Received: 16 December 2017 | Revised: 7 February 2018 | Accepted: 12 February 2018

DOI: 10.1111/jcpe.12951

2017 WORLD WORKSHOP

WILEY Journal of Clinical Periodontology

Periodontal manifestations of systemic diseases and developmental and acquired conditions: Consensus report of workgroup 3 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions

Søren Jepsen[1] | Jack G. Caton[2] | Jasim M. Albandar[3] | Nabil F. Bissada[4] | Philippe Bouchard[5] | Pierpaolo Cortellini[6] | Korkud Demirel[7] | Massimo de Sanctis[8] | Carlo Ercoli[9] | Jingyuan Fan[10] | Nicolaas C. Geurs[11] | Francis J. Hughes[12] | Lijian Jin[13] | Alpdogan Kantarci[14] | Evanthia Lalla[15] | Phoebus N. Madianos[16] | Debora Matthews[17] | Michael K. McGuire[18] | Michael P. Mills[19] | Philip M. Preshaw[20] | Mark A. Reynolds[21] | Anton Sculean[22] | Cristiano Susin[23] | Nicola X. West[24] | Kazuhisa Yamazaki[25]

[1]Department of Periodontology, Operative and Preventive Dentistry, University of Bonn, Bonn, Germany

[2]University of Rochester, Periodontics, Eastman Institute for Oral Health, Rochester, NY, USA

[3]Department of Periodontology and Oral Implantology, Temple University School of Dentistry, Philadelphia, PA, USA

[4]Case Western Reserve University, Cleveland, OH, USA

[5]U.F.R. d'Odontologie, Université Paris Diderot, Hôpital Rothschild AP-HP, Paris, France

[6]Private practice, Firenze, Italy; European Research Group on Periodontology, Bern, Switzerland

[7]Department of Periodontology, Istanbul University, Istanbul, Turkey

[8]Department of Periodontology, Università Vita e Salute San Raffaele, Milan, Italy

[9]University of Rochester, Prosthodontics & Periodontics, Eastman Institute for Oral Health, Rochester, NY, USA

[10]University of Rochester, Periodontics, Eastman Institute for Oral Health, Rochester, NY, USA

[11]Department of Periodontology, University of Alabama at Birmingham, School of Dentistry, Birmingham, AL, USA

[12]King's College London Dental Institute, London, UK

[13]Discipline of Periodontology, Faculty of Dentistry, The University of Hong Kong, Prince Philip Dental Hospital, Hong Kong SAR, China

[14]Forsyth Institute, Cambridge, MA, USA

[15]Columbia University College of Dental Medicine, Division of Periodontics, New York, NY, USA

[16]Department of Periodontology, School of Dentistry, National and Kapodistrian University of Athens, Greece

[17]Faculty of Dentistry, Dalhousie University, Halifax, Nova Scotia

[18]Private practice, Perio Health Professionals, Houston, TX, USA

[19]Department of Periodontics, University of Texas Health Science Center at San Antonio, TX, USA

[20]Centre for Oral Health Research and Institute of Cellular Medicine, Newcastle University, Newcastle upon Tyne, UK

[21]University of Maryland, School of Dentistry, Department of Advanced Oral Sciences and Therapeutics, Baltimore, MD, USA

[22]Department of Periodontology, University of Bern, Switzerland

[23]Department of Periodontics, Augusta University Dental College of Georgia, Augusta, GA, USA

[24]Restorative Dentistry and Periodontology, School of Oral and Dental Sciences, Bristol Dental School & Hospital, Bristol, UK

[25]Research Unit for Oral-Systemic Connection, Division of Oral Science for Health Promotion, Niigata University Graduate School of Medical and Dental Sciences, Niigata, Japan

Correspondence
Prof. Søren Jepsen, Department of Periodontology, Operative and Preventive Dentistry, University of Bonn, Welschnonnenstrasse 17, 53111 Bonn, Germany.
Email: jepsen@uni-bonn.de

Sources of Funding: The workshop was planned and conducted jointly by the American Academy of Periodontology and European Federation of Periodontology with financial support from the American Academy of Periodontology Foundation, Colgate, Johnson & Johnson Consumer Inc., Geistlich Biomaterials, SUNSTAR, and Procter & Gamble Professional Oral Health.

The proceedings of the workshop were jointly and simultaneously published in the *Journal of Periodontology* and *Journal of Clinical Periodontology*.

Abstract

Background: A variety of systemic diseases and conditions can affect the course of periodontitis or have a negative impact on the periodontal attachment apparatus. Gingival recessions are highly prevalent and often associated with hypersensitivity, the development of caries and non-carious cervical lesions on the exposed root surface and impaired esthetics. Occlusal forces can result in injury of teeth and periodontal attachment apparatus. Several developmental or acquired conditions associated with teeth or prostheses may predispose to diseases of the periodontium. The aim of this working group was to review and update the 1999 classification with regard to these diseases and conditions, and to develop case definitions and diagnostic considerations.

Methods: Discussions were informed by four reviews on 1) periodontal manifestions of systemic diseases and conditions; 2) mucogingival conditions around natural teeth; 3) traumatic occlusal forces and occlusal trauma; and 4) dental prostheses and tooth related factors. This consensus report is based on the results of these reviews and on expert opinion of the participants.

Results: Key findings included the following: 1) there are mainly rare systemic conditions (such as Papillon-Lefevre Syndrome, leucocyte adhesion deficiency, and others) with a major effect on the course of periodontitis and more common conditions (such as diabetes mellitus) with variable effects, as well as conditions affecting the periodontal apparatus independently of dental plaque biofilm-induced inflammation (such as neoplastic diseases); 2) diabetes-associated periodontitis should not be regarded as a distinct diagnosis, but diabetes should be recognized as an important modifying factor and included in a clinical diagnosis of periodontitis as a descriptor; 3) likewise, tobacco smoking – now considered a dependence to nicotine and a chronic relapsing medical disorder with major adverse effects on the periodontal supporting tissues – is an important modifier to be included in a clinical diagnosis of periodontitis as a descriptor; 4) the importance of the gingival phenotype, encompassing gingival thickness and width in the context of mucogingival conditions, is recognized and a novel classification for gingival recessions is introduced; 5) there is no evidence that traumatic occlusal forces lead to periodontal attachment loss, non-carious cervical lesions, or gingival recessions; 6) traumatic occlusal forces lead to adaptive mobility in teeth with normal support, whereas they lead to progressive mobility in teeth with reduced support, usually requiring splinting; 7) the term *biologic width* is replaced by *supracrestal tissue attachment* consisting of junctional epithelium and supracrestal connective tissue; 8) infringement of restorative margins within the supracrestal connective tissue attachment is associated with inflammation and/or loss of periodontal supporting tissue. However, it is not evident whether the negative effects on the periodontium are caused by dental plaque biofilm, trauma, toxicity of dental materials or a combination of these factors; 9) tooth anatomical factors are related to dental plaque biofilm-induced gingival inflammation and loss of periodontal supporting tissues.

Conclusion: An updated classification of the periodontal manifestations and conditions affecting the course of periodontitis and the periodontal attachment apparatus, as well as of developmental and acquired conditions, is introduced. Case definitions and diagnostic considerations are also presented.

KEYWORDS

anatomy, attachment loss, bruxism, classification, dental prostheses, dental restorations, diagnosis, genetic disease, gingival inflammation, gingival recession, gingival thickness, gingivitis, mucogingival surgery, occlusal trauma, periodontal disease, periodontitis, plastic periodontal surgery, systemic disease, tooth

A variety of systemic diseases and conditions can affect the course of periodontitis or have a negative impact on the periodontal attachment apparatus. Gingival recessions are highly prevalent and often associated with hypersensitivity, the development of caries and non-carious cervical lesions on the exposed root surface and impaired esthetics. Occlusal forces can result in injury of teeth and periodontal attachment apparatus. Several developmental or acquired conditions associated with teeth or prostheses may predispose to diseases of the periodontium.

The objectives of Workgroup 3 were to revisit the 1999 AAP classification for periodontal diseases and conditions, evaluate the updated evidence with regard to epidemiology and etiopathogenesis and to propose a new classification system together with case definitions and diagnostic considerations. In preparation, four position papers were provided, that had been accepted for publication. Discussions were based on these four reviews covering 1) periodontal manifestions of systemic diseases and conditions;[1] 2) mucogingival conditions around natural teeth;[2] 3) traumatic occlusal forces and occlusal trauma;[3] and 4) dental prostheses and tooth-related factors.[4] This consensus report is based on the results of these reviews and on expert opinions of the participants.

SYSTEMIC DISEASES AND CONDITIONS THAT AFFECT THE PERIODONTAL SUPPORTING TISSUES

Is it possible to categorize systemic diseases and conditions based on the underlying mechanisms of their effect on the periodontal supporting tissues?

Systemic diseases and conditions that can affect the periodontal supporting tissues can be grouped into broad categories as listed in Albandar et al.,[1] for example genetic disorders that affect the host immune response or affect the connective tissues, metabolic and endocrine disorders, and inflammatory conditions. In the future, it is anticipated that further refinement of these categories will be possible.

Are there diseases and conditions that can affect the periodontal supporting tissues?

There are many diseases and conditions that can affect the periodontal tissues either by 1) influencing the course of periodontitis or 2) affecting the periodontal supporting tissues independently of dental plaque biofilm-induced inflammation. These include:

1a. Mainly rare diseases that affect the course of periodontitis (e.g., Papillon Lefevre Syndrome, leucocyte adhesion deficiency, and hypophosphatasia). Many of these have a major impact resulting in the early presentation of severe periodontitis.

1b. Mainly common diseases and conditions that affect the course of periodontitis (e.g., diabetes mellitus). The magnitude of the effect of these diseases and conditions on the course of periodontitis varies but they result in increased occurrence and severity of periodontitis.

2. Mainly rare conditions affecting the periodontal supporting tissues independently of dental plaque biofilm-induced inflammation (e.g., squamous cell carcinoma, Langerhans cell histiocytosis). This is a more heterogeneous group of conditions which result in breakdown of periodontal tissues and some of which may mimic the clinical presentation of periodontitis.

The full list of these diseases and conditions is presented in Table 1, adapted from Albandar et al.[1]

Particularly relating to those common conditions identified in 1b) above:

Should diabetes-associated periodontitis be a distinct diagnosis?

Given the current global diabetes epidemic and the challenges with timely identification and/or achieving glycemic goals in a large percentage of affected individuals, this disease is of particular importance.[5] Because of differences in prevalence between type 1 and type 2 diabetes most of the evidence for its adverse effects on periodontal tissues is from patients with type 2 diabetes.[6] The level of hyperglycemia over time, irrespective of the type of diabetes, is of importance when it comes to the magnitude of its effect on the course of periodontitis.[7]

There are no characteristic phenotypic features that are unique to periodontitis in patients with diabetes mellitus. On this basis diabetes-associated periodontitis is not a distinct disease. Nevertheless, diabetes is an important modifying factor of periodontitis, and should be included in a clinical diagnosis of periodontitis as a descriptor. According to the new classification of periodontitis,[8,9] the level of glycemic control in diabetes influences the grading of periodontitis.

There is mounting evidence of specific mechanistic pathways in the pathogenesis of periodontitis in patients with diabetes.[10] In a more etiologically driven classification this should require further consideration in the future.

TABLE 1 Classification of systemic diseases and conditions that affect the periodontal supporting tissues (adapted from Albandar et al.[1])

Classification	Disorders	ICD-10 code
1.	**Systemic disorders that have a major impact on the loss of periodontal tissues by influencing periodontal inflammation**	
1.1.	**Genetic disorders**	
1.1.1.	**Diseases associated with immunologic disorders**	
	Down syndrome	Q90.9
	Leukocyte adhesion deficiency syndromes	D72.0
	Papillon-Lefèvre syndrome	Q82.8
	Haim-Munk syndrome	Q82.8
	Chediak-Higashi syndrome	E70.3
	Severe neutropenia	
	– Congenital neutropenia (Kostmann syndrome)	D70.0
	– Cyclic neutropenia	D70.4
	Primary immunodeficiency diseases	
	– Chronic granulomatous disease	D71.0
	– Hyperimmunoglobulin E syndromes	D82.9
	Cohen syndrome	Q87.8
1.1.2.	**Diseases affecting the oral mucosa and gingival tissue**	
	Epidermolysis bullosa	
	– Dystrophic epidermolysis bullosa	Q81.2
	– Kindler syndrome	Q81.8
	Plasminogen deficiency	D68.2
1.1.3.	**Diseases affecting the connective tissues**	
	Ehlers-Danlos syndromes (types IV, VIII)	Q79.6
	Angioedema (C1-inhibitor deficiency)	D84.1
	Systemic lupus erythematosus	M32.9
1.1.4.	**Metabolic and endocrine disorders**	
	Glycogen storage disease	E74.0
	Gaucher disease	E75.2
	Hypophosphatasia	E83.30
	Hypophosphatemic rickets	E83.31
	Hajdu-Cheney syndrome	Q78.8
1.2.	**Acquired immunodeficiency diseases**	
	Acquired neutropenia	D70.9
	HIV infection	B24
1.3.	**Inflammatory diseases**	
	Epidermolysis bullosa acquisita	L12.3
	Inflammatory bowel disease	K50, K51.9, K52.9
2.	**Other systemic disorders that influence the pathogenesis of periodontal diseases**	
	Diabetes mellitus	E10 (type 1), E11 (type 2)
	Obesity	E66.9
	Osteoporosis	M81.9
	Arthritis (rheumatoid arthritis, osteoarthritis)	M05, M06, M15-M19
	Emotional stress and depression	F32.9
	Smoking (nicotine dependence)	F17
	Medications	
3.	**Systemic disorders that can result in loss of periodontal tissues independent of periodontitis**	
3.1.	**Neoplasms**	
	Primary neoplastic diseases of the periodontal tissues	
	– Oral squamous cell carcinoma	C03.0 – 1
	– Odontogenic tumors	D48.0
	– Other primary neoplasms of the periodontal tissues	C41.0
	Secondary metastatic neoplasms of the periodontal tissues	C06.8
3.2.	**Other disorders that may affect the periodontal tissues**	
	Granulomatosis with polyangiitis	M31.3
	Langerhans cell histiocytosis	C96.6
	Giant cell granulomas	K10.1
	Hyperparathyroidism	E21.0
	Systemic sclerosis (scleroderma)	M34.9
	Vanishing bone disease (Gorham-Stout syndrome)	M89.5

Can obesity affect the course of periodontitis?

The relationship between obesity and metabolic status, including hyperglycemia, is complex and it is difficult to unravel their relative contributions to effects on periodontitis. Nevertheless, recent meta-analyses consistently show a statistically significant positive association between

obesity and periodontitis.[11,12] However there are relatively few studies with longitudinal design, and the overall effect appears to be modest.[13,14]

Can osteoporosis affect the course of periodontitis?

There is conflicting evidence regarding the association between osteoporosis and periodontitis. A recent systematic review concluded that postmenopausal women with osteoporosis or osteopenia exhibit a modest but statistically significant greater loss of periodontal attachment compared with women with normal bone mineral density.[15]

Can rheumatoid arthritis affect the course of periodontitis?

A recent meta-analysis found a statistically significant but weak positive association between rheumatoid arthritis and periodontitis.[16] There is some evidence that periodontitis may contribute to the pathogenesis of rheumatoid arthritis, and therefore, longitudinal studies are required to clarify this association.

Should smoking-associated periodontitis be a distinct diagnosis?

Tobacco smoking is a prevalent behavior with severe health consequences. Although tobacco use was once classified as a habit, it is now considered a dependence to nicotine and a chronic relapsing medical disorder (International Classification of Diseases, Tenth Revision [ICD-10 F17]).

It is well established that smoking has a major adverse effect on the periodontal supporting tissues, increasing the risk of periodontitis by 2- to 5-fold.[17] There are no unique periodontal phenotypic features of periodontitis in smokers. On this basis smoking-associated periodontitis is not a distinct disease. Nevertheless, tobacco smoking is an important modifying factor of periodontitis, and should be included in a clinical diagnosis of periodontitis as a descriptor. According to the new classification of periodontitis,[8,9] the current level of tobacco use influences the grading of periodontitis.

Case definitions and diagnostic considerations

1a. **Rare conditions that may have major effects on the course of periodontitis**. Periodontitis (see Workgroup 2 case definition, Papapanou et al.[8]) is a manifestation of these conditions. Cases are defined as periodontitis in the presence of the condition. The full list, case definitions, and diagnostic considerations are shown in Albandar et al.[1] (Tables 2 to 6).

1b. **Common conditions with variable effects on the course of periodontitis**.
Periodontitis associated with diabetes mellitus: Periodontitis (see Workgroup 2 case definition, Papapanou et al.,[8] Tonetti et al.[9]) and diagnosis of diabetes mellitus.
Periodontitis associated with smoking: Periodontitis (see Workgroup 2 case definition, Papapanou et al.,[8] Tonetti et al.[9]) and previous or current smoking in pack-years.

2. **Conditions affecting the periodontal apparatus independently of dental plaque biofilm-induced inflammation**
Periodontal attachment loss occurring in:
Neoplastic diseases
Other diseases

The full list, case definitions, and diagnostic considerations are shown in Albandar et al.[1] (Tables 9 and 10).

MUCOGINGIVAL CONDITIONS AROUND THE NATURAL DENTITION

This consensus focuses on single and multiple facial/lingual recessions that could be related to various periodontal conditions/diseases. Clinical aspects such as mucogingival conditions and therapeutic interventions that are associated with gingival recessions are evaluated. The accompanying narrative review[2] reports data supporting this consensus paper on nine focused questions, case definitions, and a novel classification for gingival recessions.

What is the definition of recession?

Recession is defined as an apical shift of the gingival margin caused by different conditions/pathologies. It is associated with clinical attachment loss. This may apply to all surfaces (buccal/lingual/interproximal).

What are the possible consequences of gingival recession and root surface exposure to oral environment?

Impaired esthetics
Dentin hypersensitivity
Caries/non-carious cervical lesions (NCCL)

Besides the esthetic impairment caused by the apical shift of the gingival margin, the group also highlights the impact of the oral environment on the exposed root surface. The prevalence of dentin hypersensitivity, cervical caries, and especially non-carious cervical lesions, is very high and the latter is increasing with age.

Is the development of gingival recession associated with the gingival phenotype?

The group strongly suggests the adoption of the definition "periodontal phenotype"[18] to describe the combination of gingival phenotype (three-dimensional gingival volume) and the thickness of the buccal bone plate (bone morphotype). Most papers use the term "biotype".

a. Biotype: (Genetics) group of organs having the same specific genotype.

b. Phenotype: Appearance of an organ based on a multifactorial combination of genetic traits and environmental factors (its expression includes the biotype).

The phenotype indicates a dimension that may change through time depending upon environmental factors and clinical intervention and can be site-specific (phenotype can be modified, not the genotype). Periodontal phenotype is determined by gingival phenotype (gingival thickness, keratinized tissue width), and bone morphotype (thickness of the buccal bone plate).

Thin phenotype increases risk for gingival recession. Thin phenotypes are more prone to develop increasing recession lesions.[19,20]

How can the periodontal phenotype be assessed in a standardized and reproducible way?

It can be assessed by using a periodontal probe to measure the gingival thickness (GT) observing the periodontal probe shining through gingival tissue after being inserted into the sulcus:

1) Probe visible: thin (≤1 mm).
2) Probe not visible: thick (> 1 mm).

Different types of probes are used to assess GT: CPU 15 UNC, HuFriedy,[21] SE Probe SD12 Yellow, American Eagle Instruments.[22]

Note: Probe visibility was tested in samples of subjects with unknown gingival pigmentation. It is unknown if the same outcomes are to be expected in populations with different gingival pigmentation. A novel electronic customized caliper has been recently proposed to measure the gingival thickness with a controlled force.[23]

Additional information on the three-dimensional gingival volume can be obtained by measuring the keratinized tissue width (KTW) from the gingival margin to the mucogingival junction. *Bone morphotypes* have been measured radiographically with cone-beam computed tomography (CBCT). The group does not recommend the application of CBCT in this context. There is evidence reporting a correlation between gingival thickness and buccal bone plate.[24,25] To date, periodontal phenotype cannot be assessed in full, while gingival phenotype (GT and KTW) can be assessed in a standardized and reproducible way.

Is there a certain amount (thickness and width) of gingiva necessary to maintain periodontal health?

Any amount of gingiva is sufficient to maintain periodontal health when optimal oral hygiene is attainable.

Does improper toothbrushing influence the development and progression of gingival recessions?

Data are inconclusive. Some studies reported a positive association, some a negative, and some no association.[26]

Does intrasulcular restorative margin placement influence the development of gingival recession?

Intrasulcular restorative/prosthetic cervical margin placement may be associated with the development of gingival recession particularly in a thin periodontal phenotype.

What is the effect of orthodontic treatment on the development of gingival recession?

1. Several studies report the observation of gingival recessions following orthodontic treatment (mainly on the effect of mandibular incisor proclination). The reported prevalence spans 5% to 12% at the end of treatment. Authors report an increase of the prevalence up to 47% in long-term observations (5 years).[27–30] One study reported a correlation between lower incisor proclination and thin phenotype.[31]
2. Direction of the tooth movement and the bucco-lingual thickness of the gingiva may play important roles in soft tissue alteration during orthodontic treatment.[32]

Do we need a new classification of gingival recession?

The group suggests the need for a new classification based upon anatomy.

Case definitions and diagnostic considerations

Mucogingival conditions

Within the individual variability of anatomy and morphology "normal mucogingival condition" can be defined as the "absence of pathosis (i.e. gingival recession, gingivitis, periodontitis)". There will be extreme conditions without obvious pathosis in which the deviation from what is considered "normal" in the oral cavity lies outside of the range of individual variability.[2]

a) Mucogingival condition with gingival recessions

A case with gingival recession presents with an apical shift of the gingival margin (*recession depth*). Relevant features contributing to the description of this condition are 1) the interdental clinical attachment level, 2) the gingival phenotype (*gingival thickness and keratinized tissue width*), 3) root surface condition (presence / absence of NCCL or caries), 4) detection of the CEJ, 5) tooth position, 6) aberrant frenum, and 7) number of adjacent recessions. Presence of recession can cause esthetic problems to the patients and be associated with dentin hypersensitivity.

b) Mucogingival condition without gingival recessions

A case without gingival recession can be described as the gingival phenotype (*gingival thickness and keratinized tissue width*), either at the entire dentition, or at individual sites. Relevant features

contributing to the description of this condition might be tooth position, aberrant frenum, or vestibular depth.

Gingival Recession

It is proposed to adopt a classification of gingival recession with reference to the interdental clinical attachment loss:[33]

- ***Recession Type 1 (RT1)***: Gingival recession with no loss of interproximal attachment. Interproximal CEJ is clinically not detectable at both mesial and distal aspects of the tooth.
- ***Recession Type 2 (RT2)***: Gingival recession associated with loss of interproximal attachment. The amount of interproximal attachment loss (measured from the interproximal CEJ to the depth of the interproximal sulcus/pocket) is less than or equal to the buccal attachment loss (measured from the buccal CEJ to the apical end of the buccal sulcus/pocket).
- ***Recession Type 3 (RT3)***: Gingival recession associated with loss of interproximal attachment. The amount of interproximal attachment loss (measured from the interproximal CEJ to the apical end of the sulcus/pocket) is higher than the buccal attachment loss (measured from the buccal CEJ to the apical end of the buccal sulcus/pocket).

Table 2 reports a diagnostic approach to classify gingival phenotype, gingival recession, and associated cervical lesions. This is a treatment-oriented classification supported by data included in the accompanying narrative review.[2]

OCCLUSAL TRAUMA AND TRAUMATIC OCCLUSAL FORCES

The group defined excessive occlusal force and renamed it *traumatic occlusal force*. *Traumatic occlusal force* is defined as any occlusal force resulting in injury of the teeth and/or the periodontal attachment apparatus. These were historically defined as excessive forces to denote that the forces exceed the adaptive capacity of the individual person or site. *Occlusal trauma* is a term used to describe the injury to the periodontal attachment apparatus, and is a histologic term. Nevertheless, the clinical presentation of the presence of occlusal trauma can be exhibited clinically as described in the case definition.

Does traumatic occlusal force or occlusal trauma cause periodontal attachment loss in humans?

There is no evidence that traumatic occlusal force or occlusal trauma causes periodontal attachment loss in humans.

Can traumatic occlusal force cause periodontal inflammation?

There is limited evidence from human and animal studies that traumatic occlusal forces can cause inflammation in the periodontal ligament.[3]

Does traumatic occlusal force accelerate the progression of periodontitis?

There is evidence from observational studies that traumatic occlusal forces may be associated with the severity of periodontitis.[34] Evidence from *animal* models indicate that traumatic occlusal forces may increase alveolar bone loss.[35,36] However, there is no evidence that traumatic occlusal forces can accelerate the progression of periodontitis *in humans*.

Can traumatic occlusal forces cause non-carious cervical lesions?

There is no credible evidence that traumatic occlusal forces cause non-carious cervical lesions.

What is the evidence that abfraction exists?

Abfraction, a term used to define a wedge-shaped defect that occurs at the cemento-enamel junction of affected teeth, has been claimed to be the result of flexure and fatigue of enamel and dentin. The existence of abfraction is not supported by current evidence.

Can traumatic occlusal forces cause gingival recession?

There is evidence from observational studies that occlusal forces do *not* cause gingival recession.[37,38]

Are orthodontic forces associated with adverse effects on the periodontium?

Evidence from animal models suggests that certain orthodontic forces can adversely affect the periodontium and result in root resorption, pulpal disorders, gingival recession and alveolar bone loss.[39,40] Conversely, there is evidence from observational studies that with good plaque control, teeth with a reduced but healthy periodontium can undergo successful tooth movement without compromising the periodontal support.[41,42]

Does the elimination of the signs of traumatic occlusal forces improve the response to treatment of periodontitis?

There is evidence from one randomized clinical trial that reducing tooth mobility may improve periodontal treatment outcomes.[43] There is insufficient clinical evidence evaluating the impact of eliminating signs of traumatic occlusal forces on response to periodontal treatment.

Should we still distinguish primary from secondary occlusal trauma in relation to treatment?

Primary occlusal trauma has been defined as injury resulting in tissue changes from traumatic occlusal forces applied to a tooth or teeth

TABLE 2 Classification of mucogingival conditions (gingival phenotype) and gingival recessions

Gingival site				Tooth site	
	REC Depth	GT	KTW	CEJ (A / B)	Step (+/-)
No recession					
RT1					
RT2					
RT3					

RT = recession type[33]
REC Depth = depth of the gingival recession
GT = gingival thickness
KTW = keratinized tissue width
CEJ = cemento-enamel junction (Class A = detectable CEJ, Class B = undetectable CEJ)
Step = root surface concavity (Class + = presence of a cervical step > 0.5 mm. Class – = absence of a cervical step > 0.5 mm)[44]

with normal periodontal support. This manifests itself clinically with *adaptive mobility* and is not progressive. Secondary occlusal trauma has been defined as injury resulting in tissue changes from normal or traumatic occlusal forces applied to a tooth or teeth with reduced support. Teeth with *progressive mobility* may also exhibit migration and pain on function. Current periodontal therapies are directed primarily to address etiology; in this context, traumatic occlusal forces. Teeth with progressive mobility may require splinting for patient comfort.

The group considered the term *reduced periodontium* related to secondary occlusal trauma and agreed there were problems with defining "*reduced periodontium*". A reduced periodontium is only meaningful when mobility is progressive indicating the forces acting on the tooth exceed the adaptive capacity of the person or site.

Case definitions and diagnostic considerations

1. *Traumatic occlusal force* is defined as any occlusal force resulting in injury of the teeth and/or the periodontal attachment apparatus. These were historically defined as *excessive forces* to denote that the forces exceed the adaptive capacity of the individual person or site. The presence of *traumatic occlusal forces* may be indicated by one or more of the following: fremitus, tooth mobility, thermal sensitivity, excessive occlusal wear, tooth migration, discomfort/pain on chewing, fractured teeth, radiographically widened periodontal ligament space, root resorption, and hypercementosis. Clinical management of traumatic occlusal forces is indicated to prevent and treat these signs and symptoms.
2. *Occlusal trauma* is a lesion in the periodontal ligament, cementum and adjacent bone caused by traumatic occlusal forces. It is a histologic term; however, a clinical diagnosis of occlusal trauma may be made in the presence of one or more of the following: progressive tooth mobility, adaptive tooth mobility (fremitus), radiographically widened periodontal ligament space, tooth migration, discomfort/pain on chewing, and root resorption.

As some of the signs and symptoms of traumatic occlusal forces and occlusal trauma may also be associated with other conditions, an appropriate differential analysis must be performed to rule out other etiologic factors.

The group agreed to a classification related to traumatic occlusal forces and occlusal trauma (Table 3).

DENTAL PROSTHESES AND TOOTH-RELATED FACTORS

Several conditions, associated with prostheses and teeth, may predispose to diseases of the periodontium and were extensively reviewed in a background paper.[4] The extent to which these conditions contribute to the disease process may be dependent upon the susceptibility of the individual patient.

What is the biologic width?

Biologic width is a commonly used clinical term to describe the apico-coronal variable dimensions of the supracrestal attached tissues. The supracrestal attached tissues are histologically composed of the junctional epithelium and supracrestal connective tissue attachment. *The term biologic width* should be replaced by *supracrestal tissue attachment*.

Is infringement of restorative margins within the supracrestal connective tissue attachment associated with inflammation and/or loss of periodontal supporting tissues?

Available evidence from human studies supports that infringement within the supracrestal connective tissue attachment is associated with inflammation and loss of periodontal supporting tissue. Animal studies corroborate this statement and provide histologic evidence that infringement within the supracrestal connective tissue attachment is associated with inflammation and subsequent loss of periodontal supporting tissues, accompanied with an apical shift of the junctional epithelium and supracrestal connective tissue attachment.

Are changes in the periodontium caused by infringement of restorative margins within supracrestal connective tissue attachment due to dental plaque biofilm, trauma, or some other factors?

Given the available evidence, it is not possible to determine if the negative effects on the periodontium associated with restoration margins located within the supracrestal connective tissue attachment is caused by dental plaque biofilm, trauma, toxicity of dental materials, or a combination of these factors.

For subgingival indirect dental restorations, are design, fabrication, materials, and delivery associated with gingival inflammation and/or loss of periodontal supporting tissues?

There is evidence to suggest that tooth supported/retained restorations and their design, fabrication, delivery, and materials can be associated with plaque retention and loss of clinical attachment. Optimal restoration margins located within the gingival sulcus do not cause gingival inflammation if patients are compliant with self-performed plaque control and periodic maintenance. Currently, there is a paucity of evidence to define a correct emergence profile.

Are fixed dental prostheses associated with periodontitis or loss of periodontal supporting tissues?

The available evidence does not support that optimal fixed dental prostheses are associated with periodontitis. There is evidence to suggest that design, fabrication, delivery and materials used for fixed dental prostheses procedures can be associated with plaque retention, gingival recession and loss of supporting periodontal tissues.

Are removable dental prostheses associated with periodontitis or loss of periodontal supporting tissues?

The available evidence does not support that optimal removable dental prostheses are associated with periodontitis. If plaque control is established and maintenance procedures performed, removable dental prostheses are not associated with greater plaque accumulation, periodontal loss of attachment and increased tooth mobility. However, if patients perform inadequate plaque control and do not attend periodic maintenance appointments, removable dental prostheses could act as dental plaque biofilm retentive factors, be associated with gingivitis/periodontitis, increased mobility and gingival recession.

Can tooth-related factors enhance plaque accumulation and retention and act as a contributing factor to gingival inflammation and loss of periodontal supporting tissues?

Tooth anatomical factors (cervical enamel projections, enamel pearls, developmental grooves), root proximity, abnormalities and fractures, and tooth relationships in the dental arch are related to dental plaque biofilm-induced gingival inflammation and loss of periodontal supporting tissues.

Can adverse reactions to dental materials occur?

Dental materials may be associated with hypersensitivity reactions which can clinically appear as localized inflammation that does not respond to adequate measures of plaque control. Additional diagnostic measures will be needed to confirm hypersensitivity. Limited in vitro evidence suggests selected ions liberated from dental materials may adversely affect cell viability and function.

TABLE 3 Classification of traumatic occlusal forces on the periodontium

1. Occlusal trauma
A. Primary occlusal trauma
B. Secondary occlusal trauma
C. Orthodontic forces

What is altered passive eruption?

Abnormal dentoalveolar relationships associated with altered passive tooth eruption is a developmental condition that is characterized by the gingival margin (and sometimes bone) located at a more coronal level. This condition may be clinically associated with the formation of pseudopockets and/or esthetic concerns.

Case definitions and diagnostic considerations

1. *Supracrestal attached tissues* are composed of the junctional epithelium and the supracrestal connective tissue attachment. This was formally referred to as the *biologic width*. The apico-coronal dimension of the supracrestal attached tissues is variable. Clinically, there is evidence that placement of restorative margins within the supracrestal connective tissues is associated with inflammation and loss of periodontal supporting tissues. Additional research is necessary to clarify the effects of placement of restorative margins within the junctional epithelium.
2. *Altered passive eruption* is a developmental condition with abnormal dento-alveolar relationships. Clinically, this condition is characterized by the gingival margin (and sometimes bone) located at a more coronal level, which leads to pseudopockets and esthetic concerns. Correction of this condition can be accomplished with periodontal surgery.

TABLE 4 Classification of factors related to teeth and to dental prostheses that can affect the periodontium

A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis
1. Tooth anatomic factors
2. Root fractures
3. Cervical root resorption, cemental tears
4. Root proximity
5. Altered passive eruption
B. Localized dental prosthesis-related factors
1. Restoration margins placed within the supracrestal attached tissues
2. Clinical procedures related to the fabrication of indirect restorations
3. Hypersensitivity/toxicity reactions to dental materials

The workgroup agreed to a classification of dental prosthesis and tooth-related factors (Table 4).

ACKNOWLEDGMENTS AND DISCLOSURES

Workshop participants filed detailed disclosure of potential conflicts of interest relevant to the workshop topics, and these are kept on file. The authors receive, or have received, research funding, consultant fees, and/or lecture compensation from the following companies: Biolase, Colgate, Dentsply Sirona, Geistlich Pharma, Nobel Biocare, OraPharma, Osteogenics Biomedical, Osteology Foundation, and Straumann.

REFERENCES

1. Albandar JM, Susin C, Hughes FJ. Manifestations of systemic diseases and conditions that affect the periodontal attachment apparatus: case definitions and diagnostic considerations. *J Clin Periodontol*. 2018;*45*(Suppl 20):S171–S189.
2. Cortellini P, Bissada NF. Mucogingival conditions in the natural dentition: narrative review, case definitions and diagnostic considerations. *J Clin Periodontol*. 2018;*45*(Suppl 20):S190–S198.
3. Fan J, Caton JG. Occlusal trauma and excessive occlusal forces: narrative review, case definitions and diagnostic considerations. *J Clin Periodontol*. 2018;*45*(Suppl 20):S199–S206.
4. Ercoli C, Caton JG. Dental prostheses and tooth-related factors. *J Clin Periodontol*. 2018;*45*(Suppl 20):S207–S218.
5. International Diabetes Federation. *IDF Diabetes Atlas*, 8th ed. Brussels, Belgium: International Diabetes Federation; 2017.
6. Sanz M, Ceriello A, Buysschaert M, et al. Scientific evidence on the links between periodontal diseases and diabetes: consensus report and guidelines of the joint workshop on periodontal diseases and diabetes by the International Diabetes Federation and the European Federation of Periodontology. *J Clin Periodontol*. 2018;*45*:138–149.
7. Lalla E, Papapanou PN. Diabetes mellitus and periodontitis: a tale of two common interrelated diseases. *Nat Rev Endocrinol*. 2011 *28*(7):738–748.
8. Papapanou PN, Sanz M, 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 Clin Periodontol*. 2018;*45*(Suppl 20):S162–S170.
9. Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: framework and proposal of a new classification and case definition. *J Clin Periodontol*. 2018;*45*(Suppl 20):S149–S161.
10. Polak D, Shapira L. An update on the evidence for pathogenic mechanisms that may link periodontitis and diabetes. *J Clin Periodontol*. 2018;*45*:150–166.
11. Chaffee BW, Weston SJ. Association between chronic periodontal disease and obesity: a systematic review and meta-analysis. *J Periodontol*. 2010;*81*:1708–1724.
12. Suvan J, D'Aiuto F, Moles DR, Petrie A, Donos N. Association between overweight/obesity and periodontitis in adults. A systematic review. *Obes Rev*. 2011;*12*:e381–404.
13. Nascimento GG, Leite FR, Do LG, Peres KG, Correa MB, Demarco FF, Peres MA. Is weight gain associated with the incidence of periodontitis? A systematic review and meta-analysis. *J Clin Periodontol*. 2015;*42*:495–505.
14. Gaio EJ, Haas AN, Rösing CK, Oppermann RV, Albandar JM, Susin C. Effect of obesity on periodontal attachment loss progression: a 5-year population-based prospective study. *J Clin Periodontol*. 2016;*43*:557–565.
15. Penoni DC, Fidalgo TK, Torres SR, et al. Bone density and clinical periodontal attachment in postmenopausal women: a systematic review and meta-analysis. *J Dent Res*. 2017;*96*:261–269.
16. Fuggle NR, Smith TO, Kaul A, Sofat N. Hand to mouth: a systematic review and meta-analysis of the association between rheumatoid arthritis and periodontitis. *Front Immunol*. 2016;*7*:80. https://doi.org/10.3389/fimmu.2016.00080.eCollection 2016.
17. Warnakulasuriya S, Dietrich T, Bornstein MM, et al. Oral health risks of tobacco use and effects of cessation. *Int Dent*. 2010;*60*:7–30.
18. *Dictionary of Biology*, 6th ed. Oxford: Oxford University Press; 2008. Print ISBN-13: 9780199204625.
19. Agudio G, Cortellini P, Buti J, Pini Prato G. Periodontal conditions of sites treated with gingival augmentation surgery compared with untreated contralateral homologous sites: an 18- to 35-year long-term study. *J Periodontol*. 2016;*87*:1371–1378.
20. Chambrone L, Tatakis DN. Long-term outcomes of untreated buccal gingival recessions: a systematic review and meta-analysis. *J Periodontol*. 2016;*87*:796–808.
21. De Rouck T, Eghbali R, Collys K, De Bruyn H, Cosyn J. The gingival biotype revisited: transparency of the periodontal probe through the gingival margin as a method to discriminate thin from thick gingiva. *J Clin Periodontol*. 2009;*36*:428–433.
22. Kan JY, Morimoto T, Rungcharassaeng K, Roe P, Smith DH. Gingival biotype assessment in the esthetic zone: visual versus direct measurement. *Int J Periodontics Restorative Dent*. 2010;*30*:237–243.
23. Liu F, Pelekos G, Jin LJ. The gingival biotype in a cohort of Chinese subjects with and without history of periodontal disease. *J Periodontal Res*. 2017;*52*:1004–1010.
24. Zweers J, Thomas RZ, Slot DE, Weisgold AS, Van der Weijden GA. Characteristics of periodontal biotype, its dimensions, associations and prevalence: a systematic review. *J Clin Periodontol*. 2014;*41*:958–971.
25. Ghassemian M, Lajolo C, Semeraro V, et al. Relationship between biotype and bone morphology in the lower anterior mandible: an observational study. *J Periodontol*. 2016;*87*:680–689.
26. Heasman PA, Holliday R, Bryant A, Preshaw PM. Evidence for the occurrence of gingival recession and non-carious cervical lesions as a consequence of traumatic toothbrushing. *J Clin Periodontol*. 2015;*42* Suppl 16: S237–255.
27. Aziz T, Flores-Mir C. A systematic review of the association between appliance-induced labial movement of mandibular incisors and gingival recession. *Aust Orthod J*. 2011;*27*:33–39.
28. Renkema AM, Fudalej PS, Renkema A, Kiekens R, Katsaros C. Development of labial gingival recessions in orthodontically treated patients. *Am J Orthod Dentofacial Orthop*. 2013;*143*:206–212.
29. Renkema AM, Navratilova Z, Mazurova K, Katsaros C, Fudalej PS. Gingival labial recessions and the post-treatment proclination of mandibular incisors. *Eur J Orthod*. 2015;*37*:508–513.
30. Morris JW, Campbell PM, Tadlock LP, Boley, Buschang PH. Prevalence of gingival recession after orthodontic tooth movements. *Am J Orthod Dentofacial Orthop*. 2017;*151*:851–859.
31. Rasperini G, Acunzo R, Cannalire P, Farronato G. Influence of periodontal biotype on root surface exposure during orthodontic treatment:a preliminary study. *Int J Periodontics Restorative Dent*. 2015;*35*:665–675.
32. Kim DM, Neiva R. Periodontal soft tissue non-root coverage procedures: a systematic review from the AAP regeneration workshop. *J Periodontol*. 2015;*86*(S2): S56-S72.
33. Cairo F, Nieri M, Cincinelli S, Mervelt J, Pagliaro U. The interproximal clinical attachment level to classify gingival recessions and predict root coverage outcomes: an explorative and reliability study. *J Clin Periodontol*. 2011;*38*:661–666.
34. Ismail AI, Morrison EC, Burt BA, Caffesse RG, Kavanagh MT. Natural history of periodontal disease in adults: findings from the Tecumseh Periodontal Disease Study, 1959 – 87. *J Dent Res*. 1990;*69*:430–435.
35. Kaku M, Uoshima K, Yamashita Y, Miura H. Investigation of periodontal ligament reaction upon excessive occlusal load – osteopontin induction among periodontal ligament cells. *J Periodontal Res*. 2005;*40*:59–66.

36. Yoshinaga Y, Ukai T, Abe Y, Hara Y. Expression of receptor activator of nuclear factor kappa B ligand relates to inflammatory bone resorption, with or without occlusal trauma, in rats. *J Periodontal Res*. 2007;*42*:402–409.
37. Bernimoulin J, Curilović Z. Gingival recession and tooth mobility. *J Clin Periodontol*. 1977;*4*(2): 107–114.
38. Harrel SK, Nunn ME. The effect of occlusal discrepancies on gingival width. *J Periodontol*. 2004;*75*:98–105.
39. Stenvik A, Mjör IA. Pulp and dentine reactions to experimental tooth intrusion. A histologic study of the initial changes. *Am J Orthod*. 1970;*57*:370–385.
40. Wennström JL, Lindhe J, Sinclair F, Thilander B. Some periodontal tissue reactions to orthodontic tooth movement in monkeys. *J Clin Periodontol*. 1987;*14*:121–129.
41. Eliasson LA, Hugoson A, Kurol J, Siwe H. The effects of orthodontic treatment on periodontal tissues in patients with reduced periodontal support. *Eur J Orthod*. 1982;*4*:1–9.
42. Boyd RL, Leggott PJ, Quinn RS, Eakle WS, Chambers D. Periodontal implications of orthodontic treatment in adults with reduced or normal periodontal tissues versus those of adolescents. *Am J Orthod Dentofacial Orthop*. 1989;*96*:191–198.
43. Cortellini P, Tonetti MS, Lang NP, et al. The simplified papilla preservation flap in the regenerative treatment of deep intrabony defects: clinical outcomes and postoperative morbidity. *J Periodontol*. 2001;*72*:1702–1712.
44. Pini-Prato G, Franceschi D, Cairo F, Nieri M, Rotundo R. Classification of dental surface defects in areas of gingival recession. *J Periodontol*. 2010;*81*:885–890.

How to cite this article: Jepsen S, Caton JG, et al. Periodontal manifestations of systemic diseases and developmental and acquired conditions: Consensus report of workgroup 3 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Clin Periodontol*. 2018;45(Suppl 20):S219–S229. https://doi.org/10.1111/jcpe.12951

FIGURE 1 Participants of Workgroup 3