Principles of bone and joint injuries and their healing

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Abstract

This article describes the mechanisms of fracture healing (direct and indirect), general fracture management, the influence of the surgeon on the biology and biomechanical environment of bone healing, the management of articular fractures, and disorders of bone union.

Keywords Articular; bone healing; delayed union; fracture callus; non-union; open

Musculoskeletal injuries are common, accounting for 3.5 million attendances to UK emergency departments each year.

Why bones fail

A fracture is a structural failure of bone, which can occur due to a combination of extrinsic and intrinsic factors.

Extrinsic factors: the mechanism of loading will influence the likelihood of failure, due to the:

- magnitude of load
- rate of load
- direction of load.

Bone is **viscoelastic**, which means a time-dependent reduction in stress occurs in bone under constant strain. Therefore the rate of loading affects the extent of injury. Under higher loading speeds, as in a road traffic accident, more energy is absorbed, resulting in far greater damage to the bone and surrounding soft tissue structures (Figure 1).

Cortical bone is **anisotropic**, which means it has different mechanical properties when loaded in different directions. Bone may fail under applied compression, tension, shear or a combination of these forces. It is strongest in compression, weaker under tension and weakest to shear forces.

Loads applied in specific directions and rates will produce predictable patterns of failure. For example, a bone that fractures as it is pulled apart in tension will have a transverse fracture pattern, whereas one subjected to a twisting force will result in a spiral fracture pattern.

Intrinsic factors: bone is a composite made of inorganic minerals and cells surrounded by a large volume of extracellular matrix; mainly type I collagen. It has an outer cortical layer, where the osteons are organized into compact Haversian systems and the bone is strong but brittle; and inner cancellous bone, where the Haversian systems are much less compact and separated by large areas of marrow or fat.

The relative amounts of cortical and cancellous bone determine how bones fail; for example, the calcaneum, which is mainly cancellous bone with very little cortex, often sustains a crush or compression fracture.

Fracture classification

There are a variety of classification systems available. It is important that a classification is easy to use, reproducible, predicts outcome reliably and is clinically useful for guiding management. The AO (**A**rbeitsgemeinschaft für **O**steosynthesefragen – Association for the Study of Internal Fixation) classification is a widely used alphanumeric system of pattern recognition, used to describe individual long bone fractures. A wide variety of other classifications have been devised for describing various specific fractures.

Fractures can be broadly classified according to:

- displacement (displaced or undisplaced)
- whether the skin remains intact (closed or open)
- fracture pattern (transverse, spiral, oblique, simple, segmental or multifragmentary)
- the section of the bone involved (intra-articular, metaphysis, diaphysis)
- cause (traumatic, stress, insufficiency, pathological).

Closed versus open fractures

A closed fracture is one in which the skin is not broken. An open fracture describes an injury where a break in the skin communicates with a fracture and its haematoma. Any fracture with a wound overlying the same limb compartment must be considered open, until proven otherwise. Even the smallest of wounds is enough to allow organisms entry to a fracture site. When a bone clinically appears reasonably well aligned at the time of presentation, it may be difficult to appreciate the degree of fracture displacement that occurred during the initial injury. A bone fragment may potentially have displaced through the skin causing significant crush, stripping and devascularization of the surrounding soft tissues.

Degloving of the soft tissues is a sign of severe soft tissue injuries; it occurs superficial to the deep fascia and may result in poor viability and subsequent necrosis.

Open fractures are classified according to the Gustilo and Anderson classification (Table 1). The main factors that affect outcome are the degree of soft tissue injury and the degree of contamination of the wound.

Fracture healing

Bone differs from other tissues due to its remarkable ability to repair and heal without leaving a scar. Fracture healing is determined by mechanical and biological factors. Adult bone, unlike bone during growth, has very little ability to correct angulation or axial rotation so both must be corrected before the bone unites, or malunion will occur.

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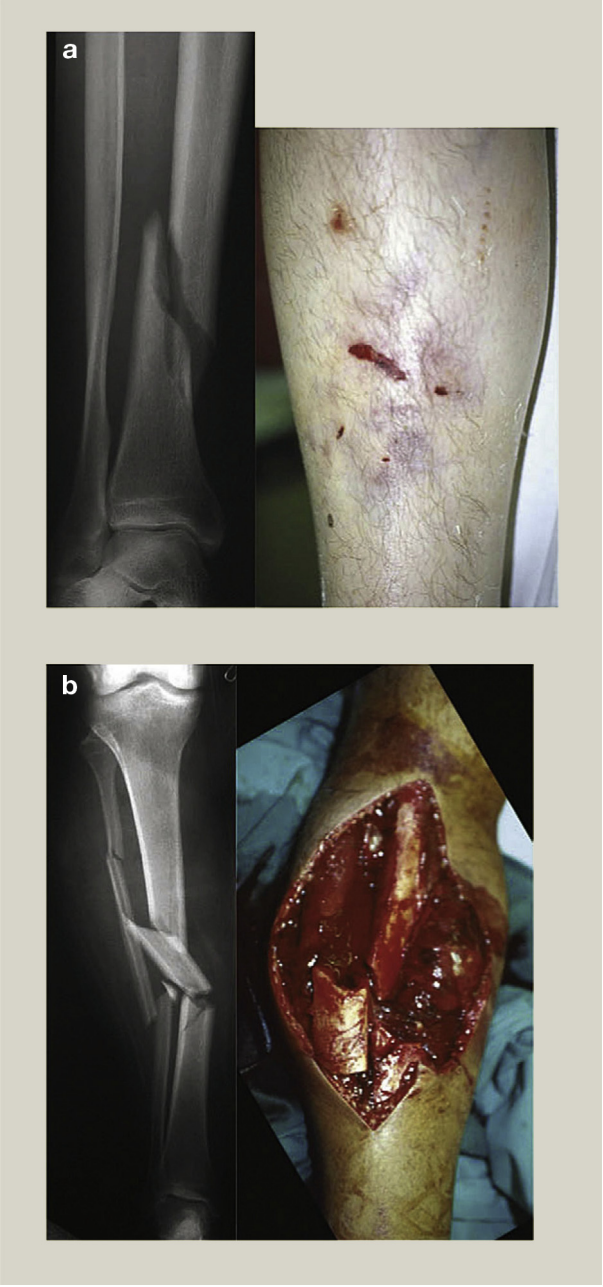

Figure 1 Effect of rate of loading to a limb. (**a**) At a low rate of loading, little energy is dissipated to the surrounding soft tissues. (**b**) At a high loading rate much surrounding soft tissue damage occurs as in this open fracture, shown post-debridement.

Healing of a fracture depends on the blood supply to the bone, the amount of force producing the fracture and the condition of the soft tissues. At the time of injury there is disruption of the Haversian systems and necrosis of osteocytes at the fracture surfaces. The extent of bone cell death depends on the degree of fracture comminution and displacement, disruption of the local blood supply and the extent of periosteal stripping (which affects the cortical blood supply and removes the cambial layer of periosteal stem cells from the surface of the bone). In general, the greater the damage to bone and surrounding soft tissues, the slower the bone is to heal.

The pattern of bone healing can be modified by the mechanical environment of the fracture and this can in turn be manipulated by surgical intervention. The purpose of fracture stabilization is to maximize the biology of fracture healing to aid early union and restore function while minimizing complications.

Direct (primary) healing: direct or primary healing can be achieved through surgery; with **anatomical reduction** of the fracture and application of rigid internal fixation to generate **absolute stability** (no interfragmentary motion under physiological load). This generates a very low interfragmentary strain.

With a fracture gap of less than 200 μm and absolute stability, osteoclasts can tunnel across the fracture line, establishing a 'cutting cone' between the bone ends (Figure 2). Osteoblasts follow, laying down bone matrix and re-establishing continuity between the Haversian systems. Revascularization occurs, with internal bone remodelling, and bone forms directly with no callus formation.

Direct fracture healing with the formation of new cortical bone occurs slowly, and is essentially the same biological process that occurs in normal bone turnover and remodelling.

Indirect (secondary) healing: the majority of fractures heal by indirect healing. This occurs in an environment of **relative stability** (where some controlled interfragmentary motion still exists under functional loading). The stages of repair last several weeks, and are described in three sequential phases (Figure 3), as follows.

- **Reactive phase**
  - ***Inflammation and haematoma formation.*** Bone has an excellent blood supply; the medullary cavity and inner two-thirds of the cortex are supplied centrifugally by endosteal arteries within the bone, whereas the outer third of the cortex is supplied by the periosteal arteries. Bleeding from the bone and surrounding soft tissues results in haematoma formation with a clot of insoluble fibrin at the fracture site.
  - The clot provides a framework of fibrin fibres for the influx of various migrating cells (e.g. neutrophils, lymphocytes, monocytes, macrophages, mast cells, platelets). These cells release various cytokines, including transforming growth factor-β, platelet-derived growth factor, fibroblast growth factor, and interleukins 1 and 6. Histamine is released from mast cells and platelets. This environment encourages an increased capillary permeability, chemotaxis and small vessel dilatation.
  - ***Granulation tissue formation.*** The initial fracture has significant interfragmentary motion, which generates a significant interfragmentary strain. (Strain is defined as the extent of deformation relative to its initial condition, i.e. a relative measure of displacement of particles as a force is applied.) Granulation tissue forms, as it tolerates conditions of high strain (up to 100%), whereas bone cannot form at this stage, as osteoblasts only tolerate very low strain (<1%). The granulation tissue matures, reducing strain across the fracture site. Osteoclasts begin to resorb the dead bone ends and phagocytes remove other necrotic tissue.

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