Harsukh Educational Charitable Society
International Journal of Community Health and Medical Research
Journal home page:www.ijchmr.com doi: 10.21276/ijchmr

ISSN E: 2457-0117 **ISSN P:** 2581-5040 **Index Copernicus** ICV 2018=62.61

REVIEW ARTICLE

HEALING IN DENTAL IMPLANTS- REVIEW ARTICLE

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ABSTRACT
Dental implants are a routine dental procedure employed these days for prosthetic rehabilitation of missing teeth. Today the continued high rate of success achieved with dental implants allows a greater number of patients to enjoy the benefits of fixed rather than removable restorations. Throughout history, many researchers have attempted to use dental implants as a solution to edentulism. The present review aimed for highlighting important aspects of healing in dental implants.
Key words: Dental implants, Healing

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This article may be cited as: Gupta R, Luthra RP, Bhuvaneshwari K. Healing in dental implants: A review. HECS Int J Comm Health Med Res 2019; 5(4):113-116.

INTRODUCTION

The successful replacement of lost natural teeth by tissue-integrated tooth root analogues is a major advancement over the last 25 years. Today the continued high rate of success achieved with dental implants allow a greater number of patients to enjoy the benefits of fixed rather than removable restorations. Throughout history, many researchers have attempted to use dental implants as a solution to edentulism. Unfortunately much of this work resulted in failure. It is critically important to understand how oral implantology has evolved in order to understand where we have been, and where we are going. Osseointegration is defined as a direct bone anchorage to an implant body which can provide a foundation to support a prosthesis; it has the ability to transmit occlusal forces directly to bone (Albrektsson, et al., 1981; Branemark, 1983; Carlsson, et al., 1986).[1]

Albrektsson et al.[2] discussed 6 different parameters that needed to be controlled for proper bone anchorage to occur: the biocompatibility, design and surface conditions of the implant, the state of the host bed, the surgical technique and the loading conditions. The goal of implantology research is to design devices that induce controlled, guided and rapid integration into surrounding tissues. More specifically, in addition to acceleration of normal wound healing phenomena, implants should result in an interfacial matrix with a composition and structure characteristic of bone, and the matrix should have adequate biomechanical properties. These outcomes would allow not only faster recuperation for the patient, but also stable fixation between bone and implant that would permit early or immediate loading of the device. To achieve these goals, however, a better understanding of tissue healing events is needed.[3]

BIOLOGY OF BONE

It is essential to have a thorough understanding of basic bone biology. Bone is classified as either Compact bone (referred to as Cortical bone) or Spongy bone (referred to as Cancellous bone). Compact bone has outer circumferential lamellae, inner circumferential lamellae, haversian lamellae, and interstitial lamellae which account for hardness and density of this bone. Within compact bone, spongy bone has a three-dimensional network called bone trabeculae. Spongy bone architecture is cavernous and less dense such that the hardness is less when compared to compact bone. Spongy bone with less density and less hardness is not a stable base for primary fixture fixation. Only compact bone can provide a stable base for primary fixture fixation.

BONE HEALING

An injured bone heals either by primary or secondary process.

Phases of bone healing:

a. Injury phase:

One way of looking at the initiation of bone repair is to regard 'injury' as the initiating mechanism. Injury is known to act as a releasing stimulus for various growth factors as well as to sensitize various cell types.

b. Granulation phase:

The second healing phase at a few weeks after injury has been termed the granulation stage. At this time new local connective

tissue, new capillaries and supportive tissue appear, whereas an abundant new bone formation is generally not seen until the next healing stage.

c. Callus phase:

In bone healing, a delicate balance is established among the several tissues that proliferate in the case of a bone injury. Information between the different cellular subgroups is maintained via chemical signals- mediators.

FOREIGN BODY REACTION:

Organization or an antigen-antibody reaction occurs when a foreign body is present in the body. This reaction occurs in the presence of protein, but with implant materials devoid of protein, there is no antigen-antibody reaction.[1]

CONDITIONS AFFECTING BONE REPAIR AT AN IMPLANT SITE: (CELLULAR BACKGROUND)

Bone tissue is highly cellular and richly vascularized. At rest, bone will receive about 11% of the cardiac output. No matter how careful the preparatory technique, a necrotic border zone will inevitably appear around any surgically created bone defect. The width of this necrotic zone around an implant site will primarily depend on the generated frictional heat at surgery, and also on factors such as the degree of vascularization etc. In principle, bone may react in three different ways as a response to the necrosis.

1. Fibrous tissue formation may occur.
2. Dead bone may remain as sequestrum without repair.
3. New bone healing or Osseointegration (bone formation = bone resorption)

Bone repair of the necrotic implant cortex will depend on the presence of

1. Adequate cells
2. Adequate nutrition to these cells
3. Adequate stimulus for bone repair.

THEORETICAL POSSIBILITIES TO REINFORCE BONE RESPONSE AT IMPLANT SITES

Morphogenic protein may act as a potential bone-accelerating substance. However, to date researches are still in progress to find out the potential in humans. Hormonal stimulation would be another way of reinforcing the osteogenic response, because it is known that osteoblastic function is stimulated by Growth Hormone (GH). However, administration of GH to healing bone grafts in a rabbit experimental model was not shown to have any beneficial effect. Fibrin Adhesive System (FAS) has been suggested to reinforce the incorporation of experimental bone implants. However, contradictory evidence has been presented by Albrektsson et al. In addition, Zilch and Noffke found no significant increase in bone formation after treatment with FAS. Electrical stimulation with direct current of magnitudes approximately 5 to 20 μA has been demonstrated to increase the interfacial strength of experimental implants by several groups.

Mode of stimulus	Examples
Hormone administration	Growth hormone
Drug influence	FAS, BMP
Electrical stimulation	DC, AC

BONE RESPONSE TO MECHANICAL LOADS

The implant-to-tissue interface is an extremely dynamic region of interaction. The interface completely changes character as it goes from its genesis (placement of the implant into the prepared bony site) to its maturity (healed condition). The biochemical environment plays an immediate role in the quality and compositional outcome of the new interface.

Indicators of the biologic response:

a. Changes in concentration of intracellular mediators:

In general, cell surface receptors relay information by activating a chain of events that alters the concentration of one or more small intracellular signaling molecules often referred to as second messengers or intracellular mediators. In turn, these messenger molecules pass the signal on by altering the behavior of selected cellular proteins.

b. Changes in cellular proliferation:

Many studies have reported increases in cell proliferation, total protein production, and DNA synthesis in response to mechanical strain. At high magnitudes of strain, osteoblasts proliferate and decrease their production of osteoblast phenotypic markers, such as alkaline phosphatase and bone matrix proteins while at lower magnitudes of strain, osteoblasts exhibit a more differentiated state, with an increase in alkaline phosphatase and matrix protein production and a decrease in proliferation.

c. Changes in cellular morphology and/or organization:

Cells respond differently to various types of strain. Buckley et al noted that the cells orient themselves perpendicular to the long axis of the applied mechanical strain. This perpendicular alignment was noted at 4 hours after loading and was significant by 12 hours. They suggested that the preferred orientation may have resulted from a mechanical effect on the osteoblast, wherein cell attachments were broken in the maximum strain direction, leaving only those attachments already present in the least strained conformation. A second hypothesis suggested that the cells may have resolved their focal contacts and migrated in an attempt to minimize the strain to which they were subjected.

d. Altered expression and/or Reorganization of osteoblast integrins:

Although changes in the distribution of the cytoskeleton in mechanically strained cells have been reported, the exact mechanism for the initial detection and transduction of mechanical force into a biologic signal has yet to be determined. One possible transduction pathway is the extracellular matrix-integrin-cytoskeletal axis.

e. Changes in gene expression:

To characterize the biologic response of osteoblast-like cells to external mechanical loading, many researchers are investigating strain-induced alterations in patterns of osteoblast gene expression. Several authors have reported that the initial response to strain is a rapid increase in c-fos mRNA expression, indicative of increased proliferation, paired with a rapid decline in levels of mRNA encoding bone matrix proteins, such as type I collagen, osteopontin, and osteocalcin. A “rebound” effect or reversal of this trend is usually seen with time as the proliferation tapers off, accompanied by an increase in expression of the matrix proteins.

THE BIOMECHANICAL RESPONSE:

a. Dependence on direction of loading:

The presence of teeth and/or implants significantly increases the trabecular bone amount and density within the residual alveolar bone.

b. Dependence on rate of loading:

Bone fails at a higher load, but with less allowable elongation (deformation) at higher as compared with lower strain rates. Thus bone behaves in a more brittle fashion at higher strain rates. Strain rate to which bone is normally exposed varies from 0.001 sec^{-1} for slow walking to 0.01 sec^{-1} for higher levels of activity.

c. Dependence on duration of loading:

Carter and Caler have reported the creep-fracture curve for adult human bone at a constant stress of 60 Mpa. Fatigue failure has been reported for in vivo bone by Carter and associates and by others at relatively low cycles (10^4 to 10^8 cycles). Given the high magnitude of cycles encountered in oral function, the relatively low in vivo fatigue life reported in bone (ie, accumulated fatigue damage) is likely to be accommodated in vivo through the normal process of bone remodeling.

d. Dependence on species and anatomic location:

Large variations have been noted in experimental measurements of elastic modulus and ultimate compressive strength of trabecular bone. Finite element models of the human mandible have shown that cortical bone plays a major role in the dissipation of occlusal loads. Thus load patterns on trabecular bone and microstructure of trabecular bone may contribute to differences in the mechanical behavior of the mandible as compared with other anatomic regions.

e. Dependence on side constraint:

The biomechanical response of trabecular bone in the mandible is highly dependent on the presence or absence of cortical plates as a "side constraint". Qu et al showed a 65% higher stiffness (elastic modulus) for trabecular bone of the mandible when constrained by cortical plates as compared with unconstrained test values.

f. Dependence on structural density:

Trabecular bone is a porous, structurally anisotropic, inhomogeneous material. Qu et al specifically reported on the mechanical properties of mandibular trabecular bone. Regional differences were noted in the human mandibular trabecular bone elastic modulus and ultimate compressive strength, exhibiting up to 47% to 68% higher mean values in the anterior region compared with the posterior region of the mandible.

Success criteria for osseointegrated implants

Smith D.E et al examined the possible criteria for implant success in the light of available supporting studies for implant success.

Consideration should be given to evaluating the following criteria:

a. Durability
b. Bone loss
c. Gingival health
d. Pocket depth
e. Effect on adjacent teeth
f. Function
g. Esthetics
h. Presence of infection, discomfort, paresthesia or anesthesia
i. Intrusion on the mandibular canal
j. Patient attitude & motivation

Revised criteria for implant success

1. Individual unattached implant is immobile when tested clinically.
2. No evidence of peri implant radiolucency is present as assessed on an undistorted radiograph.
3. Mean vertical bone loss is less than 0.2 mm after 1st year of service.
4. No persistent pain, discomfort or infection.
5. A success rate of 85% at the end of a 5-year observation period and 80% at the end of a 10-year period are minimum levels of success.

Saadoun A.P et al, discussed the keys to success in implant osseointegration. Quality of bone is the determining factor in success rates; the deeper the bone, the lower the failure rate; a failure rate is most likely to take place during the first year after placement; a higher success rate is found in the mandible; and a higher success rate is found with HA-coated implants**.**

CONCLUSION

The tooth is anchored to its neighbouring bone by soft tissue, a highly differentiated periodontal membrane. It seems natural; therefore, that early investigators of oral implants claimed that the soft tissue seen around their devices was a replication of nature and thus would lead to lifelong function. However, certain histologic differences between the proper ligament and the soft tissue that is found around these metallic devices were observed. This lead to the formulation of new nomenclature such as pseudo ligaments or fibrous osseointegration instead of much desired re - evaluation of the basic concept of a soft tissue anchorage.

To use the alternative to a soft - tissue capsule attachment, namely osseointegration, is based on only one reason,, however an essential one:: Osseointegrated or al implants have been found to result in long - term success rates.. Although various factors govern the osseointegration of implants, the successful management of implant dentistry depends on the meticulous diagnostic,, planning and surgical skills of the operator.. The operator can change the prognosis towards a better outcome. Failures can be kept at a minimum if careful planning and execution is carried out at every stage of the implant therapy.

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