Soft Tissues and Pink Esthetics
in Implant Therapy
Soft Tissues & Pink Esthetics in Implant Therapy

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Clinical Case Studies
Forewords

This work reflects the surgical and treatment procedures employed by two highly experienced clinicians. Each chapter provides readers with information on how and why to treat their patients in order to achieve and maintain an optimum esthetic outcome with implant therapy.

The diagnosis and assessment of the quantity and quality of hard and soft tissues suitable for implant placement are discussed with particular attention to the expected outcome of therapy. Placement of implants immediately after extraction can be challenging because the socket healing process is always critical. In such situations, it is crucial to assess buccal cortical bone integrity and soft tissue thickness and consider alternative options if acute infections are present. Other considerations include the use of a grafting technique to fill the gap between bone and implant and the choice of suitable biomaterials.

When specifically indicated, socket preservation techniques can be applied at the same time. This means selecting the most appropriate biomaterials, barrier membranes, and surgical techniques. In addition, soft tissue augmentation procedures are crucial when deficiencies have to be managed during implant placement to build up the site or when conditioning peri-implant tissue to receive a prosthesis. On these specific topics, Dr Cardaropoli—whom I have known since he was a university student—has made a substantial contribution to the international literature, and his works are referred to throughout the book.

This work is a must for anyone performing implant dentistry. The book offers a clear, multidisciplinary approach to dealing with implants in the esthetic zone and a thorough description of the procedures necessary for a successful long-term outcome. Every dental practice should make room on its bookshelves for this volume.

Myron Nevins, DDS
Associate Professor
Harvard School of Dental Medicine

I read this book with great interest and even some pride. I used to teach Dr Casentini, and he has achieved more in his career than any of my other students. After studying oral surgery under my guidance, he expanded his knowledge into periodontology, implant dentistry, and prosthetics. His approach is extremely constructive, and he has always combined intellectual curiosity with indubitable clinical skills and great natural talent. Last but not least, he is a remarkable teacher who is invited all over the world to share his knowledge.

The subject of this book, peri-implant soft tissue management, is very topical and has grown considerably in recent years to become the main pillar of successful prosthetic rehabilitation using implants.

Both authors are expert and creative clinicians who have contributed to the development of peri-implant soft tissue management techniques and built up 20 years of experience in the field. I was particularly struck by the book’s innovative structure: Each chapter is dedicated to a specific topic with an opening question-and-answer section that serves the twofold purpose of providing the scientific bases and answering clinicians’ frequently asked questions. A long series of lavishly illustrated case studies in the second part of each chapter explores the different clinical ramifications of the topic in question, showing various clinical situations in which readers will be able to identify cases that are similar to ones they have personally encountered.

Some of the cases Dr Casentini discusses stem from our own partnership, which dates back more than 20 years, and I am happy to have been able to contribute our joint case histories to the book. Although I was his teacher, I am proud to say that he has been a source of ongoing professional enrichment for me and has expanded my dental outlook in the broadest sense.

I am sure this book breaks new ground in terms of its structure and content. It is destined to continue providing clinicians with a very useful reference in the long term. I think it is indispensable reading for the modern dental practitioner, and I am certain that it will not disappoint its many readers.

Matteo Chiapasco, DDS, MD
Professor, Head of Oral Surgery
University of Milan

Visiting Associate Professor
Loma Linda University School of Dentistry
Preface

Why a guide to soft tissues and pink esthetics in implant therapy?

*Osseointegration*\(^1\), ie, direct contact between the implant and surrounding bone tissue, must still be considered a basic requirement of implant therapy but is no longer the only objective to be achieved in oral implantology. Today, optimal integration of the prosthetic restoration with the surrounding soft tissues must be considered a main criterion for successful implant treatment. “Pink esthetics”\(^2\) has become the holy grail of modern implant therapy. Once sufficient hard tissue volume has been built up, a series of techniques must be implemented to maintain, augment, condition, and replicate the soft tissues that will surround the future implant-supported restorations. Only in this way will it be possible to achieve satisfactory esthetic integration of hard issues with their surrounding soft tissues, ie, crowns that look like natural teeth and are not immediately recognizable as crowns on implants.

Why a book with questions, answers, and clinical case studies?

Over many years of training, during innumerable courses and lectures, we realized that the most important part of contemporary teaching is responding to our colleagues’ questions and needs and explaining treatment strategies by presenting clinical case examples.

This book, which can be considered a distillation of 20 years of clinical and teaching experience, is based on this principle. Each chapter begins with strategically chosen frequently asked questions and their answers, providing a framework for the topic covered, describing current scientific findings, and setting out guidelines for a given technique. The next section offers step-by-step analysis of clinical cases, providing detailed examples of surgical techniques applied to specific treatment options.

When selecting clinical cases, we tried to represent the different ramifications of a topic, explaining the surgical and prosthetic procedures applied to a wide range of different therapeutic needs. The range of clinical situations explored is sufficiently broad to allow readers to identify clinical cases similar to those they deal with routinely. Our clinical activities have continued along parallel tracks for many years and find a natural meeting point in our common quest for the best therapeutic option, always making the patient the focus of our decision making. Each of us contributed our own personal clinical and professional experiences to the text, but they are framed by a common working philosophy designed to enhance the reader’s learning experience.

We are sure that the systematic organization of the topics covered, the wealth of photographs, and the clear descriptions will help generate a general picture and be a useful training tool for our colleagues.

We hope we will see more clinical cases treated in a predictable manner. The true value of our professional lives is not reflected by a single action, whose success can depend on chance, but by the sum of all our actions. Randomness and chance are not equal to routine practice. It is wrong to claim success based on a chance positive result, because predictability means knowing how to achieve that result systematically. The only way to do this is to know exactly how and why this result was achieved. Without evidence-based scientific and objective knowledge, we cannot formulate proper treatment plans and achieve optimal, stable results over time.
First and foremost, my sincere thanks go to my wife, Lorena. She is my life partner; without her advice, support, and understanding, I would not be what I am or be able to do what I do.

Thank you to my children, Luca and Alessia. They are my life and my strength.

Special thanks to my parents—Annamaria and Tito. I am grateful for the way they brought me up and what they taught me and passed on to me: honesty and fairness, first and foremost.

Thank you to my grandparents. They are my roots. They have passed on, but I am sure that they are watching me from above.

Thank you to all the practice staff: Linda, Francesca, Melissa, and Sonia.

I am bound to thank my mentor, Ron Nevins, who has always been a point of reference and source of inspiration. He is an unparalleled clinician and researcher.

Thanks to all the friends and colleagues who have supported and appreciated my teaching activities over the years. They are the reason for my ongoing quest for improvement.

Thanks to the Dentalanze dental laboratory in Castagnole delle Lanze, Italy.

Unless otherwise specified, the cases I treated surgically were managed on an interdisciplinary basis by the PRoEd—Institute for Professional Education in Dentistry—team, and I would like to thank them all: Drs Alessandro Roffredo, Lorenzo Tamagnone, Andrea De Maria, Lorena Gaveglio, and Monica Ravera.

Daniele Cardaropoli

First of all, I would like to thank my family—my wife, Irene, and my children, Zeno and Sveva—for the great support they give me every day. I inevitably stole time from them to write this book, and I hope they will understand.

Thank you to my father, Augusto Casentini, and my mother, Silvana De Luca: parents, colleagues, and teachers by profession and above all by their professional ethics. Thank you for teaching me to strive to do my best for my patients every day.

Thanks to Nicolò Gruden, who is practically my second father and a very experienced clinician.

Heartfelt thanks to my teacher, Matteo Chiapasco, the most experienced, sophisticated, and creative surgeon that I’ve ever met and one of the most cultured people I have had the good fortune to know. Thank you for our fruitful partnership and for igniting my passion for teaching.

Thanks to Claudio Gatti for launching me as a speaker nearly 25 years ago.

Thanks to the team at my practice and the Narcodont Centre, who help me pursue excellence every day; thanks in particular to Nicola Balduzzi, Fabio Quarta, and Luca Pizzoni, who are fantastic colleagues and friends.

Thanks to my dental technician friends for everything they have taught me and for the outstanding results we have achieved together over the years.

A special mention to all the colleagues and dental technicians who contributed to each case in the case study section: warm thanks for all your hard work!

Paolo Casentini
We are what we repeatedly do. Excellence, then, is not an act, but a habit.

Aristotle
Quality and Quantity of Peri-implant Soft Tissue
Questions:

1. What is the biologic significance of peri-implant soft tissue?
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5. Is biologic width present around implants?
6. What are the clinical implications of forming biologic width around implants?
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9. What is the role of keratinized tissues around implants?
What is the biologic significance of peri-implant soft tissue?

The creation of a soft tissue barrier around a dental implant at the point where it emerges into the oral cavity is an important stage in the process of rendering the implant functional and ensuring the esthetic integration of the implant-supported prosthetic restoration (Fig 1-1). Maintaining this seal in a condition of health is critical to the function and long-term prognosis of the implant. The ultimate purpose of the peri-implant soft tissue seal is to protect the underlying bond between the implant and bone tissue created through the osseointegration process.

![Physiologic appearance of peri-implant soft tissues around different implant types. (a and b) Bone-level titanium implants. In these cases the depth of the peri-implant mucosal tunnel, i.e., the distance from the gingival margin to the implant connection, is greater. (c) Tissue-level titanium implant. In these cases the peri-implant mucosal tunnel is more shallow. (d) Single-component zirconia implant. With tissue-level implants, the implant prosthetic platform is located closer to the surface in a juxtagingival or slightly subgingival position.](image)

How does soft tissue heal around an implant?

Formation of a transmucosal or peri-implant attachment begins with the implant placement for single-component implants (Fig 1-2). The epithelial cells at the margin of the surgical flap adapted to the implant or abutment neck proliferate and migrate to cover the underlying connective tissue and adhere to the implant or abutment surface, forming a junctional epithelium. Apical migration of the epithelial cells ends at a band characterized by dense connective tissue and located immediately above the bone ridge, which also comes into contact with the implant surface (Fig 1-3). For two-piece implants, formation of the peri-implant attachment begins at surgical reopening and abutment attachment (Fig 1-4).
How does soft tissue heal around an implant?

Fig 1-2 (a to d) The peri-implant mucosal seal around transgingival or single-component implants begins to form immediately after implant insertion, when the soft tissues are adjusted to fit the smooth implant neck by means of sutures.

Fig 1-3 Histologic evaluation of peri-implant hard and soft tissue healing in a dog model 12 weeks after insertion. (a) With submerged healing, the implant achieves secondary stability through the osseointegration process. The soft tissues above the implant, consisting of epithelium and connective tissue, completely cover the head of the fixture. (b) After reopening and abutment connection, the soft tissues adapt around the abutment and heal by creating a mucosal tunnel consisting of an epithelial attachment and connective tissue attachment up to the first contact between bone and implant. (Courtesy of Prof J. L. Calvo Guirado, Murcia, Spain.)
Fig 1-4 (a to h) For two-component bone-level implants, submerged healing is used, and the inner implant cavity is initially sealed by means of a surgical screw. On reopening, transmucosal healing screws are then positioned. In these cases, a peri-implant soft tissue seal is therefore formed after the second surgical stage.
What are the histologic characteristics of peri-implant soft tissues?

Various histologic studies on animal and human models have allowed detailed description of the interface between soft tissues and titanium implants. This is characterized by:

- **Junctional epithelium:** Epithelial cells adhere to the implant surface through a basal lamina less than 200 nm thick through the formation of hemidesmosomes, as occurs with natural teeth. The apicocoronal extension of the junctional epithelium is 2 mm on average. Histologic examination of human biopsies has often detected the presence of an inflammatory infiltrate dominated by T lymphocytes below the junctional epithelium, although in the absence of clinical signs of soft tissue inflammation and bone resorption. The composition of this inflammatory infiltrate, which acts as a physiologic barrier against external antigenic stimuli, is very similar to that present in periodontal soft tissues in natural teeth.

- **Supracrestal connective tissue:** The supracrestal connective tissue, which is approximately 1.5 mm high, seems to be the most important component of the peri-implant seal. The formation of this connective tissue layer (50 to 100 µm in thickness) adhering to the implant surface, which is packed with collagen fibers, is poorly vascularized, contains hardly any cells, and has characteristics similar to those of scar tissue, limits the apical migration of the overlying epithelium. Unlike the tooth connective tissue attachment, where the connective tissue fibers mainly grow at right angles to the root surface so they can insert into the root cement, peri-implant connective tissue fibers usually run parallel to the implant surface and can adopt a circular pattern. In the remaining connective tissue portion (excluding the layer adhering to the implant), connective tissue fibers run in different directions, and the cellular and vascular components are higher.

![Fig 1-5](https://example.com) Histologic evaluation in a beagle model at the interface between the implant and supracrestal soft tissues 6 weeks after implant placement. (a) The junctional epithelium extends from the sulcus in an apical direction, where it is delimited by the connective tissue attachment, which ends apically at the point of first contact between bone and implant (original magnification ×10). (b) Polarized light clearly shows the direction of the collagen fibers within the supracrestal connective tissue and at the point of connective tissue attachment (original magnification ×40). (Courtesy of Prof G. Cardaropoli, Turin, Italy.)
What are the main differences between a natural tooth and an implant at the supracrestal level?

The soft tissues around implants and teeth is very similar and in both cases is more or less keratinized (depending on the presence or absence of keratinized gingiva). The subgingival interface between the soft tissues and the tooth or implant is represented by a junctional epithelium in the coronal component (approximately 2 mm wide) and supracrestal connective tissue in the apical direction (approximately 1 to 1.5 mm wide). In both cases, the connection with the junctional epithelium takes place through basal lamina and a layer of hemidesmosomes. In natural teeth, the apical limit of the junctional epithelium is the cementoenamel junction. In the soft tissue–implant interface, the epithelium stops about 1.5 mm short of the bone crest at the supracrestal connective tissue band. In natural teeth, the connective tissue fibers are predominantly horizontal and insert into the root cement (Fig 1-6).

Because implants lack a periodontal ligament, connective tissue fibers appear to originate from the bone crest periosteum, and their growth pattern is parallel to the implant surface. In the supracrestal area, the connective tissue seems to adhere closely to the thin layer of titanium oxide covering the implant surface. Connective tissue fibers growing with a circular pattern around the implant have been identified. Peri-implant supracrestal connective tissue is richer in collagen fibers, but its cell population (fibroblasts) and vascularization are lower than those of natural teeth, having the properties of scar tissue. Any fibroblasts at this level perform the function of maintaining and, if necessary, restoring cohesion between the connective tissue and implant surface.

The lower vascularization of the implant connective tissue is explained by the fact that the natural teeth have a twofold source of vascularization, from the supraperiosteal vessels and the periodontal ligament vascular plexus, while the latter source is not present in implants. The connective tissue attachment and supracrestal connective tissue both perform a barrier function in natural teeth and implants. The early response to a buildup of bacterial plaque, ie, the composition of the inflammatory infiltrate and the type of lesions from a histologic viewpoint, is entirely similar in periodontal and peri-implant soft tissues (Fig 1-7).
Is biologic width present around implants?

In natural teeth, the supracrestal soft tissues perform the function of protecting the periodontium (made up of root cementum, periodontal ligament, and alveolar bone) from external attack. From an anatomical viewpoint, supracrestal tissues consist of the sulcular epithelium, junctional epithelium, and connective tissue attachment. Together, the junctional epithelium and connective tissue attachment make up the biologic width, which tends to have constant dimensions.

There is evidence of the presence of soft tissue biologic width around osseointegrated implants (Figs 1-8 and 1-9). The average biologic width is approximately 3.0 to 3.5 mm, of which 2 mm is the junctional epithelium and about 1.0 to 1.5 mm is supracrestal connective tissue. In an experimental animal model it has been shown that if supracrestal soft tissue thickness is reduced to less than 3 mm, the healing process will still tend to re-create a biologic width of 3 mm, which in this case will involve some bone ridge resorption, with the formation of an angular bony defect near the implant platform. Formation of this soft tissue band will subsequently protect the underlying bond between the implant and bone tissue, which is known as osseointegration. Whether the implant positioning involves one or two surgical stages does not seem to affect biologic width. The type of implant system used (ie, two component, bone level versus single component, tissue level) does not appear to have any impact either.

It can therefore be concluded that, as with the interface between soft tissues and natural teeth, a biologic width has been identified around implants (Fig 1-10). It measures approximately 3 mm and tends to be re-created by reduction of the bone ridge if the soft tissue height is insufficient.

Fig 1-7 Histologic appearance of periodontal tissues around a natural tooth (a) and peri-implant soft tissues around a transmucosal single-component implant (b). (Courtesy of Prof F. Schwarz, Frankfurt, Germany.)
Fig 1-8 Biologic width is absent when the implant is still submerged (a), forming after connection of the prosthetic components and loading (b).

Fig 1-9 Histologic image of peri-implant soft tissues contributing to biologic width formation. The image shows oral epithelium (A), sulcular epithelium (B), epithelial attachment (C), and connective tissue attachment in the supracrestal portion (D). (Courtesy of Prof G. Cardaropoli, Turin, Italy.)
Formation of a peri-implant mucosal seal around an implant positioned using a single-stage protocol

Fig 1-10  (a) A titanium implant is inserted at bone level in a mandibular first molar site by detaching a full-thickness flap. (b) A healing abutment is immediately screwed onto the implant head, and the soft tissues are fitted and sutured around it. (c) An intraoral radiograph shows the correct implant position in relation to the marginal bone level. (d) Twelve weeks later, after osseointegration, a newly created peri-implant mucosal seal is evident after removing the healing abutment. (e) Enlarged image of the peri-implant mucosal tunnel shows that these tissues are perfectly healthy, without any sign of superficial inflammation or bleeding. (f) Follow-up photograph 5 years after loading. Osseointegration of the implant and mucosal seal quality help to maintain the peri-implant tissues in perfect health with a sufficiently thick band of keratinized gingiva. (g) A radiograph taken 5 years after loading shows the stability of the marginal bone level.
What are the clinical implications of forming biological width around implants?

The need for a peri-implant soft tissue seal with predominantly consistent dimensions obviously has clinical implications. Limited initial soft tissue thickness correlates to greater marginal bone loss around the coronal aspect of the implant, which could represent a potential risk for the subsequent development of peri-implant problems. A controlled clinical study on humans confirmed results found in animal models: when peri-implant soft tissue thickness is limited, ie, less than 2 mm, peri-implant bone resorption is greater (1.38 mm) than that recorded when initial soft tissue thickness is greater (0.25 mm). A subsequent clinical study conducted by the same authors to assess the behavior of bone-level implants inserted in posterior mandibular sectors using a single-stage protocol examined the possibility of preventing bone resorption in the presence of thin tissues by inserting an allogeneic dermal membrane to increase tissue thickness. Peri-implant bone resorption was similar around implants with thick tissues and those with initially thin soft tissues (< 2 mm) increased by the insertion of an allogeneic dermal membrane. If the initially thin tissues (< 2 mm) were not increased, greater initial bone resorption was recorded. Therefore, the clinical recommendation is to increase the thickness of peri-implant soft tissues surgically if they are initially thin. Soft tissue thickness can be increased by an autologous connective tissue graft or the use of biomaterials such as collagen matrices (Figs 1-11 to 1-14).

**Fig 1-11** Initial soft tissue thickness can be easily assessed using a periodontal probe marked in millimeters after detaching a full-thickness buccal access flap.
What are the clinical implications of forming biologic width around implants?

Fig 1-12  (a) An edentulous area around the mandibular first molar has an atrophic ridge with thin gingival tissue. (b and c) An osseointegrated implant is placed, and a porcine collagen matrix (Fibro-Gide, Geistlich) is grafted to increase gingival thickness. (d) The gingival tissue is then sutured around the healing abutment. (e) Three months later, the implant is osseointegrated and the soft tissues have matured. (f) Removal of the healing abutment reveals a healthy transmucosal tunnel of well over 2 mm, without signs of inflammation and ready for impression procedures.

clinical case 2

Soft tissue augmentation simultaneous with implant placement in a guided bone regeneration site. At sites that have undergone previous bone augmentation, soft tissues may be thinner as they have been stretched to cover the reconstructed area.

Fig 1-13  (a and b) In this case, in which an implant was being inserted in an area that had previously undergone guided bone regeneration, measurement of soft tissues after crestal incision showed reduced thickness (approximately 2 mm).
Once the implant was placed, soft tissue thickness was increased by means of a connective tissue graft that was harvested from the palate, de-epithelialized, and fixed to the site by trailing suspended sutures. (g) Passive flap advancement for complete graft coverage. (Surgery by Dr P. Casentini.)
What are the clinical implications of forming biologic width around implants?

Soft tissue augmentation using a 3D collagen matrix simultaneous with implant placement

**Fig 1-14** (a) Edentulous mandibular left first molar site with ridge atrophy coinciding with a soft tissue deficiency. The treatment plan involves insertion of a single implant. (b) After lifting a full-thickness flap, the implant site is prepared for insertion of a 4.8-mm-diameter, 12-mm-long tapered implant (SLActive Bone Level Tapered, Straumann). (c to e) After implant placement, a distobuccal peri-implant bone defect remains, which is managed with the use of a biomaterial with added collagen (Bio-Oss Collagen, Geistlich) and a resorbable membrane (Bio-Gide, Geistlich). (f) A 6-mm-thick porcine collagen matrix (Fibro-Gide, Geistlich) is selected to augment the supracrestal soft tissue thickness. (g and h) The matrix is trimmed to fit the site anatomy while maintaining the original thickness. The matrix absorbs the blood from the surgical site within a few seconds. The process of soft tissue regeneration begins as soon as the clot is stabilized.
clinical case 3 (cont)

Fig 1-14 (cont) (i) The flap is advanced passively over the matrix in order to obtain healing by first intention and closed using a combination of horizontal mattress and simple interrupted sutures. (j) Radiograph taken at the end of stage-one surgery. (k to n) Clinical images after 1, 2, 4, and 8 weeks, respectively, showing good progression of wound healing with maintenance of the augmented volume. (o) After 3 months, stage-two surgery can be initiated. (p) After incision, the increased soft tissue thickness can be observed.
What are the clinical implications of forming biologic width around implants?

Fig 1-14 (cont) (q and r) A healing cap is placed on the implant to allow tissue healing and the creation of biologic width. (s and t) Six weeks later, the mucosal tunnel is thick and healthy, measuring 4 mm in height. It creates a seal around the implant and limits marginal bone remodeling. (u) Follow-up clinical image showing formation of a stable mucosal tunnel after the screw-retained ceramic crown has been placed. (v) The radiograph also reveals stable marginal bone levels.
What is meant by platform switching and what is its biologic significance?

Another potential factor in peri-implant bone remodeling that occurs at an early stage, in addition to adequate biologic width, is the presence of a microgap between the implant and prosthetic abutment in two-component implants. Some authors have demonstrated the presence of predominantly anaerobic bacteria (cocci and a smaller percentage of rods) in two-component systems and the consequent presence of an inflammatory infiltrate in the microgap. This gives rise to vertical bone remodeling up to the level of the first implant thread16,17 (Fig 1-15).

The problem is significantly reduced by using the platform-switching technique18–20: Abutments of a smaller diameter than the implant platform are used to create a bone-level implant-abutment junction that is shifted inward (Fig 1-16). The change in the horizontal relationship between the external border of the implant and the prosthetic abutment creates a dimensional mismatch that moves the cellular inflammatory infiltrate away from the crestal bone because the microgap is no longer located on the external implant profile. This significantly reduces physiologic peri-implant bone remodeling after abutment or healing screw connection (Figs 1-17 to 1-23). However, the mechanism of preserving crestal bone through platform switching does not seem to work when the tissues are very thin21; the need to have sufficient biologic width therefore seems to play a predominant role.

![Fig 1-15 Sequence of radiographs showing the way that peri-implant bone remodeling typically evolves with a two-component or bone-level implant after transmucosal component connection. Bone remodeling, absent when the implant is submerged and a screw cap is present (a), begins after connection of the healing screw (b) and stabilizes at the first implant thread after connection of the implant abutment (c) and definitive prosthetic loading (d).]
**Fig 1-16** (a) Connection between implant and abutment according to the concept of platform switching. Scanning electron microscope image of (b) a standard implant-abutment connection and (c) a connection made by means of platform switching.

**Fig 1-17** (a) Standard implant-abutment connection: The presence of a microgap, and thus an inflammatory infiltrate, gives rise to marginal bone remodeling. (b) Implant-abutment connection according to the concept of platform switching: Inward horizontal displacement of the microgap prevents marginal bone remodeling.
Fig 1-18  Six-month radiographic and histologic follow-up assessment of an implant positioned in human bone using platform switching. The implant threads show close contact with the surrounding bone, indicating a successful osseointegration. (a) The microcomputed tomography image shows a good level of bone-implant contact. (b and c) Histologic evaluation reveals the absence of apical migration of the junctional epithelium around the implant threads. The bone-implant contact consists of a combination of newly formed bone and native bone. The buccal and lingual bone levels match the original platform position at the time of insertion, without subsequent resorption. (Courtesy of Dr M. Nevins, Boston, Massachusetts.)

Fig 1-19  Eighteen-week follow-up assessment of two implants positioned in a dog mandible using platform switching. (a) Radiographic view. (b) The histologic images reveal circular collagen fibers at the level of the implant platform. Mature connective tissue and vessels can be observed near the titanium surface. The collagen fibers grow at right angles, emerging from the peripheral soft tissue area. This set of radial fibers changes course near the implant surface and grows in a circular pattern to create continuity. (c) The polarized light image shows the presence of fibers above the interimplant bone septum. These fibers seem to connect the two adjacent titanium surfaces. (Courtesy of Prof X. Rodriguez, Barcelona, Spain.)

Fig 1-20  (a and b) Clinical and radiographic images of a maxillary central incisor implant prosthetically treated by means of a zirconia abutment with a platform-switched connection. Note that the peri-implant bone level has been maintained.
Fig 1-21 (a and b) Comparison between postoperative radiograph and follow-up radiograph taken 5 years after prosthetic loading with use of the platform-switching concept, revealing the absence of peri-implant bone resorption.

**clinical case 4**

Platform switching: Stability of the mucosal seal and peri-implant bone level in the esthetic zone

5 years after prosthetic loading

Fig 1-22 (a to c) Six months after extraction and simultaneous ridge preservation procedures (see chapter 5), an implant (bone-level 3.3 × 10 mm, Straumann) is inserted in the maxillary right central incisor site using a prosthetically guided technique.
Peri-implant bone volume is further augmented by means of guided bone regeneration using autologous bone mixed with deproteinized bovine bone (Bio-Oss) and a collagen membrane (Bio-Gide). After conditioning with a screw-retained provisional crown, the peri-implant soft tissues are ready for placement of the definitive restoration. Buccal and occlusal views reveal favorable tissue architecture and the complete absence of inflammation. This soft tissue seal performs the task of protecting the underlying implant-bone connection without any inflammation. Clinical and radiographic images after placement of the definitive crown.
What is meant by platform switching and what is its biologic significance?

Fig 1-22 (cont) (k to m) Clinical and radiographic images after 5 years. The precise, stable implant-crown connection achieved through platform switching made it possible to maintain a stable peri-implant bone level. The stability of the soft tissue seal and profile can also be appreciated. (n and o) The ultimate goal of any prosthetic rehabilitation using implants must always be favorable integration of the implant-supported restoration with the surrounding tissues, smile, and the patient’s facial appearance. (Implant surgery and prosthetic rehabilitation by Dr P. Casentini; prostheses by Mr A. Schoenenberger.)
clinical case 5
Platform switching: Stability of peri-implant mucosal seal and bone level 10 years after prosthetic loading in the posterior mandible

Fig 1-23 Clinical (a) and radiographic (b) views of an edentulous mandibular right first molar site. Following the lifting of a full-thickness flap (c), a tapered titanium implant (Osseotide Certain Tapered, 5 × 13 mm, Zimmer Biomet) is inserted at the bone ridge level (d). (e and f) After inserting a screw cap, the implant is left to heal in a completely submerged position. (g) Three months after placement, when osseointegration is complete, the stage-two surgery commences with the raising of a full-thickness flap. (h) A healing abutment with a diameter (4 mm) smaller than that of the implant (5 mm) is screwed onto the implant head, applying the concept of platform switching.
Fig 1-23 (cont) (i) Six weeks later the soft tissues are healed, and a transfer impression can be taken of the newly created mucosal tunnel. (j) The implant undergoes prosthetic loading after inserting a 4-mm-diameter titanium abutment (GingiHue, Zimmer Biomet) and cementing a provisional resin crown. (k) A radiograph shows that the implant and abutment are out of horizontal alignment with the marginal bone level at the time of loading. (l) When the definitive ceramic crown is cemented in place, the peri-implant soft tissues are optimally shaped and show an acceptable band of keratinized tissue. (m) The radiograph shows the marginal bone level 1 year after loading. (n) Clinical image 5 years after loading, highlighting the health of the peri-implant soft tissues. (o) A radiograph taken 5 years after loading shows that the marginal bone level has moved in a coronal direction. (p) A radiograph taken 10 years after loading shows that platform switching has helped to gain bone. The marginal bone level has moved in a coronal direction to the level of the implant-abutment connection.

What is meant by platform switching and what is its biologic significance?
What are the dynamics of soft tissue healing around immediate implants?

Immediately after extraction, soft tissue healing at the site inevitably leads to the formation of a mucosal barrier consisting of junctional epithelium and connective tissue attachment. This occurs regardless of whether implant placement is carried out using a flapless or a flap technique and whether the implant undergoes transmucosal healing through the use of a healing abutment or is treated using an immediate prosthetic loading technique. The structural and histologic characteristics of the original biologic width, intended to protect the natural tooth, undergo changes during the first few weeks following tooth extraction and implant placement.  

Four weeks after surgery, the keratinized oral epithelium is continuous with the junctional epithelium. The connective tissue apical to the epithelial cells has a dense network of collagen fibers with few vascular structures and inflammatory cells. In the innermost area, the connective tissue fibers are already lined up parallel to the implant axis. There is also nearly a total absence of blood vessels, making the tissue scarlike (Figs 1-24 and 1-25).

**Fig 1-24**  
(a) Maxillary premolar site prepared for implant placement immediately after extraction.  
(b) After implant placement, the bone-implant gap is filled with biomaterial and protected with a resorbable membrane.  
(c) A healing abutment is immediately screwed onto the implant head to achieve transgingival healing.  
(d and e) Healing of peri-implant soft tissues after 6 weeks, with re-establishment of biologic width.

**Fig 1-25**  
(a and b) Histologic evaluation of biologic width formation in implants positioned immediately after extraction in a beagle dog model. Eight weeks after transmucosal implant placement, the peri-implant mucosa is covered by a well-keratinized oral epithelium continuous with the long junctional epithelium and in contact with the abutment and the smooth portion of the implant collar. Connective tissue is interposed between the apical cells of the barrier epithelium and the bone ridge. It is free of inflammatory cells but rich in mesenchymal cells, with a collagen fiber network organized circularly around the implant. Collagen fibers close to the titanium surface tend to be aligned obliquely or parallel to the implant, while fibers located some distance away tend to be at right angles to the titanium surface. (Courtesy of Prof. J. L. Calvo Guirado, Murcia, Spain.)
What is the role of keratinized tissues around implants?

There is no absolute consensus in the scientific literature about the role played by keratinized tissue in maintaining healthy conditions in peri-implant tissues. Nevertheless, a growing number of clinical studies support the idea that a band of peri-implant keratinized tissue measuring at least 2 mm may promote healthy conditions and a favorable long-term outcome. In particular:

- When a peri-implant keratinized tissue band is absent or measures < 2 mm, patients report discomfort during brushing, particularly for mandibular rehabilitations.
- Under the same anatomical conditions (keratinized tissue < 2 mm), higher levels of plaque and bleeding on probing were recorded (Fig 1-26).
- With no keratinized tissue, the peri-implant soft tissues showed a higher incidence of recession and biologic complications.
- Implant sites with a band of keratinized tissue measuring less than 2 mm showed a higher incidence of peri-implantitis.

In general, it is therefore recommendable to achieve a band of keratinized tissue measuring at least 2 mm around the implants. Chapter 7 analyzes the different surgical techniques and time frames for increasing the peri-implant keratinized tissue band.

**Fig 1-26 (a to e)** Pain on brushing and greater plaque buildup with bleeding on probing and a tendency toward recession were noted around the implant inserted in the mandibular right first premolar site, which lacked peri-implant keratinized tissue. An absence of keratinized tissue represents a risk factor for the development of peri-implant biologic complications.
References


Recommended Reading
