



## ORIGINAL ARTICLE

# Clinical application of the new classification of periodontal diseases: Ground rules, clarifications and “gray zones”

Kenneth S. Kornman<sup>1</sup> | Panos N. Papapanou<sup>2</sup>

<sup>1</sup>Department of Periodontics and Oral Medicine, University of Michigan School of Dentistry, Ann Arbor, MI

<sup>2</sup>Division of Periodontics, Section of Oral, Diagnostic and Rehabilitation Sciences, College of Dental Medicine, Columbia University, New York, NY

**Correspondence**

Kenneth S. Kornman, Department of Periodontics and Oral Medicine, University of Michigan School of Dentistry, Ann Arbor, MI 48109.

Email: [kkornman@umich.edu](mailto:kkornman@umich.edu)

**Abstract**

**Background:** Successful dissemination of the new classification of periodontitis is facilitated by emphasis on the basic ground rules, clarification of ambiguities, and identification of “gray zones” where thoughtful application of the guidelines by an informed, experienced clinician is paramount to arrive at a correct Stage and Grade.

**Methods:** Highlighted ground rules are (1) Stage is a patient-based, not a tooth-based concept, therefore, a single Stage is assigned per patient; (2) Stage can shift upward over time, if the periodontal status deteriorates, but the initially assigned Stage is retained even after improvement post-therapy; (3) the complexity factors that determine Stage must be evaluated collectively, not in isolation, to arrive at a clinically meaningful assessment; (4) a single Grade is assigned to a patient based on a deliberate evaluation of the “biological fabric” of the case, in terms of history of/risk for further progression, interplay of risk factors, and the two-way effects of periodontitis or its treatment on general health; (v) shift of Grade over time is possible towards either direction, after thorough, collective, evaluation of changes in the above parameters. Exemplified gray zones include a radiographically intact patient with minimal attachment loss in older age; presence of “frank” periodontitis affecting a single tooth; and assessment of factors that do/do not lead to increased complexity of therapy.

**Conclusion:** Differentiating between Stage I/II versus Stage III/IV periodontitis is relatively uncomplicated; further distinction between Stages and correct assignment of Grade requires nuanced, thorough interpretation of a broad array of findings by a knowledgeable clinician.

**KEY WORDS**

classification, diagnosis, diagnostic system, periodontal diseases, periodontitis

## 1 | INTRODUCTION

A new classification of periodontal diseases and conditions was introduced in 2018,<sup>1,2</sup> following the deliberations and the consensus reports of an International Workshop that took place in November 2017. In the time since then, educational institutions and the periodontal community have begun to utilize the new classification according to the stipulated principles, and a number of dissemination efforts to the larger

dental community, allied health professionals, patients and other constituencies are underway. As is the case with all new systems that re-classify disease modalities into novel schemes, successful implementation involves a learning curve, and additional interpretations of both the “letter” and the “spirit” of the guidelines are inevitably necessary.

To facilitate this process, the Editors of the *Journal of Periodontology* decided to initiate a series of short, authoritative commentaries to address specific items in the position paper



and the consensus report of Workgroup 2<sup>3</sup> that admittedly benefit from further clarification. In this first report, we reiterate some basic principles, emphasize important “ground rules,” identify potential gray zones, and provide practical tips that will help clinicians to seamlessly navigate the new system in their everyday clinical practice. Subsequent commentaries will further dwell on specific topics that will be exemplified by means of clinical case reports.

## 2 | PATH TO THE NEW CLASSIFICATION

One of the major learnings over the past twenty years has been the realization that multiple risk factors, including environmental exposures and genetic predispositions may combine in different patients to modify an individual’s phenotypic response to the bacterial challenge and/or their response to periodontal therapy. In addition, we know from clinical experience and research evidence that the majority of periodontitis cases respond predictably to mechanical biofilm disruption and subsequent plaque control; and that further disease progression despite standard periodontal treatment will generally occur in a small subset of patients. We also know that while average levels of attachment loss at different ages are generally consistent throughout the world, there are individuals in each age group who have experienced a level of disease severity that is disproportionate to that expressed by the majority of their peers.<sup>4</sup>

These learnings and clinical observations indicated that there was a need for additional information beyond the current level of severity to more specifically characterize a patient’s type of periodontitis. Moreover, these clinically observable exceptions in periodontitis expression and the clinical response to standard principles of therapy necessitated an evolution in the classification of periodontal diseases that we have used so far. Important questions that arose and challenged older paradigms were (1) whether the clinically observed distinct disease phenotypes are truly different diseases or, rather, variations of a common disease entity; (2) whether these phenotypes were indeed the result of different infections by specific bacteria or bacterial complexes that had been earlier implicated as causative factors; and (3) the exact role of multiple risk factors.

## 3 | KEY CONCEPTS AND GROUND RULES OF THE NEW CLASSIFICATION OF PERIODONTAL DISEASES

The new classification system is quite different from the one used for almost two decades, because, with the

exception of specific forms (necrotizing periodontal diseases and periodontitis as a manifestation of systemic disease),<sup>5,6</sup> periodontitis is recognized as a single nosological entity that is further classified using a two-vector system (*Stage* and *Grade*).<sup>1</sup> *Stage* reflects the severity of the disease (expressed through *attachment loss* and *bone loss*), but also *tooth loss* that has occurred as a result of periodontitis, at least as well as can be determined. In addition, it reflects anticipated complexity of treatment required to eradicate/reduce the current level of infection and inflammation, and to restore patient masticatory function. *Grade* describes additional biological dimensions of the disease including the observed or inferred progression rate, the risk for further deterioration due to environmental exposures (such as smoking) and co-morbidities (such as diabetes), and the risk that the disease or its treatment may adversely affect the particular patient’s general health status. Bleeding on probing (BOP) is a valuable clinical parameter to help assess current levels of inflammation and residual risk post-treatment, but BOP does not influence the classification.<sup>1</sup>

We assume that the reader of this commentary is familiar with the detailed criteria described in the tables that outline the Stage and Grade criteria of the classification, so here we will briefly go through key steps of the process to be followed when implementing the new knowledge in the everyday clinical practice.

### 3.1 | Assessment of stage

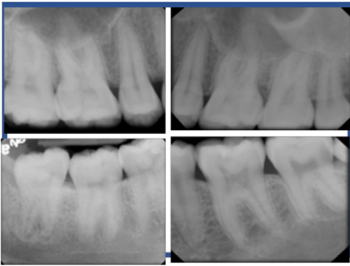
The first step is to define if the patient has periodontitis; this is ideally performed by assessing presence of clinical attachment loss but, importantly, this determination involves clinical judgement: If (1) interproximal attachment loss is present at least at two different, non-adjacent teeth, and (2) the observed attachment loss cannot be attributed to traumatic factors or non-periodontitis related etiologies (e.g., root fracture, endodontic infection, surgical trauma), then the patient has periodontitis. In the absence of interproximal attachment loss, but if attachment loss that cannot be ascribed to non-periodontitis-related causes is present at buccal or lingual surfaces, a diagnosis of periodontitis requires concomitant presence of clinical attachment loss of  $\geq 3$  mm and probing depth of  $\geq 3$  mm at  $\geq 2$  teeth. Clinicians will frequently confirm the presence of attachment loss by corresponding interproximal alveolar bone loss on radiographs. It must be remembered, however, that tissue loss needs to encompass a substantial portion of the buccal-lingual dimension before it can be visualized by conventional radiographs. Thus, absence of readily discernible bone loss does not preclude presence of frank periodontitis of incipient severity. This is exactly the reason why the diagnosis of periodontitis is based on attachment loss rather than bone loss which is admittedly more widely assessed; use of bone loss as the primary criterion

A

| Staging a Periodontitis Patient |  | Initial Periodontitis                                       | Moderate  | Severe with potential for tooth loss   | Advanced with potential for dentition loss  |
|---------------------------------|--|---|---|--|---|
| Periodontitis stage             |  | Stage I   | Stage II  | Stage III  | Stage IV  |
| Severity                        | Interdental CAL at site of greatest loss | 1 to 2 mm   | 3 to 4 mm   | ≥ 5 mm   | ≥ 5 mm  |
|                                 | Radiographic bone loss                   | Coronal third (<15%)  | Coronal third (15% to 33%)                                  | Extending to mid-third of root and beyond  | Extending to mid-third of root and beyond   |
|                                 | Tooth loss                               | No tooth loss due to periodontitis                          |   | Tooth loss due to periodontitis of ≤ 4 teeth   | Tooth loss due to periodontitis of ≥ 5 teeth  |
| Complexity                      | Local                                    | Maximum probing depth ≤ 4 mm<br>Mostly horizontal bone loss | Maximum probing depth ≤ 5 mm<br>Mostly horizontal bone loss | In addition to stage II complexity:<br>Probing depth ≥ 6 mm<br>Vertical bone loss ≥ 3 mm<br>Furcation involvement Class II or III<br>Moderate ridge defect | In addition to stage III complexity:<br>Need for complex rehabilitation due to:<br>Masticatory dysfunction<br>Secondary occlusal trauma (tooth mobility degree ≥2)<br>Severe ridge defect<br>Bite collapse, drifting, flaring<br>Less than 20 remaining teeth (10 opposing pairs) |
|                                 |  | Extent and distribution                                     | Add to stage as descriptor                                  | For each stage, describe extent as localized (<30% of teeth involved), generalized, or molar/incisor pattern   |   |

B

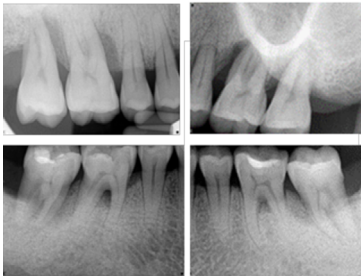
Start with a high-level assessment of the patient's periodontal status.



| Staging a Periodontitis Patient |  | Initial Periodontitis                                       | Moderate  | Severe with potential for tooth loss   | Advanced with potential for dentition loss  |
|---------------------------------|--|---|---|--|---|
| Periodontitis stage             |  | Stage I   | Stage II  | Stage III  | Stage IV  |
| Severity                        | Interdental CAL at site of greatest loss | 1 to 2 mm   | 3 to 4 mm   | ≥ 5 mm   | ≥ 5 mm  |
|                                 | Radiographic bone loss                   | Coronal third (<15%)  | Coronal third (15% to 33%)                                  | Extending to mid-third of root and beyond  | Extending to mid-third of root and beyond   |
|                                 | Tooth loss                               | No tooth loss due to periodontitis                          |   | Tooth loss due to periodontitis of ≤ 4 teeth   | Tooth loss due to periodontitis of ≥ 5 teeth  |
| Complexity                      | Local                                    | Maximum probing depth ≤ 4 mm<br>Mostly horizontal bone loss | Maximum probing depth ≤ 5 mm<br>Mostly horizontal bone loss | In addition to stage II complexity:<br>Probing depth ≥ 6 mm<br>Vertical bone loss ≥ 3 mm<br>Furcation involvement Class II or III<br>Moderate ridge defect | In addition to stage III complexity:<br>Need for complex rehabilitation due to:<br>Masticatory dysfunction<br>Secondary occlusal trauma (tooth mobility degree ≥2)<br>Severe ridge defect<br>Bite collapse, drifting, flaring<br>Less than 20 remaining teeth (10 opposing pairs) |
|                                 |  | Extent and distribution                                     | Add to stage as descriptor                                  | For each stage, describe extent as localized (<30% of teeth involved), generalized, or molar/incisor pattern   |   |

C

Start with a high-level assessment of the patient's periodontal status.



| Staging a Periodontitis Patient |  | Initial Periodontitis                                       | Moderate  | Severe with potential for tooth loss   | Advanced with potential for dentition loss  |
|---------------------------------|--|---|---|--|---|
| Periodontitis stage             |  | Stage I   | Stage II  | Stage III  | Stage IV  |
| Severity                        | Interdental CAL at site of greatest loss | 1 to 2 mm   | 3 to 4 mm   | ≥ 5 mm   | ≥ 5 mm  |
|                                 | Radiographic bone loss                   | Coronal third (<15%)  | Coronal third (15% to 33%)                                  | Extending to mid-third of root and beyond  | Extending to mid-third of root and beyond   |
|                                 | Tooth loss                               | No tooth loss due to periodontitis                          |   | Tooth loss due to periodontitis of ≤ 4 teeth   | Tooth loss due to periodontitis of ≥ 5 teeth  |
| Complexity                      | Local                                    | Maximum probing depth ≤ 4 mm<br>Mostly horizontal bone loss | Maximum probing depth ≤ 5 mm<br>Mostly horizontal bone loss | In addition to stage II complexity:<br>Probing depth ≥ 6 mm<br>Vertical bone loss ≥ 3 mm<br>Furcation involvement Class II or III<br>Moderate ridge defect | In addition to stage III complexity:<br>Need for complex rehabilitation due to:<br>Masticatory dysfunction<br>Secondary occlusal trauma (tooth mobility degree ≥2)<br>Severe ridge defect<br>Bite collapse, drifting, flaring<br>Less than 20 remaining teeth (10 opposing pairs) |
|                                 |  | Extent and distribution                                     | Add to stage as descriptor                                  | For each stage, describe extent as localized (<30% of teeth involved), generalized, or molar/incisor pattern   |   |

would result in significant under-detection of incipient periodontitis and an increase in “false negatives.”<sup>7</sup>

After ascertaining that the patient has periodontitis, the clinician should proceed with an assessment of Stage. A key element of the new classification, supported by our current knowledge, is that Stage I and Stage II adult patients are likely very different from Stage III and Stage IV patients in terms of how the host copes with and responds to the bacterial challenge. Stage I and II patients show periodontitis of incipient or moderate severity, have not lost any teeth because of the disease, and are likely to respond predictably to standard therapy based on the principles of sustainable reduction of the bacterial burden. In contrast, in Stage III and Stage IV periodontitis patients, it is most likely that one or several intrinsic or environmental risk factors adversely affect the ability of the host to respond to the bacterial infection and to contain the tissue damage; thus, these patients seem to be on a different “disease trajectory” than patients of the same age with Stage I or Stage II periodontitis. Thus, Stage III and IV represent more complex cases that require more specific knowledge, broader training and more clinical experience to manage successfully.

Based on the above, the initial staging of a case should involve a focused, high-level assessment of the patient’s medical history, radiographs, and probing chart to distinguish between Stage I or II versus Stage III or IV periodontitis, using two key discriminatory variables that can distinguish between the two aggregate groups, that is, the severity of tissue damage and the presence of periodontitis-associated tooth loss.

This high-level assessment (Figure 1A) uses a narrow set of parameters and provides a starting point for a more detailed assessment. Since the majority of adult patients in non-specialty dental offices will likely be Stage I or II, many patients can be staged by focusing on the limited parameters highlighted in Figure 1B. If the high-level assessment indicates the patient is more likely to be a Stage III or IV, the clinician will need to evaluate the more complex parameters highlighted in Figure 1C.

In this step, the clinician needs to study in detail the available full-mouth periodontal charting and full-mouth series of intra-oral radiographs. The distinction between Stage I and II periodontitis will be primarily carried out by evaluating severity of bone loss at areas of the dentition with the most advanced destruction.

A frequently raised issue is how to reliably differentiate between bone loss of up to 15% of the root length versus bone loss extending between 15% and 33% of the root length. Clearly, the point here is not to scrutinize the level of bone loss with a level of precision extending to single percentage points. Instead, the intent is to distinguish between an incipient stage of periodontitis that has barely resulted in alveolar bone loss, from more substantial bone loss that extends within the coronal third of the root length. Clearly discernible interproximal bone loss within the coronal third of the root length will, in most situations, be commensurate with Stage II rather than Stage I disease. In contrast, Stage I disease is usually characterized by incipient attachment loss in the presence of early radiographic evidence of disruption in the alveolar bone support (e.g., a break in the integrity of the *lamina dura*) rather than pronounced increase in the CEJ-bone crest distance.

If the preliminary assessment is that the patient suffers from either Stage III or Stage IV periodontitis, the distinction between these two stages will be based either on the amount of tooth loss that can be attributed to periodontitis (one to four teeth versus five or more teeth lost) or on the presence of the various complexity factors listed in Figure 1 that need to be appreciated in detail. It must be realized that either Stage III or Stage IV disease may reflect severe or very severe periodontitis. However, the primary distinction between the two requires that an experienced clinician ponders the following two central questions that essentially represent a distillation of the case’s treatment: (1) does the patient’s extent and severity of periodontitis constitute a threat for the survival of *individual teeth* or rather of the survival of *the entire dentition*? and (2) does the total therapy envisioned to address the sequelae of periodontitis in the particular patient involve extensive, multi-disciplinary oral rehabilitation? If the assessment is that the current level of periodontitis threatens the entire dentition and, consequently, treatment requires extensive oral rehabilitation involving collaboration of multiple experts (beyond the need for occasional extractions and a limited prosthetic reconstruction), then the appropriate Stage for the patient is IV rather than III. Importantly, this assessment involves a *collective* assessment of the potential complexity factors, rather than a mere “checking of a box” approach of isolated features.

It should be emphasized that Stage is a patient-based attribute, not a tooth-based assessment; consequently, a

**FIGURE 1** Figure subparts A, B, and C, staging a periodontitis patient, were reproduced with permission from the *Journal of Periodontology*, Tonetti et al.<sup>1</sup> (A) The vertical red line boundary was added to the Staging table to emphasize the distinctions between Stages I and II versus Stages III and IV and to guide the focus of a clinician’s thought process with assessment of each patient. (B) Shows selected periapical radiographs that capture one patient’s overall general radiographic bone loss, which is in the coronal third of the root length. The orange box in the figure defines characteristics of Stages I and II, which include the most likely severity of periodontitis for this patient. This initial high-level disease assessment guides clinicians to target Stages I and II based on clinical and radiographic bone loss of patients. (C) Shows selected periapical radiographs that capture one patient’s overall general radiographic bone loss, which is in the middle third or beyond of the root length. The orange box in the figure defines characteristics of Stages III and IV, which include the most likely severity of periodontitis for this patient. This initial high-level disease assessment guides clinicians to target the parameters listed for Stages III and IV based on clinical and radiographic bone loss of patients

**TABLE 1** Grading to assess patient's rate of disease progression, factors that increase risk, and potential systemic inflammatory impact

| Periodontitis grade                      |                                  | Grade A:<br>Slow rate<br>of progression           | Grade B:<br>Moderate rate<br>of progression                        | Grade C:<br>Rapid rate<br>of progression  |
|--|----------------------------------|---|--|---|
| Primary criteria                         | Direct evidence of progression   | Longitudinal data (radiographic bone loss or CAL) | Evidence of no loss over 5 years                                   | <2 mm over 5 years  |
|  | Indirect evidence of progression | % Bone loss/age<br><br>Case phenotype             | <0.25<br><br>Heavy biofilm deposits with low levels of destruction | 0.25 to 1.0<br><br>Destruction commensurate with biofilm deposits<br><br>Destruction exceeds expectation given biofilm deposits; specific clinical patterns suggestive of periods of rapid progression and/or early onset disease (e.g. molar/incisor pattern; lack of expected response to standard bacterial control therapies) |
| Grade modifiers                          | Risk factors                     | Smoking<br><br>Diabetes                           | Non-smoker<br><br>Normoglycemic/no diagnosis of diabetes           | Smoker < 10 cigarettes/day<br><br>HbA1c < 7.0% in patients with diabetes  |
| Risk of systemic impact of periodontitis | Inflammatory burden              | High-sensitivity CRP (hsCRP)                      | <1 mg/L  | 1 to 3 mg/L<br><br>> 3 mg/L   |
| Biomarkers                               | Indicators of CAL/bone loss      | Saliva, gingival crevicular fluid, serum          | ?  | ?<br><br>?  |

Table is reproduced<sup>1</sup> by permission of the *Journal of Periodontology*. Note that Grade B should be assumed for each patient unless evidence exists for a more rapid rate of progression (Grade C) or a slower rate of progression (Grade A) than expected.

single Stage is ascribed to an individual patient at a given time. Although the most severely affected segments of the dentition are the ones that inevitably define the patient's Stage, the clinician is encouraged to expand the description with additional pertinent information. The terms "localized" or "generalized" will be used to describe the extent of the dentition that is affected by the Stage-defining severity. In addition, a patient with, for example, localized Stage III periodontitis, may frequently include segments of the dentition with mild or moderate severity of attachment/bone loss; this fact should be acknowledged in the "narrative" portion of the case description.

Another frequently raised question is whether a patient's Stage can change over time. If a patient that has been staged at a given time point experiences significant disease progression or disease recurrence after therapy that results in increased severity and/or more complex treatment needs, then stage must be shifted upwards at the time of the subsequent examination, as appropriate. However, although the severity of attachment loss and/or bone loss can be reduced substantially from beyond the coronal third to within the coro-

nal third in cases of successful regeneration therapy, it is advised that the patient retains the Stage originally assigned prior to the treatment.

### 3.2 | Assessment of grade

Evidence over recent decades supported that the majority of periodontitis patients are on a trajectory that will result in predictable clinical responses if standard principles of plaque control are applied diligently to the prevention and treatment of periodontitis (Table 1). However, according to current estimates, ≈20% to 25% of our patients are on a different trajectory and, therefore, are less likely to respond predictably to standard approaches to managing periodontitis.<sup>8</sup> The primary goal of grading is to determine which of two disease paths a specific patient is traveling on, and use this information to guide the most appropriate treatment strategy that will lead to successful outcomes. Of course, there are no facts about the future, only probabilities, therefore, our classification of individual patients includes grading to help generate our best estimate at the time as to the patient's likely path.



We assume a moderate periodontitis progression rate (Grade B) until clinical or medical history provides evidence of more rapid progression or risk factors that increase the probability of more rapid progression (Grade C). In some cases, radiographic evidence suggests a slower progression rate than one might expect given the case history and patient's age (Grade A). We anticipate that planned reviews of evidence supporting modifications to staging and grading classifications will refine boundaries for defining Grades A, B, C.

## 4 | THE BASIS FOR GRADING A PATIENT

Grading is based on three fundamental principles: (1) Not all individuals are equally susceptible to periodontitis,<sup>4,9,10</sup> (2) Periodontitis progression and severity is a function of multifactorial influences on a patient's response to the microbial challenge. Multiple factors often interact to influence clinical phenotypes,<sup>8,11,12</sup> and (3) Some periodontitis cases require more intensive control of the microbial biofilm and inflammation than achieved using current principles of care.<sup>13</sup>

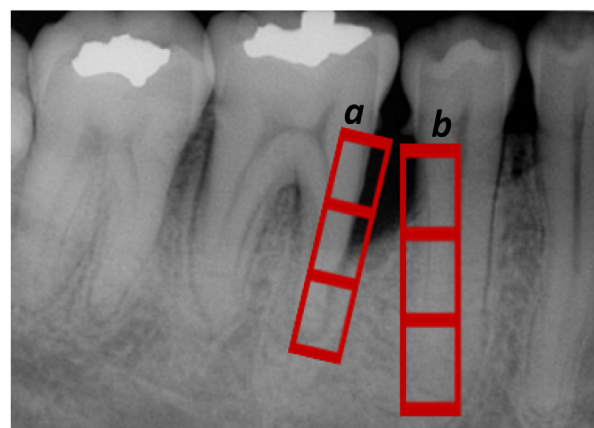
Consequently, there are three primary goals for Grading a patient with periodontitis:

1. To assist in stratifying each patient in terms of which of two general paths best capture the patient's periodontitis trajectory. A "Path 1" patient has minimal likelihood of disease progression, and clinical treatment responses are expected to be predictable after applying standard principles of periodontitis treatment based on biofilm disruption and regular plaque control; in contrast, in a "Path 2" patient, there is an increased likelihood of disease progression and less predictable clinical response to standard periodontitis prevention and treatment principles.
2. To assist new protocol development for management of periodontitis cases that are less likely to respond to current principles for periodontitis prevention and treatment.
3. To assist in development of additional approaches to management of certain periodontitis cases that may favorably influence systemic health.

Factors to be assessed to determine the patient's grade include the actual or inferred rate of periodontitis progression, presence, and control of risk factors, and status of systemic inflammation.

### 4.1 | Progression

The most reliable indication of disease progression or stability is captured by longitudinal assessments of radiographic bone loss (RBL) or CAL. For most patients progression rate must be inferred using the most severe RBL observed in relation to patient age (% bone loss/age ratio).



**FIGURE 2** Estimate radiographic bone loss at site that appears to have most severe destruction and determine whether the bone loss is likely to be in the most coronal third, the middle third, or the most apical third of the root length. Site *a* bone loss extends well into the middle third of the root length, whereas site *b* bone loss appears to be within the coronal third of the root length

**TABLE 2** Calculating the ratio of radiographic bone loss at most severe sites divided by age in years

| Bone loss as % of root length | Patient age | Ratio radiographic bone loss/age |
|-------------------------------|-------------|----------------------------------|
| 30%                           | 50          | 30/50 = 0.60                     |
| 40%                           | 50          | 40/50 = 0.80                     |
| 50%                           | 50          | 50/50 = 1.00                     |
| 60%                           | 50          | 60/50 = 1.20                     |

Bone loss assessment as a percentage of root length is inherently a rough estimate based on the clinician's interpretation of the most apical location of alveolar bone support, location of the CEJ, and location of the root apex. The example below (Figure 2) shows bone loss of  $\approx 60\%$  or greater of root length. In a 50-year-old patient, this would represent a  $>1.0$  bone loss/age ratio, as shown in Table 2. A maximum bone loss ratio by age  $>1.0$  will classify the patient as Grade C based on progression rate. Given the limited precision of assessments used to calculate the ratio of greatest RBL by age, clinicians should use clinical judgement for ratios close to 1.0.

### 4.2 | Risk factors

The multifactorial nature of periodontitis is based on evidence that multiple factors may contribute to an individual's susceptibility to develop disease and often to responsiveness to specific types of therapy. The Grading table lists the two most well-documented risk factors for periodontitis, namely smoking<sup>14,15</sup> and diabetes mellitus.<sup>16,17</sup> In general, clinicians should consider a patient's other systemic factors that may influence progression of periodontitis and treatment responses. These may include obesity, chronic



inflammatory diseases such as rheumatoid arthritis, chronic depression, genetic factors, and other factors from a comprehensive medical history.<sup>11,18–20</sup>

The goal for the clinician is to identify patients more likely to exhibit progression of periodontitis and to require intensive monitoring, intervention, and physician collaborations to help control systemic factors that may complicate host modulation of the chronic inflammatory component of severe periodontitis.

Patients classified as initial (Stage I) or moderate (Stage II) periodontitis will not routinely have sufficient periodontitis progression to qualify as a Grade C patient, unless they are very young, and therefore, may have a bone loss/age ratio of >1. However, some Stage I or II patients may be heavy smokers or have poorly controlled Type II diabetes and may therefore, qualify for a Grade C diagnosis through their risk profile. The exposures that account for Grade C should be targets for behavioral modification (i.e., smoking cessation) or additional therapeutic intervention in collaboration with the patients' physician to better achieve metabolic control in diabetes, as they entail greater risk for less predictable clinical outcomes using standard principles of disease management.

In Stage III and IV patients, assessment of Grade may often be defined indirectly by the apparent rapid bone loss relative to the patient's age; however, Grade modifiers, beyond being informative of the risk of further progression and likelihood of successful treatment outcome are obvious interventional targets.

### 4.3 | Systemic impact risk

Substantial evidence from large clinical databases involving >150,000 patients with a chronic inflammatory disease compared with >370,000 controls indicate that the presence of certain chronic inflammatory diseases influences the likelihood of a second chronic disease to be concomitantly manifested.<sup>21–23</sup> Although there is substantial evidence associating periodontitis with other diseases such as cardiovascular disease, Type II diabetes, and adverse pregnancy outcomes, evidence that treatment of periodontitis will result in predictable benefits with respect to any of those systemic conditions is rather limited.<sup>24</sup> The systemic inflammatory burden of periodontitis is well-documented, at least as measured by high sensitivity C-reactive protein (hsCRP).<sup>25,26</sup> Given the well documented role of elevated hsCRP in cardiovascular diseases,<sup>27–29</sup> as well as in other inflammatory conditions, the impact of periodontal treatment on plasma hsCRP levels may be an important parameter to monitor in certain patients with Stage III or IV periodontitis.

### 4.4 | Biomarkers

Current evidence indicates that certain combinations of salivary biomarkers may add value in the assessment of periodontal therapy relative to stability of the case post-treatment.<sup>30,31</sup>

It is expected that additional evidence of clinical utility and further advances with novel biomarkers may better inform objective assessments of Grade.

A common question is whether Grade can change over time. An upwards revision of Grade is possible if the % bone loss/age ratio increases substantially, or the risk profile of the patient deteriorates. Conversely, downgrading is also possible, if the determinants of Grade when it was originally assigned are no longer prevalent. The clinician is urged to carry out such modifications judiciously and after thorough consideration of the risk factors at play as well as of the consequences of the altered Grade on the patient's overall management plan.

## 5 | INTERPRETATIONAL CHALLENGES AND “GRAY ZONES”

In a time of evidence-based healthcare and comparative effectiveness research, some clinicians would like a simple algorithm to convert a patient's clinical findings to a stage and grade that is a robust periodontitis classification. It has become evident in medicine that in spite of extensive evidence to guide certain clinical decisions, new technologies and more evidence often expands “gray zones” which do not have simple decision guidelines.<sup>32</sup> We recognize that knowledge and clinical judgement will be required for classification of some patients. Below, we provide narrative examples of commonly encountered diagnostic “gray zones” and offer suggestions of how they can be addressed.

1. *A male 65-year-old patient has experienced no tooth loss, is radiographically intact, has no interproximal pockets with a depth >3 mm. The level of the gingival margin (GM) interproximally is, at most sites, coronal to the CEJ, except for a few surfaces located at non-adjacent teeth where the GM is located at the CEJ. A loss of attachment of 2 mm is recorded at these few surfaces. Does this patient have periodontitis?*

This is a borderline case. According to the above description, the probe tip apparently penetrates *within* the junctional epithelium to a level *apical* to the CEJ at a few interproximal sites with shallow probing depth, no visible recession and no radiographic evidence of alveolar bone loss. Since this middle-aged patient appears to be periodontally intact, a diagnosis of “periodontitis” is not justified. It must be emphasized, however, that the same phenotype in a much younger patient may signify “true” incipient periodontitis. Again, clinical judgement is paramount at arriving at a correct diagnosis after assessing the totality of the patient data.

2. *The severity of periodontitis in a 50-year old patient, based on RBL at the sites of the most advanced destruction,*

is compatible with Stage II disease (e.g., the bone loss extends within the coronal third of the root). Does presence of one or a few 6 mm pockets necessarily upshift the diagnosis to Stage III?

Not necessarily. If the severity of bone loss does not extend beyond the coronal third of the root length, presence of a couple of 6 mm pockets does not automatically entail a need for more complex treatment. Upstaging because of “complexity factors” requires a meaningful, integrated appraisal of these factors by an experienced clinician. Correct implementation of the Staging system does not lend itself to automated algorithms based on checkboxes or presence/absence of isolated features.

3. According to the new classification, a diagnosis of periodontitis requires a minimum of “at least two teeth” affected by interproximal attachment loss. Does this mean that a patient that presents with attachment loss, or bone loss, that affects only a single tooth should not be diagnosed as having periodontitis?

The requirement of “at least two affected teeth” has been incorporated in the classification to minimize false positives, that is, to preclude an inflation of periodontitis prevalence due to incidental attachment loss. This restriction was also introduced in recognition of the fact that “true” periodontitis seldom affects only a single tooth in the dentition. However, if according to the clinician’s judgment, an observed attachment loss/bone loss lesion that affects a single tooth in an otherwise intact dentition cannot be ascribed to a cause other than periodontitis (e.g., root fracture, endodontic lesion, etc.), then the clinician should bypass the rule, proceed with assigning a diagnosis of periodontitis, stage it appropriately, and further describe it as “localized.”

## 6 | THE VALUE OF THE 2018 PERIODONTITIS CLASSIFICATION


Well-controlled longitudinal clinical studies of periodontitis treatment have demonstrated that the standard principles for control of periodontitis are remarkably successful in the long-term control of the disease, but not for everyone. Over the years, classification schemes have drawn attention to different clinical phenotypes that may be expressed in some patients with periodontitis. The 2018 periodontitis classification uses the staging and grading system,<sup>1,3</sup> as discussed above, to allow clinicians to consistently (1) assess the current level of severity of periodontitis and its impact on the treatment required, and (2) determine whether a periodontitis patient is highly likely or less likely to respond predictably to standard principles for treating periodontitis. And perhaps most importantly,

the new classification guides a clinician to recognize factors that indicate that the patient’s disease trajectory is more complex and should be managed accordingly.

## ACKNOWLEDGMENTS

We would like to thank many colleagues who raised questions and offered comments that encouraged further clarifications of the new classification of periodontal diseases published in 2018. The authors have no conflicts of interest relative to the new classification or any interpretations of the classification of periodontitis that are expressed in this paper. Dr. Kornman currently serves as editor-in-chief of the *Journal of Periodontology*.

## ORCID

Kenneth S. Kornman 

<https://orcid.org/0000-0003-2252-7324>

Panos N. Papapanou 

<https://orcid.org/0000-0002-6538-3618>

## REFERENCES

1. Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: framework and proposal of a new classification and case definition. *J Periodontol*. 2018;89(Suppl 1):S159-S172.
2. Caton JG, Armitage G, Berglundh T, et al. A new classification scheme for periodontal and peri-implant diseases and conditions—introduction and key changes from the 1999 classification. *J Periodontol*. 2018;89(Suppl 1):S1-S8.
3. Papapanou PN, Sanz M, Buduneli N, et al. Periodontitis: consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Periodontol*. 2018;89(Suppl 1):S173-S182.
4. Billings M, Holtfreter B, Papapanou PN, Mitnik GL, Kocher T, Dye BA. Age-dependent distribution of periodontitis in two countries: findings from NHANES 2009 to 2014 and SHIP-TREND 2008 to 2012. *J Periodontol*. 2018;89(Suppl 1):S140-S158.
5. Albandar JM, Susin C, Hughes FJ. Manifestations of systemic diseases and conditions that affect the periodontal attachment apparatus: case definitions and diagnostic considerations. *J Periodontol*. 2018;89(Suppl 1):S183-S203.
6. Herrera D, Retamal-Valdes B, Alonso B, Feres M. Acute periodontal lesions (periodontal abscesses and necrotizing periodontal diseases) and endo-periodontal lesions. *J Periodontol*. 2018;89(Suppl 1):S85-S102.
7. Lang NP, Hill RW. Radiographs in periodontics. *J Clin Periodontol*. 1977;4:16-28.
8. Giannobile WV, Braun TM, Caplis AK, Doucette-Stamm L, Duff GW, Kornman KS. Patient stratification for preventive care in dentistry. *J Dent Res*. 2013;92:694-701.
9. Loe H, Anerud A, Boysen H, Morrison E. Natural history of periodontal disease in man. Rapid, moderate and no loss of attachment in Sri Lankan laborers 14 to 46 years of age. *J Clin Periodontol*. 1986;13:431-445.





10. Baelum V, Fejerskov O, Karring T. Oral hygiene, gingivitis and periodontal breakdown in adult Tanzanians. *J Periodontol Res.* 1986;21:221-232.
11. Morelli T, Moss KL, Beck J, et al. Derivation and validation of the periodontal and tooth profile classification system for patient stratification. *J Periodontol.* 2017;88:153-165.
12. Struch F, Dau M, Schwahn C, Biffar R, Kocher T, Meisel P. Interleukin-1 gene polymorphism, diabetes, and periodontitis: results from the Study of Health in Pomerania (SHIP). *J Periodontol.* 2008;79:501-507.
13. McGuire MK, Nunn ME. Prognosis versus actual outcome. IV. The effectiveness of clinical parameters and IL-1 genotype in accurately predicting prognoses and tooth survival. *J Periodontol.* 1999;70:49-56.
14. Johnson GK, Guthmiller JM. The impact of cigarette smoking on periodontal disease and treatment. *Periodontol 2000.* 2007;44:178-194.
15. Haber J, Wattles J, Crowley M, Mandell R, Joshipura K, Kent RL. Evidence for cigarette smoking as a major risk factor for periodontitis. *J Periodontol.* 1993;64:16-23.
16. Lalla E, Papapanou PN. Diabetes mellitus and periodontitis: a tale of two common interrelated diseases. *Nat Rev Endocrinol.* 2011;7:738-748.
17. Taylor JJ, Preshaw PM, Lalla E. A review of the evidence for pathogenic mechanisms that may link periodontitis and diabetes. *J Periodontol.* 2013;84:S113-S134.
18. Suvan J, Petrie A, Moles DR, et al. Body mass index as a predictive factor of periodontal therapy outcomes. *J Dent Res.* 2014;93:49-54.
19. Genco RJ, Ho AW, Grossi SG, Dunford RG, Tedesco LA. Relationship of stress, distress and inadequate coping behaviors to periodontal disease. *J Periodontol.* 1999;70:711-723.
20. Reynolds MA. Modifiable risk factors in periodontitis: at the intersection of aging and disease. *Periodontol 2000.* 2014;64:7-19.
21. Dregan A, Charlton J, Chowienczyk P, Gulliford MC. Chronic inflammatory disorders and risk of type 2 diabetes mellitus, coronary heart disease, and stroke: a population-based cohort study. *Circulation.* 2014;130:837-844.
22. Dregan A. Arterial stiffness association with chronic inflammatory disorders in the UK Biobank study. *Heart.* 2018;104:1257-1262.
23. Dregan A, Matcham F, Harber-Aschan L, et al. Common mental disorders within chronic inflammatory disorders: a primary care database prospective investigation. *Ann Rheum Dis.* 2019;78:688-695.
24. Beck JD, Papapanou PN, Philips KH, Offenbacher S. Periodontal medicine: 100 years of progress. *J Dent Res.* 2019;98:1053-1062.
25. Amabile N, Susini G, Pettenati-Soubayroux I, et al. Severity of periodontal disease correlates to inflammatory systemic status and independently predicts the presence and angiographic extent of stable coronary artery disease. *J Intern Med.* 2008;263:644-652.
26. Artese HP, Foz AM, Rabelo Mde S, et al. Periodontal therapy and systemic inflammation in type 2 diabetes mellitus: a meta-analysis. *PloS One.* 2015;10:e0128344.
27. Libby P, Ridker PM. Inflammation and atherosclerosis: role of C-reactive protein in risk assessment. *Am J Med.* 2004;116(Suppl 6A):9S-16S.
28. Ridker PM, Danielson E, Fonseca FA, et al. Rosuvastatin to prevent vascular events in men and women with elevated C-reactive protein. *N Eng J Med.* 2008;359:2195-2207.
29. Ridker PM, Libby P, MacFadyen JG, et al. Modulation of the interleukin-6 signalling pathway and incidence rates of atherosclerotic events and all-cause mortality: analyses from the Canakinumab Anti-Inflammatory Thrombosis Outcomes Study (CANTOS). *Eur Heart J.* 2018;39:3499-3507.
30. Kinney JS, Morelli T, Braun T, et al. Saliva/pathogen biomarker signatures and periodontal disease progression. *J Dent Res.* 2011;90:752-758.
31. Salminen A, Gursoy UK, Paju S, et al. Salivary biomarkers of bacterial burden, inflammatory response, and tissue destruction in periodontitis. *J Clin Periodontol.* 2014;41:442-450.
32. Chandra A, Khullar D, Lee TH. Addressing the challenge of gray-zone medicine. *N Engl J Med.* 2015;372:203-205.

**How to cite this article:** Kornman KS, Papapanou PN. Clinical application of the new classification of periodontal diseases: Ground rules, clarifications and “gray zones”. *J Periodontol.* 2020;91:352–360. <https://doi.org/10.1002/JPER.19-0557>