

Episode #46 – Classification of Peri-implant Diseases and Conditions
October 8, 2021

Overview
The 2017 World Workshop on the classification of periodontal and peri-implant diseases and conditions was jointly conducted by the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP). The result from the workshop was a revised disease classification system to guide clinician diagnosis and comprehensive treatment planning, while allowing for personalized client care. The revised system is also important for researchers to investigate etiology, pathogenesis, natural history, and treatment of periodontal and peri-implant diseases and conditions. The updated classification included the staging and grading system for periodontitis classification, a regrouping of the various forms of periodontitis, and the first classification for peri-implant diseases[1] and conditions. [1] [2]

There are 4 classes for peri-implant diseases and conditions:
- Peri-implant health
- Peri-implant mucositis
- Peri-implantitis
- Peri-implant soft and hard tissue deficiencies [2]

Peri-implant health
Understanding the characteristics of healthy peri-implant tissues is important to enable disease recognition. Peri-implant tissues are the hard and soft tissue components surrounding osseointegrated dental implants.[2] The hard tissue component (i.e., bone)[3] forms the secure contact relationship with the implant surface supporting the implant's function. The soft tissue component, termed peri-implant mucosa, forms the protective seal around the osseointegrated implant. Peri-implant health can be defined by both histologic and clinical characteristics. [3]

In clinical health, the peri-implant mucosa forms a tight seal around the transmucosal component of the implant (i.e., the abutment and/or restoration). The height of the soft tissue surrounding the implant influences probing depths.[4] Generally, however, the probing depth associated with peri-implant health is approximately ≤5 mm. [3] [4]

[1] Refer to Episode #24 for information on laser therapy and peri-implant diseases.
[2] Refer to Episode #18 for information on ceramic versus titanium implants.
[3] Refer to Episode #42 for information on dental implants and osteoporosis.
[4] Refer to Episode #12 for information on probing peri-implant tissues.

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The definition of peri-implant health is based on the absence of peri-implant signs of soft tissue inflammation (i.e., erythema, edema, profuse bleeding on probing, suppuration), and the absence of further additional bone loss following initial healing. Note: peri-implant tissue health can exist following treatment of peri-implantitis with variable levels of bone support (e.g., reduced bone support). [4] [5]

There are no visual differences between healthy peri-implant and periodontal tissues. However, probing depths are usually greater around dental implants versus teeth. Also, papillae between dental implants may be shorter than papillae between teeth. [5]

The main histological differences between healthy peri-implant and periodontal tissues are:

- Peri-implant tissues do not have cementum and periodontal ligament.
- Peri-implant epithelium is often longer and there are no inserting fibres into the implant surface.
- Peri-implant tissues are less vascularized in the area between the bone crest and the junctional epithelium compared to the connective tissue of the periodontium. [5]

The soft tissue collar around dental implants exhibits less resistance to probing than the gingiva surrounding teeth. This aspect of the implant mucosal seal may lead to mechanically induced bleeding on probing on dental implants that are clinically healthy. Thus, the presence of a local dot of bleeding may represent trauma rather than a sign of biofilm-induced inflammation. Trauma-induced bleeding on probing may be a result of excessive probing forces as well as difficulties in aiming the probe into the dental implant sulcus because of implant-restoration contours. [4]

Comparison between teeth and dental implants [6] [7]

	Teeth	**Dental implants**
Connection	Cementum, bone, periodontal ligament	Osseointegration, functional ankylosis (periodontal ligament and cementum absent)
Junctional epithelium	Hemidesmosomes and basal lamina[5]	Hemidesmosomes and basal lamina
Connective tissue	Perpendicular fibres to the long axis of the tooth	Circumferential parallel fibres to the long axis of the implant
Vascularity	More	Less
Probing depths	≤3 mm in health	≤5 mm (dependent on soft tissue depth)
Bleeding on probing for disease detection	More reliable	Less reliable
Hard tissue interface	Resilient connection, tooth mobility up to ~0.2 mm regarded as physiological	Rigid connection, no mobility

[5] Hemidesmosomes are multiprotein complexes that facilitate the adhesion of basal epithelial cells to the underlying basal lamina of the basement membrane, which is, in turn, attached to the underlying connective tissue. [14]

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Assessing peri-implant health
Assessing for the presence or absence of inflammation of an implant site involves visual inspection, peri-implant probing, digital palpation, and discussion with the client to determine if they have noted changes (e.g., discomfort, loose components). Periodontal measurements are necessary to assess for bleeding and/or suppuration on probing and to monitor for changes in probing depths and mucosal margin height. [4]

Assessing for peri-implant health includes:

- Evaluating the soft tissue around dental implants, including general oral hygiene with specific focus on the presence of biofilm on dental implants and their restorations;
- Probing dental implants with a light force (0.25 N or 25 grams of pressure) to avoid trauma. Peri-implant pocket depths should generally be ≤5 mm. Dental implants should be probed routinely and periodically (e.g., at least once per year);
- Observing for bleeding on probing. It is difficult to distinguish between biofilm-induced peri-implant inflammation and mechanically-induced trauma. Therefore, bleeding "dots" should be interpreted carefully because this bleeding may be a result of trauma and not due to tissue inflammation;
- Observing for suppuration on probing;
- Palpating peri-mucosal tissues for the presence of suppuration and/or discomfort;
- Evaluating for mobility and loose components; and
- Evaluating intraoral radiographs for implant bone loss beyond bone level changes from initial bone remodelling. Alveolar bone remodelling following the first year in function may depend on the type and position of the implant, but alveolar bone loss starting after the implant was placed in function should not exceed 2 mm. Bone loss ≥2 mm during or after the first year should be considered pathologic. An intraoral radiograph should be taken at baseline with the supra-structure[6] in place that clearly identifies the implant threads and mesial and distal bone levels. Radiographs should be taken based on clinical judgement after implant assessment. Subsequent radiographs are compared with baseline radiographs to assess for implant bone level changes. [4]

Case definition for peri-implant health
Peri-implant health requires:

- Absence of visual signs of inflammation (e.g., pink, firm, nonedematous tissue);
- Lack of profuse line or drop bleeding on probing;
- No increase in probing depths over time. Pocket depths could differ depending on the height of the soft tissue surrounding the dental implant. Hence, it is not possible to define a range of probing depths compatible with peri-implant health; and
- Absence of further bone loss after initial healing, which should not be ≥2 mm. [2] [4]

<u>Peri-implant mucositis</u>
Peri-implant mucositis is a disease characterized by inflammation of the soft tissues surrounding a dental implant, bleeding on gentle probing, and no signs of loss of

[6] Supra-structure is the portion of the dental implant that attaches to its implant substructure and to a fixed (e.g., a crown) or removable dental prosthesis. In other words, everything above the implant.

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supporting bone following initial bone remodelling during healing. Suppuration may be present. [5] [8]

The main etiology of peri-implant mucositis is bacterial biofilm accumulation around the dental implant. There is very limited evidence for non-plaque induced peri-implant mucositis. Peri-implant mucositis can be reversed with good biofilm control measures aimed at eliminating plaque. [2] [4] [5] [9]

Risk factors for peri-implant mucositis

- Biofilm accumulation – Research clearly demonstrated biofilm accumulation was associated with peri-implant mucositis. Also, biofilm accumulation was a risk indicator in a dose-dependent manner. Experimental peri-implant mucositis studies have shown it takes more than 3 weeks for complete resolution of clinical signs of inflammation with reinstitution of biofilm control. Thus, mechanical biofilm control is considered the standard of care for prevention and management of peri-implant mucositis administered by the client and the oral healthcare professional. [5] [9] [10]
- Lack of compliance with regular preventive maintenance therapy – Studies have demonstrated peri-implant mucositis was a common finding among individuals not adhering to regular preventive maintenance therapy.[7] Furthermore, complying with regular preventive maintenance therapy significantly reduced the prevalence of peri-implantitis. Therefore, regular preventive maintenance therapy with biofilm removal is an important strategy to prevent peri-implant mucositis and the progression of peri-implant mucositis to peri-implantitis. [9]
- Design of implant-supported prostheses – Implant-supported prostheses that provide accessible biofilm removal is important in the prevention and management of peri-implant diseases. For instance, a high proportion of implants diagnosed with peri-implantitis were associated with poor biofilm control and lack of accessibility for oral hygiene measures, while cleansable protheses and good plaque control was rarely associated with peri-implantitis. Thus, individualized self-care instruction is important to prevent peri-implant mucositis and peri-implantitis. Additionally, when possible, margins of implant-supported prostheses should be placed at or above the peri-implant mucosal margin to facilitate biofilm removable. Implant-supported prostheses impairing biofilm removal should ideally be adjusted or replaced with cleansable prostheses. [9]
- Dimensions of keratinized peri-implant mucosa – Evidence for the presence or minimum width of keratinized mucosa around implants to maintain soft tissue health and stability remains controversial. For instance, some studies reported higher rates of peri-implant mucositis with implants lacking or surrounded by an inadequate width (<2 mm) of keratinized mucosa. While other studies found no association or a positive association. In clinical situations where self-performed biofilm control is adequate around dental implants, presence or grafting of keratinized mucosa to maintain peri-implant health does not appear to be critical. [9]
- Excess cement – Research has demonstrated excess cement was associated with clinical signs of peri-implant mucositis. Thus, to avoid excess cement, restoration

[7] Refer to Episode #35 for information on air polishing and dental implants.

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margins should be located at or above the peri-implant mucosal margin to allow for proper cement removal. [9]
- Factors affecting host susceptibility to biofilm-induced peri-implant mucositis – Factors increasing host susceptibility may include smoking, radiation therapy, and poorly controlled diabetes. [9]

Assessing for peri-implant mucositis
Assessing for peri-implant mucositis is based on clinical signs of inflammation and absence of radiographic bone level changes beyond initial bone remodelling. [4]

Similar to assessing for peri-implant health, assessing for peri-implant mucositis includes:
- Evaluating for signs of clinical inflammation (e.g., localized edema, erythema, and shininess of the soft tissue surface). Having a discussion with the client. For example, soreness is a common symptom reported by clients;
- Gentle probing observing for bleeding on probing and increasing probing depths. A local dot of bleeding from probing may be the result of traumatic probing in the absence of visually inflamed tissues. Bleeding on probing (e.g., a line of bleeding or drop of bleeding within 30 seconds following probing) that is combined with visually inflamed tissues at the probing site is a sign of an inflammatory peri-implant soft tissue lesion. [5] An increase in probing depth is often observed due to edema or decrease in probing resistance;
- Observing for suppuration upon digital palpation of the tissues or following probing; and
- Radiographically evaluating the dental implant if clinical signs of inflammation are present. This would involve comparing current intraoral implant radiograph with baseline implant radiograph (with the supra-structure in place). Initial bone remodelling after the dental implant is placed in function should not exceed 2 mm. Crestal bone loss beyond the initial remodelling suggests either progressive peri-implant infection or other etiologic factors (e.g., excess cement, loose implant components, fractured implant components). [4]

Case definition for peri-implant mucositis
Peri-implant mucositis consists of:
- The presence of signs of inflammation (e.g., erythemic, edematous tissues);
- Presence of profuse line or drop bleeding on probing and/or suppuration on probing or with digital palpitation of peri-implant tissues;
- An increase in probing depths compared to baseline; and
- Absence of bone loss beyond crestal bone level changes from initial remodelling. [4]

Peri-implantitis
Peri-implantitis is a plaque-associated pathologic condition of the tissue surrounding a dental implant. It is characterized by inflammation of the peri-implant mucosa, bleeding on probing and/or suppuration, increased probing depths and/or recession of the mucosal margin, and subsequent progressive loss of the supporting bone. Bone loss is frequently circumferential. Histologically, peri-implantitis sites often have larger inflammatory lesions compared to periodontitis sites.

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It is assumed peri-implant mucositis precedes peri-implantitis. However, the histopathologic and clinical conditions leading to the transition from peri-implant mucositis to peri-implantitis are not completely understood. Peri-implantitis is associated with inadequate plaque control and a history of severe periodontitis. The onset of peri-implantitis may occur early following dental implant placement as indicated by research using clinical and radiographic data. Data also suggest the progression of peri-implantitis appears to be faster than that observed in periodontitis. Without treatment, peri-implantitis appears to progress in a non-linear and accelerating pattern. Studies on peri-implantitis treatment demonstrate anti-infective treatment strategies are effective in decreasing soft tissue inflammation and in suppressing disease progression. [2] [5] [11]

Risk factors for peri-implantitis

- History of periodontitis – Research has demonstrated strong evidence that a history of periodontitis is a risk factor for peri-implantitis. Specifically, longitudinal and cross-sectional studies have shown individuals with periodontitis have significantly higher odds of developing peri-implantitis compared to individuals without periodontitis. [11]
- Smoking – Several studies have reported an association between smoking and peri-implantitis. However, the majority of studies failed to identify smoking as a risk factor for peri-implantitis. The reasons for the conflicting findings and the weak association between smoking and peri-implantitis are currently not understood but may be related to the differences in classifying smokers and non-smokers in the research. Also, the studies relied on self-reported smoking status. Thus, it is inconclusive whether smoking is a risk factor for peri-implantitis. [11]
- Diabetes – Several studies have indicated individuals with diabetes are at higher risk for peri-implantitis. Conversely, a number of studies failed to identify diabetes as a risk for peri-implantitis. Hence, the available evidence is inconclusive as to whether diabetes is a risk factor for peri-implantitis. [11]
- Poor plaque control and lack of regular preventive maintenance therapy – Research has provided evidence that poor plaque control and lack of regular preventive maintenance therapy are risk factors for peri-implantitis. [11]
- Keratinized mucosa – Evidence for the need of keratinized mucosa to maintain peri-implant health is still limited. Some studies have indicated that a keratinized mucosa of <2 mm was associated with greater plaque accumulation and peri-implant soft tissue inflammation compared with dental implants surrounded by a keratinized margin of ≥2 mm. Studies suggest a reduced width of keratinized mucosa or its absence may negatively affect oral self-care measures (e.g., increased client discomfort). However, there is limited evidence that this factor represents a risk for peri-implantitis and further research is required. [11]
- Excess cement – Several studies have reported an association between excess cement and the prevalence of peri-implant diseases. This may be a result of the rough surface created by cement remnants facilitating biofilm formation and retention. However, the proportion of diseased implant sites with excess cement varied between 9% and 81% and several implant sites with excess cement exhibited no disease. Research has also indicated cement-retained restorations were not at a higher risk for peri-implantitis compared to screw-retained restorations. Therefore, it is suggested excess cement is a potential risk factor for peri-implantitis. [11]

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- Genetic factors – Gene polymorphisms[8] may affect traits such as gene expression, protein production, and cytokine secretion. Several observational studies have investigated the potential association between various gene polymorphisms and the occurrence of peri-implantitis, with the majority focusing on the cytokine IL-1.[9] The available evidence indicates a potential influence of various gene polymorphisms in the pathogenesis of peri-implantitis. However, more research is required, especially prospective clinical studies and studies with sufficient sample sizes. [11]
- Systemic conditions – The association of systemic conditions (other than diabetes) with peri-implantitis has rarely been studied and is therefore unclear. Thus, the evidence suggesting systemic conditions (other than diabetes) to be a risk factor for peri-implantitis is limited and further research is required. [11]
- Iatrogenic factors – The Consensus report of the 7th European Workshop on Periodontology (2011) acknowledged that peri-implantitis onset and progression may be initiated by iatrogenic factors, such as inadequate restoration-abutment seating, overcontouring of restorations, and implant-malpositioning. [12] However, studies examining the role of iatrogenic factors in the development of peri-implant diseases are still scarce. Thus, due to insufficient data, it appears that implant position and design of the supra-structure may influence access for self-performed and professionally administered biofilm removal. [11]
- Occlusal overload – A small number of animal and human studies assessed occlusal overload. Currently, there is limited evidence that occlusal overload constitutes a risk factor for peri-implantitis onset or progression. [11]
- Titanium particles – A small number of studies have identified metal particles around healthy and diseased dental implant sites. However, the available evidence does not allow for the evaluation of the role of titanium or metal particles in the pathogenesis of peri-implant diseases. Further research is required. [11]
- Additional factors – A few case reports have associated factors such as bone compression necrosis, overheating, micromotion, and biocorrosion with peri-implantitis. However, further research is necessary to evaluate such factors. [11]

Periapical peri-implantitis

Several case reports have described the occurrence of periapical peri-implantitis lesions. The affected dental implants were generally characterized by a periapical radiographic radiolucency with or without clinical signs of inflammation (e.g., erythema, edema, fistula and/ or abscess formation). These radiographic and clinical signs of pathology were noted between 2 to 8 weeks and up to 4 years after dental implant placement. Most of the studies reported a direct association between retrograde peri-implantitis[10] and existing periapical endodontic lesions at adjacent teeth. [11]

Assessing for peri-implantitis

Assessing for peri-implantitis is based on clinical signs of inflammation and radiographic bone loss beyond initial bone remodelling. The difference between peri-implant

[8] Gene polymorphism is the presence of a gene with multiple variations that can affect various traits. [15]
[9] Refer to Episodes #44 and #45 for information on cytokines.
[10] Retrograde peri-Implantitis is a clinically symptomatic periapical lesion diagnosed by a radiolucency that develops shortly after implant insertion and where the coronal portion of the implant achieves a normal bone-implant interface. [16]

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mucositis and peri-implantitis is bone loss following initial healing and bone remodelling and requires radiographic evaluation of the bone level.

Similar to assessing for peri-implant mucositis, assessing for peri-implantitis includes:

- Evaluating for signs and symptoms of inflammation (e.g., erythema, edema, pain);
- Gentle probing assessing for bleeding on probing and increasing probing depths. A local dot of bleeding from probing may be the result of traumatic probing in the absence of visually inflamed tissues. Bleeding on probing (e.g., a line of bleeding or drop bleeding) that is combined with visually inflamed tissues at the probing site is a sign of an inflammatory peri-implant soft tissue lesion;
- Observing for suppuration upon digital palpation of the tissues or following probing; and
- Radiographically evaluating the dental implant if clinical signs of inflammation are present. This would involve comparing current intraoral implant radiograph with baseline implant radiograph (with the supra-structure in place). Initial bone remodelling after the implant is placed in function should not exceed 2 mm. Crestal bone loss beyond the initial remodelling suggests pathology. [4]

Case definitions for peri-implantitis

Peri-implantitis consists of:

- Visual inflammation of the peri-implant soft tissues combined with bleeding on probing and/or suppuration;
- Increasing probing pocket depths as compared to probing depths obtained at placement of the supra-structure; and
- Progressive bone loss based on radiographic bone level assessment at 1 year following the delivery of the implant-supported prosthetics reconstruction; and
- If initial radiographs and probing depths are unavailable, radiographic evidence of bone level ≥3 mm and/or probing depths ≥6 mm in combination with profuse bleeding represents peri-implantitis. [4]

Differential diagnosis for peri-implantitis

Several case reports have described various mucosal lesions around dental implants that may mimic peri-implant diseases, such as giant cell granuloma[11] and pyogenic granuloma[12] as well as primary malignant tumours (i.e., oral squamous cell carcinoma) or metastases. These pathologic conditions share several clinical features with peri-implant diseases. However, these lesions reveal distinct differences to nonspecific inflammation at the histopathologic level. [11]

<u>Hard and soft tissue implant site deficiencies</u>

The normal healing process after tooth loss leads to diminished dimensions of the alveolar process/ridge resulting in both hard and soft tissue deficiencies. Thus, hard and

[11] Giant cell granulomas are rare benign but aggressive osteolytic tumours that can affect the head and neck region, especially the jaw bones. [17]

[12] Pyogenic granulomas are benign, vascular tumours that can affect skin and mucous membranes. They usually appear as raised, bright red lesions that may grow quickly and bleed a lot. Factors associated with development include trauma, hormonal influence, or medications, but exact cause is unknown. [18] [19]

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soft tissue deficiencies at dental implants sites are common clinical findings. The presence of these deficiencies may lead to increased marginal bone loss, soft tissue inflammation, and soft tissue recession. These problems are difficult to treat and may compromise implant survival. Thus, therapeutic interventions may be required to correct hard and soft tissue deficiencies to improve clinical outcomes of dental implant therapy. [2] [13]

Hard and soft tissue deficiencies may result from various of etiological factors including:
- Systemic diseases and conditions;
- Systemic medications;
- Tissue healing processes;
- Tissue turnover and tissue response to clinical interventions;
- Trauma to orofacial structures;
- Local diseases affecting the teeth, periodontium, bone, and mucosa;
- Biomechanical factors;
- Tissue morphology and tissue phenotype; and
- Iatrogenic factors.

These etiologic factors may occur in isolation or in combination. When more than one etiologic factor occurs, the severity of the defect(s) may increase. [13]

Hard and soft tissue deficiencies can be categorized as deficiencies prior to implant placement and deficiencies after implant placement. [13]

Hard tissue deficiencies prior to implant placement
Hard tissue deficiencies prior to dental implant placement include conditions where the available amount of bone does not allow for a standard implant to be placed fully embedded in the bone.

Factors affecting hard tissue deficiencies prior to implant placement include:
- Tooth loss – Various amounts of alveolar bone loss occurs following tooth extraction due to resorption processes. Research has demonstrated edentulous alveolar ridges continuously reduce over time (e.g., under denture bases). [13]
- Trauma from tooth extraction – Trauma during tooth extraction may affect bone healing resulting in decreased amounts of bone volume in the extraction site area. However, clinical investigations reporting hard and soft tissue defects resulting from traumatic tooth extraction are scarce. [13]
- Periodontitis – Periodontitis progression results in the continuous resorption of bone adjacent to affected teeth. Lack of bone volume may prevent implant placement. [13]
- Endodontic infections – Endodontic infections can lead to resorption of periapical bone. This resorption may reach a degree where implant placement using standard procedures is impacted even if the marginal bone is intact. Further, depending on the degree of bone resorption, apical bone deficiencies may make implant placement more difficult or not possible. [13]
- Longitudinal root fractures – Longitudinal (vertical) root fractures may lead to bone resorption and thus cause hard tissue deficiencies at implants. However, data is very

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scarce assessing implant placement and the extent of bone destruction caused by vertical root fractures and the resulting bone defects. [13]

- General trauma – Trauma is often a reason for implant placement. Trauma may affect teeth as well as mucosa and bone of the orofacial structures. Trauma to the alveolar process and/or the body of the mandible and the maxilla may reduce bone volume necessary for implant placement. However, there are no data on survival and complications of implants in prosthetically optimal position versus implants in suboptimal position after surgical reconstruction of the lost tissues. [13]
- Bone height in the posterior maxilla (area of the sinus floor) – Bone height in the posterior maxilla is bordered by the sinus floor and by the alveolar bone crest. A bone defect results when there is insufficient bone height for standard implant placement. Insufficient bone height can result from pneumatization of the maxillary sinuses (i.e., maxillary sinus expansion in an inferior direction due to aging) and bone resorption in edentulous regions. Oral surgical interventions maybe necessary to allow implant placement. [13]
- Systemic diseases – Some systemic diseases are associated with abnormal and incomplete tooth and bone formation during growth and development (e.g., ectodermal dysplasia[13]). The alveolar process is not formed or is reduced in volume when teeth do not develop. Bone deficiency will impact implant placement. However, there are no data on survival and complications of implants in prosthetically optimal position compared to implants in suboptimal position after surgical reconstruction of the lost tissues. [13]

Hard tissue deficiencies after implant placement

Hard tissue deficiencies after dental implant placement may be mostly categorized into bone deficiencies associated with healthy situations and those associated with malfunctions and diseases.

Factors affecting hard tissue deficiencies after implant placement include:

- Defects in healthy situations – Defects of the alveolar process (e.g., dehiscence,[14] fenestration,[15] infrabony defects) can exist when teeth are present. After tooth extraction, bone defects will result. Lingual undercuts are a frequent finding in the mandibular anterior and the premolar and molar areas. Bony undercuts may lead to bone defects at implants sites.
- Malpositioning of implants – Malpositioning of implants maybe associated with peri-implant tissue breakdown. For example, research has shown buccal mucosal recession was associated with more buccal implant positioning. Further, the more buccally an implant was placed from the centre of the alveolus was associated with a more apical position of the buccal crest of bone.
- Peri-implantitis – Peri-implantitis leads to hard and soft tissue loss at implant sites.
- Mechanical overload – Mechanical overload may be categorized into loading forces preventing an implant from osseointegrating during the healing phase and loading

[13] Ectodermal dysplasias are a group of more than 180 disorders in which there is abnormal development of the skin, hair, nails, teeth, or sweat glands. [20]
[14] Dehiscence is loss of alveolar bone on the facial (rarely lingual) surface of a tooth from the cementoenamel junction apically. The defect may be 1-2 mm long or extend the full root length.
[15] Fenestration is a "window" of alveolar bone loss on the facial or lingual aspect of the root.

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forces destroying previously established osseointegration. Complete absence of micromotion is not a requirement for successful osseointegration. Research has demonstrated micromotions of less than 50 µm to 150 µm are still amendable to successful bone integration. Excessive strain can lead to bone resorption, whereas amounts below this strain result in bone apposition. The data for overload of osseointegrated implants leading to hard and/or soft tissue defects is very scarce. The evidence for osseointegration loss due to overload is limited to anecdotal reports of single or multiple cases.

- Soft tissue thickness – Studies have shown an association between soft tissue thickness and bone loss indicating thin soft tissues lead to increased marginal bone loss compared to thick soft tissues at implants.
- Systemic diseases – Hard tissue deficiencies after implant placement may result from systemic diseases, bone diseases, medications, and certain types of therapies. For example, prolonged high doses of bisphosphonates increase the risk of bone necrosis of the jaws[16] in conjunction with implant therapy. High dose radiotherapy to the jawbone regions may lead to impaired bone turnover resulting in bone loss at implants. Some studies have noted increased bone loss as well as soft tissue recession at implants when individuals underwent radiotherapy for oral cancer. [13]

Soft tissue deficiencies prior to implant placement

Soft tissue deficiencies prior to dental implant placement includes situations when the available amount of soft tissue does not:

- Easily allow soft tissue coverage of bone volume augmentations;
- Allow tension free primary coverage of the site of implant placement; or
- Allow tension free adaptation of the keratinized soft tissue flap around the neck of the implant. [13]

Factors affecting soft tissue deficiencies prior to implant placement include:

- Tooth loss – The changes to the alveolar ridge after tooth loss are the most common reason leading to soft tissue deficiencies prior to implant placement. Specifically, as the alveolar ridge decreases in size following tooth extraction, the soft tissue covering is also reduced. Thus, a diminished amount of soft tissue to cover an implantation site and the regenerated bone can be problematic.
- Periodontal disease – Untreated periodontitis leads to loss of periodontal support including soft tissue recession and bone resorption.
- Systemic diseases – Some systemic diseases are associated with abnormal and incomplete bone formation (e.g., osteogenesis imperfecta[17]). The reduced bone formation may result in lack of bone volume to support implant placement. The amount of soft tissue present is related to the amount of bone present. Thus, if bone augmentation is needed for implant placement, the available soft tissue may be insufficient to cover the new bone volume during the regeneration surgery. This lack of soft tissue may make implant treatment more challenging. Currently, there is

[16] Refer to Episodes #41 and #42 for information on bisphosphonates and osteonecrosis of the jaw.
[17] Osteogenesis imperfecta is a group of genetic disorders that mainly affect the bones, leading to bones that fracture easily, often with mild injury or no apparent cause.

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limited data investigating the measures to increase the amount of soft tissue to help cover bone augmentation sites. [13]

Soft tissue deficiencies after implant placement

Factors affecting soft tissue deficiencies after implant placement include:

- Lack of buccal bone – Some studies have reported the lack of buccal bone at implants to be associated with decreased height of facial soft tissues. However, there are conflicting results from studies reporting whether or not the buccal bone plate will remain stable over time and will support the buccal soft tissue of the implant in the long-term. [5] [13]
- Papilla height – The height of the papilla between implants and teeth is affected by the level of the periodontal tissues on the teeth adjacent to the implants. The height of the papilla between implants is determined by the bone crest between the implants. A major soft tissue deficiency is the reduced papilla height between two adjacent implants, which may cause esthetic problems in visible areas. [5] [13]
- Keratinized tissue – There are numerous studies assessing the associations between clinical and radiographic parameters and the presence or absence of a band of keratinized mucosa at implant sites. Currently, the results are inconclusive regarding the effect on long-term health and maintenance of dental implants with these clinical conditions. However, it appears keratinized mucosa may have advantages in increasing client comfort and ease of plaque removal. [5] [13]
- Migration of teeth and life-long skeletal changes – Discrepancies between implants and teeth (e.g., open contacts) may develop due to tooth wear and anatomical changes of face and jawbones in adults over time. Studies have documented migration of teeth adjacent to implants. However, the clinical consequences of hard and soft tissue defects are poorly examined and understood. [13]

Take home messages

Utilizing the AAP classification of peri-implant diseases and conditions will:

- Aid in comprehensive treatment planning and enhance client care, thus increasing client oral and overall health.
- Support monitoring of dental implant health for early identification of disease to initiate treatment. If peri-implantitis develops, it initiates within the first few years after the implant is in function. This stresses the importance of carefully monitoring changes that may occur around dental implants in the early post-restorative phase, with focus on bleeding and/or suppuration on probing and in combination with radiographic evidence of bone loss.
- Improve communication with clients increasing their awareness and understanding of their oral condition and to take ownership of their oral health. For example, clients need to be aware of the importance of thorough biofilm removal and regular preventive implant therapy to help ensure implant health. Also, clients should report any changes they note with their implants, such as discomfort or loose components.
- Benefit interprofessional collaboration, for example, in communicating clinical findings with other oral health professionals and referrals to specialists.

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References

[1] American Academy of Periodontology, "Proceedings from the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions," 2021. [Online]. Available: https://www.perio.org/2017wwdc. [Accessed 27 September 2021].

[2] J. Caton, G. Armitage, T. Berglundh, I. Chapple, S. Jepsen, K. Kornman, B. Mealey, P. Papapanou, M. Sanz and M. Tonetti, "A new classification scheme for periodontal and peri-implant diseases and conditions – Introduction and key changes from the 1999 classification," *Journal of Periodontology,* vol. 89, no. 1, pp. S1-S8, 21 June 2018.

[3] M. Araujo and J. Lindhe, "Peri-implant health," *Journal of Periodontology,* vol. 89, no. S1, pp. S249-S256, 21 June 2018.

[4] S. Renvert, G. Persson, F. Pirih and P. Camargo, "Peri-implant health, peri-implant mucositis, and peri-implantitis: Case definitions and diagnostic considerations," *Journal of Periodontology,* vol. 89, no. S1, pp. S304-S312, 21 June 2018.

[5] T. Berglundh, G. Armitage, M. Araujo, G. Avila-Ortiz, et al, "Peri-implant diseases and conditions: Consensus report of workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions," *Journal of Periodontology,* vol. 89, no. S1, pp. S313-S318, 21 June 2018.

[6] S. Dhir, L. Mahesh, G. Kurtzman and K. Vandana, "Peri-implant and periodontal tissues: A review of differences and similarities," *Compendium of Continuing Education in Dentistry,* vol. 34, no. 7, July/August 2013.

[7] N. Lang and P. Bartold, "Periodontal health," *Journal of Clinical Periodontology,* vol. 45, no. S20, pp. S9-S16, 20 June 2018.

[8] American Academy of Periodontology, "Academy report: Peri-implant mucositis and peri-implantitis: A current understanding of their diagnoses and cinical implications," *Journal of Periodontology,* vol. 84, no. 4, pp. 436-443, 1 April 2013.

[9] L. Heitz-Mayfield and G. Salvi, "Peri-implant mucositis," *Journal of Periodontology,* pp. S257-S266, 21 June 2018.

[10] G. Salvi and C. Ramseier, "Efficacy of patient-administered mechanical and/or chemical plaque control protocols in the management of peri-implant mucositis. A systematic review," *Journal of Clinical Periodontology,* vol. 42, no. S16, pp. S187-S201, 11 December 2014.

[11] F. Schwarz, J. Derks, A. Monje and H. Wang, "Peri-implantitis," *Journal of Periodontology,* vol. 89, no. S1, pp. S267-S290, 21 June 2018.

[12] N. Lang and Berglundh, "Periimplant diseases: where are we now? – Consensus of the Seventh European Workshop on Periodontology," *Journal of Clinical Periodontology,* vol. 38, no. S11, pp. 178-181, 16 February 2011.

[13] C. Hämmerle and D. Tarnow, "The etiology of hard- and soft-tissue deficiencies at dental implants: A narrative review," *Journal of Periodontology,* vol. 89, no. S1, pp. S291-S303, 21 June 2018.

[14] G. Walko, M. Castañón and G. Wiche, "Molecular architecture and function of the hemidesmosome," *Cell and Tissue Research,* vol. 360, no. 2, pp. 363-378, 2015.

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[15] BiologyOnline, "Polymorphism," 2021. [Online]. Available: https://www.biologyonline.com/dictionary/polymorphism. [Accessed 2 October 2021].

[16] J. Mohamed, M. Alam, G. Singh and S. Chandrasekaran, "The management of retrograde peri-implantitis: A case report," *Journal of Clinical & Diagnostic Research,* vol. 6, no. 9, pp. 1600-1602, November 2012.

[17] P. Jeyaraj, "Management of central giant cell granulomas of the jaws: An unusual case report with critical appraisal of existing literature," *Annals of Maxillofacial Surgery,* vol. 9, no. 1, pp. 37-47, January-June 2019.

[18] P. Sarwal and K. Lapumnuaypol, "Pyogenic Granuloma," StatPearls [Internet}, 5 December 2020. [Online]. Available: https://www.ncbi.nlm.nih.gov/books/NBK556077/. [Accessed 5 October 2021].

[19] V. Ngan, M. Sivasaththivel, S. Tandon and L. Ly, "Pyogenic granuloma," DermNet NZ, January 2021. [Online]. Available: https://dermnetnz.org/topics/pyogenic-granuloma. [Accessed 5 October 2021].

[20] National Institutes of Health, "Ectodermal dysplasia," 17 July 2017. [Online]. Available: https://rarediseases.info.nih.gov/diseases/6317/ectodermal-dysplasia. [Accessed 5 October 2021].

Additional Resources

A new classification scheme for periodontal and peri-implant diseases and conditions – Introduction and key changes from the 1999 classification, Caton, J; Armitage, G; Berglundh, T; Chapple, I; Jepsen, S; Kornman, K; Mealey, B; Papapanou, P; Sanz, M; Tonetti, M. *Journal of Periodontology*, Volume 89, Issue S1, June 21, 2018, p. S1-S8 https://aap.onlinelibrary.wiley.com/doi/10.1002/JPER.18-0157

Peri-implant diseases and conditions: Consensus report of workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions, Berglundh, T; Armitage, G; Araujo, M; Avila-Ortiz, G; Blanco, J; et al. *Journal of Periodontology*, Volume 89, Issue S1, June 21, 2018, p. S313-S318 https://aap.onlinelibrary.wiley.com/doi/10.1002/JPER.17-0739

Peri-implant health, Araujo, M; Lindhe, J. *Journal of Periodontology*, Volume 89, Issue S1, June 21, 2018, p. S249-S256 https://aap.onlinelibrary.wiley.com/doi/10.1002/JPER.16-0424

Peri-implant mucositis, Heitz-Mayfield, L; Salvi, G. *Journal of Periodontology*, Volume 89, Issue S1, June 21, 2018, p. S257-S266 https://aap.onlinelibrary.wiley.com/doi/10.1002/JPER.16-0488

Peri-implantitis, Schwarz, F; Derks, J; Monje, A; Wang, H. *Journal of Periodontology*, Volume 89, Issue S1, June 21, 2018, p. S267-S290 https://aap.onlinelibrary.wiley.com/doi/10.1002/JPER.16-0350

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Peri-implant health, peri-implant mucositis, and peri-implantitis: Case definitions and diagnostic considerations, Renvert, S; Persson, G; Pirih, F; Camargo, P. *Journal of Periodontology*, Volume 89, Issue S1, June 21, 2018, p. S304-S312 https://aap.onlinelibrary.wiley.com/doi/10.1002/JPER.17-0588

The etiology of hard- and soft-tissue deficiencies at dental implants: A narrative review, Hämmerle, C; Tarnow, D. *Journal of Periodontology*, Volume 89, Issue S1, June 21, 2018, p. S291-S303 https://aap.onlinelibrary.wiley.com/doi/10.1002/JPER.16-0810

Disclaimer: This document is educational and not intended to provide clinical advice nor should it be used as a replacement for professional dental or medical advice. Dental hygienists are encouraged to consult with CDHO practice advisors and refer to CDHO guidelines. Dental hygienists are responsible for the decisions they make and for the consequences associated with those decisions. © ODHA