Saving Dental Implants

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Foreword

It is great honor to write this foreword for George Romanos's incredible textbook. Congratulations to him for bringing together experts from around the world to add their knowledge and expertise to help us better know the state of the art when it comes to prevention and management of periimplantitis. What is so impressive is that the chapters have both clinical and academic emphases so that the reader will be able to deal with the everyday problems that we all have in our practices.

What is so impressive is that there are many ways to treat this very important problem. The use of lasers, photo dynamics, and implantoplasty with and without bone and

soft tissue grafting each have their own chapters. Wow, this is very special.

I know you will enjoy this book, and it will help you manage your patients at a higher level. Congratulations again to George and all the authors.

Well done!

Dennis P Tarnow DDS Clinical Professor of Periodontology and Director of Implant Dentistry Columbia University College of Dental Medicine

Preface

The increased number of peri-implant complications, such as peri-implant mucositis and peri-implantitis demand the need for a comprehensive analysis of the anatomical conditions, the etiological and risk factors for the disease pathology, medical and prosthodontic aspects, and *last but not least* the management of these clinical entities.

The patient's request for treatment with dental implants puts the responsibility on every clinician to present the pros and cons of this type of therapy and the specifics of pre-implantological diagnostics, treatment methodology, prosthetic concepts, and further maintenance visits.

In "Saving Dental Implants," I have tried to present global treatment approaches and concepts, established philosophies in practice for the last 20 years, but also future aspects of the management of the disease progression. I am excited and proud to have recruited leaders in this field, who have seen how implant therapy has an impact on patients' lives. Our patients deserve the best functional and esthetic outcomes. The foreword in this book, written by Dr. Dennis Tarnow, highlights the past, present, and future of dental Implants.

I would like to thank everyone who provided the effort to illustrate their clinical and research expertise based

on daily practice. This book should be a tool for every clinician and a resource of knowledge, to prepare the foundation around dental implant treatment and how to manage peri-implant diseases focused on long-term predictability. I highlight in this book periodontal, prosthetic, and biomaterial aspects and challenges, for sustainable and long-term successful clinical outcomes. It is an opportunity to gain knowledge, find inspiration, and engage in clinical practices that can prepare each one of us to build the critical thinking we need to treat ailing dental implants.

I would like to thank the Publisher's team and all the authors for supporting me from the beginning. We took on the challenge of the management of peri-implant problems as an opportunity for education, research, and clinical practice advancement. Special thanks to my loving family for their continuous support of my academic work.

Georgios Romanos

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Part I

Etiology - Pathogenesis

Anatomy of the Peri-implant Soft Tissues

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Dental implants anchor into the jawbone through direct contact between the bone and the implant, a process known as "osseointegration." Recent evidence suggests that the sustained success and survival of implants are not exclusively contingent on "osseointegration" but also on the soft tissues enveloping the transmucosal section of the implant, which serves as a barrier between the peri-implant bone and the oral cavity (Figure 1.1). This soft tissue seal, often referred to as the "peri-implant mucosa," plays a crucial role in the overall health and longevity of dental implants [1]. The attachment of soft tissue to the implant functions as a biological seal, ensuring optimal conditions and thwarting the onset of periimplant infections, such as peri-implant mucositis and peri-implantitis. Consequently, the peri-implant soft tissues play a pivotal role in ensuring the long-term survival of implants [1].

As soft tissue develops around teeth during tooth eruption, it forms a seal that protects the supporting tissues – namely, the alveolar bone, periodontal ligament, and cementum – from exposure to the oral cavity [2]. In contrast, the peri-implant mucosa is established after the oral soft and hard tissues undergo a healing process to accommodate the osseointegrated implants. The following section provides a concise overview of the key anatomical features of peri-implant tissues.

Structure of Peri-implant Tissues in Health

During the process of wound healing following the accommodation of dental implants, the features of the periimplant mucosa are established [3] (Figures 1.2–1.4). Berglundh et al. [4] conducted an examination in dogs to

investigate the anatomical and histological features of the peri-implant mucosa formed in a two-stage procedure, comparing them with the gingiva around teeth.

It was revealed that the peri-implant mucosa consists of a keratinized oral epithelium located at the external surface. This epithelium is connected to a thin non-keratinized sulcular epithelium facing the abutment and terminating in junctional epithelium, equivalent to the junctional epithelium around teeth, termed as peri-implant junctional epithelium. The peri-implant junctional epithelium terminates 2mm apical to the coronal soft tissue margin and 1.0–1.5mm coronal to the peri-implant bone crest. The mean supracrestal soft tissue, including sulcus depth, measured 3.80mm around implants and 3.17mm around teeth (Figures 1.2–1.4).

While no statistically significant difference was observed in the height of the junctional epithelium and sulcus depth between implants and teeth, the height of the soft connective tissue was statistically significantly greater around implants than around teeth. The peri-implant junctional epithelium and the soft connective tissue adjacent to the abutment appeared to be in direct contact with the implant–abutment surface [4].

In summary, this study demonstrated that the periimplant mucosa exhibits comparable anatomical features to those of gingiva around teeth [4].

Subsequent studies provided evidence that a similar mucosal attachment formed on titanium in conjunction with different implant systems [5, 6] and around intentionally non-submerged and initially submerged implants [7, 8, 9]. However, the peri-implant junctional epithelium was significantly longer in initially submerged implants to which an abutment was connected later than in intentionally non-submerged implants [9].

3

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Figure 1.1 Clinical image depicting a healthy soft tissue around an osseointegrated implant. *Source:* Photo: Prof. Dr. Georgios Romanos.

Figure 1.2 Photomigrograph of an osseointegrated titanium dental implant depicting the direct bone-implant contact and the supracrestal soft tissue implant contact. *Source:* Photo: Prof. Dieter D. Bosshardt.

Figure 1.3 Higher magnification depicting the supracrestal peri-implant soft tissues consisting of oral and sulcular epithelium and connective tissue adhesion to the implant surface. *Source:* Photo: Prof. Dieter D. Bosshardt.

The biologic width (i.e., the supracrestal soft tissue) was revisited in a further dog experiment after abutment connection to the implant fixture with or without a reduced vertical dimension of the oral mucosa (Berglundh and Lindhe [10]). While the peri-implant junctional epithelium was about 2mm long, the supra-alveolar soft connective tissue was about 1.3–1.8mm high. Interestingly, sites with a reduced mucosal thickness consistently revealed marginal bone resorption, adjusting the width of the supracrestal soft tissue. Evaluating the biologic width around one- and twopiece titanium implants that healed either non-submerged or submerged in dog mandibles, Hermann et al. [11] suggested that the gingival margin is located more coronally, and the biologic width is more like teeth in association with one-piece non-submerged implants compared to either twopiece non-submerged or two-piece submerged implants. These findings were later confirmed in a comparably designed dog study with another implant system [12].

Several studies have evaluated the impact of surface topography (i.e., surface roughness measurements) on the peri-implant mucosa. Cochran et al. [13] failed to show any differences in the dimensions of the sulcus depth, periimplant junctional epithelium, and soft connective tissue

Figure 1.4 Higher magnification of the coronal portion of the supracrestal peri-implant soft tissues. The oral and sulcular epithelium are clearly visible. A more diffuse inflammatory infiltrate, located immediately adjacent to the junctional and sulcular epithelium is visible. *Source:* Photo: Prof. Dieter D. Bosshardt.

contact to implants with a titanium plasma-sprayed (TPS) surface or a sandblasted acid-etched surface. Abrahamsson et al. [14, 15] observed similar epithelial and soft connective tissue components on a rough (acid-etched) and smooth (turned) titanium surface. The biologic width (i.e., supracrestal soft tissue) was greater on the rough surface; however, without a statistically significant difference from that around a smooth surface.

Findings from two human histologic studies revealed less epithelial downgrowth and a longer soft connective tissue in conjunction with oxidized or acid-etched titanium compared to a machined surface [16]. In a study in baboons, Watzak et al. [17] showed that implant surface modifications had no significant effect on the biologic width after 18 months of functional loading. Following a healing period of 3 months, nano-porous $TiO₂$ coatings of onepiece titanium implants showed a similar length of periimplant soft connective tissue and epithelium as the uncoated, smooth neck portion of the control titanium implants in dogs [18]. Schwarz et al. [19] suggested that soft tissue integration was more influenced by hydrophilicity than by microtopography.

Several studies revealed that the epithelial cells attach to different implant materials in a comparable way to that of the junctional epithelial cells to the tooth surface via hemidesmosomes and a basal lamina [3].

Analyzing the intact interface between soft connective tissue and titanium-coated epoxy resin implants, the parallel orientation of collagen fibrils to the titanium layer was confirmed [20, 21]. Since implants lack a cementum layer that can invest the peri-implant collagen fibers, the attachment of the soft connective tissue to the transmucosal portion of an implant is regarded as being weaker than the soft connective tissue attachment to the surface of a tooth root. Therefore, improving the quality of the soft tissue-implant interface is considered to be of great relevance for maintaining healthy peri-implant tissues [3].

Studies on the distribution of the collagen types on the peri-implant soft tissues have been evaluated by different research groups. Chavrier et al. [22] examined collagen types I, III, and IV as well as noncollagenous glycoproteins (i.e., laminin and fibronectin), and they could not find any significant structural differences between peri-implant mucosa and gingiva. However, Chavrier et al. [22] underlined the clinical importance of collagen type III and fibronectin in keratinized mucosa surrounding implants, because these proteins seem to promote connective tissue repair around implants after the second surgical stage (implant uncovering) or in case of an inflammatory response.

Romanos et al. [23] emphasized the role of collagen type V in peri-implant soft tissues. Specifically, Romanos et al. [23] evaluated the extracellular matrix quality around nonsubmerged implants with clinically healthy conditions in humans and demonstrated a higher amount of collagenaseresistant matrix containing collagen type V, when implants are one-piece with a transmucosal highly polished neck (without implant-abutment microgap). These collagen fibers in a filament-type distribution were oriented in a different way in the stroma. The fibers were more intense around the blood vessels and nerves, and in some areas, formed parallel fibrillar bundles. The structural differences may be responsible for the defense of peri-implant keratinized gingival connective tissues to bacterial penetration, because of the high amount of the collagen type V-component, which is responsible for the higher collagenase stability.

In contrast to these findings, the peri-implant inflamed tissues showed no difference in terms of collagen type distribution like that around teeth with inflamed gingiva. The collagen type V is the main component of the inflamed granulation tissue around teeth and implants [24]. The authors concluded that quantitative analyses of periimplant versus periodontal soft tissues may be important to confirm morphologic studies in the peri-implant soft tissues. The localization of collagen type V in the tissue may

be of great theoretical and clinical importance and may modify significantly soft tissue management.

The wound healing sequence leading to the establishment of the soft tissue sealing at implants has been evaluated by Berglundh et al. [25]. Immediately after implant placement, a coagulum occupied the implant-mucosa interface. Numerous neutrophils infiltrated the blood clot, and at four days, an initial mucosal seal was established. In the next few days, the area with the leukocytes decreased and was confined to the coronal portion, whereas fibroblasts and collagen dominated the apical part of the implant–tissue interface. Between one and two weeks of healing, the peri-implant junctional epithelium was about 0.5mm apical to the mucosal margin. At two weeks, the peri-implant junctional epithelium started to proliferate in the apical direction. After two weeks, the periimplant mucosa was rich in cells and blood vessels. At four weeks of healing, the peri-implant junctional epithelium migrated further apically and occupied now 40% of the total soft tissue implant interface. The soft connective tissue was rich in collagen and fibroblasts and well-organized. The apical migration of the peri-implant junctional epithelium was completed between six and eight weeks, and the fibroblasts formed a dense layer over the titanium surface at that time.

References

- **1** Lindhe, J., Wennström, J.L., and Berglundh, T. (2008). The mucosa at teeth and implants. Chapter 3. In: *Clinical Periodontology and Implant Dentistry*, 5e (ed. J. Lindhe, N.P. Lang, and T. Karring), 69–85. Blackwell Munksgaard.
- **2** Bosshardt, D.D. and Lang, N.P. (2005). The junctional epithelium: from health to disease. *Journal of Dental Research* 84 (1): 9–20.
- **3** Sculean, A., Gruber, R., and Bosshardt, D.D. (2014). Soft tissue wound healing around teeth and dental implants. *Journal of Clinical Periodontology* 41 (Suppl 15): S6–S22.
- **4** Berglundh, T., Lindhe, J., Ericsson, I. et al. (1991). The soft tissue barrier at implants and teeth. *Clinical Oral Implants Research* 2 (2): 81–90.
- **5** Buser, D., Weber, H.P., Donath, K. et al. (1992). Soft tissue reactions to non-submerged unloaded titanium implants in beagle dogs. *Journal of Periodontology* 63: 225–235.
- **6** Abrahamsson, I., Berglundh, T., Wennstrom, J., and Lindhe, J. (1996). The peri-implant hard and soft tissues at different implant systems. A comparative study in the dog. *Clinical Oral Implants Research* 7: 212–219.
- **7** Arvidson, K., Fartash, B., Hilliges, M., and Kondell, P.A. (1996). Histological characteristics of peri-implant mucosa around Branemark and single-crystal sapphire implants. *Clinical Oral Implants Research* 7: 1–10.

From 6 to 12 weeks, maturation of the soft connective tissue had occurred, and the peri-implant junctional epithelium occupied about 60% of the entire implant soft tissue interface. Further away from the implant surface, the number of blood vessels was low, and fibroblasts were located between thin collagen fibers running mainly parallel to the implant surface. The findings indicated that the soft tissue adherance to transmucosal (i.e., non-submerged) implants made of commercially pure titanium with a polished surface in the neck portion requires at least six weeks [25]. The findings from animals were corroborated also in humans by Tomasi et al. [26], indicating that a soft tissue barrier adjacent to titanium implants may form completely within eight weeks. Further studies provided evidence indicating that in animals (i.e., dogs), the dimensions of the soft tissue seal (i.e., the biological width or supracrestal soft tissue) around implants are stable for at least 12 [13, 27] or 15 months, respectively [28].

In conclusion, soft peri-implant mucosa is a physiological barrier between the oral mucosa and peri-implant bone. It is the protective core of the implant surrounding bone and the anatomical structure promoting resistance to functional loads and responsible for the immunological hosttissue response.

- **8** Abrahamsson, I., Berglundh, T., Moon, I.S., and Lindhe, J. (1999). Peri-implant tissues at submerged and nonsubmerged titanium implants. *Journal of Clinical Periodontology* 26: 600–607.
- **9** Weber, H.P., Buser, D., Donath, K. et al. (1996). Comparison of healed tissues adjacent to submerged and non-submerged unloaded titanium dental implants. A histometric study in beagle dogs. *Clinical Oral Implants Research* 7: 11–19.
- **10** Berglundh, T. and Lindhe, J. (1996). Dimension of the periimplant mucosa. Biological width revisited. *Journal of Clinical Periodontology* 23: 971–973.
- **11** Hermann, J.S., Buser, D., Schenk, R.K. et al. (2001). Biologic Width around one- and two-piece titanium implants. *Clinical Oral Implants Research* 12: 559–571.
- **12** Pontes, A.E., Ribeiro, F.S., Iezzi, G. et al. (2008). Biologic width changes around loaded implants inserted in different levels in relation to crestal bone: histometric evaluation in canine mandible. *Clinical Oral Implants Research* 19: 483–490.
- **13** Cochran, D.L., Hermann, J.S., Schenk, R.K. et al. (1997). Biologic width around titanium implants. A histometric analysis of the implanto-gingival junction around unloaded and loaded nonsubmerged implants in the

canine mandible. *Journal of Periodontology* 68: 186–198.

- **14** Abrahamsson, I., Zitzmann, N.U., Berglundh, T. et al. (2001). Bone and soft tissue integration to titanium implants with different surface topography: an experimental study in the dog. *The International Journal of Oral & Maxillofacial Implants* 16: 323–332.
- **15** Abrahamsson, I., Zitzmann, N.U., Berglundh, T. et al. (2002). The mucosal attachment to titanium implants with different surface characteristics: an experimental study in dogs. *Journal of Clinical Periodontology* 29: 448–455.
- **16** Glauser, R., Schüpbach, P., Gottlow, J., and Hämmerle, C.H. (2005). Periimplant soft tissue barrier at experimental one-piece mini-implants with different surface topography in humans: a light-microscopic overview and histometric analysis. *ClinicalImplant Dentistryand RelatedResearch* 7 (Suppl 1): S44–S51.
- **17** Watzak, G., Zechner, W., Tangl, S. et al. (2006). Soft tissue around three different implant types after 1.5 years of functional loading without oral hygiene: a preliminary study in baboons. *Clinical Oral Implants Research* 17: 229–236.
- **18** Rossi S, Tirri T, Paldan H, Kuntsi-Vaattovaara H, Tulamo R, Närhi T. (2008). Peri-implant tissue response to $TiO₂$ surface modified implants. *Clinical Oral Implants Research* 19 (4): 348–355.
- **19** Schwarz, F., Sculean, A., Wieland, M. et al. (2007). Effects of hydrophilicity and microtopography of titanium implant surfaces on initial supragingival plaque biofilm formation. A pilot study. *Mund- Kiefer- und Gesichtschirurgie* 11 (6): 333–338.
- **20** Listgarten, M.A., Buser, D., Steinemann, S.G. et al. (1992). Light and transmission electron microscopy of the intact interfaces between non-submerged titanium-coated epoxy resin implants and bone or gingiva. *Journal of Dental Research* 71: 364–371.
- **21** Listgarten, M.A. (1996). Soft and hard tissue response to endosseous dental implants. *The Anatomical Record* 245: 410–425.
- **22** Chavrier, C., Couble, M.L., and Hartmann, D.J. (1994). Qualitative study of collagenous and noncollagenous glycoproteins of the human healthy keratinized mucosa surrounding implants. *Clinical Oral Implants Research* 5: 117–124.
- **23** Romanos, G.E., Schroeter-Kermani, C., Weingart, D., and Strub, J.R. (1995). Healthy human periodontal versus peri-implant gingival tissues: an immunohistochemical differentiation of the extracellular matrix. *The International Journal of Oral & Maxillofacial Implants* 10: 750–758.
- **24** Romanos, G.E., Schroeter-Kermani, C., and Strub, J.R. (1996). Inflamed human periodontal versus periimplant gingival tissues: an immunohistochemical differentiation of the extracellular matrix. *The International Journal of Oral & Maxillofacial Implants* 11 (5): 605–611.
- **25** Berglundh, T., Abrahamsson, I., Welander, M. et al. (2007). Morphogenesis of the peri-implant mucosa: an experimental study in dogs. *Clinical Oral Implants Research* 18: 1–8.
- **26** Tomasi, C., Tessarolo, F., Caola, I. et al. (2013). Morphogenesis of peri-implant mucosa revisited: an experimental study in humans. *Clinical Oral Implants Research* 25: 997–1003.
- **27** Assenza, B., Scarano, A., Petrone, G. et al. (2003). Crestal bone remodeling in loaded and unloaded implants and the microgap: a histologic study. *Implant Dentistry* 12: 235–241.
- **28** Hermann, J.S., Buser, D., Schenk, R.K. et al. (2000). Biologic width around titanium implants. A physiologically formed and stable dimension over time. *Clinical Oral Implants Research* 11: 1–11.

Prevalence and Risk Factors for Peri-implant Diseases: The Global Diseases

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Peri-implant disease is classified into two types: peri implant mucositis and peri-implantitis [1, 2]. The definitions of peri-implant diseases were first proposed at the first European Workshop on Periodontology [1]. Peri-implant mucositis was defined as a reversible inflammatory reaction in the soft tissues surrounding a functioning implant, and peri-implantitis was described as inflammatory reactions associated with loss of supporting bone around an implant in function [1]. According to the diagnostic criteria suggested in the Consensus of the Seventh EuropeanWorkshop on Periodontology, peri-implant mucositis was defined as bleeding on gentle probing($\langle 0.25 \text{ N} \rangle$, and peri-implantitis is characterized by changes in the level of the crestal bone in conjunction with bleeding on probing with or without concomitant deepening of peri-implant pockets [3, 4]. The Consensus of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions presented the main clinical characteristic of peri-implant mucositis as bleeding on gentle probing [5]. Erythema, swelling, and/or suppuration may also be present (Figures 2.1–2.3). Peri-implantitis was defined as a plaque associated pathological condition occurring in tissues around dental implants, characterized by inflammation in the peri-implant mucosa and subsequent progressive loss of supporting bone [5] (Figures 2.4–2.6).

Implants are designed to function over long term [6], yet many studies suggest that the prevalence of peri-implant disease is higher than expected [7–9]. In 2013, Atieh et al. reported in a systematic review and meta-analysis that the summary estimates for the frequency of peri-implant mucositis were 63.4%of participants and 30.7%of implants, and those of peri-implantitis were 18.8% of participants and 9.6% of implants [8]. Another systematic review from 2015 reported the prevalence of peri-implant disease ranged from 19 to 65% for peri-implant mucositis and from 1 to 47% for peri-implantitis [7]. Since there is evidence

suggesting that peri-implant mucositis is the precursor of peri-implantitis [10, 11], and the progression of peri implantitis appears to be faster than that observed in periodontitis [5], it is essential to better understand peri-implant diseases and implement preventive strategies [12].

Prevalence of Peri-implant Mucositis and Peri-implantitis

Several recent studies have provided more information about the epidemiology of peri-implant diseases in different geographical areas as shown in Table 2.1 [3, 13–18]. The prevalence of peri-implant mucositis ranged from 23.9 to 54.5% at patient-level and from 27 to 69.2% at implant level, and those of peri-implantitis ranged from 9.7 to 56.6% at patient-level and from 9.2 to 27.9% at implant-level [3, 14–18].

Variability in the prevalence of peri-implant disease might be attributable to differences between studies with different study designs, limited convenience sample, and unstandardized diagnostic criteria for peri-implant diseases. A retrospective cross-sectional study from India reported the prevalence of peri-implant mucositis to be 0.33%, which is evidently lower than various other studies done by other authors [19]. Besides, French et al. reported that the prevalence of mucositis varied from 18.2% using the "relaxed" bleeding-on-probing scores threshold (moderate multipoint bleeding) to 49.5% using a "strict" bleeding on-probing scores threshold (minimal single-point bleeding) [13], and the prevalence of peri-implantitis (combination of mucositis and marginal bone loss ≥1.0mm) varied from 3.6% using relaxed criteria to 4.7% using strict criteria, which means the prevalence of peri-implant diseases differed depending on the corresponding definition [13, 14]. Interestingly, recent studies have defined an

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Figures 2.1–2.3 A case of peri-implant mucositis: the presence of inflammation in the mucosa at an implant with no sign of loss of supporting bone.

intermediate peri-implant health category between peri implant mucositis and peri-implantitis; for example, Romandini et al. denominated peri-implant mucositis as presence of bleeding and suppuration on probing together with radiographic bone levels(BL) <1mm, pre-periimplantitis as presence of bleeding and suppuration on probing together with 1mm≦BL<2mm, and peri implantitis as presence of bleeding and suppuration on probing together with radiographic BL \geq 2mm [14]. Under this definition, the prevalence (at patient-level) of peri implant mucositis was 11.1%, of pre-peri-implantitis was 31.3%, and of peri-implantitis was 56.6%; the prevalence (at implant-level) of peri-implant mucositis was 31.9%, of pre peri-implantitis was 31.7%, and of peri-implantitis was 27.9% [14]. Therefore, epidemiological studies with a cross sectional design, adequate sample sizes, and standardized diagnostic criteria are necessary to study the prevalence and risk indicators of peri-implant diseases.

Risk Factors of Peri-implant Mucositis and Peri-implantitis

Many factors, such as diabetes mellitus [20], poor oral hygiene [20, 21], periodontitis [20–22], smoking [23–26], implant surface roughness [23], augmentation at the implant site [23], type of dentition [23], bacterial microleakage at the implant–abutment interface [27], lack of keratinized mucosa [27], residual excess cement [28], implant location [21], and prosthetic design [29] were described as factors enhancing the risk of peri-implant disease. There are limited data available to support systemic conditions as risk indicators for peri-implant mucositis and peri-implantitis; besides, many of these studies have not included all these factors in their statistical analysis.

As shown in Table 2.1, a history/presence of periodontitis, smoking, systemic disease, poor oral hygiene/plaque control record (PCR) (>20%), keratinized tissue width

Figures 2.4–2.6 A case of peri-implantitis: the presence of inflammation in the peri-implant mucosa and loss of supporting bone.

(<2mm), implant position (maxilla), surgical procedure (two-stage), frequency of maintenance, number of dental implants, and surgical technique (two-stage) [3, 15, 17, 18] were reported as risk factors for peri-implant mucositis recently. While the cross-sectional study from Spain using a network of sentinel dentists reported that none of the studied variables (including length and diameter of implants, surface roughness, keratinized tissue-mid buccal, and prosthesis) were significantly associated at univariate or multivariate level with mucositis [15]. As for peri-implantitis, a history/presence of periodontitis, smoking, poor oral hygiene/PCR (>20%), keratinized tissue

width (<2mm), implant position/location (maxilla), surgical procedure (two-stage), number of dental implants, diameter and surface of dental implants, type of prosthesis, <16 remaining teeth, implant brand, restoration type (bridge versus single crown), supportive therapy, trauma as reason of tooth loss (versus caries), autoimmune disease, gender, and bisphosphonate use [3, 13–18] were reported as risk factors for peri-implantitis recently (Table 2.1). Among those studies, the university-representative cross sectional study from Spain indicated that interproximal flossing/brushing, proton pump inhibitors, and anticoagulants were protective indicators for peri-implantitis [14].

Table 2.1 Prevalence and risk indicators of peri-implant diseases.

(*Continued*)

Table 2.1 (Continued)

Systemic Risk Factors

Periodontitis

Periodontitis (history or presence of periodontitis) was described as factors enhancing the risk of peri-implant disease in many studies [3, 17, 18, 20–22]. Gunpinar et al. [18] recently revealed the importance of current periodontal status as a risk indicator. A history of periodontitis was also indicated as a strong risk factor for peri-implant diseases in a recent cross-sectional multicenter study from Japan, and the prevalence of peri-implant mucositis may be related to the severity of periodontitis [3]. In their study, periodontal condition before implant treatment was classified into six groups: healthy, gingivitis, slight chronic periodontitis (CP), moderate CP, severe CP, and aggressive periodontitis. Among patients with slight CP, moderate CP, or severe CP before implant treatment, 17.8, 34.5, and 43.9% had peri implant mucositis and 11, 8.6, and 10.6% had peri implantitis [3]. While Wada et al. [17] did not find an association between the presence of periodontitis and peri implant disease because of strong multicollinearity of the presence of periodontitis and the PCR.

Smoking

Smoking has also been highlighted as a strong risk indicator for peri-implant disease [23–26]. A higher frequency of occurrence of peri-implant diseases was recorded for smokers in a systematic review and meta-analysis of nine studies with 1497 participants and 6283 implants included [8]. In contrast, studies by Romanos et al. showed that no negative impact of heavy smoking on implant failure and no significant difference in the bleeding on probing or crestal bone loss when implants have platform switching and are placed in one-stage approach, loaded with the final abutment at the day of surgery [30]. Recently significant correlations were found between smoking and both peri-implant mucositis and peri implantitis in a multicenter retrospective study in Japan [17]. While in another recent study among Japanese patients, among the 22 smokers, 8 (36.4%) had peri implant mucositis, but none had peri-implantitis (0%); among the 52 former smokers, 17 (32.7%) had peri implant mucositis, and 4 had peri-implantitis (8%); and among the 193 nonsmokers, 64 (33.2%) had peri-implant mucositis, and 22 had peri-implantitis (11.4%), which suggest that smoking is not associated with development of peri-implant diseases [3]. Those controversial conclusions might be interpreted as a "masking effect" due to periodontitis [14], and another university-representative cross-sectional study in 2020 by Romandini et al. found that smoking had stronger association with peri implantitis than periodontitis [14].

Systemic Diseases

A four-year retrospective analysis reported that odds ratio for an implant failure was most clearly elevated for diabetes [31], whereas another patient cohort found no association between diabetes mellitus and implant survival [26]. The impact of systemic diseases on peri-implant mucositis is still controversial. Gunpinar et al. recently reported that patients with systemic diseases (cardiovascular disease, hypertension, and diabetes) have increased risk for mucositis (OD = 2.1) and peri-implantitis (OD = 3.7) development than patients who were systemically healthy [18], while Monje et al. found that patients with diabetes were at high risk for peri-implantitis development but not for peri implant mucositis [32]. Autoimmune disease and bisphosphonate therapy were recently found as significant risk indicators for marginal bone loss [13].

Gender

It remains unclear whether and how gender acts as risk indicator for peri-implant disease. A previous retrospective analysis did not identify any significant contribution of gender to the presence of peri-implantitis [33]. Interestingly, according to two cross-sectional studies in 2018, statistically significant associations for peri-implantitis were found for gender(male gender being protective) by Rodrigo et al. [15]; meanwhile, Matarazzo et al. reported gender male to be significantly associated with the event peri-implantitis [16].

Local Risk Factors

Plaque

Based on the data available, it is clearly demonstrated that poor oral hygiene [3] were strong risk factors for peri implant disease, and plaque accumulation for peri-implant mucositis [11 17] and peri-implantitis, respectively [14, 17]. According to the Consensus of the 2017, World Workshop on the Classification of Periodontal and Peri- Implant Diseases and Conditions, there is strong evidence from animal and human experimental studies that plaque is the etiological factor for peri-implant mucositis, and peri-implantitis is a plaque-associated pathological condition [5]. In a cross-sectional analysis of 512 implants in 2015, plaque (odds ratio [OR], 8.415) was significantly correlated with the event peri-implant mucositis, and when further evaluating the study variables at the patient level, it was noted that PI scores \geq 33% were significantly associated with the event peri-implant mucositis and peri implantitis [34]. A significant correlation was also found between PCR (>20%) with both peri-implant mucositis and peri-implantitis by a recent univariate analysis [17].

These results suggest that a high plaque score increases the risk of developing peri-implant diseases.

Dimensions of Keratinized Tissue

It remains unclear whether a zone of keratinized mucosa is required to maintain the health of peri-implant tissue. According to a systematic review in 2013, a lack of adequate keratinized mucosa around implants is associated with more plaque accumulation, tissue inflammation, mucosal recession, and attachment loss [35]. And evidence for surface roughness, residual cement, the dimension of the keratinized tissue and time of implant in function was reported weak as a risk indicator for peri-implant mucositis from another systematic literature review in 2014 [11]. Recently, Sadiye et al. reported that implants with keratinized tissue <2mm have greater change for peri-implant mucositis (OD = 5.38) and peri-implantitis (OD = 8.01) development, respectively, than implants having keratinized tissue \geq 2mm [18]. According to studies by Romanos et al., a wide band of KM (KM width \geq 2mm) was associated with a significant lower bleeding index, plaque index, and mucosal recession (MR) [36]. A significant correlation was also found between peri-implant diseases and keratinized tissue width $\left($ <2mm) by univariate analysis in another study [17]. Interestingly, if the keratinized tissue width was adequate, smoking was not considered as a risk indicator for peri-implantitis development [17]; and the "regular" and "irregular" implant maintenance therapy did not reject the protective role of KM against inflammation neither [36]. Results from these studies suggested that inadequate keratinized tissue is a controversial risk indicator for peri-implant diseases and keratinized tissue <2mm may induce an indirect correlation with soft tissue health.

Prosthetic Concepts

Plaque accumulation and poor oral hygiene around dental implants have been listed as major risk factors for peri implant diseases [3, 11, 17]. In a recent review exploring the factors associated with implant restorative design and risks for peri-implant diseases, Dixon and London proposed that prosthetic designs may also be associated with peri-implant disease if the prosthesis obstructshygiene [29]. For example, bacterial penetration was observed at the implant–abutment interface and [37] and the implant– abutment interface dictated peri-implant inflammatory cell accumulation for two-piece implants [38]. According to another study investigating the use of implants designed with a long smooth hyperbolic neck placed in a transmucosal configuration, neck exposure during the healing time displayed limited plaque accumulation and was free from complications such as gingivitis, mucositis, and implant failures during the healing time [39]. Similarly, the

association of the restoration type (bridges versus single crowns) with peri-implantitis has also been reported by Rodrigo et al. in 2018 [15] and by Romandini et al. in 2020 [14], which may also be explained by the more difficult access to oral hygiene procedures [15]. The association between cemented fixed prosthesis and peri-implantitis found here has already been identified [16, 34] and explained by the presence of excess cement in the peri implant sulcus [34]. From another point of view, undesirable emergence profiles may be crucial for the plaque accumulation, poor oral hygiene, and thus peri-implant diseases [29]. Dixon and London advised a subtle, vertical emergence pattern that most closely resembles the natural tooth emergence contours, which would be amenable to probing and hygiene instrumentation [29].

There are also studies suggesting that some restorative designs have no significant effect on peri-implant diseases. For example, implant design and prosthesis type were not significantly associated with peri-implant diseases in a recent multicenter retrospective study analyzing associations of implant design (soft-tissue level or bone level) and prosthesis type (screw- or cement-retained) with peri implant diseases [3]. Interestingly, there were different conclusions in peri-implant mucositis and peri-implantitis. As Rodrigo et al. reported, none of the studied variables (including length and diameter of implants, surface roughness, keratinized tissue-mid-buccal, and prosthesis) were significantly associated at univariate or multivariate level with peri-implant mucositis, while significant associations for peri-implantitis were found for type of prosthesis and access to interproximal plaque control [15]. A significant correlation was found between peri-implant disease and implant position (maxilla) and surgical procedure (two stage) by a univariate analysis [17]. Therefore, prosthetic design allowing for daily hygiene may play a crucial role in peri-implant diseases in some conditions.

Implant Factors

Implant diameter [13, 15], implant location [13, 15, 17], and implant brand [14] have been listed as a possible factor for peri-implant diseases recently. Different studies have listed the location of the maxillary [17], anterior [13], and lower anterior [15] implants as factors for crestal bone loss or peri-implantitis. In a multiple regression analysis, statistically significant associations for peri-implantitis were found for implant diameter [15], in which higher risk for diameters of 3.3mm was reported based on a study performed in private practice. Significant risk indicators for bone loss were also found to include implant location, diameter, and design recently [13], whereby an increase of 1mm in diameter was associated with a decrease in crestal bone loss by about 0.11mm.

Additionally, different surface characteristics (implants/ abutments) may play a role in plaque accumulation, the overall evidence is weak. Some scholars proposed that microbial formation may be related more to the individual themselves rather than the material being used [40]. Some studies suggest that certain materials (e.g. zirconium) may be more resistant to bacterial colonization than pure titanium with the same surface roughness [41] and gold–platinum alloy appears to be useful material for abutments when considering the accumulation of plaque [40]. Besides, a review in 2011 has shown that based on the limited data available, there is no evidence that implant surface characteristics can have a significant effect on the initiation of peri-implant diseases [42]. Another study also validated the assumption of a significantly higher bacterial adhesion to rough titanium (e.g. sandblasted and acid-etched) than to purely machined, and therefore the micro-structured parts of an implant that are exposed to the oral cavity should be highly polished to prevent plaque accumulation and peri-implant disease [43]. While a university representative cross-sectional study in 2018 have not found significant association between surface roughness and mucositis at univariate or multivariate level [15].

Overall, the prevalence of peri-implant mucositis ranged from 23.9 to 54.5% at patient-level and from 27 to 69.2% at implant-level, and those of peri-implantitis ranged from 9.7 to 56.6% at patient-level and from 9.2 to 27.9% at implant-level according to recent studies in different geographical areas. Variability in the prevalence of peri-implant disease might be attributable to differences between studies with different study designs, limited convenience sample, and unstandardized diagnostic criteria for peri-implant diseases. Periodontitis may have association with the plaque and may mask the influence of smoking. A common finding was that peri-implant disease can primarily be attributed to plaque accumulation. A potential factor for causing inadequate hygiene measures may come from the implant prosthesis itself. Surface characteristics or width of keratinized tissue may not directly relate to peri-implant diseases, but relate more to plaque accumulation and thus indirectly affect the peri implant diseases. There is a need for more adequately powered prospective longitudinal studies to try and identify the epidemiology and potential risk indicators for peri-implant diseases.

References

- **1** Zitzmann, N.U. and Berglundh, T. (2008). Definition of peri-implant diseases. *Journal of Clinical Periodontology* 35 (8 Suppl): 286–291.
- **2** Khammissa, R., Feller, L., Meyerov, R. et al. (2012). Peri-implant mucositis and peri-implantitis: clinical and histopathological characteristics and treatment. *South African Dental Journal* 67 (3): 124–126.
- **3** Ogata, Y., Nakayama, Y., Tatsumi, J. et al. (2017). Prevalence and risk factors for peri-implant diseases in Japanese adult dental patients. *Journal of Oral Science* 59 $(1): 1-11.$
- **4** Lang, N.P., Berglundh, T., and Working Group 4 of Seventh European Workshop on Periodontology (2011). Periimplant diseases: where are we now? – Consensus of the Seventh European Workshop on Periodontology. *Journal of Clinical Periodontology* 38 (s11): 178–181.
- **5** Berglundh, T., Armitage, G., Araujo, M.G. et al. (2018). 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. *British Dental Journal* 45 (Suppl 20): S286–S291.
- **6** Horikawa, T., Odatsu, T., Itoh, T. et al. (2017). Retrospective cohort study of rough-surface titanium implants with at least 25years' function. *International Journal of Implant Dentistry* 3 (1): 42.
- **7** Derks, J. and Tomasi, C. (2015). Peri-implant health and disease. A systematic review of current epidemiology. *Journal of Clinical Periodontology* 42: S158.
- **8** Atieh, M.A., Alsabeeha, N.H., Faggion, C.M. Jr. et al. (2013). The frequency of peri-implant diseases: a systematic review and meta-analysis. *Journal of Periodontology* 84 (11): 1586.
- **9** Fransson, C., Lekholm, U., Jemt, T. et al. (2010). Prevalence of subjects with progressive bone loss at implants. *Clinical Oral Implants Research* 16 (4): 440–446.
- **10** Costa, F.O., Takenaka-Martinez, S., Cota, L.O. et al. (2012). Peri-implant disease in subjects with and without preventive maintenance: a 5-year follow-up. *Journal of Clinical Periodontology* 39 (2): 173–181.
- **11** Renvert, S. and Polyzois, I. (2015). Risk indicators for peri-implant mucositis: a systematic literature review. *Journal of Clinical Periodontology* 42: S172.
- **12** Berglundh, T., Armitage, G., Araujo, M.G. et al. (2018). 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* 89 (Suppl 1): S313–s318.
- **13** French, D., Grandin, H.M., and Ofec, R. (2019). Retrospective cohort study of 4,591 dental implants: analysis of risk indicators for bone loss and prevalence of

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peri-implant mucositis and peri-implantitis. *Journal of Periodontology* 90 (7): 691–700.

- **14** Romandini, M., Lima, C., Pedrinaci, I. et al. (2021). Prevalence and risk/protective indicators of peri-implant diseases: a university-representative cross-sectional study. *Clinical Oral Implants Research* 32 (1): 112–122.
- **15** Rodrigo, D., Sanz-Sánchez, I., Figuero, E. et al. (2018). Prevalence and risk indicators of peri-implant diseases in Spain. *Journal of Clinical Periodontology* 45 (12): 1510–1520.
- **16** Matarazzo, F., Sabóia-Gomes, R., Alves, B.E.S. et al. (2018). Prevalence, extent and severity of peri-implant diseases. A cross-sectional study based on a university setting in Brazil. *Journal of Periodontal Research* 53 (5): 910–915.
- **17** Wada, M., Mameno, T., Onodera, Y. et al. (2019). Prevalence of peri-implant disease and risk indicators in a Japanese population with at least 3 years in function-a multicentre retrospective study. *Clinical Oral Implants Research* 30 (2): 111–120.
- **18** Gunpinar, S., Meraci, B., and Karas, M. (2020). Analysis of risk indicators for prevalence of peri-implant diseases in Turkish population. *International Journal of Implant Dentistry* 6 (1): 19.
- **19** Labh, A.K., Bennis, M.A., and Mani, G. (2021). Prevalence of peri-implantitis and peri-implant mucositis among implant patients: a Dental University based study. *Journal of Long-Term Effects of Medical Implants* 31 $(1): 59-64.$
- **20** Ferreira, S.D., Silva, G.L., Cortelli, J.R. et al. (2006). Prevalence and risk variables for peri-implant disease in Brazilian subjects. *Journal of Clinical Periodontology* 33 (12): 929–935.
- **21** Aguirre-Zorzano, L.A., Estefanía-Fresco, R., Telletxea, O. et al. (2015). Prevalence of peri-implant inflammatory disease in patients with a history of periodontal disease who receive supportive periodontal therapy. *Clinical Oral Implants Research* 26 (11): 1338–1344.
- **22** Roccuzzo, M., Bonino, F., Aglietta, M. et al. (2012). Ten-year results of a three arms prospective cohort study on implants in periodontally compromised patients. Part 2: clinical results. *Clinical Oral Implants Research* 23 (4): 389–395.
- **23** Karbach, J., Callaway, A., Kwon, Y.D. et al. (2009). Comparison of five parameters as risk factors for peri-mucositis. *The International Journal of Oral & Maxillofacial Implants* 24 (3): 491–496.
- **24** Rinke, S., Ohl, S., Ziebolz, D. et al. (2011). Prevalence of periimplant disease in partially edentulous patients: a practice-based cross-sectional study. *Clinical Oral Implants Research* 22 (8): 826–833.
- **25** Rinke, S., Roediger, M., Eickholz, P. et al. (2015). Technical and biological complications of single-molar

implant restorations. *Clinical Oral Implants Research* 26 (9): 1024–1030.

- **26** Anner, R., Grossmann, Y., Anner, Y. et al. (2010). Smoking, diabetes mellitus, periodontitis, and supportive periodontal treatment as factors associated with dental implant survival: a long-term retrospective evaluation of patients followed for up to 10 years. *Implant Dentistry* 19 (1): 57–64.
- **27** Koutouzis, T., Koutouzis, G., Gadalla, H. et al. (2013). The effect of healing abutment reconnection and disconnection on soft and hard peri-implant tissues: a short-term randomized controlled clinical trial. *The International Journal of Oral & Maxillofacial Implants* 28 (3): 807–814.
- **28** Linkevicius, T., Puisys, A., Vindasiute, E. et al. (2013). Does residual cement around implant-supported restorations cause peri-implant disease? A retrospective case analysis. *Clinical Oral Implants Research* 24 (11): 1179–1184.
- **29** Dixon, D.R. and London, R.M. (2019). Restorative design and associated risks for peri-implant diseases. *Periodontology 2000* 81 (1): 167–178.
- **30** Romanos, G.E., Gaertner, K., Aydin, E. et al. (2013). Long-term results after immediate loading of platform switched implants in smokers versus nonsmokers with full-arch restorations. *The International Journal of Oral & Maxillofacial Implants* 28 (3): 841–845.
- **31** Zupnik, J., Kim, S.W., Ravens, D. et al. (2011). Factors associated with dental implant survival: a 4-year retrospective analysis. *Journal of Periodontology* 82 (10): 1390–1395.
- **32** Monje, A., Catena, A., and Borgnakke, W.S. (2017). Association between diabetes mellitus/hyperglycaemia and peri-implant diseases: systematic review and meta-analysis. *Journal of Clinical Periodontology* 44 (6): 636–648.
- **33** Renvert, S., Aghazadeh, A., Hallström, H. et al. (2014). Factors related to peri-implantitis – a retrospective study. *Clinical Oral Implants Research* 25: 522–529.
- **34** Schwarz, F., Becker, K., Sahm, N. et al. (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. *Clinical Oral Implants Research* 28 (1): 24–28.
- **35** Lin, G.H., Chan, H.L., and Wang, H.L. (2013). The significance of keratinized mucosa on implant health: a systematic review. *Journal of Periodontology* 84 (12): 1755–1767.
- **36** Romanos, G., Grizas, E., and Nentwig, G.H. (2015). Association of keratinized mucosa and periimplant soft tissue stability around implants with platform switching. *Implant Dentistry* 24 (4): 422–426.
- **37** Piattelli, A., Scarano, A., Paolantonio, M. et al. (2001). Fluids and microbial penetration in the internal part of cement-retained versus screw-retained implant-abutment connections. *Journal of Periodontology* 72 (9): 1146–1150.
- **38** Broggini, N., LM, M.M., Hermann, J.S. et al. (2006). Peri-implant inflammation defined by the implant abutment interface. *Journal of Dental Research* 85 (5): 473–478.
- **39** Prati, C., Zamparini, F., Canullo, L. et al. (2020). Factors affecting soft and hard tissues around two-pies transmucosal implants: a 3-year prospective cohort study. *The International Journal of Oral & Maxillofacial Implants* 35 (5): 1022–1036.
- **40** Yamane, K., Ayukawa, Y., Takeshita, T. et al. (2013). Bacterial adhesion affinities of various implant

abutment materials. *Clinical Oral Implants Research* 24 (12): 1310–1315.

- **41** Scarano, A., Piattelli, M., Caputi, S. et al. (2004). Bacterial adhesion on commercially pure titanium and zirconium oxide disks: an in vivo human study. *Journal of Periodontology* 75 (2): 292–296.
- **42** Renvert, S., Polyzois, I., and Claffey, N. (2011). How do implant surface characteristics influence peri-implant disease? *Journal of Clinical Periodontology* 38 (Suppl 11): 214–222.
- **43** Bürgers, R., Gerlach, T., Hahnel, S. et al. (2010). In vivo and in vitro biofilm formation on two different titanium implant surfaces. *Clinical Oral Implants Research* 21 (2): 156–164.

3

Seyed Hossein Bassir1 and Georgios E. Romanos2 **Significance of Radiographic Findings for the Long-term Success of Dental Implants**

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Introduction

Replacement of missing teeth using implant–supported prostheses is a widely accepted treatment modality [1–5]. Although survival rates of greater than 90–95% have been for dental implants [1–5], the survival of implants is not equal to a treatment success [6, 7]. Rather, the long-term success of the therapy depends on several factors that affect the stability of the implant–prosthetic complex such as health and stability of peri-implant soft and hard tissues [6–10]. One of the key elements in maintaining the stability of peri-implant tissue is early detection of any peri-implant soft and hard tissue changes [11, 12]. Longterm and regular maintenance care has been shown to be essential for maintaining the health and stability of periimplant soft and hard tissues [13–16]. Radiographs are used to evaluate and monitor the hard-tissue levels around implants at regular maintenance visits. In this chapter, the applications of radiographs in the maintenance of health and stability of peri-implant tissues are outlined. In this regard, the factors affecting marginal bone levels as well as type and frequency of radiographs for monitoring the health and stability of peri-implant tissues are discussed.

Peri-implant Bone Levels

Stability of marginal peri-implant bone levels is crucial for successful long-term treatment outcomes [17, 18], and continuous crestal bone loss results in the failure of implant therapy [19–21]. The reasons for crestal bone loss around dental implants are still debated, but in general, bone loss can occur due to several reasons, such as lack of

osseointegration, trauma from surgery, bone remodeling following loading, or peri-implantitis [21–25].

Lack of osseointegration results in early implant failure that occurs during the healing period prior to the implant loading. Several factors, such as infection, poor primary stability, or impaired wound healing can lead to the early implant failure [26–30]. On radiographs, lack of osseointegration can be diagnosed by the presence of continuous radiolucency around the implant (Figure 3.1). To rule out early implant failure, it is recommended to obtain a periapical radiograph prior to the second-stage surgery or prior to final impression for one-stage implants. The treatment for the early implant failure is the removal of failed implant.

Trauma from the surgery can result in crestal bone loss or lack of osseointegration [19, 22, 28, 31]. The trauma can be a result of overheating of bone during the surgery [32–34] or excessive insertion torque [35–37]. The excessive heat generated at the time of surgery causes a certain degree of bone necrosis [33, 38]. Poor irrigation or lack of irrigation during the implant surgery can cause thermally induced bone necrosis. Hence, the bone temperature must not exceed 44–47 °C during the site preparation [33]. In addition, excessive insertion torque can also lead to bone necrosis and result in peri-implant crestal bone loss [35–37]. Bone necrosis as a result of surgical trauma can be detected on radiographs as peri-implant bone loss of greater than 2mm before the loading of the implant (Figure 3.2). The treatment for these lesions depends on the degree of the bone loss and shape of the defect, and it can be implanting removal or bone grafting around the implant.

Bone remodeling occurs following the loading of implants, and it can result in some crestal bone loss [39–42]. two implants with early failure. Lack of osseointegration is evident by the presence of continuous radiolucency around the implants: (a) radiograph of the implant in an edentulous mandible at site # 22 four months following the placement. (b) radiograph of the implant at site # 8 three months following extraction and immediate implant placement.

Figure 3.1 Periapical radiographs of (a) (b)

months following implant placement likely due to surgical trauma: (a) radiograph of implants placed at sites # 4 and #5 at the day of the surgery. (b) crestal bone loss noted around implants # 4 and #5 three months after the placement.

It has been shown that the amount of crestal bone loss following the loading depends on several factors such as implant design [43–45], implant positioning [46–49], and location of the implant–abutment interface (microcap) [50–52]. Hence, the amount of crestal bone loss due to remodeling varies between implant different brands/systems and implant–abutment connections [42]. This bone remodeling mainly occurs during the first year following the loading (Figure 3.3) and most implants experience less than 1–2mm of bone loss during this period [53–55].

Bone loss of greater than 2mm following the loading is considered pathologic, [14, 56–58] and it is a result of periimplant diseases such as peri-implantitis (Figure 3.4), which is the main cause of implant failure after osseointegration [55, 59, 60]. Peri-implantitis is diagnosed based on

the presence of bleeding on probing and/or suppuration, increased probing depths, radiographic bone loss beyond bone level changes resulting from the initial bone remodeling following the loading [14, 58]. It has been suggested that, in clinical situations where baseline radiographs are not available, the diagnosis of peri-implantitis can be made based on the presence of bleeding on probing and/or suppuration, pocket depth of 6mm or greater, and alveolar bone loss of 3mm or greater from implant shoulder [14, 58]. Several treatment approaches have been used for the treatment of peri-implantitis [11, 55, 59–66]. In general, non-surgical mechanical treatment alone seems to be ineffective in the treatment of peri-implantitis [67–69]. A surgical approach that includes open flap debridement and surface decontamination has been shown to be more

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Figure 3.3 Bone remodeling around tissue-level and bone-level implants after one year following the loading: (a) minimal bone loss around a tissue-level implant at site # 19 one year following loading. (b) 1–2mm of bone loss noted around tissue-level implants at the mesial of implant #19 and the distal of implant # 20 one year after loading. (c) minimal bone loss around a bone-level implant at site # 28 one year after the loading. (d) a bone-level implant at site # 19 with 1–2mm bone loss after one year following the crown delivery.

effective than non-surgical approaches for the treatment of peri-implantitis [70, 71]. The decision on utilizing resective or regenerative approach following the open flap debridement is based on the morphology of peri-implant defects [72–74]. Contained three-wall and four-wall defects can be treated with regenerative approach using a combination of bone grafting materials, a barrier membrane, and/or biological factors.

Type of Radiographs

Monitoring the hard and soft tissues around dental implants is vital during maintenance care [25]. Unlike around teeth, there is no defined range of probing depth associated with peri-implant health [42, 75, 76]. However, peri-implant bone levels can be readily assessed using radiographs. Hence, radiographic evaluations are crucial not only for early diagnosis of peri-implantitis, but also in determining the therapeutic approach for treatment of peri-implantitis defects.

Parallel periapical radiographs are routinely used to monitor peri-implant hard tissue levels due to their accuracy, low radiation exposure, and cost-effectiveness [25, 77]. Nevertheless, periapical radiographs have some limitations such as two-dimensional projection of three-dimensional structures, low spatial resolution, and inability to represent the bone around buccal and lingual areas of implants (Figure 3.5) [25, 77]. In addition, periapical radiographs have been associated with relatively low sensitivity and high portion of false negative in early changes in peri-implant bone levels [25, 78–80]. Hence, the application of three-dimensional (3D) imaging such as cone-beam computed tomography (CBCT) has been advocated to monitor implants during maintenance care [81–85].

Although CBCT images provide valuable 3D volumetric data for planning the surgical placement of dental implants, application of CBCT for monitoring peri-implant hard tissue level during maintenance care is still questionable [25, 86]. The challenges for the application of CBCT for this purpose are high radiation dose and imaging artifacts that significantly affect the image quality and diagnostic value of these scans [87, 88]. High-density objects such as titanium implants and other metallic objects in the field of view result in noise and beam-hardening artifacts [88, 89]. These artifacts can mask bony defects around implants and limit visualization of buccal and lingual/palatal plates as well as bone-implant interface [25, 86].

(a)

(b)

(d)

Figure 3.4 A clinical case of peri-implantitis: (a) patient presented with clinical signs of peri-implantitis including erythema, swelling, bleeding on probing, suppuration, and increased probing depths. (b) baseline radiograph at the time of loading. (c) periapical radiograph obtained three years following the loading demonstrated more than 3mm of bone loss. (d) peri-implant defects after reflection of the flap and removal of granulation tissues.

The accuracy of periapical digital radiographs and CBCT images has been compared by several groups [77, 90, 91]. Dave and colleagues compared the accuracy of digital long cone periapical radiographs with that of limited field of view and large field of view CBCT for diagnosis of circumferential peri-implant crestal bone defects [90]. They found that periapical radiographs were more accurate in diagnosing peri-implant defects that were smaller (0.35 mm). However, there were no significant differences between periapical radiographs and CBCT scans for larger (0.675 mm) peri-implant defects [90]. In addition, a cadaver study conducted by Kuhl and colleagues compared the accuracy of intraoral periapical radiography, panoramic radiography, CBCT, and Computer Tomography (CT) for the diagnosis of peri-implant defects [91]. They created 1 mm- and 3 mmdepth two-wall, three-wall, and four-wall defects around dental implants. They reported that the highest sensitivity was found for intraoral periapical radiographs and CBCT scans compared to panoramic radiography and CT. In addition, it was found that intraoral periapical radiographs had the highest specificity for both 1 mmand 3 mm-depths defects [91]. A meta-analysis conducted on this topic compared the accuracy of different imaging techniques for the detection peri-implant defects [77]. This meta-analysis found that the sensitivity and specificity of CBCT for diagnosis of peri-implant defects were 59 and 67%, respectively. Similar values were reported for intra-oral periapical radiographs which were 60 and 59% for sensitivity and specificity, respectively. It was concluded that both CBCT and intraoral radiographs result in acceptable diagnosis accuracy for detecting peri-implant bone defects [77].

Therefore, periapical radiographs are still considered the preferred method for monitoring peri-implant bone levels during maintenance care despite their limitations.

Radiographic Intervals

There is still no clear guideline regarding the frequency of radiographs for monitoring peri-implant bone levels. Baseline radiographs need to be obtained on the day of implant placement and day of implant loading [14, 25]. In addition, obtaining one-year post-loading radiographs is recommended to monitor marginal bone level changes following the delivery of prosthesis [12, 14].

Following the implant loading, radiographs must be obtained if any clinical sign of peri-implantitis is observed at maintenance care visits [14, 58]. If peri-implantitis is diagnosed, proper treatment must be performed. Authors recommend that follow-up radiographs be obtained every six months to ensure the effectiveness of the provided treatment.

In the absence of any clinical signs of peri-implant diseases, it is recommended to obtain radiographs at one-year, three-year, and five-year recall visits following the implant loading [12, 91]. Thereafter, radiograph for monitoring the implant should be obtained every 24–36 months during recall visits.

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Figure 3.5 A clinical case demonstrating limitations of periapical radiographs. Patient is referred for evaluation of implant # 13 due to discomfort at site # 13. Implant had been placed four months ago. (a) Periapical radiograph demonstrating the crestal bone levels around # 13 are within normal limits, but a periapical radiolucency noted around tooth # 12. (b) occlusal photo demonstrating erythema around implant # 13 that is placed buccally. (c) a limited field of view CBCT was obtained to further assess implant # 13. Although, imaging artifacts affected the diagnostic quality of the scan, it was noted that the apical portion of implant # 13 is outside the alveolar housing. Therefore, removal of implant # 13 and apicoectomy for tooth # 12 was recommended. (d) surgical photo demonstrating more than 50% of implant # 13 is outside the alveolar housing. (e) implant # 13 was explanted and apicoectomy was done for tooth # 12. (f) defects were grafted using an allograft and it was covered with a resorbable collagen membrane. (g) re-entry was done four months following the surgery and an implant was placed at site # 13. (h) periapical radiograph following implant placement at site # 13 demonstrating the positioning of implant # 13 and the healing of periapical defect around tooth # 12.

References

- **1** Derks, J., Schaller, D., Håkansson, J. et al. (2016). Effectiveness of implant therapy analyzed in a Swedish population: prevalence of peri-implantitis. *Journal of Dental Research* 95 (1): 43–49.
- **2** De Boever, A.L., Quirynen, M., Coucke, W. et al. (2009). Clinical and radiographic study of implant treatment outcome in periodontally susceptible and non-susceptible patients: a prospective long-term study. *Clinical Oral Implants Research* 20 (12): 1341–1350.
- **3** Romanos, G., Froum, S., Hery, C. et al. (2010). Survival rate of immediately vs delayed loaded implants: analysis of the current literature. *The Journal of Oral Implantology* 36 (4): 315–324.
- **4** Bassir, S.H., El Kholy, K., Chen, C.Y. et al. (2019). Outcome of early dental implant placement versus other dental implant placement protocols: a systematic review and meta-analysis. *Journal of Periodontology* 90 (5): 493–506.
- **5** Schwartz-Arad, D. and Chaushu, G. (1997). Placement of implants into fresh extraction sites: 4 to 7 years retrospective evaluation of 95 immediate implants. *Journal of Periodontology* 68 (11): 1110–1116.
- **6** Papaspyridakos, P., Chen, C.J., Singh, M. et al. (2012). Success criteria in implant dentistry: a systematic review. *Journal of Dental Research* 91 (3): 242–248.
- **7** Annibali, S., Bignozzi, I., La Monaca, G., and Cristalli, M.P. (2012). Usefulness of the aesthetic result as a success criterion for implant therapy: a review. *Clinical Implant Dentistry and Related Research* 14 (1): 3–40.
- **8** Buser, D., Martin, W., and Belser, U.C. (2004). Optimizing esthetics for implant restorations in the anterior maxilla: anatomic and surgical considerations. *The International Journal of Oral & Maxillofacial Implants* 19 (7): 43–61.
- **9** Chackartchi, T., Romanos, G.E., and Sculean, A. (2019). Soft tissue-related complications and management around dental implants. *Periodontology 2000* 81 (1): 124–138.
- **10** Bassir, S.H., Alhareky, M., Wangsrimongkol, B. et al. (2018). Systematic review and meta-analysis of hard tissue outcomes of alveolar ridge preservation. *The International Journal of Oral & Maxillofacial Implants* 33 (5): 979–994.
- **11** Romanos, G.E., Javed, F., Delgado-Ruiz, R.A., and Calvo-Guirado, J.L. (2015). Peri-implant diseases: a review of treatment interventions. *Dental Clinics* 59 (1): 157–178.
- **12** Salvi, G.E. and Lang, N.P. (2004). Diagnostic parameters for monitoring peri-implant conditions. *The International Journal of Oral & Maxillofacial Implants* 19 (7): 116–127.
- **13** Klinge, B., Meyle, J., and Working Group 2 (2012). Peri-implant tissue destruction. The third EAO consensus

conference 2012. *Clinical Oral Implants Research* 23: 108–110.

- **14** Berglundh, T., Armitage, G., Araujo, M.G. et al. (2018). 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* 89: S313–S318.
- **15** Frisch, E., Vach, K., and Ratka-Krueger, P. (2020). Impact of supportive implant therapy on peri-implant diseases: a retrospective 7-year study. *Journal of Clinical Periodontology* 47 (1): 101–109.
- **16** Monje, A., Aranda, L., Diaz, K.T. et al. (2016). Impact of maintenance therapy for the prevention of peri-implant diseases: a systematic review and meta-analysis. *Journal of Dental Research* 95 (4): 372–379.
- **17** Clementini, M., Rossetti, P.H., Penarrocha, D. et al. (2014). Systemic risk factors for peri-implant bone loss: a systematic review and meta-analysis. *International Journal of Oral and Maxillofacial Surgery* 43 (3): 323–334.
- **18** Vervaeke, S., Collaert, B., Cosyn, J. et al. (2015). A multifactorial analysis to identify predictors of implant failure and peri-implant bone loss. *Clinical Implant Dentistry and Related Research* 17: e298–e307.
- **19** Albrektsson, T., Chrcanovic, B., Östman, P.O., and Sennerby, L. (2017). Initial and long-term crestal bone responses to modern dental implants. *Periodontology 2000* 73 (1): 41–50.
- **20** Snauwaert, K., Duyck, J., van Steenberghe, D. et al. (2000). Time dependent failure rate and marginal bone loss of implant supported prostheses: a 15-year follow-up study. *Clinical Oral Investigations* 4 (1): 13–20.
- **21** Qian, J., Wennerberg, A., and Albrektsson, T. (2012). Reasons for marginal bone loss around oral implants. *Clinical Implant Dentistry and Related Research* 14 (6): 792–807.
- **22** Oh, T.J., Yoon, J., Misch, C.E., and Wang, H.L. (2002). The causes of early implant bone loss: myth or science? *Journal of Periodontology* 73 (3): 322–333.
- **23** Chung, D.M., Oh, T.J., Lee, J. et al. (2007). Factors affecting late implant bone loss: a retrospective analysis. *The International Journal of Oral & Maxillofacial Implants* 22 (1): 117–126.
- **24** Albrektsson, T., Buser, D., and Sennerby, L. (2012). Crestal bone loss and oral implants. *Clinical Implant Dentistry and Related Research* 14 (6): 783–791.
- **25** De Bruyn, H., Vandeweghe, S., Ruyffelaert, C. et al. (2013). Radiographic evaluation of modern oral implants with emphasis on crestal bone level and relevance to peri-implant health. *Periodontology 2000* 62 (1): 256–270.
- **26** De Bruyn, H. and Collaert, B. (1994). The effect of smoking on early implant failure. *Clinical Oral Implants Research* 5 (4): 260–264.
- **27** Van Steenberghe, D., Jacobs, R., Desnyder, M. et al. (2002). The relative impact of local and endogenous patientrelated factors on implant failure up to the abutment stage. *Clinical Oral Implants Research* 13 (6): 617–622.
- **28** Sakka, S., Baroudi, K., and Nassani, M.Z. (2012). Factors associated with early and late failure of dental implants. *Journal of Investigative and Clinical Dentistry* 3 (4): 258–261.
- **29** Chrcanovic, B.R., Albrektsson, T., and Wennerberg, A. (2014). Reasons for failures of oral implants. *Journal of Oral Rehabilitation* 41 (6): 443–476.
- **30** Javed, F., Ahmed, H.B., Crespi, R., and Romanos, G.E. (2013). Role of primary stability for successful osseointegration of dental implants: factors of influence and evaluation. *Interventional Medicine and Applied Science* 5 (4): 162–167.
- **31** Kronström, M., Svenson, B., Hellman, M., and Persson, G.R. (2001). Early implant failures in patients treated with Brånemark system titanium dental implants: a retrospective study. *The International Journal of Oral & Maxillofacial Implants* 16 (2): 201–207.
- **32** Piattelli, A., Piattelli, M., Mangano, C., and Scarano, A. (1998). A histologic evaluation of eight cases of failed dental implants: is bone overheating the most probable cause? *Biomaterials* 19 (7–9): 683–690.
- **33** Eriksson, R.A. and Albrektsson, T. (1984). The effect of heat on bone regeneration: an experimental study in the rabbit using the bone growth chamber. *Journal of Oral and Maxillofacial Surgery* 42 (11): 705–711.
- **34** Aljateeli, M., Fu, J.H., and Wang, H.L. (2012). Managing peri-implant bone loss: current understanding. *Clinical Implant Dentistry and Related Research* 14: e109–e118.
- **35** Bashutski, J.D., D'Silva, N.J., and Wang, H.L. (2009). Implant compression necrosis: current understanding and case report. *Journal of Periodontology* 80 (4): 700–704.
- **36** Ikar, M., Grobecker-Karl, T., Karl, M., and Steiner, C. (2020). Mechanical stress during implant surgery and its effects on marginal bone: a literature review. *Quintessence International* 51 (2): 142–150.
- **37** Jimbo, R., Tovar, N., Anchieta, R.B. et al. (2014). The combined effects of undersized drilling and implant macrogeometry on bone healing around dental implants: an experimental study. *International Journal of Oral and Maxillofacial Surgery* 43 (10): 1269–1275.
- **38** Li, S., Chien, S., and Brånemark, P.I. (1999). Heat shock-induced necrosis and apoptosis in osteoblasts. *Journal of Orthopaedic Research* 17 (6): 891–899.
- **39** Hermann, J.S., Cochran, D.L., Nummikoski, P.V., and Buser, D. (1997). Crestal bone changes around titanium

implants. A radiographic evaluation of unloaded nonsubmerged and submerged implants in the canine mandible. *Journal of Periodontology* 68 (11): 1117–1130.

- **40** Broggini, N., McManus, L.M., Hermann, J.S. et al. (2003). Persistent acute inflammation at the implant-abutment interface. *Journal of Dental Research* 82 (3): 232–237.
- **41** Broggini, N., McManus, L.M., Hermann, J.S. et al. (2006). Peri-implant inflammation defined by the implantabutment interface. *Journal of Dental Research* 85 (5): 473–478.
- **42** Araujo, M.G. and Lindhe, J. (2018). Peri-implant health. *Journal of Periodontology* 89: S249–S256.
- **43** Bryant, S.R. and Zarb, G.A. (2003). Crestal bone loss proximal to oral implants in older and younger adults. *The Journal of Prosthetic Dentistry* 89 (6): 589–597.
- **44** Pilliar, R.M., Deporter, D.A., Watson, P.A., and Valiquette, N. (1991). Dental implant design–effect on bone remodeling. *Journal of Biomedical Materials Research* 25 (4): 467–483.
- **45** Chou, H.Y., Jagodnik, J.J., and Müftü, S. (2008). Predictions of bone remodeling around dental implant systems. *Journal of Biomechanics* 41 (6): 1365–1373.
- **46** Hartman, G.A. and Cochran, D.L. (2004). Initial implant position determines the magnitude of crestal bone remodeling. *Journal of Periodontology* 75 (4): 572–577.
- **47** Schwarz, F., Alcoforado, G., Nelson, K. et al. (2014). Impact of implant–abutment connection, positioning of the machined collar/microgap, and platform switching on crestal bone level changes. Camlog Foundation Consensus Report. *Clinical Oral Implants Research* 25 (11): 1301–1303.
- **48** Mailoa, J., Fu, J.H., Chan, H.L. et al. (2015). The effect of vertical implant position in relation to adjacent teeth on marginal bone loss in posterior arches: a retrospective study. *The International Journal of Oral & Maxillofacial Implants* 30 (4): 931–936.
- **49** Saleh, M.H., Ravida, A., Suárez-López del Amo, F. et al. (2018). The effect of implant-abutment junction position on crestal bone loss: a systematic review and metaanalysis. *Clinical Implant Dentistry and Related Research* 20 (4): 617–633.
- **50** Liu, Y. and Wang, J. (2017). Influences of microgap and micromotion of implant–abutment interface on marginal bone loss around implant neck. *Archives of Oral Biology* 83: 153–160.
- **51** Weng, D., José Hitomi Nagata, M., Francisco Bosco, A. et al. (2011). Influence of microgap location and configuration on radiographic bone loss around submerged implants: an experimental study in dogs. *The International Journal of Oral & Maxillofacial Implants* 26 (5): 941–946.
- **52** King, G.N., Hermann, J.S., Schoolfield, J.D. et al. (2002). Influence of the size of the microgap on crestal bone levels in non-submerged dental implants: a radiographic

study in the canine mandible. *Journal of Periodontology* 73 (10): 1111–1117.

- **53** Roos-Jansåker, A.M., Lindahl, C., Renvert, H., and Renvert, S. (2006). Nine-to fourteen-year follow-up of implant treatment. Part II: presence of peri-implant lesions. *Journal of Clinical Periodontology* 33 (4): 290–295.
- **54** Tarnow, D., Cho, S.C., and Wallace, S.S. (2000). The effect of inter-implant distance on the height of inter-implant bone crest. *Journal of Periodontology* 71 (4): 546–549.
- **55** Misch, C.E., Perel, M.L., Wang, H.L. et al. (2008). Implant success, survival, and failure: The International Congress of Oral Implantologists (ICOI) pisa consensus conference. *Implant Dentistry* 17 (1): 5–15.
- **56** Galindo-Moreno, P., León-Cano, A., Ortega-Oller, I. et al. (2015). Marginal bone loss as success criterion in implant dentistry: beyond 2 mm. *Clinical Oral Implants Research* 26 (4): e28–e34.
- **57** Derks, J., Schaller, D., Håkansson, J. et al. (2016). Peri-implantitis–onset and pattern of progression. *Journal of Clinical Periodontology* 43 (4): 383–388.
- **58** Schwarz, F., Derks, J., Monje, A., and Wang, H.L. (2018). Peri-implantitis. *Journal of Clinical Periodontology* 45: S246–S266.
- **59** Moy, P.K., Medina, D., Shetty, V., and Aghaloo, T.L. (2005). Dental implant failure rates and associated risk factors. *The International Journal of Oral & Maxillofacial Implants* 20 (4): 569–577.
- **60** Hämmerle, C.H., Chen, S.T., and Wilson, T.G. Jr. (2004). Consensus statements and recommended clinical procedures regarding the placement of implants in extraction sockets. *The International Journal of Oral & Maxillofacial Implants* 19 (Suppl): 26–28.
- **61** Romanos, G.E. and Weitz, D. (2012). Therapy of periimplant diseases. Where is the evidence? *The Journal of Evidence-Based Dental Practice* 12 (3): 204–208.
- **62** Koo, K.T., Khoury, F., Keeve, P.L. et al. (2019). Implant surface decontamination by surgical treatment of periimplantitis: a literature review. *Implant Dentistry* 28 (2): 173–176.
- **63** Khoury, F., Keeve, P.L., Ramanauskaite, A. et al. (2019). Surgical treatment of peri-implantitis–Consensus report of working group 4. *International Dental Journal* 69: 18–22.
- **64** Machtei, E.E., Romanos, G., Kang, P. et al. (2021). Repeated delivery of chlorhexidine chips for the treatment of peri-implantitis: a multicenter, randomized, comparative clinical trial. *Journal of Periodontology* 92 (1): 11–20.
- **65** Schwarz, F., Jepsen, S., Obreja, K. et al. (2022). Surgical therapy of peri-implantitis. *Periodontology 2000* 88 (1): 145–181.
- **66** Lindhe, J., Meyle, J., and Group D of the European Workshop on Periodontology (2008). Peri-implant diseases: consensus report of the sixth European

workshop on periodontology. *Journal of Clinical Periodontology* 35: 282–285.

- **67** Karring, E.S., Stavropoulos, A., Ellegaard, B., and Karring, T. (2005). Treatment of peri-implantitis by the Vector® system: a pilot study. *Clinical Oral Implants Research* 16 (3): 288–293.
- **68** Renvert, S., Roos-Jansåker, A.M., and Claffey, N. (2008). Non-surgical treatment of peri-implant mucositis and peri-implantitis: a literature review. *Journal of Clinical Periodontology* 35: 305–315.
- **69** Renvert, S., Samuelsson, E., Lindahl, C., and Persson, G.R. (2009). Mechanical non-surgical treatment of peri-implantitis: a double-blind randomized longitudinal clinical study. I: clinical results. *Journal of Clinical Periodontology* 36 (7): 604–609.
- **70** Schwarz, F., Jepsen, S., Herten, M. et al. (2006). Influence of different treatment approaches on non-submerged and submerged healing of ligature induced peri-implantitis lesions: an experimental study in dogs. *Journal of Clinical Periodontology* 33 (8): 584–595.
- **71** Parma-Benfenati, S., Tinti, C., Romano, F. et al. (2020). Long-term outcome of surgical regenerative treatment of peri-implantitis: a 2-to 21-year retrospective evaluation. *The International Journal of Periodontics & Restorative Dentistry* 40 (4): 487–496.
- **72** Renvert, S. and Polyzois, I.N. (2015). Clinical approaches to treat peri-implant mucositis and peri-implantitis. *Periodontology 2000* 68 (1): 369–404.
- **73** Figuero, E., Graziani, F., Sanz, I. et al. (2014). Management of peri-implant mucositis and periimplantitis. *Periodontology 2000* 66 (1): 255–273.
- **74** Khoshkam, V., Chan, H.L., Lin, G.H. et al. (2013). Reconstructive procedures for treating peri-implantitis: a systematic review. *Journal of Dental Research* 92 (12_suppl): 131S–138S.
- **75** Coli, P., Christiaens, V., Sennerby, L., and Bruyn, H.D. (2017). Reliability of periodontal diagnostic tools for monitoring peri-implant health and disease. *Periodontology 2000* 73 (1): 203–217.
- **76** Serino, G., Turri, A., and Lang, N.P. (2013). Probing at implants with peri-implantitis and its relation to clinical peri-implant bone loss. *Clinical Oral Implants Research* 24 (1): 91–95.
- **77** Bohner, L.O., Mukai, E., Oderich, E. et al. (2017). Comparative analysis of imaging techniques for diagnostic accuracy of peri-implant bone defects: a meta-analysis. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology* 124 (4): 432–440.
- **78** Serino, G., Sato, H., Holmes, P., and Turri, A. (2017). Intra-surgical vs. radiographic bone level assessments in measuring peri-implant bone loss. *Clinical Oral Implants Research* 28 (11): 1396–1400.

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- **79** Cassetta, M., Di Giorgio, R., and Barbato, E. (2018). Are intraoral radiographs reliable in determining peri-implant marginal bone level changes? The correlation between open surgical measurements and periapical radiographs. *International Journal of Oral and Maxillofacial Surgery* 47 (10): 1358–1364.
- **80** Christiaens, V., De Bruyn, H., De Vree, H. et al. (2018). A controlled study on the accuracy and precision of intraoral radiography in assessing interproximal bone defect morphology around teeth and implants. *European Journal of Oral Implantology* 11 (3): 361–367.
- **81** Saberi, B.V., Khosravifard, N., Ghandari, F., and Hadinezhad, A. (2019). Detection of peri-implant bone defects using cone-beam computed tomography and digital periapical radiography with parallel and oblique projection. *Imaging Science in Dentistry* 49 (4): 265–272.
- **82** Song, D., Shujaat, S., de Faria Vasconcelos, K. et al. (2021). Diagnostic accuracy of CBCT versus intraoral imaging for assessment of peri-implant bone defects. *BMC Medical Imaging* 21 (1): 1–8.
- **83** Rodrigues Pinheiro, L., Scarfe, W.C., de Oliveira Sales, M.A. et al. (2017). Effectiveness of periapical radiography versus cone beam computed tomography with different kilovoltage settings in the detection of chemically created peri-implant bone defects: an in vitro study. *The International Journal of Oral & Maxillofacial Implants* 32 (4): 741–750.
- **84** Schwindling, F.S., Hilgenfeld, T., Weber, D. et al. (2019). In vitro diagnostic accuracy of low-dose CBCT for evaluation of peri-implant bone lesions. *Clinical Oral Implants Research* 30 (12): 1200–1208.
- **85** Hilgenfeld, T., Juerchott, A., Deisenhofer, U.K. et al. (2018). Accuracy of cone-beam computed tomography, dental magnetic resonance imaging, and intraoral radiography for detecting peri-implant bone defects at single zirconia implants – an in vitro study. *Clinical Oral Implants Research* 29 (9): 922–930.
- **86** Rios, H.F., Borgnakke, W.S., and Benavides, E. (2017). The use of cone-beam computed tomography in management of patients requiring dental implants: an American Academy of Periodontology best evidence review. *Journal of Periodontology* 88 (10): 946–959.
- **87** Schulze, R., Heil, U., Groβ, D. et al. (2011). Artefacts in CBCT: a review. *Dento Maxillo Facial Radiology* 40 (5): 265–273.
- **88** Nardi, C., Borri, C., Regini, F. et al. (2015). Metal and motion artifacts by cone beam computed tomography (CBCT) in dental and maxillofacial study. *La Radiologia Medica* 120 (7): 618–626.
- **89** Schulze, R.K., Berndt, D., and d'Hoedt, B. (2010). On cone-beam computed tomography artifacts induced by titanium implants. *Clinical Oral Implants Research* 21 (1): 100–107.
- **90** Dave, M., Davies, J., Wilson, R., and Palmer, R. (2013). A comparison of cone beam computed tomography and conventional periapical radiography at detecting periimplant bone defects. *Clinical Oral Implants Research* 24 (6): 671–678.
- **91** Nevins, M., Nevins, M., Langer, B. et al. (2018). Clinically oriented patient maintenance protocol: a clinical consensus of experts. *The International Journal of Periodontics & Restorative Dentistry* 38: 281–288.

4

Clinical, Microbiological, and Immunological Risk Indicators of Peri-implant Mucositis and Early Peri-implant Bone Loss

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Introduction

Osseointegrated dental implants have undoubtedly advanced reconstructive dentistry, by adding a powerful treatment alternative to the armament of the clinician. They can be further considered in the treatment planning for restoring masticatory, occlusal, and esthetic functions of missing teeth. A unique feature, amid other types of endosseous implants, of being partially exposed to a microbial-rich environment, the oral cavity, which naturally renders them prone to biofilm colonization. Not unexpectedly, the increasing number of implants inserted worldwide coincides with an overall increase in periimplant infections [1].

Because dental implants are expected to functionally substitute to natural teeth, there is also a tendency to perceive diseases of ailing implants as pathologies analogous to gingivitis and periodontitis [2]. Yet, a number of factors attributed to the structural and immunological variability of the peri-implant tissues render them more susceptible to endogenous oral infections, compared to natural teeth [2–4]. However, as further described in this chapter, the etiopathological basis of these two groups of oral diseases is not entirely the same. Increasing evidence indicates that the peri-implant ecosystem differs from the natural one established in the gingival crevice around periodontal tissues, reflecting a compositionally distinct microbiota [5]. Whereas this microbiota may evolve in homeostasis with the host, ecological changes may readily trigger and dysbiotic compositional shifts that further induce inflammatory alterations and ultimately lead to the destruction of the implant-supporting tissues.

Diagnostic Epidemiological Aspects of Peri-implant Infections

Diagnosis of peri-implant infections primarily relies on the clinical and radiographical signs of the induced inflammatory changes. Accordingly, diagnostic criteria for periimplant mucositis include bleeding on probing (BOP), yet without radiographic signs of crestal bone loss beyond the initial remodeling [6]. In contrast, radiographic evidence of osteolysis, typically appearing as a symmetrical "saucershaped" crestal defect around the implant, is central to the diagnosis of peri-implantitis. Clinically, an increased probing pocket depth (PPD), as compared to previous examinations is also indicative of peri-implantitis; however, the angulation, insertion depth, and threads of some implants may render peri-implant probing challenging [6]. BOPpositive peri-implant sites run a higher probability to be diagnosed with peri-implantitis [7], whereas suppuration appears to be very much limited to peri-implantitis cases [8], and its presence is a good discriminant between health or peri-implant mucositis and actual periimplantitis [9]. The prevalence of peri-implantitis has been reported at 28–56% of implant-baring patients, or 12–43% of the total number of implants [10], with a weighted mean prevalence at 22% [11]. Overall, about 1/3 of all patients and 1/5 of all implants will be affected by periimplantitis [12]. Major risk factors associated with these epidemiological observations include past history of periodontitis, smoking, and diabetes, particularly in the absence of adequate oral hygiene [13–17]. Ill-fitting or ill-designed fixed and cement-retained restorations are also evident factors of local plaque accumulation [12].

Early Peri-implant Bone Loss and Associated Risk Factors

The process of wound healing following implant insertion is accompanied by bone remodeling that may eventually lead to a slight reduction of the marginal bone level and mild immunological reactions. As described further in this section, the process of early bone loss is multifactorial and involves intrinsic as well as extrinsic causes.

Stability of peri-implant crestal bone (PICB) level is one of the pivotal issues that dictate both biological and esthetic long-term success of dental implants [18]. In order to reach this goal clinicians should thoroughly understand the biological mechanisms of peri-implant bone behavior to local and systemic factors associated with its remodelation as well as how to manage them.

Immediately after dental implant surgery, bone and soft tissue structures surrounding the implant site go through acute alterations. As the consequence of surgical intervention both submerged and non-submerged implant placement procedures trigger wound healing and remodeling mechanisms in bone and mucosa that includes initiation and ultimately resolution of inflammation, matrix deposition, bone resorption/apposition, neurogenesis, and angiogenesis. This healing response is mediated by activated resident and migrating cells and related biological cascades involving cell–cell and cell–tissue interactions which are modulated by various cytokines, chemokines, and growth factors [19, 20].

Generally, 12 weeks after implant and/or healing abutment installation, integration of implant (osseointegration) at the bone-implant interface and formation of attachment apparatus/biological seal at the soft tissuetransmucosal part interface are almost entirely completed [20–23]. However, tissues, particularly peri-implant crestal bone, continue to undergo dimensional changes, a phenomenon called "early/initial peri-implant bone loss" (EPBL), which is generally accepted to continue up to one year following occlusal loading [24–26]. On marginal bone level changes around dental implants, EPBL indicates that the establishment of the implant integration with surrounding tissues to attain homeostasis is not a static state that is reached in several weeks following implant insertion, but rather a dynamic equilibrium process that may last more than a year. After one year of definitive prosthesis placement tissue, stability is expected. Minimal modeling of crestal bone around a successful implant of no more than 0.1–0.2mm per year is favorable in the following periods of implant function [27, 28]. Cochran and colleagues have reported that the most significant amount of PICB which was independent from arch, type of restoration, or implant length, accounting for the 86% of the total bone loss in five years, occurred between the time of implant placement and the time that the final prosthesis was placed [29].

Peri-implantitis is frequently defined as inflammation of the peri-implant soft tissues associated with progressive marginal bone loss (Figure 4.1) [30]. The changes in PICB at early healing and maturation phases often occur as a consequence of physiological processes alone or in combination with individual adaptive response to intrinsic and extrinsic factors [26]. Since this reduction in periimplant bone level is not primarily of infectious origin and limited $\left($ <2 mm), it is believed to be a "condition" or a "complication" rather than a disease and quite distinct from peri-implantitis-related bone loss (Figure 4.2) [26, 31, 32]. EPBL may be indicative of a remodeling response and does not represent a clinical condition that needs therapeutic intervention. Therefore, it is pivotal to distinguish between limited early physiological bone remodeling and advancing PICB namely peri-implantitis, with the latter implying a pathological state of infectious origin. According to "Statements from the Estepona Consensus Meeting on Peri-Implantitis, 2012" it is not appropriate to name entire universe of peri-implant bone losses as "peri-implantitis" [33]. Therefore, to name "EPBL" as "peri-implantitis" at the initial stage is not convenient; however, the scenario that peri-implant tissue inflammation may occur due to plaque accumulation on previously exposed rough implant surface as a result of early bone loss and can serve as a "locus minoris resistensiae" for further progressive bone loss can not be entirely ruled out [25, 26, 34, 35]. This assumption of secondary infectious etiology may be questioned by some experts because presence of increased plaque accumulation and inflammation does not necessarily lead to peri-implantitis and initially remodeled crestal bone may be maintained unchanged for prolonged periods in the absence of other risk factors [35–37]. There is a tendency to admit that bone resorption around dental implants beyond "early peri-implant bone loss" which is within acceptable limits following the implants' first year in function, \geq 2 mm may represent a disease condition namely peri-implantitis [38]. This type of additional marginal bone loss coupled with inflammation and profuse bleeding has been defined as a situation in which clinicians should be alerted and carefully monitored if the site needs therapeutic intervention [39].

Evidence also suggests that progressive PICB loss in the absence of mucosal inflammation is not likely to occur [30]. Also, inflammation may be present but may only be contained in the mucosal region farther from PICB which is in a steady equilibrium state thus limited EPBL may remain stable and may exhibit a non-progressing character [35].

Figure 4.1 Peri-implantitis refers to a biofilm-driven pathological state that causes inflammation of the peri-implant connective tissues, increased probing depths, and/or mucosal recession associated with progressive marginal bone loss. Whereas distal implants show peri-implantitis and associated clinical and radiological findings, mesial aspects of the mesial implants remain otherwise unaffected.

Figure 4.2 PICB alterations during early stages of healing and maturation are often consequences of adaptive responses to intrinsic and extrinsic factors. The above case showing a limited bone loss (<2mm) of non-infectious cause is to be distinguished from peri-implantitis-driven PICB osteolysis. EPBL may represent a remodeling response that does not require therapeutic intervention. Therefore, it is indicative of a "condition" or "complication" rather than a "pathological state" or a "disease". Following submerged and subcrestal placement (top left), uncovering implants and connecting healing abutments (top right, bottom left), slight radiological PICB loss and exposure of the implant neck six months following functional loading is not accompanied by inflammation, bleeding, or increased probing depths (bottom middle and right).

However, presence of combined potential risk factors that will result in disequilibrium of the host-implant interaction such as smoking, history of periodontal disease, lack of proper plaque removal and maintenance may favor a secondary opportunistic microbial etiology in time and thus jeopardize the long-term survival of the implants by extending the borders of the immune-inflammatory response to crestal bone [32, 39, 40]. From this point of view, EPBL especially occurring at pre-loading period may act as a predictor for future and advancing bone loss, leading to the development of peri-implantitis and resulting in implant failure (Figure 4.3) [25, 41, 42]. Wandael et al. have reported that implant surface exposure as a consequence of EPBL exceeding 0.5- and 1-mm threshold results in higher prevalence and odds ratio (four to six times higher) of peri-implantitis and implant loss, especially in combination with smoking and/or a history of periodontitis. Non-smokers presenting <5mm EPBL showed the least probability of future peri-implantitis [40]. Similarly, it has been shown that almost all implants with an 0.44mm or more at 6 months after loading had shown progression

in PICB loss and lost >2mm marginal bone at 18 months post-loading [43].

Although earlier literature has reported up to 1.5–2 mm of early bone loss to be within the limits of the acceptable thresholds, parallel to contemporary implant technology and increasing esthetic demands clinicians expect to maintain crestal bone for prolonged periods and to minimize or prevent PICB loss occurring at early periods of implant function to ensure the longevity of implants. EPBL is a multifactorial entity depending on factors related to patients, implant design, surgical protocols, and restorative protocols which are generally manageable or avoidable (Figure 4.4) [42, 44, 45]. In the following parts, these factors and recommended strategies to maintain the stability of peri-implant tissues will be discussed in detail.

Factors Related to Early Peri-implant Bone Loss

Several factors associated with EPBL should be taken into consideration from the planning phase to the loading stage

Figure 4.3 Schematic representation of early peri-implant bone loss and putative mechanism underlying the predisposition to peri-implantitis. MBL, marginal bone loss.

Figure 4.4 Factors that may result in early bone loss and that thereby may favor further biofilm accumulation may ultimately predispose to peri-implantitis. Adverse effects of these factors can be mitigated by an appropriate risk assessment/management of the patient, implant selection, and controlling surgical and restorative factors.

of the dental implant rehabilitation in order to keep the PICB loss rate at minimum.

Patient

Patient-related factors might be more effective in the long-run since their potential effects last longer than the locally-acting factors however clinicians might question their possible association with EPBL in order to assess the risk. Smoking is a well-documented risk factor for oral and periodontal health. Although smoking strongly shifts the peri-implant microbiome to a disease-associated state even in healthy sites, smokers and non-smokers may exhibit similar probing depth and inflammation scores [46, 47]. No convincing evidence has been reached to date for smoking as being a risk factor/indicator of peri-implant bone loss [30]. Similarly, the association of peri-implantitis with diabetes and patient genetics is somewhat unclear. In contrast to the above factors cause and effect relation between peri-implantitis and history of periodontitis, poor plaque control and lack of regular maintenance therapy is robust [30]. The relation between peri-implantitis and cardiovascular disease, rheumatoid arthritis, osteoporosis, thyroid disorders, hepatitis,

radiation therapy, chemotherapy, body mass index, hyperlipidemia, obesity, vitamin deficiency, serotonin reuptake inhibitors, and proton pump inhibitors present limited evidence and needs to be further cleared [21, 30].

Patients should be instructed thoroughly about the proper post-operative care to avoid flap dehiscence and infection-related EPBL including protection of the surgical site from trauma borne by wearing dentures and chewing on hard foods, usage of anti-inflammatory drugs and abstaining from consumption of hot beverages to minimize the tissue edema, gentle brushing in conjunction with application of antiseptic agents and antibiotic prescription when necessary.

Implant Design

Two-piece implant systems mainly consist of an abutment interlocking with the fixture by screws and anti-rotational geometry or friction only. Under high chewing load vectors, the interface between the two components shows elastic deformations (micromovement) and forms a void (microgap) that opens and closes repeatedly [48]. This gap interconnects the inner part of the implant and peri-implant tissues which may lead to transfer of the bacteria, bacterial toxins, and acidic compounds (microleakage) in close proximity to the peri-implant bone where they may induce inflammatory bone loss [49]. Although none of the different contemporary IAC's (integrated abutment crown) designs are free of gap and totally prevent leakage through the interface, conical (morse taper) connections are likely to present increased resistance to micromovements and reduced micro-gap/microleakage formation compared both to external and non-conical internal connections thus may be associated with reduced bone loss (Figure 4.5) [50–52]. All morse taper-like connections form a narrower abutment diameter namely horizontal offset (platform switch-PS) at the IAC due to internal cone structure. PS-structured implants have been linked with reduced bone loss compared to non-PS ones, but the association seems to be indirect [53]. Proposed mechanisms of actions for PS in decreasing bone loss are; (i) movement of the IAJ thus microleakage away from crestal bone, modification of the biological width by adding a horizontal component, and more desirable distribution of forces which may interfere with the integrity of bone-implant contact at the crestal portion of the implant [42, 50].

Based on the design of the two-piece implants the vertical position of micro-gap relative to bone may vary. The gap is located at the mucosal margin for the "soft tissue level" implants, above the crest but below the mucosal margin for the "polished collar" implants, and at or below the bone crest for the "fully rough-surfaced" implants [54]. Theoretically "soft tissue level" implants harbor the most

Figure 4.5 Abutment and fixture frequently interconnect by screws and geometrical structures in two-piece implants to provide a tight and stable seal. Contemporary internal connections are widely preferred types of interfaces although, to some extent, all types of internal connections display microgaps. Conical internal connections are likely to present increased resistance to micromovements and reduced microgap/ microleakage formation over non-conical IAC's. However, engineering quality of the implants incorporating a welldesigned IAC may be of greater importance than the type of IAC to minimize both technical and biological complications. Note the lack of crestal bone loss around an implant with an internal, non-conical IAC after six years of loading.

coronal micro-gap location, which is equimucosal; however, this design can not prevent bone loss in both early and late periods [55]. In addition, sub-crestal positioning of transmucosal part of soft tissue level implants and polished collar implants should be avoided since this approach increases bone resorption possibly due to lack of appropriate distribution of occlusal stress associated with absence of threads, approximation of the micro-gap to bone and increased possibility of submucosal cement remnants [55–57].

Neck portion design of the implants have been long focused as a site where the majority of forces are transmitted and cortical crestal bone- implant-connective tissue intersects. Closely placed tiny threads at the most coronal part (micro-threads) of the implants and gradual decreasing of the thread width from implant apex to the neck are designs directed toward reducing the excess pressure at the neck portion. Also, laser-microtextured implant design both at collar area and abutment have been introduced. Although rough-collared implants present lower PICB loss compared to smooth-collar ones, evidence is lacking on the addition of micro-threads and laser micro-texture to rough collar regarding PICB loss [58–60].

Surgical Protocols

Abundant vascularization of soft and hard tissues is critical for healing and remodeling of peri-implant bone. Therefore, proper 3D positioning of the implants in bone relative to cortical plates and nearby teeth and/or implants is critical to keep the EPBL at minimum. Implants at the crestal portion often neighbor dense cortical bone with a limited blood supply, thereby making the bone to be more prone to necrosis when excessive insertion torque is applied during placement of implants especially those of large implant diameter [45]. Hence EPBL related to surgical trauma is observed more pronounced at sites where cortical bone proportion is much greater than the cancellous bone (Figure 4.6) [61]. In order to facilitate preparation of properly shaped implant osteotomy and to reduce strain on crestal bone, usage of adequate final, pre-tap, and cortical drills according to the manufacturer's recommendations are the practical solutions to overcome necrosis of dense bone. In addition, overheating of the

Figure 4.6 Early peri-implant bone loss related to surgical trauma may be more noticeable at sites with dense cortical bone due to the limited blood supply. Therefore, large diameter implants may be a preferred choice at maxillary posterior sites, which bare abundant cancellous bone more frequently than at mandibular posterior sites. Radiographs of a 6×8mm plateau design, press-fit, locking-taper implant immediately after placement (left), and uncovering surgeries (right).

Figure 4.7 Implant's bed can be prepared with osteotoms in the maxilla in order to place an implant, which length would exceed the vertical bone height below the neighboring maxillary sinus (left). In case of insufficient bone width crest splitting may be applied. Because these techniques increase intraoperative trauma, placing a slightly submerged implant may help compensating for post-operative bone remodeling (right).

bone should be avoided by copious irrigation to prevent bone damage. When implants are placed following osteotome-assisted crestal sinus lift or crest splitting increased trauma to bone may be anticipated thus slight submerged placement might be encouraged (Figure 4.7). Novel strategies alternative to conventional drilling such as osseodensification and piezosurgery might offer some benefits. Biomaterial and biomechanical considerations to prevent risks in implant therapy [31, 62, 63].

Spontaneous exposure of cover screw is another issue associated with EPBL (Figure 4.8) [64]. To overcome this complication, principles for atraumatic surgical flap management from incision to suturing and correct vertical positioning of the implant should be strictly followed particularly when the mucosa is thin (Figure 4.9). Mucosal height determines whether the bone resorption will occur during the establishment of the biological width around the transmucosal portion of implants as evidenced by the presence of thick $(\geq 3$ mm) mucosa results in lower EPBL (Figure 4.10) [65]. Also, keratinized mucosa (KM) width <3mm might be associated with EPBL [66]. Therefore, practitioners should be familiar with the techniques to augment the mucosa and to modify the implant site

Figure 4.8 Traumatic surgical procedures may result in flap dehiscence, exposure of cover-screws and eventually in early peri-implant bone loss. The patient from the intra-oral photograph was referred 10 days following implant insertion for correction of the soft tissue problem.

accordingly to reduce the bone loss related to mucosal height and KM insufficiency.

Restorative Protocols

Abutment height that is surprisingly seemed to not relate with mucosal thickness and space for biological width establishment is a pivotal determinant of EPBL since it may influence the vertical position of the crown margin and short and divergent abutments may exert excess pressure on surrounding bone [67-70]. Intermittent connection and then disconnection of the healing abutments during the preparation of the final prosthesis causes disruption and then reestablishment of the mucosal seal which then in turn may lead to EPBL [71, 72]. In this context, choice of definitive abutment placement instead of healing abutment may be a viable method [73, 74].

Figure 4.9 Following basic principles in incision, flap reflection, management, suturing, and tension-free closure of the mucosal flap serves atraumatic implant surgery, allows sufficient vascularization, and predisposes to an uneventful and timely healing. Primary flap closure with 5.0 polypropylene interrupted, crisscross, and horizontal mattress sutures (top) and healed site at four weeks (bottom).

Planning and restorative principles should be carefully followed to decide the optimal timing of loading, to provide a suprastructure design that presents a natural restoration emergence profile angle, to prevent improper marginal fit and abutment seating, to form a concave configuration of the transmucosal component with a narrow abutment profile, to exclusively select the retaining method (cemented versus screwed) in the context of excess cement avoidance and to allow for fabricating a prosthesis with a hygienic contour (Figure 4.11) [75–80]. In addition, prosthetic

Mucosa thickness Figure 4.10 In order to avoid PICB loss related to the formation of the biological width following abutment placement, presence of at least 3mm of mucosa thickness is critical. Implants surrounded with mucosa <3mm of height experience crestal bone loss whereas implants with peri-implant mucosal thickness ≥3mm show no loss associated with the establishment of the biological width (BW) (purple arrow: Vertical dimension of $BW = 3$ mm).

Figure 4.11 Restorative and surgical principles are critical to the fabrication of prosthetic crowns that respect a natural restoration emergence angle (REA) to minimize EPBL. A REA <30° is favorable in terms of avoiding early peri-implant bone loss. Stability of the crestal bone around a 4×9mm, subcrestally-placed, platform switch, conical connection, implant after three years (left) and seven years (right) of loading can be observed.

factors associated with, material selection, occlusal relations, distribution of occlusal forces, crown to implant ratio should be analytically considered to prevent technical complications leading to loosening of the components and titanium particle release/metallic leakage and ultimately to inflammation-associated EPBL [25, 31, 81–83].

Radiologically-detected EPBL is a multifactorial entity that constitutes one of the academical controversial issues of implantology. It should be underlined that early limited reductions in PICB are not indicative of peri-implant disease. Each individual factor given above seems to exert a minimal effect on PICB loss however may be of clinical significance regarding long-term success when several of them conjoins. To sum-up, total precise engineering of the implants incorporating an IAC which presents a stable connection with biological benefits and a crestal module that allows appropriate distribution of the forces and tight tissue integrity may be critical in the longevity of implants.

Additionally, since EPBL might be associated with surgical trauma utmost attempt should be given in order not to jeopardize the healing capacity and vascularization of peri-implant tissues. Restorative protocols should be followed with the consideration of eliminating and controlling prosthesis-related issues on PICB loss. Reducing the number of peri-implant inflammatory situations by minimizing EPBL related to above factors may lower the incidence of PI. Therefore, implant practitioners are responsible to be competent in surgical and restorative interventions in the context of life-long learning attitude, to work with well-documented implant systems in which they have comprehensive and analytical knowledge, to perform adequate planning regarding aesthetics and restoration-driven implantology, to properly perform the risk assessment of the patient and plan the maintenance schedule on the patient basis to reach the goal of "predictable and long-term implant stability" (Figure 4.12).

The Oral Environment and Microbial Ecology

The oral environment represents is a unique ecosystem, which is both spatially and ecologically diverse, as it is continuously bathed in saliva [84, 85], lined by shedding epithelia of various degrees of keratinization, and framed by the non-shedding dental surfaces, whereas the gingival sulcus interface between teeth and the gingiva physiologically contains a nutrient-rich, serum-derived transudate [86]. Occasionally, dental implants will replace natural teeth, and their metallic surface will also constitute a nonshedding surface in the oral cavity, with a peri-implant crevice that is deeper than that of the natural gingival sulcus. These anatomical and spatial diversities generate ecological gradients of temperature, redox potential, pH, and nutrient availability, which differentially support growth of the residing oral microbiota [87]. The spatial gradients of these conditions determine the distinct distribution and composition of microbial communities among the different niches of the oral cavity [88], described as "biogeography" [89].

The Microbial Ecosystem of the Peri-implant Niche

Dental implants offer a pristine, non-shedding titanium surfaces onto which a salivary pellicle rapidly adsorbs, just like onto natural teeth, with nonetheless slight compositional variations (apparent absence of low-molecularweight mucins and cystatins) [90]. The dynamics of bacterial colonization on implant surfaces are initially slower than on natural teeth [91], although the sequence and composition of early colonizing communities appear unaffected [92, 93]. We now understand that early colonizers adhere to implant surfaces within thirty minutes following implant insertion [94], and evolve toward the formation of organized biofilm communities in the next two weeks [91]. The composition of the microbial communities that have colonized the peri-implant niche at this stage resemble those of healthy neighboring gingival sulci [95], but they are less diverse [96]. Typically, in times of peri-implant health, the submucosal microbiota displays high relative abundances of *Leptotrichia* spp., *Veillonella dispar*, *V. parvula, Fusobacterium* spp., *Neisseria* spp., and *Streptococcus* spp. (including *S. sanguinis*, *S. parasanguinis*, *S. gordonii*), and to a lesser extent species of *Rothia* (*R. aeria*, *R. dentocariosa*) [97–99]. These species are all commensals and from an ecological standpoint, they mostly catabolize saccharides as nutrient source and thrive in aerobic environments, aside from *Leptotrichia* spp. that favors anaerobic milieus despite aerotolerant

abilities [100]. Whereas microbial colonization and biofilm formation are completely normal processes compatible with peri-implant health, ecological shifts may drive this microbial ecosystem into a state of "dysbiosis," as described further in this chapter.

Peri-implant Microbial Biofilms

Early work on the peri-implant microbiota relied on anaerobic culture-based techniques and phase contrast microscopy [101, 102]. Based on these early methodological observations, the established notion was that the healthy peri-implant sub-mucosa is populated by Gram-positive cocci, non-motile bacilli, and few Gram-negative anaerobic species [103, 104]. Peri-implant mucositis was characterized by more cocci, motile bacilli and spirochetes [105], whereas peri-implantitis was characterized by a progressive increase in Gram-negative, black-pigmented, motile, and anaerobic species, attributed to the lower oxygen conditions that prevail in deepened peri-implant pockets [106]. Standard molecular detection methods frequently detected *Aggregatibacter actinomycetemcomitans* [107], *P. gingivalis* [107, 108], *Prevotella intermedia* [107, 109], *Fusobacterium* spp. [109], *T. forsythia,* and *T. denticola* [110, 111] as well as broader groups of coil-shaped motile *Spirochaetes* and curve-shaped non-motile *Synergistetes* [112]. Strong microbiological similarities have been identified between periodontal and peri-implant diseases, which is not surprising given that natural teeth and dental implants share a common ecological environment of the oral cavity. Some notable microbiological differences include the frequent isolation of *Peptostreptococcus* spp., *Staphylococcus* spp. in peri-implant infections [94, 113–115].

Profiling of the Full Peri-implant Microbiome

"Next generation sequencing" (NGS) technologies have given rise to considerably broader opportunities for mapping the peri-implant microbiome and discovering hundreds of species in a given sample as well as their associations. These have enabled us to understand that the microbial ecology around implants is somewhat less diverse than around natural teeth [116–120], and that notable compositional differences exist between peri-implant and periodontal sites [116–118], in both health and disease. These later studies using contemporary molecular methods have confirmed what earlier conventional method studies have shown, which is that peri-implant infections are polymicrobial opportunistic infections, caused by shifts in our endogenous "normal" microbiota. Frequently found microbial taxa at healthy peri-implant sites include *Eubacterium*, *Anaerofilum*, *Prevotella*, *Porphyromonas*, *Selenomonas*, *Streptococcus*, *Actinomyces*, *Leptotrichia*, *Veillonella*, *Propionibacterium, Peptococcus, Fusobacterium, Lactococcus, Rothia,* and *Treponema* [116–120], whereas periimplantitis sites foster more *Treponema*, *Filifactor*, *Porphyromonas*, *Prevotella*, *Tannerella*, *Campylobacter,* or *Staphylococcus* spp. [117–121]. Compared to periodontitis, peri-implantitis sites foster more *Desulfomicrobium orale*, *Sphingomonas*, *Veillonella*, *Actinomyces* spp., and various streptococci, differences which are also observed in individuals with a past history of periodontitis (Figure 4.13) [119, 120]. Despite the well-established notion that existing periodontal pockets are reservoirs of periodontopathogens for implants [123], with the help of NGS it became clearer that peri-implant and periodontal sites behave as distinct microbial ecosystems [117–120, 122]. Hence, implant colonization by specific microbes is driven primarily by local ecological features of the peri-implant environment with influence from nearby niches, such as gingival crevices or periodontal pockets [117, 119, 120].

Microbial Ecology in Transition from Peri-implant Health to Disease

Peri-implant diseases are caused by the loss of homeostasis between the microbiota located in the crevice and host tissues. Although host inflammation is ultimately responsible for the most part of peri-implant tissues breakdown, it remains a secondary event and it is shifts in the microbial ecology that instigate the transition from health to disease [124, 125]. Typically, factors that promote extensive and uninterrupted biofilm growth, such as poor oral hygiene or plaque-retentive restorations, allow the establishment of more elaborated food networks, interspecies signaling, and response interactions within microbial communities [126]. This supports colonization of proinflammatory taxa whose abundance increases relative to health, resulting into a synergistically more pathogenic community that subverts host control, i.e. a dysbiotic microbiota [127]. This disadvantageous relationship with the host propagates further inflammation in the peri-implant mucosa, thereby increasing the exudation of PICF and creating a proteinrich environment that further supports the establishment of proteolytic/pathogenic species [2]. Interruption of oral hygiene procedures for as long as three-weeks is sufficient to enable the emergence of common periodontal pathogens on the implant surface [126], which will, in turn, take advantage of the incipient inflammation as a source of their nutrients, in order to grow and stimulate further

inflammation [128, 129]. Altogether, overgrowth and shifts in the microbial composition set-off a vicious cycle between microbial dysbiosis, ecological alterations, and inflammation that overwhelms host control and leads to an excessive inflammatory response. Beyond the conceptual understanding of factors driving these microbial ecological shifts, it would be clinically relevant to identify which microbial communities and specific compositional patterns appear associated with early bone loss [5].

Microbiota Shifts Associated with Early Peri-implant Bone Loss – Infection as Risk Indicator

Most recent evidence demonstrates that the ecological shifts from health to peri-implant mucositis, and further to peri-implantitis are characterized by an increase in microbial diversity [5]. During the transition to periimplant mucositis especially, new microbial species are joining the ecological landscape of the peri-implant niche, building up on the pre-existing microbial communities (Figure 4.14) [97, 98]. Specifically, higher abundances of "classical" periodontopathogens such as *Treponema* spp., *Prevotella intermedia*, *Porphyromonas gingivalis, P. endodontalis,* and *Tannerella forsythia* are observed along with more recently emerging putative pathogens including *Anaeroglobus geminatus*, *Capnocytophaga gingivalis* or *Prevotella loescheii,* and *P. tannerae*. [97, 98]. In contrast to commensal communities, these strictly anaerobic taxa display a markedly increased pathogenic potential notably due to their proteolytic activities, cytotoxin secretions, and ability to subvert the host immune system [112, 130, 131]. Furthermore, some of these taxa such as *P. gingivalis* in leading position or *A. geminatus* may elevate the pathogenicity of the entire community by altering patterns of gene expression in the ecosystem [132–134]. These observations indicate that peri-implant mucositis acquires an inflammatory "high-at-risk-for-harm" ecosystem, that is likely to already harbor the microbiota responsible to exacerbate periimplant inflammation and cause bone loss [98]. Indeed, because a lag in time is necessary between the establishment of dysbiosis and its adverse effects, the causal dysbiotic microbiota must somehow precede the appearance of clinical peri-implantitis (bone loss). This notion is supported by evidence showing that further shifts in microbial composition that accompany the transition from peri-implant mucositis to peri-implantitis remain more subtle [97, 98]. In other terms, the peri-implant pathogens likely responsible for bone loss are already ecologically established in peri-implant mucositis, mostly persist

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Figure 4.13 Schematic aggregate of the characteristic and core microbiota associated with different peri-implant conditions. Venn diagrams attempt a summative qualitative illustration of the bacterial composition detected in the submucosa of healthy peri-implant, peri-implantitis, and periodontitis sites. Taxonomy is reported at the genus level or lower. Font size is proportionate with the frequency of detection among publications. (a) Bacterial taxonomic composition from periodontitis and peri-implantitis sites is illustrated based on Kumar et al. [116], Dabdoub et al. [117], Maruyama et al. [118], and Yu et al. [122]. (b) Bacterial taxonomic composition from healthy implants and peri-implantitis sites are illustrated based on Kumar et al. [116], Sanz-Martin et al. [99], Tsigarida et al. [98], Yu et al. [122], and Zheng et al. [97]. Note of consideration; bioinformatic pipelines, criteria of taxonomic identification and statistical significance may vary among studies. (*Source:* Belibasakis et al. [5]. Reproduced here in agreement with the terms of a Creative Commons CC-BY license with SAGE publishing <https://creativecommons.org/licenses/by/4.0/>).

Figure 4.14 Dynamics of the submucosal microbiota diversity during the typical course of peri-implant infections. The illustration depicts the increase in microbial diversity detected during the transition from peri-implant health to peri-implant mucositis, and further to peri-implantitis. (*Source:* Belibasakis et al. [5]. Reproduced here in agreement with the terms of a Creative Commons CC-BY license with SAGE publishing [https://creativecommons.org/licenses/by/4.0/\)](https://creativecommons.org/licenses/by/4.0/). The illustration was designed by the authors using the web interface BioRender.com.

during the transition to peri-implantitis, and are gradually enriched with some more bacterial taxa. Typically, additional taxa that appear enriched in peri-implantitis include *Eubacterium* spp., *Campylobacter* spp., *Fretibacterium fastidiosum,* or anaerobic Firmicutes such as *Mogibacterium* spp., *Peptococcus* spp., or *Filifactor alocis*. One also observes further increase in abundance of *Treponema* species such as *T. denticola, T. maltophilum* [97–99, 116, 117] along with other as-yet-uncultivated taxa, e.g. *Treponema* HOT-246, HOT-257, and HOT-258, HOT-260, some of which tend to positively correlate with radiographic bone loss, PPD, and suppuration [98, 99, 118, 120, 122]. Another common finding also includes species of *Staphylococcus* spp. (e.g. *S. aureus*, *S. pettenkoferi,* or *S. hominis*) that are detected in higher levels in peri-implantitis, compared to peri-implant mucositis [98, 117, 119]. These can be considered as allochthonous species (non-oral transient contaminant) [135, 136], that may survive and ecologically establish, due to their ability to colonize artificial intra-oral surfaces, such as dentures or dental implants [114, 137]. Finally, another ecological observation of the transition from health to peri-implant

mucositis and to peri-implantitis, is the decrease in abundance of certain commensals such as non-mutans *Streptococcus*, *Lactobacillus,* or *Leptotrichia* [98, 116, 122]. That said, the loss of commensals is especially pronounced upon tobacco smoking [98].

In summary, the microbial diversity of submucosal biofilms increases during the transition from health to periimplant mucositis, and then to peri-implantitis, albeit to a more limited degree during the latter stage. In this dynamic process, the clinical state of peri-implant mucositis comes out as a key transitional phase, which establishes a hostdisadvantageous microbial ecology that exacerbates periimplant inflammation and stimulates early bone loss. This early turning point is characterized by a marked increase in abundances of anaerobic and proteolytic taxa, such as *Porphyromonas* spp., *Prevotella* spp., *Capnocytophaga*, *Tannerella forsythia,* or *Aneroglobus geminatus*, with a concurrent decrease in health-associated commensals. Detection of these microbial compositional shifts could become a useful early indicator of the progression of periimplant infections and potentially be employed as risk factor for early peri-implant bone loss.

Osseointegration and Peri-implant Tissue Microenvironment

Biocompatibility, biostability, and biosafety are crucial properties of dental implants that enable the integration to the oral cavity, thus contributing to a newly established oral micro-environment. They are paramount for optimal performance of the implant before and after functional loading. Dental implant biomaterials are directly interacting with the oral environment and adapt to the oral functional needs as a living organ [138]. Clinical biocompatibility refers to the biomaterial-host interaction, which starts with an early inflammatory immune response of the recipient bone and mucosal tissue and is a hallmark of favorable wound healing [139] Minimizing surgical trauma during implant installation is crucial for an optimal and functionally successful wound healing response.

Bone tissue healing around dental implants comprises of an initial homeostasis phase, a pro-inflammatory phase, a cell proliferation phase, and a final remodeling phase [140]. These successive phases are characterized by influxes of various immune cell types (e.g. polymorphonuclear leukocytes, macrophages, and lymphocytes) and structural cells (e.g. fibroblast and osteoblasts) that interact with one another coordinated by local gradients of growth factors, cytokines, chemokines and matrix proteins [22, 141, 142]. These soluble mediators are progressively detectable in the PICF collected from the osseointegrating-healing site, peaking at week 2 after implant insertion, and then decreasing during weeks 4–8, and remaining steady at least throughout week 24 [62, 63, 143]. Hence, the micro-inflammatory environment of the healing peri-implant tissues is crucial for successful wound healing around implants. This type of immune response during the early stages of osseointegration can be considered as "sterile inflammation" and is usually resolved without complications.

The initial immune response to implant installation is further modifiable by the features of the bone-implant interface, such as the implant surface characteristics/ design, surgical procedure for implant bed preparation or implant–abutment interface configuration [144]. Early peri-implant bone loss has also been attributed to these factors as well as to a postulated foreign body or hypersensitivity reaction [145–151]. The dental implant surface is expected to wear-off over time, resulting in to titanium corrosion and titanium particle release [152]. Although it is not clear whether particle release is responsible for a potential hypersensitivity reaction, titanium ions can themselves induce inflammasome expression by macrophages, leading them to release active proinflammatory cytokines [153]. Indeed, a greater number of macrophages containing titanium particles is found in areas of close proximity to implant surface [154].

Immune Response of the Peri-implant Tissues to Biofilms

Similar to gingival tissue, peri-implant mucosa possesses mechanisms that may control the growth of biofilms, including a physical flushing process by the PICF, the rapid epithelial turnover, the influx of immune cells to the affected tissue, and the transmigration of neutrophils beyond the tissue into the peri-implant sulcus [2, 4]. Prolonged exposure to biofilms may induce both qualitative and quantitative changes of the inflammatory infiltrate around the per-implant mucosa, which can be reversible upon reinstitution of proper oral hygiene [155]. This seems to be independent of implant type, evidenced using experimental animal models [156]. The sequence of inflammatory events that take place in peri-implant mucositis are indeed similar to those in experimental gingivitis, yet the inflammation magnitude is greater and the inflammatory process extends faster toward the alveolar bone [157]. Experimental peri-implant mucositis at 3 weeks is characterized by a pronounced inflammatory cell infiltrate within the connective tissue underlying the oral epithelium [124]. The area of inflammatory lesion on the peri-implant mucosa can reach up to 0.14 mm^2 , and characterized by increased proportions of T- and B- lymphocytes [158]. The switch to peri-implantitis is accompanied by further influx of inflammatory cells into the affected area of the peri-implant mucosa, which now expands to reach the bone tissue [159, 160] whereas the size of the peri-implantitis lesions appears to be double than that of the periodontitis lesions (3.5 mm^2) versus 1.5mm² respectively) [161]. Moreover, diseased tissue obtained from peri-implantitis sites expresses higher levels of inflammatory mediators, compared to healthy or periimplant mucositis sites [162, 163]. Major osteolytic mediators, such as the cyclooxygenase-2 pathway, or the receptor activator of NF-κB ligand (RANKL)/osteoprotegerin (OPG) ratio, are significantly more upregulated in peri-implantitis, compared to periodontitis. [164, 165].

Diagnostic Potential of Peri-implant Crevicular Fluid

Periodontal indices are not as reliable diagnostic and prognostic measures for investigating dental implants and determining treatment needs, although it is well established that they are reliable diagnostic tools when it comes to natural teeth (e.g. PPD). That is because routine probing around unaffected implants could potentially result in trauma to the peri-implant soft tissues, with consequent mechanically-induced inflammation. It is for this reason that probing around dental implants is not recommended until after six months, when osseointegration is expected to have been completed [166]. Moreover, using probing to measure history of disease is less accurate around the peri-implant mucosa, since probes encounter less resistance and penetrate deeper around implants than around natural teeth [167, 168]. BOP around implants is also a poor indicator of progressive peri-implantitis, as it has been shown to be consistently present at sites with peri-mucositis, un-treated peri-implantitis, stable periimplantitis, and even in the majority of healthy sites [169].

PICF, the inflammatory exudate of the peri-implant sulcus, is the most proximal source of biomarkers for periimplant health and disease and can be collected non-invasively in less than five minutes [4, 63]. It is the result of increased vessel permeability of the underlying connective tissue, which responds to the growing biofilm at the implant–tissue interface [86], and is enriched with tissue breakdown products and inflammatory molecules [170–173]. Importantly, the molecular content of PICF can be easily quantified in the laboratory [2, 174]. The flow of PICF is minimum in health, but its traceable volume is increased in peri-implant mucositis and periimplantitis, according to the site-response to biofilm accumulation. [175, 176]. The qualitative composition of PICF is modified along with the histopathological changes of progressive peri-implant inflammation, so its molecular analysis may support the early detection of clinically undetectable disease [126].

Cytokines and Early Peri-Implant Bone Loss – Inflammation as Risk Indicator

Long-term implant survival can be facilitated by adequate surveillance and preventive measures throughout the implant-life. With appropriate surgical and restorative case management, it is possible to arrest early periimplant bone loss, regenerate lost bone, and maintain a healthy status for more than half a decade post-loading, but early detection and multidisciplinary care is paramount to ensure this [177]. Late implant failures have been associated with earlier inflammatory events, smoking, surgical approach, and posterior mandibular placement [178]. The operator's surgical and prosthetic rehabilitation capacity, the total number of implants in

the reconstructive treatment and the degree of early bone resorption appear to largely impact on the occurrence of late implant failures [179]. An important operator-related parameter to be considered for early peri-implant bone loss is retained dental cement, which can result in significant early peri-implant inflammation [180]. The diagnostic accuracy of clinical and radiographic evaluations at a given time-point on follow-up is crucial for identifying the history of peri-implant bone loss and the consequent occurrence of peri-implantitis. Baseline documentation is imperative for accurate early diagnosis of peri-implant inflammatory bone loss. A combination of BOP or suppuration on probing together with bone loss ≥ 2 mm can identify moderate or severe peri-implantitis with a high degree of specificity [181].

Detection of early peri-implant bone loss by molecular methods and non-invasive means would be an innovative and valuable approach to supplement clinical examination and support clinical diagnosis of the implant site under investigation. Several potential biomarkers have been considered in supporting clinical diagnosis of early or late peri-implant bone loss, but the conclusions reached have often been conflicting due to the heterogeneity in study designs [182]. The PICF collected at the tissueimplant interface is the ideal biological medium for this quest. Investigating early inflammatory markers in PICF is justifiable because the peri-implant niche displays cumulative levels of antigens (microbes and material) on site, which can activate a bone-destructive immuneinflammatory response [183].

Early studies showed that the presence of IL-1β in PICF could be indicative of early bone loss or disease progression to peri-implantitis [184]. The role of this particular cytokine is further supported by recent systematic reviews, which underlined a link between IL-1 genotype and early peri-implant inflammation, but not other studied genetic polymorphisms [185, 186]. Mobilization of inflammatory markers such as TNF- α and IL-1β, or markers of bone metabolism such as alkaline phosphatase, osteocalcin, and Cathepsin-K have been associated with early deterioration of peri-implant clinical parameters, as early as two weeks following implant function, which could serve as predictors of early implant complications [187]. Other bone remodeling regulators associated with early peri-implant bone loss are runt-related transcription factor-2 (Runx-2) and bone morphogenetic protein-2 (BMP-2) [188]. In addition, the cyclophilin A-EMMPRIN axis has been identified as an indicator of early peri-implant bone loss [189]. Smoking has also been associated with an increased early marginal bone loss particularly during the late phase of osseointegration, characterized by disturbances in the levels of hypoxiainducible factor 1 alpha (HIF-1 α) and IL-6 expression in PICF cells, over a period of three months [190, 191]. The PICF levels of cytokines, chemokine, and growth factors have also been evaluated during healing and osseointegration at osteotomy sites prepared either with piezosurgery or drills [62, 63]. Crestal bone levels did not depend on the osteotomy procedures modality. Regardless of the procedure, PICF levels of most studied molecular factors peaked at two weeks and decreased over time, from four or eight weeks onwards. Molecular PICF variations between the two procedures were transiently observed only in the case of RANTES and RANKL.

Of special consideration for the early detection of marginal peri-implant bone loss are molecules involved in connective tissue proteolysis and activation of osteoclastic bone resorption. The former category is largely represented by the family of MMP enzymes and their inhibitors TIMP, and particularly the active form of MMP-8 that has been invariably identified as a marker for progressive bone loss in peri-implantitis [173, 192–195]. The latter category is largely represented by the RANKL-OPG system as a regulator of bone resorption. Indeed, the levels of RANKL and OPG, as well as their relative RANKL/OPG ratio, in PICF have been associated with early peri-implant bone loss [188] or the occurrence of peri-implant mucositis, as compared to peri-implant health [175], revealing the diagnostic potential of this molecular system. The type of implant–abutment connection does not appear to influence this measure over a period of one year [144].

Conclusions

In this chapter, we identified and discussed clinical, microbiological, and immunological parameters as indications of early peri-implant inflammation (e.g. peri-implant mucositis) and early peri-implant bone loss (i.e. incipient peri-implantitis). Apart from well-established clinical risk factors, there are diagnostically underexploited possibilities for molecular verification of the early pathological events associated with the peri-implant tissues. As such, there is a finite number of immunological factors and trending microbiological profiles that need to be more routinely implemented in clinical diagnosis, in order to more definitively validate their early diagnostic and/or therapeutic potential. Pinpointing such immune and microbial signatures may indeed prove to be a valuable tool for the primary prevention of peri-implantitis and may further provide the basis for targeted antimicrobial action or outcome monitoring of therapeutic interventions. The clinical application of such novel risk indicators may ultimately allow to improve the long-term retention of dental implants.

References

- **1** Salvi, G.E., Cosgarea, R., and Sculean, A. (2017). Prevalence and mechanisms of peri-implant diseases. *Journal of Dental Research* 96 (1): 31–37.
- **2** Belibasakis, G.N. (2014). Microbiological and immunopathological aspects of peri-implant diseases. *Archives of Oral Biology* 59 (1): 66–72.
- **3** Heitz-Mayfield, L.J. and Lang, N.P. (2010). Comparative biology of chronic and aggressive periodontitis vs. peri-implantitis. *Periodontology 2000* 53: 167–181.
- **4** Belibasakis, G.N., Charalampakis, G., Bostanci, N., and Stadlinger, B. (2015). Peri-implant infections of oral biofilm etiology. *Advances in Experimental Medicine and Biology* 830: 69–84.
- **5** Belibasakis, G.N. and Manoil, D. (2021). Microbial community-driven etiopathogenesis of peri-implantitis. *Journal of Dental Research* 100 (1): 21–28.
- **6** Berglundh, T., Armitage, G., Araujo, M.G. et al. (2018). 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 Clinical Periodontology* 45 (Suppl 20): S286–S291.
- **7** Hashim, D., Cionca, N., Combescure, C., and Mombelli, A. (2018). The diagnosis of peri-implantitis: a systematic review on the predictive value of bleeding on probing. *Clinical Oral Implants Research* 29 (Suppl 16): 276–293.
- **8** Ramanauskaite, A., Becker, K., and Schwarz, F. (2018). Clinical characteristics of peri-implant mucositis and periimplantitis. *Clinical Oral Implants Research* 29 (6): 551–556.
- **9** Rakic, M., Monje, A., Radovanovic, S. et al. (2019). Is the personalized approach the key to improve clinical diagnosis of peri-implant conditions? The role of bone markers. *Journal of Periodontology* 91: 859.
- **10** Zitzmann, N.U. and Berglundh, T. (2008). Definition and prevalence of peri-implant diseases. *Journal of Clinical Periodontology* 35 (8 Suppl): 286–291.
- **11** Derks, J. and Tomasi, C. (2015). Peri-implant health and disease. A systematic review of current epidemiology. *Journal of Clinical Periodontology* 42 (Suppl 16): S158–S171.
- **12** Kordbacheh Changi, K., Finkelstein, J., and Papapanou, P.N. (2019). Peri-implantitis prevalence, incidence rate, and risk factors: a study of electronic health records at a U.S. dental school. *Clinical Oral Implants Research* 30 (4): 306–314.
- **13** Kumar, P.S. (2019). Systemic risk factors for the development of periimplant diseases. *Implant Dentistry* 28 (2): 115–119.
- **14** Roccuzzo, M., De Angelis, N., Bonino, L., and 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. *Clinical Oral Implants Research* 21 (5): 490–496.
- **15** Heitz-Mayfield, L.J. (2008). Peri-implant diseases: diagnosis and risk indicators. *Journal of Clinical Periodontology* 35 (8 Suppl): 292–304.
- **16** Mombelli, A., Muller, N., and Cionca, N. (2012). The epidemiology of peri-implantitis. *Clinical Oral Implants Research* 23 (Suppl 6): 67–76.
- **17** Lindhe, J., Meyle, J., and Group DoEWoP (2008). Peri-implant diseases: consensus report of the sixth European workshop on periodontology. *Journal of Clinical Periodontology* 35 (8 Suppl): 282–285.
- **18** Papaspyridakos, P., Chen, C.J., Singh, M. et al. (2012). Success criteria in implant dentistry: a systematic review. *Journal of Dental Research* 91 (3): 242–248.
- **19** Trindade, R., Albrektsson, T., Tengvall, P., and Wennerberg, A. (2016). Foreign body reaction to biomaterials: on mechanisms for buildup and breakdown of osseointegration. *Clinical Implant Dentistry and Related Research* 18 (1): 192–203.
- **20** Tomasi, C., Tessarolo, F., Caola, I. et al. (2016). Early healing of peri-implant mucosa in man. *Journal of Clinical Periodontology* 43 (10): 816–824.
- **21** Insua, A., Monje, A., Wang, H.L., and Miron, R.J. (2017). Basis of bone metabolism around dental implants during osseointegration and peri-implant bone loss. *Journal of Biomedical Materials Research. Part A* 105 (7): 2075–2089.
- **22** Berglundh, T., Abrahamsson, I., Lang, N.P., and Lindhe, J. (2003). De novo alveolar bone formation adjacent to endosseous implants. *Clinical Oral Implants Research* 14 (3): 251–262.
- **23** Tomasi, C., Tessarolo, F., Caola, I. et al. (2014). Morphogenesis of peri-implant mucosa revisited: an experimental study in humans. *Clinical Oral Implants Research* 25 (9): 997–1003.
- **24** Oh, T.J., Yoon, J., Misch, C.E., and Wang, H.L. (2002). The causes of early implant bone loss: myth or science? *Journal of Periodontology* 73 (3): 322–333.
- **25** Albrektsson, T., Chrcanovic, B., Ostman, P.O., and Sennerby, L. (2017). Initial and long-term crestal bone responses to modern dental implants. *Periodontology 2000* 73 (1): 41–50.
- **26** Coli, P. and Jemt, T. (2021). On marginal bone level changes around dental implants. *Clinical Implant Dentistry and Related Research* 23 (2): 159–169.
- **27** Adell, R., Lekholm, U., Rockler, B., and Branemark, P.I. (1981). A 15-year study of osseointegrated implants in

the treatment of the edentulous jaw. *International Journal of Oral Surgery* 10 (6): 387–416.

- **28** Albrektsson, T., Zarb, G., Worthington, P., and Eriksson, A.R. (1986). The long-term efficacy of currently used dental implants: a review and proposed criteria of success. *The International Journal of Oral & Maxillofacial Implants* 1 (1): 11–25.
- **29** Cochran, D.L., Nummikoski, P.V., Schoolfield, J.D. et al. (2009). A prospective multicenter 5-year radiographic evaluation of crestal bone levels over time in 596 dental implants placed in 192 patients. *Journal of Periodontology* 80 (5): 725–733.
- **30** Schwarz, F., Derks, J., Monje, A., and Wang, H.L. (2018). Peri-implantitis. *Journal of Periodontology* 89 (Suppl 1): S267–S290.
- **31** Albrektsson, T., Becker, W., Coli, P. et al. (2019). Bone loss around oral and orthopedic implants: an immunologically based condition. *Clinical Implant Dentistry and Related Research* 21 (4): 786–795.
- **32** Albrektsson, T., Canullo, L., Cochran, D., and De Bruyn, H. (2016). "Peri-implantitis": a complication of a foreign body or a man-made "disease". Facts and fiction. *Clinical Implant Dentistry and Related Research* 18 (4): 840–849.
- **33** Albrektsson, T., Buser, D., Chen, S.T. et al. (2012). Statements from the Estepona consensus meeting on peri-implantitis, February 2-4, 2012. *Clinical Implant Dentistry and Related Research* 14 (6): 781–782.
- **34** Monje, A., Galindo-Moreno, P., Canullo, L. et al. (2015). Editorial: from early physiological marginal bone loss to peri-implant disease: on the unknown local contributing factors. *The International Journal of Periodontics & Restorative Dentistry* 35 (6): 764–765.
- **35** Coli, P. and Jemt, T. (2021). Are marginal bone level changes around dental implants due to infection? *Clinical Implant Dentistry and Related Research* 23 (2): 170–177.
- **36** Doornewaard, R., Christiaens, V., De Bruyn, H. et al. (2017). Long-term effect of surface roughness and patients' factors on crestal bone loss at dental implants. A systematic review and meta-analysis. *Clinical Implant Dentistry and Related Research* 19 (2): 372–399.
- **37** Menini, M., Setti, P., Pera, P. et al. (2018). Peri-implant tissue health and bone resorption in patients with immediately loaded, implant-supported, full-arch prostheses. *The International Journal of Prosthodontics* 31 (4): 327–333.
- **38** Renvert, S., Persson, G.R., Pirih, F.Q., and Camargo, P.M. (2018). Peri-implant health, peri-implant mucositis, and peri-implantitis: case definitions and diagnostic considerations. *Journal of Periodontology* 89 (Suppl 1): S304–S312.
- **39** Klinge, B. (2012). Peri-implant marginal bone loss: an academic controversy or a clinical challenge? *European Journal of Oral Implantology* 5 (Suppl): S13–S19.

44 *Clinical, Microbiological, and Immunological Risk Indicators of Peri-implant Mucositis and Early Peri-implant Bone Loss*

- **40** Windael, S., Collaert, B., De Buyser, S. et al. (2021). Early peri-implant bone loss as a predictor for peri-implantitis: a 10-year prospective cohort study. *Clinical Implant Dentistry and Related Research* 23 (3): 298–308.
- **41** Acharya, A., Leung, M.C.T., Ng, K.T. et al. (2019). Peri-implant marginal bone loss rate pre- and postloading: an exploratory analysis of associated factors. *Clinical Oral Implants Research* 30 (5): 410–419.
- **42** Qian, J., Wennerberg, A., and Albrektsson, T. (2012). Reasons for marginal bone loss around oral implants. *Clinical Implant Dentistry and Related Research* 14 (6): 792–807.
- **43** Galindo-Moreno, P., Leon-Cano, A., Ortega-Oller, I. et al. (2015). Marginal bone loss as success criterion in implant dentistry: beyond 2 mm. *Clinical Oral Implants Research* 26 (4): e28–e34.
- **44** Tatarakis, N., Bashutski, J., Wang, H.L., and Oh, T.J. (2012). Early implant bone loss: preventable or inevitable? *Implant Dentistry* 21 (5): 379–386.
- **45** Romanos, G.E., Delgado-Ruiz, R., and Sculean, A. (2019). Concepts for prevention of complications in implant therapy. *Periodontology 2000* 81 (1): 7–17.
- **46** Pimentel, S.P., Fontes, M., Ribeiro, F.V. et al. (2018). Smoking habit modulates peri-implant microbiome: a case-control study. *Journal of Periodontal Research* 53 (6): 983–991.
- **47** Ata-Ali, J., Flichy-Fernandez, A.J., Alegre-Domingo, T. et al. (2016). Impact of heavy smoking on the clinical, microbiological and immunological parameters of patients with dental implants: a prospective crosssectional study. *Journal of Investigative and Clinical Dentistry* 7 (4): 401–409.
- **48** Zipprich, H., Weigl, P., Ratka, C. et al. (2018). The micromechanical behavior of implant-abutment connections under a dynamic load protocol. *Clinical Implant Dentistry and Related Research* 20 (5): 814–823.
- **49** Liu, Y. and Wang, J. (2017). Influences of micro-gap and micromotion of implant-abutment interface on marginal bone loss around implant neck. *Archives of Oral Biology* 83: 153–160.
- **50** Koutouzis, T. (2019). Implant-abutment connection as contributing factor to peri-implant diseases. *Periodontology 2000* 81 (1): 152–166.
- **51** Canullo, L., Penarrocha-Oltra, D., Soldini, C. et al. (2015). Microbiological assessment of the implant-abutment interface in different connections: cross-sectional study after 5 years of functional loading. *Clinical Oral Implants Research* 26 (4): 426–434.
- **52** Caricasulo, R., Malchiodi, L., Ghensi, P. et al. (2018). The influence of implant-abutment connection to peri-implant bone loss: a systematic review and meta-analysis. *Clinical Implant Dentistry and Related Research* 20 (4): 653–664.
- **53** Hsu, Y.T., Lin, G.H., and Wang, H.L. (2017). Effects of platform-switching on peri-implant soft and hard tissue outcomes: a systematic review and meta-analysis. *The International Journal of Oral & Maxillofacial Implants* 32 (1): e9–e24.
- **54** Sasada, Y. and Cochran, D.L. (2017). Implant-abutment connections: a review of biologic consequences and peri-implantitis implications. *The International Journal of Oral & Maxillofacial Implants* 32 (6): 1296–1307.
- **55** Saleh, M.H.A., Ravida, A., Suarez-Lopez Del Amo, F. et al. (2018). The effect of implant-abutment junction position on crestal bone loss: a systematic review and meta-analysis. *Clinical Implant Dentistry and Related Research* 20 (4): 617–633.
- **56** Penarrocha-Diago, M.A., Flichy-Fernandez, A.J., Alonso-Gonzalez, R. et al. (2013). Influence of implant neck design and implant-abutment connection type on peri-implant health. Radiological study. *Clinical Oral Implants Research* 24 (11): 1192–1200.
- **57** Hartman, G.A. and Cochran, D.L. (2004). Initial implant position determines the magnitude of crestal bone remodeling. *Journal of Periodontology* 75 (4): 572–577.
- **58** Zhang, Q. and Yue, X. (2021). Marginal bone loss around machined smooth neck implants compared to rough threaded neck implants: a systematic review and meta-analysis. *Journal of Prosthodontics* 30 (5): 401–411.
- **59** Messias, A., Nicolau, P., and Guerra, F. (2019). Titanium dental implants with different collar design and surface modifications: a systematic review on survival rates and marginal bone levels. *Clinical Oral Implants Research* 30 $(1): 20-48.$
- **60** Carrigy, J., Sharma, A., Perrotti, V., and Quaranta, A. (2021). Clinical outcomes of laser microtextured implants or abutments: a systematic review. *International Journal of Oral Implantology* 14 (2): 141–154.
- **61** Simons, W.F., De Smit, M., Duyck, J. et al. (2015). The proportion of cancellous bone as predictive factor for early marginal bone loss around implants in the posterior part of the mandible. *Clinical Oral Implants Research* 26 (9): 1051–1059.
- **62** Gurkan, A., Tekdal, G.P., Bostanci, N., and Belibasakis, G.N. (2019). Cytokine, chemokine, and growth factor levels in peri-implant sulcus during wound healing and osseointegration after piezosurgical versus conventional implant site preparation: randomized, controlled, splitmouth trial. *Journal of Periodontology* 90 (6): 616–626.
- **63** Peker Tekdal, G., Bostanci, N., Belibasakis, G.N., and Gurkan, A. (2016). The effect of piezoelectric surgery implant osteotomy on radiological and molecular parameters of peri-implant crestal bone loss: a randomized, controlled, split-mouth trial. *Clinical Oral Implants Research* 27 (5): 535–544.
- **64** Hertel, M., Roh, Y.C., Neumann, K., and Strietzel, F.P. (2017). Premature exposure of dental implant cover screws. A retrospective evaluation of risk factors and influence on marginal peri-implant bone level changes. *Clinical Oral Investigations* 21 (6): 2109–2122.
- **65** Suarez-Lopez Del Amo, F., Lin, G.H., Monje, A. et al. (2016). Influence of soft tissue thickness on peri-implant marginal bone loss: a systematic review and metaanalysis. *Journal of Periodontology* 87 (6): 690–699.
- **66** Radaelli, M.T.B., Federizzi, L., Nascimento, G.G. et al. (2020). Early-predictors of marginal bone loss around morse taper connection implants loaded with single crowns: a prospective longitudinal study. *Journal of Periodontal Research* 55 (2): 174–181.
- **67** Chen, Z., Lin, C.Y., Li, J. et al. (2019). Influence of abutment height on peri-implant marginal bone loss: a systematic review and meta-analysis. *The Journal of Prosthetic Dentistry* 122 (1): 14–21 e2.
- **68** Spinato, S., Stacchi, C., Lombardi, T. et al. (2019). Biological width establishment around dental implants is influenced by abutment height irrespective of vertical mucosal thickness: a cluster randomized controlled trial. *Clinical Oral Implants Research* 30 (7): 649–659.
- **69** Spinato, S., Stacchi, C., Lombardi, T. et al. (2020). Influence of abutment height and vertical mucosal thickness on early marginal bone loss around implants: a randomised clinical trial with an 18-month post-loading clinical and radiographic evaluation. *International Journal of Oral Implantology* 13 (3): 279–290.
- **70** Spinato, S., Galindo-Moreno, P., Bernardello, F., and Zaffe, D. (2018). Minimum abutment height to eliminate bone loss: influence of implant neck design and platform switching. *The International Journal of Oral & Maxillofacial Implants* 33 (2): 405–411.
- **71** Koutouzis, T., Gholami, F., Reynolds, J. et al. (2017). Abutment disconnection/reconnection affects peri-implant marginal bone levels: a meta-analysis. *The International Journal of Oral & Maxillofacial Implants* 32 (3): 575–581.
- **72** Bressan, E., Grusovin, M.G., D'Avenia, F. et al. (2017). The influence of repeated abutment changes on periimplant tissue stability: 3-year post-loading results from a multicentre randomised controlled trial. *European Journal of Oral Implantology* 10 (4): 373–390.
- **73** Tallarico, M., Caneva, M., Meloni, S.M. et al. (2018). Definitive abutments placed at implant insertion and never removed: is it an effective approach? A systematic review and meta-analysis of randomized controlled trials. *Journal of Oral and Maxillofacial Surgery* 76 (2): 316–324.
- **74** Wang, Q.Q., Dai, R., Cao, C.Y. et al. (2017). One-time versus repeated abutment connection for platformswitched implant: a systematic review and meta-analysis. *PLoS One* 12 (10): e0186385.
- **75** Schropp, L. and Wenzel, A. (2016). Timing of single implant placement and long-term observation of marginal bone levels. *European Journal of Oral Implantology* 9 (Suppl 1): S107–S122.
- **76** Majzoub, J., Chen, Z., Saleh, I. et al. (2021). Influence of restorative design on the progression of peri-implant bone loss: a retrospective study. *Journal of Periodontology* 92 (4): 536–546.
- **77** Dixon, D.R. and London, R.M. (2019). Restorative design and associated risks for peri-implant diseases. *Periodontology 2000* 81 (1): 167–178.
- **78** Sommer, M., Zimmermann, J., Grize, L., and Stubinger, S. (2020). Marginal bone loss one year after implantation: a systematic review of different loading protocols. *International Journal of Oral and Maxillofacial Surgery* 49 (1): 121–134.
- **79** Mattheos, N., Vergoullis, I., Janda, M., and Miseli, A. (2021). The implant supracrestal complex and its significance for long-term successful clinical outcomes. *The International Journal of Prosthodontics* 34 (1): 88–100.
- **80** Pesce, P., Canullo, L., Grusovin, M.G. et al. (2015). Systematic review of some prosthetic risk factors for periimplantitis. *The Journal of Prosthetic Dentistry* 114 (3): 346–350.
- **81** Delgado-Ruiz, R.A., Calvo-Guirado, J.L., and Romanos, G.E. (2019). Effects of occlusal forces on the peri-implantbone interface stability. *Periodontology 2000* 81 (1): 179–193.
- **82** Fretwurst, T., Nelson, K., Tarnow, D.P. et al. (2018). Is metal particle release associated with peri-implant bone destruction? An emerging concept. *Journal of Dental Research* 97 (3): 259–265.
- **83** Bonfante, E.A., Jimbo, R., Witek, L. et al. (2019). Biomaterial and biomechanical considerations to prevent risks in implant therapy. *Periodontology 2000* 81 (1): 139–151.
- **84** Veerman, E.C., van den Keybus, P.A., Vissink, A., and Nieuw Amerongen, A.V. (1996). Human glandular salivas: their separate collection and analysis. *European Journal of Oral Sciences* 104 (4 (Pt 1)): 346–352.
- **85** Siqueira, W.L., Salih, E., Wan, D.L. et al. (2008). Proteome of human minor salivary gland secretion. *Journal of Dental Research* 87 (5): 445–450.
- **86** Bostanci, N. and Belibasakis, G.N. (2018). Gingival crevicular fluid and its immune mediators in the proteomic era. *Periodontology 2000* 76 (1): 68–84.
- **87** Proctor, D.M. and Relman, D.A. (2017). The landscape ecology and microbiota of the human nose, mouth, and throat. *Cell Host & Microbe* 21 (4): 421–432.
- **88** Proctor, D.M., Shelef, K.M., Gonzalez, A. et al. (2020). Microbial biogeography and ecology of the mouth and implications for periodontal diseases. *Periodontology 2000* 82 (1): 26–41.

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- **89** Mark Welch, J.L., Rossetti, B.J., Rieken, C.W. et al. (2016). Biogeography of a human oral microbiome at the micron scale. *Proceedings of the National Academy of Sciences of the United States of America* 113 (6): E791–E800.
- **90** Edgerton, M., Lo, S.E., and Scannapieco, F.A. (1996). Experimental salivary pellicles formed on titanium surfaces mediate adhesion of streptococci. *The International Journal of Oral & Maxillofacial Implants* 11 (4): 443–449.
- **91** Quirynen, M., Vogels, R., Pauwels, M. et al. (2005). Initial subgingival colonization of 'pristine' pockets. *Journal of Dental Research* 84 (4): 340–344.
- **92** Leonhardt, A., Olsson, J., and Dahlen, G. (1995). Bacterial colonization on titanium, hydroxyapatite, and amalgam surfaces in vivo. *Journal of Dental Research* 74 (9): 1607–1612.
- **93** Frojd, V., Linderback, P., Wennerberg, A. et al. (2011). Effect of nanoporous TiO2 coating and anodized Ca2+ modification of titanium surfaces on early microbial biofilm formation. *BMC Oral Health* 11: 8.
- **94** Furst, M.M., Salvi, G.E., Lang, N.P., and Persson, G.R. (2007). Bacterial colonization immediately after installation on oral titanium implants. *Clinical Oral Implants Research* 18 (4): 501–508.
- **95** Lang, N.P., Berglundh, T., and Working Group 4 of Seventh European Workshop on P (2011). Periimplant diseases: where are we now? – consensus of the seventh European workshop on periodontology. *Journal of Clinical Periodontology* 38 (Suppl 11): 178–181.
- **96** Payne, J.B., Johnson, P.G., Kok, C.R. et al. (2017). Subgingival microbiome colonization and cytokine production during early dental implant healing. *mSphere* 2 (6): e00527–e00517.
- **97** Zheng, H., Xu, L., Wang, Z. et al. (2015). Subgingival microbiome in patients with healthy and ailing dental implants. *Scientific Reports* 5: 10948.
- **98** Tsigarida, A.A., Dabdoub, S.M., Nagaraja, H.N., and Kumar, P.S. (2015). The influence of smoking on the peri-implant microbiome. *Journal of Dental Research* 94 (9): 1202–1217.
- **99** Sanz-Martin, I., Doolittle-Hall, J., Teles, R.P. et al. (2017). Exploring the microbiome of healthy and diseased peri-implant sites using Illumina sequencing. *Journal of Clinical Periodontology* 44 (12): 1274–1284.
- **100** Cho, E.H., Park, K.S., Yang, M. et al. (2017). Laboratory identification of leptotrichia species isolated from bacteremia patients at a single institution. *Annals of Laboratory Medicine* 37 (3): 272–276.
- **101** Charalampakis, G. and Belibasakis, G.N. (2015). Microbiome of peri-implant infections: lessons from conventional, molecular and metagenomic analyses. *Virulence* 6 (3): 183–187.
- **102** Mombelli, A. and Lang, N.P. (2000). The diagnosis and treatment of peri-implantitis. *Periodontology 2000* 17: 63–76.
- **103** Mombelli, A., van Oosten, M.A., Schurch, E. Jr., and Land, N.P. (1987). The microbiota associated with successful or failing osseointegrated titanium implants. *Oral Microbiology and Immunology* 2 (4): 145–151.
- **104** Bower, R.C., Radny, N.R., Wall, C.D., and Henry, P.J. (1989). Clinical and microscopic findings in edentulous patients 3 years after incorporation of osseointegrated implant-supported bridgework. *Journal of Clinical Periodontology* 16 (9): 580–587.
- **105** Pontoriero, R., Tonelli, M.P., Carnevale, G. et al. (1994). Experimentally induced peri-implant mucositis. A clinical study in humans. *Clinical Oral Implants Research* 5 (4): 254–259.
- **106** Mombelli, A. and Decaillet, F. (2011). The characteristics of biofilms in peri-implant disease. *Journal of Clinical Periodontology* 38 (Suppl 11): 203–213.
- **107** Devides, S.L. and Franco, A.T. (2006). Evaluation of peri-implant microbiota using the polymerase chain reaction in completely edentulous patients before and after placement of implant-supported prostheses submitted to immediate load. *The International Journal of Oral & Maxillofacial Implants* 21 (2): 262–269.
- **108** Galassi, F., Kaman, W.E., Anssari Moin, D. et al. (2012). Comparing culture, real-time PCR and fluorescence resonance energy transfer technology for detection of Porphyromonas gingivalis in patients with or without peri-implant infections. *Journal of Periodontal Research* 47 (5): 616–625.
- **109** Al-Radha, A.S., Pal, A., Pettemerides, A.P., and Jenkinson, H.F. (2012). Molecular analysis of microbiota associated with peri-implant diseases. *Journal of Dentistry* 40 (11): 989–998.
- **110** Hultin, M., Gustafsson, A., Hallstrom, H. et al. (2002). Microbiological findings and host response in patients with peri-implantitis. *Clinical Oral Implants Research* 13 (4): 349–358.
- **111** Shibli, J.A., Melo, L., Ferrari, D.S. et al. (2008). Composition of supra- and subgingival biofilm of subjects with healthy and diseased implants. *Clinical Oral Implants Research* 19 (10): 975–982.
- **112** Belibasakis, G.N., Mir-Mari, J., Sahrmann, P. et al. (2016). Clinical association of *Spirochaetes* and *Synergistetes* with peri-implantitis. *Clinical Oral Implants Research* 27 (6): 656–661.
- **113** Salvi, G.E., Furst, M.M., Lang, N.P., and Persson, G.R. (2008). One-year bacterial colonization patterns of Staphylococcus aureus and other bacteria at implants and adjacent teeth. *Clinical Oral Implants Research* 19 (3): 242–248.
- **114** Persson, G.R. and Renvert, S. (2014). Cluster of bacteria associated with peri-implantitis. *Clinical Implant Dentistry and Related Research* 16 (6): 783–793.
- **115** Renvert, S., Lindahl, C., Renvert, H., and Persson, G.R. (2008). Clinical and microbiological analysis of subjects treated with Branemark or AstraTech implants: a 7-year follow-up study. *Clinical Oral Implants Research* 19 (4): 342–347.
- **116** Kumar, P.S., Mason, M.R., Brooker, M.R., and O'Brien, K. (2012). Pyrosequencing reveals unique microbial signatures associated with healthy and failing dental implants. *Journal of Clinical Periodontology* 39 (5): 425–433.
- **117** Dabdoub, S.M., Tsigarida, A.A., and Kumar, P.S. (2013). Patient-specific analysis of periodontal and peri-implant microbiomes. *Journal of Dental Research* 92 (12 Suppl): 168S–175S.
- **118** Maruyama, N., Maruyama, F., Takeuchi, Y. et al. (2014). Intraindividual variation in core microbiota in periimplantitis and periodontitis. *Scientific Reports* 4: 6602.
- **119** Sousa, V., Nibali, L., Spratt, D. et al. (2017). Peri-implant and periodontal microbiome diversity in aggressive periodontitis patients: a pilot study. *Clinical Oral Implants Research* 28 (5): 558–570.
- **120** Apatzidou, D., Lappin, D.F., Hamilton, G. et al. (2017). Microbiome of peri-implantitis affected and healthy dental sites in patients with a history of chronic periodontitis. *Archives of Oral Biology* 83: 145–152.
- **121** Gao, X., Zhou, J., Sun, X. et al. (2018). Diversity analysis of subgingival microbial bacteria in peri-implantitis in Uygur population. *Medicine (Baltimore)* 97 (5): e9774.
- **122** Yu, X.L., Chan, Y., Zhuang, L. et al. (2019). Intra-oral single-site comparisons of periodontal and peri-implant microbiota in health and disease. *Clinical Oral Implants Research* 30 (8): 760–776.
- **123** Papaioannou, W., Quirynen, M., Nys, M., and van Steenberghe, D. (1995). The effect of periodontal parameters on the subgingival microbiota around implants. *Clinical Oral Implants Research* 6 (4): 197–204.
- **124** Heitz-Mayfield, L.J.A. and Salvi, G.E. (2018). Periimplant mucositis. *Journal of Periodontology* 89 (Suppl 1): S257–S266.
- **125** Meyer, S., Giannopoulou, C., Cancela, J. et al. (2019). Experimental mucositis/gingivitis in persons aged 70 or over: microbiological findings and prediction of clinical outcome. *Clinical Oral Investigations* 23 (10): 3855–3863.
- **126** Schincaglia, G.P., Hong, B.Y., Rosania, A. et al. (2017). Clinical, immune, and microbiome traits of gingivitis and peri-implant mucositis. *Journal of Dental Research* 96 (1): 47–55.
- **127** Hajishengallis, G. and Lamont, R.J. (2012). Beyond the red complex and into more complexity: the

polymicrobial synergy and dysbiosis (PSD) model of periodontal disease etiology. *Molecular Oral Microbiology* 27 (6): 409–419.

- **128** Kumar, P.S., Leys, E.J., Bryk, J.M. et al. (2006). Changes in periodontal health status are associated with bacterial community shifts as assessed by quantitative 16S cloning and sequencing. *Journal of Clinical Microbiology* 44 (10): 3665–3673.
- **129** Hajishengallis, G., Darveau, R.P., and Curtis, M.A. (2012). The keystone-pathogen hypothesis. *Nature Reviews. Microbiology* 10 (10): 717–725.
- **130** Bostanci, N. and Belibasakis, G.N. (2012). Porphyromonas gingivalis: an invasive and evasive opportunistic oral pathogen. *FEMS Microbiology Letters* 333 (1): 1–9.
- **131** Ishihara, K. (2010). Virulence factors of *Treponema* denticola. *Periodontology 2000* 54 (1): 117–135.
- **132** Hajishengallis, G. (2014). Immunomicrobial pathogenesis of periodontitis: keystones, pathobionts, and host response. *Trends in Immunology* 35 (1): 3–11.
- **133** Bao, K., Bostanci, N., Thurnheer, T., and Belibasakis, G.N. (2017). Proteomic shifts in multi-species oral biofilms caused by *Anaeroglobus geminatus*. *Scientific Reports* 7 (1): 4409.
- **134** Frias-Lopez, J. and Duran-Pinedo, A. (2012). Effect of periodontal pathogens on the metatranscriptome of a healthy multispecies biofilm model. *Journal of Bacteriology* 194 (8): 2082–2095.
- **135** Smith, A.J., Jackson, M.S., and Bagg, J. (2001). The ecology of Staphylococcus species in the oral cavity. *Journal of Medical Microbiology* 50 (11): 940–946.
- **136** Marsh, P.D., Lewis, M.A.O., Rogers, H. et al. (2016). The mouth as a microbial habitat. In: *Oral Microbiology*, 6e (ed. Elsevier), 10–25. Churchill Livingstone.
- **137** Marsh, P.D., Lewis, M.A.O., Rogers, H. et al. (2016). Dental plaque. In: *Oral Microbiology*, 6e (ed. Elsevier), 81–111. Churchill Livingstone.
- **138** Williams, D.F. (2019). Specifications for innovative, enabling biomaterials based on the principles of biocompatibility mechanisms. *Frontiers in Bioengineering and Biotechnology* 7: 255.
- **139** Williams, D. (2003). Revisiting the definition of biocompatibility. *Medical Device Technology* 14 (8): 10–13.
- **140** Bosshardt, D.D., Salvi, G.E., Huynh-Ba, G. et al. (2011). The role of bone debris in early healing adjacent to hydrophilic and hydrophobic implant surfaces in man. *Clinical Oral Implants Research* 22 (4): 357–364.
- **141** Trindade, R., Albrektsson, T., and Wennerberg, A. (2015). Current concepts for the biological basis of dental implants: foreign body equilibrium and osseointegration dynamics. *Oral and Maxillofacial Surgery Clinics of North America* 27 (2): 175–183.

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- **142** Maenpaa, J., Soderstrom, K.O., Salmi, T., and Ekblad, U. (1988). Large atypical polyps of the vagina during pregnancy with concomitant human papilloma virus infection. *European Journal of Obstetrics, Gynecology, and Reproductive Biology* 27 (1): 65–69.
- **143** Atieh, M.A., Alsabeeha, N.H.M., Tawse-Smith, A., and Duncan, W.J. (2018). Piezoelectric versus conventional implant site preparation: a systematic review and meta-analysis. *Clinical Implant Dentistry and Related Research* 20 (2): 261–270.
- **144** Ozturk, V.O., Emingil, G., Bostanci, N., and Belibasakis, G.N. (2017). Impact of implant-abutment connection on osteoimmunological and microbiological parameters in short implants: a randomized controlled clinical trial. *Clinical Oral Implants Research* 28 (9): e111–e120.
- **145** Albrektsson, T., Dahlin, C., Jemt, T. et al. (2014). Is marginal bone loss around oral implants the result of a provoked foreign body reaction? *Clinical Implant Dentistry and Related Research* 16 (2): 155–165.
- **146** Trindade, R., Albrektsson, T., Galli, S. et al. (2018). Osseointegration and foreign body reaction: titanium implants activate the immune system and suppress bone resorption during the first 4 weeks after implantation. *Clinical Implant Dentistry and Related Research* 20 (1): 82–91.
- **147** Young, C.W., Lee, J.S., Le, H., and Smith, R.A. (2004). Surrogate markers of health after titanium dental implant placement. *Journal of Oral and Maxillofacial Surgery* 62 (11): 1413–1417.
- **148** Thomas, P., Iglhaut, G., Wollenberg, A. et al. (2013). Allergy or tolerance: reduced inflammatory cytokine response and concomitant IL-10 production of lymphocytes and monocytes in symptom-free titanium dental implant patients. *BioMed Research International* 2013: 539834.
- **149** Quabius, E.S., Ossenkop, L., Harder, S., and Kern, M. (2012). Dental implants stimulate expression of Interleukin-8 and its receptor in human blood--an in vitro approach. *Journal of Biomedical Materials Research. Part B, Applied Biomaterials* 100 (5): 1283–1288.
- **150** Harder, S., Quabius, E.S., Ossenkop, L. et al. (2012). Surface contamination of dental implants assessed by gene expression analysis in a whole-blood in vitro assay: a preliminary study. *Journal of Clinical Periodontology* 39 (10): 987–994.
- **151** Harder, S., Quabius, E.S., Meinke, F. et al. (2019). Changes in proinflammatory gene expression in human whole blood after contact with UV-conditioned implant surfaces. *Clinical Oral Investigations* 23 (10): 3731–3738.
- **152** Albrektsson, T., Buser, D., and Sennerby, L. (2012). Crestal bone loss and oral implants. *Clinical Implant Dentistry and Related Research* 14 (6): 783–791.
- **153** Pettersson, M., Kelk, P., Belibasakis, G.N. et al. (2017). Titanium ions form particles that activate and execute interleukin-1beta release from lipopolysaccharideprimed macrophages. *Journal of Periodontal Research* 52 (1): 21–32.
- **154** Olmedo, D., Fernandez, M.M., Guglielmotti, M.B., and Cabrini, R.L. (2003). Macrophages related to dental implant failure. *Implant Dentistry* 12 (1): 75–80.
- **155** Liljenberg, B., Gualini, F., Berglundh, T. et al. (1997). Composition of plaque-associated lesions in the gingiva and the peri-implant mucosa in partially edentulous subjects. *Journal of Clinical Periodontology* 24 (2): 119–123.
- **156** Abrahamsson, I., Berglundh, T., and Lindhe, J. (1998). Soft tissue response to plaque formation at different implant systems. A comparative study in the dog. *Clinical Oral Implants Research* 9 (2): 73–79.
- **157** Salvi, G.E., Aglietta, M., Eick, S. et al. (2012). Reversibility of experimental peri-implant mucositis compared with experimental gingivitis in humans. *Clinical Oral Implants Research* 23 (2): 182–190.
- **158** Heitz-Mayfield, L.J.A. and Salvi, G.E. (2018). Periimplant mucositis. *Journal of Clinical Periodontology* 45 (Suppl 20): S237–S245.
- **159** Gualini, F. and Berglundh, T. (2003). Immunohistochemical characteristics of inflammatory lesions at implants. *Journal of Clinical Periodontology* 30 (1): 14–18.
- **160** Lindhe, J., Berglundh, T., Ericsson, I. et al. (1992). Experimental breakdown of peri-implant and periodontal tissues. A study in the beagle dog. *Clinical Oral Implants Research* 3 (1): 9–16.
- **161** Carcuac, O. and Berglundh, T. (2014). Composition of human peri-implantitis and periodontitis lesions. *Journal of Dental Research* 93 (11): 1083–1088.
- **162** Duarte, P.M., de Mendonca, A.C., Maximo, M.B. et al. (2009). Differential cytokine expressions affect the severity of peri-implant disease. *Clinical Oral Implants Research* 20 (5): 514–520.
- **163** Venza, I., Visalli, M., Cucinotta, M. et al. (2010). Proinflammatory gene expression at chronic periodontitis and peri-implantitis sites in patients with or without type 2 diabetes. *Journal of Periodontology* 81 (1): 99–108.
- **164** Liu, Y., Liu, Q., Li, Z. et al. (2020). Long non-coding RNA and mRNA expression profiles in peri-implantitis vs periodontitis. *Journal of Periodontal Research* 55 (3): 342–353.
- **165** Belibasakis, G.N., Reddi, D., and Bostanci, N. (2011). Porphyromonas gingivalis induces RANKL in T-cells. *Inflammation* 34 (2): 133–138.
- **166** Serino, G., Turri, A., and Lang, N.P. (2013). Probing at implants with peri-implantitis and its relation to clinical
peri-implant bone loss. *Clinical Oral Implants Research* 24 (1): 91–95.

- **167** Lang, N.P., Wetzel, A.C., Stich, H., and Caffesse, R.G. (1994). Histologic probe penetration in healthy and inflamed peri-implant tissues. *Clinical Oral Implants Research* 5 (4): 191–201.
- **168** Schou, S., Holmstrup, P., Stoltze, K. et al. (2002). Probing around implants and teeth with healthy or inflamed peri-implant mucosa/gingiva. A histologic comparison in cynomolgus monkeys (Macaca fascicularis). *Clinical Oral Implants Research* 13 (2): 113–126.
- **169** Lekholm, U., Adell, R., Lindhe, J. et al. (1986). Marginal tissue reactions at osseointegrated titanium fixtures. (II) A cross-sectional retrospective study. *International Journal of Oral and Maxillofacial Surgery* 15 (1): 53–61.
- **170** Adonogianaki, E., Mooney, J., Wennstrom, J.L. et al. (1995). Acute-phase proteins and immunoglobulin G against Porphyromonas gingivalis in peri-implant crevicular fluid: a comparison with gingival crevicular fluid. *Clinical Oral Implants Research* 6 (1): 14–23.
- **171** Kaklamanos, E.G. and Tsalikis, L. (2002). A review on peri-implant crevicular fluid assays potential in monitoring and predicting peri-implant tissue responses. *Journal of the International Academy of Periodontology* 4 (2): 49–59.
- **172** Golub, L.M., Raisanen, I.T., Sorsa, T., and Preshaw, P.M. (2020). An unexplored pharmacologic/diagnostic strategy for peri-implantitis: a protocol proposal. *Diagnostics (Basel)* 10 (12): 1050.
- **173** Thierbach, R., Maier, K., Sorsa, T., and Mantyla, P. (2016). Peri-Implant Sulcus Fluid (PISF) Matrix Metalloproteinase (MMP) -8 Levels in Peri-Implantitis. *Journal of Clinical and Diagnostic Research* 10 (5): ZC34-8.
- **174** Esberg, A., Isehed, C., Holmlund, A., and Lundberg, P. (2019). Peri-implant crevicular fluid proteome before and after adjunctive enamel matrix derivative treatment of peri-implantitis. *Journal of Clinical Periodontology* 46 (6): 669–677.
- **175** Guncu, G.N., Akman, A.C., Gunday, S. et al. (2012). Effect of inflammation on cytokine levels and bone remodelling markers in peri-implant sulcus fluid: a preliminary report. *Cytokine* 59 (2): 313–316.
- **176** Schierano, G., Pejrone, G., Brusco, P. et al. (2008). TNF-alpha TGF-beta2 and IL-1beta levels in gingival and peri-implant crevicular fluid before and after de novo plaque accumulation. *Journal of Clinical Periodontology* 35 (6): 532–538.
- **177** Neely, A.L. and Maalhagh-Fard, A. (2018). Successful management of early peri-implant infection and bone

loss using a multidisciplinary treatment approach. *Clinical Advances in Periodontics* 8 (1): 5–10.

- **178** Jemt, T., Karouni, M., Abitbol, J. et al. (2017). A retrospective study on 1592 consecutively performed operations in one private referral clinic. Part II: peri-implantitis and implant failures. *Clinical Implant Dentistry and Related Research* 19 (3): 413–422.
- **179** Jemt, T. (2017). A retro-prospective effectiveness study on 3448 implant operations at one referral clinic: a multifactorial analysis. Part II: clinical factors associated to peri-implantitis surgery and late implant failures. *Clinical Implant Dentistry and Related Research* 19 (6): 972–979.
- **180** Shapoff, C.A. and Lahey, B.J. (2012). Crestal bone loss and the consequences of retained excess cement around dental implants. *The Compendium of Continuing Education in Dentistry* 33 (2): 94–96. 8-101; quiz 2, 12.
- **181** Romandini, M., Berglundh, J., Derks, J. et al. (2021). Diagnosis of peri-implantitis in the absence of baseline data: a diagnostic accuracy study. *Clinical Oral Implants Research* 32 (3): 297–313.
- **182** Carinci, F., Romanos, G.E., and Scapoli, L. (2019). Molecular tools for preventing and improving diagnosis of peri-implant diseases. *Periodontology 2000* 81 $(1): 41-47.$
- **183** Amengual-Penafiel, L., Cordova, L.A., Constanza Jara-Sepulveda, M. et al. (2021). Osteoimmunology drives dental implant osseointegration: a new paradigm for implant dentistry. *Japanese Dental Science Review* 57: 12–19.
- **184** Panagakos, F.S., Aboyoussef, H., Dondero, R., and Jandinski, J.J. (1996). Detection and measurement of inflammatory cytokines in implant crevicular fluid: a pilot study. *The International Journal of Oral & Maxillofacial Implants* 11 (6): 794–799.
- **185** Dereka, X., Mardas, N., Chin, S. et al. (2012). A systematic review on the association between genetic predisposition and dental implant biological complications. *Clinical Oral Implants Research* 23 (7): 775–788.
- **186** Agrawal, K.K., Anwar, M., Gupta, C. et al. (2021). Association of interleukin-1 gene polymorphism and early crestal bone loss around submerged dental implants: a systematic review and meta-analysis. *The Journal of the Indian Prosthodontic Society* 21 (2): 116–124.
- **187** Slotte, C., Lenneras, M., Gothberg, C. et al. (2012). Gene expression of inflammation and bone healing in peri-implant crevicular fluid after placement and loading of dental implants. A kinetic clinical pilot study using quantitative real-time PCR. *Clinical Implant Dentistry and Related Research* 14 (5): 723–736.

50 *Clinical, Microbiological, and Immunological Risk Indicators of Peri-implant Mucositis and Early Peri-implant Bone Loss*

- **188** Kim, D.J., Kim, S.K., Cha, J.K. et al. (2019). Clinical factors and cellular responses of in situ human alveolar bone-derived mesenchymal stromal cells associated with early periimplant marginal bone loss: a prospective cohort pilot study. *Implant Dentistry* 28 (5): 421–429.
- **189** Yang, L., Zhu, Q., Gong, J. et al. (2018). CyPA and Emmprin play a role in peri-implantitis. *Clinical Implant Dentistry and Related Research* 20 (2): 102–109.
- **190** Sayardoust, S., Omar, O., Norderyd, O., and Thomsen, P. (2017). Clinical, radiological, and gene expression analyses in smokers and non-smokers, Part 2: RCT on the late healing phase of osseointegration. *Clinical Implant Dentistry and Related Research* 19 (5): 901–915.
- **191** Sayardoust, S., Omar, O., and Thomsen, P. (2017). Gene expression in peri-implant crevicular fluid of smokers and nonsmokers. 1. The early phase of osseointegration. *Clinical Implant Dentistry and Related Research* 19 (4): 681–693.
- **192** Sorsa, T., Mantyla, P., Ronka, H. et al. (1999). Scientific basis of a matrix metalloproteinase-8 specific chair-side test for monitoring periodontal and peri-implant health and disease. *Annals of the New York Academy of Sciences* 878: 130–140.
- **193** Arakawa, H., Uehara, J., Hara, E.S. et al. (2012). Matrix metalloproteinase-8 is the major potential collagenase in active peri-implantitis. *Journal of Prosthodontic Research* 56 (4): 249–255.
- **194** Kivela-Rajamaki, M., Maisi, P., Srinivas, R. et al. (2003). Levels and molecular forms of MMP-7 (matrilysin-1) and MMP-8 (collagenase-2) in diseased human periimplant sulcular fluid. *Journal of Periodontal Research* 38 (6): 583–590.
- **195** Lahteenmaki, H., Umeizudike, K.A., Heikkinen, A.M. et al. (2020). aMMP-8 point-of-care/chairside oral fluid technology as a rapid, non-invasive tool for periodontitis and peri-implantitis screening in a medical care setting. *Diagnostics (Basel)* 10 (8): 562.

Metallic Nanoparticles as a Risk Factor for Peri-implant Diseases

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Dental Implants

Usage of dental implants in the treatment of edentulism has revolutionized dentistry since the discovery of osseointegration of Per-Ingvar Branemark in the 1960s [1]. During the following decade, the development of the modern dental implant and treatment protocols was made by the Swedish and a Swiss research group simultaneously [2–7]. Previous implants that had been used for rehabilitation of edentulism did not show very good success rates, and at the introduction of the modern titanium implant by P-I Branemark, it was not well received by the dental society [8, 9]. Previous implants had been made of different alloys mainly cobalt–chromium (Co–Cr), with a relatively low success rate [10]. While the implant introduced by Professor Brånemark was made of commercial-pure titanium (CP Ti) with a purity of 99–99.5% [9]. CP Ti is alloyed with only oxygen divided into grade 1–4 depending on the oxygen content, where grade 1 is the weakest [11]. Higher grades of Ti are alloyed with other elements than oxygen and the most commonly used in dental implants are Ti–6Al–4V or other Ti alloys with Zr [12]. Also, Niobium and Tantalum are non-toxic elements favorable for usage as alloying elements with Ti to improve mechanical properties [13]. Usage of other Ti alloys than CP Ti is mainly to make it possible to reduce the diameter with a maintained strength of the implant, making it possible to install implants in an area with a reduced bone width [12, 14]. As materials for endosseous implants, c.p. Ti and Ti alloys dominate the current market. However, there is ongoing research into other materials, e.g. zirconia and PEEK (polyetheretherketone), for usage in dental implants. Implants made of Zirconia might be a material competitor to Ti in the future, but more in vivo research is needed before transition to a

different implant material than Ti can be recommended [15]. At the beginning of the modern implant era, solely c.p Ti was used in the implants and abutments. Supra constructions were full arch prostheses made of acrylic with a framework made of gold (Au). Today the core for the framework varies from different metallic alloys to ceramic materials, mainly different variants of zirconia. While usage of dental Au as materials has vanished due to cost and the development of modern manufacturing techniques used instead of casting.

A now historical conference held in Toronto, Canada, 9–11 May 1982 changed the dental community's thoughts about dental implants and are today called the start of the modern dental implant era [16, 17]. At this conference, P.-I. Branemark presented long time follow-up of his patient material of edentulous mandibles reconstructed with a fixed prosthesis anchored to titanium implants [3, 4]. After this conference, implant treatment and research have exploded with thousands of papers in the subject published today. At the start of the implant era, this treatment was only performed by specialists in oral surgery and prosthetic dentistry. Today, it is seen as a standard treatment to replace lost teeth used also by general dentists. The implant market has become a billion-dollar market with an estimate of 15–20 million implants installed annually worldwide [18]. Dental caries and periodontal problems are still two major diseases that haunts humans with a prevalence in studies of 35 and 46 % for the whole population, respectively [19–21]. Edentulism among low- and high-income countries differ between 10 and 35% and are expected to increase due to a longer life expectancy for humans [19]. So, the dental implant market has a growth potential between 5 and 11% annually in the coming five years. The possibility to replace lost teeth by a fixed prosthesis anchored to dental implants instead of a removable denture has improved the quality of life for many patients [22–25].

Peri-implantitis is an inflammatory disease, causing inflammation and degradation of the supporting tissue with a progressive loss of bone around a dental implant [26]. This inflammatory condition was first reported by Levignac in 1965 and described under the term periimplantation osteolysis-, periimplantosis – periimplantitis [27]. Peri-implantitis have been reported among individuals and implants with a prevalence of 19.83% (95% Confidence Interval (*CI*) 15.38–24.27) and 9.25% (*CI* 7.57–10.93), respectively. The criteria of peri-implantitis have been heavily discussed with a wide range in the prevalence. Today, a prevalence around 20% based on subjects is accepted. Taking this number in consideration means that every fifth patient treated with dental implants will have problems with inflammation and degradation of the supporting tissue around one or more implants. With those facts, it is of great importance to find contributing factors to peri-implantitis disease and try to avoid placing implants in patients with a high risk of failure. Also, try to find a solution to treat or minimize the damages caused by this inflammatory disease. As periodontal pathogens also are associated with peri-implantitis, development of antibacterial surfaces and improved mucosal seal are important characteristics to reduce bacterial adhesion and maintain a healthy soft tissue around the implant [28, 29].

Peri-implant mucositis is an inflammation in the soft tissue around a dental implant, without degradation of the supporting bone. It is a common condition among individuals treated with dental implants, with a prevalence based on implant level and patient level of 29.48% (*CI* 22.65–36.32) and 46.83% (*CI* 38.30–55.36), respectively [30]. Bacterial induced peri-implant mucositis is a reversible condition if a good plaque control is established [31]. However, other factors than bacterial factors can induce an inflammation in the peri-implant tissue e.g. excess cement, to deep placement of the implant, unsuitable implants, and poor surgical technique [32]. Inflammation in the peri-implant mucosa caused by these factors is not possible to influence with better plaque control and if they are not handled properly destruction of the supporting tissue and bone are inevitable.

In a follow-up study of 10.092 implants placed between 1980 and 2014 at a specialist clinic in Sweden, it was found that overall, 642 implants (6.36%) failed, of which 176 (1.74%) were lost prior to second-stage surgery [33]. This shows that dental implants are a treatment for edentulism with a high survival rate over time. However, when success rate is used as the outcome instead of survival the outcome looks a little different. A review study reported a range of success between 34.9 and 100% with a mean of 89.7% (± *SD* 10.2%) and a range for survival between 73.4 and 100% with a mean of 94.6% $(\pm SD 5.97%)$ [34]. Success rate for dental implants have been defined with some different measurements over

time, which makes it harder to compare outcome of success between different studies [8, 10, 35, 36].

Titanium

Ti is a chemical element with the atomic number 22 in the periodic system. It is categorized as a shiny transition metal with a close-packed hexagonal crystal structure. It is the ninth most common element in the crust of the earth [37]. Ti was first discovered in 1791 by the geologist William Gregor; this new element was in a magnetic black sand [38]. But not until 1910, it was purified to 99.9% purity by metallurgist Matthew Hunter [39]. Ti has commercially become widely used during the second half of the twentieth century, used in items such as aircrafts, gas turbine engines, bicycles, and golf clubs. Ti is also used in various medical appliances, such as dental and orthopedic implants, metalceramic reconstructions, removable partial dentures, needles, pacemaker leads, sutures, ligature clips, orthodontic appliances, and eyeglasses frames. This wide range of applications is due to the material's combination of strength, unique density, and corrosion resistance [37]. Due to these characteristics, Ti is advantageous compared to materials such as aluminum, steel, and other highperformance alloys. Ti is very reactive, and a thin titanium dioxide (TiO₂) layer (~3–5nm) covers Ti surfaces when exposed to air [40]. This oxide layer on the surface pacifies the metal and makes it very corrosion-resistant. Also, interactions between the metal and biomolecules are prevented by the oxide layer. While interactions between the oxide layer and biomolecules will occur, for instance different types of bonding e.g. van der Waals bonding, <4.184kJ/ mol; hydrogen bonding, ~4.184–41.84kJ/mol; and covalent and ionic bonding, 41.84–418.4kJ/mol [41]

American Society for Testing and Materials (ASTM) classifies c.p. Ti in five grades (gr.; 1–4 and 7) based on the concentration of oxygen (O; 0.18–0.40wt.%) and iron (Fe; 0.2–0.5wt.%) content. Ti impurities include nitrogen (N; 0.03–0.05wt.%), carbon (C; 0.1wt.%), and hydrogen (H) $(0.015 \text{ wt.}\%)$ [42]. Ti grades, gr. 1 is the purest and softest form, with a tensile strength of 240 MPa up to gr. 4 with a tensile strength of 515MPa. Grade 2 of c.p. Ti is the most commonly used in industrial services because it offers a good combination of high corrosion and erosion resistance, good cold formability, and excellent weldability [43].

Immune System

The immune system has evolved through millennia in the evolution of mankind, with powerful defense mechanisms to protect the host against a large variety of invaders that could enter the body [44]. A working immune system is essential for the survival of the host, not only for the protection against invading pathogens (bacteria, viruses, and parasites), but also for tissue repair and eradication of damaged cells, that otherwise could evolve into tumor cells [45].

To prevent entry into the body for pathogens is the main function for the immune system. The skin could be seen as a part of the immune system; there it works as the first barrier for pathogens to enter the body [44]. An intact skin barrier works as a good protection against pathogens, while the mucosa of the mouth and gut is easier for pathogens to penetrate [44]. The immune system host defense is divided into two major groups, the innate and the adaptive immune system. There the innate immune system is the first defense against pathogens, while the adaptive immune system is more specialized to recognize specifically molecules called antigen [45]. Antigens represent a large variety of both of foreign origin (pathogens, proteins, or molecules), but also self-molecules that can provoke autoimmune responses [44].

Innate and Adaptive Immune System

The main function of the innate system (fast immune response) is to be the first responder to a pathogen or foreign molecule entering the body. This type of immune response is immediate and consists of a number of different types of cells, macrophages, dendritic cells, mast cells, neutrophil-, eosinophil-, and basophil granulocytes, and natural killer cells [45]. Also, the epithelial barrier, natural antibodies, and complement factors are important in the innate immune response [45]. Some of the cells in the innate immune system are present in the tissue, while others are recruited to the area of an invading pathogen or if a tissue damage occurs. Macrophages, dendritic cells, and mast cells are these types of guardian cells that recruit other cells to the location of e.g. an infesting pathogen that entered the body. Macrophages, dendritic cells, and neutrophil granulocytes are phagocytes, where the first two also have antigen-presenting capability of antigens to the adaptive immune cells.

Adaptive immunity is the late immune response, which takes hours till days to initiate a response against the antigen. The immune cells consist of T- and B- lymphocytes [45]. The adaptive immunity is divided into humoral- and cellmediated immunity. Humoral immunity is antibodies, proteins produced and secreted by B-lymphocytes, to block infections by binding to extracellular antigens. Activation of B-lymphocytes leads to the effector function, neutralization of microbes, activation of phagocytes, and complement factors [44, 45]. While intracellular antigen, either phagocytized or an infected cell (e.g. viruses) activates cell-mediated immunity. This cell-mediated immunity can work through a phagocyte presenting the antigen of a phagocytized microbe to a T-helper lymphocyte that secrete cytokines to active the

phagocyte to destroy the ingested microbe [45]. Effector function of activation of T-helper lymphocytes not only consist of macrophage activation and inflammation, but also activation of B-lymphocytes. While a virus-infected cell needs to be killed to stop the virus from using the cell as "factory" for the replication of the virus [44, 45]. This death mechanism is mediated through a cytotoxic T-lymphocyte that binds to a receptor where the cell expresses viral antigen that initiates cell death to stop the virus replication. All these effector functions need to be regulated thoroughly so that the immune system acts against foreign antigens, but not the host's own cells and proteins [44, 45]. Regulatory T-lymphocytes have an important role to initiate a suppression of the immune response to prevent a too powerful response that could harm the host [44].

Inflammation

Inflammation is a series of immune reactions in response to an infection or tissue injury [46, 47]. Clinical signs of inflammation are calor, rubor, tumor, and dolor in the affected soft tissue [46]. Increased heat (calor) and redness (rubor) correlate to an increase of local blood flow in the soft tissue, and swelling (tumor) by increased permeability and/or leakage of plasma proteins and fluids into the tissue, pain (dolor) are caused by release of various of inflammatory mediators e.g. prostaglandins, bradykinin, and cytokines [46, 48].

Inducer of inflammation can be divided into two main groups exogen and endogenous [47]. Further, exogen inducers can be divided into microbial and non-microbial, and endogenous in cell-, tissue-, plasma, and extracellular matrix (ECM) derived [47]. Inflammation caused by exogen inducers as e.g. microbes, viruses, or particles are more well studied than endogenous-induced inflammation associated with many modern human diseases as e.g. atherosclerosis, diabetes, obesity, and rheumatoid arthritis [47].

Blood leukocytes (e.g. monocytes and neutrophils) are normally circulating in the bloodstream and only present in small numbers in healthy tissue. Eradicate of bacteria not only by phagocytosis, but also clearance of damaged tissue are performed by neutrophil granulocytes [49]. Neutrophils are the most common phagocytes circulating in the bloodstream. During inflammation, monocytes circulate in the blood are recruited to extravascular tissue and differentiated into macrophages. A breach of the first defense barrier, the epithelium, by microbes will be detected by dendritic cells, macrophages, and mast cells [49]. These cells will release proinflammatory cytokines, e.g. IL-1 and TNF- α , which are essential in the recruitment of inflammatory cells to the infected area. The release of these cytokines causes the endothelium to express adhesion molecules directed toward leukocytes in the bloodstream used for

recruitment of these cells [50]. Endothelial cells and macrophages will produce chemokines to render the endothelium more permeable to adhered leukocytes and allow them to extravasate and migrate to the infected tissue [49]. The cytokine IL-1 will induce acute inflammation and fever [51]. Interferon-gamma (IFN-γ) production by natural killer cells is induced by IL-12 produced by macrophages after phagocytosis. IFN-γ will then activate the macrophages to kill phagocytosed microbes [49].

Naive CD4⁺ will differentiate into T-helper cells type 1 (Th1) or Th2 effector cells when encountering an antigen [52, 53]. These different strains of T cells will activate macrophages to polarize into M1 (inflammatory) and M2 (antiinflammatory) depending on if they are activated by Th1 or Th2 cells [52]. Th1 cells will drive macrophage polarization to M1 phenotype that will activate cytokine release and an inflammatory response [52, 53]. While activation of macrophages by Th2 cells will polarize to a M2 phenotypes and initiate tissue repair and wound healing. Recent knowledge has shown that M1 and M2 macrophage phenotype present in the tissue show a higher diversity with a complex response to factors present in the microenvironment [54, 55].

Phagocytosis

Élie Metchnikoff was the first to describe the phenomena of phagocytosis in invertebrates in 1883 [56]. Phagocytosis is the process of intracellular ingestion and elimination of microbes, substances, or apoptotic cells that occurs in macrophages and neutrophils [57].

Phagocytosis starts with extension of the cell membrane around a microbe, which is initiated by binding of a microbe or particle to a PAMP or DAMP receptor [49]. This process is followed by closure of the plasma cell membrane around the bound microbe or particle, which is then pinched off into a vesicle called a phagosome [58]. Lysosomes, containing reactive oxygen species (ROS), nitric oxide (NO), and lysosomal proteases, are fused to the phagosome, forming a phagolysosome. ROS, NO, and lysosomal proteases are released in the phagolysosome to surround the ingested microbe or substance to decompose it [49]. The membrane of the phagolysosome will break if the decomposition fails, and the toxic lysosomal enzymes and proteases will leak into the cytosol. Nucleotide oligomerization domain (NOD) receptors in the cytosol will be activated by leakage of these toxic components. These receptors react to pathological changes in the cytosol to initiate the production of IL-1β via the assembly of a protein complex called an inflammasome [59]. The name inflammasome is derived from the ability of the complex to trigger both inflammation and cell death through apoptosis and pyroptosis [60, 61].

Inflammasomes

Inflammasomes are large multimeric protein complexes assembled in the cytosol of the cells working as a defense mechanism upon triggering from pathogen-associated molecular patterns (PAMPs) or danger-associated molecular patterns (DAMPs) [59, 62–64]. Inflammasomes regulate maturation and release of pro-inflammatory cytokines as interleukin (IL) – 1 β and IL – 18 which are an essential part of the innate immune system [63, 65]. Also, pyroptosis for eradication of infected or cells sensing internal danger is an essential function initiated by the inflammasomes [64]. These protein complexes are a sophisticated cell system to control the immune response against an infection or a danger signal. There are a number of subsets of inflammasomes and in this chapter, only the NLRP3 inflammasome will be discussed. These inflammasomes are composed by three domains: nucleotide-binding and oligomerization (NACHT) domain; followed by C-terminal leucine-rich repeats (LRRs); and N-terminal caspase recruitment (CARD), or pyrin (PYD) domains [63, 64]. Autoregulation and ligand sensing are believed to be the function for LRRs, whereas downstream signaling by protein–protein interactions is mediated by CARD and PYD domains [63]. Caspase-1, also known as IL-1 converting enzyme (ICE), is synthesized as an inactive proenzyme, which is activated by cleavage through the assembly of the inflammasome complex [66, 67]. NLRP3 inflammasome is the most characterized and consists of three components: NLRP3 scaffold; apoptosisassociated speck-like protein containing a caspase recruitment domain (ASC); and caspase-1 [59, 63, 64]. Activation of NLRP3 is initiated by a wide range of stimuli initiating: relocation of NLRP3 to the mitochondria; generation of reactive oxygen species (ROS); potassium efflux out of the cell; release of mitochondrial DNA or cardiolipin; and lysosomal destabilization leading to release of cathepsins into the cytosol [59]. Assembly of the NLRP3 inflammasome is initiated by the activation of NLRP3 leading to activation of caspase-1 and cleavage of pro-IL-1β into bioactive IL-1β [59, 68]. A schematic illustration of particle-induced inflammasome activation is shown in Figure 5.1.

Particle-induced activation of the NLRP3 inflammasome is believed to be initiated by rupture of phagolysosomes and leakage of the content into the cytosol e.g. proteolytic enzymes and the endosomal-lysosomal protease cathepsin B [69–71]. This protease works as an internal danger signal and is required to initiate the assembly of the NLRP3 inflammasome [72]. IL-1 β is a potent cytokine that affects every organ in the human body by the initiation of immune responses through e.g. fever, acute phase proteins, hypothalamic-pituitary-adrenal (HPA) axis, chemokines, adhesion molecules, interferon-gamma (IFN-γ), and

Figure 5.1 Schematic illustration of particle-induced NLRP3 inflammasome activation in a macrophage. Upregulation of NF-κβ in the nucleus is initiated through PAMP activation of the TLR. NF-κβ expression activates the immune system by macrophage secretion of TNF-α and IL-6. Also, upregulation of intracellular of pro-IL-1β and inflammasome genes as inactive NLRP3. Phagocytized particles will work as the second signal to activate assembly of the NLRP3 inflammasome. Phagosomes and lysosomes will form a phagolysosome where lysosomal products such as e.g. proteolytic enzymes and ROS will try to degrade the phagocytized particles. Due, to the stability of some particles (e.g. silica, asbestos, and TiO₂) degradation of these molecules will lead to rupture of the phagosome membrane. Leakage of lysosomal content into the cytosol with release of cathepsin B as the result, which works as a danger signal for the cell. Assembly of the NLRP3 inflammasome components NLRP3, ASC and pro-caspase-1 are initiated by this danger signal. Caspase-1 is activated through the assembly of the inflammasome, where the activated enzyme cleaves pro-IL-1β into bioactive IL-1β. Also, activated caspase-1 will add a N-terminal to GSMD initiating pore formation in the cell membrane. Through this pore, bioactive IL-1β can be released, which will activate inflammation. These pores will also allow Ca²⁺ influx to the cell, which is believed to be an important component to initiate cell death by pyroptosis.

IL-17 [73–75]. To protect the organism from a strong immune response induced by release IL-1β, two signals are needed to activate this potent cytokine [59, 76]. Expression of pro-IL-1β and NLRP3 are initiated by upregulation of nuclear factor kappa B (NF-κB) in the cell nucleus by activation through the toll-like receptors [59, 76]. This is not enough to activate NLRP3 but works as a first signal with upregulation of the proteins in cell cytosol, but dependent on a secondary signal initiated by e.g. silica, asbestos, cholesterol crystals, or Ti to activate the assembly of the NLRp3 inflammasome [59, 63, 69, 76–78]. A synergistic effect has been shown when macrophages are primed with

lipopolysaccharides before exposure to Ti or $TiO₂$ on the NLRP3 inflammasome activated IL-1β release [77, 79].

Activation of the NLRP3 inflammasome, maturation, and release of bioactive IL-1β will trigger inflammation and pyroptosis of the cell [80]. Activation of the protein Gasdermin D (GSDMD) with a N-terminal to GSDMD-NT by caspase-1 is required for IL-1β secretion and pyroptosis to occur [80–82]. Pyroptosis and IL-1β are initiated by pore formation in the cell membrane by GSDMD-NT [81, 83]. Cell death caused by pyroptosis is not fully understood, but it has been proposed that influx through pores formed in the cell membrane by GSDMD-NT might be a factor [81, 84].

Foreign Body Reaction

Implantation of a biomaterial into living tissue will first initiate an acute inflammation and in some cases lead to a chronic inflammation/granulation tissue formation, which can start an encapsulated foreign body reaction [85, 86]. Foreign body reactions are inflammatory responses and wound healing after implantation of a foreign material into the body [87]. Biocompatibility of a biomaterial is dependent on protein adhesion; macrophage/leukocyte adhesion and activation; activation of complement factors, where activation of macrophages plays an essential role [85]. In granulomas formed around an implanted biomaterial, giant cells are formed by fusion of macrophages into multinucleated cells [85, 88]. Foreign-body giant cells (FBGCs) are one type of giant cells derived from monocytes and the end stage of monocyte differentiation [88]. Giant cells are multinucleated cells that originated from fusion of macrophages into one gigantic cell with two different types of morphological classification, Langhans giant cells and FBGCs [88]. Langhans giant cells are multi-nucleoid cells, first described by the German pathologist Theodor Langhans in granulomas of tuberculosis [89]. Although both types of giant cells are derived from monocytes or monocyte progenitor cells, there are clear differences in the way they are formed. Where formation of FBGCs is induced by IL-4 and IL-13, while formation of Langhans giant cells by interferon-γ [88]. A clear difference in the characteristics is the lymphocyte collar seen in granulomas with Langhans giant cells, which is not present in FBGCs granulomas [88]. This is typical for this particle-induced granulomas that are non-immunological showing an absence of lymphocytes in the lesions [44]. They can also be distinguished by the number of nuclei, where FBGCs have >20 nuclei, while Langhans giant cells have less than twenty [88]. Other multi-nucleated cells as osteoclasts and the HIV-1-induced $CD4^+$ -T-cell derived syncytia which also can be considered as giant cells [88, 90]. Osteoclasts are cells involved in both natural bone remodeling and pathological bone resorption processes, which is contrary to other types of giant cells that only are present in pathological processes. Surface properties of the implanted biomaterial play an important role in foreign body reaction during the initial stage of healing after implantation [87].

It has been proposed that osseointegration of a dental implant is a foreign body response to the material causing a chronic inflammation and embedding of the implant with bone to separate it from the body [32]. Also, peri-implantitis is a disbalance in the foreign body response causing marginal bone loss around the implant leading to a secondary biofilm-driven infection of the exposed implant surface [32]. Albrektsson et al. propose that a foreign-body reaction will

occur during the healing phase after implant placement and to achieve a successful outcome a foreign-body equilibrium has to be established [32]. The authors suggest that this equilibrium can be disturbed by various local patient-dependent factors, cement particles, unsuitable implants, and poor surgical techniques, leading to chronic inflammation, which might in turn lead to failed osseointegration (early failure), marginal bone loss, and eventually implant loss (late failure) [32]. One argument opposing this hypothesis is that periimplantitis consistently begins with marginal loss. If the sole factor would be an imbalance in the foreign body reaction leading to peri-implantitis, bone loss could originate from any point on the implant surface, but this is not observed in practice [91–95].

Degradation Products from Implants

Nanoparticles and the Influence of Immune Cells

Nanoparticle (NP) is defined as matter with a size from 100nm down to the size of atoms 0.2 nm (2 Å) [96, 97]. Usage of nanotechnology in a wide range of areas has been developed during the last decades, raising concern about health hazards of those ultrafine particles [98–101]. Particles found inside a cell are normally phagocytized or enter the cell by endocytosis, while NP can enter the cell by passing the cell membrane [102]. In body fluids, NP attracts protein which leads to a corona of proteins attached to the particles surface defining the biological property of the NP [103]. Metal–protein aggregates formed by Ti show different morphology depending on if it is the ionic-, Ti NP, or $TiO₂$ Figure 5.2.

 $TiO₂$ NP can cross epithelial cells reaching the bloodstream and be transported to every organ in the body as well as entering the central nervous system [104]. These NPs can not only enter the cells, but also enter the nuclei and mitochondria interacting with cytoplasmic proteins $[104]$. Toxicological, TiO₂ NP induces oxidative stress in the cells that cause inflammation, cell damage, genotoxicity, and adverse immune response [105, 106]. Also, indications that $TiO₂ NP$ can cause chromosomal damage and DNA strand breaks into cells that have been shown [107]. Due to the diversity and reactivity of these particles, the immunological response and cell damages are dependent on physical and chemical properties of the NPs [105, 106]. Reports have suggested that the rutile form of $TiO₂$ NPs can not easily enter the cells, concluding that the crystal form rather than the particle size is the most important factor for the toxicological effect on cells [108]. While other studies have demonstrated that the shape and size of the particles

Figure 5.2 TEM images showing aggregates formed by (a) Ti ions with proteins; (b) Ti NP with proteins; and (c) TiO₂ NP with proteins. A clear difference in the morphology of the formed metal–protein aggregates can be seen. If the difference in the structure of the formed metal–protein aggregates will initiate different immune responses is unknown.

are important factors for the cellular internalization pathways by endocytosis and are favorable by rod-like formed particles compared to spherical $[109]$. TiO₂ is used in a large variety of consumer products as e.g. pigments in personal care products and food, makeup, and sun creams. As new knowledge is discovered about hazards to human health by exposure to $TiO₂ NPs$, the effect on human health of these highly reactive particles should be examined thoroughly [110]. Also, usage of produced NPs in human consumer products should be limited until the health aspect of exposure is better known.

Inhalation of asbestos and silica particles is linked to inflammation in lung tissue, fibrosis, and lung cancer [78]. Inflammation in the lungs by inhalation of silica and asbestos particles is associated with phagocytosis of these particles by macrophages resulting in activation of the NLRP3 inflammasome and release of IL-1β [78]. Inhalation of $TiO₂$ NPs has been found to cause similar inflammation in lung tissue as silica and asbestos, resulting in emphysemalike damages [79, 111].

Gene expression of toll-like receptors (TLRs) 4 has been shown to be downregulated after 24 hours of exposure to Ti particles, possibly to inhibit an excessive inflammation [112]. However, this might lead to a consequence where cells and tissue are more susceptible to pathogens [112]. Bone marrow cells show a decrease in osteoclast proliferation, alkaline phosphatase activity, and osteocalcin production when challenged with polymethylmethacrylate (PMMA) or ultra-high molecular weight polyethylene (UHMWPE) particles [113, 114]. While other studies show that receptor activator of nuclear factor-κβ ligand (RANKL), interleukin (IL)-6, IL-8, IL-10, C-X-C motif chemokine ligand 10 (CXCL10), monocyte chemoattractant protein (MCP) 1, and monokine induced by

gamma interferon (MIG) are overexpressed then exposed to UHMWPE leading to periprosthetic osteolysis [115].

Orthopedic Implants

Osteolysis around an orthopedic implant caused by wear debris is a well-known complication and a major problem, causing a vast majority of prosthesis revision surgery (75%) [116]. This degradation around a hip prosthesis without a previous infectious cause, also called aseptic osteolysis, was first described by Harris et al. [116, 117]. This particleinduced osteolysis is caused by the activation of inflammatory cells and secretion of proinflammatory cytokines (e.g. IL-1β, IL-6, TNF- α , and prostaglandin E2 (PGE2) [118–120]. Inflammation in the tissue adjacent to the implant will be induced by the release of these cytokines, which in turn will activate the receptor activator of nuclear factor kappa beta ligand (RANKL). RANKL will activate osteoclasts and bone resorption will be induced. Histomorphometric analysis of failed implant sites showed an increased contact between bone and CD11 -positive cells, in sites where radiographic signs of osteolysis were present [121]. Systemic distribution of metallic wear particles was found in liver, spleen, and regional lymph nodes postmortem with a higher concentration in patients with a revised hip prosthesis compared to patients examined with a primary prosthesis [122]. Intoxication by cobalt with various organs and parts of the nerve system affected has been reported from failed hip prosthesis made of cobalt-chromium alloys [123–127]. Those rare complications correlated to wear particles released from hip prosthesis seem to be more frequent among patients with a fractured ceramic prosthesis and where radiologic and clinical signs of local inflammation are present [128, 129]. Orthopedic implants show many

similarities with dental implants but also some differences. One of the major differences is that orthopedic prostheses are embedded in a sterile compartment, while dental implants are exposed to the oral environment through the peri-implant mucosa. Additionally, orthopedic prostheses are much larger and exposed to movement, which induces much more extensive particle abrasion from the surface than that from the surface of dental implants. Inflammatory reactions similar to those observed in peri-implantitis occur around orthopedic implants. Implant revisions contribute to these inflammatory problems, which might lead to loss of the prosthesis [130].

Tribocorrosion

Tribocorrosion is wear or degradation of an implant due to a combination of mechanical-, chemical-, and electrolytic factors [131–133]. Ions and degradation particles released from the implant can induce inflammation and bone loss around a dental- or medical implant [130, 131, 134–136]. A decrease in the corrosion resistance of Ti surfaces is seen then Ti surfaces are colonized by *S. mutans* [137, 138]. Bacterial adhesion to dental implant surfaces is dependent on the surface structure; a machined surface shows less adhered bacteria compared to an implant with a surface modification e.g. acid-etched or sandblasted [139]. Biofilm formation on a surface will decrease the friction and work as a lubricant [140]. A lower friction between components should lower the wear, but a combination of a lower pH and an increase in the mechanical movement induced by a biofilm might lead to higher wear from the implant components [138, 140]. Implants with an external hexagon interface have been shown to harbor significant number of bacteria which could lead to a microleakage, while implants with an internal connection show less microleakage of bacterial products [141, 142]. Dental crowns, abutments, implant interface, and the implant are exposed to a complex environment with exposure to food particles, drinks, temperature- and pH variations, saliva, micromovement, abrasion from tooth brushing, body fluids, and the bone [143]. This combination of tribocorrosive factors will influence the degradation of the included materials [143]. In saliva, chemicals such as lactic acid, citric acid, hydrogen peroxide, and chlorides that are known to be corrosive to Ti can be found [144]. In addition, LPS has been shown to affect the corrosion behavior of Ti, with a promotion of corrosion at pH 4–7, while an inhibitory effect is seen at pH 2 [145]. Corrosion of the protective oxide layer as well as promotion of an increased bacterial attachment can be affected by *Porphyromonas gingivalis* [146]. Hydrogen sulfide (H_2S) , methanethiol (CH₃SH), and dimethyl sulfide ($[CH₃]₂S$) produced by periodontal pathogens are chemicals that can corrode Ti [147, 148]. Corrosion

products released from Ti implants by tribocorrosion will accumulate in the peri-implant mucosa resulting in an inflammatory immunological response.

Wear Particles from the Abutment-implant Interface and Implants

A good fit of the joint between the abutment and implant is essential to reduce the risk of misfit and degradation products released from the implant or the abutment [149, 150]. Zirconia-based abutments show a higher degree of wear particles from the Ti implant compared to abutments made of Ti [151]. There has been a debate surrounding whether supra- or subcrestal positioning of the implant-abutment interface could have an impact on peri-implant inflammation. A higher neutrophil accumulation at the area of the interface has been shown at implants with a subcrestal placement compared to a supracrestal [152].

Cytotoxicity of wear debris from dental implants depend on size, concentration, and composition of the particles released into the peri-implant tissue [153]. In a recent review, it is shown that debris from dental implants and abutments has an adverse effect on the soft- and hard tissue [154].

Wear Particles from Dental Implants and the Impact on Peri-implantitis Disease

In later years, the effect of particle-induced inflammation around dental implants has been in more focus in dentistry. In comparison, this has been known to be the major cause for losing orthopedic prostheses for many years. While peri-implantitis has been seen as an infectious disease caused by microorganisms such as periodontitis; a major difference between an orthopedic- and dental implant is the environment they are placed in. Orthopedic implants are installed in a sterile environment without exposure to different chemicals and microorganisms. They are larger than dental implants and exposed to more abrasion, so investigation of the effect from released wear particles has been thoroughly investigated. Dental implants are on the other hand placed in a very tough environment exposed to the oral cavity through the peri-implant mucosa. The connection to the oral cavity will continuously expose the implant and abutment to microorganisms, chemicals, and changes in temperature and pH. In these tribocorrosive environment, release of metal particles from the abutment/implants are inevitable. It has been shown that tissue around an implant with peri-implantitis has a higher content of Ti than healthy tissue [155]. It has been suggested that particles released from dental implants might aggravate the inflammation and cause faster degradation of the supporting tissue and bone around a dental implant with peri-implantitis disease [77, 156, 157]. It has been shown that peri-implant tissues affected with peri-implantitis have a high content of Ti [155]. An immune cell seen in a biopsy from peri-implantitis tissue is demonstrated in Figure 5.3.

Peri-implantitis is a disease with multiple contributing factors where particles from the implant and abutment might be an etiological factor. A patient suffering from severe peri-implantitis at one implant is shown in Figures 5.4 and 5.5. In this case, no visible particles could be

Figure 5.3 Transmission electron microscopy (TEM) images of a section from a biopsy of the soft tissue around a dental implant with peri-implantitis. A cell with a large nucleus, correlated to the morphology of a macrophage can be seen in the image.

Figure 5.4 Clinical photo of a patient with a severe periimplantitis after removal of the implant–supported supra construction. The implant in the center (#32) to the right exhibits an extensive inflammation in the peri-implant tissue, with redness, swelling, and suppuration. Calculus can be identified at the implant shoulder in all the implants.

Figure 5.5 Radiographs showing at the implant in the center an extensive loss of supporting bone around. Neighboring implant to the right also shows signs of marginal bone loss, while a stable marginal bone level is seen at the implant to the left. Formation of calculus is seen at the top of both implants with bone loss.

found in the peri-implant tissue during surgery (Figures 5.6 and 5.7). However, visible damages to the implant surface and threads could be identified in photos and with scanning electron microscopy (SEM) (Figure 5.8). Surface modification of dental implants is used to enlarge the surface leading to better primary stability and get a better wettability to achieve a faster initiation of bone formation at the implant surface. An anodized implant surface is shown in Figure 5.9a. If marginal bone loss occurs around an implant, regardless of the cause is peri-implantitis or some other factors, microorganisms will grow at the implant surface. An implant with a surface modification will be much harder if not impossible to be completely clean from bacteria. A dental implant with an anodized surface, removed due to periimplantitis, is seen in Figure 5.9b, c. This case demonstrates that the probable cause for losing the implants is caused by a microbial infection.

Degradation products from an implant are released during placement, by corrosion, tribocorrosion, and abrasion during mechanical cleaning of the implant and abutments [131, 158]. Release of these particles will accumulate in the periimplant mucosa and act as foreign bodies, triggering the immune system to release inflammatory mediators associated with bone resorption [119]. These particles are also known to show adverse effects on the peri-implant soft- and hard tissue [159]. Scaling is the standard method for

(a) (b) **Figure 5.6** Clinical photo during surgery to remove the central implant due to severe peri-implantitis. (a) Extensive bone loss is seen at the central implant. No visible signs of damages to the implant threads or the surface can be seen. (b) No metal particles were identified in the surrounding tissue after removal of the center implant.

Figure 5.7 Clinical photo during surgery to remove an Brånemark (Bmk) System mark (Mk) III TiUnite (Nobel Biocare, Zürich, Switzerland) implant showing severe bone loss due to peri-implantitis. Small signs of damages to the implant surface can be identified. No visible metal particles were found in the peri-implant tissue.

treatment of periodontitis, when it is used on dental implants Ti particles will be released which can induce inflammation and contribute to osteolysis [160]. This mechanical debridement of dental implants should be used with care and other gentler techniques should be developed. An interesting technique for biofilm removal prior to regenerative surgery has been shown using electrolytic cleaning [161]. This might be a promising method for the treatment of peri-implantitis, but the effect on long term is unknown.

Platform switching is a design with different diameter of the implant compared to the abutment neck, which will let the soft tissue attach more cervically and closer to the axis of the implant, further away from the bone crest, compared to a platform-matched implant [162]. A platform-matched and platform-switched implant are illustrated in Figure 5.10. The theory behind platform switching of a dental implant is to increase the distance between the implant shoulder and the bone, which might reduce the risk for initial bone loss and establishment of a biofilm on the implant surface. This might also reduce the risk of corrosion of Ti from the implant surface [163]. Studies have also shown that marginal bone loss is reduced with platformed switched implants compared to implant matched, with an increase of the differences over time [164]. Platform switching is an interesting concept that should be followed in long time studies to gather more show explicit evidence that marginal bone loss can be reduced.

The only present clinical study comparing implants with platform-switched implants with butt-joint and Morsetapered connections in the same patient was initiated at the Eastman Dental Center at the University of Rochester, NY in 28 patients with edentulous mandibles. The study showed, after implant placement at the bone level and immediate loading using prefabricated abutments placed at the day of surgery (without removal), a significant difference between the two implant designs. Specifically, implants with butt-joint connections (70%) lose more bone compared to the implants with Morse tapered connections (11%) [165].

The next case presented shows a different pattern compared to the previous case. Images show two implants with a severe loss of supporting bone Figure 5.11.

Figure 5.8 (a) Photo of a Bmk System MkIII TiUnite implant showing signs of damages to the threads. (b) and (c) SEM images show clear damages to the implant threads. If those damages are caused during the insertion of the implant is unknown. Magnification at (b) 30 X and (c) 150 X.

Figure 5.9 SEM images of (a) The surface of an unused Bmk System MkIII TiUnite implant. Pits and grooves are seen on implant surfaces modified with anodization to establish a surface enlargement and an improved primary stability during installation (b) Surface of a Bmk System MkIII TiUnite after removal due to peri-implantitis. The surface is covered with biological material and bacteria are seen all over the surface. (c) A bacterium at the opening of a pit at the surface-modified implant. Bacteria growing inside the pits of a surface-modified implant makes a complete removal during peri-implantitis treatment almost impossible to achieve. (a) and (b) magnification at 5 KX and (c) at 100 KX.

Figure 5.10 Schematic illustration of platform matched (left) and platform switched (right) abutment connection. Illustration from On titanium release from dental implants and the inflammatory response. *Source:* Pettersson [156]/University of Umea.

Figure 5.11 Radiograph showing extensive bone loss around dental implants supporting a full arch implant–supported denture. The two most distal implants show close proximity to each other. No visible calculus can be identified in the radiograph.

During surgery, clear signs of damages to the implants as well as metal particles in the surrounding tissue Figure 5.12. Biopsies from the peri-implant mucosa show metal content that could be identified in light microscopy, Figure 5.13. Electron microscopy also could identify metal particles Figure 5.14, complementary element analysis shows that metal particles could be identified as Ti. Compared to the previous case presented particles released into the periimplant tissue might have aggravated the peri-implantitis. Evidence that metal particles from debris from dental implants and abutments in some cases have an adverse effect and might have an impact on the pathogenesis of peri-implantitis [136, 154].

Similarly, the granulation tissue removed during surgical treatment of peri-implantitis showed a high concentration of titanium ions using an X-ray spectroscopy (Figures 5.15–5.17).

In summary, metal particles due to wear at the time of implant placement, tribocorrosion phenomena under loading conditions, and micromovements at the implant– abutment connections provide strong evidence that Ti nanoparticles are present in the peri-implant tissues and might influence the immunological host response leading alone or in conjunction with bacteria from the biofilm to marginal bone loss and severe peri-implant bony defects. In addition, cleaning the implant surfaces, promoting potential damages to the implant surface pattern has an impact on the continuous resorption of the peri-implant bone and should be revised especially when gentle procedures are not warranted. Further discussion of the role of the Ti particles and the potential systemic effects should be extensively studied to find the ideal biomaterial for implant therapy, improving clinical outcomes without risks of periimplant infections.

Figure 5.12 Clinical photos during peri-implant surgery. (a) Clear signs of damage to the implant threads are seen in the photo, white arrows. No calculus visible at the implant nor the abutment surfaces. (b) Metallic particles are visible at the soft tissue and bone, white arrows. *Source:* With permission from Dr. Carola Höglund-Åberg.

Figure 5.13 Light microscope image of granulation tissue removed during peri-implantitis surgery. Metallic particles can be identified inside the microscopic section (Original Magnification: 400 X). Image first published in Titanium release in periimplantitis. *Source:* Pettersson et al. [155]/with permission from John Wiley & Sons, Inc.

 (a) (b) 0 µm $10 \mu m$

Figure 5.14 (a) SEM image of a biopsy from the peri-implant tissue of a dental implant with peri-implantitis. The high contrasted particles in the images are metallic particles embedded inside the tissue. (b) SEM-Energy Dispersive X-Ray Spectroscopy (EDS) map showing high contrasting metal particles identified as Ti particles. Magnification at 1.5 KX.

Figure 5.15 Peri-implant inflammatory reaction presenting deep probing pocket depth and crestal bone loss.

Figure 5.16 Peri-implant bony defect after removal of granulation tissue before grafting.

Meas. cond.											
Diaphragm DE-10 mm			Atmosphere Helium		Sample spin None			Meas. order optimization Yes			
Condition name High-Z Mid-Z Low-Z Analyzed result (FP method)		Primary filter F C Open	Tube voltage (kV) 60.0 35.0 6.5		Tube current (μA) 200 342 277		ST (μ sec) 1.6 1.6 1.6	100 100 100	Meas. time (sec)	Dead time (%) 1.5 17.5 37.3	
No. 1 2 3 4 5 6 7 Spectrum	Component Al Ti Zn Ag Au Pt Pd		Unit Result 83.7 mass% 130000 ppm 3.31 mass% ND mass% ND mass% ND mass% ND mass%		Stat. Err. 3.67 8960 0.0699	18800 0.0925	LLD 2.15	LLQ 6.46 56500 0.278	Element line $L:AI-K\alpha$ M : Τί-Κα $M:Zn-K\alpha$	Intensity (cps) 119.253 8.910 46.112	
cps											
1.0 0.8 0.6 Intensity 0.4 0.2 0.0	Al-Ka 1.5 1.0	\times 10000 2.0 2.5	Ti-Ka 4	6	$\times 20$ $2n-K\alpha$ 8 10		12	14	20 30	\times 5.0 40	

Figure 5.17 Peri-implant soft tissue spectrometric analysis showing high presence of Titanium nanoparticles (130,000ppm).

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References

- **1** Branemark, P.I., Adell, R., Breine, U. et al. (1969). Intra-osseous anchorage of dental prostheses. I. Experimental studies. *Scandinavian Journal of Plastic and Reconstructive Surgery* 3 (2): 81–100.
- **2** Adell, R., Hansson, B.O., Branemark, P.I., and Breine, U. (1970). Intra-osseous anchorage of dental prostheses. II. Review of clinical approaches. *Scandinavian Journal of Plastic and Reconstructive Surgery* 4 (1): 19–34.
- **3** Branemark, P.I., Hansson, B.O., Adell, R. et al. (1977). Osseointegrated implants in the treatment of the edentulous jaw. Experience from a 10-year period. *Scandinavian Journal of Plastic and Reconstructive Surgery* 16: 1–132.
- **4** Adell, R., Lekholm, U., Rockler, B., and Branemark, P.I. (1981). A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. *International Journal of Oral Surgery* 10 (6): 387–416.
- **5** Schroeder, A., Pohler, O., and Sutter, F. (1976). Tissue reaction to an implant of a titanium hollow cylinder with a titanium surface spray layer. *SSO Schweiz Monatsschr Zahnheilkd* 86 (7): 713–727.
- **6** Schroeder, A., Stich, H., Straumann, F., and Sutter, F. (1978). The accumulation of osteocementum around a dental implant under physical loading. *SSO Schweiz Monatsschr Zahnheilkd* 88 (10): 1051–1058.
- **7** Schroeder, A., van der Zypen, E., Stich, H., and Sutter, F. (1981). The reactions of bone, connective tissue, and epithelium to endosteal implants with titanium-sprayed surfaces. *Journal of Maxillofacial Surgery* 9 (1): 15–25.
- **8** Albrektsson, T., Zarb, G., Worthington, P., and Eriksson, A.R. (1986). The long-term efficacy of currently used dental implants: a review and proposed criteria of success. *The International Journal of Oral & Maxillofacial Implants* 1 (1): 11–25.
- **9** Brånemark, P.-I. and Chien, S. (2005). *The Osseointegration Book: From Calvarium to Calcaneus*. Quintessence Publishing Company.
- **10** Schnitman, P.A. and Shulman, L.B. (1979). Recommendations of the consensus development conference on dental implants. *Journal of the American Dental Association (1939)* 98 (3): 373–377.
- **11** Bieler, T.R., Trevino, R.M., and Zeng, L. (2005). Alloys: titanium. In: *Encyclopedia of Condensed Matter Physics* (ed. F. Bassani, G.L. Liedl, and P. Wyder), 65–76. Oxford: Elsevier.
- **12** Duraccio, D., Mussano, F., and Faga, M.G. (2015). Biomaterials for dental implants: current and future trends. *Journal of Materials Science* 50 (14): 4779–4812.
- **13** Niinomi, M. (2016). Recent research and development in titanium alloys for biomedical applications and healthcare goods. *Science and Technology of Advanced Materials* 4 (5): 445–454.
- **14** Romeo, E., Lops, D., Amorfini, L. et al. (2006). Clinical and radiographic evaluation of small-diameter (3.3-mm) implants followed for 1-7 years: a longitudinal study. *Clinical Oral Implants Research* 17 (2): 139–148.
- **15** Osman, R.B. and Swain, M.V. (2015). A critical review of dental implant materials with an emphasis on Titanium versus Zirconia. *Materials (Basel)* 8 (3): 932–958.
- **16** Zarb G. (1983). Proceedings of the Toronto Conference on Osseointegration in Clinical Dentistry, May 1982. St. Louis: C.V. Mosby Company.
- **17** The American College of Prosthodontists. Prosthodontists TACo. Attendee to the Toronto conference of 1982. [https://www.prosthodontics.org/](https://www.prosthodontics.org/assets/1/7/Toronto_conference_attendees[2]1.pdf) [assets/1/7/Toronto_conference_attendees\[2\]1.pdf.](https://www.prosthodontics.org/assets/1/7/Toronto_conference_attendees[2]1.pdf)
- **18** Chrcanovic, B. (2017). *On Failure of Oral Implants*. Malmö: Department of Prosthodontics, Faculty of Odontology, Malmö University.
- **19** Petersen, P.E. and Ogawa, H. (2012). The global burden of periodontal disease: towards integration with chronic disease prevention and control. *Periodontology 2000* 60 (1): 15–39.
- **20** Kassebaum, N.J., Bernabe, E., Dahiya, M. et al. (2015). Global burden of untreated caries: a systematic review and metaregression. *Journal of Dental Research* 94 (5): 650–658.
- **21** Eke, P.I., Dye, B.A., Wei, L. et al. (2015). Update on prevalence of periodontitis in adults in the United States: NHANES 2009 to 2012. *Journal of Periodontology* 86 (5): 611–622.
- **22** Allen, P.F., McMillan, A.S., and Walshaw, D. (2001). A patient-based assessment of implant-stabilized and conventional complete dentures. *The Journal of Prosthetic Dentistry* 85 (2): 141–147.
- **23** Heydecke, G., Locker, D., Awad, M.A. et al. (2003). Oral and general health-related quality of life with conventional and implant dentures. *Community Dentistry and Oral Epidemiology* 31 (3): 161–168.
- **24** Strassburger, C., Heydecke, G., and Kerschbaum, T. (2004). Influence of prosthetic and implant therapy on satisfaction and quality of life: a systematic literature review. Part 1 – characteristics of the studies. *The International Journal of Prosthodontics* 17 (1): 83–93.
- **25** Turkyilmaz, I., Company, A.M., and McGlumphy, E.A. (2010). Should edentulous patients be constrained to removable complete dentures? The use of dental implants to improve the quality of life for edentulous patients. *Gerodontology* 27 (1): 3–10.
- **26** Schwarz, F., Derks, J., Monje, A., and Wang, H.L. (2018). Peri-implantitis. *Journal of Clinical Periodontology* 45 (Suppl 20): S246–S266.
- **27** Levignac, J. (1965). Periimplantation osteolysisperiimplantosis – periimplantitis. *Revue Française d'Odonto-Stomatologie* 12 (8): 1251–1260.
- **28** Norowski, P.A. Jr. and Bumgardner, J.D. (2009). Biomaterial and antibiotic strategies for peri-implantitis: a review. *Journal of Biomedical Materials Research. Part B, Applied Biomaterials* 88 (2): 530–543.
- **29** Ivanovski, S. and Lee, R. (2018). Comparison of periimplant and periodontal marginal soft tissues in health and disease. *Periodontology 2000* 76 (1): 116–130.
- **30** Lee, C.T., Huang, Y.W., Zhu, L., and Weltman, R. (2017). Prevalences of peri-implantitis and peri-implant mucositis: systematic review and meta-analysis. *Journal of Dentistry* 62: 1–12.
- **31** Tarnow, D.P. (2016). Increasing prevalence of periimplantitis: how will we manage? *Journal of Dental Research* 95 (1): 7–8.
- **32** Albrektsson, T., Dahlin, C., Jemt, T. et al. (2014). Is marginal bone loss around oral implants the result of a provoked foreign body reaction? *Clinical Implant Dentistry and Related Research* 16 (2): 155–165.
- **33** Chrcanovic, B.R., Kisch, J., Albrektsson, T., and Wennerberg, A. (2016). Factors influencing early dental implant failures. *Journal of Dental Research* 95 (9): 995–1002.
- **34** Moraschini, V., Poubel, L.A., Ferreira, V.F., and dos Sp Barboza, E. (2015). Evaluation of survival and success rates of dental implants reported in longitudinal studies with a follow-up period of at least 10 years: a systematic review. *International Journal of Oral and Maxillofacial Surgery* 44 (3): 377–388.
- **35** Ledermann, D. (1983). 6-year clinical trial with titanium plasma-coated ITI (Internationales Team fur Implantologie) screw implants in the mandibular interforaminal region. *SSO Schweiz Monatsschr Zahnheilkd* 93 (11): 1070–1089.
- **36** ten Bruggenkate, C.M., van der Kwast, W.A., and Oosterbeek, H.S. (1990). Success criteria in oral implantology. A review of the literature. *The International Journal of Oral Implantology* 7 (1): 45–51.
- **37** Lütjering, G. and Williams, J.C. (2003). *Titanium*. Berlin; New York: Springer.
- **38** von Crell, L.F.F. (1791). *Chemische Annalen für die Freunde der Naturlehre, Arzneygelahrtheit, Haushaltungskunst und Manufacturen*. C. G. Fleckeisen.
- **39** Hunter, M.A. (1910). Metallic titanium. *Journal of the American Chemical Society* 32 (3): 330–336.
- **40** Branemark, P.-I., Zarb, G.A., and Albrektsson, T. (1985). *Tissue-Integrated Prostheses: Osseointegration in Clinical Dentistry*. Chicago: Quintessence Publ. Co. Inc.
- **41** Kasemo, B. and Lausmaa, J. (1985). Metal selection and surface characteristics. In: *Tissue-Integrated Prostheses: Osseointegration in Clinical Dentistry* (ed. P.-I. Brånemark, G. Zarb, and T. Albrektsson), 99–116. Chicago: Quintessence Publ. Co. Inc.
- **42** Anusavice, K.J. and Phillips, R.W. (2003). *Phillips' Science of Dental Materials*, 11e. St. Louis, Mo: Saunders.
- **43** Donachie, M.J. (2000). *Titanium: A Technical Guide*, 2e. ASM International.
- **44** Male, D., Stokes Peebles, R. Jr., and Male, V. (2020). *Immunology E-Book*. Elsevier.
- **45** Abbas, A.K., Lichtman, A.H., Pillai, S., and Baker, D.H. (2019). *Basic Immunology: Functions and Disorders of the Immune System*, 6e. Philadelphia, PA: Elsevier.
- **46** Larsen, G.L. and Henson, P.M. (1983). Mediators of inflammation. *Annual Review of Immunology* 1: 335–359.
- **47** Medzhitov, R. (2008). Origin and physiological roles of inflammation. *Nature* 454 (7203): 428–435.
- **48** Millan, M.J. (1999). The induction of pain: an integrative review. *Progress in Neurobiology* 57 (1): 1–164.
- **49** Abbas, A.K., Lichtman, A.H., and Pillai, S. (2016). *Basic Immunology: Functions and Disorders of the Immune System*, 5e. Elsevier Inc.
- **50** Chong, S.Z., Evrard, M., Goh, C.C., and Ng, L.G. (2018). Illuminating the covert mission of mononuclear phagocytes in their regional niches. *Current Opinion in Immunology* 50: 94–101.
- **51** Dinarello, C.A. (2011). A clinical perspective of IL-1beta as the gatekeeper of inflammation. *European Journal of Immunology* 41 (5): 1203–1217.
- **52** Mosmann, T.R., Cherwinski, H., Bond, M.W. et al. (1986). Two types of murine helper T cell clone. I. Definition according to profiles of lymphokine activities and secreted proteins. *Journal of Immunology* 136 (7): 2348–2357.
- **53** Anderson, C.F. and Mosser, D.M. (2002). A novel phenotype for an activated macrophage: the type 2 activated macrophage. *Journal of Leukocyte Biology* 72 (1): 101–106.
- **54** Martinez, F.O. and Gordon, S. (2014). The M1 and M2 paradigm of macrophage activation: time for reassessment. *F1000Prime Reports* 6: 13.
- **55** Chistiakov, D.A., Myasoedova, V.A., Revin, V.V. et al. (2018). The impact of interferon-regulatory factors to macrophage differentiation and polarization into M1 and M2. *Immunobiology* 223 (1): 101–111.
- **56** Metchnikoff, E. Phagocytosis (1883). Untersuchung uber die mesodermalen Phagocyten einiger Wirbel tiere. *Biologicial Centralblatt* 18: 560.
- **57** Aderem, A. and Underhill, D.M. (1999). Mechanisms of phagocytosis in macrophages. *Annual Review of Immunology* 17: 593–623.
- **58** Rosales, C. and Uribe-Querol, E. (2017). Phagocytosis: a fundamental process in immunity. *BioMed Research International* 2017: 9042851.
- **59** Guo, H., Callaway, J.B., and Ting, J.P. (2015). Inflammasomes: mechanism of action, role in disease, and therapeutics. *Nature Medicine* 21 (7): 677–687.
- **60** Zou, H., Li, Y., Liu, X., and Wang, X. (1999). An APAF-1·cytochromecmultimeric complex is a functional apoptosome that activates procaspase-9. *The Journal of Biological Chemistry* 274 (17): 11549–11556.
- **61** Fink, S.L. and Cookson, B.T. (2005). Apoptosis, pyroptosis, and necrosis: mechanistic description of dead and dying eukaryotic cells. *Infection and Immunity* 73 (4): 1907–1916.
- **62** Lamkanfi, M. and Dixit, V.M. (2014). Mechanisms and functions of inflammasomes. *Cell* 157 (5): 1013–1022.
- **63** Schroder, K. and Tschopp, J. (2010). The inflammasomes. *Cell* 140 (6): 821–832.
- **64** Davis, B.K., Wen, H., and Ting, J.P. (2011). The inflammasome NLRs in immunity, inflammation, and associated diseases. *Annual Review of Immunology* 29: 707–735.
- **65** Latz, E., Xiao, T.S., and Stutz, A. (2013). Activation and regulation of the inflammasomes. *Nature Reviews Immunology* 13 (6): 397–411.
- **66** Cohen, G.M. (1997). Caspases: the executioners of apoptosis. *Biochemical Journal* 326 (Pt 1): 1–16.
- **67** Martinon, F., Burns, K., and Tschopp, J. (2002). The inflammasome: a molecular platform triggering activation of inflammatory caspases and processing of proIL-β. *Molecular Cell* 10 (2): 417–426.
- **68** Dinarello, C.A. (2018). Overview of the IL-1 family in innate inflammation and acquired immunity. *Immunological Reviews* 281 (1): 8–27.
- **69** Hornung, V., Bauernfeind, F., Halle, A. et al. (2008). Silica crystals and aluminum salts activate the NALP3 inflammasome through phagosomal destabilization. *Nature Immunology* 9 (8): 847–856.
- **70** Riteau, N., Baron, L., Villeret, B. et al. (2012). ATP release and purinergic signaling: a common pathway for particlemediated inflammasome activation. *Cell Death & Disease* 3: e403.
- **71** Duewell, P., Kono, H., Rayner, K.J. et al. (2010). NLRP3 inflammasomes are required for atherogenesis and activated by cholesterol crystals. *Nature* 464 (7293): 1357–1361.
- **72** Chevriaux, A., Pilot, T., Derangere, V. et al. (2020). Cathepsin B is required for NLRP3 inflammasome activation in macrophages, through NLRP3 interaction. *Frontiers in Cell and Development Biology* 8: 167.
- **73** Dinarello, C.A. (1984). Interleukin-1. *Reviews of Infectious Diseases* 6 (1): 51–95.
- **74** Dinarello, C.A. (2009). Immunological and inflammatory functions of the interleukin-1 family. *Annual Review of Immunology* 27: 519–550.
- **75** Garlanda, C., Dinarello, C.A., and Mantovani, A. (2013). The interleukin-1 family: back to the future. *Immunity* 39 (6): 1003–1018.
- **76** Bauernfeind, F.G., Horvath, G., Stutz, A. et al. (2009). Cutting edge: NF-kappaB activating pattern recognition and cytokine receptors license NLRP3 inflammasome activation by regulating NLRP3 expression. *Journal of Immunology* 183 (2): 787–791.
- **77** Pettersson, M., Kelk, P., Belibasakis, G.N. et al. (2017). Titanium ions form particles that activate and execute

interleukin-1beta release from lipopolysaccharide-primed macrophages. *Journal of Periodontal Research* 52 (1): 21–32.

- **78** Dostert, C., Petrilli, V., Van Bruggen, R. et al. (2008). Innate immune activation through Nalp3 inflammasome sensing of asbestos and silica. *Science* 320 (5876): 674–677.
- **79** Moon, C., Park, H.J., Choi, Y.H. et al. (2010). Pulmonary inflammation after intraperitoneal administration of ultrafine titanium dioxide (TiO2) at rest or in lungs primed with lipopolysaccharide. *Journal of Toxicology and Environmental Health Part A* 73 (5): 396–409.
- **80** He, W.T., Wan, H., Hu, L. et al. (2015). Gasdermin D is an executor of pyroptosis and required for interleukin-1beta secretion. *Cell Research* 25 (12): 1285–1298.
- **81** Liu, X., Zhang, Z., Ruan, J. et al. (2016). Inflammasomeactivated gasdermin D causes pyroptosis by forming membrane pores. *Nature* 535 (7610): 153–158.
- **82** Kuriakose, T. and Kanneganti, T.D. (2018). Gasdermin D flashes an exit signal for IL-1. *Immunity* 48 (1): 1–3.
- **83** Evavold, C.L., Ruan, J., Tan, Y. et al. (2018). The poreforming protein gasdermin D regulates interleukin-1 secretion from living macrophages. *Immunity* 48 (1): 35–44.e36.
- **84** de Vasconcelos, N.M., Van Opdenbosch, N., Van Gorp, H. et al. (2019). Single-cell analysis of pyroptosis dynamics reveals conserved GSDMD-mediated subcellular events that precede plasma membrane rupture. *Cell Death and Differentiation* 26 (1): 146–161.
- **85** Mikos, A.G., McIntire, L.V., Anderson, J.M., and Babensee, J.E. (1998). Host response to tissue engineered devices. *Advanced Drug Delivery Reviews* 33 (1–2): 111–139.
- **86** Tang, L. and Eaton, J.W. (1999). Natural responses to unnatural materials: a molecular mechanism for foreign body reactions. *Molecular Medicine* 5 (6): 351–358.
- **87** Anderson, J.M., Rodriguez, A., and Chang, D.T. (2008). Foreign body reaction to biomaterials. *Seminars in Immunology* 20 (2): 86–100.
- **88** Anderson, J.M. (2000). Multinucleated giant cells. *Current Opinion in Hematology* 7 (1): 40–47.
- **89** Langhans, T. (1868). Über Riesenzellen mit wandständigen Kernen in Tuberkeln und die fibröse Form des Tuberkels. *Archiv für pathologische Anatomie und Physiologie und für klinische Medicin* 42: 382–404.
- **90** Yagi, M., Miyamoto, T., Sawatani, Y. et al. (2005). DC-STAMP is essential for cell-cell fusion in osteoclasts and foreign body giant cells. *The Journal of Experimental Medicine* 202 (3): 345–351.
- **91** Lindhe, J., Meyle, J., and Group DoEWoP (2008). Peri-implant diseases: consensus report of the sixth European workshop on periodontology. *Journal of Clinical Periodontology* 35 (8 Suppl): 282–285.
- **92** Zitzmann, N.U. and Berglundh, T. (2008). Definition and prevalence of peri-implant diseases. *Journal of Clinical Periodontology* 35 (8 Suppl): 286–291.

68 *Metallic Nanoparticles as a Risk Factor for Peri-implant Diseases*

- **93** Heitz-Mayfield, L.J. (2008). Peri-implant diseases: diagnosis and risk indicators. *Journal of Clinical Periodontology* 35 (8 Suppl): 292–304.
- **94** Tallarico, M., Canullo, L., Wang, H.L. et al. (2018). Classification systems for peri-implantitis: a narrative review with a proposal of a new evidence-based etiology codification. *The International Journal of Oral & Maxillofacial Implants* 33 (4): 871–879.
- **95** (2013). Peri-implant mucositis and peri-implantitis: a current understanding of their diagnoses and clinical implications. *Journal of Periodontology* 84 (4): 436–443.
- **96** Council NSaT (2007). *The National Nanotechnology Initiative – Strategic Plan*, 1–52. WW.NANO.GOV2007.
- **97** Pidgeon, N., Porritt, J., Ryan, J. et al. (2004). Nanoscience and nanotechnologies: opportunities and uncertainties. *The Royal Society & The Royal Academy of Engineering* 29: 2004.
- **98** Hou, J., Wang, L., Wang, C. et al. (2019). Toxicity and mechanisms of action of titanium dioxide nanoparticles in living organisms. *Journal of Environmental Sciences (China)* 75: 40–53.
- **99** Oberdorster, G., Oberdorster, E., and Oberdorster, J. (2005). Nanotoxicology: an emerging discipline evolving from studies of ultrafine particles. *Environmental Health Perspectives* 113 (7): 823–839.
- **100** Baranowska-Wojcik, E., Szwajgier, D., Oleszczuk, P., and Winiarska-Mieczan, A. (2020). Effects of titanium dioxide nanoparticles exposure on human health-a review. *Biological Trace Element Research* 193 (1): 118–129.
- **101** Warheit, D.B., Sayes, C.M., Reed, K.L., and Swain, K.A. (2008). Health effects related to nanoparticle exposures: environmental, health and safety considerations for assessing hazards and risks. *Pharmacology & Therapeutics* 120 (1): 35–42.
- **102** Rothen-Rutishauser, B.M., Schurch, S., Haenni, B. et al. (2006). Interaction of fine particles and nanoparticles with red blood cells visualized with advanced microscopic techniques. *Environmental Science & Technology* 40 (14): 4353–4359.
- **103** Cedervall, T., Lynch, I., Lindman, S. et al. (2007). Understanding the nanoparticle-protein corona using methods to quantify exchange rates and affinities of proteins for nanoparticles. *Proceedings of the National Academy of Sciences of the United States of America* 104 (7): 2050–2055.
- **104** Gheshlaghi, Z.N., Riazi, G.H., Ahmadian, S. et al. (2008). Toxicity and interaction of titanium dioxide nanoparticles with microtubule protein. *Acta Biochimica et Biophysica Sinica Shanghai* 40 (9): 777–782.
- **105** Shabbir, S., Kulyar, M.F., Bhutta, Z.A. et al. (2021). Toxicological consequences of titanium dioxide

nanoparticles (TiO2NPs) and their jeopardy to human population. *Bionanoscience* 11: 1–12.

- **106** Chen, Z., Wang, Y., Ba, T. et al. (2014). Genotoxic evaluation of titanium dioxide nanoparticles in vivo and in vitro. *Toxicology Letters* 226 (3): 314–319.
- **107** Petkovic, J., Kuzma, T., Rade, K. et al. (2011). Preirradiation of anatase TiO2 particles with UV enhances their cytotoxic and genotoxic potential in human hepatoma HepG2 cells. *Journal of Hazardous Materials* 196: 145–152.
- **108** Hattori, K., Nakadate, K., Morii, A. et al. (2017). Exposure to nano-size titanium dioxide causes oxidative damages in human mesothelial cells: The crystal form rather than size of particle contributes to cytotoxicity. *Biochemical and Biophysical Research Communications* 492 (2): 218–223.
- **109** Gratton, S.E., Ropp, P.A., Pohlhaus, P.D. et al. (2008). The effect of particle design on cellular internalization pathways. *Proceedings of the National Academy of Sciences of the United States of America* 105 (33): 11613–11618.
- **110** Albanese, A., Tang, P.S., and Chan, W.C. (2012). The effect of nanoparticle size, shape, and surface chemistry on biological systems. *Annual Review of Biomedical Engineering* 14: 1–16.
- **111** Chen, H.W., Su, S.F., Chien, C.T. et al. (2006). Titanium dioxide nanoparticles induce emphysema-like lung injury in mice. *The FASEB Journal* 20 (13): 2393–2395.
- **112** Pajarinen, J., Mackiewicz, Z., Pollanen, R. et al. (2010). Titanium particles modulate expression of Toll-like receptor proteins. *Journal of Biomedical Materials Research Part A* 92 (4): 1528–1537.
- **113** Chiu, R., Ma, T., Smith, R.L., and Goodman, S.B. (2006). Polymethylmethacrylate particles inhibit osteoblastic differentiation of bone marrow osteoprogenitor cells. *Journal of Biomedical Materials Research Part A* 77 (4): 850–856.
- **114** Chiu, R., Ma, T., Smith, R.L., and Goodman, S.B. (2009). Ultrahigh molecular weight polyethylene wear debris inhibits osteoprogenitor proliferation and differentiation in vitro. *Journal of Biomedical Materials Research Part A* 89 (1): 242–247.
- **115** Wang, C.T., Lin, Y.T., Chiang, B.L. et al. (2010). Overexpression of receptor activator of nuclear factorkappaB ligand (RANKL), inflammatory cytokines, and chemokines in periprosthetic osteolysis of loosened total hip arthroplasty. *Biomaterials* 31 (1): 77–82.
- **116** Holt, G., Murnaghan, C., Reilly, J., and Meek, R.M. (2007). The biology of aseptic osteolysis. *Clinical Orthopaedics and Related Research* 460: 240–252.
- **117** Harris, W.H., Schiller, A.L., Scholler, J.M. et al. (1976). Extensive localized bone resorption in the femur following total hip replacement. *The Journal of Bone and Joint Surgery. American Volume* 58 (5): 612–618.
- **118** Jacobs, J.J., Roebuck, K.A., Archibeck, M. et al. (2001). Osteolysis: basic science. *Clinical Orthopaedics and Related Research* 393: 71–77.
- **119** Purdue, P.E., Koulouvaris, P., Potter, H.G. et al. (2007). The cellular and molecular biology of periprosthetic osteolysis. *Clinical Orthopaedics and Related Research* 454: 251–261.
- **120** Hallab, N.J. and Jacobs, J.J. (2009). Biologic effects of implant debris. *Bulletin of the NYU Hospital for Joint Diseases* 67 (2): 182–188.
- **121** Kadoya, Y., Revell, P.A., al-Saffar, N. et al. (1996). Bone formation and bone resorption in failed total joint arthroplasties: histomorphometric analysis with histochemical and immunohistochemical technique. *Journal of Orthopaedic Research: Official Publication of the Orthopaedic Research Society* 14 (3): 473–482.
- **122** Urban, R.M., Jacobs, J.J., Tomlinson, M.J. et al. (2000). Dissemination of wear particles to the liver, spleen, and abdominal lymph nodes of patients with hip or knee replacement. *The Journal of Bone and Joint Surgery. American Volume* 82 (4): 457–476.
- **123** Ikeda, T., Takahashi, K., Kabata, T. et al. (2010). Polyneuropathy caused by cobalt-chromium metallosis after total hip replacement. *Muscle & Nerve* 42 (1): 140–143.
- **124** Machado, C., Appelbe, A., and Wood, R. (2012). Arthroprosthetic cobaltism and cardiomyopathy. *Heart, Lung & Circulation* 21 (11): 759–760.
- **125** Pelclova, D., Sklensky, M., Janicek, P., and Lach, K. (2012). Severe cobalt intoxication following hip replacement revision: clinical features and outcome. *Clinical Toxicology (Philadelphia, Pa.)* 50 (4): 262–265.
- **126** Czekaj, J., Ehlinger, M., Rahme, M., and Bonnomet, F. (2016). Metallosis and cobalt – chrome intoxication after hip resurfacing arthroplasty. *Journal of Orthopaedic Science* 21 (3): 389–394.
- **127** Biglia, A., Morandi, V., Monti, S. et al. (2020). Cobalt hip prosthesis intoxication mimicking an autoimmune disease. *Joint, Bone, Spine* 87 (6): 652–654.
- **128** Bradberry, S.M., Wilkinson, J.M., and Ferner, R.E. (2014). Systemic toxicity related to metal hip prostheses. *Clinical Toxicology (Philadelphia, Pa.)* 52 (8): 837–847.
- **129** Tower, S.S. (2012). Arthroprosthetic cobaltism associated with metal on metal hip implants. *BMJ* 344: e430.
- **130** Harris, W.H. (1995). The problem is osteolysis. *Clinical Orthopaedics and Related Research* 311: 46–53.
- **131** Cruz, H.V., Souza, J.C.M., Henriques, M., and Rocha, L.A. (2011). Tribocorrosion and bio-tribocorrosion in the oral environment: the case of dental implants. In: *Biomedical Tribology* (ed. J.P. Davim), 1–33. New York: Nova Science Publishers, Inc.
- **132** Mathew, M.T., Kerwell, S., Lundberg, H.J. et al. (2014). Tribocorrosion and oral and maxillofacial surgical devices. *The British Journal of Oral & Maxillofacial Surgery* 52 (5): 396–400.
- **133** Apaza-Bedoya, K., Tarce, M., Benfatti, C.A.M. et al. (2017). Synergistic interactions between corrosion and wear at titanium-based dental implant connections: a scoping review. *Journal of Periodontal Research* 52 (6): 946–954.
- **134** Gallo, J., Goodman, S.B., Konttinen, Y.T. et al. (2013). Osteolysis around total knee arthroplasty: a review of pathogenetic mechanisms. *Acta Biomaterialia* 9 (9): 8046–8058.
- **135** Dapunt, U., Giese, T., Lasitschka, F. et al. (2014). On the inflammatory response in metal-on-metal implants. *Journal of Translational Medicine* 12: 74.
- **136** Mombelli, A., Hashim, D., and Cionca, N. (2018). What is the impact of titanium particles and biocorrosion on implant survival and complications? A critical review. *Clinical Oral Implants Research* 29 (Suppl 18): 37–53.
- **137** Souza, J.C., Ponthiaux, P., Henriques, M. et al. (2013). Corrosion behaviour of titanium in the presence of Streptococcus mutans. *Journal of Dentistry* 41 (6): 528–534.
- **138** Souza, J.C., Henriques, M., Oliveira, R. et al. (2010). Do oral biofilms influence the wear and corrosion behavior of titanium? *Biofouling* 26 (4): 471–478.
- **139** Badihi Hauslich, L., Sela, M.N., Steinberg, D. et al. (2013). The adhesion of oral bacteria to modified titanium surfaces: role of plasma proteins and electrostatic forces. *Clinical Oral Implants Research* 24 (Suppl A100): 49–56.
- **140** Souza, J.C., Henriques, M., Oliveira, R. et al. (2010). Biofilms inducing ultra-low friction on titanium. *Journal of Dental Research* 89 (12): 1470–1475.
- **141** Mishra, S.K., Chowdhary, R., and Kumari, S. (2017). Microleakage at the different implant abutment interface: a systematic review. *Journal of Clinical and Diagnostic Research* 11 (6): ZE10–ZE15.
- **142** Quirynen, M. and van Steenberghe, D. (1993). Bacterial colonization of the internal part of two-stage implants. An in vivo study. *Clinical Oral Implants Research* 4 (3): 158–161.
- **143** Mathew, M.T., Srinivasa Pai, P., Pourzal, R. et al. (2009). Significance of tribocorrosion in biomedical applications: overview and current status. *Advances in Tribology* 2009: 1–12.
- **144** Mabilleau, G., Bourdon, S., Joly-Guillou, M.L. et al. (2006). Influence of fluoride, hydrogen peroxide and lactic acid on the corrosion resistance of commercially pure titanium. *Acta Biomaterialia* 2 (1): 121–129.
- **145** Yu, F., Addison, O., Baker, S.J., and Davenport, A.J. (2015). Lipopolysaccharide inhibits or accelerates biomedical titanium corrosion depending on

70 *Metallic Nanoparticles as a Risk Factor for Peri-implant Diseases*

environmental acidity. *International Journal of Oral Science* 7 (3): 179–186.

- **146** Barao, V.A., Yoon, C.J., Mathew, M.T. et al. (2014). Attachment of Porphyromonas gingivalis to corroded commercially pure titanium and titanium-aluminumvanadium alloy. *Journal of Periodontology* 85 (9): 1275–1282.
- **147** Basic, A. and Dahlen, G. (2015). Hydrogen sulfide production from subgingival plaque samples. *Anaerobe* 35 (Pt A): 21–27.
- **148** Harada, R., Takemoto, S., Kinoshita, H. et al. (2016). Influence of sulfide concentration on the corrosion behavior of titanium in a simulated oral environment. *Materials Science & Engineering. C, Materials for Biological Applications* 62: 268–273.
- **149** Schwarz, M.S. (2000). Mechanical complications of dental implants. *Clinical Oral Implants Research* 11 (Suppl 1): 156–158.
- **150** Khraisat, A., Abu-Hammad, O., Al-Kayed, A.M., and Dar-Odeh, N. (2004). Stability of the implant/abutment joint in a single-tooth external-hexagon implant system: clinical and mechanical review. *Clinical Implant Dentistry and Related Research* 6 (4): 222–229.
- **151** Stimmelmayr, M., Edelhoff, D., Guth, J.F. et al. (2012). Wear at the titanium-titanium and the titanium-zirconia implant-abutment interface: a comparative in vitro study. *Dental Materials* 28 (12): 1215–1220.
- **152** Broggini, N., McManus, L.M., Hermann, J.S. et al. (2006). Peri-implant inflammation defined by the implant-abutment interface. *Journal of Dental Research* 85 (5): 473–478.
- **153** Messous, R., Henriques, B., Bousbaa, H. et al. (2021). Cytotoxic effects of submicron- and nano-scale titanium debris released from dental implants: an integrative review. *Clinical Oral Investigations* 25 (4): 1627–1640.
- **154** Romanos, G.E., Fischer, G.A., and Delgado-Ruiz, R. (2021). Titanium wear of dental implants from placement, under loading and maintenance protocols. *International Journal of Molecular Sciences* 22 (3).
- **155** Pettersson, M., Pettersson, J., Johansson, A., and Molin Thoren, M. (2019). Titanium release in

peri-implantitis. *Journal of Oral Rehabilitation* 46 (2): 179–188.

- **156** Pettersson, M. (2018). *On Titanium Release from Dental Implants and the Inflammatory Response*. Umeå: Umeå universitet.
- **157** Fretwurst, T., Nelson, K., Tarnow, D.P. et al. (2018). Is metal particle release associated with peri-implant bone destruction? An emerging concept. *Journal of Dental Research* 97 (3): 259–265.
- **158** Franchi, M., Bacchelli, B., Martini, D. et al. (2004). Early detachment of titanium particles from various different surfaces of endosseous dental implants. *Biomaterials* 25 (12): 2239–2246.
- **159** Noronha Oliveira, M., Schunemann, W.V.H., Mathew, M.T. et al. (2018). Can degradation products released from dental implants affect peri-implant tissues? *Journal of Periodontal Research* 53 (1): 1–11.
- **160** Eger, M., Sterer, N., Liron, T. et al. (2017). Scaling of titanium implants entrains inflammation-induced osteolysis. *Scientific Reports* 7: 39612.
- **161** Schlee, M., Rathe, F., Brodbeck, U. et al. (2019). Treatment of peri-implantitis-electrolytic cleaning versus mechanical and electrolytic cleaning-a randomized controlled clinical trial-six-month results. *Journal of Clinical Medicine* 8 (11): 1909.
- **162** Lazzara, R.J. and Porter, S.S. (2006). Platform switching: a new concept in implant dentistry for controlling postrestorative crestal bone levels. *International Journal of Periodontics & Restorative Dentistry* 26 (1): 9–17.
- **163** Alrabeah, G.O., Knowles, J.C., and Petridis, H. (2016). The effect of platform switching on the levels of metal ion release from different implant-abutment couples. *International Journal of Oral Science* 8 (2): 117–125.
- **164** Chrcanovic, B.R., Albrektsson, T., and Wennerberg, A. (2015). Platform switch and dental implants: a meta-analysis. *Journal of Dentistry* 43 (6): 629–646.
- **165** Romanos, G.E., Malmstrom, H., Feng, C. et al. (2014). Immediately loaded platform-switched implants in the anterior mandible with fixed prostheses: a randomized, split-mouth, masked prospective trial. *Clinical Implant Dentistry and Related Research* 16 (6): 884–892.

Part II

Management from Planning

Impact of Maintenance on Narrow Diameter Implants

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Narrow dental implant (NDI) is defined as implant with a diameter <3.5mm [1] and is divided into three categories based on their diameters, such as category 1: <3.0mm ("mini-implants"); category 2: 3.0–3.25mm; and category 3: 3.30–3.50mm [2]. In the literature, the use of these implants is well documented in cases of the anterior edentulous sectors of the ridges, not subjected to excessive chewing loads [3–5]. In these regions, the replacement of dental elements with implant-supported prostheses can not only be based on the biological principles of osseointegration described by Branemark [6] and on the success criteria of Albrektsson [7], but must also guarantee an adequate aesthetic result [8], obtaining a correct proportion between the white color of the tooth and the pink of the gingiva. The esthetic outcome can be evaluated through various indices that take into consideration fundamental anatomical parameters, including the presence of the interproximal papillae and the positioning of the gingival zenith [9, 10]. Tarnow et al. clarified that the papilla between two natural teeth is guaranteed when the distance between the bone crest and the contact point is equal to or less than 5mm [11]. Following the tooth loss, the papilla retracts, and the possibility of its reappearance will depend exclusively on the maintenance of the interproximal bone peaks of the neighboring teeth and on the positioning of a provisional or definitive prosthetic device that will again determine the interdental contact point. Likewise, the correct location of the gingival zenith is guaranteed by the preservation and/or regeneration of the buccal cortex of the edentulous alveolus [12]. To guarantee the respect of these bone structures, an adequate surgical procedure, an appropriate implant diameter, and a correct implant position are necessary [13]. Since the establishment of the implant biological width, as described by Adell et al., determines an unavoidable marginal bone [14], it is essential to respect safety distances. The ideal implant position is 1.5–2mm from the buccal plate, 1.5mm from the adjacent teeth, and 2–3mm from their cemento-enamel junction (CEJ). If these parameters are not respected a vertical bone reabsorption will occur, so that the gingival zenith and the presence of the papillae will be compromised.

In general, it is considered that a minimum of 1mm of bone tissue must surround the entire implant surface [2]; however, in the anterior sector, this measure is not sufficient to guarantee aesthetic success. When replacing dental elements with aesthetic impact, the choice of the implant diameter must be calculated by subtracting 3mm (1.5mm per side) from the mesio-distal dimension of edentulousness (distance at the crestal level between the two neighboring roots) to ensure the survival of the bone peaks following the occurrence of the Adell et al. phenomenon. Where the chosen implant diameter does not guarantee the minimum thicknesses required in the vestibule-palatine direction, regenerative surgical techniques will be required to increase the available bone. Considering that the average width of the upper lateral incisors is between 6.1 in females and 6.6 in males, it is necessary to use reduced-diameter implants for their replacement [15]. This consideration is even more evident for the lower arch incisors that have an average mesio-distal size of 5mm [16–17]. **(**CASE 1**)**

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Case 1 (provided by C. Peron)

(Figures 1 and 2) Preoperative phase. The clinical and radiographic preclinical situation presents a chronic periodontitis with third-degree mobility of the two lower lateral incisors. (B and C) The dental implants are inserted slightly palatal immediately after the teeth extractions of 3.2 and 4.1. (Figure 3) An implant of reduced diameter allows to easily maintain the correct distances with the adjacent teeth and a minimum distance of 2mm from the buccal bone wall, not interfering with the healing of bone remodeling of this wall. (Figure 4) The gap between the implant and the buccal bone wall is managed with the insertion of biomaterial up to the free gingival margin. (Figures 5 and 6) Two definitive abutments were screwed onto the implants and were no longer removed; the temporary bridge was cemented on top, creating a protective barrier to the inserted biomaterial. (Figures 7 and 8) Final cemented restoration in zirconia. Clinical and radiographical examination with final cemented restoration in translucent zirconia.

In the anterior region, where esthetics are of fundamental importance, the use of standard-diameter implants is indicated only for the replacement of teeth with a mesio-distal diameter >6.5mm.

NDI, hence, are necessary for the replacement of teeth with a mesio-distal diameter <6.5mm, such as lower incisors and upper lateral incisors in females.

Fig 4

Fig 6

Fig 5

Case 2 (provided by C. Peron)

(Figures 1–3) Clinical and radiographic pre-clinical pictures show a corono-radicular fracture of the left upper lateral incisor (Figure 4) the fundamental prerequisite for minimally invasive surgery is to perform an atraumatic extraction without damaging the surrounding tissues, in particular, the buccal bone wall (Figures 5–7) Narrow implant placement in the fresh extraction socket allows to respect the correct distances with the adjacent teeth, also in this case in which the mesio-distal distance between the two adjacent teeth is only 6 mm. The gap with the buccal bone wall is filled with biomaterial in order to contract the reabsorption of this alveolar portion following the extraction of the dental element. (Figures 8 and 9) Thanks to all these steps and also allows, and thanks to the Socket Healing Guide of the screwed provisional, to create a correct peri-implant emergence profile.

(Figures 10 and 11) Final radiography and cemented restoration in translucent zirconia.

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Fig 2

Fig 6

Fig 10

In the presence of a reduced horizontal alveolar ridge (without bone grafting or orthodontic treatment), the use of NDI would be beneficial to decrease the rate of bone grafting associated with implant insertion. This may especially help elderly patients or patients with general medical compromised conditions, who would benefit from implant therapy with reduced surgical invasiveness, or patients who decline offered bone grafting or orthodontic treatment.

It has been verified that the implant–abutment connection can also affect the maintenance of peri-implant bone levels [18]. The platform switching, obtained with abutments that are undersized with respect to the implant platform [19], and a conometric type implantabutment connection allow respectively to remove the critical area of abutment–implant union and to reduce the micro-movements and micro-infiltrations that could create an insult on the crestal bone [20]. In particular, it has been shown that the association of these two characteristics of the implant abutment allows the preservation of the peri-implant bone and increases the prognosis of soft tissue stability (gingival zenith and interproximal papillae) [21]. A recent study by Vela et al. [22] suggested that the use of platform-switched implants allows for placement 1 mm from adjacent teeth, and these implant types still maintain the bone interproximal peaks after about one year of loading. Furthermore, reducing the implant–tooth distance may result in better aesthetic and functional results in situations with limited mesio-distal space.

Case 3 (provided by C. Peron)

(Figures 1–3) Edentulous area in the first premolar area of the maxilla with a mesio-distal distance of only 6mm, clinical and radiographic view. (Figures 4 and 5) For proper space management, the use of a narrow implant is recommended; in this way, we do not interfere with the buccal bone. Below are the positioning phases. (Figure 6) The implant allows to obtain an excellent primary stability, confirmed by the insertion torque and by the value of the resonance frequency higher than 35 Ncm and 70 ISQ respectively; this made it possible to immediately load the implant with an immediate

screwed provisional. (Figure 7) Soft tissue healing after four months upon removal of the provisional restoration. (Figures 8–10) Intraoral scan with and without scan body to better capture and transfer the emergence profile to the laboratory, from the oral scan a resin model is printed through a 3D printer. (Figure 11) Final cemented restorations of first premolar onto the implant and second premolar onto the natural abutment are in lithium di-silicate. (Figure 12) Radiographic view of the stable marginal bone with the follow-up at one to two and four years.

Fig 6

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However, Romanos and Javed [23] suggested that crestal bone loss around implants seems to be associated with several factors other than platform switching, such as the cervical features of the implant design, 3D-implant positioning, prosthetic concept, width of the alveolar ridge, and micromotions at the implant–abutment interface. A subcrestal placement was recommended [24]. In a lower number of clinical cases using the same implant design was reported a minimal crestal bone loss (0.21mm) after two years of loading restoring mandibular incisors with immediate implants without removal of the abutment [25]. Finally, it has been shown that the use of guided surgery in order to perform an optimal implant placement together with the use of narrow implant with prevention system for minimizing micro-movements and micro-infiltrations at the implant–abutment interface was useful for a minimal marginal bone loss of immediately loaded NDI placed in fresh extraction sockets of mandibular incisors [26].

The correct prosthetic management of the provisional restoration loaded immediately both in the postextraction sites and in the healed sites allows to create an essential emergence profile for obtaining excellent healing of the soft tissues. [27] Survival rates of NDIs appear to be similar when compared to those of standard diameter implants. A recent systematic review reported survival rates >95% [1]. In another study, 255 NDIs underwent immediate loading, of which 194 were placed in healed bone and 61 were placed in post-extraction sites, and no statistically significant difference was highlighted, suggesting that immediate loading of NDIs in post-extraction sites should be considered a safe and predictable procedure as for standard diameter implants [28].

Long-term data on the use of NDI in the posterior regions for the replacement of premolars and molars are not available in the literature. Although their use in some studies has been reported as a promising practice, even with long-term follow-up [1, 28, 29], it is the authors' opinion that the prognosis may not be favorable due to the reduced bone contact surface, resulting these implants not adequate to support the chewing load of this sector of the mouth.

In any case, before decision is made for the use of NDI, biomechanical risk factors must be carefully evaluated, such as the possible presence of parafunctions.

The reduction of the implant diameter can lead to a reduced resistance to fracture [3]. Fatigue fracture has been reported to occur after a long period of loading [4] and is around 0.2–0.6% [5]. In another more recent study, out of 159 NDI inserted in 71 patients no fracture of the

implant fixture was reported in a follow-up period of five years [30].

This risk, therefore, must be considered very low and similar to standard implants, especially in monoedentulous ones. On the other hand, it will be greater in the posterior regions or in the case of overdenture; in the latter case, most studies in the literature recommend splinting the implants with a rigid superstructure [31–32].

In addition to the biological aspects that affect the aesthetic outcome, the long-term prognosis of implant– prosthetic therapy will also be determined by biomechanical considerations. In particular, it will be necessary to evaluate whether an implant with a reduced diameter can be adequate to support the chewing function of these dental elements.

In general, the implant surface must be equal to, or better still superior, given the absence of the periodontal ligament, to the root surface of the tooth to be replaced [33]. This objective can be achieved at a macroscopic level by managing the thread of the implant abutment (increase of up to 30% compared to a standard cylinder) and at a microscopic level, by means of surface treatments of the same (increase of up to 20–45% compared to a smooth surface of titanium). Furthermore, for an increase of 1mm in the implant length the surface is increased by about 10%, and thus an increase of 1mm in the diameter of the implant body determines a widening of 20–40% [34].

It has been determined a correlation between the occlusal load and the root surface of the dental elements, verifying a progressive increase of both parameters from the anterior to the posterior region (Table 6.1) [35]. The table shows that for the replacement of an upper lateral incisor it is sufficient to use an implant that develops a surface equal to or greater than 100 mm².

Based on biomechanical studies [34], the values of the implant surface considering length and diameter and

Table 6.1 Correlation between occlusal load (kg) and root surface (mm²) for the different dental elements (modified by 21).

Tooth	Load (Kg)	Root Surface (mm ²)
Central incisor	16	120
Lateral incisor	18	100
Canine	30	150
First premolar	35	150
Second premolar	40	150
First molar	60	220
Second molar	62	200

Table 6.2 The graph shows how the surface of sandblasted and etched implants varies as a function of length and diameter (expressed in mm²) [20].

rough surface pattern show a potential reference for better planning of an implant–prosthetic rehabilitation (Table 6.2). From this graph, it can be concluded that an implant of 3mm in diameter and 10mm in length is suitable for supporting the chewing function of a lateral incisor of the upper arch.

Therefore, the replacement of upper lateral incisors and/or lower incisors finds its scientific rationale in the use of NDI allowing to achieve aesthetic success, without neglecting the biomechanical aspects for a favorable long-term prognosis.

The success of implant–prosthetic therapy and the reduction of the risk of peri-implantitis depend on a careful assessment and management of the periodontal status in partially edentulous patients and on the assessment and verification of the patient's degree of awareness of personal care and hygiene. This is confirmed since an improvement of oral hygiene effectiveness has been associated with successful implant therapy [36, 37].

When replacing narrow teeth (lower incisors or upper lateral incisors), it is necessary to select an optimal implant diameter and correct emergence profile of the implant restoration to ensure the patient daily oral hygiene measures.

The frequency of peri-implantitis could be related with the location, where those implants are placed. Results obtained after multiple multilevel regression analysis have shown that the probability of suffering from peri-implantitis was approximately four to five times higher in implants placed in the lower-anterior area, with lack of access to interproximal hygiene [38]. In fact, in this area of the mouth, it is difficult to ensure an adequate band of attached gingiva, [39] and we also have less bone availability. Narrow implants due to their reduced diameter in association with platform switching connections can better guarantee bone thicknesses and long-term stability of peri-implant hard and soft tissues facilitating the required oral home care.

Case 4 (provided by Dr. G. Bavetta)

(Figures 1–3) Pictures show a post-extraction site on 10 after healing; the natural tooth was extracted by root fracture; (Figure 4) preoperative CBCT; (Figure 5) preoperative CBCT with virtual project; (Figures 6–11) bone defect, narrow implant placement, and G.B.R. with an absorbable membrane, graft particulate, and growth factor; (Figure 12) one week provisional after four months of healing; (Figure 13) provisional after three months; (Figure 14) final restoration.

Fig 4

Fig 7

Case 5 (provided by Dr. G. Bavetta)

(Figure 1) Preoperative picture shows a compromised #24 tooth after apicectomy with subsequent loss of both gingival and buccal bone tissues. There is an accumulation of dental tartar attached to the root following the dehiscence of the gingiva; (Figures 2 and 3) preoperative CBCT shows the absence of buccal plate; (Figure 4) gingival and bone defect after tooth extraction; (Figures 5–7) bone defect was managed with the insertion of graft particulate up to the free gingival margin and socket repair membrane for bone regeneration with ice-cream technique by Elian and Tarnow [12]; (Figure 8) Implant after

healing and soft tissue management with provisional; (Figures 9–13) Digital management for final restoration (provisional scan, pre-op scan, implant scan, matching of pre-op scan, and provisional scan) according to digital workflow by Bavetta et al. [39]; (Figure 14) Collapse of soft tissue; (Figures 15 and 16) Virtual project of final restoration as a copy of provisional profile; (Figures 17–22) Pre-milled abutment; (Figures 23 and 24) Final restoration; (Figure 25) radiographs after implant placement; (Figure 26) after one-year loading with final restoration; (Figures 27 and 28) pre-op and one-year CBCT.

Fig 4

Fig 14

Fig 20

Fig 22

Fig 23

Fig 27

Case 6 (provided by Dr. G. Bavetta)

(Figures 1 and 2) Clinical and CBCT pre-operative pictures show compromised 24 and 25 teeth with II-grade mobility, which need to be replaced with implant therapy; (Figures 3–5) CBCT pre-op virtual project shows that narrow implant is the best choice to replace these teeth in order to respect safety distance and preserve from performing GBR; (Figure 6) post-extraction site; (Figures 7 and 8) a guided approach was used to insert

implants in order to have the best accuracy in implant placement; (Figures 9–11) prosthetic steps for immediate loading protocol; (Figure 12) four-months healing after provisional; (Figure 13) final restoration; (Figures 14 and 15) radiographic pictures performed at implant placement and after one-year prosthetic loading with final restoration: a great stability in both soft and bone tissue has been obtained.

Fig 2

Fig 3

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Fig 9

Fig 11

Fig 12

Fig 13

Fig 14 Fig 15

Case 7 (provided by Dr. G. Bavetta)

(Figures 1, 2) Clinical and CBCT preoperative photographs; (Figures 3–5) CBCT virtual project; (Figures 6, 7) post-extraction sites and surgical guide in situ; (Figures 8, 9) bone defect was managed by using particulate dentine (see ref. 40) and growth factors; (Figures 10 and 13) radiographs after implant placement and dentine graft; (Figure 14) immediate loading provisional; (Figures 15 and 16) four-months healing; (Figures 17–20) CAD-CAM management; (Figure 21) final restoration.

Fig 6

Fig 7

Fig 13

Fig 18

Fig 19

Fig 15

Fig 20

Fig 21

Case 8 (provided by Dr. G. Bavetta)

(Figures 1,2) Clinical and radiographic figures show the presence of element #7 with a large area of gingival recession and loss of buccal bone tissue; (Figures 3–7) soft- and hard tissue defects were managed by using dentine particulate and growth factors with ice-cream technique; (Figure 8) provisional Maryland bridge to

replace the natural tooth; (Figures 9 and 10) four months of soft- and bone tissue healing; (Figure 11) flap opening for implant placement shows the formation of new bone tissue; (Figures 12 and 13) provisional restoration and management of soft tissues; (Figures 14 and 15) preoperative picture and final restoration.

Fig 1

Fig 4

Fig 2

Fig 9

Fig 14

Fig 15

Case 9 (provided by Dr. G. Bavetta)

(Figures 1 and 2) preoperative figure with #7 agenesia; a Maryland bridge as provisional was used; an area of concavity of the vestibular area is highlighted, indicating bone atrophy of the buccal bone; (Figures 3–8) surgical steps with flap opening, implant placement, G.B.R.; (Figures 9 and 10) four months healing and soft tissue management with provisional restoration; (Figures 11 and 12) final restoration.

Fig 4

Fig 2

Fig 7

Case 10 (provided by Dr. G. Bavetta)

(Figure 1) Preoperative picture shows edentulous area on #7 for agenesia; (Figures 2–7) implant–prosthetic virtual project was obtained by matching DICOM and .STL file; (Figure 8) virtual guided surgery project; (Figures 9–11) implant placement by using guided surgery and flapless technique; (Figures 12 and 13) intraoperative radiographs by using the guided drill and after implant placement; (Figure 14) final restoration; (Figure 15) radiograph after one-year loading with final restoration.

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Fig 14

Conclusions

Based on all these considerations, NDIs are the best choice for replacing natural teeth (especially lower and upper

References

1 Klein, M.O., Schiegnitz, E., and Al-Nawas, B. (2014). Systematic review on success of narrow-diameter dental implants. *The International Journal of Oral & Maxillofacial Implants* 29 (Suppl): 43–54.

lateral incisors) in terms of esthetic and clinical long-term outcomes providing a better stability of peri-implant hard and soft tissues and a better patient compliance in the daily oral hygiene.

2 Davarpanah, M., Martinez, H., Tecucianu, J.F. et al. (2000). Small-diameter implants: indications and contraindications. *Journal of Esthetic Dentistry* 12 (4): 186–194.

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- **3** Vigolo, P. and Givani, A. (2000). Clinical evaluation of single-tooth mini-implant restorations: a five-year retrospective study. *The Journal of Prosthetic Dentistry* 84 (1): 50–54.
- **4** Zinsli, B., Sägesser, T., Mericske, E., and Mericske-Stern, R. (2004). Clinical evaluation of small-diameter ITI implants: a prospective study. *The International Journal of Oral & Maxillofacial Implants* 19 (1): 92–99.
- **5** Romeo, E., Lops, D., Amorfini, L. et al. (2006). Clinical and radiographic evaluation of small-diameter (3.3-mm) implants followed for 1-7 years: a longitudinal study. *Clinical Oral Implants Research* 17 (2): 139–148.
- **6** Brånemark, P.I. (1983). Osseointegration and its experimental background. *The Journal of Prosthetic Dentistry* 50 (3): 399–410.
- **7** Albrektsson, T., Zarb, G., Worthington, P., and Eriksson, A.R. (1986). The long-term efficacy of currently used dental implants: a review and proposed criteria of success. *The International Journal of Oral & Maxillofacial Implants* 1 (1): 11–25.
- **8** Smith, D.E. and Zarb, G.A. (1989). Criteria for success of osseointegrated endosseous implants. *The Journal of Prosthetic Dentistry* 62 (5): 567–572.
- **9** Fürhauser, R., Florescu, D., Benesch, T. et al. (2005). Evaluation of soft tissue around single-tooth implant crowns: the pink esthetic score. *Clinical Oral Implants Research* 16 (6): 639–644.
- **10** Jemt, T. (1997). Regeneration of gingival papillae after single-implant treatment. *International Journal of Periodontics & Restorative Dentistry* 17 (4): 326–333.
- **11** Tarnow, D., Elian, N., Fletcher, P. et al. (2003). Vertical distance from the crest of bone to the height of the interproximal papilla between adjacent implants. *Journal of Periodontology* 74 (12): 1785–1788.
- **12** Elian, N., Cho, S.C., Froum, S. et al. (2007). A simplified socket classification and repair technique. *Practical Procedures & Aesthetic Dentistry* 19 (2): 99–104.
- **13** Bavetta, G. and Paderni, C. (2014). Valutazioni estetiche nella sostituzione di elementi dentari anteriori mediante tecnica socket repair modificata: studio retrospettivo. *Quintessenza Internazionale* 30 (04): 49–55.
- **14** Adell, R., Lekholm, U., Rockler, B., and Brånemark, P.I. (1981). A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. *International Journal of Oral Surgery* 10 (6): 387–416.
- **15** Kraus, B.S., Jordan, R.E., and Abrams, L. (1969). *Dental Anatomy and Occlusion; a Study of the Masticatory System*. Baltimore: Williams and Wilkins.
- **16** Ash, M.M. and Stanley, J.N. (2003). *Wheeler's Dental Anatomy, Physiology and Occlusion*, 8e (ed. Saunders). Elsevier.
- **17** Jullian, W.B. and Rickne, C.S. (2003). *Dental Anatomy: Its Relevance to Dentistry*. Philadelphia: Williams & Wilkins.
- **18** Baggi, L., Di Girolamo, M., Mirisola, C., and Calcaterra, R. (2013). Microbiological evaluation of bacterial and mycotic seal in implant systems with different implantabutment interfaces and closing torque values. *Implant Dentistry* 22 (4): 344–350.
- **19** Lazzara, R.J. and Porter, S.S. (2006). Platform switching: a new concept in implant dentistry for controlling postrestorative crestal bone levels. *International Journal of Periodontics & Restorative Dentistry* 26 (1): 9–17.
- **20** Steinebrunner, L., Wolfart, S., Bössmann, K., and Kern, M. (2005). In vitro evaluation of bacterial leakage along the implant-abutment interface of different implant systems. *The International Journal of Oral & Maxillofacial Implants* 20 (6): 875–881.
- **21** Macedo, J.P., Pereira, J., Vahey, B.R. et al. (2016). Morse taper dental implants and platform switching: the new paradigm in oral implantology. *European Journal of Dentistry* 10 (1): 148–154.
- **22** Vela, X., Méndez, V., Rodríguez, X. et al. (2012). Crestal bone changes on platform-switched implants and adjacent teeth when the tooth-implant distance is less than 1.5 mm. *International Journal of Periodontics & Restorative Dentistry* 32 (2): 149–155.
- **23** Romanos, G.E. and Javed, F. (2014). Platform switching minimizes crestal bone loss around dental implants: truth or myth? *Journal of Oral Rehabilitation* 41 (9): 700–708.
- **24** Romanos, G.E. (2015). Tissue preservation strategies for fostering long-term soft and hard tissue stability. *International Journal of Periodontics & Restorative Dentistry* 35 (3): 363–371.
- **25** Peron, C. and Romanos, G. (2020). Immediate provisionalization of single narrow implants in fresh extraction sockets and healed sites: clinical and radiographic outcomes of 2 years follow-up. *International Journal of Periodontics & Restorative Dentistry* 40 (3): 417–424.
- **26** Bavetta, G., Paderni, C., Randazzo, V. et al. (2022). Marginal bone loss of immediately loaded narrow diameter implants placed in fresh extraction sockets of mandibular incisors. *Journal of Osseointegration* 14 (1): 6–12.
- **27** Peron, C. and Romanos, G. (2017). Socket healing guide technique for single and multiple immediate implants. *Clinical Oral Implants Research (Abstract)*.
- **28** Degidi, M., Piattelli, A., and Carinci, F. (2008). Clinical outcome of narrow diameter implants: a retrospective study of 510 implants. *Journal of Periodontology* 79 (1): 49–54.
- **29** Maló, P. and de Araújo Nobre, M. (2011). Implants (3.3 mm diameter) for the rehabilitation of edentulous posterior regions: a retrospective clinical study with up to 11 years of follow-up. *Clinical Implant Dentistry and Related Research* 13 (2): 95–103.
- **30** Geckili, O., Mumcu, E., and Bilhan, H. (2013). Radiographic evaluation of narrow-diameter implants after 5 years of clinical function: a retrospective study. *The Journal of Oral Implantology* 39 (S1): 273–279.
- **31** Comfort, M.B., Chu, F.C., Chai, J. et al. (2005). A 5-year prospective study on small diameter screw-shaped oral implants. *Journal of Oral Rehabilitation* 32 (5): 341–345.
- **32** Jofre, J., Cendoya, P., and Munoz, P. (2010). Effect of splinting mini-implants on marginal bone loss: a biomechanical model and clinical randomized study with mandibular overdentures. *The International Journal of Oral & Maxillofacial Implants* 25 (6): 1137–1144.
- **33** Misch, C.E. (2006). Consideration of biomechanical stress in treatment with dental implants. *Dentistry Today* 25 (5): 80. 82, 84-5.
- **34** Misch, C.E. (2008). *Contemporary Implant Dentistry*. Mosby.
- **35** Pierazzini, A. (1996). Implantologia avanzata. UTET Collana Odontoiatrica Pratica.
- **36** Armitage, G.C. and Xenoudi, P. (2016). Post-treatment supportive care for the natural dentition and dental implants. *Periodontology 2000* 71 (1): 164–184.
- **37** Jepsen, S., Berglundh, T., Genco, R. et al. (2015). Primary prevention of peri-implantitis: managing peri-implant mucositis. *Journal of Clinical Periodontology* 42 (Suppl 16): S152–S157.
- **38** Rodrigo, D., Sanz-Sánchez, I., Figuero, E. et al. (2018). Prevalence and risk indicators of peri-implant diseases in Spain. *Journal of Clinical Periodontology* 45 (12): 1510–1520.
- **39** Brito, C., Tenenbaum, H.C., Wong, B.K. et al. (2014). Is keratinized mucosa indispensable to maintain periimplant health? A systematic review of the literature. *Journal of Biomedical Materials Research. Part B, Applied Biomaterials* 102 (3): 643–650.
- **40** Bavetta, G., Tornabene, E., Cavataio, A., and Paderni, C. (2016). Scanner intraorale: nuova metodica per la misurazione del collasso tissutale periimplantare. *Implantologia* 2: 61–68.

Prosthetic Abutment Characteristics and Implant–Abutment Maintenance

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Introduction

An implant abutment is defined by the glossary of prosthodontic terms as "that portion of a dental implant that serves to support and/or retain a prosthesis" [1]. It is also defined by Giglio [2], as an intermediate component connecting the implant and the restoration [2], its retained to the implant with a screw (abutment screw) [1] and retains the implant crown with another screw (prosthetic screw) [3] in screw-retained restorations, or with cement in cementretained restorations [1–3]. In addition, the prosthetic abutments of certain implant systems are retained by friction to the implant connection without a screw [1–3].

Considering the number of parts involved in the implant– abutment-restoration, a system of tiers (two and three) was created [2]. Tier-2 indicates that the *abutment and the crown* are a single piece retained to the implant or that the *implant and the abutment* are a single piece and the crown is retained to it. Meanwhile, in tier-3 three interconnected pieces conform the system: *the implant, the abutment, and the crown* [2].

In addition to retaining an implant restoration, implant abutments can have different purposes. For example, healing abutments are used during the healing phase on singlestage implants to assist tissue healing or to modify tissue contours [1]. Implant–angulated abutments are used to correct the implant angulation in case of positional errors, and other abutments are used to support temporary restorations until the complete integration of the implants [1]. Once the osseointegrated implants are restored, the maintenance of the implant restoration is essential to reduce the plaque deposits and their subproducts thus, preventing peri-implant diseases [4].

Different aspects of the design of implant restorations that compromise cleanability have been presented as well as some methods to reduce plaque adhesion and enhance cleanability [5–10].

For example, Walton et al. [5] described that fixed implant restorations were harder to clean compared to removable implant–supported restorations [5]. O'Mahoney et al. [6] analyzed failed implants and found plaque deposits in all the implant–abutment-restoration interfaces, the prosthesis surface, and the implant neck [6]. Teughels et al. [7] found that the transmucosal region of various materials developed robust and thicker biofilms when their surfaces were rough [7]. Zaugg et al. [8] demonstrated that hydrophilic and rough surfaces accumulate considerable amounts of biofilm and the topography influenced the cleanability of the surface [8]. Mazjoub et al. [9] found that larger emergence angles of the restoration resulted in higher marginal bone loss [9]. Finally, Chanthasan et al. [10] demonstrated that the interproximal design of single implant–supported posterior crowns influences the amount of food impaction and the health of the peri-implant gingival tissues [10] (Table 7.1).

Biofilm formation within the oral cavity cannot be eliminated and plaque deposits will evolve and mature over time on all hard dental and prosthetic implant structures if the patients cannot access hidden aspects of the implant restorations [11, 12]. Dab [13] described two aspects that should be considered in implant suprastructure designs: the running room (distance of the most apical portion of the interproximal contacts to the implant platform) and the ridge dimensions. Together, these two parameters dictate the best implant diameter and insertion depth to achieve the ideal emergence angle with the lowest resorption rates of the peri-implant marginal bone [13].

7

Table 7.1 Design parameters of implant restorations and their influence on the formation of biofilm and plaque.

In addition to the previous parameters described by Dab [13], the transmucosal portion of the prosthetic abutment is critical in preventing peri-implant diseases [14]. Therefore, the characteristics of prosthetic abutments including composition, surface, coating, design, emergence profile, and their influence on cleanability and maintenance, require a detailed analysis [14].

The goal of this chapter was to review the characteristics of the tissue interfaces around prosthetic abutments, describe the influence of prosthetic abutment material, surface characteristics, presence of coatings, design and emergence profile, and other variables in implant maintenance. This chapter is supplemented with illustrations, clinical photos, and recommendations for improving prosthetic abutments that can reduce peri-implant diseases by enhancing their maintenance.

Tissue Interfaces Around Prosthetic Abutments

The body of the prosthetic abutment consists of three parts: the connection, the transmucosal area, and the retentive walls. Its vertical dimensions, from the top to the base, encompass the length of the walls, the transmucosal area, and the connection length (Figure 7.1).

The abutment connection stabilizes the prosthetic abutment within the implant, prevents its rotation, seals it against bacteria colonization, and transfers loads to the implant $[15, 16]$. The transmucosal portion of the prosthetic abutment pierces the mucosa. It elevates the abutment/restoration micro-gap away from the bone, provides support to the soft tissue contours and the establishment of a soft tissue cuff (primary barrier to bacteria penetration), and participates in forming the emergence profile of the restoration [17, 18]. The prosthetic abutment walls support the implant restoration, and incorporating different designs, grooves, wall angles, and cement, can increase the retention of cementretained implant crowns [19–21].

The amount and hard or soft tissues in contact with prosthetic abutments depends on the implant insertion depth (subcrestal level, bone level, or tissue level), the prosthetic abutment height, the initial soft tissue thickness and previous soft/hard tissue grafts, and tissue recession [22, 23].

For example, in implants deeply inserted in bone (subcrestal implants), the prosthetic abutment may contact the cortical bone and all the soft tissue thickness. In addition, the restoration base will be in contact with soft tissues. Meanwhile, in supracrestal implants, the prosthetic abutments will have only contact with the soft tissues, and the

Figure 7.1 Illustration of a conventional prosthetic abutment for a single implant for a cement-retained restoration. Three areas can be identified: light blue area represents the walls, the dark blue area represents the transmucosal portion (collar), and the grey area, the connection. The sum of all areas constitutes the abutment length.

Figure 7.2 Effect of the implant insertion depth on the dimensions of the transmucosal portion of prosthetic abutments.

crown will have some soft tissue contact. In bone-level implants, the prosthetic abutment will be in contact mainly with the soft tissues, although some contact with bone can exist in platform-switched abutments [24]. The prosthetic abutments' length will require adjusting with long transmucosal portions in subcrestal implants. Meanwhile, supracrestal implants will require prosthetic abutments with shorter transmucosal portions (Figure 7.2).

Characteristics of the Soft Tissue in Contact with the Prosthetic Abutments

Donley et al. (1992), reported that the epithelia of the soft tissue are attached to the titanium oxide layer of the abutment surface, by a layer of glycoproteins (laminin and fibronectin) of 20nm thickness, and described that the connective tissue fibers run parallel to the abutment surface. However, the abutment's surface properties and geometry can change the fibers' orientation [25]. In addition, Berglundh et al. [26] found that the marginal implant mucosa around the titanium surface contains a collagen/ fibroblast ratio of 109 versus a collagen/fibroblast ratio of 4 in gingival tissues of teeth in canines [26].

Iglhaut et al. [27], completed an exhaustive review of the parameters and differences that can affect the attachment and apical migration of the gingival epithelia on implant abutments [27]. Their findings can be summarized as follows:

• The peri-implant soft tissues are larger $(3-3.5 \text{ mm})$ [26] than the periodontal soft tissues (2.73mm) [28]. The soft tissue dimensions around implants account for 2mm epithelium+1–1.5mm connective tissue, and in teeth, account for 1.6mm epithelium+1.06mm connective tissue [26–28].

- Fewer blood vessels are found in peri-implant soft tissues than around teeth because the blood supply is only supraperiosteal; meanwhile, blood supply around teeth comes from the vascular plexus of the periodontal ligament and the supraperiosteal blood vessels [28]. This can result in down migration of the connective tissue [29, 30]
- Material chemistry influences the apical displacement of the epithelia. Pure titanium, alumina oxide ceramics $(Al₂O₃)$, and zirconia oxide $(ZrO₂)$ abutments will develop a gingival cuff of 3.5mm [31]. Meanwhile, gold and platinum alloys can produce apical displacement of the epithelia and potential enlarged gingival cuffs and bone loss [32].
- The presence of contaminants on the abutment surface produces an apical displacement of the epithelia and increased inflammatory reactions [33].
- Butt-joint implant–abutment connections result in apical epithelial migration, but platform-switched implant– abutment connections result in medial migration of the epithelia [34–36].
- Epithelial attachment on implant abutments is weaker (few hemidesmosomes) and repeated probing can increase probing depths compared to probing around natural teeth [37].
- Multiple abutment connection-disconnection can increase the apical migration of the epithelia and bone loss [38, 39].
- The presence of horizontal microgrooves at the abutment surface can prevent the epithelial down growth by inducing transversal attachment of connective tissue fibers into the abutment [40].

Mature soft tissues around the prosthetic abutments are observed between six and eight weeks after their installation [41], and by the resolution of inflammatory cells observed at eight weeks of healing in samples obtained from the adjacent gingival tissue [42]. Maintaining a strong epithelial and connective tissue attachment to the abutment surface is necessary to resist mechanical injury and prevent the penetration of pathogens into the transmucosal area of the prosthetic abutment [43].

The following sections of this chapter will evaluate prosthetic abutment materials, surfaces, coatings, designs, and emergence profiles that can improve implant maintenance by limiting biofilm formation and establishing a robust gingival attachment.

Prosthetic Abutment Material

In general, four main materials are used for prosthetic abutments: metals and metal alloys, ceramics, hybrid abutments (composed of two or more materials), and polymerbased abutments (Figure 7.3) [44–48].

From esthetic and mechanical aspects, each abutment material possesses specific characteristics that indicate its use. For example, ceramic or coated metallic abutments will provide higher esthetics [49], metallic and zirconia abutments will provide high mechanical strength [50], hybrid abutments permit angle corrections with esthetic advantages [51], and abutments based on polymers will allow long-term temporization [52].

However, from the perspective of maintaining the stability of the soft tissues, the prosthetic abutment material may have other effects [53–55].

The effect of different abutment materials on the soft tissues around dental implants was evaluated by Ferrari et al. [53] in a randomized clinical trial [53]. Forty-seven patients received ninety-seven dental implants. The implants were restored with prosthetic abutments made in titanium (15), titanium with a titanium nitride coating (18), and zirconia (14). After two years of maintenance with a supportive periodontal care program (recalls every four or six months), the keratinized tissue, gingival biotype, peri-implant markers, and recession were similar for the three used materials without differences between groups. Thus, suggesting that plaque control is key to maintaining peri-implant health independent of the abutment composition [53].

Linkevicius et al. [54] evaluated the stability of the periimplant hard and soft tissues when abutments of different materials (titanium, gold alloy, zirconium oxide, and aluminum oxide) were used [54]. The authors completed a systematic review, including clinical studies, human histology, and animal studies. After the search and selection process, nine articles were included: three animal studies, two human histological studies, and four randomized clinical trials. The authors concluded that soft and hard tissues presented similar stability when titanium abutments were used compared to gold alloy, zirconium oxide, and aluminum oxide [54].

The abutment must also resist degradation under function in the oral environment [55]. De Avila et al. [55] reviewed different parameters of the surface and bulk materials used for implant abutments.

The authors concluded that the oxidation of the surface, contamination during handling, and the corrosion processes, can affect the integrity of metallic abutments,

Figure 7.3 Materials used for the fabrication of prosthetic abutments. Although titanium-based abutments are predominant, new hybrid materials and polymers are available thanks to new fabrication methods.

increasing the release of metallic ions, thus, increasing the release of inflammatory cytokines in the adjacent tissues [55].

Biofilm Formation on Different Abutment Materials

Biofilms around prosthetic abutments are formed in supragingival and subgingival locations, the subgingival biofilm is difficult to remove, and their characteristics are dependent on the surface energy, roughness, and partially on the chemical composition of the abutments [56].

To demonstrate the biofilm formation on modern implant abutments, Hahnel et al. [57] completed an experimental study using disks made in zirconia, polyetheretherketone (PEEK), and titanium and compared these with control disks made in polymethylmethacrylate (PMMA). The disks were highly polished and the surface roughness and energy were measured. Afterward, salivary biofilm was seeded with *S. mutans, S. gordonii, A. naeslundii, and C. albicans* and the adhered and dead microorganisms were measured.

Their results showed lower roughness for PEEK $(Ra = 0.04 \,\mu\text{m})$ and PMMA $(Ra = 0.05 \,\mu\text{m})$ compared to zirconia ($Ra = 0.16 \mu m$) and titanium ($Ra = 0.17 \mu m$). The surface energy (drop angle) was higher for PEEK (42.19), followed by PMMA (39.14), titanium (35.62), and zirconia (31.72). After 20 hours, the lowest cell availability was found in PEEK, followed by zirconia, titanium, and PMMA. After 44 hours, the biomass increased in all the materials. In regards to dead microorganisms, after 44 hours zirconia (74.87%) showed higher percentages of dead microorganisms, followed by titanium (68.82%), PEEK (63.72%), and PMMA (60.17%) [57].

Desch et al. [58], evaluated the biofilm formation on 192 zirconia and titanium disks $(n = 96)$ bonded to intraoral splints in twelve patients. Samples were collected at different time points (6 hours, 24 hours, three and five days), and volume of biofilm, and number of live and dead bacteria were measured with confocal laser scanning microscope. In addition, the microbial composition was analyzed using DNA sequencing analysis [58].

The results showed that the volume of biofilm increased with the time on both materials, reaching a plateau at three days. The microbial composition differed over time but was not influenced by material composition. Some microorganisms were detected in higher percentages at the first hours (*Abiotrophia defectiva, Lautropia mirabilis, Ralstonia solanaceraum, Rothia, Prevotella, and Streptococcus*) but decreased at three and five days. The remaining microorganisms started with low percentages and increased over time (*Actinomyces, Bergeyella, Capnocytophaga, Eikenella, Fusobacterium, Neisseria, Porphyromonas, Veillonella*). The authors concluded that there are no differences between zirconia and titanium in the quantity of adherent bacterial species after three days [58].

Zeller et al. [59] studied the biofilm formation on metallic alloys, zirconia, and PEEK [59]. A removable intraoral appliance was designed to retain 15 disks distributed equally at the palatal area and inserted in sixteen patients. The disks were designed with 5mm diameter and 1mm thickness. They were manufactured in five materials: goldbased andsilver-based noble metal alloy, zirconia, PEEK, or titanium zirconium alloy. After the appliances were delivered, samples were collected after 24 and 48 hours. The results showed that the gold and silver-based alloys presented the lowest number of colonies forming units, and titanium-zirconium alloy, zirconia, and PEEK showed similar values. Thus, indicating that silver and gold-based alloys can be used in the transmucosal portion of implants to limit the biofilm formation [59].

Herrman et al. [60] compared early and mature biofilm (formed after 3 days and after 31 days, respectively) growing in simulating implant surface, implant collar, and abutments [60]. Disks with 5mm diameter and 2mm thickness were fabricated in four materials: sandblasted and acid-etched titanium ($Ra = 1.87 \mu m$, simulating the implant surface), machined titanium ($Ra = 0.18 \mu m$, simulating implant collar), titanium-aluminum-vanadium alloy $(Ra = 0.16 \,\mu\text{m})$, and zirconia $(Ra = 0.74 \,\mu\text{m})$ simulating the abutment roughness. The disks were not polished to maintain their original roughness and topography. Mandibular acrylic appliances were developed and the disks were attached facing the lingual side. The appliances were inserted in 14 patients.

The devices were removed only during eating and for routine oral hygiene. Total bacteria were detected using polymerase chain reaction. Their results showed that bacteria colonized all the materials after exposure to the oral cavity at 3 and 31 days. Curiously in the early biofilm, bacteria colonies at three days were lowest for machined titanium $(3.83 \times 10^8 \pm 3.13 \times 10^8)$ and highest for zirconia $(5.63 \times$ $10^8 \pm 4.83 \times 10^8$). Mature biofilm at 31 days showed bacteria counts higher for zirconia (6.66 \times 10 8 \pm 4.19 \times 10 8) and lower for titanium alloys $(4.67 \times 10^8 \pm 2.81 \times 10^8)$. The comparison of machined versus sandblasted-acid etched titanium showed higher bacterial counts for sandblasted-acid etched titanium compared to machined titanium. Most of the detected bacterial species increased cell numbers in mature biofilms compared to the early biofilms. The authors concluded that the higher roughness observed in zirconia abutments explains the higher bacterial cell counts compared to machined titanium and titanium alloy [60].

More recently, Del Rey et al. [61] completed a systematic review on the biofilm formation of alternative abutment materials compared to titanium. Studies evaluating biofilm formation on metallic, ceramic, or polymeric materials were included.

The following variables were analyzed: microbial counts, biofilm thickness and area, and cell viability. In total, 19 studies were included (10 clinical and 9 *in situ*). The alternative materials used in these studies were alumina, cobalt chromium, polytetrafluoroethylene (PTFE), gold and goldplatinum alloys, and zirconia. The authors found that titanium versus zirconia abutments showed comparable bacteria and inflammation levels and health conditions, but bacteria levels increased over time. Gold alloys showed thick biofilms and more extensive bacteria surface coverage than titanium and zirconia, and all the surfaces accumulated supragingival biofilm [61].

Despite difficult comparisons between materials, and the short-term of the included studies, the authors suggest studies with extended follow-up periods.

Prosthetic Abutment Surface

The transmucosal portion of the abutment surface is confined at the basal area by the implant platform and the cortical bone, laterally by the connective tissue and the gingival epithelial tissue, and coronal by the interface abutment-restoration and the restoration. Therefore, different modifications have been completed on the transmucosal portion of the abutments to improve the behavior of the adjacent connective and epithelial tissues. These surface modifications can be divided into machined, microgrooved, coated, and functionalized surfaces.

Machined Surfaces

In in vivo studies, machined surfaces have shown that the fibroblasts of the connective tissue are oriented with their long axis parallel to the abutment [62, 63], there is a lack of connective tissue attachment to the abutment surface, and the junction epithelia is long [40]. In addition, the original machined surface changes after two months of exposure to the oral environment from smooth (surface roughness $Sa = 0.259 \,\mu m$) toward a rougher surface (surface roughness $Sa = 0.430 \,\text{\mu m}$ [64].

In in vitro studies, fibroblasts were attached firmly to the surface of machined titanium disks. Their bodies were flat, rounded, with short and thick lamellipodia, without clear orientation on the surface [65]. In in vivo studies, immunofluorescent analyses of oral epithelial-like cells showed well-developed actin filaments and expression of adhesion proteins (laminin-5) [66]. Finally, the soft tissues adjacent to machined surfaces showed expression of Intercellular Adhesion Molecule (ICAM-1), Matrix Metalloproteinases (MMP's), and MMP-9 (which regulates neutrophils migration) [67].

The epithelium facing the machined surface was analyzed by Atsuta et al. [68]. The authors described three zones: upper, middle, and lower portion. At the lower portion, adhesion structures (hemidesmosomes) were present in a zone of 100nm covering about 80% of the lower area. At the middle portion, adhesion structures were present in 40% of the area [68].

Micro-Grooved Surfaces

Early studies completed by Brunette et al. (1991) demonstrated in vitro and in vivo that organized topography (repeated patterns like grooves) on the material surface can guide cell orientation and cell spreading [69].

Weiner et al. [70] evaluated the connective tissue, epithelia, and crestal bone around micro-grooved and machined implant collars [70]. In their study, 36 implants (3.75mm diameter and 8mm length) were inserted in six dogs. Each dog received six implants (three implants with machined collar and three implants with micro-grooved collar) in the bilateral healed premolar and molar areas of the mandible. Both collars had a length of 2mm. The micro-grooved collar was divided into three zones an upper machined zone of 0.5mm, a middle zone of 0.7mm was laser microgrooved with micro-grooves of 8μm wide, and the apical zone of 0.8mm laser micro-grooved with micro-grooves of 12μm wide. Samples were obtained at three and six months after implant insertion. Histologic samples were obtained and evaluated for soft tissue recession, bone growth, bone loss, and the number of osteoclasts. The authors found that the micro-grooved surfaces presented minimal soft tissue recession, lower bone loss, and osteoclast activity compared to machined surfaces for all the evaluated periods. Collagen fibers were attached to the middle zone of the micro-grooved surface, and some microgrooves of the apical zone of the micro-grooved surface showed bone formation. Meanwhile, the machined collars did not show attachment of collagen fibers or bone formation [70].

Nevins et al. [40] completed an animal study in dogs to evaluate the influence of two implant surfaces with two types of healing abutments [40]. The implants were tapered, with or without a 0.5mm machined collar, and their surfaces were treated by resorbable blast texturing. The implants were inserted at the bone level in healed mandibular ridges and received either a micro-grooved healing abutment or a machined healing abutment. Healing abutments, soft tissues, implants, and bone were evaluated with micro-computed tomography (micro-CT), transmitted light, and scanning electron microscopy (SEM).

The histological analysis showed bone and connective tissue attached to the micro-grooved zone, and the junctional epithelium ending at the highest coronal portion of the micro-grooved zone, without the characteristics of a long junction epithelium given that the connective tissue attachment impeded the apical migration of the epithelium. The SEM analysis showed dense connective tissue attached to the micro-grooved abutment surfaces. Meanwhile, the junctional epithelium migrated apically on machined abutment surfaces, and no connective fiber attachment was observed [40].

Shin et al. [71], evaluated micro-grooves influence on the healing of peri-implant tissues in immediate implants in dogs [71]. Implants with external hexagon connection, with the same geometry (cylindric-apical taper) and dimensions but different collars. The control group possessed a machined collar, and the test group had a microgrooved collar. Four dogs received sixteen implants (eight implants of each group) inserted immediately after extracting the third and fourth mandibular premolars at a bone level or slightly apical to the lingual bone crest. After three months, the samples were processed for histological analysis. The results showed that the first bone-to-implant contact was higher in micro-grooved implant collars than in machined collars and demonstrated that the connective tissue fibers of the peri-implant mucosa were attached perpendicular to the collar. Meanwhile, the first bone-toimplant contact was more apical in machined collars, the connective tissue fibers were parallel, and the mucosa penetrated below the machined collar. Thus, the authors conclude that the micro-grooved collars improve the attachment of soft and hard tissues and reduce the marginal bone loss in immediate implants [71].

Micro-grooved abutments also can reach reattachment of the soft tissues to their original levels. Therefore, using micro-grooved components like healing abutments and micro-grooved prosthetic abutments are recommended when multiple abutment replacements are required [72]. Furthermore, enhanced cell adhesion and soft tissue sealing to the micro-grooved surface benefit against bacterial intrusion [73].

In a literature review, Ketabi et al. (2015) concluded that micro-grooves at the implant collar inhibit apical migration of the soft tissues, thus reducing crestal peri-implant bone loss. Characteristics of the micro-grooved surface, including the dimensions of the micro-grooves, their pitch, valleys distribution, and their nano topographical features, allow basal tissue attachment and stop epithelia from migration. In addition, the connective tissue surrounding micro-grooved collars resembles that located around natural teeth [74].

Geurs et al. [75] completed a clinical study to evaluate the healing and formation of the connective tissue attachment around micro-grooved and machined abutments. It was observed that connective tissue attachment occurs on micro-grooved surfaces during healing and even after replacement by new micro-grooved abutments. However, machined abutments resulted in epithelial attachment without connective tissue presence. After substitution by micro-grooved abutments, this situation did not change. However, if a wound was created after the removal of a machined abutment and a micro-grooved abutment replaced this, then connective tissue attachment was observed. The authors recommended removing the epithelium to allow connective tissue attachment when replacing machined by micro-grooved abutments [75].

Finally, a longitudinal randomized controlled clinical trial showed that micro-grooved abutments supporting cemented single implant restorations reduced the sulcus depth and crestal bone loss compared to machined prosthetic abutments supporting cemented single implant restorations. The authors concluded that using micro-grooved implant collars and micro-grooved abutments is a dual strategy that should be used to promote a more robust and healthier soft and hard tissue integration [76].

Coated Surfaces

Coatings have been applied to the surface of prosthetic abutments with two main goals: to improve soft tissue attachment and reduce bacteria colonization [77, 78]. The improvement of the soft tissue attachment to the abutment surface should be evidenced by the attachment of collagen fibers in a perpendicular direction, reduction of the apical migration of the gingival epithelium, improved tissue healing, and therefore, limited crestal bone resorption [79]. The reduction of bacterial colonization should be reflected in delayed biofilm formation, reduced plaque, reduced infection, reduced inflammation, and prevention of bone loss [80, 81].

The following antibacterial coatings have been tested on titanium abutment materials: anodic spark deposition, nanoporous titanium oxide $(TiO₂)$, titanium-bismuthgallium (Ti-Bi-Ga), doxycycline, diamond-like carbon (DLC), silver and silver nanoparticles, titanium nitride, zirconia nitride [82–90] (Table 7.2).

The following coatings for the improvement of the soft tissue behavior around titanium abutment materials have been evaluated: collagen, titanium nitride, zirconium nitride, and Vitamins D and E [77] (Table 7.3).

Although coatings have the potential for development, only Doxycycline, Silver nanoparticles, Titanium, and Zirconia nitrides, proved antibacterial activity against a limited number of microorganisms (*Staphylococcus epidermidis, S. aureus, Streptococcus mutans, S. salivarius, S. sanguinis, S. sobrinus, and Pseudomonas aeruginosa*). Furthermore, collagen was the only coating that demonstrated human fibroblast adhesion and proliferation [77, 82–90]. Finally, the effects of mechanical or chemical cleaning methods on the integrity of the coatings after long-term exposure to use and cleaning are unknown.

Functionalized Surfaces

A biofunctionalized surface uses a coating that confers properties different from the base materials by modifying their composition, structure, and morphology without altering their mechanical properties. Such modifications can improve biocompatibility (increase cell attachment and interactions), reduce bacterial adhesion, and reduce corrosion and wear [91, 92].

The methods for surface functionalization include the addition of ceramic, polymer, and polymeric gel coatings, and ceramic coatings are preferred given their high stability and osteoconductivity [93, 94]. The stability of a biofunctionalized surface in the intraoral environment can be affected by variable conditions of temperature, pH, lack of homogeneous coating and mismatch with the surface, residual stresses, and continued degradation. These mechanisms are poorly understood and can affect their efficacy [91, 95].

The Transmucosal Area of Prosthetic Abutments

The transmucosal portion of the prosthetic abutment passes through the gingival tissues and in different emergence angles continues toward the cervical contour of the implant restoration, these angles vary from 0° to 90°, and provide physical support for the peri-implant soft tissues [96].

For example, the profile of the prosthetic abutment, as per Steigmann et al. [97] can be concave, slightly concave, or convex. When the implants are too buccal, concave abutments are indicated. When the implants are centered in the ridges, slightly concave abutments could be used, and for too-palatal implants, convex abutments should be preferred. Thus, convex abutments can help in displacing the buccal tissues facially; meanwhile, slightly concave abutments maintain the position of the soft tissues, and concave abutments could be used in case of limited buccal space or thick gingival tissues [97].

Chu et al. [98] described the implant restorative emergence profile (IREP) as "the contour of the implant/ abutment crown complex as it arises from the restorative platform of the implant and emerges from the peri-implant soft tissues" and also called it the "implant restorative contour" [98].

Table 7.2 Antibacterial coatings tested on titanium abutment materials. Only Doxycycline, silver nanoparticles, and titanium and zirconia nitride coatings showed antibacterial activity.

Table 7.3 Coatings for the enhancement of the soft tissues for titanium surfaces. Only collagen coatings produced increased cell adhesion, cell viability, and proliferation.

In single implants, the IREP should imitate the emergence profile of the extracted tooth on all sides (buccal, mesial, distal, and lingual); however, this is not always possible without complementary procedures like bone and soft tissue grafts and temporization [98]. Two areas of the soft tissues that surround the whole circumference of the transmucosal portion of the prosthetic abutments were described: the critical and the subcritical contours [99]. The critical contour is an area of around 1mm (coronalapical length) immediately apical to the gingival margin. It can be facing the prosthetic abutment, the implant crown, or both, depending on the position of the interface abutment/crown. On the buccal side, the critical contour determines the level of the gingival margin; meanwhile, in the interproximal areas, it determines the shape of the implant crown (square or triangular) [99]. The subcritical contour is the area below the critical contour that extends to the implant platform. Its dimensions are variable (short in shallow implant placement and long in deep implants) and help in establishing the IREP [99].

Correlations between the peri-implant soft and hard tissues/implant prosthetic abutment/implant restorations are complicated given variable patient and implant factors (gingival biotype, location, healed versus post-extraction implants, microbiome, implant insertion depth, type of implant connection, abutment contour characteristics, interface abutment-crown, materials, and others) [100].

To overcome this issue, Mattheos et al. [101] coined the term "implant supracrestal complex" (ISC) to describe and correlate the gingival tissue, mechanical components, and bacteria in the transmucosal portion of the implant prosthesis [101]. In addition, in a recent systematic review of clinical studies analyzing the elements of the design of the ISC, Mattheos et al. [102], concluded that bone-level implants with abutments with wide emergence angles and convex abutment profiles increase the risk for peri-implantitis. Also, prosthesis with accessible and cleansable designs reduces peri-implant mucositis. If mucositis exists, its treatment is more difficult with deep peri-implant sulcus [102].

Gomez-Meda et al. [103] described a concept for the vertical distribution of the subgingival design of emergence profiles around implant restorations. In their approach, the subgingival emergence profile contains three zones: the esthetic zone (E), the bounded zone (B), and the crestal zone (C).

The esthetic zone (E) influences the position of the free gingival margin, is around 1mm thick, and its contour should be convex and establish the morphology of the clinical portion of the implant crown. In addition, it contains the free gingiva.

The bounded zone (B) possesses between 1 and 2mm thickness. It is located below the E zone. It is determined by the thickness of the surrounding soft tissues, the implant position, and the implant neck design and should be preferably concave. It contains the gingival epithelial tissue.

The crestal zone (C) is 1–1.5mm in thickness and is located below the B zone. The abutment contour should be straight or concave in this area to avoid pressuring the adjacent hard tissues. Its dimensions also depend on the implant insertion depth. The supracrestal connective tissue is present in this area [103].

Transmucosal Designs of Prosthetic Abutments

Different transmucosal designs have been used in prosthetic abutments, including parallel, concave, convex, convergent, divergent, and customized configurations [97, 103–107].

A parallel abutment typically possesses walls without angular, geometrical, or diameter changes. Concave abutments possess walls that converge toward a narrower central portion and then diverge. In convex abutments, the walls diverge from the center to an area of maximum curvature, then converge to the coronal. In a convergent abutment, the walls progressively converge toward the coronal. A divergent abutment possesses walls that gradually diverge from the center. Finally, a custom abutment imitates the contour of the contralateral tooth or adopts multiple unique geometries required for the restoration design [103–107].

Valente et al. completed a systematic review to evaluate the effect of different abutment geometries (convergent or concave versus divergent and parallel) on marginal bone loss and esthetics [104]. Five studies fulfilled the inclusion criteria. Their results showed that concave and convergent abutments were beneficial in maintaining soft tissue levels and reducing bone loss [104].

Siegenthaler et al. [108] completed a one-year clinical trial on anterior implant restorations over convex or concave abutments to evaluate the stability of the gingival margins. Their results showed that concave abutments presented a frequency of buccal gingival recessions of 14.3%; meanwhile, abutments with convex profiles presented a frequency of buccal gingival recessions of 64.3% [108], The authors concluded that after a year, concave abutments stabilized the gingival margin better than convex abutments.

Customized abutments are non-prefabricated abutments created by transferring the emergence profile from temporary restorations, natural teeth (data obtained from CT scanners), or fabricated by CAD/CAM (computer-assisted design/computer-assisted manufacturing) to accommodate the soft tissue thickness, implant depth, and angulation, to suit esthetics and other regional requirements specific for each case [109-111].

Custom abutments were developed to solve complex cases that could not be treated with traditional prosthetic
abutments (i.e. implant misplacement, esthetic failures) [112]. Cardoso et al. [112], evaluated the preferences of prosthodontics specialists for using custom or prefabricated abutments. The survey results showed that custom zirconia and titanium abutments were preferred for the anterior esthetic region. Meanwhile, prefabricated abutments were preferred for the posterior regions [112].

Schepke et al. [113] completed a clinical trial to evaluate the patient outcomes for stock (prefabricated) versus customized zirconia abutments. Their results showed that for all the parameters evaluated (peri-implant bone level, plaque index, bleeding on probing, probing depths, soft tissues, and patient satisfaction) after one year, the clinical variables were similar, and the patient satisfaction comparable favoring equally the use of prefabricated or custom abutments [113].

However, newer data suggest the opposite [114, 115]. Romanos et al. [114], in a recent long-term retrospective study, compared the crestal bone loss and implant failure of prefabricated versus customized abutments after 10 years. Their results showed that prefabricated abutments reduced crestal bone compared to custom abutments. In addition, 4.1% of implants with customized abutments were lost. Meanwhile, no implant with prefabricated abutments was lost [114].

Lin et al. [115], evaluated the marginal bone loss by comparing stock abutments versus customized CAD/CAM abutments after one year of follow-up. Their results showed that the mesial and distal bone levels were lower in custom abutments compared to stock abutments. The authors attribute these differences to some drawbacks of the manufacturing process, intolerances, connection fit, and global stability that can affect the long-term performance of customized abutments [115].

Evaluation of the Emergence Profile of a Prosthetic Abutment

As per the glossary of prosthodontic terms (ninth edition), the emergence profile is defined as "the contour of a tooth or restoration, such as the crown on a natural tooth, dental implant, or dental implant abutment, as it relates to the emergence from circumscribed soft tissues" [1].

A critical consideration for this definition is that it is global and does not clarify landmarks to evaluate the emergence profiles of prosthetic implant abutments. Therefore, there is no consensus about how they should be measured.

For example, Souza et al. (2018), [116] traced a line parallel to the central axis of the implant and measured the angle formed with the wall of the abutment. In this method, the transmucosal profile possesses straight walls, and the

Figure 7.4 Emergence profile according to Souza et al. (2019). Red dotted central line represents the central axis of the implant, the red dotted line intersecting the abutment profile represents an extension of the implant axis, the most external red dotted line represents the abutment profile, the emergence profile in this image shows a narrow-angle represented in with a white arc.

abutment sides have equal angles. In their study, narrow angles (15°) resulted in less bone resorption than abutments with wider angles (45°) (Figure 7.4).

However, when the abutment walls at the transmucosal portion are not straight and the walls acquire complex profiles, the methods recommended by Katafuchi et al. [117] and Yi et al. [118] can be used.

Their methods describe two parameters: the emergence angle and the emergence profile.

The emergence angle is the angle of the most apical portion of the abutment when it emerges from the implant connection in relation to a line parallel to the central axis of the implant. The emergence profile is the whole contour of the transmucosal portion of the abutment. It can be concave, convex, slightly concave, or slightly convex [117, 118]. These methods evaluate the emergence profile and angles of custom abutments (Figure 7.5).

Huang et al. [119] in a randomized controlled trial, evaluated the influence of the buccal emergence profile on the peri-implant tissues. Their study was done using cylindric implants restored at the implant level. Their references were the implant's long axis, implant shoulder, buccal mucosa, and the most coronal portion of the gingival tissue in contact with the restoration.

The authors defined the emergence angle as the "angle between the tangent line of the restoration at the most coronal point of the buccal mucosa and the axis of the implant."

Figure 7.5 Emergence angle and emergence profile according to Katafuchi et al. [117]. Emergence angle and emergence profile in abutments with asymmetric walls. In this buccolingual view, the abutment possesses two different emergence angles (a1 is wider, and a2 is narrower), and two different emergence profiles (p1, p2, one for each wall). In this example, the emergence profile is identified by the solid blue line. P2 is slightly concave, and p1 is concave.

The authors defined the buccal emergence profile as "the buccal profile of the restoration from the implant shoulder to the gingival margin." In addition, the authors measured the ratio resulting from the width of the buccal mucosa (W) and the gingival height (W/H ratio). They determined that a W/H ratio of 1.3 maintained an optimal emergence profile [119] (Figure 7.6a, b).

The emergence angle and profile are measured and evaluated in various forms. The whole circumference of the transmucosal portion can present different emergence angles and emergence profiles (especially in customized abutments). There are four areas of the implant abutment/ restoration in which the emergence profile and emergence angle form: buccal, mesial, distal, and lingual surfaces. In deeply inserted implants, the emergence angles and profiles form over the transmucosal portion of the abutment; meanwhile, in shallow implants or implants restored directly to the implant platform, the emergence profiles and angle can form over the restoration surface (Figure 7.7).

Landmarks are completed using tangent lines over the abutment or the restoration, and the angles usually form at the intersection with the implant axis and the gingival margin. The buccal emergence angle and profile support the esthetic gingival margin, the interproximal emergence profile and angles support the papillae, and the lingual emergence angles and profiles support the lingual gingival margin. Narrow angles and concave profiles are preferred over wide angles and convex profiles because the soft tissue attachment is more stable, and less crestal bone loss can be expected.

Figure 7.6 (a) Emergence angle as per Yuan et al. [119]. Emergence angle is formed by a tangent line from the highest soft tissue contact with the restoration to a line tangent to the implant platform, intersection occurs at the implant shoulder. (b) Emergence profile as per Yuan et al. [119]. Emergence profile is measured from the angle formed between a line tangent to the lower contour of the restoration in contact with the soft tissues and the central axis of the implant.

Figure 7.7 Example of the emergence angles and emergence profiles in evaluations from the buccal and mesial sides. The buccal view shows different emergence angles dictated by the abutment and crown contours. The transmucosal portion of the abutment is short. The mesial and distal views show slightly concave contours at the base of the buccal side and a divergent contour at the lingual. Higher metallic collar at the interproximal and lingual sides. White lines represent emergence angles and profiles, red dotted line the gingival level, and black dotted line implant axis.

Prosthetic Abutment Maintenance

In this subsection, the instruments and methods used to provide hygiene in patients with dental implants will be discussed, including the effect of instrumentation on the abutment surface and the efficacy of the different methods to remove biofilm, plaque, and calculus. In addition, recommended protocols for maintenance are presented.

Effects of Instrumentation on the Abutment Surface

The effects of different hygiene instruments on the surface of titanium implant abutments were evaluated by Rapley et al. [120]. Cylindric abutments mounted on implants were subject to mechanical debridement using ten methods combining different times and strokes. The methods were evaluated: (i) Rubber cup mounted in a handpiece rotating at slow speed, (ii) Rubber cup rotating at slow speed with fluor pumice, (iii) Air polishing

perpendicular to the abutment, (iv) interproximal brush with back-and-forth strokes, (v) Interproximal plastic tip with back-and-forth strokes, (vi) Toothbrush with reciprocal strokes, (vii) Plastic scaler with vertical movements, (viii) Ultrasonic scaler with vertical strokes, (ix) Stainlesssteel scaler, and (x) control.

After the methods were completed, the abutments were evaluated by scanning electron microscope (SEM) using different magnifications. The amount of surface damage was evaluated by comparing the surfaces after each technique with the control surface and was graded as follows: $AA =$ smoother, $A =$ comparable, $B =$ slightly rougher, $C =$ moderately rougher, $D =$ severely damaged.

Group 2 produced smoother surfaces (grade AA), Group 4 did not induce damage (grade A), groups 1, 3, 4 and 6 produced minor damage (grade B), the groups 8 and 9 (metal scaler and ultrasonic scaler) produced severely damaged surfaces (Grade D). The authors concluded that conservative plaque debridement and maintenance methods do not alter the abutment surface [120].

Mengel et al. [121] completed an in vitro study to evaluate the effect of eight cleaning methods on titanium surfaces (machined, sandblasted, and titanium plasma-sprayed) (i) Titanium curettes, (ii) Gracey curettes, (iii) Plastic curettes, (iv) Rubber cups $+$ prophy paste, (v) Ultrasonic scaler, (vi) Air polishing, (vii) Ultrasonic scaler with soft tips, and (viii) Ultrasonic scaler with hard tips. The loss of substance from the titanium surfaces was evaluated. Four grades of substance loss were assigned (none, slight, moderate, and pronounced). The evaluations were carried out with the SEM.

Their results showed that air-polishing, rubber cup, and plastic curettes did not alter the titanium surfaces; meanwhile, ultrasonic scalers with soft tips and titanium curettes produced a slight loss of substance. Finally, the Gracey curettes and ultrasonic with metallic tips produced pronounced surface alterations and material loss. The authors concluded that air-polishing, rubber cups, and plastic curettes could be used to remove supragingival calculus and plaque without risk of surface damage [121].

Recent studies by the same group in (2004) [122] evaluated the effect of different instruments on the integrity of titanium abutments coated with titanium nitride and without coating. Evaluations of damage, roughness, removed amounts of titanium and coating after a single treatment of the surface with different methods (titanium, plastic, and steel curettes), rubber cup, ultrasonic scaler with metallic tips, and air scaler + cleaning powder.

Their results showed that all the groups suffered surface alterations and material loss when steel curettes and ultrasonic scalers with metallic tips were used. The other methods produced moderate damage to the non-coated surface and minor damage to the coated surfaces. The authors concluded that the coating protected the titanium surface from direct damage, but the detachment of the coating and the increased roughness after the maintenance are of concern [122].

Gehrke et al. [123] studied the titanium nitride coating applied to CAD/CAM abutments. Acrylic scalers, titanium curettes, and ultrasonic scalers with metallic tips were used on twelve abutments. All the scalers were applied with 150 or 200 g pressure against the abutment surfaces. After scaling, the surfaces' roughness and characteristics were analyzed with optical profilometry and SEM. The results showed that ultrasonic scalers severely damaged the titanium nitride coatings and produced increased roughness, and acrylic curettes did not damage the surface. The authors concluded that acrylic instruments could be recommended for cleaning the transmucosal portion of abutments [123].

Recently, Sirinirund et al. [124] completed an experimental study to evaluate the effects of different instruments used for supportive care on titanium machined and

roughened surfaces. Topographical changes and the efficacy of instrumentation were evaluated. The implants were covered with cyanoacrylate and toluidine blue dye to resemble attached calculus. The surfaces were cleaned with curettes (stainless steel, plastic, and titanium), ultrasonic instruments with plastic and metallic tips, titanium brush, and air-polishing, simulating one year and five years of supportive care. Afterward, a stereomicroscope was used to evaluate the efficacy of calculus removal (percentage of residual calculus), and an atomic force microscope and SEM to evaluate the surface topography.

The most efficient instruments to remove simulated calculus were ultrasonics with metallic tips followed by titanium brush and ultrasonics with plastic tips. Stainless steel curettes were more efficient than titanium, and plastic curettes were inefficient. Air-polishing efficiently removed calculus deposits on rough surfaces but not in machine. Mechanical methods produced micrometric and nanometric topographical changes on the titanium surface, except for air-polishing. In addition, plastic instruments released plastic debris, and metallic instruments released metallic debris [124].

Biazussi et al. [125] completed an in vitro study using sodium bicarbonate and glycine powders applied to titanium disks with a smooth surface ($Ra = 0.1 \mu m$). The authors used air-polishing with the same device and operation settings situated at a 4mm distance from the disks. The powders differed in size (sodium bicarbonate powders with a particle size of $220 \mu m$ and glycine powders with a particle size of less than $63 \mu m$). After air-polishing, the roughness of the samples was evaluated with optical profilometry. The results showed that the surface roughness barely changed when glycine powders were used $(Ra = 0.112 \pm 0.033)$; meanwhile, the sodium bicarbonate powders slightly increased the roughness of the samples (Ra = 0.212 ± 0.058). The authors concluded that glycine powders used during simulated implant abutment prophylaxis minimally affect the surface roughness [125].

Tran et al. [126] compared cleaning techniques (air abrasion, ultrasonic scaler, carbon, and carbon stainlesssteel scalers, and 40% citric acid) on three different titanium surfaces (smooth, sandblasted, and sandblasted + acid etched). Titanium disks with the three experimental surfaces were seeded with biofilm from human saliva. After applying the debridement techniques, the remaining biofilm was evaluated with SEM and crystal violet methods. Most biofilms were eliminated with the air polishing methods (94.84–96.53% of efficacy in biofilm removal), and the mechanical methods were the least efficient in biofilm reduction. Meanwhile, citric acid was comparable to mechanical methods for biofilm reduction. The surfaces were severely damaged with all the mechanical methods, but citric acid and air-polishing resulted in the lowest surface damage.

The authors concluded that air-polishing with glycine powders is the recommended method for abutment debridement based on the efficacy of biofilm removal and minimal surface damage [126].

In summary, of the mechanical methods used for titanium abutment maintenance, cleaning, and debridement, the air polishing methods are the most efficient in removing plaque, simulated calculus, and biofilm with the lowest surface alterations. Other mechanical methods possess limited capabilities to reduce plaque or biofilm and produce significant damage to the titanium surfaces and coated titanium surfaces represented in scratches, increased roughness, and titanium particle release.

Efficacy of Cleaning Methods

Schmage et al. [127] evaluated the cleaning efficacy of instruments used for implant prophylaxis. In this in vitro study, disks with polished or acid-etched surfaces were seeded with Streptococcus mutans biofilm layers, and seven methods for prophylaxis were evaluated. The methods included: plastic curettes, carbon-fiber-reinforced plastic curettes, sonic prophylaxis brush, rubber cup + prophylaxis paste, sonic PEEK inserts, ultrasonic PEEK inserts, and air-polishing.

After cleaning, the disks were stained with a plaquedisclosing solution and analyzed by light microscopy. Their results showed that the sonic and ultrasonic PEEK tips and the air polishing with glycine powders achieved the highest cleaning efficacy. The worst cleaning efficacy was produced by manual instrumentation in polished disks (<18% of remaining biofilm) and the rubber cups in acid-etched surfaces <11% of remaining biofilm). The authors concluded that the cleaning efficacy of less than 4% of remaining biofilm is not achieved by any methods and is affected by the surface topography [127].

Louropoulou et al. [128] completed a systematic review to evaluate the effects of mechanical instrumentation on contaminated implant surfaces. Fourteen studies (major part in vitro) reported that plastic instruments and rubber cups were inefficient in the decontamination of titanium surfaces. Meanwhile, rotating and ultrasonic instruments produced some positive results. The best method was airpolishing. The authors concluded the following: metal curettes were ineffective in removing calcified deposits from machined surfaces but effective on sandblasted surfaces, scalers with carbon tips remove plaque from sandblasted surfaces, and rotating titanium brush is effective in removing non-mineralized deposits from sandblasted surfaces, air-polishing is effective in all the surfaces including machined, plasma-sprayed, and sandblasted [128]. The authors highlighted that except for air-polishing that cleaned all the surfaces, a different cleaning method should be selected for each type of surface.

Wei et al. [129] evaluated the effect of the air pressure of air-polishing devices on the cleaning efficacy for debridement of implant surfaces. The authors inserted implants in polyurethane with simulated peri-implant defects. Afterward, the implants' surfaces were stained with red ink to simulate biofilm. Sodium bicarbonates, calcium carbonates (76μm particle size), and glycine powders $(25 \mu m)$ particle size) were applied with four levels of air pressure: 25, 35, 45, and 55 pounds per square inch (psi). After cleaning, the implants were removed, and pictures were obtained with macro lenses. Their results showed that air polishing with sodium bicarbonate, calcium carbonate, or glycine powders at 35 psi,45 psi, and 55 psi removed simulated biofilm. The authors concluded that 25 psi with glycine powders should be used to eliminate biofilm and reduce surface damage [129].

There is only a single systematic review of the effects of decontamination methods on zirconia surfaces. Tan et al. [130] searched for in vitro, animal studies, and randomized controlled trials that evaluated decontamination methods on zirconia discs or implants. The authors included eleven studies that fulfilled the selected inclusion criteria. Their results showed that lasers (Er:YAG, $CO₂$, and Diodes), curettes (steel, plastic, and titanium), ultrasonic and air scalers with different tips (steel, carbon composite, and copper alloy tip), air-polishing, and prophylaxis rubber cups had been used on zirconia. The authors concluded that air-polishing and prophylaxis rubber cups were safe for zirconia surface decontamination; meanwhile, hand scalers and ultrasonic scalers damage the zirconia surface. In addition, air polishing was highly efficient in removing plaque and biofilm deposited on zirconia surfaces [130].

Summary of Mechanical, Chemical, Lasers, and Other New Techniques Used for Abutment Decontamination

Although laser methods were not included in the previous sections, different studies have shown lasers' application to decontaminate implant surfaces. In a recent systematic review, Francis et al. [131] evaluated comparative studies on titanium surface decontamination. The authors searched for studies reporting decontamination methods applied to implant and abutment titanium surfaces. Inclusion criteria were: in vitro studies evaluating decontamination methods (mechanical, chemical, or laser) on titanium implants, disks, or cylinders with any type of surface. After search and filtering, 38 studies were included. Their data summary showed that air polishing was more efficient for biofilm and plaque removal than laser and chemical methods. However, plaque and biofilm were eliminated when different modalities were combined.

New methods for microbial biofilm removal from implant surfaces were evaluated in a systematic review by Dhaliwal et al. [132]. The authors searched the literature for experimental studies that evaluated the presence of biofilm (including bacteria and fungi) on titanium surfaces and decontamination methods applied for their removal. After the initial search and screening, 116 articles were included.

The new methods to eliminate biofilm were: photodynamic therapy, ultraviolet light (UVL) radiation, sonication, thermal treatment, and ultrasounds. The treatments were applied to reduce existing biofilm or to prevent biofilm formation on surfaces. The mechanism of action is briefly explained as follows:

• Photodynamic therapy used methylene blue applied to the surfaces and activated with 405nm LED or 635nm diode laser to induce cell membrane lysis. It can be applied during maintenance

- UVL therapy uses UV radiation applied prior to the material insertion to promote the elimination of carbon and alter the surface energy to reduce bacterial cell attachment.
- Sonication produces bacterial detachment from titanium surfaces through agitation of a liquid that creates microbubbles, which detach bacteria and solids from the surface through hydrodynamic mechanisms. It can be applied prior to the insertion of the material or during maintenance.
- Thermal treatment is applied before the material insertion, using temperatures below 350 °C to reduce bacteria present at the surface and reduce adhesion by forming titania complexes.
- Ultrasounds produce low-level disinfection through bacteria dispersion and return to the planktonic state. It can be applied prior to insertion or during maintenance [132].

In Figures 7.8–7.11, the mechanical, chemical, laser, and new methods used for abutment decontamination, cleaning, and debridement are summarized [120–132].

Figure 7.8 Mechanical methods and instruments for the maintenance of abutment surfaces. Four main groups of methods are described in the literature [120–132].

Figure 7.9 Chemical methods for disinfection and decontamination of abutment surfaces [120–132].

Figure 7.10 Lasers used for disinfection and decontamination of abutment surfaces [120–132].

Figure 7.11 New methods used for biofilm removal or prevention on abutment surfaces [120–132].

Maintenance Protocols for Implant Abutments

It is well known that pathogenic microorganisms within the plaque microcosmos can produce inflammatory changes around dental implants that can induce peri-implant diseases and potential implant failure [133]. Also, it has been demonstrated that at-home or in-office maintenance can reduce the incidence of implant failures [134, 135].

The professional maintenance of patients, implants, and their restorations is essential because this allows for identifying risk factors, establishing customized maintenance programs and patient education, and intervention if required [136]. Generally, a routine maintenance visit would include the following assessments: gentle probing (once a year), bleeding on probing (BOP), evaluation of the presence of suppuration, debridement, and decontamination of implants, abutments, and restorations [137–139].

A national survey was completed by Zellmer et al. (2020) [140]. The survey with 34 questions was sent to a random sample of 10,000 dental hygienists affiliated with the American Dental Hygienists Association (ADHA). Twenty-one percent responded the survey (2100 participants), and 98% provided care to patients with dental implants.

The results of this study are significant because they reflect in detail the current state of implant maintenance in clinical practice. The distribution of instrumentation used by hygienists during maintenance visits was: 60% plastic scalers, 42% titanium scalers, 31% rubber cups, 12% graphite scalers, and 4.8% of hygienists air-polishing.

The hygienists agreed that plastic curettes were not efficient for debridement and that air- polishing was highly efficient. Also, the hygienists agreed that the recall visits should be established based on individual patient needs, and six-month recalls were recommended. In addition, recommendations for instruments for patient's self-care were the following: oral irrigators, monofilament dental floss, interproximal brushes, silicone or rubber picks, and specially designed intrasulcular instruments [140].

Lian et al. (2021) [141] performed a literature review on interproximal cleaning methods. The authors searched the literature for meta-analysis, systematic reviews, clinical trials, and case reports of studies completed on human subjects. Twenty-six articles were included.

The authors found that dental floss is a generalized method for interproximal cleaning. However, its efficiency is reduced in big embrasure spaces, lack of patient dexterity, and tight spaces. Therefore, the authors recommend customizing the interproximal cleaning device for each patient's requirement.

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Afterward, the authors created a decision tree based on the size of the embrasure, patient motivation, manual dexterity, and pattern of biofilm accumulation. The embrasure spaces were classified as type I, II, and III.

Type I (closed interproximal space filled with the papilla)

- Type II (open interproximal space filled with 51–75% of papilla)
- Type III (large embrasure opening filled with 50% of papilla) [141].

For type I embrasures and motivated patients with good dexterity, dental floss + brushing is recommended. For type I embrasures, not-motivated patients, or lacking dexterity, rubber bristle + soft picks or easy flosser or floss pick and oral irrigation are recommended.

For type II and III, open embrasure, good patient motivation, and good dexterity, interproximal brushes (medium to large sizes) are recommended, plus adjuncts, including wood sticks, dental floss, and gum stimulators [141]. For the lingual interproximal surfaces, in addition to brushing, the use of dental floss with the "C-shaping" technique demonstrated better plaque removal than wood sticks [141].

Dental implant maintenance was also evaluated among dental hygienists in Israel (I) and Germany (GE) by Eickholz et al. [142] Online questionaries were developed at Tel-Aviv and Frankfurt Universities Departments of Periodontology. The questionnaires included: gender, education level if peri-implant probing was completed and type of instruments used for probing, if the hygienists completed peri-implant instrumentation, and the type of instruments used for that purpose. Their results showed that 99% (I) and 98% (GE) of hygienists were women, and 99% were working in their own countries (I and GE), 99% (I) and 92% (GE) of hygienists instrumented, and hand and ultrasonic instruments were used at different percentages in each country 77% (I), and 2% (GE). Finally, airflow was used in different percentages in both countries, 99% (GE) and 18% (I), and lasers were used only in Germany. The authors concluded that the methods of decontamination and debridement variate among countries [142].

Clinical Examples of Implant Abutments and Recommended Decontamination Methods

Given that the techniques for implant abutment maintenance and decontamination are strongly influenced by the type of implant restoration, and to give the readers a better understanding, this section divides implant–abutment maintenance into two main groups: single implant abutments and interconnected abutments. The first group of single implant abutments encompasses maintenance methods for single implant–supported crowns and stud

abutments. The second group, interconnected abutments, encompasses the maintenance methods for implant–supported bridges, bars, and frameworks.

Maintenance for Single Implant Abutments

Single Implant–supported Crown

A single implant–supported crown can be cemented, screw-retained, or friction-fitted. In screw-retained crowns, the abutment and the crown are a single piece and therefore, the abutment is incorporated in the body of the crown at the basal area (Figure 7.12).

However, in cement-retained implant crowns, the transmucosal portion of the implant abutment can be differentiated from the implant restoration (Figure 7.13).

Figure 7.12 Screw-retained porcelain fused to metal single implant crowns. The abutment and the crown are a single piece. The transmucosal portion is short and difficult to differentiate (yellow arrows).

Figure 7.13 Cement-retained single implant zirconia crowns over titanium nitride-coated abutments. The abutment and the crown are two individual pieces. The transmucosal portion of the abutment is longer and variable depending on the implant depth and soft tissue thickness (yellow arrows pointing the white dotted lines that indicate the transmucosal area of the prosthetic abutments).

Figure 7.14 Single implant crown at site #8. Wide open embrasure space at the mesial, and closed embrasure space at distal.

The methods for cleaning and maintenance include brushing + interproximal floss (in closed embrasures) OR brushing + interproximal brushes (in open embrasures). Monofilament floss is recommended over multifilament floss. If the patient has movement limitations, the use of rotatory brushes + flosser OR rotatory brushes + oral irrigation is recommended (Figure 7.14).

Stud Abutments Supporting Overdentures (Including Locator, Ball, and Conus Abutments)

Single-stud abutments are used to support overdentures or implant-assisted retained partial dentures. Their selection depends on the interocclusal space, degree of retention, and simplicity. Although this type of abutment is relatively easy to reach by different cleaning methods, there are areas with restricted access (lingual sides, occlusal, and areas below the retentive ring) (Figures 7.15 and 7.16).

Maintenance for Interconnected Implant Abutments

Implant Bridges

Implant–supported bridges can be screwed or cementretained. Two or more implant abutments can be interconnected with pontics or connectors with different designs in two-unit bridges, three-unit bridges, four-unit bridges, and so on until full mouth rehabilitations (Figures 7.17–7.23).

Given that screw-retained bridges can occasionally be removed, the transmucosal abutments can be reached for a more exhaustive decontamination, and the same protocol for single abutments can be used (Figure 7.24).

Figure 7.15 Four Locator abutments inserted in the maxilla of an edentulous patient.

Figure 7.16 Locator abutments at the mandible. Although the whole 360° circumference of the abutments is exposed, the occlusal cavity (yellow arrow), and the area below the occlusal ring (white arrow) are difficult to reach.

Implant–supported Bar Restorations

Implant bars with multiple designs are commonly used to support overdentures as well as screw-retained hybrid restorations. Bars can be casted, milled, or 3D-printed. The

Figure 7.17 Example of three-unit zirconia bridges, and single crowns to be cemented on custom titanium nitride-coated abutments.

Figure 7.20 Occlusal view of a three-unit implant–supported porcelain fused to metal implant bridge at sites #5, #6, and #7. And a two-unit cantilevered porcelain fused to metal implant bridge at sites #10 (pontic), and #11 (abutment).

Figure 7.18 Examples of four-unit porcelain fused to metal screw-retained implant bridge to be supported by three implants.

Figure 7.21 Buccal view of both bridges. The three-unit bridge at the right side has added pink ceramics.

Figure 7.19 Example of a 10-unit porcelain fused to metal screw-retained upper implant bridge to be supported by six implants. This is a photo showing the lingual side of the bridge positioned over mirror to appreciate the abutment connection.

implant abutment can be incorporated into the base of the bar and then the bar is supported by the implant head. In addition, bars can be supported by transmucosal abutments (like multi-unit abutments). Some bars possess enough space for hygiene and others are too close to the soft tissues resulting in insufficient space for cleaning (Figures 7.25 and 7.26).

The buccal aspect of bars is usually accessible to oral hygiene methods but, the basal aspect as well as the lingual aspect of the bars are extremely difficult to reach for adequate cleaning by at-home conventional methods. Thus, bars must be removed and inspected to detect plaque and calculus and to allow a comprehensive decontamination, and polishing (Figure 7.27).

Although intraoral methods can be applied to debride and maintain implant–supported bars. The recommended action is to remove the bar and proceed with extraoral

Figure 7.22 Right and left side close-views of the a three-unit implant–supported porcelain fused to metal implant bridge at sites #5, #6, and #7, and the two-units cantilevered porcelain fused to metal implant bridge at sites #10 (pontic), and #11 (abutment).

Recommended cleaning protocol intraoral maintenance of a three-unit screw-retained implant–supported bridge with pink ceramics

Figure 7.23 Clinical photo of an upper screw-retained implant bridge with pink porcelain to provide esthetics. The cleanability is compromised by the buccal pink ceramics.

Recommended cleaning protocol intraoral maintenance of a 10-unit screw-retained implant–supported bridge

Recommended cleaning protocol intraoral maintenance of a two-unit screw-retained implant bridge with a cantilevered crown

cleaning and decontamination, additional steps including polishing are recommended (Figure 7.28).

When the bar is supported by transmucosal abutments, once the bar is removed, the transmucosal abutments can be cleaned following the same protocol recommended for multi-unit abutments.

Evaluation of Deffects of the Abutment/ Crown Interface

The previous sections assumed that the implant abutments and their restorations were fabricated in ideal conditions. However, manufacturing methods, including casting, the firing of ceramics, milling, printing, bonding of titanium bases with zirconia restorations, and finishing/polishing, may create undesired surface defects on the transmucosal interface of the prosthetic abutments. In this final section, different manufacturing defects detected in works completed by conventional dental laboratories are grouped and described.

Excess of Ceramic on the Abutment Surface

Ceramic veneering applied to the abutment surface toward the gingival tissues must end at the finish line of the abutment. Which will reduce the presence of micro-retentive

Figure 7.24 Clinical photo of transmucosal multi-unit abutments with angle correction once a screw-retained prosthesis is removed. The photo at the left is showing a buccal view of the multi-unit abutments, The photo at the right is showing an occlusal view of the achieved parallelism with the angle correction multi-unit abutments.

Recommended cleaning protocol intraoral maintenance of multi-unit type transmucosal abutments

Figure 7.25 Upper bar design with enough space for access and cleaning of the transmucosal portion and the basal zone. Different oral hygiene methods can be used.

Figure 7.26 Lower bar design without enough space for access and cleaning. Methods for intraoral hygiene are limited.

areas for plaque accumulation, improve the efficiency of cleaning methods, and improve the soft tissue's contact with the abutment surface.

Excess ceramic can be observed in UCLA abutments when ceramics are masking the buccal gingival margin of the implant abutment or when finishing and polishing are deficient.

Although more frequent at the buccal side of the abutment, this excess can also be seen around the interproximal surfaces and less frequently at the lingual surface (Figures 7.29–7.34).

Porosity

Improper ceramic layering technique, excess of temperature, improper ceramic powder/liquid ratios can produce increased porosity. Porosity can also be seen at the cement that connect ceramic crowns with titanium bases. The pore dimensions are variable and will result in increased

plaque accumulation, and improper cleaning (Figures 7.32 and 7.33).

Inadequate Finishing

Improper finishing and polishing results in thicker margins, partial/incomplete removal of ceramic excess, metal debris, scratches, and incomplete removal of cement excess at the crown/abutment interface (Figures 7.34 to 7.37).

Figure 7.27 Bar that was removed after one year of use for evaluation of plaque retention. Significant calculus and plaque deposits were observed along the whole basal surface of the bar in contact with the soft tissues.

Recommended cleaning protocol for intraoral maintenance of implant–supported bars

Excess of Micro-gap Dimensions

Restorations fabricated by cementation of the crown on a titanium base are usually completed extraoral. Proper cementation technique, adequate vertical pressure, limiting the amount of cement, and venting could minimize the gap at the titanium base/crown interface (Figures 7.38 and 7.39).

Alterations of the Abutment Surface

Coatings can be accidentally removed during finishing and polishing, creating delamination, scratches, and other surface irregularities (Figures 7.40 and 7.41).

Figure 7.28 Basal area of a bar retired for maintenance. Aspect after cleaning, disinfection, and polishing.

Recommended cleaning protocol for extraoral maintenance of implant–supported bars

- 1. Removal of the bar
- 2. Inspection of the basal, and lingual aspects
- 3. Sonication for 20 minutes in disinfecting solution. This step will remove soft plaque and calculus
- 4. Surface polishing with silicone tips
- 5. Re-insertion of the bar and potential replacement of prosthetic screws

Figure 7.29 Example of ceramic layering excess covering the transmucosal area of the implant abutment. (a) Illustrates the gingival extension on the buccal surface almost reaching the base of the abutment. (b) Illustrates an interproximal view (mesial) with overextended and irregular ceramic layering. (c) The lingual surface is not covered by ceramic but, the finishing of the abutment transmucosal metal collar is deficient.

Figure 7.30 In this screw-retained implant crown, the emergence profile is concave, and ceramic layering excess can be observed below the margin of the transmucosal abutment at the level of the abutment connection. This type of excess will critically affect the integrity of the implant–abutment interface increasing the dimension of the micro-gap between the implant and the abutment.

Figure 7.31 Appropriate finishing and polishing under proper magnification can help in the removal of excess ceramics without affecting the integrity of the abutment connection.

Concluding Remarks

Prosthetic abutments and their transmucosal portion are essential for maintaining the peri-implant soft tissue health and the long-term prognosis of dental implants.

Prosthetic implant abutments can be fabricated in different materials (metals, ceramics, polymers, and hybrid compositions). The selection of the abutment material is based on esthetic and mechanical requirements. The surfaces of the abutments can be modified to improve the soft tissue attachment and reduce bacterial adhesion. Micro-grooved surfaces and collagen-coated surfaces enhance soft tissue attachment. Biofilms attach to all hard surfaces, but

Figure 7.32 Close view of the transmucosal portion of an abutment with ceramic debris. The debris invade the basal area of the abutment and the abutment connection. Unfortunately, proper finishing and removal are complex because the instruments used for that purpose can alter the integrity of the implant/abutment interface

titanium nitride, zirconia nitride, silver nanoparticles, and doxycycline coatings can reduce the biofilms and plaque formation.

The implant prosthetic abutment transmucosal area supports the buccal gingival margin and interproximal papillae. Concave transmucosal designs maintain the tissue contours in the presence of thick tissues, and convex transmucosal designs are preferred to support thin gingival tissues. Convergent designs allow soft tissue level maintenance. Narrow emergence angles, concave, and convergent emergence profile designs at the transmucosal

Figure 7.33 Pink ceramic layering on the buccal side of frameworks must be carefully applied to avoid invading the transmucosal portion or the base of the cylinder. If the implant location is supragingival this cannot be achieved.

Figure 7.35 Ceramic layer porosity. Usually seen overlapping the abutment margin and extending to the gingival area.

Figure 7.34 Close view of excess pink ceramic layer. Irregular finishing will impede proper cleaning, and the overextension to the base of the metal cylinder will impede passive fit and increase the micro-gap dimension between the abutment and the framework.

portion of abutments prevent the apical migration of soft tissues and reduce crestal bone loss. Maintenance of the transmucosal portion of implant abutments is critical to maintaining the integrity of the soft tissue seal.

Different methods are used for the cleaning and debridement of implant abutments and restorations. However, the ideal methods should efficiently remove biofilm and plaque without altering the surface of the abutments. The best mechanical method to accomplish both goals is air polishing with glycine powders. Other mechanical methods (scaling and ultrasonic instruments) can partially remove plaque and calculus and reduce biofilm, but all produce surface alterations.

Chemicals, including chlorhexidine, sodium hypochlorite, and citric acid are helpful against biofilm formation and the establishment of young plaque but are not efficient against old plaque and calculus. New decontamination methods, including phototherapy with low-intensity lasers, radiation, and thermal treatment, seem promising. However, to accomplish adequate implant abutment maintenance and achieve better outcomes, the combination of methods is preferred, such as mechanical + chemical methods or chemical + lasers. In addition, the recalls frequency and maintenance protocol must be adapted to the patient and type of restoration.

Finally, the abutments and their restorations must be carefully inspected before insertion to detect fabrication defects, including overextended ceramic layers, porosity, poor finishing and unpolished surfaces, excessive microgap at the abutment-crown interface, and any alterations of

Figure 7.36 Closer view of the characteristics of the porous ceramic. Variable pore sizes, irregular surface finishing, and hidden location will impair both soft tissue attachment and cleanability.

Figure 7.37 Improper finishing and polishing. Metallic debris can be observed at the buccal and lingual aspects of this single implant crown. (a) Buccal view showing metal excess and debris from improper casting, these debris should have been removed before ceramic layering. (b) Lingual view showing partial elimination of metal debris.

Figure 7.38 Micro-gap between zirconia crown and implant abutment. (a) Lingual view showing the extension of the micro-gap around the whole lingual surface. (b) Detail picture illustrating the micro-gap formed at the interface crown-cement-abutment.

Figure 7.39 The micro-gap is too big for accepted standards. In this case 377.89μm.

Figure 7.40 Premolar implant crown fabricated over a laser micro-grooved abutment. The transmucosal emergence angle is divergent.

Figure 7.41 At higher magnification can be observed that the titanium nitride coating is partially removed also, scratches and delamination are observed at the micro-grooved area.

the abutment surface. If detected, excess ceramic and surface debris can be corrected by finishing and polishing. The restoration should be repeated if the abutment crown micro-gap is beyond the clinically acceptable range. This inspection is the most crucial step related to prosthetic abutment hygiene and peri-implant tissue maintenance.

References

- **1** (2017). The glossary of prosthodontic terms. Ninth edition. *The Journal of Prosthetic Dentistry* 117: e6.
- **2** Giglio, G.D. (1999). Abutment selection in implantsupported fixed prosthodontics. *International Journal of Periodontics and Restorative Dentistry* 19: 233–241.
- **3** Glossary Committee from the International Congress of Oral Implantologists (2017). The glossary of implant dentistry III. *International Congress of Oral Implantologists* 106.
- **4** Derks, J., Ichioka, Y., Dionigi, C. et al. (2022). Prevention and management of peri-implant mucositis and periimplantitis: a systematic review of outcome measures used in clinical studies in the last 10 years. *Clinical Oral Implants Research*<https://doi.org/10.1111/clr.13925>. Online ahead of print.
- **5** Walton, J. and MacEntee, M. (1994). Problems with prostheses on implants: a retrospective study. *The Journal of Prosthetic Dentistry* 71: 283–288.
- **6** O'Mahony, A., MacNeill, S., and Cobb, C. (2000). Design features that may influence bacterial plaque retention: a retrospective analysis of failed implants. *Quintessence International* 31: 249–256.
- **7** Teughels, W., Van Assche, N., Sliepen, I., and Quirynen, M. (2006). Effect of material characteristics and/or surface topography on biofilm development. *Clinical Oral Implants Research* 17 (Suppl 2): 68–81.
- **8** Zaugg, L., Astasov-Frauenhoffer, M., Braissant, O. et al. (2017). Determinants of biofilm formation and cleanability of titanium surfaces. *Clinical Oral Implants Research* 28: 469–475.
- **9** Majzoub, J., Chen, Z., Saleh, I. et al. (2021). Influence of restorative design on the progression of peri-implant bone loss: a retrospective study. *Journal of Periodontology* 92: 536–546.
- **10** Chanthasan, S., Mattheos, N., Pisarnturakit, P.P. et al. (2022). Influence of interproximal peri-implant tissue and prosthesis contours on food impaction, tissue health and patients' quality of life. *Clinical Oral Implants Research* 33: 768–781.
- **11** Engel, A., Kranz, H., Schneider, M. et al. (2020). Biofilm formation on different dental restorative materials in the oral cavity. *BMC Oral Health* 20: 162. [https://doi.](https://doi.org/10.1186/s12903-020-01147-x) [org/10.1186/s12903-020-01147-x](https://doi.org/10.1186/s12903-020-01147-x).

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- **12** de Freitas, M., Pontes, K., Fontenelle, I.S.O. et al. (2022). Clinical study of the biofilm of implant-supported complete dentures in healthy patients. *Gerodontology* 39: 148–160.
- **13** Dab, S.S. (2022). Iatrogenic peri-implantitis-factors interplaying the implant suprastructure design. *Clinicial Advances in Periodontics* <https://doi.org/10.1002/cap.10199>.
- **14** Göthberg, C., Gröndahl, K., Omar, O. et al. (2018). Bone and soft tissue outcomes, riskfactors, and complications of implant-supported prostheses: 5-Years RCT with different abtment types and loading protocols. *Clinical Implant Dentistry and Related Research* 20: 313–321.
- **15** Block, M.S. (2022). Evidence-based criteria for an ideal abutment implant connection-a narrative review. *Journal of Oral and Maxillofacial Surgery* S0278-2391 (22): 00506–00507.<https://doi.org/10.1016/j.joms.2022.07.002>. Online ahead of print.
- **16** Ceruso, F.M., Barnaba, P., Mazzoleni, S. et al. (2017). Implant-abutment connections on single crowns: a systematic review. *Oral & Implantology* 10: 349–353.
- **17** Kim, J.C., Lee, M., and Yeo, I.L. (2022). Three interfaces of the dental implant system and their clinical effects on hard and soft tissues. *Materials Horizons* 9: 1387–1411.
- **18** Abdallah, M.N., Badran, Z., Ciobanu, O. et al. (2017). Strategies for optimizing the soft tissue seal around osseointegrated implants. *Advanced Healthcare Materials* 6: 1700549.<https://doi.org/10.1002/adhm.201700549>.
- **19** Rues, S., Fugina, M., Rammelsberg, P., and Kappel, S. (2017). Cemented single crown retention on dental implants: an in vitro study. *The International Journal of Prosthodontics* 30: 133–135.
- **20** Rödiger, M., Kloß, J., Gersdorff, N. et al. (2018). Removal forces of adhesively and self-adhesively luted implantsupported zirconia copings depend on abutment geometry. *Journal of the Mechanical Behavior of Biomedical Materials* 87: 119–123.
- **21** Rathod, A., Jalaluddin, M., Shrinivas, D.T. et al. (2021). Geometry of implant abutment surface improving cement effectiveness: an in vitro study. *Journal of Pharmacy & Bioallied Sciences* 13: S1093–S1097.
- **22** Koutouzis, T., Neiva, R., Nair, M. et al. (2014). Cone beam computed tomographic evaluation of implants with platform-switched Morse taper connection with the

implant-abutment interface at different levels in relation to the alveolar crest. *The International Journal of Oral & Maxillofacial Implants* 29: 1157–1163.

- **23** Levin, B.P., Saito, H., Reynolds, M.A., and Chu, S.J. (2020). Changes in peri-implant soft tissue thickness with bone grafting and dermis allograft. Part II: a comparative retrospective case series using a subcrestal angle correction implant design. *International Journal of Periodontics and Restorative Dentistry* 40: 539–547.
- **24** Kütan, E., Bolukbasi, N., Yildirim-Ondur, E., and Ozdemir, T. (2015). Clinical and radiographic evaluation of marginal bone changes around platform-switching implants placed in crestal or subcrestal positions: a randomized controlled clinical trial. *Clinical Implant Dentistry and Related Research* 17 (Suppl 2): e364–e375.
- **25** Donley, T.G. and Gillette, W.B. (1991). Titanium endosseous implant-soft tissue interface: a literature review. *Journal of Periodontology* 62: 153–160.
- **26** Berglundh, T., Lindhe, J., Ericsson, I. et al. (1991). The soft tissue barrier at implants and teeth. *Clinical Oral Implants Research* 2: 81–90.
- **27** Iglhaut, G., Schwarz, F., Winter, R.R. et al. (2014). Epithelial attachment and downgrowth on dental implant abutments--a comprehensive review. *Journal of Esthetic and Restorative Dentistry* 26: 324–331.
- **28** Sculean, A., Gruber, R., and Bosshardt, D. (2014). Soft tissue wound healing around teeth and dental implants. *Journal of Clinical Periodontology* 41: S6–S22.
- **29** Moon, J., Berglundh, T., Abrahamsson, I. et al. (1999). The barrier between the keratinized mucosa and the dental implant. An experimental study in the dog. *Journal of Clinical Periodontology* 26: 658–663.
- **30** Berglundh, T., Lindhe, J., Jonsson, K., and Ericsson, I. (1994). The topography of the vascular systems in the periodontal and peri-implant tissues in the dog. *Journal of Clinical Periodontology* 21: 189–193.
- **31** Kohal, R.J., Weng, D., Bachle, M., and Strub, J.R. (2004). Loaded custom-made zirconia and titanium implants show similar osseointegration: an animal experiment. *Journal of Periodontology* 75: 1262–1268.
- **32** Vigolo, P., Givani, A., Majzoub, Z., and Cordili, G. (2006). A 4-year prospective study to assess peri-implant hard and soft tissues adjacent to titanium versus gold-alloy abutments in cemented single implant crowns. *Journal of Prosthodontics* 15: 250–256.
- **33** Sennerby, L. and Lekholm, U. (1993). The soft tissue response to titanium abutments retrieved from humans and reimplanted in rats. A light microscopic pilot study. *Clinical Oral Implants Research* 4: 23–27.
- **34** Becker, J., Ferrari, D., Herten, M. et al. (2007). Influence of platform switching on crestal bone changes at non-submerged titanium implants: a

histomorphometrical study in dogs. *Journal of Clinical Periodontology* 34: 1089–1096.

- **35** Farronato, D., Santoro, G., Canullo, L. et al. (2012). Establishment of the epithelial attachment and connective tissue adaptation to implants installed under the concept of "platform switching": a histologic study in minipigs. *Clinical Oral Implants Research* 23: 90–94.
- **36** Atieh, M.A., Ibrahim, H.M., and Atieh, A.H. (2010). Platform switching for marginal bone preservation around dental implants: a systematic review and meta-analysis. *Journal of Periodontology* 81: 1350–1366.
- **37** Schwarz, F., Mihatovic, I., Ferrari, D. et al. (2010). Influence of frequent clinical probing during the healing phase on healthy peri-implant soft tissue formed at different titanium implant surfaces: a histomorphometrical study in dogs. *Journal of Clinical Periodontology* 37: 551–562.
- **38** Abrahamsson, I., Berglundh, T., and Lindhe, J. (1997). The mucosal barrier following abutment dis/ reconnection. An experimental study in dogs. *Journal of Clinical Periodontology* 24: 568–572.
- **39** Iglhaut, G., Becker, K., Golubovic, V. et al. (2013). The impact of dis-/reconnection of laser microgrooved and machined implant abutments on soft- and hard-tissue healing. *Clinical Oral Implants Research* 24: 391–397.
- **40** Nevins, M., Kim, D.M., Jun, S.H. et al. (2010). Histologic evidence of a connective tissue attachment to laser microgrooved abutments: a canine study. *International Journal of Periodontics and Restorative Dentistry* 30: 245–255.
- **41** Salvi, G., Bosshardt, D., Lang, N. et al. (2015). Temporal sequence of hard and soft tissue healing around titanium dental implants. *Periodontology 2000* 68: 135–152.
- **42** Tomasi, C., Tessarolo, F., Caola, I. et al. (2016). Early healing of peri-implant mucosa in man. *Journal of Clinical Periodontology* 43: 816–824.
- **43** Gibbs, S., Roffel, S., Meyer, M., and Gasser, A. (2019). Biology of soft tissue repair: gingival epithelium in wound healing and attachment to the tooth and abutment surface. *European Cells & Materials* 38: 63–78.
- **44** Sailer, I., Philipp, A., Zembic, A. et al. (2009). A systematic review of the performance of ceramic and metal implant abutments supporting fixed implant reconstructions. *Clinical Oral Implants Research* 20 (Suppl 4): 4–31.
- **45** Edelhoff, D., Schweiger, J., Prandtner, O. et al. (2019). Metal-free implant-supported single-tooth restorations. Part I: abutments and cemented crowns. *Quintessence International* 50: 176–184.
- **46** Matos, J., Arcila, L., Ortiz, L. et al. (2022). Hybrid abutment during prosthetic planning and oral rehabilitation. *Minerva Dental and Oral Science* 71: 107–116.
- **47** Taha, D., Cesar, P.F., and Sabet, A. (2022). Influence of different combinations of CAD-CAM crown and customized abutment materials on the force absorption capacity in implant supported restorations – in vitro study. *Dental Materials* 38: e10–e18.
- **48** Rahmitasari, F., Ishida, Y., Kurahashi, K. et al. (2017). PEEK with reinforced materials and modifications for dental implant applications. *Dentistry Journal* 5: 35. <https://doi.org/10.3390/dj5040035>.
- **49** Vazouras, K., Gholami, H., Margvelashvili-Malament, M. et al. (2022). An esthetic evaluation of different abutment materials in the anterior maxilla: a randomized controlled clinical trial using a crossover design. *Journal of Prosthodontics* <https://doi.org/10.1111/jopr.13520>. Online ahead of print.
- **50** Coray, R., Zeltner, M., and Özcan, M. (2016). Fracture strength of implant abutments after fatigue testing: a systematic review and a meta-analysis. *Journal of the Mechanical Behavior of Biomedical Materials* 62: 333–346.
- **51** Al-Thobity, A.M. (2022). Titanium base abutments in implant prosthodontics: a literature review. *European Journal of Dentistry* 16: 49–55.
- **52** Suphangul, S., Rokaya, D., Kanchanasobhana, C. et al. (2022). PEEK biomaterial in long-term provisional implant restorations: a review. *Journal of Functional Biomaterials* 13: 33. <https://doi.org/10.3390/jfb13020033>.
- **53** Ferrari, M., Cagidiaco, M.C., Garcia-Godoy, F. et al. (2015). Effect of different prosthetic abutments on peri-implant soft tissue. A randomized controlled clinical trial. *American Journal of Dentistry* 28: 85–89.
- **54** Linkevicius, T. and Apse, P. (2008). Influence of abutment material on stability of peri-implant tissues: a systematic review. *The International Journal of Oral & Maxillofacial Implants* 23: 449–456.
- **55** de Avila, E.D., de Molon, R.S., Palomari Spolidorio, D.M., and de Assis Mollo, F. Jr. (2013). Implications of surface and bulk properties of abutment implants and their degradation in the health of periodontal tissue. *Materials (Basel)* 6: 5951–5966.
- **56** Elter, C., Heuer, W., Demling, A. et al. (2008). Supra- and subgingival biofilm formation on implant abutments with different surface characteristics. *The International Journal of Oral & Maxillofacial Implants* 23: 327–334.
- **57** Hahnel, S., Wieser, A., Lang, R., and Rosentritt, M. (2015). Biofilm formation on the surface of modern implant abutment materials. *Clinical Oral Implants Research* 26: 1297–1301.
- **58** Desch, A., Freifrau von Maltzahn, N., Stumpp, N. et al. (2020). Biofilm formation on zirconia and titanium over time – an in vivo model study. *Clinical Oral Implants Research* 31: 865–880.
- **59** Zeller, B., Stöckli, S., Zaugg, L.K. et al. (2020). Biofilm formation on metal alloys, zirconia and

polyetherketoneketone as implant materials in vivo. *Clinical Oral Implants Research* 31: 1078–1086.

- **60** Herrmann, H., Kern, J.-S., Kern, T. et al. (2020). Early and mature biofilm on four different dental implant materials: an in vivo human study. *Clinical Oral Implants Research* 31: 1094–1104.
- **61** Del Rey, Y.C., Parize, H., Pedrazzi, V. et al. (2022). Clinical and in situ oral biofilm formation on dental implant abutment materials: a systematic review. *The International Journal of Oral & Maxillofacial Implants* 37: 639–652.
- **62** Abrahamsson, I., Zitzmann, N., Berglundh, T. et al. (2002). The mucosal attachment to titanium implants with different surface characteristics: an experimental study in dogs. *Journal of Clinical Periodontology* 29: 448–455.
- **63** Chien, H., Schroering, R., Prasad, H., and Tatakis, D. (2014). Effects of a new implant abutment design on peri-implant soft tissues. *The Journal of Oral Implantology* 40: 581–588.
- **64** Wennerberg, A., Sennerby, L., Kultje, C., and Lekholm, U. (2003). Some soft tissue characteristics at implant abutments with different surface topography. A study in humans. *Journal of Clinical Periodontology* 30: 88–94.
- **65** Furuhashi, A., Ayukawa, Y., Atsuta, I. et al. (2012). The difference of fibroblast behavior on titanium substrata with different surface characteristics. *Odontology* 100: 199–205.
- **66** Furuhashi, A., Ayukawa, Y., Atsuta, I. et al. (2021). Soft tissue interface with various kinds of implant abutment materials. *Journal of Clinical Medicine* 10: 2386. [https://](https://doi.org/10.3390/jcm10112386) [doi.org/10.3390/jcm10112386.](https://doi.org/10.3390/jcm10112386)
- **67** Ghinassi, B., Di Baldassarre, A., D'Addazio, G. et al. (2020). Gingival response to dental implant: comparison study on the effects of new nanopore laser-treated vs. traditional healing abutments. *International Journal of Molecular Sciences* 21: 6056.
- **68** Atsuta, I., Ayukawa, Y., Furuhashi, A. et al. (2019). Epithelial sealing effectiveness against titanium or zirconia implants surface. *Journal of Biomedical Materials Research. Part A* 107: 1379–1385.
- **69** Brunette, D.M. and Chehroudi, B. (1999). The effects of the surface topography of micromachined titanium substrata on cell behavior in vitro and in vivo. *Journal of Biomechanical Engineering* 121: 49–57.
- **70** Weiner, S., Simon, J., Ehrenberg, D. et al. (2008). The effects of laser microtextured collars upon crestal bone levels of dental implants. *Implant Dentistry* 17: 217–228.
- **71** Shin, S.-Y. and Han, D.-H. (2010). Influence of microgrooved collar design on soft and hard tissue healing of immediate implantation in fresh extraction sites in dogs. *Clinical Oral Implants Research* 21: 804–814.
- **72** Nevins, M., Camelo, M., Nevins, M.L. et al. (2012). Reattachment of connective tissue fibers to a

laser-microgrooved abutment surface. *International Journal of Periodontics and Restorative Dentistry* 32: e131–e134.

- **73** Lee, H., Lee, J., Lee, J. et al. (2015). Microgrooves on titanium surface affect peri-implant cell adhesion and soft tissue sealing; an in vitro and in vivo study. *Journal of Periodontal & Implant Science* 45: 120–126.
- **74** Ketabi, M. and Deporter, D. (2013). The effects of laser microgrooves on hard and soft tissue attachment to implant collar surfaces: a literature review and interpretation. *International Journal of Periodontics and Restorative Dentistry* 33: e145–e152.
- **75** Geurs, N., Geisinger, M., Vassilopoulos, P. et al. (2016). Optimizing connective tissue integration on laser-ablated implant abutments. *Clinical Advances in Periodontics* 6: 153–159.
- **76** Ahamed, A., Prakash, P., Crena, J. et al. (2021). The influence of laser-microgrooved implant and abutment surfaces on mean crestal bone levels and peri-implant soft tissue healing: a 3-year longitudinal randomized controlled clinical trial. *International Journal of Implant Dentistry* 7: 102. [https://doi.org/10.1186/s40729-021-00382-3.](https://doi.org/10.1186/s40729-021-00382-3)
- **77** Corvino, E., Pesce, P., Mura, R. et al. (2020). Influence of modified titanium abutment surface on peri-implant soft tissue behavior: a systematic review of in vitro studies. *The International Journal of Oral & Maxillofacial Implants* 35: 503–519.
- **78** Jennes, M., Naumann, M., Peroz, S. et al. (2021). Antibacterial effects of modified implant abutment surfaces for the prevention of peri-implantitis-a systematic review. *Antibiotics (Basel)* 10: 1350. [https://doi.](https://doi.org/10.3390/antibiotics10111350) [org/10.3390/antibiotics10111350](https://doi.org/10.3390/antibiotics10111350).
- **79** Ricci, J. and Alexander, H. (2016). Cell response to surfaces: a concise summary. *International Journal of Periodontics and Restorative Dentistry* 36: s39–s46.
- **80** Pesce, P., Menini, M., Tealdo, T. et al. (2014). Periimplantitis: a systematic review of recently published papers. *The International Journal of Prosthodontics* 27: 15–25.
- **81** Pesce, P., Canullo, L., Grusovin, M. et al. (2015). Systematic review of some prosthetic risk factors for periimplantitis. *The Journal of Prosthetic Dentistry* 114: 346–350.
- **82** Kheur, S., Singh, N., Bodas, D. et al. (2017). Nanoscale silver depositions inhibit microbial colonization and improve biocompatibility of titanium abutments. *Colloids and Surfaces B: Biointerfaces* 159: 151–158.
- **83** Cardoso, M., Sangalli, J., Koga-Ito, C. et al. (2016). Abutment coating with diamond-like carbon films to reduce implant-abutment bacterial leakage. *Journal of Periodontology* 87: 168–174.
- **84** Fröjd, V., Linderbäck, P., Wennerberg, A. et al. (2011). Effect of nanoporous TiO2 coating and anodized Ca2+ modification of titanium surfaces on early microbial biofilm formation. *BMC Oral Health* 11: 8.
- **85** Odatsu, T., Kuroshima, S., Sato, M. et al. (2020). Antibacterial properties of nano-Ag coating on healing abutment: an in vitro and clinical study. *Antibiotics (Basel)* 9: 347.
- **86** Huacho, P., Nogueira, M., Basso, F. et al. (2017). Analyses of biofilm on implant abutment surfaces coating with diamond-like carbon and biocompatibility. *Brazilian Dental Journal* 28: 317–323.
- **87** Brunello, G., Brun, P., Gardin, C. et al. (2018). Biocompatibility and antibacterial properties of zirconium nitride coating on titanium abutments: an in vitro study. *PLoS One* 13: e0199591.
- **88** Xing, R., Witsø, I., Jugowiec, D. et al. (2015). Antibacterial effect of doxycycline-coated dental abutment surfaces. *Biomedical Materials* 10: 055003.
- **89** Visai, L., Rimondini, L., Giordano, C. et al. (2008). Electrochemical surface modification of titanium for implant abutments can affect oral bacteria contamination. *Journal of Applied Biomaterials & Biomechanics* 6: 170–177.
- **90** Almohandes, A., Abrahamsson, I., Dahlén, G., and Berglundh, T. (2021). Effect of biofilm formation on implant abutments with an antibacterial coating: a pre-clinical in vivo study. *Clinical Oral Implants Research* 32: 756–766.
- **91** Amirtharaj Mosas, K.K., Chandrasekar, A.R., Dasan, A. et al. (2022). Recent advancements in materials and coatings for biomedical implants. *Gels* 8: 323. [https://doi.](https://doi.org/10.3390/gels8050323) [org/10.3390/gels8050323.](https://doi.org/10.3390/gels8050323)
- **92** Kazimierczak, P. and Przekora, A. (2020). Osteoconductive and osteoinductive surface modifications of biomaterials for bone regeneration: a concise review. *Coatings* 10: 971.
- **93** Akhtar, M., Uzair, S.A., Rizwan, M., and Ur Rehman, M.A. (2022). The improvement in surface properties of metallic implant via magnetron sputtering: recent progress and remaining challenges. *Frontiers in Materials* 8: 602.
- **94** López-Valverde, N., López-Valverde, A., and Ramírez, J. (2021). Systematic review of effectiveness of chitosan as a biofunctionalizer of titanium implants. *Biology (Basel)* 10: 102. [https://doi.org/10.3390/](https://doi.org/10.3390/biology10020102) [biology10020102](https://doi.org/10.3390/biology10020102).
- **95** López-Valverde, N., Flores-Fraile, J., Ramírez, J.M. et al. (2020). Bioactive surfaces vs. conventional surfaces in titanium dental implants: a comparative systematic review. *Journal of Clinical Medicine* 9: 2047. [https://doi.](https://doi.org/10.3390/jcm9072047) [org/10.3390/jcm9072047](https://doi.org/10.3390/jcm9072047).
- **96** Davarpanah, M., Martinez, H., Celletti, R., and Tecucianu, J. (2001). Three-stage approach to aesthetic implant restoration: emergence profile concept. *Practical Procedures & Aesthetic Dentistry* 13: 761–767.
- **97** Steigmann, M., Monje, A., Chan, H., and Wang, H. (2014). Emergence profile design based on implant position in the

esthetic zone. *International Journal of Periodontics and Restorative Dentistry* 34: 559–563.

- **98** Chu, S.J., Kan, J.Y., Lee, E.A. et al. (2019). Restorative emergence profile for single-tooth implants in healthy periodontal patients: clinical guidelines and decisionmaking strategies. *International Journal of Periodontics and Restorative Dentistry* 40: 19–29.
- **99** Su, H., Gonzalez-Martin, O., Weisgold, A., and Lee, E. (2010). Considerations of implant abutment and crown contour: critical contour and subcritical contour. *International Journal of Periodontics and Restorative Dentistry* 30: 335–343.
- **100** Romanos, G., Traini, T., Johansson, C., and Piatteli, A. (2010). Biologic width and morphologic characteristics of soft tissues around immediately loaded implants. Studies performed on human autopsy specimens. *Journal of Periodontology* 81: 70–78.
- **101** Mattheos, N., Vergoulis, I., Janda, M., and Miseli, A. (2021). The Implant supracrestal complex and its significance for long term successful clinical outcomes. *The International Journal of Prosthodontics* 34: 88–100.
- **102** Mattheos, N., Janda, M., Acharya, A. et al. (2021). Impact of design elements of the implant supracrestal complex (ISC) on the risk of peri-implant mucositis and peri-implantitis: a critical review. *Clinical Oral Implants Research* 32: s181–s202.
- **103** Gomez-Meda, R., Esquivel, J., and Blatz, M.B. (2021). The esthetic biological contour concept for implant restoration emergence profile design. *Journal of Esthetic and Restorative Dentistry* 33: 173–184.
- **104** Valente, N.A., Wu, M., Toti, P. et al. (2020). Impact of concave/convergent vs parallel/ divergent implant transmucosal profiles on hard and soft peri-implant tissues: a systematic review with meta-analyses. *The International Journal of Prosthodontics* 33: 553–564.
- **105** Cabanes-Gumbau, G., Pascual-Moscardó, A., Peñarrocha-Oltra, D. et al. (2019). Volumetric variation of peri-implant soft tissues in convergent collar implants and crowns using the biologically oriented preparation technique (BOPT). *Medicina Oral, Patología Oral y Cirugía Bucal* 24: e643–e651.
- **106** Dada, K., Pariente, L., and Valois, J. (2021). A new method of conclusively creating and transferring the emergence profile on single tooth immediate implant restorations: the E-merge protocol. *International Journal of Computerized Dentistry* 24: 457–477.
- **107** Parpaiola, A., Toia, M., Norton, M. et al. (2020). CAD/ CAM implant abutments: peri-implant hard and soft tissue response with up to 4 years of follow-up- a retrospective cohort study evaluation. *International*

Journal of Periodontics and Restorative Dentistry 40: 193–201.

- **108** Siegenthaler, M., Strauss, F., Gamper, F. et al. (2022). Anterior implant restorations with a convex emergence profile increase the frequency of recession: 12-month results of a randomized controlled clinical trial. *Journal of Clinical Periodontology* [https://doi.org/10.1111/](https://doi.org/10.1111/jcpe.13696) [jcpe.13696.](https://doi.org/10.1111/jcpe.13696)
- **109** Hamilton, A., Obermaier, B., Doliveux, S. et al. (2022). Digitally fabricated provisional implant restorations prior to implant placement: a clinical case series. *The International Journal of Prosthodontics* 35: 94–108.
- **110** Teślak, M., Ziemlewski, A., Foltyn, I. et al. (2021). Development of custom anatomic healing abutment based on cone-beam computer tomography measurement on human teeth cross-section. *Materials (Basel)* 14: 4570. [https://doi.org/10.3390/ma14164570.](https://doi.org/10.3390/ma14164570)
- **111** Costa, A., Burgoa, S., Rayes, A. et al. (2021). Digital workflow for CAD-CAM custom abutments of immediate implants based on natural emergence profile of the tooth to be extracted. *The Journal of Oral Implantology* [https://doi.org/10.1563/](https://doi.org/10.1563/aaid-joi-D-20-00214) [aaid-joi-D-20-00214.](https://doi.org/10.1563/aaid-joi-D-20-00214)
- **112** Cardoso, R.C., Gerngross, P.J., Dominici, J.T., and Kiat-amnuay, S. (2013). Survey of currently selected dental implants and restorations by prosthodontists. *The International Journal of Oral & Maxillofacial Implants* 28: 1017–1025.
- **113** Schepke, U., Meijer, H.J., Kerdijk, W. et al. (2017). Stock versus CAD/CAM customized zirconia implant abutments – clinical and patient-based outcomes in a randomized controlled clinical trial. *Clinical Implant Dentistry and Related Research* 19: 74–84.
- **114** Romanos, G., Gurbanov, S., Hess, P. et al. (2022). Crestal bone loss and implant failure of prefabricated versus customized abutments: a 10-year retrospective radiological study. *Clinical Oral Investigations* 26: 2879–2886.
- **115** Lin, H., Lin, J., Salamanca, E. et al. (2022). Marginal bone level evaluation of fixed partial dental prostheses using preformed stock versus CAD/CAM customized abutments. *Journal of Personalized Medicine* 12: 1051. <https://doi.org/10.3390/jpm12071051>.
- **116** Souza, A., Alshihri, A., Kämmerer, P. et al. (2018). Histological and micro-CT analysis of peri-implant soft and hard tissue healing on implants with different healing abutments configurations. *Clinical Oral Implants Research* 29: 1007–1015.
- **117** Katafuchi, M., Weinstein, B.F., Leroux, B.G. et al. (2018). Restoration contour is a risk indicator for periimplantitis: a cross-sectional radiographic analysis. *Journal of Clinical Periodontology* 45: 225–232.
- **118** Yi, Y., Koo, K.-T., Schwarz, F. et al. (2020). Association of prosthetic features and peri-implantitis: a crosssectional study. *Journal of Clinical Periodontology* 47: 392–403.
- **119** Wang, J., Tang, Y., Qiu, L., and Yu, H. (2022). Influence of buccal emergence profile designs on peri-implant tissues: a randomized controlled trial. *Clinical Implant Dentistry and Related Research* 24: 329–338.
- **120** Rapley, J., Swan, R., Hallmon, W., and Mills, M. (1990). The surface characteristics produced by various oral hygiene instruments and materials on titanium implant abutments. *The International Journal of Oral & Maxillofacial Implants* 5: 47–52.
- **121** Mengel, R., Buns, C., Mengel, C., and Flores-de-Jacoby, L. (1998). An in vitro study of the treatment of implant surfaces with different instruments. *The International Journal of Oral & Maxillofacial Implants* 13: 91–96.
- **122** Mengel, R., Meer, C., and Flores-de-Jacoby, L. (2004). The treatment of uncoated and titanium nitride-coated abutments with different instruments. *The International Journal of Oral & Maxillofacial Implants* 19: 232–238.
- **123** Gehrke, P., Spanos, E., Fischer, C. et al. (2018). Influence of scaling procedures on the integrity of titanium nitride coated CAD/CAM abutments. *The Journal of Advanced Prosthodontics* 10: 197–204.
- **124** Sirinirund, B., Garaicoa-Pazmino, C., and Wang, H.L. (2019). Effects of mechanical instrumentation with commercially available instruments used in supportive peri-implant therapy: an in vitro study. *The International Journal of Oral & Maxillofacial Implants* 34: 1370–1378.
- **125** Biazussi, B., Perrotti, V., D'Arcangelo, C. et al. (2019). Evaluation of the effect of air polishing with different abrasive powders on the roughness of implant abutment surface: an in vitro study. *The Journal of Oral Implantology* 45: 202–206.
- **126** Tran, C., Khan, A., Meredith, N., and Walsh, L. (2022). Influence of eight debridement techniques on three different titanium surfaces: a laboratory study. *International Journal of Dental Hygiene* 21: 1–13.
- **127** Schmage, P., Kahili, F., Nergiz, I. et al. (2014). Cleaning effectiveness of implant prophylaxis instruments. *The International Journal of Oral & Maxillofacial Implants* 29: 331–337.
- **128** Louropoulou, A., Slot, D.E., and Van der Weijden, F. (2014). The effects of mechanical instruments on contaminated titanium dental implant surfaces: a systematic review. *Clinical Oral Implants Research* 25: 1149–1160.
- **129** Wei, M., Tran, C., Meredith, N., and Walsh, L. (2017). Effectiveness of implant surface debridement using particle beams at differing air pressures. *Cllinical and Experimental Dental Research* 3: 148–153.
- **130** Tan, N., Khan, A., Antunes, E. et al. (2021). The effects of physical decontamination methods on zirconia implant surfaces: a systematic review. *Journal of Periodontal & Implant Science* 51: 298–315.
- **131** Francis, S., Iaculli, F., Perrotti, V. et al. (2022). Titanium surface decontamination: a systematic review of in vitro comparative studies. *The International Journal of Oral & Maxillofacial Implants* 37: 76–84.
- **132** Dhaliwal, J.S., Abd Rahman, N.A., Ming, L.C. et al. (2021). Microbial biofilm decontamination on dental implant surfaces: a mini review. *Frontiers in Cellular and Infection Microbiology* 11: 736186. [https://doi.](https://doi.org/10.3389/fcimb.2021.736186) [org/10.3389/fcimb.2021.736186.](https://doi.org/10.3389/fcimb.2021.736186)
- **133** Dreyer, H., Grischke, J., Tiede, C. et al. (2018). Epidemiology and risk factors of peri-implantitis: a systematic review. *Journal of Periodontal Research* 53: 657–681.
- **134** Monje, A., Aranda, L., Diaz, K.T. et al. (2016). Impact of maintenance therapy for the prevention of peri-implant diseases: a systematic review and meta-analysis. *Journal of Dental Research* 95: 372–379.
- **135** Berglundh, T., Jepsen, S., Standlinger, B. et al. (2019). Periimplantitis and its prevention. *Clinical Oral Implants Research* 30: 150–155.
- **136** American Academy of Periodontology (2003). Position paper: periodontal maintenance. *Journal of Periodontology* 74: 1395–1401.
- **137** Armitage, G. and Xenoudi, P. (2016). Post-treatment supportive care for the natural dentition and dental implants. *Periodontology 2000* 71: 164–184.
- **138** Bidra, A.S., Daubert, D.M., Garcia, L.T. et al. (2016). Clinical practice guidelines for recall and maintenance of patients with tooth-borne and implant-borne dental restorations. *Journal of Dental Hygiene* 90: 60–69.
- **139** Todescan, S., Lavigne, S., and Kelekis-Cholakis, A. (2012). Guidance for the maintenance care of dental implants: clinical review. *Journal of the Canadian Dental Association* 78: c107.
- **140** Zellmer, I.H., Couch, E.T., Berens, L., and Curtis, D.A. (2020). Dental hygienists' knowledge regarding dental implant maintenance care: a national survey. *Journal of Dental Hygiene* 94: 6–15.
- **141** Liang, P., Ye, S., McComas, M. et al. (2021). Evidencebased strategies for interdental cleaning: a practical decision tree and review of the literature. *Quintessence International* 52: 84–95.
- **142** Eickholz, P., Winkler, P., Elez, I. et al. (2022). Common practices of dental implants maintenance among dental hygiene professionals in Israel and Germany. *International Journal of Dental Hygiene* [https://doi.](https://doi.org/10.1111/idh.12612) [org/10.1111/idh.12612](https://doi.org/10.1111/idh.12612).

8

Considerations of Implant–abutment Connections for the Longevity of Dental Implants

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Introduction

There are two types of implant–abutment configurations: one-piece and two-piece. One-piece design lacks interfaces as the implant body and abutment are continuous [1]. On the contrary, two-piece configurations have an interface (implant abutment interface [IAC]) [1]. The IAC is defined as "the interlocking interfacial geometry between a dental implant and an abutment" and "the surface where the dental implant and prosthetic abutment connect". These two components (implant and abutment) can be fastened via an abutment screw or by a friction system [2].

Mechanically, the resistance of the abutment against rotation is granted by an index system provided by different geometric configurations of the implant and abutment walls. This reduces rotational freedom to less than two degrees [3]; and resists dislodgement and micromovement at the IAC $[4, 5]$. In addition, tightening the abutment screw until preload is achieved stabilizes and retains both parts together thereby minimizing the risk of screw loosening [6, 7].

Biologically, appropriate sealing at the IAC interface reduces the migration of microorganisms inside the implant connection [8], and its apical-coronal location determines the insertion levels for the stability of soft and hard peri-implant tissues [9]. Clinically, the IAC also dictates the maintenance methods to support the longevity of osseointegrated implants [10].

The most coronal portion of the implant where the prosthetic abutment, restorations, and other prosthetic components connect with the implant is called the implant platform [11]. When correlated with the osseous crest, the implant platform serves as a reference to determine if an implant is subcrestal (apical to the crestal bone), crestal (at

the same level with the crestal bone), or supracrestal located (coronal to the crestal bone). The edges of the implant platform coincide with the abutment edges in butt-joint connections. However, it is not coincident in platform-switching/ shifting connections in which, edges of the implant abutment are mesial to the edges of the implant platform [11]. Significant geometrical differences exist in the different available IACs that may impact the frequency of mechanical and biological complications [12].

This chapter has three aims: first, to classify the different IACs designs; second, to compare their sealing capability against microorganisms; and third, to describe strategies for the maintenance of IACs.

Types of IAC

Characterization of IAC is based on the following features: (i) method of retention of the abutment to the implant, (ii) vertical positioning of the IAC in relation to the bone level and in relation to the implant platform, (iii) horizontal position of the IAC in relation to the implant platform edges, and (iv) geometric designs of the IAC (Figure 8.1).

Abutment Retention to the Implant Body

One-piece Implants

One-piece dental implants have been available for over five decades, and early examples were: blade implants, ceramic implants (synthetic sapphire and aluminum oxide), and titanium implants. The main characteristic of these designs is that the abutment is incorporated together within the same implant body [13]. Given that one-piece implants are exposed to the oral cavity immediately after insertion, a

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Figure 8.1 Classification of implant–abutment connections (IACs). Five groups are presented based on the method of retention, vertical position of the IAC in relation to the crestal bone, vertical position of the IAC in relation to the implant platform, horizontal position of the IAC in relation to the platform edges, and their designs. Multiple combinations and possibilities of these four groups are found in current dental implant systems.

second surgery is not required, and therefore they are also called "one-stage" implants [14].

The configuration of one-piece implants (including the implant body and the abutment in a single piece) reduces the surgical trauma by eliminating a second surgery and simplifies the clinical procedure by eliminating the requirement for additional prosthetic components. In addition, the microgap is absent, which may reduce the bacteria counts [15]. Other advantages attributed to one-piece implants are reduced crestal bone loss and inflammatory reactions, and biologic width dimensions similar to natural teeth [16]. Furthermore, surgical and clinical procedures for one-piece dental implants results in less swelling, pain, time, and fewer appointments than procedures for twopiece dental implants [17–19].

One-piece dental implants are exposed to the oral cavity before they are osseointegrated; thus, they require primary stability (<30Ncm) sufficient to withstand force vectors and to reduce micromovement-related complications. Consequently, their use is recommended in areas with good bone quality and quantity [20].

When strict inclusion criteria are followed, one-piece implants with immediate load have shown stable marginal bone and soft tissue levels and a high cumulative survival rate (98.8%) after two years in function [21]. The inclusion criteria used in the study by Finne et al. [21] described the following pre-requisites for immediate loading on onepiece implants: healthy subjects, good oral hygiene, stable occlusal relationships, no history of bruxism, sufficient bone volume, implants with at least 10mm length, insertion torque between 35–45Ncm, implant bed free of infection, and in the case of immediate implants, no extraction remnants. The authors also described the exclusion criteria for the use of one-piece implants: lack of patient compliance, chronic bone diseases, bone tumors, previous irradiation, implant angles exceeding 10–15 degrees, and cantilevered prostheses [21].

Baer et al. [22] reported the results of a three-years multicenter study using the same one-piece implant system. In their protocol, implants were inserted and immediately temporized, and the final restorations were delivered after six months. One-piece implants showed a cumulative survival rate of 98.3%, with minimal resorption of crestal bone, and preservation of the soft tissue levels [22].

Current one-piece implants are also available in materials other than titanium, exemplified by one-piece zirconia dental implants. Silva et al. [23] evaluated fatigue and failure modes of zirconia implants with a 30-degree angle related to the vertical axis. The abutment portion of the implants was prepared to simulate the preparation for an anterior crown. The characteristics of the preparation included: a 2mm occlusal reduction, a six-degree wall angle, and a 0.5mm chamfer reduction along the entire circumference. Forces over 600Ncm were applied. The main failure mode for preparing an unprepared implant was a fracture, which occurred in the areas of exposed treads below the prepared abutment. Thus, demonstrating that a minimal preparation of the abutment portion of one-piece zirconia implants did not increase the fracture incidence [23].

When the abutment portion of one-piece implants is modified, the temperature can increase significantly at the level of the first threads. This was demonstrated in the study by Gabay et al. [24] which investigated the effects of the preparation of the coronal portion of one-piece dental implants with and without irrigation on the temperature at the first thread of the implants. Their results showed temperatures over 47 °C reached between 5 and 11 seconds after the beginning of the abutment preparation in the group without irrigation. Furthermore, the temperature continued increasing after the drill stopped. Meanwhile, in the group with irrigation, the temperature elevation did not reach 47 °C. Thus, the authors demonstrated that the preparation of the abutment portion of a one-piece dental increases the temperature and is essential to provide enough volume of irrigation to prevent fast temperature elevation at the level of the first thread [24].

In case of space limitations when narrow implants are recommended, it seems that the mechanical strength of one-piece implants is superior to that of two-piece implants; however, proper implant positioning is critical because the abutment orientation cannot be changed, and structural modifications of the abutment portion can result in a lack of retention of the implant crown or phase transformation in the case of ceramic implants [25].

It seems that the diameter of the one-piece implant can influence the stability of the crestal bone [26]. Forty-four narrow Implants (3mm diameter) were inserted in healed ridges and in post-extraction areas, and all the implants were immediately loaded with temporary crowns. Survival and radiographic bone levels were evaluated for one year. The survival rate after one year was 98%. The radiographic analysis of the bone le levels showed that 18% of implants presented <3mm of bone loss, 20% of implants presented between 2 and 3mm of bone loss, and 62% of implants presented >2mm of bone loss after one year. Based on the excessive bone loss after one year, the authors recommended the cautious use of 3mm diameter one-piece implants [26]. There are also some limitations in using one-piece implants, in case of implant malposition, the abutment requires modifications which results in titanium particle release, and the exposure to non-axial loads can result in implant micromovement, lower success rates, and higher crestal bone loss [27–29].

Two-piece Implants

Two-piece implants are versatile and possess a different number of applications [30]. For example, in case of implant misalignment (17-degrees up to 25-degrees), custom or angled abutments can be used, if the implants lack primary stability, they can be submerged. In such cases, they require additional surgery after the bone healing to expose the implant platform and connect a healing abutment, also called "two-stage" implants [31].

Two-piece titanium implants (100%) have survival rates comparable to those of one-piece implants (100%) after five years [32, 33]; however, it seems that they present more technical complications (screw loosening and porcelain fractures) [32, 33].

The transmucosal portion of the prosthetic abutments of two-piece dental implants can be customized to accommodate soft tissue thickness and biologic width [34], the abutment angle can be modified to correct up to 25-degree implant misalignment [35-37]. The implant can be submerged in augmented areas, lack of stability, or if a removable denture is being inserted and risk of excessive uncontrolled forces is present [38–40].

Some differences between two-piece and one-piece dental implants include: the gingival margin being more coronal in

one-piece implants and the biologic width dimensions being more similar to natural teeth in one-piece implants [16]. Furthermore, the presence of an interface in two-piece dental implants and microgap, allows potential micromovement, bacteria colonization, and screw loosening [41].

The prosthetic abutment can be retained in two-piece dental implants by an abutment screw or a friction system (screw-less). In screw-retained abutments, the retention screw is tightened to produce an elastic deformation (flattening of the microrough edges of the screw threads and elongation of the screw) achieving enough vertical compressive force between the implant and the abutment (preload) to reduce screw-loosening [42, 43]. It has been demonstrated that 90% of the tightening force is spent as frictional force and 10% of the force is converted to preload [44].

In implant abutments retained by friction systems, a morse taper connection produces high tension between the abutment and the implant and a horizontal cold-welding effect. Taper angles around 1.5° and 4.0° degrees ensure an excellent coupling between the mating surfaces of the abutment and the implant connection [45, 46]. The frictionretained abutment systems seem to function comparably to screw-retained abutment systems [47].

Vertical Implant Position of the IAC in Relation to the Bone Level

The implant platform can be placed at three alternative vertical positions at the moment of implant insertion: supracrestal, crestal, or subcrestal. In supracrestal positions, the implant platform is elevated over the crestal bone; therefore, the IAC is also located over the bone crest. In crestal positions, the implant platform edges, and the IAC are leveled with the adjacent bone, and in subcrestal positions, the implant platform and the IAC are below the crestal bone level [2].

The consequences of different implant insertion depths are variable. For example, it was found that the placement of the IAC at or below the alveolar crest did not influence the peri-implant crestal bone levels after five years in human studies [48]. However, posterior studies that used implants with platform-switching (abutment diameter narrower than implant platform diameter) demonstrated that subcrestal placement presented less crestal bone loss than platform-switched implants placed at the crestal level [49]. Increased number of neutrophils and moderate bone resorption occur when the IAC is located at the level of the bone crest and If the IAC is superior to the crestal bone (in platform-switching or one-piece implants), minimal inflammatory infiltrates and lower bone resorption can be expected [1].

Contradictory results were presented in recent systematic reviews and meta-analyses that evaluated the bone loss of implants placed at subcrestal or crestal level [50, 51]. In the study by Palacios-Garzón et al. [50] sixteen studies were included. Ten studies did not show differences in bone loss between crestal or subcrestal placement, three studies found more crestal bone loss in subcrestal placement, and the other three found more bone loss in crestal placement.

In the most recent systematic review and metaanalysis by Cruz et al. [51] the marginal bone loss, implant survival, and soft tissue parameters around implants placed crestal or subcrestal were evaluated. The authors included ten studies that fulfilled the inclusion criteria. The meta-analysis showed that implant depths did not influence implant survival or other periimplant parameters (marginal bone loss, probing depths, or keratinized tissue).

Rationale for Crestal or Subcrestal Implant Placement

Implant placement is followed by a remodeling process that results in a marginal bone loss between 0.25 and 0.65mm during the first year [52, 53]. In both cases (crestal and subcrestal placement), the remodeling is comparable, but in subcrestal placement lower risk of exposed threads can be expected [54]. This occurs because re-establishing the biological width around implants requires a minimum distance of 2mm between the implant platform and the gingival surface. Therefore, by adapting the implant insertion level to the surface of the soft tissues bone remodeling can be controlled [55, 56].

In the case of soft tissues with thin phenotypes, the establishment of the biologic width can occur by causing bone resorption [57]; meanwhile, in the case of thick gingival phenotypes, the marginal bone resorption is reduced [58, 59]. Finally, the marginal bone level is affected by the implant design + implant insertion depth combinations [53]. For example, tissue-level implants (with an incorporated transmucosal portion) preserve the bone if inserted at crestal level; meanwhile, bone-level implants preserve the bone when placed subcrestally [53].

All the previous can be applied to implants inserted in the healed bone. However, when implants are inserted in post-extraction sockets, more bone remodeling is expected, specially at the buccal bone area, where resorption up to 2.2mm has been reported after tooth extraction [60, 61]. Therefore, in post-extraction sockets, the bone resorption can be compensated by completing a deeper implant insertion from 2.5 to 3.0mm below the crestal bone [62]. The insertion depth range in the case of post-extraction implants with platform-switching is smaller than implants without platform-switching, and 1.08mm of subcrestal insertion is enough in platform-switched implants to avoid exposure of the implant neck [63].

Vertical Position of the IAC in Relation to the Implant Platform

Two main configurations for IACs have been described in two-piece dental implants: external or internal. In external IACs, the connection occurs over the body of the implant at the implant platform, and the center of rotation of the abutment is high resulting in abutment instability. Meanwhile, in internal IACs, the connection occurs within the implant and below the implant platform, and the center of rotation of the abutment is low, resulting in higher abutment stability [64].

The external connection was intended to serve for the insertion of the implant and to stabilize the implant supra structure [65]. However, the height of the geometrical feature of external connections (mainly hexagonal) was inefficient in reducing micromovement against non-axial loads of elevated magnitude. Thus, conducting to screw loosening, and fatigue and fracture of prosthetic screws [66].

Internal connections were developed to limit the drawbacks of external connections (screw loosening, micromovement, and fracture). Internal connections possess different depths, geometries, and mating properties (between the internal walls of the implant and the abutment connection) that seem to improve the transfer of forces and reduce abutment micromovement and screw loosening [64, 67].

Other claimed benefits of internal connections over external connections have been reported in in vitro and in vivo studies, including better stress distribution along the implant body when non-axial loads are applied, better maintenance of the screw preload, lower Interleukin 1 (IL-1) and tumoral necrosis factor- α (TNF α) around the implants [68], and lower screw loosening and mechanical complications observed in clinical studies with at least three years of follow-up [69].

Finally, it has been demonstrated that wider microgaps appear in external connections when lateral forces are applied, favoring bacterial microleakage [70], and better esthetic outcomes are observed around implants with internal connections placed in the anterior region compared to those with external connections [71].

Horizontal Position of the IAC in Relation to the Platform Edges

Two horizontal positions of the IAC in relation to the implant platform edges have been described: butt-joint and platform-switched positions. The main difference is that the implant and the abutment match in butt-joint positions and not in platform-switched positions [72]. In butt-joint IACs, the edges of the abutment and the implant platform are coincident without overlapping. Therefore, the diameter of the platform and the basal diameter of the

prosthetic abutment are the same. Meanwhile, in platformswitched, the basal diameter of the implant abutment is narrower than the implant platform [73].

In platform-switching, the inward displacement of the abutment walls toward the central axis of the implant creates a step on the implant platform [74], (a horizontal space) that allows for the horizontal establishment of the biologic width and bone ingrowth [75]. Once the abutment contour is moved away from the edges of the implant platform, the implant–abutment microgap and the associated inflammatory infiltrate are also distanced from the marginal bone thus, reducing bone resorption [76].

Given that implant diameters vary in range from narrow to wide implants, a platform-switched abutment may possess different levels of mismatch. Canullo et al. [77] studied the effects of an abutment with 3.8mm diameter inserted on implants with different diameters (3.8, 4.3, 4.8, and 5.5mm). Thus, total mismatches of 0, 0.25, 0.5, and 0.85mm were evaluated. The authors observed that after three years of follow-up, larger mismatch resulted in lower marginal bone resorption [77]. This was confirmed by histological studies completed in minipigs with platform-switching of 0.25mm [78], and in radiographic studies with platformswitching up to 0.85mm (in which implants with platforms with 5.8mm diameter received 4.1mm abutments) that demonstrated decreased marginal bone resorption compared with non-platform-switched abutments [79, 80].

Another aspect related to the marginal bone preservation observed in platform-switching is the soft tissue thickness. When sufficient apical-coronal and buccal soft tissue thickness are present, the marginal bone is preserved in both platform-switched and non-platform-switched implants. However, when the tissue thickness is reduced, platform-switching is efficient in preserving the marginal bone level [81].

Vandeweghe et al. [81] completed a randomized controlled trial in patients who received implants with wide platforms (7mm) and connections centered in the implant axis or with an offset (two platform-switching sizes 0.225 or 1.225mm). After 3, 6, and 12 months radiographs were obtained to evaluate the marginal bone levels at the mesial and distal sides. Their results showed that the marginal bone loss decreased by 30% in platform-switched implants when the soft tissue thickness was sufficient [81].

The biomechanics of implants with platform-switched abutments have also been considered responsible for reduced bone loss [82–84]. When oblique forces are applied on platform-switched abutments, the functional stresses are re-directed to the center of the implant, thus transferring the strains to the cancellous bone, and protecting the cortical bone [82–84].

Moreover, under function and simulated accelerated corrosion, implants with platform-switching released fewer

metal ions than implants with butt joint connections. Implants with butt joint connection presented active corrosion at the outer borders of the contacting surfaces. Meanwhile, platform-switched connections released minimal amounts of titanium ions which may explain the reduced bone loss observed around platform-switched IACs [85, 86].

A recent systematic review and meta-analysis by Mishra et al. [87] evaluated randomized controlled trials with at least three years of follow-up comparing platform matching with platform-switching. The results showed that platform-switching limited the marginal bone loss better when the mismatch between the implant and the abutment was greater, the maxilla reacted better than the mandible to platform-switching, and better bone preservation with platform-switched implants with splinted prostheses inserted at bone level [87].

Geometric Design of the IAC Index

Implant manufacturers are working on improving the IAC index designs to reduce abutment rotation, improve load distribution, reduce bacteria filtration, and minimize screw loosening to limit mechanical and biological complications [88]. Common IAC index designs are based on angles, channels, morse-taper (conical), and combinations of different geometries [89]. Each IAC index design possesses certain variability. For example, the angled index connections can be hexagonal, octagonal, and dodecagonal (this means 6, 8, or 12 angles); channel index type connections can have 3, 4, 6, 7, or 8 channels (with straight or round contours) with variable dimensions, and morse tapered connections are available with 4-degree to 11-degree taper angles and different depths and can have or not an index.

Further available IACs include a combination of geometries like morse taper $+$ hexagon, or conical $+$ hexagon, and unique geometries like spline type, and triangle conical. The following subsections review the characteristics of each group.

Angled Indexed Connections

Angled connections are available with 6, 8, or 12 angles, and all possess different rationales that support their use. In the following subsection, details about each type of connection are provided.

Connections with Six Angles (Hexagonal)

The oldest of the angled IACs is the hexagonal connection initially described by Branemark in 1969 [90]. This design simplified the implant insertion, provided anti-rotational support for the restoration on single implants [70], and allowed for one- or two-stages implant surgery [70, 88-90].

Figure 8.2 (a) Internal hexagon. The dark outline is the contour of the implant. The silver ring is the implant platform. (b) Illustrates in red color the hexagonal indexed area, and in green, each one of the vertex of the hexagon.

The hexagonal connection can be internal or external, and some implant systems simultaneously possess internal and external hexagons [91]. Hexagonal connections present some limitations; for example, the short height of the external hexagon under non-axial forces can allow micro movement and frequent screw loosening in single implant restorations, and the prosthetic screw is the only component retaining the butment [64, 92]. Also, when non-axial loads are applied on external hexagonal connections, the dimensions of the microgap increase, conducting in increased counts of microorganisms inside the IAC [93]. In addition, as the microgap increases the range of micromotion also increases. This, in turn, increases stresses that are transferred to the vertices of the hexagon initiating a cycle of wear, more micromotion, and potential fractures [94] (Figures 8.2 and 8.3).

Connections with Eight Angles (Octagonal)

Octagonal connections were designed to improve the alsocalled "morse-taper" connection. The morse-taper connection consists of a cone – fitting within – a cone, and the precise matting between both conical surfaces achieves the stability of this connection [95]. The taper angle variates between 4 and 11%, and total convergence angles can be up to 16 degrees [96]. However, there were some difficulties in transferring positional information between the clinic and the laboratory because the cones are symmetric and do not possess an anti-rotational element [97]. Including an index (hexagon, octagon, and others) below the cone, provided

reproducibility between the dental laboratory and the clinical procedures and reduced micromotion [94, 98].

Setbacks of conical connections with octagonal index are that a vertical sliding movement occurs during the insertion of the abutment to achieve friction fit. Some octagonal connections do not have a stop element, and a settlement phenomenon (the abutment is slightly intruded into the implant) can occur when high vertical occlusal loads are applied, resulting in changes in the occlusal height of the restoration [99].

Another potential issue of conical implant–abutment conical + octagonal index is that after simulated repeated loading (vertical load of 250 N for 100,000 cycles) representing the stresses received for one month, the abutment removal torque decreases from 24 ± 0.43 to 17.66 ± 0.59 Ncm, and the vertical displacement can reach up between 5.0 ± 1.8 and 8.8 ± 1.10 µm [100] (Figure 8.4).

Connections with 12 Angles (Dodecagonal)

Connections with 12 angles were designed to increase the restorative options by adding more positional possibilities to the prosthetic abutment. The implants with a dodecagonal index also possess a hexagon that engages the implant driver. Thus, the dodecagonal index is protected from deformation in high insertion torques [101, 102]. The dodecagonal index enables the surgeon to place the flat surfaces of the implant connection in any position during the surgery. The prosthetic abutment can be oriented in 12 different positions. This allows increased rotational flexibility [101].

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Figure 8.3 Internal hexagon with internal bevel. The picture is showing an implant system that possesses a platform beveled internally in a slight tapered angle. The external silver-light ring is the implant platform, the dark ring is the superior part of the bevel, the third silver-ring is the lower part of the bevel, the hexagonal index area is highlighted in red color. The vertex of the angles is indicated with blue numbers. Digital microscope image at 50X.

Figure 8.4 Internal octagon. This system presents a platform narrower than the implant diameter. The external dark ring is the implant body, the internal silver ring is the platform, and inner to the platform is the octagonal index area highlighted in red color. The vertex of the angles is indicated with green numbers. Digital microscope image at 50X.

In mechanical studies with finite element analysis, it was determined that oblique forces applied to three different IAC designs (external hexagon, internal dodecagon, and internal conical tapered hexagon with 45° angle), were more stable distributed and less displacement observed in dodecagonal internal connections [102] (Figure 8.5).

Channel Indexed Connections

The channeled connections are also called grooved indexed connections, which are present in tube-in-tube and conical connections. The main characteristic is that in the tube-intube connection, the channel (or groove) is present at the coronal level of the main tube; meanwhile, in conical

Figure 8.5 Implant with dodecagonal index. This figure illustrates an implant with a flat implant platform in yellow color. Internally, in red color a hexagonal area is highlighted. The center, a dodecagonal index area is indicted in blue color. The angles of the hexagon are indicated in red, and the angles of the dodecagon in black color. Digital microscope image at 50X.

connections, the tapered wall ends in a short cylinder that contains the channels (thus, the channels are more apical) [103–108].

Channeled connections can have three, four, six, or eight symmetric channels (i.e. trilobe®, conelog®, cross fit®), which allow for three, four, six, or eight abutment positions. In addition, there are also channeled connections with asymmetric channels (indexing dots) that allow only one abutment position [103–108].

The channels can be rounded or squared form (like boxes) and have different widths and depth sizes. The channeled index design possesses certain advantages, including good reproducibility of implant/abutment position, excellent rotational stability, and good distribution of loads [103–105].

One disadvantage of the channel in some tube-intube connections is that their location at the coronal area and the channel size can produce a thinning of the implant wall that can result in fractures in narrow implants [94, 95]. However, in wider implants, the tubein-tube channeled connection can increase the fracture strength and the stability of the restoration, and the apical location of the channels in conical connections results in thicker implant walls and a lower risk of fracture [94, 106, 107]. Overall, the presence of channels or grooves significantly reduces the vertical, horizontal, and rotational movement of the prosthetic abutment [108] (Figures 8.6–8.10).

Morse Tapered Connections

Morse tapered connections are also called conical connections. The morse taper design possesses conical walls with internal angles of different degrees. For example, implants with morse tapered internal walls with 8- to 11-degrees taper angles, showed increased resistance to screw loosening, and increased mechanical strength [109–111]. Conical connections with a 4-degrees tapering angle increase the frictional contacts between the implant and the abutment, providing enhanced anti-rotation and improved sealing against fluids and bacteria leakage. This precise fit is more efficient in transferring horizontal forces from the abutment to the implant and reduces the stresses received by the abutment screw [112, 113].

The fracture resistance of abutments with morse tapered connection (conical) compared with internal hexagonal connection was tested under oblique compressive forces. Morse tapered connections with 3.5 connection depth, and 11.5-taper angle showed higher deformation torque values $(90 \pm 6.72 \text{ Kgf})$ than internal hexagonal connections $(83.73 \pm 4.94 \,\text{Kgf})$ [114].

The performance of morse tapered designs was evaluated in a systematic review that compiled in vitro and in vivo studies. In vitro studies demonstrated that morse tapered conical connections presents a superior bacterial seal, increased resistance to abutment displacement, minimal microgap, higher resistance to torque loss and fatigue, and less transmission of stresses to the abutment screw. In

Figure 8.6 Implant with six channels index. This figure illustrates an implant with flat platform, internally, a small cone that ends in six symmetric boxes separated by the channels indicated with green numbers. In red color, the area between boxes that contain the channels is highlighted. Digital microscope image at 50X.

Figure 8.7 Implant with seven channels index. This figure illustrates an implant with flat platform, internally, a cone that ends in seven asymmetric boxes separated by the seven channels indicated with green numbers. In red color, the area between boxes that contain the channels is highlighted. Digital microscope image at 50X.

vivo studies showed reduced crestal bone loss, success, and survival comparable to other connection systems [115]. In addition, the conical abutment that engages in the morse tapered connections presents a smaller diameter than the implant platform, which provides a platform-switched configuration. Therefore, morse tapered designs allow increased space for establishing the biologic width and increased connective tissue thickness surrounding the abutment [116].

Morse taper conical abutments were subject to cyclic axial loads in the presence of biofilm, and the removal torque values were evaluated against the baseline. The results showed that the removal torque increased significantly after repeated load from 27.2 ± 7.5 Ncm to maximum values of

Figure 8.8 Implant with an index with eight rounded channels. This implant shows an outer dark gray ring (implant body), a silver ring (a flat implant platform), and eight symmetric rounded channels in red. Each round channel is indicated with green numbers. Digital microscope image at 50X.

Figure 8.9 Zirconia implant with a four channels index. These photos are showing a zirconia implant with a flat implant platform. Internally in red color, four channels are highlighted in red color. Digital microscope image at 50X.

 160.6 ± 15.8 Ncm after 100,000 cycles and up to 154 ± 14 Ncm after 500,000 cycles [117]. This confirms the increased removal and the active settlement under load experienced by morse taper connections without a stop design.

Finally, a recent prospective study with follow-up from one to nine years evaluated morse taper connections with

and without positional index. The study showed that indexed abutments suffered more fractures than nonindexed abutments in molar positions. In addition, the survival of non-indexed morse cone abutments was higher (99%) than morse cone indexed abutments (95%) [118] (Figures 8.11–8.13).

Figure 8.10 Implant with a tube- in tube connection with three channels. (a) The inner walls of a tube-in-tube connection are almost parallel however, this image example shows 1-degree taper angle to facilitate the insertion of the abutment. (b) Coronal view of a 3.5mm diameter implant ant the three rounded channels. (c) 3D-reconstruction from the characteristic inner cavity of the implant with three channels. Digital microscope image at 50X.

Figure 8.11 Implant with morse cone tapered connection. This implant system shows the platform (flat and wide) in gray color. Inside, a blue area indicates the internal cone. In yellow a six channels index, and in red is indicated the basal space for the prosthetic screw. Microscope image at 50X.

Figure 8.12 Section of the IAC of an implant with morse cone tapered connection. The cone is located at the most coronal portion of the IAC. The index area is inferior to the cone, and in red the basal area is the space for the apical portion of the prosthetic screw. Obtained by vertical scanning with the digital microscope and lenses of 100X.

Figure 8.13 Implant with morse tapered connection. The dark color is the implant body beveled internally toward a silver ring (implant platform). In yellow, the morse tapered area, and inside in red, a hexagonal index. The numbers in green color are indicating the angles of the hexagon. Microscope image at 50X.

IAC Design and Sealing Capability Against Bacteria Colonization

Microorganisms can penetrate and colonize the implant– abutment interface demonstrated by the presence of colonies of microorganisms at different areas inside the implant connection, in screw-retained and in cementretained restorations [119, 120, 121] Given that the dimension of the microgap differs between IAC designs, the percentage of bacteria that can penetrate inside the implant connection variates. For example, higher counts of microorganisms are measured for external connections

compared to internal connections. Furthermore, within internal connection designs, the conical connections have shown less bacterial counts compared to non-conical connections [122, 123]. The morse-taper connection is the most resistant to bacteria penetration although, smaller molecules (endotoxins) can penetrate all types of connections including morse-taper connections [124, 125]. The microbial contamination of the IAC can occur by multiple causes (handling, lack of fit between components, design of the IAC, and translocation of microorganisms). In this section, the potential mechanisms that can produce microbial contamination inside the IAC, as well as the effect of the IAC design in the bacteria colonization are summarized.

Mechanisms for Microbial Contamination of the IAC

Microbial contamination of the IAC can be induced by implant insertion [126–132], before the restoration is delivered, and during function [129]. During the fabrication of the implant restoration, microbial contamination of the abutments can occur, and contaminated abutments can transfer this contamination to the IAC during the insertion of the implant restorations [130].

Penarrocha et al. [127] evaluated the microbial colonization produced by the insertion of dental implants after five years post-loading. Two types of implants were compared pre-mounted and mount-less dental implants. The difference between both systems is that pre-mounted implants come with an insert that is released after the implant insertion, and mount-less implants come without an insert and are transferred to the implant bed using universal implant drivers.

After five years, samples were obtained from the implant connection, and higher counts of bacteria were observed in the implant inserted with the universal drivers compared to implants inserted with pre-mounts. Given that the implant driver transfers rotational forces to the implant index, and the fit of the pre-mounted insert surpasses the fit of the universal implant driver, the authors explained that the deformation of the connection during insertion induced by the driver was higher than the deformation of the connection induced by the pre-mount and recommend the use of pre-mounted dental implants and reduce the insertion torques [127].

In addition, De barros Lucena et al. [130] obtained samples of the implant connection at the second stage of surgery. Careful isolation of the implant area was completed and after the removal of the cover screws, paper points were used to obtain samples of the implant's intaglio. The samples were evaluated by checkerboard DNA hybridization. Their results detected 22 different species of bacteria in 62.5% of the subjects, and a higher percentage of bacteria was found at the mandible (77.42%) compared to the maxilla (36.17%) [130] (Figure 8.14).

After exposure to the oral environment, healing abutments will suffer from surface contamination by saliva, food debris, microbial and epithelial cells, and nicotine (in smokers) [132, 133]. The removal of all contaminants from healing abutments by conventional cleaning, or their complete sterilization has not been achieved because traces of proteins and amino acids are still detectable in microbiological evaluations of healing abutments after different cleaning and sterilization regimes [134].

Cover screws removed during the second stage surgery also showed contamination, including tissue debris, bacteria, and corrosion products not observed in new cover screws [135]. The histological analysis of the soft tissues located over cover screws also showed chronic inflammatory infiltrate. This could mean that although clinically the mucosa appears healed, a communication tract persisted between the cover screw and the oral environment, or the cover screws were contaminated during insertion [136]. Therefore, healing abutments and cover screws can be other sources of contamination of the IAC (Figure 8.15).

Once microorganisms colonize the inner aspect of the implant, the IAC becomes the perfect reservoir where bacteria are protected, larger colonies can grow and where a transition to a microflora that is mainly anaerobic and more pathogenic, is common [137]. The dynamics of fluid flow within implant connections still need to be understood. Factors that include saliva viscosity, composition and volume of saliva, and geometrical constraints can affect the microorganism's attachment and accumulation at the implant connection [138] (Figure 8.16).

Also, fluid flow can affect the transport and physiology of the microbial residents by inducing phenotypic changes [139]. In small spaces, fluid tension produces an accumulation of motile bacteria close to the sidewalls and decreases bacterial concentration in the central regions of channeled spaces [140–143]. Taking into consideration the microgap and other physical spaces within the IAC, the following areas can be colonized by microorganisms or endotoxins: all the inner walls of the IAC, basal space of the abutment screw, screw threads, internal threads of the implant connection, and implant platform [144, 145] (Figure 8.17).

Furthermore, the bacterial counts are influenced by the IAC design. For example, D'Ercole et al. [147] found that morse cone connections presented 20% of microbial contamination, compared to external (45%) and internal (55%) hexagonal connections. In their study, implants with three
Figure 8.14 Microbial contamination of the IAC can occur early during implant surgery when the implants are inserted for (example using the same implant driver to insert multiple implants), or when saliva or blood flows into the IAC.

Loss of healing abutment or cover screws After removal of healing abutment or cover screws

Figure 8.15 Microbial contamination of the IAC can occur during prosthetic procedures. Microorganisms can enter the IAC when during the connection and disconnection of prosthetic parts. Also, the premature loss of healing abutments and cover screws as well as the reuse of parts are causative of microbial contamination.

Figure 8.16 Microbial contamination of the IAC can occur during function. Multiple factors are involved but, here the design and the screw-loosening are the sources of a cascade of events that influence the number of microbial counts within contaminated IACs.

Figure 8.17 Common locations for contamination deposits at the IAC. 1. Contamination includes acids, enzymes, bacteria, endotoxins, and corrosion subproducts. Six areas are described in this figure. 1. The basal area of the implant connection is illustrated in green color, 2. The implant connection threads are illustrated in gray color, 3. Walls of the implant connection illustrated in red color, 4. The abutment screw is illustrated in dark gray, 5. The abutment channel is illustrated in blue color, 6. The union between the platform and the abutment walls is illustrated in yellow color. The locations vary in different implant designs, and all can show different levels of contamination. Modified from Liu and Wang [146].

IAC designs were inoculated with *S. oralis* and *P. aeuroginosa*. Afterward abutments were connected with their recommended torque values, and the abutments were sealed with polyetheretherketone -PEEK) caps and immersed in a culture media solution. The penetration of the bacteria in the solution was evaluated by the turbidity of the samples. There are differences regarding microbial diversity and microorganism counts in oral biofilm associated with titanium or zirconia [148] and morse taper connections were more efficient in reducing bacteria filtration [149].

This bacteria leakage is variable because the microgap (space between the implant connection and the abutment) differs between IAC designs. The microgap varies in size from 0.1 (morse taper connections) to $10 \mu m$ (external hexagonal connections) before load and can increase under loading conditions [150]. The repeated micromovement of the abutment can produce a bi-directional and constant fluid flow from the peri-implant sulcus to the implant connection; thus, bacteria and their products, as well as beneficial molecules for microbial growth, are always present and perpetuating the infection [151, 152].

Another parameter related to bacteria contamination is screw loosening [153–156]. Once the preload of the abutment screw is lost, micromovement increases, microgap is enlarged, and the fluid flow increases which can result in increased counts of bacteria and yeast at the implant cavities [155].

Finally, the type of restoration (cemented versus screwretained) seems to influence the microbial contamination of the IAC. In a cross-sectional study, Peñarrocha-Oltra et al. [154] compared the microbial colonization of implants restored with cemented versus screw-retained restorations. Samples were obtained from the implant sulcus, adjacent teeth, and IACs. Their results showed that both implant groups (with cemented and screw-retained restorations) were contaminated. But differed in that implants with cement-retained restorations possessed lower bacteria counts inside the connection and more bacteria counts at the peri-implant sulcus. Meanwhile, implants with screw-retained restorations presented fewer bacteria counts at the peri-implant sulcus and more bacteria counts at the implant connection [154].

Comparison of Microbial Contamination in Different IAC Designs

Multiple clinical, animal, and in vitro studies evaluated the influence of the IAC design on bacterial microleakage. However, comparisons are not feasible because of differences in the experimental methods, time of sampling, presence of load or not, and only some of the IAC designs have been compared against each other.

For this section, a focused review of the literature was completed from January to September 2022. Only in vitro studies were included to reduce the variability of human and animal studies. The search included the terms "bacterial leakage" AND "implant connection" OR "bacterial microleakage" AND "implant–abutment connection" OR "microbial contamination" AND "implant–abutment connection". The following terms were also included: "internal connection" AND "contamination" OR "external connection" AND "contamination" OR "hexagon" AND "contamination" OR "octagon" AND "contamination" OR "dodecagon" AND "contamination" OR "morse taper" AND "contamination" OR "conical" AND "contamination" OR "cone" AND "contamination" OR "tube-in-tube" AND "contamination."

Inclusion criteria were in vitro studies evaluating the implant connection or implant–abutment connection and its microbial contamination, with or without restoration, with or without load, comparison of two- or more- types of IAC designs, the connection type is identified, time of sampling, presence of microorganisms (bacteria and yeast), and endotoxin counts. Exclusion criteria were human and animal studies, studies that evaluated only a type of IAC design, studies that evaluated leakage but did not include bacteria, and in general, studies that did not fulfill the inclusion criteria.

The collected information was summarized and registered in tables including authors, year, type of connection, presence, load, and quantification of bacteria, yeast, or endotoxins, results, and conclusions.

The initial results of the search returned 524 articles. The titles and abstracts were reviewed, and 350 articles were eliminated because these were not in vitro studies [164]. The full texts of the remaining 174 articles were read in full to determine the fulfillment of inclusion criteria. Then, 153 articles were removed because were repeated, did not compare at least two connections, did not properly clear the type of implant connection, and evaluated leaking but not microbial contamination. From the 21 remaining articles, 16 articles were excluded because they did not compare different IAC designs. Finally, only 5 articles that fulfilled the inclusion criteria were included.

The following subgroup comparisons were found external and internal hexagons versus morse taper and tube-intube versus flat-to-flat connections.

External and Internal Hexagon Versus Morse Taper

D'Ercole et al. [147] in an in vitro study, evaluated bacteria microleakage from the IAC of implants inserted into a culture solution. Three designs were compared: external hexagon, internal hexagon, and morse taper. In total, sixty implants (20 per group) were used.

Ten sterile implants of each group were inoculated with 1.0μl solution containing *S. oralis* and other ten sterile implants were inoculated with 1.0μl solution containing *P. aeuroginosa*. Afterward, sterile prosthetic abutments were connected to their respective implants with 35Ncm torque, and sterile polyetheretherketone (PEEK) caps were inserted into the most coronal portion of the abutments. Finally, the coupled implant/abutment/PEEK caps were inserted into individual containers filled with culture media.

The culture media was evaluated every four days by visual inspection (turbidity of the solution indicated bacterial contamination) and by taking a sample of 1.0μ of the solution. The collected samples were evaluated by Gram staining and the morphology of the colonies cultured in agar plates for *S. oralis* and *P. aeuroginosa*. The last samples were retrieved after 14 days.

The results of the study showed that after 14 days, P. aeuroginosa leaked from all the IAC designs to the cultural media. Meanwhile, *S. oralis* leaked from the internal and external hexagonal connections but not from the morse taper connection. The leaking from external and internal was higher than from the morse cone [147].

D'Ercole et al. [157] compared the microbial leakage in morse taper versus internal hexagon. Twenty implants (ten from each group) were inoculated with a solution containing 0.1μl of *P. aeuroginosa* (0.5–1.0μm wide and 1.5–5μm long) or *Aggregatibacter actinomycetemcomitans* (AA) $(0.4-1.0 \,\mu m)$ long). The respective abutments were connected with 35Ncm torque. The coupled implants/ abutments were inserted in Eppendorf tubes leaving the

IAC level below the solution containing culture media. After 28 days, the bacterial leakage was evaluated by inspection for the presence of turbidity in the culture solution. In addition, 1μl of the solution was analyzed by Gram staining and colony morphology in agar.

The results showed that 80% of the samples leaked and contaminated the culture solution in the internal hexagon connection meanwhile, 50% of the samples leaked to the culture solution in the Morse cone connection. The authors concluded that both connections suffer bacterial contamination, and the differences in microgap between both connections can explain the differences in leakage [157].

Guerra et al. [143] evaluated the bacterial contamination of five abutment designs. External hexagon and internal hexagon implants were compared with implants with morse cone connections. The implants with the morse cone received three different abutments morse cone alone, morse cone with a hexagonal index, and short morse cone. 150 implants (30 implants per group) received their respective abutments. The abutment screws were inoculated with 0.5μl solution containing *Escherichia coli* (*E. coli*) (0.5–1.0μm size) and *Streptococcus sanguinis* (*S. sanguinis*) $(1.1-1.5 \mu m)$ diameter and 2–6 μ m length) and then, inserted into the implants and torqued with 20Ncm.

Afterward, the implant/abutment couples were suspended in culture media broth. The abutments were not sealed. After 14 days, the solutions were inspected for bacterial contamination by evaluating the turbidity of the sample, number of colonies, and Gram staining. The results of the study, contrary to other studies, showed no contamination from implants with an external connection. Meanwhile, the morse cone implants showed contamination of the culture media (16.47%). The authors attributed the lack of bacteria filtration from external hex connections to the lack of loading and the filtration of bacteria from differences in the machining of parts in the morse cone connections. The authors concluded that bacterial infiltration is low without differences between morse cone connections [143].

Larrucea et al. [158] compared the microbial leakage of four morse cone designs (three with hexagonal index and one squared index) against three straight internal connection designs (two with hexagon and one with three channels). Six samples per group (*N* =42) were subject to load, thermocycling, and bacterial leakage tests. Perpendicular load of 10kg was applied to the abutments for 2000 cycles every 0.5 seconds. Afterward, the samples were subject to thermocycling during 300 cycles of 5 seconds each in cold (5 °C) and hot (50 °C) temperatures. The bacterial leakage after loading was evaluated at two independent chambers, one at the level of the IAC and the second at the level of the abutment inoculated with *Porphyromonas gingivalis* *(P. gingivalis)*. Samples of each chamber were obtained after 15 days.

The results of the study showed that the samples with conical connections did not filtrate *P. gingivalis* to the upper or lower chambers. Meanwhile, the straight internal connections filtrated *P. gingivalis* to the upper and lower chambers. The authors concluded that the precision of the fit of conical connections provided better sealing than internal hex and channeled connections. In addition, in internal connections, the length of the connection determined less bacterial microfiltration [158] (Table 8.1).

Tube-in-tube Versus Flat-to-Flat

Baggi et al. [159] evaluated the resistance to fungi and bacteria contamination of implant systems with different IAC designs and different preload values. In their study, seven different implant brands with tube-in-tube connections were compared with implant brands with flat-to-flat connections. In sterile conditions, the implants were contaminated with 0.1 µl of microbial solution (0.05μ) of bacterial solution $+ 0.05 \mu$ l of yeast suspension). The solution contained the following microorganisms *Candida albicans* (*C. albicans*), *Candida glabrata* (*C. glabrata*), *Streptococcus sanguis* (*S. sanguis*), *Fusobacterium nucleatum* (*F. nucleatum*), and *Actinomyces odontolyticus* (*A. odontolyticus*). Then, the implants and abutments were connected with the torque values recommended by the manufacturers (from 15 to 35 Ncm) or with manual torque $(\pm 5$ Ncm) and immersed in a culture solution for 7 and 14 days. The contamination of the solution was evaluated based on the presence of cloudiness, Gram staining, and colony-forming units. The results showed that implant–abutment pairs connected with the torque recommended by the manufacturer resisted microbial leakage more than implant–abutment pairs connected by hand torque. In addition, tube-in-tube connections presented lower microbial leakage than flat-to-flat connections. The *C. Albicans* and *C. glabrata,* although with a bigger dimension than the bacteria, were able to penetrate through the IACs forming complex biofilms [159] (Table 8.1).

Maintenance of IACs

In the previous subsections, we discussed the differences in microgap between IACs designs, the influence of the IAC design on the microbial leakage from and toward the implant connection, and the potential contamination ways of the implant and abutment connections.

Dental implant therapy does not end with the delivery of the implant restorations; it also must involve maintenance protocols that include, among others: updates on the general **Table 8.1** Microbial leakage comparisons between different IAC designs.

(*Continued*)

Table 8.1 (Continued)

health and oral health, evaluation of the patient's oral healthcare, assessments of the peri-implant tissues (soft and hard), and supra- and sub-gingival cleaning [160–162]. Establishing supportive therapy and reparative therapy has shown increased implant survival rates [163–165].

In addition to the maintenance of the peri-implant tissues, prosthetic maintenance is also essential and includes the establishment of cleansable contours of the implant restorations, adequate emergence profiles, removal of undercuts and food trapping areas, granting polished surfaces with reduced roughness, evaluation of the integrity of the restorations, confirmation of the prosthetic screw stability, and repair or replacement of defective restorations [166–169]. The long-term success and survival of the implant restorations and the health of peri-implant tissues also rely on maintaining the stability of the implant–abutment connection [89, 146]. However, nothing is said about how we should specifically maintain the IACs.

Therefore, considering the importance of the IACs, it is necessary to provide recommendations to address the microgap, reduce abutment micromovement, and reduce screw loosening. In addition, we summarize methods to clean and disinfect the implant connection. Finally, based on the exhaustive literature review of this subsection, we aim to describe a workflow for an ideal maintenance protocol for the IAC.

Addressing the Microgap

First, it is necessary to understand the microgap and its influence on the stability of the crestal bone. Microgaps are formed inside the implant connection, between the implant and abutment walls, and at the implant platform.

Four types of microgap are considered at the level of the implant platform [170]: Type I (no horizontal or vertical microgaps between the implant and the abutment), Type II (only horizontal misfit between the implant platform expressed by under-contoured or over-contoured abutments), Type III (vertical microgap), Type IV (horizontal and vertical microgap). The horizontal microgaps are usually bigger than the vertical microgaps in different abutments (castable, pre-machined, or pre-machined with cast-on) [170].

Hermann et al. [171] evaluated six IAC designs (3 IACs without microgap and 3 IACs with microgaps) with different microgap dimensions ($\langle 10, 50,$ and $100 \mu m$). Implants and their abutments were inserted, and after three months, the crestal bone loss was evaluated by histological analysis. The study showed that none of the single-body implants (implants without microgap) presented bone loss. However, the implants with microgap showed bone loss even with

the smallest microgap of $10 \mu m$. The authors conclude that the microgap allowed micromovement between the implant and the abutment, inducing crestal bone loss. Therefore the presence of a microgap, not its size, is responsible for bone loss [171].

Posteriorly, King et al. [172] in a similar experiment, evaluated the position of the microgap and its dimensions on crestal bone levels. Sixty implants (30 single-body and 30 two-piece) were inserted in the edentulous mandibles of foxhound dogs, leaving the IAC 1mm over the bone crest. The two-piece implants possessed three microgap sizes (\lt 10, 50, and 100 μ m), and the single-body implants did not have microgap. Radiographic evaluations were completed at one, two, and three months, and then histological samples were obtained to evaluate the bone levels. After one month, significantly higher bone loss was observed in the two-piece implants compared to single-body implants, independently of the microgap size.

The authors concluded that interfaces (no matter the size) would be associated with biological changes, and the potential micromovement between implant–abutment may have an early influence on peri-implant wound healing [172].

When a microgap is not present, mild inflammation of the peri-implant connective tissue is observed; meanwhile, moderate and severe inflammatory infiltrates are observed when the microgap is present [1, 173] If the microgap is moved coronally, less bone resorption is observed; however, if the microgap is displaced apically, bone resorption increases (in butt-join IACs) [174]. Larger microgaps exist in screw-retained restorations $(<60 \,\mu m$) compared to microgaps in cement-retained restorations $(40 \mu m)$, and larger microgaps will accumulate more bacteria [121]. Histologic analysis of failed implants showed heavy contamination with a heterogeneous microbial population along the entire IAC within the implant and the abutment interfaces, which supports the theory that the microgap placed at the bone level and colonized by bacteria or their subproducts represents a risk for bone loss [175].

The microgap size and the IAC design can produce different patterns of bone resorption [176]. Morse taper IACs resulted in less bone resorption and flat bone angles; meanwhile, internal hex produced acute bone angles with more bone resorption [177]. But, even the microgap of tight morse cone connections changes when loads are applied. Synchrotron-based radiography studies of the microgap of morse cone IACs showed that under different lateral forces, the microgap increased. Thus, minimal microgap $(>4 \mu m)$ was observed when no forces were applied. When a lateral force of 60N was applied, the microgap increased to $11 \,\mu m$; and under 100 N, the microgap increased up to $22 \mu m$ at the top and up to $15 \mu m$ at the bottom of the IAC [178].

The microgap can be detected clinically or radiographically. However, only microgaps over 150.9μm are detectable in radiographic evaluations. Meanwhile, the tactile clinical evaluation is not accurate enough to determine the presence of microgaps [179]. Within the same implant system, the abutment's fabrication method influences the dimensions of the microgap.

Rismanchian et al. [180] compared the microgaps between implants and castable abutments, castable on machined titanium abutments, solid titanium abutments, and synOcta abutments. The microgap was measured at four locations in each coupled implant–abutment system by scanning electron microscope (SEM) analysis. Their results showed that solid titanium abutments had the smallest microgap $(7.03 \pm 5.98 \,\mu m)$ compared to synOcta $(10.36 \pm 7.01 \,\mu m)$, castable on machined titanium ($95.92 \pm 8.75 \,\mu$ m), and castable abutments $(74.43 \pm 45.02 \,\mu\text{m})$ [180]. In addition, the microgap is smaller in IACs of implants connected with original abutments than IACs of implants connected with non-original components [181–183]. The consequences of smaller microgaps and better fit of original abutments are that original abutments provide better bacterial sealing, more homogeneous load distribution, and reduced micromovement compared to compatible but not-original abutments

Recommendations to Reduce the Microgap and its Effects

Although the microgap is present in all the IACs, conical and morse taper designs present the smallest microgaps and should be preferred. If butt joint IACs are used, the microgap should be maintained at the supracrestal level to reduce bone loss. Platform-switched IACs are preferred because the microgap is displaced inward, thus, distancing the bacteria colonies away from the adjacent bone. Standard and original abutments should be preferred because the microgap is smaller than in castable abutments. Original abutments must be chosen over non-original ones because the microgap is smaller and presents a better internal fit.

Addressing the Micromovement

Weinberg [184] listed the following definitions: macro movement (movement of a tooth or prostheses by more than 500μm and clinically detectable), micromovement (movement of tooth, prostheses, or implant components by 100–500μm not detectable clinically), and micronmovement (movement inferior to $100 \mu m$). In this subsection, micromovement will refer to the lateral or apical-coronal displacement of the implant abutment by $>100 \,\mu m$.

Yamanishi et al. [185] evaluated the effect of the implant neck and the abutment connection design on crestal bone stress and abutment micromovement. Finite elements analysis (FEA) was used to compare three IAC designs: external connection with a conical tapered neck, internal connection with a straight neck, and conical connection with a reverse conical neck. A simulated oblique force of 100 N was applied on the abutment in a 45°angle with the implant axis. The results showed that conical connections minimized the stress concentration on the labial side compared to external and internal connections. In addition, the micromovement was higher in external connection $(277.8 \,\mu m)$ compared to Internal $(238 \,\mu m)$ and conical connections (237.5μm) [185].

Repeated loading can produce fatigue of the IAC, increased microgap, deformation of the IAC, and micromovement in narrow implants. Rack et al. [186] evaluated the deformations and microgaps in narrow implants with a conical connection. Implants and abutments were connected and subject to 5 million cycles of fatigue loading under 120 N forces. The forces were applied axially and in different angulations (30° degree or 90° degree). The authors measured the microgap using synchrotron-based radiography with hard X-ray phase contrast before the loads were applied and after loading. Four areas were measured at the IAC (two at the coronal and two at the base of the IAC). Their results showed that microgaps changed after loading from $0.1-11$ to $0.1-11 \mu m$ [186]. Therefore, it is inferred that micromovement can also increase from increased microgap and deformation of the IAC.

Furthermore, the micromovement of original and nonoriginal abutments at the IAC was evaluated by Berberi et al. [187] Fifteen implants with 4.0mm diameter were connected with three abutments (five original, and five non-original from two different manufacturers). Increasing forces (From 25 up to 200 N) were applied in a 30°angle. A radiograph amplifier and X-ray videos were used to evaluate the microgap. The study showed that microgaps were only present before load application in the non-original abutments group $(5.2 \pm 3.63$ and $4.8 \pm 3.03 \,\mu$ m, respectively). When the forces reached 200 N, microgaps were detected in the original abutments group $(8.4 \pm 1.67 \,\text{\mu m})$; meanwhile, larger microgaps were observed in the non-original abutments $(12.4 \pm 3.29$ and $22.8 \pm 5.76 \,\mu$ m, respectively). The authors concluded that non-original components present significantly higher micromovements when compared with original abutments [187]. Finally, other parameters that can increase the micromovement at the IAC are connection and disconnection of the implant abutments [188], wear, and corrosion [189], and screw loosening [190].

Recommendations to Reduce the IAC Micromovement

Conical connections present the smallest micromovement and distribute the stress more efficiently than internal and external hexagonal connections and, therefore, should be preferred. However, due to repeated loading, deformation of the IAC can occur, and micromovement can appear. Therefore, it is recommended to use original abutments because they possess less micromovement than nonoriginal abutments.

Addressing Screw Loosening

Axial occlusal loads are transferred from the implant restoration apically toward the implant platform and the implant apex. Meanwhile, non-axial loads are transferred to the prosthetic screws, inner walls of the implant connection, and abutment connection [72, 191]. The forces are divided into vectors that induce deformation of the weakest part of the system (the abutment screws) [192]. The deformation of the abutment screws is expressed as loss of preload, which results in screw loosening, and screw loosening is the most frequent mechanical complication of single dental implants [193, 194].

Different causes for screw loosening have been evaluated in the literature, including lack or loss of preload [195], crown offset or crown dimensions and cantilevers [196, 197], misfit of the prostheses [198], and inadequate tightening protocols [199, 200].

A six-year retrospective clinical study evaluated the characteristics of screw loosening in 1928 implants. The study showed that screw loosening occurred in 7.2% of the implants, was detected before six months of loading, repeated in 22.3% of implants, was frequent in the molar region (8.5%), and in external connections (8.9%) and more frequent in single crowns (14%). The study demonstrated that screw loosening would be an early complication that can relapse and will be determined by the implant location and type of implant restoration [201].

Recommendations to Reduce Screw Loosening

The following measures can reduce the frequency of screw loosening:

- Reduce the vertical and horizontal offset of the implant restorations.
- Reduce the extension of the cantilevers.
- Apply repeated tightening protocols during the delivery of the implant restorations and use torque controllers to achieve adequate preload.

Confirm a proper fit of the implant and abutment.

Addressing the Decontamination of the IAC

Different methods can be used to decontaminate the implant connection. For example, the irrigation of the implant connection can remove non-attached debris and non-attached bacteria. Various antibacterial substances (like chlorhexidine and hydrogen peroxide) and modern laser therapies can be used to reduce microbial contamination. In this subsection, different methods for the decontamination of the IAC will be summarized.

Chlorhexidine

Paolantonio et al. [202] evaluated the effect of 1% chlorhexidine gel used to fill the implant connection on bacteria colonization of the IAC. Thirty patients received a single crown cemented temporarily, and after three months, the crowns were removed, gingival parameters recorded, and the patients were divided into two groups. In the control group, the patients received the screw-retained abutment and a temporary cemented crown. In the test group, the implant connections were filled with a 1% chlorhexidine gel before the insertion of the abutments.

After six months, the crowns were removed, and microbiological samples were obtained with paper points from the implants' connections. The results showed reduced bacterial counts and fewer periodontopathogen microorganisms in the test groups compared to controls. The authors concluded that a 1% chlorhexidine gel used to fill the implant cavity efficiently reduces microbial colonization for six months [202].

These results were confirmed by D'Ercole et al. [203] in a similar study of 25 patients. The only difference was that the microbiological samples were retrieved at baseline and after three months. The study's results also showed that a gel containing 1% chlorhexidine reduced the bacterial counts compared to controls. In addition, the controls showed increased levels of Aspartate aminotransferase (AST), which indicates increased cell death and tissue destruction [203].

Koutouzis et al. [124] evaluated the effect of a solution containing 0.2% chlorhexidine in preventing bacterial endotoxins' penetration into the IAC of morse taper implants. In this in vitro experiment. Thirty implants were used in this experiment. Twenty implants were inoculated with 1μl of *Escherichia coli* (*E. coli*) and divided into three groups. In group 1, after 24 hours of exposure to the bacteria, the supernatant was removed, and 0.5μl of sterile saline was added; in Group 2, after 24 hours, the supernatant was removed and 0.5μl of a solution containing 0.2% chlorhexidine was added. The remaining ten implants of Group 3 were inoculated with 0.5μ l of sterile saline to

serve as controls. Then, all the implants were connected with their respective abutments immersed in culture media and subjected to a simulated load of 160 N for 200,000 cycles. Samples were obtained from the culture solution at time 0 and every 9 hours until the completion of 54 hours (6 samples per implant were obtained).

The results showed that endotoxins penetrated the IAC of morse taper implants even containing chlorhexidine. These findings mean that although chlorhexidine at higher concentrations can reduce bacteria colonization, at lower concentrations (0.2% chlorhexidine) cannot prevent the penetration of endotoxins [124].

Chlorhexidine Used in Combination with Channel Sealers

Podhorsky et al. [204] compared the efficacy of a 2% chlorhexidine gel, a special silicone filler, sealing grease, or no filler to prevent the microbial contamination of implants with internal hexagon and conical connection immersed in a solution with a suspension of *E. coli* for one week. The implants were subject to thermocycling, the abutments were removed, and samples from the implant connections were obtained. The presence of bacteria was evaluated with real-time polymerase chain reaction (PCR). The results showed that bacteria penetrated all the implant connections, but the bacterial load was minimal in the treated implants. Therefore, the authors concluded that "the application of products to reduce bacterial invasion can reduce the bacterial load and therefore can help minimize the cofactors that contribute to the development of peri-implantitis." [204]

Similar results were obtained by Ozdiler et al. [205] who evaluated the same type of sealants (2% chlorhexidine gel, special silicone filler, and sealing grease) but used conical implants with different taper angles at the IAC. After applying the sealants to the implant intaglio, the abutments were connected and immersed in a solution containing *Enterococcus faecalis* (*E. faecalis*). In addition, low-intensity loads (50 N) were applied, but more cycles were used (500,000).

Afterward, the abutments were disconnected, and samples were obtained to measure bacterial contamination. The authors found bacteria in all the implants, the taper angle did not influence the number of bacteria and applying a 2% chlorhexidine gel and sealants reduced the bacteria counts [205].

Photodynamic Therapy and Chlorhexidine

Alresheedi et al. [206] in a randomized clinical trial evaluated the impact of abutment disinfection with 0.2% chlorhexidine gel or photodynamic therapy before its delivery on the clinical status of dental implants. Forty-five patients received implants at the first mandibular molar area. Three disinfection protocols were applied to the abutments before insertion.

Group 1 received photodynamic therapy, Group 2 received 0.2% chlorhexidine gel, and Group 3 was steamed. Peri-implant parameters (modified plaque and gingival indices, probing depths, and radiographic crestal bone loss) were evaluated after one year. The results showed that all the implants maintained normal peri-implant indexes without evidence of bone loss. Also, the patients reported that the minimum frequency for brushing and flossing was two-times per day. The authors concluded that implant abutment decontamination with any of the three methods and strict oral hygiene efficiently maintain peri-implant health [206].

Other Decontamination Methods

Fletcher et al. [207] used a new decontamination method for implant surfaces including mechanical debridement and disinfection with dilutions of hypochlorite, hydrogen peroxide, and sterile saline. After the application of this methodology, the implants demonstrated successful histological re-osseointegration.

Canullo et al. [208, 209] described the use of Plasma of Argon for the decontamination of implant abutments. In a randomized controlled trial with a five-year follow-up, the authors evaluated two methods for the decontamination of implant abutments before insertion (steaming or plasma of Argon). The evaluation parameters relevant to the decontamination protocol were biological complications, marginal bone loss, and periodontal parameters. The study showed a higher bone loss in the steaming decontamination protocol and higher soft tissue gain in the plasma argon decontamination protocol. The authors conclude that the plasma of Argon can be used as a disinfection protocol for the prosthetic abutments before insertion to minimize crestal bone loss [208].

Considerations and Recommended Protocols for Maintenance of the IACs

Protocols for decontamination of the implant connection after implant insertion, during the delivery of the implant restoration, and during maintenance are described.

In addition, protocols for the reduction of screw loosening are presented. In the present protocols, only actions that can increase the stability of the IACs and reduce their contamination are discussed. Therefore, the evaluations of peri-implant parameters are not part of this section (Figures 8.18–8.20).

Figure 8.18 Recommended protocol for maintenance of the IAC immediately after implant insertion.

Figure 8.19 Recommended protocol for maintenance of the IAC during delivery of implant restorations.

Figure 8.20 Recommended protocol for maintenance of the IAC during regular maintenance visits.

References

- **1** Broggini, N., McManus, L., Hermann, J. et al. (2003). Persistent acute inflammation at the implant-abutment interface. *Journal of Dental Research* 82: 232–237.
- **2** ICOI (2017). Glossary of Implant Dentistry. *International Congress of Oral Implantologist* 117: e6.
- **3** Binon, P. and McHugh, M. (1996). The effect of eliminating implant/abutment rotational misfit on screw joint stability. *The International Journal of Prosthodontics* 9: 511–519.
- **4** Binon, P. (1995). Evaluation of machining accuracy and consistency of selected implants, standard abutments, and laboratory analogs. *The International Journal of Prosthodontics* 8: 162–178.
- **5** Binon, P. (1996). Evaluation of three slip fit hexagonal implants. *Implant Dentistry* 5: 235–248.
- **6** Binon, P. (1998). Evaluation of the effectiveness of a technique to prevent screw loosening. *The Journal of Prosthetic Dentistry* 79: 430–432.
- **7** Binon, P. (2000). Implants and components: entering the new millennium. *The International Journal of Oral & Maxillofacial Implants* 15: 76–94.
- **8** Lorenzoni, F., Coelho, P., Bonfante, G. et al. (2011). Sealing Capability and SEM Observation of the Implant-Abutment Interface. *International Journal of Dentistry* 2011: 864183. <https://doi.org/10.1155/2011/864183>.
- **9** Welander, M., Abrahamsson, I., and Berglundh, T. (2009). Subcrestal placement of two-part implants. *Clinical Oral Implants Research* 20: 226–231.
- **10** Guo, T., Gulati, K., Arora, H. et al. (2021). Race to invade: Understanding soft tissue integration at the transmucosal region of titanium dental implants. *Dental Materials* 37: 816–831.
- **11** Almas, K., Javed, F., and Smith, S. (2018). *Glossary of Dental Implantology*, 178–179. Wiley Blackwell.
- **12** Vinhas, A., Aroso, C., Salazar, F. et al. (2020). Review of the mechanical behavior of different implant-abutment connections. *International Journal of Environmental Research and Public Health* 17: 8685. [https://doi.](https://doi.org/10.3390/ijerph17228685) [org/10.3390/ijerph17228685](https://doi.org/10.3390/ijerph17228685).
- **13** Hahn, J. (2005). One-piece root-form implants: a return to simplicity. *The Journal of Oral Implantology* 31: 77–84.
- **14** Jones, A. and Cochran, D. (2006). Consequences of implant design. *Dental Clinics of North America* 50: 339–360.
- **15** Rosenlicht, J. (2002). SwissPlus implant system, part 1: surgical aspects and intersystem comparisons. *Implant Dentistry* 11: 144–153.
- **16** Hermann, J., Buser, D., Schenk, R. et al. (2001). Biologic Width around one- and two-piece titanium implants. *Clinical Oral Implants Research* 12: 559–571.
- **17** Nkenke, E., Eitner, S., Radespiel-Tröger, M. et al. (2007). Patient-centered outcomes comparing transmucosal implant placement with an open approach in the maxilla: a prospective, non-randomized pilot study. *Clinical Oral Implants Research* 18: 197–203.
- **18** Engquist, B., Astrand, P., Anzén, B. et al. (2005). Simplified methods of implant treatment in the edentulous lower jaw: a 3-year follow-up report of a controlled prospective study of one-stage versus twostage surgery and early loading. *Clinical Implant Dentistry and Related Research* 7: 95–104.
- **19** Singh, S., Mishra, S., and Chowdhary, R. (2022). Patient satisfaction and crestal bone changes with one-piece and two-piece single implant-retained mandibular overdenture: a randomized controlled clinical study. *Journal of Prosthodontic Research* [https://doi.org/10.2186/](https://doi.org/10.2186/jpr.JPR_D_20_00315) [jpr.JPR_D_20_00315](https://doi.org/10.2186/jpr.JPR_D_20_00315).
- **20** Cehreli, M., Akça, K., and Iplikçioğlu, H. (2004). Force transmission of one- and two-piece morse-taper oral implants: a nonlinear finite element analysis. *Clinical Oral Implants Research* 15: 481–489.
- **21** Finne, K., Rompen, E., and Toljanic, J. (2007). Prospective multicenter study of marginal bone level and soft tissue health of a one-piece implant after two years. *The Journal of Prosthetic Dentistry* 97 (6 Suppl): S79–S85.
- **22** Baer, R., Shanaman, R., Clark, P., and Medley, M. (2013). A 3-year multicenter study of marginal bone level and soft tissue health of a 1-piece implant. *Implant Dentistry* 22: 366–373.
- **23** Silva, N., Coelho, P., Fernandes, C. et al. (2009). Reliability of one-piece ceramic implant. *Journal of Biomedical Materials Research. Part B, Applied Biomaterials* 88: 419–426.
- **24** Gabay, E., Cohen, O., and Machtei, E. (2010). Heat production during prosthetic preparation of a one-piece dental implant. *The International Journal of Oral & Maxillofacial Implants* 25: 1131–1136.
- **25** Parel, S. and Schow, S. (2005). Early clinical experience with a new one-piece implant system in single tooth sites. *Journal of Oral and Maxillofacial Surgery* 63 (S2): 2–10.
- **26** Zembić, A., Johannesen, L., Schou, S. et al. (2012). Immediately restored one-piece single-tooth implants with reduced diameter: one-year results of a multi-center study. *Clinical Oral Implants Research* 23: 49–54.
- **27** Ostman, P., Hellman, M., Albrektsson, T., and Sennerby, L. (2007). Direct loading of nobel direct and nobel perfect one-piece implants: a 1-year prospective clinical and radiographic study. *Clinical Oral Implants Research* 18: 409–418.
- **28** Prithviraj, D., Gupta, V., Muley, N., and Sandhu, P. (2013). One-piece implants: placement timing, surgical technique, loading protocol, and marginal bone loss. *Journal of Prosthodontics* 22: 237–244.
- **29** Barrachina-Díez, J., Tashkandi, E., Stampf, S., and Att, W. (2013). Long-term outcome of one-piece implants. Part II: prosthetic outcomes. A systematic literature review with meta-analysis. *The International Journal of Oral & Maxillofacial Implants* 28: 1470–1482.
- **30** Andreasi Bassi, M., Lopez, M., Confalone, L. et al. (2016). Clinical outcome of a two-piece implant system with an internal hexagonal connection: a prospective study. *Journal of Biological Regulators and Homeostatic Agents* 30: 7–12.
- **31** Engquist, B., Astrand, P., Anzén, B. et al. (2002). Simplified methods of implant treatment in the edentulous lower jaw. A controlled prospective study. Part I: one-stage versus two-stage surgery. *Clinical Implant Dentistry and Related Research* 4: 93–103.
- **32** Ormianer, Z., Duda, M., Block, J., and Matalon, S. (2015). One- and two-piece implants placed in the same patients: clinical outcomes after 5 years of function. *The International Journal of Prosthodontics* 29: 608–610.
- **33** Gamper, F., Benic, G., Sanz-Martin, I. et al. (2017). Randomized controlled clinical trial comparing one-piece and two-piece dental implants supporting fixed and removable dental prostheses: 4- to 6-year observations. *Clinical Oral Implants Research* 28: 1553–1559.
- **34** Prati, C., Zamparini, F., Canullo, L. et al. (2020). Factors affecting soft and hard tissues around two-piece transmucosal implants: a 3-year prospective cohort study. *The International Journal of Oral & Maxillofacial Implants* 35: 1022–1036.
- **35** ten Bruggenkate, C.M., Sutter, F., Oosterbeek, H.S., and Schroeder, A. (1992). Indications for angled implants. *The Journal of Prosthetic Dentistry* 67: 85–93.
- **36** Cardelli, P., Montani, M., Gallio, M. et al. (2009). Angulated abutments and perimplants stress: F.E.M. analysis. *Oral & Implantology (Rome)* 2: 3–10.
- **37** Cavallaro, J. Jr. and Greenstein, G. (2011). Angled implant abutments: a practical application of available knowledge. *Journal of the American Dental Association (1939)* 142: 150–158.
- **38** Garber, D.A., Salama, H., and Salama, M.A. (2001). Two-stage versus one-stage – is there really a controversy? *Journal of Periodontology* 72: 417–421.
- **39** Esposito, M., Grusovin, M.G., Chew, Y.S. et al. (2009). One-stage versus two-stage implant placement. A Cochrane systematic review of randomised controlled clinical trials. *European Journal of Oral Implantology* 2: 91–99.
- **40** Byrne, G. (2010). Outcomes of one-stage versus two-stage implant placement. *Journal of the American Dental Association (1939)* 141: 1257–1258.
- **41** Assenza, B., Tripodi, D., Scarano, A. et al. (2012). Bacterial leakage in implants with different

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implant-abutment connections: an in vitro study. *Journal of Periodontology* 83: 491–497.

- **42** Khraisat, A., Abu-Hammad, O., Al-Kayed, A.M., and Dar-Odeh, N. (2004). Stability of the implant/abutment joint in a single-tooth external-hexagon implant system: clinical and mechanical review. *Clinical Implant Dentistry and Related Research* 6: 222–229.
- **43** Sun, F., Cheng, W., Zhao, B. et al. (2022). Evaluation the loosening of abutment screws in fluid contamination: an in vitro study. *Scientific Reports* 12: 10797. [https://doi.](https://doi.org/10.1038/s41598-022-14791-w) [org/10.1038/s41598-022-14791-w.](https://doi.org/10.1038/s41598-022-14791-w)
- **44** Martin, W., Woody, R., Miller, B., and Miller, A. (2001). Implant abutment screw rotations and preloads for four different screw materials and surfaces. *The Journal of Prosthetic Dentistry* 86: 24–32.
- **45** Bagegni, A., Weihrauch, V., Vach, K., and Kohal, R. (2022). The mechanical behavior of a screwless morse taper implant-abutment connection: an in vitro study. *Materials (Basel)* 15: 3381. [https://doi.org/10.3390/](https://doi.org/10.3390/ma15093381) [ma15093381](https://doi.org/10.3390/ma15093381).
- **46** Moon, S., Kim, H., Son, M., and Chung, C. (2009). Sinking and fit of abutment of locking taper implant system. *The Journal of Advanced Prosthodontics* 1: 97–101.
- **47** Urdaneta, R., Marincola, M., Weed, M., and Chuang, S. (2008). A screwless and cementless technique for the restoration of single-tooth implants: a retrospective cohort study. *Journal of Prosthodontics* 17: 562–571.
- **48** Al Amri, M.D. (2016). Crestal bone loss around submerged and non-submerged dental implants: a systematic review. *The Journal of Prosthetic Dentistry* 115: 564–570.
- **49** Valles, C., Rodríguez-Ciurana, X., Clementini, M. et al. (2018). Influence of subcrestal implant placement compared with equicrestal position on the peri-implant hard and soft tissues around platform-switched implants: a systematic review and meta-analysis. *Clinical Oral Investigations* 22: 555–570.
- **50** Palacios-Garzón, N., Velasco-Ortega, E., and López-López, J. (2019). Bone loss in implants placed at subcrestal and crestal level: a systematic review and meta-analysis. *Materials (Basel)* 12: 154. [https://doi.](https://doi.org/10.3390/ma12010154) [org/10.3390/ma12010154.](https://doi.org/10.3390/ma12010154)
- **51** Cruz, R., Lemos, C., de Luna Gomes, J. et al. (2022). Clinical comparison between crestal and subcrestal dental implants: a systematic review and meta-analysis. *The Journal of Prosthetic Dentistry* 127: 408–417.
- **52** Glibert, M., Vervaeke, S., Jacquet, W. et al. (2018). A randomized controlled clinical trial to assess crestal bone remodeling of four different implant designs. *Clinical Implant Dentistry and Related Research* 20: 455–462.
- **53** Maghsoudi, P., Slot, D.E., and van der Weijden, F.G.A. (2022). Bone remodeling around dental implants

after 1-1.5 years of functional loading: a retrospective analysis of two-stage implants. *Cllinical and Experimental Dental Research* 8: 680–689.

- **54** Romanos, G.E., Aydin, E., Gärtner, K., and Nentwig, G.H. (2015). Long-term results after subcrestal or crestal placement of delayed loaded implants. *Clinical Implant Dentistry and Related Research* 17 (1): 133–141.
- **55** Vervaeke, S., Matthys, C., Nassar, R. et al. (2018). Adapting the vertical position of implants with a conical connection in relation to soft tissue thickness prevents early implant surface exposure: a 2-year prospective intra-subject comparison. *Journal of Clinical Periodontology* 45: 605–612.
- **56** Spinato, S., Bernardello, F., Lombardi, T. et al. (2022). Influence of apico-coronal positioning of tissue-level implants on marginal bone stability during supracrestal tissue height establishment: a multi-center prospective study. *Clinical Implant Dentistry and Related Research* 24: 611–620.
- **57** Berglundh, T. and Lindhe, J. (1996). Dimension of the periimplant mucosa. Bio- logical width revisited. *Journal of Clinical Periodontology* 23: 971–973.
- **58** Di Gianfilippo, R., Valente, N., Toti, P. et al. (2020). Influence of implant mucosal thickness on early bone loss: a systematic review with meta-analysis. *Journal of Periodontal & Implant Science* 50: 209–225.
- **59** Sun, P., Yu, D., Luo, X. et al. (2022). The effect of initial biologic width on marginal bone loss: a retrospective study. *The International Journal of Oral & Maxillofacial Implants* 37: 190–198.
- **60** Cardaropoli, G., Araújo, M., and Lindhe, J. (2003). Dynamics of bone tissue for- mation in tooth extraction sites. An experimental study in dogs. *Journal of Clinical Periodontology* 30: 809–818.
- **61** Huynh-Ba, G., Pjetursson, B., Sanz, M. et al. (2010). Analysis of the socket bone wall dimensions in the upper maxilla in relation to immediate implant placement. *Clinical Oral Implants Research* 21: 37–42.
- **62** Pellicer-Chover, H., Rojo-Sanchís, J., Peñarrocha-Diago, M. et al. (2020). Radiological implications of crestal and subcrestal implant placement in posterior areas. A cone-beam computed tomography study. *Journal of Clinical and Experimental Dentistry* 12: e870–e876.
- **63** de Siqueira, R., Savaget Gonçalves Junior, R., Dos Santos, P. et al. (2020). Effect of different implant placement depths on crestal bone levels and soft tissue behavior: a 5-year randomized clinical trial. *Clinical Oral Implants Research* 31: 282–293.
- **64** Gracis, S., Michalakis, K., Vigolo, P. et al. (2012). Internal vs. external connections for abutments/reconstructions: a systematic review. *Clinical Oral Implants Research* 23 (S6): 202–216.
- **65** Balfour, A. and O'Brien, G. (1995). Comparative study of anti-rotational single tooth abutments. *The Journal of Prosthetic Dentistry* 73: 36–43.
- **66** Weinberg, L. (1993). The biomechanics of force distribution in implant-supported prostheses. *The International Journal of Oral & Maxillofacial Implants* 8: 19–31.
- **67** Huang, Y. and Wang, J. (2019). Mechanism of and factors associated with the loosening of the implant abutment screw: a review. *Journal of Esthetic and Restorative Dentistry* 31: 338–345.
- **68** Machtei, E., Oved-Peleg, E., and Peled, M. (2006). Comparison of clinical, radiographic and immunological parameters of teeth and different dental implant platforms. *Clinical Oral Implants Research* 17: 658–665.
- **69** Theoharidou, A., Petridis, H.P., Tzannas, K., and Garefis, P. (2008). Abutment screw loosening in single-implant restorations: a systematic review. *The International Journal of Oral & Maxillofacial Implants* 23: 681–690.
- **70** Ceruso, F., Barnaba, P., Mazzoleni, S. et al. (2017). Implant-abutment connections on single crowns: a systematic review. *Oral & Implantology (Rome)* 10: 349–353.
- **71** Vetromilla, B., Brondani, L.P., Pereira-Cenci, T., and Bergoli, C. (2019). Influence of different implantabutment connection designs on the mechanical and biological behavior of single-tooth implants in the maxillary esthetic zone: a systematic review. *The Journal of Prosthetic Dentistry* 121: 398–403.
- **72** Merz, B., Hunenbart, S., and Belser, U. (2000). Mechanics of the implant-abutment connection: an 8-degree taper compared to a butt joint connection. *The International Journal of Oral & Maxillofacial Implants* 15: 519–526.
- **73** Lazzara, R. and Porter, S. (2006). Platform-switching: a new concept in implant dentistry for controlling postrestorative crestal bone levels. *International Journal of Periodontics and Restorative Dentistry* 26: 9–17.
- **74** Hermann, F., Lerner, H., and Palti, A. (2007). Factors influencing the preservation of the periimplant marginal bone. *Implant Dentistry* 16: 165–175.
- **75** Canullo, L. and Rasperini, G. (2007). Preservation of peri-implant soft and hard tissues using platformswitching of implants placed in immediate extraction sockets: a proof-of-concept study with 12- to 36-month follow-up. *The International Journal of Oral & Maxillofacial Implants* 22: 995–1000.
- **76** Luongo, R., Traini, T., Guidone, P. et al. (2008). Hard and soft tissue responses to the platform-switching technique. *International Journal of Periodontics and Restorative Dentistry* 28: 551–557.
- **77** Canullo, L., Fedele, G., Iannello, G., and Jepsen, S. (2010). Platform-switching and marginal bone-level alterations:

the results of a randomized-controlled trial. *Clinical Oral Implants Research* 21: 115–121.

- **78** Farronato, D., Santoro, G., Canullo, L. et al. (2012). Establishment of the epithelial attachment and connective tissue adaptation to implants installed under the concept of "platform-switching": a histologic study in minipigs. *Clinical Oral Implants Research* 23: 90–94.
- **79** Cocchetto, R., Traini, T., Caddeo, F., and Celletti, R. (2010). Evaluation of hard tissue response around wider platform-switched implants. *International Journal of Periodontics and Restorative Dentistry* 30: 163–171.
- **80** Fickl, S., Zuhr, O., Stein, J.M., and Hürzeler, M.B. (2010). Peri-implant bone level around implants with platformswitched abutments. *The International Journal of Oral & Maxillofacial Implants* 25: 577–581.
- **81** Vandeweghe, S. and De Bruyn, H. (2012). A withinimplant comparison to evaluate the concept of platformswitching: a randomised controlled trial. *European Journal of Oral Implantology* 5: 253–262.
- **82** Maeda, Y., Miura, J., Taki, I., and Sogo, M. (2007). Biomechanical analysis on platform-switching: is there any biomechanical rationale? *Clinical Oral Implants Research* 18: 581–584.
- **83** Canay, S. and Akça, K. (2009). Biomechanical aspects of bone-level diameter shifting at implant-abutment interface. *Implant Dentistry* 18: 239–248.
- **84** Chang, C., Chen, C., and Hsu, M. (2010). Biomechanical effect of platform-switching in implant dentistry: a threedimensional finite element analysis. *The International Journal of Oral & Maxillofacial Implants* 25: 295–304.
- **85** Alrabeah, G.O., Knowles, J.C., and Petridis, H. (2016). The effect of platform-switching on the levels of metal ion release from different implant-abutment couples. *International Journal of Oral Science* 8: 117–125.
- **86** Alrabeah, G.O., Knowles, J.C., and Petridis, H. (2018). Reduction of tribocorrosion products when using the platform-switching concept. *Journal of Dental Research* 97: 995–1002.
- **87** Mishra, S.K., Gaddale, R., Sonnahalli, N.K., and Chowdhary, R. (2021). Platform-switching concept in dental implants: a systematic review and meta-analysis of randomized controlled trials with a minimum follow-up of 3 years. *The International Journal of Oral & Maxillofacial Implants* 36: e97–e109.
- **88** Pita, M.S., Anchieta, R.B., Barão, V.A. et al. (2011). Prosthetic platforms in implant dentistry. *The Journal of Craniofacial Surgery* 22: 2327–2331.
- **89** Block, M.S. (2022). Evidence-based criteria for an ideal abutment implant connection-a narrative review. *Journal of Oral and Maxillofacial Surgery* 80: 1670–1675.
- **90** Brånemark, P., Adell, R., Breine, U. et al. (1969). Intraosseous anchorage of dental prostheses. I. Experimental

studies. *Scandinavian Journal of Plastic and Reconstructive Surgery* 3: 81–100.

- **91** Davi, L., Golin, A., Bernardes, S. et al. (2008). In vitro integrity of implant external hexagon after application of surgical placement torque simulating implant locking. *Brazilian Oral Research* 22: 125–131.
- **92** Jemt, T. (2008). Single implants in the anterior maxilla after 15 years of follow-up: comparison with central implants in the edentulous maxilla. *The International Journal of Prosthodontics* 21: 400–408.
- **93** do Nascimento, C., Miani, P.K., Pedrazzi, V. et al. (2012). Leakage of saliva through the implant-abutment interface: in vitro evaluation of three different implant connections under unloaded and loaded conditions. *The International Journal of Oral & Maxillofacial Implants* 27: 551–560.
- **94** Saidin, S., Abdul Kadir, M., Sulaiman, E., and Abu Kasim, N. (2012). Effects of different implant-abutment connections on micromotion and stress distribution: prediction of microgap formation. *Journal of Dentistry* 40: 467–474.
- **95** Sutter, F., Weber, H., Sorenson, J., and Belser, U. (1993). The new restorative concept of the ITI dental implant system: design and engineering. *International Journal of Periodontics and Restorative Dentistry* 13: 409–431.
- **96** Schlosser (2001). Morse taper. [http://showcase.netins.](http://showcase.netins.net/web/schlosser/mttaper.htm) [net/web/schlosser/mttaper.htm](http://showcase.netins.net/web/schlosser/mttaper.htm) (accessed August 2022).
- **97** Norton, M. (1997). An in vitro evaluation of the strength of an internal conical interface compared to a butt joint interface in implant design. *Clinical Oral Implants Research* 8: 290–298.
- **98** Perriard, J., Wiskott, W., Mellal, A. et al. (2002). Fatigue resistance of ITI implant abutment connectors: a comparison of the standard cone with a novel internally keyed design. *Clinical Oral Implants Research* 13: 542–549.
- **99** Kim, K. and Lim, Y. (2020). Axial displacements and removal torque changes of five different implantabutment connections under static vertical loading. *Materials (Basel)* 13: 699. [https://doi.org/10.3390/](https://doi.org/10.3390/ma13030699) [ma13030699](https://doi.org/10.3390/ma13030699).
- **100** Kim, K., Han, J., and Lim, Y. (2014). Settling of abutments into implants and changes in removal torque in five different implant-abutment connections. Part 1: cyclic loading. *The International Journal of Oral & Maxillofacial Implants* 29: 1079–1084.
- **101** Finger, I., Castellon, P., Block, M., and Elian, N. (2003). The evolution of external and internal implant/ abutment connections. *Practical Procedures & Aesthetic Dentistry* 15: 625–632.
- **102** Tang, C., Liul, S., Zhou, G. et al. (2012). Nonlinear finite element analysis of three implant- abutment interface

designs. *International Journal of Oral Science* 4: 101–108.

- **103** Semper, W., Kraft, S., Kruger, T., and Nelson, K. (2009). Theoretical considerations: implant positional index design. *Journal of Dental Research* 88: 725–730.
- **104** Semper, W., Heberer, S., Merhof, J. et al. (2010). Effects of repeated manual disassembly and reassembly on the positional stability of various implant-abutment complexes: an experimental study. *The International Journal of Oral & Maxillofacial Implants* 25: 86–94.
- **105** Semper-Hogg, W., Kraft, S., Stiller, S. et al. (2013). Analytical and experimental position stability of the abutment in different dental implant systems with a conical implant-abutment connection. *Clinical Oral Investigations* 17: 1017–1023.
- **106** Nicoll, R., Sun, A., Haney, S., and Turkyilmaz, I. (2013). Precision of fit between implant impression coping and implant replica pairs for three implant systems. *The Journal of Prosthetic Dentistry* 109: 37–43.
- **107** Graf, T., Güth, J., Edelhoff, D. et al. (2021). Influence of butt joint connections with long guiding areas on the stability of single crowns and 3-unit bridges – an in-vitro-study. *Journal of Prosthodontic Research* 65: 455–460.
- **108** Kofron, M., Carstens, M., Fu, C., and Wen, H. (2019). In vitro assessment of connection strength and stability of internal implant-abutment connections. *Clinical Biomechanics (Bristol, Avon)* 65: 92–99.
- **109** Sutter, F., Schroeder, A., and Buser, D. (1988). The new concept of ITI hollow-cylinder and hollow-screw implants: Part 1. Engineering and design. *The International Journal of Oral & Maxillofacial Implants* 3: 161–172.
- **110** Levine, R., Clem, D. 3rd, Wilson, T. Jr. et al. (1999). Multicenter retrospective analysis of the ITI implant system used for single-tooth replacements: results of loading for 2 or more years. *The International Journal of Oral & Maxillofacial Implants* 14: 516–520.
- **111** Arvidson, K., Bystedt, H., Grykholm, A. et al. (1998). Five-year prospective follow-up report of the Astra Tech dental implant system in the treatment of edentulous mandibles. *Clinical Oral Implants Research* 9: 225–234.
- **112** May, D. and Romanos, G. (2002). Immediate implantsupported mandibular overdentures retained by conical crowns: a new treatment concept. *Quintessence International* 1: 5–12.
- **113** Weigl, P. (2004). New prosthetic restorative features of Ankylos implant system. *The Journal of Oral Implantology* 30: 178–188.
- **114** Coppedê, A., Bersani, E., de Mattos Mda, G. et al. (2009). Fracture resistance of the implant-abutment connection in implants with internal hex and internal

conical connections under oblique compressive loading: an in vitro study. *The International Journal of Prosthodontics* 22 (3): 283–286.

- **115** Schmitt, C., Nogueira-Filho, G., Tenenbaum, H. et al. (2014). Performance of conical abutment (Morse Taper) connection implants: a systematic review. *Journal of Biomedical Materials Research. Part A* 102: 552–574.
- **116** Macedo, J., Pereira, J., Vahey, B. et al. (2016). Morse taper dental implants and platform-switching: the new paradigm in oral implantology. *European Journal of Dentistry* 10: 148–154.
- **117** Asmarz, H., Magrin, G., Prado, A. et al. (2021). Evaluation of removal torque and internal surface alterations in frictional morse taper connections after mechanical loading associated or not with oral biofilm. *The International Journal of Oral & Maxillofacial Implants* 36: 492–501.
- **118** Yang, F., Ruan, Y., Liu, Y. et al. (2022). Abutment mechanical complications of a Morse taper connection implant system: A 1- to 9-year retrospective study. *Clinical Implant Dentistry and Related Research* 24: 683–695.
- **119** Quirynen, M. and Van Steenberghe, D. (1993). Bacterial colonization of the internal part of two stage implants. An in vivo study. *Clinical Oral Implants Research* 4: 158–161.
- **120** Piattelli, A., Scarano, A., Paolantonio, M. et al. (2001). Fluids and microbial penetration in the internal part of cement-retained versus screw-retained implantabutment connections. *Journal of Periodontology* 72: 1146–1150.
- **121** Scarano, A., Assenza, B., Piattelli, M. et al. (2005). A 16-year study of the microgap between 272 human titanium implants and their abutments. *The Journal of Oral Implantology* 31: 269–275.
- **122** Jaworski, M., Melo, A., Picheth, C., and Sartori, I. (2012). Analysis of the bacterial seal at the implantabutment interface in external hexagon and Morse taper-connection implants: an in vitro study using a new methodology. *The International Journal of Oral & Maxillofacial Implants* 27: 1091–1095.
- **123** Candotto, V., Gabrione, F., Oberti, L. et al. (2019). The role of implant-abutment connection in preventing bacterial leakage: a review. *Journal of Biological Regulators and Homeostatic Agents* 33 (Suppl. 1): 129–134.
- **124** Koutouzis, T., Gadalla, H., Kettler, Z. et al. (2015). The role of chlorhexidine on endotoxin penetration to the implant-abutment interface (IAI). *Clinical Implant Dentistry and Related Research* 17 (476): 482.
- **125** Romanos, G.E., Biltucci, M.T., Kokaras, A., and Paster, B.J. (2016). Bacterial composition at the

implant-abutment connection under loading in vivo. *Clinical Implant Dentistry and Related Research* 18 (1): 138–145.

- **126** Jansen, V., Conrads, G., and Richter, E. (1997). Microbial leakage and marginal fit of the implant-abutment interface. *The International Journal of Oral & Maxillofacial Implants* 12: 527–540.
- **127** Peñarrocha-Oltra, D., Rossetti, P., Covani, U. et al. (2015). Microbial leakage at the implant-abutment connection due to implant insertion maneuvers: cross-sectional study 5 years postloading in healthy patients. *The Journal of Oral Implantology* 41: e292–e296.
- **128** Jinno, Y., Johansson, K., Stocchero, M. et al. (2019). Impact of salivary contamination during placement of implants with simultaneous bony augmentation in iliac bone in sheep. *The British Journal of Oral & Maxillofacial Surgery* 57: 1131–1136.
- **129** Jinno, Y., Jimbo, R., Hjalmarsson, J. et al. (2019). Impact of surface contamination of implants with saliva during placement in augmented bone defects in sheep calvaria. *The British Journal of Oral & Maxillofacial Surgery* 57: 41–46.
- **130** de Barros Lucena, G.A., de Molon, R.S., Moretti, A.J. et al. (2018). Evaluation of microbial contamination in the inner surface of titanium implants before healing abutment connection: a prospective clinical trial. *The International Journal of Oral & Maxillofacial Implants* 33: 853–862.
- **131** Canullo, L., Tallarico, M., Penarrocha, M. et al. (2017). Plasma of argon cleaning treatment on implant abutments in periodontally healthy patients: six years postloading results of a randomized controlled trial. *International Journal of Periodontics and Restorative Dentistry* 37: 683–690.
- **132** Yin, L., Zhu, Y., Yu, H., and Qiu, L. (2022). Evaluating the efficiency of three methods to clean and disinfect screw- and cement-retained prostheses. *The Journal of Prosthetic Dentistry* 127: 775–782.
- **133** Callan, D., Cobb, C., and Williams, K. (2005). DNA probe identification of bacteria colonizing internal surfaces of the implant-abutment interface: a preliminary study. *Journal of Periodontology* 76: 115–120.
- **134** Wadhwani, C., Schonnenbaum, T., Audia, F., and Chung, K. (2016). Invitro study of the contamination remaining on used healing abutments after cleaning and sterilizing in dental practice. *Clinical Implant Dentistry and Related Research* 18: 1069–1074.
- **135** Sahin, S.C. and Dere, K.A. (2021). Evaluation of residual contamination on reused healing abutments. *Clinical Oral Investigations* 25: 5889–5895.

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- **136** Sennerby, L., Lekholm, U., and Ericson, L. (1989). Soft-tissue response to clinically retrieved titanium cover screws reimplanted in the rat abdominal wall. *The International Journal of Oral & Maxillofacial Implants* 4: 233–239.
- **137** Nahas, A., Dechichi, P., de Magalhães, D., and Vieira, A. (2010). Histologic analysis of the oral mucosa lining osseointegrated implant cover screws: a study in humans. *The Journal of Oral Implantology* 36: 3–10.
- **138** Caldara, M., Belgiovine, C., Secchi, E., and Rusconi, R. (2022). Environmental, Microbiological, and Immunological Features of Bacterial Biofilms Associated with Implanted Medical Devices. *Clinical Microbiology Reviews* 35 (2): e0022120. [https://doi.](https://doi.org/10.1128/cmr.00221-20) [org/10.1128/cmr.00221-20.](https://doi.org/10.1128/cmr.00221-20)
- **139** Wheeler, J., Secchi, E., Rusconi, R., and Stocker, R. (2019). Not just going with the flow: the effects of fluid flow on bacteria and plankton. *Annual Review of Cell and Developmental Biology* 35: 213–237.
- **140** Rusconi, R., Guasto, J., and Stocker, R. (2014). Bacterial transport suppressed by fluid shear. *Nature Physics* 10: 212–217.
- **141** Secchi, E., Vitale, A., Miño, G. et al. (2020). The effect of flow on swimming bacteria controls the initial colonization of curved surfaces. *Nature Communications* 2020 (11): 2851. [https://doi.org/10.1038/](https://doi.org/10.1038/s41467-020-16620-y) [s41467-020-16620-y](https://doi.org/10.1038/s41467-020-16620-y).
- **142** Fernandes, P., Grenho, L., Fernandes, M. et al. (2021). Microgap and bacterial microleakage during the osseointegration period: an in vitro assessment of the cover screw and healing abutment in a platformswitched implant system. *The Journal of Prosthetic Dentistry* S0022-3913 (21): 00501–00501. [https://doi.](https://doi.org/10.1016/j.prosdent.2021.07.030) [org/10.1016/j.prosdent.2021.07.030.](https://doi.org/10.1016/j.prosdent.2021.07.030)
- **143** Guerra, E., Pereira, C., Faria, R. et al. (2016). The impact of conical and nonconical abutments on bacterial infiltration at the implant-abutment interface. *International Journal of Periodontics and Restorative Dentistry* 36: 825–831.
- **144** Cannata, M., Grandi, T., Samarani, R. et al. (2017). A comparison of two implants with conical vs internal hex connections: 1-year post-loading results from a multicentre, randomised controlled trial. *European Journal of Oral Implantology* 10: 161–168.
- **145** Tripodi, D., D'Ercole, S., Iaculli, F. et al. (2015). Degree of bacterial microleakage at the implant- abutment junction in Cone Morse tapered implants under loaded and unloaded conditions. *Journal of Applied Biomaterials & Functional Materials* 13: 367–371.
- **146** Liu, Y. and Wang, J. (2017). Influences of microgap and micromotion of implant-abutment interface on marginal bone loss around implant neck. *Archives of Oral Biology* 83: 153–160.
- **147** D'Ercole, S., Dotta, T.C., Farani, M. et al. (2022). Bacterial microleakage at the implant-abutment interface: an in vitro study. *Bioengineering (Basel)* 9 (7): 277. [https://doi.org/10.3390/bioengineering9070277.](https://doi.org/10.3390/bioengineering9070277)
- **148** Nascimento, C., Pita, M., Santos Ede, S. et al. (2016). Microbiome of titanium and zirconia dental implants abutments. *Dental Materials* 32: 93–101.
- **149** Teixeira, W., Ribeiro, R., Sato, S., and Pedrazzi, V. (2011). Microleakage into and from two-stage implants: an in vitro comparative study. *The International Journal of Oral & Maxillofacial Implants* 26: 56–62.
- **150** Broggini, N., McManus, L., Hermann, J. et al. (2006). Peri-implant inflammation defined by the implantabutment interface. *Journal of Dental Research* 85: 473–478.
- **151** Goodacre, C., Bernal, G., Rungcharassaeng, K., and Kan, J. (2003). Clinical complications with implants and implant prostheses. *The Journal of Prosthetic Dentistry* 90: 121–132.
- **152** Wolfinger, G. (2003). Implant prosthodontic and restorative complications. *The International Journal of Oral & Maxillofacial Implants* 18: 766–767.
- **153** Calcaterra, R., Di Girolamo, M., Mirisola, C., and Baggi, L. (2016). Effects of repeated screw tightening on implant abutment interfaces in terms of bacterial and yeast leakage in vitro: one-time abutment versus the multiscrewing technique. *International Journal of Periodontics and Restorative Dentistry* 36: 275–280.
- **154** Penarrocha-Oltra, D., Monreal-Bello, A., Penarrocha-Diago, M. et al. (2016). Microbial colonization of the peri-implant sulcus and implant connection of implants restored with cemented versus screw-retained superstructures: a cross-sectional study. *Journal of Periodontology* 87: 1002–1011.
- **155** Steinebrunner, L., Wolfart, S., Bössmann, K., and Kern, M. (2005). In vitro evaluation of bacterial leakage along the implant-abutment interface of different implant systems. *The International Journal of Oral & Maxillofacial Implants* 20: 875–881.
- **156** Harder, S., Dimaczek, B., Açil, Y. et al. (2010). Molecular leakage at implant-abutment connection – in vitro investigation of tightness of internal conical implantabutment connections against endotoxin penetration. *Clinical Oral Investigations* 14: 427–432.
- **157** D'Ercole, S., Scarano, A., Perrotti, V. et al. (2014). Implants with internal hexagon and conical implantabutment connections: an in vitro study of the bacterial contamination. *The Journal of Oral Implantology* 40: 30–36.
- **158** Larrucea, C., Navarro, C., Larrucea, K. et al. (2021). Comparative study of bacterial microfiltration in the implant-abutment interface, with straight and conical

internal connections, in vitro. *Cllinical and Experimental Dental Research* 7: 1014–1024.

- **159** Baggi, L., Di Girolamo, M., Mirisola, C., and Calcaterra, R. (2013). Microbiological evaluation of bacterial and mycotic seal in implant systems with different implantabutment interfaces and closing torque values. *Implant Dentistry* 22: 344–350.
- **160** Cheung, M., Hopcraft, M., and Darby, I. (2021). Dental implant hygiene and maintenance protocols: a survey of oral health practitioners in Australia. *Journal of Dental Hygiene* 95: 25–35.
- **161** Bunk, D., Eisenburger, M., Häckl, S. et al. (2020). The effect of adjuvant oral irrigation on self-administered oral care in the management of peri-implant mucositis: a randomized controlled clinical trial. *Clinical Oral Implants Research* 31: 946–958.
- **162** Rösing, C., Fiorini, T., Haas, A. et al. (2019). The impact of maintenance on peri-implant health. *Brazilian Oral Research* 33 (suppl 1): e074. [https://doi.](https://doi.org/10.1590/1807-3107bor-2019.vol33.0074) [org/10.1590/1807-3107bor-2019.vol33.0074](https://doi.org/10.1590/1807-3107bor-2019.vol33.0074).
- **163** Lin, C., Chen, Z., Pan, W., and Wang, H. (2019). The effect of supportive care in preventing peri-implant diseases and implant loss: a systematic review and meta-analysis. *Clinical Oral Implants Research* 30: 714–724.
- **164** Hatzimanolakis, P., Tsourounakis, I., and Kelekis-Cholakis, A. (2019). Dental implant maintenance for the oral healthcare team. *The Compendium of Continuing Education in Dentistry* 40: 424–429.
- **165** Mombelli, A. (2000). Maintenance therapy for teeth and implants. *Periodontology 2000* 2019 (79): 190–199.
- **166** Göthberg, C., Gröndahl, K., Omar, O. et al. (2018). Bone and soft tissue outcomes, risk factors, and complications of implant-supported prostheses: 5-years RCT with different abutment types and loading protocols. *Clinical Implant Dentistry and Related Research* 20: 313–321.
- **167** Sasada, Y. and Cochran, D. (2017). Implant-abutment connections: a review of biologic consequences and peri-implantitis implications. *The International Journal of Oral & Maxillofacial Implants* 32: 1296–1307.
- **168** Todescan, S., Lavigne, S., and Kelekis-Cholakis, A. (2012). Guidance for the maintenance care of dental implants: clinical review. *Journal of the Canadian Dental Association* 78: c107.
- **169** Bidra, A., Daubert, D., Garcia, L. et al. (2016). A systematic review of recall regimen and maintenance regimen of patients with dental restorations. Part 2: implant-borne restorations. *Journal of Prosthodontics* 25 (Suppl 1): S16–S31.
- **170** Kano, S., Binon, P., and Curtis, D. (2007). A classification system to measure the implant-abutment microgap. *The International Journal of Oral & Maxillofacial Implants* 22: 879–885.
- **171** Hermann, J., Schoolfield, J., Schenk, R. et al. (2001). Influence of the size of the microgap on crestal bone changes around titanium implants. A histometric evaluation of unloaded non-submerged implants in the canine mandible. *Journal of Periodontology* 72: 1372–1383.
- **172** King, G., Hermann, J., Schoolfield, J. et al. (2002). Influence of the size of the microgap on crestal bone levels in non-submerged dental implants: a radiographic study in the canine mandible. *Journal of Periodontology* 73: 1111–1117.
- **173** Tenenbaum, H., Schaaf, J., and Cuisinier, F. (2003). Histological analysis of the Ankylos peri-implant soft tissues in a dog model. *Implant Dentistry* 12: 259–265.
- **174** Piattelli, A., Vrespa, G., Petrone, G. et al. (2003). Role of the microgap between implant and abutment: a retrospective histologic evaluation in monkeys. *Journal of Periodontology* 74: 346–352.
- **175** Covani, U., Marconcini, S., Crespi, R., and Barone, A. (2006). Bacterial plaque colonization around dental implant surfaces. *Implant Dentistry* 15: 298–304.
- **176** Norton, M. (2006). Multiple single-tooth implant restorations in the posterior jaws: maintenance of marginal bone levels with reference to the implantabutment microgap. *The International Journal of Oral & Maxillofacial Implants* 21: 777–784.
- **177** Weng, D., Nagata, M., Bell, M. et al. (2008). Influence of microgap location and configuration on the periimplant bone morphology in submerged implants. An experimental study in dogs. *Clinical Oral Implants Research* 19: 1141–1147.
- **178** Rack, A., Rack, T., Stiller, M. et al. (2010). In vitro synchrotron-based radiography of micro-gap formation at the implant-abutment interface of two-piece dental implants. *Journal of Synchrotron Radiation* 17: 289–294.
- **179** Konermann, A., Zoellner, A., Chang, B., and Wright, R. (2010). In vitro study of the correlation between the simulated clinical and radiographic examination of microgaps at the implant-abutment interface. *Quintessence International* 41: 681–687.
- **180** Rismanchian, M., Hatami, M., Badrian, H. et al. (2012). Evaluation of microgap size and microbial leakage in the connection area of 4 abutments with Straumann (ITI) implant. *The Journal of Oral Implantology* 38: 677–685.
- **181** Duraisamy, R., Krishnan, C., Ramasubramanian, H. et al. (2019). Compatibility of nonoriginal abutments with implants: evaluation of microgap at the implantabutment interface, with original and nonoriginal abutments. *Implant Dentistry* 28: 289–295.
- **182** Rizvi, N., Alyahya, Y., Rizvi, A. et al. (2022). Accuracy of original vs. non-original abutments using various

176 *Considerations of Implant–abutment Connections for the Longevity of Dental Implants*

connection geometries for single unit restorations: a systematic review. *Journal of Prosthodontics* 31: e21–e52.

- **183** Alonso-Pérez, R., Bartolomé, J., and Pradíes, G. (2022). Original vs compatible stock abutment- implant connection: an in vitro analysis of the internal accuracy and mechanical fatigue behaviour. *Journal of Prosthodontic Research* 66: 476–483.
- **184** Weinberg, L. (2001). Therapeutic biomechanics concepts and clinical procedures to reduce implant loading. Part I. *Journal of Oral Implantology* 27: 293–301.
- **185** Yamanishi, Y., Yamaguchi, S., Imazato, S. et al. (2012). Influences of implant neck design and implantabutment joint type on peri-implant bone stress and abutment micromovement: three-dimensional finite element analysis. *Dental Materials* 28: 1126–1133.
- **186** Rack, T., Zabler, S., Rack, A. et al. (2013). An in vitro pilot study of abutment stability during loading in new and fatigue-loaded conical dental implants using synchrotron-based radiography. *The International Journal of Oral & Maxillofacial Implants* 28: 44–50.
- **187** Berberi, A., Maroun, D., Kanj, W. et al. (2016). Micromovement Evaluation of Original and Compatible Abutments at the Implant-abutment Interface. *The Journal of Contemporary Dental Practice* 17: 907–913.
- **188** Messias, A., Rocha, S., Calha, N. et al. (2017). Effect of intentional abutment disconnection on the micromovements of the implant-abutment assembly: a 3D digital image correlation analysis. *Clinical Oral Implants Research* 28: 9–16.
- **189** Apaza-Bedoya, K., Tarce, M., Benfatti, C. et al. (2017). Synergistic interactions between corrosion and wear at titanium-based dental implant connections: a scoping review. *Journal of Periodontal Research* 52: 946–954.
- **190** Dixon, D., Breeding, L., Sadler, J., and McKay, M. (1995). Comparison of screw loosening, rotation, and deflection among three implant designs. *The Journal of Prosthetic Dentistry* 74: 270–278.
- **191** Delgado-Ruiz, R., Calvo-Guirado, J., and Romanos, G. (2019). Effects of occlusal forces on the peri-implantbone interface stability. *Periodontology 2000* 81: 179–193.
- **192** Bakaeen, L., Winkler, S., and Neff, P. (2001). The effect of implant diameter, restoration design, and occlusal table variations on screw loosening of posterior single-tooth implant restorations. *The Journal of Oral Implantology* 27: 63–72.
- **193** Kim, Y., Kim, S., Yun, P. et al. (2010). Prognosis of single molar implants: a retrospective study. *International Journal of Periodontics and Restorative Dentistry* 30: 401–407.
- **194** Schwarz, M.S. (2000). Mechanical complications of dental implants. *Clinical Oral Implants Research* 11 (Suppl 1): 156–158.
- **195** Vinhas, A., Aroso, C., Salazar, F. et al. (2022). In vitro study of preload loss in different implant abutment connection designs. *Materials (Basel)* 15 (4): 1392. <https://doi.org/10.3390/ma15041392>.
- **196** Kourtis, S., Damanaki, M., Kaitatzidou, S. et al. (2017). Loosening of the fixing screw in single implant crowns: predisposing factors, prevention and treatment options. *Journal of Esthetic and Restorative Dentistry* 29: 233–246.
- **197** Chen, J., Wang, L., Yang, L. et al. (2021). The prosthetic screw loosening of two-implant supported screwretained fixed dental prostheses in the posterior region: a retrospective evaluation and finite element analysis. *Journal of Biomechanics* 122: 110423. [https://doi.](https://doi.org/10.1016/j.jbiomech.2021.110423) [org/10.1016/j.jbiomech.2021.110423.](https://doi.org/10.1016/j.jbiomech.2021.110423)
- **198** Katsoulis, J., Takeichi, T., Sol Gaviria, A. et al. (2017). Misfit of implant prostheses and its impact on clinical outcomes. Definition, assessment and a systematic review of the literature. *European Journal of Oral Implantology* 10 (Suppl 1): 121–138.
- **199** Xu, Y., Li, W., and Su, M. (2021). Clinical assessment of preload maintenance in the abutment screws of single posterior implants after 1 month of use. *The International Journal of Oral & Maxillofacial Implants* 36: 177–181.
- **200** Khalili, M., Luke, A., El-Hammali, H. et al. (2019). Efficacy of several retightening protocols for maintaining clamping force in the implant-abutment joint: an in vitro pilot study. *The International Journal of Oral & Maxillofacial Implants* 34: 1084–1090.
- **201** Lee, K., Shin, K., Jung, J. et al. (2020). Clinical study on screw loosening in dental implant prostheses: a 6-year retrospective study. *Journal of the Korean Association of Oral and Maxillofacial Surgeons* 46: 133–142.
- **202** Paolantonio, M., Perinetti, G., D'Ercole, S. et al. (2008). Internal decontamination of dental implants: an in vivo randomized microbiologic 6-month trial on the effects of a chlorhexidine gel. *Journal of Periodontology* 79: 1419–1425.
- **203** D'Ercole, S., Tetè, S., Catamo, G. et al. (2009). Microbiological and biochemical effectiveness of an antiseptic gel on the bacterial contamination of the inner space of dental implants: a 3-month human longitudinal study. *International Journal of Immunopathology and Pharmacology* 22: 1019–1026.
- **204** Podhorsky, A., Biscoping, S., Rehmann, P. et al. (2016). Transfer of bacteria into the internal cavity of dental implants after application of disinfectant or sealant agents in vitro. *The International Journal of Oral & Maxillofacial Implants* 31: 563–570.
- **205** Ozdiler, A., Bakir-Topcuoglu, N., Kulekci, G., and Isik-Ozkol, G. (2018). Effects of taper angle and sealant agents on bacterial leakage along the implant-abutment interface: an in vitro study under loaded conditions. *The International Journal of Oral & Maxillofacial Implants* 33: 1071–1077.
- **206** Alresheedi, B. and Alazmi, S. (2022). Disinfection of implant abutment connection using antimicrobial photodynamic therapy and 0.2% chlorhexidine gel applications immediately before prosthesis delivery: clinical and radiographic status at 1-year of follow-up. *Photodiagnosis and Photodynamic Therapy* 38: 102790. <https://doi.org/10.1016/j.pdpdt.2022.102790>.
- **207** Fletcher, P., Deluiz, D., Tinoco, E. et al. (2017). Human histologic evidence of reosseointegration

around an implant affected with peri-implantitis following decontamination with sterile saline and antiseptics: a case history report. *International Journal of Periodontics and Restorative Dentistry* 37: 499–508.

- **208** Canullo, L., Tallarico, M., Peñarrocha-Oltra, D. et al. (2016). Implant abutment cleaning by plasma of argon: 5-year follow-up of a randomized controlled trial. *Journal of Periodontology* 87: 434–442.
- **209** Canullo, L., Tallarico, M., Chu, S. et al. (2017). Cleaning, disinfection, and sterilization protocols employed for customized implant abutments: an international survey of 100 universities worldwide. *The International Journal of Oral & Maxillofacial Implants* 32: 774–778.

The Role of Bone Regeneration in the Maintenance of Dental Implants

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The number of patients receiving dental implants has rapidly increased since the mid-1960's until present. In 1970, the success of dental implants was around 50%. One of the reasons was the risk of infection [1]. Number of factors have changed since then, allowing for their 10-year survival rate to be around 94.6%, ranging from 73.4 to 100% [2]. Approximately 70% of the implant failures occurred after placement of the abutment and the final restoration. The absence of a periodic preventive maintenance program may affect implant survival rates [2]. It is of paramount importance that patients with periodontal disease should have a stable/healthy periodontium, without flareups, and very low plaque score <10%, before they begin implant treatment [3, 4]. In addition, all the restorations should have cleansable and clear margins [3, 4]. Presence of residual pocket depths ≥5mm and BOP>30% indicates a significant factor for peri-implantitis and subsequent implant loss [4]. The bacteria that colonize the peri-implant tissues are similar to those found in the periodontal tissues [5–8]. *Porphymonas gingivalis, prevotella intermedia,* and *fusobacterium nucleatum* have been associated with the onset of peri-mucositis and peri-implantitis [5–8]. After the placement of the final restorations, the patients must take part in a strict maintenance program [5].

According to the Glossary of Periodontal terms of the American Academy of Periodontology, maintenance procedures are all the procedures performed at selected intervals to assist the patient in maintaining oral health [9]. Maintenance procedures include an update of the medical and dental histories, radiographic review, extraoral and intraoral soft tissue examination, dental examination, periodontal evaluation, removal of the bacterial flora from crevicular and pocket areas, scaling and root planing where indicated, polishing of the teeth, and a review of the patient's plaque control efficacy [9].

Schallhorn and Snider described the four types of maintenance around teeth and implants: (i) preventive, (ii) trial, (iii) compromise, and (iv) post-treatment [10].

- **Preventive periodontal maintenance (PM)**: to prevent inception of peri-implant disease in patients without pathology,
- **Trial PM**: to maintain borderline peri-implant conditions, to assess over time any progression of disease, and the need for further treatment,
- **Compromise PM**: to slow disease progression in patients who would benefit from corrective treatment but are not surgical candidates because of health, economics, inadequate oral hygiene, or other considerations, and
- **Post-treatment PM**: Designed to prevent recurrence of disease after successful corrective therapy. The posttreatment PM is the most applied among patients [11].

The aim of maintenance around dental implants is to minimize the bacterial plaque, to limit the risk of further disease activity that causes continued peri-implant attachment and bone loss, and to ensure the long-term success of dental implant care. It also allows for close monitoring of the patient, so if a recurrence of peri-implant disease takes place, an intervention can be provided [11]. Several different maintenance protocols have been suggested regarding dental implants; (i) initial placement: 6 months, 12 months and then every 2 years, (ii) initial placement: 1, 3, and 5 years if no pathology present, and (iii) initial placement: 6 and 12 months, annually if no pathology present $-$ if pathology present, every 6 months until resolution [12, 14]. Regardless of the suggested protocols, maintenance should be tailored to the specific patient needs [11]. Due to the high volume of implants being placed worldwide, the maintenance is mainly performed by general dentists and dental hygienists [3].

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Patient home care plays a very important role, and it needs to be kept simple. Multiple oral hygiene devices confuse and discourage the patients. When patients applying several toothbrushing techniques, either with manual or electric toothbrushes, miss the most lingual aspects of the implant surface, so this finding should be reinforced at the time of the oral hygiene instructions (OHI) [15]. The use of sonic toothbrush was suggested to be very effective in reduction of bacterial plaque, gingival inflammation, and BOP [16].

An interdental brush has been recommended to massage the gingival tissue around an implant to increase blood flow and enhance the tone of the surrounding gingiva (Figure 9.1). The patient should be instructed to insert the tip interdentally, parallel to the occlusion, allowing the tip to insert the marginal gingiva in a passive way and then apply a rotary motion [17]. Patients to ensure the optimal peri-implant health should maintain daily biofilm removal (up to at least 85%) and be compliant to professional care through periodontal maintenance [18].

The establishment of baseline data before the enrollment of a patient in a maintenance program is another important factor [3]. For implant restorations, a periapical radiograph at the time of the placement of the final crown is a prerequisite, along with initial probing depth as it shows the baseline-reference information to compare with the findings, in future maintenance appointments [3]. Especially, when the implants are placed more than 4mm apically from the adjacent CEJ, they tend to have deeper baseline probing depths (Figure 9.2) [11].

Any bone loss \geq 2mm from the baseline bone level with associated probing depth (PD) ≥ 6 mm and bleeding on probing (BOP) is considered peri-implantitis. In the absence of baseline-reference bone level, ≥3mm bone loss with the associated BOP and PD, are indicative of peri-implantitis [19].

Figure 9.1 Interdental brushes around dental implants in fixed dental prostheses (FDP), inserted in a passive way and parallel to the occlusion, allowing for the patient to have proper oral hygiene.

Figure 9.2 An example of an implant placed more than 4mm subcrestal (5.26mm) from the adjacent restorative CEJ, allowing for an initial deeper PD. In addition, one year after the placement of the final cement-retained restoration, crestal bone loss was noted circumferentially to the first thread. Finally, in addition to the bone loss from the position of the implant, the implant crown has a characteristic misfit (open margins) allowing for a cement-induced peri-implantitis.

The need for keratinized tissue or attached mucosa for the maintenance of dental implants has been controversial. Chung et al. reported that, regardless of the implant surface configuration, the absence of adequate keratinized tissue or attached mucosa was associated with higher plaque scores and gingival inflammation but not with more alveolar bone loss [20]. Romanos et al. are in accordance with the previous findings, showing that absence of keratinized tissue around dental implants, was associated with significant higher plaque scores, bleeding, and recession, compared to a wide band of ≥2mm of keratinized tissue [21].

Patients exhibiting poor plaque control, peri-mucositis and not attending regular maintenance therapy are at higher risk of developing peri-implantitis [22]. The incidence of peri-implantitis was lower in subjects enrolled in a regular maintenance program (18%) compared to patients without regular maintenance care (43%) [23]. Another important factor that does not facilitate the proper oral hygiene and maintenance is the post-restorative presence of submucosal cement, therefore screw-retained restorations are recommended [24]. In addition, the use of angled screw channel abutments allows the design of screwretained restorations even in areas in the anterior zone, which require more angular correction of the final restoration, such as the lateral incisors [25].

Figure 9.4 Buccal periosteal release of 20 mm, on the posterior mandible, using the technique described by Romanos [42].

Several etiological factors, such as developmental defects, congenital missing teeth, tooth extractions without ridge preservation, periodontal disease, odontogenic cysts, tumors, and trauma may lead to Seibert class I, II, or III defects [26, 27]. These defects require rehabilitation with the use of horizontal (HRA) and/or vertical ridge augmentation (VRA) procedures [28–30]. HRA and VRA can be achieved with the use of guided bone regeneration (GBR), the use of bone blocks, or distraction osteogenesis. GBR has become more predictable with less morbidity for the patients over the years, due to advancement of the surgical techniques and the barrier membranes, and the use of

autogenous, allografts, and xenografts [31–38]. A better aesthetic outcome can be achieved with VRA due to more favorable anatomy, along with better access for oral hygiene and maintenance [30, 39, 40]. However, it is a technique-sensitive approach performed by trained and experienced clinicians due to the potential for severe complications [38, 39, 40].

It is of paramount importance to follow the prescribed surgical principles for buccal and lingual flap management, to predictably attain, and maintain primary closure and avoid any complications (Figures 9.3a–c and 9.4) [34, 40–46].During the lingual flap management, severe complications may occur including the risk of damaging the lingual nerve, the sublingual artery, the Wharton's duct, the sublingual and submandibular glands, and perforation/over-thinning of the lingual flap [42, 44, 45, 47]. These events may result in hematoma which can be a lifethreatening complication due to possible respiratory obstruction [42, 44–49].

The rate of complications, especially for the VRA procedures, range between 2.78 and 17%, and include, but are not limited to, membrane exposure and post-operative site infection. Sites exhibiting primary exposure gained only 65% of the anticipated bone height compared with sites without membrane exposure. Cases with late exposures, compared to early exposures (<four weeks postoperatively), showed sufficient bone consolidation, which allowed for measurable bone regeneration [50, 51]. Defect morphology plays an additional significant role in the regenerative potential; wider and flat ridges are better candidates for VRA due to better blood supply, stability of the grafting materials and the numerous growth factors acting as chemoattractant and powerful modifiers of the healing

Figures 9.3 (a–c) Mylohyoid muscle release on the lingual aspect of the posterior mandible, ranging from 25 to 29 mm, using the finger sweep technique.

process [30, 52–55]. Finally, proper case selection and execution of the treatment plan, strict post-operative protocol, and appropriate implant loading can ensure the success of ridge augmentation procedures [50, 56].

However, due to the aforementioned parameters, in several cases, it is preferred the option of short implants, placement of implants where the alveolar bone allows, placement of implants in adjacent sites, or the use of pink ceramic to mimic the normal soft tissue anatomy [56]. Restorative therapies employing short implants (<7mm) are well documented in the literature. Reduced costs and treatment times, fewer surgical complications with similar treatment outcomes as longer implants, provide justification as alternative treatments to ridge augmentation procedures [57–59]. Although short implants do not demonstrate more crestal bone loss per se, compared to standard length implants, by default there is a lower percentage of bone-to-implant contact. Therefore, crestal bone loss or bone remodeling around the implant platform of a short implant, might be detrimental for the long-term survival rate $[60]$. The use of ultrashort implants (<5mm) needs further investigation, before the clinical application on a regular basis [61, 62].

There is no statistically significant difference between implants placed in pristine native bone compared to augmented sites both at patient and implant levels [39]. Minimum buccal bone thickness of 2mm is required for the placement of implants in the anterior region, and 1.5–2mm for the posterior region [63]. The interproximal distances between teeth and implants, adjacent implants and crestal bone, to the contact point should be followed in order to have harmonious restorations that allow for proper cleansibility and maintenance [64]. Ideally, between adjacent implants, the distance in a vertical dimension, from the contact point to the crestal bone is 3 and 3–5mm between a tooth and an implant. In a horizontal dimension, 3–4mm distance is critical between either teeth and implants or between implants [63, 64]. Generally, implants are placed around 3–4mm apical to the neighboring/restorative CEJ (Figures 9.5a, b, 9.6a–d, 9.7, 9.8a, b, 9.9a–c, 9.10, 9.11a–c, 9.12–9.14, 9.15a, b, 9.16a, b, and 9.17a–c) [35].

Unfortunately, most of the locations used for placement of dental implants, are not in the ideal prosthetic position, when the distance from the neighboring CEJ is more than 4mm, creating several problems such as lack of proper cleansability of the final restoration, alveolar/crestal bone loss in the proximal areas, high crown to implant ratio, and lack of proper distribution of the occlusal forces. Specifically, when implants are placed apically more than 4mm from the restorative CEJ, significantly greater bone loss on the mesial and distal surfaces was noted, compared to implants placed within 3mm from the neighboring CEJ (Figures 9.18a–d).

Bone loss around implants is the most important etiological factor that may lead to implant failure. In restorations with high crown-to-implant ratio, the occlusal stress is distributed around the implant platform, which may cause implant failure. Ideally, all the occlusal discrepancies and parafunctional habits should be addressed before the beginning of the implant treatment [65–71].

Other reasons causing crestal bone loss around an implant are: (i) iatrogenic factors, such as poor surgical skills, (ii) early exposure of a submerged implant, (iii) high temperatures during the preparation of the osteotomy, and (iv) compression necrosis during a high-torque implant

Figure 9.5 (a, b) Clinical and radiographic view of tooth #9 showing pathologic tooth migration, Miller class III recession, moderate interproximal bone loss, mobility class II, 7mm probing depth on the mesial aspect and 6mm on the distal aspect, external root resorption and composite restorations, on the cervical aspect and the incisal edge. The prognosis of the tooth was hopeless.

Figure 9.6 (a, b) Upon reflection of a papilla preservation flap and after the extraction of #9, a fenestration was noted in the most apical part of the socket. Horizontal and vertical ridge augmentation was performed with amnion-chorion resorbable membrane along with 50:50 mixture of mineralized cortical allograft and deproteinized bovine bone. The technique used is described by Soldatos et al. [35]. (c, d) CBCT before and after the extraction and ridge augmentation of #9, showing the area of external resorption and a very thin buccal plate. Nine months post-operatively, horizontal and ridge augmentation was achieved, allowing for placement of a dental implant.

placement (Figures 9.19a–d, 9.20a, b, 9.21a–c, 9.22, 9.23a, b) [72, 73]. This type of crestal bone loss is usually seen before the placement of the healing abutment [60].

Furthermore, the implant design and rough surface might play a role in crestal bone level [74–77]. Huang et al. supported the sealing ability of the tapered implant connection, minimizing the bacterial penetration at the implant–abutment surface and the crestal bone loss [74].

Microbiological studies compared butt-joint with Morse tapered connections in vivo with patients having implants with the different types of implant–abutment geometries and found a bacterial sealing in the Morse-tapered connections [78].

All these factors and implant design characteristics are fundamental to create a crestal bone stability and improve clinical outcomes over time [79].

Figure 9.6 (Continued)

Figure 9.7 A periapical radiograph at the area of #9, after the placement of a tapered bone level dental implant within 3mm of the neighboring CEJs.

Figures 9.8 (a, b) A periapical radiograph and clinical view of implant #9, three (3) months after the placement of the single cement-retained final restoration, allowing for proper esthetics, oral hygiene, and periodontal maintenance.

Figures 9.9 (a–c) Pre-operative clinical and CBCT view of areas #3,4 showing Siebert class I defect.

Figure 9.10 Upon reflection of the full thickness flap, Siebert class I defect was noted at the area of #3, 4. Flap management was performed with buccal periosteal release using the technique described by Romanos [42].

(a) (b) (c)

Figures 9.11 (a–c) Two (2) tenting screws (4 mm height) were placed for space maintenance, cortical perforations (using #8 finishing round bur) and two cross-linked resorbable membranes (sutured together with 4-0 chromic gut sutures). The membranes were placed initially on the palatal aspect and then after the placement of the bone grafting material, on the buccal aspect. The technique applied is described by Soldatos et al. [35].

Figure 9.12 Horizontal ridge augmentation was performed with 50:50 mixture of mineralized cortical allograft and deproteinized bovine bone (large particles) placed over the tenting screws at the area of #3, 4.

Figure 9.13 Suturing the flaps in a connective tissue to connective tissue approach, with 3 horizontal mattress and 13 simple interrupted PTFE sutures. Tension-free primary closure was achieved and maintained.

Figure 9.14 Nine (9) months post-operative CBCT showing 3–4.7mm horizontal ridge augmentation. At the time of the placement of dental implants, internal sinus elevation is required at the area of #3.

Figures 9.15 (a, b) Buccal and occlusal post-operative view, nine months after the horizontal ridge augmentation, showing the underlying new bone formation.

Figures 9.16 (a, b) Buccal and occlusal view, upon reflection of the full thickness flap, showing new bone formation at the level of the head of the tenting screws.

Figure 9.17 (a) Two implants (areas #3, 4) were placed (a) with simultaneous internal sinus elevation (area of #3). The primary stability of the implants was ≥40 N/cm allowing for the placement of healing abutments. (b, c) Final radiographic and clinical view of the screw retained independent final restorations within 3 mm of the neighboring restorative CEJs. The design of the final restorations allows for proper esthetics, oral hygiene, and periodontal maintenance.

Figure 9.17 (Continued)

Figure 9.18 (a–d) Implants at the areas of #24 and 9, placed apically more than 3 mm from the neighboring restorative CEJ, presenting crestal bone loss after the placement of the final restoration. In both cases, the implants should be removed, and vertical ridge augmentation should be performed, allowing for a better new implant placement.

Figure 9.19 (a) Virtual implant planning for the areas #12–14. The three implants were planned to be placed in different levels allowing for restorative CEJ discrepancies which will lead to crestal bone loss along with difficulty in oral hygiene. (b–d) Intraoperative buccal and palatal view of implants #12–14. The implants' position (#12–14) was corrected during the surgical intervention, placing them at a similar restorative level. In addition, vertical ridge augmentation was performed around all three implants, to restore the Seibert class II defect.

Figure 9.19 (Continued)

Figures 9.20 (a, b) At the area of #29, an implant was placed without the proper digital planning in a non-restorable prosthetic position. In addition, the implant had 4mm crestal bone loss before the second stage (placement of the healing abutment). The implant was removed, and horizontal and vertical ridge augmentation was performed. The red line shows the amount of bone that must be regenerated in order to restore the proper alveolar bone level.

Figure 9.21 (a–c) Horizontal and vertical ridge augmentation was performed at the area of #29 with 2 tenting screws (5mm height), cortical perforations with #8 round bur, 50: 50 mixture of mineralized cortical allograft with deproteinized bovine bone. Two membranes were used, a cross-linked resorbable and a high density PTFE non-resorbable membrane. The site was sutured with a horizontal mattress and eight single interrupted PTFE sutures. The technique applied is described by Soldatos et al. [37].

(c)

Figure 9.21 (Continued)

Figure 9.22 Nine months post-operatively, where clinically, a wide band of keratinized tissue is seen along with new bone formation in a vertical level.

Figures 9.23 (a, b) Nine months post-operatively, 5mm of vertical ridge augmentation was achieved at the site #29, allowing for the placement of a new implant in the ideal restorative position and level compared to neighboring CEJs.

References

- **1** Albrektsson, T. and Wennerberg, A. (2005). The impact of oral implants – past and future, 1966–2042. *Journal of the Canadian Dental Association* 71: 327.
- **2** Moraschini, V., Poubel, L.A.d.C., Ferreira, V.F., and Barboza, E.d.S.P. (2015). Evaluation of survival and success rates of dental implants reported in longitudinal studies with a follow-up period of at least 10 years: a systematic review. *International Journal of Oral and Maxillofacial Surgery* 44: 377–388.
- **3** Chen, S. and Darby, I. (2003). Dental implants: Maintenance, care, and treatment of peri-implant infection. *Australian Dental Journal* 48: 212–220.
- **4** Pjetursson, B.E., Helbling, C., Weber, H. et al. (2012). Peri-implantitis susceptibility as it relates to periodontal therapy and supportive care. *Clinical Oral Implants Research* 23: 888–894.
- **5** Leonhardt, A., Grondahl, K., Bergstrom, C., and Lekholm, U. (2002). Long-term follow-up of osseointegrated titanium implants using clinical, radiographic, and microbiological parameters. *Clinical Oral Implants Research* 13: 127–132.
- **6** Weyant, R.J. and Burt, B.A. (1993). An assessment of survival rates and with in-patient clustering of failures for endosseous oral implants. *Journal of Dental Research* 72: 2–8.

- **7** Tonetti, M.S. and Schmid, J. (2000). Pathogenesis of implant failures. *Periodontology* 1994 (4): 127–138.
- **8** Pontoriero, R., Tonelli, M.P., Carnevale, G. et al. (1994). Experimentally induced peri-implant mucositis. A clinical study in humans. *Clinical Oral Implants Research* 5: 254–259.
- **9** American Academy of Periodontology (2022). Glossary of periodontal terms. perio.org (accessed November 2022).
- **10** Schallhorn, R.G. and Snider, L.E. (1981). Periodontal maintenance therapy. *Journal of the American Dental Association (1939)* 103: 227–231.
- **11** Shumaker, N., Metcalf, B., Toscano, N., and Holtzclaw, D. (2009). Periodontal and peri-implant maintenance: a critical factor in long-term treatment success. *Compendium* 30: 388–390.
- **12** Mombelli, A. and Lang, N.P. (1998). The diagnosis and treatment of peri–implantitis. *Periodontology 2000* 17: 63–76
- **13** Humphrey, S. (2006). Implant maintenance. *Dental Clinics of North America* 50: 463–478.
- **14** Wyatt, C.C. and Pharoah, M.J. (1998). Imaging techniques and image interpretation for dental implant treatment. *The International Journal of Prosthodontics* 11: 442–452.
- **15** Rasmussan, R.A. (1992). *The Branemark System of Oral Reconstruction: A Color Atlas*. Kyiyaku EuroAmerica, Inc.
- **16** Wolff, L., Kim, A., Nunn, M. et al. (1998). Effectiveness of a sonic toothbrush in maintenance of dental implants. A prospective study. *Journal of Clinical Periodontology* 25: 821–828.
- **17** Biesbrock, A., Bartizek, R., Gerlach, R., and Terezhalmy, G. (2007). Oral hygiene regimens, plaque control, and gingival health: a two-month clinical trial with antimicrobial agents. *The Journal of Clinical Dentistry* 18: 103–107.
- **18** Misch, C.E. (2005). *Dental Implant Prosthetics*. St. Louis, Mo: Mosby.
- **19** Renvert, S., Persson, G.R., Pirih, F.Q., and Camargo, P.M. (2018). Peri-implant health, peri-implant mucositis, and peri-implantitis: case definitions and diagnostic considerations. *Journal of Periodontology* 89: 304–312.
- **20** Chung, D.M., Oh, T.-J., Shotwell, J.L. et al. (2006). Significance of keratinized mucosa in maintenance of dental implants with different surfaces. *Journal of Periodontology* 77: 1410–1420.
- **21** Romanos, G., Grizas, E., and Nentwig, G.H. (2015). Association of keratinized mucosa and peri-implant soft tissue stability around implants with platform switching. *Implant Dentistry* 24: 422–426.
- **22** Costa, F.O., Takenaka-Martinez, S., Cota, L.O.O. et al. (2012). Peri-implant disease in subjects with and with-out preventive maintenance: a 5-year follow-up. *Journal of Clinical Periodontology* 39: 173–181.
- **23** Schwarz, F., Derks, J., Monje, A., and Wang, H.-L. (2018). Peri-implantitis. *Journal of Periodontology* 89: 267–290.
- **24** Berglundh, T., Armitage, G., Araujo, M. et al. (2018). 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* 89: 313–318.
- **25** Edmondson, K., Trejo, P., Soldatos, N., and Weltman, R. (2021). The ability to screw-retain single implantsupported restorations in the anterior maxilla: a CBCT analysis. *The Journal of Prosthetic Dentistry* 17: 0022-3913(21)00032-9. [https://doi.org/10.1016/j.](https://doi.org/10.1016/j.prosdent.2021.01.004) [prosdent.2021.01.004.](https://doi.org/10.1016/j.prosdent.2021.01.004) Online ahead of print.
- **26** Seibert, J.S. (1983). Reconstruction of deformed, partially edentulous ridges, using full thickness onlay grafts. Part I. Technique and wound healing. *The Compendium of Continuing Education in Dentistry* 4: 437–453.
- **27** Wang, H.L. and Al-Shammari, K. (2002). HVC ridge deficiency classification: a therapeutically oriented classification. *International Journal of Periodontics and Restorative Dentistry* 22: 335–343.
- **28** Urban, I., Monje, A., Lozada, J., and Wang, H.L. (2015). Vertical ridge augmentation and soft tissue reconstruction of the anterior atrophic maxillae: a case series. *International Journal of Periodontics and Restorative Dentistry* 35: 613–623.
- **29** Schropp, L., Wenzel, A., Kostopoulos, L., and Karring, T. (2003). Bone healing and soft tissue contour changes following single-tooth extraction: a clinical and radiographic 12-month prospective study. *International Journal of Periodontics and Restorative Dentistry* 23: 313–323.
- **30** Soldatos, N. and Weltman, R. (2022). Vertical ridge augmentation (VRA): success and predictability. *Current Oral Health Reports* [https://doi.org/10.1007/](https://doi.org/10.1007/s40496-022-00318-9) [s40496-022-00318-9](https://doi.org/10.1007/s40496-022-00318-9).
- **31** Dahlin, C., Linde, A., Gottlow, J., and Nyman, S. (1988). Healing of bone defects by guided tissue regeneration. *Plastic and Reconstructive Surgery* 81: 672–676.
- **32** Urban, I., Monje, A., Lozada, J., and Wang, H.-L. (2017). Principles for vertical ridge augmentation in the atrophic posterior mandible: a technical review. *International Journal of Periodontics and Restorative Dentistry* 37: 639–645.
- **33** Simion, M., Jovanovic, S.A., Trisi, P. et al. (1998). Vertical ridge augmentation around dental implants using a membrane technique and autogenous bone or allografts in humans. *International Journal of Periodontics and Restorative Dentistry* 18: 8–23.

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- **34** Simion, M., Jovanovic, S.A., Tinti, C., and Benfenati, S. (2001). Long term evaluation of osseointegrated implants inserted at the time or after vertical ridge augmentation. A retrospective study on 123 implants with 1-5-year follow-up. *Clinical Oral Implants Research* 12: 35–45.
- **35** Soldatos, N., Al ramli, R., Nelson-Rabe, L. et al. (2021). Vertical ridge augmentation (VRA) with the use of cross-linked resorbable membrane, tenting screws, and a combination grafting technique: a report of three cases. *Quintessence International* 52: 328–339.
- **36** Soldatos, N., Stylianou, P., Koidou, V. et al. (2017). Limitations and options using resorbable versus resorbable membranes for successful guided bone regeneration procedures. *Quintessence International* 48: 131–147.
- **37** Soldatos, N., Garcia, M., Umoh, E. et al. (2022). Vertical ridge augmentation around dental implants with the use of a dense PTFE membrane to correct previously failed augmentations. *Clinical Advances in Periodontics* 12: 51–56.
- **38** Jepsen, S., Schwarz, F., Cordaro, L. et al. (2019). Regeneration of alveolar ridge defects. Consensus report of group 4 of the 15th European workshop on periodontology on bone regeneration. *Journal of Clinical Periodontology* 46: 277–286.
- **39** Salvi, G.E., Monje, A., and Tomasi, C. (2018). Long-term biological complications of dental implants placed either in pristine or in augmented sites: a systematic review and meta-analysis. *Clinical Oral Implants Research* 29: 294–310.
- **40** Urban, I., Monje, A., Wang, H.-L. et al. (2017). Mandibular regional anatomical landmarks and clinical implications for ridge augmentation. *International Journal of Periodontics and Restorative Dentistry* 37: 347–353.
- **41** Pikos, M.A. (2005). Atrophic posterior maxilla and mandible: alveolar ridge reconstruction with mandibular block autografts. *The Alpha Omegan* 98: 34–45.
- **42** Romanos, G.E. (2010). Periosteal releasing incision for successful coverage of augmented sites. A technical note. *The Journal of Oral Implantology* 36: 25–30.
- **43** Urban, I., Traxler, H., Romero-Bustillos et al. (2018). Effectiveness of two different lingual flap advancing techniques for vertical bone augmentation in the posterior mandible: a comparative, split-mouth cadaver study. *International Journal of Periodontics and Restorative Dentistry* 38: 35–40.
- **44** Ronda, M. and Stacchi, C. (2011). Management of coronally advanced lingual flap in regenerative osseous surgery: a case series introducing a novel technique.

International Journal of Periodontics and Restorative Dentistry 31: 505–513.

- **45** Ronda, M., Rebaudi, A., Torelli, L., and Stacchi, C. (2014). Expanded vs. dense polytetrafluoroethylene membranes in vertical ridge augmentation around dental implants: a prospective randomized controlled clinical trial. *Clinical Oral Implants Research* 25: 859–866.
- **46** Simion, M., Trisi, P., and Piattelli, A. (1994). Vertical ridge augmentation using a membrane technique associated with osseointegrated implants. *International Journal of Periodontics and Restorative Dentistry* 14: 496–511.
- **47** Urban, I., Lozada, J.L., Jovanovic, S.A. et al. (2014). Vertical ridge augmentation with titanium- reinforced dense PTFE membranes and a combination of particulate autogenous bone and anorganic bovine bone-derived mineral: a prospective case series in 19 patients. *The International Journal of Oral & Maxillofacial Implants* 29: 185–193.
- **48** Niamtu, J. 3rd. (2001). Near-fatal airway obstruction after routine implant placement. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics* 92: 597–600.
- **49** Soldatos, N., Immonen, J., Romanos, G., and Weltman, R. (2023). The significance of mylohyoid muscle release (MMR) in the vertical and horizontal ridge augmentation surgeries. *Genes (Basel)* 14 (3): 595. doi: 10.3390/genes14030595.
- **50** Urban, I., Montero, E., Monje, A., and Sanz-Sanchez, I. (2019). Effectiveness of vertical ridge augmentation interventions: a systematic review and meta-analysis. *Journal of Clinical Periodontology* 46: 319–339.
- **51** Tay, J.R.H., Ng, E., Lu, X.J., and Lai, W.M.C. (2022). Healing complications and their detrimental effects on bone gain in vertical-guided bone regeneration: a systematic review and meta-analysis. *Clinical Implant Dentistry and Related Research* 24: 43–71.
- **52** Lindhe, A., Thoren, C., Dahlin, C., and Sandberg, E. (1993). Creation of new bone by an osteopromotive membrane technique: an experimental study in rats. *Journal of Oral and Maxillofacial Surgery* 51: 892–889.
- **53** Schmid, J., Hammerle, C.H.F., Stich, H., and Lang, N.P. (1991). Supraplant, a novel implant system based on the principle of guided bone regeneration, A preliminary study in the rabbit. *Clinical Oral Implants Research* 2: 199–202.
- **54** Jovanovic, S., Schenk, R., Orsini, M., and Kenney, E.B. (1995). Supracrestal bone formation around dental implants: an experimental dog study. *The International Journal of Oral & Maxillofacial Implants* 10: 23–31.
- **55** Garaicoa, C., Suarez, F., Fu, J.H. et al. (2015). Using cone beam computed tomography angle for predicting the outcome of horizontal bone augmentation. *Clinical Implant Dentistry and Related Research* 17: 717–723.
- **56** Urban, I., Klokkevold, P., and Cochran, D. (2015). Advanced implant surgical procedures. In: *Carranza's Clinical Periodontology*, 12e, chapter 77, 750–763. Elsevier.
- **57** Esposito, M., Barausse, C., Pistilli, R. et al. (2019). Posterior atrophic jaws rehabilitated with prostheses supported by 5×5 mm implants with a nanostructured calcium-incorporated titanium surface or by longer implants in augmented bone. Five-year results from a randomized controlled trial. *The International Journal of Oral Implantology* 12: 39–54.
- **58** Pieri, F., Forlivesi, C., Caselli, E., and Corinaldesi, G. (2017). Short implants (6 mm) vs. vertical bone augmentation and standard-length implants (9 mm) in atrophic posterior mandibles: a 5-year retrospective study. *International Journal of Oral and Maxillofacial Surgery* 46: 1607–1614.
- **58** Terheyden, H., Meijer, G.J., and Raghoebar, G.M. (2021). Vertical bone augmentation and regular implants versus short implants in the vertically deficient posterior mandible: a systematic review and metaanalysis of randomized studies. *International Journal of Oral and Maxillofacial Surgery* 50: 1249–1258.
- **60** Linkevicious, T. (2019). *Zero Bone Loss Concepts*, chapter 1., 3–15. Quintessence Publishing.
- **61** Barausse, C., Piattelli, A., and Del Fabbro, M. (2020). Are <7-mm long implants in native bone as effective as longer implants in augmented bone for the rehabilitation of posterior atrophic jaws? A systematic review and meta-analysis. *Clinical Implant Dentistry and Related Research* 22: 552–566.
- **62** Barausse, C., Pistilli, R., Canullo, L. et al. (2022). A 5-year randomized controlled clinical trial comparing 4-mm ultrashort to longer implants placed in regenerated bone in the posterior atrophic jaw. *Clinical Implant Dentistry and Related Research* 24: 4–12.
- **63** Su, C.Y., Fu, J.H., and Wang, H.L. (2014). The role of implant position on long-term success. *Clinical Advances in Periodontics* 4: 187–193.
- **64** Gastaldo, J.F., Cury, P.R., and Sendyk, W.R. (2004). Effect of the vertical and horizontal distances between adjacent implants and between a tooth and an implant on the incidence of interproximal papilla. *Journal of Periodontology* 75: 1242–1246.
- **65** Mailoa, J., Fu, J.H., Chan, H.L. et al. (2015). The effect of vertical implant position in relation to adjacent teeth on marginal bone loss in posterior arches: a retrospective study. *International Journal of Oral & Maxillofacial Implants* 30: 931–936.
- **66** Hermann, J.S., Buser, D., Schenk, R.K., and Cochran, D.L. (2000). Crestal bone changes around titanium implants. A histometric evaluation of unloaded nonsubmerged and submerged implants in the canine mandible. *Journal of Periodontology* 71: 1412–1424.
- **67** Pontes, A.E., Ribeiro, F.S., da Silva, V.C. et al. (2008). Clinical and radiographic changes around dental implants inserted in different levels in relation to the crestal bone, under different restoration protocols, in the dog model. *Journal of Periodontology* 79: 486–494.
- **68** Cardaropoli, G., Wennstrom, J.L., and Lekholm, U. (2003). Peri-implant bone alterations in relation to inter-unit distances. A 3-year retrospective study. *Clinical Oral Implants Research* 14: 430–436.
- **69** Chang, M., Chronopoulos, V., and Mattheos, N. (2013). Impact of excessive occlusal load on successfullyosseointegrated dental implants: a literature review. *Journal of Investigative and Clinical Dentistry* 4: 142–150.
- **70** Zitzmann, N.U. and Berglundh, T. (2008). Definition and prevalence of peri-implant diseases. *Journal of Clinical Periodontology* 35: 286–291.
- **71** Roos-Jansaker, A.M., Lindahl, C., Renvert, H., and Renvert, S. (2006). Nine- to fourteen-year follow-up of implant treatment. Part II: presence of peri-implant lesions. *Journal of Clinical Periodontology* 33: 290–295.
- **72** Soldatos, N., Nelson-Rabe, L., Palanker, N. et al. (2022). Temperature changes during implant osteotomy preparations in fresh human cadaver tibiae, comparing straight with tapered drills. *Materials* 15: 2369.
- **73** Soldatos, N., Pham, H., Fakhouri, W. et al. (2022). Temperature changes during implant osteotomy preparations in human cadaver tibiae comparing MIS® straight drills with Densah® burs. *Genes* 10: 1716.
- **74** Huang, B., Meng, H., Piao, M. et al. (2012). Influence of placement depth on bone remodeling around tapered internal connection implant: a clinical and radiographic study in dogs. *Journal of Periodontology* 83: 1164–1171.
- **75** Norton, M.R. (1998). Marginal bone levels at single tooth implants with a conical fixture design. The influence of surface macro- and microstructure. *Clinical Oral Implants Research* 9: 91–99.
- **76** Garaicoa-Pazmino, C., Suarez-Lopez del Amo, F., Monje, A. et al. (2014). Influence of crown/implant ratio on marginal bone loss: a systematic review. *Journal of Periodontology* 85: 1214–1221.
- **77** Romanos, G.E., Delgado-Ruiz, R., and Sculean, A. (2019). Concepts for prevention of complications in implant therapy. *Periodontology 2000* 81: 7–17.
- **78** Romanos, G.E., Biltucci, M.T., Kokaras, A., and Paster, B.J. (2016). Bacterial composition at the implantabutment connection under loading in vivo. *Clinical Implant Dentistry and Related Research* 18: 138–145.
- **79** Romanos, G.E. (2015). Tissue preservation strategies for fostering long-term soft and hard tissue stability. *International Journal of Periodontics and Restorative Dentistry* 35: 363–371.

10

Soft Tissue Around Implants to Maintain/Reestablish Peri-implant Tissue Health

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Over the past few decades, the replacement of missing teeth with dental implants has become a standard procedure due to its high predictability [1].

However, the incidence of biological peri-implant complications, such as peri-implant mucositis (PIM) and peri-implantitis, has been increasing reaching up to 47% [2, 3]. These complications present a growing global health problem and pose challenges with unpredictable treatment outcomes. As a result, there has been a shift in clinicians' decision-making towards a more conservative treatment approach, where questionable teeth are treated and implant placement is postponed [4].

According to the current classification of periodontal and peri-implant diseases, PIM is defined as an inflammatory condition of the peri-implant mucosa without accompanying marginal bone loss [5]. The main cause of PIM is an imbalance in the host–microbial homeostasis at the mucosa–implant interface. Clinically, PIM is characterized by inflammation, redness, swelling, bleeding on gentle probing, and sometimes suppuration. Elevated peri-implant probing depths may also be observed [6]. With appropriate treatment leading to inflammation resolution, PIM is reversible. However, if left untreated, it may progress to peri-implantitis.

Peri-implantitis is an irreversible pathological condition primarily caused by the accumulation of biofilm and is clinically characterized by inflammation, bleeding or suppuration on probing, swelling, increased peri-implant probing depths, radiographic evidence of supporting bone loss, and sometimes mucosal recessions [6]. Risk factors for its onset and/or progression include a history of severe periodontitis, poor plaque control, irregular peri-implant maintenance therapy, smoking, diabetes mellitus, limited access to oral hygiene, improper implant positioning or prosthetic suprastructure, and the presence of submucosal cementation material [7]. Additionally, factors such as the absence of keratinized/attached peri-implant mucosa, surgical aspects (bone compression necrosis and overheating), micromotion, biocorrosion, and occlusal overload have been proposed as influencing the development of peri-implant pathological conditions [7]. Peri-implantitis may manifest shortly after prosthetic restoration and, if left untreated, may progress more rapidly than periodontitis, leading to implant loss [6, 7].

However, the quantity and characteristics of peri-implant tissue play a crucial role in maintaining peri-implant health and achieving successful esthetic outcomes. These aspects are encompassed by the peri-implant phenotype, which refers to the morphological and dimensional features characterizing the clinical presentation of the tissues surrounding and supporting osseointegrated implants, including the soft tissues (such as keratinized mucosa (KM) width, mucosal thickness, and supracrestal tissue height) and peri-implant bone thickness [8]. Like the periodontal phenotype, the peri-implant phenotype is site-specific, and irreversible changes can occur due to environmental factors or clinical interventions.

Soft-tissue Issues Around Dental Implants

Keratinized Tissue Width

Ensuring the long-term success of dental implants heavily relies on maintaining healthy peri-implant tissues. However, the role of peri-implant soft tissue and the

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necessity of having a minimal amount of keratinized mucosa around dental implants have been a subject of controversy in the literature [9–12].

Different studies have presented conflicting views on the importance of the width of keratinized mucosa (KMW) around dental implants. Some researchers have suggested that a KMW of less than 2mm is associated with increased inflammation and plaque accumulation compared to sites with a KMW of 2mm or greater [13, 14]. However, other studies have failed to establish a clear association between the lack of a minimum width of keratinized mucosa and inflammation [15–18].

A recent meta-analysis reported significantly lower plaque scores, bleeding indices, mucosal recessions, and attachment loss for sites with wider keratinized tissue (KT) [14]. In an observational study involving 110 implants and 36 patients, significantly more plaque accumulation, peri-implant inflammation, mucosal recession, and bleeding were reported after six months at sites with a KMW of less than 2mm [19]. Ueno and colleagues also observed that implants with a KMW of less than 2mm exhibited significantly higher plaque accumulation, bleeding on probing, and pocket depths compared to sites with a KMW of 2mm or greater [19]. Similar findings, with statistically significantly higher plaque levels, bleeding, and patient discomfort, were reported by Souza et al. for implants with less than 2mm of keratinized mucosa [20].

Even when patients were highly motivated and able to maintain long-term low full-mouth plaque scores for over 10 years, significantly higher plaque accumulation occurred at sites with insufficient keratinized mucosa [21]. In another long-term observational study spanning eight years, implants with a KMW of less than 2mm showed significantly higher bleeding and plaque scores than sites with sufficient keratinized tissue width (KTW) [22].

Some authors have observed a positive association between the lack of a minimal band $\left($ <2mm $\right)$ of keratinized mucosa and the occurrence of PIM [23] as well as a higher prevalence of PIM in such cases [24–27]. However, other studies have found no association [18, 28, 29].

The width of sufficient KT also appears to have an impact on diseased implants, specifically peri-implantitis. In a cross-sectional study involving 534 patients, 53 (10.3%) were affected by peri-implantitis. These implants showed significantly more plaque accumulation and bleeding on probing and were more likely to have an attached mucosa width of less than 2mm [30].

From a clinical standpoint, it is important to highlight that when the peri-implant KT is less than 2mm, there is greater plaque accumulation, bleeding on probing, and more gingival recessions are diagnosed around these implants, particularly in cases with irregular maintenance

protocols. However, these differences are not statistically significant when patients have regular recall appointments with professional cleaning every six months, regardless of whether the KTW is less than 2mm or greater than 2mm [31]. This clinical study involved 118 patients with 320 dental implants utilizing platform switching.

A recent study has examined the natural regeneration of the implanto–mucosal and dento-gingival units following the complete removal of KT [32]. The results of the study revealed that after the excision of KT at implant sites, the spontaneous regeneration of the soft tissue is characterized by a non-keratinized epithelium similar to alveolar mucosa. On the other hand, at tooth sites, the spontaneous regeneration resulted in soft tissue resembling gingiva. These findings are clinically significant as they demonstrate, for the first time, that once KT is lost at implant sites, it does not regenerate naturally. Therefore, the restoration of KT can only be achieved through soft tissue grafting (Figure 10.1a–h).

Mucosal Thickness

In recent years, emerging evidence supports the idea that peri-implant mucosal thickness represents an important factor in the maintenance of peri-implant health. Historically, soft tissue thickness has been shown to play a critical role in peri-implant marginal bone loss [33]. Greater stability of the interproximal bone in cases with adequate mucosal thickness has been reported in a recent systematic review [34]. Nonetheless, the adequate amount for tissue thickness has not been clearly defined yet [35], but a threshold of 2mm thickness has been proposed [8]. The assessment of peri-implant mucosal thickness may vary depending on the implant location (e.g. lingual or buccal) and is commonly determined as 1–2mm apical to the mucosal margin [8]. Most studies assessing horizontal mucosal thickness focused on esthetic outcomes in relation to the transparency of the abutment material [36–38], and a minimum thickness of about 2mm is necessary for masking restorative materials [37, 39]. However, a thickness >1mm was reported as beneficial against mucosal recessions following immediate implant placement [40, 41]. On the other hand, augmentation of mucosal thickness has been proposed for compensating expected bone remodeling after immediate implant placement and for stabilizing the mucosal margin [42–45].

Moreover, tissue thickness influences majorly esthetic outcomes [43]. Recent evidence indicates that a thick mucosa assures a more stable mucosal margin as compared to a thinner one [43, 45]. Thin mucosa is also considered an etiological factor for the development of peri-implant mucosal recessions [41, 46].

(e)

 (g) (h)

Figure 10.1 (a) In this clinical scenario, a single-unit crown (SUC) was placed on a 4.1mm regular neck tissue level implant in the area of tooth #46. However, there was a noticeable absence of attached keratinized mucosa (KM). The patient also reported difficulties in maintaining proper oral hygiene. To address these issues, the patient was treated with a peri-implant free gingival graft (FGG) procedure. The goal of this treatment is to enhance both the quantity and quality of the soft tissue surrounding the implant. (b) The single-unit crown was removed, and a healing abutment was placed in preparation for surgery. The decision to remove the crown was made to facilitate the surgical intervention. (c) Clinical image depicting the preparation of a peri-implant split-thickness flap. It is important to note that a submarginal incision was made to preserve an adequate amount of peri-implant soft tissue, which facilitates the suturing of the graft. (d) Clinical appearance of the FGG harvested from the palate. (e) Clinical image depicting the adaptation and suturing of the FGG to the recipient site. (f) Clinical appearance of the grafted area during suture removal, which took place two weeks after surgery. It is important to observe the partial healing of the graft without any clinical signs of infection. (g) Clinical appearance of the grafted area during the replacement of the restoration. The presence of a newly formed, thick, and well-attached peri-implant mucosa is evident. (h) Clinical appearance of the grafted area two years after the surgical intervention. Notice the stability of the grafted area and the absence of plaque.

 (a) (b)

Additionally, current studies have also reported that vertical mucosal thickness plays an important role in maintaining/preventing marginal bone loss. Cases with a thin supracrestal tissue $\left($ <2mm) height (measured from the bone crest to the mucosal margin in apico-coronal direction) were associated with greater marginal bone loss than those with thick mucosa [47, 48], especially at bone level implants [49]. Surgical thickening of the vertical periimplant mucosa resulted in reduced marginal bone loss over time [34].

Implants with a thin soft-tissue phenotype with a reduced horizontal mucosal thickness seem to be more often associated with PIM and peri-implantitis compared to implants with a thick phenotype [50].

Nonetheless, there seems to be limited evidence to support the thickening of the peri-implant soft tissue in order to lower the incidence of peri-implant diseases [51–53].

In conclusion, peri-implant tissue thickness, in both horizontal and vertical direction, appears to represent important structural components for maintaining periimplant tissue health, thus affecting marginal bone remodeling, esthetic outcomes, restorative material transparency, and patient satisfaction.

Peri-implant Mucosal Recessions

Peri-implant mucosal recessions can significantly impact esthetic outcomes and patient satisfaction. The etiology of these recessions includes factors such as the mucosal phenotype [41], inadequate width of keratinized mucosa, the height of peri-implant marginal bone [54], improper orovestibular implant positioning [55, 56], characteristics of the implant abutment and prosthetic superstructure (emergence profile), and the angle of the implant fixture [46].

There are limited data available on the prevalence of peri-implant mucosal recessions. A systematic review reported peri-implant recessions in 0–64% of implants, based on data from only four studies [57]. Other studies have observed recessions occurring as early as one-year post-implant placement in 24% of cases [58].

Soft Tissue Augmentation Procedures

Soft tissue grafting procedures have been proposed to modify the soft tissue phenotype, with the goal of increasing tissue thickness, the width of KT, and the height of supracrestal tissue. These procedures aim to achieve optimal functional, biological, and esthetic outcomes for implants with thin or no keratinized mucosa, soft tissue deficiencies, or recessions [34, 52, 59].

Autogenous soft tissue grafts have demonstrated satisfactory results in natural dentition and were the initial approaches utilized for dental implants, including techniques such as free gingival grafts (FGGs) or connective tissue grafts (CTGs) [60, 61]. However, due to the higher morbidity associated with autogenous grafts, alternative materials such as xenogeneic collagen membranes (CMs) or acellular dermal matrix (ADM) have been introduced for peri-implant soft tissue augmentation. These materials serve as replacements for autogenous grafts.

Improvement of Keratinized Tissue Width Around Dental Implants

In a consensus report and accompanying systematic review, four randomized controlled clinical trials (RCTs) were included. These studies indicated a positive association between soft tissue grafting aimed at increasing the KTW and greater reductions in plaque and gingival indices compared to non-augmented sites [34, 62].

Techniques based on apical shifting (i.e. apically positioned flap [APF]) [63–65], or bilaminar procedures like the tunneling technique [66–69] have been the subject of investigation.

In a systematic review and meta-analysis conducted by Tavelli et al. [52], which included 23 RCTs, it was reported that both autogenous soft tissue grafts and soft tissue substitutes in combination with an APF, can lead to a statistically significant increase in KTW. When compared to non-augmented control sites, the use of APF and various graft types (FGG, CTG, ADM, and CM) resulted in superior outcomes. Notably, FGG demonstrated the highest improvements in KTW, with the following results: APF (2.48mm, 95% CI [1.35, 3.62], *p*=0.04), CM (2.96mm, 95% CI [1.82, 4.19], *p*=0.002), CTG (2.82mm, 95% CI [1.91, 4.14], *p*=0.007), FGG (3.67mm, 95% CI [3.03, 4.31], *p*=0.01), and ADM (3.02mm, 95% CI [1.87, 4.17], *p*=0.03) [52].

Moreover, the combination of APF with grafting procedures resulted in a statistically significant increase in KTW, leading to reductions in pocket depth, plaque accumulation, and mucosal recessions [52]. In some cases, although unpredictable, there may be observed a slight gain in attachment level when autogenous grafts are used for augmentation procedures [61, 70].

Interestingly, when comparing different treatment approaches including grafting procedures to APF alone, only FGG demonstrated a statistically significant increase in KTW. In contrast, bilaminar techniques did not achieve significant gains in KTW [52].

Increasing KTW has been shown to result in greater reductions in plaque accumulation (WMD = 0.344 , 95%) CI $[0.179, 0.509]$, $p < 0.001$) and gingival indices (WMD = 0.863, 95% CI [0.658, 1.067], *p* < 0.001), but it has no significant effect on bleeding on probing when compared to non-augmented sites [34, 62]. Moreover, an increase in KTW appears to contribute to a more stable position of the marginal bone levels, particularly when using APF with autogenous grafts (WMD = -0.175 mm, 95% CI $[-0.313, -0.037], p = 0.013$.

Improving KTW also has a positive impact on peri-implant marginal bone stability. In the meta-analysis conducted by Tavelli and colleagues, augmentation procedures involving APF and soft tissue grafts not only improved oral hygiene and reduced plaque accumulation but also resulted in less marginal bone loss [52]. Similar results have been reported by other authors who observed a positive association between KTW improvement and proper plaque control [21, 68, 69].

The timing of soft tissue augmentation procedures seems to have no impact on the gain in KTW with either the bilaminar technique or APF. Therefore, soft tissue augmentation can be performed at the time of implant placement, during the second stage, or at any delayed time [52, 71].

Improvement of Peri-implant Mucosal Thickness

Soft tissue augmentation procedures are commonly recommended to increase peri-implant mucosal thickness for esthetic improvements and to address volume deficiencies [42, 72, 73] as well as to facilitate tissue adaptation during implant placement [74].

In a recent meta-analysis utilizing a network model to assess mucosal thickness as an independent parameter, it was found that all evaluated augmentation methods (CTG, ADM, and CM) used in combination with a bilaminar surgical technique resulted in a statistically significant increase in mucosal thickness. CTG demonstrated the highest improvements (1.13mm, 95% CI [0.94, 1.31], *p* <0.001), followed by ADM (1.08mm, 95% CI [0.80, 1.35], *p* <0.001), and CM (0.76mm, 95% CI [0.55, 0.97], *p* <0.001) [52]. CM showed statistically significant lower mucosal thickness gain compared to CTG and ADM but showed greater gain compared to non-augmented sites: CTG (0.36 mm, 95% CI [0.23, 0.49], $p < 0.001$), ADM (0.31mm, 95% CI [0.04, 0.57], *p* =0.02), non-augmented sites (-0.76 mm, 95% CI [-0.97, -0.55], *p* <0.001). However, no statistically significant difference was observed between ADM and CTG $(p = 0.69)$ [52]. These findings are consistent with those obtained in previous studies that have also failed to demonstrate statistically significant differences in terms of mucosal thickness gains between ADM and CTG [75, 76].

It is important to note that the results of the afore mentioned meta-analysis [52] were generated by a network model, and direct clinical comparisons between ADM and CM have not been conducted. Therefore, these comparisons should be interpreted cautiously. Nevertheless, CTG remains the gold standard for augmenting peri-implant sites aiming to improve mucosal thickness, showing superior results over CM [34, 59, 62].

Positive effects of soft tissue augmentation were also observed on marginal bone loss. Augmented sites with CTG or CM using the bilaminar technique showed statistically significantly less marginal bone loss [52, 65, 77]. Another study highlighted that thicker peri-implant mucosa was associated with lower marginal bone loss [78]. These findings are supported by other authors, emphasizing that non-augmented sites exhibit statistically significantly higher marginal bone loss compared to sites augmented with CTG or CM, which appear to provide a certain level of stability for the supporting marginal bone [74, 79, 80].

Two systematic reviews and meta-analyses have failed to demonstrate statistically significant associations between mucosal thickness gain and reductions in plaque indices or pocket depth [34, 52]. Plaque indices remained stable regardless of the intervention used. However, it is interesting to note that one RCT reported a statistically significant reduction in probing pocket depth following mucosal thickening [74].

Similarly, soft tissue augmentation procedures for mucosal thickening had no significant impact on bleeding on probing values compared to non-augmented sites [34].

It appears that the timing of soft tissue augmentation procedures for mucosal thickening can be performed at the time of implant placement, during the second stage, or at a delayed time, with no statistically significant differences in outcomes [52]. These findings are supported by another systematic review and meta-analysis where mucosal thickening was performed either at the time of implant placement or during the second stage, three months after implant placement but before prosthetic reconstruction [34]. The findings by Tavelli et al., Thoma et al. [34, 52] are consistent with those of other studies where mucosal thickening mucosal-thickening procedures were performed either at the time of implant placement [44, 74, 81] or during the second stage, three months after implant placement but prior to prosthetic reconstruction [42, 72, 73].

Improvement of Soft Tissue Height

Augmentation for the purpose of gaining soft tissue height has been subject to limited investigation, although there have been suggestions regarding the effectiveness of ADM in increasing soft tissue height [82]. However, several authors have highlighted the negative impact of thin initial soft tissue height on marginal bone loss [49, 83]. In certain studies, non-augmented sites exhibited significant marginal bone loss, whereas implants with ADM augmentation and an increase in soft tissue height demonstrated reduced marginal bone loss [82, 84].

Coverage of Soft Tissue Dehiscence at Dental Implants

The evidence supporting mucosal recession coverage at implants is currently limited to a few case series with a small number of patients [85–89].

Different techniques have been used by various authors, including a coronally advanced flap combined with a subepithelial CTG [85, 89], a bilaminar technique (such as a split-thickness flap) with a xenogeneic CM [88] or CTG [90] harvested from the tuberosity.

The success rate of mucosal recession coverage ranges from 66% at 6 months [85, 86] to 96.3% after 12–20 months [89, 90]. Factors that can influence these outcomes include the surgical technique employed (coronally advanced flap with or without vertical incisions versus tunneling technique), the graft harvesting technique (single incision technique versus de-epithelialized FGG), the initial amount of peri-implant KT, and the accessibility determined by the maintenance or removal of the prosthetic superstructure during the surgical procedure.

Promising results have also been achieved when using a xenogeneic CM membrane with a modified coronally advanced tunnel technique, compared to a subepithelial CTG [88].

However, at present, there is only very limited evidence available on the long-term stability following coverage of mucosal recessions at osseointegrated dental implants.

Roccuzzo et al. [91] reported the five-year outcomes of a coverage procedure for shallow maxillary soft tissue dehiscences around single tissue-level implants. A CTG harvested from the maxillary tuberosity was inserted underneath a split-thickness envelope flap. Following treatment, patients received individually tailored supportive periodontal therapy. After five years, complete soft tissue coverage was achieved in 8 out of 13 cases (62%), with a mean recession coverage of 86%. Patients' esthetic evaluation revealed high visual analog scale (VAS) scores.

However, due to the limited available evidence, no definitive long-term conclusions can be drawn regarding the stability of mucosal recession coverage.

Overall, the current evidence suggests that currently only shallow mucosal recessions (up to 2–3mm) at implants that are properly positioned in three jaw dimensions can be successfully covered. However, data on the efficacy of these techniques for larger and deeper mucosal recessions are still lacking [46] (Figure 10.2a–e).

In conclusion, soft tissue augmentation procedures around dental implants contribute to improved periimplant health by increasing the width of keratinized mucosa and the thickness of the mucosal tissue. This leads

Figure 10.2 (a) This clinical photograph shows a deep and extensive gingival recession at tooth #3, along with two shallow adjacent mucosal recessions at implants located in areas #4 and #5. (b) The preparation involves a combination of a tunnel and envelope flap to create space for a palatal subepithelial CTG. (c) The palatal subepithelial CTG was carefully adapted and sutured to provide complete coverage of the exposed recessions at tooth #3 and the implants in #4 and #5. (d) The envelope and tunneled flap were coronally advanced and meticulously sutured to ensure full coverage of both the palatal subepithelial CTG and the recessions. (e) At the two-year follow-up, it is evident that complete coverage of all recessions has been achieved.

Figure 10.2 (Continued)

to positive effects on bleeding indices and a reduction in marginal bone loss. These procedures also result in esthetic improvements, facilitate oral hygiene and plaque removal, and enhance patient satisfaction. Overall, soft tissue augmentation plays a crucial role in the long-term maintenance of peri-implant health and the stability of peri-implant tissues.

References

- **1** Jung, R.E., Pjetursson, B.E., Glauser, R. et al. (2008). A systematic review of the 5-year survival and complication rates of implant-supported single crowns. *Clinical Oral Implants Research 19* (2): 119–130. <https://doi.org/10.1111/j.1600-0501.2007.01453.x>.
- **2** Lee, C.T., Huang, Y.W., Zhu, L., and Weltman, R. (2017). Prevalences of peri-implantitis and peri-implant mucositis: systematic review and meta-analysis. *Journal of Dentistry 62*: 1–12. [https://doi.org/10.1016/j.jdent.](https://doi.org/10.1016/j.jdent.2017.04.011) [2017.04.011](https://doi.org/10.1016/j.jdent.2017.04.011).
- **3** Salvi, G.E., Cosgarea, R., and Sculean, A. (2017). Prevalence and mechanisms of peri-implant diseases. *Journal of Dental Research 96* (1): 31–37. [https://doi.](https://doi.org/10.1177/0022034516667484) [org/10.1177/0022034516667484.](https://doi.org/10.1177/0022034516667484)
- **4** Rasperini, G., Siciliano, V.I., Cafiero, C. et al. (2014). Crestal bone changes at teeth and implants in periodontally healthy and periodontally compromised patients. A 10-year comparative case-series study. *Journal of Periodontology 85* (6): e152–e159. [https://doi.org/](https://doi.org/10.1902/jop.2013.130415) [10.1902/jop.2013.130415](https://doi.org/10.1902/jop.2013.130415).
- **5** Heitz-Mayfield, L.J.A. and Salvi, G.E. (2018). Peri-implant mucositis. *Journal of Clinical Periodontology 45* (Suppl 20): S237–S245. [https://doi.org/10.1111/jcpe.12953.](https://doi.org/10.1111/jcpe.12953)
- **6** Berglundh, T., Armitage, G., Araujo, M.G. et al. (2018). 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 Clinical Periodontology 45* (Suppl 20): S286–S291. [https://doi.org/10.1111/jcpe.12957.](https://doi.org/10.1111/jcpe.12957)
- **7** Schwarz, F., Derks, J., Monje, A., and Wang, H.L. (2018). Peri-implantitis. *Journal of Periodontology 89* (Suppl 1): S267–S290.<https://doi.org/10.1002/JPER.16-0350>.
- **8** Avila-Ortiz, G., Gonzalez-Martin, O., Couso-Queiruga, E., and Wang, H.L. (2020). The peri-implant phenotype. *Journal of Periodontology 91* (3): 283–288. [https://doi.](https://doi.org/10.1002/JPER.19-0566) [org/10.1002/JPER.19-0566](https://doi.org/10.1002/JPER.19-0566).
- **9** Berglundh, T., Lindhe, J., Ericsson, I., and Liljenberg, B. (1992). Enhanced gingivitis in the deciduous and permanent dentition. An experimental study in the dog. *Journal of Clinical Periodontology 19* (2): 135–142. <https://doi.org/10.1111/j.1600-051x.1992.tb00452.x>.
- **10** Lim, H.P., Lee, K.M., Koh, Y.I., and Park, S.W. (2012). Allergic contact stomatitis caused by a titanium nitridecoated implant abutment: a clinical report. *The Journal of Prosthetic Dentistry 108* (4): 209–213. [https://doi.org/](https://doi.org/10.1016/S0022-3913(12)60163-2) [10.1016/S0022-3913\(12\)60163-2.](https://doi.org/10.1016/S0022-3913(12)60163-2)
- **11** Lopez-Jornet, P., Camacho-Alonso, F., and Sanchez-Siles, M. (2014). Dental implants in patients with oral lichen planus: a cross-sectional study. *Clinical Implant Dentistry and Related Research 16* (1): 107–115. [https://doi.org/](https://doi.org/10.1111/j.1708-8208.2012.00455.x) [10.1111/j.1708-8208.2012.00455.x](https://doi.org/10.1111/j.1708-8208.2012.00455.x).
- **12** Siddiqi, A., Payne, A.G.T., De Silva, R.K., and Duncan, W.J. (2011). Titanium allergy: could it affect dental implant integration? *Clinical Oral Implants Research 22* (7): 673–680. [https://doi.](https://doi.org/10.1111/j.1600-0501.2010.02081.x) [org/10.1111/j.1600-0501.2010.02081.x.](https://doi.org/10.1111/j.1600-0501.2010.02081.x)
- **13** Gobbato, L., Avila-Ortiz, G., Sohrabi, K. et al. (2013). The effect of keratinized mucosa width on peri-implant health: a systematic review. *International Journal of Oral and Maxillofacial Implants 28* (6): 1536–1545. [https://doi.](https://doi.org/10.11607/jomi.3244) [org/10.11607/jomi.3244.](https://doi.org/10.11607/jomi.3244)
- **14** Lin, G.H., Chan, H.L., and Wang, H.L. (2013). The significance of keratinized mucosa on implant health: a systematic review. *Journal of Periodontology 84* (12): 1755–1767. [https://doi.org/10.1902/jop.2013.120688.](https://doi.org/10.1902/jop.2013.120688)
- **15** Schou, S., Holmstrup, P., Hjorting-Hansen, E., and Lang, N.P. (1992). Plaque-induced marginal tissue reactions of osseointegrated oral implants: a review of the literature. *Clinical Oral Implants Research 3* (4): 149–161. [https://](https://doi.org/10.1034/j.1600-0501.1992.030401.x) [doi.org/10.1034/j.1600-0501.1992.030401.x.](https://doi.org/10.1034/j.1600-0501.1992.030401.x)
- **16** Strub, J.R., Gaberthuel, T.W., and Grunder, U. (1991). The role of attached gingiva in the health of peri-implant

tissue in dogs. 1. Clinical findings. *International Journal of Periodontics and Restorative Dentistry 11* (4): 317–333.

- **17** Warrer, K., Buser, D., Lang, N.P., and Karring, T. (1995). Plaque-induced peri-implantitis in the presence or absence of keratinized mucosa. An experimental study in monkeys. *Clinical Oral Implants Research 6* (3): 131–138. <https://doi.org/10.1034/j.1600-0501.1995.060301.x>.
- **18** Wennstrom, J.L. and Derks, J. (2012). Is there a need for keratinized mucosa around implants to maintain health and tissue stability? *Clinical Oral Implants Research 23* (Suppl 6): 136–146. [https://doi.org/10.1111/](https://doi.org/10.1111/j.1600-0501.2012.02540.x) [j.1600-0501.2012.02540.x.](https://doi.org/10.1111/j.1600-0501.2012.02540.x)
- **19** Ueno, D., Nagano, T., Watanabe, T. et al. (2016). Effect of the keratinized mucosa width on the health status of periimplant and contralateral periodontal tissues: a cross-sectional study. *Implant Dentistry 25* (6): 796–801. [https://doi.org/10.1097/ID.0000000000000483.](https://doi.org/10.1097/ID.0000000000000483)
- **20** Souza, A.B., Tormena, M., Matarazzo, F., and Araujo, M.G. (2016). The influence of peri-implant keratinized mucosa on brushing discomfort and peri-implant tissue health. *Clinical Oral Implants Research 27* (6): 650–655. [https://doi.org/10.1111/clr.12703.](https://doi.org/10.1111/clr.12703)
- **21** Roccuzzo, M., Grasso, G., and Dalmasso, P. (2016). Keratinized mucosa around implants in partially edentulous posterior mandible: 10-year results of a prospective comparative study. *Clinical Oral Implants Research 27* (4): 491–496. [https://doi.org/10.1111/](https://doi.org/10.1111/clr.12563) [clr.12563.](https://doi.org/10.1111/clr.12563)
- **22** Ladwein, C., Schmelzeisen, R., Nelson, K. et al. (2015). Is the presence of keratinized mucosa associated with periimplant tissue health? A clinical cross-sectional analysis. *International Journal of Implant Dentistry 1* (1): 11. [https://doi.org/10.1186/s40729-015-0009-z.](https://doi.org/10.1186/s40729-015-0009-z)
- **23** Roos-Jansaker, A.M., Renvert, H., Lindahl, C., and Renvert, S. (2006). Nine- to fourteen-year follow-up of implant treatment. Part III: factors associated with peri-implant lesions. *Journal of Clinical Periodontology 33* (4): 296–301. [https://doi.org/10.1111/j.1600-051X.2006.00908.x.](https://doi.org/10.1111/j.1600-051X.2006.00908.x)
- **24** Adibrad, M., Shahabuei, M., and Sahabi, M. (2009). Significance of the width of keratinized mucosa on the health status of the supporting tissue around implants supporting overdentures. *The Journal of Oral Implantology 35* (5): 232–237. [https://doi.org/10.1563/](https://doi.org/10.1563/AAID-JOI-D-09-00035.1) [AAID-JOI-D-09-00035.1](https://doi.org/10.1563/AAID-JOI-D-09-00035.1).
- **25** Bouri, A. Jr., Bissada, N., Al-Zahrani, M.S. et al. (2008). Width of keratinized gingiva and the health status of the supporting tissues around dental implants. *The International Journal of Oral & Maxillofacial Implants 23* (2): 323–326.
- **26** Crespi, R., Cappare, P., and Gherlone, E. (2010). A 4-year evaluation of the peri-implant parameters of immediately loaded implants placed in fresh extraction sockets.

Journal of Periodontology 81 (11): 1629–1634. [https://doi.](https://doi.org/10.1902/jop.2010.100115) [org/10.1902/jop.2010.100115.](https://doi.org/10.1902/jop.2010.100115)

- **27** Schrott, A.R., Jimenez, M., Hwang, J.W. et al. (2009). Five-year evaluation of the influence of keratinized mucosa on peri-implant soft-tissue health and stability around implants supporting full-arch mandibular fixed prostheses. *Clinical Oral Implants Research 20* (10): 1170–1177. <https://doi.org/10.1111/j.1600-0501.2009.01795.x>.
- **28** Frisch, E., Ziebolz, D., Vach, K., and Ratka-Kruger, P. (2015). The effect of keratinized mucosa width on peri-implant outcome under supportive postimplant therapy. *Clinical Implant Dentistry and Related Research 17* (Suppl 1): e236–e244. [https://doi.org/10.1111/](https://doi.org/10.1111/cid.12187) [cid.12187.](https://doi.org/10.1111/cid.12187)
- **29** Zigdon, H. and Machtei, E.E. (2008). The dimensions of keratinized mucosa around implants affect clinical and immunological parameters. *Clinical Oral Implants Research 19* (4): 387–392. [https://doi.](https://doi.org/10.1111/j.1600-0501.2007.01492.x) [org/10.1111/j.1600-0501.2007.01492.x.](https://doi.org/10.1111/j.1600-0501.2007.01492.x)
- **30** Canullo, L., Penarrocha-Oltra, D., Covani, U. et al. (2016). Clinical and microbiological findings in patients with peri-implantitis: a cross-sectional study. *Clinical Oral Implants Research 27* (3): 376–382. [https://doi.org/](https://doi.org/10.1111/clr.12557) [10.1111/clr.12557](https://doi.org/10.1111/clr.12557).
- **31** Romanos, G., Grizas, E., and Nentwig, G.H. (2015). Association of keratinized mucosa and peri-implant soft tissue stability around implants with platform switching. *Implant Dentistry* 24 (4): 422–426. [https://doi.](https://doi.org/10.1097/ID.0000000000000274) [org/10.1097/ID.0000000000000274.](https://doi.org/10.1097/ID.0000000000000274) PMID: 26200163.
- **32** Imber, J.C., Roccuzzo, A., Stähli, A. et al. (2023, 2023). Spontaneous regeneration of keratinized tissue at implants and teeth. *Journal of Clinical Periodontology* 50 (8): 1064–1074.<https://doi.org/10.1111/jcpe.13820>. Epub 2023 Apr 26. PMID: 37186090.
- **33** Berglundh, T. and Lindhe, J. (1996). Dimension of the periimplant mucosa. Biological width revisited. *Journal of Clinical Periodontology 23* (10): 971–973. [https://doi.](https://doi.org/10.1111/j.1600-051x.1996.tb00520.x) [org/10.1111/j.1600-051x.1996.tb00520.x](https://doi.org/10.1111/j.1600-051x.1996.tb00520.x).
- **34** Thoma, D.S., Naenni, N., Figuero, E. et al. (2018). Effects of soft tissue augmentation procedures on peri-implant health or disease: a systematic review and meta-analysis. *Clinical Oral Implants Research 29* (Suppl 15): 32–49. [https://doi.org/10.1111/clr.13114.](https://doi.org/10.1111/clr.13114)
- **35** Thoma, D.S., Muhlemann, S., and Jung, R.E. (2014). Critical soft-tissue dimensions with dental implants and treatment concepts. *Periodontology 2000 66* (1): 106–118. <https://doi.org/10.1111/prd.12045>.
- **36** Bressan, E., Paniz, G., Lops, D. et al. (2011). Influence of abutment material on the gingival color of implantsupported all-ceramic restorations: a prospective multicenter study. *Clinical Oral Implants Research 22* (6): 631–637.<https://doi.org/10.1111/j.1600-0501.2010.02008.x>.
- **37** Cosgarea, R., Gasparik, C., Dudea, D. et al. (2015). Peri-implant soft tissue colour around titanium and zirconia abutments: a prospective randomized controlled clinical study. *Clinical Oral Implants Research 26* (5): 537–544. [https://doi.org/10.1111/](https://doi.org/10.1111/clr.12440) [clr.12440.](https://doi.org/10.1111/clr.12440)
- **38** Park, S.E., Da Silva, J.D., Weber, H.P., and Ishikawa-Nagai, S. (2007). Optical phenomenon of peri-implant soft tissue. Part I. Spectrophotometric assessment of natural tooth gingiva and peri-implant mucosa. *Clinical Oral Implants Research 18* (5): 569–574. [https://doi.org/](https://doi.org/10.1111/j.1600-0501.2007.01391.x) [10.1111/j.1600-0501.2007.01391.x](https://doi.org/10.1111/j.1600-0501.2007.01391.x).
- **39** Lops, D., Stellini, E., Sbricoli, L. et al. (2017). Influence of abutment material on peri-implant soft tissues in anterior areas with thin gingival biotype: a multicentric prospective study. *Clinical Oral Implants Research 28* (10): 1263–1268.<https://doi.org/10.1111/clr.12952>.
- **40** Kan, J.Y., Morimoto, T., Rungcharassaeng, K. et al. (2010). Gingival biotype assessment in the esthetic zone: visual versus direct measurement. *International Journal of Periodontics and Restorative Dentistry 30* (3): 237–243.
- **41** Kan, J.Y., Rungcharassaeng, K., Lozada, J.L., and Zimmerman, G. (2011). Facial gingival tissue stability following immediate placement and provisionalization of maxillary anterior single implants: a 2- to 8-year follow-up. *The International Journal of Oral & Maxillofacial Implants 26* (1): 179–187.
- **42** Bienz, S.P., Jung, R.E., Sapata, V.M. et al. (2017). Volumetric changes and peri-implant health at implant sites with or without soft tissue grafting in the esthetic zone, a retrospective case-control study with a 5-year follow-up. *Clinical Oral Implants Research 28* (11): 1459–1465.<https://doi.org/10.1111/clr.13013>.
- **43** Hosseini, M., Worsaae, N., and Gotfredsen, K. (2020). Tissue changes at implant sites in the anterior maxilla with and without connective tissue grafting: a five-year prospective study. *Clinical Oral Implants Research 31* (1): 18–28.<https://doi.org/10.1111/clr.13540>.
- **44** Migliorati, M., Amorfini, L., Signori, A. et al. (2015). Clinical and aesthetic outcome with post-extractive implants with or without soft tissue augmentation: a 2-year randomized clinical trial. *Clinical Implant Dentistry and Related Research 17* (5): 983–995. [https://](https://doi.org/10.1111/cid.12194) doi.org/10.1111/cid.12194.
- **45** Zuiderveld, E.G., Meijer, H.J.A., den Hartog, L. et al. (2018). Effect of connective tissue grafting on periimplant tissue in single immediate implant sites: A RCT. *Journal of Clinical Periodontology 45* (2): 253–264. [https://doi.org/10.1111/jcpe.12820.](https://doi.org/10.1111/jcpe.12820)
- **46** Sculean, A., Chappuis, V., and Cosgarea, R. (2017). Coverage of mucosal recessions at dental implants.

Periodontology 2000 73 (1): 134–140. [https://doi.org/](https://doi.org/10.1111/prd.12178) [10.1111/prd.12178.](https://doi.org/10.1111/prd.12178)

- **47** Linkevicius, T., Apse, P., Grybauskas, S., and Puisys, A. (2009). The influence of soft tissue thickness on crestal bone changes around implants: a 1-year prospective controlled clinical trial. *International Journal of Oral and Maxillofacial Implants 24* (4): 712–719.
- **48** Linkevicius, T., Linkevicius, R., Alkimavicius, J. et al. (2018). Influence of titanium base, lithium disilicate restoration and vertical soft tissue thickness on bone stability around triangular-shaped implants: a prospective clinical trial. *Clinical Oral Implants Research 29* (7): 716–724.<https://doi.org/10.1111/clr.13263>.
- **49** Diaz-Sanchez, M., Soto-Penaloza, D., Penarrocha-Oltra, D., and Penarrocha-Diago, M. (2019). Influence of supracrestal tissue attachment thickness on radiographic bone level around dental implants: a systematic review and meta-analysis. *Journal of Periodontal Research 54* (6): 573–588. [https://doi.org/10.1111/jre.12663.](https://doi.org/10.1111/jre.12663)
- **50** Gharpure, A.S., Latimer, J.M., Aljofi, F.E. et al. (2021). Role of thin gingival phenotype and inadequate keratinized mucosa width (<2 mm) as risk indicators for peri-implantitis and peri-implant mucositis. *Journal of Periodontology 92* (12): 1687–1696. [https://doi.org/10.1002/JPER.20-0792.](https://doi.org/10.1002/JPER.20-0792)
- **51** Carra, M.C., Blanc-Sylvestre, N., Courtet, A., and Bouchard, P. (2023). Primordial and primary prevention of peri-implant diseases: a systematic review and meta-analysis. *Journal of Clinical Periodontology* [https://doi.org/10.1111/jcpe.13790.](https://doi.org/10.1111/jcpe.13790)
- **52** Tavelli, L., Barootchi, S., Avila-Ortiz, G. et al. (2021). Peri-implant soft tissue phenotype modification and its impact on peri-implant health: a systematic review and network meta-analysis. *Journal of Periodontology 92* (1): 21–44.<https://doi.org/10.1002/JPER.19-0716>.
- **53** Valles, C., Vilarrasa, J., Barallat, L. et al. (2022). Efficacy of soft tissue augmentation procedures on tissue thickening around dental implants: a systematic review and meta-analysis. *Clinical Oral Implants Research 33* (Suppl 23): 72–99. [https://doi.org/10.1111/clr.13920.](https://doi.org/10.1111/clr.13920)
- **54** Buser, D., Martin, W., and Belser, U.C. (2004). Optimizing esthetics for implant restorations in the anterior maxilla: anatomic and surgical considerations. *The International Journal of Oral & Maxillofacial Implants 19* (Suppl): 43–61.
- **55** Chen, S.T. and Buser, D. (2016). Esthetic complications due to implant malpositions: etiology, prevention, and treatment. In: *Dental Implant Complications Etiology, Prevention and Treatment* (ed. S.I. Froum), 209–232. Chichester: Wiley-Blackwell.
- **56** Evans, C.D. and Chen, S.T. (2008). Esthetic outcomes of immediate implant placements. *Clinical Oral Implants Research 19* (1): 73–80. [https://doi.org/10.1111/j.1600-](https://doi.org/10.1111/j.1600-0501.2007.01413.x) [0501.2007.01413.x.](https://doi.org/10.1111/j.1600-0501.2007.01413.x)
- **57** Cosyn, J., Hooghe, N., and De Bruyn, H. (2012). A systematic review on the frequency of advanced recession following single immediate implant treatment. *Journal of Clinical Periodontology 39* (6): 582–589. [https://doi.org/](https://doi.org/10.1111/j.1600-051X.2012.01888.x) [10.1111/j.1600-051X.2012.01888.x.](https://doi.org/10.1111/j.1600-051X.2012.01888.x)
- **58** Cosyn, J., Sabzevar, M.M., and De Bruyn, H. (2012). Predictors of inter-proximal and midfacial recession following single implant treatment in the anterior maxilla: a multivariate analysis. *Journal of Clinical Periodontology 39* (9): 895–903. [https://doi.org/](https://doi.org/10.1111/j.1600-051X.2012.01921.x) [10.1111/j.1600-051X.2012.01921.x.](https://doi.org/10.1111/j.1600-051X.2012.01921.x)
- **59** Cairo, F., Barbato, L., Selvaggi, F. et al. (2019). Surgical procedures for soft tissue augmentation at implant sites. A systematic review and meta-analysis of randomized controlled trials. *Clinical Implant Dentistry and Related Research 21* (6): 1262–1270. [https://doi.org/10.1111/](https://doi.org/10.1111/cid.12861) [cid.12861.](https://doi.org/10.1111/cid.12861)
- **60** Tavelli, L., Barootchi, S., Cairo, F. et al. (2019). The effect of time on root coverage outcomes: a network metaanalysis. *Journal of Dental Research 98* (11): 1195–1203. [https://doi.org/10.1177/0022034519867071.](https://doi.org/10.1177/0022034519867071)
- **61** Zucchelli, G., Tavelli, L., McGuire, M.K. et al. (2020). Autogenous soft tissue grafting for periodontal and peri-implant plastic surgical reconstruction. *Journal of Periodontology 91* (1): 9–16. [https://doi.org/10.1002/](https://doi.org/10.1002/JPER.19-0350) [JPER.19-0350.](https://doi.org/10.1002/JPER.19-0350)
- **62** Giannobile, W.V., Jung, R.E., Schwarz, F., and Groups of the 2nd Osteology Foundation Consensus, M (2018). Evidence-based knowledge on the aesthetics and maintenance of peri-implant soft tissues: osteology foundation consensus report part 1-effects of soft tissue augmentation procedures on the maintenance of peri-implant soft tissue health. *Clinical Oral Implants Research 29* (Suppl 15): 7–10. [https://doi.org/10.1111/](https://doi.org/10.1111/clr.13110) [clr.13110.](https://doi.org/10.1111/clr.13110)
- **63** Anderson, L.E., Inglehart, M.R., El-Kholy, K. et al. (2014). Implant associated soft tissue defects in the anterior maxilla: a randomized control trial comparing subepithelial connective tissue graft and acellular dermal matrix allograft. *Implant Dentistry 23* (4): 416–425. [https://doi.org/10.1097/ID.0000000000000122.](https://doi.org/10.1097/ID.0000000000000122)
- **64** D'Elia, C., Baldini, N., Cagidiaco, E.F. et al. (2017). Peri-implant soft tissue stability after single implant restorations using either guided bone regeneration or a connective tissue graft: a randomized clinical trial. *International Journal of Periodontics and Restorative Dentistry 37* (3): 413–421. [https://doi.org/10.11607/](https://doi.org/10.11607/prd.2747) [prd.2747.](https://doi.org/10.11607/prd.2747)
- **65** De Bruyckere, T., Eeckhout, C., Eghbali, A. et al. (2018). A randomized controlled study comparing guided bone regeneration with connective tissue graft to re-establish convexity at the buccal aspect of single implants:

a one-year CBCT analysis. *Journal of Clinical Periodontology 45* (11): 1375–1387. [https://doi.org/10.](https://doi.org/10.1111/jcpe.13006) [1111/jcpe.13006.](https://doi.org/10.1111/jcpe.13006)

- **66** Basegmez, C., Ersanli, S., Demirel, K. et al. (2012). The comparison of two techniques to increase the amount of peri-implant attached mucosa: free gingival grafts versus vestibuloplasty. One-year results from a randomised controlled trial. *European Journal of Oral Implantology 5* (2): 139–145.
- **67** Basegmez, C., Karabuda, Z.C., Demirel, K., and Yalcin, S. (2013). The comparison of acellular dermal matrix allografts with free gingival grafts in the augmentation of peri-implant attached mucosa: a randomised controlled trial. *European Journal of Oral Implantology 6* (2): 145–152.
- **68** Lorenzo, R., Garcia, V., Orsini, M. et al. (2012). Clinical efficacy of a xenogeneic collagen matrix in augmenting keratinized mucosa around implants: a randomized controlled prospective clinical trial. *Clinical Oral Implants Research 23* (3): 316–324. [https://doi.](https://doi.org/10.1111/j.1600-0501.2011.02260.x) [org/10.1111/j.1600-0501.2011.02260.x.](https://doi.org/10.1111/j.1600-0501.2011.02260.x)
- **69** Oh, S.L., Masri, R.M., Williams, D.A. et al. (2017). Free gingival grafts for implants exhibiting lack of keratinized mucosa: a prospective controlled randomized clinical study. *Journal of Clinical Periodontology 44* (2): 195–203. [https://doi.org/10.1111/jcpe.12660.](https://doi.org/10.1111/jcpe.12660)
- **70** Yu, S.H., Tseng, S.C., and Wang, H.L. (2018). Classification of soft tissue grafting materials based on biologic principles. *International Journal of Periodontics and Restorative Dentistry 38* (6): 849–854. [https://doi.org/](https://doi.org/10.11607/prd.3622) [10.11607/prd.3622.](https://doi.org/10.11607/prd.3622)
- **71** Lin, C.Y., Chen, Z., Pan, W.L., and Wang, H.L. (2018). Impact of timing on soft tissue augmentation during implant treatment: a systematic review and metaanalysis. *Clinical Oral Implants Research 29* (5): 508–521. [https://doi.org/10.1111/clr.13148.](https://doi.org/10.1111/clr.13148)
- **72** Cosyn, J., Eghbali, A., Hermans, A. et al. (2016). A 5-year prospective study on single immediate implants in the aesthetic zone. *Journal of Clinical Periodontology 43* (8): 702–709.<https://doi.org/10.1111/jcpe.12571>.
- **73** Fenner, N., Hammerle, C.H., Sailer, I., and Jung, R.E. (2016). Long-term clinical, technical, and esthetic outcomes of all-ceramic vs. titanium abutments on implant supporting single-tooth reconstructions after at least 5 years. *Clinical Oral Implants Research 27* (6): 716–723.<https://doi.org/10.1111/clr.12654>.
- **74** Bianchi, A.E. and Sanfilippo, F. (2004). Single-tooth replacement by immediate implant and connective tissue graft: a 1-9-year clinical evaluation. *Clinical Oral Implants Research 15* (3): 269–277. [https://doi.org/10.1111/j.1600-](https://doi.org/10.1111/j.1600-0501.2004.01020.x) [0501.2004.01020.x.](https://doi.org/10.1111/j.1600-0501.2004.01020.x)
- **75** Hutton, C.G., Johnson, G.K., Barwacz, C.A. et al. (2018). Comparison of two different surgical approaches to

increase peri-implant mucosal thickness: a randomized controlled clinical trial. *Journal of Periodontology 89* (7): 807–814.<https://doi.org/10.1002/JPER.17-0597>.

- **76** Paolantonio, M., Dolci, M., Esposito, P. et al. (2002). Subpedicle acellular dermal matrix graft and autogenous connective tissue graft in the treatment of gingival recessions: a comparative 1-year clinical study. *Journal of Periodontology 73* (11): 1299–1307. [https://doi.org/10.](https://doi.org/10.1902/jop.2002.73.11.1299) [1902/jop.2002.73.11.1299.](https://doi.org/10.1902/jop.2002.73.11.1299)
- **77** Cairo, F., Barbato, L., Tonelli, P. et al. (2017). Xenogeneic collagen matrix versus connective tissue graft for buccal soft tissue augmentation at implant site. A randomized, controlled clinical trial. *Journal of Clinical Periodontology 44* (7): 769–776.<https://doi.org/10.1111/jcpe.12750>.
- **78** Puzio, M., Hadzik, J., Blaszczyszyn, A. et al. (2020). Soft tissue augmentation around dental implants with connective tissue graft (CTG) and xenogenic collagen matrix (XCM). 1-year randomized control trail. *Annals of Anatomy 230*: 151484. [https://doi.org/10.1016/j.aanat.](https://doi.org/10.1016/j.aanat.2020.151484) [2020.151484](https://doi.org/10.1016/j.aanat.2020.151484).
- **79** Eghbali, A., De Bruyn, H., Cosyn, J. et al. (2016). Ultrasonic assessment of mucosal thickness around implants: validity, reproducibility, and stability of connective tissue grafts at the buccal aspect. *Clinical Implant Dentistry and Related Research 18* (1): 51–61. [https://doi.org/10.1111/cid.12245.](https://doi.org/10.1111/cid.12245)
- **80** Thoma, D.S., Gasser, T.J.W., Jung, R.E., and Hammerle, C.H.F. (2020). Randomized controlled clinical trial comparing implant sites augmented with a volume-stable collagen matrix or an autogenous connective tissue graft: 3-year data after insertion of reconstructions. *Journal of Clinical Periodontology 47* (5): 630–639. [https://doi.org/](https://doi.org/10.1111/jcpe.13271) [10.1111/jcpe.13271](https://doi.org/10.1111/jcpe.13271).
- **81** Yoshino, S., Kan, J.Y., Rungcharassaeng, K. et al. (2014). Effects of connective tissue grafting on the facial gingival level following single immediate implant placement and provisionalization in the esthetic zone: a 1-year randomized controlled prospective study. *The International Journal of Oral & Maxillofacial Implants 29* (2): 432–440. <https://doi.org/10.11607/jomi.3379>.
- **82** Puisys, A., Vindasiute, E., Linkevciene, L., and Linkevicius, T. (2015). The use of acellular dermal matrix membrane for vertical soft tissue augmentation during submerged implant placement: a case series. *Clinical Oral Implants Research 26* (4): 465–470.<https://doi.org/10.1111/clr.12401>.
- **83** Suarez-Lopez Del Amo, F., Lin, G.H., Monje, A. et al. (2016). Influence of soft tissue thickness on peri-implant marginal bone loss: a systematic review and metaanalysis. *Journal of Periodontology 87* (6): 690–699. <https://doi.org/10.1902/jop.2016.150571>.
- **84** Tavelli, L., McGuire, M.K., Zucchelli, G. et al. (2020). Extracellular matrix-based scaffolding technologies for

periodontal and peri-implant soft tissue regeneration. *Journal of Periodontology 91* (1): 17–25. [https://doi.](https://doi.org/10.1002/JPER.19-0351) [org/10.1002/JPER.19-0351](https://doi.org/10.1002/JPER.19-0351).

- **85** Burkhardt, R., Joss, A., and Lang, N.P. (2008). Soft tissue dehiscence coverage around endosseous implants: a prospective cohort study. *Clinical Oral Implants Research 19* (5): 451–457. [https://doi.org/10.1111/j.1600-0501.](https://doi.org/10.1111/j.1600-0501.2007.01497.x) [2007.01497.x.](https://doi.org/10.1111/j.1600-0501.2007.01497.x)
- **86** Le, B., Borzabadi-Farahani, A., and Nielsen, B. (2016). Treatment of labial soft tissue recession around dental implants in the esthetic zone using guided bone regeneration with mineralized allograft: a retrospective clinical case series. *Journal of Oral and Maxillofacial Surgery 74* (8): 1552–1561. [https://doi.org/10.1016/](https://doi.org/10.1016/j.joms.2016.04.015) [j.joms.2016.04.015](https://doi.org/10.1016/j.joms.2016.04.015).
- **87** Rotundo, R., Pagliaro, U., Bendinelli, E. et al. (2015). Long-term outcomes of soft tissue augmentation around dental implants on soft and hard tissue stability: a systematic review. *Clinical Oral Implants Research 26* (Suppl 11): 123–138. [https://doi.org/10.1111/clr.](https://doi.org/10.1111/clr.12629) [12629.](https://doi.org/10.1111/clr.12629)
- **88** Schallhorn, R.A., McClain, P.K., Charles, A. et al. (2015). Evaluation of a porcine collagen matrix used to augment keratinized tissue and increase soft tissue thickness around existing dental implants. *International Journal of Periodontics and Restorative Dentistry 35* (1): 99–103. <https://doi.org/10.11607/prd.1888>.
- **89** Zucchelli, G., Mazzotti, C., Mounssif, I. et al. (2013). A novel surgical-prosthetic approach for soft tissue dehiscence coverage around single implant. *Clinical Oral Implants Research 24* (9): 957–962. [https://doi.](https://doi.org/10.1111/clr.12003) [org/10.1111/clr.12003.](https://doi.org/10.1111/clr.12003)
- **90** Roccuzzo, M., Gaudioso, L., Bunino, M., and Dalmasso, P. (2014). Surgical treatment of buccal soft tissue recessions around single implants: 1-year results from a prospective pilot study. *Clinical Oral Implants Research 25* (6): 641–646. <https://doi.org/10.1111/clr.12149>.
- **91** Roccuzzo, M., Dalmasso, P., Pittoni, D., and Roccuzzo, A. (2019). Treatment of buccal soft tissue dehiscence around single implant: 5-year results from a prospective study. *Clinical Oral Investigations* 23 (4): 1977–1983. [https://doi.org/10.1007/s00784-018-2634-4.](https://doi.org/10.1007/s00784-018-2634-4)

Part III

Management of Peri-Implant Diseases

11

Periodontal Maintenance in Patients with Peri-implantitis

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Introduction

Regular professional supportive oral health care is considered central to controlling the progression of inflammatory periodontal and peri-implant disease. This chapter reviews the rationale, treatment approaches, and evidence for periodontal maintenance in patients with healthy implants and after active treatment for peri-implantitis.

Periodontal maintenance (formerly referred to as Supportive Periodontal Therapy) is defined as procedures performed at selected intervals to assist in maintaining oral health in individuals with a history of periodontitis. Periodontal maintenance procedures following active therapy are not synonymous with a prophylaxis [1]. Periodontal maintenance (PM) is started after completion of active periodontal therapy, both non-surgical and surgical therapies, and at varying intervals for the life of the dentition, including dental implants. Studies have demonstrated that periodontitis can be successfully treated by both non-surgical and surgical protocols [2] as part of active periodontal therapy. However, the benefits of active periodontal therapy cannot be maintained without a regular program of PM [3].

Maintenance procedures are under the supervision of the dentist and typically include an update of the medical and dental histories, radiographic review, extraoral and intraoral soft tissue examination, dental examination, periodontal evaluation, removal of supragingival and subgingival bacterial plaque (biofilm) and calculus, selective root planing where indicated, polishing of the teeth, and a review of the patient's plaque control efficacy [4, 5]. These procedures are performed at prescribed intervals established for periodic ongoing care to assist the patient in maintaining periodontal health. This phase of periodontal therapy focuses on the control of etiologic factors and monitoring of periodontal health status. It is distinct from, but integrated with, active periodontal therapy. Patients receiving PMT may require active therapy to control disease progression [6].

The traditional therapeutic goals of PM include (i) minimizing the recurrence and progression of periodontal disease in patients previously treated for gingivitis and periodontitis, (ii) reducing the incidence of tooth loss by monitoring the dentition and any prosthetic replacements of the natural teeth, and (iii) increasing the probability of locating and treating, in a timely manner, other diseases or conditions found within the oral cavity [7]. Since the goal of periodontal maintenance is to minimize the recurrence and progression of periodontitis, the authors propose that the protocols associated with traditional periodontal maintenance can be applied – with several specific considerations and modifications – to control the progression of peri-implantitis.

In this chapter, Implant Maintenance (IM) is used to distinguish treatment approaches for the control of periimplantitis from PMT for the control of periodontal infection. Three common clinical scenarios are summarized below that help guide questions related to the application of IM:

● **The patient without a history of periodontitis and peri-implant mucositis/peri-implantitis ("healthy" dental implants):** Do dental implants increase the risk of developing periodontal infection and disease – periodontitis or peri-implantitis?

- **The patient with a history of peri-implant mucositis**: Is the temporal progression of peri-implant mucositis to peri-implantitis similar to that of gingivitis to periodontitis?
- **The patient with a history of peri-implantitis**: (i) Is PM in a patient with periodontitis the same as IM in a patient with peri-implantitis? (ii) What is the role of IM in the patient with active peri-implantitis versus following active treatment of peri-implantitis?

In consideration of the above scenarios, this chapter will discuss the rationale for IM, elements of an IM appointment, and guidelines for the determination of maintenance intervals.

Modifying Factors and Considerations

The current model of disease progression for periodontitis is well-established and supported by decades of clinical evidence documenting the vital role of PM in controlling the progression of periodontitis – with lifelong maintenance, periodontitis can be stabilized for a vast majority of our patients [6]. With the increased use of dental implants to replace missing teeth, there has been a significant rise in prevalence of peri-implant disease, with estimated weighted mean prevalence of 43% for peri-implant mucositis and 22% for peri-implantitis [8, 9]. The risk profiles associated with the progression of peri-implant mucositis and peri-implantitis resemble the risk profile that drives the progression of gingivitis and periodontitis. Therefore, similar strategies have been adopted from periodontal protocols for implant maintenance therapy to prevent further breakdown. For example, without supportive therapy, implant sites with peri-implant mucositis are at risk for progression to peri-implantitis [10] and those sites with peri-implantitis risk further breakdown. Given the similarities in the clinical presentation of periodontitis and peri-implantitis, it is reasonable to conclude that similar approaches to supportive care after treatment of peri-implant disease should prove beneficial in controlling disease progression.

Multiple studies have investigated the impact of periodontal maintenance on the occurrence of peri-implant diseases [11–13]. As with periodontal maintenance, a primary focus is on identifying and addressing host-level and sitespecific (implant) factors in the personalized maintenance plan to an individual patient. The modifying factors and considerations of peri-implant disease progression will be presented, organized into two main categories for the reader: host-level and implant-level considerations.

Host-level

There are several host-level considerations identified in the literature that are essential to review when prescribing an

IM maintenance plan, such as a history of periodontitis, smoking, systemic diseases, e.g. type II diabetes mellitus, and poor plaque control [14].

Systemic Considerations

Although it might be intuitive to assume that systemic disease can have a significant impact on peri-implant health, the evidence on whether specific systemic conditions are a risk factor for peri-implant disease remains equivocal [14]. However, systemic conditions, such as uncontrolled diabetes, and environmental exposures, such as tobacco smoking and radiation therapy of the oral/head-neck region, are generally considered factors with the potential to modify peri-implant disease susceptibility and progression, including implant failure [6, 9, 15, 16].

Polymorphisms in genes for cytokines, such as IL-1 and IL-6, have been associated with an increase in the expression of these inflammatory cytokines and with the development of chronic diseases, such as periodontitis and peri-implant disease [17]. The latter findings suggest that genetic background may also predispose certain individuals to implant failure, consistent with observations of multiple implant failures (cluster failure behavior) of implants at the patient level. Consideration of potential risk factors is important for the targeted management of peri-implant disease and the long-term stabilization of peri-implant health [16, 17].

Additional host-level factors that should be considered when building a peri-implant maintenance plan are disturbances in cognitive function and advanced age [16, 18, 19]. There is evidence that loss of cognitive function and dementia negatively impact periodontal health due to an increase in plaque accumulation and bacterial burden; oral hygiene is further compromised from the barriers associated with cognitive decline and compromised dexterity [18]. Patients exhibiting a decline in cognition, dexterity, or both may benefit from shorter intervals between appointments, engagement of family or caretakers in oral hygiene care, and instruction on the use of oral hygiene devices, such as customized or electric toothbrushes and flossers, to help patients perform oral hygiene care. Contrast this situation with a patient who struggles with compliance because of their own lack of motivation or interest in oral hygiene. In this case, the patient is best treated with customized oral hygiene instruction that focuses on the justification for the emphasis on plaque control [6] to empower the patient to take responsibility for his/her oral health care ownership of factors within their control.

History of Periodontitis

Given that periodontitis and peri-implant disease share similar risk profiles for disease progression, maintenance therapy is considered essential to reduce disease recurrence and progression following active therapy [6, 20]. Not only does periodontitis share risk factors with periimplant disease, but a history of periodontitis appears to predispose individuals to peri-implant disease. Periimplantitis has been reported to be found twice as often in patients with periodontitis compared to those without periodontal disease; moreover, the extent of peri-implant breakdown was shown to parallel the severity of the preexisting periodontitis [14, 21, 22]. Clinical studies have reported that a history of periodontitis prior to implant placement or the presence of residual periodontal pockets at the time of implant placement were associated with a higher occurrence of implant loss and peri-implant diseases [11, 22–24]. Residual deep probing depth $(\geq 6$ mm) and marginal bone loss constituted risk factors for recurrence/progression of peri-implantitits. Furthermore, patients with an aggressive or molar/incisor pattern periodontitis may need additional active therapy following implant placement and should be also considered candidates for shorter recall intervals [10].

History of Peri-implant Mucositis

As with the progression of gingivitis to periodontitis, it is acknowledged that peri-implant mucositis can be a precursor to peri-implantitis, often progressing in an accelerated pattern [15]. Without supportive therapy, implant sites with peri-implant mucositis are at risk for progression to peri-implantitis [10]. In a five-year retrospective study in Brazil, patients diagnosed with peri-implant mucositis at baseline had a significantly higher incidence of periimplantitis (43.9% versus 18.0%) if they attended less than one visit to the dentist annually [11]. These results are in agreement with the findings of a systematic review, which found a higher prevalence of peri-implantitis in persons not receiving maintenance care compared to those undergoing routine follow-up care (\approx 35% versus \approx 10%, respectively) [5]. Patients in IM following active therapy for peri-implantitis must be evaluated closely for of signs of inflammation, which could indicate peri-implant mucositis or active peri-implantitis.

Implant-level

Bacterial Biofilm

Periodontal pockets are ideal environments for the establishment and maturation of subgingival biofilm [3]. The temporal relationship between inflammation, dysbiotic subgingival microbiota, and disease onset and progression remain incompletely understood [25]. Nevertheless, it is important to understand the dynamic role of the polymicrobial biofilm in disease pathogenesis [26]. Studies also highlight differences in bacterial profiles of healthy and diseased dental implants [27].

Socransky's landmark study of periodontal disease identified six bacterial complexes that commonly occur together. For descriptive purposes, these complexes were color-coded as blue, green, yellow, purple, orange, and red. The orange and red complexes are thought to be associated with periodontitis and include members of the yellow, green, and purple complexes [28]. The orange complex bacteria generally appear after the early colonizers and include many putative periodontal pathogens, such as *Fusobacterium nucleatum*. The red complex (*Porphyromonas gingivalis*, *Tannerella forsythia* [formerly *Bacteroides forsythus*], and *Treponema denticola*) is comprised of these late colonizers forming a stable, or climax, bacterial community that significantly drive periodontal destruction; the red complex bacteria are putative periodontal pathogens [29]. The subgingival/submarginal microbiota has been found to exhibit significant differences in composition between health and disease [28, 30, 31].

The development of gingivitis has been extensively studied in humans using a controlled paradigm to induce experimental gingivitis [32, 33]. The transition to gingivitis is initiated by inflammatory changes accompanied initially by the appearance of gram-negative rods and filaments and subsequently by spirochetal and motile microorganisms [34]. The analysis of subgingival/submucosal microbial samples from experimental gingivitis and peri-implant mucositis has revealed relatively small differences over time, with essentially no differences between gingivitis and peri-implant mucositis sites [35]. Nonetheless, it has been postulated that a prolonged exposure of implants to the bacterial challenge may yield quantitative and qualitative compositional changes of the inflammatory infiltrate due to the structural and immunologic differences between the gingiva around teeth and the mucosa around titanium implants. These changes include the density of collagen fibers and fibroblasts, collagen fiber orientation, and vascular structure, as well as expression of cell adhesion molecules and inflammatory cell populations [36–38]. Preexisting peri-implant mucositis, in conjunction with a lack of adherence to maintenance care, has been associated with a higher incidence of peri-implantitis [11]. Despite the fact that mucositis may be reversible, early diagnosis and appropriate management are clinically important.

Recent studies focused on the precise characterization of microbiome composition associated with periodontitis and peri-implantitis using 16s rRNA sequencing have shown that red complex pathogens can be found in patients and in periodontal sites in the absence of disease and that other bacterial species, some of which were not necessarily gramnegative (*Filifactor alocis*, *Peptostreptococcus*), appear associated with periodontal and peri-implant disease [28, 31, 39, 40]. Differences in the composition of the subgingival microbiota between periodontal and peri-implant diseases are discrete [41–43]. Orange complex microorganisms were

more frequent in peri-implantitis. *Porphyromonas gingivalis* was the most frequently found red complex organism in peri-implantitis followed by *T. forsythia*, *P. intermedia,* and *Prevotella nigrescens* [44]. In addition, surface properties of dental implants can influence the adhesion of cells and microbial colonizers and the surface modification limits the effectiveness of mechanical biofilm removal [45]. Although IM is performed as non-surgical therapy, some degree of surface decontamination, such as use of plastic or metal curettes designed to be used on implant surface, may need to be performed as part of IM [46].

Following active treatment, periodontal and peri-implant sites are subject to recurrent polymicrobial succession, maturation of a dysbiotic plaque biofilm, and increase in biomass of the microbiota. The rapidity of these changes in microbiota appears dependent on factors such as treatment protocol, the distribution patterns of periodontal microorganisms elsewhere in the oral cavity, and the effectiveness of the patient's oral hygiene [47]. Bacterial species from the red and orange complex showed a progressive trend toward expansion to pretreatment levels in periodontal sites at 42 days post-therapy. Two species from the orange complex have also been shown to return to pretreatment levels [48]. Despite the proven overall treatment effectiveness of subgingival biofilm removal, deep periodontal pockets may not revert rapidly or completely to a physiologic sulcus, with shallower probing depths. As a consequence, regular debridement by professional intervention is necessary to prevent recurrence of disease [47].

Prosthesis Design

Prosthesis design influences the risk for biologic complications as it can significantly impact access for oral hygiene care as well as access for effective IM [49, 50]. When the design of a prosthesis limits access to one or more of its surfaces, such as a tissue-borne bridge or long-span hybrid restoration with inadequate space for hygiene devices, the maintenance strategy will be more complex. For example, a two-implant supported three-unit bridge with ideal interdental distances and adequate space underneath the pontic enables the insertion of interdental brush, whereas a single implant–supported restoration with less than 1.5mm horizontal distance to adjacent tooth and more than 3mm height discrepancy (more apical) hampers a patient's access and ability to effectively clean the implant.

The emergence profile of the prosthesis of the implant/ abutment platform must have hygienic contours, and the inter-implant pontic sections should be reasonably selfcleansable [51]. The inability to adequately remove microbial biofilm, by the patient or oral health professional, has been associated with the development of peri-implant disease, thus proper prosthetic design plays an important role [52]. Convex emergence profiles have been found to be a risk factor for peri-implantitis [53]. Restorations must be contoured to function effectively while protecting and stimulating the gingival tissues. For single-unit implant restorations, a closed emergence angle $(>30^{\circ})$ has been shown to be associated with peri-implantitis in bone-level implants but not tissue-level implants. In natural teeth, using a straight emergence profile in the restoration design has been shown to improve the effectiveness of oral hygiene near the gingival sulcus and facilitates oral hygiene near the gingival sulcus [54]. These design characteristics also hold true for implants [55].

Peri-implant health and control of inflammatory disease may be influenced by physical access to perform proximal hygiene and by a patient's mechanical dexterity to perform oral hygiene [56]. Poorer access for proximal hygiene was associated with peri-implant diseases, especially periimplant mucositis. Lack of access to perform proximal hygiene measures was found to be negatively associated to peri-implant soft tissue conditions. Consequently, periimplant disease is associated with greater reported discomfort, pain, and difficulties while performing oral hygiene; moreover, patients generally viewed unfavorably the professional recommendations for oral hygiene care around implants. Importantly, therefore, resolution of peri-implant mucositis has been documented through modification of the prosthetic contour [57].

Ramfjord and Yuodelis have reported that overcontouring a prosthesis is a greater hazard to periodontal health than under-contouring a prosthesis [58, 59]. An over-contoured implant restoration is a possible a risk indicator of peri-implantitis. In addition, splinting adjacent implant–supported restorations could be another risk indicator. The prevalence of peri-implantitis of an implant splinted to both mesial and distal adjacent implants has been found to be 4.6-fold higher than for a single unit implant restoration [55].

The position of the individual implant should also be considered. Although formation of the biologic width around the dental implant is widely considered a biological phenomenon to create soft tissue seal around the abutment, studies have documented greater peri-implant probing depth around conventional implants placed subcrestally compared to implants placed at the crest or supracrestally [60, 61]. The difference in the height of the platform of the prosthesis might pose difficulty in accessing the interproximal area of the implant [62]. A deeper position of the implant results in a higher crown-to-implant ratio and a shallower emergence angle [55]. Implants placed at the subcrestal position displayed greater infra-osseous defects than those implants placed at the crestal position in a ligature induced peri-implantitis model in animal model [63].

Another factor associated with higher incidence of periimplantitis was encountered in sites where implants were positioned with <3mm inter-implant distance. Given adequate interproximal distance, this small distance can often be attributed to clinician error in implant positioning and placement. The design of the splinted restoration or bridge can impair effective oral hygiene; for example, concavities in the internal portion of the restorations must be avoided to reduce plaque accumulation and present mucosal irritation from impaired cleaning. In addition, oral hygiene performance has been shown to be impaired when the prosthesis contacts the mucosa, consistent with patient reports of mucosal irritation due to the lack of space for the use of the devices [64]. Insufficient inter-implant distance also influences the formation of "black triangle" in the interproximal space which may cause esthetic and functional problems including food impaction [65, 66].

Compared to single-unit restorations, full-mouth implant rehabilitations were found to pose a 16-time greater risk for peri-implantitis. The latter may be attributed to the difficulty in plaque removal by patients and dental professionals [49]. Full-arch fixed prosthesis supported by a number of implants, commonly four to six, can be made of a milled metal bar screwed directly onto implant abutments and a resin portion for the replacement of teeth and mucosal tissues [67]. Removal of a fixed, screw-retained implant prosthesis for evaluation is not needed unless there are signs of peri-implantitis, a demonstrated inability to maintain adequate oral hygiene, or there are mechanical complications that require removal [68]. However, ineffective oral hygiene around the prosthetic abutments related to difficulties in using oral hygiene devices is a common cause of chronic peri-implant mucositis and risk of periimplantitis. As part of a periodontal maintenance regimen, one should review the plaque index. Comparatively high plaque index scores have been observed on lingual surfaces of full arch restorations, which may be attributable to conformation of the prosthesis and to objective difficulties in reaching those surfaces using only interdental brushes [69]. In instances where prosthetic design hampers access for effective oral hygiene, periodic removal of the prosthesis may be considered (Figure 11.1).

In addition to the prosthetic considerations, other implant-level modifying factors to consider when designing an IM treatment plan include a lack of (or absence of) supportive keratinized tissue, the presence of deep probing depths, and the presence of foreign bodies such as titanium particles and remaining cement [14, 70–72]. Furthermore, implants with modified surfaces may warrant special attention, because surface modifications appear to predispose implants to disease progression [73]. A lack of keratinized tissue or a limited zone of

Figure 11.1 Mandibular implant-supported prosthesis design exhibiting poor access to underlying implants and supportive tissues.

keratinized tissue may also predispose implants to disease progression, warranting regular professional maintenance to promote peri-implant health [14]. If a lack of keratinized tissue or a limited zone of keratinized tissue is associated with pain and/or discomfort for a patient in performing oral hygiene, the clinician should implement a more targeted maintenance plan. Similarly, the exposure of implant threads due to peri-implantitis or active treatment may increase the risk of disease progression. A shorter maintenance interval is recommended once implant thread exposure is recognized, especially for rough surface implants since effective oral hygiene is difficult to achieve [74]. Even with surgical exposure (e.g. open flap debridement), the mechanical and chemical debridement of rough surface implants is difficult.

When designing an IM maintenance plan, there are multiple considerations to help guide the clinician in establishing an overall maintenance plan, including recall interval. The clinician must assess all host-level considerations, such as risk factors, systemic health status, compliance, and environmental modifiers, as well as implant-level considerations, such as prosthesis design, position, access, quality of supportive gingival tissues, and presence of foreign bodies.

Elements of a Implant Maintenance Appointment

PM and IM have a shared goal of controlling disease progression by regular surveillance of periodontal and periimplant health status and by reducing the biomass of the bacterial plaque. Thus, a practitioner familiar with the elements of a periodontal maintenance appointment will find

Figure 11.2 The elements of a supportive implant care at the maintenance appointment.

little difference with the elements of an IM maintenance appointment as presented below [68]. The dentist must monitor for adverse factors that persist despite treatment and maintenance as well as evaluate new findings that arise during the maintenance session, all of which will have an effect on the future IM regimen and interval (Figure 11.2).

Review of Health Status

At the start of each appointment, it is important to review with the patient changes to systemic health status; this includes but is not limited to updates to medical diagnoses, new or changed medications, surgeries, and procedures since the last visit, as well as updated laboratory results. The name and contact information for the patient's medical team (primary care manager, specialists, pharmacists, etc.) should also be confirmed. During this portion of the visit, new information that might shorten the maintenance interval include signs of systemic disease decline, such as worsening diabetic control as evident by rising HbA1c levels over 7%, or a new medical diagnosis that could impede a patient's home care, such as a cognitive or physical impairment. In contrast, if it is determined that the patient has had a considerable improvement in the control of their systemic disease, this may signal an opportunity to lengthen the interval between maintenance visits.

Review of Dental Status

It is equally important to review any changes to the patient's dental health status. This includes inquiring about visits to other dentists, procedures completed, and pending treatment plans. Likewise, confirm the current recall interval and other practitioners involved in the patient's dental treatment team – this is especially important in the case the patient splits maintenance appointments between two offices, such as between a general dentist and a periodontist.

Review Radiographs

In line with the review of dental status, the dentist should review radiographs and determine the need for updates to the radiographic record. A general rule for implant health surveillance is to obtain new radiographs yearly after implant loading. A change of greater than 0.5mm (the radiographic measurement error) indicates a need to act, whether it be to shorten the maintenance interval or to place the patient back into active therapy [15].

Oral Examination

The oral examination is the critical component of the IM maintenance appointment. The examination should include an oral cancer screening exam and the assessment of the color, consistency, and contour of the gingiva. Periodontal and peri-implant charting should be performed that includes probing depths, clinical attachment levels, mobility of teeth or implants, furcation involvement, assessment of keratinized gingiva/mucosa, presence of plaque and calculus, and bleeding on probing. Additionally, the evaluation includes an assessment of the color, consistency, and contour of the lining or masticatory mucosa at the interface of the implant. Data obtained from the examination that signals a need to shorten maintenance interval or to return the patient to active therapy would be probing depths greater than or equal to 6mm, increased plaque retention, increased bleeding on probing, and other clinical signs of a worsening status.

Oral Hygiene Assessment

During the examination, the dentist should be assessing the patient's home care effectiveness and compliance by the presence of plaque and calculus. Attention should be paid to the location of the plaque as a guide to where the patient needs to improve their oral hygiene efficiency. Patients who are struggling with effective home care despite targeted oral hygiene instructions are candidates for shorter maintenance intervals.

Biofilm Removal

Once the examination and assessment portion of the IM appointment is complete, the clinician should complete a full mouth biofilm removal. For areas with no loss of attachment or signs of disease progression, this may be completed with supragingival polish. In areas of deep or advancing probing depths, scaling and root planing of the implant surface is advised. For implant sites it is important to pay special attention to the instrument type used, implant surfaces, and access for biofilm removal. Surface modification of dental implants contributes more bacterial adhesion and biofilm formation compared to natural teeth. This requires mechanical debridement like scaling to eliminate bacteria from the site of inflammation, the use of additional antiseptic treatment to disinfect the implant surface, and the application of antibiotics to remove bacteria in the surrounding peri-implant tissues [75].

During this portion of the appointment, the dentist should also assess the need for prosthesis removal. As previously mentioned, the American College of Prosthodontists's position paper states that removal of screw-retained implant fixed prostheses for evaluation is not needed unless there are signs of peri-implantitis, a demonstrated inability to maintain adequate oral hygiene, or there are mechanical complications that require removal (Figure 11.3) [51]. The latter guidance is consistent with

Figure 11.3 Implant-supported prothesis with biofilm build-up despite design allowing for home care. *Source:* Courtesy of Keith Merchant, DDS, MS and David Bell DDS, MS.

the management of periodontal disease, when periodic removal of the prosthesis is necessary to perform effective mechanical debridement and root planing. The periodic removal of the prosthesis should also be performed to facilitate IM, although removal may not be indicated at every maintenance visit.

Methods for Biofilm Removal During Implant Maintenance

The removal of supra- and subgingival biofilm deposits is a critical component of professional maintenance visits, with recall intervals determined by multiple clinical considerations, including risk factors. Disruption of the biofilms is most commonly achieved by mechanical intervention. In implant sites that exhibit no inflammation and are in peri-implant health, treatment should be limited to coronal polishing. Subgingival instrumentation is generally indicated at implant sites exhibiting bacterial deposits, mucosal inflammation, or signs of peri-implantitis.

Mechanical Methods

A broad selection of mechanical instruments is available for use in the non-surgical maintenance of implants at risk for disease progression. This list of instruments includes curettes, scalers, and modified piezoelectric or ultrasonic tips. It is widely considered a best practice to employ instruments that are manufactured expressly for use against an implant surface composed of materials that minimize the risk of damaging the implant surface, such as plastics, graphite, or titanium. Unintentional modifications of the implant surface can hamper plaque control and, thereby, increase risk of disease progression [6]. Additional instruments available for the mechanical debridement of implant surfaces include titanium brushes, lasers, and glycine

air-abrasives [76]. However, these options may not be practical considering maintenance therapy is conducted in a closed-scaling environment [77, 78].

Chemical Adjuncts

A variety of chemical agents can be used to aid in biofilm removal during IM, including chlorhexidine, ethylenediaminetetraacetic acid (EDTA), povidone-iodine, and hydrogen peroxide [76, 77, 79]. The clinician can also consider the use of a locally delivered antibiotic. In a six-month study of a maintenance program for implants following surgical treatment of peri-implantitis, topically applied minocycline gel improved clinical and radiographic parameters [80]. A recent multicenter randomized study conducted by Machtei et al. showed the efficacy of a Chlorhexidine-containing slow-release device (PerioChip) in terms of pocket reduction and clinical attachment gain when used as an adjunct to biweekly supragingival debridement for 12 weeks as compared to supragingival debridement alone [81]. Emerging evidence on the local administration of antimicrobials for the treatment of periimplant disease is encouraging; however, no commercially available antimicrobials in the United States have received regulatory approval for the treatment of peri-implant diseases. Clinicians should exercise careful professional judgment in the off-market clinical application of antimicrobial agents or compounds.

Miscellaneous Adjuncts

To improve access and ease of biofilm removal during IM appointments, the dentist might consider the adjunctive application of lasers (Nd:Yag, Er:YAG, Er,Cr:YSGG, Diodes) to boost disinfection and endoscopes to improve visualization of calculus and other foreign objects [18, 78, 82]. However, long-term outcomes have not been well-established following laser therapy during regular maintenance care for the treatment of peri-implant disease [83].

Oral Hygiene Instruction

Upon completion of biofilm removal, the patient should be instructed on appropriate, customized oral hygiene instructions. Emphasis should be placed on oral hygiene techniques and specific implant sites that would benefit from improvement. A toothbrush and dental floss remain the most widely recommended oral hygiene devices for patients. Many patients will have a need for additional hygiene adjuncts, such as floss threaders, specialized stiffended floss, interdental brushes, end-tufted brushes, sulcular brushes, and irrigating devices. The need for these adjunct devices will depend on the patient's dexterity, location of troubled areas, and the impact the prosthesis has on patient access.

Assess the Effectiveness of the Current Maintenance Plan

Intricately linked to all the elements and steps of a maintenance appointment is evaluation of the effectiveness of the maintenance recall interval. As stated throughout this chapter, in addition to evidence of poor oral hygiene effectiveness or compliance, other factors such as systemic disease status and evidence of disease progression will shorten or lengthen the recall interval. The next section will discuss specific guidelines about setting maintenance intervals.

Interval and Considerations

Clinicians often recommend an initial maintenance interval of three months for patients with a history of periodontitis. However, the appropriate maintenance interval is not the same for all patients. A five-year follow-up study demonstrated the importance of IM to prevent peri-implant mucositis progressing to peri-implantitis [11]. The prevalence of peri-implantitis (PD≥5mm, bleeding on probing and/or suppuration, and bone loss) was found to be higher among patients not in professional maintenance care (43.9%) compared to those receiving regular supportive care (18%). Longitudinal clinical results from treated and maintained populations, as well as data from randomized controlled clinical trials, show that supportive periodontal care at three- to four-month intervals is sufficient to control disease in most patients [6, 7, 84, 85]. However, shorter or longer maintenance intervals may be appropriate depending on risk assessment. The clinician must customize the professional maintenance interval to the patient, with the goal of achieving long-term outcomes in peri-implant health.

Host-level Considerations

The evidence from periodontal literature suggests that, in general, patients previously treated for chronic periodontitis and/or peri-implantitis appear to require more frequent periodontal maintenance. An increase in susceptibility to peri-implantitis also has been reported in individuals with a history of periodontitis [10]. A relatively short maintenance interval of three to four months is based, in part, on the risk for disease progression. A recent five-year prospective study assessing the risk for recurrence/progression of peri-implantitis following surgical therapy showed 44% of implants displayed recurrence/progression of disease during the follow-up period [73]. In addition, longitudinal clinical results from treated-and-maintained populations, and data from randomized controlled clinical trials, show that supportive periodontal care at three- to four-month intervals is effective in controlling disease progression in most persons [6, 7, 84, 85].

Additional host-level considerations that may guide the recommendation to shorten the maintenance interval include systemic considerations, such as uncontrolled diabetes, smoking, and head and neck radiation [6,14–16, 20, 86], as well as patient age and loss of cognitive or physical function impacting home care [18]. Patient compliance with home care instructions or effectiveness in oral hygiene should be considered in developing a maintenance program. Changes in host-level factors, such as glycemic control or radiation therapy, must be considered in determining the interval of IM [16, 87].

Implant-level Considerations

As we have discussed above, complexity in the biofilm removal and prosthetics design as well as other implantlevel factors, such as a lack of keratinized tissue, the presence of exposed or modified implant threads, as well as clinical signs of unstable parameters like residual probing depths greater than 5mm and unresolved inflammation, can be used to set the baseline for the MI interval [6].

The decision algorithm can be considered a two-step process.

First, determine whether either (i) the prosthesis impedes the patient's access to perform home care or (ii) if the residual probing depth(s) limits the professional care. Second, determine whether systemic factors, such as diabetes, neurologic or musculoskeletal diseases, or local/implant-level factors, such as pontic design or a lack of keratinized gingiva are present and their subsequent impact on maintenance success. The final recommendation must be informed by consideration of all relevant local and systemic factors. In certain instances, the presence of one factor (such as need of periodical removal of the prosthesis) may drive the decision.

When a Return to Active Therapy is Warranted

The purpose of IM is to maintain peri-implant health as well as to recognize the development and progression of periimplantitis; clinical findings may present early during followup indicating a return to a diseased state of peri-implantitis. In addition, untreated peri-implantitis appears to progress in a non-linear, accelerating pattern, and at a faster rate than observed in periodontitis [8]. Persistent residual probing depths ≥6mm is a predictor for additional bone loss in patients diagnosed with peri-implantitis [73]. For this reason, a radiographic exam should be completed to evaluate bone levels for evidence of new loss. If a pattern of continuous bone loss is established, a problem-focused procedure such as local debridement should be performed at the time of IM. The latter also holds true with the return of other clinical signs such as thread exposure, bleeding and suppuration on probing, swelling of the peri-implant soft tissues, and symptoms of pain and discomfort. After completion of focused treatment and re-evaluation, the clinician should consider initiating a new round of active implant therapy.

Applying These Guidelines to Three Clinical Scenarios

The Patient Without a History of Periodontitis and Peri-implant Mucositis/Periimplantitis ("Healthy" Dental Implants)

One may start with four to six months interval of IM. However, with the reported increased risk of developing periodontal infection and disease, the patient with host level (e.g. uncontrolled diabetes or smoking) and/or implant level risk factors (e.g. prosthesis design that limits access) will need close monitoring of the disease development, especially in patients receiving implant therapy for the first time.

The Patient with a History of Peri-implant Mucositis

Studies have shown that good plaque control is associated with peri-implant health and peri-implant mucositis can be successfully treated by control of biofilm [4]. Although peri-implant mucositis may be reversible, with known faster progression from peri-implant mucositis to peri-implantitis, three to four months interval of IM is

recommended. This interval can be modified further with host level (e. g. the patient with limited manual dexterity) and/or implant level risk factors, such as the prosthesis limits access or a limited amount of KG, to prevent and monitor the progression to peri-implantitis.

The Patient with a History of Peri-implantitis

Periodontal surgery with regular follow-up care can delay further periodontal destruction although it may not prevent it completely [6]. In this patient population, the current status of the implant sites needs to be evaluated carefully. With the resolution of the peri-implantitis (i.e. reduced probing depths and/or arrested bone loss), one may start on a three months interval and use the IM as an opportunity for periodic monitoring with "regular" course of biofilm removal. However, host-level factors such as dexterity or cognitive impairment, and/or implant level factors that may decrease the effectiveness of the IM such as exposed thread, a more "proactive" or additional

element of IM, including removal of the prosthesis periodically but not necessarily at every visit, may need to be performed to establish the effectiveness (Figure 11.4).

In patients with implants without the resolution of peri-implantitis after their active therapy, i.e. residual PD≥5mm, it may lead to additional visits between the MI appointments for observation or a temporary shortening interval. The addition of a problem-focused procedure such as local debridement at the time of IM should be considered (Figure 11.5). After completion of focused treatment and re-evaluation, the clinician should consider initiating a new round of active implant therapy.

Summary

Regular professional supportive therapy is considered key to controlling the progression of periodontal and peri-implant disease. Emerging evidence underscores the critical role of IM in the long-term outcome of dental implants, particularly in persons with a history of periodontitis, peri-implantitits, or both. With increased complexity in the structure of dental implants and restorations, when compared to the natural dentition, a customized interval of IM and close monitoring are recommended to achieve long-term success. In patients without history of peri-implantitis, IM allows for the

References

- **1** American Academy of P. Glossary of Periodontal Terms. <https://members.perio.org/libraries/glossary>
- **2** Harrel, S.K. and Nunn, M.E. (2001). Longitudinal comparison of the periodontal status of patients with moderate to severe periodontal disease receiving no treatment, non-surgical treatment, and surgical treatment utilizing individual sites for analysis. *Journal of Periodontology* 72: 1509–1519.
- **3** Costa, F.O., Vieira, T.R., Cortelli, S.C. et al. (2018). Effect of compliance during periodontal maintenance therapy on levels of bacteria associated with periodontitis: a 6-year prospective study. *Journal of Periodontology* 89: 519–530.
- **4** Rosing, C.K., Fiorini, T., Haas, A.N. et al. (2019). The impact of maintenance on peri-implant health. *Brazilian Oral Research* 33: e074.
- **5** Monje, A., Aranda, L., Diaz, K.T. et al. (2016). Impact of maintenance therapy for the prevention of peri-implant diseases: a systematic review and meta-analysis. *Journal of Dental Research* 95: 372–379.
- **6** Armitage, G.C. and Xenoudi, P. (2016). Post-treatment supportive care for the natural dentition and dental implants. *Periodontology 2000* 71: 164–184.
- **7** (2000). Parameter on periodontal maintenance. American Academy of Periodontology. *Journal of Periodontology* 71: 849–850.<https://pubmed.ncbi.nlm.nih.gov/10875688>.
- **8** Derks, J. and Tomasi, C. (2015). Peri-implant health and disease. A systematic review of current epidemiology. *Journal of Clinical Periodontology* 42 (Suppl 16): S158–S171.
- **9** Fu, J.H. and Wang, H.L. (2020). Breaking the wave of peri-implantitis. *Periodontology 2000* 84: 145–160.
- **10** Jepsen, S., Berglundh, T., Genco, R. et al. (2015). Primary prevention of peri-implantitis: managing peri-implant mucositis. *Journal of Clinical Periodontology* 42 (Suppl 16): S152–S157.

early identification and management of peri-implant mucositis or peri-implantitis.

Consideration of each risk factor is critical in developing a customized maintenance program and interval, especially for patients with a history of periodontitis, periimplantitis, or both.

Patients who choose dental implant therapy to replace lost dental function should be carefully informed and educated on the importance IM following their treatment. This conversation should start early in treatment planning prior to commencing treatment, and the importance of adherence to the IM protocol should be continuously emphasized and reinforced over time.

- **11** Costa, F.O., Takenaka-Martinez, S., Cota, L.O. et al. (2012). Peri-implant disease in subjects with and without preventive maintenance: a 5-year follow-up. *Journal of Clinical Periodontology* 39: 173–181.
- **12** Pjetursson, B.E., Thoma, D., Jung, R. et al. (2012). A systematic review of the survival and complication rates of implant-supported fixed dental prostheses (FDPs) after a mean observation period of at least 5 years. *Clinical Oral Implants Research* 23 (Suppl 6): 22–38.
- **13** Roos-Jansaker, A.M., Lindahl, C., Renvert, H., and Renvert, S. (2006). Nine- to fourteen-year follow-up of implant treatment. Part I: implant loss and associations to various factors. *Journal of Clinical Periodontology* 33: 283–289.
- **14** Schwarz, F., Derks, J., Monje, A., and Wang, H.L. (2018). Peri-implantitis. *Journal of Periodontology* 89 (Suppl 1): S267–S290.
- **15** Berglundh, T., Armitage, G., Araujo, M.G. et al. (2018). 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* 89 (Suppl 1): S313–S318.
- **16** Heitz-Mayfield, L.J., Aaboe, M., Araujo, M. et al. (2018). Group 4 ITI consensus report: risks and biologic complications associated with implant dentistry. *Clinical Oral Implants Research* 29 (Suppl 16): 351–358.
- **17** Campos, M.I., Godoy dos Santos, M.C., Trevilatto, P.C. et al. (2005). Interleukin-2 and interleukin-6 gene promoter polymorphisms, and early failure of dental implants. *Implant Dentistry* 14: 391–396.
- **18** Persson, G.R. (2018). Periodontal complications with age. *Periodontology 2000* 78: 185–194.
- **19** Sekundo, C., Langowski, E., Kilian, S., and Frese, C. (2020). Periodontal and peri-implant diseases in centenarians. *Journal of Clinical Periodontology* 47: 1170–1179.
- **20** Heitz-Mayfield, L.J.A., Salvi, G.E., Mombelli, A. et al. (2018). Supportive peri-implant therapy following anti-infective surgical peri-implantitis treatment: 5-year survival and success. *Clinical Oral Implants Research* 29: 1–6.
- **21** Mombelli, A., Muller, N., and Cionca, N. (2012). The epidemiology of peri-implantitis. *Clinical Oral Implants Research* 23 (Suppl 6): 67–76.
- **22** Roccuzzo, M., Bonino, F., Aglietta, M., and Dalmasso, P. (2012). Ten-year results of a three arms prospective cohort study on implants in periodontally compromised patients. Part 2: clinical results. *Clinical Oral Implants Research* 23: 389–395.
- **23** Pjetursson, B.E., Helbling, C., Weber, H.P. et al. (2012). Peri-implantitis susceptibility as it relates to periodontal therapy and supportive care. *Clinical Oral Implants Research* 23: 888–894.
- **24** Hardt, C.R., Grondahl, K., Lekholm, U., and Wennstrom, J.L. (2002). Outcome of implant therapy in relation to experienced loss of periodontal bone support: a retrospective 5-year study. *Clinical Oral Implants Research* 13: 488–494.
- **25** Van Dyke, T.E., Bartold, P.M., and Reynolds, E.C. (2020). The nexus between periodontal inflammation and dysbiosis. *Frontiers in Immunology* 11: 511.
- **26** Colombo, A.P., Sakellari, D., Haffajee, A.D. et al. (1998). Serum antibodies reacting with subgingival species in refractory periodontitis subjects. *Journal of Clinical Periodontology* 25: 596–604.
- **27** Perez-Chaparro, P.J., Duarte, P.M., Shibli, J.A. et al. (2016). The current weight of evidence of the microbiologic profile associated with peri-implantitis: a systematic review. *Journal of Periodontology* 87: 1295–1304.
- **28** Socransky, S.S. and Haffajee, A.D. (1992). The bacterial etiology of destructive periodontal disease: current concepts. *Journal of Periodontology* 63 (Suppl 4S): 322–331.
- **29** Dewhirst, F.E., Chen, T., Izard, J. et al. (2010). The human oral microbiome. *Journal of Bacteriology* 192: 5002–5017.
- **30** Aas, J.A., Paster, B.J., Stokes, L.N. et al. (2005). Defining the normal bacterial flora of the oral cavity. *Journal of Clinical Microbiology* 43: 5721–5732.
- **31** Mason, M.R., Preshaw, P.M., Nagaraja, H.N. et al. (2015). The subgingival microbiome of clinically healthy current and never smokers. *The ISME Journal* 9: 268–272.
- **32** Theilade, E. (1996). The experimental gingivitis studies: the microbiological perspective. *Journal of Dental Research* 75: 1434–1438.
- **33** Theilade, E., Wright, W.H., Jensen, S.B., and Loe, H. (1966). Experimental gingivitis in man. II. A

longitudinal clinical and bacteriological investigation. *Journal of Periodontal Research* 1: 1–13.

- **34** Salvi, G.E., Aglietta, M., Eick, S. et al. (2012). Reversibility of experimental peri-implant mucositis compared with experimental gingivitis in humans. *Clinical Oral Implants Research* 23: 182–190.
- **35** Listgarten, M.A. and Hellden, L. (1978). Relative distribution of bacteria at clinically healthy and periodontally diseased sites in humans. *Journal of Clinical Periodontology* 5: 115–132.
- **36** Berglundh, T., Lindhe, J., Ericsson, I. et al. (1991). The soft tissue barrier at implants and teeth. *Clinical Oral Implants Research* 2: 81–90.
- **37** Berglundh, T., Lindhe, J., Jonsson, K., and Ericsson, I. (1994). The topography of the vascular systems in the periodontal and peri-implant tissues in the dog. *Journal of Clinical Periodontology* 21: 189–193.
- **38** Salvi, G.E., Cosgarea, R., and Sculean, A. (2017). Prevalence and mechanisms of peri-implant diseases. *Journal of Dental Research* 96: 31–37.
- **39** Kumar, P.S., Mason, M.R., Brooker, M.R., and O'Brien, K. (2012). Pyrosequencing reveals unique microbial signatures associated with healthy and failing dental implants. *Journal of Clinical Periodontology* 39: 425–433.
- **40** Faveri, M., Figueiredo, L.C., Shibli, J.A. et al. (2015). Microbiological diversity of peri-implantitis biofilms. *Advances in Experimental Medicine and Biology* 830: 85–96.
- **41** Persson, G.R. and Renvert, S. (2014). Cluster of bacteria associated with peri-implantitis. *Clinical Implant Dentistry and Related Research* 16: 783–793.
- **42** Mougeot, J.L., Stevens, C.B., Cotton, S.L. et al. (2016). Concordance of HOMIM and HOMINGS technologies in the microbiome analysis of clinical samples. *Journal of Oral Microbiology* 8: 30379.
- **43** Schincaglia, G.P., Hong, B.Y., Rosania, A. et al. (2017). Clinical, immune, and microbiome traits of gingivitis and peri-implant mucositis. *Journal of Dental Research* 96: 47–55.
- **44** Lafaurie, G.I., Sabogal, M.A., Castillo, D.M. et al. (2017). Microbiome and microbial biofilm profiles of periimplantitis: a systematic review. *Journal of Periodontology* 88: 1066–1089.
- **45** Han, A. (2016). Bacterial adhesion mechanisms on dental implant surfaces and the influencing factors. *International Journal of Adhesion and Adhesives* 69: 58–71.
- **46** Schwarz, F., Hegewald, A., John, G. et al. (2013). Fouryear follow-up of combined surgical therapy of advanced peri-implantitis evaluating two methods of surface decontamination. *Journal of Clinical Periodontology* 40: 962–967.
- **47** Mombelli, A. (2018). Microbial colonization of the periodontal pocket and its significance for periodontal therapy. *Periodontology 2000* 76: 85–96.
- **48** Feres, M., Bernal, M., Matarazzo, F. et al. (2015). Subgingival bacterial recolonization after scaling and root planing in smokers with chronic periodontitis. *Australian Dental Journal* 60: 225–232.
- **49** Dalago, H.R., Schuldt Filho, G., Rodrigues, M.A. et al. (2017). Risk indicators for peri-implantitis. A crosssectional study with 916 implants. *Clinical Oral Implants Research* 28: 144–150.
- **50** Curtis, D.A., Lin, G.H., Fishman, A. et al. (2019). Patient-centered risk assessment in implant treatment planning. *The International Journal of Oral & Maxillofacial Implants* 34: 506–520.
- **51** American College of Prosthodontists. Maintenance of Full-Arch Implant Restorations. 2016. [https://www.](https://www.prosthodontics.org/assets/1/7/Maintenance_of_Full-Arch_Implant_Restorations.pdf) [prosthodontics.org/assets/1/7/Maintenance_of_Full-](https://www.prosthodontics.org/assets/1/7/Maintenance_of_Full-Arch_Implant_Restorations.pdf)[Arch_Implant_Restorations.pdf](https://www.prosthodontics.org/assets/1/7/Maintenance_of_Full-Arch_Implant_Restorations.pdf)
- **52** Serino, G. and Strom, C. (2009). Peri-implantitis in partially edentulous patients: association with inadequate plaque control. *Clinical Oral Implants Research* 20: 169–174.
- **53** Katafuchi, M., Weinstein, B.F., Leroux, B.G. et al. (2018). Restoration contour is a risk indicator for periimplantitis: a cross-sectional radiographic analysis. *Journal of Clinical Periodontology* 45: 225–232.
- **54** Croll, B.M. (1990). Emergence profiles in natural tooth contour. Part II: clinical considerations. *The Journal of Prosthetic Dentistry* 63: 374–379.
- **55** Yi, Y., Koo, K.T., Schwarz, F. et al. (2020). Association of prosthetic features and peri-implantitis: a crosssectional study. *Journal of Clinical Periodontology* 47: 392–403.
- **56** Pons, R., Nart, J., Valles, C. et al. (2021). Self-administered proximal implant-supported hygiene measures and the association to peri-implant conditions. *Journal of Periodontology* 92: 389–399.
- **57** Monje, A., Pons, R., Insua, A. et al. (2019). Morphology and severity of peri-implantitis bone defects. *Clinical Implant Dentistry and Related Research* 21: 635–643.
- **58** Ramfjord, S.P. (1974). Periodontal aspects of restorative dentistry. *Journal of Oral Rehabilitation* 1: 107–126.
- **59** Yuodelis, R.A., Weaver, J.D., and Sapkos, S. (1973). Facial and lingual contours of artificial complete crown restorations and their effects on the periodontium. *The Journal of Prosthetic Dentistry* 29: 61–66.
- **60** Saito, H., Aichelmann-Reidy, M.B., and Oates, T.W. (2020). Advances in implant therapy in North America: improved outcomes and application in the compromised dentition. *Periodontology 2000* 82: 225–237.
- **61** Atsuta, I., Ayukawa, Y., Kondo, R. et al. (2016). Soft tissue sealing around dental implants based on histological interpretation. *Journal of Prosthodontic Research* 60: 3–11.
- **62** Abrahamsson, I., Berglundh, T., Moon, I.S., and Lindhe, J. (1999). Peri-implant tissues at submerged and nonsubmerged titanium implants. *Journal of Clinical Periodontology* 26: 600–607.
- **63** Huang, B., Zhang, L., Xu, L. et al. (2018). Effect of implant placement depth on the peri-implant bone defect configurations in ligature-induced peri-implantitis: an experimental study in dogs. *Medicina Oral, Patologia Oral y Cirugia Bucal* 23: e30–e37.
- **64** Schuldt Filho, G., Dalago, H.R., Oliveira de Souza, J.G. et al. (2014). Prevalence of peri-implantitis in patients with implant-supported fixed prostheses. *Quintessence International* 45: 861–868.
- **65** Tarnow, D., Elian, N., Fletcher, P. et al. (2003). Vertical distance from the crest of bone to the height of the interproximal papilla between adjacent implants. *Journal of Periodontology* 74: 1785–1788.
- **66** Jeong, J.S. and Chang, M. (2015). Food impaction and periodontal/peri-implant tissue conditions in relation to the embrasure dimensions between implant-supported fixed dental prostheses and adjacent teeth: a crosssectional study. *Journal of Periodontology* 86: 1314–1320.
- **67** Francetti, L., Corbella, S., Taschieri, S. et al. (2015). Medium- and long-term complications in full-arch rehabilitations supported by upright and tilted implants. *Clinical Implant Dentistry and Related Research* 17: 758–764.
- **68** Bidra, A.S., Daubert, D.M., Garcia, L.T. et al. (2016). Clinical practice guidelines for recall and maintenance of patients with tooth-borne and implant-borne dental restorations. *Journal of Prosthodontics: Official Journal of the American College of Prosthodontists* 25 (Suppl 1): S32–S40.
- **69** Corbella, S., Del Fabbro, M., Taschieri, S. et al. (2011). Clinical evaluation of an implant maintenance protocol for the prevention of peri-implant diseases in patients treated with immediately loaded full-arch rehabilitations. *International Journal of Dental Hygiene* 9: 216–222.
- **70** Wilson, T.G. Jr. (2009). The positive relationship between excess cement and peri-implant disease: a prospective clinical endoscopic study. *Journal of Periodontology* 80: 1388–1392.
- **71** Wilson, T.G. Jr. (2021). Bone loss around implants-is it metallosis? *Journal of Periodontology* 92: 181–185.
- **72** Safioti, L.M., Kotsakis, G.A., Pozhitkov, A.E. et al. (2017). Increased levels of dissolved titanium are associated with peri-implantitis – a cross-sectional study. *Journal of Periodontology* 88: 436–442.
- **73** Carcuac, O., Derks, J., Abrahamsson, I. et al. (2020). Risk for recurrence of disease following surgical therapy of peri-implantitis-A prospective longitudinal study. *Clinical Oral Implants Research* 31: 1072–1077.
- **74** Renvert, S., Polyzois, I., and Claffey, N. (2011). How do implant surface characteristics influence peri-implant disease? *Journal of Clinical Periodontology* 38: 214–222.
- **75** Lang, N.P. and Tonetti, M.S. (1996). Periodontal diagnosis in treated periodontitis. Why, when and how to use clinical parameters. *Journal of Clinical Periodontology* 23: 240–250.
- **76** John, G., Sahm, N., Becker, J., and Schwarz, F. (2015). Nonsurgical treatment of peri-implantitis using an air-abrasive device or mechanical debridement and local application of chlorhexidine. Twelve-month follow-up of a prospective, randomized, controlled clinical study. *Clinical Oral Investigations* 19: 1807–1814.
- **77** Garaicoa, J.L., Bates, A.M., Avila-Ortiz, G., and Brogden, K.A. (2020). Antimicrobial prosthetic surfaces in the oral cavity–a perspective on creative approaches. *Microorganisms* 8: 1247.
- **78** Lin, G.H., Suarez Lopez Del Amo, F., and Wang, H.L. (2018). Laser therapy for treatment of peri-implant mucositis and peri-implantitis: an American Academy of Periodontology best evidence review. *Journal of Periodontology* 89: 766–782.
- **79** Serino, G., Turri, A., and Lang, N.P. (2015). Maintenance therapy in patients following the surgical treatment of peri-implantitis: a 5-year follow-up study. *Clinical Oral Implants Research* 26: 950–956.
- **80** Cha, J.K., Lee, J.S., and Kim, C.S. (2019). Surgical therapy of peri-implantitis with local minocycline: a 6-month

randomized controlled clinical trial. *Journal of Dental Research* 98: 288–295.

- **81** Machtei, E.E., Romanos, G., Kang, P. et al. (2021). Repeated delivery of chlorhexidine chips for the treatment of peri-implantitis: a multicenter, randomized, comparative clinical trial. *Journal of Periodontology* 92: 11–20.
- **82** Mettraux, G.R., Sculean, A., Burgin, W.B., and Salvi, G.E. (2016). Two-year clinical outcomes following non-surgical mechanical therapy of peri-implantitis with adjunctive diode laser application. *Clinical Oral Implants Research* 27: 845–849.
- **83** Mills, M.P., Rosen, P.S., Chambrone, L. et al. (2018). American Academy of Periodontology best evidence consensus statement on the efficacy of laser therapy used alone or as an adjunct to non-surgical and surgical treatment of periodontitis and peri-implant diseases. *Journal of Periodontology* 89: 737–742.
- **84** Chambrone, L.A. and Chambrone, L. (2006). Tooth loss in well-maintained patients with chronic periodontitis during long-term supportive therapy in Brazil. *Journal of Clinical Periodontology* 33: 759–764.
- **85** Hirschfeld, L. and Wasserman, B. (1978). A long-term survey of tooth loss in 600 treated periodontal patients. *Journal of Periodontology* 49: 225–237.
- **86** Fu, J.H., Yeh, C.Y., Chan, H.L. et al. (2010). Tissue biotype and its relation to the underlying bone morphology. *Journal of Periodontology* 81: 569–574.
- **87** Khandelwal, N., Oates, T.W., Vargas, A. et al. (2013). Conventional SLA and chemically modified SLA implants in patients with poorly controlled type 2 diabetes mellitus – a randomized controlled trial. *Clinical Oral Implants Research* 24: 13–19.

Surgical Treatment of Peri-implantitis

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The surgical treatment of peri-implantitis is a complex surgical procedure and a clinical challenge in today's daily practice. The surgical approach seems not to be complicated and a straightforward process for a well-skilled and competent clinician, but the clinical outcome is not always predictable. A comprehensive review of the literature with the different studies performed for the treatment of periimplantitis was recently published [1]. Different etiological factors are associated with the disease and especially the disease progression.

Peri-implantitis is defined as a biofilm-associated pathological condition characterized by inflammation in the periimplant mucosa and subsequent progressive bone loss of supporting bone [2]. However, biofilm accumulation is dependent on the implant surface characteristics, the implant macro-geometry [3], the type of the prosthesis as well as other local and systemic factors (smoking, diabetes, metalosis, etc.) contributing to the disease progression [4]. In addition, inadequate maintenance, and oral hygiene (Figure 12.1) are risk indicators for peri-implantitis.

The non-surgical treatment, which consists of mechanical debridement, the use of ultrasonics or laser devices, alone or combined with antiseptic and/or antibiotic agents, has not demonstrated the resolution of the disease due to inconsistent results according to the current literature. In contrast to this therapy, surgical therapy, in conjunction with the implant surface detoxification and debridement of peri-implant tissues, as a regenerative or resective approach, has shown greater effectiveness in terms of disease resolution as demonstrated by preclinical and clinical studies. Surgical augmentative peri-implantitis therapy

resulted in improved clinical and radiographic treatment outcomes compared with the baseline in most studies with six months to 7–10 years of follow-up [5].

There is no reliable evidence suggesting which could be the most effective intervention for treating periimplantitis [6] and no single method can be promoted based on the evidence [7, 8]. Evaluating the different published surgical treatment methods, a recent review of the literature concluded that the available evidence to support the superiority of augmentative surgical techniques for peri-implantitis management on the treatment outcomes over non-augmentative methods is limited [9]. There is no "gold standard," and several techniques have been recommended to treat peri-implantitis, from non-surgical to complex resective and regenerative surgical procedures, which aim at preventing inflammation and encouraging the regeneration of hard and soft tissues surrounding the implant [10].

The treatment is focused on detoxification of the implant surface and surrounding tissues with finally the grafting.

Different detoxification methods of the implant surface have been reported in the literature, such as chlorhexidine [11], hydrogen peroxide 3% [12], 24% EDTA [13], tetracycline powder [14, 15] (Figure 12.2), air-polishing with sodium bicarbonate and glycine [14] and recently the use of an electrolytic cleaning in conjunction with bone grafting [16].

Also, laser irradiation for implant surface detoxification like the use of Er:YAG laser ($[17-21]$), CO₂ laser $[22-25]$, Er,Cr:YSGG [26], 810nm diode laser [27] or a high power 980nm laser [28] have been utilized. Additional options have been reported, like a low-power 980nm diode laser [29] and photodynamic therapy [30] (see also chapter 13).

The peri-implant bone defect debridement, methods of implant surface decontamination, and detoxification

With contribution of Drs.: Kristen Brocavich, Sharlene Demehri, Nathan Estrin, Paul Fletcher, Jeffrey Ganeles, Maria Hernandez, Dimitios Papadimitriou, Eduard Rexha, John Rose, and Stavros Sofos.

Figure 12.1 Inadequate plaque control around one-piece implants (Ledermann screw) leading to an excessive inflammatory reaction of the peri-implant soft tissues and crestal bone loss.

Figure 12.2 Detoxification of the peri-implant bony defect with tetracycline.

have been extensively reported in the literature associated with the treatment of peri-implantitis. However, the peri-implant bony defect configuration seems to impact the treatment outcome, and previous experiences reported that the defect morphology dictates the treatment approach [14]. Specifically, contained circumferential defects (Figure 12.3a, b) seem to have favorable outcomes following surgical regenerative therapy using natural mineral in combination with a collagen membrane [31].

Since contained defects have a favorable regenerative potential, any type of bone and bone substitutes can be recommended. Non-contained defects (one- or two-wall) need the presence of good bone vascularization and stability of the bone particles. Growth factors (Figure 12.4) and/or stabilization of the blood clot, can be performed with the use of $CO₂$ or high-power diode lasers (Figure 12.5).

Therefore, autogenous bone (containing autogenous growth factors) or bone substitutes in combination with recombinant growth factors (platelet-derived growth factor/ PDGF, GEM21S) provide an excellent treatment approach.

The strategy for the surgical treatment of peri-implantitis is presented in the chart below.

Figure 12.3 Contained defects provide the best potential for regeneration.

Figure 12.4 (a, b) The non-contained defects (clinically) and radiographically do not provide the best prognosis and require additional biological factors for blood clot- as well as bone particle stabilization.

Figure 12.5 Decontamination of the implant surface using a $CO₂$ (a) or a diode laser (b) demonstrating adequate blood clot stabilization.

(a)

(b)

Also, the removal of the prosthesis for better access to the defects, to achieve better decontamination and defect debridement has been documented. Preclinical studies in dogs [32] showed that open surgical treatment and submerged healing improved the outcome. Also, clinical studies showed that treatment of peri-implant defects using a bone

graft substitute combined with a resorbable membrane and submerged healing results in defect-fill and clinically healthier situations [33–35].

However, a clinical study with 32 clinical cases showed no statistically significant differences in response to treatment when removing or keeping the prosthesis after regenerative surgery in peri-implant defects [36]. In a recent retrospective study, Parma-Benfenati et al. [37] showed successful longterm clinical outcomes after regenerative treatment of advanced peri-implantitis defects (vertical defects). They concluded that a success rate of 70.2% in 40 implants in an average follow-up period was 6.9 years (range: 2–21 years).

Consecutive case series of 170 treated peri-implantitis– affected implants in 100 patients with follow-up measurements from 2 to 10 years were published by Froum et al. [15]. A total of 51 implants in 38 patients previously

reported were followed for an additional 2.5 years, and 119 different implants in 62 other patients were treated with the same protocol and monitored for at least 2 years posttreatment. The results concluded that of the 170 implants treated, 2 were lost, both at six months post-surgery, yielding a 98.8% survival rate with a mean probing depth reduction (168 implants) of 5.10mm (range of 2–12mm) and average bone gain of 1.77mm (range of 0.4–9.0mm).

A variety of grafting materials have been tested clinically to evaluate the clinical outcome, with the main goal to get a bone fill and re-osseointegration (Figures 12.6 and 12.7).

The bone reconstruction was obtained with a variety of bone substitutes like autogenous bone [38–41], deproteinized bovine xenograft (cancellous BioOss®) ([25]), vancomycin and tobramycin impregnated allografts [42] and collagen-contained grafting materials [13, 43], porous titanium granules [44], and enamel-matrix derivatives [45].

Comparison between different xenografts (Endobon® and BioOss®; both having bovine origin) did not show any superiority when used in reconstructive surgery of periimplant osseous defects [46].

Recent studies from private practices using combination therapy of flap reflection, surface decontamination, use of enamel-matrix derivative (EMD) or platelet-derived growth factor (PDGF, GEM21S®), and guided bone regeneration (GBR) with mineralized freeze-dried bone and/or inorganic bovine bone combined with PDGF or EMD and covered with an absorbable membrane and/or subepithelial connective tissue graft appear to be encouraging [15]. The present evidence does not show any superiority to the specific debridement method (decontamination) and the use of a specific bone substitute. There is no standardized recommendation for submerged or non-submerged healing after grafting or the adjunctive administration of antibiotics [1].

The effectiveness of the outcome in the treatment of peri-implantitis after combination therapy with implantoplasty and augmentation was similar, independent if the implants were initially placed in pristine or grafted bone [47].

However, the main topic in any case of peri-implantitis therapy is the aspect of re-osseointegration. Studies have also reported that though new bone generation occurs to some extent, a "true" re-stabilization could not be regained without adjunctive GBR [48–50].

Following preclinical [48, 51, 52] and clinical [53] evidence of the possibility of obtaining re-osseointegration around dental implants affected by peri-implantitis, several case reports and studies proposed different reconstructive protocols using autogenous bone and/or various bone substitutes utilizing also barrier membranes to treat peri-implantitis defects [24, 25, 33, 34, 38–40].

Preclinical studies found that while substantial "re-osseointegration" occurred to an implant with a rough surface (SLA), the bone growth on a previously exposed smooth surface (turned) was minimal [48]. Nevins et al. [54] showed re-osseointegration after decontamination of the infected implant surface using the Er,Cr:YSGG laser [54].

Fletcher et al. [53] presented human histologic evidence of re-osseointegration after the use of calcium sulfate and bovine bone as grafting materials and a porcine collagen barrier for connective tissue and epithelial exclusion in conjunction with guided bone regeneration. The implant surface was debrided with a plastic curette, and diluted sodium hypochlorite, hydrogen peroxide, and sterile saline were used for implant surface detoxification.

In a recent systematic review on the topic of reosseointegration, Lollobrigida et al. [55] showed in animal studies with induced peri-implantitis that rough surfaces can enhance re-osseointegration as compared to smooth surfaces. Submerged healing and barrier membranes have shown a positive effect on re-osseointegration. Growth factors have been shown to improve re-osseointegration in animal models, though additional studies are required to confirm the data. Several decontamination treatments have been shown to promote re-osseointegration compared to control, and no specific procedure has proven superior to others in achieving re-osseointegration.

The following goals should be achieved in the surgical treatment of peri-implantitis:

- Removal of the surface pathogens (i.e. biofilm) and degranulation in the peri-implant defect
- Decontamination of the implant surface and surrounding bone
- Creation of an attached keratinized mucosa around the defect to create an adequate width of mechanically stable mucosa
- Decrease the probing pocket depth and bleeding on probing
- Regeneration of the bone
- Access for implant maintenance; if needed, a modification of the implant–supported restoration is required to achieve a sustainable clinical outcome.

Clinical Cases

Case 1 Peri-implantitis Therapy and Implant Surface Decontamination with Hydroxen Peroxide

Figure 12.6 Circumferential intrabony defect due to peri-implantitis. The defect (a) was debrided and rinsed with 3% H₂O₂ solution (b). The bone augmentation was performed with particulate bovine mineral in particulate form (c, d. Endobon granules S° , Biomet, Merck, France) and covered with a collagen (e) membrane (OsseoGuard®, 3i, Palm Beach Gardens, FL), immobilized with titanium tacks (f, g). The healing was uneventful, and the defect showed no bone resorption compared to the preoperative condition after two years (h,i).

Figure 12.6 (Continued)

Case 2 Peri-implantitis Therapy in a Heavy Smoker

Implants were placed in a heavy smoker (heavy cigar smoker for more than 10 years) with type 2 diabetes (HbA1c: 7.2%). One year after implant placement the patient developed peri-implantitis. The defects were debrided with titanium curettes (a). The decontamination occurred with 3% H₂O₂ irrigation for one minute. A non-resorbable PTFE membrane was used without the use of any grafting material. The membrane was adapted around the implants (b), stabilized with titanium tacks (Frios[®], Dentsply, Mannheim, Germany), and the flap was sutured without tension (c, d). The membrane was exposed after six weeks (e). Chlorhexidine mouth rinses were recommended and the membrane including the tacks was removed carefully after eight weeks (f). The bone regeneration after two years with the prosthetic restoration showed a significant improvement and new bone fill (g).
(a) (b)

Figure 12.7 (a) Circumferential peri-implant defects in a heavy smoker; flap elevation with a buccal incision design. Immobilization of the membrane around the implant platform with titanium tacks and placement of healing abutments (b); flap closure with interrupted sutures (c). (d) Postoperative radiograph representing the size of the defect. (e) Slight membrane exposure without signs of suppuration. (f) Removal of the membrane and demonstration of well-vascularized granulation tissue around the implants. (g) Radiographic evidence of the bone fill two years after peri-implantitis therapy.

(g)

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The following clinical cases present laser-assisted periimplantitis therapy based on the cases published in the recent book: Romanos GE. *Advanced Laser Surgery in Dentistry* (Wiley, 2021).

Case 3 Laser-assisted Implant Surface Decontamination and Peri-implantitis Therapy

The treatment of the peri-implant lesions in the area #19-21 was performed after removal of the restoration (Figure 12.8a), periosteal flap elevation under local anesthesia, degranulation with curettes (Figure 12.8b) and decontamination with a pulsed mode $CO₂$ laser (4W, noncontact). The blood clot was stabilized in the defects (Figure 12.8c) and was preserved during the bone augmentation with bovine mineral bone grafting material (Figure 12.8d). A collagen membrane covered the defects and immobilized them with titanium tacks (Figure 12.8e). The flap was closed with conventional interrupted sutures. The wound healing was uneventful and new bone formation around the implants was shown after two years (Figure 12.8f). No other clinical findings were presented.

Case 4 Peri-implantitis Treatment Before Functional Loading

The case presents a peri-implantitis clinical case before delivery of the final prosthesis. The patient complained for acute pain and swelling in the mesial implant (#21 area). After irrigation with chlorhexidine solution to clean up the acute infection, a mucoperiosteal flap was elevated and the peri-implant bony defect was de-granulated with curettes. The maximum depth of the infrabony defect was 12mm. The decontamination with a $CO₂$ laser (4W; pulsed mode)

 (a) (b)

Figure 12.8 Peri-implant lesions in the area #19–21 after removal of the restoration (a); defect after degranulation with curettes (b); decontamination with a pulsed mode $CO₂$ laser (4 Watts, non-contact) providing blood clot stabilization in the defects (c); bone augmentation with bovine mineral (d); collagen membrane coverage of the defect immobilized with titanium tacks (e); new bone fill, around implants after two years (f).

was performed and the entire defect was irradiated in an ablative mode. Bone grafting with a bovine mineral was finally used to fill the defect and a collagen membrane

covered the augmented site. The healing was uneventful and the clinical and radiological condition improved over the 3-years of follow-up (Figure 12.9).

(c)

(e)

(g)

Figure 12.9 Peri-implantitis diagnosed immediately before delivery of the final prosthesis associated with pain and suppuration (a, b); scaling of the defect with plastic curette (c) and measurement intraoperatively with a periodontal probe showing a depth of 12 mm (d); decontamination with a CO₂ laser (e); bone grafting with a bovine mineral (f); collagen membrane covered the augmented site (g); flap closure (h); uneventful healing after three months (i); radiological condition presenting bone fill after three years (j).

Figure 12.9 (Continued)

Case 5 Peri-implantitis Treatment with Autogenous Bone Grafting Material

The presented case shows an advanced peri-implant bony defect with symptoms of peri-implantitis and no implant mobility. The patient was interested to maintain this implant and asked for surgical treatment. A flap elevation and a meticulous degranulation of the defect were initially performed. The decontamination of the defect was performed with a $CO₂$ laser (4W, pulsed mode, non-contact) and final bone augmentation with autogenous bone harvested from the chin region (particulate with a bone mill). Coverage of the augmentation site with a collagen membrane stabilized the particulate bone and condensed within the defect. The flap was closed with conventional sutures. The bone regeneration occurred in the next few months and showed an excellent crestal bone after four months when the implants were restored prosthetically (Figure 12.10).

Case 6 Laser-Supported Treatment of Severe Peri-implantitis Defect

The present case demonstrates a severe bone loss of an implant, splinted with additional implants in the fixed maxillary restoration. The female 84-year-old patient asked me to try to do my best to save her implant, which

was the terminal (distal) implant of the restoration and was an additional anchorage for a partial prosthesis. The patient signed the informed consent to harvest autogenous bone from the tuberosity (#1) and to use this particulate graft with allograft as a composite graft (b, c) after debridement and detoxification of the implant surface using the pulsed $CO₂$ laser in a non-contact mode. The detoxification occurred for 30 seconds around the entire exposed implant surface (Figure 12.11d). The defect was filled with the composite graft and a collagen membrane was used to cover the augmented site (Figure 12.11e, f). The flap was advanced coronally and closed with silk sutures (Figure 12.11g, h). The patient presented a significant reduction of the probing pocket depth and a recession of the soft tissue (Figure 12.11i). She asked for a new surgical procedure to improve the clinical outcome. The probing was reduced without bleeding on probing or suppuration (Figure 12.11j). A new flap was elevated and presented significant bone fill. The defect was filled with a combination of BioOss collagen® and BioOss® 1–2 mm large granules were used to augment the site. A collagen membrane was used to cover the grafted site (Figure 12.11k–m). The two-year radiological evaluation showed a complete bone fill without resorption (Figure 12.11n) compared to the preoperative radiograph (Figure 12.11o) *(Surgery performed together with the periodontal resident: D. Papadimitriou, Rochester, NY)*

Figure 12.10 Advanced peri-implant bony defect with symptoms of peri-implantitis and no implant mobility (a, b); after flap elevation and meticulous degranulation of the defect (c); decontamination of the defect with a CO $_2$ laser (d); bone augmentation with autogenous particulate bone (e); coverage of the augmentation site with a collagen membrane (f); flap closure (g); excellent bone crest after four months (h).

Figure 12.11

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Figure 12.11 (Continued)

Case 7 Laser-Supported Treatment of Peri-implantitis

A 55-year-old patient was diagnosed with peri-implant inflammation (Figure 12.12a) associated with pain and suppuration and presented radiological findings of an advanced peri-implant osseous lesion (Figure 12.12b). The

implant prosthesis was a screw-retained restoration, which was fabricated 10 years ago. The prosthesis and the abutment of the failing implant were removed. Under local anesthesia, a mucoperiosteal flap was elevated, the periimplant granulation tissue was conventionally removed with plastic curettes (Figure 12.12c) and subsequently, the implant surface and the surrounding bone were decontaminated with the $CO₂$ laser (average power: 4W, c.w.). The laser-assisted decontamination permits good hemostasis (Figure 12.12d). The osseous defect was filled with bovine mineral bone grafting material (Figure 12.12e) and covered with an absorbable collagen membrane, based on the principles of the Guided Bone Regeneration for submerged healing (Figure 12.12f). After four months the implant was uncovered, the previous abutment was placed in the same position and the older prosthesis was delivered. Healing

succeeded with no complications and after four months the clinical situation (as well as the radiological examination showed excellent peri-implant conditions. The clinical situation was stable for four years (Figure 12.12g, h). However, 13 years later the patient decided to replace the bridge for esthetic reasons and mesial migration of the anterior teeth. The peri-implant condition showed after 21 years a stability of the peri-implant bone, proof of the longterm success of the peri-implantitis treatment concept (Figure 12.12i–k).

Figure 12.12 Peri-implant bone loss associated with pain and suppuration (a); radiological findings of an advanced peri-implant osseous lesion (b); Demonstration of the defect after degranulation (c); decontamination with the CO₂-laser (d); bovine mineral bone grafting material (e) and coverage with a collagen membrane (f); clinical and radiographic evaluation after 4 years (g, h); The peri-implant condition showed after 21 years stability of the peri-implant bone without periodontal pocket (i, j, k).

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(k)

Figure 12.12 (Continued)

(j)

Case 8 Treatment in a Heavy Smoker using CO² Laser Decontamination and Bone Mineral as a Grafting Material (see Figure 12.13)

Case 9 Peri-implantitis Treatment with a Diode Laser

The clinical case presents a female patient with endosseous dental implants placed almost 4 years ago and loaded with a fixed prosthesis (Figure 12.14a). The patient complained about sub-acute pain, especially during tooth brushing. The clinical examination showed bleeding on probing and suppuration in conjunction with peri-implant intrabony defects at #19–20 (Figure 12.14b). After local anesthesia and mucoperiosteal flap elevation with meticulous degranulation of the peri-implant defects (Figure 12.14c), a 980nm diode laser with a 300μm glass fiber was used, in a non-contact mode and 2W power settings for 3x20 seconds irradiation period. (Figure 12.14d).

Autogenous, particulate bone grafting material was used (patient decision) to fill the defects (Figure 12.14e), and a collagen membrane was applied and fixated with titanium tacks (Figure 12.14f). The flap was closed with interrupted silk sutures. The sutures were removed after one week. The healing was uneventful and after one year the clinical and radiological evaluation (Figure 12.14g, h) showed a significant increase of the bone fill around the failing implants and no clinical symptoms of inflammation.

 $\qquad \qquad \text{(c)}$

Figure 12.14 (Continued)

Figure 12.14 Peri-implant infrabony defects at #19–20 (a, b); meticulous degranulation of the peri-implant defects (c); a 980 nm diode laser was used to irradiate the defects (d); autogenous, particulate bone grafting material was used to fill the defects (e); a collagen membrane was fixated around the augmentation area (f); clinical and radiological evaluation (g); bone fill around the failing implants one year after grafting (h).

Case 10 Treatment of Peri-implantitis in a Diabetes Patient Using a Pulsed 980 nm Diode Laser

A circumferential defect with vertical component (a) due to peri-implantitis was debrided and irradiated with a 980 nm pulsed diode laser (b) for 30 seconds (noncontact, non-initiated tip, 320-micron glass fiber, 2W). The blood clot was stabilized (c) before bone grafting allograft material (Puros®, ZimVie, Palm Beach Gardens, FL) filled the defect (d). A collagen membrane (e) covered the augmented site. The implant was healed in submerged mode. Therefore, the cover screw was placed, and the flap was advanced for primary closure. Six months after treatment, the implant was loaded with the previous prosthesis. The pre-operative, postoperative condition, six-month follow-up, and the five years follow-up presenting a complete bone fill are demonstrated in Figure 12.15f–i. The peri-implant clinical measurements showed a general healthy condition (j) after five years.

(Surgery was performed together with the resident: Dr. K. Brocavich, Stony Brook University, NY).

Case 11 Treatment of Peri-implantitis Using a Pulsed 980nm Diode Laser

Peri-implantitis defect before implant loading was debrided by titanium curettes and the implant was decontaminated with a pulsed 980 nm diode laser for 20–30 seconds (320 micron glass non-initiated fiber) and 2-w average power in a non-contact mode (Figure 12.16a).

A composite graft (allograft – Puros® and autogenous bone from the ramus) was used to fill the vertical two-wall defect (Fig. 12.16b). The bone particles were compressed under a collagen membrane (Fig. 12.16c, d), which was stabilized with titanium tacks (Fig. 12.16c, d). The flap was advanced and closed with vertical mattress sutures. The implant was healed in submerged mode (Fig. 12.16e). Nine months later, the implant was uncovered (Fig. 12.16 f). The complete bone fill is presented in Figures 12.16f, g when the flap was elevated for the removal of the titanium tacks. The final restoration was delivered 7 months later. The follow-up radiograph 14 months later shows the crestal bone stability (Fig. 12.16h). *(The patient was treated with the residents E. Rexha and N. Estrin, Stony Brook University, NY)*

(d) (e) (f)

Figure 12.15

闸

Figure 12.16

Case 12 Peri-implantitis Therapy in Conjunction with Growth Factors

Peri-implant subacute swelling in #14 under antibiotic treatment before drainage (Fig. 12.17a–d). Peri-implant defect due to excess of cement after flap elevation demonstrating the severe bone loss. A long fracture was diagnosed at the tooth #13 and required a tooth extraction (Fig. 12.17e, f). After debridement of the defect and decontamination with H2O2 3% for one minute the defect was filled with a composite graft of allograft and platelet-derived growth factor, covered with a collagen membrane (Fig. 12.17g). The membrane was stabilized with titanium tacks and the flap was closed (Fig. 12.17h, i). The clinical outcome was improved over this healing period (Fig. 12.17j–l) After a full-thickness flap elevation for implant placement at the site #13, a significant defect closure was demonstrated at the area #14 and complete bone regeneration palatally. However, new grafting was required for complete bone regeneration. Eight months later implant uncovering (#13) and removal of the tacks shows a complete bone fill around the implants #13 and #14 (Fig. 12.17m). The implant was restored two months later representing good clinical and esthetic outcome and radiographically crestal bone stability (Fig. 12.17n-p). Postoperatively, the patient was rinsing the oral cavity 3–5×/day with the herbal-based mouth rinse StellaLife, which supports the wound healing and has also beneficial effects in the tissue regeneration (Fig. 12.17q, r).

Figure 12.17 (a) Peri-implant subacute swelling in #14 under antibiotic treatment before drainage. (b) Peri-implant bleeding on probing with deep probing more than 15mm in the #14. (c, d) *Peri-implant defect due to excess of cement after flap elevation demonstrating the severe bone loss in conjunction with palatal vertical defect*. (e) A long fracture was diagnosed at the tooth #13 and required a tooth extraction (f). After debridement of the defect and decontamination with H_2O_2 3% for one minute (f) the defect was filled with a composite graft of allograft (Lynch Biologics) (g) and platelet-derived growth factor (PDGF, GEM 21S) and covered with a collagen membrane. The membrane was stabilized with titanium tacks (h) and the flap was closed without tension (i). The patient was recommended to use mouth rinse with StellaLife[®] for five months. The clinical outcome was improved over this healing period (j). The radiographic evaluation before, after grafting and five months later is shown in the periapical radiographs (k). (l) After a full-thickness flap elevation for implant placement at the site #13, a significant defect closure was demonstrated at the area #14 and complete bone regeneration palatally. However, new grafting was required for complete bone regeneration. Eight months later implant uncovering (#13) and removal of the tacks shows a complete bone fill around the implants #13 and #14 (m). The implant was restored two months later representing good clinical (n) and esthetic outcomes with radiographically crestal bone stability two years after treatment (o, p). Postoperatively, the patient was rinsing the oral cavity 3-5×/day with the herbal-based mouth rinse StellaLife® which supports the wound healing and has also beneficial effects in the tissue regeneration. (q, r) Mouthrinse for surgical site decontamination (herbal basis) promoting wound healing after surgery.

(j)

(k)

Before grafting

Figure 12.17 (Continued)

Post grafting extending the state of the S mo. after grafting

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Figure 12.17 (Continued)

Figure 12.17 (Continued)

The following clinical cases have been treated by different clinicians and included here to provide further information about various concepts of surgical treatment of peri-implantitis. These cases show multiple decontamination methods of the implant **surface as well as grafting materials including growth factors to contribute to the bone growth around implants and possibly re-osseointegration (implant long-term success).**

Case 1 (Treatment provided by Dr. Paul Fletcher, Stony Brook, NY)

The patient presented with 8mm pocketing, a buccal mucogingival problem, and radiographic symmetrically appearing vertical bone loss around tooth #19 (Figure 12.18a, b, c). After obtaining informed consent, the

patient was premedicated with 500mg of amoxicillin and anesthetized with 2% xylocaine w/1 : 100,000 epinephrine. Buccal and lingual sulcular incisions were placed from #'s 18–20, the soft tissue was reflected, and the bony

 (a) (b)

 (g) (c) (h)

Figure 12.18

defect visualized. Granulomatous tissue in the defect was debrided using a carbon fiber curette, and residual dental cement was seen apical to the crown margin (Figure 12.18d). Following removal of the cement, the implant surface was mechanically decontaminated with a carbon fiber curette (Figure 12.18e) and was chemically detoxified by firmly and thoroughly burnishing it for one minute with a cotton pellet soaked in sterile saline (Figure 12.18f). The osseous defect was filled with a cortico-cancellous bone combination (Puros, Zimmer Biomet) (Figure 12.18g), and a dermal allograft (Perioderm, Dentsply) was placed over the buccal and lingual aspects of the defect to act both as a barrier membrane [56] and to serve as a connective tissue graft to aid in the resolution of the mucogingival problem (Figure 12.18h). Dermis has been shown to remain occlusive to vascular and connective tissue ingrowth for at least 2 months while an autogenous connective tissue graft will show vascular ingrowth within 10 days. The soft tissue

flaps were re-approximated utilizing 4-0 Vicryl sutures (Figure 12.18i) and the patient was placed on amoxicillin 500mg tid×10 days and ibuprofen 400mg prn q4-6h. Seventeen-month follow-up images show the resolution of both the mucogingival defect and the infrabony bone loss (Figure 12.18j, k). While the result was satisfying to all involved, it is important to note the only outcome of the osseous grafting aspect of this procedure that can validly be claimed is the achievement of radiographic evidence of bone fill. In the absence of prior histologic proof of principle confirmation, re-osseointegration (defined as new direct bone-to-implant contact on a previously contaminated implant surface), cannot accurately be claimed, using this or any other decontamination and grafting technique.

(Surgical treatment performed in collaboration with Siyan Lin, DDS, former post-doctoral resident in Periodontics, Columbia University College of Dental Medicine, New York, NY).

Case 2 (Treatment provided by Dr. Paul Fletcher, Stony Brook, NY)

The patient presented with inflammation, 9mm pocketing, radiographic evidence of both horizontal and vertical bone loss, and a buccal mucogingival problem due to a shallow vestibule around an implant in the #20 position (Figure 12.19a, b). After receiving the patient's informed consent, she was premedicated with 500mg of amoxicillin and anesthetized with 2% xylocaine w/1 : 100,000 epinephrine. Following an unsuccessful attempt to remove the incompletely seated splinted implant restoration, buccal and lingual sulcular incisions were placed to elevate full-thickness flaps from #'s 19–21. The peri-implantitis defect was visualized and titanium curettes were used for soft tissue degranulation and to mechanically decontaminate the implant surface. To chemically achieve surface detoxification the implant threads and collar were meticulously burnished with sterile saline on a small cotton pellet, and copious irrigation

with sterile saline from a Monojet syringe was used in the osseous defect and on the implant surface to further dilute the microbial concentration to a sub-inflammatory level (Figure 12.19c). The osseous defect was filled with slowly resorbing calcium sulfate (Nanogen, Orthogen LLC) and was covered on the buccal and lingual with a porcine collagen membrane (Dynamatrix, Keystone Inc.) (Figure 12.19d). The flaps were sutured with 4-0 Vicryl sutures (Figure 12.19e). Amoxicillin 500mg and metronidazole 500mg were prescribed three times daily for 10 days. The patient was advised not to brush the surgical site for two weeks and to rinse with 0.12% chlorhexidine for 30 seconds twice daily.

(Surgical treatment performed in collaboration with Christos Constantinides, DDS, former post-doctoral resident in Periodontics, Columbia University College of Dental Medicine, New York, NY).

Figure 12.19 (f) is a seven-month postoperative clinical image of the surgical site showing healthy tissue and the height of the gingiva in relation to the crown margin of #20, and (g) shows a 3mm probing depth on the buccal of #20. (h) At the 12-month post-surgical visit the patient complained of soft tissue soreness on the buccal of #20. The gingiva had receded 3mm and the sulcular margin was slightly retractable due to the pull of the vestibule. It was decided re-enter the area and deepen the vestibule using an apically positioned split thickness flap and augment the keratinized gingiva using a free gingival graft, while simultaneously clinically evaluating the results of the prior surgery. After receiving the patient's informed consent and following the administration of local anesthetic, a buccal sulcular incision was placed with a vertical release mesial to #20. A split thickness flap was elevated, with the periosteum on the buccal of #20 being moved aside to allow for visualization of what appeared to be a calcified fill of the infrabony component of the original defect with a bone like material closely abutting the implant surface (i). As in the previous example, as favorable as the result was using this technique, true re-osseointegration cannot accurately be claimed without a histological section or prior proof of principle histology [57]. A free gingival graft from the palate was subsequently placed on the buccal of #'s 19 and 20 (j), the soft tissue flap was sutured apically to deepen the vestibule, and periodontal pack was used for protection during initial healing process (k). The final result shows resolution of the peri-implantitis defect, a deepening of the vestibule, and an adequate zone of buccal keratinized gingiva (l), (m) is a 12- month post- operative radiograph showing a radiopaque fill of the infrabony component of the lesion.

Figure 12.19 (Continued)

Case 3 (Treatment provided by Dr. Paul Fletcher, Stony Brook, NY)

The patient presented at the dental clinic of the Department of Periodontics of the State University of Rio de Janeiro Brazil with advanced bone loss around five implants supporting a mandibular screw-retained implant overdenture (Figure 12.20a–k). The proposed treatment plan was to remove the implants labeled #'s 2 and 3 in Figure 12.20a and augment the extraction sites for future implant placement while simultaneously attempting to regenerate bone around the implants labeled #'s 1, 4 and 5. After further evaluation of a cross-sectional scan showing the size and shape of the bony defect around implant #3 (Figure 12.20b), the patient agreed to allow us to keep this implant and graft and treat it similarly to the implants being retained and treated, prior to subsequently removing it for histological evaluation. A study protocol was developed at the State University of Rio de Janeiro and approved by the Ethics Committee of the University Hospital Pedro Ernesto (CE-HUPE) in full accordance with the World Medical Association Declaration of Helsinki. It was accepted by the patient and his oral and written informed consent was received prior to treatment.

Following the removal of implant #2, Figure 12.20c shows plaque and calculus on the surface of implant #3 being mechanically debrided with a plastic curette. Figure 12.20d shows the implant surface being chemically detoxified by thorough burnishing with cotton pellets soaked in sequential solutions of 0.25% sodium hypochlorite (NaClO), a 50 : 50 3% hydrogen peroxide, and sterile water solution (H_2O_2 -H2O). Additionally, the 6mm wide horizontal and 11 exposed thread vertical components of the osseous defect can be visualized. Figure 12.20e shows a 50 : 50 graft combination by volume of bovine bone (Bio-Oss, Geistlich) and calcium sulfate (NanoGen, OrthoGen) in the defect, with the platform of the implant removed so it could be submerged during the healing process. Figure 12.20f shows a porcine collagen membrane (Dynamatrix, Keystone Dental Group) with a punch hole the diameter of the implant placed over the implant to serve as a barrier membrane. The other remaining implants were treated in a similar manner. Figure 12.20g shows the surgical site sutured with the test implant submerged.

Upon surgical re-entry at six months only 8 threads of implant #3 were now exposed (Figure 12.20h). The implant was removed *en bloc* and the supracrestal threads were sectioned horizontally from those embedded in bone. A low-power SEM of the super osseous implant surface shows the characteristic microanatomy of the pores of an additive anodized implant surface. The surface is free of bacteria, but some residual inorganic mineralized material is evident adjacent to and within the pores of the surface (Figure 12.20i).

Following processing of the implant/bone core, a SEM shows pre-existing apical bone, remodeling pre-existing bone, new bone, and particles of bovine bone graft material. The pre-existing bone is denoted by the presence of interstitial bone (see arrow and blue star) which stains lighter as it's more mineralized and has no rings. The darker-appearing bone around the remnants of interstitial bone is actively remodeling old bone. Above the most coronal remnants of old bone, in direct contact with the implant surface, are three threads of well-integrated new bone, evidenced by its relative lack of organization, increased cellularity, vascularity, number of marrow spaces, and incorporated particles of bovine graft material (Figure 12.20j).

The histologic section (Figure 12.20k) expands on the findings of the subcrestal SEM photomicrograph. The section confirms the presence of dense new bone-toimplant contact against the top 3 threads coronal to the old bone with no evidence of epithelial or connective tissue down growth between the bone and the implant. There are no inflammatory cells visible against the implant or in the bone away from the implant surface. The particles of bovine bone that remain have moved away from the implant surface during the healing process to allow for direct apposition of new bone to the implant.

This proof of principle case demonstrates that reosseointegration, defined as direct bone-to-implant contact on a previously contaminated implant surface, is possible in a human using readily available, low-cost medicaments such as NaClO, H_2O_2 and NaCl for decontamination purposes [53].

(Surgical treatment performed in collaboration with Daniel Deluiz, DDS, PhD, former post-doctoral researcher, Faculty of Dentistry, State University of Rio de Janeiro, Rio de Janeiro, Brazil).

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250 *Surgical Treatment of Peri-implantitis*

Peri-implantitis case presentation utilizing air abrasion using glycine powder, hydrogen peroxide 3% chemical

detoxification and mineralized cancellous allograft in conjunction with rhPDGF (Fig. 12.21).

Figure 12.21 (a) Preoperative radiograph: note the severe bone loss at implants #18 and 20. (b) Intraoperative buccal view: prosthesis was removed, and gingival flap elevated showing severe bone loss at both fixtures. (c) Intraoperative lingual view showing the moat like characteristic of the defects. Implant surfaces would be debrided with air abrasion using glycine powder and hydrogen peroxide 3% chemical detoxification. (d) Intraoperative buccal view: mineralized cancellous allograft, soaked in rhPDGF. (e) Intraoperative view: a crosslinked collagen membrane was cut and fitted over the allograft. (f) Immediate post-operative radiograph showing the allograft filled to the coronal level of the defects. (g) Postoperative radiograph taken at two months. (h) Postoperative radiograph taken at eight months showing evidence of radiographic bone fill, prosthesis was replaced. (i) Postoperative photograph at 8 months prior to replacement of the prosthesis. Note: healthy appearance of the soft tissue surrounding the fixtures.

photograph showing the placement of mineral cancellous allograft which was soaked in rhPDGF. (e) Intraoperative photograph of a crosslinked collagen membrane cut and fitted over the bone graft. (f) Intraoperative photograph showing complete closure with a 4-0 Vicryl suture. (g) Immediate postoperative radiograph showing allograft placement. (h) Postoperative radiograph taken at 18 months showing radiographic evidence of bone fill.

Figure 12.24 (a) Pre-operative photograph of a two-unit splinted implant prosthesis and a single-unit prosthesis. (b) Preoperative radiograph. (c) Intraoperative photograph: implant #28 with a severe moat like defect and dehiscence of the buccal plate. Local factors present on the surface of the implant fixture. (d) Intraoperative photograph: implant #28 treated with Titanium curette, glycine-air abrasion, hydrogen peroxide for two minutes and the sterile saline rinse for 30 seconds. (e) Intraoperative view of bone grafting. (f) Intraoperative photograph: bone graft covered with a collagen membrane, secured around the fixtures and healing abutments. (g) Intraoperative radiograph: Flaps adapted with PTFE sutures. (h) Immediate postoperative radiograph. (i) Postoperative radiograph at eight months. Prosthesis was sectioned a single unit was replaced at #28 for esthetics. The bone fill is well demonstrated (i) compared to the preoperative condition (j).

severe bone loss at implant #9. (c) Intraoperative photograph showing severe buccal dehiscence at #9. There is a moat like palatal defect not visible in this picture. (d) Intraoperative photograph showing application of hydrogen peroxide at the implant surface. Air abrasion with glycine proceeded this with hydrogen peroxide. (e) Intraoperative photograph showing the positioning of a collagen membrane on the palatal aspect of the implant. (f) Intraoperative photograph showing the application of mineralized cancellous graft. (g) Intraoperative photograph of a collagen membrane covering the buccal and interproximal aspects of the bone graft. (h) Postoperative radiograph of site sutured with 4-0 vicryl. (i) Immediate postoperative radiograph of implant #9.

Peri-implantitis defect at the buccal aspect of the implant using air-abrasion with glycine powder and 3% hydrogen

peroxide to decontaminate the exposed implant surface (Fig. 12–26).

showing bone loss at implant #8. (c) Intraoperative buccal view showing severe bone loss on the buccal aspect of the implant. (d) Intraoperative buccal view showing the use of air-abrasion with glycine powder to decontaminate the exposed implant surface. (e) Intraoperative buccal view showing the use of 3% hydrogen peroxide to decontaminate the exposed implant surface. (f) Intraoperative buccal view showing the use of citric acid to decontaminate the exposed implant surface. (g) Intraoperative buccal view showing the use of doxycycline slurry to decontaminate the exposed implant surface. (h) Intraoperative buccal view of the addition of mineralized cancellous allograft previously soaked in rhPDGF. (i) Intraoperative buccal view of the addition of a crosslinked collagen membrane sutured in place. (j) Intraoperative buccal view of the addition of enamel matrix derivative added to a secured crosslinked collagen matrix and the surround hard and soft tissues. (k) Immediate postoperative buccal view of flap replaced with 6-0 polypropylene sutures. (l) Immediate postoperative radiograph.

Case 10 (Treatment provided by Drs. Stavros Sofos and Maria Hernandez, Nova Southeastern University, Fort Lauderdale, FL)

A patient with a history of hypothyroidism and hypercholesterolemia (properly medicated for both) was referred due to the presence of BoP and SoP on implants in positions 30 and 31 (Straumann BL implants placed in 2009). Crowns were removed by the restorative provider (Figure 12.27a–c).

Intraoral radiographs indicated a 1C circumferential bone defect with the presence of excess cement distal to both implants (Figure 12.27d–e).

Treatment:

- 1) **Non-surgical therapy**: The site was debrided using titanium curettes and copious irrigation with chlorhexidine 0.12%. The patient then rinsed with chlorhexidine 0.12% twice daily for 14 days.
- 2) **Surgical therapy**: Three weeks later, the combined approach for the treatment of peri-implantitis was conducted. Implant abutments were removed and substituted with cover screws. A midcrestal incision was made extending from the mesial of tooth #27 to the distal of the last implant onto the ascending ramus. A full-thickness flap was then reflected, and all granulomatous tissue was removed. The clinical configuration of the defects closely matched the radiographic findings (Figure 12.27f–g). The suprabony portions of the implants were treated with implantoplasty (Figure 12.27h), while the intrabony portions were treated with EDTA for three minutes

(Figure 12.27i), followed by copious irrigation with saline, followed by chlorhexidine 0.12%, and finally with saline again. A slow resorbing bone graft (DBBM) was placed in both defects (Figure 12.27j) and covered with a non-crosslinked native CM. Buccal and lingual periosteal releases were made and the surgical site was closed by primary intention using PTFE 3/0 sutures (Figure 12.27k). The patient was properly medicated.

3) The patient returned for a seven-day post-op. presenting uneventful healing. Sutures were removed after three weeks (Figure 12.27l).

After six months, the patient returned for re-entry. Periapical radiographs showed radiographic bone fill of the defect with 1 mm of the implant remaining above the alveolar crest, corresponding to portions of the implants that received implantoplasty (Figure 12.27m–n).

Implants were uncovered, healing abutments were placed, and a free gingival graft (FGG) was placed buccally to correct the lack of attached gingiva. Four months later the patient was cleared for restorations (Figure 12.27o), and the patient received final abutments and crowns (Figure 12.27p–q). To date (two years later), the area shows no inflammation nor increased PPDs, and radiographic bone levels appear to be stable (Figure 12.27r).

(c) (d)

Figure 12.27 (a) Initial presentation (occlusal view). Crowns were previously removed by the restorative provider. (b) Buccal view. Note the suppuration buccal of the implant in position #31. (c) Lingual view. (d, e) Intraoral radiographs show radiopaque image distal of both implants, compatible with restorative material. (f) Surgery (three weeks after non-surgical treatment). Restorative abutments replaced by cover screws. Flap elevation. Granulomatous tissue around both implants. (g) Peri implant defect and implant surface after removal of granulomatous tissue. (h) Implantoplasty was perfomed buccally on implant surface with suprabony exposure (suprabony defect). (i) Implant surface treated/detoxified with cotton pellets soaked in EDTA 24% for 2 minutes. (j) DBBM bone particulate bone graft added to the defects. A native collage membrane was placed on top of the bone graft (no picture). (k) Flap was released by means of a buccal periosteal releasing collagen, then repositioned tension-free and sutured using PTFE 3/0 sutures. (l) Three-week follow-up. (m, n) Radiographs were taken at day of uncovering (six month after the surgical procedure). (o) Four months after uncovering and FGG. The patient was cleared for restoration. (p, q) Final restoration. (r) Two-year radiographic follow-up. (Treatment provided by Dr. Stavros Sofos, Fort Lauderdale, FL)

Case 11 (Treatment provided by Drs. Stavros Sofos and Sharlene Demehri, Nova Southeastern University, Fort Lauderdale, FL)

Patient with a history of hypertension (medicated and controlled), presented for evaluation of implant bridge from #3 to #6 position (cement-retained). Implants were placed in 2008 on a healed ridge). Upon clinical evaluation, increased probing depths (4–9mm), bleeding on probing (BoP) and suppuration (SoP) on the implant in position #6 were noted (Figure 12.28a–c), having a cement-retained restoration. Radiographic examination revealed approximately 50% bone loss mesially, and 30% distally, likely with the absence of buccal and lingual plates (Figure 12.28d). Implant #6 was diagnosed with peri-implantitis, with possible excess cement as a contributing factor.

Treatment:

- 1) The restoration was removed (being able to adequately preserve it), and a cover screw was placed in #6 and a healing abutment in #3 position, to allow for soft tissue healing over implant #6.
- 2) On the day of surgery, soft tissue had started to cover implant site #6 (Figure 12.28e). A full-thickness flap was raised (Figure 12.28f), and granulomatous tissue was removed (Figure 12.28g). Implant surface

detoxification mechanically with titanium curettes and titanium brushes with saline. Chemically surface was treated with 24% EDTA (Figures 12.28h–i)

- 3) GBR was completed with rhPDGF + FDBA, adding DBBM over FDBA, and a crosslinked collagen membrane (Figure 12.28j).
- 4) The periosteal release was performed achieving flap passivity, and the membrane was stabilized with periosteal sutures (Figure 12.28k).
- 5) Primary closure was achieved, suturing with PTFE 4-0 (Figure 12.28l–m). Healing occurred uneventfully and sutures were removed 4 weeks (Figure 12.28n).
- 6) The uncovering of the #6 implant was done at six months (Figure 12.28o–s). PA radiograph shows bone fill mesial and distal, CBCT shows buccal bone fill (Figures 12.28t–u). The healing abutment was left in place for two months in order to assess for periimplant tissue stability.
- 7) At eight months, peri-implant tissues showed stability, without the presence of inflammation (Figures 12.28v–x).
- 8) Replacement of original bridge at eight months postsurgery. Recession present, reduction in PPD, and no suppuration (Figure 12.28y).

Figure 12.28 (a) Initial presentation, frontal view. (b) Initial presentation, buccal view. (c) Initial presentation, palatal view. (d) Initial presentation PA radiograph. Note approximately 50% of bone loss mesially, 30% of bone loss distally. (e) Four weeks after bridge removal, replacement of restorative abutment with cover screw in #6. (f) Open flap. Note granulomatous tissue. (g) Granulomatous tissue removal. (h) Debrided implant surface (after being treated with EDTA). Evidence of large bone defect 9. around the implant, lack of buccal bone, and partial vertical component on the lingual wall. (i) Same as 8, occlusal view. (j) Particulate bone graft in place (allograft embedded in rh-PDGF, veneered with DBBM). (k) Site was over-augmented and covered with a crosslinked collagen membrane, held with periosteal sutures. (l, m) Flap closure using PTFE sutures. (n) Suture removal after four weeks. (o, p) Six months post-op. healed ridge, uncover visit. (q–s) Implant uncovering. (t) Periapical radiograph, six-month post-op. (u) CBCT, six-month post-op. (v–x) Two months following uncover. Note the stability of peri-implant soft tissues. (y) Definitive restoration in place.

(m)

(n)

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Case 12 (Treatment provided by Drs. Sharlene Dehmeri and Jeffrey Ganeles, Nova Southeastern University, Fort Lauderdale, FL)

Healthy patient presents to clinic for assessment of implant in #19 position. The implant was placed in 2015, non-platform-switched, and loaded with a screwretained crown (Figure 12.29a).

Clinical examination revealed increased PPDs, BoP and SoP (Figure 12.29b–d). Diagnosed as peri-implantitis.

Treatment:

- 1) The crown was removed.
- 2) A full-thickness flap opened buccal and lingually. Granulomatous was tissue removed (Figure 12.29e). Implant surface detoxification was done mechanically with titanium curettes and titanium brushes and with Saline. Implantoplasty was completed on micro threads (Figure 12.29f). Placed cover screw.
- 3) Flap advanced via a periosteal releasing incision. GBR was completed with rhPDGF + DBBM + FDBA and noncrosslinked collagen membrane (Figure 12.29g).
- 4) Membrane stabilized with periosteal sutures Chromic Gut 4-0.
- 5) Sutured using PTFE 4/0. Primary closure was achieved; an immediate radiograph was taken (Figure 12.29h– i). Sutures were removed at four weeks post-op.
- 6) Uncover the implant at six months (crown altered to close mesial open contact). The periapical radiograph shows bone fill mesial and distal (Figure 12.29j).
- 7) A 2-year follow-up: Recession present, reduction in PPD, and no suppuration. The radiograph shows stable crestal bone levels (Figure 12.29k–n).

(d) (e) (f)

 (9) (h) and the contract of the contract of

Figure 12.29 (a) Initial radiograph. (b, c) Initial presentation. Note suppuration on probing. (d) Initial presentation lingual. (e) Open flap. (f) Implantoplasty performed in most coronal aspects (micro threads). (g) Bone regeneration. (h) Flap repositioned and sutured. (i) Radiograph immediately after surgery. (j) Radiograph at the time of uncover and crown reconnection. (k–m) Two years follow up. (n) Two-year follow-up radiograph.

References

- **1** Schwarz, F., Jepsen, S., Obreja, K. et al. (2022). Surgical therapy of peri-implantitis. *Periodontology 2000* 88: 145–181.
- **2** Berglundh, T., Armitage, G., Araujo, M.G. et al. (2018). Periimplant 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* 89 (Suppl 1): S313–s318.
- **3** Rakasevic, D., Lazic, Z., Soldatovic, I. et al. (2022). Influence of titanium implant macrodesign on periimplantitis occurrence: a cross-sectional study. *Clinical Oral Investigations* [https://doi.org/10.1007/](https://doi.org/10.1007/s00784-022-04492-zas) [s00784-022-04492-zas.](https://doi.org/10.1007/s00784-022-04492-zas) Online ahead of print.
- **4** Javed, F. and Romanos, G.E. (2018). *Evidence-based Implant Dentistry and Systemic Conditions*. Hoboken: Wiley Publ.
- **5** Khoury, F., Keeve, P.L., Ramanauskaite, A. et al. (2019). Surgical treatment of peri-implantitis – consensus report of working group 4. *International Dental Journal* 69 (Suppl 2): 18–22.
- **6** Esposito, M., Grusovin, M.G., and Worthington, H.V. (2012). Treatment of peri-implantitis: what interventions are effective? A Cochrane systematic review. *European Journal of Oral Implantology* 5 (Suppl): S21–S41.
- **7** Renvert, S., Polyzois, I., and Claffey, N. (2012). Surgical therapy for the control of peri-implantitis. *Clinical Oral Implants Research* 23 (Suppl 6): 84–94.
- **8** Romanos, G.E. and Weitz, D. (2012). Therapy of periimplant diseases. Where is the evidence? *The Journal of Evidence-Based Dental Practice* 12 (3 Suppl): 204–208.
- **9** Ramanauskaite, A., Obreja, K., Sader, R. et al. (2019). Surgical treatment of periimplantitis with augmentative techniques. *Implant Dentistry* 28 (2): 187–209.
- **10** Yi, Y., Koo, K.T., Schwarz, F. et al. (2020). Association of prosthetic features and peri-implantitis: a cross-sectional study. *Journal of Clinical Periodontology 47*: 392–403.
- **11** Schou, S., Berglundh, T., and Lang, N.P. (2004). Surgical treatment of peri-implantitis. *The International Journal of Oral & Maxillofacial Implants* 19 (Suppl): 140–149.
- **12** La Monaca, G., Pranno, N., Annibali, S. et al. (2018). Clinical and radiographic outcomes of a surgical reconstructive approach in the treatment of periimplantitis lesions: A 5-year prospective case series. *Clinical Oral Implants Research* 29 (10): 1025–1037.
- **13** Mercado, F., Hamlet, S., and Ivanovski, S. (2018). Regenerative surgical therapy for peri-implantitis using deproteinized bovine bone mineral with 10% collagen,

enamel matrix derivative and Doxycycline-A prospective 3-year cohort study. *Clinical Oral Implants Research* 29 (6): 583–591.

- **14** Parma-Benfenati, S., Roncati, M., and Tinti, C. (2013). Treatment of peri-implantitis: surgical therapeutic approaches based on peri-implantitis defects. *International Journal of Periodontics and Restorative Dentistry* 33 (5): 627–633.
- **15** Froum, S.J., Froum, S.H., and Rosen, P.S. (2015). A regenerative approach to the successful treatment of peri-implantitis: a consecutive series of 170 implants in 100 patients with 2- to 10-year follow-up. *International Journal of Periodontics and Restorative Dentistry* 35 (6): 857–863.
- **16** Zipprich, H., Weigl, P., Di Gianfilippo, R. et al. (2022). Comparison of decontamination efficacy of two electrolyte cleaning methods to diode laser, plasma, and air-abrasive devices. *Clinical Oral Investigations* 26 (6): 4549–4558.
- **17** Renvert, S., Lindahl, C., Roos Jansåker, A.M., and Persson, G.R. (2011). Treatment of peri-implantitis using an Er:YAG laser or an air-abrasive device: a randomized clinical trial. *Journal of Clinical Periodontology* 38 (1): 65–73.
- **18** Schwarz, F., John, G., Schmucker, A. et al. (2017). Combined surgical therapy of advanced peri-implantitis evaluating two methods of surface decontamination: a 7-year follow-up observation. *Journal of Clinical Periodontology* 44 (3): 337–342.
- **19** Norton, M.R. (2017). Efficacy of Er:YAG laser in the decontamination of peri-implant disease: a one-year prospective closed cohort study. *International Journal of Periodontics and Restorative Dentistry* 37: 781–788.
- **20** Wang, C.W., Ashnagar, S., Gianfilippo, R.D. et al. (2021). Laser-assisted regenerative surgical therapy for periimplantitis: a randomized controlled clinical trial. *Journal of Periodontology 92*: 378–388.
- **21** Clem, D. and Gunsolley, J.C. (2019). Peri-implantitis treatment using Er:YAG laser and bone grafting. a prospective consecutive case series evaluation: 1 year posttherapy. *International Journal of Periodontics and Restorative Dentistry* 39: 479–489.
- **22** Deppe, H., Horch, H.H., and Neff, A. (2007). Conventional versus $CO₂$ laser-assisted treatment of peri-implant defects with the concomitant use of pure-phase beta-tricalcium phosphate: a 5-year clinical report. *The International Journal of Oral & Maxillofacial Implants* 22 (1): 79–86.

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- **23** Romanos, G.E., Ko, H., Froum, S., and Tarnow, D. (2009). The use of $CO₂$ laser in the treatment of periimplantitis. *Photomedicine and Laser Surgery* 27: 381–386.
- **24** Romanos, G.E. (1999). Laseranwendung bei der Periimplantitistherapie. *Ein Fallbericht. ZMK* 15: 818–819. (German).
- **25** Romanos, G.E. and Nentwig, G.H. (2005). Regenerative Therapie von periimplantären Knochendefekten: Literaturanalyse und eigene Daten. *Z. Zahnärztl. Implantol.* 21: 76–81. (German).
- **26** Al-Falaki, R., Cronshaw, M., and Hughes, F.J. (2014). Treatment outcome following use of the erbium, chromium:yttrium, scandium, gallium, garnet laser in the non-surgical management of peri-implantitis: a case series. *British Dental Journal 217*: 453–457.
- **27** Bach, G., Neckel, C., Mall, C., and Krekeler, G. (2000). Conventional versus laser-assisted therapy of periimplantitis: a five-year comparative study. *Implant Dentistry* 9: 247–251.
- **28** Romanos, G.E. (2002). Treatment of periimplant lesions using different laser systems. *Journal of Oral Laser Applications* 2: 75–81.
- **29** Papadopoulos, C.A., Vouros, I., Menexes, G., and Konstantinidis, A. (2015). The utilization of a diode laser in the surgical treatment of peri-implantitis. A randomized clinical trial. *Clinical Oral Investigations* 19 (8): 1851–1860.
- **30** Sculean, A., Deppe, H., Miron, R. et al. (2021). Effectiveness of photodynamic therapy in the treatment of periodontal and peri-implant diseases. *Monographs in Oral Science* 29: 133–143. Eick S (ed): Oral Biofilms.
- **31** Schwarz, F., Sahm, N., Schwarz, K., and Becker, J. (2010). Impact of defect configuration on the clinical outcome following surgical regenerative therapy of periimplantitis. *Journal of Clinical Periodontology* 37 (5): 449–455.
- **32** Schwarz, F., Jepsen, S., Herten, M. et al. (2006). Influence of different approaches on non-submerged and submerged healing of ligature induced peri-implantitis lesions: An experimental study in dogs. *Journal of Clinical Periodontology 33*: 584–595.
- **33** Monje, A., Pons, R., Roccuzzo, A. et al. (2020). Reconstructive therapy for the management of periimplantitis via submerged guided bone regeneration: a prospective case series. *Clinical Implant Dentistry and Related Research* 22 (3): 342–350.
- **34** Roos-Jansåker, A.M., Renvert, H., Lindahl, C., and Renvert, S. (2007). Submerged healing following surgical treatment of peri-implantitis: a case series. *Journal of Clinical Periodontology* 34 (8): 723–727.
- **35** Wen, S.C., Barootchi, S., Huang, W.X., and Wang, H.L. (2022). Surgical reconstructive treatment for

infraosseous peri-implantitis defects with a submerged healing approach: a prospective controlled study. *Journal of Periodontology* 93 (2): 195–207.

- **36** Astolfi, V., Gómez-Menchero, A., Ríos-Santos, J.V. et al. (2021). Influence of removing or leaving the prosthesis after regenerative surgery in peri-implant defects: retrospective study: 32 clinical cases with 2 to 8 years of follow-up. *International Journal of Environmental Research and Public Health* 18 (2): 645.
- **37** Parma-Benfenati, S., Tinti, C., Romano, F. et al. (2020). Long-term outcome of surgical regenerative treatment of peri-implantitis: a 2- to 21-year retrospective evaluation. *International Journal of Periodontics and Restorative Dentistry* 40 (4): 487–496.
- **38** Schwarz, F., Sculean, A., Bieling, K. et al. (2008). Twoyear clinical results following treatment of periimplantitis lesions using a nanocrystalline hydroxyapatite or a natural bone mineral in combination with a collagen membrane. *Journal of Clinical Periodontology 35*: 80–87.
- **39** Behneke, A., Behneke, N., and d'Hoedt, B. (2000). Treatment of peri-implantitis defects with autogenous bone grafts: Six-month to 3-year results of a prospective study in 17 patients. *The International Journal of Oral & Maxillofacial Implants 15*: 125–138.
- **40** Khoury, F. and Buchmann, R. (2001). Surgical therapy of peri-implant disease: a 3-year follow-up study of cases treated with 3 different techniques of bone regeneration. *Journal of Periodontology 72*: 1498–1508.
- **41** Aghazadeh, A., Persson, R.G., and Renvert, S. (2012). A single-centre randomized controlled clinical trial on the adjunct treatment of intra-bony defects with autogenous bone or a xenograft: results after 12 months. *Journal of Clinical Periodontology 39*: 666–673.
- **42** Nart, J., de Tapia, B., Pujol, À. et al. (2018). Vancomycin and tobramycin impregnated mineralized allograft for the surgical regenerative treatment of peri-implantitis: a 1-year follow-up case series. *Clinical Oral Investigations* 22 (6): 2199–2207.
- **43** Roccuzzo, M., Gaudioso, L., Lungo, M., and Dalmasso, P. (2016). Surgical therapy of single peri-implantitis intrabony defects, by means of deproteinized bovine bone mineral with 10% collagen. *Journal of Clinical Periodontology 43*: 311–318.
- **44** Wohlfahrt, J.C., Lyngstadaas, S.P., Rønold, H.J. et al. (2012). Porous titanium granules in the surgical treatment of peri-implant osseous defects: A randomized clinical trial. *The International Journal of Oral & Maxillofacial Implants 27*: 401–410.
- **45** Isehed, C., Holmlund, A., Renvert, S. et al. (2016). Effectiveness of enamel matrix derivative on the clinical and microbiological outcomes following surgical regenerative treatment of peri-implantitis. A randomized
controlled trial. *Journal of Clinical Periodontology 43*: 863–873.

- **46** Polymeri, A., Anssari-Moin, D., van der Horst, J. et al. (2020). Surgical treatment of peri-implantitis defects with two different xenograft granules: a randomized clinical pilot study. *Clinical Oral Implants Research* 31 (11): 1047–1060.
- **47** Ramanauskaite, A., Becker, K., Juodzbalys, G., and Schwarz, F. (2018). Clinical outcomes following surgical treatment of peri-implantitis at grafted and non-grafted implant sites: a retrospective analysis. *International Journal of Implant Dentistry* 4 (1): 27.
- **48** Persson, L.G., Berglundh, T., Lindhe, J., and Sennerby, L. (2001). Re-osseointegration after treatment of periimplantitis at different implant surfaces. An experimental study in the dog. *Clinical Oral Implants Research* 12 (6): 595–603.
- **49** Wetzel, A.C., Vlassis, J., Caffesse, R.G. et al. (1999). Attempts to obtain re- osseointegration following experimental periimplantitis in dogs. *Clinical Oral Implants Research* 10: 111–119.
- **50** Hämmerle, C.H., Fourmousis, I., Winkler, J.R. et al. (1995). Successful bone fill in late peri-implant defects using guided tissue regeneration. A short communication. *Journal of Periodontology* 66 (4): 303–308.
- **51** Stübinger, S., Henke, J., Donath, K., and Deppe, H. (2005). Bone regeneration after peri-implant care with the CO2 laser: a fluorescence microscopy study. *The International Journal of Oral & Maxillofacial Implants* 20 (2): 203–210.
- **52** Deppe, H., Horch, H.H., Henke, J., and Donath, K. (2001). Peri-implant care of ailing implants with the carbon dioxide laser. *The International Journal of Oral & Maxillofacial Implants* 16 (5): 659–667.
- **53** Fletcher, P., Deluiz, D., Tinoco, E.M. et al. (2017). Human histologic evidence of reosseointegration around an implant affected with peri- implantitis following decontamination with sterile saline and antiseptics: a case history report. *International Journal of Periodontics and Restorative Dentistry 37*: 499–508.
- **54** Nevins, M., Benfenati, S.P., Galletti, P. et al. (2020). Human histologic evaluations of the use of Er,Cr:YSGG laser to decontaminate an infected dental implant surface in preparation for implant reosseointegration. *International Journal of Periodontics and Restorative Dentistry* 40 (6): 805–812.
- **55** Lollobrigida, M., Fortunato, L., Lamazza, L. et al. (2020). Reosseointegration after the surgical treatment of induced peri-implantitis: systematic review on current evidence and translation from the animal to the human model. *Minerva Stomatologica* 69 (1): 37–54.
- **56** Tarnow, D. and Fletcher, P. (2015). Histologic evidence of the ability of dermal allograft to function as a barrier during guided bone regeneration-a case report. *Clinical Advances in Periodontics* 5: 201–207.
- **57** Fletcher, P. and Constantinides, C. (2015). Resolution of a peri-implantitis defect using sterile saline for surface detoxification: A case report with clinical re-entry. *Clinical Advances in Periodontics* 5: 235–241.

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Lasers in Surgical Therapy of Peri-implantitis

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Introduction

Similar to periodontitis, surgical therapy of peri-implantitis includes both regenerative and resective approaches [1]. During resective surgery of peri-implantitis, the goal is to remove granulation tissue, eliminate osseous cratering, and apically position the tissues. This is often done in combination with implantoplasty in which the goal is to smooth the implant threads and rough surface of the implant to provide a smoother surface unfavorable to biofilm formation [1]. However, implantoplasty is controversial in the amount of titanium particles expelled into the surrounding tissue during the process [2, 3]. Surgical therapy also involves decontaminating the implant surface and applying biological materials, such as bone grafts, membranes, and growth factors in an attempt to regenerate the lost implant–supporting structures [4].

Whether the treatment is non-surgical therapy, or surgical therapy, the goal in treating peri-implantitis is to disinfect the implant surface [5]. Even in resection of the peri-implant tissues, implantoplasty is often done in order to remove the contaminated titanium oxide layer and threads of the implant and provide a smoother surface less favorable for biofilm formation [4], while managing periodontitis on the other hand, teeth are debrided with curettes removing a superficial layer of cementum and bacterial load in the periodontal pocket prior to periodontal repair. However, curettes have shown to be ineffective against implant surfaces [5]. Additionally, utilizing curettes against implant surfaces has been shown to create titanium particles in the surrounding tissues, potentially contributing to the inflammatory response [3]. Therefore, alternative methods are pursued in decontaminating the implant surfaces including chemotherapeutics, such as antiseptics and antibiotics, as well as laser irradiation and photodynamic therapy [6]. Laser irradiation allows for the decontamination of the implant providing a useful adjunct in the management of peri-implantitis [7].

The term "laser" is an acronym for "Light Amplification for Stimulated Emission of Radiation" [8, 9]. Laser light differs from standard light in that it is monochromatic, coherent, and collimated [8, 10]. Each laser system has an active medium that directly contributes to the monochromatic wavelength produced. Knowledge of the various wavelengths is critical as this directly relates to the type of laser-tissue interaction utilized [8, 10]). Specifically in medicine, there are four different types of interactions with tissues [8, 9]. These include absorption, reflection, transmission, and scattering of the laser light [8, 9]. While absorption is the most desired outcome, the type of lasertissue interactions depends on both the laser wavelength and the characteristics of the tissue [8, 9]. Depending on the laser wavelength, there are different effects in both hard and soft tissues as well as titanium surfaces [7, 8, 9]. Given that different wavelengths exhibit different lasertissue interactions depending on the targeted chromophore, each wavelength will exhibit different interactions with the titanium surface. Lasers in the treatment of periimplantitis in general lacks significant evidence due to the difficulties of conducting randomized controlled clinical trials in a split-mouth fashion. Therefore, more publications are needed from basic in-vitro studies, animal studies, and various clinical case series and randomized clinical trials. The purpose of this chapter was to review the characteristics of various laser wavelengths available in dentistry and discuss their effects on current evidence on interactions with titanium implant surfaces and treatment in peri-implantitis.

Diode Lasers for Peri-implantitis Therapy

Diode lasers are classified by having a semi-conductor as their active medium with resulting wavelengths in the infrared and near-infrared spectrum. While the most commonly utilized laser wavelengths in dentistry range from 810 to 980nm, a new 445nm blue light has shown promising results in wound healing and warrants further investigation [11, 12] (Figure 13.1).

Given that diode lasers have a relatively high absorption coefficient for melanin and hemoglobin, near-infrared diode lasers are advantageous when utilized for coagulation and soft tissue surgery [10]. Diode lasers are commonly used in clinical settings due to the relatively low cost and smaller size when compared to other wavelengths such as the Nd:YAG and Er:YAG [13, 14]. The delivery method for diode lasers is fiberoptic tips which provide versatility to its use [15, 16] (Figure 13.2). Initiators such as articulating

Figure 13.1 New developed diode laser with visible blue light (445nm) and near-infrared 970nm laser wavelength (Sirona, Bensheim, Germany).

paper or a cork provide a dark pigmented mass at the end of the fiberoptic tip which focuses the laser beam. This provides concentrated maximum energy on the targeted tissue, creating a "hot tip" for precise cutting of the soft tissues [15, 16]. Diode lasers are very effective in cutting soft tissue precisely and are known for stalling and dragging during the cutting process if not reinitiated [14]. However, this is an advantage of diode lasers as the tissue can be "felt" while cutting unlike the non-contact devices, such as $CO₂$ and erbium lasers [14]. In contrast, non-initiated tips can be used in a non-contact modality to provide a defocused beam aiding in coagulation or for photobiomodulation [10]. To further increase its versatility, diode lasers are commonly utilized during photodynamic therapy where a photosensitizer is activated by the laser light to carry out a desired photochemical effect. When evaluating the potential and use of diode lasers in peri-implantitis, it is important to consider the method utilized and the combination of initiated, versus non-initiated tip in addition to the wavelength. Additionally laser safety protocols to control overheating and risks from laser irradiation.

Effects of Diode Lasers on Titanium Surfaces

Previous studies on laser-irradiated titanium discs with either Nd:YAG and 980nm diode lasers showed that the 980nm diode laser displayed no surface damage even at 15W compared to the use of pulsed Nd:YAG laser which melted the titanium surface and created cracks and other surface damages [17]. This is consistent with the study by Kreisler et al. in which the Nd:YAG, Ho:YAG, Er:YAG, CO2, and GaAlAs (diode) lasers were compared on various implant surfaces. Sandblasted, acid-etched, plasma-sprayed, hydroxyapatite-coated, and smooth titanium disks were

Figure 13.2 Irradiation of an implant for decontamination of the implant surface (810nm diode, Ivoclar, Amherst, NY).

irradiated with different laser groups and analyzed via SEM and energy dispersive spectroscopy [18]. Consistent with the previous studies mentioned, the Nd:YAG laser-induced melting, cracking, and crater formation on all implant surfaces. The Ho:YAG laser also produced these surface alterations on all tested surfaces. The Er:YAG and the $CO₂$ laser caused surface alterations only when utilized at higher power output, specifically with superpulsed mode for the $CO₂$ laser. The GaAlAs (809 nm) appeared to be the safest laser and did not induce any damage on any of the titanium disks [18]. Another in-vitro study analyzed the thermal changes on titanium surfaces after irradiation with diode, Er:YAG, and $CO₂$ lasers [19]. The diode laser demonstrated clinically unacceptable temperature changes for both the 810 and 980nm when utilized in continuous mode [19]. Therefore, carefully selected parameters are required when utilizing diode lasers on implant surfaces. Similarly, in-vitro studies assessed the thermal effect of the 445nm diode laser on five dental implant systems. The studies showed that laser irradiation systems can prevent harmful rises in temperature and surface alteration when used at moderate laser parameters [20]. It was shown that the 445nm wavelength caused temperature increases of more than 10°C at or above the 0.8W power level working in continuous wave mode for 5 seconds and in pulsed mode at 3W for 20 seconds with 10% duty cycle. The highest rises in temperature were seen in the Straumann Pure ceramic implant, lowest temperature observed in the Ankylos system.

Clinical Studies

In a clinical, split-mouth trial involving 10 patients with 48 implants, a diode laser (810nm) as an adjunct to traditional scaling was evaluated. However, the trial concluded that the diode lasers did not yield any positive influence on periimplantitis when used in conjunction with mechanical therapy [21]. In another randomized clinical trial, 27 patients requiring treatment for peri-implantitis were treated with either conventional non-surgical therapy alone or in combination with the 810nm diode laser [22]. With a total of 125 implants (750 sites) included, the authors found a statistically significant reduction in PPD and BOP in the treatment group compared to the control group. However, the study only had a follow-up of six months. Both of these studies were included in a systematic review evaluating the management of peri-implantitis with the diode laser in non-surgical therapy [23]. All three studies included utilized the 810nm wavelength. While the two studies discussed above showed statistical significance with the diode as an adjunctive therapy, the randomized clinical trial by Bach et al. [24] failed to report a statistical significance

between the two groups after a five-year follow-up [24]. Due to the high heterogeneity of the studies, no meta-analysis was completed. Therefore, the results of the systematic review were that additional studies are needed, and the evidence thus far does not support the adjunctive use of diode lasers in non-surgical peri-implantitis therapy.

In regard to surgical therapy, a randomized clinical trial evaluated the efficacy of utilizing a diode laser as an adjunct in conventional open-flap debridement of failing implants. With 19 patients evaluated, both groups showed significant improvement in periodontal parameters after six months of treatment. However, the group irradiated with the diode laser only provided marginal improvement in CAL, while no difference between test and control groups was noted for PD and BOP [25]. However, more studies are necessary with the 810nm and other diode wavelengths in the surgical management of peri-implantitis.

Summary of Diode Lasers in Peri-implant Therapy

While there is a lack of clinical trials on the use of diode lasers in peri-implantitis therapy, in-vitro results are promising. Studies by Kreisler et al. and Romanos et al. displayed no surface alterations to the titanium disks with an 810 and 980nm diode laser when compared to the Nd:YAG laser which produced irreversible changes [17, 18]. While the adjunctive use of surgical therapy with the 810nm diode laser showed promising results by Papadopoulos et al. [25], additional studies are necessary. Additionally, a newer diode wavelength of a visible blue light with a 445nm wavelength has been suggested to have improved hemostatic effects and wound healing potential [12, 26]. However, this wavelength has not yet been investigated clinically in periimplantitis treatment. Given that the goal of laser irradiation in peri-implant and periodontal protocols is to produce a blood coagulum in the base of the pocket to promote regeneration, the blue light might be a useful tool in periimplantitis. However, more research with this wavelength is necessary, especially in in-vivo applications. While there is a general lack of study on diode lasers in peri-implantitis therapy, it is important for future studies to evaluate both initiated and non-initiated tips as well as the variable wavelengths available in the diode family.

Nd:YAG Lasers in Peri-implantitis Therapy

Nd:YAG lasers have a solid-state active medium in which about 1% of the yttrium is replaced with neodymium. The resulting wavelength is 1064 nm therefore having a deep penetration depth in soft tissues [7, 9] a strong coagulation affect [5, 10] and has also been successful in decontaminating the periodontal pockets [27]. While its first use in dentistry was for the removal of caries [28] the Nd:YAG laser is primarily utilized for periodontal therapy and soft tissue excision [7]. The Nd:YAG laser is most commonly known today by its trademarked technique: Laser-Assisted New Attachment Procedure (LANAP®), which includes a specific protocol for periodontal regenerative therapy that has been verified with histological evidence [29, 30]. Although it is one of the most commonly utilized lasers in periodontal therapy, it has the deepest penetration depth in soft tissue [7] and due to a high risk of overheating (Figure 13.3), this laser is only utilized in dentistry in a pulsed setting.

While it's success in periodontal therapy is well documented, the Nd:YAG has actually shown to have detrimental effects when utilized on implant surfaces. Block et al. [31] reported irreversible changes including melting, loss of porosity, cracking, and ruptures on the surfaces of the HA-coated implant in an in-vitro setting [31]. Alarmingly, this occurred at all power settings tested. In a different study by Romanos et al. [17], three different titanium discs which were either hydroxyapatite (HA)-coated, titanium plasma sprayed (TPS), or sandblasted were irradiated with an Nd:YAG or 980nm diode laser [17]. Scanning electron microscopy displayed extensive melting in all Nd:YAG laser irradiated areas. All disks that were TPS and HA-coated displayed damage from the dental pulsed Nd:YAG laser, even at the lowest power setting of 2.0W [17].

Summary of Nd:YAG Lasers in Peri-implant Therapy

In summary, the undesirable outcomes with the Nd:YAG laser on titanium implants are well documented [17, 18, 32, 33. Only when utilized in a soft setting the Nd:YAG achieved desirable results [34]. Therefore, it is becoming increasingly understood by clinicians that utilizing the Nd:YAG laser on implant surfaces should be avoided.

Erbium Lasers in Peri-implantitis Therapy

The pulsed Er:YAG laser (2940nm) has the dual ability to effectively ablate both soft and hard biological tissues with minimal thermal side-effects, due to its high absorption by water (Figure 13.4) [35, 36]. The Er,Cr:YSGG laser (2780nm), another laser type from the erbium laser family, also shows performance similar to the Er:YAG, with a difference being that the Er,Cr:YSGG laser's energy is absorbed more by hydroxyl (OH−) ions than by water molecules [37]. Thus,

Figure 13.3 Titanium surface showing melting after Nd:YAG laser irradiation.

erbium lasers can be applied not only for oral soft tissue surgery but also for hard tissue treatments such as caries removal, root surface debridement, bone ablation, as well as implant surface debridement [38]. Currently, erbium laser systems have the broadest range of periodontal and periimplant applications (Figure 13.5) [7, 38, 39].

Tissue Ablation and Wound Healing Following Erbium Laser Irradiation

Erbium lasers evaporate soft tissues via photo-thermal effects, delivering precise ablation with the advantage (among available dental lasers) of minimal thermal effects [40–42], particularly when used in combination with a water spray [38, 40]. Between the Er:YAG and Er,Cr:YSGG lasers, the effect of soft tissue ablation is reportedly greater for Er:YAG, with less thermal sideeffects, compared to Er,Cr:YSGG [40, 43]. The width of the coagulation layer on porcine gingival tissue following erbium laser ablation is minimal: approximately 18μm with water spray for Er:YAG and 33μm for Er,Cr:YSGG with water spray [40]. Compared to electrosurgery, Er:YAG laser surgery shows much less thermal change in gingiva as well as faster and more favorable gingival wound healing [44]. These characteristics make the erbium laser a suitable choice for peri-implant soft tissue management [7]. Erbium lasers can also ablate hard tissues such as calculus, root cementum and dentin, and bone without major thermal damage [38, 39, 45]. This is accomplished through "thermo-mechanical" effects based on the production of "micro-explosions" of water molecules within the hard tissues [38, 39, 46, 47].

Figure 13.5 Erbium Laser apparatuses and associated handpieces. (a) Er:YAG, Erwin AdvErLTM Evo, J. Morita Mfg. Corp., Kyoto, Japan (b) Er:YAG, LightTouch, Light Instruments Ltd., Yoknea, Israel (c) Er:YAG, LightWalker AT S NE, Fotona, d.o.o., Ljubljana, Slovenia (d) Er,Cr:YSGG, Waterlase iPlus, BIOLASE, Inc., California, USA.

In addition, lasers possess the interesting and characteristic property of bio-stimulation effects (photobiomodulation: PBM) induced by low power laser energy [48, 49]. Briefly, during soft tissue surgery with high power laser irradiation, a certain amount of low energy simultaneously scatters or penetrates into the surrounding or underlying tissues, stimulating biological response and promoting wound healing/ regeneration [50]. The Er:YAG laser, also capable of PBM effects, induces promotion of cell proliferation [51–54] and enhances calcification on osteogenic cells [55–57].

Effects of Erbium Lasers on Titanium Surfaces

Erbium lasers cause no visible changes to the titanium surface under appropriate irradiation conditions, although irradiation at high energy levels produces distinct surface changes on titanium [58–64] (Figure 13.6). The use of water spray minimizes temperature elevations of titanium during Er:YAG laser irradiation [58, 65]. The Er,Cr:YSGG laser can also be applied safely and effectively on the titanium surface in a manner similar to that with the Er:YAG laser [62, 66, 67].

The erbium lasers are capable of effectively removing calcified deposits and plaque on micro structured surfaces [64, 67–70], when compared to conventional mechanical methods such as ultrasonics, plastic or titanium curettes, or cotton pellets [71–73] (Figure 13.7). The Er:YAG laser possesses a high bactericidal potential on implants with different surface characteristics, even at low energy densities [74]. Erbium laser decontaminated surfaces recover their biocompatibility, showing the attachment of osteoblasts or fibroblasts [61, 67, 75, 76]. Thus, both Er:YAG laser and Er,Cr:YSGG laser irradiation are effective methods for decontamination without surface alterations under suitable irradiation conditions [7, 77].

The erbium lasers can preserve most of the microstructured surfaces under suitable irradiation conditions, but the anodic oxidized surface cannot be preserved by the Er:YAG laser [59, 64, 78]. With the anodized microstructure, instead of being preserved, complete peeling off the oxidized layer followed by exposure of a fresh rough titanium surface has been proposed [79, 80]. Thus, erbium lasers generally show effective and safe application for the debridement of microstructured titanium surfaces except for anodized surfaces. Nonetheless, when erbium lasers are used on titanium surfaces, the operator always needs to be mindful of the irradiation parameters and conditions so as not to induce the concentration or accumulation of heat during irradiation [7, 62, 64].

Figure 13.6 Stereomicroscopy and scanning electron microscopy (SEM) images of commercially available micro-structured titanium implants after Er:YAG laser irradiation. Er:YAG laser spot irradiation was performed at 30, 40, and 50mJ/pulse (energy density: 10.6, 14.2, and 17.7J/cm²/pulse) and 30Hz in near contact mode for five seconds. For the SLA® surface, 30 and 40 mJ/pulse with water spray showed no changes in stereomicroscopy observations, but 40mJ/pulse produced a slight change. In SEM images, 40 and 50mJ/pulse with water spray showed slight melting. Without water, 50 mJ/pulse produced severe thermal changes. For the TiUnite® surface, the anodized microstructure was always destructed, resulting in exposure of a fresh rough titanium surface (arrowheads) below the original microstructure. Arrows indicate color change, melting, cracking, or resolidification of the micro structured surface. Control: non-irradiated control; Water: water spray. (*Source:* Lasers in Medical Science © 2013 / with permission from Springer Nature).

(a)

(b)

Figure 13.7 Stereomicroscopy images of contaminated microstructured surface of an explanted implant after Er:YAG laser irradiation and ultrasonic scaling (US) with a plastic tip. Before treatment, calcified deposits (arrowheads) were evident on the microstructured surface (a). Er:YAG laser irradiation was performed at 20 and 30mJ/pulse (energy density: 7.1 and 10.6 J/cm²/pulse) and 30 Hz under water spray in near contact mode. Calcified deposits were effectively removed without visible damage to the microstructured surface in all treated areas (b). Thin white-gray fragments (possibly plastic debris) were observed throughout the US-treated area (b). (*Source:* Lasers in Medical Science © 2013 / with permission from Springer Nature).

In-vivo Studies of Erbium Lasers in Peri-implantitis Therapy

Erbium lasers, different from other high-power lasers, can be advantageously applied not only for fixture debridement but also for degranulation in the peri-implant bone defect [60, 81–83].

In animal studies on surgical therapy of periimplantitis, Schwarz et al. [84] reported that the application of Er:YAG laser irradiation during flap surgery seemed to promote re-osseointegration on contaminated implant surfaces more than plastic curettes plus metronidazole gel and ultrasonic devices in a circumferential crater-like bone defect. Takasaki et al. [85] also demonstrated that the Er:YAG laser debridement of bone defects as well as implant surfaces tended to produce greater bone-implant contact than curette treatment in flap surgery for experimentally-induced peri-implant infection in dehiscence-type defects in dogs (Figure 13.8). Nevins et al. [80] observed in dog experiments that Er:YAG laser decontamination of implant surfaces allowed for regeneration and improved bone-to-implant contact.

Clinical Studies

Recently, erbium lasers have been increasingly applied non-surgically and surgically in peri-implantitis treatment [7, 86, 87].

Non-surgical Therapy

In non-surgical applications, Schwarz et al. [72, 88] reported that Er:YAG laser treatment led to significant clinical improvements six months after therapy with a significant higher reduction of BOP compared to mechanical debridement using plastic curettes and antiseptic therapy, but its effectiveness seemed to be limited to a period of six months and may not be sufficient for the maintenance of failing implants in the long term due to a remaining mixed chronic inflammatory cell infiltrate in the connective tissue stroma [89]. Also, Renvert et al. [1] and Persson et al. [90] reported that clinical improvements following the non-surgical treatment of peri-implantitis lesions using an Er:YAG laser was limited at six months.

From systematic reviews, although Muthukuru et al. in 2012 [91] and Yan et al. in 2015 [92] reported that Er:YAG laser non-surgical treatment could potentially provide short-time additional benefits in comparison to conventional treatment, Kotsakis et al. in 2014 [93] and Mailoa et al. [94] could not identify any superiority of laser treatment.

Thus, based on the current evidence of short-term benefits of laser use in peri-implant disease, non-surgical laser therapy might be useful as initial treatment for controlling acute inflammation before surgical therapy [86]. Nonsurgical therapy seems to not always be sufficient for

Figure 13.8 Surgical treatment of experimentally induced dehiscencetype peri-implantitis lesions in dogs. At the time of access for therapeutic surgery, all defects were filled with granulation tissue (a and b). Degranulation and implant surface decontamination were performed by either an Er:YAG laser device (c), or plastic curets (d). At four months following therapy, favorable formation of new bone was observed in the laser group (e) when compared to the control group (f). Villanueva golder stain, bar = $500 \mu m$, BBD, bottom of the bone defect; CBD, coronal level of bone defect. (*Source:* Lasers in Medical Science © 2007 / with permission from Springer Nature.

complete healing of peri-implantitis sites [88], which indicates that depending on the case, a surgical approach may be necessary for the treatment of peri-implant lesions in order to achieve favorable clinical healing.

On the other hand, Al-Falaki et al. [95] reported that the use of Er,Cr:YSGG laser is effective as a non-surgical aid in the management of peri-implantitis in the majority of their cases, but precisely speaking, their method included not only implant surface debridement but also bone defect debridement, suggestive of flapless surgery. Mizutani et al. [86] also reported the method of minimally invasive flapless surgery (Figure 13.9).

Surgical Therapy

In the surgical approach, erbium lasers have some advantageous therapeutic effects allowing thorough debridement of implant surfaces as well as bone defects, and thus have potential to induce improved or favorable bone healing [60, 82, 96]. Compared to conventional mechanical instruments, erbium lasers allow more effective debridement of the narrow and deep bone defects in the early stage of periimplantitis (Figure 13.10).

Previously, several case studies have demonstrated successful treatment outcomes, with significant pocket depth reduction as well as excellent bone regeneration, following surgical therapy with Er:YAG laser debridement and bone augmentation (Figure 13.11) [79, 80, 97–99]. Reported that surgical therapy is a predictable method for treating periimplant disease but that laser treatment of the exposed implant surface during surgery of peri-implantitis did not prove to be beneficial. In a similar vein, Schwarz et al. [100–102] indicated in their RCT studies, including Er:YAG laser treatment, that the method of surface debridement and decontamination had no significant impact on clinical outcomes following surgical therapy of advanced peri-implantitis lesions.

Recently, Norton et al. [103] reported in their prospective closed cohort study that the Er:YAG laser can decontaminate the implant surface and allow for re-osseointegration. The baseline mean PPD for the original 27 implants was 5.9 mm while at the one-year review, the remaining 24 implants had a mean PPD of 3.1 mm

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Figure 13.9 Non-surgical therapy (minimally invasive flapless surgery) of peri-implantitis with Er:YAG laser in a 57-year-old male. After the use of systemic antibiotics in the acute phase of peri-implantitis, probing pocket depth at the mesial-palatal site of the #26 (FDI) was reduced from 6mm two weeks earlier to 5mm with bleeding on probing (BOP) (a). Following crown removal, a gingival cleft was evident in the mesial interdental gingiva (b). The inflamed gingival tissue was removed and diseased granulation tissue was thoroughly removed from the bone defect in the mesial site using only a Er:YAG laser at approximately 35mJ/pulse (panel setting 70 mJ/pulse, energy density: approximately 12.0J/cm²/pulse) and 25 Hz in contact mode under water spray leaving two threads of fixture surface partially exposed. The exposed microstructured fixture surface was also carefully debrided with the Er:YAG laser. The buccal and palatal gingival tissue was further ablated with the Er:YAG laser to prevent a distinct gingival defect production and to obtain smooth interdental gingival architecture following wound healing. Effective and safe debridement was achieved without any visible signs of major thermal damage such as carbonization and coagulation on the laser-treated gingival, bone, and implant fixture surfaces. After debridement, a sufficient amount of bleeding was observed in the defect, and non-contact defocused irradiation without water spray was used to perform coagulation according to the Er:YAG laser-assisted comprehensive periodontal therapy (Er-LCPT) protocol [7]. The coagulation was performed in order to establish a stable blood clot in the gingival defect and surrounding gingival tissue (c) Following surgery, an uneventful healing was observed, and no complications or side effects were observed in the treated tissues. Three weeks after surgery, a concave gingival contour was still observed in the mesial interdental area (d). The defect gradually diminished and disappeared around four months due to the regeneration of healthy gingival tissue (e). Six years following surgery, the condition was well maintained, and no signs of gingival inflammation were observed. The probing depth decreased to 2mm without BOP (f) (Case details provided by Akira Aoki) (*Source:* Mizutani K et al. Lasers in minimally invasive periodontal and peri-implant therapy. Periodontol 2000 © copyright 2016 / with permission from John Wiley & Sons) [86].

and signs of inflammation were notably diminished overall. In 2021, Wang et al. [104] reported, in their randomized controlled clinical trial, that following the adjunctive use of Er:YAG laser to modulate and remove inflammatory tissue as well as to decontaminate the implant surface, the Er:YAG laser group demonstrated significantly higher PPD reductions at the site level compared 13 to the control group and that Er:YAG laser treatment during peri-implantitis regenerative therapy may aid in better probing PD reduction. Thus, in the case of Er:YAG laser application, its use for bone defect debridement might have more advantageous impacts, compared to conventional mechanical therapy, due to its antibacterial and PBM effects.

Summary of Er:YAG Lasers in Surgical Peri-implant Therapy

Lasers are gradually being applied in the surgical treatment of peri-implantitis. Based on current evidence, erbium lasers are promising laser systems as they can be used to debride both contaminated fixture surfaces and bone defects. Although erbium laser peri-implant therapy tends to show better results in animal studies, significant benefits of erbium laser therapies have not yet been demonstrated in the limited number of clinical studies. Recently, the American Academy of Periodontology best evidence consensus statement in 2018 [105, 106] reported that "When lasers are used to treat peri-implantitis surgically, most studies show a reduction in bleeding on probing; however,

 $\qquad \qquad \text{(g)}\qquad \qquad \text{(i)}$

Figure 13.10 Surgical therapy of peri-implantitis with Er:YAG laser. 64-year-old male. Before surgery (a), the probing depth around implants at the site of #14 -16 (FDI) was maximally 7mm with bleeding on probing (BOP). The mesial site of the #14 implant (14i) showed a probing depth of 7mm with BOP (b). The palatal view before surgery (c). After flap reflection, debridement of bone defects and fixture surfaces was effectively performed with the Er:YAG laser at 30-40mJ/pulse (panel 60-80mJ/pulse, 10.7-14.2J/cm²/pulse) and 20–25Hz under saline water spray. In particular, the bone defect around 14i was very narrow and deep, and only the thin 400μm-diameter contact tip was available for insertion and debridement of the defect (d). In such a case, entire fixture debridement using the laser was incomplete. Nonetheless, a decontamination effect from the explosive ablation of water was expected during irradiation. After debridement, moderate to severe vertical bone defects were observed around 14i and the #16 implant (16i), and a fenestration (arrow) was observed in the buccal bone around the apex of the #15 implant fixture (15i) (e). The palatal site of 14i also demonstrated vertical bone resorption with reduced marginal bone height (f). Before flap replacement, osseous recontouring was performed around implants to correct irregular bone contours using the Er:YAG laser. Autogenous bone collected from the area distal to 16i was grafted in the vertical bone defects around all the implants, and guided bone regeneration (GBR) using absorbable membranes was performed at the buccal fenestration site of 15i and palatal vertical bone defect of 14i. One week after surgery, wound healing was uneventful (h) except for membrane exposure (arrow) which was noted at the palatal inter-implant area of 14i and 15i (i). Later, the exposure was gradually minimized and eventually resolved. Eight months post-surgery, peri-implant tissues stabilized without inflammation although some gingival recession occurred (k, l). The probing depth at all sites, including the mesial site of 14i, decreased to 2–3mm without BOP. GBR: guided bone regeneration. (*Source:* Aoki A et al. Periodontal and peri-implant wound healing following laser therapy. Periodontol 2000 © 2015 / with permission from John Wiley & Sons, Inc) [7].

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short-term data demonstrate inconsistency with regard to pocket depth reduction, clinical attachment level gains, and bone fill." Thus, further long-term randomized controlled clinical trials are needed to establish the superiority of erbium laser applications in the non-surgical and surgical management of peri-implantitis. Nevertheless, based on previous reports and clinical experiences, it can be concluded that the application of erbium lasers has some technical and therapeutic advantages as an alternative or adjunctive modality in the management of peri-implant diseases.

CO2 Lasers

The $CO₂$ laser was the first laser used in dentistry [14] and is available at the 10,600nm and the newer 9300nm wavelengths. The carbon dioxide laser has a high affinity for absorption of water (although it absorbs water 10 times less efficiently than Er:YAG), and hydroxyapatite with a shallow penetration depth [107]. With its ability to coagulate, it is ideal for soft tissue use and for removing epithelial tissue without causing damage to the underlying connective

tissues [107, 108]. Here, the wavelength 9300nm should be indicated with the $CO₂$ laser. The 10600nm $CO₂$ cannot ablate enamel and dentin. Melcer et al. [109] provided histological evidence in an animal study displaying formation of secondary dentin and sterilization of the dentin and pulp with the $CO₂$ laser [109]. Given that the laser is noncontact, it is more technique-sensitive with precise aiming and manual control of the focal point [14].

CO2 Lasers in Peri-implantitis Therapy

The first clinical case report was presented in 1999 in a German scientific journal [110].

In a later study by Kreisler et al. (2002) the $CO₂$ wavelength was utilized without damaging the implant surface only when proper parameters were utilized [18]. This is consistent with an in-vitro study by Deppe et al. [111] in which the $CO₂$ laser displayed melting and other surface alterations only on specific parameters, specifically in superpulsed mode [111]. However, in continuous mode, no surface alterations were found, even at higher power outputs [111]. The bactericidal efficacy of the carbon dioxide laser on a contaminated implant surface was also evaluated as well as surface changes, temperature rise, and cell adhesion [112]. Titanium disks contaminated with *P. gingivalis* and *S. sanguis* were irradiated with an expanded $CO₂$ laser beam with temperature measured with a thermograph and the surrounding fibroblasts and osteoblasts were monitored for migration into the irradiated area [112]. They observed that all microbial cells were killed in the irradiated area, without a rise in surface temperature. Additionally, osteoblasts and fibroblasts did not display any damage outside the zone of irradiation and had actually migrated onto the irradiated surface afterwards [112]. Romanos et al. [66] also evaluated the osteoblastic activity on the different titanium surfaces (machined; titanium plasma-sprayed (TPS); hydroxyapatite-coated; sandblasted) with both $CO₂$ and Er,Cr:YSGG lasers [66]. All titanium surfaces were able to colonize osteoblasts in both laser-irradiated groups, initially suggesting that titanium surfaces treated with lasers could lead to re-osseointegration [66]. This was shown by Deppe et al. in an animal study involving beagle dogs [111]. Peri-implantitis-induced defects, which successfully underwent decontamination with $CO₂$ laser and GBR were able to regenerate previously lost bone which was confirmed with histological samples [111, 113]. Later on, a case series by Romanos and Nentwig [114], 19 implants with peri-implantitis underwent regenerative therapy utilizing a $CO₂$ laser to decontaminate the implant surface and form a coagulum in the peri-implant defects [114]. All treated implants resulted

in resolution of the inflammatory reaction and complete bone fill was observed radiographically 27 months post-operatively [114]. Of clinical interest is that many of the treated implants had bone loss of more than 50% of the implant length.

Summary of CO2 Lasers in Surgical Peri-implant Therapy

Current evidence suggests that the $CO₂$ laser is a useful tool in decontaminating the implant surface and aid in regeneration of the peri-implant defects. In-vitro studies seem to conclude that the $CO₂$ laser does not cause damage to the implant surface if proper power outage and settings are utilized. In a review article by Romanos et al. [115], the protocol for decontaminating the implant surfaces was suggested to have a power setting from 2 to 4W in a pulsed mode with a non-contact defocused handpiece to avoid risks of overheating [115]. The hemostatic ability of the $CO₂$ laser and its ability to form a coagulum in the peri-implant defects without damaging the implant surfaces is an advantage of the $CO₂$ laser in peri-implantitis therapy. However, additional multicenter clinical studies are necessary to confirm and further analyze the benefits of $CO₂$ laser irradiation in peri-implantitis therapy (Figure 13.12).

Photodynamic Therapy

Photodynamic therapy (PDT) is an emerging adjunctive therapy utilized to detoxify implant surfaces [116]. Photodynamic therapy involves the use of a chemical dye (so-called "photosensitizer") in conjunction with laser light and oxygen [117]. When activated by laser light, the photosensitizer enters an excited state initiating an electronic transition chain that produces short-lived reactive oxygen species (ROS) that cause oxidative damage to the targeted microbial or cancer cells [117, 118]. Thus, photodynamic therapy is a method in which laser light indirectly targets microbial cells and can be utilized as a method of decontamination.

The primary pathogens of peri-implantitis, and periodontitis for that matter, are gram-negative bacteria in the red complex, such as *P. gingivalis* [119, 120]. However, targeting gram-negative bacteria through photodynamic therapy has been a challenge due to the double lipid bilayer cell wall preventing the uptake of both anionic and neutral photosensitizers [117]. Therefore, alternative strategies including more cationic photosensitizers, or utilizing permeabilizing agents such as EDTA to disrupt the gram-negative cell membrane prior to PDT have been developed [117, 118]. Most commonly utilized in dentistry are phenothiazinium dyes, such as toluidine blue (TBO) and methylene blue (MB) [121].

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Figure 13.12 Peri-implant defect after degranulation (a) and irradiation with a CO₂ laser (b). Observe the blood clot stabilization on the implant surface (c). Bone grafting with bovine-based mineral (d) and coverage of the defect with a collagen membrane (e); flap closure with conventional interrupted sutures (f); preoperative clinical photograph (g) and a five-year follow up (h); preoperative radiograph demonstrating the peri-implant defect (i) and five-year postoperative radiograph (j) (*Source:* Dr. Georgios Romanos (faculty) and Dr. Lauren Spevack (resident) at the Dept. of Periodontology, Eastman Institute for Oral Health, Rochester, NY).

Figure 13.12 (Continued)

A systematic review by Vohra et al. [116] compared the efficacy of mechanical debridement in peri-implantitis patients with and without photodynamic therapy [116]. While the experimental animal studies suggested promising results with PDT as an adjunctive therapy, the results were underwhelming when translated to clinical studies. Out of six clinical studies included in their review, four of them reported comparable results to standard mechanical therapy when adjunctive PDT was utilized [116]. The authors hypothesized that the severity of peri-implantitis played a role in the success of the photodynamic treatment. One of their studies by Deppe et al. [122] only showed increased efficacy with PDT in pockets less than 5mm while probing depths from 5 to 8mm was ineffective at maintaining crestal bone levels [122]. The authors also suggested that in the experimental studies, it is a more controlled environment and easier to apply the photosensitizers than in the more narrow and hard-to-reach peri-implantitis defect in-vivo setting [116]. As we know, deeper probing depths encompass higher quantities of red complex bacteria and a potentially more mature biofilm [123]. Therefore, it may be difficult for the dye to penetrate deeper probing depths in in-vivo settings. Specifically, the clinical study by Doerbudak et al. [124] showed that the TBO photosensitizer with laser irradiation resulted in significant bacterial reduction in three periodonto-pathogenic bacteria. Even without laser irradiation, the photosensitizer alone resulted in a statistically significant reduction in A. *actinomycetemcomitans* and P*. intermedia*, however, the reduction in *P. gingivalis* was not significant [124]. However, with laser irradiation a significant reduction in all three species was observed but complete elimination of the tested bacteria was not able to be achieved. As we know, it is critical to decontaminate implant surfaces during maintenance appointments for peri-implantitis.

Additionally, the authors acknowledged that there was a significant variability in the frequency and duration of PDT and laser parameters utilized. The authors also noted that true comparison was difficult as there was a lack of control groups (mechanical debridement only) and clear treatment groups utilizing only adjunctive PDT [116]. Therefore, the authors decided that adjunctive PDT to traditional PDT

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remains inconclusive. While the laser parameters can easily be changed for experimental studies, the pharmacokinetics of the photosensitizer are not as controllable. Therefore the pursuit of an ideal photosensitizer is of major focus [118]. Eleven out of the twelve studies included in the review by Vohra et al. [116] utilized methylene blue or toluidine blue as their photosensitizers. However, the effectiveness of these dyes may fail in comparison to a newer generation of photosensitizers [118]. Nanoparticle-encapsulated methylene blue has shown an increase in efficacy over traditional methylene blue [118, 125]. Additionally, Safranine O and SAPYR are budding photosensitizers for biofilm disruption [126, 127]. Therefore, more experiments with these newer photosensitizers are necessary.

The effects of photodynamic therapy in peri-implantitis were investigated by Al Amri et al. [128] in a clinical trial involving 67 patients with peri-implantitis and type 2 diabetes mellitus [128]. The control group received mechanical debridement alone while the test group received mechanical debridement in combination with photodynamic therapy utilizing phenothiazine chloride as the photosensitizer and a 660nm diode laser as the light source [128]. Both probing depths and bleeding on probing were significantly lower in the test group at the 6-month and 12-month follow-ups suggesting that mechanical debridement with adjunctive PDT is a superior treatment to peri-implantitis compared to mechanical debridement alone [128]. Interestingly, the test group showed significantly lower levels in HbA1c at 6- and 12-month follow-ups where the control group did not report any changes in HbA1c levels [128]. The authors hypothesized that the adjunctive photodynamic therapy aided in decreasing the systemic as well as local inflammatory burden [128]. Utilizing the same photosensitizer, Javed et al. [129] conducted a clinical trial evaluating mechanical curettage with and without adjunctive PDT in the treatment of periimplant mucositis in cigarette smokers [129]. Fifty-four male patients were included in the study and divided into two different groups, one with adjunctive photodynamic therapy. The peri-implant parameters improved at 12 weeks in both groups; however, the plaque index and probing depths were decreased greater in the group without the photodynamic therapy.

Photodynamic Therapy in the Surgical Therapy of Peri-implantitis

Another systematic review by Chambrone et al. [130] concluded that photodynamic therapy did not give a significant benefit as an adjunct to peri-implantitis management; however, there was insufficient evidence provided as only two

studies were included [130]. One of which photodynamic therapy was utilized in conjunction with surgical management of peri-implantitis [131]. In a randomized case-control study, 40 patients with peri-implantitis underwent open flap debridement with and without PDT with TBO as the initiator and a diode 810nm as the laser. Anaerobic bacteria counts were taken before, three months, and six months post-operatively. In this study there was a statistically significant reduction in BOP, PPD, and inflammatory exudate (IE) at 24 months [131]. However, there was no statistically significant difference in the amount of periodontopathogens on the implant surfaces at the follow-up visit. The authors attributed this to the threads of the implant interfering with the light distribution on the targeted area [131].

Summary of PDT Remarks

The mechanism of action of PDT is dependent on various factors including type, dose, and localization of the photosensitizer, laser wavelength, power, frequency and settings, time of laser irradiation, target cell genotype, and intracellular ATP levels. Therefore, it is difficult to compare studies when many of these variables differ from study to study. This may explain the variability of outcomes in the nonsurgical management of peri-implantitis. In regard to surgical therapy of peri-implantitis, very few studies are available warranting more research to be done. While photodynamic therapy has a lot of potential in decontaminating the implant surface in the management of peri-implantitis, additional evidence in clinical studies is needed documenting consistent success, specifically in surgical peri-implantitis therapy. With new photosensitizers continuing to develop, the improvement of this field is inevitable.

Conclusions

A key goal to surgical therapy of peri-implantitis is to decontaminate the implant surface. Laser therapy provides a significant advantage in the surgical management of peri-implantitis for several reasons including its ability to destroy targeted bacteria without altering the implant surface if the right parameters are utilized. Giannini et al. [132] displayed successful decontamination of the implant surface with the Nd:YAG laser [132]. However, it has been shown that the Nd:YAG causes overheating of the implant surface leaving irreversible damage [17]. Therefore, the Nd:YAG laser Is not suitable for peri-implantitis therapy due to high risks of overheating. Comparatively, the diode lasers did not have the same surface altering effects and were beneficial as an adjunct to conventional therapy in both non-surgical and **Figure 13.13** Summary remarks of existing laser systems on surgical therapy of peri-implantitis.

surgical therapy of peri-implantitis [21, 25]. However, additional studies are necessary since the current literature provides insufficient data. Er:YAG and Er,Cr:YSGG are the most documented lasers to decontaminate the implant surface and have demonstrated success in many studies. A prominent clinical study by Schwarz et al. [96] compared the Er:YAG laser with plastic curettes and saline irrigation [72] and showed a reduction in bleeding on probing in the laser group but in a later study the same group showed no difference in bone fill in advanced peri-implantitis defects after use of the Er:YAG laser or plastic curettes with saline irrigation $[102]$. The $CO₂$ laser has documented success in many clinical cases, but some studies have shown that it might be harmful to the implant surfaces when utilized in superpulsed mode. However, when the correct settings are utilized, resulting bone fill has been documented [114]. Photodynamic therapy with low-power diode lasers is a relatively new and innovative field with success in treating periimplantitis via non-surgical therapy but requires more research with various wavelengths and photosensitizers in the surgical treatment of peri-implant diseases. Overall, utilizing lasers provides an alternative method to decontaminate the implant surface which is a critical component of treating peri-implantitis (Figure 13.13). As new wavelengths and settings are tested, more efficient protocols will continuously emerge.

References

- **1** Renvert, S., Lindahl, C., Roos Jansaker, A.M., and Persson, G.R. (2011). Treatment of peri-implantitis using an Er:YAG laser or an air-abrasive device: a randomized clinical trial. *Journal of Clinical Periodontology* 38 $(1): 65 - 73.$
- **2** Barrak, F.N., Li, S., Muntane, A.M., and Jones, J.R. (2020). Particle release from implantoplasty of dental implants and impact on cells. *International Journal of Implant Dentistry* 6 (1): 1–9.
- **3** Delgado-Ruiz, R. and Romanos, G. (2018). Potential causes of titanium particle and ion release in implant dentistry: a systematic review. *International Journal of Molecular Sciences* 19 (11): 3585.
- **4** Renvert, S., Polyzois, I., and Claffey, N. (2012). Surgical therapy for the control of peri-implantitis. *Clinical Oral Implants Research* 23 (Suppl 6): 84–94.
- **5** Aoki, A., Ando, Y., Watanabe, H., and Ishikawa, I. (1994). In vitro studies on laser scaling of subgingival calculus with an erbium: YAG laser. *Journal of Periodontology* 65 (12): 1097.
- **6** Romanos, G.E., Javed, F., Delgado-Ruiz, R.A., and Calvo-Guirado, J.L. (2015). Peri-implant diseases: a review of treatment interventions. *Dental Clinics of North America* 59 (1): 157–178.
- **7** Aoki, A., Mizutani, K., Schwarz, F. et al. (2015). Periodontal and peri-implant wound healing following laser therapy. *Periodontology 2000* 68 (1): 217–269.

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- **8** Elavarasu, S., Naveen, D., and Thangavelu, A. (2012). Lasers in periodontics. *Journal of Pharmacy and Bioallied Sciences* 4 (6): 260–263.
- **9** Romanos, G. (2015). Current concepts in the use of lasers in periodontal and implant dentistry. (Invited Review) (Report). *Journal of Indian Society of Periodontology* 19 (5): 490.
- **10** Romanos, G. (2021). *Advanced Laser Surgery in Dentistry*. Hoboken: Wiley Publ 2021.
- **11** Estrin, N.E. (2021). Wound healing aspects of diode lasers in oral soft tissues. In: *Laser Light Therapy in Dentistry: Efficacy, Uses and Limitations* (ed. G. Romanos), 401–407. Nova Science Publishers.
- **12** Reichelt, J., Winter, J., Meister, J. et al. (2017). A novel blue light laser system for surgical applications in dentistry: evaluation of specific laser-tissue interactions in monolayer cultures. *Clinical Oral Investigations* 21 (4): 985–994.
- **13** Romanos, G. and Nentwig, G.H. (1999). Diode laser (980 nm) in oral and maxillofacial surgical procedures: clinical observations based on clinical applications. *Journal of Clinical Laser Medicine & Surgery* 17 (5): 193–197.
- **14** Romanos, G.E. (2013). Diode laser soft-tissue surgery: advancements aimed at consistent cutting, improved clinical outcomes. *Compendium of continuing education in dentistry (Jamesburg, NJ: 1995)* 34 (10): 752–757. quiz 758.
- **15** Romanos, G.E., Belikov, A.V., Skrypnik, A.V. et al. (2015). Uncovering dental implants using a new thermo-optically powered (TOP) technology with tissue air-cooling. *Lasers in Surgery and Medicine* 47 (5): 411–420.
- **16** Romanos, G.E., Sacks, D., Montanaro, N. et al. (2018). Effect of initiators on thermal changes in soft tissues using a diode laser. *Photomedicine and Laser Surgery* 36 (7): 386–390.
- **17** Romanos, G.E., Everts, H., and Nentwig, G.H. (2000). Effects of diode and Nd:YAG laser irradiation on titanium discs: a scanning electron microscope examination. *Journal of Periodontology* 71 (5): 810–815.
- **18** Kreisler, M., Götz, H., Duschner, H., and d'Hoedt, B. (2002). Effect of Nd: YAG, Ho: YAG, Er: YAG, CO 2, and GaAlAs laser irradiation on surface properties of endosseous dental implants. *International Journal of Oral & Maxillofacial Implants* 17 (2): 202–211.
- **19** Leja, C., Geminiani, A., Caton, J., and Romanos, G.E. (2013). Thermodynamic effects of laser irradiation of implants placed in bone: an in vitro study. *Lasers in Medical Science* 28 (6): 1435–1440.
- **20** Deppe, H., Ahrens, M., Behr, A.V. et al. (2021). Thermal effect of a 445 nm diode laser on five dental implant systems: an in vitro study. *Scientific Reports* 11 (1): 20174.
- **21** Arısan, V., Karabuda, Z.C., Arıcı, S.V. et al. (2015). A randomized clinical trial of an adjunct diode laser application for the nonsurgical treatment of peri-implantitis. *Photomedicine and Laser Surgery* 33 (11): 547–554.
- **22** Lerario, F., Roncati, M., Gariffo, A. et al. (2016). Non-surgical periodontal treatment of peri-implant diseases with the adjunctive use of diode laser: preliminary clinical study. *Lasers in Medical Science* 31 (1): 1–6.
- **23** Mattar, H., Bahgat, M., Ezzat, A. et al. (2021). Management of peri-implantitis using a diode laser (810 nm) vs conventional treatment: a systematic review. *Lasers in Medical Science* 36 (1): 13–23.
- **24** Bach, G., Neckel, C., Mall, C., and Krekeler, G. (2000). Conventional versus laser-assisted therapy of periimplantitis: a five-year comparative study. *Implant Dentistry* 9 (3): 247–251.
- **25** Papadopoulos, C.A., Vouros, I., Menexes, G., and Konstantinidis, A. (2015). The utilization of a diode laser in the surgical treatment of peri-implantitis. A randomized clinical trial. *Clinical Oral Investigations* 19 (8): 1851–1860.
- **26** Matys, J., Flieger, R., and Dominiak, M. (2017). Effect of diode lasers with wavelength of 445 and 980 nm on a temperature rise when uncovering implants for second stage surgery: an ex-vivo study in pigs. *Advances in Clinical and Experimental Medicine* 26 (4): 687–693.
- **27** Cobb, C.M., McCawley, T.K., and Killoy, W.J. (1992). A preliminary study on the effects of the Nd: YAG laser on root surfaces and subgingival microflora in vivo. *Journal of Periodontology* 63 (8): 701–707.
- **28** Myers, T. and Myers, W. (1985). In vivo caries removal utilizing the YAG laser. *The Journal of the Michigan Dental Association* 67 (2): 66–69.
- **29** Harris, D.M., Gregg, R.H. 2nd, McCarthy, D.K. et al. (2004). Laser-assisted new attachment procedure in private practice. *General Dentistry* 52 (5): 396–403.
- **30** Nevins, M.L., Camelo, M., Schupbach, P. et al. (2012). Human clinical and histologic evaluation of laser-assisted new attachment procedure. *International Journal of Periodontics & Restorative Dentistry* 32 (5): 497–507.
- **31** Block, C.M., Mayo, J.A., and Evans, G.H. (1992). Effects of the Nd: YAG dental laser on plasma-sprayed and hydroxyapatite-coated titanium dental implants: surface alteration and attempted sterilization. *International Journal of Oral & Maxillofacial Implants* 7 (4): 441–449.
- **32** Romanos, G.E., Gupta, B., Yunker, M. et al. (2013). Lasers use in dental implantology. *Implant Dentistry* 22 (3): 282–288.
- **33** Park, C.-Y., Kim, S.-G., Kim, M.-D. et al. (2005). Surface properties of endosseous dental implants after NdYAG and CO2 laser treatment at various energies. *Journal of Oral and Maxillofacial Surgery* 63 (10): 1522–1527.
- **34** Pick, R.M. and Colvard, M.D. (1993). Current status of lasers in soft tissue dental surgery. *Journal of Periodontology* 64 (7): 589–602.
- **35** Hale, G.M. and Querry, M.R. (1973). Optical constants of water in the 200-nm to 200-μm wavelength region. *Applied Optics* 12 (3): 555–563.
- **36** Niemz, M.H. (1996). *Laser-Tissue Interaction. Fundamentals and Applications*. Berlin: Springer-Verlag.
- **37** Featherstone, J.D.B. (2000). Caries detection and prevention with laser energy. *Dental Clinics of North America* 44: 955–969.
- **38** Aoki, A., Sasaki, K., Watanabe, H., and Ishikawa, I. (2004). Lasers in non-surgical periodontal therapy. *Periodontology 2000* 36: 59–97.
- **39** Aoki, A., Mizutani, K., Takasaki, A.A. et al. (2008. Erratum in: Gen Dent 57(1):94, 2009). Current status of clinical laser applications in periodontal therapy. *General Dentistry* 56 (7): 674–687.
- **40** Kawamura, R., Mizutani, K., Lin, T. et al. (2020). Ex vivo evaluation of gingival ablation with various laser systems and electroscalpel. *Photobiomodulation, Photomedicine, and Laser Surgery* 38 (6): 364–373.
- **41** Merigo, E., Clini, F., Fornaini, C. et al. (2013). Laserassisted surgery with different wavelengths: a preliminary ex vivo study on thermal increase and histological evaluation. *Lasers in Medical Science* 28 (2): 497–504.
- **42** Walsh, J.T. Jr., Flotte, T.J., and Deutsch, T.F. (1989). Er:YAG laser ablation of tissue: effect of pulse duration and tissue type on thermal damage. *Lasers in Surgery and Medicine* 9 (4): 314–326.
- **43** Walsh, J.T. Jr. and Cummings, J.P. (1994). Effect of the dynamic optical properties of water on midinfrared laser ablation. *Lasers in Surgery and Medicine* 15 (3): 295–305.
- **44** Sawabe, M., Aoki, A., Komaki, M. et al. (2015). Gingival tissue healing following Er:YAG laser ablation compared to electrosurgery in rats. *Lasers in Medical Science* 30 (2): 875–883.
- **45** Ting, C.C., Fukuda, M., Watanabe, T. et al. (2007). Effects of Er,Cr:YSGG laser irradiation on the root surface: morphologic analysis and efficiency of calculus removal. *Journal of Periodontology* 78 (11): 2156–2164.
- **46** Nelson, J.S., Yow, L., Liaw, L.H. et al. (1988). Ablation of bone and methacrylate by a prototype mid-infrared erbium: YAG laser. *Lasers in Surgery and Medicine* 8 (5): 494–500.
- **47** Nuss, R.C., Fabian, R.L., Sarkar, R., and Puliafito, C.A. (1988). Infrared laser bone ablation. *Lasers in Surgery and Medicine* 8 (4): 381–391.
- **48** Mester, E., Mester, A.F., and Mester, A. (1985). The biomedical effects of laser application. *Lasers in Surgery and Medicine* 5 (1): 31–39.
- **49** Mester, E., Spiry, T., Szende, B., and Tota, J.G. (1971). Effect of laser rays on wound healing. *American Journal of Surgery* 122 (4): 532–535.
- **50** Ohsugi, Y., Niimi, H., Shimohira, T. et al. (2020). In vitro cytological responses against laser photobiomodulation for periodontal regeneration. *International Journal of Molecular Sciences* 21 (23): 9002.
- **51** Aleksic, V., Aoki, A., Iwasaki, K. et al. (2010). Low-level Er:YAG laser irradiation enhances osteoblast proliferation through activation of MAPK/ERK. *Lasers in Medical Science* 25 (4): 559–569.
- **52** Kong, S., Aoki, A., Iwasaki, K. et al. (2018). Biological effects of Er:YAG laser irradiation on the proliferation of primary human gingival fibroblasts. *Journal of Biophotonics* 11 (3): e201700157.
- **53** Ogita, M., Tsuchida, S., Aoki, A. et al. (2015). Increased cell proliferation and differential protein expression induced by low-level Er:YAG laser irradiation in human gingival fibroblasts: proteomic analysis. *Lasers in Medical Science* 30 (7): 1855–1866.
- **54** Pourzarandian, A., Watanabe, H., Ruwanpura, S.M. et al. (2005). Effect of low-level Er:YAG laser irradiation on cultured human gingival fibroblasts. *Journal of Periodontology* 76 (2): 187–193.
- **55** Niimi, H., Ohsugi, Y., Katagiri, S. et al. (2020). Effects of low-level Er:YAG laser irradiation on proliferation and calcification of primary osteoblast-like cells isolated from rat calvaria. *Frontiers in Cell and Development Biology* 8: 459.
- **56** Ohsugi, Y., Katagiri, S., Hirota, T. et al. (2020). Laser irradiation decreases sclerostin expression in bone and osteogenic cells. *The FASEB Journal* 34 (9): 12877–12893.
- **57** Shimohira, T., Katagiri, S., Ohsugi, Y. et al. (2021). Comprehensive and sequential gene expression analysis of bone healing process following Er:YAG laser ablation. *Photobiomodulation Photomedicine and Laser Surgery* 39 (2): 100–112.
- **58** Matsuyama, T., Aoki, A., Oda, S. et al. (2003). Effects of the Er:YAG laser irradiation on titanium implant materials and contaminated implant abutment surfaces. *Journal of Clinical Laser Medicine & Surgery* 21 (1): 7–17.
- **59** Park, J.H., Heo, S.J., Koak, J.Y. et al. (2012). Effects of laser irradiation on machined and anodized titanium disks. *The International Journal of Oral & Maxillofacial Implants* 27 (2): 265–272.
- **60** Schwarz, F., Aoki, A., Sculean, A., and Becker, J. (2009). The impact of laser application on periodontal and peri-implant wound healing. *Periodontology 2000* 51: 79–108.
- **61** Schwarz, F., Rothamel, D., Sculean, A. et al. (2003). Effects of an Er:YAG laser and the Vector ultrasonic system on the biocompatibility of titanium implants in

cultures of human osteoblast-like cells. *Clinical Oral Implants Research* 14 (6): 784–792.

- **62** Smeo, K., Nasher, R., and Gutknecht, N. (2018). Antibacterial effect of Er,Cr:YSGG laser in the treatment of peri-implantitis and their effect on implant surfaces: a literature review. *Lasers in Dental Science* 2: 63–71.
- **63** Stübinger, S., Etter, C., Miskiewicz, M. et al. (2010). Surface alterations of polished and sandblasted and acid-etched titanium implants after Er:YAG, carbon dioxide, and diode laser irradiation. *The International Journal of Oral & Maxillofacial Implants* 25 (1): 104–111.
- **64** Taniguchi, Y., Aoki, A., Mizutani, K. et al. (2013). Optimal Er:YAG laser irradiation parameters for debridement of microstructured fixture surfaces of titanium dental implants. *Lasers in Medical Science* 28 (4): 1057–1068.
- **65** Kreisler, M., Al Haj, H., and d'Hoedt, B. (2002). Temperature changes at the implant-bone interface during simulated surface decontamination with an Er:YAG laser. *The International Journal of Prosthodontics* 15 (6): 582–587.
- **66** Romanos, G., Crespi, R., Barone, A., and Covani, U. (2006). Osteoblast attachment on titanium disks after laser irradiation. *The International Journal of Oral & Maxillofacial Implants* 21 (2): 232–236.
- **67** Schwarz, F., Nuesry, E., Bieling, K. et al. (2006). Influence of an erbium, chromium-doped yttrium, scandium, gallium, and garnet (Er,Cr:YSGG) laser on the reestablishment of the biocompatibility of contaminated titanium implant surfaces. *Journal of Periodontology* 77 (11): 1820–1827.
- **68** Eick, S., Meier, I., Spoerle, F. et al. (2017). In vitro-activity of Er:YAG laser in comparison with other treatment modalities on biofilm ablation from implant and tooth surfaces. *PLoS One* 12 (1): e0171086.
- **69** Gholami, G.A., Karamlou, M., Fekrazad, R. et al. (2018). Comparison of the effects of Er, Cr: YSGG laser and super-saturated citric acid on the debridement of contaminated implant surfaces. *Journal of Lasers in Medical Sciences* 9 (4): 254–260.
- **70** Miller, R.J. (2004). Treatment of the contaminated implant surface using the Er,Cr:YSGG laser. *Implant Dentistry* 13 (2): 165–170.
- **71** Hakki, S.S., Tatar, G., Dundar, N., and Demiralp, B. (2017). The effect of different cleaning methods on the surface and temperature of failed titanium implants: an in vitro study. *Lasers in Medical Science* 32 (3): 563–571.
- **72** Schwarz, F., Sculean, A., Rothamel, D. et al. (2005). Clinical evaluation of an Er:YAG laser for nonsurgical treatment of peri-implantitis: a pilot study. *Clinical Oral Implants Research* 16 (1): 44–52.
- **73** Takagi, T., Aoki, A., Ichinose, S. et al. (2018). Effective removal of calcified deposits on microstructured titanium

fixture surfaces of dental implants with erbium lasers. *Journal of Periodontology* 89 (6): 680–690.

- **74** Kreisler, M., Kohnen, W., Marinello, C. et al. (2002). Bactericidal effect of the Er:YAG laser on dental implant surfaces: an in vitro study. *Journal of Periodontology* 73 (11): 1292–1298.
- **75** Friedmann, A., Antic, L., Bernimoulin, J.P., and Purucker, P. (2006). In vitro attachment of osteoblasts on contaminated rough titanium surfaces treated by Er:YAG laser. *Journal of Biomedical Materials Research. Part A* 79 (1): 53–60.
- **76** Kreisler, M., Kohnen, W., Christoffers, A.B. et al. (2005). In vitro evaluation of the biocompatibility of contaminated implant surfaces treated with an Er: YAG laser and an air powder system. *Clinical Oral Implants Research* 16 (1): 36–43.
- **77** Park, S.H., Kim, O.J., Chung, H.J., and Kim, O.S. (2020). Effect of a Er, Cr:YSGG laser and a Er:YAG laser treatment on oral biofilm-contaminated titanium. *Journal of Applied Oral Science* 28: e20200528.
- **78** Shin, S.I., Min, H.K., Park, B.H. et al. (2011). The effect of Er:YAG laser irradiation on the scanning electron microscopic structure and surface roughness of various implant surfaces: an in vitro study. *Lasers in Medical Science* 26 (6): 767–776.
- **79** Yamamoto, A. and Tanabe, T. (2013). Treatment of peri-implantitis around TiUnite-surface implants using Er:YAG laser microexplosions. *International Journal of Periodontics & Restorative Dentistry* 33 (1): 21–30.
- **80** Nevins, M., Nevins, M.L., Yamamoto, A. et al. (2014). Use of Er:YAG laser to decontaminate infected dental implant surface in preparation for reestablishment of bone-toimplant contact. *International Journal of Periodontics & Restorative Dentistry* 34 (4): 461–466.
- **81** Sculean, A., Schwarz, F., and Becker, J. (2005). Anti-infective therapy with an Er:YAG laser: influence on peri-implant healing. *Expert Review of Medical Devices* 2 (3): 267–276.
- **82** Ishikawa, I., Aoki, A., Takasaki, A.A. et al. (2009). Application of lasers in periodontics: true innovation or myth? *Periodontology 2000* 50: 90–126.
- **83** Reyhanian, A. and Coluzzi, D.J. (2008). Peri-implantitis therapy with an Er:YAG laser. *Journal of Laser Dentisry* 16 (2): 69–74.
- **84** Schwarz, F., Jepsen, S., Herten, M. et al. (2006). Influence of different treatment approaches on non-submerged and submerged healing of ligature induced peri-implantitis lesions: an experimental study in dogs. *Journal of Clinical Periodontology* 33 (8): 584–595.
- **85** Takasaki, A.A., Aoki, A., Mizutani, K. et al. (2007). Er:YAG laser therapy for peri-implant infection: a histological study. *Lasers in Medical Science* 22 (3): 143–157.
- **86** Mizutani, K., Aoki, A., Coluzzi, D. et al. (2016). Lasers in minimally invasive periodontal and peri-implant therapy. *Periodontology 2000* 71 (1): 185–212.
- **87** Schwarz, F. and Becker, J. (2005). Treatment of periodontitis and peri-implantitis with an Er:YAG laser: experimental and clinical studies. *Medical Laser Application* 20: 47–59.
- **88** Schwarz, F., Bieling, K., Nuesry, E. et al. (2006). Clinical and histological healing pattern of peri-implantitis lesions following non-surgical treatment with an Er:YAG laser. *Lasers in Surgery and Medicine* 38 (7): 663–671.
- **89** Schwarz, F., Bieling, K., Bonsmann, M. et al. (2006). Nonsurgical treatment of moderate and advanced periimplantitis lesions: a controlled clinical study. *Clinical Oral Investigations* 10 (4): 279–288.
- **90** Persson, G.R., Roos-Jansaker, A.M., Lindahl, C., and Renvert, S. (2011). Microbiologic results after nonsurgical erbium-doped: yttrium, aluminum, and garnet laser or air-abrasive treatment of peri-implantitis: a randomized clinical trial. *Journal of Periodontology* 82 (9): 1267–1278.
- **91** Muthukuru, M., Zainvi, A., Esplugues, E.O., and Flemmig, T.F. (2012). Non-surgical therapy for the management of peri-implantitis: a systematic review. *Clinical Oral Implants Research* 23 (Suppl 6): 77–83.
- **92** Yan, M., Liu, M., Wang, M. et al. (2015). The effects of Er:YAG on the treatment of peri-implantitis: a metaanalysis of randomized controlled trials. *Lasers in Medical Science* 30 (7): 1843–1853.
- **93** Kotsakis, G.A., Konstantinidis, I., Karoussis, I.K. et al. (2014). Systematic review and meta-analysis of the effect of various laser wavelengths in the treatment of peri-implantitis. *Journal of Periodontology* 85 (9): 1203–1213.
- **94** Mailoa, J., Lin, G.H., Chan, H.L. et al. (2014). Clinical outcomes of using lasers for peri-implantitis surface detoxification: a systematic review and meta-analysis. *Journal of Periodontology* 85 (9): 1194–1202.
- **95** Al-Falaki, R., Cronshaw, M., and Hughes, F.J. (2014). Treatment outcome following use of the erbium, chromium: yttrium, scandium, gallium, garnet laser in the non-surgical management of peri-implantitis: a case series. *British Dental Journal* 217 (8): 453–457.
- **96** Schwarz, F., Sculean, A., Romanos, G. et al. (2005). Influence of different treatment approaches on the removal of early plaque biofilms and the viability of SAOS2 osteoblasts grown on titanium implants. *Clinical Oral Investigations* 9 (2): 111–117.
- **97** Lin, T., Taniguchi, Y., Aoki, A., and Chang, Y.C. (2019). Clinical evaluation of multiple peri-implant bony defect management by Er:YAG laser-assisted bone regenerative therapy. *Journal of Dental Sciences* 14 (4): 430–432.
- **98** Azzeh, M.M. (2008). Er,Cr:YSGG laser-assisted surgical treatment of peri-implantitis with 1-year reentry and 18-month follow-up. *Journal of Periodontology* 79 (10): 2000–2005.
- **99** Yoshino, T., Yamamoto, A., and Ono, Y. (2015). Innovative regeneration technology to solve periimplantitis by Er:YAG laser based on the microbiologic diagnosis: a case series. *International Journal of Periodontics & Restorative Dentistry* 35 (1): 67–73.
- **100** Schwarz, F., Hegewald, A., John, G. et al. (2013). Four-year follow-up of combined surgical therapy of advanced peri-implantitis evaluating two methods of surface decontamination. *Journal of Clinical Periodontology* 40 (10): 962–967.
- **101** Schwarz, F., John, G., Mainusch, S. et al. (2012). Combined surgical therapy of peri-implantitis evaluating two methods of surface debridement and decontamination. A two-year clinical follow up report. *Journal of Clinical Periodontology* 39 (8): 789–797.
- **102** Schwarz, F., Sahm, N., Iglhaut, G., and Becker, J. (2011). Impact of the method of surface debridement and decontamination on the clinical outcome following combined surgical therapy of peri-implantitis: a randomized controlled clinical study. *Journal of Clinical Periodontology* 38 (3): 276–284.
- **103** Norton, M.R. (2017). Efficacy of Er:YAG laser in the decontamination of peri-implant disease: a one-year prospective closed cohort study. *International Journal of Periodontics & Restorative Dentistry* 37 (6): 781–788.
- **104** Wang, C.W., Ashnagar, S., Gianfilippo, R.D. et al. (2021). Laser-assisted regenerative surgical therapy for periimplantitis: a randomized controlled clinical trial. *Journal of Periodontology* 92 (3): 378–388.
- **105** Lin, G.H., Suarez Lopez Del Amo, F., and Wang, H.L. (2018). Laser therapy for treatment of peri-implant mucositis and peri-implantitis: an American Academy of Periodontology best evidence review. *Journal of Periodontology* 89 (7): 766–782.
- **106** Mills, M.P., Rosen, P.S., Chambrone, L. et al. (2018). American Academy of Periodontology best evidence consensus statement on the efficacy of laser therapy used alone or as an adjunct to non-surgical and surgical treatment of periodontitis and peri-implant diseases. *Journal of Periodontology* 89 (7): 737–742.
- **107** Tuncer, I., Tomruk, C.Ö., Sencift, K., and Cöloğlu, S. (2009). Comparison of conventional surgery and CO2 laser on intraoral soft tissue pathologies and evaluation of the collateral thermal damage. *Photomedicine and Laser Surgery* 28: 75–79.
- **108** Rossmann, J.A., McQuade, M.J., and Turunen, D.E. (1992). Retardation of epithelial migration in monkeys using a carbon dioxide laser: an animal study. *Journal of Periodontology* 63 (11): 902–907.

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- **109** Melcer, J., Chaumette, M.T., and Melcer, F. (1987). Dental pulp exposed to the CO2 laser beam. *Lasers in Surgery and Medicine* 7 (4): 347–352.
- **110** Romanos, G.E. (1999). Laseranwendung bei der Peri-implantitistherapie. Ein Fallbericht. *ZMK* 15: 818–819. (German).
- **111** Deppe, H., Horch, H.H., Henke, J., and Donath, K. (2001). Peri-implant care of ailing implants with the carbon dioxide laser. *The International Journal of Oral & Maxillofacial Implants* 16 (5): 659–667.
- **112** Kato, T., Kusakari, H., and Hoshino, E. (1998). Bactericidal efficacy of carbon dioxide laser against bacteria-contaminated titanium implant and subsequent cellular adhesion to irradiated area. *Lasers in Surgery and Medicine* 23 (5): 299–309.
- **113** Romanos, G.E. (2002). Treatment of peri-implant lesions using different laser systems. *Journal of Oral Laser Applications* 2: 75–81.
- **114** Romanos, G.E. and Nentwig, G.H. (2008). Regenerative therapy of deep peri-implant infrabony defects after CO2 laser implant surface decontamination. *International Journal of Periodontics & Restorative Dentistry* 28 (3): 245–255.
- **115** Romanos, G., Ko, H.H., Froum, S., and Tarnow, D. (2009). The use of CO(2) laser in the treatment of peri-implantitis. *Photomedicine and Laser Surgery* 27 (3): 381–386.
- **116** Vohra, F., Al-Rifaiy, M.Q., Lillywhite, G. et al. (2014). Efficacy of mechanical debridement with adjunct antimicrobial photodynamic therapy for the management of peri-implant diseases: a systematic review. *Photochemical & Photobiological Sciences* 13 (8): 1160–1168.
- **117** Sperandio, F., Huang, Y.-Y., and Hamblin, M.R. (2013). Antimicrobial photodynamic therapy to kill Gramnegative bacteria. *Recent Patents on Anti-Infective Drug Discovery* 8 (2): 108–120.
- **118** O'Connor, A.E., Gallagher, W.M., and Byrne, A.T. (2009). Porphyrin and nonporphyrin photosensitizers in oncology: preclinical and clinical advances in photodynamic therapy. *Photochemistry and Photobiology* 85 (5): 1053–1074.
- **119** Pérez-Chaparro, P.J., Duarte, P.M., Shibli, J.A. et al. (2016). The current weight of evidence of the microbiologic profile associated with peri-implantitis: a systematic review. *Journal of Periodontology* 87 (11): 1295–1304.
- **120** Shibli, J.A., Melo, L., Ferrari, D.S. et al. (2008). Composition of supra-and subgingival biofilm of subjects with healthy and diseased implants. *Clinical Oral Implants Research* 19 (10): 975–982.
- **121** Takasaki, A.A., Aoki, A., Mizutani, K. et al. (2009). Application of antimicrobial photodynamic therapy in

periodontal and peri-implant diseases. *Periodontology 2000* 51 (1): 109–140.

- **122** Deppe, H., Mücke, T., Wagenpfeil, S. et al. (2013). Nonsurgical antimicrobial photodynamic therapy in moderate vs severe peri-implant defects: a clinical pilot study. *Quintessence International* 44 (8): 609–618.
- **123** Socransky, S., Haffajee, A., Cugini, M. et al. (1998). Microbial complexes in subgingival plaque. *Journal of Clinical Periodontology* 25 (2): 134–144.
- **124** Dörtbudak, O., Haas, R., Bernhart, T., and Mailath-Pokorny, G. (2001). Lethal photosensitization for decontamination of implant surfaces in the treatment of peri-implantitis. *Clinical Oral Implants Research* 12 (2): 104–108.
- **125** Tang, W., Xu, H., Park, E.J. et al. (2008). Encapsulation of methylene blue in polyacrylamide nanoparticle platforms protects its photodynamic effectiveness. *Biochemical and Biophysical Research Communications* 369 (2): 579–583.
- **126** Cieplik, F., Späth, A., Regensburger, J. et al. (2013). Photodynamic biofilm inactivation by SAPYR – an exclusive singlet oxygen photosensitizer. *Free Radical Biology & Medicine* 65: 477–487.
- **127** Voos, A.C., Kranz, S., Tonndorf-Martini, S. et al. (2014). Photodynamic antimicrobial effect of safranine O on an ex vivo periodontal biofilm. *Lasers in Surgery and Medicine* 46 (3): 235–243.
- **128** Al Amri, M.D., Kellesarian, S.V., Ahmed, A. et al. (2016). Efficacy of periimplant mechanical debridement with and without adjunct antimicrobial photodynamic therapy in patients with type 2 diabetes mellitus. *Photodiagnosis and Photodynamic Therapy* 14: 166–169.
- **129** Javed, F., BinShabaib, M.S., Alharthi, S.S., and Qadri, T. (2017). Role of mechanical curettage with and without adjunct antimicrobial photodynamic therapy in the treatment of peri-implant mucositis in cigarette smokers: a randomized controlled clinical trial. *Photodiagnosis and Photodynamic Therapy* 18: 331–334.
- **130** Chambrone, L., Wang, H.L., and Romanos, G.E. (2018). Antimicrobial photodynamic therapy for the treatment of periodontitis and peri-implantitis: an American Academy of Periodontology best evidence review. *Journal of Periodontology* 89 (7): 783–803.
- **131** Bombeccari, G.P., Guzzi, G., Gualini, F. et al. (2013). Photodynamic therapy to treat periimplantitis. *Implant Dentistry* 22 (6): 631–638.
- **132** Giannini, R., Vassalli, M., Chellini, F. et al. (2006). Neodymium:yttrium aluminum garnet laser irradiation with low pulse energy: a potential tool for the treatment of peri-implant disease. *Clinical Oral Implants Research* 17 (6): 638–643.

Implant Surface Modification as a Method of Implant Maintenance Applications, Limitations, and Risks of Implantoplasty

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Introduction

Peri-implant diseases are defined as any kind of inflammatory reaction in the tissues surrounding the implants' [1], whereas peri-implantitis was introduced as an inflammatory process on hard and soft tissue resulting in pathological pocket formation and loss of supporting bone [2].

The wide range in prevalences of peri-implantitis varying between 2.7 and 47.1% of implants $[1, 3, 4]$ can be attributed to the missing of exact diagnostic parameters, differences in study population, disease dentition, and implant micro-and macrostructures. An effective strategy for treating this disease is required, as otherwise a debilitating condition around the affected implants will result in risk of further loss of function and esthetics.

The development of an adherent biofilm on the implant surface plays an important role in the multifactorial etiology of peri-implantitis [2]. One key factor in the initiation and progress of peri-implant diseases is the presence of a bacterial biofilm; therefore, the elimination of the established biofilm from the implant surface and prevention of adhesion of a new biofilm are the main objectives in the treatment of peri-implant mucositis and peri-implantitis [5]. Thus, incomplete decontamination of the implant surface could be the biggest obstacle in maintaining the remaining peri-implant bone [6].

When referring to peri-implant inflammation, peri implantitis is an irreversible progressive inflammation of the surrounding bone [7]. For the classification of periimplantitis and corresponding peri-implant bone lesions, a classification of different defect morphologies – especially focused on the various therapeutic options – is recommended. If there is a combination of bleeding/suppuration with an increasing probing depth compared to previous

examinations, or probing depths of ≥ 6 mm and radiological detectable bone resorption beyond the initial bone level after implant placement, this is referred to as peri implantitis [8]. In the absence of initial radiological findings, bone resorption ≥3mm apical to the intrabony part of the implant is considered indicative of peri-implantitis [9]. Furthermore, intrabony defects (Class I) are distinguished from horizontal supracrestal defects (Class II). The supracrestal portion is defined as the distance between the transition from the smooth to the machined implant portion and the peri-implant crestal alveolar bone [10]. Intrabony defects can be divided into purely vestibular or oral dehiscence defects (Class Ia), vestibular or oral dehiscence defects with additional semicircular portions (Class Ib), vestibular or oral dehiscence defects with additional circular bone resorption (class Ic), and circular bone resorption with vestibular and oral dehiscence defects (class Id) or with bilaterally preserved compact bone (class Ie). Horizontal and intrabony defects mostly occur together. According to current data, 55.3% of peri-implant bone defects are a combination of Class Ie and Class II [11].

Patient age, gender, gene polymorphisms, cardiovascular disease, rheumatoid disease, osteoporosis, condition of residual dentition, implant design, and surface as well as implant site and type of restoration are possible risk factors for peri-implant disease. Several clinical protocols for treatment of peri-implantitis have been suggested, including mechanical debridement, the use of antiseptics and local or systemic antibiotics $[12, 13]$, surgical access $[14–16]$, and regenerative [17–20] or resective surgical procedures [21–24]. Yet, re-osseointegration as desired goal in implant dentistry has not been achieved after treatment of infected treated implants in long term clinical trials until current days [25].

Among all clinical approaches to treat peri-implantitis, the non-surgical treatment involving mechanical debridement with local or systemic antimicrobials as adjuncts has not been effective to resolve peri-implantitis since it cannot provide an adequate access to remove infected tissues around affected implants, especially in advanced lesions [26]. Yet, in a randomized clinical trial mechanical therapy reduced bleeding and plaque accumulation indexes after a six-month follow up [27]. Hence, the non-surgical approach as a preparatory phase has been recommended before any surgical intervention to treat PI since it can reduce inflammation and biofilm accumulation improving the preoperative conditions of the surgical area [28]. Compared to the non-surgical approach, peri-implantitis surgical intervention has been claimed as a more effective therapy since it provides an adequate access to decontaminate the infected implant. Removing biofilm accumulation and granulation tissue around implants can reduce the risk of further disease progression since the local etiological factors are eliminated [29].

Different surgical approaches to treat peri-implantitis have been evaluated including resective therapy, implantoplasty, bone recontouring, and regenerative techniques with adjuncts such as bone filler particles and membranes [5]. All these techniques depend especially on the configuration of the peri-implant bone defect and the treated region and aim to either fill up bony defects by so-called regenerative approaches or to provide effective access to osseodisintegrated areas for adequate daily oral hygiene by resective measures [30]. Regeneration, however, is not appropriate in many cases either due to an unfavorable defect morphology or due to patient-related factors like continuous heavy smoking or incompliance regarding oral hygiene, leaving the resective intervention as a more reliable alternative [23, 24]. For an effective resective, peri-implantitis therapy comprises a discreet osteoplasty of the marginal peri-implant bone, an implant surface modification called implantoplasty through the removal of exposed threads, and antibacterial decontamination of the implant surface are key factors. Using resective techniques the main aim is to create a predominantly horizontal defect and therefore allow for better access during intervention and – importantly – during maintenance later on. Implant surface roughness and the size of initial peri-implant lesion seem to have an influence on the progression rate of peri-implantitis [31]. Studies have shown that implant and abutment surface roughness significantly influence the type and quantity of microbiota supra- and subgingivally [32]. Therefore, the commonly accepted rationale of implantoplasty consists of mechanical removal of implant threads (or any rough implant surface) using sequential drills [33], thus yielding a smooth-finished implant structure that will reduce biofilm accumulation [34]. This has been found to influence the outcome of

peri-implantitis treatment in terms of extent and/or severity of residual peri-implant inflammation [35]. Successful clinical and radiographic outcomes of peri-implantitis treatment have been reported in literature when a resective procedure was combined with implantoplasty [23, 24, 26].

However, none of them has been proven superior to the others concerning peri-implantitis resolution in long term. The combined resective and implantoplasty approach has shown favorable results in some studies [20, 23, 24, 29, 36]. The mechanical removal (grinding) of the implant threads and the rough implant surface, rendering thus a relatively "smooth" implant surface can be performed either at the parts of the implant, where due to defect anatomy only a limited potential for bone regeneration and/or re osseointegration after healing is expected, e.g. the supra bony and dehiscence-aspects of the implant or on the whole exposed rough implant surface [37].

Both implantoplasty approaches aim to reduce biofilm accumulation by smoothening the rough exposed implant surface, since numerous studies have shown that a rough surface can protect biofilm from shear forces carried out by mechanical debridement [38, 39], thus allowing biofilm growth and persistence as a local factor for progession of peri-implantitis.1 Regarding that, the smoothening and decontamination of the exposed implant surface will provide a more favorable area for soft tissue adaptation during the healing process. In vitro studies have also reported that a smooth surface enhances fibroblast adherence and consequently can help to get peri-implant soft tissue health [36].

Unpleasantly, implantoplasty may cause perforation of the implant body, destruction of the implant-abutment connection, overheating of the implant during grinding causing thermal damage to the surrounding bone, or induction of mucosal staining and/or increased risk for late inflammatory reactions due to titanium particle deposition [40], generated from the grinding procedure. Furthermore, reduction of the implant mass (implant diameter) at its coronal aspect, occasionally also involving the implant collar [41, 42], may compromise implant strength and lead to an increased rate of late mechanical complications, like for example implant collar deformation, loosening of the supra-structure, fixation screw fracture and implant fracture. Altogether this may lead to recurrent peri-implant biological complications and/ or require explantation.

Treatment

Patient-specific treatment of peri-implant inflammation is a synoptic treatment concept with attention to individual risk factors in order to prevent the development or renewed progression of peri-implant infections and anti-inflammatory, if possible reconstructive treatment of the peri-implant lesion. The aimed success of implant treatment is only possible, if in the long-term biological, technical, and esthetic complications can be avoided. Biologically, the absence of peri-implant mucositis, peri-implantitis, and the establishment of stable soft tissue conditions is necessary, especially during maintenance therapy after active treatment of peri implant infections. Biological complications at implants differ in frequency and severity in patients with and without periodontitis. Thus, the implementation of a careful anti-infective periodontal therapy with reduction of inflammatory signs and probing depth values before treatment of implantoplasty is mandatory.

For a successful long-term treatment of peri-implant inflammation, particularly from the patient point of view, it is essential to design the prosthetic restoration as similar as possible to the natural appearance of the teeth with corresponding good oral hygiene characteristics to satisfy the patient optically and functionally; often, this can only be achieved by restoring the lost tissue dimensions.

The extent to which the crown-to-implant length ratio has an influence on the survival, on the marginal bone level, or on prosthetic complications in the absence of augmentation is controversially discussed. Some reviews concluded that no negative influences exist [43, 44]. In contrast, other systematic reviews observed a higher incidence of prosthetic complications such as abutment loosening or fractures, mainly in posterior jaw regions. Restoration of near-original dimensions of hard and soft tissues can minimize these risks in the long term [45]. Moreover, the esthetic result is significantly improved, and the ability to maintain oral hygiene, thus ensuring the prevention of inflammatory processes [46].

If peri-implantitis develops despite consideration of these recommendations, the causal therapy of existing risk

factors needs to start with the utmost priority; this includes smoking cessation, control of diabetes mellitus, and specific oral hygiene instructions. Localized plaque-induced inflammation should be eliminated by non-surgical mechanical plaque removal, optimization of oral hygiene skills, and inclusion in a regular maintenance therapy program [47]. To enable an efficient plaque removal is the primary goal [48]. Home-based oral hygiene can be carried out using manual or electric toothbrushes and appropriate interdental brushes [49].

Peri-implantitis with a maximum of 75% mean bone loss can more often result in implant loss depending on implant position, prosthetic importance, inflammation, oral hygiene access, and implant type. An implant with mean bone loss of 25–50% is 8.86 times more likely to fail than an implant with mean bone loss <25%. Moreover, 1 mm of additional mean bone loss increases the risk of failure by 74%. The removal of the affected implant is usually indicated after clinical and radiological diagnosis (Figures 14.1–14.8), as well as very low Resonance Frequency Analysis (RFA) values or very high Damping Capacity Analysis (DCA) values, deep tapping sounds, mobility, and large probing values as indicators for insufficient osseointegration [47]. In all other cases, the peri implant inflammation must permanently be brought back to a stagnation phase, beginning with a non-surgical treatment phase and the adjustment of all oral hygiene parameters.

The systematic and continuous prevention and treatment of peri-implant diseases is based on the original CIST concept (cumulative interceptive supportive therapy or antiseptic cumulative supportive therapy) according to Mombelli and Lang [50, 51]. The CIST concept is a step-by step model divided into four treatment steps. Depending on the diagnostic course, the modular therapy guide

Figure 14.1 Panoramic radiograph: peri-implantitis with severe bone loss.

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Figure 14.2 Baseline situation with removal of the bar. **Figure 14.5** Free gingival graft harvested from the palate.

Figure 14.3 Implant removal of posterior implants and removal of granulation tissue.

Figure 14.6 Clinical situation two weeks post-operatively.

Figure 14.4 Implantoplasty at anterior two implant with the saving bar-retained restauration.

initially includes hygiene instructions and professional dental cleanings(part A), followed by chlorhexidine rinses, gel applications (part B) and systemic antibiotic medication (part C), and finally subsequent surgical interventions

Figure 14.7 Clinical situation four years post-operatively.

with either resective or regenerative treatment approaches (part D) (Figures 14.9–14.15). However, especially in the further development of patient-specific treatments, the existing risk factors must be recognized and adjusted, and the evaluation of each treatment step must not be made

Figure 14.8 Post-operative radiograph four years after treatment.

according to rigid consideration of probing values, but according to the change in probing values over time [52, 53].

Non-surgical treatment of peri-implantitis can be expected to reduce bleeding on probing, but results in a limited improvement in probing values [54]. When adjuvant

Figure 14.9 Baseline situation: peri-implantitis regio #3.

irrigation solutions or antibiotics were used, such as minocycline products and tetracycline derivatives, the effectiveness was shown by improved bleeding on probing values and reduced probing depths [13, 55–57] (Figure 14.11). However, the administration of systemic antibiotics should be avoided for non-surgical procedures [14, 58]. The adjuvant use of Nd:YAG and Er:YAG lasers in addition to mechanical therapy resulted in only short-term success, which lasted a few months in terms of bleeding on probing and probing depths reduction [59, 60].

Six weeks after the non-surgical procedure, a surgical, mechanical debridement including chemical de-contamination of the implant surface should be performed. Access flaps, resective therapy approaches with or without implantoplasty, or augmentative procedures can be used in this operative intervention. The decisive factors in surgical treatment planning in this context are bony defect morphology and position of the affected implant – either inside or outside the esthetic area (Figures 14.12–14.14). In principle, augmentative measures for intrabony components such as bowl-shaped defects (class Ie [61]) and three- or

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Figure 14.14 Apical reposition flap.

Figure 14.11 Non-surgical debridement and decontamination with application of local antibiotics.

Figure 14.12 Open flap debridement six weeks after non-surgical approach.

Figure 14.13 Implantoplasty with antimicrobial irrigation and laser application.

Figure 14.15 Clinical situation five years after treatment without bleeding on probing.

four-walled bone defects can achieve improved clinical, radiological therapeutic, and anti-inflammatory results. The remaining bony defect morphologies are usually treated with resective procedures optionally combined with implant surface modification.

Indications

Surgical access flaps and resective treatment approaches are indicated for supracrestal bone defects (horizontal bone resorption) with exposed implant threads [62, 63]. Resective treatment of peri-implant inflammation can recontour the bone and reduce probing values. This can be performed with or without smoothening of the implant surface. Peri-implant bone defect morphology can limit the angulation of the bur and, thus, affect the outcome of implantoplasty. Concerns have also been raised regarding biological and mechanical complications associated with implantoplasty [64].

In the esthetic region, an access flap with a strictly intrasulcular incision preserving the soft tissue is preferred, whereas in the posterior region, an apically repositioned flap can be used [62]. In esthetic regions with moderate bone loss and shallow bone defects, the combination of surgical debridement with a free connective tissue graft is a recommended option to achieve significant clinical improvement while avoiding the high risk of recession (Figures 14.16–14.21) [15, 20]. Especially in posterior areas, resective treatment with implantoplasty leads to improved clinical and radiological results after a three-year follow-up compared to the resective approach without implantoplasty (STM: 1.64 ± 1.29 versus 2.3 ± 1.45 mm) [23, 24].

Figure 14.19 Connective tissue graft harvested from the palate.

Figure 14.16 Periimplantitis in the esthetic region.

Figure 14.20 Wound closure.

Figure 14.17 Open flap debridement with granulation tissue removal.

Figure 14.18 Implantoplasty and antimicriobial irrigiation.

Figure 14.21 Clinical situation without bleeding and probing 3 years post-operatively.

Surgical Procedure

The aims of resective surgery are reducing pocket probing depths and gaining a soft tissue morphology that enhances good self-performed oral hygiene and peri-implant health. The therapeutical approach of peri-implantitis with suprabony defects surrounding rough implants comprises several aspects [65]: (i) removal of supragingival bacterial plaque, (ii) surgical approach with access flap, (iii) removal of granulation tissue and detoxification of the exposed implant surface, (iv) correction of the anatomical architecture of the bone, (v) modification of the roughness of implant surface, (vi) establishment of an efficient plaque control regime (Figures 14.22–14.28) [48, 66].

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Figure 14.22 Peri-implantitis at mal-positioned implants.

Figure 14.23 Radiographic situation with mostly horizontal bone loss.

Figure 14.24 Removal of suprastructure and suppuration.

Implantoplasty can be performed at the surgical peri implantitis treatment. Implantoplasty is done on the exposed implant threads by polishing and smoothing implant surface in order to reduce bacterial recolonization [34, 67]. Removal of the threads and surface polishing

Figure 14.25 Implantoplasty and minimal-invasive resective osseous treatment.

Figure 14.26 Laser application and wound closure.

Figure 14.27 Clinical situation one year post-operatively.

can generally be performed using different sequences of instruments. While Chan et al. [68] and Ramel et al. [33] described the use of diamond burs followed by an Arkansas stone, Sahrmann et al. [69] suggested the use of a rough carborundum stone followed by an Arkansas stone and – like Chan et al. [68] – silicon polishers. Beyond that,

Figure 14.28 Clinical situation five years post-operatively. **Figure 14.29** Baseline situation with suppuration.

several other sequences of instruments for implantoplasty have been proposed [23, 70]. They vary in different ways that might potentially influence the mechanical stability of the instrumented implant [71, 72]. Firstly, the invasiveness or quickness of abrasion that depends on the rotational speed and the coarseness of the instrument is likely to have an effect: the quicker the abrasion the less material loss will be controllable. Secondly, instruments with unchangeable morphology like diamonds might not perfectly adapt to the implant surface while abrasive stones can be trimmed in order to fit perfectly. However, diamonds might have a smaller diameter and therefore might be less prone to injure neighboring teeth, which should be considered as a potential damage during instrumentation [69].

The so-called "modified" implantoplasty technique [26] forming a bottleneck format beginning from the most apical part of the exposed implant body is commonly performed following a sequence of steps and burs (Figures 14.29–14.33). First, a bud-shaped diamond bur (grit size 106μm) at 120,000rpm is used to smoothen the exposed threads and to perform a "platform switch" area in the exposed apical part of the implant. In this step, the smoothing procedure is mainly restricted to the threads and does, obviously, not affect the core diameter of the implants. A waist shape, narrowing the medial region of the exposed implant body is achieved by using football-shaped fine (grit size 46μm) and extra-fine (grit size $25 \mu m$). Afterward, the rough surface is polished at 40,000rpm with flame-shaped fine (grit size $46 \mu m$) and extra fine (grit size $25 \mu m$) diamond burs (8379) 314.021; 379EF 314.023, Gebr. Brasseler GmbH & Co. KG, Lemgo, Germany) [73] (Figure 14.34). Flame or ellipseshaped carbide burs (30mm length) can be used with normal (12 cutting edges) and ultra-fine (30 cutting edges) finishing grades (Figure 14.35). Damage of the neighboring teeth, however, is a rather frequent finding with a prevalence of one-third of all cases. That is why football-shaped burs should only be used carefully in situations with good

Figure 14.30 Removal of the suprastructure and application of healing cap.

Figure 14.31 Bone defect.

accessibility [74]. The smoothening of the surface is finalized with Arkansas stone torpedo-or round-shaped white aluminum oxide burs at 40,000rpm (601.314, extra fine, grit 420; Gebr. Brasseler GmbH & Co. KG) (Figure 14.36) [75]. The final polishing of the implant's waist-like surface is

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Figure 14.32 Implantoplasty achieving a platform switch area.

Figure 14.33 Wound closure and insertion of suprastructure.

Figure 14.34 Flame or ellipse-shaped fine and extra fine diamond burs.

done using a mini-point-shaped abrasive-impregnated silicone polisher (Greenie) until the smoothened areas look evenly machined macroscopically (Figure 14.36). The complete burs sequence is performed under copious irrigation with sterile saline. Using this technique, the median

Figure 14.35 Flame or ellipse-shaped carbide burs (normal and ultra-fine).

Figure 14.36 Torpedo-shaped Arkansas stone or round-shaped white aluminum oxide burs and abrasive silicone polisher (Greenie).

implant diameters in tissue-level or bone-level implants are normally reduced by about 0.10–0.20mm. The surface modification with a sequence of burs leads to smoother surfaces compared to diamond sonic tips. Followed by Arkansas stones, equivalent final surface roughness could be detected in both implantoplasty procedures. In cases of narrow diameter implants, it could be helpful to use sonic devices as a more conservative method with less structure loss [64]. Bone recontouring can be performed with a spherical bur under copious irrigation (Figures 14.37–14.46).

Implantoplasty commonly results in a smoothing of the sharp-edged microstructure. However, it is also associated with the presence of irregular grooves and flat pits, which appear to be more uneven than the structure noted for machined surfaces. In particular, implantoplasty-treated and machined surfaces are characterized by a comparable quantity (Wt%) of elements carbon (C) , oxygen (O) , sodium (Na), chloride (Cl), potassium (K), and silicon (Si), but a

Figure 14.37 Baseline situation with peri-implantitis and insufficient attached mucosa.

significantly different quantity of elements nitrogen (N), titanium (Ti) and aluminum (Al) [73]. Finally, the peri implant mucosa is thinned out and repositioned in a more apical direction, leaving the previously smoothened surface accessible for self-conducted oral hygiene by the patient [20].

The remaining titanium particles in the tissue should be reduced by means of gauze exposure and excision of the granulation tissue after implantoplasty or, depending on the indication and diagnosis, implantoplasty should be limited to the supramucosal areas before flap formation, since the effect of tissue reactions to the remaining titanium with regard to progressive peri-implant inflammation is currently unclear [62, 76]. It is recommended to remove the suprastructure before resective intervention, especially in case of implantoplasty; as hereby, the suprastructure can be adapted to as sufficient oral hygiene design before being reinserted [62]. Resective procedures with adjuvant systemic antibiotics did not result in significant clinical and radiological long-term improvement [58].

Figure 14.39 Removal of suprastructure.

Figure 14.40 Open flap debridement and removal of granulation tissue.

Especially, implant position and design as well as the hygienic suitability of the prosthetic reconstruction should be critically evaluated preoperatively [47]. The question of

Figure 14.38 Panoramic radiograph at baseline.

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Figure 14.41 Implantoplasty and antimicrobial irrigation.

Figure 14.44 Free gingival graft harvested from the palate.

Figure 14.42 Clinical situation 12 weeks after implantoplasty treatment and healthy peri-implant conditions.

Figure 14.43 Vestibulumplasty with incision 5mm apart from sulcus.

Figure 14.45 Clinical situation six years after treatment.

open or closed healing [77] and the benefit of adjuvant systemic antibiotics [78] also cannot be clearly answered with the current state of literature. In cases of implant surface modifications and open flap debridement, there is no explicit need for a closed healing and suprastructure can be inserted directly after the surgical procedure [63].

Decontamination

The efficacy of titanium decontamination can be assessed using different protocols under consideration of surface changes in roughness, chemical composition, and wettability on the microimplant surface. Common protocols for decontamination are irrigation with 24% EDTA, 2% chlorhexidine (CHL), gauze soaked in 2% chlorhexidine (GCHL), gauze soaked in ultrapure water (GMQ), irrigation with citric acid (20%) or scaling (SC), use of titanium

Figure 14.46 Radiographic situation six years after treatment.

brushes (TiB) and implantoplasty (IP) [64]. All protocols resulted in changes in roughness, wettability, and chemical composition, yet gauze-soaked chlorhexidine, scaling, titanium brushes, and implantoplasty presented the best decontamination outcomes. Changes in surface roughness were observed after all treatment protocols, but implantoplasty presented the smoothest and least hydrophilic surface [26]. In both initially smooth and rough implants, the lowest values of residual contamination were observed using gauze-soaked chlorhexidine, scaling, titanium brushes, and implantoplasty. There was a statistically significant reduction in hydrophilia after implantoplasty, with no difference if implants were initially smooth or rough. Concurrently, the topographic implant changes are most effective in removing biofilm [34]. Almost no bacterial remnants could be observed after implantoplasty (Figures 14.47–14.52) [79]. In accordance, most effective decontamination protocols do not preserve the elemental integrity of the titanium surface, affecting the interaction of the implant with surrounding tissues [80]. The benefits of a combination of chemical disinfection and protocols with implantoplasty are still controversial [81].

Figure 14.47 Periimplantitis with recessions and bone loss (suprastructure removed).

Figure 14.48 Bone cup defects with buccal fenestrations.

Figure 14.49 Implantoplasty and removal of titanium remnants with saline-sauked gauze.

Wheelis et al. investigated the effects of using various antimicrobial chemical agents for titanium decontamination [82]. As a result, they noted that all protocols invariably left contaminants on their surfaces, which influenced the physical properties. In all experimental groups, a reduction in titanium signal was observed, as well as the

Figure 14.50 Wound closure after laser application.

Figure 14.51 Clinical situation two weeks after treatment.

Figure 14.52 Clinical situation five years after treatment.

deposition of O, C, Al, Na, Cl, and other contaminants. All these contaminants may reduce the surface energy of Ti since the wettability of a surface can be altered by topographic changes or by changesin the chemical components of its surface [83]. This may be a concern due to its potential to compromise cell viability [84]. Besides, it is important to highlight that the selection of a peri implantitis treatment cannot be solely based on its antimicrobial potential, since the effectiveness of decontamination is not the only outcome related to biological success [79, 85].

Overall, implantoplasty-treated implants showed high disease resolution rate and peri-implant marginal bone stability. The surgical antibiofilm strategy can altogether counteract peri-implantitis progression providing an adequate environment for implant function and longevity over a moderate to long-term period [26].

Biofilm Formation

The steps of biofilm development on implant and natural teeth are very similar [86]. Regarding the composition of the biofilm, no significant differences between natural roots and titanium implants could be highlighted [25, 87, 88]. Untreated surfaces showed mature, complex biofilm structures with wide morphological diversity, whereas, implantoplasty treated surfaces did notshow the formation of mature biofilm structures. Implantoplasty is efficient in reducing plaque adhesion and influencing biofilm formation by firstly removing the most external bacterial contaminated layer of titanium while creating an uncontaminated surface and secondly reducing the implant roughness in order to make its surface less retentive for plaque. Five days after implantoplasty, only 16% of implant surfaces (SD = 0) are covered with plaque in contrast to 65% of plaque coverage in the control group without implantoplasty $(SD = 7.07)$ [89].

Equivalent to those findings, in vivo studies show a correlation between plaque adhesion and surface roughness on natural teeth surfaces [32]. Similar correlations are shown on acetate strips [90] and on titanium [32], where rough abutments harbored 25 times more bacteria than standard abutments [91–93]. In accordance, Albouy et al. [94] found that the progression of the peri-implant lesion seems to be influenced by the roughness of the surface possibly predisposed by different biofilm quantity and composition.

On day 1, there are no significant differences between implant surfaces treated with or without implantoplasty regarding plaque accumulation. Starting from day 2, a highly significant difference is found between implantoplasty treated and untreated surfaces. Untreated surfaces of supragingival implant parts show mature and complex biofilm structures with wide morphological diversity expressing active replication and inter-species communication through pili. On surfaces treated with implantoplasty, less mature biofilm structures are seen, showing a prevalence of cocci embedded in the extra-cellular matrix. The biofilm on subgingival parts of implant surfaces biofilms
has a smaller diversity, with mostly bacilli and filamentous microorganisms in both groups, possibly as a consequence of the subgingival-like conditions created by the silicon envelope. Small surface irregularities are used by pioneer bacteria that are almost exclusively cocci. A first layer of pioneer cocci adheres to the surfaces, immediately starts extra-cellular matrix production, and colonizes the smooth surfaces starting from the crestal part of the implant. Biofilm develops as a complex multilayered structure in which mainly bacilli and some hyphae can be observed, while the surface disappears under the mature biofilm structure, especially in spaces between the threads [89]. Mature biofilm structures composed of mixed bacterial species with more than 100 μm of thickness can be observed at some points. In the upper parts of the implant, mature biofilm shows higher ratio of dead to alive microorganisms, which are the layers more exposed to the environment. Dead cells are mostly found on the outer layers of the microcolonies. Implantoplasty does not completely prevent bacterial adherence and biofilm formation, but biofilm structures are much more easily removed by shear forces and mechanical action of oral structures than on conventional implant surfaces [89]. Thus, it can play a major role in preventing plaque accumulation and biofilm formation (Figures 14.53–14.56).

Roughness

A reduction of titanium surface roughness has been correlated positively with reduced plaque colonization up to average threshold roughness of 0.2mm [35, 95]. Implantoplasty modifies implant surface topography. Arithmetical mean roughness values ranging between 0.32 and $1.67 \mu m$ have been reached after implantoplasty, with the lowest value obtained using a sequence of diamond burs and silicone polishers [33, 34, 41].

Figure 14.54 Implantoplasty without osseous resection and creation of a platform switch area.

Figure 14.55 Wound closure.

Figure 14.56 Clinical situation seven years after treatment.

The time for implantoplasty with sonic devices was significantly longer than with burs, with a greater implant weight loss in the latter treatment (−11 and −3%, respectively). Surface roughness values (arithmetical mean [Ra] **Figure 14.53** Baseline situation after removal of suprastructure. and average roughness [Rz]) were higher for ultrasonic

procedures versus using burs and were similar after polishing with Arkansas stones in both groups. SEM analysis showed a faceted surface after implantoplasty with burs, whereas the sonic device left a more homogeneous surface, and differences between the treatments were minimized after use of Arkansas stones, reflecting surface topography findings [64]. Sahrmann et al. [69] compared a sequence of bud-shaped diamond burs followed by Arkansas stones and a sequence of conical silicon carbide stones followed by Arkansas stones and silicone polishers: both procedures recorded a total treatment time of 15 minutes. Ramel et al. [33] had an implantoplasty duration of 21 ± 4 minutes with a sequence of diamond burs followed by silicone polishers and 13 ± 2 minutes with a sequence of diamond burs followed by Arkansas stones. Altogether, the profile of the rotating instruments used has a greater influence on the surface roughness than the duration of surface treatment [96].

The performance of rotary instruments is significantly determined by their average cutting speed and also depends on the bur diameter. Surprisingly larger round-end burs showed rather worse roughness values than the smaller spherically shaped burs. Spherical carbide burs work on a minimal surface in the apical region due to their geometric property. Conically shaped carbide burs, however, maintain a contact surface to the substrate, which explains their better cutting performance. In addition, carbide burs produce rather rougher surface profiles at higher than lower rotational speeds [96]. Spherical burs with larger diameter have higher cutting speeds with a possible negative influence on surface roughness. Generally speaking, carbide burs with lateral grooves showed higher average roughness values (Ra and Rz) compared to carbide burs without transverse grooves, thus preferring the latter for implantoplasty. In the literature, no influence on the quantity and composition of the biofilm is proven below an Ra value of 0.2 microns [95].

Looking at different protocols using a sequence of diamond burs followed by a Greenie silicone polisher reaches Ra levels of $0.59 \,\mu m$ (SD 0.19, Rz $4.35 \,\mu m$, SD 1.37) [33], a sequence of diamond burs followed by Brownie and Greenie silicone polishers achieves Ra levels of 0.32 μm (SD 0.14; Rz 2.31 μm, SD 0.95), a sequence of bud-shaped diamond burs followed by Arkansas stones aims Ra levels of $0.76 \,\mu$ m (SD 0.14, Rz 4.12 $\,\mu$ m, SD 0.72 and) and a sequence of conical silicon carbide stone followed by Arkansas stones, Brownie, and Greenie obtains values of Ra $0.38 \,\mu$ m (SD 0.15, Rz 1.87 $\,\mu$ m, SD 0.69) [69]. Silicone polishers are commonly associated with a pronounced pollution of the surgical site, contrary to the beneficial effect on surface roughness [20]. From the clinical point of view, the use of carbide burs without transverse grooves and an additional surface treatment with polishers (Brownies, Greenies, and Super-Greenies) is required to achieve the desired Ra value of 0.2 microns (Figures 14.57–14.61) [64, 96].

Sequences of diamond burs require a longer amount of time to achieve a subjectively evaluated smooth implant surface and thus have a higher risk of induced necrosis of bone cells [97, 98]. Clinically, the extent of the peri-implant defect, the type and shape of the suprastructure (e.g. crowns, various retention elements bolted or cemented), the geometry of the implant, as well as the presence of an implant shoulder can considerably influence the accessibility to the implant surface and the treatment possibilities. Basically, whenever possible, the pre-operative removal of the suprastructure is recommended even in funnel- or spiral-shaped defects to ensure sufficient accessibility. Due to these morphological particularities, carbide bur no. 9 and all spherically shaped burs are suitable for implantoplasty and provide an ideal contact area to the implant surface because of their shape and size. The best results with respect to the time required are achieved with carbide bur

Figure 14.57 Panoramic radiograph: periimplantitis in the lower jaw.

Figure 14.58 Open flap debridement.

Figure 14.59 Implantoplasty and removal of granulation tissue with particle remnants.

No. 8, probably due to the shallow angle of twist, the deep turns and the grooves of the cutting blades. Subsequent polishing, however, is also recommended for these types of burs [96].

Figure 14.60 Clinical situation five years after treatment.

Biocompatibility

Specific implantoplasty procedures (i.e. fine/extra-fine diamond burs and arkansas stone or carbide burs without transverse grooves and with polishers) result in a smooth transmucosal area with a close adhesion of the subepithelial connective tissue [99] The formation of new bone to implant contact after implantoplasty can not be identified, thus indicating that these surfaces do obviously not attract osteoblasts [24]. A positive effect of extension by implantoplasty on cell viability of gingival fibroblasts has also been confirmed by Schwarz et al. [36]. Furthermore, spreading and proliferation of fibroblasts are pronounced smooth when compared with rough titanium surfaces [100]. Besides, cellular interactions may also be influenced by the chemical elemental composition of the implant surface. In this regard, the presence of potential contaminants from the atmosphere, such as hydrocarbon and carbon has been shown to reduce the free surface energy and subsequently reduce cell adhesion [101]. Comparing these data with the specific characteristics obtained after implantoplasty,

Figure 14.61 Panoramic radiograph five years after treatment.

smoothened and machined surfaces reveal a comparable quantity (Wt%) of elements C, O, Na, Cl, K, and Si, but a significantly different quantity of elements Ti and Al. However, compared with rough surfaces, implantoplasty resulted in a significantly different quantity of elements C, O, Na, Ti, and Al, especially when using 30-mm particle size diamond egg-shaped burs [102]. The elemental composition of implantoplasty-modified surfaces closely resembles conventional machined surfaces [36].

Overheating

Overheating of the implant and the surrounding bone can occur during implantoplasty. An increase in temperature between 42 and 45 °C is known to result in a reversible heat shock [103], with a threshold for irreversible thermal damage to the bone at 47 °C for 1 minutes [97]. Indeed, studies assessing the impact of implant-abutment grinding on temperature levels at the implant/bone interface have shown, depending on the type of bur used and the contact time of the bur with the abutment, there is quite some variation in temperature increase, which under standard water cooling remains generally below the threshold of thermal damage [104]. During implantoplasty, however, the grinding is performed directly on the implant; thus, the temperature increase at the implant/bone interface may be different (i.e. higher) than is observed in studies involving abutment grinding [105]. In two laboratory studies, temperature increase at the implant/bone interface during implantoplasty under standard water cooling was momentarily $\langle 2^{\circ}$ C, irrespective of type of bur or duration of grinding $[105, 106]$, which is well below the 47^oC threshold. Thus, overheating of the neighboring bone tissues during implantoplasty with the potential danger of osseous thermal damage appears easy to control by means of standard cooling. Implantoplasty performed under proper irrigation, only leads to a minimal and temporarily increase (max. $1.8 \degree C$) in the surrounding tissue temperature, irrespective of the type of bur used. Thus, temperature remains appropriate for soft or peri-implant bone tissue integrity.

Particle Remnants

The role of titanium debris dispersion in hard and soft peri-implant tissues, which might sustain the inflammatory process, is still controversial [64]. There is no definitive evidence that the presence of titanium particles in peri-implant tissues is correlated to peri-implantitis [73]. Deposition of titanium particles in the neighboring hard and soft tissues due to grinding is difficult to control and variable amounts of such particles should be expected remaining in the tissues surrounding the implant after implantoplasty. No post-operative complications after implantoplasty are reported [99, 107]. Post-operatively, a slight to moderate deposition of titanium particles in the adjacent tissues, associated with a localized inflammatory cell infiltrate, can be observed histologically. In another study, one single case of slight mucosal pigmentation, attributed to titanium particle deposition from grinding, was observed [10]. One preclinical in vivo study identified the presence of titanium particles in the surrounding soft tissues after implantoplasty [99, 107], which was associated with only a limited extent, low-grade, chronic inflammatory response. Additionally except from the above-mentioned single case of tissue discoloration, no clinical study included in this review mentioned any other adverse events related to titanium particle deposition, despite the variable type of burs used and most likely the variable amount of deposition. Furthermore, some studies describe the ultramicroscopic morphology and elemental implant surface composition after implantoplasty [64], yet a definitive correlation with hard and soft tissue healing deficiencies in humans cannot be drawn. In this context, a consensus report concluded on the basis of the available evidence, although the possibility cannot unequivocally be excluded that it is not likely for titanium particles to cause adverse biological reactions in the peri-implant tissues [108].

As mentioned before, the use of silicone polishers can result in silicone debris contamination of both implant surface and peri-implant tissues [33, 41, 70, 109]. It has been suggested that the release of titanium and silicone micro and nano-particles can stimulate a number of cytokines, which, in turn, sustain a local inflammatory response [110]. In a recent in vitro study performing implantoplasty on pure grade 4 titanium and grade 5 titanium alloy implants, the released particles were analyzed in terms of size, ionic product release, and effects on human gingival fibroblasts. In addition to titanium, both aluminum and vanadium were detected in grade 5 particles. Carbon was detected in both grade 4 and 5 particles. Cells were cultured with the grade 4 particles and showed no adverse effects. Grade 5 particles, contain vanadium and thus can influence the reduction of metabolic activity and cell viability in higher concentrations. The exposure of particles (especially pure grade 4 titanium and grade 5 titanium alloy implants) and the presence of both titanium and carbon in the debris in usually low concentrations did not reduce cell viability in vitro [75]. Sufficient irrigation around the implant and suction of metallic debris during implantoplasty can reduce the deposition of metallic particles and ions into surrounding tissues [111].

Fracture/Reducing Diameter

While the progress of peri-implantitis can be effectively interrupted by implantoplasty [23, 24], material removal in the area of the implant shoulder raised doubts regarding the implants' future resistance to fracture. Implantoplasty reduces both the diameter of implant and the thickness of its walls (Figures 14.62–14.66) [42, 68, 112], thereby reducing the ability of the implant to resist fracture in response to functional load. In combination with an increase in crown-implant ratio due to bone loss, this can increase the lever effect, which in presence of high masticatory forces or parafunctional habits, can lead to complications such as fracture of the implant or loosening of the prosthetic screw. Recent evidence noted that implantoplasty significantly reduces the fracture resistance of implants with a 2.5 : 1 crown-to-implant ratio [113]. Implantoplasty and cyclic loading can reduce the implants' maximum bending strength and influence the elastic limits of internal connection [74].

Figure 14.62 Removal of suprastructure: peri-implantitis and mal-positioned implants.

Figure 14.63 Buccal fenestration defects and horizontal bone loss.

Figure 14.64 Implantoplasty to apical extension.

Figure 14.65 Wound Closure after laser application.

Figure 14.66 Clinical situation four years after treatment.

Cyclic loading in a laboratory masticator, simulating a five-year equivalent of chewing, is used to measure implant failure strength after different implantoplasty protocols. Assessing the implant radius, implant walls are reduced by implantoplasty ranging from 0.36 ± 0.04 mm. Subtracting

the width of the threads, there is only an unintended excess loss of the implant wall of 0.076 ± 0.18 mm [74]. Among instrumented implants of the different groups, fracture load and the coefficient of elasticity do not vary considerably. However, non-instrumented implants show higher fracture load values. This might not be surprising, since – by definition – implant material loss is a logic consequence of implantoplasty, which suggests – likewise – a loss of resistance to fracture. On the other hand, material removal should ideally refer only to the threads and should not or at least not relevantly affect the implant body. Implantoplasty does not affect implant strength and resistance to fracture of wide-diameter implants significantly (i.e., 4.7mm diameter; [68]). In contrast, variable results are reported for narrow/regular diameterimplants(i.e. 3.75–4.1mm diameter). A minimal reduction (i.e. 0.2mm) in core diameter of the implant does not significantly affect implant strength [41], while a reduction of about 17–40% in core diameter leads to average strength loss – depending on the implant platform/connection design [42, 68]. In a finite element analysis [112], implantoplasty resulted in stress increase on the implant body of 44–85%, more or less independent of the simulated bone loss height and height of implant grinding. Implantoplasty does not affect bone micro-strain, which depends on the extent of simulated bone loss and is critical when the endo-osseous portion of the implant is smaller than the exposed portion. In implants with a tapered design, implantoplasty may weaken the strength especially in types of narrow-diameter [68]. In particular, the bending strength was significantly reduced by performing implantoplasty with 30/15-μm egg-shaped diamond burs, Arkansas stones, and fine silicon polishers, thus resulting in a body fracture of all 3.75-mm diameter implants. In contrast, at 4.7-mm implants, the fractures solely occurred at the level of the abutment screws [68]. Implant material has a significant impact on tissue-level implants and regular-diameter implants, with zirconia being stronger than titanium. Implantoplasty significantly reduces the maximum implant failure strength, irrespective of the implant type/design, diameter, or material, yet the maximum implant failure strength of regular diameter and of narrow bone level implants remains high. There seems to be no clinically relevant impact on the majority of cases, except for those of single narrow titanium tissue implants, which may have an increased risk for mechanical complications. There is no significant higher failure rate in standard/regular diameterimplants, even though implantoplasty leads to a maximum of 40% strength reduction [42]. The highest failure rate is in narrow-diameter titanium tissue level implants. Within the laboratory settings, maximum implant failure strength of regular diameter and narrow Bone Level implants remained high despite implantoplasty $(i.e. >650$ and 440 N, respectively), while $>50\%$ of the narrow titanium tissue level implants fractured after implantoplasty during dynamic loading, simulating regular mastication. Altogether, implantoplasty seems to have no clinically relevant impact on the majority of cases, except from single narrow titanium tissue level implants, which may have an increased risk for mechanical complications [114]. This should be considered for peri-implantitis treatment planning (e.g. patient communication about potential complications).

Furthermore, there are no reported cases of implant fracture during clinical survival [74]. Therefore, the clinical impact of well-performed implantoplasty on the incidence of fracture seems to be low. Especially when gently reduction of the implant diameter will allow to separate the supracrestal area from the intra-bony area like in a platform switch format, counteracting possible future mean bone loss, the results show that after implantoplasty the implant is narrower in the medial than in the coronal region to achieve the desired "platform switch concept," This kind of implantoplasty leads to a distinct diameter reduction due to the waist-like format, and even here no implant fracture was detected after two to six years in function, proving the low risk of this complication [26].

Concerning the connection design, Gehrke et al. show a varying implant fracture strength after implantoplasty. Morse tapered implant connection is proven to be the most resistant followed by external and finally internal hexagon connection [42]. In reference to whether the connection is external hexagonal, internal hexagonal, or cone morse, the author observes a mean reduction of 37, 40, and 20%, respectively, in resistance to fatigue [6]. Even though internal hexagonal connection shows the greatest decrease in resistance to fatigue after implantoplasty, the external hexagonal connection has the lowest resistance values in total. Internal hexagon and conical connection implants seem to be more prone to fracture after implantoplasty. Implantoplasty significantly decreases the elastic limit of internal connection in dental titanium implants, especially in combination with a smaller diameter (3–3.5mm). The greater decrease in the elastic limit could be due to the connection occupying a greater space within the implant body, leading to a greater reduction in the wall thickness of treated implants, especially in the neck region. All tested bone-level implants are mostly deformed in the coronal area close to the abutment connection, whereas tissue-level implants are deformed in the apical area at the surface where implantoplasty has been performed [6]. The decrease in the elastic limit of implants with a narrower diameter is probably more clinically relevant, as it could be exceeded by occlusal forces in the posterior jaw in some patients [115]. The elastic limit of internal connection of narrow diameter implants treated with implantoplasty should be considered while planning the placement of the implant [116]. The risk of plastic deformation can increase if the implant is unsplinted and there is considerable bone loss.

Nevertheless, no clinical study has reported any remarkable complication due to implantoplasty; among 217–291 implants with implantoplasty, no implant fracture was reported during a follow-up of 3–126 months. Based on all currently available, yet limited, in vivo preclinical and clinical evidence, implantoplasty seems not associated with any remarkable mechanical or biological complication on the short-to medium-term [37].

Results

The treatment approach for implantoplasty requires the elimination of the contamination on the implant surface, thus mitigating the inflammatory process and stopping the progression of bone loss. Ultimately, hereby clinical outcomes portrayed by the decrease of purulence, bleeding, and reduction of periodontal pockets will be enhanced and increase implant survival. The survival and the success of implant therapy seem to be associated to patient-related factors such as smoking, poor oral hygiene, plaque formation, operator-related factors, and to implant-related factors such as implant shape, implant surface topography, and roughness [52]. Many in vitro and in vivo studies show a significant impact of parameters like surface-free energy and surface roughness on de novo plaque formation [65].

In a review, clinical peri-implant parameters such as PD and BOP improved after access apical surgery only [15, 22–24], after surface decontamination with chlorhexidine (CHX) and/or cetylpyridinium chloride (CPC) [22, 117] and after systemic antibiotics [15, 21, 58, 118]. In one study, no clinical benefits were seen using adjunctive systemic azithromycin in combination with open flap debridement [14]. No potential benefits of systemic antibiotics were found over three years [21]. The clinical parameters improved significantly especially after implantoplasty [23] or after the use of systemic antimicrobials [15, 58]. In detail, implantoplasty (diamond/arkansas burs + silicone polishers) as an adjunct to open flap debridement with bone recontouring and apical flap repositioning resulted in better BOP and PD scores, but higher mean mucosal recessions compared to the control group with open flap debridement alone (PD: 1.64 ± 1.29 versus 2.3 ± 1.45 mm), where persistent active signs of peri-implant inflammation recurred in all patients after 24 months. Adjunctive benefits in resective surgical treatment with an apically repositioned flap, bone recontouring, and surface debridement with addition of 0.12% CHX + 0.05% CPC to a placebosolution (without CHX/CPC) [22, 117] tend to show greater immediate suppression of anaerobic bacteria on the implant surface than a placebo-solution, but do not lead to superior clinical results or differences in mean marginal bone loss at 12-month follow-up. Similarly, the adjunctive use of 980-nm diode laser to mechanical open flap debridement alone [16] failed to reveal any significant clinical improvements in mean BOP and PD scores at six-months follow-up. The probing depth is a controversial criterion in terms of disease resolution [119]. A pre-surgical initial probing depth of more than 7mm negatively influences the clinical prognosis of implants treated with non augmentative therapy [3]. The clinical signs of inflammation seem more important since peri-implant health can also exist around implants with high mean bone loss and consequently high probing depths [25]. Overall implantoplasty results in positive clinical and radiographic findings, such as low bleeding rates, shallow probing pocket depths, increased clinical attachment levels, and increased or stable bone levels on the short- to medium-term (Figures 14.67–14.71) [23, 24, 62, 120, 121]. Increased PPD can be correlated with a higher degree of inflammation of the peri-implant mucosa [122].

Stable radiographic peri-implant bone levels result after systemic application of antibiotics [14, 15, 58] or local use of chemical compounds [22, 117]. In studies with significant improvement of clinical peri-implant parameters after implantoplasty, radiographic bone levels and marginal bone loss were significantly lower after a three-year follow up [24] with an interproximal bone loss at control sites up to 1.45–1.54mm.

A total number of 35 implants were removed after non augmentative surgery due to progressive recurrence of peri-implant inflammation [21–24, 58]. One implant was removed because of implant neck fracture [117].

Figure 14.67 Baseline situation: peri-implantitis with horizontal bone loss and cemented suprastructure.

Figure 14.68 Open flap debridement and implantoplasty. **Figure 14.71** Clinical situation six years after treatment.

Figure 14.69 Wound closure.

Figure 14.70 Clinical situation three years after treatment.

Four studies reported on smoking status of the patients, ranging from 25% [15] to 59.1% [117]. Despite two studies [14, 15] with no negative effect of smoking on treatment outcome, smoking influences treatment success when adjusted for baseline and follow-up [22, 117].

Treatment Success

Implantoplasty as an adjunct to surgical therapy proved effective in terms of disease resolution and implant survival rate. For a therapeutic success, implant location, defect morphology, and width of KM are also important. In a nine-year follow-up retrospective analysis about implantoplasty, Pommer et al. reports a success rate 87.2% [121]. Similarly, Romeo et al. found a cumulative survival rate of 87.5% for resective implantoplasty therapy in a three-year follow-up [24]. Englezos et al. have a 0% failure rate and 92.5% marginal bone stability rate with a mean PPD reduction of ∼5mm after implantoplasty and osteoplasty therapy in a prospective two-year follow-up case series [123]. According to Carcuac et al. overall success is defined as no MBL >0.5mm, no BOP, no suppuration, and no PD >4mm and is seen in 33% of the cases over a three-year follow-up after resective therapy $[21]$. In a two-year follow-up, prospective study resective and bone recountoring treatment has a 48% peri-implantitis resolution index [124]. Bianchini et al. see stable bone levels at 87% in their case series about implants after implantoplasty combined with resective therapy [26]. Ravidà et al. analyses implant survival and clinical/ radiographic outcomes of peri-implantitis after resective therapy with (test) or without (control) adjunct implantoplasty. Interestingly, implants in the test group respond more favorably considering the higher survival rate (90 versus 81.6%) over a mean ∼three-year period, although not reaching statistical significance [125]. Other authors have reported implantoplasty as adjunct to a combined therapeutic approach (so-called combined approach).

Follow-Up Care/SPT

The lack of compliance during maintenance therapy is associated with tooth loss and attachment loss [126, 127]. A relationship between microbial plaque and diseases such

as gingivitis and periodontitis is numerously demonstrated [128, 129]. Plaque removal as casual therapy shows improvements in inflammatory lesions due to its plaque associated etiopathogenesis. A subsequent study involving mucositis patients demonstrates that efficient plaque control is critical for the prevention of peri-implantitis [130]. In this study, the incidence of peri-implantitis over a five year period is significantly lower in patients undergoing maintenance therapy (18%) than in patients without maintenance therapy (44%). Roccuzzo et al [131] also find a higher prevalence of peri-implantitis over a 10-year period in the absence of maintenance therapy (41%) than with maintenance therapy (27%). Patients who attend maintenance therapy less than two times a year show an increased risk of developing peri-implantitis (OR 4.69; 95% CI 1.17–18.79). Moreover, a strong association between inadequate home-based oral hygiene and peri-implantitis is shown in 4 studies with an odds ratio ranging from 5 to 14 [9, 76, 132]. However, conflicting findings are also published [18, 30, 45], despite the fact that one singular plaque index recorded in these studies generally does not reflect a complete long-term oral hygiene status. Serino and Stroem investigate the oral hygiene ability of patients with peri implantitis at implant-supported restorations [133] and are able to show that peri-implantitis is diagnosed in only 18% of the areas accessible to oral hygiene and in 65% of the areas not accessible to sufficient oral healthcare.

Implant failure is influenced by the amount of mean bone loss at the time of treatment. Although hardly reported, the severity of mean bone loss is a commonsensical risk for implant survival following peri-implantitis therapy, and there seems to be an agreement in literature regarding its significance [125, 134]. Furthermore, a systematic review and meta-analysis by Monje et al. [135] show not only the significance of SPT but also how the variable of SPT frequency is vital for the prevention of peri implant diseases. Consistent with these findings, implants placed in patients receiving \leq 2 annual SPT visits and $>$ 2 annual visits have 0.53 and 0.75mm less mean bone loss per year, respectively, compared to patients without SPT.

Follow-up care (supportive periodontal therapy) is the key to a successful, long-term treatment of peri-implant inflammation [78] and only functions when potential patient-specific risk factors are taken into consideration. During maintenance therapy, intensive, repetitive instruction, demonstration, and motivation of the patient is indispensable [136]. Moreover, the peri-implant probing depth must gently be recorded, and the reevaluation of effective home-based oral care and a professional oral hygiene must be carried out. The recall interval should be selected according to the individual's risk profile [137, 138], whereby patients with previous peri-implant inflammation

are generally considered to be at an increased risk [78]. Maintenance frequency interval for most studies range between three and six months. Taking the bacterial etiology into account, a strict maintenance program with more than two visits is imperative for stability of the results in the long-term [37]. For this reason, a close-meshed three month interval for supportive periodontal therapy should always be selected initially, which can regularly be adapted on a patient-specific basis according to existing risk factors.

Discussion

Surgical techniques are used depending on multiple factors including patient general health condition, oral hygiene, type of bony defects, implant surface, post-operative maintenance program and other factors that cannot completely be assumed in a systematic literature review. The non augmentative treatment concept is indicated for supracrestal bone defects (horizontal bone loss) with exposed threads in esthetically non-demanding areas based on patient needs and satisfaction. It involves reduction or elimination of pathological peri-implant pockets, the apical positioning of a mucosal flap, or recontouring the bone with or without implant surface modification; the so-called implantoplasty. In case of modifications of implant surfaces, the rough design should be removed and polished. However, a concern about remaining titanium particles should be addressed.

Even in cases of peri-implant mucositis, it can be advisable to perform an access flap for proper mechanical and chemical decontamination (i.e. additional removal of cement remnants) of the implant surface.

The resolution of infection can be achieved by a proper method of implant surface decontamination. Clinical studies comparing different decontamination methods or placebo fail to reveal significant differences between different, locally applied decontamination methods on clinical treatment outcomes [22, 117]. Nevertheless, a combined mechanical and chemical removal of biofilm is recommended [5]. Furthermore, adjunctive systemic antibiotics have no impact on the treatment's success for implants with a machined surface, and a positive effect was observed for the treatment success of implants with a rough surface only in the first year [58]. Local antibiotic delivery in addition to mechanical debridement and irrigation with an antimicrobial agent may be an effective option for treating peri-implantitis [12, 13]. Moreover, the development of bacterial resistance seems to be unlikely even in the event of repeated applications [139].

Resective peri-implantitis therapy combined with implantoplasty leads to superior clinical and radiographic

treatment outcomes compared to resective treatment alone. In particular, implantoplasty, which can be performed with diamond or carbide burs and metal polishing instruments with irrigation, positively influence implant survival rates (100% test, 87.5% control group), significantly reduced peri-implant pocket depths, and reduced bleeding scores [23]. Implantoplasty is associated with stabile interproximal bone levels at the three-year follow-up, while significant radiographic bone loss at control sites is detected [24]. However, the gingival recession index is significantly higher when implant surface modification is performed [23]. Therefore, this type of surgical technique can only be of benefit primarily in the nonesthetic areas and alternative treatments should be developed for the esthetic zone [5].

The resective therapy outcomes are influenced by the experience of the surgical team, the amount of peri-implant bone loss, maximum probing pocket depth at baseline, patient smoking habits, and the presence of plaque during the follow-up [117].

Analysis of studies and achieved reliability of treatments revealed similarities in treatment approaches between the protocols including (i) pretreatment phase, (ii) cause related therapy, and (iii) maintenance phase. Oral hygiene instructions and their importance must be stressed to patients before and after the treatment [140]. Non-surgical subgingival mechanical debridement in conjunction with local antibacterials, like chlorhexidine digluconate or locally delivered antibiotics are effective in reducing soft tissue inflammation $[13, 141]$ and should be the first step in successful treatment [140]. The supportive treatment phase is mandatory for the success of peri-implantitis treatment [140]. In a five-year follow-up observational study, healthy peri-implant tissue conditions in patients with high oral hygiene standards (maintenance every six months) could be maintained for the majority of patients following resective peri-implant surgery [142]. Further disease progression occurred only in 10% of treated implants, revealing that risk factors like residual pockets after surgery, smoking, poor oral hygiene, untreated periodontal disease, and diabetes may modify both the initial and long term outcome [140].

All studies included implants with both cemented or screw-retained suprastructures. Hence, some surgeries were performed with suprastructures in place and some after removal of the suprastructures. All clinical assessments were made with suprastructures in place. The suprastructure probably might affect quality of implant decontamination and modification, flap design, and possibly the measurement of clinical parameters If possible, it is strongly recommended to remove the suprastructure during surgical intervention and to adapt it for better cleanability.

Limitations of the revised studies are relatively small sample sizes and short follow-up periods. Furthermore, there are inconsistencies in methodology with various treatment modalities, limited control groups, different implant systems, and follow-up results from the same research groups. Unclear or high risk of bias of included studies may have an effect on the conclusions [143]. The evidence of the review is limited due to significant variations observed in included studies. Therefore, there is a substantial need for randomized controlled studies with a proper design and powerful sample size to provide strong and comparable evidence of benefits regarding non augmentative procedures for treating peri-implantitis.

Limitations

Due to the subtractive nature of implantoplasty, it is reasonable to consider the possibility of various mechanical complications either during the procedure (e.g. perforation of the implant body or destruction of the implant-abutment connection) or at a later stage (e.g. implant collar deformation and loosening of the supra-structure, fixation-screw fracture, and implant fracture). While the complications during the procedure might be avoidable, if taken care, the late complications might not be able to control, as they are dependent on the altered (weakened) mechanical properties of the implant. Indeed, the laboratory studies included in this review have indicated that narrow/standard – but not wide – diameter implants suffer from a variable, mostly significant, extent of weakening due to implantoplasty. In this context, the information regarding the clinical performance of implants after implantoplasty is based on relatively limited numbers, with short- to medium-term follow-up. Secure conclusions may be drawn with caution, because of (i) different brands/systems, thus representing different connection types, (ii) various positions in the mouth, implying most likely the use of only wide diameter implants, (iii) different types of reconstructions, including single crowns, (iv) different follow-up length, and (v) remarkable mechanical complications, it is reasonable to claim that implantoplasty does not appear to significantly compromise the mechanical properties of implants, at least on the short-to medium-term; nevertheless, mechanical complications cannot definitely be excluded.

Conclusion

It was shown that surgical non-regenerative modalities treating peri-implantitis can reduce the amount of inflammation in a short-term follow-up, but seem limited effective in the long-term. Using implantoplasty in surgical non-regenerative treatment leads to a significant reduction in bleeding on probing and probing depths and may result in improvement of clinical and radiographic parameters, up to three years after surgery compared to mechanical debridement alone. Application of systemic antibiotics, chemical compounds, or diode lasers does not

result in significant clinical or radiographic long-term improvements. Implantoplasty as adjunct to surgical therapy followed by regular supportive maintenance therapy is effective in terms of disease resolution and implant survival rate. Implant location, defect morphology, and width of keratinized mucosa are indicators of therapeutic success.

References

- **1** Zitzmann, N.U. and Berglundh, T. (2008). Definition and prevalence of peri-implant diseases. *Journal of Clinical Periodontology* 35: 286–291.
- **2** Lang, N.P. and Berglundh, T. (2011). Periimplant diseases: where are we now? – consensus of the seventh European workshop on periodontology. *Journal of Clinical Periodontology* 38: 178–181.
- **3** Koldsland, O.C., Scheie, A.A., and Aass, A.M. (2010). Prevalence of peri-implantitis related to severity of the disease with different degrees of bone loss. *Journal of Periodontology* 81 (2): 231–238.
- **4** Mir-Mari, J., Mir-Orfila, P., Figueiredo, R. et al. (2012). Prevalence of peri-implant diseases. A cross-sectional study based on a private practice environment. *Journal of Clinical Periodontology* 39 (5): 490–494.
- **5** Renvert, S. and Polyzois, I.N. (2015). Clinical approaches to treat peri-implant mucositis and peri-implantitis. *Periodontology 2000* 68 (1): 369–404.
- **6** Dieguez-Pereira, M., Chavarri-Prado, D., Viteri-Agustin, I. et al. (2021). Effect of implantoplasty on the elastic limit of dental implants of different diameters. *Internationl Journal of Implant Dentistry* 7 (1): 88.
- **7** Albrektsson, T.O., Johansson, C.B., and Sennerby, L. (1994). Biological aspects of implant dentistry: osseointegration. *Periodontology 2000* 4: 58–73.
- **8** Berglundh, T., Jepsen, S., Stadlinger, B., and Terheyden, H. (2019). Peri-implantitis and its prevention. *Clinical Oral Implants Research* 30 (2): 150–155.
- **9** Rokn, A., Aslroosta, H., Akbari, S. et al. (2017). Prevalence of peri-implantitisin patients not participating in well-designed supportive periodontal treatments: a cross-sectional study. *Clinical Oral Implants Research* 28 (3): 314–319.
- **10** Schwarz, F., Sahm, N., Iglhaut, G., and Becker, J. (2011). Impact of the method of surface debridement and decontamination on the clinical outcome following combined surgical therapy of peri-implantitis: a randomized controlled clinical study. *Journal of Clinical Periodontology* 38 (3): 276–284.
- **11** Schwarz, F., Derks, J., Monje, A., and Wang, H.L. (2018). Peri-implantitis. *Journal of Periodontology* 89 (Suppl 1): S267–S290.
- **12** Mombelli, A., Feloutzis, A., Bragger, U., and Lang, N.P. (2001). Treatment of peri-implantitis by local delivery of tetracycline. Clinical, microbiological and radiological results. *Clinical Oral Implants Research* 12 (4): 287–294.
- **13** Renvert, S., Lessem, J., Dahlen, G. et al. (2006). Topical minocycline microspheres versus topical chlorhexidine gel as an adjunct to mechanical debridement of incipient peri-implant infections: a randomized clinical trial. *Journal of Clinical Periodontology* 33 (5): 362–369.
- **14** Hallstrom, H., Persson, G.R., Lindgren, S., and Renvert, S. (2017). Open flap debridement of peri-implantitis with or without adjunctive systemic antibiotics: a randomized clinical trial. *Journal of Clinical Periodontology* 44 (12): 1285–1293.
- **15** Heitz-Mayfield, L.J.A., Salvi, G.E., Mombelli, A. et al. (2012). Anti-infective surgical therapy of peri-implantitis. A 12-month prospective clinical study. *Clinical Oral Implants Research* 23 (2): 205–210.
- **16** Papadopoulos, C.A., Vouros, I., Menexes, G., and Konstantinidis, A. (2015). The utilization of a diode laser in the surgical treatment of peri-implantitis. A randomized clinical trial. *Clinical Oral Investigations* 19 (8): 1851–1860.
- **17** Jepsen, K., Jepsen, S., Laine, M.L. et al. (2016). Reconstruction of peri-implant osseous defects: a multicenter randomized trial. *Journal of Dental Research* 95 (1): 58–66.
- **18** Khoury, F. and Buchmann, R. (2001). Surgical therapy of peri-implant disease: a 3-year follow-up study of cases treated with 3 different techniques of bone regeneration. *Journal of Periodontology* 72 (11): 1498–1508.
- **19** Roccuzzo, M., Gaudioso, L., Lungo, M., and Dalmasso, P. (2016). Surgical therapy of single peri-implantitis intrabony defects, by means of deproteinized bovine bone mineral with 10% collagen. *Journal of Clinical Periodontology* 43 (3): 311–318.
- **20** Schwarz, F., John, G., Schmucker, A. et al. (2017). Combined surgical therapy of advanced peri-implantitis evaluating two methods of surface decontamination: a 7-year follow-up observation. *Journal of Clinical Periodontology* 44 (3): 337–342.

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- **21** Carcuac, O., Derks, J., Abrahamsson, I. et al. (2017). Surgical treatment of peri-implantitis: 3-year results from a randomized controlled clinical trial. *Journal of Clinical Periodontology* 44 (12): 1294–1303.
- **22** de Waal, Y.C., Raghoebar, G.M., Huddleston Slater, J.J. et al. (2013). Implant decontamination during surgical peri-implantitis treatment: a randomized, double-blind, placebo-controlled trial. *Journal of Clinical Periodontology* 40 (2): 186–195.
- **23** Romeo, E., Ghisolfi, M., Murgolo, N. et al. (2005). Therapy of peri-implantitis with resective surgery. A 3-year clinical trial on rough screw-shaped oral implants. Part I: clinical outcome. *Clinical Oral Implants Research* 16 (1): 9–18.
- **24** Romeo, E., Lops, D., Chiapasco, M. et al. (2007). Therapy of peri-implantitis with resective surgery. A 3-year clinical trial on rough screw-shaped oral implants. Part II: radiographic outcome. *Clinical Oral Implants Research* 18 (2): 179–187.
- **25** Berglundh, T., Armitage, G., Araujo, M.G. et al. (2018). 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 Clinical Periodontology* 45 (Suppl 20): S286–S291.
- **26** Bianchini, M.A., Galarraga-Vinueza, M.E., Apaza-Bedoya, K. et al. (2019). Two to six-year disease resolution and marginal bone stability rates of a modified resective implantoplasty therapy in 32 peri-implantitis cases. *Clinical Implant Dentistry and Related Research* 21 (4): 758–765.
- **27** Karring, E.S., Stavropoulos, A., Ellegaard, B., and Karring, T. (2005). Treatment of peri-implantitis by the vector system. *Clinical Oral Implants Research* 16 (3): 288–293.
- **28** Renvert, S., Roos-Jansåker, A.-M., and Claffey, N. (2008). Non-surgical treatment of peri-implant mucositis and peri-implantitis: a literature review. *Journal of Clinical Periodontology* 35: 305–315.
- **29** Schwarz, F., Hegewald, A., John, G. et al. (2013). Four year follow-up of combined surgical therapy of advanced peri-implantitis evaluating two methods of surface decontamination. *Journal of Clinical Periodontology* 40 (10): 962–967.
- **30** Renvert, S., Polyzois, I., and Claffey, N. (2012). Surgical therapy for the control of peri-implantitis. *Clinical Oral Implants Research* 23 (Suppl 6): 84–94.
- **31** Berglundh, T., Gotfredsen, K., Zitzmann, N.U. et al. (2007). Spontaneous progression of ligature induced peri-implantitis at implants with different surface roughness: an experimental study in dogs. *Clinical Oral Implants Research* 18 (5): 655–661.
- **32** Quirynen, M., van der Mei, H.C., Bollen, C.M. et al. (1993). An in vivo study of the influence of the surface roughness of implants on the microbiology of supra-and subgingival plaque. *Journal of Dental Research* 72 (9): 1304–1309.
- **33** Ramel, C.F., Lussi, A., Ozcan, M. et al. (2016). Surface roughness of dental implants and treatment time using six different implantoplasty procedures. *Clinical Oral Implants Research* 27 (7): 776–781.
- **34** Toma, S., Behets, C., Brecx, M.C., and Lasserre, J.F. (2018). In vitro comparison of the efficacy of peri-implantitis treatments on the removal and recolonization of Streptococcus gordonii biofilm on titanium disks. *Materials (Basel)* 11 (12): 2484.
- **35** Albouy, J.P., Abrahamsson, I., Persson, L.G., and Berglundh, T. (2011). Implant surface characteristics influence the outcome of treatment of peri-implantitis: an experimental study in dogs. *Journal of Clinical Periodontology* 38 (1): 58–64.
- **36** Schwarz, F., John, G., and Becker, J. (2017). The influence of implantoplasty on the diameter, chemical surface composition, and biocompatibility of titanium implants. *Clinical Oral Investigations* 21 (7): 2355–2361.
- **37** Esteves Lima, R.P., Abreu, L.G., Belem, F.V. et al. (2021). Is implantoplasty efficacious at treating peri-implantitis? A systematic review and meta-analysis. *Journal of Oral and Maxillofacial Surgery* 79 (11): 2270–2279.
- **38** Galarraga-Vinueza, M.E., Passoni, B., Benfatti, C.A.M. et al. (2017). Inhibition of multi-species oral biofilm by bromide doped bioactive glass. *Journal of Biomedical Materials Research. Part A* 105 (7): 1994–2003.
- **39** Teughels, W., Van Assche, N., Sliepen, I., and Quirynen, M. (2006). Effect of material characteristics and/or surface topography on biofilm development. *Clinical Oral Implants Research* 17 (Suppl 2): 68–81.
- **40** Apaza-Bedoya, K., Tarce, M., Benfatti, C.A.M. et al. (2017). Synergistic interactions between corrosion and wear at titanium-based dental implant connections: a scoping review. *Journal of Periodontal Research* 52 (6): 946–954.
- **41** Costa-Berenguer, X., Garcia-Garcia, M., Sanchez-Torres, A. et al. (2018). Effect of implantoplasty on fracture resistance and surface roughness of standard diameter dental implants. *Clinical Oral Implants Research* 29 (1): 46–54.
- **42** Gehrke, S.A., Aramburu Junior, J.S., Dedavid, B.A., and Shibli, J.A. (2016). Analysis of implant strength after implantoplasty in three implant-abutment connection designs: an in vitro study. *The International Journal of Oral & Maxillofacial Implants* 31 (3): e65–e70.
- **43** Mombelli, A. and Lang, N.P. The diagnosis and treatment of peri-implantitis. *Periodontology 2000* 1998 (17): 63–76.
- **44** Ramanauskaite, A., Obreja, K., Sader, R. et al. (2019). Surgical treatment of periimplantitis with augmentative techniques. *Implant Dentistry* 28 (2): 187–209.
- **45** Maminskas, J., Puisys, A., Kuoppala, R. et al. (2016). The prosthetic influence and biomechanics on peri-implant strain: a systematic literature review of finite element studies. *Journal of Oral & Maxillofacial Research* 7 (3): e4.
- **46** Keeve, P.L. and Khoury, F. (2017). Long-term results of peri-implant conditions in periodontally compromised patients following lateral bone augmentation. *The International Journal of Oral & Maxillofacial Implants* 32 (1): 137–146.
- **47** Petropoulos, G., McKay, I.J., and Hughes, F.J. (2004). The association between neutrophil numbers and interleukin-1alpha concentrations in gingival crevicular fluid of smokers and non-smokers with periodontal disease. *Journal of Clinical Periodontology* 31 (5): 390–395.
- **48** Louropoulou, A., Slot, D.E., and Van der Weijden, F. (2014). The effects of mechanical instruments on contaminated titanium dental implant surfaces: a systematic review. *Clinical Oral Implants Research* 25 (10): 1149–1160.
- **49** Renvert, S. and Polyzois, I. (2015). Risk indicators for peri-implant mucositis: a systematic literature review. *Journal of Clinical Periodontology* 42 (Suppl 16): S172–S186.
- **50** Lang, N.P., Mombelli, A., Tonetti, M.S. et al. (1997). Clinical trials on therapies for peri-implant infections. *Annals of Periodontology* 2 (1): 343–356.
- **51** Mombelli, A. and Lang, N.P. (1992). Antimicrobial treatment of peri-implant infections. *Clinical Oral Implants Research* 3 (4): 162–168.
- **52** Keeve, P. (2022). Patient-specific treatment of peri implant inflammation. *Dtsch Zahnärztl Z International* 4: 18–29.
- **53** Keeve, P.L. and Conrad, T. (2021). Implantate im parodontal vorgeschädigten Gebiss. *der Junge zahnarzt* 12 (3): 14–25.
- **54** Renvert, S., Aghazadeh, A., Hallstrom, H., and Persson, G.R. (2014). Factors related to peri-implantitis -a retrospective study. *Clinical Oral Implants Research* 25 (4): 522–529.
- **55** Bassetti, M., Schar, D., Wicki, B. et al. (2014). Anti infective therapy of peri-implantitis with adjunctive local drug delivery or photodynamic therapy: 12-month outcomes of a randomized controlled clinical trial. *Clinical Oral Implants Research* 25 (3): 279–287.
- **56** Buchter, A., Meyer, U., Kruse-Losler, B. et al. (2004). Sustained release of doxycycline for the treatment of peri-implantitis: randomised controlled trial. *The British Journal of Oral & Maxillofacial Surgery* 42 (5): 439–444.
- **57** Renvert, S., Hirooka, H., Polyzois, I. et al. (2019). Diagnosis and non-surgical treatment of peri-implant diseases and maintenance care of patients with dental implants -Consensus report of working group 3. *International Dental Journal* 69 (Suppl 2): 12–17.
- **58** Carcuac, O., Derks, J., Charalampakis, G. et al. (2016). Adjunctive systemic and local antimicrobial therapy in the surgical treatment of peri-implantitis: a randomized controlled clinical trial. *Journal of Dental Research* 95 (1): 50–57.
- **59** Romanos, G.E. and Nentwig, G.H. (2008). Regenerative therapy of deep peri-implant infrabony defects after CO2 laser implant surface decontamination. *International Journal of Periodontics Restorative Dentistry* 28 (3): 245–255.
- **60** Wang, C.W., Renvert, S., and Wang, H.L. (2019). Nonsurgical treatment of periimplantitis. *Implant Dentistry* 28 (2): 155–160.
- **61** Schwarz, F., Sahm, N., Schwarz, K., and Becker, J. (2010). Impact of defect configuration on the clinical outcome following surgical regenerative therapy of peri implantitis. *Journal of Clinical Periodontology* 37 (5): 449–455.
- **62** Keeve, P.L., Koo, K.T., Ramanauskaite, A. et al. (2019). Surgical treatment of periimplantitis with non augmentative techniques. *Implant Dentistry* 28 (2): 177–186.
- **63** Khoury, F., Keeve, P.L., Ramanauskaite, A. et al. (2019). Surgical treatment of peri-implantitis - consensus report of working group 4. *International Dental Journal* 69 (Suppl 2): 18–22.
- **64** Sivolella, S., Brunello, G., Michelon, F. et al. (2021). Implantoplasty: carbide burs vs diamond sonic tips. An in vitro study. *Clinical Oral Implants Research* 32 (3): 324–336.
- **65** Mombelli, A. (1999) Prevention and therapy of peri implant infections. *Proceedings of the 3rd European Workshop on Periodontology,* Thurgau, Switzerland (30 January–3 February 1999), pp. 281–303. Quintessence.
- **66** Ntrouka, V.I., Slot, D.E., Louropoulou, A., and Van der Weijden, F. (2011). The effect of chemotherapeutic agents on contaminated titanium surfaces: a systematic review. *Clinical Oral Implants Research* 22 (7): 681–690.
- **67** Burgers, R., Gerlach, T., Hahnel, S. et al. (2010). In vivo and in vitro biofilm formation on two different titanium implant surfaces. *Clinical Oral Implants Research* 21 (2): 156–164.
- **68** Chan, H.L., Oh, W.S., Ong, H.S. et al. (2013). Impact of implantoplasty on strength of the implant-abutment complex. *The International Journal of Oral & Maxillofacial Implants* 28 (6): 1530–1535.

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- **69** Sahrmann, P., Luso, S., Mueller, C. et al. (2019). Titanium implant characteristics after implantoplasty: an in vitro study on two different kinds of instrumentation. *The International Journal of Oral & Maxillofacial Implants* 34 (6): 1299–1305.
- **70** Tawse-Smith, A., Kota, A., Jayaweera, Y. et al. (2016). The effect of standardised implantoplasty protocol on titanium surface roughness: an in-vitro study. *Brazilian Oral Research* 30 (1): e137.
- **71** Gealh, W.C., Mazzo, V., Barbi, F., and Camarini, E.T. (2011). Osseointegrated implant fracture: causes and treatment. *The Journal of Oral Implantology* 37 (4): 499–503.
- **72** Gehrke, S.A., Dos Santos, S., Vianna, M., and Dedavid, B.A. (2014). Influence of bone insertion level of the implant on the fracture strength of different connection designs: an in vitro study. *Clinical Oral Investigations* 18 (3): 715–720.
- **73** Stavropoulos, A., Bertl, K., Eren, S., and Gotfredsen, K. (2019). Mechanical and biological complications after implantoplasty-a systematic review. *Clinical Oral Implants Research* 30 (9): 833–848.
- **74** Jorio, I.C., Stawarczyk, B., Attin, T. et al. (2021). Reduced fracture load of dental implants after implantoplasty with different instrumentation sequences. An in vitro study. *Clinical Oral Implants Research* 32 (8): 881–892.
- **75** Barrak, F.N., Li, S., Muntane, A.M., and Jones, J.R. (2020). Particle release from implantoplasty of dental implants and impact on cells. *International Journal of Implant Dentistry* 6 (1): 50.
- **76** Schwarz, F., Becker, K., Sahm, N. et al. (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. *Clinical Oral Implants Research* 28 (1): 24–28.
- **77** Romandini, M., Berglundh, J., Derks, J. et al. (2021). Diagnosis of peri-implantitis in the absence of baseline data: a diagnostic accuracy study. *Clinical Oral Implants Research* 32 (3): 297–313.
- **78** Polyzois, I. (2019). Treatment planning for periimplant mucositis and periimplantitis. *Implant Dentistry* 28 (2): 150–154.
- **79** Stuani, V.T., Kim, D.M., Nagai, M. et al. (2021). Effectiveness and surface changes of different decontamination protocols at smooth and minimally rough titanium surfaces. *Journal of Periodontology* 92 (5): 704–715.
- **80** Kotsakis, G.A., Lan, C., Barbosa, J. et al. (2016). Antimicrobial agents used in the treatment of peri implantitis alter the physicochemistry and cytocompatibility of titanium surfaces. *Journal of Periodontology* 87 (7): 809–819.
- **81** Valderrama, P. and Wilson, T.G. Jr. (2013). Detoxification of implant surfaces affected by peri-implant disease: an

overview of surgical methods. *International Journal of Dentistry* 2013: 740680.

- **82** Wheelis, S.E., Gindri, I.M., Valderrama, P. et al. (2016). Effects of decontamination solutions on the surface of titanium: investigation of surface morphology, composition, and roughness. *Clinical Oral Implants Research* 27 (3): 329–340.
- **83** Gittens, R.A., Olivares-Navarrete, R., Cheng, A. et al. (2013). The roles of titanium surface micro/ nanotopography and wettability on the differential response of human osteoblast lineage cells. *Acta Biomaterialia* 9 (4): 6268–6277.
- **84** Zhao, G., Schwartz, Z., Wieland, M. et al. (2005). High surface energy enhances cell response to titanium substrate microstructure. *Journal of Biomedical Materials Research. Part A* 74 (1): 49–58.
- **85** Schwarz, F., Papanicolau, P., Rothamel, D. et al. (2006). Influence of plaque biofilm removal on reestablishment of the biocompatibility of contaminated titanium surfaces. *Journal of Biomedical Materials Research. Part A* 77 (3): 437–444.
- **86** Quirynen, M., Vogels, R., Peeters, W. et al. (2006). Dynamics of initial subgingival colonization of 'pristine' peri-implant pockets. *Clinical Oral Implants Research* 17 (1): 25–37.
- **87** Mombelli, A., van Oosten, M.A., Schurch, E. Jr., and Land, N.P. (1987). The microbiota associated with successful or failing osseointegrated titanium implants. *Oral Microbiology and Immunology* 2 (4): 145–151.
- **88** Shibli, J.A., Melo, L., Ferrari, D.S. et al. (2008). Composition of supra-and subgingival biofilm of subjects with healthy and diseased implants. *Clinical Oral Implants Research* 19 (10): 975–982.
- **89** Azzola, F., Ionescu, A.C., Ottobelli, M. et al. (2020). Biofilm formation on dental implant surface treated by implantoplasty: an in situ study. *Dentistry Journal (Basel)* 8 (2): 40.
- **90** Quirynen, M., Marechal, M., Busscher, H.J. et al. (1990). The influence of surface free energy and surface roughness on early plaque formation. An in vivo study in man. *Journal of Clinical Periodontology* 17 (3): 138–144.
- **91** Schwarz, F., Becker, J., Civale, S. et al. (2018). Onset, progression and resolution of experimental peri-implant mucositis at different abutment surfaces: a randomized controlled two-centre study. *Journal of Clinical Periodontology* 45 (4): 471–483.
- **92** Schwarz, F., Mihatovic, I., Golubovic, V. et al. (2014). Experimental peri-implant mucositis at different implant surfaces. *Journal of Clinical Periodontology* 41 (5): 513–520.
- **93** Zitzmann, N.U., Abrahamsson, I., Berglundh, T., and Lindhe, J. (2002). Soft tissue reactions to plaque formation at implant abutments with different surface topography. An experimental study in dogs. *Journal of Clinical Periodontology* 29 (5): 456–461.
- **94** Albouy, J.P., Abrahamsson, I., and Berglundh, T. (2012). Spontaneous progression of experimental peri implantitis at implants with different surface characteristics: an experimental study in dogs. *Journal of Clinical Periodontology* 39 (2): 182–187.
- **95** Bollen, C.M., Papaioanno, W., Van Eldere, J. et al. (1996). The influence of abutment surface roughness on plaque accumulation and peri-implant mucositis. *Clinical Oral Implants Research* 7 (3): 201–211.
- **96** Meier, R.M., Pfammatter, C., Zitzmann, N.U. et al. (2012). Surface quality after implantoplasty. *Schweizer Monatsschrift für Zahnmedizin* 122 (9): 714–724.
- **97** Eriksson, A.R. and Albrektsson, T. (1983). Temperature threshold levels for heat-induced bone tissue injury: a vital-microscopic study in the rabbit. *The Journal of Prosthetic Dentistry* 50 (1): 101–107.
- **98** Sharawy, M., Misch, C.E., Weller, N., and Tehemar, S. (2002). Heat generation during implant drilling: the significance of motor speed. *Journal of Oral and Maxillofacial Surgery* 60 (10): 1160–1169.
- **99** Schwarz, F., Sahm, N., Mihatovic, I. et al. (2011). Surgical therapy of advanced ligature-induced peri implantitis defects: cone-beam computed tomographic and histological analysis. *Journal of Clinical Periodontology* 38 (10): 939–949.
- **100** Nothdurft, F.P., Fontana, D., Ruppenthal, S. et al. (2015). Differential behavior of fibroblasts and epithelial cells on structured implant abutment materials: a comparison of materials and surface topographies. *Clinical Implant Dentistry and Related Research* 17 (6): 1237–1249.
- **101** Rupp, F., Scheideler, L., Olshanska, N. et al. (2006). Enhancing surface free energy and hydrophilicity through chemical modification of microstructured titanium implant surfaces. *Journal of Biomedical Materials Research. Part A* 76 (2): 323–334.
- **102** Toma, S., Lasserre, J., Brecx, M.C., and Nyssen-Behets, C. (2016). In vitro evaluation of peri-implantitis treatment modalities on Saos-2osteoblasts. *Clinical Oral Implants Research* 27 (9): 1085–1092.
- **103** Li, S., Chien, S., and Branemark, P.I. (1999). Heat shock-induced necrosis and apoptosis in osteoblasts. *Journal of Orthopaedic Research* 17 (6): 891–899.
- **104** Huh, J.B., Eckert, S.E., Ko, S.M., and Choi, Y.G. (2009). Heat transfer to the implant-bone interface during preparation of a zirconia/alumina abutment. *The International Journal of Oral & Maxillofacial Implants* 24 (4): 679–683.
- **105** Sharon, E., Shapira, L., Wilensky, A. et al. (2013). Efficiency and thermal changes during implantoplasty in relation to bur type. *Clinical Implant Dentistry and Related Research* 15 (2): 292–296.
- **106** de Souza Junior, J.M., Oliveira de Souza, J.G., Pereira Neto, A.L. et al. (2016). Analysis of effectiveness of

different rotational instruments in implantoplasty: an in vitro study. *Implant Dentistry* 25 (3): 341–347.

- **107** Schwarz, F., Mihatovic, I., Golubovic, V. et al. (2014). Immunohistochemical characteristics of regenerated bone after surgical therapy of advanced ligature-induced peri-implantitis defects. *Clinical Oral Investigations* 18 (6): 1679–1686.
- **108** Schliephake, H., Sicilia, A., Nawas, B.A. et al. (2018). Drugs and diseases: summary and consensus statements of group 1. The 5(th) EAO Consensus Conference 2018. *Clinical Oral Implants Research* 29 (Suppl 18): 93–99.
- **109** Beheshti Maal, M., Aanerod Ellingsen, S., Reseland, J.E., and Verket, A. (2020). Experimental implantoplasty outcomes correlate with fibroblast growth in vitro. *BMC Oral Health* 20 (1): 25.
- **110** Noronha Oliveira, M., Schunemann, W.V.H., Mathew, M.T. et al. (2018). Can degradation products released from dental implants affect peri-implant tissues? *Journal of Periodontal Research* 53 (1): 1–11.
- **111** Rashad, A., Sadr-Eshkevari, P., Weuster, M. et al. (2013). Material attrition and bone micromorphology after conventional and ultrasonic implant site preparation. *Clinical Oral Implants Research* 24 (Suppl A100): 110–114.
- **112** Tribst, J.P.M., Dal Piva, A.M.O., Shibli, J.A. et al. (2017). Influence of implantoplasty on stress distribution of exposed implants at different bone insertion levels. *Brazilian Oral Research* 31: e96.
- **113** Leitao-Almeida, B., Camps-Font, O., Correia, A. et al. (2020). Effect of crown to implant ratio and implantoplasty on the fracture resistance of narrow dental implants with marginal bone loss: an in vitro study. *BMC Oral Health* 20 (1): 329.
- **114** Bertl, K., Isidor, F., von Steyern, P.V., and Stavropoulos, A. (2021). Does implantoplasty affect the failure strength of narrow and regular diameter implants? A laboratory study. *Clinical Oral Investigations* 25 (4): 2203–2211.
- **115** Ogawa, T., Suzuki, T., Oishi, N. et al. (2011). Tactile sensation and occlusal loading condition of mandibular premolars and molars. *Odontology* 99 (2): 193–196.
- **116** Suarez, F., Monje, A., Galindo-Moreno, P., and Wang, H.L. (2013). Implant surface detoxification: a comprehensive review. *Implant Dentistry* 22 (5): 465–473.
- **117** de Waal, Y.C., Raghoebar, G.M., Meijer, H.J. et al. (2015). Implant decontamination with 2% chlorhexidine during surgical peri-implantitis treatment: a randomized, double-blind, controlled trial. *Clinical Oral Implants Research* 26 (9): 1015–1023.
- **118** Koldsland, O.C., Wohlfahrt, J.C., and Aass, A.M. (2018). Surgical treatment of peri-implantitis: prognostic indicators of short-term results. *Journal of Clinical Periodontology* 45 (1): 100–113.
- **119** Doornewaard, R., Jacquet, W., Cosyn, J., and De Bruyn, H. (2018). How do peri-implant biologic parameters

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correspond with implant survival and peri-implantitis? A critical review. *Clinical Oral Implants Research* 29 (Suppl 18): 100–123.

- **120** Monje, A., Pons, R., Amerio, E. et al. (2022). Resolution of peri-implantitis by means of implantoplasty as adjunct to surgical therapy: a retrospective study. *Journal of Periodontology* 93 (1): 110–122.
- **121** Pommer, B., Haas, R., Mailath-Pokorny, G. et al. (2016). Periimplantitis treatment: long-term comparison of laser decontamination and implantoplasty surgery. *Implant Dentistry* 25 (5): 646–649.
- **122** Romeo, E. and Storelli, S. (2012). Systematic review of the survival rate and the biological, technical, and aesthetic complications of fixed dental prostheses with cantilevers on implants reported in longitudinal studies with a mean of 5 years follow-up. *Clinical Oral Implants Research* 23: 39–49.
- **123** Englezos, E., Cosyn, J., Koole, S. et al. (2018). Resective treatment of peri-implantitis: clinical and radiographic outcomes after 2 years. *International Journal of Periodontics & Restorative Dentistry* 38 (5): 729–735.
- **124** Serino, G. and Turri, A. (2011). Outcome of surgical treatment of peri-implantitis: results from a 2-year prospective clinical study in humans. *Clinical Oral Implants Research* 22 (11): 1214–1220.
- **125** Ravida, A., Siqueira, R., Saleh, I. et al. (2020). Lack of clinical benefit of implantoplasty to improve implant survival rate. *Journal of Dental Research* 99 (12): 1348–1355.
- **126** Axelsson, P. and Lindhe, J. (1981). Effect of controlled oral hygiene procedures on caries and periodontal disease in adults. Results after 6 years. *Journal of Clinical Periodontology* 8 (3): 239–248.
- **127** Axelsson, P., Nystrom, B., and Lindhe, J. (2004). The long-term effect of a plaque control program on tooth mortality, caries and periodontal disease in adults. Results after 30 years of maintenance. *Journal of Clinical Periodontology* 31 (9): 749–757.
- **128** Berglundh, T., Zitzmann, N.U., and Donati, M. (2011). Are peri-implantitis lesions different from periodontitis lesions? *Journal of Clinical Periodontology* 38: 188–202.
- **129** Loe, H., Theilade, E., and Jensen, S.B. (1965). Experimental gingivitis in man. *Journal of Periodontology* 36: 177–187.
- **130** Costa, F.O., Takenaka-Martinez, S., Cota, L.O. et al. (2012). Peri-implant disease in subjects with and without preventive maintenance: a 5-year follow-up. *Journal of Clinical Periodontology* 39 (2): 173–181.
- **131** Roccuzzo, M., De Angelis, N., Bonino, L., and 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. *Clinical Oral Implants Research* 21 (5): 490–496.

- **132** Aguirre-Zorzano, L.A. et al. (2014). Prevalence of peri-implant inflammatory disease in patients with a history of periodontal disease who receive supportive periodontal therapy. *Clinical Oral Implants Research* 26: 1338–1344.
- **133** Serino, G. and Strom, C. (2009). Peri-implantitis in partially edentulous patients: association with inadequate plaque control. *Clinical Oral Implants Research* 20 (2): 169–174.
- **134** Froum, S.J., Froum, S.H., and Rosen, P.S. (2015). A regenerative approach to the successful treatment of peri-implantitis: a consecutive series of 170 implants in 100 patients with 2-to 10-year follow-up. *Int J Periodontics Restorative Dent* 35 (6): 857–863.
- **135** Monje, A., Aranda, L., Diaz, K.T. et al. (2016). Impact of maintenance therapy for the prevention of peri-implant diseases: a systematic review and meta-analysis. *Journal of Dental Research* 95 (4): 372–379.
- **136** Kelekis-Cholakis, A. and Rothney, J. (2019). Maintenance of implant patients: a narrative review. *Implant Dentistry* 28 (2): 161–172.
- **137** Lang, N.P. and Tonetti, M.S. (2003). Periodontal risk assessment (PRA) for patients in supportive periodontal therapy (SPT). *Oral Health & Preventive Dentistry* 1 (1) : 7-16.
- **138** Oliveira Costa, F., Miranda Cota, L.O., Pereira Lages, E.J. et al. (2011). Progression of periodontitis in a sample of regular and irregular compliers under maintenance therapy: a 3-year follow-up study. *Journal of Periodontology* 82 (9): 1279–1287.
- **139** Raghavendra, M., Koregol, A., and Bhola, S. (2009). Photodynamic therapy: a targeted therapy in periodontics. *Australian Dental Journal* 54 (Suppl 1): S102–S109.
- **140** Heitz-Mayfield, L.J. and Mombelli, A. (2014). The therapy of peri-implantitis: a systematic review. *The International Journal of Oral & Maxillofacial Implants* 29 (Suppl): 325–345.
- **141** Sahm, N., Becker, J., Santel, T., and Schwarz, F. (2011). Non-surgical treatment of peri-implantitis using an air-abrasive device or mechanical debridement and local application of chlorhexidine: a prospective, randomized, controlled clinical study. *Journal of Clinical Periodontology* 38 (9): 872–878.
- **142** Serino, G., Turri, A., and Lang, N.P. (2015). Maintenance therapy in patients following the surgical treatment of peri-implantitis: a 5-year follow-up study. *Clinical Oral Implants Research* 26 (8): 950–956.
- **143** Higgins, J.P., Altman, D.G., Gotzsche, P.C. et al. (2011). The cochrane collaboration's tool for assessing risk of bias in randomised trials. *BMJ* 343: d5928.

Non-surgical Antimicrobial Photodynamic and Photothermal Therapy in Treatment of Peri-implant Mucositis and Peri-implantitis

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Introduction

Peri-implant mucositis and peri-implantitis are pathological conditions affecting tissues surrounding dental implants. Many factors are contributing etiologically to the pathology of these diseases [1]. The major factor is considered to be microbial plaque (or microbial biofilm). Patients who do not follow daily oral hygiene routine and are not under regular maintenance therapy have been shown to be at a higher risk for developing peri-implant disease [2]. There are also secondary etiological and predisposing factors such as smoking, diabetes mellitus, radiation, genes associated with host response that contribute to peri implant pathology [2, 3].

The main goal of treatment of peri-implant diseases of microbial etiology is the (i) effective removal of microbial biofilm, (ii) disinfection of implant and prosthetic restoration surfaces, (iii) inflammation resolution and stabilization of marginal bone level, and (iv) regeneration of peri-implant tissues (if feasible) [4]. Treatment can be (i) surgical (resective and/or regenerative techniques), or (ii) non-surgical (mechanical removal of microbial biofilm using ultrasonic scaling, curettes, or air-polishing without raising a flap) [4]. Additional methods that have been studied include (i) systemic administration of antibiotics or application of antimicrobial mouthwashes, (ii) local application of antibiotic or antimicrobial gels in peri-implant pocket, (iii) mechanical or chemical alteration of implant surfaces, as well as (iv) application of dental lasers [5].

The efficacy of dental lasers in treating peri-implant diseases has been studied either as monotherapy or adjunctive therapy during i) disease control phase, ii) surgical phase (implant placement), or iii) irradiation phase of implant surfaces for disinfection or modification. Various types of lasers have been investigated: i) neodymium lasers (Nd:YAG, Neodymium-Doped Yttrium Aluminum Garnet, 1064nm), ii) Erbium lasers (Er:YAG, Erbium Yttrium Aluminum Garnet, 2940nm and Er,Cr:YSGG, Erbium, Chromium-Doped Yttrium Scandium Gallium Garnet, 2780 nm), iii) Carbon Dioxide lasers (CO₂, Carbon Dioxide, 10600nm), as well as iv) Diode lasers (810, 940, 980, and 1064nm) [4]. Low-level lasers have been studied in a specific type of therapy termed as "Photodynamic Therapy" or "Photothermal Therapy".

Photodynamic Therapy

Photodynamic therapy is a treatment modality that includes application of low-level laser light for activation of a non-toxic hydrophilic photosensitive substance, termed as photosensitizer, usually in an oxygen-rich environment. This method was discovered at the beginning of the twentieth century by Raab who reported that *Paramecium caudatum* could be killed after incubation with acridine hydrochloride or eosin and concomitant exposure to daylight (but not to the dark) [6]. At that period, therapy with local eosin and light was studied in skin carcinoma treatment for first time [7]. At the same time, the requirement for oxygen during this photosensitive reaction was reported and the term "photodynamic action" was introduced for first time a few years later by von Tappeiner [8]. Antimicrobial photodynamic therapy (aPDT) was first introduced by MacMillan and his colleagues in 1966, who applied a continuous wave gas laser (632nm) to activate Toluidine Blue Ο and killed bacterial microorganisms including bacteria and yeast [9]. Due to discovery of antibiotics and their bactericidal activity, PDT application was forgotten for many years, until resistance to antibiotics developed by many bacteria and multi-drug resistance has led to the increasing scientific interest and research on alternative antibacterial treatment modalities such as aPDT.

Photodynamic therapy has been applied as a method for treating conditions such as carcinoma and actinic keratosis until Wilson et al. reported its application for inactivation of oral and periodontopathogenic bacteria [10–12]. More specifically, *Streptococcus sanguis, Fusobacterium nucleatum, Porphyromonas gingivalis,* and *Actinobacillus actinomycetemcomitans* were reported to be killed by applying Toluidine Blue O, Azure B Chloride, or Methylene Blue and activating them by a He:Ne laser. The technique was effective in inactivation of these species not only in culture form but even when they were organized into biofilms. In a report from the same group of colleagues at the same period, subgingival microbial plaque samples from patients with chronic periodontitis were incubated with Toluidine Blue O and activated by He:Ne laser [13]. Photodynamic Therapy led to viability reduction of all different periodontopathogenic species of the samples (aerobes, anaerobes, black-pigmented anaerobes, *P. gingivalis*, *F. nucleatum*, and streptococci) (only a minor toxicity was caused by the dye alone). Due to its targeted activity against microorganisms, Photodynamic Therapy was also termed as Photodynamic Antimicrobial Chemotherapy (PACT), antimicrobial Photodynamic Therapy (aPDT), or Lethal Photosensitization [14, 15].

As it was mentioned above, the main principle of this treatment modality lies in the application of a photoactive dye which is termed as "photosensitizer." Photosensitizers, applied either locally, or been administered intravenously, consequently bind to target bacterial cells and are activated by light of specific wavelength in the presence of oxygen [16]. Theoretically, neither light of a specific wavelength nor photosensitizers alone can cause cytotoxic effects on cells. Lasers can transmit monochromatic, high intensity but low-level energy to the photosensitizers which can be highly effective for activation of photodynamic reaction.

After light activation, the photosensitizer is promoted from its stable ground state to an excited triplet state. Electrons from highly excited states can return to their ground states, and during this transition, light or heat emission can be observed [17]. The excited triplet state photosensitizers react with their substrate following two different pathways termed as Type I and Type II reactions, depending on the photosensitizer applied and the existing conditions [18–21]. In Type I reaction, electrons are transferred from the excited triplet state photosensitizer to organic substrate molecules within the cells. This leads to

production of other reactive agents (free radicals and radical ions) that react with molecular oxygen and produce reactive oxygen species such as hydroxyl radical, superoxide and hydrogen peroxide. These reactive oxygen species react with molecules inside bacterial cells and cause damage to them. Type II reaction includes the interaction of excited triplet-state photosensitizer with ground-state oxygen that leads to production of singlet oxygen ($^1\mathrm{O}_2$). Singlet oxygen can also interact with many molecules inside cells leading to their oxidation. Both reactions can eventually damage the cell wall and cytoplasmic membrane, leading to cell lysis and cell death by necrosis or apoptosis [22].

Singlet oxygen $({}^{1}O_{2})$ is considered to be the primary reactive agent of photodynamic reaction, so most of the effects inside microbial cells are mediated by Type II reaction [23]. Due to the short lifetime of singlet oxygen in cells, the radius of activity of singlet oxygen is also short. As a result, photodynamic reaction is limited to the region of photosensitizer application, and its effects are restricted to a small targeted region of activity, without affecting cells and tissues at a distance.

aPDT offers many advantages in comparison to surgical or non-surgical treatment modalities (mechanical instrumentation, local or systemic administration of antibiotics, etc.) [23]. When aPDT is performed, patients avoid undergoing the unpleasant experience of a surgical procedure. Furthermore, this treatment modality can be performed even in patients with compromised medical history. Due to application of low-level lasers, the energy and heat that is transferred to the implant surface is very low. Photosensitizers require less energy to become activated and inactivate pathogenic bacteria. As a result, the overall reaction does not cause any damage to the implant surface and neighboring healthy tissues (carbonization, cracking, or melting). Furthermore, aPDT presents a promising treatment method for overcoming limitations of classical mechanical instrumentation methods to decontaminate implant surfaces, even in difficult-to-access regions without raising a flap, and this effect can be completed in a very short time period which is controllable in a light-dependent manner (wavelength, power, and time of irradiation), and without causing trauma to the neighboring tissues. In contrast to antibiotics, aPDT is advantageous as it overcomes one major disadvantage of antibiotics administration (as it has been reported for periodontal microflora) and does not lead to development of antibiotic-resistant strains [24]. This is due to non-target specific mechanism of action of aPDT which is based on the production of highly reactive cytotoxic molecules, as mentioned above. Due to the target-specific activity of antibiotics, many of them would be required to inactivate different species of bacteria in microbial biofilm. However, aPDT can inactivate different microorganisms non-specifically due to its mechanism of action. Furthermore, the effect of laser irradiation on the photosensitizer can be diffused to the inner layers of microbial biofilm, an effect that is difficult to achieve by antibiotics [24].

Many photosensitizers have been applied in aPDT, in general, and more specifically in treatment of periodontal and peri-implant diseases. Ideally, they should have the following features: (i) absence of toxicity, (ii) high binding affinity, specificity and absorption for target diseased cells/ tissues and low for healthy cells/tissues, (iii) quick accumulation at the target cells/tissues and short half-life, in order to be dispersed at a specific radius from the desired target region of tissue and not affect non-desired tissue regions, (iv) have a broad spectrum of activity, (v) should be activated by a specific wavelength of light and be able to produce a significant quantity of cytotoxic molecules [25].

Since the first in vivo human study published in 2001 by Doertbudak et al. (that applied aPDT after raising a flap), many reports have been published in PubMed database of the US National Library of Medicine so far that have applied non-surgical aPDT in peri-implant disease treatment (Table 15.1) [14]. The most common photosensitizers applied in these studies are Toluidine Blue O [Tolonium Chloride: (7-amino-8-methyl-phenothiazin-3-ylidene)-dim ethyl-ammonium $(C_{15}H_{16}N_3S_+)$, Methylene Blue [3,7-bis(dimethyl-amino) phenazathionium chloride tetramethylthionine chloride $(C_{16}H_{18}N_3C_1S)$ or phenothiazine-5ium, 3,7-bis(dimethylamino)-chloride], and Phenothiazine Chloride (Table 15.1).

Toluidine Blue has a blue–violet color, while MB in its oxidized form is blue and becomes colorless in its reduced state. Both photosensitizers are similar in their features and are effective against periodontopathogenic bacteria (both Gram+ and Gram−) [59]. MB is a cationic dye with its absorption peak at light of 660nm wavelength, while Toluidine blue is also cationic with its absorption peak at light of 635nm wavelength for inactivating bacterial cells. Phenothiazine Chloride is also a blue photosensitizer (its main ingredient isToluidine blue) and its maximum absorbance is at 670nm wavelength of light. In seven studies published so farin PubMed database of the US National Library of Medicine, aPDT with MB was performed together with mechanical debridement or glycine air-polishing (Table 15.1). MB was activated by (i) 630nm, (ii) 635nm, (iii) 660nm, or (d) 670nm wavelength of light. In six out of seven studies, aPDT resulted in improvement of clinical and/or microbiological and immunological parameters in these studies. The only study that did not report any additive positive result after aPDT with MB was that done by Alresheedi and Alazmi [55]. In five studies published so far in PubMed database of the US National Library of Medicine, aPDT with Toluidine Blue was performed in combination

with scaling and/or mechanical debridement and/or subgingival glycine air-polishing and/or professionally administered plaque removal and/or home-based administration of probiotics, and/or irrigation of peri-implant pocket with saline solution. In these studies, Toluidine Blue was activated by (i) LED, (ii) 630 or (iii) 635 wavelength of light. In all of these studies, aPDT with Toluidine Blue resulted in improvement of clinical parameters examined in each study. Finally, nine studies have been published so far in PubMed database of the US National Library of Medicine, reporting aPDT with phenothiazine chloride. In all of these studies, phenothiazine chloride was activated by 660nm wavelength of light. APDT was performed as sole treatment or in combination with air-polishing and/or mechanical debridement and/or scaling and root planing.

An overview of all of these studies (samples, methodology followed, results) investigating the effectiveness of non-surgical photodynamic therapy in treatment of peri implant diseases published in PubMed database of the US National Library of Medicine so far are presented in Table 15.1. A brief comment is that in many studies, aPDT either as sole or adjunctive treatment leads to short-term improvement (three to six months) of clinical, microbiological, and immunological parameters in patients with peri-implant disease in comparison to control groups that have undergone only classical treatment methods (e.g. mechanical debridement, air-polishing, local, or systemic administration of antibiotics). In some of the studies, no additional benefit from aPDT in any of the parameters measured was reported. There are some studies that presented some limitations (no control groups). APDT is a technique, that as mentioned above, due to its advantage of not causing resistant strain growth, could be repeated many times to achieve better results if possible without having adverse effects (in some studies, repeated application of therapy in regular sessions after initial therapy has been reported). There are some studies in which aPDT has reported improvement levels of Hb1Ac in diabetic patients with peri-implant disease, while in others no additional benefit was observed. All these results are shown in Table 15.1. Although in some of the studies, no details of methodology are provided either in abstract or in full text, it is obvious that there has been increasing interest in ongoing research around application of this treatment in therapy of peri-implant mucositis and peri-implantitis.

Photothermal Therapy

In recent years, another photosensitizer reported to be applied in a more contemporary version of aPDT is Indocyanine Green (Table 15.2). This photosensitizer **Table 15.1** In vivo human studies of non-surgical antimicrobial photodynamic therapy in treatment of peri-implant diseases.

intervals

(*Continued*)

Table 15.1 (Continued)

(*Continued*)

Table 15.1 (Continued)

Table 15.2 In vivo human studies of non-surgical photothermal therapy in treatment of peri-implant diseases.

is anionic, hydrophilic, and lipophilic, and shows its absorption peak at 810 nm. This wavelength is different from wavelengths applied with the other photosensitizers in aPDT (Methylene Blue, Toluidine Blue, and Phenothiazine Chloride), has a greater penetration depth compared to them, which is favorable in deep periodontal and peri-implant pockets. In contrast with photosensitizers used in aPDT, Indocyanine Green's main effect derives primarily from its molecules' vibration and heat release inside target cells/tissues, and at a secondary degree through production of cytotoxic reactive agents (thought to be 80% photothermal and 20% photochemical) [23, 63]. Due to its different mechanism of action, this type of aPDT is termed as "Photothermal Therapy." Photothermal therapy with Indocyanine Green was initially applied in treatment of acne bulgaris [64]. Indocyanine green has been shown to be selectively absorbed by periodontopathogenic bacteria (*P. gingivalis* and *A. actinomycetemcomitans*) [63].

Photothermal therapy includes application (with a syringe with a blunt tip) of indocyanine green (mixed according to manufacturer's instructions) in peri-implant pocket before removal of microbial biofilm from implant surface and peri-implant tissues (Figures 15.1–15.3). This is performed to avoid bleeding that would lead to absorption of laser energy by the hemoglobin, and consequently lead to reduced absorption of light by the bacterial target cells. At first stage, a specific handpiece for transgingival irradiation is irradiating at a vertical direction from a distance of ~15 mm on the outer surface of peri-implant tissues (Figure 15.4). The power that is used is 500mW and the duration of irradiation lasts from 5 to 15 seconds per

Figures 15.2 Application of Indocyanine Green in peri-implant pocket buccally and palatally.

Figures 15.3 Application of Indocyanine Green in peri-implant pocket buccally and palatally.

Figure 15.1 Initial situation of an implant in position 21 with peri-implantitis.

Figure 15.4 Transgingival irradiation with a specific therapy handpiece provided by the manufacturer.

Figure 15.5 The bulb tip of the optical fiber is applied for 360° intra-pocket irradiation.

Figure 15.7 Intra-pocket irradiation with the bare tip of the optical fiber.

Figure 15.6 Intra-pocket irradiation with the bulb tip of the optical fiber.

targeted area (buccally and lingually). The photosensitizer is activated transgingivally. At the second stage, a $300 \mu m$ optic fiber with a special tip (bulb fiber) is used (Figure 15.5). This fiber tip enables irradiation inside peri implant pocket at a 360° angle (Figure 15.6). The power that is used is 200mW and the duration of irradiation lasts from 5 to 15 seconds per targeted area (buccally and lingually). The optic fiber is placed in the peri-implant pocket and circular movements are performed inside it in all directions. At the third stage, the same $300 \mu m$ – optic fiber is used, but with a flat tip (bare fiber) (Figure 15.7). The power that is implemented is 200mW and the duration of irradiation lasts from 5 to 15 seconds per targeted area

(buccally and lingually). The optic fiber is placed into the pocket, against its external wall, and circular movements are performed to achieve de-epithelialization and removal of the granulation tissue from the infected peri-implant pocket to an extent that is determined by the clinician. It is recommended that microbial deposits can then be removed from the implant surface during the same session. Two studies investigating the effectiveness of non surgical photothermal therapy in treatment of peri-implant diseases have been published so far in PubMed database of the US National Library of Medicine (Table 15.2). Photothermal therapy with Indocyanine Green was applied in combination with mechanical debridement, and/or scaling and root planing, and/or air-polishing with sodium bicarbonate powder. In both studies, photothermal therapy resulted in additional improvement in some of the clinical and microbiological parameters examined (three to six months). In a recent study, a home-based protocol of photothermal therapy was examined for first time [62]. Researchers reported that they provided their patients with peri-implant disease, with a special kit of ICG mouth rinse and an apparatus for radiant exposure to a combination of 810 and 405nm light at very low levels of energy for four weeks before performing mechanical debridement. At four weeks, improvement of clinical and immunological parameters was reported (however, there is no control group in the study).

The following clinical cases present the application of photodynamic therapy in the treatment of peri-implantitis to maintain health and stability of dental implants (Figures 15.8–15.13).

Figure 15.8 Radiographic images of implant in position 21 before non-surgical therapy and during follow-ups of a 63-year-old woman, at maintenance phase of periodontal therapy, with hypothyroidism. Implants were placed at sites #21, 36, 37, 26, 27 (at the age of 53). Seven years after implant placement, severe peri-implantitis was identified (radiographic bone loss, suppuration, and bleeding on probing). Implants at sites 36, 37, 26, 27 were extracted. Sessions of Ultrasonic Scaling, Supra- and Sub-gingival Air-Polishing with Glycine or Erythritol Powder, antimicrobial Photothermal Therapy with Indocyanine Green (810 nm diode laser), Local application of Blue®m gel or 2% w/w Minocycline gel or 0.3% Chlorhexidine gel + 1% Hyaluronic Acid, were performed in regular three month-recalls. Peri-implant probing depth has been reduced, suppuration and bleeding on probing have resolved and radiographic bone levels are presented. (a) Initial situation (seven years after implant placement). (b) one-year recall. (c) two-year diastema needed recall. (d) three-year recall.

Figure 15.9 A 52-year-old man, at maintenance phase of periodontal therapy, with free medical history. Peri-implantitis was identified (radiographic bone loss, bleeding on probing). Sessions of Ultrasonic Scaling, Supra-gingival Air-Polishing with Glycine or Erythritol Powder, antimicrobial Photothermal Therapy with Indocyanine Green (810nm diode laser), were performed in regular three month-recalls. Peri-implant probing depth has been reduced, bleeding on probing has resolved and radiographic bone levels are presented. (a) Initial situation. (b) one-year recall. (c) two-year recall. (d) two-year recall. Clinical situation.

Figure 15.9 (Continued)

Figure 15.10 About 73-year-old woman, at maintenance phase of periodontal therapy, with free medical history. Peri-implantitis was identified (radiographic bone loss, bleeding on probing). Clinically a vertical fracture of implant at site 37 was identified. Sessions of Ultrasonic Scaling, Supra-gingival Air-Polishing with Glycine or Erythritol Powder, antimicrobial Photothermal Therapy with Indocyanine Green (810nm diode laser), were performed in regular three month-recalls. Peri-implant probing depth has been reduced, minor bleeding on probing has been observed and radiographic bone levels are presented. (a) Initial situation. (b) one-year recall. (c) two-year recall. (d) two-year recall. Clinical situation.

(a)

Figure 15.11 A 62-year-old woman, at maintenance phase of periodontal therapy, with free medical history. Extraction of tooth 46 was performed due to tooth fracture, and an implant was placed (at the age of 51). Seven years after implant placement, severe peri-implantitis was identified (radiographic bone loss, suppuration, and bleeding on probing) due to inadequate compliance of patient with regular periodontal recalls. Sessions of Ultrasonic Scaling, Supra- and Sub-gingival Air-Polishing with Glycine or Erythritol Powder, antimicrobial Photothermal Therapy with Indocyanine Green (810nm diode laser), Local application of Blue®m gel or 2% w/w Minocycline gel or 0.3% Chlorhexidine gel + 1% Hyaluronic Acid, were performed in regular three-month recalls. Peri-implant probing depth has been reduced, suppuration and bleeding on probing have resolved and radiographic bone levels are presented. (a) Initial situation (seven years after implant placement). (b) two-year recall. (c) four-year recall.

Figure 15.12 A 71-year-old woman, with hyperlipidemia. No previous periodontal treatment. Three implants were placed at sites #44, #45, #46 (at the age of 59). Nine years after implant placement, peri-implantitis was identified (radiographic bone loss, bleeding on probing). Sessions of Ultrasonic Scaling, Supra-gingival Air-Polishing with Glycine or Erythritol Powder, antimicrobial Photothermal Therapy with Indocyanine Green (810nm diode laser), Local application of Blue®m gel or 2% w/w Minocycline gel, were performed in regular three month-recalls. Peri-implant probing depth has been reduced, bleeding on probing has resolved and radiographic bone levels are presented. (a) Initial situation (nine years after implant placement). (b) one-year recall. (c) three-year diastema needed recall.

Figure 15.13 A 77-year-old woman, with hypothyroidism and hyperlipidemia. No previous periodontal treatment. Among others, 2 implants were placed at sites #12, #13 (at the age of 65). Nine years after implant placement, severe peri-implantitis was identified (radiographic bone loss, suppuration, and bleeding on probing). Sessions of Ultrasonic Scaling, Supra- and Sub-gingival Air-Polishing with Glycine or Erythritol Powder, antimicrobial Photothermal Therapy with Indocyanine Green (810nm diode laser), were performed in regular three-month recalls. Peri-implant probing depth has been reduced, suppuration has resolved, minor bleeding on probing has been observed. Radiographic bone levels are presented. Surgical therapy of peri-implantitis has not been performed due to patient's decision. (a) Initial situation (nine years after implant placement). (b) one-year recall. (c) three-year recall.

Abbreviations

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References

- **1** Salvi, G.E., Cosgarea, R., Sculean, A. et al. (2019). Prevalence of periimplant diseases. *Implant Dentistry* 28 (2): 100–102.
- **2** Berglundh, T., Armitage, G., Araujo, M.G. et al. (2018). 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 Clinical Periodontology* 45 (Suppl 20): S286–S291.
- **3** Fourmousis, I. and Vlachos, M. (2019). Genetic risk factors for the development of periimplantitis. *Implant Dentistry* 28 (2): 103–114.
- **4** Romanos, G.E., Javed, F., Delgado-Ruiz, R.A. et al. (2015). Peri-implant diseases: a review of treatment interventions. *Dental Clinics of North America* 59 (1): 157–178.
- **5** Renvert, S., Roos-Jansaker, A.M., Claffey, N. et al. (2008). Non-surgical treatment of peri-implant mucositis and peri-implantitis: a literature review. *Journal of Clinical Periodontology* 35 (8 Suppl): 305–315.
- **6** Raab, O. (1900). Ueber die wirkung fluoreszierenden stoffen. *Infusoria Z Biol* 39: 524.
- **7** Jesionek, A. and Von Tappeiner, H. (1905). Zur behandlung der hautcarcinome mit fluorescierenden stoffen. *Deutsches Archiv für Klinische Medizin* 85: 223–239.
- **8** von Tappeiner, H. and Jodlbauer, A. (1907). *Die sensibilisierende Wirkung fluorescierender Substanzen: gesammelte Untersuchungen über die photodynamische Erscheinung*. Vogel.
- **9** Macmillan, J.D., Maxwell, W.A., Chichester, C.O. et al. (1966). Lethal photosensitization of microorganisms with light from a continuous-wave gas laser. *Photochemistry and Photobiology* 5 (7): 555–565.
- **10** Dobson, J. and Wilson, M. (1992). Sensitization of oral bacteria in biofilms to killing by light from a low-power laser. *Archives of Oral Biology* 37 (11): 883–887.
- **11** Wilson, M., Dobson, J., Harvey, W. et al. (1992). Sensitization of oral bacteria to killing by low-power laser radiation. *Current Microbiology* 25 (2): 77–81.
- **12** Wilson, M., Dobson, J., Sarkar, S. et al. (1993). Sensitization of periodontopathogenic bacteria to killing by light from a low-power laser. *Oral Microbiology and Immunology* 8 (3): 182–187.
- **13** Sarkar, S. and Wilson, M. (1993). Lethal photosensitization of bacteria in subgingival plaque from patients with

TNF-α Tumor Necrosis Factor-α

chronic periodontitis. *Journal of Periodontal Research* 28 (3): 204–210.

- **14** Dortbudak, O., Haas, R., Bernhart, T. et al. (2001). Lethal photosensitization for decontamination of implant surfaces in the treatment of peri-implantitis. *Clinical Oral Implants Research* 12 (2): 104–108.
- **15** Wainwright, M. (1998). Photodynamic antimicrobial chemotherapy (PACT). *The Journal of Antimicrobial Chemotherapy* 42 (1): 13–28.
- **16** Sharman, W.M., Allen, C.M., Van Lier, J.E. et al. (1999). Photodynamic therapeutics: basic principles and clinical applications. *Drug Discovery Today* 4 (11): 507–517.
- **17** Maisch, T. (2007). Anti-microbial photodynamic therapy: useful in the future? *Lasers in Medical Science* 22 (2): 83–91.
- **18** Foote, C.S. (1987). Type I and type II mechanisms of photodynamic action. In: *Light-Activated Pesticides*, vol. 339, 22–38. American Chemical Society.
- **19** Foote, C.S. (1991). Definition of type I and type II photosensitized oxidation. *Photochemistry and Photobiology* 54 (5): 659–659.
- **20** Gollnick, K. (1968). Type II photosensitized oxygenation reactions. In: *Oxidation of Organic Compounds*, vol. 77, 78–101. American Chemical Society.
- **21** Schenck, G.O. (1963). Photosensitization. *Industrial and Engineering Chemistry* 55 (6): 40–43.
- **22** Buytaert, E., Dewaele, M., Agostinis, P. et al. (2007). Molecular effectors of multiple cell death pathways initiated by photodynamic therapy. *Biochimica et Biophysica Acta* 1776 (1): 86–107.
- **23** Takasaki, A.A., Aoki, A., Mizutani, K. et al. (2009). Application of antimicrobial photodynamic therapy in periodontal and peri-implant diseases. *Periodontology 2000* 51: 109–140.
- **24** Walker, C.B. (1996). The acquisition of antibiotic resistance in the periodontal microflora. *Periodontology 2000* 10: 79–88.
- **25** Jori, G., Fabris, C., Soncin, M. et al. (2006). Photodynamic therapy in the treatment of microbial infections: basic principles and perspective applications. *Lasers in Surgery and Medicine* 38 (5): 468–481.
- **26** Deppe, H., Mucke, T., Wagenpfeil, S. et al. (2013). Nonsurgical antimicrobial photodynamic therapy in moderate vs severe peri-implant defects: a clinical pilot study. *Quintessence International* 44 (8): 609–618.
- **27** Schar, D., Ramseier, C.A., Eick, S. et al. (2013). Anti infective therapy of peri-implantitis with adjunctive local drug delivery or photodynamic therapy: six-month outcomes of a prospective randomized clinical trial. *Clinical Oral Implants Research* 24 (1): 104–110.
- **28** Thierbach, R. and Eger, T. (2013). Clinical outcome of a nonsurgical and surgical treatment protocol in different types of peri-implantitis: a case series. *Quintessence International* 44 (2): 137–148.
- **29** Bassetti, M., Schar, D., Wicki, B. et al. (2014). Anti infective therapy of peri-implantitis with adjunctive local drug delivery or photodynamic therapy: 12-month outcomes of a randomized controlled clinical trial. *Clinical Oral Implants Research* 25 (3): 279–287.
- **30** Al Amri, M.D., Kellesarian, S.V., Ahmed, A. et al. (2016). Efficacy of periimplant mechanical debridement with and without adjunct antimicrobial photodynamic therapy in patients with type 2 diabetes mellitus. *Photodiagnosis and Photodynamic Therapy* 14: 166–169.
- **31** Javed, F., Abduljabbar, T., Carranza, G. et al. (2016). Efficacy of periimplant mechanical debridement with and without adjunct antimicrobial photodynamic therapy in the treatment of periimplant diseases among cigarette smokers and non-smokers. *Photodiagnosis and Photodynamic Therapy* 16: 85–89.
- **32** Karimi, M.R., Hasani, A., Khosroshahian, S. et al. (2016). Efficacy of antimicrobial photodynamic therapy as an adjunctive to mechanical debridement in the treatment of peri-implant diseases: a randomized controlled clinical trial. *Journal of Lasers in Medical Sciences* 7 (3): 139–145.
- **33** Romeo, U., Nardi, G.M., Libotte, F. et al. (2016). The antimicrobial photodynamic therapy in the treatment of peri-implantitis. *International Journal of Dentistry* 2016: 7692387.
- **34** Abduljabbar, T. (2017). Effect of mechanical debridement with adjunct antimicrobial photodynamic therapy in the treatment of peri-implant diseases in type-2 diabetic smokers and non-smokers. *Photodiagnosis and Photodynamic Therapy* 17: 111–114.
- **35** Abduljabbar, T. (2017). Effect of mechanical debridement with and without adjunct antimicrobial photodynamic therapy in the treatment of peri-implant diseases in prediabetic patients. *Photodiagnosis and Photodynamic Therapy* 17: 9–12.
- **36** Al-Sowygh, Z.H. (2017). Efficacy of periimplant mechanical curettage with and without adjunct antimicrobial photodynamic therapy in smokeless tobacco product users. *Photodiagnosis and Photodynamic Therapy* 18: 260–263.
- **37** Javed, F., BinShabaib, M.S., Alharthi, S.S. et al. (2017). Role of mechanical curettage with and without adjunct antimicrobial photodynamic therapy in the treatment of

peri-implant mucositis in cigarette smokers: a randomized controlled clinical trial. *Photodiagnosis and Photodynamic Therapy* 18: 331–334.

- **38** Mongardini, C., Pilloni, A., Farina, R. et al. (2017). Adjunctive efficacy of probiotics in the treatment of experimental peri-implant mucositis with mechanical and photodynamic therapy: a randomized, cross-over clinical trial. *Journal of Clinical Periodontology* 44 (4): 410–417.
- **39** Al Rifaiy, M.Q., Qutub, O.A., Alasqah, M.N. et al. (2018). Effectiveness of adjunctive antimicrobial photodynamic therapy in reducing peri-implant inflammatory response in individuals vaping electronic cigarettes: a randomized controlled clinical trial. *Photodiagnosis and Photodynamic Therapy* 22: 132–136.
- **40** Zeza, B., Farina, R., Pilloni, A. et al. (2018). Clinical outcomes of experimental gingivitis and peri-implant mucositis treatment with professionally administered plaque removal and photodynamic therapy. *International Journal of Dental Hygiene* 16 (2): e58–e64.
- **41** Alqahtani, F., Alqhtani, N., Alkhtani, F. et al. (2019). Efficacy of mechanical debridement with and without adjunct antimicrobial photodynamic therapy in the treatment of peri-implantitis among moderate cigarette smokers and waterpipe-users. *Photodiagnosis and Photodynamic Therapy* 28: 153–158.
- **42** Wang, H., Li, W., Zhang, D. et al. (2019). Adjunctive photodynamic therapy improves the outcomes of peri-implantitis: a randomized controlled trial. *Australian Dental Journal* 64 (3): 256–262.
- **43** Ahmed, P., Bukhari, I.A., Albaijan, R. et al. (2020). The effectiveness of photodynamic and antibiotic gel therapy as an adjunct to mechanical debridement in the treatment of peri-implantitis among diabetic patients. *Photodiagnosis and Photodynamic Therapy* 32: 102077.
- **44** Al Deeb, M., Alresayes, S., Mokeem, S.A. et al. (2020). Clinical and immunological peri-implant parameters among cigarette and electronic smoking patients treated with photochemotherapy: a randomized controlled clinical trial. *Photodiagnosis and Photodynamic Therapy* 31: 101800.
- **45** Al Deeb, M., Alresayes, S., Mokeem, S.A. et al. (2020). Clinical peri-implant health and biological bone marker levels in tobacco users treated with photodynamic therapy. *Photodiagnosis and Photodynamic Therapy* 31: 101821.
- **46** Al Hafez, A.S.S., Ingle, N., Tashery, H.M. et al. (2020). Effectiveness of mechanical debridement with and without adjunct antimicrobial photodynamic for treating peri-implant mucositis among prediabetic cigarette smokers and non-smokers. *Photodiagnosis and Photodynamic Therapy* 31: 101912.

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- **47** Almohareb, T., Alhamoudi, N., Al Deeb, M. et al. (2020). Clinical efficacy of photodynamic therapy as an adjunct to mechanical debridement in the treatment of per implantitis with abscess. *Photodiagnosis and Photodynamic Therapy* 30: 101750.
- **48** Alresayes, S., Al Deeb, M., Mokeem, S.A. et al. (2020). Influence of body fat in patients with dental implant rehabilitation treated with adjunctive photodynamic therapy. *Photodiagnosis and Photodynamic Therapy* 31: 101831.
- **49** Deeb, M.A., Alsahhaf, A., Alhamoudi, N. et al. (2020). Clinical and microbiological outcomes of photodynamic and systemic antimicrobial therapy in smokers with peri-implant inflammation. *Photodiagnosis and Photodynamic Therapy* 29: 101587.
- **50** Ohba, S., Sato, M., Noda, S. et al. (2020). Assessment of safety and efficacy of antimicrobial photodynamic therapy for peri-implant disease. *Photodiagnosis and Photodynamic Therapy* 31: 101936.
- **51** Al-Askar, M.H., Abdullatif, F.A., Alshihri, A.A. et al. (2022). Comparison of photobiomodulation and photodynamic therapy as adjuncts to mechanical debridement for the treatment of peri-implantitis. *Technology and Health Care* 30 (2): 389–398.
- **52** Alasqah, M.N. (2022). Influence of adjunctive non surgical periimplant therapy on clinical and salivary cytokine profile in obese patients. *Photodiagnosis and Photodynamic Therapy* 37: 102721.
- **53** Al-Hamoudi, N., Alali, Y., Al-Aali, K. et al. (2022). Peri implant parameters and bone metabolic markers among water-pipe users treated with photodynamic therapy. *Photodiagnosis and Photodynamic Therapy* 37: 102655.
- **54** Alqutub, M.N. (2022). Peri-implant parameters and cytokine profile among peri-implant disease patients treated with Er Cr YSGG laser and PDT. *Photodiagnosis and Photodynamic Therapy* 37: 102641.
- **55** Alresheedi, B. and Alazmi, S. (2022). Disinfection of implant abutment connection using antimicrobial photodynamic therapy and 0.2% chlorhexidine gel applications immediately before prosthesis delivery: clinical and radiographic status at 1-year of follow-up. *Photodiagnosis and Photodynamic Therapy* 38: 102790.
- **56** Shetty, B., Ali, D., Ahmed, S. et al. (2022). Role of antimicrobial photodynamic therapy in reducing

subgingival oral yeasts colonization in patients with peri-implant mucositis. *Photodiagnosis and Photodynamic Therapy* 38: 102803.

- **57** ALHarthi, S.S., Alamry, N.Z., MS, B.S. et al. (2022). Effect of multiple sessions of photodynamic therapy on bone regeneration around dental implants among patients with periimplantitis. *Photodiagnosis and Photodynamic Therapy* 37: 102612.
- **58** ALHarthi, S.S., Divakar, D.D., Alwahibi, A. et al. (2022). Effect of mechanical instrumentation with adjunct photodynamic therapy on salivary TNFalpha levels and clinical periodontal and peri-implant status in patients with depression: a randomized controlled trial. *Photodiagnosis and Photodynamic Therapy* 40: 103042.
- **59** Usacheva, M.N., Teichert, M.C., Biel, M.A. et al. (2001). Comparison of the methylene blue and toluidine blue photobactericidal efficacy against gram-positive and gram-negative microorganisms. *Lasers in Surgery and Medicine* 29 (2): 165–173.
- **60** Birang, E., Talebi Ardekani, M.R., Rajabzadeh, M. et al. (2017). Evaluation of effectiveness of photodynamic therapy with low-level diode laser in nonsurgical treatment of peri-implantitis. *Journal of Lasers in Medical Sciences* 8 (3): 136–142.
- **61** Labban, N., Shibani, N.A., Al-Kattan, R. et al. (2021). Clinical, bacterial, and inflammatory outcomes of indocyanine green-mediated photodynamic therapy for treating periimplantitis among diabetic patients: a randomized controlled clinical trial. *Photodiagnosis and Photodynamic Therapy* 35: 102350.
- **62** Lahteenmaki, H., Patila, T., Räisänen, I.T. et al. (2022). Repeated home-applied dual-light antibacterial photodynamic therapy can reduce plaque burden, inflammation, and aMMP-8 in peri-implant disease-a pilot study. *Current Issues in Molecular Biology* 44 (3): 1273–1283.
- **63** Boehm, T.K. and Ciancio, S.G. (2011). Diode laser activated indocyanine green selectively kills bacteria. *Journal of the International Academy of Periodontology* 13 (2): 58–63.
- **64** Genina, E.A., Bashkatov, A.N., Simonenko, G.V. et al. (2004). Low-intensity indocyanine-green laser phototherapy of acne vulgaris: pilot study. *Journal of Biomedical Optics* 9 (4): 828–834.

Removal of Implants: Guidelines of Explantation Techniques

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Introduction

Implants are generally a successful and well-established treatment for tooth replacement. However, like everything in dentistry, things can and will fail and clinicians placing implants need to be well versed in algorithms of diagnostic assessment [1]. Profound knowledge in therapeutic interventions in order to save an ailing implant is equally important as skillsets in procedures where implants have not succeeded and are deemed to fail. The consequences of implant removal jeopardize the clinician's efforts to accomplish satisfactory function and esthetics. For the patient, this usually involves further costs and additional procedures.

The current literature provides ample data on the high success rates of dental implant treatment, which range from 90 to 97% [2–5]. But over time, the percentage of dental implants that fail increases. Clinicians must be knowledgeable in different methods regarding removal of dental implants [6–8] on the basis of several clinical and anatomical factors and the patient's desire for reimplantation in failed implant site. Regardless of the reason for explantation, being able to safely remove an osseointegrated implant is imperative.

Most late-failing implants are not mobile and remain at least partially osseointegrated in the apical aspect. The attempt to remove the implants can therefore still be very challenging; the explantation might be invasive, and neighbor teeth or vital structures can also be potentially harmed [9].

Once the decision for the removal of an implant has been made, the selection of the appropriate removal technique should be addressed. The selected option should be fast, as minimally traumatic as possible and cost-effective for patient and dentist. Because the implant bed configuration is of outmost importance to a stable implant placement,

the expected morphology of the remaining defect should also guide the choice over the method of the implant removal.

Multiple explantation techniques have been reported in the literature with variable success rates. But thus far, very little is known regarding the different aspects of each technique with respect to benefits, limitations, and overall success rates. Moreover, universal explantation protocols with clear indications and guidelines are still lacking.

Since studies on the potential advantages and disadvantages of different removal techniques are still scarce, the basic techniques are summarized according to the available literature. Ideally, the explantation procedure should be followed either by the installation of another implant or by guided bone regeneration (GBR), or both procedures during the same session, when indicated.

Overview of Removal Techniques and Factors for Decision-making

Implant removal is quite different from extracting a tooth. Great caution has to be exercised not to remove a buccal or lingual plate during explanation. Therefore, numerous techniques were identified as seen in Figure 16.1:

Factors for Decision-making

Proper planning is fundamental in order to minimize treatment time, side effects, and costs. The quantity of periimplant residual bone, the proximity of vital anatomical structures, and the implant's design should be evaluated. Maximal preservation of bone and the feasibility of future implant placement should be prioritized. Clinical and anatomical factors that may govern the explantation protocol (Table 16.1) also have to consider the retrievability of the

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Table 16.1 Clinical and anatomical factors that determine the explantation protocol.

prosthetic suprastructures. Their complete destruction may sometimes be required in order to access the implant's inner structure. Broken screws or damaged abutments could also complicate implant removal and limit the choice between different explantation techniques.

Removal is easier when the implant driver connects directly to the internal connector. Therefore, an implant with an internal connection is much easier to remove than one with an external hex or spline connection because the internal connection is usually more rigid [9].

However, complications may occur when removing an implant with an internal "trilob" connections using the counter-torque ratchet. The neck portion of this type of implant system, especially those with smaller diameters (i.e. 3.5mm), has been observed to fracture during removal procedure if the reverse torque is more than 45Ncm [9].

Figure 16.1 Overview of Explantation

techniques.

Increased thread depth – which is the distance between the outer diameter and the implant body – is another characteristic of overall implant geometry, which can make implant removal more difficult.

A tapered implant with the same platform diameter as a straight-walled implant has a decreasing outer diameter because of the taper. Thus, the thread depth decreases toward the apical region. The tapered, threaded implant has less implant-to-bone contact in the apical region and is, therefore, easier to remove with a more conservative technique.

Reasons for Implant Removal

There are multiple reasons identified in the literature for removal of implants and the prostheses retained to these implants (Figure 16.2). Some of these, but not limited to, are broadly categorized into the following groups:

- 1) Mobility of the implant
- 2) Extensive crestal bone loss
- 3) Chronic pain, including sinus problems, close proximity to the canal with neurological disturbances or nerve damage
Figure 16.2 Reasons for explantation as reported in 16 studies according to Roy et al. [2]. (a) Most common reasons for implant removals as pie chart, demonstrating that peri-implantitis is by far the most common reason. (b) Most common reasons for implant removals in percent (%) as table. *Source:* Adapted from Roy et al. [2].

(b)

- 4) Advanced peri-implantitis
- 5) Fractured implant
- 6) Malpositioned and unrestorable implant

There is also an established association between biocorrosion, presence of titanium particles (titanium intolerance) and other different metals alloys, and peri-implant aseptic bone loosening leading to a failure of implant osseointegration [10, 11]. Since no metal or alloy is completely .inert in vivo, the possible detrimental immunological consequences of its by-products are contributing factors of clinical implant failure requiring removal of these fixtures [10, 11].

Irrespective of the reasons mentioned above: if radiographically, an implant has a radiolucent line along the entire implant surface, the implant is tender to percussion and/or mobile, the only option is removal of the implant.

Implant Removal Techniques

Non-bone Removal Techniques

Tooth Extraction Set

Dental implants, which are mobile or show only little residual bone-to-implant contacts, can usually be removed with instruments, which are also used for tooth extraction including elevators and/or extraction forceps. If the threads oppose no resistance, rotating movements are not even required.

The application of counter-torque techniques has been proposed as another conservative and atraumatic alternative for the explantation of clinically stable dental implants. Counter-torque technique (CTT) and the reverse screw technique (RST) may be useful, where damage to the surrounding tissues is to be avoided. Both of these techniques engage the implant and with a counterclockwise force and reverse screw the implant out of the bone.

Counter-torque-Technique

The treatment is performed using an implant removal kit (i.e. Biomet 3i Implant removal kit $\widetilde{ }$, Figures 16.3 and 16.4) that allows the application of a counter-torque to the boneimplant interface.

The procedure commences with the removal of the cover screw or the abutment of the implant to be removed. The technique is applied by placing an abutment or an engaging extraction tool into the implant and reverse torquing the implant counterclockwise (Figure 16.3). The need to raise a flap or not is made according to the surgical needs. A ratchet is engaged into the implant connection and then a counter-torque is exerted by a wrench in counterclockwise direction. When the reverse torque reaches

Figure 16.3 Counter-torque set (a) and technique (b) used to remove a malpositioned implant.

200Ncm without success, a piezoelectric device can be utilized to remove the coronal third (3–4mm) of the boneimplant contacts. If not available, trephine drills or burs can be utilized at low speeds and under copious saline irrigation. The implant explantation is then continued with the reverse torque wrench.

The CTT has a high success rate but is not exempt from complications, although at a very low rate. Typical complications are usually identified as fractures of the implant [12]. As a minimally invasive technique, Solderer et al. [5] have recommended the CTT as the first option for the removal of failed nonmobile dental implants.

This technique is recommended in predominantly poor bone quality (∼maxilla). In higher bone density, bone damage or fracture of the implant body or adjacent bone may occur. The existing bone density is the most critical factor, which affects the ease of implant removal via the CTT or RST.

Reverse Screw Technique

A reverse screw removal drill is usually indicated when the internal connection of the implant (threads) is damaged, or an external connection implant requires removal and the Counter torque technique (CTT) technique may not be advisable, or when the counter-torque method is unsuccessful.

A screw removal device is inserted clockwise into the implant and tightened using the torque to engage the implant. This technique is recommended only for poorly dense bones. In higher bone density bone, damage or fracture of the implant body or adjacent bone may occur. The existing bone density is the most critical factor which affects the ease of implant removal via the CTT or RST. Caution is also advised with smaller-diameter internal implants (∼3.0mm) because fracture of the implant body may occur.

The counter-torque tool kit (Figure 16.4) is made up of three components:

- 1) internal implant removal screw, which is screwed into the implant
- 2) implant remover driver, which is screwed on the implant removal screw anticlockwise
- 3) torque ratchet which has two torque gauges one side measures the correct torque for the implant removal screw to be driven into the implant, and the other side measures the correct torque for the removal of the implant.

The system has been designed to fit a large variety of implant systems and, therefore, is a universal kit and not implant-specific. The correct implant removal screw is chosen according to the thread pitch of the implant connection to drive into the implant. This screw is for one use only and essentially creates its own thread inside the implant – this is torqued to a maximum of 60Ncm. The implant remover driver is selected according to the implant design platform. The implant remover driver has a thread inside it that screws down onto the implant removal screw – this is torqued to a maximum of 300 Ncm. It is essential that when the torque wrench is used to remove the implant; the clinician turns it slowly anticlockwise working their way up to the maximum torque. Once the osseointegration has broken between the implant and the bone, the implant can be removed by hand.

Some implant designs have an anti-rotational feature, which is usually incorporated into the implant body in the apical region. The most common design employs a hole or vent. In theory, bone can grow through the apical hole and resist rotational loads applied to the implant. These implants may require the use of a trephine or bur to cut to the apex to sever the bone anchorage and remove the implant if the implant cannot be rotated with the CTT or RST.

Bone Removal Techniques

Surgical Removal Using High Speed Burs and Elevator/Forceps

This surgical technique uses burs and elevators/forceps by creating mesial and distal slots on the implant. An elevator is used with lateral movements to loosen and remove the implant. Alternatively, bone is removed mesially and distally around the implant using a round bur or fissure burs, the implant is then grasped with the dental forceps, similar to a surgical tooth extraction.

In the presence of thin cortical plates but without any critically adjacent structures, the mesial and distal bones can be removed to allow insertion of elevators and extraction forceps.

In cases with thick cortical plates, or in the presence of buccal bone defects, an osteotomy can be performed on the vestibular aspect of the implant and up to the apex. Extraction forceps can then be employed for implant removal. Yet, flap reflection has to be performed in all cases.

Despite the successful implant removal, this technique remains more invasive and highly dependent on the operator's skills. One study [13] described a two-part separation of the implant to minimize bone damage. This technique is described as difficult, especially when separating the apical portion of the implant. Nevertheless, successful explantation and simultaneous implant placement could be performed using a similar implant in both length and diameter.

Moreover, the significant amount of titanium particle debris generated by the implant separation represents a major limitation [14]. Particles of the implant surface may enter the wound and should be removed as part of the procedure [9].

The additional risk of injury to adjacent bones and vital anatomical structures should also be considered when utilizing this technique. When the implant is in close proximity to a vital structure before using the burs, accurate radiographs, and eventually a cone beam scan (CBCT) are recommended to locate and identify relevant anatomical structures [9].

Piezo-surgical Removal

As the use of ultrasonic inserts enables precise and selective cuts. Implant removal using piezosurgery is an attractive alternative to trephine burs or rotary drills [15, 16].

After careful clinical and radiographic analysis and flap selection according to the position and size of the implant, a full-thickness mucoperiosteal flap is recommended. The appropriate ultrasonic insert is selected (case by case) based on several factors, including the inclination of the fractured implant, dimensions of the implant fragment, type of bone density, and vital anatomical structures in close proximity to the implant. It is always preferable to begin the osteotomy using inserts with thin tips. A circumferential osteotomy is performed with a piezoelectric device using a diamond-coated insert. The piezo-surgical device must be used in the "BONE"-mode under maximum irrigation with cold saline solution. The handpiece is guided around the implant firmly but without excessive force. Heavy pressure which could impede insert vibration, should be avoided, since pressure might transform energy into heat.

A diamond-tipped ultrasonic insert is initially used to create circumferential peri-implant holes. The insert must be placed parallel to the long axis of the implant and gradually pushed up to a depth as close as possible to the apical portion of the implant.

Care must be taken to ensure minimal invasiveness; the clinician must work in close contact with the fixture threads, and the ultrasonic insert must be gently moved around the implant to remove only the necessary bone and safely remove the implant. Following osteotomy, the fixture can be easily luxated and removed using common extraction tools. This approach enables the clinician to evaluate the potential of bone regeneration in the defect and simultaneous implant placement or future implant placement in the same surgical site.

Conventional explantation techniques always require protection of adjacent soft tissue, because cutting is not limited to bone and can easily affect other tissues if not performed properly. In particular, complex explantation of implants located at technically or anatomically challenging sites requires adequate surgical exposure to ensure the safety of adjacent structures, and this resulting in an extended surgical time. In contrast, piezoelectric surgery does not cause visible damage to adjacent soft tissues, even when the tissues are only slightly touched by the instrument. Piezoelectric surgery reduces the existing risks of damaging the vascular network of the lingual plate of the mandible and the preservation of intact soft tissues. The ultrasonic vibrations break down the irrigation liquid into very small particles that wash away from the surgical field and make it appear bloodless. Furthermore, the bone healing response following piezoelectric surgery seems to be more favorable than osteotomies performed with burs (Figure 16.5) [17].

Piezoelectric and Grafted Removal Technique for Removal of Blade Implants

The piezoelectric and grafted removal technique of blade implants (Figure 16.6a–e) can be recommended for the successful removal of fixed mandibular blade implants with minimal intraoperative trauma, maximal conservation of the existing alveolar bone, and shortening of the total treatment time by simultaneous grafting for immediate or future implant placement [18].

In the case presented in Figure 16.6, a piezosurgical device was used to perform an ostectomy for the removal of the left blade implant to save as much bone tissue as possible, so that root-form implants might later be inserted. The patient underwent surgery for the removal of two blade implants that were no longer functional. The peri-implant ostectomy was carried out with conventional burs on the right side and a piezoelectric surgery device on the left side. The piezo instrument demonstrated to be effective and precise during ostectomy, providing an extremely thin cutting line. During the course of the operation and at postoperative controls after 7 and 30 days, patients did not show any relevant complications and still had sufficient alveolar bone to be treated with root-formed implants. The piezosurgical device proved to be an effective instrument in interventions requiring a significant saving of bone tissue, extreme precision in cutting, and respect for soft tissues [31].

Trephine Drill Technique

There are several sizes of trephines available that correspond to various implant diameters. The smallest effective size trephine should be selected to avoid collateral damage to the neighboring bone, teeth, and/or implants [9]. Ideally, an appropriate trephine drill with a diameter and length corresponding to the size of the implant to be removed is selected (Figure 16.7).

The healing abutment or abutment/crown is unscrewed and a mucoperiosteal flap raised, if necessary. The trephine drill is sunk over the implant into the bone using low-speed drilling and light pressure with running saline cooling. A hole is drilled taking care that the trephine has been sunk to the exact depth by controlling the outside rings on the drill. For implant systems that do not provide a guiding cylinder/pin, a healing abutment of the smallest emergence profile diameter is mounted before using the trephine. In the event that the implant is still firm after the trephine has been lifted (in cases of insufficient drilling depth), an elevator is placed into the empty space and lightly twisted to break the bony anchorage, enabling easy removal of the implant using the fingertips.

Trephine burs can be used to remove implants, but this technique can be very invasive, and thus should be used only when absolutely necessary. There are a variety of sizes of trephines and the smallest trephine should be used (the internal diameter of the trephine should be slightly larger

Figure 16.5 Algorithm for non-bone removal techniques, modified according to Froum et al. [9]. *Source:* Adapted from Froum et al. [9].

Figure 16.6 (a–e) While the right blade implant was removed traditionally with bone cutting burs, the left blade implant was explanted with piezo electric tips (b+c), the defect was grafted with an allograft (d). The differences in postoperative defect healing can clearly be seen: right side in bone cutting technique, left side with piezoelectric device (e).

than the implant to avoid engaging the implant body). The preferred cutting speed is 1200–1500 rpm [3]. Once the coronal half of the bone has been removed, elevators, forceps, and torque drivers can be used to remove the implant. A number of complications have been noted in the use of trephine burs including fatigue fracture of the mandible and osteomyelitis [19, 20].

Furthermore, using trephine drills may complicate immediate implant placement. The new implants often need to be larger in diameter and/or longer than the explanted ones to ensure primary stability. Whether

implant placement is immediately planned or not, care should be taken when utilizing resective techniques.

However, anatomical considerations and surrounding vital structures should be evaluated. If the apex of the implant is in approximation to a vital structure, the trephine bur should not be used at the apex to avoid vital structure damage.

Finally, trephine drills should be utilized at a low speed with copious irrigation in order to avoid overheating and the consequent risk of necrosis and impaired bone regeneration [2].

Figure 16.7 Trephine drill kit with different diameters and lengths. (a) Trephine drill kit with trephine burs of different diameter and length. (b) Trephine drill set with 3 different trephine burs of different diameter but same length. Burs show depth precision markers.

Laser-assisted Explantation

Only one case report described the removal of a single dental implant using an Er,Cr:YSGG-laser [21]. The procedure is described as being similar to piezo-surgical interventions, because a circumferential destruction of the boneimplant interface by the laser light is achieved. The specific laser wavelength has a high absorption by hydroxyapatite generating micro-explosions and destruction on the surrounding target tissue and leads to clean cuts without any thermal damage. As a further advantage of laser-assisted surgery, an optimal hemostatic control is reported, thus facilitating good visualization, and therefore accelerating the intervention [22].

Another in vitro study on human mandibles used the same laser and compared it to the conventional trephine approach [23]. Assessed parameters were the amount of the removed bone, duration of the procedure, and morphological alterations on the surface of the bone. The results showed almost half of removed bone through the laser method compared with the trephine burs. Regarding the duration of the procedure, the trephine burs were more than twice as fast as the laser. Assessing morphological bone alterations in the laser group, the authors found welldefined bone edges without any thermal alteration, whereas the trephine bur group showed abnormal bone formations with some microcracks.

In a case study, Worni et al. [24] heated the implant's inner connection using a $CO₂$ laser, which resulted in localized laser-induced thermo-necrosis at the bone-to-implant contact. One week following laser application, explantation could be performed easily with a torque slightly more

than 35Ncm. No complications occurred during the healing period. The result was a very easily performed explantation while preserving a maximum of the surrounding bony structure. Healing was uneventful, and no further visible bone loss could be observed during the healing time.

Laser surgical removal might be a less invasive and traumatic intervention as compared with trephine burs; however, the procedure is more time-consuming. Although laser surgery is already established in the fields of periodontology and implant dentistry, and some preliminary data seem promising, more research is needed before this technique can be recommended for routine implant removal.

Alternative Techniques

Bony Lid Approach

The bony lid technique, as a bone-saving method, has been described for various indications in the literature by Khoury et al. The main indication is endodontic apical surgery of mandibular molars [25–27].

The bony lid is prepared with a MicroSaw (Dentsply, Mannheim, Germany) or a piezosurgical unit. The MicroSaw consists of a diamond disc mounted on a contraangle or handpiece with a soft tissue protector. The cuts are large enough to achieve wide uncovering of the implant after luxation of the buccal window (Figure 16.8a). Careful handling is necessary to avoid injuring the roots of adjacent teeth with the diamond disc. Therefore, a safety margin of 1–2mm has to be considered. The bony lid is then luxated with a thin chisel. If the implant to be removed is still covered by a layer of cancellous bone, the

Figure 16.8 Bony lid approach. (a) Exposing and removal of the implant after luxation of the buccal bone lid. (b) After removal of the implant, replantation of the bony lid.

bone is luxated with a curette and preserved with the main cortical bony lid in saline solution. After removal of the implant, the bony lid is then reimplanted (Figure 16.8b). An exact fit can be achieved due to mesial and distal beveling so that generally there is no need for further fixation [28].

In many patients, the bony lid approach can be performed simultaneously with an implant insertion or with a bone grafting procedure. Implants can be placed in the grafted area after three months. Normal healing without any infection is usually observed.

Advantages of the bone lid technique over traditional methods for implant removal are considered to be a minimization of the bony defect while preserving the contour of the explantation socket. The technique decreases the overall treatment time, improves outcomes, and allows a combination with other non-surgical techniques (counter-torque and RST).

Partial Explantation/Coronal Resection

Partial explantation or coronal resection of osseointegrated but failing or fractured dental implants that were previously placed too close to the mandibular canal is a useful technique to avoid possible injury of the inferior alveolar nerve. The advantage of this technique is to avoid a mandibular canal entry by total explantation that can likely cause nerve injury by direct mechanical trauma or indirect postoperative injuries due to infection, induced hematoma, or pressure that compresses the nerve.

The remaining apexes of these implants can even act as a barrier to further insults to the nerve when followed by implant replacement [29]. However, this implant resection technique is only appropriate when the peri-implant bone is very thin or well-osseointegrated part of the implant is very close to the crucial anatomical landmarks (Figure 16.9a). The remaining part of the resected implant should be at least 2–3mm below the crestal bone to allow bony overgrowth (Figure 16.9b).

The technique could be an alternative to other reported techniques for the explantation of failed implants. Through the partial explantation, the preservation of existing hard tissue is possible in terms of avoiding serious complications during explantation surgery such as jaw fracture in highly atrophic mandibles or nerve injuries [30].

Clinical Guidelines for Explantation of Osseointegrated Oral Implants

The proper removal technique depends on:

- the thickness of the cortical bone plates
- the proximity of the implant to adjacent vital structures (alveolar canal, teeth, implants, and sinus maxillaris)
- timing of future implant placement
- concomitant bone defects.

The reverse torque technique is the most conservative and should be the first choice but only be utilized when extraction inserts can be screwed into the implant.

With the exception of reverse torque techniques, mucoperiosteal flap elevation is recommended for sufficient access and visibility.

When the reverse torque reaches 200Ncm without success, a piezoelectric device is recommended to remove the coronal third of the bone. If not available: trephine drills or surgical burs at low speeds and under copious saline

Figure 16.9 Partial explantation/coronal resection technique. (a) The well-osseointegrated apical part of the implant is very close to the crucial anatomical landmark (IAN). (b) The remaining part of the resected implant should be at least 2–3mm below the crestal bone to allow bony overgrowth.

irrigation are an acceptable alternative before reverse torque can be reapplied.

If the CTT fails at this point or in case of fractured implants, an osteotomy can be completed up to the implant's apex using piezosurgery or burs.

In the presence of thin cortical plates but without any critically adjacent structures: mesial and distal bone removal, insertion of elevators, and extraction forceps are recommended.

In cases with thick cortical plates or presence of buccal bone defects, an osteotomy on the vestibular aspect of the implant and up to the apex is possible before an extraction forcep is applied for implant removal.

Conclusion

Implant removal always should be performed in an atraumatic, tissue-preserving and time-saving, manner.

The choice of technique should be based on the specific clinical factors such as the design of the implant–abutment connection, remaining amount of bone-implant contact, anatomical conditions at the implant site, and bone quality.

The use of the counter-torque ratchet technique appears to be the most elegant and least invasive technique for removing an implant with the highest predictability for reinsertion of another implant, but a combination of different techniques especially the use of piezosurgery might be necessary in other clinical situations.

The author recommends reverse torque as the first choice for explantation. Despite a 100 % success rate is not guaranteed, it is the most conservative technique in terms of bone removal and flap access, meaning there is a greater opportunity for immediate implant placement. Ideally, the explantation procedure should be followed either by the installation of another implant or by GBR, or both at the same visit, when indicated.

References

- **1** Bergamini, M., Gomez, J., Wiedemann, T. et al. (2020). An algorithmic approach to evaluation of an ailing implant. *The Compendium of Continuing Education in Dentistry* 41 (8): e1–e9.
- **2** Roy, M., Loutan, L., Garavaglia, G., and Hashim, D. (2020). Removal of osseointegrated dental implants: a systematic review of explantation techniques. *Clinical Oral Investigations* 24 (1): 47–60.<https://doi.org/10.1007/s00784-019-03127-0>.
- **3** Bowkett, A., Laverty, D., Patel, A. et al. (2016). Removal techniques for failed implants. *British Dental Journal* 220: 109–114.
- **4** Greenstein, G. and Cavallaro, J. (2014). Failed dental implants: diagnosis, removal and survival of reimplantations. *Journal of the American Dental Association (1939)* 145 (8): 835–842. [https://doi.org/](https://doi.org/10.14219/jada.2014.28) [10.14219/jada.2014.28](https://doi.org/10.14219/jada.2014.28).
- **5** Solderer, A., Al-Jazrawi, A., Sahrmann, P. et al. (2019). Removal of failed dental implants revisited: questions and answers. *Cllinical and Experimental Dental Research* 1–13.
- **6** Stajčić, Z., Stojčev Stajčić, L.J., Kalanović, M. et al. (2016). Removal of dental implants: review of five different techniques. *International Journal of Oral and Maxillofacial Surgery* 45 (5): 641–648. [https://doi.](https://doi.org/10.1016/j.ijom.2015.11.003) [org/10.1016/j.ijom.2015.11.003](https://doi.org/10.1016/j.ijom.2015.11.003).
- **7** Anitua, E., Murias-Freijo, A., Piñas, L. et al. (2016). Nontraumatic implant explantation: a biomechanical and biological analysis in sheep tibia. *The Journal of Oral Implantology* 42 (1): 3–11.
- **8** Young, L., Brown, T., and Lamont, T.J. (2020). A comparison of techniques for the explantation of osseointegrated dental implants. *Evidence-Based Dentistry* 21 (4): 126–127.
- **9** Froum, S., Yamanaka, T., Cho, S.C. et al. (2011). Techniques to remove a failed integrated implant. *The Compendium of Continuing Education in Dentistry* 32 (7): 22–32.
- **10** Wiedemann, T. and Bergamini, M. (2020). Titanium intolerance and its relevance in clinical practice. *Journal of Oral Ceramic Implantology December* 11: 32–38.
- **11** Zampara, I., Bergamini, M., Talib, H., and Wiedemann, T. (2021). Is aseptic bone necrosis a cause for early implant failure in patients with metal allergies? – a case report and review of the literature. *Journal of Oral Ceramic Implantology* 11: 30–38.
- **12** Anitua, E., Murias-Freijo, A., and Alkhraisat, M.H. (2016). Conservative implant removal for the analysis of the cause, removal torque, and surface treatment of failed nonmobile dental implants. *The Journal of Oral Implantology* 42 (1): 69–77. [https://doi.](https://doi.org/10.1563/aaid-joi-D-14-00207) [org/10.1563/aaid-joi-D-14-00207.](https://doi.org/10.1563/aaid-joi-D-14-00207)
- **13** Li, C.H. and Chou, C.T. (2014). Bone sparing implant removal without trephine via internal separation of the titanium body with a carbide bur. *International Journal of Oral and Maxillofacial Surgery* 43 (2): 248–250.
- **14** Mombelli, A., Hashim, D., and Cionca, N. (2018). What is the impact of titanium particles and biocorrosion on implant survival and complications? A critical review. *Clinical Oral Implants Research* 29 (Suppl 18): 37–53.
- **15** Messina, A.M., Marini, L., and Marini, E. (2018). A step-by-step technique for the piezosurgical removal of fractured implants. *The Journal of Craniofacial Surgery* 29 (8): 2116–2118.
- **16** Marini, E., Cisterna, V., and Messina, A.M. (2013). The removal of a malpositioned implant in the anterior mandible using piezosurgery. *Oral Surgery, Oral Medicine, Oral Pathology, and Oral Radiology* 115 (5): e1–5.
- **17** Preti, G., Martinasso, G., Peirone, B. et al. (2007). Cytokines and growth factors involved in the osseointegration of oral titanium implants positioned using piezoelectric bone

surgery versus a drill technique: a pilot study in minipigs. *Journal of Periodontology* 78 (4): 716–722.

- **18** Sivolella, S., Berengo, M., Fiorot, M., and Mazzuchin, M. (2007). Retrieval of blade implants with piezosurgery: two clinical cases. *Minerva Stomatologica* 56 (1–2): 53–61.
- **19** Schroeder, A., Sutter, F., Buser, D. et al. (1996). Oral implantology. In: *Basics, ITI Hollow Cylinder System*, 445–476. Stuttgart: Georg Thieme Publ.
- **20** Esposito, M., Hirsch, J., Lekholm, U., and Thomsen, P. (1999). Differential diagnosis and treatment strategies for biologic complications and failing oral implants: a review of the literature. *The International Journal of Oral & Maxillofacial Implants* 14: 473–490.
- **21** Smith, L.P. and Rose, T. (2010). Laser explantation of a failing endosseous dental implant. *Australian Dental Journal* 55 (2): 219–222.
- **22** Romanos, G.E. (2021). *Advanced Laser Surgery in Dentistry*. Hoboken: Wiley Publ.
- **23** Hajji, M., Franzen, R., Grümer, S. et al. (2016). Removal of dental implants using the Erbium,Chromium:Yttrium-Scandium-Gallium-Garnet Laser and the conventional trephine bur: an in vitro comparative study. *Photomedicine and Laser Surgery* 34 (2): 61–67.
- **24** Worni, A., Marchand, L., Sailer, I. et al. (2018). Explantation of an osseointegrated titanium implant using laser-induced thermo-necrosis: a case report. *The International Journal of Oral & Maxillofacial Implants* 33 (6): e151–e155.
- **25** Khoury, F. (1986). Une nouvelle technique pour le curetage periapical des molaires inferieures. *Actualités Odonto-Stomatologiques* 154: 181–188.
- **26** Khoury, F., Hemprich, A., and Sass, T. (1985). Die Anwendung des freien Knochendeckels bei verschiedenen Eingriffen im Unterkiefer. *Dtsch Z Mund-, Kiefer-, Gesichtschir* 9: 298–304.
- **27** Khoury, F. and Hensher, R. (1987). The bony lid approach for the apical root resection of lower molars. *International Journal of Oral and Maxillofacial Surgery* 16: 166–170.
- **28** Khoury, F. (2013). The bony lid approach in pre-implant and implant surgery: a prospective study. *European Journal of Oral Implantology* 6 (4): 375–384.
- **29** Sahl, E., Alqahtani, A., Alqahtani, N.M., and Gallez, F. (2018). Partial explantation of failed dental implants placed in mandibular canal: a case report. *The Journal of Oral Implantology* 44 (6): 456–461.
- **30** Kutlu, H.B., Goyushov, S., and Tözüm, T.F. (2020). An alternative treatment for hopeless implants: coronal resection. *Journal of Oral Biology and Craniofacial Research* 10 (2): 104–109.
- **31** Wiedemann, T. (2023). Review of Contemporary Applications of Piezoelectric - Surgery in Modern Implant Dentistry. *Acta Scientific Dental Sciences* 7 (10): 95–104.

Part IV

Contributing Factors

17

Risks and Opportunities for Cement-retained Implant Restorations

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Introduction

The original Branemark high water design fixed prosthesis was a watershed moment in dental history. It addressed the vexing problem of mandibular complete denture therapy by creating a predictable fixed prosthetic alternative for those patients lacking lower teeth. This treatment alternative had only one method of retaining the prosthesis to the transmucosal implant; abutment-screw retention. As implantology evolved, sectional fixed prosthetic and single tooth prosthetic designs were developed after the success of the Branemark full arch screw-retained prosthesis.

An early solution for partial edentulism in either arch was to utilize either a solid or angle-correcting stock abutment, or a custom cast then later a custom-milled abutment that was designed to retain the replacement crown, held in position by the selected luting agent. Many factors common to tooth preparation were employed: features to optimize retention and resistance form, and margin placement all evolved over time, driven by idealizing patient treatment outcomes.

Design features vary between the single tooth and fixed partial denture abutments. A single implant has been used in conjunction with a natural tooth to retain a three-unit fixed partial denture. In this instance, the only means of retaining the prosthesis is to utilize a luting agent. A review of the scientific literature reveals high success rates for this approach, although it is not considered a mainstream dental therapeutic $[1-3]$.

A commercially available solid abutment can be attached to an implant at the manufacturer's recommended screw torque value. A prosthetic finishing line can then be developed and demarcated either on an analog cast or intraorally, and a conventional or digital impression can be generated. A final implant crown can be cast, milled, or printed, and attached to the abutment with a luting agent. Sometimes, a manufacturer-supplied prefabricated solid abutment cannot adequately support the required tooth form due to implant body angulation issues. This requires a workaround of some sort. Either a machined angle correcting abutment can be attached to the implant, or a more costly custom-fabricated abutment can be manufactured via a casting or milling process [4].

The major disadvantage of cement retention is the process of safely applying the correct volume of luting agent. The selected agent could be a provisional or final cement. Ideally, supra-, or epi-gingival margin design is incorporated into the abutment fabrication, as well as optimal digital or analog cement spacing is also incorporated into the process. Various strategies have been undertaken to minimize excess cement in the gingival sulcus. An inadvertent excess of cement in the sulcus can be associated with the development of peri-implant mucositis or peri-implantitis [5] (Figure 17.1). The unexpected outcome of cement in the implant sulcus can lead to chronic peri-implant inflammation proceeding on to crestal bone loss and ultimately can result in implant failure (Figure 17.2). Strategies to minimize excess cement include internal venting of the supporting abutment. Wadhwani et al. [6] advocate the placement of two holes in abutments, one high and one low in the abutment profile. This allows the excess luting agent to vent internally into

Figure 17.1 Cement visible below crown margin on implant surface. (*Source:* Courtesy: Dr. G. Romanos, Stony Brook, NY).

Figure 17.2 Visible cement excess on a periapical radiograph. (*Source:* Courtesy: Dr. G. Romanos, Stony Brook, NY).

the screw access chamber which minimizes the amount of cement that can be squeezed into the gingival sulcus by crown seating. Haas and Haas [7] advocate an approach described as the "Cement shield membrane technique" in which a specified length polytetrafluoroethylene (PTFE) is stretched over the implant abutment and adjacent teeth, and then the luting agent is applied to the intaglio surface of the implant restoration. The crown is removed, and the PTFE is removed, leaving a nearly optimal amount of cement to retain the crown atop the abutment.

Retrievability

One of the main advantages of screw retention is retrievability in the event of screw loosening, screw fracture, or other prosthetic and biological complications. Without a screw-access hole, cement-retained restorations can be difficult if not impossible to remove and often require irreversible damage to the restoration and potential damage to the implant. There are solutions to combat the issue of retrievability with these types of restorations, both of which can be incorporated into the design of the prosthesis itself or employed if separating the restoration from the abutment(s) intraorally is not possible.

One method of improving retrievability for cementretained restorations is the inclusion of additional screws into the framework design, such as the lingual set screw. The crown is cemented using the luting agent of choice and the screw is inserted. If the crown must be removed, removing the screw will cause shear forces that break the cement adhesion. Additional retention from the screw also makes it possible to use an interim cement more predictably, which can make removal easier. Disadvantages to this approach are that the additional screws/access channels can be bulky, especially in the lingual area, which can be uncomfortable to the tongue and cause plaque accumulation. The design of such a restoration also takes additional laboratory time and skill, which can be costly.

Another method of retrievability is the lingual retrieval slot mechanism, proposed by Schweitzer et al. [8] for metal ceramic implant prostheses. A 3mm deep horizontal slot is incorporated in the mid-marginal position of the lingual cast metallic surface, and this area is not covered by ceramic. If the crown must be removed, an instrument can be inserted into this slot and turned to break the cement barrier. Though slightly less laboratory technique sensitive in its fabrication than the previously described design, this will also take additional time and skill to fabricate and can also be a plaque trap. This area can be filled on a provisional basis with composite to negate the plaque trap, but this material would require removal by rotary instrumentation.

A screw access channel can be cut through the crown to access the abutment screw if the crown is cement-retained. This will cause weakening of the ceramic and potential for future fracture, as it will then need to be filled with a different material such as composite resin after completion of the screw-tightening procedure. Without knowing precisely where the abutment screw access is, this hole can become quite large and compromise the integrity of the restoration. Several techniques have been proposed to register the location of the screw-access hole to minimize the

damage. Farzin et al. [9] proposed designing a ledge in the framework in the location of the screw access hole to support the remaining porcelain after perforation of the restoration.

Doerr [10] proposed the fabrication of a retaining-screw location guide utilizing the original implant– and abutment–level casts. A cast of the cemented restoration is made and used to fabricate a vacuum clear guide, like a surgical guide, with the screw-access hole prepared.

Another option is to place the abutment on the master cast and mount this on a surveyor. After recording the screw-access position using a surveyor pin, the restoration is placed on the abutment and placed back on the surveyor. The surveyor pin is replaced with a fine-tip brush loaded with brown or white stain, which can be applied to the occlusal surface of the restoration in the screw-access hole area. This requires that a small concavity first be made in the restoration so that the stain can be received and not worn away. However, this is limited due to esthetic concerns as well as the potential for removal of stains if the restoration needs intraoral adjustment after insertion or the patient exhibits parafunctional habits.

Several authors proposed the use of digital photographs prior to the insertion of the restoration to document the position of the screw-access hole. The camera is mounted on a tripod to maintain the same position for both photos. One photo is taken of the definitive abutment(s) on the cast and one with the definitive prosthesis placed on the abutment(s) on the cast. The photos are superimposed to locate the screw – access hole using a photo-editing software of presentation software program and can be stored digitally in the patient's file.

CAD/CAM

Finally, the use of provisional cement may be advised to provide better potential retrievability. This is acceptable only if the abutment has been designed appropriately with maximal retention, but even then, degree of retention may be unpredictable. Extra appointments, loss or damage to the restoration, and patient dissatisfaction can arise if recements are required. Provisional cement tends also to be more soluble and not advised for long-term use. However, it must be noted that different prostheses may warrant different luting agents, and not all restorations should be managed in the same manner. Using definitive versus provisional cement for cement-retained implant restorations is dependent upon a multitude of factors such as provider preference, the design and span of the restoration, the ability to achieve a dry field, access to clean and remove excess cement, and the potential for future biologic or prosthetic complications.

Cement Selection

Material properties such as viscosity, flow rate, and working time will influence the *potential* for remaining excess cement. Factors such as the amount, site, and radiographic density will influence the *detection* of residual cement [11]. Ideally, the cement used to lute cement-retained implant restorations would be as radiopaque as possible, to help distinguish remaining subgingival cement from surrounding anatomic structures and materials. The site of cement extrusion will also influence its radiographic detectability, as even a highly radiopaque cement may be indetectable if it is directly facial or lingual and obstructed by the implant– abutment complex. In comparison, a less radiopaque cement such as RMGI may be detected even in small amounts if the cement flows in a circumferential layer around the abutment, since the radiographic beam will pass through it tangentially many times, described as the "peripheral egg-shell effect" [11]. In summary, it is easier to identify cement remnants radiographically in the proximal areas compared to buccal/lingual.

Radiographic detectability of cement varies widely, some allowing for easy detection and others undetectable even at a thickness of 2mm [12]. Zinc is highly radiopaque, making zinc oxide eugenol and zinc phosphate readily detectable. In comparison, glass ionomers, calcium hydroxide, and resin cement have poor radiodensity properties, especially with a thickness less than 1 mm, unless specific radiopacifiers are added during formulation [12]. Other factors that influence the detectability of luting agents are the exposure settings, beam angulation, and evaluation methodology [13].

Biocompatibility of luting agents must also be considered, and not all cements that are used on natural teeth are suitable for implant restorations. Resin types of cement can be damaging to tissues as there can be a release of free monomers after polymerization and the formaldehyde present in their air-inhibited non-polymerized superficial layer [14]. In comparison, zinc-containing cements show potentially beneficial antibacterial effects due to zinc ion release [13]. Though commonly used in clinical practice, Durelon® (ESPE Co.), a polycarboxylate luting agent, according to the manufacturer's instructions has a corroding effect on titanium and is only recommended for Ti-free restorations on natural teeth [15]. The application of methods to reduce excess cement can help avoid the biological side effects of remnant cement.

As previously discussed, several factors influencing the retentiveness of the restoration, along with the span of the restoration and the provider preference, contribute to the choice between a provisional or definitive cement. Definitive types of cement (resin cement and glass ionomer cement) have shown to be more difficult to remove and result in more residual cement when compared with zinc oxide eugenol cement [16].

Strategies to Minimize Cement Excess Expressed into Sulcus

The soft tissue attachment around an implant differs from that surrounding a natural tooth [15]. A reduced number of collagen fibers and the direction of these fibers, as well as the lack of perpendicular gingival fibers, make the soft tissue-implant interface significantly more delicate [15]. For this reason, the potential for cement extrusion deep into the gingival sulcus is more common around implant restorations. Implants and their corresponding restorative surfaces (i.e. titanium and zirconia) also provide a more difficult surface for cement debridement compared to dentin [17]. The presence of excess cement may cause an adverse biological response, with the potential for periimplantitis and even implant failure [18]. As a result, various methods have been reported to help reduce the amount of cement expelled into the gingival sulcus.

Schwedhelm et al. [19] proposed the use of a vent or escape way in the metal-ceramic implant restoration. In this design, an 18-gauge stainless steel rod is incorporated into the wax pattern at the thickest portion of the restoration's lingual surface to produce a small, accessible vent hole. When the prosthesis is inserted, excess cement theoretically escapes through the vent hole as opposed to being expressed down into the sulcus. At the time of insertion, a steel rod is placed into the hole, and cement is allowed to set. The rod is then cut to be flush with the restoration surface and the restoration is therefore sealed.

Multiple authors have proposed the use of a copy abutment/abutment analog for a two-step cementation [15, 20, 21]. Various materials and methods have been studied demonstrating this concept, which ultimately establishes an ideal and uniform cement layer prior to insertion on the definitive abutment intraorally. This can therefore reduce if not eliminates excess cement. Per Wadhwani et al. [11], the optimal cement volume necessary for cementation is estimated to be 3% of the total crown volume, which fills approximately 40–50μm of space. An abutment analog can be fabricated using CAD/CAM technology and milled/printed to a smaller dimension than the definitive abutment, accounting for ideal cement space. Or, this can be accomplished via analog procedures, for example using a die spacer or PTFE tape along the intaglio surface of the restoration to represent ideal cement thickness and then fabricating the copy abutment using PVS [15]. When using a two-step cementation approach, cement with a longer working time must be used [22].

Within the restoration, where the cement is placed before seating has been studied. Dumbringue et al. [22] proposed placing a luting agent only on the occlusal half of the intaglio of the restoration, allowing flow along the axial walls upon seating. However, this has been shown to lead to incomplete sealing of the restorative margin which can cause biological complications due to microgap formation harboring bacteria [23]. Haas and Haas [7], on the other hand, found that cement placement around the marginal area, less in the occlusal portion of restoration aids in less extrusion. Romanos [24] suggests introducing liquified petroleum jelly into the abutment sulcus immediately after cementation and then using the air-water spray with a suction tip to remove the sulcular excess of cement.

Seo and Seo [25] propose the use of a rubber dam when cementing implant restorations, which can block the extrusion of excess cement into the sulcus as well as provide isolation and protection of the gingiva from direct contact with cement. This method is appealing as it requires no additional laboratory steps, is not significantly timeconsuming and can ultimately reduce patient discomfort during the insertion process. According to Wadhwani et al. [6], one possible way to reduce excess cement flow is to leave the screw access channel open or partially open as it will act as a reservoir for excess cement. In addition to limiting excess cement, it has been shown that partial filling of the screw channel can improve prosthesis retention [26].

Digital Versus Analog

CAD/CAM procedures allow for more accurate fabrication – precise control of cement space compared to analog fabrication [27].

Abutment Margination

CAD/CAM or analog procedures can be used to fabricate custom abutments whose margins precisely follow the soft tissue contours, bringing the gingival margin of the restoration to an ideal position, despite the depth and position of the implant. Ideally, the gingival margin of a cement-retained restoration should be supra or epigingival for ease of cement removal. Though supra-gingival margins are recommended, this is not always possible, especially in the esthetic zone. An anterior implant restoration must often have a subgingival margin to meet the demand for a proper emergence profile. Buser et al. [28] recommended leaving the restoration margin 1–2mm subgingivally (Figures 17.3–17.6).

Figure 17.3 Screw retained single tooth provisional for left central incisor (#9).

Figure 17.5 Custom ceramic abutment corrects angulation and allows supragingival crown margination and cementation.

Figure 17.4 Impression coping demonstrates poor angulation of a well-integrated implant in the #9 position.

Removal of excess cement becomes complicated, particularly if the sulcus depth is greater than 3mm [17]. Deep gingival margin placement makes it not only difficult to ensure excess cement removal, but also to assess the damage to the surface of the implant restoration caused by the removal [19]. An in-vitro study by Ayyadanveettil et al. [16] showed a statistically significant relationship between the location of the finish line and the amount of cement trapped in the peri-implant sulcus. The findings were consistent with those of Agar et al. [17] in that subgingival margins between 1.5 and 3mm may lead to inadequate cement removal. Consistent with the findings of Wilson [29], even after the operator believed all cement had been adequately removed, some remained adhered to the restoration in the gingival sulcus.

Figure 17.6 Cemented all-ceramic crown 9 atop custom zirconia abutment with gingival ceramic applied to the abutment.

Peri-implantitis Incidence Relating to Cement Displaced into the Sulcus

A major disadvantage of cement-retained restorations is the extrusion of excess cement into the peri-implant area, which accounts for 81% of peri-implant disease [29]. The presence of residual cement in the sulcus is a risk factor for bleeding, abscess formation, and inflammation in the periimplant tissues with the potential for attachment loss and implant failure [27]. It is believed that the etiology of periimplant disease related to residual cement is a result of bacterial colonization of the foreign material [11]. This response may happen quickly; it can also occur several years after the restoration has been completed [29]. Per Wilson [29], upon identification and removal of residual

cement in these clinical situations, resolution of the signs of periimplantitis occurred in 76% of the patient treatments [29]. It has also been seen that residual excess cement will not always cause this response, suggesting different variables such as host factors, type and amount of cement, and location of cement likely play a role. In a retrospective treatment analysis by Linkevicius et al. [30], the authors found that residual cement around implant restorations was more likely to cause peri-implantitis in patients with a history of periodontitis.

Several attempts to combat the issue of residual cement have been discussed; however, sometimes it is very difficult to avoid. Irrespective of the type of instrument used scratches on the abutment and some amount of cement remnants are consistently present which is undesirable due to the potential for increased biofilm formation on rough surfaces [17]. Resin cement has been reported to be more difficult than zinc phosphate or glass ionomer cement to remove from titanium abutments [31]. Titanium abutments have long been used as the gold standard to support implant restorations due to their excellent physical properties and biocompatibility [31]. However, zirconia has become a popular material selection due to improved esthetics, excellent physical properties, and biocompatibility. Specific properties such as low surface free energy, low surface wettability, low cement bond strength, and high surface hardness make zirconia a desirable choice with regards to cement removal, as these may facilitate ease of removal and minimize scratching of the abutment surface [31].

Multiple studies have compared peri-implant soft tissues of cement versus screw-retained restorations and reported poorer soft tissue health associated with cementretained restorations [32]. In a systematic review by Sailer et al. [33], it was found that the incidence of serious biologic complications such as bone loss exceeding 2mm was higher with cemented reconstructions compared to screw retained. While screw-retained restorations encountered

more frequent mechanical and technical complications, cement-retained restorations experienced more serious biological complications which may be more difficult to resolve clinically. There was no statistically significant difference in the five-year survival rate of implants supporting single crowns when comparing screw- versus cement-retained restorations. However, partial FDPs showed a trend toward more implant failures with cemented reconstructions, and for full-arch FDPs, significantly more implant failures compared to screw-retained restorations. The incidence of implant loss increased with the size of the reconstruction, which could theoretically be in part due to difficulty in cement removal with increasing span [33].

*In summary, w*hen considering implant prosthesis retention, carefully designed abutments, which incorporate two vent holes can be successfully utilized with cement retention. There are numerous techniques elucidated previously that minimize uncontrolled cement expression into the implant gingival sulcus. Unfortunately, when not carefully controlled, excess cement has been implicated as a primary etiology for cement-induced peri-implantitis. Clinically the author's preference is screw retention. For an implantfixed prosthesis, this can include a combination of "implant support" which utilizes a custom abutment to support a prosthesis with unfavorable screw access angulation in combination with a direct-to-the-implant engaging screw connection. For example, a 3-unit implant FPD 12-13-14 with an unfavorable facial screw access channel on the 1st maxillary bicuspid in the esthetic zone can have a single custom abutment for implant support. The final screw-retained fixed prosthesis can attach by screw retention directly to the 1st molar implant using engaging componentry. In this manner, the three-unit FPD is a one-piece construct that inserts over the custom abutment and screws into the terminal molar implant. Finally, the clinician's treatment philosophy should change over time based on best practice evidence that is continuously evolving in implant dentistry.

References

- **1** Shadid, R. and Sadaqa, N. (2012). A comparison between screw and cement-retained implant prostheses. a literature review. *The Journal of Oral Implantology* 38: 298–307.
- **2** Sherif, S., Susarla, H.K., Kapos, T. et al. (2014). A systematic review of screw- versus cement-retained implant-supported fixed restorations. *Journal of Prosthodontics* 23: 1–9.
- **3** Wittenben, J.G., Joda, T., Weber, H.P., and Bragger, U. (2017). Screw-retained vs. Cement-retained

implant-supported fixed dental prosthesis. *Periodontology 2000* 73: 141–151.

- **4** Malpartida-Carrillo, V., Tinedo-Lopez, P.L., Ortiz-Culca, F. et al. (2020). Techniques for retrievability and for registering screw access holes in cement-retained implantsupported prostheses: a scoping review of the literature. *The Journal of Prosthetic Dentistry* 123: 427–433.
- **5** Tsigarida, A., Chochlidakis, K., Fraser, D. et al. (2020). Peri-implant diseases and biologic complications at

implant-supported fixed dental prostheses in partially edentulous patients. *Journal of Prosthodontics* 29: 429–435.

- **6** Wadhwani, C., Hess, T., Pineyro, A. et al. (2013). Effects of abutment and screw access channel modification on dislodgement of cement-retained implant-supported restorations. *The International Journal of Prosthodontics* 26: 54–56.
- **7** Haas, R.C. and Haas, S.E. (2020). Cement shield membrane technique to minimize residual cement on implant crowns: a dental technique. *The Journal of Prosthetic Dentistry* 123: 223–227.
- **8** Schweitzer, D.M., Berg, R.W., and Mancia, G.O. (2011). A technique for retrieval of cement-retained implantsupported prostheses. *The Journal of Prosthetic Dentistry* 106: 131–135.
- **9** Farzin, M., Derafshi, R., Giti, R. et al. (2021). A special design to facilitate retrieval of cement-retained zirconiabased implant-supported restorations. *Journal of Dentistry* 22 (2): 132–137.
- **10** Doerr, J. (2002). Simplified technique for retrieving cemented implant restorations. *The Journal of Prosthetic Dentistry* 88 (3): 352–353.
- **11** Wadhwani, C., Rapoport, D., La Rosa, S. et al. (2012). Radiographic detection and characteristic patterns of residual excess cement associated with cement-retained implant restorations: a clinical report. *The Journal of Prosthetic Dentistry* 107: 151–157.
- **12** Wadhwani, C., Hess, T., Faber, T. et al. (2010). A descriptive study of the radiographic density of implant restorative cements. *The Journal of Prosthetic Dentistry* 103: 295–302.
- **13** Nematollahi, F., Beyabanaki, E., and Alikhasi, M. (2016). Cement selection for cement-retained implant-supported prostheses: a literature review. *Journal of Prosthodontics* 25: 599–606.
- **14** Goldberg, M. (2008). In vitro and in vivo studies on the toxicity of dental resin components: a review. *Clinical Oral Investigations* 12: 1–8.
- **15** Wadhwani, C. and Pinero, A. (2009). Technique for controlling the cement for an implant crown. *The Journal of Prosthetic Dentistry* 102: 57–58.
- **16** Ayyadanveettil, P., Thavakkara, V., Koodakkadavath, S. et al. (2021). Influence of collar height of definitive restoration and type of luting cement on the amount of residual cement in implant restorations: a clinical study. *The Journal of Prosthetic Dentistry* 129: 109.
- **17** Agar, J.R., Cameron, S.M., Hughbanks, J.C., and Paker, M.H. (1997). Cement removal from restorations luted to titanium abutments with simulated subgingival margins. *The Journal of Prosthetic Dentistry* 78: 43–47.
- **18** Gapski, R., Neugeboren, N., Pemeraz, A.Z., and Reissner, M.W. (2008). Endosseous implant failure influenced by

crown cementation: a clinical case report. *The International Journal of Oral & Maxillofacial Implants* 23: 943–946.

- **19** Schwedhelm, E.R., Lepe, X., Aw, T.C. et al. (2003). A crown venting technique for the cementation of implant-supported crowns. *The Journal of Prosthetic Dentistry* 89: 89–90.
- **20** Bhushan, P., Aras, M.A., Chitre, V. et al. (2017). A novel universal cementation technique for implant-supported crowns with subgingival margins. *Journal of Prosthodontics* 26: 628–632.
- **21** Yuzbasioglu, E. (2014). A modified technique for extraoral cementation of implant retained restorations for preventing excess cement around the margins. *The Journal of Advanced Prosthodontics* 6: 146–149.
- **22** Dumbrigue, H.B., Abanomi, A.A., Cheng, L.L. et al. (2002). Techniques to minimize excess luting agent in cement-retained restorations. *The Journal of Prosthetic Dentistry* 87: 112–114.
- **23** Quirynen, M. and van Steenberghe, D. (1993). Bacterial colonization of the internal part of two-stage implants. An in vivo study. *Clinical Oral Implants Research* 4: 158–161.
- **24** Romanos, G.E. (2019). A simplified technique to control excess cement material underneath cementretained implant restorations: technical note. *The International Journal of Oral & Maxillofacial Implants* 34: e17–e19.
- **25** Seo, C.W. and Seo, J.M. (2017). A technique for minimizing subgingival residual cement by using rubber dam for cement-retained implant crowns. *The Journal of Prosthetic Dentistry* 117 (2): 327–328.
- **26** Tan, K.M., Masri, R., Driscoll, C.F. et al. (2012). Effect of axial wall modification on the retention of cementretained, implant-supported crowns. *The Journal of Prosthetic Dentistry* 107: 80–85.
- **27** Wang, W., Chang, J., Wang, H.M. et al. (2020). Effects of pre-cementation on minimizing residual cement around the marginal area of dental implants. *The Journal of Prosthetic Dentistry* 123: 622–629.
- **28** Buser, D., Martin, W., and Belser, U. (2004). Optimizing esthetics for implant restorations in the anterior maxilla: anatomic and surgical considerations. *The International Journal of Oral & Maxillofacial Implants* 19: 43–61.
- **29** Wilson, T.G. Jr. (2009). The positive relationship between excess cement and peri-implant disease: a prospective clinical endoscopic study. *Journal of Periodontology* 80: 1388–1392.
- **30** Linkevicius, T., Vindasiute, E., Puisys, A. et al. (2013). The influence of the cementation margin position on the amount of undetected cement. A prospective clinical study. *Clinical Oral Implants Research* 24 (1): 71–76.

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- **31** Hsu, K.W., Liang, C.H., Peng, Y.C. et al. (2021). Comparison of the residual cement on custom computeraided design and computer-aided manufacturing titanium and zirconia abutments: a preliminary cohort study. *The Journal of Prosthetic Dentistry* 128: 618.
- **32** Weber, H.P., Kim, D.M., Ng, M.W. et al. (2006). Periimplant soft-tissue health surrounding cement- and

screw-retained implant restorations: a multi-center, 3-year prospective study. *Clinical Oral Implants Research* 17: 375–379.

33 Sailer, I., Mühlemann, S., Zwahlen, M. et al. (2012). Cemented and screw-retained implant reconstructions: a systematic review of the survival and complication rates. *Clinical Oral Implants Research* 23 (Suppl 6): 163–201.

Implant Supportive Maintenance for Fixed Prosthetic Rehabilitations: The Patient with the Complete Arch Fixed Implant–supported Rehabilitation: Prosthetic Concepts to Optimize Maintenance Protocols

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The introduction of dental implants as a treatment modality in dentistry has helped millions of edentulous people to overcome the need for removable complete dentures by making fixed implant–supported prosthetic reconstructions possible. Implants have thus offered a great service to edentulous patients with maladaptive denture experiences. While the success of dental implants has been reported to be outstanding, early and late implant failures, as well as failure of implant prostheses, are well-documented and considered to be serious issues [1–3]. That is especially true for patients with advanced cases of bone resorption and limited bone quantity and quality for implant placement. Soft and hard tissue ridge deficiencies not only hinder the surgical and prosthetic success of implant therapy, but subsequent implant and/or prosthetic failure will cause further tissue loss, complicating treatment or leaving the patient with traditional removable prosthetics as the only remaining option. Maintenance is a critical component of dental care, and implant therapy is no exception. As such, it is the aim of the authors to present an evidence-based discussion of factors influencing the short- and long-term maintenance of fixed Implant–supported prosthetic rehabilitations.

The complications associated with complete arch fixed implant–supported rehabilitations are numerous as discussed in this chapter, but most of these complications can be summarized by the following etiologies: bacterial adhesion and load, host response, mechanical overload, and physical deterioration. In analyzing these etiologies, the authors have found that the choice of prosthetic materials is a key factor in prognosis affecting biocompatibility, occlusal surface, and color stability. Additional factors include the prosthesis design, especially at the intaglio surface, location of the prosthetic platform, and the mode of retention influencing the presence of residual cement.

Restorative treatment involving dental implants presents multiple interfaces, which impact the maintenance and prognosis of the restoration. Some of those interfaces exist within the prosthetic apparatus: implant to abutment, abutment to prosthesis framework, and prosthetic framework to veneering material, if any (Figures 18.1–18.2); some are between the prosthetic components and the surrounding hard and soft tissues: implant to bone, implant, and abutment to emergence soft tissue (non-keratinized), intaglio surface of the prosthetic framework to the attached mucosa, polished surface of the prosthesis to the buccal and lingual mucosa, lips, and tongue, and occlusal surface to the opposing dentition or prosthesis (Figures 18.3–18.5). The topography of interfaces between the prosthetic components and the surrounding tissues, the properties of the materials, and the biology of those tissues in contact with the prosthesis would all influence proper hygiene, bacterial adhesion, and biological response. Passivity of fit, the use of screw or cement retention, the presence of micro-gaps, as well as occlusal stability would additionally influence bacterial and mechanical factors, and thus, long-term success.

The materials used in the fabrication of complete arch fixed implant prostheses include *framework* materials such as gold, silver-palladium, titanium, zirconia, and high-strength polymers, and *veneering* materials including acrylic resin, composite resin, and ceramics. These materials all have different physical and mechanical properties which influence the performance of the prosthesis and long-term success. On the other hand, surface properties, such as roughness, hydrophobicity, and electric charge, can significantly modify the extent of bacterial adhesion [4]. Biological complications will then follow the increase in pathogenic bacterial load adjacent

Figures 18.1–18.2 Interfaces of prosthetic apparatus. (a) implant to abutment. (b) abutment to prosthesis. (c) framework to veneer.

to a prosthesis. While oral bacteria can adhere to hydrophobic and hydrophilic surfaces, fluctuating shear conditions that exist in vivo prevent much biofilm from accumulating on hydrophobic surfaces. As such, biofilm tends to accumulate on hydrophilic surfaces, particularly those that are more rough than smooth. Multiple bacterial strains make up oral biofilms, but their character and impact can be material-specific. For example, Candida species tend to attach to acrylic resin. Gold has been found to harbor thick biofilms in vivo, but they are barely viable. Biofilms on ceramics, on the other hand, are thin and highly viable. As for composites, biofilms cause surface deterioration, which bolsters the process of further biofilm formation and attachment [5].

The Metal-acrylic Hybrid Prosthesis

Understanding the current advances in implant–supported prosthetic rehabilitation requires a historical understanding of the development of prosthetic materials used in this domain. This no doubt goes hand-in-hand with the earliest

Figures 18.3–18.5 Interfaces of prosthetic components to tissues. (a) implant to bone (subcrestal). (b) abutment/prosthesis to soft tissue (subgingival), (c) prosthesis to soft tissue (intaglio/ pontic), (d) prosthesis to buccal and lingual mucosa (polished), (e) occlusal surface to opposing dentition/prosthesis.

Figures 18.3–18.5 (Continued)

applications of implantology in dentistry. Immediately after comprehending the biologic process of osseointegration, Professor Brånemark and his co-workers realized the therapeutic value of implants and foresaw the great impact implants would make in the dental community [6]. Therefore, based on their long-time scientific research and observations on tissue repair and osseointegration of commercially pure titanium implants in jaw bones of dogs they started further experimental work aiming at the development of surgical and prosthetic protocols for the rehabilitation of mandibular completely edentulous patients [7]. Their primary aim was the prosthetic reconstruction of severely resorbed mandibular alveolar processes. Today, more than 50 years since the first placement of implants for the rehabilitation of an edentulous patient, it is known that complete arch implant–supported fixed dental prostheses have very high long-term success rates [8–10]. The first prostheses were supported by five implants placed between the mental foramina and were fabricated by a metal framework with beads and other retentive elements, so as to retain the heat-processed polymethyl methacrylate (PMMA) resin and ten to twelve denture teeth. As the implants were placed between the mental foramina, the posterior segments on each side were cantilevered. A biomechanical analysis was attempted at that time, without however taking into consideration differences in arch shapes, interarch distance, length of implants, and length of cantilevers (Figures 18.6–18.7) [6].

This type of prosthesis has been widely known as a *hybrid prosthesis*, as it has the characteristics of two types of prostheses. It is made of multiple materials used in a traditional removable complete denture, while at the same time, it is fixed on the osseointegrated implants through a metal framework and abutments, and therefore can only

Figures 18.6–18.7 The metal framework of a mandibular hybrid prosthesis with beads and rods for the retention of PMMA resin. The hybrid prosthesis intraorally.

be removed by the dentist. According to the 9th edition of the Glossary of Prosthodontic Terms (GPT-9), the term "*hybrid prosthesis*" is *a nonspecific term applied to any prosthesis that does not follow conventional design; frequently used to describe a dental prosthesis that is composed of different materials, types of denture teeth (porcelain, plastic, composite resin), variable acrylic denture resins, differing metals or design, etc.; may refer to a fixed dental prosthesis, removable dental prosthesis, or maxillofacial prosthesis* [11]. Most often, a hybrid prosthesis consists of an alloy framework with retentive elements (i.e. beads, rods, loops, etc.), supported by the implants. These retentive elements are necessary, because acrylic polymers cannot bond to metal, at least without surface treatment. This fact necessitates the incorporation of the retentive elements to mechanically interlock the processed acrylic. The denture teeth that are used in these restorations are chemically bonded with the PMMA resin retained on the alloy framework. This type of prosthesis is also known as the "Toronto bridge," due to a series of clinical studies that Professor George Zarb and his colleagues conducted on that subject in the 1980s and 1990s, at the University of Toronto [12–14].

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Removable overdentures supported by dental implants have also been utilized for the treatment of complete edentulism, as they are less expensive than their fixed counterparts, due to the smaller number of implants required for their support and the lower cost of the involved materials and laboratory procedures needed for their fabrication [15–17]. Additionally, they are preferred when patients have limited bone volume at the host sites, and they are unwilling to proceed with bone grafting procedures, due to financial or other restrictions. Moreover, a consensus conference in the early 2000s has advocated that implant–supported overdentures should be considered as the first prosthetic treatment of choice in case of complete edentulism in the mandible and should always be presented to edentulous patients as a better alternative, given that there are no contraindications for implant placement [18] (Figures 18.8–18.13).

Figures 18.8–18.9 Loss of facial support due to extensive residual alveolar ridge resorption in an elder patient.

Figures 18.10–18.11 Two implants placed in the mandibular canine regions and an implant–supported complete denture fabricated to restore function and facial support.

It should also be mentioned that the selection of a removable complete denture as a treatment option, is many times necessitated because of the substantial loss of volume of the residual alveolar ridges and loss of lip support, which can be better served by removable prostheses. Finally, this type of prosthesis is many times indicated in cases of geriatric patients and/or patients with diminished manual dexterity, due to Parkinson's disease, stroke, arthritis, peripheral neuropathy, etc., which prevent the application of proper oral hygiene measures [19]. These measures, in the cases of hybrid prostheses, are quite demanding, involving the use of interproximal brushes, floss threaders, and oral irrigator units.

Prognosis and Maintenance of Metal-acrylic Hybrid Prostheses

In the initial era of implant prosthodontic therapy, the use of PMMA acrylic resin in combination with denture teeth was considered as an advantage, due to the absorption of occlusal forces, which would not be transmitted to

Figures 18.12–18.13 Maxillary complete denture and implant–supported mandibular overdenture in place. Restored lip support and vertical dimension of occlusion.

the supporting implants. To compensate for the stark difference in expected biomechanical behavior between the periodontal ligament compared to implants, Brånemark recommended that implants and natural teeth not be used as abutments for the same fixed prosthesis, that implants be restored with resilient restorative materials, and that prostheses be planned with a retrievable design [20]. However, acrylic denture teeth wear down much faster than enamel and porcelain (Figures 18.14–18.17). As a result, long-term occlusal stability is very often compromised, with deleterious effects in the function of the stomatognathic system and the prognosis of the restoration. A clinical study by Ohlmann et al. found that after a period of six months, the mean wear of acrylic teeth was $20.5 \mu m$. The wear was significantly higher in males than in females. Moreover, this study has shown that the wear was higher when the acrylic teeth had natural teeth as antagonists [21]. A very recent systematic review has concluded that even new-generation denture acrylic teeth wear substantially. Factors that play a role in this wear include the antagonists, the age and the sex of the patient, the type of the prosthesis, and the

Figures 18.14–18.15 Extensive wear of acrylic denture teeth after many years of service.

amount of the occlusal superficial layer removed during equilibration, at the delivery appointment [22]. Several studies in the past have also demonstrated similar results [23–25]. These studies have also shown that it is always better to use the same restorative materials in opposing arches.

The cumulative implant survival rates for metal-acrylic hybrid prostheses range between 87.9 and 100% for followup periods of 5–10 years and 78.3–98.9% for 10 years or more. Cumulative prosthetic survival rates are 93.3–100% and 82–100%, for periods of 5–10 years and 10 years or more, respectively [26]. Bone loss around implants supporting hybrid prostheses has been reported to be greater than 2mm in 20.1% of the cases after a 5-year period, and in 40.3% of the cases after 10 years [27]. Prosthetic complications in these prostheses usually include loosening or fracture of fastening screws, fracture of the PMMA denture acrylic resin, as well as fracture or debonding of acrylic resin teeth (Figures 18.18–18.19) [26]. A systematic review has reported that screw loosening or fracture occurs at rates of 10.4 and 20.8% after periods of 5 and 10 years, while chipping or fracture of veneering materials occurs at a rate of 30.3% after 5 years and 66.6% after 10

Figures 18.16–18.17 Hybrid metal-acrylic bar-retained removable implant prosthesis with gold occlusal surfaces to provide stable occlusal support and circumvent high wear and fracture rate of acrylic resin.

years [27]. Framework fractures have also been reported, but at a rate of 8.8% after a period of 15 years [28]. The highest incidence of prosthetic incidences was reported in a retrospective study by Krennmair et al., where 256 complications were recorded in 42 individuals, after five years of service [29]. As already mentioned, phonetic and esthetic issues have also been reported by patients treated with hybrid prostheses [30]. Esthetic deficiencies due to progressive deterioration of the resin material of the prosthetic base and of the denture teeth were reported at a 9% rate [28].

Bacterial Adhesion to Metal-acrylic Hybrid Prostheses

Apart from the fact that denture teeth wear down, classic hybrid prostheses are associated with inherent problems of PMMA denture acrylics, namely weak strength, residual monomer content, low dimensional stability, water

Figures 18.18–18.19 Fractured and debonded acrylic resin denture teeth of metal acrylic hybrid fixed implant prosthesis with limited prosthetic space, replaced with new denture teeth.

absorption, as well as color changes. Internal and external porosity in heat-processed acrylic resins is one additional disadvantage. Porosity usually results from the presence of residual monomer, which may boil if the peak temperature of the resin rises, due to the exothermic reaction of the polymerization, above the boiling point of monomer [31]. Then, the evaporation of the unreacted monomer may produce porosity, which can compromise esthetic properties and physical characteristics, adversely affecting hygiene. Retention of fluids and harboring of micro-organisms in the porous surface of PMMA acrylics may negatively impact the health of the peri-implant tissues [32]. Recent studies have demonstrated that acrylic resins are prone to microbial colonization, with anaerobe species prevailing over aerobes. This research has shown that the most predominant bacteria in acrylic resins were actinomyces, oral streptococci, and Enterobacteriaceae [33, 34]. Bacteria present possible threats to treatment success, as they can lead to

peri-implant mucositis and peri-implantitis. Peri-implant mucositis has been found to be present in 80% of the cases, while peri-implantitis was reported to be present in 56% of the cases [35]. Previous research has shown that surface irregularities, voids, or pits can be colonized as fast as 30 minutes from the moment a prosthesis is placed in the oral cavity [36, 37]. Surface roughness significantly increases the available surface area for the attachment of bacteria and offers support for adhesion [38].

Bacterial adhesion starts at sites protected from shear forces and is driven by hydrophobic, electrostatic, and van der Waals forces, which can take bacteria in a proximity with the acquired pellicle on implant surfaces [39, 40]. Bacteria's direct adhesion to the proteins of the acquired pellicle leads to the establishment of an irreversible attachment. This is a critical point as next, upregulation of bacterial activity and migration on implant surfaces is noticed. From that moment on, a maturation of dental plaque and an increase in the diversity of the bacterial community is observed [41–43]. In the initial stages of biofilm formation, gram-positive cocci, non-motile bacilli, as well as a limited number of gram-negative anaerobic species dominate supra- and sub-gingival plaque [44, 45]. Different bacteria have been identified by numerous studies in the dental literature. It has also been observed that bacterial species have some dynamic specific interrelationships which may produce shift patterns in the colonization process [46]. That explains the fact that a discrepancy both in the quality and quantity of peri-implant microbiota is very often noticed between screened populations [47].

Besides material's surface roughness, other surface properties may contribute to a stronger or weaker adhesion too. These primarily include: (i) surface topography, (ii) surface charge density, (iii) surface wettability. Other environmental factors, such as the fluid dynamics and bacterial mobility also affect bacterial adhesion on dental material surfaces [48]. (i) *Surface topography* is a term that is related to surface roughness, as it refers both to surface roughness and the profile shape. Bacteria can sense mechanical cues related to surfaces. Generally, surface microscale topography, in the size order of bacteria can impact their adhesion via hydrodynamics. Surface topography characteristics in the nanoscale affect bacterial attachment through deformation of cell membrane, physicochemical forces, and chemical gradients [49]. Furthermore, it has been demonstrated that the expression of bacterial adhesions can be modified when the surface topography is altered [50, 51]. Research has shown that bacterial attachment is enhanced when material surface has characteristic features larger than a single bacterium, as the latter can find shelter and greater available contact area, [52, 53] (ii) *surface charge density* of the dental material affects bacterial adhesion, as

electrostatic interactions and van der Waals forces are contributing greatly in this process [54]. According to published data, bacterial adhesion is easier on surfaces that are positively charged, as usually bacteria have a negative charge on their cell wall surfaces, due to the presence of amino, carboxyl, and phosphate groups [55–59]. Specifically, for PMMA acrylics which are positively charged, it was shown that adhesion and growth of Pseudomonas aeruginosa was twice as much, compared to negatively charged surfaces [60]. Similar results regarding *Pseudomonas*, *Staphylococcus aureus*, and *Escherichia coli* have been reported by other researchers too [61]. It should be mentioned, however, that there is evidence showing that some bacteria have the ability to adhere to negatively charged surfaces through the help of lipopolysaccharides (LPS) or appendages, like fimbriae, existing on their surfaces [62, 63], (iii) *surface wettability* is defined by how much a liquid phase is spread over a solid surface. Therefore, an increased contact area between a liquid and a solid shows enhanced surface wettability, and it is one of the major properties determining solid–liquid interactions in biological systems [48]. Material's surface energy and roughness affect wettability, and several models have been developed to demonstrate the association between bacterial adhesion and surface energy. For that purpose, a thermodynamic approach has shown that bacteria with hydrophilic cell surfaces tend to favor hydrophilic materials, while bacteria with hydrophobic cell surfaces show a preference to hydrophobic materials [64]. As mentioned previously, van der Waals and Coulomb interactions affect bacterial adhesion to material surfaces. In that sense, the distance between bacteria and surfaces, as well as the ionic strength of the surrounding fluid is very important. Generally, as ionic strength increases, so does the bacterial adhesion to materials [65]. The fact that PMMA denture acrylic presents increased porosity and favors bacterial adhesion combined with the difficult access for hygiene under hybrid prostheses has led to the use of highly polished metal at the gingival surface (Figures 18.20–18.21).

Dental alloys can be highly polished and therefore present minimal surface roughness, which favors bacterial adhesion. Research has shown that resin and synthetic materials are more prone to bacterial adhesion than metals, especially chrome–cobalt [4]. Therefore, if a hybrid prosthesis is chosen as a treatment modality, a proper convex design with metal finishing should be selected. Moreover, space between the prosthesis and the residual alveolar ridge should be provided to facilitate oral hygiene measures. This space, which is required for dental hygiene, may pose a problem if used in the maxilla, as it is many times associated with speaking problems, due to the air passage between the residual alveolar ridge and the

Figures 18.20–18.21 Implant supported hybrid prosthesis with denture teeth and PMMA acrylic resin at the coronal surface and highly polished metal at the gingival surface.

prosthesis. Some esthetic concerns have also been reported in the literature, due to the aforementioned space [30].

Although highly polished metal presents smaller bacterial adhesion than acrylic resin, dental ceramics demonstrate an even better behavior than dental alloys. An in vitro study has demonstrated that artificial plaque synthesized of *Streptococcus sobrinus* and glucans was retained significantly less on dental ceramics than on metals and resins [66].

The Shift to Metal–Ceramics

With the advent of new methods and materials in the early 1990's, full-arch implant–supported prostheses shifted from denture acrylic PMMA to metal–ceramics in an effort to avoid the shortcomings of acrylic resin.

Although there is a lot of published evidence on the clinical outcome of hybrid prostheses and their supporting implants, there is not enough data on full arch metal– ceramic implant-retained restorations. However, as metal– acrylic restorations are currently rarely in use, it is very important for clinicians to know the technical and biologic complications of metal–ceramic implant–supported fixed complete dentures. This information is critical for treatment planning and design of the prostheses. There is a need for clinical studies with large sample sizes and long follow-up times, in order to draw some meaningful conclusions. A recent retrospective study that included 52 patients with both metal-acrylic and metal-ceramic full-arch fixed complete dentures found that while the 5-year and the 10 year failure rate for the metal-acrylic was 7.1 and 58.2% respectively, the corresponding failure rates for metalceramic full-arch prostheses were 0% [67].

Technical complications occur quite often in full-arch fixed implant–supported prostheses. These can be divided into two broad categories: minor and major complications. Minor complications include all these complications which are easily managed and can be usually fixed chairside. These include minor wear or chipping of the veneering material, loss of the screw access filling, and abutment- or prostheticscrew loosening. On the contrary, major complications include those complications that are quite serious and cannot be easily managed chairside. These include fracture of a big part of the veneering material, fracture of the framework, fracture of the abutment, fracture of a fastening screw, and fracture of an implant (Figures 18.22–18.26).

It should be indicated that the same retrospective study has found that more than 40% of the full-arch fixed implant– supported prostheses (for both the hybrid and the metalceramic) presented a complication in the first 5 years, while the estimated complication rates for the 10-year period was 98% [67]. Although a bigger number of complications was identified in that study in the metal–ceramic group (78.6%) than in the metal–resin (hybrid) group (42.3%), it should be mentioned that more metal-ceramic (55) than metal–resin (16) prostheses were included.

Modern CAD/CAM Framework Materials

By the start of the twenty-first century, framework materials had been traditionally metal-based. This was to provide implant–supported fixed dental prostheses with sufficient rigidity [68]. Gold alloy was considered the benchmark material for frameworks of implant–supported fixed dental prostheses. Due to the high cost associated with gold, a demand for less expensive options developed. This included titanium, which presented with a technically difficulty manufacturing process and higher framework dimension requirements, which subsequently affected the level of patient comfort [68]. The arrival of computer-aided design/ computer-aided manufacturing (CAD/CAM) provided an alternative manufacturing process that bypassed the shortcomings of the traditional process (waxing/investing/

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Figures 18.22–18.23 (Top) Multiple fractures of the veneering material in a non-esthetic area. This can be polished with fine-grit diamonds and abrasive points. (Bottom) Fracture of the framework is one of the most serious technical complications. Although the occlusogingival and buccolingual size of the connector is appropriate (>3mm), the fact that the soldering metal did not reach the central part of the connector caused the fracture.

casting) and the associated procedural errors. With CAD/ CAM technology, large frameworks could now be manufactured precisely from homogeneous blanks of various high-strength materials [68]. This included zirconia, which has now been in use in dentistry for about two decades, dominating the market owing to its high strength, low cost, and natural white color [69].

Figures 18.24–18.25 Fracture of an abutment screw. In some cases, these screws can be retrieved by a counterclockwise motion with an explorer. If this is not possible, an ultrasonic scaler can be employed, taking care not to approach the internal threads of the implant, as they can be damaged. Some retrieval systems are also available from implant companies.

Figure 18.26 Fracture of two implants in a patient with parafunctional habits. Angular bone loss around the distal implant can also be observed. This fact may contribute to the fracture, especially if the end of the fastening screw coincides with the new bone level.

Zirconia-based Prostheses

Zirconia, or zirconium dioxide, has been predominantly used in dentistry in the form of yttria-stabilized tetragonal zirconia polycrystal (Y-TZP). With a flexural strength of 900–1400MPa, zirconia is considered the strongest of all dental ceramics presently available [70]. Early zirconia materials were quite opaque, so full contour ceramic veneering was employed to fulfill esthetic requirements. With little understanding of appropriate veneering protocols for zirconia compared to the long-standing experience with PFM, the primary clinical complication associated with porcelain-fused-to-zirconia (PFZ) was the substantially high rate of fracture or chipping of veneered porcelain, at a range of 15–54% documented in the literature, while framework fracture was reported at less than 1% [69].

A careful selection of good-quality zirconia blanks, optimized rates of heating and cooling during the firing stage, and digital rather than physical cut-back techniques effectively reduced the rate of ceramic chipping when veneered over zirconia [69]. Another solution was to restrict veneering to non-occlusal loading areas such as facially and gingivally, or to eliminate the veneer material entirely by using monolithic zirconia, with the addition of stains for tooth and gingival shading, and internal coloration for improved esthetics [69]. The availability of various compositions of zirconia has greatly influenced the design of full-arch zirconia frameworks. The early 3 mol% yttria-stabilized tetragonal polycrystal (3Y-TZP) zirconia offered phenomenal strength but inadequate esthetics for full-contour restorations. This necessitated veneering with feldspathic porcelain. Improved framework designs involved a minimal facial cutback to maximize strength in occlusal stressbearing areas (Figures 18.27–18.29). In an effort to produce more translucent zirconia restorations, yttria content was altered to produce 4 and 5 mol% partially stabilized zirconia (4Y/5Y-PSZ). This zirconia does not exhibit the same strength and resistance to crack propagation as 3Y-TZP and is contraindicated for long-span multiple-unit prostheses. Further improvements, however, brought multilayer zirconia blanks to the scene, available from multiple manufacturers. These materials present with various degrees of translucency and strength built into the same blank, maximizing esthetics toward the occlusal surfaces, while maintaining high overall strength to serve most restorative indications. This allows an even more conservative framework design with no cut-back while using internal and external stains to produce characterization and the appearance of roots and artificial gingiva (Figures 18.30–18.32).

Comparing technical complications in metal–acrylic and zirconia-based prostheses, Barootchi et al. found that metal-acrylic prostheses presented a higher rate of

Figures 18.27–18.29 Maxillary full arch fixed implant– supported prosthesis consisting of a screw-retained abutmentlevel 3Y-TZP zirconia framework with minimal facial porcelain layering. Cutback was completed digitally before milling of the zirconia framework.

minor complications, such as single tooth fracture or chipping, as well as a greater tendency for delayed complications. While zirconia fixed implant–supported prostheses come with higher initial costs, there is an overall reduction in complications and higher survival rates than metal–acrylic hybrids [71]. The shortcomings with zirconia are certainly of importance as well, including

Figures 18.30–18.32 Maxillary full arch fixed implant– supported prosthesis fabricated as a screw-retained abutmentlevel full contour multilayer (3Y-TZP/5Y-PSZ) zirconia framework with internal and external staining. *Source:* Courtesy of Dr. Esteban Molina, DDS, CAGS, Boston, MA.

the heavy weight, and the difficult adjustment and polishing of the framework. Delucchi et al. reported complications, such as ceramic veneer chipping (Figures 18.33 and 18.34) and the less frequent failure of the framework [72]. According to some authors, a nonlayered monolithic zirconia could solve the high incidence of ceramic chipping, by eliminating the interface between the zirconia framework and veneering ceramic (Figures 18.30–18.32) [72]. Table 18.1 summarizes the advantages and disadvantages of zirconia as a framework material for full-arch fixed implant prostheses.

Figures 18.33–18.34 Chipping of facial ceramic veneering at the canine incisal tip, on minimally layered full arch fixed implant–supported zirconia prosthesis in a patient with chronic bruxism after one year of follow-up.

In a study comparing porcelain-veneered zirconia frameworks (PVZ) to monolithic zirconia with nonfunctional porcelain veneering (MZ), Caramês et al. suggests that zirconia is a robust material for fixed implant–supported full-arch frameworks, with low reported rate of technical complications, high survival rate of the prosthesis and short/medium term success of the implants involved [73].

Bacterial growth on zirconia surfaces has been a controversial topic. An in vitro study comparing feldspar-based porcelain, CoCr alloy, commercially pure titanium grade IV, and yttria-stabilized zirconia showed the highest values of formation and absorbance for biofilms grown on CoCr alloys when compared to the other test materials for 24–48 hours, while no significant differences were found between biofilms grown on zirconia, porcelain, and titanium [74]. Research has also shown that the bacterial community colonizing zirconia and titanium were similar to those found in the natural dentition. These results suggest a selective adhesion of different microflora for either titanium or zirconia surfaces [75]. Furthermore, an in vivo study has demonstrated that zirconia exhibited low plaque accumulation compared to lithium disilicate glass–ceramic after 24 hours [76].

Table 18.1 Advantages and disadvantages of using zirconia in fixed implant–supported prostheses.

Milled Titanium Frameworks

While casted titanium frameworks have been commonly used as alternatives to traditional castings, they can also be milled using CAD/CAM technology. The beneficial properties of titanium for use as a framework material include biocompatibility, good resistance to corrosion, and high mechanical properties, as well as possibly better passive fit to the implants. A passive fit is critical, and a lack thereof may cause screw loosening and consequential fractures in prosthetics components. Although laser-welded titanium frameworks have been used too, they were associated with significantly more complications, and these were

increasing after the third year of function. Laser-welded areas on titanium framework could possibly act as the weakest link. Computer Numerical Controlled (CNC) milling techniques to fabricate one-piece titanium frameworks were introduced in order to make up for this disadvantage. Through such procedures, there is a better control of distortion, compared to conventional casting fabrication [77].

Surface roughness, contact angle, bacterial count, bacterial adherence, biofilm thickness, and bacterial distribution on zirconia and titanium surfaces have been studied by a meta-analysis of 39 in vitro and in vivo studies [78]. It was shown that results in the literature were inconsistent, as 10 studies concluded that zirconia accumulated less initial oral biofilm parameters, 16 trials showed negligible intermaterial differences, and only one study demonstrated that the most biofilm adhered to zirconia [78]. This metaanalysis also did not yield any conclusive differences between zirconia and titanium for the remaining parameters. An important point is that studies presented a substantial degree of heterogeneity, and thus no clear preference for the use of zirconia or titanium can be inferred [78]. Research is still ongoing, and a promising five-year prospective randomized controlled trial commenced by Jia et al. in 2020, comparing the peri-implant submucosal microbiota in arches with zirconia or titanium implant–supported fixed complete dental prostheses may shed light on this discussion [79]. With regards to soft tissue adhesion, animal studies showed that zirconia demonstrated connective tissue adhesion that was like that seen on the machined titanium surface while exhibiting limited plaque formation and improved esthetics [70].

High-strength Polymers

Going back to Brånemark's recommendations, specifically that implants be restored with resilient restorative materials, clinicians have since tried to replicate the damping effect of the periodontal ligament with various implant, abutment, and restorative systems. Researchers and clinicians have then incorporated various resilient and viscoelastic components to be part of implant prosthetics [80–82]. These components have predominantly been composed of polymeric materials to emulate the viscoelasticity of the PDL. Historically, this has been the approach in dentistry when using acrylic-based composite resins. More recently, several high-strength CAD/CAM polymers have emerged that are more suitable for this role.

High-strength CAD/CAM polymers include fiberreinforced polymers such as composite resin, epoxy resin, or acrylic resin reinforced by glass fibers or carbon fibers, and a class of polymers known as high-performance polymers, which are already widely used materials in aerospace and in orthopedic surgery. Examples of high-performance polymers include PEEK and PEKK, fiber-reinforced or non-reinforced, and this class of polymers has been in increasing use in dentistry as CAD/CAM materials for applications in fixed and removable prostheses. As these materials have been introduced recently in the dental market, a lack of studies on their performance as framework materials for full arch fixed implant prostheses, is noted [72]. Carbon fiber-reinforced composite (CFRC) frameworks have been employed recently for full-arch implant prostheses. Research shows that when CFRC frameworks are compared with cast metal frameworks, the former exhibit greater implant survival, less bone resorption, and no prosthetic complications [72]. CFRC boasts the benefits of high rigidity and tenacity comparable to metal, excellent fatigue resistance, good energy dissipation capacity, low cost, low weight, and a compatible surface for veneering acrylic resin to adhere to. This may explain the low occurrence of chipping of the veneering material in this study [72]. The combination of full-arch fiberreinforced resin fixed prostheses on four ultrashort implants in patients with highly atrophic jaws has also been studied. An average follow-up of 19.5 months has shown that 97.25% success and survival rates were reached, with minimal bone loss. Although no chipping or fracture of the prostheses was observed, these prostheses were bonded to telescopic copings that abutted the implants, and the main complication involved decementation [83].

Prosthetic Design – Impact on Maintenance

Early complete arch fixed implant prosthesis was a treatment modality employed for patients who have been completely edentulous and were initially restored with removable complete dentures, which are quite often associated with a significant loss of soft and hard tissues. This necessitated the prosthesis to restore both teeth and gingivae. Later on, as implants were used more often for the rehabilitation of patients whose teeth with questionable or hopeless prognosis were being extracted, as part of a restorative treatment plan, alveolar bone resorption was not advancing to the same degree [84–88]. Therefore, the need to replace, with implant–supported fixed prostheses, not only teeth but also the neighboring soft tissues was smaller. As a result, the design of the prostheses changed, with an impact on dental hygiene. Today, full-arch fixed implant prostheses can restore [89]:

1) Only the anatomic crowns of the missing natural teeth, where there has usually been minimal loss of soft and hard tissues. In these cases, the implants can be placed in an ideal position, mimicking the roots of the lost teeth. Proper planning and execution of implant placement, along with gingival contouring, usually results in a natural esthetic outcome, which is of utmost importance in the anterior esthetic region. The prosthesis-tissue interface is most coronal in these cases, and the intaglio surface is usually convex and festooned. These qualities provide for an accessible design for the cleansability of full-arch fixed implant prostheses (Figures 18.35–18.36).

2) The anatomic crown and part of the root of the missing natural tooth, where there has been considerable bone loss, either due to advanced periodontal disease or bone resorption from previously performed extractions. In these cases, if no bone augmentation procedures are implemented, implants are placed more apically and more lingually than the original position of the roots of the teeth to be restored. Ideally, each one of the implants supporting the fixed complete denture should correspond to a tooth, to facilitate proper hygiene and esthetic result, especially in cases where exposure of both teeth and neighboring soft tissues exist. The patient must be informed that the teeth will look longer than normal, and the roots of the teeth will have to be mimicked to achieve a more natural result. If the patient objects to this outcome bone grafting should be planned to be

Figures 18.35–18.36 Screw-retained full arch fixed zirconia prosthesis, with minimum facial porcelain veneering, restoring the anatomical crowns of missing teeth.

performed prior to implant placement, after a careful evaluation of the benefits and risks [90, 91], or artificial gingiva would need to be employed (Figures 18.37–18.42).

3) the anatomic crowns of natural missing teeth and a part of the neighboring soft tissues, usually due to extensive bone resorption after teeth extraction and/or prolonged use of removable complete dentures. These fixed complete dentures resemble the hybrid prostheses that have been described earlier but are made of metal–ceramic

Figures 18.37–18.42 Screw-retained full-arch fixed implant prosthesis restoring anatomic crowns and parts of the roots.

Figures 18.37–18.42 (Continued)

or zirconia. In these prostheses, implants are placed also in a more apical and lingual position, than the original position of the natural teeth. This fact complicates the biomechanical behavior of the prosthesis, as prosthesis-to-implant ratios are unfavorable, and greater moments of force are generated at the prosthesis/abutment/implant connection. A proper occlusal scheme should be planned to minimize technical complications. However, in contrast to the previous category, each implant does not necessarily have to

correspond to a tooth, as pink ceramics or zirconia are added, and the esthetics are not compromised. A careful framework design should be made to avoid technical complications related to unsupported ceramics. Additionally, highly skilled dental technologists should be employed, as matching soft tissues with pink ceramics is very challenging and usually requires additional firings if feldspathic porcelain is used. Additional problems include the increased dimensions of the metal framework, which retains more heat leading to ceramic fracture upon cooling. As these prostheses extend to the soft tissues of the residual alveolar ridge, careful planning should be made and instructions should be given to the patient to facilitate proper hygiene, which is crucial for long-term maintenance (Figures 18.43–18.45).

Expanding on this classification, the design of complete arch-fixed implant prostheses can be further categorized by prosthetic platform position, mode of retention, segmentation, and framework design (Table 18.2). The various combinations of designs are determined by existing patient condition, availability of hard and soft tissues, clinician preference, and laboratory systems available. Various configurations have evolved alongside the available technology and laboratory systems. Each design has a different impact on maintenance, and the most contemporary approaches are not well supported in the literature due to the lack of longterm follow-up and increasingly heterogenous studies. Figures 18.46–18.51 demonstrate various examples of design configurations based on the classification presented below.

● **Position of Prosthetic Platform:**

- Implant level (bone level implants and tissue level implants)
- Abutment level (screw-retained prosthesis over multiunit abutments, cement-retained prosthesis over stock or custom abutments)

Figure 18.43 Mandibular screw-retained fixed full arch implant metal-ceramic prosthesis restoring the anatomic crowns and neighboring gingival tissues.

Figures 18.44–18.45 Removal of the metal–ceramic fixed complete denture for peri-implant soft tissue evaluation. Minimal inflammation is noted on the lingual side of two implants. Good oral hygiene in combination with proper restorative materials helps minimize peri-implant inflammation.

● **Mode of Retention:**

- Screw-retained (e.g. metal–ceramic prostheses using UCLA abutments, NobelProcera® screw-retained fixed partial dentures with/without friction-fit titanium adapters)
- Cement-retained (e.g. framework cemented directly over stock or custom abutments – no screw access)
- Screw/Cement-retained (e.g. suprastructure cemented intraorally over screw-retained bar or individual crowns cemented over screw-retained framework with studs – no screw access)
- Screw/Cement-retained (e.g. screwmentable prosthesis cemented extraorally over stock or custom abutments, cylinders, or adapters with screw channels through prosthesis)

● **Segmentation:**

- One-piece prosthesis
- Segmented prosthesis

Soft tissue adherence and stability can be influenced by additional factors, such as prosthesis contour, platform switching, number of abutment disconnections from

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Table 18.2 Classification of full arch fixed implant prosthesis designs.

Figures 18.46–18.47 Maxillary and mandibular full arch fixed implant–supported rehabilitation utilizing screw-retained segmented facially veneered zirconia restoring teeth only extraorally cemented on titanium custom abutments (screwmentable). Screwmentable implant restorations combine the benefits of cement-retained and screw-retained restorations while avoiding the drawback of residual cement which may be a potential source of biological failure [80]. Segmented prostheses have the advantage of the option of replacing a segment rather than the whole arch if any mechanical or biological failure occurs.

Figures 18.48–18.49 Maxillary full arch fixed implant– supported rehabilitation utilizing segmented facially veneered zirconia restoring teeth only intraorally cemented on screwretained titanium custom abutments (cement-retained). Custom abutments in this case compared to the previous case are designed such that the prosthetic margin is no more than 1mm subgingival to provide adequate access for cleaning after intraoral cementation of suprastructure. Cement-retained prostheses can be considered to be esthetically superior from the patient's perspective due to the absence of visible screw channels. However, retrievability is difficult and there is a risk of residual cement in the peri-implant tissues [93].
Prosthetic Design – Impact on Maintenanc **373**

Figures 18.50–18.51 Maxillary full arch fixed implant– supported rehabilitation utilizing one-piece facially veneered zirconia restoring teeth and tissue screw-retained directly over multi-unit abutments (screw-retained). Multi-unit abutments allow for easier retrievability in cases of prosthetic screw failure [92]. Abutments can be easily replaced, if necessary, while risk of damage to implant platform due to screw failure is low.

implant, quantity and quality of available soft and hard tissues, and the presence of multi-unit abutments. Multi-unit abutments can potentially influence the bacterial pumping during micromotion by moving the microgap that results from inherent inaccuracies of long-span restorations further away from the bone crest [90, 92]. Pre-prosthetic bone recontouring can be done to allow for non-plaque-retentive intaglio surface to residual ridge interface [91].

On the other hand, bone reduction for esthetic reasons to hide the artificial to natural gingiva transition zone

may move the interface further apically and make hygiene more difficult. In those cases, a careful pre-prosthetic planning is necessary to visualize the need for soft tissue replacement and to assess the esthetics of the planned prosthesis (Figures 18.52–18.55). After the prosthetically driven placement of implants (Figures 18.56 and 18.57), a provisional restoration is delivered, and esthetics, function, and oral hygiene can be carefully evaluated (Figures 18.58 and 18.59). It is the restorative dentist's duty to determine the fine line between optimal esthetics and adequate access for hygiene. Although modern materials, like zirconia, do not retain as much plaque as PMMA, the patient's ability to use effective oral hygiene practices is the most important prerequisite for a longlasting result. The definitive implant–supported prosthesis is fabricated only after a careful assessment of patient's function and their ability to clean the provisional prosthesis effectively (Figures 18.60–18.62).

Figures 18.52–18.53 Patient with terminal maxillary dentition planned for implant rehabilitation.

Figures 18.54–18.55 3D-printed removable complete denture prostheses designed digitally against ideal mandibular occlusion and used as a prototype for implant planning. Flangeless and flanged design used to visualize the need for soft tissue replacement and to mark the exaggerated smile line.

Conclusions

Various combinations of materials are available for complete arch-fixed implant–supported prostheses. At the time of this writing, zirconia, and titanium frameworks with various designs of ceramic veneering or zirconia suprastructures appear to be the predominant materials in use due to mechanical and biological predictability and cost-effectiveness. CAD/CAM technology has also unlocked the possibility of utilizing high-strength, highperformance polymers which may present a promising future alternative. Design options range across implantlevel or abutment-level, screw-retained or cement-retained, one-piece or segmented, and tooth-only replacement or tooth-tissue replacement based on clinical scenario, patient desire, and clinician preference.

Materials and prosthesis configurations all have an impact on maintenance strategies and can be considered prognostic factors for the success of fixed implant rehabilitation. As such, careful planning is needed to modify these factors starting with the desired tooth position based on esthetics, phonetics, and function, prosthetic margin position based on dynamic esthetics and restorative space, and implant position based on anatomical factors, esthetic parameters, and mechanics. Attention to intaglio surface

Figures 18.56–18.57 Prosthetically driven implant placement, simulating the use of angled multi-unit abutments to maximize native bone support of implants while achieving a screwretained prosthesis with vertical prosthetic margin position hidden beyond the smile line.

topography and the need for sculpting to allow access for maintenance should be a major concern in every step of the treatment including planning, surgical phase, provisionalization, finalization, and recare. Moreover, the consideration of opposing dentition, material wear, and occlusal stability is crucial for long-term success and effective maintenance. Of utmost importance is a strict restorative and periodontal maintenance and re-care program, in order to ensure a long-term successful result. In cases of non-compliant patients, other therapeutic approaches should be considered, as implant supported restorations will ultimately fail (Figures 18.63–18.65).

Figures 18.58–18.59 Tooth extraction, bone reduction, and immediate implant placement completed. Multi-unit abutments inserted at time of implant placement and were not removed throughout treatment (one abutment one-time concept). Screw-retained provisional prosthesis inserted over multi-unit abutments. Provisional demonstrated good esthetics and function, including the position of the transitional zone of artificial to natural gingiva hidden beyond the smile line. Moving the transition line apically for esthetics does compromise the difficulty to access the prosthesis-tissue interfaces for proper home care and maintenance.

Figures 18.60–18.62 Full arch screw-retained monolithic 3Y-TZP zirconia framework with facial cutback was milled and veneered with pink and white ceramic on the facial surface to achieve adequate esthetics. Convex intaglio surface of prosthesis achieved for interfacing with residual ridge. Definitive maxillary full arch fixed implant prosthesis and mandibular posterior full coverage restorations delivered after a careful evaluation of patient's function and ability to use effective oral hygiene measures.

Figures 18.63–18.65 Patient restored with a mandibular full-arch implant supported prosthesis, which failed after 10 years, due to patient's negligence to follow oral hygiene measures and maintenance program.

References

- **1** Moraschini, V., Poubel, L.A., Ferreira, V.F., and Barboza, E.S. (2015). Evaluation of survival and success rates of dental implants reported in longitudinal studies with a follow-up period of at least 10 years: a systematic review. *International Journal of Oral and Maxillofacial Surgery* 44 (3): 377–388. [https://doi.org/10.1016/j.ijom.](https://doi.org/10.1016/j.ijom.2014.10.023) [2014.10.023.](https://doi.org/10.1016/j.ijom.2014.10.023) Epub 2014 Nov 20. PMID: 25467739.
- **2** Charyeva, O., Altynbekov, K., Zhartybaev, R., and Sabdanaliev, A. (2012). Long-term dental implant success and survival--a clinical study after an observation period up to 6 years. *Swedish Dental Journal* 36 (1): 1–6. PMID: 22611899.
- **3** Malo, P., de Araújo Nobre, M., Lopes, A. et al. (2011). A longitudinal study of the survival of All-on-4 implants in the mandible with up to 10 years of follow-up. *Journal of the American Dental Association (1939)* 142 (3): 310–320. [https://doi.org/10.14219/jada.archive.2011.0170.](https://doi.org/10.14219/jada.archive.2011.0170) PMID: 21357865.
- **4** Kozmos, M., Virant, P., Rojko, F. et al. (2021). Bacterial adhesion of *Streptococcus mutans* to dental material surfaces. *Molecules* 26 (4): 1152. [https://doi.org/10.3390/](https://doi.org/10.3390/molecules26041152) [molecules26041152](https://doi.org/10.3390/molecules26041152).
- **5** Busscher, H.J., Rinastiti, M., Siswomihardjo, W., and van der Mei, H.C. (2010). Biofilm formation on dental restorative and implant materials. *Journal of Dental Research* 89 (7): 657–665. [https://doi.](https://doi.org/10.1177/0022034510368644) [org/10.1177/0022034510368644.](https://doi.org/10.1177/0022034510368644) Epub 2010 May 6.
- **6** Brånemark, P.I., Zarb, G., and Albrektsson, T. (1992). *Tissue-integrated Prostheses: Osseointegration in Clinical Dentistry*. Chicago: Quintessence.
- **7** Brånemark, P.I. (1983). Osseointegration and its experimental background. *The Journal of Prosthetic Dentistry* 50 (3): 399–410. [https://doi.org/10.1016/](https://doi.org/10.1016/s0022-3913(83)80101-2) [s0022-3913\(83\)80101-2.](https://doi.org/10.1016/s0022-3913(83)80101-2) PMID: 6352924.
- **8** Papaspyridakos, P., Mokti, M., Chen, C.J. et al. (2014). Implant and prosthodontic survival rates with implant

fixed complete dental prostheses in the edentulous mandible after at least 5 years: a systematic review. *Clinical Implant Dentistry and Related Research* 16 (5): 705–717.<https://doi.org/10.1111/cid.12036>. Epub 2013 Jan 11. PMID: 23311617.

- **9** Papaspyridakos, P., Barizan Bordin, T., Kim, Y.J. et al. (2018). Implant survival rates and biologic complications with implant-supported fixed complete dental prostheses: a retrospective study with up to 12-year follow-up. *Clinical Oral Implants Research* 29 (8): 881–893. [https://doi.org/10.1111/clr.13340.](https://doi.org/10.1111/clr.13340) Epub 2018 Jul 24. PMID: 30043456.
- **10** Mori, G., Oda, Y., Sakamoto, K. et al. (2019). Clinical evaluation of full-arch screw-retained implant-supported fixed prostheses and full-arch telescopic-retained implant-supported fixed prostheses: a 5-12 year follow-up retrospective study. *Clinical Oral Implants Research* 30 (3): 197–205. <https://doi.org/10.1111/clr.13406>. Epub 2019 Feb 5. PMID: 30667092.
- **11** (2017). The glossary of prosthodontic terms: ninth edition. *The Journal of Prosthetic Dentistry* 117 (5S): e1–e105. [https://](https://doi.org/10.1016/j.prosdent.2016.12.001) [doi.org/10.1016/j.prosdent.2016.12.001.](https://doi.org/10.1016/j.prosdent.2016.12.001) PMID: 28418832.
- **12** Zarb, G.A. and Schmitt, A. (1990). The longitudinal clinical effectiveness of osseointegrated dental implants: the Toronto study. Part I: surgical results. *The Journal of Prosthetic Dentistry* 63 (4): 451–457. [https://doi.](https://doi.org/10.1016/0022-3913(90)90237-7) [org/10.1016/0022-3913\(90\)90237-7](https://doi.org/10.1016/0022-3913(90)90237-7). PMID: 2184231.
- **13** Zarb, G.A. and Schmitt, A. (1990). The longitudinal clinical effectiveness of osseointegrated dental implants: the Toronto Study. Part II: the prosthetic results. *The Journal of Prosthetic Dentistry* 64 (1): 53–61. [https://doi.](https://doi.org/10.1016/0022-3913(90)90153-4) [org/10.1016/0022-3913\(90\)90153-4](https://doi.org/10.1016/0022-3913(90)90153-4). PMID: 2200880.
- **14** Zarb, G.A. and Schmitt, A. (1992). Osseointegration and the edentulous predicament. The 10-year-old Toronto study. *British Dental Journal* 172 (4): 135. [https://doi.org/](https://doi.org/10.1038/sj.bdj.4807804) [10.1038/sj.bdj.4807804](https://doi.org/10.1038/sj.bdj.4807804). PMID: 1610456.
- **15** Mericske-Stern, R. (1998). Treatment outcomes with implant supported overdentures: clinical considerations. *The Journal of Prosthetic Dentistry* 79: 66–73.
- **16** Bergendal, T. and Engquist, B. (1998). Implant-supported overdentures: a longitudinal prospective study. *The International Journal of Oral & Maxillofacial Implants* 13: 253–262. 27.
- **17** Trakas, T., Michalakis, K., Kang, K., and Hirayama, H. (2006). Attachment systems for implant retained overdentures: a literature review. *Implant Dentistry* 15: 24–34.
- **18** Feine, J.S., Carlsson, G.E., Awad, M.A. et al. (2002). The McGill consensus statement on overdentures. Mandibular two-implant overdentures as first choice standard of care for edentulous patients. *Gerodontology* 19 (1): 3–4. PMID: 12164236.
- **19** Müller, F., Duvernay, E., Loup, A. et al. (2013). Implantsupported mandibular overdentures in very old adults: a randomized controlled trial. *Journal of Dental Research* 92 (12 Suppl): 154S–160S. [https://doi.org/10.1177/](https://doi.org/10.1177/0022034513509630) [0022034513509630.](https://doi.org/10.1177/0022034513509630)
- **20** Cherilyn, G. and Sheets, J.C.E. (1997). Tooth intrusion in implant-assisted prostheses. *The Journal of Prosthetic Dentistry* 77: 39.
- **21** Ohlmann, B., Rohstock, K., Kugler, J. et al. (2007). Influences on clinical wear of acrylic denture teeth: a pilot study. *The International Journal of Prosthodontics* 20 (5): 496–498. PMID: 17944339.
- **22** Mudliar, V.L., Tieh, M.T., Aarts, J.M. et al. (2022). Wear of modern denture teeth – a systematic review. *Oral* 2 (1): 95–111. [https://doi.org/10.3390/oral2010011.](https://doi.org/10.3390/oral2010011)
- **23** Ekfeldt, A. and Oilo, G. (1988). Occlusal contact wear of prosthodontic materials. An in vivo study. *Acta Odontologica Scandinavica* 46 (3): 159–169. [https://doi.](https://doi.org/10.3109/00016358809004762) [org/10.3109/00016358809004762.](https://doi.org/10.3109/00016358809004762) PMID: 3044001.
- **24** Ekfeldt, A. and Oilo, G. (1990). Wear of prosthodontic materials – an in vivo study. *Journal of Oral Rehabilitation* 17 (2): 117–129. [https://doi.org/10.1111/](https://doi.org/10.1111/j.1365-2842.1990.tb01400.x) [j.1365-2842.1990.tb01400.x](https://doi.org/10.1111/j.1365-2842.1990.tb01400.x). PMID: 2341953.
- **25** Ekfeldt, A., Fransson, B., Söderlund, B., and Oilo, G. (1993). Wear resistance of some prosthodontic materials in vivo. *Acta Odontologica Scandinavica* 51 (2): 99–107. [https://doi.org/10.3109/00016359309041154.](https://doi.org/10.3109/00016359309041154) PMID: 8498167.
- **26** Kwon, T., Bain, P.A., and Levin, L. (2014). Systematic review of short- (5-10 years) and long-term (10 years or more) survival and success of full-arch fixed dental hybrid prostheses and supporting implants. *Journal of Dentistry* 42 (10): 1228–1241. [https://doi.org/10.1016/](https://doi.org/10.1016/j.jdent.2014.05.016) [j.jdent.2014.05.016](https://doi.org/10.1016/j.jdent.2014.05.016). Epub 2014 Jun 26. PMID: 24975989.
- **27** Papaspyridakos, P., Chen, C.J., Chuang, S.K. et al. (2012). A systematic review of biologic and technical complications with fixed implant rehabilitations for edentulous patients. *The International Journal of Oral & Maxillofacial Implants* 27 (1): 102–110. PMID: 22299086.
- **28** Bozini, T., Petridis, H., Garefis, K., and Garefis, P. (2011). A meta-analysis of prosthodontic complication rates of implant-supported fixed dental prostheses in edentulous patients after an observation period of at least 5 years. *The International Journal of Oral & Maxillofacial Implants* 26 (2): 304–318. PMID: 21483883.
- **29** Krennmair, G., Seemann, R., Weinländer, M. et al. (2013). Clinical outcome and peri-implant findings of fourimplant-supported distal cantilevered fixed mandibular prostheses: five-year results. *The International Journal of Oral & Maxillofacial Implants* 28 (3): 831–840. [https://doi.](https://doi.org/10.11607/jomi.3024) [org/10.11607/jomi.3024.](https://doi.org/10.11607/jomi.3024) PMID: 23748316.
- **30** Rosén, A. and Gynther, G. (2007). Implant treatment without bone grafting in edentulous severely resorbed maxillas: a long-term follow-up study. *Journal of Oral and Maxillofacial Surgery* 65 (5): 1010–1016. [https://doi.org/](https://doi.org/10.1016/j.joms.2006.11.023) [10.1016/j.joms.2006.11.023](https://doi.org/10.1016/j.joms.2006.11.023). PMID: 17448855.
- **31** Phoenix, R.D. (2003). Denture base resins. In: *Phillips Science of Dental Materials*, 11e (ed. K.J. Anusavice), 730–733. St. Louis: Saunders.
- **32** Wolfaardt, J.F., Cleaton-Jones, P., and Fatti, P. (1986). The occurrence of porosity in a heat-cured poly (methyl methacrylate) denture base resin. *The Journal of Prosthetic Dentistry* 55 (3): 393–400.
- **33** Takeuchi, Y., Nakajo, K., Sato, T. et al. (2012). Quantification and identification of bacteria in acrylic resin dentures and dento-maxillary obturator-prostheses. *American Journal of Dentistry* 25 (3): 171–175.
- **34** Delgado, A.H.S., Carvalho, J., Borrecho, G. et al. (2018). *In situ* multispecies colonization of an acrylic resin: comparison to oral microbiome and potential for inflammatory response. *Contemporary Clinical Dentistry* 9 (3): 400–405.
- **35** Romanos, G.E. and Weitz, D. (2012). Therapy of periimplant diseases. Where is the evidence? *The Journal of Evidence-Based Dental Practice* 12 (3 suppl): 204–208.
- **36** Quirynen, M., Vogels, R., Pauwels, M. et al. (2005). Initial subgingival colonization of 'pristine' pockets. *Journal of Dental Research* 84 (4): 340–344. [https://doi.org/](https://doi.org/10.1177/154405910508400409) [10.1177/154405910508400409](https://doi.org/10.1177/154405910508400409). PMID: 15790740.
- **37** van Winkelhoff, A.J., Goené, R.J., Benschop, C., and Folmer, T. (2000). Early colonization of dental implants by putative periodontal pathogens in partially edentulous patients. *Clinical Oral Implants Research* 11 (6): 511–520. [https://doi.org/10.1034/](https://doi.org/10.1034/j.1600-0501.2000.011006511.x) [j.1600-0501.2000.011006511.x.](https://doi.org/10.1034/j.1600-0501.2000.011006511.x) PMID: 11168244.
- **38** Yoda, I., Koseki, H., Tomita, M. et al. (2014). Effect of surface roughness of biomaterials on Staphylococcus epidermidis adhesion. *BMC Microbiology* 14: 234. [https://doi.org/10.1186/s12866-014-0234-2P](https://doi.org/10.1186/s12866-014-0234-2)MID: 25179448; PMCID: PMC4161769.
- **39** Boks, N.P., Norde, W., van der Mei, H.C., and Busscher, H.J. (2008). Forces involved in bacterial adhesion to hydrophilic and hydrophobic surfaces. *Microbiology (Reading)* 154 (Pt 10): 3122–3133. [https://doi.org/10.1099/](https://doi.org/10.1099/mic.0.2008/018622-0) [mic.0.2008/018622-0](https://doi.org/10.1099/mic.0.2008/018622-0). PMID: 18832318.
- **40** Charalampakis, G. and Belibasakis, G.N. (2015). Microbiome of peri-implant infections: lessons from conventional, molecular and metagenomic analyses. *Virulence* 6 (3): 183–187. [https://doi.org/10.4161/](https://doi.org/10.4161/21505594.2014.980661) [21505594.2014.980661](https://doi.org/10.4161/21505594.2014.980661). Epub 2015 Feb 5. PMID: 25654499; PMCID: PMC4601299.
- **41** Gristina, A.G., Naylor, P., and Myrvik, Q. (1988–1989). Infections from biomaterials and implants: a race for the

surface. *Medical Progress through Technology* 14 (3-4): 205–224. PMID: 2978593.

- **42** Costerton, J.W., Montanaro, L., and Arciola, C.R. (2005). Biofilm in implant infections: its production and regulation. *The International Journal of Artificial Organs* 28 (11): 1062–1068. [https://doi.](https://doi.org/10.1177/039139880502801103) [org/10.1177/039139880502801103](https://doi.org/10.1177/039139880502801103). PMID: 16353112.
- **43** Al-Ahmad, A., Wiedmann-Al-Ahmad, M., Fackler, A. et al. (2013). In vivo study of the initial bacterial adhesion on different implant materials. *Archives of Oral Biology* 58 (9): 1139–1147. [https://doi.org/10.1016/j.archoralbio.2013.](https://doi.org/10.1016/j.archoralbio.2013.04.011) [04.011](https://doi.org/10.1016/j.archoralbio.2013.04.011). Epub 2013 May 18. PMID: 23694907.
- **44** Pye, A.D., Lockhart, D.E., Dawson, M.P. et al. (2009). A review of dental implants and infection. *The Journal of Hospital Infection* 72 (2): 104–110. [https://doi.org/](https://doi.org/10.1016/j.jhin.2009.02.010) [10.1016/j.jhin.2009.02.010.](https://doi.org/10.1016/j.jhin.2009.02.010) Epub 2009 Mar 28. PMID: 19329223.
- **45** Belibasakis, G.N. (2014). Microbiological and immunopathological aspects of peri-implant diseases. *Archives of Oral Biology* 59 (1): 66–72. [https://doi.org/10.1016/](https://doi.org/10.1016/j.archoralbio.2013.09.013) [j.archoralbio.2013.09.013](https://doi.org/10.1016/j.archoralbio.2013.09.013). Epub 2013 Oct 9. PMID: 24209597.
- **46** Peyyala, R. and Ebersole, J.L. (2013). Multispecies biofilms and host responses: "discriminating the trees from the forest". *Cytokine* 61 (1): 15–25. [https://doi.org/](https://doi.org/10.1016/j.cyto.2012.10.006) [10.1016/j.cyto.2012.10.006.](https://doi.org/10.1016/j.cyto.2012.10.006) Epub 2012 Nov 6. PMID: 23141757; PMCID: PMC4131722.
- **47** Pokrowiecki, R., Mielczarek, A., Zaręba, T., and Tyski, S. (2017). Oral microbiome and peri-implant diseases: where are we now? *Therapeutics and Clinical Risk Management* 13: 1529–1542. [https://doi.org/10.2147/](https://doi.org/10.2147/TCRM.S139795) [TCRM.S139795.](https://doi.org/10.2147/TCRM.S139795) PMID: 29238198; PMCID: PMC5716316.
- **48** Zheng, S., Bawazir, M., Dhall, A. et al. (2021). Implication of surface properties, bacterial motility, and hydrodynamic conditions on bacterial surface sensing and their initial adhesion. *Frontiers in Bioengineering and Biotechnology* 9: 643722.<https://doi.org/10.3389/fbioe.2021.643722>. PMID: 33644027; PMCID: PMC7907602.
- **49** Cheng, Y., Feng, G., and Moraru, C.I. (2019). Micro- and Nanotopography Sensitive Bacterial Attachment Mechanisms: a review. *Frontiers in Microbiology* 10: 191. [https://doi.org/10.3389/fmicb.2019.00191.](https://doi.org/10.3389/fmicb.2019.00191) PMID: 30846973; PMCID: PMC6393346.
- **50** Rizzello, L., Galeone, A., Vecchio, G. et al. (2012). Molecular response of Escherichia coli adhering onto nanoscale topography. *Nanoscale Research Letters* 7 (1): 575. [https://doi.org/10.1186/1556-276X-7-575.](https://doi.org/10.1186/1556-276X-7-575) PMID: 23078758; PMCID: PMC3552789.
- **51** Rizzello, L., Cingolani, R., and Pompa, P.P. (2013). Nanotechnology tools for antibacterial materials. *Nanomedicine (London, England)* 8 (5): 807–821. [https://doi.org/10.2217/nnm.13.63.](https://doi.org/10.2217/nnm.13.63) PMID: 23656266.
- **52** Scardino, A.J., Guenther, J., and de Nys, R. (2008). Attachment point theory revisited: the fouling response to a microtextured matrix. *Biofouling* 24 (1): 45–53. <https://doi.org/10.1080/08927010701784391>. PMID: 18066730.
- **53** Helbig, R., Günther, D., Friedrichs, J. et al. (2016). The impact of structure dimensions on initial bacterial adhesion. *Biomaterials Science* 4 (7): 1074–1078. <https://doi.org/10.1039/c6bm00078a>. Epub 2016 May 27. PMID: 27232637.
- **54** Renner, L.D. and Weibel, D.B. (2011). Physicochemical regulation of biofilm formation. *MRS Bulletin* 36 (5): 347–355.<https://doi.org/10.1557/mrs.2011.65>. PMID: 22125358; PMCID: PMC3224470.
- **55** Rijnaarts, H.H., Norde, W., Bouwer, E.J. et al. (1993). Bacterial adhesion under static and dynamic conditions. *Applied and Environmental Microbiology* 59 (10): 3255–3265. [https://doi.org/10.1128/aem.](https://doi.org/10.1128/aem.59.10.3255-3265.1993) [59.10.3255-3265.1993](https://doi.org/10.1128/aem.59.10.3255-3265.1993). PMID: 16349063; PMCID: PMC182446.
- **56** Kovačević, D., Pratnekar, R., Godič Torkar, K. et al. (2016). Influence of polyelectrolyte multilayer properties on bacterial adhesion capacity. *Polymers (Basel)* 8 (10): 345. [https://doi.org/10.3390/polym8100345.](https://doi.org/10.3390/polym8100345) PMID: 30974625; PMCID: PMC6432465.
- **57** Guo, S., Kwek, M.Y., Toh, Z.Q. et al. (2018). Tailoring polyelectrolyte architecture to promote cell growth and inhibit bacterial adhesion. *ACS Applied Materials & Interfaces* 10 (9): 7882–7891. [https://doi.org/10.1021/](https://doi.org/10.1021/acsami.8b00666) [acsami.8b00666.](https://doi.org/10.1021/acsami.8b00666) Epub 2018 Feb 26. PMID: 29437375.
- **58** Oh, J.K., Yegin, Y., Yang, F. et al. (2018). The influence of surface chemistry on the kinetics and thermodynamics of bacterial adhesion. *Scientific Reports* 8 (1): 17247. [https://](https://doi.org/10.1038/s41598-018-35343-1) [doi.org/10.1038/s41598-018-35343-1.](https://doi.org/10.1038/s41598-018-35343-1) PMID: 30467352; PMCID: PMC6250697.
- **59** Chen, C., Petterson, T., Illergård, J. et al. (2019). Influence of cellulose charge on bacteria adhesion and viability to PVAm/CNF/PVAm-modified cellulose model surfaces. *Biomacromolecules* 20 (5): 2075–2083. [https://doi.org/](https://doi.org/10.1021/acs.biomac.9b00297) [10.1021/acs.biomac.9b00297](https://doi.org/10.1021/acs.biomac.9b00297). Epub 2019 Apr 10. PMID: 30901196.
- **60** Gottenbos, B., Van Der Mei, H.C., Busscher, H.J. et al. (1999). Initial adhesion and surface growth of Pseudomonas aeruginosa on negatively and positively charged poly(methacrylates). *Journal of Materials Science. Materials in Medicine* 10 (12): 853–855. <https://doi.org/10.1023/a:1008989416939>. PMID: 15347964.
- **61** Zhu, X., Jańczewski, D., Guo, S. et al. (2015). Polyion multilayers with precise surface charge control for antifouling. *ACS Applied Materials & Interfaces* 7 (1): 852–861.<https://doi.org/10.1021/am507371a>. Epub 2014 Dec 19. PMID: 25485625.
- **62** Ueshima, M., Tanaka, S., Nakamura, S., and Yamashita, K. (2002). Manipulation of bacterial adhesion and proliferation by surface charges of electrically polarized hydroxyapatite. *Journal of Biomedical Materials Research* 60 (4): 578–584. [https://doi.org/10.1002/jbm.10113.](https://doi.org/10.1002/jbm.10113) PMID: 11948516.
- **63** Rzhepishevska, O., Hakobyan, S., Ruhal, R. et al. (2013). The surface charge of anti-bacterial coatings alters motility and biofilm architecture. *Biomaterials Science* 1 (6): 589–602.<https://doi.org/10.1039/c3bm00197k>. Epub 2013 Mar 4. PMID: 32481834.
- **64** Krasowska, A. and Sigler, K. (2014). How microorganisms use hydrophobicity and what does this mean for human needs? *Frontiers in Cellular and Infection Microbiology* 4: 112.<https://doi.org/10.3389/fcimb.2014.00112>. PMID: 25191645; PMCID: PMC4137226.
- **65** Sheng, X., Ting, Y.P., and Pehkonen, S.O. (2008). The influence of ionic strength, nutrients and pH on bacterial adhesion to metals. *Journal of Colloid and Interface Science* 321 (2): 256–264. [https://doi.org/10.1016/](https://doi.org/10.1016/j.jcis.2008.02.038) [j.jcis.2008.02.038](https://doi.org/10.1016/j.jcis.2008.02.038). Epub 2008 Feb 29. PMID: 18343395.
- **66** Kawai, K. and Urano, M. (2001). Adherence of plaque components to different restorative materials. *Operative Dentistry* 26 (4): 396–400. PMID: 11504440.
- **67** Papaspyridakos, P., Bordin, T.B., Kim, Y.J. et al. (2020). Technical complications and prosthesis survival rates with implant-supported fixed complete dental prostheses: a retrospective study with 1- to 12-year follow-up. *Journal of Prosthodontics* 29 (1): 3–11. [https://doi.org/10.1111/](https://doi.org/10.1111/jopr.13119) [jopr.13119.](https://doi.org/10.1111/jopr.13119) Epub 2019 Nov 21. PMID: 31650669.
- **68** Gallucci, G.O., Doughtie, C.B., Hwang, J.W. et al. (2009). Five-year results of fixed implant-supported rehabilitations with distal cantilevers for the edentulous mandible. *Clinical Oral Implants Research* 20 (6): 601–607. [https://doi.org/10.1111/j.1600-](https://doi.org/10.1111/j.1600-0501.2008.01699.x) [0501.2008.01699.x.](https://doi.org/10.1111/j.1600-0501.2008.01699.x) Epub 2009 Mar 11. PMID: 19302389.
- **69** Bidra, A.S., Rungruanganunt, P., and Gauthier, M. (2017). Clinical outcomes of full arch fixed implant-supported zirconia prostheses: a systematic review. *European Journal of Oral Implantology* 10 (Suppl 1): 35–45. PMID: 28944367.
- **70** Tetè, S., Mastrangelo, F., Bianchi, A. et al. (2009). Collagen fiber orientation around machined titanium and zirconia dental implant necks: an animal study. *The International Journal of Oral & Maxillofacial Implants* 24 (1): 52–58. PMID: 19344025.
- **71** Barootchi, S., Askar, H., Ravidà, A. et al. (2020). Longterm clinical outcomes and cost-effectiveness of full-arch implant-supported zirconia-based and metal-acrylic fixed dental prostheses: a retrospective analysis. *The International Journal of Oral & Maxillofacial Implants* 35 (2): 395–405.<https://doi.org/10.11607/jomi.7833>. PMID: 32142577.
- **72** Delucchi, F., De Giovanni, E., Pesce, P. et al. (2021). Framework materials for full-arch implant-supported rehabilitations: a systematic review of clinical studies. *Materials (Basel)* 14 (12): 3251. [https://doi.org/10.3390/](https://doi.org/10.3390/ma14123251) [ma14123251](https://doi.org/10.3390/ma14123251). PMID: 34204681; PMCID: PMC8231547.
- **73** Caramês, J., Marques, D., Malta Barbosa, J. et al. (2019). Full-arch implant-supported rehabilitations: a prospective study comparing porcelain-veneered zirconia frameworks to monolithic zirconia. *Clinical Oral Implants Research* 30 (1): 68–78. [https://doi.org/10.1111/clr.13393.](https://doi.org/10.1111/clr.13393) Epub 2018 Dec 30. PMID: 30521106.
- **74** Souza, J.C., Mota, R.R., Sordi, M.B. et al. (2016). Biofilm formation on different materials used in oral rehabilitation. *Brazilian Dental Journal* 27 (2): 141–147. [https://doi.org/](https://doi.org/10.1590/0103-6440201600625) [10.1590/0103-6440201600625](https://doi.org/10.1590/0103-6440201600625). PMID: 27058375.
- **75** de Freitas, A.R., Silva, T.S.O., Ribeiro, R.F. et al. (2018). Oral bacterial colonization on dental implants restored with titanium or zirconia abutments: 6-month follow-up. *Clinical Oral Investigations* 22 (6): 2335–2343. [https://doi.](https://doi.org/10.1007/s00784-018-2334-0) [org/10.1007/s00784-018-2334-0](https://doi.org/10.1007/s00784-018-2334-0). Epub 2018 Jan 18. PMID: 29349504.
- **76** Bremer, F., Grade, S., Kohorst, P., and Stiesch, M. (2011). In vivo biofilm formation on different dental ceramics. *Quintessence International* 42 (7): 565–574. PMID: 21716984.
- **77** Ortorp, A., Jemt, T., Bäck, T., and Jälevik, T. (2003). Comparisons of precision of fit between cast and CNC-milled titanium implant frameworks for the edentulous mandible. *The International Journal of Prosthodontics* 16 (2): 194–200. PMID: 12737254.
- **78** Kniha, K., Heussen, N., Modabber, A. et al. (2021). The effect of zirconia and titanium surfaces on biofilm formation and on host-derived immunological parameters. *International Journal of Oral and Maxillofacial Surgery* 50 (10): 1361–1374.<https://doi.org/10.1016/j.ijom.2021.01.021>. Epub 2021 Feb 19. PMID: 33618967.
- **79** Jia, P., Yang, J., Yue, Z. et al. (2020). Comparison of peri-implant submucosal microbiota in arches with zirconia or titanium implant-supported fixed complete dental prostheses: a study protocol for a randomized controlled trial. *Trials* 21 (1): 979. [https://doi.org/10.1186/](https://doi.org/10.1186/s13063-020-04853-7) [s13063-020-04853-7](https://doi.org/10.1186/s13063-020-04853-7). PMID: 33246481; PMCID: PMC7694361.
- **80** Sarafidou, K., Vasileiadi, G., Louvrou, M.K. et al. (2021). Screwmentable implant-supported prostheses: a systematic review. *The Journal of Prosthetic Dentistry* S0022-3913 (21): 00506-0. [https://doi.org/10.1016/](https://doi.org/10.1016/j.prosdent.2021.08.027) [j.prosdent.2021.08.027](https://doi.org/10.1016/j.prosdent.2021.08.027).
- **81** Lin, J., Lin, Z., and Zheng, Z. (2020). Effect of different restorative crown design and materials on stress distribution in endodontically treated molars: a finite element analysis study. *BMC Oral Health* 20 (1): 226. <https://doi.org/10.1186/s12903-020-01214-3>. PMID: 32811484; PMCID: PMC7437004.
- **82** Sevimay, M., Usumez, A., and Eskitascioglu, G. (2005). The influence of various occlusal materials on stresses transferred to implant-supported prostheses and supporting bone: a three-dimensional finite-element study. *Journal of Biomedical Materials Research. Part B, Applied Biomaterials* 73 (1): 140–147. [https://doi.org/](https://doi.org/10.1002/jbm.b.30191) [10.1002/jbm.b.30191](https://doi.org/10.1002/jbm.b.30191). PMID: 15742379.
- **83** Seemann, R., Marincola, M., Seay, D. et al. (2015). Preliminary results of fixed, fiber-reinforced resin bridges on four 4- × 5-mm ultrashort implants in compromised bony sites: a pilot study. *Journal of Oral and Maxillofacial Surgery* 73 (4): 630–640. [https://doi.org/10.1016/](https://doi.org/10.1016/j.joms.2014.10.031) [j.joms.2014.10.031](https://doi.org/10.1016/j.joms.2014.10.031). Epub 2014 Nov 14. PMID: 25542605.
- **84** Johnson, K. (1969). A study of the dimensional changes occurring in the maxilla following tooth extraction. *Australian Dental Journal* 14 (4): 241–244. [https://doi.](https://doi.org/10.1111/j.1834-7819.1969.tb06001.x) [org/10.1111/j.1834-7819.1969.tb06001.x](https://doi.org/10.1111/j.1834-7819.1969.tb06001.x). PMID: 5259350.
- **85** Atwood, D.A. (1963). Postextraction changes in the adult mandible as illustrated by microradiographs of midsaggital sections and serial cephalometric roentgennograms. *The Journal of Prosthetic Dentistry* 13: 810–815.
- **86** Tallgren, A. (1972). The continuing reduction of the residual alveolar ridges in complete denture wearers: a mixed-longitudinal study covering 25 years. *The Journal of Prosthetic Dentistry* 27 (2): 120–132. [https://doi.org/](https://doi.org/10.1016/0022-3913(72)90188-6) [10.1016/0022-3913\(72\)90188-6](https://doi.org/10.1016/0022-3913(72)90188-6). PMID: 4500507.
- **87** Denissen, H.W. and Kalk, W. (1991). Preventive implantations. *International Dental Journal* 41 (1): 17–24. PMID: 1848531.
- **88** Denissen, H.W., Kalk, W., Veldhuis, H.A., and van Waas, M.A. (1993). Anatomic consideration for preventive implantation. *The International Journal of Oral & Maxillofacial Implants* 8 (2): 191–196. PMID: 8359876.
- **89** Misch, C.E. (1999). *Contemporary Implant Dentistry*. St. Louis: Mosby.
- **90** Esposito, M., Grusovin, M.G., Felice, P. et al. (2009). The efficacy of horizontal and vertical bone augmentation procedures for dental implants – a Cochrane systematic review. *European Journal of Oral Implantology* 2 (3): 167–184. PMID: 20467628.
- **91** Sheikh, Z., Sima, C., and Glogauer, M. (2015). Bone replacement materials and techniques used for achieving vertical alveolar bone augmentation. *Materials* 8 (6): 2953–2993.<https://doi.org/10.3390/ma8062953>.
- **92** Hjalmarsson, Smedberg, J.-I., Pettersson, M., and Jemt, T. (2011). Implant-level prostheses in the edentulous maxilla: a comparison with conventional abutment-level prostheses after 5 years of use. *The International Journal of Prosthodontics* 24 (2): 158–167.
- **93** Renvert, S. and Quirynen, M. (2015). Risk indicators for peri-implantitis. A narrative review. *Clinical Oral Implants Research* 26 (Suppl. 11): 15–44.

Prosthetic Rehabilitations to Optimize Maintenance and Implant Long-term Success

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Introduction

Decisions between fixed and removable implant–supported restorations are usually governed by clinical considerations (anatomy and esthetics). Financial constraints and patient requests may also enter the equation, and there is a growing tendency for sociodemographic aspects to become relevant in selecting treatment modalities [1, 2]. Due to a rapidly changing population, dental clinicians in the United States will be treating an ever-increasing proportion of older patients, whose percentage in the age range of 65 and over is expected to increase from currently 16.5% to almost 20% by the late 2020s and to 25% by 2060 [1, 3]. Dental clinicians will have to develop treatment concepts in line with the requirements and limitations of this continuously growing population. This particularly applies to implant dentistry, where the goal is to achieve high success rates combined with low complication rates. Advanced fabrication methods – such as computer-aided design and manufacturing (CAD/CAM) technology – have increased the precision and time efficiency of achieving treatment goals. At the same time, there is a mandatory need to ensure that the resultant prostheses can be readily handled by patients whose motor abilities will foreseeably decrease with age. There is also a need, notably, with a view to the longevity of dental implants, that good cleansability of the superstructures by routine self-performed hygiene is ensured. With a view to these manipulation and maintenance requirements, implant–supported removable prostheses are considered beneficial, but attention should also be devoted to their attachment systems [4, 5].

Attachment Systems for Removable Superstructures

Different attachment systems are the main distinguishing feature between implant–supported removable superstructures. Two major subtypes exist: freestanding (unsplinted) and splinted systems. All of these have in common that they comprise a patrix (male) and a matrix (female) component, the latter forming an integral part of the superstructure and acting as a receptacle, the former being connected to the implant and engaging them [6]. Freestanding systems notably include ball, locators, magnetic, and telescopic attachments. Splinted systems may take the form of either flexible (cast) or rigid (milled) bar attachments (Figure 19.1).

Long-term Evaluation

For all these attachment systems, adequate outcomes are documented with regard to function, esthetics, and "oral health-related quality of life" (OHRQoL) [7, 8]. They have been shown to offer high rates of five-year overall survival ranging from 95 to 100% [5, 9]. While a complete assessment of long-term prognoses will also, of course, have to consider the risk of complications, a uniform approach to classifying complications of implant–supported removable prostheses does not currently exist. Back in 2001, Payne et al. [10] proposed that a distinction should be made between routine maintenance performed during periodic recall visits on the one hand, and repair or corrective maintenance requirements on the other. Reports in the literature actually do tend to differentiate between technical

Unsplinted (freestanding) attachments Magnetic **Double crowns** Ball Ball **Locator**

Figure 19.1 Unsplinted Attachment systems for implant–supported removable superstructures.

complications (often used interchangeably with mechanical complications) and routine maintenance requirement.

Complications and Maintenance

Technical complications in the sense that patrix or matrix components need to be activated, replaced, or repositioned and are the most common type of intervention with implant–supported removable prostheses [11–14]. While ball attachments seem particularly susceptible to requiring such interventions notably within the first year of delivery, these inventions do not translate into a significant difference in overall comparisons between freestanding ball or Locator attachment systems [14, 15]. Within splinted systems, a difference has been reported in favor of rigid (milled) bars, where significantly fewer interventions were required for the retentive part [14].

Inconclusive rates are documented for screw (including abutment screw) loosening or fracture. On one hand, all attachment systems appear to require interventions for these events similarly often [13]. On the other, the incidence of this complication with maxillary prostheses has been reported to be especially high for bar attachments [12].

The risk of prosthetic fractures – including failure of a resin tooth or the denture framework – is influenced by how the restoration has been fabricated in the dental laboratory. Current evidence in the literature indicates a higher rate of technical complications associated with the denture itself for maxillary restorations that do not feature a metal reinforcement or coverage of the palate [12, 16]. Another modifier of intervention rates is for how long a denture has been worn. Systematic reviews have unanimously disclosed that complications increase after a five-year period [12, 13].

How to Select an Attachment System?

A variety of parameters come into play in deciding which attachment system should be selected in each patient. What follows is a short discussion of three diagnostic levels from which the decision should eventually be derived.

Decision Level 1: People-related Factors

At this level, the clinician and the patient are the main two factors contributing to the decision. Other modifiers include the clinical and technical expertise of the entire team, the patient's desires and expectations, and how much he or she can afford to pay for the treatment. Selectable attachment systems may also be limited by health-related issues, most notably regarding the patient's motor abilities.

Decision Level 2: Tissue-related Factors

This level, too, combines various aspects to be considered in the decision-making. Most importantly, these include the existing prosthetic space, number of implants, interimplant distance, implant angulation, and implant position. Of particular note in this context, removable prostheses are generally considered to involve greater horizontal and vertical space requirements than fixed prostheses. It is mandatory to make sure in the diagnostic phase that adequate space will be present for both the attachment and its housing, as well as for the denture itself. In the literature, vertical dimension requirements of 7 or 11mm have been reported for freestanding or splinted (i.e. bar) attachments, respectively [17]. These values should be regarded as the minimum height capable of avoiding undersized components (Figure 19.2).

Figure 19.2 (a) and (b): Intraoral view of a case where inadequate space resulted in undersized prosthetic components, thereby causing fracture of the denture and loss of the matrix component.

Figure 19.3 (a) and (b): Intraoral views of differently designed full-mouth restorations with double crown attachments. Those on the left are designed as removable dentures, while those on the right are similar to fixed prostheses.

Decision Level 3: Simulation-related Factors

Factors related to the quality of simulation need to enter the decision process as well. They are not only concerned about the question if palatal coverage is required, but also about the dimensions of the future prosthesis. Whenever coverage of the palate is not tolerated by a patient, inserting an adequate number of implants will eliminate this need. Four or more implants are required to this end [18]. The use of double crowns for attachments is considered beneficial by allowing for substantially reduced dimensions of the prosthesis. Experience has shown that implant–supported removable prostheses of this type, similar in design to fixed bridgework, meet with high patient acceptance (Figure 19.3).

This author believes that an ideal attachment system, which will reliably meet a given patient's individual requirements, can only be selected by taking a synoptic view (Figure 19.4).

Prostheses Retained by Double-crowns

Of the aforementioned retention systems for implant– supported removable prostheses, the variant of doublecrowns will be discussed in depth for the remainder of this chapter. Compared to other freestanding systems, this attachment mechanism has several distinguishing features and is extremely versatile.

Figure 19.4 Overview of modifiers to be considered in selecting an ideal attachment system.

History of Telescopic Crowns

Telescopic crowns were invented for fixed dental prostheses. Their use was first described by a Philadelphian dentist named R. W. Starr in 1886 [19]. Initially used for bridgework segmentation, they were later harnessed for removable prostheses.

The conventional use for double-crown telescopes is to retain dentures on residual teeth. A telescopic double crown is made up of a primary (internal) crown fixed to a natural tooth or implant abutment, and a secondary (external) crown, which, being a rigid component of the denture framework, forms an integral part of the prosthesis.

Friction-type (Cylindrical Crown) Telescopes

These telescopes are based on a primary crown milled to 0−2° of parallelism, which slides into the secondary crown much like a plunger into a cylindrical receptacle, having contact with its wall circumferentially. Retention is thus achieved by friction, i.e. by the frictional resistance developing between the two contacting surfaces. The original design of these friction-type telescopes, and their further development into parallel attachments and bars, dates back to Karl Häupl und Hermann Böttger [20].

Resilience-type (Clearance Crown) Telescopes

This non-friction variant of telescopic double crowns was introduced by Manfred Hofmann in 1966. Here retention is achieved mainly by mucosal support. Given around 0.5mm of occlusal clearance between the primary and secondary crowns, retention develops not immediately but after mucosal resilience is established [20]. In European countries, these prostheses are known as "cover dentures." Good results with this system have been obtained mainly in the presence of up to three natural tooth abutments. Resilience-type telescopes are not used in implant dentistry.

Cone-type (Conical Crown) Telescopes

In 1968, Karl-Heinz Körber worked on refining the frictiontype telescopes by developing a primary crown no longer cylindrical in shape, but featuring a taper of 4°–8°, which reduced frictional resistance to telescope closure [20]. Depending on the convergence angle of the primary crown, either a retentive or a supportive function is accomplished. Smaller angles translate into more retention, the strength of which is also influenced by different surface characteristics and materials. A high-gold alloy has proven to be the material of choice for the components of this system (Figure 19.5).

Figure 19.5 Different primary crowns used in double crown attachment systems.

Figure 19.6 (a) and (b): Principle of the "tribological system" according to Weigl et al. [21].

Gold-on-zirconia Double Crowns

This system also uses a conical primary crown with a defined cone angle of 2° but with a high-strength oxide ceramic as the primary crown material and a secondary crown made of electroformed gold. This principle was developed and introduced by Paul Weigl at the turn of the millennium, has entered the daily clinical practice with the introduction of advanced CAD/CAM technology, and is today widely accepted. This system, also known as the "Frankfurt principle," does not rely on friction between the patrix and matrix components for retention (which therefore remains unaffected by intraoral service). Rather, the retention is accomplished tribologically, with the surfaces interacting in relative motion via a gap of 5μm that is created between the primary and secondary crowns. In contact with saliva, this feature serves as a capillary gap allowing cohesive forces to develop that will ensure continuous retention of the prosthesis. The design and material selected for the primary crowns are also beneficial, as zirconia minimizes the "demasking" effect and given a predefined vertical height of at least 7mm, acts as a rest seat allowing for a delicate removable prosthesis similar to what would normally be the design of a fixed prosthesis. In addition, primary crowns of zirconia offer more stable retention than crowns made of gold alloy (Figure 19.6) [5, 21–23].

To summarize, a variety of double-crown telescopic systems are available, all of them having their own strengths and weaknesses. The characteristics of the most widely used systems are listed in Table 19.1.

The "Frankfurt Principle"

Indications and Limitations

A challenge routinely posed by severely reduced dentitions is for the clinical team and the patient to decide whether the proposed rehabilitation should incorporate any compromised residual teeth or should be supported and retained exclusively by implants. While there is never going to be a universally applicable solution to this dilemma, the concept of telescopic retention by double crowns, notably in accordance with the "Frankfurt principle," does offer several advantages in prostheses combining both existing teeth and strategically inserted implants.

Combinations of tooth and implant abutments are known to offer mutual stabilization. While the tactile sensitivity of natural abutments, given intact periodontal receptors, will counteract any excessive masticatory forces, the much higher stability of the implants will protect the

Source: From Lehmann and Gente [24].

*Cover Denture=resilience-type telescopes

compromised abutment teeth from excessive loading [25]. The goal is to achieve total integration of abutments, i.e. of all residual natural teeth with all implants placed. As an additional consideration, the prosthetic usefulness of each potential abutment tooth should be evaluated in advance based on periodontal, endodontic, and reconstructive criteria [26, 27]. Its suitability for a hybrid prosthesis of this type can be derived from the result of this evaluation outlined in Table 19.2.

Another important advantage of these restorations is that they can potentially survive any loss of one or several tooth abutments. In a scenario like this, the prosthesis will usually continue to serve its purpose. Later, an additional implant can be inserted to re-establish stability at the site of the lost abutment tooth.

A distinctive feature of the Frankfurt treatment protocol is to fabricate and provide these patients with a "travel denture" (described in detail later in this chapter). As a result, the patient can resort to an acceptable copy of the definitive denture whenever the need arises, so that any maintenance and/or repair jobs can be conducted in the laboratory as required without creating an unpleasant waiting period for the patient.

Compared to fixed restorations, *it is easier to maintain high levels of plaque control with a prosthesis that can be removed by the patient at all times*. Compared to dentures retained by splinted bars, it is also much easier for the patient to maintain self-performed hygiene of the freestanding implant–supported crowns serving as primary telescopes. One potential limitation to keep in mind, however, is the long-term prognosis of the patient's general health, including his or her motor abilities (Figure 19.7 and 19.8).

Correct implant positioning is essential to the longterm success of the prosthesis and, therefore, should be driven by the design of the future prosthesis, to avoid any malpositioning that might cause deviations from the uniform orientation that is needed for all abutments to properly insert into the secondary crowns. These requirements may necessitate a more extensive surgical approach, which, in turn, might exceed the patient's willingness to accept risks or be contraindicated in the presence of health problems. While few compromises are acceptable with regard to the position, length, and angulation of implants for fixed prostheses, there is nothing wrong with selecting between several options of implant positioning for removable prostheses to strike an appropriate

Table 19.2 Suitability of natural teeth for use as abutments, modified and expanded.

Source: From Zitzmann [26] and Naumann [27].

balance between the extensiveness, cost, and benefit of treatment. For instance, major surgical procedures like bone augmentation can often be avoided or their invasiveness reduced without repercussions on the function or esthetics attainable by prosthetic treatment. An effort should be made, however, to achieve an abutment distribution as quadrangular as possible for optimal support of the prosthesis. Pertinent follow-up examinations have demonstrated a statistically significant difference between the success rates offered by quadrangular versus both triangular and linear transverse distributions, in the absence of

a difference between these latter two support distributions (Figure 19.9) [5].

Also, removable restorations supported by implants allow for better flexibility in optimizing the prosthetic design for aesthetics and function. The denture-mucosa interface can be positioned more freely, and the tooth axes more readily corrected. Occlusal relationships can be transformed or adjusted in the vertical and horizontal planes with modest effort. Limitations of these prostheses, notably when natural tooth abutments are to be incorporated, arise from the total space requirements for a primary crown, secondary crown, tertiary structure, and veneer layer. Also, preparing natural teeth with abrasive instruments for use as abutments is somewhat invasive. Esthetic limitations may arise from a need for over-contoured

Figure 19.7 Reinforcement of oral-hygiene training for cleansing of freestanding primary crowns required by dental providers. These ceramic primary telescopes had been in situ for eight years.

prosthetic restorations notably in the anterior segment of the maxilla or mandible. Giving due consideration to all these limitations in the planning stage is absolutely required.

The treatment and fabrication process are more complex for hybrid prostheses than for other restorations. A team approach to planning is required, followed by close coordination of the successive clinical and laboratory steps. Meticulous care should be devoted to "passivation," which is required to compensate for the physiological mobility of natural tooth abutments as compared to the relative rigidity of osseointegrated implants. The process of laboratory fabrication, too, is technique-sensitive and should be performed by a considerably experienced and skilled technician.

Clinical and Technical Process

The rest of this chapter will be dedicated to case reports illustrating the chairside and laboratory workflows for prostheses retained by gold-on-zirconia double crowns.

Figure 19.8 Abutment distributions as classified by Steffel [28]. (a) punctiform support. (b) linear sagittal (unilateral anteroposterior) support. (c) linear transverse support. (d) linear diagonal support. (e) triangular support. (f) quadrangular support.

Specific Workflow Considerations

Primary and Secondary Crowns

Using CAD/CAM technology, the primary crowns are milled from zirconia, an oxide-ceramic material known for its high strength. Based on an esthetic try-in (mock-up) of a digitized denture setup, a backward-planned design process is carried out first for ideal abutment shapes and then for the primary crowns. Once milled, these primary crowns are then processed using diamond finishing instruments without polishing.

Digitized setup (mock-up) $\qquad \qquad$ Setup-based designing of abutments

Finished abutment designs **Finished primary-crown** designs

Figure 19.9

Milled abutments Primary crowns milled from zirconia

Workflow: figures 1–6.

A milling device is used to create the contact surfaces of the conical crowns. Care should be taken to ensure smooth surfaces, as any tiny "dent" will be replicated in the process of electroforming the matrix component, which then, in the process of being dislodged from the conical crown,

is bound to expand by the amount of this dent, thus compromising the high precision of fit. Any polishing will indent these smooth surfaces in ways not always visible to the naked eye. Therefore, following processing in the milling unit, the conical crowns should never be polished.

Processing of the primary crowns Surface smoothening (no polishing!) Workflow: figures 7 and 8

The secondary crowns, or matrix components, are created directly on the surface of the conical primary crowns using a fully automated process of electroforming by deposition of fine-gold particles. Thus, rather than duplicating the primary crown for indirect forming of the matrix component, the internal surface of the fine-gold coping is directly defined by the external surface of the conical crown. To render the surface conductive for electroforming, the conical crown is covered with a very thin layer of silver varnish by air-spraying, which also creates a separating layer allowing the electroformed coping, once finished, to be dislodged from the conical crown. The layer thickness (and especially evenness) of conductive silver varnish is critical for an accurate fit of the electroformed matrix component. Given a correct procedure, a 5 μm fit is created between the primary and secondary crown.

Workflow: figures 9−12: Fabrication of the secondary crown by deposition of fine-gold particles.

Case Report 1

This female patient presented with existing maxillary and mandibular restorations. Being dissatisfied with her chewing function and esthetic appearance, she requested

treatment to replace the old prostheses and resolve her masticatory muscle pain.

Case report 1: figures 1−9: Baseline situation.

Right lateral view Left lateral view

Orthopantomogram (first visit)

The diagnostic examinations revealed, in addition to inadequate prostheses in the maxilla and mandible, findings of complete anterior disc displacement in both temporomandibular joints.

In the pretreatment stage, before restorations were completely removed, an occlusal splint was fabricated to

correct the inter arch relationship. Once the patient no longer reported feeling any pain, the existing blade implants were removed and all teeth except for 13 (#6) and 33 (#22) were extracted. The next step was implant placement in both jaws.

Case report 1: figures 10−13: Pretreatment.

Case report 1: figures 14−19: First stage of denture fabrication.

Tooth preparation and impression-taking (maxilla)

Tooth preparation and impression-taking (mandible)

Provisional intermaxillary analysis (laboratory view)

Provisional intermaxillary analysis (clinical view)

Esthetic try-in (mock-up) of the denture setup.

Case report 1: figures 20−28: Second stage of denture fabrication.

Definitive abutments (occlusal view) Primary crowns (ceramic crowns) cemented to the abutments; covered by secondary crowns

Try-in of tertiary framework (maxilla) Try-in of tertiary framework (mandible)

Situation after intraoral bonding **Situation after final intermaxillary analysis** Situation after final intermaxillary analysis

Primary crowns (ceramic crowns) after intraoral bonding

Overimpression of tertiary framework Delivery of interim dentures

Case report 1: figures 29−32: Treatment stage of intraoral delivery.

Intraoral view of prostheses in situ **Intraoral view of esthetic zone**

Occlusal view of maxillary prosthesis Occlusal view of mandibular prosthesis

Fabrication of the Tertiary Structure

The tertiary structure, designed to become an integral part of the finished prosthesis, is bonded to the secondary crowns in the patient's mouth. It was previously fabricated with the framework loosely and passively seated on the secondary crowns. Due to the gap involved, the precision

requirements are relatively low for such an extensive framework. What is required is a massive build (ideal: 9 m^2) notably where the cantilever-style distal denture bases start, as these removable prostheses with primary crowns telescoping into electroformed gold are supported purely by teeth and/or implants. Forces are distributed in exactly the same way as in fixed dental prostheses!

Workflow: figures 13 and 14: Tertiary framework designed by CAD and milled by CAM.

Intraoral Bonding

For the high-precision 5-μm fit between the primary and secondary crowns, the framework needs to be assembled with the secondary crowns right in the patient's mouth. This assembly cannot possibly take place on a cast (as would be possible for traditional conical crowns). What follows is a brief summary of the required workflow:

- 1) Definitive intraoral tightening of implant abutments and/or trying in of primary crowns on natural abutment teeth.
- 2) First try-in of the tertiary framework with the primary and secondary crowns placed underneath.

Workflow, figure 15

3) Definitive cementation of the primary crowns with the secondary crowns seated on top.

Workflow: figure 16: Definitive tightening of the implant abutments, followed by cementation of the primary crowns.

4) Second try-in of the tertiary framework. The fit should be completely passive.

Workflow: figure 17

5) Intraoral bonding of the tertiary framework to the secondary crowns (using an adhesive cement).

Workflow: figures 18 and 19: Cemented primary crowns with secondary crowns seated on top.

Workflow: figures 20 and 21: Loading adhesive cement into the tertiary framework.

Workflow: figure 22: Intraoral bonding of the tertiary framework to the secondary crowns.

Workflow: figures 23 and 24: Occlusal and frontal views of the ceramic primary crowns after the tertiary framework now integrated with the secondary crowns has been removed.

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Workflow, figures 25–27: Over-impression of the tertiary framework in polyether with a custom tray.

Case Report 2

This female patient presented with a request to have her existing mandibular denture replaced. She reported having a hard time with multiple pressure sores. She also

expressed a desire for better esthetics particularly in the esthetic zone.

Case report 2: figures 1−4: Baseline situation.

After in-depth counselling, four interforaminal implants were inserted.

Case report 2: figures 5 and 6: Impression of the patient's edentulous maxilla (left) and impression of abutments attached to the implants (right).

Case report 2: figure 7: Following a provisional intermaxillary analysis, an esthetic mock-up was tried in with

the main goal of verifying the anterior maxilla (esthetic zone).

Case report 2: figures 8 and 9: Mandible: prosthetic components were fabricated in accordance with the "Frankfurt" protocol (implant abutments, primary and

secondary telescopic crowns, a tertiary framework, and a mandibular travel denture). Maxilla: finishing of the mucosa-supported denture.

Case report 2: figures 10 and 11: Tightening definitive abutments to the mandibular implants.

Case report 2: figures 12 and 13: Definitive cementation of the primary crowns (with their matrix

counterparts of electroformed gold seated on top) to the abutments.

Case report 2: figures 14–18: Intraoral bonding of the tertiary framework to the matrix components, intermaxillary analysis against the definitive maxillary denture, and insertion of the travel denture.

Case report 2: figure 19 and 20: Delivery of the final maxillary and mandibular prostheses.

Case report 2: figures 21 and 22: Finished maxillary and mandibular setups from the laboratory.

Case report 2: figure 23 and 24: Five years after insertion of the prostheses, minor coffee-related

discolorations were seen, which easily came off by surface polishing in the laboratory.

Any restorative steps were preceded by a comprehensive pretreatment regimen for systematic control of periodontitis. Subsequently, following removal of all restorations, implants were strategically inserted in the maxilla (sites $#3, #5, #12, #14$) and mandible (sites $#20$ and #29). Then a mock-up was tried in to define the esthetic appearance of the future prostheses. As the patient expressed a desire for designs as delicate as possible at this point, prosthetic dimensions similar to fixed bridgework were planned.

Case report 3: figures 9 and 10: Situation after pretreatment and intraoral esthetic mock-up.

Case report 3: figures 11 and 12: Definitive tightening of the implant abutments and preparation of the residual natural teeth for use as abutments.

Case report 3: figures 13 and 14: Cementation of primary crowns and try-in of tertiary framework.

Case report 3: figures 15 and 16: After the try-in had revealed direct contact of the seated tertiary framework, the intaglio surface was relieved.

Case report 3: figures 17 and 18: Situation after intraoral bonding, followed by intermaxillary analysis.

Case report 3: figures 19 and 20: An overimpression of the tertiary framework was taken, transferred to the laboratory, and segmented for divestment. Case report 3:

figures 21 and 22: The frameworks were mounted in an articulator and veneered with composite resin.

Case report 3: figures 23 and 24: Finished prostheses for the maxilla and mandible.

Case report 3: figure 25 and 25a: Intraoral delivery of the maxillary and mandibular prostheses.

Case report 3: figures 26 and 27: Occlusal views of the inserted maxillary and mandibular prostheses.

Case report 3: figure 28: Follow-up examination 1 year after intraoral delivery.

Case report 3: figure 29: Follow-up examination 8 years after intraoral delivery.

Travel Denture

The initial requirement for a temporary restoration arises from the modified treatment process, particularly because the primary crowns are definitively cemented before the denture is finished. Therefore, concurrently with the primary crowns, an interim removable denture is fabricated, which is referred to as a "travel denture" since it can be worn even after the definitive prosthesis has been delivered. This option comes in handy whenever the definitive prosthesis may need to be repaired, and the patient will always have two restorations (the standard one and a backup) to choose from.

Once the aforementioned processing of the definitive tertiary framework (intraoral bonding and over impression) has been completed, this future "travel" denture, whose basal intaglio surfaces have been generously relieved, is relined with a soft material and inserted, at this point in time, as an interim denture in the patient's mouth.

Workflow: figures 28–31: Maxillary travel denture before, during, and after soft relining (basal views), as well as after intraoral insertion (perioral view)

The case reports that have been presented in this chapter illustrate a number of different applications for the "Frankfurt" attachment system of gold-on-zirconia double crowns. It should be emphasized that this system lends itself well to efforts in the laboratory aimed at meeting patient expectations. Good results can be obtained even in complex cases associated with functional problems.

Long-term performance of "Frankfurt" gold-on-zirconia double crowns.

A large-scale observation study has been performed to evaluate the long-term clinical outcomes of prostheses retained by gold-on-zirconia telescopes.

A total 126 prostheses with 603 abutments in 111 patients, with observation periods of up to 11.5 years, resulted in Kaplan–Meier survival rates of $92.9 \pm 0.05\%$ (maxilla) or 100% (mandible). Based on all prostheses, the authors reported Kaplan-Meier survival rates (time to failure) of $82.5 \pm 8.6\%$ (95% CI: 65.7–99.6) at 5 years and $82.5\% \pm 4.8\%$ (95% CI: 65.7–99.6) at 10 years. These findings

Figure 19.10 Abutment based maintenance [5].

are consistent with current evidence in the literature and indicate favorable outcomes of functional survival for these prostheses [5, 29]. Prostheses with any abutment configurations (natural teeth and/or implants) were included, the largest subgroup being prostheses supported exclusively by implants $(n = 77)$. Which abutment configurations were used was not found to influence survival or 'time to first repair' in a significant way.

Another essential question, in addition to functional survival, concerns the incidence of complications and maintenance requirements. In this regard, the study focused on whether the laboratory requirements for these prostheses, which can fairly be called extensive, are justified by an appropriately low incidence of technical complications. Moreover, patient-centered measures were to be used in the overall assessment.

Repair requirements were found to equal 1.9 events for each maxillary prosthesis and 1.89 events for each mandibular prosthesis. Maintenance requirements were found to be associated with corresponding event rates of 0.72 or 1.3 for each maxillary or mandibular prosthesis, respectively. Low maintenance requirements were identified at the abutment level (Figure 19.10).

These findings are consistent with the complication rates emerging from a systematic review and meta-analysis [30]. To summarize, removable prosthesis featuring the "Frankfurt" attachment system of gold-on-zirconia double crowns are a low-complication treatment modality with manageable maintenance requirements.

The patients' own ratings were found to indicate high satisfaction and that the handling of these prostheses daily was not a significant problem (Table 19.3).

Maxilla (MX)	Mandible (MD)	MX+MD
$n=22$	$n = 39$	$n=13$
87.77 ± 13.59	89.79 ± 9.81	87.88 ± 14.89
92.9 ± 7.47	90.85 ± 8.62	90.12 ± 14.4
90.36 ± 16.88	89.79 ± 12.68	87.96 ± 19.33
94.77 ± 7.11	90.95 ± 9.48	86.42 ± 17.79
$94 + 6.32$	88.36 ± 13.09	86.15 ± 16.42
88.5 ± 15.72	90.23 ± 8.93	$82.23 + 22.53$
90.54 ± 14.02	87.44 ± 13.22	79.42 ± 26.36
92.54 ± 12.11	91.026 ± 7.34	85.08 ± 22.14
90.23 ± 12.72	91.21 ± 9.76	84.38 ± 20.66

Table 19.3 Patient satisfaction scoring of the prosthetic rehabilitation.

Even though the current literature gives no clear evidence to justify recommending one specific attachment system for implant–supported removable prostheses, the "Frankfurt" double-crown system presented in this chapter, consisting of gold-on-zirconia double crowns, covers a broad spectrum of indications and offers a high degree of safety with regard to complications and maintenance requirements. Moreover, it appears that this treatment modality also meets with high patient acceptance and ensures the long-term success of dental implants.

References

- **1** Inglehart, M.R., Albino, J., Feine, J.S., and Okunseri, C. (2022). Sociodemographic changes and oral health inequities: dental workforce considerations. *JDR Clinical & Translational Research* 7 (1_suppl): 5S–15S.
- **2** Sailer, I., Karasan, D., Todorovic, A. et al. (2022). Prosthetic failures in dental implant therapy. *Periodontology 2000* 88 (1): 130–144.
- **3** Mather, M., Jacobsen, L.A., and Pollard, K.M. (2015). *Aging in the United States*. Population Bulletin, vol. 70,. no. 2., 1–20. Washington (DC): Population Reference Bureau.
- **4** Schubert, O., Güth, J.F., Beuer, F. et al. (2020). Double crown rescue concept: clinical and dental technical workflow. *International Journal of Computerized Dentistry* 23 (3): 281–292. PMID: 32789315.
- **5** Brandt, S., Winter, A., Weigl, P. et al. (2019). Conical zirconia telescoping into electroformed gold: a retrospective study of prostheses supported by teeth and/or implants. *Clinical Implant Dentistry and Related Research* 21 (2): 317–323.
- **6** Anas El-Wegoud, M., Fayyad, A., Kaddah, A., and Nabhan, A. (2018). Bar versus ball attachments for implant-supported overdentures in complete edentulism: a systematic review. *Clinical Implant Dentistry and Related Research* 20 (2): 243–250.
- **7** Feine, J.S., Maskawi, K., de Grandmont, P. et al. (1994). Within-subject comparisons of implantsupported mandibular prostheses: evaluation of masticatory function. *Journal of Dental Research* 73 (10): 1646–1656.
- **8** Reissmann, D.R., Dard, M., Lamprecht, R. et al. (2017). Oral health-related quality of life in subjects with implantsupported prostheses: a systematic review. *Journal of Dentistry* 65: 22–40. [https://doi.org/10.1016/j.](https://doi.org/10.1016/j.jdent.2017.08.003) [jdent.2017.08.003](https://doi.org/10.1016/j.jdent.2017.08.003). Epub 2017 Aug 5. PMID: 28789860.
- **9** Slot, W., Raghoebar, G.M., Vissink, A. et al. (2010). A systematic review of implant-supported maxillary overdentures after a mean observation period of at least 1 year. *Journal of Clinical Periodontology* 37 (1): 98–110.
- **10** Payne, A.G., Walton, T.R., Walton, J.N., and Solomons, Y.F. (2001). The outcome of implant overdentures from a prosthodontic perspective: proposal for a classification protocol. *The International Journal of Prosthodontics* 14 (1): 27–32.
- **11** Assaf, A., Daas, M., Boittin, A. et al. (2017). Prosthetic maintenance of different mandibular implant overdentures: a systematic review. *The Journal of Prosthetic Dentistry* 118 (2): 144–152.e145.
- **12** Osman, R.B., Payne, A.G., and Ma, S. (2012). Prosthodontic maintenance of maxillary implant overdentures: a systematic literature review. *The International Journal of Prosthodontics* 25 (4): 381–391.
- **13** Cehreli, M.C., Karasoy, D., Kokat, A.M. et al. (2010). Systematic review of prosthetic maintenance requirements for implant-supported overdentures. *The International Journal of Oral & Maxillofacial Implants* 25 (1): 163–180.
- **14** Goodacre, C. and Goodacre, B. (2017). Fixed vs removable complete arch implant prostheses: a literature review of prosthodontic outcomes. *European Journal of Oral Implantology* 10 (Suppl 1): 13–34.
- **15** Brandt, S., Lauer, H.-C., Fehrenz, M. et al. (2021). Ball versus Locator® attachments: a retrospective study on prosthetic maintenance and effecton oral-health-related quality of life. *Materials* 14 (4): 1051.
- **16** Andreiotelli, M., Att, W., and Strub, J.R. (2010). Prosthodontic complications with implant overdentures: a systematic literature review. *The International Journal of Prosthodontics* 23 (3): 195–203.
- **17** Carpentieri, J., Greenstein, G., and Cavallaro, J. (2019). Hierarchy of restorative space required for different types of dental implant prostheses. *Journal of the American Dental Association (1939)* 150 (8): 695–706.
- **18** S3-Leitlinie (Langfassung) Implantatprothetische Versorgung des zahnlosen Oberkiefers AWMF-Registernummer: 083-010.
- **19** Starr, R.W. (1886). Removable bridge-work, porcelain cap crowns. *Dental Cosmos* 28: 17.
- **20** Szentpetry, S. (2016). *Das Stark Reduzierte Testzahngebiss-Versorgung Mit Teleskopprothetik*. Quintessenz Verlag.
- **21** Weigl, P., Hahn, L., and Lauer, H.C. (2000). Advanced biomaterials used for a new telescopic retainer for removable dentures. *Journal of Biomedical Materials Research* 53 (4): 320–336.
- **22** Weigl, P. and Lauer, H.C. (2000). Advanced biomaterials used for a new telescopic retainer for removable dentures. *Journal of Biomedical Materials Research* 53 (4): 337–347.
- **23** Bayer, S., Zuziak, W., Kraus, D. et al. (2011). Conical crowns with electroplated gold copings: retention force changes caused by wear and combined off-axial load. *Clinical Oral Implants Research* 22: 323–329.
- **24** Lehmann, K.M. and Gente, M. (1988). *Doppelkronen als Verankerung für herausnehmbaren Zahnersatz* (ed. W. Ketterl), 106–120. München: Deutscher Zahnärztekalender.
- **25** Mericske-Stern, R. (1994). Oral tactile sensibility recorded in overdenture wearers with implants or natural roots: a comparative study. Part 2. *The International Journal of Oral & Maxillofacial Implants* 9 (1): 63–70.
- **26** Zitzmann, N.U., Krastl, G., Hecker, H. et al. (2010). Strategic considerations in treatment planning:

deciding when to treat, extract, or replace a questionable tooth. *The Journal of Prosthetic Dentistry* 104 (2): 80–91.

- **27** Naumann, M., Heydecke, G., and Joda, T. (2010). Wissenschaftliche Mitteilung der DGZPW Prothetische Rehabilitation im parodontal geschädigten (aber sanierten). *Gebiss* 1–11.
- **28** Steffel, V.L. (1962). Planning removable partial dentures. *The Journal of Prosthetic Dentistry* 12: 524–535.
- **29** Schwarz, S., Bernhart, G., Hassel, A.J., and Rammelsberg, P. (2014). Survival of double-crownretained dentures either tooth-implant or solely implant-supported: an 8-year retrospective study. *Clinical Implant Dentistry and Related Research* 16 (4): 618–625.
- **30** Lian, M., Zhao, K., Feng, Y., and Yao, Q. (2018). Prognosis of combining remaining teeth and implants in double-crown-retained removable dental prostheses: a systematic review and meta-analysis. *The International Journal of Oral & Maxillofacial Implants* 33 (2): 281–297.

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Implant Prostheses Planning and Maintenance for the Aging Population

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Introduction

"Older adult" or "elderly" are terms conventionally referred to individuals older than 65 years of age [1]. The World Health Organization estimates an increase in the elderly population from 900 million to 2 billion by 2050 worldwide; an increase from 12 to 22% of the total global population (WHO) [2]. Not only is this age group increasing in number, but also exhibits a notable increase in life expectancy, educational level, and personal wealth, factors that play a role in overall health. The impact of social determinants of health such as these, in oral and overall health should be considered when treatment planning a patient affected by any combination of the factors. On the other hand, geriatric dental patients are biologically compromised adults, that may or may not be older than 65, with one or more chronic, debilitating, physical, or mental illness [3]. Older adults as well as geriatric patients can present with unique challenges that impact treatment planning for implant prosthodontics rehabilitation. Even though treatment planning for these cohorts of patients has been recognized more so now than in the past, including greater emphasis in dental school curriculum, providing ideal and safe care with long-term clinical and patient outcomes is still a challenge. The concept of rationale treatment planning applies to this patient population when considering dental implants as long-term solution in oral rehabilitation.

From an oral health perspective, an increase in chronological age has traditionally been associated with an increase in tooth loss and partial edentulism [4–6], and

prevalence of total edentulism ranging from 1.3 to 78% worldwide (Figure 20.1) [7–9]. Prevalence of edentulism in most developed countries has shown a dramatic decline more recently [10]. Between 1990 and 2010, a decrease in global age-standardized prevalence of complete edentulism in individuals decreased from 4.4 to 2.4%, respectively [7]. This decrease is notable in developed countries as a result of various public health initiatives. However, when considering age, there exists a steep increase in prevalence of edentulism around the seventh decade of life. Reasons may include low utilization of dental services by this cohort, the lack of financial support from the government or thirdparty payment systems, and absence of relevant oral health policies [11]. The data indicates that even with a clear decline in edentulism, there will continue to be a need for replacement of teeth in the future, most especially among older patients. Dental care in the elderly can no longer be limited to removable complete prostheses. Retaining natural teeth in the older patient population has inadvertently led to dentistry's achievement of an adverse "consequence of success" as described by Joshi et al in 1996 [12]. With the increased retention of natural teeth in the elderly population, there is also an increased risk for developing periodontal disease or dental caries as well as increased need and demand for complex restorative care that provides not only function and form, but also includes expectations for esthetic outcomes. Furthermore, treatment planning for partially edentulous patients is a complex challenge as compared to edentulous patients, further necessitating the use of dental implants in a practitioner's armamentarium.

Studies have shown that tooth loss results in a reduced quality of life. Notable consequences of tooth loss include compromised masticatory ability with subsequent poor nutrition [13, 14], as well as restriction on social activities or

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Figure 20.1 Worldwide prevalence of edentulism among both sexes for ages 70 and above in 2019. Latin America and Caribbean had 47.69% of total prevalent cases compared to Sub-Saharan Africa at 13.95% among 70 years and older individuals. *Source:* Institute for Health Metrics and Evaluation. Used with permission. All rights reserved. Access 8/23/2021.

psychological well-being are observed [15]. With the advent of implant-retained and implant–supported prostheses, there has been a significant shift in treatment strategies to help improve stability and support in removable prostheses in edentulous patients and improve esthetics and function in partially edentulous patients. As patients' demand for more complex, multidisciplinary interventions increase, along with dental training in implant dentistry, it can be expected elderly individuals in high-income countries will choose the aforementioned to address their oral quality of life [6, 16, 17]. In the United States, implant dentistry was the only prosthodontic procedure that increased per capita from 1992 to 2007 and from prevalence of 0.7% in 1999–2000 to 5.7% in 2015–2016 [18, 19]. The largest absolute increase in prevalence of dental implants was observed among 65- to 75-year-old individuals [19]. In 2020, North America accounted for 38.2% revenue share, leading the worldwide implant market [20]. Driving forces include increased geriatric population with high oral healthcare needs and increased awareness of dental implants as well as introduction of advanced technologies and digital workflows [20].

Review of peer-reviewed scientific literature overwhelmingly supports the use dental implants as a reliable, predictable treatment modality for replacement of missing teeth in elderly patients [6, 21–24]. Studies have shown high implant

survival rates in patients 65 years and older. However, implant and prosthesis maintenance have become an important factor impacting long-term success that needs further investigation with increased use of implants surgically placed and restored using various prosthodontic designs. An elderly patients' ability to (i) maintain dental implants long-term considering their co-morbidities, (ii) perform oral hygiene habits, (iii) seek and obtain regular supportive care, and (iv) manage care of fixed and removable prostheses associated with the implants are important aspects reviewed in this chapter.

Rapid Oral Health Deterioration in Elderly Patients

Even though most studies focus on age when grouping patients based on demographic profiles, a functional definition for an elderly adult is at times more appropriate than a chronological one. In 1984, Ettinger and Beck developed a means of categorizing elderly patients based on their functional abilities as opposed to their age alone. The aging population can be categorized in one of three functional groups: functionally independent older adult, frail older adult, or functional dependent older adult [3]. Those

persons who are frail and functionally dependent are known to have higher rates of oral disease that develops over time due to the overall decline in their general health and function [25]. The rapid oral health deterioration (ROHD) concept was introduced to help identify patients who experience a decline in general health later in life and subsequently, a decline in oral health [3].

Advanced chronologic age is correlated with age-related diseases, with 92 age-related diseases accounting for 51.3% of the adult global burden [26]. According to the World Health Organization (WHO), the most common chronic conditions in the elderly include cardiovascular disease, cancer, respiratory diseases, diabetes mellitus, cirrhosis of the liver, osteoarthritis, and neurocognitive impairments like unipolar depression, Alzheimer's, and dementia. In addition, polypharmacy is a complex issue for the older population and can result in several oral side effects, such as xerostomia. These systemic diseases can affect oral health including indirect impact on dental implants and prostheses in various ways. For instance, the consequences of systemic disease can reduce a patient's ability to maintain proper oral hygiene, especially in those with dementia, depression, arthritis, and stroke; or predispose patients to more aggressive oral disease as seen with those with diabetes; and finally, diminish the effects of protective factors by reducing salivary flow as in patients with medication-induced xerostomia [27].

Social determinants can also lead to a reduction in access to needed care and ultimately, place a patient at risk for ROHD [3, 28–30]. The World Health Organization defines social determinants of health as "the conditions in which people are born, grow, work, live, and age, and the wider set of forces and systems shaping the conditions of daily life. These forces and systems include economic policies and systems, development agendas, social norms, social policies and political systems." [31] Our goal as clinicians is to provide the optimal care not just for today but for years to come, thus aiming for a patient-centered long-term outcome as opposed to mere procedure-centered outcomes. As best stated by Marchini and colleagues, it is critical to "look at a patient as a moving picture rather than a snapshot" [27].

Evidence-based risk factors for ROHD (Figure 20.2) should be reviewed when assessing a patient for any implant–supported or implant-retained treatment. A thorough medical and social history will ultimately help ascertain data relative to general health, social support, and oral conditions that may contribute to ROHD risk. Providers can categorize patients within one of four stages:

Stage 1: Risk factors for ROHD not present

Stage 2: Patient is at risk of ROHD, but not currently experiencing ROHD

Stage 3: Patient is currently experiencing ROHD Stage 4: ROHD has happened

Evaluating and assessing a patient for stages of ROHD ultimately enables a clinician to properly formulate a treatment plan, while also planning for failures and determining the complexity or aggressiveness nature for a proposed preventive plan. Again, the goal is to envision the patient as a *moving picture*. For instance, if a patient is a wellcontrolled diabetic via medication (HbA1c level of 6.5% or less), this condition is unlikely to affect the treatment plan for implant therapy and restorations at this point of time. In this example, diabetes is not considered a risk factor and does not impact implant survival up to five years after implant loading based on current evidence available [32]. In contrast, if a patient has been recently diagnosed with dementia, reports indicate that these types of patients have lower motivation to perform regular oral hygiene maintenance along with diminished cognitive and manual skills to perform adequate hygiene maintenance protocol [33]. Thus, without consistent home-care support, the longterm outcome of the implant and prostheses may be at risk. Implant and prosthesis maintenance should be taken into consideration at the time of treatment planning. In addition, long-term maintenance costs should be considered and included as part of the treatment plan discussion [34].

Treatment Modalities

Treatment plans should be formulated based on a thorough medical and dental evaluation of elderly patients and customized to everyone following the patient-centered approach to assess the possibilities for medical and oral deterioration [27]. There are multiple terminologies referring to this decision-making process of formulating a treatment plan. Examples include cost-effective care, minimally invasive dentistry, and rational dental care. In medicine, alternative treatment strategies have been proposed to develop "best sequence" treatment decisions in order to optimize long-term outcomes with both present and delayed benefits [35]. The concept of rational dental care applies to the elderly patient population when considering treatment plans that incorporate the use of implants for long-term clinical outcomes. Rational care, as described by Ettinger and Beck, is not a new concept; it emphasizes the importance of making thoughtful decisions with regards to patient treatment plans while considering modifying factors that will play a large role in the patient's systemic and oral health (Figure 20.2). Rational dental care has been described as clinical decision-making based on a patient's characteristics and consideration for modifying factors to formulate the best, individualized treatment plan [36]. Rational treatment plans can range from complex treatment, limited treatment, and emergency care to decisions that reflect no treatment for the individual. The plan should **Rational evaluation of elderly patients for dental implants**

Figure 20.2 Diagram shows a modified model for decision-making for older adults while considering risk factors for rapid oral health deterioration (ROHD). *Source:* Adopted from Ettinger and Beck, and Marchini et al. [27].

also include preventive and therapeutic treatments. Recall and professional maintenance regimen should be prescribed and individualized based on the soft and hard tissue risk assessment and supportive care [37].

When an elderly patient is categorized as Stage 1: "Risk factors for ROHD not present," an advanced treatment approach including a fixed-retained prosthesis can be proposed to restore form, function, and esthetics. For instance, implant–supported fixed complete dental prostheses have been documented to provide patients with improved function, esthetics, and quality of life [38]. Potential prosthetic complications in this prosthodontic approach include wear, chipping, and fracturing of the prosthetic teeth [39]. High patient satisfaction and oral health-related quality of life have been documented with favorable outcomes. However, sustained hygienic maintenance of a prosthesis has been reported as the most significant complaint by patients [40].

For a patient in the category of Stage 2: "Patient is at risk of ROHD, but not currently experiencing ROHD," a more conservative treatment approach [41] should be considered. Changing from a complex, intricate design for an implant– supported prosthesis to a simplistic, straightforward prosthetic design may be needed as the patient ages. As described by Muller, "today's fixed implant prosthesis should be considered for tomorrow's overdenture, at first retained by a bar, then a stud-type attachment, and converting to a lower retention attachment when the patient is in a palliative care setting." In addition, the prosthetic design should support a cleansable, hygienic environment that will allow a patient and their caregiver to maintain optimal oral health.

When a patient falls in the category of Stage 3: "Patient is currently experiencing ROHD," oral hypofunction, mastication functions, tongue pressure, tongue, and lip movement function deterioration will most likely be observed [42]. Therefore, a treatment plan involving removable implantretained prosthesis may be indicated to provide function and form. Esthetics may not be the top priority for this cohort, but comfort should be [43].

When a patient is determined to be in Stage 4: "ROHD has happened," the treatment plan should incorporate simple prosthetic design for ideal hygiene maintenance to be managed by the patient and if needed, by caregivers. Planning for an implant-retained prosthesis with complex and intricate design elements may not be indicated for a patient in the ROHD stage. Alternatively, a conventional prosthesis should be considered for these patients. The maintenance of a healthy intraoral environment and a prosthetic design with relatively easy access to home care cleaning and maintenance should be the primary goals for this cohort. Cognitive deterioration function should be assessed for the patient's understanding of the treatment plan and maintenance [43]. Finally, home caregivers should be educated and trained in providing care for the elderly, both the oral cavity and the prosthesis.

Recall and Maintenance in Elderly Patients

Routine patient recall appointments, professional hygiene maintenance appointments, and at-home hygiene maintenance regimens are essential for long-term success in elderly patients to decrease the frequency of biological, mechanical, and technical complications commonly associated with implant–supported prostheses [44]. Frequent, prescribed dental implant maintenance over a long-term period has been shown to be critical in implant survival [45]. According to recently published clinical practice guidelines for recall and maintenance of patients with implant–borne dental prostheses, patient recall frequencies should be more often than every six months if patients are considered as higher risk based on their age and ability to perform oral self-care and potential biological or mechanical complications [44] (Figure 20.3). During professional maintenance appointments, thorough extraoral and intraoral health and dental examinations, intraoral hygiene instructions, and hygiene instructions for cleaning the prostheses and oral hygiene intervention are performed. In addition, the prostheses should be professionally cleaned with established mechanical and chemical debridement and cleaning protocols.

In regards to professional prosthesis maintenance, the prostheses should be evaluated and assessed to determine need for adjustment, repair, replacement, or remake upon evaluation. Lastly, patient education is important for the person to learn about potential for anticipated issues to respond and seek consultation with the dentist, learn use of individualized home care instruments or cleaning aids, and learn cleaning protocol based on their specific needs. The various aspects addressed through the clinical practice guidelines are vital to help oral healthcare providers and caregivers improve clinical outcomes for patients.

There are confounding factors such as systemic health status, transportation, access to care, and financial standing of an elderly patient that may influence the commitment necessary for professional and at-home maintenance regimens and consequently, compromise outcomes related to tissue health and prosthesis longevity. Advanced age may be related to more difficulties in adapting to the new prostheses, delayed muscular function and adaptation, and post-insertion problems [22, 46]. Further, deteriorating maintenance conditions, such as reduced dexterity in the elderly [43], may also be observed. This may make it challenging for elderly patients to manage a removable implant

Figure 20.3 Clinical Practice Guidelines for implant–borne dental restorations from the American College of Prosthodontists. Accessed 8/23/2021 from the American College of Prosthodontists website [\(https://www.prosthodontics.org/assets/1/7/Final_ACP_](https://www.prosthodontics.org/assets/1/7/Final_ACP_Chairside_guide_July_2016.pdf) Chairside quide July 2016.pdf). Used with permission.

prosthesis since proper hygiene maintenance requires patient agility to manage insertion and removal of the prosthesis multiple times during a single day. Deterioration of a patient's social environment defined as lacking supportive care from either family members or caregivers, can influence access to care and prevent the elderly from receiving adequate professional care and maintenance.

There are some recommendations to promote recall and professional maintenance and preserve the prostheses outcomes in the elderly in anticipation of the patient's aging. For instance, anticipating or converting an implant– supported or implant-assisted prosthesis from a fixedretained design to a removable prosthesis for ease of cleaning and maintaining is highly recommended [34, 43, 47]. It may be challenging for those elderly in nursing home care with limited access to seek or conduct off-site professional care or continue self-care to keep up with their oral health follow-up and professional maintenance care [48]. Many elderlies in a nursing home or assisted living environments depend on the caregiver and other social services to provide oral hygiene care. Therefore, educating the caregivers with the appropriate information from journals, books, audio-visual media, and in-house training should be planned to provide oral health support for the elderly [49]. At the same time, preparing for increased home health and oral health care involving oral health professionals visiting the personal residences, assistedliving to transitional nursing home facilities, and hospitals for those patients who cannot attend regular maintenance care may be needed to support the elderly oral maintenance care [50].

At the policy level, recommendations to standardize implant components help ensure compatibility of implant instrumentation across various manufacturers, in order to evaluate and perform repair of implant superstructures. In

addition, it is important to ensure all oral health providers are competent in providing implant prosthetic maintenance and post-insertion, and post-delivery maintenance for their patients, especially those who provide care in nursing homes [43]. At a patient level, the use of a specific manufacturer for an implant system should be selected to ensure the availability to supply component parts over the lifetime of a patient. In addition, if a patient continues to experience natural tooth loss, a record of the implant system and components employed can be recorded on a personalized "implant card." This documentation of the implant system, component parts, and manufacturer identification codes should be provided to the patient in case they relocate, move, or transfer care elsewhere. In certain situations, it may be prudent as oral healthcare providers to provide the patient their casts or models used to fabricate the implant prostheses so that they may safely retain them for future reference in prostheses maintenance or replacement.

Conclusion

As a person ages, it is important to anticipate potential challenges due to consequences associated with aging when providing implant treatment. The Rapid Oral Health Deterioration staging classification is one means of rationalizing a patient's current status relative to general health, social support, and intraoral conditions while taking into consideration the potential of future adverse changes.

Key perspectives when considering incorporation of dental implants in treatment planning for elderly patients include the following:

● **Life expectancy and quality of life**: It is important to note that clinical decision-making relative to implant– supported prostheses should not be solely based on survival or success rates. The focus should be a patient's subjective gain of quality of life and overall well-being as well as anticipated life expectancy based on the patient's systemic conditions.

- **Rational treatment planning**: Careful consideration as to the final treatment plan, prosthesis type, and the ability to change from a fixed to a removable restoration or vice versa can be critical especially as the patient's general health, social support, and oral conditions deteriorate with time.
- **Prosthesis design**: Clinicians must also take into consideration the type of implant prosthesis and design in addition to a patient's ability and motivation to wear and/or maintain it as the person ages. An implant may successfully integrated with minimal bone loss; however, if the prosthesis cannot be utilized for its intended purpose due to the patient's inability to maintain it, then this cannot be considered a successful outcome [41]. Prosthodontic treatment plans should be ultimately designed to be cleansable for long-term maintenance, but also retrievable, repairable, and replaceable.
- **Implant systems and manufacturers**: Consideration to utilize one implant system over time for a patient is preferred as opposed to use of multiple implant systems with various implant component manufacturers along with providing the patient with a listing detailing implant specifications.
- **Access to care**: As the patient ages, their ability to schedule and commit to professional maintenance care may depend on several factors such as transportation and assistance to gain access to care. Identifying an appropriate support system is important for an elderly patient to maintain good oral health for overall health, yet the support system must adapt to increased needs of the patient and/or unexpected complications. Many elderly depend on their caregivers to maintain their oral health and general health. Therefore, educating the caregivers to raise the level of oral health literacy is essential in providing optimal care for the elderly.

Case reports of patients that required implant prostheses management over time, impacting clinical decision-making.

Case Report 20.1 Implant-retained overdenture

A 71-year-old edentulous male patient in good health presented for placement of two, mandibular implants, in September 2007. Following the prescribed healing period to achieve osseointegration, the mandibular denture was converted to mandibular implant-retained overdenture opposing a maxillary denture. At the time of active treatment, the patient was categorized as Stage 1: "Risk factors for ROHD not present." The patient continued to

report to the clinic on a regular basis for maintenance follow-ups, periodic exams, cleanings, attachment (Locator®; Zest Dental Solutions, Carlsbad, CA) inserts replacement, and ultimately, new prostheses as the old ones naturally wore away and required relines. In 2014, the patient stopped visiting the clinic regularly. Even though the patient's general health continued to be relatively good, the patient's support system diminished and evidence of ROHD surfaced after his wife passed away and children moved to other states. Patient was fully self-reliant and managed all his personal needs, including dental care. Patient returned to the clinic in 2021 at the age of 85 with the chief complaint of "my implant broke, and it took me two months to even find the number for this clinic." Clinical examination revealed soft tissue inflammation surrounding

Figure 20.4 Occlusal view of the mandibular abutments (Locator® patrices).

#27 implant Locator[®] attachment (patrix), absence of Locator® housing attachment, and a fragment of the insert lodged within the Locator® abutment (Figure 20.4). After the abutment was removed, it was noted that the remainder of the insert fragment had shifted apically along the abutment and determined to be the likely source of the inflammation (Figures 20.5 and 20.6).

Figure 20.5 After the Locator® implant abutment was removed, a portion of the matrix is shown as drifted apically

Figure 20.6 The portion of the matrix and the remainder of the matrix are shown after intraoral removal of the abutment.

and subgingivally.

Case Report 20.2 Implant-supported fixed dental prosthesis

A 62-year-old female reported to the clinic in 2011 with the chief complaint of missing mandibular anterior teeth. At the time, patient had a history of well-controlled schizophrenia, hypertension, goiter, and borderline diabetes. The patient was categorized as Stage 2: "At risk of ROHD, but not currently experiencing ROHD." Patient was deemed to have a strong social support system that tended to her needs and scheduled her for regular dental and medical appointments. Thus, an implant–supported fixed dental prosthesis was planned, and implants surgically placed. When the patient returned to her home country, the implants were restored by a local dentist (Figure 20.7). Because of the economic deterioration in her home country in approximately 2016, the patient was unable to obtain the necessary hygiene maintenance care. The patient's diagnosis of schizophrenia, need for hospitalization on multiple occasions, medicationinduced xerostomia, and lack of trust in the dental care

within her country ultimately led to poor follow-up and professional maintenance care. In 2019, the 70-year-old patient returned to the United States for follow-up with the chief complaint of "I think my implants are failing. I have a bad taste in my mouth." Based on a review of her medical and social history, patient had and continued to experience Rapid Oral Health Deterioration. Risk factors for ROHD had increased markedly in a short period of time. Her implants showed clinical signs of periimplantitis, and the patient was treatment planned for removal of the implants; the prosthesis was poorly maintained and multiple teeth had deteriorated and were non-restorable (Figure 20.8). One implant and several teeth were extracted, and the patient was temporarily restored with interim partial dentures in anticipation of alternating her treatment plan to an implant–supported overdenture. The patient passed away within 1.5 years due to a rapid decline in her medical and mental health.

Figure 20.7 Radiographs of mandibular dentition, images dated 2014.

Figure 20.8 Radiographs of mandibular dentition, images dated 2019.

Case Report 20.3 Fixed complete denture

A 65-year-old patient reported to the hospital with a chief complaint of fractured front tooth of the fixed complete denture and difficulty in cleaning (Figures 20.9 and 20.10). The patient was categorized as Stage 1: "Risk factors for ROHD not present" and treatment was completed. The patient was motivated and demonstrated the ability to perform at-home care of the prosthesis. The patient returned after completion of treatment approximately 4.5 years later and since the initial treatment, the patient's dexterity decreased, and he no longer was as motivated in cleaning his prosthesis. After

Figure 20.9 Maxillary fixed implant denture shows signs of wear, including chipping of the anterior tooth.

removal of the maxillary fixed implant complete denture and assessing aspects of the prosthesis design, it was observed that the cameo surface (tissue surface) contours did not incorporate ideal convexity for at-home cleaning and maintenance of the prosthesis (Figure 20.11). Due to the difficulty in the patient's ability to clean the prosthesis intraorally, the decision was made to convert his existing maxillary fixed complete denture to an implant–supported overdenture (Figures 20.12–20.14). *Case and photos courtesy of Dr. Alvin G. Wee; VA Hospital, Omaha, Nebraska*.

Figure 20.10 Occlusal view of the fixed implant denture shows the signs of wear, including chipping of the anterior tooth.

Figure 20.11 Intaglio view of the maxillary fixed implant denture revealed the contours were not ideal and not conducive for proper home care hygiene maintenance and for patient ease in cleaning the prosthesis intraorally.

Figure 20.12 Intraoral view of the new prosthesis, redesigned with a fixed bar infrastructure, that incorporates overdenture attachments (patrices) for the suprastructure – maxillary overdenture.

Figure 20.13 Intaglio view of the maxillary overdenture that incorporates the custom suprastructure housing for the attachment retention elements (matrices).

Figure 20.14 Frontal view of the completed prostheses in the patient.

References

- **1** Orimo, H., Ito, H., Suzuki, T. et al. (2006). Reviewing the definition of "elderly". *Geriatrics & Gerontology International 6* (3): 149–158.
- **2** WHO (n.d.). 10 Facts on ageing and the life course. World Helath Organization: WHO Retrieved from [https://www.](https://www.who.int/features/factfiles/ageing/ageing_facts/en/) [who.int/features/factfiles/ageing/ageing_facts/en/](https://www.who.int/features/factfiles/ageing/ageing_facts/en/) (accessed 22 August 2021).
- **3** Ettinger, R.L. and Beck, J.D. (1984). Geriatric dental curriculum and the needs of the elderly. *Special Care in Dentistry 4* (5): 207–213.
- **4** Feine, J., Carlsson, G., Awad, M. et al. (2002). The McGill consensus statement on overdentures. Mandibular two-implant overdentures as first choice standard of care for edentulous patients. *Gerodontology 19* (1): 3–4.
- **5** Müller, F., Naharro, M., and Carlsson, G.E. (2007). What are the prevalence and incidence of tooth loss in the adult and elderly population in Europe? *Clinical Oral Implants Research 18*: 2–14.
- **6** Schimmel, M., Müller, F., Suter, V., and Buser, D. (2017). Implants for elderly patients. *Periodontology 2000 73* (1): 228–240. [https://doi.org/10.1111/prd.12166.](https://doi.org/10.1111/prd.12166)
- **7** Kassebaum, N., Bernabé, E., Dahiya, M. et al. (2014). Global burden of severe tooth loss: a systematic review and meta-analysis. *Journal of Dental Research 93* (7_suppl): 20S–28S.
- **8** Petersen, P.E. (2009). Global policy for improvement of oral health in the 21st century–implications to oral health research of World Health Assembly 2007, World Health Organization. *Community Dentistry and Oral Epidemiology 37* (1): 1–8.
- **9** Polzer, I., Schimmel, M., Müller, F., and Biffar, R. (2010). Edentulism as part of the general health problems of elderly adults. *International Dental Journal 60* (3): 143–155.
- **10** Khazaei, S., Firouzei, M.S., Sadeghpour, S. et al. (2012). Edentulism and tooth loss in Iran: SEPAHAN systematic review No. 6. *International Journal of Preventive Medicine* 3 (Suppl1): S42.
- **11** Petersen, P.E., Kandelman, D., Arpin, S., and Ogawa, H. (2010). Global oral health of older people-call for public health action. *Community Dental Health 27* (4): 257–267.
- **12** Joshi, A., Douglass, C.W., Feldman, H. et al. (1996). Consequences of success: do more teeth translate into more disease and utilization? *Journal of Public Health Dentistry 56* (4): 190–197.
- **13** Fontijn-Tekamp, F., Slagter, A., Van Der Bilt, A. et al. (2000). Biting and chewing in overdentures, full dentures, and natural dentitions. *Journal of Dental Research 79* (7): 1519–1524.
- **14** Sheiham, A., Steele, J., Marcenes, W. et al. (2001). The relationship among dental status, nutrient intake, and nutritional status in older people. *Journal of Dental Research 80* (2): 408–413.
- **15** Bouma, J., Boerrigter, L.M., Van Oort, R.P. et al. (1997). Psychosocial effects of implant-retained overdentures. *International Journal of Oral and Maxillofacial Implants 12* (4): 515–522.
- **16** Madianos, P., Papaioannou, W., Herrera, D. et al. (2016). EFP Delphi study on the trends in periodontology and periodontics in Europe for the year 2025. *Journal of Clinical Periodontology 43* (6): 472–481.
- **17** Meijer, H.J., Batenburg, R.H., and Raghoebar, G.M. (2001). Influence of patient age on the success rate of dental implants supporting an overdenture in an edentulous mandible: a 3-year prospective study. *International Journal of Oral and Maxillofacial Implants 16* (4): 522–526.
- **18** Eklund, S.A. (2010). Trends in dental treatment, 1992 to 2007. *The Journal of the American Dental Association* 141 (4): 391–399.
- **19** Elani, H.W., Starr, J.R., Da Silva, J.D., and Gallucci, G.O. (2018). Trends in dental implant use in the U.S., 1999-2016, and projections to 2026. *Journal of Dental Research 97* (13): 1424–1430. [https://doi.org/10.1177/](https://doi.org/10.1177/0022034518792567) [0022034518792567.](https://doi.org/10.1177/0022034518792567)
- **20** Grand View Research (2021). Dental Implant Market Size, Share & Trends Analysis Report By Implants Type (Titanium, Zirconium), By Region (North America, Europe, Asia Pacific, Latin America, MEA), And Segment Forecasts, 2021–2028. [https://www.grandviewresearch.](https://www.grandviewresearch.com/industry-analysis/dental-implants-market) [com/industry-analysis/dental-implants-market](https://www.grandviewresearch.com/industry-analysis/dental-implants-market) (accessed 23 August 2021).
- **21** De Baat, C. (2000). Success of dental implants in elderly people-a literature review. *Gerodontology 17* (1): 45–48.
- **22** Jemt, T. (1993). Implant treatment in elderly patients. *International Journal of Prosthodontics 6* (5): 456–461.
- **23** Schimmel, M., Srinivasan, M., McKenna, G., and Muller, F. (2018). Effect of advanced age and/or systemic medical conditions on dental implant survival: a systematic review and meta-analysis. *Clinical Oral Implants Research 29* (Suppl 16): 311–330. [https://doi.org/10.1111/clr.13288.](https://doi.org/10.1111/clr.13288)
- **24** Srinivasan, M., Meyer, S., Mombelli, A., and Muller, F. (2017). Dental implants in the elderly population: a systematic review and meta-analysis. *Clinical Oral Implants Research 28* (8): 920–930. [https://doi.org/](https://doi.org/10.1111/clr.12898) [10.1111/clr.12898](https://doi.org/10.1111/clr.12898).
- **25** Chen, X., Clark, J.J., Preisser, J.S. et al. (2013). Dental caries in older adults in the last year of life. *Journal of the American Geriatrics Society 61* (8): 1345–1350.
- **26** Chang, A.Y., Skirbekk, V.F., Tyrovolas, S. et al. (2019). Measuring population ageing: an analysis of the global burden of disease study 2017. *The Lancet Public Health 4* (3): e159–e167.
- **27** Marchini, L., Hartshorn, J.E., Cowen, H. et al. (2017). A teaching tool for establishing risk of oral health deterioration in elderly patients: development, implementation, and evaluation at a U.S. Dental School. *Journal of Dental Education 81* (11): 1283–1290. <https://doi.org/10.21815/JDE.017.086>.
- **28** Friedman, P.K., Kaufman, L.B., and Karpas, S.L. (2014). Oral health disparity in older adults: dental decay and tooth loss. *Dental Clinics of North America 58* (4): 757–770.
- **29** Manski, R.J., Cohen, L.A., Brown, E. et al. (2014). Dental service mix among older adults aged 65 and over, United States, 1999 and 2009. *Journal of Public Health Dentistry 74* (3): 219–226.
- **30** Manski, R.J., Moeller, J.F., Chen, H. et al. (2015). Dental use and expenditures for older uninsured Americans: the simulated impact of expanded coverage. *Health Services Research 50* (1): 117–135.
- **31** WHO (n.d.). Social determinants of health. [https://www.](https://www.who.int/health-topics/social-determinants-of-health#tab=tab_1) [who.int/health-topics/social-determinants-of-health#tab=](https://www.who.int/health-topics/social-determinants-of-health#tab=tab_1) [tab_1](https://www.who.int/health-topics/social-determinants-of-health#tab=tab_1) (accessed 22 August 2021).
- **32** Fretwurst, T. and Nelson, K. (2021). Influence of medical and geriatric factors on implant success: an overview of systematic reviews. *The International Journal of Prosthodontics 34*: s21–s26. [https://doi.org/10.11607/](https://doi.org/10.11607/ijp.7000) [ijp.7000](https://doi.org/10.11607/ijp.7000).
- **33** Gil-Montoya, J.A., Sánchez-Lara, I., Carnero-Pardo, C., Fornieles-Rubio, F., Montes, J., Barrios, R., Gonzalez-Moles, M.A. and Bravo, M. (2017). Oral hygiene in the elderly with different degrees of cognitive impairment and dementia. *Journal of the American Geriatrics Society*, *65*(3) 642–647.
- **34** Bartold, P.M., Ivanovski, S., and Darby, I. (2016). Implants for the aged patient: biological, clinical and sociological considerations. *Periodontology 2000 72* (1): 120–134. <https://doi.org/10.1111/prd.12133>.
- **35** Simoneau, G., Moodie, E.E., Azoulay, L., and Platt, R.W. (2020). Adaptive treatment strategies with survival outcomes: an application to the treatment of type 2 diabetes using a large observational database. *American Journal of Epidemiology 189* (5): 461–469.
- **36** Ettinger, R. (2015). Treatment planning concepts for the ageing patient. *Australian Dental Journal 60*: 71–85.
- **37** Afshari, F.S., Campbell, S.D., Curtis, D.A. et al. (2019). Patient-specific, risk-based prevention, maintenance, and supportive care: a need for action and innovation in education. *Journal of Prosthodontics 28* (7): 775–783.
- **38** Del Fabbro, M., Bellini, C.M., Romeo, D., and Francetti, L. (2012). Tilted implants for the rehabilitation of

edentulous jaws: a systematic review. *Clinical Implant Dentistry and Related Research* 14 (4): 612–621.

- **39** Box, V.H., Sukotjo, C., Knoernschild, K.L. et al. (2018). Patient-reported and clinical outcomes of implantsupported fixed complete dental prostheses: a comparison of metal-acrylic, milled zirconia, and retrievable crown prostheses. *The Journal of Oral Implantology 44* (1): 51–61.
- **40** Martín-Ares, M., Barona-Dorado, C., Guisado-Moya, B. et al. (2016). Prosthetic hygiene and functional efficacy in completely edentulous patients: satisfaction and quality of life during a 5-year follow-up. *Clinical Oral Implants Research 27* (12): 1500–1505.
- **41** Müller, F. and Schimmel, M. (2016). Revised success criteria: a vision to meet frailty and dependency in implant patients. *The International Journal of Oral & Maxillofacial Implants 31* (1): 15–15.
- **42** Liu, B., Dion, M.R., Jurasic, M.M. et al. (2012). Xerostomia and salivary hypofunction in vulnerable elders: prevalence and etiology. *Oral Surgery, Oral Medicine, Oral Pathology, and Oral Radiology 114* (1): 52–60.
- **43** Sato, Y., Kitagawa, N., and Isobe, A. (2020a). Current consensus of dental implants in the elderly – what are the limitations? *Current Oral Health Reports 7* (3): 321–326. <https://doi.org/10.1007/s40496-020-00268-0>.
- **44** Bidra, A.S., Daubert, D.M., Garcia, L.T. et al. (2016). Clinical practice guidelines for recall and maintenance of patients with tooth-borne and implant-borne dental restorations. *Journal of Prosthodontics 25* (Suppl 1): S32–S40. [https://doi.org/10.1111/jopr.12416.](https://doi.org/10.1111/jopr.12416)
- **45** Gay, I., Tran, D., Weltman, R. et al. (2016). Role of supportive maintenance therapy on implant survival: a university-based 17 years retrospective analysis. *International Journal of Dental Hygiene 14* (4): 267–271.
- **46** Engfors, I., Örtorp, A., and Jemt, T. (2004). Fixed implant-supported prostheses in elderly patients: a 5-year retrospective study of 133 edentulous patients older than 79 years. *Clinical Implant Dentistry and Related Research 6* (4): 190–198.
- **47** Feine, J., De Grandmont, P., Boudrias, P. et al. (1994). Within-subject comparisons of implant-supported mandibular prostheses: choice of prosthesis. *Journal of Dental Research 73* (5): 1105–1111.
- **48** Sweeney, M., Williams, C., Kennedy, C. et al. (2007). Oral health care and status of elderly care home residents in Glasgow. *Community Dental Health 24* (1): 37–42.
- **49** Hopcraft, M., Morgan, M., Satur, J., and Wright, F. (2008). Dental service provision in Victorian residential aged care facilities. *Australian Dental Journal 53* (3): 239–245.
- **50** Hagiwara, Y., Ohyama, T., Yasuda, H. et al. (2021). Dental implant status in elderly individuals requiring domiciliary dental care in Japan. *International Journal of Implant Dentistry 7* (1): 53. [https://doi.org/10.1186/](https://doi.org/10.1186/s40729-021-00340-z) [s40729-021-00340-z.](https://doi.org/10.1186/s40729-021-00340-z)

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Disinfection of Implant Prosthetic Components Before Delivery

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Background

The subgingival oral biofilm is generally criticized for the initiation and progression of peri-implant diseases, periimplant mucositis, and peri-implantitis [1]. The oral biofilm harbors pathogenic bacteria such as *Aggregatibacter actinomycetemcomitans* (*A. actinomycetemcomitans*), *Prevotella intermedia* (*P. intermedia*), and *Porphyromonas gingivalis* (*P. gingivalis*) that have been directly related with the etiopathogenesis and prognosis of peri-implant diseases [2–5]. Local and systemic risk factors that are commonly associated with the increased colonization of pathogenic bacteria around implants include poor oral hygiene status and tobacco-smoking and poorly controlled diabetes mellitus, respectively [6–8]. However, handling of implant prosthetic components by staff and laboratory personnel can also increase the risk of microbial contamination [9, 10]. A major challenge in two-phase implant treatment (delayed loaded implants) is the risk of microbial leakage at the implant–abutment connection (IAC) due to the presence of microgaps [11, 12]. An increased bacterial colonization has also been identified on customized titanium abutments [9]. Biologically, molecules such as toxins and constituents of bacterial wall are accountable for peri-implant inflammatory reactions. These molecules (endotoxins) penetrate the microgaps at the IAC instead of the whole bacterial cell and enhance an inflammatory response and jeopardize the surrounding bone [13, 14]. Moreover, the extent of bacterial contamination at the IAC depends on the accuracy of fit between the fixture and the abutment, tightening torque and micro-movements between the connected components during chewing. This is concerning as presence of pathogenic bacteria at the IAC increases the risk of future peri-implant soft tissue

inflammation and crestal bone loss (CBL) even in individuals stringently following routine oral hygiene maintenance protocols such as toothbrushing and flossing of interproximal spaces. It has also been proposed that complete removal of contaminants and pollutants from implant abutments improves the preload maintenance of the screw/abutment complex [15].

Objective

This chapter aims to provide an evidence-based review of methods that are used to disinfect implant abutments before insertion.

Literature Search Strategy

Indexed databases namely PubMed/Medline, Google Scholar, and ISI Web of Knowledge were searched without time and language barriers to identify studies that assessed methods of disinfection of implant abutments before delivery. The pattern of this chapter was customized to mainly summarize the relevant information.

Methods of Implant Abutment Disinfection

Argon Plasma

Mixtures of gases such as argon (Ar) and oxygen are metastable rare gases that carry significant amounts of energy; and lead to the formation of reactive oxygen species (ROS) via energy transfer reactions. The final manufacturing

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process of Titanium implants involves treatment with argon plasma (AP) prior to sterilization by gamma rays. In this technique, electronic mantle of materials is activated by a spray of argon under room temperature and pressure. This procedure helps remove contamination and microbiologic pollution from metal surfaces. Simultaneously, AP also helps modify the physico-chemical, and biological features of implant surfaces, and their ability to adapt with the surrounding soft and osseous tissues [16–18]. In vitro studies [19, 20] have assessed the effect of AP on different implant surfaces with reference to adhesion of osteoblasts, fibroblasts, and microbial decontamination. The scanning electron microscopy results showed that AP reduces bacterial adhesion to implant discs and promotes spreading and attachment of fibroblasts and osteoblasts [19, 20]. In an experimental study on canine-models, Coelho et al. [18] assessed the effect of AP applications immediately prior to dental implant placement on peri-implant bone. Histomorphometric examinations performed at three weeks showed a significantly higher bone-to-implant contact percentage (>300%) and mean bone area fraction occupancy (>30%) in AP-treated implant surfaces compared with controls (untreated surfaces) [18]. In a randomized controlled trial (RCT), Canullo et al. [21] investigated radiographic osseous changes around customized, platform-switched abutments placed according to the "one-abutment-one-time" protocol, with (test-group) and without (control-group) PA cleaning treatment in 30 patients with a history of periodontal disease and thin gingival phenotype (<1mm). Patients were randomly divided into test and control groups immediately before abutment connection. In the control group, cleaning protocol comprised steaming. Outcome measures were: (i) implant and prosthesis success rate; (ii) prosthetic and biological complications; (iii) CBL; (iv) esthetic and periodontal parameters; and (v) patient satisfaction [21]. At five-years'

follow-up, none of the implants in either group were lost; however, CBL was significantly higher in the control than the test group. Periodontal parameters were stable in both groups at 60 months of follow-up. This study [21] concluded that AP treatment is a useful technique for disinfection of implant abutments before connection. This study was basically a long-term (5-years follow-up) of an interim RCT, which was followed up for 24 months [22]. Outcomes of this interim study [22] were also similar with those reported in the five-year follow-up RCT [21], that is, disinfection of implant abutments using AP removes bacteria and minimizes the risk of CBL to a much greater extent compared with steam disinfection (SD). It is known that a history of periodontitis is a risk factor for peri-implant diseases including peri-implantitis [23–25]. In this context, the risk of future peri-implantitis is higher in these patients in case traditional methods, such as SD are used to decontaminate implant abutments before delivery. Therefore, abutment disinfection using AP is a reliable strategy to minimize bacterial colonization and leakage of their biproducts at the IAC. This may help facilitate long-term success and survival of dental implants. An overview of studies, which assessed the role of AP in abutment disinfection is presented in Table 21.1.

Chlorhexidine

In a double blinded RCT [26], investigators applied 0.2% chlorhexidine (CHX) gel (16 patients) or a placebo gel $(n = 16$ patients) to the IAC during all surgical and prosthetic phases and assessed the effect on CBL for up to one year. The one-year follow-up results showed a significantly higher CBL around implants treated with the placebo gel compared with those in which, 0.2% CHX as applied at the IAC [26]. However, implants in both groups showed minimal peri-implant plaque and gingival bleeding scores at

Table 21.1 An overview of studies, which assessed the role of AP in abutment disinfection.

AP, Argon plasma; NA, Not applicable; RCT, Randomized controlled trial.

one-year of follow-up. This study [26] concluded that application of 0.2% CHX gel at the IAC minimizes the risk of CBL for up to 12 months. One limitation of this study is that microbiological assessment of subgingival oral biofilm samples and immunological investigations (for instance, assessment of destructive inflammatory cytokines such as tumor necrosis factor-alpha and interleukin 1-beta in the peri-implant sulcular fluid [PISF]) were not performed. It is tempting to speculate that application of 0.2% CHX gel to the IAC during all surgical and prosthetic phases reduces the counts of pathogenic bacteria (such as *A. actinomycetemcomitans, P. gingivalis*, and *P. intermedia*) in the subgingival oral biofilm; and reduces the expression of proinflammatory cytokines in the PISF thereby contributing toward the maintenance of peri-implant health. Another double-blinded RCT [27] quantitative investigated, the presence of *P. gingivalis* on the healing abutment when 0.20% CHX gel or a placebo gel were applied during the first and second implant surgical stages. Besides recording peri-implant plaque and gingival bleeding scores, inflammatory infiltrate and the micro-vessel density surrounding peri-implant soft tissues were also assessed at three-four points (at the time of implant insertion, second stage surgery, prosthetic phase, and 12 months of follow-up). At one-year follow-up, all implants showed low scores of plaque and gingival bleeding; however, a significantly low count of *P. gingivalis* was observed in healing abutments belonging to the 0.2% CHX group compared with the placebo group [27]. Moreover, the placebo group demonstrated the highest expression of inflammation markers for B-lymphocytes, T-lymphocytes, and macrophages cluster definitions (CD) (CD3, CD20, and CD68). Thus, use of 0.2% CHX is most likely a reliable treatment option in clinical implant dentistry as it significantly reduces *P. gingivalis* counts and host inflammatory response around implant abutments. However, in a recent in vitro study [28], antibacterial efficacy of 0.2% CHX was compared with 0.8% hyaluronic acid (HA) against *P. gingivalis* strains. The experimental results showed that 0.8% HA is a more potent antibacterial agent against *P. gingivalis* strains compared with 0.2% CHX [28]. By no means should this result tempt readers to perceive that 0.2% CHX is inferior to other pharmacologic preparations in terms of its antibacterial efficacy as there was a marked difference in the concentrations of both tested preparations. There is a possibility that 0.2% CHX gel is as effective as 0.2% HA in demonstrating antibacterial actions against *P. gingivalis* strains; however, an in vitro study reported that 0.2% CHX has no effect on bacterial endotoxin that possesses the ability to invade the IAC [29]. With regards to CHX rinsing, results from a clinical study [10] on 10 patients showed that CHX rinsing does not have any effect toward decontamination at IAC.

The study showed presence of bacteria (incl. anaerobic species) after a continuous irrigation (for 3mm) within the butt-joint connections after the use of CHX. However, results of a six-months follow-up RCT [30] showed that 0.1% CHX gel displays antibacterial activity against a variety of microbes including *P. intermedia*, *A. actinomycetemcomitans*, *P. gingivalis*, *Fusobacterium nucleatum,* and *Treponema denticola*. In summary, the antibacterial effectiveness of CHX at the IAC remains debatable predominantly due to limited clinical evidence. Moreover, it remains to be determined what concentration (0.1%, 0.2%, or another concentration) is most relevant in terms of minimizing the risk of bacterial contamination at the IAC immediately after prosthetic loading (Table 21.2).

Nanoparticles

Nanostructures or nanoparticles are derived from a variety of metals such as copper and zinc [32]. Studies have shown that nanoparticles possess unique physiochemical properties [33]; and their varying shapes (cylindrical, cubic, and spherical), sizes (5−100nm), and larger contact surfaces compared with microparticles generate a change in the physical properties, which results in different degrees of damage to bacterial cells [34]. In Gram-negative bacteria, nanoparticles act chiefly in the range of 1−10nm [35]; and small-sized oxidized nanoparticles are more toxic than larger non-oxidized particles [36]. To date, one in vitro study [32] has assessed the effects of zinc oxide (ZnO) and copper nanoparticles against *Streptococcus sanguinis* and *P. gingivalis* models in cell cultures of human gingival fibroblasts (HGFs). Both NPs induced mitochondrial dosedependent cytotoxicity, ZnO nanoparticles increases actate dehydrogenase release and intracellular ROS production. The results showed that nanoparticles exhibit a bactericidal effect against pathogenic bacteria [32]. It has been reported that nanoparticles improve the proliferation and adhesion of osteoblast cell lines [37]. The mode of action of nanoparticles is summarized in Figure 21.1. There are several unanswered questions related to the significance of nanoparticles with reference to clinical implant dentistry; however, it is hypothesized that incorporation of nanoparticles in custom abutments minimized bacterial colonization at the IAC and helped maintain crestal bone height. However, there are no clinical studies in this regard.

Hydrogen Peroxide

Hydrogen peroxide $(H₂O₂)$ possesses antibacterial properties in biological systems [38]. The broad-spectrum antimicrobial activity, ease of use, and safety profile makes $H₂O₂$ a potent agent for sterilization and disinfection

Table 21.2 An overview of studies, which assessed the role of chlorhexidine in abutment disinfection.

CB, Crestal bone loss; CHX, Chlorhexidine; *E. Coli, Escherichia coli*; IAC: Implant abutment connection; RCT: Randomized controlled trial. *a* Microbiological assessment was not performed.

Figure 21.1 Mode of action of nanoparticles.

purposes [39]. In clinical implant dentistry and related research, antibacterial efficacy of 10% H₂O₂ irrigation has been shown to be superior to 0.9% saline and 0.2% CHX [40]. A recent study [41] compared the effect of different protocols (0.2% CHX, 0.9% saline, 10% H₂O_{2,} and 70% alcohol) toward implant internal decontamination (IID). The results showed that out of all the disinfection agents assessed, 10% $H₂O₂$ was most effective toward IID [41]. However, in a literature review in which, Claffey et al. [42] reviewed 43 clinical and experimental studies showed that none of the decontamination protocols (saline, CHX, and/or H_2O_2) are superior to the other in

terms of implant surface disinfection. The current understanding is that H_2O_2 has been used to eradicate microbes from contaminated implant surfaces; however, there are no studies in indexed literature that have evaluated the efficacy of H_2O_2 in disinfecting implant prosthetic components (including restorations) immediately before delivery.

Ethyl Alcohol

Denaturation of microbial proteins is the most likely mechanism through which ethyl alcohol exerts its antimicrobial effects. Absolute ethyl alcohol is a dehydrating agent, which is less bactericidal than mixtures of alcohol and water as proteins are denatured more rapidly in an aqueous medium [43]. Ethanol, at a concentration of at least 70% has been labeled as an "antimicrobial lock solution" [44]; however, ethanol concentrations over 28% have been associated with side effects including protein denaturation, dizziness, and blood clotting [44]. There is a possibility that ethyl alcohol is suitable for disinfecting implant abutments immediately before implant prosthesis delivery. However, to date, there are no clinical trials and/or in vitro experiments that have tested this hypothesis. Further welldesigned and power-adjusted studies are needed to test this hypothesis.

Iodine

In orthopedic surgery, iodine-coated titanium implants can be advantageous in terms of prevention of post-operative infections [45]. Tsuchiya et al. [45] performed a clinical trial to evaluate the antimicrobial activity of iodine-coated and non-coated titanium hip implants. In this trial [45], 222 patients (mean age approximately 49 years) with postoperative infections were treated with iodine-coated titanium implants. The 12-months follow-up showed that all implants had osseointegrated and there were no complications (such as loosening of implant and infections at the surgical site). Also, bone ingrowth was radiographically observed around all hip prostheses. Since achievement of osseointegration accompanied by the least possible risk of infection is a prerequisite for a successful dental implant therapy, it is speculated that coating implant abutments with iodine immediately before prosthetic loading plays a role in achieving osseointegration and minimizing the risk of microbial colonization at the IAC. Regrettably, there are no clinical trials and/or experimental studies that have tested this hypothesis. This warrants additional welldesigned and power-adjusted clinical and experimental investigations.

Citric Acid

In oral implantology, citric acid (CA) is used to disinfect infect implant surfaces due to its antibacterial properties against biofilm formation without jeopardizing periodontal tissues [46, 47]. Immersion of titanium implants in CA may slightly increase implant surface roughness $\left($ <0.2 μ m); however, this does not facilitate adherence of biofilm on implant surfaces [48]. The CA is characterized by high concentration and low pH; however, this does not affect cellular activity on the titanium surfaces [49]. In a radiologic, histologic, and histomorphometric evaluation on canine models, Htet et al. [46] reported that CA possesses great

Figure 21.2 The unknown dilemma. What is the best way to disinfect an implant-retained prosthesis, an abutment, or other prosthetic components (alcohol, iodine, H_2O_2 , or CHX) in Implant Dentistry?

potential for disinfection of implant surfaces and facilitates stability of peri-implant crestal bone height. It is therefore likely that immersion of implant abutments in CA immediately before prosthetic loading minimizes the risk of bacterial contamination at the IAC, which in turn reduces the chances of future occurrences of peri-implant diseases. Lamentably, so far this hypothesis remains under investigation. This warrants additional studies.

Authors' Perspective

Prevention of microbial colonization around implant surfaces including the IAC plays an important role in minimizing the risk of soft tissue-related and osseous complications, peri-implant mucositis, and periimplantitis. To date, the ideal disinfection protocol for disinfecting implant abutments, as well as implant-retained prostheses, immediately before prosthetic loading remains unknown. Moreover, the precise concentrations and duration/frequency of disinfection treatment remain undetermined. Based upon currently available indexed scientific evidence, it seems that the subject "Disinfection of implant prosthetic components (including restorations) before delivery" deserves further attention from clinicians and researchers (Figure 21.2).

Abbreviations

- A. *actinomycetemcomitans*
	- *Aggregatibacter actinomycetemcomitans*
- AP Argon plasma
- Ar Argon
- CA Citric acid
- CBL Crestal bone loss

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References

- **1** Belibasakis, G.N., Charalampakis, G., Bostanci, N., and Stadlinger, B. (2015). Peri-implant infections of oral biofilm etiology. *Advances in Experimental Medicine and Biology* 830: 69–84. [https://doi.](https://doi.org/10.1007/978-3-319-11038-7_4) [org/10.1007/978-3-319-11038-7_4.](https://doi.org/10.1007/978-3-319-11038-7_4)
- **2** Ingendoh-Tsakmakidis, A., Mikolai, C., Winkel, A. et al. (2019). Commensal and pathogenic biofilms differently modulate peri-implant oral mucosa in an organotypic model. *Cellular Microbiology* 21 (10): e13078. [https://doi.](https://doi.org/10.1111/cmi.13078) [org/10.1111/cmi.13078](https://doi.org/10.1111/cmi.13078).
- **3** Szafrański, S.P., Winkel, A., and Stiesch, M. (2017). The use of bacteriophages to biocontrol oral biofilms. *Journal of Biotechnology* 250: 29–44. [https://doi.org/10.1016/](https://doi.org/10.1016/j.jbiotec.2017.01.002) [j.jbiotec.2017.01.002.](https://doi.org/10.1016/j.jbiotec.2017.01.002)
- **4** Kim, S.G., Hong, J.Y., Shin, S.I. et al. (2016). Prevalence of Porphyromonas gingivalis fimA genotypes in the periimplant sulcus of Koreans assessed using a new primer. *Journal of Periodontal & Implant Science* 46 (1): 35–45. [https://doi.org/10.5051/jpis.2016.46.1.35.](https://doi.org/10.5051/jpis.2016.46.1.35)
- **5** Polymeri, A., van der Horst, J., Buijs, M.J. et al. (2021). Submucosal microbiome of peri-implant sites: a crosssectional study. *Journal of Clinical Periodontology* 48 (9): 1228–1239. [https://doi.org/10.1111/jcpe.13502.](https://doi.org/10.1111/jcpe.13502)
- **6** Javed, F., Rahman, I., and Romanos, G.E. (2019). Tobaccoproduct usage as a risk factor for dental implants. *Periodontology 2000* 81 (1): 48–56. [https://doi.org/10.1111/](https://doi.org/10.1111/prd.12282) [prd.12282.](https://doi.org/10.1111/prd.12282)
- **7** Alsahhaf, A., Al-Aali, K.A., Alshagroud, R.S. et al. (2019). Comparison of yeast species in the subgingival oral biofilm of individuals with type 2 diabetes and peri-implantitis and individuals with peri-implantitis without diabetes. *Journal of Periodontology* 90 (12): 1383–1389. [https://doi.](https://doi.org/10.1002/jper.19-0091) [org/10.1002/jper.19-0091.](https://doi.org/10.1002/jper.19-0091)
- **8** Javed, F. and Romanos, G.E. (2019). Chronic hyperglycemia as a risk factor in implant therapy. *Periodontology 2000* 81 (1): 57–63. [https://doi.org/10.1111/](https://doi.org/10.1111/prd.12283) [prd.12283.](https://doi.org/10.1111/prd.12283)
- **9** Canullo, L., Micarelli, C., Lembo-Fazio, L. et al. (2014). Microscopical and microbiologic characterization of customized titanium abutments after different cleaning procedures. *Clinical Oral Implants Research* 25 (3): 328–336. [https://doi.org/10.1111/clr.12089.](https://doi.org/10.1111/clr.12089)
- **10** Romanos, G.E., Biltucci, M.T., Kokaras, A., and Paster, B.J. (2016). Bacterial composition at the implantabutment connection under loading in vivo. *Clinical Implant Dentistry and Related Research* 18 (1): 138–145. [https://doi.org/10.1111/cid.12270.](https://doi.org/10.1111/cid.12270)
- **11** Jansen, V.K., Conrads, G., and Richter, E.J. (1997). Microbial leakage and marginal fit of the implantabutment interface. *The International Journal of Oral & Maxillofacial Implants* 12 (4): 527–540.
- **12** King, G.N., Hermann, J.S., Schoolfield, J.D. et al. (2002). Influence of the size of the microgap on crestal bone levels in non-submerged dental implants: a radiographic study in the canine mandible. *Journal of Periodontology* 73 (10): 1111–1117. [https://doi.org/10.1902/jop.2002.](https://doi.org/10.1902/jop.2002.73.10.1111) [73.10.1111.](https://doi.org/10.1902/jop.2002.73.10.1111)
- **13** Harder, S., Dimaczek, B., Açil, Y. et al. (2010). Molecular leakage at implant-abutment connection – in vitro investigation of tightness of internal conical implantabutment connections against endotoxin penetration. *Clinical Oral Investigations* 14 (4): 427–432. [https://doi.](https://doi.org/10.1007/s00784-009-0317-x) [org/10.1007/s00784-009-0317-x.](https://doi.org/10.1007/s00784-009-0317-x)
- **14** Assenza, B., Tripodi, D., Scarano, A. et al. (2012). Bacterial leakage in implants with different implantabutment connections: an in vitro study. *Journal of Periodontology* 83 (4): 491–497. [https://doi.org/10.1902/](https://doi.org/10.1902/jop.2011.110320) [jop.2011.110320](https://doi.org/10.1902/jop.2011.110320).
- **15** Micarelli, C., Canullo, L., Baldissara, P., and Clementini, M. (2013). Implant abutment screw reverse torque values before and after plasma cleaning. *The International Journal of Prosthodontics* 26 (4): 331–333. [https://doi.](https://doi.org/10.11607/ijp.3396) [org/10.11607/ijp.3396](https://doi.org/10.11607/ijp.3396).
- **16** Aronsson, B.O., Lausmaa, J., and Kasemo, B. (1997). Glow discharge plasma treatment for surface cleaning and modification of metallic biomaterials. *Journal of Biomedical Materials Research* 35 (1): 49–73. [https://doi.](https://doi.org/10.1002/(sici)1097-4636(199704)35:1<49::aid-jbm6>3.0.co;2-m) [org/10.1002/\(sici\)1097-4636\(199704\)35:1<49::aid-jbm6>](https://doi.org/10.1002/(sici)1097-4636(199704)35:1<49::aid-jbm6>3.0.co;2-m) [3.0.co;2-m.](https://doi.org/10.1002/(sici)1097-4636(199704)35:1<49::aid-jbm6>3.0.co;2-m)
- **17** Chang, Y.C., Feng, S.W., Huang, H.M. et al. (2015). Surface analysis of titanium biological modification with glow discharge. *Clinical Implant Dentistry and Related Research* 17 (3): 469–475. [https://doi.org/10.1111/](https://doi.org/10.1111/cid.12141) [cid.12141.](https://doi.org/10.1111/cid.12141)
- **18** Coelho, P.G., Giro, G., Teixeira, H.S. et al. (2012). Argonbased atmospheric pressure plasma enhances early bone response to rough titanium surfaces. *Journal of Biomedical Materials Research. Part A* 100 (7): 1901–1906. [https://doi.org/10.1002/jbm.a.34127.](https://doi.org/10.1002/jbm.a.34127)
- **19** Canullo, L., Genova, T., Wang, H.L. et al. (2017). Plasma of argon increases cell attachment and bacterial decontamination on different implant surfaces. *The International Journal of Oral & Maxillofacial Implants* 32 (6): 1315–1323. [https://doi.org/10.11607/jomi.5777.](https://doi.org/10.11607/jomi.5777)
- **20** Canullo, L., Genova, T., Gross Trujillo, E. et al. (2020). Fibroblast interaction with different abutment surfaces: in vitro study. *International Journal of Molecular Sciences* 21 (6): [https://doi.org/10.3390/ijms21061919.](https://doi.org/10.3390/ijms21061919)
- **21** Canullo, L., Tallarico, M., Peñarrocha-Oltra, D. et al. (2016). Implant abutment cleaning by plasma of argon: 5-year follow-up of a randomized controlled trial. *Journal of Periodontology* 87 (4): 434–442. [https://doi.org/10.1902/](https://doi.org/10.1902/jop.2015.150549) [jop.2015.150549.](https://doi.org/10.1902/jop.2015.150549)
- **22** Canullo, L., Peñarrocha, D., Clementini, M. et al. (2015). Impact of plasma of argon cleaning treatment on implant abutments in patients with a history of periodontal disease and thin biotype: radiographic results at 24 month follow-up of a RCT. *Clinical Oral Implants Research* 26 (1): 8–14. [https://doi.org/10.1111/clr.12290.](https://doi.org/10.1111/clr.12290)
- **23** Carcuac, O. and Berglundh, T. (2014). Composition of human peri-implantitis and periodontitis lesions. *Journal of Dental Research* 93 (11): 1083–1088. [https://doi.](https://doi.org/10.1177/0022034514551754) [org/10.1177/0022034514551754.](https://doi.org/10.1177/0022034514551754)
- **24** Chambrone, L., Wang, H.L., and Romanos, G.E. (2018). Antimicrobial photodynamic therapy for the treatment of periodontitis and peri-implantitis: an American Academy of Periodontology best evidence review. *Journal of Periodontology* 89 (7): 783–803. [https://doi.org/10.1902/](https://doi.org/10.1902/jop.2017.170172) [jop.2017.170172.](https://doi.org/10.1902/jop.2017.170172)
- **25** Sgolastra, F., Petrucci, A., Severino, M. et al. (2015). Periodontitis, implant loss and peri-implantitis. A meta-analysis. *Clinical Oral Implants Research* 26 (4): e8–e16. [https://doi.org/10.1111/clr.12319.](https://doi.org/10.1111/clr.12319)
- **26** Sinjari, B., D'Addazio, G., De Tullio, I. et al. (2018). Peri-implant bone resorption during healing abutment placement: the effect of a 0.20% chlorhexidine gel vs. placebo-a randomized double blind controlled human study. *BioMed Research International* 2018: 5326340. [https://doi.org/10.1155/2018/5326340.](https://doi.org/10.1155/2018/5326340)
- **27** D'Ercole, S., D'Addazio, G., Di Lodovico, S. et al. (2020). Porphyromonas gingivalis load is balanced by 0.20% chlorhexidine gel. a randomized, double-blind, controlled, microbiological and immunohistochemical human study. *Journal of Clinical Medicine* 9 (1): [https://doi.org/10.3390/jcm9010284.](https://doi.org/10.3390/jcm9010284)
- **28** Binshabaib, M., Aabed, K., Alotaibi, F. et al. (2020). Antimicrobial efficacy of 0.8% hyaluronic acid and 0.2% chlorhexidine against porphyromonas gingivalis strains: an in-vitro study. *Pakistan Journal of Medical Sciences* 36 (2): 111–114. [https://doi.org/10.12669/pjms.36.2.1456.](https://doi.org/10.12669/pjms.36.2.1456)
- **29** Koutouzis, T., Gadalla, H., Kettler, Z. et al. (2015). The role of chlorhexidine on endotoxin penetration to the implant-abutment interface (IAI). *Clinical Implant Dentistry and Related Research* 17 (3): 476–482. [https://](https://doi.org/10.1111/cid.12158) [doi.org/10.1111/cid.12158.](https://doi.org/10.1111/cid.12158)
- **30** Paolantonio, M., Perinetti, G., D'Ercole, S. et al. (2008). Internal decontamination of dental implants: an in vivo randomized microbiologic 6-month trial on the effects of a chlorhexidine gel. *Journal of Periodontology* 79 (8): 1419–1425. [https://doi.org/10.1902/jop.2008.070660.](https://doi.org/10.1902/jop.2008.070660)
- **31** de Waal, Y.C., Raghoebar, G.M., Meijer, H.J. et al. (2015). Implant decontamination with 2% chlorhexidine during surgical peri-implantitis treatment: a randomized, doubleblind, controlled trial. *Clinical Oral Implants Research* 26 (9): 1015–1023. [https://doi.org/10.1111/clr.12419.](https://doi.org/10.1111/clr.12419)
- **32** Vergara-Llanos, D., Koning, T., Pavicic, M.F. et al. (2021). Antibacterial and cytotoxic evaluation of copper and zinc oxide nanoparticles as a potential disinfectant material of connections in implant provisional abutments: an in-vitro study. *Archives of Oral Biology* 122: 105031. [https://doi.](https://doi.org/10.1016/j.archoralbio.2020.105031) [org/10.1016/j.archoralbio.2020.105031.](https://doi.org/10.1016/j.archoralbio.2020.105031)
- **33** Isani, G., Falcioni, M.L., Barucca, G. et al. (2013). Comparative toxicity of CuO nanoparticles and CuSO4 in rainbow trout. *Ecotoxicology and Environmental Safety* 97: 40–46. [https://doi.org/10.1016/j.ecoenv.2013.07.001.](https://doi.org/10.1016/j.ecoenv.2013.07.001)
- **34** Salata, O. (2004). Applications of nanoparticles in biology and medicine. *Journal of Nanobiotechnology* 2 (1): 3. [https://doi.org/10.1186/1477-3155-2-3.](https://doi.org/10.1186/1477-3155-2-3)
- **35** Rudramurthy, G.R., Swamy, M.K., Sinniah, U.R., and Ghasemzadeh, A. (2016). Nanoparticles: alternatives against drug-resistant pathogenic microbes. *Molecules* 21 (7): [https://doi.org/10.3390/molecules21070836.](https://doi.org/10.3390/molecules21070836)
- **36** Lok, C.N., Ho, C.M., Chen, R. et al. (2007). Silver nanoparticles: partial oxidation and antibacterial activities. *Journal of Biological Inorganic Chemistry* 12 (4): 527–534. [https://doi.org/10.1007/s00775-007-0208-z.](https://doi.org/10.1007/s00775-007-0208-z)
- **37** Della Valle, C., Visai, L., Santin, M. et al. (2012). A novel antibacterial modification treatment of titanium capable to improve osseointegration. *The International Journal of Artificial Organs* 35 (10): 864–875. [https://doi.](https://doi.org/10.5301/ijao.5000161) [org/10.5301/ijao.5000161.](https://doi.org/10.5301/ijao.5000161)
- **38** Mohanan, P.V., Sangeetha, V., Sabareeswaran, A. et al. (2021). Safety of 0.5% hydrogen peroxide mist used in the disinfection gateway for COVID-19. *Environmental Science and Pollution Research International* 28 (47): 66602–66612[. https://doi.org/10.1007/s11356-021-15164-y.](https://doi.org/10.1007/s11356-021-15164-y)
- **39** McEvoy, B. and Rowan, N.J. (2019). Terminal sterilization of medical devices using vaporized hydrogen peroxide: a review of current methods and emerging opportunities. *Journal of Applied Microbiology* 127 (5): 1403–1420. [https://doi.org/10.1111/jam.14412.](https://doi.org/10.1111/jam.14412)
- **40** Ntrouka, V., Hoogenkamp, M., Zaura, E., and van der Weijden, F. (2011). The effect of chemotherapeutic agents on titanium-adherent biofilms. *Clinical Oral Implants Research* 22 (11): 1227–1234. [https://doi.org/10.1111/](https://doi.org/10.1111/j.1600-0501.2010.02085.x) [j.1600-0501.2010.02085.x.](https://doi.org/10.1111/j.1600-0501.2010.02085.x)
- **41** Jervøe-Storm, P.M., Hablützel, A.S., Bartels, P. et al. (2021). Comparison of irrigation protocols for the internal decontamination of dental implants-results of in vitro and in vivo studies. *Clinical Oral Implants Research* 32 (10): 1168–1175. [https://doi.org/10.1111/clr.13814.](https://doi.org/10.1111/clr.13814)
- **42** Claffey, N., Clarke, E., Polyzois, I., and Renvert, S. (2008). Surgical treatment of peri-implantitis. *Journal of Clinical Periodontology* 35 (8 Suppl): 316–332. [https://doi.org/](https://doi.org/10.1111/j.1600-051X.2008.01277.x) [10.1111/j.1600-051X.2008.01277.x.](https://doi.org/10.1111/j.1600-051X.2008.01277.x)
- **43** Boyce, J.M. (2018). Alcohols as surface disinfectants in healthcare settings. *Infection Control and Hospital Epidemiology* 39 (3): 323–328. [https://doi.org/10.1017/](https://doi.org/10.1017/ice.2017.301) [ice.2017.301](https://doi.org/10.1017/ice.2017.301).
- **44** Sivaranjani, M., Liu, F., and White, A.P. (2021). Synergistic activity of tetrasodium EDTA, ethanol and

chlorhexidine hydrochloride against planktonic and biofilm cells of clinically relevant pathogens. *Journal of Global Antimicrobial Resistance* 24: 148–157. [https://doi.](https://doi.org/10.1016/j.jgar.2020.12.002) [org/10.1016/j.jgar.2020.12.002.](https://doi.org/10.1016/j.jgar.2020.12.002)

- **45** Tsuchiya, H., Shirai, T., Nishida, H. et al. (2012). Innovative antimicrobial coating of titanium implants with iodine. *Journal of Orthopaedic Science* 17 (5): 595–604.<https://doi.org/10.1007/s00776-012-0247-3>.
- **46** Htet, M., Madi, M., Zakaria, O. et al. (2016). Decontamination of anodized implant surface with different modalities for peri-implantitis treatment: lasers and mechanical debridement with citric acid. *Journal of Periodontology* 87 (8): 953–961. [https://doi.org/10.1902/](https://doi.org/10.1902/jop.2016.150615) [jop.2016.150615](https://doi.org/10.1902/jop.2016.150615).
- **47** Meyle, J. (2012). Mechanical, chemical and laser treatments of the implant surface in the presence of marginal bone loss around implants. *European Journal of Oral Implantology* 5 (Suppl): S71–S81.
- **48** Castner, D.G. and Ratner, B.D. (2002). Biomedical surface science: foundations to frontiers. *Surface Science* 500 (1-3): 28–60.
- **49** Verdeguer, P., Gil, J., Punset, M. et al. (2022). Citric acid in the passivation of titanium dental implants: corrosion resistance and bactericide behavior. *Materials (Basel)* 15 (2): <https://doi.org/10.3390/ma15020545>.

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Home Care for the Implant Patient

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Introduction and Key Issues

The following statements schematically summarize the premises as well as represent a number of absolutely significant conclusions, therefore proposed as an introduction to the chapter.

- **Proper home care** for effective plaque removal is undeniably **crucial** for the **prevention**, **management,** and **control** of peri-implant infections, induced by bacterial biofilm [1–6].
- The mechanism of biofilm formation and the role of the peri-implant microbiome is a core component in **preventing** most biologic complications [6].
- Poor oral hygiene and lack of regular maintenance are strongly correlated with the development of both periimplant mucositis and peri-implantitis [7].
- Implants should be placed only in patients who can master **oral hygiene skills** required for disease prevention and already adhering to a recommended and tailored recall frequency (Figures 22.1–22.9).
- It is extremely valuable to emphasize that implant placement and **prosthetic reconstructions** need to allow **proper personal cleaning**, diagnosis by probing (Figures 22.3 and 22.7) and professional plaque removal [8].
- A dedicated dental team must **inform** the patient about the need for a constructive therapeutic alliance and **educates** patients to mechanically and efficiently remove biofilm (Figures 22.4, 22.6, 22.9) [9].
- Home care for the implant patient is still extensively neglected or **delegated**, but it is an indisputable cause of success [10] or failure even in case of implant treatment [11].
- It is valuable to properly coach the patient on how to use **dedicated home care tools** and more importantly in

adjunct to routine oral care procedures. Dental implants require **some additional tools** beyond routine oral hygiene care.

- The patient should be instructed to always **begin daily oral hygiene from implant–supported restorations**.
- The presence of an "adequate" amount of **peri-implant keratinized mucosa** is beneficial to improving proper daily oral care [3, 12].
- Various other factors can affect home care efficacy: tissue anatomy, periodontal status, or phenotype [3].
- At each follow-up recall appointment the clinician must update the diagnosis (Figures 22.3 and 22.7) and **continually remotivate** the patient after assessing home care performance [9].
- Consider **implant subjects always "high-risk" patients**.
- Peri-implant diseases are frequent and prevalence of peri-implantitis is increasing over time [8, 13]. As periimplant diseases could be effectively controlled by accurate prevention, **the importance** of giving proper oral hygiene instructions to the patients who are rehabilitated with dental implant and of fitting prosthetic constructions that allow accessibility for oral hygiene around implants [14–16] **is not stressed enough**.

Rationale and Relevance of Home Care

In implant dentistry, **plaque control** and **oral hygiene practices** are essential to limit the risk of complication and failure in the long term [3]. The identified etiological factor is certainly the biofilm [1].

Home care is the most important factor in impacting prevention, treatment, and prognosis of complications [11, 17].

However, effective Oral Hygiene remains a daily challenge.

Figure 22.1 (a–b). Full mouth X-ray before (a) and twenty years after (b) implant treatment in the posterior sectors.

Figure 22.2 Clinical case aspect with a 20-year follow-up.

A considerable body of evidence indicates that patients with implant–borne removable and fixed restorations require **lifelong professional recall regimens** to provide home care instructions, biological and mechanical maintenance, customized for each patient [18].

Experimental biofilm accumulation leads to a higher frequency of bleeding sites around implants as compared with teeth [19]. Consequently, in the case of a **positive bleeding index**, the clinician is responsible for providing **site-specific recommendations** so that, in addition to professional hygiene, proper home care can resolve plaqueinduced inflammation.

Neglected or poorly treated periodontal disease increases the threat for peri-implantitis and implant loss [20]. Prevalence of peri-implantitis is higher in noncompliant patients demonstrating poor home care adherence. Patients with a history of periodontitis presented lower survival rates and a statistically significantly higher number of sites with peri-implant bone loss [20]. A higher implant failure rate was found in periodontally compromised patients who

Figure 22.4 Electric toothbrush with oscillating-rotating technology (Oral B Procter and Gamble Company, USA). It is crucial to teach the patient proper brushing technique to ensure effective biofilm removal while preserving periimplant tissue integrity.

did not completely adhere to the recommended supportive periodontal therapy and showed improper home care [20].

Lack of regular supportive therapy in patients with periimplant mucositis is associated with increased risk for onset of peri-implantitis [8].

These important concepts should be highlighted: **the periodontal patient is at greater risk of complications than periodontally healthy patients** [9]. **Consequently, all implant patients should be considered at high risk**.

In contrast, stable clinical results could be achieved up to five years after initial therapy but **adequate oral cleanliness** across this period appeared to be an essential prerequisite [21, 22].

 (a) (b)

Figure 22.3 (a–b). The periodontal probe detects normal probing values in the absence of bleeding. The clinician should always update the diagnosis at each recall appointment by circumferential probing.

Figure 22.5 (a–b). Clinical use of a tritonal plaque disclosing solution (a) (Tri Plaque ID Gel, GC Corporation, Tokyo, Japan), to more effectively highlight the biofilm to be removed (b).

Figure 22.6 (a–b). Clinical use of a very useful and practical gauze soaked in 0.12% chlorhexidine (Digital Brush, Enacare, Micerium, Avegno, GE, Italy), wrapped around the finger of the dominant hand (a). The medicated gauze is initially placed on the keratinized gingiva or on the alveolar mucosa and then rolled in an apico-coronal direction, as indicated by the arrow (a). This procedure results in effective biofilm removal respecting the integrity of the peri-implant soft tissues. In addition, the use of the gauze is supplemental to the brushing (b).

Figure 22.8 (a–b). Plaque disclosing solution (Tri Plaque ID Gel, GC Corporation, Tokyo, Japan) is applied with a micro brush (a). Clinical appearance after rinsing with the air-water syringe (b). The clinician thus has the opportunity to motivate the patient to rigorously cleanse the residual biofilm (b).

Figure 22.9 (a–b). The interproximal toothbrush is the most appropriate tool for the removal of microbial biofilm from the interdental site. It is important to teach the patient to insert it correctly with an oblique movement in the apico-coronal direction (Interdental brush, Enacare, Micerium, Avegno, GE, Italy).

Therefore, home care reinforcement is so crucial to control reinfection and limit biologic complications, enhancing the long-term outcomes of implant therapy, particularly in patients with previous history of periodontitis [23].

The paramount importance of periodontal treatment with the goal of complete resolution of any periodontal inflammation before implant placement in partially edentulous patients has been highlighted throughout the dental literature [24, 25].

Prevention is the ideal treatment of peri-implantitis It is also advisable to begin supportive peri-implant therapy before positioning the implant. Patients must receive

detailed and, above all, personalized instructions on home care procedures based on their particular clinical situation, **including any hopeless teeth planned for extraction**, prior to implant placement [9]. Use of medicated gauze or a digital brush (Figure 22.6 a) is recommended for **hopeless teeth** [9, 11].

Periodontal pathogens can be transmitted from natural teeth to implant; therefore, **meticulous oral hygiene regimens of the entire oral cavity** are an indispensable prerequisite.

The **soft-tissue conditions around the implant**, the width of keratinised mucosa, and the phenotype and thickness of the mucosa are also considered major risk

indicators, as the presence of any mucosal defect around an implant can increase plaque accumulation and result in tissue inflammation [3].

As periodontists became concerned about cleanability and ease of hygiene with mucosal margins around implants, further revisions in techniques focused on the **importance of the preservation of attached gingiva** for maintenance of the implants [26].

Most clinicians agree that an "adequate" amount of attached gingiva facilitates proper daily oral care [3, 12].

The prosthetic suprastructure design and the possibility for the patient to easily access each implant for plaque removal is crucial [3]. The coronal portion of the implant, which is exposed to the bacterial challenge of the mouth, should be smooth enough not to foster plaque accumulation [26].

How to Motivate and Inform Implant Patients on Effective Home Care

Implants patient education must be the **combined team efforts** of the dental office staff and patients themselves for long-term success of dental implantology.

The clinician must ascertain the patient's awareness and understanding that effective oral hygiene is absolutely crucial. Poor oral hygiene resulting in plaque accumulation leads to peri-implant disease [27].

The patient should also be informed that peri-implant tissues respond to plaque accumulation in a way similar to that of periodontal tissues and that disease may develop in the tissues around the implants jeopardizing their longevity [27].

Implant–supported prosthetic restorations are more vulnerable to plaque-induced inflammation. Such concept is well known by all clinicians; however, the question is: do we do enough [9]?

In other words, do we as patient educators, do as much patient training in oral hygiene as necessary? Special awareness should be devoted to exemplifying tactics for the patient's approach to achieve appropriate plaque control, reinforcing desirable behaviors and producing results in a short time.

Always strengthen motivation at every possible opportunity, asking commitment to change, for autonomy and not with authority, and especially boost self-efficacy.

It is important to make the patient aware that dental health care habits, such as brushing and interdental cleaning, are crucial for long-term maintenance.

Patients should be guided in the selection of instruments best suited to their individual needs and trained in their use so to perform proper daily oral hygiene care [9].

During all subsequent sessions, it is essential to assess oral hygiene status and check if the cleaning procedures applied are actually appropriate, offering additional home care reinforcement or changing tools and techniques as needed [9].

A dedicated clinician educates patients to mechanically remove biofilm. Invest time in demonstrating how to brush teeth/implant–supported restorations properly either with an electric or manual tooth brush.

Dental office staff devote time to teaching interproximal hygiene techniques with the proper instrument for the existing interdental dimension.

Emphasize to start brushing from posterior areas, frequently hosting implant–supported restorations (Figure 22.10).

People usually brush their teeth with special attention to the anterior areas, both as a matter of habit and because of cosmetic considerations. Consequently, the posterior teeth and particularly the palatal and lingual surfaces are brushed hastily [9].

Suggest specific and dedicated implant care, i.e. the patient **should use a supplementary tool,** and/or add some extra antimicrobial/antiseptic substance, or specific toothpaste for implants beyond the appropriate routine for natural teeth.

There is consensus that **prevention** is more necessary than cure.

Figure 22.10 Implants are frequently inserted in the posterior sectors, especially in periodontal patients who have replaced completely compromised teeth. The clinician should always recommend starting the brushing maneuver from the areas where the implants are placed, and particularly from the palatal and/or lingual aspect, as indicated in the drawing. In the initial stages of brushing, the patient should be more careful and meticulous for effective biofilm removal precisely at those sites most vulnerable to plaque-induced inflammation.

Even in the case of implant treatment an adequate prevention could avoid plaque-induced peri-implant diseases. Nevertheless, peri-implant inflammatory complications are increasing, so the strategies that should modify this trend, according to the author, would be: convince the patient to use additional tools, always giving priority to the cleansing of areas where implant reconstructions are located.

Advise patients that mouthwash rinsing is **ineffective** for adhesive microbial plaque.

When we began to successfully place dental implants, the entire surgical team, adopted a strict protocol, provided by Branemark, including an intra-oral pre-operative chlorhexidine "scrub."

The take home message is to educate the population to a mechanical removal of biofilm.

During the infancy years of dental implantology, the emphasis for long-term success of osseointegrated implants was the surgical phase [28]. In the years that followed, the significance for long-term maintenance had switched to focusing more on the proper fixture placement, dictated by the prosthetic and aesthetic needs of each specific case. In more recent years, **professional implant maintenance** [18] and **meticulous patient home care** [17] has been recognized as two critical factors for the long-term success of dental implants.

Bleeding on probing is considered as key clinical measure to distinguish between peri-implant health and disease [8].

Consequently, wherever bleeding on probing is detected, the entire dental team must be involved in scheduling a suitable time to remotivate the patient in order to effectively remove the biofilm and resolve plaque-induced inflammation.

Dental assistants and dental hygienists have a significant role in the education of implant patients so that meticulous oral self-care can be accomplished. However, **it is important that dentists do not give the impression that they are delegating the motivation phase only to auxiliary personnel**. On the other hand, the dentist should emphasize the importance of giving first-hand instructions and participating in the practical sessions of oral hygiene instruction.

The clinician should NOT GIVE THE FEELING to DELEGATE OHI.

Current evidence also demonstrates that the use of specific oral topical agents and oral hygiene aids can improve professional and at-home maintenance of implant–borne restorations.

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professional and at-home maintenance of implant–borne restorations [18].

How Much Time Should be Spent on Motivation?

It is recommended that the 1st appointment of etiological therapy of all implant patient should last 2 hours, so at least 20/30 minutes can be devoted to motivation and very thorough home oral hygiene instructions.

Approximately 15 minutes at subsequent appointments should be scheduled to remotivating the patient and assessing his/her home care performance efficacy, based on instructions given at the previous appointment.

During periodontal support therapy, motivation time ranges from 5 to 15 minutes, depending on the level of patient compliance.

These times are approximate; all patients deserve their own personalized reinstruction sessions when indicated. It is always important to set aside enough time to explain the home care techniques: **the therapist needs to make certain that the patient has mastered the oral hygiene skills.**

Most importantly, it is imperative that the **oral hygiene instructions be practical**, i.e. the clinician must demonstrate the proper use of the hygiene instruments in the patient's mouth, and then supervise home care procedures to ensure the patient is able to implement all the techniques correctly.

The clinician must not assume that once the instructions are given, the patient will remember them in all their details, for long time.

Continuous Patient Remotivation is a Never-Ending Job.

Audiovisual educational materials are recommended as a supplement to home oral hygiene explanations.

The author has released a DVD in Italian addressed to the patient, with the catchy title: "If I say implant, what do you think?," focused mainly on the motivation of the patient, as well as providing useful practical information especially in the short and long term post-surgical follow-up [29].

Essential Home Care Tools and Technique

A plethora of home care tools have become commercially available.

To keep high compliance, clinician should avoid overwhelming the patient with several devices or complicated protocols, most of which, other than brushing, are dropped following a short trial period [9].

Therefore, only the essential instruments should be selected, fully describing the technique to effectively using them.

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It is crucial to recommend to the implant patient the following [9]:

- Effective brushing technique (with manual or electric toothbrush).
- An interproximal cleaning aid (definitely an interdental brush). Oral hygiene should always include an interproximal device, which could be either an interdental brush or dental floss (Figures 22.19–22.22), depending on interproximal space viability and width.
- An additional home care device. (e.g. a Digital Brush, Enacare, Micerium) [9] The patient should always use some extra instrument, specific to the area where the implants have been placed, in addition to the oral hygiene routine for the rest of his or her mouth.

Home care effectiveness is mainly determined by the dexterity of the patient. Therefore, the technique used is more important than the instrument selected, which must be appropriate for the peri-implant site.

In terms of patient-centered care, the presence of a periimplant keratinized tissue is beneficial to easing proper oral hygiene. The keratinized mucosal collar offers greater resistance to possible trauma resulting from brushing maneuvers and the use of auxiliary tools such as interdental brushes, tufts, and special flosses [12].

This is crucial especially for **less motivated patients**, who could **justify** their **oral hygiene negligence** if they are experiencing localized **discomfort** or soft tissue lesions following self-injury due to inappropriate tool use. This does not mean that oral hygiene maneuvers cannot be carried out effectively if the peri-implant keratinized mucosa is minimal or even absent, but they are undoubtedly more difficult to perform properly [12]. (Figures 22.10–22.35) However, the clinician must commit even more to providing very specific and tailored instructions.

Important and Useful Clinical Recommendation

The clinician should always suggest to the patient to follow a precise sequence of use of the various devices, preferably starting from the posterior areas, and from the internal surfaces, to avoid any omission [12], since it is easier to clean the anterior areas while neglecting the posterior teeth.

Figure 22.11 (a–e) The periodontal probe detects 2mm probing depth on the mesial aspect (a) and 3mm on the distal aspect (b), both in the absence of bleeding (a–b), of the implant replacing the left maxillary lateral incisor. The peri-implant tissue is in good health, although keratinized gingiva is absent in this clinical case at 19-year follow-up (a–b). The patient's oral hygiene is satisfactory. In addition to tooth- and interdental-brushing, the patient uses a special implant toothpaste (Implaclean®, Dyna Dental, Netherlands), which can also be applied by the clinician with a micro brush ($c-e$) or at home by the patient with a single tufted toothbrush. It is important to train the patient to use a specific tool and especially additional to routine oral care methods.

Figure 22.12 (a–b). Educational drawings illustrating the Roll Brushing technique. The bristles are initially placed parallel to the vertical axis of the implant–supported prosthetic restoration and in the apical direction (a). Subsequently, the brush is rotated coronally in the arrow direction (b).

Figure 22.13 (a–b). Sulcular type of brushing. An adequate amount of keratinized tissue is present in the clinical photograph (a). The presence of a stable mucosal seal can endure a Bass technique. The patient uses a sulcular toothbrush, with only two bristle rows, gently inserting it into the peri-implant sulcus (b), according to the modified Bass technique.

Figure 22.14 (a–b). Clinical aspect of a keratinized mucosa present in ideal quantity and quality (a). The periodontal probe is applied at the alveolar mucosa and kept perpendicular to the vertical axis of the implants, exposing the generous amount of attached gingiva (a). The manual brush is applied initially with the bristles parallel to the vertical axis of the implants (b), according to the Roll technique, followed by a rotational movement in the coronal direction.

Figure 22.15 (a–b). The same patient and the same brushing technique as in the previous figures (Figure 22.14), but using a smaller toothbrush, which better, more easily reaches the most posterior areas (a–b). Children's toothbrushes are often ideal for facilitating hygiene maneuvers especially in the inner and/or rearmost areas. According to the Roll technique, the bristles are initially positioned parallel to the vertical axis of the implant–supported prosthetic restoration, well distanced from the gingival margin, on the attached gingiva (a), and then are rotated in the coronal direction (b).

Figure 22.16 The clinician should always recommend that the patient begin brushing from the more posterior, often neglected, inner areas of the oral cavity, where implant–supported prosthetic restorations are frequently located.

nondominant hand to facilitate toothbrush correct placement,

especially if it is of normal size and/or voluminous, into the most posterior areas of the buccal aspect.

Figure 22.17 The image illustrates how the clinician recommends that the patient retract the cheek with the

It is also crucial that the clinician take every opportunity to remotivate the patient: for example, even during the polishing phase. The clinician might draw the patient's attention to the fact that he/she has started this procedure from the posterior areas and not from the esthetic, anterior area, which will nevertheless be addressed later.

Toothbrush and Brushing Technique

Peri-implant phenotype assessment should guide the clinician in recommending the most appropriate instrument for routine brushing [9].

Powered toothbrushes were found to result in an improvement in clinical parameters over time [17, 30].

It is equally significant not to cause tissue trauma to the peri-implant mucosa while brushing.

To date, no one method of brushing has been recognized as undoubtedly superior to the others [31].

Cleaning efficacy has an impact on preferring one technique over another. Effective biofilm removal is the most critical factor when assessing brushing technique, thoroughness is more important than method [9].

(b)

 $\qquad \qquad \text{(c)}$

Figure 22.18 (a–e). Initial, baseline radiographic status (a), full mouth X-ray (b), and current clinical condition (c) at 24-year follow-up (b–c). The patient shows adequate plaque control. The clinician should always monitor the effectiveness of biofilm removal from both implant–supported prosthetic restorations (d) and from natural teeth (e). The interdental brush should be consistently introduced in an apico-coronal direction (d–e), explaining to the patient that the interproximal device should be inserted from top to bottom for the maxillary arch (d) and from bottom to top for the mandibular arch (e).

(e)

Figure 22.18 (Continued)

In case the patient prefers a manual toothbrush, in the presence of implant–supported prosthetic reconstructions, the author recommends the **Roll method** (Figure 22.15), combined with a soft toothbrush, as it is not traumatic and effective [9].

The Roll technique seems to be appropriate to effectively eliminate plaque accumulation without causing soft tissue distress. It is particularly recommended for thin periodontal phenotypes or when the implants are surrounded by alveolar mucosa and not by keratinized mucosa (Figure 22.13).

In case the Modified Bass sulcular brushing technique is preferred by the clinician, the bristles are introduced just inside the peri-implant sulcus at 45° to the vertical axes of the implant (Figure 22.13a, b). Slight oscillation and vibration are then applied on the area to disrupt the bacterial colonies that have become established within the sulcus.

Figure 22.19 The didactic drawing illustrates the correct insertion of the interdental brush, always at 45° to the vertical axis of the implant supported prosthesis, and also always in the apico-coronal direction, therefore from the bottom to the top for the mandibular arch and from the top to the bottom for the maxillary one.

Single bristle layered brushes are also available to better accommodate for sulcular adaption (Figure 22.13b).

In case of an implant–supported prosthesis that is surrounded by a generous quantity of well-keratinized adherent gingiva (Figure 22.14), the roll brushing technique is, however, also suitable, as it is adequate in the case of a thick periodontal phynotype (Figure 22.15) [9, 32].

 (a) (b)

Figure 22.20 Clinical images showing the interdental brush insertion (a) complying with the correct angulation in the apico-coronal direction (a). Two different sizes of interdental brushes fit the two different interproximal spaces (b). Verifying the proper space adaptation is important, especially at the stage of provisional prosthetic restoration.

Figure 22.21 (a–c). The interdental brush (Enacare interdental brush, Micerium Ge, Italy) can be soaked in various antimicrobial substances, such as 10-volume 3% hydrogen peroxide, into a small plastic cup (a). Alternatively, it can be dipped in prophylactic paste at the end of the professional hygiene session to demonstrate its use (b). The patient at home can associate the use of an implantspecific toothpaste (Implaclean®, Dyna Dental, Netherlands) (c). The goal is to lubricate the bristles to make insertion of the interdental brush more easily (b–c).

Supragingival plaque is the first deposit to form; its prevention and control are critical to the prevention of subgingival plaque formation [9]. If the patient is able to remove most of supragingival plaque, very modest subgingival deposits will develop [9, 33].

Recommend starting the brushing routine in the inner and more posterior surfaces of the mouth. Typically, people brush their teeth paying particular attention to the anterior areas, both as a matter of habit and aesthetic demand. However, the surfaces with the greatest accumulation of soft, calcified deposits are the lingual areas of mandibular molars and bicuspids [9], which are often neglected or reached hastily, possibly "fooled" by a feeling of freshness, by the use of toothpaste. Therefore, advising the patient to start the cleansing of the oral cavity, starting from the most posterior areas, and from the

lingual and palatal surfaces (Figures 22.10 and 22.17), can be a practical and valid suggestion to improve plaque control.

Tongue Brushing

Recommend that patients never forget to brush their tongue.

To thoroughly cleanse the oral cavity, the tongue must also be brushed [9, 34].

The toothbrush, either electric or manual, should be placed at a right angle to the tongue, initially orienting the bristles toward the throat. The instrument is then rotated to the tongue tip, exerting a gentle pressure. This movement should be repeated at least four to five times until the instrument feels effectively removes the deposit coating the dorsal surface of the tongue [9]. Microorganisms can

Figure 22.22 (a–c). Medicated wipes soaked in chlorhexidine at 0.12% (Digital Brush, Enacare, Micerium, Avegno, GE, Italy) (a) are commercially available (the author co-patented such digital brush). These devices are indicated for the biofilm removal from teeth, prosthesis and mucous membranes and are particularly suitable for peri-implant tissue scrubbing. In particular, the tissues allow an effective and gentle cleansing, even outdoors. As an alternative to the product on the market (a), a simple gauze can be wrapped around the index finger of the dominant hand (b), and then soaked in a disinfectant substance, chlorhexidine-based or other antimicrobial and then passed with a roller movement from the mucous membranes to the teeth/restorations, as indicated by the blue arrow in the drawing (c), both by the patient or by the clinician.

accumulate in the tongue, especially in the presence of deep fissures and grooves, receptacle for the bacteria responsible for halitosis [34].

Especially the posterior part of the tongue, as less oxygenated, also not as affected by self-cleaning very easily develop a patina and even bad breath.

Interdental Brush

The interproximal brush is unquestionably the best-performing instrument for interproximal cleaning [35], also in case of implant-borne fixed restorations [9].

Interdental brushes are to be considered always preferable to floss for interdental plaque removal [36].

In the case of implant–supported prosthetic restorations, it should always be possible to use an interproximal brush in both the posterior and anterior sites [12].

The use of interdental brushes is essential for biofilm removal in patients during initial periodontal and followup therapy [37].

The **interdental space should have a 2-mm minimum interproximal dimension to allow easy insertion of the interdental brush** in an oblique direction, from bottom to top, at mandibular sites and from top to bottom maxillary.

Recommending interdental brushes, clinician has to select a wide enough device, to fill the viable space, adequately flexible to pass through to the other side (Figure 22.21c).

Figure 22.23 The didactic drawing illustrates the use of a gauze, drawing a letter "C," wrapped around the distal surfaces of the most posterior element in the dental arch, for effective mechanical biofilm removal.

Digital Brush

Medicated Gauze, Disposable Towel The use of a medicated gauze or disposable towel/digital brush soaked in 0.12% chlorhexidine is recommended [9].

Medicated wipes or gauze, i.e. soaked in chlorhexidine at 0.12%, are commercially available and very useful and practical (Figures 22.22–22.25) (Digital Brush, Enacare, Micerium, Avegno, GE), in convenient disposable, pocket-sized packages, as an alternative to many other products on the market (the author co-patented such digital brush). In this period in which chlorhexidine is one of the substances indicated for an effective disinfection against Corona virus [38, 39], its use could be recommended.

Such device can be wrapped around the second (index) finger of the dominant hand and used to clean the periimplant mucosa, teeth, and implant supported prosthesis with rolling movements in an apico-coronal direction (Figure 22.22).

Alternatively, or better in addition, the use of a gauze, always wrapped around the index finger of the dominant hand (Figure 22.22), and soaked in an antimicrobial substance poured into a plastic cup (Figure 22.22), is proposed to remove soft deposits from tooth surfaces, with rotational movements from the gingiva to the tooth crown (Figure 22.22), resulting in a mechanical removal of bacteria in combination with chemical decontamination [11, 40–42].

This procedure is simple and highly effective for removing plaque and can supplement the use of a toothbrush, potentially improving its effectiveness [11, 40–42].

When the patient is holding the toothbrush handle, he or she is not able to check whether the brush is effectively reaching all the surfaces of the teeth. Conversely, the fingertips contain tactile receptors that make it possible to systematically control the cleaning action when it is performed with a gauze pad wrapped around the finger. After taking out and unfolding a gauze pad or a medicated disposable towel [9], the individual places the forefinger of his or her dominant hand in the center of the pad before wrapping it completely around the finger (Figure 22.22). The gauze pad is then used all over the oral cavity. The correct movement is vertical (Figure 22.22), from the peri-implant soft tissue to the prosthetic crown, and one implant/tooth is cleaned at a time.

Figure 22.24 In case of very wide interdental space, a gauze fragment is recommended, drawing a letter "C," wrapped to encircle both the mesial (a) and distal (b) surfaces of adjacent teeth.

Figure 22.25 (a–e). The periodontal probe detects normal readings (a–b). The patient reports cutting out tissue fragments (c–d) and applying them to the buccal aspect, like a patch, for local decontamination action (Digital Brush, Enacare, Micerium, Avegno, GE, Italy).

Use of a gauze pad is recommended immediately post implant placement and in the case of serious localized sensitivity or when the toothbrush bristles can cause an unpleasant sensation with a consequent avoidance reaction. In such cases, the patient tends to skip the sensitive area, which thus leads to greater plaque buildup, can trigger a vicious cycle and worsen the pain symptoms. Conversely, use of the gauze pad, or a medicated disposable towel, is never traumatic. Other indications include all clinical conditions where the use of a conventional toothbrush can be difficult, troublesome, or not fully effective, for example, in patients who have had oral, periodontal, or

Figure 22.26 (a–b) Clinical use of a plaque disclosing agent applied with a micro brush (a). It is necessary to rinse, using the dental unit syringe (b) to remove excess product, and then assess for soft deposits present.

Figure 22.27 (a–c) The plaque disclosing solution (Tri Plaque ID Gel, GC) is applied with a micro brush (a), water is sprayed with the dental unit syringe (b) to highlight to the patient the areas neglected during homecare hygiene, giving further suggestions for a more effective brushing, recommending, for example, to move away the cheek using the non-dominant hand, to make it easier for the toothbrush to reach the most posterior areas in the vestibular aspect (c).

Figure 22.28 The single tufted toothbrush is certainly an appropriate tool for implant–supported prosthetic restorations. Thanks to the small size of this instrument, bristles insertion between the prosthesis and the peri-implant tissue is particularly easy, even in case of poor dexterity. The bristles can also be dipped in an antimicrobial substance for an additional decontamination effect.

implant surgery [43, 44]; who have difficultly opened their mouth; who have poor dexterity [5] or who are bedridden or have a disability [45]. The use of gauze, following the specified instructions, is never traumatic.

The gauze pad or a medicated disposable towel, wrapped around the crown of the tooth as shown in Figure 22.23, is also useful to clean surfaces adjacent to edentulous areas. A rubbing movement is performed, in a shoeshining motion.

It is useful for operators to suggest to patients the use of medicated or ordinary gauze, which can easily be wrapped around fixed implant–supported restorations (Figure 22.24). This manual method is effective and can be performed to clean the microbial plaque off the exposed threads of an implant, for cleansing areas subject to inflammation or following surgery [26, 44].

The presence of chlorhexidine does not appear to cause pigmentation of the tooth/prosthetic surfaces, like

(f) (g) (c) (h)

Figure 22.29 (a–h) Clinical use of an implant-specific toothbrush (a–c), recently introduced to the US market and protected by patent. The bristles are positioned on either side of the handle, angled (d), to facilitate cleansing while simultaneously retracting the cheek (a), to better reach the most posterior sites (a–c). The periodontal probe detects peri-implant tissue health (e, g) and adequate bone support (f). The patient also uses the interdental toothbrush (h) necessary to clean the interproximal spaces, but the dedicated implant toothbrush is however and certainly a particularly effective additional tool (a–d), to be recommended especially in the presence of implant–supported prosthetic restorations in the posterior areas.

 (e) (f)

Figure 22.30 (a–f) The Single-tufted brush (TePe Implant Care™, Malmö Sweden) is particularly effective for cleaning biofilm under the prosthetic flange (a, e), and all surfaces in the posterior and palatal region (b–c) and all prosthetic structures that are quite articulated and not easy to clean (a–f). The single-brush toothbrush can be used in combination with antimicrobial substances (d–e) for a better decontamination action of the implant supported prosthesis.

Figure 22.31 Didactic drawing illustrating the correct technique for the use of dental floss. It is important to draw a letter "C" with the index fingers of both hands, kept in direct contact with the surfaces to be cleaned, trying to embrace the element as circumferentially as possible, for a more effective cleansing of the interproximal surfaces.

chlorhexidine-based antimicrobial agents in the form of mouthwashes or gel, because the substance is present in reduced concentrations, is combined with discoloration prevention systems, and mainly remains on the gauze pad.

Disclosing Solution

The use of a liquid plaque-disclosing solution (Figures 22.26–22.27) is recommended for patient motivation, identifying any areas neglected during home hygiene techniques [12].

Furthermore the biofilm can be identified **to perform site-specific and effective instrumentation** that is never excessive, especially in the presence of healthy periimplant tissues, in the immediate post-treatment phases, or even after the provisional and/or definitive prosthetic restorations have been placed [12].

The plaque-disclosing solution can also be used at the end of nonsurgical periodontal instrumentation **to evaluate site-specific instrumental performance**, indicating any areas where additional treatment is required to complete an effective, comprehensive decontamination phase [12].

In particular, when a tri-colored substance (e.g. Tri Plaque ID Gel, GC) is used, acidophilic plaque takes on a bright turquoise color (Figure 22.27b).

Tufts

Single-tufted Brushes A single-tufted toothbrush is particularly indicated for cleaning all surfaces that the patient is unable to reach effectively, such as prosthetic restorations supported by osseointegrated implants [9]. (Figures 22.28–22.30). It is a useful tool for cleansing relatively complex and bulky implant–supported prosthetic superstructures (Figures 22.28–22.30). It is particularly indicated also in the case of the gag reflex, preventable because of the small size of this tool [9].

Floss

The ideal instrument for cleansing the interproximal spaces remains the interdental brush [9, 35]. The dental floss (Figures 22.31–22.41), therefore, is a certainly less indicated tool in case of prosthetic restorations supported

Figure 22.32 (a–b). Clinical images illustrating the correct use of dental floss. It is important to wrap the implant–supported prosthetic restoration as circumferentially as possible by drawing a letter "C" on both the mesial aspect (a) and distal aspect (b) of the interproximal space of two adjacent elements.

Figure 22.33 (a–b). A floss cut (a) is evident in the clinical images, resulting from a traumatic use of this instrument. The interdental brush (b) is always to be preferred and recommended.

Figure 22.34 The clinical image shows the rigid tip of the special implant wire.

Figure 22.36 The figure illustrates the use of a special implant floss for prosthetic restorations with bridge elements. The floss should be guided to bypass the bridge element by cleansing the microbial biofilm from the mucosal-facing surface.

Figure 22.35 The clinical image illustrates another correct technique of implant special floss use. By crossing the thread on the buccal aspect, a circumferential contact is made with the implant surface below the prosthetic margin.

Figure 22.37 Special implant floss is required to clean the surface of the bridge pontic facing the mucosal tissue, but always in conjunction with an interdental brush to clean the interproximal spaces.

(e)

Figure 22.38 (a–e). Clinical use of the special implant floss (a) to bypass the pontic in mesial position. Follow-up periapical radiograph (b). The use of interdental brushes is always necessary for interproximal space cleansing (c), in order to maintain a clinical stability of the case, assessed with an update of the diagnosis by circumferential probing at each recall appointment (d–e). The periodontal probe detects 3mm probing readings in the absence of bleeding.

Figure 22.39 Didactic drawing illustrating how the special implant floss cleans the surface of the prosthetic restoration facing the soft tissue.

by implants, both for its lower cleansing efficiency and especially for its greater difficulty in the appropriate technique of use. The patient's manual dexterity is generally poor even in the presence of natural teeth, and therefore finds major obstacles in the presence of implant supported prostheses. The technique for the efficient use of the dental floss is illustrated in the didactic drawing, where the fingers of both hands are placed very close to each other and in direct contact with the surfaces to be scrubbed, drawing a letter "C" (Figures 22.31 and 22.32).

The floss should clean circumferentially in a shoeshining motion (Figure 22.32a, b). But not all patients will be able to perform this action. Many patients may have dexterity issues, creating an overexaggerated C-shape is the next best option.

Special Floss

Floss specifically designed for implants and bridges is also available.

Figure 22.40 (a–d). Flossing is indicated to remove biofilm in the presence of prosthetic restorations with pontics (a–b). It is recommended to use a special spongy floss, (a–b) which is inserted with a stiffer termination in the interproximal space (a) and then guided to pass under the pontic (b) to effectively remove the biofilm at that site. However, it is recommended that interproximal brushes (c–d) also be used in conjunction with dental floss, as they remain absolutely necessary in the home care routine. This particular type of interdental brush has bristles of different sizes, so that the larger ones, as they flex, face less resistance from the shorter bristles (Interdental Brush, Enacare, Micerium, Avegno (Ge) Italy) (d) (The author co-patented such anatomical interdental brushes).

 $\qquad \qquad \textbf{(b)} \qquad \qquad \textbf{(c)}$

Figure 22.41 (a–c). At each recall appointment, the clinician has a responsibility to re-evaluate the patient's home hygiene performance: the titanium-coated stainless-steel curette (Universal Curet, ENACARE, Micerium, Avegno, Ge, Italy) detects abundant biofilm deposits, especially at the pontic, a particularly difficult area to successfully clean (a). The radiographic (b) and clinical (c) evaluations, however, are satisfactory.

A special implant floss with a spongy portion is indicated in the presence of pontic elements to clean the area facing the soft tissue. However, the patient must have enough dexterity to use such an instrument effectively and especially safely, with no soft tissue injury (Figure 22.33).

In case of prosthetic reconstruction with pontics, it is necessary to use a special implant floss featuring a stiffer end that facilitates its insertion (Figure 22.34).

Recommend an extra hygiene tip in case of implant patient. Suggest to all implant patients to pay specific attention to that area of their mouth, which is more vulnerable to inflammatory, plaque-induced diseases, especially in case of previous history of periodontal disease and in the presence of other risk factors [46]. **All implant patients are, however, associated with a high risk**. It is also important to recommend dedicating additional time to the area where there are implants.

The patient is commended to use a specific instrument, which could be the mono tufted toothbrush (Figures 22.28–22.30) or the 0.12% medicated gauze (Figures 22.22–22.25) or an implant-specific toothpaste or other antimicrobial substance at the clinician's discretion, applied with a micro brush or cotton fioc, in addition to the daily oral hygiene routine for the rest of the oral cavity [12].

Continuous patient remotivation is the single most significant factor in determining and maintaining the long-term success following effective etiologic therapy. Our professional concern must be focused on promoting the most appropriate home hygiene behavior possible, hoping that these simple suggestions will inspire others so that our patients will be able to perform consistent and effective "smart home care."

The benefits of plaque reduction around teeth and implants **can improve oral and overall systemic health**. We can no longer accept a one-tool-for-all approach; at-home maintenance routines need to be customized.

References

- **1** Berglundh, T., Armitage, G., Araujo, M.G. et al. (2018). 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* 89 (Suppl 1): S313–S318.
- **2** Costa, F.O., Takenaka-Martinez, S., Cota, L.O. et al. (2012). Peri-implant disease in subjects with and without preventive maintenance: a 5-year follow-up. *Journal of Clinical Periodontology* 39 (2): 173–81.
- **3** Giovannoli, J.L., Roccuzzo, M., Albouy, J.P. et al. (2019). Local risk indicators – consensus report of working group 2. *International Dental Journal* 69 (Suppl 2): 7–11.
- **4** Renvert, S., Persson, G.R., Pirih, F.Q., and Camargo, P.M. (2018). Peri-implant health, peri-implant mucositis, and peri-implantitis: case definitions and diagnostic considerations. *Journal of Clinical Periodontology* 45 (Suppl 20): S278–S285.
- **5** Roncati M. Periodontal Maintenance in G. Ricci, Periodontal Diagnosis and Therapy, Quintessence Pubishing, Chicago, U.S.A., 2013, pp 725–738.
- **6** Romanos, G.E., Delgado-Ruiz ,R., Sculean, A. (2019). Concepts for prevention of complications in implant therapy. *Periodontol* 2000 81 (1): 7–17.
- **7** Wada, M., Mameno, T., Otsuki, M. et al. (2021). Prevalence and risk indicators for peri-implant diseases: a literature review. *Japanese Dental Science Review* 57: 78–84.
- **8** Jepsen, S., Berglundh, T., Genco, R. et al. (2015). Primary prevention of peri-implantitis: managing peri-implant mucositis. *Journal of Clinical Periodontology* 42 (Suppl 16): S152–S157.
- **9** Roncati, M. (2017). *nonsurgical periodontal therapy indications, limits, and clinical protocols with the adjunctive use of a diode laser*. Milano, Italy: Quintessence Pubishing.
- **10** Preus, H.R., Al-Lami, Q., Baelum, V., and Oral hygiene revisited. (2020). The clinical effect of a prolonged oral hygiene phase prior to periodontal therapy in periodontitis patients. A randomized clinical study. *Journal of Clinical Periodontology* 47 (1): 36–42.
- **11** Roncati, M. (2021). Impact of home and professional care on laser-assisted management of peri-implant complications: a case report. *International Journal of Periodontics and Restorative Dentistry* 41 (6): 819–825.

Promote Healthy Lifestyle

Clearly that future is bright, as periodontists embrace the responsibility of **placing and maintaining dental implants**.

- **12** Roncati, M. (2019). The importance of dedicated maintenance therapy for long-term success. In: *Implant Therapy: Clinical Approaches and Evidence of Success*, 2e, vol. 27 (ed. M. Nevins and H. Wang), 475–489. Chicago: Quintessence Pubishing.
- **13** Lee, C.T., Huang, Y.W., Zhu, L., and Weltman, R. (2017). Prevalences of peri-implantitis and peri-implant mucositis: systematic review and meta-analysis. *Journal of Dentistry* 62: 1–12.
- **14** Renvert, S., Aghazadeh, A., Hallström, H., and Persson, G.R. (2014). Factors related to peri-implantitis – a retrospective study. *Clinical Oral Implants Research* 25 (4): 522–529.
- **15** Serino, G. and Ström, C. (2009). Peri-implantitis in partially edentulous patients: association with inadequate plaque control. *Clinical Oral Implants Research* 20 (2): 169–174.
- **16** Saltmarsh, H. and Healy, S.R. (2017). Guideline on recall and maintenance for patients with restorations based mostly on expert opinion. *International Journal of Evidence-Based Practice for the Dental Hygienist* 3 (1): 25–30.
- **17** Louropoulou, A., Slot, D.E., and Van der Weijden, F. (2014). Mechanical self-performed oral hygiene of implant supported restorations: a systematic review. *The Journal of Evidence-Based Dental Practice* 14 (Suppl): 60–9.e1.
- **18** Bidra, A.S., Daubert, D.M., Garcia, L.T. et al. (2016). A systematic review of recall regimen and maintenance regimen of patients with dental restorations. part 2: implant-borne restorations. *Journal of Prosthodontics* 25 (Suppl 1): S16–S31.
- **19** Salvi, G.E., Cosgarea, R., and Sculean, A. (2017). Prevalence and mechanisms of peri-implant diseases. *Journal of Dental Research* 96 (1): 31–37.
- **20** Roccuzzo, M., Bonino, F., Aglietta, M., and Dalmasso, P. (2012). Ten-year results of a three arms prospective cohort study on implants in periodontally compromised patients, part 2: clinical results. *Clinical Oral Implants Research* 23: 389–395.
- **21** Renvert, S., Polyzois, I., and Persson, G.R. (2013). Treatment modalities for peri-implant mucositis and peri-implantitis. *American Journal of Dentistry* 26 (6): 313–318. PMID: 24640434.

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- **22** Roccuzzo, M., Layton, D.M., Roccuzzo, A., and Heitz-Mayfield, L.J. (2018). Clinical outcomes of periimplantitis treatment and supportive care: a systematic review. *Clinical Oral Implants Research* 29 (Suppl 16): 331–350.<https://doi.org/10.1111/clr.13287>. PMID: 30328195.
- **23** Sanz, M. and ChappleI, L. (2012). Working group 4 of the VIII european workshop on periodontology. clinical research on periimplant diseases: consensus report of working group 4. *Journal of Clinical Periodontology* 39 (Suppl 12): 202–206.
- **24** Matuliene, G., Pjetursson, B.E., Salvi, G.E. et al. (2008). Influence of residual pockets on progression of periodontitis and tooth loss: Results after 11 years of maintenance. *Journal of Clinical Periodontology* 35: 685–695.
- **25** Salvi, G.E., Mischler, D.C., Schmidlin, K. et al. (2014). Risk factors associated with the longevity of multi-rooted teeth: Longterm outcomes after active and supportive periodontal therapy. *Journal of Clinical Periodontology* 41: 701–707.
- **26** Parma Benfenati, S. and Roncati, M. (2017). Soft tissue management-reconstruction techniques. In: *Atlas text book Periodontology and implant therapy*, 915–998. Milano, Italy: Quintessence Publishing.
- **27** Lindhe, J., Meyle, J., and Group D of European Workshop on Periodontology (2008). Peri-implant diseases: consensus report of the sixth european workshop on periodontology. *Journal of Clinical Periodontology* 35 (8 Suppl): 282–285.
- **28** Silverstein, L., Garg, A., Callan, D., and Shatz, P. (1998). The key to success: maintaining the long-term health of implants. *Dentistry Today* 17 (2): 104. 106, 108-11.
- **29** Roncati, M and Marzola, P. (2011). If I say implant what you think? Educational DVD on implant therapy. Turin, Italy: Tueor Ed,. Italian, Tueor services.
- **30** Magnuson, B., Harsono, M., Stark, P.C., et al. (2013). Comparison of the effect of two interdental cleaning devices around implants on the reduction of bleeding: a 30-day randomized clinical trial. *Compendium of Continuing Education in Dentistry* 34 Spec No 8: 2–7. PMID: 24568169.
- **31** Van der Weijden, F. (2015). Slot DE Efficacy of homecare regimens for mechanical plaque removal in managing gingivitis a meta review. *Journal of Clinical Periodontology* 42 (Suppl. 16): S77–S91.
- **32** Wilkins, E. (1999). *Clinical Practice of the Dental Hygienist*, 8e. Philadelphia: Lea and Febiger.
- **33** West, N.X. and Moran, J.M. (2008). Home-use preventive and therapeutic oral products. *Periodontology 2000* 48: 7–9.
- **34** Kuo, Y.W., Yen, M., Fetzer, S., and Lee, J.D. (2013). Toothbrushing versus toothbrushing plus tongue cleaning in reducing halitosis and tongue coating: a

systematic review and meta-analysis. *Nursing Research* 62 (6): 422–429.

- **35** Salzer, S., Slot, D.E., Van der Weijden, F.A., and Dörfer, C.E. (2015). Efficacy of inter-dental mechanical plaque control in managing gingivitis – a meta-review. *Journal of Clinical Periodontology* 42 (Suppl 16): S92–S105.
- **36** Christou, V., Timmerman, M.F., Van der Velden, U., and Van der Weijden, F.A. (1998). Comparison of different approaches of interdental oral hygiene: interdental brushes versus dental floss. *Journal of Periodontology* 69 (7): 759–764.
- **37** Wehner, C., Husejnagic, S., Laky, B. et al. (2021). Effect of interdental brush design on plaque during nonsurgical periodontal therapy. *Clinical Oral Investigations* 25 (1): 87–94.
- **38** D'Accolti, M., Roncati, M., Soffritti, I. et al. (2021). Scrubbing wipes to dislodge dental biofilm can remove and inactivate viruses from tooth surface: perspectives for Covid-19 prevention. *Biomedical Journal of Scientific & Technical Research* 37 (4): 29592. BJSTR. MS.ID.006028.
- **39** Testori, T., Wang, H.L., Basso, M. et al. (2020). COVID-19 and Oral Surgery: a narrative review of preoperative mouth rinses. *Acta Stomatologica Croatica* 54 (4): 431–441.
- **40** Roncati, M. and Adriaens, L.M. (2013). Treatment of peri-implantitis: nonsurgical therapeutic approaches. *Annals of Oral & Maxillofacial Surgery* 1: 21.
- **41** Roncati, M. and Lucchese, A. (2013). Plaque removal efficacy of a novel oral care device: a microbiological assessment. *Journal of Dentistry and Oral Hygiene* 5 (8): 83–88.
- **42** Sparabombe S., Roncati M., Monterubbianesi R., et al. (2018). Assessment of antiplaque effectiveness of chlorhexidine-soaked gauze compared to chlorhexidine mouth rinse: randomized clinical trial. *Journal of Investigative and Clinical Dentistry* 9 (3): e12328.
- **43** McCracken G., Asuni A., Ritchie M., et al. (2017). Failing to meet the goals of periodontal recall programs. What next? *Periodontology 2000* 75 (1): 330–352.
- **44** Parma-Benfenati, S., Roncati, M., Galletti, P., and Tinti, C. (2014). Resorbable dome device and GBR: an alternative bony defect treatment around implants: a case series. *International Journal of Periodontics and Restorative Dentistry* 34 (5): 2–9.
- **45** Stefanini, M., Sangiorgi, M., Roncati, M. et al. (2016). Effect on plaque control in children patients with down syndrome using digital brush with or without chlorhexidine: a randomized clinical trial. *Special Care in Dentistry* 36 (2): 66–70.
- **46** Roccuzzo, M., DeAngelis, N., Bonino, L., and 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. *Clinical Oral Implants Research* 21: 490–496.

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Role of Compliance in Oral and Implant Health Maintenance: Significance, Risk Factors and Suggestions

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Background

Advancements in implant dentistry and related research have played a role in improving the overall success and survival of dental implants. Modernizations in surgical protocols, implant surface characteristics, bone and tissue regeneration techniques, and implant geometry are worth mentioning in this regard. Nevertheless, the risk of peri-implant diseases (peri-implant mucositis and peri-implantitis) cannot be overlooked. Habitual use of combustible and non-combustible tobacco products, routine alcohol consumption, and a state of immunosuppression are common risk factors that jeopardize the integrity of peri-implant soft tissues; and if left unaddressed and treated in a timely manner can lead to loss of osseointegration even implant loss. This book entitled "Saving Dental Implants" used references from indexed scientific literature to describe various protocols that can be used to augment peri-implant soft tissues and supporting bone. Home-care regimes, for young and elderly patients, which could help maintain the integrity of peri-implant soft and hard tissues are also comprehensively discussed; and the contribution of potential adjunct therapies such as osseous grafting, photobiomodulation, and photodynamic therapy are also discussed in detail. In summary, from the authors' perspective, a critical factor that supersedes all risk factors is routine oral and implant hygiene maintenance; and this is dependent on "compliance." By no means do the authors of this chapter intend to solely nominate the patient for unfavorable clinical scenarios such as occurrence of peri-implant diseases and implant failure;

however, poor patient compliance in terms of factors including oral hygiene maintenance, routine dental visits, (such as checkups and prophylaxis) and noncompliance toward medications and control of risk factors (such as smoking and hyperglycemia) may turn out to be calamitous particularly toward the long-term (preferably lifetime) implant success and survival. In this chapter, the authors emphasize on the significance of compliance (both from the patients' and professionals' perspectives); and highlights the common risk factors associated with non-compliance. The author have also documented suggestions that could help improve patient compliance; which in turn, can improve and maintain the soft-tissue-based and osseous integrity of oral tissues as well as dental implants.

Introduction

Modern Dentistry and Critical Thinking

Advancements in healthcare technology have revolutionized clinical dentistry and related research. Over the years, much of the research has emphasized on the etiopathogenesis, diagnosis, and management of oral conditions such as dental caries, periodontal and periimplant diseases, endodontic diseases, oral premalignant and malignant lesions, and malocclusion [1–9]. Moreover, various modern diagnostic (such as use of artificial intelligence), and therapeutic (including photobiomodulation, photodynamic therapy, and probiotic therapy) strategies have been incorporated into routine dental practice and have shown to facilitate diagnosis and management of oral diseases. There are, however, a variety of factors that influence the outcomes of such diagnostic and therapeutic protocols. Firstly, oral healthcare providers are routinely challenged with difficult situations in terms of making decisions that are in the best interest of patients with regards to their oral health-related conditions. A situation commonly encountered in a dental setting is "to extract or not to extract" questionable dentition. From a periodontal perspective, placement of dental implants in patients with a history of periodontitis may compromise the long-term success and survival of dental implants. In such situations, operator and/or oral health care provider's knowledge, adequate training, and clinical experience/skills are critical factors help formulate a diagnosis and treatment plan that is in the best interest of the patient. Such factors may also assist early diagnosis and successful management of oral health-related conditions such as those mentioned above [10-12]. Thus, clinicians must follow an evidence-based diagnostic and treatment approach that is unbiased for the determination of the most effective yet realistic therapeutic plan for patients [13–16].

Complications in Dental Procedures and Their Consequences

Complications and failures during and after dental treatment are not unusual in clinical dentistry; and may occur for procedures performed by the most experienced and knowledgeable dental professionals [17]. Moreover, it is not uncommon for patients to solely nominate dental professionals as the primary cause of complications and/ or failure of the treatment performed. Such attitudes and perceptions are risk factors for dental anxiety and phobia [18]; and may compromise communication and feeling of professional "trust" among patients and dental professionals [19]. Furthermore, postage of criticisms and comments on the internet and social media platforms based on dissatisfactory/negative personal experiences of patients with healthcare usually brings dental providers and related offices to the spotlight. People read online reviews about dental professionals and their offices; and this information usually influences the perceptions of readers toward dental professionals and their practices. Readers often use public opinions, which may either be constructive or critical to judge the skills of dental professionals; however, it is important to mention

that negative feedback that often targets dental professionals may not necessarily patients' own compliance toward post-operative care [20]. In other words, the ultimate objective of any form of medical treatment is to attain certain desired outcomes; and an essential factor that influences the occurrence and prevention of postoperative complications and success of the performed dental procedure is "patient compliance." [21] "Noncompliance" is a shortfall that may compromise and even have serious effects from the aspect of disease management [21–23]. In this context, "patient compliance" is a continuous area of concern for clinicians as well as researchers in healthcare practice and related research, respectively.

What is Compliance?

The Oxford Advanced American dictionary defines compliance as "the practice of obeying rules or requests made by people in authority." In other words, it is the act of obeying an instruction, rule, or request by an inspector or officer-in-charge.

Compliance and Non-compliance in Healthcare

In healthcare, compliance is described as the patient's attitude or behavior in terms of taking medications, following dietary and pre/post-operative instructions, routine follow-up visits and/or implementing changes in life-style (such as quitting smoking and routine exercise) based upon advice and recommendations from healthcare professionals [24, 25]. The World Health Organization has defined compliance as an extent to which, a patient is willing to take a medication, follow recommendations on nutrition or lifestyles, and comply with healthcare provider's instructions [26]. The term "adherence" is often used as a synonym for compliance in a healthcare setting [27].

"Non-compliance" or "non-adherence" occurs when patients' attitudes toward advices and recommendations by healthcare providers demonstrate a lack of analogy. Non- or lack of compliance is associated with lower quality of life, poor clinical outcomes, increased hospitalizations, and financial burdens in relation to healthcare [28]. Various types of non-compliance associated with general and oral healthcare systems are summarized in Figures 23.1 and 23.2.

Figure 23.1 General overview of types of non-compliance encountered in healthcare settings. *Source:* Adapted from Jin et al. [29].

Figure 23.2 Overview of types of non-compliance encountered toward oral health.

Common Risk Factors of Non-compliance

Risk factors of non-compliance can be categorized into five themes as shown in Figure 23.3. In the following text, each of these risk factors are discussed in further detail using references from evidence-based research.

Patient-related Factors

Dental Anxiety/Phobia

Research studies [30–34] have shown that dental anxiety (DA) or dental phobia (DP) affects up to 20% of the adult population; and compromises dental attendance and related therapy and oral health in general [34]. Results from a survey-based study reported that nearly 15.5% of the

Figure 23.3 Risk factors of non-compliance.

respondents avoided dental visits due to DP [35]; and 36.5% of the respondents had not visited a dentist for over 12 months [35]. A common reason for DA and DP is "needle phobia or NP," and the thought of being "poked" in the mouth with a sharp needle often contributes toward worsening DA and DP [36]. It has been reported that NP has psychologic (such as unconsciousness and convulsions), physiologic (such as bradycardia and hypotension) and social (such as avoidance behavior) consequences. People with high levels of DA often encounter increased DP which, in turn, compels them to (i) avoid routine dental visits and treatment, and (ii) seek dental care only when pain becomes inevitable. When such patients finally present themselves for treatment, the therapeutic needs are often extensive as well as invasive. Patients with DA and/or DP may not only skip dental check-ups and routine dental visits but may even demonstrate reluctance to seek help from a dental professional in times of emergency such as severe dental pain [37]. It is worth mentioning that high levels of DA and DP are directly associated with a poorer oral health status and an increased number of missing teeth [38].

Gender

Results from a survey-based study performed in Saudi Arabia reported that non-compliance toward dental visits is more common in male patients compared with females [39]. In other words, females have a more positive attitude toward dental visits than their counterparts. Moreover, female patients demonstrate a more positive behavior toward dental flossing compared with males [39]. Similar results were reported in a study done in Japan [40]. However, DP is more often manifested in women than men [34].

Age

A statistically significant association exists between advancing age and non-compliance [41]. By no means should this be interpreted as older patients are ignorant toward oral health maintenance and management of related diseases. However, it is known that forgetfulness and dementia are more often manifested in the senior population or elderly patients compared with relatively younger individuals [42]; and it is quite uncommon for elderly patients to forget abiding to pre- and post-operative instructions such as taking medications at the recommended time, dosage, and frequency. This seems to be a reason for missing scheduled dental and medical visits, especially in the elderly patient populations. Other factors that are linked with a lack of compliance among the elderly populations include but are not limited to lack of dexterity, compromised cognitive skills, and barriers in transportation to healthcare facilities [43, 44].

Previous Unpleasant Experience/s of Patients

Before seeking consultation and/or treatment from particular dental professional, patients often read reviews and opinions published online by individuals about the provider. These reviews and options are usually based on the experiences of patients with the healthcare provider and office staff. Commonly, the number of "stars" allotted to a practitioner and his/her office staff is often sufficient for readers to "judge" the skills of the healthcare professionals. The purpose of this chapter by no means is to criticize patients' perceptions and/or skills toward writing reviews for health professionals; however, positive reviews often decorated with "golden 5-stars" are usually a source of encouragement especially for new patients who are interested in seeking professional healthcare consultation and/ or treatment; however, despite the professional receiving numerous positive feedbacks, negative criticisms are not uncommon. Patients' options played by one or two stars on social media and search engines' review segments are sometimes sufficient to demoralize vulnerable patients and perhaps raise their latent or evident anxiety levels.

Demographics-related Factors

Education and Socioeconomic Status

Underprivileged socioeconomic and education standards are risk factors of oral diseases such as periodontitis [45]; however, these factors are also linked with poor patient compliance toward oral health maintenance and related treatment. Moreover, limited financial resources among patients with low education and underprivileged income statuses may compel them to strive to attain or reach the basic standards of living than focusing on oral health maintenance and related visits. These individuals often perceive that visit to dental professionals and attaining therapy for oral health-related diseases is a luxury reserved for the affluent populations [46]. According to Syrjälä et al. [47] low education standards are significantly associated with a lack of patient compliance toward visiting oral healthcare providers.

Treatment-related Factors

Complexity and Duration of Treatment

Healthcare providers may often change the therapeutic strategy such as alterations in types/dosage and frequency of medication intake. Moreover, in complex scenarios where preliminary treatment is unable able yield desired outcomes, a invasive treatment approach such as surgery is considered and recommended to patients. Such situation, despite being in the best interest of the patient from the healthcare professionals' perspective, may be a source of

psychological and/or financial stress for patients. Under such scenarios, patients tend to either (i) evade continuing/seeking treatment from the same provider; (ii) look for alternative treatment regimens including consultation with other healthcare professionals; (iii) consider complementary treatments including but not limited to herbal treatment, homeopathic medicine, and spiritual therapy; (iv) disregard all treatment options at least until the signs and symptoms of the underlying condition become inevitable.

Moreover, treatments that require routine maintenance such as glycemic control among patients with diabetes mellitus (DM) warrants long-term dietary restrictions and regular exercise. It has been reported that rate of compliance with diet among patients with DM varies from 25 to 65% [48]. Here it is worth mentioning that poorly controlled DM is also a classical risk factor for oral diseases such as periodontitis and peri-implantitis [49–51]. Research has shown that compliance with oral health-related recommendations is correlated with hemoglobin A1c levels [47]. This suggests that a state of chronic hyperglycemia among diabetic populations is a risk factor for poor compliance toward routine oral hygiene maintenance and related therapy.

Non-effectiveness of Therapy

Patients often expect fruitful outcomes related to treatment within a short-time frame after treatment if not immediately. Failure of attaining relief or achievement of the expected outcomes can compromise patient compliance toward healthcare-related visits and ongoing treatment. Here, it is important to note that a variety of factors that may compromise the outcomes of therapeutic interventions include tobacco smoking, malnutrition, immunosuppression, and habitual alcohol intake [52, 53]. Such factors may in turn compromise patient compliance toward their healthcare needs.

Communication and Trust Between the Patient and Dental Professional

A major barrier toward lack of compliance is lack of communication between the patient and dental professional. There are a variety of factors that may influence this relationship. Firstly, rushing with consultation and/or treatment often leaves patients with the perception of being ignored and/or disrespected. Secondly, a lack of communication with the patient undergoing treatment (even conscious sedation) may increase vulnerability among patients. Being attentive toward the treatment and/or prescription is critical and ethical; however, merely focusing on the region of interest in the maxillofacial region and notes/

prescriptions without intermittent direct "eyeball to eyeball" contact with the patient is demotivating for some patients. Furthermore, communication distortion or language barriers patients and practitioners may discourage patients from asking questions and pursuing future visits and treatment with dental professionals [54].

Suggestions

- **Recognizing patients with anxiety and dental phobia**: It is important for dental professionals to recognize patients with DA and DP prior to any form of examination or intervention to minimize the risk of unpleasant consequences such as those mentioned above.
- **Non-pharmacologic behavioral management**: A welcoming attitude from the professional team toward the patient especially those with diagnosed DA and DP may help improve compliance in vulnerable patient populations.
- **Communication**: It is critical for dental professionals to be attentive and "patient" with the patient. In other words, "good listening skills" or "being a good listener" influence the perception of the patient about the provider and the future or ongoing treatment. Patients should be invited to ask questions; and it is the responsibility of the dental professional to answer all questions raised in a simple, understandable, and yet logical manner. Motivation procedures such as reminding the patient of upcoming and future visits, follow-up communication via phone, letter, and/or email with patients, especially after invasive procedures and expressing appreciation even for minor efforts made by patients for improvements in oral health may help build the patient–dentist relationship and likely improve compliance. Interpreter or translator services should be utilized in situation where the patient and dental professional do not share the same primary language. Such patients should be identified prior to appointment; and in case the dental office is not equipped with interpreter services, then relatives, friends, and/or acquaintances that can communicate with the dental professional on behalf of the patient should be encouraged to accompany at the visit.
- **Sedation dentistry**: Treatment under partial/conscious (such as nitrous-oxide sedation) or intravenous/complete sedation (general anesthesia) is often performed for invasive procedures including removal of impacted third molars, full mouth extractions and extensive periodontal and endodontic interventions [55–57]. These approaches are also adopted for dental management of pediatric patients, and individuals with autism spectrum disorders and special needs [58–60]. Although dental interventions

performed under sedation address the individuals current needs, there is insufficient scientific evidence for its long-term impact on patients' dental attendance. Moreover, research has shown that patients with dental fear who undergo dental treatment under sedation demonstrate significantly less regular dental attendance at 2 and 10-year follow-up compared with patients who receive treatment without sedation [61, 62]. In this context, sedation dentistry is a useful strategy for addressing the dental needs of vulnerable patient populations; however, the contribution of sedation dentistry toward improvement in patient compliance toward oral health remains perplexed.

● **Community-based dental education programs**: Patient education with respect to oral and systemic health maintenance and importance of routine visits to healthcare providers plays a role in improving patient compliance [63, 64]. Community-based health awareness programs are particularly helpful for vulnerable patient communities, such as those with a deprived education and socioeconomic status [65–68]. In this context, community-based health insurance (CHI) can be a protuberant and promising concept in tackling financial healthcare issues confronting especially the underprivileged communities particularly in developing countries. Results from a cross-sectional study performed in Ethiopia showed that CHI was effective in improving awareness, attitude as well as compliance toward healthcare needs [69]. Execution of such insurance-based policies particularly in developing countries may also help patients visit oral healthcare providers, seek related therapy in a timely manner and improve compliance. Irrespective of the strategy adopted, identifying vulnerable patients (such as non-compliant individuals) is important and every possible level of support should be extended to these patients to educate them that can help improve the overall quality of life of these individuals.

Steps Toward Compliance: Authors' Opinion

From a general perspective, patient compliance is linked with "patience and persistence." Ideally, such characteristics should be demonstrated by the patient as well as the provider; however, realistically, it is the healthcare provider who is expected to demonstrate such traits in order to gain patients' confidence and trust. Such a strategy is a possible "first step" for non-compliant individuals such as with patients demonstrating DA, DP, and/or those who have had unpleasant experiences in the past with other healthcare settings. Communicating with patients in simple language is another factor that can help patients gain compliance.

Figure 23.4 Additional factors that may boost patient compliance.

Even minor efforts made by patients toward improvements in their health conditions should be acknowledged by healthcare providers; which in turn may further augment patient compliance. In addition, efforts such as facilitating and offering assistance related to communication (interpreter service) and arraigning transport for patients are additional factors that may boost patient compliance (Figure 23.4).

References

- **1** Patel, M. (2020). Dental caries vaccine: are we there yet? *Letters in Applied Microbiology* 70: 2–12.
- **2** Kim, S. and Kratchman, S. (2006). Modern endodontic surgery concepts and practice: a review. *Journal of Endodontia* 32: 601–623.
- **3** Luczaj-Cepowicz, E., Marczuk-Kolada, G., Obidzinska, M., and Sidun, J. (2019). Diagnostic validity of the use of ICDAS II and DIAGNOdent pen verified by microcomputed tomography for the detection of occlusal caries lesions-an in vitro evaluation. *Lasers in Medical Science* 34: 1655–1663.
- **4** Allan, B., Ruan, R., Landao-Bassonga, E. et al. (2021). Collagen membrane for guided bone regeneration in dental and orthopedic applications. *Tissue Engineering. Part A* 27: 372–381.
- **5** Gilligan, G., Garola, F., Piemonte, E. et al. (2021). Lichenoid proliferative leukoplakia, lichenoid lesions with evolution to proliferative leukoplakia or a continuum of the same precancerous condition? A revised hypothesis. *Journal of Oral Pathology & Medicine* 50: 129–135.
- **6** Warnakulasuriya, S. and Kerr, A.R. (2021). Oral cancer screening: past, present, and future. *Journal of Dental Research* 100: 1313, 220345211014795–1320 [https://doi.](https://doi.org/10.1177/00220345211014795) [org/10.1177/00220345211014795.](https://doi.org/10.1177/00220345211014795)
- **7** Almadi, K., Alkahtany, M., Alamam, Y. et al. (2021). Influence of propolis, ozone and photodynamic therapy in root canal disinfection on resin bond strength to radicular dentin. *Photodiagnosis and Photodynamic Therapy* 33: 102131.
- **8** Saghiri, M.A., Karamifar, K., Nath, D. et al. (2021). A novel polyurethane expandable root canal sealer. *Journal of Endodontia* 47: 612–620.
- **9** Shen, H., Zhi, Y., Zhu, F. et al. (2021). Experimental and clinical evaluation of BMP2-CPC graft versus deproteinized bovine bone graft for guided bone regeneration: a pilot study. *Dental Materials Journal* 40: 191–201.
- **10** Carrard, V.C. and van der Waal, I. (2018). A clinical diagnosis of oral leukoplakia; a guide for dentists. *Medicina Oral, Patología Oral y Cirugía Bucal* 23: e59–e64.

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- **11** Fernández-Feijoo, J., Carro, B., Branco, A. et al. (2019). The effect of dentist experience on the treatment of individuals with disability under general anesthesia. *Special Care in Dentistry* 39: 281–286.
- **12** Sun, T.M., Lee, H.E., and Lan, T.H. (2019). The influence of dental experience on a dental implant navigation system. *BMC Oral Health* 19: 222.
- **13** Abdellatif, H., Dechow, P.C., and Jones, D.L. (2011). Principles of evidence-based dental practice (EBDP). *Texas Dental Journal* 128: 155–164.
- **14** Afrashtehfar, K.I., Eimar, H., Yassine, R. et al. (2017). Evidence-based dentistry for planning restorative treatments: barriers and potential solutions. *European Journal of Dental Education* 21: e7–e18.
- **15** Rawat, P., Goswami, R.P., Kaur, G. et al. (2018). Knowledge, attitude, and behavior toward evidencebased dentistry among dental professionals in Jodhpur Rajasthan, India. *The Journal of Contemporary Dental Practice* 19: 1140–1146.
- **16** Straub-Morarend, C.L., Marshall, T.A., Holmes, D.C., and Finkelstein, M.W. (2013). Toward defining dentists' evidence-based practice: influence of decade of dental school graduation and scope of practice on implementation and perceived obstacles. *Journal of Dental Education* 77: 137–145.
- **17** Barsekow, F. (1990). Complications of daily dental work and expert opinion aspects. *Zahnärztliche Welt, Zahnärztliche Rundshau* 99: 629–635.
- **18** Appukuttan, D.P. (2016). Strategies to manage patients with dental anxiety and dental phobia: literature review. *Clinical, Cosmetic and Investigational Dentistry* 8: 35–50.
- **19** Yuan, S., Freeman, R., Hill, K. et al. (2020). Communication, trust and dental anxiety: a personcentred approach for dental attendance behaviours. *Dentistry Journal (Basel)* 8: 118.
- **20** Lu, X., Zhang, R., Wu, W. et al. (2018). Relationship between internet health information and patient compliance based on trust: empirical study. *Journal of Medical Internet Research* 20: e253.
- **21** Amerio, E., Mainas, G., Petrova, D. et al. (2020). Compliance with supportive periodontal/peri-implant therapy: a systematic review. *Journal of Clinical Periodontology* 47: 81–100.
- **22** Donovan, J.L. and Blake, D.R. (1992). Patient noncompliance: deviance or reasoned decision-making? *Social Science & Medicine* 34: 507–513.
- **23** Keim, R.G. (2016). The non-compliance issue. *Journal of Clinical Orthodontics* 50: 399–400.
- **24** Morris, L.S. and Schulz, R.M. (1992). Patient compliance – an overview. *Journal of Clinical Pharmacy and Therapeutics* 17: 283–295.
- **25** Ormel, H.L., van der Schoot, G.G.F., Sluiter, W.J. et al. (2018). Predictors of adherence to exercise interventions during and after cancer treatment: a systematic review. *Psychooncology* 27: 713–724.
- **26** Tachalov, V.V., Orekhova, L.Y., Isaeva, E.R. et al. (2018). Characteristics of dental patients determining their compliance level in dentistry: relevance for predictive, preventive, and personalized medicine. *The EPMA Journal* 9: 379–385.
- **27** Osterberg, L. and Blaschke, T. (2005). Adherence to medication. *The New England Journal of Medicine* 353: 487–497.
- **28** Gold, D.T. and McClung, B. (2006). Approaches to patient education: emphasizing the long-term value of compliance and persistence. *The American Journal of Medicine* 119: S32–S37.
- **29** Jin, J., Sklar, G.E., Min Sen Oh, V., and Chuen Li, S. (2008). Factors affecting therapeutic compliance: a review from the patient's perspective. *Therapeutics and Clinical Risk Management* 4: 269–286.
- **30** Milgrom, P., Fiset, L., Melnick, S., and Weinstein, P. (1988). The prevalence and practice management consequences of dental fear in a major US city. *Journal of the American Dental Association* 116: 641–647.
- **31** Armfield, J.M., Spencer, A.J., and Stewart, J.F. (2006). Dental fear in Australia: who's afraid of the dentist? *Australian Dental Journal* 51: 78–85.
- **32** Moore, R., Birn, H., Kirkegaard, E. et al. (1993). Prevalence and characteristics of dental anxiety in danish adults. *Community Dentistry and Oral Epidemiology* 21: 292–296.
- **33** Jeddy, N., Nithya, S., Radhika, T., and Jeddy, N. (2018). Dental anxiety and influencing factors: a cross-sectional questionnaire-based survey. *Indian Journal of Dental Research* 29: 10–15.
- **34** Milgrom, P., Newton, J.T., Boyle, C. et al. (2010). The effects of dental anxiety and irregular attendance on referral for dental treatment under sedation within the national health service in London. *Community Dentistry and Oral Epidemiology* 38: 453–459.
- **35** Gatchel, R.J., Ingersoll, B.D., Bowman, L. et al. (1983). The prevalence of dental fear and avoidance: a recent survey study. *Journal of the American Dental Association (1939)* 107: 609–610.
- **36** Majstorovic, M. and Veerkamp, J.S. (2004). Relationship between needle phobia and dental anxiety. *Journal of Dentistry for Children (Chicago, Ill.)* 71: 201–205.
- **37** Skaret, E., Berg, E., Kvale, G., and Raadal, M. (2007). Psychological characteristics of Norwegian adolescents reporting no likelihood of visiting a dentist in a situation with toothache. *International Journal of Paediatric Dentistry* 17: 430–438.
- **38** Hägglin, C., Berggren, U., Hakeberg, M., and Ahlqwist, M. (1996). Dental anxiety among middle-aged and elderly women in Sweden. A study of oral state, utilisation of dental services and concomitant factors. *Gerodontology* 13: 25–34.
- **39** Hamasha, A.A., Alshehri, A., Alshubaiki, A. et al. (2018). Gender-specific oral health beliefs and behaviors among adult patients attending King Abdulaziz Medical City in Riyadh. *The Saudi Dental Journal* 30: 226–231.
- **40** Fukai, K., Takaesu, Y., and Maki, Y. (1999). Gender differences in oral health behavior and general health habits in an adult population. *The Bulletin of Tokyo Dental College* 40: 187–193.
- **41** Parajuli, J., Saleh, F., Thapa, N., and Ali, L. (2014). Factors associated with nonadherence to diet and physical activity among Nepalese type 2 diabetes patients; a cross sectional study. *BMC Research Notes* 7: 758.
- **42** Grande, G., Qiu, C., and Fratiglioni, L. (2020). Prevention of dementia in an ageing world: evidence and biological rationale. *Ageing Research Reviews* 64: 101045.
- **43** Syed, S.T., Gerber, B.S., and Sharp, L.K. (2013). Traveling towards disease: transportation barriers to health care access. *Journal of Community Health* 38: 976–993.
- **44** Cramer, J.A. (1998). Enhancing patient compliance in the elderly. Role of packaging aids and monitoring. *Drugs & Aging* 12: 7–15.
- **45** Javed, F., Näsström, K., Benchimol, D. et al. (2007). Comparison of periodontal and socioeconomic status between subjects with type 2 diabetes mellitus and non-diabetic controls. *Journal of Periodontology* 78: 2112–2119.
- **46** Duane, B., Harford, S., Ramasubbu, D. et al. (2019). Environmentally sustainable dentistry: a brief introduction to sustainable concepts within the dental practice. *British Dental Journal* 226: 292–295.
- **47** Syrjälä, A.M., Kneckt, M.C., and Knuuttila, M.L. (1999). Dental self-efficacy as a determinant to oral health behaviour, oral hygiene and HbA1c level among diabetic patients. *Journal of Clinical Periodontology* 26: 616–621.
- **48** Cerkoney, K.A. and Hart, L.K. (1980). The relationship between the health belief model and compliance of persons with diabetes mellitus. *Diabetes Care* 3: 594–598.
- **49** Akram, Z., Alqahtani, F., Alqahtani, M. et al. (2020). Levels of advanced glycation end products in gingival crevicular fluid of chronic periodontitis patients with and without type-2 diabetes mellitus. *Journal of Periodontology* 91: 396–402.
- **50** Javed, F., Al-Askar, M., and Al-Hezaimi, K. (2012). Cytokine profile in the gingival crevicular fluid of periodontitis patients with and without type 2 diabetes: a literature review. *Journal of Periodontology* 83: 156–161.
- **51** Javed, F. and Romanos, G.E. (2009). Impact of diabetes mellitus and glycemic control on the osseointegration of dental implants: a systematic literature review. *Journal of Periodontology* 80: 1719–1730.
- **52** Natoli, R.M., Yu, H., Meislin, M.C. et al. (2018). Alcohol exposure decreases osteopontin expression during fracture healing and osteopontin-mediated mesenchymal stem cell migration in vitro. *Journal of Orthopaedic Surgery and Research* 13: 101.
- **53** Xu, B., Chen, L., and Lee, J.H. (2020). Smoking and alcohol drinking and risk of non-union or delayed union after fractures: a protocol for systematic review and dose-response meta-analysis. *Medicine (Baltimore)* 99: e18744.
- **54** Plainfield, S. (1969). Communication distortion. The language of patients and practitioners of dentistry. *The Journal of Prosthetic Dentistry* 22: 11–19.
- **55** Fiorillo, L. (2019). Conscious sedation in dentistry. *Medicina (Kaunas)* 55: 778.
- **56** Southerland, J.H. and Brown, L.R. (2016). Conscious intravenous sedation in dentistry: a review of current therapy. *Dental Clinics of North America* 60: 309–346.
- **57** Trieger, N. (1989). Intravenous sedation in dentistry and oral surgery. *International Anesthesiology Clinics* 27: 83–91.
- **58** Orellana, L.M., Martínez-Sanchis, S., and Silvestre, F.J. (2014). Training adults and children with an autism spectrum disorder to be compliant with a clinical dental assessment using a TEACCH-based approach. *Journal of Autism and Developmental Disorders* 44: 776–785.
- **59** Sacoor, S. (2017). Anaesthesia and sedation for the autistic patient. *Society for the Advancement of Anaesthesia in Dentistry Digest* 33: 40–43.
- **60** Wang, Y.C., Lin, I.H., Huang, C.H., and Fan, S.Z. (2012). Dental anesthesia for patients with special needs. *Acta Anaesthesiologica Taiwanica* 50: 122–125.
- **61** Berggren, U. (1986). Long-term effects of two different treatments for dental fear and avoidance. *Journal of Dental Research* 65: 874–876.
- **62** Hakeberg, M., Berggren, U., Carlsson, S.G., and Gröndahl, H.G. (1993). Long-term effects on dental care behavior and dental health after treatments for dental fear. *Anesthesia Progress* 40: 72–77.
- **63** Javed, F. and Michelogiannakis, D. (2019). Is oral health maintenance/promotion crucial in patients treated with the left ventricular assist device? *The American Journal of the Medical Sciences* 358: 168.
- **64** Javed, F., Khan, J., Youssef, M. et al. (2020). Dental management of patients with congestive heart failure before and after implantation of ventricular assist devices: linking the missing protocol. *Scandinavian Cardiovascular Journal* 54: 206–211.

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- **65** Siddique, I. and Mitchell, D.A. (2013). The impact of a community-based health education programme on oral cancer risk factor awareness among a Gujarati community. *British Dental Journal* 215: E7.
- **66** Smith, P.D., Thullen, M., Evans, C. et al. (2019). Benefits and challenges of partner site engagement with communitybased dental education programs: the partner sites' perspectives. *Journal of Dental Education* 83: 1012–1018.
- **67** Strauss, R.P., Stein, M.B., Edwards, J., and Nies, K.C. (2010). The impact of community-based dental education on students. *Journal of Dental Education* 74: S42–S55.
- **68** Yang, K.I., Chung-Do, J.J., Fujitani, L. et al. (2019). Advancing community-based participatory research to address health disparities in Hawai'i: perspectives from academic researchers. *Hawai'i Journal of Medicine & Public Health* 78: 83–88.
- **69** Workneh, S.G., Biks, G.A., and Woreta, S.A. (2017). Community-based health insurance and communities' scheme requirement compliance in Thehuldere district, northeast Ethiopia: cross-sectional community-based study. *ClinicoEconomics and Outcomes Research* 9: 353–359.

Systemic Factors and Peri-implant Health

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Introduction

For centuries, dental practitioners have relied on partial dentures, complete dentures, and fixed prosthesis (such as bridges) for the replacement of missing teeth. Dental implants have revolutionized modern clinical practice and are a contemporary substitute to such traditional fixed and removable dental prosthesis. It is well known that dental implants can osseointegrate and remain functionally and aesthetically stable over long durations in patients with missing teeth. Studies have reported high success and survival rates of dental implants in systemically healthy individuals; however, dental implant therapy has also been reported to be successful among patients with systemic disorders, such as diabetes mellitus and acquired immune deficiency syndrome (AIDS). This chapter provides essential information on the osseointegration and survival of dental implants in medically challenged patients. In the present chapter, an evidence-based approach was adopted to identify original research studies that had assessed the success and/or survival of dental implants in patients with systemic diseases. In this chapter, we compiled original studies from indexed databases (including PubMed, MEDLINE, OVID, ISI Web of Knowledge, Scopus, and EMBASE) with reference to their impact/influence on the survival and success of dental implants. These studies have formulated into individual subheadings focusing on specific focused questions and data has been presented using a systematic review approach. The content of this chapter is arranged on evidence-based dental implant therapy among patients with systemic diseases. Moreover, each sub-category discusses the outcomes of the respective studies and recommendations for future research are also presented.

Dental Implants in Patients with Diabetes Mellitus

It is well known that systemic disorders, such as poorlycontrolled diabetes mellitus (DM), jeopardize periodontal health and are also a significant risk factor for dental implant failure $[1-3]$. One explanation for this is that chronic hyperglycemia increases the formation and accumulation of glucose-mediated advanced-glycation-endproducts (AGEs) in the periodontal and systemic tissues in patients with poorly controlled DM [4, 5]. These AGEs play a role in the pathogenesis and altered periodontal wound healing by activating the receptors for AGEs (RAGE) located on the periodontium [5] (Figure 24.1). In addition, it has been reported that endothelial cells take up glucose passively in an insulin-independent manner, thereby causing tissue damage [6]. Also, chronic hyperglycemia has also been associated with alterations in host resistance by defective migration of polymorphonuclear leukocytes, impairment in phagocytosis, and exaggeration in inflammatory response to microbial products [7]. These factors may also be held responsible for the increase prevalence of peri-implant diseases (peri-implant mucositis and periimplantitis) in patients with poorly controlled DM as compared to non-diabetic controls [8, 9].

On the contrary, glycemic control in patients diagnosed with DM has been reported to improve the overall health status [10]. Studies have reported that dental implants and osseointegrate and remain functionally stable in patients with well-controlled DM in a manner contrast to patients with a poor metabolic control of DM [11–13]. For example, in a clinical study by Tawil et al. [11], implant survival rates were comparable among patients with well-controlled diabetes and nondiabetic controls. Likewise, in an experimental study, Casap et al. [14] assessed the osseointegration of

Figure 24.1 Pathophysiology of soft tissue inflammation and alveolar bone loss in connection with a state of persistent hyperglycemia. AGEs, Advanced glycation endproducts; IL, Interleukin; TNF-α, Tumor necrosis factor alpha.

implant discs placed in tibial medullary spaces of 140 male diabetic and control rats. Histomorphometric results showed significantly higher trabecular bone volumes among controls and rats with well-controlled diabetes, as compared to those with poorly-controlled diabetes. Similar results were reported by de Molon et al. [15] A strict glycemic control has also been shown to reduce microvascular complications in diabetes [16]. It has been reported that maintenance of serum glycemic levels may help to improve the function of osteoblasts, and the progression of periodontal bone loss is markedly reduced in subjects with wellcontrolled diabetes compared to individuals with poorly controlled diabetes in subjects with diabetes [17]. The serum and GCF concentrations of proinflammatory cytokines are also significantly reduced in subjects with well-controlled diabetes compared to individuals with poorly controlled diabetes [18, 19]. Therefore, under optimal glycemic control, diabetic subjects can have a periodontal bone height similar to that of healthy individuals.

Although maintenance of serum glycemic levels plays an important role in successful osseointegration, there are other factors that may also assist in enhancing the implant survival rates in diabetic patients. Maintenance of periodontal healthy environment is essential for successful dental implant treatment [20]. Dental plaque is a major etiological factor in periodontal destruction and studies have reported higher scores of plaque index, BOP, and PPD in diabetic patients compared to nondiabetic controls [3, 21]. It has been reported that inflammatory periodontal diseases may increase insulin resistance in a way similar to obesity, thereby aggravating glycemic control [20]. Inflammatory reactions in the peri-implant tissues have been associated with the presence of dental plaque around implants [22]. Periodontal therapy has been shown to improve glycemic control in hyperglycemic patients [23]. In high-fat-fed diabetic rats, periodontitis accelerated the onset of severe insulin resistance and impaired glucose homeostasis [24]. Therefore, control and treatment of periodontal infections should thus be an important part of the overall management of diabetes mellitus patients and consequently could play an important role in the success of implant therapy.

The routine use of antibiotics in oral implantology is widespread; however, there is a controversy over the use of antimicrobial agents in healthy candidates for dental implant therapy [25]. In diabetic patients undergoing implant surgery, the use of antimicrobial agents reduces the risk of surgical wound infection and improves the implant survival rate. It has been shown that a preoperative antibiotic cover in diabetic patients improves the implant survival rate by 10.5% compared to healthy candidates for implants (improvement with antibiotics was 4.5%) [26]. In another study, data for 2973 implants were investigated with reference to success of osseointegration at different stages of implant treatment [27]. The results showed that at each stage of treatment, the implant survival rate was notably higher in subjects, who had received preoperative antibiotics [27]. It has been reported that the use of chlorhexidine mouth-rinse is effective at reducing the viability of *Porphyromonas gingivalis* infection and peri-implant mucositis [28]. A twice-daily use of an antiseptic mouthwash has been suggested for the maintenance of dental implants [29]. In conclusion, dental implants can osseointegrate and remain functionally stable in patients with DM provided the glycemic levels are maintained. Nevertheless, the role of routine dental hygiene maintenance and regular dental checkups in this regard should not be overlooked.

Dental Implants in Patients with Cardiovascular Diseases

Cardiovascular diseases (CVD) are a group of diseases that include atherosclerosis, congestive heart failure, coronary artery disease, hypertension, and vascular stenosis. It has been proposed that restricted supply of oxygen and nutrients to tissues may negatively affect osseointegration in patients with CVD [30]. To our knowledge from indexed literature, only a limited number of studies have assessed the influence of CVD on osseointegration of dental implants [31]. It has

Authors et al.	Study design	Participants	Outcome
Khadivi et al. [31]	Retrospective	Group 1:39 patients with CVD Group 2: 98 without CVD	There was no difference in implant survival rates among patients with and without controlled CVD.
van Steenberghe et al. [40]	Prospective	399^a	CVD was not associated with implant failure.
Alsaadi et al. [41]	Retrospective	2004^a	CVD was not associated with implant failure.
Alsaadi et al. [42]	Retrospective	283°	CVD was not associated with implant failure.
Neves et al. [43]	Retrospective	721^a	CVD was not associated with implant failure.
Nobre Mde et al. [44]	Retrospective	Group 1:38 patients with CVD	CVD was not associated with implant failure.
		Group 2: 32 without CVD	

Table 24.1 Outcome of assessing the outcome of implant therapy among patients with cardiovascular diseases.

CVD, cardiovascular diseases.

a Patients had systemic diseases, including CVD. The exact number of patients with CVD remained unknown.

been reported that the risk of stroke is 80% higher for nonsmoking patients with up to 24 teeth as compared to individuals who had 25 or more teeth [32]. Likewise, results from another study reported an association to exist between periodontitis and increased risk of ischemic stroke compared with patients without periodontitis, gingivitis, or tooth loss [33]. The correlation between periodontitis and CVD has several possible pathophysiologic links. An increased systemic burden of bacteria, endotoxin, and other bacterial products could induce an abundant production of proinflammatory cytokines, cause inflammatory cell proliferation into large arteries, and increase the production of clotting factors (such as fibrinogen) through the liver, which may contribute to atherogenesis and thromboembolic events [34, 35]. Moreover, periodontopathogenic microbes may induce platelet aggregation that may be thrombogenic when entering the systemic circulation as in periodontitis [36, 37]. Furthermore, bacterial toxins (lipopolysaccharides) may also damage the endothelial cells by attacking the arterial lining [38]. These results indicate that there is a relationship between periodontitis and CVD. Since a previous history of periodontitis is a significant risk factor for peri-implant diseases [39], it is hypothesized the outcome of dental implant therapy is compromised in patients with CVD compared with systemically healthy controls.

Results from a retrospective analysis of patients with certain types of CVD showed no statistically significant difference in the implant failure rates among patients with and without CVD [31]. This study concluded that patients with controlled CVD are not at an increased risk of failure of osseointegration [31]. In another retrospective study, influence of CVD on implant failure, up to one week after the second stage of surgery was evaluated [40]. The results showed that there was no statistically significant association between CVD and failure of osseointegration [40].

Alsaadi and co-workers [41] CVD were not associated with an increased incidence of early implant failures. In another retrospective analysis, the authors reported that CVD such as hypertension and ischemic heart diseases are not significantly associated with early implant loss [42]. Although results from a publication showed that a history of CVD is associated with dental implant failure [43]; it has also been reported that oral rehabilitation with dental implants among patients with or without CVD is a valid treatment [44]. Table 24.1 summarizes the outcomes of studies that have assess the influence of CVD on the survival of dental implants. In conclusion, it seems that dental implants can osseointegrate and remain functionally stable in patients with CVD; however, further long-term follow-up studies are needed in this regard.

Dental Implants in Patients with Hepatic Disorders

Aspartate aminotransferase (AST) is a pyridoxal phosphatedependent transaminase enzyme, which plays an essential role in the metabolism of amino acids [45]. AST is mainly found in the liver but is also present in other tissues including heart, red blood cells, and skeletal muscles. When body tissue or an organ such as the liver is diseased, additional AST is released into the bloodstream, thereby elevating its levels. In this regard, assessment of AST levels is commonly performed for the diagnosis of hepatic disorders [45, 46]. Elevated AST levels in the saliva and gingival crevicular fluid have been associated with periodontal inflammatory parameters (probing depth and gingival bleeding) [47, 48]. It has been reported that salivary AST levels are significantly higher in patients

with chronic periodontitis (CP) [49]; however, mechanical debridement of plaque and calculus from teeth surfaces results in a statistically significant decrease in salivary AST levels in patients with CP [49]. Similarly, increased AST activity in the peri-implant sulcular fluid, have been associated with peri-implant bleeding on probing and bone loss [50, 51]. Since levels of AST in blood are significantly higher in patients with hepatic disorders and that increased AST activity has been associated with periodontal and peri-implant diseases [49], it is hypothesized that the outcomes of implant therapy are compromised in patients with hepatic disorders. Following a vigilant review of pertinent indexed literature, no studies that had assessed the influence of hepatic disorders on the success and survival of dental implants were identified. However, it is imperative to interpret this outcome with caution as the possible contribution of elevated AST levels toward the initiation and progression of peri-implant diseases (peri-implant mucositis and peri-implantitis) cannot be disregarded.

Dental Implants in Patients with Psychological Disorders

It has been reported that scores of periodontal inflammatory parameters (including bleeding on probing, probing depth, and dental calculus index) are poorer among patients with psychiatric disorders, such as schizophrenia and mood disorders [52]. *Porphyromonas gingivalis* (*P. gingivalis*) (a classical microbe associated with the etiology of periodontal disease) can transmigrate to the brain and modulate an innate inflammatory response [53]. In patients with Alzheimer's disease, *P. gingivalis* inhibits the local interferon-gamma response by preventing entry of immune cells into the brain [54]. Moreover, an increased number of missing teeth has also been associated with an increased incidence and prevalence of dementia [55]. Since patients with psychological/psychiatric disorders are more susceptible to periodontal disease [52, 55]; it is hypothesized that the outcome of dental implant therapy is compromised in patients with psychological/psychiatric disorders as compared to healthy individuals.

A limited number of studies have assessed the success and survival of dental implants in patients with psychological/psychiatric disorders [56–61]. In the retrospective study, personal grief and depression were nominated as significant risk-factors associated with dental implant failure [56]. In the study by Addy et al. [58], dental implant retained prostheses were provided to 3 patients with psychiatric disorders. The study concluded that psychiatric

disorders are not a contraindication to dental implant therapy [58]. Results from a five-year follow-up study showed that dental implant retained prosthesis can remain functionally stable in patients with Schizophrenia [59]. Similarly, results from another case report showed successful implant surgery in the maxilla in a 72-year-old patient with Parkinson's disease; however, follow-up results were not reported in this study [61]. On the contrary, in another case report, Kromminga et al. [60] concluded that outcomes of dental implant therapy are compromised among patients with psychological disorders.

The literature with respect to the success and survival of dental implants in patients with psychiatric/psychological diseases is sparse and inconsistent. However, poor oral hygiene status, oral parafunctional habits such as bruxism, habits such as repeated insertion of fingers in the mouth and behavioral issues are common in patients with psychiatric/psychological diseases. Such factors may complicate dental implant therapy in these patients and jeopardize the long-term success and survival of dental implants.

Dental Implants in Patients with HIV/ Acquired Immune Deficiency Syndrome

In a mono-centric study, 66 HIV-infected patients with a stable disease with good oral hygiene, requiring implant rehabilitation, were included [62]. In this study, each patient received at least 1 dental implant with a total of 190 implants placed in the study population. The primary outcome variables were implanting failure, prosthetic failure, peri-implant marginal bone loss, and biological complications including peri-implantitis, pus discharge, pain, and paresthesia [62]. The peri-implant status was assessed at the time in intervention and after 12 months of followup. At follow-up, there was no evidence of fractures of fixtures or paresthesia and implant failure occurred in patients (15 out of the 190 fixtures) [62]. The study concluded that dental implant therapy is a suitable therapeutic strategy for oral rehabilitation in HIV-infected patients provided the disease is well-controlled and appropriate infection control protocols are adopted [62]. Similar results were reported in another study [63]. In total, 13 studies were identified [62–74]. The study participants ranged between 1 and 68 patients and the duration of follow-up ranged between 4 weeks and 10 years. Results from nearly 50% of the studies were based on case reports and case series [65–67, 71–74]. All studies reported that dental implants can remain functionally stable in HIV-positive patients with stable disease (Table 24.2). There is insufficient evidence to determine whether in the long-term,

Authors et al.	Study design	Participant/s	HIV/AIDS	Follow-up	Result
Gherlone et al. [62]	Prospective	68 patients	$HIV+$	1 year	Dental implants can remain functionally stable in HIV-positive patients with stable disease
Gherlone et al. [63]	Retrospective	66 patients	$HIV+$	1 year	Dental implants can remain functionally stable in HIV-positive patients with stable disease
May et al. $[64]$	Retrospective	16 patients	AIDS	5 years	Dental implants can remain functionally stable in HIV-positive patients with stable disease
Vidal et al. [65]	Case series	3 patients	$HIV+$	Up to 10 years	Dental implants can remain functionally stable in HIV-positive patients with stable disease
Gay-Escoda et al. [66]	Case-series	9 patients	$HIV+$	$~6$ years	Dental implants can remain functionally stable in HIV-positive patients with stable disease
Romanos et al. [67]	Case report	43-year-old male	$HIV+$	4 years	Immediately loaded dental implants can remain functionally stable in HIV-positive patients with stable disease
Oliveira et al. [68]	Prospective	40 patients	$HIV+$	1 year	Dental implants can remain functionally stable in HIV+ patients with stable disease
Kolhatkar et al. [69]	Retrospective	2 patients	$HIV+$	Up to 4 weeks	Dental implants can remain functionally stable in HIV+ patients with stable disease
Stevenson et al. [70]	Prospective	20 patients	$HIV+$	6 months	Dental implants can remain functionally stable in HIV+ patients with stable disease
Strietzel et al. [71]	Case series	3 patients	$HIV+$	$~2$ years	Dental implants can remain functionally stable in HIV+ patients with stable disease
Achong et al. [72]	Case series	3 patients	$HIV+$	2 years	Dental implants can remain functionally stable in HIV+ patients with stable disease
Baron et al. [73]	Case report	1 patient	$HIV+$	2 years	Dental implants can remain functionally stable in HIV+ patients with stable disease

Table 24.2 An overview of studies, which have investigated the survival of dental implants in HIV-positive patients.

dental implants can remain functionally stable in HIV+ patients and patients with AIDS. Hence, further studies are warranted in this regard.

Dental Implants in Patients with Eating Disorders

Eating disorders (EDs) are abnormal eating habits, which may involve either insufficient or excessive food consumption thereby jeopardizing an individual's physical and/or mental health status [75]. The most common forms of EDs include anorexia nervosa (AN) and bulimia nervosa (BN) [76, 77]. AN is characterized by malnutrition and restricted food consumption [78, 79]; and BN is characterized by excessive food consumption (binge eating), followed by inappropriate compensatory behaviors such as selfinduced vomiting, use of laxatives, and excessive exercise [78, 79]. In general, EDs are a heterogeneous combination of AN- and BN-like EDs [80]. Prevalence of EDs has been documented in a limited number of studies [81–83]. EDs are a major health issue among children and adolescents of all ethnic groups in the United States [84]. According to the American Dietetic Association [85], EDs are dominant among adolescent females with prevalence rates of up to 5%. In Europe, lifetime estimated prevalence of 0.48, 0.51, 1.12, 0.72, and 2.15% have been reported for AN, BN, bingeeating disorder, sub-threshold binge-eating disorder, and any binge eating, respectively [83]. These values are up to eight times higher in females as compared to males with EDs [83]. In a recent cohort study, prevalence of AN, BN, and binge-eating disorders were reported to be 1.7, 0.8, and 2.3%, respectively, among Dutch females [82]. In this study [82], EDs were relatively rare among males.

Following an exhaustive search of indexed literature, 13 studies were initially identified. Twelve studies, that did **Table 24.3** Characteristics and outcome of the study that assessed osseointegration and survival of dental implants in a patient with eating disorders.

not fulfill the eligibility criteria, were excluded (Table 24.3). Therefore, one case report was included and processed for data extraction [86]. In this case report, oral rehabilitation using dental implants was performed in a 31-year-old nonsmoking female with a 10-year history of BN [86]. Clinical examination showed multiple crowns with extensive cervical caries, and prognosis of all teeth was poor. Periimplant tissues were inflamed, and lack of keratinized tissue necessitated additional periodontal procedures before a definitive restoration could be placed in this patient. Most of the remaining teeth were endodontically treated. According to the treatment plan, these teeth were extracted and followed by implant placement and fixed restorations. The goal of the prosthetic plan was to provide first molar occlusion to the patient using eight maxillary and six mandibular implants. The one-year follow-up results showed that the implants were aesthetically and functionally stable. The patients' self-esteem had also improved at follow-up [86]. To date, there is insufficient evidence to determine whether dental implants can remain functionally stable in patients with EDs. Hence, further studies are warranted in this regard.

Dental Implants in Patients with Autism Spectrum Disorders

Autism spectrum disorder, or ASD, is a group of developmental disorders that includes a variety/spectrum of symptoms, skills, and levels of disability [87]. Patients with ASD usually have these characteristics: (i) Ongoing social problems such as resistance to communication and interaction with others; (ii) repetitive behaviors and limited interests or activities; (iii) Symptoms that hurt an individuals' ability to function in various areas of life [87]. The precise etiology of ASD remains unclear; however, a number of risk factors have been associated with the etiology of ASD. These include genetic factors (genetic differences associated with

the X chromosome), neurobiological factors (problems with genetic code development involving multiple brain regions), and environmental factors (exposure to drugs and environmental toxicants) [88–91].

A compromised oral health status has been reported in adult patients with ASD. In a study from Sweden, dental caries and periodontal health status were assessed among 47 adults with ASD and 69 age- and gendermatched controls [92]. The results showed a significantly reduced stimulated salivary flow rate among patients with ASD as compared to controls. Buccal gingival recession was more often manifested in patients with ASD than controls [92]. Although there was no statistically significant difference in dental caries among patients with and without ASD, the authors hypothesized that adults with ASD are more susceptible to dental caries than healthy adults most likely as a consequence to a reduced salivary flow rate in ASD adults [92]. However, in another study, a statistically significant association was reported between ASD and the prevalence of dental caries. Other oral manifestations among adults with ASD are bruxism, self-perpetrated oral lesions, and dental malocclusions (most commonly anterior open bite) [93]. A vigilant review of indexed literature revealed no studies that assessed the success and survival of dental implants in adults with ASD. Hence, further studies are warranted in this regard.

Dental Implants in Patients with Crohn's Disease

Crohn's disease (CD) is an idiopathic chronic inflammatory disorder of the gastrointestinal tract, which may also affect the oral cavity [94]. CD is characterized by the presence of several antibody-antigen complexes, leading to autoimmune inflammatory processes in many parts of the body – for example, enteritis, recurrent oral ulceration, vasculitis, arthritis, or keratoconjunctivitis. Increasing trends in the incidence and prevalence of CD have been reported almost globally, with Canada, France, Italy, New Zealand, Scotland, and Scandinavia being among the most distinct regions [95, 96]. In the United States, there are 400,000–600,000 patients with CD. In the US state of Minnesota, an incidence rate of $7/10^5$ has been reported or CD [97]. Early reports from Canada (Quebec and Ontario) have shown a lower incidence and prevalence of CD $(0.7/10^5 \text{ and } 33/10^5, \text{ respectively})$ [95, 98]. An Australian study conducted between the years 1971 and 2001 showed an increase in annual rates from 0.13 to $2/10^5$ in a typical urban patient population [99]. Although the incidence of CD remains low in Asian countries (including China, India, and Malaysia), cases of CD are often misdiagnosed as enteric tuberculosis, or amebic colitis [95, 100–102]. Therefore, the Asian continent may also harbor a vast majority of CD patients worldwide.

The prevalence rates of oral manifestations in patients with CD vary between 5 and 20%; however, in pediatric patients, the prevalence is much higher (48–80%) [103]. It has been reported that patients with CD perceive their oral

health to be worse than individuals without CD (controls) [104]. In this study, oral health-related problems, such as gingivitis and dental caries, were more often reported by patients with CD than controls [104]. In addition, clinical scores of the markers of periodontitis (bleeding on probing, probing pocket depth, and clinical attachment loss) have been reported to be higher in patients with CD than controls [105]. Furthermore, ulcerative lesions involving the lips, tongue, buccal mucosa, and posterior pharynx have also been reported in patients with CD as compared to controls [106].

The literature search showed five studies [40–42, 107] that had assessed the survival of dental implants in patients with CD. Results from four studies [40–42, 107] reported that CD is significant factor for implant therapy. In a retrospective study by van Steenberghe et al. [40] two of three patients with CD had implant failures (3 out of 10 inserted implants failed). In their study, Peron et al. [108] reported two cases with CD in whom dental implants successfully osseointegrated and remained functionally stable up to 13 and 12 months of follow-up (Table 24.4).

a The total population comprised of 399 individuals (including 3 patients with Crohn's disease). CD: Crohn's disease; NA: Not available.

Conclusion

Systemic diseases are not a contraindication for dental implant therapy. Dental implants can osseointegrate and remain stable for prolonged durations in medically compromised individuals. Routine oral hygiene maintenance and timely medical diagnosis and treatment of systemic

References

- **1** Oates, T.W., Dowell, S., Robinson, M., and McMahan, C.A. (2009). Glycemic control and implant stabilization in type 2 diabetes mellitus. *Journal of Dental Research* 88: 367–371.
- **2** Javed, F. and Romanos, G.E. (2009). Impact of diabetes mellitus and glycemic control on the osseointegration of dental implants: a systematic literature review. *Journal of Periodontology* 80: 1719–1730.
- **3** Javed, F., Nasstrom, K., Benchimol, D. et al. (2007). Comparison of periodontal and socioeconomic status between subjects with type 2 diabetes mellitus and non-diabetic controls. *Journal of Periodontology* 78: 2112–2119.
- **4** Murillo, J., Wang, Y., Xu, X. et al. (2008). Advanced glycation of type i collagen and fibronectin modifies periodontal cell behavior. *Journal of Periodontology* 79: 2190–2199.
- **5** Holla, L.I., Kankova, K., Fassmann, A. et al. (2001). Distribution of the receptor for advanced glycation end products gene polymorphisms in patients with chronic periodontitis: a preliminary study. *Journal of Periodontology* 72: 1742–1746.
- **6** Ebersole, J.L., Holt, S.C., Hansard, R., and Novak, M.J. (2008). Microbiologic and immunologic characteristics of periodontal disease in hispanic americans with type 2 diabetes. *Journal of Periodontology* 79: 637–646.
- **7** Soory, M. (2002). Hormone mediation of immune responses in the progression of diabetes, rheumatoid arthritis and periodontal diseases. *Current Drug Targets. Immune, Endocrine and Metabolic Disorders* 2: 13–25.
- **8** Gomez-Moreno, G., Aguilar-Salvatierra, A., Rubio Roldan, J. et al. (2014). Peri-implant evaluation in type 2 diabetes mellitus patients: a 3-year study. *Clinical Oral Implants Research*<https://doi.org/10.1111/clr.12391>.
- **9** Ferreira, S.D., Silva, G.L., Cortelli, J.R. et al. (2006). Prevalence and risk variables for peri-implant disease in brazilian subjects. *Journal of Clinical Periodontology* 33: 929–935.

disorders contribute toward long-term success and survival of dental implants.

Conflict of Interest Disclosure

The author declares that he does not have conflict of interest related to this chapter.

- **10** Mager, D.R., Iniguez, I.R., Gilmour, S., and Yap, J. (2013). The effect of a low fructose and low glycemic index/load (fragile) dietary intervention on indices of liver function, cardiometabolic risk factors, and body composition in children and adolescents with nonalcoholic fatty liver disease (nafld). *JPEN Journal of Parenteral and Enteral Nutrition* [https://doi.org/10.1177/0148607113501201.](https://doi.org/10.1177/0148607113501201)
- **11** Tawil, G., Younan, R., Azar, P., and Sleilati, G. (2008). Conventional and advanced implant treatment in the type ii diabetic patient: surgical protocol and long-term clinical results. *The International Journal of Oral & Maxillofacial Implants* 23: 744–752.
- **12** Kwon, P.T., Rahman, S.S., Kim, D.M. et al. (2005). Maintenance of osseointegration utilizing insulin therapy in a diabetic rat model. *Journal of Periodontology* 76: 621–626.
- **13** Lee, S.B., Retzepi, M., Petrie, A. et al. (2013). The effect of diabetes on bone formation following application of the gbr principle with the use of titanium domes. *Clinical Oral Implants Research* 24: 28–35.
- **14** Casap, N., Nimri, S., Ziv, E. et al. (2008). Type 2 diabetes has minimal effect on osseointegration of titanium implants in psammomys obesus. *Clinical Oral Implants Research* 19: 458–464.
- **15** de Molon, R.S., Morais-Camilo, J.A., Verzola, M.H. et al. (2013). Impact of diabetes mellitus and metabolic control on bone healing around osseointegrated implants: removal torque and histomorphometric analysis in rats. *Clinical Oral Implants Research* 24: 831–837.
- **16** Rocha, M., Nava, L.E., Vazquez de la Torre, C. et al. (2001). Clinical and radiological improvement of periodontal disease in patients with type 2 diabetes mellitus treated with alendronate: a randomized, placebo-controlled trial. *Journal of Periodontology* 72: 204–209.
- **17** Taylor, G.W., Burt, B.A., Becker, M.P. et al. (1998). Glycemic control and alveolar bone loss progression in type 2 diabetes. *Annals of Periodontology* 3: 30–39.
- **18** Iwamoto, Y., Nishimura, F., Nakagawa, M. et al. (2001). The effect of antimicrobial periodontal treatment on circulating tumor necrosis factor-alpha and glycated

hemoglobin level in patients with type 2 diabetes. *Journal of Periodontology* 72: 774–778.

- **19** Javed, F., Al-Daghri, N.M., Wang, H.L. et al. (2014). Short-term effects of non-surgical periodontal treatment on the gingival crevicular fluid cytokine profiles in sites with induced periodontal defects: a study on dogs with and without streptozotocin-induced diabetes. *Journal of Periodontology* [https://doi.org/10.1902/jop.2014.](https://doi.org/10.1902/jop.2014.1401501-11) [1401501-11.](https://doi.org/10.1902/jop.2014.1401501-11)
- **20** Mealey, B.L. and Oates, T.W. (2006). Diabetes mellitus and periodontal diseases. *Journal of Periodontology* 77: 1289–1303.
- **21** Javed, F., Klingspor, L., Sundin, U. et al. (2009). Periodontal conditions, oral candida albicans and salivary proteins in type 2 diabetic subjects with emphasis on gender. *BMC Oral Health* 9: 12.
- **22** Maximo, M.B., de Mendonca, A.C., Renata Santos, V. et al. (2009). Short-term clinical and microbiological evaluations of peri-implant diseases before and after mechanical anti-infective therapies. *Clinical Oral Implants Research* 20: 99–108.
- **23** Javed, F., Ahmed, H.B., Mehmood, A. et al. (2014). Effect of nonsurgical periodontal therapy (with or without oral doxycycline delivery) on glycemic status and clinical periodontal parameters in patients with prediabetes: a short-term longitudinal randomized case-control study. *Clinical Oral Investigations* [https://doi.org/10.1007/](https://doi.org/10.1007/s00784-014-1185-6) [s00784-014-1185-6.](https://doi.org/10.1007/s00784-014-1185-6)
- **24** Watanabe, K., Petro, B.J., Shlimon, A.E., and Unterman, T.G. (2008). Effect of periodontitis on insulin resistance and the onset of type 2 diabetes mellitus in zucker diabetic fatty rats. *Journal of Periodontology* 79: 1208–1216.
- **25** Javed, F., Alghamdi, A.S., Ahmed, A. et al. (2013). Clinical efficacy of antibiotics in the treatment of peri-implantitis. *International Dental Journal* 63: 169–176.
- **26** Morris, H.F., Ochi, S., and Winkler, S. (2000). Implant survival in patients with type 2 diabetes: placement to 36 months. *Annals of Periodontology* 5: 157–165.
- **27** Laskin, D.M., Dent, C.D., Morris, H.F. et al. (2000). The influence of preoperative antibiotics on success of endosseous implants at 36 months. *Annals of Periodontology* 5: 166–174.
- **28** Leyes Borrajo, J.L., Garcia, V.L., Lopez, C.G. et al. (2002). Efficacy of chlorhexidine mouthrinses with and without alcohol: a clinical study. *Journal of Periodontology* 73: 317–321.
- **29** Ciancio, S.G., Lauciello, F., Shibly, O. et al. (1995). The effect of an antiseptic mouthrinse on implant maintenance: plaque and peri-implant gingival tissues. *Journal of Periodontology* 66: 962–965.
- **30** Elsubeihi, E.S. and Zarb, G.A. (2002). Implant prosthodontics in medically challenged patients: the university of toronto experience. *Journal of the Canadian Dental Association* 68: 103–108.
- **31** Khadivi, V., Anderson, J., and Zarb, G.A. (1999). Cardiovascular disease and treatment outcomes with osseointegration surgery. *The Journal of Prosthetic Dentistry* 81: 533–536.
- **32** Joshipura, K.J., Hung, H.C., Rimm, E.B. et al. (2003). Periodontal disease, tooth loss, and incidence of ischemic stroke. *Stroke* 34: 47–52.
- **33** Wu, T., Trevisan, M., Genco, R.J. et al. (2000). Periodontal disease and risk of cerebrovascular disease: the first national health and nutrition examination survey and its follow-up study. *Archives of Internal Medicine* 160: 2749–2755.
- **34** Carroll, G.C. and Sebor, R.J. (1980). Dental flossing and its relationship to transient bacteremia. *Journal of Periodontology* 51: 691–692.
- **35** Mask, A.G. Jr. (2000). Medical management of the patient with cardiovascular disease. *Periodontology 2000* 23: 136–141.
- **36** Herzberg, M.C. and Meyer, M.W. (1996). Effects of oral flora on platelets: possible consequences in cardiovascular disease. *Journal of Periodontology* 67: 1138–1142.
- **37** Herzberg, M.C. and Weyer, M.W. (1998). Dental plaque, platelets, and cardiovascular diseases. *Annals of Periodontology* 3: 151–160.
- **38** Reidy, M.A. and Bowyer, D.E. (1977). Scanning electron microscopy: morphology of aortic endothelium following injury by endotoxin and during subsequent repair. *Atherosclerosis* 26: 319–328.
- **39** Romanos, G.E., Javed, F., Delgado-Ruiz, R.A., and Calvo-Guirado, J.L. (2015). Peri-implant diseases: a review of treatment interventions. *Dental Clinics of North America* 59: 157–178.
- **40** van Steenberghe, D., Jacobs, R., Desnyder, M. et al. (2002). The relative impact of local and endogenous patient-related factors on implant failure up to the abutment stage. *Clinical Oral Implants Research* 13: 617–622.
- **41** Alsaadi, G., Quirynen, M., Komarek, A., and van Steenberghe, D. (2007). Impact of local and systemic factors on the incidence of oral implant failures, up to abutment connection. *Journal of Clinical Periodontology* 34: 610–617.
- **42** Alsaadi, G., Quirynen, M., Michiles, K. et al. (2008). Impact of local and systemic factors on the incidence of failures up to abutment connection with modified surface oral implants. *Journal of Clinical Periodontology* 35: 51–57.

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- **43** Neves, J., de Araujo Nobre, M., Oliveira, P. et al. (2016). Risk factors for implant failure and peri-implant pathology in systemic compromised patients. *Journal of Prosthodontics* [https://doi.org/10.1111/jopr.12508.](https://doi.org/10.1111/jopr.12508)
- **44** Nobre Mde, A., Malo, P., Goncalves, Y. et al. (2016). Outcome of dental implants in diabetic patients with and without cardiovascular disease: a 5-year post-loading retrospective study. *European Journal of Oral Implantology* 9: 87–95.
- **45** Miura, Y., Kanda, T., Yasui, S. et al. (2017). Hepatitis a virus genotype ia-infected patient with marked elevation of aspartate aminotransferase levels. *Clinical Journal of Gastroenterology* 10: 52–56.
- **46** Pokorska-Spiewak, M., Kowalik-Mikolajewska, B., Aniszewska, M. et al. (2016). Predictors of liver disease severity in children with chronic hepatitis B. *Advances in Clinical and Experimental Medicine* 25: 681–688.
- **47** Fiorellini, J.P., Nevins, M.L., Sekler, J. et al. (2000). Correlation of peri-implant health and aspartate aminotransferase levels: a cross-sectional clinical study. *The International Journal of Oral & Maxillofacial Implants* 15: 500–504.
- **48** Totan, A., Greabu, M., Totan, C., and Spinu, T. (2006). Salivary aspartate aminotransferase, alanine aminotransferase and alkaline phosphatase: possible markers in periodontal diseases? *Clinical Chemistry and Laboratory Medicine* 44: 612–615.
- **49** Kudva, P., Saini, N., Kudva, H., and Saini, V. (2014). To estimate salivary aspartate aminotransferase levels in chronic gingivitis and chronic periodontitis patients prior to and following non-surgical periodontal therapy: a clinico-biochemical study. *Journal of Indian Society of Periodontology* 18: 53–58.
- **50** Ruehling, A., Jepsen, S., Kocher, T., and Plagmann, H.C. (1999). Longitudinal evaluation of aspartate aminotransferase in the crevicular fluid of implants with bone loss and signs of progressive disease. *The International Journal of Oral & Maxillofacial Implants* 14: 428–435.
- **51** Paolantonio, M., Di Placido, G., Tumini, V. et al. (2000). Aspartate aminotransferase activity in crevicular fluid from dental implants. *Journal of Periodontology* 71: 1151–1157.
- **52** Nayak, S.U., Singh, R., and Kota, K.P. (2016). Periodontal health among non-hospitalized chronic psychiatric patients in mangaluru city-india. *Journal of Clinical and Diagnostic Research* 10: Zc40–Zc43.
- **53** Harding, A., Robinson, S., Crean, S., and Singhrao, S.K. (2017). Can better management of periodontal disease delay the onset and progression of alzheimer's disease? *Journal of Alzheimer's Disease* [https://doi.org/10.3233/jad-170046.](https://doi.org/10.3233/jad-170046)
- **54** Ide, M., Harris, M., Stevens, A. et al. (2016). Periodontitis and cognitive decline in alzheimer's disease. *PLoS One* 11: e0151081.
- **55** Stein, P.S., Desrosiers, M., Donegan, S.J. et al. (2007). Tooth loss, dementia and neuropathology in the nun study. *Journal of the American Dental Association (1939)* 138: 1314–1322. quiz 1381-1312.
- **56** Ekfeldt, A., Christiansson, U., Eriksson, T. et al. (2001). A retrospective analysis of factors associated with multiple implant failures in maxillae. *Clinical Oral Implants Research* 12: 462–467.
- **57** Tinner, D. and Marinello, C.P. (2002). Implantsupported dental prostheses in a depressed patient – a case report. *Schweizer Monatsschrift für Zahnmedizin* 112: 495–507.
- **58** Addy, L., Korszun, A., and Jagger, R.G. (2006). Dental implant treatment for patients with psychiatric disorders. *The European Journal of Prosthodontics and Restorative Dentistry* 14: 90–92.
- **59** Griess, M., Reilmann, B., and Chanavaz, M. (1998). The multi-modal prosthetic treatment of mentally handicapped patients--necessity and challenge. *The European Journal of Prosthodontics and Restorative Dentistry* 6: 115–120.
- **60** Kromminga, R., Habel, G., and Muller-Fahlbusch, H. (1991). Failure of dental implants following psychosomatic disturbances in the stomatognathic system – a clinical-catamnestic study. *Deutsche Stomatologie* 41: 233–236.
- **61** Kubo, K. and Kimura, K. (2004). Implant surgery for a patient with parkinson's disease controlled by intravenous midazolam: a case report. *The International Journal of Oral & Maxillofacial Implants* 19: 288–290.
- **62** Gherlone, E.F., Cappare, P., Tecco, S. et al. (2016). Implant prosthetic rehabilitation in controlled HIVpositive patients: a prospective longitudinal study with 1-year follow-up. *Clinical Implant Dentistry and Related Research* 18: 725–734.
- **63** Gherlone, E.F., Cappare, P., Tecco, S. et al. (2016). A prospective longitudinal study on implant prosthetic rehabilitation in controlled HIV-positive patients with 1-year follow-up: the role of cd4+ level, smoking habits, and oral hygiene. *Clinical Implant Dentistry and Related Research* 18: 955–964.
- **64** May, M.C., Andrews, P.N., Daher, S., and Reebye, U.N. (2016). Prospective cohort study of dental implant success rate in patients with aids. *International Journal of Implant Dentistry* 2: 20.
- **65** Vidal, F., Vidal, R., Bochnia, J. et al. (2017). Dental implants and bone augmentation in HIV-infected patients under haart: case report and review of the literature. *Special Care in Dentistry* 37: 150–155.
- **66** Gay-Escoda, C., Perez-Alvarez, D., Camps-Font, O., and Figueiredo, R. (2016). Long-term outcomes of oral rehabilitation with dental implants in HIV-positive patients: a retrospective case series. *Medicina Oral, Patología Oral y Cirugía Bucal* 21: e385–e391.
- **67** Romanos, G.E., Goldin, E., Marotta, L. et al. (2014). Immediate loading with fixed implant-supported restorations in an edentulous patient with an HIV infection: a case report. *Implant Dentistry* 23: 8–12.
- **68** Oliveira, M.A., Gallottini, M., Pallos, D. et al. (2011). The success of endosseous implants in human immunodeficiency virus-positive patients receiving antiretroviral therapy: a pilot study. *Journal of the American Dental Association (1939)* 142: 1010–1016.
- **69** Kolhatkar, S., Khalid, S., Rolecki, A. et al. (2011). Immediate dental implant placement in HIV-positive patients receiving highly active antiretroviral therapy: a report of two cases and a review of the literature of implants placed in HIV-positive individuals. *Journal of Periodontology* 82: 505–511.
- **70** Stevenson, G.C., Riano, P.C., Moretti, A.J. et al. (2007). Short-term success of osseointegrated dental implants in HIV-positive individuals: a prospective study. *The Journal of Contemporary Dental Practice* 8: 1–10.
- **71** Strietzel, F.P., Rothe, S., Reichart, P.A., and Schmidt-Westhausen, A.M. (2006). Implant-prosthetic treatment in HIV-infected patients receiving highly active antiretroviral therapy: report of cases. *The International Journal of Oral & Maxillofacial Implants* 21: 951–956.
- **72** Achong, R.M., Shetty, K., Arribas, A., and Block, M.S. (2006). Implants in HIV-positive patients: 3 case reports. *Journal of Oral and Maxillofacial Surgery* 64: 1199–1203.
- **73** Baron, M., Gritsch, F., Hansy, A.M., and Haas, R. (2004). Implants in an HIV-positive patient: a case report. *The International Journal of Oral & Maxillofacial Implants* 19: 425–430.
- **74** Rajnay, Z.W. and Hochstetter, R.L. (1998). Immediate placement of an endosseous root-form implant in an HIV-positive patient: report of a case. *Journal of Periodontology* 69: 1167–1171.
- **75** Bell, C., Waller, G., Shafran, R., and Delgadillo, J. (2017). Is there an optimal length of psychological treatment for eating disorder pathology? *The International Journal of Eating Disorders* [https://doi.org/10.1002/eat.22660.](https://doi.org/10.1002/eat.22660)
- **76** Tortorella, A., Brambilla, F., Fabrazzo, M. et al. (2014). Central and peripheral peptides regulating eating behaviour and energy homeostasis in anorexia nervosa and bulimia nervosa: a literature review. *European Eating Disorders Review* [https://doi.org/10.1002/](https://doi.org/10.1002/erv.2303) [erv.2303](https://doi.org/10.1002/erv.2303).
- **77** Fischer, S., Meyer, A.H., Dremmel, D. et al. (2014). Short-term cognitive-behavioral therapy for binge eating disorder: long-term efficacy and predictors of long-term treatment success. *Behaviour Research and Therapy* 58C: 36–42.
- **78** Romanos, G.E., Javed, F., Romanos, E.B., and Williams, R.C. (2012). Oro-facial manifestations in patients with eating disorders. *Appetite* 59: 499–504.
- **79** Swinbourne, J., Hunt, C., Abbott, M. et al. (2012). The comorbidity between eating disorders and anxiety disorders: prevalence in an eating disorder sample and anxiety disorder sample. *The Australian and New Zealand Journal of Psychiatry* 46: 118–131.
- **80** Johansson, A.K., Norring, C., Unell, L., and Johansson, A. (2012). Eating disorders and oral health: a matched case-control study. *European Journal of Oral Sciences* 120: 61–68.
- **81** Garner, D.M. and Garfinkel, P.E. (1980). Socio-cultural factors in the development of anorexia nervosa. *Psychological Medicine* 10: 647–656.
- **82** Smink, F.R., van Hoeken, D., Oldehinkel, A.J., and Hoek, H.W. (2014). Prevalence and severity of DSM-5 eating disorders in a community cohort of adolescents. *The International Journal of Eating Disorders* [https://doi.](https://doi.org/10.1002/eat.22316) [org/10.1002/eat.22316.](https://doi.org/10.1002/eat.22316)
- **83** Preti, A., Girolamo, G., Vilagut, G. et al. (2009). The epidemiology of eating disorders in six european countries: results of the esemed-wmh project. *Journal of Psychiatric Research* 43: 1125–1132.
- **84** Nicholls, D. and Viner, R. (2005). Eating disorders and weight problems. *BMJ* 330: 950–953.
- **85** Position of the american dietetic association (2001). Nutrition intervention in the treatment of anorexia nervosa, bulimia nervosa, and eating disorders not otherwise specified (ednos). *Journal of the American Dietetic Association* 101: 810–819.
- **86** Ambard, A. and Mueninghoff, L. (2002). Rehabilitation of a bulimic patient using endosteal implants. *Journal of Prosthodontics* 11: 176–180.
- **87** Scott, M., Jacob, A., Hendrie, D. et al. (2017). Employers' perception of the costs and the benefits of hiring individuals with autism spectrum disorder in open employment in australia. *PLoS One* 12: e0177607.
- **88** Landrigan, P.J., Lambertini, L., and Birnbaum, L.S. (2012). A research strategy to discover the environmental causes of autism and neurodevelopmental disabilities. *Environmental Health Perspectives* 120: a258–a260.
- **89** Abrahams, B.S. and Geschwind, D.H. (2010). Connecting genes to brain in the autism spectrum disorders. *Archives of Neurology* 67: 395–399.

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- **90** Coe, B.P., Girirajan, S., and Eichler, E.E. (2012). The genetic variability and commonality of neurodevelopmental disease. *American Journal of Medical Genetics. Part C, Seminars in Medical Genetics* 160c: 118–129.
- **91** Coe, B.P., Girirajan, S., and Eichler, E.E. (2012). A genetic model for neurodevelopmental disease. *Current Opinion in Neurobiology* 22: 829–836.
- **92** Blomqvist, M., Bejerot, S., and Dahllof, G. (2015). A cross-sectional study on oral health and dental care in intellectually able adults with autism spectrum disorder. *BMC Oral Health* 15: 81.
- **93** Orellana, L.M., Silvestre, F.J., Martinez-Sanchis, S. et al. (2012). Oral manifestations in a group of adults with autism spectrum disorder. *Medicina Oral, Patología Oral y Cirugía Bucal* 17: e415–e419.
- **94** Scheper, H.J. and Brand, H.S. (2002). Oral aspects of crohn's disease. *International Dental Journal* 52: 163–172.
- **95** Economou, M. and Pappas, G. (2008). New global map of crohn's disease: genetic, environmental, and socioeconomic correlations. *Inflammatory Bowel Diseases* 14: 709–720.
- **96** Galeone, C., Pelucchi, C., Barbera, G. et al. (2017). Crohn's disease in italy: a critical review of the literature using different data sources. *Digestive and Liver Disease* <https://doi.org/10.1016/j.dld.2016.12.033>.
- **97** Jess, T., Loftus, E.V. Jr., Harmsen, W.S. et al. (2006). Survival and cause specific mortality in patients with inflammatory bowel disease: a long term outcome study in olmsted county, minnesota, 1940-2004. *Gut* 55: 1248–1254.
- **98** Hiatt, R.A. and Kaufman, L. (1988). Epidemiology of inflammatory bowel disease in a defined northern california population. *The Western Journal of Medicine* 149: 541–546.
- **99** Phavichitr, N., Cameron, D.J., and Catto-Smith, A.G. (2003). Increasing incidence of crohn's disease in victorian children. *Journal of Gastroenterology and Hepatology* 18: 329–332.
- **100** Zheng, J.J., Zhu, X.S., Huangfu, Z. et al. (2005). Crohn's disease in mainland china: a systematic analysis of 50 years of research. *Chinese Journal of Digestive Diseases* 6: 175–181.
- **101** Hilmi, I., Tan, Y.M., and Goh, K.L. (2006). Crohn's disease in adults: observations in a multiracial asian population. *World Journal of Gastroenterology* 12: 1435–1438.
- **102** Desai, H.G. and Gupte, P.A. (2005). Increasing incidence of crohn's disease in india: is it related to improved sanitation? *Indian Journal of Gastroenterology* 24: 23–24.
- **103** Orosz, M. and Sonkodi, I. (2004). Oral manifestations in crohn's disease and dental management. *Fogorvosi Szemle* 97: 113–117.
- **104** Rikardsson, S., Jonsson, J., Hultin, M. et al. (2009). Perceived oral health in patients with crohn's disease. *Oral Health & Preventive Dentistry* 7: 277–282.
- **105** Vavricka, S.R., Manser, C.N., Hediger, S. et al. (2013). Periodontitis and gingivitis in inflammatory bowel disease: a case-control study. *Inflammatory Bowel Diseases* 19: 2768–2777.
- **106** Estrin, H.M. and Hughes, R.W. Jr. (1985). Oral manifestations in crohn's disease: report of a case. *The American Journal of Gastroenterology* 80: 352–354.
- **107** Alsaadi, G., Quirynen, M., Komarek, A., and van Steenberghe, D. (2008). Impact of local and systemic factors on the incidence of late oral implant loss. *Clinical Oral Implants Research* 19: 670–676.
- **108** Peron, C., Javed, F., and Romanos, G.E. (2017). Immediate loading of tantalum-based implants in fresh extraction sockets in patient with sjogren syndrome: a case report and literature review. *Implant Dentistry* 26: 634–638.

Part V

Potential Future Solutions

25

Zirconia as a Viable Implant Material in Implant Dentistry

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Current Problems in Implant Dentistry

There have been over five decades of successful implant dentistry. Starting in the sixties, the material of choice was and continues to be titanium and titanium alloys. Dental implants simply revolutionized dentistry and as time went by dental implants evolved micro and macroscopically. Thread design and surface enhancements all contributed to successful and predictable implantation. The improvements continued with the alloying providing implants with improved biomechanical performance and stability. All these improvements and developments led to implants being adopted and used at an exponential rate; teeth replacement with dental implants became more widespread with increasingly diverse and divergent outcomes. One of the emerging concerns with dental implants among others was a rising trend toward degradation of periimplant tissues which initially was mostly explained as a result of systemic health issues or poor oral hygiene. Then reports came about with regard to some patients being sensitive to the alloy components of the implants, a fact that has been very well investigated and established in orthopedics. It has also been well documented that just like orthopedic devices, dental implants are vulnerable to corrosion and release in the process alloy components which can lead to a wide range of health issues including peri-implant inflammation and bone loss. This has led to the search for alternative materials started in the sixties by Prof. Sami Sandhaus who investigated ceramics (Alumina) as implant materials. In the mid to late nineties, many of the initial challenges with ceramics as implant materials were solved; today there is a growing popularity and demand especially

from patients for metal-free teeth replacement solutions and an increasing number of manufacturers are adding ceramic implants to their portfolio. The trend recently is pointing toward a fast adoption of zirconia implants; however, the few questions that remain in the minds of many clinicians are among others can zirconia be regarded as a viable implant material in implant dentistry, does zirconia have the same micro- and macroscopic features as their metal alloy counterparts. In this chapter, we will present and discuss the evolution and challenges faced by metals (especially the complications due to peri-implant diseases) and how ceramics are living up or offering to address those challenges.

Corrosion

Corrosion is a phenomenon that is characterized by the gradual degradation of metals by chemical and/or electrochemical reaction with their environment. The type of corrosion observed with dental implants falls under the category of wet corrosion since they are placed and function within the oral cavity which is a wet environment with saliva and bodily fluids acting as electrolytes. Altogether implant corrosion is an electromechanical event most specifically with regards to restored dental implants. Given the number of dissimilar alloys used in implant prosthetic components, the most prominent factor of corrosion on restored implants in the oral cavity is galvanism. The use of fluoride-containing oral hygiene products has also been found to have an impact on the corrosion behavior of implants and higher incidence of corrosion have been reported with increased anion concentrations [1]. There are two major factors in the breakdown of titanium alloys: first, the environment in which they are placed and second the material's chemical and physical properties. In the sixties, Ferguson et al. first documented elevated titanium levels in peri-implant tissues and 30 years later Bumgardner and co-workers reported that there was increased release of metal ions from implants because of galvanic couples correlated with increased implant and peri-implant tissue breakdown [2, 3].

Furthermore, it is well known that diet, lifestyle, and oral habits greatly influence the pH level of the oral cavity thus the implant's reaction to the environment within which it is placed. Another factor in implant corrosion is electrical since upon prosthetic rehabilitation implants are connected to components such as abutments and screws, which alloys are dissimilar to that of the implants themselves. This results in coupling between the implants and the prosthetic components resulting in a galvanic reaction where the implant generally is the anode and releases ions in the peri-implant tissues. It has been found and proven that titanium and various alloying elements leach into the crevicular space around implants [4]. Cortada et al. used the inductively coupled plasma-atomic emission spectroscopy (ICP-AES) technique to confirm the release of metallic ions as a result of titanium implant galvanism [5]. Other factors such as scratching of the implant surface, wear, fretting, erosion, cracking, mechanical stress, and material fatigue have been documented to accelerate or at the very least facilitate the corrosion process on dental implants (Figure 25.1) [6, 7]. Titanium, like other active-passive metals when exposed to oxygen, is covered by a protective oxide layer of TiO₂; even though TiO₂ is thermodynamically stable it remains vulnerable to chemical and mechanical challenges which result in the release of alloy elements through a passive dissolution process.

Figure 25.1 Tribo-Corrosion on the implant platform and prosthesis.

Clinical Significance of Corrosion

Although titanium alloys have better corrosion resistance properties compared to Co–Cr and other implant materials, their corrosion leads not only to the dissolution of titanium but also to other alloys in their composition, such as aluminum, vanadium, chromium, etc. Kirkpatrick et al. in an in vitro study isolated and demonstrated the complex sequence of events at the biomaterial-tissue interface. In the study, they evaluated the modulation role and influence of specific metal ions and corrosion products in the function of the participating cell types. They investigated and presented the mechanism and pathophysiology of impaired wound healing around dental and maxillofacial implants and their impact on healing and maintaining peri-implant health [8]. Galvanic corrosion in dental implants manifests itself in two ways; first through biological effects, such as soft tissue discoloration, pigmentation, and localized inflammatory reaction to the leached alloy components, and secondly by the loss of bone as a result of osteolysis. The osteoclastic activity increases in presence of inflammatory mediators. The release of these alloy elements and metal ions induces a host response that can be limited to the peri-implant tissues or extend to distant organs and even the brain in the case of aluminum. The leached metal ions may induce potentially osteolytic cytokines into tissues leading to implant loosening and may even cause severe allergic reactions or hypersensitivity. There have also been reports in the literature of tumors and malignancies related to metal ions released from dental implants [9].

Osteolysis and Bone Loss

Olmedo et al. demonstrated that corrosion-induced ionic leaching could be responsible for peri-implantitis and implant failure. The particles that are leached as a result of the corrosion process are phagocytized by macrophages as part of host response release mediators of inflammation in the form of cytokines through the inflammatory cascade. These cytokines interfere with the production of osteoblasts thereby causing an elevated osteoclastic activity which leads to peripheral osteolysis and eventual loosening of the implant. Cytotoxic tissue response to corrosion release of several elements from various titanium alloys used in dentistry has been widely investigated and documented [10]. Furthermore, ions released because of corrosion have been demonstrated to contribute to peri-implant bone loss by inducing localized pain and inflammation without the presence of infection around the implant, this interesting phenomenon is called aseptic osteolysis. Osteolysis takes place because of the phagocyte cascade activation when osteoclastic activity is increased [11, 12] but without the presence of infection. Several studies have further shown that the cellular uptake of hexavalent

chromium is many folds greater than the trivalent chromium ion and its increased uptake causes a reduction of the alkaline phosphatase activity of the osteoblastic cells [13]. Furthermore, there have been reports of periimplant carcinogenic and mutagenic activity associated with these metal ions release [14].

Peri-implantitis

Articles on the prevalence of peri-implant diseases showed that 90% of peri-implant tissues had some form of inflammatory response and a prevalence of peri-implantitis from 28 to 51% according to a recent paper [15]. Peri-implantitis is an inflammatory process around an osseointegrated implant, including soft tissue inflammation and progressive loss of supporting bone beyond biological bone remodeling. This inflammatory process is the leading cause of implant failures in the long term. Peri-implantitis results from biofilm dysbiosis deposits surrounding the periimplant mucosa, meaning that bacterial deposition is essential to develop the inflammatory reaction. In addition, research on bacterial adhesion on titanium has revealed that titanium corrosion increases with plaque accumulation. On this matter, zirconia implants have been suggested as a peri-implant mucosa-friendly alternative. Recent studies show intriguing results promoting the advantage of zirconia implants in this matter regarding biofilm formation and bacterial adhesion on the surface of zirconia implants. An in vitro study comparing biofilm formation on zirconia versus titanium disks using threespecies biofilm or human plaque samples in an anaerobic flow chamber mode found that zirconia showed a lesser biofilm thickness and mass compared with titanium [16]. However, the biofilm metabolism was similar for both materials. These results suggest that zirconia implants accumulate less plaque therefore leading to less periimplant inflammation compared to titanium implants. With regards to the bacterial adhesion on zirconia, the Grossner–Schreiber group found that bacterial counts were higher on titanium discs compared to zirconia, while Scarano et al. showed that bacterial adhesion was significantly higher with pure titanium surfaces as compared with zirconium dioxide surfaces [17]. Similar data by Sadid-Zadeh and collaborators also supported these findings. Moreover, these results are also supported in animal models [18]. A study comparing ligature-induced periimplantitis around zirconia and titanium implants in dogs found that the first presented less crestal peri-implant bone loss and no implant failure. Whereas the titanium implant group showed greater levels of bone loss and one implant failure due to peri-implantitis [19]. Additionally, a recent systematic review of 15 studies with meta-analysis indicated that zirconia accumulated less initial oral biofilm parameters thanks to the lesser roughness of the zirconia surface [20]. On the other hand, taking into consideration the role of host response in peri-implantitis, a recent study compared the expression of immunoinflammatory markers in the tissues around ceramic and titanium implants. The authors found a similar number of inflammatory cells (T- and B-cells) in mucosa surrounding ceramic implants when compared with titanium ones, concluding that the cellular response to the inflammatory challenge provoked by the biofilm around implants is the same, independent of the material with which the implant is made [21]. Besides, the hypothesis has been raised in the literature that not only biofilm is responsible for peri-implantitis around titanium implants, but also foreign body reaction, allergies, cement excess, or even metallosis. Considering all the factors discussed above, peri-implantitis is multifactorial and primarily a clinical manifestation of a complex inflammatory response. However, it could be assumed that zirconia implants present a lower incidence of peri-implantitis when compared with titanium implants. Furthermore, when loosening occurs with ceramic implants, it is mostly aseptic, possibly pointing to a different mechanism of de-osseointegration. Nevertheless, according to a systematic review and meta-analysis, owing to the heterogeneity of assessment and short-term evaluation of zirconia implants in clinical studies, results are found to be inconclusive with regards to inflammatory markers in zirconia peri-implant mucosa [22]. On the other hand, one prospective study that followed up zirconia implants for 7.8 years found no difference between teeth (control group) and implants regarding the bleeding index, attachment level, or bacterial colonization. Interestingly, zirconia implants presented less plaque accumulation and recession, but greater probing depths when compared with natural dentition. Moreover, another systematic review concluded that zirconia implants had less plaque accumulation and less inflammation around the peri-implant mucosa [21–23].

Immune Reactions to Metal Alloy Implants

Cellular and humoral immunity are the principal pathway mechanisms of the immune system. The immune system response and inflammation require well-engaged and active biochemical processes to restore homeostasis, which ultimately leads to osseointegration of the implant. The immune response has critical factors such as cytokines and soluble substances produced by different kinds of immunocompetent cells whereby, they affect each other. Up to this date, there are several studies carried out in the field of implant rejection from the immunologic viewpoint and the alterations in the peri-implant environment.

Cellular Immunity

Poor osseointegration and chronic inflammation are two primary and interrelated factors that cause periimplantitis. Infiltration of immune cells is an important component of the immune response that significantly impacts the biocompatibility of dental implants and can lead to implant failure or the absence of osseointegration. The initial injury of peri-implant tissue triggers an inflammatory response mediated by the cells of innate immunity, such as macrophages, dendritic cells (DC), mast cells, and neutrophils [24].

Humoral Immunity

Most of the interrelation between cells of the immune system is modulated by several factors such as cytokines, growth factors, and hormones. Cytokines have an undeniable role in the intercommunication of the immune cells in the inflammatory cascade, which manifest itself clinically. Cytokines are messengers in cell-to-cell interaction that are critical for the pathogenic process of peri-implantitis.

TNF- α is a proinflammatory cytokine and is at the beginning of almost every immune response based on the individual's sensitization to titanium. Thus, TNF-α plays a key role in the failure of implants especially in the case of genetic incompatibility. During the function, metal particles are released from the implant as a result of wear and fatigue, they mostly accumulate in the peri-implant tissues causing an increase in proinflammatory activity of nonspecific immune cells (tissue macrophages and monocytes), as a result of the exposure to particulate debris, i.e. Ti particles. It is known that such particles (diameter $1-10 \mu m$) are consistently released into the environment surrounding implants as a result of mechanical abrasion, chemical, bacterial, and galvanic corrosion which as mentioned above promote hyperinflammatory conditions. Sterner et al. investigated the effects of clinically relevant aluminum ceramic, zirconium ceramic, and titanium particles of varying size and concentration on TNF release in a human macrophage system. They suggested that analysis of cytokines in the peri-implant crevicular fluid (PICF) may be a good indicator to estimate the prognosis of dental implant treatment [25]. Several studies confirmed the negative role of proinflammatory cytokines on the longevity of dental implants; therefore, measuring these cytokine levels, especially IL-1*β*, TNF-ɑ, and IL-6, seems more reliable to predict the fate of implants [26].

Allergies/Metal Hypersensitivity

The scientific literature has for the longest reported on the success of titanium implants; however, in the last ten years, the amount of research and clinical reports on

peri-implantitis and titanium sensitivity has increased significantly. A general search of the literature containing the word "peri-implantitis" on PubMed between 2012 and 2022 returned 2995 results and for the first half of 2022, 345 results were returned. This is reflective of the seriousness of the peri-implantitis problem (Figure 25.2).

With regards to metal sensitivity, it has not been directly correlated with peri-implantitis but appears to have some common causative factors such as release of ions which in the case of dental implants is a result of corrosion. Dental research and literature on metal hypersensitivity related to dental implants have also been gaining momentum, between 2006 and 2022 there were 83 results on the subject (Figure 25.3) and in the last four years 37 publications were published on the problem (Figure 25.4).

Nevertheless, the tribo-corrosion process releases titanium ions into the surrounding tissues that trigger a cascade of reactions that can be localized, in distant organs or even systemic reactions. As demand grows for implantbased dental treatments, the range of implants available on the market is expanding exponentially [27]. When it comes to product development, the choice of implant material, surface treatment, and post-manufacturing cleaning techniques, and packaging are key factors that will characterize the finished product. However, competition between brands is tough, a situation that sadly sometimes places business interests and cost-cutting before quality control. Duddeck et al. showed greater carbon contamination, possibly related to cleaning procedures after surface treatments, which affect the protein and cell adsorption process [28]. Likewise, Massaro et al. mention that the presence of Na, K, and Cl contaminants, occasionally detected by X-ray Photoelectron Spectroscopy (XPS) and High Vacuum X-ray Energy Dispersive Microanalysis (HV-EDX) on implant surfaces, may be residues of proprietary cleaning treatments performed after sandblasting. The biological role of these contaminants remains unclear [29]. Various tests for allergy to titanium are described in the literature, the patch test being the most frequently used [30]. There are other modalities to assess or predict the likelihood of sensitivity to certain metals; the MELISA test is one that uses lymphocytes' response to metals and assists with an 85% accuracy in determining the likelihood an individual will be sensitive to a given metal. The scientific literature on this topic is very scarce, which highlights the need to establish a protocol for those patients who are sensitized to titanium before and after the placement of dental implants. In patients with a history of metal allergies, it is advisable to perform an allergy test and preferably the implant panel from the MELISA test. The alloy and its purity must be identified together with any trace elements it may contain in order to optimize diagnosis. When dental

Figure 25.2 Trends in the Peri-implantitis literature. (a) Last 10 year data, (b) 2022 (till date).

Figure 25.3 Dental implant metal hypersenstivity [2006–2022].

implants are placed in the jaws, and the patient eventually proves allergic to titanium or its alloys, the option of explanation must be considered, basing the decision on the ratio of risk/benefit to the patient. After having removed titanium implants, if the patient shows no clinical improvement, the option of removing other metal prostheses from

the organism should also be considered. In this context, zirconium dioxide implants $(ZrO₂)$ offer a promising alternative to titanium as no case of allergy to zirconium oxide has ever been reported. Nevertheless, in animal experiments, zirconia implants have been shown to release negligible non-reactive particles [27].

Figure 25.4 Dental implant metal hypersentivity [2017–2022].

Zirconia as an Implantable Material

Over the past few decades, dental implant procedures were applied to support single crowns as well as fixed or removable partial or full dentures. Early osseointegration is a critical factor for the clinical success of oral implants. Titanium has shown a high biocompatibility, resistance to corrosion, and favorable mechanical properties, these characteristics are particularly relevant in the early osseointegration phase of the healing period. Nevertheless, with the popularity and the exponential number of implants placed, failures of titanium implants have become quite common. To improve titanium surfaces and enhance osseointegration, several methods have been used; the most commonly used method is to coat the surface of a titanium implant with a non-loading bioceramic, such as hydroxyapatite. Zirconia was first introduced to implant dentistry in the form of coatings to improve osseointegration of the titanium implants. As it was further investigated, the unique characteristics of zirconia such as its superior toughness, strength, corrosion resistance, and good aesthetic value made it an ideal implant material [16, 30, 32–36]. Once, material scientists, clinicians, and patients realized that the accumulation of titanium particles was detectable in tissues close to the implants, local lymph nodes, and even distant organs, they began to reconsider their view of titanium as the ultimate, viable, and biocompatible implant material. Reports of sensitivity and allergy resulting from the placement of titanium implants have been on the rise [36–39]. As of now, there are no reports on the toxicity of zirconia implants and its tooth-like color offers the possibility of staining zirconia thereby elevating the aesthetic scores and patient satisfaction. The successful use of zirconia as a material for dental implants also lies in

its excellent osseointegration. In vivo studies have shown that zirconia osseointegration is on par with that of titanium with regard to cell metabolism and positive tissue response under immediate and delayed loading conditions [39–43]. Furthermore, the inflammatory response and bone loss around zirconia implants are less intense than that induced by titanium implants, suggesting its superior biocompatibility [44]. In the quest to achieve more predictable osseointegration, especially in difficult clinical sites, researchers have attempted to modify zirconia surfaces to enhance bone cell attachment, growth, proliferation, and differentiation. Various methods of surface enhancement of zirconia implants have been investigated and reported for zirconia, some protocols have been reported as preclinical results and others are commercially available. Most of the surface modification procedures studied promoted superior cell responses and qualitative osseo- and muco-integration [45, 46].

Manufacturing Processes of Zirconia

It should be noted that zirconium, like titanium in its earthen state, is also a metal. However, they both belong to the group of transitional metals that when exposed to oxygen under controlled conditions and proprietary protocols produce the ceramic zirconium dioxide which is also known as zirconia. This process is not reversible therefore zirconium can be formed from the zirconia and reversal to the metal state and properties is not possible. However, not all zirconium dioxides are the same. There are major differences in the manufacturing process, powder selection, surface design, and handling of individual systems [46].

Whether its tetragonal zirconia polycrystals (TZP), tetragonal zirconia polycrystals-Alumina (TZP-A), or Alumina toughened zirconia (ATZ), processing of the starting material is important. Grain size, purity, and density have a decisive influence on hardness, stability, and quality. Although very recently 3D printing of ceramics has appeared, hard machining and injection molding are the two main processes for the manufacture of commercially available zirconia implants [47]. Zirconia at 100% responds poorly to humidity and will age prematurely under such conditions. Therefore, modern ceramic implants consist of partially stabilized tetragonal zirconia polycrystals (TZP), which are polycrystalline ceramic composites and consist of zirconia, yttria, and alumina; depending on the manufacturer these components vary in percentages with good mechanical properties and have an average bending strength of 1100MPa. To increase the bending strength to 1200MPa, 0.5% by volume of aluminum oxide (alumina) is added to the starting powders (TZP-A). Zirconia being hydrophobic, most manufacturers add Yttria (Yttrium dioxide) to the composite to stop the low-temperature aging process caused by hydrothermal degradation [48]. In the case of ATZ ceramics, manufacturers will increase the volume percentage of alumina to 20% thereby obtaining a hybrid polycrystalline ceramic that can achieve flexural strength up to 2000MPa. This ATZ-ceramic significantly reduces the risk of fracture. The aging process (phase transformation) from tetragonal to monoclinic $(t \rightarrow m)$ no longer has any clinical relevance thanks to these procedures and the addition of yttria [49].

The state of zirconia implants once the manufacturing is complete is in the tetragonal phase which is maintained with the addition of stabilizers such as yttria. Stabilizers are included to limit the phase transformation of the ceramic which occurs with the ceramic reverting to a less stable and fracture-prone phase also known as monoclinic. However, once the implant is placed, clinicians must be careful with manufacturers' recommendations as phase transformation of the material must be expected when chairside grinding of the zirconia with rotating instruments. Uncontrolled grinding with coarse-grained diamonds and insufficient cooling of the material can induce micro-cracks within the outer surface layer of the material that can propagate and grow into the underlying material structure. However, the stabilizers confer "self-healing" properties to zirconia by volume expansion as a result of phase transformation which effectively helps stop the propagation of mechanically induced micro-cracks. There are however limits to this "self-healing mechanism" of zirconium dioxide because once this unique compensatory capacity has been exceeded, it weakens the zirconia, makes it vulnerable to humidity, and leads to premature aging and ultimately material catastrophic failure [46, 49].

During the early stages of improvement of zirconia implant surfaces, high blasting pressure and large particles were used to roughen the surfaces of ceramic implants. As a result, micro-cracks were found in the surface structure of the implants, which led to fractures under function. A great number of fractures of ceramic implants can be traced back to an improper industrial manufacturing process or the result of poor post-production mechanical handling by the clinician. The processes by which the surface micro-roughness of a zirconia implant is achieved have evolved with the advances in material processing and a better understanding of phase transformation. Today manufacturers are using proprietary acid etching techniques or in the case of injection molded manufacturing, the surface roughness is built into the mold thereby negating the need to grind or use etchants. Some manufacturers claim this is a way to avoid manufacturing-induced contamination of the implant surface [50].

Hard Machining Process

White machining is used to machine zirconia prior to sintering in order to obtain complex profiles such as dental crowns and bridges. It is a dry milling process using tungsten carbide milling tools. This process enables a rapid and cost-effective generation of intricate profiles of zirconia components. However, white machining can also produce extensive surface damage on machined surfaces and induce preload cracks [49, 50].

Hard machining is the most complex and costly way of machining ceramics but also the most reliable and accurate. A block is compressed through Hot Isostatic Post Compaction (HIP) under high pressure (2000 bar) and temperatures of up to 2000 °C. The block is then slowly brought back down to room temperature, sintered, and only then ground to the desired implant shape. Hard machining is performed on sintered zirconia to obtain high precision in tolerance, dimension, and shape using highprecision, high-stiffness, and conventional or high-speed grinding machines [51–53].

Powder Injection Molding

The high price linked with the production of ceramic implants remains a significant deterrent for dental rehabilitation with ceramic implants. As such less expensive manufacturing protocols have been developed.

Powder injection molding (PIM) has been used for a long time in many industries including ceramics. It is a near-net shape method, owing to its advantages such as accuracy

and efficient manufacturing with no need for surface finishing. The implant surface characteristics are embedded in the mold thereby negating the need for postmanufacturing and potentially destructive surface modification. The process of powder injection molding has five stages: (i) the selection of raw materials, (ii) combining the powder with a feedstock binder, which is reused to stabilize and prevent pre-sintering breakage of the green ceramic during production, (iii) feedstock injection molding into the mold using the preferred form, (iv) de-binding to loosen the binder, and (v) sintering to produce the implant. It is critical to assess the structure, shape, particle size, and particle distribution in order to select the yttria-stabilized zirconia powder for the production of feedstock. Binders are used to formulate zirconia parts by ceramic injection molding (CIM). It is additionally essential to understand the melting and degradation temperatures of the binder, to outline the processing conditions for mixing and injection, alongside the removal cycle of the binder (Figure 25.5).

Feedstocks have to be homogeneous, and their preparation is important in the production of defect-free implants. After homogeneous mixing, the feedstock is prepared for injection molding. The parts namely implants and/or abutments are initially in the green stage then they are extracted from the molds and submitted to debinding and sintering. The debinding and sintering thermal cycles are carried out at high temperatures under a controlled atmosphere of argon and hydrogen in order to avoid a stoichiometric change of the zirconia.

The powder injection molding method is suitable to produce one and two-piece ceramic dental implants. A drawback can be that based on the quality of the mold, the roughness level of the produced implant surfaces can be insufficient for optimal bone apposition. Therefore, the surface roughness of CIM-manufactured implants is often enhanced with the post-production application of nanostructured coatings or surface roughening by acid etching or sandblasting. The powder injection molding method is a cost-effective method for manufacturing Yttria-Stabilized Zirconia dental implants that have outstanding biological and mechanical characteristics [50, 54–56].

3D Printing

Using this technology, it is possible to manufacture, through additive manufacturing, highly complexed shape implants in great numbers, which possess good strength and preserve geometry through the manufacturing process [54].

With the development of the lithography-based ceramic manufacturing process (LCM), the 3D printing of these materials is now feasible, and offers some advantages in comparison to standard fabrication procedures. In this context, additive manufacturing offers significant advantages as these procedures can reproduce detailed inner geometries with high accuracy [55, 56]. However, LCM ceramic products are still lagging in terms of translucency and coloring, conversely, 3D printing is unique in that it allows for individual coloring, and customized color gradients can be created since they are manufactured using a layering technology. They however require further clinical and scientific research and evaluation with regard to aging and behavior in the oral environment and occlusal stresses [47, 57, 58] (Figure 25.6).

Figure 25.5 Ceramic injection molding reprinted with reference.

Figure 25.6 Lithography-based ceramic 3D printed implant (Lithoz).

Surface Modification Techniques

The early ceramic implants only had smooth, machined surfaces available. Today with modern and advanced processing methods, rougher ceramic implant surfaces are attainable with minimal effect on the structural integrity of the material. The rougher an implant surface is, the more bone cells can attach to it, and the stronger the bond between implant and bone. Since the volume of the implant itself cannot be increased, the surface of the implants is now increased by "roughening." Surface roughening is important in the colonization of the implant surface by bone-forming cells and the bone-implant contact (BIC) percentages play an important role in reliable, effective, and long-term osseointegration. The preferred methods of commercially available ceramic implant surface roughening are subtractive namely sandblasting, acid etching, laser etching, or a combination [45]. There are no commercially available ceramic implants roughened by addition.

Sandblasting

Sandblasting, which is also known as airborne particle abrasion, produces a surface that has micro-roughness. Various parameters affect the roughness that is present on the implant surface, this includes the size, shape, and kinetic energy of the particles. During the process of sandblasting, particles are projected toward the implant surface and their kinetic energy which depends on their density, volume, and velocity creates irregularities that contribute to the implant surface roughness. The main advantage of sandblasting is that a homogenous and gentle anisotropic abrasion can be obtained on hard materials like ceramic, glass, and silicon. Alumina particles are generally preferred blasting material because of their low cost, hardness, and

needle-like shape. The major disadvantage of using the sandblasting technique is that it may slightly change the surface chemistry because the alumina particles can become embedded in the implant surface and cause contamination but also, they can induce micro-cracks that lead to premature aging, which coalesce and expand once the implant or prosthetic part is placed under functional stresses. Such events can lead to implant fracture and failure [50, 59].

Acid-etching

The process of acid etching is performed with either hydrofluoric acid, nitric acid, or sulphuric acid. Acid-etching treatment can also be used to overcome alumina contamination as it has been proven to remove the alumina residues. This explains why a growing number of manufacturers combine sandblasting and acid etching during the surface roughening of their implants [60, 61]. Heat treatment is done following acid etching to smoothen the sharp edges caused by the etching process. Advantages of acid etching include the homogenous roughening of the material, regardless of its size and shape. This method imposes no risk of delamination as it does not exert much stress on the material. But it might cause chemical changes that are undesirable, thus making this a disadvantage of the process. The topography formed after acid-etching, depends on the prior treatment, composition of the acid mixture, temperature, and the time of exposure to the acid. Acidetching is generally used to generate a micro-scale surface texture, which has the ability to facilitate interlocking between the implant and the bone. Recent studies show that combining sandblasting and acid etching techniques enhances the degree of micro-roughness on zirconia. Such a combination is currently used by some manufacturers to optimize micro-roughness, which would also provide a better material surface for osteoblast cell initial attachment and its proliferation [62–65].

Selective Infiltration Technique

This technique involves coating the surface of the material with a specific infiltration glass and then heating it at a temperature higher than its glass transition temperature. This is followed by the infiltration of molten glass that occurs between the material grains. But this technique can be limited only to a particular extent as only the surface grains joined with the infiltration glass get involved in the process, thereby allowing control over the area required to be treated. Traces of infiltration agent left behind can further be removed by immersion in a solution of 5% hydrofluoric acid, with a nanoscale produced upon rinsing with water. The selective infiltration etching technique is often used to create a nano-porous surface on zirconia implants. The only advantage of this technique is that the actual surface chemistry of the material remains unchanged, and the nanoscale roughness of the surface can be enhanced without losing any material or changing the microscopic surface roughness [66, 67]. However, currently, it is not available on clinically approved ceramic implants.

Polishing

The majority of commercially available ceramic implants are tissue level; therefore, they have a smooth collar that is designed to be transgingival. This area however needs a roughness of a different type. Polishing is a method of surface treatment of the supra-bony area of tissue-level implants, it yields a smoother surface compared to acid etching and sandblasting. Epithelial cells are more likely to adhere to the rough surfaces (acid etching and sandblasting) as compared to smoother (polishing) surfaces, whereas fibroblasts adhere well onto both types of surfaces [67]. This explains the clinical and histological observations of soft tissue adaptation to the transgingival collars of tissuelevel ceramic implants. Polishing of a zirconia surface is performed by using a silicon carbide polishing paper and a diamond or silica suspension using a polishing machine. Mechanical surface treatment that includes polishing, permits a change in the surface topography without modifying the actual surface chemistry. The average surface roughness of a polished zirconia biomaterial is between 8 and 200nm. Polishing also serves to clean the implant surface to a certain extent, along with giving a smooth texture to it. Polishing is mostly done in the transgingival area of tissuelevel ceramic implants and preferably should not be countersunk in bone at the time of implant placement as it may lead to bone loss over time [68, 69].

Laser Treatment

In contrast with sandblasting and acid-etching techniques, laser treatment exerts zero risk of surface contamination as there is no direct contact between the laser and the biomaterial. The laser surface treatments also tend to improve the material wettability by altering the surface properties, which further plays a major role in cell adhesion. Optimal wettability is when the contact angle of a drop of blood with the treated surface is low, this allows the blood and bone-forming cells to spread quickly on the implant surface [70]. The higher the contact angle, the lower the wettability of the material. High wettability improves the protein adsorption and cellular adhesion therefore it is important that the implant surface wettability be elevated to allow optimal protein adsorption with greater and faster bone formation. However, the use of lasers on zirconia ceramic has been reported to disrupt its chemical and mechanical structure with the risk of inducing pre-function cracks and premature aging because of partial transformation to the monoclinic phase [71, 72].

Ultraviolet Light Treatment

Various studies have shown that bone-implant contact of the implants treated with ultraviolet (UV) light was deeply enhanced because of the effect of super-hydrophilicity [73]. A material is described to be super-hydrophilic when the contact angle of the water droplet is less than 5°. Hydrophilicity is one of the key factors involved in the initial interaction with proteins and cells that is beneficial for the early phases of wound healing and osseointegration. When the hydrophilic oxide surface binds to water, hydroxyl (OH−) and oxygen (O²−) groups are formed on the outermost layer. The formation of hydroxylated oxide surfaces improves the surface reactivity with the surrounding ions, amino acids, and proteins in the tissue fluid. As compared to the hydrophobic surfaces, osteoblasts cultured on hydrophilic surfaces have shown to exhibit higher levels of differentiation markers, including alkaline phosphatase and osteocalcin [74, 75]. The effect of hydrophilic surfaces on osseointegration can be supported by improvements in the bone-implant contact and bone anchorage that occurs in the early stages of bone healing. Other than hydrophilicity, the atomic percentage of hydrocarbon changes after UV treatment, according to various studies [76–78].

Coating

Coating of Yttria Stabilized Zirconia (YSZ) with Reinforced Hydroxyapatite (HA) has shown positive in vitro results in the enhancement of adhesive strength and coating stability. Because of their versatility, calcium phosphate {Ca (PO)4} based coatings are generally fabricated using the plasma-spraying techniques in the industry. Despite its numerous drawbacks, such as variation in coating or implant bond strength and modification of hydroxyapatite structure, this technique is said to provide low cost and a high deposition rate. For depositing CP-based coatings, new techniques are constantly being developed in order to address the issues associated with plasma spraying. For an in vitro study, Pardun et al. synthesized a YSZ/HA coating by wet powder spraying (WPS) using a double-action airbrush spray. However, the long-term stability of these coatings has not yet been determined [79–86].

Biofunctionalization

Biomimetic surface modification, also called biofunctionalization, involves the immobilization of biomolecules on the surface to change their biochemical properties and biological responses. Biofunctionalization also allows the anchorage of organic components such as proteins, enzymes, and peptides on the implant surface in order to take control of the implant tissue interface, thereby determining the type of tissue that develops. Arginine–glycine– aspartate (RGD) is commonly used as an adhesive peptide. Many other adhesive proteins are found to possess RGD as their cell recognition site, including fibronectin, fibrinogen, and collagen. These RGD sequences are identified using at least one of the integrins. Adhesion proteins and integrins make a pair to provide cell anchorage, differentiation, and growth signal. RGD peptide has shown to be successfully immobilized on the Y-TZP with this method and thereby enhancing the biocompatibility of the material as well as cell attachment to its surface [87–89].

Self-assembly

An autonomous process by which the components are organized into patterns or structures without any external intervention is called self-assembly. Self-assembled monolayers (SAMs) are formed by the process of solution deposition i.e. the immersion of a particular substrate into the solution of an active surfactant in a particular solvent which may either be organic or aqueous, or the solid surface vapor deposition of an active organic compound, or by aerosol spraying. The driving force for self-assembly is generally the specific interaction between the head group of the surfactant and substrate surface. Most of the surfactants consist of three distinctive parts namely, the surface active head group which binds strongly to the surface, the terminal group which is located at the monolayer surface shows the interfacial properties of the assembly, and the alkane chain which serves as a linker between the head and the terminal groups and facilitates the packing of the molecules in the monolayer with the van der Waals interactions between adjacent methylene groups that orient and stabilize the monolayer. Therefore, by carefully composing the mixture of substrate, SAM solution, and subsequent terminal functionalization, a multitude of subsequent molecule adhesion is feasible [90–95].

Fracture Resistance of Zirconia Implants

Since the beginning of the 2000s, the clinical performance of commercially available zirconia implants has significantly improved. From a mechanical perspective, one-piece designs may be superior to two-piece systems, due to increased fracture resistance and reduced susceptibility for low-grade temperature degradation. In addition, the absence of a microgap between the assembled implant and abutment reduces the potential of hosting bacteria, thus preventing peri-implant inflammation and bone resorption. However, from a practitioner's perspective, there are multiple surgical and prosthodontic shortcomings associated with a one-piece system, as the transmucosal part of the implant cannot be detached. As such, submerged implant healing is not possible, and there are also limited routes to compensate for positional challenges or errors with implants with the provisional and final restoration [96]. In cases of positional challenges, wherein in situ grinding of the zirconia abutment becomes unavoidable, fracture resistance of the implant and osseointegration are potentially impacted due to the vibration produced and the cracks created at the surface of the implant. Several two-piece zirconia implants are being commercially marketed and clinically utilized wherein the implant–abutment attachment is usually achieved through cementing or screwing the abutment and the crown to the endosseous implant. Although cement can seal the microgaps and initial adjustment of implant angulation is possible, the processes are mostly irreversible, and therefore, limited options for replacement of restorations over the implant exist. In contrast, ceramic implants restored by screw retention are still utilizing titanium screws (Figure 25.7) for the most part. The Zeramex XT implant was the first and continues to be the only ceramic implant that has a metal-free

Figure 25.7 Camlog with titanium screw.

Figure 25.8 Zeramex with peek screw.

screw available (Figure 25.8). Previous in vitro studies have noted that the biomechanical stability of both one-piece and two-piece zirconia implants could withstand a loading duration of 10 million cycles at 95 N force. Although the bending moment to fracture (BMF) was unaffected by loading conditions, the BMF of zirconia implants (onepiece and two-piece) were significantly less than that of the control Group [97].

Furthermore, Spies et al. investigated the long-term stability of a commercially available two-piece zirconia implant under the same chewing simulator used in Reimer's study. Spies et al. agreed with Riemer's findings that two-piece zirconia implants are capable of withstanding masticatory forces for several years [98]. In a metaanalysis by Roehling et al., one of 510 zirconia implants fractured (0.20%) suggested that fracture resistance is like that of Ti implants. Fracture rate increases with the diameter of zirconia implants below 3.25mm [99].

Biological Outcome of Zirconia Implants

Muco-integration

Zirconia and titanium elicit different responses from the peri-implant soft tissue. Soft tissue around zirconia abutments and implants is consistently more healthy, better vascularized, and more keratinized with minimal to no visible inflammation [100]. Histologically the collagen fibers surrounding zirconia implants are oblique in a similar fashion to natural teeth. Studies have investigated this

phenomenon and it appears that vascularization and the topography of the ceramic surface contribute greatly [101]. Kajiwara et al. conducted a clinical study where they observed and compared the vascular pattern around zirconia and titanium abutments [102]. They used microcirculation dynamics and thermography and the soft tissue around the implant abutments was evaluated with twodimensional laser speckle imaging and a thermograph. The blood flow within soft tissue around natural teeth was also measured to correct for differences among the subjects. The results showed that the blood flow was significantly higher in the zirconia abutment group and that the metal abutments had nearly 20% lower blood flow in comparison with natural teeth. These results suggest that blood flow around zirconia is similar to that of soft tissue around natural teeth; In a 7.8-year clinical follow-up report of onepiece ceramic implants on 28 patients, Lorenz et al evaluated multiple parameters namely approximal plaque index, sulcus bleeding index, probing pocket depth, probing attachment level, and creeping or recession of the mucosa/ gingiva were analyzed [23]. Changes in the marginal bone level were evaluated by comparing radiographs recorded immediately after implant placement and at the follow-up investigation. Furthermore, the pink esthetic score, periotest values, microbial colonization of the implant/dental sulcus fluid, and patient satisfaction were collected and analyzed. The results showed that compared to natural teeth adhesion of plaque was lower on the implants and creeping attachment was superior; furthermore, the soluble biotinylated-collagen assay (SBA) and periodontal attachment loss were comparable to natural teeth. These short- and medium-term findings explain the consistently superior quality of soft tissue around ceramic implants and abutments (Figure 25.9) [33, 100, 103].

Figure 25.9 Soft tissue healing around zirconia implants after three months of healing.

Osseointegration

Thirty years of translational research in zirconia ceramics has led to significant improvements in the biomedical field especially in dental Implantology. Oral implants made of Yttria-tetragonal zirconia polycrystals (Y-TZP) because of their excellent mechanical properties, good biocompatibility, and aesthetically acceptable color have emerged as an attractive metal-free alternative to titanium implants. Multiple studies in vitro, in vivo on humans and animals have proven that ceramic implants osseointegrate as well as titanium implants and some reports have shown BIC (bone-to-implant contact) values comparable to or even higher than that of metal implants [45, 104].

Today ceramic implant research continues, and they are fast becoming a solution in an ever-growing range of clinical situations. The area of ceramic implant that seems to be of great focus is the surface modification of the infra-bony area of the implants. Ceramic implants unlike titanium do not benefit from additive coating, therefore they are not coated and osseointegration takes place directly on the surface of the implant material. Early ceramic implants were milled with very smooth and glassy surfaces and their osseointegration was poor. Sandblasting soon came to offer roughness but the downside was that the blasting process induced cracks in the implants making them prone to fracture when placed in function and most were made of 100% alumina or zirconia as ceramic composites were initially not widely available for clinical use in dentistry. Today surface modification protocols are mainly acid etching for roughness and other manufacturers have used laser etching but this modality is also technique sensitive and fraught with risk for zirconia as it can induce cracks and lead to implant fractures [45]. Kubasiewicz-Ross and coworkers in an animal model evaluated three surface textures of zirconia implant surfaces. The surfaces studied were blasted (M1), etched (M2), and blasted/etched (M3) against Ti and Zr controls and evaluated the bone-to-implant contact (BIC) percentages. It was found that the zirconia acid-etched surface had the highest BIC percentage proving that zirconia ceramics will integrate into bone for effective tooth replacement [105, 106].

Although the mechanisms behind early or late ceramic implant failures remain to be investigated, the literature available today allows us to say that there is no leaching of ions from zirconia implants. However, the implants themselves after exposure to a permanently wet environment compounded with mechanical stresses have been known to undergo aging during which the implant or at least some areas of the implant undergo a transition in structure from tetragonal to monoclinic. It is now known what percentage of transformation is necessary to induce implant failures. In vitro studies have demonstrated that over 60 years of

Figure 25.10 Osseointegration after five years of zirconia implant placement.

aging would be necessary to have a complete structural failure of the material [107]. Most of the failures with ceramic implants are early loss or lack of osseointegration and for loosening of the implants is aseptic with inflammation but absence of infection. Furthermore, a direct correlation between aging and loss of osseointegration has not been established. However, based on 10 years of clinical observation by the authors, barring prosthetic misfit or abuse, the loss or failure to integrate for zirconia ceramic implants tends to be early or soon after placement.

It appears that since ceramic implants do not release toxic compounds or ions that can interfere with osteogenesis and osseointegration, attention should be turned toward the bone and the recipient's systemic health. There are known factors such as hyperglycemia, elevated cholesterol, micronutrient deficiencies (D3, K2, C, etc.), and low RBC-Mg (red blood cell magnesium) to name a few that have an impact on bone healing and remodeling. The authors have been able to observe a 20% decrease in peri-implant bone failure after systemic optimization of some of the above-listed factors prior to implant placement (Figure 25.10) [104, 107, 108].

Market Growth with Zirconia Implants

The global dental implant market in 2016 was approximately US\$3.8 billion. Between 2018 and 2024, it is anticipated to grow at a compound annual growth rate (CAGR) of 7.7% to nearly US\$6.8 billion ([https://www.grandviewresearch.](https://www.grandviewresearch.com/industry-analysis/dental-implants-market)

Figure 25.11 Market Share (2021) on dental implants.

[com/industry-analysis/dental-implants-market](https://www.grandviewresearch.com/industry-analysis/dental-implants-market)). In 2016, titanium implants dominated the dental implant market with a portion of approximately 92.5%. The remaining 7.5% of the share was distributed among implants fabricated out of zirconia and other materials. Although not as large as the titanium, the zirconia implant market is estimated to be worth over US\$0.4 billion by the end of 2027, with a CAGR of 6.1% [\(https://www.futurewiseresearch.com/](https://www.futurewiseresearch.com/healthcare-market-research/Zirconia-Implant-Market/4254) [healthcare-market-research/Zirconia-Implant-Market/4254\)](https://www.futurewiseresearch.com/healthcare-market-research/Zirconia-Implant-Market/4254). According to www.absolutereports.com the global market for zirconia implants in 2021 was 4.84% (Figure 25.11).

These data show that the market of zirconia implants is small in comparison to the titanium implant market at present, but it is growing rapidly with a CAGR almost identical to that of titanium alloy implants. As of today, there are over twenty ceramic implant manufacturers around the world and in 2023 five new ceramic implant systems were launched

References

- **1** Nakagawa, M., Matsuya, S., Shirasishi, T., and Ohta, M. (1997). Effect of fluoride concentration and pH on corrosion behavior of titanium for dental use. *Journal of Dental Research 78*: 1568–1572.
- **2** Ferguson, S.J., Langhoff, J.D., Voelter, K. et al. (2008). Biomechanical comparison of different surface modifications for dental implants. *The International Journal of Oral & Maxillofacial Implants 23* (6): 1037.
- **3** Bhola, R., Bhola, S.M., Mishra, B., and Olson, D.L. (2011). Corrosion in titanium dental implants/prostheses – a review. *Biomaterials and Artificial Organs 25* (1): 34–46.
- **4** Soares, F.M.S., Elias, C.N., Monteiro, E.S. et al. (2021). Galvanic corrosion of Ti dental implants coupled to CoCrMo prosthetic component. *International Journal of Biomaterials 2021*: 1313343, 11 pages.
- **5** Cortada, M., Giner, L., Costa, S. et al. (1997). Metallic ion release in artificial saliva of titanium oral implants coupled

globally. Zirconia oral implants are of particular interest to our patients because they can positively support an esthetic outcome of reconstruction due to their tooth-like color.

Conclusion

Titanium, being a biomaterial of choice, has been and is still largely employed in the fabrication of dental implants. However, its disadvantages related to peri-implantitis, aesthetics, immunology, and corrosion are still looming and of increased concern. Zirconia is proving to be a viable alternative in replacing titanium. Although the trend has been changing over the last five years, unfortunately, the demand for ceramic implants remains driven by the secondary consumers (patients) who in the majority of cases originate the requests for ceramic implants and inform their dentists on the viability of this excellent metal-free alternative. The need for substantial background data stemming from long-term clinical trials is of paramount importance for zirconia dental implants. There also needs to be evaluation studies and protocol standards so as to have comparable study outcomes that can contribute to developing even more effective clinical guidelines for clinicians. However, the number of clinicians who can attest over a decade of evidence of success for zirconia implants has never stopped growing. The ceramic implantology community has been proactive and such endeavors are already and continuously being developed and improved by the various industryspecific groups in North and South America and Europe. This will give zirconia even more solid ground to stand as a flagbearer in this new chapter in dental implantology.

with different metal superstructures. *Bio-medical Materials and Engineering 7*: 213–220.

- **6** Alrabeah, G.O., Knowles, J.C., and Petridis, H. (2018). Reduction of tribocorrosion products when using the platform-switching concept. *Journal of Dental Research* 97 (9): 995–1002.
- **7** Sansone, V., Pagani, D., and Melato, M. (2013). The effects on bone cells of metal ions released from orthopaedic implants. A review. *Clinical Cases in Mineral and Bone Metabolism* 10 (1): 34.
- **8** Kirkpatrick, C.J., Barth, S., Gerdes, T. et al. (2002). Pathomechanisms of impaired wound healing by metallic corrosion products. *Mund-, Kiefer-und Gesichtschirurgie: MKG 6* (3): 183–190.
- **9** Jané-Salas, E., López-López, J., Roselló-Llabrés, X. et al. (2012). Relationship between oral cancer and implants: clinical cases and systematic literature review. *Medicina Oral, Patología Oral y Cirugía Bucal 17* (1): e23–e28.
- **10** Olmedo, D.G., Tasat, D.R., Duffó, G. et al. (2009). The issue of corrosion in dental implants: a review. *Acta Odontológica Latinoamericana 22* (1): 3–9.
- **11** Rogers, S.D., Howie, D.W., Graves, S.E. et al. (1997). In vitro human monocyte response to wear particles of titanium alloys containing niobium and vanadium. *Journal of Bone and Joint Surgery 79* (B): 311–315.
- **12** Debetto, P. and Luciani, S. (1988). Toxic effect of chromium on cellular metabolism. *Science of the Total Environment 71*: 365–377.
- **13** Arillo, A., Melodia, F., and Frache, R. (1987). Reduction of hexavalent chromium by mitochondria: methodological implications and possible mechanisms. *Ecotoxicology and Environmental Safety 14*: 164–177.
- **14** Cohen, M.D., Kargacin, B., Klein, C.B., and Costa, M. (1993). Mechanism of chromium carcinogenicity and toxicity. *Critical Reviews in Toxicology 23*: 255–281.
- **15** Schwarz, F., Derks, J., Monje, A., and Wang, H.L. (2018). Peri-implantitis. *Journal of Clinical Periodontology 45*: S246–S266.
- **16** Scarano, A., Piattelli, M., Caputi, S. et al. (2004). Bacterial adhesion on commercially pure titanium and zirconium oxide disks: an in vivo human study. *Journal of Periodontology 75* (2): 292–296.
- **17** Größner-Schreiber, B., Teichmann, J., Hannig, M. et al. (2009). Modified implant surfaces show different biofilm compositions under in vivo conditions. *Clinical Oral Implants Research 20* (8): 817–826.
- **18** Sadid-Zadeh, R., Willis, J., Forgo, G., and Haraszthy, V. (2020). Comparative analysis of biofilm formation on materials used for the fabrication of implant-supported prostheses. *Brazilian Dental Journal 31*: 380–384.
- **19** Roehling, S., Gahlert, M., Janner, S. et al. (2019). Ligatureinduced peri-implant bone loss around loaded zirconia and titanium implants. *International Journal of Oral and Maxillofacial Implants 34* (2): 357–365.
- **20** Kniha, K., Heussen, N., Modabber, A. et al. (2021). The effect of zirconia and titanium surfaces on biofilm formation and on host-derived immunological parameters. *International Journal of Oral and Maxillofacial Surgery 50* (10): 1361–1374.
- **21** Serichetaphongse, P., Chengprapakorn, W., Thongmeearkom, S., and Pimkhaokham, A. (2020). Immunohistochemical assessment of the peri-implant soft tissue around different abutment materials: a human study. *Clinical Implant Dentistry and Related Research 22* (5): 638–646.
- **22** Webber, L.P., Chan, H.L., and Wang, H.L. (2021). Will zirconia implants replace titanium implants? *Applied Sciences 11* (15): 6776.
- **23** Lorenz, J., Giulini, N., Hölscher, W. et al. (2019). Prospective controlled clinical study investigating

long-term clinical parameters, patient satisfaction, and microbial contamination of zirconia implants. *Clinical Implant Dentistry and Related Research 21* (2): 263–271.

- **24** FDA, U (2019). *Biological Responses to Metal Implants*, 1–143. *Food and Drug Administration*.
- **25** Sterner, T., Schütze, N., Saxler, G. et al. (2004). Effects of clinically relevant alumina ceramic, zirconia ceramic and titanium particles of different sizes and concentrations on TNF-alpha release in a human macrophage cell line. *Biomedizinische Technik. Biomedical Engineering 49* (12): 340–344.
- **26** Lechner, J., Noumbissi, S., and von Baehr, V. (2018). Titanium implants and silent inflammation in jawbone – a critical interplay of dissolved titanium particles and cytokines TNF-α and RANTES/CCL5 on overall health? *The EPMA Journal 9* (3): 331–343.
- **27** Siddiqi, A., Payne, A.G., De Silva, R.K., and Duncan, W.J. (2011). Titanium allergy: could it affect dental implant integration? *Clinical Oral Implants Research 22* (7): 673–680.
- **28** Duddeck, D., Iranpour, S., Derman, M. et al. (2013). Surface characteristics and quality of implants in sterile packaging. *European Journal for Dental Implantologists 1* (9): 48–58.
- **29** Massaro, C., Rotolo, P., De Riccardis, F. et al. (2002). Comparative investigation of the surface properties of commercial titanium dental implants. Part I: chemical composition. *Journal of Materials Science: Materials in Medicine 13* (6): 535–548.
- **30** Schalock, P.C., Menné, T., Johansen, J.D. et al. (2012). Hypersensitivity reactions to metallic implants– diagnostic algorithm and suggested patch test series for clinical use. *Contact Dermatitis 66* (1): 4–19.
- **31** Yoshinari, M. (2020). Future prospects of zirconia for oral implants – a review. *Dental Materials Journal 39* (1): 37–45.
- **32** Branemark, P.I., Chien, S., Grondahl, H.G., and Robinson, K. (2005). *The Osseointegration Book-From Calvarium to Calcaneus*. Batavia, IL: Quintessence Publishing Co Ltd.
- **33** Kniha, K., Kniha, H., Grunert, I. et al. (2019). Esthetic evaluation of maxillary single-tooth zirconia implants in the esthetic zone. *International Journal of Periodontics and Restorative Dentistry 39* (5): e195–e201.
- **34** Albrektsson, T., Chrcanovic, B., Mölne, J., and Wennerberg, A. (2018). Foreign body reactions, marginal bone loss and allergies in relation to titanium implants. *European Journal of Oral Implantology 11*: S37–S46.
- **35** Lugowski, S.J., Smith, D.C., McHugh, A.D., and Van Loon, J.C. (1991). Release of metal ions from dental implant materials in vivo: determination of Al, Co, Cr, Mo, Ni, V, and Ti in organ tissue. *Journal of Biomedical Materials Research 25* (12): 1443–1458.
- **36** Mombelli, A., Hashim, D., and Cionca, N. (2018). What is the impact of titanium particles and biocorrosion on implant survival and complications? A critical review. *Clinical Oral Implants Research 29*: 37–53.
- **37** Noumbissi, S., Scarano, A., and Gupta, S. (2019). A literature review study on atomic ions dissolution of titanium and its alloys in implant dentistry. *Materials 12* (3): 368.
- **38** Fretwurst, T., Nelson, K., Tarnow, D.P. et al. (2018). Is metal particle release associated with peri-implant bone destruction? An emerging concept. *Journal of Dental Research 97* (3): 259–265.
- **39** Degidi, M., Artese, L., Scarano, A. et al. (2006). Inflammatory infiltrate, microvessel density, nitric oxide synthase expression, vascular endothelial growth factor expression, and proliferative activity in peri-implant soft tissues around titanium and zirconium oxide healing caps. *Journal of Periodontology 77* (1): 73–80.
- **40** Andreiotelli, M., Wenz, H.J., and Kohal, R.J. (2009). Are ceramic implants a viable alternative to titanium implants? A systematic literature review. *Clinical Oral Implants Research 20*: 32–47.
- **41** Derks, J. and Tomasi, C. (2015). Peri-implant health and disease. A systematic review of current epidemiology. *Journal of Clinical Periodontology 42*: S158–S171.
- **42** Moraschini, V., Poubel, L.D.C., Ferreira, V.F., and dos Sp Barboza, E. (2015). Evaluation of survival and success rates of dental implants reported in longitudinal studies with a follow-up period of at least 10 years: a systematic review. *International Journal of Oral and Maxillofacial Surgery 44* (3): 377–388.
- **43** Lalor, P.A., Revell, P.A., Gray, A.B. et al. (1991). Sensitivity to titanium. A cause of implant failure? *The Journal of Bone and Joint Surgery. British Volume 73* (1): 25–28.
- **44** Daubert, D.M., Weinstein, B.F., Bordin, S. et al. (2015). Prevalence and predictive factors for peri-implant disease and implant failure: a cross-sectional analysis. *Journal of Periodontology 86* (3): 337–347.
- **45** Gupta, S., Noumbissi, S., and Kunrath, M.F. (2020). Nano modified zirconia dental implants: advances and the frontiers for rapid osseointegration. *Medical Devices & Sensors 3* (3): e10076.
- **46** Hisbergues, M., Vendeville, S., and Vendeville, P. (2009). Zirconia: established facts and perspectives for a biomaterial in dental implantology. *Journal of Biomedical Materials Research Part B: Applied Biomaterials: An Official Journal of The Society for Biomaterials, The Japanese Society for Biomaterials, and The Australian Society for Biomaterials and the Korean Society for Biomaterials 88* (2): 519–529.
- **47** Osman, R.B., van der Veen, A.J., Huiberts, D. et al. (2017). 3D-printing zirconia implants; a dream or a reality? An in-vitro study evaluating the dimensional accuracy, surface topography and mechanical properties of printed

zirconia implant and discs. *Journal of the Mechanical Behavior of Biomedical Materials 75*: 521–528.

- **48** Nistor, L., Grădinaru, M., Rîcă, R. et al. (2019). Zirconia use in dentistry-manufacturing and properties. *Current Health Sciences Journal 45* (1): 28.
- **49** Miyazaki, T., Nakamura, T., Matsumura, H. et al. (2013). Current status of zirconia restoration. *Journal of Prosthodontic Research 57* (4): 236–261.
- **50** Alao, A.R., Stoll, R., Song, X.F. et al. (2017). Surface quality of yttria-stabilized tetragonal zirconia polycrystal in CAD/CAM milling, sintering, polishing and sandblasting processes. *Journal of the Mechanical Behavior of Biomedical Materials 65*: 102–116.
- **51** Yin, L. and Huang, H. (2004). Ceramic response to high speed grinding. *Machining Science and Technology 8* (1): 21–37.
- **52** Shih, A.J., Scattergood, R.O., Curry, A.C. et al. (2003). Cost-effective grinding of zirconia using the dense vitreous bond silicon carbide wheel. *Journal of Manufacturing Science and Engineering 125* (2): 297–303.
- **53** Shih, A.J., Grant, M.B., Yonushonis, T.M. et al. (1998). Vitreous bond CBN wheel for high speed grinding of zirconia and M2 tool steel. *Transactions-north American Manufacturing Research Institution of Sme XXVI*: 195–200.
- **54** Kim, H.K., Woo, K.M., Shon, W.J. et al. (2015). Comparison of peri-implant bone formation around injection-molded and machined surface zirconia implants in rabbit tibiae. *Dental Materials Journal 34* (4): 508–515.
- **55** Dobrzański, L.A. and Matula, G. (2017). Powder injection molding of tool materials and materials containing one-dimensional nanostructural elements. In: Dobrzanski, L.A. (Ed.) *Powder Metallurgy–Fundamentals and Case Studies*, Leszek A Dobrzanski, 223–243. Rijeka, Croatia: *InTech*.
- **56** Ferreira, T.J., Vieira, M.T., Costa, J., and Gato, P.T. (2016). Manufacturing dental implants using powder injection molding. *Journal of Orthodontics & Endodontics 2* (1): 21.
- **57** Gatto, M.L., Groppo, R., Furlani, M. et al. (2022). Lithography-based ceramic manufacturing (LCM) versus milled zirconia blocks under uniaxial compressive loading: an in vitro comparative study. *Journal of Dentistry 116*: 103886.
- **58** Bomze, D. and Ioannidis, A. (2019). 3D-printing of high-strength and bioresorbable ceramics for dental and maxillofacial surgery applications-the LCM process. *Ceramic Applications 7* (1): 38–43.
- **59** Bacchelli, B., Giavaresi, G., Franchi, M. et al. (2009). Influence of a zirconia sandblasting treated surface on peri-implant bone healing: an experimental study in sheep. *Acta Biomaterialia 5* (6): 2246–2257.
- **60** Hempel, U., Hefti, T., Kalbacova, M. et al. (2010). Response of osteoblast-like SAOS-2 cells to zirconia

ceramics with different surface topographies. *Clinical Oral Implants Research 21* (2): 174–181.

- **61** Kohal, R.J., Bächle, M., Att, W. et al. (2013). Osteoblast and bone tissue response to surface modified zirconia and titanium implant materials. *Dental Materials 29* (7): 763–776.
- **62** Iwaya, Y., Machigashira, M., Kanbara, K. et al. (2008). Surface properties and biocompatibility of acid-etched titanium. *Dental Materials Journal 27* (3): 415–421.
- **63** Saulacic, N., Erdösi, R., Bosshardt, D.D. et al. (2014). Acid and alkaline etching of sandblasted zirconia implants: a histomorphometric study in miniature pigs. *Clinical Implant Dentistry and Related Research 16* (3): 313–322.
- **64** Hoshing, U.A., Patil, S., Medha, A., and Bandekar, S.D. (2014). Comparison of shear bond strength of composite resin to enamel surface with laser etching versus acid etching: an in vitro evaluation. *Journal of Conservative Dentistry: JCD 17* (4): 320.
- **65** Langhoff, J.D., Voelter, K., Scharnweber, D. et al. (2008). Comparison of chemically and pharmaceutically modified titanium and zirconia implant surfaces in dentistry: a study in sheep. *International Journal of Oral and Maxillofacial Surgery 37* (12): 1125–1132.
- **66** Aboushelib, M.N., Kleverlaan, C.J., and Feilzer, A.J. (2007). Selective infiltration-etching technique for a strong and durable bond of resin cements to zirconiabased materials. *The Journal of Prosthetic Dentistry 98* (5): 379–388.
- **67** Aboushelib, M.N., Osman, E., Jansen, I. et al. (2013). Influence of a nanoporous zirconia implant surface on cell viability of human osteoblasts. *Journal of Prosthodontics: Implant, Esthetic and Reconstructive Dentistry 22* (3): 190–195.
- **68** Klinge, B. and Meyle, F. (2006). Soft-tissue integration of implants-Consensus report of Working Group 2. *Clinical Oral Implants Research 17* (Suppl. 2): 93–96.
- **69** Kimura, Y., Matsuzaka, K., Yoshinari, M., and Inoue, T. (2012). Initial attachment of human oral keratinocytes cultured on zirconia or titanium. *Dental Materials Journal 31*: 2011-189.
- **70** Hao, L. and Lawrence, J. (2003). Effects of CO2 laser irradiation on the wettability and human skin fibroblast cell response of magnesia partially stabilised zirconia. *Materials Science and Engineering: C 23* (5): 627–639.
- **71** Hao, L., Ma, D.R., Lawrence, J., and Zhu, X. (2005). Enhancing osteoblast functions on a magnesia partially stabilised zirconia bioceramic by means of laser irradiation. *Materials Science and Engineering: C 25* (4): 496–502.
- **72** Ogawa, T. (2012). Ultraviolet photofunctionalization of titanium implants. *Oral & Craniofacial Tissue Engineering 2* (2): 95–102.
- **73** Hsu, S.K., Hsu, H.C., Ho, W.F. et al. (2014). Biomolecular modification of zirconia surfaces for enhanced biocompatibility. *Thin Solid Films 572*: 91–98.
- **74** Sawase, T., Jimbo, R., Baba, K. et al. (2008, 2008). Photo-induced hydrophilicity enhances initial cell behavior and early bone apposition. *Clinical Oral Implants Research 19* (5): 491–496.
- **75** Tengvall, P. and Lundstrom, I. (1992). Physico-chemical considerations of titanium as a biomaterial. *Clinical Materials 9*: 115–134.
- **76** Zhao, G., Schwartz, Z., Wieland, M. et al. (2005). High surface energy enhances cell response to titanium substrate microstructure. *Journal of Biomedical Materials Research Part A: An Official Journal of The Society for Biomaterials, The Japanese Society for Biomaterials, and The Australian Society for Biomaterials and the Korean Society for Biomaterials 74* (1): 49–58.
- **77** Sartoretto, S.C., Alves, A.T.N.N., Resende, R.F.B. et al. (2015). Early osseointegration driven by the surface chemistry and wettability of dental implants. *Journal of Applied Oral Science 23*: 279–287.
- **78** Tuna, T., Wein, M., Swain, M. et al. (2015). Influence of ultraviolet photofunctionalization on the surface characteristics of zirconia-based dental implant materials. *Dental Materials 31* (2): e14–e24.
- **79** Pardun, K., Treccani, L., Volkmann, E. et al. (2015). Characterization of wet powder-sprayed zirconia/ calcium phosphate coating for dental implants. *Clinical Implant Dentistry and Related Research 17* (1): 186–198.
- **80** Herman, H. (1988). Plasma spray deposition processes. *MRS Bulletin 13* (12): 60–67.
- **81** Yang, Y., Kim, K.H., and Ong, J.L. (2005). A review on calcium phosphate coatings produced using a sputtering process – an alternative to plasma spraying. *Biomaterials 26* (3): 327–337.
- **82** Pardun, K., Treccani, L., Volkmann, E. et al. (2015). Mixed zirconia calcium phosphate coatings for dental implants: tailoring coating stability and bioactivity potential. *Materials Science and Engineering: C 48*: 337–346.
- **83** Zhang, B., Kwok, C.T., Cheng, F.T., and Man, H.C. (2011). Fabrication of nano-structured HA/CNT coatings on Ti6Al4V by electrophoretic deposition for biomedical applications. *Journal of Nanoscience and Nanotechnology 11* (12): 10740–10745.
- **84** Sandhyarani, M., Rameshbabu, N., and Venkateswarlu, K. (2014). Fabrication, characterization and in-vitro evaluation of nanostructured zirconia/hydroxyapatite composite film on zirconium. *Surface and Coatings Technology 238*: 58–67.
- **85** Stanic, V., Aldini, N.N., Fini, M. et al. (2002). Osteointegration of bioactive glass-coated zirconia in healthy bone: an in vivo evaluation. *Biomaterials 23* (18): 3833–3841.
- **86** Bosetti, M., Verne, E., Ferraris, M. et al. (2001). In vitro characterisation of zirconia coated by bioactive glass. *Biomaterials 22* (9): 987–994.
- **87** Frandsen, C.J., Noh, K., Brammer, K.S. et al. (2013). Hybrid micro/nano-topography of a TiO2 nanotubecoated commercial zirconia femoral knee implant promotes bone cell adhesion in vitro. *Materials Science and Engineering: C 33* (5): 2752–2756.
- **88** Morra, M. (2006). Biochemical modification of titanium surfaces: peptides and ECM proteins. *European Cells & Materials 12* (1): 15.
- **89** Schwarz, F., Herten, M., Sager, M. et al. (2007). Histological and immunohistochemical analysis of initial and early osseous integration at chemically modified and conventional SLA® titanium implants: preliminary results of a pilot study in dogs. *Clinical Oral Implants Research 18* (4): 481–488.
- **90** Ruoslahti, E. and Pierschbacher, M.D. (1987). New perspectives in cell adhesion: RGD and integrins. *Science 238* (4826): 491–497.
- **91** Whitesides, G.M. and Grzybowski, B. (2002). Selfassembly at all scales. *Science 295* (5564): 2418–2421.
- **92** Love, J.C., Estroff, L.A., Kriebel, J.K. et al. (2005). Self-assembled monolayers of thiolates on metals as a form of nanotechnology. *Chemical Reviews 105* (4): 1103–1170.
- **93** Ulman, A. (1996). Formation and structure of selfassembled monolayers. *Chemical Reviews 96* (4): 1533–1554.
- **94** Mastrangelo, F., Fioravanti, G., Quaresima, R. et al. (2011). Self-assembled monolayers (SAMs): which perspectives in implant dentistry? *Journal of Biomaterials and Nanobiotechnology 2* (05): 533.
- **95** Gupta, S. (2016). A recent updates on zirconia implants: a literature review. *Dental Implants Dentures 1* (113): 18–26.
- **96** Hannink, R.H., Kelly, P.M., and Muddle, B.C. (2000). Transformation toughening in zirconia-containing ceramics. *Journal of the American Ceramic Society 83* (3): 461–487.
- **97** Kohal, R.J. and Dennison, D.K. (2020). Clinical longevity of zirconia implants with the focus on biomechanical and biological outcome. *Current Oral Health Reports 7* (4): 344–351.
- **98** Spies, B.C., Kohal, R.J., Balmer, M. et al. (2017). Evaluation of zirconia-based posterior single crowns supported by zirconia implants: preliminary results of a prospective multicenter study. *Clinical Oral Implants Research 28* (5): 613–619.
- **99** Roehling, S., Schlegel, K.A., Woelfler, H., and Gahlert, M. (2018). Performance and outcome of zirconia dental implants in clinical studies: a meta-analysis. *Clinical Oral Implants Research 29*: 135–153.
- **100** Kniha, K., Schlegel, K.A., Kniha, H. et al. (2018). Evaluation of peri-implant bone levels and soft tissue dimensions around zirconia implants – a three-year follow-up study. *International Journal of Oral and Maxillofacial Surgery 47* (4): 492–498.
- **101** Tetè, S., Mastrangelo, F., Bianchi, A. et al. (2009). Collagen fiber orientation around machined titanium and zirconia dental implant necks: an animal study. *The International Journal of Oral & Maxillofacial Implants 24* (1): 52.
- **102** Kajiwara, N., Masaki, C., Mukaibo, T. et al. (2015). Soft tissue biological response to zirconia and metal implant abutments compared with natural tooth: microcirculation monitoring as a novel bioindicator. *Implant Dentistry 24* (1): 37–41.
- **103** Gahlert, M. and Roehling, S. (2015). Peri-implantitis and ceramic implants: first clinical observations. *Implantologie 23* (3): 305–310.
- **104** Siddiqi, A., Khan, A.S., and Zafar, S. (2017). Thirty years of translational research in zirconia dental implants: a systematic review of the literature. *The Journal of Oral Implantology 43* (4): 314–325.
- **105** Kubasiewicz-Ross, P., Hadzik, J., and Dominiak, M. (2018). Osseointegration of zirconia implants with 3 varying surface textures and a titanium implant: a histological and micro-CT study. *Advances in Clinical and Experimental Medicine 27* (9): 1173–1179.
- **106** Depprich, R., Zipprich, H., Ommerborn, M. et al. (2008). Osseointegration of zirconia implants compared with titanium: an in vivo study. *Head & Face Medicine 4* $(1): 1-8.$
- **107** Choukroun, J., Khoury, G., Khoury, F. et al. (2014). Two neglected biologic risk factors in bone grafting and implantology: high low-density lipoprotein cholesterol and low serum vitamin D. *The Journal of Oral Implantology 40* (1): 110–114.
- **108** Pieralli, S., Kohal, R.J., Hernandez, E.L. et al. (2018). Osseointegration of zirconia dental implants in animal investigations: a systematic review and meta-analysis. *Dental Materials 34* (2): 171–182.

26

Ceramic Implants

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Introduction

Without a doubt, titanium dental implants have been considered the gold standard for tooth replacement since their introduction in the 1960s, demonstrating excellent biocompatibility and offering a wide range of treatment options for patients. Nonetheless, reports on possible unwanted reactions to titanium have increased in recent years. Hypersensitivity and allergic reactions, corrosion, Ti particle release, and unfavorable esthetic results have been reported [1–4]. In addition, the emerging trend of a holistic or metal-free approach to dentistry has prompted patients and clinicians to seek for an alternative to titanium and other metals.

The quest for an alternative to metal implants dates back to the 1930s, when M. Rock applied for a patent on Aluminum Oxide. In 1965, Prof. Sami Sandhaus, considered the father of ceramic implantology, patented a screwshaped dental implant made of alumina powder (Degussit AL23). This would be the first step in a new era in ceramic implantology [5]. Sandhaus continued his investigations with aluminum oxide and introduced the Crystalline Bone Screw (C.B.S) in 1968, with a reported success rate of only 25% after five years [6]. Sandhaus would later commercialize these implants under the brand name CeraSand in Switzerland, but no long-term data exists on their performance [7]. These efforts were followed by Schulte and Heimke in 1976, who introduced the Tübingen implant (Frialit I; Friadent, Mannheim, Germany) for immediate implant placement in the maxillary anterior region (Figure 26.1). A success rate of 92.5% was reported after an investigation period of 10 years [8]. The Bionit system, another aluminum oxide implant, was introduced by

Müller and co-workers in the late 1980s in Germany [9], but little data exists regarding their long-term behavior. In conjunction with the aforementioned efforts with aluminum oxide, single crystal alumina (Sapphire) implants were also investigated as a means for tooth replacement. The BioCeram implant, developed by Kyocera (Kyoto, Japan) and introduced in the 1970s was perhaps the most documented of them all (Figures 26.2 and 26.3). Steflik et al. [10] reported 10-year results with 81% success rates for tooth– implant–supported fixed partial dentures. Fartash and Arvidson [11] reported on the treatment of total or partial edentulism with fixed prosthesis with a follow-up of 10 years, demonstrating a survival rate of 100% of their toothimplant restorations. The same group later reported only implant–supported fixed partial dentures, with an estimated survival rate of 96.6%. The Bioceram implant system was also utilized for the treatment of complete edentulism. Berge and Gronningsaeter [12] reported on 30 patients who had been treated with implant-assisted mandibular overdentures, with a cumulative survival rate of 69% over a period of 11 years. All of the above systems were eventually removed from the market due to poor mechanical properties and unfavorable long-term clinical performance [13].

Zirconia

Introduced in the 1990s as a dental material, its current clinical applications range from posts, frameworks, and abutments to dental implants. Zirconia is white, has excellent biocompatibility, low affinity to plaque, and is able to achieve osseointegration levels comparable to its titanium counterparts [4, 13].

Manufacturing

Zircon is the raw material by which Zirconia is obtained. A mineral belonging to the group of neosilicates, its chemical name is zirconium silicate $(ZrSiO₄)$, and is a source of the metal zirconium (Zr) and the ceramic Zirconia (ZrO₂). About 80% of the world's Zircon mining occurs in Australia and South Africa. To obtain the Zr metal, the material is separated via the *Van Arkel* process. The result is a zirconium (Zr) sponge called the Zirconia Crystal Bar, which can be melted to obtain zirconium rods. Pure zirconium is used in nuclear reactors as containers of nuclear fuel and in combination with other metals to form compounds. Once the metal is obtained, it can undergo a calcination process, by which an oxide is produced. The result is zirconium dioxide or Zirconia $(ZrO₂)$, a ceramic with no metallic properties. Zirconia occurs in three temperature-dependent forms: monoclinic (room temperature to 1170°C), tetragonal $(1170\text{°C}-2370\text{°C})$, and cubic $(2370\text{°C} - \text{up to melting})$ point) [14].

Zirconia enters the *monoclinic phase* after the calcination process of zirconium. At this stage, the material is very fragile and requires stabilization with other compounds to **Figure 26.1** The Tübingen implant. *Source:* SpotImplant. enter the *tetragonal phase* and make it suitable for dental

Figures 26.2–26.3 The BioCeram implants, developed by Kyocera (Kyoto, Japan). Available in blade and root form. *Source:* KYOCERA Corporation.

applications. Ytria and Alumina are most commonly utilized to stabilize zirconia. Zirconia dental implants today are manufactured either with **Y-TZP** (Ytria-stabilized Tetragonal Zirconia Policrystals) or **ATZ** (Alumina-Toughened Zirconia). The manufacturing process occurs via subtractive milling. A zirconia blank is milled to achieve its final macro and microscopic characteristics. This blank is then subjected to a sintering process and Hot Isostatic Pressing (HIP), where a controlled shrinkage occurs by about 15–20%. The implants are then post-processed to achieve the final surface characteristics via sandblasting, acid etching, laser treatment, or a combination of those.

Advancements in manufacturing technology such as the introduction of injection molding for the production of zirconia implants have streamlined the production processes. Injection molding is a novel technology that eliminates the post-processing procedures that take place after sintering. The macro, micro, and nano-characteristics of the implants are all incorporated into a pre-designed mold. The raw material is a ceramic feedstock that is injected at high pressure (2500 PSI) into the molds and then sintered at high temperature for a controlled shrinkage. Since there is no post-processing via sandblasting/acid etching, this process yields an impurity-free surface. Injection molding also provides a reduction in the manufacturing costs, tool wear, and lowers the environmental impact of the manufacturing process (Figure 26.4).

Commercially available Zirconia implants can be one or two-piece. One-piece zirconia implants with an integrated abutment and pre-determined restorative margin have limitations due to their inability to allow for angle correction, require a high degree of surgical precision, and only accept a cementable restoration. Two-piece systems with either cement or screw-retained abutments allow clinicians to broaden their prosthetic options, including angle correction via angled abutments, the ability to deliver screwretained restorations, as well as improved options for soft tissue management. Commercially available two-piece systems can accept either a cementable or screw-retained abutment. Spies and coworkers [15] compared the fracture resistance of cement and screw-retained systems with one-piece implants. Despite the fact that one-piece implants outperformed the two-piece systems, the bending moment values for the two-piece system remained above the maximum values measured in the mouth. The authors concluded that the fracture-resistance values of two-piece systems could withstand physiological bite forces in vivo. The stability of screw retention in two-piece systems was further investigated by Joos et al. [16]. Two-piece zirconia and titanium implants were subjected to artificial aging, including thermal cycling and chewing simulation/ mechanical loading equivalent to five years of function.

Figure 26.4 One and two-piece zirconia implants manufactured via Ceramic Injection Molding. The macro, micro, and nano- characteristics are incorporated into the mold. *Source:* Zinova Zirconia Implant System.

Fracture tests resulted in mean forces well above physiologic thresholds and the stability of the connection between the restoration and the implant seemed comparable for both zirconia and titanium implants, making them suitable for clinical application (Figures 26.5–26.12).

Osseointegration

There is no doubt that zirconia has the same capacity for osseointegration as titanium. Histological studies have shown direct osseous integration without any connective tissue formation at the bone–implant interface. Gahlert et al. compared the bone tissue responses to surfacemodified zirconia and titanium implants in pigs. Histological evaluation showed osseous integration for both materials at 4, 8, and 12 weeks, with no statistically significant differences between the 2 groups at any time period. Similarly, there were no differences found with respect to peri-implant bone density or bone-to-implant contact ratios between the two [17, 18]. Akagawa et al. [19] evaluated loaded and unloaded zirconia dental implants in monkeys. Histology analysis performed three months after implant placement demonstrated direct bone apposition to the implant in both treatment groups. The bone-to-implant contact ratio was 81.9% for the non-loaded implants and 69.8% for the loaded group. The authors reported absence of mobility and no fractures occurred during the observation period. These findings have been corroborated in recent clinical trials in humans. One-piece zirconia implants have been evaluated with up to 10 years followup. Gahlert et al. [20] reported on the performance of onepiece Y-TZP zirconia implants in single tooth gaps. Three months after implant placement, provisional restorations

Figures 26.5–26.12 Extraction of hopeless tooth #8 and immediate implant placement utilizing a two-piece zirconia implant. *Source:* Institut Straumann AG.

were delivered, followed by final prosthesis at six months. Crestal bone levels were measured at placement, six months and one year. Twelve months after surgery, the mean bone level change was 1.02mm (0.90mm SD). The implant survival and success rates after 12 months were 97.6%. In a prospective cohort investigation, Balmer et al. [21] evaluated the clinical and radiological outcomes of one-piece zirconia implants restored with single crowns (SCs) or fixed dental prostheses (FDPs) over an observation period of five years in function. About 71 one-piece implants were placed in 60 patients with all but 3 implants placed in PM or M sites. About 49 implants were restored as single crowns and 22 as 3-unit fixed dental prosthesis. Only one implant was lost five weeks after implantation and the mean marginal bone loss at the five-year follow-up was 0.7mm (SD 0.6mm). The overall success and survival rate was 98.4%. Ferrario et al. [22] presented at the 28th Annual Scientific Meeting of the European Association for Osseointegration on 10-year follow-ups of edentulous rehabilitation with zirconia implants. About 25 one-piece zirconia implants were placed in 10 patients. At the end of the observation period, the average crestal bone loss was 0.81mm (SD 2.4mm). No implant failure was reported. The osseointegration of ceramic implants, much like that of titanium, is dependent on the surface characteristics. The removal torque of machined zirconia implants is significantly lower when compared to roughened zirconia [23–25]. Similarly, a comparative study with sandblasted or machined zirconia implants and titanium implants in the maxilla of miniature pigs bone apposition was enhanced by the roughened zirconia surface [26]. Clinical data demonstrates that Zirconia implants have equal capacity for osseointegration as titanium and they are a suitable alternative material as a means for tooth replacement.

Soft Tissue Integration

Although osseointegration has been a primary focus in optimizing the success of dental implants, there has been an emerging emphasis on the importance of mucointegration and the peri-implant soft-tissue barrier for their long-term maintenance and success. In that regard, zirconia implants have already shown excellent osseointegration capability and biocompatibility in comparison to other implant materials, while exhibiting significantly improved color response of the peri-implant mucosa, indicating not only a better esthetic outcome but potentially healthier peri-implant soft-tissues. Muco-integration could prove zirconia implants to be capable of forming a long-term softtissue barrier that promotes the health of these implants, reinforcing the rising demand for metal-free options for

tooth replacement. It is well established that the quality of the peri-implant mucosal environment is highly dependent on the surface characteristics of the implants. The proliferation and spread of gingival fibroblasts appear to be faster on zirconia compared to titanium, [27] and it has been suggested that the wettability of zirconia could promote protein adsorption and the attachment and spreading of fibroblasts [28]. Welander et al. [29] investigated the dimensions of the peri-implant mucosa around zirconia and titanium abutments. After an observation period of five months, the length of the epithelium remained at 1.75mm in the zirconia group, with a lower presence of leukocytes compared to titanium. Their study suggested that zirconia could enhance the epithelial attachment during the healing phase. When compared to titanium implants, zirconia implants were found to exhibit a shallower probing depth in monkeys [30]. Similarly, an animal study comparing one-piece zirconia and titanium implants found a higher content of collagen and shorter length of the sulcular epithelium in zirconia implants, suggesting an improvement in the soft tissue seal and reduction of the peri-implant inflammatory infiltrate [31]. Furthermore, it is well-known that ceramic materials are less prone to bacterial biofilm and plaque accumulation than metal substrates [32, 33], suggesting that zirconia implants could offer not only a healthier peri-implant environment, but also one that is more resistant to inflammation and infection (Figures 26.13 and 26.14).

Figure 26.13 Soft Tissue integration around a one-piece zirconia implant. *Source:* Institut Straumann AG.

Figure 26.14 Soft Tissue integration around a two-piece zirconia implant. *Source:* Z5-BL, Z Systems AG, Oensingen, Switzerland.

Long-term Maintenance

The peri-implant environment plays a crucial role in the long-term maintenance and success of dental implants. Inflammation of the peri-implant mucosa can lead to periimplantitis if left untreated. Similarly, there is a direct causeeffect relationship between bacterial plaque accumulation and peri-implantitis [34–36]. Zirconia 's low surface energy and wettability results in a reduction of the adhesion capacity of bacteria [37]. In an in vivo experiment on bacterial adhesion, zirconia disks showed less accumulation of bacteria compared with titanium. The same study showed that the bacterial plaque growing on zirconia surfaces was less

References

- **1** Depprich, R., Naujoks, C., Ommerborn, M. et al. (2014). Current findings regarding Zirconia Implants. *Clinical Implant Dentistry and Related Research* 16 (1): 124–137.
- **2** Sicilia, A., Cuesta, S., Coma, G. et al. (2008). Titanium Allergy in dental implant patients: a clinical study on 1500 consecutive cases. *Clinical Oral Implants Research* 19 (8): 823–835.
- **3** Siddiqi, A., Milne, T., Cullinan, M.P., and Seymour, G.J. (2016). Analysis of P. gingivalis, T. forsythia and S. aureus levels in edentulous mouths prior to and 6 months after placement of one – piece zirconia and titanium implants. *Clinical Oral Implants Research* 27: 288–294.
- **4** Cionca, N., Hashim, D., and Mombelli, A. (2017). Zirconia dental implants: where are we now, and where are we heading? *Periodontology 2000* 73 (1): 241–258.
- **5** Rock, M. (1933). Künstliche Ersatzteile für das Innere und Aussere des menschliche und tierische Körpers. German Patent DRP 583.589.

mature compared with the bacterial plaque growing on titanium [32]. These findings were corroborated by Scarano and coworkers [33]. In a ligature-induced peri-implantitis study comparing Ti and Zr implants, Roehling et al. [38] demonstrated a more favorable hard and soft tissue response to the disease. Zirconia implants showed a statistically significant reduction in the active and spontaneous progression of the disease, as well as a reduction of the crestal bone loss. This tissue-friendly environment observed around zirconia is also evidenced by an increased microcirculation in the periimplant soft tissues compared to titanium and an ability to form a mucosal seal [29, 39]. Altogether, zirconia provides a unique peri-implant environment that closely resembles that of a natural tooth and the ability to outperform titanium implants in the long term.

Conclusions

Zirconia implants have proven to be a reliable alternative to titanium as a means for tooth replacement both in vitro and in vivo. It is clear that zirconia has the same capacity for osseointegration as titanium, as well as the ability to withstand occlusal forces. It's superior biocompatibility, low affinity to plaque accumulation, and increased resistance to peri-implantitis provides a tissue-friendly environment that positively impacts their survival, success, and long-term maintenance. Zirconia implants are proving to be a game changer in implant dentistry, potentially replacing titanium implants in the future.

- **6** Strub, J.R., Rohner, D., and Schärer, P. (1987). Die Versorgung des Lückengebißes mit implantatgetragenen Brücken. Eine Longitudinalstudie über 7,5 Jahre. *Z Zahnärztl Implantol* 3: 242-254.
- **7** Sandhaus, S. (1987). The Cerasand endosseous implant (In French). *Actualités Odonto-Stomatologiques* 41: 607–626.
- **8** D'Hoedt, B. (1986). 10 Jahre Tübinger Implantat aus Frialit. Eine Zwischenauswertung der Implantatdatei. *Z Zahnärztl Implantol* 2: 6–10.
- **9** Müller, W., Piesold, J., and Glien, W. (1988). Eigenschaften und klinische Anwendung von Kieferimplantaten aus Aluminium- oxidkeramik Bionit. *Stomatologie der DDR* 38: 673–678.
- **10** Steflik, D.E., Koth, D.L., Robinson, F.G. et al. (1995). Prospective investigation of the single-crystal sapphire endosteal dental implant in humans: tenyear results. *The Journal of Oral Implantology* 21: 8–18.
- **11** Fartash, B. and Arvidson, K. (1997). Long-term evaluation of single crystal sapphire implants as abutments in fixed prosthodontics. *Clinical Oral Implants Research* 8: 58–67.
- **12** Berge, T.I. and Gronningsaeter, A.G. (2000). Survival of single crystal sapphire implants supporting mandibular overdentures. *Clinical Oral Implants Research* 11: 154–162.
- **13** Kohal, R.J., Att, W., Bachle, M., and Butz, F. (2008). Ceramic abutments and ceramic oral implants. An update. *Periodontology 2000* 47: 224–243.
- **14** Denry, I. and Kelly, J. (2008). State of the art zirconia for dental applications. *Dental Materials* 24: 299–307.
- **15** Spies, B.C., Nold, J., Vach, K., and Kohal, R.-J. (2016). Two-piece zirconia oral implants withstand masticatory loads: an investigation in the artificial mouth. *Journal of the Mechanical Behavior of Biomedical Materials* 53: 1–10.
- **16** Joos, M., Sailer, I., Filippi, A. et al. (2020). Stability of screw-retention in two-piece zirconia implants: an in vitro study. *Clinical Oral Implants Research* 31 (7): 607–614.
- **17** Gahlert, M., Röhling, S., Wieland, M. et al. (2009). Osseointegration of zirconia and titanium dental implants: a histological and histomorphometrical study in the maxilla of pigs. *Clinical Oral Implants Research* 20: 1247–1253.
- **18** Gahlert, M., Roehling, S., Sprecher, C.M. et al. (2012). In vivo performance of zirconia and titanium implants: a histomorphometric study in mini pig maxillae. *Clinical Oral Implants Research* 23: 281–286.
- **19** Akagawa, Y., Ichikawa, Y., Nikai, H., and Tsuru, H. (1993). Interface histology of unloaded and early loaded partially stabilized zirconia endosseous implant in initial bone healing. *The Journal of Prosthetic Dentistry* 69: 599–604.
- **20** Gahlert, M., Kniha, H., Weingart, D. et al. (2016). A prospective clinical study to evaluate the performance of zirconium dioxide dental implants in single- tooth gaps. *Clinical Oral Implants Research* 27: e176–e184.
- **21** Balmer, M., Spies, B.C., Kohal, R.J. et al. (2020). Zirconia implants restored with single crowns or fixed dental prostheses: 5-year results of a prospective cohort investigation. *Clinical Oral Implants Research* 31 (5): 452–462.
- **22** Ferrario, S., Borgonovo, A.E., Dino, R., and Maiorana, C. (2019). Edentulous rehabilitation with zirconia dental implants – 10 years follow-up. *Clinical Oral Implants Research* 30: 470. [https://doi.org/10.1111/](https://doi.org/10.1111/clr.425_13509) [clr.425_13509](https://doi.org/10.1111/clr.425_13509).
- **23** Sennerby, L., Dasmah, A., Larsson, B., and Iverhed, M. (2005). Bone tissue responses to surface- modified zirconia implants: a histomorphometric and removal torque study in the rabbit. *Clinical Implant Dentistry and Related Research* 7: S13–S20.
- **24** Schliephake, H., Hefti, T., Schlottig, F. et al. (2010). Mechanical anchorage and peri-implant bone formation of surface-modified zirconia in minipigs. *Journal of Clinical Periodontology* 37: 818–828.
- **25** Hoffmann, O., Angelov, N., Zafiropoulos, G.-G., and Andreana, S. (2012). Osseointegration of zirconia implants with different surface characteristics: an evaluation in rabbits. *International Journal of Oral and Maxillofacial Implants* 27: 352–358.
- **26** Gahlert, M., Gudehus, T., Eichhorn, S. et al. (2007). Biomechanical and histomorphometric comparison between zirconia implants with varying surface textures and a titanium implant in the maxilla of miniature pigs. *Clinical Oral Implants Research* 18: 662–668.
- **27** Rompen, E., Domken, O., Degidi, M. et al. (2006). The effect of material characteristics, of surface topography and of implant components and connections on soft tissue integration: a literature review. *Clinical Oral Implants Research* 17: 55–67.
- **28** Noro, A., Kaneko, M., Murata, I., and Yoshinari, M. (2013). Influence of surface topography and surface physicochemistry on wettability of zirconia (tetragonal zirconia polycrystal). *Journal of Biomedical Materials Research. Part B, Applied Biomaterials* 101: 355–363.
- **29** Welander, M., Abrahamsson, I., and Berglundh, T. (2008). The mucosal barrier at implant abutments of different materials. *Clinical Oral Implants Research* 19: 635–641.
- **30** Kohal, R.J., Wolkewitz, M., and Mueller, C. (2010). Alumina-reinforced zirconia implants: survival rate and fracture strength in a masticatory simulation trial. *Clinical Oral Implants Research* 21: 1345–1352.
- **31** Linares, A., Grize, L., Munoz, F. et al. (2016). Histological assessment of hard and soft tissues surrounding a novel ceramic implant: a pilot study in the minipig. *Journal of Clinical Periodontology* 43: 538–546.
- **32** Rimondini, L., Cerroni, L., Carrassi, A., and Torricelli, P. (2002). Bacterial colonization of zirconia ceramic surfaces: an in vitro and in vivo study. *The International Journal of Oral & Maxillofacial Implants* 17: 793–798.
- **33** Scarano, A., Piattelli, M., Caputi, S. et al. (2004). Bacterial adhesion on commercially pure titanium and zirconium oxide disks: an in vivo human study. *Journal of Periodontology* 75: 292–296.
- **34** Meyer, S., Giannopoulou, C., Courvoisier, D. et al. (2016). Experimental mucositis and experimental gingivitis in persons aged 70 or over. Clinical and biological responses. *Clinical Oral Implants Research* 28: 1005–1012.
- **35** Salvi, G.E., Aglietta, M., Eick, S. et al. (2012). Reversibility of experimental peri-implant mucositis compared with experimental gingivitis in humans. *Clinical Oral Implants Research* 23: 182–190.

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- **36** Pontoriero, R., Tonelli, M., Carnevale, G. et al. (1994). Experimentally induced peri-implant mucositis. A clinical study in humans. *Clinical Oral Implants Research* 5: 254–259.
- **37** Al-Radha, A.S.D., Dymock, D., Younes, C., and O'Sullivan, D. (2012). Surface properties of titanium and zirconia dental implant materials and their effect on bacterial adhesion. *Journal of Dentistry* 40: 146–153.
- **38** Roehling, S., Gahlert, M., Janner, S. et al. (2019). Ligature-induced peri-implant bone loss around loaded zirconia and titanium implants. *The International Journal of Oral & Maxillofacial Implants* 34 (2): 357–365.
- **39** Kajiwara, N., Masaki, C., Mukaibo, T. et al. (2015). Soft tissue biological response to zirconia and metal implant abutments compared with natural tooth: microcirculation monitoring as a novel bioindicator. *Implant Dentistry* 24 (1): 37–41.

Photofunctionalization in the Treatment of Peri-implantitis

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The acceptance of oral implantology as the standard of care in oral reconstruction has led to an exponential increase in the use of dental implants by clinicians of all specialties. Concurrent with this increased use of implantable devices has been the increased incidence of periimplantitis [1]. While the causes of peri-implantitis are varied, the one common thread has been alterations in the surface characteristics of implanted devices, and many different treatment modalities have been suggested for treatment of these implant site defects [2]. However, treatment of peri-implantitis can be refractory, with a significant number of treatment attempts failing to achieve the desired results $[3, 4]$.

The conditions leading to the development of periimplantitis are multifactorial, and there is one aspect of peri-implant disease that is not well understood. That parameter is the condition of the implant surface. Metallic oxide surfaces are quite complex, and they exist in a range of electron states that affect the quality of fluid and tissue adhesion [5]. When titanium or zirconium implants are manufactured, the oxide layers that immediately form are pristine and, consequently, extremely hydrophilic and electrostatically active [6]. Within days of manufacture, however, inorganic ions from the atmosphere start to populate the active receptor sites on these oxide layers [7]. Implant surfaces undergo a fundamental change, becoming less electrostatically active and convert to a hydrophobic form (Figure 27.1). The term "biologic aging of titanium" has been coined to describe the time-related degradation of physiochemical properties of implant surfaces, with a direct effect on the ultimate quality and quantity of bone at the implant interface.

This will diminish the degree of protein and fibrin attachment and may affect the quality of osseointegration [8]. (Figures 27.2 and 27.3) A lower quality of fibrin

adhesion may delay cell migration and result in a lower percentage of bone-to-implant contact. Early implant failures may be related to this phenomenon.

In addition, implants that suffer from peri-implant disease are not only contaminated with inorganic ions, but there is now a layer of organic contamination that potentially complicates treatment outcomes. Strategies to treat periimplantitis have previously focused on removal of organic bioburden and decontamination. We now recognize that treatment of the oxide surface at the molecular and atomic levels is now becoming an integral part of the treatment protocol to ensure reintegration of the implant body.

A critical factor in implant integration is biocompatibility, and successful integration and survival of dental implants in function are directly related to the percentage of bone-to-implant contact. It is reported that percentage of vital bone in successfully integrated implants ranges from 45 to 65% [9]. Early failure of dental implants is most often related to insufficient load-bearing bone, resulting in breakdown and resorption of the remaining bone complex. Therefore, biocompatibility becomes the most important factor in forming the highest percentage of vital bone in direct apposition to the implant surface.

There are several physiochemical properties that contribute to biocompatibility, which includes surface topography, hydrophilicity, and chemical composition [10]. Morra et al. commenced a study of 34 different implant systems and analyzed hydrocarbon deposition on titanium oxide surfaces. They found a range of 17.9–76.5% carbon deposition [11]. This hydrocarbon contamination existed on all implants tested, regardless of surface characteristics.

Early failures of dental implants may be directly related to a low percentage of bone-to-implant contact. These percentages under normal conditions may range from 50 to 65% [12].

Figure 27.1 Hydrophobic implant surface repelling fibrin attachment. (*Source:* Reprinted courtesy Professor Dr. Nelson R. Pinto, Department of Periodontics and Implant Dentistry, Faculty of Dentistry, University of the Andes, Santiago, Chile).

Surface Characteristics of Metallic Oxide Surfaces

Studies have shown that the electron state of metallic oxide surfaces can affect protein absorption, and osteoblast migration, differentiation, and proliferation.

Surface analysis of implant surfaces can be accomplished by using SEM (Scanning Electron Microscope), AES (Auger Electron Spectroscopy), and XPS (X-ray Photoelectron Spectroscopy). These instruments can perform elemental analysis of the oxide surfaces to measure both morphology and chemistry. Within the titanium oxide or zirconium oxide surfaces, we can detect hydrocarbon contamination of the oxide receptors and ultimately test the affinity of these surfaces for biologic fluids and cell attachment [13]. The primary contaminant is carbon, with smaller percentages of nitrogen, oxygen, aluminum, and fluoride.

In a study by Roy et al. they measured contaminant deposition corresponding to the presence of carbon-containing molecules on implant surfaces. The peak attachment occurred at 285 eV for all species, and then the population of these molecules was measured after UVC irradiation. They found that the percentage of hydrocarbon contamination decreased by a factor of 4X. Another peak at 534 eV was found for contamination by H_20 . Photofunctionalization was found to decrease the $H₂0$ concentration and covert the receptor site to TiOH, resulting in an increase in hydrophilicity by changing the electron charge of the surface [14]. (Figure 27.4) Changing the binding energy (eV) of titanium receptors will result in the uncoupling of hydrocarbon contaminants and conversion to electropositive species that attract biologic fluids. (Figure 27.5)

It is hypothesized that, in the biologic model, additional contamination will occur through the attachment of lipopolysacharrides, bacterial endotoxins, calculus, and dental plaque. Debridement of the implant surface must precede photofunctionalization. This can be accomplished by using ultrasonic instrumentation, chemotherapeutic

Figures 27.2–27.3 Poor quality fibrin adhesion on aged titanium. (*Source:* Reprinted courtesy Professor Dr. Nelson R. Pinto, Department of Periodontics and Implant Dentistry, Faculty of Dentistry, University of the Andes, Santiago, Chile).

Figure 27.4 The core lines recorded for as received and after photofunctionalization samples. The lines in red show the decontamination effect of the UVC irradiation decreasing the hydrocarbons peak (a) and increasing the oxygen peak (b). The red lines demonstrate the decontamination effect of UVC irradiation by decreasing hydrocarbon peaks and increasing oxygen binding. (*Source:* Reprinted courtesy Roy et al. [14]).

Figure 27.5 Comparing the intensity of the peak at 285eV corresponding to carbon contamination and oxygen binding before (a, c) and after (b, d) UVC irradiation. (*Source:* Reprinted courtesy Roy et al. [14]).

agents, citric acid, prophyjet, or ablative lasers [15]. Following removal of the bioburden on the implant surface, UVC energy can then reach the oxide layer [16]. The final removal of organic and hydrocarbon residues results in a change in electrostatic condition, allowing adsorption of organic components in biologic fluid, primarily carboxyl, and amine groups. This early phenomenon now facilitates adhesion and migration of regenerative cells, resulting in integration of the implanted device [17–19] (Figure 27.6).

Osteoblast Attachment and Migration

Adhesion and motility of osteoblasts can be affected by surface properties of implant surfaces [20]. Reduction of the ability of osteoblasts to adhere to the implant surface and reduction of cell spreading across that surface has a direct effect on the production of signaling proteins in the osteoblast. These Rho GTPases help to regulate actin fibers in the osteoblast and are responsible for cell mobility. Any reduction of these signaling proteins will interfere with cell

Figure 27.6 Biochemical changes to aged titanium surfaces following photofunctionalization. (*Source:* Reprinted courtesy Yang Cheng, Department of Stomatology, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, 1277, Jiefang Avenue, Wuhan 430022, Hubei, P.R. China, Tel: +008613517199412).

Figure 27.7 ECM collagen and fibronectin interact with transmembranous integrins. Vinculin binds integrins with actin filaments aiding cellular migration, adhesion, and spreading. (*Source:* Reprinted courtesy Yang Cheng, Department of Stomatology, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, 1277, Jiefang Avenue, Wuhan 430022, Hubei, P.R. China, Tel: +008613517199412).

attachment and spreading. As hydrocarbon contamination increases, there is a linear reduction in cell migration and spreading. Photofunctionalization helps to indirectly stimulate GTPase gene expression, upregulating osteoblast metabolism [21] (Figure 27.7).

Osteoblast Differentiation and Bone Density

Osseointegration is a complex metabolic process, but measurement of certain bone markers can be used to determine the relative rates of early bone production. These markers

Figure 27.8 Comparison of cellular morphology before and after UVC treatment. (*Source:* Reprinted courtesy Gao et al. [23]).

include Col-1, RUNx-2, Osterix, Osteocalcin, Osteopontin, and Alkaline Phosphatase. Maturation of the bone complex can be observed by measuring Alkaline Phosphatase levels (ALP). Elevated ALP levels are directly correlated with osteogenic activity and bone deposition. It can be demonstrated that ALP levels are reduced by up to 50% on aged titanium surfaces as a function of inhibition of osteoblast attachment and reduced metabolic activity. Photofunctionalization enhances protein adsorption, osteoblast adhesion, and proliferation [22] (Figure 27.8).

Electrostatic Condition and Hydrophilicity

While the wettability of an implant surface may not be directly related to bioactivity, in vitro testing shows alterations in bone marker activity [6]. Aged titanium exists in an electronegative state, resulting in a hydrophobic response [24]. When UVC light interacts with the implant oxide layer, the surface is converted to electropositive, enhancing fibrin attachment [13, 25]. The proposed mechanism is the conversion of Ti4+ to Ti3+, resulting in the formation of Ti-OH groups [26]. The result is the conversion of the oxide surface to superhydrophilic, enhancing the adhesion of blood proteins as a precursor to osteoblast attachment (Figure 27.9).

Antibacterial Effect

Bacterial adhesion to transmucosal elements of dental implants is unavoidable. Bacteria use salivary proteins to enhance the colonization on abutment components and may induce an inflammatory response in the periimplant tissue. Chronic inflammatory response degrade

the dentogingival complex leading to even greater bacterial attachment to the implant body. The charged cell surface of bacteria interacts with the oxide surface of implants, enabling colonization of the transmucosal portion of the implant body or the roughened surface of the implant body following bone loss. Photofunctionalization enhances osteogenic adhesion and competes with microbial colonization. This effect appears to be long-lasting following treatment, and fewer relapses are seen on treated implant surfaces [27] (Figure 27.10).

Intraoral Use of Photofunctionalization

All previous research in photofunctionalization (PTF) has been conducted on implants prior to placement and commercial systems are currently available (Figure 27.11).

Elemental analysis of implants before and after UVC treatment can then be measured and correlated with treatment outcomes. The refractive nature of peri-implantitis treatment is now driving research into additional treatment modalities for contaminated implant surfaces to enhance potential regeneration. However, the increased complexity of the type of contaminants found on implant surfaces requires additional instrumentation for PTF to be effective. In the presence of bioburden, UVC application cannot reach the titanium oxide surface if the layer of organic contamination is not removed. Complete ablation of the implant surface prior to PTF is therefore an absolute requirement for conversion of the oxide surface. While several different modalities for surface ablation have been proposed in the literature, hard tissue lasers have shown the greatest promise [28]. The remaining defect must be grafted to prevent reattachment

Figure 27.9 The degree of osteoblast attachment to titanium surfaces as a function of time and surface treatment. (*Source:* Reprinted courtesy Gao et al. [23]).

New research is being conducted using intraoral UVC handpieces. This is the first time PTF has been proposed after implant placement and development of periimplantitis. Treatment of peri-implant defects in situ is showing great promise, particularly in treating refractory cases where previous graft surgery has failed.

Treatment of the infected implant surface requires sequential treatment steps. Unlike the sterile implant prior to placement, the roughened implant surface has bacterial colonization with a bioburden layer that can be difficult to remove. This organic layer must be removed to allow UVC energy to reach the titanium oxide layer.

Prototype handpieces have been designed to deliver UVC energy in the 254nm range. Mercury ray tubes are used to create UVC energy primarily in the 254nm range. They are shielded to direct the energy toward the target tissue and implant body (Figures 27.12a, b).

The first step is full-thickness flaps to expose the implant body. Surgical curettes can be used to remove the gross amounts of granulation tissue. Following complete

Figure 27.10 A summary of the effects of photofunctionalization. (*Source:* Reprinted courtesy Yang Cheng, Department of Stomatology, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, 1277, Jiefang Avenue, Wuhan 430022, Hubei, P.R. China, Tel: +008613517199412).

Dental implant surface conditioning system

Figure 27.11 Commercial UVC treatment system for preplacement treatment of implants and transmucosal elements.

debridement and UVC treatment of the implant surface and remaining bone architecture, the defect is grafted to prevent soft tissue reattachment to the implant body (Figures 27.13–c).

A treatment sequence for the treatment of periimplantitis has been proposed. Proper diagnosis is essential prior to treatment. Implants with advanced bone loss should be removed and the height of bone levels around the remaining implants assessed (Figure 27.14a). Pocket

depths are measured and the amount of keratinized tissue and vestibular depth evaluated (Figure 27.14b). Whenever possible, the prosthesis should be removed. This will allow better access to the implant bodies circumferentially and for grafting following debridement (Figure 27.14c.). The remaining implants can now be evaluated. Mobile implants are removed (Figure 14d) and a strategy to debride and graft the remaining defect is developed. Flaps are better managed with the prosthesis removed and the defects can be fully visualized (Figure 27.14e).

Full-thickness flaps are raised and all gross granulation tissue removed with surgical curettes. An ablative laser can now be used to remove any soft tissue and bioburden adherent to the implant body (Figure 27.14f). This step must be efficiently completed before the final surface treatment. Any visible soft tissue remnants will prevent reintegration of the implant body.

While all visible bioburdens can be removed, the next step is to treat the implant surface at the nano level. Surface ablation may appear to completely clear the implant body of diseased tissue, but the oxide surface will still be contaminated. It is proposed that acid etching can denature any remaining bacterial endotoxins (Figure 27.14g). However, the breakdown products of the surface contaminants are still present. An application of EDTA will remove any smear layer that remains (Figure 27.14h). The final chemotherapeutic step is the application of hydrogen peroxide. This will remove any last tissue remnants or breakdown products of bacterial colonization (Figure 27.14i).

The surface of the implant is now free of any biologic contaminants. The UVC handpiece can now be used to effectively change the electron state of the oxide surface, converting it back to a hydrophilic state (Figure 27.14j). The handpiece is used for 60–90 seconds at proximity.

The treated implant surface is now ready to be grafted. The graft of choice is determined by the defect size and the

Figure 27.12 (a) Prototype UVC handpiece with mercury ray tube. (b) Gated shield directing the UVC energy to the target tissue.

Figure 27.13 (a) Failure of particulate bone graft in peri-implantitis treatment. (b) Graft placement following surface debridement and application of UVC handpiece. (c) Regeneration of peri-implant bone after three months healing.

number of remaining walls. The graft must be stable and protect the space while bone is regenerating. Whenever possible, autologous fibrin is mixed with the graft material to aid in handling and to accelerate the rate of bone regeneration (Figure 27.14k).

If key walls are missing, a longer resorption time collagen membrane is used to protect the graft complex (Figure 27.14l). Some clinicians advocate the use of a final L-PRF membrane to aid in early anastomosis of flap margins (Figure 27.14m). Primary closure is recommended

and placement of cover screws with primary closure may aid in full regeneration of the defect whenever possible.

The defect should be monitored radiographically, and the prosthesis replaced when the regenerated bone is mature (Figure 27.14n).

In early studies, UVC photo functionalization showed great promise in revision surgery for the treatment of periimplantitis. More controlled studies will be necessary to prove the efficacy of this treatment for a very vexing problem in oral implantology.

Figure 27.14 (a) Pre-op radiograph of infected implant bridge. (b) Pre-op photograph demonstrating bone loss and suppuration. (c) Sectioning and removal of implant prosthesis. (d) Removal of mobile implant. (e) Remaining implants to be grafted. (f) Laser ablation of implant surfaces with hard tissue laser. (g) Acid etching to denature bacterial endotoxin. (h) Application of EDTA to remove remaining smear layer. (i) Application of hydrogen peroxide to remove any remnants of bioburden within the defect. (j) UVC treatment to photofunctionalize the implant surface. (k) Corticocancellous graft of defect to abutment level. (l) Resorbable collagen membrane to cover graft area. (m) Final L-PRF membrane to facilitate flap margin anastomosis. (n) three-month post-op radiograph of healing graft complex.

Figure 27.14 (Continued)

References

- **1** Lang, N.P. and Berglundh, T. (2011). Peri-implant diseases: where are we now? consensus of the seventh european workshop on periodontology. *Journal of Clinical Periodontology* 38 (Suppl. 11): 178–181.
- **2** Khoury, F. and Buchmann, R. (2001). Surgical therapy of peri-implant disease: a 3-year follow-up study of cases treated with 3 different techniques of bone regeneration. *Journal of Periodontology* 72: 1498–1508.
- **3** Fransson, C., Wennström, J., and Berglundh, T. (2008). Clinical characteristics at implants with a history of progressive bone loss. *Clinical Oral Implants Research* 19: 142–147.
- **4** Wagner, T.P., Pires, P.R., Rios, F.S. et al. (2021). Surgical and non-surgical debridement for the treatment of periimplantitis: a two-center 12-month randomized trial. *Clinical Oral Investigations* [https://doi.org/10.1007/](https://doi.org/10.1007/s00784-021-03874-z) [s00784-021-03874-z.](https://doi.org/10.1007/s00784-021-03874-z) Online ahead of print. PMID: 33725166.
- **5** Roy, M. and Hędzelek, W. (2014). Photofunctionalization: a new method to bio-activate the titanium implant surface – review of literature. *Journal of Stomatology (Czasopismo Stomatologiczne)* 67: 682–691.
- **6** Zhao, G., Schwartz, Z., Wieland, M. et al. (2005). High surface energy enhances cell response to titanium substrate microstructure. *Journal of Biomedical Materials Research Part A* 74: 49–58. PMID.
- **7** Hori, N., Att, W., Ueno, T., Sato, N., Yamada, M., Saruwatari, L., Suzuki, T., Ogawa, T. (2009). Agedependent degradation of the protein adsorption capacity of titanium. *Journal of Dental Research* 88(7): 663–667. doi: 10.1177/0022034509339567. PMID: 19641155.
- **8** Att, W., Hori, N., Takeuchi, M. et al. (2009). Timedependent degradation of titanium osteoconductivity: an implication of biological aging of implant materials. *Biomaterials* 30: 5352–5363. [https://doi.org/10.1016/j.](https://doi.org/10.1016/j.biomaterials.2009.06.040) [biomaterials.2009.06.040.](https://doi.org/10.1016/j.biomaterials.2009.06.040)
- **9** Att, W. and Ogawa, T. (2012). Biological aging of implant surfaces and their restoration with ultraviolet light treatment: a novel understanding of osseointegration. *The International Journal of Oral & Maxillofacial Implants* 27: 753–761.
- **10** Massaro, C., Rotolo, P., De Riccardis, F. et al. (2002). Comparative investigation of the surface properties of commercial titanium dental implants. Part I: chemical composition. *Journal of Materials Science. Materials in Medicine* 13: 535–554.
- **11** Morra, M., Cassinelli, C., Bruzzone, G. et al. (2003). Surface chemistry effects of topographic modification of titanium dental implant surfaces: 1. surface analysis. *The International Journal of Oral & Maxillofacial Implants* 18: 40–45.
- **12** Ogawa, T. and Nishimura, I. (2003). Different bone integration profiles of turned and acid-etched implants associated with modulated expression of extracellular matrix genes. *The International Journal of Oral & Maxillofacial Implants* 18: 200–210.
- **13** Rapuano, B.E. and MacDonald, D.E. (2011). Surface oxide net charge of a titanium alloy; modulation of fibronectinactivated attachment and spreading of osteogenic cells. *Colloids and Surfaces, B: Biointerfaces* 82: 95–103.
- **14** Roy, M., Pompella, A., Kubacki, J. et al. (2016). Photofunctionalization of titanium: an alternative explanation of its chemical- physical mechanism. *PLoS One* 11 (6): e0157481. [https://doi.org/10.1371/journal.](https://doi.org/10.1371/journal.pone) [pone.](https://doi.org/10.1371/journal.pone) 0157481.
- **15** Kamel, M.S., Khosa, A., Tawse-Smith, A., and Leichter, J. (2014). The use of laser therapy for dental implant surface decontamination: a narrative review of in vitro studies. *Lasers in Medical Science* 29 (6): 1977–1985.
- **16** Suzuki, T., Hori, N., Att, W. et al. (2009). Ultraviolet treatment overcomes time-related degrading bioactivity of

titanium. *Tissue Engineering. Part A* 15 (12): 3679–3688. [https://doi.org/10.1089/ten.](https://doi.org/10.1089/ten) TEA. 2008.0568.

- **17** Funato, A., Yamada, M., and Ogawa, T. (2013). Success rate, healing time, and implant stability of photofunctionalized dental implants. *The International Journal of Oral & Maxillofacial Implants* 28 (5): 1261–1271. [https://doi.org/10.11607/jomi.](https://doi.org/10.11607/jomi) 3263.
- **18** Tsukimura, N., Yamada, M., Iwasa, F. et al. (2011). Synergistic effects of UV photofunctionalization and micro-nano hybrid topography on the biological properties of titanium. *Biomaterials* 32: 4358–4368.
- **19** Hori, N., Ueno, T., Suzuki, T. et al. (2010). Ultraviolet light treatment for the restoration of age-related degradation of titanium bioactivity. *The International Journal of Oral & Maxillofacial Implants* 25 (1): 49–62.
- **20** Aita, H., Att, W., Ueno, T. et al. (2009). Ultraviolet light- mediated photofunctionalization of titanium to promote human mesenchymal stem cell migration, attachment, proliferation and differentiation. *Acta Biomaterialia* 5: 3247–3257.
- **21** Hayashi, R., Ueno, T., Migita, S. et al. (2014). Hydrocarbon deposition attenuates osteoblast activity on titanium. *Journal of Dental Research* 93 (7): 698–703.
- **22** Hirakawa, Y., Jimbo, R., Shibata, Y. et al. (2013). Accelerated bone formation on photo-induced hydrophilic titanium implants: an experimental study in the dog mandible. *Clinical Oral Implants Research* 24 (Suppl A100): 139–144. [https://doi.org/10.1111/j.](https://doi.org/10.1111/j.1600-0501.2011.02401) [1600-0501.2011.02401.](https://doi.org/10.1111/j.1600-0501.2011.02401)
- **23** Gao, Y., Liu, Y., Zhou, L. et al. (2013). The effects of different wavelength UV photofunctionalization on micro-arc oxidized titanium. *PLoS ONE* 8 (7): e68086. [https://doi.org/10.1371/journal.pone.0068086.](https://doi.org/10.1371/journal.pone.0068086)
- **24** Iwasa, F., Hori, N., and Ueno, T. (2010). Enhancement of osteoblast adhesion to UV-photofunctionalized titanium via an electrostatic mechanism. *Biomaterials* 31: 2717–2727.
- **25** Rapuano, B.E., Lee, J.J., and MacDonald, D.E. (2012). Titanium alloy surface oxide modulates the conformation of adsorbed fibronectin to enhance it's binding to integrins in osteoblasts. *European Journal of Oral Sciences* 120: 185–194.
- **26** Aita, H., Hori, N., and Takeuchi, M. (2009). The effect of ultraviolet functionalization of titanium on integration with bone. *Biomaterials* 30: 1015–1110.
- **27** Yamada, Y., Yamada, M., Ueda, T., and Sakurai, K. (2014). Reduction of biofilm formation on titanium surface with ultraviolet-C pre- irradiation. *Journal of Biomaterials Applications* 29 (2): 161–171.
- **28** Romanos, G.E., Gupta, B., Yunker, M. et al. (2013). Lasers use in dental implantology. *Implant Dentistry* 22: 282–288.

Improvement of Osseointegration Through Autologous Growth Factors

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Introduction

Dental implants are relatively innovative and superior treatments in dentistry and are widely used in an increasing number of cases. Most of the techniques are evidencebased and predictable. However, in many cases, the implant site is inadequate for poor bone quality and quantity.

For these reasons, tissue regeneration is frequently required in implant dentistry, and autologous platelet concentrates are promising and innovative therapeutic approaches.

Implant osseointegration is a concept widely supported by the literature. In 1990, Zarb and Alberktsson defined osseointegration from a clinical point of view as a rigid and clinically asymptomatic fixation process of an alloplastic material with bone subjected to functional load [1, 2].

Since then a lot is changing and a lot is still to come.

The most important aspects for successful osseointegration are the biological characteristics of the host site (the patient) and the macro- and microstructure of the implant. Dental implant surfaces have now achieved outstanding performances, which were previously unimaginable.

Traditional implant surfaces can be classified into two broad categories: smooth (machined) and rough (treated). It has now been demonstrated that implants with micro-rough surfaces stimulate a greater and faster apposition of new bone than implants with smooth surfaces. The differentiation of osteoblasts and their response during osseointegration varies and depends on the implant surface in its nano- and microtopographic aspects. Various phenomena, such as clot formation, retention of the fibrin texture, and differentiation of the cell population, are influenced by surface topography.

The biomechanical phenomena that arise at the interface between biological tissues and the implant surface as well as the implant–prosthetic material are governed by

the design (external micro and macrostructure), the patient's response, the surgical technique, and by the conditions and/or times of loading. Exposure of the implant surface to air leads to the formation of an oxide layer, which constitutes a benevolent substrate in the interaction with body fluids, the first and fundamental mediator of all biological phenomena. The insertion of the implant and the consequent surgical trauma cause the interruption of the bone blood vessels, with subsequent bleeding: this determines the contact between the biological fluids of the host and the surface of the newly inserted implant. The absorption on the implant surface of ions and macromolecules of blood origin and fibrin is fundamental for the platelet adhesion itself and the consequent osteogenesis. This ensures an extremely high percentage of osseointegration, even in the most complex situations.

On the other hand, this means that the margins for further improvement of modern surfaces through mechanical or chemical procedures are very small. Major improvement can be achieved biologically by adding autologous growth factors, obtained by processing the patient's venous blood, to the implant surface.

The study on tissue reparative processes has highlighted the fundamental role played by platelets (in this context), which are physiological reservoirs of growth factors and proteins. There are various platelet concentrates, such as platelet-rich plasma (PRP), platelet-rich fibrin (PRF), and concentrated growth factor (CGF), which reconstruct bone defects [3].

Numerous studies have shown that PRF provides positive results in tissue engineering [4]. Research by Sohn et al. has demonstrated the greater regeneration capacity of the CGF and its multi-purpose use [5]. After a long phase of the study, our therapeutic choice was the use of the CGF, for the following reasons.

It is 100% autologous and biocompatible, requires a simple preparation, is easily identifiable, has a very high concentration of platelets in a fibrin network, has a presence of growth factors and no manipulation of the product is necessary when exclusively using autologous blood products, without the addition of other substances.

Platelets, in particular, contain biologically active proteins at high concentrations and support cell healing, growth, and morphogenesis [6]. In addition to platelets, CGF contains fibroblasts, leukocytes, and endothelial cells for angiogenesis and tissue remodeling; and provides a matrix for cell migration [7, 8]. CGF is a fibrin biomaterial rich in the growth factors obtained by centrifugation of venous blood, at alternating speeds based on a special device (Silfradent Co.) [8].

CGF, associated with guided bone regeneration, has been used to accelerate new bone formation. Due to its special characteristics, including lack of immune reaction, the capability of accelerating tissue healing and vascularization, and anti-swelling properties, CGF is widely used in implant surgery [9–11]. However, the interaction between CGF and the dental implant is not clear. The addition of autologous growth factors to the implant surface is hindered by titanium's characteristics of extremely low wettability [12, 13]. This means that simply wetting the implant with autologous growth factors is of little use unless it is left in immersion for more than 30 minutes [13]. This makes the procedure difficult to include in the clinical routine. Because of these difficulties, the challenge of producing a biologically active surface remains. Our study experiences demonstrate a protocol that could produce a biologically active implant surface. The growth factors are incorporated onto the implant surface, using a dedicated implant ampoule, which enables the procedure to be carried out in a closed field and a second device (Roundup) to incorporate GF onto the surface in a few seconds. We verified the adhesion of CGF on the titanium implant surface and then quantified the release of the vascular endothelial growth factor (VEGF) from CGF and the liquid phase of CGF (LPCGF) permeated implants. Later on, we tested other platelet-derived growth factors release, bone morphogenetic proteins, and stem cells. In the end, we evaluated the clinical outcome on patients.

This justifies the assertion that the first biologically active implant surface was produced, permeated with both fibrin, essential for hosting the cellular network, and growth factors, essential for creating intercellular communication and neo-angiogenesis. The osseointegration process thus becomes bilateral, operating both from the bone toward the implant and the implant toward the bone. This reduces the healing time and enhances the physiological response by four to six times [14, 15].

The research of this activity was focused on the adhesion of autologous growth factors, extracted with innovative techniques from the patient's venous blood, on the surface of the device, thus obtaining a biologically active implant surface. These growth factors are appropriately characterized to evaluate the synergistic effect from the point of view of their activity.

Growth Factors

An ideal bone graft in dentistry should be biomimetic and should have the ability to induce differentiation of the appropriate cells for bone formation; it should be costeffective and it should be able to achieve consistent and predictable results without being affected by different levels of technical ability of the clinician.

The dream of any surgeon would be to have a miraculous substance that could heal all wounds quickly and avoid the secondary complications of surgical procedures, such as edema, pain, dehiscences, infections, and hematomas.

The bad news is that this substance does not exist.

To meet these demands, research has focused on the use of bioactive molecules to induce bone formation and reduce post-surgery complications.

The growth factors associated with repair and regeneration coordinate important cellular processes such as stimulation and inhibition of growth, development and differentiation, mitogenesis, chemotaxis, angiogenesis, and apoptosis [16, 17].

Growth factors can be distinguished in:

Natural: Extracted from cells or tissues and then reprocessed;

Synthetic: Completely synthesized in the laboratory.

The characteristic common to both is their exceptional reactivity even at very low concentrations.

At present, we consider only autologous growth factors.

Synthetic growth factors could tomorrow be an alternative as far as legislative and even medico-legal problems pose today a serious difficulty to their clinical use. On the other hand, the mechanism of action of growth factors is only partly known and for this reason, the application of synthetic growth factors poses difficulties also from an ethical point of view.

What normally happens is that the degranulating platelets release growth factors, which stimulate cell proliferation, intercellular communication, and neoangiogenesis. They, therefore, give way to a process aimed at hemostasis and healing.

This is a well-known biological process that we are going to enhance and accelerate through the on-site application of the patient's autologous growth factors. The safety and the manageability in the application of growth factors (GF) are out of the question since it is an autologous product to which no exogenous components are added.

There are different methods and protocols to obtain GF from a patient's blood. The most used are Platelet Rich Plasma (PRP), Plasma Rich in Growth Factors (PRGF), Advanced Platelet-Rich Fibrin (A-PRF), and Concentrated Growth Factors (CGF).

Platelet-rich plasma (PRP) is a concentrated preparation of platelets now termed "first-generation platelet concentrate." PRP is a rich source of growth factors and promoted significant changes in monocyte-mediated proinflammatory cytokine/chemokine release. Leukotriene A4 (LXA4) was increased in PRP, suggesting that PRP may suppress cytokine release, limit inflammation, and, thereby, promote tissue regeneration [17]. Platelet activation allows access to autologous growth factors, which by definition, are neither toxic nor immunogenic and are capable of accelerating the normal processes of bone regeneration. In general, a large body of PRP studies demonstrated that PRP stimulates the proliferation and differentiation of fibroblasts, osteoblasts, chondrocytes, and mesenchymal stem cells [18, 19]. PRP can thus be considered a useful instrument for increasing the quality of regenerated bone [20] and wound healing of injuryassociated soft tissue defects [21–25] for chronic nonhealing tendon injuries.

Platelet-rich fibrin (PRF), a self-clotted preparation of PRP derivative, also overcame these matters. Blood samples collected in the absence of anticoagulants are immediately centrifuged to form fibrin clots. This simple preparation procedure has been widely accepted in various medical fields and spread worldwide. The developer of PRF further modified it to an advanced form (A-PRF), which is expected to contain a relatively greater number of white blood cells (WBC) [26]. Because of low-speed centrifugation, this fibrin clot is softer than that of the original PRF. On the other hand, concentrated growth factors (CGF), another modified form of PRF, are prepared by repeatedly switching the centrifugation speed and are characterized as a relatively stiffer fibrin clot [27]. Therefore, it has been anticipated that the difference in mechanical characteristics may produce a difference in the growth factor content.

Novel technologies (such as the cell concentrator Silfradent®) make possible the extraction of the so-called **CGF**. CGF is an autologous leukocyte-rich and plateletrich fibrin (L-PRF) biomaterial termed "second-generation platelet concentrate." CGF contains autologous osteoinductive platelet growth factors and an osteoconductive fibrin matrix [28]. It is also present in CGF: TGF-b1, VEGF,

and CD34 positive cells [29]. The application of CGF resulted in excellent healing of critical-size bone defects in vivo [28], hair loss [30], and promissory in the periphery and myocardial ischemia [31].

Platelet count is the first variable to consider. Absolute platelet count varies depending on the platelet concentration in the subjects' peripheral blood. PRP devices can be usually divided into lower (2.5–3 times baseline concentration) and higher (5–9 times baseline concentration) systems. It would seem intuitive that a higher platelet count would yield more growth factors and better clinical results; however, this has not yet been determined. Graziani et al. suggested that the optimal concentration of PRP was 2.5 x baseline and above this, there may be an inhibitory effect [32]. The optimal concentration was estimated at 2.5-fold to stimulate soft tissues. However, 2.5/3-folds upper baseline was optimal to stimulate bone regeneration [33, 34].

PRP containing white blood cells will have different biologic activity than PRP in which they are absent. The lower platelet count systems separate the whole blood into two components: one with the cellular components and the other consists of serum in which the platelets are suspended. The higher platelet count systems separate the whole blood into three fractions: the red cells, serum, and buffy coat. The buffy coat contains both platelets and WBC.

WBC can be further classified into different types. These include neutrophils, monocytes/macrophages, and lymphocytes. Their roles in tissue healing are different. Neutrophils are phagocytic and contain over 40 hydrolytic enzymes. Their activation leads to phagocytosis of debris and the release of oxygen-free radicals and proteases. This release of toxic molecules from the neutrophils can lead to secondary damage to the muscle [35, 36]. Whether or not neutrophils have a negative or positive effect on acute or chronically injured soft tissue is unknown.

Macrophages are the tissue forms of the circulating monocytes. Their role is the removal of debris and they are primarily phagocytic. They also have a role in balancing the pro-inflammatory and anti-inflammatory aspects of the healing [36, 37]. Since it is not possible to fractionate different types of white blood cells out of PRP, it may be that the absence of macrophages is more detrimental to healing than any secondary damage inflicted by neutrophils.

The growth factor contents in PRF and CGF preparations and their bioactivities have been demonstrated in vitro studies by several independent scientific groups [22, 24–35], the regenerative effects of PRF/CGF are not solely due to fibrin clots. The major source of growth factors in PRF preparations is its exudate; however, as a minor source, growth factors are thought to be secured by fibrin fibers [25]. The angiogenic activity of PRF/CGF preparations in endothelial cell cultures and the chick embryo chorioallantoic membrane (CAM) assay [36], demonstrated that PRF/CGF preparations are somewhat more potent in angiogenesis than PRP preparations. The comparison of the growth factor contents in four types of PRP derivatives (PRP, PRGF, A-PRF, and CGF) prepared from the same donors showed that both A-PRF and CGF preparations contained TGF-β1, PDGF-BB, VEGF, IL-1β, and IL-6 at levels similar to or higher than PRP preparations [25]. The expected proliferative effects of both A-PRF and CGF extracts were demonstrated in the in vitro assay using human periosteal cells, which give rise to osteoblasts involved in periodontal skeletal regeneration. Therefore, as do PRP preparations, these self-clotted PRP derivatives are expected to function not only as a scaffolding material but also as a reservoir to deliver certain growth factors and proinflammatory cytokines at the implantation sites [25].

For all these reasons, we decided to evaluate the outcome of the application of CGF on the implant surface with in vitro and in vivo studies.

Recent publications underline moved in the right direction as Concentrated Growth Factors (CGF) Induce Osteogenic Differentiation in Human Bone Marrow [33].

In the first part of our research, we decided to test the release of GF from the biologically coated implant surface in vitro. Later on, we did it in vivo.

For the in vitro test, we used good health volunteers to take the blood, elaborate, and incorporate it into the implant. For the in vivo use, we decided to focus our attention on elderly patients with dysmetabolic diseases.

Centrifugation of blood samples

Figure 28.1 Medifuge device. *Source:* Thermo Fisher Scientific Inc.

Figure 28.2 White fraction and red fraction.

Preparation of CGF

Blood samples (8mL) were taken by puncture of a vein from five donor patients, non-smokers, and in good general health. The five donors involved in the study (three men and two women) were aged between 25 and 45 years, with a BMI (Body Mass Index) between 21 and 23 points. The general medical history was negative. Patients were not on therapy with any type of drug and the blood samples were taken separately from the main meals, on empty stomachs. Informed consent was obtained from the patients included in this study. Tubes of blood were processed by a device (Medifuge MF200; Silfradent) to obtain CGF, following the manufacturer's instructions (Figure 28.1).

The evaluation of the quality of the CGF was made on two fractions: the so-called **white fraction** and the **red fraction** (Figure 28.2).

White Fraction: LPGF (liquid face of CGF) liquid uncured fibrin obtained with a white cap tube. It is a

completely smooth test tube without any additive inside that delivers the material in a liquid state, which will polymerize at room temperature in the next 15 minutes (Figure 28.3).

Red fraction: CGF polymerized fibrin, which is obtained with a red cap tube and has rough walls to promote polymerization through an exclusively physical process. It has a greater consistency, a gelatinous appearance, and a higher cellular concentration of uncured fibrin due to the activated fibrin network that traps more cells. Within the network, cells can migrate and differentiate (Figure 28.4).

It was, therefore, possible to evaluate the different results of the two preparations.

The resulting CGF was then inserted into dedicated implant ampoules (Figure 28.5), each containing an implant (IML®, Swiss dental implants, diameter 4mm and height 8mm). To incorporate the CGF onto the implant surface, these tubes were inserted into a second device,

Figure 28.3 Isolation of the white fraction.

Figure 28.6 Tubes ready for incorporation of CGF in Roundup device.

Figure 28.4 Isolation of the red fraction, polymerized fibrin.

Figure 28.5 Implant a CGF in the dedicated tubes.

Roundup (Silfradent), and centrifuged for 16 seconds, following the manufacturer's instructions (Figure 28.6).

After centrifuging the growth factors with the 8 implants for 16 seconds, the tubes obtained were placed in two different culture materials: DMEM and PBS (Figure 28.7).

Figure 28.7 Implants placed in DMEM and PBS.

In vitro Trial

After preparing Concentrated Growth Factors **two main experiments** were conducted:

- **the first** to assess the presence of the VEGF in the CGF and LPGF (Figure 28.8), and with the implant plus CGF/ LPGF (Figure 28.9).
- **the second** to evaluate: (i) the ability of the implant surface to be permeated by the CGF (Figures 28.10–28.12); (ii) the extent of the diffusion of the VEGF present in the CGF with the implantation in the culture medium; (iii) the implant's ability to release VEGF by moving it to another culture medium (Figure 28.13).

The analysis of the first graph shows how the CGF has a much higher concentration of VEGF than the LPGF, however, after one day, the concentrations in both preparations are superimposable.

On the other hand, considering the second graph, the solid CGF with the implant presents and maintains over

Figure 28.9 Implants with white portion and red portion of CGF.

Figure 28.10 Implant permeated with CGF.

time an ever-greater concentration of VEGF compared to the LPGF with implant (Figure 28.9).

To assess whether the VEGF was closely linked to the implant or was immediately released into the culture medium, after a day the implant was moved by inserting it into a new well with DMEM or pure PBS.

The culture medium left without implant corresponds to the **2d sv** sample (two days "senza vite" – two days without screw) because it was taken two days after the start of the experiment.

The well into which the implant was transferred corresponds to sample **2d** (two days).

As can be seen from the graph, VEGF remains within the implant surface and is gradually released over time.

Figure 28.11 Implant permeated with CGF SEM photomicrograph.

Consequently, VEGF pertains to the implant surface and is not linked to the presence of the culture medium. This deduction is supported by the fact that the 2d sv sample is comparable to 12 hours, moreover, the VEGF value of the 2 g corresponds almost perfectly to the sum of $2sv+3g$.

In neutral soil, the relevant aspect is that when the implant is removed, the VEGF immediately drops. When the implant is placed back in another culture medium, the VEGF rises again.

So it means that the release of VEGF starts from the implant, and it can be deduced that the implant has a biologically active surface.

Results

The results obtained from the in vitro experiments show that the concentration of growth factors (VEGF) is higher in the CGF rather than in the LPGF and by centrifuging the

Figure 28.12 Strong magnification of the CGF-permeated implant surface.

implant with the CGF, the presence of VEGF is very high at zero time and then decreases.

Subsequently, the results of the second experiment show that VEGF remains within the implant surface and is gradually released over time.

It is also important to emphasize that the use of specific implants is essential for a better implant micro-surface, allowing the growth factors to settle inside it.

From a practical point of view, the insertion of the LPGF can be achieved through a closed system by direct injection into the implant tube. And it is an easily reproducible situation in an outpatient setting, thanks to dedicated implant tubes also.

However, the use of CGF (already polymerized – red tube), although it has a much higher presence of VEGF at zero time than LPGF, instead involves opening the implant tube to insert the material and then closing it again before insertion in the Roundup device to incorporate on the surface. This procedure of opening the tube and inserting the growth factor, however, takes place in an open system, but with contiguity of time and space that does not limit its clinical application.

Implants with CGF In vivo

So starting from this, we decide to apply this technology to patients whose biological response is compromised because of their age and health conditions:

21 patients, aged between 27 and 72 years old, were enrolled to be rehabilitated with 45 IML®, swiss dental implants of which 22 were CGF permeated implants. They were patients whose general health conditions were not always favorable for bone healing.

In particular: six patients with mild osteoporosis not on therapy; three patients with moderate osteoporosis on therapy or who have finished bisphosphonate therapy; two patients with compensated diabetes; six patients with metabolic diseases (hypercholesterolemia, thyroid hormone alterations); four patients with hypertension or on antiplatelet/anticoagulant therapy.

All patients rehabilitated with implants plus CGF were appropriately examined before surgery and two radiographic examinations were required: (i) dental arches orthopantomography to have a global assessment of the patient, to detect sites of monoedentulism or to plan multiple implant insertions in the absence of two or more dental elements; (ii) cone beam computed tomography examination of the affected arch to study the thickness, height, and quality of the rehabilitated bone tissue.

Patients with poor oral hygiene, insufficient space available for the introduction of the fixture, bruxism, rehabilitations with angulation correction of over 25° concerning the implant axis, or rehabilitations with excessive cantilever were excluded from the study.

Our work evaluated the effect of CGF on implant healing. The particular clinical evaluation was performed through a survey filled in 24 hours after surgery and the analysis of the post-operative course (edema, pain, and bleeding) after surgery. For this evaluation propose the healing times, any problems on the implant prosthesis, the intraoral radiographic (bone resorption, any exposed implant threads) as well as the peri-implant probing (bleeding or pain on probing) at 3, 6, and 12 months after surgery were evaluated. The split-mouth design, which provides rehabilitating half-arch with traditional implants and the other half-arch with CGF-permeated implants in the same patient, was used when applicable.

Informed consent was obtained from all patients.

The use of implants permeated with CGF demonstrated a significant improvement in the final results when compared with those without CGF permeation in terms of postsurgery complications such as edema, pain, and bleeding (Figure 28.14) and bone level (Figures 28.15–28.18).

The pain was assessed with the Visual Analogue Scale (VAS).

The measurement of the edema was reported by the oral surgeon after a post-surgery check-up.

Additionally, patients experienced faster and uncomplicated healing, especially on the rehabilitated side with growth factors. The peri-implant probing performed 3, 6, and 12 months after surgery did not create bleeding or pain. From 24 hours after the analysis of the intervention survey, 89% of patients declare that they do not feel much pain, 93% do not have bruises and edema and 96% declare that they have quickly resumed normal daily activity.

Discussion

To obtain maximum implant success, it is necessary to achieve and maintain the stability of the implant from a morphological point of view [1]. The concept of osseointegration has led to the emergence of the concept of primary stability, influenced by the quality of the bone in

Figure 28.14 Post-operative course for traditional and CGF-permeated implants.

Figure 28.16 Patient treated with split-mouth with IML implants, with CGF (a), and without CGF (b).

Figure 28.17 Implant treated with CGF in a diabetic patient, note the perfect preservation of crestal bone levels.

Figure 28.15 Patient treated with implants without CGF (b) in the frontal area and CGF-permeated implants were used in the posterior areas (a).

Figure 28.18 Detail of the mandible of two implants in an elderly patient undergoing treatment for osteoporosis with bisphosphonates.

the surgical site [1, 2]. Conversely, secondary stability is the stability of the implant after soft tissue and bone healing [11]. Davies [12] has studied the healing phases of the peri-implant bone and has shown that, following a traumatic event, the formation of lamellar bone is not immediate; rather, an irregular bone is formed called a woven bone.

Human studies have shown that CGF can be very beneficial; it is simple to apply during surgery or other types of therapy. Furthermore, together with CD 34+ stem cells, CGF has shown significant benefits in terms of accelerating wound healing, soft tissue repair, and new bone formation [7, 9, 38–44]. Autologous CGF treatment is generally considered safe in carefully evaluated patients.

Potential contraindications are patients with anemia, thrombocytopenia, hemodynamic instability, unstable angina, sepsis, anticoagulants, or fibrinolytic therapy. The advantages of this simple method are particularly evident in patients with systemic or metabolic diseases or undergoing regenerative treatments. It is capable of expanding the success rate in all patients, being safe, biologically compatible, and economical.

These findings were evident in preclinical models, known as reliable tools for studying osteogenesis and the endothelial biocompatibility of CGF-permeated devices. However, our in vitro experimental results do not necessarily translate directly into the in vivo situation. Therefore, further investigations were performed to evaluate the in vivo efficacy of CGF-permeated implants in promoting osseointegration and tissue regeneration and reducing post-surgical complications.

Conclusion

Optimal crestal level, probing, and less bleeding were obtained using the CGF-permeated implants compared to the un-permeated implant interventions, where bone resorption was often observed. These results were also obtained in elderly subjects with conditions that are not always eligible for dental implant surgery. Overall, these data highlight interesting new perspectives for the improvement of CGF-coated implant osteointegration and healing.

Based on the histological and clinical results obtained, we can affirm that we have created the first biologically active implant surface that is permeated both by fibrin, essential to accommodate the cellular network, and by growth factors essential to creating intercellular communication and neo-angiogenesis. This implies that the osseointegration process becomes bilateral, that is, from the bone surface toward the implant and also from the implant toward the bone.

In the future, further studies will be needed to investigate the use of biologically active surfaces and improve the implant micro-surface to be permeated more and more by autologous growth factors to maximize osseointegration and reduce complications.

References

- **1** Albrektsson, T. and Zarb, G.A. (1999). Determinants of correct clinical reporting. *Ontario Dentist* 76 (4): 29–33.
- **2** Albrektsson, T. and Wennerberg, A. (2005). The impact of oral implants – past and future, 1966-2042. *Journal of the Canadian Dental Association* 71 (5): 327.
- **3** Ananth, H., Kundapur, V., Mohammed, H.S. et al. (2015). Mankar S.A review on biomaterials in dental implantology. *International Journal of Biomedical Sciences* 11 (3): 113–120.
- **4** Coetzee, J.C., Pomeroy, G.C., Watts, J.D., and Barrow, C. (2005). The use of autologous concentrated growth

factors to promote syndesmosis fusion in the Agility total ankle replacement.A preliminary study. *Foot & Ankle International* 26: 840–846.

- **5** Sohn, D.S., Heo, J.U., Kwak, D.H. et al. (2011). Bone regeneration in the maxillary sinus using an autologous fibrin-rich block with concentrated growth factors alone. *Implant Dentistry* 20: 389–395.
- **6** Nurden, A.T., Nurden, P., Sanchez, M. et al. (2008). Platelets and wound healing. *Frontiers in Bioscience* 13: 3532–3548.
- **7** Gassling, V.L., Acil, Y., Springer, I.N. et al. (2009). Plateletrich plasma and platelet-rich fibrin in human cell culture.

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Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics 108: 48–55.

- **8** Bernardi, S., Mummolo, S., Tecco, S. et al. (2017). Histological characterization of sacco's concentrated growth factors membrane. *International Journal of Morphology* 35: 114–119.
- **9** Takeda, Y., Katsutoshi, K., Matsuzaka, K., and Inoue, T. (2015). The effect of concentrated growth factor on rat bone marrow cells in vitro and on calvarial bone healing in vivo. *The International Journal of Oral & Maxillofacial Implants* 30: 1187–1196.
- **10** Kotsakis, G.A., Boufidou, F., Hinrichs, J.E. et al. (2016). Extraction socket management utilizing platelet rich fibrin: a proof-of-principle study of the "accelerated-early implant placement" concept. *The Journal of Oral Implantology* 42: 164–168.
- **11** Herrmann, M., Binder, A., Menzel, U. et al. (2014). CD34/ CD133 enriched bone marrow progenitor cells promote neovascularization of tissue-engineered constructs in vivo. *Stem Cell Research* 13: 465–477.
- **12** Davies, J.E. (1996). In vitro modeling of the bone/implant interface. *The Anatomical Record* 245 (2): 426–445.
- **13** Kim, I.H., Son, J.S., Choi, S.H. et al. (2016). Nano- and micro-scale oxidative patterning of titanium implant surfaces for improved surface wettability. *Journal of Nanoscience and Nanotechnology* 16: 1883–1886.
- **14** Sánchez-Ilárduya, M.B., Trouche, E., Tejero, R. et al. (2013). Time-dependent release of growth factors from implant surfaces treated with plasma rich in growth factors. *Journal of Biomedical Materials Research. Part A* 101 (5): 1478–1488.
- **15** Jhin, M.J., Kim, K.H., Kim, S.H. et al. (2013). Ex vivo bone morphogenetic protein-2 gene delivery using bone marrow stem cells in rabbit maxillary sinus augmentation in conjunction with implant placement. *Journal of Periodontology* 84 (7): 985–994.
- **16** Gardner, R.L. (2002). Stem cells: potency, plasticity and public perception. *Journal of Anatomy* 277–282.
- **17** El-Sharkawy, H., Kantarci, A., Deady, J. et al. (2007). Platelet-rich plasma: growth factors and pro- and anti-inflammatory properties. *Journal of Periodontology* 78: 661–669.
- **18** Sampson, S., Gerhardt, M., and Mandelbaum, B. (2008). Platelet rich plasma injection grafts for musculoskeletal injuries: a review. *Current Reviews in Musculoskeletal Medicine* 1: 165–174.
- **19** Lee, K.S., Wilson, J.J., Rabago, D.P. et al. (2011). Musculoskeletal applications of platelet-rich plasma: fad or future? *AJR. American Journal of Roentgenology* 196: 628–636.
- **20** Pacifici, L., Casella, F., and Maggiore, C. (2002). Platelet rich plasma (PRP): potentialities and techniques of extraction. *Minerva Stomatologica* 51: 341–350.
- **21** Gao, F., Wang, J.X., and Han, Y. (2009). Research advance on application of platelet-rich plasma in wound repairreview. *Zhongguo Shi Yan Xue Ye Xue Za Zhi* 17: 840–843.
- **22** Cieslik-Bielecka, A., Bielecki, T., Gazdzik, T.S. et al. (2009). Autologous platelets and leukocytes can improve healing of infected high-energy soft tissue injury. *Transfusion and Apheresis Science*.
- **23** Kawase, T. (2015). Platelet-rich plasma and its derivatives as promising bioactive materials for regenerative medicine: basic principles and concepts underlying recent advances. *Odontology* 103: 126–135.
- **24** Anitua, E. (2001). The use of plasma-rich growth factors (PRGF) in oral surgery. *Practical Procedures & Aesthetic Dentistry* 13: 487–493.
- **25** Masuki, H., Okudera, T., Watanebe, T. et al. (2016). Growth factor and pro-inflammatory cytokine contents in platelet-rich plasma (PRP), plasma rich in growth factors (PRGF), advanced platelet-rich fibrin (A-PRF), and concentrated growth factors (CGF). *International Journal of Implant Dentistry* 2: 19.
- **26** Choukroun, J. (2014). Advanced PRF, A-PRF: platelet concentrates or blood concentrates? *Journal of Periodontal Medicine & Clinical Practice* 1: 3.
- **27** Corigliano, M., Sacco, L., and Baldoni, E. (2010). CGF- una proposta terapeutica per la medicina rigenerativa. *Odontoiatria* 1: 69–81.
- **28** Honda, H., Tamai, N., Naka, N. et al. (2012). Bone tissue engineering with bone marrow-derived stromal cells integrated with concentrated growth factor in Rattus norvegicus calvaria defect model. *Journal of Artificial Organs* 16: 305–315.
- **29** Rodella, L.F., Favero, G., Boninsegna, R. et al. (2011). Growth factors, CD34 positive cells, and fibrin network analysis in concentrated growth factors fraction. *Microscopy Research and Technique* 74: 772–777.
- **30** Kang, J.S., Zheng, Z., Choi, M.J. et al. (2014). The effect of CD34+ cell-containing autologous platelet-rich plasma injection on pattern hair loss: a preliminary study. *Journal of the European Academy of Dermatology and Venereology* 28: 72–79.
- **31** Mackie, A.R. and Losordo, D.W. (2011). CD34-positive stem cells: in the treatment of heart and vascular disease in human beings. *Texas Heart Institute Journal* 38: 474–485.
- **32** Graziani, F., Ivanovski, S., Cei, S. et al. (2006). The in vitro effect of different PRP concentrations on osteoblasts and fibroblasts. *Clinical Oral Implants Research* 17: 212–219.
- **33** Rochira, A., Siculella, L., Damiano, F. et al. (2020). Concentrated growth factors (CGF) induce osteogenic differentiation in human bone marrow stem cells. *Biology* 9: 370.
- **34** Weibrich, G., Hansen, T., Kleis, W. et al. (2004). Effect of platelet concentration in platelet-rich plasma on peri-implant bone regeneration. *Bone* 34: 665–671.
- **35** Smith, C., Kruger, M.J., Smith, R.M., and Myburgh, K.H. (2008). The inflammatory response to skeletal muscle injury: illuminating complexities. *Sports Medicine* 38: 947–969.
- **36** Tuomi, H. and Best, T. (2003). The inflammatory response: friend or enemy to muscle tissue? *British Journal of Sports Medicine* 37: 284–286.
- **37** Tidball, J.G. and Wehling-Henricks, M. (2007). Macrophages promote muscle membrane repair and muscle fibre growth and regeneration during modified muscle loading in mice in vivo. *The Journal of Physiology* 578: 327–336.
- **38** Dohan Ehrenfest, D.M., Rasmusson, L., and Albrektsson, T. (2009). Classification of platelet concentrates: from pure platelet-rich plasma (P-PRP) to leucocyte- and plateletrich fibrin (L-PRF). *Trends in Biotechnology* 27: 158–167.
- **39** Kobayashi, M., Kawase, T., Horimizu, M. et al. (2012). A proposed protocol for the standardized preparation of PRF membranes for clinical use. *Biologicals* 40: 323–329.
- **40** Nishimoto, S., Fujita, K., Sotsuka, Y. et al. (2015). Growth factor measurement and histological analysis in platelet rich fibrin: a pilot study. *Journal of Maxillofacial and Oral Surgery* 14: 907–913.
- **41** Lundquist, R., Dziegiel, M.H., and Agren, M.S. (2008). Bioactivity and stability of endogenous fibrogenic factors in platelet-rich fibrin. *Wound Repair and Regeneration* 16: 356–363.
- **42** Su, C.Y., Kuo, Y.P., Tseng, Y.H. et al. (2009). In vitro release of growth factors from platelet-rich fibrin (PRF): a proposal to optimize the clinical applications of PRF. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics* 108: 56–61.
- **43** Clipet, F., Tricot, S., Alno, N. et al. (2012). In vitro effects of Choukroun's platelet-rich fibrin conditioned medium on 3 different cell lines implicated in dental implantology. *Implant Dentistry* 21: 51–56.
- **44** He, L., Lin, Y., Hu, X. et al. (2009). A comparative study of platelet-rich fibrin (PRF) and platelet-rich plasma (PRP) on the effect of proliferation and differentiation of rat osteoblasts in vitro. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics* 108: 707–713.

Plasma Cleaning for Implant Surfaces to Improve Implant Success

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Introduction

Osseointegration is the foundation of modern oral implantology. As discovered by Brånemark et al. [1], osseointegration is the process by which surrounding bone becomes fused to a load-bearing implant. The strength of bone-toimplant attachment dictates implant success, as dental implants must be able to withstand masticatory forces whilst remaining mechanically stable. Osseointegration is reminiscent of fracture healing – an injury is made to bony tissue during drilling for implant placement, initiating a phase of wound healing marked by bone remodeling [2]. Insufficient osseointegration can result in fibrous tissue development at the site of implant placement, loosening oral prosthesis, and causing failure [3].

The material composition of dental implants is one of the key determinants of implant success. Pure titanium and Titanium–Aluminum–Vanadium alloy-based implants are the most common implant materials used in dentistry, although zirconium dioxide and novel ceramic alloys are gaining popularity among implant materials due to esthetic appeal [4]. In a study conducted by Simonis et al. [5], the clinical survival rates of titanium-based implants 10- and 16 years after placement were 89.23 and 82.94%, respectively [5]. Several studies report similar findings, with the long-term success of titanium-based implants being widely accepted in oral implantology [6–8]. In order to increase the success of oral implants, their surface properties can be modified to improve osseointegration and promote bone remodeling at the implant site.

Conventional surface modification methods, such as sandblasting, acid-etching, and anodization, cause microscale changes to implant topography that increase

the surface area and roughness of the implant surface [9–11]. These modifications lead to more bone deposition and integration of the implant; however, studies have shown a potential correlation between roughened implant surfaces and the maintenance or promotion of peri-implant diseases, thereby prompting investigation into nanoscale and plasma treatment as substituted or additive surface modification methods for implants [12]. Nanoscale modifications can improve the wettability, bioactivity, and bacterial resistance of implant surfaces using 3D-laser texturing and biomimetic modification; therefore, researchers suggest that nanoscale modifications should be performed in conjunction to microscale techniques to encourage osseointegration and maintain implant vitality [13].

Plasma is a fundamental state of matter that exists as an ionized gas composed of positive ions, free electrons, molecular particles, and free radicals. Due to its ability to generate reactive oxygen and nitrogen species, plasma is thought to be a safe surface modification technique that decontaminates implant surfaces whilst increasing surface hydrophilicity to facilitate protein and cell adhesion for osseointegration [14]. Plasma treatment has also been found to be bactericidal and non-damaging, decontaminating implants, and disrupting oral biofilm formation without altering surface topography [15]. Plasma treatment is thus a safe, non-toxic method for cleaning implant surfaces and favorable for clinical application over other conventional methods of surface modification. The present study will review plasma treatment for implant surfaces, highlighting the types of plasma, benefits of plasma, and systems of plasma cleaning along with commercially available plasma devices.

Plasma Cleaning

Types of Plasma

There are two main types of plasma for implant surface modification – thermal plasma or atmospheric-pressure plasma. Thermal, or hot, plasmas have conventionally been used for plasma spraying hydroxyapatite coatings onto implant surfaces, whereas, atmospheric-pressure, or cold, plasmas have been used to modify implant surface chemistry and increase surface area (Figure 29.1) [17, 18]. The ability of atmospheric-pressure plasmas to generate reactive oxygen and nitrogen species causes implant surface modification, thereby enhancing its osseointegrative properties.

Compared to thermal plasmas, atmospheric-pressure plasmas have garnered much attention in the fields of dentistry and biomedicine for their practicality, efficiency, and cost-effectiveness. Atmospheric-pressure plasma can drive high-temperature chemistry at low ambient temperatures while also serving as a suitable benchtop method of implant surface treatment that improves osseointegration immediately prior to insertion [19]. Additionally, atmospheric-pressure plasma devices do not require the use of vacuum equipment, thus making them more portable and affordable [17]. In contrast, thermal plasmas have limited applications and researchers have argued against its use for treating implant surfaces [20]. It is for these reasons that atmospheric-pressure plasma has dominated research

related to implant surface modification. As a result, the following discussion primarily references studies involving the use of atmospheric-pressure plasma.

Benefits

Plasma cleaning is a key method of improving the osseointegrative properties of dental implants. Plasma treatment can refine the implant surface for osseointegration by removing organic matter, increasing hydrophilicity, and eliminating microbial challenges. Plasma treatment is an efficient method of cleansing implant surfaces of organic contamination to promote osseointegration. Bone-to-implant contact percentages, representative of osseointegration, are commonly less than ideal, ranging between 52 and 78% [21]. These values may be attributed to the biological aging of implant surfaces; that is, organic matter and carbon compounds can accumulate on the surface of implants from atmospheric exposure during the manufacture-to-insertion period [22]. Studies have shown that plasma cleaning reduces the amount of carbon compound pollutants on the surface of titanium-based implants, improving osseointegration and bone response. Using atmospheric-pressure plasma treatment, Danna et al. [23] noted reduced carbon compounds on the surface and increased titanium and oxygen on pure titanium [23]. Tsujita et al. [24] also observed a decrease in the number of carbon pollutants on the surface of titanium screws that were subject to atmospheric-pressure plasma treatment [24].

Figure 29.1 Application of non-thermal (atmospheric pressure) plasma for implant surface modification. *Source:* Adapted from Lata et al. [16].

The wettability (or hydrophilicity) of implant surfaces improves the absorption of proteins and cell adherence as well as connective tissue network interaction. The role of surface energy and osteoblast cell adhesion is well defined in the literature – higher surface energy correlates with enhanced osteoblast adhesion and proliferation which modulates bone remodeling and osseointegration – and it is favorable to improve the hydrophilicity of implant surfaces to achieve higher surface energy [25–27]. Plasma is capable of conferring hydrophilicity to implant surfaces by modifying the oxide layer, which lies at the interface with the cells of the surrounding tissue, generating reactive species, and thus improving tissue adhesion [15, 28]. Studies from Coelho et al. [19] revealed higher surface hydrophilicity, enhanced cell recruitment, and increased surrounding tissue contact during healing for plasma-treated titanium implants compared with those that were left untreated [19]. The researchers were also able to observe a higher quantity of bone surrounding roughened plasma-treated implant surfaces, indicating improved osteoblast adhesion [19]. Duske et al. [15] similarly reported increased hydrophilicity and osteoblast cell attachment to plasma-treated titanium implant surfaces [15]. The findings from these studies corresponded with current understandings of hydrophilic implant surfaces and cell adhesion reported in the literature [14, 29, 30].

Plasma cleaning of implant surfaces is also an effective method to improve osseointegration whilst disrupting bacterial attachment and aggregation. Titanium and titanium alloy implants that are roughened from sandblasting, grit blasting, and acid-etching techniques promote bacterial adhesion due to the microscopic pits and grooves that form. These changes to implant surface topography create a crevice for the bacteria to latch onto, ultimately triggering the host-inflammatory response and increasing the risk for peri-implant diseases [2, 31]. Plasma treatment can inhibit the attachment of adherent hydrophobic bacteria, like *Streptococcus sanguinis*, by carbon-cleaning implant surfaces and producing reactive oxygen and nitrogen species to increase hydrophilicity [32]. The surface energy and chemical properties of implants play a role in facilitating bacterial attachment, that is, materials that have high surface energy generally bind bacteria; however, the hydrophilic or hydrophobic properties of the surface and bacteria dictate attachment to materials with high surface energy [33, 34]. Hydrophobic surfaces bind hydrophobic bacteria rather than hydrophilic bacteria [35]. Interactions between reactive oxygen and nitrogen species from plasma and the implant surface change surface chemistry and topography by improving hydrophilicity and surface energy without altering surface roughness, thus decontaminating the implant, and discouraging further hydrophobic bacterial colonization. In addition to

improving the hydrophilicity of the implant surface, the reactive oxygen and nitrogen species generated during plasma cleaning can directly modify bacterial membranes and invoke enzyme inhibition, leading to reactive oxygen and nitrogen species accumulation and bacterial cell death [36, 37]. Through decontamination experiments of titanium-based implant nanonetwork structures, Zeng et al. further demonstrated that plasma treatment could effectively eradicate the biofilm of biofilm-contaminated implant surfaces while preserving the nanostructure. Taken together, plasma cleaning is one of the best clinically available surface modification methods to sterilize dental implants and improve osseointegration.

Regarding clinical delivery, plasma cleaning is a costeffective, non-toxic, and efficient means of implant surface modification. At atmospheric pressure, plasma can be obtained from plasma torch, transferred arc, corona discharge, dielectric barrier discharge, or plasma jet devices. From an efficiency standpoint, plasma cleaning is a superior method of surface modification for its ability to disrupt oral biofilm formation and simultaneously generate a hydrophilic surface favorable for osseointegration. Commercially, plasma is advantageous due to its portability, low cost, ease of operation, and versatile applications for implant treatment. For instance, atmospheric-pressure plasma can be used to treat implants prior to insertion or as a therapeutic approach for peri-implantitis. Zeng et al. report the benefits of a handheld, nonthermal atmosphericpressure plasma device that utilizes piezoelectric technology to eliminate oral biofilm and decontaminate implant surfaces in a more environmentally friendly manner than conventional UV and laser surface modification treatments.

Atmospheric-pressure plasmas have also been found to be more convenient compared to earlier plasma technology, taking advantage of ambient pressure and temperature to drive plasma cleaning instead of lowpressure, high-temperature conditions that were previously required [19, 38]. This improves the functionality and usability of atmospheric-pressure plasma devices, making them capable of use in the operating room prior to implant insertion. Moreover, a study by Berger et al. [39] investigated the benefits of an on-site benchtop technique of plasma treatment for implants at the time of placement using a dielectric barrier discharge plasma device, reporting improved hydrophilicity, osteoblast adherence, and implant vitality with increased accessibility from using the dielectric barrier discharge device [39]. Atmosphericpressure microplasma devices may also prove to be clinically favorable for their portability and potential to generate plasma that increases implant surface energy under safe atmospheric conditions [19].

Atmospheric-pressure Plasma Systems and Devices for Cleaning Implant Surfaces

Plasma can be generated at atmospheric pressure using electric transferred arcs, corona, discharge, dielectric barrier discharge, and plasma jet systems. In essence, the input of energy from these systems activates a fraction of gas molecules from the atmosphere, turning them into charged particles (electrons and ions) and reactive oxygen and nitrogen species while the remainder remains inert. It is these charged ions that make plasma active and confer its ability to modify implant surfaces. Traditionally, high temperatures (exceeding 3000 °C) are required to drive atmospheric-pressure plasma generation (e.g. electric transferred arc plasma); however, modern systems (e.g. dielectric barrier discharge and plasma jet) have been able to generate plasma at lower temperatures (50–400 °C). These systems of atmospheric-pressure plasma generation will be reviewed in this section.

Transferred Arcs

Transferred arc devices have a rather simple apparatus – gas is pumped into a chamber, surrounded by a water-cooled shield, housing a cylinder-shaped cathode that generates a transferred arc discharge to an anode workpiece sitting external to the chamber (Figure 29.2). A DC power voltage of up to 200kW is supplied to the system, energizing the gaseous mixture of air and argon that passes between the cathode and shield and producing an arc that can be sustained between the two electrodes [40]. Transferred arcs

Figure 29.2 Transferred Arc apparatus. *Source:* Adapted from Schutze et al. [40].

have been studied in metallurgy and used in industry to cut steel plates; however, variations of the transferred arc apparatus have been developed for use in biomedicine. Plasma arc heaters stem from transferred arc devices but instead utilize the shield as the anode rather than the workpiece. By extension, plasma torches are a variation of plasma arc heaters that contain an injection port for precursor compounds that can be used for thin film deposition. It is these plasma torches that are clinically relevant for implant surface modification, as precursor compounds in the form of solid pellets can be inserted into the plasma torch apparatus, undergo vaporization, and generate reactive species in the form of a high-powered gas discharge that projects outward to be sprayed onto a given surface [40].

SteriPlas (Adtec Europe Ltd., London, UK) is an atmospheric-pressure gas plasma torch device that has been proven to be bactericidal, promote healing, manage infection, eliminate biofilms, and sterilize wounds and surfaces [41]. The device features a serrated design with six electrodes inside of an aluminum cylinder set at a fixed distance away from the inner surface of the plasma torch (Figure 29.3). This design maximizes electric field strength to facilitate plasma generation and charge stability. As little evidence exists, future studies may wish to utilize the SteriPlas device for dental implant surface modification.

Dielectric Barrier Discharge

The apparatus of dielectric barrier discharge devices consists of two metallic electrode plates, one of which is coated with a dielectric layer (Figure 29.4). Gas passed between the dielectric-coated electrode plate becomes ionized by energy from a voltage source supplying ~20kV to the system, producing a series of micro arcs that generate nonuniform plasma capable of producing reactive oxygen and nitrogen species [40].

The implaPrep concept device (Relyon Plazma GmbH, Regensburg, and Germany) alters implant surfaces by emitting an atmospheric dielectric barrier discharge plasma. Implant surfaces treated with the implaPrep concept device demonstrate increased surface area due to the removal of organic, carbon-based molecules, improving cell adhesion, and surface hydrophilicity [42]. In addition, the device is favorable compared to other commercially available plasma devices for its short treatment time (~30 seconds), portability, safety, efficiency, and ability to activate implant surfaces without changing surface topography. The implaPrep concept device features a plasma reactor with a dielectric barrier discharge plasma generator that uses the implant surface as the counter electrode and a plasma driver with a high voltage generator (25V) that can be easily controlled with analog processing signals (Figure 29.5). Using a guide attached to the device, the user

Figure 29.3 Image of SteriPlas Device (left) and CAD model (right). *Source:* Bennett et al. [41]/from MDPI/CC BY 4.0.

Figure 29.4 Dielectric barrier discharge apparatus (1) Metallic electrode plates. (2) Dielectric barrier-coated surface. *Source:* Adapted from Schutze et al., [40].

places the implant into the plasma reactor for ~30 seconds to be processed (Figure 29.6). The portability, inability to overtreat the implant, and lack of auxiliary materials that are needed to operate the implaPrep concept device make

it user-friendly. Future studies may wish to build upon the findings of Yan et al. [42], examining its effectiveness as a standalone, chairside method of surface modification immediately prior to implantation.

Active+ (Nova Plasma Ltd., Hevel Megiddo, and Israel) is a portable dielectric barrier discharge plasma device that sterilizes implants and improves surface hydrophilicity (Figure 29.7). Factory sterilized implants that are presealed in a disposable capsule are placed into the plasma chamber by the user and processed for ~30 seconds [43]. Active+ consists of a plasma reactor and plasma driver; however, the capsule serves as the counter electrode. Berger et al. [44] reported that implants treated with the Active+ device demonstrate improved osseointegration by increasing surface hydrophilicity compared to untreated implants [44].

Figure 29.5 Schematic of the implaPrep concept device. *Source:* Courtesy of RELYON PLASMA GMBH.

Figure 29.6 Images of a user placing an implant into the implaPrep concept device. *Source:* Courtesy of RELYON PLASMA GMBH.

Figure 29.7 Image of the Active+ Device (Nova Plasma Ltd., Hevel Megiddo, Israel). *Source:* Courtesy of Nova Plasma Ltd.

Plasma Jet

Atmospheric-pressure plasma jets consist of two concentric electrodes through which gas flows at temperatures ranging from 25–200 °C. Typically, atmospheric-pressure plasma jets operate under Radiofrequency (RF) power, supplying 100–250V to the inner electrode causing the gas to be ignited and released through a nozzle directed toward an object (Figure 29.8) [40]. Plasma jets are a favorable system of plasma generation for their ability to minimize operating temperatures, voltages, and the diameter of the capillary needed to generate plasma [45].

The Piezobrush® (Relyon Plazma GmbH, Regensburg, and Germany) is a handheld atmospheric-pressure plasma jet device that utilizes Piezoelectric Direct Discharge technology. The technology consists of a high-voltage plasma generator and direct electrical discharge transformer, allowing low input voltage to be transformed into electric fields that can ionize gas to create plasma (Figure 29.9) [46]. The Piezobrush® is particularly useful in clinical practice for its portability, benchtop capabilities, and low input, high output efficiency at low-room temperatures. Studies examining the impact of atmospheric-pressure plasma treatment on the osseointegration of titanium-based implant surfaces using the Piezobrush® reported enhanced surface decontamination, hydrophilicity, and osseointegration with unchanged surface topography for plasmatreated implants compared to untreated controls.

Low-pressure Plasma Systems and Devices for Cleaning Implant Surfaces

Low-pressure plasma systems reduce bacterial aggregations and activate implant surfaces for osseointegration by generating reactive oxygen and nitrogen species that can increase surface tension. Unlike atmospheric-pressure plasma systems, low-pressure systems utilize a vacuum chamber that partially ionizes gas that is then pumped out of the chamber and replaced with a processed gas at a controlled pressure. In doing so, the gas is transformed to a plasma state at a constant rate with the continuous replacement of gasses. Advantages of low-pressure plasma systems include amplified process uniformity, the ability to achieve high surface area from materials that atmosphericpressure systems cannot, and the generation of superior implant surface chemistry due to the use of vacuum chambers, which minimize the time between the activated state of the surface and bonding.

Diener electronic GmbH & Co KG, (Ebhausen, Germany), offers several low-pressure plasma systems for dental applications. Diener low-pressure systems can

piezobrush®

Figure 29.8 Atmospheric-pressure plasma apparatus. *Source:* Adapted from Schutze et al. [40].

Figure 29.9 Three generations of the piezoelectric direct discharge based piezo brushes. *(*Source:* Adapted from Korzec et al. [46]).

Diezo brush* PZ2

ezo brush PZ3

Figure 29.10 Diener electronic low-pressure plasma devices. (Left) DENTAPLAS MAN; (Middle) DENTAPLAS IMP; (Right) DENTAPLAS PC. *Source:* Courtesy of Diener electronic GmbH & Co KG.

clean, etch, activate, and coat implant materials to improve osseointegration (Figure 29.10). The DENTAPLAS MAN and DENTAPLAS PC devices are simple, user-friendly, entry-level plasma systems that operate at low-pressure, featuring aluminum chambers, two process gasses, and an integrated vacuum chamber (Figures 29.11 and 29.12). The key difference between these two devices is the automatic operations system of DENTAPLAS PC versus the manual operations system of DENTAPLAS MAN. Despite this, both systems can reduce microbial attachment to surfaces and increase surface energy for material activation through the generation of oxygen radicals. Using Diener low-pressure systems for plasma treatment, Fu et al. [47] report increased hydrophilicity and adhesion of osteoblasts to plasma-treated

Figure 29.11 Diener DENTAPLAS MAN. *Source:* Courtesy of Diener electronic GmbH & Co KG.

Figure 29.12 Diener DENTAPLAS PC. *Source:* Courtesy of Diener electronic GmbH & Co KG.

poly-ether-ether-ketone (PEEK) surfaces (a suitable metal-free alternative implant material) compared to untreated surfaces [47]. These results demonstrate the ability of Diener low-pressure plasma systems to biologically activate implant materials, making these devices a viable option for implant surface modification. Unlike the DENTAPLAS MAN and DENTAPLAS PC devices, DENTAPLAS IMP is a chair-side low-atmospheric pressure plasma system with an integrated vacuum pump, aluminum chamber, and one process gas (Figure 29.13). The portability of DENTAPLAS IMP makes it attractive for clinical practice, offering fast cleaning and surface activation of implants and oral prostheses prior to placement.

Conclusions

Plasma cleaning is a promising method of surface modification for implants. Plasma cleaning is capable of improving implant surface hydrophilicity, decontamination, and cell adhesion, thereby promoting osseointegration and implant vitality. Commercially, plasma cleaning is

References

- **1** Brånemark, P.-I., Breine, U., Adell, R. et al. (1969). Intra-osseous anchorage of dental prostheses:I. experimental studies. *Scandinavian Journal of Plastic and Reconstructive Surgery 3* (2): 81–100. [https://](https://doi.org/10.3109/02844316909036699) [doi.org/10.3109/02844316909036699.](https://doi.org/10.3109/02844316909036699)
- **2** Hudecki, A., Kiryczyński, G., & Łos, M. J. (2019). Chapter 7 biomaterials, definition, overview. In M. J. Łos, A. Hudecki,

& E. Wiecheć (Eds.) *Stem Cells and Biomaterials for Regenerative Medicine* (pp. 85–98). Academic Press. [https://](https://doi.org/10.1016/B978-0-12-812258-7.00007-1) doi.org/10.1016/B978-0-12-812258-7.00007-1

3 Jin, W. and Chu, P.K. (2019). Orthopedic implants. In: *Encyclopedia of Biomedical Engineering* (ed. R. Narayan), 425–439. Elsevier. [https://doi.org/10.1016/b978-0-12-80123](https://doi.org/10.1016/b978-0-12-801238-3.10999-7) [8-3.10999-7.](https://doi.org/10.1016/b978-0-12-801238-3.10999-7)

Figure 29.13 Diener DENTAPLAS IMP. *Source:* Courtesy of Diener electronic GmbH & Co KG.

advantageous for its affordability, portability, and reduced toxicity compared to conventional methods of implant surface modification. These advantages of plasma cleaning make it a viable, real-time option for implant treatment prior to insertion. To support its use in clinical practice, future studies may wish to investigate the longterm efficacy of plasma-treated implants immediately prior to insertion in vivo. Furthermore, studies may wish to investigate the clinical benefit of the plasma cleaning for bone grafting materials and oral appliances to improve scientific understandings of the applications of plasma cleaners in implant dentistry as well as an opportunity to utilize plasma cleaning for the treatment of peri-implant diseases.

- **4** Mandracci, P., Mussano, F., Rivolo, P., and Carossa, S. (2016). Surface treatments and functional coatings for biocompatibility improvement and bacterial adhesion reduction in dental implantology. *Coatings 6* (1): 7. <https://doi.org/10.3390/coatings6010007>.
- **5** Simonis, P., Dufour, T., and Tenenbaum, H. (2010). Long-term implant survival and success: a 10-16-year follow-up of non-submerged dental implants. *Clinical Oral Implants Research 21* (7): 772–777. [https://doi.](https://doi.org/10.1111/j.1600-0501.2010.01912.x) [org/10.1111/j.1600-0501.2010.01912.x.](https://doi.org/10.1111/j.1600-0501.2010.01912.x)
- **6** Karoussis, I.K., Muller, S., Salvi, G.E. et al. (2004). Association between periodontal and peri-implant conditions: a 10-year prospective study. *Clinical Oral Implants Research 15* (1): 1–7. [https://doi.](https://doi.org/10.1111/j.1600-0501.2004.00982.x) [org/10.1111/j.1600-0501.2004.00982.x.](https://doi.org/10.1111/j.1600-0501.2004.00982.x)
- **7** Karoussis, I.K., Salvi, G.E., Heitz-Mayfield, L.J.A. et al. (2003). Long-term implant prognosis in patients with and without a history of chronic periodontitis: a 10-year prospective cohort study of the ITIR dental implant system. *Clinical Oral Implants Research 14* (3): 329–339. <https://doi.org/10.1034/j.1600-0501.000.00934.x>.
- **8** Roos-Jansaker, A.M., Lindahl, C., Renvert, H., and Renvert, S. (2006). Nine- to fourteen-year follow-up of implant treatment. Part I: implant loss and associations to various factors. *Journal of Clinical Periodontology 33* (4): 283–289. [https://doi.org/10.1111/j.1600-051x.](https://doi.org/10.1111/j.1600-051x.2006.00907.x) [2006.00907.x.](https://doi.org/10.1111/j.1600-051x.2006.00907.x)
- **9** Cochran, D.L., Schenk, R.K., Lussi, A. et al. (1998). Bone response to unloaded and loaded titanium implants with a sandblasted and acid-etched surface: a histometric study in the canine mandible. *Journal of Biomedical Materials Research 40* (1): 1–11.
- **10** Wennerberg, A., Hallgren, C., Johansson, C., and Danelli, S. (1998). A histomorphometric evaluation of screwshaped implants each prepared with two surface roughnesses. *Clinical Oral Implants Research 9* (1): 11–19. <https://doi.org/10.1034/j.1600-0501.1998.090102.x>.
- **11** Zhu, G., Wang, G., and Jiao Li, J. (2021). Advances in implant surface modifications to improve osseointegration. *Materials Advances 2* (21): 6901–6927. [https://doi.org/10.1039/D1MA00675D.](https://doi.org/10.1039/D1MA00675D)
- **12** Atieh, M.A., Alsabeeha, N.H.M., Faggion, C.M., and Duncan, W.J. (2012). The frequency of peri-implant diseases: a systematic review and meta-analysis. *Journal of Periodontology 84* (11): 1–15. [https://doi.org/10.1902/](https://doi.org/10.1902/jop.2012.120592) [jop.2012.120592](https://doi.org/10.1902/jop.2012.120592).
- **13** Souza, J.C.M., Sordi, M.B., Kanazawa, M. et al. (2019). Nano-scale modification of titanium implant surfaces to enhance osseointegration. *Acta Biomaterialia 94*: 112–131.<https://doi.org/10.1016/j.actbio.2019.05.045>.
- **14** Park, J.H., Olivares-Navarrete, R., Baier, R.E. et al. (2012). Effect of cleaning and sterilization on titanium implant surface properties and cellular response. *Acta Biomaterialia 8* (5): 1966–1975. [https://doi.org/10.1016/j.](https://doi.org/10.1016/j.actbio.2011.11.026) [actbio.2011.11.026](https://doi.org/10.1016/j.actbio.2011.11.026).
- **15** Duske, K., Koban, I., Kindel, E. et al. (2012). Atmospheric plasma enhances wettability and cell spreading on dental implant metals. *Journal of Clinical Periodontology 39* (4): 400–407. [https://doi.org/10.1111/j.1600-051x.2012.01853.x.](https://doi.org/10.1111/j.1600-051x.2012.01853.x)
- **16** Lata, S., Chakravorty, S., Mitra, T. et al. (2022). Aurora Borealis in dentistry: the applications of cold plasma in biomedicine. *Materials Today Bio 13*: 100200. [https://doi.](https://doi.org/10.1016/j.mtbio.2021.100200) [org/10.1016/j.mtbio.2021.100200.](https://doi.org/10.1016/j.mtbio.2021.100200)
- **17** Foest, R., Kindel, E., Ohl, A. et al. (2005). Non-thermal atmospheric pressure discharges for surface modification. *Plasma Physics and Controlled Fusion 47* (12B): B525–B536. [https://doi.org/10.1088/0741-3335/47/](https://doi.org/10.1088/0741-3335/47/12B/S38) [12B/S38](https://doi.org/10.1088/0741-3335/47/12B/S38).
- **18** Guastaldi, F.P.S., Yoo, D., Marin, C. et al. (2013). Plasma treatment maintains surface energy of the implant surface and enhances osseointegration. *International Journal of Biomaterials 2013*: 1–6. [https://doi.](https://doi.org/10.1155/2013/354125) [org/10.1155/2013/354125](https://doi.org/10.1155/2013/354125).
- **19** Coelho, P.G., Giro, G., Teixeira, H.S. et al. (2012). Argon-based atmospheric pressure plasma enhances early bone response to rough titanium surfaces. *Journal of Biomedical Materials Research. Part A 100* (7): 1901–1906. [https://doi.org/10.1002/jbm.a.34127.](https://doi.org/10.1002/jbm.a.34127)
- **20** Albrektsson, T. (1998). Hydroxyapatite-coated implants: a case against their use. *Journal of Oral and Maxillofacial Surgery 56* (11): 1312–1326. [https://doi.org/10.1016/](https://doi.org/10.1016/s0278-2391(98)90616-4) [s0278-2391\(98\)90616-4](https://doi.org/10.1016/s0278-2391(98)90616-4).
- **21** Von Wilmowsky, C., Moest, T., Nkenke, E. et al. (2013). Implants in bone: part II. Research on implant osseointegration. *Oral and Maxillofacial Surgery 18* (4): 355–372. [https://doi.org/10.1007/](https://doi.org/10.1007/s10006-013-0397-2) [s10006-013-0397-2.](https://doi.org/10.1007/s10006-013-0397-2)
- **22** Att, W. and Ogawa, T. (2012). Biological aging of implant surfaces and their restoration with ultraviolet light treatment: a novel understanding of osseointegration. *The International Journal of Oral & Maxillofacial Implants 27* (4): 753–761. [https://pubmed.ncbi.nlm.nih.](https://pubmed.ncbi.nlm.nih.gov/22848875/) [gov/22848875/.](https://pubmed.ncbi.nlm.nih.gov/22848875/)
- **23** Danna, N.R., Beutel, B.G., Tovar, N. et al. (2015). Assessment of atmospheric pressure plasma treatment for implant osseointegration. *BioMed Research International 2015*: 1–8. [https://doi.org/10.1155/2015/761718.](https://doi.org/10.1155/2015/761718)
- **24** Tsujita, H., Nishizaki, H., Miyake, A. et al. (2021). Effect of plasma treatment on titanium surface on the tissue surrounding implant material. *International Journal of*

Molecular Sciences 22 (13): 6931. [https://doi.org/10.3390/](https://doi.org/10.3390/ijms22136931) [ijms22136931](https://doi.org/10.3390/ijms22136931).

- **25** Buser, D., Broggini, N., Wieland, M. et al. (2004). Enhanced bone apposition to a chemically modified SLA titanium surface. *Journal of Dental Research 83* (7): 529–533.<https://doi.org/10.1177/154405910408300704>.
- **26** Lai, H.-C., Zhuang, L.-F., Liu, X. et al. (2010). The influence of surface energy on early adherent events of osteoblast on titanium substrates. *Journal of Biomedical Materials Research. Part A 93* (1): 289–296. [https://doi.](https://doi.org/10.1002/jbm.a.32542) [org/10.1002/jbm.a.32542.](https://doi.org/10.1002/jbm.a.32542)
- **27** Lim, J.Y., Shaughnessy, M.C., Zhou, Z. et al. (2008). Surface energy effects on osteoblast spatial growth and mineralization. *Biomaterials 29* (12): 1776–1784. <https://doi.org/10.1016/j.biomaterials.2007.12.026>.
- **28** Zhao, G., Schwartz, Z., Wieland, M. et al. (2005). High surface energy enhances cell response to titanium substrate microstructure. *Journal of Biomedical Materials Research. Part A 74* (1): 49–58. [https://doi.org/10.1002/](https://doi.org/10.1002/jbm.a.30320) [jbm.a.30320](https://doi.org/10.1002/jbm.a.30320).
- **29** Aita, H., Hori, N., Takeuchi, M. et al. (2009). The effect of ultraviolet functionalization of titanium on integration with bone. *Biomaterials 30* (6): 1015–1025. [https://doi.](https://doi.org/10.1016/j.biomaterials.2008.11.004) [org/10.1016/j.biomaterials.2008.11.004](https://doi.org/10.1016/j.biomaterials.2008.11.004).
- **30** Bang, S.-M., Moon, H.-J., Kwon, Y.-D. et al. (2013). Osteoblastic and osteoclastic differentiation on SLA and hydrophilic modified SLA titanium surfaces. *Clinical Oral Implants Research 25* (7): 831–837. [https://doi.](https://doi.org/10.1111/clr.12146) [org/10.1111/clr.12146.](https://doi.org/10.1111/clr.12146)
- **31** Drake, D.R., Paul, J., and Keller, J.C. (1999). Primary bacterial colonization of implant surfaces. *International Journal of Oral and Maxillofacial Implants 14* (2): 226–232.
- **32** Lee, J.-H., Jeong, W.-S., Seo, S.-J. et al. (2017). Nonthermal atmospheric pressure plasma functionalized dental implant for enhancement of bacterial resistance and osseointegration. *Dental Materials 33* (3): 257–270. [https://doi.org/10.1016/j.dental.2016.11.011.](https://doi.org/10.1016/j.dental.2016.11.011)
- **33** Quirynen, M. and Bollen, C.M.L. (2005). The influence of surface roughness and surface-free energy on supra- and subgingival plaque formation in man. *Journal of Clinical Periodontology 22* (1): 1–14. [https://doi.org/10.1111/](https://doi.org/10.1111/j.1600-051x.1995.tb01765.x) [j.1600-051x.1995.tb01765.x](https://doi.org/10.1111/j.1600-051x.1995.tb01765.x).
- **34** Thevenot, P., Hu, W., and Tang, L. (2008). Surface chemistry influences implant biocompatibility. *Current Topics in Medicinal Chemistry 8* (4): 270–280. [https://doi.](https://doi.org/10.2174/156802608783790901) [org/10.2174/156802608783790901](https://doi.org/10.2174/156802608783790901).
- **35** An, Y.H. and Friedman, R.J. (2002). Concise review of mechanisms of bacterial adhesion to biomaterial surfaces. *Journal of Biomedical Materials Research 43* (3): 338–348. [https://doi.](https://doi.org/10.1002/(SICI)1097-4636(199823)43:3<338::AID-JBM16>3.0.CO;2-B)

[org/10.1002/\(SICI\)1097-4636\(199823\)43:3%3C338::](https://doi.org/10.1002/(SICI)1097-4636(199823)43:3<338::AID-JBM16>3.0.CO;2-B) [AID-JBM16%3E3.0.CO;2-B](https://doi.org/10.1002/(SICI)1097-4636(199823)43:3<338::AID-JBM16>3.0.CO;2-B).

- **36** Flynn, P.B., Higginbotham, S., Alshraiedeh, N.H. et al. (2015). Bactericidal efficacy of atmospheric pressure non-thermal plasma (APNTP) against the ESKAPE pathogens. *International Journal of Antimicrobial Agents 46* (1): 101–107. [https://doi.org/10.1016/j.ijantimicag.](https://doi.org/10.1016/j.ijantimicag.2015.02.026) [2015.02.026](https://doi.org/10.1016/j.ijantimicag.2015.02.026).
- **37** Lunov, O., Zablotskii, V., Churpita, O. et al. (2016). The interplay between biological and physical scenarios of bacterial death induced by non-thermal plasma. *Biomaterials 82*: 71–83. [https://doi.org/10.1016/j.](https://doi.org/10.1016/j.biomaterials.2015.12.027) [biomaterials.2015.12.027.](https://doi.org/10.1016/j.biomaterials.2015.12.027)
- **38** Becker, K.H., Kogelschatz, U., Schoenbach, K.H., and Barker, R.J. (2004). *Non-equilibrium Air Plasmas at Atmospheric Pressure*. CRC Press.
- **39** Berger, M.F., Bosh, K.B., Cohen, D. et al. (2021). Benchtop plasma treatment of titanium surfaces enhances cell response. *Dental Materials: Official Publication of the Academy of Dental Materials 37* (4): 690–700. [https://doi.](https://doi.org/10.1016/j.dental.2021.01.026) [org/10.1016/j.dental.2021.01.026](https://doi.org/10.1016/j.dental.2021.01.026).
- **40** Schutze, A., Jeong, J.Y., Babayan, S.E. et al. (1998). The atmospheric-pressure plasma jet: a review and comparison to other plasma sources. *IEEE Transactions on Plasma Science 26* (6): 1685–1694. [https://doi.](https://doi.org/10.1109/27.747887) [org/10.1109/27.747887.](https://doi.org/10.1109/27.747887)
- **41** Bennett, A., Urayama, T., Papangelis, K. et al. (2021). Characterisation of a cold atmospheric pressure plasma torch for medical applications: demonstration of device safety. *Applied Sciences 11* (24): 11864. [https://doi.](https://doi.org/10.3390/app112411864) [org/10.3390/app112411864](https://doi.org/10.3390/app112411864).
- **42** Yan, M., Hartjen, P., Gosau, M. et al. (2021). Effects of a novel cold atmospheric plasma treatment of titanium on the proliferation and adhesion behavior of fibroblasts. *International Journal of Molecular Sciences 23* (1): 420. [https://doi.org/10.3390/ijms23010420.](https://doi.org/10.3390/ijms23010420)
- **43** Nova Plasma Ltd (2020). Innovating Medical Solutions. Novaplasma.com. [https://novaplasma.com/](https://novaplasma.com/projects/#cold-plasma-dentistry) [projects/#cold-plasma-dentistry](https://novaplasma.com/projects/#cold-plasma-dentistry) (accessed 5 June 2023).
- **44** Berger, M.F., Cohen, D., Levit, M.M. et al. (2022). Hydrophilic implants generated using a low-cost dielectric barrier discharge plasma device at the time of placement exhibit increased osseointegration in an animal pre-clinical study: an effect that is sex-dependent. *Dental Materials: Official Publication of the Academy of Dental Materials 38* (4): 632–645. [https://doi.](https://doi.org/10.1016/j.dental.2022.02.002) [org/10.1016/j.dental.2022.02.002](https://doi.org/10.1016/j.dental.2022.02.002).
- **45** Omran, A.V., Busco, G., Ridou, L. et al. (2020). Cold atmospheric single plasma jet for RONS delivery on large biological surfaces. *Plasma Sources Science and*

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Technology 29 (10): 105002. [https://doi.org/10.1088/](https://doi.org/10.1088/1361-6595/abaffd) [1361-6595/abaffd.](https://doi.org/10.1088/1361-6595/abaffd)

- **46** Korzec, D., Hoppenthaler, F., and Nettesheim, S. (2021). Piezoelectric direct discharge: devices and applications. *Plasma 4* (1): 1–41. [https://doi.org/10.3390/](https://doi.org/10.3390/plasma4010001) [plasma4010001](https://doi.org/10.3390/plasma4010001).
- **47** Fu, Q., Gabriel, M., Schmidt, F. et al. (2020). The impact of different low-pressure plasma types on the physical, chemical and biological surface properties of PEEK. *Dental Materials 37* (1): [https://doi.org/10.1016/j.](https://doi.org/10.1016/j.dental.2020.09.020) [dental.2020.09.020](https://doi.org/10.1016/j.dental.2020.09.020).

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Dental Implant Maintenance and Prosthetic Preventive Care for Implant Longevity

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Introduction

During the delivery of implant–supported restorations, base reference values (BRVs) should be registered. These BRVs include different variables of the peri-implant tissues, prosthetic parameters, and patient outcomes (Figure 30.1).

BRVs for the peri-implant tissues:

- The soft tissue characteristics (thickness, height, location, color, gingival papilla, and attributes).
- Probing depths (measured with stable reference points in the implant or the prostheses).
- Bleeding on probing (evaluated with standardized periodontal probes).
- Mesial and distal radiographic bone levels (using as reference implant or prosthetic landmarks).

BRVs for the implant prostheses:

- The occlusal relationships (occlusal contacts and occlusal scheme)
- Interproximal relationships (interproximal contacts, embrasure spaces)
- Prostheses fit (implant–abutment/crown–abutment fit and emergence profile, both evaluated by clinical and radiographic methods)
- Integrity and quality of the implant restorations (presence of cracks, surface porosity, finishing, and contamination)
- Color matching and mimicking with the adjacent structures (including adjacent teeth and gingival tissue).

The BRVs for patient outcomes are subjective, usually measured through questionnaires filled by the patient, and can include some of the following:

- Patient satisfaction
- Patient function
- Patient's quality of life

In addition to the BRVs recorded immediately after delivery, periodical follow-ups can detect changes in the BRVs during the implants' expected functional life. As consequence of the implant/prosthesis function and the exposure to multiple environmental factors, the health of the periimplant tissues as well as their survival can be impaired [1].

Local and general factors can alter the peri-implant tissues' health. Among the local factors that can affect the health of peri-implant tissues can be included: plaque, bacteria, sub-products (toxins), residual cement and foreign bodies, material surface porosity and chemistry, and maintenance protocols. General factors that can affect the health of the peri-implant tissues are immune system status, medications, diabetes, and smoking [2]. Therefore, with longterm implantable medical devices and prostheses, the periodical evaluation of the peri-implant and prosthetic parameters is obligatory to detect and control as early as possible any alterations of the BRVs parameters [3].

When periodical evaluations are completed, any deviation from the original BRVs indicates a potential impairment of biological (mucositis, peri-implantitis), prosthetic, or patient outcomes is in progress. These changes must be addressed to return the dental implants, the peri-implant tissues, the implant restorations, and the patient to the baseline or stability (Figure 30.2).

The prevalence of biologic (mucositis and peri-implantitis) (Table 30.1) and prosthetic complications (Table 30.2) are present in different percentages. Both increase with the time of implant service [3]. Given that fluctuations between health and disease occur and co-exist over the long term,

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Figure 30.1 Base Reference Values (BRVs) recorded immediately after the insertion of the implant restorations. Parameters will be recorded periodically during follow-up visits or maintenance visits. Any changes and date of occurrence must be registered (By Delgado, Javed and Romanos).

Time (Years)

Figure 30.2 Different factors can influence dental implants, where a thin line separates health and disease. The peri-implant health and disease status are variable and adjacent to each other. Maintenance protocols can stabilize the line on the healthy side. Although health is considered the lack of disease, the disease can be present with different severity levels, which can mask the early stages of the disease. Severe disease conditions are away from health, and mild disease conditions are near to health status. The black dotted line indicates the equilibrium. The broken spaces in the line indicate periods of interruption or change. The oscillating line shows alternant health and disease. When the disease appears and is detected early, therapeutic and corrective actions are required to restore the equilibrium (By Delgado, Javed and Romanos).

Table 30.1 Compilation of the prevalence of peri-implant diseases (mucositis and peri-implantitis) upon a follow-up period of 15 years presented in the most recent systematic reviews. Mucositis is more frequent than peri-implantitis.

The prevalence of both diseases appears to increase with the time of follow-up. CI, Confidence Interval; I, Implants; NA, Not applicable; NR, Not reported; P, Patients; PPD, Peri-implant probing depth.

Table 30.2 Compilation of technical/mechanical/prosthetic complications upon a follow-up period of 15 years collated from recent systematic reviews. Two frequent complications were described in all the reviews (Screw loosening and material fractures); prosthetic designs were responsible for the variability. The complication rates increase with the time of prosthesis use, which can be related to material fatigue. Finally, cemented restorations will present higher biological complications, and screw-retained restorations will develop more mechanical complications.

follow-up evaluations are required to detect changes, and maintenance protocols are essential to support the health and longevity of implants and their restorations. Because it has been demonstrated that the incidence of peri-implant diseases is significantly reduced, and implant survival is increased when maintenance protocols are applied [3].

Definition of Implant Health, Maintenance, and Patient's General Health

Peri-implant health was defined as the absence of inflammation, bleeding on probing, swelling, suppuration, or bone loss at the Proceedings of the World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions [14]. The health of the peri-implant tissues can be sustained through implant maintenance therapy [15]. Thus, implant maintenance can be defined as all these procedures that measure the peri-implant soft and hard tissue parameters, detect and remove plaque and other risk factors, apply corrective actions, educate the patient to perform correct home care, and attempt to improve the patient's compliance [15].

Although there are different ways to describe implant maintenance, like supportive peri-implant therapy (SPIT) [16] and cumulative interceptive supportive therapy (CIST) [17], the goals are the same. They are comparable to conventional supportive periodontal treatment (SPT). Essential implant maintenance includes the following steps analog to SPT: clinical examination, radiographic evaluation, oral hygiene instructions, plaque control, and mechanical debridement [18].

Beyond peri-implant health is general health, defined by the World Health Organization (WHO) as a "state of complete physical, psychological, and social well-being and not merely the absence of disease or infirmity." Therefore, not just the peri-implant tissues but also the patient's general health, prostheses integrity, prosthesis's function, and patient's quality of life (evaluated through specific questionnaires) should be included within the goals of the implant maintenance and patient's health maintenance procedures [1].

Implant Maintenance Protocols

Since the middle of the '90s, different approaches have been recommended for the maintenance of dental implants; this occurred because some of the first long-term follow-up studies on dental implants demonstrated that a small percentage of implants could fail [20, 21]. Wilson T., in 1996, described a list of seven aspects that should be evaluated in the maintenance visit for patients restored with dental implants (Figure 30.3).

As can be observed, the maintenance visit included more than the simple evaluation of the peri-implant tissues but extended far to cover prostheses, the patient's health, and preventive and corrective steps. Regarding the examination of the peri-implant tissues, the method of probing was not described. However, the assessment of the soft tissues included bleeding on probing and detecting the presence of suppuration (Figure 30.4–30.6).

In addition, the occlusal examination evaluated, among others, the screening for evidence of wear, the evaluation of screws (looking for loosening or fractures of

Figure 30.3 List of evaluations proposed by Wilson [22]. Evaluations were covering the general and oral health, implant, peri-implant tissues, hygiene, patient's behavior toward oral hygiene, evaluation of bone levels, cleaning the prostheses, and setting the maintenance intervals.

Figure 30.4 Peri-implant tissue health after abutment connection (a). The peri-implant mucosa with good tissue stability presents capillary formation without any inflammatory reaction (b).

implants and parts), and recording patient complaints. The implant stability was also included in the evaluations when permitted by the type of restoration.

Coulthard et al. [23], described a staged approach (preventive strategies) for the patients with dental implants that included preventive approaches during implant planning, preventive actions during the implant surgery, and preventive actions during the maintenance visits (Figure 30.7).

In this proposed protocol, the implant evaluation is extensive. The mobility of the implant is considered as implant failure, manual methods, and electronic devices

(Periotest device and Osstell device) are used to evaluate the implant stability. For the radiographic evaluation, a baseline radiography is obtained at the moment of insertion of restoration using standardized holders. Posterior radiographs are obtained using the same holder to allow comparison. Landmarks are established in the implant or prostheses and changes in the bone levels are evaluated. Changes including reduced bone levels, radiolucent defects around the implant neck, body, or apex, indicate bone loss meanwhile a full radiolucent image around an implant indicates implant failure.

Figure 30.5 Peri-implant inflammatory reaction due to lack of attached mucosa without bleeding on probing (a); the tissue consistence was improved after soft tissue grafting utilizing an epithelialized gingival graft from the hard palate after 14 months of healing improving the home care and reducing the pathological probing depth (b).

Figure 30.6 Peri-implant pathology associated with probing of 7mm, suppuration and crestal bone loss (peri-implantitis).

Todescan et al. [24] suggested a maintenance protocol that started with updating of medical and dental history, followed by the clinical examination of the implant (Figure 30.8). The clinical evaluation begins with evaluating plaque index, which should be completed using plaque index scores specific to dental implants [25, 26].

Afterward, implant health is associated with no bleeding on probing combined with no changes in probing depths. Suppuration must be absent no recessions or inflamed tissues should be observed. Occlusion should provide adequate posterior support, and technical complications should not be documented. When the disease appears, appropriate treatment, including oral hygiene instructions, removal of supra- and sub-gingival plaque and calculus, occlusal

Figure 30.7 Preventive strategies for maintenance for patients with dental implants described by Coulthard et al. in 2003. In this approach, a six-month period between maintenance visits is recommended. However, the frequency of such visits is determined based on the patient's needs. The prosthesis's evaluation considered mainly mechanical factors. The implants' evaluation is focused on mobility, bone levels (evaluated in radiography), and peri-implant tissue parameters.

Figure 30.8 Guidance for maintenance and care of dental implants described by Todescan et al. [24]. The authors described four steps in which the clinical evaluation of the implants was improved compared to other protocols. The authors also described the importance of using specific plaque score indexes for implants.

Figure 30.9 Components of maintenance after implant therapy described by Mombelli [27]. The author discusses three levels of implant maintenance. The first is the patient-care level, which involves the patient's oral hygiene. The second is preventive care, and the third is the intervention therapy, administered by the healthcare professional.

adjustment, and relining of a removable prosthesis or surgery, should be completed.

In Mombelli's [27] view, there are two main levels of prevention within the implant maintenance (preventive measures provided by the patient and preventive procedures provided by the healthcare professional), plus a level of supportive care which address any detected problems (Figure 30.9).

In this protocol, for the first time, the responsibility of implant maintenance and preventive care is presented as a group effort shared between the patient and the healthcare provider, thus, certain level of patient compliance, management of existing medical conditions, and reduction of environmental risk factors (like smoking) is expected from the patient. Meanwhile, the healthcare provider provides preventive care, eliminates local risk factors, and provides intervention therapy.

Leone et al. [28], completed a cross-sectional study to evaluate the effect of patient compliance on the development of

peri-implantitis. They found that patients non-compliant with preventive measures and erratic attendance to professional preventive care presented a significantly higher probability of peri-implantitis (88% more than compliant patients).

Implant Prosthodontics Maintenance Protocols

In the previous section, different implant maintenance protocols were presented and summarized. However, none of these protocols described how the implant restorations should be evaluated and how they should be maintained. Besides, prosthetic risk factors must be controlled to ensure the prostheses' longevity and survival.

It is well known that the type of prostheses will condition the type of technical failure or mechanical complication. Walton and MacEntee [29] documented that implant–supported fixed

restorations required tightening of the prosthetic screws (27%), assisted cleaning of plaque and calculus (18%), occlusal adjustments (14%), corrections of the prosthesis contour (14%), and other adjustments like color changes, or fracture repairs (14%). These findings differed in percentages with removable implant prostheses in which contour correction was more frequent than in fixed restorations (50%), followed by tightening of screws (32%), occlusal adjustments (11%), and other corrections (7%). The most common adjustments for implant fixed restorations were reparation of material fractures (47%), resealing the screw access openings (31%), and others (22%); meanwhile, the most common repairs for removable implant prosthesis were the replacement of retentive components (55%), relines (10%), and others 35%. The author concluded that the time and cost of repair and maintenance of fixed or removable implant restorations were substantial, and removable prostheses required twice the number of repairs than fixed prostheses, and a significant part of the adjustments was required the first year of service (Figure 30.10).

Bidra et al., completed a systematic review of implant prosthetic complications and concluded that the type of prosthesis, the prosthetic components, and the restorative materials can influence the quality of the implant maintenance (provided by the health professional or during home care). In addition, Vahidi and Pinto-Sinai [30], presented an updated review of the specific complications that could be expected in the current types of removable implant–supported restorations.

The authors recommended the following measures to reduce the incidence of patients' mechanical and technical problems with implant–supported removable dentures and partial dentures:

1) The overdenture must have proper extension and support; the fitting should be reevaluated periodically, relines, or rebases if indicated.

- 2) The matrices of the attachment system must be checked and replaced periodically.
- 3) Metal reinforcement reduces base fractures; the thickness of base materials and frameworks must adequately support the teeth.
- 4) Special instructions and homecare for patients with bars.
- 5) The distal extension should not be extended beyond the second premolar.
- 6) Use more implants to support the prosthesis; thus, cantilevers will not be required, material and implant fractures could be reduced.

Finally, Bidra et al. [31] presented evidence-based clinical recommendations for the recall and maintenance of patients with removable and fixed implant–supported restorations and home-care maintenance (Figure 30.11a–c). In this protocol, for each recommendation, the strength of the available evidence is assigned. There are five categories identified with letters as follows:

- A) (evidence-based in systematic reviews of randomized controlled trials)
- B) (evidence-based in one or more randomized controlled trials)
- C) (evidence from comparative studies, cohort studies, and case-control studies)
- D) (evidence from expert committee reports).

Patient recall periods are supported by a low level of evidence (consensus and expert opinions); therefore, the best approach is to customize the recall frequency to the patient's needs. Mixed levels of evidence support the recommendations for the biological and mechanical maintenance of removable implant restorations (from A to D).

Similarly, patient recall periods in fixed implant restorations are supported by a low level of evidence (consensus

Figure 30.10 Components of maintenance for removable implant prosthesis by Vahidi and Pinto-Sinai [30]. The implant overdentures and the implant–supported removable dentures presented similar maintenance requirements. Patient satisfaction increased for patients using implant-assisted removable devices compared to conventional dentures.

Figure 30.11 (a) Guidelines for recall and maintenance of patients with implant–borne removable restorations by Bidra et al. (2016). (b) Guidelines for recall and maintenance of patients with implant–borne fixed restorations by Bidra et al. (2016). (c) Guidelines for at-home maintenance for patients with implant–supported restorations by Bidra et al. (2016).

and expert opinions), and customization of the recall frequency to the patient's needs is recommended. Mixed levels of evidence support the recommendations for the biological and mechanical maintenance of fixed implant restorations (from A to D). Those differ from the guidelines for removable in difficulty for retrievability, potential replacement of prosthetic screws, extraoral cleaning is not possible, use of occlusal appliances is recommended, and the required use of antimicrobials is considered essential in fixed implant restorations.

The home care of fixed and removable restorations requires brushing, cleaning, and using hygiene aids. In addition, the use of topical antimicrobials is recommended. The main differences are that removable implant prostheses will require intraoral and extraoral cleaning, evaluation of intraoral parts, and evaluation of removable parts (Figure 30.12a–c). Meanwhile, fixed restorations will require intraoral assessment and cleaning. Both receive benefits from occlusal devices during sleep.

In cases of implant-assisted removable partial dentures the implant abutments, the removable partial denture and their retentive elements, the abutments, and the natural teeth require detailed evaluation and care. The presence of reduced support increases the mechanical loads on the remaining teeth and implant abutments. Full palate coverage, alternative clasps, and materials can help in reducing the stress received by the supporting structures (Figure 30.13a and b).

The evidence for home-care maintenance is mid-low, specifically C (evidence from comparative studies, cohort studies, and case-control studies), and D (evidence from expert committee reports). The authors (Bidra et al. 2016) highlighted that the introduced guidelines were prepared using expert judgment and consensus. They included the best possible interpretation of best clinical practices, clinical feasibility, and patient risk-benefit ratio. Different organizations reviewed and approved these clinical practice guidelines until new evidence emerged.

Patient's Function and Quality of Life in Maintenance Protocols

Outcomes in dental implants have relied on clinical measures from the clinician's point of view [32]. Those provide quantitative information and allow health tracking but do not consider functional and psychosocial aspects of oral health [33, 34], which resulted in the development of the Oral Health-Related Quality of Life (OHRQoL) questionnaire.

Diverse patient-related outcomes can be assessed during the implant maintenance visit, including patient satisfaction, patient function, and patient quality of life (QoL). Given that patient satisfaction is influenced by deep psychosocial factors challenging to quantify [35, 36] and by the contradiction between high expectations and the clinical outcomes of the finished treatment [37], only patient function and quality of life measured through welldeveloped questionnaires are included in this section.

Figure 30.12 (a) Intraoral evaluation of an implant–supported bar (Try-in) will allow to detect passive fit, also, distances from the bar to structure to the soft tissues. (b) Intraoral inspection of the hybrid prostheses every six months of use. This will allow for the detection of fractures, wear, and other issues like plaque accumulation (−1). Also, the evaluation of the antagonist teeth or prostheses determines the degree of wear (−2). Repairs are possible but, if the wear involves loss of vertical dimension of occlusion, impaired function, or changes of the facial thirds, new prostheses should be fabricated. (c) After the removal of the hybrid prostheses, the soft tissue recession and calculus deposits indicate that changes in oral cleaning techniques, re-designing the prostheses, or changing the type of prostheses towards a removable appliance are needed.

(a)

Figure 30.13 (a) Complicated cases with limited support (teeth or implants) require alternative prosthetic solutions. An occlusal view shows only four remaining teeth with reduced periodontal support in a compromised patient requesting to preserve the remaining natural teeth. In implant-assisted removable partial dentures, a single implant can make the difference (−1). A removable prosthesis with flexible clasps can reduce the stress on the abutment teeth, a long-arm I-clasp, provides stability to the prostheses (−2). (b) A full palate coverage can increase the prostheses' stability and can reduce palatal fractures (−1). In addition, the incorporation of retentive nylon inside the locator housing (-2) can be customized to the patients' needs.

The mastication and adaptation to occlusal changes presented by Bourdiol et al. [38] described that with the occlusal changes (provided by the loss of teeth or a partial or full-mouth restoration), a patient could have normal mastication, adapt to changes (compensatory adaptation), or be incapable of proper chewing. The patient reacts differently to the occlusal changes. In the compensatory adaptation, an increased number of masticatory cycles is required to achieve the particle size comparable to the normal mastication's food bolus, changes in the jaw movement and force could be observed, but the mastication still occurs.

In mastication incapacity, the food bolus' particle sizes are too large, the patient is forced to swallow unprepared bolus, and sometimes the patient refuses some foods because they cannot be eaten. These factors can result in dietary changes, malnutrition, health, and mental issues. The same incapacity can appear when a patient has a decreased number of teeth, periodontal disease, and neurological disabilities that affect motility and muscle strength. Therefore, it is essential to monitor patients' function, expressed as the ability of proper mastication during implant maintenance.

Health Impact Profile-14

The OHIP-14 survey is a shortened version of the 49-item OHIP questionnaire [39].

It evaluates functional restrictions, pain, subjective discomfort, physical impediment, psychological disability, social disability, and handicap.

Fourteen questions from Q1 to Q14 are included as follows:

- Q1. Articulation
- Q2. Sense of taste
- Q3. Pain
- Q4. Eating
- Q5. Self-conscious
- Q6. Felt tense
- Q7. Diet unsatisfactory
- Q8. Interrupt meals
- Q9. Relax
- Q10. Embarrassed
- Q11. Irritable
- Q12. Usual work
- Q13. Less satisfied
- Q14. Unable to function.

The questions Q1, Q4, Q8, and Q14, pertain to the functional aspect (Articulation, eating, interrupted meals, unable to function), and can be used to evaluate functional improvements or functional changes in the patient. This questionnaire owns plausible reliability and constructs validity concerning subjective global oral health indicators.

Oral Implant Impact Profile

The OIIP-Q questionnaire contains fifteen questions that score 1–5, where 1 is "no satisfaction," and 5 is "satisfaction" with the treatment provided [40]. It is indicated for use in patients with single implants. This questionnaire evaluated the following groups of items: the provider's professionalism, time of operation and healing, pain, and complications, chewing and function and aesthetics, and psychology. The questions from Q1 to Q15 are as follows: Q1. Preoperative introduction, Q2. Pain during the implant surgery, Q3. Post-operatory pain, Q4. Duration of the

operation, Q5. Soft-tissue edema, Q6. Wound healing duration, Q7. Waiting time until restoration delivery, Q8. Color of implant crown, Q9. The difference of the restoration with the natural teeth, Q10. Oral hygiene maintenance, Q11. Cost of treatment, Q12. Chewing ability, Q13. Comfort, Q14. Food impaction, Q15. Dental professionalism. The questions Q12, Q13, and Q14 pertain to chewing and function. This questionnaire has shown that almost 16% of patients have issues with the prostheses function, and 8.63% of patients have issues with food impaction after eating. The use of this questionnaire also could help in the optimization of implant maintenance.

Food Intake Ability

The Food Intake Ability (FIA) Index, compares subjective mastication before and after the implant restoration is delivered [41]. The patient answered questions in the FIA questionnaire according to the following score: 0 points – "have never eaten," 20 points – "cannot chew at all," 40 points – "difficult to chew," 60 points – "cannot say either way," 80 points – "can chew some," and 100 points – "can chew well." Overall high scores represent a satisfactory function, and lower scores represent a limited/impaired function.

Concluding Remarks

Ideally, the rehabilitation of oral implants improves the quality of life and sometimes the nutritional state of patients with dental implants [42]. Given the two-directional impact of the patient's function on the implant restorations and the effect of the implant restoration on the patient's function, the implant maintenance visit should include evaluating the implant restorations and their proper function using methods that could be quantified and tracked.

We presented three questionnaires representing the implant restorations' function, which are valuable tools for clinical decision-making.

Incorporating the implant function questionnaires in the maintenance visit will change the clinician's approaches toward a more involved and compliant patient, resulting in improved implant survival and oral and general health.

References

- **1** Dawson, D. and Jasper, S. (2015). Key systemic and environmental risk factors for implant failure. *Dental Clinics of North America* 59: 25–39.
- **2** Daubert, D., Weinstein, B., Bordin, S. et al. (2015). Prevalence and predictive factors for peri-implant disease

and implant failure: a cross-sectional analysis. *Journal of Periodontology* 86: 337–347.

3 Rösing, C., Fiorini, T., Nogueira-Haas, A. et al. (2019). The impact of maintenance on peri-implant health. *Brazilian Oral Research* 33 (Suppl 1): e074.

- **4** Atieh, M.A., Alsabeeha, N.H., Faggion, C.M. Jr., and Duncan, W.J. (2013). The frequency of peri-implant diseases: a systematic review and meta-analysis. *Journal of Periodontology* 84: 1586–1598.
- **5** Derks, J. and Tomasi, C. (2015). Peri-implant health and disease. A systematic review of current epidemiology. *Journal of Clinical Periodontology* 42 (Suppl 16): S158–S171.
- **6** Muñoz, V., Duque, A., Giraldo, A., and Manrique, R. (2018). Prevalence of peri-implant disease according to periodontal probing depth and bleeding on probing: a systematic review and meta-analysis. *The International Journal of Oral & Maxillofacial Implants* 33: e89–e105.
- **7** Natto, Z.S., Almeganni, N., Alnakeeb, E. et al. (2019). Peri-implantitis and peri-implant mucositis case definitions in dental research: a systematic assessment. *The Journal of Oral Implantology* 45: 127–131.
- **8** Jung, R.E., Pjetursson, B.E., Glauser, R. et al. (2008). A systematic review of the 5-year survival and complication rates of implant–supported single crowns. *Clinical Oral Implants Research* 19: 119–130.
- **9** Romeo, E. and Storelli, S. (2012). Systematic review of the survival rate and the biological, technical, and aesthetic complications of fixed dental prostheses with cantilevers on implants reported in longitudinal studies with a mean of 5 years follow-up. *Clinical Oral Implants Research* 6: 39–49.
- **10** Papaspyridakos, P., Chen, C.J., Chuang, S.K. et al. (2012). A systematic review of biologic and technical complications with fixed implant rehabilitations for edentulous patients. *The International Journal of Oral & Maxillofacial Implants* 27: 102–110.
- **11** Rabel, K., Spies, B.C., Pieralli, S. et al. (2018). The clinical performance of all-ceramic implant-supported single crowns: a systematic review and meta-analysis. *Clinical Oral Implants Research* 18: 196–223.
- **12** Pieralli, S., Kohal, R.J., Rabel, K. et al. (2018). Clinical outcomes of partial and full-arch all-ceramic implantsupported fixed dental prostheses. A systematic review and meta-analysis. *Clinical Oral Implants Research* 29 (Suppl 18): 224–236.
- **13** Gaddale, R., Mishra, S.K., and Chowdhary, R. (2020). Complications of screw- and cement-retained implant– supported full-arch restorations: a systematic review and meta-analysis. *The International Journal of Oral Implantology* 13: 11–40.
- **14** Caton, J., Armitage, G., Berglundh, T. et al. (2018). A new classification scheme for periodontal and peri-implant diseases and conditions – introduction and key changes from the 1999 classification. *Journal of Clinical Periodontology* 89 (Suppl. 20): S1–S8.
- **15** Salvi, G.E. and Zitzmann, N.U. (2014). The effects of anti-infective preventive measures on the occurrence of biologic implant complications and implant loss: a systematic review. *The International Journal of Oral & Maxillofacial Implants* 29 (Suppl): 292–307.
- **16** Monje, A., Aranda, L., Diaz, K.T. et al. (2016). Impact of maintenance therapy for the prevention of peri-implant diseases: a systematic review and meta-analysis. *Journal of Dental Research* 95: 372–379.
- **17** Lang, N.P., Wilson, T.G., and Corbet, E.F. (2000). Biological complications with dental implants: their prevention, diagnosis and treatment. *Clinical Oral Implants Research* 11 (Suppl 1): 146–155.
- **18** Frisch, E., Ziebolz, D., Vach, K., and Ratka-Krüger, P. (2014). Supportive post-implant therapy: patient compliance rates and impacting factors: 3-year follow-Up. *Journal of Clinical Periodontology* 41: 1007–1014.
- **19** Rovesti, M., Fioranelli, M., Petrelli, P. et al. (2018). Health and illness in history, science and society. *Open Access Macedonian Journal of Medical Sciences* 6: 163–165.
- **20** Adell, R., Lekholm, U., Rockler, B., and Branemark, P.-I. (1981). A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. *International Journal of Oral Surgery* 10: 387–416.
- **21** Albrektsson, T., Dahl, E., Enbom, L. et al. (1988). Osseointegrated oral implants – a Swedish multicenter study of 8139 consecutively inserted Nobelpharma implants. *Journal of Periodontology* 59: 287–296.
- **22** Wilson, T. (1996). A typical maintenance visit for patients with dental implants. *Periodontology 2000* (12): 29.
- **23** Coulthard, P., Esposito, M., Slater, M., and Worthington, H. (2003). Preventive strategies for patients requiring osseointegrated oral implant treatment. *British Dental Journal* 195: 187–194.
- **24** Todescan, S., Lavigne, S., and Kelekis-Cholakis, A. (2012). Guidance for the maintenance care of dental implants: clinical review. *Journal of the Canadian Dental Association* 78: 1–8.
- **25** Lindquist, L.W., Rockler, B., and Carlsson, G.E. (1988). Bone resorption around fixtures in edentulous patients treated with mandibular fixed tissue-integrated prostheses. *The Journal of Prosthetic Dentistry* 59: 59–63.
- **26** Mombelli, A., Marxer, M., Gaberthuel, T. et al. (1995). The microbiota of osseointegrated implants in patients with a history of periodontal disease. *Journal of Clinical Periodontology* 22: 124–130.
- **27** Mombelli, A. (2019). Maintenance therapy for teeth and implants. *Periodontology 2000* (79): 190–199.
- **28** Leone, F.D., Blasi, G., Amerio, E. et al. (2023). Influence of the level of compliance with preventive maintenance therapy upon the prevalence of peri-implant diseases: a

cross-sectional study. *Journal of Periodontology* [https://doi.org/10.1002/JPER.23-0048.](https://doi.org/10.1002/JPER.23-0048)

- **29** Walton, J. and MacEntee, M. (1997). A prospective study on the maintenance of implant prostheses in private practice. *The International Journal of Prosthodontics* 10: 453–458.
- **30** Vahidi, F. and Pinto-Sinai, G. (2015). Complications associated with implant-retained removable prostheses. *Dental Clinics of North America* 59: 215–226.
- **31** Bidra, A., Daubert, D., Garcia, L. et al. (2016b). Clinical practice guidelines for recall and maintenance of patients with tooth-borne and implant-borne dental restorations. *Journal of Prosthodontics* 25 (Suppl 1): S32–S40.
- **32** Slade, G., Strauss, R., Atchison, K. et al. (1998). Conference summary: assessing oral health outcomes– measuring health status and quality of life. *Community Dental Health* 15: 3–7.
- **33** Locker, D. and Jokovic, A. (1996). Using subjective oral health status indicators to screen for dental care needs in older adults. *Community Dentistry and Oral Epidemiology* 24: 398–402.
- **34** Sischo, L. and Broder, H. (2011). Oral health related quality of life: what, why, how and future implications. *Journal of Dental Research* 90: 1264–1270.
- **35** Al-Omiri, M., Abu Hantash, R., Abu Yunis, M., and Lynch, E. (2012). Relationship between personality and impacts of implant treatment on daily living. *Clinical Implant Dentistry and Related Research* 14 (Suppl 1): e2–e10.
- **36** Baracat, L., Teixeira, A., dos Santos, M. et al. (2011). Patients' expectations before and evaluation after dental implant therapy. *Clinical Implant Dentistry and Related Research* 13: 141–145.
- **37** Yao, J., Tang, H., Gao, X.L. et al. (2014). Patients' expectations to dental implant: a systematic review of the literature. *Health and Quality of Life Outcomes* 12: 153.
- **38** Bourdiol, P., Hennequin, M., Peyron, M., and Woda, A. (2020). Masticatory adaptation to occlusal changes. *Frontiers in Physiology* 11: 263.
- **39** Hongxing, L., List, T., Nilsson, I. et al. (2014). Validity and reliability of OIDP and OHIP-14: a survey of Chinese high school students. *BMC Oral Health* 14: 158.
- **40** Dong, H., Zhou, N., Liu, H. et al. (2019). Satisfaction analysis of patients with single implant treatments based on a questionnaire survey. *Patient Preference and Adherence* 13: 695–704.
- **41** Kang, S., Lee, S., Kwon, H., and Kim, B. (2015). Shortterm improvement of masticatory function after implant restoration. *Journal of Periodontal and Implant Science* 45: 205–209.
- **42** Bugone, E., Vicenzi, C., Cardoso, M. et al. (2019). The impact of oral rehabilitation with implants in nutrition and quality of life: a questionnaire-based survey on self-perception. *Journal of Clinical and Experimental Dentistry* 11: e470–e475.

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