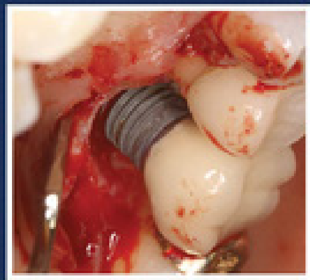
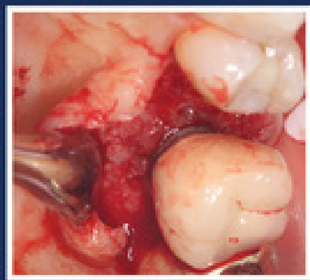
Saving Dental Implants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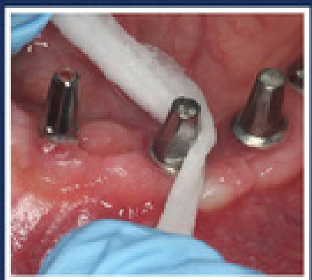


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Saving Dental Implants

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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 peri-implantitis. 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!

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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

1

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 peri-implant 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 peri-implant 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 2 mm apical to the coronal soft tissue margin and 1.0–1.5 mm coronal to the peri-implant bone crest. The mean supracrestal soft tissue, including sulcus depth, measured 3.80 mm around implants and 3.17 mm 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 peri-implant 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].

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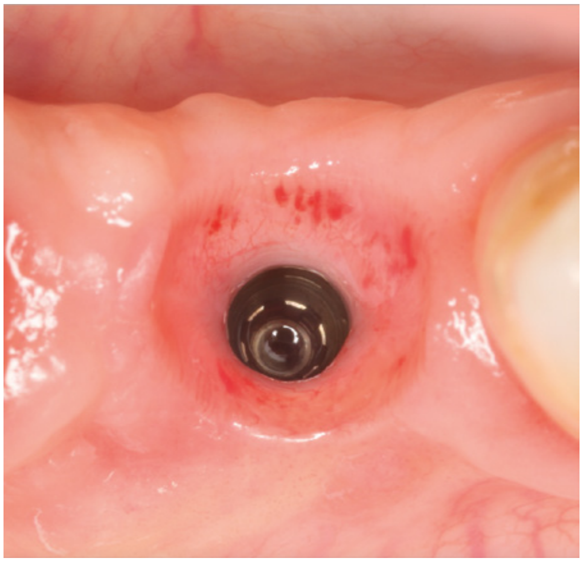

Figure 1.1 Clinical image depicting a healthy soft tissue around an osseointegrated implant. *Source:* Photo: Prof. Dr. Georgios Romanos.

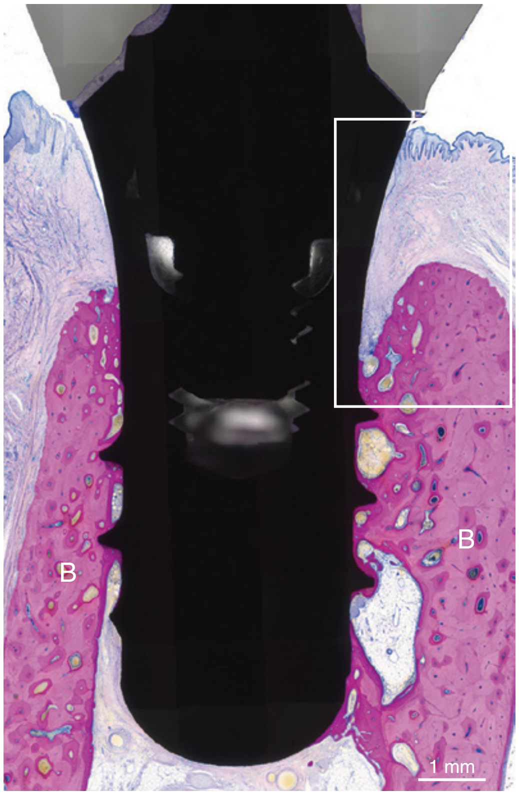

Figure 1.2 Photomigrograph of an osseointegrated titanium dental implant depicting the direct bone-implant contact and the supracrestal soft tissue implant contact. *Source:* Photo: Prof. Dieter D. Bosshardt.

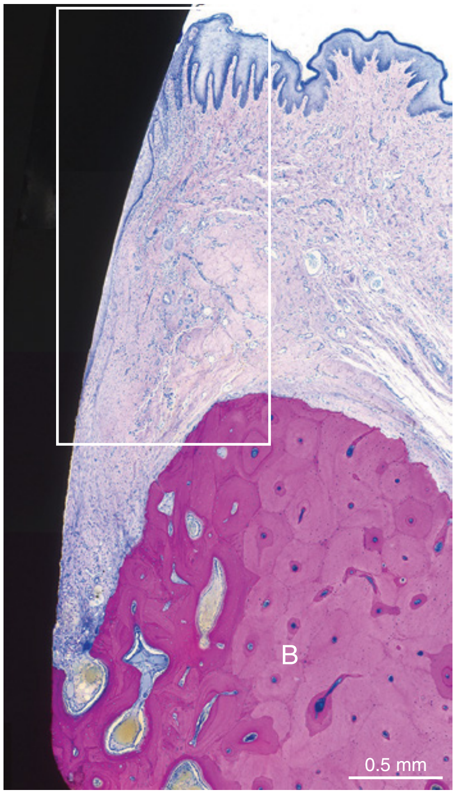

Figure 1.3 Higher magnification depicting the supracrestal peri-implant soft tissues consisting of oral and sulcular epithelium and connective tissue adhesion to the implant surface. *Source:* Photo: Prof. Dieter D. Bosshardt.

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 2 mm long, the supra-alveolar soft connective tissue was about 1.3–1.8 mm 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 two-piece 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 two-piece 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, peri-implant junctional epithelium, and soft connective tissue

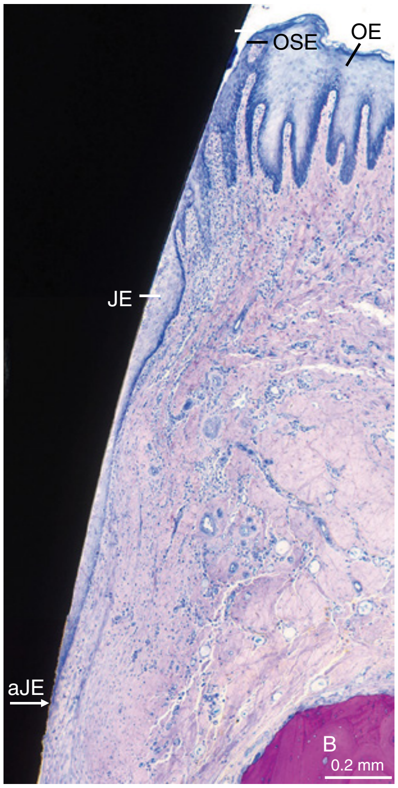

Figure 1.4 Higher magnification of the coronal portion of the supracrestal peri-implant soft tissues. The oral and sulcular epithelium are clearly visible. A more diffuse inflammatory infiltrate, located immediately adjacent to the junctional and sulcular epithelium is visible. *Source:* Photo: Prof. 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 TiO_2 coatings of one-piece titanium implants showed a similar length of peri-implant 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 non-submerged implants with clinically healthy conditions in humans and demonstrated a higher amount of collagenase-resistant 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 peri-implant 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 0.5 mm apical to the mucosal margin. At two weeks, the peri-implant junctional epithelium started to proliferate in the apical direction. After two weeks, the peri-implant 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. 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 host-tissue response.

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