Bone healing

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FIGURE 1

Malunited 4-part fracture, five months after accident.

The first step in fracture healing is stabilization of the fracture site, mechanically. Healing cannot occur if the bone fragments are not reduced and held securely in position. As surgical technologists, we are aware of the surgical interventions that are used to reduce and stabilize a fracture. The procedure may be as simple as closed reduction and casting or may be more invasive. Many types of devices are available for surgical fracture stabilization, including the internal and external fixation devices with which we are all familiar. In the extreme case, partial or total joint replacement may be indicated.

If a fracture has been properly immobilized and aligned, the normal physiologic mechanisms of the human body should facilitate bone healing. Sometimes, however, that process gets disrupted. This article, the second part in a series, discusses disrupted bone healing and the factors that enhance proper bone healing. To review normal physiology, see "Bone Healing-Normal" in the March 2002 *Journal.*

Disrupted bone healing

A disruption at any stage of bone healing or maintenance can be responsible for a variety of abnormal conditions.

Avascular necrosis occurs when the capillary network or collateral circulation cannot be reestablished following a traumatic injury of when the vascular system is disrupted by other

means. This can be pharmacologic (eg steroid use), pathologic (eg diabetes), or idiopathic. Decreased blood supply to the bone may lead to irreversible necrosis.

Osteomyelitis is an inflammation of the bone, marrow, and possibly the surrounding tissue commonly due to a *Staphylococcus aureus* infection. Prevention is the main issue; although, occasionally wound contamination cannot be avoided due to the type of injury sustained (especially in the case of a compound fracture). Chronic infection can result if the acute condition is not recognized or treated properly.

Compartment syndrome is an increase in pressure within a closed space. Excess pressure leads to neurovascular compromise. Tissue viability may be affected, increasing the risk for infection, and permanent nerve damage can occur.

Malunion is solid union of the fractured bone in a deformed position (Figure 1). This results from either inadequate reduction or immobilization. Patient noncompliance is often a factor.

Delayed union may have one or several determining causative factors including pathologic (eg osteoporosis), mechanical (eg distraction of the fracture site or inadequate immobilization), traumatic, referring to the type of injury sustained (eg comminuted fracture).

Nonunion is a failure of the bone fragments to calcify together. Often the space between the fragments is too large or soft tissue may be entrapped between the fragments. Improper immobilization and excess activity by the patient can disrupt an otherwise normal cycle of bone healing. Infection, nutrition, hormones, and circulation are also factors to be considered.

Factors that enhance bone healing

Several options are available to the clinician and the patient to enhance fracture healing.

- Good nutrition and overall health are two very important influences on fracture healing. Calcium (RDA for the average, healthy adult is 800 mg) and Vitamin D (RDA for the

Bone healing terminology

Endosteum—thin vascular membrane that lines the marrow cavity of long bones.

Fibroblast—immature, fiber producing, connective tissue cell proliferated at the site of inflammation. Fibroblasts are capable of differentiation into a chondroblast, a collagenoblast, or an osteoblast.

Osteoblast—cell associated with bone formation that arises from a fibroblast.

Osteoclast—cell associated with destruction and resorption of bone.

Osteocyte—mature, functional bone cell found in osteons.

Osteomalacia—softening of bone due to inadequate or delayed mineralization.

Osteomyelitis—inflammation of bone, marrow, and possibly the surrounding tissue usually due to infection.

Osteon—structural unit of mature compact bone.

Periosteum—dense fibrous membrane that covers the exterior surface of bone. Contains nerves, blood vessels, lymphatics, and osteoblasts. Responsible for nourishment and bone growth. Allows for attachment of tendons.

Proliferate—to reproduce or multiply.

Calcitonin—thyroid hormone that promotes the deposit of calcium in bone.

Calcium—chemical element responsible for hard bones and teeth, essential for normal heart, nerve, and muscle function, and is involved in normal blood coagulation (Factor IV).

Parathyroid Hormone—promotes movement of calcium from bone to blood.

Vitamin D—increases calcium in the body by allowing absorption from the GI tract.

average, healthy adult is 5 g) supplements are extremely helpful.

- Loading or placing stress on the bone is thought to produce a small electrical field that stimulates new bone formation.
- Treatment of osteoporosis (a gradual decrease in bone density begins in the late 30s in both men and women—osteoporosis does NOT only affect postmenopausal women).
- Grafting-bone may be taken from the patient (autograft), another human—most likely a cadaver (allograft), or a non-human source (xenograft). Xenograft materials include marine coral (coralline hydroxyapatite) and bovine collagen (collagraft). Xenografts are not actually bone replacements, but are considered scaffolds or structural foundations for natural bone regrowth. The graft must be capable of being included in the new growth and undergoing the remodeling process. The use of a fixation device may be necessary in addition to the graft to stabilize the fracture site.
- Injectable growth factor proteins are under investigational use. Additional amounts of the growth factor proteins, such as morphogenic protein and transforming growth factor-beta, which are normally found in the body, may be capable of encouraging faster and stronger bone healing.
- Bone filler paste is also being evaluated for fracture stabilization. The paste is injected at the fracture site and within 12 hours, the tensile strength of the bone is restored. The paste is reported to stabilize the fracture during healing and undergo the remodeling process.
- Electrical bone growth stimulators and ultrasonic devices stimulate the normal cellular processes at the fracture site. The stimulator may be noninvasive or implantable and may be used alone or in conjunction with open reduction internal fixation (ORIF), external fixation devices, and various grafting techniques. The external electrical stimulation is thought to reproduce the same type of electric force that is naturally created when the bone is loaded.

Many new therapies are on the horizon that will enhance fracture healing by improving the natural course of healing. Some of these techniques will require surgical expertise and others will encourage bone healing, thereby making the patient's post-injury course less painful and shorter in duration.

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Bone healing—normal

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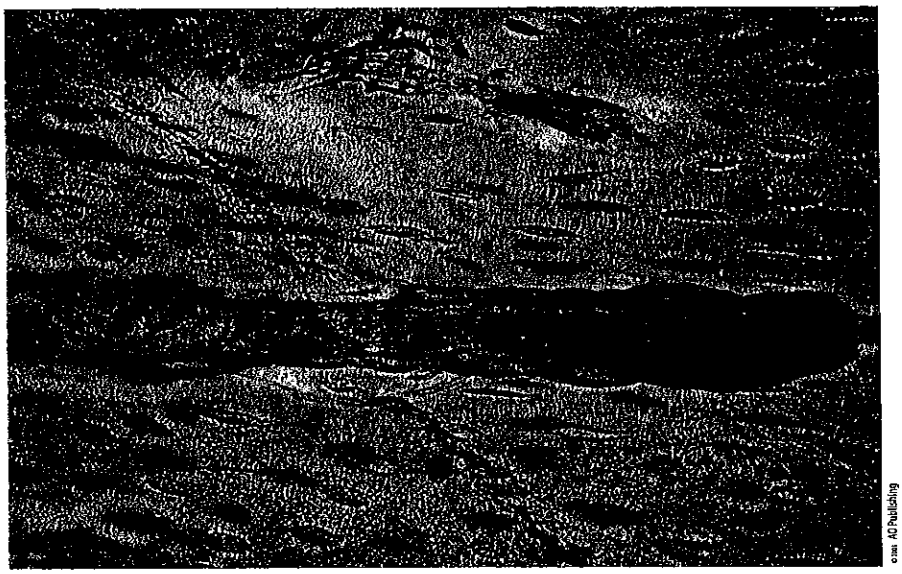
Following a fracture, complete bone healing is expected in 8-12 weeks under optimal circumstances (Figure 1). For complete union of a fracture, the site of injury should be completely immobilized (by means of an internal or external fixation device) and be in proper alignment. Additionally, the patient should be in good general health, well nourished, infection free; and all physiologic mechanisms to facilitate the normal process of bone healing should be intact.

FIGURE 1

Histological appearance of direct cortical bone healing. The fracture line has been graphically enhanced.



The normal process of bone healing involves the following five stages:

1. Inflammation
2. Cellular proliferation
3. Callus formation
4. Ossification Remodeling

The inflammatory stage begins at the time of injury and lasts approximately two days. The fracture hematoma, which is a result of the extravasation of blood caused by the injury, is formed during this time. The blood clot serves as a foundation for the subsequent cellular proliferation stage.

The cellular proliferation stage begins approximately on the second day following the traumatic event. Macrophages debride the area and allow for the formation of a fibrin mesh that seals the approximated edges of the fracture site. The fibrin mesh serves as the foundation for capillary and fibroblastic ingrowth. A soft tissue or periosteal callus is formed on the outer surface or cortex of the fractured bone by the collagen producing fibroblasts and osteoblasts.

The callus formation stage lasts three to four weeks. The soft tissue growth continues and the bone fragments grow toward one another, bridging the gap. Osteoblasts form a matrix of collagen that invades the periosteal callus, bridging the fracture site and uniting the two ends of the bone. Fibrous tissue, cartilage, and immature bone stabilize the fracture site.

The ossification stage begins two to three weeks following the injury and can last three to four months. The matrix of osteoblasts-now called the osteoid-calcifies, firmly uniting the bone. The bone is able to accept mineral deposits.

The remodeling stage is the maintenance state of normal bone. Following a fracture, any devitalized tissue is removed and the new bone is organized to provide maximum support and function. Osteoblastic and osteoclastic activity should be equal, constantly resorbing, and reforming the bone. The process of remodeling continues throughout the life cycle and is affected by local stress on the individual bone, circulation, nutrition, and hormones. Any disruption of the homeostasis will result in a pathologic condition.

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