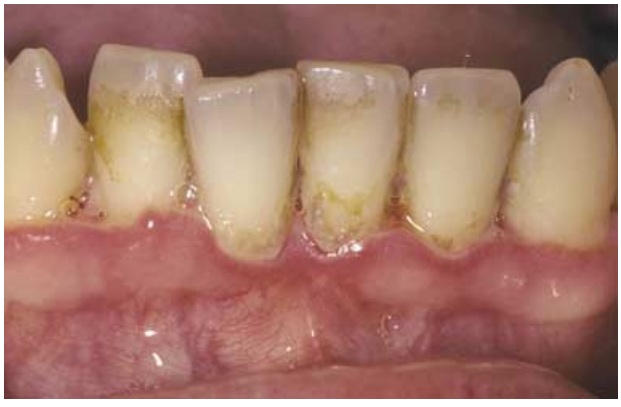

Fig. 5. Necrotizing ulcerative gingivitis in a 25-year-old Caucasian male. Note the cratering of the interproximal gingiva in most areas. The gingival lesions were painful.

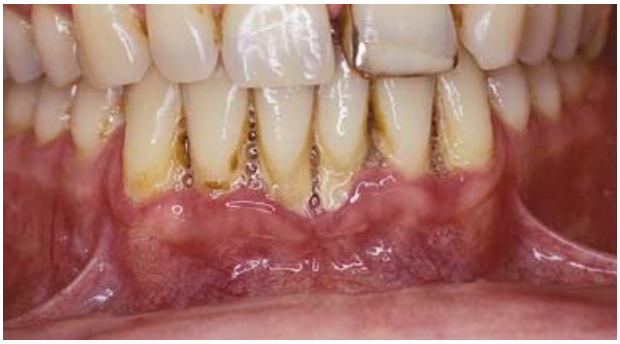

Fig. 6. Necrotizing ulcerative periodontitis in a medically healthy 37-year-old Caucasian male. The severity and pattern of attachment loss in the mandibular anterior region is similar to that often observed in HIV-positive patients. However, the photograph was taken in 1968, well before the AIDS epidemic. The patient complained of pain from the affected areas.

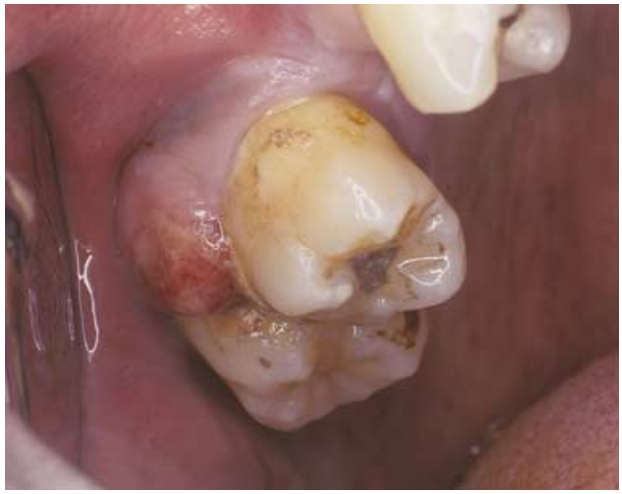

Fig. 7. Periodontal abscess that rapidly developed over a 2-day period between the maxillary second and third molars in a 43-year-old African American male with untreated chronic periodontitis. Both molars were free of pulpal disease.

(46, 61). If there is uncertainty, clinicians often use what is sometimes called a *working diagnosis* to guide the information-gathering process. The relevant information is identified and then analyzed within the context of the entire patient. A decision is then made as to what label or diagnosis should be placed on the patient's disease or condition. It should be emphasized that patients may have more than one disease or condition simultaneously affecting the periodontium. For example, Fig. 8 shows a 50-year-old white female who has both chronic periodontitis and benign mucous membrane pemphigoid.

Table 2. Partial list of possible diagnoses for destructive types of periodontal diseases (1999 Classification) (10)

Chronic periodontitis (Localized/Generalized)	(30)
Localized aggressive periodontitis	(52, 95)
Generalized aggressive periodontitis	(52, 95)
Periodontitis as a manifestation of systemic diseases	
Associated with hematologic disorders:	
Acquired neutropenia	(43)
Leukemias	(6, 91)
Associated with genetic disorders	
Familial and cyclic neutropenia	(76, 90)
Down's syndrome	(78)
Leukocyte adhesion deficiency syndromes	(65, 97)
Papillon-Lefèvre syndrome	(38, 50)
Chediak-Higashi syndrome	(37)
Langerhans cell disease (histiocytosis syndromes)	(68, 96)
Glycogen storage disease	(50)
Chronic granulomatous disease	(17)
Infantile genetic agranulocytosis	(81)
Cohen syndrome	(1, 50)
Ehlers-Danlos syndrome (Types IV and VIII)	(50, 75)
Hypophosphatasia	(18)
Crohn disease (inflammatory bowel disease)	(30, 47)
Marfan syndrome	(92)
Necrotizing ulcerative periodontitis (NUP)	(23, 69)
Abscesses of the periodontium	(23, 39, 63)
Combined periodontic–endodontic lesions	(64, 79)

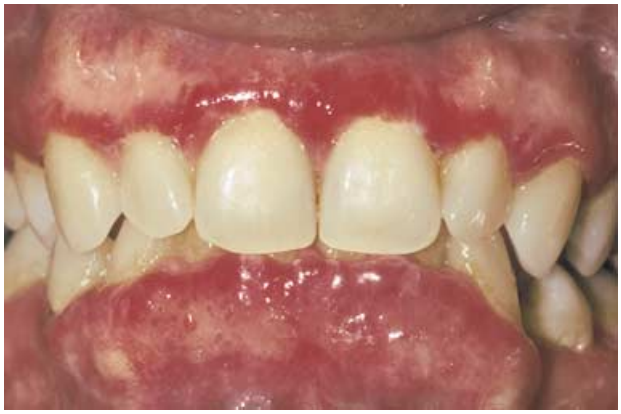

Fig. 8. A 50-year-old female with chronic periodontitis and benign mucous membrane pemphigoid.

The second question is a bit easier to answer since it only requires one to decide on a way to characterize the *severity* of the patient's plaque-induced periodontal disease. Most clinicians designate severity by using a 3-tiered system: 1 – slight, 2 – moderate, or 3 – severe. Sometimes ''initial'' and ''mild'' are used interchangeably with ''slight''; ''advanced'' may be used instead of ''severe.'' It makes no difference what words are chosen as long as the terms are understood by other clinicians. In cases of periodontitis, it has been recommended that severity be categorized on the basis of the amount of clinical attachment loss as follows: Slight = 1–2 mm, Moderate = 3–4 mm, and Severe ≥5 mm clinical attachment loss (10). Clinical attachment loss is measured with a periodontal probe and is the distance from the cementoenamel junction to the base of the probeable crevice (7). In cases of gingivitis, a similar 3-tiered system is frequently used: 1 – slight, mild, 2 – moderate, and 3 – severe. However, these designations are based on subjective clinical assessments of the intensity of the gingival inflammation (i.e. degree of redness, swelling, bleeding).

In answering the third question a decision needs to be made as to whether a patient's periodontal disease is localized or generalized. This decision is arbitrary since there are no hard-and-fast rules that can be applied for determining the *extent* or intraoral distribution of a patient's periodontal disease. Nevertheless, it has been recommended that the disease be called localized if up to 30% of the teeth are affected and generalized if >30% of the teeth are involved (10). Since during a routine periodontal examination, probing depths and clinical attachment loss measurements are taken from six sites around each tooth, it is theoretically possible to assign a diagnosis to a single surface on a given tooth, a single tooth, a quadrant, or the entire patient. However, for practical reasons, clinicians usually combine all of the clinical findings in a summary or global statement by assigning a diagnosis for the entire patient. Exceptions to this practice are common. The diagnosis can be phrased many different ways depending on how precise or detailed one wants to be. For example, Table 3 shows multiple ways to correctly phrase the periodontal diagnosis for a middle-aged patient with slight gingivitis around most teeth, who also has severe chronic periodontitis (clinical attachment loss = 6 mm) localized to the mesial surfaces of three teeth on the mandibular left where there are 8 mm probing depths and bleeding on probing at the periodontitis-affected sites.

Table 3. Some of the multiple ways to correctly phrase a periodontal diagnosis for a middle-aged patient with slight gingivitis around most teeth, who also has severe chronic periodontitis (clinical attachment loss = 6 mm) localized to the mesial surfaces of three teeth on the mandibular left where there are 8 mm probing depths and bleeding on probing at the periodontitis-affected sites

Diagnostic field and emphasis	Phrasing of the diagnosis
Focus on one disease (periodontitis)	
Single surfaces of affected teeth	Severe chronic periodontitis on the mesial surfaces of teeth #18, 19, and 20
Single teeth	Severe chronic periodontitis localized to teeth #18, 19, and 20
Quadrant	Severe chronic periodontitis localized to the mandibular left quadrant
Patient	Localized severe chronic periodontitis
Focus on both diseases (gingivitis and periodontitis)	
Patient and single surfaces of affected teeth	Generalized slight gingivitis with severe chronic periodontitis on the mesial surfaces of teeth #18, 19, and 20
Patient and single teeth	Generalized slight gingivitis with severe chronic periodontitis localized to teeth #18, 19, and 20
Patient and quadrant	Generalized slight gingivitis with severe chronic periodontitis localized to the mandibular left quadrant
Patient	Generalized slight gingivitis with localized severe chronic periodontitis

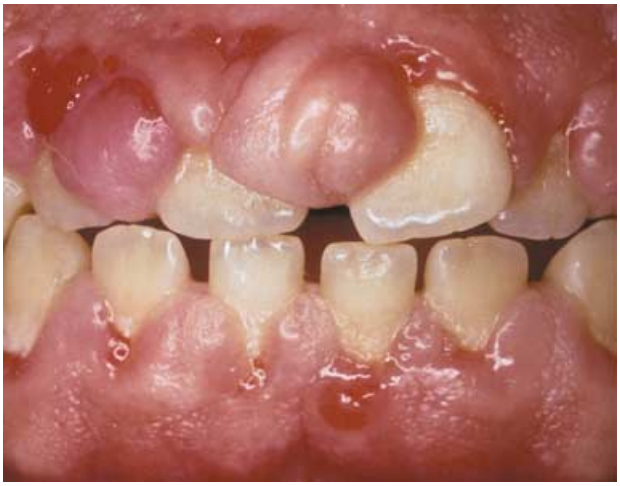

Fig. 9. Gingival enlargement in a patient taking phenytoin for cerebral seizures. The gingival margins on most teeth are coronal to the cementoenamel junction (CEJ).

It should be noted that probing depth measurements, while not used as the primary criterion for establishing the severity of periodontitis, are a useful piece of information. Probing depth is measured with a periodontal probe and is the distance from the gingival margin to the base of the probeable crevice. It is not the main criterion for severity because the gingival margin is not a fixed reference point from which to measure. For example, in cases where there is gingival enlargement and swelling, the gingival margin can be considerably coronal to the cementoenamel junction (Fig. 9). In other instances, where there has been recession, the gingival margin is apical to the cementoenamel junction (Fig. 10). Most importantly, the gingival margin can move coronal or apical to the cementoenamel junction over time and therefore is not a good reference point from which to assess longitudinal changes in clinical attachment.

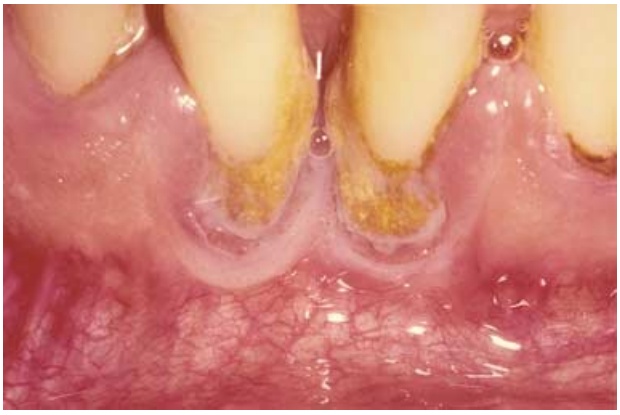

Fig. 10. Gingival recession in a 40-year-old female with chronic periodontitis. The gingival margin is apical to the CEJ on the facial surfaces of the two mandibular central incisors.

Nevertheless, probing depth measurements are of considerable importance since they provide a useful assessment of location and size of the principal habitats (i.e. periodontal pockets) of subgingival bacteria. Deep pockets are of concern because they are difficult for both the patient and therapist to clean (7). It is because of this that one of the goals of periodontal therapy is probing depth reduction.

Establishing a diagnosis in treated patients

Important steps in the management of patients with plaque-induced periodontal diseases are evaluations of post-treatment results. Such evaluations can occur at two critical points in monitoring the outcomes of therapy. The first is performed at the end of active therapy when the clinician must decide if treatment has been successful. Has the desired short-term clinical outcome been attained (9)? The second set of evaluations are repeatedly performed at multiple stages during the maintenance phase of therapy. Has the patient remained free of recurrent disease (9)? At each of these post-treatment evaluations a periodontal examination is performed and a diagnosis is made.

Treatment of plaque-induced periodontal diseases often results in the resolution of the patient's periodontal infection. It is important to note that periodontal therapy can change the pretreatment diagnosis to a more favorable post-treatment diagnosis. For example, effective treatment routinely converts plaque-induced gingivitis into a state of periodontal health (i.e. a Gingivitis to Periodontal Health shift). Successful treatment of plaque-induced periodontitis is often converted to a state of *periodontal health with a reduced periodontium*. In such cases, damage persists from the previous periodontitis in the form of gingival recession. An interesting diagnostic problem arises when successfully treated patients who once had periodontitis subsequently develop gingival inflammation during the maintenance phase of therapy. Do such patients have gingivitis superimposed on a reduced periodontium or do they have a recurrence of periodontitis? There is no simple answer to this question. However, depending on a variety of circumstances, most clinicians err on the side of caution and entertain the notion that the periodontitis may be recurring.

At a single evaluation visit one cannot determine if previously treated periodontitis is recurring. Data collected during multiple maintenance visits are required to make this determination. Perhaps the

earliest indication that the disease might be returning is the presence of bleeding on probing at multiple maintenance visits. This notion is supported by the results of a meta-analysis of treated populations of patients receiving periodontal maintenance care. In this analysis sites that exhibited the repeated presence of bleeding on probing were at a threefold higher risk of losing additional attachment compared to sites that did not bleed at most of the maintenance visits (9). Higher clinical attachment loss with time is the best single indication that periodontitis has probably recurred. One can be certain that the disease has recurred if there is bleeding on probing, increased probing depth, and higher clinical attachment loss measurements.

Although treatment of plaque-induced periodontitis usually results in resolution of the patient's periodontal infection, in some cases therapy is unsuccessful. Usually the causes for the unsuccessful treatment are unknown. One might reasonably suspect that the infection did not resolve because of poor plaque control by the patient or incomplete removal of subgingival calculus and plaque by the therapist. Additional plaque control instructions and further scaling and root planing are often successful in controlling the periodontal infections in these ''incompletely treated'' patients. However, there are some cases where the patient has been compliant with all recommendations and performs excellent plaque control. In addition, the therapist has provided a course of conventional periodontal therapy that succeeds in most patients. Nevertheless, the treatment fails to prevent the further progression of periodontitis. Such patients are sometimes assigned the diagnosis of ''refractory'' (21, 22, 98) or ''therapy-resistant'' (12, 13, 26) periodontitis. The diagnostic category of refractory periodontitis is a heterogeneous grouping since there are probably multiple forms of periodontitis that are nonresponsive to conventional therapy. However, most of the patients who have been studied started out with an initial diagnosis of chronic (adult) periodontitis. In retrospect, an appropriate diagnosis for this group of patients is refractory or treatment-resistant chronic periodontitis. Diagnostic approaches for this group of patients are discussed elsewhere in this volume (56).

Diagnosis and classification of developmental or acquired deformities and conditions of the periodontium

There are a large number of developmental or acquired deformities and conditions that affect the periodontium (Table 4). Some of them are simply departures from normal periodontal anatomy that may cause functional or esthetic problems for patients. Others can create an environment that promotes the development of plaque-induced periodontal diseases. Methods for the detection and diagnosis of these conditions are discussed elsewhere in this volume (62). Diagnostic methods for identifying the clinical effects of an injury-producing occlusion are also discussed (36).

Table 4. List of developmental or acquired deformities and conditions affecting the periodontium (1999 Classification) (10)

Tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis (15, 62)
Tooth anatomic factors
Dental restorations/appliances
Root fractures
Cervical root resorption and cemental tears
Mucogingival deformities and conditions (62, 77)
Gingival/soft tissue recession
Facial or lingual surfaces
Interproximal (papillary)
Lack of keratinized gingiva
Decreased vestibular depth
Aberrant frenum/muscle position
Gingival excess
Pseudopocket
Inconsistent gingival margin
Excessive gingival display
Gingival enlargement
Abnormal color
Occlusal trauma (35, 36)
Primary occlusal trauma
Secondary occlusal trauma

Distinguishing between chronic and aggressive forms of periodontitis

Most patients with plaque-induced periodontitis will have the chronic form (2, 72). The main clinical features and characteristics of chronic periodontitis are listed in Table 5. The typical patient is over 30 years of age with substantial deposits of plaque and calculus associated with the presence of gingival inflammation, periodontal pockets, and attachment loss. In most cases the disease is slowly progressing (16, 55, 73), but short periods of rapid attachment loss can occur (45, 89). Chronic periodontitis was once called ''adult periodontitis'' since it was believed that only adults developed the disease (5). However, epidemiologic

Table 5. Main clinical features and characteristics of chronic periodontitis (1999 Classification) (10)

- 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 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)
- Can be modified by factors other than systemic disease such as cigarette smoking and emotional stress

data clearly show that the disease can also be found in children and adolescents (3, 72). Although chronic periodontitis can occur in localized or generalized patterns, the two forms appear to be identical with regards to their etiology and pathogenesis.

Aggressive periodontitis is less common than chronic periodontitis and principally affects young patients (3, 72). It occurs in localized and generalized forms that differ in many respects with regard to their etiology and pathogenesis (52). Localized aggressive periodontitis (LAP) and generalized aggressive periodontitis (GAP) were previously called ''localized and generalized juvenile periodontitis,'', respectively (5). Features of aggressive periodontitis that are common to both the localized and generalized forms of the disease are shown in Table 6.

The thought process followed in distinguishing between chronic and aggressive forms of periodontitis initially focuses on 1) the amount and pattern of periodontal destruction and 2) the patient's age and medical status. One begins to suspect that a patient might have a form of aggressive periodontitis if they are young, medically healthy and present with extensive periodontal destruction. If the periodontal destruction is localized to interproximal areas of permanent first molars and incisors, the diagnosis of LAP is usually made. In addition, in most cases of LAP the traditional view is that the, ''amounts of microbial deposits are inconsistent with the severity of periodontal tissue destruction'' (52). If the destruction is found around at least three permanent teeth other than first molars and incisors, the diagnosis of GAP is usually made (52).

As in the case of chronic periodontitis, both forms of aggressive periodontitis are plaque-induced infections and host responses to plaque bacteria are responsible for most of the tissue destruction. The plaque biofilms are, however, often clinically thinner than in cases of chronic periodontitis. As mentioned above, this is particularly true in cases of LAP.

Based on several specific clinical and host-response differences between LAP and GAP, it is clear that LAP is not merely a localized form of GAP. Features that are said to be specific for each of these forms of aggressive periodontitis are shown in Table 7. Importantly, LAP generally has a circumpubertal onset or is first detected and diagnosed during puberty, whereas GAP is usually detected and diagnosed in people under 30 years of age. However, some patients with GAP may be older than 30 years of age. It has been suggested that patients with LAP usually mount a robust serum antibody response to

Table 6. Features of aggressive periodontitis that are common to both the localized and generalized forms of the disease (1999 Classification) (52)

Primary features
Except for the presence of periodontitis, patients are otherwise clinically healthy
Rapid attachment loss and bone destruction
Familial aggregation
Secondary features (often present)
Amounts of microbial deposits are inconsistent with the severity of periodontal tissue destruction
Elevated proportions of *Actinobacillus actinomycetemcomitans* and, in some populations, *Porphyromonas gingivalis* may be elevated
Phagocyte abnormalities
Hyperresponsive macrophage phenotype, including elevated levels of prostaglandin E_2 (PGE_2) and interleukin-1β (IL-1β)
Progression of attachment loss and bone loss may be self-arresting

Table 7. Specific features of localized and generalized aggressive periodontitis (1999 Classification) (52)

Localized aggressive periodontitis
Circumpubertal onset
Robust serum antibody to infecting agents
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 more than two teeth other than first molars and incisors
Generalized aggressive periodontitis
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

periodontal pathogens, whereas patients with GAP exhibit a poor antibody response to the infecting agents (52). Comparisions of the main clinical characteristics of chronic, localized aggressive, and generalized aggressive periodontitis are shown in Table 8.

Noninflammatory destructive periodontal disease (NDPD) – does it exist?

Most of the destructive forms of periodontal disease discussed in this chapter are primarily caused by dental plaque biofilms. Classification of these afflictions follows an infection/host response paradigm in which it is held that noxious materials from dental plaque bacteria induce an inflammatory response in the adjacent periodontal tissues (11). The lytic activities associated with inflammation are the primary way in which periodontal tissues are destroyed. Central to this paradigm is the notion that destruction of periodontal tissues is accompanied by an inflammatory response.

However, for the past 250 years some clinicians have described what they believed were ''noninflammatory destructive periodontal diseases'' in which periodontal tissues were gradually lost without the presence of inflammation (71). Indeed, from approximately 1920 to 1970, most classification systems for periodontal diseases included noninflammatory (i.e. degenerative, atrophic) categories. The primary reasons for including these categories were the clinical impressions (i.e. opinions) of clinicians and the long-held assumption that periodontal diseases followed the ''principles of general pathology'' in which ''there are three major tissues reactions: inflammatory, dystrophic, neoplastic'' (11, 70). Despite the lack of supporting scientific data, the existence of such diseases was never seriously challenged until it was convincingly shown in the 1970's that ''periodontosis,'' a name given to a destructive periodontal disease that was presumably a degenerative noninflammatory condition, was actually an infection with a definite inflammatory component (11). As a consequence of this discovery, most classification systems for periodontal diseases published after 1977 do not include degenerative or noninflammatory categories.

Page & Sturdivant have recently proposed the existence of a form of ''noninflammatory destructive periodontal disease (NDPD)'' and that it ''...is seen rather commonly by practicing periodontists'' (71). These authors supported their suggestion by presenting two case reports of patients who progressively experienced gingival recession and loss of attachment over a period of several years. Importantly, both patients practiced intense and frequent toothbrushing and use of ''...multiple forms of interdental cleaning including dental floss and interdental brushes and various probes, picks and sticks'' (71). In addition, the authors reviewed the early literature in which references have been made to the ''...absorption, or wasting of the alveolar processes.'' They quote an early book by Joseph Fox (1823) who stated, ''Sometimes this disease proceeds without the appearance of any assignable cause, the gums retain a very healthy aspect, are quite free of pain or inflammation, and yet will gradually recede, until the teeth become very loose'' (31). Finally, Page & Sturdivant state that the 1999 classification of periodontal diseases and conditions adopted by the American Academy of Periodontology (AAP) makes ''...no allowance for the possible existence of forms of periodontal disease that may not fulfil [*sic*] the prescribed characteristics'' (71).

Although their paper is interesting and thought provoking, the arguments presented by Page & Sturdivant for the existence of a specific noninflammatory destructive periodontal disease are weak and unconvincing. Examination of the two case reports strongly suggests that the progressive gingival recession and loss of attachment were secondary to self-inflicted injury caused by longstanding abusive oral

Table 8. Comparison of the main clinical characteristics of chronic periodontitis, localized aggressive periodontitis, and generalized aggressive periodontitis

Chronic periodontitis	**Localized aggressive periodontitis**	**Generalized aggressive periodontitis**
Most prevalent in adults, but can occur in children	Usually occurs in adolescents (circumpubertal onset)	Usually affects people under 30 years of age, but patients may be older
Slow to moderate rates of progression	Rapid rate of progression.	Rapid rate of progression (pronounced episodic periods of progression)
Amount of microbial deposits consistent with severity of destruction	Amount of microbial deposits *not* consistent with severity of destruction	Amount of microbial deposits sometimes consistent with severity of destruction
Variable distribution of periodontal destruction; no discernible pattern • No marked familial aggregation • Frequent presence of subgingival calculus	Periodontal destruction localized to permanent first molars and incisors • Marked familial aggregation. • Subgingival calculus usually absent	Periodontal destruction affects many teeth in addition to permanent first molars and incisors • Marked familial aggregation. • Subgingival calculus may or may not be present

hygiene procedures. Similar cases appear in Hirschfeld's classic book, *The Toothbrush: its Use and Abuse* (40). It should be pointed out that the 1999 AAP classification includes a category of ''Non-plaque-induced gingival lesions'' with a subgroup of ''Traumatic lesions'' under which ''physical injury'' is specifically listed (10). It is unclear why the gingival recession and loss of attachment observed in the two cases was not attributed to the cumulative effects of repeated physical injury to the periodontium. Certainly, repetitive physical injury to the gingiva and periodontium can result in gingival recession and loss of attachment (51, 74, 94). Contrary to the statements of Page & Sturdivant, there is nothing in the AAP classification to indicate that ''*. . .all* forms of destructive periodontal disease are infectious, and thay [*sic*] they are *all* characterized by chronic inflammation, pocket formation and progressive deepening, and loss of attachment and alveolar bone'' (71). Indeed, periodontal destruction can be a feature of some non-plaque-induced gingival lesions (28, 51, 57, 74, 83, 94).

The most troublesome suggestion regarding the existence of NDPD is the notion that it is ''noninflammatory.'' If the observed periodontal destruction is due to repetitive physical injury to the periodontium (which is likely), it is difficult to imagine the existence of small and short-lived traumatic wounds without the development of transient inflammation during healing. Therefore, use of the word ''noninflammatory'' is ill-advised since it suggests that inflammation is not part of the process associated with tissue destruction and remodeling. Although it is possible that inflammation may not be a central component of the tissue-destructive processes involved in the two cases described by Page & Sturdivant, it is highly unlikely that the injured tissues were free of inflammation at all times during the development of attachment loss and gingival recession. Alternatively, no data are presented to rule out the possibility that injury-induced inflammation is involved in the process leading up to the observed periodontal damage. It is unlikely that intermittent clinical observations on two patients would permit the detection of transient episodes of periodontal inflammation. Without a sound scientific basis, it is unwise to resurrect the old concept that some forms of destructive periodontal disease are non-inflammatory.

Finally, Page & Sturdivant appear to have uncritically interpreted some very old literature and assumed that what was written by John Hunter (*circa* 1771) and Joseph Fox (*circa* 1806) must be true.

Although Hunter and Fox made significant and insightful contributions to the field of their day, over-interpretation of what they wrote should be avoided. Misinterpretation of clinical observations was common among many early authors (11). In retrospect, with the limited technology and information base available to them, the failure of the pioneers of periodontics to recognize the infectious and inflammatory nature of such diseases as localized aggressive periodontitis is understandable. Indeed, early failures to recognize the presence of periodontal inflammation is not surprising since in many cases of chronic and aggressive periodontitis the tissues superficially look healthy. It is only upon closer inspection with calibrated periodontal probes, which were not invented until 1925 (88), that bleeding on probing (now a universally accepted sign of periodontal inflammation) permitted the detection of hidden-from-view periodontal inflammation.

Nevertheless, the two cases presented by Page & Sturdivant clearly demonstrate that not all patients who experience progressive periodontal destruction must necessarily have clinical features commonly associated with chronic periodontitis (i.e. formation of periodontal pockets, overt and persistent signs of inflammation, abundant accumulation of dental plaque biofilms) (71). The important question that might have been raised by these authors is: Does progressive periodontal destruction presumably caused by self-inflicted vigorous oral hygiene procedures fit under the 1999 AAP category of ''Non-plaque-induced gingival lesions'' (subcategory ''Traumatic lesions'')? Should the classification be modified to include a category of ''Non-plaque-induced periodontal lesions'' (subcategory ''Traumatic lesions'')? If one chooses to make inflexible or rigid interpretations of the 1999 AAP classification, it could be argued that *gingival* lesions and *periodontal* lesions should be separate items.

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