Osseous Healing Around Endosteal Dental Implants

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Abstract

To understand how an implant can function within physiologic limits of health, one should consider information from many areas including physiology, biochemistry, biomaterials and biomechanics in relation to normal repair and regeneration mechanisms. Providing that the implant material is biocompatible and that correct insertion techniques are employed, healing after insertion of an implant can be described using known repair mechanisms.

When an endosteal implant is inserted, epithelium, connective tissue and periosteum are incised and bone is removed in creating the osteotomy to receive the implant. This causes tissue injury and induces an expected slight inflammatory reaction. A cellular response is induced in which pluripotential cells undergo cell differentiation into a variety of cell types required for healing.[1] Biochemical and bioelectric signals have been described to occur to influence the processes of angiogenesis and ostoegenesis.[2] Although these responses occur naturally in response to tissue injury, factors within the clinical protocol directly affect them and therefore influence the healing.

Introduction

During bone healing, the ph changes at the site of the injury. Bleeding, local changes in pressure and edema follow injury. Within 48 hours, a clot is organized and the fibroblasts begin to lay down threadlike collagen fibers. Meanwhile, blood-borne cells continue to lyse and remove debris. With circulation partially interrupted, bone cells at the osteotomy can lose vitality. This dead skeletal tissue can act as scaffolding, and collagen fibres fill in around the implant and walls of the osteotomy. The dead bone is slowly replaced and the regions including the collagen fibres gradually ossify. Thus as old bone is removed; new bone regenerates in its place around the implant.[3]

The shock waves generated and transmitted into living tissue by the torque, speed, and vibration of the drill are important factors that may partially account for differences in the rate and quality of bone healing. The shock waves produced under low-speed drilling, commonly thought to be of a greater magnitude than those produced under high-speed drilling, are speculated to be partially responsible for the lower rate and quality of healing observed after low-speed osteotomy preparation.[4]

Bone physiology is controlled by an interaction of mechanical and metabolic factors. Under most physiologic circumstances, bone formation is primarily regulated by functional loading.

Success of endosseous dental implants is dependent on the establishment and maintenance of rigid implant-to-bone fixation, a condition referred to as "functional osseointegration." Adequate blood supply at the implant site and provision of minimal relative movement at the implant-bone interface during postimplantation healing period are essential. Since osseointegration with currently available implants relies primarily on mechanical interlock of newly formed bone with implant, implants should be characterized by both macroscopic form and microscopic surface features to ensure effective long term mechanical anchorage. Implant surfaces designed to increase the rate of new bone formation, leading to osseointegration, are of obvious benefits in the field of implant dentistry.

Osseous healing around dental implants

It is suggested to occur in 4 stages[5]:

1. Vascular Sprouting stage
2. Early bone formation stage
3. Bone growth stage
4. Bone maturation stage

1. Vascular Sprouting Stage

The vascular sprouting period occurs 3-7 days following implantation. It is the earliest angiogenic and osteogenic phase, corresponding to the beginning of vascularization stage following tooth extraction. This early evidence of angiogenesis is found where elongation of broken ends of fine blood vessels occurs, located in the walls of prepared osteotomy. There is vascular sprouting observed from immature sinusoidal capillaries invading the granulation tissue. Both of these expand into the peri-implant space from the walls of the bone marrow cavities lining the osteotomy. Pluripotential cells are activated and are observed in the threaded grooves or acute angles of interface geometry. After the 1st week, these are rapidly filled with fine collagen fibres and fibroblasts resulting in fibroblastic proliferation.

In case of 1-stage and 2-stage implants, tissue responses and micro-vascularization start in the grooves and threads of the interface architecture. In case of plate/blade form implants, new bone trabeculae isolated from one another are observed early in the blood clot period. This corresponds to the crestal portion of the implant osteotomy, which is expected to show ossification earlier because it was originally narrow. Root form threads and grooves, and the shoulder of blade forms are known to facilitate early fixation of the implant.

2. Early Bone formation Stage. (Fig. 1)

This occurs 2 weeks after implantation. It is the intial angiogenic stage and corresponds to the formation of bone trabeculae which is preceded by vascular ingrowth and osteogenesis.Woven bone (bony callous) originates from the untraumatized endosteal and periosteal areas. If sufficient space is available between the pre-existing bone structures and the implant surface, it can be filled with woven bone, which provides secondary implant stabilization. On osteo-conductive implant surfaces, bone can be produced directly by osteoprogenitor cells or mature osteobalsts. Woven bone may bridge distances upto 1mm within a relatively short time, provided that living or non-vital bone

surfaces can be used as stepping stones for bone formation and there is adequate vascularization.[6] On the other hand close implant-cortical bone contacts will undergo resorption, because the traumatized peri-implant bone has to be replaced which occurs as early as 4 weeks often beginning as peripheral cutting cones.[7]

3. Bone Growth Stage/ Lamellar Compaction and Remodeling: (Fig. 2)

This occurs 6-18 weeks following implantation. The intial primary spongiosa transforms to secondary spongiosa and proliferates to form new alveolar bone.Four weeks after implantation, bone trabeculae originating from the osteotomy over the peri-implant space perpendicular to the interface form a bone plate on and tangential to th einterface, referred to as stalked bone trabeculae. When a scanned section is viewed from the centre of the implant socket, island like bone plates and their capillary network occupy th peri-implant space twining around the stalked bone trabeculae.

Lamellar bone is appositioned to the newly generated woven bone as well as tops the pre-existing peri-implant cancellous trabeculae by typical osteoblastic layers, and primary osteons are formed. This modeling process leads to a more loadable stabilization of the implant which is anchored in lamellar bone (more mineralized) and to lamellar compacted bone.[8]

Lamellar compaction and increased callous formation are accompanied by remodeling that affect both the new bone generated in early bone formation stage and the pre-existing peri-implant bone tissue traumatized during preparation of the host site. Even in an optimal preparation of osteotomy, compact bone, adjacent to the surgically created implant bed will undergo necrosis as a result of disruption of nutritive vessels and inadequate collateral blood suppy. This will result in undermining resorption of the existing necrotic bone and replacement with new bone to guarantee stable and adaptable long-term implant integration (secondary stability).[8]

Frost described a remodeling process as a coupled activation, resorption and formation phases which last for about 17 weeks in humans. This was called the sigma cycle of bone healing.

4. Bone Maturation Stage: (Fig. 3)

This extends from 18-54 weeks. The final stage involves maturation and adaptation of the implant-bone interface, peri-implant bone and the entire implant supporting skeletal element. At this time formation of bone around the implant nears completion.The new and old bone interconnect, with their vascularization originating in bone marrow. At the implant socket base, several strong thick plates of trabeculae bone appear, resembling the cribriform plate of alveolar bone. In case of plate/blade form implants, vascularization and bone formation at the interface occurred in approximately I week less time than it was seen in root-form implants.

Factores That Stimulate Bone Repair

1. Biomechanical Stress-generated Bioelectric Signals.

Healthy strong bone is maintained when it is biomechanically stressed within physiologic limits. The cancellous bone around an osteopreserved implant can be densely packed, forming a cribriform plate similar to the socket or dental alveolus around a natural tooth. In case of implants, the collagen fibres originate at trabeculae of cancellous bone on one side, weave their way around a portion of the implant, and insert at another trabeculae, preferably not to far away.[9, 10] These collagen fibres have been shown to functionally tie and anchor the implant in place. Forces of occlusion load the collagen fibres, which can deform the trabeculae into which they insert, producing an osteostimulatory effect. Plate/Blade form configurations designed to control collagenous fibre length have properly dimensioned struts and Endopore root forms have porosities formed by diffusion-bonded microspheres, around which short collagen fibres may wrap to achieve this osteostimulatory effect. Biomechaniocal stress is hypothesized to promote growth in part because bone exhibits the piezoelectric effect. When collagen fibres inserted into a trabeculae of bone are stressed sufficiently to deform the trabeculae, a difference of electric potential is induced and bioelectric current flows.[11, 12] In case of trabeculae forming the dental alveolus, or implant alveolus in the osteopreserved mode of implant tissue integration, the surface closest to the tooth or implant exhibits a net negative charge. It is in compression as a result of deformation, which promotes bone deposition. On the other hand, the trabeculae surface farthest from the natural tooth or implant exhibits a net positive charge, because it is in tension as a result of deformation and therefore exhibits resorption.[13] It is the deposition of bone on the trabeculae surface closest to the implant that controls the thickness of the cribriform plate, and thus the thickness of the peri-implant ligament, ensuring stability and long-term function.

2. Cell- Generated Biochemical Signals

Osteoblasts accumulate around the negative pole of an implanted electrode; because this area's local ph is more alkaline that the usual body ph of 7.4. The functional enzyme of osteoblasts, alkaline phosphatase (ALK) works best at this slightly alkaline ph.[14] It is believed that the alkaline phosphatase produced by the osteoblasts breaks down phosphate compounds found in the interstitial fluids, yielding various byproducts including free phosphate. This free phosphate then combines with calcium to form calcium phosphate, an important building block of new bone.

3. Ground Substance-Generated Biochemical Signals.

Fibrblasts and osteoblasts are also responsible for the generation of components of the extracellular matrix that surrounds them.[15] This material seems to be amorphous, composed of a hydrated, semi-solid, gel-like mass that provides a mechanism for regulating water tissue levels. Although the exact mechanism are not clearly understood, ground substance components, cellular and chemical are known to influence the rate and quality of healing through generated biochemical signals that intensify following injury and repair.

Biological Considerations

Although endosseous dental implants have been used for many years, there is still much speculation in the literature about the bone-physiologic mechanisms that allow formation of bone-implant contacts.

Direct apposition of mineralized bone tissue to the implant surface appears to be a crucial factor in preventing persistent foreign-body reactions and connective tissue encapsulation of implants. Both bone stem cells (osteoprogenitor cells) and mature osteoblasts seem to promote the formation of woven bone and subsequently lamellar bone matrix on adequate bone-friendly (osteoconductive) surface, either directly or after apposition of a cementing substance (cement line).[16]

A sequence of biologic events leading to eventual osseointegration are suggested,[17] staring with the osteotomy, which is made for implant placement resulting in haematoma formation as the blood vessels get cut in the bone during the osteotomy preparation and fibrin clot formation within any gaps or spaces at the implanthost bone interface. This is followed by angiogenesis and resorption of the fibrin clot and the necrotic tissues as a result of trauma to the bone. The cells from the vasculature, like osteoblasts (or preosteoblasts) migrate and get activated either at the host bone interface (Distance Osteogenesis) or at the implant surface (Contact Osteogenesis) resulting information of an intial non-collagenous layer that becomes mineralized (cement line) with bone interdigitated with this layer. (Fig 4)

The healing process in endosteal implants is similar to primary bone healing. Initially, blood is present between the fixture and bone, then a blood clot forms. The blood clot is transformed by phagocytic cells, such as polymorphonuclear leukocytes, lymphoid cells, and macro-phages. The phagocytic activity level peaks during the time between the first and third day after surgery. During this period, formation of the procallus occurs, containing fibroblasts, fibrous tissue, and phagocytes. The procallus occurs, containing fibroblasts, fibrous tissue, and phagocytes.

The procallus becomes dense connective tissue and mesenchymal cells differentiate into osteoblasts and fibroblasts. The connective tissue is referred to as a callus, including osteoblasts that appear on the fixture surface. Osteogenetic fiber formed by osteoblasts has a potential to calcity. The dense connective tissue then forms a fibrocarti-laginous callus, usually forming between the fixture and bone. New bone penetrates and the new bone matrix is called the bone callus. This new bone matures, increasing in density and hardness. About this time, the prosthesis is attached to the fixtures and with stimulation; bone remodeling occurs (Branemark, et al., 1984). Haversian bone calcifies becoming dense and homogenous. Occlusal stresses stimulate the surrounding bone to remodel and the osseointegrated fixtures can withsand masticatory functions.

Conclusion

The osseointegration process observed after implant insertion can be compared to bone fracture healing. Logically, a certain immobility of the implant surface toward the bone should be maintained. A mild inflammatory response, as triggered by movements or appropriate electrical stimuli, may enhance the bone-healing response, but above a certain threshold, this is detrimental. It has been reported that when micromovements at the interface exceed 150µm, differentiation to osteoblasts will not occur; rather, a fibrous scar tissue is laid down between the bone and implant surface. Therefore avoiding such forces as occlusal load during the early healing period seems to be a safe approach. If the neighboring bone has been overheated or crushed during drilling, however, the necrotic area will prevent ingrowth of stem cells, and a scar formation or sequestered formation will result. The critical temperature for bone cells is as low as 47° C at an exposure time of 1 minute. This corresponds to the denaturing temperature of alkaline phosphatase, the main bone cell enzyme. Implant placement thus implies profuse cooling with intermittent moderate-speed drilling with sharp drills. Another complicating factor, well recognized from open wound fractures, is that microbial contamination jeopardizes the normal bone repair. Thus, when oral implants are placed, strict aseptic techniques should be maintained.

Woven Bone is the first to be formed in the gap between the implant and bone. Second after several months this is progressively replaced by lamellar bone under the load stimulation. Third, a steady state is reached after about 1 ½ years. Often, for oral implants, occlusal load is allowed as early as 2-3 months, while mostly woven bone is present. Woven bone grows fast, upto 100µm per day, and in all directions. It is characterized by a random orientation of its collagen fibrils, high cellularity, and limited degree of mineralization. Limited mineralization means that the bone's biomechanical capacity is poor, and thus occlusal load should be controlled. Woven bone can grow by apposition, originating from the bone lesion or by conduction, using the implant surface as a scaffold. The implant surface characteristics, such as material properties, surface free energy, and roughness profile, are determining factors that influence bone apposition. Altered implant surface topographies, such as those created by acid etching, blasting, combinations of acid etching and blasting, or increasing the titanium oxide layer appear to result in greater bone apposition to the implant surface as compared to a turned or machined surface.

References

1. Roberts, GAretto: Bone physiology and metabolism. In Misch, editor: Contemporary Implant dentistry, ed 2, Mosby.
2. Turner: Functional determinants of bone structure: beyond Wolff's law of bone transformation, Bone 13: 403, 1992.
3. Babbush: Implant Dentistry: a long term survey and comparative study with fixed bridgework, J Oral Implantol 7: 1, 1977.
4. Eirich: the role of friction and abrasion in the drilling of teeth. Dept of Health, Education and Welfare, DHEW Pub No 76-670: 234-235, 1976.
5. Ohta Y: Comparative changes in microvasculature and bone during healing of implant and extraction sites, J Oral Implantol 19: 184, 1993.
6. Schenik, Wehrli: Zur Reaktion des Knochens auf eine zement freie. Orthopade 18: 454, 1989.
7. Haider, Watzek: Histologisch-morphometrische Analyse der Knochenstruktur und Bohrer-Kuhlmethode Stomatol, 88: 540, 1991
8. Roberts: Bone tissue interface. J Dent Educ 52: 804-809, 1988.
9. Weiss: Tissue integration of dental endosseous implants: description and comparative analysis of the fibro-osseous and osseous integration systems, J Oral Implantol 12: 169, 1986.
10. James RA: Tissue response to dental implant devices. In Hardin JF, editor: Clark's Clinical Dentistry, Philadelphia, 1986.
11. Chiarenza: Cathode stimulation of alveolar bone around endosteal blade implants, Implantogist 1: 90, 1977.
12. Hayes, Carter: Biomechanics of bone. In Simmons, kunin, editors: Skeletal research; an experimental approach, New York, 1979, Academic Press.
13. Malkani: Cytoplasmic modifications at the contact zone of osteoclast and calcified tissues, Calcif Tissue Res 11: 258, 1973.
14. Parfitt AM: The cellular basis of bone remodeling: the quantum concept re-examined in light of recent advances in cell biology of bone, Calcif Tissue Int 36: 37, 1984.
15. Sampath, Muthukumaran, Reddi: Isolation of osteogenin, an extracellular matrix associated bone-inductive protein, by heparin affinity chromatography, Proc Natl Acad Sci 84: 7109, 1987.
16. Davies JE: The Bone Biomaterial interface. University of Toronto Press, 1991.
17. Davies J. Mechanisms of endosseous integration. Int j Prosthodont , 11: 391-401, 1998.

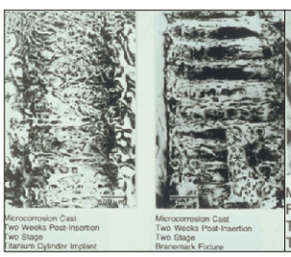

Fig. 1

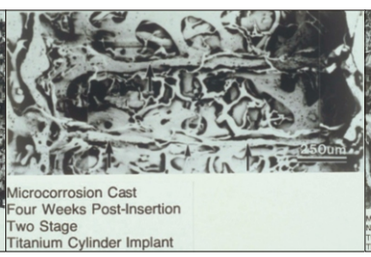

Fig. 2

Fig. 3

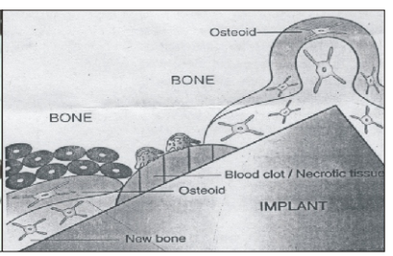

Fig. 4