PERIODONTAL LITERATURE REVIEW: 
THE NEXT GENERATION

The Periodontal Literature Review: The Next Generation is a living document and will be continuously updated as sections are added and reviews of new, relevant research are published.

One of the most important elements of any post-graduate training is the Literature Review Program. This particular course has been a part of the Periodontology training programs at all major institutions for decades. An important resource in the review of “classic” literature was and still is the Periodontal Literature Reviews: A summary of current knowledge (PLR). This volume has been a required reading for our program since its publication since 1996. This book was not only a useful text for students preparing for their board examinations but was also a wonderful reference guide for the practicing periodontist. However, as with any type-set book, the material held within is stagnant. Millions of pages have been written on the treatment of periodontal disease and tooth loss since 1996 yet there is no resource available that has continuously updated “classic” literature since that time.

Therefore, the American Academy of Periodontology sought to find a way to update this invaluable guide. Our proposal from the University of Toronto Department of Periodontology was to create a “web-based” document that would allow for continuous updating and editing, such that the website might be considered a “living document” for all post-graduate students studying for their board exams as well as practicing dentists and periodontists who want to read up on a particular topic. The website should not only be a complement to the original version of the text, but it should also broaden the scope of subjects as well as provide enhanced visual capabilities and ease of navigation to match the available technology of today. By providing an electronic version of the review, direct links to the articles being reviewed are embedded within the text and thus made available within an instant of a click allowing students to critically evaluate the original material - a real tenant of this educational endeavor.

Articles were selected through a web-based search using key words and titles for the various topics to be discussed. Articles were read and reviewed by residents of the University of Toronto’s Graduate Program in Periodontology and selected based on their clinical relevance. As with the original 1996 textbook developed by the Air Force Residency Program, this version was also meant to provide an overview of the subject only and was not meant to be a comprehensive as a textbook on a given subject.

We hope that this website will continue to expand and prove to be a useful resource for students and teachers alike.

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PERIODONTAL LITERATURE REVIEW:
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A review of periodontal literature from 1996 – 2010

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IMPLANT THERAPY

The field of dental implant therapy has exploded in terms of volume and scope since the initial AAP literature review was published in 1996. At that time 5 (?) pages were devoted to the topic. Since then, scores of research papers, textbooks, symposia and implant systems have entered (and left) the field. This review will only touch the surface of the subject. However, as the database grows, so too will our understanding of this field increase such that this section will, in future, provide a good foundation in the understanding of dental implant therapy.

BIOMECHANICS OF IMPLANTS

Complications and failures due to mechanical factors including component fracture, coronal bone resorption and fixture loss have frequently been reported during implant-prosthetic treatment. Biomechanically, forces derived from functional or parafunctional occlusal contacts on implant-supported restorations usually induce physiological adaptation of supporting tissues since implants are tightly anchored into the bone. However, if the stress generated is beyond the adaptive capacity of the host, the response of the supporting tissues and prosthetic components may result in mechanical and/or biologic failures. Mechanical failures comprised of screw bending, loosening, fracturing or implant fracture in extreme cases whereas a biologic failure results from a resorptive-remodeling of the bone surrounding the implant that leads to progressive bone loss. Schrotenboer et al (2008) discussed several factors that are known to affect the stress/strain distribution on bone surrounding implants in their article the studied the effect of microthreads and platform switch on crestal bone stress levels. These factors included the design and position of the implant, implant-abutment connection, cantilever length, surface roughness, bone quality and type, depth of insertion, arch configuration, the nature of bone-implant interface, and occlusal conditions. However, Goodacre et al (2003) noted that biological failures are the most common implant-related complications that occur after implants are loaded. Implant failure primarily occurs within 18 months of initial loading. These early loading failures occur most often in poor-quality bone (16% failure) and with shorter implant lengths (17% failure).

Several investigators have tried to gain insight into implant loading magnitudes by performing tests using experimental, analytical, and computer-based simulations of various implant-supported prosthesis types. The biting force on implants was generally measured as gentle biting force, biting as when chewing and maximal biting force. Duyck et al (2000) assessed the distribution and magnitude of forces in an in vivo situation where a fixed prosthesis was in place. The prosthesis was supported by a variable number of implants (3 vs. 5-6) and compression/tension measurements were gathered. As one might suspect, higher forces were observed on implants where fewer supports were present. Kim et al (2005) showed that a moderate vertical biting force of 250 N applied at the mesial end of an implant supported prosthesis generates a large compressive force of 450 N on the mesial implant and a tensile loading of 200 N on the distal implant. It is considered that possible overloading factors include overextended cantilever (>15 mm in the mandible and >10-12 mm in the maxilla), parafunctional habits or heavy bite force, excessive premature contacts (>100 μm), large occlusal table, steep cusp inclination, poor bone quality/quantity and inadequate number of implants will influence to forces produced on an implant.

In 2006, Isador reviewed the literature on the effects of occlusal loading and its effects on supporting bone. The response to increased stress on bone, up to a certain threshold, would be that of strengthening the support bone. However, beyond the threshold, fatigue microdamage may occur, leading to a possible loss of crestal bone and possibly the demise of the implant. To overcome this effect, there have been a number of studies on the effects of coronal microthreads on the maintenance of crestal bone levels. Lee et al (2007) looked at 17 patients who had Astra Tech implants placed and the effect of their microthread design on the maintenance of
crestal bone levels after three years. Depending on the implant design used, there was a significant difference in crestal bone levels noted over a one year period. Crestal bone loss was also greatest in the first year, but stabilized such that there was no difference moving into year two and year three of the study. Three dimensional finite element models appear to confirm reduced forces in crestal bone with microthread models. However, how this may translate clinically still requires further investigation (Hudieb et al. 2011).

**BILOGIC WIDTH AND IMPLANTS**

Understanding the relationship between the peri-implant soft tissue and the implant surface is critical to ensuring successful rehabilitations. The dentogingival junction surrounding natural teeth has been well documented since 1959, when Sicher described both epithelial and connective tissue attachment to a tooth. In 1961, the term biologic width was introduced by Gargulio based on his work describing vertical dimension of this dentogingival junction in human cadaver jaws. He described the average sulcus depth (0.69 mm), length of the epithelial attachment (0.97 mm), and connective tissue attachment (1.07 mm) that occurs around natural teeth. It has since been hypothesized that a similar relationship between the bone and overlying soft tissue around dental implants exists as well. Peri-implant tissues have many similarities with periodontal tissues and dentogingival junction, but there are some obvious anatomic differences as well, such as the lack of periodontal ligament. Peri-implant biological width has been studied and measured in both histological animal studies and clinical human studies and the literature surrounding the concept of biologic width around implants will be discussed below.

One of the known potential sequelae following dental implant placement is crestal alveolar bone loss. The establishment of biologic width around the implant may be one of the reasons behind this occurrence. Some bone loss is to be expected: as described by Albrektsson, 1.5 mm of crestal bone loss within the first year of implant function would be considered success. Implant overload, microgap, polished implant neck, and infection are all factors that have also been implicated in early peri-implant bone loss, but the formation of biologic width may play a role in this as well.

Both human and animal studies have been conducted to describe the structure of biologic width around implants. Berglundh (1991) first demonstrated that the peri-implant mucosa formed a cuff-like barrier around the surface of the titanium abutment. The tissue is essentially scar tissue that repairs the injury of the implant insertion and additional tissue is formed to protect the exposed bone and seal the emergence of the implant. As is seen the gingiva around natural teeth, the peri-implant mucosa has a well-keratinized oral epithelium that is continuous with a junctional epithelium that faces the titanium surface. In a rat study, Ikeda (2000) described the presence of a basal lamina and hemidesmosomes in peri-implant junctional epithelium. As well, Abrahamson (1999) showed that the apical portion of the epithelium around titanium implants is very thin and attached to the implant with hemidesmosome-like structures. Arvidson (1996) also evaluated the peri-implant seal with soft tissue biopsies of human samples and found the junctional epithelium attached to the implant surface through hemidesmosome-like structures, which is the same attachment as is seen around teeth.

However, other studies have shown differences in the junctional epithelium tissues. Shioya (2009) found that eight weeks after implant placement, the implant interface appeared to be sealed by aligned special cells with surrounding elongated fibroblasts and bundles of collagen fibers. No hemidesmosomes and no basal lamina were found in this tissue. With regard to the connective tissue compartment, Moon (1999) described this zone to be similar to scar-like tissue that has contacted the implant but had no attachment to it. Circular fibers were found in the inner zone of connective tissue, next to the titanium surface; in the outer layer, horizontal and vertical fibers were found. These fibers were running from the periosteum and the alveolar crest towards the
oral epithelium. Glauser (2005) conducted a human histological study and calculated the total height of this peri-
implant soft tissue to be between 4-4.5 mm, including the sulcus (0.2-0.5 mm), junctional epithelium (1.4-2.9 mm) and connective tissue (0.7-2.6 mm). It has been suggested by Linkevicius (2008) that the function of this soft tissue collar is similar to that of biologic width around teeth and may serve as a protective mechanism for the underlying bone.

Berglundh and Lindhe (1996) also demonstrated that a constant gingival dimension was observed around the peri-implant tissues. They also went on to evaluate some of the factors that may impact on the dimension of this tissue. The authors showed that by surgically reducing the thickness of the gingival flap prior to suturing, crestal bone loss would subsequently occur allowing for the reestablishment of biologic width. Other factors that can potentially influence biologic width include one- versus two-stage procedures, immediately loaded or unloaded implants, different implant surfaces, and different implant structures and positions.

The effect of one- versus two-stage procedures on the resultant biologic width has been studied extensively. Abrahamsson (1996) evaluated the effect of a one-stage versus two-stage procedure on the soft tissue healing around three different implant systems (Astra Tech, Brånemark and Bonefit-ITI). The histological results demonstrated similar dimension and composition of the epithelial and connective tissue components around all systems in both protocols. Ericsson (1996) also noted similar soft tissue adaptation and proper osseointegration in Brånemark implants installed according to a one-stage or a two-stage procedure.

The influence of timing of loading an implant has been studied as well. Cochran (1997) evaluated the biologic width around non-submerged unloaded and immediately loaded implants. At the three-month point, the constituents of the biological width in the unloaded group were 0.49 mm (sulcus depth), 1.16 mm (junctional epithelium), and 1.36 mm (connective tissue component). The measurements in the loaded group were 0.50 mm, 1.44 mm, and 1.01 mm, respectively. The results were also similar after 12 months of loading, confirming that the biological width around implants resembles the one present around teeth and that the dimension of its constituents are independent from the loading variable. Glauser (2006) later concluded that once an immediately-loaded implant has successfully integrated, it appears to show a similar soft-tissue reaction with regard to periodontal as well as morphologic aspects in comparison to conventionally loaded implants.

Implants are produced with various designs, such as one piece implants, with a transmucosal part in continuity with the endosseous part, or two piece implants, which present an interface between the endosseous component and the transmucosal component, resulting in the formation of a microgap between the components. In Abrahamsson’s (1996) study comparing one- and two-piece implant systems, no significant differences were noted in the dimension or composition of the epithelial and connective tissue components. This group (1997) went further on to investigate the influence of the abutment disconnection/reconnection on the marginal peri-implant tissues. They found that this abutment manipulation compromised the mucosal barrier and caused an apical migration of the connective tissue. Thus, while normal proportions and dimensions of the surrounding tissues were observed in a control group, at test sites the abutment manipulation resulted in a mechanical injury to the soft tissue barrier that had to reestablish more apically, causing a marginal bone resorption of approximately 1.5 mm. Further, Todescan (2002) investigated the influence of the position of the implant shoulder on the soft tissue healing. Twenty-four Brånemark implants were placed into dogs, and these were divided into Group 1 (implants placed 1 mm above the bone crest), Group 2 (implant shoulder was placed at the level of the bone crest) and Group 3 (implants placed 1 mm below the bone crest). The junctional epithelium showed a mean value of 1.67 mm in Group 1, 1.93 mm in Group 2 and 2.78 mm in Group 3. The corresponding values for the band of connective tissue were 1.13 mm, 0.92 mm and 1.60 mm, respectively. Differences between Groups 2 and 3 were significantly different, demonstrating a
tendency towards longer junctional epithelium and connective tissue component the deeper the implants were placed.

**BONE QUALITY AND IMPLANTS**

Implant stability plays a major role in the eventual osseointegration of a dental implant. A significant factor in establishing this stability is bone quality. Primary stability is related to the quality and quantity of bone surrounding an implant at its initial placement. Secondary stability is established during the healing process whereby bone formation and bone remodeling occurs adjacent to the implant surface. Meredith (1997) described several techniques for measuring bone quality and implant stability in his review paper on the subject. As related to the time of this article, such instruments as Periotest®, and the Dental Fine Tester® were used to assess the stability of both teeth and dental implants. Cutting resistance measurements were also employed to determine the quality of bone during osteotomy preparation. Prior to restoring the implant, the application of a “reverse torque” was used applied to the implant up to 20 Ncm in a counter-clockwise direction. Well-integrated implant will resist this torqueing movement. Currently, such an instrument as the ISQ (Implant Stability Quotient, Osstell) is used to determine implant stability and by extension, bone quality through a resonance frequency analysis.

In reviewing bone quality at the time of implant placement, Friberg et al (1999) established a classification of cutting torque measurements that could be ascertained at the time of implant site preparation. This particular study looked at a 3-year follow-up of 105 consecutively treated individuals, with the goal of reinforcing the classification established in 1994 on the variations of cutting torque which were dependent on the site of implant placement. The cutting torque comprises the “true cutting resistance of bone” and the friction torque. As expected, cutting torque resistance was higher in the mandible than the maxilla, with declining values from anterior to posterior. The corresponding success of implant survival was correlated to the quality of bone insofar as those implants placed in the anterior mandible had a higher survival rate than those placed in the posterior maxilla. Johansson et al. (2004) looked at cutting torque measurements in conjunction with implant placement in grafted and non-grafted sites. In this study, 40 subjects were treated, 27 having grafted sites, 13 with no grafting performed. Significantly lower cutting torque was noted in the grafted versus the non-grafted sites. The cutting torque showed a similar distribution as previously described. However, after 6 months of onlay graft healing, the differences in torque between the non-grafted sites were not significantly different. In all regions, cutting torque values in failed implant sites were lower than those where implant success was recorded. It may be surmised that cutting torque resistance may be used as a means of judging the quality of the osteotomy site and the potential for implant success.

More recently, a number of groups have looked at the use of Resonance Frequency Assessment (RFA) as a non-invasive means of assessing bone quality. Huang et al (2002), using bone block models, demonstrated a linear relationship between bone quality type and resonance frequency results. Resonance frequency was be reduced with worsening bone quality. Similar findings were noted with Lachmann et al (2006), and Ostman et al (2006).

Turkyilmaz reported in a series of papers (2006, 2007) on the use of computerized tomography as a means of determining bone quality. In these studies, patients’ CT scans were assessed with the relative density being expressed in Hounsfield units. In one study, 85 patients from two clinics were assessed. 158 implants in total were placed. Once implants were placed, stability was assessed using resonance frequency values and torque strength. The recorded mean bone densities based on CT scan results were consistent with all previous studies, with the anterior mandible being most dense and the posterior maxilla demonstrating less than half the density (970 HU, 417 HU). The posterior mandible and anterior maxilla demonstrated similar density (669 HU,
696 HU respectively). Implant stability, as recorded by the ISQ was also similarly correlated. These studies indicated the potential utility of CT scans to determine site density prior to implant placement. With the advent of Cone Beam CT scans, which produce less radiation than conventional CT scan, there may be a greater place for these tools in the planning of implant therapy.

**BONE TO IMPLANT CONTACT**

A significant amount of funds and research has been directed at improving surface characteristics of implants in order to improve bone to implant contact. However, the mechanisms of the concept of “osseointegration” were best described by Davies (1998) where he described the sequence of bone healing around a dental implant. In this review, Davies juxtaposes the terms “distance osteogenesis” from “contact osteogenesis”. These terms, originally coined in the 1980’s generally relate to the bone formation on and around an implant surface. The two concepts suggest different mechanisms by which osteogenic cells either line “old bone” surfaces and secrete bone towards the implant (distance osteogenesis) or osteogenic cells coming in intimate contact with the implant surface with new bone being laid down along the implant itself (contact osteogenesis). One can suspect that a combination of both mechanisms may occur in osteogenesis around an implant. The cells involved in contact osteogenesis are likely derived from undifferentiated peri-vascular connective tissue cells. Once osseoconduction has been completed, de novo bone formation occurs, resulting in a mineralized bone matrix. It is these phases that will be effected profoundly by the surface characteristics and topography of the implant itself. The third phase, that of bone remodeling, will complete the integration process, and again be quite dependent on the surface characteristics of the implant.

Masuda et al (1998) reiterated the complexities of the integration process and the importance of surface characteristics and topography in this process. This review assessed the literature of its time in terms of light microscopic evaluations of bone-implant contact, histological findings, and the ultrastructural characteristics of the bone-implant interface. The results of this review suggested that surface characteristics did have a significant influence on the amount of bone formed around the implant. However the mechanism of these differences was still up for debate.

In 2001, Khang et al. initiated a multi-centered trial that looked at the difference between acid-etched and machine-surface implants and the bone quality around the given implant. In this study, 97 patients were enrolled in either a private practice setting or a university clinic. 247 acid-etch and 185 machined-surface implants were placed and allowed to integrate for up to 6 months. Implant-supported fixed prostheses or overdentures were placed, depending on the subject's individual needs. Of the 432 implants placed, 36 failed. There was statistically significant differences in the pre-loading success rate of the acid etch (95%) versus the machined surface implant (86.7%).

**IMPLANT RISK FACTORS**

Although implants have been shown to be an effective method by which to restore missing teeth in a healthy individual, it has come to the attention of many that there are certain health-related issues that may affect the outcomes of implant therapy. Klokkevold and Han (2007) conducted a systematic review to determine the effects of smoking, diabetes and periodontal disease on implant therapy. Implant survival rates were compared between smokers and non-smokers, diabetics and non-diabetics as well as those treated for periodontal disease versus those with no treatment for periodontal disease. The findings revealed a statistically significant difference between success and survival of implants in non-smokers versus smokers. There was no difference noted in survival in those with and without diabetes. There was also no difference between those treated for periodontitis in the past and those who had never been treated. This particular meta-analysis indicated that
smokers were at higher risk for complications, as compared to the other systemic issues study. Another systematic review by Heitz-Mayfield et al (2009) reinforced the findings that well-controlled periodontal conditions do not increase the risk of implant complications and/or failures. This study also found the majority of studies reviewed found smoking to be a statistically significant risk factor for adverse implant outcomes with the implant survival rate ranging from 80% to 96% in 5-10 year studies.

Smoking has local and systemic negative effects in terms of tissue healing. Nicotine local absorption causes vasoconstriction and compromises the blood supply needed for the nutrition and irrigation to the implant bed during the healing stage. In fact, Sanchez-Perez et al (2007) found that the use of tobacco involves a 15.8% risk of implant failure and that the consumption of a pack of cigarettes (20) or more daily increases the risk of implant failure to 30.8%. As well, a study by Moy et al. (2005) reported an implant success rate of 80% in smokers compared to 91% in non-smokers and that most failures occurred in the first year after placement and were twice as likely to occur in the maxilla compared with the mandible. However, in a retrospective study, Kumar et al (2002) looked at the initial osseointegration before loading in 461 patients with 1183 surface-modified ITI SLA implants and found that the success rate of integration was 98% in both smokers and non-smokers. There was also no difference in outcome between maxillary and mandibular implants. Bain (2002) also noted that surface modifications of implants may resist the effects of smoking.

Diabetes

Uncontrolled diabetes has an impact on success rate of the implants. Patients with uncontrolled diabetes have been found to have 2.59 higher risk of implant failure (Zupnik, 2011). In uncontrolled diabetic state, chronic hyperglycemia maintains chronic inflammation that could lead to bone loss and implant failure long term (Mellado-Valero, 2007). It also prevents normal osteoblastic formation by altering the PTH response. Patients with uncontrolled diabetes also suffer from higher risk of infections, which could delay the wound healing, in this case the implant integration. Therefore, uncontrolled diabetes or hyperglycemic state is a risk factor for implants failure and patients should be informed about their condition and the effect on the success rate of the implants.

Osteoporosis

Osteoporosis is reported to occur in one third of the North American female population above 65 years of age, and is treated by either estrogen hormone therapy or bisphosphonate medications. The bone quality in post-menopausal women and osteoporotic patients became an issue of interest due to the association of poor bone quality to implant failure (Virdie, 2007). Implant success has been reported to be higher in good quality bone such as the bone found in anterior mandible (Chee, 2007). Animal studies have shown that estrogen deficiency interferes with bone healing around the implant and that most failures occurred in the first year after placement and were twice as likely to occur in the maxilla compared with the mandible. However, in a majority of clinical trials, the differences between the osteoporotic patients and normal group in terms of implant success and osseointegration have not been found to be statistically significant.

History of Periodontitis

Periodontitis is one of the main causes of the loss of teeth and patients with this condition benefit majorly from implant therapy; however the risk of implant failure has been a point of interest in these patients. According to
literature, the risk of implant failure and the mean bone loss are higher in patients with treated generalized aggressive periodontitis (88.8% 5 year success rate) compared to chronic periodontitis patients (Mengel, 2001). In a comparison of patients with a history of chronic periodontitis to patients with no history of periodontitis, the risk of implant failure and bone loss is higher and the survival and success rate is reported to be lower in patients with history of periodontitis (96.5% survival rate compared to 90.5% in PG) (Hardt, 2002). The plausible mechanism of this kind of failure is that the implants in patients with history of periodontitis or current periodontal condition are more prone by bacterial colonization, inflammation and the bone loss.

Oral and IV bisphosphonates

Oral and IV bisphosphonate are the medications used mainly for the treatment of osteoporosis, Paget’s disease bone pain, hypercalcemia malignancy such as multiple myeloma, breast and other cancers. The concern with bisphosphonate therapy and dental treatments is the risk of osteonecrosis of the jaw, which is reported to have incidence of 0.8%-12% (IV bisphosphonate) and 0.7 per 100,000 person years of exposure (oral bisphosphonate). Osteonecrosis caused by oral bisphosphonate is less common and less severe if it occurs and more responsive to the treatments. According to the majority of the studies, the use of oral or IV bisphosphonate does not have a negative effect on the implant success. Overall, there is not enough evidence to avoid any dental treatment or implant therapy in patients with bisphosphonate therapy but these patients need to be informed of the risk and monitored in case of complications to receive the appropriate treatment promptly (Javed, 2010).

PERIOIMPLANT MUCOSITIS/PERI-IMPLANTITIS

As the popularity of implant therapy grows, the prevalence of peri-implant disease steadily increases. A systematic review, with a mean follow-up period ranging from 5-13 years, estimated that peri-implant mucositis affects 63% of implant patients, and peri-implantitis affects 19% of patients (31% and 9.5% of implants, respectively) (Atieh, 2012). Previous systematic reviews on peri-implant disease found similar and often even higher incidences of disease (Zitzman, 2008).

Peri-implant mucositis and peri-implantitis are inflammatory conditions affecting soft and hard peri-implant tissues. Peri-implant mucositis is defined as a reversible inflammatory condition solely affecting the mucosa surrounding the dental implant. Peri-implantitis is defined as an inflammatory process affecting both soft and hard tissues surrounding the dental implant associated with pathological bone loss around the implant. Similar to the accepted relationship between gingivitis and chronic periodontitis, peri-implant mucositis is considered the precursor of peri-implantitis, yet not all peri-implant mucositis cases will progress to peri-implantitis (AAP, 2013).

A peri-implantitis classification was recently proposed by Froum and Rosen (2012). Similar to chronic periodontitis, this classification allows clinicians to diagnose peri-implantitis lesions based on their severity, utilizing bleeding on probing (BOP), pocket depth (PD) and radiographic bone loss to differentiate between early, moderate and advanced peri-implantitis lesions.

Classification of Peri-implantitis

Early

PD>4mm, BOP/suppuration on 2 or more aspects of the implant
Bone loss of <25% of implant length (compared to at time of loading)
Moderate

PD>6mm, BOP/suppuration on 2 or more aspects of the implant
Bone loss of 25-50% of implant length (compared to at time of loading)

Advanced

PD>8mm, BOP/suppuration on 2 or more aspects of the implant
Bone loss of >50% of implant length (compared to at time of loading)

(Froum and Rosen 2012)

Clinical signs associated with peri-implant mucositis are similar to those associated with gingivitis: bacterial plaque accumulation, erythema of the implant-supporting tissue, bleeding on probing (BOP), and suppuration. Recession may or may not be evident. However, no pathologic bone loss is detected around the implant (AAP, 2013).

In an experimental peri-implant mucositis study, Pontoriero (1994) demonstrated no significant differences in any of the measured clinical parameters (including BOP, PD, and plaque index) around teeth and implants. The study demonstrated a causative effect between bacterial biofilm accumulation and peri-implant mucositis.

Clinical signs associated with peri-implantitis are similar to those observed during peri-implant mucositis, with the presence of pathologic bone loss detected on radiographic examination. Further signs may include increased PD (>5 mm) and implant mobility (AAP, 2013). Most peri-implantitis sites (66%) present circumferential bony defects (Serino, 2013). Bone loss of 2 mm (from immediate post implant placement bone levels) is suggested as a threshold for diagnosing peri-implantitis (Sanz, 2012).

The pathogenesis of peri-implant mucositis follows that of gingivitis through the accumulation of bacterial biofilm on the implant and abutment surfaces leading to a local inflammatory response. The pathogenesis of peri-implantitis was investigated using an animal ligature model. Both ligated teeth and ligated implants demonstrated similar clinical disease progression and alveolar bone loss during the course of the 8 month trial (Lang, 1993). These findings suggest that similar to gingivitis and chronic periodontitis, bacterial biofilm is the main etiologic factor for peri-implant diseases. As such, biofilm formation and the host response, have a significant role in the development of peri-implant mucositis and peri-implantitis.

The acquired pellicle layer is deposited on the implant surface immediately following implant exposure to the oral environment. Bacterial biofilm was demonstrated to mature within two weeks following implant exposure (Heitz-Mayfield, 2010). Within 7 days, sites presented similar pathogenic microbiota found on natural teeth. Biofilm colonization around implants is complete within 7-14 days. The ability of pathogenic bacteria to spread from pockets around natural teeth to peri-implant sites within a short time span highlights the need for complete disease control prior to implant placement.

Several studies investigated the bacterial biofilm composition in healthy peri-implant sites, as well as in peri-implant mucositis and peri-implantitis sites. In general, most studies demonstrated that the composition of the biofilm established on the implant surfaces corresponded closely to that identified on natural teeth (Heitz-Mayfield, 2010). That was demonstrated for both healthy and diseased implant sites. The microbiota associated with healthy implant sites is characterized by gram-positive facultative cocci and rods. Experimental peri-implant mucositis trials demonstrated similar subgingival bacterial biofilm composition in peri-implant mucositis and gingivitis sites (Pontoriero, 1994).
Further investigation of the host innate immune response in the peri-implant tissue was recently published. Authors analyzed peri-implant gingival biopsies and concluded that implant patients with history of chronic periodontitis present similar increase in pro-inflammatory potential (as measured by inflammatory infiltrate and pro-inflammatory molecules) to that found in chronic periodontitis, suggesting similar host innate immune response around teeth and implants (Koutouzis, 2013).

As the bacterial biofilm is presumed to be the main etiologic factor in the development of both peri-implant mucositis and peri-implantitis, treatment is usually aimed at its removal, followed by proper maintenance. Studies investigating the efficacy of different treatment modalities are limited by the lack of a gold standard treatment protocols with which to compare. Suggested treatments are reported as case reports/series or as RCT’s comparing different treatment modalities. As studies report different treatment protocols, clinical measurements and follow-up time, the ability to identify an ideal treatment protocol is limited (Sanz, 2012).

The goal of peri-implant mucositis treatment is the elimination of bacterial biofilm as well as patient education regarding proper home care. No specific treatment protocol was demonstrated to be superior to others for the treatment of peri-implant mucositis, including different home care techniques (Esposito, 2012). Chlorhexidine gel (0.5%) has been suggested as an adjunct treatment following mechanical debridement (Heitz-Mayfield, 2011). Results showed no significant improvement in clinical parameters within 3 months following treatment when compared to mechanical debridement only. The use of systemic antibiotics has also been suggested. Antibiotics were prescribed following mechanical debridement (Azithromycin 500 mg day 1 and 250 mg days 2–4) (Hallstrom, 2012). No significant clinical or microbial differences were found within 6 months following treatment when compared to mechanical debridement only.

Treatment goals include infection control and cessation of tissue destruction, and creation of an environment which allows proper home care and reduces biofilm accumulation. Resective or regenerative procedures may be indicated to provide ideal bone support and architecture. A regular periodontal maintenance plan should follow.

The goal of peri-implantitis treatment is to remove all bacterial deposits from the exposed implant surface. However, due to implant surface characteristics, mechanical debridement is not as effective as root planning of natural teeth. Persson et al. (2010) investigated biofilm samples at baseline, 1 week, 1, 3 and 6 months following scaling (hand instrumentation vs. ultrasonic device). Limited clinical improvement was observed in both treatment groups within 6 months. Furthermore, no differences in counts of bacterial species were recorded between baseline and 6 month time point, suggesting that scaling as a sole treatment for peri-implantitis is not effective at biofilm elimination.

Non-surgical treatment has limited successes in re-osseointegration of exposed implants. For this reason, advanced peri-implantitis sites may benefit from surgical intervention, which will aim at implant surface debridement and decontamination, as well as resective or regenerative procedures (Renvert, 2012). Re-osseointegration of decontaminated implants was demonstrated to be biologically possible in several animal studies, though results are greatly varied. Access surgery was shown to have a positive effect on re-osseointegration rate when compared to non-surgical decontamination. Positive results were obtained with different surgical techniques and different decontamination methods. However, none of the reported animal studies demonstrated full length re-osseointegration. Several human studies reported full length defect fill, yet it is not a predictable result (Renvert, 2009). Surgical techniques include access surgeries, resective surgeries, bone graft and bone graft substitute placement, and regenerative procedures. The success of surgical treatment is affected not only by surface decontamination and the technique used, but also by other site or implant-specific factors (Aghazadeh, 2012). Roccuzzo et al. (2011) compared surgical treatment with bone
graft around 2 different implant surfaces – titanium plasma-sprayed surface (TPS) and sand-blasted large grit acid-etched surface (SLA)(64). It was suggested that implant surface characteristics as well as time in function prior to peri-implantitis, may have an impact on the clinical outcome following surgical treatment of peri-implantitis.

The long-term success of implant therapy should be measured based on function, esthetics and healthy-peri-implant tissues. Early diagnosis and continued management of peri-implant diseases are essential to the success of dental implants. Our knowledge of chronic periodontitis has prepared us well for preventing and treating peri-implant disease. Periodontitis and peri-implantitis share a similar etiology, risk factors and disease course. They do however present unique challenges as well. Perio-implantitis should be diagnosed using probing depths measured without the prosthesis in place whenever possible. Non-surgical treatment of peri-implantitis should include decontamination of the implant surface following debridement. Surgical treatments should also be considered as treatment options after non-surgical treatment has been successful. Access flap procedures may facilitate the debridement and decontamination process. Resective or regenerative procedure are not predictable, however positive results have been reported. The treating doctor should decide on the need for surgical intervention, as there is no proven gold standard with respect to surgical treatment of peri-implantitis. This decision should be based on implant mobility, esthetic zone considerations (surgery is not recommended due to mucosal recession), defect morphology and disease severity. Further research into the specific pathogenesis and treatment options of peri-implant disease is essential to allow clinicians to provide successful and predictable treatments.
IMPLANT THERAPY REFERENCES

BIOMECHANICS OF IMPLANTS


**BIOLOGIC WIDTH AND IMPLANTS**


**BONE QUALITY AND IMPLANTS**


**BONE TO IMPLANT CONTACT**


Schure


**IMPLANT RISK FACTORS**


**PERI-IMPLANTITIS/PERI-IMPLANT MUCOCITIS**


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