



Periodontal health and gingival diseases and conditions on an intact and a reduced periodontium: Consensus report of workgroup 1 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions

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Abstract

Periodontal health is defined by absence of clinically detectable inflammation. There is a biological level of immune surveillance that is consistent with clinical gingival health and homeostasis. Clinical gingival health may be found in a periodontium that is intact, i.e. without clinical attachment loss or bone loss, and on a reduced periodontium in either a non-periodontitis patient (e.g. in patients with some form of gingival recession or following crown lengthening surgery) or in a patient with a history of periodontitis who is currently periodontally stable. Clinical gingival health can be restored following treatment of gingivitis and periodontitis. However, the treated and stable periodontitis patient with current gingival health remains at increased risk of recurrent periodontitis, and accordingly, must be closely monitored.

Two broad categories of gingival diseases include non-dental plaque biofilm-induced gingival diseases and dental plaque-induced gingivitis. Non-dental plaque biofilm-induced gingival diseases include a variety of conditions that are not caused by plaque and usually do not resolve following plaque removal. Such lesions may be manifestations of a systemic condition or may be localized to the oral cavity. Dental plaque-induced gingivitis has a variety of clinical signs and symptoms, and both local predisposing factors and systemic modifying factors can affect its extent, severity, and progression. Dental plaque-induced gingivitis may arise on an intact periodontium or on a reduced periodontium in either a non-periodontitis patient or in a currently stable “periodontitis patient” i.e. successfully treated, in whom clinical inflammation has been eliminated (or substantially reduced). A periodontitis patient with gingival inflammation remains a periodontitis patient (Figure 1), and comprehensive risk assessment and management are imperative to ensure early prevention and/or treatment of recurrent/progressive periodontitis.

Precision dental medicine defines a patient-centered approach to care, and therefore, creates differences in the way in which a “case” of gingival health or gingivitis is defined for clinical practice as opposed to epidemiologically in population prevalence surveys. Thus, case definitions of gingival health and gingivitis are presented for both purposes. While gingival health and gingivitis have many clinical features, case definitions are primarily predicated on presence or absence of bleeding on probing. Here we classify gingival health and gingival diseases/conditions, along with a summary table of diagnostic features for defining health and gingivitis in various clinical situations.

KEYWORDS

allergic reaction, amalgam tattoo, aspergillosis, biofilm, blastomycosis, calcifying fibroblastic granuloma, candidosis, chemical trauma, clinical health, coccidioidomycosis, condylomata acuminatum, contact allergy, coxsackie virus, Crohn's disease, dental plaque-induced gingivitis, disease control, disease remission, disease stability, drug-induced gingival enlargement, drug-induced pigmentation, dysbiosis, erythema multiforme, erythroplakia, factitious injury, fibrous epulis, focal epithelial hyperplasia, frictional keratosis, geotricosis, gingival pigmentation, hand foot and mouth, hereditary gingival fibromatosis, herpangina, herpes simplex, histoplasmosis, Hodgkin lymphoma, hyperglycemia, hyposalivation, intact periodontium, leukemia, leukoplakia, lichen planus, local risk factors, lupus erythematosus, melanoplakia, Melkersson-Rosenthal, menstrual cycle, modifying factors, molluscum contagiosum, mucormycosis, *Mycobacterium tuberculosis*, necrotizing periodontal diseases, *Neisseria gonorrhoeae*, non-dental plaque-induced gingival conditions, non-Hodgkin lymphoma, oral contraceptive, orofacial



granulomatosis, paracoccidioidomycosis, pemphigoid, pemphigus vulgaris, periodontal disease, peripheral giant cell granuloma, plasma cell gingivitis, predisposing factors, pregnancy, puberty, pyogenic granuloma, reduced periodontium, resolution of inflammation, restoration margins, sarcoidosis, scurvy, smoker's melanosis, smoking, squamous cell carcinoma, squamous cell papilloma, stable periodontitis, streptococcal gingivitis, symbiosis, systemic risk factors, thermal trauma, toothbrush trauma, *Treponema pallidum*, varicella zoster, vascular epulis, verruca vulgaris

“Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity”.¹ Based upon this definition from the World Health Organization (WHO), it follows that periodontal health should be defined as a state free from inflammatory periodontal disease that allows an individual to function normally and avoid consequences (mental or physical) due to current or past disease. Based upon this overall framework of health, periodontal health should be predicated upon the absence of disease, as assessed clinically, associated with gingivitis, periodontitis, or other periodontal conditions, and may include patients who have had a history of successfully treated gingivitis or periodontitis, or other periodontal conditions, who have been and are able to maintain their dentition without signs of clinical gingival inflammation. Additionally, clinical periodontal health embraces physiological immune surveillance involving levels of biological and inflammatory markers compatible with homeostasis.² Periodontitis is a chronic inflammatory disease that currently can be successfully controlled, and teeth can be retained for life. Periodontitis can remain stable (in remission) or enter periods of exacerbation. A stable periodontitis patient remains at higher risk for recurrent disease compared to a gingivitis patient or a healthy patient. Therefore, precision dental medicine requires ongoing, individual risk assessment as part of optimal patient management.

A definition of periodontal health and wellness is critical to establish ideal and acceptable therapeutic end points to periodontal therapies, to systematically assess the biological burden of periodontal inflammation, to categorize gingival and periodontal disease prevalence in populations, and to evaluate individualized risk for future disease development. Periodontal health must be assessed and defined at both the patient and site level to achieve these goals. Furthermore, definitions of periodontal health that are used to inform treatment decisions for individual patients may differ from those used in epidemiological studies.

Is there a level of gingival inflammation that is consistent with clinical periodontal health at a site level?

There is a biological level of immune surveillance, manifesting as a predominantly neutrophilic infiltrate that is consistent with clinical gingival health.²

What is the spectrum of clinical periodontal health at a site level?

What is the biology of clinical gingival health?

Clinical gingival health is generally associated with an inflammatory infiltrate and a host response consistent with homeostasis.

On a site level, how do we classify clinical gingival health?

- Clinical gingival health on an intact periodontium
- Clinical gingival health on a reduced periodontium
 - Stable periodontitis patient
 - Non-periodontitis patient (e.g. recession, crown lengthening)

What are the clinical features of gingival health on an intact periodontium?

Clinical gingival health on an intact periodontium is characterized by the absence of bleeding on probing, erythema and edema, patient symptoms, and attachment and bone loss. Physiological bone levels range from 1.0 to 3.0 mm apical to the cemento-enamel junction.

What are the clinical features of gingival health on a reduced periodontium?

Clinical gingival health on a reduced periodontium is characterized by an absence of bleeding on probing, erythema, edema and patient symptoms in the presence of reduced clinical attachment and bone levels. However, it should be recognized that successfully treated and stable periodontitis patients remain at increased risk of recurrent progression of periodontitis. In non-periodontitis patients, there is no current evidence for increased risk of periodontitis.

What are the clinical features of gingival health following treatment of gingivitis on an intact periodontium?

Clinical gingival health following treatment of gingivitis on an intact periodontium is characterized by the absence of bleeding on probing, erythema and edema, patient symptoms, and attachment and bone loss.

What are the clinical features of gingival health following successful treatment of periodontitis?

Clinical gingival health following successful treatment of periodontitis is characterized by an absence of bleeding on probing, erythema, edema, and patient symptoms in the presence of reduced clinical attachment and bone levels.

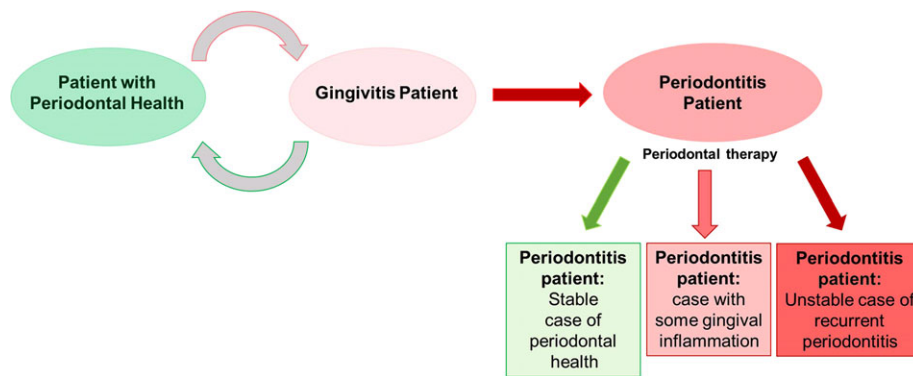


FIGURE 1 The transition from periodontal health to gingivitis is reversible following treatment that resolves gingival inflammation. The transition to periodontitis results in attachment loss which, at the present time is irreversible. More importantly, it signposts patients who are at lifelong high risk of recurrent periodontitis. Optimal periodontal therapy can restore gingival health on a reduced periodontium, or may result in mild marginal gingival inflammation at shallow probing pocket depths (≤ 3 mm). However, a history of periodontitis places patients at high risk of recurrent periodontitis and such patients require careful site-specific monitoring during periodontal maintenance programs

CASE DEFINITIONS FOR PERIODONTAL HEALTH AND GINGIVITIS

Based on available methods to assess gingival inflammation, a gingivitis case can be simply, objectively and accurately defined and graded using a bleeding on probing score (BOP%),³ assessed as the proportion of bleeding sites (dichotomous yes/no evaluation) when stimulated by a standardized (dimensions and shape) periodontal probe with a controlled (~ 0.25 N) force to the apical end of the sulcus at six sites (mesio-buccal, buccal, disto-buccal, mesio-lingual, lingual, disto-lingual) on all teeth present. Limitations of these clinical criteria arise from a lack of standardized periodontal probes (e.g. probe dimensions, taper), examiner variability (probe pressure, angle), patient related factors (biotype, medications, etc.) and smoking.

In all references to an “intact periodontium” within this consensus, an absence of detectable attachment and/or bone loss is implicit.

How do we define a case of gingival health on an intact and a reduced periodontium for epidemiological purposes?

For an intact periodontium and a reduced and stable periodontium, gingival health is defined as $< 10\%$ bleeding sites^{4,5} with probing depths ≤ 3 mm.

How do we define a case of gingival health on an intact and a reduced periodontium for clinical practice?

Due to limitations in, and a lack of uptake of, standardized ISO probes and techniques leading to inherent measurement variability in the parameters of gingival health, a patient with

periodontal health may exhibit one or two sites with some evidence of clinical gingival inflammation. Moreover, localized mild and delayed bleeding to probe at isolated sites is ubiquitous, but may fall within the spectrum of “clinical health”.

In clinical practice, a case of gingival health on an intact periodontium would be a patient with no signs of gingivitis as defined above.

In clinical practice, the goal of periodontal treatment on a reduced periodontium is a patient with no signs of gingivitis as defined above. A case of gingival health on a reduced periodontium in a stable periodontitis patient must be distinguished from a case of periodontal health in a reduced periodontium in a non-periodontitis patient (recession, crown lengthening), because there is a difference in risk for periodontal disease progression.

Following treatment of periodontitis, periodontitis patients may not attain a status of complete gingival health based on the above definition. However, evidence has demonstrated that a patient may achieve periodontal stability. Periodontal stability is characterized by successful treatment through control of local and systemic risk factors, resulting in minimal ($< 10\%$ of sites⁴) BOP, no probing depths of 4 mm or greater that bleed on probing, optimal improvement in other clinical parameters and lack of progressive periodontal destruction.⁶ The treated and stable periodontitis patient with current gingival health remains at increased risk of recurrent periodontitis and accordingly must be closely monitored. Figure 1 summarizes the various scenarios that may arise following the transition from health, to gingivitis and ultimately periodontitis.

How do we define gingivitis at a site level (biological & clinical)?

Defining inflammation at a site level is quite distinct from defining a case of gingivitis. A universal case definition is



essential to facilitate population surveillance, for clinicians setting therapeutic targets, and to enable assessment of the efficacy of prevention and/or treatment regimes.

There are broadly two categories of gingival disease:

- Dental plaque biofilm-induced gingivitis
- Non-dental plaque-induced gingival diseases

Dental plaque biofilm-induced gingivitis is defined at the site level as “*an inflammatory lesion resulting from interactions between the dental plaque biofilm and the host's immune-inflammatory response, which remains contained within the gingiva and does not extend to the periodontal attachment (cementum, periodontal ligament and alveolar bone). Such inflammation remains confined to the gingiva and does not extend beyond the mucogingival junction and is reversible by reducing levels of dental plaque at and apical to the gingival margin*”.

Depending on whether dental plaque biofilm-induced gingival inflammation occurs on an intact or reduced periodontium, or in a patient diagnosed with periodontitis, gingivitis can be further classified as:

- Gingivitis on an intact periodontium
- Gingivitis on a reduced periodontium in a non-periodontitis patient (e.g., recession, crown lengthening)
- Gingival inflammation on a reduced periodontium in a successfully treated periodontitis patient (Note that recurrent periodontitis cannot be ruled out in this case)

Since the 1999 classification, there have been advances in knowledge of the microbiome and the gingival transcriptome. Gingivitis is a non-specific inflammatory condition and is therefore a consequence of sustained plaque biofilm accumulation at and apical to the gingival margin.⁷ Longitudinal studies have demonstrated that sites that do not progress to attachment loss are characterized by less gingival inflammation over time, whereas those sites that do progress have persistently greater levels of gingival inflammation.^{8–14} Therefore, gingivitis is a major risk factor, and a necessary pre-requisite, for periodontitis. The management of gingivitis is thus a primary prevention strategy for periodontitis.

Periodontitis patients who are currently stable but develop gingival inflammation at specific sites should remain on periodontal maintenance and should be closely monitored during periodontal maintenance for any reactivation of periodontitis. Such patients may not be managed in the same way as non-periodontitis patients with gingivitis.

What are the determinants of the rate of development of gingivitis, its severity and extent?

The threshold of plaque accumulation necessary to induce gingival inflammation and impact upon its rate of progression at specific sites or at a whole mouth level varies between individuals according to both local risk factors, known as predisposing factors, and systemic risk factors, referred to as modifying factors, respectively.

1. Local risk factors (predisposing factors)

Local risk factors for gingivitis are those that encourage plaque accumulation at a specific site by either inhibiting its removal during daily oral hygiene practices, and/or creating a biological niche that encourages increased plaque accumulation.⁷ These include:

- a. Dental plaque biofilm retention factors (including certain tooth anatomical factors) – facilitate plaque accumulation at and apical to the gingival margin, enabling biofilm adherence and maturation and increasing the difficulty of mechanical plaque removal. Several clinical studies providing a moderate level of evidence have demonstrated that subgingival restoration margins may be detrimental to gingival health.^{15,16}
- b. Oral dryness is a clinical condition often associated with symptoms of xerostomia. Oral dryness manifesting as a lack of salivary flow, availability, or changes in quality of saliva, leading to reduced cleansing of tooth surfaces is associated with reduced dental plaque biofilm removal and enhanced gingival inflammation. Common causes include medications that have anti-parasympathetic action, Sjögrens syndrome when the salivary acini are replaced by fibrosis following autoimmune destruction, and mouth breathing in people who may have enhanced gingival display and/or an incompetent lip seal.¹⁷

2. Systemic risk factors (modifying factors)

Systemic risk or modifying factors are those characteristics present in an individual, which negatively influence the immune-inflammatory response to a given dental plaque biofilm burden, resulting in exaggerated or “hyper” inflammation. Examples include:

- a. Smoking – is one of the major lifestyle/behavioral risk factors for periodontitis, but which also has profound effects upon the gingival tissues. Systemic circulatory uptake of components of cigarette smoke as well as local uptake are reported to induce microvascular vasoconstriction and fibrosis. This can mask clinical signs of gingivitis, such as bleeding on probing, despite a significant underlying pathological inflammatory cell infiltrate.¹⁸

- b. Metabolic factors – hyperglycemia in people with or without diabetes. Excess glucose is toxic and directly induces mitochondrial stress and an enhanced respiratory burst in inflammatory cells that may activate various proinflammatory mediator cascades. Formation of advanced glycation end-products (AGEs) may also result in AGE binding to its cell surface receptor (RAGE), which activates proinflammatory signaling cascades and downstream proinflammatory events.¹⁹
- c. Nutritional factors – Severe Vitamin C deficiency, or scurvy, results in compromised antioxidant micronutrient defenses to oxidative stress and also negatively impacts collagen synthesis, resulting in weakened capillary blood vessel walls and a consequent propensity to enhanced gingival bleeding.²⁰
- d. Pharmacological agents (prescription, non-prescription, and recreational agents) – can act via diverse mechanisms to increase susceptibility to gingivitis. This may include drugs that reduce salivary flow, drugs that impact endocrine function (see below), and drugs that may induce gingival enlargement and pseudo-pocketing.
- e. Elevations in sex steroid hormones – at puberty, during pregnancy, or following medication with first generation oral contraceptives may modify the gingival inflammatory response. Complex biological reactions within the gingival tissues result from such elevated sex steroid levels and generate more than expected inflammation, in response to relatively small levels of plaque. However, modern oral contraceptive dosages have been reduced and there is little evidence for exaggerated gingival inflammatory responses to plaque with such drugs.²¹
- f. Hematological conditions – particular blood malignancies such as leukemia or pre-malignant conditions such as myelodysplasia are associated with signs of excess gingival inflammation in the absence of excessive plaque biofilm accumulation. Signs include swollen, purple or occasionally pale gingiva due to leukemic cell infiltration, gingival bleeding that is inconsistent with levels of dental plaque biofilm accumulation, due to thrombocytopenia and/or clotting-factor deficiencies.²²

What are the diagnostic criteria for a gingivitis case?

Given the “spectrum” of presentation of gingival health and gingival inflammation in terms of severity and extent of gingival involvement, it is important to define the features of a universally accepted case of gingivitis.

Current epidemiological data on the prevalence of gingivitis suffer from the lack of a universally adopted case definition and vary as widely as 6% to 94%, due to the use of indices that measure gingival inflammation at individual sites rather than considering the patient's mouth as a whole. Therefore, mild localized clinical inflammation is reported to affect almost 95% of the population, a figure that would incorrectly suggest gingivitis as being a variation of “normality” and thus consistent with the spectrum of “clinical health” rather than being a disease. By contrast, the more extensive the manifestation of disease employed in a case definition, the lower the reported prevalence. A universally agreed case definition should be based upon a pragmatic appraisal of the evidence base derived from longitudinal observation and intervention studies.

Clinical, radiological, and biological signs and symptoms

1. Gingivitis is a clinical diagnosis. While emerging technologies are starting to shed light on the microbiological, molecular, and pathophysiological characteristics of gingivitis, definitive knowledge is not sufficient to supersede current clinical parameters.⁷
2. The clinical signs of inflammation are erythema, edema, pain (soreness), heat, and loss of function.
3. These may manifest clinically in gingivitis as:
 - a. Swelling, seen as loss of knife-edged gingival margin and blunting of papillae
 - b. Bleeding on gentle probing
 - c. Redness
 - d. Discomfort on gentle probing
4. The symptoms a patient may report include:
 - a. Bleeding gums (metallic/altered taste)
 - b. Pain (soreness)
 - c. Halitosis
 - d. Difficulty eating
 - e. Appearance (swollen red gums)
 - f. Reduced oral health-related quality of life
5. Radiographs cannot be used to diagnose gingivitis.

Should we classify dental plaque biofilm-induced gingivitis?

There is utility in defining the severity of gingivitis as a patient communication tool, but there are no objective clinical criteria for defining severity. Thus, in this context alone, the extent of gingivitis can be used to communicate “mild, moderate, and severe” gingivitis. Moreover, emerging evidence suggests that the contained gingivitis lesion may have systemic inflammatory consequences.^{23,24}

**TABLE 1** Diagnostic look-up table for gingival health or dental plaque-induced gingivitis in clinical practice

Intact periodontium	Health	Gingivitis
Probing attachment loss	No	No
Probing pocket depths (assuming no pseudo pockets) ^a	≤3 mm	≤3 mm
Bleeding on probing ^a	<10%	Yes (≥ 10%)
Radiological bone loss	No	No
Reduced periodontium		
Non-periodontitis patient	Health	Gingivitis
Probing attachment loss	Yes	Yes
Probing pocket depths (all sites & assuming no pseudo pockets) ^a	≤3 mm	≤3 mm
Bleeding on probing ^a	<10%	Yes (≥ 10%)
Radiological bone loss	Possible	Possible
NB: In conditions where there is treatment but not cure, e.g. rheumatoid arthritis, periodontitis, the post-treatment parameters that define stability/health or gingivitis may differ from the parameters for health/gingivitis in a non-periodontitis patient. The threshold for “clinical health” in a treated and stable periodontitis patient is therefore set at ≤ 4 mm.		
Successfully treated stable periodontitis patient	Health	Gingivitis in a patient with a history of periodontitis
Probing attachment loss	Yes	Yes
Probing pocket depths (all sites & assuming no pseudo pockets) ^a	≤4 mm (no site ≥ 4 mm with BOP) ^b	≤3 mm
Bleeding on probing ^a	<10%	Yes (≥ 10%)
Radiological bone loss	Yes	Yes
NB: A successfully treated periodontitis patient in whom sites of gingival bleeding appear remains at high risk of disease recurrence at those sites and of progressive attachment loss. Therefore, gingivitis is defined as bleeding at a shallow site of ≤ 3 mm rather than ≤ 4 mm, as is the case in gingival health. Where the probing depth is 4 mm or higher with bleeding, this is no longer a “closed pocket.” ^{21,25}		

^aAssumes a light probing pressure of 0.2 to 0.25 N.

^bThere was a rational minority view expressed that the threshold for defining a clinical case of health in a successfully treated periodontitis patient should be set at ≤ 3 mm with no BOP to acknowledge the elevated risk of recurrent disease. However, the counter and majority view was that the ≤ 3 mm threshold is rarely achieved at 100% of treated sites and could lead to over-treatment, since any non-bleeding site > 3 mm would not be classified as “health” and thus open to further invasive treatment, rather than monitoring and supportive care. The threshold was therefore set at ≤ 4 mm acknowledging that post-treatment clinical phenotypes need to be considered differently to pre-treatment phenotypes.

There is no robust evidence to clearly differentiate mild, moderate, and severe gingivitis, and definitions remain a matter of professional opinion. Methods of defining gingivitis may include:

Defining percentages (e.g. mild = < 10%, moderate = 10%-30%, severe = > 30% sites)

Grading (e.g. grade 1 to 5 in 20% quintiles for % sites bleeding on probing).

How do we define a case of dental plaque-induced gingivitis on an intact and a reduced periodontium for epidemiological purposes?

For epidemiological purposes, gingivitis on an intact periodontium and gingivitis on a reduced periodontium in a patient without a history of periodontitis, is defined as ≥10% bleeding sites^{4,5} with probing depths ≤3 mm. Localized

gingivitis is defined as 10%-30% bleeding sites; generalized gingivitis is defined as > 30% bleeding sites.

For epidemiological purposes alone, a periodontitis case cannot simultaneously be defined as a gingivitis case. Therefore, a patient with a history of periodontitis, with gingival inflammation is still a periodontitis case.

How do we classify a patient with dental plaque-induced gingivitis on an intact and a reduced periodontium for clinical practice?

In clinical practice, a case of gingivitis on an intact periodontium, or a reduced periodontium in a patient without a history of periodontitis, would be a patient with signs of gingival inflammation as defined above (Table 1).

In clinical practice, periodontitis patients, if successfully treated can achieve a reduced and stable periodontium where



probing pocket depths are ≤ 4 mm²⁵ and there is an absence of clinical inflammation (bleeding on probing). Gingival inflammation may arise at specific sites, and where probing depths are ≤ 3 mm is termed gingival inflammation in a stable periodontitis patient. However, such patients remain at high risk of recurrent periodontitis and require close monitoring as such sites are at high risk of reverting to periodontitis (Table 1).

How do we classify non-dental plaque-induced gingival conditions?

Although oral health and systemic health are frequently considered as separate entities, both are strongly interrelated. There are numerous examples of how oral diseases may impact systemic health and how the oral cavity may be a window to general health. Consequently, it is crucial for all health-care providers to understand these interrelationships, inform patients of such conditions, and make appropriate referrals.

Non-dental plaque-induced gingival conditions encompass a variety of conditions that are not caused by plaque and usually do not resolve following plaque removal. Such lesions may be manifestations of a systemic condition or may be localized to the oral cavity.²⁶ Although these lesions are not caused by the dental plaque biofilm, the severity of the clinical manifestations often depends on plaque accumulation and subsequent gingival inflammation.²⁷

The proposed classification considers those conditions listed in Table 2.

Which non-dental plaque-induced gingival conditions may have associated systemic involvement and how does that impact upon patient-centered care pathways?

In recent years, the traditional treatment model in which the patient was a passive receiver of care is changing toward patient-centered care in precision dental medicine (PDM). In PDM, an individual's specific health needs and desired health outcomes are the driving force behind all health-care decisions and quality measurements. One of the elements in PDM is that care is collaborative, coordinated, and accessible. The right care is provided at the right time and the right place. Considering that the conditions marked with an "a." (Table 2) have associated systemic involvement or are oral manifestations of systemic conditions, other health-care providers may be involved in diagnosis and treatment.

TABLE 2 Classification of gingival health and gingival diseases/conditions

1. **Periodontal health**²
 - A. Clinical health on an intact periodontium
 - B. Clinical gingival health on a reduced periodontium
 - (i) Stable periodontitis patient
 - (ii) Non-periodontitis patient
2. **Gingivitis – dental plaque-induced: intact periodontium; reduced periodontium in non-periodontitis patient; reduced periodontium in successfully treated periodontitis patient.**⁷
 - A. Associated with biofilm alone
 - B. Mediated by systemic or local risk factors
 - i. Systemic risk factors (modifying factors)
 - (a) Smoking
 - (b) Hyperglycemia
 - (c) Nutritional factors
 - (d) Pharmacological agents (prescription, non-prescription and recreational)
 - (e) Sex steroid hormones
 - Puberty
 - Menstrual cycle
 - Pregnancy
 - Oral contraceptives
 - (f) Hematological conditions
 - ii. Local risk factors (predisposing factors)
 - (a) Dental plaque biofilm retention factors (e.g., prominent restoration margins)
 - (b) Oral dryness
 - C. Drug-influenced gingival enlargement
3. **Gingival diseases – non-dental plaque-induced**²⁶
 - A. Genetic/developmental disorders
 - i. Hereditary gingival fibromatosis^a
 - B. Specific infections
 - i. Bacterial origin
 - (a) *Neisseria gonorrhoeae*^a
 - (b) *Treponema pallidum*^a
 - (c) *Mycobacterium tuberculosis*^a
 - (d) Streptococcal gingivitis
 - ii. Viral origin
 - (a) Coxsackie virus (hand-foot-and-mouth disease)^a
 - (b) Herpes simplex I & II (primary or recurrent)^a
 - (c) Varicella zoster (chicken pox & shingles – V nerve)^a
 - (d) Molluscum contagiosum^a
 - (e) Human papilloma virus (squamous cell papilloma; condyloma acuminatum; verruca vulgaris; focal epithelial hyperplasia)

(Continues)



TABLE 2 (Continued)

- iii. Fungal origin
 - (a) Candidosis
 - (b) Other mycoses, e.g., histoplasmosis, aspergillosis
- C. Inflammatory and immune conditions
 - i. Hypersensitivity reactions
 - (a) Contact allergy^a
 - (b) Plasma cell gingivitis^a
 - (c) Erythema multiforme^a
 - ii. Autoimmune diseases of skin and mucous membranes
 - (a) Pemphigus vulgaris^a
 - (b) Pemphigoid^a
 - (c) Lichen planus^a
 - (d) Lupus erythematosus^a
 - Systemic lupus erythematosus
 - Discoid lupus erythematosus
 - iii. Granulomatous inflammatory lesions (orofacial granulomatoses)
 - (a) Crohn's disease^a
 - (b) Sarcoidosis^a
- D. Reactive processes
 - i. Epulides
 - (a) Fibrous epulis
 - (b) Calcifying fibroblastic granuloma
 - (c) Vascular epulis (pyogenic granuloma)
 - (d) Peripheral giant cell granuloma^a
- E. Neoplasms
 - i. Premalignancy
 - (a) Leukoplakia
 - (b) Erythroplakia
 - ii. Malignancy
 - (a) Squamous cell carcinoma^a
 - (b) Leukemic cell infiltration^a
 - (c) Lymphoma^a
 - Hodgkin
 - Non-Hodgkin
- F. Endocrine, nutritional & metabolic diseases
 - i. Vitamin deficiencies^a
 - (a) Vitamin C deficiency (scurvy)
- G. Traumatic lesions
 - i. Physical/mechanical trauma
 - (a) Frictional keratosis
 - (b) Mechanically induced gingival ulceration
 - (c) Factitious injury (self-harm)
 - ii. Chemical (toxic) burn
 - iii. Thermal insults
 - (a) Burns to gingiva

(Continues)

TABLE 2 (Continued)

- H. Gingival pigmentation
 - i. Melanoplakia^a
 - ii. Smoker's melanosis
 - iii. Drug-induced pigmentation (antimalarials, minocycline)
 - iv. Amalgam tattoo

^aConditions marked with an "a" have associated systemic involvement or are oral manifestations of systemic conditions; therefore, other health-care providers may be involved in diagnosis and treatment.

FUTURE RESEARCH NEEDS

Regarding classification and diagnosis of periodontal health and gingival diseases/conditions, future research is needed on the:

- development and validation of non-invasive diagnostic tools (e.g., saliva-based diagnostics), especially as they relate to detection of gingival inflammation;
- identification of the characteristics (e.g., genetic factors) that distinguish persons who are resistant to the development of dental plaque biofilm-induced or non-dental plaque biofilm-induced gingival diseases from those who are susceptible;
- expansion of our limited knowledge of the determinants that affect the reliability of currently available diagnostic tools (e.g., effects of probe design on bleeding on probing responses);
- characterization of the possible differences (e.g., molecular determinants) between gingivitis on an intact periodontium and other forms of gingival inflammatory disease.

Regarding the current primary periodontal diagnostic tool, the graduated periodontal measuring probe, the following are recommendations for an ISO periodontal probe:

The reliability and reproducibility of any case definition for health, gingival or periodontal conditions relies upon standardization of probing protocols, which is only possible with the implementation of an ISO probe. The current International Organization for Standardization (ISO) for periodontal probes is – ISO 21672, but requires updating in order to define the features of a global standard periodontal probe. These characteristics are:

1. Tip diameter 0.5 mm
2. Cylindrical tine structure
3. Constant force limiter of 0.25 N
4. 15-mm scale with precise individual or banded millimeter markings
5. A taper of 1.75°



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FIGURE 1 Participants of Workgroup 1