

Episode #49 – Classification of Periodontal & Peri-Implant Diseases & Evidence-Based Treatment Guideline for Periodontitis

November 26, 2021

Introduction

In 2018, the American Academy of Periodontology (AAP) published a new global classification system for periodontal health, diseases and conditions, as well as peri-implant diseases and conditions. The classification system was the outcome of a joint workshop held by the European Federation of Periodontology (EFP) and AAP in 2017. The updated system comes almost two decades after the last comprehensive classification of these conditions and reflects the substantial amount of new evidence and knowledge gained in that time. [1]

In 2020, EFP published the first formal evidence-based guideline for treating periodontitis to assist oral health practitioners globally in providing the best possible treatment for their clients. The guideline was formulated at the EFP-led Perio Workshop 2019 (XVI European Workshop on Periodontology), it was attended by 90 experts from 19 countries who evaluated 15 systematic reviews on various types of periodontal therapy. [2]

The EFP S3 level clinical practice guideline was published in the *Journal of Clinical Periodontology* (July 2020), along with the systematic reviews and their respective meta-analyses that were examined to formulate the guideline. [2]

The guideline was developed using the highest level of methodology (S3) and is based on the client's diagnosis according to the AAP classification of periodontal diseases. The guideline provides oral healthcare professionals with 62 clinical recommendations for periodontal therapy for the treatment of stage I-III periodontitis utilizing 4 sequential steps:

1. Reducing inflammation by motivating clients to adopt good oral hygiene practices and a healthy lifestyle, and through professional removal of supragingival deposits.
2. Providing professional subgingival debridement with adjunctive therapies as required.
3. Administering additional complex treatments, such as surgery, when indicated.
4. Preventing relapse through long-term supportive care, healthy lifestyle, good oral hygiene, and regular assessments. [2] [3]

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The guideline, however, does not address the treatment of gingivitis (although management of gingivitis is an indirect goal in some of the interventions), stage IV periodontitis, necrotizing periodontitis, periodontitis as manifestation of systemic diseases, or mucogingival conditions. [3]

Recommendations for periodontitis stage IV therapy took place at Perio Workshop 2021 (XVII European Workshop on Periodontology) in July 2021. A collection of systematic reviews has been published to support the eventual publication of an evidence-based guideline for the treatment of stage IV periodontitis. The collection of studies is in progress and the EFP webpage will be updated accordingly. [3] [4]

Considering the EFP's guideline is based on client diagnosis of stage I-III periodontitis according to the AAP classification of periodontal diseases, an overview of the AAP classification system is warranted.

AAP classification of periodontal and peri-implant diseases and conditions
AAP classification of periodontal and peri-implant diseases and conditions consists of 4 main categories:
1. periodontal health, gingival diseases and conditions;
2. periodontitis;
3. other conditions affecting the periodontium; and
4. peri-implant diseases and conditions.

Each category contains several sub-categories.

1. Periodontal health, gingival diseases and conditions

a. Periodontal health and gingival health

i. *Pristine periodontal health* – Total absence of clinical inflammation (no bleeding on probing [BOP]) along with no attachment or bone loss. Pristine periodontal health is unlikely to be observed clinically in adults. Modifying factors (conditions that alter an individual's responds to subgingival plaque accumulation, such as smoking, systemic conditions [e.g., diabetes, genetics, immune function], medications, stress, nutrition) and predisposing factors (conditions that contribute to dental plaque accumulation, such as tooth anatomy, tooth position, restorations) are controlled.

ii. *Clinical periodontal health* – Absence or minimal levels of clinical inflammation with normal support (e.g., no/minimal BOP [i.e., <10% of sites], normal sulcus depth and bone height). Modifying and predisposing factors are controlled.

iii. *Periodontal disease stability in a reduced periodontium* – Periodontitis was successfully treated and clinical signs of the disease do not appear to worsen in extent or severity despite the presence of a reduced periodontium. Modifying and predisposing factors are controlled.

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iv. *Periodontal disease remission/control in a reduced periodontium* – A period in the course of the disease when symptoms become less severe (e.g., BOP is significantly reduced) but may not be fully resolved. Modifying and/or predisposing factors are not fully controlled. [1] [5]

b. Gingivitis: dental biofilm-induced – This is described as inflamed gingival tissues (e.g., BOP, erythema, edema, tenderness, hypertrophy) resulting from bacterial plaque accumulation at and below the gingival margin. There is stable (i.e., non-changing) periodontal attachment levels, which may or may not involve a loss of attachment or alveolar bone. For example, plaque-induced gingivitis on a reduced periodontium may occur after periodontal treatment has resolved the inflammation from periodontitis, but biofilm-induced gingival inflammation recurs with no evidence of progressive attachment loss (i.e., no indication of active disease). The extent and severity of inflammation are influenced by various systemic or local factors. Localized gingivitis is defined as 10-30% bleeding sites, while generalized gingivitis is defined as >30% bleeding sites. [6]

   The severity (intensity) of gingival inflammation can be categorized as mild, moderate, or severe:
   Mild gingival inflammation: area with a minor change in colour and little change in texture of the tissue.
   Moderate gingival inflammation: area with glazing, redness, edema, hypertrophy, and BOP.
   Severe gingival inflammation: area of overt redness and edema with a tendency toward bleeding when touched or probed.
   Inflammation is reversible by biofilm removal or disruption. [6]

   i. *Associated with dental biofilm only* - Inflammatory response of the gingival tissues resulting from bacterial plaque accumulation.

   ii. *Mediated by systemic or local risk factors* – Systemic modifying factors (e.g., hormones [puberty, menstrual cycle, pregnancy, oral contraceptives], systemic disorders [e.g., hyperglycemia, leukemia, malnutrition]) and local modifying factors (e.g., prominent subgingival restoration margins, smoking, hyposalivation) can alter the severity of the plaque-induced inflammation.

   iii. *Drug-induced gingival enlargement* – Several medications may cause gingival tissue enlargement (hypertrophy), such as antiepileptic drugs (e.g., phenytoin, sodium valproate), calcium channel-blocking drugs (e.g., nifedipine, verapamil, diltiazem, amlodipine, felodipine), immunoregulating drugs (e.g., cyclosporine), and high-dose oral contraceptives. For drug-influenced gingival conditions, plaque bacteria and the drug are required to produce a gingival response. However, not everyone taking these drugs develop gingival

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enlargement and some individuals with drug-influenced gingival enlargement exhibit little to no gingivitis. [1] [6]

c. Gingival diseases: non-biofilm induced – Non-plaque induced gingival diseases are less common as compared to plaque-induced gingivitis and are often manifestations of systemic conditions. They may also represent pathologic changes limited to gingival tissues. These diseases are not directly caused by plaque, but their clinical course may be affected by plaque accumulation and subsequent gingival inflammation.

   i. *Genetic/developmental disorders* – Hereditary gingival fibromatosis[1] is a rare disorder that may present with gingival overgrowth.

   ii. *Specific infections* – Other pathogens may infect the gingiva, such as certain bacteria (e.g., *Neisseria gonorrhoeae*,[2] *Treponema pallidum*[3]); viruses (e.g., coxsackieviruses,[4] herpes simplex virus types 1 [HSV-1] and 2 [HSV-2], varicella-zoster virus,[5] molluscum contagiosum virus,[6] human papilloma virus); and fungi (e.g., candidosis, histoplasmosis,[7] aspergillosis[8]).

   iii. *Inflammatory and immune conditions* – Several conditions can impact the gingiva. These include hypersensitivity reactions (e.g., contact

[1] Hereditary gingival fibromatosis is a benign, non-hemorrhagic, fibrous gingival overgrowth that can cover all or part of the teeth and in many cases prevents eruption. The disorder usually arises during the transition from primary to permanent teeth. [19]
[2] *Neisseria gonorrhoeae* is a bacterial pathogen responsible for gonorrhea. It can manifest as fiery red, edematous, and painful ulcerations, asymptomatic chancres, mucous patches or atypical non-ulcerated, highly inflamed gingiva. [20]
[3] *Treponema pallidum* is a spirochaete bacterium that causes syphilis. It can manifest as fiery red, edematous, and painful ulcerations, asymptomatic chancres, mucous patches or atypical non-ulcerated, highly inflamed gingiva. [20]
[4] Coxsackieviruses are divided into group A and group B viruses. Group A tend to infect the skin and mucous membranes, causing diseases such as hand-foot-and-mouth (HFM) disease. HFM symptoms include a blister-like rash involving the hands, feet, and mouth (tongue, gingiva, buccal mucosa). [21]
[5] Varicella-zoster virus causes chickenpox, primarily in children, and later reactivation of the virus in adults causes herpes zoster (shingles). Both can involve the gingiva, presenting as vesicle lesions that burst leaving fibrin-covered lesions. [20]
[6] Molluscum contagiosum is a self-limited viral infection that commonly affects the skin. Involvement of mucous membranes is rare. Clinically, the infection manifests as single or multiple asymptomatic, small (2-6 mm), flesh-coloured or pink papules with a central depression. [22]
[7] Histoplasmosis is caused by the fungus *Histoplasma capsulatum*, mainly found in soil containing large amounts of bird, bat, or cat feces. Oral lesions have been reported involving the gingiva, tongue, palate, buccal mucosa, oropharynx, retromolar area, lip, and floor of mouth. The lesions can be flat, plaque-like, non-tender elevations, either papillary or nodular, which ulcerate and become painful. Other descriptions include an erythematous, granulomatous appearance, a pattern of scattered red and white granulomatous lesions, and ulcerations as large as 2.5 cm in diameter. [20]
[8] Aspergillosis is an opportunistic infection usually affecting the lower respiratory tract and is caused by inhaling spores of the filamentous fungus *Aspergillus*. Aspergillosis of the oral cavity is an uncommon condition occurring most frequently in immunocompromised individuals. In most cases, oral aspergillosis lesions are yellow or black, with a necrotic ulcerated base. [23] [24]

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allergy, plasma cell gingivitis,[9] erythema multiforme[10]); autoimmune diseases of skin and mucous membranes (e.g., pemphigus vulgaris,[11] pemphigoid, lichen planus, lupus erythematosus); and granulomatous inflammatory conditions (e.g., Crohn's disease,[12] sarcoidosis[13]).

iv. *Reactive processes* – Reactive processes include epulides,[14] such as fibrous epulis,[15] calcifying fibroblastic granuloma,[16] pyogenic granuloma (vascular epulis), peripheral giant cell granuloma[17] (or central).

v. *Neoplasms* – Conditions affecting the gingiva include premalignant neoplasms (e.g., leukoplakia, erythroplakia) and malignant neoplasms (e.g., squamous cell carcinoma, leukemia, lymphoma).

vi. *Endocrine, nutritional & metabolic diseases* – This includes vitamin deficiencies, such as vitamin C deficiency (scurvy).

vii. *Traumatic lesions* – Traumatic lesions of the gingiva can result from a variety of causes and can be self-inflicted, iatrogenic, or accidental in nature. Causes of traumatic lesions include physical/mechanical insults (e.g., frictional keratosis, toothbrushing-induced gingival ulceration, factitious injury [self-harm]); chemical (toxic) insults (e.g., etching, chlorhexidine, acetylsalicylic acid, cocaine, hydrogen peroxide, dentifrice detergents, paraformaldehyde, calcium hydroxide); and thermal insults (e.g., mucosa burns).

[9] Plasma cell gingivitis is a rare condition characterized by diffuse and massive infiltration of plasma cells into the sub-epithelial connective tissue. It presents as diffuse reddening and edematous swelling of the gingiva with a sharp demarcation along the mucogingival border. [25]
[10] Oral lesions from erythema multiforme may occur in the labial and buccal mucosa, tongue, palate, and gingiva. Mucosal lesions consist of swelling and redness with blister formation. Blisters quickly rupture leaving large, shallow, irregular shaped, painful ulcers covered by a white pseudomembrane. [20] [26]
[11] Refer to Episode #30 for additional information on pemphigus.
[12] Crohn's disease is an inflammatory disease of the intestines that can involve any part of the gastrointestinal tract. It can cause small, shallow, round- to oval-shaped, painful ulcers in the oral mucosa. Oral lesions are sometimes the first sign of the disease. [20] [27]
[13] Sarcoidosis is a relatively rare acquired systemic granulomatous disease affecting multiple organs and tissues. Gingival involvement presents as red gingival enlargement. [28]
[14] Epulides are lesions associated with the gingival tissues (epulides mean "on the gums").
[15] Fibrous epulis is a hyperplastic fibrous tissue mass located at the gingiva. It is usually the same colour as the oral mucosa and firm on palpation, yet sometimes hard if bone is present within the lesion. [29]
[16] Calcifying fibroblastic granuloma is a relatively rare gingival overgrowth possibly triggered by dental calculus or plaque, microorganisms, dental appliances, and restorations. It occurs exclusively on the gingiva as a pedunculated or sessile nodular mass, usually emanating from the interdental papilla. It ranges from red to pink in colour and is frequently ulcerated. [30]
[17] Peripheral giant cell granuloma has an unknown etiology, although local irritation due to dental plaque or calculus, periodontal disease, poor dental restorations, ill-fitting dental appliances, or dental extractions may contribute to lesion development. Most lesions present as a dusky purple, sessile or pedunculated, smooth-surfaced, dome-shaped papule or nodule <1.5 cm in diameter. [31]

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viii. *Gingival pigmentation*[18] – Types of gingiva pigmentation or melanoplakia include smoker's melanosis, drug-induced pigmentation (e.g., antimalarials, minocycline), and amalgam tattoo. [1] [7]

2. Periodontitis

a. Periodontitis – A chronic multifactorial inflammatory disease associated with bacterial dysbiosis and characterized by progressive destruction of tooth supporting structures. Its primary features include clinical attachment loss (CAL),[19] radiographic bone loss, periodontal pocketing, and gingival bleeding. [1] [8]

b. Necrotizing periodontitis – An inflammatory process characterized by interdental papillae necrosis, gingival bleeding, halitosis, pain, and rapid bone loss. Necrotizing periodontal diseases are strongly associated with compromised host immune response. Predisposing conditions include HIV/AIDS, immunosuppression, severe malnutrition, acute psychological stress, insufficient sleep, smoking, and inadequate oral hygiene. [1] [8] [9]

c. Periodontitis as a manifestation of systemic disease – Systemic disorders can impact periodontal inflammation via an immunological response (e.g., Chédiak-Higashi syndrome,[20] leukocyte adhesion deficiency syndromes,[21] Down syndrome), by affecting the oral mucosa and gingival tissues (e.g.., epidermolysis bullosa[22]) or connective tissues (i.e., systemic lupus erythematosus, Ehlers-Danlos syndromes[23]), or through metabolic or endocrine disorders (e.g., glycogen storage diseases,[24] obesity, osteoporosis, diabetes mellitus, hypophosphatasia[25]). Similarly, systemic inflammatory diseases such as rheumatoid arthritis or inflammatory bowel disease can

[18] Refer to Episode #8 for additional information on gingival pigmentation related to classification.
[19] Refer to Episode #20 for more information on CAL.
[20] Chédiak-Higashi syndrome (CHS), also called Begnez-Cesar's Syndrome and several other names, is characterized by reduced pigment in the skin and eyes, immune deficiency with an increased susceptibility to infections, and a tendency to bruise and bleed easily. It commonly causes mouth sores, gingivitis, and periodontitis. [32]
[21] Leukocyte adhesion deficiency (LAD) syndromes are a group of rare disorders affecting the immune system. They are characterized by recurrent, life-threatening infections and poor wound healing. Individuals with this condition are predisposed to periodontitis. [33]
[22] Epidermolysis bullosa is a group of rare diseases that cause fragile, blistering skin. Blisters appear in response to minor trauma (e.g., heat, rubbing, scratching). In severe cases, blisters may occur in the mucous membrane of the digestive tract. Ulcerations can affect all oral mucosa including the tongue. Lesions heal with scarring. The continual process of blister formation and healing with scarring results in marked changes in the oral architecture. [34] [35]
[23] Ehlers-Danlos syndromes (EDS) are a group of related disorders caused by different genetic defects in collagen. They are generally characterized by joint hypermobility, instability, and dislocations; scoliosis and other joint deformities, skin hyperextensibility, and abnormal scarring. Some forms impact the periodontium resulting in periodontitis. [36] [37] [38]
[24] Glycogen storage diseases are a group of disorders in which stored glycogen cannot be metabolized (due to enzyme deficiency) into glucose to supply energy and maintain steady blood glucose levels. Certain forms increase susceptible to oral ulcers, periodontitis, and oral infections. [39] [40]
[25] Hypophosphatasia (HPP) is a rare genetic disorder characterized by impaired mineralization of bones and teeth, increasing caries risk, early loss of primary teeth, and loss of permanent teeth. [41]

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impact periodontal health and influence the pathogenesis of periodontitis. [1] [10]

3. Other conditions affecting the periodontium

a. Systemic diseases or conditions affecting the periodontal supporting tissues – Many diseases and conditions can affect periodontal tissues either by influencing the course of periodontitis or affecting the periodontium independent of biofilm-induced inflammation. Diseases and conditions affecting the course of periodontitis can be rare (e.g., Papillon-Lefèvre syndrome,[26] leucocyte adhesion deficiency syndromes, and hypophosphatasia) or common (e.g., diabetes mellitus, smoking). Conditions affecting the periodontium independently of dental plaque biofilm-induced inflammation include neoplastic diseases (e.g., oral squamous cell carcinoma, secondary metastatic neoplasms) and other conditions (e.g., Langerhans cell histiocytosis[27]). [1] [10] [11]

b. Periodontal abscesses and endodontic-periodontal lesions – A periodontal abscess may develop in a pre-existing periodontal pocket (e.g., in clients with untreated periodontitis, under supportive therapy, or after scaling and root planing or systemic antimicrobial therapy). A periodontal abscess developing in a previously periodontally healthy site is often associated with subgingival foreign body impaction (e.g., toothpicks, popcorn hulls) or harmful habits (e.g., biting wire, nail biting, clenching) causing foreign body impaction. An endodontic-periodontic lesion may be caused by a carious or traumatic lesion that affects the pulp and, subsequently, affects the periodontium; by periodontal destruction that subsequently affects the root canal; or by both events concurrently. [8] [9]

c. Mucogingival deformities and conditions – Periodontal health can be maintained in most clients with optimal oral home care even with minimal amounts of keratinized tissue. There are several mucogingival characteristics and other predisposing conditions or treatments that may be associated with the occurrence of gingival recession. For example, thin periodontal phenotype[28] is at greater risk for developing gingival recession. Also,

[26] Papillon-Lefèvre syndrome is an extremely rare genetic disorder that typically becomes apparent from approximately 1 to 5 years of age. It is associated with severe inflammation and degeneration of the periodontium. Primary teeth frequently become loose and exfoliate by about age five. Without treatment, most of the permanent teeth may also be lost by around age 17. [42]

[27] Langerhans cell histiocytosis (LCH) is an inflammatory myeloid neoplasia caused by mutations of several genes. It can damage tissue or cause lesions to form in single or multiple locations (e.g., bone, oral cavity, skin, lung, etc.). Signs and symptoms are dependent on tissues involved. Jaw involvement in children may result in early tooth eruption and gingival swelling and bleeding. Adults are more likely to have lesions in the maxilla and maxilla resulting in tooth loss. Individuals with skin involvement may experience ulcerative lesions on the tongue, gingiva, and buccal mucosa. [43] [44]

[28] In the AAP consensus report, the term periodontal biotype was replaced by periodontal phenotype. Periodontal phenotype is determined by gingival phenotype (gingival thickness, keratinized tissue width), and bone morphotype (thickness of the buccal bone plate). [11]

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inadequate oral hygiene, orthodontic treatment, and cervical restorations may increase risk of gingival recession. [1] [11] [12]

d. Traumatic occlusal forces – There is no evidence traumatic occlusal forces lead to periodontal attachment loss, non-carious cervical lesions (abfractions), or gingival recession. Traumatic occlusal forces can lead to adaptive mobility in teeth with normal support, whereas they can lead to progressive mobility in teeth with reduced support, usually requiring splinting. Reducing tooth mobility may enhance the effect of periodontal therapy. [1] [11] [13]

e. Tooth and prosthesis-related factors – Tooth anatomic factors (e.g., enamel pearls, developmental grooves, root proximity, fractures) and tooth relationships in the dental arch and with the opposing teeth can increase plaque retention. Restoration margins located within the gingival sulcus do not cause gingival inflammation if clients maintain good homecare and receive periodic maintenance. Tooth-supported and/or tooth-retained restorations and their design, fabrication, delivery, and materials have been associated with plaque retention and attachment loss. Hypersensitivity reactions can occur to dental materials which can appear as localized inflammation that does not respond to plaque control. Restoration margins placed within the junctional epithelium and supracrestal connective tissue attachment[29] can be associated with inflammation and, potentially, recession. [1] [11] [14]

Periodontitis

Periodontitis is defined when loss of periodontal tissue support through inflammation is the primary feature. It is important to note when an individual is diagnosed with periodontitis, they remain at risk of disease progression for life, even following successful therapy, and require life-long supportive care to prevent disease recurrence. [1] [15]

A client is diagnosed with periodontitis when:

- Interdental CAL is detected at 2 or more non-adjacent teeth, or
- Buccal CAL of ≥3mm with pocketing of >3mm is detected at 2 or more teeth, and
- CAL is not a result of non-periodontal factors, such as gingival recession caused by trauma; cervical caries; CAL on the distal surface of a second molar associated with malposition or third molar extraction; vertical root fracture; or drainage of an endodontic lesion through the gingival margin.

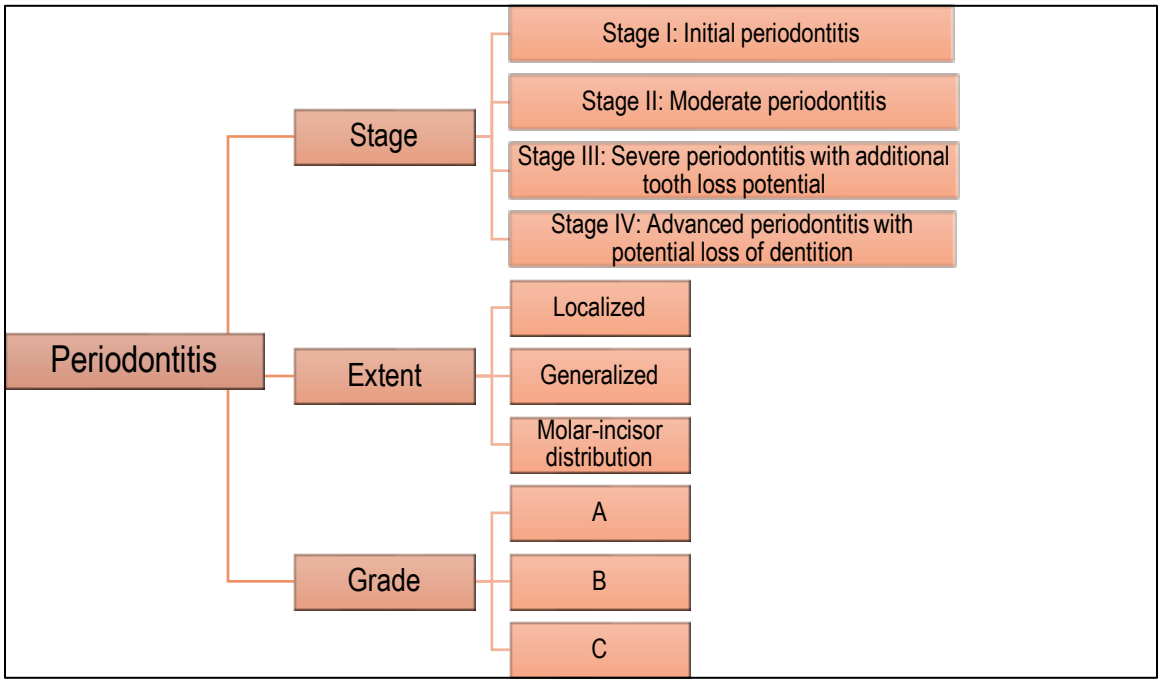
Periodontitis is further characterized by staging the disease (i.e., severity and complexity of management) and grading the disease (i.e., evidence or risk of progression and potential risk for systemic impact, which also influences the complexity of managing periodontitis).[30] There are four stages (I-IV) and three grades (A-C). [16]

[29] The term biologic width is replaced by supracrestal tissue attachment consisting of junctional epithelium and supracrestal connective tissue. [11]

[30] Refer to Episode #8 for additional information on staging and grading.

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Periodontitis classification



Staging

Staging involves assessing disease severity and extent and the complexity to control the disease and manage long-term function and esthetics of the dentition. The severity is primarily based on interdental attachment loss attributable to periodontitis and is assessed by measuring CAL and radiographic bone loss. It is assigned based on the worst-affected tooth. These measurements include the number of teeth lost due to periodontitis. Complexity considers factors such as the presence of deep probing depths, vertical defects, furcation involvement, tooth hypermobility, drifting and/or flaring of teeth, tooth loss, ridge deficiency, and loss of masticatory function. [16]

Stage I periodontitis

Stage I periodontitis represents the early stages of attachment loss. Clients with clinical attachment loss at a relatively early age may have increased susceptibility to disease progression. Early diagnosis offers opportunities for early intervention and monitoring.

Stage II periodontitis

Stage II represents established periodontitis. At this stage of the disease process management remains relatively simple for many cases.

Stage III periodontitis

At stage III, periodontitis has produced significant damage to the periodontium and, without advanced treatment, tooth loss may occur.

Stage IV periodontitis

At the more advanced stage IV, periodontitis causes considerable damage to the periodontium and may cause significant tooth loss, which translates to loss of

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masticatory function. In the absence of proper control of the periodontitis and adequate rehabilitation, the dentition is at risk of being lost. [16]

Framework for staging [16]

Periodontitis stage		**Stage I** Initial periodontitis	**Stage II** Moderate periodontitis	**Stage III** Severe periodontitis with potential for additional tooth loss	**Stage IV** Advanced periodontitis with extensive tooth loss and the potential for complete loss of the dentition
Severity	Interdental CAL at site of greatest loss	1-2 mm	3-4 mm	≥5 mm	
	Radiographic bone loss	Coronal third (<15%)	Coronal third (15-33%)	Extending to middle third of root or beyond	
	Periodontitis-associated tooth loss	No tooth loss from periodontitis		≤4 teeth lost from periodontitis	≥5 teeth lost from periodontitis
Complexity	Local	Maximum probing depth ≤4 mm Mostly horizontal bone loss	Maximum probing depth ≤5 mm Mostly horizontal bone loss	In addition to stage II complexity: ▪ Probing depth ≥6 mm ▪ Vertical bone loss ≥3mm ▪ Furcation involvement (class II or III) ▪ Moderate ridge defect	In addition to stage III complexity: ▪ Need for complex rehabilitation due to: ▪ Masticatory dysfunction ▪ Secondary occlusal trauma, bite collapse, drifting, flaring ▪ Severe ridge defect ▪ <20 remaining teeth (10 opposing pairs)
Extent & distribution	Add to stage as descriptor	For each stage, describe extent as: ▪ Localized (<30% of teeth involved) ▪ Generalized, or ▪ Molar-incisor pattern			

Note:

- The initial stage should be determined using CAL. If CAL is not available, radiographic bone loss (RBL) should be used.
- Tooth loss from periodontitis will affect the stage, even without complexity factors.
- Complexity factors may move the stage to a higher level (e.g., furcation II or III would move the stage to either stage III or IV regardless of the CAL).
- Difference between stages III and IV is mainly based on complexity factors (e.g., considerable tooth mobility and/or posterior bite collapse would denote stage IV).
- Generally, it only takes 1 complexity factor to move to a higher stage.
- CAL and RBL are still the main stage determinants for post-treatment clients. If a complexity factor(s) was eliminated by treatment, the stage should not revert to a

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lower stage since the original stage complexity factor should be considered in the maintenance phase. [16]

Staging [16]

	Stage	CAL	Probing depth	Tooth loss from periodontitis	Bone loss trend	Furcation	RBL
Initial periodontitis	I	1-2 mm	≤4 mm	None	Horizontal	Class I	<15%
Moderate periodontitis	II	3-4 mm	≤5 mm	None	Horizonal	Class I	15-33%
Severe periodontitis	III	≥5 mm	≥6 mm	≤4 teeth	Vertical	Class II to III	Extending to middle 3rd of root or beyond
Advanced periodontitis	IV	≥5 mm	≥6 mm	≥5 teeth	Vertical	Class II to III	Extending to middle 3rd of root or beyond

Grading

Regardless of the stage at diagnosis and depending on the individual, periodontitis may progress at different rates, may respond less predictably to treatment, and may or may not influence general health or systemic disease. [16]

Thus, grading helps to provide additional information on the disease's biological features. These features include analyzing the rate of attachment loss overtime, assessing future progression risk, analyzing possible poor treatment outcomes, and assessing the risk the disease or its treatment may have on the client's general health. Risk factors, such as cigarette smoking and diabetes, affect the rate of progression of periodontitis and may increase the advancement from one stage to the next.

<u>Grade A</u>

- Slow rate of progression
- No radiographic bone loss/CAL over 5 years
- <0.25 bone loss/age
- Heavy biofilm deposits with low levels of destruction
- Non-smoker
- Normoglycemia, no diagnosis of diabetes

<u>Grade B</u>

- Moderate rate of progression
- <2 mm bone loss/CAL over 5 years
- 0.25 to 1.0 bone loss/age
- Destruction corresponds with amount of biofilm deposit
- <10 cigarettes/day for smokers
- HbA1c <7.0%[31] in clients with diabetes

[31] HbA1c or glycated hemoglobin A1c is measured via a blood test. It indicates the average level of blood glucose levels over the past 2 to 3 months.

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Grade C

- Rapid rate of progression
- ≥2 mm bone loss/CAL over 5 years
- >1.0 bone loss/age
- Destruction exceeds amount of biofilm deposits
- Evidence of periods of rapid progression and/or early-onset disease (e.g., molar/incisor pattern)
- Expected poor response to therapy
- ≥10 cigarettes/day for smokers
- HbA1c ≥7.0% in clients with diabetes [16]

Framework for grading [16]

Grade			**Grade A**	**Grade B**	**Grade C**
Primary criteria	Direct evidence of progression	Long-term data (RBL or CAL)	No CAL or RBL over 5 years Slow rate of progression	<2 mm over 5 years Moderate rate of progression	≥2 mm over 5 years Rapid rate of progression
	Indirect evidence of progression	Percent bone loss/age	<0.25	0.25-1.0	>1.0
		Case phenotype	Heavy biofilm deposits with low levels of destruction	Destruction reflects biofilm deposits	Destruction exceeds biofilm deposits Evidence of periods of rapid progression and/or early-onset disease Expected poor response to therapy
Grade modifiers	Risk factors	Smoking	Non-smoker	Smoker <10 cigarettes/day	Smoker ≥10 cigarettes/day
		Diabetes	No diagnosis of diabetes	HbA1c <7%	HbA1c ≥7%

Note:

- Grade is used as an indicator of the rate of periodontitis progression.
- The primary criteria are either direct or indirect evidence of progression. Whenever available, direct evidence is used; in its absence indirect estimation is made using bone loss as a function of age at the most affected tooth or case presentation (radiographic bone loss expressed as percentage of root length divided by the age of the client, RBL/age).

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- Clinicians should initially assume Grade B and then look for evidence to move to grade A or C, if available. For example, poorly controlled diabetes would shift grade B to grade C.
- Once grade is established based on evidence of progression, it can be modified based on the presence of risk factors. [16]

Criteria for grading [16]

	Direct evidence	Indirect evidence		Grade modifiers	
	RBL or CAL over 5 years	% Bone loss/age	Biofilm	Smoking	HbA1c
Grade A	None	<0.25	Heavy biofilm Low destruction	Non-smoker	No diabetes diagnosis
Grade B	<2 mm	0.25-1.0	Destruction reflects biofilm deposits	<10 cigarettes/day	HbA1c <7%
Grade C	≥2 mm	>1.0	Destruction exceeds biofilm deposits Rapid progression Early onset	≥10 cigarettes/day	HbA1c ≥7%

Grade based on percent bone loss/age

Age (years)	Percentage of radiographic bone loss based on root length					
	15%	25%	33%	50%	66%	75%
15-20	B	C	C	C	C	C
21-25	B	C	C	C	C	C
26-30	B	B	C	C	C	C
31-35	B	B	B	C	C	C
36-40	B	B	B	C	C	C
41-45	B	B	B	C	C	C
46-50	B	B	B	C	C	C
51-55	B	B	B	B	C	C
56-60	B	B	B	B	C	C
61-65	A	B	B	B	C	C
66-70	A	B	B	B	B	C
71-75	A	B	B	B	B	C
76-80	A	B	B	B	B	B
81-85	A	B	B	B	B	B
86-90	A	B	B	B	B	B
91-95	A	B	B	B	B	B
96-100	A	B	B	B	B	B

Grade A <0.25; Grade B 0.25-1.0; Grade C >1.0;
Grades are approximated for each age cohort.

After periodontal therapy is completed, stable periodontitis is defined by gingival health on a reduced periodontium (BOP in <10% of the sites; shallow probing depths of ≤4 mm

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and no 4 mm sites with BOP). However, if BOP is still present at >10% of sites after therapy, the client is diagnosed with stable periodontitis with gingival inflammation. [3]

Sites with persistent probing depths ≥4 mm and BOP are likely unstable and require further treatment. Clients with periodontitis who were successfully treated and their condition is considered stable still remain at increased risk of recurrent periodontitis. Therefore, if gingival inflammation is present adequate measures to control inflammation should be implemented to prevent recurrent periodontitis. [3]

4. Peri-Implant diseases and conditions[32]

a. Peri-implant health – Absence of erythema, bleeding on gentle probing,[33] swelling, and suppuration, and no further bone loss has occurred following initial healing.

b. Peri-implant mucositis – Inflammation is confined to the soft tissue and no further bone loss following initial healing. The main clinical characteristic is bleeding on gentle probing. Erythema, swelling, and/or suppuration may also be present. An increase in probing depth is often detected due to swelling or decreased probing resistance.

c. Peri-implantitis – Peri-implant mucosa inflammation and progressive bone loss. For example, clinical signs of inflammation, bleeding on gentle probing, and/or suppuration, increased probing depths and/or recession of the mucosal margin in addition to radiographic bone loss.

d. Peri-implant soft and hard tissue deficiencies – Deficiencies at implant sites may result from various factors that can lead to complications affecting implant survival, such as natural resorption after tooth extraction, trauma, infectious diseases (e.g., periodontitis, peri-implantitis, endodontic infections), growth and development, sinus floor expansion, anatomical preconditions, mechanical overload, thin soft tissues, lack of keratinized mucosa, implant malposition, tooth migration, and systemic diseases. [1] [17] [18]

EFP clinical treatment guideline for stages I−III periodontitis

The guideline entitled *Treatment of stage I-III periodontitis – The EFP S3 level clinical practice guideline* offers 62 clinical treatment recommendations. Each recommendation includes the grade of recommendation and the strength of consensus.

Strength of recommendation:

- Grade A: strong recommendation: i.e., recommended (↑↑) or not recommended (↓↓)
- Grade B: recommendation: i.e., suggested (↑) or not suggested (↓)
- Grade O: open recommendation: i.e., may be considered (←→)

[32] Refer to Episode #46 for additional information on peri-implant diseases and conditions classification.
[33] Dental implants should be probed with a light force (0.25 N or 25 grams of pressure) to avoid trauma.

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If the group felt the evidence was not clear enough to support a recommendation, statements were formulated, including the need for additional research.

Strength of consensus:
- Unanimous consensus (agreement of 100% of participants)
- Strong consensus (agreement of >95%)
- Consensus (agreement of 75-95%)
- Simple majority (agreement of 50-74%) [3]

Step-by-step approach to treatment
Once a client is diagnosed with periodontitis, the guideline recommends periodontal therapy based on a 4-step approach. Naturally, before any treatment is rendered, the client is first informed of the diagnosis, including etiology of the disease, risk factors, and treatment options. Informed consent is obtained and a personalized treatment care plan is devised with client input. The plan may need revising during treatment, depending on client preferences, clinical findings, and overall health changes. [3]

Step 1
This step involves individualized advice aimed at guiding behaviour change to motivate clients to adopt good oral hygiene and a healthy lifestyle to reduce inflammation. It also includes professional mechanical supragingival plaque and calculus removal, as well as removal of possible plaque-retentive factors that may impair oral hygiene practices.

Risk factor control includes all health behavioural change interventions to eliminate or mitigate risk factors for periodontitis onset and progression, such as smoking cessation, improved metabolic control of diabetes, and possibly physical exercise, dietary counselling, and weight loss.

The first step of therapy should be implemented with all clients with periodontitis, regardless of the stage of their disease. Frequent re-evaluation is important to continue client motivation and adherence; assess, improve, and modify home care as required; and assess the provided interventions. [3]

Step 2
This step of therapy (cause-related therapy) is targeted at reducing/eliminating subgingival biofilm and calculus through subgingival instrumentation.

In addition, other nonsurgical interventions may be utilized as required, such as the use of adjunctive:
- Physical or chemical agents
- Host-modulating agents (local or systemic)
- Subgingival locally delivered antimicrobials
- Systemic antimicrobials

The second step of therapy should be used for all clients with periodontitis, regardless of their disease stage, only in teeth with loss of periodontal support, and/or periodontal pocket formation. Note the first and second steps of therapy can be delivered

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simultaneously, especially if deep probing depths are present to prevent periodontal abscess formation.

Periodontal re-evaluation after second step therapy occurs once periodontal tissues have healed. If the goals of therapy were not achieved (i.e., no periodontal pockets >4 mm with BOP or no deep periodontal pockets ≥6 mm), the third step of therapy should be considered. If therapy goals were achieved, clients should be placed in a supportive periodontal maintenance program. [3]

Step 3
This step is targeted at treating areas that do not respond to second step therapy (i.e., presence of pockets ≥4 mm with BOP or presence of deep periodontal pockets ≥6 mm). The purpose of step 3 interventions is to gain access for subgingival instrumentation or to regenerate or resect lesions that add complexity in the management of periodontitis (e.g., intra-bony and furcation lesions).

Step 3 interventions may include:
- Repeated subgingival instrumentation with or without adjunctive therapies
- Access flap periodontal surgery
- Resective periodontal surgery
- Regenerative periodontal surgery

The individual response to the third step of therapy should be re-evaluated. Ideally goals of therapy should be achieved and clients should be placed in supportive periodontal care. However, therapy goals may not be achievable in all teeth in clients with severe stage III periodontitis. [3]

Step 4
This step provides long-term supportive periodontal care to maintain periodontal stability and prevent relapse. It combines preventive and therapeutic interventions outlined in step 1 and 2 of therapy, depending on the client's periodontal status. Interventions include advice on healthy lifestyles and good oral hygiene, regular assessments, and professional debridement.

This step should be provided at regular intervals according to client needs. During any of these recare visits recurrent disease may be detected requiring retreatment. In these situations, a proper diagnosis and treatment plan should be reinstituted.

During any of the steps of therapy, tooth extraction may be considered if the affected teeth have a hopeless prognosis. [3]

Take home messages
Untreated or inadequately treated periodontitis can lead to tooth loss negatively impacting chewing, eating, speech, aesthetics, and quality of life. Periodontitis is preventable and treatable in the majority of cases. The EFP guideline is evidence-based and supports oral health professionals in their clinical decision-making for periodontal therapy. The guideline aims to improve the quality of periodontal treatment, reduce

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tooth loss associated with periodontitis, and essentially improve overall general health and quality of life.

The AAP classification system for periodontal and peri-implant diseases is necessary in order to provide a framework to scientifically study the etiology, pathogenesis, and treatment of periodontal diseases. In addition, the AAP classification system allows clinicians to properly diagnose and treat individuals with periodontal and peri-implant diseases and conditions. Incorporating the medical model of staging and grading into the diagnosis of periodontitis allows for a more individualized diagnosis and enhances communication and interprofessional collaboration between oral health and medical professionals.

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Client Resources

Periodontal (Gum) Disease, ODHA Fact Sheet
https://odha.on.ca/wp-content/uploads/2016/08/Gum-Disease.14.1-copyright.pdf

Oral Health and Overall Health, ODHA Fact Sheet
https://odha.on.ca/wp-content/uploads/2016/08/Overall-Health.14.1-copyright.pdf

From Head to Toe: Preventing and Treating Gum Disease for Overall Health, CDHO Fact Sheet
https://www.cdho.org/docs/default-source/pdfs/oral-health-rpt/oralhealthmatters_gumdisease.pdf

From Head to Toe: Smoking and Your Oral Health, CDHO Fact Sheet
https://www.cdho.org/docs/default-source/pdfs/oral-health-rpt/oralhealthmatters_smoking.pdf

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5 Medical Conditions That Hurt Your Mouth, CDHO Fact Sheet
https://www.cdho.org/docs/default-source/pdfs/oral-health-rpt/article-5-medical-conditions.pdf

Additional Resources

Classification at-a-Glance, American Academy of Periodontology, 2018
https://www.perio.org/wp-content/uploads/2019/08/Classification-at-a-glance.pdf

Staging and Grading Periodontitis, American Academy of Periodontology
https://www.perio.org/wp-content/uploads/2019/08/Staging-and-Grading-Periodontitis.pdf

Three Steps to Staging and Grading a Patient, American Academy of Periodontology
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Frequently Asked Questions on the 2018 Classification of Periodontal and Peri-Implant Diseases and Conditions, American Academy of Periodontology
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https://www.efp.org/fileadmin/uploads/efp/Documents/Campaigns/New_Classification/Guidance_Notes/report-02.pdf

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Peri-implant health, peri-implant mucositis, and peri-implantitis, Berglundh, T. European Federation of Periodontology, March 2019, p 1-8
https://www.efp.org/fileadmin/uploads/efp/Documents/Campaigns/New_Classification/Guidance_Notes/report-04.pdf

EFP Videos

Periodontal health and gingival diseases and conditions; Chapple, I. European Federation of Periodontology, March 18, 2020
Five short videos are available:

- Periodontal Health and Gingivitis: Introduction (10:26)
- Expert paper 1 Periodontal health (11:56)
- Expert paper 2 Plaque induced gingival conditions (3:35)
- Expert paper 3 - Non-plaque-induced gingival diseases (17:20)
- Expert paper 4 - Plaque-induced gingivitis - Case definition and diagnostic considerations (14:40)

https://www.efp.org/publications/special-publications/clinical-practice-guideline-new-classification/resources/periodontal-health-and-gingival-diseases-and-conditions-29062/

Periodontitis, Sanz, M; Tonetti, M. European Federation of Periodontology, March 18, 2020
Ten short videos are available:

- Periodontitis: Introduction (10:24)
- Periodontitis: Step1 new patient. Screening for signs of periodontitis presence (4:09)
- Periodontitis: Step2 Patient suspected of periodontitis (2:46)
- Periodontitis: Step3a. Patient is a periodontitis case whose stage needs to be established (1:42)
- Periodontitis: Step3b. Stages III and IV versus I and II (2:35)
- Periodontitis: Step3c. Stages I II III and IV (1:53)
- Periodontitis: Step4a. Grading when there are not existing records (2:42)
- Periodontitis: Step4b. Grading when there are existing records (1:23)
- Periodontitis: Treatment plan - Criteria Stages – Frameworks (8:27)
- Periodontitis: Clinical examples and points to keep in mind (11:34)

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Six short videos are available:

- Systemic and other periodontal conditions – Introduction (1:46)
- Expert paper 1 - Manifestations of systemic diseases and conditions (7:50)
- Expert paper 2 - Mucogingival conditions in the natural dentition (12:50)
- Expert paper 3 - Occlusal trauma and excessive occlusal forces (3:00)
- Expert paper 4 - Dental prosthesis and tooth-related factors (5:40)
- Expert paper - Periodontal abscesses lesions and endo-periodontal lesions (5:31)

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Five short videos are available:

- Peri-implant diseases and conditions. Introduction (3:13)
- Peri-implant health (2:34)
- Peri-implant mucositis (3:25)
- Peri-implantitis (5:54)
- Case definitions and diagnostic considerations (3:40)

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