

Peri-Implant Diseases And Conditions: An Executive Summary Of The New Global Classification System For The Busy Dental Professional – Part II

October 8, 2019

by Amanda B. Longo, BSc, MSc, PhD; Peter C. Fritz, BSc, DDS, FRCD(C), PhD (Perio), MB

In 2017, the world of periodontology was redefined with significant updates to the classification system for periodontal and peri-implant disease. The work of more than 170 leading clinicians, scientists, and educators from around the globe culminated in the publication of 17 articles and four consensus statements summarizing a contemporary, evidence-based and clinically relevant system. This system is modelled after others used in medicine and stratifies the severity, rate of progression, and the extent of periodontal disease and helps to clarify clinical approaches for treatment. Since its unveiling in 2017, this comprehensive classification system has become the new standard of clinical practice around the world. It encourages clinicians to view the patient through a systemic lens, connecting and reinforcing the link between oral health and overall health. An executive summary of the updates to the new global classification system for periodontal disease have been shared in an earlier version of this publication.¹

The previous periodontal classification system was published in 1999, at a time when dental implants had only been in North America for approximately 20 years. Since then, dental implants have become an increasingly popular treatment option for the replacement of missing teeth. It soon became apparent that much like natural teeth, the supporting structures of dental implants can experience disease and therefore require specialized considerations for diagnosis of a healthy versus diseased state. It is obvious today that there can be no implantology without periodontology and the periodontal classification system from 1999 did not capture this relationship. The new system elegantly defines peri-implant diseases and conditions in great detail. The aim of this executive summary is to highlight the principal concepts and key updates in regard to peri-implant health and disease culminating from the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions.

Peri-Implant Health, Mucositis, and Implantitis

As previously defined, clinical periodontal health is the ideal oral condition defined as a state free from inflammation or infection that allows an individual to function normally.² A state of clinical periodontal health can be achieved on an intact or reduced periodontium and relies heavily on optimal control of both local and systemic factors contributing to inflammation.

Clinical peri-implant health shares many clinical features with periodontal health.³ Of course, anatomical and histological differences exist, primarily in the absence of a periodontal ligament and cementum around a dental implant. The hard and soft tissues surrounding an implant carry out two features similar to those supporting a natural tooth. The hard tissues form a secure relationship between the bone and the implant supporting ongoing function. The soft tissues, termed peri-implant mucosa, form a protective seal around the osseointegrated implant. After implant surgery, a well-established pattern of wound healing occurs to form the peri-implant mucosal seal. Some deficiencies in soft and hard tissues can occur prior to implant placement which may be linked to a poor outcome. Soft tissue deficiencies due to tooth loss, periodontal disease, or even some systemic disease do not allow tension-free, primary closure at the site of implant placement, potentially compromising peri-implant health. Tooth loss, periodontal disease, some systemic diseases, as well as endodontic

infection, root fracture, bone volume and height, and general trauma (from extraction or other) may also cause deficiencies in hard tissues prior to implant placement. These soft and hard tissue deficiencies may compromise peri-implant health even prior to implant placement.⁴

In the absence of tissue deficiencies, peri-implant health can be defined by the absence of erythema, bleeding on probing, swelling, and suppuration. In a model of health, there are no visual signs of inflammation. A periodontal probe is an essential tool in determining a diagnosis of peri-implant health. However, unlike in the diagnosis of periodontal disease around natural teeth, no exact defining threshold for probing depth can be set for peri-implant health. The clinical diagnostic considerations for health and disease are outlined in **Table 1**.

Table 1

Baseline Clinical Parameters	<ul style="list-style-type: none"> • Radiographic measurement • Probing measurement
Peri-Implant Health	<ul style="list-style-type: none"> • Absence of clinical signs of inflammation • Absence of bleeding and/or suppuration on gentle probing • No increase in probing depth compared to previous examinations • Absence of bone loss beyond crestal bone level changes due to initial bone remodelling following implant placement
Peri-Implant Mucositis	<ul style="list-style-type: none"> • Presence of clinical signs of inflammation • Presence of bleeding and/or suppuration on gentle probing with or without increased probing depth compared to previous examinations • Absence of bone loss beyond crestal bone level changes due to initial bone remodelling
Peri-Implantitis:	<ul style="list-style-type: none"> • Presence of clinical signs of inflammation • Presence of bleeding and/or suppuration on gentle probing • Increased probing depth compared to previous examination • Presence of bone loss beyond crestal bone level changes due to initial bone remodelling
Peri-Implantitis In the Absence of Baseline Clinical Parameters	<ul style="list-style-type: none"> • Presence of clinical signs of inflammation • Presence of bleeding and/or suppuration on gentle probing • Probing depths of $\geq 6\text{mm}$ • Bone levels of $\geq 3\text{mm}$ apical of the most coronal portion of the intraosseous part of the implant

Clinical Diagnostic Criteria for Peri-Implant Health, Peri-Implant Mucositis, and Peri-Implantitis

At implant sites, probing depths are often greater than at natural tooth sites. The peri-implant mucosal seal around implants often exhibits less resistance to probing than the gingiva at neighbouring teeth due to a lack of cementum on the implant surfaces and the orientation of the newly formed collagen fibres supporting the soft tissue seal.^{5,6} However, the height of the soft tissue around the implant following initial healing and the formation of the mucosal seal influences the initial probing depth and should always be documented as a means for future comparison.⁷ All things considered, a probing depth up to 5 mm may be associated with peri-implant health⁷ however, there are exceptions.

Any deviation from peri-implant health is a cause for concern and requires immediate identification and action by the clinician. Peri-implant mucositis develops following the accumulation of a bacterial biofilm around dental implants. The accumulation of bacteria results in the development of an inflammatory response of the peri-implant mucosa without the loss of supporting peri-implant bone.⁸ Just as in bio-film induced gingivitis, there is substantial variability in humans and their response to the quantity and type of bacterial biofilm present and the development of an inflammatory response around a dental implant.⁸ The presentation is however, the same. In routine clinical examinations, peri-implant mucositis presents with local swelling, redness, and shininess of the soft tissues. Patients may report a soreness that goes along with the visual signs of clinical inflammation. An increase in probing depth from a healthy reference is often observed due to swelling and/or a decrease in probing resistance secondary to inflammation. The main clinical characteristic of peri-implant mucositis is bleeding upon gentle probing. A line of bleeding or drop of bleeding should be used as an indication of a positive bleeding on probing finding. Important to note, a local dot of bleeding can occur following forceful probing of greater than 0.25 N (approximately 25 grams) of pressure and can be mistaken for a mucositis. Suppuration can occur in peri-implant mucositis, but it is rapidly destructive and soon induces bone loss turning the situation into a case of peri-implantitis.

With any amount of clinical inflammation at the site of an implant, radiographic evaluation should be conducted to assess bone levels around implants. Just as initial probing depths should be documented, an initial radiograph should be taken after implant placement to be used for all future

comparisons. Accounting for some crestal bone remodeling (0.5 – 2mm) within the first year after implant placement, radiographic bone loss should not exceed 2 mm.⁷

Similar to biofilm induced gingivitis at natural tooth sites⁹, peri-implant mucositis can be reversed with the reinstatement of effective biofilm (plaque) control. However, experimental peri-implant mucositis may take up to or longer than 21 days to be clinically reversed¹⁰, but full resolution and a return to peri-implant health is possible. An innovative and effective implant care protocol can be found in this publication. As a compliment to the identification and diagnosis of peri-implant disease, a clinically responsible method of prevention, management, and treatment should be followed.

If left untreated, peri-implantitis is assumed to progress following peri-implant mucositis, although the mechanism by which this occurs has not yet been identified.¹¹ The term “peri-implantitis” is slightly misleading given the implant is made of metal and cannot be inflamed. It is of course the surrounding tissues of the implant that experiences the inflammation. Peri-implantitis is characterized by the inflammation of the peri-implant mucosa, similar to peri-implant mucositis, along with the progressive loss of the supporting bone structure.^{12,13} This pathological condition is detected in the soft tissues by probing (bleeding on probing, increase in probing depth, suppuration) and in the hard tissues by radiographic analysis. Clinical studies have reported on the configuration of peri-implantitis bone defects. The majority of peri-implantitis affected implants exhibited uniform bone loss at all four aspects, suggesting that peri-implantitis lesions commonly progress in a circumferential pattern.¹⁴⁻¹⁶

Several studies have sought out to map the factors triggering the onset and progression of peri-implantitis. While the cause of onset remains elusive, the progression of bone loss follows a non-linear, accelerating pattern.¹⁷ Research supports an early onset of peri-implantitis (≤ 2 years) in some cases.¹⁸⁻²⁰ This pattern is not homogenous, likely due to the high variability in the individual response to plaque and bacterial biofilm accumulation, to the complex and heterogeneous nature of the bacterial infection itself, and to other environmental risk factors associated with the onset and progression of peri-implant diseases.

Risk Indicators

Observational, cross-sectional and retrospective studies have been conducted to evaluate potential risk factors associated with peri-implant health and disease. A varying body of evidence exists for each risk factor and therefore, more research that is interventional (while ethical in design) is required to identify true risk factors with a high level of certainty.

The strongest evidence exists around a history of periodontitis as a risk factor for peri-implantitis.²¹⁻³⁶ These findings support the need for compliant and regular supportive periodontal therapy^{26,27,37} and proper oral hygiene^{26,36,38}. This strongly supports the notion that implantology should never be considered without supportive periodontal monitoring and assessment. Responsible clinicians must always ensure completion of a full periodontal examination and compliance with an oral hygiene program in all of their patients before their consideration as a candidate for a dental implant.

Another factor within the clinician’s control that is a risk factor in both the onset and progression of peri-implant mucositis and implantitis are iatrogenic factors such as poor surgical planning and execution, inadequate restoration, lack of full seating of the prostheses, and over-contouring of restorations. Similarly, the design and positioning of the implant supported prostheses should be easy to clean by the patient to reduce the likelihood of bacterial accumulation and the formation of biofilm.

Just as deficiencies to the hard and soft tissues prior to implant placement can contribute to future complications, tissue⁴ and restorative deficiencies can also impact the health of the implant post-surgical placement.⁴ Hard and soft tissue deficiencies can generally be categorized as either being associated with healthy or diseased situations. As an example, defects to the alveolar process and anatomical anomalies will influence the placement of an implant which may result in it being malpositioned, improperly loaded, with a lack of buccal bone and a dearth of keratinized tissue. These factors can increase the likelihood of developing peri-implant disease as well as aesthetic complications. Systemic, inflammatory, genetic or acquired disease may also influence the quality of the hard and soft tissue following implant placement, ultimately influencing the long-term health of

the implant. Further supporting research is required to substantiate each of these individual deficiencies and their relationship to implant health. In a clinical setting, they should always be integral when treatment planning.

Currently, there is not a large enough body of supporting evidence in the literature for a strong causal relationship between either diabetes mellitus or smoking status with peri-implant diseases. However, with the known risk that both uncontrolled blood glucose levels and current smoking status pose on periodontal disease, caution should be taken when treating patients with either such condition. It is clear, that there is reduced periodontal healing ability in both smokers and poorly controlled diabetics.

Although occlusal factors, such as overloading or non-axial loading, are suspect as risk factors, to date there is no consensus or direct evidence that these can initiate, sustain or lead to progression of peri-implant disease. One possible scenario is that occlusal factors can lead to initial bone loss due to biomechanically-induced bone remodeling without inflammation (mucositis) creating the environment for inflammation secondary to biofilm accumulation.

Similarly, other areas for future research as potential risk factors in the development of peri-implant diseases include the quantity of keratinized mucosa surrounding the implant, the presence of excess cementum or titanium particles. Further research into genetic polymorphisms and other diseases with a known systemic link such as cardiovascular disease, immune function disorders, osteoporosis, obesity, and hepatitis is also warranted.

Measurement Of Implant Related Complications

With the diagnostic considerations for peri-implant related diseases now clearly defined, a standardized classification and reporting system should also be established for reporting the occurrence of these negative outcomes. The Fonthill Dental Complication Classification Scale, proposes such a scale that can be used in daily dental practice for the measurement of complications. This scale consists of seven classes, each increasing in severity and based on the time and the dental personnel required to fully resolve the complication arising from initial dental treatment.³⁹ The complication classes are outlined in **Table 2** along with examples of implant-related complications that can arise in any practice. Specifically, the Class IV complication rate for dental implant placement can be calculated and is directly reflective of the implant failure rate. While its value may encompass some implant failures that are not directly due to biofilm-induced peri-implantitis, such as occlusal overload, the majority of cases will be as a result of non-reversible peri-implantitis.

Table 2

Class	Definition	Example
1	Any deviation from the normal postoperative course without the need for physical intervention. Full resolution can be obtained over the phone/ email/text by dental personnel or by the dental surgeon.	Patient requests additional information regarding the timeline of treatment or instructions regarding prescriptions.
2	Any deviation from the normal postoperative course without the need for intervention by the dental surgeon. The patient may need to return to the clinic but does not need to see the dental surgeon for full resolution of the complication.	Staining from chlorhexidine rinse.
3	Any deviation from the normal postoperative course requiring surgical, dental, or prosthetic intervention by the dental surgeon. The patient and dental surgeon need to meet for exactly one appointment for full resolution of the complication.	Loose/lost healing abutment
4	Any deviation from the normal postoperative course requiring surgical, dental, or prosthetic intervention by the dental surgeon. The patient and dental surgeon need to meet more than once for full resolution of the complication.	Peri-implantitis
5	Any deviation from the normal postoperative course requiring surgical, dental, or prosthetic intervention by the dental surgeon and a third-party dental specialist. Full resolution of the complication requires one or more appointments with the dental surgeon and with the third-party dental specialist.	Damage to adjacent tooth during implant placement requiring endodontic management.
6	Any deviation from the normal postoperative course requiring intervention outside of the scope of dentistry (i.e., requiring medical intervention).	Aspiration of a healing abutment during regularly scheduled dental surgery requiring medical intervention for removal.
7	Death	

The Fonthill Dental Complication Classification Scale with Implant-Related Examples
Adapted from Fritz and Longo (2019) Clin Exp Dent Res. 1-6.
<https://doi.org/10.1002/cre2.23>

Conclusions

The updates and consensus from the new global classification system present a framework to define and to classify oral health and disease around teeth and dental implants. Attention has been paid to the differences that exist between natural and artificial root structures and the individual response to the presence of bacterial biofilm. This important and comprehensive update provides a framework for clinicians to increase transparency and to create a common language when discussing complications observed in implantology, ultimately progressing the field of dentistry and allowing us to better care for our patients.

Oral Health welcomes this original article.

References

1. Fritz PC, Ward WE, Longo AB. (2018) The New Global Classification System for Periodontal and Peri-Implant Diseases: An Executive Summary for the Busy Dental Professional. *Oral Health*.
2. Lang NP, Bartold PM. (2018) Periodontal health. *J Clin Periodontol*. 45(Suppl 20): S9-S16.
3. Araujo MG, Lindhe J. (2018) Peri-implant health. *J Clin Periodontol*. 45(Suppl 20):S230-S236.
4. Hammerle CHF, Tarnow D. (2018) The etiology of hard- and soft-tissue deficiencies at dental implants: A narrative review. *J Clin Periodontol*. 45(Suppl 20): S267-S277.
5. Lang NP, Wetzel AC, Stich H, Caffesse RG. (1994) Histological probe penetration in healthy and inflamed tissues. *Clin Oral Implants Res*. 5: 191-201.
6. Abrahamsson I, Soldini C. (2006) Probe penetration in periodontal and peri-implant tissues. An experimental study in the beagle dog. *Clin Oral Implants Res*. 17: 601-605.
7. Renvert S, Persson GR, Pirih FQ, Camargo PM. (2018) Peri-implant health, peri-implant mucositis, and peri-implantitis: Case definitions and diagnostic considerations. *J Clin Periodontol*. 45(Suppl 20): S278-S285.
8. Heitz-Mayfield LJA, Salvi GE. (2018) Peri-implant mucositis. *J Clin Periodontol*. 45(Suppl 20): S237-S245.
9. Loe H, Theilade E, Jensen SB. (1965) Experimental gingivitis in man. *J Periodontol*. 36: 177-187.
10. Salvi GE, Aglietta M, Eick S, Sculean A, Lang NO, Ramseier CA. (2012) Reversibility of experimental peri-implant mucositis compared with experimental gingivitis in humans. *Clin Oral Implants Res*. 23: 182-190.
11. Schwarz F, Derks J, Monje A, Wang HL. (2018) Peri-implantitis. *J Clin Periodontol*. 45(Suppl 20): S246-S266.
12. Lang NP, Berglundh T. (2011) Working Group 4 of Seventh European Workshop on P. Periimplant diseases: Where are we now?-Consensus of the Seventh European Workshop on Periodontology. *J Clin Periodontol*. 38(Suppl 11): 178-181.
13. Lindhe J, Meyle J, Group DoEWOP. (2008) Peri-implant diseases: consensus report of the Sixth European Workshop on Periodontology. *J Clin Periodontol*. 35(Suppl 8): 282-285.
14. Serino G, Turri A, Lang NP. (2013) Probing at implants with peri-implantitis and its relation to clinical peri-implant bone loss. *Clin Oral Implants Res*. 24: 91-95.
15. Schwarz F, Herten M, Sager M, Bieling K, Sculean A, Becker J. (2007) Comparison of naturally occurring and ligature-induced peri-implantitis bone defects in humans and dogs. *Clin Oral Implants Res*. 18: 161-170.
16. Garcia-Garcia M, Mir-Mari J, Benic F, Figueiredo R, Valmaseda-Castellon E. (2016) Accuracy of periapical radiography in assessing bone level in implants affected by peri-implantitis: a cross-sectional study. *J Clin Periodontol*. 43: 85-91.
17. Fransson C, Tomasi C, Pikner SS, et al. (2010) Severity and pattern of peri-implantitis associated bone loss. *J Clin Periodontol*. 37: 442-448.
18. Derks J, Schaller D, Hakansson J, Wennstrom JL, Tomasi C, Berglundh T. (2016) Peri-implantitis – onset and pattern of progression. *J Clin Periodontol*. 43: 383-388.
19. Schwarz F, Becker K, Sahm N, Horstkemper T, Rousi K, Becker J. (2017) The prevalence of peri-implant diseases for two-piece implants with an internal tube-in-tube connection: a cross-sectional analysis of 512 implants. *Clin Oral Implants Res*. 28: 24-28.
20. Becker J, John G, Becker K, Mainusch S, Diedrichs G, Schwarz F. (2017) Clinical performance of two-piece zirconia implants in the posterior mandible and maxilla: a prospective cohort study over 2 years. *Clin Oral Implants Res*. 28: 29-35.
21. Costa FO, Takenaka-Martinez S, Cota LO, Ferreira SD, Silva GL, Costa JE. (2012) Peri-implant disease in subjects with and without preventive maintenance: a 5-year follow-up. *J Clin Periodontol*. 39: 173-181.
22. Derks J, Schaller D, Hakansson J, Wennstrom JL, Tomasi Cm Berglundh T. (2016) Effectiveness of implant therapy analyzed in a Swedish population: prevalence of peri-implantitis. *J Dent Res*. 95: 43-49.
23. Karoussis IK, Salvi GE, Heitz-Mayfield LJ, Bragger U, Hammerle CH, Lang NP. (2003) Long-term implant prognosis in patients with and without a history of chronic periodontitis: a 10-year prospective cohort study of the ITI Dental Implant System. *Clin Oral Implants Res*. 14: 329-339.

24. Rocuzzo M, Bonino F, Aglietta M, Dalmasso P. (2012) Ten-year results of a three arms prospective cohort study on implants in periodontally compromised patients, Part 2: clinical results. *Clin Oral Implants Res.* 23: 389-395
25. Rocuzzo M, De Angelis N, Bonino L, Aglietta M. (2010) Ten-year results of a three-arm prospective cohort study on implants in periodontally compromised patients. Part 1: implant loss and radiographic bone loss. *Clin Oral Implants Res.* 21: 490-496.
26. Roos-Jansaker AM, Lindahl C, Renvert H, Renvert S. (2006) Nine- to fourteen-year follow-up of implant treatment, Part II: presence of peri-implant lesions. *J Clin Periodontol.* 33: 290-295.
27. Roos-Jansaker AM, Renvert H, Lindahl C, Renvert S. Nine- to fourteen-year follow-up of implant treatment. Part III: factors associated with peri-implant lesions. *J Clin Periodontol.* 33: 296-301.
28. Koldslund OC, Scheie AA, Aass AM. (2010) Prevalence of peri-implantitis related to severity of the disease with different degrees of bone loss. *J Periodontol.* 81: 231-238.
29. Koldslund OC, Scheie AA, Aass AM. (2011) The association between selected risk indicators and severity of peri-implantitis using mixed model analyses. *J Clin Periodontol.* 28: 285-292.
30. Casado PL, Pereira MC, Duarte ME, Granjeiro JM. (2013) History of chronic periodontitis is a high risk indicator for peri-implant disease. *Braz Dent J.* 24: 136-141.
31. de Araujo Nobre M, Mano Azul A, Rocha E, Malo P. (2015). Risk factors of peri-implant pathology. *Eur J Oral Sci.* 123: 131-139.
32. Renvert S, Aghazadeh A, Hallstrom H, Persson GR. (2014) Factors related to peri-implantitis – a retrospective study. *Clin Oral Implants Res.* 25: 522-529.
33. Dalago HR, Schuldt Filho G, Rodrigues MA, Renvert S, Bianchini MA. (2017) Risk indicators for peri-implantitis. A cross-sectional study with 916 implants. 28: 144-150.
34. Maximo MB, de Mendonca AC, Alves JF, Cortelli SC, Peruzzo DC, Duarte PM. (2008) Peri-implant diseases may be associated with increased time loading and generalized periodontal bone loss: preliminary results. *J Oral Implantol.* 34: 268-273.
35. Daubert DM, Weinstein BF, Bordin S, Lerouz BG, Flemming TF. (2015) Prevalence and predictive factors for peri-implant disease and implant failure: a cross-sectional analysis. 86: 337-347.
36. Ferreira SD, Silva GL, Cortelli JR, Costa JE, Costa FO. (2006). Prevalence and risk variables for peri-implant disease in Brazilian subjects. *J Clin Periodontol.* 33: 929-935.
37. Roos-Jansaker AM, Lindahl C, Renvert H, Renvert S. (2006) Nine- to fourteen-year follow-up of implant treatment. Part I: implant loss and associations to various factors. *J Clin Periodontol.* 33: 283-289.
38. Konstantinidis IK, Kotsakis GA, Gerdes S, Walter MH. (2015) Cross-sectional study on the prevalence and risk indicators of peri-implant diseases. *Eur J Oral Implantol.* 8: 75-88.
39. Fritz PC, Longo AB. (2019) The Fonthill Dental Surgery Complication Classification Scale. *Clin Exp Dent Res.* 1-6.

About The Author



Dr. Amanda B. Longo is the Chief Innovation Officer and Director of Strategy of a private periodontal surgery clinic in Fonthill, ON. She is internationally recognized through her influence in research, innovation and knowledge mobilization in the fields of periodontology, CBCT technologies, nutrition, and employee engagement.



Dr. Fritz is a full-time periodontist in Fonthill, ON. He leads an extraordinary, collaborative, empowered team of clinicians, makers, scientists and artists who are all working together to innovate the dental specialty of periodontics and redefine the patient experience. He has recently created the Implant Care Practitioner Program for dental professionals across the globe.

RELATED ARTICLE: Peri-implantitis

Follow the Oral Health Group on Facebook, Instagram, Twitter and LinkedIn for the latest updates on news, clinical articles, practice management and more!