

Principles of fracture healing

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One must consider why and how bones break, the stages of fracture healing, and the factors that influence these processes in order to understand the principles of fracture healing. Only then is it possible to understand the treatment of these fractures.

Fracture causation

A fracture describes a loss of continuity in the substance of a bone. Fractures in normal bones are usually caused by stress that exceeds the normal limits of tensile or compressive strain. The exact location of the fracture, the nature and direction of the fracture line and the rate of eventual healing process relate to age, the bone involved and the precise mechanism of injury.

Stress fractures: cyclical loading of low intensity (e.g. after marching, marathon running or training for sport) are capable of producing small, but clinically significant, 'stress fractures', usually in the lower limb. The microfractures cannot heal due to the repeated minor trauma. The commonest sites are the tibia and neck of the second metatarsal ('March fracture').

Pathological fractures: a structurally abnormal bone should be suspected if a bone fractures due to stress considered to be within the normal physiological tolerance of that bone. This is called a 'pathological fracture' and is particularly common in patients with osteoporosis, but can occur in other metabolic disorders (e.g. osteomalacia, rickets), Paget's disease of bone, infection or neoplasm. The bone may fracture after trivial injury or spontaneously during normal activity.

Force and fracture patterns: fractures occur due to direct force (e.g. a fracture of the distal phalanx after a hammer blow) or in-direct violence (e.g. a tibia fracture secondary to a twisting injury). The fracture pattern in adults is determined by the magnitude and direction of the applied force. The size of the force determines whether the fracture is:

- complete (both cortices broken as seen on radiograph) or incomplete (one cortex broken)
- displaced or undisplaced
- simple or comminuted (multiple fragments)
- open (fracture communicates with skin surface) or closed.

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The direction of the force influences the pattern of the fracture, and may be transverse, oblique or spiral (Figure 1). The direction of force must be diagnosed from clinical and radiological assessment of the fracture because it must be reversed during manipulative reduction and helps to determine the duration and success of fracture healing. Direct blows and bending forces tend to produce transverse fractures in diaphyseal bone; axial loading results in oblique fractures and torsional forces, causing spiral fractures. The fracture pattern determines the stability of the fracture. Simple transverse fractures have inherent stability to axial loading but not bending. Oblique and spiral fractures are inherently unstable and shortening and displacement is common. Comminuted fractures where there are multiple fragments are also unstable.

Fractures in children and the elderly: fractures are common in children due to accidents and in the elderly due to osteoporosis and falls. Children's bones often break intraperiosteally, with part of the cortical circumference remaining intact: these are 'greenstick' fractures and heal rapidly. Growing bones can sustain injuries to the growth plate that can affect growth. Elderly patients with reduced bone mass secondary to osteoporosis, and who fall frequently, are susceptible to 'insufficiency fractures'; this means the force that caused the fracture would not be expected to fracture a normal bone (e.g. a fracture of the neck of femur or a distal radial fracture due to a simple fall from a standing height). Osteoporosis can cause spontaneous vertebral body crush fractures and it is very common for elderly patients to state that they have 'lost height'.

Open versus closed fractures

An open fracture is one in which a break in the skin and underlying soft tissues leads directly to, or communicates with, the fracture and its haematoma. The term 'compound fracture' refers to the same injury and should not be used. A closed fracture is one in which the skin is not broken.

Diagnosis of an open fracture can be difficult because the wound may be far from the fracture site, but the fracture must be considered to be open until proven otherwise if a wound occurs in the same limb segment as a fracture. An open fracture has many consequences:

- bacterial contamination from the external environment
- crushing, stripping and devascularization of soft tissues render the soft tissues and the underlying bone more susceptible to the contaminating bacteria
- the destruction or loss of the normal soft tissue envelope that normally surrounds the bone may affect the way in which the fracture can be immobilized, may result in delayed healing of the fracture, and there may be direct loss of function due to damage to muscles, tendons, nerves, vessels and skin.

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 (Figure 2).

Fracture management

Fracture management consists of initial assessment of the patient, then the limb or fracture, and finally treatment of the fracture.

Fracture patterns

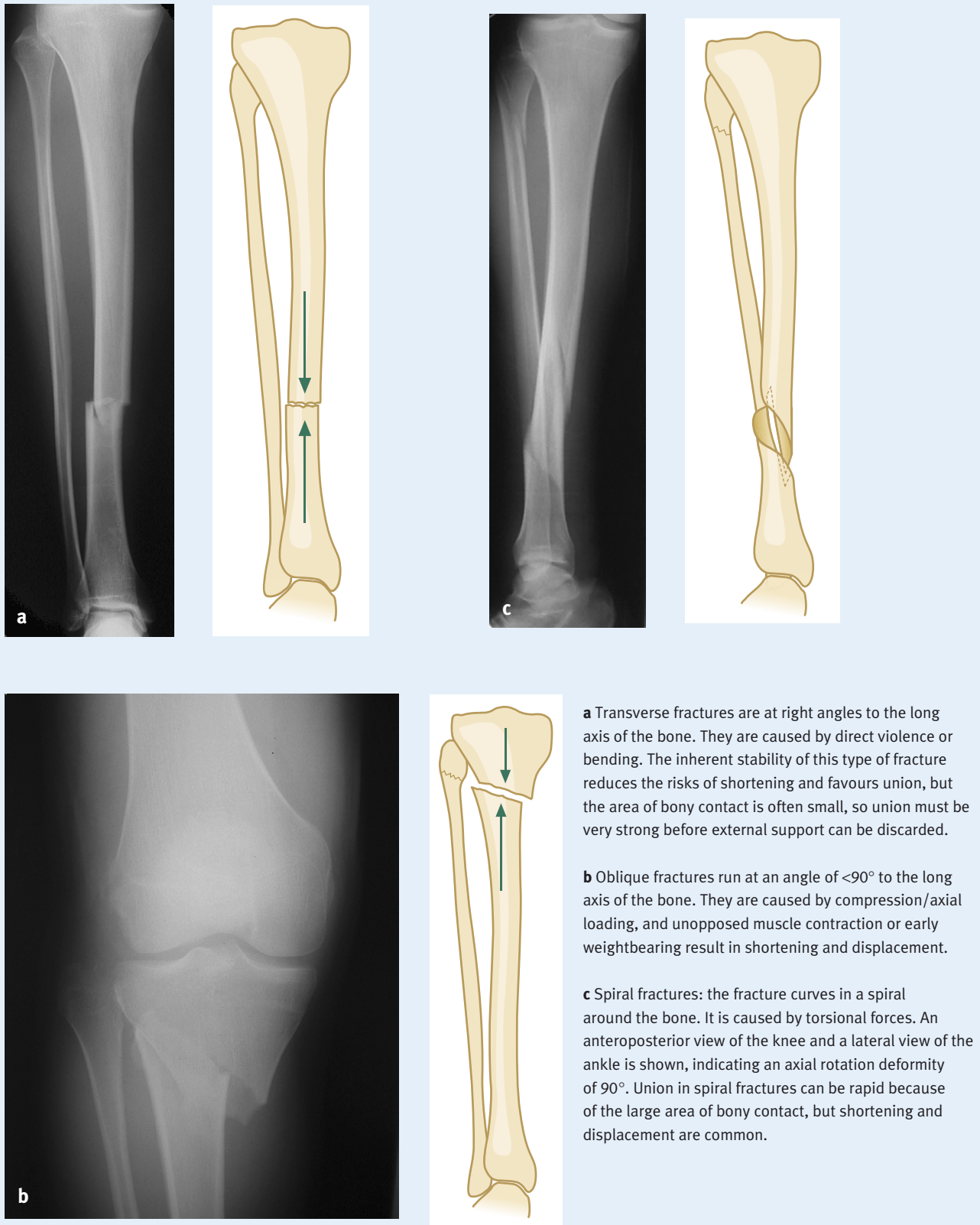


Figure 1

Classification of open fractures

Type	Wound	Level of contamination	Soft tissue injury	Bone injury
I	<1 cm long	Clean	Minimal	Simple, minimal comminution
II	>1 cm long	Moderate	Moderate, some muscle damage	Moderate comminution
III*				
A	Usually >10 cm long	High	Severe, with crushing	Usually comminuted soft tissue coverage of bone possible
B	Usually >10 cm long	High	Very severe loss of coverage	Bone coverage poor; usually requires reconstructive surgery of soft tissue
C	Usually >10 cm long	High	Very severe loss of coverage plus vascular injury requiring repair	Bone coverage poor; usually requires reconstructive surgery of soft tissue

*Segmental fractures, farmyard injuries, fractures occurring in a highly contaminated environment, shotgun wounds, or high-velocity gunshot wounds are automatically classified as type III open fractures.

Table 1

Initial assessment and management: the Advanced Trauma Life Support™ protocol of airway, breathing and circulation must be applied to all patients. History-taking determines the magnitude and direction of the force. All wounds near a fracture must be assumed to communicate with the fracture and must be treated as open fractures. Antibiotics must be given as soon as an open fracture is suspected and the wound covered with a sterile dressing until formal exploration and debridement can take place in the operating theatre. A cephalosporin should be given (i.v.) for open fractures, adding an aminoglycoside (i.v.) for open fractures with a high level of contamination. High-dose penicillin (i.v.) is added for gross contamination (e.g. farmyard and clostridial infections). Antibiotics are continued for 24 hours after the last debridement or until the bone is covered by soft tissue. The status of tetanus vaccination must be considered for open fractures.



Figure 2 Open fracture. This is the result of a high-velocity motorcycle crash. There is a grade III B open fracture of the mid-shaft of the right tibia, a wound across the knee and a degloving injury to the sole.

The principles of debridement include wound extension to see the extent of the injury, removing all devitalized bone and tissue, followed by at least six litres of irrigation. Patients with highly contaminated wounds and severely damaged soft tissue should return to theatre every 48 hours until the wound is clean and only healthy soft tissues remain. Exposed bone requires soft tissue coverage with a rotational or free flap as soon as possible (usually within five days). The infection rates with appropriate initial care are 5–30%.

The state of the soft tissues surrounding the fracture dictate management, even in closed injuries. Do not operate through bruised and highly swollen tissues where the wound may be impossible to close or break down later. Wait until the swelling and bruising have improved, even if this means a delay of several days (Figure 3).

Clinical examination must include assessment of the neurovascular status of the limb, particularly distal to the fracture. Radiographs should include the whole of the fractured limb and the joint above and below the fracture. Monteggia fracture dislocations (where a fracture of the ulna is associated with a dislocation of the radial head at the proximal radioulnar joint) are often overlooked.



Figure 3 Closed fracture with severe bruising. This fracture of the tibial plateau occurred four days ago, but the degree of skin blistering and bruising indicate that it is too early to consider open reduction.

Fracture treatment involves reduction (if necessary), stabilization and rehabilitation. The Royal College of Surgeons of England uses the mnemonic RIP (**r**eduction, **i**mmobilization, **p**hysiotherapy).

Reduction is required if there is significant displacement, angular deformity or rotation that is likely to result in functional disability if left to heal in that position. For example, angular deformity in the forearm due to mid shaft fractures of the radius and ulna must be corrected; otherwise, pronation and supination will be restricted, resulting in significant impairment of function. Axial rotational deformity must be corrected to allow normal function of limbs (e.g. an internal rotation deformity of the distal tibia causes the patient to trip over the affected foot).

A fracture is considered stable if it maintains its position when treated by simple conservative methods (e.g. plaster cast). Unstable fractures can be treated by non-surgical methods (e.g. traction, plaster casts), but often require internal (e.g. plate and screws, intramedullary nail) or external fixation devices. This often allows earlier mobilization of joints and the precise reduction and fixation required for intra-articular fractures. Plaster casts can control only angulation and do not prevent shortening. Moulding a well-padded cast helps to prevent redisplacement of the fracture. The

biology of fracture healing must be considered because fracture stabilization can interfere with normal repair processes.

Bone healing

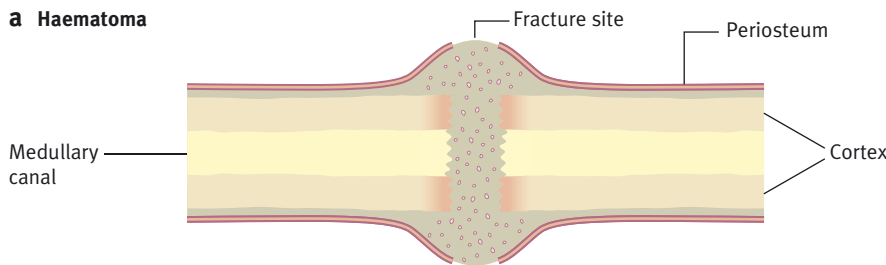
Bone differs from other musculoskeletal tissue due to its ability to repair itself and heal without leaving a scar. The processes involved depend on the biomechanical stability and biological environment of the fracture. Direct bony union or primary fracture healing occurs when there is absolute stability (no motion between fracture surfaces under functional load), as found with anatomical reduction and rigid internal fixation. Secondary fracture healing occurs when there is relative stability (some controlled motion between fracture surfaces under functional load), for example fractures treated in a plaster cast or by external fixation. Secondary fracture healing is by far the commonest type of healing.

Secondary fracture healing

Secondary fracture healing occurs in four overlapping phases (Figure 4).

Secondary fracture healing: bridging of a fracture by external callus

a Haematoma

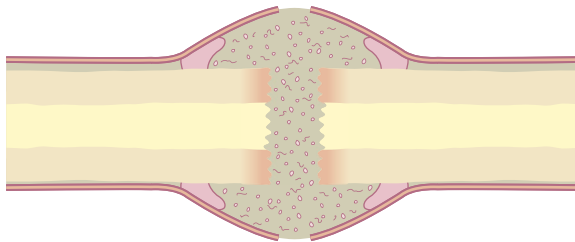


Haematoma forms at the fracture site and the periosteum is torn. The bone ends die and are resorbed by osteoclasts.

Haematoma

Dead bone at fracture site

b Inflammation

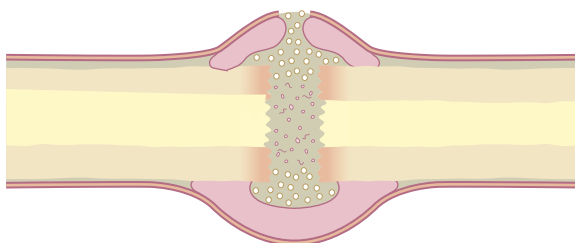


Granulation tissue replaces the haematoma and woven bone or hard callus starts to form the abutments of the bridge from the cambium layer of the periosteum by intramembranous ossification.

Granulation tissue

New woven bone/external hard callus

c Repair



The fracture gap is bridged by soft callus or cartilage. This is replaced with bone by the process of endochondral ossification. The gap is also bridged by hard external callus arching over the soft cartilaginous callus as shown in the lower half of the diagram. Internal or medullary callus forms more slowly and finally cortical continuity is restored.

Cartilage/soft callus

Figure 4

Haematoma: bleeding occurs from the medullary cavity and the periosteum as well as tearing of soft tissue and adjacent muscle, particularly on the convex side of the fracture. Damage to neighbouring blood vessels may occur; the combined result is the formation of a fracture haematoma.

There is disruption of the Haversian systems with death of osteocytes at the fracture surface. The extent of bone cell death varies with the:

- degree of fracture comminution and displacement
- amount of periosteal stripping
- extent of injury to the medullary contents.

In general, the greater the bone damage, the slower the fracture healing.

Inflammation: the fibrin mesh within the haematoma forms a framework for the influx of various migrating cells (e.g. neutrophils, lymphocytes, monocytes, macrophages, mast cells, platelets). The haematoma organizes over the next week into granulation tissue. The inflammatory cells and platelets release various cytokines: transforming growth factor- β , platelet-derived growth factor, fibroblast growth factor, and interleukin-1 and -6. These activate cell migration, proliferation and differentiation of osteoprogenitor cells, leading to repair. Inflammatory mediators increase vascular permeability, causing an exudate of plasma and promotion of phagocytosis of necrotic material. Angiogenesis occurs as the fracture haematoma is rapidly organized and osteoclasts start to resorb the dead bone ends.

Bone is formed by osteoblasts that function best at very low strain. (Strain is the change in length of a material when a given force is applied.) Osteoblasts cannot tolerate strains of $>1\%$. Immediately after a fracture, the area between the bone ends has a strain of $>100\%$, so bone cannot form. Initial healing is via granulation tissue, which can tolerate very high strains.

Repair: as the granulation tissue matures, it reduces strain at the fracture site as it increases in size. Cartilage forms when strains are $<10\%$, and this is eventually replaced by bone when strains are $<1\%$.

Angiogenesis and an intact periosteum are very important in the formation of fracture callus. The term 'hard callus' refers to woven bone that is ossified and therefore visible on radiographs, 'soft callus' refers to a cartilaginous phase before differentiation into hard callus. The soft callus is replaced by the process of endochondral ossification into woven bone. External callus forms on the outside of the fractured bone to bridge the gap. Internal callus forms more slowly from the medullary canal. Finally, the cortical continuity is restored.

Osteoblasts beneath the periosteum (cambium layer) deposit a subperiosteal layer of woven bone at the fracture site. This woven bone forms the abutments of the bridging external callus by the process of intramembranous ossification with no intermediate cartilaginous phase. In embryonic skeletal development, the flat bones of the skull, pelvis and clavicle also form by intramembranous ossification.

The initial haematoma matures into granulation tissue at the fracture gap. Soft callus or cartilage then forms, and finally the soft callus is replaced by endochondral ossification to become hard callus or woven bone. The chondrocytes become hypertrophic, calcify and die. Angiogenesis occurs and osteoblasts

lay down bone matrix on the collagen framework left by the chondrocytes.

External bridging callus is under the control of humoral and mechanical influences and is reliant on viable adjacent soft tissues. Cyclical micro-movements stimulate the growth of cartilage and then bone. The optimum size of these movements is about 1 mm. The increasing size and stiffness of the callus reduces movement and facilitates the conversion of cartilage at the fracture site into woven bone. Formation of an external bridging callus is the predominant form of healing when a simple fracture is treated by a sling or plaster-cast immobilization, external fixation or intramedullary nailing.

Late medullary callus ('internal callus') is formed mainly within the medullary cavity at the fracture site. The capacity of the medulla to form new bone occurs more slowly and forms later.

Remodelling may continue long after the fracture has clinically healed (up to seven years), but eventually there is consolidation and remodelling of the woven bone 'osteoid' into lamellar bone and reconstitution of the medullary canal and restoration of the bone shape.

Excess callus is formed in the early stages of callus formation. The callus is distributed preferentially on the concave surface of the fracture if the bone is not aligned. Remodelling then occurs, with the osteoblasts and osteoclasts working in unison. The shape is regained based on stresses to which the bone is exposed; bone is laid down in areas of excess stress and removed from areas where there is too little ('Wolff's law'). Bone is resorbed by osteoclasts in areas of bone with too little stress. Under physiological stresses, woven bone is replaced with lamellar bone and the medullary cavity is also restored.

In children, remodelling is so effective that even completely displaced fractures may heal and remodel without trace. The younger the child, the greater is the propensity for remodelling. As well as increased cellular activity, the physis of a bent bone grows eccentrically to help restore the alignment and growth in bone length and width to conceal the deformity. There is some ability to correct angulation, although this decreases as the child approaches adolescence. Axial malrotation must not be accepted because it will probably remain.

In adults, there is very little correction of angulation or axial rotation. Axial rotation deformity must be corrected and angulation, particularly in the adult or older child, must be corrected before bone healing.

Primary fracture healing

In primary fracture healing, there is absolute stability, and no motion occurs between the fracture surfaces under functional load. Anatomical reduction results in maximal friction, and internal fixation by interfragmentary compression with a lag screw and plate prevents motion. Under these conditions, strain is very low and there is no functional requirement for external bridging callus, which is minimal or absent.

With a fracture gap between the bone ends of $<200\ \mu\text{m}$, osteoclasts can tunnel across the fracture line, and establish a 'cutting cone' across the fracture (Figure 5). Osteoblasts follow and lay down bone matrix and re-establish continuity between the Haversian systems. Vessel ingrowth is absent and the bone

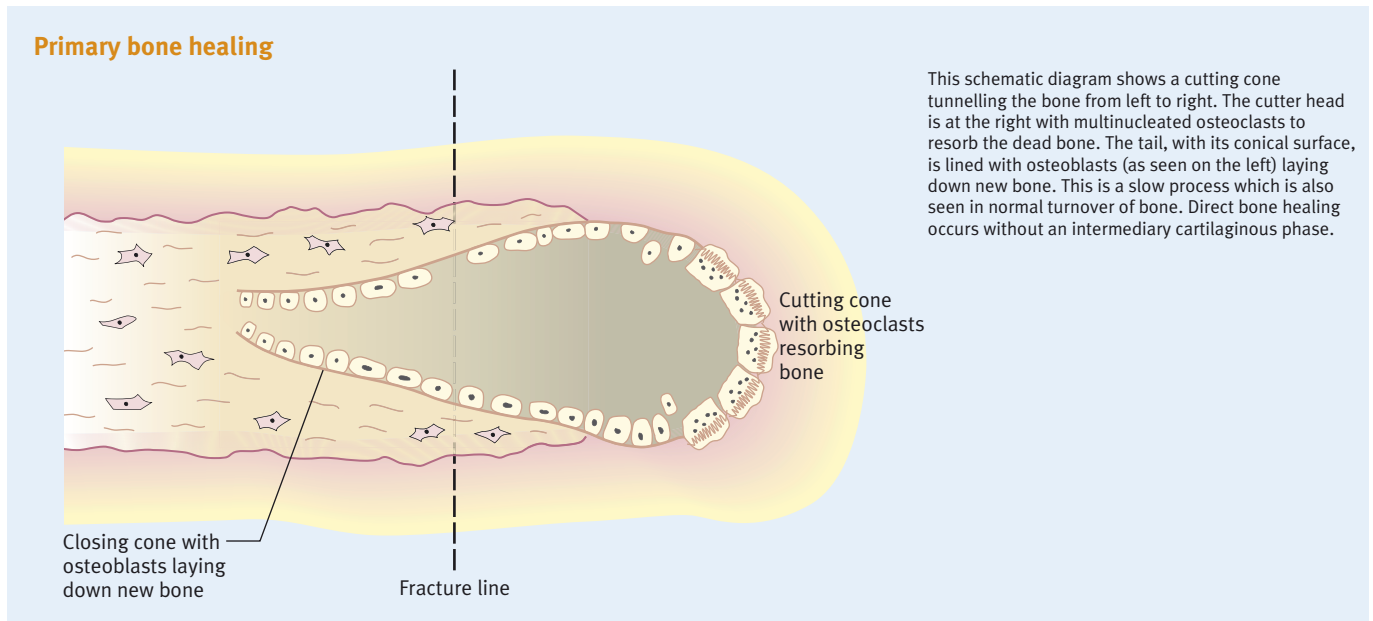


Figure 5

filling the interfracture gap appears without the intermediate formation of cartilage or connective tissue. This process cannot occur if the fracture ends are mobile or if there is interposition of soft tissue between the bone ends.

Fracture healing with the formation of new cortical bone between the bone ends occurs slowly and is essentially the same biological process as occurs in normal bone turnover and late remodelling. The internal fixation must be maintained until this healing process is complete.

Healing with different methods of stabilization

The purpose of fracture stabilization is to utilize the mechanisms of fracture healing outlined above to unite a fracture rapidly while minimizing complications and obtaining good functional outcome. Fracture healing varies with the method of treatment. The amount of callus formation is inversely proportional to the extent of immobilization of the fracture.

Treatment involves reduction (if necessary), stabilization and subsequent rehabilitation in fractures of long bones, and can be achieved by conservative or surgical methods.

Plaster casts prevent angulation and malrotation. Only transverse fractures have axial stability in a cast and oblique fractures may displace and shorten. A cast provides relative stability with some controlled motion between the fracture surfaces under functional load. Fracture callus is seen on radiographs and secondary fracture healing occurs.

Traction is considered old-fashioned, but is a safe way of maintaining reduction while allowing axial micro-movement to encourage callus formation.

Intramedullary nails prevent angulation and provide axial stability. They also provide rotational stability if locking screws are

used. Healing is by callus formation if there is a small fracture gap, but primary bone healing can occur if the bone ends are anatomically reduced and compressed. There is always a small amount of micro-motion with an intramedullary nail, so external bridging callus is seen on the radiograph (Figure 6a).

Unilateral external fixation is particularly useful if the soft tissue injury precludes internal fixation. All external fixators allow micro-motion and some are designed to allow about 1 mm of movement to encourage early formation of callus and careful early weightbearing.

Circular frames are particularly useful if the fracture is very close to a joint and associated soft tissue injuries preclude internal fixation. They provide stability in three planes and allow axial micro-movement to encourage callus formation.

Internal fixation with absolute stability: an anatomical reduction allows maximal friction at the fracture site. Absolute stability is achieved if combined with interfracture compression to prevent motion. Absolute stability occurs when there is no motion between the fracture surfaces under functional load i.e. there is very low strain across the fracture and primary bone healing occurs without formation of an external callus (Figure 6b). This is a slow process that relies on internal remodelling of the bone. Interfracture compression can be achieved with a lag screw across the fracture or a special plate that causes compression as the screws are tightened, or both combined.

Anatomical reduction is required in two special situations:

- the forearm when the radius and ulna are fractured and displaced (pronation and supination will be reduced unless anatomical reduction is achieved; the bones are held reduced with a lag screw and plate)
- intra-articular fractures to reconstruct the articular surface.

Examples of primary and secondary bone healing

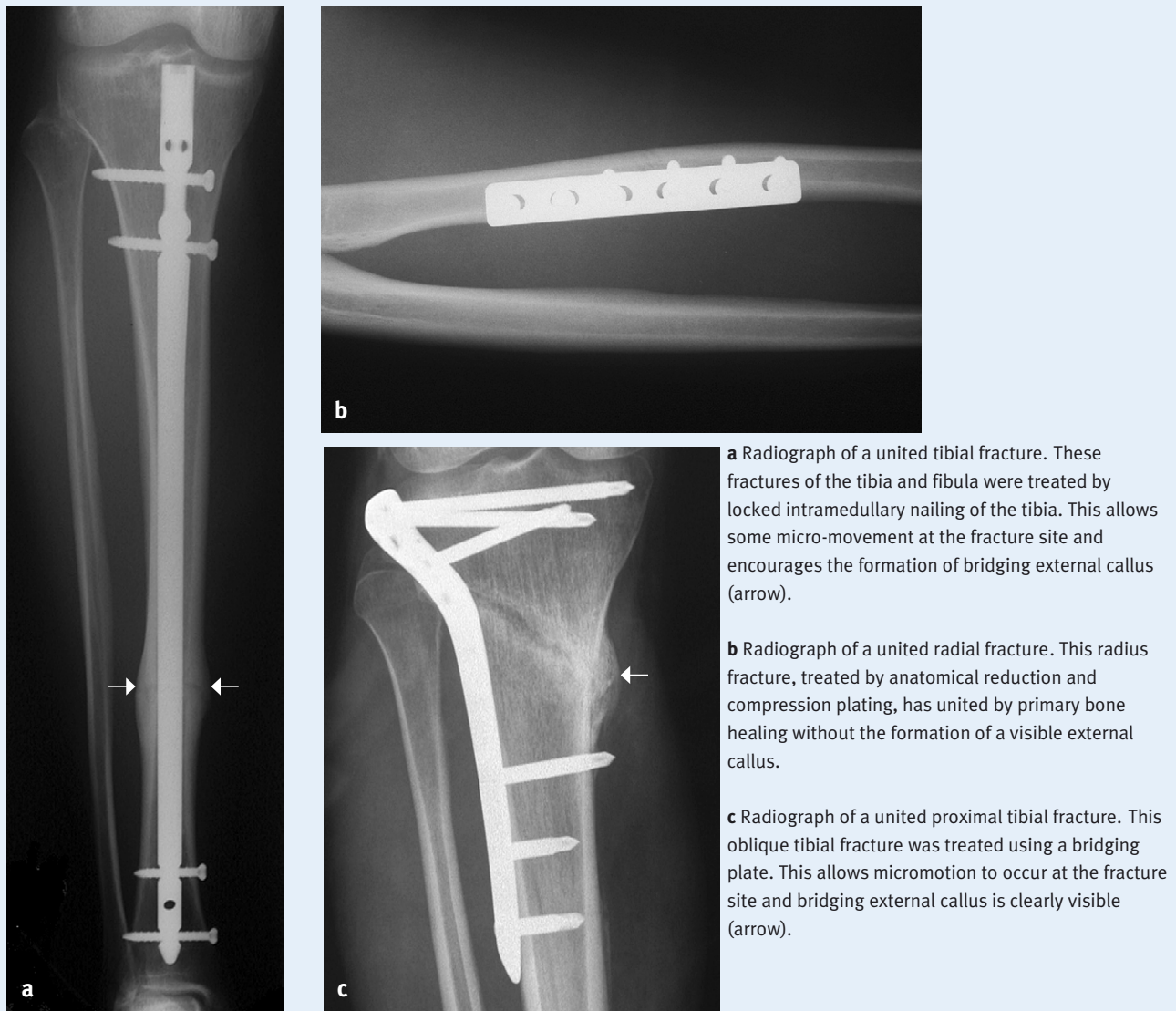


Figure 6

There are different methods of treating the same fracture, but an understanding of the type of healing you wish to achieve avoids adverse outcomes. An example of poor treatment is rigid internal fixation of a diaphyseal fracture with damage to surrounding soft tissues, and periosteal stripping without achieving compression and leaving separation between the bone ends. This prevents primary bone healing because of separation of the bone ends, and inhibits external bridging callus because of the absolute stability. The result is non-union of the fracture and risk of implant failure.

Stability can be further improved by screws that lock via a thread onto the plate ('locking plate'). This can be useful in biomechanically weaker bone such as in metaphyseal fractures or osteoporotic bone. Anatomical reduction and compression across the fracture site must be achieved, otherwise the locking plate holds the fracture ends apart; primary bone healing will

not occur, and neither will secondary bone healing due to the absolute stability.

Plate fixation with relative stability: relative stability permits some controlled motion between the fracture surfaces under functional load, encouraging callus formation. Plates have many functions:

- a protection plate reduces the load placed upon a lag screw
- a buttress plate resists axial load by applying a force at 90° to the potential deformity
- a compression plate presses together the bone fragments to increase stability
- a plate on the tensile surface of the bone resists tensile force and dynamically compresses the far cortex
- a bridging plate fixes the two main fragments, leaving the fracture zone undisturbed ('biological plating'; Figure 6c).

Factors affecting the rate of fracture healing

General factors

Age: fractures in children unite more rapidly; the speed decreases as skeletal maturity approaches. A femur may be expected to unite in:

- four weeks in a three-year-old child
- at nine weeks for an eight-year-old child
- 3–6 months in an adult.

This is due to greater activity of bone and often more minor fractures with an intact periosteal sleeve in children. Children's bones also have great capacity for remodelling (except for axial rotation).

Nutrition and drug therapy: nutritional status and general health influence the healing of bone and soft tissue. Many drugs adversely affect fracture healing. Corticosteroids and NSAIDs impair the inflammatory response and delay bony union.

Bone pathology: pre-existing abnormal bone at the fracture site (pathological fracture) may be genetically determined (e.g. osteogenesis imperfecta) or the result of acquired conditions (e.g. malignant disease, metabolic disease of bone). A fracture through a malignant deposit is unlikely to heal and requires stabilization with a sturdy device to resist the cyclical loading imposed by the non-united fracture.

The type of bone involved in the fracture influences the rate of union. Cancellous bone tends to heal more rapidly than cortical bone and is generally well advanced at six weeks, when stabilization of the fracture can be abandoned (e.g. removal of the plaster cast after wrist fracture). This is due to the large area of bony contact and the greater number of active bone cells that are present. Union of cancellous bone occurs with little external callus, particularly if the two fragments impact into each other.

Cortical bone heals via endosteal (medullary) callus that may take many months to become well established (e.g. tibial fractures treated conservatively take about 16–20 weeks to unite).

Local factors

Mobility at the fracture site: excess movement at the fracture site may interfere with vascularization of the fracture haematoma, cause high strains and may disrupt early bridging callus and prevent endosteal new bone growth. This may result in delayed or non-union and a fibrocartilage envelope can develop. One of the primary aims of external or internal splintage is to reduce the mobility and thus strain at the fracture site and hence encourage union.

Separation of the bone ends: bony union may be delayed or prevented if the bone ends are separated. This may be the result of bone loss or resorption at the fracture site, soft tissue interposition between the bone ends, excessive traction or after surgical intervention if the bone ends are held apart by internal fixation.

Disturbance of blood supply: a fundamental factor affecting healing is blood flow in the bone. A fracture through the neck of femur may interrupt the normal blood supply to the femoral head, resulting in avascular necrosis. Surgical treatment, such

as excision of the head and replacement with a metallic prosthesis (hemiarthroplasty), is often the first-line treatment in the elderly.

With injury of the talar neck or waist of scaphoid, a fracture may disrupt the blood supply to one side of the fracture. The devitalized side of the fracture acts as a scaffold for fracture union and is osteoconductive and osteoinductive, but does not contribute actively to bony union by providing osteogenic precursor cells. These fractures may eventually unite slowly if adequate stabilization is achieved.

High-energy comminuted fractures and fractures involving a disturbance of soft tissue attachment and periosteal stripping heal more slowly. In uninjured long bones, the medullary canal, cancellous bone and inner two-thirds of the cortex receive their blood supply centrifugally from within the medullary canal from a central nutrient artery, and the outer one-third of the cortex receives its blood supply from the periosteum and surrounding soft tissues. In fractures, the blood supply from surrounding soft tissues is very important in forming granulation tissue and bridging external callus. Fractures of long bones treated by intramedullary nailing usually unite extremely well with external callus formation.

A segmental or double fracture (where the bone is fractured at two distinct levels) heals slowly for two reasons. The intervening bone fragment may be devitalized; a compromise in the optimal biomechanical environment is likely as the two sites attempt to unite simultaneously.

Property of bone involved: there is a variation in the speed at which bones heal in the same individual. Fractures of the upper limb generally heal more quickly than fractures of the lower limb. The clavicle has remarkable healing powers and non-union is extremely rare despite the inability to effectively control movement at the fracture site. Fractures of the tibial shaft tend to unite more slowly than fractures of the femoral shaft probably because of a poor intrinsic blood supply and relatively poor soft tissue cover.

Type of fracture: displaced and comminuted fractures frequently result in delayed healing. The avascular fragments of splintered bone require resorption, a more extensive inflammatory and callus phase, and more time to remodel.

Transverse fractures take longer to heal than spiral fractures because they usually have more displacement of the periosteum and a smaller surface area of contact.

Infection in the region of the fracture can delay or prevent union by directing cellular activity from the process of bony union to combat the infection. Infection results in a prolonged inflammatory phase and is a common cause of delayed or non-union. Infection is a particular problem if associated with internal fixation because it can be difficult to eradicate from the metalwork.

Biomechanical environment: there is an optimal balance between stability and micro-motion to encourage callus formation. Small, cyclical movements of about 1 mm increase the rate of healing by 25% and are a feature of many external fixation devices.

Electromagnetic environment: stressed bone produces electrical current and these local currents, as well as magnetic fields,

have been postulated to improve bone healing. Direct current helps stimulate an inflammatory-type response and pulsed electromagnetic fields initiate calcification of fibrocartilage. Their therapeutic use is controversial.

Ultrasound: low-intensity pulsed ultrasound accelerates fracture healing and improves mechanical strength by increasing the stiffness and torque of fracture callus. This is thought to be the result of transfer of mechanical energy to the cells by the ultrasound signal.

Biochemical environment: circulating hormones influence the rate and quality of healing. Nicotine from smoking increases the time to fracture union and the risk of non-union, and weakens callus formation. Smoking also has an adverse effect on oxygenation of soft tissue and wound healing.

High-dose irradiation is associated with a decrease in cellularity, long-term changes within the Haversian system and an increased risk of non-union.

Complications

One must consider potential complications of the injury as well as complications of the fracture and its management.

General complications

General complications include hypovolaemic shock, adult respiratory distress syndrome, fat emboli and venous and pulmonary emboli.

Early local complications include:

- injury to vessels and nerves
- injury to soft tissue
- compartment syndrome
- injury to associated joints
- infection.

Compartment syndrome is a serious complication that can result in loss of the limb. It can occur in low-velocity injuries (e.g. football injury), where the fascial compartments stay intact. Soft tissue injury and bleeding into a fascial compartment results in the tissue pressure exceeding the capillary pressure, leading to muscle ischaemia. The cardinal sign is increasing pain made worse by passively stretching the affected muscle; the commonest site is the tibia. Constricting splints and plaster casts must be removed and the limb is placed on a pillow in line with the level of the heart to optimize blood inflow. An urgent surgical fasciotomy must be done if this does not result in immediate relief of pain.

Late local complications include:

- malunion
- delayed union and non-union

- avascular necrosis of bone
- ischaemic contractures
- joint stiffness and myositis ossificans
- poor range of motion and functional disability
- reflex sympathetic dystrophy
- growth disturbance
- osteoarthritis
- pressure sores.

Slow, delayed and non-union: some fractures are slow to unite or fail to heal completely despite optimal treatment. In slow union, the fracture proceeds through the normal stages of healing clinically and radiologically, albeit at a slower rate.

In delayed union, healing fails to occur within the expected time; it ceases to be active in non-union. Non-union occurs if there is wide separation of the bone ends due to bone loss or muscle interposition, lack of blood supply, and/or an adverse biomechanical environment. Infection after open fractures can also result in delayed and non-union. Radiographically, the non-union may be described as 'atrophic' or 'hypertrophic'. In atrophic non-union, there is no attempt at healing, the bone ends are resorbed and rounded; the biology is faulty and bone grafting plus internal fixation are required.

Inadequate stability of the fracture leads to hypertrophic non-union, with normally viable bone ends that appear sclerotic and flared giving the typical appearance of 'elephant's feet'. The gap between the bone ends is filled with cartilage and fibrous tissue. The biomechanical environment is faulty, with too much movement preventing the fracture from healing. The blood supply at the bone ends is good and the cartilaginous and fibrous tissue will mineralize and be converted to bone without the need to disturb the bone ends. Immobilizing the fracture (e.g. with an intramedullary nail) usually results in rapid union.

Local bone mass can be improved by fracture reduction, bone grafting or bone transport. Bone activity can be enhanced by bone morphogens with cancellous bone grafting or indirectly by intramedullary reaming. Exogenous human recombinant bone morphogenic proteins are currently being evaluated for the treatment of non-unions, segmental fractures and avascular necrosis. ♦

FURTHER READING

- Burnand KG, Young AE, eds. The new Aird's companion to surgical studies, 2nd edn. Edinburgh: Churchill Livingstone, 1999.
- McRae R, Esser M, eds. Practical fracture management, 4th edn. Edinburgh: Churchill Livingstone, 2002.