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MANUAL OF CLINICAL IMPLANTOLOGY

En collaboration avec

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INTRODUCTION

History of dental implantology from antiquity to the present day

M. Davarpanah, S. Szmukler-Moncler, P. Rajzbaum

Objectives

At the end of the introduction the reader should be able to describe:

- The different steps of the development of implantology
- The specificities of these steps.

Replacing lost teeth with a prosthetic device has been a human concern since the dawn of time. Many archaeological discoveries attest to this throughout the history of mankind and in all places. The devices are of various origins, such as mineral, animal, and human (Bremner, 1964; Ouvreard, 1987; McKinney, 1991). Six distinct periods characterize the evolution of implantology (McKinney, 1991):

- Antiquity.
- Medieval period.
- Fundamental period.
- Pre-modern period.
- Modern period.
- Contemporary period.

Antiquity (BC to 1000 AC)

The first attempts to use implants were made at the time of the Ancient Egyptian dynasties and pre-Columbian civilization.

Geographic location

Traces of this period have been found in Africa (Egypt), Latin and Central America (Mayas, Aztecs, Incas), and in the Middle East.

Materials used

Animal teeth or teeth carved in ivory were used.

Special features

Radiographic examinations of exhumed skulls showed good osseointegration of artificial roots made of carved ivory (pre-Columbian culture). In Egyptian culture, toothless corpses were treated before mummification.

Medieval period (1000–1800 AC)

During this period, implantology was essentially limited to transplantation.

Geographic location

Europe.

Materials used

Human teeth.

Special features

Transplantation was carried out from one patient to another by barber surgeons. Teeth were taken from members of the disadvantaged social classes. From the beginning of the 18th century, the risk of infection and bacterial contamination are mentioned.

Fundamental period (1800–1910)

Endosseous implantology truly begins at this time.

Geographic location

North America.

Materials used

Gold, porcelain, wood, and metal (platinum, silver, tin).

Special features

In 1809, Maggilio placed a gold implant in a post-extraction socket. The restauration was only realized after tissue healing.

Biocompatibility and primary stability principles were developed by Berry in 1888. The latter insisted on the need for immediate stability of the implant and the use of 'safe' materials, avoiding disease transmission of any kind.

Premodern period (1910–1930)

Payne and Greenfield were the precursors of implantology (at the beginning of the 20th century).

Geographic location

North America.

Materials used

Gold, porcelain.

Special features

Payne described the implantation of a cylindrical basket made of gold. This implant was placed after the diameter of the socket had been enlarged with a drill. Rubber filled the gaps. A crown with its porcelain post was immediately fixed in the inner, hollow part of the implant (Fig 1). Greenfield (1913) described a hollow cylinder made of rough porcelain. A delayed loading of six to eight weeks was suggested and the importance of a bone implant close connection was emphasized. A comparison with the principles of orthopedic surgery was made and 'clean' surgery and delayed loading were discussed.

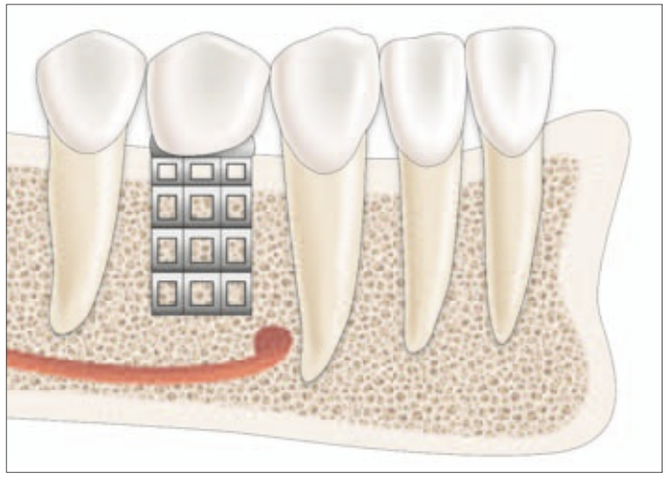


Fig 1 Basket implant of the modern period. Surgery is performed in two stages and the implant is submerged. During the first stage, osseointegration is obtained. The prosthetic connection is made after the second surgery.

Modern period (1930–1978)

This period really begins in the late 1930s. It is characterized by the study of different biomaterials and the introduction of surgical and prosthetic innovations.

Geographic location

Europe and North America.

Materials used

Porcelain, vitallium alloy, titanium.

Special features

Three types of implants were developed.

Endosseous implants I

In 1939, Strock (1939) created a screw implant made of vitallium alloy (Fig 2) and then developed (Strock & Stock, 1949) the endodontic implant (Figs 3a, b). Satisfactory results were reported at 17 years of age and a histologic study was carried out for the first time on dogs.

Subperiosteal implants

These were developed in 1941 by Dahl in Sweden. The first subperiosteal implants had standard shapes with extensive surfaces. They were locally adapted according to the bone environment. In 1951, Lew was the first to make direct bone impressions to ensure a more precise adaptation of the subperiosteal implant to the implant site (Figs 4a, b). In a second step, Weinberg and Linkow described the unilateral subperiosteal implant, which is smaller in extent (Linkow & Cherchève 1970). Several other modifications were made later. For example, James proposed the use of a support at the level of the rising branches of the mandible to prevent the subperiosteal implant from sagging. James was one of the first to suggest the use of the dental scanner to visualize a three-dimensional reconstruction of the mandible or maxilla. The latter made it possible to avoid taking an impression in direct contact with the bone.

Endosseous implants II

From the 1940s onwards, different shapes of implants were created. The Formiggini helicoidal spiral implant developed in 1947 was made of stainless steel or tantalum. Cherchève (1962), inspired by Formiggini, developed a double helix spiral implant (Figs 5a, b). It comes with a surgical kit for its insertion. Scialom promoted a tripod implant (needle implant). The three parts of

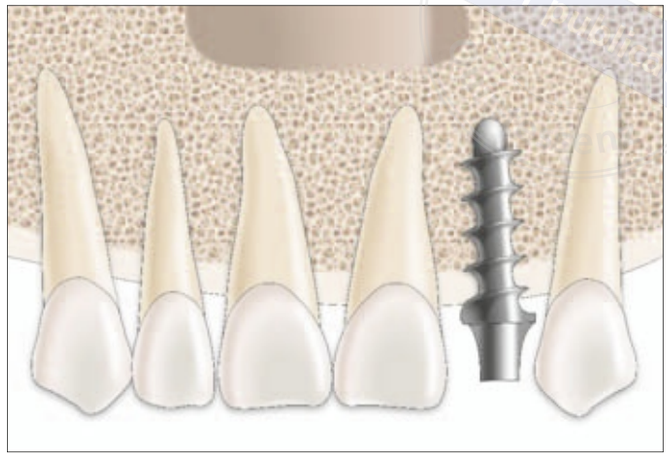
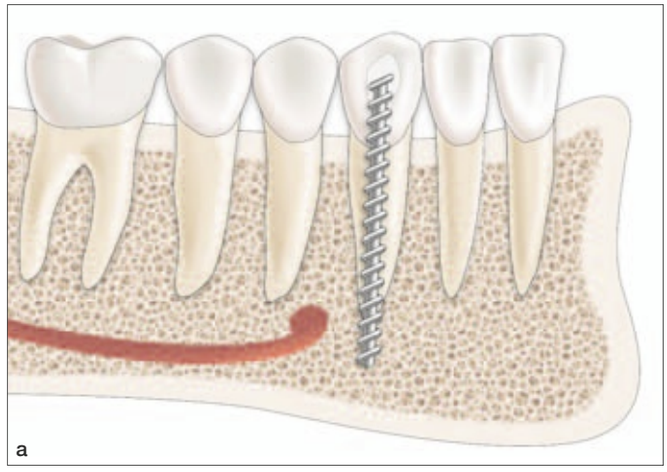


Fig 2 Vitallium alloy implant of the modern period. Surgery was performed in one stage and the implant was not submerged. Apart from the choice of material, this implant is very similar to the one-piece implants of today.



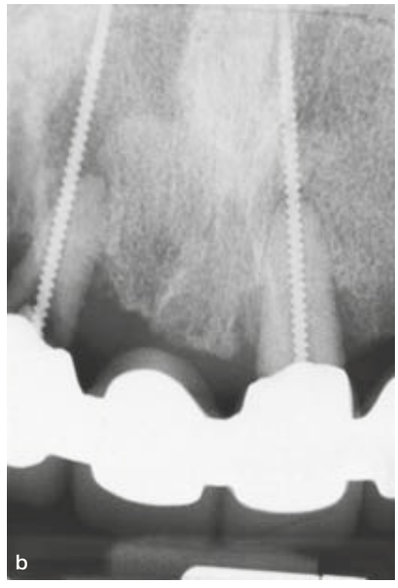


Fig 3 Endodontic implant of the modern period.

a Diagram of an endodontic implant in the mandible. The root of the tooth supports the implant. The implant protrudes beyond the root and seeks transapical bone integration. No bone surgery is involved.

b Radiograph of an endodontic implant in a partially edentulous anterior maxillary. Note the bone, crestal, and periapical lesions. A certain amount of bone height is required to anchor the endodontic screw. The treatment is doomed to fail within a more or less short period of time.

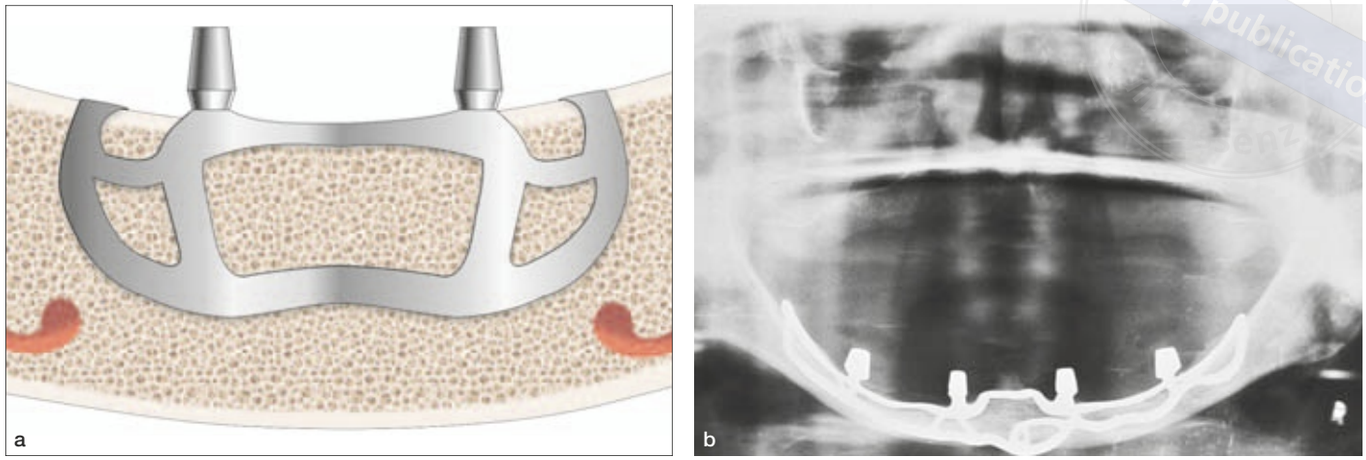
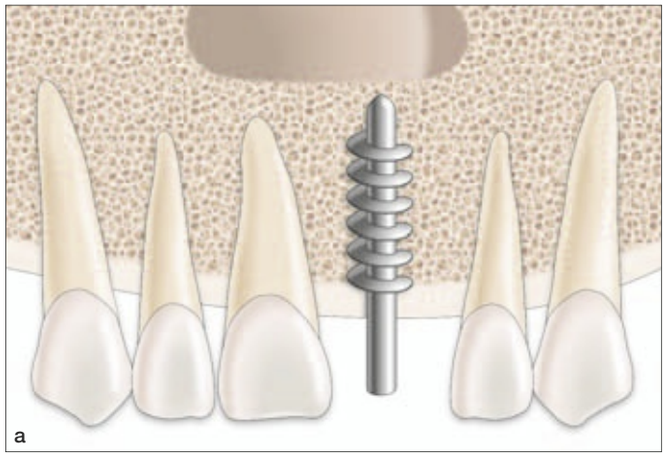


Fig 4 Subperiosteal implant.

a Diagram of a periosteal implant.

b Radiograph of a periosteal implant in the edentulous mandible. The implant is fitted as close as possible to the edentulous ridge. Over time, it causes significant bone resorption.



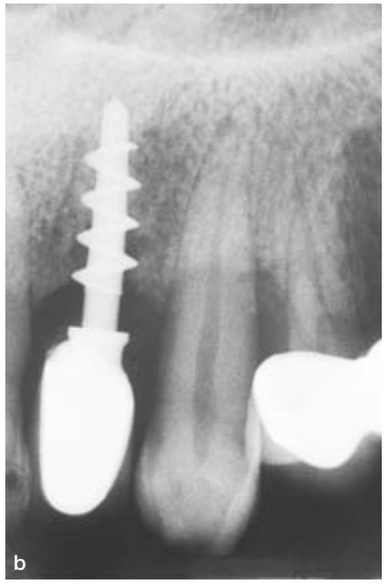
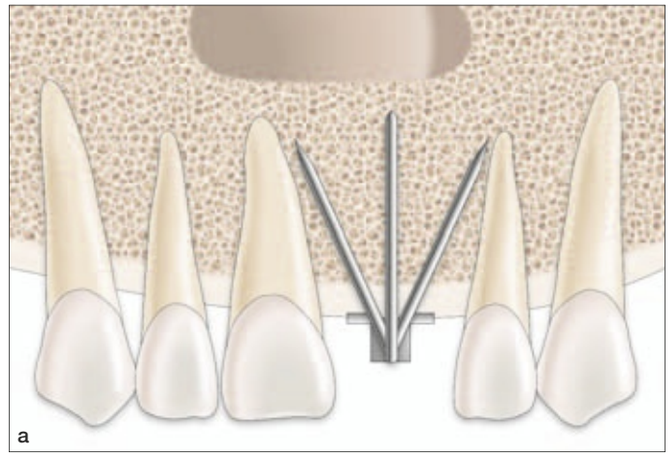


Fig 5 Cherchève implant.

a Diagram of a Cherchève implant.

b Radiograph of a Cherchève implant in the partially edentulous maxilla. The implant is osseointegrated. Crestal bone loss is the consequence of excessive mechanical stress.



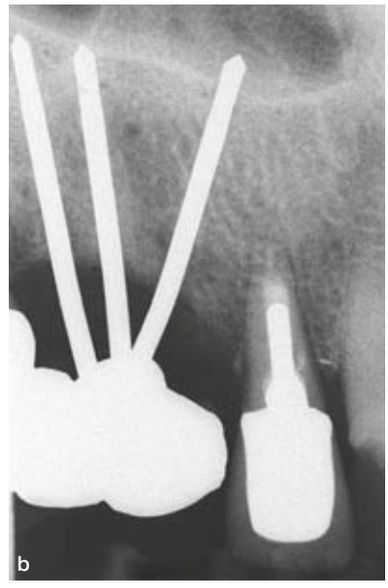


Fig 6 Scialom implant.

a Diagram of a Scialom implant.

b Radiograph of a Scialom implant in the partially edentulous maxilla. The tantalum needles are perfectly integrated in this case, without crestal bone loss.

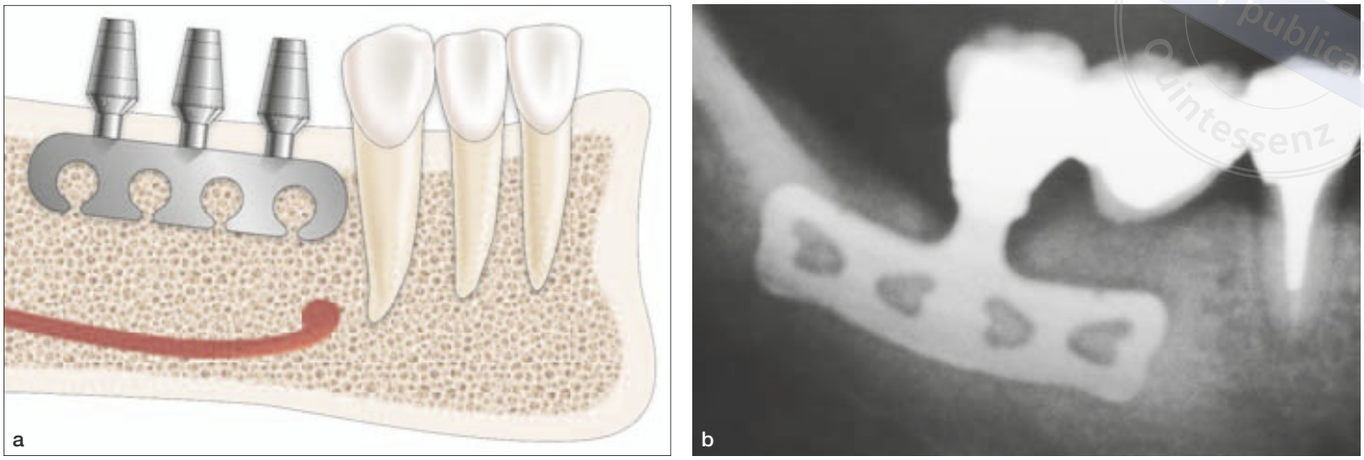


Fig 7 Linkow implant.

a Diagram of a Linkow implant. The blade has three pillars that, if necessary, can be oriented in the right direction using pliers.

b Radiograph of a Linkow implant in the partially edentulous mandible. Blade with a pillar.
Note the bone loss at the neck of the blade and at the upper mesial edge of the blade.

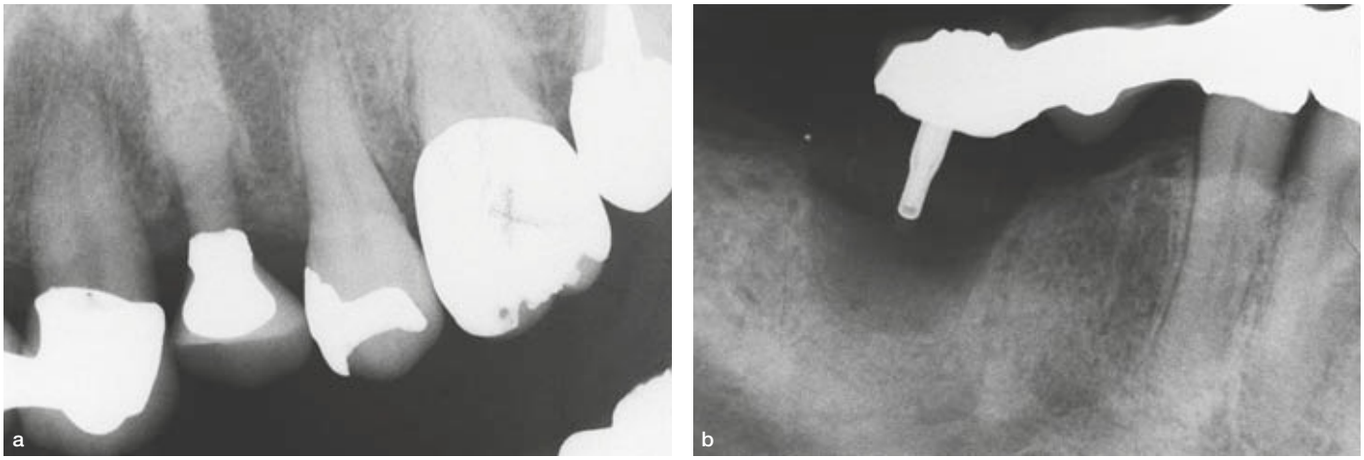


Fig 8 Nonmetallic implants.

a Radiograph of an alumina ceramic implant in a partially edentulous mandible.
The implant is osseointegrated. Its radiodensity is lower than that of metals; however, it is easily detectable.

b Radiograph of a vitreous carbon implant in a partially edentulous mandible.
The radiodensity of the implant is very low and its contours cannot be determined on X-ray. This is a permanent issue.

the tripod come together to support the restoration (Figs 6a, b). In 1967, Linkow introduced first the titanium and then the titanium alloy blade implant. It was used frequently until the late 1980s (Figs 7a, b). Implants made of sintered ceramic (Sandhaus, 1966) and vitreous carbon were produced in the early 1970s (Figs 8a, b).

Implantology in the 1950s to the 1970s was a time of trial and error. At that time, a fibrous peri-implant interface was sought. The goal was to mimic the periodontal ligament to absorb the shocks at the interface. Ankylosis, with its direct bone–implant contact, was considered a handicap in implant prognosis. In 1970, James introduced the transmandibular implant. Around 1975, Juillet elaborated the three-dimensional (3D) implant (Figs 9a, b), which requires lateral placement across the buccal aspect of the cortical ridge. All of these implants led to many short- and medium-term failures. Success rates of up to 50% in the short term were well accepted and deemed encouraging.

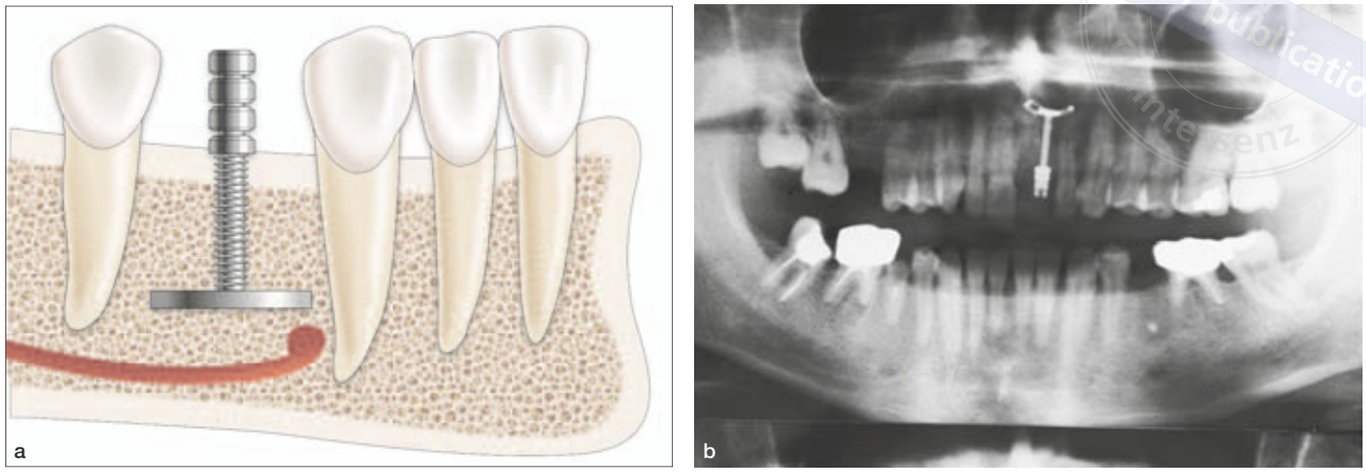


Fig 9 Juillet implants.

a Diagram of a Juillet 3D implant in a partially edentulous mandible. Placement is performed laterally, in the manner of disk implants.

b Radiograph of a Juillet implant in the anterior area of the maxilla. The implant is osseointegrated without any noticeable bone resorption.

Contemporary period

(Osseointegration or Brånemark period)

This period started in the late 1970s. The endosseous implant (fixture consistent with Brånemark) is the result of a philosophy that evolved over the years. The first Harvard conference in 1978 and the Swedish scientific studies published in 1969 and 1977 by Brånemark *et al.* marked the beginning of this period (Figs 10a, b).

Geographic location

North America, Europe.

Materials used

Titanium, titanium alloys, hydroxyapatite, ceramic.

Development of osseointegration

The first research on tissue integration of materials was carried out in Sweden in the early 1950s (Emneus, 1959). Knowledge about different types of materials and the influence of surgical trauma on tissue healing was generated. Experiments on various tissues, such as nerve, muscle, tendon, bone, skin, and mucous membrane were carried out on different animal models. The influence on tissue healing of specific factors, such as hormones, age, and temperature, were also evaluated.

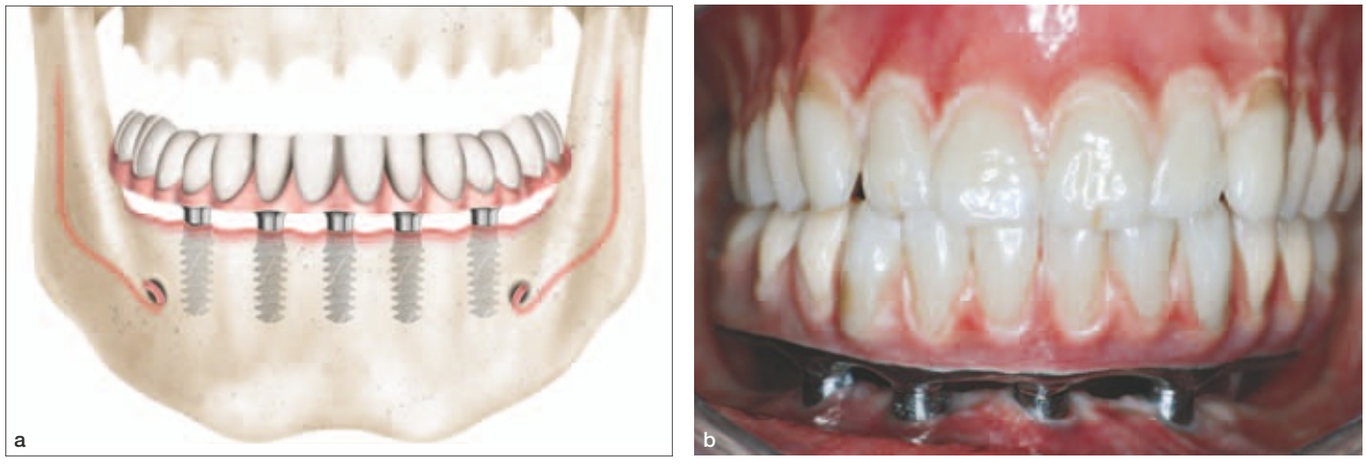
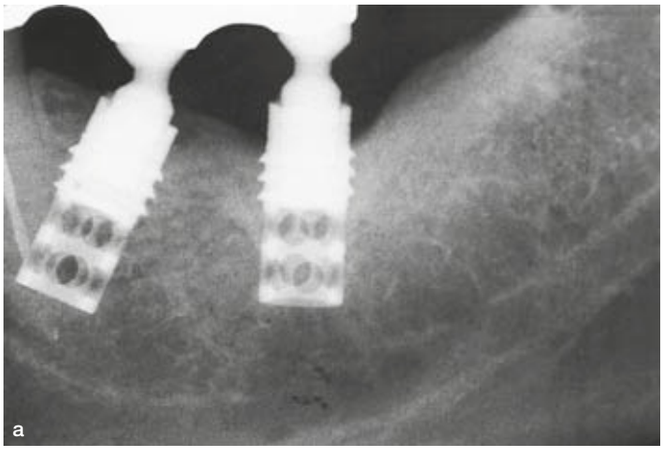


Fig 10 Brånemark implant.

a Diagram of the treatment of the edentulous mandible using the hybrid prothesis concept. The restoration is fixed on 5–6 implants placed in the anterior area of the mandible and is supported by the mucosa in the posterior area.

b Clinical condition of a mandibular restoration. Note the clearly visible abutments.

The first bone study was conducted in 1952 on a rabbit fibula (Brånemark *et al.*, 1985). The technique consisted of grinding the bone on the surface and observing under the microscope, in situ, bone repair and marrow reaction. Different traumatic models were then applied to these tissues to determine the factors influencing complete tissue regeneration. Factors identified as altering bone repair were: relative ischemia; local temperature elevation; and use of topical products.



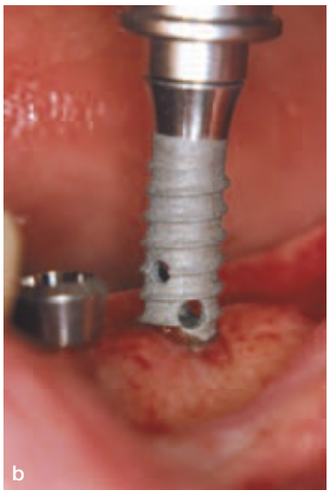


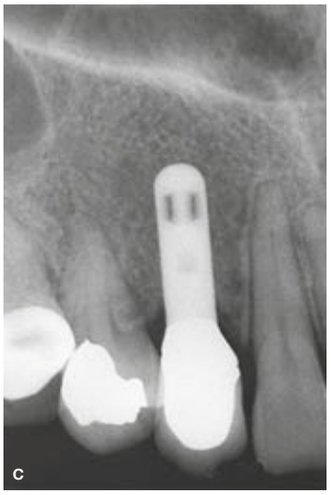
Fig 11 Shapes of implants used in the 1980s other than the 'Brånemark fixture'.

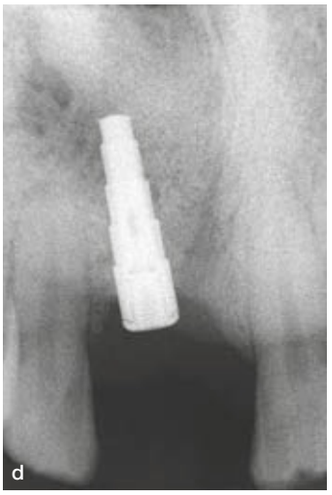
a Core-Vent-machined hollow screw implant with many apical vents.

b Bonefit hollow screw implant with titanium plasma spray coating with apical vents.

c IMZ impacted cylinder with titanium plasma spray coating with apical vents.

d Frialit impacted implant.





A first clinical protocol was developed in animals to test the restoration of an edentulous zone with a fixed prosthesis (Brånemark *et al.*, 1969). Partially edentulous dogs were rehabilitated with implant-supported restorations. Fixtures were previously submerged to achieve a bone healing period of 3–4 months. In 1965, the first patient was treated according to the principles of osseointegration (Brånemark *et al.*, 1977).

Brånemark *et al.* (1977) introduced the concept of osseointegration using titanium implants (Brånemark fixtures) and submersion during bone healing. Subsequently, other implant systems were tested with equal short-term success (Figs 11 and 12).

Silhouette of an implant

The silhouette of an implant refers to its general shape and it is the most easily perceived feature. Variation in silhouettes available on the market demonstrates the lack of a universal ideal shape. This reinforces the idea that each silhouette expresses a preference, a specific compromise, or a particular specification. It corresponds to an emphasis placed on a certain feature. For example, a conical shape increases primary stability whereas a more cylindrical shape simplifies placement. A more in-depth study of their properties makes it possible to define specific applications for each one.

Implants are subdivided into three parts: neck; body; and apex (Fig 13). Each part has its own specific features and a distinct role. This division into different parts simplifies the description of an implant and makes it part of a larger family.

The three parts (neck, body, and apex) are perfectly differentiated.

In 1976, Schroeder *et al.* proposed the use of a monobloc transgingival unsubmerged implant. This implant consisted of two parts: an intraosseous part with a rough surface and a transgingival part with a polished surface (Fig 12).

Fig 12 a Cylindrical bone-level implant with external connection.

b One-stage cylindrical 'tissue level' implant with internal connection.

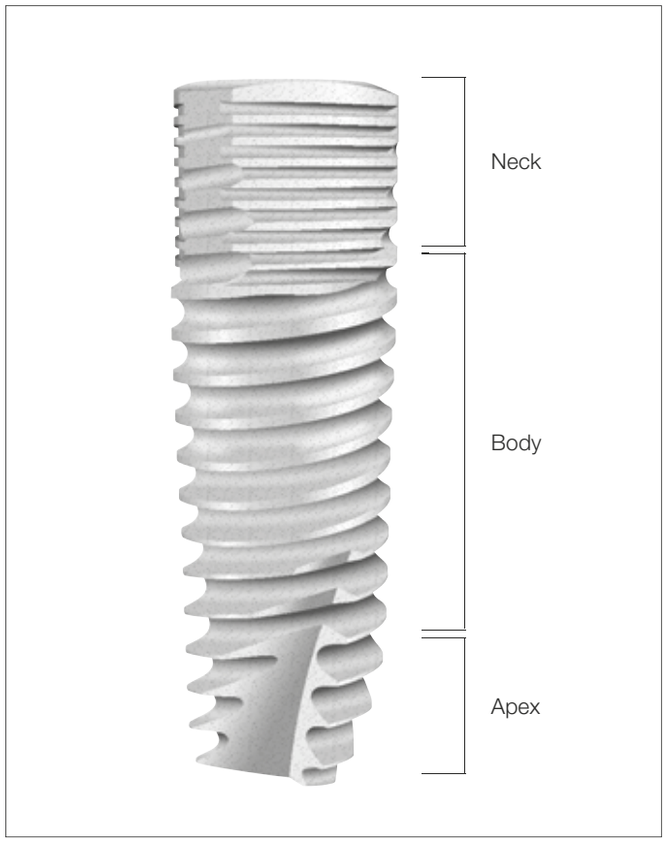


Fig 13 (V3 Implant)
Description of the constituent parts of an implant.

The junction or transition zone between the rough and polished surfaces is, according to the surgical protocol, positioned at the alveolar crest level.

Osseointegration was defined by Brånemark (Brånemark *et al*., 1985) as 'a direct anatomical and functional junction between the remodeled living bone and the loaded implant surface'. This type of interface allows high long-term success rates to be maintained. Thus, Brånemark and his team are recognized for developing the biologic principles of contemporary implantology, namely osseointegration. The most fundamental features compared to the previous period are:

- the search for direct apposition at the bone–implant interface, whereas previously fibrointegration was sought to mimic the alveolar-dental ligament;
- submerging the implant and its delayed loading in the face of the most immediate loading possible.

Since then, implantology has undergone unprecedented development. Millions of implants are placed every year on all continents. Many implant systems have been developed and success rates of 95–100% are commonplace in the literature.

Post-Brånemark period

(Immediate loading with osseointegrated implants)

A few clinical and animal studies published in the early 1990s showed that immediate loading can lead to a high rate of osseointegration. At the end of the 1990s, the number of these articles increased (Szmukler-Moncler *et al*., 1998, 2000). In 1999, Brånemark (Brånemark *et al*., 1999) gave an update and published a clinical article with a new implant system for immediate loading.

Geographic location

North America, Europe.

Materials used

Titanium, titanium alloys, hydroxyapatite.

Development of immediate loading

Immediate loading was common practice in the modern period before the Brånemark period. However, it was characterized by high failure rates. Brånemark implantology, a departure from the previous period, had many surgical and prosthetic prerequisites, nine in all (Szmukler-Moncler *et al*., 2000), including delayed loading. By complying with these prerequisites, many teams in several countries achieved high success rates (Esposito *et al*., 1998). There was then interest in simplifying the surgical and prosthetic techniques presented by the Swedish school as prerequisites.

Thus, we started using an armamentarium, such as pliers and kidney dishes, made of stainless steel instead of titanium. The occlusal surfaces of the implant-supported restorations are made of ceramic instead of resin. Control radiographs are taken immediately after implant placement instead of waiting until the end of the osseointegration period. One of the most significant simplifications is that there is no need to submerge the implants for 3–6 months. Finally, the deferred loading period in the patient with an edentulous mandible is eliminated by implementing protocols for immediate loading within 72 hours (Szmukler-Moncler *et al*., 2000). High success rates have been maintained despite prescriptions dating from the mid-1980s from the Swedish team.

Brånemark and his collaborators (1999) eventually questioned their principles, which had been issued some 30 years earlier. The number of studies on immediate loading, both clinical and experimental, escalated from the year 2000 onwards (Davarpanah *et al*., 2007). Indications initially limited to the edentulous mandible were extended to all clinical situations in healed or post-extraction sites (Fig 14). Implant success rates are now high (Del Fabbro *et al*., 2006) and similar to those achieved with conventional delayed loading methods.

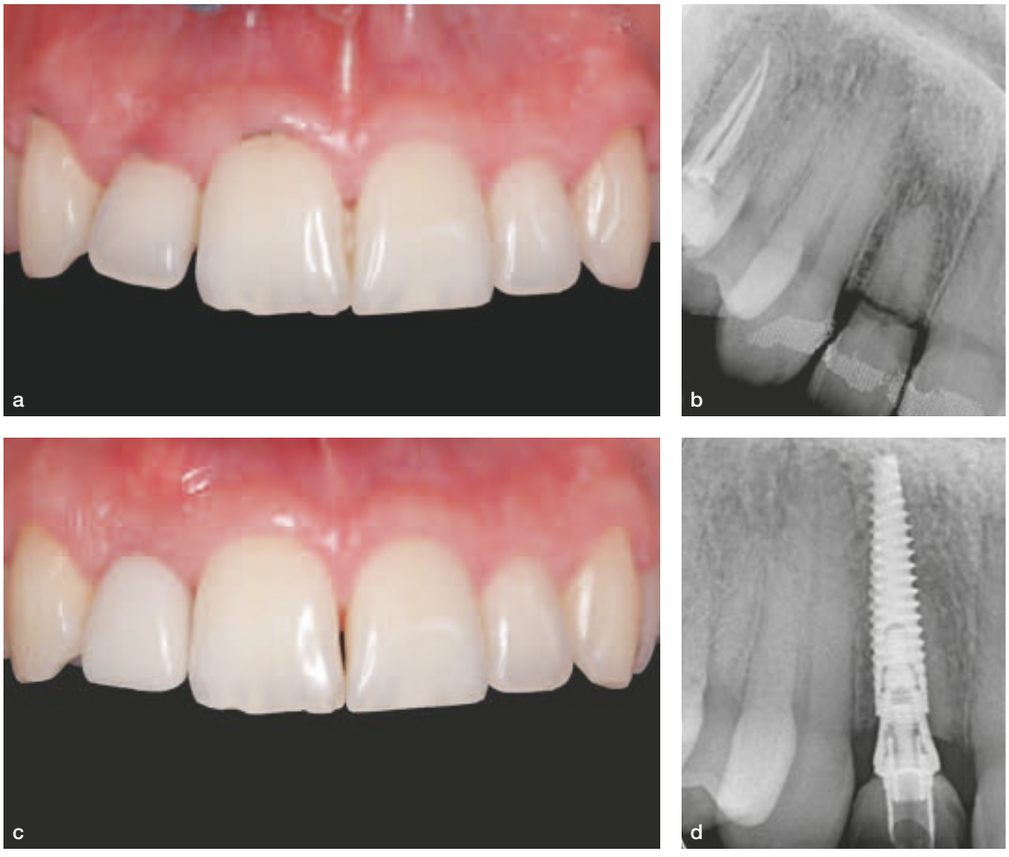


Fig 14

a, b Buccal view and radiograph of the fracture in 12.

c, d Final clinical and radiographic status.

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1

Maxillary and mandibular anatomy

C. Vachet, S. Szmukler-Moncler, M. Davarpanah

Objectives

At the end of this chapter the reader should be able to be familiar with:

- maxillary and mandibular bone anatomy
- maxillary and mandibular vascularization and innervation
- the evolution of the intermaxillary relationships induced by edentulism
- the evolution of anatomical obstacles induced by edentulism.

Introduction

The clinician must be familiar with maxillary and mandibular bone anatomy, whether the patient presents with or without teeth. They must also have knowledge of vascularization and innervation. Indeed, these areas are part of clinical practice and they should know which hard and soft tissue elements they are likely to encounter when making an incision, raising a flap, drilling into the bone or collecting a bone graft. A patient who comes for treatment should never leave with an additional disability.

The dynamics of resorption of the different bone parts after tooth loss must also be taken into account because new occlusion-related issues must be anticipated and treated appropriately.

The maxilla are described first, followed by the mandible.

Maxilla

Two maxillary bones form the skeleton of the upper jaw (Figs 1-1 and 1-2). The maxillary bone is articulated with all the other bones of the face and contains a vast cavity, the maxillary sinus. It borders the nasal fossae on the outside, the orbital cavities at the bottom and the oral cavity at the top; it supports the maxillary teeth.

Anatomical description

Notably, the jaw bone is hollowed out of a cavity, the maxillary sinus, which belongs to the group of nasal sinuses. It occupies the entire maxillary pyramid and sometimes has extensions, especially in the palatine process.

Its detailed anatomical description can be found in many specialized books (Gaudy, 2007) as well as in previous editions of this book (Davarpanah *et al.* 2008, 2012). Briefly, the jaw bone is a skeletal element shaped as a triangular pyramid with a truncated lateral apex. It has three faces and a truncated outer apex:

- the orbital upper surface, which is a part of the orbit floor.
- the posterior surface, which constitutes the maxillary tuberosity.
- the anterior jugular face, the internal base, which forms the side wall of the nasal cavities. It is hollowed out by the maxillary hiatus, which connects the maxillary sinus to the nasal cavity.

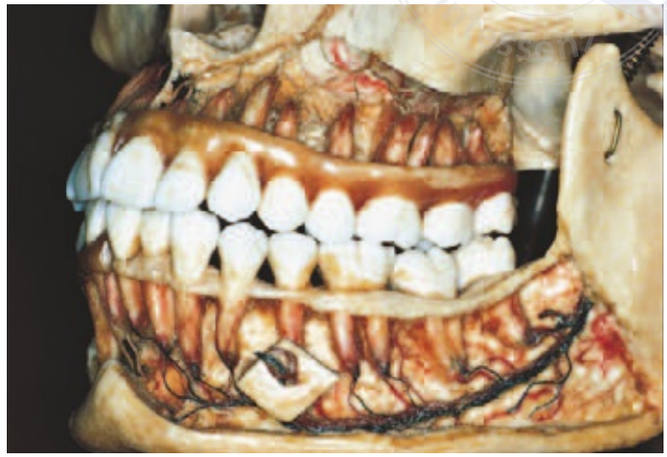


Fig 1-1 Left lateral view of the two maxillary bones. The relationship between the two maxillary bones and their vascularization and innervation are shown.

Vascularization

The vascularization of the maxilla depends on several branches of the maxillary artery (MA) (Figs 1-2a, b):

- The infraorbital artery (IA) ensures vascularization of the upper orbital surface and anterior surface of the maxilla and anterior teeth.

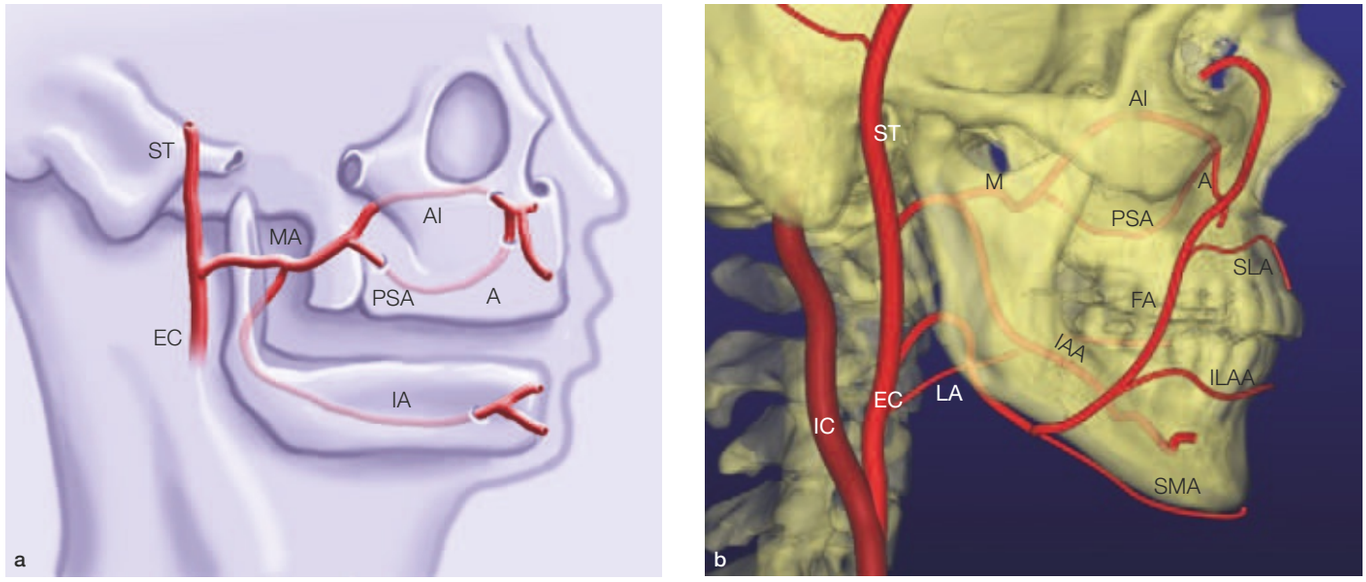


Fig 1-2 Vascularization of the maxilla.

a Side view of the face. The external carotid artery (ECA) is divided into two terminal branches, the superficial temporal artery and MA. The latter is the origin the IA; it passes through the orbital floor and exits through the infraorbital foramen. It anostomoses (A) with the PSAA.

b Three-dimensional (3D) reconstruction of the face with its vascularization. This three-dimensional view illustrates the same vessels shown in the previous diagram. In addition, the internal carotid artery, lingual artery (LA), facial artery (FA) and its successive branches, submental artery (SMA), inferior labial artery and superior labial artery are shown.

- The posterior superior alveolar artery (PSAA) ensures vascularization of the posterior surface of the maxilla and posterior teeth. The IA and PSAA are anastomosed by an artery (A) usually located between the sinus mucosa and anterolateral wall of the maxillary sinus; it is duplicated in 33–44% of cases (Solar *et al.* 1999; Rosano *et al.*, 2011; Traxler *et al.*, 1999) (Figs 1-2c, d). In 47–55% of cases (Mardinger *et al.* 2007; Rosano *et al.* 2011), it goes through the anterolateral wall and can then be considered as an anatomical obstacle when performing a sinus lift using a lateral approach.
- The greater palatine artery (GPA), a branch of the descending palatine artery, ensures vascularization of the posterior part of the palatal mucosa (Figs 1-2e-g).
- The nasopalatine artery (NPA), a branch of the sphenopalatine artery vascularizes the anterior part of the palatal mucosa. The GPA anastomoses with the NPA (Fig 1-2g).

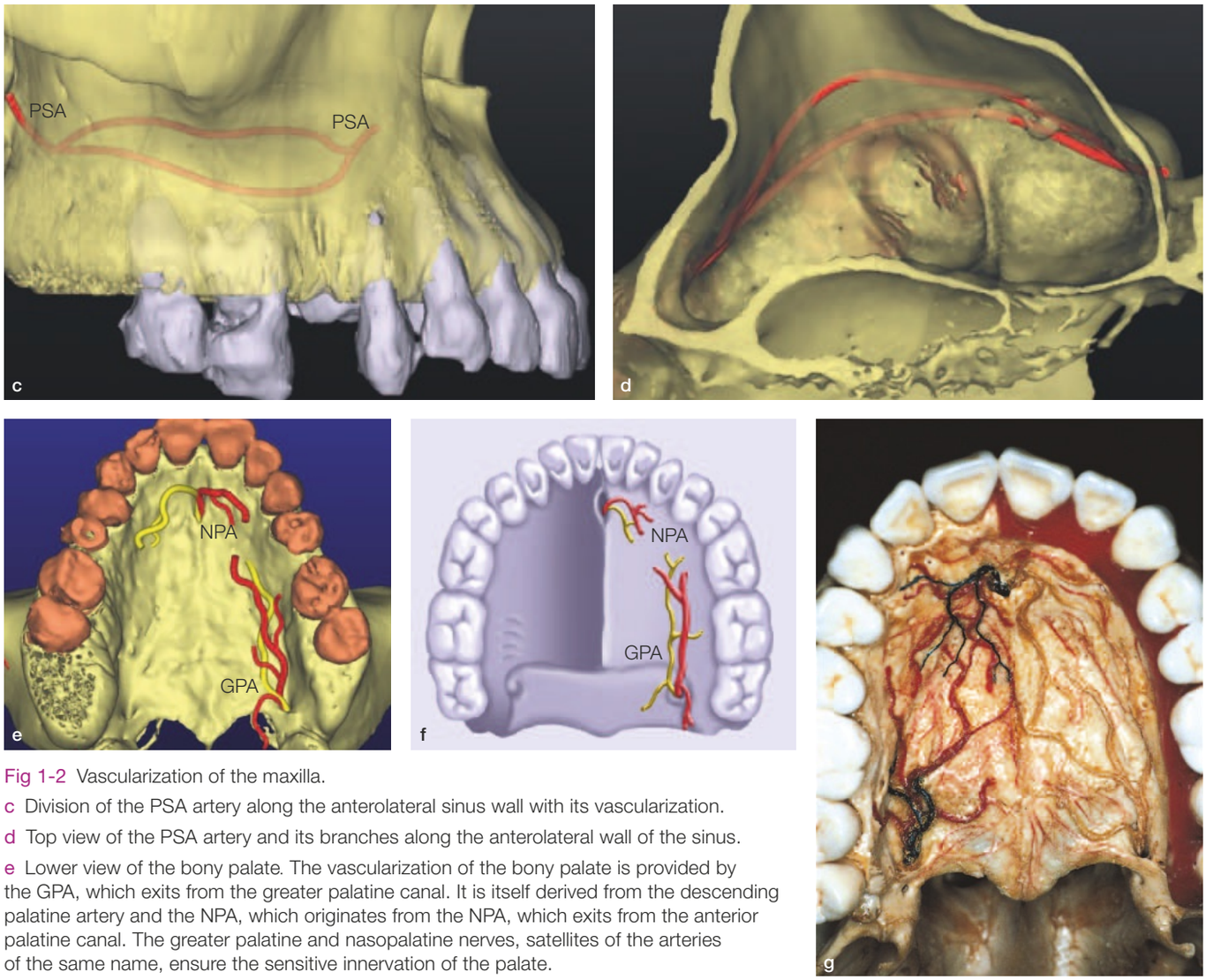


Fig 1-2 Vascularization of the maxilla.

c Division of the PSA artery along the anterolateral sinus wall with its vascularization.

d Top view of the PSA artery and its branches along the anterolateral wall of the sinus.

e Lower view of the bony palate. The vascularization of the bony palate is provided by the GPA, which exits from the greater palatine canal. It is itself derived from the descending palatine artery and the NPA, which originates from the NPA, which exits from the anterior palatine canal. The greater palatine and nasopalatine nerves, satellites of the arteries of the same name, ensure the sensitive innervation of the palate.

f 3D reconstruction of palate vascularization and innervation. The anterior and posterior palatine canals with their arteries and nerves were reconstructed in this 3D view of the palate.

g Anatomical cross section of the palate vascularization and innervation. Note the anterior and greater palatine canals and associated vessels. Note the anastomosis of the two networks.

Innervation

Innervation of the maxilla depends on the maxillary nerve, the second branch of the trigeminal nerve (V2), which is the sensory nerve of the face (Figs 1-3a, b). The maxillary nerve originates from the trigeminal ganglion (TG) of the trigeminal nerve. It passes through the round foramen at the base of the skull, hollowed out in the greater wing of the sphenoid. In the pterygopalatine fossa it gives:

- the zygomatic nerve (ZN), which anastomoses with the lacrimal nerve from the ophthalmic nerve V1 at the side wall of the orbit, to provide sensitivity to the skin of the cheekbone.
- the pterygopalatine (PP) nerve, which engages in the greater palatine canal to ensure sensitive innervation of the soft palate and posterior part of the hard palate (Figs 1-2e, f).
- the posterior superior alveolar nerves, which run down along the maxillary tuberosity and penetrate into canals in the bone to ensure sensitive innervation of the maxillary molars.

Then, it passes along the infraorbital canal and provides:

- the middle superior alveolar nerve, which runs down into the side wall of the maxillary sinus.
- the anterior superior alveolar (ASA) branches, which get separated from the nerve at the end of the infraorbital canal and run in the anterior wall of the maxillary sinus to ensure sensitive innervation of the maxillary anterior teeth.
- the terminal branch of the maxillary nerve, called the infraorbital nerve (ION), which crosses the infraorbital foramen and provides sensitive branches for the lower eyelid, cheek, nose and upper lip.

Any trauma to this nerve in the infraorbital foramen during subperiosteal detachment of the anterior surface of the maxilla results in hypoesthesia of the upper lip and homolateral nose wing.

Anatomy applied to the maxilla

The following case illustrates issues related to the knowledge of maxillary anatomy.

A patient presents for rehabilitation of her left posterior maxillary. The cone-beam computed tomography examination shows reduced subsinusal bone height (Fig 1-4a). Any implant rehabilitation should be preceded by a sinus bone graft using a lateral approach. This consists of penetrating into the sinus through the anterolateral wall, lifting the sinus membrane and grafting the gap thus delimited using a bone substitute. A knowledge of the anatomy requires us to examine the anterolateral bone wall of the maxillary sinus to locate the PSAA. The latter is often found in the bone wall or membrane. If the clinician does not pay attention to this, he might need to manage subsequent intraoperative bleeding.

In this case, the artery is not detectable on the cross section that is supposed to highlight it. However, knowing its anatomical presence allows us to carefully conduct surgery using diamond-surfaced piezo inserts (Blus *et al.*, 2008). The PSAA, caught in the sinus membrane (Fig 1-4b, c), can then be visualized. Our anatomical knowledge allowed us to suspect its presence without damaging it. A lesion would have exposed us to the risk of hemorrhage and increased the duration of the procedure.

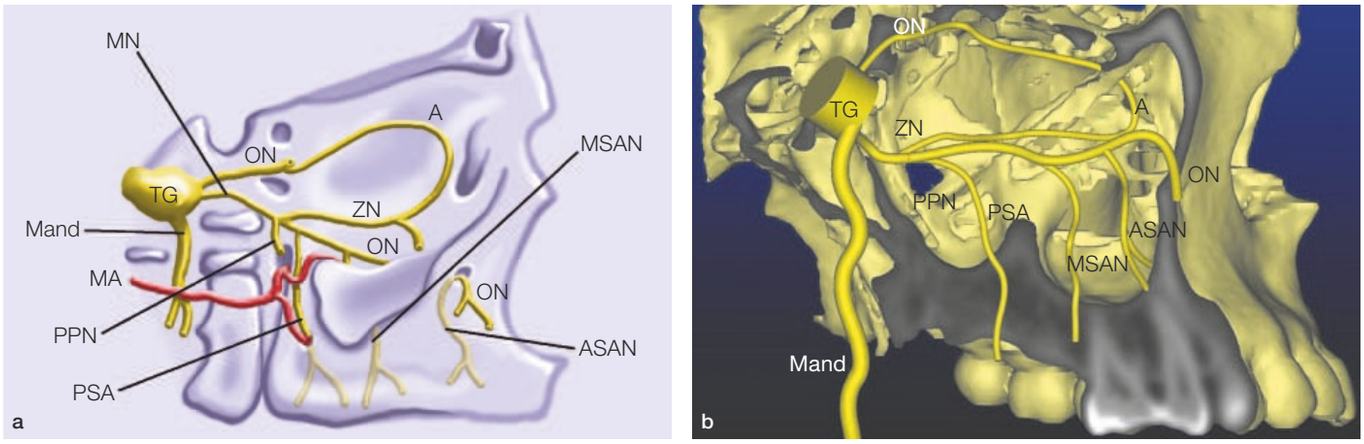
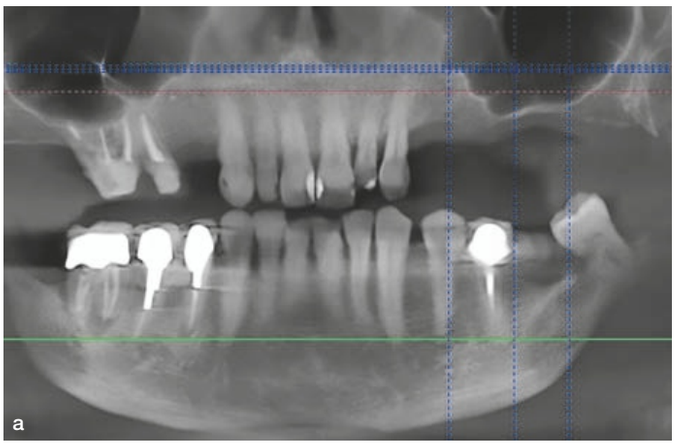
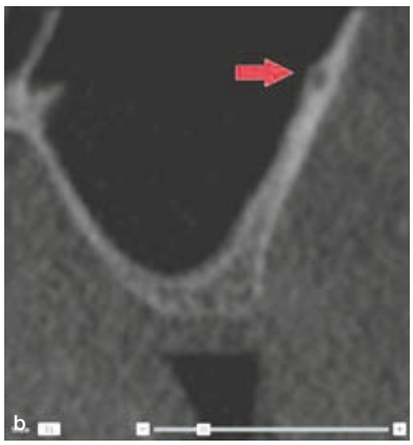


Fig 1-3 Innervation of the maxilla.

a Side view of the face. The TG gives rise to three nerves: ophthalmic (ON), maxillary (MN) and mandibular (Mand). The maxillary nerve gives rise to the ZN, which anastomoses (A) with the ON, the pterygopalatine nerve (PPN) and posterior branches of the ASAN. The terminal branch of the MN, the ION gives rise to the MSAN and ASAN.

b Side view of a 3D reconstruction. This 3D view from a computed tomography radiograph is based on the information given in the previous diagram.





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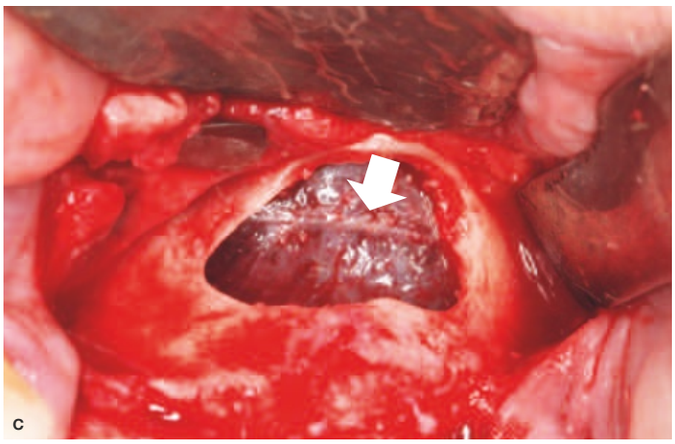


Fig 1-4 Anatomy applied to the maxilla.

a Panoramic section showing the low subsinusal bone height. A sinus bone graft is needed.

b Transverse section at the posterior sector. It confirms the reduced subsinusal bone height. The PSAA is not detectable on this section.

c Preparation of the lateral approach window with identification of the PSAA. The PSAA is caught in the membrane and was not detectable on the transverse section; however, its presence was suspected.

Mandible

The mandible is an odd and symmetrical bone. It is the only mobile bone of the face and constitutes the skeleton of the lower face (Fig 1-5a). It articulates with the temporal bones and supports the mandibular teeth.

Anatomical description

The mandible consists of a body and two rami.

The body is curved, horseshoe-shaped and open towards the rear (Figs 1-5a-c).

It has two sides:

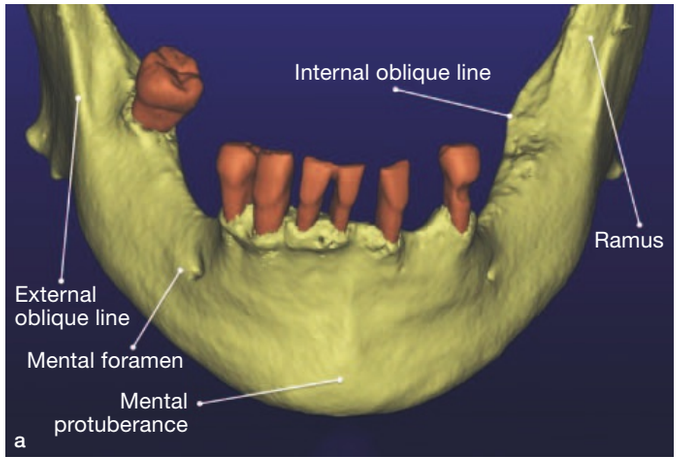
- An anterior, convex face, the mandibular symphysis; it is marked by an anterior and median relief. It has a lateral orifice, the mental foramen, from which the mental nerve originates. It is located at the mid-height of the mandible opposite the premolar teeth.
- A posterior, concave face, marked by the reliefs of the superior and inferior mandibular spines. The superior spines insert into the genioglossus muscles, while the lower spines insert into the geniohyoid muscles.

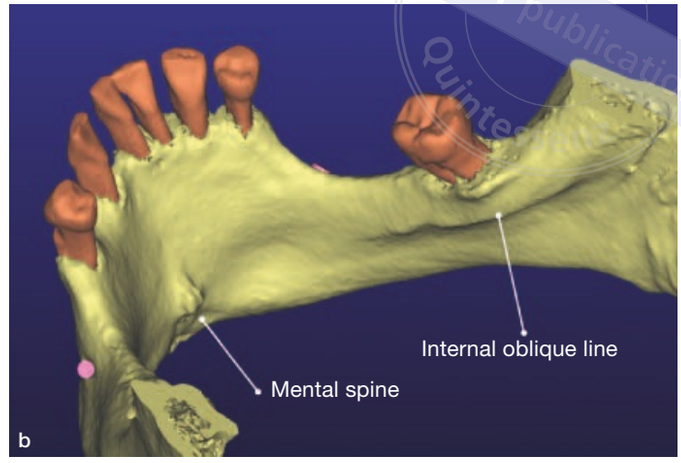
It also has two edges:

- An upper edge, hollowed out with alveoli that receive the mandibular teeth.
- A corticalized and very thick lower edge.

The rami are detached on each side of the posterior end of the body. On their inner side is the entrance to the mandibular canal, inside which the inferior alveolar vascular-nervous pedicle passes; this pedicle provides the lower alveolar nerves and vessels. Anterior to this entrance is the lingula of the mandible (or Spix spine), which serves as a reference point during a mandibular nerve (MN) block. From the lingula the mylohyoid groove starts, which marks the insertion of the muscle of the same name. This muscle located at the floor of the mouth divides cellulitis into dental cellulitis, which drains above it, and submandibular cellulitis, which drains below it.

The external face of the rami inserts into the masseter muscle. Their upper edge is marked by two bony protrusions. At the front is the coronoid process into which the temporal muscle inserts. At the back is the condylar process, which articulates with the temporal bone and inserts into the lateral pterygoid muscle. They are separated by the mandibular notch.





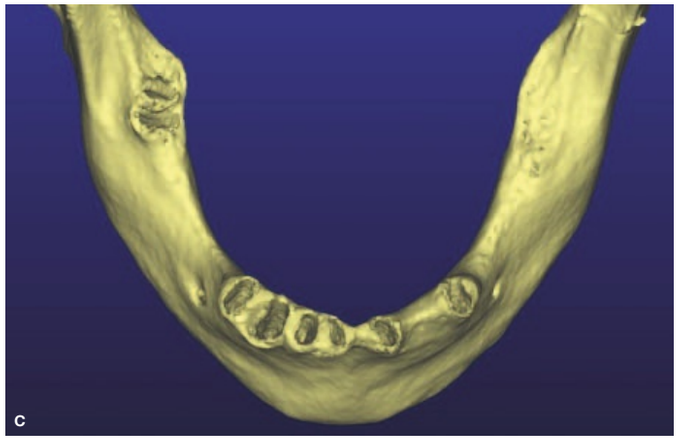


Fig 1-5 Anatomy of the mandible.

a Anterior view of a 3D reconstruction of the mandible with its different landmarks. This bone consists of a horizontal body and two vertical rami on each side.

b Lateral view of the mandible with its internal landmarks.

c Occlusal view of the mandible after digital extraction of residual teeth from their sockets.

VIDÉO

Vascularization

The mandible is vascularized (Fig 1-6) by an external periosteal network and an internal endosseous network.

The external vascular network is formed by:

- the FA, which reaches the submandibular region (Figs 1-6a, b) by passing over the medial surface, then reaching the upper edge of the submandibular gland (SMG). It is positioned along the periosteum of the mandible at its lower edge and on the lower section of the lateral surface, after which it is more superficial and passes into the cheek.
- the SMA (Figs 1-6a, b), which originates from the facial artery, runs forward obliquely and reaches the lower border of the mandible in the anterior zone. It can be damaged if the lower cortical bone of the mandible is perforated during an implant procedure, resulting in a cervical hematoma.
- the sublingual artery, which originates from the LA and passes along the anterior part of the floor of the mouth until it reaches the posterior cortical part of the symphysis. This artery can be damaged if this cortical process is perforated.
- the masseteric and pterygoid arteries, branches of the maxillary and mylohyoid arteries, and a branch of the inferior alveolar artery (IAA) (Figs 1-6c, d).

The internal vascular network is dependent on the IAA, which is a branch of the MA. It enters the mandibular canal behind the lingula.

The artery is located above the inferior alveolar nerve (IAN). If the roof of the mandibular foramen is injured, bleeding generally precedes nerve injury.

From the IAA, branches intended for each dental apex can be visualized. The artery divides into two terminal branches, an incisor artery that continues its course in the mandible and provides branches for the canine and lower incisors, and a mental artery that exits through the mental foramen and anastomoses with the SMA.

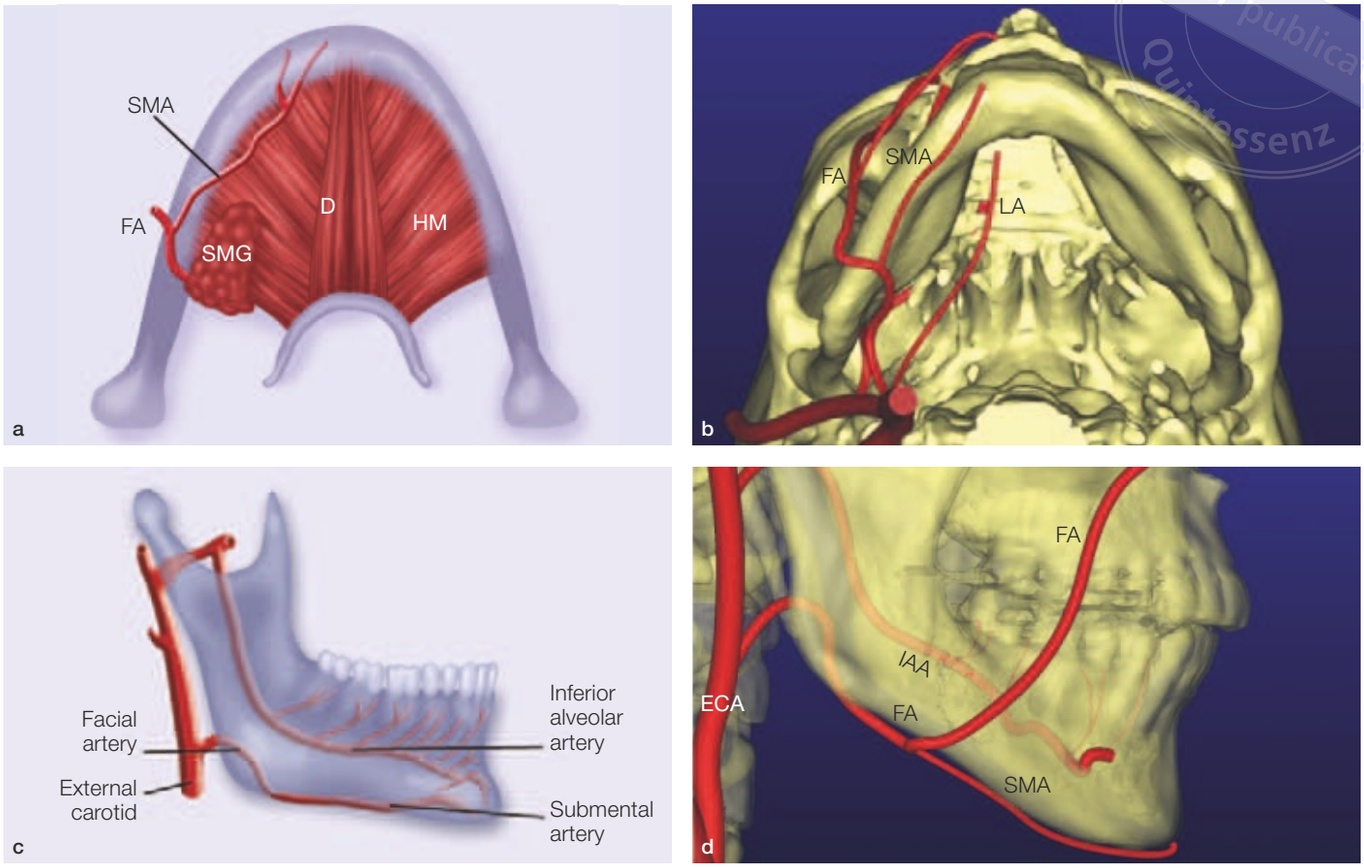


Fig 1-6 Vascularization of the mandible.

a Underside of the floor of the mouth. The floor is essentially made up of the two joining mylohyoid muscles reinforced underneath by the anterior belly of the digastric muscle. In the region of SMG, the FA gives rise to the SMA whose terminal branches lie below the lower edge of the symphysis.

b 3D view of the underside of the floor of the mouth.

c, d 3D view of the external side of the mandible with its different vessels.
From the posterior region, the FA starts from the ECA and leads forward to the SMA, whose terminal branches are located below the lower edge of the lower mandibular symphysis. Note the IAA and the branches it sends to the teeth in the mandible.

Innervation

The IAN (Fig 1-7a) provides sensitive innervation of the mandible and mandibular teeth. It is a terminal branch of the MN (V3). The MN, after passing through the base of the skull via the foramen ovale, penetrates the infratemporal fossa and, in particular, provides three motor nerves for the temporal muscle from back to front:

- the posterior deep temporal nerve (DTN), which originates from the temporomasseteric nerve
- the medial DTN
- the anterior DTN, which, together with the buccal nerve, originates from the temporobuccal nerve

It also has two terminal branches, the lingual nerve (LN), which runs against the inner side of the mandible in the molar region and the IAN, which penetrates the mandibular canal (Figs 1-7b, c).

This canal is usually located under the dental roots, in contact with the internal cortical bone. However, when the canal is located high up, its position is lateral with regard to the dental roots (Gaudy, 2007).

Opposite the premolars, the nerve divides into an incisive nerve that continues its intraosseous path in the mandibular incisive canal to ensure sensitive innervation of the canines and lower incisors and a mental nerve that crosses the mental foramen, which is often described as a bend in the bone. This mental nerve divides into multiple sensory branches for the lower lip and chin.

The LN (Figs 1-7b, c) descends between the lateral and medial pterygoid muscle and lies against the medial side of the retromolar trigone. It then comes into close contact with the submandibular duct and reaches the mobile tongue, whose sensitivity it ensures.

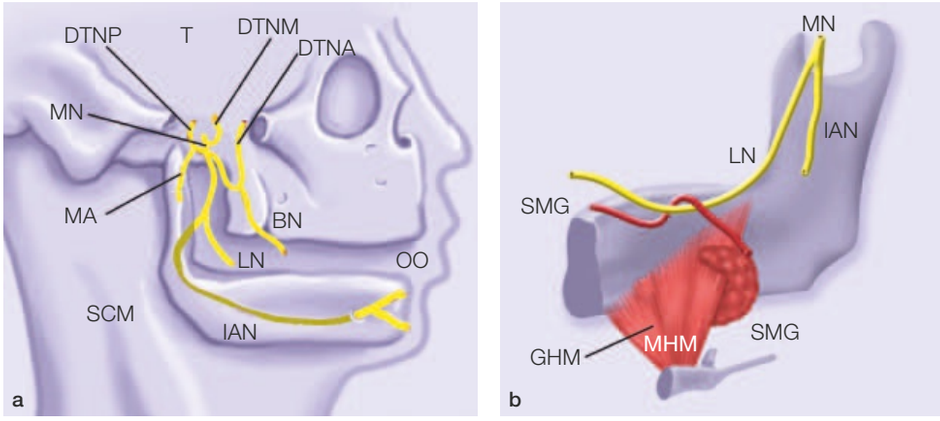
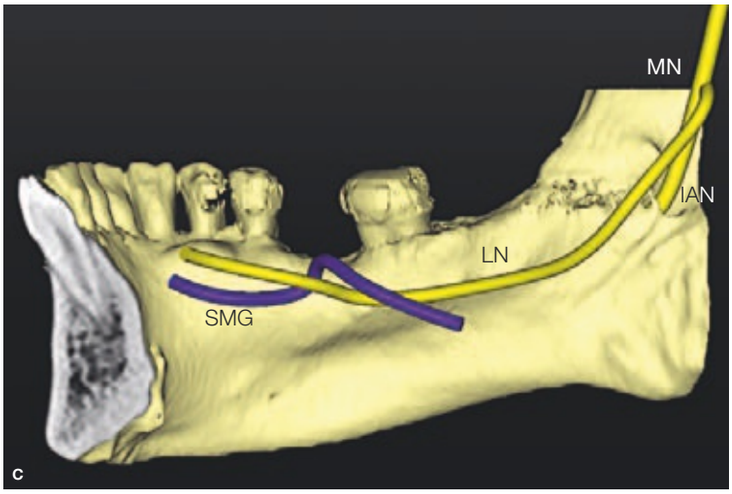


Fig 1-7 Innervation of the mandible.

a External side after sectioning of the zygomatic arch and resection of part of the mandibular ramus. The sternocleidomastoid muscle (SCM) marks a relief on the lateral side of the neck. The MN gives origin to the temporal nerve, which in turn gives origin to the anterior DTN, and the buccal nerve that innervates the buccinator muscle, the middle DTN and temporomasseteric nerve, which gives rise to the posterior DTN, and the masseteric nerve, which innervates the masseter muscle. The three DTNs provide motor innervation of the temporal muscle.



The maxillary nerve passes within the two bundles of the lateral pterygoid muscle and gives rise to its two terminal branches: on the one hand, the LN, which comes into contact with the inner side of the mandible; on the other hand, the IAN, which penetrates the mandible. The lower lip (represented here by the orbicularis oris muscle, whose motor innervation depends on the facial nerve) has a sensitive innervation that is dependent on the mental nerve, itself the terminal branch of the IAN.

b Medial view of the floor of the mouth. The LN originates from the bifurcation of the MN into the IAN, which penetrates the mandible, and the LN, which has a very close relationship with the submandibular canal, originating from the SMG. The floor of the mouth is viewed from its upper side with the mylohyoid and geniohyoid muscles. The sublingual gland is located above the floor of the muscles.

c 3D view of the inner wall of the mandible with the nerves and submandibular canal of the previous diagram.

Anatomy as applied to the mandible

The following case illustrates the issues related to the knowledge of the anatomy of the mandible.

A patient presents to the clinic to rehabilitate her right posterior mandible. Radiological examination reveals reduced bone height above the mandibular canal (Fig 1-8a). Short implants are recommended, especially since a safe distance of 2 mm must be maintained. Three C1 implants (MIS implants) with Ø 3.75 × 8 mm are placed; when a molar is extracted after an acute infection, an additional implant with Ø 5 × 8 mm is also placed (Fig 1-8b). The short implants make it possible to rehabilitate the patient without the need for a vertical bone graft.

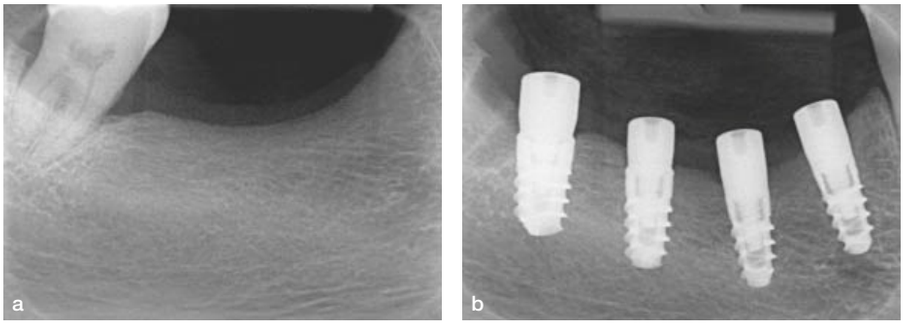


Fig 1-8 Anatomy as applied to the mandible.

a Periapical radiograph revealing a reduced height above the mandibular canal.

b Short implants placed in the posterior region while maintaining a safe distance of 2 mm between the implant apex and upper part of the mandibular canal.

Microscopic anatomy and bone typology

The bone response to the contact with the implant surface is different in cortical bone than in cancellous bone. Classification of the bone densities encountered during implant placement is that proposed by Lekholm and Zarb (1985), taking into account the distribution between cortical and cancellous bone (Fig 1-9a):

- type I bone: the jaw consists almost entirely of homogeneous compact bone (Fig 1-9b);
- type II bone: a thick layer of compact bone surrounds a core of dense trabecular bone (Fig 1-9c);
- type III bone: a thin layer of cortical bone surrounds a core of dense trabecular bone (Fig 1-9d);
- type IV bone: a thin layer of cortical bone surrounds a core of low-density trabecular bone (Fig 1-9e).

This is histologic classification is easily revealed by examining a histologic section under a microscope. Clinically, the relevance of this classification is limited, and the classification by Trisi & Rao is more suitable and preferred (Trisi & Rao, 1999). In this classification bone is:

- dense: the clinician does not feel any noticeable delineation between cortical and cancellous bone;
- normally, the clinician can clearly feel the transition from cortical to less resistant bone;
- low-density: the cortical and cancellous parts of the bone offer little resistance; they are easily passed.

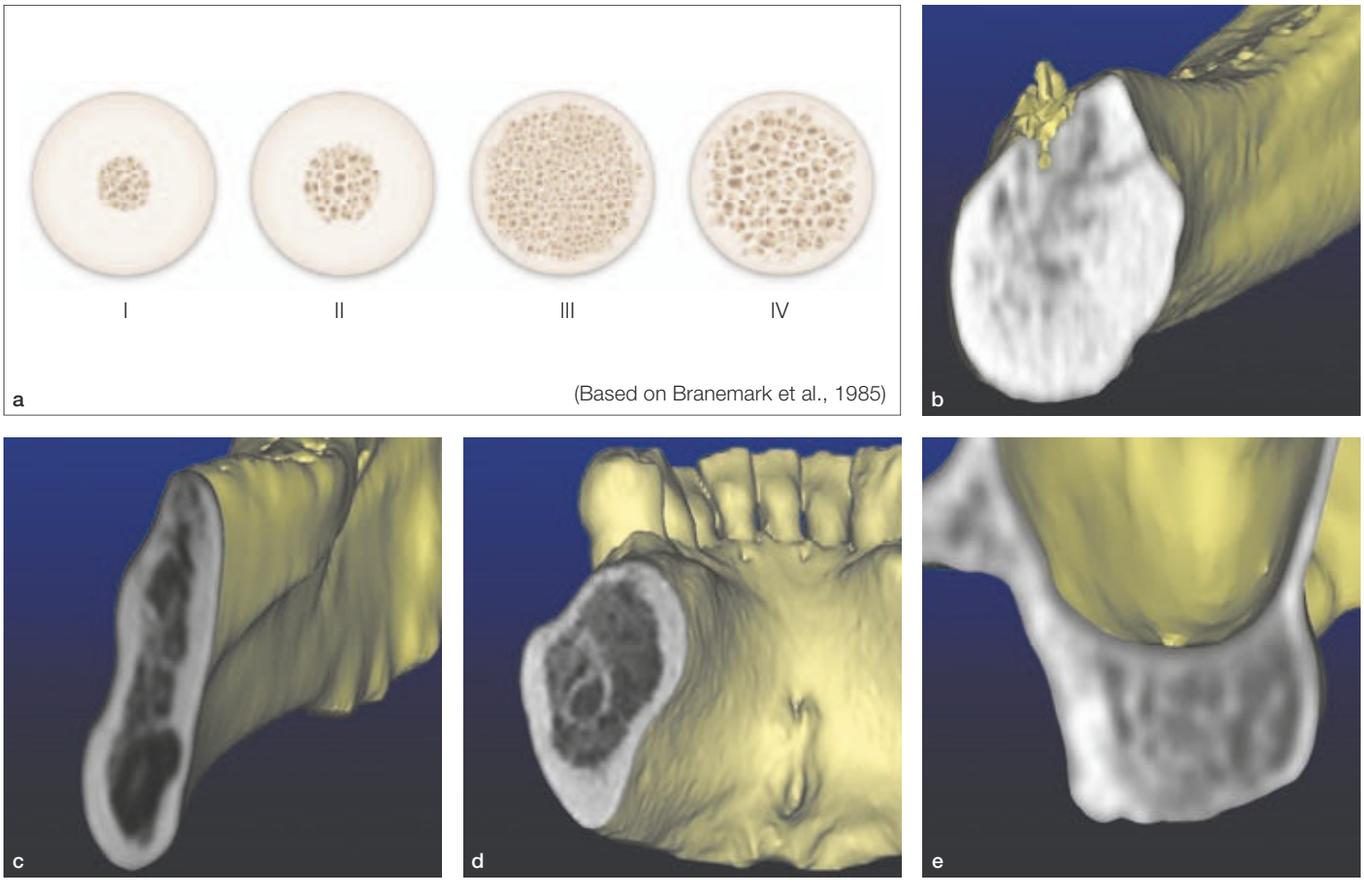


Fig 1-9 Bone typology based on a histologic approach according to Lekholm & Zarb.

a Diagram of the four types of classification.
Types I and II are more common in the mandible; types III and IV are more common in the maxilla.

b Transverse section of a 3D reconstruction showing a type I bone.

c Transverse section of a 3D reconstruction showing a type II bone.

d Transverse section of a 3D reconstruction showing a type III bone.

e Transverse section of a 3D reconstruction showing a type IV bone.

Anatomical variations and implantology

Maxillary and mandibular resorption

The size and shape of the bone ridges after tooth loss is of particular interest in the field of implantology since the new anatomy will help inform the positioning of implants. In fact, alveolar bone remodeling always occurs after dental extraction. It combines osteoclastic resorption of the alveolar bone with bone apposition in the extraction socket. Resorption is very active during the first few months of bone healing. Resorption occurs in the alveolar portion of the bone (Fig 1-10) and to a lesser extent in basal bone. Resorption of basal bone is more age-related.

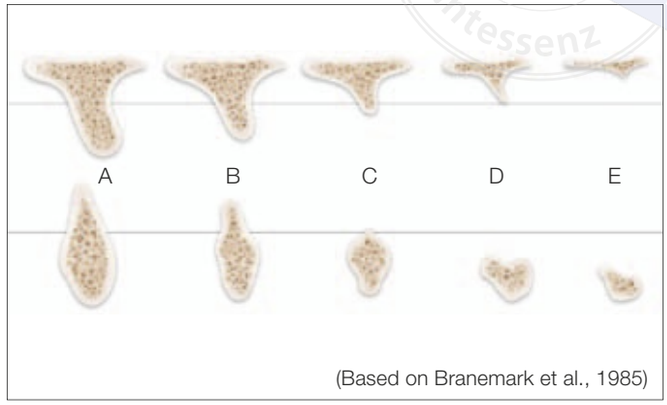


Fig 1-10 Maxillary and mandibular resorption typology. Each class from A to E corresponds to a more advanced degree of resorption. The classes concern the maxilla and the mandible.

Anatomical changes induced by edentulism

Certain changes in the bony bases must be taken into consideration because they may affect the surgical technique employed or the choice of implant length.

Generally speaking:

- In the mandible, resorption in the anterior ridge is four times faster than in the maxilla. In addition, resorption is faster on the lingual side (centrifugal resorption). Thus, the alveolar crest progressively loses its height and mesiodistal width. Vertical resorption brings the mandibular canal closer to the crestal margin (Figs 1-11 and 1-12).
- In the maxilla, vertical resorption is associated with greater resorption on the buccal side (centripetal resorption). These skeletal changes transform the maxillo–mandibular relationship (Fig 1-12). Vertical maxillary resorption often limits the available bone volume below the maxillary sinuses (Fig 1-11c).

In the maxilla, the residual bone under the sinus decreases. In the mandible, the distance between the ridge and IAN is also decreasing.

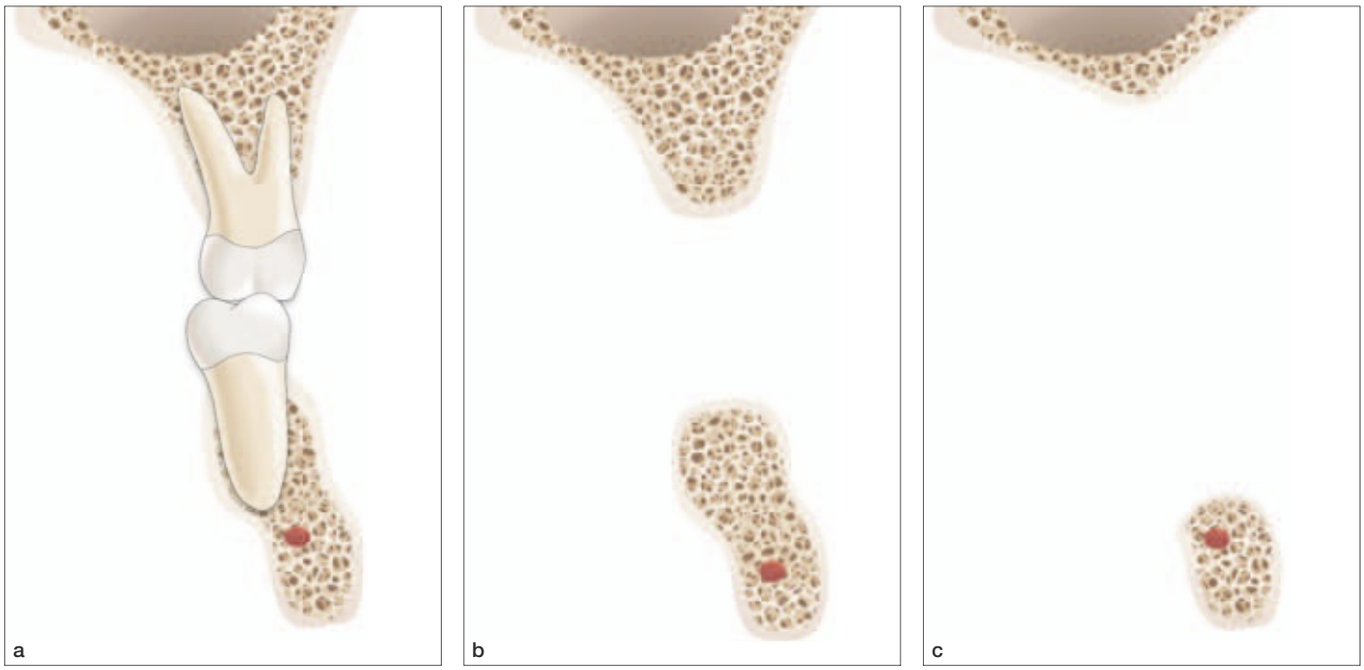


Fig 1-11 Evolution of the inter-maxillary relationships after tooth extraction.
a Situation when teeth are present in the posterior region.
b Situation shortly after tooth extraction.
c Situation long after tooth extraction has taken place.

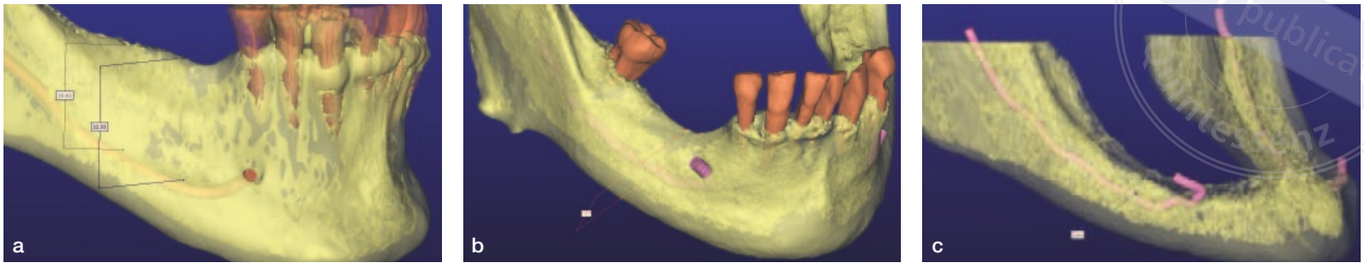


Fig 1-12 Bone resorption in the posterior area of the mandible.

a Edentulous area with low bone resorption. Implant placement in this area is not a major issue.

b Edentulous area with significant resorption. With 9 mm above the ridge, only short 7-mm implants can be used to rehabilitate this area.

c Atrophied posterior area. A height of less than 4 mm precludes a standard implant procedure in this area.

Depending on the area, tooth loss changes the anatomical relationships (Vacher, 2004):

- In the posterior mandibular area, the mandibular canal is usually located below the dental apexes (Fig 1-11a); it gets closer to the ridge since mandibular resorption is important (Figs 1-11b, c and 1-12a-c). In the presence of extensive resorption (Fig 1-12c), implant placement behind the mental foramina is contraindicated.
- In the anterior mandible, the floor of the mouth and mylohyoid line approach the alveolar ridge (Fig 1-13); bone quality is increasingly cortical at the expense of cancellous bone.
- In the mandible and generally, muscle attachments are more superficial due to bone resorption and limit the available space left for restorations. In the buccal zones, the attachments of the buccinator muscle are involved. Because of bone resorption, they may be located close to the crestal margin. On the lingual sides of the mandible, it is the mylohyoid and geniohyoid muscles that affect the available space. The floor of the mouth is closer to the mandibular crest. Laterally, this situation leads to a more superficial position of the LN; behind the symphysis, the genioglossus muscles are also more superficial.
- In the posterior maxillary area, for the maxillary sinus, ridge resorption after tooth loss is associated with pneumatization (Fig 1-14). This limits the subsinusal bone volume available for implant placement (Figs 1-14d, e). Sometimes the combined action of sinus resorption and pneumatization is such that only a thin slice of bone remains under the maxillary sinuses.
- The tuberosities and pterygomaxillary regions undergo less bone resorption than other parts of the maxilla: placement of implants in this area is sometimes recommended when there is insufficient bone volume in the maxillary molar area. This is a challenging procedure due to the many anatomical obstacles in the region (the descending palatine artery in particular).
- In the anterior regions of the maxilla, resorption may be vertical and bring the alveolar ridge back towards the nasal spine (Figs 1-15a, b), which may limit the length of the implants to be placed. It can also be horizontal and prevent the placement of implants without surgery (Figs 1-15c-e).

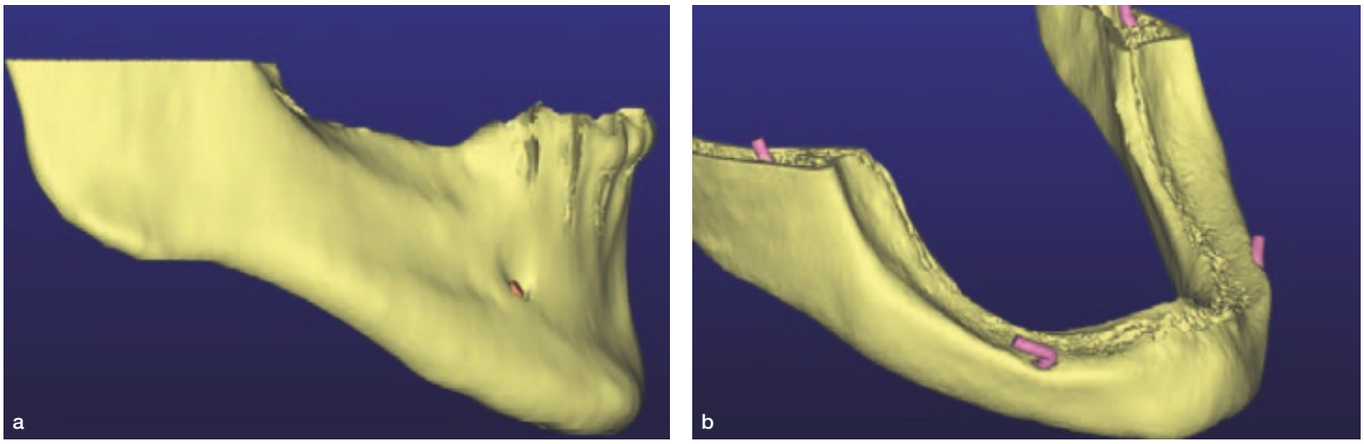
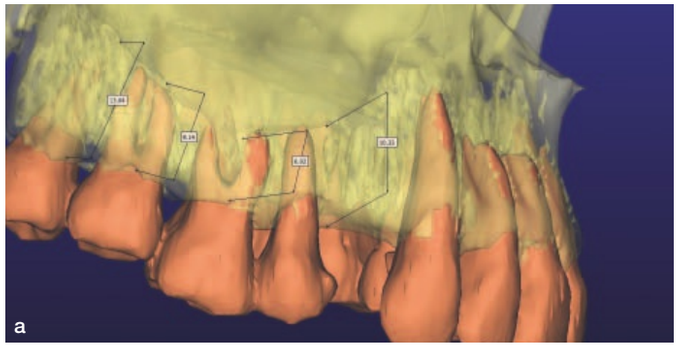
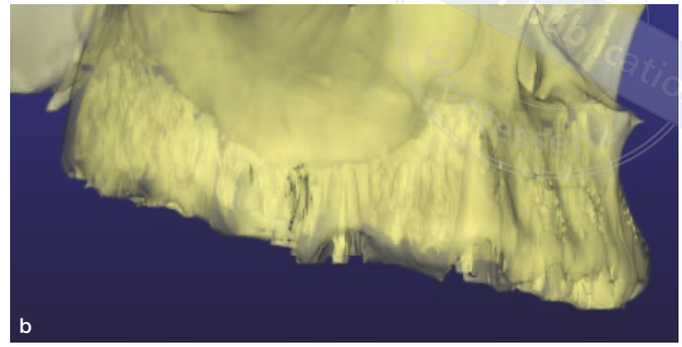


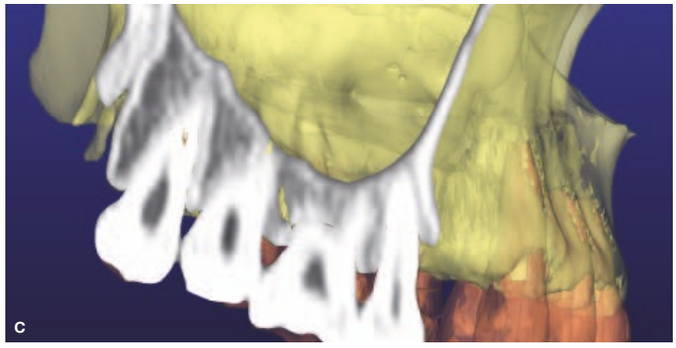
Fig 1-13 Bone resorption in the anterior area of the mandible.

a Edentulous area with very little bone resorption. Implant placement in this area is not a major issue.

b Atrophied anterior zone. The entire mandible is atrophied, the bone is corticalized and the mandibular canal is flush below the ridge.







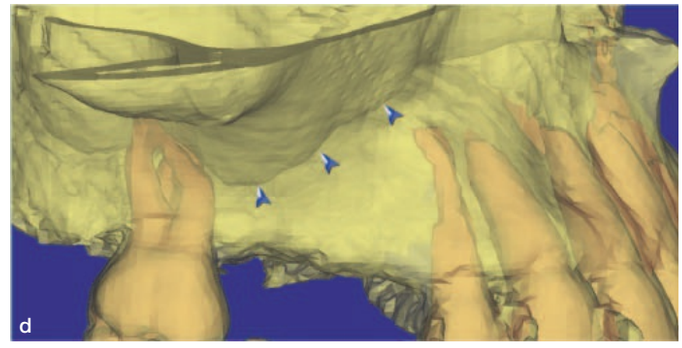


Fig 1-14 Bone resorption in the posterior area of the maxilla.

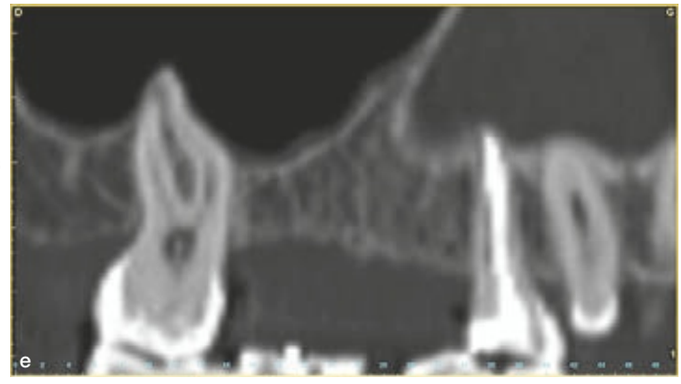
a Toothed area showing in transparency the lower delimitation of the maxillary sinus. The single-tooth gap in the premolar region does not concern the pneumatized area.

b Image identical to the previous one with all teeth digitally extracted. The purpose of this figure is to illustrate the relationship between sinus and the teeth roots in the area.

c Section highlighting the limits of the floor and anterior wall of the sinus.

d View of the anterolateral sinus wall with the limits of the sinus floor shown. Coronal section showing that tooth extraction below the sinus floor may cause the sinus floor to collapse.
The blue markings show the floor delineation.

VIDÉO



e Panoramic section corresponding to the previous figure. Disappearance of the teeth led to the collapse of the floor.
The remaining tooth retains a local supporting action on the floor.

Conclusion

Skeleton anatomy differs according to the degree of horizontal or vertical bone resorption.

In implantology, the clinician must have an excellent knowledge of:

- bone structure and shape of the maxilla and mandible;
- the location of vessels and nerves that constitute anatomical obstacles;
- anatomical variations related to tooth loss and age, especially the position of the maxillary sinuses and mandibular canal.

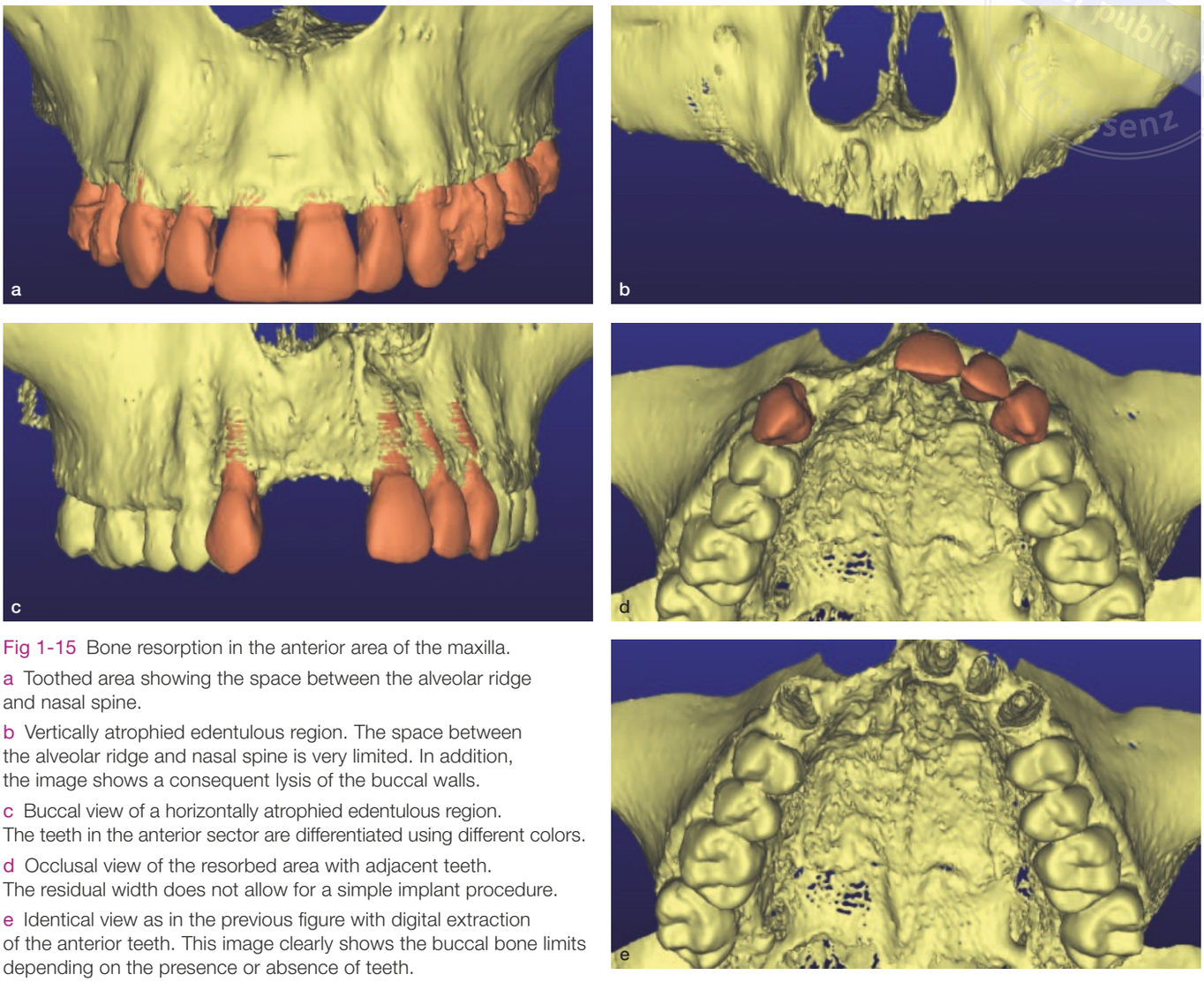


Fig 1-15 Bone resorption in the anterior area of the maxilla.

a Toothed area showing the space between the alveolar ridge and nasal spine.

b Vertically atrophied edentulous region. The space between the alveolar ridge and nasal spine is very limited. In addition, the image shows a consequent lysis of the buccal walls.

c Buccal view of a horizontally atrophied edentulous region. The teeth in the anterior sector are differentiated using different colors.

d Occlusal view of the resorbed area with adjacent teeth. The residual width does not allow for a simple implant procedure.

e Identical view as in the previous figure with digital extraction of the anterior teeth. This image clearly shows the buccal bone limits depending on the presence or absence of teeth.

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2

Hard and soft tissue physiology

S. Szmukler-Moncler, M. Davarpanah, J.P. Bernard

Objectives

At the end of the chapter the reader should be able to understand:

- The different stages of bone healing.
- Bone responses as a function of surface condition.
- The local and general factors that influence the bone response.
- The bone response when placing a healing abutment.
- The various elements of the arsenal that can minimize crestal bone loss.
- Bone response in case of peri-implantitis and how to distinguish it from occlusal overload.

Bone response to implantation: introduction

History of osseointegration

In the 1950s, Brånemark (1959) studied blood circulation and bone repair. He then planned to implant a metallic optical chamber in a long bone but did not know which metal to use so that the chambers would be well tolerated. An orthopedic surgeon, Emneus, studied several metals with the aim of creating hip prostheses (Emneus *et al*., 1960). Titanium, a little-known material at the time, seemed promising to him; it was mainly used in the then Soviet Union in the nuclear industry. Brånemark managed to obtain some and implanted his optical chambers in titanium. At the end of animal experimentation, these chambers were difficult to remove. He then had the idea of using titanium in bone surgery, more specifically as a dental implant to stabilize an implant-supported prosthesis (first patient in 1965). An experiment on dogs was carried out with implant-supported prostheses and was published in 1969 (Brånemark *et al*., 1969). It reported the long-term stability of the bone–implant interface (for up to 4 years).

With this publication, Brånemark and his collaborators were among the first to support the idea that the durability of a dental implant depends on direct contact, without fibrous interposition, between bone and implant. In 1977 (Brånemark, 1977), they devised a new term to reflect this concept, which was completely new in implantology. They called it 'osseointegration' (in French it becomes 'osteointegration'). They defined osseointegration as 'a direct anatomical and functional junction between the remodeled living bone and the surface of the loaded implant'.

This definition, which refers to the micron scale, is based on histologic observations made with an optical microscope (Fig 2-1). It consists of ankylosis that leads to clinical stability; it can be tested manually or with devices capable of delivering an objective, intensity-graded measurement. On radiographs, a direct bone apposition is visible around the implant surface and is characterized by the lack of a radiolucent zone (Fig 2-2).

Bone response to implantation according to Brånemark

In the 1980s, Brånemark and colleagues (1985, 1988) described a model of physiological bone response to implantation. According to them, any surgical preparation, however atraumatic, cannot avoid the creation of a zone of peripheric necrosis at the drill line (Fig 2-3). The extent of this zone of necrotic bone depends essentially on the local temperature rise during drilling and bone vascularization. After placement of an endosseous implant, necrosis of the adjacent bone may extend up to 1 mm.

According to this model, the first step in the healing process is the removal of the peri-implant bone necrotic tissue during drilling. At the same time, the blood clot that has formed in the gaps between bone and implant becomes calcified. The newly formed bone fills the gap left between the bone walls of the surgical site and the implant surface (Fig 2-3). This immature bone has low resistance to mastication forces. Bone remodeling into lamellar and Haversian bone strengthens its mechanical properties and extends from the 3rd to the 18th month. If sufficient time is kept before loading the implant, the implant surface will be covered with lamellar bone, which will later differentiate into Haversian bone. The new balanced situation between apposition and resorption at the bone–implant interface is finally achieved 18 months after implantation.

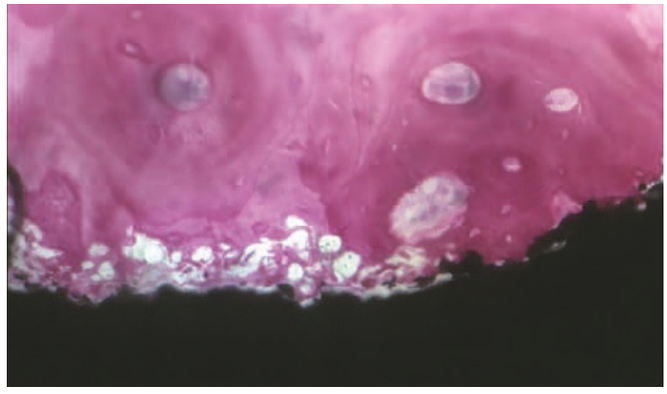


Fig 2-1 Histologic section obtained with an optical microscope of a bone–implant interface.
The implant surface is coated with titanium plasma spray and then hydroxyapatite. At this magnification, the contact is close and direct, with no apparent interposition of any fibrous tissue. Note the fragmentation of the coating (white grains) and its resorption (to the right of the interface). The implant is black, the bone is colored purple. The coating is white.

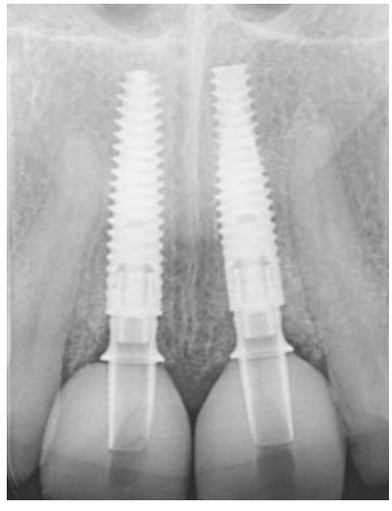


Fig 2-2 Radiograph of a conical implant after 5 months of loading. The implant is osseointegrated and there is no visible interposition between the implant and the surrounding bone. The resolution of the radiograph is 0.1 mm (100 µm).

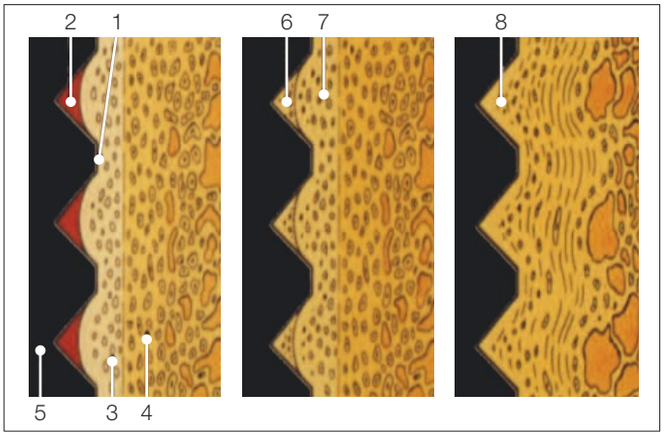


Fig 2-3 Bone physiologic response after implant placement according to Brånemark (Brånemark *et al.*, 1985).
A zone of bone necrosis around the drill line is noted.
Left: Situation immediately after implant placement: *(1)* immobilization of the implant in the bone; *(2)* hematoma contained in the gap delimited by the threads; *(3)* damaged bone area due to unavoidable necrosis caused by mechanical and thermal trauma; *(4)* undamaged bone at a distance; *(5)* implant.
Middle: The events taking place during the healing phase take place in a stress-free environment: *(6)* the hematoma is transformed into bone by means of a callus; *(7)* once the damaged bone has healed, remodeling takes place according to a demineralization/remineralization process.
Right: Situation at the end of the bone-healing period: *(8)* bone remodeling occurs at the bone–implant interface in response to the transmitted forces of mastication.

Prerequisites for achieving osseointegration and its long-term maintenance according to Brånemark

Between 1977 and the mid-1980s, the Swedish school argued that osseointegration can only be achieved and maintained over time if certain conditions are met (Szmukler-Moncler *et al.*, 2000). These are explained in Table 2-1. These conditions are restrictive and dogmatic. They require the use of titanium instruments (pliers and kidney dishes) to prevent contamination of the implant site.

The most characteristic prerequisite is placement using a two-step surgical protocol, with subgingival healing in 3–6 months. The goal is to protect the bone-healing process from:

- a possible inflammation;
- biomechanical stresses at the level of the implant neck;
- a possible epithelial exfoliating invagination.

This latency is essential to optimize the mechanical properties of the newly formed bone–implant interface before undergoing mastication stresses.

Review of Brånemark's prerequisites for osseointegration

The principles outlined by the Swedish school were strictly followed for about 10 years. Implant success rates were high, over 90% for many indications (Esposito *et al.*, 1998). Over time, simplifications were successively attempted. It was then realized that most of these recommendations were not essential; rather they served as a safeguard to allay people's concerns (Table 2-1).

Clinical and experimental studies proved that the Swiss school led by Schroeder (Schroeder *et al.*, 1976, 1979, 1981), which advocated implantation in one surgical stage, to be correct. Both methods led to identical bone reactions (Schroeder *et al.*, 1981; Gotfredsen *et al.*, 1991) and osseointegration rates (Buser *et al.*, 1991).

Thus, the original Swedish model of physiologic bone response to implant placement has been questioned. Several groups (Szmukler-Moncler *et al.*, 2000b; Massei *et al.*, 2001; Buser *et al.*, 2004) showed that drilling was not necessarily followed by a surrounding bone necrosis. Therefore, the bone apposition

1	Use of a compatible material, titanium in this case	Still current
2	Submerging the implants (two-stage surgical protocol)	Obsolete
3	Delayed loading of at least 3–8 months	Obsolete
4	Atraumatic bone drilling (low speed)	Still current
5	Making an incision in the vestibule with an offset gingival incision	Obsolete
6	Surgery in aseptic conditions, similar to the operating room	Obsolete
7	Use of titanium instruments (pliers, kidney dishes)	Obsolete
8	Radiographs contraindicated during the healing phase	Obsolete
9	Acrylic occlusal surfaces recommended	Obsolete

Table 2-1 Conditions enacted to achieve osseointegration according to Brånemark *et al.* (1985)
Of these nine conditions, only two are still in effect; the others are obsolete.

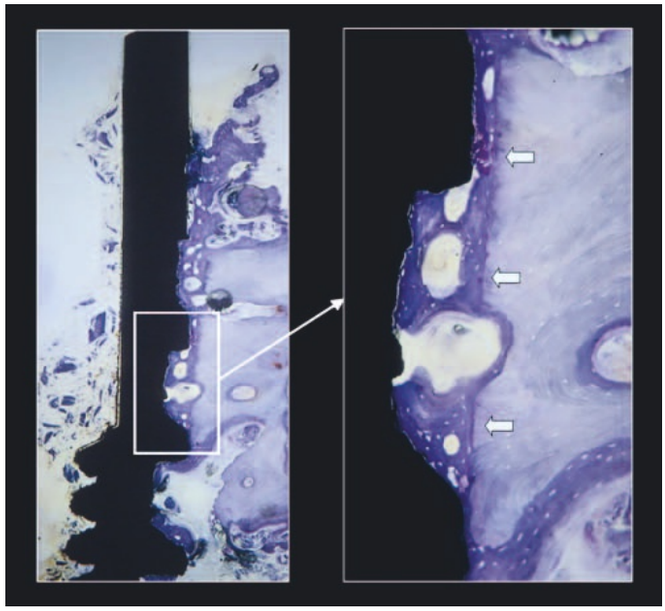


Fig 2-4 Histologic section of an immediately loaded implant in a human after 10 weeks.
Right: Magnification of the one on the left. The drilling line is easily visible, separating the existing bone from the newly formed bone. Thus, bone apposition did not pass through a remodeling step before newly formed bone is in contact with the implant (Massei *et al.*, 2001) (Histology Dr P. Trisi).

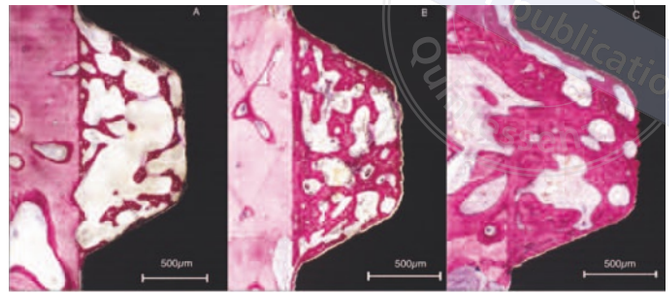


Fig 2-5 Histologic sections after 2, 4, and 8 weeks in miniature pigs.
a Section at 2 weeks. Bone apposition is performed directly from the original bone at the drill line, without going through a bone remodeling phase.
b Section at 4 weeks. The boundary between old and new bone is clearly defined. Bone apposition was started from the original bone.
c Section at 8 weeks. After filling the gap delimited by the implant, the new bone is finally remodeled. The boundary of the drill line that separated the old and new bone is no longer visible.
The implant is shown in black, the original bone is colored light pink, and the color of the newly formed bone is darker (Buser *et al.*, 2004).

(modeling) stage could begin without a prior remodeling stage of the necrotic peri-implant bone, as shown in the Figs 2-4 and 2-5. In addition, newly formed bone can cope with the stresses resulting from mastication (Piattelli *et al.*, 1998; Corso *et al.*, 1999; Szmukler-Moncler *et al.*, 2000b).

Integration of all these new physiologic data at the clinical level has made it possible to significantly reduce the time allocated to bone healing. Under certain conditions, loading an implant within 1 hour of placement is possible and accepted (Balshi *et al.*, 2005; Davarpanah *et al.*, 2007).

Bone responses leading to osseointegration

Definitions

The term 'osseointegration' describes a functional bone response to an implant; however, it does not describe the very different events that can lead to it. Therefore, these events need to be better defined.

Contact osteogenesis

Contact osteogenesis occurs when the newly formed bone around an implant starts directly from its surface, for example, titanium implant with a rough, etched surface (Davies, 2003).

Distance osteogenesis

Distance osteogenesis occurs when the newly formed bone around an implant does not start directly from its surface because it can only start from the pre-existing adjacent bone, for example, a titanium implant with a smooth surface (Davies, 2003).

Osteoconduction

A material or surface is said to be 'osteoconductive' when it leads to contact osteogenesis. However, osteoconductive capabilities can be variable, for example, hydroxyapatite is more osteoconductive than titanium (Davies, 2003).

Osteoinduction

A material or surface is said to be 'osteoinductive' when it induces the formation of new bone in a tissue environment not intended for bone formation. For example, porous hydroxyapatite can induce osteogenic activity on immediate contact when implanted in a muscle or the abdominal wall (Ripamonti *et al.*, 2012).

Common factors in bone healing

Bone reaction to the placement of an implant is not specific. It conforms to the rules and sequences of bone healing, which is

common to any bone fracture, drilling, or grafting. Unlike soft tissues, bone healing does not produce scar tissue under good conditions. At the end of healing, newly formed bone is no longer distinguishable from the pre-existing bone.

The necessary conditions for bone healing are:

- a stable surface on which to form;
- presence of adequate cells;
- adequate nutrition for these cells;
- an appropriate biomechanical environment.

The cells involved in bone formation are osteoblasts and osteoclasts (Fig 2-6). They are recruited from bone marrow or from undifferentiated mesenchymal cells in the bloodstream. At the bone site, the latter are expected to differentiate according to their osteoblastic lineage.

A vascular network must provide nutrition to these cells undergoing a differentiation process. Therefore, the conditions for bone healing in a trabecular bone surrounded by blood vessels are more favorable and a bleeding bone site has better osteogenic capacity.

Bone healing requires some mechanical stimulation. In the absence of any mechanical stimulation, the osteogenic capacity of the site to repair is reduced (Rubin & McLeod, 1994; Vandamme *et al.*, 2007). When this mechanical stimulation is too high, it results in large micromovements of the implant at the bone site. The differentiation of mesenchymal cells arriving at the site to be repaired is then diverted from the osteoblastic to the fibroblastic pathway (Büchler *et al.*, 2003). Around an implant undergoing too much micromovements, fibroblasts form a peri-implant fibrous bundle that is parallel to the implant axis: this fibrous integration is synonymous with osseointegration failure. Interest in a controlled mechanical stimulation is therefore understandable.

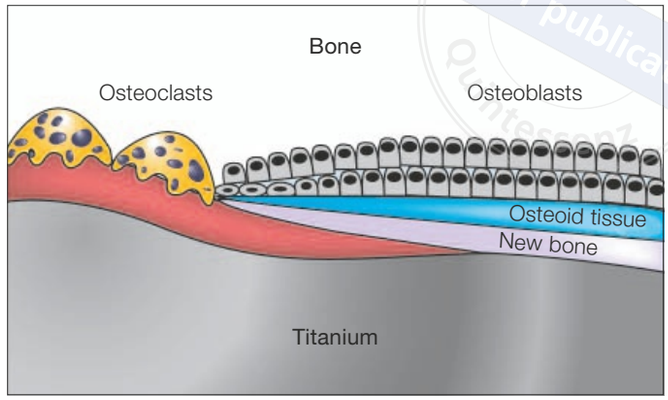


Fig 2-6 Cells involved in bone healing.
Osteoclasts resorb pre-existing bone. They are followed by the osteoblasts that adhere to newly formed bone. These cells turn into osteocytes and become quiescent cells.

The bone response around an implant is different in cortical than in cancellous bone because the bone and cell environments are distinct. We first consider the response in cancellous bone and then in cortical bone.

Bone typology (reminder)

This section is a reminder of what was described in Chapter 1. Classification of the bone quality encountered during implant placement is essential to categorize the various applications. The most commonly used classification is the one proposed by Lekholm & Zarb (1985), based on the distribution between cortical and cancellous bone tissue (Figs 2-7a-d):

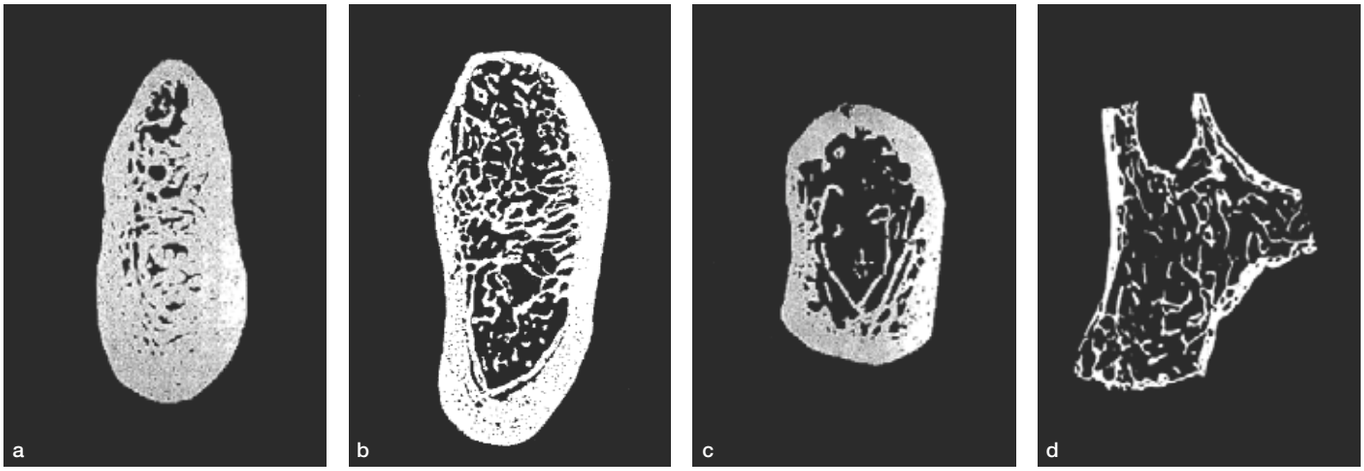


Fig 2-7 Classification according to Lekholm & Zarb (1985). Section of the mandible and maxilla corresponding to the typology stated.
a Type I in the mandible.
b Type II in the mandible.
c Type III in the mandible.
d Type IV in the maxilla (Ulm *et al.*, 1997, 1999).

- Type I bone is a dense bone, mainly composed of compact bone. Cancellous bone is almost nonexistent.
- Type II bone is composed of cortical and cancellous parts.
- Type III bone. The cortical part is fine and the cancellous part is consistent.
- Type IV bone. The cortex is almost nonexistent and the cancellous part is very reduced.

However, this classification is only relevant during a histologic evaluation. It is not appropriate clinically because a weak correlation between clinical sensation and histologic evidence has been found (Trisi & Rao, 1999). Therefore, clinically, only the classification into three groups with distinct bone quality proposed by Trisi & Rao is relevant. According to this classification, bone is (Table 2-2):

1. Dense. The clinician does not feel a noticeable transition between cortical and cancellous zones.

2. Regular. The clinician can clearly feel the transition from cortical to a weaker bone substance.

3. Low-density. The cortical and cancellous parts offer little resistance and both are easily passed.

Trisi & Rao classification	Lekholm & Zarb classification
Dense bone	Type I
Regular bone	Types II and III
Low-density bone	Type IV

Table 2-2 Trisi & Rao (1999) and Lekholm & Zarb (1985) classification of clinically perceptible bone qualities.

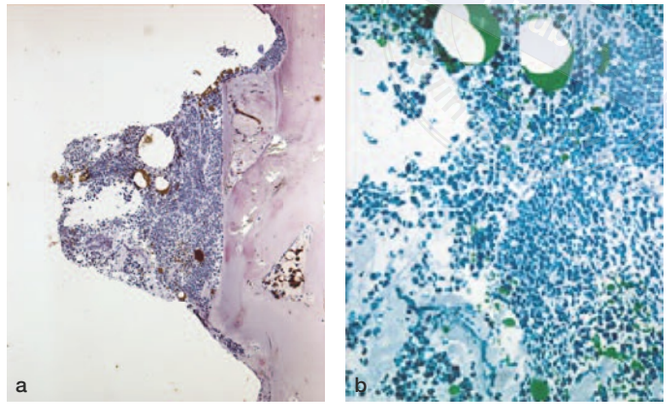


Fig 2-8 Clot formation within hours after implant placement. Example at 2 hours in a dog.

a Presence of a blood clot. It invades all available gaps between the bone and implant surface. Original magnification ×100.

b Characteristics of a blood clot. Erythrocytes and inflammatory cells are present but platelets are not visible at this magnification. Original magnification ×400 (Berglundh *et al.*, 2003).

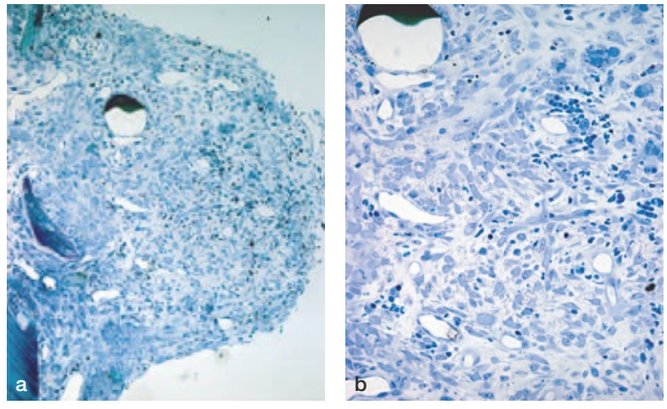


Fig 2-9 Angiogenesis in the days after implant placement. Example at 4 days in a dog.

a Clot persistence. The clot has invaded all the gaps between the bone and the implant surface and is still present. Original magnification ×200.

b Characteristics of a blood clot. Erythrocytes and inflammatory cells are present while platelets are not visible at this magnification. Original magnification ×400 (Berglund *et al.*, 2003).

Cancellous bone response

Stage 1: clot formation

Blood is the first substance to come into contact with the implant surface. After implant placement, a blood clot forms in the gaps between the drill line and material (Fig 2-8a). The cellular part contains red blood cells, platelets, and white blood cells (Fig 2-8b). Fibrinogen, the protein part, is deposited on the titanium material and allows preferential absorption of platelets at the surface. Immediately after absorption, platelets degranulate and release growth factors. The latter, by chemotaxis, attract undifferentiated cells to the wound site.

Stage 2: three-dimensional formation of a fibrin network

Once the clot has settled, a three-dimensional (3D) fibrin network is formed. It is followed by local angiogenesis (Fig 2-9). Through the newly formed capillaries, undifferentiated mesenchymal cells reach the healing site. If all biomechanical conditions are met locally, cells differentiate according to their osteoblastic lineage.

Osteogenic newly differentiated cells migrate toward the surface because they are attracted by signals emitted during platelet degranulation close to the surface. Their migration to the immediate vicinity of the implant surface is accompanied by fiber tension, causing some retraction. Depending on whether or not the fibers attached to the surface can resist traction, osteogenesis will continue in the direction of contact or distance osteogenesis.

Stage 3: first bone apposition

■ Contact osteogenesis

If the fibers are well anchored to the surface and resist cell traction, osteogenic cells can directly reach the implant surface (Fig 2-10). They recognize this surface as a stable and biocompatible surface, continue their differentiation into osteoblasts, and then express their phenotype. These osteogenic cells will first secrete a noncollagenous protein matrix, rich in osteopontin and sialoprotein, which is immediately mineralized. It is the equivalent of the cementation line, which is systematically encountered during any remodeling activity. Cells continue their bone apposition activity by producing woven bone. The latter is recognizable by the disorganized nature of its mineralized collagen fibers. Cells continue their bone apposition; secreting osteoblasts are included in the bone matrix and differentiate into osteocytes. Bone apposition continues centrifugally (from the implant surface to the pre-existing bone) (Fig 2-11) and centripetally (from the pre-existing bone to the implant surface; not shown) to immobilize the implant in the bone structure.

■ Distance osteogenesis

When the anchoring of the fibers to the implant surface is weak, usually because the surface does not have enough roughness for gripping, the fibers do not resist the traction of the osteogenic cells and detach from the implant surface (Fig 2-12). The migrating cells cannot directly reach the implant surface, so they remain at a distance from it. Bone apposition will be performed from the most stable adjacent surface, namely the edges of the drill line. As

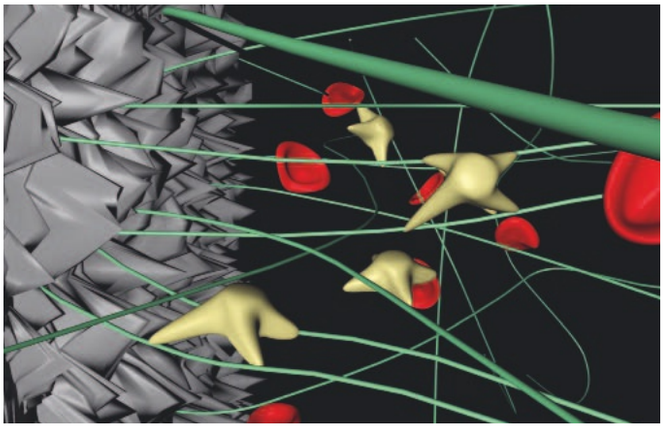


Fig 2-10 Contact osteogenesis.
The arrival of newly differentiated (yellow) cells is made possible up to the implant surface. This contiguity is due to the fibers (green), which maintain their attachment to the implant surface despite the traction caused by cell migration along the fibers. Red blood cells are shown in red.

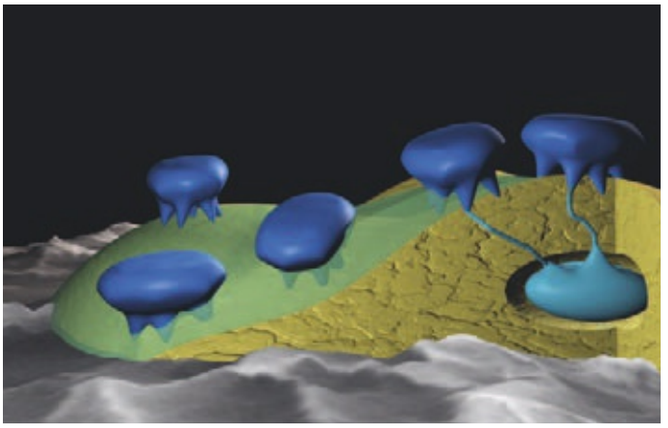


Fig 2-11 Centrifugal bone apposition from the rough surface.
The mineralized phase in direct contact with the implant is identical to the cementation line of the bone tissue. Shown on the section are bone (yellow), osteocytes (light blue) embedded in bone in their osteocyte gap, and osteoblasts (dark blue), which secrete a protein matrix (green) that is then mineralized.

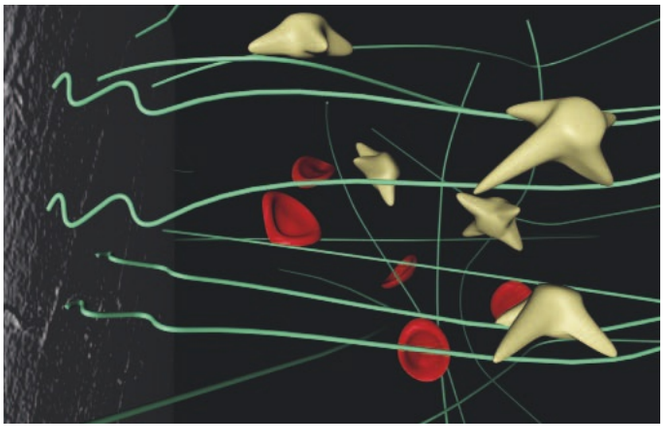


Fig 2-12 Distance osteogenesis.
The arrival of newly differentiated (yellow) cells to the implant surface is impossible. This is because the cells progressing along the fibers (green) generate a pulling force that is reflected at the surface. If the fibers are not sufficiently attached, they separate from the surface and (yellow) cannot reach the surface.

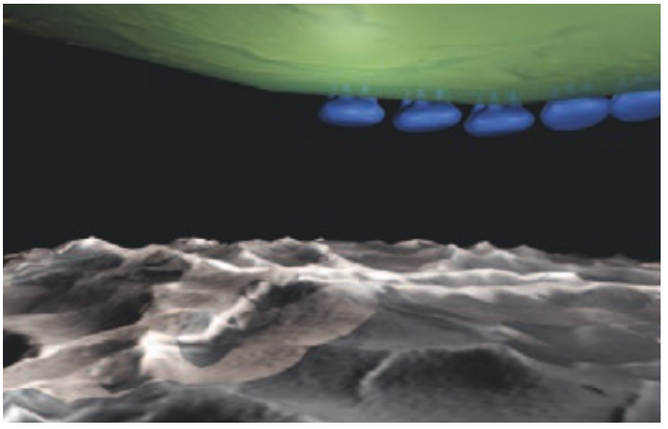


Fig 2-13 Centripetal bone apposition from the bone surface to the implant surface.
Progression is made from the bone edges and not from the implant surface. The bone tissue (yellow) and osteoblasts (dark blue) that secrete a protein matrix (green) that will be mineralized, can be identified.

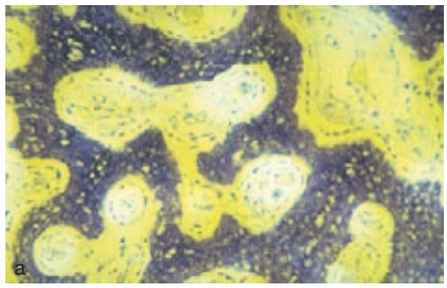


Fig 2-14 Woven bone.
Collagen fibers run in all directions randomly, without any particular organization. The view in polarized light with a quarter wave reveals the disordered character of the fibers.

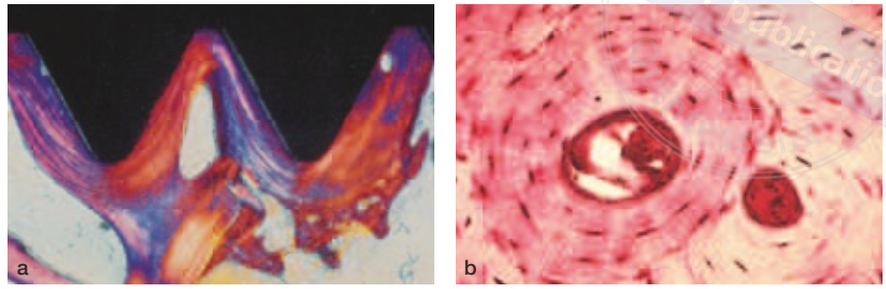


Fig 2-15 Lamellar and Haversian bone.

a Lamellar bone. Collagen fibers are organized in parallel lamellae. They are the result of a first remodeling after the woven bone stage.

b Haversian bone. Collagen fibers are organized in concentric circles and cells are arranged according to this geometry. They are the result of a secondary remodeling.

before, the noncollagenous protein matrix rich in osteopontin and sialoprotein is secreted and mineralized. The cells continue their bone apposition activity toward the implant (centripetal activity) (Fig 2-13), producing a woven bone (Fig 2-14) that is remodeled into lamellar (Fig 2-15a) and then Haversian bone (Fig 2-15b).

Stage 4: bone apposition and osseointegration

After the initiation of bone apposition, woven bone goes through all the stages of maturation and remodeling, that is, from woven (Fig 2-14) to lamellar bone (Fig 2-15a) with a parallel organization of collagen fibers, then to Haversian bone with a concentric circular organization of collagen fibers (Fig 2-15b). As the maturation stages progress, the mechanical properties of the bone increase.

However, the initial response, that is, contact or distance osteogenesis, is not without consequences on the long-term organization of the peri-implant bone structure (Table 2-3).

The surface condition induces a specific initial bone response, leading to a distinct bone structure that is maintained in the long term.

	Surface	
	Smooth	**Rough**
Initial bone response	Remote osteogenesis	Contact osteogenesis
Osseointegration	Corticalization	Trabeculation

Table 2-3 Osteogenesis and type of bone response.

■ 'Trabeculization' reaction

When the initial bone reaction is a contact osteogenesis reaction, bone apposition continues as 'trabeculization' (Fig 2-16a). Around the implant, the bone forms a thin, generally continuous layer of bone on which bone trabeculae oriented more or less perpendicular to the vertical axis of the implant are embedded. These trabeculae are connected to the surrounding bone; this organization persists over the long term (Fig 2-16b). This reaction is typical of a rough or bioactive surface.

■ 'Corticalization' reaction

When the initial bone reaction is a distant osteogenesis reaction, bone apposition continues as a 'corticalization' reaction (Fig 2-17a). Around the implant, the bone forms an enveloping shell of a certain thickness. Organization of this structure persists in the long term (Fig 2-17b). Corticalization is slow and takes time to reach the Haversian stage. This reaction is typical of a machined surface.

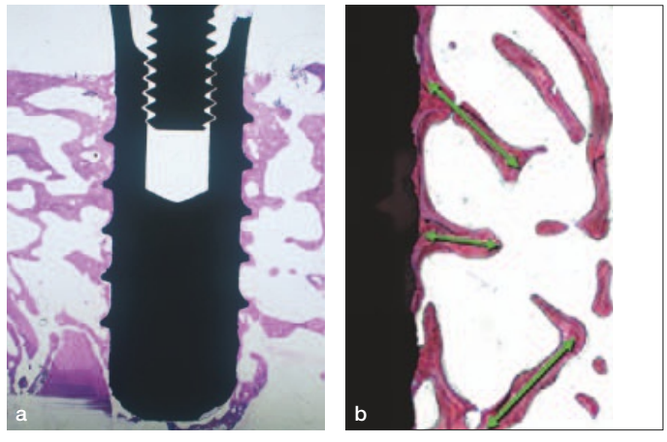


Fig 2-16 Bone reaction of trabeculization around rough-surfaced implants.

a Response at 3 months of healing. Bone trabeculae run toward the rough surface, without forming a dense shell around the implant. Implant placed for 3 months in a dog's jawbone is shown (Bernard *et al.*, 2003).

b Response after 18 months of loading. This cylinder with a surface roughened by plasma-sprayed titanium (PST) was biomechanically stressed for 18 months. Despite the long period of occlusal loading, trabeculization did not evolve into corticalization of the surrounding bone as shown in Fig 2.17.

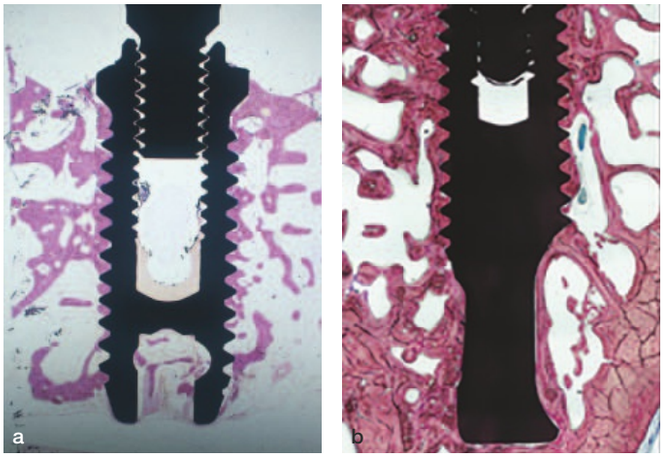


Fig 2-17 Bone corticalization around implants with machined surfaces.

a Response at 3 months. A bone shell condenses around the implant.

b Response after 18 months of loading. This implant with its machined surface was subjected to biomechanical stress for 18 months. Corticalization was even more extensive under the loading forces (Watzak *et al.*, 2005).

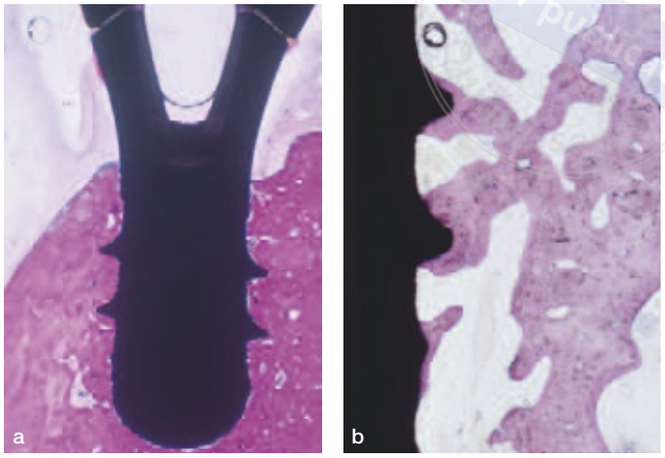


Fig 2-18 Different bone responses depending on bone quality.

a Cortical bone response. Example of bone reaction in dense bone to immediate loading in a dog for 7 months. Remodeling is slower.

b Cancellous bone response. Example of bone reaction in cancellous bone to immediate loading in a dog for 6 months. Bone trabeculae are projected preferentially on the threads to immobilize the implant in the fastest and most efficient way.

Cortical bone response

Cortical bone response is different from cancellous bone response (Figs 2-18a, b). The influence of surfaces on the cortical bone response is small compared to its effect in a cancellous bone environment (Jansen *et al.*, 1991). Thus, placing a bioactive surface in cancellous bone significantly increases the percentage of bone apposition around the implant. On the other hand, if placed in cortical bone, the bioactive surface cannot express its osteoconductive properties in the same way as in cancellous bone. In this environment, it loses all its relevance.

In the case of direct bone contact with the implant surface, bone remodeling at this location is delayed compared to the 2 weeks needed for cancellous bone (Berglundh *et al.*, 2003; Franchi *et al.*, 2005). It only occurs later, within 3 months. To achieve osseointegration, a local resorption phase is necessary to allow osteoblastic cells to express their phenotype, that is, to secrete the cementation line and achieve bone apposition. This stage takes longer in cortical bone than in cancellous bone because in the latter bone apposition can begin immediately at the implant surface (Berglundh *et al.*, 2003).

Bone response and local factors

Many factors can influence bone healing. The percentage of bone–implant contact is affected by:

- bone quality;
- the condition of the implant surface;
- the implant material and its shape;
- the surgical procedure;
- the healing time.

Bone response and bone quality

Subjectively, the clinician prefers placing an implant in dense (type I) bone to ensure the highest possible primary stability. However, the clinician should keep in mind that from a bone dynamics point of view, type 3 cancellous bone or even type 4 has a shorter healing period than type I or II dense bone. In addition, type III bone has a superior osteogenetic ability than corticalized type I bone (Wang *et al.*, 2017).

In implants with a rough surface (Fig 2-19a), the more cancellous the bone is, the faster and more effective the bone response will be (Lazzara *et al.*, 1999). A study conducted in the posterior jaw of patients with low bone density confirmed this assertion. After 3 and 12 months of healing, the rate of bone apposition increased over time from 59.0% to 76.7% (Trisi *et al.*, 1999b). On the other hand, it is low and stable over time for machined implants (<10%) (Trisi *et al.*, 1999b; Ivanoff, 2001). It is understandable why longer healing times have been recommended for machined implants in this type of bone (Ivanoff, 2001).

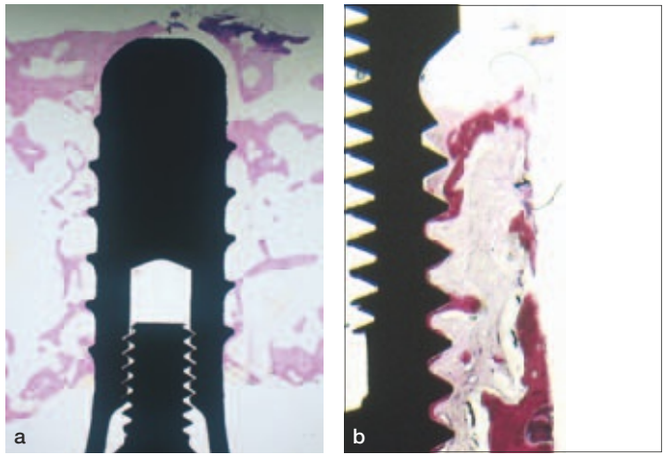


Fig 2-19 Bone response in low-density bone.

a Bone reaction in the maxilla of a dog after 3 months. The implant is immobilized by a few bone trabeculae, without the need for cortical bone. At higher magnification (not shown) a thin layer of bone covering the roughened PST implant is visible.

b Bone reaction of an immediately loaded implant in the mandible of a human. A thin but continuous layer of bone tissue covers the rough surface of the etched implant (Testori *et al.*, 2002).

It even seems that a bone response consisting of the formation of a thin bone shell around the implant (Fig 2-19b) is sufficient to distribute occlusal stresses and allow the implant to be loaded (Testori *et al.*, 2002).

Bone response and primary stability

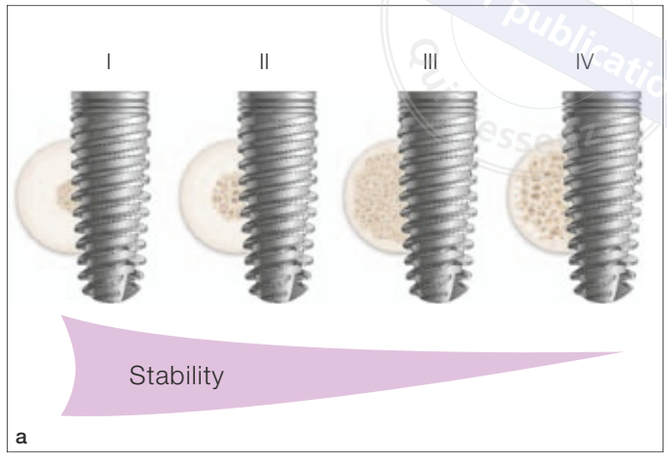
Primary stability is a determining factor (Fig 2-20a) in achieving osseointegration (Albrektsson *et al.*, 1981; Brånemark *et al.*, 1985). It is obtained essentially by the implant portion that is in contact with the cortical bone. The maxilla often has a thin external wall (Fig 2-7), which explains the greater difficulty in achieving primary stability (Fig 2-20b).

In low-density bone, primary stability can still be achieved by underpreparing the implant site. Tapping is omitted as well as the final drilling. It is also possible to prepare the implant site using osteotomes that locally condense a low-density bone or with self-tapping implants (Davarpanah *et al.*, 2011).

Bone response and drilling temperature

A local rise in temperature during drilling (Fig 2-21a) causes necrosis of the bone tissue in the vicinity of the drill line. This necrosis induces the formation of peri-implant fibrous tissue instead of osseointegration (Fig 2-21b).

Studies have shown that maintaining a temperature of 47°C for 1 minute induces cell necrosis (Lundskog, 1972; Eriksson *et al.*, 1984). On the other hand, maintaining a temperature of 50°C for more than 1 minute irreversibly disrupts bone healing (Fig 2-21b).



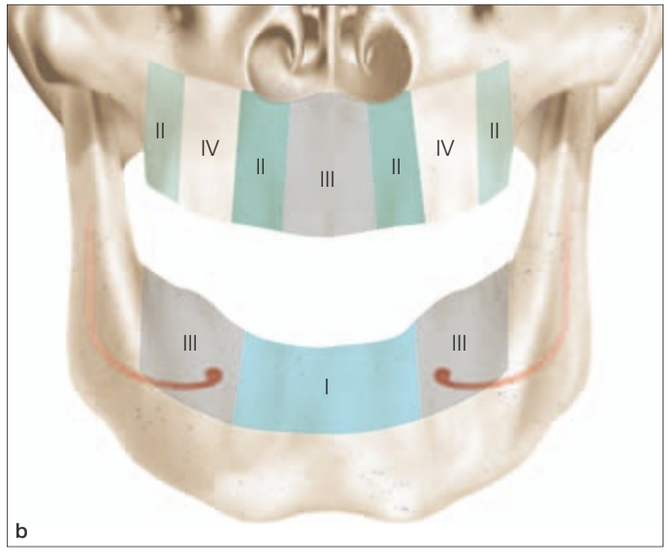


Fig 2-20 Primary stability and bone quality.

a Primary stability in various types of bone. Stability is more and more difficult to achieve as we progress through the typology. In low-density bone, specific techniques must be used.

b Repartition of bone quality in the upper and lower jaw. The difficulty in obtaining primary stability can be anticipated and it is possible to anticipate the use of an alternative method.

However, these data are further removed from clinical reality. Indeed, a temperature peak is immediately followed by a rapid decrease unless a strong force is still applied (Reingewirtz *et al.*, 1997). Thus, maintaining a temperature peak for 1 minute does not correspond to clinical reality.

To prevent excessive thermal increase, it is necessary to use sharp drills, appropriate drilling speeds in the range of 800–1,500 rpm (Reingewirtz *et al.*, 1997) (Fig 2-21c), and a graduated drilling sequence so as not to create a 3 mm or 4 mm implant socket in a single step, all of which must be accompanied by abundant irrigation.

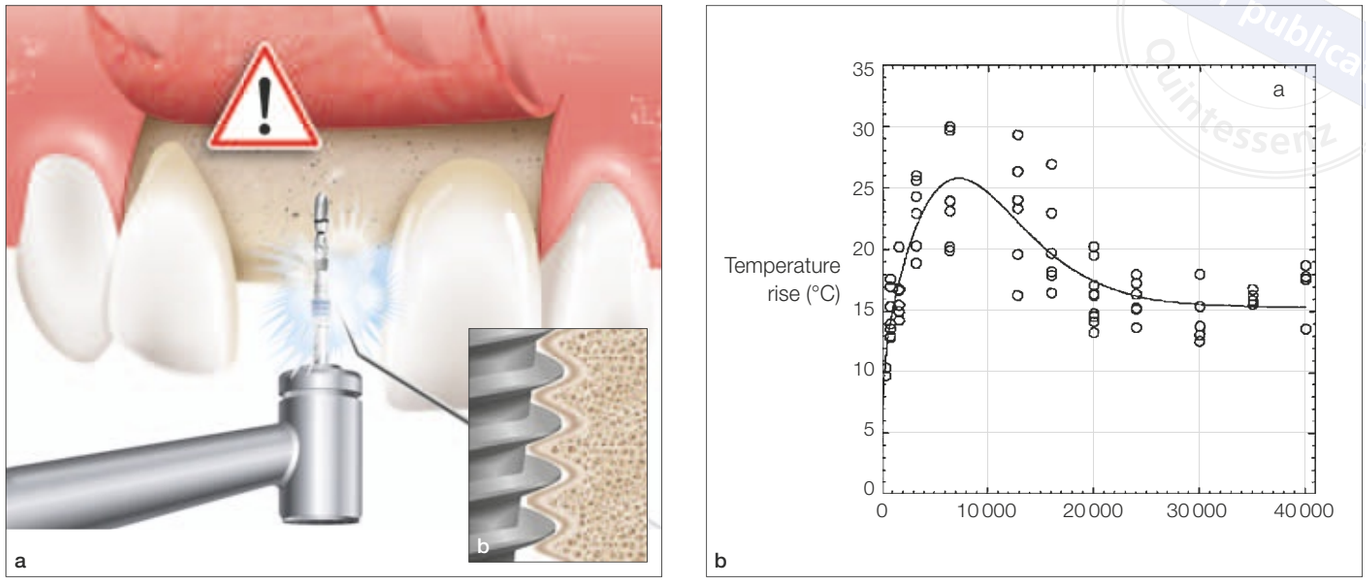


Fig 2-21 Bone response to local temperature rise.

a Threshold temperature not to be exceeded. This threshold is not exceeded when the drilling speed is less than 1,500 rpm and is accompanied by saline irrigation.

b Bone response when thermic necrosis occurs. The bone tissue at the interface is not newly formed; a capsule of fibrous tissue is formed.

c Evolution of temperature according to the rotation speed of the drill. By increasing the rotation speed, temperature increases until it peaks and then decreases again. The drilling speed will be limited to 1,500 rpm (Reingewirtz *et al.*, 1997).

Bone response and implants

Biomaterials

Dental implants are made from metallic and ceramic biomaterials (Table 2-4). The most biocompatible metals are:

- commercially pure (CP) titanium (grades 1–4);
- titanium alloys (titanium grade 5, titanium-zirconium);
- nobium;
- tantalum;
- zirconium (not to be confused with zirconia, which is the ceramic, that is, the oxide ZrO_2 made from metallic zirconium).

Metals	Ceramics	
	Bioinerts	Bioactives
Titanium CP grades 1–4	Al_2O_3	HA
Titanium alloys (grade 5, 23)	ZrO_2	TCP
Titanium-zirconium alloy		Brushite
Zirconium		
Nobium		
Tantalum		

Table 2-4 Materials used in implantology.

The corrosion resistance of these metals in contact with physiological fluids is high because the corrosion currents recorded are very low but not nonexistent; they are of the order of 2 nA/cm^2 due to the dense and stable titanium oxide layer that covers them (Fig 2-22). It is therefore the source of the excellent biocompatibility of these materials. The technology used to produce metal implants is machining from metal bars.

Unalloyed titanium is known as 'commercially pure' or 'CP'; it is different from titanium alloys. It nevertheless contains several other elements in the form of trace metals, of the order of 1%, which are called impurities. There are four grades of CP titanium, depending on the impurities (Table 2-5). The existence of trace elements increases the mechanical properties substantially. Thus, the breaking strength of grade 2 titanium is 345 MPa; it increases

Fig 2-22 Passivation of titanium due to a dense layer of corrosion-resistant titanium oxide.

	Titanium grade				
	Grade 1	Grade 2	Grade 3	Grade 4	Grade 5
Trace element, maximum in %					
N	0.03	0.03	0.05	0.05	0.05
C	0.10	0.10	0.10	0.10	0.08
H	0.015	0.015	0.015	0.015	0.015
Fe	0.20	0.30	0.30	0.50	0.50
O	0.18	0.25	0.35	0.5	0.2
Al	–	–	–	–	6
V	–	–	–	–	4
Breaking strength (MPa)	240	345	450	550	1,000

Table 2-5 The different grades of titanium.

to 550 MPa for grade 4 titanium, which has more trace elements in low concentration (Table 2-5). The use of grade 5 or 23 titanium alloy with 90% titanium, 6% aluminum and 4% vanadium (TAV) further increases the breaking strength up to 1,000 MPa (Table 2-5). Some CP-grade titanium is treated to further increase its mechanical properties to reach those of the TAV alloy.

The choice between the different titanium grades from 1 to 5 is made by the manufacturers according to their perception of the biocompatibility of the titanium in question. Users of grade 2 will emphasize the purity of the material despite its lower mechanical strength than grade 4 titanium or TAV alloy, which contains aluminum and vanadium. In fact, the TAV titanium alloy is widely used clinically in dental and orthopedic implantology and does not differ from CP titanium (Brunette *et al.*, 2001; Shah *et al.*, 2016). Some authors observed a tissue response to this alloy that is open to negative interpretation (Salaucic *et al.*, 2012). However, this result can be attributed to the questionable surface treatment used in this experimental study (sandblasting and acid rinsing) rather than to the biocompatibility of the alloy.

An alloy of titanium and 13–17% zirconium has recently been introduced in the market with the aim of increasing the mechanical properties of implants with reduced diameters (Barter *et al.*, 2012; Altuna *et al.*, 2016). However, the increase in its mechanical properties is poor, about 13–20% (Fig 2-23). They still remain below the mechanical properties of the TAV alloy. A study on implant fatigue (Fig 2-24) showed that the gain was variable, ranging from only 11% to 21%, depending on the type of implant (Medvedev *et al.*, 2016).

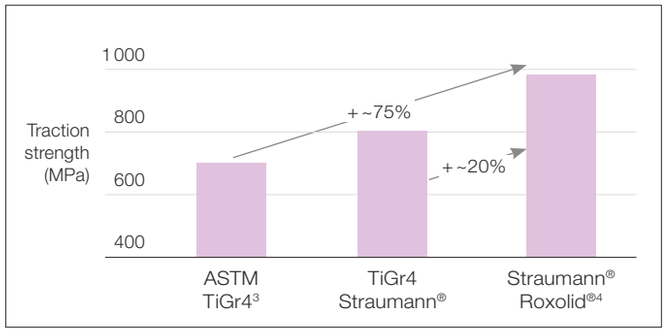


Fig 2-23 Mechanical properties improvement of the titanium-zirconium alloy compared to grade 4 titanium.
The improvement compared to the material in clinical use is only about 20%.

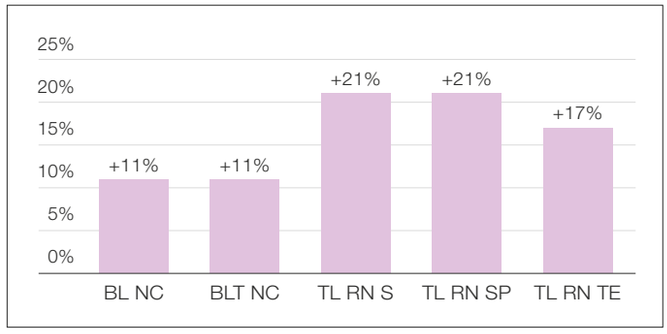


Fig 2-24 Improvement of the mechanical fatigue properties of Ø 3.3 mm titanium-zirconium alloy implants compared to grade 4 titanium depending on the implant type.
The fatigue gains of titanium-zirconium implants are modest, ranging from 11% to 21% at best (Medvedev *et al.*, 2016). BL, bone-level implant; TL, tissue-level implant; NC, narrow CrossFit; RN, regular neck; S, standard; SP, standard plus; TE, tapered effect.

Tantalum is another material that has long been known for its biocompatibility. It first appeared in oral implantology in 2012 after being used in orthopedics as porous tantalum (Kamath *et al.*, 2011; Unger & Duggan, 2011). It is known as 'trabecular metal' (Fig 2-25a). This tantalum surface is brought into contact with cancellous bone, mimicking the trabecular structure of cancellous bone (Fig 2-25b); studies have shown bone growth within this metallic reticular system (Bobyn *et al.*, 1999).

Implants can also be made of ceramics. The ceramics family is divided into inert and bioactive ceramics (Table 2-4). The biomechanical properties of bioactive ceramics are insufficient to use them for implants. However, they can be used as a surface coating to modify implant surface reactivity.

The best-known and most documented inert ceramics for their biocompatibility are alumina (Al_2O_3) and zirconia (ZrO_2) (Table 2-4). These materials consist of electrochemically stable, refractory metal oxides. Thus, ceramics are not subject to electrochemical corrosion. Their impact resistance is lower than that of metallic materials; however, in recent years it has greatly increased to 10. This is still below that of titanium, which is 100 (Brunette *et al.*, 2001).

They are implemented using 'powder technology', a process that is much more complex and sophisticated than machining.

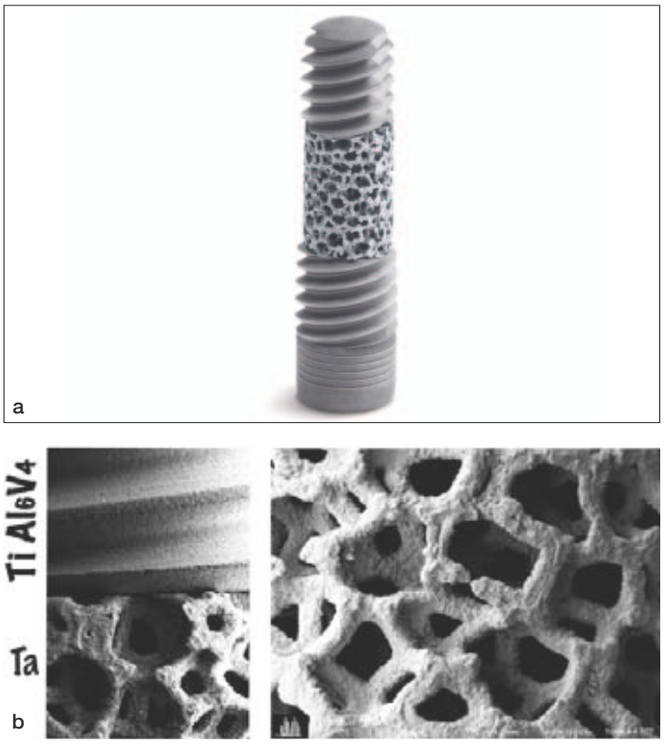


Fig 2-25 Tantalum implant surface structure.

a Implant with combined surface, with sandblasted TAV and porous tantalum mesh with a macrostructure resembling cancellous bone.

b Tantalum macroporous structure at higher magnification (original ×50).

A ceramic powder of a specific grain size is sintered at high temperature and under high pressure (high isostatic pressure) in a mold of a specific shape. The mold accommodates the shrinkage that occurs during sintering and produces a densified material with the required implant dimensions.

Bioactive ceramics dissolve at least partially in contact with biological fluids. The degree of dissolution depends on their physical and chemical properties. From the circulating fluids, they leave on their surface a more abundant quantity of proteins than titanium. The best-known bioactive ceramics are calcium hydroxyapatite ($Ca_{10}(PO_4)_6(OH)_2$) and calcium phosphate ($Ca_3(PO_4)_2$) (Table 2-4). The latter constitute a complex family formulated from a mixture of various metal oxides. They partially dissolve on the surface to form a layer rich in Ca^{2+} and phosphate (PO_4) ions that interacts with osteoblastic lineage cells. Due to their poor mechanical properties, bioactive ceramics are used as a surface coating or graft material. Their solubility depends strongly on their physical properties (porosity, grain size, impurities).

As a surface coating, they modify the osteoconduction of the implant surface. Osseointegration is achieved more quickly and the percentage of (cancellous) bone in direct contact with the implant surface is increased compared to bioinert metallic surfaces. Their use is optimal where low-density bone is involved (Iamoni *et al.*, 1999; Szmukler-Moncler *et al.*, 2003). These properties are responsible for their popularity in the early 1990s. However, the initial clinical benefit was severely compromised by poorly controlled side effects appearing in the longer term, generally after 5 years (Szmukler-Moncler *et al.*, 2003).

Over time, when peri-implant tissue recession occurs, the hydroxyapatite coating is exposed to the oral cavity. This happens all the more easily when hydroxyapatite-coated cylinders are placed in the posterior areas of the oral cavity. Bone cratering followed by gingival recession occurs (Watson *et al.*, 1999) in response to the high mechanical loads in these posterior areas, which are poorly distributed in the bone by the thread-free cylindrical design of the implants.

The high affinity between plaque and hydroxyapatite means that the coating is rapidly colonized by bacterial plaque organisms. Like tooth enamel and dentin, the coating dissolves when the pH value decreases locally. The dissolution progresses rapidly and simultaneously in all directions and spreads along areas of weak resistance. It can even reach the implant's most apical area without causing any external symptoms. Hydroxyapatite exacerbates peri-implant tissue inflammation, which can lead to considerable bone loss around the implant (Fig 2-26). Several studies have shown numerous long-term complications with this kind of surface coating. In France, hydroxyapatite coating

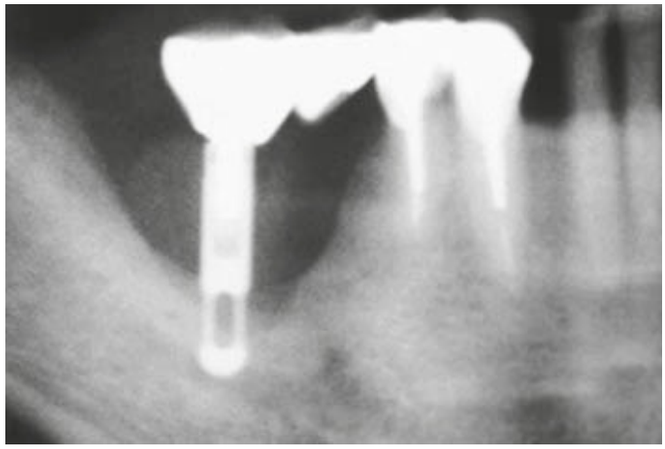


Fig 2-26 Significant bone resorption around a cylindrical implant, coated with plasma-sprayed hydroxyapatite.
Note that the cylindrical implant is located in the posterior area of the mandible. Bone loss is cup-shaped and suggests an infectious origin.

created by plasma spraying lost its marketing authorization in 2001. However, other bioactive coating formulations can avoid such long-term issues (Szmukler-Moncler *et al.*, 2003). They allow the immediate benefits of bioactive coatings without the long-term disadvantages (Davarpanah *et al.*, 2008).

Implant shape

Historically, several different implants designs have existed, shaped as baskets, blades, discs, solid or hollow screws, and solid or hollow cylinders. Some shapes have completely disappeared. The most common form is the screw implant (Fig 2-27), which has greater clinical benefit. Its functionality without clinical complications over three decades has been amply demonstrated (Esposito *et al.*, 1998). The presence of a screw thread improves primary stability but above all it allows good stress distribution in the bone.

Cylindrical (non-threaded) implants have shown a consistent clinical decline (Willer *et al.*, 2003; Karousis *et al.*, 2004). However, circumferential resorption in the form of cratering has been reported often, especially in posterior areas (Mersicke-Stern *et al.*, 2001) where occlusal stresses are higher. The reason for this marginal bone loss is due to the reduced ability of the cylindrical design to distribute stress in the bone tissue compared to a threaded implant.

Implant surface

The peri-implant tissue response depends on the physical and chemical properties of the implant surface (Fig 2-28). Surface treatment serves two distinct purposes: *(1)* to create surface roughness; and *(2)* to make the surface bioactive. In both cases, osteoconductive properties are improved and the bone response in cancellous bone is positively altered. Instead of corticalization, trabeculization is established. The bone percentage at the bone–implant interface and anchoring of the implant in the bone are increased; osseointegration is achieved more rapidly. In addition, the distribution and rate of failures are altered. With smooth surfaces, the failure rate is higher during the healing phase (primary failure) but also during the first year of loading (secondary failure). With rough surfaces, the primary and secondary failure rates are lower. It is rare for an osseointegrated implant to lose its integration during the first year due to loading.

The various surface treatments and variations in bone response between smooth and rough surfaces are detailed in Chapter 3, where the various surface types available on the market are discussed.

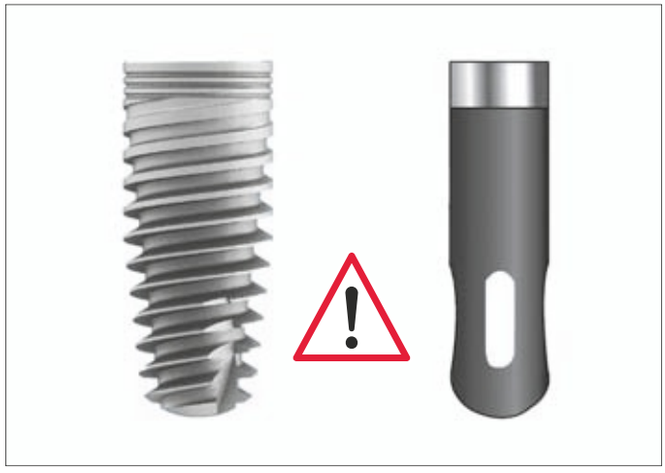


Fig 2-27 Benefits of tapered implants versus cylindrical (non-threaded) ones.

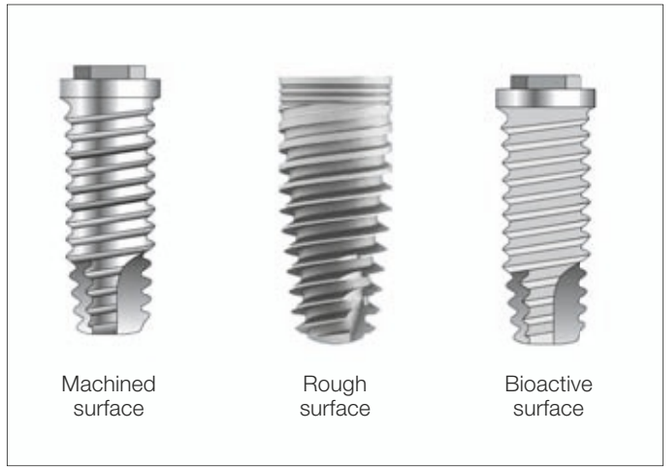


Fig 2-28 Various types of implant surfaces.
The machined surface (left) can be roughened by sandblasting, acid etching, or anodic oxidation (middle) or made bioactive by depositing hydroxyapatite nanoparticles (right).

Bone response after loading

Once osseointegration has been achieved and controlled, the actual prosthetic step can begin. A prosthetic abutment is connected to the implant, usually by screw connection. This custom abutment or non-custom abutment is then fitted with a crown.

However, the connection of the prosthetic abutment to the implant neck will induce soft and hard tissue reactions. The purpose of this section is to describe these reactions, which take 2–4 months to occur and reach a balanced situation.

These reactions depend on whether the implants were submerged, under the soft tissues, or whether healing was transmucosal in contact with the oral environment. Similarly, when healing is transmucosal, reactions can differ depending on whether the connection between the prosthetic abutment and implant is made at the soft tissue level with a single-stage implant (no soft tissue perturbation during the connection of the prosthetic abutment; so-called 'tissue-level' implant) or at the bone crest level with a two-stage implant (soft tissue perturbation during the connection of the prosthetic abutment; so-called 'bone-level' implant).

Single-stage procedure

At the end of the osseointegration period, the healing abutment is disconnected. It does not disturb the soft tissues that matured during the bone-healing period. The prosthetic abutment is connected to its crown without disturbing the soft tissue.

The greatest bone loss, of the order of 0.6 mm to 1 mm, occurs during the healing period (Pham *et al.*, 1994; Fiorellini *et al.*, 1999). After implant loading, it continues but it is smaller than before, of the order of 0.2 mm (Pham *et al.*, 1994; Weber *et al.*, 2000).

Two-stage procedure

For 2-stage implants, there is little or no bone loss when implants are submerged (Fiorellini *et al.*, 1999; Testori *et al.*, 2002). Crestal bone loss begins with the connection of the healing abutment and stabilizes over the next 3–4 months (Pilliar *et al.*, 1991). During the first year, it results in a vertical lysis of 1 mm to 1.5 mm around the first thread (Albrektsson *et al.*, 1986; Jung *et al.*, 1996). It is a physiological and non-pathological response to implant loading.

Two-stage implants are defined by two distinct clinical realities:

- The implants are completely submerged by soft tissue during the bone-healing phase.
- The implants are transmucosal during the healing phase because a healing abutment was connected immediately after placement.

In both cases, connecting the prosthetic abutment will disrupt the mucogingival junction that was put in place during the osseointegration phase. This disturbance will induce tissue remodeling around the abutment–implant junction. It will affect the crestal bone and the soft tissue. The response to disturbances is in accordance with the principle of biologic width conservation. The purpose of the following section is to explain this principle of hard and soft tissue biologic response to disturbances, whether biologic or biomechanical in origin, and explain how they lead to a physiologic response of vertical and horizontal bone loss at the crestal level.

The principle of conservation of biological space and its clinical implications within the three planes of space are successively considered:

- In the mesiodistal plane: it is expressed as the minimum distance between two implants or between a tooth and an implant.
- In the bucco-palatine plane: it is expressed as the minimum distance between the outer edge of the implant and the edge of the vestibular cortex.
- In the apico-coronal plane: it is expressed as supra, juxta, or subcrestal positioning of the implant–abutment junction and consequently of the implant neck.

Conservation of biologic width

As with teeth, a biologic width has been identified with implants. The concept of biologic width requires explanation. It means that regardless of implant loading, the tissue junction or tissue seal that separates the internal from the external oral environment around an implant always consists of three levels of relatively stable and constant dimensions over time (Hermann *et al.*, 2000); it varies very little (Fig 2-29a):

- a sulcus of 0.5 mm to 1 mm;
- an epithelial attachment or junctional epithelium of 1–2 mm;
- a 1–2 mm connective tissue attachment.

These three levels form a mucoepithelial attachment that is at least 3-mm thick.

If aggression, whether bacterial, mechanical, or iatrogenic in origin, damages the structure of the mucoepithelial attachment or reduces it to less than 3 mm, conservation of biologic space requires that the bone in the vicinity of any aggression is always protected by the same tissue structure, identical to the structure that exists when tissue is healthy. To preserve bone tissue located at a distance from the disturbance, the structure reorganizes itself at a distance and the original dimensions of the three components are reformed. To protect against aggression,

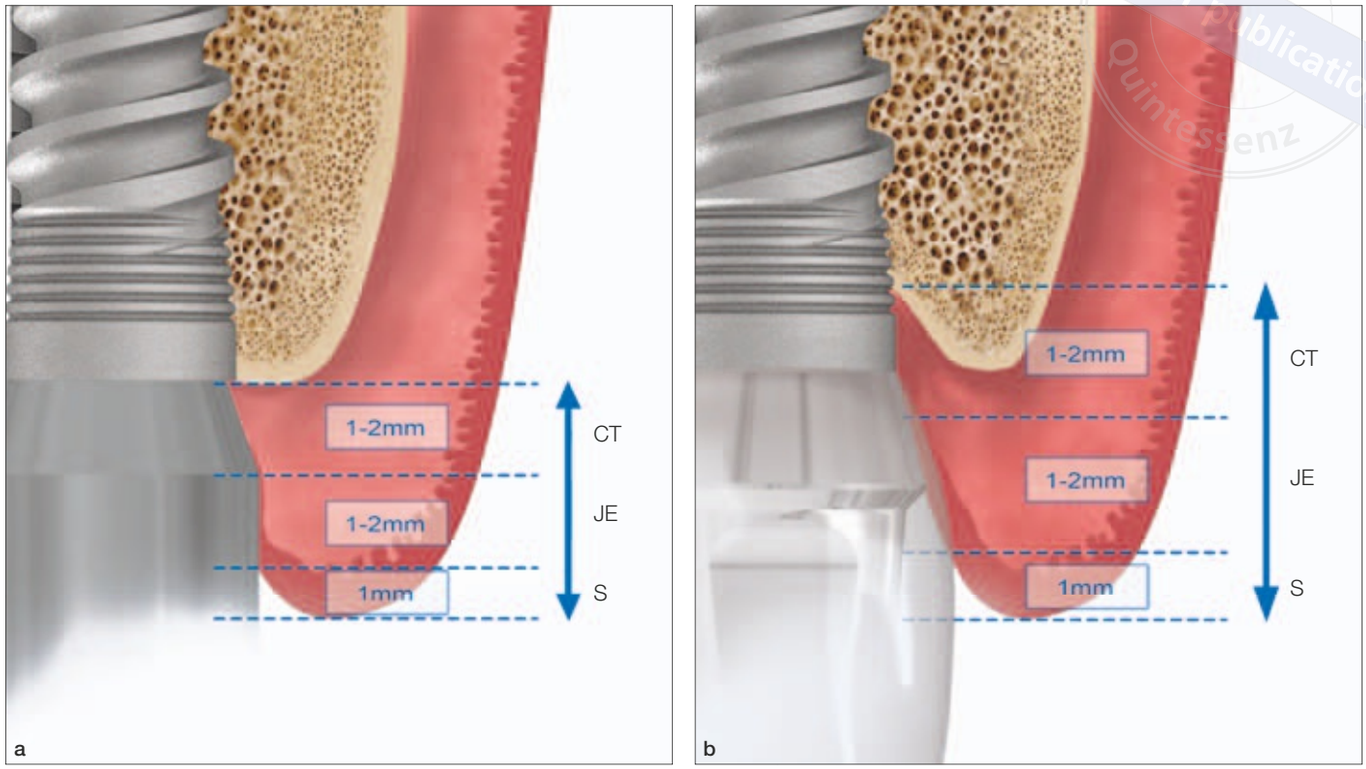


Fig 2-29 Biologic width establishment.

a Situation immediately after placement of the healing abutment.

b Situation after expression of the principle of the biologic width conservation. Apical bone lysis must take place to conserve the structural levels.

the level of the most coronal bone–implant contact is pushed back in an apical direction, until the 3 mm of biologic space is restored. Thus, application of the principle of biologic space conservation may lead to apical bone lysis (Fig 2-29b).

There is another reason for local disturbance of the mucoepithelial attachment. This occurs during the connection step of the prosthetic abutment. This is the repeated disconnection of the healing abutment for two-stage implants (Abrahamsson *et al.*, 1997). This local and time-determined disturbance causes the epithelial attachment to migrate from the lower or middle part of the abutment to the implant neck.

To comply with the principles of the biologic width conservation, epithelial migration must result in an apical transposition of the attachment structure. For it to take place and respect its dimensional organization, bone lysis must occur, around the entire implant circumference, in a cratering shape around the implant neck (Figs 2-31a, b).

Aggression on peri-implant mucoepithelial attachment

The implant device consists of the implant, prosthetic abutment, and crown. Assembly of these components results in junctions, namely the implant–prosthetic abutment and crown-prosthetic abutment junctions. Under the effect of masticatory forces, the assembly is made dynamic and generates mechanical and bacterial disturbances at the newly formed biologic junction. These disturbances constitute an aggression to which the peri-implant tissues respond by protecting themselves depending on their specific mode, that is, by complying with biologic width conservation.

Different sources of chronic disturbances have been identified (Fig 2-30). According to the hypotheses formulated in the literature, they are due to the presence of:

- a micro-hiatus at the implant–abutment junction (microgap) (Hermann *et al.*, 2001);
- a bacterial infiltrate along the implant–abutment junction (Ericsson *et al.*, 1996; Piattelli *et al.*, 2001; Broggini *et al.*, 2003);
- abutment micromovements in relation to the implant (King *et al.*, 2002);
- loadings related to occlusal function (Miyata *et al.*; 2002).

Transient disturbances have also been identified and are due to repeated prosthetic manipulations (screwing and unscrewing the abutment) (Abrahamsson *et al.*, 1997; Canullo *et al.*, 2010a,b).

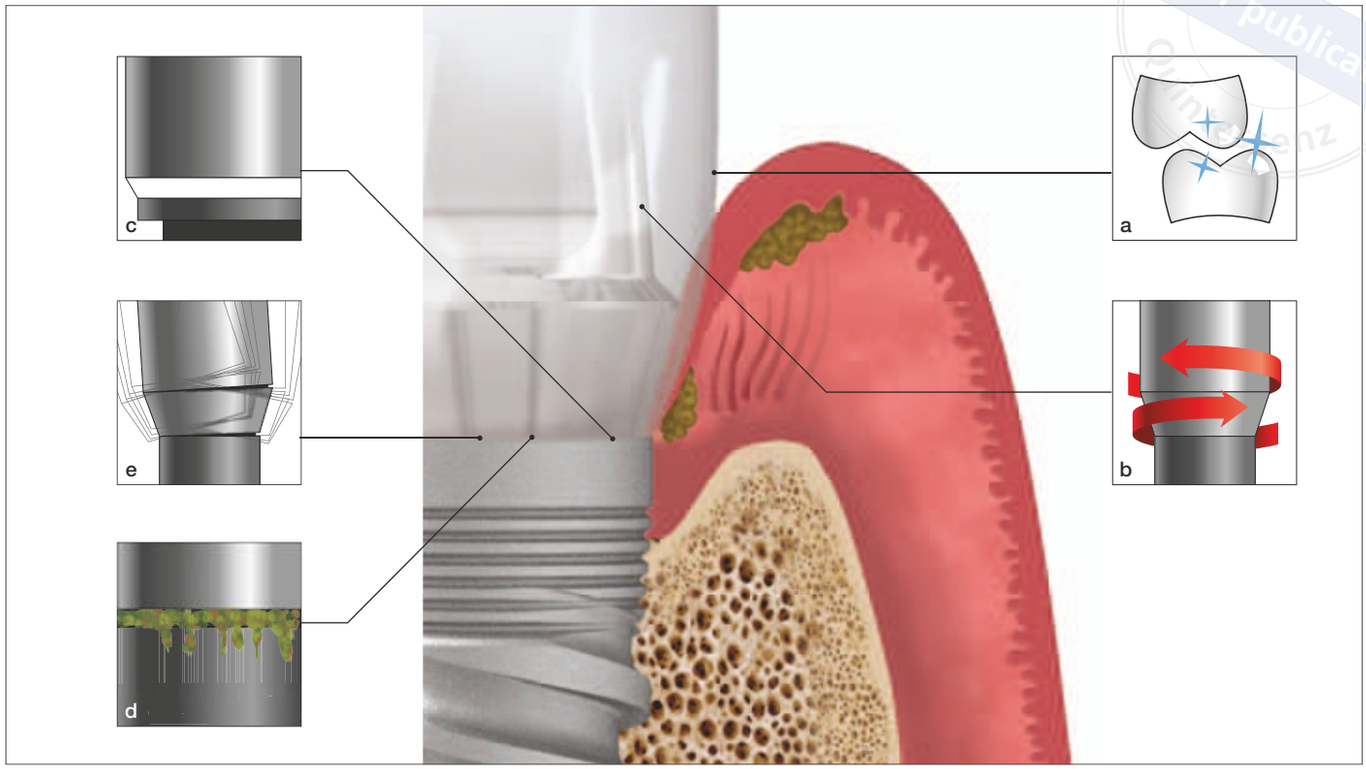


Fig. 2-30 Reasons for aggression and expression of conservation of biologic width.

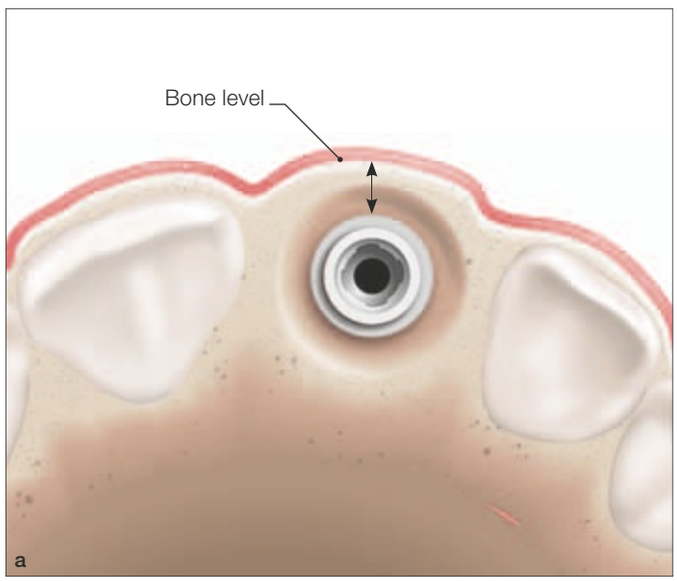
a Presence of a micro-hiatus at the implant–abutment junction.

b Unscrewing and screwing at the implant–abutment junction.

c Micromovements at the implant–abutment junction.

d Presence of bacterial infiltrate at the implant–abutment junction.

e Occlusal stresses.



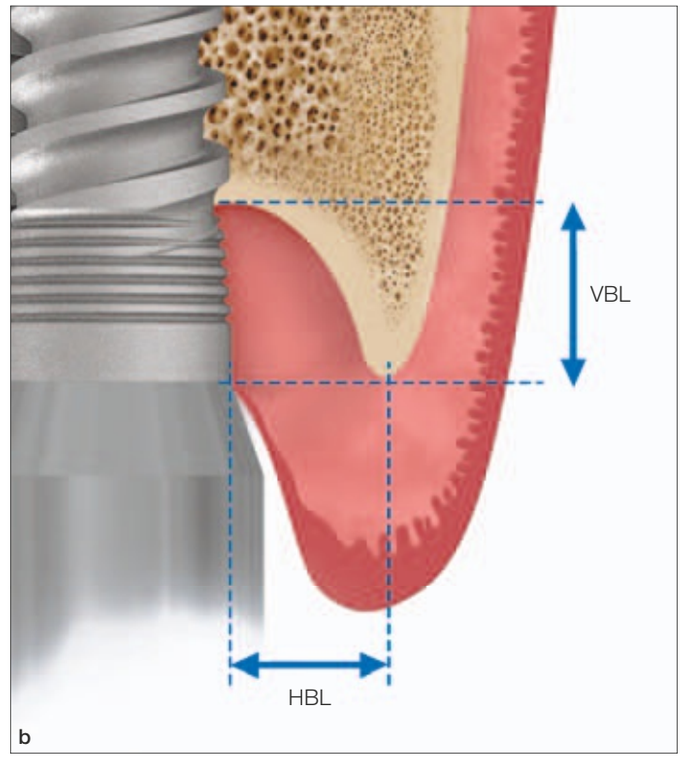


Fig 2-31 Consequence of the expression of conservation of biologic width.

a View from above. Note the cratering all around the implant at the implant–abutment junction.

b Longitudinal view. Cratering results in vertical and horizontal bone loss.

Biologic consequences of aggression on the implant/peri-implant tissue relationship

■ Minimum mesiodistal distance required between implant and adjacent structures (implant or tooth)

The principle of keeping a minimum distance between two elements originates from the principle of conservation of biologic width in the mesiodistal plane. The minimum distance to be maintained between 2 implants is 3 mm. It is 1.5 mm between implant and adjacent teeth.

In fact, during the first months of loading and after aggression whose origin is not yet well defined, a simultaneous vertical and horizontal bone lysis takes place for the two-stage implants, as shown in Fig 2-31a. This lysis involves the surrounding bone over a radius of 1 mm to 1.5 mm. Vertically, it results in apical migration of the peri-implant bone (Fig 2-31b). Horizontally, it results in lateral bone loss over an equivalent distance (Fig 2-31b).

When the distance between two implants is:

- less than 3 mm (2 mm × 1.5 mm), the interimplant bone ridge is resorbed over its entire height (Figs 2-32a, b) (Tarnow *et al.*, 1992). It can no longer provide support for the papilla, which migrates in an apical direction until sufficient bone support is regained.
- greater than 3 mm, the interimplant bone ridge is preserved (Figs 2-32c, d). The papillae have sufficient bone support and are present. Esthetics can be maintained over the long term.

The origin of this bone lysis is probably related to the presence of the chronic disturbances mentioned previously. However, the precise reason(s) have not yet been clearly identified.

■ Buccolingual distance from the outer edge of the implant to the edge of the buccal plate

The principle of a minimum distance between the edge of the implant and the edge of the buccal plate (Saadoun *et al.*, 2004) also originates from the principle of conservation of the biologic width in the buccolingual plane. The minimum distance to be maintained is 2 mm (Figs 2-33a, b).

When the thickness of the buccal plate is less than 2 mm, the 1.5-mm circumferential bone lysis reaches the buccal plate over its entire thickness. Thus, the first bone–implant contact is more apical than at the time of implant placement. The thinner the buccal plate, the greater the gingival recession. When the thickness of the buccal plate is greater than 2 mm, circumferential bone lysis does not reach the buccal plate and its coronal limit is not affected. The bone fulfills its role of supporting the marginal gingiva and esthetics are preserved.

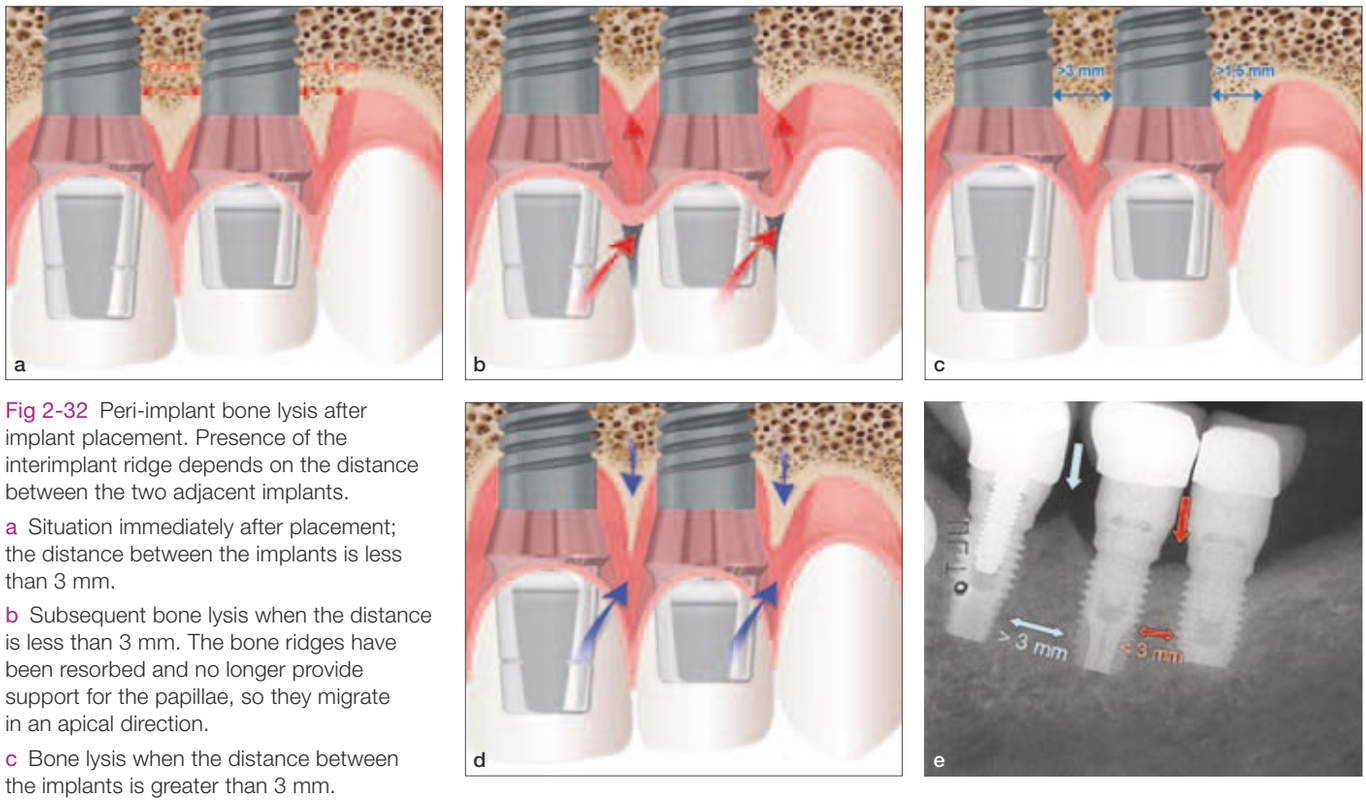


Fig 2-32 Peri-implant bone lysis after implant placement. Presence of the interimplant ridge depends on the distance between the two adjacent implants.

a Situation immediately after placement; the distance between the implants is less than 3 mm.

b Subsequent bone lysis when the distance is less than 3 mm. The bone ridges have been resorbed and no longer provide support for the papillae, so they migrate in an apical direction.

c Bone lysis when the distance between the implants is greater than 3 mm.

d Diagram of bone lysis when the distance is greater than 3 mm. The bone ridges still provide support for the papillae, which are then stabilized.

e Radiograph showing bone lysis depending on the distance between the implants. The bone ridge is present when the minimum distance between implants is maintained. The ridge is no longer present when the minimum distance is not maintained.

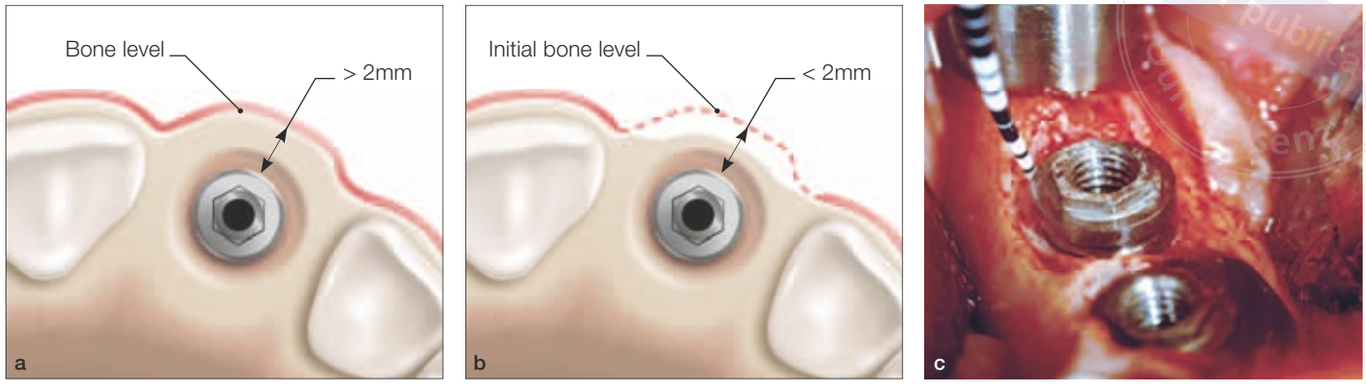


Fig 2-33 Bone lysis in the buccolingual direction.

a When the distance between the outer edge of the buccal plate and the implant is greater than 2 mm. The outer plate is not affected by cratering. Soft tissue support is preserved.

b When the distance between the outer edge of the buccal plate and the implant is less than 2 mm. The outer plate is then reached by cratering. Tissue support is no longer assured, resulting in gingival recession.

c Craterization observed after implant placement (photo: Prof T. Testori). The probe shows a depth approaching 3 mm.

Figure 2-33c shows a clinical case where the clearly visible cratering did not affect the buccal plate because the distance between the outer edge of the implant and the edge of the buccal plate was greater than 2 mm.

The optimal positioning of the implant neck and thus the abutment–implant junction also originates from the principle of conserving biologic width in the apico-coronal plane.

It would seem that:

- The implant–abutment junction apically pushes back the first bone–implant contact by approximately 1–2 mm. The more subcrestal the junction, the more lysis of the ridge is to be expected.
- A supracrestal positioning of 1–2 mm of the implant-abutment junction is necessary to avoid ridge lysis due to the presence of the implant–abutment junction.

Thus, the optimal placement of an implant in this case should take into account:

- bone resorption due to:
  - surgical trauma (of the order of 0.6 mm to 1.0 mm);
  - subcrestal placement of the implant–abutment junction.
- but also the height required by the dental technician to achieve a harmonious prosthetic emergence profile.

Bone response to the occurrence of infectious or biomechanical disease

During the functional life of an implant, several factors can lead to bone loss; they can be physiologic (Marcelis *et al.*, 2012) or pathologic. In the latter case, the pathologic origin is infectious, biomechanical, or combined (Giovannoli & Renvert, 2012). A few studies showed that bone resistance to occlusal overload is weakened in the presence of infection (Miyata *et al.*, 2002; Kozlowski *et al.*, 2007).

When bone loss is detected radiographically, it is important to be able to recognize its most obvious origin. This allows the appropriate clinical measures to be taken, that is, either to treat the infection in the case of peri-implantitis or identify the reason for the occlusal overload and treat it.

Infectious disease: peri-implantitis

Peri-implant tissue inflammation after bacterial infection can lead to bone resorption. This resorption is recognizable by its cup shape (Figs 2-34a-c). This specific shape was attested by causing experimental peri-implantitis in dogs using ligatures (Figs 2-34b, c) (Persson *et al.*, 2001; Schou *et al.*, 2003; Kozlowski *et al.*, 2007).

Cup-shaped bone loss due to infection is easily recognizable clinically on a radiograph, both around implants with rough surfaces (Fig 2-35a), coated with hydroxyapatite (Fig 2-35b) and around machined implants (Fig 2-35c).

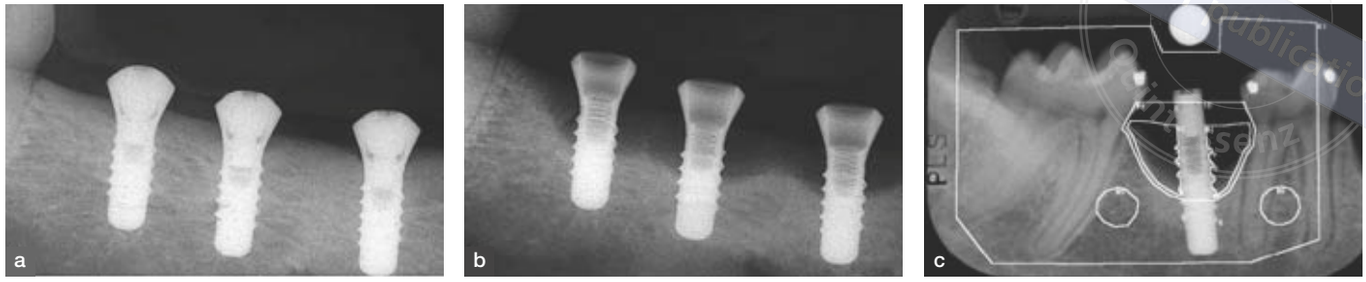


Figure 2-34 Bone defect of infectious origin experimentally created in dogs using ligatures.

a Radiograph before ligature placement (Persson *et al.*, 2001). Note the bone level near the tulip-shaped neck of the implant.

b Radiograph after ligature placement (Persson *et al.*, 2001). Cup-shaped bone loss is visible. The inflammation is so extensive in this case that horizontal loss is also noticeable.

c Cup-shaped bone loss (Schou *et al.*, 2003). The white line shows the extent of bone damage. The cup shape is attested in dogs.

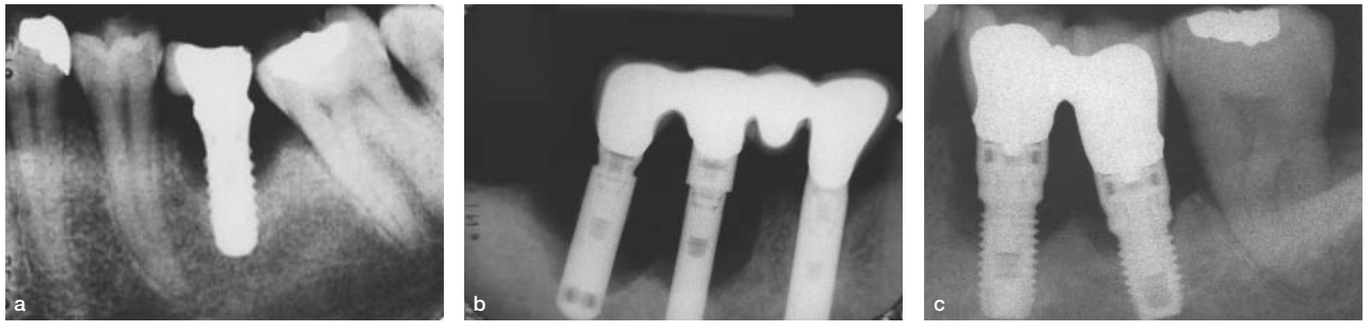


Figure 2-35 Bone defects of infectious origin in a patient, cup-shaped and radiographically revealed.

a Implant with rough surface obtained with a titanium plasma spray (Meriscke-Stern *et al.*, 2001).

b Implant coated with a hydroxyapatite plasma spray. Bone loss is more extensive due to the presence of the bioactive coating, which promotes inflammation.

c Implant with machined surface. Inflammation reaches the mesial and distal aspects of the most mesial implant. On the other hand, only the mesial aspect of the distal implant is affected (radiograph by Dr J.L Giovannoli).

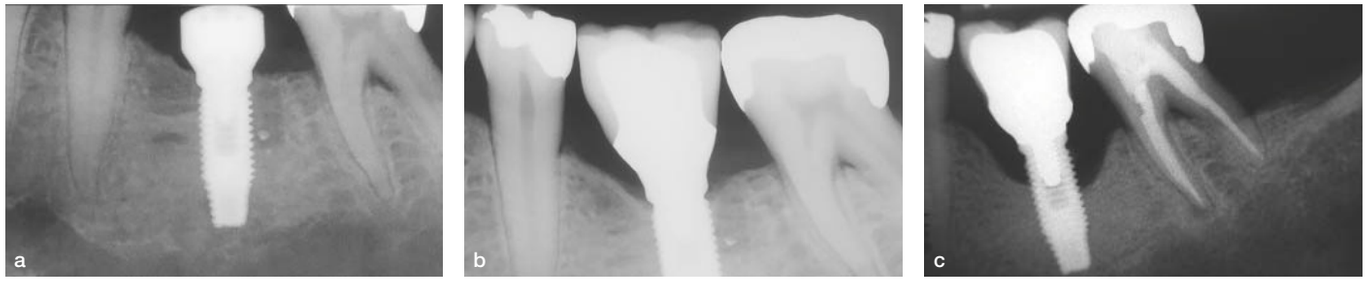


Fig 2-36 Bone adaptation to occlusal overload in the mandible.

a Postoperative situation, before implant loading. The bone ridge is homogeneous and the bone is at the implant neck level.

b Ridge densification at the 1-year recall. The latter is particularly visible on the mesial aspect.

c Peri-implantitis after 4.5 years of loading. The combination of occlusal overload and an infectious episode due to poor hygiene has potentiated bone loss. Bone loss then takes on a cup-like shape, which is characteristic of peri-implantitis. When inflammation is absent, the bone can better resist occlusal overload.

Biomechanical disease

When occlusal trauma occurs, bone tissue adapts its response. If the trauma is mild, the bone trabeculae strengthen to respond to the mechanical stress. Crestal bone densification is visible on radiographs (Figs 2-36a, b). Infectious disease added to biomechanical stress potentiates bone loss (Fig 2-36c).

When occlusal trauma takes precedence, bone tissue reacts with bone resorption. It consists of moving the bone away from the areas of highest stress density (Fig 2-37a). On radiographs, the bone defect takes on a characteristic V shape, with a sharply accentuated slope (Fig 2-37b). Horizontal bone loss is poor compared to vertical bone loss (Figs 2-38a-c).

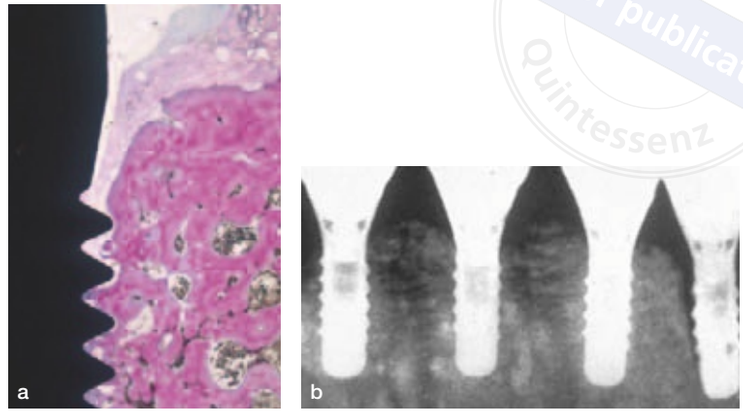


Figure 2-37 V-shaped bone defects of biomechanical origin observed in dogs.

a Histologic section after 6 months of loading of an immediately loaded implant. Note the V-shaped bone loss. On the radiograph, bone loss would have a V-shaped appearance (Szmukler-Moncler *et al.*, 2000b).

b Radiograph after 1 month of a loaded implant 2 days after placement. The V-shaped bone loss is very deep vertically. At the end of the 6 months of experimentation, this implant became mobile (Corso *et al.*, 1999).

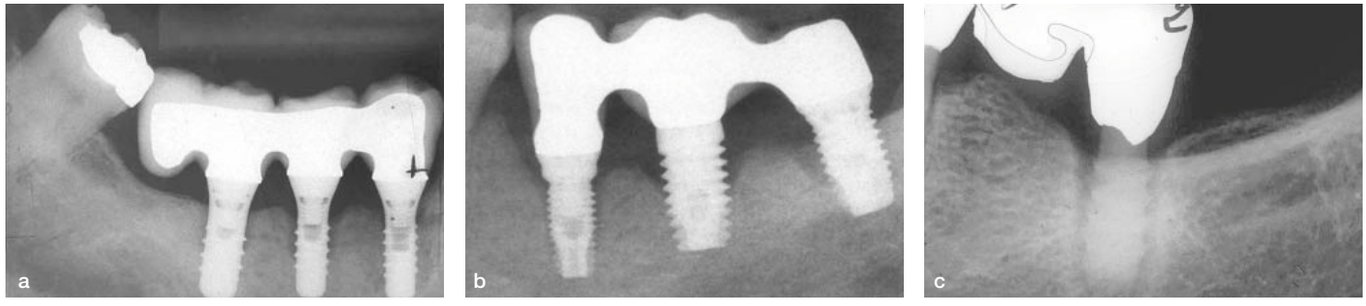


Figure 2-38 Bone defects of biomechanical origin, observed in the clinic.

a On implants with a rough plasma spraying technology surface (Dr Bischof). Note the V-shaped bone loss of the middle implant. The two adjacent implants also begin to show signs of bone loss. Occlusal equilibration is required.

b On machined surface implants. Only the most mesial implant has a V-shaped bone loss. The other two implants are cup-shaped, which is typical of peri-implantitis. Please note that the distal aspect of the most distal implant has no bone damage and is protected from inflammation. All shapes of bone loss are observed in this patient (Giovannoli & Renvert, 2012).

c On an aluminous ceramic implant. Bone loss is relevant around the implant and corresponds to a radiolucent border that indicates implant mobility. The cause of implant failure is of biomechanical origin (radiograph Professor J.H. Dubruille).

The peri-implant mucosa

Characteristics

The behavior of the peri-implant mucosa depends on the quality of the soft tissue (Berglundh *et al.*, 1991, 1994). At the end of the gingival healing period, the soft tissue–implant interface consists of three well-defined areas (Fig 2-39b):

- the sulcus;
- the epithelial attachment;
- connective tissue.

These structures are similar to those of the superficial periodontium, but not identical to it (Figs 2-39a, b).

Epithelial attachment

The sulcular epithelium is a nonkeratinized extension of the buccal epithelium (Fig 2-40). It is in continuity with the keratinized epithelium on its coronal portion. It consists of 5–15 cell layers and its structure is similar to that of the periodontal sulcular epithelium. The sulcular epithelium decreases in thickness as it becomes apical (Fig 2-40). The average probing depth in this sulcus is 2 mm; measurements can reach 3–4 mm without evidence of pathology (Figs 2-41a, b).

The junctional epithelium is in close contact with the implant surface. This biologic barrier plays a major role in the durability of the implant. Its height is about 1–2 mm. The most apical epithelial cells are located 1–1.5 mm coronally to the crestal bone.

Epithelial cells can adhere to inert biomaterials such as titanium and ceramics by means of hemidesmosomes and a basal lamina, just like the tooth surface. Gingival tearing may occur when unscrewing an abutment that was screwed immediately after implant placement. Close observation of the abutment shows the presence of tissue remnants that have remained adhered to the abutment. They show the biologic bonding that took place between epithelial cells and inert material.

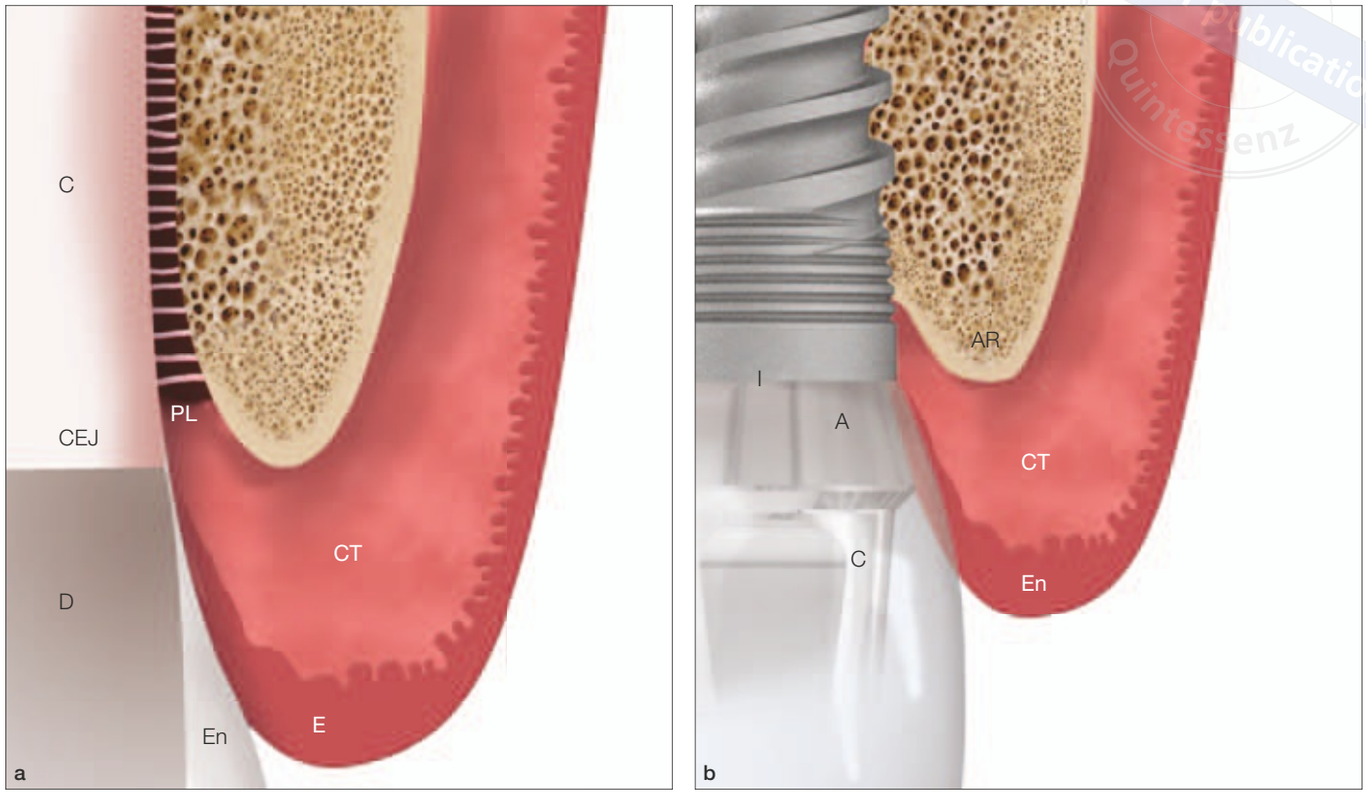


Figure 2-39 Organization of the mucoepithelial junction of a tooth and an implant. The structure of the gingiva is not very different. The significant difference is the existence of the periodontal ligament for the natural tooth and osseointegration for the implant.

a Natural tooth. C, cementum; PL, periodontal ligament; CEJ, cementoenamel junction; D, dentin; En, enamel; E, epithelium; CT, connective tissue. The total structure is about 3 mm.

b Implant. I, implant–prosthetic abutment junction; A, abutment; C, crown ; AR, alveolar ridge.

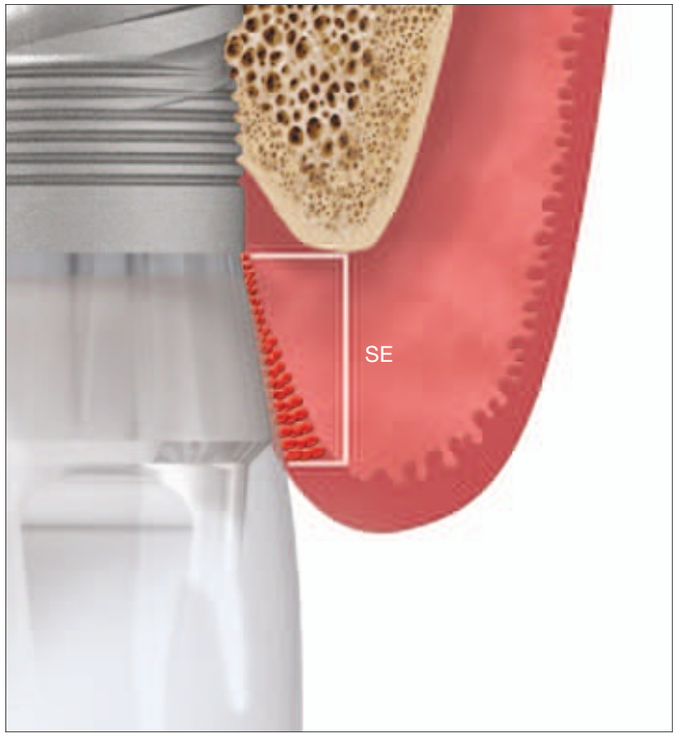


Fig 2-40 Sulcular epithelium.
The sulcular epithelium is the more or less extended junction between the implant neck and internal part of the sulcus.

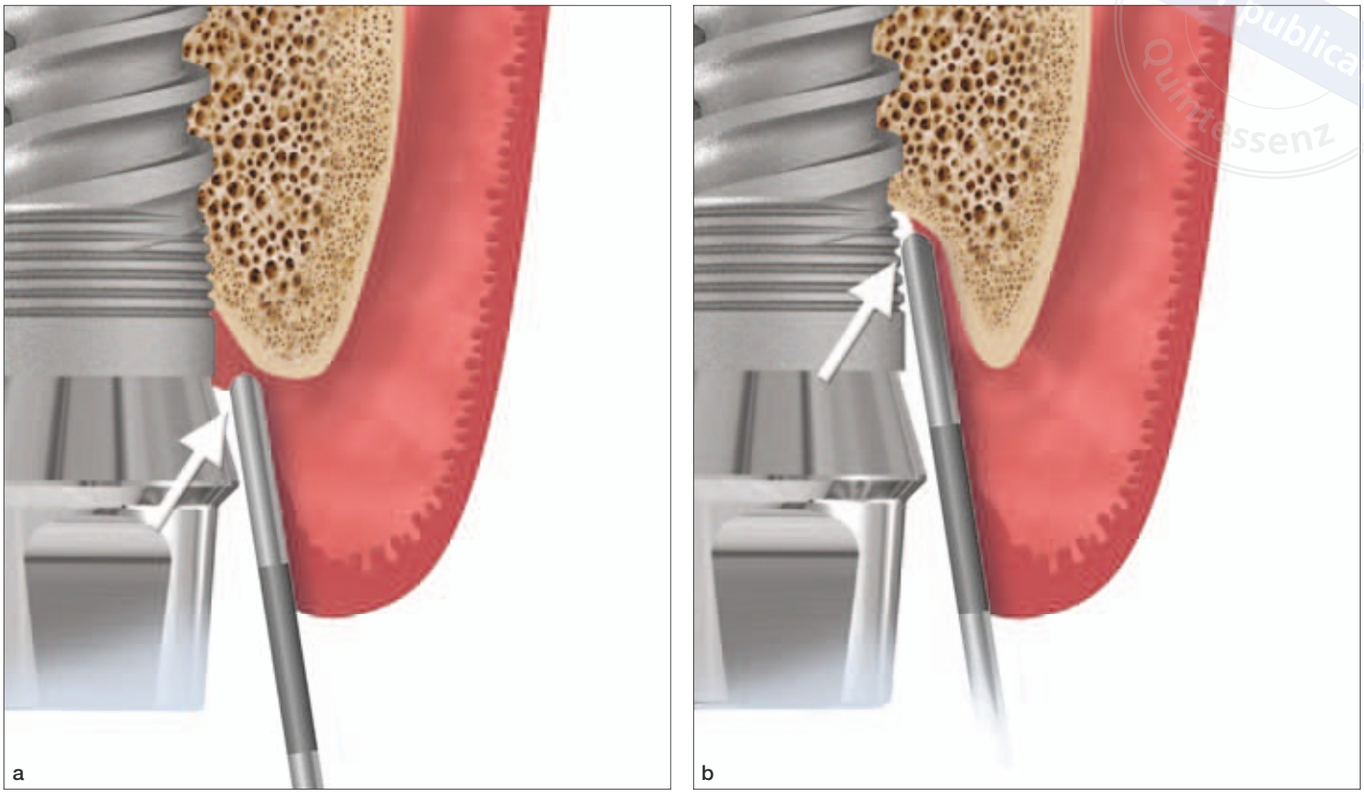


Figure 2-41 Probing depth in the implant sulcus.
a Implant with no pathology. The normal nonpathologic depth of a pocket is 2–4 mm.
b Implants with pathology. Pocket depth is greater than 4 mm.

Connective attachment

Some connective tissue is always interposed between the apex of the bone ridge and the apical portion of the epithelium (Fig 2-42). Regardless of whether the surface is machined, roughened, or covered with a bioactive material, collagen fibers are parallel to the implant axis (Fig 2-43); they do not insert perpendicularly to the implant surface (Comut *et al*., 2001) like collagen fibers in cement. They are perpendicular to the crestal bone and attach to the periosteum (Fig 2-42). In addition, circular fibers participate in junction sealing (Buser *et al*., 1992).

Connective tissue is organized in two layers:

- a dense, avascular inner layer of 50-100 µm, with circularly oriented fibers arranged in a collar shape around the implant;
- a richly vascularized outer layer with fibers parallel to the implant surface (Buser *et al*., 1992).

The gap left for the connective attachment is reduced during inflammation (Comut *et al*., 2001).

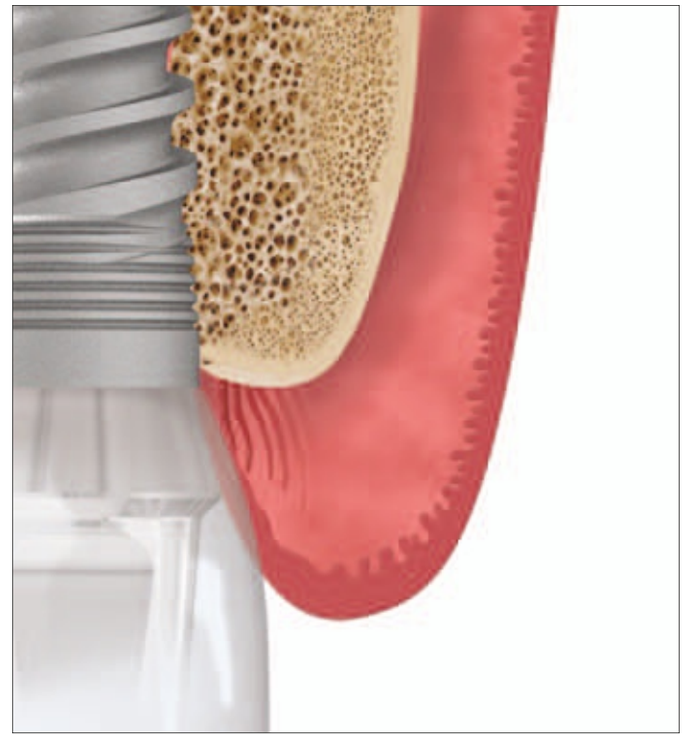


Fig 2-42 Parallel arrangement of the connective tissue fibers of the attachment with the implant.

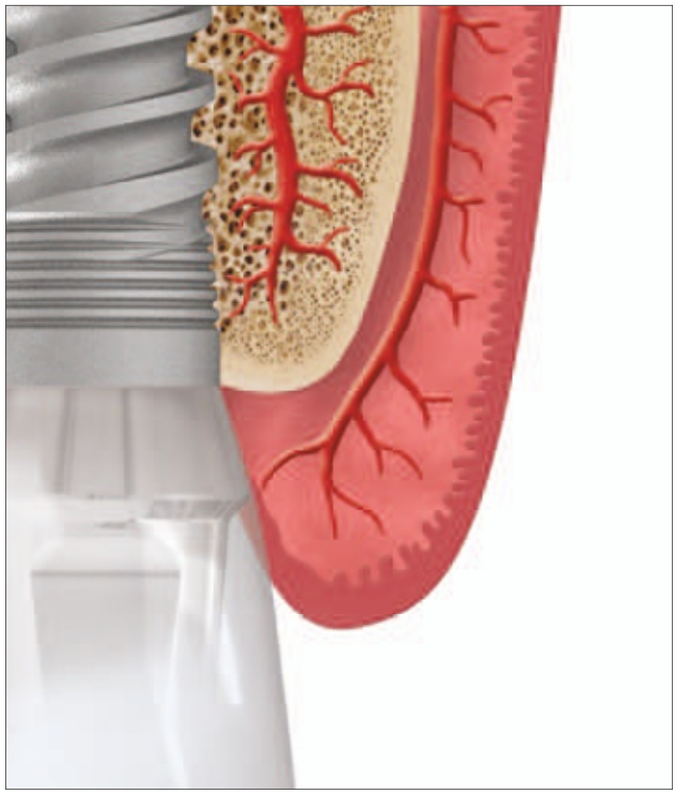


Fig 2-43 Vascularization of the peri-implant mucosa.
The rich vascularization of the periodontal ligament is absent.

Vascularization

Vascularization of the peri-implant mucosa is derived from periosteal vessels (Fig 2-42). Vascularization is less developed and less important than around a tooth because the latter benefits from the vascularization originating from the periodontal ligament. In particular, an avascular connective tissue aera exists and is adjacent to the implant.

Physiology of the peri-implant mucosa

The peri-implant mucosa acts as a barrier to the oral environment, which is septic (Weyant, 1994; Comut *et al.*, 2001). It presents specificities in relation to the gingiva. Tissue response to bacterial aggression is similar but not identical at the gingival and peri-implant mucosa levels. Dental plaque colonizes the implant surface and develops in the same way as on the tooth surface. It leads to migration of leukocytes to the junctional epithelium and formation of an inflammatory cell infiltrate.

The defense ability around an implant is less important. The number of fibroblasts is reduced, apical blood vessels at the epithelial junction are absent, and arrangement of the connective collagen fibers (in greater numbers) is parallel to the implant surface. The mucosa–implant interface, although vulnerable, prevents direct contamination of the implant by the oral environment.

However, indirect contamination can occur. Infiltration passes through the abutment screws (Fig 2-44a) but also through the attachment (Fig 2-44b). For this reason, a clinically healthy gingiva is the site of chronic low-level gingival inflammation (Ericsson *et al.*, 1996). It is particularly confined to the implant–abutment junction, centered on the junction (Fig 2-44b) (Ericsson *et al.*, 1996; Broggini *et al.*, 2003). Its presence could explain bone lysis in the form of a 1–1.5-mm craterization during the first months of loading (Hermann *et al.*, 2000, 2001).

In the healthy peri-implant sulcus, the pocket probing depth varies between 2 mm and 4 mm. Clinically, lower tissue resistance is observed at probing. It is caused by a lateral displacement of the peri-implant soft tissue. The thicker the peri-implant mucosa, the greater the depth of the pockets. Bleeding on probing does not always indicate a pathologic situation. It may be related more simply to the low resistance of the peri-implant mucosal attachment.

The presence of a keratinized mucosa is not essential for the maintenance of good peri-implant health (Chiu *et al.*, 2015). Opinions are divided and the level of evidence of relevance is low (Wennström & Derks, 2012). Some clinicians believe that bacterial mucositis and hyperplasia are aggravated in the presence of mobile peri-implant mucosa and lack of keratinized tissue (Chung *et al.*, 2006). Nevertheless, the tendency to gingival recession is all the more important as the gingiva is keratinized.

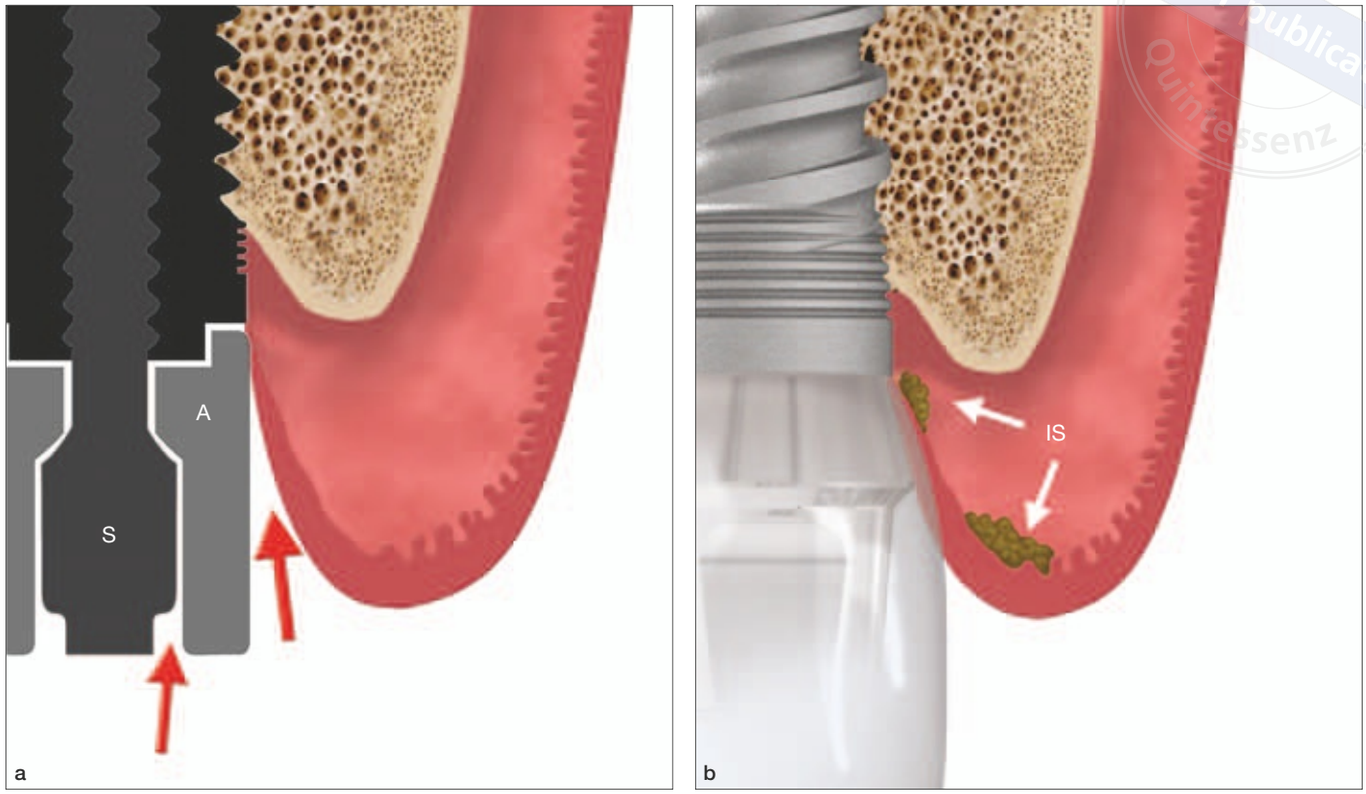


Fig 2-44 Bacterial infections at the mucoepithelial attachment.

a Bacterial infiltration pathways.

b Chronic inflammation of the attachment. Two inflammatory sites are maintained. One around the implant–abutment junction, the other at the upper level of the connective tissue.

However, it does not play a decisive role in increasing pocket depth. On the other hand, hygiene is easier to maintain in the presence of keratinized gingiva (Chiu *et al.*, 2015).

A 10-year comparative study (Roccuzzo *et al.*, 2016) recently observed that sites with a lack of keratinized gingiva were more prone to plaque accumulation and gingival recession. With a view to good long-term maintenance of gingival hygiene and health, there is therefore a trend toward strengthening the gingiva with soft tissue grafts (Chiu *et al.*, 2015; Roccuzzo *et al.*, 2016).

Physiologic response of the peri-implant mucosa to bone lysis

To maintain the esthetics of an implant-supported restoration, it is necessary to stabilize the peri-implant soft tissue, that is, the edge of the marginal gingiva and adjacent papillae. To best achieve this goal, it is necessary to be aware of the interactions between soft and hard tissues.

The vertical level of soft tissue depends on the presence of an underlying bone structure. Any gingival recession is preceded by apical migration of the underlying supporting bone.

The clinical literature reported the existence of a maximum distance between the bone ridge level and contact points between two adjacent crowns, between a tooth and an implant, and between two implants.

Figure 2-45a illustrates clinically performed measurements (Tarnow *et al.*, 1992). Between two teeth, when a distance of 5 mm is observed between the apex of the papillae and the bone ridge, papillae are present in 100% of cases. When this distance is 6 mm, papillae are present in 55% of cases. It is present in 25% of cases when the distance is 7 mm and in 10% of cases when the distance is 8 mm. By mathematically extrapolating the curve thus formed, the papillae should completely disappear when this distance is greater than 9 mm. Similar distances have been measured between a tooth and an implant.

On the other hand, these distances are much smaller between two implants. When the distance from the apex of the papillae to the apex of the interimplant bone ridge is greater than 5 mm, papillae are almost systematically absent (Tarnow *et al.*, 2000). It is present when this distance is 2–4 mm (Fig 2-45b). Between a tooth and an implant, the rules for the presence of papillae are those specific to the tooth. Indeed, the papillae between tooth and implant receive their vascularization from the periodontal ligament of the adjacent tooth.

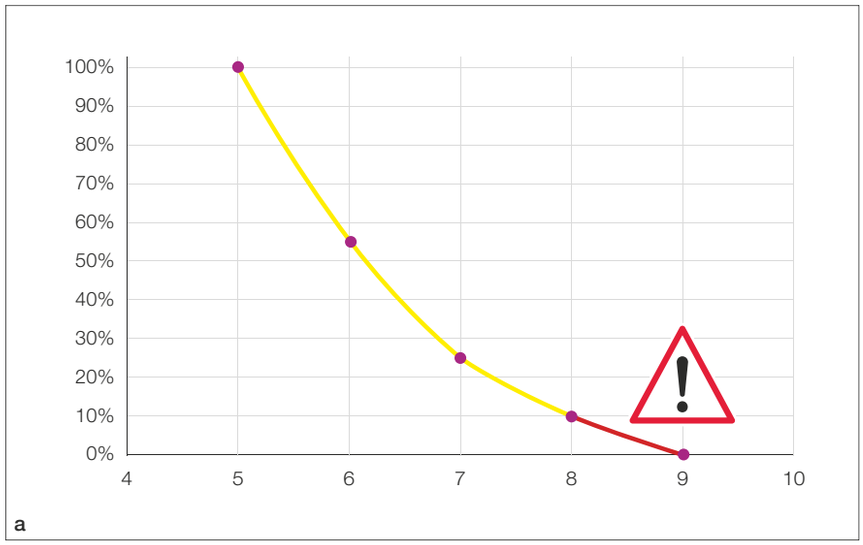


Fig 2-45 Relationship between the distance between the bone ridge and the presence of soft tissue.

a Relationship between the distance between the bone ridge and the contact point between two adjacent tooth crowns, corresponding to the apex of the papillae. Note that as the distance increases, the chances of having a papilla decrease.

b Distance between the apex of the papillae and the bone ridge between two implants. This maximum distance is smaller; it varies between 2 mm and 4 mm. Here it is 3 mm.

Therapeutic arsenal to minimize peri-implant bone loss

Recently, several parameters involved in alveolar ridge lysis have been identified. They allow the best strategy to be put in place to minimize bone loss as soon as implants are loaded.

Platform switching

Definition of platform switching

If the previously described bone loss could be avoided, esthetic management would be easier. In fact, in some otherwise unexpected circumstances, crestal bone loss does not seem to occur (Fig 2-46).

In 1991, the implant manufacturer 3i introduced large-diameter implants (Ø 5 mm and Ø 6 mm) without accompanying abutments with matching diameters. Thus, some restorations in the posterior zone of the oral cavity were based on Ø 3.75 mm implants as well as large-diameter implants, namely Ø 5 mm and Ø 6 mm (Fig 2-46). All these implants of different diameters were connected to abutments of the same diameter. Lazzara and Porter (2006) then observed that around standard implants, vertical bone loss reached the first thread, in accordance with classical radiologic observations (Fig 2-46). However, around large-diameter implants, bone height was unexpectedly maintained initially; the expected bone loss was absent (Fig 2-46). For a long time, this observation was neither interpreted nor exploited.

After learning how to control the prognosis of osseointegration, management of the esthetics has gained the full attention of the scientific and clinical community. Attempts were then made to avoid or minimize crestal bone loss, long considered physiologic and inevitable. Accidental maintenance of the crestal level initially attracted attention and was again analyzed. It was attributed to the removal of the abutment–implant junction from the implant neck (Lazzara & Porter, 2006). It is this offset between abutment and implant neck diameter that is referred to as platform switching (Figs 2-47a-d).

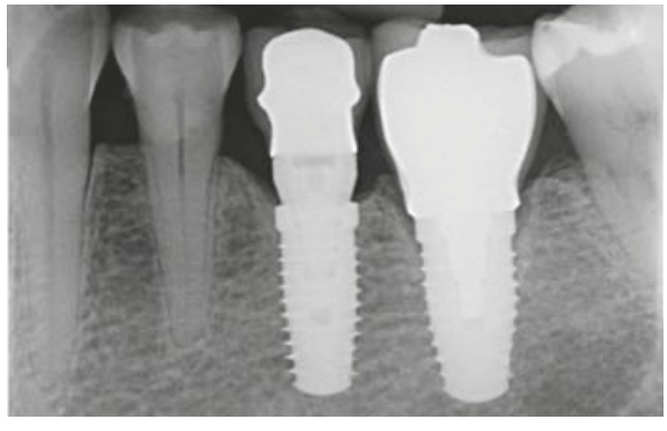


Fig 2-46 Periapical radiograph at 5 years.
Mesial implant (second premolar) with platform switching (implant neck 5 mm; prosthetic seat 4 mm) and physiologic emergence profile. Distal implant (first molar) without platform switching (6 mm) with convex soft tissue-compressing emergence profile: note the peri-implant bone loss.

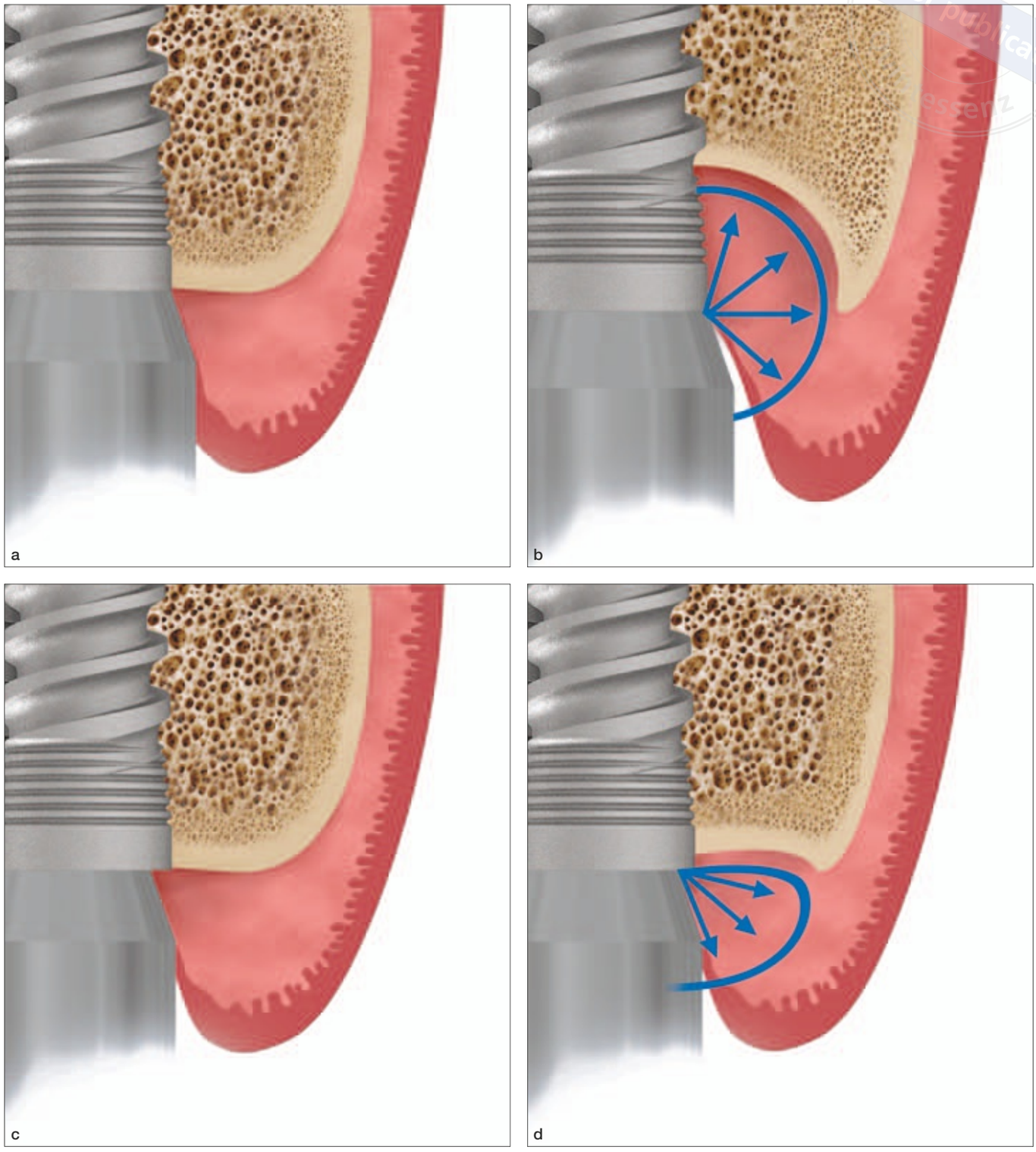


Fig 2-47 Principles of platform switching.

a Conventional abutment–implant junction.

b Possible diffusion reaction of chronic inflammation at the implant–abutment junction.
Diffusion would occur according to a diameter of 1–1.5 mm and would lead to vertical and horizontal resorption in the form of cratering.

c Recessed offset of the implant–abutment junction or application of platform switching.
The diameter of the abutment is smaller than the implant neck.

d Possible diffusion reaction of chronic inflammation when applying platform switching.
The diffusion path would be modified by the protruding edge of the implant neck. Vertical loss would be prevented and horizontal loss would be limited.

The observation by Lazzara & Porter (2006) was quickly relayed by other authors who confirmed its validity, as shown in Fig 2-48 (Serrano-Sánchez *et al.*, 2011; Siffert & Etienne, 2011). However, some animal studies could not systematically demonstrate a decisive advantage of platform switching compared to the classical butt joint connection (Becker *et al.*, 2009). Thus, it seems that:

- For implants with platform switching, vertical bone loss is low and restricted to the implant neck; horizontal loss is limited (Figs 2-46 and 2-49a, b).
- For implants without platform switching, horizontal and vertical bone loss is more pronounced. Vertical loss usually reaches the level of the first thread and horizontal loss extends more widely around the implants (Fig 2-49).

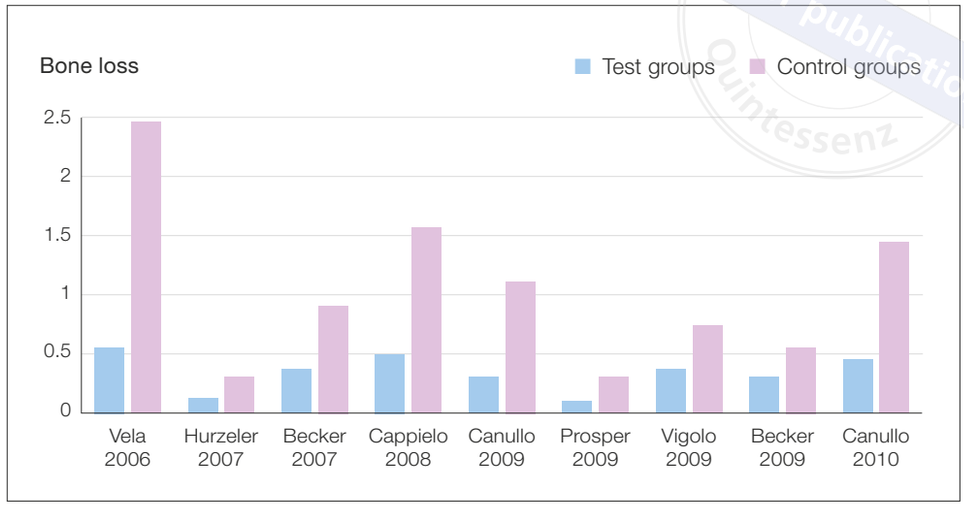
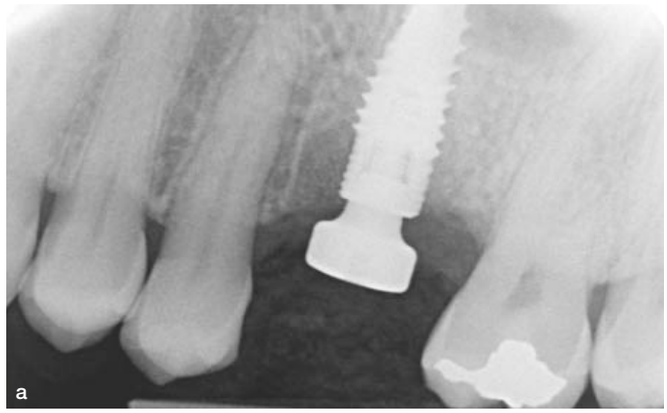


Fig 2-48 Studies showing the positive effect of platform switching. Bone loss in the test groups (blue) was consistently lower than in the respective control groups (red) (after Siffert & Etienne, 2011).

Interpretation of the platform-switching effect

It is not easy to understand this phenomenon nor formulate a link between platform switching and vertical and horizontal bone reactions because various theories are competing to explain the existence of crestal bone loss up to the first thread:

- Those who attribute vertical loss up to the first thread to the positioning of the biologic width would suggest that platform switching allows biologic width to be positioned along the implant–abutment recess. Projected on a vertical axis, the corresponding bone loss would be more limited and not reach the first thread.
- Those who attribute the vertical bone lysis observed on conventional implant–abutment junction implants to the presence of chronic inflammation (Fig 2-47b) would suggest that the chronic inflammatory flare-up is redirected in a horizontal or upward direction (Fig 2-47d). This change in trajectory would result in vertical preservation of the bone ridge and weak horizontal bone loss. Thus, the better-preserved bone capital would provide better support to the soft tissue at the cervical or papillary zone.
- Those who attribute apical bone loss to the presence of micromovements at the implant–abutment junction would suggest that when platform switching is applied, micromovements are no longer exerted in the immediate vicinity of the bone. Therefore, the bone would not need to retract away from the mechanical disturbance but could remain in the immediate vicinity of the implant neck.
- Still others suggest a change in the distribution of crestal-level stresses and their intensities (Canullo *et al.*, 2011a).



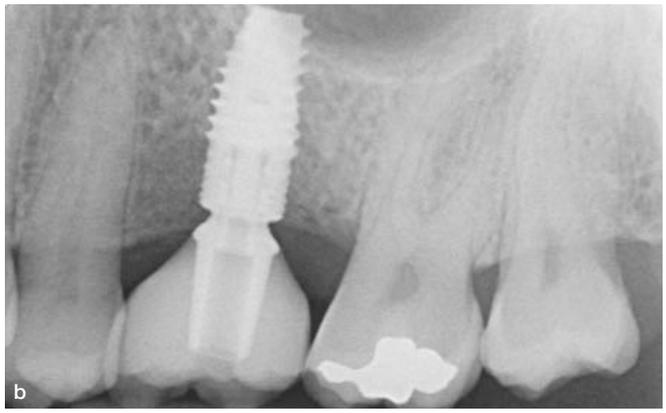


Fig 2-49 Platform switching to avoid vertical bone loss reaching the first thread.
a Postoperative radiograph showing the healing abutment respecting the platform switching principle.
b Radiograph of the final restoration. Application of platform switching prevented vertical bone loss up to the first thread.

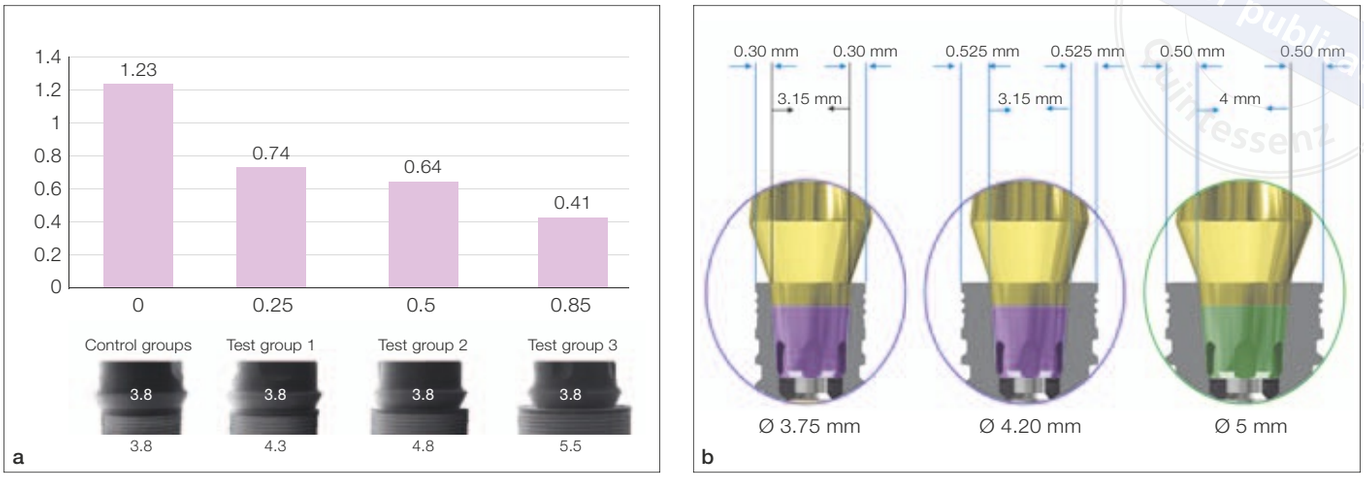


Fig 2-50 Extent of platform switching and crestal bone loss.

a Comparison of the level of crestal bone loss at 9 months. Four groups are compared: a control group without stall and 3 test groups with increasing stall amplitude, 0.25 mm, 0.50 mm, and 0.85 mm, respectively. Corresponding crestal losses decrease by 1.23 ± 0.67 mm, 0.74 ± 0.39 mm, 0.64 ± 0.40 mm, and 0.41 ± 0.28 mm, respectively (Canullo *et al.*, 2010a).

b Implant system where the extent of platform switching varies according to the implant diameter (C1, MIS).

In an attempt to answer this question, the extent of the stall between the implant neck and prosthetic abutment was modulated in a specific clinical study (Fig 2-50a) (Canullo *et al.*, 2010a) as it responds to a clinical reality in some implant systems (Fig 2-50b). The authors were able to observe a correlation between the decrease in crestal bone loss and the extent of platform switching (Fig 2-50a). To verify whether these differences in bone responses could be attributed to varying degrees of local inflammation at the implant–abutment junction, gingival samples were taken from the same patients. No differences were found in the extent of connective tissue inflammation, microvascular density, or collagen content of the gingiva at the implant–abutment junction (Canullo *et al.*, 2011b). Similarly, study of germs present could not reveal any statistically significant differences between control and test groups or between the various test groups themselves (Canullo *et al.*, 2010).

Preliminary work in the posterior area carried out by our team showed that platform switching is effective in 44% of the sites (each implant has 2 sites, 1 mesial and 1 distal). That is to say that platform switching allowed to maintain the crestal level close to its initial level in about one case out of two instead of seeing it usually reach the level of the first thread. This non-systematic character of maintaining the bone level shows, on the one hand, that progress has been made but, on the other hand, that all the parameters for understanding the phenomenon have not been sufficiently defined. It would seem that we are moving toward a more accurate and specific understanding of crestal bone loss. It should take more account of the patient's own bone quantity and quality (Petrie & Williams, 2007; Canullo *et al.*, 2011c) and its immune capabilities (Canullo *et al.*, 2011c).

Clinical relevance of platform switching

From the previous section, it would seem that a 'simple' offset of the abutment from the implant neck may prevent apical and horizontal bone loss from occurring, observed under usual conditions. Whatever the reasons, application of the platform switching principle with its low, even random, bone loss could offer new clinical perspectives (Davarpanah *et al.*, 2007; Vela-Nebot *et al.*, 2011):

- It would allow the minimum distances between tooth and implant and between implants to be reduced (Figs 2-51a-d, 2-52a, b) (Rodriguez-Cuirana *et al.*, 2009; Vela-Nebot *et al.*, 2011; Vela *et al.*, 2012).
- The rules for implant positioning could be simplified (Davarpanah *et al.*, 2007; Vela-Nebot *et al.*, 2011).
- Management of esthetics would be simplified.
- More implants could be placed in a given space to meet biomechanical criteria without compromising esthetic requirements, for example, in the cases of immediate loading in the maxilla.
- It could even influence the treatment strategy for the anterior area when the incisive zone of the maxilla is edentulous. In this indication, it is customary to avoid placing two implants in the central incisor sites as this involves delicate and unpredictable soft tissue management. The alternative strategy is to place two implants at the lateral incisor sites and pontics at the central incisor sites or alternate implants and pontics. With a newly recorded minimal bone loss between two implants, it would be possible to consider placing two adjacent implants at the central incisor sites (Fig 2-52) without compromising soft tissue prognosis (Vela-Nebot *et al.*, 2011).

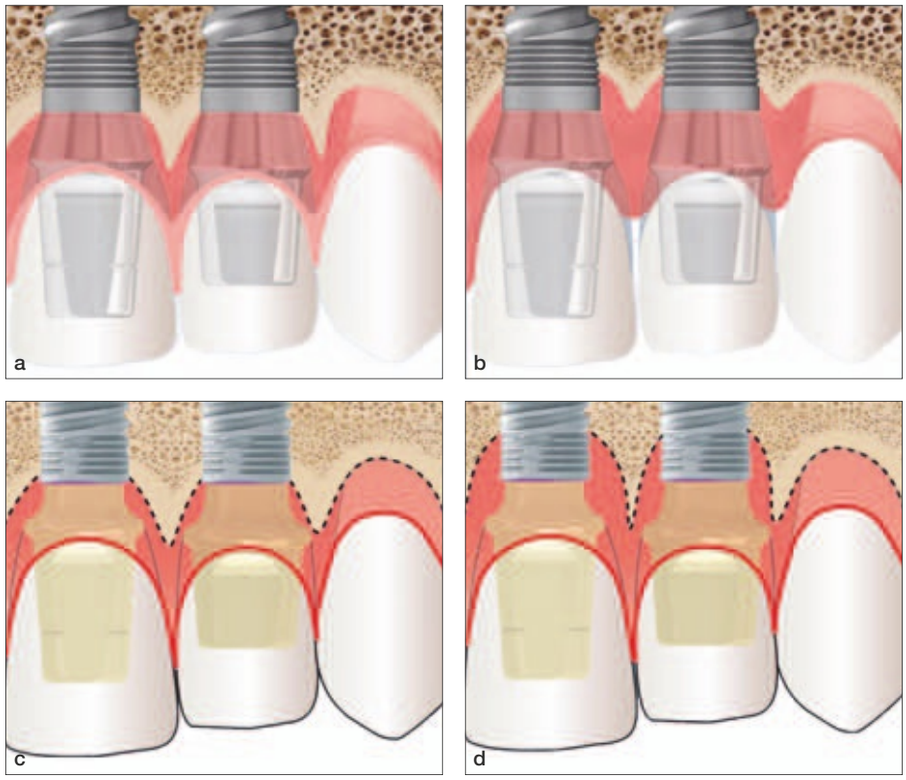


Fig 2-51 Possible simplifications brought about by the application of platform switching to the implant positioning rules.

a Interimplant distance < 3 mm without platform switching, postoperatively. The bone level is located at the level of the implant neck.

b Interimplant distance < 3 mm without platform switching, after loading. Note the bone loss of the interimplant ridge because the interimplant distance is < 3 mm. The papilla is no longer supported; recession takes place at this level.

c Interimplant distance < 3 mm with platform switching, postoperatively. The bone level is located at the level of the implant neck.

d Interimplant distance < 3 mm with platform switching, after loading. Bone resorption is limited. Despite an implant distance of < 3 mm, the interimplant ridge was not lost. It can then support the papilla and avoid gingival recession at this level.

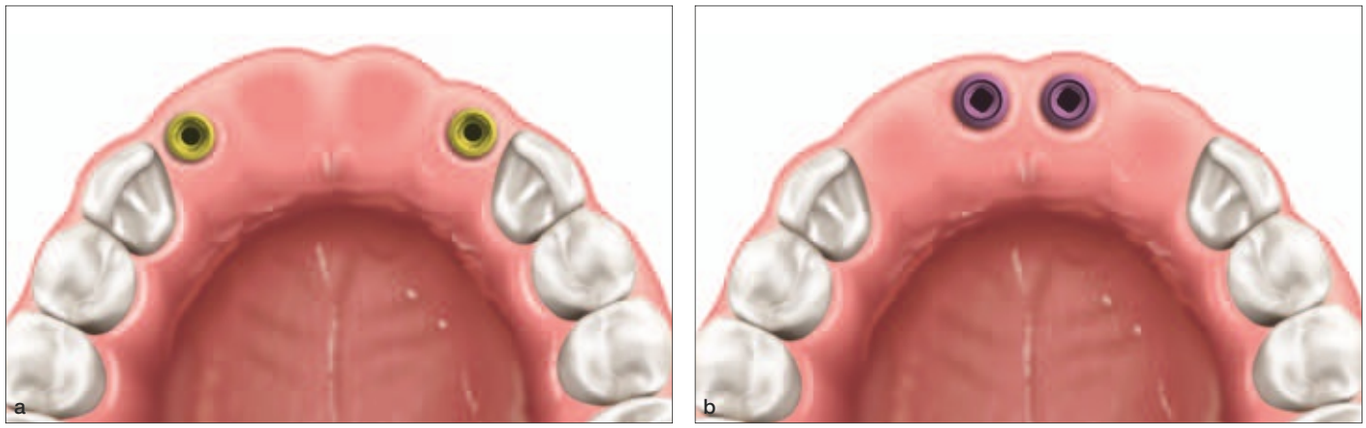


Fig 2-52 Possible impact of platform switching on processing strategy in the edentulous anterior region.

a Conventional strategy of treatment without platform switching or if platform switching does not reduce peri-implant crestal bone resorption. The implants are not adjacent with regard to the central incisors.

b Alternative strategy thanks to platform switching, which allows reducing peri-implant crestal bone resorption. It is possible to place two adjacent implants in the position of the central incisors.

Thus, the literature does not offer a clear answer to our question about platform switching. Nevertheless, two main ideas emerge: the first is that platform switching as such should not be seen as a panacea in the fight against bone loss. The second is that many other parameters seem to influence bone loss.

It seems that we have a multifactorial phenomenon; the importance of each parameter has not yet been identified.

We will now consider other factors that may be involved in the control of crestal bone resorption.

Implant site biotype

It has long been known that biologic seal reconstitution during healing of the peri-implant gingiva may cause vertical bone loss in the apical direction in relation to gingival thickness (Ericsson *et al.*, 1996). This introduces the presumption of a relationship between gingival biotype and peri-implant crestal bone loss. A Lithuanian team embarked on the further development of this hypothesis.

Linkevicius *et al.* (2009) began by comparing bone remodeling according to gingival biotype for implants without platform switching. Bone loss was more pronounced for implants with a thin biotype (≤ 2 mm) compared to a thick biotype (> 2.5 mm), 1.61 mm versus 0.26 mm (Fig 2-53a). This result was all the more unexpected since implants were placed 2 mm supracrestally to shift the abutment–implant junction beyond the alveolar ridge.

The second part of their work was to study the effect of thin-to-thick biotype modification on peri-implant bone response when the connection was conventional (Linkevicius *et al.*, 2013). Crestal bone loss was compared in three groups of patients. The mean thickness of the first group with a thin biotype was 1.51 mm. The second group had their originally thin gingiva augmented with an allogeneic membrane, with a thickness of 3.83 mm. The last group was characterized by a thick biotype of 2.98 mm. At the 1-year recall, the most pronounced bone loss, 1.65 mm, occurred in the implants with the thin biotype. Bone loss in the augmented biotype group was much less (0.31 mm), slightly less than in the originally thick biotype group (o.44 mm) (Fig 2-53b). It was concluded that the peri-implant tissue biotype could play a much more prominent role than expected for implants with a conventional connection.

At this stage, it was necessary to check whether platform switching could affect the decisive influence of the biotype observed previously on conventional connection. The solution was to compare the different abutment–implant connections in sites with a thin gingival biotype. Less crestal bone loss around implants with platform switching would be conclusive. This study was undertaken in a pilot clinical trial of six implants (Linkevicius *et al.*, 2010). In sites with a thin biotype <2 mm (1.79 mm), implants of 2 distinct systems with different diameters (Ø 3.5 mm and 4.0 mm for Prodigy and BioHorizons and Ø 4.1 mm for Prevail and Biomet *3i*) were implanted. In both cases, crestal lysis was pronounced, 1.88 mm and 1.76 mm, respectively, and platform switching could not be observed (Fig 2-53c). This result, although supported by a reduced number of implants, seems to suggest that a fundamental parameter has been omitted in many clinical studies. This would help to understand why some studies report platform switching while others report its absence.

To further support this finding, the same authors compared 40 sites with thin and thick biotypes using a platform switching implant (Linkevicius *et al.*, 2014). At the 1-year follow-up, they found that platform switching did not prevent the bone lysis usually observed (Fig 2-53d). At sites with a thin biotype, it was 1.17 mm versus 0.21 mm for sites with a thick biotype. At the end of this study, it was possible to consider the biotype parameter as more powerful in contributing to crestal bone loss than platform switching in opposing it.

< 2 mm vs > 2 mm	Bone loss (mm)
Thin biotype 1.95 ± 0.30	1.45 ± 0.55
Thick biotype 3.34 ± 0.76	0.17 ± 0.19

a

< 2 mm vs > 2 mm	Bone loss (mm)	
	Mesial	Distal
Thin biotype 1.51 ± 0.09	1.65 ± 0.08	1.81 ± 0.06
Thick biotype 2.98 ± 0.08	0.44 ± 0.06	0.47 ± 0.07
Thicken biotype 3.83 ± 0.13	0.31 ± 0.05	0.34 ± 0.05

b

1.5-2 mm, 1.79 ± 0.26	Bone loss (mm)	
	Mesial	Distal
Platform-shift	1.81 ± 0.39	1.70 ± 0.35
Standard	1.60 ± 0.46	0.76 ± 0.45

c

PS	Bone loss (mm)
Thin biotype 1.53 ± 0.07 1-2 mm	1.18 2.1-0.1 mm
Thick biotype 2.98 ± 0.53 2.5-4 mm	0.22 1.1-0.0 mm

d

PS	Bone loss (mm)	
	Mesial	Distal
Thin biotype < 2 mm	1.22 ± 0.08	1.14 ± 0.07
Thick biotype > 2 mm	0.22 ± 0.06	0.20 ± 0.06
Thick biotypen	0.24 ± 0.06	0.19 ± 0.06

e

Fig 2-53 Influence of biotype on bone loss

a Biotype effect with a conventional connection. Bone loss is greater for implants placed in a site with a thin biotype (Linkevicius *et al.*, 2009).

b Biotype augmentation effect with a conventional connection. Both thick and thickened biotypes show very little peri-implant crestal loss (Linkevicius *et al.*, 2015).

c Platform-switching effect in the presence of a thin biotype. The presence of platform switching does not reduce crestal bone loss when the biotype is thick (Linkevicius *et al.*, 2010).

d Biotype effect in the presence of platform switching. The presence of platform switching does not override the biotype parameter. Crestal bone loss is observed around implants placed in a site with a thin biotype (Linkevicius *et al.*, 2014).

e Biotype augmentation in the presence of platform switching. The biotype parameter is decisive in relation to the platform-switching parameter. Indeed, only the thin biotype suffers significant crestal bone loss (Puisys & Linkevicius, 2015).

To definitively establish the weakness of platform switching, this same group (Puisys *et al.*, 2015) replicated on platform switching implants the work they had previously carried out on implants with a conventional connection, which had highlighted the influence of the 'biotype modification' parameter on bone loss. Sites with an originally thin biotype augmented with an allogeneic membrane were compared (test group 1) to sites that were not augmented (test group 2). Sites with thick biotypes served as negative controls (group C). Bone loss was more pronounced in group T1 (1.22 mm), while it was similar in group T2 with the modified thin biotype (0.24 mm) and Group C with the initially thick biotype (0.22 mm) (Fig 2-53e).

The work of this team allows us to conclude that platform switching as such definitely does not prevent bone loss. Lack of consideration of this important parameter could explain the reason for the discrepancies between studies, but also why we found that, far from being systematic, platform switching only worked for 44% of the analyzed implant 'sides' when the mesial and distal aspects of each implant were counted separately (Szmukler-Moncler *et al.*, 2012).

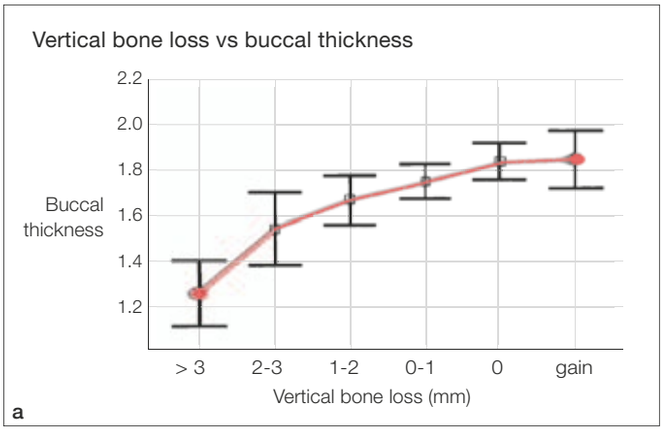
Similarly, it may lead to questionable interpretations as may have been the case in the study by Wang *et al.* (2015), who compared bone loss of posterior implants with and without platform switching. The gingival biotype parameter was not recorded. At the 1-year follow-up, bone loss for both groups was particularly low, 0.04 mm for the platform-switching group and 0.19 mm for the non-platform-switching group. The difference between groups was statistically significant but clinically irrelevant. The authors attributed this unexpected finding to the geometry of the internal connection and its hermeticity to bacterial colonization, whereas the biotype could have played a decisive role for these posterior implants.

Thickness of the bone lamellae

The notion of optimal 3D placement is well established (Saadoun *et al.*, 1999; Grunder, 2005). It implies that a bone thickness > 1.5 mm must remain between the edge of the implant and the edge of the residual buccal plate, otherwise resorption of the buccal plate may occur. However, the influence of the variable 'buccal plate thickness' on implant success rate or crestal bone loss has been little studied (Spray *et al.*, 2000; Bischof *et al.*, 2006; Dam *et al.*, 2014).

Some clinical work on transgingival implants has shown the absence of a relationship between bone loss and buccal plate thickness (> 1 mm versus ≤ 1 mm) (Bischof *et al.*, 2006; Dam *et al.*, 2012). In contrast, for 2-stage surgical implants, the sites without bone loss were those with an average thickness of 1.8 mm. Sites with bone loss > 3 mm were precisely those with the weakest thickness, 1.3 mm (Spray *et al.*, 2000) (Fig 2-54a).

Only one group studied the relationship between platform switching and buccal table thickness. Unfortunately, this parameter was entered in their database as a discontinuous qualitative variable (abundant bone volume) and not as a continuous quantitative variable with measurements taken at each site (Glibert *et al.*, 2016). Nevertheless, the authors noted that platform switching was less important in the preservation of the bone ridge (0.63 mm versus 1.02 mm) than the residual width of the bone plate (0.45 mm versus 1.20 mm) for all implants combined (Fig 2-54b). Further studies are needed to refine this question. It should be noted that a correlation between biotype and thickness of the buccal plate of the incisor-canine maxillary area was demonstrated in healthy dentate patient. Where an average buccal plate thickness was 0.34 mm for patients with a thin biotype, it was 0.75 mm when the biotype was intermediate or thick (Cook *et al.*, 2011), more than double the thickness.



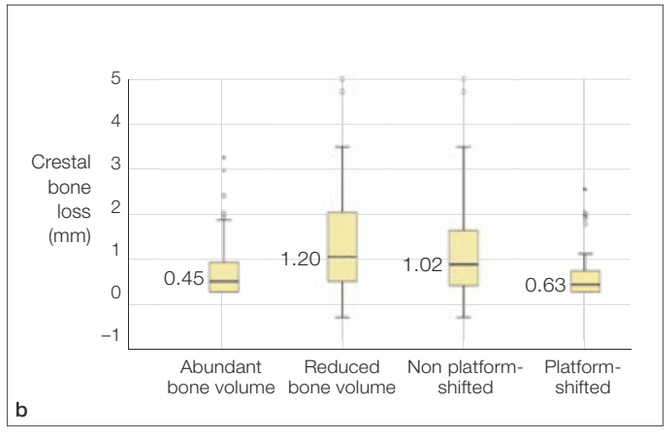
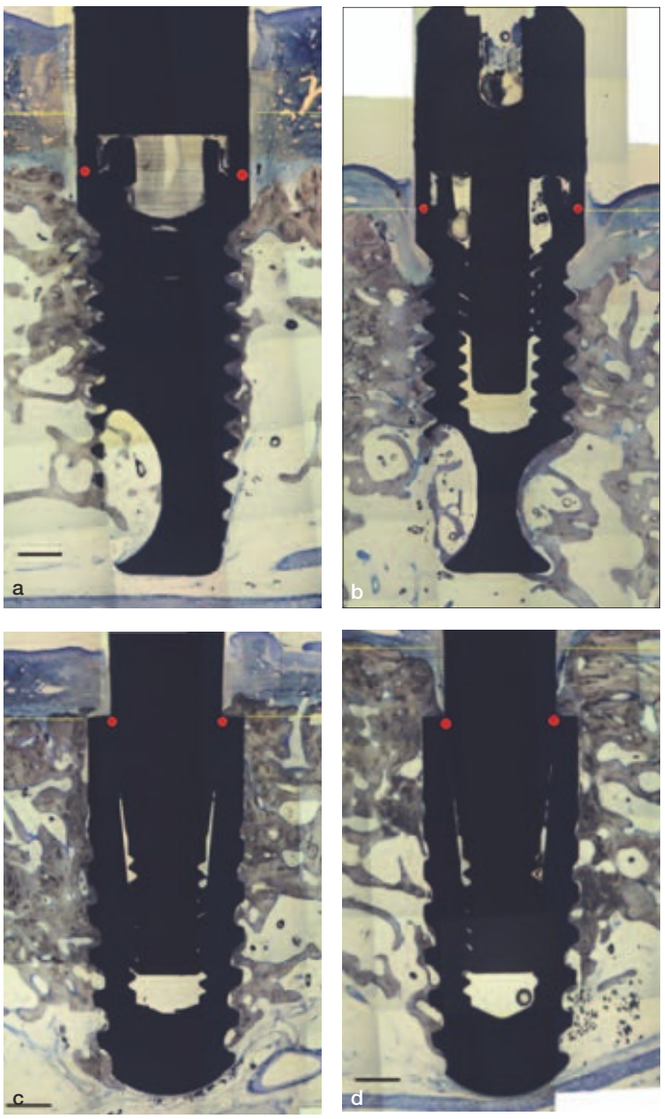
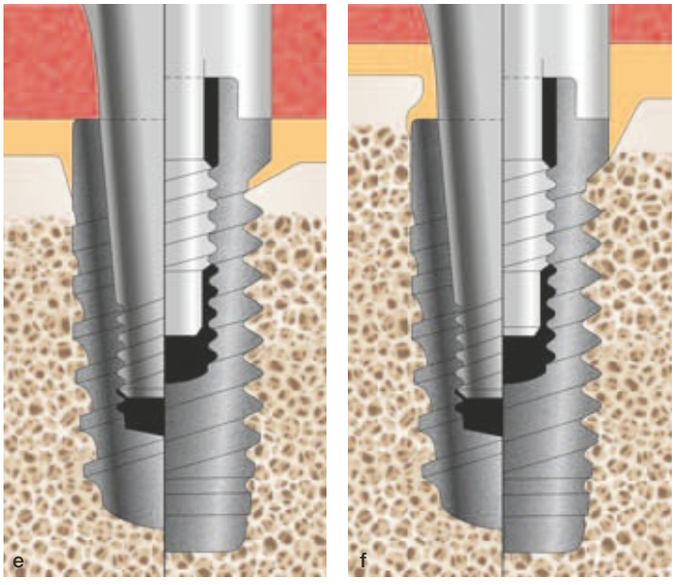


Fig 2-54 Influence of the thickness of the buccal plate on crestal bone loss.

a Retrospective highlighting of vertical bone loss depending on the initial thickness of the buccal plate (Spray *et al.*, 2000).

b Relationship between crestal bone loss and available bone volume and in the presence of platform switching. Abundant (class A, B, C) or limited (class D) bone volume is shown. Bone loss is more affected by bone volume than by the presence of platform switching (Glibert *et al.*, 2013).





Crestal, supracrestal, or subcrestal position

It is generally acknowledged that the position of the implant neck in relation to the alveolar ridge plays a decisive role in the extent of bone loss. Several animal studies showed that a more subcrestal position leads to a more extensive bone loss (Hermann *et al.*, 2000; Weng *et al.*, 2008).

The relevant question then is whether this rule also applies to implants with platform switching. Clinical studies have invalidated this principle, showing that the subcrestal situation does not induce specific crestal bone loss. For platform-switching implants, it is neutral (Romanos *et al.*, 2014) or even has a positive impact (Veis *et al.*, 2010). Based on animal studies, some authors (Weng *et al.*, 2008) have proposed a model of bone loss dependent on neck position beside the ridge and whether or not platform switching is integrated (Figs 2-55a-f).

Digital simulations suggest that the subcrestal position induces less stress in the bone than a crestal position (Zanardi *et al.*, 2015). This may explain why some authors experimentally found less crestal bone loss when the subcrestal position is chosen (Calvo-Guirado *et al.*, 2014, 2015).

Fig 2-55 Effect of the crestal or subcrestal position on bone loss depending on the presence of platform switching.

a Bone loss when the crestal position is chosen for an implant without platform switching. The red dots indicate the level of the implant neck.

b More extensive bone loss when the subcrestal position is chosen for an implant without platform switching.

c Bone loss when the subcrestal position is chosen for an implant with platform switching.

d Absence of bone loss when the subcrestal position is chosen for an implant with platform switching.

e Simulation of bone loss for an implant in crestal position with and without platform switching. Crestal bone loss occurs in both configurations but is less important for the implant with platform switching.

f Simulation of bone loss for an implant in subcrestal position with and without platform switching. Crestal bone loss is only relevant for the implant without platform switching (Weng *et al.*, 2008).

Implant neck design

Neck structure

Over the past 30 years, changing the implant neck has been undertaken in many directions. The originally straight and smooth implant neck has undergone many changes (Fig 2-56). The neck, the most coronal part of the implant, which corresponds to the emergence of the implant at the ridge, has been the focus of much attention. The aim of optimizing neck design was to retain the peri-implant ridge level closest to its initial level. It was enlarged in relation to the implant body, either slightly or markedly. Threads were added, either mini-threads or threads rising from the implant body to the top of the neck. The surface of the implant neck was also variable, either machined or rough.

Many studies have been carried out on this subject (Al-Thobity *et al.*, 2017). It has long been known that a smooth surface does not retain bone tissue (Wiskott *et al.*, 1999; Gotfredsen *et al.*, 2001). Bone migration in the apical direction occurs until it encounters threads or a rough surface (Hermann *et al.*, 1997).

Neck design meets two contradictory requirements. On the one hand, the crestal bone loss that occurs after implant placement must be minimized. The neck will then have rough microthreads or threads reaching the top of the implant. On the other hand, from a long-term perspective, it is better to have a smooth surface that is easier to clean in case of peri-implantitis. The neck will then be machined to a height of 1 mm to 2 mm depending on the system.

Clinical studies showed that neck design that better maintains the bone level of the alveolar ridge is that of a neck with a micro-thread with a rough surface. A rough neck with no particular geometry maintains it less well but better than a neck with a machined surface (Abrahamson *et al.*, 2006; Shin *et al.*, 2006; Bratu *et al.*, 2009).

Neck geometry

Since the introduction of modern implantology by Brånemark *et al.* (1977), implant design has changed significantly. Starting with a standard 'off-the-shelf' implant of different lengths, successive modifications were made to meet the requirements of new specific indications. Implants were then adapted to the changing views that were to permeate implantology.

In terms of diameter, larger implants were introduced to the market. They were larger than Ø 3.75–4.0 mm, reaching Ø 6 mm, or even Ø 7 mm. This was followed by implants with smaller diameters of Ø 3.5 mm, Ø 3.25 mm, and Ø 3.0 mm. The shape of the apex also changed from flat to round, sharp or not; the early apical foramen also disappeared. Implant bodies also underwent evolution, from strictly cylindrical to cylindrical-conical and conical, with or without symmetrical or asymmetrical threads, with the most varied designs.

Fig 2-56 Evolution of implant design where each implant incorporates several new features.

At the same time, another change concerning a similar issue was taking place. In the area of maxillary central incisors, when a post-extraction site had to be rehabilitated, placing large implants was recommended to best fill the freshly obtained socket (Martinez *et al.*, 1999). The aim of this approach was to preserve peri-implant bone volume and limit resorption of the buccal place supporting the marginal gingiva. Studies began to show that this type of recommendation did not achieve the goal (Grunder *et al.*, 2005). It was suggested that the best way to maintain the buccal plate was to maintain at least 2 mm of cortical bone between the edge of the implant and the buccal plate (Saadoun *et al.*, 1999; Grunder 2005) or even 4 mm (Capelli *et al.*, 2013). For this purpose, it was more appropriate to place an implant with a regular diameter slightly angled in the palatal direction instead of an implant with a wide neck and graft the gap left between implant and buccal plate (Davarpanah *et al.*, 2012).

To reduce peri-implant bone loss, this awareness led manufacturers to design implants with a particular geometry at the crestal emergence of the implant. The aim was to increase the thickness of the buccal bone lamella facing the implant as it emerges beyond the bone crest. This resulted in a reverse conical neck (Figs 2-57a, b).

In fact, the combination of these two properties, platform switching and reverse conical neck, effectively reduced bone loss below the first thread of the implant body without making it completely disappear (Figs 2-57c, d).

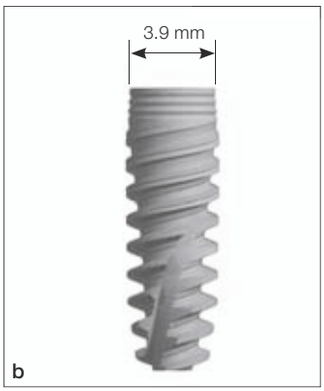
Evolution of implant neck design

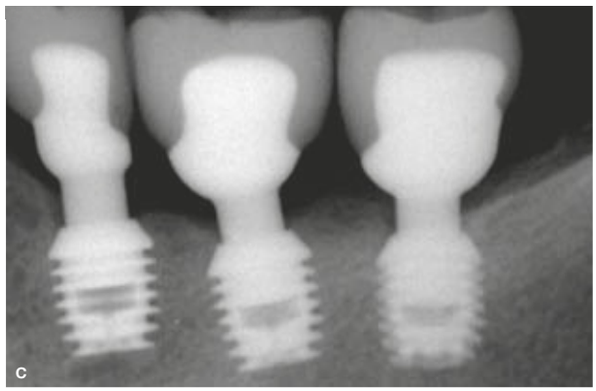
Maintaining the widest possible buccal bone plate at the level of the crestal emergence is a determining issue. The creativity of implant manufacturers has recently opened up a new avenue in maintaining a thicker buccal bone plate in relation to the implant.

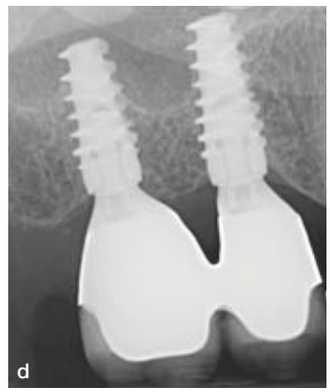
Experienced clinicians and micromechanical engineers devised an implant with a triangular neck (V3, MIS Implants Technologies) with three flats on the circumference of the implant neck. When the flat is placed in front of the buccal plate, the geometry of the implant neck allows the thickness of the buccal bone lamella to be increased (Fig 2-58a). The introduction of this particular design was possible without deviating from the

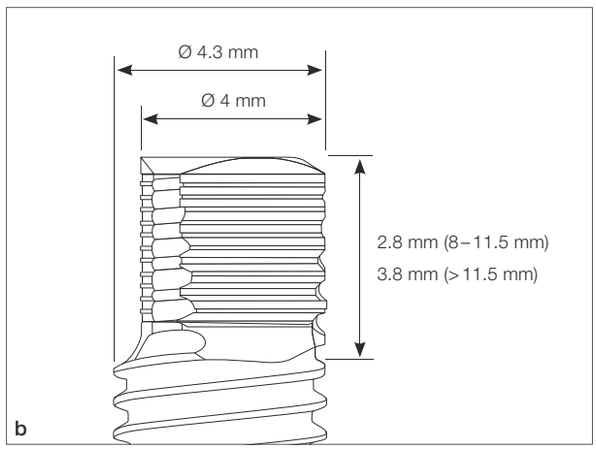
biomechanical requirements because the implant material is a specific titanium alloy (grade 23 titanium) with superior biomechanical properties than titanium CP. Depending on the diameter of the implant, this flattening provides an additional bone table space of 0.1–0.5 mm compared to a cylindrical design (Figs 2-58b, c). Most importantly, this horizontal bone gain extends in the apical direction over a distance of 2.8–3.8 mm depending on the length of the implant (Figs 2-58b, d).

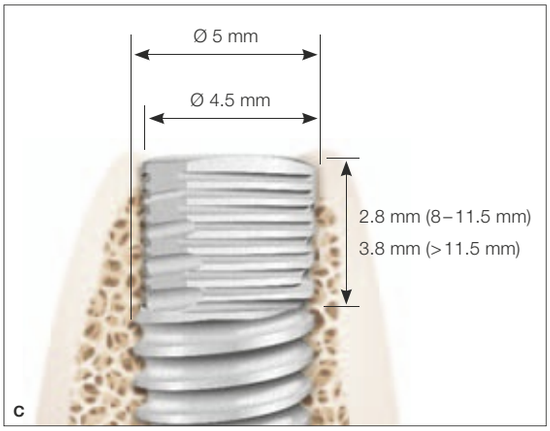
Fig 2-57 Implant with a neck diameter smaller than its body.
a, b Implant with a neck diameter smaller than its body diameter. (Courtesy of Morgan VJ. The Bicon Short Implant. Quintessence Publishing; 2018).
c, d Radiographs showing crestal level conservation.











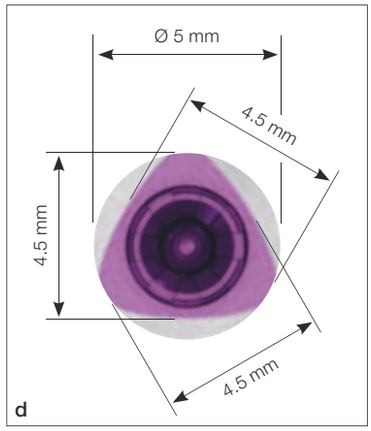


Fig 2-58 Implant with cervical flats to be positioned facing the buccal plate.
a Diagram of the V3 implant with three flat spots on the implant neck.
b Flat releasing a 0.3 mm × 2.8 mm gap compared to a Ø 4.3 mm conventional implant.
c Top view of the neck flat compared to a conventional implant. In the case of a Ø 5-mm implant, the flat spot leaves a 0.5-mm gap between the edge of the implant and the buccal plate.
d Gain in bone volume of the buccal plate created by this flat extending vertically from 2.8 mm to 3.8 mm in height depending on the length of the implant (Courtesy of MIS).

This increases the overall buccal bone volume available between the edge of the implant and the edge of the buccal plate. This should provide increased support for the marginal gingiva and a more generous blood supply.

Clinically, in areas where the alveolar ridge is thin (Fig 2-59a), placement of a conventional implant with a Ø 5 mm would leave a cortical plate thickness of only 0.5 mm, whereas an implant of equivalent diameter with a flattened surface leaves a bone cortical thickness of 1 mm (Fig 2-59b). Similarly, in the mesiodistal plane, when the distance between 2 standard implants is less than the required 3 mm (Fig 2-60a), placement of 2 implants with their flats facing each other allows this effective distance to be maintained (Fig 2-60b).

The drills required for the placement of these implants are circular; therefore, there is no bone compression around the whole perimeter of the implant neck. Compression stresses are limited to three well-defined areas (Fig 2-61). In these areas, compressive stresses are less intense when an implant is inserted into a healed cortical site. This is because the centrifugal compressive stresses in the direction of the bone tissue can relax thanks to the gaps between the periphery of the implant neck and the drilled bone socket (Fig 2-61).

For V3 implants with smaller diameters of Ø 3.5 and Ø 3.9 mm, the bone gap between the outer edge of the neck and the implant socket is only 0.1 mm and 0.2 mm, respectively. Gains in the bone lamella are very modest; however, these gaps are sufficient to create cortical areas that are not in contact with the implant surface and delimit a bone chamber. Several authors (Berglundh *et al.*, 2003; Franchi *et al.*, 2005; Colombo *et al.*, 2012) showed that osseointegration is delayed in areas of the implant surface that come into direct contact with bone tissue, whereas bone apposition starts immediately when the implant surface is not in contact with bone tissue (Fig 2-62). At the cortical ridge level, the remodeling phase can thus be bypassed when the entire neck circumference is in close contact with the bone tissue. Regardless of its volume, the gap adjacent to the flat is immediately occupied

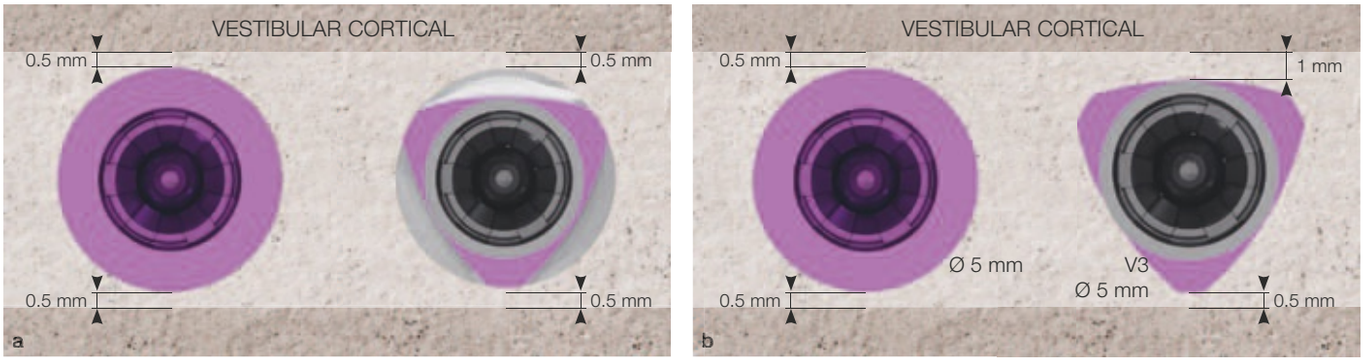


Fig 2-59 Indication of the flat to optimize buccal plate volume.

a Comparison between the placement of a conventional Ø 5 mm implant and that of an implant of the same diameter with a flat in the vestibular direction.

b Comparison of bone healing around the 2 implant types showing the possibility of obtaining a 1.2-mm bone lamella compared to 0.5 mm for the conventional implant.

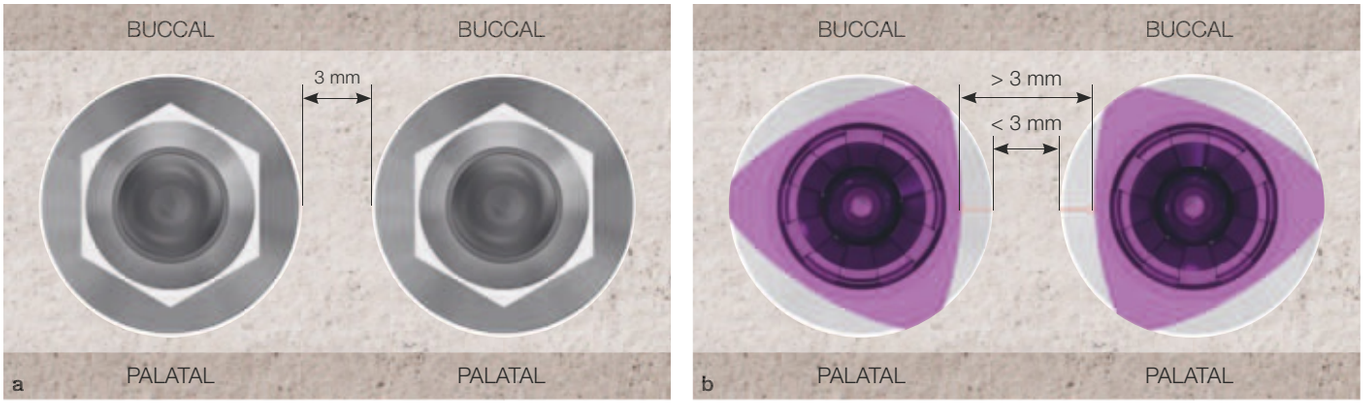


Fig 2-60 Indication of the flat to optimize the interimplant distance in the mesiodistal plane.

a Comparison between the placement of 2 conventional implants of Ø 5 mm where the interimplant distance is < 3 mm and 2 implants of the same diameter with a flat placed in the mesial direction where the interimplant distance is > 3 mm.

b Bone healing with an interimplant distance > 3 mm using a mesial flat.

by a blood clot. The blood clot undergoes all the transformations that lead to the formation of corticalized bone.

The implant design of the reverse conical neck, such as the Nobel Active implant, had already begun the process of increasing peri-implant bone volume at the emergence of the bone plate. The horizontal and vertical dimensions of the recess between the implant neck and body varied depending on the implant diameter, ranging from 0 mm to 0.55 mm depending on the implant diameter over a height of 0.9 mm. With the V3 implant, the volume facing the buccal plate is significantly increased for standard diameters and beyond (Ø 4.3 mm and Ø 5 mm); it is 0.3 mm over a height of 2.8–3.8 mm. It is in line with the evolution of implant design at the level of the crestal emergence of the neck.

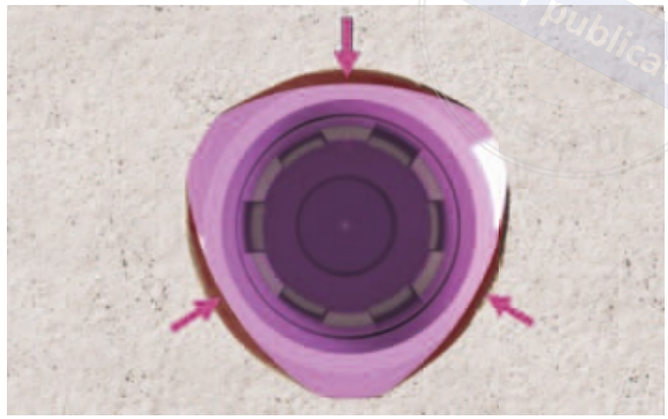


Fig 2-61 Intraoperative gaps around implants caused by the presence of cervical flats.
Cortical contact and gaps determined (arrows) by the cervical flat geometry. A stable blood clot immediately occupies these peri-implant gaps.

Conclusion

Knowledge of tissue response, both hard and soft tissue, to implantation is fundamental. Our understanding of this physiology has significantly progressed over the past decade. It allows us to better adapt our treatment in terms of time and flexibility. It also allows for shorter bone-healing protocols.

A better understanding of the hard tissue-soft tissue interaction has enabled us to improve the final esthetic result. However, these results must be maintained over the long term, over several decades. This imperative is dictated by the increasing number of young adults who come to benefit from the esthetic advances of implantology. The parameters that allow us to anticipate the long-term esthetic result and resist aging of the peri-implant tissues are not yet fully known (Buser *et al.*, 2013; Davarpanah *et al.*, 2014). With this in mind, treatment attempts from the implant surgery stage were initiated in the early 2000s in the form of an overcontour that could evoke overtreatment at the bone and soft tissue level (Buser *et al.*, 2013). It is still too early to say whether this approach will be successful in the long term; however, when an esthetic case arises it is best to put all chances aside if it is still impossible to guarantee the effectiveness of this approach in terms of esthetic success (Davarpanah *et al.*, 2016).

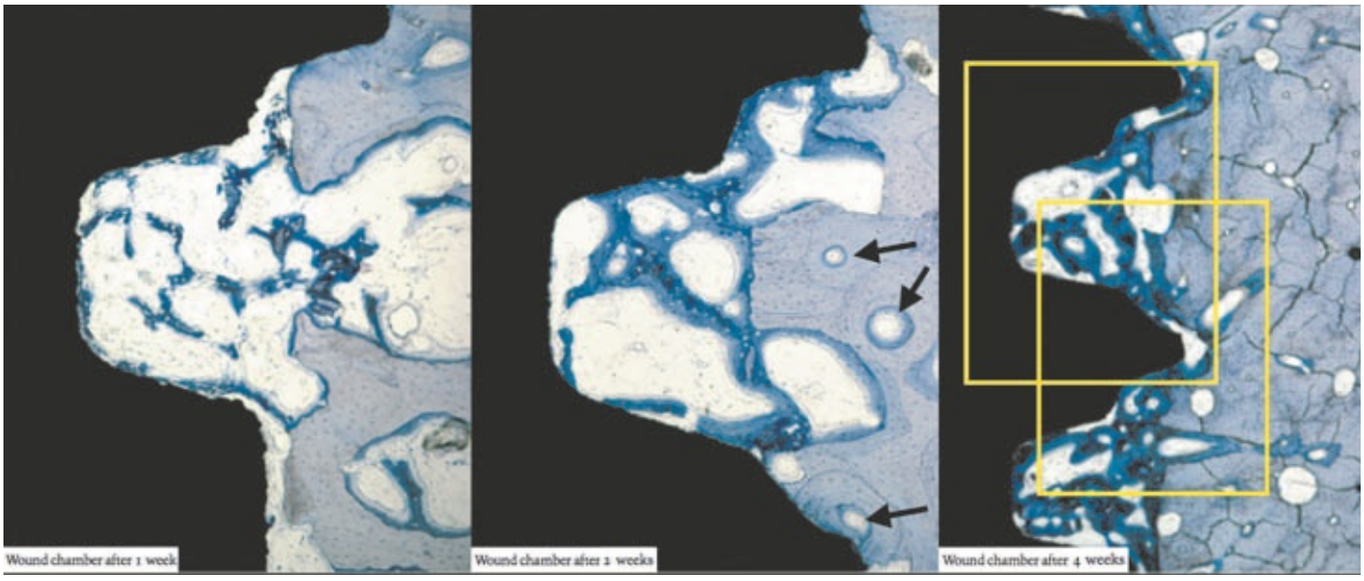


Fig 2-62 Bone reaction of immediate or delayed apposition, depending on whether the implant surface is in direct contact with or away from tissues.
Center and right: The bone areas in direct contact with the implant must pass through a remodeling reaction before participating in osseointegration as shown in the 2- and 4-week sections. Left: In the gaps in direct contact with the implant surface, bone apposition can start immediately, as shown in the 1-week section.