

Chapter 2:
Fracture Healing and Its Histogenesis
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FRACTURE HEALING:

The fractures may be displaced due to force of injury, gravity, or pull by muscles. The fractures usually unite as long as sufficient contact is there. It may heal even if reduction is imperfect, but bone to bone contact is more than 50%. most commonly fractures are splinted not to get union of bone, but to 1) alleviate pain 2) maintain reduction 3) to get early Limb function.

Healing occurs as primary or secondary i.e. healing by callus formation or by direct union

A) Healing by Callus:

There are five stages of fracture healing.

1) Haematoma formation: when fracture occurred, there is haematoma formation due to torn blood vessels in and around bone. Bone is deprived of blood supply and it dies or two millimetres back from bone ends.

2) Inflammatory and cellular proliferation: Inflammatory cells appear in the haematoma with initiation and differentiating of mesenchymal stem cells from periosteum. Inflammatory mediators like cytokines and growth factors are involved resolving the haematoma and new capillaries grow into the affected part.

3) Callus formation: The differentiating stem cells provide chondrogenic and osteogenic cells which starts forming new bone formation. The cell population of osteoclasts derived from new blood vessels causes mop up of dead bone. This cellular mass having islands of immature bone and cartilage is called Callus. When immature bone becomes densely mineralized and fractures usually unites at about 4weeks.

4) Consolidation: With continuing osteoclastic and osteoblastic activity, the newly formed woven bone is transformed into a lamellar bone. This takes several months and fractures must be protected till consolidation.

5) Remodelling: The newly formed bone is reshaped by a continuous process of alternate resorption and formation. Thick lamellar bone is formed where stresses are high. (Wolfs Law)

B) Healing by Direct union:

If fracture is absolutely immobilized either due to impaction or by external or internal metal implant, then there will be no callus formation at fracture site. But osteoblastic new bone formation occurs between bone fracture fragments. The gaps between fracture fragment surfaces were invaded by new capillaries and osteoprogenitor cells which are growing inwards from edges. The new bone is formed on these exposed surfaces. (Gap healing) When the crevices are very narrow i.e. less than 200 nm then ontogenesis produces lamellar bone. And wider gaps are first filled with woven bone which are remodelled with lamellar bone. The bone remodelling units i.e. osteoclast followed by osteoblasts penetrate at 3-4 weeks.

In impacted fractures and fixed with implants, internal bridging may occasionally occur without any intermediate stages (contact healing). In rigid fixation, bone depends entirely upon metal implant for long periods for integrity, so there is absence of callous formation.

CLASSIFYING INJURY:

Patients with open fractures may have multiple injuries. A rapid assessment of patient for open wound and any life threatening injury has to be dealt emergently.

Gustilo's has classified open fractures depending upon type of fractures, soft tissue injury and degree of contamination.

Type-1: the wound is less than 1cm, clean puncture wound through which a bone spike has protruded. No crushing of soft tissues and fracture is not comminuted. (Low energy injury)

Type-2: Wound is more than 1CM, but there is no skin flap. No more than moderate crushing or comminution is there. (Low to moderate injury)

Type-3: There is a large lacerated wound with extensive damage to skin and soft tissues. Contamination is significant. (High energy injury) It is divided into three types:

3A: Fractured bone is adequately covered by soft tissues despite lacerations.

3-B: Extensive periosteal stripping and fracture is not covered.

3-C: this has arterial injury along with extensive soft tissue damage.