The dynamic interface: A review

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Abstract

The implant-to-tissue interface is an extremely dynamic region of interaction. Generally, a surgical procedure is performed on a patient to insert a foreign material into the bone, and the body is called on to "heal" the wound. The time schedule crucial for a healing process that is expected to result in *restitution ad integrum* must be determined with respect to the condition of the individual patient and tissue to be treated. There are various factors responsible for the formation of an adequate bone–implant interface. A comprehensive review of the response of bone to implant is described.

Key words: *Bone, implant, interface, osseointegration, risk factors, tissue*

INTRODUCTION

The term interface[1] is defined as a plane forming the common boundary between two parts of matter or space. It may represent a discrete boundary between the two materials or may consist of a region or zone of interactions between the two materials, i.e. the interface that exists between a dental implant and bone. The implant-to-tissue interface is an extremely dynamic region of interaction. The interface completely changes in character as it goes from its genesis (placement of the implant into the prepared bony site) to its maturity (healed condition). Relative movements (micromotion) between the implant and the bone at the time of placement are more likely to favor the development of a fibro-osseous interface. The healing phase of the interface is only the beginning of its dynamic nature. Generally, a surgical procedure is performed on a patient to insert a foreign material into bone, and the body is called on to "heal" the wound. A basic prerequisite for establishing true and lasting tissue integration of a non-biologic prosthesis with minimal risk of adverse local and general tissue reactions consists of a detailed understanding of the response behavior of highly differentiated hard and soft tissue to surgical preparation of the recipient site and installation of the prosthesis, as well as the long-term tissue adaptation to functional demands on the anchorage unit. The time schedule crucial for a healing process that is expected to result in *restitution ad integrum* (Latin term which means restoration to the original condition) must be determined with respect to the condition of the individual patient and the tissue to be treated.

HISTORICAL PERSPECTIVE

In 1952, Dr. Per Ingvar Branemark,[2] a Swedish anatomist, studied the functioning of the bone marrow and developed a technique known as *vital microscopy*. In this technique, a lens encased in titanium was introduced into the rabbit's tibia. After a month, this chamber was supposed to be retrieved, but to his

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annoyance, Sir Branemark found that he was unable to retrieve the chamber. He was not stuck by the idea of events until he accepted to join as professor in the University of Gothenburg, Sweden. There he used the same technique in the upper arm of human volunteers known as "graduate students." After a month, the chamber was retrieved and a lot of information was gathered regarding the working of the bone marrow; one additional finding was that titanium is compatible with human tissues. By 1968, Sir Branemark was ready to give a new name to this bone–implant interaction as "osseointegration." Osseointegration is a direct structural and functional connection between ordered living bone and the load carrying implant surface. He compared this equivalent to pseudoarthrosis, if there was a soft tissue interposition between the two. According to Schroeder *et al.* (1976),[3] it is the intimate contact of the bone to the implant and he referred to it as "functional ankylotic" adaptation. According to the american academy of implant dentistry (AAID) glossary of terms,[4] it is a contact established between normal and remodeled bone and an implant surface without the interposition of the nonbone or connective tissue. There is never a 100% bone-to-implant interface.

RESPONSE OF BONE AROUND THE NECROTIC IMPLANT CORTEX

Bone will heal with new bone formation only if certain local conditions are optimized. In other words, bone around the necrotic implant cortex depends on three factors: Adequate cells, nutrition to these cells, and stimulus for bone repair.[2] The differentiated bone cells are osteoblasts which are the bone-forming cells and these form bone at a rate of 0.17 mm^3/day. Osteocytes are the main stress detectors of bone. They play a role in bone remodeling. Osteoclasts are derived from the mononuclear cells of the blood. These resorb bone at a rate of 100 µm/day. In terms of adequate bone cell nutrition, in *cancellous bone*, the maximal vascular penetration rate has been established to 0.5 mm/day as compared to 0.05 mm/day in *cortical bone*. Adequate stimulus for bone repair may be cell-to-cell contact that is dependent on cellular activity, action of soluble matrix molecules, and action dependent on stress-generated electric potentials. Thus, taking these conditions into consideration, bone around the implant is supposed to respond in three different ways: If there is excessive trauma, fibrous tissue will form around the implant (fibrointegration); if vascularization has not been established, dead bone will be present around the implant and like a dead branch of a tree, it will be capable of carrying some kind of load; and if the local conditions have been fulfilled, normal bone will form around the implant, i.e., *osseointegration*.

STAGES AND TYPES OF OSSEOINTEGARTION

Bone healing is a fascinating biological accomplishment and one of the few examples in which regenerative procedures fully restore the original structure and function.[5] Once activated, it follows a biologically determined program that is divided into three stages which are: *Incorporation by woven bone formation, adaptation of bone mass to load (lamellar and parallel-fibered bone deposition), and adaptation of bone structure to load (bone remodeling). Osseointegration can be of three types, namely,* biointegration, chemical integration, and mechanical integration. Biointegration is achieved by a bioactive material like hydroxyapatite which bonds with collagen of bone. There exists some kind of a physiochemical bond. It is independent of any mechanical interlocking. Chemical integration is achieved by the formation of various bonds like van der Waals force, ionic bond, covalent bond, hydrogen bond, etc. Mechanical integration occurs when the bone to implant contact is aided by screws, vents, dimples, etc. This type of integration is independent of any chemical bond.[6]

FACTORS RELIABLE FOR OSSEOINTEGRATION

According to Albrektsson *et al.* (1981),[7] the various factors responsible for osseointegration are biocompatibility, design, surface conditions, the status of the host bed, the surgical technique, and the loading conditions applied afterward. Biocompatible materials are passive toward the tissue healing process.[8] Based on biocompatibility, these materials are further classified as shown in Table 1.

Table 1: Classification of materials according to their biocompatibility		
Degree of compatibility	**Characteristics of reactions of the bony tissue**	**Materials**
Biotolerant	Implants separated from adjacent bone by a soft tissue layer along most of the interface: Distance osteogenesis	Stainless steels, PMMA bone cements, CoCrMo and CoCrMoNi alloys
Bioinert	Direct contact to bony tissue: Contact osteogenesis	Alumina, zirconia, ceramics, titanium, carbon
Bioactive	Bonding to bony tissue: bonding osteogenesis	Calcium phosphate-containing glasses, glass-ceramics, ceramics, titanium

PMMA: Poly(methyl methacrylate)

Hoffmann *et al.* (2008)[9] showed that zirconia was also a biocompatible material with a similar bone apposition rate around the implant as that of titanium. Implant design determines the type of force that will be transmitted to the implant–bone interface. The various aspects of implant design that are important for the dynamic interface are geometry of the implant, implant length, implant width, and thread geometry. Desai *et al.* (2013)[10] evaluated the influence of implant length on stress distribution at the bone–implant interface in single, immediately loaded implants when placed in D4 bone quality. They concluded that short implants can be successfully placed in poor-quality bone under immediate loading protocol. Surface conditions are a key element in the reaction of hard and soft tissue to an implant and involve the implant surface characteristics. Surface roughness at the nanometer scale will influence the local electromagnetic fields close to the implant surface and may cause changes in the bonding by the van der Waals interaction. Colnot *et al.* (2007)[11] concluded that osteoblastic differentiation was favored by surface characteristics at a molecular level; thus, surface modification helps in the osseointegration process. On the contrary, AlFarraj *et al.* (2014)[12] showed in their study that implant design and surface characteristics had little influence at the bone–implant interface. The bone–implant interface is influenced by status of the implant bed and alveolar ridge resorption, irradiation, systemic conditions of the patient, and the bone quality/quantity. The factors influencing the surgical process for implantation include drilling out of the bone, mucosal characteristics, loading forces, and also the loading conditions applied afterward. The implant–bone interface is an extremely dynamic region and it can change its character even after the implant has osseointegrated. Excessive load can be detrimental to the interface. Strategies to avoid implant overload include inserting the implants perpendicular to the occlusal plane, placing the implants in tooth position, avoiding connecting implants to teeth, and if necessary, using a rigid connection. Implant placement with high insertion or low insertion torques also has an effect on bone resorption around the implant.[13] Placement of implants with high insertion torques doubles this zone of dead and dying osteocytes, thus increasing the microfractures and bone resorption around the implant.[14]

BONDING FORCES AND CHEMICAL PROCESSESS AT THE INTERFACE

There are several types of bonds by which biomolecules may be bound to the solid surfaces. The various bonds that occur at the interface zone are van der Waals force, hydrogen bonding, covalent bonding, and ionic bonding. van der Waals and hydrogen forces have bond strength of about 10 kcal/mole. Covalent and the ionic bonds are the strongest types of bonds that have a bond strength of 10–100 kcal/mole. One fact that is neglected is that the biological environment is aqueous, i.e. the most commonly occurring molecule is water. Water molecules can participate in hydrogen bonding, but will also modify, for instance, ionic bonding as the ions will be hydrated, that is, surrounded by a shell of water molecules. Furthermore, the presence of water may give rise to quite strong repulsive interactions at distances beyond the range of the short-range attractive forces. Persegian presented a hypothetical picture of the interface: Some molecules are weakly bound by van der Waals or hydrogen bonds, while others are much more strongly bound, may be at defect sites.[15]

Biomolecules with a high specificity for the implant surfaces are expected to build up the first monoatomic layer, on top of which a subsequent molecular layer builds up. Eventually, at larger distances (>1 nm), more organized biological complexes and cell structures are expected to appear. A look at the development of the implant zone with time after implantation indicates that in the first fraction of seconds, the surface is exposed to an aqueous medium containing various biomolecules (blood). Some of them will rapidly be attached through the formation of bonds. Many or all of them will eventually be replaced due to the appearance of more efficiently competing biomolecules. In the entire healing period of a few months, there is a continuous change in the chemical environment. The oxide starts to grow perhaps due to oxidizing radicals created in the metabolic processes. It is obvious that the chemical properties of the outermost atomic layer of the implant are the key factors in the integration processes.

ULTRASTRUCTURAL INVESTIGATIONS OF THE INTERFACE BETWEEN BONE AND IMPLANT

Albrektsson *et al.*[2,16] studied the interface zone around bone-integrated dental implant that had to be removed after 2½ years of clinical function in spite of an undisturbed bone anchorage. Scanning electron microscopy (SEM) and transmission electron microscopy revealed a *fibrous tissue-free interface.* Collagen bundles were seen at a distance of 1–3 μm from the interface. Collagen filaments were observed closer to the interface, but were always separated from the titanium surface by a proteoglycan layer of minimal thickness of 200 Å. This proteoglycan layer was partly calcified, and

calcified tissue was observed in direct continuity with the implant surface at a resolution level of the used equipment, i.e. 30–50 Å. Bone cells and processes from them were likewise separated from the titanium surface by a proteoglycan layer of thickness of a few hundred angstroms.[17] An analysis with the help of confocal laser scanning microscopy (CLSM) technique suggested that bone first formed as thin processes toward and across the implant surface, followed by further bone formation behind these processes. The interface between calcified bone tissue and the implant surface was characterized by a 10 µm space.[18]

HISTOLOGICAL OBSERVATIONS

Primary stability is obtained by congruency and press fitting, which leads to direct bone–implant contact. Press fitting often causes local overload, with plastic deformation of the lamellae and even fissures and microcracks. The local blood supply is disturbed by rupture and compression of vessels. The bone becomes avascular and necrotic, but still provides stability. At 3 months, it is partially or completely filled by lamellar bone that is formed in the second stage of osseointegration. Bone remodeling, the dominant mechanism in stage three, finally replaces the avascular areas with mature living bone. *Cortical remodeling substantially contributes to the increase in interface between implant and living bone that amounts to 60–70% after 15 months in this material.* Histomorphometric study of the bone–implant interface reveals that it varies from the time of implantation until complete healing has occurred. This process is known as *progressive osseointegration*.[6,19]

INTERRELATIONSHIP BETWEEN SEAL AND SUPPORT

It is convenient to study both these phenomena separately, but if we look at the pathologic processes at the implant surface, then it is in this area that the seal and the support come together. Crestal bone loss has been a consistent finding related to dental implants. Various factors are related to these phenomena and they include biomechanical load and bacterial flora surrounding the implant. In each case, either of them may be considered a primary etiologic factor and the other one as secondary.

RISK FACTORS FOR OSSEODISINTEGRATION

Dental implant osseointegration failure is a complex, multifactorial trait that has been investigated by several clinical follow-up and retrospective studies. The process is divided into early (smoking, aging, systemic disease, bone quantity and quality) and late (peri-implantitis, occlusal overload) events.[20-22]

CLINICAL SIGNIFICANCE OF THE DYNAMIC INTERFACE

Clinical application of the improving knowledge about the risk factors and indicators associated with implant loss and/or disease is urgently needed to improve and maintain the high success rate of dental implants over time. Site-specific monitoring of periodontal parameters around implants will help in early diagnosis of marginal infection and in effective monitoring of soft tissue health around these devices during the necessary recall appointments.

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Conflicts of interest

There are no conflicts of interest.

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