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Periodontal health

Niklaus P. Lang[1] | P. Mark Bartold[2]

[1]Universities of Bern and Zurich, Switzerland

[2]University of Adelaide, South Australia

Correspondence
Prof. Dr. Niklaus P. Lang, Professor Emeritus, University of Bern, Scheuermattweg 33, CH-3043 Uettligen, Switzerland.
Email: nplang@switzerland.net

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Abstract

Objectives: To date there is a paucity of documentation regarding definitions of periodontal health. This review considers the histological and clinical determinants of periodontal health for both intact and reduced periodontium and seeks to propose appropriate definitions according to treatment outcomes.

Importance: Defining periodontal health is can serve as a vital common reference point for assessing disease and determining meaningful treatment outcomes.

Findings: The multifactorial nature of periodontitis is accepted, and it is recognized that restoration of periodontal health will be defined by an individual's response to treatment, taking into account allostatic conditions.

Conclusions: It is proposed that there are 4 levels of periodontal health, depending on the state of the periodontium (structurally and clinically sound or reduced) and the relative treatment outcomes: (1) pristine periodontal health, with a structurally sound and uninflamed periodontium; (2) well-maintained clinical periodontal health, with a structurally and clinically sound (intact) periodontium; (3) periodontal disease stability, with a reduced periodontium, and (4) periodontal disease remission/control, with a reduced periodontium.

KEYWORDS

Clinical health, gingiva, periodontal remission, periodontal stability, pristine health

INTRODUCTION

"Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity."[1] In accordance with this definition by the World Health Organization, periodontal health should be defined as a state free from inflammatory periodontal disease that allows an individual to function normally and not suffer any consequences (mental or physical) as a result of past disease. However, while this definition is holistic and patient-outcome based, it seems an impractical and limiting definition for the purposes of clinical management of periodontal diseases. Therefore, a more practical definition of periodontal health would be a state free from inflammatory periodontal disease. This, in turn, means that absence of inflammation associated with gingivitis or periodontitis, as assessed clinically, is a prerequisite for defining periodontal health.

It is a matter of debate if altered morphological conditions resulting from previous exposure to disease processes (eg, gingival recession, loss of attachment, and bone loss) may be redefined as novel healthy conditions in the absence of clinical signs and symptoms of inflammation.

Interestingly, there are almost no studies or reports attempting to define periodontal health.[2] Defining periodontal health is very important if we are to have a common reference point for assessing periodontal disease and determining meaningful treatment outcomes. Health can be evaluated at both the histological and clinical levels and should be considered in the context of a preventive starting point and a therapeutic end point. Thus, periodontal health can exist before disease commences but, conversely, periodontal health can be restored to an anatomically reduced periodontium. In this review, the clinical criteria for distinguishing pristine health from health on a reduced periodontium are presented and discussed.

HISTOLOGICAL EVIDENCE OF HEALTH

Animal studies – pristine periodontal health and early gingival inflammation

During the 1970 and 1980s, various animal studies assessed the health status of gingival tissues that were exposed to oral biofilms.[3–5] These tissues were usually taken as baseline prior to commencement of plaque accumulation for studies on the development and pathogenesis of gingivitis. Clearly, most of the evidence for such healthy conditions (homeostasis) was defined by a complete absence of inflammatory infiltrate concomitant with gingival and plaque indices, yielding zero values.

Histologically, composition of the gingival biopsies was analyzed using a sampling microscope. It was stated that biopsies at day zero (commencement of plaque accumulation) did not contain any inflammatory cell infiltrates. The original delicate vascular capillary network of uninflamed gingival tissue has been described using vital microscopy, perfusion, and histological techniques in young dogs and cats.[4,5] However, after 4 days of plaque accumulation, a significant number of leukocytes were found in the collagen-poor connective tissue immediately beneath the junctional epithelium. The size of the infiltrated connective tissue (ICT) gradually increased during the experimental period and the volumetric density of collagen in the noninfiltrated connective tissue (NCT) was always much higher than in the ICT. In the ICT, however, collagen density remained constant throughout the study. By days 4 and 7, neutrophilic granulocytes constituted 60% to 70% of the leukocyte population. On day 28, the infiltrate comprised mainly mononuclear leukocytes, especially plasma cells; at that time neutrophils occupied only a small fraction of the infiltrate.[3]

The presence of biofilm and overt inflammation, occurring with eruption of deciduous teeth in dogs and cats, has been related to concomitant changes in gingival vascular morphology. Although localized, acute inflammation accompanies biofilm formation at the time of weaning, it rarely develops into a chronic inflammation. Infiltration of gingival tissue by chronic inflammatory cells occurred in only a few specimens and was associated with increased biofilm and replacement of the gingival vessel network by loop patterns. This, in turn, meant the original delicate vascular network was replaced with loop configurations of capillaries once challenged by biofilm-induced inflammation.[4]

A subsequent study investigated whether the regular vasculature of noninflamed marginal gingiva would become re-established following plaque control, scaling, and gingivectomy in dogs with and without pre-experimental gingivitis at 4, 8, and 12 weeks.[5] Noninflamed gingiva that was previously inflamed was characterized by a series of looped vessels that could be readily distinguished from the regular network of vessels described for the marginal gingiva that had neither been inflamed nor resected previously.[5]

Human histological studies on health and gingivitis

The cellular composition of developing infiltrated connective tissue was analyzed in human volunteers participating in a 21 day experimental gingivitis model in which oral hygiene practices were abolished.[6] As the clinical index (gingival index) for inflammation increased, the volumetric density of infiltrated connective tissue within the noninfiltrated connective tissue in an area subjacent to the junctional epithelium increased significantly and almost linearly.[7] The infiltrated connective tissue demonstrated a significant increase in lymphocytes from health to inflammation (17.0% to 29.9%) concomitant with a decrease in the numerical density of fibroblasts, from 48.1% to 34.9%. Moreover, the numerical density of polymorphonuclear leukocytes was between 20.8% and 22.6% at all stages, from health to gingivitis. These results indicate that an inflammatory infiltrate subjacent to the junctional epithelium is always present in gingival tissues that are clinically healthy.

To study the influence of long-term gingival health, 5 dental hygienists with optimal personal oral hygiene were supervised regarding their oral hygiene performance for 6 months.[8] It was assured that at all observation times (0, 1, 4, and 6 months) clinical indices for plaque and inflammation were close to zero. The volumetric density of infiltrated connective tissue versus noninfiltrated connective tissue decreased significantly from month 1 to month 4. This indicated that a long-standing optimal oral hygiene regime is necessary for any histological improvement of the inflammatory infiltrate. Nevertheless, even after 6 months of supervised oral hygiene practices, the infiltrate was still present.[8]

While the numerical density of lymphocytes within the infiltrate decreased significantly, from 18.4% to 5.6%, after 6 months of meticulous oral hygiene, the numerical density of fibroblasts increased significantly, from 57.7% to 71.0%. This clearly reflected a positive healing outcome. However, it must be recognized that even during this 6 month period of optimal oral hygiene, the numerical density of polymorphonuclear leukocytes remained relatively stable, varying from 20.6% to 17.7%. This, in turn, means that in humans a status of clinically healthy gingiva, even for a prolonged period, is always histologically characterized by a small inflammatory cell infiltrate.[7,8] This indicates polymorphonuclear leukocyte surveillance, which is a very important physiological (not pathological) process. Most recently, in human biopsies from clinically healthy sites, memory B cells were identified within the connective tissue subjacent to the junctional epithelium. This suggests a role for memory B cells in maintaining homeostasis.[9]

Thus, the term *pristine clinical health* represents a rare, but realistic entity, ie, no attachment loss, no bleeding on probing (BoP), no sulcular probing >3 mm and no redness, clinical swelling/edema, or pus. It should be recognized that this condition is associated with physiological immune surveillance rather than pathological inflammation. The term *clinically healthy* should refer to tissue that demonstrates an absence, or very low level, of clinical indicators of inflammation such as BoP and inflammatory markers in gingival crevicular fluid. This review did not consider gingival crevicular fluid biomarker research in periodontal health and disease, as crevicular fluid analysis is not generally practical to implement in clinical practice at this time due to the need for specialized equipment.

DETERMINANTS OF CLINICAL PERIODONTAL HEALTH

No longer can periodontal diseases be considered simple bacterial infections. Rather, they are complex diseases of multifactorial nature involving an intricate interplay between the subgingival microbiota, the host immune and inflammatory responses, and environmental modifying factors.[10] Thus, periodontal health must not be considered solely in the context of plaque/bacteria levels and control but must embrace a holistic consideration and evaluation of all factors responsible for the emergence of disease, as well as the restoration and maintenance of health.[11]

Determinants of periodontal health fall into 3 major categories, namely, microbiological, host, and environment (Table 1). Because many of these factors are addressed in the paper dealing with plaque induced gingival diseases,[12] we will only consider the clinical indicators of clinical periodontal health in this article.

The relevance of recognizing such important determinants of periodontal health and disease as controllable and uncontrollable predisposing and modifying factors cannot be underestimated, and their assessment for each patient is crucial to attaining and maintaining clinical periodontal health. In this context, predisposing factors are defined as any agent or condition that contributes to the accumulation of dental plaque (eg, tooth anatomy, tooth position, restorations). Modifying factors are defined as any agent or condition that alters the way in which an individual responds to subgingival plaque accumulation (eg, smoking, systemic conditions, medications). The threshold(s) to establish when such factors are controlled versus not fully controlled await further elaboration, but it is reasonable to expect that many factors will be determined controllable (eg, removal of overhangs, smoking cessation, good diabetes control) while others will not (eg, genetic predisposition, immune status, use of critical medications).

TABLE 1 Determinants of clinical periodontal health

Microbiological Determinants of Clinical Periodontal Health
Supragingival plaque composition
Subgingival biofilm composition
Host Determinants of Clinical Periodontal Health
1. Local predisposing factors
1.1 Periodontal pockets
1.2 Dental restorations
1.3 Root anatomy
1.4 Tooth position and crowding
2. Systemic modifying factors
2.1 Host immune function
2.2 Systemic health
2.3 Genetics
Environmental Determinants of Clinical Periodontal Health
Smoking
Medications
Stress
Nutrition

PLAQUE AND CLINICAL PERIODONTAL HEALTH

Subgingival biofilm

The bacterial composition of the subgingival biofilm associated with gingivitis and periodontitis results from dynamic interactions with its microenvironment. In general, the microbial composition is a collection of commensal organisms that coexist in relative harmony. However, should the environment change, either as a result of inflammation within the gingival tissues or other, as yet unidentified, processes within the biofilm, a state of dysbiosis may result in the overgrowth of more virulent components of the biofilm, with ensuing exacerbation of periodontal inflammation.[13] Thus, gingivitis can be considered a relatively nonspecific inflammatory response to nonspecific (indigenous) subgingival microbiota. With the resultant inflammation and development of periodontitis, a shift in microbial composition occurs and several putative pathogens emerge, leading to heightened host-driven tissue damage. Thus, for periodontal health to be attained, or maintained, the composition of the subgingival microbiota needs to be redirected toward one compatible with gingival health.[14]

Oral hygiene

Good oral hygiene has always been considered a mainstay of periodontal health.[15] It is usually achieved by a combination of good personal oral hygiene and regular professional care.[16,17] It must be remembered that plaque accounts for only 20% of the direct risk of developing periodontitis, thus it must not be forgotten that the remaining 80% of direct and indirect risk and modifying factors may be responsible for the development of periodontal diseases.[18] While oral hygiene remains the most important factor in obtaining and maintaining periodontal health, it should not be the sole focus of attention. Additional factors must be addressed in the quest for attaining or maintaining periodontal health.

INDICATORS OF CLINICAL PERIODONTAL HEALTH

In its pristine form, periodontal health would be defined as the absence of histological evidence of periodontal inflammation and no evidence of anatomical change to the periodontium. However, it must be recognized that in most (if not all) adults this is unlikely. Therefore, the term *clinically healthy* should be adopted to cover the absence of (or very significant reduction in) clinical periodontal inflammation on either an anatomically intact periodontium or a reduced periodontium. Furthermore, a compromised definition or paradigm for periodontal clinical health needs to be developed for individuals who have experienced periodontal disease (gingivitis or periodontitis), undergone treatment, then returned to a state of clinical health on either a full periodontium (in the case of gingivitis) or a reduced periodontium (in the case of periodontitis).

Bleeding on probing

Monitoring health or inflammation of the gingival tissues is best documented by the parameter of BoP.[19] Bleeding on probing, in the absence of pocketing, should be understood as bleeding provoked in the coronal marginal gingiva following the application of pressure to the lateral wall of a periodontal sulcus or pocket, reflecting microulceration of the sulcus lining. However, BoP is usually measured as bleeding provoked by applying a probe to the bottom of a sulcus/pocket. In most studies on BoP as a clinical parameter, this latter definition is applied. The histological characteristics of the gingival tissues associated with BoP have been evaluated.[20] Sites that bleed following probing with light pressure applied to the tissues (0.25 N) are associated with a significantly increased percentage of cell-rich and collagen-reduced connective tissue but no increase in vascularity or vessel lumen size that would justify the bleeding tendency. Moreover, clinical and histological data suggest that bleeding is an earlier sign of gingivitis than are the visual signs of inflammation (redness and swelling).

Obviously, BoP may be provoked by trauma to the tissues using a periodontal probe. Hence, the probing pressure to be applied to the tissue (bottom of the sulcus/pocket) when evaluating BoP should not be sufficient to create trauma; rather it should only be enough to provoke tissue to bleed if there is increased blood vessel fragility resulting from inflammation. It has been demonstrated that BoP provoked with pressures greater than 0.25 N results in false-positive readings. By incrementally increasing pressure by 0.25 N, an increase of approximately 13% in BoP sites has been noted.[21,22]

An early retrospective study evaluated the prognostic value of BoP compared with repeated visits in identifying sites at risk for periodontal attachment loss during supportive care following periodontal therapy.[23] The results indicated that sites with a probing depth of ≥5 mm had significantly higher incidence of BoP. Sites with an incidence of BoP at 4 of 4 visits had a 30% chance of attachment loss. This decreased to 14% with an incidence of BoP at 3 of 4 visits, to 6% with an incidence of BoP at 2 of 4 visits, to 3% with an incidence of BoP at 1 of 4 visits, and finally, to 1.5% with no BoP at any of 4 visits. Sensitivity and predictability calculations revealed that BoP is a limited but useful prognostic indicator in monitoring periodontal tissue after active therapy.[23]

Subsequent studies investigated the predictive value of absence of BoP as an indicator for periodontal stability.[24,25] While the positive predictive value remained rather low for repeated BoP prevalence (≤30%), the negative predictive value in the same studies was nearly 100%. This demonstrated that absence of BoP at repeated examinations represented periodontal health and was a very reliable indicator for periodontal stability.[24] Hence, from a clinical point of view, absence of BoP would indicate clinically healthy periodontal tissue. These findings were later validated in a prospective follow-up study applying BoP as a clinical indicator for disease progression or periodontal stability.[25]

As the absence of BoP at 0.25 N indicates periodontal health, with a negative predictive value of 98% to 99%, this clinical parameter appears the most reliable for monitoring patients in daily practice over time.[24,25] Nonbleeding sites may be considered as clinically healthy and periodontally stable. It would be logical to assume that the positive outcomes of periodontal treatment, in receptive patients, would reach a status of nonbleeding on gentle probing.

Because various factors, such as probe dimension, angulation of probe, and applied pressure, can affect the assessment of gingival inflammation, it is imperative to standardize BoP as resulting from a defined level of force (pressure to the tissue), preferably not exceeding 0.25 N.[26]

A multilevel analysis of various site-specific and patient-related factors influencing BoP in 601 adult patients demonstrated that BoP may be associated with site-specific factors (periodontal probing depth [PPD], tooth type, and aspects) as well as patient-related factors (eg, sex and smoking status).[27] While the severity and extent of gingival bleeding are often associated with the degree of bacterial plaque accumulation, it is noted that other factors can lead to increased gingival bleeding. For example, vitamin C deficiency or ingestion of aspirin can cause significant gingival bleeding through mechanisms that may not be primarily related to plaque accumulation.[28,29] In a recent retrospective study of 445 patients in periodontal supportive therapy for at least 5 years, increased mean BoP in patients on supportive periodontal therapy was related to disease severity and periodontal instability irrespective of smoking status; smokers demonstrated lower mean BoP concomitant with increased prevalence of residual PPD.[30]

Standardization of periodontal probe design

The characteristics of an ideal periodontal probe will be central to a future determination of periodontal health. There is need to develop an International Organization for Standardization periodontal probe to ensure not only that probe dimension is consistent but that probing force is standardized to 0.25 N, thus removing the confounding issue of BoP induced by too much pressure, as well as unnecessary bleeding resulting from trauma. This critical issue is discussed in more detail by Trombelli and Tatakis (fourth paper for Working Group 1; in this issue).[31]

Periodontal probing depth

While it would seem intuitive that shallow pockets are consistent with health and deep pockets consistent with disease, there is ample evidence to indicate this may not necessarily be true. For example, deep pockets may remain stable and uninflamed, particularly if careful supportive periodontal care is provided, over very long periods of time.[32,33] Thus, deep pockets may exist as so-called healthy pockets.

It is important to recognize that, following successful treatment, recurrent inflammatory periodontitis can recur at individual sites despite most of the dentition remaining well maintained and in a state of relative health.[33] This has been interpreted to indicate that mean values of clinical parameters such as PPD, attachment levels, and bone height are not adequate predictors for sites that may become

reinfected and undergo recurrent disease.[33] Thus, PPD or probing attachment levels alone should not be used as evidence of gingival health or disease. They must be considered in conjunction with other important clinical parameters such as BoP, as well as modifying and predisposing factors. This highlights, as stated above, that the most useful indicator of disease is clinical evidence of inflammation, and that historical evidence of disease (increased PPD, recession and loss of attachment, bone loss) may be of less relevance in the context of periodontal health on a reduced periodontium.[34]

Radiographic features of periodontal health

Radiographic assessment forms a critical component of clinical assessment of the periodontium. Radiographic features of a normal, anatomically intact periodontium would include an intact lamina dura (both laterally and at the alveolar crest), no evidence of bone loss in furcation areas, and a 2 mm distance, on average, from the most coronal portion of the alveolar bone crest (AC) to the cementoenamel junction (CEJ). The distance from the CEJ to AC in healthy individuals can vary between 1.0 and 3.0 mm.[35,36] It is important to note that factors such as patient age, tooth type, angulation of teeth, and severe attrition can all influence the CEJ-AC height, thus caution must be exercised when assessing this parameter as a measure of periodontal health. While periodontal ligament space is also appraised radiographically, it can vary and is not considered a useful indicator of health (see section below on tooth mobility).

Once periodontitis has developed, by definition, alveolar bone loss has occurred because of the inflammatory process. Thus, clinical periodontal health on a reduced periodontium cannot be determined using radiographs alone; they provide information regarding historical destruction and are of value for longitudinal determination of progressive bone loss.

Tooth mobility

Clinicians often assess the status of a tooth by estimating its mobility. Because teeth are not ankylosed, or *osseointegrated*, as are implants, but are suspended in the alveolar bone by a network of collagenous fibers, they exhibit a degree of physiological mobility. This is usually assessed as the amplitude of crown displacement resulting from the application of a defined force.[37] The magnitude of this movement has been used to distinguish between physiological and pathological tooth mobility, with up to 0.2 mm regarded as physiological. In teeth with noninflamed periodontal tissue, 2 fundamental histological factors determine tooth mobility: 1) the height of the periodontal tissue support and 2) the width of the periodontal ligament.

In a clinically healthy situation, increased tooth mobility associated with widening of the periodontal ligament most likely represents a tooth in occlusal trauma. Furthermore, increased tooth mobility cannot be used as a sign of disease for a tooth with a reduced, but healthy, periodontium. Such increased mobility may be permanently increased due to reduced periodontal support, yet the periodontium may be completely healthy. If the height of the periodontal support is reduced but the width of the ligament is unchanged (approximately 250 μm), it should be appreciated that the amplitude of root mobility within the remaining periodontium is the same as for a tooth with normal height of periodontal support. Hence, the so-called hypermobility of a periodontally healthy tooth with reduced support but normal width of periodontal ligament should be considered physiological tooth mobility.

Increased tooth mobility due to a widening in the periodontal ligament is the result of uni- or multidirectional forces to the crown that are sufficiently high and frequent to induce resorption of the alveolar bone walls in pressure zones. In a series of controlled animal experimental studies in periodontally healthy teeth, the alveolar bone resorption resulted in increased tooth mobility but no loss of connective tissue attachment, irrespective of the height of the supportive bone.[38,39] Because alveolar bone loss has been demonstrated to be reversible upon cessation of applied forces, it was concluded that increased tooth mobility as a result of a widened periodontal ligament represents a physiological adaptation to altered function rather than a sign of pathology.[37] Hence, tooth mobility is not recommended to be used as a sign of either health or disease status.

PERIODONTAL HEALTH AND TREATMENT TARGETS FOR A DISEASED OR REDUCED PERIODONTIUM

While maintaining periodontal health over a lifetime with no adverse changes in the periodontium is desirable, it must be recognized this is unlikely for most of the population.

In Table 2, periodontal conditions and their expected outcomes with respect to periodontal health are detailed within the context of an intact and a reduced periodontium. For the treatment of gingivitis, it is not realistic to return to pristine periodontal health; restoration to full clinical health (no BoP, no anatomical loss of periodontal structures) would be expected following removal of biofilm and calculus and ongoing effective oral hygiene and maintenance. In treating periodontitis, which by definition manifests as loss of periodontal support (both attachment and bone), restoration to pre-disease attachment and bone levels is an unlikely event at the majority of sites; therapeutic targets are to control local and modifying factors, minimize inflammation, and stabilize attachment and bone. Therefore, for a large proportion of the population, the issue of periodontal health must be considered in the context of returning to clinical health from disease (either gingivitis or periodontitis) and what this return entails. According to recent epidemiological data, gingivitis affects up to 95% of the population and chronic periodontitis up to 60% to 65% of the North American population 65 years and older.[40,41] While some variance is to be expected across communities, these figures are likely to be relatively accurate for most populations worldwide.

In the context of our current understanding of the multifactorial nature of (plaque-associated) periodontal diseases, reducing

TABLE 2 Outcomes of periodontal health for plaque-associated periodontal diseases

				Periodontitis (reduced periodontium)	
	Pristine periodontal health	**Clinical periodontal health (intact periodontium)**	**Gingivitis**	**Periodontal disease stability**	**Periodontal disease remission/control**
Bleeding on probing	No	No/Minimal	Yes	No/Minimal	Significantly reduced
Normal gingival sulcus depth	Yes	Yes	Yes	No	No
Normal bone heights	Yes	Yes	Yes	No	No
Modifying factors	Controlled	Controlled	May be present	Controlled	Not fully controlled
Predisposing factors	Controlled	Controlled	May be present	Controlled	Not fully controlled

Pristine periodontal health is defined as no bleeding on probing and no anatomical loss of periodontal structures. *Gingivitis* is defined as a nonspecific inflammatory reaction to a nonspecific accumulation of plaque that is confined to the gingival tissue, with no underlying destruction of the attachment apparatus. *Periodontitis* covers the major plaque-associated periodontal diseases, and treatment outcomes are expected to be either *periodontal disease stability* or *periodontal disease remission/control*. *Periodontal disease stability* is defined as a state in which the periodontitis has been successfully treated and clinical signs of the disease do not appear to worsen in extent or severity despite the presence of a reduced periodontium. *Periodontal disease remission/control* is defined as a period in the course of disease when symptoms become less severe but may not be fully resolved.

inflammation and improving clinical health for a reduced periodontium may be achieved at 2 levels, namely stability and remission/control. These are variants of therapeutic outcomes to restore health on a reduced periodontium.

Periodontal disease stability will be defined as a state in which periodontitis has been successfully treated through control of local and systemic factors, resulting in minimal BoP, optimal improvements in PPD and attachment levels, and a lack of progressive destruction. The principal signs of successful periodontal treatment would be as detailed above with regard to BoP, PPD, and clinical attachment levels. In addition, control of modifying factors such as reduction of daily cigarette smoking and good control of diabetes are achieved. In many respects, attainment of periodontal disease stability can be considered a prognostic definition.

Periodontal disease remission/control is defined as a period in the course of disease during which treatment has resulted in reduction (although not total resolution) of inflammation and some improvement in PPD and attachment levels, but not optimal control of local or systemic contributing factors. This may be a reasonable treatment outcome for individuals with uncontrollable modifying factors. Indeed for many chronic inflammatory medical conditions (eg, diabetes, cardiovascular disease, hyperlipidemia, and rheumatoid arthritis), the goal of disease remission is important and is based on the emerging concept of *treat to target*.[42] This is a treatment paradigm that utilizes specific and well-defined treatment outcomes to monitor the control of the clinical signs and symptoms of a disease and is aimed at attaining a state of putative *health*. For patients with longstanding disease and/or uncontrolled contributing factors, for example smoking or diabetes, low disease activity may be an acceptable therapeutic goal. Thus, the definition of disease remission/control is related to the achievement of other (ie, different from those obtained in the disease stability definition) treatment end points that testify to an improvement in periodontal condition (with respect to baseline status) that, if not achieved, may be associated with progression of attachment loss.

If the concept of disease remission/control is embraced as a treatment target for the management of periodontal diseases, periodontal treatment will move from a solely biofilm-based protocol to a more holistic, inflammation-based model. It is important to note that this model does not discount or diminish the importance of the periodontal microbiome, but refocuses attention on controlling the inflammation to control the infection and the ongoing destruction of the periodontium.

This model requires that modifiable indicators of periodontitis such as traditional markers of periodontitis (attachment and bone loss and PPD), modifiable inflammatory markers (periodontal inflammation score, inflammatory mediators in gingival crevicular fluid) and modifiable systemic risk factors (eg, diabetes, smoking) be accounted for when evaluating the outcome of periodontal treatment and whether there has been a positive response to treatment consistent with progression toward periodontal health and stability. Thus, specific measurable biological and clinical outcomes should be determined to form the basis for assessment of periodontal health based largely (but not exclusively) on the inflammatory response.

CONCLUSIONS

It is proposed that there are 4 levels of periodontal health, depending upon whether the periodontium has normal attachment and

bone level or reduced support, as well as the ability to control modifying factors and relative treatment outcomes. These 4 categories include 1) *pristine periodontal health*, defined as a total absence of clinical inflammation and physiological immune surveillance on a periodontium with normal support (no attachment or bone loss). Pristine periodontal health is not likely to be observed clinically; 2) *clinical periodontal health*, characterized by an absence or minimal levels of clinical inflammation in a periodontium with normal support; 3) *periodontal disease stability* in a reduced periodontium; 4) *periodontal disease remission/control* in a reduced periodontium. Periodontal disease stability and periodontal disease remission/control are differentiated based on the ability to control modifying factors and therapeutic response. Stability is characterized by minimal inflammation and optimal therapeutic response, with control of modifiable risk factors; it is a major treatment goal for periodontitis. For patients in whom it is not possible to fully control modifying and predisposing factors, remission/control may be the more realistically achievable therapeutic goal. Remission/control is characterized by a significant decrease in inflammation, some improvement in other clinical parameters, and a stabilization of disease progression. Ideally, restoration to periodontal stability should be a major treatment goal and can be attained by controlling inflammation and infection, reducing predisposing factors, and controlling any modifying factors. While remission/control should be a clear target, based on available evidence, low disease activity may be an acceptable alternative therapeutic goal, particularly in long-standing disease.

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