

Chapter 5

Current Trends in Periodontal Diagnosis & Disease Recognition - A Perspective from the USA

L.L. Cabanilla

School of Dentistry, University of Detroit-Mercy, United States of America

Introduction

Arriving at an accurate diagnosis is one of the most important tasks a prudent clinician has to carry out as this is what a treatment plan will be based upon. It is therefore not surprising to see the amount of time a periodontist allots in examining a patient. One usually starts with responsibly gathering and/or updating medical, dental and other pertinent histories of the patient. After this process, several diagnostic techniques are utilized to acquire the necessary data to successfully determine the patient's disease category. Despite the tremendous increase in current knowledge with regards to the etiology and pathogenesis of periodontal diseases, the classification and diagnosis of periodontal diseases are still largely based on traditional clinical assessments (Armitage 1995, Armitage 1996).

A clinical diagnosis of periodontitis is still based on clinical attachment loss and bone loss. This information reflects past periodontal destruction but does not provide any information regarding current disease activity or susceptibility to further periodontal destruction. Most clinicians assign a diagnosis of "periodontitis" to inflamed sites with clinical and radiographic bone loss. This

diagnosis is made under the assumption that such sites are at increased risk for disease progression and thus would require active periodontal therapy. Here lies the reason behind the continued quest for developing advanced diagnostic tests.

The parameters that are currently used, such as pocketing and bleeding on probing, have inherent shortcomings in determining risk for further attachment loss, and in the case of probing depths, there is also the question of accuracy. Although Lang (Lang *et al* 1986) reported that sites that bled on probing at several visits had a higher probability (30%) of losing attachment than those that bled at one visit or did not bleed, well-controlled studies failed to demonstrate a significant correlation between bleeding on probing and other clinical signs and subsequent loss of attachment (Badersten *et al* 1985, Haffajee *et al* 1983). The periodontal probe is still the most widely used tool for the assessment of clinical attachment loss. Unfortunately, it carries problems in sensitivity and reproducibility. Probing is very subjective since it can be affected by several factors such as degree of tissue inflammation, probing technique, force, size of the probe, angle of insertion and precision of the probe calibration (Listgarten *et al* 1976).

Over the past decade, several developments have occurred in the diagnosis of periodontal disease, the most noticeable of which is the change in the classification of the disease. This manuscript highlights some important aspects of the revised classification. The reader is referred to the *Annals of Periodontology* for a more detailed discussion of each category, the revisions and the rationale for the changes (American Academy of Periodontology 1999).

Revised Classification of Periodontal Diseases and Conditions (1999 International Workshop)

The revised classification of periodontal diseases includes seven general types of plaque-induced periodontal diseases namely; gingivitis, chronic periodontitis, aggressive periodontitis, periodontitis as a manifestation of systemic diseases, necrotizing periodontal diseases, abscesses of the periodontium and periodontitis associated with endodontic lesions. It has a detailed classification of gingival diseases and lesions that are either plaque-induced or not primarily associated with dental plaque. This section also includes other gingival lesions and disorders that affect the gingiva.

The term “Adult Periodontitis” was replaced with “Chronic Periodontitis” mainly in order to eliminate the restrictive use of age of onset as a major factor in determining diagnosis. In the new classification, the nonspecific term “Chronic Periodontitis” is characterized by the fact that it is most prevalent in adults, but can also occur in children (Papapanou 1996). It is said to progress in a slow to moderate rate (Loe *et al* 1986, Papapanou *et al* 1989), but may have periods of rapid progressions (Socransky *et al* 1984, Jeffcoat and Reddy 1991). It is consistent with a variable microbial pattern and can be associated with local predisposing

factors as well.

“Early-onset periodontitis” was discarded since it is assumed that one has temporal knowledge of when the disease started. In addition, the age by which an “adult” is defined in the old classification is considered arbitrarily chosen. It is therefore recommended that a diagnosis of “Aggressive Periodontitis” should be based on clinical, radiographic, historical and laboratory findings. It is characterized by the following: rapid attachment loss and bone destruction, generally clinically healthy individuals, familial aggregation and the amounts of microbial deposits are inconsistent with the severity of periodontal destruction (American Academy of Periodontology 1999). Although the microbial etiology of the early onset syndromes has been primarily associated with *A. actinomycetemcomitans* (Slots *et al* 1980), recent studies have indicated that differences exist among various ethnicities (Lee *et al* 2003, Dogan *et al* 2003, Takeuchi *et al* 2003).

Advances in Traditional Diagnostic Methods

The increased understanding in the etiology and pathogenesis of periodontal diseases underscores the importance of advancing diagnostic techniques. Since the most common means of determining clinical attachment loss is through probing, attempts have been made to improve its accuracy and reproducibility. One of which is the development of the computer linked, controlled-force electronic periodontal probes. Although electronic probes offer the advantage of controlled insertion forces, automatic recording of data into a computer (Greenstein 1997, Armitage 1996) and better resolution (Jeffcoat and Reddy 1991), there are still several drawbacks associated with it. Electronic probing requires more time to use, is more costly, can be uncomfortable and may

potentially underestimate deep probing depths (Perry *et al* 1994). It has also been reported that in order to improve reproducibility of clinical measurements, a double pass method (measuring each site twice) should be utilized (Osborn *et al* 1990).

Significant advances have also been made in radiographic imaging to aid in periodontal diagnosis. Conventional radiographs have been shown to routinely underestimate the amount of bone loss (Greenstein, 1997) It also requires 30-50% bone mineral resorption before changes can be detected by routine radiographic examination (Jeffcoat and Reddy 1991). The introduction of subtraction radiography to the dental field, allowed detection of changes in bone density as low as 5%. Both hardware and software have been improving and will continue to do so in attempts to correct some of the problems initially associated with subtraction radiography such as; subtle differences in contrast, projection geometry and other repeatability errors (Hausmann *et al* 1994). The use of digital radiography in periodontal diagnosis has tremendous potential especially since studies have demonstrated an 80% agreement between probing and radiographic methods in identifying sites that have lost attachment (Jeffcoat 1992, Hausmann *et al* 1994).

Supplemental Diagnostic Tests

Supplemental diagnostic tests have been developed and investigated since there is an enormous potential in using the results to successfully identify therapeutic targets, monitor the response to therapy, identify sites at high risk for progression and to assist in determining a patient specific recall. As of to date, these tests can be used to detect the presence of: substances associated with putative pathogens, host derived enzymes, tissue breakdown products and inflammatory

mediators. There are numerous examples of supplemental diagnostic tests, however, this paper will only mention some of the more commonly used clinical tests.

The most common means of identifying the microbial composition of plaque samples is through bacterial culturing. The main advantage of this method is that one can obtain relative and absolute counts of the cultured species. It is also the only *in vitro* method able to assess for antibiotic susceptibility of the microbes. The microbial composition of subgingival plaque has always been a point of interest in the diagnosis and treatment planning for periodontal disease. It has been demonstrated that progressing periodontitis is associated with certain periodontopathogenic bacteria (Machtei *et al* 1997). However, a clinician should not rely on the microbial testing alone to determine diagnosis and individualized treatment, since it has also been determined that the presence or absence of specific bacteria cannot discriminate subjects from different disease categories (Mombelli *et al* 2002). In addition, another study concluded that the presence of putative pathogens can only predict future attachment loss in only 20% of the sites. Their absence was considered a better predictor of no further attachment loss than their presence was of disease progression (Wennstrom *et al* 1987).

Another means by which bacterial identification can be accomplished is through an enzymatic test such as the BANASCAN. This enzymatic assay provides a rapid chair side test (15 minutes) to detect *Porphyromonas gingivalis*, *Treponema denticola* and *Tannerella forsythensis* (formerly *Bacteroides forsythus*).

Other supplemental diagnostic tests are designed to provide information regarding the ongoing inflammatory process. One example is the Periocheck, which tests for collagenase as a marker of inflammation. Compared with healthy sites, locations with gingivitis or

periodontitis had higher levels of collagenase (Larivee *et al* 1986). However, there are no data showing a relationship between the level of collagenase and progressive periodontitis. In addition, this test cannot differentiate between gingivitis and periodontitis.

One of the most comprehensively studied host derived enzyme associated with periodontal disease is aspartate amino transferase (AST), which is released by dead or dying cells. It has been widely utilized to detect heart damage after a myocardial infarction as well liver damage during hepatitis. Gingival crevicular fluid samples taken from sites with severe gingival inflammation demonstrated a marked increase in AST levels (Chambers *et al* 1991). It is however, unable to discriminate between sites with severe inflammation but with no attachment loss from sites that are losing attachment. It remains to be seen whether this test offers some advantage over existing clinical measures of disease (Magnusson *et al* 1996, Persson *et al* 1995).

It has been well documented that susceptibility to periodontal diseases is highly variable and depends on host responses to pathogens (Tonetti 1994, Ishikawa *et al* 1997, Offenbacher 1996). Thus, attempts have been made to develop a reliable test for host-based susceptibility. The only test for host susceptibility that is available to practitioners is a genetic test for polymorphisms in the interleukin gene cluster (PST) (Kornman *et al* 1997). Approximately 30% of Caucasians are positive for a composite genotype of IL-1A and IL-1B polymorphisms. People who test positive for this composite genotype are said to be at increased risk of the following: bleeding on probing (Lang *et al* 2000), severe chronic periodontitis (Kornman *et al* 1997), tooth loss (McGuire and Nunn 1999), and attachment loss after therapy (De Sanctis and Zucchelli 2000) or increased secretion of IL-1 β (Pociot *et al* 1992). However, other studies

have shown conflicting results (Cattabriga *et al* 2001, Papapanou *et al* 2001, Ehmke *et al* 1999, Mark *et al* 2000). The prevalence of this genotype also varies among different populations (Armitage *et al* 2000), thus may be of little value in determining the risk for susceptibility to periodontitis.

Conclusion

Despite the astounding improvements in both traditional and supplemental diagnostic techniques, the majority of clinicians still rely heavily on basic clinical and radiographic assessments gathered from using a periodontal probe and conventional radiographs. Recent developments in diagnostic techniques, without a doubt, have significant potential and scientific merit. However, several of these advancements still require definition of their clinical utility and cost-effectiveness in order to promote a more widespread use. Addressing these issues and continuing the quest for diagnostic techniques that will not only provide clinicians with current periodontal disease status but also future risk of periodontal breakdown that are both patient and site-specific, will have a tremendous impact on the way we diagnose periodontal diseases and ultimately, how we develop our treatment plan.

References

- American Academy of Periodontology. 1999 International Workshop for Classification of Periodontal Diseases and Conditions. Chicago: *Ann Periodontol* 1999;4:1-112.
- American Academy of Periodontology. 1999 International Workshop for Classification of Periodontal Diseases and Conditions. Consensus Report: Aggressive Periodontitis Chicago: *Ann Periodontol* 1999;4:53.
- Armitage GC, Wu Y, Wang HY, Sorrell J, di Giovine FS, Duff GW. Low prevalence of a periodontitis-

- associated interleukin-1 composite genotype in individuals of Chinese heritage. *J Periodontol* 2000;71:164-171.
- Armitage GC. Clinical evaluation of periodontal diseases. *Periodontol 2000* 1995;7:39-53.
- Armitage GC. Manual periodontal probing in supportive periodontal treatment. *Periodontol 2000* 1996;12:33-39.
- Armitage GC. Periodontal Diseases: Diagnosis. *Ann Periodontol* 1996;1:37-215.
- Badersten A, Nilveus R, Egelberg J. Effect of non-surgical periodontal therapy. VII. Bleeding, suppuration and probing depth in sites with probing attachment loss. *J Clin Periodontol* 1985;12:432-440.
- Cattabriga M, Rotundo R, Muzzi L, Nieri M, Verrocchi G, Cairo F, Pini Prato G. Retrospective evaluation of the influence of the interleukin-1 genotype on radiographic bone levels in treated periodontal patients over 10 years. *J Periodontol* 2001;72:767-773.
- Chambers DA, Imrey PB, Cohen RL, Crawford JM, Alves ME, McSwiggin TA. A longitudinal study of aspartate aminotransferase in human gingival crevicular fluid. *J Periodont Res* 1991;26:65-74.
- De Sanctis M, Zucchelli G. Interleukin-1 gene polymorphisms and long term stability following guided tissue regeneration therapy. *J Periodontol* 2000;71:606-613.
- Dogan B, Antinheimo J, Cetiner D, Bodur A, Emingil G, Buduneli E, Uygur C, Firatli E, Lakio L, Asikainen S. Subgingival microflora in Turkish patients with periodontitis. *J Periodontol* 2003;74:803-814.
- Ehmke B, Kress W, Karch H, Grimm T, Klaiber B, Flemmig TF. Interleukin-1 haplotype and periodontal disease progression following therapy. *J Clin Periodontol* 1999;26:810-813.
- Greenstein G. Contemporary interpretation of probing depth assessments: Diagnostic and therapeutic indications. *J Periodontol* 1997;68:1194-1205.
- Haffajee AD, Socransky SS, Goodson JM. Clinical parameters as predictors of destructive periodontal activity. *J Clin Periodontol* 1983;10:257-265.
- Haffajee AD, Socransky SS. Microbial etiological agents of destructive periodontal diseases. *Periodontol 2000* 1994;5:78-111.
- Hausmann E, Allen K, Norderyd J, Ren W, Shibly O, Machtei E. Studies on the relationship between changes in radiographic bone height and probing attachment. *J Clin Periodontol* 1994;21:128-132.
- Ishikawa I, Nakashima K, Koseki T. Induction of the immune response to periodontopathic bacteria and its role in the pathogenesis of periodontitis. *Periodontol 2000* 1997;14:70-111.
- Jeffcoat MA, Reddy MS. Comparison of probing and radiographic methods for detection of periodontal disease progression. *Curr Opin Dent* 1991;1:45-51.
- Jeffcoat MK, Reddy MS. Progression of probing attachment loss in adult periodontitis. *J Periodontol* 1991;62:185-189.
- Jeffcoat MK. Radiographic methods for the detection of progressive alveolar bone loss. *J Periodontol* 1992;63:367-72.
- Kornman KS, Crane A, Wang HY, di Giovine FS, Newman MG, Pirk FW, Wilson TG Jr, Higginbottom FL, Duff GW. The interleukin-1 genotype as a severity factor in adult periodontitis. *J Clin Periodontol* 1997;24:72-77.
- Kornman KS, Page RC, Tonetti MS. The host response to the microbial challenge in periodontitis. Assembling the players. *Periodontol 2000* 1997;14:33-53.
- Lang NP, Joss A, Orsanic T, Gusberti FA, Siegrist BE. Bleeding on probing. A predictor for the progression of periodontal disease? *J Clin Periodontol* 1986;13:590-596.
- Lang NP, Tonetti MS, Suter J, Sorrell J, Duff GW, Kornman KS. Effect of interleukin-1 gene polymorphisms on gingival inflammation assessed by bleeding on probing in a periodontal maintenance population. *J Periodont Res* 2000;35:102-107.
- Larivee J, Sokek J, Ferrier JM. Collagenase and collagenase inhibitor activities in crevicular fluid of patients receiving treatment for localized juvenile periodontitis. *J Periodont Res* 1986;21:702-714.
- Lee JW, Choi BK, Yoo YJ, Choi SH, Cho KS, Chai

- JK, Kim CK. Distribution of periodontal pathogens in Korean aggressive periodontitis. *J Periodontol* 2003; 74:1329-1335.
- Listgarten MA, Mao R, Robinson PJ. Periodontal probing and the relationship of the probe tip to periodontal tissues. *J Periodontol* 1976;47:511-513.
- Loe H, Anerud A, Boysen H, Morrison E. Natural history of periodontal diseases in man. Rapid, moderate and no loss of attachment in Sri Lankan laborers 14-46 years of age. *J Clin Periodontol* 1986;13:431-440.
- Machtei EE, Dunford R, Hausmann E, Grossi SG, Powell J, Cummins D, Zambon JJ, Genco RJ. Longitudinal study of prognostic factors in established periodontitis patients. *J Clin Periodontol* 1997;24:102.
- Magnusson I, Persson RG, Page RC, DeRouen TA, Crawford JM, Cohen RL, Chambers DA, Alves ME, Clark WB. A multicenter clinical trial of a new chairside test in distinguishing between diseased and healthy periodontal sites. II. Association between site type and test outcome before and after therapy. *J Periodontol* 1996;67:589-596.
- Mark LL, Haffajee AD, Socransky SS, Kent RL Jr, Guerrero D, Kornman K, Newman M, Stashenko P. Effect of the interleukin-1 genotype on monocyte IL-1b expression in subjects with adult periodontitis. *J Periodont Res* 2000;35:1172-1177.
- McGuire MK, Nunn ME. Prognosis versus actual outcome. IV. The effectiveness of clinical parameters and IL-1 genotype in accurately predicting prognoses and tooth survival. *J Periodontol* 1999;70:49-56.
- Mombelli A, Casagni F, Madianos PN. Can presence or absence of periodontal pathogens distinguish between subjects with chronic and aggressive periodontitis? A systematic review. *J Clin Periodontol* 2002;29 Suppl 3:10-21; discussion 37-38.
- Offenbacher S. Periodontal diseases: Pathogenesis. *Ann Periodontol* 1996;1:821-878.
- Osborn J, Stoltenberg J, Huso B, Aepli D, Pihlstrom B. Comparison of measurement variability using a standard and constant force periodontal probe. *J Periodontol* 1990;61:497-503.
- Papapanou PN, Neiderud AM, Sandros J, Dahlen G. Interleukin-1 gene polymorphism and periodontal status. A case-control study. *J Clin Periodontol* 2001;28:389-396.
- Papapanou PN, Wenstrom JL, Grondahl K. A 10-year retrospective study of periodontal disease progression. *J Clin Periodontol* 1989;16:403-411.
- Papapanou PN. Periodontal diseases: Epidemiology. *Ann Periodontol* 1996;1:1-36.
- Perry DA, Taggart EJ, Leung A, Newburn E. Comparison of a conventional probe with electronic and manual pressure regulated probes. *J Periodontol* 1994;65:908-913.
- Persson GR, Alves ME, Chambers DA, Clark WB, Cohen R, Crawford JM, DeRouen TA, Magnusson I, Schindler T, Page RC. A multicenter clinical trial of PerioGard in distinguishing between diseased and healthy periodontal sites. *J Clin Periodontol* 1995;22:794-803.
- Pociot F, Molvig J, Wogensen L, Worsaae H, Nerup J. A Taq1 polymorphism in the human interleukin-1 beta (IL-1b) gene correlates with secretions in vitro. *Eur J Clin Invest* 1992;22:396-402.
- Slots J, Reynolds HS, Genco RJ. Actinobacillus actinomycetemcomitans in human periodontal disease: a cross-sectional microbiological investigation. *Infect Immun* 1980;29:1013-1020.
- Socransky SS, Haffajee AD, Goodson JM et al. New concepts of destructive periodontal disease. *J Clin Periodontol* 1984;11:21-32.
- Takeuchi Y, Umeda M, Ishizuka M, Huang Y, Ishikawa I. Prevalence of periodontopathic bacteria in aggressive periodontitis patients in a Japanese population. *J Periodontol* 2003;74:1460-1469.
- Tonetti MS. Etiology and pathogenesis. In: *Proceedings of the 1st European Workshop on Periodontology*. 1994:54-89.
- Wennstrom JL, Dahlen G, Svensson J, Nyman S. Actinobacillus actinomycetemcomitans, Bacteroides gingivalis and Bacteroides intermedius: Predictors of attachment loss? *Oral Microbiol Immunol* 1987;2:158-162.