Bone healing

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 a 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
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.

References
7. Wheeless’ Textbook of Orthopaedics. meddic.com Accessed 1-3-02

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Bone healing

1. Under optimal circumstances, complete healing of a bone fracture takes ___.
   a) 4-6 weeks
   b) 8-12 weeks
   c) 4-6 months
   d) 1 year

2. The inflammatory stage of normal bone healing begins at injury and lasts approximately ___.
   a) two days
   b) seven days
   c) 10 days
   d) two weeks

3. In which stage do osteoblasts form a matrix of collagen that invades the periosteal callus, bridging the fractured bone?
   a) cellular proliferation
   b) callus formation
   c) ossification
   d) remodeling

4. Osteomyelitis is ___.
   a) decreased blood supply
   b) wound contamination
   c) inflammation of bone and marrow
   d) a disrupted vascular system

5. Avascular necrosis can be caused by ___.
   a) steroid use
   b) diabetes
   c) disease
   d) all of the above

6. Which is not a cause of malunion?
   a) inadequate reduction
   b) patient noncompliance
   c) inadequate immobilization
   d) all are causes

7. ___ is softening of the bone due to inadequate or delayed mineralization.
   a) osteomyelitis
   b) osteomalacia
   c) calcitonin
   d) bone coagulation

8. Which type of cell is responsible for the destruction and resorption of bone?
   a) osteoclast
   b) osteoblast
   c) osteocyte
   d) fibroblast

9. ___ allows for the attachment of tendons.
   a) endosteum
   b) parathyroid hormone
   c) calcitonin
   d) periosteum

10. ___ are not actual bone replacements, but are structural "scaffolds" to encourage new growth.
    a) ORIF devices
    b) bone fillers
    c) allografts
    d) xenografts

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