CHAPTER 4. DISEASE ACTIVITY

Section 1. Bleeding Upon Probing

Assessment of bleeding upon probing (BOP) is an important part of the periodontal examination. Bleeding has been demonstrated in clinical and histological studies to be a more sensitive sign of gingival inflammation than visual alterations. Meitner et al. (1979) examined 6,990 gingival surfaces visually, then probed for bleeding 1, 2, and 3 months following prophylaxis. At the first exam, 1,678 sites (24%) demonstrated a combined absence of visual inflammation and BOP. One month later, only 766 of these surfaces were still healthy. Data from surfaces that had changed since the first examination showed that there were a significantly greater number of surfaces which bled upon probing compared to either a color change only or a combination of color change and BOP. The authors concluded that gingival indices based upon BOP are more accurate than those using visual changes.

BLEEDING INDICES

Interdental bleeding following stimulation with wooden interdental cleaners forms the basis of the Eastman Interdental Bleeding Index (EIBI), while bleeding following sweeping a probe in the sulcus from the line angle to the interproximal contact forms the basis of the Papilla Bleeding Index (PBI). Caton et al. (1988) compared visual evaluation, PBI, and EIBI in 82 subjects not currently undergoing periodontal therapy. Visual signs of inflammation were seen at 71% of the sites, EIBI was positive at 65%, and PBI was positive at 42% of the sites. At sites rated visually as non-inflamed, almost twice as many inflammatory lesions were detected by EIBI versus PBI. The authors concluded that the Eastman Interdental Bleeding Index was a more reliable clinical indicator for detecting interdental inflammation than the Papilla Bleeding Index.

HISTOLOGY OF BLEEDING SITES

Histological studies have verified the presence of significantly more inflammation in the bleeding sites compared to non-bleeding sites. Greenstein et al. (1981) evaluated 60 gingival biopsies for visual presence or absence of bleeding after probing using a pressure-controlled probe set at 25g. Sites which bled on probing exhibited a significantly greater percentage (28.7% versus 19.9%) of cell rich-collagen poor connective tissue than non-bleeding sites without an increase in blood vessel lumens. Other studies have characterized the histopathological features of active periodontal lesions using bleeding as one of the criteria of disease activity. Davenport et al. (1982) found that the percentage of infiltrated connective tissue was consistently larger in bleeding lesions with suppuration than in non-bleeding lesions with suppuration. Significant differences were observed between bleeding and non-bleeding sites in terms of the mean percent volume occupied by plasma cells (68% versus 24%) and mononuclear cells (5.5% versus 22%). The pocket epithelium of bleeding lesions demonstrated thinned and ulcerated areas along with proliferation of rete pegs.

Passo et al. (1988) examined the histological features of bleeding sites with and without suppuration. They reported that the percentage of collagen-poor area in bleeding (but not suppurating) sites was similar (27.7%) to the above finding of Greenstein et al. (1981), but was higher in the suppurating area. However, suppuration was not always associated with extensive inflammation, and intense inflammation was also seen in non-suppurating sites. Thus, suppuration did not appear to be a specific indicator of a destructive periodontal lesion. Reinhardt et al. (1988) examined the lymphocyte subpopulation in periodontal active lesions characterized by bleeding and progressive attachment loss.

Polymorphonuclear leukocytes (PMNs) were seen only in 3.7% of the sulcular third of stable sites and in 5.6% to 8.3% of the sulcular third of active sites. Lymphocyte density was greater in the sulcular thirds than in the oral thirds. When compared to stable sites, active sites showed an increased number of plasma cells and a reduced T/B ratio. The T helper/T suppressor ratio did not vary significantly between blood and gingival tissue of any disease group but seemed to follow a trend toward lower numbers of T helper cells. These results indicated that active periodontal lesions characterized by bleeding and attachment loss displayed elevated B cell population and abnormal immune regulation possibly involving the T helper cell subset.

Badersten et al. (1990) studied the longitudinal effects of non-surgical therapy on nonmolar teeth. The diagnostic value of clinical scores of plaque, bleeding, suppuration, and probing depth in predicting probing attachment loss during the maintenance phase was investigated. A combination of linear regression and end-point analysis was used to determine probing attachment loss over the 0 to 60 month period. The authors found that all of the investigated scores were associated with clinical attachment loss and that improved diagnostic predictability was noted with an increased length of time in recording scores. The positive predictive value (Pv+) of accumulated plaque or accumu-

lated bleeding scores reached a maximum of about 30%, corroborating the findings of Lang et al. (1986). A residual probing depth of ≥ 7 mm had about a 50% Pv+ while increased probing depths ≥ 1.0 mm reached 80% Pv+ after 60 months. This led to the conclusion that an increase in probing depth, as opposed to the presence of bleeding on probing, was most valuable in predicting probing attachment loss (our "gold standard" of disease activity?). The use of lower probing pressure (< 0.75 Newton [N]) used in this study) and selection of only those sites with marked bleeding might have improved the predictive value of bleeding upon probing. Suppuration only reached a maximum of 20% positive predictive value, probably because it was a rare occurrence.

Claffey et al. (1990) did a parallel study which included molars. For 42 months following periodontal therapy, plaque, bleeding, suppuration, and probing depth were recorded for 17 subjects. A probing force of 0.50 N was used and 4 of the subjects received no subgingival instrumentation during the 42 months of maintenance. Similar results to Badersten et al. (1990) were obtained: 1) accumulated plaque scores had low Pv+; 2) 41% of sites that bled on probing at 75% or more of the examinations between 3 to 42 months had undergone probing attachment loss; 3) suppuration on probing reached a Pv+ of 40 to 50% but was not a frequent finding; and 4) increase in probing depth of greater than 1 mm reached a Pv+ of 68% at 42 months. The combination of increasing probing depth with bleeding frequency at 75% or more of examinations yielded a predictability score of 87% at 42 months. The best positive predictive value was found using a combination of bleeding upon probing and an increased probing depth of 1 mm or more. A comparison of this group with that of Badersten et al. (1990) revealed higher plaque and bleeding scores in this group of patients. Furcations showed the highest incidence of probing attachment loss. The authors conclude that longer observation periods are needed if the commonly used clinical signs such as BOP and suppuration are to reach meaningful diagnostic values.

In another longitudinal study of maintenance patients, Kaldahl et al. (1990) investigated the relationship of bleeding, suppuration, and presence of supragingival plaque to attachment loss during the second and third years following active therapy which included either coronal scaling, root planing, modified Widman surgery, or flap with osseous resection. A probing force of 25 grams was used to detect bleeding on probing, while an increased pressure of 50 grams was used to record the probing attachment level. They found that as the frequency of bleeding on probing increased, the sensitivity decreased (0.82 to 0.15), the specificity increased (0.20 to 0.88), and positive and negative predictive values remained constant (Pv+ = .15–.18; Pv− = .86). For suppuration, sensitivity was extremely low but specificity was nearly 1.0 while positve predictive value increased (from 0.27 to 0.50) because of the low frequency of suppuration in the population. For supragingival plaque, as the frequency of presence of plaque increased, the sensitivity decreased (0.67 to 0.15), the specificity increased (0.40 to 0.87), and the positive and negative predictive values remained constant (Pv+ = .16–.17; Pv− = .87–.86). The authors conclude that bleeding and plaque are not good prognosticators while suppuration is a weak prognosticator over the 2-year maintenance period. The bleeding symptom associated with a non-aggressive state (gingivitis) is probably much more frequent and therefore may mask bleeding associated with an aggressive inflammatory state (periodontitis).

Greenstein and Caton (1990) published a critical assessment of periodontal disease activity concepts. Several important issues were addressed, including the fact that at any given moment, there is no practical clinical test to determine if disease activity is occurring. Longitudinal monitoring is required. If a 3 mm increase in probing attachment loss is used, as selected by Haffajee et al. (1983), as equivalent to 3 times the standard deviation association with difference between replicate probing measurements, and rounded upward, clinically significant loss may occur before initiating therapy. In this instance, the sensitivity of the test will be low and the specificity high, resulting in a high number of false-negatives and possible undertreatment. Conversely, if a 1 mm increase in probing depth is used as the standard for disease activity, variability in probing accuracy may produce significant numbers of false-positive results, with subsequent overtreatment.

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Section 2. Gingival Crevicular Fluid

DEFINITION

Gingival Crevicular Fluid (GCF): Tissue fluid that seeps through the crevicular epithelium. It is increased in the presence of inflammation.

It has been reported that the flow of GCF can be detected a few days before other clinical signs of inflammation are evident. Crevicular fluid appears during altered states of vascular permeability which may accompany gingival inflammation (Abbott and Caffesse, 1977). On this basis, its measurement has been proposed as an indicator of periodontal disease activity.

METHODS OF COLLECTION AND MEASUREMENT

GCF may be collected by two basic techniques: filter paper strips and capillary micropipettes. Measurements are accomplished by ninhydrin stain assessment or by use of the Periotron (measures filter strip wetness impedance). GCF collection methods using filter paper strips may be divided into the intracrevicular and extracrevicular techniques:

Intracrevicular

The Brill (1962) technique includes placement of a filter strip into the gingival sulcus until resistance is felt. After 3 minutes in place, the strips are removed, dried, and stained with ninhydrin (stains proteins). Löe and Holm-Pedersen (1965) proposed a method involving minimal irritation to the gingival sulcus, in which the paper strip is placed at the entrance of the crevice.

Extracrevicular

Brill and Krasse (1959) and Löe and Holm-Pedersen (1965) also employed an extracrevicular collection technique. The strips were closely adapted to the buccal surfaces of the teeth, across the gingival margin and onto the attached gingiva.

GCF collected with capillary pipettes permits the measurement of fluid volume; however, to assure accurate measurement, relatively large volumes have to be collected. This has the disadvantage of requiring a considerable amount of time in order to obtain a sample, rendering the technique impractical for clinical use. A more precise method is the use of micropipettes which determines the actual volume of GCF (Kaslick, 1970).

The Periotron employs an electronic transducer to measure GCF. This instrument measures electrical capacitance, as the insulating properties of the filter paper strip vary according to the quantity of fluid absorbed within the strip (Suppipat, 1977). The instrument evaluates the flow of current based on the wetness of the strip (impedance). A digital readout registers the area wetted and is indicative of the volume of fluid collected on the paper strip.

QUANTITATIVE ASSESSMENT

Hancock et al. (1979) studied crevicular fluid and gingival inflammation, reporting weak correlations between GCF and clinical or histologic parameters. They concluded that the quantity of fluid may have potential as a clinical indicator of presence but not severity of gingival inflammation. Shapiro et al. (1979) found no statistically significant correlation between the amount of GCF and the histological degree of inflammation. However, there was a positive correlation between the GCF and the clinical assessment of inflammation. Cimasoni (1983), in reviewing this subject, noted that a "positive correlation was always found between the clinical appreciation of gingival inflammation and the amount of gingival fluid." He also reported that the correlation between gingival crevicular fluid flow and histological inflammatory changes was poor.

It is generally agreed that gingival crevicular fluid reflects vascular permeability and thus gingival inflammation. It may indicate the presence of gingival inflammation but there is no evidence showing that it can predict periodontal breakdown or disclose the degree of inflammation. Factors such as circadian periodicity, hormonal alteration, and differences in collection technique may provide sources of error, making interpretation of findings difficult.

QUALITATIVE ASSESSMENT

Hydrogen sulfide is a cytotoxic metabolite of bacterial origin. Solis-Gaffer et al. (1980) proposed that the level of GCF hydrogen sulfide could be an indication of periodontal disease and used a quantitative method to determine the production of hydrogen sulfide from 240 GCF samples collected with paper strips. A strong positive correlation between hydrogen sulfide production and the degree of gingival inflammation was shown. Horowitz and Folke (1973) analyzed hydrogen sulfide production in patients with gingival health (probing depth 2 mm or less), periodontitis (PD $>$ 4 mm), and surgical treatment (pocket elimination). Results indicated that hydrogen sulfide generation increased with sulcular depth and that toothbrushing failed to reduce hydrogen sulfide production in periodontal pockets. These findings appear to favor pocket reduction surgical procedures.

PMN RESPONSE TO CHEMOTACTIC CHALLENGE

Neutrophils (PMNs) are the predominant cell type in the gingival sulcus during the initiation and progression of per-

iodontal disease (Attstrom, 1971; Page and Schroeder, 1976). These cells are responsive to a number of chemotactic factors produced by the gingival microflora and by activated products of the complement system. Most studies have indicated that neutrophils play a protective role in periodontal tissues (Van Dyke et al., 1980). PMN dysfunction (especially defective chemotaxis) has been observed in many cases of severe periodontal disease. It has been shown that PMNs migrate through the gingival connective tissue and junctional epithelium into the gingival sulcus (Van Dyke et al., 1980). Monitoring crevicular PMNs has been advocated as a means of assessing periodontal disease activity.

Golub et al. (1981) described a technique (the sulcular technique) for assessing neutrophil chemotaxis in vivo. Singh et al. (1984) utilized this method to monitor patients with gingivitis, chronic periodontitis, and LJP. In gingivitis and chronic periodontitis, the response to the chemotactic agent (casein) was similar to normal subjects except that the peak cell count was greater. LJP patients showed an abnormal response with two leukocyte peaks compared with a single peak in controls. This in vivo assay of neutrophil response could be useful in determining susceptibility to periodontal breakdown and individual treatment regimens.

Lamster et al. (1985) studied enzymatic profiles in gingival fluid. Arylsulfatase and B-glucuronidase were selected as indicators of the breakdown of connective tissue ground substance. Evidence suggests that ground substance integrity is important for maintenance of the collagen component of the connective tissue. The authors found that the ground substance degrading enzyme activity in crevicular fluid plateaued 2 to 3 weeks after the initiation of experimental gingivitis. The authors observed that this peak in enzyme activity corresponded with the established lesion of gingivitis. Similarly, Oshrain et al. (1984) found greater arylsulfatase volume activity in patients with gingivitis and periodontitis than in normal subjects.

Elevation of serum levels of aspartate aminotransferase is frequently used as a diagnostic indicator of myocardial infarction and as a quantitative measure of the extent of tissue destruction. Chambers et al. (1984) hypothesized that cell death during active periodontitis would release aspartate aminotransferase into the crevicular fluid. They studied the levels of this enzyme in GCF in a ligature-induced periodontitis model in the beagle dog. Results showed significant increases in aspartate aminotransferase levels 2 weeks after ligation. The authors suggested that this peak represented the acute destructive episodes of active periodontal disease.

Lamster et al. (1988) performed a longitudinal evaluation of GCF levels of B-glucuronidase, arylsulfatase, and lactate dehydrogenase in patients with chronic adult periodontitis. They determined that increased GCF levels of B-glucuronidase (a marker for primary granule release from PMNs) was a very strong predictor of impending clinical attachment loss. In contrast, neither arylsulfatase nor lactate dehydrogenase was able to predict attachment loss.

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Section 3. Bacterial Flora

In recent years, traditional clinical assessments of disease progression have been questioned. Haffajee et al. (1983) demonstrated that common clinical assessments of inflammation (gingival erythema, bleeding on probing, and

suppuration) were poor predictors of progression in an untreated population. Similar results were reported by Badersten et al. (1985) and by Lang et al. (1986) who showed that repeated measurements (4/4) of bleeding on probing at maintenance visits was followed by a loss of attachment at only 30% of possible sites. These and other studies suggest that since it is impossible to clinically detect sites which are actually undergoing attachment loss, treatment of all inflamed sites is necessary.

Although bacterial plaque has long been accepted as the primary etiology of periodontal disease, there is some debate over the exact etiologic mechanism of disease activity. The "non-specific plaque" hypothesis proposes that the etiologic mechanism in periodontitis is related to total plaque mass, therefore, reducing the amount of plaque will curtail disease activity. According to the "specific plaque hypothesis," periodontal disease activity is related to specific bacterial species and their products. The latter theory suggests that identification and eradication of putative periodontopathogens should be the goal of treatment. Microbial monitoring is based on the specific plaque hypothesis. In reality, mechanisms mediating periodontal diseases probably relate to both hypotheses or theories. The non-specific theory may be more relevant to chronic adult periodontitis, whereas the specific plaque theory may be more applicable to distinctive periodontal disorders; e.g., localized juvenile periodontitis (Listgarten, 1988).

Since clinical tests are currently unable to provide adequate disease assessment and predictability and since specific microbiologic species seem to be related to disease activity, microbiologic tests were sought as viable alternatives or adjuncts to clinical parameters.

COMPOSITION OF SUBGINGIVAL FLORA TO DISEASE ACTIVITY

Analysis of Bacterial Morphotypes (Darkfield Microscopy [DFM])

With DFM, light enters the microscopic field peripherally so that microorganisms are obliquely illuminated and glow against a dark background.

Listgarten and Hellden (1978) described a technique for classifying bacterial samples on the basis of constituent shape, size, and motility. Two diseased and two healthy sites from 12 patients with severe periodontitis were studied. Clinical parameters (gingival fluid flow, gingival index [GI], periodontal index [PI], and probing depth) were recorded and plaque samples obtained from apical sites of each crevice. Samples were diluted and observed with darkfield microscopy; bacteria were categorized into 9 morphotypes. Results indicated significant differences between flora at healthy and diseased sites. At healthy sites, coccoid cells predominated (74.4% versus 22.3%), while diseased sites presented a greater number of motile rods, curved rods, and spirochetes. The ratio of motile to non-motile cells at healthy sites was 1:49, while at diseased sites the ratio increased to 1:1. The authors suggest that this technique may be used to predict sites with active disease based on percentages of certain bacterial morphotypes.

Listgarten and Levin (1981) studied the correlation between proportions of subgingival spirochetes and motile bacteria and susceptibility of subjects to periodontal deterioration. At baseline and every 2 months for 1 year, clinical and microbiological parameters were recorded for 20 patients treated for moderate-advanced periodontitis. Microbial samples from the deepest pocket in each quadrant were pooled for analysis using DFM. Microbial forms were recorded as coccoid, motile rods, spirochetes, or others. When probing depth at any surface exceeded baseline by $\geq$ 3 mm, the tooth was exited from the study. A significant positive correlation was found between proportions of motile rods and PI and GI scores. Also, a positive correlation between PD and proportions of spirochetes was observed. Spirochetes and/or motile rods predominated in subjects who had 2 or more teeth exited from the study, whereas coccoid forms predominated in sites without exited teeth. The authors concluded that the proportion of spirochetes is a good predictor of periodontal deterioration within the upcoming year.

Armitage et al. (1982) correlated the percentage of motile bacteria with clinical assessments of inflammatory periodontal disease. Two sites were selected from each of 60 subjects and categorized into 6 disease categories, ranging from health to advanced periodontitis. These findings were compared to DFM analysis of subgingival plaque from each site. Bacterial morphotypes were classified as non-motiles, spirochetes, or other motiles. The authors reported that severity of disease correlated significantly with an increase in the percentage of spirochetes, although no such relationship was observed for other motile bacteria. There was also a significant correlation between each clinical parameter (PI, GI, BOP, PD, gingival exudate, attachment loss) and the percentage of spirochetes at individual sites. Bleeding on probing and/or probing depth or loss of attachment $>$ 3 mm were the parameters most closely related to the percentage of spirochetes. In fact, there was an 80% correlation between BOP and high levels (2 to 3 fold increase) of spirochetes. Due to the high correlation of clinical parameters with DFM findings, the need for microbiologic assessment of subgingival flora was questioned.

Rosenberg et al. (1981) examined composite bacterial populations before, during, and after various stages of periodontal therapy. Eighteen (18) patients with moderate periodontitis were scored for PI, GI, and PD. The tooth surface with the deepest pocket in each sextant (per patient) was chosen as the microbial sampling site. Subgingival plaque samples were obtained at baseline, after initial preparation, and at various times after surgical therapy. A pooled sample from each patient was studied by DFM for each time period. There was a significant increase in the mean percentage of coccoid cells and significant decrease in the

proportions of spirochetes and motile rods from baseline to post-initial therapy and from post-initial therapy to post-surgery. In spite of continued improvements in microbiological parameters, little or no change in clinical indices was noted after the surgical phase. Two of 18 patients exhibited increases in motile rods and spirochetes, accompanied by a deterioration of clinical parameters during the treatment period. The authors suggest that despite treatment rendered, microflora of these sites were related to increased disease susceptibility rather than periodontal health.

Evian et al. (1982) correlated proportions of bacterial morphotypes at different diseased sites within the same mouth, determining the extent of correlation with the clinical parameters of periodontitis in 14 patients with untreated moderate to advanced disease. After clinical indices (PD, PI, GI) were recorded, microbial samples were obtained from the deepest pocket in each sextant and examined individually by DFM. Bacterial morphotypes were categorized as cocci, motile rods, spirochetes, or others. Proportions of morphotypes among the 6 sites in any individual mouth varied considerably. In accordance with earlier studies, the mean percentage of spirochetes varied directly with increased PD, PI, and GI scores. However, no significant correlations could be determined when measurements of individual sites were analyzed. The authors suggest that differences in disease activity among sites as well as variability due to the sampling process may account for the results. The authors suggest that by pooling samples from individual patients, it may be possible to minimize sampling errors and still yield useful information about sites at risk.

CULTURAL STUDIES

Tanner et al. (1979) characterized the microbial flora at the apical region of advancing periodontitis lesions. Criteria of disease activity in 8 subjects with advanced periodontitis consisted of at least 2 mm of radiographic bone loss in the previous year. Anaerobic bacterial samples were obtained and cultured. Correlations between bacterial and clinical parameters were statistically evaluated. Microbiota of 2 young adult patients with generalized bone loss and inflammation were dominated by *Porphyromonas gingivalis* and *Actinobacillus actinomycetemcomitans*. Sites of 2 patients with extensive bone loss but minimal inflammation were dominated by *Prevotella intermedia, Eikenella corrodens*, and fusiform-shaped *Bacteroides* (probably *Bacteroides forsythus*). Sites of the remaining 4 patients had moderate inflammation with continued bone loss and demonstrated high levels of *P. gingivalis, F. nucleatum, "fusiform" Bacteroides*, and anaerobic *Vibrios*. Sites with minimal disease revealed higher proportions of Gram-positive organisms. The authors concluded that significant differences do exist in the apical plaque of adult periodontitis patients and that it seems likely that specific groups of organisms are related to certain clinical diseases.

RAPID IDENTIFICATION OF POTENTIAL PATHOGENS

Investigators have attempted to develop methods for rapidly identifying certain bacteria or disease activity based on bacterial antigenic profiles (DNA probes, latex agglutination), enzymatic activity (B-glucuronidase, collagenase, etc.), metabolic end products and/or antibody titers. Review articles by Armitage (1987) and Greenstein (1988) discuss some of the advantages and disadvantages of various rapid identification techniques.

DNA Probes. These are comprised of radio-labeled DNA fragments of specific bacteria such as *P. gingivalis*. Theoretically, when the labeled fragments are added to a digested plaque sample adhering to nitrocellulose, they will combine with any analogous fragments (*P. gingivalis* if present), allowing identification and quantification of the specific organism if present. This method is site specific and simple, but disadvantages include decay of the label, inter-reaction of labeled fragments, and cross-reactivity with DNA of other species.

Latex Agglutination. In this technique, plaque samples are mixed with latex beads that are coated with antibodies specific for certain periopathogens. If the pathogen is present in the plaque sample, cross-bridging of the antibody-pathogen-antibody results in visibly detectable agglutination of the beads. This technique has potential, but is not widely used.

Flow Cytometry. This technique involves reaction of plaque with fluorescein-tagged antibodies specific for selective pathogens. The sample is then dispersed and "tagged" pathogens are counted as they flow past a spectrometer. Disadvantages include equipment expense and an inadequate data base related to plaque microbe determination.

Use of ELISA. In this enzyme-linked immunosorbent assay, plaque samples are diluted and attached to a polystyrene plate to which pathogen specific antibodies (tagged with enzyme substrate) are added. Unattached antibody is then washed off and the plate reacted with an enzyme that is specific for the substrate tag. The remaining antibodies which are attached to the pathogens can be detected by spectrophotometry for color change.

Direct Immunofluorescence (IF). In this technique, fluorescein tagged antibodies are reacted with plaque dilutions and specific staining of organisms is observed using a fluorescent microscope.

Indirect IF. Here untagged antibodies are initially complexed with the antigen. The second step involves addition of fluorescein-tagged antibodies which will react with ("piggybacking") the first antibody (antisera). This gives a much higher fluorescein signal and has been used successfully to detect specific microbes in plaque samples (good for qualitative analysis, time consuming for quantitative analysis). Disadvantages: monoclonal antibodies are so specific they may overlook some of the organisms and polyclonal anti-

bodies may cross-react with other species. Indirect immunofluorescence is probably not clinically feasible.

When complex mixed infections make it difficult to single out individual pathogens, increased antibody titers to particular organisms may suggest pathogenicity. The problem with measuring serum antibody titers is that high levels are not always associated with disease progression, and non-specific cross-reactivity may occur. Titers are sometimes low in the presence of disease progression. This is explained by the fact that the systemic response (blood serum antibodies) may lag behind disease initiation. This lag may continue well beyond resolution of diseased sites and may also influence antibody titers to specific organisms.

RELEVANCE AND CONCERNS

Initially, darkfield studies appeared to offer a means for relating disease activity to proportions of motile bacteria, especially spirochetes. Evian et al. (1982), however, suggested that spirochetes are so ubiquitous in nearly all forms of periodontal disease that their mere presence in bacterial sample is of limited diagnostic value.

The initial study by Listgarten and Levin (1981) was encouraging, because it suggested that spirochetes and motile rods could be used to identify subjects at risk for future breakdown. In a subsequent study, however, Listgarten et al. (1984) were unable to use the percentage of spirochetes in a subgingival sample to identify specific sites at risk for breakdown. In addition, a separate study by the same group (Listgarten et al., 1986) was unable to effectively use microscopic monitoring to prevent recurrence of periodontitis by altering maintenance regimens. When pooled subgingival samples from a patient were examined for spirochete prevalence, levels of spirochetes tended to correlate with disease activity although site of the activity is not identified by this method. However, when levels of spirochetes at similar (> 6 mm) sites within a patient are examined, a correlation between disease activity and number of spirochetes could be shown. Other factors discouraging the use of DFM to monitor disease activity are that the major putative periodontopathogens (e.g., *P. gingivalis, A. actinomycetemcomitans*, and *P. intermedia* are non-motile and would be discounted by a method based solely on bacterial motility and/or number of spirochetes (Slots and Listgarten, 1980).

Technical difficulties associated with cultural studies include: 1) location of plaque sample; 2) tendency of various methods of dispersing bacterial plaque to favor growth of one species over another; and, 3) unavailability of a single culture media/method capable of recovering all bacterial species in subgingival plaque. Selective media may disinfranchise important species and purportedly "non-selective media" may "select" for different segments of microbiota. These 3 factors may be partially responsible for the variety of microbiological findings reported by different laboratories. Furthermore, probing accuracy may present a problem when attempting to associate cultural findings with probing depth. The time and expense associated with culturing presently limit its use to that of a periodontal research diagnostic tool.

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Section 4. Indices Used in Assessment of Periodontal Status

DEFINITION

Index: A relative or arbitrary system of measurement which describes or quantitates a condition. Such indices are appropriate for use in an individual patient or for epidemiological studies.

PURPOSE

Periodontal indices are designed to assess disease prevalence and/or incidence within a population or a given pa-

tient. Prevalence is defined as the number of existing cases in the population at a specific point in time, while incidence is the number of new cases of a disease from one time point to another. The long latent period of periodontal disease makes it difficult to determine a definite time of disease onset, a fact needed to establish incidence rates. Prevalence of disease is usually the main determinant in population health need surveys. For periodontal disease, the degree and severity of inflammation must be determined in order to determine treatment needs. All periodontal indices in current use measure morbidity. However, none are designed to measure tooth mortality related to periodontal disease.

TYPES

Indices may be reversible, irreversible, or composite (See Tables 1 through 8) (Barnes et al., 1986). An irreversible index measures the permanent tissue damage caused by disease. Indices which record radiographic bone loss and attachment loss are considered irreversible. Reversible indices assess active disease and allow for changes in periodontal health status. Examples are: Russell's periodontal index (PI), Ramfjord's periodontal disease index (PDI), and Greene and Vermillion's oral hygiene index (OHI), and OHI-simplified. The PI and PDI indices have reversible and irreversible components and are considered to be composite indices.

Löe (1967) described the gingival index, the plaque index, and the retention index systems. The gingival index (GI) describes qualitative changes in the gingival soft tissue at 4 areas on the tooth (buccal, mesial, distal, and lingual). A score of 0 to 3 is given for each area based on visual characteristics of inflammation after drying of the tissues and the presence or absence of bleeding when a probe is run along the soft tissue wall of the entrance of the gingival crevice. The GI may be used to determine a GI for the tooth (sum of the 4 areas divided by 4), a GI for a group of teeth (grouping scores of incisors, premolars, molars), or a GI for the individual (adding the indices for the teeth and dividing by the total number of teeth examined). Criteria for the GI are: 0 = normal gingiva; 1 = mild inflammation-slight change in color, slight edema, no bleeding on probing; 2 = moderate inflammation-redness, edema, and glazing with bleeding on probing; 3 = severe inflammation-marked redness and edema, ulceration, and spontaneous bleeding.

The plaque index (PI) describes the thickness of the soft debris aggregates in the gingival area of the tooth surfaces, and no attention is paid to the coronal extension of the plaque. A score from 0 to 3 is given for the same areas evaluated in the GI, but is based on the amount of soft matter detected following drying of the tissues and running a probe across the tooth surface at the entrance of the gingival crevice. PI should precede GI when the two indices are used together. Criteria for the PI are: 0 = no plaque in the gingival area; 1 = a film of plaque adhering to the free gingival margin and adjacent area of the tooth, recognized by running a probe across the tooth surface; 2 = moderate accumulation of soft deposits within the gingival pocket, on the gingival margin and/or adjacent tooth surface which can be seen by the naked eye; 3 = abundance of soft matter within the gingival pocket and/or on the gingival margin and adjacent tooth surface. The retention index describes the quality of the tooth surface as it relates to the presence of retentive factors such as calculus, ill-fitted margins, and carious lesions. Criteria are: 0 = no caries, no calculus, no imperfect margin of dental restoration in gingival location; 1 = supragingival cavity, calculus, or imperfect margin of restoration; 2 = subgingival cavity, calculus, or imperfect margin of restoration; 3 = large cavity, abundance of calculus or grossly insufficient marginal fit of restoration in a supra- and/or subgingival location.

Chaves et al. (1993) evaluated the association between gingival index (GI) bleeding (GI = 2 or 3) and bleeding on probing (BOP) in 125 gingivitis patients (19 to 62 years old). Clinical parameters included GI and PI with a manual probe, probing depth (PD), and BOP with a Florida probe. BOP was considered present if bleeding occurred within 20 seconds of probing. PD ranged from 0.1 to 5.9 mm; 98.6% of depths were 0.1 to 4 mm. Mean GI bleeding was 35.3% and mean BOP bleeding was 40.9%. When sites were evaluated, BOP showed a positive correlation with PD as did GI with PI. There was a good overall correlation between GI and BOP, and agreement varied with PD. Highest agreement between GI and BOP bleeding was for PD > 4 mm (85.4%) and PD > 2 mm (72.3%). In shallow pockets (0.1 mm to 2 mm), the highest percent agreement was for sites with GI = 0 or 1, or negative BOP (77.7%). When the relationship between BOP and visual signs of inflammation was related to PD, the percent BOP increased with increasing PD.

Muhlemann and Son (1971) concluded that bleeding from the sulcus is the earliest clinical symptom of gingivitis and that it precedes discoloration and swelling of gingival units. Sulcus bleeding index (SBI) was scored at time intervals over 17 days in 13 dental students refraining from oral hygiene measures. SBI was determined using a periodontal probe (diameter 0.5 mm) placed in the sulcus parallel to the tooth long axis at facial/lingual sites and directed towards the col at interproximal sites. A score of 1 represented a bleeding point which occurred up to 30 seconds after probing in the absence of gingival swelling or color change. At the start of the test period, 738 gingival units appeared healthy and did not bleed upon sulcus probing (score 0). After 17 days without hygiene, 264 apparently healthy units (score 0) remained. The number of score-1 units (bleeding upon gentle probing in the absence of color change or swelling) increased from 88 to 470. At this time, score-2 units (bleeding with change of color) increased from 6 to 89, while only 9 gingival units were slightly swollen (score 3). Using SBI, 64.2% of gingival units at

TABLE 1. INDICES USED TO EVALUATE SIGNS, SYMPTOMS, AND ETIOLOGIC FACTORS ASSOCIATED WITH DENTAL DISEASE (PLAQUE AND HYGIENE)

Index (source)	Measures	Scored by	Soft tissue area or teeth examined	Aspect or surfaces examined	Presence or severity measured	Uses
Oral hygiene index (OHI) (Greene and Vermillion)	Debris calculus	Segments	All except 3rd molars	Buccal, lingual	Severity	Epidemiologic surveys, clinical trials, monitoring, individual patient
Oral hygiene index simplified (OHI-S) (Greene and Vermillion)	Debris calculus	Teeth	Max (R) 1st molar Max (R) central incisor Max (L) 1st molar Mnd (L) 1st molar Mnd (L) central incisor Mnd (R) 1st molar	Buccal Facial Buccal Lingual Facial Lingual	Severity	Epidemiologic surveys
OHI-S debris modification (Glass)	Debris	Teeth	Same as OHI-S	Same as OHI-S	Severity	Epidemiologic surveys
Hygiene analysis index (HAI) (Love et al.)	Plaque	Teeth	All	Mesial, distal, facial, lingual	Presence/ absence	Patient motivation, monitoring patient progress
Patient hygiene performance (PHP) (Podshadley and Haley)	Plaque	Teeth	Same as OHI-S	Same as OHI-S	Severity	Epidemiologic surveys
Plaque index (Lennox and Kopczyk)	Plaque bleeding	Teeth	All	Mesial, distal, facial, lingual	Presence/ absence	Patient motivation, monitoring patient progress
Plaque index (Quigley and Hein)	Plaque	Teeth	All except 3rd molars	Buccal, lingual	Severity	Clinical trials, epidemiologic surveys

TABLE 2. INDICES USED TO EVALUATE SIGNS, SYMPTOMS, AND ETIOLOGIC FACTORS ASSOCIATED WITH DENTAL DISEASE (CALCULUS)

Index (source)	Measures	Scored by	Soft tissue area or teeth examined	Aspect or surfaces examined	Presence or severity measured	Uses
Calculus Surface Index (CSI) (Ennever et al.)	Calculus	Teeth	Mandibular central and lateral incisors	Mesial, distal, facial, lingual	Presence/ absence	Clinical trials
Probe Method Calculus assessment (Volpe et al.)	Calculus	Teeth	All mandibular incisors and cuspids	Lingual (and lingual aspects of inter-proximals)	Severity	Clinical trials, epidemiologic surveys
Marginal Line Calculus Index (MLC) (Muhlemann and Villa; Villa et al.)	Calculus	Teeth	Mandibular central and lateral incisors	Lingual	Severity	Clinical trials, patient progress, patient motivation

TABLE 3. INDICES USED TO EVALUATE SIGNS, SYMPTOMS, AND ETIOLOGIC FACTORS ASSOCIATED WITH DENTAL DISEASE (BLEEDING AND GINGIVAL)

Index (source)	Measures	Scored by	Soft tissue area or teeth examined	Aspect or surfaces examined	Presence or severity measured	Uses
Gingival Bleeding Index (GBI) (Carter and Barnes)	Bleeding	Teeth	All except between 2nd and 3rd molars	Interproximal	Presence/ absence	Clinical trials, patient progress, patient motivation
Sulcus Bleeding Index (SBI) (Muhlemann and Major; Muhlemann and Son)	Bleeding, gingival, color change, edema	Teeth	All maxillary and mandibular central incisors, lateral incisors, cuspids, and 1st bicuspids	Mesial, distal, facial, lingual	Presence/ absence and severity	Clinical trials, epidemiologic surveys, patient progress, patient motivation
PMA Index papillary, marginal, attached gingiva (Massler et al.; Schour and Massler)	Gingival inflammation (edema, bleeding necrosis, recession pocket formation)	Papillary marginal and attached gingival units	Around all teeth except 3rd molars	Facial, lingual	Severity and presence	Epidemiologic surveys
Gingival Index (GI) (Soumi and Barbano)	Gingival inflammation	Segments and teeth (papillary and marginal gingival units)	All	Facial, lingual	Severity	Epidemiologic surveys, clinical trials, patient progress
Gingival Index (GI) (Löe; Löe and Silness)	Inflammation (color changes, edema, bleeding, ulceration)	Teeth	Gingival, plaque and retention indices are intended to be used together as a system. All use the same teeth and same surfaces.			
Plaque Index (used with GI) (Löe)	Debris and plaque	Teeth	Max (R) 1st molar Max (R) lateral incisor	Mesial, distal, facial, lingual	Severity	Epidemiologic surveys, clinical trials, monitoring patient progress
Retention Index (used with GI) (Löe)	Calculus, caries, overhanging restorations, other retentive agents	Teeth	Max (L) 1st bicuspid Mnd (L) 1st molar Mnd (L) lateral incisor Mnd (R) 1st bicuspid			

TABLE 4. INDICES USED TO EVALUATE SIGNS, SYMPTOMS, AND ETIOLOGIC FACTORS ASSOCIATED WITH DENTAL DISEASES (PERIODONTAL)

Index (source)	Measures	Scored by	Soft tissue area or teeth examined	Aspect or surfaces examined	Presence or severity measured	Uses
Periodontal Disease Rate Index (PDR) (Sandler and Stahl)	"Periodontal Disease": inflammation crevice depth, mobility, alveolar bone resorption	Teeth	All	Mesial, distal, facial, lingual, (uses radiographs)	Presence/absence	Crude epidemiologic surveys
Periodontal Index (PI) (Russell)	Inflammation pocket depth tooth mobility	Teeth	All	Facial, lingual, mesial, distal	Severity	Epidemiologic surveys
Periodontal Disease Index (PDI) (Ramfjord)	Pocket depth, crevice depth (from CEJ)	Teeth	Max (R) 1st molar Max (R) lateral incisor Max (L) 1st bicuspid Mnd (L) 1st molar Mnd (L) lateral incisor Mnd (R) 1st bicuspid	Mesial, facial	Severity	Epidemiologic surveys, clinical trials
Gingival Index (a part of PDI) (Ramfjord)	Inflammation, bleeding, edema, ulceration	Teeth	Same as PDI	Mesial, facial	Severity	Epidemiologic surveys, clinical trials
Plaque Index (used with PDI) (Ramfjord [Shick and Ash Modification])	Plaque	Teeth	Same as PDI	Facial, lingual, mesial, distal	Presence and severity	Epidemiologic surveys, clinical trials
Calculus Index (used with PDI) (Ramfjord)	Calculus	Teeth	Same as PDI	Facial, lingual, mesial, distal	Severity	Epidemiologic surveys
Periodontal screening examination (O'Leary)	Gingival Index: inflammation	Segments	All	Gingival index, facial, lingual, interproximals	Severity	Monitor patient progress, epidemiologic surveys
Includes: Gingival Index Periodontal Index Irritant Index	Periodontal Index: pocket depth Irritant Index: plaque, calculus, overhanging restorations, unfilled	Segments Segments All	All	Periodontal index, mesial, line angle Irritant index: all tooth surfaces	Severity Severity	

TABLE 5. COMPARISON OF SCORES IN FREQUENTLY USED PLAQUE AND DEBRIS INDICES

	Scores					
Index	0	1	2	3	4	5
Greene and Vermillion OHI-S Debris Index	No debris or stain	Soft debris covering not more than one third of the tooth surface	Soft debris covering more than one third, but not more than two thirds of the exposed tooth surface	Soft debris covering more than two thirds of the exposed tooth		
Glass modification of OHI-S	No visible debris	Debris visible at gingival margin, but discontinuous. Less than 1 mm in height	Debris continuous at gingival margin. Greater than 1 mm in height	Debris involving entire gingival third of tooth	Debris generally scattered over tooth surface	
Löe Plaque Index	No plaque in the gingival area	A film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may only be recognized by running a probe across the tooth surface	Moderate accumulation of soft deposit is within the gingival margin, which can be seen by the naked eye	Abundance of soft matter within the gingival pocket and/or on the gingival margin		
Ramfjord Plaque Index	No plaque	Plaque present on some but not all interproximal buccal and lingual surfaces of the tooth	Plaque present on all interproximal, buccal and lingual surfaces, but covering less than one half of these surfaces	Plaque extending over all interproximal buccal and lingual surfaces, but covering more than one half of these surfaces		
Quigley and Hein Plaque Index (modified)	No plaque	Separate flecks of plaque at the cervical margin of the tooth	A thin continuous band of plaque (up to 1 mm) at the cervical margin	A band of plaque wider than 1 mm but covering less than one third of crown	Plaque covering at least one third but less than two thirds of the crown	Plaque covering two thirds or more of crown
Podshadley and Haley PHP	No plaque	The tooth surface if mentally divided into 5 areas. Plaque in only one area	Plaque in two areas	Plaque in three areas	Plaque in four areas	Plaque in five areas
Lennox and Kopczyk Plaque Index	Plaque severity is not scored; only presence or absence on four tooth surfaces.					
Love Hygiene Analysis Index	Plaque severity is not scored; only presence or absence on four tooth surfaces					

TABLE 6. COMPARISON OF SCORING SYSTEM IN FREQUENTLY USED CALCULUS INDICES

Scores	Greene and Vermillion OHI-S Calculus Scores	Ramfjord Calculus Index	Ennever et al.		Volpe and Manhold probe method of calculus assessment	Muhlemann and Villa marginal line calculus index	O'Leary Irritant Index
			Calculus Surface Index	Calculus Surface Severity Index			
0	No calculus present	No calculus	Has no severity component. Scores 16 tooth surfaces for presence or absence of calculus	No calculus present	Uses a periodontal probe to measure the height and width of calculus deposits on tooth surface. Results are reported in millimeters	Uses a probe to measure height of calculus deposits on tooth surfaces. Results reported in percentage of surface covered (0, 12.5, 25, 50, 75, and 100%)	No detectable plaque, or calculus, either supragingival or subgingival is found on any tooth in the segment
1	Supragingival calculus covering not more than one third of the exposed tooth surface	Supragingival calculus extending only slightly below the free gingival margin		Calculus observable, but less than 0.5 mm in width and/or thickness			A slight amount of plaque or supragingival calculus not extending more than 2 mm from the gingival margin is found on any tooth in the segment
2	Supragingival calculus covering one third to two thirds of the exposed tooth surface and/or flecks of subgingival calculus	Moderate amount of supra- and subgingival calculus or subgingival calculus alone		Calculus not exceeding 1.0 mm in width and/or thickness			Plaque or supragingival calculus covers up to one half the exposed clinical crown on any tooth in the segment
3	Supragingival calculus covering more than two thirds of the exposed tooth surface and/or a continuous band of subgingival calculus	An abundance of supra- and subgingival calculus		Calculus exceeding 1.0 mm in width and/or thickness			Plaque or supragingival calculus covers more than one half the clinical crown or subgingival calculus deposits or overhanging or deficient restorations are detectable by probing

TABLE 7. COMPARISON OF SCORES IN FREQUENTLY USED GINGIVAL AND BLEEDING INDICES

Index	Scores					
	0	1	2	3	4	5
Schour and Massler PMA Index Papillae	Normal: no inflammation	Mild papillary engorgement; slight increase in size	Obvious increase in size of gingival papilla: hemorrhage on pressure	Ecessive increase in size with spontaneous hemorrhage	Necrotic papilla	Atrophy and loss of papilla (through inflammation)
Marginal	Normal; no inflammation visible	Engorgement; slight increase in size; no bleeding	Obvious engorgement; bleeding upon pressure	Swollen collar; spontaneous hemorrhage; beginning infiltration into attached gingiva	Necrotic gingivitis	Recession of the free marginal gingiva below the CEJ due to inflammatory changes
Attached gingiva	Normal; pale rose; stippled	Slight engorgement with loss of stippling; change in color may or may not be present	Obvious engorgement of attached gingiva with marked increase in redness. Pocket formation present	Advanced periodontitis. Deep pockets evident		
Löe and Silness Gingival Index	Normal gingiva	Mild inflammation, slight change in color, slight edema. No bleeding on probing	Moderate inflammation, redness, edema and glazing. Bleeding on probing	Severe inflammation—marked redness and edema. Ulceration. Spontaneous bleeding		
Ramfjord Gingival Index	Absence of signs of inflammation	Mild to moderate inflammatory gingival changes, not extending around the tooth	Mild to moderately severe gingivitis extending all around the tooth	Severe gingivitis characterized by marked redness, swelling, tendency to bleed and ulceration		
O'Leary Gingival Score	Normal gingiva	Slight to moderate inflammation not surrounding any teeth	Slight to moderate inflammation surrounding one or more teeth	Marked inflammation ulceration, spontaneous bleeding, loss of surface continuity, clefts of gingival tissue		
Muhlemann and Major, Muhlemann and Son Sulcus Bleeding Index	Healthy appearance, no bleeding on sulcus probing	Apparently healthy P and M showing no change in color and no swelling, but bleeding from sulcus on probing	Bleeding on probing and change of color due to inflammation. No swelling, macroscopic edema	Bleeding on probing, change in color and slight edematous swelling	Bleeding on probing, change in color obvious swelling	Bleeding on probing and spontaneous bleeding and change in color, marked swelling with or without ulceration
Soumi and Barbano Gingival Index	Absence of inflammation. Gingiva is pale pink in color and firm in texture. Swelling is not evident and stippling can usually be noted	Presence of severe inflammation. A distinct color change to red or magenta is evident. There may be swelling and loss of stippling. The gingiva may be spongy in texture	Presence of severe inflammation. A distinct color change to red or magenta: swelling, loss of stippling and a spongy consistency. Gingival bleeding upon gentle probing. The inflammation has spread to the attached gingiva			
Carter and Barnes Gingival Bleeding Index	Bleeding severity is not scored. Only the presence or absence of bleeding from a maximum of 28 gingival units following flossing is recorded.					

TABLE 8. COMPARISON OF SCORES IN FREQUENTLY USED PERIODONTAL INDICES

Scores	Russell Periodontal Index	Ramfjord Periodontal Disease Index (Crevice Depth)	O'Leary Periodontal Screening Examination	Scores
0	Negative. There is no overt inflammation		Upon probing of the gingival crevices, a score of 0 is given if the probe does not extend 1 mm apical to the CE Junction of any tooth in the segment and there is no exposure of the CEJ on any surface of any tooth in the segment	0
1	Mild gingivitis. There is an overt area of inflammation in the free gingiva which does not circumscribe the tooth	If the gingival crevice in none of the measured areas extends apically to the CEJ, the gingival score is the PDI score for that tooth (it can range from 0 to 3)		1
2	Gingivitis. Inflammation completely circumscribes the tooth, but there is no apparent break in the epithelial attachment			2
3				3
4		The gingival crevice extends apical to the CEJ, but not more than 3 mm	The probe extends up to 3 mm apical to the CEJ of any tooth in the segment	4
5		The gingival crevice extends apically from 3 to 6 mm in relation to the CEJ	The probe extends from 3 to 6 mm apical to the CEJ of any tooth in the segment	5
6	Gingivitis with pocket formation. The epithelial attachment has been broken and there is a pocket. There is no interference with normal masticatory function, the tooth is firm in its socket	The gingival crevice extends more than 6 mm apically to the CEJ	The probe extends 6 mm or more apical to the CEJ of any tooth in the segment	6
7				7
8	Advanced destruction with loss of masticatory function			8

risk (score 0 at baseline) revealed inflammation. Without using SBI and with visual inspection alone (GI), only 11.9% of units would have been detected as having become inflamed. Bleeding from the gently probed sulcus precedes the appearance of gingival color changes and is the leading and first clinical symptom of marginal gingivitis. The authors conclude that the higher sensitivity of SBI makes it possible to use non-hygiene periods of a reasonable length of time (2 to 3 weeks) in areas with moderate gingivitis activity. Characteristics of the SBI which differentiate it from the GI are: 1) inflammation is diagnosed only if bleeding occurs upon gentle probing of the sulcus; 2) apparently healthy gingival units without color changes or swelling are diagnosed as inflamed (score 1) if the bleeding occurs upon gentle probing; and 3) resulting SB scores are roughly 1 unit higher.

Quirynen et al. (1991) examined 5 plaque indices (Harrap index, Quigley and Hein index, Navy plaque index modified by Clemmer and Barbano, Navy plaque index modified by Hancock and Wirthlin, planimetrical plaque index) to determine which could best discriminate the difference in the rate of plaque formation associated with healthy tissue (phase I) versus with gingival inflammation (phase II) where plaque was allowed to accumulate over a 96-hour period. Significant differences between phase I and phase II plaque accumulations could not be detected using the Harrap index and the Navy index (Hancock and Wirthlin). The Navy index (Clemmer and Barbano) detected a difference at 82 hours while the Quigley and Hein index detected a phase I and phase II difference at 72 hours. The planimetrical index detected a significant difference at 36 hours. The planimetrical plaque index demonstrated the

highest discriminating power; the Harrap and Navy index (Hancock and Wirthlin) was the least discriminating. Although the planimetrical index is highly reproducible, it is time consuming and difficult to use. The Quigley and Hein or Navy index (Clemmer and Barbano) seem to be the best alternative of those indices studied if the planimetrical index is not feasible. Each index has its own advantages and disadvantages and the aim of study should determine the plaque index chosen.

Almas et al. (1991) evaluated the capacity of the community periodontal index of treatment needs (CPITN) to reflect the clinical periodontal status in 52 patients as represented by the standard clinical indices of PI, GI, papilla bleeding index (PBI), and PD. CPITN scores determined for each sextant are as follows: code 4 = pathologic pockets 6 mm or more; code 3 = pathologic pockets 4.0 to 5.5 mm; code 2 = supra-or subgingival calculus; code 1 = gingival bleeding after probing; and code 0 = none of the above signs present. PD was assessed using a pressure-sensitive probe with 0.34 mm tip and 0.25 N force. CPITN was assessed using a (WHO 612) CPITN probe with a ball end of 0.5 mm and 0.25 N force. Six sites per tooth were recorded. CPITN did not correlate with the amount of plaque present, number of sites affected in a given sextant, or GI. There was a tendency for an association between the CPITN and both the PD and the PBI. In spite of the above correlations, extreme ranges for all indices were found within a sextant regardless of the CPITN code; 25% of sites had pockets > 6 mm deep and 70% of sextants had a CPITN score of 4. This study demonstrated that the CPITN functions as an epidemiologic tool for population planning and treatment needs but is not indicated for assessment of individual treatment needs.

Kaldahl et al. (1990) evaluated gingival suppuration and supragingival plaque following 4 modalities of periodontal therapy: coronal scaling (CS) only; coronal and subgingival scaling and root planing (RP); root planing followed by modified Widman surgery (MW); root planing followed by flap reflection with osseous surgery (FO). Clinical assessments were made at baseline (exam 1), 4 weeks following oral hygiene instruction (OHI) and CS or Sc/RP (exam 2), 10 weeks following respective therapies (exam 3), and annually prior to maintenance therapy appointments (exams 4 and 5). This assessment included presence of supragingival plaque, gingival suppuration, probing depth, and clinical attachment level. Patients were seen every 3 months for 2 years for maintenance therapy. The authors found that the prevalence of supragingival plaque between the groups except for FO-treated sites showed more plaque accumulation after surgical therapy. The presence or absence of supragingival plaque at specific sites was dynamic, frequently converting to a new status between 2 examinations. Sites that were not suppurating at 1 exam but were suppurating at the subsequent exam or at both exams had a less favorable response in both probing depth and probing attachment level.

USES

There are 4 ways in which a periodontal index may be used: 1) clinical trials; 2) epidemiologic surveys; 3) evaluation of patient progress; and 4) motivation of the patient to improve hygiene. The ideal index should: 1) be simple to use; 2) require minimum time; 3) require minimum armamentarium; 4) be clear, understandable, and reproducible; 5) be amenable to statistical analysis; 6) be equally sensitive throughout its scale of variable measure; and 7) be acceptable to the patient (Barnes et al., 1986).

CONSIDERATIONS

When using indices or evaluating studies using indices, the following should be considered: 1) studies conducted with different indices should be compared only as to general findings rather than specific details; 2) an index should be selected according to its ability to best evaluate the variable (i.e., presence/absence of plaque rather than quantity of plaque); 3) index methodology should not be modified; 4) those that measure presence or absence of plaque are more applicable to motivation at assessment while those measuring quantity may be more suited for clinical trials and epidemiologic surveys; 5) full-mouth scoring should be reserved for clinical use, a single patient or small population groups, while simplified indices are more useful for epidemiologic surveys and large clinical studies; 6) examiner intra- and inter-reliability should be established prior to use and repeated throughout the study time period.

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