Roy C Page, John A Martin

Quantification of periodontal risk and disease severity and extent using the Oral Health Information Suite (OHIS)

Roy C Page
Emeritus Professor,
Department of Periodontics,
Box 357480 School of Dentistry,
University of Washington,
Seattle, WA 98195, USA
Tel: 206-543-5599
Fax: 206-685-8024
Email: roypage@u.washington.edu

John A Martin
Private Practice,
2521 Carnegie Dr.,
State College,
PA 16803, USA

KEY WORDS *disease severity and extent, periodontal disease, risk assessment, therapeutic outcomes*

Change has occurred in dentistry and periodontics where improved methods to accurately assess risk for periodontal disease and quantify periodontal disease status would benefit all stakeholders of oral health care. This paper focuses on a new technology that provides these measurements. The Periodontal Assessment Tool (PAT) is an integral component of the Oral Health Information Suite™ (OHIS). Using only data gathered during a traditional periodontal examination, PAT accurately assesses the risk and quantifies severity and extent for periodontal disease, reporting them as a risk score and disease score, respectively. The risk score is expressed on a scale of 1 (lowest risk) to 5 (highest risk) and the disease score on a scale of 1 (healthy) to 100 (generalised severe periodontitis). The risk score has been shown to be valid and accurate where the study population was observed for 15 years. The extent of agreement between calculated disease scores and actual severity measured as mean alveolar bone height using digitised radiographs was strong (r = 0.735). Furthermore, there was strong agreement between the disease score and the diagnostic opinions of periodontists, general dentists and dental hygienists. Both risk and disease scores are objective, precise, and sensitive to minor changes, either improvement or deterioration. These novel scores provide a powerful new tool for monitoring periodontal status, quantifying therapeutic outcomes, and for communication with patients, third party payers and other health professionals.

■ Introduction

Several developments have occurred in dentistry that mandate fundamental changes in oral health care. Periodontal disease is no longer ubiquitous. To the contrary, in the United States and some other industrialised countries, stratification of severity exists, with the highest severity occurring in roughly 10% of the population[1-3]. While bacteria are essential for disease to occur and progress, bacteria alone are insufficient; a susceptible host is also essential, as disease is the result of a complex interplay between host

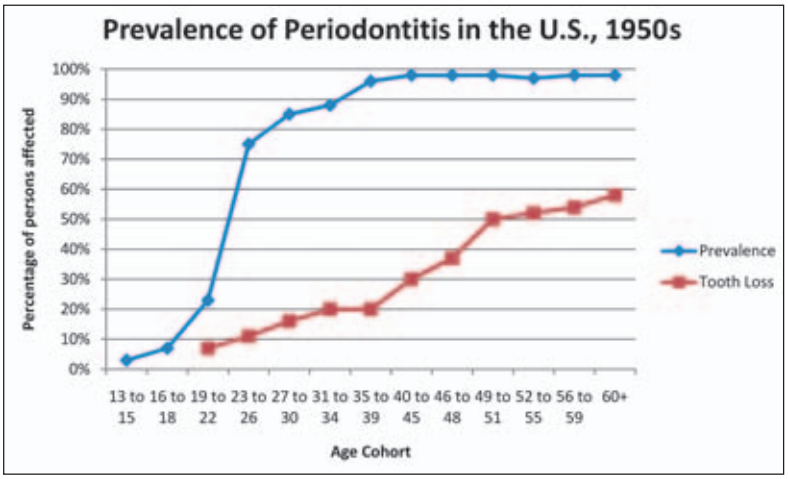

Fig 1 Prevalence of periodontitis in the 1950s. Periodontitis (blue diamonds), tooth mortality (red squares). Adapted from Marshall-Day et al[20].

and bacteria[4,5]. The evidence shows that bacteria account for about 20% of the variance of disease, with host risk factors being major determinants, where the net result is large differences in individual risk and susceptibility[6]. Achieving and maintaining a high level of oral health requires addressing risk factors as well as the infectious causes of disease, making risk assessment an integral component of diagnosis and essential for treatment planning[2].

In the US and some other countries, the cost of oral health care is escalating at a rate that is not sustainable. Payers for health care services have attempted to examine the value of these large expenditures[7] but outcomes measurements remain incomplete, patients cannot evaluate the technical quality of care, and evidence is lacking that correlates biological surrogate measures to outcomes of importance to a patient[8-11]. Numerous experts have explained how these deficiencies interfere with providing quality health care[7-15].

Long-term studies by Axelsson et al[2] demonstrated that periodontal disease, dental caries and tooth loss are preventable. These results were achieved by assessing disease status and the level of risk and providing interventions based on the patient's unique risk and disease profile. Using this approach, the quality of oral health care can be improved with a large net saving in health care cost. Axelsson's findings are consistent with projected savings in medicine due to improving quality[7].

The day-to-day practice of dentistry has not yet taken into account the demographic changes in the population, the role of risk and susceptibility, or variability in disease progression. A significant proportion of treatments and preventive measures being provided appear to be either inappropriate or not needed[7,8,13,16-18]. These conditions fuel the escalating costs for oral health care. Clinicians generally acknowledge that patients manifest greatly differing levels of risk for periodontitis. Nevertheless, risk is not being assessed with objective consistency, nor is needs-based prevention and treatment provided. Clinicians continue to apply the same treatment plans and preventive care, including its frequency for all patients, regardless of their level of risk.

Because of the relationship between periodontitis and other systemic diseases and conditions, greatly improved communications are needed between general dentists and their patients and with other health professionals such as physicians and periodontists. Patients are becoming increasingly interested and involved in their own care and they want and need more and better information about their oral health status, including its change.

The fundamental problem is the lack of methods to objectively quantify risk and disease status. Therapeutic outcomes cannot be optimally improved nor the cost of care effectively reduced without these measurements. The Oral Health Information Suite

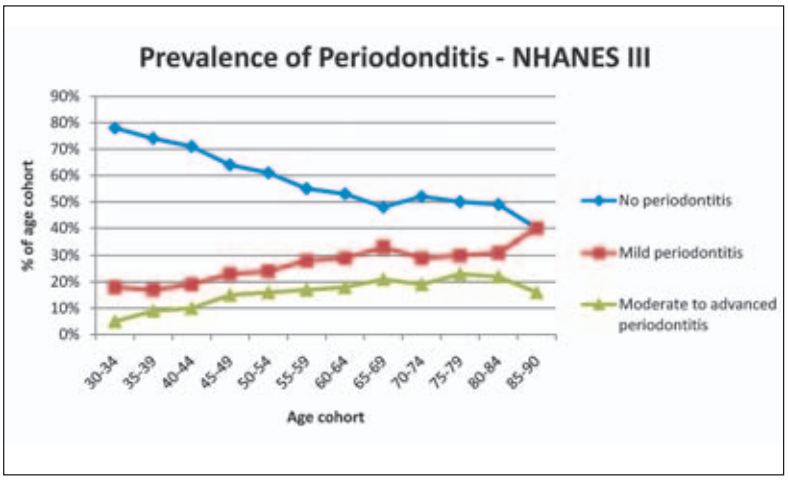

Fig 2 Proportions of the American population who do not develop periodontitis (blue diamonds), who develop mild periodontitis (red squares), and who develop moderate to severe periodontitis (green triangles) by age cohort, based on the NHANES III data set. Original data from Albandar et al[3].

(OHIS™)[19] appears to meet these needs. The purpose of this paper is to describe the component of OHIS™ for periodontal disease, the Periodontal Assessment Tool (PAT), and provide the scientific basis for its validity and accuracy. The companion paper in this issue by Loeb demonstrates the benefits of use of the technology in the day-to-day practice of dentistry. We will describe the relationship of risk to diagnosis, discuss the validity of traditional ways to assess risk, and describe traditional methods used for diagnosis. Then we will turn to a presentation of a new approach to diagnosis and treatment planning using PAT.

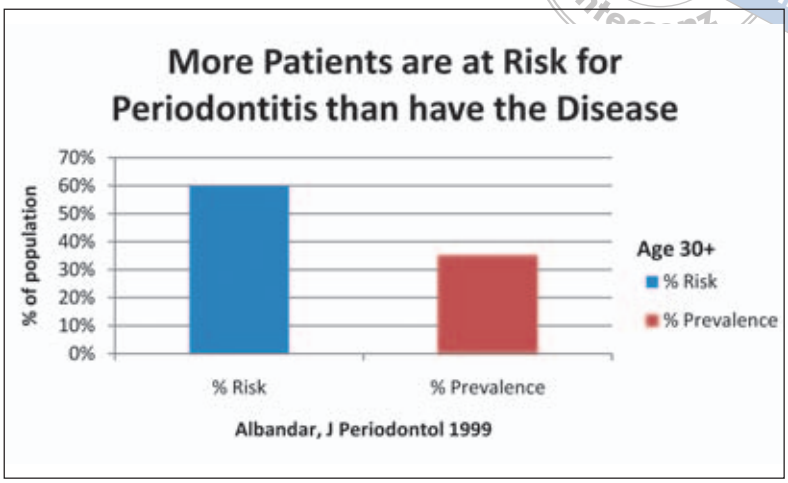

Fig 3 Proportion of the American population that have enhanced susceptibility for periodontitis, and the proportion that manifest the disease by age cohort. Original data from Page et al[24].

■ Disease prevalence and progression: diagnosis and risk

Prior to the 1950s, periodontitis affected almost all adults in the US and other industrialised countries. As shown in Fig 1[20], which is typical for epidemiological data reported at that time, periodontitis began to appear in the teenage years and the prevalence increased almost linearly until early middle age, when virtually all adults were reported to be affected, which meant that risk was nearly 100%. Over recent decades, demographics of the disease have changed significantly. Results of a national survey of adults in the US conducted in the mid-1980s by the National Institute of Dental Research show that moderate to severe periodontitis affected about 13% to 15% of Americans between the ages of 18 and 65 years[21]. In the more recent NHANES III national survey, 10% to 12% of adult Americans were affected[3]. When this data set was analysed by age cohort (Fig 2), 40% percent of the population did not manifest periodontitis at any time during their lifespan, while 35% developed mild periodontitis and 25% developed moderate to severe periodontitis. Thus, today about 60% of the US adult population is at elevated risk for periodontitis at some time during their lifespan compared to 35% who currently manifest the disease (Fig 3). Fig 4 focuses on the 60% who are at risk for periodontitis[19]. Only a negligible proportion of the susceptible group who are <30 years of age will show any signs or symptoms of periodontitis. As the group passes into and through the older age cohorts, an increasing proportion will develop periodontitis until, in the oldest age cohort, 60% will have developed the disease. Prior to the onset of signs or symptoms of periodontitis, individuals who are resistant cannot be distinguished from those at elevated risk, presenting a fundamental problem for clinicians as the latter have a higher level of treatment needs.

The realisation in the early to mid-1980s that the prevalence of periodontitis is not universal led clinicians and investigators for the first time to look at periodontitis in terms of risk and to identify characteristics that distinguish groups that have periodon-

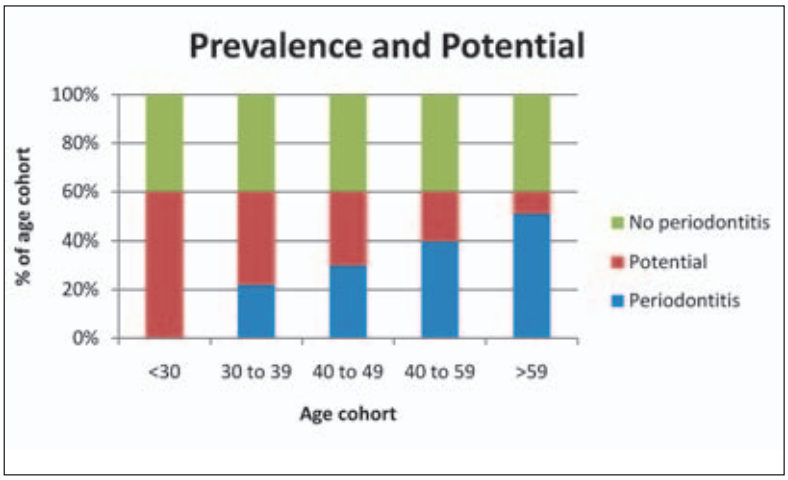

Fig 4 Changes in the proportion of individuals who were at risk for developing periodontitis but did not yet manifest the disease, those who were at risk and developed disease, and those who were not at risk and did not develop disease by age cohort. Based on the NHANES III data set. Original data from Albander et al[3].

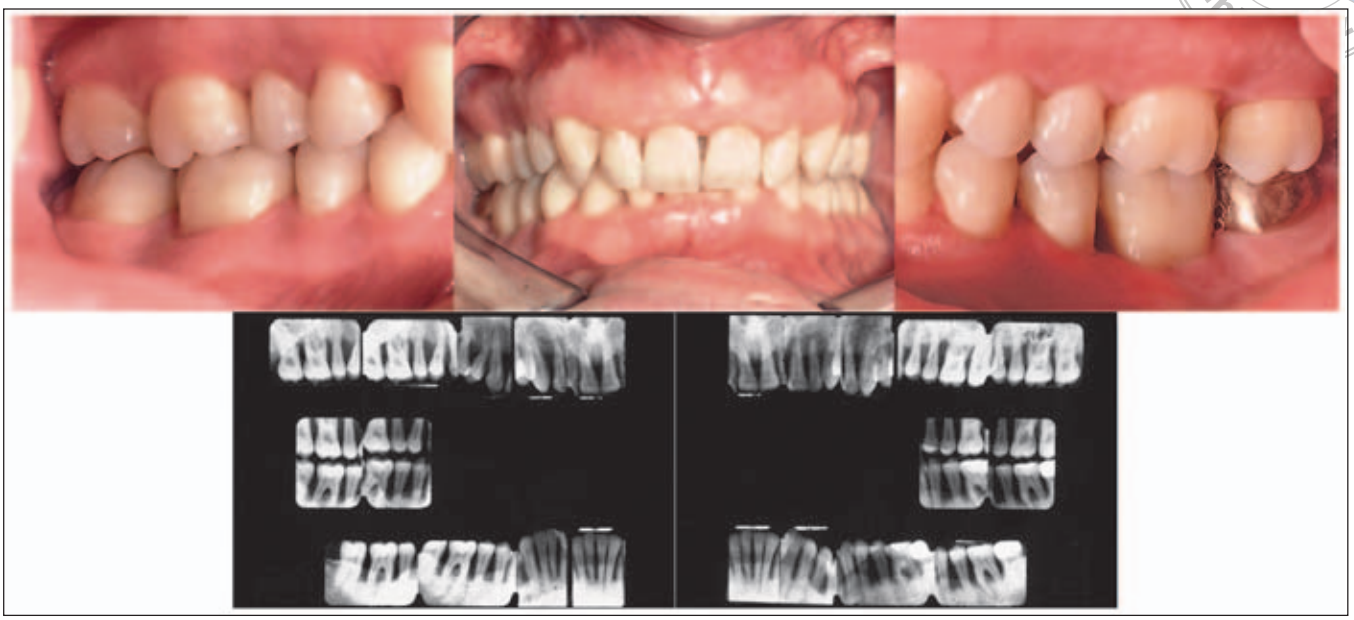

Fig 5 Clinical and radiographic manifestation of advanced periodontitis in a 27-year-old male. Note the relatively normal appearance of the teeth and gingival tissue and the extreme alveolar bone destruction.

titis from groups that are periodontally normal. The result has been the identification of genetic and acquired factors that place individuals at elevated risk[4,6,22,23]. While bacteria are essential for disease to occur, disease occurrence is determined by the complex interplay of the host and infecting bacteria[5]. The evidence shows that bacteria alone account for only about 20% of the variance for disease[6], with the remainder attributable to host risk factors, which indicates that modulation of host risk factors must be incorporated into prevention and treatment.

The case shown in Fig 5 serves to illustrate the problem. This patient, a 27-year-old male who presented with a 28-tooth dentition, was relatively free of plaque and calculus and had apparently clinically healthy gingival tissue. The radiographs, however, revealed extreme alveolar bone loss affecting all of the teeth. Periodontitis of this severity virtually ensures that all of the teeth will be lost at a young age. He has not always had this condition; at some time in the past he was healthy, although he would have been at high risk. Methods that would have detected his high-risk level prior to or soon after the onset of his disease process, when intervention could have been successful, did not exist at that time.

■ The relationship of risk to periodontal disease diagnosis

For a given patient, most practitioners, understandably, but incorrectly, equate risk for periodontitis with the extent and severity of periodontal disease; patients who have little to no disease are assumed to be at low risk for disease, while patients having disease are considered to be at high risk. Risk and diagnosis (or disease status) are entirely different entities. Risk predicts the disease state at some future point in time, or the rate at which an existing disease condition is likely to progress. Severe disease logically implies high risk. However, an individual can, in fact, be at elevated risk for periodontal disease and have little clinical or radiographic evidence of disease. An example would be a 25-year-old, poorly controlled diabetic patient who is a frequent tobacco smoker and has one 5 mm pocket that bleeds on probing and one defective restoration. Additionally, an individual can have periodontitis, especially of mild or moderate severity, but be at low risk. An example would be a previously untreated 61-year-old patient who formerly smoked frequently, has generalised 2 mm crestal bone loss, and 7 mm pockets affecting the posterior teeth.

In contrast to risk, diagnosis is an expression of the current disease conditions as determined by the

Fig 6 Role of risk assessment in diagnosis and treatment planning. Original data from Page et al[19].

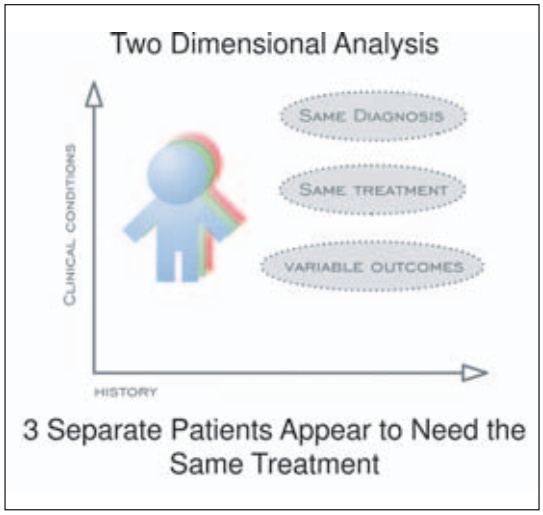

Fig 6a Periodontal diagnosis of three patients in the absence of risk information.

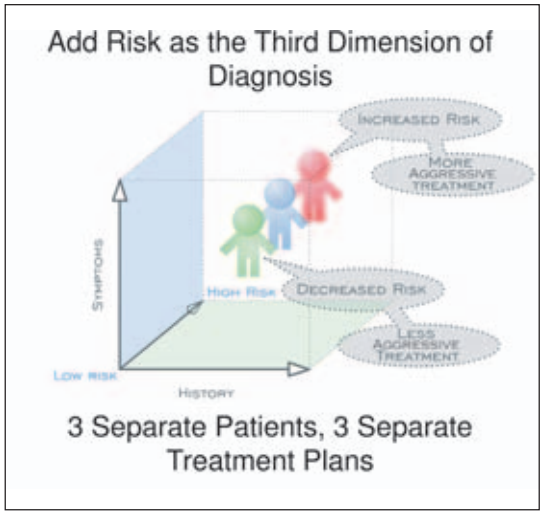

Fig 6b Periodontal diagnosis of the same three patients including consideration of their levels of risk.

presence and extent of inflammation, attachment loss, pocket depth and the extent and pattern of alveolar bone loss. Traditionally, diagnosis has not included measurement of risk. As illustrated in Fig 6a[19], when levels of risk are not considered, three patients with the same clinical and radiographic conditions and comparable histories appear to have the same diagnosis, and all three would appear to require the same treatment plan. When the level of risk is considered in the diagnostic equation (Fig 6b)[19], the same three patients require three different treatment plans, even though they have the same clinical conditions and histories. The individual at lowest risk may require only scaling and root planing with recalls at 6-month or longer intervals. The patient at moderate risk may require scaling and root planing with surgery in some areas followed by 4-month recalls, while the high-risk patient may require referral to a periodontist, along with root planing, more aggressive and extensive surgery and shorter recall intervals. As demonstrated in the Axelsson et al[2] research, matching the intensity of intervention with the risk profile of the patient can significantly reduce disease incidence as well as the cost of oral health care. Without consideration of risk, over- or under-treatment will probably occur, and a successful therapeutic outcome becomes less likely[24].

■ Traditional periodontal risk assessment and diagnosis

■ Periodontal risk assessment

Major risk factors and risk indicators for periodontal disease have been identified and they include frequent cigarette smoking, poorly controlled diabetes mellitus, stress, poor oral hygiene, past periodontal disease experience and heredity[4,6,22,23]. Although most dentists generally agree that risk assessment is important, are knowledgeable about risk factors, and collect information required for risk assessment, they are not trained to objectively assess risk, and tools for objective quantification have not been available. Consequently, as currently performed, risk assessment consists of identifying risk factors an individual patient may manifest during the examination and history taking process, and then making a subjective qualitative judgment as to the magnitude and role these factors may be playing in the disease process. The evidence, as explained in the next paragraph, shows, however, that this method is inconsistently accurate. Risk is dependent on the presence, strength, and interactions of risk factors. These factors manifest different and variable weights and the interaction of factors is non-linear and synergistic or antagonistic. Consequently, it should come as no

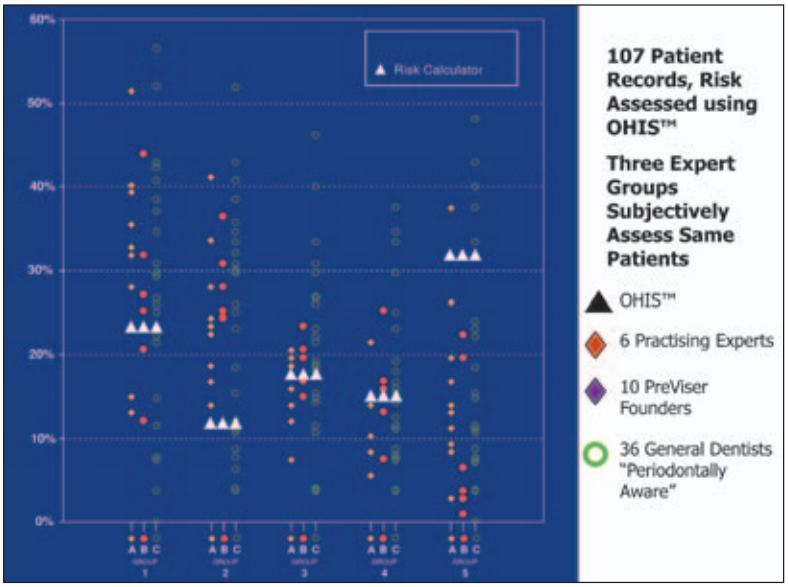

Fig 7 Percentages of subjects assigned to risk score group 1 (low risk of periodontal disease) to 5 (high risk of periodontal disease) by the Periodontal Risk Calculator (Previser, Inc, Mount Vernon, WA, USA) and the percentages of total subjects assigned by each expert evaluator in Group A (10 periodontists), Group B (six periodontists), and Group C (36 general dentists) and the evaluator group consensus scores (average scores). Reproduced from Persson GR, Mancl LA, Martin JA, Page RC. Assessing periodontal disease risk. J Am Dent Assoc 2003;134:575–582. Copyright ©2003 American Dental Association. All rights reserved. Reprinted by permission.

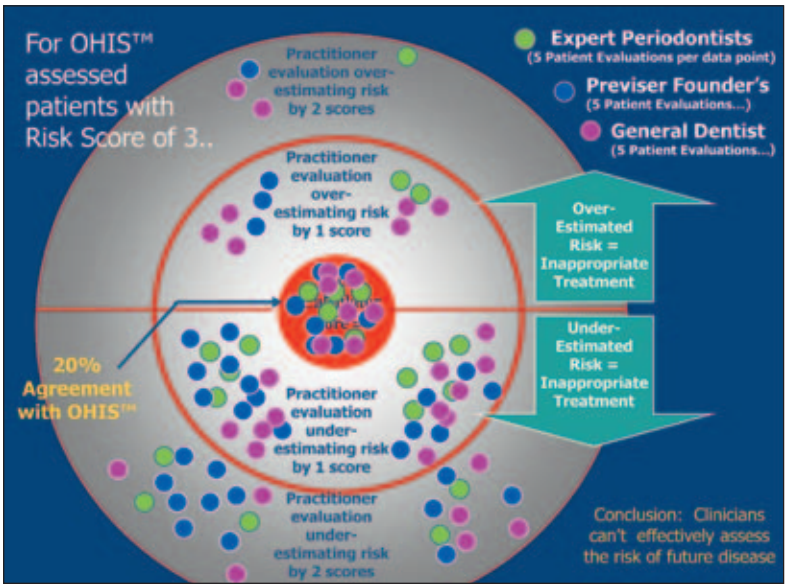

Fig 8 The extent of agreement for subjects assigned a risk score of 3 by the risk assessment tool and risk scores assigned by expert evaluator groups A (blue), B (green), and C (red). Each circle represents 5 risk assessments. For agreement, circles are located on the bulls' eye; when scores of 4 or 2 instead of 3 were assigned, circles are located in the inner circle; when 1 or 5 were assigned instead of 3, circles are located in the outer circle. Original data from Page et al[24].

surprise that assessment of risk by subjective qualitative judgment may yield less than ideal results.

A significant advance was made in assessment of periodontal risk when Lang and Tonetti[25] developed a multifactorial system known as the functional periodontal pentagon risk diagram (PPRD). The system consists of six graded vectors, each of which designates a risk factor or indicator. Periodontal risk assessments performed using the PPRD show a high degree of association with periodontitis as measured by the extent of alveolar bone loss[26]. However, the system is very complex and somewhat cumbersome for routine use in dental practice.

A study was designed and conducted by Persson et al[27] to evaluate the validity of subjective risk assessment. A study group of 107 subjects was assembled to have the broadest possible range of risk for periodontitis; a comprehensive periodontal examination was performed on each subject. The examination data were entered into the PAT and a risk score on a scale of 1 (lowest risk) to 5 (highest risk) was calculated for each subject. Fifty-two clinicians were divided into three groups; Group A consisted of 10 periodontists, Group B consisted of six periodontists, and Group C consisted of 36 general dentists. Each clinician assigned a risk score to each subject using the scale of 1 to 5. The scores assigned by each evaluator group were compared with one another and with PAT-calculated risk scores, and inter-evaluator and inter-group variation was determined[27]. A very large inter-evaluator variation was observed in all three groups (Fig 7). Fig 8 shows the distribution of dentist-assigned risk compared with PAT risk 3. Subjects assigned a risk score of 3 by the PAT are shown in the red circle while those that were assigned a score of 2 or 4 are shown in the inner circle outside the red circle, and those that were scored 1 or 5 are shown in the outermost circle. All three groups of evaluators behaved in the same manner. There was only a 20% agreement between scores assigned by the clinicians and the PAT[24]. In other words, relative to the risk assessment tool, 80% of the subjects were scored either too low or too high. Thus, over- or under-treatment would probably have been prescribed for about 80% of the risk group 3 subjects. If the PAT-calculated risk scores are accurate, as demonstrated by a previous study[28,29] and described below, then assessment of risk by subjective judgment appears to be too variable and inaccurate to

be useful in clinical decision-making. These observations demonstrate the need for a clinical method that is consistent, accurate and objective to assess risk for periodontitis. The periodontal assessment component of the OHIS™ and the PPRD described by Lang and Tonetti appear to satisfy that need[19,25].

Periodontal diagnosis

Early in the 20th Century, chronic periodontitis was known as pyorrhea. By mid-century, the terms gingivitis and periodontitis had been introduced and Gottlieb, Orban and others had suggested the existence of multiple forms of periodontitis. However, by the early 1960s only one form of periodontitis was recognised by the American Academy of Periodontology and it was designated chronic marginal periodontitis. The classification currently accepted by the American Academy of Periodontology (AAP) was devised by the 1999 International Workshop for Classification of Periodontal Diseases and Conditions (Table 1)[30]. Although this classification is attractive because of its completeness, it is deficient as a diagnostic tool for practical day-to-day use by clinicians and as a means of communication with patients and other health professionals. Chronic and aggressive periodontitis account by far for most cases of periodontitis encountered by clinicians. These two categories encompass a variety of patients ranging from those with a single bleeding pocket with mild loss of attachment and bone to those with terminal periodontitis affecting every tooth in the dentition. Use of qualitative terms such as localised and generalised, and mild, moderate and severe adds some specificity to the diagnostic terms chronic and aggressive periodontitis, but they provide no quantitative information about disease extent and severity, and the terminology becomes increasingly cumbersome as the description is made more precise (Table 2). Existing diagnostic acumen does not permit the clinician to distinguish among these large numbers of patients or to adequately express their condition.

Diagnosis of periodontal disease is generally based on the presence and extent of gingival inflammation frequently measured as bleeding on probing (BOP)[31,32], probing pocket depth (PPD)[33-35], clinical attachment level (CAL)[36-39], and the pattern and extent of alveolar bone loss assessed radiographically[38]. In addition, consideration may be given to age, gingival recession, tooth mobility, history, previous treatment, and signs and symptoms, including pain, ulceration, and microbial deposits[36,38,39]. A plethora of diagnostic terms has been used (Tables 2 and 3)[40]. Some of these have been based on severity, including mild, moderate and severe periodontitis, modified by terms for extent, including localised or generalised. In many clinical studies, investigators have used their own diagnoses and case definitions for substantial disease, such as advanced, serious or severe, based on varying combinations of CAL, PPD, BOP and extent and pattern of bone loss[22,37,41-43].

Table 1 Classification of periodontal disease.

Classification of periodontal disease
Gingivitis
Chronic periodontitis
Aggressive periodontitis
Periodontits as a manifestation of systemic disease
Necrotising periodontal disease
Abscesses of the periodontium
Periodontitis associated with endodontic lesions

Case definitions for mild, moderate and severe periodontitis were defined and used for the first time in the NHANES III national survey of periodontal disease conducted from 1988 to 1994[3], but different definitions were used in prior and subsequent national epidemiological surveys and a large number and variety of case definitions for periodontitis have been proposed and used in clinical studies (Table 3)[3,21,22,31,37,42-46]. Use of case definitions was an important step toward quantification of periodontal disease severity and extent. In order to establish case definitions for periodontitis, one must determine the threshold value for CAL and/or PPD and radiographic alveolar bone loss at a given site that constitutes unequivocal evidence of periodontitis at that site, and the number or percentage of such sites that must be present in a mouth to establish disease presence[38,39]. Selection of threshold values is of high significance, since minor changes result in major differences in who is designated as affected and who is not[40]. The most distinctive feature of these case definitions is their extreme variation and lack of consensus. Some of the definitions use a combination of PPD and CAL while others are based on either PPD

Table 2 Disease score, severity category and representative text descriptions.

Disease score	Severity category	Representative text descriptions
1	Health	Health
2–3	Gingivitis	Gingivitis
4–10	Mild periodontitis	Localised mild periodontitis Generalised mild periodontitis
11–36	Moderate periodontitis	Localised mild and moderate periodontitis Localised moderate periodontitis Generalised mild to moderate periodontitis Generalised mild and localised moderate periodontitis Generalised moderate periodontitis
37–100	Severe periodontitis	Localised mild and severe periodontitis Localised moderate and severe periodontitis Localised severe periodontitis Generalised mild to severe periodontitis Generalised mild and localised severe periodontitis Generalised moderate to severe periodontitis Generalised moderate and localised severe periodontitis Generalised severe periodontitis

or CAL alone. Only rarely has any indication of severity and extent of alveolar bone loss been included. Although two-level case definitions for periodontitis were accepted by a group of experts at the 5th European Workshop in Periodontology[47] and by an expert working group of the Centers for Disease Control and Prevention in the US[40], the two sets of definitions differed greatly in the required number of affected teeth or sites and the magnitude of attachment loss or pocket depth. There has been no widely accepted consensus, either on what threshold values for PPD, CAL and alveolar bone loss constitute a diseased site, or on the numbers of sites or teeth that must be affected to constitute a specific disease diagnosis for a person. A genuine consensus seems unlikely in the future, since case definitions generally have been tailored to the needs of the group of experts defining the consensus.

Carlos et al[48] were the first to develop an index for periodontitis based on disease severity and extent. They measured attachment loss and the percentage of teeth having attachment loss at 14 sites in one quadrant in one arch and 14 sites in the contra-lateral arch. The purpose of their effort was to be able to compare data from multiple populations and from time to time. Their index is inappropriate for routine clinical use, as the teeth in half of the mouth were not examined.

■ Quantification of risk and periodontal disease status

The Oral Health Information Suite (OHIS™) is an information system that compiles, analyses and quantifies clinical information about current oral health status and risk. The system generates a list of treatment options that may be effective for the patient's unique set of clinical and radiographic conditions, and graphically displays the outcome using the change in oral health status[19,24,28,29]. OHIS™ has been developed and patented (U.S. Patent #6,484,144) by PreViser, Inc., and is available on the Internet (www.previser.com). The Periodontal Assessment Tool (PAT) is an integral component of the OHIS™ specifically for periodontal disease. PAT reports oral health status as a disease score on a scale of 1 (healthy) to 100 (severe periodontitis) along with a 'best-fit' traditional description (Table 2) and risk score on a scale of 1 (very low risk) to 5 (very high

Table 3 Case definitions (modified from Page and Ecke[40]).

Study	Age (years)	Case definition (mm)
HES 1960–62[46]	18–79	1 tooth with pocket
HES 1971–74[46]	18–74	≥1 tooth with pocket
1981 National survey[45]	19+	Periodontitis: ≥1 site, ≥ 4 PPD Moderate: ≥1 site, 4–6 PPD Severe: >1 site, ≥6 PPD
NIDR 1984–85[21]	18–64	≥1 site, ≥3 CAL ≥1 site, 4–6 PPD ≥1 site, 5 CAL ≥1 site, ≥7 PPD
NHANES III[3]	30–90	Periodontitis: ≥1 site ≥3 CAL + ≥4 PPD (same site) Mild: ≥1 site 3 PPD or ≥1 Grade I Furcation + ≥3 PPD Moderate: ≥1 site ≥3 PPD or ≥2 sites ≥4 PPD or ≥1 Grade I Furcation + 3 PPD Severe: ≥2 sites ≥5 PPD or ≥4 sites ≥4 PPD or ≥1 Grade II Furcation ≥3 CAL ≥3 PPD
HANES 1999–2000[43]		Periodontitis: ≥3 sites, ≥ 4CAL + ≥2 sites ≥5 PPD
Machtei et al[37]		Established periodontitis: ≥2 teeth, ≥6 CAL + ≥1 site, ≥5 PPD
Moore et al[42]		Severe generalised periodontitis: ≥8 teeth, ≥5 CAL, ≥6 PPD
Burmeister et al[41]		Severe generalised periodontitis: ≥8 teeth, ≥5 CAL, at least 3 teeth (not 1st molars)
Beck et al[22]		Severe destructive periodontitis: ≥4 sites, ≥5 CAL; ≥1 same sites ≥4 PPD
Tomar and Asma[43]		Periodontitis: ≥1 site, ≥4 CAL, ≥4 PPD

risk). The disease score, risk score, and list of possible treatment interventions provide the clinician with precise, objective information that is comparative among all patients for a diagnosis, formulating needs-based treatment options and monitoring treatment outcomes. Its use is not intended to substitute for the clinician's training, experience or clinical judgment any more than a report on blood chemistry replaces the skills and knowledge of the endocrinologist.

Table 4 Information Required by the Periodontal Assessment Tool (PAT).

Information Required by the PAT
Patient age
Frequency of dental visits
Smoking history
Diabetes status
Oral hygiene status
History of pocket-reducing periodontal surgery
Pocket depth (deepest pocket in each sextant)
Bleeding on probing
Restorations below the gingival margin
Root calculus below the gingival margin
Radiographic bone height (greatest bone loss in each sextant)
Furcation involvement
Vertical bone lesions

PAT assessment of risk: the risk score

PAT determines risk from an algorithm that assigns relative weights to the various risk factors that enhance a patient's susceptibility for periodontitis. PAT is user friendly and requires only information gathered by a traditional periodontal examination (Table 4).

Validity and accuracy of risk scores were established from the clinical records and radiographs of 523 subjects enrolled in the VA Dental Longitudinal Study of Oral Health and Disease covering a period of 15 years[28,29]. Data from the baseline examinations of these subjects were entered into PAT and a risk score for periodontal deterioration was calculated for each subject. Actual periodontal status in terms of alveolar bone loss determined using digitised radiographs, and tooth loss determined from the clinical records, were assessed at years 3, 9 and 15. The strength of the association between the risk prediction and actual outcome was determined statistically.

The calculated risk scores were strong predictors of future periodontal status measured as worsening severity and extent of alveolar bone loss and tooth loss. Over the entire 15-year period, risk scores consistently ranked subject groups from least to most alveolar bone loss (Fig 9) and tooth loss (Fig 10). Risk groups differed greatly from one another. By year 3, the incidence rate of bone loss of risk group 5 was 3.7-fold greater than for risk group 2, and by year 15, loss of periodontally affected teeth was 22.7-fold greater than for risk group 2 ($p < 0.001$). As compared to a risk score of 2, the relative risk (RR) for any tooth loss was RR=3.2 for risk score of 3, RR=4.5 for risk score of 4 and RR=10.6 for risk score of 5.

PAT quantification of disease severity and extent: the disease score

The criteria defined for a system to quantify periodontal status numerically were:

1. Accuracy: scores generated must accurately reflect disease severity and extent.
2. Consistency: scores must be consistent among patients regardless of disease severity and the number of dentate sextants present in the mouth.

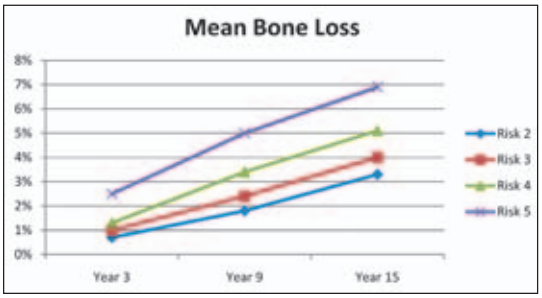

Fig 9 Mean alveolar bone loss from baseline for risk groups 2 to 5, at sites exceeding the threshold of 2% loss of alveolar bone height for all sites that could be compared. Reproduced from Page RC, Krall EA, Martin JA, Mancl LA, Garcia RI, Validity and accuracy of risk calculator in predicting periodontal disease. J Am Dent Assoc 2002;133:569–576. Copyright ©2002 American Dental Association. All rights reserved. Reprinted by permission.

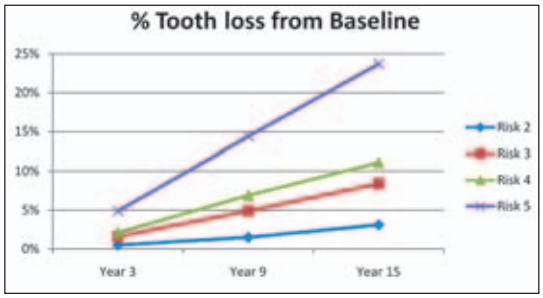

Fig 10 Mean tooth loss from baseline for risk groups 2 to 5, defined as the percentage of teeth present at baseline that were subsequently extracted. Reproduced from Page RC, Krall EA, Martin JA, Mancl LA, Garcia RI, Validity and accuracy of risk calculator in predicting periodontal disease. J Am Dent Assoc 2002;133:569–576. Copyright ©2002 American Dental Association. All rights reserved. Reprinted by permission.

Table 5 Severity ranking.

		Radiographic bone height distance from CEJ		
		<2 mm	**2–4 mm**	**>4 mm**
Pocket depth	**<5 mm**	(1)† Health‡ or (2) gingivitis	(3) Mild periodontitis	(4) Moderate periodontitis
	5–7 mm	(3) Mild periodontitis	(4) Moderate periodontitis	(5) Severe periodontitis
	>7 mm	(4) Moderate periodontitis	(5) Severe periodontitis	(5) Severe periodontitis

† Number in parentheses indicates the severity rank of the combination of pocket depth, radiographic bone height and inflammation.

‡ Health is defined as <5 mm pocket depth, <2 mm radiographic bone height and no inflammation, whereas gingivitis is defined when inflammation exists.

3. Understandable: scores must be clinically meaningful and understandable to clinicians, patients and all stakeholders.
4. Ease of use: the system must be user friendly and easy to use.

The PAT appears to satisfy these criteria. PAT is based on the concept of Carlos et al[48] that periodontal disease severity and extent can be quantified. The calculation of the PAT disease score requires only a small portion of the information gathered during a traditional periodontal examination (Table 4) and the computer performs all of the calculations described below automatically.

Each sextant of the dentition is assigned one of five severity levels using the traditional diagnostic terms of health and gingivitis, and mild, moderate and severe periodontitis. This classification of severity is based on BOP, PPD (categorised as <5 mm, 5–7 mm, >7 mm) and radiographic bone height (designated as <2 mm, 2–4 mm, >4 mm) from the cemento-enamel junction to the alveolar bone crest. Sextants are ranked as shown in Table 5 with bone loss and pocket depth given almost equal weighting.

Each severity level is assigned a numeric value, selected to ensure that all possible combinations of severity in a 1- to 6-sextant dentition represent a unique number that satisfies the requirement of accuracy and consistency among patients. The lowest number assignable to five severity level combinations in a 6-sextant dentition that ensures uniqueness regardless of the number of dentate sextants and their levels of severity was determined to be exponential values of the maximum possible sextants[3] plus 1, where the exponential value is based on a sequential ranking for severity from 0 for health to 4 for severe periodontitis. Thus, the assigned numerical values are severe periodontitis 7^4 (2401), moderate periodontitis 7^3 (343), mild periodontitis 7^2 (49), gingivitis 7^1 (7) and health 7^0 (1). Each assigned score equates to and represents a specific set of clinical and radiographic conditions.

The assigned scores are summed to yield the assigned score for the dentition (Fig 11).

Using assigned scores, the system correctly accounts for dentitions with any number of dentate sextants. For example, a patient with 5 dentate sextants all of which exhibit severe periodontitis has an assigned score of 5 x 2401, or 12,005, while a patient with 6 such sextants has an assigned score of 14,406 (Table 6). The same holds true for any number of sextants (Table 7).

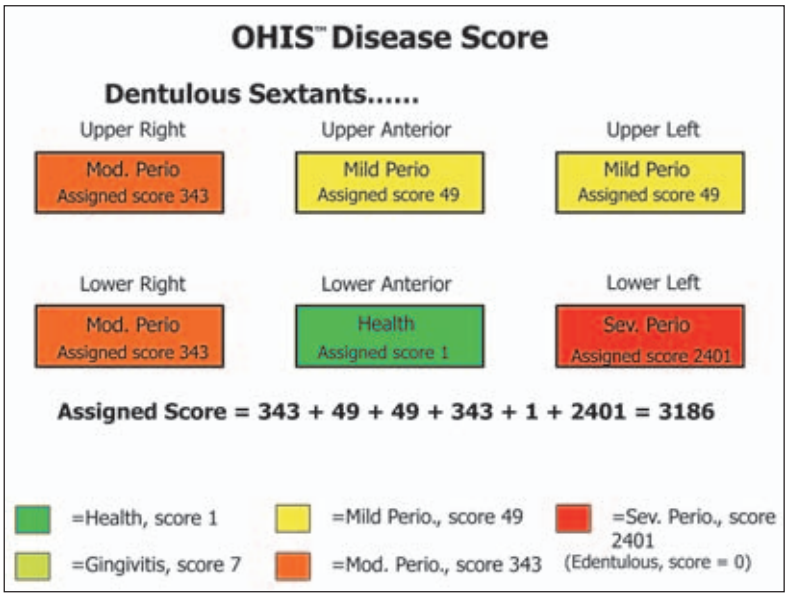

Fig 11 Calculation of assigned scores.

Table 6 Assigned scores.

	Sextants of dentition					
	One	Two	Three	Four	Five	Six
Assigned score, where health = 1	1	2	3	4	5	6
Assigned score, where severe disease = 2401	2401	4802	7203	9604	12,005	14,406
Number of possible unique sextant severity score combinations	5	15	35	70	126	210

Because assigned scores become too large to be clinically useful, they are converted to the disease score by correlating them to a scale of 1 to 100 where, regardless of the number of sextants present, 100 equals severe periodontitis throughout whatever dentition is present and 1 equals health (Fig 12, Table 8). This conversion results in compression of assigned scores for a 6-sextant dentition by a factor of 2.6 to 1 (from 210 sextant severity combinations to 100), and for a 5-sextant dentition by a factor of 1.26 to 1, etc. This compression is acceptable because the combined entities, although not identical, are very similar, and each digit in the 1–100 scale still designates a defined set of clinical and radiographic conditions (Table 9). For dentitions with less than 5 dentate sextants, there is no compression of the scale as the maximum number of unique severity and extent combinations is less than 100.

The disease score scale of 1–100 describes all possible severity combinations. It is consistent among patients regardless of the number of dentate sextants present or disease severity (Tables 7, 9 and 10). It is accurate in that it is based on accepted clinical disease descriptors (BOP, PPD and bone loss) and the numerical values and their change over time are easy to understand by patients, clinicians and other stakeholders. The system is user friendly in that it requires only a small fraction of the information collected during a traditional periodontal examination, PPD and bone loss measurements are categorised for ease of measurement and accuracy, and the computer accurately performs the calculations automatically.

The traditional diagnostic nomenclature that is the 'best-fit' for each combination of sextant-severity is applied to each disease score, as illustrated in Tables 2, 7 and 10. The disease score is not intended to replace but rather to supplement a diagnosis; nor is it intended to replace a comprehensive periodontal examination and charting.

Accuracy and validity of the disease scores was demonstrated by statistical analysis of the strength of agreement of disease scores, with actual periodontal status determined using values of alveolar bone height obtained from digitised radiographs. Bone height is generally accepted as a more accurate measure of periodontitis severity than PPD. While PPD, CAL and bone loss run in parallel at younger ages, because of gingival recession, increases in PPD do not keep pace with bone loss and CAL past middle age[40]. The study population consisted of subjects enrolled in the VA Dental Longitudinal Study of Oral Health and Disease[28]. Most of the 523 subjects had 6 dentate sextants. In order to test the accuracy of the disease scores for dentitions that had 5, 4, 3, 2, and 1 dentate sextants, we created data sets simulating these partially edentulous conditions by sequentially omitting from the calculations for each patient the PPD and bone height values for a sextant in the following sequence: upper right, upper left,

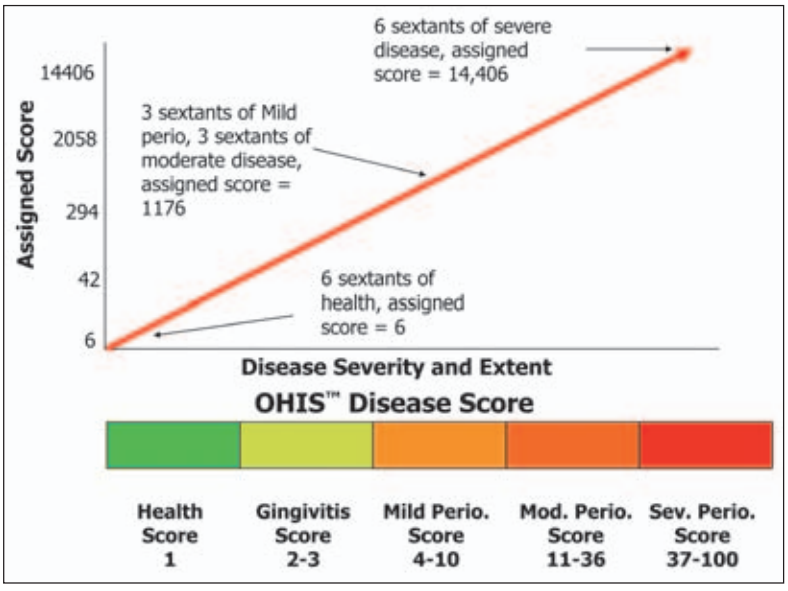

Fig 12 Conversion of assigned scores to disease scores.

Table 7 Disease score, assigned score, text description of all dentitions.

Disease score	'Best-fit' text description	Assigned score					
		6 dentate sextants	5 dentate sextants	4 dentate sextants	3 dentate sextants	2 dentate sextants	1 dentate sextant
100	Generalised severe periodontitis	14406	12005	9604	7203	4802	2401
99	Generalised severe periodontitis	12348	9947				
99	Generalised severe periodontitis	12054	9653				
98	Generalised severe periodontitis	12012	9611				
98	Generalised severe periodontitis	12006	9605				
97	Generalised severe periodontitis	10290	7889	7546	5145		
97	Generalised severe periodontitis	9996	7595				
96	Generalised severe periodontitis	9954	7573				
96	Generalised severe periodontitis	9948	7547				
95	Generalised severe periodontitis	9702	7301	7252	4851		
95	Generalised severe periodontitis	9660	7259				
94	Generalised severe periodontitis	9654	7253				
94	Generalised severe periodontitis	9618	7217	7210	4809		
93	Generalised severe periodontitis	9612	7211				
93	Generalised severe periodontitis	9606	7205	7204	4803		
92	Generalised moderate to severe periodontitis	8232	5831	5488		2744	
▼	▼	▼	▼	▼	▼	▼	▼
36	Generalised moderate periodontitis	2058	1715	1372	1029	686	343
36	Generalised moderate periodontitis	1764	1421	1078			
35	Generalised moderate periodontitis	1722	1379	1036			
35	Generalised moderate periodontitis	1716	1373	1030			
34	Generalised mild to moderate periodontitis	1470	1127		735		
34	Generalised mild to moderate periodontitis	1428	1085				
33	Generalised moderate periodontitis	1422	1079				
33	Generalised moderate periodontitis	1386	1043		693		
▼	▼	▼	▼	▼	▼	▼	▼
10	Generalised mild periodontitis	294	245	196	147	98	49
10	Generalised mild periodontitis	252	203	154			
10	Generalised mild periodontitis	246	197	148			
10	Generalised mild periodontitis	210	161		105		
9	Generalised mild periodontitis	204	155				
9	Generalised mild periodontitis	198	149		99		
9	Generalised mild periodontitis	168	119	112			
▼	▼	▼	▼	▼	▼	▼	▼
3	Gingivitis	42	35	28	21	14	7
3	Gingivitis	36	29	22			
3	Gingivitis	30	23		15		
2	Gingivitis	24	17	16		8	
2	Gingivitis	18	11	10	9		
2	Gingivitis	12					

Gaps in the table are indicted by filled triangles.

Table 8 Combinations of severity, disease score, and 'best-fit' text description.

Fully dentulous (6 sextants)					Partially edentulous (5 sextants)					Partially dentulous (3 sextants)						
Severe	Moderate	Mild	Gingivitis	Health	Severe	Moderate	Mild	Gingivitis	Health	Severe	Moderate	Mild	Gingivitis	Health	Disease Score	'Best-fit' text description
6	0	0	0	0	5	0	0	0	0	3	0	0	0	0	100	Generalised severe periodontitis
5	1	0	0	0	4	1	0	0	0						99	Generalised moderate to severe periodontitis
5	0	1	0	0	4	0	1	0	0						99	Generalised mild to severe periodontitis
5	0	0	1	0	4	0	0	1	0						98	Generalised severe periodontitis
5	0	0	0	1	4	0	0	0	1						98	Generalised severe periodontitis
4	2	0	0	0	3	2	0	0	0	2	1	0	0	0	97	Generalised moderate to severe periodontitis
4	1	1	0	0	3	1	1	0	0						97	Generalised mild to severe periodontitis
4	1	0	1	0	3	1	0	1	0						96	Generalised severe periodontitis
▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼
2	2	2	0	0	1	2	2	0	0	1	1	1	0	0	80	Generalised mild to severe periodontitis
2	2	1	1	0	1	2	1	1	0						80	Generalised mild to severe periodontitis
2	2	1	0	1	1	2	1	0	1						79	Generalised mild to severe periodontitis
2	2	0	2	0	1	2	0	2	0	1	1	0	1	0	79	Generalised moderate to severe periodontitis
2	2	0	1	1	1	2	0	1	1						78	Generalised moderate to severe periodontitis
2	2	0	0	2	1	2	0	0	2	1	1	0	0	1	78	Generalised moderate to severe periodontitis
▼		▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼
0	6	0	0	0	0	5	0	0	0	0	3	0	0	0	36	Generalised moderate periodontitis
0	5	1	0	0	0	4	1	0	0						36	Generalised mild to moderate periodontitis
0	5	0	1	0	0	4	0	1	0						35	Generalised moderate periodontitis
0	5	0	0	1	0	4	0	0	1						35	Generalised moderate periodontitis
▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼	▼
0	0	0	2	4	0	0	0	1	4	0	0	0	1	2	2	Gingivitis
0	0	0	1	5											2	Gingivitis
0	0	0	0	0	0	0	0	0	5	0	0	0	0	3	1	Health

Not all combinations of sextant severity levels, disease scores, and 'best-fit' text descriptions are shown. Gaps in the table are indicted by filled triangles.

Table 9 Examples of the relationship of assigned and disease scores.

Example 1: sextants of dentition where all sextants are healthy; health = value 1						
	One	**Two**	**Three**	**Four**	**Five**	**Six**
Assigned score	1	2	3	4	5	6
PAT disease score	1	1	1	1	1	1
Example 2: sextants of dentition where all sextants have mild disease; value = 49						
Assigned score	49	98	147	196	245	294
PAT disease score	10	10	10	10	10	10

lower left, lower right, and upper anterior. The number of subjects in each category ranged from 481 to 520. Minitab® Statistical Software was used to determine the Pearson correlation coefficient including p-value between mean bone height and disease scores (Fig 13). The correlation coefficients were 0.735, 0.683, 0.720, 0.694, 0.678, and 0.666 for dentitions with 6, 5, 4, 3, 2, and 1 dentulous sextants respectively. In each situation the p-value was 0.000. These results demonstrate a moderate to

Table 10 Relationship of sextant diagnoses, assigned score, and disease score.

Severe periodontitis	Moderate periodontitis	Mild periodontitis	Gingivitis	Health	Assigned score	Disease score
6	0	0	0	0	14406	100
5	1	0	0	0	12348	99
5	0	1	0	0	12054	99
5	0	0	1	0	12012	98
5	0	0	0	1	12006	98
4	2	0	0	0	10290	97
4	1	1	0	0	9996	97
4	1	0	1	0	9954	96
4	1	0	0	1	9948	96
4	0	2	0	0	9702	95
4	0	1	1	0	9660	95
4	0	1	0	1	9654	94
4	0	0	2	0	9618	94
4	0	0	1	1	9612	93
4	0	0	0	2	9606	93
3	3	0	0	0	8232	92
▼	▼	▼	▼	▼	▼	▼
3	0	0	0	3	7206	83
2	4	0	0	0	6174	82
▼	▼	▼	▼	▼	▼	▼
2	0	0	0	4	4806	65
1	5	0	0	0	4116	64
1	4	1	0	0	3822	64
1	0	0	0	5	2406	37
0	6	0	0	0	2058	36
▼	▼	▼	▼	▼	▼	▼
0	1	0	0	5	348	11
0	0	6	0	0	294	10
▼	▼	▼	▼	▼	▼	▼
0	0	1	0	5	54	4
0	0	0	6	0	42	3
▼	▼	▼	▼	▼	▼	▼
0	0	0	1	5	12	2
0	0	0	0	6	6	1

Gaps in the table are indicted by filled triangles.

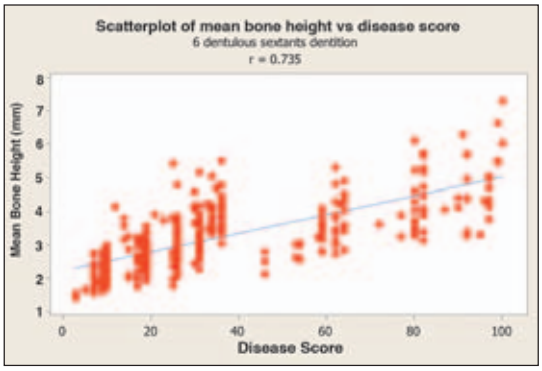

Fig 13 The plot of mean alveolar bone height (mm) and disease score for 481 subjects who had 6 dentulous sextants of teeth. The relationship between both variables was linear and Pearson's correlation coefficient was 0.735 with p-value 0.000.

strong linear relationship between periodontal disease scores and disease severity-extent as measured by mean bone loss, which means that higher disease scores are associated with greater mean bone loss.

Ranking disease severity is fundamental to calculating a disease score using the PreViser method. The validity of the logic for ranking severity was tested by asking 36 clinicians comprising periodontists, general dentists, and hygienists, to complete 7 of 9 cells in a semi-blank matrix with rows and columns identical to Table 5 using a 4-level severity ranking (gingivitis and mild, moderate and severe periodontitis). '1' was entered in the cell in the upper left and '5' was entered in the cell in the lower right. When this task was completed we then showed the clinicians Table 5 and asked them to indicate on a 10-point Likert scale their level of agreement (1 strongly disagree, 10 strongly agree). Four clinicians did not follow the instructions and were omitted from the statistical analysis. An attribute agreement analysis including calculating the kappa statistic was performed and the geometric similarity of uniquely different responses was determined. The analysis showed good agreement between the clinicians' choices and our standard. While the attribute agreement analysis is a measure of global agreement, the small differences between the clinicians' responses demonstrated very good agreement. The mean and median scores on a Likert scale of 1–10 were 7.875 and 8.000 respectively. These observations provided further evidence of good agreement with the model.

The agreement with our 'best-fit' nomenclature by clinicians was determined as follows. Each of 7 periodontists was asked to select from their patient pop-

ulation 25 records representative of a wide range of disease severity and extent. PAT used the requisite information from these records to determine a disease score and a diagnostic description for each patient expressed in traditional terminology. These were transmitted back to the periodontists, who were asked to indicate the extent of their agreement or disagreement with the diagnostic description on a Likert scale of 1 (strongly disagree) to 5 (strongly agree). Of 170 cases, the periodontists agreed or agreed strongly with the PreViser disease score and 'best-fit' text description 88% of the time. Only descriptive statistics are possible for this section since each periodontist subjectively rated his or her unique 25 records, which precluded analysis of a common set of data.

■ Discussion

We present a new approach for determining the severity and extent of a periodontal disease state. Our method uses the combination of sextants, each of which is diagnosed on the basis of severity as health or gingivitis, or mild, moderate or severe periodontitis, and correlates the results to a numerical l–100 scale. Disease severity of a sextant was determined by BOP, the deepest pocket and the site of greatest alveolar bone loss. The three-level categories for PPD and radiographic bone height were selected for clinical efficiency and accuracy of measurement, for their precision to determine treatment needs, their sensitivity to change and accuracy to describe the clinical situation. Other definitions of severity could have been chosen such as average pocket depth and mean radiographic bone loss for each sextant. It is notable that our method of quantifying a patient's severity and extent of periodontal disease will retain its validity with any set of rules or definitions that assign a severity category to a sextant. The new approach does not replace accepted text-linguistic diagnoses, but rather it supplements, augments and amplifies traditional diagnoses by objectively quantifying disease extent and severity and changes that may occur in disease status over time. Nor is the disease scoring system intended to replace a comprehensive periodontal examination and charting. The method is equally applicable to chronic and aggressive periodontitis, gingivitis and periodontal health.

Ideally, quantifying severity and extent of periodontal disease would require a diagnosis of disease severity for each tooth and consideration of all tooth severity combinations. Such an approach would result in more than 36,000 tooth combinations, a number too large for practical use. It is possible to use a sextant severity diagnosis. For patients with 6, 5, 4, 3, 2, or 1 dentate sextants, the number of possible sextant disease severity combinations is 210, 126, 70, 35, 15, and 5, respectively. We selected a 100-point scale because it is workable when the sextant is used as the unit of measure, although its use required compressing the numbers of sextant combinations. This compression is acceptable because the combined entities, although not identical, are very similar (Table 6).

Our method of numerically calculating disease severity and extent satisfies the requirement that each assigned score uniquely identifies a distinct severity-extent combination. The values assigned for severity sextants are the lowest possible number that satisfies this requirement and avoids differing sextant disease severity combinations sharing the same assigned score. Conversion of assigned scores to a scale of 1 (6 sextants of health) to 100 (6 sextants of severe periodontitis) creates 64 possible combinations where one or more sextants have severe periodontitis (our disease score 37–100), 26 combinations where no sextant has severe periodontitis and one or more has moderate periodontitis (our disease score 11–36), 7 combinations where no sextant has severe or moderate periodontitis and one or more has mild periodontitis (our disease score 4–10), and 2 combinations for the gingivitis category (our disease score 2–3) and only 1 for health (Table 2, Fig 12).

The disease score expands the number of diagnostic states within the chronic and aggressive periodontitis categories in the standard AAP classification[30] to 100 clinically definable conditions. It enables clear distinction among the large number of patients within these categories. Since the score is a measure of disease severity-extent it applies to chronic and aggressive periodontitis and does not distinguish between them. The method has precision and high sensitivity. It detects very minor changes in clinical conditions, either improvement or deterioration, and thereby enables monitoring of treatment outcomes. It is especially useful in preventive dentistry for detecting the very earliest stages of periodontitis and

Table 11 Precision of disease scores in detecting minor changes in disease status.

	Patient 1		Patient 2		Patient 3	
Sextant	**Description**	**Value**	**Description**	**Value**	**Description**	**Value**
Upper right	Severe periodontitis	2401	Severe periodontitis	2401	Severe periodontitis	2401
Upper anterior	Health	1	Gingivitis	7	Health	1
Upper left	Moderate periodontitis	343	Moderate periodontitis	343	Moderate periodontitis	343
Lower left	Mild periodontitis	49	Mild periodontitis	49	Mild periodontitis	343
Lower anterior	Health	1	Gingivitis	7	Health	1
Lower right	Health	1	Gingivitis	7	Health	1
Total assigned score		2796		2814		3090
Disease score		50		51		55

for monitoring adult orthodontic patients for onset of periodontitis during tooth movement. For example, a single 5 mm pocket or minor decrease alveolar bone height or both trigger an increase in disease score from 1 to 4.

The disease score is a clinically useful representation of periodontal status. It is determined by approximately equal weighting of pocket depth and bone loss, which is reflected in the variation from the linear trend line in Fig 13, and indicates that bone loss is not the gold standard outcome measure against which to test the method. In fact, no gold standard exists. The disease score is objective and correlates with radiographic bone level, making the score a useful guide of patient progress, with the caveat that the score is based on sextants and not on teeth or sites. This caveat precludes using the score as a definitive statement of oral disease or health status. The three patients described in Table 11 serve to illustrate the degree of precision of the system. Patient 1 differs from patient 2 only in having three sextants of gingivitis rather than health. Patient 3 differs from patient 1 only in having one sextant with moderate instead of mild periodontitis. Patient 2 differs from patient 3 by having three sextants of gingivitis instead of health and one sextant of mild rather than moderate periodontitis. The disease scores for patients 1–3 are 50, 51, and 55 respectively.

■ Concluding remarks

The Oral Health Information Suite is unique in clinical dentistry by virtue of its capacity to quantify the risk for future disease and the severity-extent of the current periodontal disease state, which enables consistently and objectively measuring changes in risk and disease status over time. These quantifications provide the means to apply guidelines to manage patients based on objective measures, to measure periodontal health care, and determine its effectiveness, including robust economic analyses of cost-benefit, cost-effectiveness, and value. Describing periodontal status by means of two scores improves communication and understanding of periodontal status, including its change over time, by all stakeholders of oral health care.

Objective measurement of outcomes is a mandatory first step in any effort to improve any aspect of a system of health care. The wellness model, by its focus on preventing disease, is enabled by our assessment of risk and disease status, and generation of customised treatment interventions. We believe that widespread use of the tool has the potential to reduce oral health care costs and improve the quality of oral health care.

■ References

1. Axelsson P, Paulander J, Svardstrom G, Kaiser H. Effects of population based preventive programs on oral health conditions. J Parodontol d'Implantol Orale 2000;19:255–269.
2. Axelsson P, Nystrom B, Lindhe J. The long-term effect of a plaque control program on tooth mortality, caries and periodontal disease in adults: results after 30 years of maintenance. J Clin Periodontol 2004;31:749–757.
3. Albandar JM, Brunelle JAS, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States 1988–1994. J Periodontol 1999;70:13–29.
4. Page RC, Beck JD. Risk assessment for periodontal diseases. Int Dent J 1997;47:61–87.

5. Page RC, Offenbacher S, Schroeder HE, Seymour GJ, Kornman KS. Advances in pathogenesis of periodontitis: summary of developments, clinical implications and future directions. Periodontol 2000 1997;14:216–248.
6. Grossi SG, Zambon JJ, Ho AW, Koch G, Dunford RG, Machtei EE et al. Assessment of risk for periodontal disease. 1. Risk indicators for attachment loss. J Periodontol 1994;65:260–267.
7. Midwest Business Group on Health, Juran Institute, Inc. The Severn Group, Inc. Reducing the costs of poor quality health care through responsible purchasing leadership. Chicago, IL 2002. Available at: http://www.mbgh.org/costquality.html. Accessed on 11 July 2004.
8. Bader JD, Ismail AI. A primer on outcomes in dentistry. J Public Health Dent 1999;59:131–135.
9. Eddy DM. Clinical decision making: from theory to practice. Anatomy of a decision. J Am Med Assoc 1990;263:441–443.
10. McGlynn EA. Six challenges in measuring the quality of health care. Health Aff 1997;16:7–21.
11. Reinhardt UE. Harness information to make health care work. Manag Care 2002;11:28.
12. Enthoven AC, Vorhaus CB. A vision of quality in health care delivery. Health Aff 1997;16:44–57.
13. Institute of Medicine. Available at: http://www.iom.edu/focuson.asp?id=8089 Accessed 11 July 2004.
14. Reinhardt UE. Quality: the Achilles' heel of market strategy. Hospitals 1988;62:24.
15. Reinhardt UE. Can efficiency in health care be left to the market? Health Polit Policy Law 2001;26:967–992.
16. Bader JD, Shugars DA. Variation, treatment outcomes, and practitioner guidelines in dental practice. J Dent Educ 1995; 59:61–95.
17. Grembowski D, Fiset L, Milgrom P, Forrester K, Spadafora A. Factors influencing the appropriateness of restorative dental treatment: an epidemiological perspective. J Public Health Dent 1997;57:19–30.
18. Shugars DA, Bader JD. Appropriateness of restorative treatment recommendations: a case for practice-based outcomes research. J Am Coll Dent 1992;59:7–13.
19. Page RC, Martin JA, Loeb CF. The oral health information suite (OHIS): its use in the management of periodontal disease. J Dent Educ 2005;69:509–520.
20. Marshall-Day CD, Stephens RG, Quigley LF Jr. Periodontal disease: prevalence and incidence. J Periodontol 1955;26: 185–203.
21. Brown LJ, Oliver RC, Loe H. Evaluating periodontal status of US employed adults. J Am Dent Assoc 1990;121:226–232.
22. Beck JD, Koch GG, Rozier RG, Tudor GE. Prevalence and risk indicators for periodontal attachment loss in a population of older community-dwelling blacks and whites. J Periodontol 1990;61:521–528.
23. Hart T, Kornman KS. Genetic factors in the pathogenesis of periodontitis. Periodontol 2000 1997;14:202–215.
24. Page RC, Martin JA, Loeb CF. Use of risk assessment in attaining and maintaining oral health. Compend Contin Educ Dent 2004;25:657-660, 663-666, 669, Erratum 2004;25:905.
25. Lang NP, Tonetti MS. Periodontal risk assessment (PRA) for patients in supportive periodontal therapy (SPT). Oral Health Prev Dent 2003;1:7–16.
26. Renvert S, Persson GR. Patient-based assessments of clinical periodontal conditions in relation to alveolar bone loss. J Clin Periodontol 2004;31:208–213.
27. Persson GR, Mancl LA, Martin JA, Page RC. Assessing periodontal disease risk. J Am Dent Assoc 2003;134:575–582.
28. Page RC, Krall EA, Martin JA, Mancl LA, Garcia RI. Validity and accuracy of a risk calculator in predicting periodontal disease. J Am Dent Assoc 2002;133:569–576.
29. Page RC, Martin JA, Krall EA, Mancl LA, Garcia RI. Longitudinal validation of a risk calculator for periodontal disease. J Clin Periodontol 2003;30:819–827.
30. Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol 1999;4:1–6.
31. Jenkins WMM, MacFarlane TW, Gilmour WH. Longitudinal study of untreated periodontitis. I. Clinical findings. J Clin Periodontol 1988;15:324–330.
32. Lang NP, Joss A, Tonetti MS. Monitoring disease supportive periodontal treatment by bleeding on probing. Periodontol 2000 1996;12:44–48.
33. Armitage GC. Manual periodontal probing in supportive periodontal treatment. Periodontol 2000 1996;12:33–39.
34. Greenstein G. Contemporary interpretation of probing depth assessments: diagnostic and therapeutic indications. A literature review. J Periodontol 1997;68:1194–1205.
35. Robertson PB, Buchanan SA, Armitage GC, Newbrun E, Taggart EJ, Hoover Cl. Evaluation of clinical and microbiological measures to predict treatment response in severe periodontitis. J Periodont Res 1987;22:230–232.
36. Armitage GC. Diagnosing periodontal diseases and monitoring the response to periodontal therapy. In: Perspectives on Oral Antimicrobial Therapeutics. Littleton, MA: PSG Publishing Co. 1987:47–60.
37. Machtei EE, Christerson LA, Grossi SG, Dunford R, Zambon JJ, Genco RJ. Clinical criteria for the definition of "established periodontitis". J Periodontol 1992;63:207–215.
38. Position paper. American Academy of Periodontology. Diagnosis of periodontal diseases. J Periodontol 2003;74:1237–1247.
39. Position paper. Epidemiology of periodontal diseases. American Academy of Periodontology. J Periodontol 2005;76:1406–1419.
40. Page RC, Eke PI. Case definitions for use in population-based surveillance of periodontitis. J Periodontol 2007;78(Suppl): 1387–139.
41. Burmeister AJ, Best AM, Palcanis KG, Caine FA, Ranney RR. Localized juvenile periodontitis and generalized severe periodontitis: clinical findings. J Clin Periodontol 1984;11: 181–192.
42. Moore WEC, Holderman LV, Smibert RM, Hash DE, Burmeister JA, Ranney RR. Bacteriology of severe periodontitis in young adult humans. Infect Immun 1982;38:1137–1148.
43. Tomar SL, Asma S. Smoking-attributable periodontitis in the United States: Findings from NHANES III. J Periodontol 2000;71:743–751.
44. Borrell LN, Burt BA, Taylor GW. Prevalence and trends in periodontitis in the USA: The NHANES 1988–2000. J Dent Res 2005;84:924–930.
45. Brown LJ, Oliver RC, Loe H. Periodontal diseases in the US in 1981: Prevalence, severity, extent, and role in tooth mortality. J Periodontol 1989;60:363–370.
46. Douglass CW, Gillings D, Sallecito W, Gammon M. National trends in the prevalence of periodontal disease. J Am Dent Assoc 1983;407–412.
47. Tonetti MS, Claffey N. Advances in the progression of periodontitis and proposal of definitions of a periodontitis case and disease progression for use in risk factor research. Group C consensus report of the 5th European workshop in periodontology. J Clin Periodontol 2005;32(Suppl 6):210–213.
48. Carlos JP, Wolfe MD Kingman A. The extent and severity index: a simple method for use in epidemiologic studies of periodontal disease. J Clin Periodontol 1986;13:500–505.