Contemporary Concepts in the Diagnosis of Periodontal Disease

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THE CHALLENGES OF PERIODONTAL DIAGNOSIS

Periodontitis is an inflammatory disease of bacterial origin that results in the progressive destruction of the tissues that support the teeth, specifically the gingiva, periodontal ligament, and alveolar bone. Although there have been significant advances in the understanding of the cause and pathogenesis of periodontal disease over the past 40 years, the traditional methods by which clinicians diagnose periodontal disease have remained virtually unchanged. The diagnosis of periodontal disease relies almost exclusively on clinical parameters and traditional dental radiography. Clinicians use clinical and radiographic findings to diagnose patients according to the classification scheme developed at the 1999 International Workshop for the Classification of Periodontal Diseases and Conditions (**Table 1**). These traditional diagnostic tools have some significant shortcomings. Clinical assessments such as probing depth (PD) and clinical attachment level (CAL) are somewhat subjective and time consuming and therefore underutilized in general dental practice.[1] Studies of the progression of periodontitis have demonstrated that there are periods of active tissue destruction separated by periods of inactive disease;[2–4] however, traditional clinical assessments do not enable a practitioner performing a single routine periodontal examination to determine whether active tissue destruction is occurring. There are, for example, no definitive means of determining whether gingival inflammation in a successfully treated case of periodontitis represents early recurrent disease or gingivitis on a stable but reduced periodontium. Demonstrating progressive loss of periodontal support requires longitudinal assessment. Current diagnostic methodologies do not enable us to accurately predict

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Table 1	
Classification of periodontal diseases and conditions	
I. Gingival Diseases	A. Plaque-associated gingival diseases Non–plaque-induced gingival diseases
II. Chronic Periodontitis	B. Localized Generalized
III. Aggressive Periodontitis	C. Localized Generalized
IV. Periodontitis as a Manifestation of Systemic Disease	D. Associated with hematologic disorders Associated with genetic disorders Not otherwise specified
V. Necrotizing Periodontal Diseases	E. Necrotizing ulcerative gingivitis Necrotizing ulcerative periodontitis
VI. Abscesses of the Periodontium	F. Gingival abscess Periodontal abscess Pericoronal abscess
VII. Periodontitis Associated with Endodontic Lesions	G. Combined periodontic-endodontic lesions
VIII. Developmental or Acquired Deformities and Conditions	H. Localized tooth-related factors that modify or predispose Mucogingival deformities and conditions around teeth Mucogingival deformities and conditions on edentulous ridges Occlusal trauma

Adapted from Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol 1999;4(1):2, 3; with permission.

which periodontal sites, teeth, or individuals are susceptible to further periodontal breakdown. Given the limitations of current diagnostic tools, researchers are working to develop techniques that address some of these inadequacies. In this article, the authors review the diagnostic techniques currently used and present new approaches and technologies that are being developed to improve the diagnosis of periodontal disease.

CURRENT DIAGNOSTIC STRATEGIES

In response to pathogenic bacteria in dental plaque (a biofilm adherent to the tooth surface), an innate inflammatory response as well as cellular and humoral immune responses are mounted locally in the periodontal tissues. The complex host response is aimed at containing the infectious stimulus and preventing bacterial invasion into the tissues. If the infection cannot be contained, the local release of proinflammatory cytokines and tissue degrading enzymes results in damage to the supporting hard and soft tissues of the tooth. The junctional epithelium becomes ulcerated and migrates apically, and there is destruction of the gingival connective tissue, periodontal ligament, and alveolar bone. Although pathogenic bacteria are capable of degrading host tissues, it is the host's response to pathogenic bacteria rather than the bacteria themselves that is responsible for most of the tissue breakdown associated with periodontitis.[5] The traditional diagnostic methods described in the following sections aim to identify the etiologic factors, assess the clinical signs of the inflammatory process, and determine the degree to which periodontal destruction has occurred.

Assessment of Etiologic Factors

Bacteria are necessary but insufficient by themselves to cause periodontal disease.[6] It is generally accepted that even in the presence of pathogenic bacteria, individuals are variably susceptible to tissue breakdown. Identifying the presence of specific periodontal pathogens in dental plaque is not currently a strategy used to establish a periodontal diagnosis.[1] Nonetheless, because bacteria are the initiating factor (and primary target of most of the present therapeutic modalities), it is important to assess the degree of bacterial plaque present and counsel patients on proper plaque control. It is also important to identify any factor that might make an individual susceptible to the accumulation of dental plaque. Some of these factors include a lack of manual dexterity associated with arthritis or other conditions, a reduced frequency of oral hygiene practices, improper technique, and tooth anatomy that promotes plaque retention.

Susceptibility to periodontitis is conferred by several established risk factors, such as diabetes mellitus and cigarette smoking.[7] Certain genetic syndromes are also associated with periodontal disease.[8] It is for these reasons that a thorough medical history, including a history of cigarette smoking, is an important part of establishing a periodontal diagnosis.

Assessment of Gingival Inflammation

Much information regarding the degree of gingival inflammation can be obtained from a simple visual inspection of the tissues. Healthy gingiva is typically pink and firm and has a knife-edged margin. Inflamed tissues exhibit cardinal signs of inflammation, such as redness and swelling (**Fig. 1**). Bleeding on probing (BOP) is an important indicator of gingival inflammation within the periodontal pocket. It occurs because of microulcerations in the junctional epithelium. BOP is influenced by repeated probe insertions in a short time as well as by the use of excessive force (>25 N). Purulent exudate can also be an important sign of gingival inflammation; however, true suppuration may be difficult to distinguish from plaque that is expressed from the gingival crevice.

Calor, or heat, is another cardinal sign of inflammation and has been investigated as a diagnostic measure of periodontal status. Haffajee and colleagues[9] used a periodontal temperature probe (Periotemp, ABIO-DENT, Inc, Danvers, MA, USA) to

Fig. 1. A patient with plaque-induced gingivitis. There is evidence of the cardinal signs of inflammation, including erythema and edema.

assess subgingival temperature and found that elevated mean subgingival temperature was related to subsequent attachment loss.

Assessment of Loss of Periodontal Attachment

PD assessment is probably the most commonly used clinical measure for detecting loss of periodontal support. It is measured from the free gingival margin (FGM) to the depth of the probable crevice. The depth of a healthy gingival sulcus ranges from 1 to 3 mm. PD is not the most objective measure of loss of periodontal tissues because the position of the FGM is variable. When there is gingival inflammation, the FGM may be located more coronal than normal because of edema in the tissues (the FGM is normally located 1.5–2 mm coronal to the cementoenamel junction [CEJ]). In this situation, there may be a deeper-than-normal PD even in the absence of loss of periodontal attachment. Such a deepened pocket is described as a pseudopocket. A true periodontal pocket occurs when there has been apical migration of the junctional epithelium and loss of supporting tissues of the tooth. The PD may also be normal in the presence of significant attachment loss. This may occur in the case of treated periodontitis or when the disease process manifests with gingival recession rather than pocket formation.

CAL is a more objective measure of loss of periodontal support because it is measured from a fixed point on the tooth, usually the CEJ, if detectable. CAL is defined as the distance between the CEJ and the base of the probable pocket (**Fig. 2**). Because the apical termination of the junctional epithelium is normally located at the CEJ, there should be no CAL when the periodontal tissues are healthy and there is no history of periodontitis. When the gingival margin is located coronal to the CEJ, CAL is measured by subtracting the distance of the FGM to the CEJ from the PD (PD−[FGM−CEJ]). When there is gingival recession, CAL is calculated by adding the PD and the amount of recession. Calculating the CAL can be challenging, and this variable is more often used in research than in everyday clinical practice. Existing CAL also does not give any indication of current disease activity.

When interpreting the PD and CAL measurements made with conventional periodontal probes, it is important to consider that these values depend on the inflammatory state of the tissues. When probing healthy gingival tissues, the periodontal probe generally stops coronal to the apical extent of the junctional epithelium, which is at the CEJ.[10] When the gingiva is inflamed and the gingival connective tissue has been infiltrated by inflammatory cells, there is less resistance to probe penetration and the periodontal probe generally passes apical to the level of the connective tissue attachment.[11,12] As a result, PD and CAL values may be overestimated in inflamed sites and underestimated in healthy sites. The depth of probe penetration may also be influenced by factors such as the diameter of the probe tip, insertion force, and angulation of the probe. Electronic probes were developed to overcome some of these technical difficulties. Electronic probes, such as the Florida probe (Florida Probe Company, Gainesville, FL, USA), have the advantage of controlling insertion force, automatic data capture into a computer, and a higher resolution than manual probes.[13] Electronic probes have the disadvantage of underestimating PD and CAL in untreated patients. Despite some acknowledged problems, manual probes are perfectly acceptable for routine periodontal examinations and provide results comparable to those with electronic probes.[14]

Other clinical variables used to assess the degree of existing periodontal destruction include mobility and the degree of furcation involvement. Tooth mobility may be caused by several factors, but loss of periodontal attachment is one of the more common causes.[15] Both mobility and furcation involvement are important determinants of a tooth's prognosis.
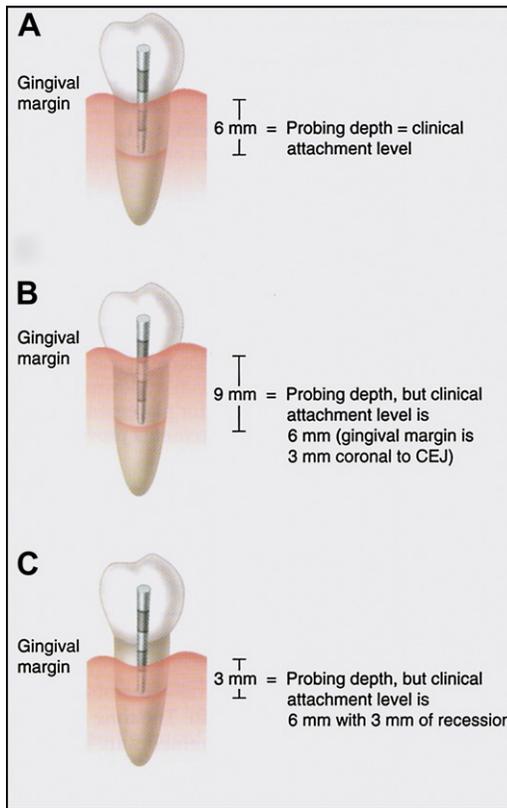
Fig. 2. A depiction of how the parameters PD and CAL relate to one another. (*A*) The gingival margin is at the level of the CEJ, so PD is equal to the CAL. (*B*) The gingival margin is coronal to the CEJ, so the PD is greater than the CAL. (*C*) The PD is within normal limits, but there is gingival recession and significant CAL. (*Adapted from* Armitage, G. Clinical periodontal examination. In: Rose LF, Mealey BL, Genco RJ, et al, editors. Periodontics: medicine, surgery, and implants. St Louis (MO): Elsevier Mosby; 2004. p. 140; with permission.)

RADIOGRAPHIC ASSESSMENT OF PERIODONTAL DISEASE

Radiographs are an essential component of the periodontal examination and indispensable in establishing a periodontal diagnosis. Important information regarding the position and architecture of the alveolar crest of bone is obtained from radiographs. Bite-wings are considered the most accurate intraoral radiographs for determining the height of the alveolar crest. In the absence of bone loss, the alveolar crest is generally located 1 to 2 mm apical to the CEJ (**Fig. 3**A).[16] Vertical bite-wings may need to be taken to visualize the osseous crest in a patient with attachment loss (**Fig. 3**B). Periapical radiographs give the clinician important information regarding crown to root ratio, the periodontal ligament space, and the presence of periapical abnormality. Intraoral radiographs are generally considered preferable to panoramic radiographs for use in periodontal assessment; however, some studies have demonstrated that panoramic radiographs can be used to assess alveolar bone height.[17]

Despite their value in periodontal diagnosis, radiographs have several limitations as diagnostic tools. First, they do not give any information about disease activity or
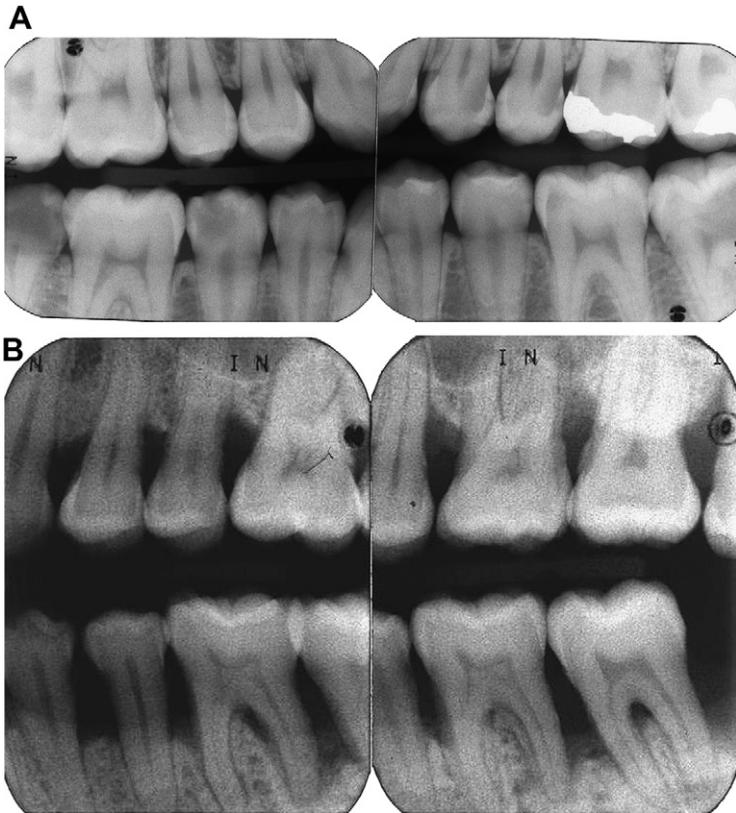
Fig. 3. (*A*) Horizontal bite-wing radiographs show that the height of the alveolar bone is in a normal position. (*B*) Vertical bite-wing radiographs demonstrating vertical and horizontal bone loss.

progression. A successfully treated case of periodontitis is likely to have similar pretreatment and posttreatment levels of radiographic bone loss. Second, studies in the periodontal literature have generally demonstrated that radiographs tend to underestimate the amount of attachment loss[18,19] and that clinical changes (attachment loss) precede radiographic changes.[20]

Subtraction radiography is a technique that longitudinally assesses change in bone density. Two radiographs with the same geometry are exposed at 2 different times. The image present in the first film is subtracted from that in the second film. The difference reflects bone gain or loss. The technique can detect bone density changes as low as 5%, whereas sequentially taken conventional radiographs reveal bone changes only after 30% to 50% of the bone has been resorbed.[21] Subtraction radiography has evolved with the introduction of intraoral digital radiography, and some of the shortcomings of the original technique have been addressed.[22] Nonetheless, this technique is generally not used in clinical practice.

SUPPLEMENTAL DIAGNOSTIC TESTS

The clinical and radiographic assessments described earlier are the most commonly used measures of periodontal disease. However, there are several supplemental tests

that have been developed to address the fact that traditional approaches do not adequately identify patients or sites with progressive disease (or at risk for progressive disease). Supplemental tests may also be used to assess the response to therapy and determine appropriate recall intervals.[23] For several reasons, these tests, which include microbial, biochemical, and genetic tests, are not routinely used in clinical practice. However, many of these tests offer the clinician information that is not available from current diagnostic procedures.

Microbial Testing

Although there are different bacterial species associated with gingival health and disease, microbial testing is not currently used to establish a periodontal diagnosis. Whether the presence of certain bacteria may help distinguish between different forms of periodontitis (chronic vs aggressive) is currently a matter of controversy.[24] In a systematic review, Mombelli and colleagues[25] found that the presence or absence of certain identified periodontal pathogens could not distinguish cases of chronic periodontitis from cases of aggressive periodontitis. Other possible uses of microbial testing are for selection of an appropriate systemic antibiotic, assessment of therapeutic outcomes, and/or risk assessment. There are several methods for detecting bacteria in dental plaque. These include bacterial culture, immunologic assays, enzymatic assays, and molecular biologic techniques that detect bacterial DNA or RNA.

Bacterial culture is the gold standard against which new microbial tests are compared. It involves growing bacteria in either aerobic or anaerobic conditions on different media and performing tests to identify and quantify specific species. This technique enables the characterization of pathogens as well as the determination of antibiotic susceptibility. The drawbacks of culture are that the plaque sample must contain viable bacteria and that some putative pathogens are difficult to cultivate.[24]

Immunologic methods use antibodies that target specific bacterial antigens. When the antibodies bind their antigen, the reaction can be visualized by techniques such as direct and indirect immunofluorescent microscopic assays, flow cytometry, and enzyme-linked immunosorbent assay.[24] Immunologic techniques enable the identification and quantification (or semiquantification) of bacteria. However, the only bacteria that are identified are those for which specific antibodies are available.

Several putative periodontal pathogens such as *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Aggregatibacter actinomycetemcomitans* possess in common a trypsinlike enzyme that hydrolyzes a substrate *N*-benzoyl-DL-arginine-2-naphthylamide (BANA). A diagnostic test that measures the activity of this trypsinlike enzyme was developed so as to identify the presence of oral bacteria that produce the enzyme. Loesche and colleagues[26] published a study comparing the BANA test to other methods of microbial testing and found that the BANA test had similar sensitivity as the other techniques that were evaluated. The BANA test is easy to perform chairside and was commercially available for a brief period in the 1990s. Serious limitations of the test include its inability to distinguish between individual bacteria, the ability to detect pathogens only when they are present in high numbers, and the fact that its diagnostic utility has not been validated in clinical trials.[24]

Molecular biologic techniques use the bacterial genome as a means of identifying specific bacteria. DNA isolated and purified from plaque samples can be analyzed via nucleic acid probes or polymerase chain reaction (PCR). Nucleic acid probes are synthesized sequences of DNA or RNA that are complementary to specific nucleic acid sequences in the bacterial genome. Bacteria can be identified when DNA isolated from dental plaque is hybridized (paired with complementary DNA) with species-specific probes that are labeled to allow visualization.[24] Checkerboard hybridization

is a technique that uses probes to simultaneously test for the presence of up to 43 bacterial species.[27] Checkerboard hybridization enables rapid processing of numerous plaque samples and is often used for research purposes. PCR uses a DNA replicating enzyme (polymerase) to amplify target sequences of DNA. Standard PCR is not a quantitative assessment of identified bacteria, although a technique called real-time PCR does enable quantification. Tests that use the genome have the advantage of not requiring viable bacteria, but they are costly and require sophisticated laboratory equipment, so they are not practical for routine clinical use.[24]

Biochemical Analysis as Part of Periodontal Diagnosis

The biochemical assessment of periodontal disease can be accomplished using several approaches. The most practical and least-invasive approach involves analysis of biologic fluids that are derived from the periodontal tissues or contain specific mediators that are present as a result of periodontal disease. The biologic fluids that have been studied to understand the nature of destructive periodontitis and to identify potential diagnostic markers of active disease include serum (blood), gingival fluid, and saliva.

As the understanding of the pathophysiology of periodontal disease advanced in the 1970s, researchers began to analyze serum to identify the nature of the host's response in periodontal disease.[28,29] Studies of serum antibody levels to periodontal bacteria were among the earliest investigations demonstrating that a humoral immune response occurs in patients with periodontitis. More recent studies have demonstrated that patients with periodontitis have elevated antibody titers to subgingival pathogens.[30] Recently, serum levels of markers of the inflammatory response have been studied for their relationship to periodontitis. The levels of inflammatory cytokines (ie, interleukin [IL]-6) and general markers of inflammation (ie, C-reactive protein) have been shown to be elevated in the blood of patients with periodontitis.[31] Nevertheless, serum markers of periodontitits, or of inflammation, are not currently used as diagnostic tests for periodontitis.

Gingival crevicular fluid (GCF) is a serum transudate, or more commonly an inflammatory exudate, that emanates from the gingival crevice and can be collected from the orifice of the crevice. A great deal of attention has been placed on the analysis of GCF for diagnostic purposes. GCF collection is most commonly accomplished with the use of small methylcellulose filter paper strips placed within the crevice or at the orifice. There has been a debate regarding the technical aspects of collecting GCF and reporting the data (as concentration or total amount of mediator in a timed collection). The reasons for this debate include the variable amount of fluid that can be collected at different tooth sites and the observation that the collection procedure can influence fluid volume, because the insertion of a filter strip can, over time, cause disruption of the underlying capillary bed. At present, most studies use a timed (30 seconds) insertion of the GCF strip to the depth of the sulcus or pocket. This procedure eliminates the need for the determination of the volume of fluid that was collected (this volume can be determined using an electronic meter known as the Periotron [Oraflow Inc, Smithtown, NY, USA])[32] and allows for the comparison of samples based on the standardized collection time.

A wide variety of mediators have been studied in GCF.[33] These mediators can generally be classified as assessing the host immune or inflammatory response or metabolic markers associated with periodontitis. The former includes antibodies, proteases and other enzymes (including the matrix metalloproteinases [MMPs]), proinflammatory cytokines (ie, IL-1β, IL-6, IL-17, tumor necrosis factor [TNF]-α), and other molecules in the different inflammatory cascades (ie, prostaglandin E$_2$). Measures of

tissue metabolism include markers of cell necrosis (ie, the enzymes lactate dehydro-genase and aspartate transaminase), molecules that play a role in the response to oxidative stress (ie, glutathione), growth factors (ie, transforming growth factor β), and measures of bone remodeling and turnover (ie, receptor activated nuclear factor-κB ligand [RANKL] and osteoprotegerin). At present, markers of inflammation have received the most attention. Several of these markers have been evaluated for their relationship to active, progressive periodontitis in clinical trials, and 2 diagnostic tests based on the analysis of elastase and aspartate aminotransferase were available commercially as chair-side tests for the diagnosis of periodontitis.

GCF has also been analyzed with infrared (IR) spectroscopy. IR spectroscopy is a technique that involves the analysis of biologic fluids to quantitatively determine ana-lytes of interest.[34] Vibrating covalent bonds of organic molecules absorb a character-istic wavelength of IR light. The spectrum of absorbed light may be used to establish a molecular fingerprint of a tissue or fluid.[35] IR analysis of GCF has recently been shown to distinguish between periodontal health and disease.[36] Longitudinal studies are needed to determine whether IR can be used to predict the risk for progressive disease.

Despite the enormous interest in the biochemical analysis of GCF, the use of this fluid as the basis of a diagnostic test has not been embraced by the profession. The reasons relate to difficulty in developing a logical and practical strategy for sampling GCF. The filter paper strips collect fluid only from a 2- to 3-mm wide portion of the crevice. To assess the entire dentition, sampling of GCF has traditionally occurred at a preestablished site on each tooth (ie, the mesiobuccal line angle of each tooth). This procedure is time consuming and may not capture a sample that is representative of the entire periodontium.

A more practical approach to the biochemical diagnosis of periodontal disease is offered by the analysis of saliva. Saliva has been analyzed as a diagnostic fluid in medicine,[37] and the analysis of saliva also offers intriguing possibilities as the basis of diagnostic tests for oral disease. Whole saliva can be collected noninvasively and analyzed for the presence of markers that, in general, are derived from GCF and have been shown to be associated with the risk for active periodontal destruction. The markers include enzymes that indicate cell necrosis and tissue destruction and inflammatory markers, such as TNF-α, IL-1β, MMP-8, and the neutrophil-derived enzyme, β-glucuronidase. Studies have shown these markers to be elevated in the saliva of patients with periodontitis.[38]

As technologies evolve, saliva may also be analyzed for genomic and microbial markers of periodontal disease.[39] Salivary RNA has been identified and used in the diagnosis of oral cancer and Sjögren syndrome; however, to date there are no salivary DNA or RNA biomarkers for periodontal disease.[39] The National Institute of Dental and Craniofacial Research has recognized the potential in salivary diagnostics and is fund-ing research that uses microfluidic and microelectromechanical systems for point-of-care testing for oral disease. These systems use small sample and reagent volumes to detect and measure proteins, DNA, RNA, bacteria, electrolytes, and other molecules in saliva. Researchers are developing lab-on-a-chip devices that will enable rapid and simultaneous detection of multiple biomarkers. Herr and colleagues[40] have been working on developing a portable device that can measure multiple biomarkers and are looking to characterize groups of proteins that are associated with different stages of periodontal disease.

As the detection of biomarkers in saliva improves, this biologic fluid may become an important part of periodontal diagnosis. Saliva-based diagnostic tests do not give tooth- or site-specific information, rather they give patient measures that may be

used in several ways. A salivary test may be used as a home screening tool that is based on a color change or simple color scale. A positive test result would indicate the need for a comprehensive dental evaluation. Alternatively, a salivary diagnostic test can be used as a quantitative in-office test that is used as part of the initial patient evaluation to assess the effectiveness of treatment and to monitor patient status during regular recall visits.

Genetic Testing

Although it is generally accepted that there is a genetic susceptibility to periodontitis,[41,42] the genes that confer susceptibility have not been definitively established. Several candidate genes have been proposed as putative risk or prognostic factors. In 1997, Kornman and colleagues[43] published a landmark study that found polymorphisms (interindividual differences in DNA sequences coding for 1 specific gene, giving rise to different functional and/or morphologic traits) in the gene for IL-1 to be a severity factor for periodontitis. A diagnostic test based on the carriage of the IL-1 polymorphism was developed and is commercially available. The test has not been widely adopted because it is of questionable clinical utility and it is unclear whether available data support its use. Since the publication of the Kornman study, polymorphisms in several genes have been proposed as risk markers for periodontitis.[44–46]

Overall, the literature on the role of individual polymorphisms is conflicting and difficult to interpret. Because periodontitis is a complex disease, it is likely that multiple genes contribute to disease susceptibility. A more comprehensive approach to the search for candidate genes should be considered. For example, Brett and colleagues[47] investigated multiple polymorphisms and their carriage among subjects with chronic and aggressive periodontitis. A relatively new method of genetic analysis, microarray technology, enables the analysis of thousands of genes at once. Using microarrays, investigators can examine which genes are differentially expressed in periodontitis. Demmer and colleagues[48] extracted messenger RNA from healthy and diseased gingival tissue and used microarrays to assess gene expression. Genes involved in apoptosis, antigen presentation, and antimicrobial humoral response were among those differentially expressed among diseased and healthy tissues. Microarray technology is a valuable tool for insight into the genetic susceptibility and pathobiology of periodontitis. As technologies evolve, it is easy to imagine the availability of a chair-side test for genetic susceptibility to periodontitis.

Newly Emerging Noninvasive Methods for Periodontal Diagnosis

Near infrared (NIR) spectroscopy is a test that provides a measure of oxygen saturation of the tissues.[35] Liu and colleagues[49] assessed multiple indices of periodontal inflammation using NIR spectroscopy and found that the tissue oxygenation at periodontitis sites was significantly decreased compared with that in patients with gingivitis and healthy controls. The investigators postulated that the tissue hypoxia reflects increased oxygen consumption that occurs with persistent inflammation. This finding is consistent with the fact that putative periodontal pathogens are generally anaerobic. NIR spectroscopy of the periodontal tissues was performed using a special intraoral probe.

Other noninvasive methods that have been suggested for imaging of the periodontal tissues include optical coherence tomography (OCT), acoustic microstreaming (ultrasonography), and cone beam computed tomography (CBCT). OCT creates high-resolution, cross-sectional images using a focused light beam that is scanned across the tissues of interest. Preliminary data have demonstrated that OCT could provide high-resolution, 3-dimensional imaging of periodontal soft and hard tissues.[35] Although

some researchers have considered ultrasonography as an imaging tool for the perio-dontium,[50,51] use of this technology for periodontal diagnosis is not developed.

Computed tomography (CT) enables cross-sectional, 3-dimensional analysis of mineralized tissue without distortion. CT scans are potentially informative for peri-odontal diagnosis; however, they are not used for this purpose because of the high cost of the machine, high levels of radiation, and relatively low resolution.[52] CBCT scan-ners on the other hand are much cheaper and impart much less radiation to the patient. Misch and colleagues[52] compared the accuracy of CBCT, periapical radiography, and direct measurement with a periodontal probe in measuring artificially created osseous defect in mandibles of dry skulls. Misch and colleagues suggested that CBCT has advantages over radiographs because it enables visualization of defects in 3 dimen-sions and visualization of buccal and lingual defects. A more recent in vitro study simi-larly reported better diagnostic and quantitative information on periodontal bone levels from CBCT compared with conventional radiography.[53] Additional research is indi-cated to determine the feasibility of using CBCT in the assessment of periodontitis.

SUMMARY

For all health care disciplines, clinical signs and symptoms play a critical role in estab-lishing a diagnosis. Diagnostic tests are used adjunctively to provide information that is not available from clinical findings. Such tests include microscopic evaluation of tissue (biopsy), evaluation of bodily fluids for markers of disease, imaging studies, and identification of specific microbial pathogens. Genetic analysis is also becoming an important area of study as the genetic contribution to specific diseases is elucidated.

The addition of a diagnostic test to patient evaluation is meaningful only if the test provides additional diagnostic information over what is obtained from the clinical assessment or if it helps guide the treatment more effectively. At present, diagnostic tests that aid in the assessment and management of patients with periodontitis are not a routine part of dental practice. Historically, this is likely because of the accessibility of the oral cavity to clinical and radiographic examination. Nevertheless, it has become clear that clinical and radiographic assessments fail to provide important information regarding the patient's disease, including whether there is the risk for transition from gingivitis to periodontitis, the disease is in a quiescent or destructive phase, adequate treatment has been provided, or there is the risk for disease recurrence. Development of a diagnostic test with appropriate sensitivity and specificity that provides this infor-mation would be invaluable in the management of patients with periodontal disease (Fig. 4).

One innovative approach to diagnosis and risk assessment for periodontal disease is the PreVisor software program (Previser Corporation, Mount Vernon, WA, USA), which is a risk assessment tool for patients with periodontal disease. Based on the longitudinal data that followed the progression of periodontal disease over a 15-year period, an algorithm that allows patients to be assessed for risk for periodontal destruction and tooth loss was developed.[54] The clinician enters specific information about each patient, including a history of dental care, smoking history, presence of diabetes mellitus, and existing dental or periodontal findings. These data are used to calculate the severity of disease and the risk for future disease progression. The disease state is expressed on a 0 to 100 scale, and a risk score is expressed on a 1 to 5 scale, with 1 as very low risk and 5 as very high risk. The severity of the patient's periodontitis and the risk score can be plotted against time (Fig. 5). Thus far, the advantages of using this risk assessment tool have not been fully explored.
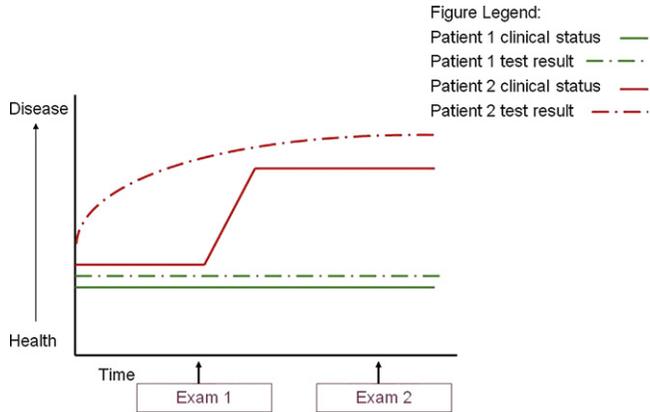
Fig. 4. The ideal diagnostic test would be able to predict the development of disease before clinical signs and symptoms. At the first examination, patients 1 (*green*) and 2 (*red*) have similar levels of disease; however, the test results are different. At the second examination, the level of disease of patient 1 has remained the same, whereas that of patient 2 has worsened. The test result remains elevated for patient 2.
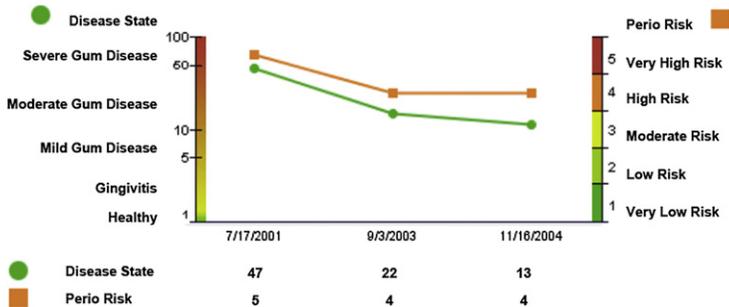

Fig. 5. Sample report from Previser.com. (Available at: http://www.previser.com/documents/reports/f39000c7-81a1-45fd-bb1f-f45d99245dbe_pf.html. Accessed May 26, 2010; with permission.)

With the enormous focus on the potential impact of periodontal disease and oral inflammation on diseases and disorders at distant sites, a diagnostic test based on the presence of important inflammatory mediators may offer a quantitative measure of the oral inflammatory burden. Such a test would help guide the clinician concerned with the effect of periodontal inflammation on morbidity associated with cardiovascular or cerebrovascular disease, adverse obstetric outcomes, diabetes mellitus, and other disorders. The test could be used to assess whether periodontal therapy has successfully reduced this risk.

New approaches to periodontal diagnosis, including biochemical tests and the application of devices that assess the periodontal tissues, have been shown to provide the clinician with information not available by traditional means. The widespread application of these tests will depend on several factors, including ease of use, cost, the strength of the data supporting the value of the tests, and the ability of the test to aid in patient management. Use of validated diagnostic tests for periodontal disease will also require a paradigm shift in the approach of the dental profession to disease management. Dentists will spend more time on the diagnostic phase of treatment, and the result will be better treatment outcomes.

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