A Guide to Canine and Feline Orthopaedic Surgery

Fourth Edition

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Preface

A Guide to Canine and Feline Orthopaedic Surgery was first published in 1980 as a rapid reference guide for veterinary students and busy practitioners trying to keep pace with current trends in small animal orthopaedic surgery. Advances continue to be made in orthopaedics and the book has been regularly updated, the second edition was published in 1985 and the third in 1993. The fourth edition has been written by Hamish R. Denny and Steven J. Butterworth. This latest edition retains the same practical approach but has been completely rewritten to provide a comprehensive review of orthopaedic and spinal conditions in the dog and cat. The illustrations have also undergone a major overhaul and the many line drawings are now combined with photographs and radiographs to clarify diagnosis and surgical technique.

Although the size of the book has been considerably increased when compared with previous editions, its regional approach to problems should still enable the reader to use it as a rapid reference guide. The book should allow veterinary practitioners to diagnose and treat most orthopaedic and spinal problems encountered in general practice, while postgraduate students taking further qualifications in orthopaedics will find a sound basis for their studies and further reading provided here.

The authors gratefully acknowledge the help of their colleagues both at Bristol University and in practice who have made this book possible.

Section 1
General

Chapter 1

Fracture Healing

Normal biology of bone

Bone is a living system with a number of functions besides that of providing a framework on which muscles can act. The skeleton also protects vital organs and houses bone marrow, which is essential for the production of cells for the haematopoietic and immune systems. Owing to its mineral content, it plays a role in calcium homeostasis, though in the normal animal this is relatively insignificant in comparison to the role of the kidneys and intestines in regulating serum calcium levels. The cells within bone include osteoblasts, osteoclasts and osteocytes. Osteoblasts are of mesenchymal origin and are important in the synthesis and mineralisation of matrix, in the initiation of bone resorption, and communication with osteocytes. Osteoclasts are derived from the monocyte–macrophage system and are involved with remodelling and resorption of bone. Osteocytes are osteoblasts that have become trapped within compact bone, residing within lacunae and maintaining contact with neighbouring cells via canaliculi. Their function is unknown but it is speculated that they are involved with calcium homeostasis since they have the ability to mobilise calcium from the lacunar borders which, when considering the whole skeleton, provide a massive surface area from which physiological amounts of calcium could easily be liberated without significant alteration to the structural integrity of the bone.

Development and growth of bone

Apart from some of the flat bones of the skull, the skeleton develops first as a cartilage model which is then replaced with bone by a process of endochondral ossification. This begins *in utero* with the development of primary centres of ossification in the diaphyses and secondary centres in the epiphyses and some other sites (for example the anconeal process and greater trochanter). The process is incomplete at birth resulting in radiographs of the limbs of patients a few weeks old showing large spaces between very round-ended 'bones' because of incomplete mineralisation of the cartilage model (Fig. 1.1). By about 5 months of age most of the cartilage model has been converted into bone and the only remaining endochondral ossification to take place is within the physes and deeper layers of the articular cartilage. By this age the general anatomy of each long bone is such that an epiphysis at each end, supporting articular cartilage, is separated from the metaphysis by a physis. The diaphysis constitutes the longest part of the bone, between the metaphyses (Fig. 1.2). Apart from where articular cartilage is present, or at sites of tendon attachment, the external surface of the bone is covered by periosteum whilst internally all surfaces are covered by endosteum.

Growth of the bone until skeletal maturity is reached involves two main processes. Firstly, the girth of the diaphysis is increased by *appositional growth* whereby new bone is deposited by periosteal osteoblasts and resorbed from the endosteal surface by osteoclastic activity. Secondly, the diaphyseal length and epiphyseal size are increased by the process of *endochondral ossification* within the physis and deeper layers of articular cartilage respectively. This process is most easily understood by consideration of events within a physis which can be divided, histologically, into several zones (Fig. 1.3).

Closest to the epiphysis is a *resting zone* in which reside relatively inactive chondrocytes

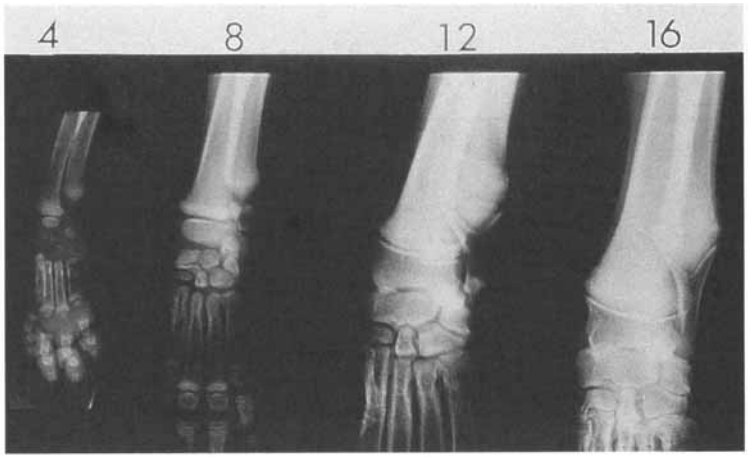

Fig. 1.1 Serial radiographs of the carpus of a puppy taken at 4, 8, 12 and 16 weeks of age showing incomplete mineralisation of the bones with large radiolucent gaps between them initially. (Courtesy C. Gibbs.)

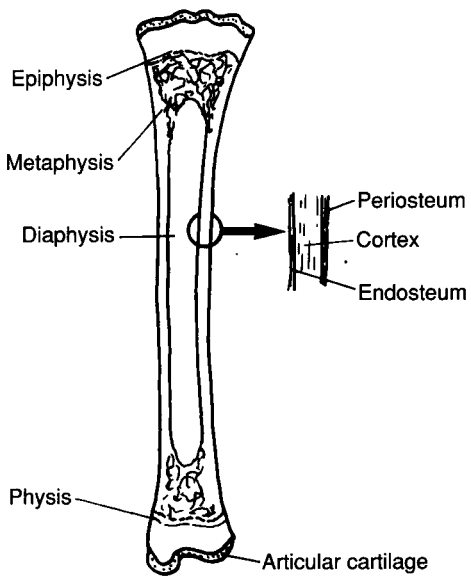

Fig. 1.2 Schematic illustration of the general anatomy of a long bone.

arranged in clusters surrounded by matrix. Further from the epiphysis comes the *zone of proliferation* where chondrocytes undergo mitosis. The dividing cells tend to form columns aligned with the longitudinal axis of the bone. These then enter the *zone of hypertrophy* where the cells swell due to accumulation of glycogen and hypoxia resulting from the cells moving away from their vascular supply which comes from the epiphyseal artery. The histological appearance of this zone is one of cell columns divided by longitudinal septae of matrix together with transverse septae between the cells within each column. At the distal end of the hypertrophic zone mineralisation of the intercellular matrix begins, forming an additional barrier to the diffusion of nutrients from the epiphysis to the chondrocytes. The resulting hypoxia is probably the cause of chondrocyte death. A *zone of vascular invasion* follows whereby capillary loops, from the nutrient artery centrally and the metaphyseal arteries peripherally, penetrate the transverse septum of the dead chondrocyte in each column. This gradual process leaves only the longitudinal septae which form the scaffold on which the internal trabecular network is created. Precursors of osteoblasts and osteoclasts arrive with the invading capillaries. Some of the longitudinal septae are removed by osteoclastic activity (chondroclastic might be considered more appropriate but no clear distinction has been made between cells having osteoclastic or chondroclastic properties). Other septae have osteoid deposited upon them by osteoblasts creating primary trabeculae which, because the whole process is in three dimensions, form a honeycomb structure.

A combination of continued bone deposition on and resorption from these trabeculae allows their movement towards or away from one another, through space, by a process referred to as

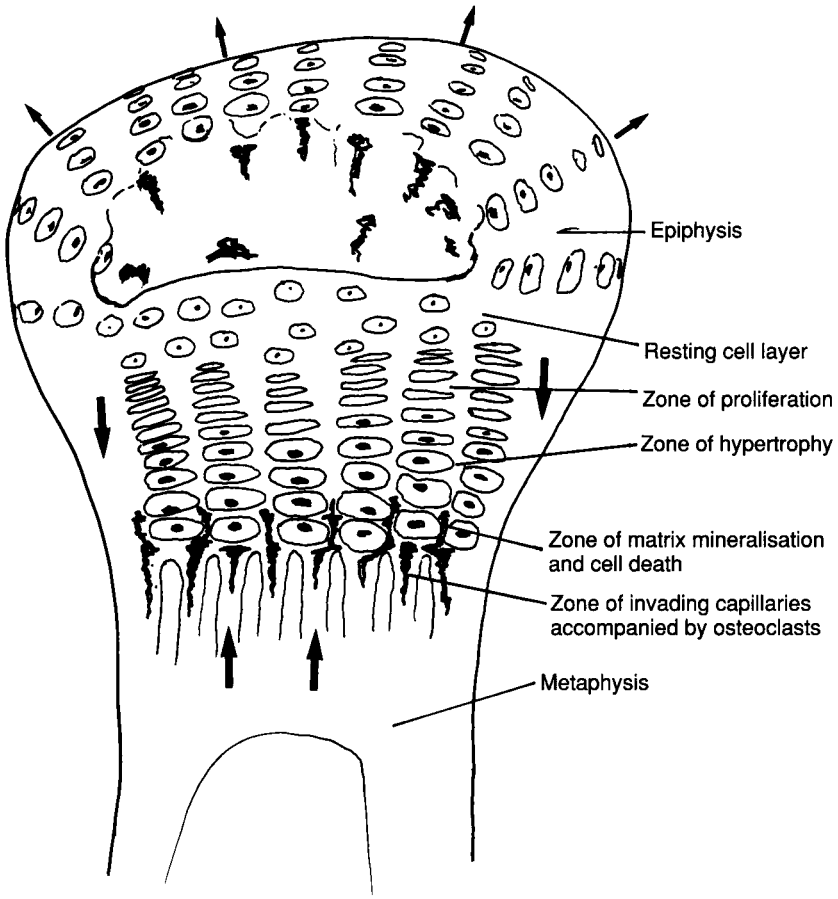

Fig. 1.3 Schematic illustration of the zones within an active physis.

'modelling'. The union of primary trabeculae forms secondary and then tertiary trabeculae. Modelling creates the trabecular architecture within the metaphyseal region of the bone and, further distal to the physis, the trabeculae unite to form the compact bone making up the cortex of the diaphysis. Some cells become trapped within this compact bone, becoming osteocytes residing within lacunae and maintaining connections with neighbouring cells through very narrow channels, termed canaliculi. A similar process occurs in the epiphysis with the proliferation of chondrocytes in the deeper layers of the articular cartilage, invasion of vessels derived from the epiphyseal artery into the zone of hypertrophy, and the formation of trabeculae which then undergo modelling.

The control mechanisms for chondrocyte activity, matrix mineralisation, trabecular formation and modelling have yet to be fully elucidated but involve both biochemical and biomechanical factors. Hormones such as somatotropin (growth hormone), acting via the production of insulin-like growth factor 1, may promote physeal activity, whilst oestrogen and testosterone may reduce chondrocyte proliferation and influence physeal closure. Vitamin D, or rather its metabolite 1,25-dihydroxycholecalciferol, influences mineralisation of the cartilage matrix. Biomechanical influence on bone formation is expressed clearly by Wolff's law which states that 'the internal architecture and external form of a bone are related to its function and change when that function is

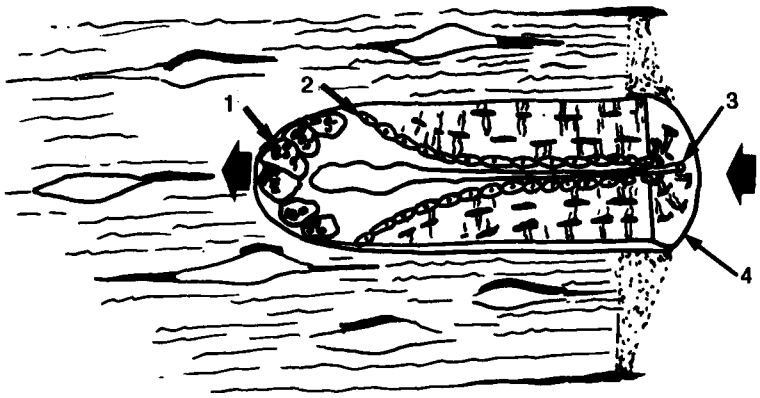

Fig. 1.4 Schematic illustration of an osteon or 'cutting cone'. 1 – Osteoclasts; 2 – osteocytes; 3 – blood vessel; 4 – Haversian system.

altered'. This means that the number and orientation of the internal trabeculae and the overall shape of both the epiphysis and diaphysis are related to the forces to which they are subjected.

After bone has formed in this way it then becomes subject to a process of 'remodelling', whereby the skeleton is constantly undergoing renewal because of gradual resorption and formation of bone. This process is important in allowing the skeleton to: (1) participate in calcium homeostasis (since it makes its mineral content dynamic rather than inert), (2) adapt its structure in line with Wolff's law, and (3) repair minor injuries such as microfractures. The rate at which this takes place is governed primarily by an animal's age but may be influenced by other systemic or local factors. In the first instance non-osteonal compact bone is osteonised by the action of 'cutting cones' (Fig. 1.4). A cutting cone is spearheaded by a cluster of osteoclasts which 'drill' a tunnel some 100–200μm in diameter. Behind the osteoclasts the tunnel becomes lined with osteoblasts which begin to deposit new bone circumferentially. Ultimately, the tunnel remaining is only of sufficient diameter to carry blood vessels and nerves. The unit formed by this activity, i.e. a tunnel filled with concentric layers of bone with a central canal (also termed a *Haversian canal*) for nerves and blood vessels, is referred to as an 'osteon' and the bone is referred to as being osteonal (or Haversian). The Haversian canals are aligned with the longitudinal axis of the bone and are connected by transverse canals called *Vaulkmann canals*. This process occurs continually and older osteons will eventually be superseded by newer osteons.

The role of bone in calcium homeostasis

It has already been stated that the role of bone in this process is small compared to those of the kidneys and intestines. However, during periods of increased calcium demand, such as lactation, it may prove necessary for an animal to rely on the skeletal reservoir. Although it is well recognised that osteoclasts are the cells responsible for bone resorption they first have to gain access to its surface. Normally, the bone surface is covered with a layer of resting osteoblasts. Osteoclasts are unable to make contact with the bone because of these cells and also, on many bone surfaces, a thin layer of unmineralised collagen which lies between the bone and osteoblasts (Fig. 1.5). Parathyroid hormone (PTH) levels rise in response to a reduced serum calcium level and there are receptors for this hormone on osteoblasts. The effect of PTH is to cause a change in osteoblast shape, whereby they become rounder and lose contact with one another. It also causes the release of a collagenase which removes the underlying collagen film, thus exposing some of the bone surface. The osteoblasts may also release cytokines which attract osteoclasts. The plasma membrane of the osteoclast in contact with the bone forms a brush border to increase the active surface area. Hydrogen ions are pumped out of the cell, through the brush border, and reduce the pH of the environment causing the mineral

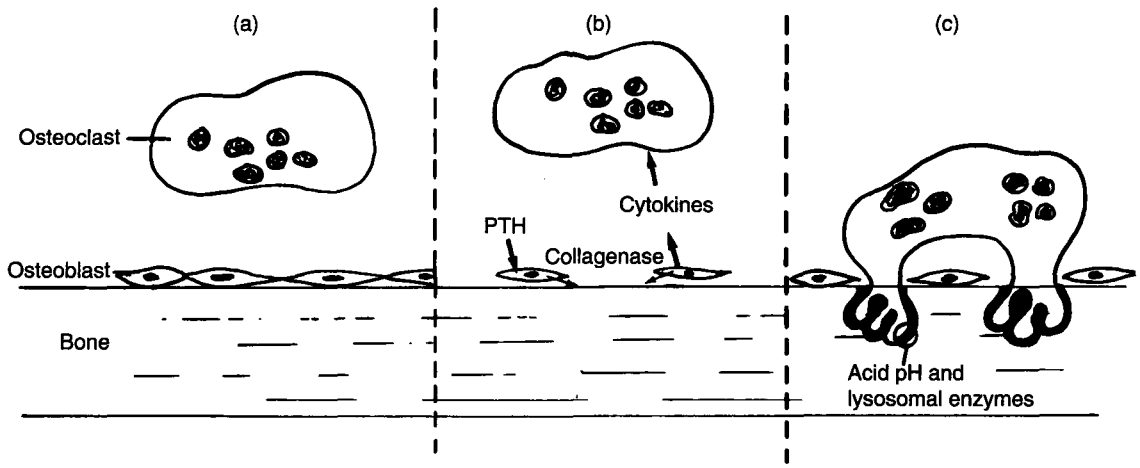

Fig. 1.5 Schematic illustration of the interaction between cells in the control of bone resorption. (a) Inactive osteoclast separated from bone surface by a layer of osteoblasts. (b) Parathyroid hormone (PTH) causes the osteoblasts to contract, exposing the bone surface, and to release collagenase which removes the collagen film from the surface. The osteoblasts also release cytokines which recruit macrophages to form osteoclasts and attract the osteoclasts to the bone surface. (c) The osteoclast contacts the bone surface by way of a brush border. Hydrogen ions are pumped out of the cell causing a drop in pH which in turn causes the mineral content of the matrix to degrade. Lysosomal enzymes are also released which degrade the organic component of the matrix. These two processes release calcium ions into the tissue fluid.

content of the matrix to dissolve. This makes calcium ions available for absorption and transport through the cell to be delivered into the tissue fluid and so into the blood stream. Lysosomal enzymes are also released through the brush border and these degrade the organic components of the matrix making more mineral available for dissolution. The defect left in the bone surface by the action of such an osteoclast is termed a *Howship lacuna.*

Biomechanical properties of bone

These can be examined in terms of the structural or material properties of bone. If any given structure is loaded by a force then it will become deformed (measured as change in length) and the relationship between these two events can be measured and plotted as a force–deformation curve (Fig. 1.6a). The characteristics of this curve are related to the structural properties of the object concerned. The area under this curve is a measure of the energy absorbed by the structure when that force is applied. When lesser forces are removed the structure will return to its previous form and in this part of the curve it is said to have undergone *elastic deformation.* With increasing forces there will come a point, termed the *yield point,* at which their removal will not allow the structure to return to its original form. This is then referred to as *plastic deformation.* Eventually a force is reached where the energy imparted to the structure cannot be absorbed by deformation resulting in it breaking (i.e. in the case of bone, it becoming fractured). This is called the *failure point.*

Deformation within a structure may be referred to as 'strain' (change in length per unit length) and this strain generates internal forces referred to as 'stress' (force per unit area). The two are mathematically linked and come in two forms, *normal* and *shear.* Normal strain causes either compression or stretching of the structure with the creation of stress that acts perpendicular to the surface. Shear strain causes torsional or angular deformation and creates stress that acts parallel to the surface. The relationship between stress and strain can be plotted out as a stress–strain curve and the characteristics of this are related to the material properties of the object (Fig. 1.6b). This

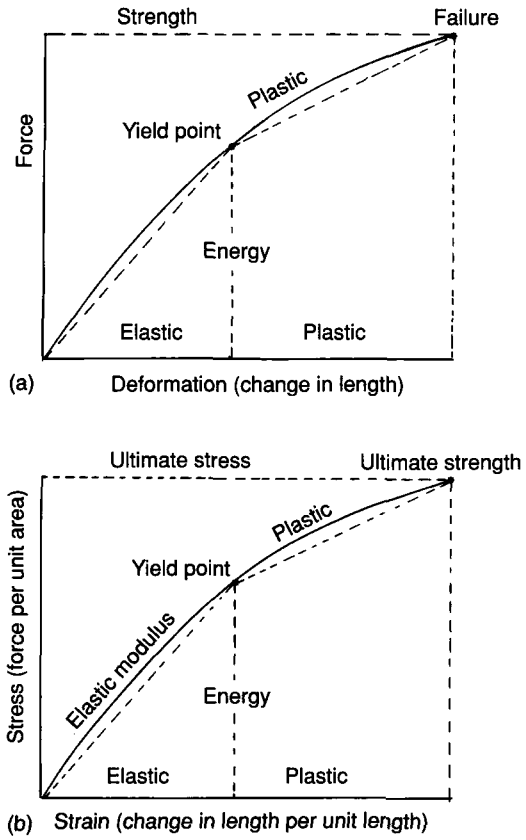

Fig. 1.6 (a) A force–deformation curve representing the structural properties of bone. (b) A stress–strain curve representing the material properties of bone.

curve is very similar to the force–deformation curve for that object, with elastic and plastic regions, a yield point between the two and a point of failure referred to as *ultimate strength*. Again, the area under the curve is a measure of the energy absorbed by the subject under those conditions of stress and strain. In addition, the gradient of the curve in the elastic region represents a measure of stiffness and is known as *Young's modulus of elasticity*.

When considering bone it is clear that neither its structural nor its material properties are uniform or static. Cancellous bone has a honeycomb-like network of trabeculae and, under compression, its stress–strain curve first shows elastic properties but then proceeds into a very prolonged region of plastic deformation, created by progressive collapse of the trabecular network, before failing. Conversely, under conditions of tension, cancellous bone fails at low loads because of trabecular distraction. Thus, cancellous bone is designed to accommodate compression and these properties are appropriate for a material found in the metaphyses where compressive forces predominate.

Cortical bone is much more dense than cancellous bone and has properties which vary according to rate and direction of loading. The more rapidly it is loaded the greater becomes its elastic modulus and ultimate strength. Thus the amount of energy absorbed before failure is far greater when the load is applied more rapidly. Any material with this property is referred to as *viscoelastic*. When cortical bone is loaded perpendicular to the direction of its osteons it will behave in a brittle manner with less plastic deformation than when loaded parallel to the osteons. Thus, a bone is able to withstand greater forces applied along its axis before the failure point is reached compared to when forces are applied across its axis. Any material whose properties depend on the direction of load application is termed *anisotropic*. Not only do the material properties of bone vary with type but they also alter with age. Immature bones are able to resist fracture by absorbing energy through deformation by virtue of a low modulus of elasticity. As the bone matures it becomes stiffer, with an increase in elastic modulus. As it loses one method of coping with forces applied to it, bone accommodates by adopting a shape that best resists the forces affecting it, a feature which has been encapsulated in Wolff's law, mentioned previously. This adaptation of bone to withstand applied forces is most probably driven by a piezoelectric effect resulting from electrical potentials generated by strain within the bone itself.

A basic understanding of these aspects of bone biomechanics aids a comprehension of the way in which bones fracture when the forces applied exceed the point of failure (Fig. 1.7). Under tension the fracture line should be transverse whereas under compression an oblique fracture line will develop because all bones have a slight curvature and so develop a tension and compression side, and thus some bending (or angular) forces when under compression. Pure bending

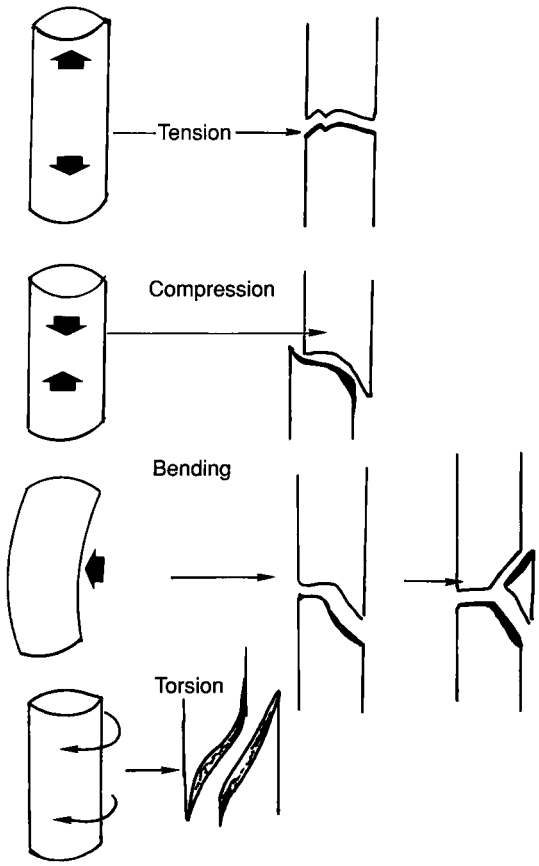

Fig. 1.7 An idealised representation of the relationship between the direction of force applied to a bone and the pattern of fracture resulting from this.

forces will produce tension and compression on opposite sides of the bone. Fracture of the bone should begin transversely on the side under tension, becoming more oblique as compressive forces are added on the opposite side. If more than one oblique fracture plane develops on the compressed side then a butterfly fragment will result. Torsional forces will tend to produce a spiral fracture but such fractures may also be influenced by the shape of the bone concerned because they are more common in the tibia and humerus whose diaphyses both have a natural 'twist' around their longitudinal axes. In clinical situations the forces applied to a bone are a combination of compression, tension, bending and torsion and a resulting fracture will often have a mixture of the aforementioned patterns. Recognising the predominant pattern(s) in a fracture can be helpful in determining which forces will be most disruptive during healing. This will then allow an appropriate treatment plan to be established.

In addition, the pattern of bone failure may provide other significant information about the injury. Mature bone loaded rapidly will absorb a great deal of energy before reaching the point of failure. Thus, if it does fail then that amount of energy will not only cause severe comminution of the bone itself but will also result in major injury to the surrounding soft tissues. Therefore, the degree of comminution seen radiographically provides some idea as to the degree of soft tissue damage. Conversely, it is unusual for a normal, mature bone to suffer a simple fracture as a result of relatively minor trauma, as the bone is able to absorb a relatively large amount of energy without developing a fracture. In such circumstances the injury might be explained by the direction of the forces applied to the bone (see earlier) but the possibility of fracture through diseased bone, or 'pathological fracture', should always be considered.

Blood supply to bone

In a mature long bone there are three functional vascular systems (Fig. 1.8). These are referred to as the *afferent*, *efferent* and *intermediate* vascular systems. The *afferent* system includes three main sources of blood. The first is the principal nutrient artery which, after penetrating the bone cortex, divides into ascending and descending medullary arteries with smaller branches supplying blood to the endosteal surface of the entire diaphysis. The second is the metaphyseal arteries which are multiple, forming a ring around the metaphysis at each end and penetrating the bone from all aspects. The metaphyseal supply anastomoses with the medullary vessels and, although it does not normally supply blood to the diaphysis, it may do so in situations where the medullary supply has been compromised. The third is the periosteal blood supply which is relatively vestigial in the mature bone except at sites of fascial or tendon attachment. At these points vessels enter the cortex in a perpendicular fashion, anastomosing

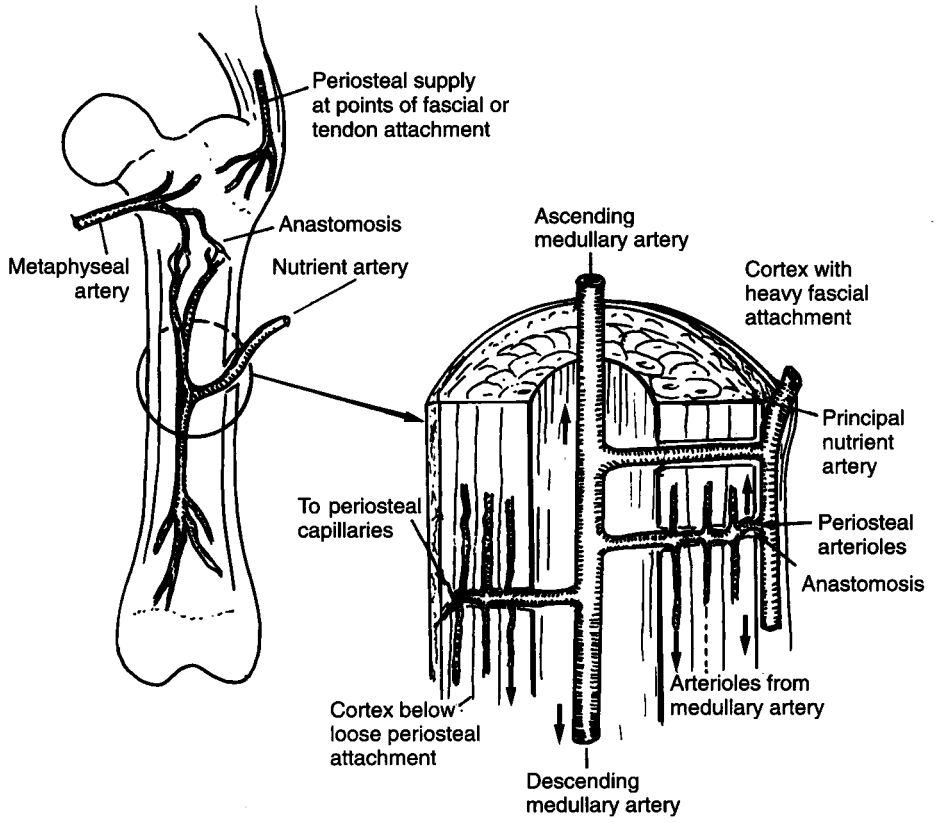

Fig. 1.8 Schematic illustration of the blood supply to an adult bone.

with vessels derived from the medullary arteries. The periosteal vessels supply the outer one-quarter to one-third of the cortical bone directly below it. In areas where there are no soft tissue attachments the periosteal vasculature supplies none of the cortical thickness in a mature bone. Additionally, despite anastomoses in the cortex, in situations where the medullary supply is compromised the periosteal supply cannot compensate and, in such circumstances, it is the metaphyseal rather than the periosteal vessels which supply the diaphyseal cortex.

The *efferent* system allows drainage of blood from the bone. The regions supplied by the metaphyseal vessels and those areas of cortical bone where a periosteal supply exists are drained by corresponding metaphyseal and periosteal venous systems. Cortex supplied by medullary arteries is almost entirely drained by periosteal vessels and it is only cortex adjacent to the medullary cavity which drains via a corresponding medullary venous system. The medullary contents drain via a system of sinusoids connecting to the nutrient vein.

The *intermediate* vascular system is that which links the afferent and efferent systems. In cancellous bone it comprises the vessels between the trabeculae, whereas in cortical bone it involves the vessels located within the osteonal (Haversian) system.

There are two main differences from the details above when considering immature bone (Fig. 1.9). Firstly, since vessels do not traverse the physis, the epiphysis and metaphysis must receive separate blood supplies. The non-articular surface of the epiphysis is covered with arborising capillary loops which penetrate the bone at the margins of the articular cartilage whilst the metaphysis is sup-

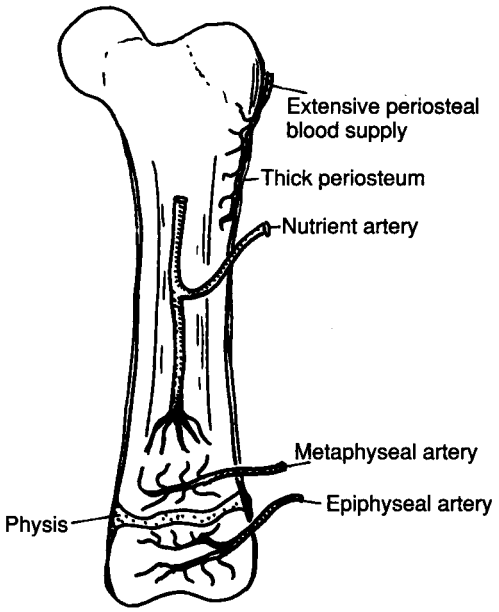

Fig. 1.9 Schematic illustration of the blood supply to an immature bone.

plied by several larger vessels which penetrate the bone around its circumference, anastomose with medullary vessels, and form several hairpin extensions towards the physis. The importance of this difference from the adult is that the supply to the epiphysis, though adequate, has no collateral alternative, making the possible consequences of injury far greater; and the presence of numerous capillary loops might predispose to haematogenous osteomyelitis due to bacteria becoming lodged at these sites. Secondly, the periosteal supply is far more extensive with longitudinal arteries and innumerable vessels radiating from these to supply the highly active osteogenic layer (cambium) of the periosteum.

Classical fracture healing

This is taken as the pattern of events which follows fracture of a long bone when it is treated conservatively, but is also seen after closed reduction of a stable fracture which is then supported by external coaptation. At the time a bone is fractured there is also damage to local soft tissues and a haematoma forms at the site (Fig. 1.10a) which contains numerous chemical mediators from both the bone itself (e.g. bone morphogenetic protein, BMP) and the supporting tissues. In addition, the coagulation activates the complement cascade, leading to an influx of inflammatory cells which then act as a source of interleukins. This, in turn, leads to the production of prostaglandins, and the platelets within the clot are a rich source of growth factors such as transforming growth factor beta (TGFβ). These chemical mediators stimulate mitosis and differentiation of mesenchymal cells, and also angiogenesis.

The sources of both cells and new blood vessels include the periosteum and endosteum but there is also an important extraosseous supply derived from the damaged, supporting soft tissues. The fibrin within the clot is the first supportive tissue at the fracture site and provides the structure required for invading blood vessels and mesenchymal cells. During this period of early response to fracture the bone ends themselves tend to resorb, for two main reasons. Firstly, the ends of the fragments are deprived of an intrinsic blood supply and so 'die back'. Secondly, increasing the fracture gap reduces the stresses in the interposed tissues caused by movement between the fragments and thus avoids those stresses exceeding the physiological limits of the invading cells.

Within a few days of injury there is a proliferation of mesenchymal cells from both the endosteum and periosteum and these cells invade the clot already formed. This proliferation is a result of both physical and biological stimuli. The cells respond to the physical disruption or lifting of the endosteum/periosteum, and also to the presence of the growth factors in the clot. Also in response to mediators from the clot there is a concurrent invasion of blood vessels which are derived from the medullary canal, the periosteum and neighbouring vascular soft tissues (extraosseous source). After invading the clot the mesenchymal cells differentiate into fibroblasts, chondroblasts or osteoblasts, depending on their local environment. Under ideal conditions of compression and adequate oxygen tension the cells become osteoblasts and woven bone is produced rapidly. In less ideal conditions of stability and oxygen tension, where osteoblasts would not survive, the

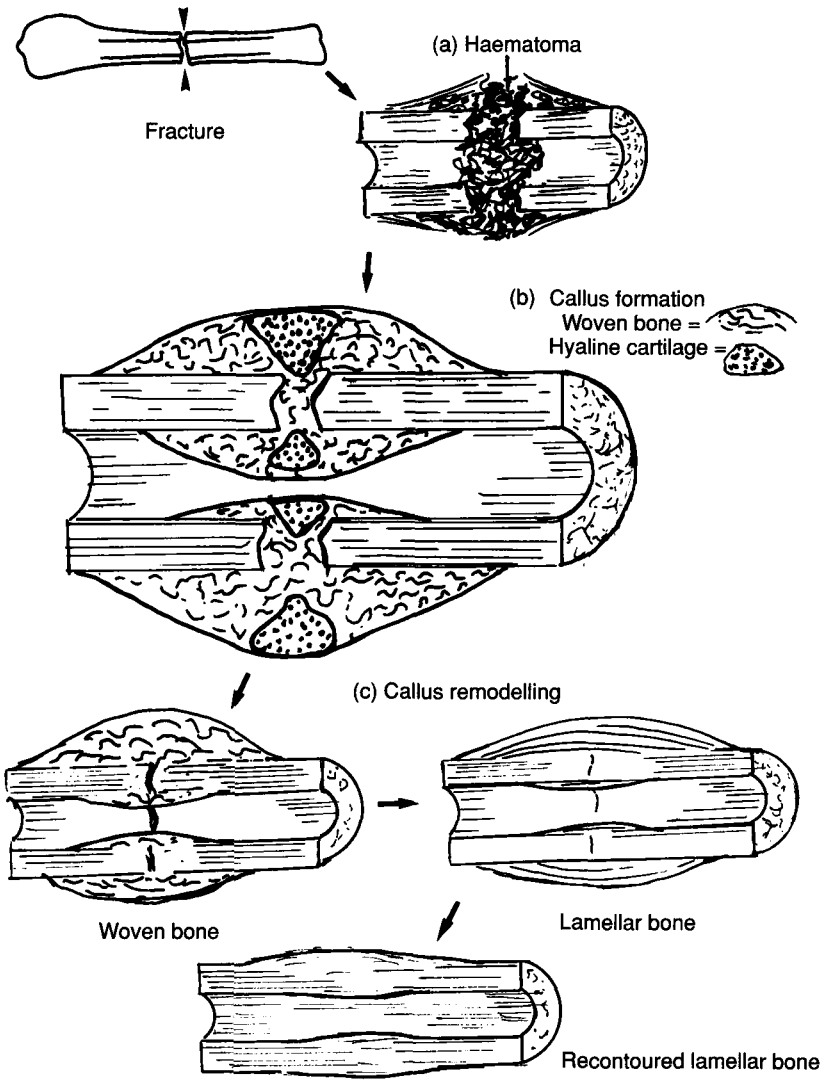

Fig. 1.10 Schematic illustration of 'classical fracture healing'. (a) Haematoma formation occurs at the site of the fracture. (b) Callus replaces the haematoma and is made up of woven bone and hyaline cartilage. (c) Once all the callus has become woven bone a process of remodelling begins and continues over a long period of time.

mesenchymal cells differentiate into chondroblasts, producing hyaline cartilage which becomes mineralised and converted to bone by the process of endochondral ossification. When the tissue is under tension (e.g. avulsion fractures) the mesenchymal cells differentiate into fibroblasts which produce fibrous tissue. This is an undesirable situation since such tissue does not enhance stability and allow the invasion of more appropriate cell types, nor does it have the ability to mineralise and become more stable. Thus, the production of fibrous tissue within a fracture gap creates a barrier to healing rather than a contribution to the bone union.

The callus so formed by the invasion and differentiation of mesenchymal cells can be divided

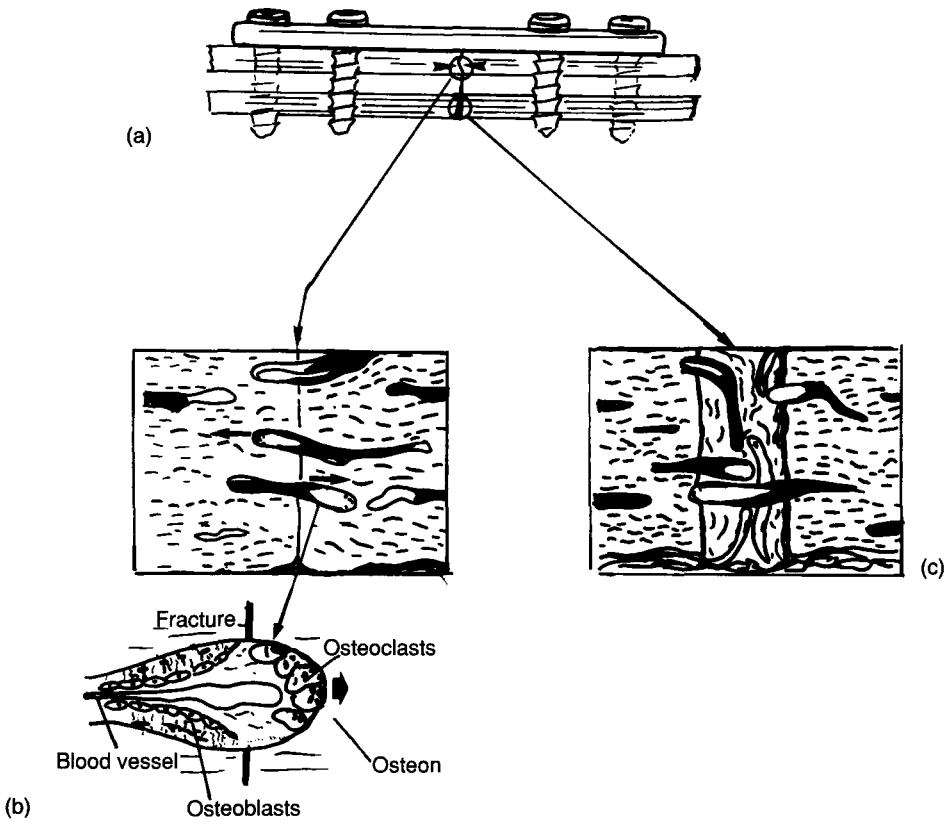

Fig. 1.11 Schematic illustration of primary bone union. (a) Fracture stabilised with compression plate. (b) 'Direct' or 'contact' healing whereby osteons pass from one fragment to another across fracture surfaces that are in contact and completely stable with respect to one another. (c) 'Gap healing' where lamellar bone forms quickly in small gaps (<1 mm) between fragments that are completely stable with respect to one another and osteons then cross between the fragments through the lamellar bone.

into *external* (derived from the periosteum) and *internal* (derived from the endosteum). As the callus advances from both sides of the fracture gap it replaces the initial clot and will usually have created a *bridging callus* by 2 weeks after injury, although this will only be faintly visible radiographically. At that stage the callus will be made up of woven bone over the fracture ends with, in most cases, an area of hyaline cartilage at the level of the fracture gap, i.e. where there is least stability and the callus is thickest, creating a lower oxygen tension within (Fig. 1.10b). In a stable fracture with adequate blood supply, bridging with bony callus ('clinical union') may be expected within 6 weeks, but a longer period will be required in less ideal circumstances. The size of the callus is directly related to the stability at the fracture site. At an unstable site the quantity of callus produced is greater so as to spread the stresses at that site through more tissue thus keeping the stress at any one point below the maximum that can be tolerated by the cells present.

Once bridging of the fracture gap with woven bone is complete the callus undergoes compaction and remodelling (Fig. 1.10c). Woven bone is converted into compact bone by osteoblasts laying down bone on the trabeculae, thus filling in the spaces. Remodelling of the callus occurs as cutting cones of osteoclasts create tunnels through the compact bone which are then filled in concentrically by the action of osteoblasts to re-establish an

osteonal (Haversian) system. As normal structure, and therefore strength, returns to the bone the callus can be reduced in size, according to Wolff's law, and so the remodelling process also leads to a gradual flattening of the callus as the bone regains more of its original shape. This remodelling occurs at the same pace as is occurring throughout the skeleton and may take months or years, depending, to some extent, on the age of the patient.

Primary bone healing

If fracture reduction is accurate and the stability rigid then healing may occur without external callus formation and is termed *direct* or *contact* healing (Fig. 1.11a,b). This requires the fracture ends to be perfectly apposed and then compressed together firmly. Since this will eliminate movement at the fracture line, there will be no signal for callus to be formed. The fracture then heals by the normal process of remodelling, whereby the bone at the ends of the fragments is replaced by new bone through the activity of 'cutting cones' (see p. 6) forming new osteons that traverse the fracture line. As these bone-filled tunnels are formed across the fracture line so the fragments gradually become reconnected to one another.

Where a small (less than 1 mm) gap exists between bone ends, but stability is sufficient, the gap will become filled with lamellar bone orientated perpendicular to the longitudinal axis. Although the gap becomes filled with bone very quickly it remains a site of weakness until it is integrated into the normal bone architecture by virtue of the remodelling process. This process is referred to as *gap healing* (Fig. 1.11a,c).

Although in an ideal situation of compression and stability it might be expected that fracture healing would take place without the formation of callus, in reality some callus will often form in response to the mechanical stimulus of periosteal or endosteal injury, particularly in skeletally immature patients.

The advantage of 'primary bone healing' over 'classical healing' is that, because the fragments are extremely stable, the bone as a whole is able to be loaded. This allows early return to limb function during fracture healing. The disadvantage is that because the process of remodelling takes a long time, the implants used to stabilise the fracture cannot be removed in the near future. It has been known for fracture healing of this type to have been in progress for several months and, on removing the implants, for the fracture to simply fall apart! Thus, primary healing is not faster than classical healing and bone union tends to be weaker in the early stages of healing. Its advantage lies in allowing an earlier return to use of the limb and thus avoidance of fracture disease (i.e. joint stiffness, muscle wastage, soft tissue adhesions and disuse osteoporosis).

Bridging osteosynthesis

The concept of *bridging osteosynthesis* has developed in an attempt to combine the advantages of 'classical' and 'primary' bone healing whilst avoiding their disadvantages. It involves the stabilisation of the two ends of a fractured bone, relative to one another, without the anatomical reduction of each bone fragment. The site of fracture is left as undisturbed as possible so as to avoid unnecessary removal of fracture haematoma (with its valuable chemical mediators) and also minimise any further compromise of the vascular supply to the region. Thus, the fracture is encouraged to heal by callus formation but in an environment of stability created by bridging of the fracture site, usually with a bone plate or an external skeletal fixator (with or without an intramedullary pin) (Fig. 1.12). In order to minimise further injury to the regional vascularity an external skeletal fixator may be applied in a closed fashion and, although a bone plate does require an open approach, this can be modified to simply allow attachment of the plate to each end of the bone without interfering with the intermediate fragments (the so-called 'open but do not touch' approach).

Such techniques are most appropriate in fractures involving the mid-diaphysis of a long bone since reasonably accurate anatomical reduction of a fracture at such sites is well tolerated as long as overall joint alignment is maintained, whereas closer to the bone end, or where an articular surface is involved, poor fragment alignment may well compromise joint and limb function. These

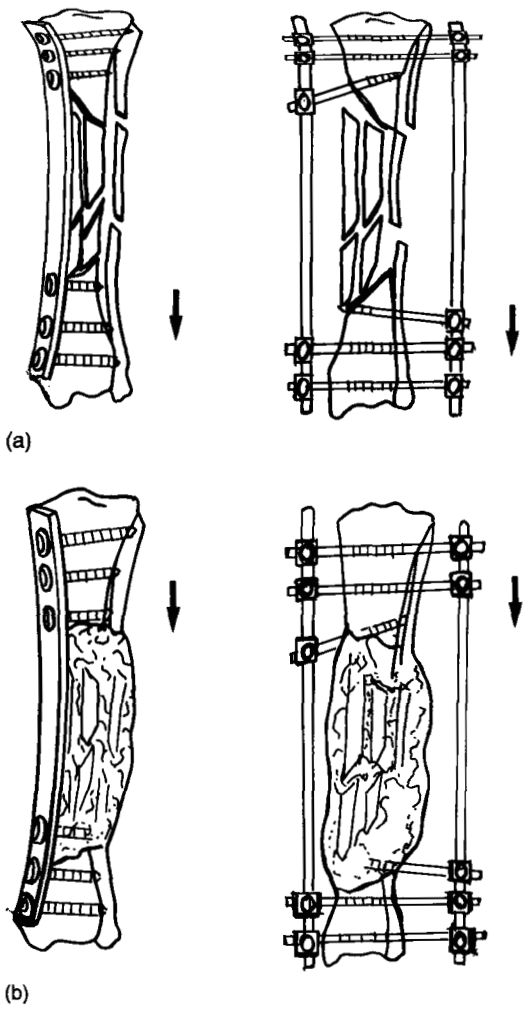

Fig. 1.12 Schematic illustration of bridging osteosynthesis. (a) A comminuted fracture may be stabilised by the application of a bone plate or external skeletal fixator to the most proximal and distal fragments with no, or minimal, exposure of the intermediate fragments. (b) Over several weeks the fracture will undergo 'biological healing' with callus forming which incorporates the fragments and creates clinical union under the protection of the bone plate or external skeletal fixator.

techniques are also most commonly used when the fracture configuration is such that anatomical reconstruction of the bone cylinder may be difficult or impossible to achieve. For example, this can occur when there are numerous small fragments

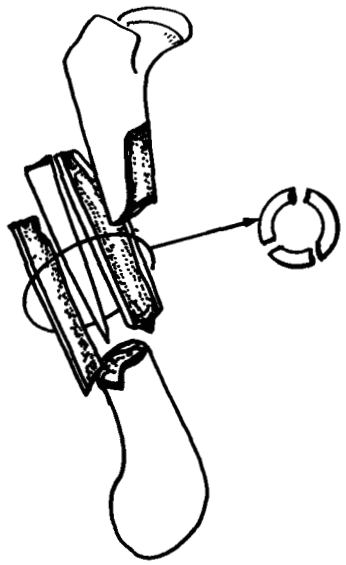

Fig. 1.13 Schematic illustration of the 'three-piece' cylinder which makes compression of the diaphyseal fragments difficult and thus attempts at fracture reduction inherently unstable.

that are of insufficient size to accommodate implants or when any transverse section of the reconstructed bone would have more than two fragments making up the circumference (the 'three-piece cylinder', Fig. 1.13), thus having a tendency to collapse into the medullary canal under compression with cerclage wires and making placement of lagged bone screws difficult because the centre of one fragment is often opposite a fracture line. In some cases a combination of reconstructive and bridging fixation may be employed whereby one or two large fragments are reduced and compressed whilst the others are bridged.

In general the rule should be that biological healing should not be disturbed unless a mechanical advantage can be gained. Except in fractures where there are two, three, or sometimes four substantial fragments it is likely that at least part of the fracture healing will involve bridging osteosynthesis.

Bridging osteosynthesis carries with it the more rapid return of bone strength seen with classical

Table 1.1 Time to reach clinical union (Brinker, 1978).

Age of animal	External coaptation, external skeletal fixation, intramedullary pinning	Plate fixation
Under 3 months	2–3 weeks	1 month
3–6 months	4–6 weeks	2–3 months
6–12 months	5–8 weeks	3–5 months
Over 12 months	7–12 weeks	5–12 months

healing, whilst allowing the limb function during healing associated with primary union by virtue of the injured bone being protected. Conversely, it reduces the likelihood of fracture disease often associated with methods of treatment relying on classical healing, and avoids the prolonged reliance on orthopaedic implants seen with primary healing.

Rate of fracture healing

The rate of fracture healing in small animals is influenced by many factors such as:

- Type of bone involved
- Type of fracture
- Age of patient
- Method of treatment
- Other systemic disease

Cancellous bone has a more abundant blood supply and a greater inherent cellular activity than does cortical bone. Therefore, a fracture involving the epiphysis or metaphysis of a bone tends to heal more quickly than do those involving the diaphysis. Impacted fractures and long spiral or oblique fractures where the fragment surfaces are in close proximity heal more quickly than in those where the fragments are widely separated. Comminuted fractures tend to heal more slowly because of inherent instability and disruption of blood supply to the numerous fragments. On the other hand simple fractures (e.g. transverse fractures), whilst having less disruption to their blood supply compared to comminuted fractures, may also heal slowly because of relative instability caused by the concentration of stresses over a small area. Healing is also delayed in the presence of infection (e.g. an open fracture) and may be delayed or not occur in fractures involving diseased bone (pathological fractures). The initial union and subsequent remodelling of a fracture will be much more rapid in a skeletally immature patient than in one that is middle-aged. It will also be influenced by concurrent, systemic diseases such as hyperadrenocorticism (Cushing's disease), chronic renal failure, or dietary inadequacies such as nutritional secondary hyperparathyroidism. The method of treatment chosen for any given fracture configuration will also influence the speed of healing depending, to a large extent, on whether it favours classical healing, primary healing or bridging osteosynthesis. Brinker (1978) defined clinical union as that point in time during the recovery when fracture healing had progressed sufficiently for the fixation device to be removed. Based on the average time taken for a simple fracture in a dog to achieve clinical union he produced a table (Table 1.1) illustrating the variation with age and method of fixation.

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Chapter 2

Bone Grafts

Bone grafting is now a well-established procedure in veterinary orthopaedic surgery. Grafts can be classified by the type of bone used, i.e.:

- Cancellous bone
- Cortical bone
- Corticocancellous bone

Grafts can also be classified according to their origin:

- *Autograft*: refers to the transfer of tissue from one site to another in the same animal.
- *Allograft*: refers to the transfer of tissues taken from one animal and transplanted to another animal of the same species.
- *Xenograft*: refers to the transfer of tissues taken from one animal and transplanted to another of a different species.

Both autografts and allografts are used successfully in orthopaedic surgery. However, xenografts tend to undergo rejection and have not proved to be of use in bone grafting procedures.

The biological functions required of a bone graft include:

- Osteogenesis
- Osteoinduction
- Osteoconduction
- Structural support

These functions are considered below in relation to the different types of graft.

Autogenous cancellous bone grafting

The two main biological functions of autogenous cancellous bone grafts are osteogenesis and osteoinduction. *Osteogenesis* is the production of new bone from viable cells which survive from the transplant. *Osteoinduction* refers to the property of the graft to stimulate the potential of the host's pleuripotential mesenchymal cells at the recipient site to produce new bone. Certain diffusible growth factors have been identified and perhaps the most important of these is bone morphogenetic protein (BMP), a non-species-specific glycoprotein which maintains osteoinductive activity following extraction and also after common storage procedures used for bone grafts such as freezing, decalcification, freeze drying or ethylene oxide treatment.

Cancellous bone is highly cellular trabecular bone and as such provides little structural support at the recipient site.

Indications for autogenous cancellous bone grafting

- In fracture repair especially the management of comminuted fractures:
  - to fill bone defects
  - to encourage revascularisation of bone fragments.
- Treatment of delayed union and non-union fractures.
- Fresh cancellous grafts can also survive in the presence of infection and can be used to fill defects resulting from debridement of bone in cases of osteomyelitis.
- Cancellous bone grafts are used in joint arthrodesis.
- Vertebral fusion.
- Treatment of bone cysts.

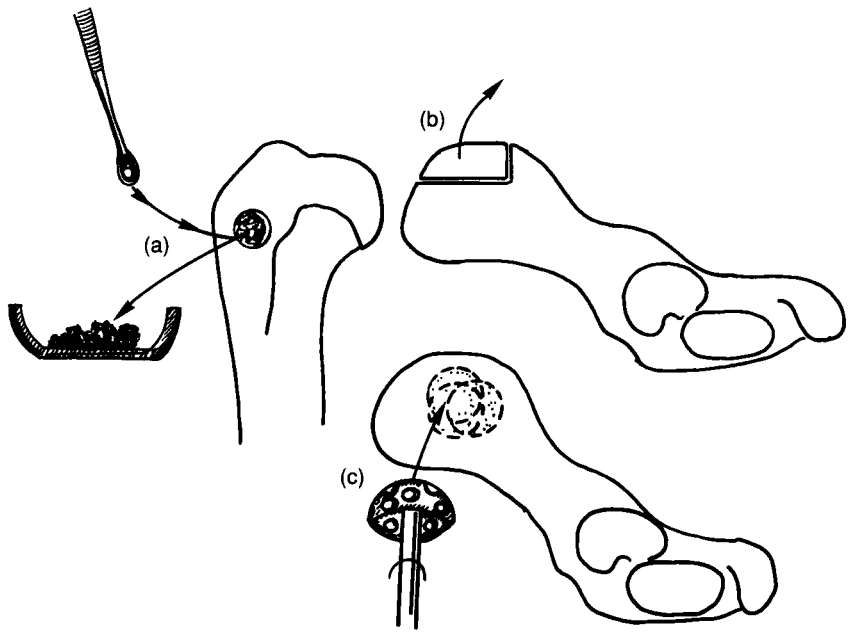

Fig. 2.1 Sites of the collection of autogenous cancellous bone include: (a) the proximal humerus and (b) the wing of the ilium. (c) An acetabular reamer is useful for the collection of bone from the ileum.

Collection of autogenous cancellous bone grafts

Preoperative planning is essential since, ideally, the site of collection of the graft should be in the same leg as the recipient site. Common sites for collection of cancellous bone include the:

- Proximal humerus (craniolateral approach)
- Proximal femur (lateral approach)
- Proximal tibia (medial approach)
- Wing of the ilium (dorsolateral approach)

The *proximal humerus* is the most easily accessible and provides the largest quantities of cancellous bone (Fig. 2.1a). A vertical incision is made directly over the greater tuberosity of the humerus and extended down to the underlying bone. Exposure is maintained with self-retaining retractors. A window is cut through the cortex with an osteotome, large Steinmann pin or Mitchel trephine. The underlying cancellous bone is curetted out with a Volkman scoop or bone marrow scoop and collected in a blood-soaked sponge prior to transfer to the recipient site. This method of collection prevents the bone graft from drying out. The graft should not be placed in saline solution as this impedes its osteogenic potential. It should be transferred to the recipient site as soon as possible after collection. Once packed into a bone defect, or around a fracture site, it is retained in position by careful suturing of the adjacent soft tissues. If a plate is used for fixation then this will overlie the graft and hold it in position.

Collection of cancellous bone from the proximal femur or tibia involves a similar technique. Complications associated with collection do occasionally arise. These include wound infection, haemorrhage and seroma formation, and, rarely, fracture at the donor site.

The *wing of the ilium* is easily accessible for the collection of bone grafts (Fig. 2.1b). An incision is made directly over the dorsolateral aspect of the wing. The middle gluteal muscle is elevated from the lateral aspect and the sacrospinalis muscle from the medial aspect of the bone. The crest can

then be removed with rongeurs and cut into small pieces of corticocancellous bone ('morselised') ready for grafting. Further amounts of cancellous bone can be curetted out from between the lateral and medial walls of the ilium. The amounts of cancellous bone collected in this way are relatively small.

A more efficient method of harvesting bone is to use a standard acetabular reamer (of the type used in hip replacement surgery). Only a lateral approach to the ilum is necessary. After elevation of the middle gluteal muscle the reamer is positioned over the lateral ilium in the caudal aspect of the gluteal depression. The reamer is attached to a low-speed drill and used to ream out the lateral cortical and intervening cancellous bone through to the level of the medial cortex (Fig. 2.1c). The amount of bone collected in the basket of the reamer can be increased by tipping the reamer cranially and caudally. A paste of corticocancellous bone is collected in the acetabular basket ready for immediate transfer to the recipient site.

Cortical bone grafts

Cortical, and some corticocancellous, bone grafts possess rigidity and strength which allow them to be used like a bone plate to restore the continuity of a bone. Cortical grafts provide structural support for *osteoconduction* which is the process whereby the graft acts as a scaffold for new bone formation. Most of the graft dies and is gradually removed and replaced by mesenchymal cells from the host bed which differentiate to form osteoblasts and osteoclasts. This slow breakdown and eventual replacement of the graft by host bone is known as 'creeping substitution'. Rigid immobilisation of the graft is essential during this healing process.

Cortical grafts seldom survive in the presence of infection and are rejected as sequestra.

Indications for cortical bone grafting

- Severely comminuted fractures, where reconstruction is considered impracticable. The fragments can be removed and replaced with a segmental diaphyseal graft.
- Certain limb-sparing techniques used in the management of bone tumours where the defect left in the bone following block resection of the tumour is filled with a cortical graft.

Sources of autogenous cortical and corticocancellous bone grafts

A large section of the ulnar shaft can be removed without any long-term effect on limb function and this provides a good source of cortical bone. A rib or the wing of the ilium are the most common sources of corticocancellous bone. This type of graft possesses the desirable properties of both types of bone graft.

Allografts of cortical and corticocancellous bone

The main disadvantages of using autogenous cortical bone grafts is that two operations have to be performed on the same animal and there are obvious limitations to the size and shape of graft that is obtained. These problems are overcome most readily by the use of allografts. Although the allograft provokes an immunological response, this does not prevent incorportion of the graft at the recipient site. The allograft dies and is gradually replaced by bone from the recipient site. The cortical allograft does not provide cells which are capable of new bone formation, but allografts of cancellous bone will induce osteogenesis at the recipient site. Although there is a delay in the incorporation of allografts compared with autogenous grafts, this delay is not sufficient enough to affect the use of allografts in clinical cases. It has been shown that there is little difference between the healing times using viable or frozen allografts and it is probable that the antigenicity of the graft is decreased by freezing.

Allograft collection

Allografts of cancellous, cortical or corticocancellous bone can be collected from a donor animal immediately after euthanasia. The bone must be collected under conditions of strict asepsis and freed from periosteum and soft tissue. Bone kept in sterile containers can be stored at –20°C in a deep freeze and can be kept for use at any time

from 4 weeks up to 2 years after collection. The 4-week delay allows the protein content of the graft to be denatured by freezing, thus decreasing its antigenicity. Before freezing, the grafts may be radiographed to provide an accessible 'library' of what is in storage.

Although aseptic collection and deep freezing for storage is the commonest way of obtaining autogenous bone grafts, bone segments can also be preserved by freeze drying, or ethylene oxide sterilisation (Anprolene, H.W. Anderson Products). In the case of the latter, after euthanasia collection of bone from the donor animal is done cleanly but strict asepsis is not essential. The diaphyses are harvested and then all soft tissue, periosteum and marrow is removed. The bones are washed in tap water and dried for 12–24 hours. They are placed in polythene tubing and sealed. The tubes are placed in 84% ethylene oxide at room temperature and normal atmospheric pressure for 12 hours. They are then allowed to aerate at room temperature for 24 hours. The prepared grafts are then stored at –20°C until used. Bone sterilised in ethylene oxide can be stored at room temperature but freezing the treated bone reduces dehydration and maintains the mechanical properties of the graft. However, it has been shown that bone sterilised in ethylene oxide has a reduced screw pullout load if stored for more than 8 months and so it should not be used after this time.

Use of frozen allografts

The frozen graft is allowed to thaw at room temperature for approximately 30 minutes before being inserted at the recipient site. If a cortical or corticocancellous graft is being used to bridge a defect then it must be rigidly immobilised, preferably with a compression plate. In addition, some fresh autogenous cancellous bone should be collected and packed around the proximal and distal ends of the graft as this will speed up its incorporation in the recipient site.

If a large bone defect is to be filled then a tubular or hemicylindrical corticocancellous allograft taken from the metaphyseal region should be used. The graft consists mainly of cancellous bone which is rapidly vascularised while the thin layer of cortical bone provides structural support. This type of bone graft is more readily vascularised than a graft of compact bone taken from the diaphysis.

Bone morphogenetic proteins (BMPs)

The main function of bone morphogenetic proteins (BMPs) is to induce the transformation of undifferentiated mesenchymal cells into chondroblasts and osteoblasts in a dose-dependent manner. Bone morphogenetic proteins, as their name implies, are isolated from bone. The proteins are able to induce new bone formation both *in vitro* and *in vivo*. DNA technology has enabled the production of BMPs in large quantities. Potential uses in clinical situations include stimulation of bone healing in delayed union and non-union fractures, as an alternative to bone grafts, to coat implants and enhance bonding to bone and perhaps the enhancement of tendon or ligament reunion with bone during reconstructive surgery. Extensive developments in the use of BMPs are likely over the next few years.

Bone grafts in cats

The basic principles of collection, storage and use of bone grafts in the cat are very similar to the dog. In the cat, however, collection of autogenous cancellous bone from the proximal humerus, femur or tibia may prove frustrating as only small quantities of cancellous bone can be obtained, especially in adult cats. The wing of the ilium provides a much more satisfactory site for collection and the bone can be used as a flat piece or is cut into small pieces, 'morselised', which are packed into the recipient site. If large defects have to be bridged with bone, then frozen allografts of cortical or corticocancellous bone can be used.

Cartilage grafts

Extensive research has been carried out in the field of cartilage and joint transplantation. Fresh and frozen osteocartilaginous allografts tend to undergo extensive degeneration with destruction of cartilage which is thought to be due to lack of revascularisation.

Shell grafts consisting of articular cartilage and

a relatively thin layer of subchondral bone stand a better chance of being revascularised. This type of graft has potential clinical application for resurfacing joints in which defects in the articular cartilage have been caused by osteochondritis dissecans or trauma, or where the natural articular cartilage has deteriorated in cases of chronic osteoarthritis. Articular cartilage has very limited healing capacity and defects in the surface persist or heal by inferior fibrocartilage formation. The end result is often degenerative joint disease. Osteochondral shell allografts provide a means of restoring a hyaline cartilage surface. The shell graft carries only a thin layer of subchondral bone which reduces the immune response at the recipient site and also improves potential for revascularisation of the graft.

Survival of the graft is influenced not only by immune response and revascularisation but also by the method of fixation used to hold the graft *in situ*. Methods of fixation of osteochondral shell autografts have been evaluated experimentally in rabbits. These include fixation with:

- Mattress sutures of polydioxanone
- Small Kirschner wires
- Small pins of polydioxanone
- Polymethylmethacrylate (bone cement) plugs

Sutures of polydioxanone provided the best results. The use of polydioxanone or stainless steel pins was satisfactory but tended to cause defects in the articular cartilage, while severe cartilage degradation was noted with bone cement.

In the management of patella luxation in the dog it is often necessary to deepen the femoral trochlea. This is done by a wedge recession technique which basically involves creating an osteochondral shell autograft from the shallow trochlea and recessing this graft deeper into the femur. In this situation the graft is held in place by friction and pressure from the overlying patella. For further detail see the section on management of medial luxation of the patella in Chapter 42 (p. 521).

Arthrodesis

The surgical fusion of a joint is called an arthrodesis. Arthrodesis is a salvage procedure and is indicated in the treatment of:

- Irreparable joint fractures
- Chronic joint instability
- The relief of pain associated with chronic osteoarthritis
- Block resection of some bone tumours

The successful arthrodesis of a joint involves four basic procedures:

(1) Removal of all articular cartilage down to bleeding subchondral bone.
(2) Where possible, flat surfaces should be cut on opposing joint surfaces to ensure optimal contact for bony union. If the contours of the joint are such that this cannot be done then the joint space should be packed with an autogenous cancellous bone graft. Cancellous bone grafts are indicated in most arthrodeses to speed up bony union.
(3) The joint should be fused at a functional angle.
(4) Rigid internal fixation is essential and in the case of the carpus and hock this is reinforced with an external splint for 6–8 weeks.

Details of the techniques for arthrodesis of individual joints have been reviewed (Denny, 1990) and can also be found in the relevant chapters of this book.

The carpus and intertarsal joints are arthrodesed most often. Fusion of these joints produces virtually no change in gait. Limb function following shoulder arthodesis is also good. Arthodesis of the elbow or stifle has a more profound affect on gait. Initially the animal advances the leg by circumduction and tends to drag the toes. Within 3–6 weeks, compensatory movement of the adjacent joints allows a reasonable degree of limb function and a return to 'normal' activity after about 3 months. However, with the exception of short legged breeds, a degree of circumduction will always be noticeable when the leg is advanced.

Arthrodesis of a major joint like the elbow or stifle creates a much longer lever arm than normal. There is an increased risk of fracture at the junction between plate and bone distal to the joint as a result of relatively minor trauma. This risk may be reduced by ensuring a gradient between rigid bone under the plate and normal elastic bone by placing the most proximal and most distal screw in the plate through one cortex

only. In addition, the plate and screws may be removed once there is radiographic evidence of bony fusion across the joint.

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