Healing of Tooth Extraction Socket

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Abstract

Bone is a highly organized composite material that consists of organic matrix and inorganic mineral substances. It serves as depository for calcium, phosphates, and other minerals and firm skeletal support for soft of the human body.

Introduction

During bone healing, the ph changes at the site of the injury. Bleeding, local changes in pressure and edema follow injury. Some cells burst, spilling toxins into the surrounding area, and certain bioelectric and biochemical phenomena are known to occur. In response to these and other factors, pluripotential cells, marrow cells, and cells lining the periosteum and endothelium act as sources of fibroblasts, osteoblasts and osteoclasts. Within 48 hours, a clot is organized and the fibroblasts begin to lay down threadlike collagen fibres. 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.[1]

Biology of Bone

Bone is a highly organized composite material that consists of organic matrix and inorganic mineral substances. It serves as depository for calcium, phosphates, and other minerals and firm skeletal support for soft of the human body.The fundamental principle of bone architecture in humans, is that compact and cancellous structures are distributed within the skeleton in such a fashion that they can best sustain mechanical loads, thus reflecting the loading conditions in all three dimensions.

Bone Morphology

Macroscopically bone can be classified as:

1. Compact /Cortical bone
2. Spongy / Cancellous bone

Compact Bone : Consists of lamellae or layers of cells, and a matrix made of inorganic and organic components. The cells present are called osteocytes; they are located in lacunae and have cell processes for nutrient diffusion within small channels or canaliculi.

The matrix component or osteoid is approximately 40% by weight and consists of Type 1 collagen, gylcosa-minoglycans and adhesive protein, osteonectin. The inorganic component is also 40% by weight and consists of hydroxyapatite, the apatite crystal of calcium and phosphate. Compact bone has outer circumferential lamellae, inner circumferential lamellae, haversian lamellae, and interstitial lamellae, which account for the hardness and density of this bone.It is covered by periosteum and has collagen fibres, osteoblasts, and osteoclasts. Periosteum is attached tightly to the bone surface by Sharpey's fibres and serves as protection for bone. Osteoblasts and Osteoclasts in periosteum are involved with remodeling, bone resorption and apposition.

Spongy / Cancellous Bone : Within compact bone, spongy bone has a three dimmesional network called bone trabeculae. Spongy bone architecture is cavernous and less dense such that the hardness is less when compared to compact bone. The bone trabeculae configuration creates a large surface area for an abundance of osteoblasts and osteoclasts, which are associated with bone formation and resorption. Large blood vessels transverse within bone trabeculae.[2] microscopically; depending on the age, function and systemic factors, bone can be classified into 4 types:

1. Woven Bone
2. Lamellar Bone
3. Bundle Bone
4. Composite Bone

Woven (embryonic) Bone (Fig 1)

This is a highly cellular osseous tissue that is formed rapidly (30-50 µm/ day) in response to growth, injury, or biomechanical adaptation. Compared to mature bone, it has relatively low mineral content, a more random fibre orientation and minimal strength[3].

Lamellar Bone (Fig 2)

It is the principal load bearing tissue of the adult skeleton. It is formed relatively slowly (0.6 µm/day) and mineralizes by a primary and secondary mechanism[4]. Full maturation of lamellar bone requires 6-12 months.

Bundle Bone

It is a special kind of woven bone that can be found in the zones of attachment of tendons, ligaments or joint capsules if mineralized bone is penetrated by collagen fibres. This has striations that are extensions of Sharpey's fibres and is similar to lamellar bone in strength. It is characteristic of fibro-osseous attachments and would be expected in a stable fibro-osseous attachment.[5]

Composite Bone

This is formed on cortical surfaces during wound healing, growth, and biomechanical adaptation. Intially, a porous lattice of woven bone captures blood vessels along a periosteal or endosteal surface. This lattice then fills with load-bearing, lamellar bone. Lamellar compaction of composite bone is an important step in achieving a load bearing osseous interface.[6]

Physiologic Adaptation of Bone

To fulfill its dual functional role of support and metabolism, bone responds to a complex array of mechanical, bioelectric, metabolic and local mediators like cytokines and growth factors.Under steady-state conditions, osteoblast differentiation is mechanically mediated and is stress-strain dependant.[7]Surgical placement of a dental implant elicits an osteogenic response which is vascularity dependant.

Modeling of Bone (Fig 3)

It is a surface-specific activity, (apposition or resorption) that produces a net change in the size and/or shape of bone. It is an uncoupled process, meaning that cell

activation (A) proceeds independently to bone formation (F) or resorption (R). It refers to a generalized change in overall dimensions of a bone's cortex or spongiosa, hence a mechanism of differential growth and structural adaptation.It is a fundamental mechanism of growth, atrophy, and reorientation.

Remodeling of Bone (Fig 3 & Fig 4)

Remodeling is defined as turnover or internal restructuring of previously existing bone. It is a couled tissue level phenomenon. Remodeling, bone resorption and appostion helps maintain blood calcium levels and does not change the mass quantity of bone. In spongy bone, remodeling occurs on the surface of bone trabeculae due to the abundance of osteoblasts and osteoclasts available, which get stimulated by the occlusal forces applied to the spongy bone. This stimulation causes bone cells to differentiate into osteoclasts involved in bone resorption and osteoprogenitor cells to differentiate into osteoblasts involved in bone formation. The same phenomenon occurs in compact bone at the remodeling site. Remodeling includes localized changes in individual osteons or trabeculae: turnover, hypertrophy, atrophy, or reorientation.

Remodeling Cycle/ Sigma Cycle (17 wks) (Table 1) (Fig 5)

Since remodeling of bone is a coupled tissue phenomenon, the following stages occur;

1. Activation Phase (A): Stimulus to the bone causes circulating preosteoclast cells to cross the blood vessel wall, enter the connective tissue and form osteoclasts. The osteoprogenitor cells differentiate into osteoblasts from the paravascular connective tissue cells.In this phase of hours to days the osteoclasts form the cutting head.

2. Resorption Phase (R): The osteoclasts result in active resorption at the remodeling site and results in gradual decrease in bone density. During this phase, the cutting cone opens a cavity of 120 to 180µm in diameter. Once this phase is complete it is followed by the

3. Quiescence or Reversal Phase (Q): Here the quiescent stage of osteoblasts changes into an active stage. There is reversal of bone resorption stage into a bone deposition stage as there is a cessation of osteoclastic activity and beginning of oesteoblastic bone formation, resulting into the next stage of ;

4. Formation Phase (F): Active Osteoblasts produce proteins for collagen formation which is a step in bone formation.

The duration of the A→R→Q→F remodeling cycle, also called the **sigma cycle** is about 6 weeks in rabbits and 17 weeks in humans.To maintain a constant level of bone remodeling, there should be local stimulation[8,9] as well as crucial levels of thyroid hormone, calcitonin and vitamin D within the system. In case of implant placement sites, occlusal force stimulus and general health management are both important to optimal bone remodeling criteria.[10]

Stages of Healing

The progression of osseous healing after tooth extraction is nearly equivalent to that observed for usual wound healing in the following sequential manner.

1. Granulation stage
2. Initial angiogenic/neurovascular stage
3. New bone formation stage
4. Bone growth stage
5. Bone reorganization stage

1. The Granulation Stage (Fig. 6, Fig. 7)

The granulation stage extends for 5 days from the time of extraction. Early granulation tissue is observed at the base of the socket, extending crestally along the socket wall. A blood clot occupies the central portion of the socket. The earliest angiogenesis observed is sprouting or budding extensions of the preexisting blood vessels sinusoidal capillaries developing from broken ends of blood vessels in the remains of the periodontal ligament at the cribriform plate. This angiogenesis starts at the base of the socket where thick, strong trabeculae already exist and along with their accompanying cappiliary plexes. This is the area at the socket base which is injured the least during tooth removal and maintains its vascular pattern intact, is the most active area initially.

2. Initial Angiogenic / Neurovascularization Stage

This period extends for 1 week from the time of extraction. The blood clot becomes smaller. The new sinusoids extending along the socket wall form the base move beyond the height of the clot, until about two thirds of the socket is filled with newly formed sinusoids. At the base of the socket, the first new bone trabeculae may be observed (Fig 8A).

3. New Bone Formation Stage

This occurs 2 weeks form the time of extraction. Now the entire socket is filled with granulation tissue replete with newly formed sinusoids. The bony wall of the base and the side of the socket presents a dense lattice of trabeculae (Fig 8B). There is intimate interrelationship between immature sinusoids exhibiting anastomosis and new bone. No new bone trabeculae are observed in areas of nonanastomosing sinusoids of blind ends of sinusoids. Woven bone is delineated by incompletely ossified trabeculae. Bone trabeculae formation is governed by the expansion and location of sinusoids. This activity reaches its peak in the 2nd week following tooth extraction and bone development becomes rapid.

4. Bone Growth Stage (Fig 8C)

This occurs 4-5 weeks following tooth extraction. Additional trabeculae are deposited, and the base and the walls of the socket have thickened and now occupy about two thirds of the original socket volume. The secondary spongiosa of the next stage begins to develop. In areas where sinusoids are still evident, new bone forms.

5. Bone reorganization Stage (Fig. 8D)

This stage occurs 6 weeks after tooth extraction. Primary spongiosa reorganize into an irregular and larger framework as secondary spongiosa, again starting at or near the base of the socket extending upwardly.

Prerequisites for Optimal Bone Healing Response

The vascularization and bone formation that follow implant insertion require the presence of following factors to promote healing:

1. Adequate Cells
2. Nutritional elements
3. Required signal stimuli.

Summary

Primary bone healing occurs at a fracture site with a clean break. The sites are positioned by pressed fixation or closely approximated. In primary bone healing, there is well-organized bone formation with minimal granulation tissue formation.

Secondary healing occurs where a large defect or large fracture site precludes close approximation of the two sites. In contrast to primary bone healing, secondary bone healing may have granulation tissue formation.

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Legends

Fig. 1: Woven bone at sites of intremambrenous bone formation. Osteoblasts produce the nonmenirelized bone matrix osteoid, which is later mineralized (dark blue)

Fig. 2: Lamellar bone formation. Osteoblasts lay down new lamellar bone onto previously resorbed mature lamellar bone during bone remodeling. Newly formed lamellar bone is more intensely stained (dark purple) than the preexisting bone (bright purple)

Fig. 3: This schematic drawing of diaphyseal (midshaft) cross-section reveals differential sites of bone modeling (M) and remodeling (R). Remodeling is turnover of previously existing bone. Modeling (change in shape or form) can be anabolic (formation) or catabolic (resorption). Bone modeling is mechanism of differential growth and structural adaption. (From Roberts WE, Garetto LP, DeCastro RH: : J Indiana Dent Assoc 68:19, 1989)

Fig. 4: Remodeling of newly formed woven bone into mature lamellar bone. Osteoblasts first form woven bone, which starts growing from the edges of local bone

Fig. 5: Schematic drawing of cutting/filling cone (evolving secondary osteon) demonstrates mechanism of cortical bone remodeling. (Roberts WE et al: Am J Orthod 86:95, 1984)

Fig. 6 :

Fig. 7 :

Fig. 8 A.

Fig. 8 B

Fig. 8C;

Fig. 8D:

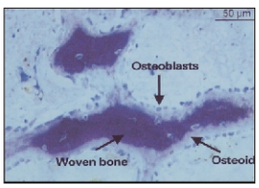

Fig. 1

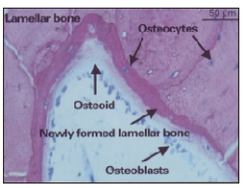

Fig. 2

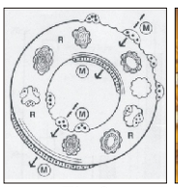

Fig. 3

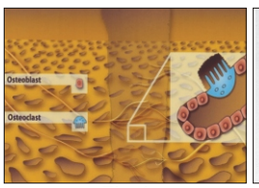

Fig. 4

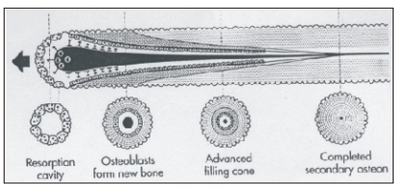

Fig. 5

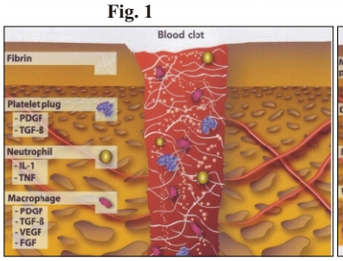

Fig. 6

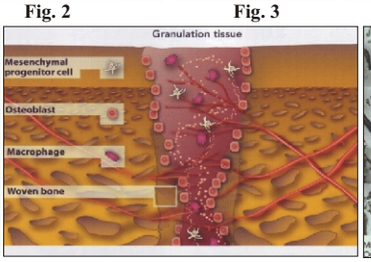

Fig. 7

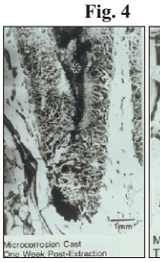

Fig. 8A

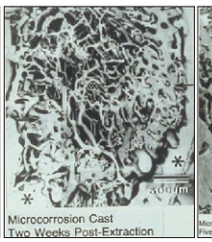

Fig. 8B

Fig. 8C

Fig. 8D