Task Force Report on the Update to the 1999 Classification of Periodontal Diseases and Conditions*

In 2014, the American Academy of Periodontology Board of Trustees charged a Task Force to develop a clinical interpretation of the 1999 Classification of Periodontal Diseases and Conditions to address concerns expressed by the education community, the American Board of Periodontology, and the practicing community that the current Classification presents challenges for the education of dental students and implementation in clinical practice.

The Academy announced that an update of the 1999 Classification would commence in 2017. This focused update addresses three specific areas of concern with the current classification: attachment level, chronic versus aggressive periodontitis, and localized versus generalized periodontitis.

Use of Attachment Levels in Diagnosis of Periodontitis

In the 1999 International Workshop for a Classification of Periodontal Diseases and Conditions, the authors of the Consensus Report on Chronic Periodontitis stated that chronic periodontitis is “An infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment, and bone loss.” It is characterized by pocket formation and/or gingival recession.” In addition, the consensus report stated that periodontitis can be further characterized by extent and severity: “As a general guide, severity can be categorized on the basis of the amount of clinical attachment loss (CAL) as follows: Slight = 1 to 2 mm CAL, Moderate = 3 to 4 mm CAL, and Severe = >5 mm CAL.” Numerous important studies since 1999 have used similar parameters to define periodontitis. For example, the recent epidemiologic studies outlining the prevalence of periodontitis in the United States used attachment loss parameters to define various severities of periodontitis. It is recognized that CAL is of importance for the scientific advancement of the knowledge of periodontitis. However, in clinical practice, measurement of CAL has proven to be challenging, and is time consuming. Measuring the location of the cemento-enamel junction (CEJ) when the gingival margin is located coronal to the CEJ is difficult and may involve some guesswork when the CEJ is not readily evident via tactile sensation. These issues can result in examinations being performed in which, rather than charting attachment levels at all sites, the clinician may chart probing depths alone or probing depths with a single recession measure at the mid-facial or mid-lingual and only when recession is actually present. Another common error occurs when gingival margin measures are charted as “0 mm” when in fact the gingival margin is not right at the level of the CEJ, resulting in attachment levels that are incorrectly charted as being equal to probing depth.

Formulation of a diagnosis of periodontitis is based on multiple clinical and radiographic parameters, all of which may not be required. In general, a patient would have periodontitis

*This paper was developed under the direction of the Task Force to Update the Classification of Periodontal Diseases and Conditions and approved by the Board of Trustees of The American Academy of Periodontology in April 2015.
when one or more sites had inflammation bleeding on probing (BOP), radiographic bone loss, and increased probing depth or clinical attachment loss. Table 1 summarizes the recommended guidelines for determining the severity of periodontitis in patients.

**Table 1. Guidelines for determining severity of periodontitis.**

<table>
<thead>
<tr>
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<th>Slight (Mild)</th>
<th>Moderate</th>
<th>Severe (Advanced)</th>
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<tbody>
<tr>
<td>Probing depths</td>
<td>&gt;3 &amp; &lt;5 mm</td>
<td>≥5 &amp; &lt;7 mm</td>
<td>≥7 mm</td>
</tr>
<tr>
<td>Bleeding on probing</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Radiographic bone loss</td>
<td>Up to 15% of root length or ≥ 2mm &amp; ≤3 mm</td>
<td>16-30% or &gt; 3mm &amp; ≤5 mm</td>
<td>&gt;30% or &gt; 5mm</td>
</tr>
<tr>
<td>Clinical attachment loss</td>
<td>1-2 mm</td>
<td>3-4 mm</td>
<td>5+ mm</td>
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Patients with gingival recession or patients following active treatment and on periodontal maintenance therapy could present with attachment loss, probing depths 3mm or less and no clinical signs of inflammation. This patient should be diagnosed with a healthy but reduced periodontium. If inflammation is noted in a patient with attachment loss, recession and probing depths 3 mm or less the diagnosis should be a reduced periodontium with inflammation (gingivitis). However when probing depths deepen in this patient to greater than 3 mm and inflammation is present the diagnosis should be periodontitis with the severity guided by the above table.

**Chronic Versus Aggressive Periodontitis**

The 1999 Armitage review summarized the rationale for the introduction of the diagnostic terms “chronic periodontitis” and “aggressive periodontitis” as the recommended nomenclature for the two principal forms of destructive periodontal disease. The review also acknowledged that both chronic and aggressive periodontitis were broad or “collective” designations, each comprising a heterogeneous “constellation” of destructive diseases. Chronic periodontitis was recommended as the descriptor to denote the slowly progressive, common form (replacing “adult periodontitis”). While Armitage and the workshop participants noted that chronic periodontitis may be characterized by limited short periods of rapid destruction in certain patients, they dismissed the notion that disease progression rates should be used to “exclude patients from receiving the diagnosis of chronic periodontitis.” Similarly, Armitage and the workshop participants discarded any age-dependent limits or criteria to differentiate between chronic or aggressive periodontitis. Two accompanying consensus reports were published as part of the workshop proceedings that
further tried to define the primary and secondary features of chronic versus aggressive periodontitis (Table 2).\textsuperscript{1,5}\n
**Table 2.** Features recognized in the 1999 consensus reports differentiating chronic versus aggressive periodontitis.

<table>
<thead>
<tr>
<th>Chronic Periodontitis\textsuperscript{1}</th>
<th>Aggressive Periodontitis\textsuperscript{5}</th>
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<tbody>
<tr>
<td>• Most prevalent in adults, but can occur in children and adolescents</td>
<td>• Except for the presence of periodontitis, patients are clinically (medically) healthy</td>
</tr>
<tr>
<td>• Amount of destruction is consistent with the presence of local factors (i.e., primary and secondary etiologic factors)</td>
<td>• Rapid attachment loss and bone destruction</td>
</tr>
<tr>
<td>• Subgingival calculus is a frequent finding</td>
<td>• Familial aggregation</td>
</tr>
<tr>
<td>• Associated with a variable microbial pattern</td>
<td>• Secondary features (generally but not universally present):</td>
</tr>
<tr>
<td>• Slow to moderate rate of progression, but may have periods of rapid destruction</td>
<td>o Amounts of microbial deposits (biofilm) are inconsistent with the severity of periodontal tissue destruction</td>
</tr>
<tr>
<td>• Can be further classified on the basis of extent and severity</td>
<td>o Elevated proportions of <em>A. actinomyctetemcomitans</em> and in some populations <em>P. gingivalis</em></td>
</tr>
<tr>
<td>• Can be associated with pre-disposing factors (e.g., tooth-related and iatrogenic factors)</td>
<td>o Phagocyte abnormalities</td>
</tr>
<tr>
<td>• May be modified by and/or associated with systemic diseases (e.g., diabetes mellitus, HIV infection)</td>
<td>o Hyper-responsive macrophage phenotype (elevated levels of PGE\textsubscript{2} and IL-1β)</td>
</tr>
<tr>
<td>• Can be modified by factors other than systemic diseases such as cigarette smoking and emotional stress</td>
<td>o Progression of attachment loss and bone loss may be self-arresting</td>
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<td></td>
<td>• Discrimination of localized versus generalized forms of aggressive periodontitis as unique “sub-classifications”</td>
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After reviewing the original 1999 workshop proceedings and updated published literature on periodontal disease diagnosis (i.e., case definitions), epidemiology, and diagnostic markers,\textsuperscript{6-13} the Task Force affirmed the use of the terms “chronic periodontitis” and “aggressive periodontitis” as separate, distinct clinical entities, both presenting with signs of periodontal destruction and inflammation.

For aggressive periodontitis, the Task Force recommended that age at onset (detection) be considered as a general guideline to distinguish patients within this broad, high-risk diagnostic classification. Following a recent review by Albandar,\textsuperscript{7} the Task Force recommended that patient age, younger than 25 years at the time of disease onset, be
used along with other signs or criteria to support a diagnosis of aggressive periodontitis. The Task Force further recognized that there are localized versus generalized forms (sub-classifications) for aggressive periodontitis. Accordingly, there are young patients (typically pubertal) whose periodontal destruction follows a characteristic pattern of affected teeth (i.e., predominantly first molars and incisors) and who may be further classified as having “localized aggressive periodontitis.” Meanwhile, the Task Force recognized that there may be other young individuals who demonstrate a history of rapid periodontal destruction (via successive charting examinations or radiographs) following no overt pattern of affected teeth and who may be diagnosed with “generalized aggressive periodontitis.” The Task Force further reiterated that the relative low levels of biofilm and secondary etiology (calculus) in affected patients may further support a broad diagnosis of aggressive periodontitis.

In contrast, the Task Force recognized that, more commonly, patients present with signs of periodontal destruction and inflammation at an older age (at time of onset) along with abundant biofilm and calculus. Within the classification of chronic periodontitis, the Task Force acknowledged that there is a spectrum of disease progression rates among such patients. Some individuals may follow a slow, continuous pattern of disease progression. Others may experience bursts of periodontal destruction around certain teeth in relatively short periods (random burst pattern). Still others may experience many bursts of destructive periodontal disease activity at a high frequency during certain periods (multiple burst pattern).

After reviewing the level of evidence on microbial and host markers, the Task Force concluded that there are no definitive biomarkers that can currently differentiate between aggressive versus chronic periodontitis or between the localized versus generalized forms of aggressive periodontitis. Hence, the clinician must base these diagnostic decisions on the patient history, clinical, and radiographic signs. Ongoing and future research may help to define prognostic subtypes or profiles within aggressive versus chronic periodontitis indicating a higher risk for rapid periodontal progression or a poorer response to therapy.

The Task Force affirmed that the diagnosis of aggressive or chronic periodontitis has important implications related to therapy, long-term prognosis, and specialty referral. Patients with aggressive (localized or generalized) periodontitis or chronic (moderate or severe) periodontitis in general require advanced periodontal therapy (i.e., beyond scaling and root planing). Such therapy may include adjunctive chemotherapeutics, regenerative or resective periodontal surgery, dental implant placement, and more frequent maintenance therapy to reduce, reverse, and/or control the disease process. The Task Force recommends that patients diagnosed with aggressive (localized or generalized) periodontitis or chronic (moderate or severe) periodontitis should be considered for referral to a periodontist for co-management, evaluation and indicated therapy.
Localized Versus Generalized Periodontitis

In the 1999 classification, the guidelines for differentiating localized versus generalized disease are based on the percentage of affected sites. As a general guide, extent can be characterized as localized ≤30% of sites involved and generalized >30% of sites involved.¹

However, confusion remains among clinicians about the distinction between localized and generalized disease that is difficult to resolve by simply calculating the percentage of sites involved.

Patterns of tooth involvement are well defined for aggressive periodontitis. Localized aggressive periodontitis is often characterized by a first molar/incisor pattern that is well described in the literature¹⁵,¹⁶ and was formerly diagnosed as localized juvenile periodontitis¹⁷ but in less distinct cases may be described by the specific localization of the disease. Generalized aggressive periodontitis is commonly characterized by involvement of almost all teeth. The terms “localized” and “generalized” for aggressive periodontitis, therefore, have recognizable patterns that are well understood by most clinicians. The extent of aggressive periodontitis is not determined by the percentage of affected sites.

In chronic periodontitis, there is more heterogeneity in the presentation of the disease. Therefore, to describe the pattern of disease in chronic periodontitis, it may be more beneficial to use descriptive terms to clarify the distribution of the affected teeth, e.g., “chronic periodontitis localized to maxillary molars with severe lesions on the premolars.” The Task Force preferred to use the percentage of affected teeth rather than the percentage of affected sites as an extent descriptor for chronic periodontitis. For localized chronic periodontitis, the criteria to be met is either a clear pattern of the affected teeth (e.g., distal of both maxillary second molars) or ≤30% of teeth.¹⁸ Generalized chronic periodontitis may be defined as periodontitis without a clear pattern of disease distribution of affected teeth or >30% of teeth are affected.

Concluding Remarks

As mentioned in the introductory paragraphs, this is a focused update with a comprehensive update planned for 2017. At this future workshop, a classification for peri-implant diseases should be developed. Additionally, risk assessment for periodontal disease progression and prognostic subtypes or profiles within aggressive versus chronic periodontitis should also be included.

References

2. Eke PI, Dye BA, Wei L, Thornton-Evans GO, Genco RJ; CDC Periodontal Disease Surveillance workgroup: James Beck (University of North Carolina, Chapel Hill, USA), Gordon Douglass (Past President, American Academy of Periodontology), Roy Page


