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

A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny,Steven J. Butterworth

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

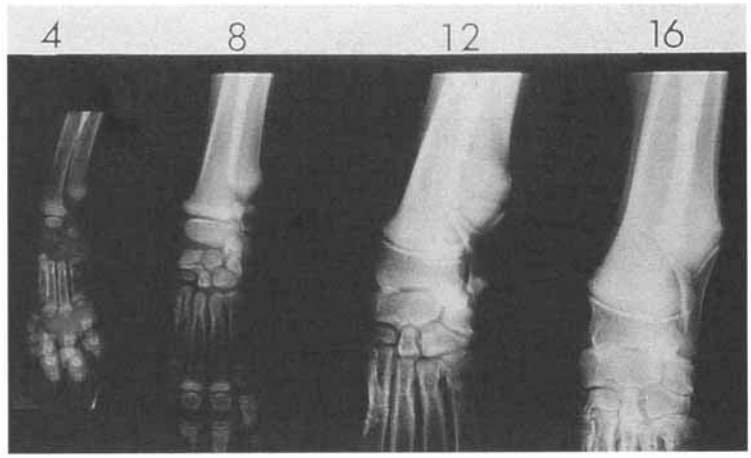

Fig. 1.1 Serial radiographs of the carpus of a puppy taken at 4, 8, 12 and 16 weeks of age showing incomplete mineralisation of the bones with large radiolucent gaps between them initially. (Courtesy C. Gibbs.)

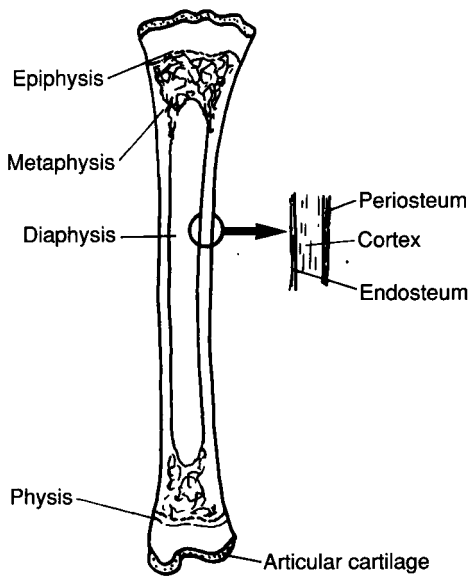

Fig. 1.2 Schematic illustration of the general anatomy of a long bone.

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

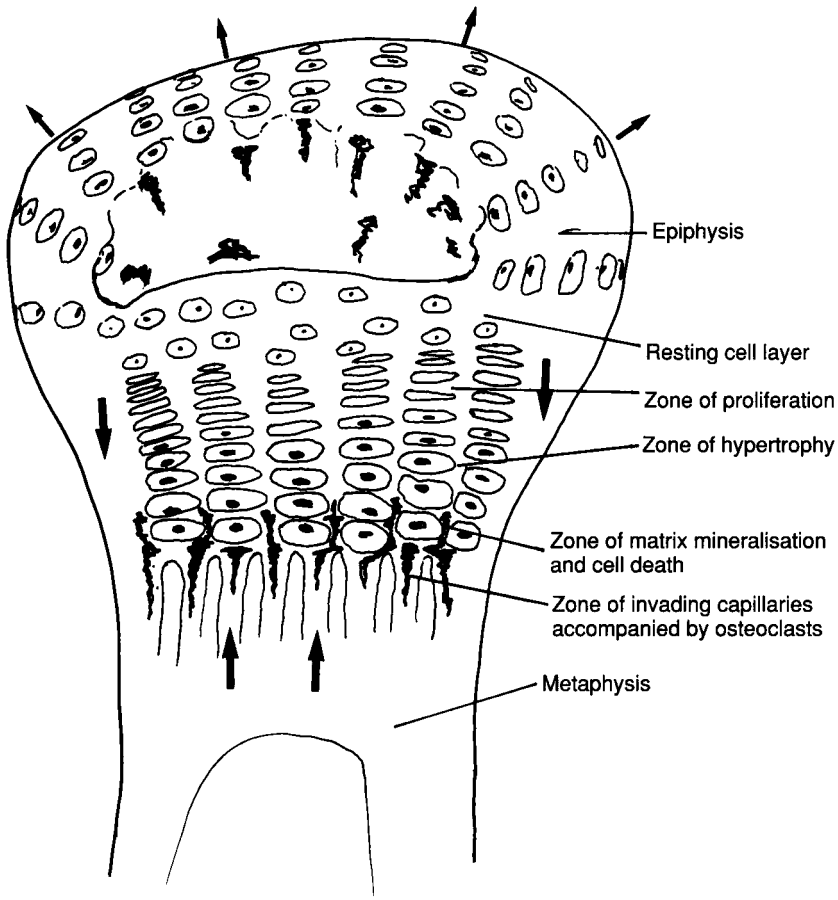

Fig. 1.3 Schematic illustration of the zones within an active physis.

'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

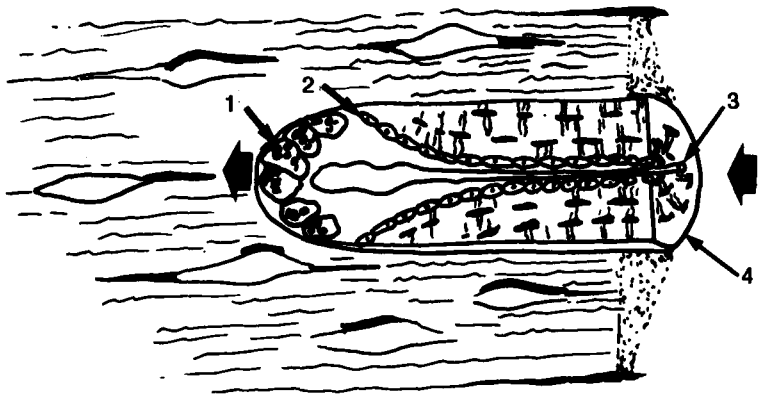

Fig. 1.4 Schematic illustration of an osteon or 'cutting cone'. 1 – Osteoclasts; 2 – osteocytes; 3 – blood vessel; 4 – Haversian system.

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

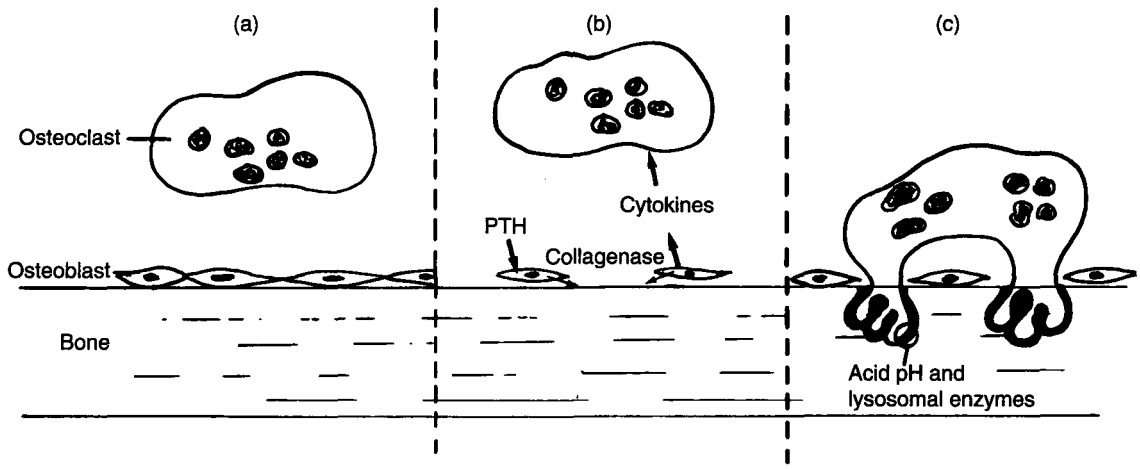

Fig. 1.5 Schematic illustration of the interaction between cells in the control of bone resorption. (a) Inactive osteoclast separated from bone surface by a layer of osteoblasts. (b) Parathyroid hormone (PTH) causes the osteoblasts to contract, exposing the bone surface, and to release collagenase which 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

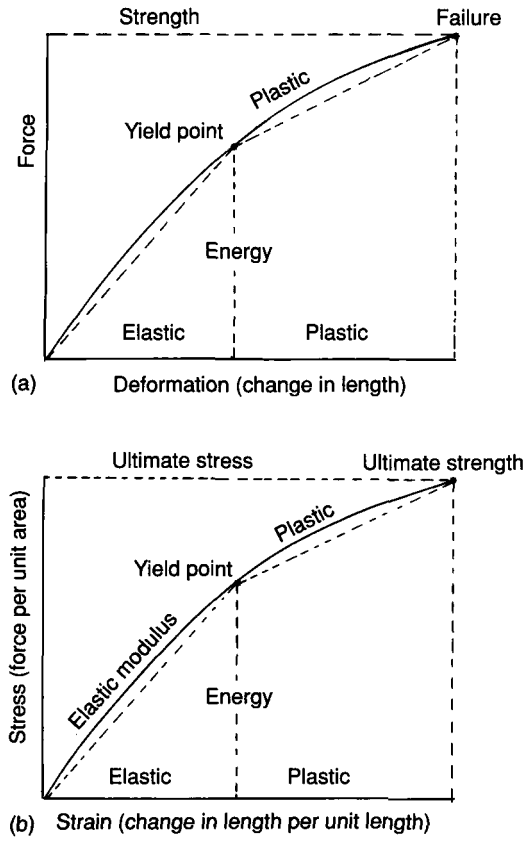

Fig. 1.6 (a) A force–deformation curve representing the structural properties of bone. (b) A stress–strain curve representing the material properties of bone.

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

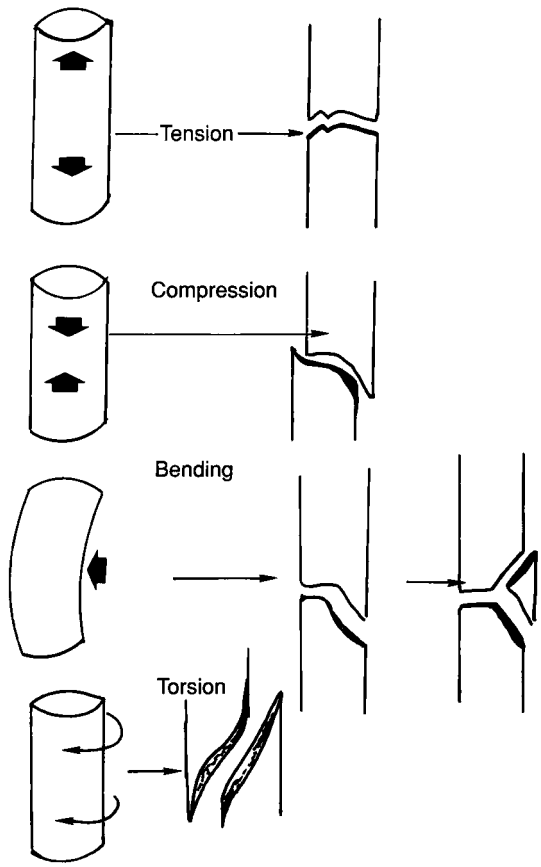

Fig. 1.7 An idealised representation of the relationship between the direction of force applied to a bone and the pattern of fracture resulting from this.

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

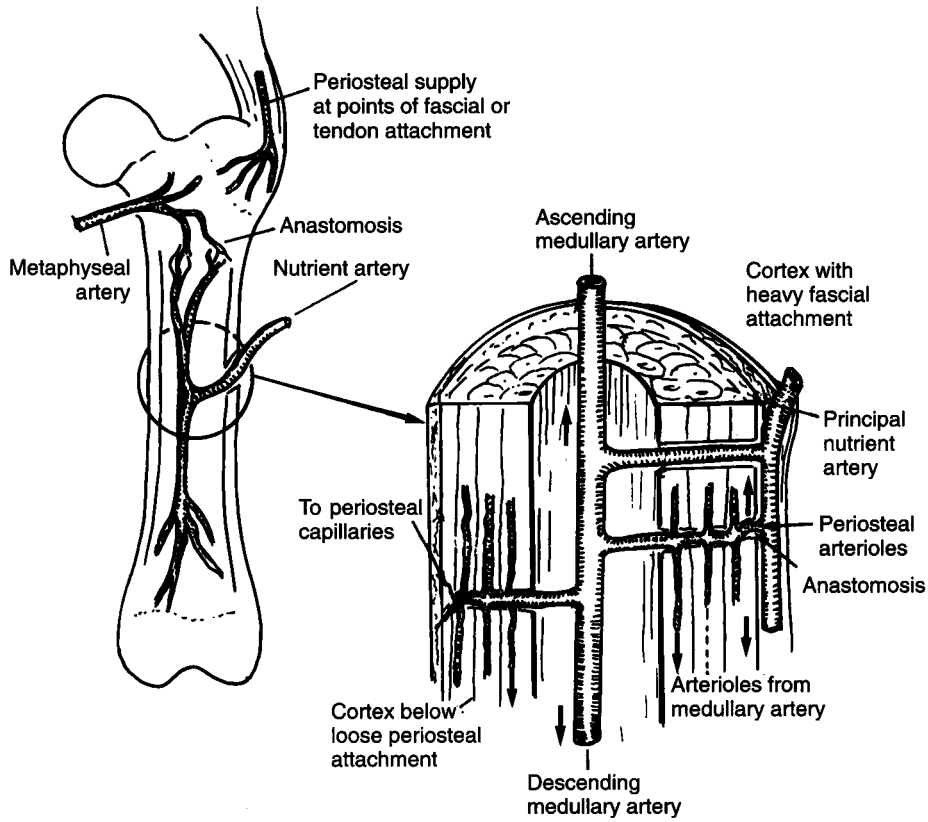

Fig. 1.8 Schematic illustration of the blood supply to an adult bone.

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-

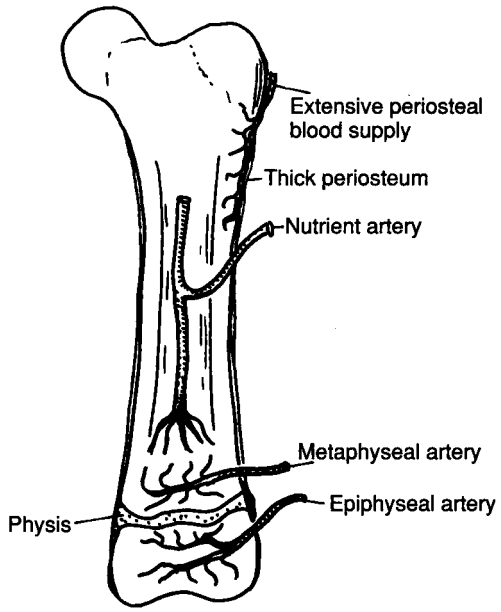

Fig. 1.9 Schematic illustration of the blood supply to an immature bone.

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

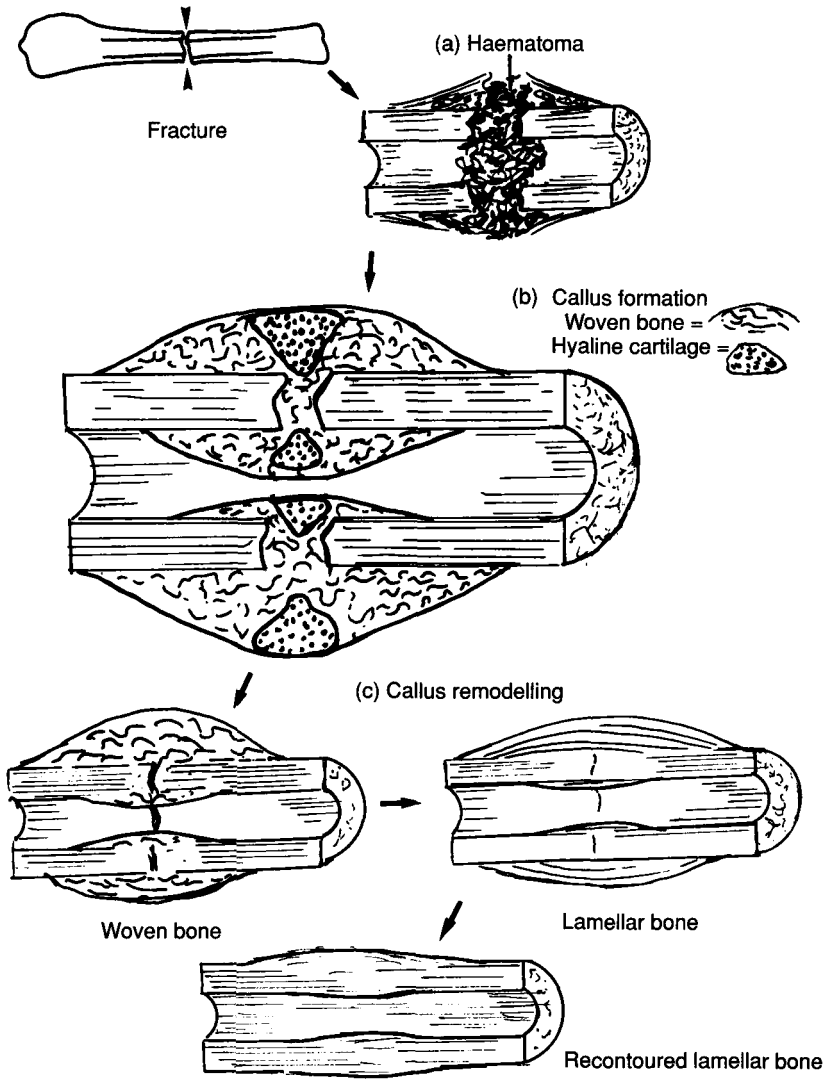

Fig. 1.10 Schematic illustration of 'classical fracture healing'. (a) Haematoma formation occurs at the site of the fracture. (b) Callus replaces the haematoma and is made up of woven bone and hyaline cartilage. (c) Once all the callus has become woven bone a process of remodelling begins and continues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

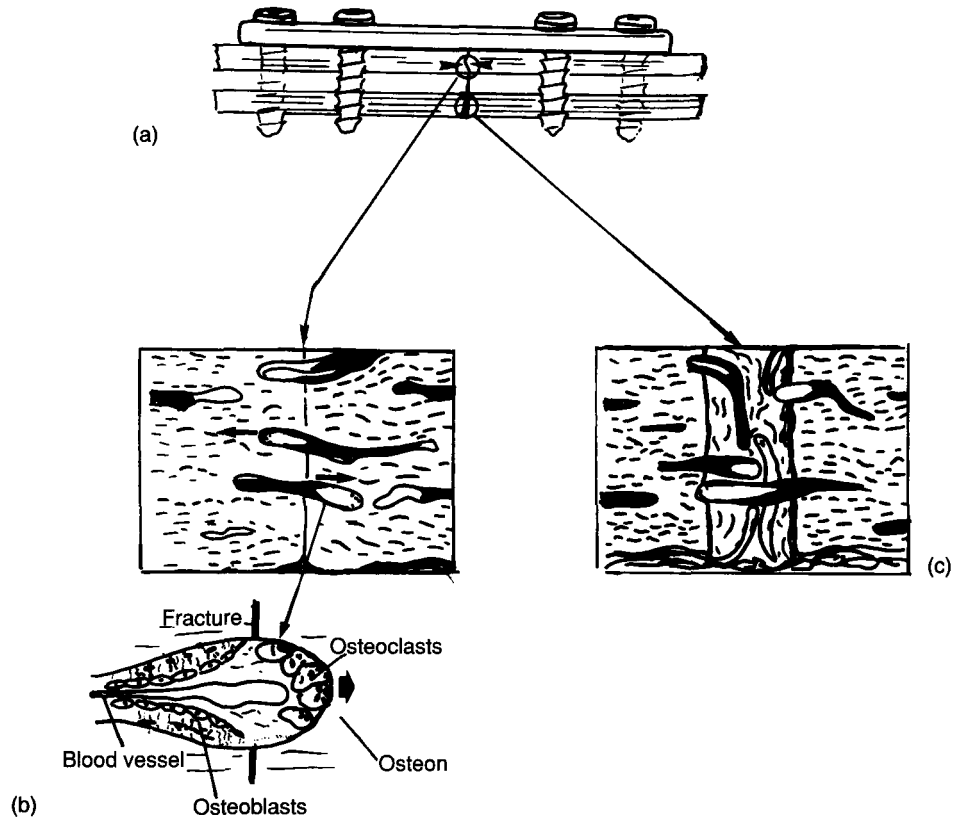

Fig. 1.11 Schematic illustration of primary bone union. (a) Fracture stabilised with compression plate. (b) 'Direct' or 'contact' healing whereby osteons pass from one fragment to another across fracture surfaces that are in contact and completely stable with respect to one another. (c) 'Gap healing' 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

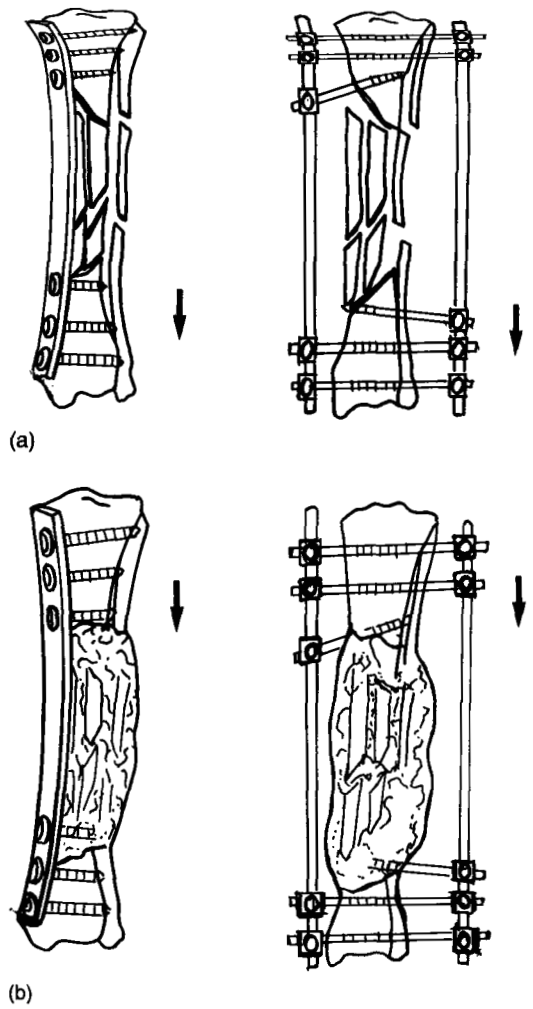

Fig. 1.12 Schematic illustration of bridging osteosynthesis. (a) A comminuted fracture may be stabilised by the application of a bone plate or external skeletal fixator to the most proximal and distal fragments with no, or minimal, exposure of the intermediate fragments. (b) Over several weeks the fracture 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

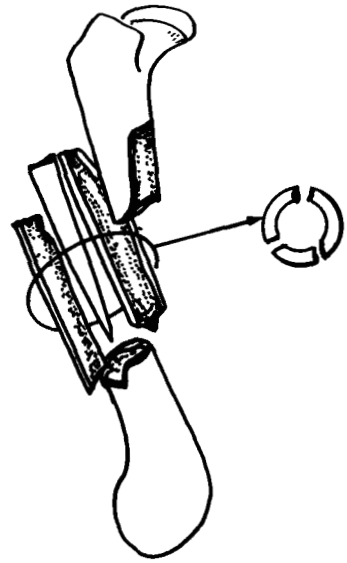

Fig. 1.13 Schematic illustration of the 'three-piece' cylinder which makes compression of the diaphyseal fragments difficult and thus attempts at fracture reduction inherently unstable.

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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A Guide to Canine and Feline Orthopaedic Surgery
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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.

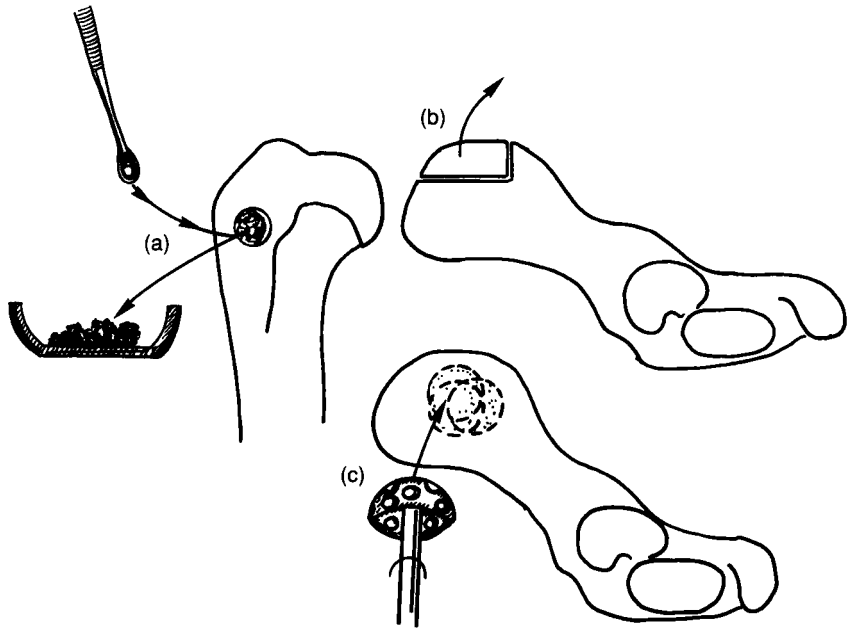

Fig. 2.1 Sites of the collection of autogenous cancellous bone include: (a) the proximal humerus and (b) the wing of the ilium. (c) An acetabular reamer is useful for the collection of bone from the ileum.

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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Chapter 3
Healing of Soft Tissue Injuries

Ligament injuries

Structure

Ligaments consist of longitudinally orientated bundles of collagen fibres. The ligaments which support a joint are either incorporated in the joint capsule as cord-like thickenings or are separated from it by outpouchings of the synovial lining termed bursae. Near their attachment to bone, the ligaments undergo a transition to fibrocartilage. Collagen fibres extend into the substance of the bone where they are attached as Sharpey's fibres.

Sprains

Ligamentous injuries are called sprains and they can be classified into three degrees:

(1) *First degree*: there is minimal tearing of the ligament, healing is rapid and lameness tends to be transient.
(2) *Second degree*: there is partial rupture of the ligament associated with haemorrhage and inflammatory oedema. A support bandage or splint is used for 3–4 weeks and exercise is controlled for up to 3 months to allow healing to occur.
(3) *Third degree*: there is complete rupture of the ligament or avulsion from its bony attachments. Surgical repair, reattachment or replacement of the ligament is usually necessary.

Healing

Healing of ligaments follows a similar pattern to that of tendons which is described below.

Muscle tendon unit injuries

The muscle tendon unit (MTU) consists of its origin or insertion, muscle belly and musculotendinous junction. Injury to any part of the MTU is called a strain. Strains and their classification are discussed in further detail below under tendon injuries.

Tendon injuries

Structure

Tendons consist of tightly packed bundles of collagen fibres (or fibrils) which surround parallel rows of tendon fibroblasts which are responsible for fibre synthesis. The collagen fibres and cells are packaged into fascicles. Although the fibres in the fascicle are essentially parallel, examination of the collagen fibres under polarised light shows evidence of a planar waveform or symmetrical crimp morphology. The crimping provides considerable lateral cohesion in the tendon and this together with interfibrillar ground substance makes slippage between fibres difficult. The organisation of tendon structure allows non-linear load deformation when subjected to tension; the crimp straightens under stress and recoils to its normal position when the load is removed. The tensile strength of tendon is similar to bone and is able to resist much higher loads than are ordinarily demanded. The collagen bundles are surrounded by peritenon and the epitenon surrounds the entire tendon unit. Both these connective tissue elements carry blood vessels to the internal structures of the tendon. Non-sheathed tendons are covered by the paratenon (loose connective tissue) which allows

the tendon to glide and also supplies the blood vessels. When the tendon crosses a joint it is surrounded by a sheath. The tendon sheath consists of a sac, containing synovial fluid, folded around the tendon, the fold is formed by the mesotenon which carries the blood supply to the tendon unit.

Tendon injury and healing

When a tendon is subjected to loads above the safe limit (a tendon of cross-sectional area of 1 cm^2 will normally support a load of 600–1000 kg), then there is loss of lateral cohesion with slippage and rupture of fibrils. Fibre rupture is associated with severe capillary haemorrhage within the tenon. There is immediate fibrin deposition and ischaemia at the site of injury, which produces congestion, fibroblast necrosis and fluid accumulation between the fibres. The acute inflammatory exudate also contains hydrolytic enzymes which cause further damage to the collagen fibrils and interfibrillar matrix. Healing following severe tendon injury involves the formation of granulation tissue and the sequence of events is detailed below.

When a tendon is cut, the wound is invaded by fibroblasts from the inner tendon sheath, or, if the wound involves a non-sheathed area of tendon, then the fibroblasts are derived from the paratenon. The fibroblasts lay down randomly orientated collagen fibres in the gap between the severed tendon ends. These fibres then become organised so that by 3 months postinjury they are found to be longitudinally orientated between the severed tendon ends. This organisation of orientation is controlled by the directions of strain within the healing tissues.

Investigations into the tensile strength of tendons during the healing process have shown that function of the tendon during the early stages of exudation, fibroplasia and fibrous union has a deleterious effect on healing, whereas function of the tendon during the latter stages of maturation and organisation accelerated the process. Consequently, following tendon repair complete immobilisation is essential for 4–6 weeks postoperatively and then a gradual increase in movement is allowed during the following 2 months. In tendon repair, restoration of tensile strength is the prime objective, but at the same time gliding function should be maintained if normal limb function is to continue. Gliding function of the tendon is commonly compromised by excessive scar tissue formation, this can be minimised by careful surgical technique and also by controlled exercise during the latter stages of healing.

Types of tendon injury

Strains and ruptures (tendinitis)

Injuries to an MTU are called strains. Injury to any part of the MTU, i.e. the tendon of origin, the muscle belly, or tendon of insertion, will result in dysfunction of the unit as a whole. Strains can be classified as first degree (mild), second degree (moderate), or third degree (severe injury).

The majority of strains resolve with conservative treatment. In the acute phase the aim of treatment is to limit the haemorrhage, fibrin exudation and tissue fluid and this reduces the extent of subsequent scarring and contracture. Treatment will therefore involve the use of cold compresses, immobilisation and the prevention of further tensile stress. Examples of these are listed below; however, further detail is given elsewhere in this book:

- Strain of the tendon of insertion of the flexor carpi ulnaris, strains of the semitendinosus and the sartorious muscles, common calcanean tendon, and digital flexor tendons, which are seen particularly in the racing Greyhound.
- Bicipital tenosynovitis.
- Contracture of the infraspinatus and/or supraspinatus muscles.
- Quadriceps muscle contracture.
- Gracilis muscle contracture.

Complete ruptures, especially those involving the musculotendinous junction or tendon, require surgical repair. Examples of these include:

- Gracilis muscle rupture in the racing Greyhound.
- Gastrocnemius muscle rupture.

Severed tendons and tendon ruptures

When treating a minor cut in a region where tendons are relatively superficial, e.g. the plantar

aspect of the metatarsus, any tendon injuries are easily overlooked, especially when the main concern with many such wounds is the degree of haemorrhage. It is important that the integrity of local tendons is ensured by observing paw/limb position during weight-bearing and that, if injury is suspected, the wound is inspected thoroughly (often requiring the defect in the skin to be enlarged). This is important since early repair of a tendon injury leads to far fewer complications when compared to delayed attempts at treatment. Monofilament nylon sutures are used for repair. The suture patterns include the traditional Bunnell tendon suture (Fig. 3.1), the Pennington locking-loop tendon/ligament suture (Fig. 3.2) and the three-loop pulley (Fig. 3.3). The Pennington suture is easier to apply and allows accurate apposition of the tendon ends. However, the three-loop pulley pattern has been shown to provide more support, less tendon distraction, and less tendon matrix constriction and distortion than the locking-loop pattern. In general, the Pennington locking loop is used for apposing the severed ends of flat tendons, whilst the three-loop pulley is used for round tendons. In chronic tendon injuries filamentous carbon fibre or polyester is used as a scaffold to induce collagen formation and bridge the gap in the tendon. Immobilisation

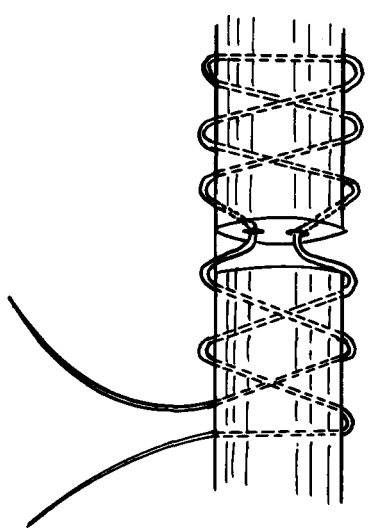

Fig. 3.1 Tendon suture – a Bunnell suture.

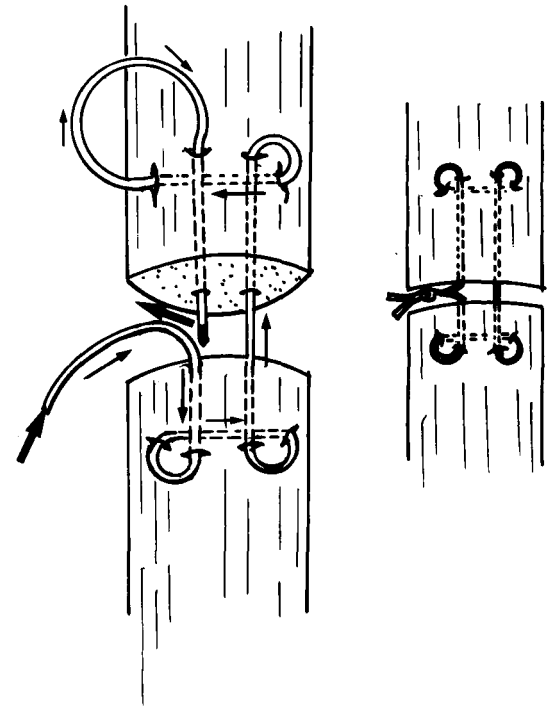

Fig. 3.2 Tendon suture – a Pennington locking-loop suture.

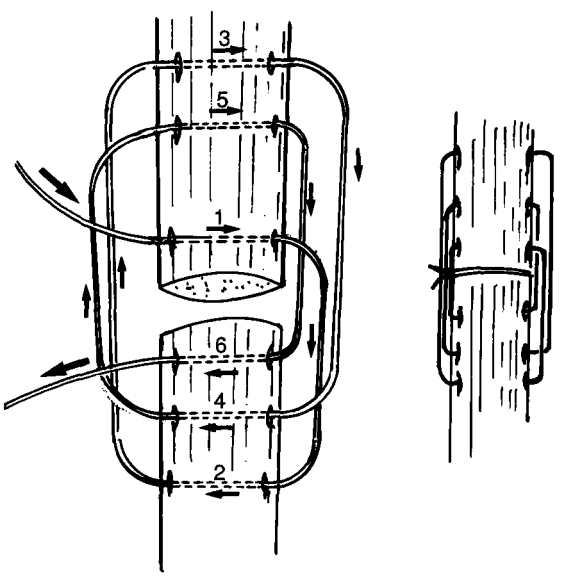

Fig. 3.3 Tendon suture – the three-loop pulley suture. For diagrammatic clarity the bites in the tendon are show in the same plane. However, in practice as much rotation as possible should be obtained between each bite when placing the suture.

of tendon repairs is essential for some 4–6 weeks postoperatively.

Examples of severed or ruptured tendons and the type of support used following repair include:

- Digital extensor tendons (splint following repair).
- Digital flexor tendons (flexion bandage or cast following repair).
- Common calcanean tendon (fix hock in extension with lagged bone screw through os calcis and distal tibia, remove screw after 6 weeks).

Tendon avulsions

Most tendon avulsions, except common calcanean tendon avulsions, tend to be seen in immature dogs and result in the tendon being pulled away from its point of attachment with a fragment of bone. The tendon is reattached with a lagged bone screw placed through the bone fragment. If the bone fragment is too small to take a screw then a ligament staple or a screw and spiked washer are used to reattach the tendon to the bone. Alternatively, the tendon can be sutured to the adjacent soft tissues.

Examples of tendon avulsions include:

- The various avulsion fractures, i.e. scapular tuberosity, olecranon, tibial crest, etc. (Kirschner wires and tension band wire used for fixation).
- Avulsion of the medial epicondyle of the humerus.
- Avulsion of the long digital extensor tendon in the stifle.
- Avulsion of the insertion of the gastrocnemius muscle from the os calcis. This injury tends to be seen as a chronic problem in older Dobermann Pinschers. The hock is temporarily fixed in extension with a lagged bone screw for 6 weeks to allow healing to occur in partial avulsions. In addition, in complete avulsions the tendon is reattached to the os calcis by passing suture material through the tendon and a transverse tunnel in the os calcis.

Displaced tendons

Specific examples of this type of problem include:

- Displacement of the superficial digital flexor tendon off the os calcis. The injury results from rupture of the medial retinaculum. The torn retinaculum is repaired or in chronic cases tightened by an overlap procedure.
- Displacement of the tendon of origin of the biceps brachii muscle following rupture of the transverse humeral ligament. The tendon is retained in its normal position by replacing the ligament with a wire prosthesis.

Skeletal striated muscle

Structure

Striated muscle consists of muscle fibres arranged in fascicles. The space between fibres in a fascicle is filled with connective tissue, the endomysium. Surrounding each fascicle is a strong connective tissue sheath, the perimysium. Strong connective tissue, the epimysium, also forms the outer sheath of the muscle body. The structure of muscle and its connective tissue sheaths is illustrated in Fig. 3.4.

Healing

Wounds involving skeletal muscle can heal both by fibrosis and by regeneration of myofibrils. If there is poor alignment of the wound edges then healing tends to be by scar tissue; however, if the muscle wound is carefully debrided allowing accurate apposition of healthy muscle at the wound edges, then ideal conditions are provided for the

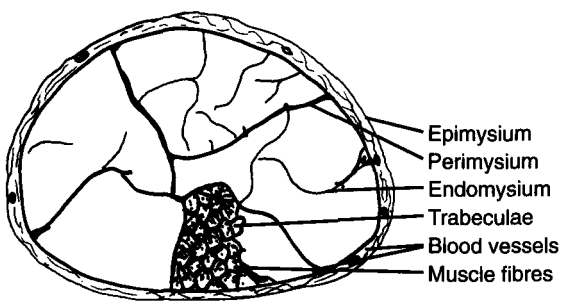

Fig. 3.4 Section through a striated muscle, showing connective tissue sheaths.

formation of fibrous tissue and the regeneration of myofibrils. The rate of muscle regeneration is said to be 1.5 mm per day.

The epimysium, or external fascial sheath of the muscle, offers the main holding power for sutures and a horizontal mattress pattern offers the best resistance to pull-out.

Myopathies See Chapter 49.

Peripheral nerves

Structure

The basic structure of a peripheral nerve is illustrated in Fig. 3.5. The outer covering of the nerve, the epineurium, consists of a loose network of collagen, elastin and fibrocytes. The epineurium has considerable elastic properties and tensile strength which allows the peripheral nerve to undergo elastic deformation without rupture. A cross-section of the nerve reveals bundles of nerve fibres called fascicles, or funiculi, and each of these fascicles is surrounded by a connective tissue sheath, the perineurium. Individual nerve fibres (Fig. 3.5) have a myelin sheath and neurolemma. Each fibre lies within an endoneurial tube which plays an important part in nerve regeneration following injury. The endoneurial tubes lie within a connective tissue medium called the endoneurium.

Classification of injury

Neurapraxia

Neurapraxia is the mildest form of nerve injury and occurs after minor stretch or pressure injuries which cause nerve contusion. Nerve conduction is interrupted but this damage is seldom permanent and function returns within hours or days.

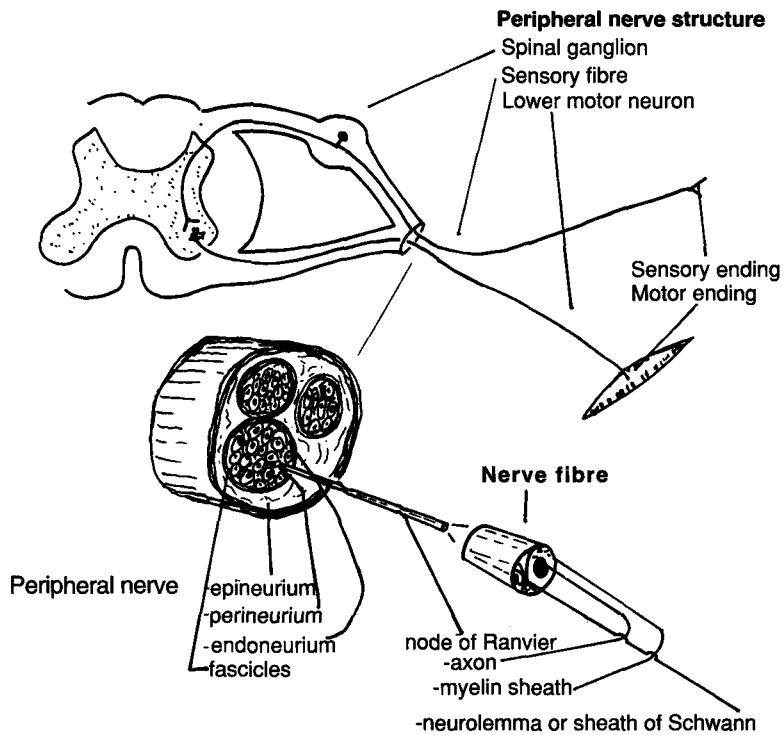

Fig. 3.5 Peripheral nerve structure.

Axonotmesis

Axonotmesis results in disruption of axons. However, the Schwann cell and connective tissue elements of the nerve remain intact. Depending on the extent of axonal damage there will be partial or complete loss of motor and sensory function and subsequent neurogenic atrophy of skeletal muscle. Axonal degeneration occurs distal to the injury but, owing to the continuity of the endoneurial tubes being maintained at the site of injury, there is potential for recovery as these tubes guide newly formed axoplasm through the site of injury. Regeneration of the nerve proceeds at approximately 1 mm per day and as a result it takes several weeks for any improvement to be seen.

Neurotmesis

Neurotmesis describes complete transection of the peripheral nerve trunk resulting in loss of all motor and sensory innervation distal to the site of injury. Transection of a nerve results in degeneration of all the fibres distal to the injury with fragmentation of axons and myelin; similar degenerative changes occur for a short distance proximal to the site of transection, this classical response is known as *Wallerian degeneration*. Schwann cells grow into the gap between the nerve ends and unite the stumps while macrophages remove degenerated axon and myelin. Schwann cells form cords in the distal endoneurial tubes. Axoplasmic regeneration starts within 2–20 days of injury. Axons bud out into the network of Schwann cells. If the cut ends of the nerve are in approximation some axons will align and migrate distally in the endoneurial tubes where they are enfolded by Schwann cells and, later, new myelin is formed. Any resulting reinnervation takes several months to occur.

Further classification of nerve injury

There will obviously be some overlap between degrees of nerve injury described and a more accurate classification was presented by Sunderland in 1951 in which he divided the injuries into five degrees of severity, first degree (mildest) injury corresponding with neuropraxia and the most severe, or fifth degree, corresponding with neurotmesis.

Repair (neurorrhaphy)

Primary repair within 8–12 hours of injury is indicated in all sharp clean wounds involving a nerve, while delayed or secondary repair at 2–6 weeks is used when nerve damage is associated with major trauma and contamination.

An end-to-end anastomosis is carried out using fine sutures (9–0 monofilament nylon or polypropylene with a swaged taper-point needle). A series of simple interrupted sutures are placed through the epineurium only. The smallest number of sutures possible should be used to minimise inflammatory response. However, accurate alignment of the nerve ends is essential to ensure any chance of a successful result. The use of optical magnification such as a binocular magnifying loupe and supplemental lighting will help improve accuracy of the surgery. The main complications of healing are ingrowth of connective tissue and aberrant axon migration. These two problems have lead to the use of a sleeve or cuff which is placed around the anastomosis to prevent ingrowth of fibrous tissue. Many biological and synthetic materials have been used for this purpose but silastic silicone rubber is used most often in clinical situations.

Specific nerve injuries See Chapter 20.

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Chapter 4
Osteochondrosis

Aetiology

Osteochondrosis is a condition which causes a disturbance in endochondral ossification in which either parts of the physis or the deeper layers of an articular surface fail to mature into bone at a normal rate. The cause of osteochondrosis is not fully understood. However, based on clinical and research observations, it is accepted that certain factors contribute to the development of osteochondrosis. These factors include:

- *Genetic involvement* is suggested not only by a well-recognised breed incidence but also by hereditability studies carried out with respect to elbow osteochondrosis. It is unlikely that the genes involved cause osteochondrosis per se but more likely that they control other factors such as growth rate, conformation and even temperament (activity level) which may then influence the development of osteochondrosis.
- *Nutritional effects* on the development of osteochondrosis have long been recognised and overfeeding of a hypercalorific diet or excessive mineral supplementation, especially calcium overload, may create a higher incidence of osteochondrosis in experimental animals. However, most cases seen nowadays with clinical osteochondrosis are pets fed correct amounts of commercially balanced diets without mineral supplementation.
- *Trauma*. The influence of trauma is very difficult to quantify. It has long been hypothesised that the areas of articular cartilage within a particular joint that develop osteochondritis dissecans lesions are those where there is the greatest pressure change between weight-bearing and non-weight-bearing. If this is the case then it is likely that conformation will influence the susceptibility of a particular dog and that a relatively large bodyweight and overactivity could predispose it further to the development of clinical disease.

There is little doubt that more factors can be identified which might influence the development of osteochondrosis and it is also clear that these factors may all interact. Therefore, trying to evaluate the influence of any one factor in isolation is very difficult and any conclusions from such a study may or may not be relevant to the naturally occurring disease.

Pathogenesis

Four types of osteochondrosis are recognised:

- Articular cartilage thickens, *osteochondrosis* (OC), and may separate from the underlying bone, *osteochondritis dissecans* (OCD).
- Physeal cartilage is involved, resulting in failure of a separate centre to unite, e.g. ununited anconeal process (UAP) or else a reduced rate of growth of a long bone. Disturbances in normal endochondral ossification which affect a growth plate may have various effects on long bone growth ranging from shortening to angular deformities, e.g. carpal valgus deformity secondary to premature closure of the distal ulnar growth plate or genu valgum secondary to uneven closure of the distal femoral growth plate. These growth disturbances are discussed in Chapters 34 and 42, respectively.
- A primary centre of ossification fails to develop normally, e.g. radial carpal bone.

- A secondary centre of ossification fails to develop normally, e.g. ununited medial humeral epicondyle.

Osteochondrosis affects well-recognised areas of certain joints. In these sites the orderly process of endochondral ossification is disrupted resulting in failure of matrix mineralisation and thus an increase in cartilage thickness (OC) (Fig. 4.1). Owing to the lack of adequate diffusion of nutrients from the synovial fluid, the cells in the deeper layers of cartilage may undergo necrosis creating a cleft between the cartilage and the subchondral bone. Vertical fissures may develop between the cleft and the articular surface creating a flap of cartilage, osteochondritis dissecans (OCD), which may mineralise (Fig. 4.2). Detachment of the flap may occur, after which it is either resorbed or else forms a joint mouse. The cartilage defect fills with granulation tissue which will eventually become fibrocartilage. Secondary osteoarthritis is almost inevitable once the articular surface has been breached. However, whether this becomes clinically significant depends on the joint involved and the degree of secondary change.

Manifestations of 'articular osteochondrosis'

Common or well-recognised lesions are given in *italics* whilst a question mark (?) indicates there remains uncertainty over whether this disease is a form of osteochondrosis:

- **Shoulder**
  - *OC and OCD of the caudal humeral head*
  - fragmentation of the caudal rim of the glenoid

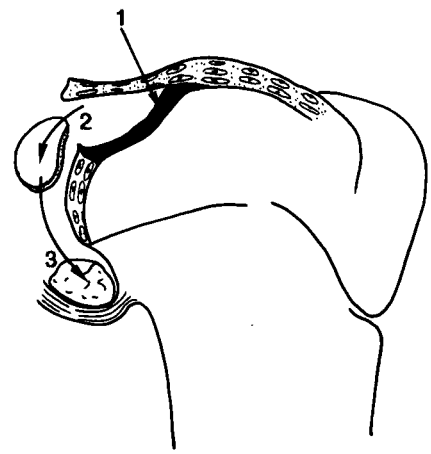

Fig. 4.2 Osteochondritis dissecans of the caudal humeral head. 1 – Cleavage; 2 – cartilage flap; 3 – joint mouse.

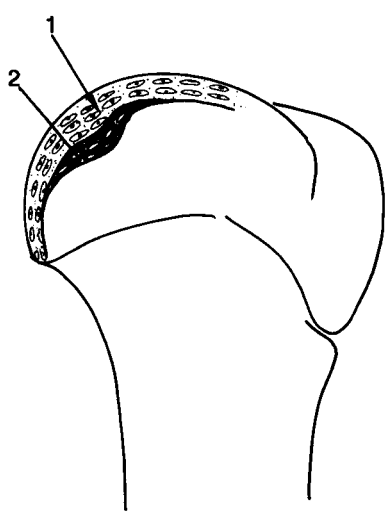

Fig. 4.1 Osteochondrosis of the caudal humeral head. 1 – Thickened articular cartilage; 2 – zone of chondromalacia.

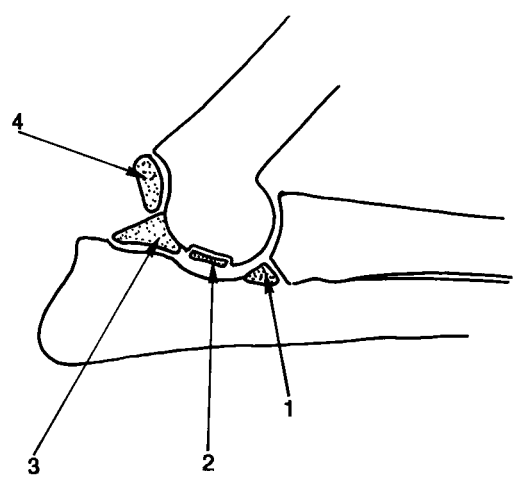

Fig. 4.3 Medial view of the left elbow showing types of articular osteochondrosis. 1 – Fragmented coronoid process; 2 – osteochondritis dissecans; 3 – ununited anconeal process; 4 – ununited medial epicondyle.

- **Elbow** (Fig. 4.3)
  - *OC and OCD of the medial aspect of the humeral condyle*
  - *fragmentation or fissuring of the medial coronoid process of the ulna (FCP or coronoid disease)*
  - *ununited anconeal process (UAP)*
  - ununited medial epicondyle (UME)
- **Carpus**
  - carpal bone malformation (?)
- **Hip**
  - OC and OCD of the femoral head (rare)
  - OC of the dorsal acetabular rim (?)
  - Legg Perthes' disease (?)
- **Stifle**
  - *OC and OCD of the lateral or medial femoral condyle*
  - OC and OCD lateral (or medial) trochlear ridge (rare)
- **Hock**
  - *OC and OCD of the medial* or occasionally lateral *talar ridge*
  - medial malleolus

History and clinical signs

This condition affects the growing dog and clinical signs are most likely to develop between 4 and 7 months of age, although the patient may not be presented until the signs have been present for some time. Those dogs in which lameness develops at 2 or more years of age will not have signs because of osteochondrosis, though they may be suffering with osteoarthritis secondary to osteochondrosis. In general, the management of these older dogs is related to their osteoarthritis and only in a few cases is the typical surgery used for 'articular' osteochondrosis appropriate.

Osteochondrosis is seen predominantly in medium to large breeds of dog. Although it is often quoted in the literature that males are affected more frequently than females, this depends very much on the literature reviewed and only really holds true for shoulder osteochondrosis. In practical terms it is safest to assume that either sex may be affected. There are certain breed predispositions to the various manifestations of osteochondrosis:

• *Shoulder*	Great Dane, Border Collie, Flat Coated Retriever, Bernese Mountain Dog, Irish Wolfhound, Pyrenean Mountain Dog
• *Elbow*	
○ OCD/FCP	Labrador Retriever, Rottweiler, Golden Retriever, Bernese Mountain Dog
○ UAP	German Shepherd, Irish Wolfhound, Basset Hound
○ UME	Labrador Retriever
• *Stifle*	Labrador Retriever, Irish Wolfhound
• *Hock*	Labrador Retriever, Rottweiler, English Mastiff, English Bull Terrier

A variable degree of lameness will be seen which is usually reported to worsen with exercise. Stiffness is often seen, particularly after rest following exercise. If clinical signs are bilateral then lameness may not be the presenting feature. Instead, the dog may show a stiff/shuffling gait (especially with bilateral elbow involvement), or a crouching hindleg gait (with bilateral stifle or hock involvement) easily mistaken as being caused by hip dysplasia. Palpation of the affected joint should reveal pain towards the limits of normal joint movement. In addition, a joint effusion, periarticular thickening, a decreased range of motion and muscle atrophy may be detected depending on the joint involved and the chronicity of the pathology.

Diagnosis

A high index of suspicion should be gained from the age and breed combined with clinical evidence of joint pathology. Definitive diagnosis is achieved by imaging the joint using one or more of the following methods:

- Radiography
- Arthrography
- Arthroscopy
- Computerised tomography (CT)
- Magnetic resonance imaging (MRI)

In general, radiography is sufficient to reach a diagnosis although arthrography or arthroscopy

can provide more information about the integrity of the articular cartilage. CT and MRI scans are rarely used due to lack of availability and their cost, even though they may allow earlier, non-invasive definitive diagnosis.

Treatment and prognosis

The shoulder and stifle are large joints with plenty of room for detached fragments of cartilage and consequently many cases with osteochondrosis of these joints will recover spontaneously with conservative management, although the recovery period can be prolonged. In cases that fail to respond to conservative measures, or where a partially detached cartilage flap has been identified, surgery to remove the flap and any underrun cartilage around the edges of the lesion, is to be advocated. The elbow and hock are 'tight-fitting' joints; there is little room for fragments and their presence rapidly initiates secondary osteoarthritis. Therefore, as a general rule, early surgical removal of cartilage flaps resulting from OCD is indicated to give the best long-term functional end results, although the optimal treatment for cases with FCP/coronoid disease or UAP remains controversial.

For detailed descriptions of the diagnosis, management and prognosis of osteochondrosis as it affects specific joints the reader is referred to the relevant chapters later in the book.

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Chapter 5
Intervertebral Disc Disease and Spinal Cord Injury

Intervertebral disc disease is an extremely common condition in dogs, predominantly affecting the chondrodystrophic breeds and, in particular, the Miniature Dachshund. The clinical signs associated with disc prolapse may vary from spinal pain, through paresis to paralysis and, worst of all, loss of conscious pain sensation (CPS). Although the signs can be very dramatic in onset and severity, the condition carries a relatively good prognosis, if managed correctly, as long as CPS is present. If this is absent then urgent treatment is required but even then the prognosis is guarded.

An intervertebral disc is made up of the annulus fibrosus and the nucleus pulposus (Fig. 5.1). The nucleus pulposus (derived embryonically from the notochord) is a gel-like structure in the young animal which becomes progressively dehydrated and less gel-like with age. The annulus fibrosus has a perinuclear zone consisting of fibrocartilage and an outer zone consisting of collagen lamellae and interlamellar ground substance. The nucleus pulposus is eccentrically positioned with the ventral annulus being one-and-a-half times as thick as the dorsal annulus. Longitudinal ligaments pass dorsal and ventral to the discs. In addition to these the disc spaces between thoracic vertebrae one and ten have a conjugal ligament passing dorsal to them between the rib heads.

Degenerative disc disease

Disc degeneration takes one of two forms depending on whether the dog is of a chondrodystrophoid or non-chondrodystrophoid breed. In *chondrodystrophoid* breeds, the nucleus pulposus undergoes chondroid metamorphosis with the nucleus being gradually replaced with hyaline cartilage starting at the perinuclear zone. By 1 year of age, 90–100% of chondrodystrophoid dogs have chondroid changes in the nucleus pulposus. In *non-chondrodystrophoid* breeds, the nucleus pulposus undergoes fibroid metamorphosis with the nucleus pulposus being gradually replaced with collagenous tissue. This starts later and progresses more slowly than chondroid metamorphosis. Changes also occur in the annulus fibrosus with loosening and fragmentation of the lamellae. Following either form of metamorphosis, degeneration may occur which, in the chondroid nucleus, usually shows itself as calcification. The latter is less common in the fibroid nucleus.

Total rupture of the annulus fibrosus will allow extrusion of varying amounts of the nucleus pulposus into the vertebral canal. This is known as a *Hansen type I protrusion* and is more common in the chondrodystrophoid breeds. These protrusions have been classified further according to the amount and distribution of material extruded (Funkquist, 1962) and whether they are dorsolateral or dorsomedian, with the latter being associated with concurrent rupture of the dorsal longitudinal ligament. Partial rupture of the annulus fibrosus (the inner layers) will cause the disc to bulge causing a *Hansen type II protrusion*. These are more common in the non-chondrodystrophoid breeds. Recently the terms *intervertebral disc extrusion* (where nuclear material has escaped into the vertebral canal) and *intervertebral disc protrusion* (where the disc is bulging into the vertebral canal) have been used to classify disc 'prolapses' as an alternative to the Hansen system (Fig. 5.2).

After extrusion of disc material into the vertebral canal, continued leakage of nucleus pulposus

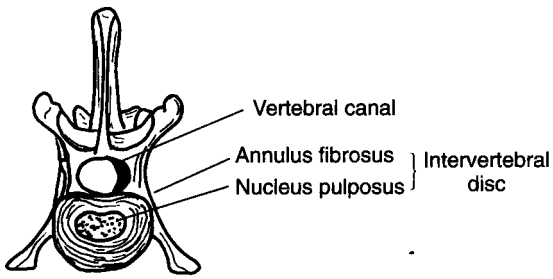

Fig. 5.1 Schematic illustration of the anatomy of an intervertebral disc.

may serve to maintain the inflammatory reaction and clinical signs. This process was termed the 'dynamic factor' by Olsson (1958).

Pathophysiology of spinal cord injury

Intervertebral disc prolapses and fracture/luxations of the vertebral column account for the majority of spinal cord injuries. Gradual onset disc prolapses, usually protrusions (type II) may not cause any signs at all since nerve tissue is capable of tolerating gradual compression far better than rapid onset compression (Kearney *et al.*, 1988). Those protrusions that do cause signs usually produce a localised myelopathy. This is thought to be a combination of malacia and demyelination resulting from not only compression of the cord itself but also, or alternatively, compression on the ventral spinal artery or venous drainage leading to cord ischaemia.

In contrast, disc extrusions and fracture luxations may cause both a concussive and compressive injury to the spinal cord and blood vessels. The concussive component results in direct (primary) injury to the spinal cord and also leads on to secondary mechanisms which cause additional tissue damage. The primary injury results in axonal disruption and/or demyelination, haemorrhagic necrosis of grey matter and a reduction in spinal cord blood flow. The secondary injury can be subdivided into metabolic and vascular mechanisms. The metabolic mechanisms are triggered by ischaemia caused by the primary injury leading to the increased production of free oxygen radicals which may exceed the capacity of natural

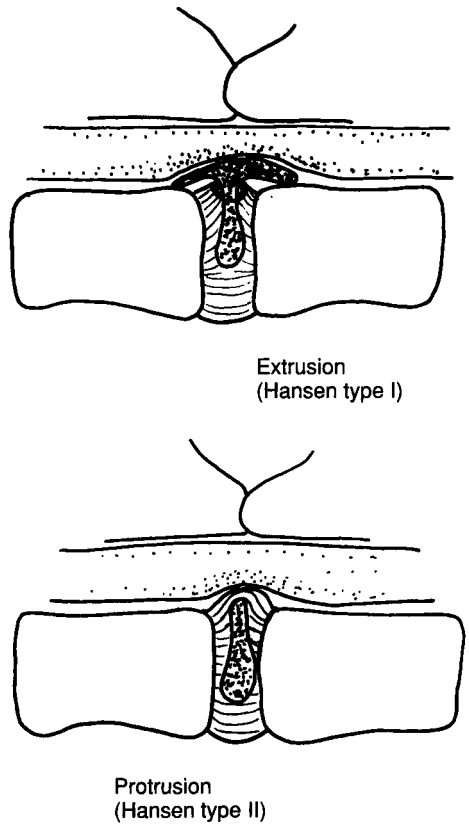

Fig. 5.2 Schematic illustration of the classification of disc prolapses.

scavenging systems. Raised levels of such radicals may then cause lipid peroxidation within the cell membranes, so reducing their integrity and leading to the release of more free oxygen radicals and also arachidonic acid. Additionally, the loss of membrane integrity and associated damage to cell membrane channels may cause raised levels of intraneuronal calcium which are cytotoxic. The free radicals may damage the microvasculature whilst the arachidonic acid may contribute to pathways involving lipoxygenase and cycloxygenase, leading to production of vasoactive leucotrienes and prostaglandins, all of which will contribute to local ischaemia. In a small number of cases suffering severe injury, the cyclic release of free radicals *may* perpetuate this autodestructive process leading to an extensive myelopathy

(ascending and descending myelomalacia). If this becomes established the changes are irreversible and euthanasia is indicated once its occurrence becomes evident since the process will always result in death if left to run its natural course (see Chapter 25, p. 247).

Vascular mechanisms add to the spinal cord ischaemia caused by the primary and secondary metabolic effects discussed above. There is a loss of autoregulation of spinal cord blood flow and release of endogenous opioids leading to systemic hypotension, both of which may further reduce the spinal cord blood flow.

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A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny, Steven J. Butterworth

Chapter 7
Osteoarthritis

Osteoarthritis (OA) is also known as osteoarthrosis or degenerative joint disease (DJD). 'Osteoarthritis' is the preferred term and, although not perfect, it does at least incorporate the inflammatory component of the disease. 'Osteoarthrosis' is used by some pathologists because they would argue that cartilage is avascular and therefore cannot have an inflammatory reaction. However, this approach disregards the important tissues of the joint other than cartilage, i.e. the synovium and subchondral bone, which are also involved in the disease process. The term DJD implies that the disease only involves degenerative mechanisms. It is clear that cartilage does have repair mechanisms and whilst they may be aberrant mechanisms in OA they are definitely active.

Osteoarthritis is the most common arthropathy of dogs (and man) and the cause of much chronic suffering in elderly animals but it may also affect some young dogs. However, it is an impossible disease to define since the term 'osteoarthritis' covers a broad spectrum of poorly understood joint disorders. Osteoarthritis is a disease which mainly affects the articular cartilage but there are also changes in the synovium and subchondral bone. The clinician's perspective of 'what OA is' may differ from that of the pathologist or radiologist. Clinically, reference may be made to a stiff, painful joint, whilst the pathologist concentrates on morphological changes in the cartilage and the veterinary radiologist identifies osteophytes and sclerosis of subchondral bone with the joint being classified as osteoarthritic without even considering the cartilage. When we consider that there is a poor correlation between what are considered significant findings in these disciplines, there is clearly a problem with defining the disease. It is currently thought that the typical changes we see on a radiograph and call 'osteoarthritis' simply represent the response of the joint to insult, i.e. the final common pathway with a variety of initiating factors.

Osteoarthritis is a heterogenous disease with assessment and staging of the disorder proving difficult. The poor correlation between radiographic and clinical data highlights this problem with a typical example of this in dogs being the dysplastic hip with secondary OA where severe radiographic changes may be present in a clinically asymptomatic joint (Fig. 7.1). This may reflect the fact that as small animal radiologists we simply assess osteophytosis and not cartilage destruction. This is not the case in man (and to some extent the horse) where weight-bearing radiographs of the knee, for example, can give a reliable indication of cartilage thickness. Expression of different facets of the disease seem to vary between individuals and even between different joints in the same individual. In small animal medicine this is exemplified by differences in osteophyte expression which clearly do not tally with the clinical picture. The current model of OA (Fig. 7.2) attempts to incorporate the heterogenetic nature of OA and how various contributing factors may interact. Overall, it is helpful to think of OA as a disease process rather than a disease entity. In trying to encompass the various aspects of the process a working definition for osteoarthritis would be:

> 'A disorder of synovial joints characterised by aberrant repair and eventual degeneration of articular cartilage and also by the formation of new bone at the articular margins, sclerosis of subchondral bone and variable low-grade synovial inflammation.'

Pathogenesis

The main tissue involved in OA is cartilage but the subchondral bone and synovium are also affected and indeed may be important in terms of disease progression. The mainstay of cartilage is the chondrocyte which produces the extracellular matrix. The matrix is composed of glycosaminoglycans (hyaluronic acid and proteoglycan) and collagens (mainly type II) which form a dense network that retains the proteoglycan. The proteoglycan is highly charged and attracts water into the tissue which makes up 75% of the cartilage. In normal cartilage there is a very slow turnover of collagens but the proteoglycan is constantly being renewed. The proteoglycans are aggregated into large molecules ('aggrecan') by means of a protein core. This core is in turn bound to hyaluronic acid chains with each chain containing many proteoglycan molecules (Fig. 7.3).

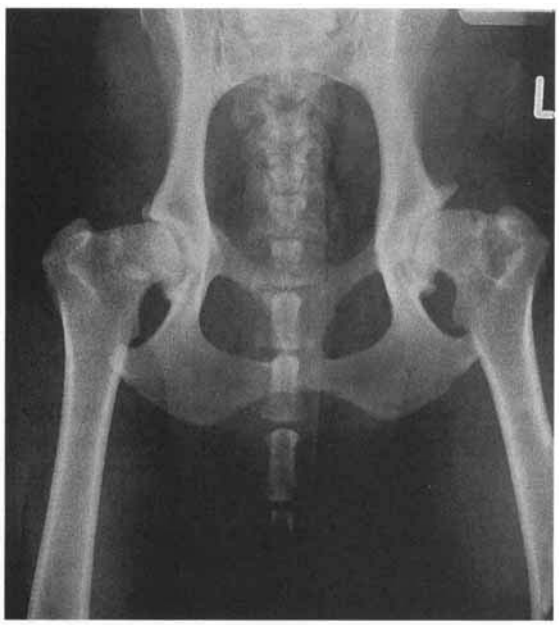


Fig. 7.1 Ventrodorsal radiograph of the pelvis of an 8-year-old Labrador presented because of hindlimb weakness. Despite a restricted range of movement in both coxofemoral joints and marked radiographic change bilaterally, the dog had never shown any lameness. The cause of its hindlimb weakness was found to be neoplasia of the spinal cord within the cervical region.

The *morphological changes* seen in OA include:

- Cartilage loss, especially in areas of increased load
- Subchondral bone sclerosis
- Marginal osteophytosis
- Variable synovial inflammation

The *biochemical changes* in the cartilage include:

- Loss of proteoglycan
- Upregulation in the degradative and synthetic activities of chondrocytes
- Increase in space between collagen fibres
- Increase in water content

These changes reduce the elasticity of the cartilage leading to fibrillation and fissuring of the cartilage with eventual loss of tissue. If this continues eburnation of subchondral bone may result. The chondrocytes themselves are upregulated and the rates of proteoglycan synthesis and degradation are increased with the overall balance towards matrix depletion. It seems likely that the activity of the chondrocytes is increased following the binding of cytokines to the cell surface. Cytokines are cellular messengers produced locally in the tissues in response to various

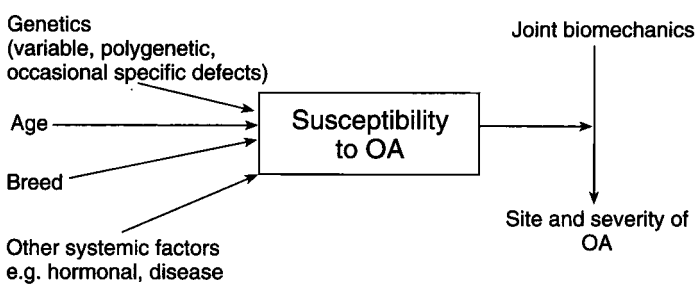


Fig. 7.2 Current model of osteoarthritis (OA) (adapted from Dieppe & Kirwan, 1994). By permission of Oxford University Press.

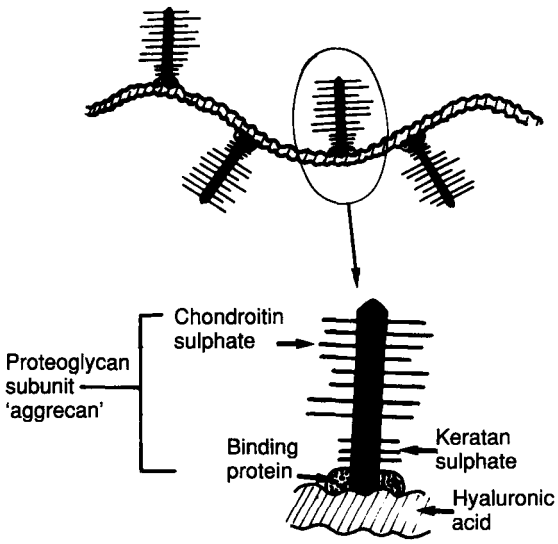


Fig. 7.3 The structure of cartilage proteoglycan.

biological stimuli such as inflammation. It is generally proposed that the cytokines responsible for stimulating cartilage degradation in OA are interleukins 1 and 6 (IL-1 and IL-6) and tumour necrosis factor-α (TNFα). These are produced from synovial cells and activated monocytes. Binding of these cytokines to a chondrocyte stimulates the production of enzymes that have been shown to be capable of degrading all the components of the cartilage matrix. The enzymes studied in most detail are the metalloproteinases – collagenase and stromelysin. Under normal circumstances the chondrocyte also produces a natural inhibitor of these enzymes known as tissue inhibitor of metalloproteinase (TIMP). TIMP production is decreased in OA. Figure 7.4 summarises the disease mechanisms in OA as discussed above. In addition, synovial cells release natural inhibitors of these cytokines such as IL-1 receptor antagonist (IL-1ra). There are also cytokines which stimulate synthesis of matrix and likely candidates for these include the insulin-like growth factors 1 and 2 (IGF-1 and IGF-2) and transforming growth factor-β (TGFβ).

Some other experimental evidence suggests that these intercellular mechanisms are relatively unimportant if the mechanical environment of the chondrocyte is abnormal. This is based on work using the cruciate deficient canine stifle with and without synovial inflammation. In these studies the damage to the articular cartilage was the same irrrespective of synovial activity.

In summary, like many body tissues, cartilage exists in equilibrium between anabolism and catabolism. In OA the balance is shifted towards breakdown. Clearly there are possibilities for intervention in these mechanisms and these are discussed later (p. 59).

Classification of canine OA

Whilst at present OA is classified as primary (idiopathic), secondary, or erosive (atrophic), it seems likely that this classification will continue to evolve as understanding of the disease progresses. It may be that the classification draws unwarranted boundaries, or conversely is grossly oversimplified. For example, human rheumatologists would now consider hip OA separately from knee OA because there are different sets of risk factors for the two 'diseases'. Indeed OA in different sites within the same joint may have different initiating factors and dominant disease mechanisms. We are not at this stage in veterinary rheumatology but these developments and changes in the way we think about the disease will undoubtedly come.

Primary (idiopathic) OA

The term primary OA, which is used to describe disease where no initiating factor can be identified, is losing favour. This is because it is likely that there are, as yet unidentified, factors and therefore the term idiopathic OA is to be preferred. Idiopathic OA is not common in dogs and usually one can identify a cause for the degenerative changes. However, a generalised disease is occasionally seen in certain breeds such as the Chow Chow, Dalmatian, Labrador Retriever and Spaniels. This is usually a symmetrical disease affecting, for example, both carpi, both stifles or both elbows.

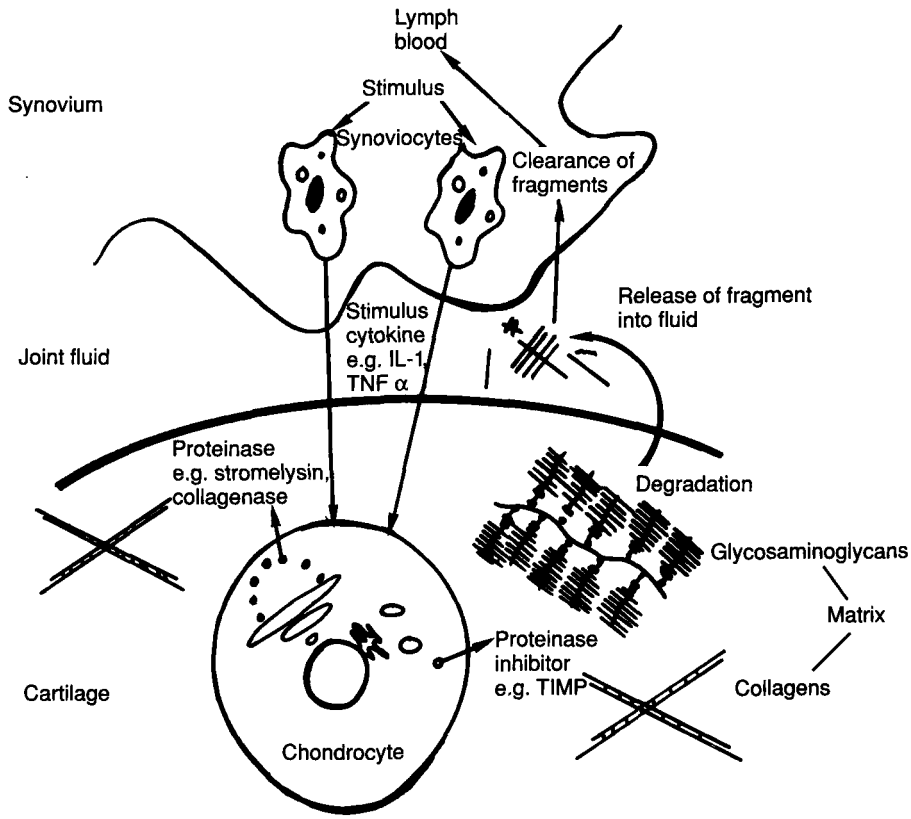


Fig. 7.4 Diagram illustrating matrix turnover in cartilage (based on Lohmander, 1992).

Secondary OA

This would seem to be the most common form of OA in dogs and frequently recognised causes are listed below:

- Osteochondrosis
- Hip dysplasia
- Cruciate disease
- Collateral ligament damage
- Joint luxation
- Articular fractures
- Legg Perthes' disease
- Other forms of arthritis (e.g. immune-mediated)

It may be that the OA is not the major consideration at the time of presentation and the primary disease may be more pressing (e.g. cruciate disease) but there will be other cases where the contribution of the OA may be more important (e.g. hip dysplasia with secondary change in a mature dog).

Erosive (atrophic) OA

Occasionally canine OA joints may show local erosive changes on a radiograph. The cause of this change is not known but in humans it has been associated with deposits of basic calcium phosphate crystals of the apatite type. The role of crystals in canine OA is unexplored. Whether this erosive change represents a different subset of OA or merely one end of a spectrum of change remains unclear. However, it emphasises the need for further diagnostic work apart from radiography. Erosive changes in a joint may be seen

also in diseases such as rheumatoid arthritis and infectious arthritis.

Diagnosis

History and clinical examination

The typical OA patient presents with lameness or stiffness. However, in cases of secondary OA signs may be related mainly to the underlying disease. It is important to remember that the course of OA is variable with some osteoarthritic joints remaining clinically silent and non-progressive, and some being very painful and rapidly progressive. Usually, though, the lameness is chronic in nature and insidious in onset. The signs may be worse following exercise and may be particularly noticeable after a period of rest following exercise. Stiffness after rest usually persists for a matter of minutes only. Cold and damp weather may exacerbate the clinical signs.

The osteoarthritic joint may be palpably thickened due to capsular fibrosis and/or osteophyte production. The range of motion of the joint may be decreased in association with this. It may be possible to detect a moderate joint effusion and manipulation of the joint may reveal crepitation and a pain response.

Occasionally dogs will present with severe lameness of acute onset. This may be due to a so-called acute 'flare-up' of OA. The reasons for this scenario are obscure but may be related to an inflammatory phase of the disease or fracture of osteophytes. One should always be careful to look for another complicating factor such as ligament damage or infection.

Radiography

The pattern of radiographic changes in OA varies between individuals and between joints. It may also vary with the 'type' of OA within a particular joint. In small animal radiology the main features of radiographic OA are osteophytosis, subchondral sclerosis and intra-articular mineralisation, with joint effusion a feature in some joints, and erosive changes a rare observation (Fig. 7.5). The radiographs should always be examined for signs of an underlying disease which may have initiated the OA.

Osteophytes tend to arise at the joint margin often where the joint capsule attaches, e.g. the femoral neck in hip OA. New bone formation may also occur at the attachment of ligaments or tendons when it is termed enthesiophytosis, e.g. distal pole of the patella in stifle OA. Each joint tends to have its own specific pattern with respect to osteophyte formation (Fig. 7.6).

Subchondral bone sclerosis tends to be seen in long-standing cases. However, this probably only reflects the insensitivity of the standard radiograph in detecting this change. Microfocal radiographical studies have shown that this change is not the reserve of chronic OA.

Intra- or periarticular mineralisation is a fairly frequent finding in OA. The deposits may be in cartilage, synovium, tendons, ligaments or menisci. Studies in man have revealed that there may be several mechanisms at work in the formation of these deposits and their importance is unknown.

Erosive changes in subchondral bone may occasionally be seen in canine OA. A more common example of this type of OA in veterinary medicine would be the erosive type of bone spavin seen in the horse. Erosive lesions vary in size and shape and may be difficult to distinguish from subchondral cysts. The latter are another occasional feature of OA but are more often seen in man and horses than dogs. They have a sharp border and may appear to communicate with the joint space.

Arthrocentesis

Synovial fluid from osteoarthritic joints tends to be found in normal or increased volumes, with normal or low viscosity (resulting from effusion rather than a qualitative change in the hyaluronic acid content), and a total cell count of up to 5×10^9/l with less than 5% polymorphs (though both of these parameters may be elevated in joints showing an acute flare-up). These features can prove useful in helping to differentiate from other types of joint pathology (see Table 6.1). It may be that in the future synovial fluid becomes much more important in terms of disease diagnosis, estimating prognosis and possibly monitoring treatment.

Further diagnostic techniques

Arthroscopy

Arthroscopy is becoming more commonplace in small animal orthopaedics. The real benefits in canine cases have yet to be proven, particularly with respect to OA. Human rheumatologists are already starting to use arthroscopy in the diagnosis and staging of OA but whether it will become commonplace in this setting remains to be seen.

Scintigraphy

Scintigraphy involves the use of a radioisotope (usually ^{99m}technetium methylene diphosphonate) to produce an image of bone activity using a gamma camera. It has long been recognised that OA joints retain isotope but it has recently been shown in human studies that scintigraphy predicts subsequent radiographic OA. This is, therefore, the first technique identified that may give an indication regarding the outcome of OA.

Ultrasound

The use of ultrasound in OA has been limited because of the problems of definition. Newer, high-frequency, high-resolution probes have shown some usefulness in assessing articular cartilage.

Magnetic resonance imaging (MRI)

Magnetic resonance imaging (MRI) is certainly unavailable to the vast majority of veterinary clinics at the moment. In human medicine, work is concentrating on using MRI to detect

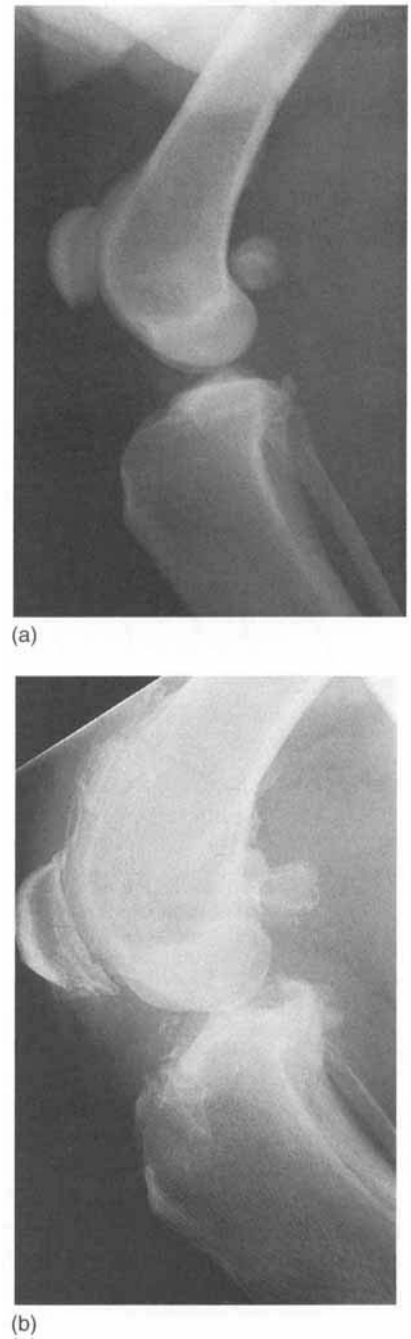
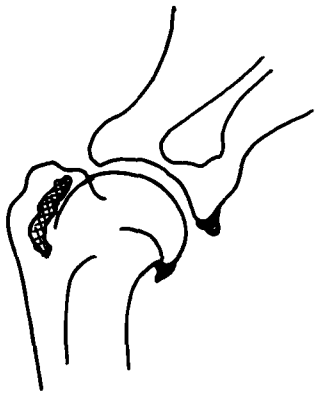
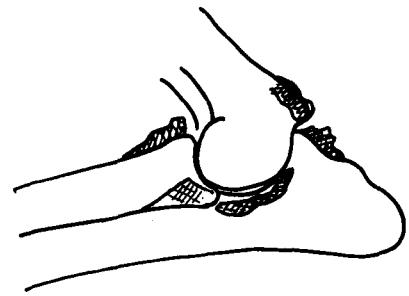


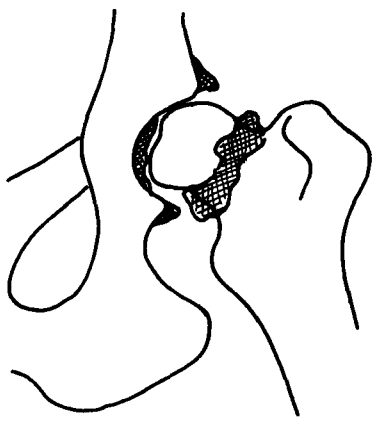
Fig. 7.5 Radiographs illustrating the typical changes seen in stifle osteoarthritis (OA) secondary to cranial cruciate ligament rupture/disease.
(a) Mediolateral radiograph of a normal stifle for comparison.
(b) Mediolateral radiograph showing osteophytosis alongside the trochlear ridges, proximal to the trochlear groove and on the tibial plateau. Enthesiophytes are present on the poles of the patella. The presence of a joint effusion is suggested by partial loss of the infrapatellar fat pad shadow and caudal distension of the joint capsule. There is some evidence of subchondral sclerosis within the distal femur but such radiodensity could also be explained by superimposition of osteophytes upon the normal bone density rather than an increase in the bone density itself.



Shoulder:
Caudal humeral head
Caudal rim of glenoid
Intertubercular groove



Elbow:
Caudal aspect of anconeal process
Coronoid process (seen better on mediolateral view)
Alongside semilunar notch
Medial humeral epicondyle



Hip:
Around acetabular rim
Within acetabular fossa
Around femoral neck

Stifle:
Proximal to trochlear groove
Alongside trochlear ridges
Poles of patella (enthesiophytes)
Tibial plateau
Around fibular head

Fig. 7.6 Diagrams illustrating the predilecion sites for osteophyte formation (hatched areas) in the proximal joints of the fore- and hindlimb.

very early changes in the cartilage. Whilst MRI promises to become the gold standard in imaging joints, there are problems with resolution and also defining the calcified zone of articular cartilage.

Biochemical markers of joint disease

Much research is concentrating on biochemical methods to detect changes in joint tissues. The hope is that these tests will allow changes to be

detected early and allow targeting of patients for disease-modifying therapy. They might also allow the effectiveness of such treatments to be monitored.

Treatment

Conservative management

Exercise

The activity of the dog will have an effect on the course of the disease. When acute flare-ups occur then complete rest for a period of 10–14 days seems to allow the majority of these to settle. The long-term activity of a dog with problematic OA will have to be altered. Short lead walks seem to be preferable with adequate periods of rest allowed should signs worsen.

Diet

Obesity is to be avoided. In human OA it has been shown that obesity is a major contribution to progression of knee OA but this is not the case for hip OA. The variability of risk factors for canine OA at different sites is not known. However, anecdotally most obese dogs suffering with clinical OA seem to improve if weight is reduced.

Medical treatment

There are a variety of approaches for medical treatment of OA but the agents fall into three broad categories:

- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Corticosteroids
- Slow-acting drugs in OA (SADOA)

Non-steroidal anti-inflammatory drugs (NSAIDs)

These are the mainstay of medical treatment of OA in small animal medicine. Whilst the rationale for their use is to relieve signs there has been much work on the effect these drugs may have on joint tissues. One reason for using NSAIDs was the premise that synovial inflammation would be decreased. However, the symptomatic relief afforded to human OA patients with a variety of prostaglandin-blocking NSAIDs has been shown to be no better than the use of a simple analgesic such as acetaminophen (paracetamol). There are several NSAIDs licensed for use in the dog and these are mostly prostaglandin-blocking. Carprofen (Rimadyl, Pfizer) is a prostaglandin-sparing NSAID with an obscure mode of action, but it may have some parallels with acetaminophen in that it too seems to be effective in the relief of OA pain.

There has been much debate on the effects NSAIDs have on cartilage. There is some evidence that some of this group have a deleterious effect on cartilage when compared to others in the group; equally some are heralded as chondroprotective. Much of this work has been carried out *in vitro* and extrapolation to the *in vivo* situation is difficult. The decrease in synovitis may be negated by the analgesic-induced overloading of a joint. Add to this the effect a drug may have on chondrocyte metabolism and clearly the situation is complex. However, the effects in the long term are generally felt to be minimal.

Several factors may influence the choice of drug:

- Clinical response
- Side-effects (important if there is concurrent hepatic, gastrointestinal or renal disease)
- Cost
- Type of preparation (capsules, tablets, drops)
- Convenience of dosing regime

Table 7.1 provides a list of commonly used products available in the UK for the treatment of OA. The products will vary in their effectiveness between individuals in keeping with the heterogenetic nature of OA.

It would seem unnecessary to consider the use of unlicensed products in the treatment of canine OA now that there is an adequate range of licensed drugs. NSAIDs available for use in humans without prescription, such as ibuprofen, should be avoided since their half-lives are much longer in the dog, compared with humans, and so it is easy to overdose. Flurbiprofen should

Table 7.1 Non-steroidal anti-inflammatory drugs (NSAIDs) used in the treatment of canine and feline osteoarthritis (OA).

NSAID	Patient	Dose
Aspirin	dog	25 mg/kg/day in divided doses (not licensed)
	cat	10–20 mg/kg every 2–3 days (not licensed)
Phenylbutazone (Phenycare)	dog	2–20 mg/kg/day in divided doses
	cat	1–5 mg/kg/day
Carprofen (Rimadyl)	dog	2–4 mg/kg/day in two doses for up to 7 days, 2 mg/kg/day as single dose thereafter
Ketoprofen (Ketofen)	dog	1 mg/kg/day for up to 5 days
	cat	1 mg/kg/day for up to 5 days
Tolfenamic acid (Tolfedine)	dog	4 mg/kg/day for up to 3 days
	cat	4 mg/kg single injection (upper respiratory tract disease only)
Meloxicam (Metacam)	dog	0.2 mg/kg/day as single dose on day 1 and 0.1 mg/kg/day or less thereafter
Flunixin (Finadyne)	dog	1 mg/kg/day for up to 3 days contraindicated in pregnant bitches
Mefenamic acid (Ponstan)	dog	10–30 mg/kg/day in divided doses (not licensed)
Piroxicam (Feldene)	dog	0.3 mg/kg every 48 hours (not licensed)
Prednisolone, **Cinchophen**, **Hexamine** (Predno-Leucotropin)	dog	1–2 tablets/10 kg/day in divided doses
	cat	0.5 tablet twice daily

Further information for those that are licensed can be found in the corresponding data sheets published annually as *Compendium of Data Sheets for Veterinary Products* by National Office of Animal Health Ltd.

definitely not be used since it may lead to an idiosyncratic reaction and death.

All NSAIDs may cause gastrointestinal irritation resulting in vomiting or diarrhoea (possibly with blood). Recently the enzyme cyclo-oxygenase (COX) has been shown to have two isomers. COX1 is responsible for 'house keeping' functions and COX2 is induced in inflammatory conditions. It has been proposed that NSAIDs acting selectively on COX2 will have a lower incidence of side-effects and this has been supported by research study results. Of the drugs available, meloxicam (metacam, Boehringer Ingelheim Ltd.) and carprofen (Rimadyl, Pfizer) show most promise.

If severe side-effects occur the drug should be withdrawn. To try and avoid such side-effects these drugs are best given with some food. Flunixin may cause severe haemorrhagic gastroenteritis and the 3-day limit should not be exceeded. If such side-effects result in withdrawal of a drug then, once the signs have settled down, another one may be tried. Particular dogs are often sensitive to certain agents.

'Trial and error' will determine a suitable drug for an individual dog.

Corticosteroids

The use of corticosteroids in the treatment of OA is another controversial area. Some work in experimental OA suggested that corticosteroids hasten the degeneration of cartilage and this evidence seemed to make the use of corticosteroids (particularly intra-articular use) contraindicated. However, there is recent evidence that intra-articular (I/A) methylprednisolone (Depo-Medrone V, Pharmacia & Upjohn), at a dose of 20 mg for an average Labrador's shoulder or stifle, may have beneficial effects in canine OA. Certainly there may be dramatic relief from clinical signs although the response is variable. Human rheumatologists may repeat intra-articular injections up to three times a year. The response to I/A corticosteroid seems to vary depending on the joint. It is probably important to rest the patient strictly for at least 2 days following the injection to encourage the drug to stay within the joint and

limit the systemic effect. If corticosteroids are to be administered systemically, low-dose prednisolone (0.25–0.5 mg/kg once daily or every other day) is the preferred drug. A course of 2–3 weeks should be used initially.

Corticosteroids seem to be particularly helpful in certain situations, namely:

- When other medical treatments have failed and the dog's quality of life has declined
- In elderly dogs where there is concern over NSAID side-effects
- After an acute flare-up of OA, which may be associated with a high level of polymorphs (15–30%) in synovial fluid
- In erosive OA

Slow-acting drugs in OA (SADOA)

The International League Against Rheumatism (ILAR) has proposed two categories in this group: disease-modifying OA drugs (DMOADS) and symptomatic slow-acting drugs in OA (SYSADOA). This classification aims to clearly define the concept of 'chondroprotection' and distinguish drugs that have this property from those that merely give symptomatic relief. The concept of chondroprotection evolved from clinical trials of certain agents which appeared to retard the progression of OA. However, the pharmacology of these drugs could not be related to the pathology of OA and hence there is a need for a much tighter definition of chondroprotective drugs.

There is some evidence that certain agents can alter the environment or activity of the chondrocyte *in vitro* such that the degeneration of cartilage is stopped or decreased. Unfortunately, although *in vitro* results may suggest that certain agents have these capabilities, *in vivo* work is hampered by the fact that there are, at present, no readily quantifiable criteria for measuring outcome of OA. This is even more applicable in clinical trials, many of which have been uncontrolled and poorly designed. Thus, there is much conflicting evidence in the literature.

At present there is only one drug in this class licensed for use in the dog. This is sodium pentosan polysulphate (PPS) (Cartrophen-Vet, Arthropharm, Australia) which is a polysaccharide sulphate ester prepared from beech hemicellulose. This drug has been shown to inhibit certain proteinases but it is structurally similar to heparin and so has anticoagulant properties. The mode of action in clinical cases is obscure and recent studies suggest that the fibrinolytic action may shift microthrombi in the vascular supply of subchondral bone resulting in pain relief. Validated clinical trials with the drug are lacking but anecdotal evidence suggests that it can give impressive clinical results in certain cases. However, suitable case selection is a large stumbling block. An oral preparation of PPS has been used experimentally in dogs and is licensed in some countries but not yet in the UK.

More recently the terms 'structure modifying' and 'symptom modifying' drugs have been used to simplify the terminology whereby PPS would be viewed as an example of the former and a NSAID of the latter, although some NSAIDs are also claimed to have structure-modifying properties.

Dietary supplementation

Recently there have been a number of dietary supplements put onto the market which claim to be able to modify the course of osteoarthritis. These generally contain chondroitin sulphate (e.g. Cartivet, Arnolds) or this together with glucosamine (e.g. Cosequin, Xeipon). Their possible methods of action have been suggested to include an anti-inflammatory action and also an indirect influence on the viscosity of synovial fluid by inhibiting the action of hyaluronidase and/or increasing the rate of hyaluronic acid production. Thus far, there is no scientific evidence to support their use although there are certainly anecdotal reports to suggest that they may help in particular cases. Because of this the authors do not generally recommend their use but would not oppose an owner's wishes to try out such a preparation since they are, at the very least, unlikely to be harmful to the osteoarthritic joint or patient.

Future possibilities for disease modification

There are many avenues of research in the

treatment of OA. Certain forms of treatment have been around for some time but have yet to be proven useful. A good example of this would be polysulphated glycosaminoglycan preparations used for so-called *viscosupplementation*. These have been licensed, in various forms, for treatment of human and equine arthritis for some time (e.g. Adequan, Janssen Animal Health). Due to the difficulties in quantifying outcome in OA, their effectiveness in clinical trials is still variable. A recent study showed no difference between patients receiving intra-articular hyaluronic acid and those receiving intra-articular saline. The explanation for this might lie in reports on the usefulness of articular lavage in the symptomatic relief of OA in humans. Clearly any preparation delivered after arthrocentesis would have to take this possible effect into account.

The theories of viscosupplementation revolve around improving the properties of the synovial fluid. This may have many effects such as decreasing abnormal load on cartilage or altering inflammatory mechanisms. There may be more efficacy in newer formulations of hyaluronic acid which have a high degree of cross linking which creates a very high molecular weight and improves the viscoelasticity.

Other medical developments are centred on attempting to *alter the activity of chondrocytes* by blocking the action of certain cytokines, for example, IL-1-blocking agents are reaching the clinical trial stage. Cytokines which stimulate synthetic activity of chondrocytes have also received attention, for example IGF-1 has been used experimentally (in combination with PPS) and shown to have some promise in maintaining matrix integrity.

Gene therapy is a concept receiving attention throughout medicine. Recent experimental work has shown that the gene for IL-1ra can be implanted into synovial cells in joints and these cells will express this gene for several weeks. Clearly there is exciting potential in such an approach to arthritis.

Surgical management

There are several potential aims of surgery in managing an osteoarthritic joint:

- To attend to the initiating cause
- To alleviate certain sources of pain caused by the osteoarthritic process
- To remove or replace the osteoarthritic joint

Other than when surgery is aimed at eliminating the initiating cause of the OA, it is generally reserved for those cases that show an unsatisfactory response to the conservative and medical measures detailed above. The options available vary with different joints and the surgical management of OA for each joint is discussed in detail within the relevant chapters of Sections 5 and 6. A general overview of what may be achieved will, however, be considered here.

Cheilectomy

Cheilectomy involves the debridement of the joint with the removal of osteophytes or 'loose bodies', possibly in combination with synovectomy. Although there is no hard evidence on the beneficial effects of such a procedure, many cases will improve for a variable period of time afterwards. The joint in which this is, perhaps, most appropriate for assisting management of OA would be the stifle.

Metaphyseal forage

Metaphyseal forage involves drilling across the metaphyseal scar(s) adjacent to the joint in question. The procedure has evolved from the pain relief reported after the performance of metaphyseal osteotomies in human patients. One belief is that venous stasis in the subchondral bone creates ischaemia and is a source of pain. Metaphyseal forage may improve the blood flow through this region, thereby reducing any such ischaemia. The exact mechanism by which this procedure may have a beneficial effect is yet to be elucidated but there are reports of such an effect being maintained for several months. It may be carried out alone or in combination with cheilectomy. Surgeons who use this technique might apply it, in decreasing likelihood, to the stifle, elbow or hip joint.

Joint replacement

Joint replacement is an ideal way to treat an end-stage osteoarthritic joint. At present a prosthesis is only available for replacement of the coxofemoral joint in the dog. The development of components for other joints is currently underway and the elbow is most likely to be the next in line.

Excision arthroplasty

Excision arthroplasty may be used as a salvage procedure in certain joints such as the hip. The success of such techniques depends on several factors such as the joint involved and the size of the patient.

Arthrodesis

Arthrodesis may be employed in appendicular joints other than the hip when end-stage OA cannot be controlled by other means. The success of such a procedure varies between the joints with the carpus being an example of a joint that can be arthrodesed with very good results, whereas fusion of an elbow or stifle will present far more mechanical problems for the dog.

Cartilage grafts

Cartilage grafts might offer a potential treatment for patients with advanced OA. Much work is being directed at trying to 'resurface' osteoarthritic joints with hyaline cartilage from either a donor or from tissue culture. Although this technique is not yet available, and there remain several problems regarding its application, it might provide an alternative method of management in the future. The topic of cartilage grafting is also discussed in Chapter 2 (p. 21).

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A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny, Steven J. Butterworth

Chapter 7
Osteoarthritis

Osteoarthritis (OA) is also known as osteoarthrosis or degenerative joint disease (DJD). 'Osteoarthritis' is the preferred term and, although not perfect, it does at least incorporate the inflammatory component of the disease. 'Osteoarthrosis' is used by some pathologists because they would argue that cartilage is avascular and therefore cannot have an inflammatory reaction. However, this approach disregards the important tissues of the joint other than cartilage, i.e. the synovium and subchondral bone, which are also involved in the disease process. The term DJD implies that the disease only involves degenerative mechanisms. It is clear that cartilage does have repair mechanisms and whilst they may be aberrant mechanisms in OA they are definitely active.

Osteoarthritis is the most common arthropathy of dogs (and man) and the cause of much chronic suffering in elderly animals but it may also affect some young dogs. However, it is an impossible disease to define since the term 'osteoarthritis' covers a broad spectrum of poorly understood joint disorders. Osteoarthritis is a disease which mainly affects the articular cartilage but there are also changes in the synovium and subchondral bone. The clinician's perspective of 'what OA is' may differ from that of the pathologist or radiologist. Clinically, reference may be made to a stiff, painful joint, whilst the pathologist concentrates on morphological changes in the cartilage and the veterinary radiologist identifies osteophytes and sclerosis of subchondral bone with the joint being classified as osteoarthritic without even considering the cartilage. When we consider that there is a poor correlation between what are considered significant findings in these disciplines, there is clearly a problem with defining the disease. It is currently thought that the typical changes we see on a radiograph and call 'osteoarthritis' simply represent the response of the joint to insult, i.e. the final common pathway with a variety of initiating factors.

Osteoarthritis is a heterogenous disease with assessment and staging of the disorder proving difficult. The poor correlation between radiographic and clinical data highlights this problem with a typical example of this in dogs being the dysplastic hip with secondary OA where severe radiographic changes may be present in a clinically asymptomatic joint (Fig. 7.1). This may reflect the fact that as small animal radiologists we simply assess osteophytosis and not cartilage destruction. This is not the case in man (and to some extent the horse) where weight-bearing radiographs of the knee, for example, can give a reliable indication of cartilage thickness. Expression of different facets of the disease seem to vary between individuals and even between different joints in the same individual. In small animal medicine this is exemplified by differences in osteophyte expression which clearly do not tally with the clinical picture. The current model of OA (Fig. 7.2) attempts to incorporate the heterogenetic nature of OA and how various contributing factors may interact. Overall, it is helpful to think of OA as a disease process rather than a disease entity. In trying to encompass the various aspects of the process a working definition for osteoarthritis would be:

> 'A disorder of synovial joints characterised by aberrant repair and eventual degeneration of articular cartilage and also by the formation of new bone at the articular margins, sclerosis of subchondral bone and variable low-grade synovial inflammation.'

Pathogenesis

The main tissue involved in OA is cartilage but the subchondral bone and synovium are also affected and indeed may be important in terms of disease progression. The mainstay of cartilage is the chondrocyte which produces the extracellular matrix. The matrix is composed of glycosaminoglycans (hyaluronic acid and proteoglycan) and collagens (mainly type II) which form a dense network that retains the proteoglycan. The proteoglycan is highly charged and attracts water into the tissue which makes up 75% of the cartilage. In normal cartilage there is a very slow turnover of collagens but the proteoglycan is constantly being renewed. The proteoglycans are aggregated into large molecules ('aggrecan') by means of a protein core. This core is in turn bound to hyaluronic acid chains with each chain containing many proteoglycan molecules (Fig. 7.3).

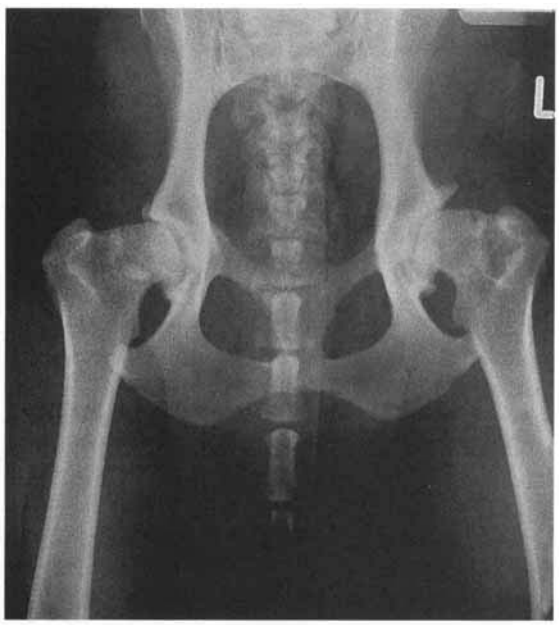


Fig. 7.1 Ventrodorsal radiograph of the pelvis of an 8-year-old Labrador presented because of hindlimb weakness. Despite a restricted range of movement in both coxofemoral joints and marked radiographic change bilaterally, the dog had never shown any lameness. The cause of its hindlimb weakness was found to be neoplasia of the spinal cord within the cervical region.

The *morphological changes* seen in OA include:

- Cartilage loss, especially in areas of increased load
- Subchondral bone sclerosis
- Marginal osteophytosis
- Variable synovial inflammation

The *biochemical changes* in the cartilage include:

- Loss of proteoglycan
- Upregulation in the degradative and synthetic activities of chondrocytes
- Increase in space between collagen fibres
- Increase in water content

These changes reduce the elasticity of the cartilage leading to fibrillation and fissuring of the cartilage with eventual loss of tissue. If this continues eburnation of subchondral bone may result. The chondrocytes themselves are upregulated and the rates of proteoglycan synthesis and degradation are increased with the overall balance towards matrix depletion. It seems likely that the activity of the chondrocytes is increased following the binding of cytokines to the cell surface. Cytokines are cellular messengers produced locally in the tissues in response to various

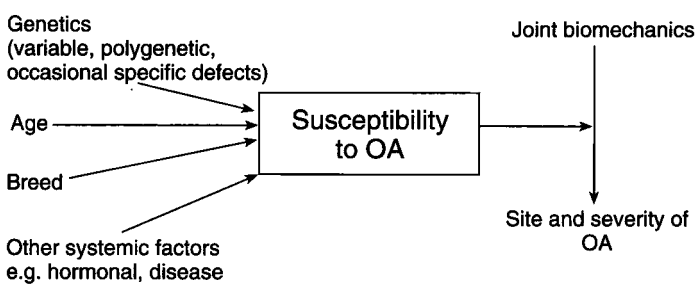


Fig. 7.2 Current model of osteoarthritis (OA) (adapted from Dieppe & Kirwan, 1994). By permission of Oxford University Press.

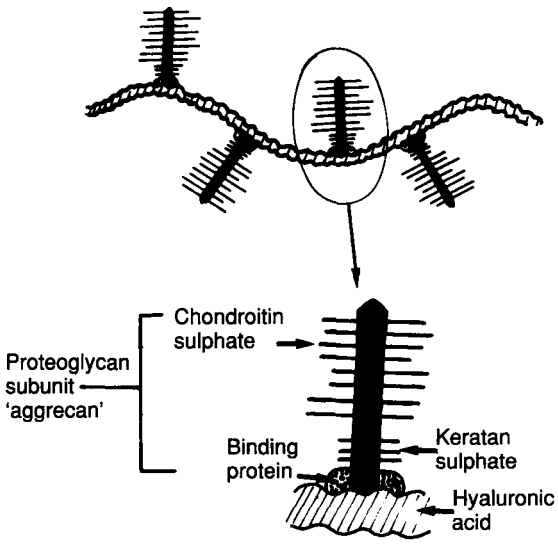


Fig. 7.3 The structure of cartilage proteoglycan.

biological stimuli such as inflammation. It is generally proposed that the cytokines responsible for stimulating cartilage degradation in OA are interleukins 1 and 6 (IL-1 and IL-6) and tumour necrosis factor-α (TNFα). These are produced from synovial cells and activated monocytes. Binding of these cytokines to a chondrocyte stimulates the production of enzymes that have been shown to be capable of degrading all the components of the cartilage matrix. The enzymes studied in most detail are the metalloproteinases – collagenase and stromelysin. Under normal circumstances the chondrocyte also produces a natural inhibitor of these enzymes known as tissue inhibitor of metalloproteinase (TIMP). TIMP production is decreased in OA. Figure 7.4 summarises the disease mechanisms in OA as discussed above. In addition, synovial cells release natural inhibitors of these cytokines such as IL-1 receptor antagonist (IL-1ra). There are also cytokines which stimulate synthesis of matrix and likely candidates for these include the insulin-like growth factors 1 and 2 (IGF-1 and IGF-2) and transforming growth factor-β (TGFβ).

Some other experimental evidence suggests that these intercellular mechanisms are relatively unimportant if the mechanical environment of the chondrocyte is abnormal. This is based on work using the cruciate deficient canine stifle with and without synovial inflammation. In these studies the damage to the articular cartilage was the same irrrespective of synovial activity.

In summary, like many body tissues, cartilage exists in equilibrium between anabolism and catabolism. In OA the balance is shifted towards breakdown. Clearly there are possibilities for intervention in these mechanisms and these are discussed later (p. 59).

Classification of canine OA

Whilst at present OA is classified as primary (idiopathic), secondary, or erosive (atrophic), it seems likely that this classification will continue to evolve as understanding of the disease progresses. It may be that the classification draws unwarranted boundaries, or conversely is grossly oversimplified. For example, human rheumatologists would now consider hip OA separately from knee OA because there are different sets of risk factors for the two 'diseases'. Indeed OA in different sites within the same joint may have different initiating factors and dominant disease mechanisms. We are not at this stage in veterinary rheumatology but these developments and changes in the way we think about the disease will undoubtedly come.

Primary (idiopathic) OA

The term primary OA, which is used to describe disease where no initiating factor can be identified, is losing favour. This is because it is likely that there are, as yet unidentified, factors and therefore the term idiopathic OA is to be preferred. Idiopathic OA is not common in dogs and usually one can identify a cause for the degenerative changes. However, a generalised disease is occasionally seen in certain breeds such as the Chow Chow, Dalmatian, Labrador Retriever and Spaniels. This is usually a symmetrical disease affecting, for example, both carpi, both stifles or both elbows.

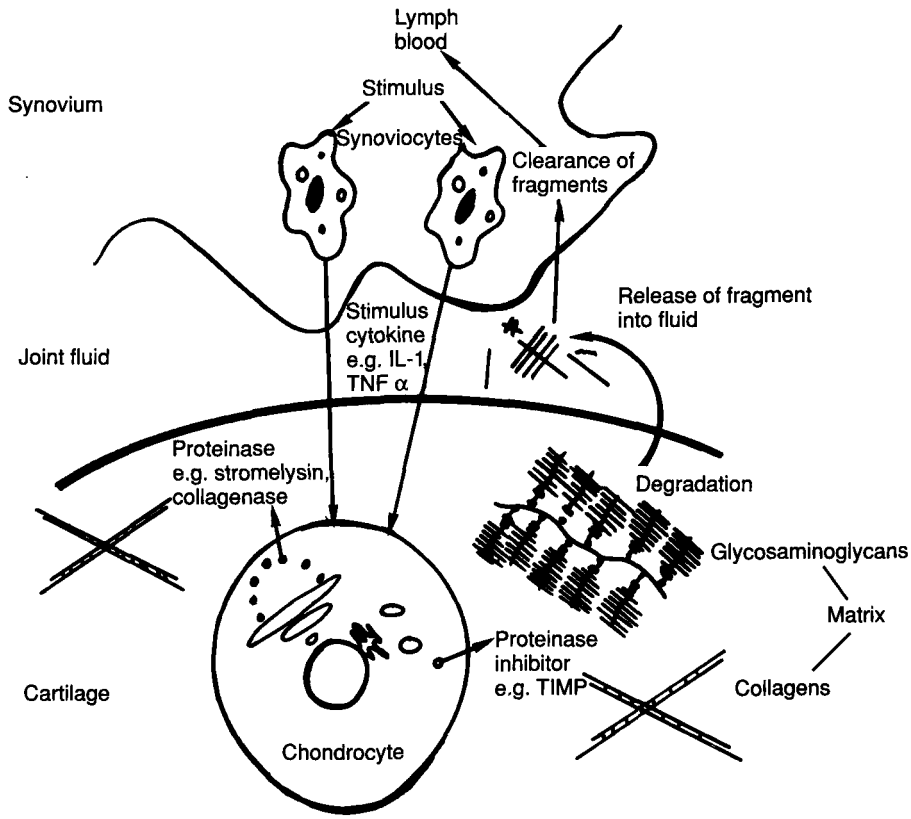


Fig. 7.4 Diagram illustrating matrix turnover in cartilage (based on Lohmander, 1992).

Secondary OA

This would seem to be the most common form of OA in dogs and frequently recognised causes are listed below:

- Osteochondrosis
- Hip dysplasia
- Cruciate disease
- Collateral ligament damage
- Joint luxation
- Articular fractures
- Legg Perthes' disease
- Other forms of arthritis (e.g. immune-mediated)

It may be that the OA is not the major consideration at the time of presentation and the primary disease may be more pressing (e.g. cruciate disease) but there will be other cases where the contribution of the OA may be more important (e.g. hip dysplasia with secondary change in a mature dog).

Erosive (atrophic) OA

Occasionally canine OA joints may show local erosive changes on a radiograph. The cause of this change is not known but in humans it has been associated with deposits of basic calcium phosphate crystals of the apatite type. The role of crystals in canine OA is unexplored. Whether this erosive change represents a different subset of OA or merely one end of a spectrum of change remains unclear. However, it emphasises the need for further diagnostic work apart from radiography. Erosive changes in a joint may be seen

also in diseases such as rheumatoid arthritis and infectious arthritis.

Diagnosis

History and clinical examination

The typical OA patient presents with lameness or stiffness. However, in cases of secondary OA signs may be related mainly to the underlying disease. It is important to remember that the course of OA is variable with some osteoarthritic joints remaining clinically silent and non-progressive, and some being very painful and rapidly progressive. Usually, though, the lameness is chronic in nature and insidious in onset. The signs may be worse following exercise and may be particularly noticeable after a period of rest following exercise. Stiffness after rest usually persists for a matter of minutes only. Cold and damp weather may exacerbate the clinical signs.

The osteoarthritic joint may be palpably thickened due to capsular fibrosis and/or osteophyte production. The range of motion of the joint may be decreased in association with this. It may be possible to detect a moderate joint effusion and manipulation of the joint may reveal crepitation and a pain response.

Occasionally dogs will present with severe lameness of acute onset. This may be due to a so-called acute 'flare-up' of OA. The reasons for this scenario are obscure but may be related to an inflammatory phase of the disease or fracture of osteophytes. One should always be careful to look for another complicating factor such as ligament damage or infection.

Radiography

The pattern of radiographic changes in OA varies between individuals and between joints. It may also vary with the 'type' of OA within a particular joint. In small animal radiology the main features of radiographic OA are osteophytosis, subchondral sclerosis and intra-articular mineralisation, with joint effusion a feature in some joints, and erosive changes a rare observation (Fig. 7.5). The radiographs should always be examined for signs of an underlying disease which may have initiated the OA.

Osteophytes tend to arise at the joint margin often where the joint capsule attaches, e.g. the femoral neck in hip OA. New bone formation may also occur at the attachment of ligaments or tendons when it is termed enthesiophytosis, e.g. distal pole of the patella in stifle OA. Each joint tends to have its own specific pattern with respect to osteophyte formation (Fig. 7.6).

Subchondral bone sclerosis tends to be seen in long-standing cases. However, this probably only reflects the insensitivity of the standard radiograph in detecting this change. Microfocal radiographical studies have shown that this change is not the reserve of chronic OA.

Intra- or periarticular mineralisation is a fairly frequent finding in OA. The deposits may be in cartilage, synovium, tendons, ligaments or menisci. Studies in man have revealed that there may be several mechanisms at work in the formation of these deposits and their importance is unknown.

Erosive changes in subchondral bone may occasionally be seen in canine OA. A more common example of this type of OA in veterinary medicine would be the erosive type of bone spavin seen in the horse. Erosive lesions vary in size and shape and may be difficult to distinguish from subchondral cysts. The latter are another occasional feature of OA but are more often seen in man and horses than dogs. They have a sharp border and may appear to communicate with the joint space.

Arthrocentesis

Synovial fluid from osteoarthritic joints tends to be found in normal or increased volumes, with normal or low viscosity (resulting from effusion rather than a qualitative change in the hyaluronic acid content), and a total cell count of up to 5×10^9/l with less than 5% polymorphs (though both of these parameters may be elevated in joints showing an acute flare-up). These features can prove useful in helping to differentiate from other types of joint pathology (see Table 6.1). It may be that in the future synovial fluid becomes much more important in terms of disease diagnosis, estimating prognosis and possibly monitoring treatment.

Further diagnostic techniques

Arthroscopy

Arthroscopy is becoming more commonplace in small animal orthopaedics. The real benefits in canine cases have yet to be proven, particularly with respect to OA. Human rheumatologists are already starting to use arthroscopy in the diagnosis and staging of OA but whether it will become commonplace in this setting remains to be seen.

Scintigraphy

Scintigraphy involves the use of a radioisotope (usually ^{99m}technetium methylene diphosphonate) to produce an image of bone activity using a gamma camera. It has long been recognised that OA joints retain isotope but it has recently been shown in human studies that scintigraphy predicts subsequent radiographic OA. This is, therefore, the first technique identified that may give an indication regarding the outcome of OA.

Ultrasound

The use of ultrasound in OA has been limited because of the problems of definition. Newer, high-frequency, high-resolution probes have shown some usefulness in assessing articular cartilage.

Magnetic resonance imaging (MRI)

Magnetic resonance imaging (MRI) is certainly unavailable to the vast majority of veterinary clinics at the moment. In human medicine, work is concentrating on using MRI to detect

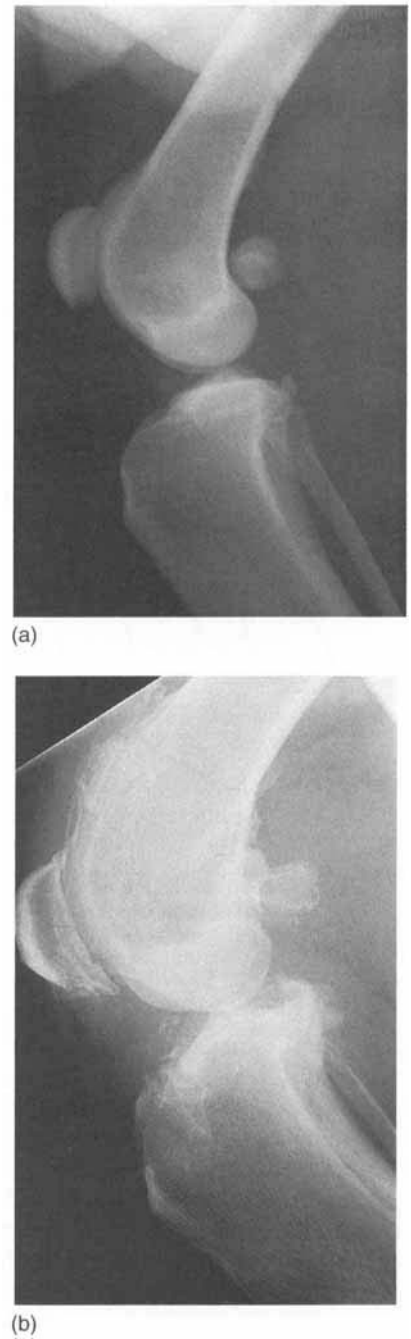
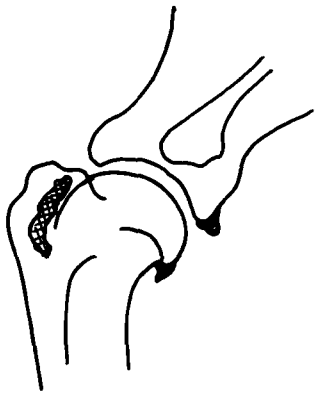
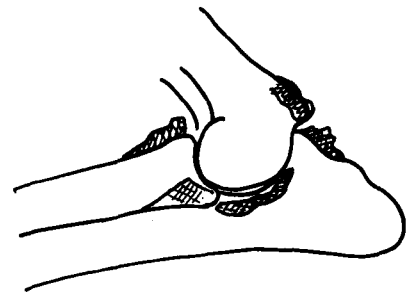


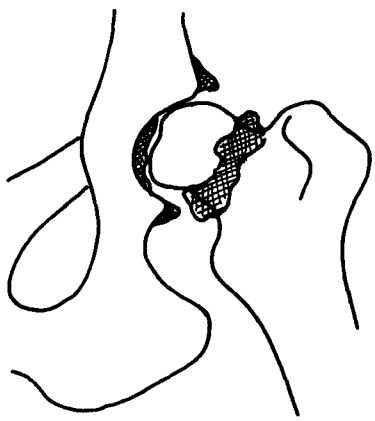
Fig. 7.5 Radiographs illustrating the typical changes seen in stifle osteoarthritis (OA) secondary to cranial cruciate ligament rupture/disease.
(a) Mediolateral radiograph of a normal stifle for comparison.
(b) Mediolateral radiograph showing osteophytosis alongside the trochlear ridges, proximal to the trochlear groove and on the tibial plateau. Enthesiophytes are present on the poles of the patella. The presence of a joint effusion is suggested by partial loss of the infrapatellar fat pad shadow and caudal distension of the joint capsule. There is some evidence of subchondral sclerosis within the distal femur but such radiodensity could also be explained by superimposition of osteophytes upon the normal bone density rather than an increase in the bone density itself.



Shoulder:
Caudal humeral head
Caudal rim of glenoid
Intertubercular groove



Elbow:
Caudal aspect of anconeal process
Coronoid process (seen better on mediolateral view)
Alongside semilunar notch
Medial humeral epicondyle



Hip:
Around acetabular rim
Within acetabular fossa
Around femoral neck

Stifle:
Proximal to trochlear groove
Alongside trochlear ridges
Poles of patella (enthesiophytes)
Tibial plateau
Around fibular head

Fig. 7.6 Diagrams illustrating the predilecion sites for osteophyte formation (hatched areas) in the proximal joints of the fore- and hindlimb.

very early changes in the cartilage. Whilst MRI promises to become the gold standard in imaging joints, there are problems with resolution and also defining the calcified zone of articular cartilage.

Biochemical markers of joint disease

Much research is concentrating on biochemical methods to detect changes in joint tissues. The hope is that these tests will allow changes to be

detected early and allow targeting of patients for disease-modifying therapy. They might also allow the effectiveness of such treatments to be monitored.

Treatment

Conservative management

Exercise

The activity of the dog will have an effect on the course of the disease. When acute flare-ups occur then complete rest for a period of 10–14 days seems to allow the majority of these to settle. The long-term activity of a dog with problematic OA will have to be altered. Short lead walks seem to be preferable with adequate periods of rest allowed should signs worsen.

Diet

Obesity is to be avoided. In human OA it has been shown that obesity is a major contribution to progression of knee OA but this is not the case for hip OA. The variability of risk factors for canine OA at different sites is not known. However, anecdotally most obese dogs suffering with clinical OA seem to improve if weight is reduced.

Medical treatment

There are a variety of approaches for medical treatment of OA but the agents fall into three broad categories:

- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Corticosteroids
- Slow-acting drugs in OA (SADOA)

Non-steroidal anti-inflammatory drugs (NSAIDs)

These are the mainstay of medical treatment of OA in small animal medicine. Whilst the rationale for their use is to relieve signs there has been much work on the effect these drugs may have on joint tissues. One reason for using NSAIDs was the premise that synovial inflammation would be decreased. However, the symptomatic relief afforded to human OA patients with a variety of prostaglandin-blocking NSAIDs has been shown to be no better than the use of a simple analgesic such as acetaminophen (paracetamol). There are several NSAIDs licensed for use in the dog and these are mostly prostaglandin-blocking. Carprofen (Rimadyl, Pfizer) is a prostaglandin-sparing NSAID with an obscure mode of action, but it may have some parallels with acetaminophen in that it too seems to be effective in the relief of OA pain.

There has been much debate on the effects NSAIDs have on cartilage. There is some evidence that some of this group have a deleterious effect on cartilage when compared to others in the group; equally some are heralded as chondroprotective. Much of this work has been carried out *in vitro* and extrapolation to the *in vivo* situation is difficult. The decrease in synovitis may be negated by the analgesic-induced overloading of a joint. Add to this the effect a drug may have on chondrocyte metabolism and clearly the situation is complex. However, the effects in the long term are generally felt to be minimal.

Several factors may influence the choice of drug:

- Clinical response
- Side-effects (important if there is concurrent hepatic, gastrointestinal or renal disease)
- Cost
- Type of preparation (capsules, tablets, drops)
- Convenience of dosing regime

Table 7.1 provides a list of commonly used products available in the UK for the treatment of OA. The products will vary in their effectiveness between individuals in keeping with the heterogenetic nature of OA.

It would seem unnecessary to consider the use of unlicensed products in the treatment of canine OA now that there is an adequate range of licensed drugs. NSAIDs available for use in humans without prescription, such as ibuprofen, should be avoided since their half-lives are much longer in the dog, compared with humans, and so it is easy to overdose. Flurbiprofen should

Table 7.1 Non-steroidal anti-inflammatory drugs (NSAIDs) used in the treatment of canine and feline osteoarthritis (OA).

NSAID	Patient	Dose
Aspirin	dog	25 mg/kg/day in divided doses (not licensed)
	cat	10–20 mg/kg every 2–3 days (not licensed)
Phenylbutazone (Phenycare)	dog	2–20 mg/kg/day in divided doses
	cat	1–5 mg/kg/day
Carprofen (Rimadyl)	dog	2–4 mg/kg/day in two doses for up to 7 days, 2 mg/kg/day as single dose thereafter
Ketoprofen (Ketofen)	dog	1 mg/kg/day for up to 5 days
	cat	1 mg/kg/day for up to 5 days
Tolfenamic acid (Tolfedine)	dog	4 mg/kg/day for up to 3 days
	cat	4 mg/kg single injection (upper respiratory tract disease only)
Meloxicam (Metacam)	dog	0.2 mg/kg/day as single dose on day 1 and 0.1 mg/kg/day or less thereafter
Flunixin (Finadyne)	dog	1 mg/kg/day for up to 3 days contraindicated in pregnant bitches
Mefenamic acid (Ponstan)	dog	10–30 mg/kg/day in divided doses (not licensed)
Piroxicam (Feldene)	dog	0.3 mg/kg every 48 hours (not licensed)
Prednisolone, **Cinchophen**, **Hexamine** (Predno-Leucotropin)	dog	1–2 tablets/10 kg/day in divided doses
	cat	0.5 tablet twice daily

Further information for those that are licensed can be found in the corresponding data sheets published annually as *Compendium of Data Sheets for Veterinary Products* by National Office of Animal Health Ltd.

definitely not be used since it may lead to an idiosyncratic reaction and death.

All NSAIDs may cause gastrointestinal irritation resulting in vomiting or diarrhoea (possibly with blood). Recently the enzyme cyclo-oxygenase (COX) has been shown to have two isomers. COX1 is responsible for 'house keeping' functions and COX2 is induced in inflammatory conditions. It has been proposed that NSAIDs acting selectively on COX2 will have a lower incidence of side-effects and this has been supported by research study results. Of the drugs available, meloxicam (metacam, Boehringer Ingelheim Ltd.) and carprofen (Rimadyl, Pfizer) show most promise.

If severe side-effects occur the drug should be withdrawn. To try and avoid such side-effects these drugs are best given with some food. Flunixin may cause severe haemorrhagic gastroenteritis and the 3-day limit should not be exceeded. If such side-effects result in withdrawal of a drug then, once the signs have settled down, another one may be tried. Particular dogs are often sensitive to certain agents.

'Trial and error' will determine a suitable drug for an individual dog.

Corticosteroids

The use of corticosteroids in the treatment of OA is another controversial area. Some work in experimental OA suggested that corticosteroids hasten the degeneration of cartilage and this evidence seemed to make the use of corticosteroids (particularly intra-articular use) contraindicated. However, there is recent evidence that intra-articular (I/A) methylprednisolone (Depo-Medrone V, Pharmacia & Upjohn), at a dose of 20 mg for an average Labrador's shoulder or stifle, may have beneficial effects in canine OA. Certainly there may be dramatic relief from clinical signs although the response is variable. Human rheumatologists may repeat intra-articular injections up to three times a year. The response to I/A corticosteroid seems to vary depending on the joint. It is probably important to rest the patient strictly for at least 2 days following the injection to encourage the drug to stay within the joint and

limit the systemic effect. If corticosteroids are to be administered systemically, low-dose prednisolone (0.25–0.5 mg/kg once daily or every other day) is the preferred drug. A course of 2–3 weeks should be used initially.

Corticosteroids seem to be particularly helpful in certain situations, namely:

- When other medical treatments have failed and the dog's quality of life has declined
- In elderly dogs where there is concern over NSAID side-effects
- After an acute flare-up of OA, which may be associated with a high level of polymorphs (15–30%) in synovial fluid
- In erosive OA

Slow-acting drugs in OA (SADOA)

The International League Against Rheumatism (ILAR) has proposed two categories in this group: disease-modifying OA drugs (DMOADS) and symptomatic slow-acting drugs in OA (SYSADOA). This classification aims to clearly define the concept of 'chondroprotection' and distinguish drugs that have this property from those that merely give symptomatic relief. The concept of chondroprotection evolved from clinical trials of certain agents which appeared to retard the progression of OA. However, the pharmacology of these drugs could not be related to the pathology of OA and hence there is a need for a much tighter definition of chondroprotective drugs.

There is some evidence that certain agents can alter the environment or activity of the chondrocyte *in vitro* such that the degeneration of cartilage is stopped or decreased. Unfortunately, although *in vitro* results may suggest that certain agents have these capabilities, *in vivo* work is hampered by the fact that there are, at present, no readily quantifiable criteria for measuring outcome of OA. This is even more applicable in clinical trials, many of which have been uncontrolled and poorly designed. Thus, there is much conflicting evidence in the literature.

At present there is only one drug in this class licensed for use in the dog. This is sodium pentosan polysulphate (PPS) (Cartrophen-Vet, Arthropharm, Australia) which is a polysaccharide sulphate ester prepared from beech hemicellulose. This drug has been shown to inhibit certain proteinases but it is structurally similar to heparin and so has anticoagulant properties. The mode of action in clinical cases is obscure and recent studies suggest that the fibrinolytic action may shift microthrombi in the vascular supply of subchondral bone resulting in pain relief. Validated clinical trials with the drug are lacking but anecdotal evidence suggests that it can give impressive clinical results in certain cases. However, suitable case selection is a large stumbling block. An oral preparation of PPS has been used experimentally in dogs and is licensed in some countries but not yet in the UK.

More recently the terms 'structure modifying' and 'symptom modifying' drugs have been used to simplify the terminology whereby PPS would be viewed as an example of the former and a NSAID of the latter, although some NSAIDs are also claimed to have structure-modifying properties.

Dietary supplementation

Recently there have been a number of dietary supplements put onto the market which claim to be able to modify the course of osteoarthritis. These generally contain chondroitin sulphate (e.g. Cartivet, Arnolds) or this together with glucosamine (e.g. Cosequin, Xeipon). Their possible methods of action have been suggested to include an anti-inflammatory action and also an indirect influence on the viscosity of synovial fluid by inhibiting the action of hyaluronidase and/or increasing the rate of hyaluronic acid production. Thus far, there is no scientific evidence to support their use although there are certainly anecdotal reports to suggest that they may help in particular cases. Because of this the authors do not generally recommend their use but would not oppose an owner's wishes to try out such a preparation since they are, at the very least, unlikely to be harmful to the osteoarthritic joint or patient.

Future possibilities for disease modification

There are many avenues of research in the

treatment of OA. Certain forms of treatment have been around for some time but have yet to be proven useful. A good example of this would be polysulphated glycosaminoglycan preparations used for so-called *viscosupplementation*. These have been licensed, in various forms, for treatment of human and equine arthritis for some time (e.g. Adequan, Janssen Animal Health). Due to the difficulties in quantifying outcome in OA, their effectiveness in clinical trials is still variable. A recent study showed no difference between patients receiving intra-articular hyaluronic acid and those receiving intra-articular saline. The explanation for this might lie in reports on the usefulness of articular lavage in the symptomatic relief of OA in humans. Clearly any preparation delivered after arthrocentesis would have to take this possible effect into account.

The theories of viscosupplementation revolve around improving the properties of the synovial fluid. This may have many effects such as decreasing abnormal load on cartilage or altering inflammatory mechanisms. There may be more efficacy in newer formulations of hyaluronic acid which have a high degree of cross linking which creates a very high molecular weight and improves the viscoelasticity.

Other medical developments are centred on attempting to *alter the activity of chondrocytes* by blocking the action of certain cytokines, for example, IL-1-blocking agents are reaching the clinical trial stage. Cytokines which stimulate synthetic activity of chondrocytes have also received attention, for example IGF-1 has been used experimentally (in combination with PPS) and shown to have some promise in maintaining matrix integrity.

Gene therapy is a concept receiving attention throughout medicine. Recent experimental work has shown that the gene for IL-1ra can be implanted into synovial cells in joints and these cells will express this gene for several weeks. Clearly there is exciting potential in such an approach to arthritis.

Surgical management

There are several potential aims of surgery in managing an osteoarthritic joint:

- To attend to the initiating cause
- To alleviate certain sources of pain caused by the osteoarthritic process
- To remove or replace the osteoarthritic joint

Other than when surgery is aimed at eliminating the initiating cause of the OA, it is generally reserved for those cases that show an unsatisfactory response to the conservative and medical measures detailed above. The options available vary with different joints and the surgical management of OA for each joint is discussed in detail within the relevant chapters of Sections 5 and 6. A general overview of what may be achieved will, however, be considered here.

Cheilectomy

Cheilectomy involves the debridement of the joint with the removal of osteophytes or 'loose bodies', possibly in combination with synovectomy. Although there is no hard evidence on the beneficial effects of such a procedure, many cases will improve for a variable period of time afterwards. The joint in which this is, perhaps, most appropriate for assisting management of OA would be the stifle.

Metaphyseal forage

Metaphyseal forage involves drilling across the metaphyseal scar(s) adjacent to the joint in question. The procedure has evolved from the pain relief reported after the performance of metaphyseal osteotomies in human patients. One belief is that venous stasis in the subchondral bone creates ischaemia and is a source of pain. Metaphyseal forage may improve the blood flow through this region, thereby reducing any such ischaemia. The exact mechanism by which this procedure may have a beneficial effect is yet to be elucidated but there are reports of such an effect being maintained for several months. It may be carried out alone or in combination with cheilectomy. Surgeons who use this technique might apply it, in decreasing likelihood, to the stifle, elbow or hip joint.

Joint replacement

Joint replacement is an ideal way to treat an end-stage osteoarthritic joint. At present a prosthesis is only available for replacement of the coxofemoral joint in the dog. The development of components for other joints is currently underway and the elbow is most likely to be the next in line.

Excision arthroplasty

Excision arthroplasty may be used as a salvage procedure in certain joints such as the hip. The success of such techniques depends on several factors such as the joint involved and the size of the patient.

Arthrodesis

Arthrodesis may be employed in appendicular joints other than the hip when end-stage OA cannot be controlled by other means. The success of such a procedure varies between the joints with the carpus being an example of a joint that can be arthrodesed with very good results, whereas fusion of an elbow or stifle will present far more mechanical problems for the dog.

Cartilage grafts

Cartilage grafts might offer a potential treatment for patients with advanced OA. Much work is being directed at trying to 'resurface' osteoarthritic joints with hyaline cartilage from either a donor or from tissue culture. Although this technique is not yet available, and there remain several problems regarding its application, it might provide an alternative method of management in the future. The topic of cartilage grafting is also discussed in Chapter 2 (p. 21).

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A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny,Steven J. Butterworth

Chapter 8
Immune-mediated Polyarthritides

Classification

The main feature used to classify an immune-mediated polyarthritis is whether or not there are associated erosive changes in the joints. Each of these broad groups (erosive and non-erosive) contain several well-recognised conditions (Table 8.1) which are characterised by specific features. However, it is important to bear in mind that, to an extent, these divisions are somewhat arbitrary and there may be considerable overlap between them.

Investigation

A general approach to the investigation of joint disease is detailed in Chapter 6. Features that are of particular note with respect to the immune-mediated joint diseases will now be outlined.

History and clinical signs

Although there are certain breed-related syndromes within this group of diseases, immune-mediated polyarthritides can affect any breed, age or sex of dog or cat. The signs noted by an owner may vary from generalised stiffness (which may be so severe as to prohibit ambulation) or malaise (usually associated with pyrexia), through a shifting lameness (which may be quite subtle) to deformity of joints (which, in small breeds, may become apparent after a visit to the grooming parlour!), particularly in long-standing, erosive arthritides.

On examination there will usually be bilaterally symmetrical swelling of multiple joints, though in some cases only one pair of joints may appear to be involved. The feature of symmetry is helpful in distinguishing these conditions from that of haematogenous septic arthritis where multiple joints may be involved but is usually asymmetric. Affected joints are usually swollen and painful but crepitation is a inconsistent feature. Instability may result from ligament failure secondary to the inflammatory pathology and it may be that the patient showed no obvious clinical lameness until the instability developed (e.g. a dog with long-standing, bilateral, immune-mediated stifle joint pathology might suddenly become lame and be presented with what appears, at face value, to be a degenerative failure of the cranial cruciate ligament). In addition, 20% of dogs presented with pyrexia of unknown origin were found to have an immune-mediated polyarthritis (Dunn & Dunn, 1998).

Radiology

Plain radiographs of all clinically affected joints, with or without others that are commonly affected (i.e. carpi, hocks, stifles, elbows) should be taken. The main features to be observed include:

- Evidence of a joint effusion (Fig. 8.1a)
- Erosive changes (Fig. 8.1b)
- Disruption of normal joint anatomy causing deformity (Fig. 8.1c)
- Proliferative changes (secondary osteophyte formation or periosteal bone formation)

Plain radiographs of the thorax and abdomen should be included in the investigation, especially in cases with non-erosive polyarthritis, since if a diagnosis of canine idiopathic polyarthritis is made then pathology with which this might be

Table 8.1 Classification of the immune-mediated arthritides seen in dogs and cats.

Erosive polyarthritides

- Rheumatoid arthritis
- Periosteal proliferative polyarthritis (mainly cats)
- Greyhound polyarthritis
- Felty's syndrome (rare)

Non-erosive polyarthritides

- Systemic lupus erythematosus
- Polyarthritis/polymyositis syndrome (especially Spaniels)
- Polyarthritis/meningitis syndrome
- Polyarteritis nodosa
- Idiopathic polyarthritides
  - Type I uncomplicated
  - Type II associated with infection (including Lyme disease in dogs and calciviral arthritis in cats)
  - Type III associated with gastrointestinal disease
  - Type IV associated with neoplasia
- Breed-associated syndromes
  - Japanese Akita arthritis
  - Chinese Shar Pei fever syndrome
- Idiosyncratic drug reactions (particularly antibiotics, e.g. Dobermann Pinschers given sulphonamides)

associated (e.g. an abscess or neoplasm) needs to be screened for.

Clinical pathology

Haematology

Anaemia may be seen in some cases, but may be marked in those with systemic lupus erythematosus (SLE). Such cases may also show a thrombocytopenia. The white cell count is often normal but may be raised due to a neutrophilia, particularly in cases with arthritides associated with infection elsewhere in the body.

Serology

There may be raised enzyme levels (e.g. serum alkaline phosphatase, alanine transferase and aspartate transferase) as a non-specific result of inflammation. Finding elevated levels of creatinine phosphokinase may be helpful in cases suspected of having polymyositis. Protein levels may be altered with globulin levels tending to increase in patients with inflammatory pathology and albumin levels decreasing if a protein-losing nephropathy is present. Tests relating more specifically to the polyarthritides include assays for rheumatoid factor (RhF), antinuclear antibody (ANA) and antibodies to *Borrelia burgdorferi* (Lyme disease). Although these tests are helpful in trying to reach a specific diagnosis, it must be remembered that, unfortunately, they are not definitive either as positive or negative findings. RhF may be produced in chronic polyarthritides other than rheumatoid arthritis, some cases with SLE are negative for ANA (so-called seronegative SLE!), and the presence of antibodies to a specific infective agent proves only that the patient has been exposed to that agent in the past.

Urinalysis

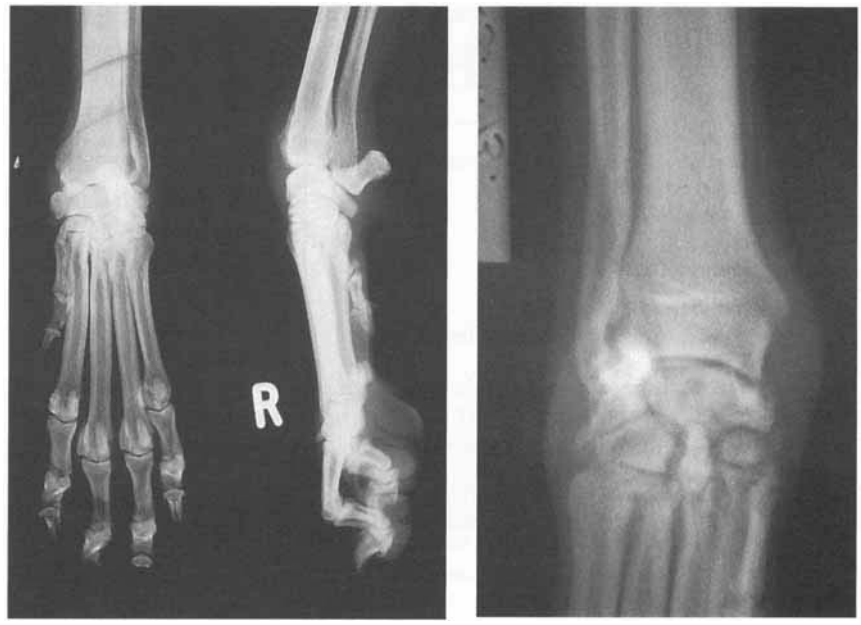
The main feature to be looked for in urinalysis is the presence of protein, though if evidence of infection is found then it might relate to a type II idiopathic polyarthritis. Proteinuria may result from glomerulonephritis (e.g. as a part of SLE) or renal amyloidosis (e.g. as a part of Chinese Shar Pei fever syndrome).

Synovial fluid analysis

Arthrocentesis should be attempted for all affected joints. Analysis of the acquired fluid will normally show a lowered viscosity, white cell counts in the region of $5–90 \times 10^9$/l with a significant proportion of polymorphs (20–80%). These features help to rule out other causes of joint disease (see Table 6.1).

Synovial membrane pathology

If a diagnosis cannot be made from the tests above then several synovial membrane biopsies should be taken from at least one joint and submitted for histopathology. The microscopic features of synovial membrane in these diseases can be variable and are not consistent throughout all parts of an affected joint (hence the reason for taking several biopsies). In general, a moderate to extensive



(a) (b)

(c)

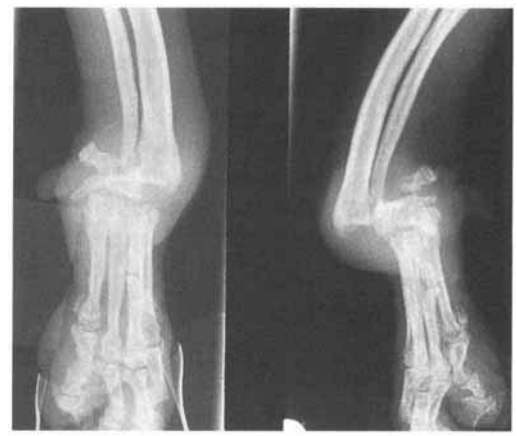


Fig. 8.1 (a) Mediolateral and dorsopalmar radiographs of the right carpus from a 7-year-old cross-breed showing clinical signs of polyarthritis. There is evidence of soft tissue swelling suggestive of a joint effusion but the arthropathy appears non-erosive.
(b) Dorsopalmar radiograph of the left carpus from an 11-year-old Golden Retriever showing clinical signs of polyarthritis. There is evidence of soft tissue swelling suggestive of a joint effusion but also erosive changes which are most easily noted within the radial carpal bone.
(c) Mediolateral and dorsopalmar radiographs of the left carpus from an 8-year-old Yorkshire Terrier showing signs of bilateral carpal and tarsal collapse. There is gross deformity of normal joint anatomy with irregular joint spaces and antebrachiocarpal luxation.

white cell infiltrate is expected with a predominance of polymorphonuclear cells in rheumatoid arthritis (though this would also be true in septic arthritis).

Bacteriology

Bacterial culture is not generally required but if haematogenous, septic arthritis cannot be ruled out from other features then synovial fluid, blood or, perhaps best of all, synovial membrane may be cultured in order to try and isolate the organism responsible.

Erosive polyarthritides

Rheumatoid arthritis (RhA) (Bennett, 1987a,b)

The aetiology of this disease is not known but it is suspected that the presence of raised levels of rheumatoid factor (RhF) in the majority of cases is of significance. RhF is an autoantibody to IgG and the interaction between these two will produce circulating immune complexes. If these were to localise to the synovial membrane and fix complement then a type III hypersensitivity

(i.e. that involving immune complex deposition) would result, creating an inflammatory joint disease.

In comparison to its incidence in man, rheumatoid arthritis (RhA) is uncommon in the dog and rare in the cat. When it does affect these patients it causes a severe, often progressive and sometimes deforming polyarthritis. The signs are as described in the general comments above but the specific features include the presence of subchondral erosions seen radiographically (Figs 8.1b and 8.1c), raised RhF levels (seen in about 75% of cases) and normal levels of ANA (or certainly not as elevated as in cases with SLE), and a predominance of polymorphonuclear cells in the raised white cell count found on synovial fluid analysis. The American Rheumatism Association base the diagnosis of RhA on eleven criteria:

(1) Stiffness after rest
(2) Pain or tenderness in at least one joint
(3) Swelling in at least one joint
(4) Swelling of a second joint within 3 months
(5) Symmetrical joint swelling
(6) Subcutaneous nodules (most often over bony prominences)
(7) Destructive/erosive radiographic changes suggestive of RhA
(8) Raised serum levels of RhF
(9) Abnormal synovial fluid (in particular a poor mucin precipitate)
(10) Characteristic histopathological changes in the synovial membrane, including three or more of the following: marked villous hypertrophy; proliferation of superficial synovial cells; marked infiltration of chronic inflammatory cells (lymphocytes/plasma cells); the presence of aggregates of lymphoid cells; foci of cell necrosis; deposition of compact fibrin on the surface or interstitially
(11) Characteristic histopathological findings within the subcutaneous nodules (granulomatous foci with central necrosis surrounded by proliferating cells bounded by peripheral fibrosis and chronic inflammatory cell infiltration)

Seven of these eleven have to be fulfilled before a diagnosis of 'classical' RhA can be made whilst five provide a diagnosis of 'definite' RhA, particularly if two of criteria 7, 8 and 10 are satisfied. Subcutaneous nodules are rarely seen in small animal patients with RhA and so criteria 6 and 11 can be discounted in practical terms.

Treatment involves the use of immunosuppressive drugs as outlined below. Alternatively, aspirin may be used at a dose of 25–35 mg/kg per os every 8 hours. This helps to relieve the clinical signs and is believed to retard the disappearance of cartilage. A final option which may help control the signs in cases with RhA is that of gold therapy. This may be injected as sodium aurothiomalate (Myocrisin, May & Baker; six injections of 0.75–1 mg/kg gold salt at weekly intervals) or given by mouth as auranofin (Ridaura, Smith-Kline Beecham; 0.05–2 mg/kg twice daily for 6 weeks – maximum of 9 mg/day/dog). Such gold therapy is generally combined with prednisolone treatment starting with immunosuppressive doses and then gradually reducing this over the 6 weeks. Possible side-effects of gold injections include bone marrow suppression, corneal ulceration, dermatoses and renal failure. Oral treatment is believed to cause less systemic side-effects but can result in severe diarrhoea. The prognosis is very guarded as the joint destruction is progressive and even if the inflammatory process is controlled or 'burns out' with time the associated joint deformity may still cause marked disability, though this is not always the case.

Periosteal proliferative polyarthritis of cats (Bennett, 1994)

The cause of this disease is uncertain. There may be a link with feline leukaemia virus or feline syncytia-forming virus but neither of these hypotheses have been proven. This bilaterally symmetrical polyarthritis most commonly affects the carpi and hocks. Radiographs will usually demonstrate the presence of erosive change and also marked, proliferative, periosteal new bone formation around the affected joints. There may also be erosions at points of attachment of ligaments or tendons as a feature of associated enthesitis. Other features of the disease are as described in the general comments above. Although it may appear similar to RhA clinically, it may be differentiated on the basis that it generally affects fewer joints, serology reveals no increase in RhF levels,

the periarticular bone proliferation is more pronounced, and enthesitis is rarely seen as a component of RhA.

Greyhound polyarthritis (Huxtable & Davis, 1976; Barton *et al.*, 1985)

A number of Greyhounds less than 3 years of age have been reported as developing erosive polyarthritides. Although the aetiology is uncertain, the isolation of *Mycoplasma spumans* from one case might be of significance in this respect.

Felty's syndrome (Bennett, 1990)

This condition is well recognised in man and comprises a combination of RhA in association with splenomegaly and a neutropenia. It is very rare in the dog.

Non-erosive polyarthritides

Systemic lupus erythematosus (SLE) (Bennett, 1987c)

The cause of this uncommon disease of dogs remains unclear but two main components of its pathogenesis have been recognised as autoimmunity and immune complex hypersensitivity. Antibodies are produced against erythrocytes, platelets and leucocytes. The immune complexes which form may then become deposited in synovium, glomeruli, the dermal/epidermal junction and/or one of several other body systems, where subsequent complement fixation results in a type III hypersensitivity. This accounts for the multisystemic nature of the disease and its wide range of presentations.

The clinical features related to synovitis are as outlined in the general comments above, often with concurrent pyrexia. Other clinical signs may be associated with multisystemic involvement including: haemolytic anaemia; thrombocytopenia; leukopenia; protein-losing nephropathy; pleuritis; sterile meningitis; skin or mucocutaneous lesions. Any age or breed of dog can be affected and the disease has been recognised in the cat.

The diagnosis of this disease needs to be considered carefully as its multisystemic nature may lead to over-diagnosis. The investigation of a case suspected of having this condition is as given in the general comments above. The general findings in a case presented with lameness should be a symmetrical polyarthritis that is non-erosive radiographically (Fig. 8.1a) and shows typical synovial fluid abnormalities associated with an immune-mediated synovitis. Synovial membrane biopsy will show thickening due to hypertrophy/hyperplasia of mainly type B cells, without villous hypertrophy. Cellular infiltration usually involves macrophages, lymphocytes and plasma cells. Obviously, other body systems need to be examined and investigated appropriately. Routine haematology and serum biochemistry is crucial, including assays to measure titres of ANA and RhF. Another 'specific' test for the disease is observation of the LE-cell phenomenon. If a blood sample is incubated then ANAs may react with leucocytes causing the release of nuclear material which is then phagocytosed by other white cells to form LE-cells which may then be identified on examination of a blood smear. Criteria which should be fulfilled before making a diagnosis of SLE include:

(1) Involvement of more than one body system, demonstrable clinically or on laboratory analysis.
(2) Significantly raised serum levels of ANA. This antibody can be produced as a result of a wide range of chronic pathologies and the significance attributed to a specific titre varies between laboratories. In general, anything >1/16 is considered significant and most SLE patients will have titres >1/64.
(3) Immunopathological features consistent with the clinical syndromes present should be proven (e.g. the presence of antibodies to erythrocytes in cases showing haemolytic anaemia, or immune complex deposition in synovial membrane/glomeruli in cases with synovitis/protein-losing nephropathy).

If criteria 1 and 2 are fulfilled then a diagnosis of 'probable' SLE can be made, but if 3 can also be satisfied then 'definite' SLE can be diagnosed.

Treatment involves the use of immunosuppres-

sive chemotherapy (as outlined below) but attention may be required to manage other aspects of the disease whilst such treatment begins to take effect (e.g. blood transfusions may be necessary in cases with haemolytic anaemia). The prognosis is guarded with the disease being controlled by chemotherapy in about 50% of cases. In some of these it may be possible to withdraw treatment after several weeks whilst in others the continued administration of immunosuppresive drugs is required to maintain control of the disease.

Polyarthritis/polymyositis syndrome (Bennett & Kelly, 1987)

The combination of polyarthritis and polymyositis may be seen in some dogs, particularly Spaniels. A non-erosive, inflammatory polyarthopathy will be found on carrying out the investigation detailed previously, but the patient will be seronegative for SLE. The associated polmyopathy will be recognised as pain on palpation of particular muscle groups, atrophy and sometimes contracture. These changes will usually be bilaterally symmetrical. Raised serum creatinine phosphokinase levels support the diagnosis and muscle biopsy can be used to confirm it. Other possible diagnoses that might mimic these signs need to be ruled out, in particular SLE and toxoplasmosis.

The prognosis is considered guarded with only two of the six dogs treated in one series (Bennett & Kelly, 1987) recovering after treatment with immunosuppresive doses of prednisolone and cyclophosphamide (see later, p. 71).

Polyarthritis/meningitis syndrome (Pedersen *et al.*, 1989)

The combination of immune-mediated polyarthritis and meningitis has been reported in a number of breeds, including Beagles, Bernese Mountain Dogs, Boxers, German Short Haired Pointers and Weimaraners. The polyarthritis causes signs as described above. The associated meningitis usually presents as spinal pain and/or depression. Its presence may be confirmed by analysis of cerebrospinal fluid which will demonstrate a raised white cell count, protein level and creatinine phosphokinase level.

The prognosis is considered fair with some patients achieving long periods of remission following immunosuppresive treatment.

Polyarteritis nodosa (Pedersen *et al.*, 1989; Bennett, 1990)

This is an extremely rare disease in dogs and its specific feature is the presence of a fibrinous infiltrate around arterioles within the synovial membrane.

Idiopathic polyarthritides (Bennett, 1987d, 1994)

This group of arthritides comprise those which are non-infective and non-erosive but cannot be classified into any of the other recognised catagories. They are the most common form of immune-mediated arthritis seen in small animal orthopaedics with a predisposition to certain breeds such as German Shepherd Dogs, Irish Setters, Shetland Sheepdogs and Spaniels. Though dogs of any age can be affected they are most common in those aged 1–3 years and uncommon in those less than 1 year of age. There may be a higher incidence in dogs compared to bitches.

The history and clinical features are as discussed in the general comments above, namely lameness associated with bilaterally symmetrical joint swelling, a radiographically non-erosive polyarthropathy, typical changes on synovial fluid analysis and negative serum titres to ANA and RhF. Muscle atrophy is a common finding and some show pain on palpation/manipulation of the vertebral column (possibly indicating involvement of the intervertebral facet joints). About two-thirds of these cases will show systemic signs of illness, such as pyrexia, lethargy, inappetance, as well as lameness. In addition, ulceration of the buccal or lingual mucosa is often seen (possibly as a result of a toxic epidermal necrosis).

The pathogenesis of these conditions may involve the deposition of immune complexes in the synovium that have formed as a result of pathology elsewhere in the body. The fixation of complement by the complexes may then lead on to a type III hypersensitivity and a resulting inflammatory synovitis. Any such circulating complexes could also be deposited elsewhere in the body resulting in other conditions, such as

glomerulonephritis, dermatitis or uveitis/retinitis. The idiopathic polyarthritides have been subdivided into the following four types according to the nature of the pathology with which they are associated.

Type I (uncomplicated idiopathic polyarthritis)

This type accounts for about 50% of the cases classed as idiopathic. No associated pathology has been identified and so this may be the truly 'idiopathic' form. However, some dogs initially classified as this may go on to develop erosive changes and become reclassified as RhA. What factors control whether or not this progression occurs remain unknown.

Treatment involves the use of immunosuppressive drugs (detailed below under 'Treatment') which may be withdrawn after 6–8 weeks. Some cases will show complete remission whilst in others continued treatment with such drugs will be found to control the disease. The prognosis is, therefore, fair to good.

Type II (reactive idiopathic polyarthritis)

This type accounts for about 25% of the cases classed as idiopathic. The condition is associated with an infective process remote from the joint, e.g. respiratory tract infection, urinary tract infection, pyoderma, pyometra, which are hypothesised to produce circulating immune complexes. The prognosis is generally good as the associated lameness will often resolve after the source of immune complexes, i.e. the infective process, has been eliminated either by treatment with antibiotics or surgery. In some cases, however, the synovitis persists and may require immunosuppressive drug therapy for a few weeks (as detailed below under 'Treatment'). It is possible that the arthropathy resulting from certain infections such as feline calcivirus or borreliosis (Lyme disease) may be, in part, due to this type of reaction, and again immunosuppressive treatment may be required if the lameness persists after treatment/resolution of the infection.

Type III (enteropathic idiopathic polyarthritis)

This type accounts for about 15% of the cases classed as idiopathic. The condition is associated with some form of gastrointestinal disease, usually involving chronic vomiting or diarrhoea. The source of the circulating immune complexes may be antibodies forming to antigens that are usually unable to cross the alimentary tract wall but do so in the face of disease that increases its permeability. The prognosis is generally good as the associated lameness will often resolve if the gastrointestinal disease can be brought under control. In some cases, however, the synovitis persists and may require immunosuppressive drug therapy for a few weeks (as detailed below under 'Treatment').

Type IV (neoplasia-related idiopathic polyarthritis)

This type accounts for about 10% of the cases classed as idiopathic. The condition is associated with some form of neoplasia remote from the joints affected. The source of the circulating immune complexes may be antibodies forming to foreign antigens found on or in the tumour cells. The associated lameness will often resolve if the neoplasia can be eliminated. In some cases, however, the synovitis persists and may require immunosuppressive drug therapy for a few weeks (as detailed below under 'Treatment'). The prognosis with this type of idiopathic polyarthritis is variable and directly related to how feasible it is to treat the associated neoplasia.

Breed-associated syndromes

Non-erosive, inflammatory polyarthropathies may be seen sporadically in certain specific breeds. For example, in Japanese Akitas and as a feature of Chinese Shar Pei fever (May *et al.*, 1992). The prognosis is variable but has to be stated as guarded, particularly in the case of the Shar Pei where they tend to develop renal amyloidosis (DiBartola *et al.*, 1990).

Idiosyncratic drug reactions

Certain drugs, most notably antibiotics (including penicillins, sulphonamides, cephalosporins, erythromycin and lincomycin) have been reported as causing idiosyncratic reactions resulting in an inflammatory polyarthropathy. The most well-reported incidence of such reactions is probably

that seen after prescribing sulphonamides to Dobermann Pinschers. In most cases the clinical signs resolve after the causative agent is withdrawn.

Treatment

Drug therapy

Immunosuppressive drug therapy is the mainstay in treatment of the immune-mediated polyarthritides, with the possible exception of those associated with other pathologies (i.e. idiopathic types II, III and IV). The principles of using prednisolone alone or in combination with cytotoxic drugs will be considered here. Mention of other medical treatments that are specific to certain types of arthritis has already been made under the relevant subheading.

Prednisolone

Prednisolone alone often suffices in the treatment of type I idiopathic polyarthritis and may allow control of signs in others such as SLE or RhA. It is given at an initial dose of 2–4 mg/kg daily per os in divided doses for 1 week and then half that dose until clinical improvement is seen. After that the dose may be gradually reduced over several weeks until it has been completely withdrawn or a maintenance level is found.

Prednisolone and cyclophosphamide or azathioprine

These are the most commonly used combinations involving cytotoxic drugs in the treatment of SLE and polyarthritis/polymyositis syndrome in particular, but also other cases with immune-mediated polyarthritis that have not responded to prednisolone alone. Cyclophosphamide is given at a dose of 50 mg/m^2 daily per os on the first 4 days of each week until 2–3 months after clinical remission has been achieved. The body surface area in square metres is equal to the bodyweight in kilograms raised to the power 0.66 and then divided by 10, i.e.:

$$\text{Body surface area } (m^2) = \frac{\text{Bodyweight } (kg)^{0.66}}{10}$$

Prednisolone is commenced at a dose of 2–4 mg/kg daily per os in divided doses for 1 week and then continued at half that dose for at least 1 month after cyclophosphamide has been withdrawn. Azathioprine is used either as well as or instead of cyclophosphamide in dogs but *not cats* at a dose rate of 2 mg/kg daily per os for 2–3 weeks and then on an every-other-day basis, alternating with the prednisolone.

Whenever cytotoxic drugs are combined with prednisolone it is important to monitor for side-effects such as bone marrow suppression. Blood samples should be taken at weekly intervals in the early stages and then every 2–4 weeks as treatment progresses. If the white cell count falls below 6×10^9/l then the dose should be reduced by 25% and if it falls below 4×10^9/l then the drug should be withdrawn for 1 week and then reinstated at half the original dose. Cyclophosphamide treatment may also be associated with sterile haemorrhagic cystitis but this can usually be avoided if the period for which the drug is given does not exceed 3 months.

Gold therapy

Gold therapy has a role in the management of RhA and was discussed earlier (p. 67).

Non-steroidal anti-inflammatory drugs (NSAIDs)

Non-steroidal anti-inflammatory drugs (NSAIDs) may have a role in the symptomatic treatment of some cases with immune-mediated polyarthritis. This is perhaps most often the case in RhA where aspirin has been used to good effect. This was discussed earlier (p. 67).

Surgery

Surgery is not often indicated in the management of immune-mediated polyarthritis. However, in some instances synovectomy, excision arthroplasty or arthrodesis may be used to treat a particularly painful joint. Indeed, it may seem preferable to consider bilateral arthrodesis in a patient suffering with a deforming arthritis affecting only, say, the carpal joints, rather than with medical treatment that might not improve joint stability. However, achieving arthrodesis in such

cases can be more problematic than when such surgery is indicated for other reasons.

Prognosis

The prognosis for each type of immune-mediated polyarthritis varies and so a definitive diagnosis is paramount if an accurate prognosis is to be given to an owner. The likely outcome for each type has been discussed under the relevant headings above.

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Chapter 9
Infective (Septic) Arthritis

A wide variety of infective agents can cause an inflammatory arthropathy. Bacteria, particularly *Streptococcus* spp. and *Staphylococcus intermedius*, are the most common cause of infective arthritis in the dog (Bennett & Taylor, 1988). In the cat, bite wounds are the most common cause, involving bacteria normally found in the mouth such as *Pasteurella multocida*, *Bacteroides* spp. and *Streptococcus* spp. Other infective agents include mycoplasmata, fungi, rickettsiae, spirochaetes, protozoa and viruses.

Bacterial arthritis

Aetiology

Bacterial arthritis may occur as a result of direct infection from a penetrating wound, as a complication of arthrotomy, by extension from a local purulent focus or it may result from haematogenous spread. Although haematogenous spread of infection to joints is common in young farm animals, the condition is relatively uncommon in the dog. Nevertheless, in a series of 58 cases with confirmed bacterial arthritis reported by Bennett & Taylor (1988), most of the infections appeared to be haematogenous in origin. Infection tends to localise in joints which have already been damaged as a result of trauma or osteoarthritis.

History and clinical signs

Two syndromes are recognised, the classic acute onset case and those with a more chronic low-grade infection. The condition occurs more commonly in large breeds of dog. The carpus is the joint most frequently affected but the shoulder, hip and stifle joints have also been implicated. Affected joints are swollen or thickened and painful. Lameness is often severe. Once infection is established in the joint, there is rapid and extensive destruction of articular cartilage and subchondral bone but few cases show evidence of systemic illness.

Radiology

Radiographic changes will depend on the stage of the disease and the type of bacteria present. Inititially, radiographic examination may reveal very little except for soft tissue swelling, but later, destructive changes will be seen extending down into the subchondral bone and there will be varying degrees of periarticular new bone formation.

Laboratory tests

Evidence of inflammation is not always consistent on haematological and serological tests. Although a neutrophilia, low-grade anaemia and mild thrombocytopenia are to be expected in positive cases, normal levels do not preclude a diagnosis of infective arthritis. Synovial fluid analysis (see Table 6.1) will usually reveal the following:

- Increased volume
- Reduced viscocity
- Haemorrhagic or purulent appearance
- Increased turbidity and tendency to clot on exposure to air
- Elevated white cell count with polymorphonuclear leukocytes predominating

- Elevated protein levels
- Low blood glucose ratio

Synovial fluid or membrane may be submitted for bacteriology as culturing the infective agent from the joint leads to a definitive diagnosis. Swabs taken from synovial fluid should be plated out both on aerobic and anaerobic culture media. However, even when infection is present, culture of the organism is only successful in some 50% of cases (Montgomery *et al.*, 1989). Culture from synovial membrane biopsies is said to be more successful than direct culture from synovial fluid (Sledge, 1978) but the results are not consistent. The most accurate and reliable method is to culture from synovial fluid following incubation in blood culture medium for 24 hours (Montgomery *et al.*, 1989). The most commonly isolated bacterial causes of infective arthritis are *Staphylococcus intermedius* and β-haemolytic *Streptococci* (Bennett & Taylor, 1988). Other bacteria which may be encountered include:

- Coliforms
- *Pasteurella multocida*
- *Pseudomonas aeruginosa*
- *Proteus* spp.
- *Nocardia asteroides*
- *Brucella abortus* (Clegg & Rorrison, 1968)
- *Erysipelothrix rhusiopathiae* (Houlton & Jefferies, 1989)
- *Salmonella typhimurium*

Treatment

Cases of suspected infective arthritis require urgent treatment. A course of broad-spectrum antibiotic such as amoxycillin and clavulanic acid (Synulox, Pfizer) or cephalosporin (Ceporex, Schering-Plough Animal Health) should be initiated while the results of culture and sensitivity are awaited. An appropriate antibiotic should then be given for at least 3 weeks and it may be necessary to continue for 6 weeks or more before the infection is eradicated. The majority of cases, 88% according to Bennett & Taylor (1988), will have a satisfactory outcome provided treatment is started early, before extensive joint damage has occurred.

Treatment of septic arthritis following joint surgery

Strict asepsis is a prerequisite for successful joint surgery. Nevertheless, infections do occur occasionally and can usually be traced back to faults in asepsis, prolonged operating time or rough handling of tissues. In the acute case there is joint swelling, pain and pyrexia within a few days of surgery. Most cases, particularly those with low-grade infection, will respond to antibiotic therapy, as discussed above. In addition to antibiotics, particularly if infection is thought to be severe, the joint should be flushed out with large quantities of sterile, lactated Ringer's solution. This can be achieved using two needles or catheters introduced into the joint, one for ingress and the other for egress of fluid. Pain relief is provided by the administration of a non-steroidal anti-inflammatory drug (NSAID), such as carprofen (Rimadyl, Pfizer) and, when possible, joint immobilisation using a Robert Jones bandage (see later in Fig. 12.1). If infection has been present for more than 3 days then arthrotomy, rather than needle flushing, is necessary for removal of fibrin deposits, necrotic tissue and purulent exudate. After the joint has been debrided and lavaged with Ringer's solution, the arthrotomy may be left open to granulate and heal by second intention or drains can be placed in the joint prior to wound closure. Irrigation and drainage is achieved by way of these for 3–4 days and then the drains are removed. The wound and drains (if used) must be carefully protected with sterile dressings to prevent interference from the patient and further contamination.

Chronic infections are seen most often following replacement of ligaments with synthetic materials such as braided nylon or Terylene. If infection becomes established in a joint where such an implant has been used, eradication of the causal organism becomes impossible unless the implant is removed, due to its capillary nature which harbours the infection. Lameness, which is often severe, persists for weeks or months following the initial surgery. The joint tends to be thickened and painful and sinus tracts appear over the region. Radiographic examination reveals soft tissue swelling, erosion of the articular surfaces, bone lysis around implants and often extensive

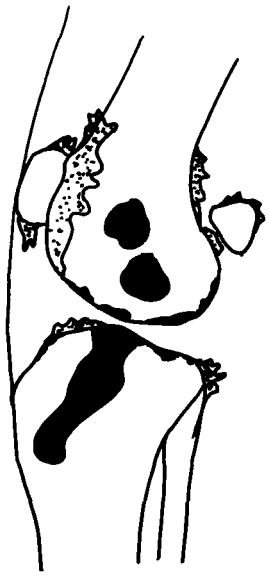

Fig. 9.1 Septic arthritis following cranial cruciate ligament replacement using a nylon prosthesis placed through bone tunnels. There are areas of subchondral bone erosion and extensive periarticular osteophyte formation. Bone lysis around the implant has increased the size of the bone tunnels.

periarticular osteophyte formation (Fig. 9.1). Treatment involves arthrotomy, removal of implanted material, debridement, synovectomy, lavage and irrigation drainage, together with prolonged antibiotic therapy. If culture and sensitivity resulting from an infected joint indicate the use of an antibiotic such as gentamicin, which can have toxic side-effects if given systemically for long periods, then local administration may be achieved by using beads of polymethylmethacrylate impregnated with gentamicin (Septopal, E. Merck). The beads are placed in the joint for up to 3 weeks and slowly release the gentamicin, providing good intra-articular levels of the antibiotic but very low systemic levels, thus minimising the risk of toxic side-effects (Walenkamp *et al.*, 1986; Brown & Bennett, 1988).

Other causes of infective arthritis

With the exception of Lyme disease in the dog and viral arthritis in cats, which are briefly described below, other causes of infective arthritis will not be discussed here as either they do not occur in Britain and/or they are rare. Such infective agents include bacterial L-forms, leishmaniasis, *Mycoplasma*, *Rickettsia* and viruses. For further detail the reader is referred to Abercromby (1994).

Lyme disease or borrelial arthritis

Lyme disease is a multisystem inflammatory disorder associated with infection by the spirochaete *Borrelia burgdorferi* which is carried by ticks. In Europe the vector is the sheep tick *Ixodes ricinus*. Lyme disease was first recognised in the United States in humans and has been diagnosed in a number of domesticated animals during the past 15 years. The first case of Lyme disease in the dog in Britain was reported by May *et al.* (1990). Diagnosis was based on contact with sheep ticks and a positive antibody titre to *B. burgdorferi*. The dog had a chronic foreleg lameness, pyrexia, localised lymphadenopathy and leucocytosis with neutrophilia showing a left shift. Synovial fluid from the elbows had inflammatory characteristics. The lameness responded to antibiotic therapy with clavulanate-potentiated amoxycillin (Synulox, Pfizer).

Viral arthritis in cats

Feline calicivirus (FCV) is a well-recognised pathogen in cats and is responsible for the majority of cases of 'cat flu'. Feline calicivirus has been associated with other clinical conditions besides upper respiratory tract disease. These include a chronic stomatitis and a lameness syndrome (Dawson, 1991). Lameness has been seen in cats following both vaccination and natural infection with FCV. Systemic signs of pyrexia and anorexia often accompany the lameness. The condition tends to be self-limiting and resolves in 24–48 hours. Lameness has also been produced experimentally using an FCV isolate from a lame cat (Pedersen *et al.*, 1983). Feline leukaemia virus and feline syncytia-forming virus infections have been linked to a chronic progressive polyarthritis in cats (Pedersen *et al.*, 1980) (see 'Periosteal proliferative polyarthritis of cats' in Chapter 8, p. 67).

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Chapter 10
Joint-related Neoplasia

Tumours affecting joints have been reviewed by several authors (Dyce, 1994; Whitelock *et al.*, 1997). Primary joint tumours are uncommon, the majority are malignant and synovial sarcomata have been reported most frequently. Tumours such as fibrosarcomas and osteosarcomas may also arise from extra-articular tissues and invade the joint. These tumours must be differentiated from benign primary tumours (rare) and benign conditions which resemble neoplasia, for example synovial osteochondromatosis in the dog or hypervitaminosis A in the cat. A classification of joint tumours and conditions which resemble them is shown in Table 10.1.

Diagnosis of joint tumours

Radiological evidence of periarticular soft tissue swelling with areas of bone destruction on both sides of the joint in an older dog of a larger breed is said to be strongly suggestive of synovial sarcoma (Pool, 1978). However, other malignant tumours may have identical radiographic features and histopathological examination is essential to confirm the diagnosis.

Synovial sarcoma

Synovial sarcomata are uncommon. The tumour occurs most frequently in middle-aged dogs of medium to large breeds. There is a gradual onset of lameness with the development of a soft tissue mass around a joint. The stifle is the most commonly reported site followed by the elbow and hock joints. The tumour is locally invasive and radiographic examination will reveal periarticular soft tissue swelling with varying degrees of bone destruction on one or both sides of the joint (Fig. 10.1). Biopsy and histopathological examination is essential to confirm the diagnosis.

Amputation appears to be the most effective form of treatment (McGlennon *et al.*, 1988) but only some 25% of cases survive for more than a year following diagnosis. The most common sites of metastases are the lungs and local lymph nodes. Chemotherapy (doxorubicin hydrochloride and cyclophosphamide) has been used successfully in the management of one case of synovial sarcoma (Timant *et al.*, 1986).

Synovial osteochondromatosis

Synovial osteochondromatosis is a condition in which numerous foci of cartilage develop in the synovial membrane of a joint or occasionally of a bursa or a tendon sheath. It is thought that metaplasia of the sublining connective tissue of the membrane occurs. The foci of cartilage detach, remain free in the joint space and frequently become ossified. The condition is uncommon with sporadic reports of cases in dogs (Flo *et al.*, 1987; Gregory & Pearson, 1990) and cats (Kealy, 1979). Lesions have been recorded in the shoulder, carpus, hip, stifle and hock joints. The disease is most often seen in mature animals of large breeds. There is chronic lameness with swelling and discomfort evident on manipulation of the affected joint. Radiographs demonstrate extensive calcified deposits in and around the joint (Fig. 10.2). The treatment of choice is total synovectomy with removal of loose bodies but this may prove difficult. Good results have been reported with this type of surgery (Flo *et al.*, 1987). Carpal synovial osteochondromatosis secondary

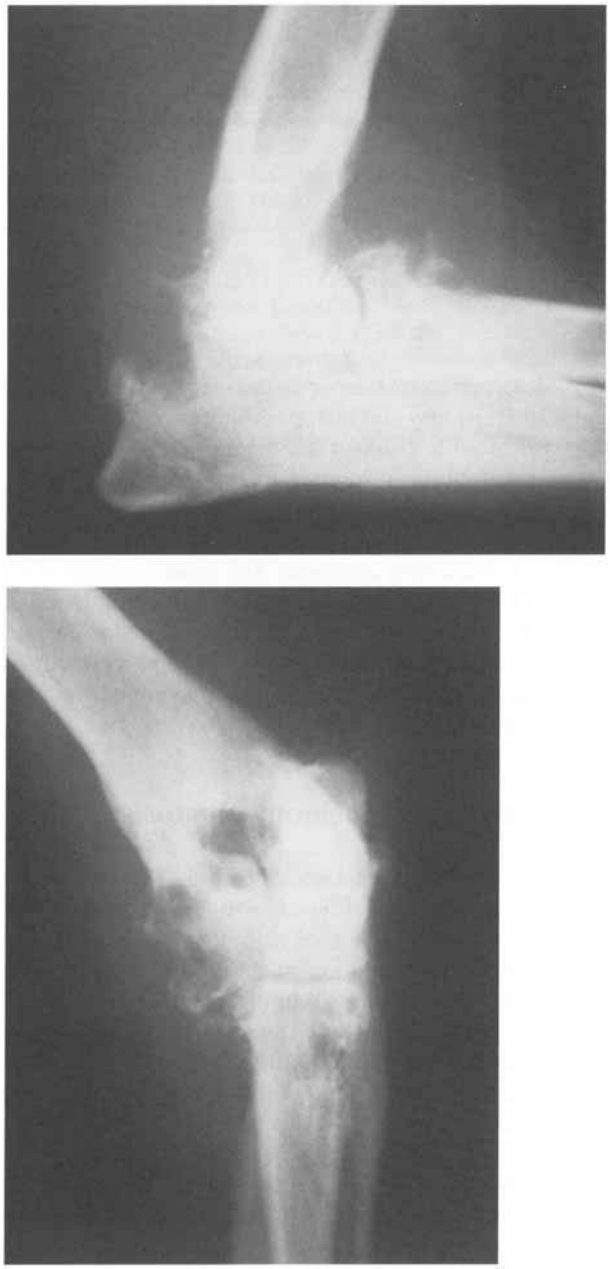

Fig. 10.1 Radiographs of the elbow of a 7-year-old Rottweiler with a synovial sarcoma. There are extensive areas of bone destruction involving the distal humerus and the proximal radius and ulna.

to a chronic subluxation has been treated by pancarpal arthrodesis. Not only did the procedure relieve pain but follow-up radiographs showed extensive remodelling of the periarticular changes associated with osteochondromatosis (Denny, unpublished).

Hypervitaminosis A (see also Chapter 47)

Hypervitaminosis A is seen in cats fed large amounts of liver (Armstrong & Hand, 1989). Although the classical clinical sign is neck

Table 10.1 Types of joint neoplasia and benign conditions which may resemble them.

Primary malignant tumours

- Synovial sarcoma
- Synovial chondrosarcoma
- Liposarcoma

Primary benign tumours

- Synovioma (cats)
- Lipoma

Extra-articular tumours which may invade joints

- Osteosarcoma
- Chondrosarcoma
- Fibrosarcoma
- Rhabdomyosarcoma
- Haemangiosarcoma
- Malignant cell fibrous histiocytoma
- Mast cell tumour
- Squamous cell carcinoma
- Plasma cell tumour
- Undifferentiated sarcoma

Benign intra-articular conditions resembling neoplasia

- Synovial osteochondromatosis
- Villonodular synovitis
- Septic arthritis

Benign extra-articular conditions resembling neoplasia

- Calcinosis circumscripta
- Cartilaginous exostosis
- Craniomandibular osteopathy
- Hypervitaminosis A (cats)
- von Willebrand heterotopic osteochondrofibrosis

Fig. 10.2 Synoval osteochondromatosis. Multiple calcified masses surround the joint.

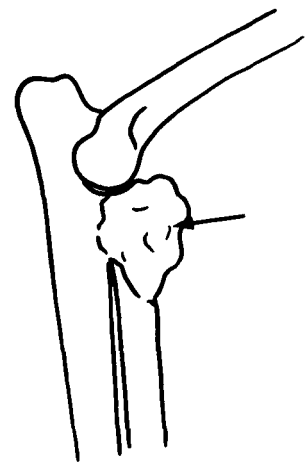

Fig. 10.3 Cat with hypervitaminosis showing elbow exostosis (arrow).

stiffness due to cervical vertebral spondylosis deformans, it is not unusual for exostoses to develop at other sites, particularly around joints such as the elbow (Fig. 10.3). A radiographic example is given later in Chapter 47 (Fig. 47.3). The exostoses may cause lameness and in extreme cases joint ankylosis. Treatment involves a change of diet. Feeding liver should be stopped and lameness treated with non-steroidal anti-inflammatory agents (see Table 7.1). Although this may result in a clinical improvement, the pathological changes seldom resolve.

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Section 3
Fracture Management

A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny,Steven J. Butterworth

Chapter 11
Classification of Fractures

A fracture may be defined as a disruption in the continuity of a bone. The majority of fractures are caused by direct injury in road traffic accidents or falls, the fracture occurring at or near the point of impact. A fracture may also be caused by an indirect force transmitted through bone or muscle to a vulnerable area of bone which breaks in a predictable manner; for example, fractures of the tibial tuberosity, olecranon or lateral epicondyle of the humerus. An incoordinate movement or excessive muscle contraction can result in this type of fracture. Factors which predispose to fracture include the shape and position of the bone; hence long, relatively exposed bones, such as the radius and ulna and the tibia, are more prone to fracture than the short compact bones of the carpus or tarsus. The mechanical strength of bone may be reduced locally, by bone tumour formation, or generally, by disease caused by dietary or hormonal imbalance so that even minor trauma causes a fracture: this is called a *pathological fracture*.

The aim of fracture classification is to form a basis on which to make decisions regarding appropriate management. Using such a system it should be possible to relate the features of a fracture to another clinician who will then have a clear idea of that particular fracture's characteristics. Fractures may be classified according to:

- Anatomical location
- External wounds
- Extent of bone damage
- Direction of fracture line
- Relative displacement of the bone fragments
- Stability

Anatomical location

It is customary to refer to specific fractures of a long bone according to their anatomical location, i.e. *proximal*, *distal* or *diaphyseal* (Fig. 11.1). The proximal or distal fractures can be further subdivided into *articular*, *epiphyseal*, *physeal* or *metaphyseal* fractures. Fractures of the physis are divided into six types (Salter & Harris, 1963; Fig. 11.2). Diaphyseal fractures can be further classified according to the direction of the fracture line or the number of fragments.

External wounds

A *closed fracture* is one in which the overlying skin remains intact (Fig. 11.3a). An *open fracture* is one in which there is a communication between the fracture site and a skin wound (Fig. 11.3b). Open fractures are classified as first, second or third degree according to the severity of soft tissue injury and contamination (see Chapter 14, p. 153).

Extent of bone damage

A *complete fracture* is one in which there is total disruption of the continuity of the bone and usually marked displacement of the fragments (Fig. 11.4a). An *incomplete fracture* is one in which partial continuity of the bone is maintained as in the greenstick (bending) fractures of young animals (Fig. 11.4b) or fissure fractures in mature animals (Fig. 11.4c).

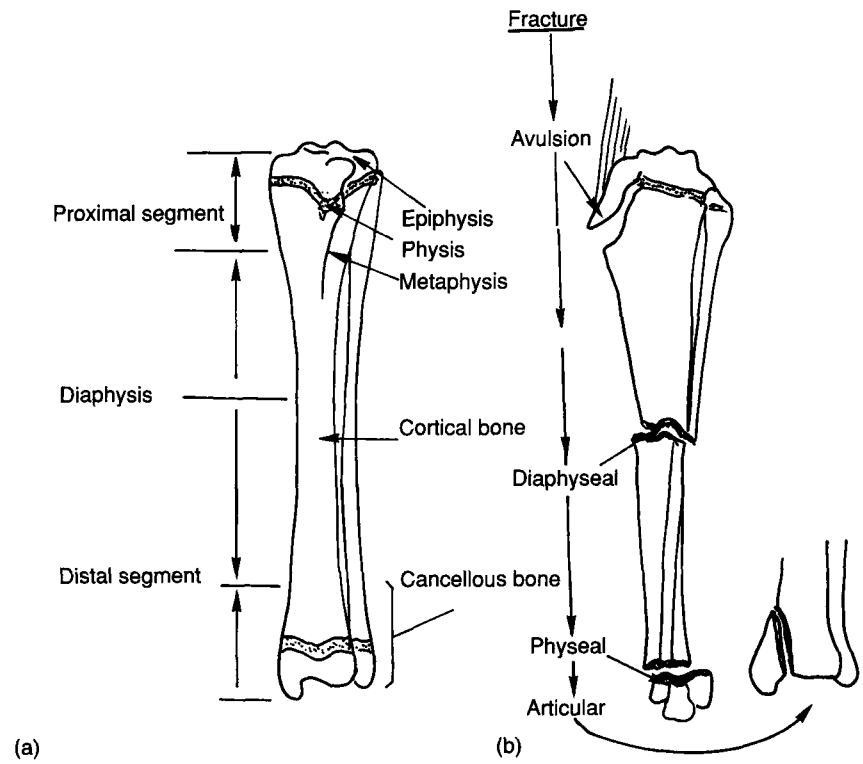

Fig. 11.1 Based on anatomical location (a) classification of fractures and (b) type of fracture.

Direction of fracture line

- A *transverse fracture* is one in which the fracture line is at right angles to the long axis of the bone (Fig. 11.5a).
- An *oblique fracture* is one at an angle to the long axis of the bone (Fig. 11.5b). A short, oblique fracture line has a length that is less than about twice the diameter of the bone.
- A *spiral fracture* curves around the bone (Fig. 11.5c).
- A *comminuted fracture* is one in which there are several fragments and the fracture lines communicate (Fig. 11.5d). The percentage of bone length involved is often estimated in these fractures.
- A *segmental (or multiple) fracture* is one in which the bone is broken into three or more segments such that the fracture lines do not communicate (Fig. 11.5e).

Relative displacement of the fragments

- An *avulsion fracture* is one in which a bone fragment is distracted by the pull of the muscle tendon or ligament which attaches to it, for example avulsion of the tibial tuberosity (Fig. 11.1b).
- An *impacted fracture* is one in which the fractured bone ends are driven into one another (Fig. 11.5f).
- A *compression fracture* refers, typically, to a fracture of a vertebra where a compressive force has resulted in shortening of a vertebra.
- A *depression fracture* usually refers to skull fractures in which the affected bone is 'pushed in', giving a concave deformity.

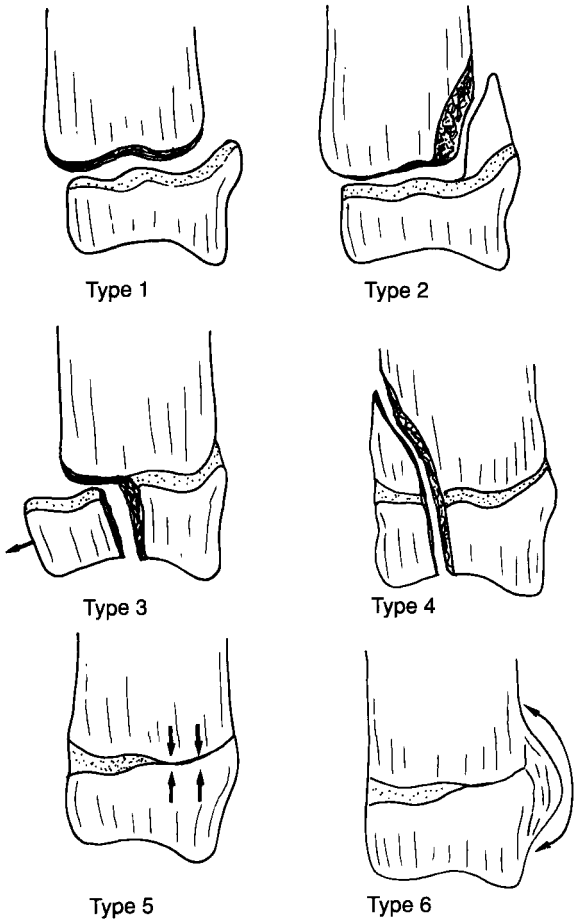

Fig. 11.2 Salter Harris classification of growth plate (physeal) injuries.

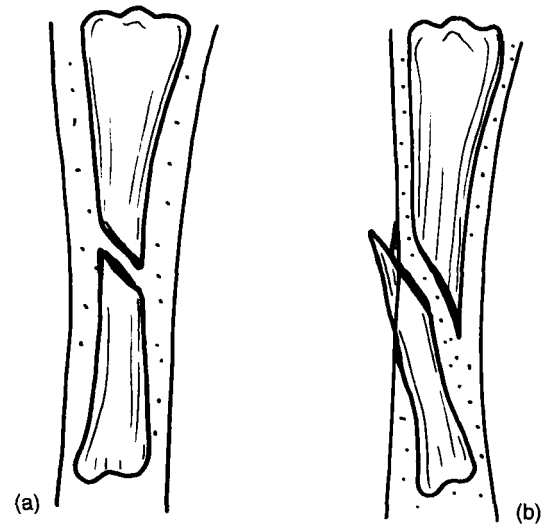

Fig. 11.3 Classification of fractures. (a) Closed fracture, (b) open fracture.

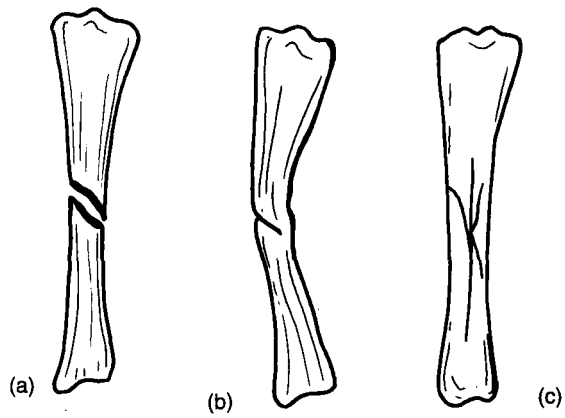

Fig. 11.4 Classification of fractures according to the extent of bone damage. (a) Complete fracture, (b) incomplete (greenstick) fracture, (c) incomplete (fissure) fracture.

Stability of the fracture

The assessment of a fracture's inherent stability is of particular importance when selecting the appropriate method of fracture fixation. What is the fracture's resistance to shortening, rotation and angulation? Diaphyseal fractures can be broadly divided into stable and unstable fractures.

Stable fractures are the transverse, blunt (short) oblique or greenstick fractures in which the fragments interlock and resist shortening forces. The only fixation necessary is to prevent angular deformity and, sometimes, rotation. Depending on site, this can be achieved either by external coaptation, or by application of an intramedullary pin, an external fixator or a plate.

Unstable fractures are oblique, spiral or comminuted. The fragments do not interlock and a method of fixation is needed which will maintain

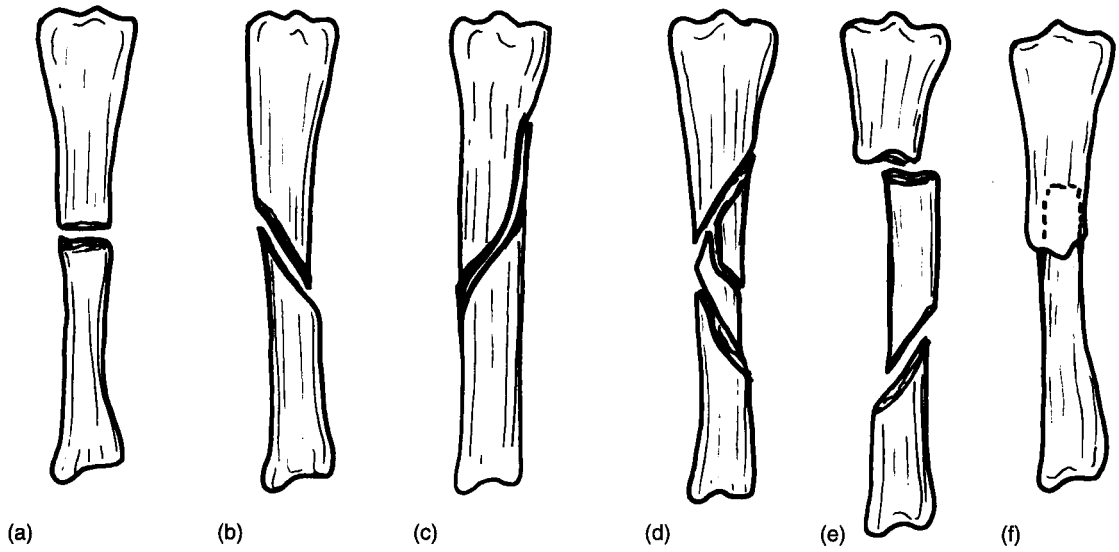

Fig. 11.5 Classification of fractures according to direction of the fracture line. (a) Transverse, (b) oblique, (c) spiral, (d) comminuted, (e) segmental, (f) impacted.

the length of the bone and prevent angular deformity and rotation. This usually involves application of a plate and screws or an external fixator.

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Chapter 12
Options in Fracture Management

Preliminary examination and management of the fracture patient

Initial examination

Orthopaedic injuries alone are seldom life-threatening unless they are associated with gross haemorrhage. A soft tissue swelling the size of a clenched fist around a fracture site is equivalent to approximately 750 ml of blood. Wounds, fractures and dislocations are usually obvious on clinical examination and there is a natural tendency to concentrate on these and miss the more serious internal injuries. Thoracic injuries, particularly pneumothorax, are common complications of fractures, particularly those involving the humerus and scapula. In *all* road traffic accident cases, a careful clinical and radiological examination should be done to rule out significant thoracic injury, which should be treated before embarking on fracture fixation. Many chest injuries, including diaphragmatic rupture, are easily missed on clinical examination but are detected on radiographic examination. Cases with tension pneumothorax or extensive pulmonary haemorrhage are obviously an anaesthetic risk and surgery should be delayed (usually a matter of days) until resolution occurs. Nitrous oxide will rapidly increase the volume of an existing pneumothorax and should not be used in the anaesthesia of such cases. In addition, a serious potential risk of pelvic fracture is rupture of the bladder or urethra. Fortunately, this type of injury is uncommon. In a review of 123 pelvic fracture cases (Denny, 1978), only one had bladder rupture and two had rupture of the urethra. Nevertheless, if there is any doubt about the integrity of the bladder or urethra, then cystography and/or urethrography should be undertaken. The protocol for initial assessment and management of acute trauma cases is well documented and for further information the reader is referred to Houlton & Taylor (1987).

The priorities in the assessment and management of accident cases can be divided into 'general priorities' and 'local priorities'. These are listed below. Orthopaedic injuries are at the bottom of the list and it is important to attend to the other problems first before undertaking fracture fixation or reduction of dislocations.

General priorities:

- Maintain an airway
- Maintain blood volume
- Relieve pain

Local priorities:

- Head injuries (see Chapter 16)
- Thoracic injuries
- Abdominal injuries
- Spinal injury (see Chapter 21)
- Orthopaedic injuries (see Sections 5 and 6)

Dislocations and ligamentous ruptures tend to be seen in mature dogs and cats over 1 year of age. The same trauma in immature animals is more likely to cause a fracture or physeal separation.

Basic management of dislocations

In all dislocations, reduction should be undertaken as soon as possible after the accident, preferably within 24 hours. After reduction of a hip dislocation, the leg may be strapped in flexion using an Ehmer sling for 5 days (see Chapter 40, Fig. 40.6). In cases of shoulder dislocation, a sling or body cast may be applied for 2–3 weeks to prevent redislocation (see Chapter 31, Figs

31.22–31.24). The elbow, by contrast, requires little external support as good stability is restored immediately following reduction in most cases, although some cases may show incomplete reduction whereupon surgery may be indicated (see Chapter 33, p. 382).

First-aid procedures for the temporary immobilisation of fractures or injured joints

Robert Jones bandage

The Robert Jones bandage is a thick cotton-wool bandage which acts as a splint and controls oedema. For these reasons, it is useful not only as a first-aid measure for the temporary immobilisation of fractures, but also as a postoperative bandage for fractures which have been treated surgically. The bandage is comfortable to wear and is generally well tolerated, despite its bulk.

Although the Robert Jones bandage is ideal to splint fractures distal to the elbow or stifle, it should not be used for fractures of the humerus or femur as the bandage will simply act as a weight causing further fracture displacement and pain through a 'pendulum' effect.

Application of a Robert Jones bandage (Fig. 12.1):

(1) Strips of adhesive tape (Elastoplast, Smith & Nephew) or zinc oxide are placed down the dorsal and palmar/plantar aspects of the foot (Fig. 12.1a). These will prevent the bandage slipping and can be used to produce traction during application of cotton wool layers.

(2) A 1 lb roll of cotton wool is split into two narrower $\frac{1}{2}$lb rolls (possibly split further for small dogs) and these are used like a bandage and wrapped around the leg (Fig. 12.1b). The first layer of cotton wool is 'compressed' with an open weave or conforming bandage (Fig. 12.1c). Another layer of cotton wool is applied, then more bandage, and so on until there is sufficient support. Generally, two layers of cotton wool will suffice but more are added if necessary. The total amount of cotton wool required ranges from $\frac{1}{2}$ to 2 lb depending on the size of the dog and site of the fracture.

(3) The ends of the Elastoplast strips are flapped back to reveal the pads of the central toes and then attached to the end of the bandage using Elastoplast (Fig. 12.1d). The whole bandage is then tightly compressed with a Vetrap (3M) elasticated bandage or Elastoplast.

Thomas extension splint (Fig. 12.2)

Fractures proximal to the elbow and stifle are difficult to immobilise by external coaptation. Fortunately, the humerus and femur are surrounded by large muscle masses which provide a degree of natural splintage, but if further immobilisation is needed prior to surgical treatment then the Thomas extension splint is used. Although this splint can also be used as the sole method of fixation for stable fractures below the elbow or stifle, it is generally used only as a temporary splint for limb bone fractures.

The splint is usually constructed from an aluminium rod. The mid-section of the rod is fashioned into a ring which fits around the base of the leg (Fig. 12.2a). The free ends of the rod are then bent down to form bars on both the cranial and caudal aspects of the leg (Fig. 12.2b). If the splint is applied to the hind leg, the ring is first bent at an angle to avoid excessive pressure on the inguinal region and femoral blood vessels. The ring is well padded with cotton wool (Fig. 12.2c). The splint is pushed firmly up into the inguinal, or axillary, region and the cranial bar of the splint is bent to conform to the leg's normal angulation in a standing position. Elastoplast (Smith & Nephew) strips are used to fix the foot to the end of the bar (Fig. 12.2d). The upper part of the leg is also attached to the cranial bar with Elastoplast (Smith & Nephew). Finally, a thick band of Elastoplast (Smith & Nephew) is placed around both bars and the hock (Fig. 12.2e).

Non-weight-bearing sling bandages (Fig. 12.3)

These bandages are used to immobilise shoulder and scapula injuries and to prevent weight-bearing. A conforming gauze bandage is wrapped around the paw, the leg is flexed and the bandage is brought up over the lateral aspect of the shoul-

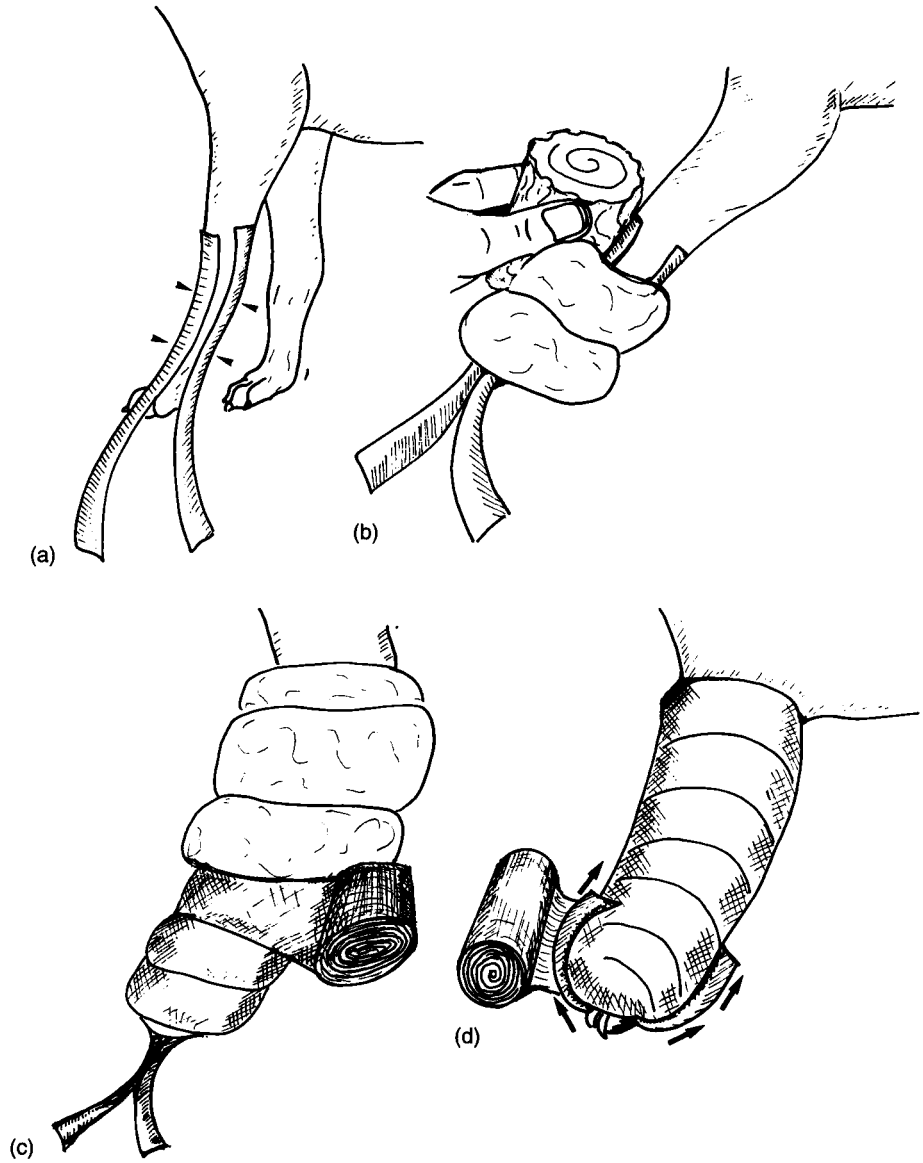


Fig. 12.1 Application of the Robert Jones bandage. See text for detail.

der and around the chest. Several layers are applied and then covered with Elastoplast (Smith & Nephew). For further details see Chapter 31, Figs 31.22 and 31.23.

The Ehmer sling (Fig. 12.4)

The Ehmer sling prevents weight-bearing on the hind limb. Its main use is to provide partial immobilisation of the hip after reduction of a dislocation or the repair of a hip fracture. Details of its application are given in Chapter 40,

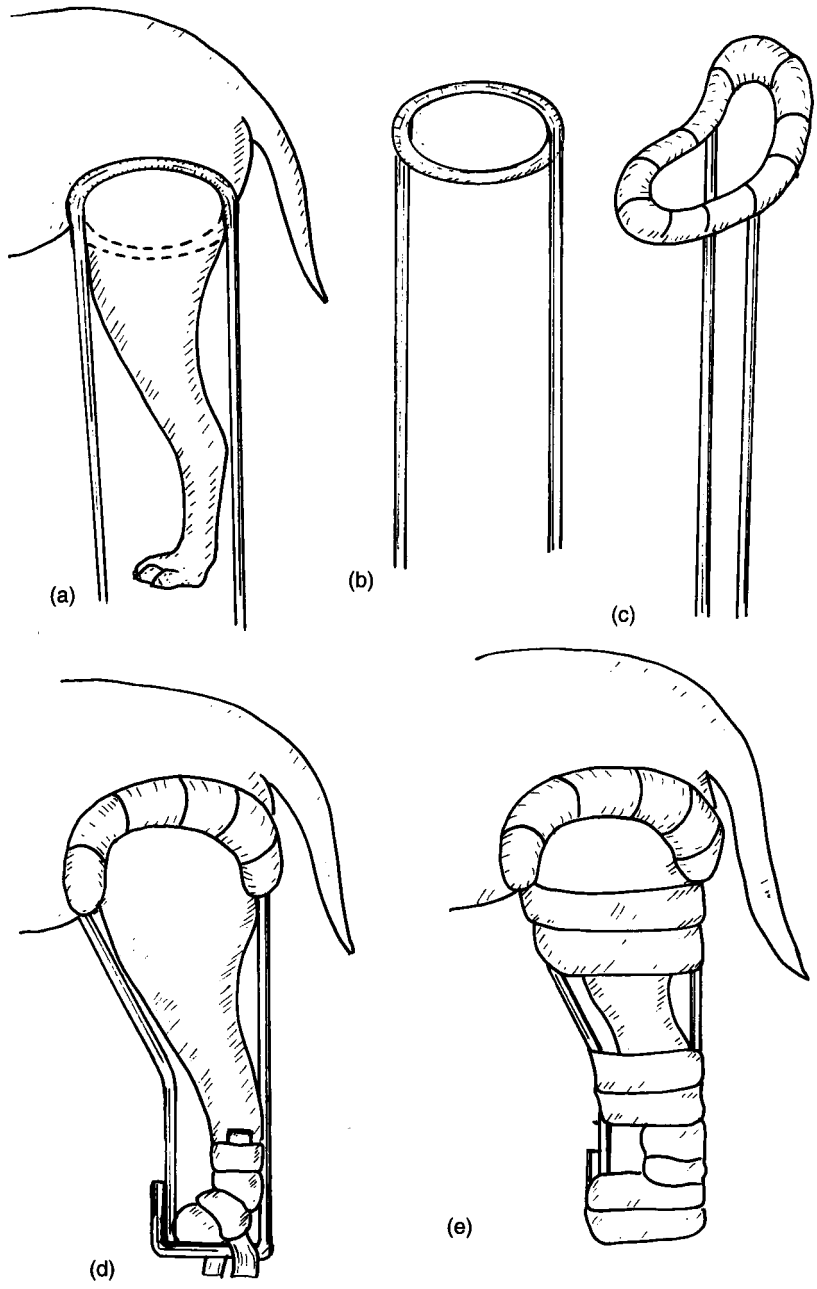


Fig. 12.2 Application of the Thomas extension splint. See text for detail.

Fig. 40.6. The Ehmer sling *should not* be used as a first-aid dressing in hip fractures as it tends to displace the fragments and cause the animal more pain.

Considerations in choosing a definitive method of treatment

Once a fracture has been reduced, and provided the blood supply to the fragments is intact, the

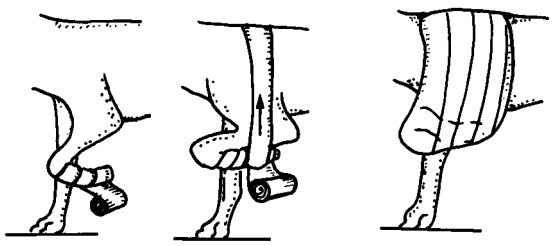


Fig. 12.3 Application of a non-weight-bearing sling.

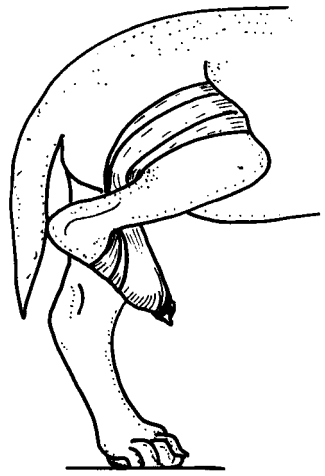


Fig. 12.4 Ehmer sling.

main requirement for successful healing is the provision of adequate immobilisation. Selection of the appropriate method of fracture fixation will primarily depend on the site of fracture and the inherent stability of the fragments. In evaluating such stability of a reduced fracture an assessment is made of its resistance to:

- Axial forces, i.e. will it resist shortening if compressed longitudinally?
- Angular forces, i.e. will it resist bending?
- Torsional or rotational forces, i.e. will it resist rotation?

Fractures can be divided into two main groups. The *stable fractures* (transverse or blunt oblique), where the fragments interlock and resist shortening, only require fixation to prevent bending or angular deformity and certainly one of the ways of doing this is with a splint or cast. Conversely, non-surgical methods of immobilisation are generally unsatisfactory for the management of *unstable* fractures (oblique, spiral or comminuted), where the fragments do not interlock and cannot resist shortening, angulation or rotation. Fixation is needed to maintain length, alignment and prevent rotation and, as a general rule, this is best done by internal fixation rather than using external coaptation but, as with all rules, there are exceptions.

Non-surgical, or conservative, methods of fracture immobilisation include:

- Cage rest
- Robert Jones bandage
- Casts and splints (external coaptation)

Surgical methods of fracture immobilisation include:

- External skeletal fixation
- Intramedullary devices (Steinmann pins, Rush pins, interlocking nails)
- Orthopaedic wire
- Bone screws
- Bone plates
- Combinations of more than one of the above

Cage rest

Many fractures of the scapula and of non-weight-bearing areas of the pelvis, i.e. pubis and ischium, will heal satisfactorily with cage rest. The animal is confined to a cage for 4–6 weeks. This form of management should include standard nursing procedures such as the provision of adequate analgesia, attention to urinary and bowel function and prevention of decubitus ulcers.

Many cats with orthopaedic injuries seem to get better despite treatment. They respond well to cage rest and if there is any doubt about the correct management of a pelvic or limb bone fracture in a cat then cage rest often gives satisfactory results, though this is not to say that the likelihood and degree of success could not be improved by considering surgery.

Robert Jones bandage

Although the Robert Jones bandage is used most frequently as a first-aid measure for temporary

immobilisation of fractures, the bandage can be used as the definitive method of immobilisation when treating puppies with greenstick fractures and undisplaced epiphyseal separations below the elbow and stifle. The bandage is usually well tolerated and can be left on for periods of 2–4 weeks. The method of application has been described above.

Casts and splints (external coaptation)

The use of a cast or splint to immobilise a fracture until healing occurs is referred to as *external fixation* or *coaptation*. The latter term is preferred to avoid confusion with external skeletal fixation.

The basic criteria for using external coaptation are:

- It must be possible to immobilise the joint proximal and distal to the fracture. Therefore, the method is limited to fractures distal to the elbow and stifle.
- Casts and splints only resist angular deformity at the fracture site and so they are reserved for transverse or short oblique fractures which can resist all but angular forces once reduced.
- At least 50% of the fracture surfaces should be in contact following reduction for satisfactory healing to occur using these methods.
- Fractures in young animals tend to heal remarkably quickly and so casts may be used with greater success in these patients.
- Articular fractures and avulsion fractures should not be treated by external coaptation.
- External coaptation may be used to provide additional support when a fracture has been treated by internal fixation, in order to prevent excessive weight-bearing which might lead to implant failure.

The advantages of external coaptation are:

- It requires no surgical intervention and so the soft tissues are not compromised further
- It requires a shorter anaesthetic time
- It requires little in the way of materials
- It carries a *potentially* lower cost

Fracture reduction

Fracture of a long bone is invariably associated with overriding of the fragments and shortening as a result of muscle contracture and spasm. The process of returning the bone fragments to their normal anatomical position is referred to as *fracture reduction*. Slow, steady traction is used to overcome muscle spasm. The fracture is bent to allow the ends of the fragments to be brought into partial contact and thus provide a fulcrum for leverage until reduction is completed (often referred to as 'toggling' the fragments). If the fracture is very unstable following reduction, or proves difficult to reduce, then open (surgical) reduction and internal fixation should be used rather than external coaptation.

Gutter splints

Although gutter splints can be improvised from plastic, metal or cast material it is easier to use commercial, plastic gutter splints (Veterinary Drug Company). This type of straight gutter splint is used most often for immobilising the radius and ulna or carpus. The splint is applied to the palmar surface of the leg. The leg is bandaged with at least two layers of cast padding (Soffban, Smith & Nephew), followed by a layer of Elastoplast (Smith & Nephew). Next the splint is applied to the palmar aspect of the leg and held in place with a final layer of Elastoplast (Smith & Nephew). *Note* the splint should not be applied directly to the layer of Soffban (Smith & Nephew) as it will tend to slip and cause pressure sores.

If a gutter splint is used on the hindlimb then a degree of hock flexion is required to allow the animal to weight-bear. The leg is bandaged as above. Angulation and immoblisation of the hock is achieved by moulding a slab of casting material to the plantar aspect of the joint to form a gutter splint or half-cast. Once the material is dry it is held firmly in place with Elastoplast (Smith & Nephew).

Dynacast Prelude (Smith & Nephew) is a padded fibreglass bandage designed specifically as a synthetic acute splint system. It is ideal for moulding to the contours of the leg to form a comfortable, strong gutter splint. A cheaper alterna-

tive is to use Orthoboard (Millpledge). This is a thermoformable, cohesive, orthopaedic support material which is first softened by immersion in boiling water. It can then be easily moulded to the shape of the leg and sets within a few minutes, reaching maximum strength within 15 minutes.

The advantage of gutter splints over conventional casts is that they are easier to change or remove, particularly when wound dressings need replacement.

Casts

The use of casts for the immobilisation of fractures is widely practised in veterinary orthopaedics. The ideal properties of a casting material include:

- Easy to apply
- Have good conformability
- Have a high strength:weight ratio
- Should set quickly
- Should achieve maximum strength quickly
- Should be resilient to wear
- Cheap/reusable
- Easily removed
- Non-irritant
- Radiolucent

Although the traditional casting material, plaster of Paris, is still popular, a variety of alternative casting materials have been introduced during the past 20 years. These can be divided into three broad types:

(1) *Plaster of Paris and its modifications* (e.g. Cellamin, Lohmann). Plaster of Paris is cheap and easy to apply. However, it takes 8 hours to dry, is heavy and is relatively radiodense.
(2) *Thermoplastic materials* (e.g. Hexcelite, Hexcel UK; Vetlite, Runlite UK). These materials are stronger and lighter than plaster. However, they require a much higher temperature to activate and can be difficult to conform to small limbs. Setting time is 8–10 minutes.
(3) *Fibreglass materials* (e.g. Vetcast, 3M; Zimflex, Zimmer UK). These are very light and strong. They conform well, set within 3–5 minutes and have the advantage of being reasonably radiolucent.

A comparison and assessment of different casting materials (Table 12.1) currently available for use in small animals was made by Langley-Hobbs *et al.* (1996a) and as a result of this study they produced a guide to suitable uses for the different casting materials (Table 12.2).

Principles of cast application

(1) Casts are normally applied to limbs so as to approximate a normal standing position.
(2) The cast should immobilise the joint above and below the fracture.
(3) The cast should include the whole foot or extend to the tips of the toes, leaving the

Table 12.1 Casting materials (modified from Langley-Hobbs *et al.*, 1996a).

Product	Manufacturer	Composition	Setting time (minutes)
Gypsona	Smith & Nephew	Gypsum	10
Cellamin	Lohmann	Gypsum + resin	10
Dynacast Pro	Smith & Nephew	Polypropylene + resin	3–5
Dynacast Extra	Smith & Nephew	Fibreglass substrate + resin	3–5
Vetcast 2	3M	Fibreglass substrate + resin	3–5
Vetcast plus	3M	Fibreglass substrate + resin	3–5
Zimflex	Zimmer UK	Fibreglass substrate + resin	3–5
Dynacast Prelude	Smith & Nephew	Fibreglass	2–4
Turbocast	Transthermo Systems	Thermoplastic sheets	10–15
Hexcelite	Hexcel UK	Thermoplastic polymer	8–10
Vetlite	Runlite UK	Thermoplastic polymer	8–10
Orthoboard	Millpledge	Thermoformable board	5–15

Table 12.2 A guide to suitable uses for different casting materials (modified from Langley-Hobbs *et al.*, 1996a).

Bodyweight of animal	Mature animal or cast as sole method of stabilisation	Immature animal or cast as supplement to internal fixation
>25 kg	Fibreglass	Cellamin Hexcelite Vetlite Fibreglass
10–25 kg	Fibreglass Cellamin Hexcelite Vetlite	Cellamin Dynacast Pro
5–10 kg	Dynacast Pro Cellamin Gypsona	Turbocast Gypsona
<5 kg	Turbocast Gypsona	Turbocast

central pads exposed for weight-bearing. Casts which end above the foot are potentially dangerous as they can compromise the circulation distally and might result in gangrene.

(4) When applying a cast many authors recommend the use of slabs (see 'Application of a plaster of Paris cast' below). This improves strength and allows for more even application than circumferential wrapping of the bandage around the limb. However, although the newer materials, such as Vetcast (3M), can be applied as slabs they are easier to apply by circumferential wrapping and this is unlikely to result in significant weakening of casts made from these materials.

Application of a plaster of Paris cast to immobilise an undisplaced fracture of the radius and ulna (Fig. 12.5)

(1) The dog or cat should be anaesthetised for application of the cast. Strips of Elastoplast (Smith & Nephew) are applied down the dorsal and palmar aspects of the leg starting just proximal to the carpus (Fig. 12.5a). These strips can be used to exert some traction on the leg during application of the cast.

(2) A stockinette is applied to the limb. This should extend from above the elbow and end below the foot (Fig. 12.5b).

(3) Two or three layers of cast padding (Soffban, Smith & Nephew) are applied next, bandaging from the toes to the elbow (Fig. 12.5c).

(4) An assistant holds the leg in extension. One hand is used to steady the leg above the elbow while the other exerts traction on the Elastoplast (Smith & Nephew) foot tapes. Two slabs of plaster of Paris are prepared; they should be four layers thick and of sufficient length to extend from elbow to foot (Fig. 12.5d).

(5) The slabs are soaked in lukewarm water and then applied to the cranial and caudal aspects of the leg (Fig. 12.5e). Light but firm pressure is then applied up and down the cast to ensure that the slabs are evenly applied and conform to the shape of the leg.

(6) A roll of plaster of Paris is taken, the free end of the bandage held and the roll immersed in water until it is completely saturated and all air bubbles have stopped coming out of it. Excess water is squeezed from the plaster and the roll is applied over the slabs in a wrap-around fashion starting from the foot and working up the leg (Fig. 12.5f).

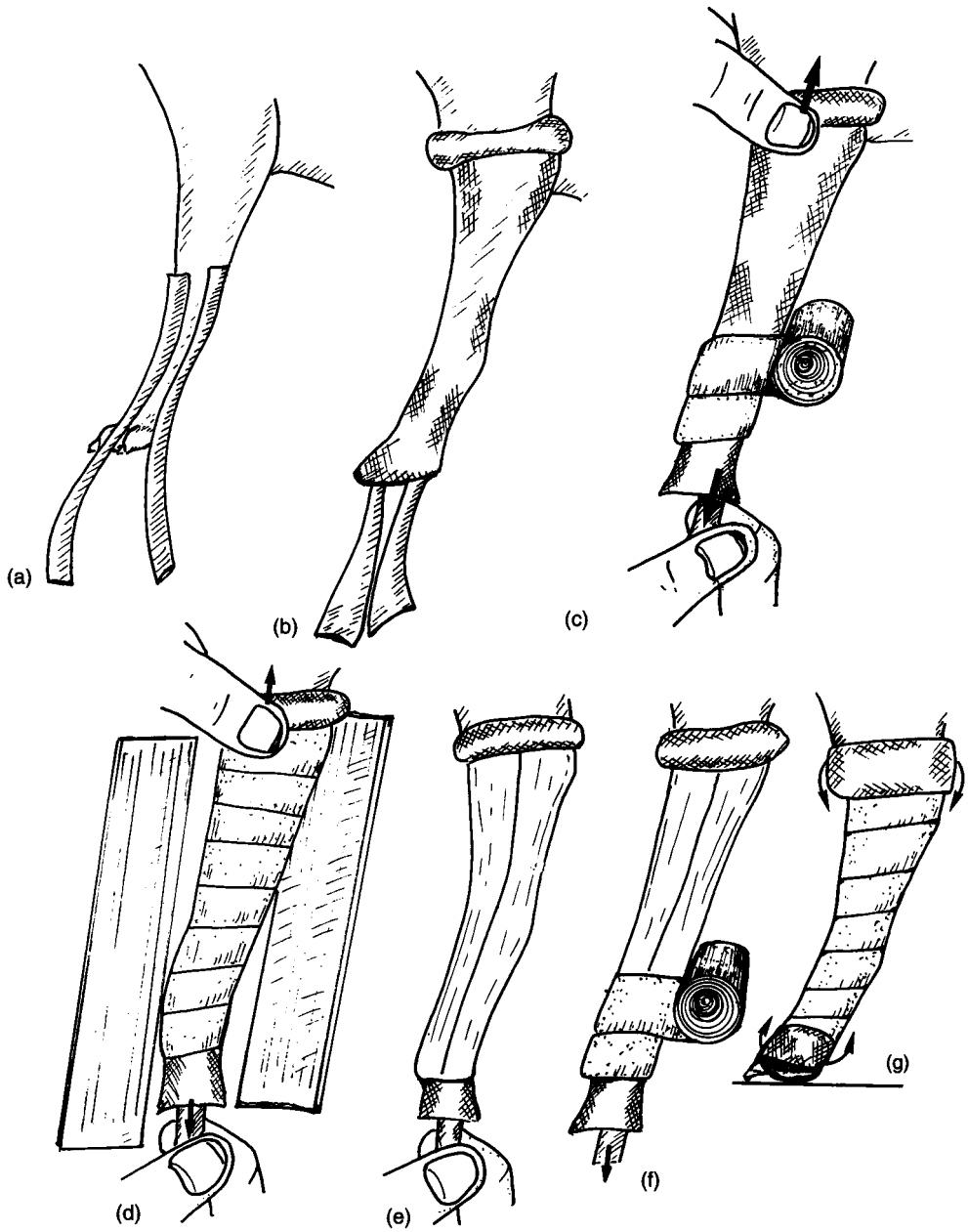


Fig. 12.5 Application of a plaster cast to immobilise a radius and ulna fracture. See text for detail.

(7) The stockinette is folded over at the top and bottom of the cast to give it a smooth edge (Fig. 12.5g). Next, the foot tags are also folded back and fixed to the distal end of the cast with a little more plaster. It is important that the stockinette at the distal end of the cast is folded back sufficiently for the pads to be exposed so that they can be checked regularly to ensure that the circulation to the foot has not been compromised. Although

plaster of Paris sets rapidly the cast will take at least 8 hours to dry out. Once it has dried, several layers of Elastoplast (Smith & Nephew) are used to protect it.

Care of casts

The cast should be inspected by a veterinary surgeon or nurse at least once a week. The owner should check the cast each day to ensure that it feels warm and is not rubbing. The foot pads should be checked daily to ensure that the circulation to the foot is not being compromised. Warn owners that if the cast begins to smell or the dog starts to chew frantically at the cast, stops putting weight on the leg or goes off its food then veterinary attention must be sought immediately and the cast changed.

Casts will usually need changing during the course of treatment, especially if applied over a swollen leg because once the swelling reduces in size the cast will become loose. To overcome this problem the leg may be supported initially with a Robert Jones bandage to reduce limb swelling before application of the cast. Alternatively, the cast is changed after 2 weeks when swelling has subsided and a second cast applied for a further 4 weeks. Typically, a fracture will require support for about 6 weeks before there is sufficient bridging callus to maintain stability. If healing is not expected within 6 weeks, for example because of the dog's age or the degree of comminution, then external coaptation is probably an inappropriate choice for management.

Potential complications of external coaptation

These include:

- *Soft tissue problems.* The most common complication associated with casts is the development of *pressure sores* due to poor casting technique or loosening. Casts which end above the foot or are chewed back by the patient to leave an edge which digs into the dorsum of the foot can lead to swelling and ischaemia which, if untreated, can lead to disaster with gangrene of the foot. This situation should be preventable by proper casting technique and aftercare. If an animal will not leave a cast alone then an alternative method of management may need to be found.
- *Fracture disease.* This is the syndrome of muscle wasting, joint stiffness, soft tissue adhesions and osteoporosis which occur when a limb is immobilised for a long time. Application of a cast is one way of creating such immobilisation and may lead to fracture disease, particularly if the cast is left on for longer than about 6 weeks.
- *Malunion.* This results from improper reduction and/or poor immobilisation of the fracture during the healing process and is more likely to occur with external coaptation than with surgical treatment. Mild degrees of malunion involving fractures of the mid-diaphysis of long bones may be clinically acceptable and cause no functional problem. This will become less true as the severity of malunion worsens or the closer to a joint it is located.
- *Delayed or non-union.* These are also more common complications with external coaptation than with internal fixation and are invariably due to inadequate immobilisation of the fracture.

Economics of external coaptation

When the cost of fracture treatment using casts is considered, the initial reaction is that a cast is going to be cheaper than internal fixation. It is important to remember, however, that it may be necessary to change a cast several times and this involves both time and general anaesthesia so that, ultimately, it can prove less expensive and certainly less time-consuming to treat a fracture surgically in the first place rather than using a cast.

Surgical treatment of fractures – general principles

Surgical or open reduction of fractures and internal fixation is widely used in veterinary orthopaedics. Major advances in the surgical management of fractures continue to be made, particularly with the work of the AO/ASIF and the AO-VET groups.

The AO group (Association for the Study of Osteosynthesis) was formed by a group of Swiss surgeons in 1958. Later the group also became known as ASIF (Association for the Study of Internal Fixation). In 1970 the veterinary equivalent of this association was formed and is know as AO-VET. The AO/ASIF group defined biomechanical principles for the successful treatment of fractures by internal fixation. The basic research was done at the Laboratory for Experimental Surgery in Davos, Switzerland. Metallurgical expertise was gained from the watch industry and an entire system of implants and instruments was developed for fracture treatment.

The aim of the AO/ASIF method is to restore full function to the injured limb as quickly as possible whilst fracture healing is taking place, so as to avoid the repercussions of fracture disease mentioned above under 'Potential complications of external coaptation'. This is achieved by:

(1) Atraumatic surgical technique.
(2) Accurate anatomical reduction of the fragments, especially in intra-articular fractures.
(3) Rigid internal fixation.
(4) The avoidance of soft tisue damage and fracture disease, i.e. joint stiffness, muscle wasting and osteoporosis, by early mobilisation of the limb.

Rigid fixation is achieved by using compression techniques. This may take the form of:

- Functional compression as in tension band wiring.
- Interfragmental compression, for example by use of lag screws or cerclage wires.
- Axial compression, which is achieved with a plate or a wire using the tension band principle, in which the fixation device is placed on the tension side of the bone (see later).
- Interfragmental compression used in combination with a neutralisation plate or external fixator.

These compression techniques are discussed in more detail later in the chapter. Although the work of the AO/ASIF group has been directed towards compression techniques which will promote primary bone union (see Chapter 1, p. 14), there has been a change in emphasis in recent years, particularly in the management diaphyseal fractures, towards less rigid fixation techniques such as the external skeletal fixator which result in 'bridging osteosynthesis' at the fracture site (see Chapter 1, p. 14). In some cases bone reconstruction cannot be achieved and so compression techniques cannot be used. In these instances there is little option other than bridging the fracture gap with a buttress plate, external skeletal fixator or interlocking nail.

External skeletal fixators (ESF)

The use of external fixators to treat human injuries was first reported in 1897 and a fixator specifically for veterinary use was designed in the late 1940s by Ehmer, based on a human design. Their use in man declined following a high incidence of complications associated with the treatment of fractures during World War II. This was compounded by interest being drawn to recent advances in the application of bone plates and screws in the treatment of fractures by internal fixation. More recently, extensive work has allowed significant improvements in fixator design, the materials used and in the understanding of the principles and techniques of application. This led to a significant reduction in complications since a lack of such understanding was responsible for the majority of the earlier failures. As a result, there has been a resurgence of interest in their use in both human and veterinary orthopaedics.

Equipment

The components of an external skeletal fixator (ESF) are illustrated in Fig. 12.6. Percutaneous fixation pins are secured into the bone and then to one another externally, usually by means of a connecting bar and clamps or a 'tube' of acrylic. Depending on the type of ESF constructed, the pins may pass through the skin and both bone cortices ('half-pins') or continue on and through the skin on the opposite side when constructing a bilateral frame ('full pins').

Fixation pins

Threaded or non-threaded Steinmann pins, Ellis pins, Imex pins or Kirschner wires can be used for

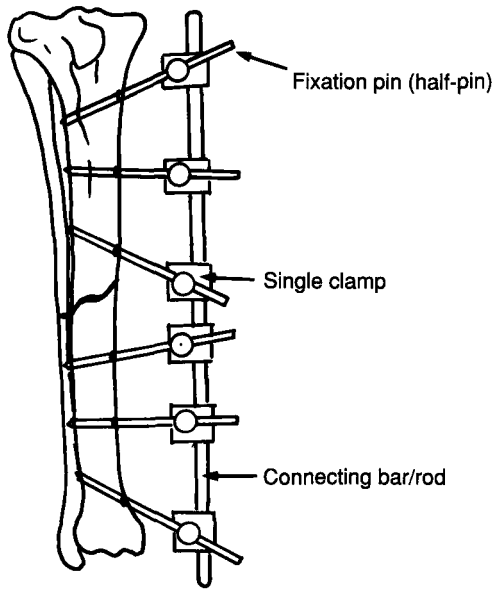


Fig. 12.6 Six-pin, single bar, unilateral, uniplanar (type I) configuration illustrating the components of a fixator.

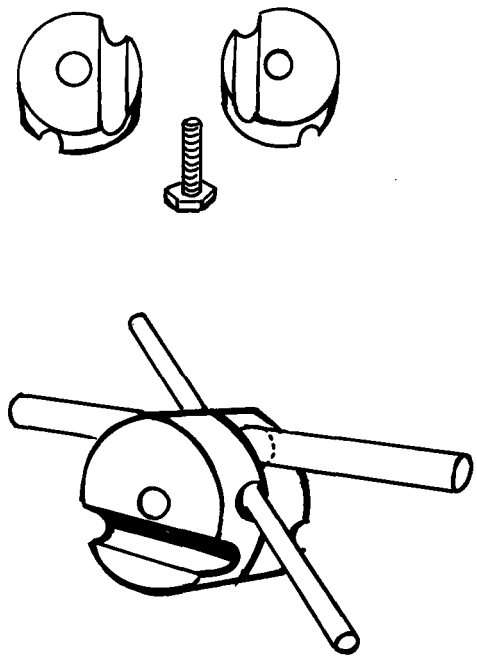


Fig. 12.7 A Maynard clamp. See text for further details.

this purpose. Threaded pins offer certain advantages in that once they are engaged in the bone they tend to drive themselves through and also gain better purchase (Howard & Brusewitz, 1983; Anderson *et al.*, 1993). The disadvantage of pins with negative profile (or 'cut in') threads is that they develop a point of stress concentration at the end of the threaded portion. Although this can be protected to some extent by placing the junction within the medullary cavity, there is still a high incidence of fixation pin failure when such pins are used (Palmer & Aron, 1990). In addition, placement of a threaded Steinmann pin such that the end of the thread lies within the medullary canal leaves the pin tip protruding too far through the far cortex (because of the length of the threaded portion). For these reasons the use of threaded Steinmann pins, Ellis pins and Kirschner wires has largely been superseded by pins with positive profile (or 'rolled on') threads (for example, the Imex pins which have an end thread for half-pins and a mid-shaft thread for full pins). Commonly a mixture of positive profile threaded pins and non-threaded Steinmann pins are used to create an ESF.

The fixation pins are connected to one another using one of the following systems.

Connecting clamps: Traditionally a system of clamps and connecting bars have been used to stabilise the fixation pins. Various types based largely on the Kirschner–Ehmer apparatus have been marketed and are available in 'small', 'medium' and 'large' kits (see below). *Single clamps* are used to anchor each fixation pin to a connecting bar (Fig. 12.6). Occasionally, short connecting bars are used to connect the pins in the proximal and distal fragments and then these two are connected by a third using *double connecting clamps*.

Maynard clamps can be used in a similar fashion (supplied in the UK through Shorline Ltd, Cowbridge and Veterinary Instrumentation, Sheffield). These comprise two discs of metal with grooves of different sizes on each face (Fig. 12.7). One disc has a hole through its centre without threads through which the bolt 'glides' and the other possesses a central hole which is threaded. Each clamp is adaptable in terms of which of the grooves on each disc faces 'inwards' and this determines the size of connecting bar and fixation

pin to be used, with the latter not having to be the same throughout the frame. The fact that the clamp is assembled around each pin/bar junction can be advantageous, especially when more than two threaded pins are required (see below under 'Application of a unilateral, uniplanar (type I) ESF'). The clamps also allow some variation with respect to the planes in which the fixation pins lie, more so than with the traditional style of clamp. Although this system is less stable than the traditional Kirschner–Ehmer apparatus, it is far more versatile when constructing complex frames and its lack of strength can easily be overcome by adding more fixation pins and/or connecting bars.

Acrylic connections: Instead of using metal clamps and connecting bars, the fixation pins may be joined together using an acrylic material such as polymethylmethacrylate (e.g. Technovit, Kulzer; Palacos LV-30, Kirby-Warrick) or epoxy putty (Armstrong, 1991; Roe & Keo, 1997). The acrylic may be applied directly over the pin ends or else injected into some form of tubing which is first placed over the pin ends. With respect to strength of an acrylic bar, in comparison to stainless steel, it has been established that a bar of $\frac{3}{4}$–1″ diameter exceeds the strength of a $\frac{3}{16}$″ steel bar (as used for the medium-sized Kirschner apparatus – see below under 'Pin/bar sizes') (Willer *et al.*, 1991). The Acrylic Pin External Fixation (APEF) System has been developed by Innovative Animal Products and is marketed in the UK by Veterinary Instrumentation, Sheffield. It comes as a complete kit including fixation pins, clamps that are used for temporary fixation whilst the acrylic tubes are applied, corrugated tubing with stoppers, and packs of acrylic (Fig. 12.8). A variation on this theme is to use a metal connecting bar, which is temporarily fixed to the pins using wire, and then using acylic to form the individual pin/bar connections. This has been reported as advantageous in avoiding the cost of connecting clamps and also one problem which may be seen with acrylic bars, namely that of the patient chewing through it (McCartney, 1998).

The main advantage of an acrylic system is that a variety of sizes and orientations of pins can easily be accommodated due to the flexibility of the connecting bar which removes some of the constraints of using metal clamps and bars. This is particularly useful for awkwardly shaped bones such as the mandible. Although using such materials is less expensive in terms of initial outlay, the metal clamps and connecting bars are reusable. One disadvantage of acrylic systems is that it is far more difficult to make adjustments to the frame once the material has set and post-operative radiographs have been taken. The APEF system overcomes this by allowing temporary clamping of the fixation pins and then, after radiographs have been taken, the acrylic bars are added. However, once the cost of such a system is taken into account then the expense of fixation becomes comparable to using traditional clamps and bars.

Pin/bar sizes

The fixation pin size chosen depends not only on the size of the animal but also on the bone involved. As a general rule, the diameter of the pin should not exceed one-third the width of the bone into which it is being placed, otherwise stress-related fractures may occur. The size of connecting bar is determined by the size of fixation pin and the type of connecting clamp being utilised. A guide to the sizes of fixation pins used, along with the corresponding size of connecting clamp and bar, in the major long bones of animals of varying bodyweight are given in Table 12.3.

Classification of ESF frames/constructs

The types of frame and associated nomenclature are a frequent source of confusion. The frames have traditionally been referred to as types I, II and III according to on how many sides of the bone there were connecting bars. More recently the terms unilateral and bilateral have been used (Carmichael, 1991), depending on whether the fixation pins occupy up to or more than 90° of the bone's circumference, and uniplanar or biplanar, according to whether they lie in one or two planes. Fixation pins are termed half-pins when they pass through the skin once and full pins when they pass through it twice. However, all fixation pins should pass through two bone cortices.

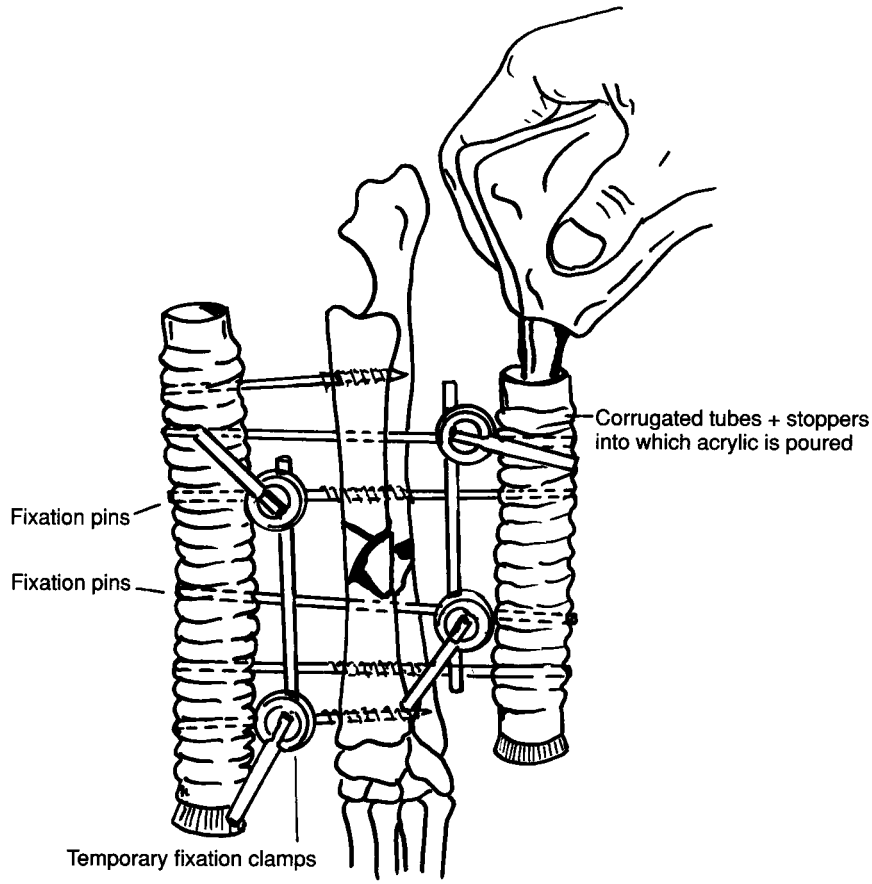


Fig. 12.8 An acrylic pin external fixator showing the components of such a system. Redrawn with permission from Innovative Animal Products.

Table 12.3 Fixation pin sizes, and corresponding connecting clamp/bar sizes, in relation to the animal's bodyweight (K-E clamps supplied through Veterinary Instrumentation and Maynard clamps supplied through Shorline or Veterinary Instrumentation).

Bodyweight	Fixation pin diameter	K-E clamps		Maynard clamps	
		Clamp size	Connecting bar diameter	Clamp size	Connecting bar diameter
<7 kg	5/64″ (2 mm)	Small	1/8″	11 mm	5/64″ (2 mm)
	5/64″ (2 mm)			13 mm	5/64″ or 7/64″
	3/32″			13 or 14 mm	7/64″ (tight)
7–20 kg	3/32″	Medium	3/16″	13 or 14 mm	7/64″ (tight)
	7/64″			13 mm	7/64″ or 1/8″
	7/64″			16 mm	1/8″
	1/8″			16 mm	1/8″ or 5/32″
20–50 kg	1/8″	Large	5/16″	16 mm	1/8″ or 5/32″
	9/64″			16 mm	5/32″ (tight)
	5/32″			16 mm	5/32″

Unilateral, uni- or biplanar (type I) fixators

Unilateral, uniplanar fixators use only half-pins and the simplest has only a single connecting bar (Fig. 12.9a). The adjustability of the frame may be increased by creating a type I frame on each fragment and then joining the two connecting bars together with a third, using double connecting clamps (Fig. 12.9b). However, this configuration is the least rigid as the double clamps act as weak points and such a frame would rarely be appropriate in fracture management. The unilateral, uniplanar fixator can be given increased rigidity by application of a second connecting bar (double bar type I).

A unilateral, biplanar construct comprises two unilateral frames placed at right angles to one another and joined together proximally and distally (Fig. 12.9c). With respect to axial compression, this configuration is stronger than a unilateral, uniplanar frame and weaker than a bilateral, uniplanar frame, but it is stronger than the latter with respect to rotational forces.

Bilateral, uniplanar (type II) fixators

Traditionally these have used only full pins (Fig. 12.9d). However, the placement of fixation pins in alignment, such that they can be attached to bars on both sides of the limb, may lead to technical difficulties (see step 7 under 'Application of a unilateral, uniplanar (type I) ESF' below). It is possible to modify the frame so that only two pins (usually the most proximal and distal) are full pins and half-pins are then added from one or both sides (Fig. 12.9e).

Bilateral, biplanar (type III) fixators

These comprise a combination of a bilateral, uniplanar frame with a unilateral, uniplanar frame placed at right angles to one another. The two component frames are joined together proximally and distally such that, end on, the appearance resembles a tent frame (Fig. 12.9f). This is the most rigid of the basic configurations. It is generally only required in open fractures with extensive loss of bone stock (e.g. high velocity ballistic injuries) and although such ESFs are used regularly in North America they are seldom required for fracture management in UK patients.

Ring fixators

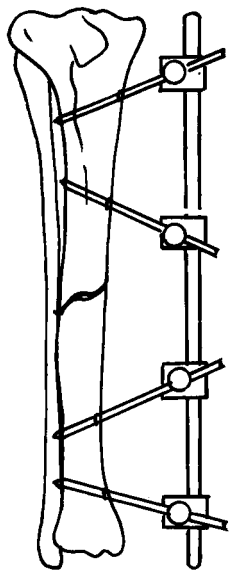
Ring fixators, such as the Ilizarov system, comprise circular elements which encompass the limb, are attached to fixation pins and are, in turn, connected to one another by parallel threaded bars (Fig. 12.9g). Although they can be used to stabilise fractures (Thommasini & Betts, 1991) they provide a rigidity comparable to a bilateral, biplanar pin fixator (Lewis *et al.*, 1998) and so are rarely an absolute necessity. They are more commonly used to correct growth deformities, especially where limb shortening is a problem, as they can be constructed in such a way as to allow distraction osteogenesis (see Chapter 34).

Application of a unilateral, uniplanar (type I) ESF (Fig. 12.10)

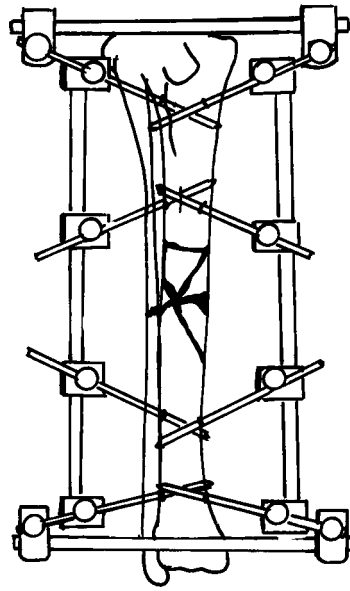
In order to create a four-pin, single bar type I (unilateral, uniplanar) fixator using a connecting bar and clamps, the following steps are taken.

(1) The limb is prepared aseptically as if for any form of open reduction and fixation. Fracture reduction (closed or open) is achieved prior to the placement of fixation pins, otherwise skin tension around the pins will develop subsequently.

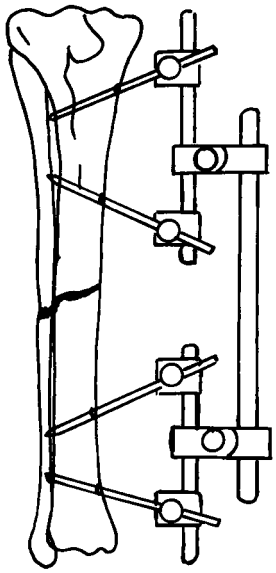
(2) With respect to which side of the bone a unilateral, uniplanar (type I) fixator is applied, several factors need to be considered; the location of the body wall, for example, restricts the aspects of the humerus and femur which can be utilised. However, the most important consideration is that of the soft tissues. The term 'safe corridor' is used to define the passage of a fixation pin from the skin to the bone without penetrating either neurovascular bundles or musculotendinous units (Marti & Miller, 1994a,b). Fortunately, the former tend not to be penetrated by pins but rather are pushed aside, although the use of drill bits or threaded pins might lead to them becoming damaged. Fixation of muscle bellies to the underlying bone may cause the formation of permanent adhesions with a detrimental effect on limb function in the future. In addition, premature loosening of the pin may occur for two reasons. Firstly, muscular activity will tend to rock the pin and, secondly, soft tissue movement around the pin will cause necrosis and a pin tract discharge which may lead to a pin tract infection.



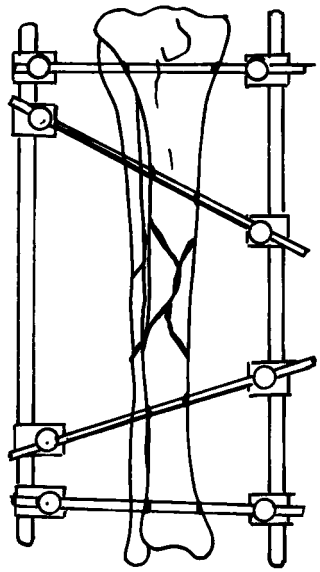
(a) Single bar, unilateral, uniplanar (type I) fixator.



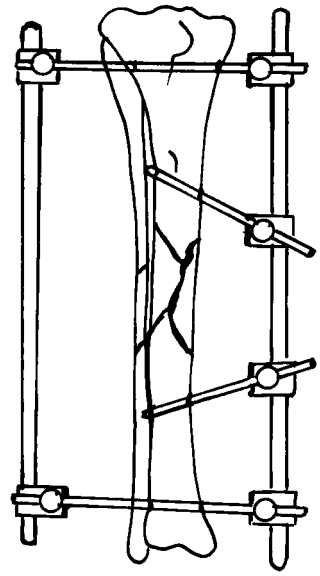
(c) Unilateral, biplanar (quadrilateral, type I) fixator.



(b) Double clamp, unilateral, uniplanar (type I) fixator.

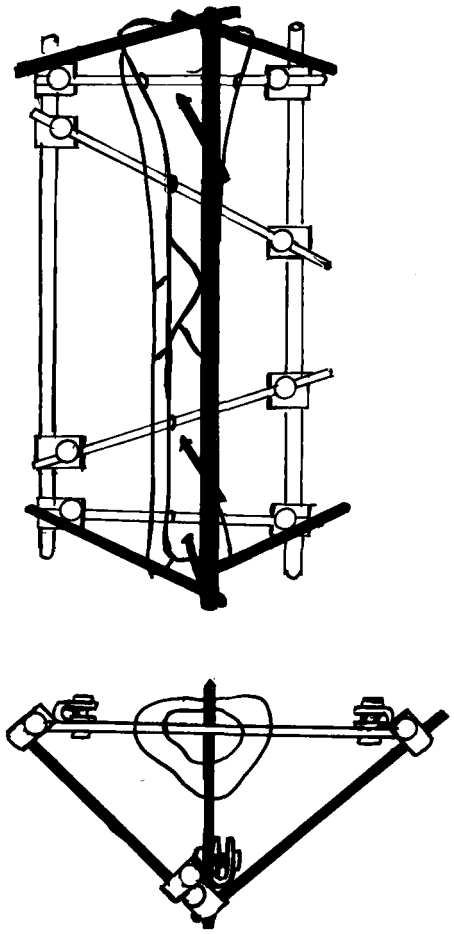


(d) Bilateral, uniplanar (type II) fixator (full pins only).

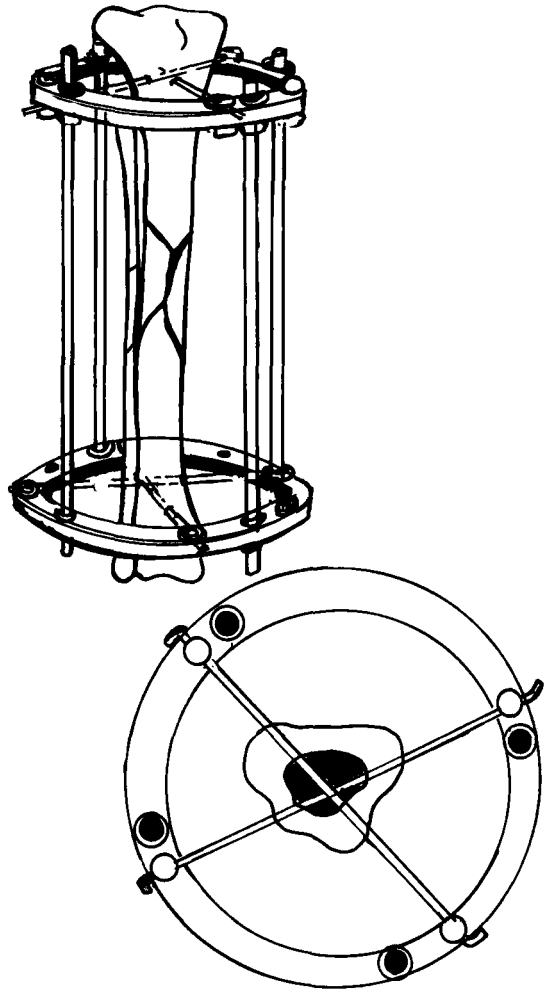


(e) Modified bilateral, uniplanar (type II) fixator (full and half-pins used).

Fig. 12.9 Classification of external skeletal fixator (ESF) frames/constructs.



(f) Bilateral, biplanar (type III) fixator ('tent frame').



(g) Ring fixator.

Fig. 12.9 *Contd.*

Bones such as the femur and humerus have no real safe corridors because they are surrounded by muscles. The use of external fixators is, therefore, generally restricted to fractures distal to the elbow or stifle. When a unilateral, uniplanar (type I) fixator is applied to the femur, an open approach may be used to allow visualisation of the fascial planes while the pins are placed. A guide to which side of a bone a unilateral, uniplanar (type I) fixator is applied is as follows:

- Humerus lateral
- Radius medial
- Femur lateral
- Tibia medial

(3) The most proximal and distal pins are placed as far apart as possible with avoidance to the joint spaces and also the physes in immature animals (Fig. 12.10a). They pass through stab incisions in the skin separate to any wound used to allow fracture reduction and enter the bone at about 45–65° to its longitudinal axis. Angulation of the pins is mainly to assist frame stability and resistance to 'pull-out' when plain pins are used. If threaded pins are utilised then this angulation is far less

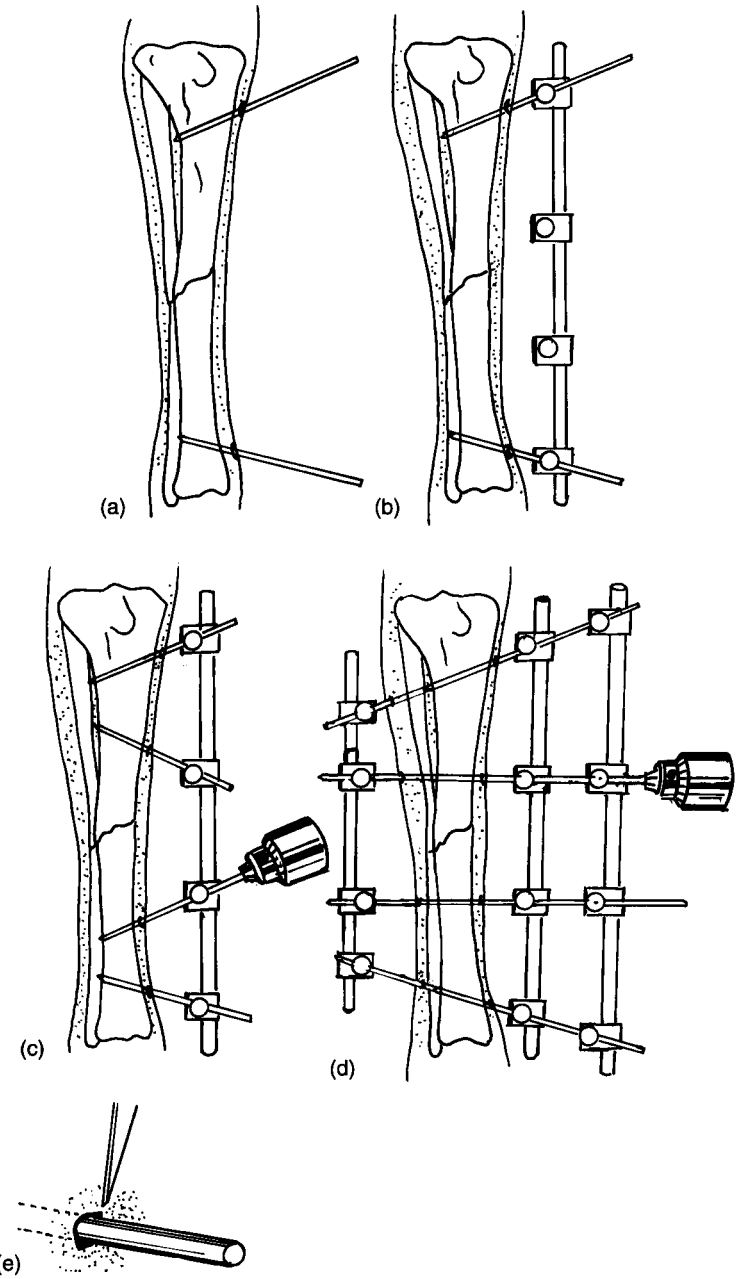


Fig. 12.10 Application of a four-pin, unilateral, uniplanar (type I) fixator (see text for additional information).
(a) The most proximal and distal pins are placed as far apart as possible through stab incisions in the skin.
(b) The connecting bar is attached to these pins leaving a gap of 5–10 mm between the skin and the clamps. With some systems the clamps for the remaining pins must be added to the bar first, whereas in others the clamps are added as the pins are placed.
(c) The remaining pins are placed at an angle to the others in the fragment to help prevent 'pull-out'.
(d) To place the remaining pins as full pins a second bar with guide clamps may be added temporarily so that the pins meet the connecting bar on the far side of the limb (necessary only for the standard bilateral, uniplanar fixator).
(e) After all the pins have been placed the skin–clamp distance is checked and also any tension in the skin around the pins is released using a no. 11 scalpel blade.

important and the first two pins are often placed almost parallel.

It has been recommended that pins are placed using a hand chuck as high-speed drills will cause thermonecrosis of surrounding bone and premature pin loosening. However, the use of hand chucks can be associated with 'wobbling' during pin placement, especially when several pins have been placed through hard cortical bone. This will result in too large a hole being produced which may cause premature loosening of the pin. Predrilling with a slightly smaller drill bit followed by hand chuck placement of the pin avoids these problems. However, there is a greater tendency for the surrounding soft tissues to become entangled with a drill compared to a pin, even when drill guides are used, and it may prove difficult to locate the predrilled hole. Therefore, although this technique is technically superior, it may be reserved for fractures where prolonged stability is required. Where good progress with respect to healing is expected (i.e. within 6–8 weeks) it is generally satisfactory to place the fixation pins using low-speed drills (Egger *et al.*, 1986). This avoids the wobble of the hand chucks and, if the speed is kept below 150 rpm, thermal necrosis is minimal.

(4) Having placed the first two pins on the appropriate side of the bone, a suitable connecting bar is chosen. Onto this are placed a number of single clamps corresponding to the number of fixation pins to be used. If the fixator alone is used to stabilise a fracture then a minimum of two pins per fragment is required. Increasing the number of pins to three per fragment results in a 66% increase in axial stiffness and a fourth pin per fragment gives an additional 33% increase (Egger, 1991). Little effect is seen by using more than four pins.

If Maynard clamps or the Securos system (supplied by Veterinary Instrumentation, Sheffield) are used then it is not necessary to preplace the intermediate connecting clamps as they can be added later.

(5) The two pins are then clamped to the connecting bar (Fig. 12.10b). The closer the bar is to the bone the more rigid is the frame. However, a gap should be left between the clamps and the skin to allow for some swelling and to avoid ulceration caused by pressure necrosis. In general, a gap of 5–10 mm is sufficient, depending on the size of the patient.

(6) The remaining pins are placed through stab incisions using the clamps as guides (Fig. 12.10c). There should be an angle of 35–55° between the most proximal and distal pins in each fragment to prevent the fragment sliding along the pins and to help maintain stability should slight loosening of the pins occur. Again the need for this angulation is aimed at reducing the likelihood of 'pull-out' when plain pins are used. Far less angulation is necessary if threaded pins are used.

If Maynard clamps or the Securos system are used then the pins are placed using the connecting bar as the guide and then each clamp is assembled around the pin/bar junction. These are particularly helpful in allowing threaded pins to be used throughout the frame. When traditional clamps are used, pins with positive profile threads will only pass through the clamp if a smaller pin diameter is chosen.

(7) If pins are being placed to form a bilateral, uniplanar (type II) configuration, then it is necessary to use either a second, temporary, bar with clamps so that the pins pass through two guiding clamps (Fig. 12.10d), or else a jig. This will ensure that they are all aligned for connection to a bar on the other side of the limb. Because of this complexity it is most common to use only two full pins to create a modified type II frame with half-pins added from one or both connecting bars.

(8) Having tightened all the clamps, the fracture site is assessed for stability and normal joint movement/alignment. It is ensured that a sufficient gap exists between the skin and each clamp and that there is no puckering of the skin around any of the pins. Any such tension should be released by incising the affected skin and, if necessary, by placing a single suture on the opposite side to close the enlarged stab wound (Fig. 12.10e). If tension is not released, necrosis will occur and the resulting discharge will increase the likelihood of a pin tract infection.

(9) The pins may then be cut to a manageable size. The patient is easier to position if this is carried out before postoperative radiography, although any subsequent adjustments required as

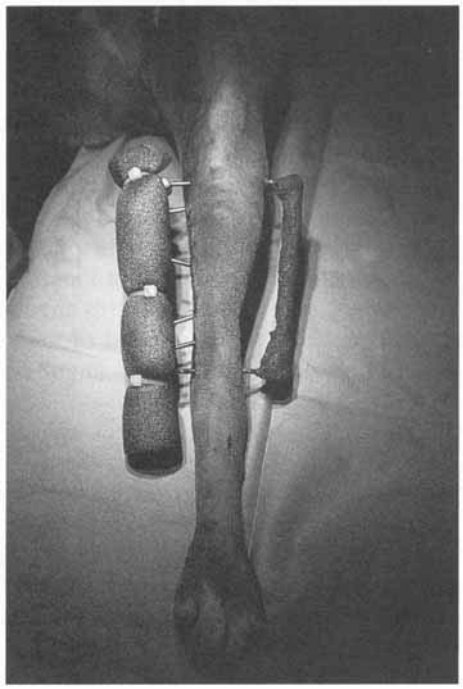


Fig. 12.11 A slide showing a bilateral, uniplanar (type II) fixator applied to a Boxer's tibia. One connecting bar has been covered with Soffban (Smith & Nephew) and Vetrap (3M) whilst the other has been covered with pipe insulating foam.

a result of the radiological finding might be limited by a lack of spare pin length.

(10) The clamps and pin ends are covered with a dressing (Vetrap, 3M; or foam tubing and ties as used for pipe insulation, see Fig. 12.11) to protect the patient's contralateral limb as well as the owners and their furniture. In cases with severely comminuted or open fractures, where considerable swelling might be anticipated, it may be necessary to place the limb and external fixator in a Robert Jones bandage and then it is advisable to include the foot in any such bandage to avoid it becoming swollen.

Postoperative care of ESFs

Postoperative care of the fixator is minimal. A light discharge from the pin tracts is not uncommon and will generally form a crust thereby sealing the pin/skin interface. In the event of a more copious discharge it may be necessary to bathe the skin at regular intervals. The pin/skin junction should be avoided because bathing this area will tend to create a soup of commensal bacteria and increase the likelihood of pin tract infection.

It is important for a veterinary surgeon or nurse to check the frame on a weekly basis to ensure that the clamps and/or pins have not loosened and that pin tract infection has not occurred. As healing progresses and the animal begins to use the limb more, one or more pins will sometimes loosen slightly and this is often accompanied by an increase in pin tract discharge and/or lameness. If this produces clinical problems it may prove necessary to remove or replace a loose pin. Radiography of the fracture site should be repeated after 4–8 weeks (or sooner if problems are suspected).

Fixator removal

It is generally found that the clinical union (i.e. healing sufficient to allow the bone to bear weight without the need for 'bone splinting') precedes radiographic union, and it is the former that is more important when considering frame removal. An important feature of external fixators is that the strength of the healing fracture can be assessed without removing the implants. By loosening the clamps and removing the connecting bar(s), the site can be tested for its strength against angular and torsional forces. If instability persists then the connecting bar(s) may be replaced.

A second, important feature of the external fixator is that if healing is progressing well, but is not yet complete, then a process of 'staging down' or 'disassembly' of the fixator may be utilised. Its aim is to provide less external support for the bone, thereby increasing the bone's share of the weight-bearing load. The avoidance of 'stress protection' of the bone by the implants (which may be seen with all methods of fracture fixation to a greater or lesser extent) is thought to increase the rate of healing. There are limits to the staging-down process, in that the healing callus must be strong enough to withstand the new forces to which it is being exposed. The methods of staging down or disassembling an external fixator include:

- Removing a connecting bar
- Moving a connecting bar further away from the bone
- Removing some of the fixation pins

It is generally recommended that a fixator is removed once the fracture site is clinically stable and preferably once there is a continuity of three out of four cortices visible on two orthogonal radiographs. It is possible to remove fixators with the patient sedated but anaesthesia may be preferable as it makes radiography, clinical assessment of the degree of healing and pin removal somewhat easier in most patients.

Indications for ESFs

Situations where the use of ESFs may be considered can be listed as:

- Simple diaphyseal fractures of the radius/ulna or tibia/fibula
- Comminuted diaphyseal fractures of the radius/ulna or tibia/fibula
- Auxiliary fixation (most commonly combined with intramedullary pins) for diaphyseal fractures of the tibia, femur and humerus
- Mandibular fractures
- Diaphyseal fractures in young animals
- Open or infected fractures
- Comminuted diaphyseal fractures that encroach on the metaphysis, creating limited bone stock

Simple diaphyseal fractures

Simple transverse or short oblique fractures of the radius/ulna or tibia/fibula may be reduced in a closed or limited open fashion and then stabilised by application of a unilateral, uniplanar (type I) fixator (Fig. 12.9a). Such an ESF provides a more positive means of stabilising the fracture than does a cast and also allows better limb/joint function during the healing period. It also provides a less invasive means of stabilising such a fracture compared to a plate, though the latter may be more appropriate where healing might be slow or problematic (e.g. in an older animal or in a toy breed).

Comminuted diaphyseal fractures

Where the degree of comminution makes reconstruction of the bone difficult, or impossible, a fixator may be used to maintain limb length and correct joint alignment while the fracture heals by bridging osteosynthesis. Large fragments might be stabilised with the limited application of implants such as lag screws. Alternatively, the whole procedure may be carried out in a closed fashion with correct alignment being assessed externally. Although the visual assessment of limb/joint orientation is more important than the radiographic appearance, the more accurate the reduction the quicker the healing is likely to be.

When used in this fashion a bilateral, uniplanar (type II) fixator is likely to be required (Fig. 12.9e) and this limits its application to fractures of the radius/ulna or tibia/fibula. However, successful use of unilateral ESFs in the management of comminuted humeral and femoral fractures has been reported (Langley-Hobbs *et al.*, 1996b, 1997).

Auxiliary fixation

Although simple fractures of the femur and humerus could be managed using a unilateral, uniplanar ESF, the lack of safe corridors for fixation pin placement makes this less than optimal. However, use of an intramedullary pin to stabilise such fractures will often not control rotational forces and it is in this situation where the ESF may be useful. A unilateral, uniplanar fixator will control such rotational forces and so provides excellent stability when combined with an intramedullary pin (McPherron *et al.*, 1992) and, although the resistance to such forces increases with the number of fixation pins, a two-pin fixator will usually suffice (Foland *et al.*, 1991) (see later in Fig. 12.14). The overall stiffness of such a construct applied to the humerus or femur may be increased by 'tying in' the intramedullary pin to the external fixator (Aron *et al.*, 1991) (Fig. 12.12). The combination of an intramedullary pin and fixator is also applicable to the tibia. In some cases with fractures of the radius/ulna, a four-pin fixator applied to the radius may be combined with an intramedullary pin placed in the ulna.

The use of such an ESF in conjunction with an intramedullary pin could also be considered for

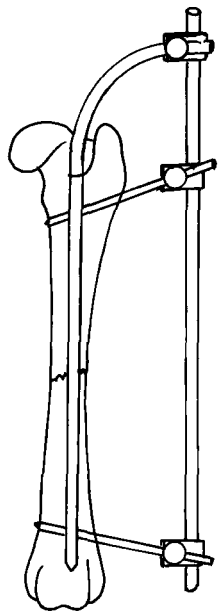


Fig. 12.12 A 'tied-in' configuration for a unilateral, uniplanar (type I) fixator used in combination with an intramedullary pin to stabilise a femoral fracture.

managing comminuted fractures of the humerus or femur in smaller patients (Langley-Hobbs *et al.*, 1996b, 1997), although the authors prefer to use buttress plates in such situations. Guerin *et al.* (1998) reported the use of modified type I ESFs in conjunction with intramedullary pins to treat a series of comminuted supracondylar humeral fractures.

Mandibular fractures

There are several reasons why fixators are useful in the management of mandibular (or unstable maxillary) fractures: the bone shape may preclude the use of certain implants; the majority of these fractures are open; and the most important factor to be borne in mind when treating them is to regain good dental occlusion. Fixation pins may be placed into the fragments so as to avoid the tooth roots and alveolar nerve. The pin size can be varied according to the size of the fragment and the pins may be stabilised using a steel rod (simple fractures) or an acrylic bar/bent rod with acrylic connections (more complex or bilateral fractures). For more details on the management of mandibular fractures the reader is referred to Chapter 16.

Young animals

Young patients tend to heal more rapidly (see Table 1.1) and so a fracture does not require prolonged bone splinting. They also have a tendency to produce excessive periosteal reaction where iatrogenic damage has resulted from placement of an implant, such as a plate. In addition, because they are young it is more likely that internal implants would need to be removed at a later date. Therefore, in fractures of the radius/ulna or tibia/fibula where the options decided upon, for reasons of fracture configuration, etc., are either bone plate or external fixator then the fact that an animal is less than 1–2 years of age may push the balance in favour of the ESF.

Open or infected fractures

Fixators provide a valuable means of stabilising open or infected fractures for three main reasons. First, the fixation pins are placed away from the infected site and so complications such as implant loosening are less likely to be encountered. Second, the fracture site need not be disturbed at all, avoiding the risk of increased soft tissue damage, decreased vascularity and the spread of contamination. Third, wound management is simplified as dressings may be changed daily without the need for the limb to be incorporated within a strong supporting bandage. External fixators may be used either as the sole means of stabilising an infected fracture during healing or else as a temporary measure whilst the wound and infection are treated.

Generally speaking, bilateral frames are required to provide sufficient stability for the duration of fracture healing. Although this tends to restrict their use, for this purpose, to fractures of the radius/ulna or tibia/fibula, open fractures are more commonly encountered in these bones compared to the humerus and femur, particularly in the UK where gunshot injuries are less common. For more details on the management of open or infected fractures the reader is referred to Chapters 13 and 14.

Limited bone stock

In cases where a comminuted fracture ends very close to a joint, the problem of low bone stock may be overcome by either placing two pins at right angles to one another (to gain two-point fixation in the fragment) or else creating a transarticular fixator which gains purchase in the bone distal or proximal to the one that is fractured.

ESFs are also used in situations other than fractures and some of these are discussed elsewhere in this book, including:

- To stabilise corrective osteotomies used in the treatment of growth deformities (see Chapter 34)
- To stabilise injured carpal or hock joints, particularly those in which there is a shearing injury (see Chapters 35 and 44)
- To stabilise a joint, particularly a hock or carpus, during arthrodesis (see Chapters 35 and 44)
- To stabilise fractures in exotic species, especially avian species

Advantages of ESFs

These may be listed as:

- Minimal instrumentation required
- Minimal soft tissue damage during application
- Very adaptable
- Allow staged load transfer to the healing bone
- Allow access to open wounds
- Allow implants at the fracture site to be avoided
- Relatively inexpensive

In terms of the amount of *instrumentation* required and the *soft tissue trauma* caused to an already compromised site during application, the ESF is similar to the intramedullary pin and both of these should be far less traumatic than the application of a bone plate or interlocking nail. This has a direct effect on the biology of the healing tissues. ESFs are *very adaptable* in terms of accommodating awkwardly shaped bones (e.g. the mandible) or in situations where there is limited bone stock. The frame can be built up to match the stability required by the specific fracture concerned and then, if necessary, 'staged down' again to allow progressive *load transfer to the healing bone.* The application of an ESF allows *good access to wounds* and regular dressing changes are not complicated by allowing movement at the fracture site compared to when a splint or cast is used. Furthermore, because the dressing is only for wound management rather than providing stability, the cost of replacing the dressing is reduced. In the case of open fractures, the use of an ESF not only minimises the amount of surgical trauma to already compromised tissues and allows access to the wound, but it also *allows the avoidance of leaving foreign bodies* (implants) in a contaminated site. It is often said that the use of ESFs is *more financially viable* than, say, using plates. Although there can be a saving in the cost of implants, if positive profile pins and/or the APEF system (where none of the components are reusable) are used then this cost may approach that of a plate. In addition, the use of an ESF arguably requires more postoperative attention and will certainly require removal. It is the authors' experience that the final cost of using an ESF to manage a complex fracture is very similar to that of using a plate (unless the plate has, ultimately, to be removed) and the choice of which system to use should be generally based on factors other than finance.

Disadvantages of ESFs

These may be listed as:

- Soft tissue complications
- Limited application to the proximal limb
- Application technique requires practice
- Owner acceptance

Unlike a bone plate, which sits between the bone and soft tissues, the fixation pins of an ESF pass through the soft tissues. This may lead to *soft tissue complications* such as adhesions, pin tract discharge, premature loosening of the pin and pain leading to reduced weight-bearing on the limb. As a result of this the morbidity, with respect to limb function during healing, may be greater with ESFs than with internal fixation. These complications are more likely if the pin passes through a non-safe corridor and this makes application of an ESF to the *proximal limb difficult.* Understanding the principles and *techniques of application* is mandatory for the successful use of these

systems and the fact that they appear simple belies the fact that they require practice and experience to master. Indeed, their perception as a panacea for fracture management has led to misuse of the systems. The problem of *owner acceptance* is becoming less as many people have seen such frames on other people or animals either in real life or on the television.

Intramedullary fixation

Intramedullary fixation is a simple method of fracture repair which is widely used in veterinary orthopaedics. Unfortunately, it is also a technique which is often misused.

The intramedullary devices include:

- Steinmann pin
- Rush pin
- Kirschner wire
- Kuntscher nail
- Interlocking nail

The Steinmann pin is used most commonly. The main indication for such a pin, used alone, is in the treatment of stable fractures, i.e. transverse or blunt oblique fractures of the middle third of long bones. The pin lies within the medullary cavity and is, therefore, able to resist bending in any direction (Fig. 12.13). Fracture stability is related to:

- The tightness of pin fit within the medullary cavity, which resists rotation
- Interlocking of the fragments, which resists rotation
- Muscle tone, which creates functional compression

The medullary cavity of long bones in the cat tends to be of a uniform diameter so a tight pin fit can be achieved. In the dog, however, the medullary cavity varies in diameter through a bone's length and so it is usually only possible to achieve three-point fixation: at the point of insertion, at the fracture site or narrowest point of the medullary cavity, and by impacting the distal end of the pin into the cancellous bone of the metaphysis and epiphysis.

A round intramedullary pin resists bending in all directions but has little resistance to shortening or rotation at the fracture site. Rotation or torsion is most likely to occur when a loose fitting pin is used for fixation of fragments that do not interlock adequately and this will often lead to non-union.

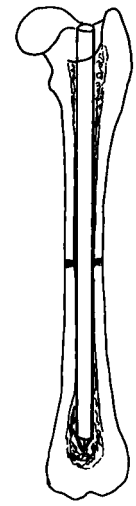


Fig. 12.13 Intramedullary pin for femoral diaphyseal fracture.

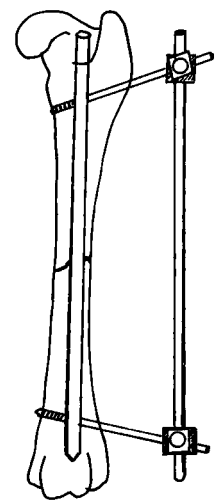


Fig. 12.14 Combination of intramedullary pin and external fixator to immobilise a femoral diaphyseal fracture.

Stability and resistance to rotation can be improved in one of several ways:

(1) The intramedullary pin can be used in combination with an external fixator (Fig. 12.14).

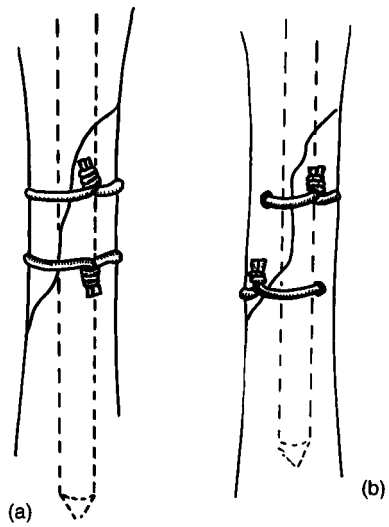


Fig. 12.15 (a) Cerclage wires and (b) hemicerclage wires used in combination with an intramedullary pin to minimise rotation.

(2) In oblique fractures, stability can be improved with cerclage (Fig. 12.15a) or hemicerclage wiring (Fig. 12.15b).
(3) Instead of a single intramedullary pin, two or three smaller pins (multiple pins) can be placed within the medullary cavity (Fig. 12.16) so as to increase the number of points of fixation.
(4) A Kuntscher nail which is V-shaped or clover leaf in cross-section can be used instead of an intramedullary pin. However, this method is seldom used in small animal orthopaedics and has largely been superseded by multiple pinning or, more recently, interlocking nailing.

Although an intramedullary pin disturbs endosteal callus formation it causes little interference with the healing of the cortex and periosteum. Size of the callus varies with the stability achieved. If stability is good there will be minimal callus formation but if it is poor, due to a loose fitting pin, there will be extensive periosteal callus formation. Young animals have a high osteogenic potential, fractures heal very rapidly and pins are, therefore, often very appropriate for use in such cases.

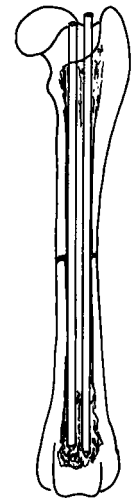


Fig. 12.16 Multiple pins used for fracture fixation.

Intramedullary pinning *should be avoided for treating comminuted fractures in dogs*. In this type of fracture the pin provides no longitudinal support or resistance to shortening forces and collapse, rotation and non-union are to be expected. Cats are an exception. Most diaphyseal fractures in the cat, even if they are severely comminuted, can be successfully managed using a Steinmann pin in combination with cerclage wire. Having said that, many cats with severely comminuted diaphyseal fractures will regain limb function more rapidly and more completely using alternative techniques such as buttress plating. In the dog, however, as a general rule, the intramedullary pin should be reserved for stable fractures of the middle third of the femoral or humeral diaphysis. However, there are some exceptions to this rule, particularly when dealing with fractures in immature animals.

Steinmann pin (Veterinary Instrumentation)

These are smooth pins, 30cm in length and varying in diameter from 1.6mm to 5.6mm. Each end has a trochar tip designed to cut into bone. This type of pin does have a tendency to migrate and pins of the same dimensions are available which have a fine thread cut into one trochar end. The thread is designed to give a better purchase in cancellous bone and minimise migration.

However, this claim has been questioned by *in vitro* studies which showed no difference in the holding strength of the two designs (Howard & Brusewitz, 1983). In addition, a threaded pin has a weak point at the thread–non-thread junction because the thread has a negative profile. If used, therefore, it must be ensured that this point of the pin is nowhere near the fracture site, otherwise pin failure should be anticipated.

Equipment required for the insertion of the Steinmann pin is as follows:

- Introduction of pin
  - Jacobs chuck
  - variable speed orthopaedic drill
- Cutting the pin
  - pin cutters
  - hacksaw

The technical aspects of choosing the correct size of intramedullary pin and its mode of insertion are described in the chapters on management of humeral, femoral and tibial fractures (Chapters 32, 41 and 43, respectively). The main disadvantage of the intramedullary pin is that rotational stability is poor. However, in appropriate cases this is outweighed by advantages such as:

- Ease and speed of insertion
- Minimal exposure (possibly none) of the fracture site is required
- Requires less technical expertise and equipment than other methods of internal fixation
- Low cost
- The intramedullary pin affords less stress protection than a plate and gives a stronger repair of the fracture site in the early stages of healing (Braden *et al.*, 1973)
- A pin which crosses a physeal plate causes minimal disturbance in longitudinal bone growth when compared with other methods of fixation

Pins can be left *in situ* after fracture healing is complete provided they do not protude from the end of the bone and cause soft tissue irritation (recognised as lameness). In immature animals, rapid longitudinal growth of bone can seal a pin within the medullary cavity. However, in mature dogs there is a tendency for the pin to eventually migrate proximally (ride up) and cause soft tissue damage. In this event the pin is removed provided fracture healing is complete. Some surgeons prefer to remove all intramedullary pins and use a longer pin which is cut off just below the skin surface to allow for easy removal. If an intramedullary pin is combined with an ESF (usually in humeral or femoral fractures) then the proximal end may be bent over and 'tied in' to the frame. In such cases the pin is removed either at the same time as the fixator pins or a few weeks later if the construct is 'staged down' (see above under 'External skeletal fixators').

Arthrodesis wires and Kirschner wires

Arthrodesis wires look like small intramedullary pins. They are 127 mm (5″) in length and have a trochar point at each end. They are availiable in four diameters ranging from 0.9 mm to 2 mm. They are used for fixation of small bone fragments. Kirschner wires are available in the same sizes as arthrodesis wires and are used for the same purposes. One tip of the Kirschner wire has a bayonet point, the other end has a trochar point.

Indications for the use of arthodesis wires and Kirschner wires are:

- As crossed wires for the repair of physeal fractures
- To hold small fragments *in situ* during fracture healing
- As intramedullary pins to stabilise fractures in small bones such as the metacarpi
- To achieve temporary fixation whilst other implants are applied
- In combination with a lag screw to give a second point of fixation and minimise rotation of a fragment, for example in the repair of a lateral humeral condylar fracture
- To maintain alignment in avulsion fractures before application of a tension band wire

The advantages of Kirschner wires and arthrodesis wires are:

- Low cost
- Ease of insertion with minimal exposure
- No effect on physeal growth

The wires do have the disadvantage that they often have to be removed because of their tendency to migrate or because sharp ends of the wires cause soft tissue irritation.

Rush pin

The design of the Rush pin is illustrated in Fig. 12.17. The pin has a pointed 'sledge runner' tip for ease of insertion whilst the other end is hooked to ensure good fixation, reduce the likelihood of migration and to simplify removal. The pins are available in various sizes ranging in diameter from $\frac{5}{32}$ to $\frac{1}{4}$" and lengths from 1 to 7".

The Rush pin immobilises the fracture by its spring-like action which results in three-point pressure within the medullary cavity (Fig. 12.17d). This method of fixation is most commonly used for supracondylar fractures of the femur and humerus.

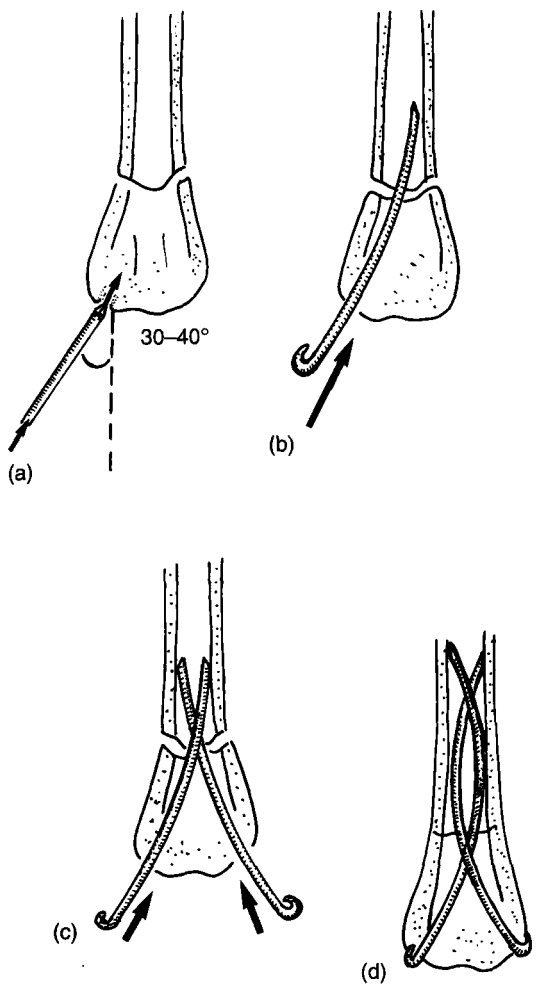


Fig. 12.17 Rush pins used for fixation of a supracondylar fracture of the femur. (a) Correct angle for insertion of Rush pin, (b) first pin introduced and used to maintain fracture reduction, (c) second Rush pin introduced and (d) pins hammered alternately until each is seated and the hook head grips the cortex.

The use of two Rush pins for fixation of a supracondylar fracture of the femur (Fig. 12.17)

The fracture is reduced and an awl-reamer or Steinmann pin is used to penetrate the cortex of the distal fragment to allow insertion of the Rush pin. The angle of insertion should be 30–40° to the long axis of the bone. The second Rush pin is introduced. Once the fracture is correctly held in alignment the pins are hammered alternately until each is seated and the hook head grips the cortex.

Rush pins, like Kirschner wires, have no effect on physeal growth. However, the technique for insertion of Rush pins is not easy and takes practice to master. For this reason, Kirschner or arthrodesis wires are often used in preference to Rush pins for stabilising fractures.

The interlocking nail (IN)

The interlocking nail evolved as a modification of the Kuntsher nail. It is basically an intramedullary pin secured in position by proximal and distal transfixing screws which engage the bone to the nail, thus providing axial and torsional stability (Fig. 12.18). The technique is particularly useful in comminuted long bone fractures. The interlocking nail is the preferred method of fixation for most diaphyseal fractures of the femur and tibia in humans. Clinical application of the method in dogs for the treatment of diaphyseal fractures of the humerus (Durall *et al.*, 1994) and fractures of the femur (Durall & Diaz, 1996) has given good results.

The structural properties of interlocking nails in canine femora have been described (Dueland *et al.*, 1996) using a system designed specifically for veterinary surgery (The Interlocking Nail [IN] System, Innovative Animal Products, supplied in the UK through Veterinary Instrumentation, Sheffield).

The sizes of interlocking nails for use in small animals include:

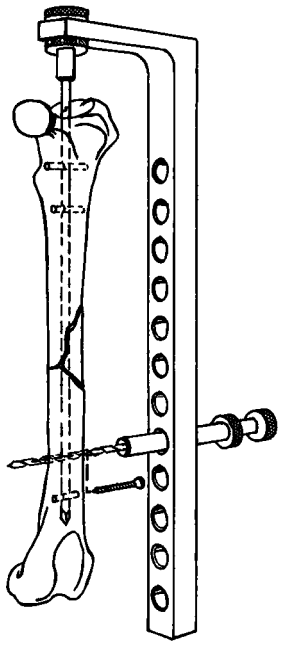


Fig. 12.18 The Interlocking Nail, IN System (Innovative Animal Products).

- 4.7mm diameter nail for use with 2.0mm cortex screws
- 6mm diameter nail for use with 3.5mm cortex screws
- 8mm diameter nail for use with 4.5mm cortex screws

The advantages of interlocking nails include:

- They resist axial shortening, rotational and angular forces better than standard intramedullary pins and so have a wider role in fracture fixation, including the bridging of severely comminuted diaphyseal fractures.
- They are centred within the bone and therefore are less likely to fail than an eccentrically positioned plate of similar size.

The disadvantages of interlocking nails include:

- They require special instrumentation to allow placement of the nail and the interlocking screws.
- They require an exposure that is similar to that for plate fixation.
- Their use is generally limited to fractures within the middle third of the diaphysis to ensure adequate room for interlocking screw placement proximally and distally.

This technique will no doubt find its niche in small animal orthopaedics but the limitations on when an interlocking nail system may be used is more restricted when compared to plating systems. As a result, the expense of adding an interlocking nail system to an already comprehensive armoury of pins, plates and ESFs is not a necessity.

Orthopaedic wire

Orthopaedic wire is made from monofilament stainless steel and is available in the following diameters:

- 18 gauge – 1.2mm
- 20 gauge – 1.0mm
- 22 gauge – 0.8mm
- 24 gauge – 0.6mm

Orthopaedic wire is monofilament and should not be confused with suture wire which is braided. *The latter is not suitable for fixation of bone fragments.*

Bone sutures

Orthopaedic wire can be used as the sole method of fixation, particularly in fractures of the mandible and skull. Simple interrupted sutures are used to retain the fragments in place. A small diameter drill bit (1.5 or 2mm) is used to drill holes through the bone fragments. The wire is passed through the holes and twisted tight.

Cerclage wire

Orthopaedic wire is frequently used in combination with intramedullary pins for fracture fixation, either to retain fragments in alignment or to provide rotational stability. If an intramedullary pin is used as the sole method of fixation for an oblique, midshaft fracture, the fragments tend to override and there will be a degree of rotation at the fracture site. A simple way of overcoming this problem is to supplement the pin with a 360° cerclage wire (Fig. 12.19). A tight cerclage wire will

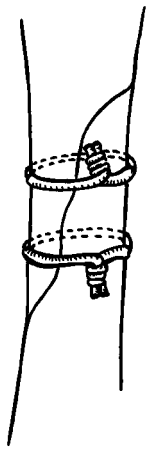


Fig. 12.19 Two cerclage wires used to stabilise an oblique fracture.

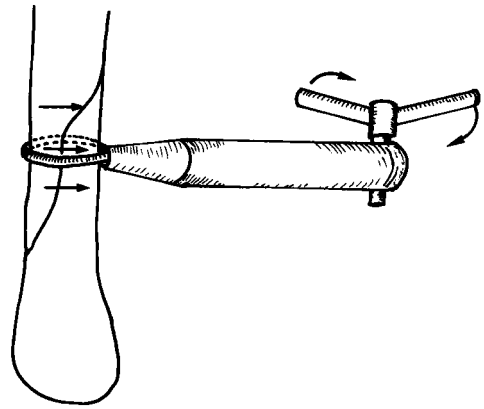


Fig. 12.20 AO wire tightener.

compress the fracture surfaces, creating friction and thus resistance to shortening or rotation.

Although the 360° cerclage wire is a useful method of fixation, it is a method which is frequently abused. Common faults are:

- Use of wire that is too thin
- Use of suture wire or other inappropriate suture materials instead of orthopaedic wire
- Failure to tighten the wire adequately
- Using insufficient numbers of cerclage wires (a single cerclage wire is particularly at risk of loosening)

These faults all result in a loose cerclage wire leading to bone lysis and the potential for non-union. Historically, the use of cerclage wires has caused some controversy. The method was condemned by some (Newton & Hohn, 1974; Vaughan, 1975) as it was said to cause non-union by interference with the periosteal blood supply. Others (Withrow & Holmberg, 1977) favoured cerclage and attributed failures of the method to poor case selection and poor technical application rather than interference with blood supply.

Normal bone receives blood from a variety of sources, including the nutrient artery, the metaphyseal vessels and through fascial attachments to the periosteum. If the blood supply in cortical bone was longitudinal to the long axis, then this would be 'strangled' by a cerclage wire. However, this is not the case (see Chapter 1). Vascular supply within cortical bone is perpendicular, rather than longitudinal, to the long axis and it has been shown (Cohen & Harris, 1958) that the longitudinal supply is limited to 1 or 2 mm only. Consequently, when two cerclage wires are used to stabilise an oblique fracture (Fig. 12.19) the segment of bone between the wires does not become necrotic. It is vascularised initially, and in a centripetal fashion, by vessels arising from the surrounding soft tissues and the periosteum. As healing progresses the normal centrifugal flow within the Haversian systems is restored.

The proper application of cerclage wires should result in compression of the fracture and primary bone union will often occur. Conversely, a loose cerclage wire results in resorption of bone or lysis under the wire and may lead to non-union of the fracture. The potential for bone necrosis may be exacerbated by excessive periosteal stripping and poor standards of asepsis.

The 360° cerclage wires should be reserved for oblique or spiral fractures. It is important to use wire of the correct thickness:

- For animals over 20 kg bodyweight use 18 gauge (1.2 mm diameter) wire
- For animals under 20 kg bodyweight use 20 gauge (1 mm diameter) wire

The cerclage wire should be applied tightly and specific wire tighteners are available for this purpose (Fig. 12.20). The wire is tightened either by twisting the free ends (Fig. 12.21a), or by the

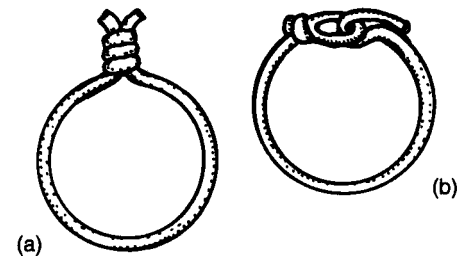


Fig. 21.21 Cerclage wire tightened by (a) a twist knot and (b) an AO/ASIF loop lock.

use of an AO/ASIF loop (Fig. 12.21b). If a twist knot is used, it is imperative to ensure that the first few twists are equally distributed on each wire (Fig. 12.21a). Uneven twisting may cause the wire to break before it is fully tightened or may form a slip knot which is likely to loosen. The question 'how tight is tight enough?' is difficult to answer specifically. However, the surgeon should not be afraid to apply a clamp to the knot and give the wire a wiggle. If the wire moves or the surgeon is reluctant to do this then the wire is not tight enough!

Movement of a cerclage wire can cause lysis of the underlying bone. This complication can be minimised by ensuring that the wire is applied tightly and, if necessary, the cortex of the bone is notched to prevent the wire from slipping. However, if absolute stability is to be ensured, the wire should penetrate the cortex of the bone before being passed around the fragments. A wire placed in this fashion is called a *hemicerclage wire* (Fig. 12.22).

When cerclage wire is combined with an intramedullary pin to reconstruct a cylinder of cortical bone where there has been comminution, the technique should be limited to two, three or possibly four piece fractures. If there are more fragments then they will tend to shake loose if incorporated within cerclage wires (the so-called 'bundle of sticks' phenomenon). Under these circumstances a 'buttress' plate, external fixator or interlocking nail should be used to maintain length and alignment of the fractured bone rather than an intramedullary pin and cerclage wires.

Wire may also be used in combination with Kirschner wires to create a *tension band* effect.

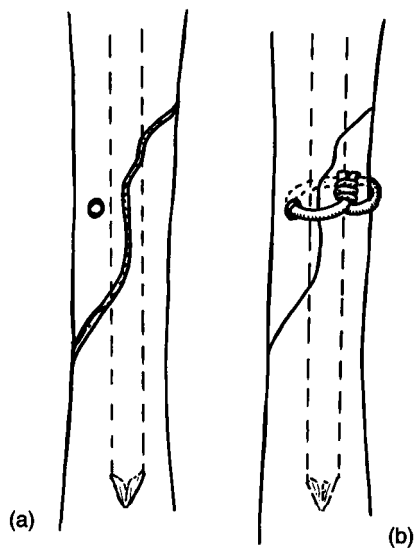


Fig. 12.22 Application of a hemicerclage wire for optimal stability. (a) To stabilise an oblique fracture a hole is drilled in the cortex and then (b) a wire is placed through the hole and tightened.

This technique is used, in particular, in the management of avulsion fractures (see below).

Tension band wiring

The main indication for tension band wiring is the treatment of avulsion fractures such as those involving the:

- Olecranon
- Greater trochanter
- Patella
- Tibial tuberosity
- Malleoli
- Os calcis

In all these fractures the fragment is distracted by the muscle, tendon or ligament which originates from/inserts on it. The tension band is placed so that it counteracts the tensile force acting on the fragments and redirects it to compress the fragment against the adjacent bone (Pauwels, 1965). Tension band wiring is also used in the repair of osteotomies, for example transolecranon osteotomy in the caudal approach to the elbow (Chapter 32) or osteotomy of the greater trochanter in the dorsal approach to the hip

(Chapter 39). Another indication is intertarsal arthrodesis (Chapter 44).

The treatment of an avulsion fracture of the tibial tuberosity is used here as an example of tension band wiring. This is a fairly common injury in the immature dog (especially Greyhounds and Terriers in the authors' experience) and the fracture occurs through the growth plate. The tibial tuberosity is distracted by the tensile force of the quadriceps muscles exerted through the straight patellar ligament (Fig. 12.23a). The fracture is reduced and intitial fixation is achieved with two Kirschner wires driven through the tuberosity into the metaphysis (Fig. 12.23b). A hole is drilled transversely though the tibia distal to the fracture site. A length of orthopaedic wire (size in line with the recommendations for cerclage wire given above) is passed through the hole. The ends of the wire are brought across the cranial aspect of the tibia in a figure-of-eight pattern and then passed through the straight patellar ligament before being twisted tight. As the wire is tightened its proximal loop engages on the protruding ends of the Kirschner wires (Fig. 12.23c). Each Kirschner wire is then bent and cut leaving a hook about 0.5 cm long which is rotated to fit snugly against the tibial tuberosity and insertion of the straight patellar ligament (Fig. 12.23d). Although, theoretically, a twist should be placed in each side of the figure-of-eight to achieve adequate tension in the wire (Fig. 12.24), in many 'short' tension band wires adequate tightening can be achieved with a single twist on one side of the figure-of-eight.

The tension band wire counteracts the pull of the straight patellar ligament and the resultant vector (v) compresses the fracture site (Fig.

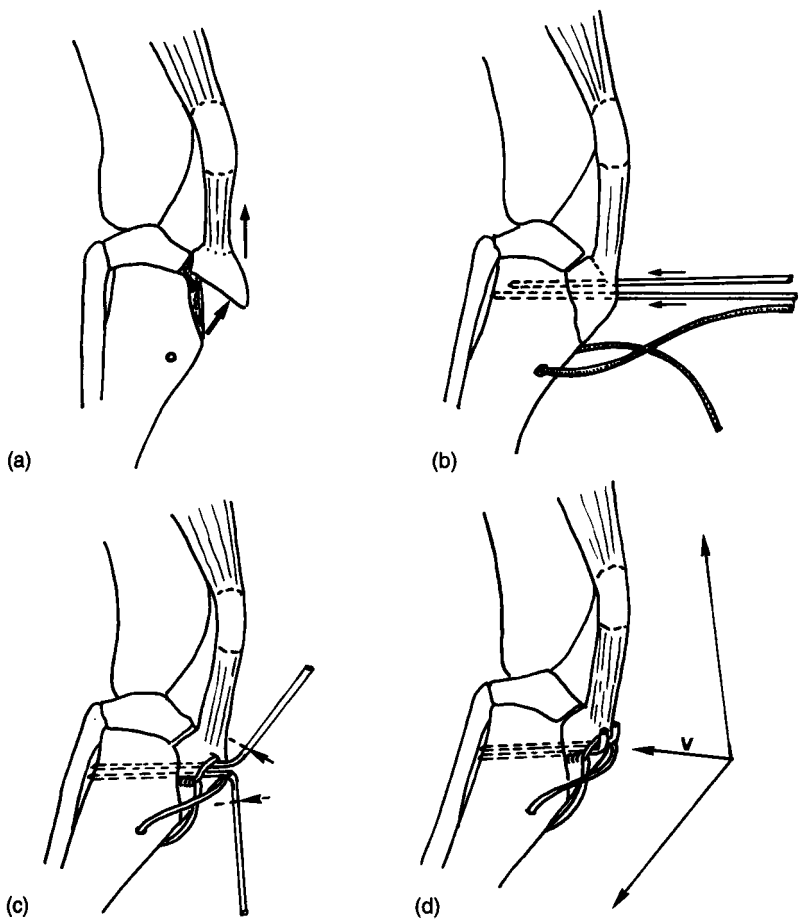


Fig. 12.23 Use of the tension band wiring technique for an avulsion fracture of the tibial tuberosity. See text for details.

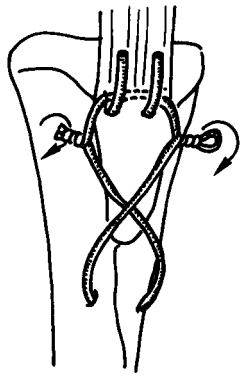


Fig. 12.24 Tightening both sides of the tension band wire.

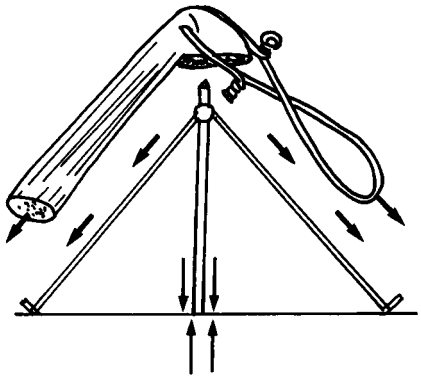


Fig. 12.25 Tension band acting like a guy rope to give functional compression at the fracture site.

12.23d). The tension band is acting in exactly the same way as a guy rope holding up a tent pole (Fig. 12.25). Imagine the tent pole is the tibial tuberosity, the straight patellar ligament is one guy rope and the tension band wire is the other. The opposing pull of the two guy ropes will cause compression between the tent pole and the ground or, in this example, between the tibial tuberosity and the metaphysis.

Details of tension band wiring of other avulsion fractures are given elsewhere in the book.

Bone screws

Bone screws used in orthopaedic surgery can be divided into three broad groups:

Fig. 12.26 Sherman self-tapping screw.

(1) Self-tapping screws (e.g. the Sherman screw)
(2) Cortical screws (first designed by the AO/ASIF group)
(3) Cancellous screws (first designed by the AO/ASIF group)

Self-tapping (e.g. Sherman) screws

This type of screw is illustrated in Fig. 12.26. It has a slotted head, the threads are at an angle to the long axis and it has a fluted end for cutting a thread in the bone. Screws are available in three diameters: 2.0 mm, 2.7 mm ($\frac{7}{64}$″) and 3.5 mm ($\frac{9}{64}$″). The appropriately sized drill bits to be used with these screws are shown in Table 12.4.

Basic steps for insertion of a self-tapping screw

(1) Drill a pilot hole through both cortices of the bone. A clearance or gliding hole, of the same diameter as the screw, should be drilled in near to the fragment if the screw crosses a fracture line – see below for lag screw effect. If the screw is being placed directly into diaphyseal bone (i.e. rather than being placed through a plate) then the hole should be countersunk so that the screw head engages the bone more evenly.
(2) Measure the depth of the hole with a depth gauge. Select a screw 2 mm longer than indicated to ensure that the threads of the screw get full purchase in the far cortex.
(3) Insert the screw using a screwdriver.

Table 12.4 Drill sizes required for self-tapping screws (Veterinary Instrumentation).

Screw diameter (mm)	Drill size for pilot hole (mm)	Drill size for clearance, or gliding, hole (mm)
2.0	1.5	2.0
2.7	2.4	2.7
3.5	2.7	3.5

Table 12.5 Drill sizes required for cortical screws (Synthes, Stratec Medical; Aesculap, Veterinary Instrumentation).

Screw diameter (mm)	Drill size for pilot hole (mm)	Drill size for clearance, or gliding, hole (mm)
1.5	1.1	1.5
2.0	1.5	2.0
2.7	2.0	2.7
3.5	2.5	3.5
4.5	3.2	4.5

Cortical screws

The design of a cortical screw is illustrated in Fig. 12.27. The threads are perpendicular to the long axis of the screw and are designed to achieve maximum purchase in the hard cortical bone of the diaphysis. The screw has a hexagonal head. The screw sizes available and the appropriately sized drill bits to be used with these screws are shown in Table 12.5.

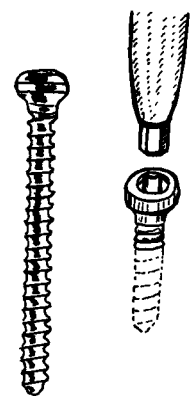


Fig. 12.27 Cortical screw.

Basic steps for insertion of a cortical screw

(1) Drill a pilot hole through both cortices of the bone. A clearance or gliding hole, of the same diameter as the screw, should be drilled in near to the fragment if the screw crosses a fracture line – see below for lag screw effect. If the screw is being placed directly into diaphyseal bone (i.e. rather than being placed through a plate) then the hole should be countersunk so that the screw head engages the bone more evenly.

(2) Measure the depth of the hole with a depth gauge. Select a screw 2 mm longer than indicated to ensure maximum purchase in the far cortex, unless the depth gauge already takes account of this (there is variation between systems in this respect).

(3) Use a tap to cut a thread for the screw in the pilot hole.

(4) Insert the screw using a screwdriver.

Cancellous screw

The design of a cancellous screw is illustrated in Fig. 12.28. They have a coarser thread than the cortical screw which is designed to get a better grip in the soft cancellous bone of the metaphysis and epiphysis. Cancellous screws can be fully or partially threaded. The screw sizes available and

Table 12.6 Drill sizes required for cancellous screws (Synthes, Stratec Medical; Aesculap, Veterinary Instrumentation).

Screw diameter (mm)	Drill size for pilot hole (mm)	Drill size for clearance, or gliding, hole (mm)
3.5 (fully threaded)	2.0	3.5
4.0 (partially or fully threaded)	2.0	4.0

Fig. 12.28 Cancellous screw.

the appropriately sized drill bits to be used with these screws are shown in Table 12.6. The steps for insertion of a cancellous screw differ from those used for a cortical screw in three ways.

(1) If partially threaded screws are used as lag screws then it is not generally necessary to drill a clearance (or gliding) hole in the near fragment.
(2) Since the screw is being placed in the metaphysis or epiphysis, where the cortex is relatively thin, countersinking is contraindicated as it will tend to encourage the screw head to sink into the bone, indeed in these regions it is often necessary to combine a bone screw with a washer to reduce the degree of sinking.
(3) Once the pilot hole has been drilled, it is not usually necessary to use a tap to cut a thread in the soft cancellous bone before insertion of the cancellous screw. However, the tap may be required in older animals with harder bone.

Indications for bone screws

- To provide interfragmental compression using the lag screw principle
- As a position screw
- As a plate screw

Lag screw

When a screw is used to compress two fragments of bone together it is referred to as a lag screw. The technique involves a clearance or gliding hole, which is the same diameter as the screw, being drilled though the near fragment, while a smaller, pilot hole is drilled through the far fragment (Fig. 12.29a). This method of drilling holes ensures that when the screw is inserted the screw thread grips in the far fragment only (Fig. 12.29b) and as the screw is tightened the two fragments of bone are drawn together producing interfragmentary compression (Fig. 12.29c). If the screw thread gripped in both fragments (position screw) it would be impossible to compress the fracture as the screw was tightened.

When a lag screw is used for repair of a condylar fracture of the humerus it is usually combined with a Kirschner wire which is placed as a second point of fixation to prevent rotation of the fragment (Fig. 12.30). If lag screws are used to compress mid-diaphyseal oblique or spiral fractures then they must be protected from angular, torsional or compressive forces in some way. This most often involves the use of a neutralisation plate but may also be achieved with such devices as an ESF. The orientation of an oblique fracture may mean that a lag screw has to be placed through one of the plate holes. It is important to remember that whenever a screw crosses a

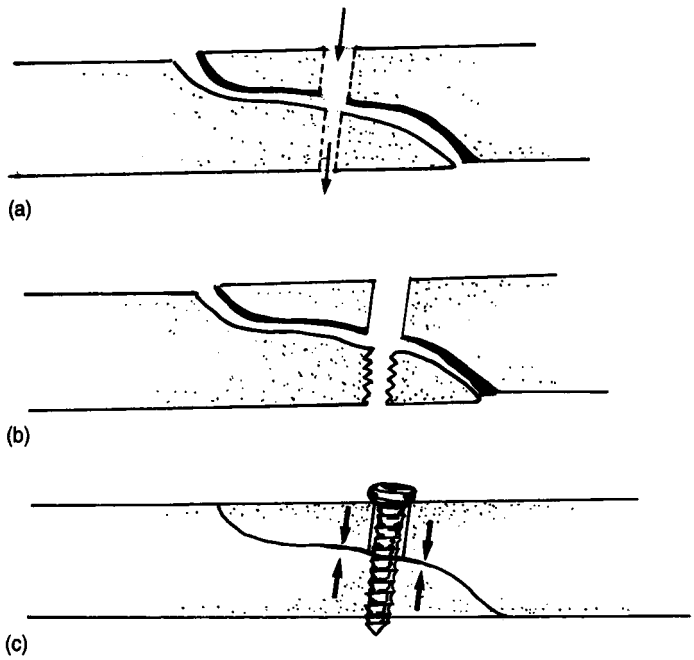


Fig. 12.29 Lag screw fixation. (a) Preparation of a clearance hole (near fragment) and a pilot hole (far fragment) for the screw. (b) Screw thread is in the far fragment only. (c) As the screw is tightened interfragmentary compression results.

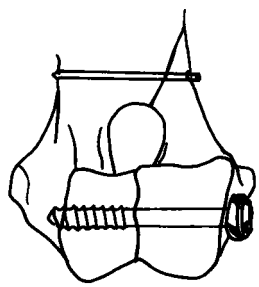


Fig. 12.30 Condylar fracture of the humerus. Cancellous screw used as a lag screw. Second point of fixation provided with Kirschner wire.

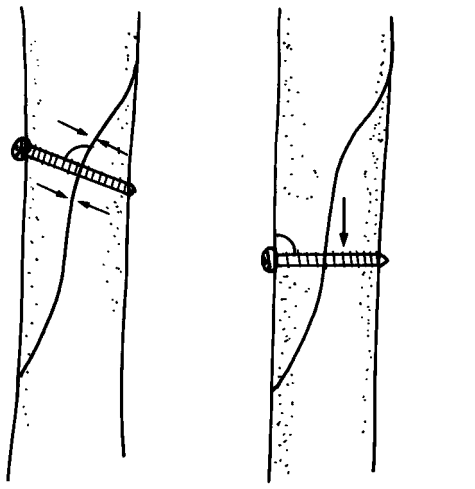
fracture line then that screw should be placed as a lag screw.

The angle of insertion of lag screws in oblique or spiral fractures also needs to be considered. A screw placed at right angles to the fracture line will give maximum interfragmental compression (Fig. 12.31a), while a screw placed at right angles to the long axis of the diaphysis (Fig. 12.31b) will offer greatest resistance to shortening forces but not maximal interfragmental compression. Maximum use of the benefits of these two screw positions can be made by insertion of a screw along an imaginary line that bisects the angle between a perpendicular to the long axis of the diaphysis and a perpendicular to the fracture plane (Fig. 12.31c). In practice several screws are inserted at different angles to each other to counteract shearing and torsional forces and at least one of the screws is usually placed at right angles to the long axis of the bone (Fig. 12.31d). Screws should not be used as the sole method of fixation of oblique or spiral fractures. They should always be used in conjunction with a neutralisation plate or external fixator.

The selection of an appropriate size of bone screw to act as a lag screw is related to the size of the bone fragment involved. As a general guide, the diameter of the screw should not exceed one-third of the width of the fragment at its site of proposed insertion.

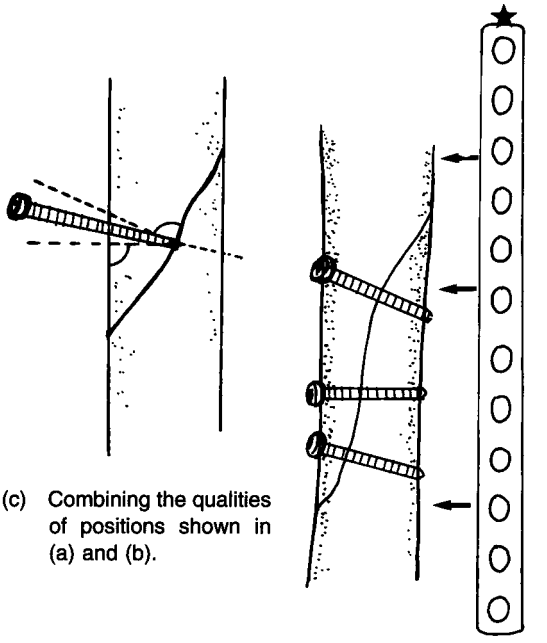
Position screws

In fractures of small bones a screw may be used to hold a fragment in position once the fracture has been reduced. The fragment may be too small, for example in an accessory carpal bone fracture, to allow the screw to be used as a lag screw because it will not withstand either overdrilling of



(a) Maximum interfragmentary compression.

(b) Maximum resistance to shortening forces.



(c) Combining the qualities of positions shown in (a) and (b).

(d) Always protect the screw repair with a neutralisation plate (*) or external fixator.

Fig. 12.31 Lag screw fixation in oblique fractures. The qualities of different screw positions.

the clearance hole or the stresses generated by such compression. Under these circumstances a pilot hole is drilled through both fragments and the screw inserted. The screw thread grips in both fragments and holds them in position. When used to serve this function the screw is termed a position screw.

The selection of an appropriate size of bone screw to act as a position screw is related to the size of the bone fragment involved. As a general guide, the diameter of the screw should not exceed one-third of the width of the fragment at its site of proposed insertion, though when dealing with small fragments the smaller screws may still tend to exceed this rule.

Plate screw

Bone screws are used to secure plates to bones. The screws pass through the plate and both cortices of the bone, in effect lagging the plate to the bone (further details are given under plate application, p. 123).

Advantages of the non-self-tapping cortical and cancellous screws over self-tapping, Sherman type screws

Traditionally the advantages listed have been:

(1) Better purchase in bone due to:
   (a) Thread design which is perpendicular to the screw rather than at an angle and so is less likely to allow pull-out.
   (b) The use of a tap to cut the thread removes bone debris giving a better fit for the screw.

(2) The hexagonal hole in the screw head allows the screw to be driven home without needing direct pressure which would put stress on the threads already cut in the bone and also reduces the likelihood of the screwdriver slipping and causing further soft tissue trauma.

(3) A screw may be changed if the length is found to be wrong. With self-tapping screws, replacing a screw could cause further threads to be cut which might reduce considerably the bone's holding ability.

(4) The AO/ASIF-type systems come with high-quality instrumentation which allows screws to be placed more accurately and this makes

these systems as a whole more adaptable, particularly in awkward fractures with limited bone stock, proximally or distally.

Although much emphasis was originally placed on the non-self-tapping screws' ability to gain better purchase, it is now considered that the main advantages of these bone screws relate to the high-quality instrumentation designed for their accurate application. This advantage may become somewhat eroded by the recent development of self-tapping screws by the AO/ASIF group to be used with similar instrumentation. Although such systems are relatively new and limited in use, they may become more widely accepted in the not too distant future.

Bone plates

The correct application of a bone plate and screws should result in optimal stability at the fracture site and allow early, pain-free limb function. A large variety of plates is available for use in veterinary orthopaedics. These can be broadly divided into three groups:

(1) The traditional round hole plates (Sherman, Lane, Venables, Burns) which are most commonly used with self-tapping Sherman screws (Fig. 12.32). These plates are usually applied without compression since to do so requires the use of a complicated piece of apparatus.
(2) AO/ASIF plates, the most common of which would be the dynamic compression plates (DCPs).
(3) Special plates (Fig. 12.33)
    - reconstruction plate
    - triple pelvic osteotomy plate
    - T-plate
    - acetabular plate
    - limb lengthening plate
    - cuttable plate

Application of a standard round hole plate using 3.5 mm ($\frac{9}{64}$") diameter Sherman screws (Fig. 12.34)

(1) The fracture is reduced and any interfragmentary implants (lag screws) placed accordingly.

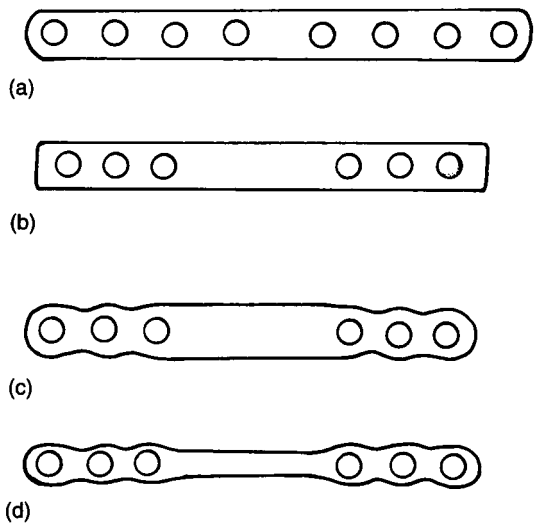


Fig. 12.32 Round hole plates. (a) Round hole plate (Veterinary Instrumentation), (b) Venable plate, (c) Burns plate, (d) Sherman plate (a Lane plate is similar but weaker).

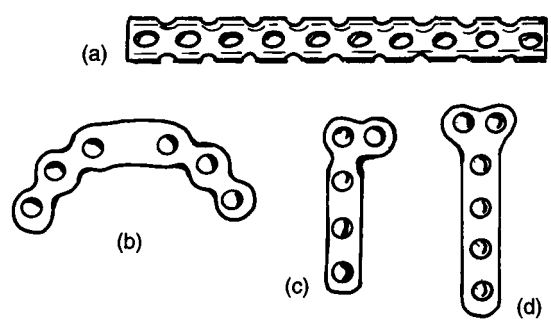


Fig. 12.33 Special plates. (a) Reconstruction plate, (b) acetabular plate, (c) angled plate, (d) 'T' plate.

(2) The longest plate that can easily be inserted is chosen (Fig. 12.34a). This should allow at least three screws to be placed on either side of the fracture site if the plate is functioning as a neutralisation plate and, ideally, four if the plate functions as a buttress plate (see below). The size of plate chosen is determined by the size of the bone. Generally, the plate screws should not exceed one-third of the width of the bone as seen radiographically and the size of these screws, in turn, determines the size of plate used.
(3) The plate is accurately contoured to the shape of the bone using plate benders.

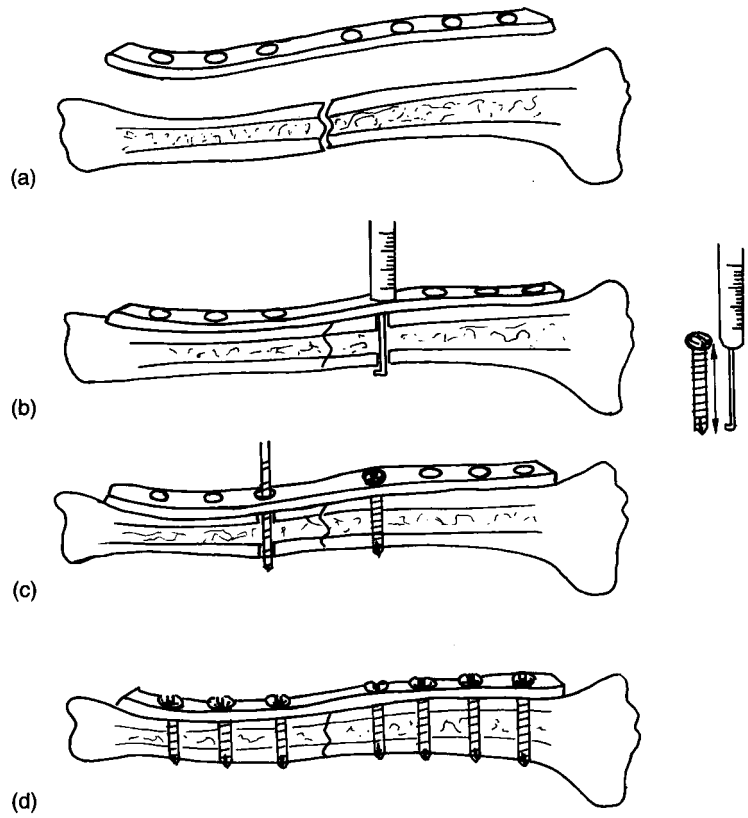


Fig. 12.34 Application of a round hole plate. See text for details.

(4) A 2.7 mm ($\frac{7}{64}$″) drill bit is used to drill the first screw hole about 1 cm from the fracture site. The hole should penetrate both cortices.
(5) A depth gauge is used to measure the length of the hole and a screw is chosen about 2 mm longer than indicated to ensure that the screw thread grips in both cortices (unless the depth gauge already accounts for this in the reading it gives – this varies between systems) (Fig. 12.34b).
(6) The screw is inserted using a screwdriver.
(7) After insertion of the first screw, the next screw hole is drilled on the other side of the fracture site and the appropriately sized screw is inserted (Fig. 12.34c).
(8) The rest of the screws are inserted, working away from the fracture site (Fig. 12.34d).

The weakest point on any plate is the screw hole and care should be taken in the application of the plate to avoid leaving an empty screw hole directly over the fracture site, otherwise the plate may well break at this point before fracture healing is complete.

With respect to which aspect of a bone a plate should be applied, this is governed by a number of factors, including:

- Which aspect of the bone is easiest to expose
- The position of lag screws in relation to the plate
- Which is the tension side of the bone, since a plate is stronger under tension than compression (see below)

In fact the easiest side to expose is often the tension side but where these are not the same the ease of exposure usually takes precedence.

Tension and compression sides of a bone

If a bone is thought of as a column, and a load is placed over its centre (Fig. 12.35a), then within that column there are only compressive forces.

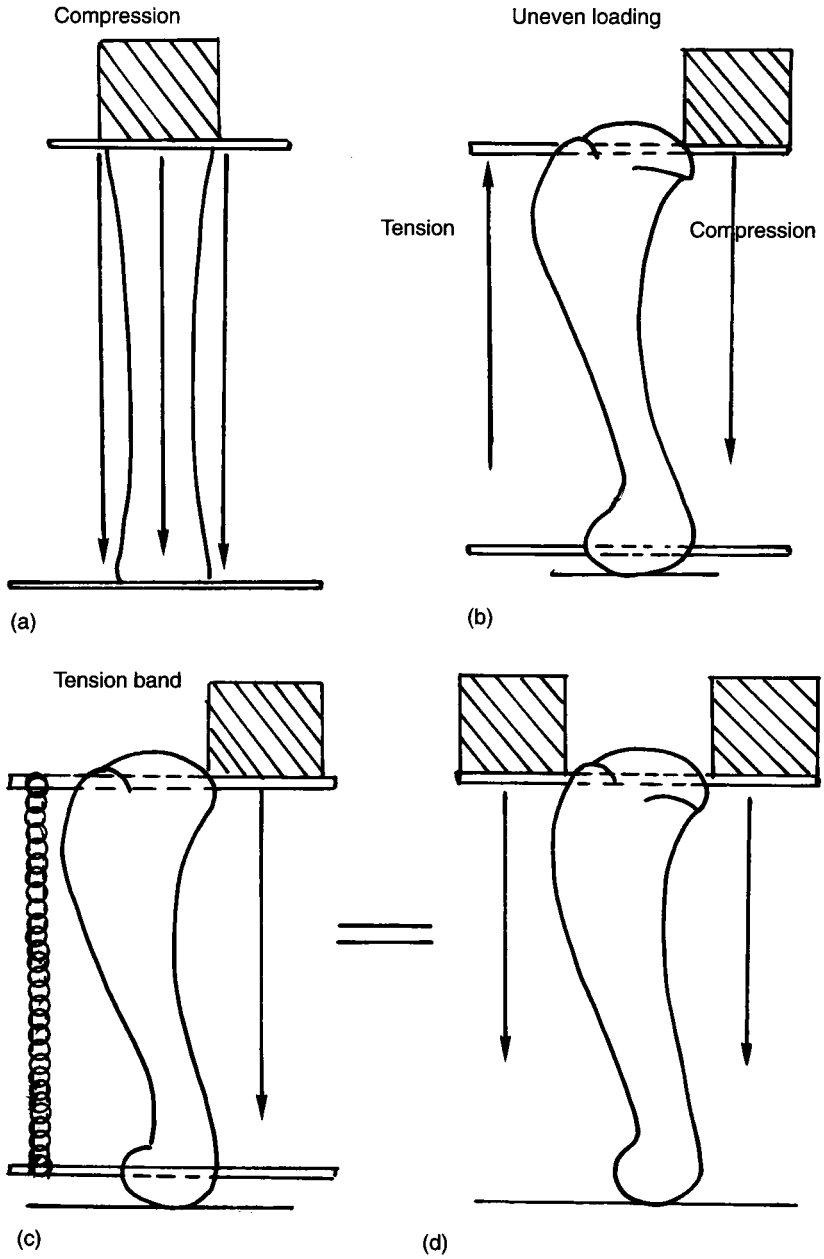


Fig. 12.35 The tension band principle. See text for details.

However, if the load is placed to one side of the column (Fig. 12.35b) then there are extra bending or compressive forces exerted on this side of the column with equal and opposite tensile forces on the other side of the column. These tensile forces can be neutralised with a tension band, which is shown as a chain in the illustration (Fig. 12.35c) but in a clinical situation would be a compression plate or wire. This then acts as if a load had been placed on the tension side of the column creating compression (Fig. 12.35d). It is this tension band principle that is utilised in the application of

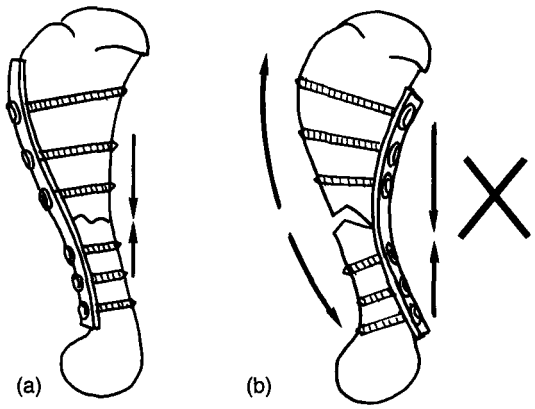


Fig. 12.36 Application of compression plates. (a) Apply plate to tension side. (b) Avoid compression side – risk of bending and implant failure.

compression plates to achieve axial compression (Pauwels, 1965) (Fig. 12.36a).

Normally bones are unevenly loaded so that one side of the bone is under compression and the other side is under tension. Implants should be placed on the tension side of the bone, otherwise they will be subjected to repeated bending and compressive forces and may break before fracture healing is complete (Fig. 12.36b). Unfortunately, the loading of bone is not as simple as shown in Fig. 12.35 and the stresses in bone are continually changing depending on:

- Weight-bearing
- Locomotion
- Muscle pull

In addition, a fracture causes a complete disruption in the normal stresses. Nevertheless, the tension side of certain long bones has been established as follows:

- The cranial aspect of the proximal humerus
- The caudal aspect of the olecranon
- The lateral aspect of the proximal femur
- The craniomedial aspect of the distal tibia

When a plate is applied it can be used to serve one of three functions, as a:

- Compression plate
- Neutralisation plate
- Buttress plate

Compression plate

A compression plate is used to create axial compression at a transverse, or short oblique, fracture line. The plate is applied to the tension side of the bone in order to achieve functional compression (as discussed above).

To achieve this with standard, round-hole plates a special 'jig' has to be applied to the bone during the operation and this requires exposure of more bone distally or proximally for attachment of the apparatus. Because of the limitations in using such apparatus the application of compression using plates is now generally restricted to the use of dynamic compression plates (DCPs) (Allgower *et al.*, 1973). Such plates include those made by Synthes (supplied through Stratec Medical) and Aesculap (supplied through Veterinary Instrumentation, Sheffield).

The main feature of the DCP is the design of the screw hole which is based on the spherical gliding principle. This enables the DCP to be used as a self-compressing plate. Insertion of the screw in the load position will displace the plate resulting in compression of the fracture site as the screw head is tightened down against the hemicylindrical slope of the screw hole (Fig. 12.37a). The spherical geometry of the screw hole also ensures that there is a congruent fit between the screw and the plate in any position along the screw hole, while permitting a degree of tilt (25° longitudinally and 7° to each side) between the screw and the plate. The DCP is ideal for treating multiple fractures of a long bone in that individual fragments can be compressed together by the introduction of successive screws in the plate.

The application of the DCP as a self-compressing plate is illustrated in Fig. 12.37:

(1) The fracture is reduced and the plate is carefully contoured to fit the bone. The plate is then prestressed by contouring it to leave a 1 mm gap under the plate directly over the fracture line. This ensures that, as the screws are placed, both the near and far cortices are compressed. Failing to prestress the plate will mean that as the screw heads tighten down the near cortex will compress whilst the far cortex will produce a gap which is far from ideal.

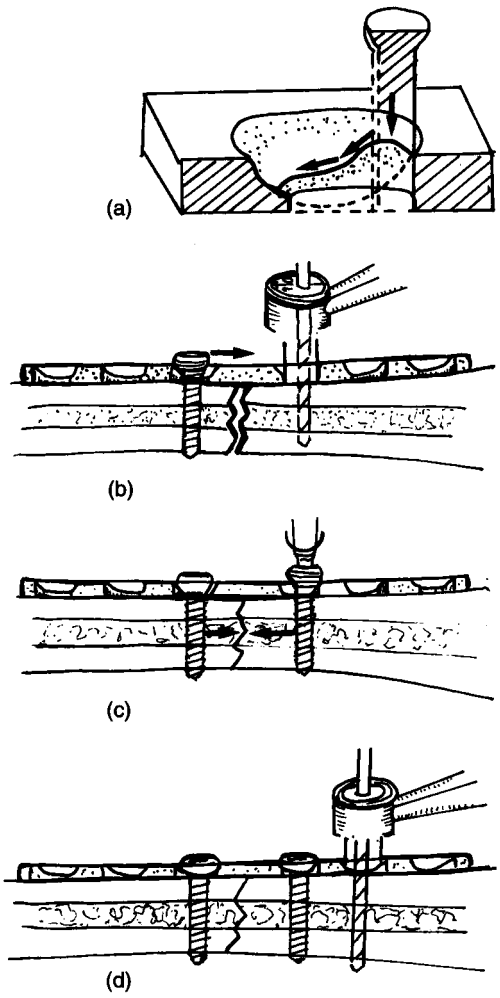


Fig. 12.37 Design of the dynamic compression plate (DCP) and its application. (a) Design of the DCP screw hole. (b) The loaded drill guide. (c) Tightening the first two screws compresses the fracture. (d) The rest of the screws are placed using the neutral drill guide. (From Allgower, M., Matter, P., Perren, S.M. & Reudi, T. (1973) *The Dynamic Compression Plate (DCP)*. Springer Verlag, Berlin. Redrawn with permission.)

(2) The plate is then secured to one of the main fragments by means of a screw inserted about 1 cm from the fracture line. A neutral drill guide is used for positioning this screw. The screw is not fully tightened and the plate is slid towards the fracture site.

(3) A second screw is placed in the opposite fragment but with the aid of the loaded drill guide (positioned with the arrow pointing towards the fracture line) (Fig. 12.37b).

(4) The two screws on either side of the fracture are tightened and the fracture is compressed (Fig. 12.37c).

(5) The remaining screws are inserted with the aid of a neutral drill guide working away from the fracture site (Fig. 12.37d).

The loaded drill guide is generally used once on either side of the fracture. However, if further compression is required another screw can be inserted in the load position on either side of the fracture before placing the remaining screws with a neutral drill guide (except in the case of the 2.0 mm DCPs).

The size of a DCP relates to the diameter of screw with which it is combined. Hence a 2.7 DCP is used with 2.7 mm diameter cortical screws and a 4.5 DCP takes 4.5 mm cortical screws. The greatest fracture gap which can be closed with the different sized DCPs is as follows:

2.7 DCP (used small dogs and cats)	will close a gap of	3.2 mm
3.5 DCP (used in medium to large breeds of dog)	will close a gap of	4.0 mm
4.5 DCP (used in large or giant breeds of dog)	will close a gap of	4.0 mm

(A mini (2.0) DCP which can be used with 1.5 or 2 mm cortical screws is available for use in cats and toy/miniature breeds of dog.)

However, it must be stressed that the aim is to achieve anatomical reduction before applying the plate to create compression rather than trying to use the plate to reduce the fracture.

A prime indication for compression plate fixation is a simple, transverse, diaphyseal fracture, e.g. radius and ulna. Compression plate fixation provides optimal stability and primary bone union should result.

Neutralisation plate

In oblique or comminuted fractures of a long bone initial reconstruction of the bone is generally achieved with lag screws. A plate is then applied without compression to transmit disruptive torsional, compressive and bending forces from the proximal to the distal end of the bone, thus protecting the area which has been reconstructed with lag screws from excessive loading (Fig. 12.38). In this situation the plate is functioning as a neutralisation plate. Ideally the plate is applied to the tension side of the bone and a minimum of three screws should be used to attach the plate to the proximal fragment and three to the distal fragment. In this function the bone and plate 'share' the load created by weight-bearing. Any plate, including a DCP, can be used in this function. When a DCP is used then all the screws are placed using the neutral guide.

Buttress plate

A buttress plate is a plate used to maintain bone length and angular relationships between joint surfaces. The plate can be used to shore up an articular surface, e.g. the tibial plateau (Fig. 12.39) or, more commonly, the plate is used to span a gap in a bone caused by severe comminution where the fragments are too small for reconstruction using lag screws. In the latter situation the plate needs to be strong enough to take the weight of the patient thus transferring the load from the proximal to the distal fragment and bypassing the area of comminution. There are special plates known as limb lengthening plates (Fig. 12.40) which are designed for this purpose. The central span of the plate which protects the area of comminution does not contain any screw holes which

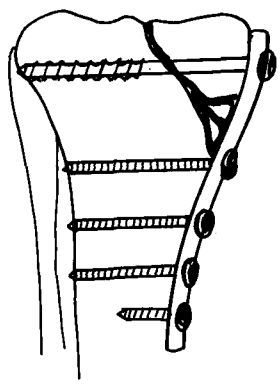


Fig. 12.39 A buttress plate being used to shore up the tibial plateau in the treatment of a proximal diaphyseal fracture.

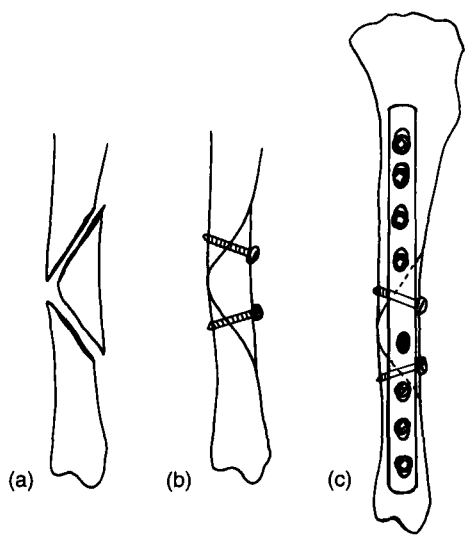


Fig. 12.38 (a) A comminuted fracture. (b) Reconstruction using lag screws. (c) The repair is protected with a neutralisation plate.

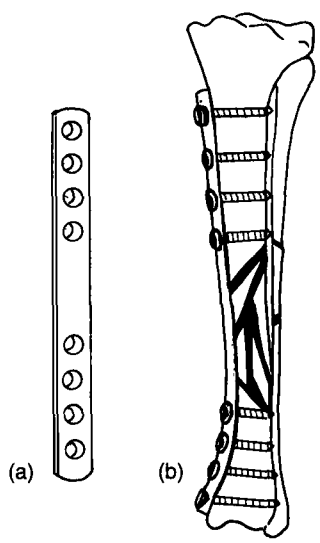


Fig. 12.40 (a) A lengthening plate and (b) a lengthening plate used as a buttress plate.

increases the strength of the plate in this region. Ideally a minimum of four screws are used to attach the plate to the proximal fragment and four to the distal fragment. Other plates, including DCPs, can be used as buttress plates. In general, for any given bone, a larger size of plate is used if serving as a buttress plate compared to one serving as a neutralisation plate, e.g. where a 3.5 mm DCP might be used for neutralisation, a broad 3.5 mm DCP might be chosen to buttress a fracture. When a DCP is used for this purpose the screw holes are kept close to the end of the plate hole nearest the fracture, i.e. nearer than that achieved by the neutral guide. Correct screw placement can be achieved by using a normal drill guide or else by turning the load guide around so that the arrow points away from the fracture.

Fracture healing is always a race between metal fatigue and bone union. To increase the odds in favour of bone union an autogenous cancellous bone graft is often used in the area of comminution to speed up the rate of bone healing.

It is important to note that when a DCP is used as a neutralisation plate or a buttress plate then the plate is applied without compression.

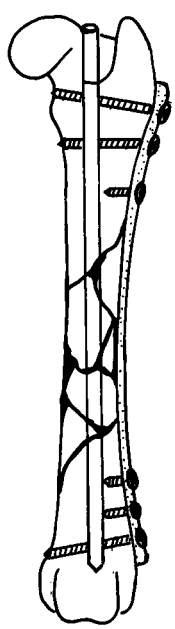


Fig. 12.41 A plate/rod system used to stabilise a comminuted femoral fracture.

Plate/rod systems

The use of plates in buttress fashion creates significant strain in an implant which is eccentrically positioned. It has been shown (Hulse *et al.*, 1997) that this plate strain can be reduced by addition of an intramedullary pin to the construct (Fig. 12.41). In general the pin is applied first to regain bone length and alignment between the proximal and distal fragments. In some cases the distal end of the pin may be cut off to leave a blunt end so that as it is introduced into the distal fragment it distracts the bone ends more efficiently, so regaining bone length. (This technique can also be used to assist in the application of a plate by holding the bone ends still. The pin is removed once some screws have been placed and this allows more of the plate screws to be bicortical.) A plate is then applied as described above. Because of the presence of the intramedullary pin, it is often only possible to use one bicortical plate screw in each of the proximal and distal fragments. If possible, more than this are applied, otherwise the remaining screws are monocortical.

Indications for plate removal

The general indications for plate removal may be listed as:

- Once the fracture has healed and the implant becomes redundant
- Persistent sinus formation over the plate associated with low-grade infection
- Thermal conduction/lameness
- Soft tissue irritation/lick granuloma
- Plate becomes non-functional

Post-fracture healing

Normally there is no need to remove plates which have been used to stabilise fractures in middle-aged, or older, dogs. Plates that have been used for fixation of the jaw or pelvis in dogs of any age are usually left *in situ* after healing is complete. When plates are used in treatment of long bone fractures in puppies under 1 year of age removal may be recommended as soon as fracture union is complete (2–4 months). Similarly, in dogs aged 1–3 years, particularly working dogs, it is usual to remove the plate after 5–6 months.

One of the reasons for removing the plate in these young dogs is to prevent the phenomenon of *stress protection*. Normal bone is constantly subjected to mechanical stresses that result in some bone deformation or strain every time weight is put on it or when isometric contraction of muscle occurs. These constantly changing stresses seem to be essential for the maintenance of the normal functional architecture of bone. When a rigid plate is used for fracture fixation the underlying bone is protected from stress (Allgower *et al.*, 1973). According to Wolff's law of adaptation to functional demand, this stress protection will cause bone destruction to prevail over osteogenesis in the course of remodelling leading to osteoporosis and a risk of refracture at one or other end of the plate if it is left *in situ*, or refracture of the bone soon after plate removal. Fortunately, stress protection in animals is seldom encountered as a clinical problem and when it occurs it is usually caused by using a plate which is too large or has been left *in situ* too long. There is considerable evidence now that in many cases of so-called stress protection the loss of bone which occurs beneath a plate is due to interference with the local vasculature rather than protection from natural stresses.

Although it may be desirable to remove plates after fracture healing of long bone fractures in young dogs, it can be difficult to persuade owners to have this second procedure carried out when the dog has made a full recovery after its fracture treatment. In the case of humeral fractures it is often safer to leave the plate *in situ* to avoid the risk of iatrogenic damage to the radial nerve. It is often difficult to identify the nerve during exposure of the plate because of the scar tissue resulting from the initial open reduction.

Low-grade infection

When fracture healing is complicated by infection and osteomyelitis, sinus tracts over the plate will not heal until the implants and any sequestra are removed (see Chapter 13, p. 140).

Thermal conduction/lameness

When a plate is used for fixation of fracture of the radius and ulna or tibia there is very little soft tissue cover for the plate. This can be associated with temperature changes between the plate and bone, particularly in cold weather, causing lameness. Plate removal solves the problem.

Irritation

When there is very little soft tissue cover over a plate, not only may this be associated with thermal changes but also local irritation and lick granulomas. Implant removal usually solves the problem.

Non-functional implant

Plates which are loose, bent or broken and are no longer serving a useful function should be removed, particularly if they are causing pain/lameness or are impeding healing.

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Chapter 13
Fracture Complications

Fracture complications can be broadly divided into complications of fracture management and those resulting from fracture treatment. Complications of management would include the fracture being open or associated with other injuries such as concurrent nerve damage or rupture of the diaphragm or bladder. Such complications are discussed elsewhere in this section. In this chapter, complications arising from the treatment of fractures will be considered, including:

- Delayed and non-union
- Osteomyelitis
- Malunion
- Fracture disease
- Fracture-associated sarcoma
- Fat embolism

However, it cannot be overstated that fracture complications are best prevented and, that having understood the contributing factors in their aetiology, the aim of fracture treatment should always be to avoid complications.

Delayed and non-union

The time taken for a fracture to heal is dependent upon a number of factors including:

- The age of the patient (see Table 1.1)
- The method of treatment chosen
- The type of fracture
- The bone involved
- Any concurrent systemic disease

The term 'delayed union' is, to an extent, subjective and applies when the healing time has exceeded what would be considered normal in that particular case. With time, the fracture might heal or else progress to 'non-union', at which point it is considered healing will not take place, however long the fracture is given. Delayed or non-union may result from one or a combination of factors, including:

- Inadequate stabilisation
- Poor vascularity
- Excessive fracture gap (+/– interposition of soft tissue, a loose implant or devitalised bone fragment)
- Infection
- Systemic or local disease
- Idiopathic factors (generally relating to atrophic non-union)

Radiology and classification of delayed and non-union

The radiographic appearance of these types of fracture complication may be useful in determining whether union is delayed or halted and also contributes to classifying any such non-unions.

Radiographic features of delayed union (Fig. 13.1):

- Persistent fracture line with evidence of healing
- Open medullary cavity
- Uneven fracture surfaces
- No sclerosis

Radiographic features of non-union (Fig. 13.2):

- Gap between fracture ends
- Closed medullary cavity (unless pinned)
- Smooth fracture surfaces
- Sclerosis
- +/– Hypertrophy or atrophy of bone ends

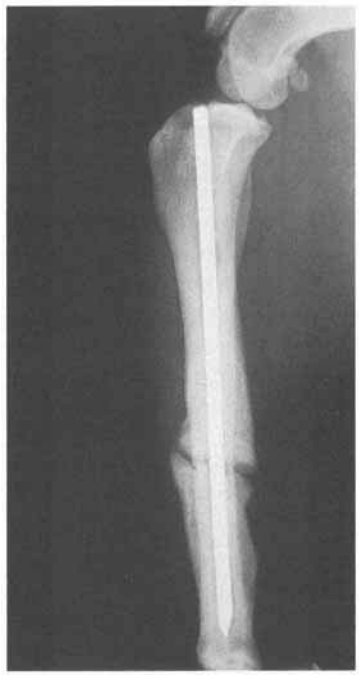


Fig. 13.1 Mediolateral radiograph of a 2-year-old Border Terrier's tibia taken 6 weeks after treatment of an interlocking, transverse fracture by intramedullary pinning. Although callus formation is present, the dog was clinically sound and the site felt stable, a clear fracture gap remains and thus union would have to be considered delayed (see Fig. 13.5).

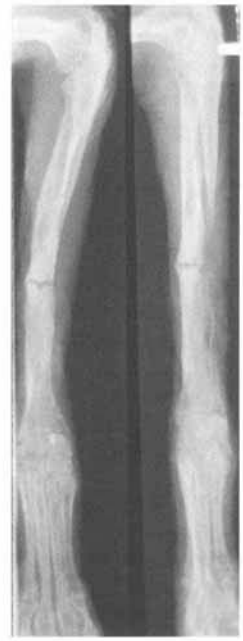


Fig. 13.2 Mediolateral and craniocaudal views of a 4-year-old Toy Poodle's antebrachium taken 2 months after treatment of transverse radius and ulna fractures by application of a cast. The site was clinically unstable. The radial fragments show sclerosis and mild formation of periosteal callus but a clear fracture gap remains and a diagnosis of slightly hypertrophic non-union was made. In the case of the ulna, the fracture ends have receded and a diagnosis of atrophic non-union would apply.

Traditionally, non-unions have been classified according the Weber–Cech system first described in 1976 (Weber & Cech, 1976). This classification divides non-unions into two broad groups: those that are biologically active (or viable) and those that are biologically inactive (or non-viable). These two groups are then further subdivided according to their cause and/or radiographic appearance (Fig. 13.3).

Biologically active or viable non-unions
These usually result from instability at the fracture site and may be classified as:

(1) *Hypertrophic non-union* ('elephant's foot' callus). There is abundant callus formation but failure to bridge the fracture gap, usually due to rotational instability. This type is most commonly seen in simple (transverse or short oblique) humeral or femoral fractures treated with an intramedullary pin.

(2) *Slightly hypertrophic non-union.* There is some callus formation but without bridging of the fracture gap. The cause is usually rotational and/or angular instability in simple (transverse or short oblique) radius/ulna or tibial fractures treated by external coaptation or intramedullary pinning.

(3) *Oligotrophic non-union.* In these cases there is no, or very limited, callus formation. The usual causes are either an avulsion injury treated conservatively or as above for slightly hypertrophic non-unions except that the dogs involved are usually small or toy breeds. Additionally, if non-union results from a related systemic disease, e.g. hyperadrenocorticism, then it is this type of non-union that might be expected.

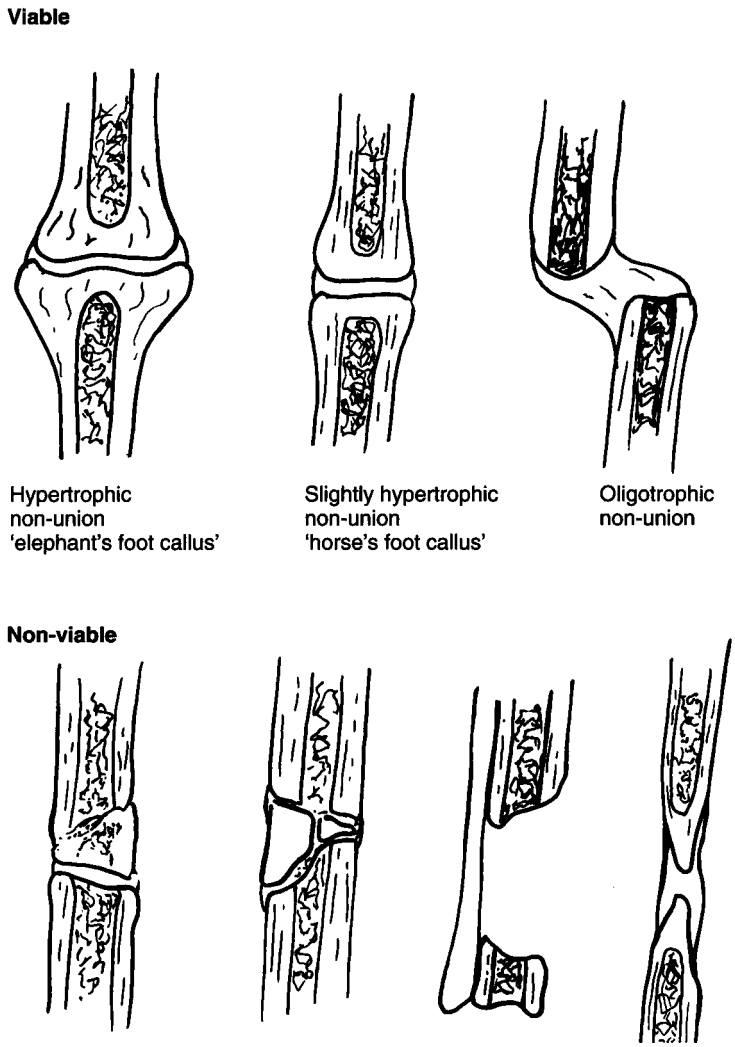


Fig. 13.3 Classification of non-union fractures (Weber & Cech, 1976).

Biologically inactive or non-viable non-unions
Although instability may play a role in the aetiology of these non-unions there is usually a barrier to healing which gives rise to a number of different types:

(1) *Dystrophic non-union.* An intermediate fragment becomes united with one of the two main fragments but not the other and this creates a devitalised portion of bone which does not participate in healing and, indeed, tends to create an obstruction.

(2) *Necrotic non-union.* These generally involve comminuted fractures in which the original injury, together with the surgeon's interference in most cases, creates avascular, necrotic pieces of bone within the fracture site and it is these that obstruct healing.

(3) *Defect non-union.* These occur where there has been significant loss of bone at the fracture site as a result of the injury with or without overzealous removal by the surgeon. The resulting gap between the remaining segments may exceed the bone's

ability to unite, especially if a cancellous bone graft has not been used.

(4) *Atrophic non-union.* Fortunately, these are rare in small animal orthopaedics. When seen they almost inevitably involve simple radius/ulna fractures in toy breeds of dog that have been treated by external coaptation or intramedullary pinning. In these breeds the ulna will often form an atrophic non-union even when the radius heals, but this is of no clinical significance. It must also be stressed that many non-unions of the radius in toy breed dogs are slightly hypertrophic or oligotrophic and the breed and fracture site should not lead directly to a diagnosis of atrophic non-union.

It may be considered that the terms biologically active/inactive and viable/non-viable would be more appropriately related to cellular activity which is more likely to correlate with scintigraphic, rather than radiographic, evaluation. The terminology used for these classifications may be misleading in that the term non-viable suggests healing will not take place whereas it will in a viable non-union. In fact healing will not occur in either without surgical intervention and the prognosis for non-viable non-unions is reasonable, with appropriate management, except, perhaps, in the case of atrophic non-union where the cause is poorly understood and treatment is recognised as difficult and often unsuccessful. Therefore, the classification system above is of limited value in planning treatment. It is more appropriate to consider the overall causes of non-union in each case in order to understand its particular management requirements (Fig. 13.4). The only non-unions that consistently defy treatment are the atrophic ones, which fortunately are rare, and perhaps only these should be considered truly 'non-viable'.

Treatment of delayed union

The main decision in these cases is whether to intervene or simply allow more time. If the duration of the fracture is nearing the time by which it should be healed, radiographic union is incomplete but manipulation detects no instability then allowing more time would be a sensible option (Fig. 13.5). However, if instability can be detected then simple intervention at that stage may avoid the need for more aggressive revision surgery at a later date. This is particularly true if instability is the only identifiable cause of the delayed

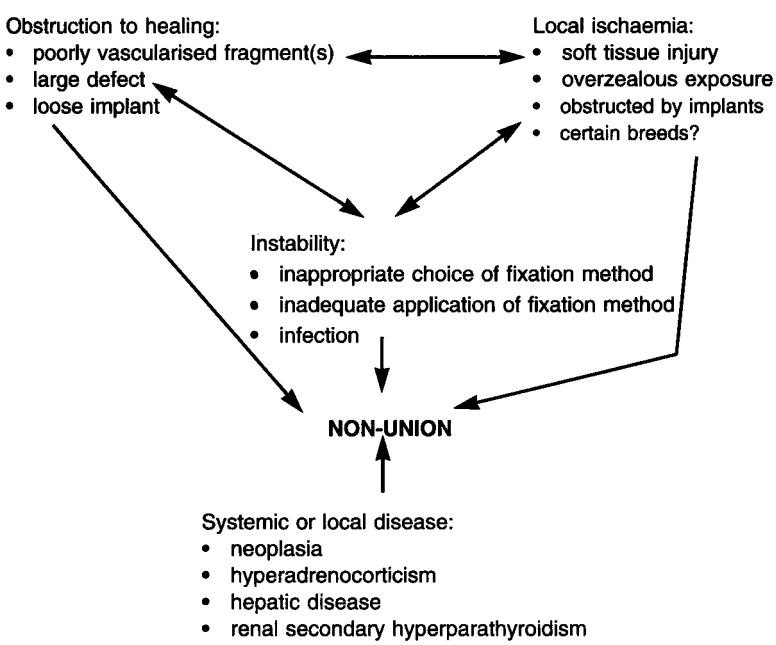


Fig. 13.4 Aetiology of non-union.

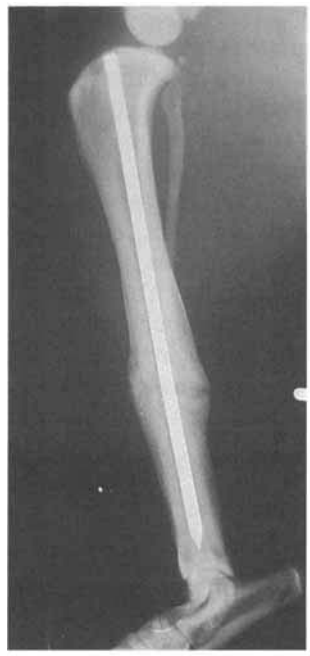


Fig. 13.5 Mediolateral radiograph of the tibia of the same dog as in Fig. 13.2, taken 12 weeks after surgery. Mineralised callus can now be seen bridging the fracture site.

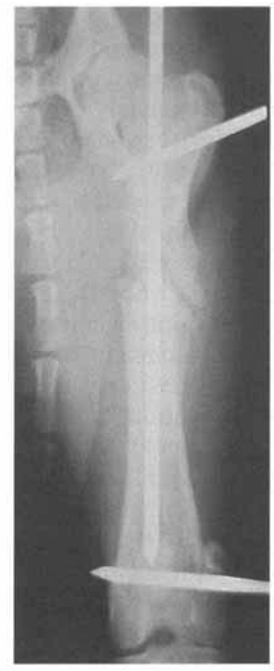


Fig. 13.6 Craniocaudal view of a 5-year-old Collie's femur taken 6 weeks after treatment of a diaphyseal fracture by intramedullary pinning. Although there is evidence of callus formation it does not appear to be bridging the fracture and the site showed mild rotational instability at that time. A diagnosis of delayed union was made. This radiograph was taken immediately after the closed application of a two-pin unilateral, uniplanar external skeletal fixator (ESF) to control rotational instability. Six weeks later fracture healing was complete and all the implants were removed.

union since the use of an external skeletal fixator (ESF) to replace a cast or as an adjunct to an intramedullary pin may be all that is required (Fig. 13.6).

Treatment of non-union

When managing non-unions it is first necessary to identify the cause(s) of the complication as described above and depicted in Fig. 13.4 under the headings of: instability, obstruction to healing, local ischaemia, and systemic or local disease.

Instability

This is the commonest cause of non-union and usually results from inappropriate use of implants, either in terms of the wrong choice of method or the incorrect application. For example, the use of an intramedullary pin to manage a simple fracture in a large, adult dog where rotational and some angular instability is likely to exist and may overcome the ability of the callus to bridge the fracture site (Fig. 13.7). This would constitute a wrong choice of method. Incorrect application would be exemplified by the use of a bone plate that is too small for its proposed function (Fig. 13.8) or with only one plate screw in either of the proximal or distal fragments (Fig. 13.9). Infection may also play a role in the creation of instability since it may cause lysis around adequately applied implants, resulting in their premature loosening and failure of the repair.

In cases where inherent instability of the fracture site is responsible, the result is often a hypertrophic or slightly hypertrophic non-union (Figs 13.2 and 13.7a). Treatment first involves removal of the inadequate implant system. Callus is resected only to gain adequate bone alignment and a surface on which to apply a plate (if this is

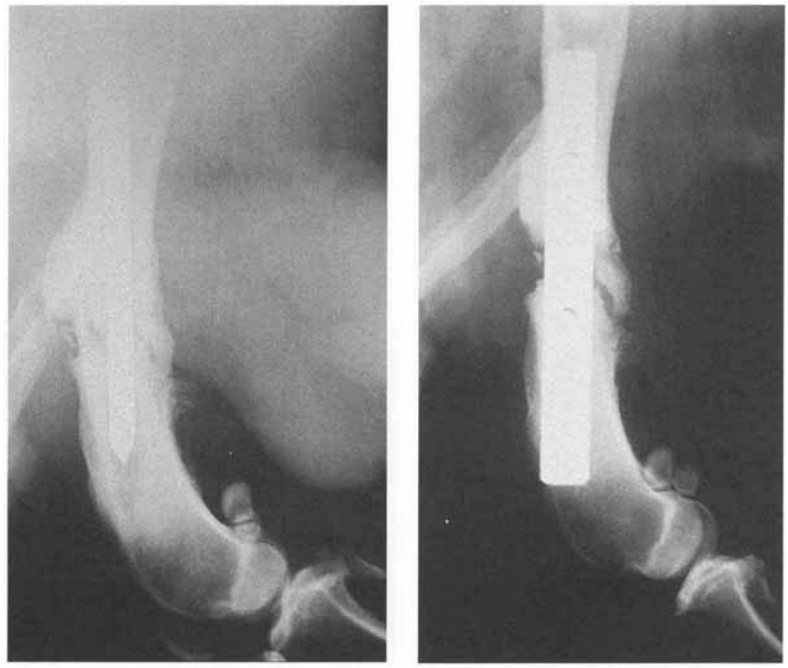


Fig. 13.7 (a) Mediolateral radiograph of a 5-year-old German Shepherd Dog's femur taken 4 months after treatment of a diaphyseal fracture by intramedullary pinning. The pin has not afforded sufficient rotational stability and movement at the fracture site has allowed proximal migration of the pin, increasing the degree of instability. A hypertrophic non-union is present.
(b) Mediolateral radiograph of the same femur taken after removal of the pin and application of a dynamic compression plate (DCP) (in compression mode) across the fracture site.

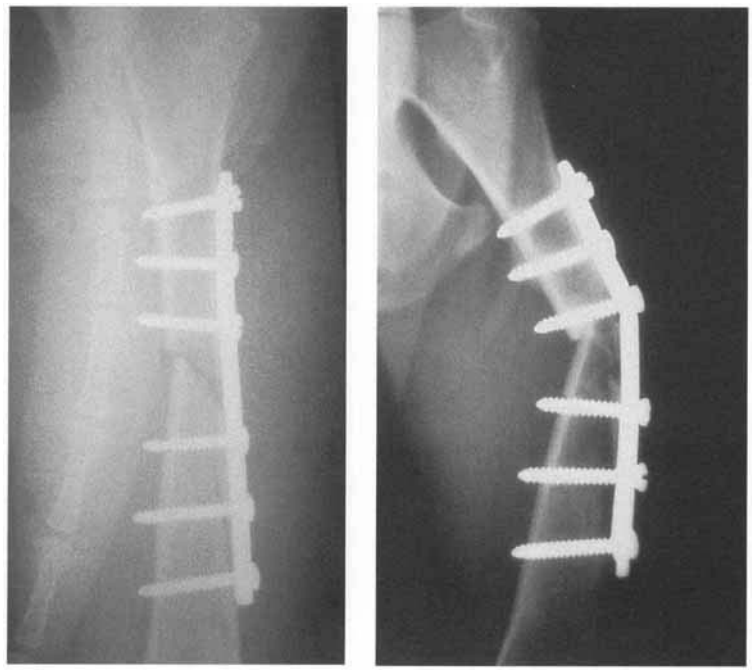
the chosen method of fixation). Stability and compression at the fracture site is then achieved, usually by application of a compression plate (Figs 13.7b and 13.10). An alternative to this in the distal limb is to achieve the same using a 'dynamic' ESF, incorporating threaded connecting bars which allow compression of the site (for example, an Ilizarov fixator). The need for a cancellous bone graft in such cases is questionable since the callus already formed should be able to produce bone union once the inhibitory affect of instability has been removed. However, there is never any harm in using such a graft if the surgeon is in any doubt about the viability of the site.

In cases where instability has resulted from implant failure these implants have to be removed and replaced by more adequate implants. If the fracture is simple then the same approach as given above applies (Fig. 13.9b). However, in these cases the fracture is often comminuted. Secondary reconstruction of the fragments is generally not advisable as they do not 'fit' together anatomically at this late stage and dissection around the site only serves to further reduce the vascular supply. Following implant removal, the fracture site is best bridged with a bone plate, interlocking nail or ESF (Fig. 13.11). It is also advisable to incorporate a cancellous bone graft into the repair.

In those cases where infection is also a complicating feature, the management may have to be modified as outlined below under 'Osteomyelitis'.

Obstruction to healing

Fracture union may be precluded by the presence of an obstruction such as necrotic piece(s) of bone (Fig. 13.12a), a loose implant (especially cerclage wire) or too large a fracture gap. Infection may contribute to the problem of bone necrosis or implant loosening. Instability may ultimately contribute as a result of implant loosening or failure due to infection or the lack of healing within the



(a) (b)

Fig. 13.8 (a) Craniocaudal radiograph of a 5-year-old Lurcher's femur taken after plating of a short, oblique, diaphyseal fracture. The two fragments should have been reduced and compressed using either a plate or a lag screw (which would then be protected by application of a plate in neutralisation mode). Instead the plate has been applied leaving a gap at the fracture site and is, therefore, functioning as a buttress plate. This is a common mistake and creates considerable stress at a specific point in the plate.
(b) Craniocaudal radiograph of the same femur taken 4 weeks later. Collapse of the medial side of the fracture and bending of the plate through the screw hole closest to the fracture is evident. This plate may well have been of sufficient size to act as a neutralisation plate but not as a buttress plate.

functional lifespan of the implants used (clinical success is always a race between fracture healing and implant failure!).

Treatment involves resection of the obstruction, packing of the fracture gap with cancellous bone and stabilisation of the site with a bone plate, interlocking nail or ESF (Fig. 13.12b). An alternative to this method would involve bone transport, whereby an osteotomy is created at a distance fom the fracture gap and the segment of bone created is transported using a 'dynamic' fixator so as to close the fracture gap and allow distraction osteogenesis within the osteotomy gap. Such techniques are rarely required in small animals. In those cases where infection is also a complicating feature, the management may have to be modified as outlined below under 'Osteomyelitis'.

In cases with atrophic non-union the same principles apply since resection of the fibrous tissue from the gradually increasing fracture gap will leave a defect needing to be packed with cancellous bone. However, the receding of the bone ends may continue such that the implants used to bridge the fracture gap eventually lose purchase and the site becomes unstable once again. In such cases amputation is an appropriate treatment option.

Local ischaemia

Poor vascularity at a fracture site may be an inherent feature, as is believed to be the case in fractures of the distal third of the radius/ulna in toy breeds, or a result of excessive stripping of soft tissues from bone during fracture repair, i.e. poor surgical technique. In either case, once non-union has resulted there is little to be done except by optimising the opportunity for neovascularisation

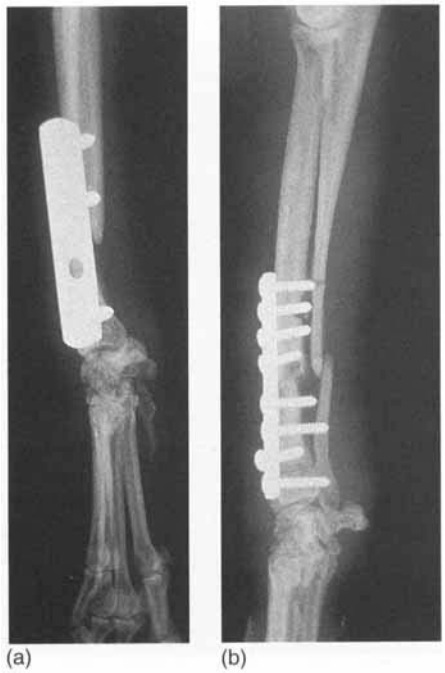


Fig. 13.9 (a) Mediolateral radiograph of a 4-year-old Toy Poodle's antebrachium 6 weeks after sustaining a transverse fracture of the radius and ulna. Initially the injury had been treated by casting. Six weeks later the site remained unstable and so was plated. Only one screw has been secured into the distal fragment and 4 weeks later the site was again unstable.
(b) Mediolateral radiograph of the same dog now 10 weeks after the injury. The inadequate plate has been removed, the site debrided and packed with a cancellous bone graft, and a mini-DCP (dynamic compression plate) applied to the radius with four screws on each side of the fracture.

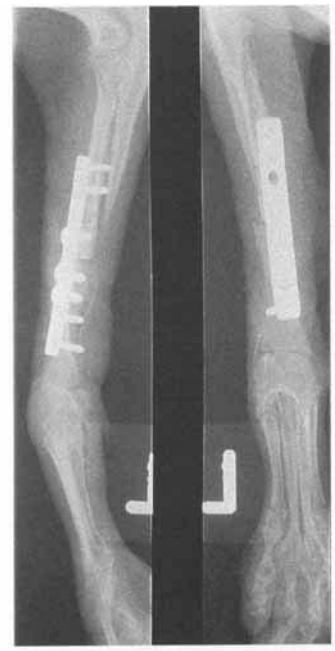


Fig. 13.10 *Mediolateral and craniocaudal radiographs of the* same dog as in Fig. 13.2. The radial non-union was debrided and compressed using a mini-DCP (dynamic compression plate).

by stabilising the site whilst causing as little further soft tissue damage as possible. In the future it may become possible to stimulate angiogenesis by applying chemical mediators (cytokines) to the site but none are currently readily available.

Systemic or local disease

If systemic disease such as nutritional secondary hyperparathyroidism or hyperadrenocorticism (Cushing's disease) is thought to be associated with non-union of a fracture, then the disease process may require appropriate management before successful healing of the fracture can be achieved. In the case of local disease, the pathology will often be neoplastic in which case the prognosis is even more guarded and the treatment options are as outlined in Chapter 51.

Prognosis

With appropriate management most cases of delayed union will heal and attain satisfactory or normal limb function. Similarly, most cases of non-union will have a satisfactory outcome if managed along the lines detailed above, with those caused by instability having the best prognosis, those requiring resection of tissue and bridging osteosynthesis having an intermediate prognosis, and those with an atrophic non-union having a very guarded prognosis. However, with many non-unions, limb function is not as good as if uncomplicated fracture healing had been achieved and so, as with all fracture complications, they are better avoided than treated.

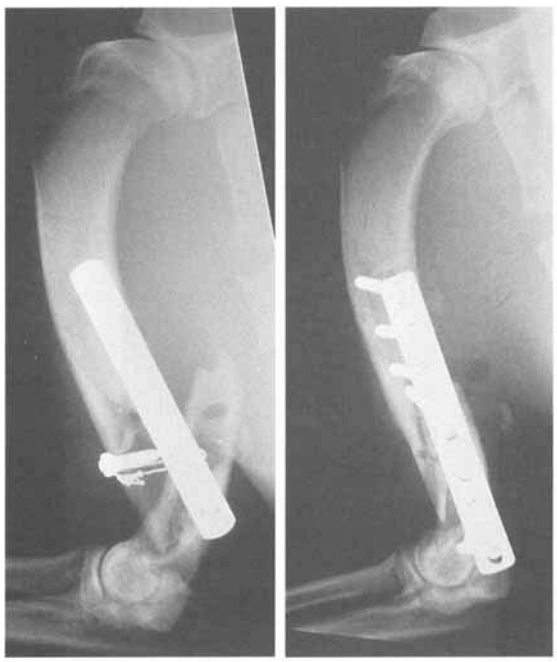


Fig. 13.11 (a) Mediolateral radiograph of a 10-month-old Boxer's humerus taken 2 weeks after an oblique/spiral fracture had been treated by open reduction and fixation. Although the reduction on the postoperative radiograph looked good and a lag screw had been used to create compression between the fragments, the neutralisation plate had only one screw gaining good purchase in the distal fragment. In addition, the cerclage technique has been misused in that suture wire, rather than orthopaedic wire, was used and the wire has been placed around the plate (cerclage wire can only compress fragments before they are 'fixed' with a bone plate). Implant failure is clearly apparent.
(b) Mediolateral radiograph of the same humerus after surgery to remove the failed implants and apply a more suitable bone plate with adequate plate screw fixation on either side of the fracture. Fragment reconstruction is often precluded by the presence of early callus at this stage and so the plate has been applied in buttress fashion.

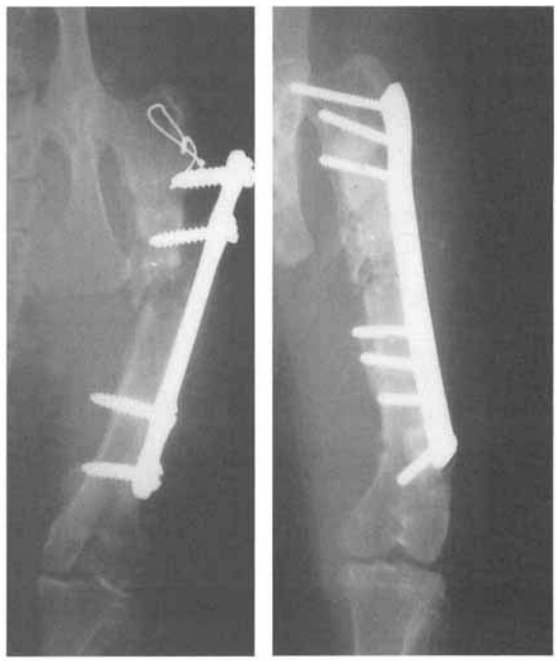


Fig. 13.12 (a) Craniocaudal radiograph of a 3-year-old Domestic Short Haired cat's femur taken 26 months after treatment of fractures sustained in a road traffic accident. An intramedullary pin and cerclage wires were used initially but although the comminuted proximal region healed, a transverse non-union remained 3 months later. The pin and wires were removed and a plate was applied to the lateral aspect. Lameness then recurred and this radiograph was taken. Healing has not taken place and implant failure has occurred by the proximal screws pulling out of the proximal bone (presumably weak through being comminuted originally). Some of the original fragments remain isolated at the fracture site and the non-union may be considered 'necrotic'.
(b) Craniocaudal radiograph of the same limb taken after revision surgery. The plate has been removed, the necrotic bone debrided, the fracture space packed with corticocancellous bone from the iliac wing and the fracture site 'bridged' with a contoured 2.7 mm dynamic compression plate (DCP).

Osteomyelitis

Although bone may become infected by way of haematogenous spread from another focus elsewhere in the body, most cases of osteomyelitis in small animals are associated with fracture of the bone and, unfortunately, the majority of these animals have had closed fractures treated by open reduction and fixation. In one study (Caywood *et al.*, 1978) it was estimated that in 58% of cases with osteomyelitis (not just fracture-related) the blame could be laid squarely at the feet of poor surgical techinque during open reduction and fixation of a fracture. A difference in incidence of osteomyelitis between dogs and bitches (approximately 2:1) and between different bones in the skeleton (Caywood *et al.*, 1978) may be, to some extent, a reflection of the comparative incidence of fractures in relation to gender or skeletal location (evidence cited by Braden, 1991; Ness *et al.*,

1996) and the likelihood of the fracture being open in relation to its anatomical location.

Pathogenesis

The pathogenesis is outlined in Fig. 13.13. Essentially three factors are required to allow osteomyelitis to become established at a fracture site: an infected wound, an environment conducive to bacterial multiplication, and avascular bone. Sequestra are avascular pieces of bone which appear radiodense compared to the surrounding bone and are surrounded by granulation tissue which fills a 'hole' in the bone referred to as an involucrum. Such sequestra may act as a nidus for infection. Fracture healing will take place in the presence of infection as long as stability is maintained but, as discussed above, infection is one cause of implant loosening and the resulting instability may then lead on to non-union. If osteomyelitis becomes established at a fracture site then, even if healing takes place, the infective agent will not be eliminated unless all foreign material, including implants, and any bone sequestra are removed.

Clinical signs

The signs associated with osteomyelitis may take two forms: those associated with acute infection and those with chronic infection. In the acute form the animal will show malaise, inappetance and pyrexia whilst the fracture site will show the cardinal signs of inflammation (heat, pain, swelling and reddening). In the chronic form, the systemic effects are usually not realised until after successful treatment when the animal is often considered to be 'more his/her old self' again. The local effects of the chronic state include recurrent discharging sinuses (Fig. 13.14) and/or lameness.

Radiology

This is very useful in confirming a suspicion of osteomyelitis but it must always be remembered that radiographic change may lag behind the clinical signs by up to 4–6 weeks. Features which may be noted include (Fig. 13.15):

- Soft tissue swelling (acute form)
- Bone lysis (especially around implants)
- Irregular periosteal reaction

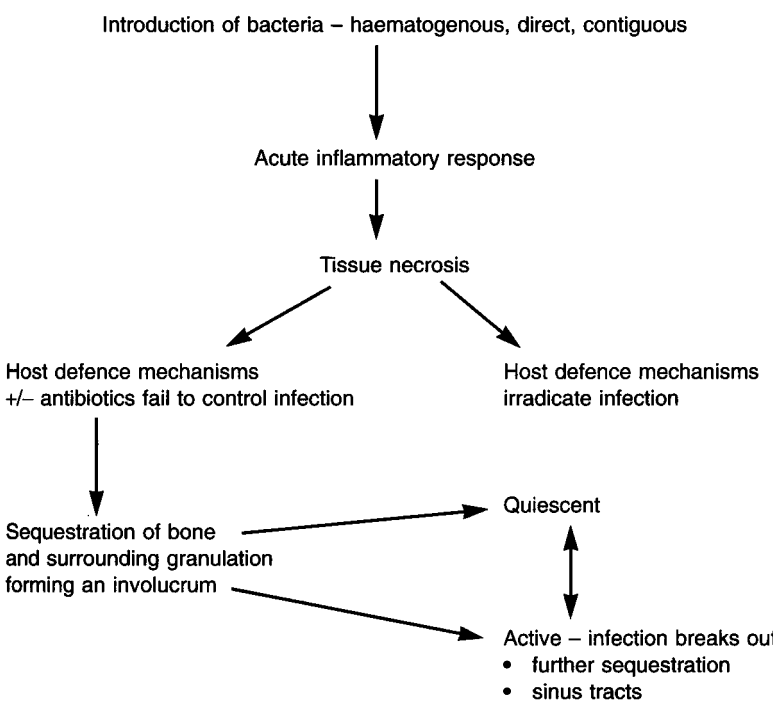


Fig. 13.13 The pathogenesis of osteomyelitis.

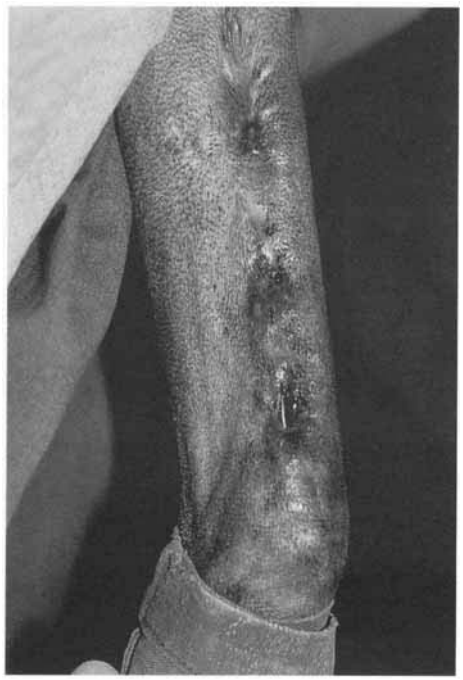


Fig. 13.14 View of a 4-year-old Collie's antebrachium taken 2 years after repair of a radius/ulna fracture by application of a bone plate. Discharging sinuses had been present since soon after the original treatment, with temporary response to antibiotic treatment. Several sinuses are present and through the most distal of these the plate can be seen. Fracture union was complete and removal of the plate, together with debridement of the sinuses, allowed the problem to resolve.

- Increased density of surrounding bone
- Sequestrum/involucrum formation (chronic form)

Treatment

The treatment of osteomyelitis associated with fracture management has to address a number of aspects as detailed below.

Infection

Antimicrobial agents will be required to eliminate the infection and/or control it until such time as fracture healing is complete and the implants can be removed. Since bacteria are the organisms most often involved these agents will usually be antibiotics. In most cases, such antibiotics are administered systemically but in a few it may be necessary to consider the use of local administration, for example gentamicin-impregnated polymethylmethacrylate beads (Septopal, E. Merck). In cases where there is an open wound then topical treatment may be achieved by regular flushing with a suitable antibacterial preparation.

With respect to choice of antibiotic, it is best to base this upon laboratory culture from swabs taken from either needle aspirates of the fracture site or directly at the time of any revision surgery. Taking swabs from a discharging sinus is non-informative as any bacteria cultured will reflect the commensal population on the surface rather than the pathological population within. Whilst awaiting the laboratory results, or if nothing is cultured but osteomyelitis is still suspected, it is most appropriate to use a broad-spectrum, bacteriocidal antibiotic such as a cephalosporin (e.g. Ceporex, Schering-Plough Animal Health), a clavulonic acid potentiated penicillin (e.g. Synulox, Pfizer) or clindamycin (Antirobe, Pharmacia & Upjohn). In general, and at least until the infection is brought under control, the authors recommend the use of the latter in combination with one of the first two drugs. Antibiotic treatment should continue for at least 4–6 weeks and 1 week beyond the removal of implants and/or surgical debridement of the site.

Fracture stability

If the fracture site remains stable then healing will continue whilst the infection is controlled, as described above. Ultimately, any implants at the fracture site will need to be removed, once healing is complete, before the infection will be eliminated.

If the infection has caused implant failure then stability has to be regained, otherwise healing will not take place. In general, in the distal long bones this is achieved by removal of the original implants and stabilising the bone using an ESF, so as to avoid the need for implants at the fracture site. In the upper limb a plate may have to be used despite concern over further surgical dissection and the presence of implants at the fracture site. At the time of removal of the original implants it is also necessary to remove any necrotic tissue, including devitalised bone, from the fracture site.

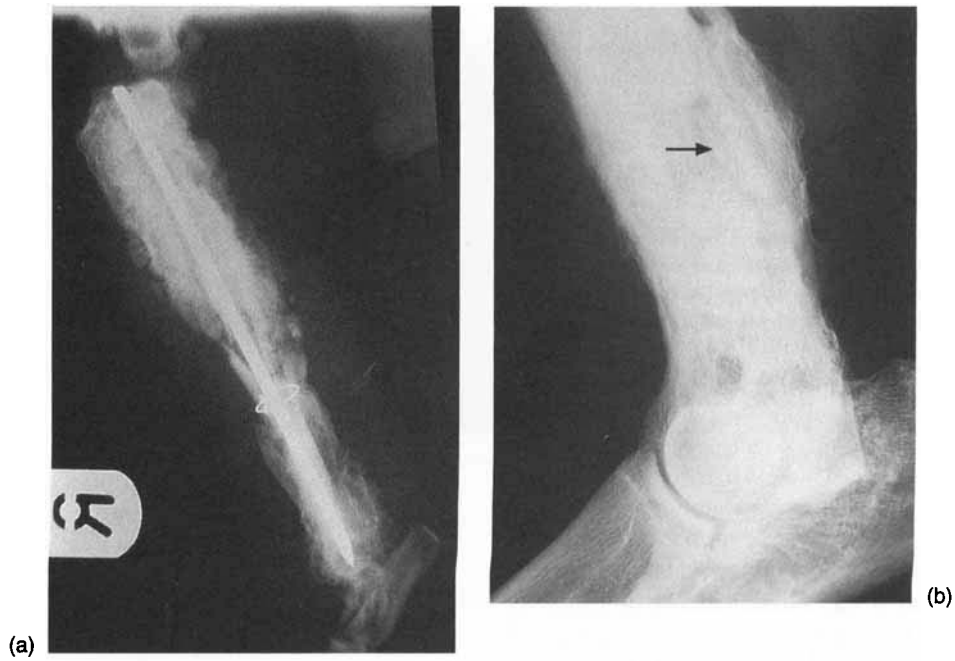


Fig. 13.15 (a) Mediolateral radiograph of a young adult Terrier's tibia 1 month after treatment of a comminuted fracture by intramedullary pinning. Extensive periosteal reaction is present throughout the length of the bone with the exception of the fracture site. A diagnosis of osteomyelitis was made and confirmed by culture of aspirates from the site. Treatment involved amputation due, in part, to financial constraints.
(b) Mediolateral radiograph of a 7-year-old German Shepherd Dog's distal humerus taken 4 years after internal fixation of a 'Y'-fracture. Discharging sinuses had appeared intermittently since that time with only temporary improvement with antibiotic treatment. Removal of all the implants had not resolved the problem. At the level of the greatest periosteal bone formation there are two linear densities lying within involucra (arrow maks the most obvious one) and a diagnosis of chronic osteomyelitis associated with persistent sequestra was made.

It may also be considered prudent to pack the site with a cancellous bone graft, either at this time or after a delay of 7–14 days whilst control of the infection is established (although cancellous bone can survive in the presence of infection).

Implants

As has been mentioned above, implants will have to be replaced if they are not stabilising the fracture adequately and will normally have to be removed, once fracture healing is complete, to allow the infection to be eliminated.

Sequestra

An avascular fragment of bone, or sequestrum, may act as a nidus for infection which will then not be eliminated without removing these fragments. In planning such surgery it is also necessary to consider dissecting out the sinus tracts since the fibrous tissue surrounding these may also harbour infection (Fig. 13.16). Identification of the tracts leading to the sequstra can be aided by local infiltration with a 1% solution of methylene blue 12–24 hours before surgery. Avascular bone can also be readily identified alongside vital bone by the intravenous injection of disulphine blue 1 hour before surgery since the avascular bone is the only tissue which does not take on a bluish hue.

Removal of the sequestra may weaken the bone, in which case it may be necessary to pack the defect with a cancellous bone graft and protect the bone from refracturing by application of an ESF.

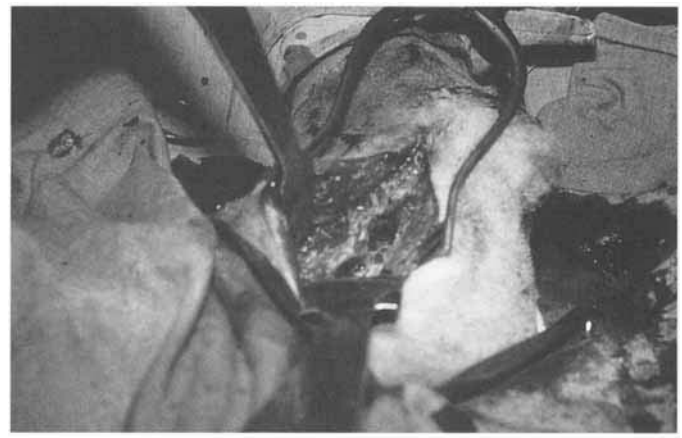


Fig. 13.16 Intraoperative view of the same dog as in Fig. 13.15b. The cavernous nature of the healed fracture site is apparent and two sequestra were removed from the site (one is sitting on the drape to the far left of the wound). No more sinuses developed subsequently but a degree of lameness persisted due to soft tissue adhesions, prolonged immobility, etc.

Prognosis

The prognosis in cases with fracture-associated osteomyelitis is variable depending on the severity of infection and requirements of treatment. In some cases it is low grade and only requires that antibiotic treatment be continued until a few weeks after fracture healing is complete and the implants used have been removed. Such cases may achieve a level of limb function close to that expected after non-infected fracture healing. However, in other cases the infection may cause considerable interference with soft tissue function due to adhesions caused by: local inflammation, sinus formation, and repeated surgeries to remove implants or sequestra. The outlook in these animals is not good and reduced limb function should be expected. Indeed, in some cases the recurrent sinuses cannot be halted with the resulting options of permanent antimicrobial therapy or amputation.

Prevention

Although the outcome of osteomyelitis associated with fracture management may be satisfactory, it is not always so and will definitely require more monitoring and cost than management of the same fracture without infection. As a result, it is best to prevent wound contamination and/or infection becoming established. The management of open fractures is discussed in Chapter 14 and the treatment of contaminated wounds so as to reduce the likelihood of them becoming infected (i.e. the number of colony forming units per gram of tissue exceeding 10^5) will not be discussed further here.

The prevention of 'iatrogenic' osteomyelitis essentially comes down to good theatre practice together with the administration of prophylactic antibiotics. Use of the latter, however, *is not a substitute* for aseptic technique. The fundamental principles of good theatre technique are well described and illustrated by Piermattei (1993) and can be summarised as:

- *Operating theatre*:
  - clean environment
  - minimise 'theatre traffic'
  - ventilation system (optimal is filtered, positive pressure system)
- *Patient preparation*:
  - clip adequate area
  - thorough skin preparation
  - draping of both the field and then the surgical wound (with either an adhesive film or by suturing/clipping the drape edge to the wound edge) using an impermeable material
- *Surgeon preparation*:
  - wear hat and mask
  - adequate hand scrub
  - wear long-sleeved, sterile gown and surgical gloves
- *Surgical technique*:
  - aseptic technique
  - careful handling of soft tissues (to minimise further vascular damage)

- minimise operating time (wounds open for longer than 1 hour begin to show an exponential rise in the incidence of infection) – assistant useful in this respect

Although prophylactic antibiotics are justifiable as an *additional* measure to all of the above, it is important that they are used perioperatively rather than postoperatively. Assuming that only the exceptional wound or operating environment will prevent all contamination, it is more effective for the antibiotic to be present when the contaminants arrive than after they are beginning to become established. Also, the postoperative fracture haematoma will contain antibiotic if this is administered perioperatively whereas diffusion of drugs administered postoperatively into the haematoma is less predictable. An intravenous bolus of antibiotic can be used just after induction of anaesthesia (though this usually requires the use of a human preparation [for example, Kefzol, Eli Lilly & Company, which is a cephalosporin] since there are no currently licensed products in small animals that are suitable for this purpose) or else systemic treatment can be introduced several hours before surgery. As far as choosing a prophylactic antibiotic, apart from it needing to be broad spectrum and bacteriocidal, this is based on surgeon's preference and experience of what works in that particular clinic. A scientifically based decision can be made by leaving agar plates out overnight and during use of the operating theatre. Culture of these plates will help to establish the resident population in the environment and sensitivity tests will determine an appropriate choice of antibiotic. However, it is pertinent to remember that *Staphylococcus* spp. are isolated from nearly half the cases of canine osteomyelitis (Caywood *et al.*, 1978) and it is likely that the source of these organisms is the dog's own skin.

Malunion

When fractured fragments of bone heal in a non-anatomical orientation then malunion results. Such malunion may create an angular or rotational deformity or a shortening of the bone. In the case of the pelvis, malunion may create narrowing of the pelvic canal. Treatment of such complications following pelvic fractures is detailed in Chapter 39 and will not be discussed further here. Except in cases with simple fractures treated by anatomical reduction and rigid internal fixation, a degree of malunion is the rule rather than the exception. This has led to the adoption of terms such as functional (or insignificant) and non-functional (or significant) malunions. A degree of angular deformity is easily accommodated at fracture sites involving the mid-diaphysis but there is less tolerance of this as the site moves towards the metaphyses since joint alignment becomes affected. Limited rotational deformity can be accommodated in a limb by alteration of posture. Because dogs and cats walk with their major joints in flexion, they have a considerable ability to cope with shortening of a bone. Therefore, a certain degree of malunion is acceptable since it will not affect limb function, i.e. would be classed as a functional malunion (Fig. 13.17). Such malunions

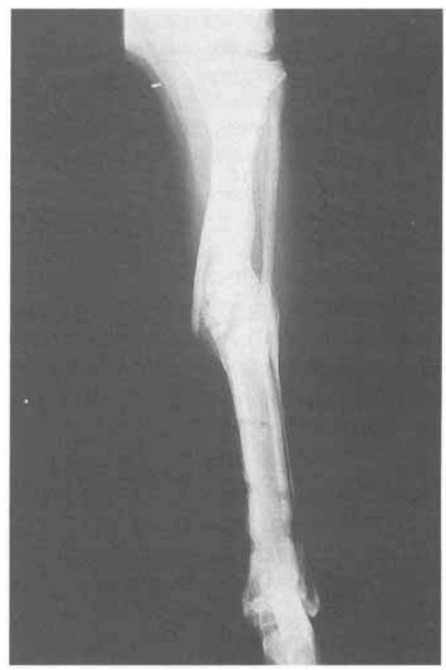


Fig. 13.17 Craniocaudal radiograph of a 7-year-old Lurcher's tibia taken 4 months after treatment of an open, comminuted fracture by application of a bilateral, uniplanar external skeletal fixator (ESF). The fixator was applied in a closed fashion with the limb held so as to regain limb length and joint alignment. A malunion has resulted but is fairly mild, located mid-diaphysis and did not prevent full limb function. It can, therefore, be described as 'functional'.

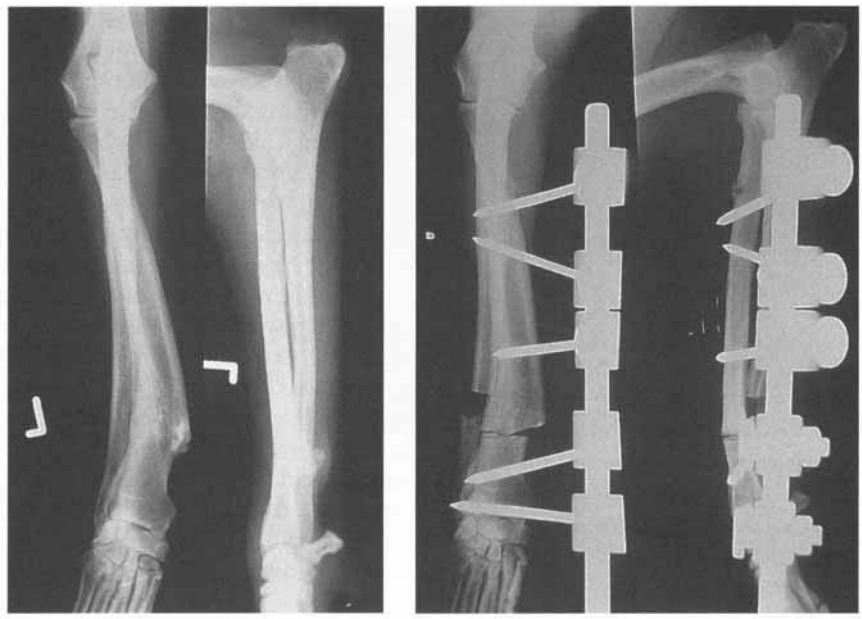


Fig. 13.18 (a) Mediolateral and craniocaudal radiographs of a 2-year-old Border Collie's antebrachium showing malunion of distal fractures of the radius and ulna creating a marked valgus deformity. The degree of visible deformity means that this can be described as a 'non-functional' malunion.
(b) Mediolateral and craniocaudal radiographs of the same limb after corrective osteotomy. A segment of ulna has been removed so as not to interfere with manipulation of the radius. A closing wedge osteotomy of the radius has been created and stabilised with a five-pin, unilateral, uniplanar external skeletal fixator (ESF).

do not require treatment. However, in cases where the deformity is so great that it affects joint alignment and/or limb function then it would be classed as a non-functional malunion (Fig. 13.18) and treatment options would have to be considered. In addition, any displaced fracture involving an articular surface will form either a non-functional malunion (Fig. 13.19) or a non-union. However, the presence of a non-functional malunion does not preclude some, or even reasonable, limb function but it does preclude normal limb use.

Treatment

As has already been stated, functional malunions do not require treatment (Fig. 13.17) and in some non-functional malunions surgical correction is likely to be problematic and unlikely to benefit the patient (Fig. 13.19). In addition, it must be remembered that in some cases the malunion may have been accommodated functionally, and correcting it some time after it formed may actually worsen the limb function rather than improve it. However, some non-functional malunions, particularly those causing lameness due to the angular or rotational deformity created, may be considered surgical candidates and such treatment involves corrective osteotomy. In the case of a rotational deformity, a transverse osteotomy allows 'derotation' of the distal fragment relative to the proximal fragment and stabilisation involves application of a bone plate or ESF. Angular deformity is the commonest malunion to be treated by corrective osteotomy. There are several types of osteotomy that can be used (as discussed in Chapter 34 and Figs 34.15 to 34.17) but in general the types used by the authors are detailed in Table 13.1. An example of correction of a malunion is shown in Fig. 13.18.

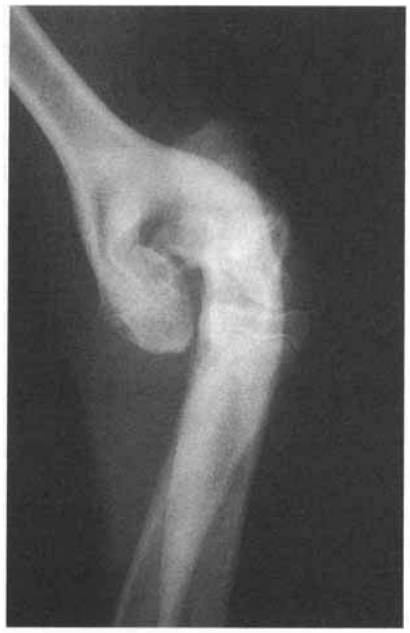


Fig. 13.19 Craniocaudal radiograph of a 5-month-old Springer Spaniel's elbow taken after the dog had shown persistent forelimb lameness after a minor fall 2 months previously. The deformity seen is most likely to be a result of malunion of a moderately displaced lateral condylar fracture, especially when the age and breed of dog are taken into consideration. Because there is deformity of the joint surface the malunion is 'non-functional' and corrective surgery is not advisable. Limited limb function could be expected in the long term and the only surgical option would be arthrodesis.

Prognosis

Functional malunions have a good prognosis whereas non-functional malunions have a variable prognosis depending on:

- To what extent they impair limb function
- Whether they lead to the development of degenerative pathology in adjacent joints
- Whether corrective surgery is (a) possible and (b) undertaken

Fracture disease

This term is used to describe a syndrome associated with the following changes:

- Muscle wastage (+/– adhesions)
- Joint stiffness
- Osteoporosis

In general, these changes result from disuse or immobilisation of a limb during fracture management. The effect of immobilisation, with or without trauma, results in atrophic changes in (Anderson, 1991):

- Bone (disuse osteoporosis)
- Muscle (wastage +/– contracture)
- Ligament
- Articular cartilage
- Synovium
- Soft tissue adhesions – only after trauma

Table 13.1 Corrective osteotomies for the treatment of malunion.

Deformity	Type of osteotomy	Fixation method
Lateral or medial bowing of the radius	Closing wedge Oblique	Cranial plate ESF
Lateral bowing of the femur	Closing wedge	Lateral plate
Medial bowing of the femur	Open wedge	Lateral plate
Lateral bowing of the tibia	Open wedge	Medial plate ESF +/– I/M pin
Medial bowing of the tibia	Closing wedge Oblique	Medial plate ESF

ESF = external skeletal fixator; I/M = intramedullary.

The severity and reversibility of these changes are influenced by such factors as the age of the patient (Hall, 1963; Jaworski *et al.*, 1980) and duration of the immobility (Akeson *et al.*, 1980). It has also been shown by several workers, including Keller *et al.* (1994), that a joint may be protected, to an extent, from some of the effects of immobilisation, or else the rate of improvement increased post-immobilisation, by the intra-articular administration of agents such as hyaluronic acid. Whether such findings would be likely to help in the management of clinical cases has not yet been determined.

Overall, from the research reviewed by Anderson (1991) it may be concluded that limb function will reach normality within an acceptable period of time if the duration of immobility/disuse is in the region of 6 weeks, particularly in the young patient.

Treatment

When the management of a fracture has involved immobilising the limb for 4–6 weeks the signs will usually resolve within a few weeks or months once the limb is weight-bearing again. In cases where joint stiffness is particularly notable, physiotherapy may be of help in making recovery quicker and more complete. In general, it is those cases where soft tissue adhesions or contractures have occurred that present significant problems (Fig. 13.20). Although physiotherapy, using modalities such as heat therapy, cold therapy, ultrasound or hydrotherapy, is the main feature in the management of such cases, a return of normal function should not be expected and considerable effort will be required to achieve any limb function in these severe cases. In those where discrete contractures can be identified, there may be a way of improving joint and/or limb function by tenotomy coupled with physiotherapy or arthrodesis (see Chapter 41, p. 504 for details of quadriceps contracture).

Prevention

The prognosis for cases showing significant fracture disease is often poor and so it is most important to consider how this complication can be avoided:

(a) (b)

Fig. 13.20 (a) Mediolateral radiograph of a 2-year-old Lurcher's humerus taken 3 months after treatment of a diaphyseal fracture. Initially an intramedullary pin had been used to stabilise the site but this migrated proximally and was removed 2 weeks later. Since then the limb had been strapped to the chest wall. A hypertrophic non-union is evident. Treatment involved application of compression, using a dynamic compression plate (DCP), across the fracture site.
(b) Clinical appearance of the same dog 4 months after plating of the humerus. The fracture had healed but fracture disease in the form of soft tissue contractures causing reduced extension of the elbow and carpus continued to prevent weight-bearing. These contractures must be considered a direct result of strapping the limb, in flexion, to the chest wall for a prolonged period. Limited limb function was regained by sectioning of the carpal and digital flexor tendons, arthrodesis of the carpus and physiotherapy on the elbow joint.

(1) Wherever possible normal (or controlled) limb function should be maintained during fracture healing by 'splinting' of the bone rather than the limb, i.e. the aim is to achieve a mobile limb with an immobile fracture site.
(2) If casting a limb is felt to be appropriate to manage a fracture then it should be removed as soon as clinical union is achieved and preferably within 6 weeks (see Chapter 12, p. 92 for the indications relating to application of a cast).
(3) Whenever internal fixation is used, the bone should load share with the implants wherever possible so as to reduce any tendency towards disuse osteoporosis.
(4) Careful attention to the principles of fracture management, in terms of methodology and technique, will help to minimise morbidity resulting from fracture disease.

Fracture-associated sarcoma

Although this condition is of great interest there are less than about 100 cases documented in the literature and the general features of such cases were reviewed by Stevenson (1991). Although most reports involve dogs, the condition has been reported in the cat (Fry & Jukes, 1995). Most commonly, clinical signs develop more than 5 years after primary treatment of the fracture and most of the fractures involved occurred when the dogs were between 1 and 3 years of age. In some cases the lag period for the development of a sarcoma is much less (6–9 months), and it must then be considered that the fracture might well have been a result of pathology in the bone rather than a forerunner to its development. Large breeds of dog are more commonly affected than the smaller breeds. At the time of sarcoma development the dogs show lameness or a mass that is gradually increasing in size. Radiographs show changes typically associated with bone tumours, i.e. a mixture of osteolysis, osteoproduction and soft tissue mineralisation (Fig. 13.21). They tend to affect the diaphyses rather than the metaphyses, and show a difference in relative incidence between the various long bones, when compared to primary bone tumours (as detailed in Table 13.2). The aetiology of these tumours is not understood and although there are hypotheses regarding factors which might contribute to their development, none of these have been proven.

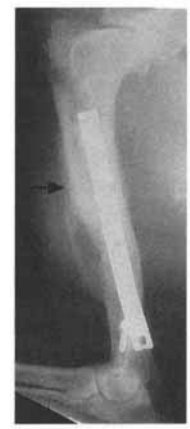


Fig. 13.21 Mediolateral radiograph of the humerus of the same dog as in Fig. 13.20 taken 4 years later. An acute deterioration in limb function was associated with the development of a painful swelling in the proximal limb. The radiograph shows periosteal new bone formation around the humerus associated with the proximal half of the plate (arrow). A fracture-associated sarcoma was diagnosed.

Table 13.2 The incidence of primary bone tumours and fracture-associated tumours in relation to the long bone concerned (Stevenson, 1991).

Long bone	Relative incidence (%)	
	Primary bone tumour	Fracture-associated tumour
Humerus	20	24
Femur	16	49
Radius	39	22
Tibia	25	5

Treatment

Treatment of these tumours follows the same principles as those outlined in Chapter 51, and the prognosis is similarly guarded to hopeless. Because the majority involve proximal limb bones, and they tend to involve the surrounding soft tissues even more than with primary osteosarcomata, it is far less likely that limb-sparing procedures could even be considered. Treatment, therefore, tends to be symptomatic or else involve amputation +/– chemotherapy to try and control the metastases.

Prevention

The aetiology of these tumours is poorly understood and is probably multifactorial which makes specific measures to avoid their occurrence difficult to identify. Furthermore, their low incidence in small animals creates a certain inertia in developing such strategies. The removal of implants after healing is complete may reduce the likelihood of a sarcoma developing. However, some cases with this problem have not had implants used at all and it is always difficult to balance the cost and risks of implant removal against the benefits in terms of an unknown reduction in the incidence of a rare complication. Although sarcomata may develop at fracture sites where healing has been uncomplicated (Fry & Jukes, 1995), most cases are associated with problematic healing of the original fracture (for example, a delay in union resulting from instability or infection). It may be concluded from this that the best way to avoid sarcoma formation is to achieve uncomplicated fracture healing.

Fat embolism

Two cases of fat embolism in the dog were reported by Furneaux (1974) and both had recently undergone internal fixation of hindlimb fractures. Both died acutely, one 5 days after treatment of a femoral fracture and the other 3 days after treatment of a pelvic and a femoral fracture. It would appear that such cases do not show the pulmonary signs associated with fat embolism seen in humans, for example following total hip replacement, and death seems to be associated with emboli in the central nervous system. Furneaux also stated that in their clinic a higher rate of postoperative mortality had been noted following skeletal surgery compared with general surgery and that the highest incidence was associated with treatment of fractures involving the pelvis. The problem would appear, fortunately, to be of low incidence in small animal surgery and it has not been considered necessary to undertake prophylactic measures. Since the clinical signs are acute death, there is no treatment.

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Chapter 14
Open Fractures

An open fracture is one in which there is a communication between the fracture site and a skin wound. Open fractures may be classified as first, second or third degree according to the severity of tissue injury and contamination (Fig. 14.1).

Basic principles of treatment of open fractures

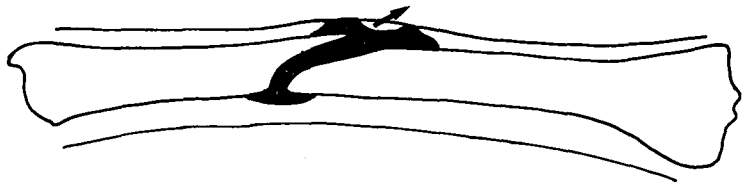
Normal periosteum provides bone with an adequate defence against invading organisms, but when this protection is removed, as a result of a fracture or surgery, then the bone becomes extremely susceptible to infection (Peacock & Van Winckle, 1970). Primary bacterial infection occurs in only about a third of open fractures and any necrotic tissue which is left in the wound serves as a nidus for bacterial multiplication. Secondary bacterial infection occurs after 6–8 hours (Muller *et al.*, 1970).

First- and second-degree wounds treated within 6–8 hours may be managed by primary closure of the wound following debridement. This period is known as the 'golden period' and is thought to be the time taken after injury and contamination before infection becomes established (defined as a presence of microorganisms in excess of 1×10^5 colony-forming units per gram of tissue). When the open fracture is over 8 hours old or there is a severe second- or third-degree wound, then, following a thorough debridement and excision, the wound is left open and covered with a protective bandage. Secondary closure can then be carried out at a later date once any infection has been eliminated. Infected wounds are treated as open wounds and allowed to heal by second intention.

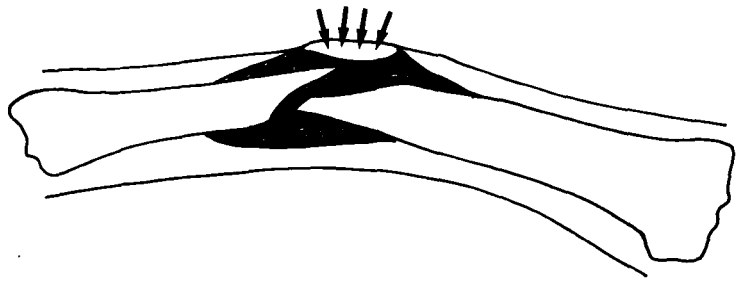
Practical management of open fractures

Open fractures are regarded as contaminated and, therefore, potentially infected. Second- and third-degree open fractures are more likely to become infected. These injuries should be treated as an emergency. First-aid management of the wound involves covering it with a sterile dressing, applying a pressure bandage and using external coaptation when possible. Radiographs are taken to assess the extent of the fracture and decisions can then be made on the method of fixation that will be used. First-degree open fractures can be treated in the same way as closed fractures using standard methods of fracture fixation after thorough wound cleaning. However, second- (Fig. 14.2a) and third-degree open fractures are, generally, stabilised using an external fixator.

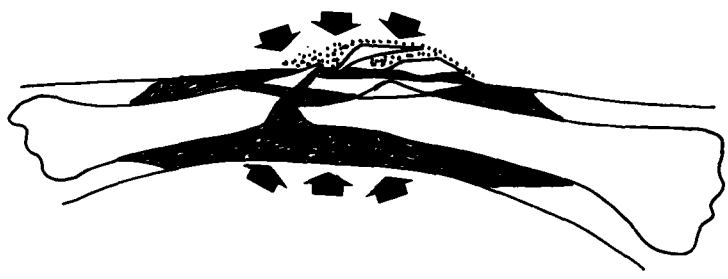
Wound infection is eliminated by wound cleansing and debridement, and prompt antiobiotic therapy. Systemic administration of antibiotics is essential to achieve adequate concentrations in the fracture haematoma. The antibiotic is given, initially, as a single intravenous bolus as soon as possible after the injury (within hours) to prevent bacterial proliferation. Swabs should be taken from the wound prior to this for culture and sensitivity testing. The most commonly used antibiotic is a cephalosporin (Ceporex, Schering-Plough Animal Health, at 10 mg/kg twice daily preceded by the intravenous administration of 10 mg/kg Kefzol, Eli Lilly & Co). This antibiotic is bacteriocidal, broad spectrum, and is especially useful for staphylococcal infections. It also achieves good levels in muscle and bone. The incidence of Gram-negative infections is higher in second- and third-degree open fractures and here



(a) First-degree open fracture – skin penetration (arrow) from within.



(b) Second-degree open fracture – skin penetration (arrows) from outside, more extensive soft tissue injury and contamination.



(c) Third-degree open fracture – gross contamination, bone loss and extensive tissue damage.

Figure 14.1 Classification of open fractures.

a cephalosporin can be combined with an aminoglycoside, e.g. gentamicin (Gentovet, Arnolds, at 2–5 mg/kg bodyweight/day divided into three equal doses), or clindamycin (Antirobe, Pharmacia & Upjohn, at 11 mg/kg bodyweight twice daily).

Antibiotics are no substitute for adequate wound debridement. In the preparation room any protective wound dressing is removed and swabs are taken from the wound for aerobic and anaerobic culture. K-Y Jelly (Johnson & Johnson) is applied to the wound surfaces to prevent contamination with hair and debris while the surrounding skin is clipped and the whole area is prepared aseptically with dilute iodine solution (Betadine Surgical Scrub, Napp Laboratories). Gross debris is removed from the wound and initial lavage carried out by flushing the wound surfaces using lactated Ringer's solution or Hartmann's solution and a syringe. The dog is then transferred to the operating theatre and sterile drapes are applied around the wound. Irrigation of the wound is continued with copious amounts of fluid. Water Pik-type cleansing is useful but if the system is not available a large syringe, +/– wide-bore hypodermic needle, can be used to flush out the wound. Final irrigation can be done using an antibacterial solution (e.g. neomycin–polymyxin and 50000 units of bacitracin per litre of Ringer's solution). Alternatively, the wound can be irrigated with dilute povidine

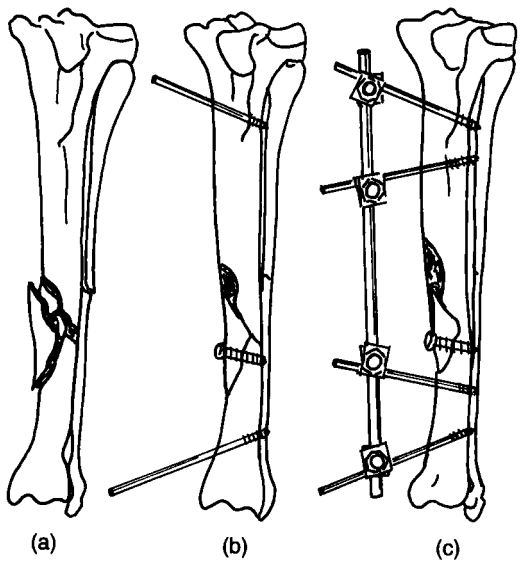


Figure 14.2 (a) Second-degree open fracture of the tibia. (b) Wound debrided, small fragment removed, large fragment fixed with lag screw. (c) External fixator used to stabilise the fracture.

iodine solution (1 in 10). All non-viable tissue should be removed from the wound (if the tissue bleeds it is viable!). All large bone fragments which can be rigidly fixed (lag screw fixation) should remain but smaller fragments, which cannot be fixed, especially those which have lost their soft tissue attachments, are removed (Fig. 14.2b). The fracture should then be rigidly stabilised and, as a general rule, the safest way of doing this is by the use of an external fixator, placing the pins in healthy bone at some distance from the fracture site (Fig. 14.2c). Primary wound closure should be avoided if the wound is over 6–8 hours old as this causes a significant rise in infection rates. Instead, the wound is left open and protected with a bandage. Once the wound appears to be filling with healthy granulation tissue, and is no longer discharging, then secondary closure can be safely undertaken if deemed necessary. One of the advantages of the external fixator in the management of open fractures is that it allows easy access for treatment of open wounds.

Prolonged antibiotic cover does not appear to reduce wound infection rates and consequently antibiotics are often only given for 5–7 days following primary or secondary closure.

Bone grafting and open fractures

Bone grafting is sometimes considered necessary because of bone loss through the wound. Autogenous cancellous bone grafts can be used at the time of surgery or, if the wound vascularity is questionable, grafting can be delayed for a week or two until healthy granulation tissue is filling the wound. If the cancellous bone graft cannot be covered with soft tissue it is protected with Vaseline-impregnated gauze (e.g. Jelonet, Smith & Nephew) and a bandage.

Autogenous cancellous bone grafts can survive in the presence of infection whereas cortical bone grafts do not because they are slow to revascularise and will tend to sequestrate in the presence of infection. If cortical grafting is indicated this should be delayed until all infection has been eliminated from the fracture site.

Further details relating to bone grafting techniques can be found in Chapter 2.

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Chapter 15
Treating Fractures in Immature Patients

The same general principles of fracture management that are applied to skeletally mature patients also apply to immature patients. However, there are certain considerations in the young patient that do not apply to the more mature animal and these can be grouped under the following headings:

- Physes
- Periosteum
- External coaptation
- Implants left *in situ*

Physes

Linear growth of long bones is achieved by physeal osteogenesis (see also Chapter 1, Fig. 1.3) and the cartilagenous zones, in particular the zone of hypertrophy, create a focal weak point in the bone which is, therefore, more prone to injury. Trauma to the bone is most likely to result in fracture through the physis rather than through the neighbouring diaphysis, and is seen more frequently than joint luxation in immature patients. Although skeletal maturity is generally considered to be reached by about 12 months of age, physeal fractures may occur in patients older than this. Houlton & McGlennon (1992) documented a possible explanation for this when they noted physeal closure was delayed by early neutering in a series of cats. Physeal injury may damage the germinal layers of the physis without causing a detectable fracture and the importance of this is seen at a later stage when a growth deformity develops.

The types of physeal injury have been classified according to Salter & Harris (1963) (see Chapter 11 and Fig. 11.2). The relevance of this classification system in man is that in type 1 and 2 injuries the growth plate itself is disrupted through the zone of hypertrophy and the germinal layer is not involved. Therefore, continued normal growth is expected after appropriate treatment. Injury types 3 and 4 involve a fracture crossing the entire thickness of the physis and so there is an increased likelihood of abnormal growth postinjury. Unfortunately, in small animals this classification system does not have such an influence on the prognosis for continued growth postinjury. Johnson *et al.*, (1994) demonstrated that Salter–Harris types 1 and 2 canine physeal fractures do not involve only the hypertrophic zone and this may help to explain why premature closure of the physis is often unavoidable following such injuries. However, this is rarely of clinical significance – presumably due to compensation from increased growth in the other physis of that bone or of other bones in that limb (Denny, 1989; Schaefer *et al.*, 1995), or by adjustment of limb posture to compensate for mild loss of bone length by increasing the degree of joint extension.

In treating physeal fractures there are certain factors to bear in mind:

- Good anatomical reduction is of paramount importance since the articular surface may be involved or epiphyseal displacement may have a profound effect on joint alignment and therefore limb function.
- When reducing the fracture the epiphyseal surface should be handled as little as possible since the germinal layer of cells should remain on that side of the fracture line.
- Stabilisation should be planned so as to minimise interference with any potential

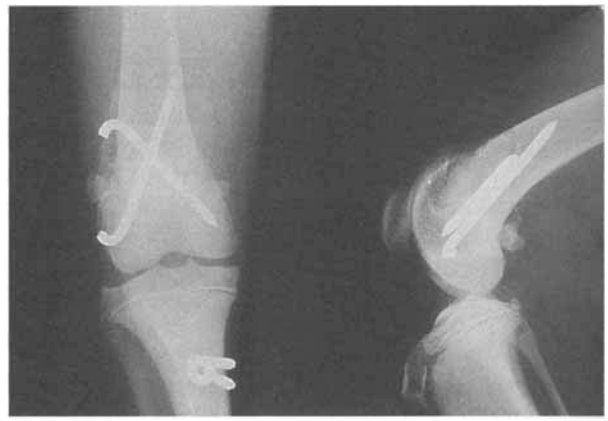


Fig. 15.1 Craniocaudal and mediolateral radiographs, taken immediately postoperatively, of a 6-month-old Border Collie's stifle. A distal femoral physeal fracture (Salter Harris type 1) has been stabilised using crossed K-wires such that neither wire crosses two cortices. This is one of several methods of application that should reduce any influence the implants have on further growth. However, the physis will often close despite such measures (see text for further details).

remaining physeal growth. Linear fixation of the physis, e.g. bone plating or crossed pins that engage the transcortex, should be avoided. Transphyseal implants should not occupy more than 10–20% of its cross-sectional area. Parallel pins, Rush pins or divergent pins that do not cross two cortices (Fig. 15.1) will have minimal effect on any future growth potential. More recently, the use of biodegradable implants (Räihä *et al.*, 1993a,b) has been developed but to a large extent these are cost prohibitive since they address a potential problem that is infrequently of clinical significance. The one exception to this may be in treating an apophyseal fracture (e.g. avulsion of the tibial tuberosity) where continued growth may be expected and lack of this may lead to significant deformity. Because these constitute avulsion injuries it is often necessary to apply a tension band. Use of orthopaedic wire in this situation may lead to deformity even if the wire is removed 4 weeks postoperatively. An alternative is to use a figure-of-eight PDS suture.

The management of specific physeal injuries is discussed elsewhere, mainly in Sections 5 and 6, in this book.

Even when treating diaphyseal fractures in immature patients, the physes must not be forgotten. If the fracture site is stabilised by application of a bone plate or external fixator then it is important to ensure that the implants do not also bridge a nearby physis as normal growth may be considerable during the period of fracture healing whilst the implants are *in situ* (Fig. 15.2). Even if they are

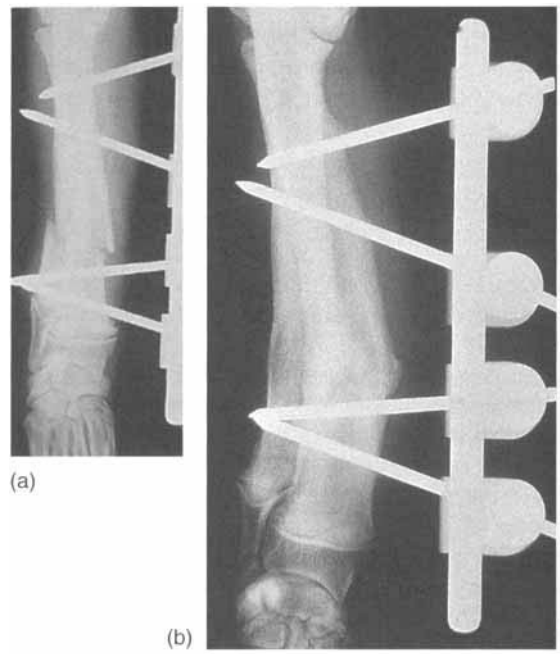
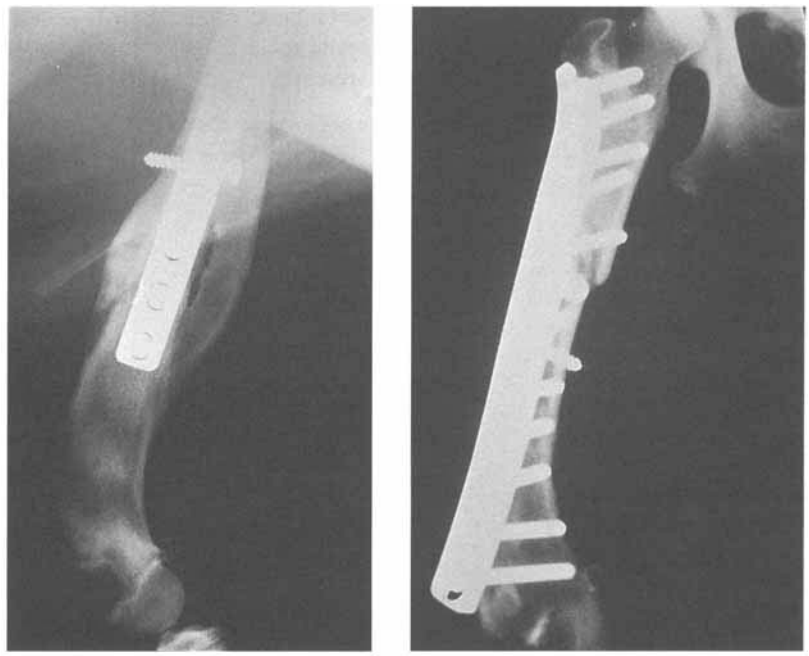


Fig. 15.2 (a) Craniocaudal radiograph, taken immediately postoperatively, of a 5-month-old Labrador's antebrachium. Transverse fractures of the radius and ulna have been stabilised with a four-pin, unilateral, uniplanar fixator. Note that the distal fixation pin is proximal to the distal radial physis. (b) Craniocaudal radiograph of the same fracture taken 6 weeks later. The fracture has healed but note the increase in distance from the distal physis to the distal pin which illustrates the rapid growth rate at this age.



(a) (b)

Fig. 15.3 (a) A mediolateral radiograph of a 7-month-old Collie's femur taken 2 months after stabilisation of an oblique femoral fracture with a lag screw and neutralisation plate. Note the extensive callus formation which may be explained by the highly active periosteum present in a patient of this age.
(b) A mediolateral radiograph of a 5-year-old Collie's femur taken 2 months after stabilisation of a segmental femoral fracture with a dynamic compression plate. Despite the more extensive surgery required in this case the periosteal reaction is minimal and this was presumed to be a result of good stability and a relatively inactive periosteum compared to a younger patient.

then removed, normal growth may not be resumed.

Periosteum

Circumferential growth of long bones is achieved by periosteal bone production and endosteal bone resorption. Thus in the immature patient the periosteum is much more active, and thus much thicker, when compared to that of an adult which is very thin and tightly apposed to the bone surface. The immature periosteum influences fracture healing in ways that may affect management:

- It may not be completely disrupted, or else reapposing the torn ends with sutures may be possible, resulting in it contributing significantly to stability.
- Its greater level of activity will result in a much more rapid and extensive periosteal reaction to injury. Thus, the rate at which callus formation creates fracture stability is much greater due to the amount of callus formed and the higher level of oxygenation (resulting from greater periosteal vascularity – see Fig. 1.9) leading to a larger proportion of the callus being mineralised at an earlier stage.
- A significant periosteal component to the callus can be expected even when accurate reduction and compression would have led to expectations of primary bone healing in the adult patient (Fig. 15.3).
- Any further interference with the periosteum, e.g. during open reduction, will again lead to a reaction way and above that which would be seen in an adult.
- The relative immaturity of the cortical bone reduces bone screw purchase to a small extent. This is also true of the cancellous bone in the epiphyses which often makes it advisable to consider only partially tapping a pilot hole to reduce the risk of 'stripping' the thread.

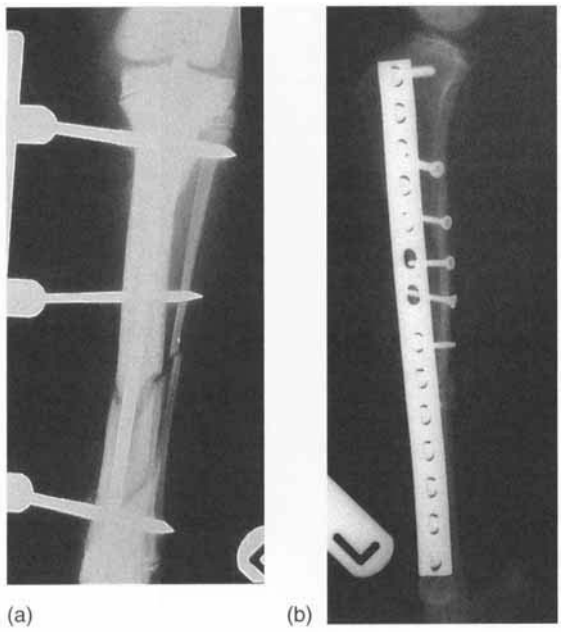


Fig. 15.4 (a) Craniocaudal radiograph of a 5-month-old Labrador's tibia. A comminuted, distal diaphyseal fracture has been stabilised using an intramedullary pin and unilateral, uniplanar fixator. Stability was also considered to be enhanced by incomplete disruption of the periosteum, as judged by preoperative manipulation.
(b) Mediolateral radiograph of a 7-year-old Rough Collie's tibia. A comminuted diaphyseal fracture has been reconstructed with lag screws which have then been protected by the application of a neutralisation plate.

- The increased periosteal activity might contribute to soft tissue adhesions if 'normal' limb function is not restored during fracture healing.

These features might well lead to an alteration in the method of treatment chosen, for example intramedullary pinning (+/– ESF) rather than bone plating (Fig. 15.4).

External coaptation

The use of external casts or splints is an attractive option in young patients since their periosteal activity, and therefore rapid healing (see Table 1.1), will often lead to a successful outcome when such treatment of a similar fracture in an adult might prove problematic. However, there are certain considerations to be kept in mind:

- These patients are growing and a careful eye needs to be kept on them to ensure that they are not 'outgrowing' their cast.
- The inclusion of joints within the cast may have a detrimental effect on the normal development of that joint.
- Fractures above the elbow or stifle are no more amenable to casting than they are in adults. The use of casts to treat mid-femoral fractures has been associated with an unacceptable incidence of quadriceps 'tie-down' which will usually cause very poor long-term limb function and may even require that amputation be considered (see also Chapter 41, p. 504).

Although external casting still has a role to play in fracture management, particularly in immature patients, in the light of advances in this field alternative options should always be kept in mind as they might offer a more appropriate means of achieving fracture healing and normal limb function/development (see Chapter 12).

Implants left *in situ*

Concern is sometimes expressed over leaving implants *in situ* since the patient is very young and this has been used to argue in favour of biodegradable implants (Räihä *et al.*, 1993a,b). However, the complications of leaving implants *in situ* are the same as in the mature patient and most problems can be resolved by their removal if a complication arises. The one complication that cannot be resolved in this way is that of fracture-related sarcoma and the long-term presence of implants has been held partly responsible for these, though without any scientific evidence. This subject is discussed towards the end of Chapter 13.

One particular point worth mentioning concerns intramedullary pins. If these are precut and broken off flush with the bone then they may become inaccessible after continued bone growth. Therefore, if removal was required at a later date this could prove highly problematic. Instead it

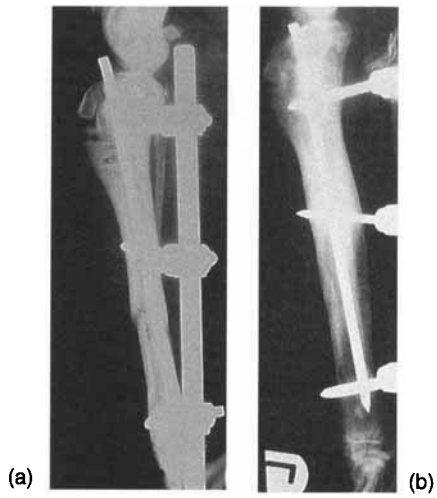


Fig. 15.5 (a) Mediolateral radiograph of the same tibia as in Fig. 15.4a. Note that the intramedullary pin has been left quite long.
(b) Mediolateral radiograph of the same tibia taken 8 weeks later. Fracture healing is complete but note how much less of the intramedullary pin sits proud of the proximal tibia. All the implants were removed at that time.

may be better to leave the pin long so that it can be retrieved (Fig. 15.5). However, doing so makes it more likely that removal will be necessary as the protruding end may irritate the surrounding soft tissues.

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Section 4
The Skull and Spine

A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny,Steven J. Butterworth

Chapter 16
The Skull

Fractures of the mandible

Mandibular fractures are common in cats and account for 14.5% of all fracture cases (Umphlet & Johnson, 1988). Dogs are presented less frequently with an incidence of only 2.7% (Umphlet & Johnson, 1990). The site of mandibular fracture also differs between the two species. The mandibular symphysis is the most common jaw fracture in the cat, 73.3% of cases (Umphlet & Johnson, 1988), while in the dog the majority of fractures involve the premolar (31%) or the molar (18%) regions (Umphlet & Johnson, 1990).

The common sites of mandibular fracture are the:

- Symphysis
- Horizontal ramus between the canine and first premolar teeth
- Horizontal ramus at the level of the carnassial tooth
- Junction of the horizontal and vertical ramus

The vertical ramus is less prone to fracture because it is well protected by muscle and the zygomatic arch. Care is required in evaluating cats with obvious symphyseal fractures since they may also have fractures involving the mandibular condyle(s) which may affect the prognosis given (Sullivan, 1989).

The majority of mandibular fractures result from involvement in road traffic accidents. Falls, kicks, bites, gun-shot wounds and dentistry account for the remaining cases. Periodontal disease predisposes to fracture and could account for the relatively high incidence of the injury in Poodles and Pekingese.

Clinical signs

Clinical signs of mandibular fracture include bleeding from the mouth, excessive salivation and malocclusion of the teeth. The jaw is displaced towards the side of the fracture. Upper airway obstruction can occur in the cat or dog with fractures of the mandible and/or maxilla, particularly if the animal is concussed, because blood and mucus tend to accumulate in the back of the pharynx. The first priority is to remove this material and, if necessary, pass an endotracheal tube to maintain an airway.

Although many fractures of the mandible are open fractures, infection is seldom a serious problem due to the antibacterial and cleansing action of saliva (Weinmann & Sicher, 1955). Nevertheless, antibiotics are generally used as a precaution in the perioperative period. The aim of treatment is to immobilise the fracture and restore normal occlusion of the teeth to allow an early return to normal feeding.

Treatment

First-aid immobilisation of mandibular fractures is achieved simply by closing the mouth and applying a muzzle so that the upper jaw acts as a splint for the lower. The muzzle can be used as the sole method of treatment for some fractures (i.e. conservative management) and is loosened at feeding times to allow the animal to drink liquids. The methods of fixation for mandibular fractures are listed below according to the region of the mandible involved.

Separation of the mandibular symphysis

Methods of stabilising the symphysis include:

- Cerclage wire
- Transmandibular lagged bone screw
- Transmandibular pin

Cerclage wire (Winstanley, 1976) Good fixation of the symphysis in cats and dogs can be achieved with a cerclage wire placed around the mandible just caudal to the canine teeth (Fig. 16.1c). The wire is passed under the soft tissues so that it lies in close contact with the bone. A large hypodermic needle is bent into a half circle; it can be used as a guide to pass the wire around the symphysis (Figs 16.1a and 16.1b). The cerclage wire tends to become buried beneath the mucous membrane and can be left *in situ*. The symphysis should heal within 6 weeks; if the wire is still visible or causing soft tissue reaction it is removed after this period.

Alternatively, one end of the wire can be placed from just lateral to each mandible such that the ends pass ventrally towards the midline and exit through a stab incision (Fig. 16.1d). The ends are drawn tight and twisted with or without first being placed through a button. Removal of the wire is generally required after healing but may be achieved easily by cutting the wire on the floor of the mouth and pulling the twisted end ventrally.

Transmandibular lag screw (Lawson, 1963; Wolff, 1974) The mucous membrane is elevated and a lag screw inserted tranversely just behind the canine teeth and rostral to the middle mental foramen (Fig. 16.2). This method provides optimal stability; care should be taken to avoid the roots of the canine teeth.

Transmandibular pin (Leonard, 1971; Spellman, 1972) An alternative to the lag screw is to transfix the mandible with a pin (site of entry is as in Fig. 16.2). If additional stability is required a figure-of-eight wire is then placed rostral to the incisors with the loops of the wire around the pin ends.

Fractures of the horizontal ramus of the mandible

Opinions differ considerably between dentists and orthopaedic surgeons concerning the best methods of treating fractures of the mandible. Dentists will favour interdental wiring and intraoral splints of dental composite or acrylic. However, these techniques depend on the animal having stable teeth for fixation. Fractures of the

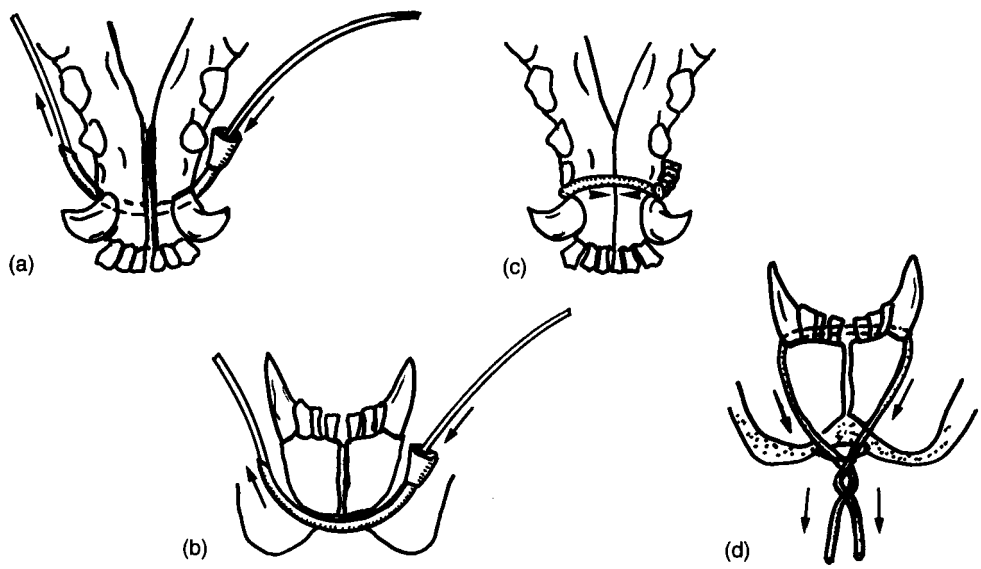

Fig. 16.1 Cerclage wire for fixation of mandibular symphyseal separation. (a and b) Hypodermic needle used as a guide to pass wire around the symphysis. (c) Cerclage wire *in situ*. (d) An alternative method of placing the wire with ventral exit through a stab incision.

ramus rostral to the first molar teeth are particularly suitable for these dental techniques. The orthopaedic surgeon tends to rely on conventional methods of fracture repair using internal or external fixation, particularly for fractures caudal to the premolar teeth. Whatever method of fixation is used for fractures of the mandible, an attempt should be made to repair torn gums and buccal mucous membrane to limit further contamination of the fracture site.

Fracture of the ramus rostral to the first premolar tooth

In some cases the buccal mucous membrane can be sutured securely enough to provide adequate stability, even in some bilateral injuries. Methods of fixation include:

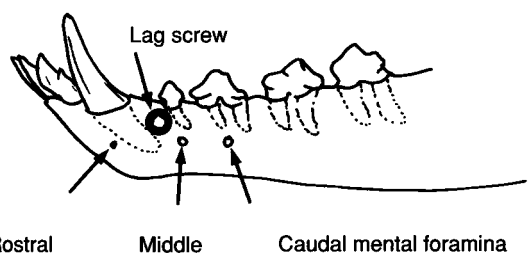

Fig. 16.2 Transmandibular lag screw for fixation of symphyseal separation.

- Wiring
- Intra-oral acrylic splints

Wiring Interdental wiring is used either alone or in combination with interfragmentary wire. The interdental wire is placed first. A hole is drilled transversely through the ramus just caudal to the fracture to anchor the wire (Fig. 16.3a), the end of the wire is passed around the canine tooth in a figure-of-eight and the free ends are twisted tight on the lateral aspect of the gingival margin (Fig. 16.3b). If necessary, stability can be improved by placing a figure-of-eight wire on the ventral aspect of the fracture (Fig. 16.3c). Exposure is achieved through a ventral skin incision.

Intra-oral acrylic splints Ideally, the intra-oral acrylic splint (Coe-Kooliner, Henry Schein Veterinary Dental) incorporates two teeth rostral and two teeth caudal to the fracture (Davidson, 1993). The teeth are first etched with acid (37% phosphoric acid) to promote adherence of the acrylic to the teeth (Fig. 16.4a). An alternative to etching the teeth is to place an interdental wire (Fig. 16.3) which is then reinforced with dental acrylic, applied so as to incorporate the wire and teeth (Fig. 16.4b). This should give the acrylic a better fix to the teeth. Light-cure dental adhesive followed by light-cure composite can be used in the same way as acrylic for small areas of mandible. Once the fracture has healed the acrylic or dental

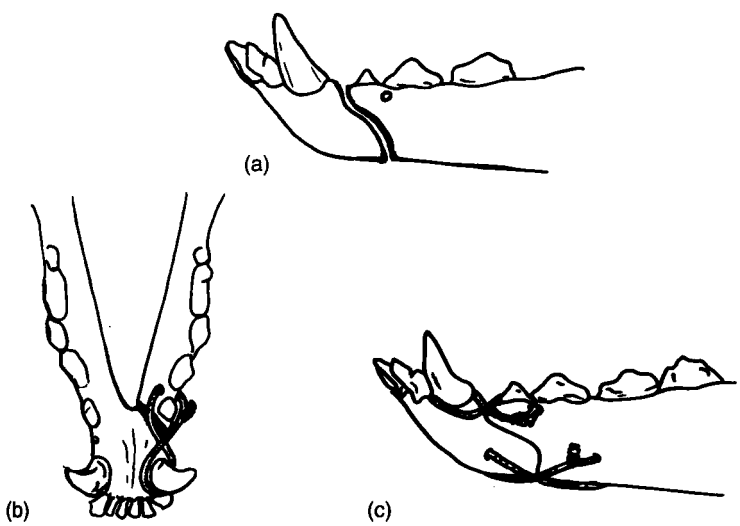

Fig. 16.3 Wiring rostral fractures of the mandible. (a) Hole drilled to anchor interdental wire. (b) Interdental wire placed. (c) Tension band wire placed.

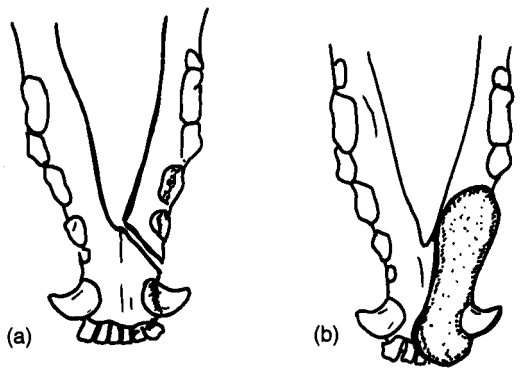

Fig. 16.4 Intra-oral acrylic splint. (a) Teeth etched with acid. (b) Stabilisation of fracture with the splint.

composite is removed from the teeth using a burr and rongeurs.

Fractures of the horizontal ramus caudal to the second premolar tooth

Methods of fixation include:

- Plate fixation
- External fixators
- Transverse pinning
- Wire sutures

Plate fixation Most fractures of the horizontal ramus in both dogs and cats are best managed by plate fixation (Sumner-Smith & Dingwall, 1971, 1973; Boudrieau & Kudisch, 1996). It is important to achieve accurate anatomical reduction of the fracture to ensure normal dental occlusion. The best way of checking dental occlusion is to close the mouth which, unfortunately, cannot be done with an endotracheal tube in the normal position. This problem can be overcome after induction of anaesthesia by making a pharyngotomy incision (through the lateral pharyngeal wall of the piriform fossa, just caudal to the articulation between the stylohyoid and epihyoid bones), through which the endotracheal tube is passed and then directed down the larynx. Anaesthesia is then maintained in the normal way and the endotracheal tube does not interfere with normal jaw occlusion during surgery. The plate is applied to the lateral surface of the mandible close to the ventral border of the ramus. It is essential that the plate is carefully contoured to the shape of the

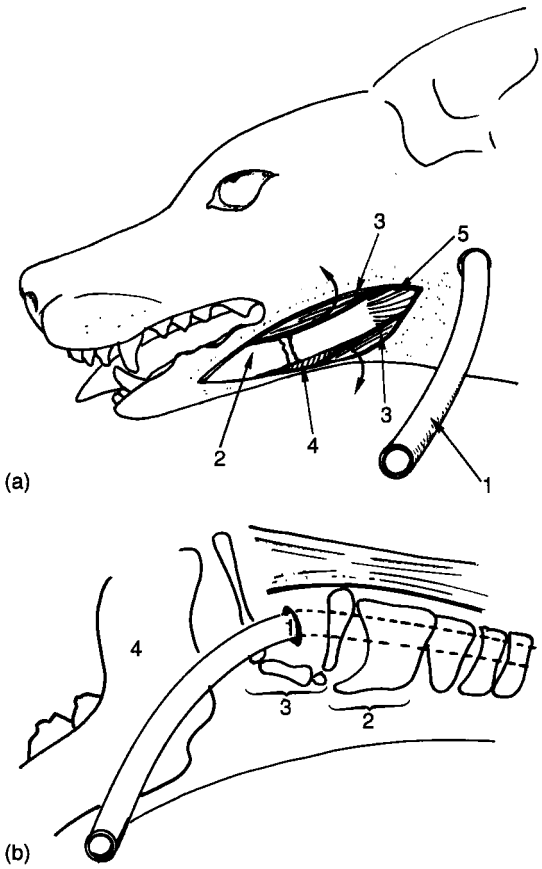

Fig. 16.5 (a) Exposure of the horizontal ramus of the mandible. 1 – Endotracheal tube placed through pharyngotomy incision; 2 – skin incision over ventral ramus; 3 – platysma muscle incised and reflected to expose mandible; 4 – myelohyoideus muscle; 5 – digastricus muscle. (b) Landmarks for placement of endotracheal tube. 1 – Pharyngotomy incision; 2 – hyoid apparatus; 3 – laryngeal cartilage; 4 – vertical ramus of the mandible.

bone to prevent malocclusion. Being able to do this is helped by the fact that the plate is not required to have weight-bearing strength which allows the use of relatively small plates. The AO/ASIF reconstruction, mini- and cuttable plates are particularly useful in these cases. Exposure of the fracture is through a skin incision over the ventral aspect of the ramus. The platysma muscle is incised and retracted dorsally to expose the bone (Fig. 16.5).

Application of the plate close to the ventral border of the ramus (Fig. 16.6) avoids the risk of

penetration of tooth roots and the mandibular nerve by bone screws.

External fixator In comminuted fractures of the ramus, or in dogs with poor bone stock as a result of periodontal disease, the use of an external fixator may be more readily applicable than a plate. The shape of the mandible in breeds like the Pekingese may make use of a conventional connecting bar on the fixator difficult and in this situation the pins or Kirschner wires are first driven into the bone fragments (at least two in each) and then joined on the lateral aspect of the jaw with bone cement or dental acrylic (Acrylic Pin External Fixation, Innovative Animal Products) (Fig. 16.7).

Transverse pinning (Lawson, 1957) This is a simple and effective form of fixation. The pin passes horizontally caudal to the fracture through the sublingual tissue and must be inserted at right angles to the median plane through all cortices (Fig. 16.8).

Wire sutures These may be placed around the base of teeth or through holes drilled in the bone and can be used for fixation in small dogs (Fig. 16.9).

Fractures of the vertical ramus

The vertical ramus is well protected by muscle and, provided there is little displacement of the fragments, fractures of this region can be treated conservatively (Lawson, 1963). However, plate fixation has been described (Sumner-Smith & Dingwall, 1971; Boudrieau & Kudisch, 1996). A skin incision is made over the angle of the jaw (Fig. 16.10). The periosteum is incised along the caudal aspect of the ramus and elevated with the attached masseter muscle to expose the vertical ramus, taking care to avoid the vital structures shown in Fig. 16.11. Small ASIF plates are ideal for fracture fixation using 1.5 mm, 2.0 mm or 2.7 mm cortical screws. Alternatively, wire sutures may be used for fixation.

Aftercare

Liquid or soft food should be fed for a least 3 weeks after fixation. Cats with severe trauma to the jaw may need feeding through a pharyngotomy tube for a few days after surgery. Systemic antibiotics are indicated in all cases with open fractures. If malocclusion, due to malunion, prevents eating then extraction of the offending teeth may be necessary.

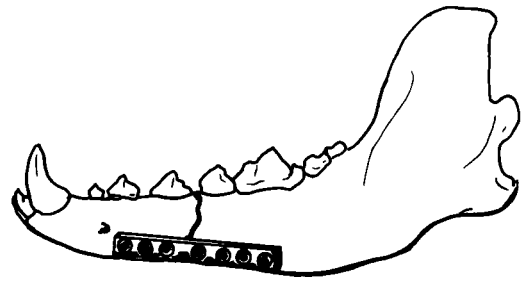

Fig. 16.6 Plate applied close to the ventral border of the mandibular ramus.

Fractures of the nasal, premaxilla and maxillary bones

Although fractures of these bones are often initially associated with epistaxis and obstruction of the nasal passages, this is usually a transient problem and most fractures will heal without surgical interference. Occasionally a dog will be encountered with gross instability of the nose resulting from multiple fractures. Some of these can be readily treated by suturing the mucosa over the hard palate. In others, fixation may be required and can be provided by half-pin splintage. The fragments are transfixed with pins and the ends of the pins are incorporated in an acrylic (Acrylic Pin External Fixation, Innovative Animal Products) 'bumper' moulded around the nose (Fig. 16.12). Alternatively, such acrylic bars can be placed on either side of the nose.

Feline high-rise syndrome and hard palate fractures

The term 'high-rise syndrome' was first used by Robinson in 1976 to describe the injuries seen in cats which had fallen two or more storeys (7 m or more). Injuries most commonly sustained include thoracic, facial and oral trauma and limb injuries.

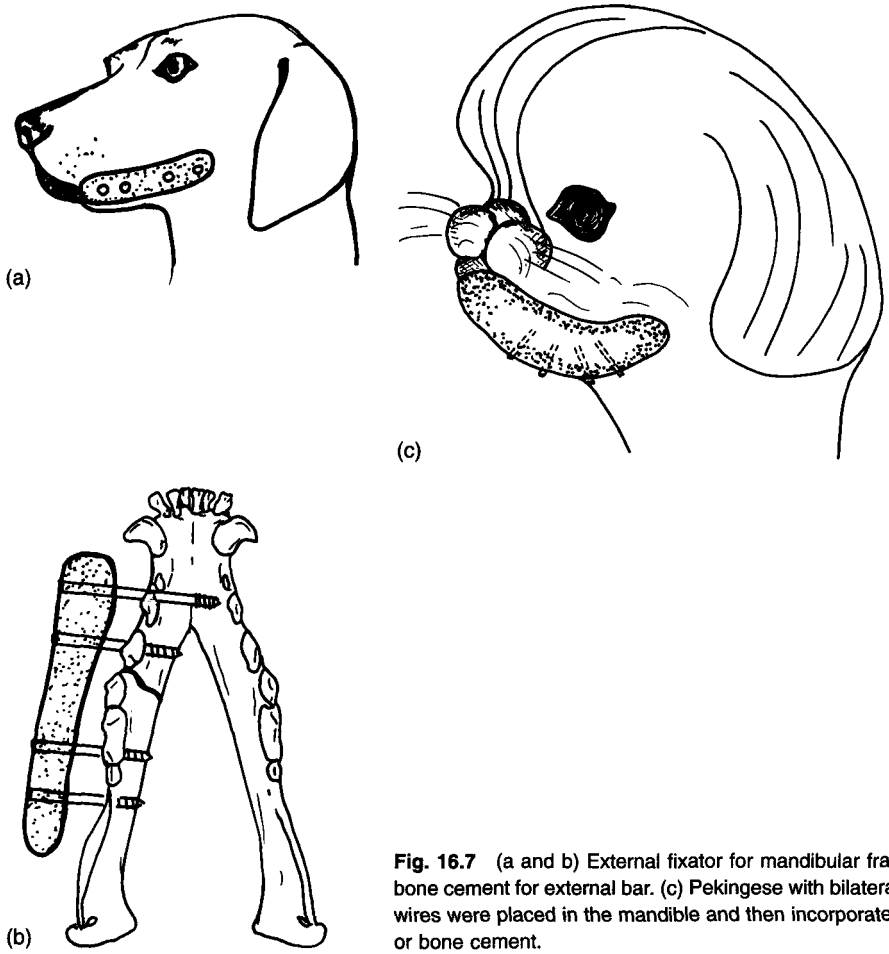

Fig. 16.7 (a and b) External fixator for mandibular fracture using dental acrylic or bone cement for external bar. (c) Pekingese with bilateral ramus fractures. Kirschner wires were placed in the mandible and then incorporated externally in dental acrylic or bone cement.

The survival rate for treated cats is 90% (Whitney & Mehlhaff, 1987). Because cats fall in a splay-legged position they often sustain either direct trauma to the head or secondary injury through bouncing. Therefore, facial injuries are frequently seen in cats, particularly splitting of the mandibular symphysis or hard palate. Whitney & Mehlhaff (1987) recommended conservative treatment for hard palate fractures in cats (soft food for at least 1 month) and they reported that all healed without surgery. The authors prefer a tension band wiring technique to close the split in the hard palate. The wire is placed between the roots of the carnassial teeth (Fig. 16.13) and is removed 4–6 weeks later. This provides rapid relief from pain and the cat is usually willing to take soft food within a day or two of surgery.

Fractures of the neurocranium

(Hoerlien, 1971; Oliver, 1975; Dewey *et al.*, 1993)

Fractures of the neurocranium may be associated with brain damage, either directly or indirectly through haemorrhage into the cranial vault. Few cases are presented for treatment, presumably because the injury is often fatal. Depression fractures may impinge on, or lacerate, the cerebral cortex and associated signs will, of course, vary with the degree and location of the brain damage. Linear fractures may require no treatment except the administration of methylprednisolone (Soumedrone V, Pharmacia & Upjohn) to control oedema (see p. 207).

Intracranial haemorrhage and oedema may be associated with:

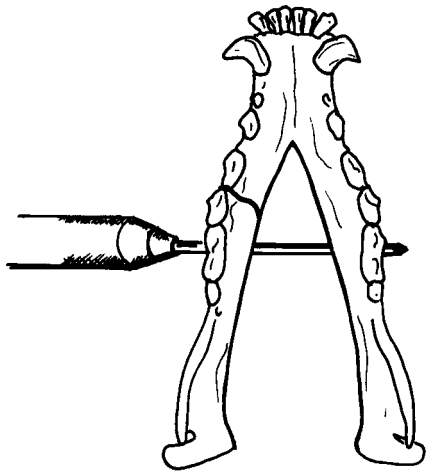

Fig. 16.8 Transverse pinning for fixation of a mandibular fracture.

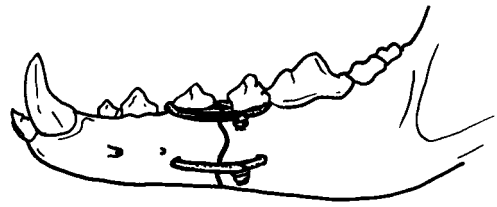

Fig. 16.9 Wire sutures for fixation of a mandibular fracture.

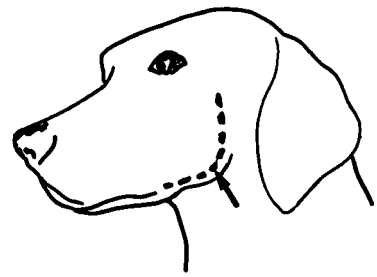

Fig. 16.10 Skin incision for exposure of the vertical ramus of the mandible.

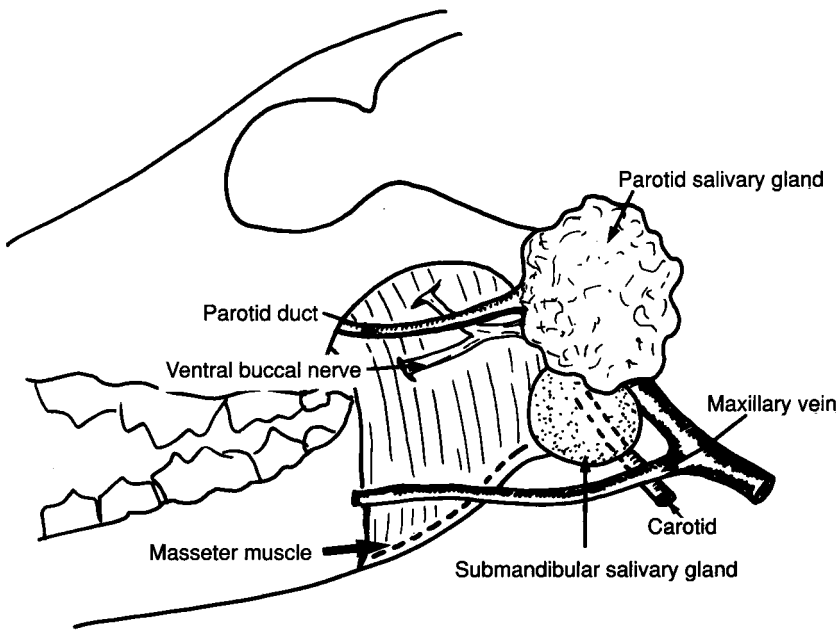

Fig. 16.11 Exposure of the vertical ramus, elevation of the masseter muscle protecting vital structures shown. (From Sumner Smith, G. & Dingwall, J.S. (1973) The plating of mandibular fractures in giant dogs. *Veterinary Record*, **92**, 39–43. Redrawn with permission.)

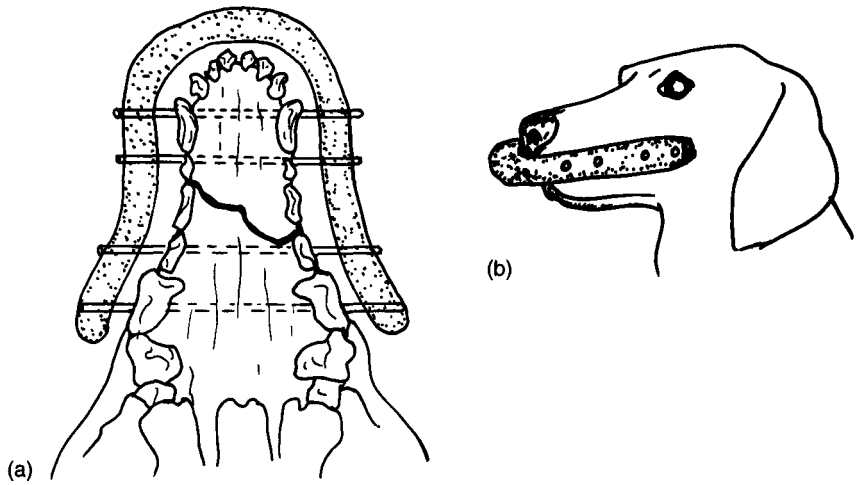

Fig. 16.12 (a) Open mouth view and (b) external view of acrylic 'bumper' external fixator for maxillary fracture.

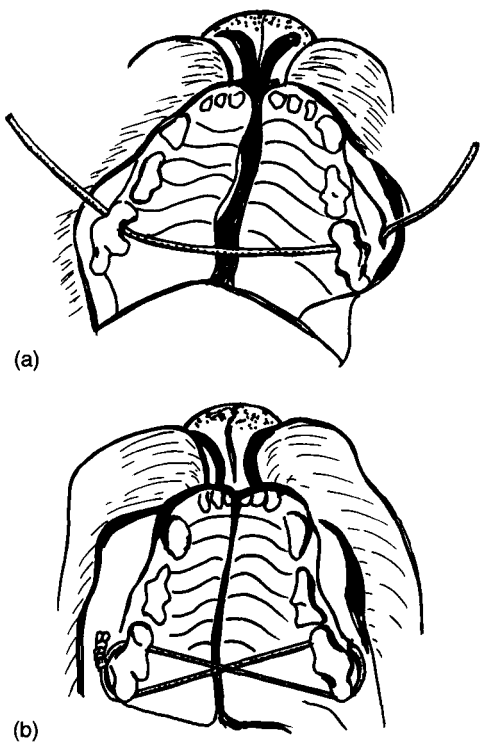

Fig. 16.13 Cat with split hard palate. (a) Wire is placed between the roots of the carnassial teeth. (b) Wire tension band used to close split in the hard palate.

- Loss of consciousness
- Dilatation of one or both pupils or other evidence of cranial nerve injury
- Motor dysfunction such as hemiparesis or decerebrate rigidity

If any of these signs are present and progressive, then cranial decompression should be considered as an emergency procedure. Pressure may be relieved by trephining the skull close to the fracture. In the case of depression fractures, pressure may be relieved by careful elevation of the fragments from the dura mater. Haemorrhage is controlled and the dura closed either by direct suturing or by application of a temporal fascia graft if a defect is present. Closure of the dura mater is important because of its function as a barrier to infection for the central nervous system. The defect in the skull is covered with the temporal muscle. Alternatively, if the fragment is large, it may be retained in postion with wire sutures.

Conditions of the temporomandibular joint (TMJ)

Luxation of the temporomandibular joint

Open 'jaw locking' as a result of temporomandibular luxation is a well recognised but uncommon clinical entity in the dog (Leonard,

1971; Knecht & Schiller, 1974). Traumatic overextension of the temporomandibular joint (TMJ) results in rostral and upward displacement of the mandibular condyle. Radiographs should always be taken before reduction is attempted to ensure that there are no concurrent fractures that might complicate treatment. Under general anaesthesia manual reduction is achieved by using a fulcrum (1–3 cm diameter wooden rod) transversely across the mouth. The fulcrum helps to move the mandibular condyle backwards and ventrally to re-engage the temporal joint surface as the rostral end of the mandible is pushed towards the maxilla. The fulcrum is removed and occlusion is checked. It may be necessary to tape the mouth closed (between meals) for 10–14 days to maintain reduction.

Subluxation of the temporomandibular joint

Subluxation of the TMJ may result in repeated bouts of open mouth jaw locking (Cameron *et al.*, 1975; Robins & Grandage, 1977; Lantz, 1987, 1991). The condition is encountered in a variety of breeds of dog but the Basset Hound and Irish Setter appear to be overrepresented. A single case has also been reported in the cat (Lantz, 1985, 1991). It has been suggested that the primary aetiological factor is temporomandibular dysplasia. This allows the coronoid process to become displaced laterally to the rostral part of the zygomatic arch when the mouth is wide open, which then prevents closure (Fig. 16.14a). The condition can be relieved by resection of the ventral part of the zygomatic arch (Fig. 16.14b).

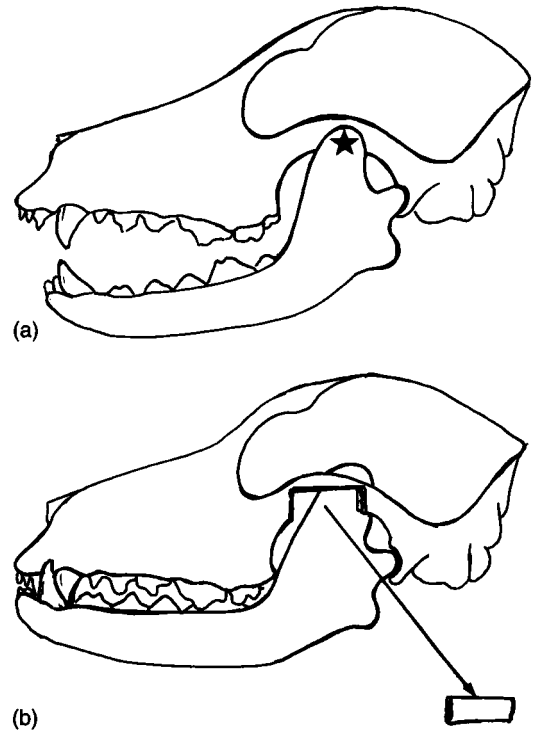

Fig. 16.14 Temporomandibular joint dysplasia. (a) Open jaw locking with coronoid process of the mandible displaced laterally to the zygomatic arch. (b) Surgical correction by removal of part of the zygomatic arch. (From Robins, G.N. & Grandage, G. (1977) Temporomandibular joint dysplasia and open jaw locking in the dog. *Journal of the American Veterinary Medical Association*, **171**, 1072–9. Redrawn with permission.)

Temporomandibular ankylosis in the cat

Fractures or luxations involving the TMJ, particularly in the cat, may result in temporomandibular ankylosis (Sullivan, 1989). The range of jaw movement becomes increasingly restricted so the cat has difficulty feeding and grooming itself. Administration of corticosteroids does little to halt the progression of the ankylosis or to prevent recurrence if the jaw has been forcibly stretched open under general anaesthesia. The best option in these cases is excision arthroplasty of the affected joint with removal of the mandibular condyle, adjacent osteophytes and scar tissue. Sullivan (1989) reported good results in two cats treated in this way, both being able to eat and groom normally when examined 18 months after surgery. Excision arthroplasty also gave good long-term results in four cases treated by one of the authors (unilateral procedure in three; bilateral in one).

Technique for excision arthroplasty of the temporomandibular joint

The first problem is of course intubation. Be prepared to do a tracheotomy if the mouth cannot be quickly prised open soon after induction of anaesthesia. To open the mouth insert the tips of a sturdy pair of artery forceps or stifle distractors (Veterinary Instrumentation, Sheffield) between

the caudal molars of the upper and lower jaw nearest the ankylosed joint. Gradually open the artery forceps and this should create enough force to slowly open the jaw. Having opened the mouth the endotracheal tube is passed and anaesthesia maintained in the usual way.

The TMJ is best approached by a horizontal lateral incision directly over the joint which is located immediately ventral to the caudal extremity of the zygomatic arch. The masseter muscle attachments to the zygomatic arch are freed and the muscle is reflected ventrally to expose the joint while taking care of the branches of the facial nerve. The mandibular condyle is removed using small rongeurs or a high-speed burr. However, in most cases, because of the extensive periarticular new bone formation around the joint, it is first necessary to remove the caudal third of the zygomatic arch before advancing in a ventromedial direction removing proliferative bone, part of the vertical ramus and the mandibular condyle until the jaw can be readily opened (Fig. 16.15).

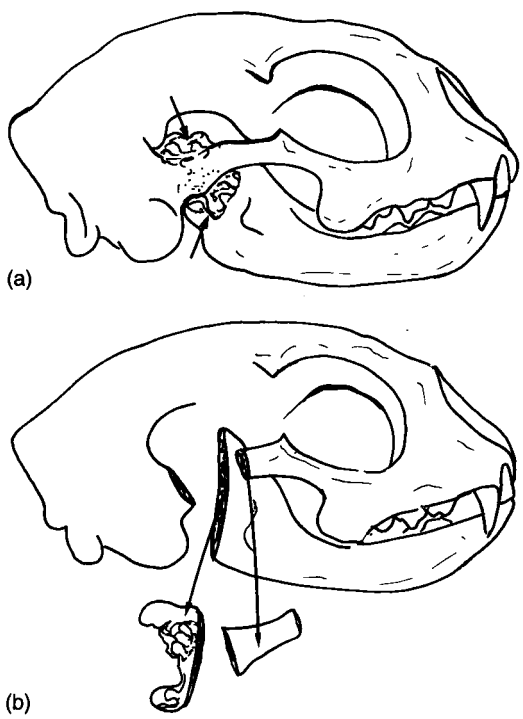

Fig. 16.15 (a) Temporomandibular joint ankylosis in the cat and (b) treatment by excision arthroplasty.

Temporomandibular joint ankylosis in dogs

Temporomandibular joint ankylosis has been reported in dogs following craniofacial trauma or ear infection (Lantz, 1985). The basic principles of treatment are the same as those described above for the cat.

Mandibular neurapraxia

Mandibular neurapraxia (Robins, 1976) is the term used to describe a condition in the dog which is probably caused by bilateral, temporary paralysis of the mandibular branch of the trigeminal nerve as a result of wide opening of the mouth. In affected cases the lower jaw hangs down passively but manipulation of the mandible is not resented and the mouth can be closed manually. However, the lower jaw drops as soon as it is released. The condition usually resolves within 3 weeks. The mouth is kept loosely muzzled during this period and the dog is fed a semi-liquid diet with the muzzle in place.

Mandibular neurapraxia should be differentiated from other conditions which cause an inability to close the mouth such as:

- Oral foreign body
- Fracture of the mandible
- Luxation or subluxation of the TMJ

Craniomandibular osteopathy

This condition is seen particularly in West Highland White Terriers, leading to it being referred to as 'Westie disease'. Due to the outward appearance of dogs suffering with the condition it is also termed 'lion jaw'. Other breeds may be affected, mainly the terriers but it has also been recorded in several other breeds including medium- and large-sized breeds (as listed by Stead, 1994). The condition is idiopathic but a genetic influence is likely.

The lesion is essentially a periostitis affecting one or more of the mandibular rami, the temporal bones or other bones of the skull (Fig. 16.16). If the proliferative changes are extensive then temporomandibular ankylosis may result. Occa-

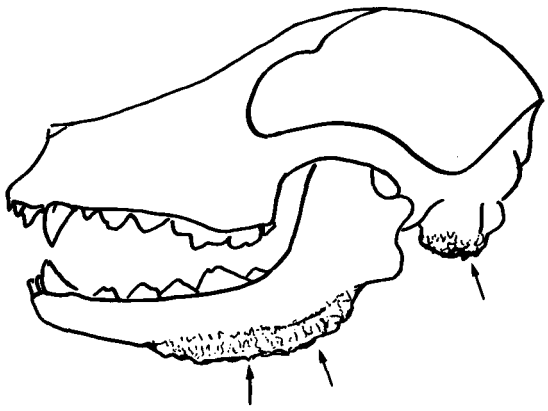

Fig. 16.16 Craniomandibular osteopathy, periosteal reaction on horizontal mandibular ramus and petrous temporal bone.

sionally, lesions may also be seen affecting appendicular long bones and heterotopic mineralisation of soft tissues adjacent to affected bone occurs, but infrequently. Riser (1967) described the histopathology of the disease. The normal lamellar bone is replaced by coarse fibrous bone and the marrow adopts a fibro-cellular appearance.

Clinically affected patients present at 3–7 months of age showing swelling of the jaws, inappetance, lethargy and pyrexia. Palpation of the mandible is painful, as is any attempt to open the mouth. Confirmation of the diagnosis is by radiography which will show dramatic periosteal new bone formation on the mandible and/or around the TMJ, with similar changes on any affected long bones. In the early stages the margins of the lesion are irregular, radiographically, but generally become more smooth with time as remodelling takes place.

Treatment is symptomatic and supportive. Prednisolone is the most effective analgesic and food should be softened or liquidised. The condition tends to run an undulating course and resolves by 8–12 months of age. As long as the TMJ remains functional the prognosis for a full recovery is good. If, however, TMJ ankylosis occurs and there remains insufficient function for the dog to be able to eat, then excision arthroplasty would need to be considered (as described above) and the prognosis would become much more guarded.

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A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny,Steven J. Butterworth

Chapter 17
Neurological Examination

This chapter will concentrate on the aspects of patient evaluation that help to localise a spinal cord lesion and determine its severity. It is not the intention of the authors to cover the localisation of intracranial lesions as the diagnosis and treatment options for such problems are beyond the scope of this book and for further information on such matters the reader is referred to standard neurology texts.

Neuroanatomy

Spinal cord (Fig. 17.1)

This arises from the brain stem and terminates within either the sixth lumbar vertebra (medium to large breed dogs) or the seventh lumbar vertebra (small breed dogs and cats). This foreshortening of the spinal cord is discussed further in Chapter 26 and is illustrated in Fig. 26.1. The spinal cord is divided into segments (8 cervical, 13 thoracic, 7 lumbar, 3 sacral, variable coccygeal/caudal). It is composed of grey matter centrally (comprising the cell bodies of lower motor neurons) and white matter peripherally (comprising myelinated and non-myelinated axons forming the descending motor and ascending sensory tracts). The spinal cord is surrounded by the meninges (pia mater, arachnoid mater and dura mater) with cerebrospinal fluid being located in the subarachnoid space.

Upper and lower motor neurons (UMNs and LMNs) (Fig. 17.2)

Upper motor neurons (UMNs) arise within motor centres of the brain, mostly decussate in the brain stem, travel down the spinal cord in the lateral and ventral funiculi and synapse, via interneurons, with the lower motor neurons (LMNs). The cell bodies of the LMNs lie within the ventral horn of the grey matter. Their corresponding axons leave the spine via the ventral spinal nerve root and finally reach the target muscle via a peripheral nerve. The LMNs may be the efferent arm of a reflex arc with the afferent arm comprising a sensory nerve fibre originating from a receptor detecting, for example, pain or muscle stretch. Such sensory fibres enter the spinal cord via the dorsal spinal nerve root and have their cell bodies located within a dorsal root ganglion. They may synapse directly with the efferent LMN or else communicate with it indirectly through interneurons. Ascending tracts in the spinal cord comprise, mainly, sensory tracts in the dorsal funiculi. However, in the dog, there is an ascending motor tract arising in the lumbar region and travelling cranially, in the contralateral lateral funiculus, to the thoracic limb in which it inhibits extensor activity. Damage to this tract is responsible for the Schiff–Sherrington phenomenon (see later).

Involuntary (or reflex) movement in each limb is, therefore, dependent on the integrity of the sensory nerve, the motor nerve (LMN) and the relevant spinal cord segments. Voluntary movement is, on the other hand, dependent on UMN and LMN function. Testing of the LMN reflexes in all four limbs allows differentiation between UMN and LMN lesions which directly assists in localising spinal cord lesions (see later).

History

As in most clinical cases, the taking of a thorough clinical history will usually be highly indicative of

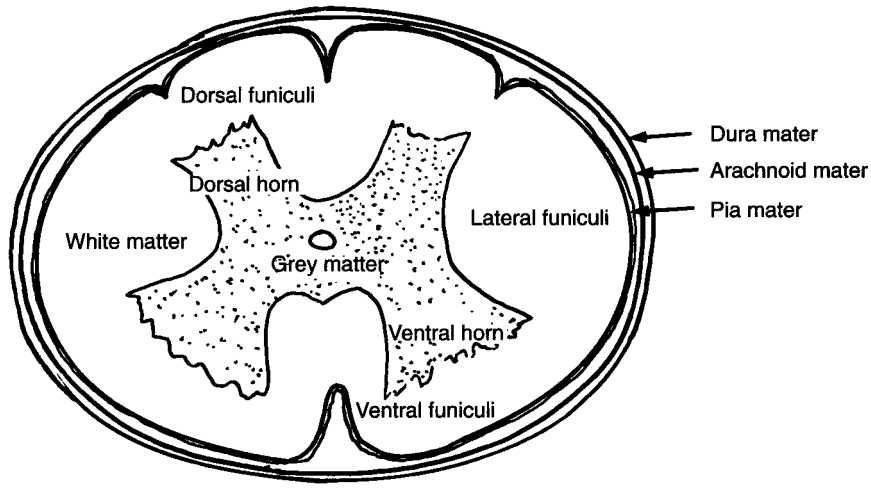

Fig. 17.1 Schematic cross-section of a spinal cord illustrating normal anatomical features.

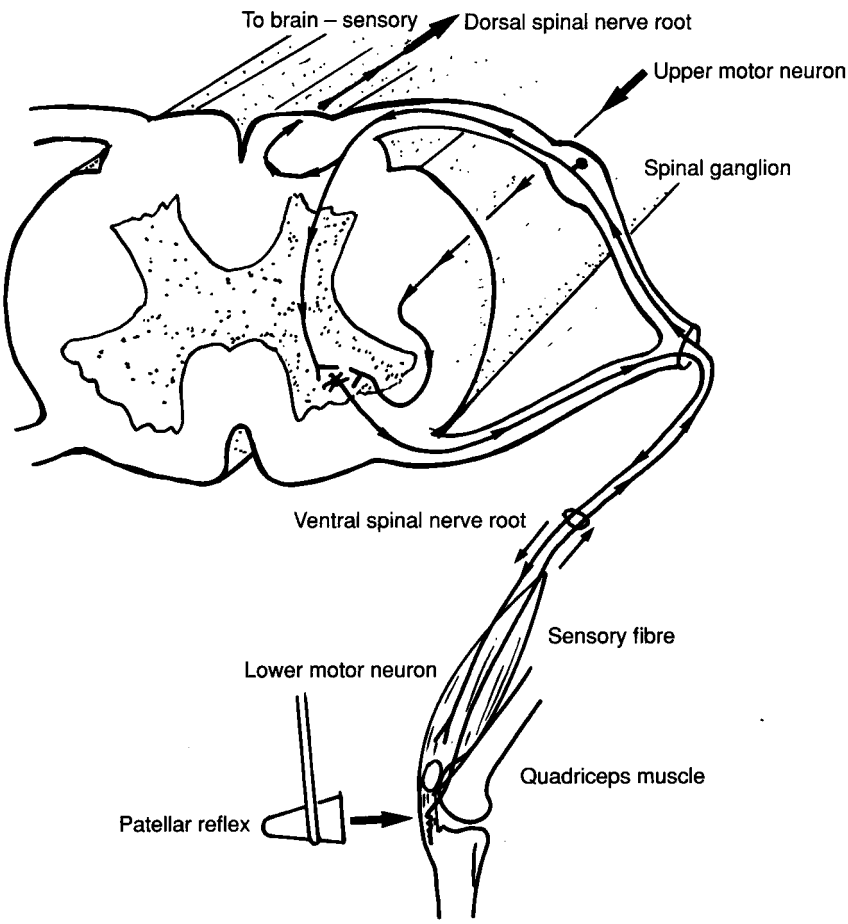

Fig. 17.2 Illustration of a spinal reflex arc showing sensory (afferent) input via the dorsal spinal nerve root, and the motor output (efferent), involving a lower motor neuron (LMN), leaving the spine via the ventral spinal nerve root.

the final diagnosis, whereas disregard of such information may well lead directly to a convoluted and less than ideal investigation. Information such as the breed and age of the patient can be highly suggestive of a specific diagnosis, but care should be taken not to assume this is the only possibility or the outcome could be disasterous. *General* patient details would include:

- Breed
- Age
- Bodyweight
- Sex
- Previous illness, surgery or similar problem
- Time been with current owner
- Other, current problems
- Use (pet, sheep dog, etc.)
- Vaccination status
- Diet
- Problems with related or in-contact animals

More *specific* details would include:

- Rate of onset of signs
- Duration of signs
- History of trauma
- Overt evidence of pain
- Which limbs affected
- Evidence of urinary or faecal incontinence

Spinal disease should be suspected in any animal developing a reluctance to jump (up steps, on its favourite chair, into the car, etc.), showing intermittent, unprovoked pain, or showing incoordination (especially if further examination reveals proprioceptive deficits).

Clinical examination

This is mandatory in all clinical cases and is no less so for the apparent 'spinal' case since general medical conditions, e.g. diabetes mellitus or iliac thrombosis resulting from cardiomyopathy, can produce neuropathies which may mimic spinal disease. Similarly, these cases should be subjected to an orthopaedic examination since problems such as bilateral cranial cruciate or plantar ligament failure may produce a disability that could easily be mistaken for a spinal problem. Conversely, spinal problems may mimic other conditions, for example a small breed dog suffering with pain from thoracolumbar disc disease may present with a tense abdomen and most spinal surgeons can recall having operated on at least one dog to treat disc disease only a matter of days after an exploratory laparotomy had been performed elsewhere.

Neurological examination

Observation

Except in cases where vertebral column trauma is suspected from the outset, posture of the head, trunk and limbs should be noted with the patient standing and the gait should be observed at the walk and trot. The dog should also be turned in moderate and tight circles and made to step up and down over a low step or kerbstone. Features to look for include:

- *Neck pain* tends to produce a low head carriage and a patient that 'looks with its eyes' rather than its head. If an object is not in their field of vision they may 'shuffle' round in order to look at it, rather than turning their head. A low head carriage tends to be counterbalanced by drawing the hindlimbs cranially which creates an arched back (easily mistaken as a sign of 'back pain'). In many cases, trauma excepted, neck pain may be intermittent in which case the dog may appear normal most of the time, then shrieks out and holds its neck rigid for a few moments or minutes at a time.
- *Thoracolumbar pain* tends to produce arching (or roaching) of the back with the hindlimbs drawn forwards and a tense abdomen. In order to counterbalance this the dog may put its head down and so the presentation could be mistaken for that of neck pain. Except in cases with trauma, the degree of pain from thoracolumbar lesions is rarely sufficient to cause unprovoked yelping, but a response may be noted if the patient is picked up.
- *Lumbosacral pain* tends to produce an appearance that the hindquarters are being 'tucked in' and the tail is usually held down. This stance would also be seen in many cases with bilateral orthopaedic disease of the hindlimbs.
- *Lameness or lifting, knuckling or trembling of*

a limb may indicate nerve root involvement.

- *Incoordination* may be noted during ambulation as a swaying gait, tendency to fall on cornering, dragging of the nails, tripping over a low step or kerb, or intermittent knuckling of a paw.
- *Muscle tone.* Atrophy may be noted and, if marked, will usually denote LMN involvement. Alternatively, increased muscle tone may be noted in some instances. For example, following severe injury to the thoracic spinal cord with damage to ascending inhibitory pathways from the hindlimbs to the forelimbs, extensor muscle tone in the forelimbs may be vastly increased (the Schiff-Sherrington phenomenon). This indicates severe cord damage and a very guarded prognosis. The phenomenon wanes after 24–48 hours which may be misinterpreted as a sign of improvement by the unwary. In such cases with severe injury to the thoracic spinal cord, where several segments may be involved, there may be a resulting paralysis of the associated intercostal muscles. If this is the case then paradoxical respiration may be seen whereby the rib cage collapses during inspiration and expands during expiration.

Head

Cranial nerves

Evaluating cranial nerve function is aimed at ruling out the possibility of multifocal or generalised neurological disease. Each test will be taken in turn and which cranial nerves are required to produce a normal response will be outlined. This discussion will not be comprehensive and for information relating to the examination of dogs suspected of having intracranial disease, the reader is referred elsewhere.

- *Menace reflex.* The eye is threatened and this should prompt a blink. An intact reflex relies on the integrity of cranial nerves II and VII.
- *Pupillary light reflex.* Shining a pen torch into each eye should cause a reduction in the size of both pupils. An intact reflex relies on the integrity of cranial nerves II and III.
- *Nystagmus.* The presence of nystagmus indicates pathology involving one or all of the following cranial nerves: III, IV, VI and VIII.
- *Facial sensation.* Touching of the skin over the face usually causes a twitch or blink. An intact reflex relies on the integrity of cranial nerves V and VII.
- *Hearing.* An ability to hear requires a functional nerve VIII.
- *Swallowing or gag reflex.* An ability to swallow food normally or produce a gag reflex relies on the integrity of cranial nerves IX and X.
- *Tongue.* Normal function of the tongue, seen as an ability to prehend food adequately, relies on the integrity of cranial nerve XII.
- *Facial symmetry.* Lack of this is usually indicative of pathology involving nerve VII.
- *Head tilt.* May be indicative of pathology involving nerve VIII.

This series of tests/observations should rule out pathology in all but cranial nerve I (olfactory). The latter is difficult to evaluate and rarely of significance in clinical cases.

Eye

The eye should be examined grossly and ophthalmoscopically. Horner's syndrome (reduced pupillary diameter, ptosis, retracted globe, protrusion of the third eyelid) indicates pathology involving the sympathetic nerve supply to the eye. Although such pathology could be in the vicinity of the eye itself or else the middle ear, through which the supply courses, it could also indicate a cervical or cranial thoracic lesion as the nerve supply travels down the cervical spine before exiting the vertebral column with nerve roots T1–3 and then passing back up the neck as part of the vagosympathetic trunk. Fundic examination may reveal evidence of papilloedema (possibly indicating raised intracranial pressure) or generalised inflammatory disease of the central nervous system (CNS) (e.g. toxoplasmosis).

Jaw

An ability to open and close the mouth should be observed. A patient may be unable to close his/her mouth in the presence of trigeminal palsy (cranial nerve V) but also in the presence of mandibular

fractures or temporomandibular luxation. An inability, or reluctance, to open the mouth may result from temporal muscle myositis or cranimandibular osteopathy.

Vertebral column

If the condition is painful then *palpation and manipulation* of the vertebral column should detect regions of muscle spasm and elicit a painful reaction which should indicate the region of interest, possibly more specifically than the neurological signs. However, it must be remembered that a site of pain, say at the lumbosacral junction due to degenerative pathology, may be longstanding and not necessarily related to an acute onset hindlimb incoordination which may be the result, for example, of a thoracolumbar ischaemic myelopathy.

The panniculus reflex

Pinching of the skin about 1–2 cm off the dorsal midline should cause a twitching of the panniculus carnosus muscle bilaterally. The sensory input for this reflex is segmental from T3 to L1, with the skin just behind the last rib relating to the T11 nerve root and the L1 nerve root relating to an area just cranial to the iliac crest (Fig. 17.3), whilst the motor outflow originates from spinal cord segments C8 and T1. Thus, an intact reflex depends on the integrity of the segmental nerves, the spinal cord from T1 to L1 and the lateral thoracic nerve. A spinal cord lesion sufficient to cause marked hindlimb paresis will generally cause an absence of the panniculus reflex to a stimulus whose sensory input to the cord is caudal to the lesion.

It must be remembered that the point at which this reflex is lost indicates the cranial margin of the lesion and its greatest use perhaps comes from allowing the clinician to observe whether this cranial limit is migrating forwards over a period of days. If it does then the explanation may be an ascending malacia which would make the prognosis hopeless (see later).

Similarly, it must be borne in mind that injury to the lateral thoracic nerve, for example in some cases with brachial plexus avulsion or neoplasia, the reflex will be absent unilaterally.

Limbs

Voluntary movement

Voluntary movement in all four limbs may be observed as the patient moves, or tries to move, around the consulting room. In those that are non-ambulatory it may be necessary to support their

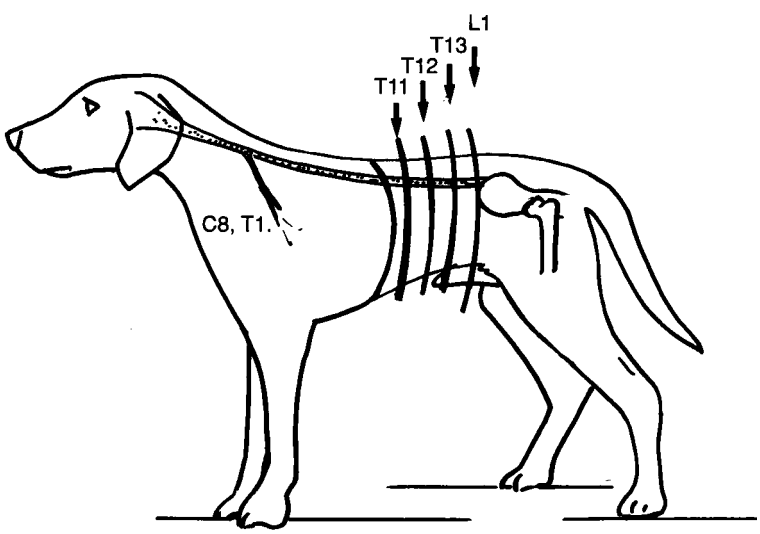

Fig. 17.3 Illustration of the panniculus reflex showing the sensory fields relating to spinal nerves T11–L1 and the single motor output at C8–T1.

weight in order to better evaluate whether some movement is present. For example, a small breed dog that is 'off his hindlimbs' because of a thoracolumbar disc extrusion should be made to walk forwards on his forelimbs whilst the rear end is supported by either holding his tail or placing two or more fingers under his pelvis. As he moves forwards any purposeful motor activity in one or both hindlimbs should be observed.

Conscious proprioception

This is, essentially, the spatial awareness of joint position and an ability to counteract any perceived abnormality thereof. It involves all levels of the nervous system (i.e. brain, spinal cord and peripheral nerves). Postural reactions are used to test proprioception but, being complex, they do not test any specific part of the nervous system. Rather they are useful for identifying neurological disease per se. In most cases with spinal cord lesions, deficits in these reactions in combination with a lack of evidence of LMN dysfunction is taken to indicate UMN involvement.

Conscious proprioception in each limb can be evaluated using one or more of the following tests:

- *Paw knuckling* involves positioning the paw on the ground in a knuckled fashion. It should be picked up immediately and returned to its normal position.
- *Reflex stepping* involves placing the paw on a piece of paper which is then drawn laterally. As the dog's centre of gravity moves laterally the limb should be picked up and replaced so as to support the animal's weight.
- The *sway response* involves gently swaying the hindquarters or shoulder region and observing that the dog transfers weight from one side to the other in order to oppose the manoeuvre.
- *Wheelbarrowing* the dog by supporting the rear end under the abdomen and walking him/her forwards may reveal subtle forelimb deficits.
- The *extensor postural thrust reaction* involves holding the dog around the thorax and lowering the hindlimbs to the table or floor. As the paws touch the surface they should push against it and then step backwards. A lack of such a reaction indicates hindlimb deficits.
- *Visual and tactile placing reactions.* If the dog is lifted off the table and then moved towards the table edge the leading limb should be flexed and placed onto the table top. This can easily be repeated for each of the four limbs (in small dogs at least!). Each test is then repeated with the dog's eyes covered when the response should occur as the limb touches the table.

Other postural reactions, such as hopping/hemihopping, hemistanding and hemiwalking, may be used to supplement these tests for proprioception as in some cases they may detect more subtle abnormalities, though they are possibly of most use when trying to localise intracranial lesions.

Spinal reflexes

These test the reflex arcs, of which LMNs make up the efferent arm (see earlier) and thus are used primarily as tests for LMN function, though it must be remembered that sensory deficits or damage to specific spinal cord segments would also affect these reflexes. The response to any particular stimulus may be:

- Normal.
- Reduced or absent: indicating partial or complete loss of sensory and/or motor function.
- Increased: indicating loss of the inhibitory descending pathways from the brain, i.e. a UMN lesion, or a 'pesudohyperreflexia' due to loss of tone in antagonistic muscle groups, for example sciatic palsy may result in an exaggerated patellar reflex.

Pedal or withdrawal reflex A minimally painful stimulus is applied to the digits causing the limb to be withdrawn. This test is predominantly evaluating function of the nerves supplying the flexor muscle groups. However, sensation in the manus also involves the radial nerve and sensation in the medial toe of the pes involves the femoral nerve. The reflex also requires the integrity of spinal cord segments C6–T1, in the case of the forelimb, and segments L6–S1 (overlying the L4–5 disc space) in the case of the hindlimb. As each limb is withdrawn, flexion of all joints should be observed. It is not uncommon to see partial dysfunction of the sciatic nerve evidenced

by poor flexion of the hock whilst stifle flexion is normal. This may reflect damage to the peroneal branch of the sciatic nerve specifically or, more commonly, a greater susceptibility to injury of the peroneal nerve fibres within the lumbosacral trunk due to their having a greater diameter than those contributing to the tibial branch of the sciatic nerve. Injury to the lumbosacral trunk is most commonly associated with pelvic fractures.

It cannot be overstressed that the presence of a withdrawal reflex does not *indicate that the patient can feel the painful stimulus.*

Patellar reflex This is a myotactic or 'stretch' reflex. In a relaxed patient with the stifle in mild flexion, tapping of the straight patellar tendon causes a 'knee jerk'. The sensory organs are stretch receptors in the quadriceps muscle spindles and the effector organ is the quadriceps muscle mass. Both the afferent and efferent arms of the reflex arc are carried in the femoral nerve but the integrity of spinal cord segments L4–6 (all within the fourth lumbar vertebra) is also required.

Other tendon reflexes Stretch reflexes, similar to the one described for the quadriceps tendon, can be elicitied in the biceps and triceps tendons. However, the response is inconsistent in normal dogs and they are probably not worthwhile including in the neurological evaluation.

Conscious pain sensation (CPS)

This may be considered one of the most important single tests in terms of prognosis, especially in cases of acute spinal trauma resulting from a disc extrusion or vertebral fracture. In those cases where conscious pain sensation (CPS) is present there is a good chance of dogs recovering a normal or near normal ability to ambulate. If CPS has been lost then the likelihood of regaining a satisfactory ability to ambulate is poor to moderate, depending on the nature and duration of the injury and treatment options available. The reason for this is that the spinal tracts carrying pain sensory fibres are well distributed throughout the cord and the fibres themselves are of small diameter and therefore very resistant to injury. Therefore, loss of CPS indicates severe and widespread damage to the spinal cord.

Testing for the presence of CPS has to be undertaken most diligently in any cases showing loss of voluntary movement in a limb since those showing voluntary movement will not have lost CPS in that limb. The lightest stimulus is that of pinching the interdigital web of the paw with finger nails (testing superficial pain sensation). *The response needs to indicate a conscious awareness of the painful stimulus, for example biting or snarling, rather than merely a pedal withdrawal reflex.* If there is no response to finger pressure then artery forceps are applied to each of the nail beds in turn (deep pain sensation). If there is still no response from the dog's head end then 'slip joint' pliers are applied to the metatarsi.

Generally speaking it is in the hindlimbs that CPS is tested for since loss of this response in the forelimbs would have to be caused by such a severe injury in the cervical spine that the phrenic outflow should have been affected, causing the patient to have stopped breathing. If no response to severe pressure can be found in either hindlimb or the tail then a total absence of CPS has been confirmed. If there is any concern that analgesics administered to the dog, or stoicism, is causing an apparent loss of CPS in the hindlimbs then a simple test is to try stimulating the forelimbs – but starting with the mildest stimulus first of all!

Bladder

Whether a patient has voluntary control over its bladder function can be difficult to evaluate in the early stages since associated urinary retention with overflow (URO) will only develop once the bladder is full. In observing patients which may or may not have URO it is important to differentiate 'bed wetting', i.e. an ability to control micturition but an inability to ambulate to an appropriate site before doing so, from true overflow. Dogs that are bed wetting will have dry beds for long periods of time and then very wet beds followed by another period of dryness once the bed has been changed. The beds of dogs with URO will become progressively wetter with time and will be damp again soon after being changed (assuming the bladder is not manually expressed). In addition, those with URO tend to pass urine when lifted

with support under their abdomen once the bladder is full, though some dogs that are simply 'holding on' may also do this if the raised intra-abdominal pressure exceeds their voluntary urethral tone.

Lesions involving the UMN supply to the bladder (i.e. cranial to the sacral cord segments) will tend to cause URO where manual expression of the bladder is made difficult by the increase in urethral tone caused by a loss of inhibitory influence of the UMNs on the LMNs. Lesions involving the LMN supply to the bladder (i.e. caudal to the sacral cord segments/the fifth lumbar vertebra) will tend to cause URO where manual expression of the bladder is relatively easy due to reduced urethral tone caused by a loss of LMN function. Determining whether URO is associated with a UMN or LMN lesion is important, both in terms of medically assisted management and also prognosis (with UMN bladders being far more likely to recover than LMN bladders).

An indication of whether LMN function is likely to be compromised may be gained by observing anal sphincter tone and the perineal reflex since their presence requires a functional pudendal nerve (originating from spinal cord segments S1–3) which also supplies the urethra. The perineal reflex involves pinching or pricking the skin of the perineum which should result in reflex twitching of the anus and downward movement of the tail base.

Localisation of the lesion

From the neurological examination detailed above conclusions will have been drawn as to whether each limb is:

- Normal: normal reflexes
- Showing LMN dysfunction: reduced or absent spinal reflexes
- Showing UMN dysfunction: reduced proprioception with normal or exaggerated spinal reflexes

Alongside other features, such as a site of pain and/or a panniculus reflex 'cut-out', these features aid in determining the site of the lesion according to the details in Table 17.1.

Table 17.1 Localisation of a spinal cord lesion according to lower motor neuron (LMN) and upper motor neuron (UMN) involvement.

Level of cord injury*	Forelimb	Hindlimb
C1–C5	UMN	UMN
C6–T2	LMN	UMN
T3–L3	Normal	UMN
L4–S3	Normal	LMN

* This relates to spinal cord segments rather than vertebrae.

Severity of a spinal cord lesion

This is an important aspect to the neurological evaluation as, particularly in acute cases involving intervertebral disc extrusions or vertebral fractures, it influences the prognosis. Several systems have been described over the years aimed at grading the degree of spinal cord white matter damage, and then some grades have been further subdivided. Determining whether bladder paralysis is present may be difficult in the first instance (as discussed above) and, although its presence may make management more difficult, it does not influence the prognosis compared to the presence of hindlimb paralysis alone. A typical grading system, which will be adopted in this text, is detailed below:

- **Grade 1**: pain only
- **Grade 2**: ambulatory paraparesis/quadriparesis
- **Grade 3**: non-ambulatory paraparesis/quadriparesis
- **Grade 4**: paraplegia/quadriplegia (i.e. voluntary movement absent)
- **Grade 5**: paraplegia/quadriplegia + urinary retention with overflow (URO)
- **Grade 6**: paraplegia/quadriplegia + URO + loss of conscious pain sensation (CPS)
- **Grade 7**: ascending/descending myelomalacia

The features of grade 7 pathology (malacia) are discussed in Chapter 5 (p. 36).

Further reading

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Chapter 18
Differential Diagnosis

On completing the general and neurological examination of a patient suffering with a spinal cord lesion, the clinician should be able to determine which region of the spinal cord is involved (i.e. C1–C5, C6–T2, T3–L3, L4–S3, bearing in mind that these relate to spinal cord segments and not vertebrae). The list of differential diagnoses is fairly extensive but certain features of the particular case (e.g. whether the patient is showing evidence of pain, rate of onset of clinical signs, patient age and region of spinal cord involved) may assist in prioritising the list of potential diagnoses and thus expedite further investigation (see Tables 18.1 and 18.2). Most of these will be dealt with, to varying degrees, within this section and the reader is directed to the index to locate further information on each specific problem. Those not covered within these pages are relatively uncommon and the reader is directed to other standard neurology texts for further information.

Table 18.1 Differential diagnosis of a vertebral column lesion (in alphabetical order and under the catagory headings defined by the 'VITAMIN D' mnemonic).

Mnemonic	Type of pathology	Diagnosis (examples)
V	Vascular	Ischaemic myelopathy (a.k.a. fibrocartilagenous embolism)
		Spinal arteritis
		Spontaneous spinal haemorrhage
I	Inflammatory	Granulomatous meningoencephalomyelitis
		Steroid-responsive meningitis
	Immune-mediated	Degenerative myelopathy??
		Feline infectious peritonitis??
	Infectious	Bacterial meningitis
		Canine distemper virus
		Discospondylitis – bacterial (fungal)
		Feline infectious peritonitis
		Protozoal (toxoplasmosis, neosporosis)
	Idiopathic	The aetiology of many of the diagnoses listed here is far from fully understood
	Iatrogenic	!!
T	Traumatic	Fracture/luxation
		Intervertebral disc extrusion
A	Anomalous (congenital)	Arachnoid cysts
		Atlantoaxial subluxation
		Cervical spondylopathy
		Lumbosacral stenosis
		Miscellaneous vertebral malformations (block, transitional, etc.)
		Occipitoatlantal subluxation
		Spina bifida
		Spinal dysraphism
		Syringomyelia

Table 18.1 *Contd.*

Mnemonic	Type of Pathology	Diagnosis (examples)
M	Metabolic	Calcinosis circumscripta Lysosomal storage diseases Osteochondrosis (articular facets, end plates)
I	(As above)	
N	Neoplasia	Vertebral osteochondroma osteosarcoma Extradural haemangiosarcoma lymphosarcoma Dural meningioma meningiosarcoma Intradural astrocytoma glioma nephroblastoma
	Nutritional	Hypervitaminosis A
D	Degenerative	Cervical spondylopathy Degenerative myelopathy Disc disease (protrusion) Lumbosacral stenosis

Table 18.2 Likely differential diagnoses of vertebral column lesions according to: (1) whether pain is a major feature; (2) the rate of onset of the clinical signs; and (3) the age of the patient (with * denoting likely diagnoses in immature patients).

Painful		**Non-painful**	
Acute onset	Chronic onset	Acute onset	Chronic onset
Disc disease (extrusion)	Disc disease (extrusion/protrusion)	Ischaemic myelopathy (may appear painful at first)	Cervical spondylopathy
Neoplasia	Neoplasia		Neoplasia
Inflammatory CNS disease*	Cervical spondylopathy		Disc disease (protrusion)
Trauma	Lumbosacral stenosis		Congenital vertebral malformations*
Discospondylitis*	Inflammatory CNS disease*		Degenerative myelopathy (DM or CDRM)
Atlantoaxial subluxation*	Discospondylitis*		Breed-associated myelopathy*
Cervical spondylopathy	Atlantoaxial subluxation*		Lysosomal storage disease
Lumbosacral stenosis	Calcinosis circumscripta*		Arachnoid cysts*
			Spina bifida*
			Spinal dysraphism*
			Syringomyelia

CNS = central nervous system; DM = degenerative myelopathy; CDRM = chronic degenerative radiculomyelopathy.

Further reading

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A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny,Steven J. Butterworth

Chapter 19
Further Investigation of Spinal Diseases

The general and neurological examination of a patient suffering with a spinal cord lesion should allow detection of which region of the spinal cord is involved and this, coupled with the patient details and the list of differential diagnoses given in Chapter 18 should enable the clinician to draw up a short list of likely diagnoses. Reaching a definitive diagnosis will then require further investigation involving one or more of the following:

- Imaging:
  - plain radiography
  - contrast radiography:
    - myelography
    - epidurography
    - discography
    - vertebral sinography
  - computerised tomography
  - magnetic resonance imaging
  - scintigraphy
- Cerebrospinal fluid analysis
- Electrophysiology

Plain radiography

The complex anatomy of the vertebrae makes careful patient positioning for radiography essential, otherwise the diagnostic value of the films obtained will be compromised. The *only time* when such compromise may be justified would be in the traumatised patient where there may be concern over iatrogenic worsening of any neurological damage by any unnecessary manipulation. A selection of foam pads/wedges, sandbags and radiopaque troughs must be available. The exact requirements for positioning of the patient will depend on the area being radiographed but general rules apply to many films. Ventrodorsal views do not often cause problems in this respect as long as troughs are used to support the patient and sandbags used to hold the limbs evenly positioned. It is in the lateral views that patients are more often malpositioned. In order to keep the vertebral column parallel to the film it is necessary to pad the mid-cervical and mid-lumbar regions. If this is not done adequately then the resulting 'sag' in the column will create artefactual narrowing of the disc spaces and an overdiagnosis of intervertebral disc (IVD) extrusion. The only part of the spine which is often impossible to make parallel to the film is the thoracic segment due to the conical nature of the chest. Although this does make certain interpretations difficult, it is perhaps fortunate that disc prolapses are rare in this region. Rotation of the vertebral column also creates numerable artefacts and avoidance of this generally requires a foam wedge to support the ventral thorax together with foam pads or sandbags to keep the limbs parallel. In the cervical region it is also necessary to support the nose on a foam wedge as rotation of the skull will create rotation in the cranial cervical spine. A combination of this and mid-cervical 'sagging' is responsible for the overdiagnosis of such conditions as an IVD prolapse or atlantoaxial subluxation (Fig. 19.1). In some dogs, good, straight views of the cervical spine are best achieved by the application of gentle traction. A tape is tied around the upper jaw, just behind the canine teeth and a loop formed in the other end as this passes over the end of the table. With the forelimbs tied caudally and/or the table tipped slightly (head up) the dog's nose can be 'drawn' gently up the table and held in this position with a sandbag placed through the loop at the end of the tape. A foam

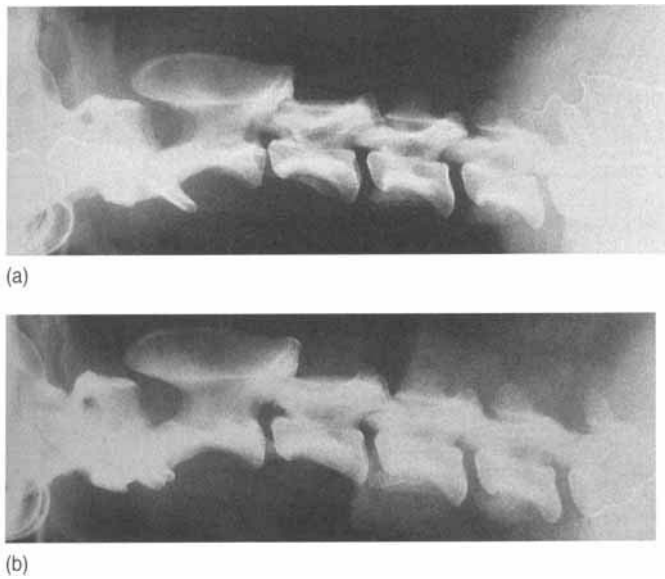


Fig. 19.1 Plain radiographs of a West Highland White Terrier's cervical spine taken (a) with reasonable efforts made to position the dog correctly and (b) with the neck under mild traction. Note that in (a) the C2–3 intervertebral disc space appears narrow, whereas in (b) the appearance is normal.

wedge is still required under the dog's nose to avoid rotation. Aids to patient positioning for radiography of the vertebral column are illustrated in Fig. 19.2.

Obtaining diagnostic films also requires attention to collimation of the primary beam. Views of the abdomen are not adequate to evaluate the lumbar spine critically, and although abnormalities may be suspected the radiography will have to be repeated for confirmation. Radiographing too long a portion of vertebral column on the same film will create distortion due to divergence of the X-ray beam.

In addition to lateral and ventrodorsal projections, sometimes it may be necessary to consider oblique views, especially in trauma patients when the integrity of the articular facets might be in question.

The use of grids, choice of screen/film combinations and the avoidance of processing faults are also important topics. They are, however, beyond the remit of this chapter and for further information the reader is directed to standard radiology texts.

Radiographic interpretation

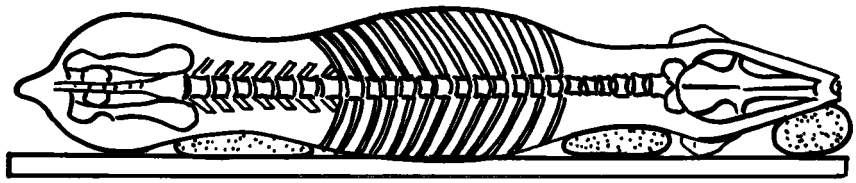
In observing a radiographic film of the vertebral column there are several features to consider. These can either be taken in turn or else each examined repeatedly moving along the vertebral column from cranial to caudal. The following features of the vertebrae should be examined:

- Vertebral body:
  - (number)
  - alignment
  - shape
  - internal architecture (destruction, fracture)
- Vertebral processes:
  - fracture
  - destruction
  - absence (especially ribs)
- Disc space:
  - width (increased or decreased)
  - mineralisation
- Intervertebral foramen:
  - size (larger or smaller)
  - opacification
- Articular facets:
  - subluxation
  - fracture

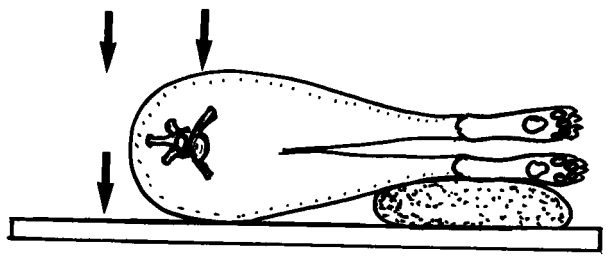
'Normal abnormalities' which may be observed include:

- Variation in number
- Block vertebrae
- Hemivertebrae
- Transitional vertebrae

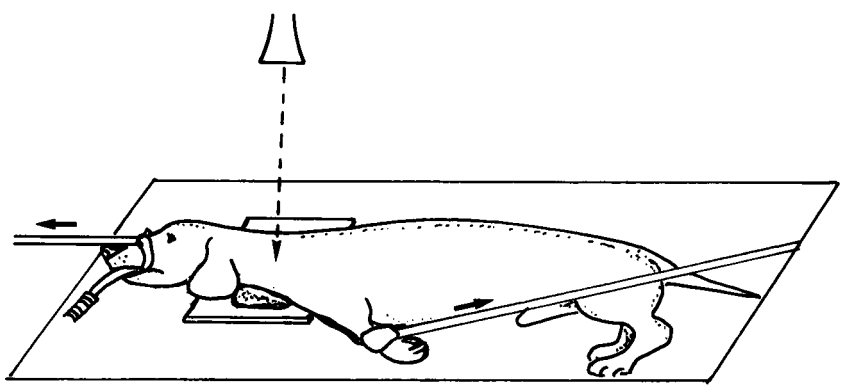
However, even these may be important in terms of



(a) Foam pads/wedges should be used to prevent 'sagging' of the cervical and lumbar spine and also to support the nose so as to prevent rotation of the head, thus keeping the vertebral column parallel to the film.



(b) Sandbags or foam pads should be used to support the limbs so as to eliminate rotation, keep the sagittal plane of the vertebral column parallel to the film and perpendicular to the primary beam.



(c) Gentle traction on the dog's head can help to eliminate 'sagging' of the cervical spine.

Fig. 19.2 Illustration of aids for good radiographic positioning.

surgical landmarks or possibly creating asymmetry (e.g. at the lumbosacral junction) which may contribute, in the long term, to degenerative changes.

Myelography

Myelography involves the injection of a radiopaque contrast agent into the subarachnoid space such that it mixes with the cerebrospinal fluid and thus outlines the spinal cord. It is generally employed to extend the radiographic study in one of three situations:

- When a patient is suffering pain or neurological signs that are related to the vertebral column and a diagnosis cannot be reached from plain radiographs.
- When an abnormality has been noted on plain radiographs but its significance is questionable, or where multiple abnormalities have been noted and the study is used to determine their relative significance.

- When disc disease is apparent but exactly which disc is protruding, or on which side of the canal the extruded material is lying, needs to be defined before decompressive surgery is undertaken.

The technique appears to be of most use in helping to evaluate the significance of plain radiographic abnormalities or when a specific focus of pain is clinically detectable in the absence of plain radiographic abnormalities. Conversely, it appears to be of least use in cases with chronic, progressive neurological signs without a focus of pain or plain radiographic changes.

Myelography is *not* a perfectly safe procedure and an estimate of mortality, either directly, or indirectly as a result of euthanasia due to a permanent neurological deterioration, would be 1%, although this is somewhat dependent on the site of injection and the contrast used as well as operator experience. Before performing myelography it may be well worthwhile asking the question 'will the result of this study affect what I decide to do?' and if the answer is no then the study is probably not justified. Furthermore, *diagnostic myelography requires good quality radiography and it is inappropriate to rely on myelography to demonstrate lesions that are not evident on poor quality plain radiographs.*

Materials

The contrast agents used today are much less toxic than the ones used in the past and the non-ionic, isosmolar agents are the ones of choice. Agents with an iodine concentration of 300 mg iodine/ml are often used but those with slightly higher concentrations (370 mg iodine/ml) for dogs and lower concentrations (270 mg iodine/ml) for small dogs and cats may also be employed. The two agents most commonly used are iohexol (Omnipaque, Nycomed) and iopamidol (Niopam, E. Merck Pharmaceuticals).

The contrast agent may be introduced by way of a hypodermic needle but spinal needles are easier to use. In most cases, access to 19g × 3″ and 22g × 1″ spinal needles (Becton Dickinson) will be all that is required but occasionally a 22g × 3″ spinal needle may be more suitable.

The volume used depends on the size of the patient and the site of interest. For a cisternal myelogram, 2–3 ml would be suitable for a cat or small dog, through to 7–8 ml for a Labrador, to 10 ml for an Irish Wolfhound. Less may be used if a cervical lesion is suspected. For a lumbar myelogram similar volumes may be used but, in general, this route is used to define thoracolumbar lesions and less agent may then be required due to the lesion's proximity to the site of injection.

Technique (Wright, 1984a,b)

Cisternal puncture

The dog is placed in lateral recumbency under general anaesthesia with the table tipped 5–10° (head up). The dog's neck is flexed as fully as possible. The anatomical landmarks are the wings of the atlas and the external occipital protuberance. A line drawn between the two wings crosses a point in the midline. Halfway between this and the occiput is the point of entry for the needle (Fig. 19.3). The area is clipped and prepared aseptically. A spinal needle is most useful for this technique

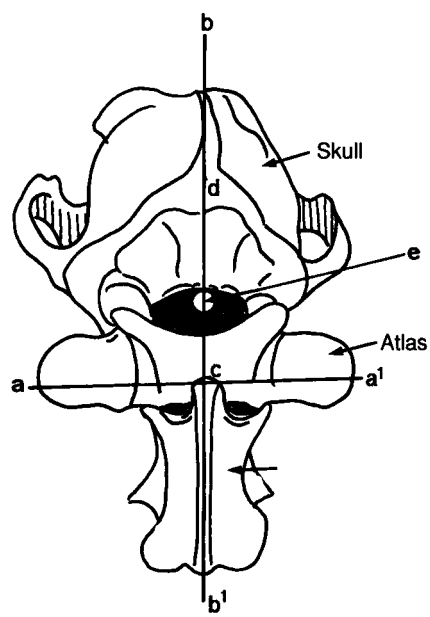


Fig. 19.3 Illustration of the landmarks used for cisternal punctures. a–a¹ – Line between wings of the atlas; b–b¹ – midline; c – point where a–a¹ crosses midline; d – caudal-most point of the occiput; e – point of entry, halfway between c and d.

(Becton Dickinson 1″ and 3″ spinal needles). The needle is introduced at a right angle to the skin. Once through the skin the stylet is withdrawn completely. The needle is then advanced until it 'pops' through the ligamentum flavum. Cerebrospinal fluid (CSF) should run out freely and can be collected for analysis (Fig. 19.4). Cerebrospinal fluid is allowed to run until the drip rate begins to slow. On advancing the needle, bone may be struck. It may be redirected or walked off the bone to find the cisterna magnum. If blood is seen then the needle is off the midline and must be withdrawn. Another attempt may be made, preferably with a new needle. Sometimes a small vessel is hit as the dura mater is penetrated. In these cases, the CSF will be blood-tinged to begin with but will run clear after a few moments.

Once the contrast has been introduced the needle is withdrawn and radiographs are taken of the cervical region. Lateral views are taken following the contrast down the spine to the lumbosacral junction. If an abnormality is found a ventrodorsal view is taken. If the contrast stops then the angle of the table is increased. Alternatively, traction may be applied to the vertebral column by securing a tape bandage to the dog's upper jaw, drawing the dog's head up the table by the scruff and then maintaining this position by suspending sandbags through the loop of bandage passing over the end of the table. In general if the contrast fails to flow with the table at an angle of 30–45°, with traction applied to the patient's spine, then the study should be abandoned, perhaps in favour of a lumbar myelogram.

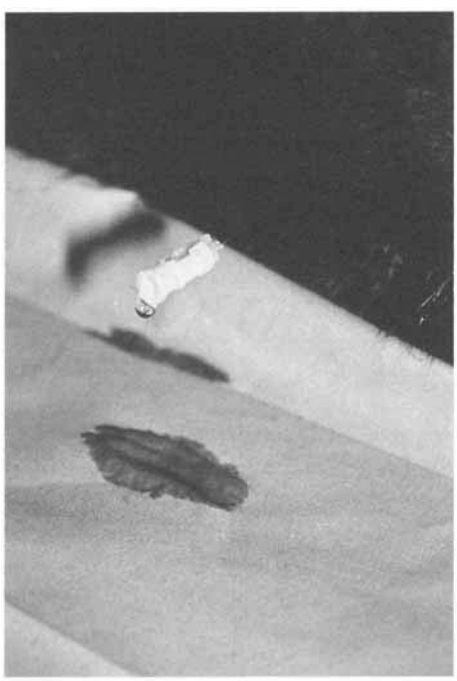


Fig. 19.4 Photograph of spinal needle in place for cisternal puncture. The dog is being held in lateral recumbency with the neck flexed such that the nose touches the sternum without allowing rotation of the head. Note clear cerebrospinal fluid (CSF) appearing in the hub of the needle.

Lumbar puncture (Figs 19.5 and 19.6)

The dog is positioned in lateral recumbency with its back hyperflexed. Alternatively, with the dog in sternal recumbency, the hindlimbs may be drawn cranially to achieve the same result. The aim is to pass the needle through the dorsal intervertebral space between L5 and L6 (the space between L4 and L5, or L6 and L7 may be used, although the former is more likely to incur damage to the spinal cord whilst the latter is more likely to result in leakage of contrast into the epidural space). The area is prepared aseptically. The landmarks are the dorsal spinous processes (that of L7 being smaller than the others and lying between the iliac wings). Although the introduction of the needle at L5–6 has always been considered preferable to further cranial, owing to the reduced likelihood of iatrogenic spinal cord damage, a recent paper (McCartney, 1997) described the introduction of contrast agent between T13 and L5 without the observation of such iatrogenic damage. The advantage of introducing the needle further cranial is that greater flexion of the vertebral column can generally be achieved, making needle placement easier, and there is less likelihood of epidural leakage of contrast agent (such leakage may produce a confusing image). Currently, the authors still prefer to introduce contrast agent at the more caudal site.

Placement of the needle can be achieved in one of several ways:

- The needle enters perpendicular to the skin in the midline halfway between the two dorsal spines.

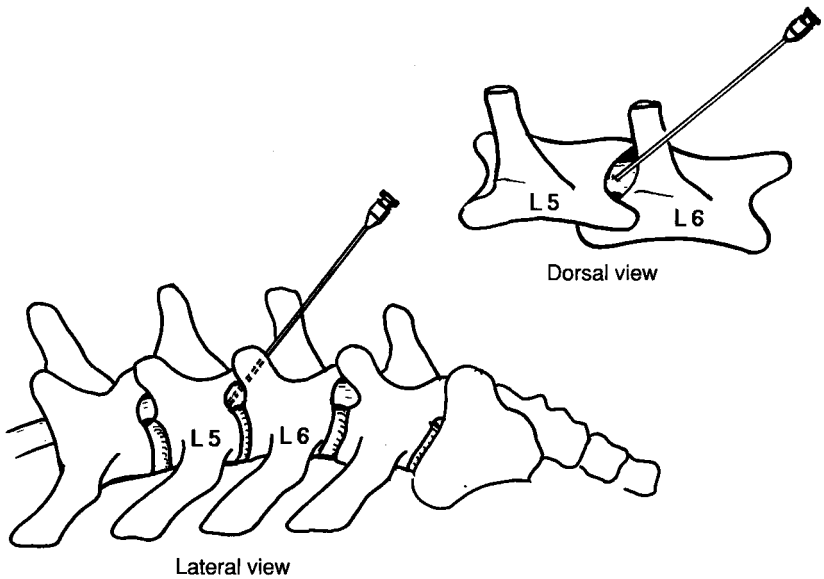


Fig. 19.5 Illustration of the landmarks used for lumbar puncture. The needle is inserted alongside the spinous process of L6 and directed cranially and ventrally through the ligamentum flavum between L5 and L6 and into the vertebral canal.

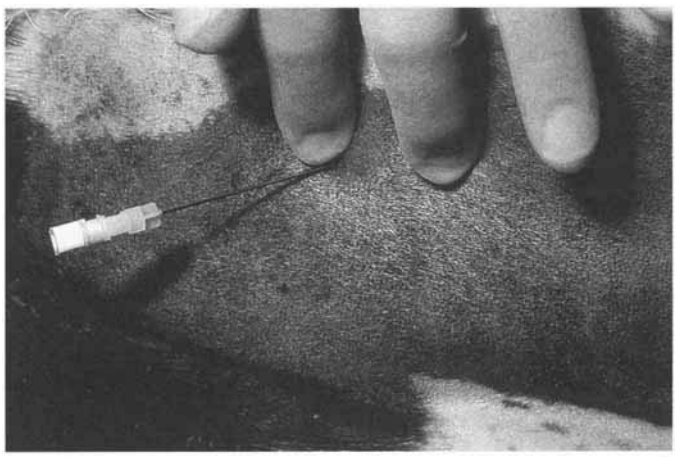
- The needle enters perpendicular to the skin just lateral to the dorsal spine, caudal to the space and is slid down the side of the process. Once bone is reached, the needle is walked forwards off the vertebra.
- The needle enters just cranial to the dorsal spine, cranial to the space and is directed backwards at an angle of 30–45°.

Once through the intervertebral ligament the needle moves on to hit bone on the floor of the canal. As the needle passes through the cord or cauda equina, the hindlimbs or tail may jerk or twitch but this will cause no long-term problems! In chondrodystrophic breeds, the needle is left against the bone whereas in the other breeds it is backed off a fraction before the contrast medium is injected. Cerebrospinal fluid may be seen and can range from a hub-full to up to a few millilitres. If no CSF is found, then a test dose of contrast agent (0.25–0.5 ml) is injected and a radiograph taken. If blood is seen in the needle hub then it is best to start afresh since the internal vertebral plexus has been penetrated. If the contrast is put into the epidural space then a sine-wave is produced as an epidurogram and the diagnostic value of the film is reduced. This may be seen even after CSF flows from the needle.

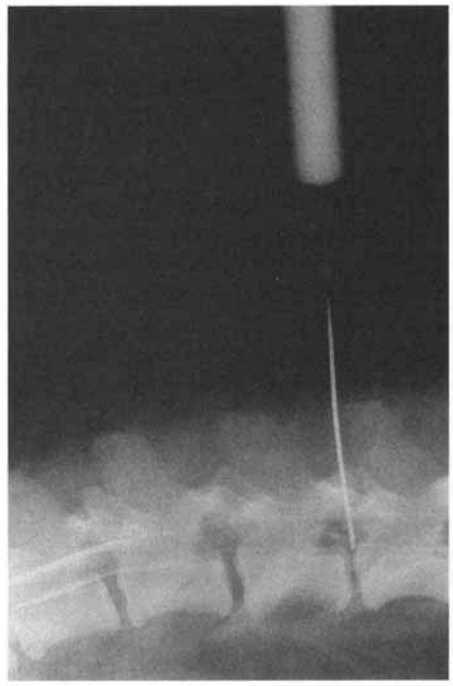
Myelographic interpretation

Essentially, the films will show lines of contrast on either side of the spinal cord (dorsally and ventrally in a lateral view, and on the right and left in a ventrodorsal view) (Fig. 19.7). The columns often show thinning at the cervicothoracic and thoracolumbar junctions due to normal anatomy/posture in lateral recumbency. In the cervical region the ventral columns always show a slight lift over the disc spaces and a kink is normally seen over the C2–3 disc. The dural sac tapers towards the lumbosacral junction and ends anywhere between L6 and S3 although most end in the region of S1.

Deviation or absence of these columns are the main abnormalities to look for (Fig. 19.8). Deviation of both columns in the same direction in either view suggests the presence of a mass outside the meninges (extradural) (Fig. 19.8a), the absence of only one column at a site with a 'golf-tee' effect in either view suggests the



(a)



(b)

Fig. 19.6 (a) Photograph of spinal needle in place for lumbar puncture. The dog is being held in right lateral recumbency with the vertebral column flexed. (b) Radiograph taken after introduction of a test dose of contrast agent to ensure correct needle placement.

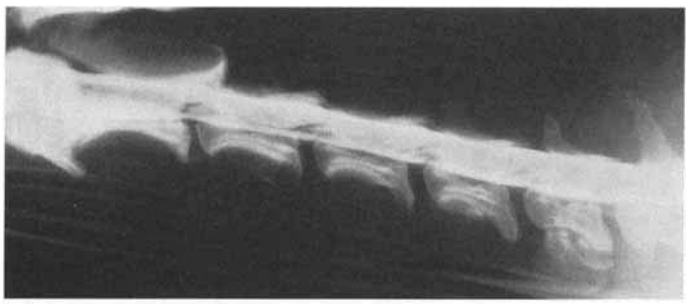
presence of an intradural but extramedullary mass (Fig. 19.8b), whilst divergence of the columns in both views suggests the presence of a mass within, or swelling of, the spinal cord (intramedullary) (Fig. 19.8c).

In addition, once the subarachnoid space in the region of interest has been opacified it may be possible to further characterise a compressive lesion in terms of the influence of positional changes. For example in cases of instability, such as in the lower cervical spine or lumbosacral spine, lateral views can be taken in flexion and extension as well as in neutral positions (Lang, 1988), or in middle-aged dogs with signs of cervical spondylopathy and a type II disc prolapse in the caudal cervical spine, the effects of linear traction can be evaluated (Butterworth, 1995). Such studies are intended to establish whether such lesions are 'dynamic' or 'traction responsive' and this may influence the choice of treatment.

The myelographic identification of such a space-occupying lesion does not give a definitive diagnosis. Myelography can only be used to localise the lesion. The decision as to the most likely cause of the lesion is dependent on the patient's history, clinical signs and plain radiographic features.

Complications

It has already been said that myelography is not a perfectly safe procedure and this appears to be the case more when cisternal, rather than lumbar, puncture is used. The latter may be due to the increased likelihood of contrast agent entering CSF surrounding the brain with cisternal myelography. Apart from serious complications caused, for example, by haemorrhage around the brain stem (which is uncommon) other, more treatable, complications include fitting on recovery from anaesthesia (controlled with diazepam) and neurological deterioration (which is usually only temporary and resolves within a few days, with or without corticosteroid treatment). It is probable that 'minor' (i.e. those readily treatable and responsive) complications will be seen in 10–25% of cases whilst 'major' complications (leading to death or permanent neurological incapacity) will occur in less than about 1% of cases (Butterworth & Gibbs, 1992).



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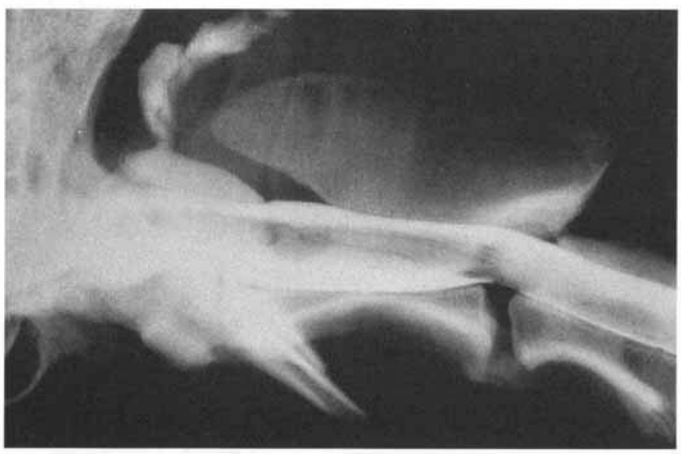

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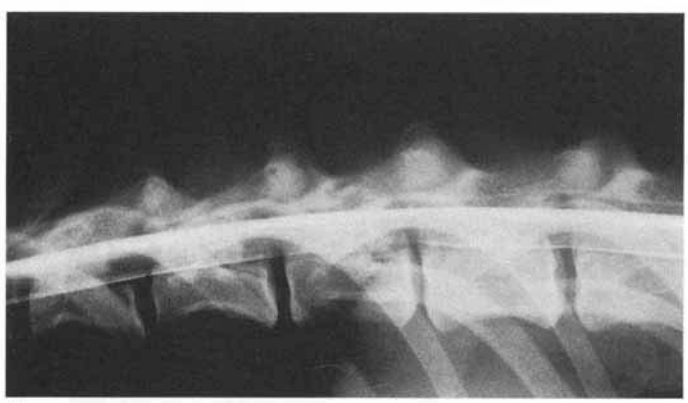


(c)

(d)

Fig. 19.7 Normal lateral myelograms.
(a) Cervical region. In this case the appearance is completely normal but often there is mild elevation of the ventral column over one or several disc spaces which is also considered a normal feature but is easily mistaken for a disc prolapse.
(b) Cranial cervical region. Note the mild 'kink' at C2–3 which is often seen and is a normal variation.
(c) Thoracolumbar region. There is no evidence of any myelographic abnormality, though one of the intervertebral disc spaces is narrowed. This image was produced by lumbar puncture. In those produced by cisternal puncture the contrast columns in the caudal thoracic spine are often faint.
(d) Lumbar region. Note widening of the spinal cord in the region of the lumbosacral intumescence and tapering of the dural sac which may terminate at any point caudal to L6.

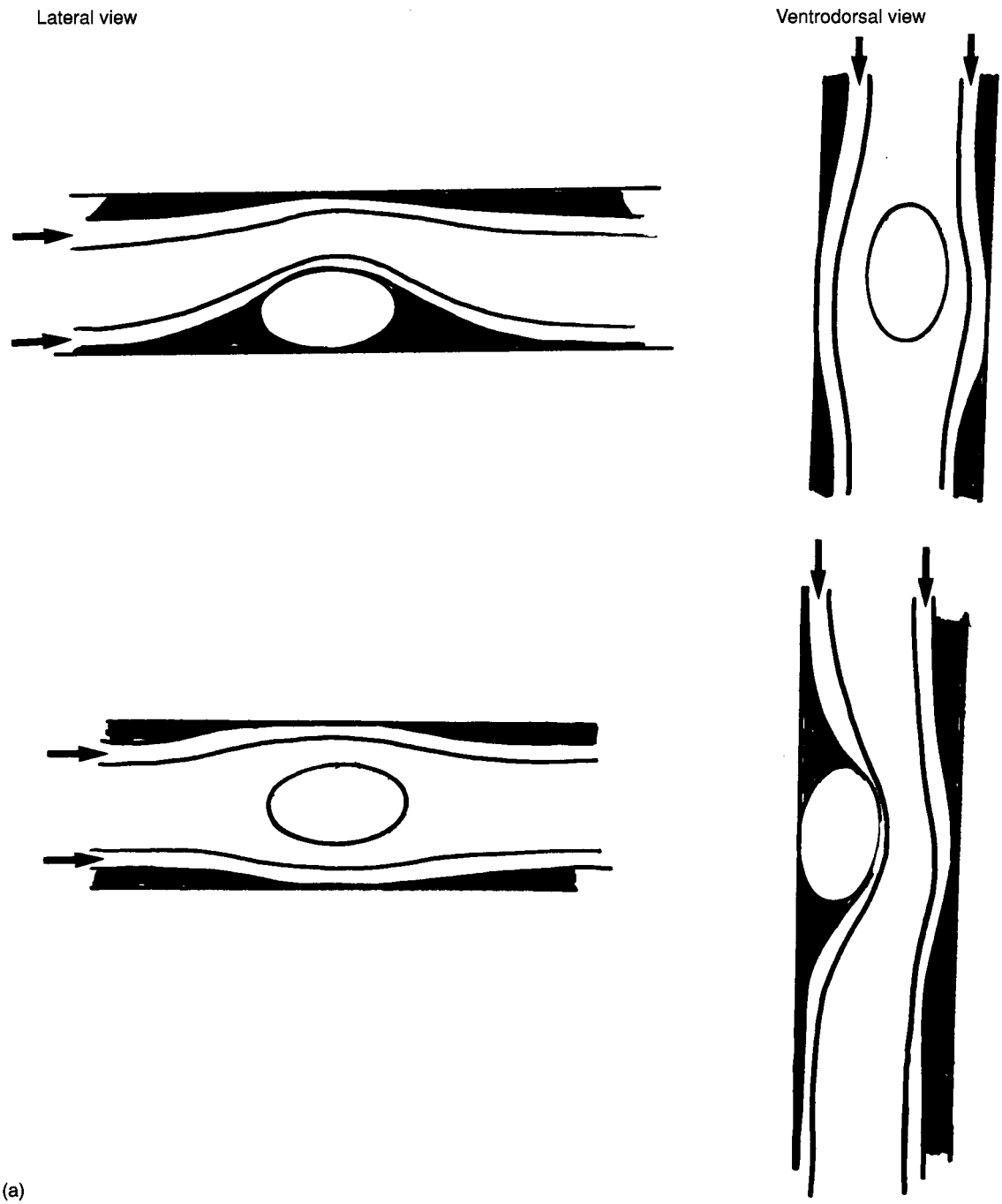
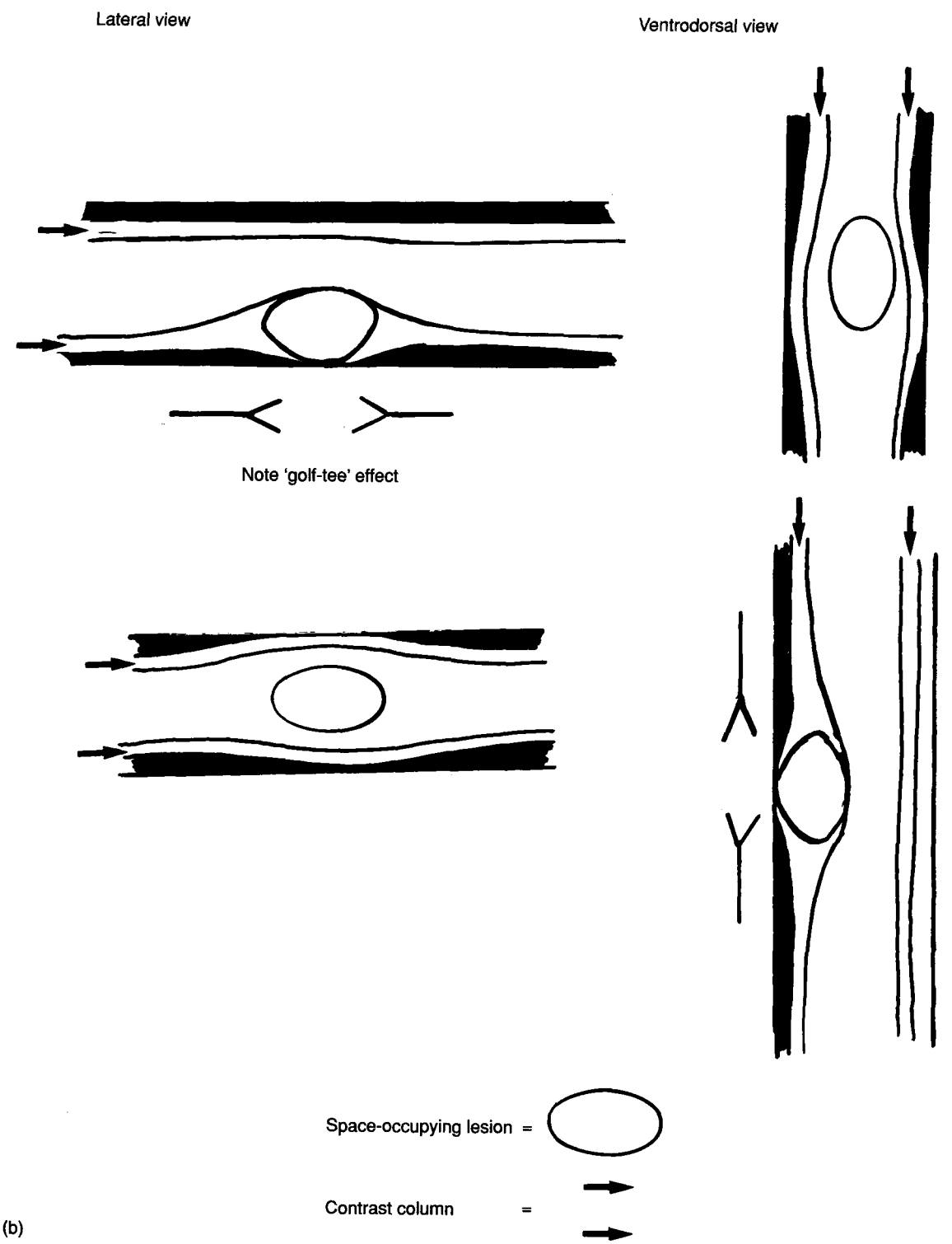
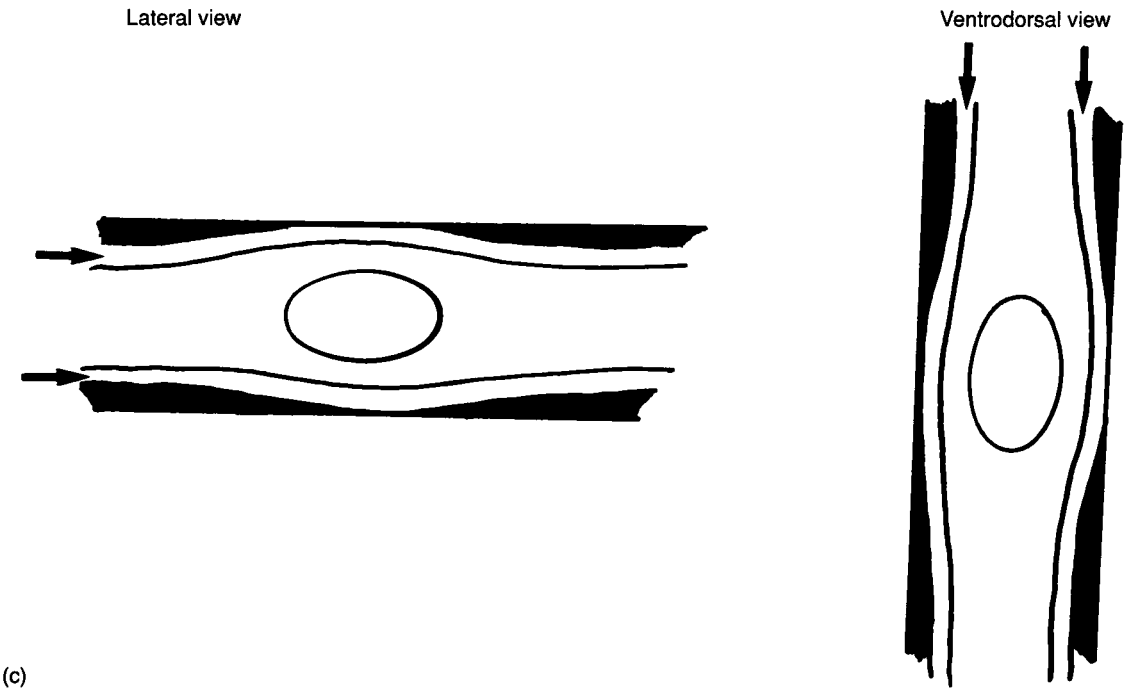


Fig. 19.8 A diagrammatic representation of myelographic abnormalities. For further details of spinal tumours see Fig. 28.1. (a) Extradural lesions,(b) intradural/extramedullary lesions, (c) intramedullary lesion.



(b)

Fig. 19.8 *Contd.*



(c)

Fig. 19.8 *Contd.*

Other contrast radiographic techniques

Epidurography (Feeney & Wise, 1981)

Epidurography involves the introduction of contrast agent into the epidural space. It is generally only used to try and delineate space-occupying lesions in the vicinity of the cauda equina where much of the space within the vertebral canal becomes epidural as the dural sac tapers off. The same contrast agents and needles can be used as described above under 'Myelography' and the injection sites are most commonly the lumbosacral junction or the cranial region of the coccygeal spine. Since CSF will not be obtained, it is usual to inject a test dose of contrast agent to ensure correct needle placement. The final dose of agent required depends on the size of the dog and the area of interest and may range from 1 to 8 or 10 ml. Films are examined for defects in the contrast. Wave patterns and cuffing around the nerve roots are normal findings (Fig. 19.9).

Interpretation of these studies can be problematic owing to the irregular appearance of the normal epidurogram. Although some radiologists use this as a preferred method of investigating clinical problems related to this region, many investigators, including the authors, use it only as a last resort.

Discography

Discography involves the injection of contrast agent into the nucleus pulposus of an intervertebral disc. It is not possible to inject into a normal disc but a degenerate nucleus, in a medium-sized breed of dog, will accept 2 ml of contrast with little resistance. The same contrast agent and needles can be used as described under 'Myelography'. The needle is passed through the ligamentum flavum, in the midline, across the vertebral canal and through the dorsal ligament and dorsal annulus. This route of injection precludes discography anywhere except the region of the cauda equina if significant spinal cord damage is to be avoided. It is, therefore,

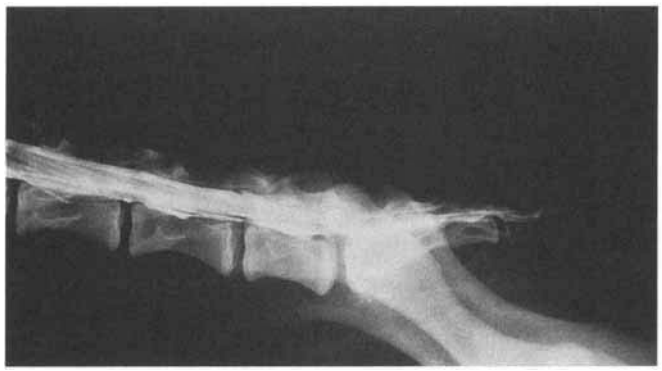


Fig. 19.9 A normal lateral epidurogram of a caudal lumbar spine.

most commonly used to investigate cases of degenerative lumbosacral disease where myelography has not revealed abnormalities that allow a definitive diagnosis to be made.

Needle placement is between the two adjacent dorsal spinous processes in the midline and radiographs need to be taken in both planes if correct needle placement is to be ensured. The dog may be placed in sternal recumbency with the hindlimbs drawn cranially, or in lateral recumbency with the lumbosacral junction fully flexed. If the signs are lateralised, and the procedure is performed with the dog in lateral recumbency, then the affected side should be uppermost so that any leakage from the disc in that direction cannot be attributed to gravity. Lateral and ventrodorsal (or dorsoventral) views are taken following injection, often with the needle still in place. An ability to inject contrast into the nucleus pulposus is evidence of degeneration (see later in Fig. 26.5), leakage into the dorsal annulus suggests the presence of a disc protrusion, and contrast entering the epidural space demonstrates rupture of the annulus with or without extrusion of the nucleus pulposus. If necessary, an epidurogram can then be produced by partially withdrawing the needle and injecting additional contrast agent.

Vertebral sinography

This involves the injection of contrast agent into the internal vertebral plexus (venous sinuses) or into the vertebral body with simultaneous compression of the caudal vena cava which encourages retrograde flow of contrast into the venous plexus. Such techniques have little, if any, use in small animal patients.

Computerised tomography (CT)

Computerised tomography (CT) involves X-rays being generated from an anode which rotates around the patient at speed. The emergent X-ray beam is picked up by a circle of electronic detectors. Computer analysis of the electronic signals recorded creates an image representing a transverse section through the patient at that level. As in conventional radiographs, bone is seen as white and gas as black. The technique is particularly useful for bony detail but the image can be improved, for purposes of spinal investigations, by myelographic enhancement. Less contrast is required (approximately a quarter of the dose) than for conventional myelography.

Magnetic resonance imaging (MRI)

Magnetic resonance imaging (MRI) involves placing the body in a strong magnetic field which causes the positively charged protons to come into alignment. Hydrogen atoms are particularly sensitive to the magnetic field and it is on this basis that contrast between tissues emerges. Tissues with a high hydrogen content are clearly depicted whilst those with low content, such as bone and air, are not. The protons are then bombarded with pulsed radio waves which disorientates them.

Between pulses the protons realign and emit a small radio signal which is recorded and converted by computer analysis into an image. This is referred to as the T1-weighted image and is useful for looking at anatomical structure. Radio signals are also emitted by the protons when they dephase once the magnetic field is removed. Analysis of these signals creates a T2-weighted image which is most useful for evaluating the molecular nature and pathology of tissues.

Again the contrast may be enhanced chemically using an agent such as gadodiamide (Omniscan, Nycomed). This affects the magnetism of the tissues to varying degrees, thus increasing contrast, particularly in T1-weighted images.

Scintigraphy

This involves the use of a radioisotope (usually ^{99m}technetium methylene diphosphonate which is a gamma radiation emitter). The agent is injected intravenously and activity in the patient is monitored using a gamma camera. A few minutes after injection the soft tissue phase can be recorded and after a few hours the bone phase can be evaluated, which is of most use in the limited clinical application of the technique. The half-life of this isotope is about 6 hours and dogs are kept in isolation for 48 hours after injection. Certain precautions are necessary regarding handling of the material and also with respect to protective clothing, the biggest danger being the dog's urine because it is by way of the kidneys that the isotope is excreted.

The uptake of isotope is dependent on the rate of bone turnover and so active lesions will show increased uptake of the radiopharmaceutical ('hot spots'). Although there are limited applications for the use of scintigraphy in spinal cases it may be of use in certain circumstances (Lamb, 1987; Hillyer *et al.*, 1996) and it may show pathology before radiographic changes become evident in conditions such as discospondylitis and bone neoplasia (especially metastatic disease). An increased uptake of isotope at a site gives no indication as to diagnosis and can only be used to show the presence of a (non-specific) bone lesion. The definitive diagnosis would come from the history, clinical signs and other diagnostic tests.

Cerebrospinal fluid analysis

Cerebrospinal fluid (CSF) analysis can provide valuable information regarding the nature of a central nervous system (CNS) problem and should be considered a routine part of many such investigations.

Collection of CSF (Wright, 1984a)

Cerebrospinal fluid may be obtained by placement of needles into the cerebellomedullary cistern (cisterna magna) or by lumbar puncture, as described earlier under 'Myelography'. In most cases the former is the easiest but, because CSF flows in a cranial to caudal direction, abnormalities are more likely to be found if it is taken from a point caudal to a localised lesion. In many cases, however, the pathology is generalised or multifocal and any abnormalities are present throughout the CSF.

Sample handling

Cerebrospinal fluid should be collected into a plain tube (biochemistry) and EDTA (cytology). Cytology on fresh samples of CSF is best performed within about 30 minutes as the cells deteriorate rapidly, though useful information can still be gained thereafter. If this is not possible then the sample may be preserved with an equal volume of 4% formalin solution.

Analysis

Routine analysis of CSF generally involves gross examination, white cell counts (total and differential) and total protein values. In some cases microbiological examination is required and involves preparing samples for microscopy and also culture. Immunological tests and protein electrophoresis can be performed but are not generally available. The normal findings for routine CSF analysis are shown in Table 19.1. Variation from these normal values is indicative of CNS or nerve root disease. Common abnormalities seen and their associated pathologies are listed in Table 19.2.

It must be remembered that abnormal CSF indi-

Table 19.1 Normal cerebrospinal fluid (CSF) parameters.

Parameter	Result
Colour/turbidity	Clear/non-turbid
White cell count	<5 cells/μl
Differential white cell count	Predominantly mononuclear
Total protein	
cisternal	10–30 mg/dl
lumbar	10–45 mg/dl

Table 19.2 Common cerebrospinal fluid (CSF) abnormalities and their interpretation.

Abnormality	Interpretation
High numbers of red blood cells	Iatrogenic contamination at collection
Xanthochromia or red blood cells in macrophages	Haemorrhage in the central nervous system
Mononuclear pleocytosis*	Uncommon. Chronic inflammation – CDV, GME, FIP, toxoplasmosis, neoplasia
Neutrophilic pleocytosis	Acute inflammation – infectious or aseptic meningitis, CDV (acute), GME, FIP
Mixed pleocytosis	Non-specific, seen with neoplasia, especially meningiomas
Neoplastic cells	Not common. Mostly in lymphosarcoma and choroid plexus tumours
Increased protein without pleocytosis	Usually neoplasia or extradural cord compression

CDV = canine distemper virus; GME = granulomatous meningoencephalomyelitis; FIP = feline infectious peritonitis.

* Pleocytosis = a greater than normal number of cells.

cates CNS or nerve root disease but normal CSF does not necessarily indicate the absence of such pathology.

Electrophysiology (Duncan, 1995)

Electromyography (EMG) may be of use in evaluating spontaneous activity in resting muscle, which should not be present in a normal, anaesthetised patient. Assessment of this may help determine whether a lesion relates to either upper or lower motor neurons (UMN or LMN). In the presence of LMN injury, spontaneous activity will take about 5–7 days to appear. If specific nerves or nerve roots are damaged then these changes will only be found in specific muscles or muscle groups whereas in the case of peripheral polyneuropathies or myopathies the abnormal activity will be widespread.

Nerve conduction studies are used to investigate peripheral neuropathies and have no real value in investigating spinal disease. Although the spinal cord evoked response has the potential to give information about the functional state of the spinal cord after trauma, it is rarely used in clinical practice.

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Further reading

Papers relating to further investigation of more specific diagnoses (e.g. thoracolumbar disc disease) will be listed at the end of the chapter concerning that specific subject.

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Chapter 20
Peripheral Nerve Injury

Peripheral nerve injury is an uncommon but well-recognised diagnosis, particularly in the forelimb where it may result from traction on the brachial plexus. The forelimb may be more prone to such injury because of the relatively weak omothoracic junction compared to the sacroiliac junction of the hindlimb. In the hindlimb, injury to the nerve roots of the cauda equina resulting from caudal lumbar fractures may cause peripheral nerve deficits as discussed in Chapter 21. The healing of peripheral nerve injuries is discussed in Chapter 3 on p. 29.

Forelimb

The brachial plexus is derived from the last three cervical and the first two thoracic nerves. The nerves of the forelimb and the muscles they innervate are illustrated in Fig. 20.1.

Worthman (1957) carried out experimental neurectomies in the dog and demonstrated that, with the exception of the radial nerve, nerves of the forelimb could be sectioned without producing an alteration in the dog's gait, although the dependent muscle groups atrophied. Section of the radial nerve resulted in paralysis of the extensors of the elbow, carpus and digits. The limb was held in flexion and could bear no weight. There was desensitisation of the dorsal and lateral aspects of the antebrachium and dorsum of the paw.

Worthman (1957) considered that fractures of the first rib or humerus were the commonest causes of radial nerve paralysis. However, most dogs presenting with radial nerve paralysis have usually been involved in a road traffic accident which has resulted in traction on the limb causing an avulsion of the brachial plexus or, more accurately, an avulsion of the nerve roots since the damage is nearly always at the point of origin of the nerve roots from the spinal cord. Such an avulsion injury may involve the cranial, caudal or entire plexus:

- *Cranial plexus injury*. Avulsion of the C6 and C7 nerve roots may result from caudally directed traction on the limb. The nerves affected control shoulder movement and elbow flexion. Thus, such a case would be able to weight-bear on the limb but, perhaps most notably, would be unable to 'shake a paw'.
- *Caudal plexus injury*. Avulsion of the C7–T2 nerve roots may result from cranially directed traction on the limb. Involvement of the radial nerve will result in an inability to weight-bear on the limb (the animal will be unable to hold the elbow and/or carpus in extension). If sufficient of the more cranial nerve roots are unaffected then the limb will be carried with the elbow flexed. In addition, Horner's syndrome or unilateral loss of the panniculus reflex may be noted.
- *Complete plexus injury*. Avulsion of all the nerve roots may result from extreme traction in either direction or else forced abduction. Complete paralysis of the limb may be noted, with or without other deficits such as Horner's syndrome or unilateral loss of the panniculus reflex (Wheeler *et al.*, 1986), with the limb literally being dragged.

Treatment and prognosis

Most cases of avulsion of the brachial plexus have a very poor prognosis with respect to useful

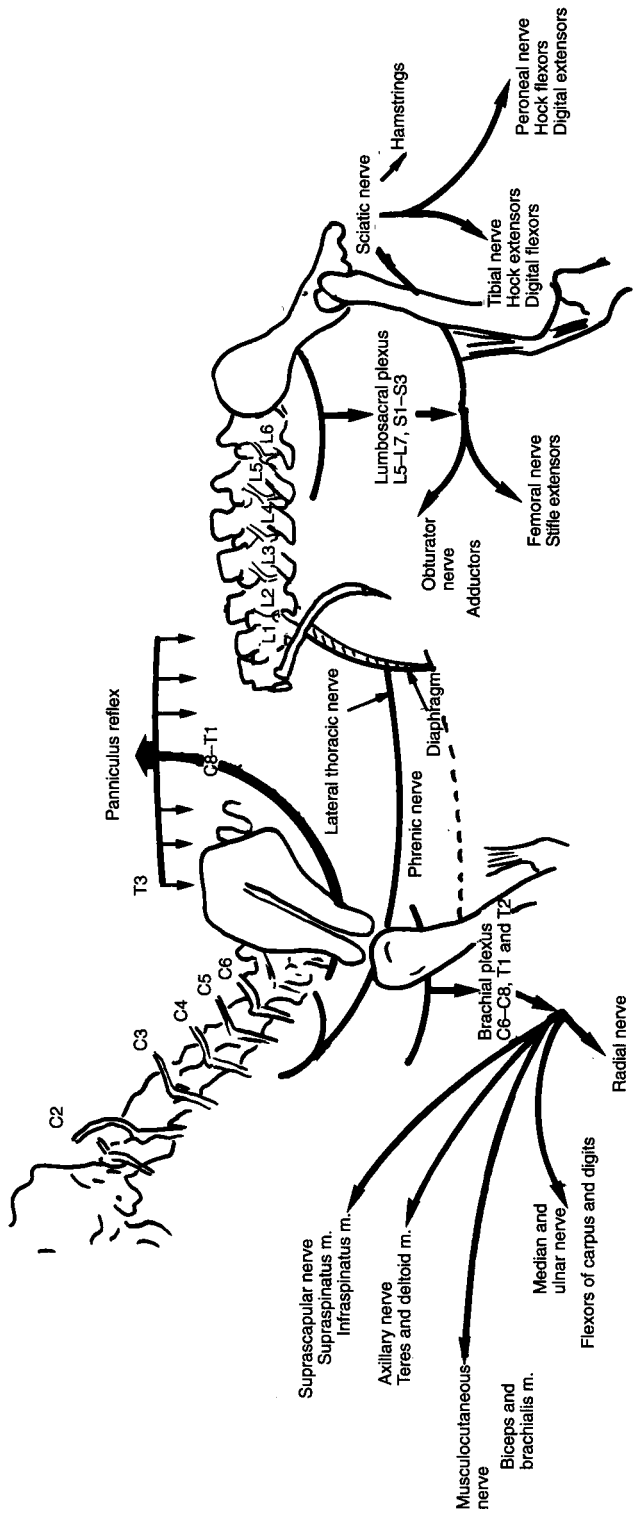

Fig. 20.1 Diagram illustrating the nerve roots supplying the forelimb and the hindlimb and the muscle groups they innervate.

recovery of limb function (Griffiths *et al.*, 1974). The exception to this is where the injury is limited to the cranial plexus (see above). If any nerve root avulsions are only partial and if some of the dysfunction is a result of oedema, then improvement may be seen over the following 1–3 months. After that time it is reasonable to assume that, in practical terms, limb function will not improve further.

Muscle relocation techniques (Bennett & Vaughan, 1976)

Limb function may be improved or restored in dogs with certain peripheral nerve injuries by means of muscle relocation techniques. Successful application of such methods is dependent on the availability of a muscle from a different group to the paralysed muscles, which must have an intact nerve supply and must be in a position to allow physical transposition. It is often difficult to fulfil these criteria as more than one nerve is usually injured in cases with brachial plexus avulsion.

After isolated radial nerve paralysis, elbow function may be restored by cutting the tendon of the insertion of the biceps brachii muscle and relocating this to the caudomedial aspect of the olecranon where the tendon is sutured to the periosteum. Ability to extend the carpus and digits is restored by side-to-side anastomosis between the tendon of the flexor carpi radialis or flexor carpi ulnaris muscle and the common digital extensor tendon. When treatment has been successful an improvement in limb function should occur within 6 weeks, reaching its best by 3 months.

The authors' experience with these techniques in the forelimb have been generally disappointing, despite careful evaluation of muscle group function. If such surgery is to be considered it may be prudent to use electromyography (EMG) and nerve conduction velocity studies to evaluate the patient so as to try and ensure that the muscle groups to be relocated are not also showing signs of denervation.

Carpal arthrodesis

In dogs showing radial nerve palsy that knuckle the carpus but are able to extend the elbow and weight-bear through this joint, carpal arthrodesis may help to improve limb function (see Chapter 35 for details of surgery). However, in the authors' experience, dogs treated in this way may continue to have problems due to knuckling of the digits leading to excoriation of the skin, abnormal wear on the digital pads causing ulceration, or paraesthesia causing chewing of the digits or skin over the plate. In the cat, arthrodesis of the carpus in a hyperextended position (20–30°) may be an effective way of managing distal radial nerve paralysis (p. 419).

Amputation

Amputation may be considered the most practicable option for the management of these injuries where limb function remains unsatisfactory after 2–3 months.

Hindlimb

The lumbosacral plexus is derived from the last three lumbar and the three sacral nerves. The nerves of the hindlimb and the muscles they innervate are illustrated in Fig. 20.1. The lumbosacral plexus is not predisposed to traction in the same way as the brachial plexus (see above) but may be subjected to such trauma in association with sacrococcygeal fractures/luxations. Such injuries will not be considered further here as they are discussed in Chapter 21. The clinical signs associated with transection of specific nerves have been described by Worthman (1957).

Obturator nerve

This innervates the external obturator, pectineus, adductor and gracilis muscles. Obturator paralysis, therefore, results in an inability to adduct the limb and the animal will tend to 'splay' its hindlimb(s). Injury to this nerve is rarely seen in small animals and is more common in cows following either traumatic calvings, when pressure may have been exerted on the nerve as it passes over the pubis on its way to the obturator foramen, or the animal 'doing the splits' on wet concrete. Treatment involves 'hobbling' the animal and most injuries recover with time

leading to a successful outcome as long as no complications arise.

Femoral nerve

This innervates the quadriceps muscle group and paralysis results in an inability to extend the stifle or weight-bear through that joint which, in turn, leads to collapse of the hock. Sensation is lost on the medial aspect of the limb and the patellar reflex will be absent or reduced. Injury to this nerve is very uncommon, presumably because it is well protected by musculature. If it was severed by laceration or iatrogenically then repair should be attempted (see 'Sciatic nerve' below). Failing this then, theoretically, limb function might be improved by stifle arthrodesis.

Sciatic nerve

This nerve innervates the hamstring muscle group and then divides into the peroneal and tibial nerves which supply all the muscles distal to the stifle. The peroneal nerve innervates the flexors of the hock and the extensors of the digits and injury to this results in hyperextension of the hock and knuckling of the digits. The tibial branch innervates the extensors of the hock and flexors of the digits and injury will result in hyperflexion of the hock ('dropped hock') and hyperextension of the digits. Complete transection of the sciatic nerve would result in loss of the withdrawal reflex and complete paralysis below the stifle apart from some sensation in the medial digit which is supplied by a branch of the femoral nerve.

Injury to this nerve may result from vertebral column trauma or injuries such as bites or lacerations, but is more often associated with pelvic fractures or iatrogenic trauma resulting from procedures such as: internal fixation of pelvic or femoral fractures, especially iliac shaft or acetabular fractures and intramedullary pinning of femoral diaphyseal fractures (Fanton *et al.*, 1983; Chambers & Hardie, 1986); internal stabilisation of hip luxations; excision arthoplasty; triple pelvic osteotomy; perineal hernia repair; or intramuscular injection into the hamstrings. The peroneal component of the lumbosacral trunk is more prone to injury than its tibial counterpart due to its fibres being of greater diameter. Therefore, peroneal dysfunction is relatively common and most easily detected as causing a reduction in flexion of the hock during testing of the withdrawal reflex. Selective involvement of the tibial branch of the sciatic nerve is rarely a result of injury and the most common cause of tibial nerve palsy is as part of a feline diabetic neuropathy.

The vast majority of cases with sciatic palsy following external trauma will recover spontaneously. As in the forelimb, injuries that do not involve complete neurotmesis may improve over 1–3 months and surgical treatment may be delayed for that period unless it is known that the nerve has been transected (i.e. iatrogenic where severance of the nerve was recognised). Treatment of sciatic nerve injury may involve primary repair or muscle relocations/arthrodesis.

Repair of the sciatic nerve

The sciatic nerve is most easily located deep to the biceps femoris muscle. Further proximal it may be exposed by a caudolateral approach to the hip but in the region where the lumbosacral trunk lies medial to the iliac shaft/acetabulum exposure becomes very difficult, requiring much soft tissue dissection.

The site of injury will usually be incorporated within a fibrous mass or bony callus and careful dissection to release the two ends is required. The aim is to release sufficient length of nerve so that the ends can be apposed without tension and the epineurium can be sutured with fine monifilament material (Shores, 1994). A silicone nerve cuff can be used to protect the repair but must be a loose fit with a cross-sectional area of two to three times that of the nerve (i.e. for a nerve of 6 mm diameter a cuff of 8–10 mm diameter would suffice). The cuff is secured to the epineurium with fine sutures of monofilament material.

Salvage procedures – muscle relocation +/– arthrodesis

In cases with peroneal paralysis, muscle relocation may be used to improve limb function (Bennett & Vaughan, 1976). A side-to-side anastomosis is performed between the tendon of the functional long digital flexor and the tendon of the non-functional long digital extensor muscle.

In cases with complete sciatic paralysis it may be possible to improve limb function by combining tarsal arthrodesis with relocation of the long digital extensor tendon of origin, which is moved from its attachment to the lateral femoral condyle and sutured to the lateral aspect of the patellar retinaculum. After this the stifle is able to support weight by virtue of the functional quadriceps, the hock will be stable by virtue of the arthrodesis and the digits will extend under the influence of the tension created in the long digital extensor tendon when the quadriceps complex becomes activated.

Patient selection for, and the complications of, such salvage procedures in the hindlimb to treat sciatic nerve paralysis are the same as those discussed above for treatment of radial paralysis in the forelimb. However, it is the authors' experience that muscle relocation techniques alone are more successful in treating peroneal paralysis than for treating radial paralysis, presumably because the muscle identified as functional is more likely to be so.

Amputation

This may be used for treatment of those with lumbosacral plexus injuries but it should not be considered as appropriate as in the brachial plexus avulsion because in the hindlimb the peripheral nerve injury is usually confined to one nerve, or a branch of one nerve, compared to the forelimb where the injury most commonly involves multiple nerves.

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A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny,Steven J. Butterworth

Chapter 21
Spinal Fractures and Luxations

Acute trauma to the spinal cord is relatively common and is usually the result of road traffic accidents. The various types of injury encountered include:

- Vertebral fracture
- Vertebral subluxation or luxation
- Vertebral fracture–luxation
- Intervertebral disc extrusion
- Spinal haemorrhage/haematoma
- Spinal cord concussion

Fractures and luxations of the spine can involve any level of the vertebral column. However, the caudal thoracic region and the lumbosacral junction are involved most frequently. Commonly encountered types of spinal fracture and luxation are illustrated in Fig. 21.1. Animals with suspect spinal injury must be handled with care to avoid further injury and, if necessary, the spine should be supported with a board or temporary splint. A thorough clinical, neurological and radiographic examination is essential to decide on appropriate treatment options and prognosis. The neurological examination should enable the level of vertebral column injury to be identified and the severity of spinal cord damage graded (see Chapter 17). The clinical grading of spinal cord injury is based on the system described by Griffiths (1982). However, some of the original grades have been subdivided and the system used in this text, as stated in Chapter 17 and repeated here for convenience, is based on seven grades, namely:

- **Grade 1**: pain only
- **Grade 2**: ambulatory paraparesis/quadriparesis
- **Grade 3**: non-ambulatory paraparesis/quadriparesis
- **Grade 4**: paraplegia/quadriplegia (i.e. voluntary movement absent)
- **Grade 5**: paraplegia/quadriplegia + urinary retention with overflow (URO)
- **Grade 6**: paraplegia/quadriplegia + URO + loss of conscious pain sensation (CPS)
- **Grade 7**: ascending/descending myelomalacia

Radiographic examination

Initial evaluation of injury to the spinal column is based on lateral radiographs of the spine. If additional information is required then ventrodorsal (+/– oblique) views are also taken. Extreme care must be taken to support the spine while the animal is positioned for such views to minimise the risk of further damage to the spinal cord. It should be noted that the radiographic findings will not necessarily represent the position of the vertebrae at the time of injury. Even when there is apparently little displacement of the vertebrae, as in a subluxation, severe cord trauma has often resulted because such injuries tend to occur in a 'whiplash' fashion, the vertebra being suddenly displaced at the time of the accident and then sliding back to near normal alignment, in part due to muscle spasm. It is safer, therefore, to evaluate the severity of injury on the basis of neurological examination rather than the radiographic findings. Myelography may be necessary to detect spinal cord compression due to disc extrusion or spinal haemorrhage.

Management of spinal trauma – general comments

In making decisions for a specific case the option chosen will be influenced by a number of factors, including:

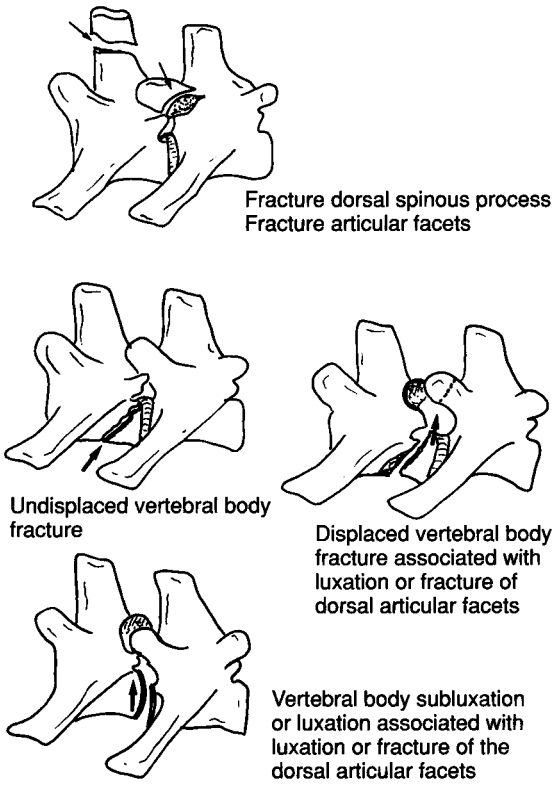

Fig. 21.1 Types of spinal fracture and luxation.

- Severity of cord injury
- Site of injury within the vertebral column
- Whether the injury is judged to be relatively stable or unstable
- Size (and age?) of the patient
- How amenable the fracture configuration is to internal fixation
- Surgical facilities and experience available
- Owner's ability to cope with the aftercare (see later)
- Finance

In cases of acute spinal cord trauma damaged cells release large amounts of oxygen free radicals which can overwhelm natural scavenging systems and cause further damage to cell membrane phospholipids resulting in lipid peroxidation. This accounts for secondary spinal cord injury. Methylprednisolone sodium succinate (Solumedrone V, Pharmacia & Upjohn) has a neuroprotective action *in vitro* which minimises secondary spinal cord injury. Since it is the only available drug with some evidence suggesting its usefulness it is the medication of choice in animals with spinal trauma, if any such drugs are to be used. Methylprednisolone should be given by slow intravenous injection within 8 hours of injury. The initial dose is 30 mg/kg, followed by 15 mg/kg every 4 hours. Treatment should not exceed 24 hours.

The options to be considered when managing cases with trauma to the vertebral column include:

- Cage rest
- External splinting or casting
- Internal fixation by:
  - plating of the vertebral bodies
  - cross-pinning of the vertebral bodies
  - application of pins or screws and polymethylmethacrylate (PMMA or bone cement) to the vertebral bodies
  - plating of the dorsal spinous processes
  - spinal stapling or modified segmental fixation using pins and wire
  - bone screw fixation of the articular facets
- Decompression of the spinal cord

When selecting methods of spinal fixation it is important to consider the forces acting at the site of injury. It is convenient to divide the spinal column into two compartments:

- The *dorsal compartment* consists of all structures dorsal to the floor of the neural canal, i.e. the articular facets, vertebral laminae and pedicles, dorsal spinous and interspinous ligaments.
- The *ventral compartment* consists of the structures ventral to the floor of the neural canal, i.e. the vertebral body, intervertebral disc, dorsal and ventral longitudinal ligaments.

Injuries to both compartments occur most commonly. The most serious injury is a vertebral body fracture in combination with injury to the dorsal compartment structures which results in angulation and lateral displacement, together with collapse of the vertebral body. Internal fixation is required in this situation. Injury to dorsal compartment structures alone is less serious because a ventral buttress remains and these cases can often be managed by external splintage. Isolated injuries to dorsal spinous or transverse processes

usually do not result in any instability of the neural canal, rarely pose a threat to the spinal cord and, in the main, can be managed successfully with conservative measures only.

Summary of management options and prognosis for spinal trauma cases

Indications for surgical management of spinal trauma and the results of treatment have been reviewed by McKee (1990a,b) and can be summarised as follows, in relation to the grades described above:

- Animals with grade 1 or 2 dysfunction are usually treated conservatively initially, with strict confinement +/– methylprednisolone +/– external splintage. Surgery is indicated in these grades if there is a deterioration in neurological status as a result of vertebral instability. The prognosis in these cases is generally good.
- Cases with grade 3, 4 or 5 dysfunction are generally classed as surgical emergencies. Prompt reduction and stabilisation of the spinal column and decompression of the spinal cord should be undertaken as soon as is practicable. Animals with grade 3 injuries usually carry a good prognosis for recovery while prognosis for grades 4 and 5 is guarded.
- Animal with grade 6 injuries (i.e. loss of conscious pain sensation [CPS]) generally carry a hopeless prognosis, with an estimated 5% regaining an ability to walk within 1 month of surgery.

Cervical fractures and luxations

The majority of cervical fractures involve the atlas or axis. The axis is the most frequently involved with the fracture occurring through the dens and/or vertebral body, often resulting in traumatic atlantoaxial subluxation. The reason for this is that the axis has firm attachments to the atlas and skull, via ligaments arising from the dens, and also to the remainder of the cervical vertebrae, via the nuchal ligament. Therefore, if the skull is flexed forcibly with respect to the vertebral column the axis is most likely to be the point of failure. Such injuries usually result from running head-on into a stationary object, or else involvement in a road traffic accident.

Although there may be considerable displacement of fractures of the cranial cervical vertebrae, there is plenty of room for the spinal cord within the neural canal and the resultant neurological deficits are often remarkably mild. The main presenting sign is cervical pain (De Lahunta, 1977). Death can occur from respiratory arrest when haemorrhage and oedema resulting from the fracture involve the brain stem (Gage, 1968). Fractures of the caudal cervical vertebrae may cause quadriparesis or quadriplegia, and death may occur from respiratory failure if there is involvement of the phrenic outflow (De Lahunta, 1977).

Suspicion of cervical fracture can usually be confirmed from a lateral radiograph of the neck but in some cases ventrodorsal views may be necessary. Animals with suspected cervical fracture must be handled with extreme care to prevent further fracture displacement and the neck should be supported at all times, particularly if the animal is anaesthetised for radiography.

Conservative management

Cases with cervical fractures are often managed successfully by cage confinement for 4–6 weeks or have their neck immobilised in a reinforced splint or cast (Fig. 21.2) for the same amount of time. The cast is applied with the dog conscious in a normal standing position with the neck extended. The cast extends rostrally to include the ears and extends just caudal to the forelegs so that the entire neck is immobilised. The dog may require hand feeding initially but later should manage to take food from a shallow, raised bowl with good results. In this region of the vertebral column the neural canal is large relative to its content and so vertebral displacement can be tolerated without necessarily causing permanent neurological dysfunction.

Internal fixation

Displaced cervical fractures, particularly those associated with motor dysfunction, tend to be managed surgically. Fractures of C2 usually involve the dens and may result in atlantoaxial subluxation. The subluxation may be stabilised by

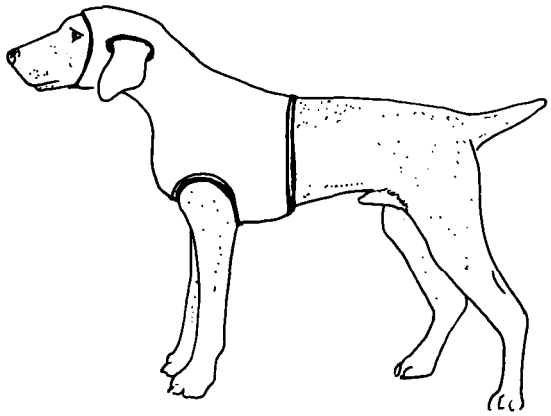

Fig. 21.2 Treatment of cervical fractures – neck cast.

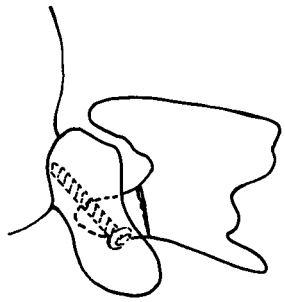

Fig. 21.3 Fracture of the axis. Lag screw fixation of the ventral articular facets.

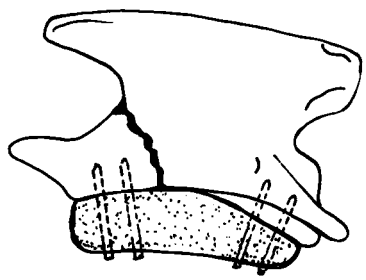

Fig. 21.4 Fracture of the axis. Kirschner wires plus bone cement used for fixation.

lag screw fixation of the ventral articular facets (Fig. 21.3). In fractures which involve any of the cervical vertebral bodies, a ventral approach is used and four pins are driven into the vertebral body, two cranial and two caudal to the fracture; the pins are left protruding about 1 cm below the ventral aspect of the vertebral body. The fracture is reduced and the pins are incorporated in bone cement (Fig. 21.4) which acts as an internal splint (Rouse, 1979). An alternative to pins is to place screws in the vertebral bodies to anchor the bone cement.

Thoracolumbar fractures and luxations

Neurological dysfunction is commonly seen with injuries in this region of the vertebral column because the spinal cord almost fills the neural canal. The injuries are rarely stable and so it is the exceptional case that warrants nothing more than cage rest.

External splinting

One of the simplest and most effective ways of managing thoracolumbar fractures and luxations is the use of an external splint or back brace (Patterson & Smith, 1992). It can be used as the sole method of providing stability or it can be used in combination with internal fixation. Aluminium sheeting (0.54 mm thickness) is used most often for the splint but in dogs under 10 kg bodyweight a thermoplastic material (Orthoboard, Millpledge) can be used. The splint is conformed to a gutter shape which will extend from the withers to the tail base with the spine in extension (Fig. 21.5a). Ideally the splint is applied with the dog conscious or lightly sedated so that protective muscle tone is retained. At presentation most dogs with fractures have a kyphotic deformity of the spine. While the dog is lying on its side the splint is carefully slid under the dog so that the dorsal curvature of the splint is lined up with the spinous processes. Two people are needed to hold the dog and apply the splint. The splint is held against the spine while the dog is carefully rotated into dorsal recumbency. Once the animal is lying on its back it will tend to relax allowing the spine to extend into the cradle of the back splint. Axial alignment of the fragments is often dramatically improved and relief from pain may

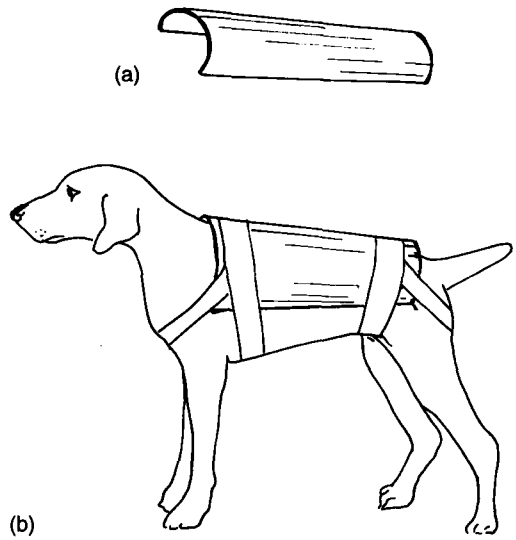

Fig. 21.5 (a) Back splint made from aluminium sheet. (b) Application of the back splint.

be immediately apparent. With the dog still lying on its back in the splint, the caudal end of the splint is slid over the edge of the table to allow Elastoplast (Smith & Nephew) straps to be applied in a cruciate fashion to secure the splint to the hindquarters. The same procedure is used to secure the cranial end of the splint with Elastoplast (Smith & Nephew) straps around the forelegs (Fig. 21.5b).

The splint provides an effective way of resisting bending and compressive forces at the fracture site, and has mechanical advantages over internal fixation which can only stabilise a relatively small area of the spine. The ideal candidate for spinal bracing is one which has:

- Conscious pain sensation present in the hindlimbs
- An injury that is confined to the dorsal elements of the vertebra, with an intact vertebral body and disc providing a ventral buttress

In a series of 16 cases treated by external splinting reported by Patterson & Smith (1992) 10 made good recoveries. The main complications associated with back splinting are sores resulting from the bandages chaffing the inner sides of the legs, and urine scalding.

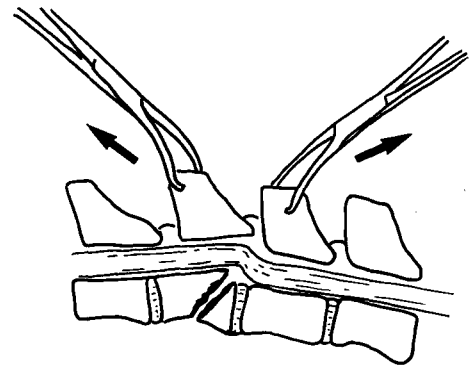

Fig. 21.6 Reduction of a spinal fracture by direct traction on the spinous processes.

Internal fixation

Surgical treatment is recommended in displaced or unstable fracture luxations of the thoracolumbar spine, particularly when both the dorsal and ventral compartments of the vertebral column are involved.

A dorsal midline skin incision is made over the fracture and the lumbodorsal fascia is incised. A periosteal elevator is used to separate the lumbar musculature from the spinous proceses and the muscle attachments are severed from the articular and accessory processes of the vertebrae involved. Blunt dissection and lateral retraction of muscle is continued down to the transverse processes of the vertebrae or the rib heads. Exposure may be uni- or bilateral depending on the method of fixation intended.

An alternative is the lateral approach (see under the heading 'Fenestration' in Chapter 25 and Fig. 25.3) which can be used for plate fixation or transfixion pinning of the vertebral bodies.

Direct traction with bone forceps applied to the adjacent dorsal spinous processes is used to help reduce the displaced vertebrae (Fig. 21.6).

Vertebral body plating

The application of a small bone plate to adjacent vertebral bodies provides excellent fixation for fractures involving the vertebral body and also luxations (Swaim, 1971, 1972). The method is limited to the fixation of fractures/luxations in the thoracolumbar region between T11 and L3.

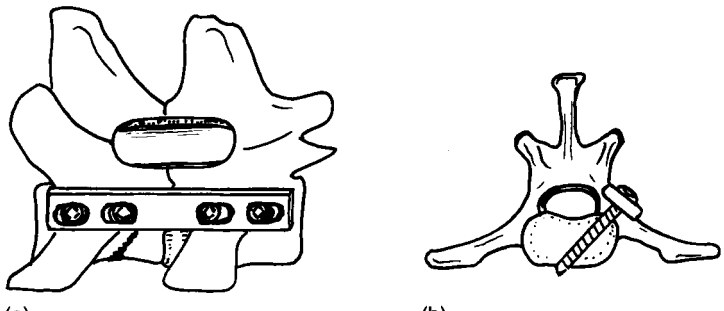

Fig. 21.7 (a) Fracture fixation by vertebral body plating. (b) Transverse section showing vertebral body plating.

Cranial to T11 exposure and application is impracticable, and caudal to L3 there is the risk of damage to nerve roots contributing to the lumbosacral plexus. In the lumbar region the plate is positioned at the junction of the transverse processes and vertebral bodies (Fig. 21.7a). In the thoracic region, resection or disarticulation of the rib heads is necessary to give a sufficiently flat surface for application of the plate to the vertebral bodies. Screws should engage a minimum of four cortices cranial and caudal to the fracture or luxation and are directed ventral to the neural canal (Fig. 21.7b).

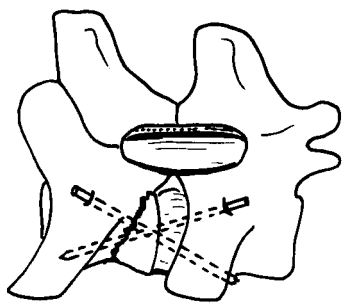

Fig. 21.8 Vertebral body fracture showing fixation with crossed Kirschner wires.

Vertebral body cross-pinning

Kirschner wires can be used for interfragmentary fixation of vertebral body fragments (Gage, 1969), particularly in small dogs (Fig. 21.8).

Pins or screws and polymethylmethacrylate (PMMA or bone cement)

Multiple vertebral body pins, which are anchored in bone cement, provide a means of stabilising fractures and luxations at any level of the spinal column (Blass & Seim, 1984; Blass *et al.*, 1988).

In the thoracic and lumbar regions, a dorsal approach is used to give bilateral exposure of the articular facets and transverse processes. At least two, and preferably three or four, pins are inserted into the vertebral bodies on either side of the fracture or luxation (Fig. 21.9a,b). The pins should penetrate two cortices, and the ends of the pins should be notched or bent to give good fixation in the bone cement. The fracture/luxation is reduced and a cylinder of bone cement is prepared. This is moulded to the articular facets and the protruding ends of the pins on one side of the spinous processes (Fig. 21.9c). A second cylinder of cement is applied to the pins on the contralateral side. A considerable amount of heat is generated

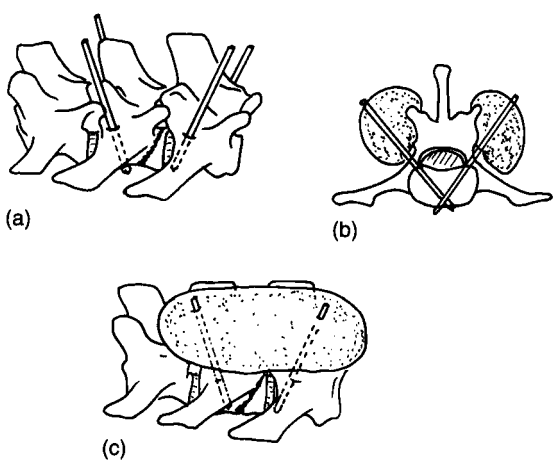

Fig. 21.9 Illustrating the use of pins or screws plus bone cement to stabilise vertebral fractures or luxations. See text for details.

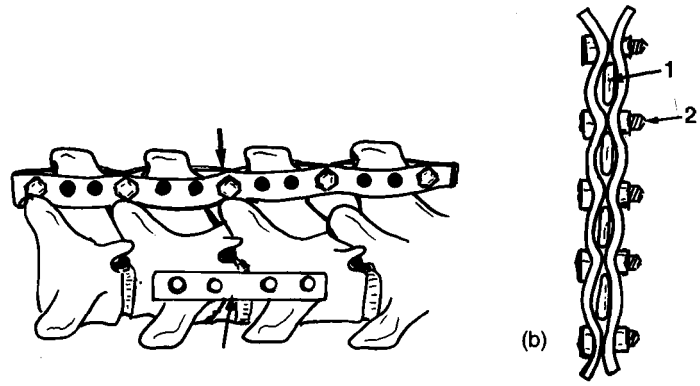

Fig. 21.10 (a) Use of plastic plates bolted (large arrow) to the dorsal spines of the vertebrae. This method of fixation can be used to reinforce vertebral body plating (small arrow). (b) Dorsal view of vertebral spines (1) showing plates bolted (2) on either side of the spines.

as the cement sets. Consequently the cord and the surrounding tissues should be irrigated with cooled lactated Ringer's solution to prevent thermal damage and necrosis. Implant failure can occur if an attempt is made to span too large an area with bone cement. It is preferable, therefore, to span only one intervertebral space.

Screws can be used instead of pins, they are often preferred since they have greater pull-out resistance and, in addition, they do not need to be notched or bent to anchor the bone cement. Pins, however, have two advantages over screws. First, pins are easier to insert and, second, pins are stronger than screws, which have a tendency to break (Garcia *et al.*, 1994). The pull-out resistance of pins may be increased by using those that are threaded. However, only those with a positive profile thread should be used as negative profile pins are at a greater risk of breaking. Pins and screws can be combined for this method of fixation and some of the implants may be placed across the articular facets in order to increase stability further.

Plating the dorsal spinous processes (Lumb & Brasmer, 1970)

Two plastic spinal plates are applied on either side of the dorsal spinous processes, sandwiching the spines between them (Fig. 21.10). Bolts are placed through the plates, passing between the spinous processes, and these are secured by washers producing friction between the plate and the spines. This technique has been used alone for fractures and luxations of the thoracolumbar spine but it offers poor resistance to bending and rotational forces. Therefore, it is better to use it in combination with other methods of internal fixation such as vertebral body plating.

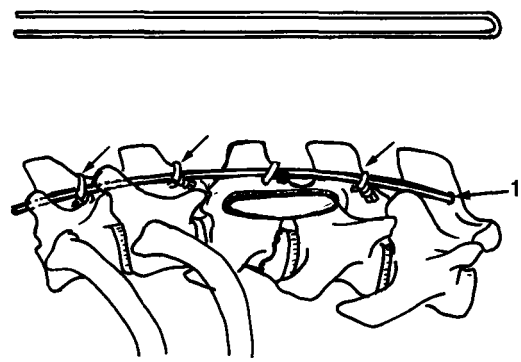

Fig. 21.11 Spinal stapling. A U-shaped pin (top and arrowed) is attached to the dorsal spines with wire sutures (1).

Spinal stapling or modified segmental fixation using pins and wire

Midlumbar and caudal lumbar fractures can be stabilised using a U-shaped pin which is wired to the articular processes, or the dorsal spinous processes in smaller dogs (Fig. 21.11). This method was described by McAnulty *et al.* (1986) and is a modification of a technique reported by Gage (1971). Like plating of the dorsal spinous processes, this technique offers little resistance to bending and rotational forces. As a result it may only be considered as a sole means of fixation in cases with an intact ventral compartment (see earlier).

Lag screw fixation of the articular facets

This method is particularly suitable for fracture luxations involving the caudal three lumbar vertebrae and the lumbosacral junction (see below) (Denny *et al.*, 1982).

Decompression of the spinal cord

It is in the thoracolumbar region that decompression of the spinal cord has to be considered most often in cases with spinal fractures. This is a direct result of the spinal cord occupying most of the neural canal in this region and thus relatively minor displacement of the vertebrae, or disc extrusion, will create cord compression. Reduction of the fracture/luxation will obviously reduce or eliminate the cord compression but in some cases it may be necessary to consider a laminectomy. In general, such an adjunct to internal fixation should be considered when there has been a loss of voluntary hindlimb movement (i.e. grade 4 and above) and be taken as mandatory when there is no evidence of CPS in the hindlimbs (i.e. grade 6), if surgery is to be considered at all in such cases (see above regarding prognosis), to ensure that the spinal cord has not been totally transected.

Hemilaminectomy is used in preference to dorsal laminectomy as it causes less instability and there is less risk of constrictive laminectomy membrane formation (Smith & Walter, 1985). Hemilaminectomy allows removal of blood clots, bone fragments and any extruded disc material from the neural canal. It also permits inspection of the spinal cord and a final decision can be made between continuing treatment or recommending euthanasia. Obviously, if it is apparent that there has been gross trauma or transection of the cord then euthanasia would have to be recommended.

Caudal lumbar/lumbosacral fractures and luxations

In the lumbosacral region, the diameter of the neural canal is large relative to its content, the cauda equina. Considerable displacement of fractures/luxations of the caudal three lumbar vertebrae can be tolerated, without necessarily causing permanent neurological dysfunction. However, the injuries tend to result in intense pain.

Conservative management

Cases with caudal lumbar fractures are often managed by cage confinement with good results for the reasons given above. A back splint (as described for thoracolumbar fractures) may be used to help stabilise the region and reduce the level of pain whilst healing takes place.

Internal fixation

In displaced fractures/luxations involving the caudal three lumbar vertebrae or lumbosacral junction, a variety of internal fixation methods are available including:

- Pins or screws plus polymethylmethacrylate (Fig. 21.9)
- Pins attached to the dorsal articular facets with wire sutures (Fig. 21.12)

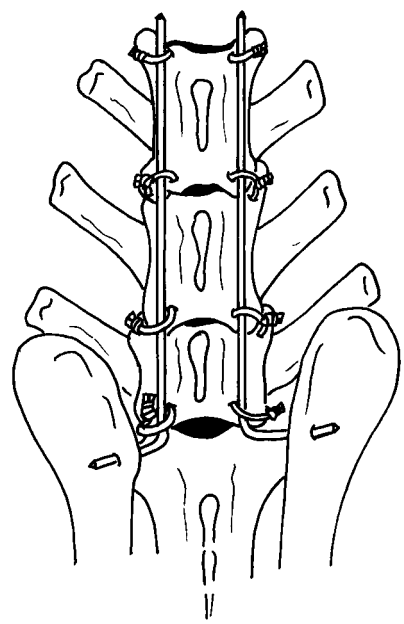

Fig. 21.12 Dorsal stabilisation of fractures and luxations in the caudal lumbar region using pins wired to the articular facets. For extra stability the ends of the pins should pass through the ilium.

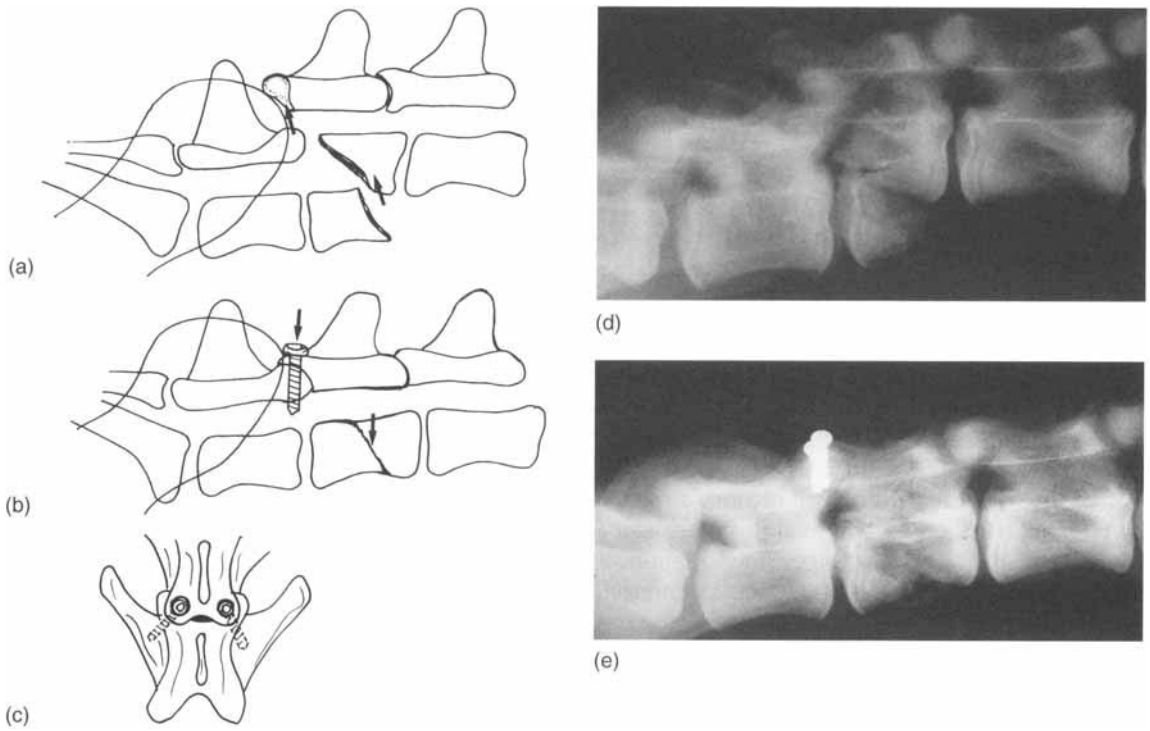

Fig. 21.13 (a) Tracing of preoperative lateral radiograph of lumbar spine showing fracture of the centrum of the 6th lumbar vertebra associated with dislocation of the dorsal articular facets. (b) Tracing of postoperative radiograph showing reduction and fixation using lag screws placed through the dorsal articular facets. (c) Dorsal view of the vertebra showing position of the lag screws. (d) Lateral radiograph showing fracture of the centrum of L6 with dislocation of the articular facets between L6 and L7 in a 6-month-old German Shepherd Dog. (e) Radiograph taken after surgical reduction and immobilisation with two screws through the articular facets. The dog recovered within 6 weeks of surgery and had no further problems during a 1-year follow-up period. (Reproduced from Denny *et al.* (1982) from the *Journal of Small Animal Practice* with permission from BVA Publications.)

- Lag screw fixation of the dorsal articular facets (Fig. 21.13a,b)
- Transilial pin (Fig. 21.14)

Lag screw fixation and the transilial pin are used most frequently.

In the treatment of oblique fractures of the vertebral body of L6 or L7 associated with luxation of the caudal articular facets of the vertebra, reduction of the articular facets tends to give simultaneous reduction of the vertebral body fracture. Fixation is achieved with lagged bone screws placed through the articular facets (Fig. 21.13). The area should be protected by external splintage for 2–3 weeks following surgery.

In fractures/luxations of L7/S1, reduction of the articular facets of L7 can be maintained by a

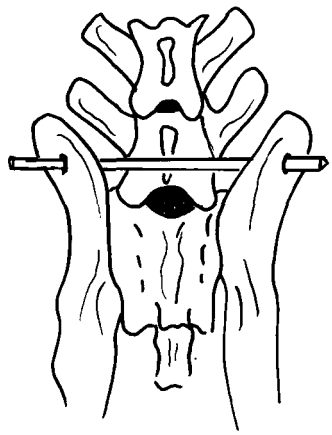

Fig. 21.14 Lumbosacral luxations and fracture of L7 can be stabilised using a transilial pin which passes over the dorsal surface of L7 holding the articular facets in position.

transilial pin (Fig. 21.14) as an alternative to lag screw fixation (Slocum & Rudy, 1975).

Prognosis

Most dogs and cats with fractures/luxations involving the cauda equina will walk again within 3–4 weeks of the accident. Severely displaced lumbosacral luxations resulting in urinary retention with overflow carry a guarded prognosis as flaccid paralysis of the bladder tends to persist even after reduction and internal fixation and despite many such cases regaining satisfactory hindlimb function.

Fractures/luxations of the sacrococcygeal area

Sacrococcygeal fractures or luxations are common in cats. Hindlimb dysfunction occurs if there has been a traction injury to the more proximal nerve roots of the cauda equina. Limb function usually improves within a week of injury. The prognosis varies and the following guide to recovery is given by Sharp (1995) based on a review of over 50 cats with sacrococcygeal injuries (Smeak & Olmstead, 1985).

- All cats with coccygeal deficits only (i.e. a paralysed tail) should recover.
- Seventy-five per cent of cats which have urinary retention in addition to the coccygeal deficit will recover.
- Cats with both these deficits and reduced anal tone or perineal sensation have only a 60% recovery rate.
- Those in which the situation is further complicated by lack of urethral sphincter tone (bladder readily expressed by pressure) have the worst prognosis and only about 50% will recover. The series demonstrated that if the cats did not regain the ability to urinate normally within 1 month of injury then incontinence was likely to be permanent.

Tail amputation can always be carried out later if faecal and urinary soiling are a problem. However, there is an argument for early amputation in order to reduce the risk of further injury to the cauda equina resulting from the paralysed tail becoming trapped and creating further traction on the nerve roots.

Postoperative care of animals with spinal injuries

Animals recovering from spinal injury require a high standard of nursing care. Pain relief using appropriate analgesics is of paramount importance. Paraplegics and quadriplegics require particular attention.

Patients should be kept clean and dry on well-padded bedding. Frequent turning helps prevent decubital ulceration. If the animal is incontinent, the bladder should be emptied (preferably by manual expression but utilising catheterisation if necessary) at least three or four times a day. Prophylactic antibiotic therapy may reduce the risk of bladder infection. Both active (whirlpool bath) and passive physiotherapy (flexion and extension exercises plus massage) are important to minimise muscle wasting. Owners should be made aware that the recovery period can often be prolonged (several months is not uncommon) and also given an idea of the amount of nursing that may be required during this period prior to making a decision in relation to treatment.

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Chapter 22
Atlantoaxial Subluxation

Atlantoaxial subluxation is an uncommon cause of neck pain and neurological deficits relating to compression of the cervical spinal cord and is seen mainly in small breeds of dog. The instability may be traumatic or congenital in origin. Reaching a definitive diagnosis requires minimal investigation and cases may be managed successfully with appropriate treatment.

Aetiology and pathogenesis

The normal atlantoaxial joint allows rotation of the skull about the long axis but should contribute little, if anything, to movement in the sagittal plane. Stability in this direction is achieved by a series of ligaments securing the dens of the axis to the occiput and atlas (Fig. 22.1). The dens is attached to the occipital bone by three ligaments: the *apical ligament* to the ventral aspect of the foramen magnum, and two *alar ligaments* to points just medial to the caudal part of the occipital condyles. It is attached to the atlas by two small *lateral ligaments* and also held in position by the *transverse ligament* passing over its dorsal aspect. The dens is phylogenetically a part of the atlas which develops as a separate centre of ossification and normally fuses onto the body of the axis by 7–9 months of age.

Acquired atlantoaxial subluxation may result from trauma in any breed resulting in fracture of the dens or body of the axis or rupture of the transverse ligament. *Congenital atlantoaxial subluxation* is most often a result of malformation of the dens or failure of it to unite with the axis (Fig. 22.2). Other associated abnormalities include a shortened atlas, deformity of the occipitoatlantal joint and hydrocephalus. The effect of these abnormalities is to create a joint with poor inherent stability and signs develop as the ligaments fail to maintain stability in the face of normal forces. As a result the signs will develop as the ligamentous support gradually fails or when minor trauma leads to rupture of already compromised ligaments which would have resisted such forces in an anatomically normal joint.

History and clinical signs

Cases can be divided into two distinct groups depending on the aetiology of the subluxation. Trauma-related cases will generally have a history of major injury such as involvement in a road traffic accident or running at speed into a solid object. It is, however, worth bearing in mind that cases with congenital deformities that are insufficient to cause a clinical problem in their own right might predispose to 'trauma-related' subluxation following a fairly minor accident. In truly traumatic cases marked pain will be noted and the degree of neurological involvement may vary from mild paresis through to quadriplegia. In those cases where the transverse ligament has ruptured, and the dens is relatively normal, the signs may be very severe due to the degree of spinal cord compression created by the subluxation.

Amongst those cases presenting with congenital subluxation the toy breeds are overrepresented, for example the Yorkshire Terrier, Pomeranian and Chihuahua. More than 50% of cases will present before they reach 1 year of age and the commonest sign is that of neck pain, although this may be associated with neurological deficits including paresis in one or more limbs or intermittent collapse.

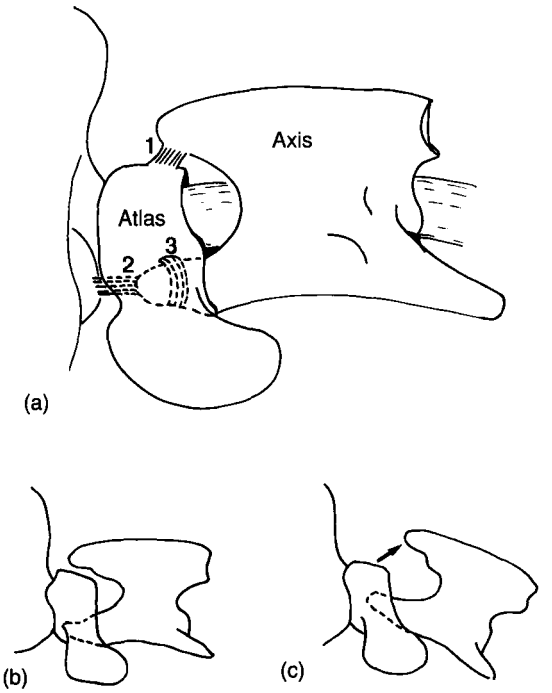

Fig. 22.1 Atlantoaxial subluxation. (a) Normal anatomy of the atlas and axis showing 1 – dorsal atlantoaxial ligament, 2 – alar and apical ligament from dens to occiput and 3 – transverse ligament passing from dorsal to dens. (b) Normal alignment of the atlas and axis. (c) Atlantoaxial subluxation.

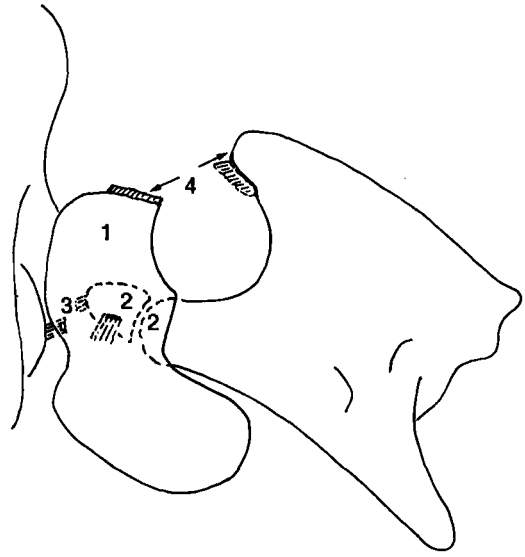

Fig. 22.2 Abnormalities seen in congenital atlantoaxial subluxation. 1 – Shortened atlas, 2 – deformed or ununited dens, 3 – absent or ineffective occipitoatlantoaxial ligaments, 4 – increased interarcuate space.

Differential diagnosis

Causes of the signs described above include:

- Atlantoaxial subluxation
- Inflammatory central nervous system (CNS) diseases (e.g. granulomatous meningoencephalomyelitis)
- Vertebral trauma at other sites in the cervical spine
- Intervertebral disc prolapse
- Discospondylitis
- Neoplasia

Diagnosis

The history and clinical signs should provide a high index of suspicion and confirmation is obtained by means of radiography. In order to obtain diagnostic films general anaesthesia is mandatory but it is *important to remember that catastrophic iatrogenic damage may occur if these patients are handled carelessly*, particularly once anaesthetised when all protective reflexes are lost. In general, it is to be recommended that they are placed in dorsal recumbency, within a foam trough, with their head and neck in extension. Whenever they are moved from this position great care must be taken to avoid sudden jarring of the cranial cervical spine!

Lateral radiographs are usually sufficient to allow a diagnosis to be made although a ventrodorsal view may allow better appreciation of any deformity of the dens. Theoretically, the latter can be best visualised using an intra-oral view taken with the head flexed at 90° to the vertebral column *but this view should be avoided* as it may result in severe iatrogenic damage. Flexed lateral views of the neck may help to demonstrate instability but these should be undertaken with care since, once again, iatrogenic damage may result.

Radiological abnormalities include (Fig. 22.3):

- Abnormal atlantoaxial alignment
- Deformed, ununited or fractured dens

Fig. 22.3 Lateral (slightly flexed) radiograph of the cervical spine of a 2-year-old Yorkshire Terrier exhibiting neck pain and moderate quadriparesis after minor trauma to the head. The wide interarcuate space, which was far less marked in the extended lateral view, demonstrates atlantoaxial instability. In addition, the atlas appears shortened which may have a bearing on the pathogenesis.

- Shortened atlas
- Increased interarcuate space (may require flexed lateral view)

In addition, trauma cases may show evidence of other fracture lines and congenital cases may show concurrent hydrocephalus (beaten copper appearance to the cranium).

Myelography and cerebrospinal fluid analysis are rarely required to rule out other causes of the clinical signs but if they are deemed necessary it is better to perform either technique using lumbar puncture rather than cerebellomedullary cistern ('cisterna magna') puncture because of the flexed neck positioning required for the latter.

Treatment

Conservative management

Conservative management requires the application of a neck brace (Fig. 21.2) together with enforced cage rest and non-steroidal anti-inflammatory treatment for pain relief. Such measures may be adequate in the case of fractures where healing of the bone should result in the restoration of stability. However, where ligament insufficiency is present such measures may allow improvement but this is usually followed by progressive deterioration once the neck brace is removed. Therefore, the indications for conservative management of congenital cases are limited and such measures should be reserved for those dogs showing only mild and non-progressive signs.

Surgical treatment

Surgical stabilisation of the atlantoaxial joint is the most appropriate treatment for cases showing severe or progressive clinical signs, particularly where instability is due to deformity and/or inadequate ligament support. The techniques can be broadly divided into those aimed at fusion of the articular facets and those involving prosthetic replacement, or augmentation, of the dorsal atlantoaxial ligament.

Fusion of the articular facets

A standard ventral approach is made to the atlantoaxial region and the longus colli muscle elevated to expose the vertebral bodies. The joint capsule is resected from the ventral aspect of the articular facets and the articular cartilage curetted. Cancellous bone, taken from the proximal humerus, is packed between and ventral to the facets. There are several methods of stabilising the atlas relative to the axis and these are illustrated in Fig. 22.4. Stabilisation of the joint is most simply achieved by placement of cortical bone screws, preferably in a lagged fashion, across the articular facets (Denny *et al.*, 1988). Pins may be used as an alternative (Sorjonen & Shires, 1981), although these cannot be used to compress the sites and migration may be a problem. Instead of placing implants across the facets, stability may be achieved by securing a small plate, e.g. ASIF mini H-plate (Stead *et al.*, 1993), to the vertebral bodies or placing two screws into each vertebra and joining them with polymethylmethacrylate (PMMA) bone cement.

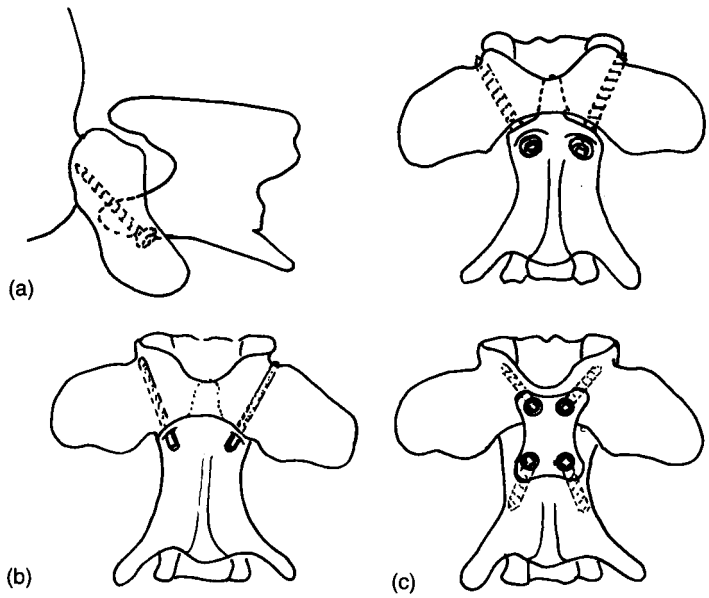

Fig. 22.4 Methods of surgical management of atlantoaxial subluxation via a ventral approach. (a) Lag screw fixation of ventral articular facets. (b) Transarticular Kirschner wires. (c) H-plate and screws (or PMMA).

The advantage of these techniques is that they require the patient to be kept in a 'safe' position with respect to iatrogenic cord damage and that they do not rely, ultimately, on the implants used but on the promotion of a biological fusion.

Prosthetic replacement or augmentation of the dorsal atlantoaxial ligament

These techniques require a dorsal approach to the dorsal spine of the axis and the dorsal arch of the atlas. A ligament prosthesis of wire or heavy-gauge multifilament non-absorbable suture material is passed through the atlas, dorsal to the spinal cord and through holes drilled in the dorsal spine of the axis. Following reduction of the subluxation the prosthesis is tightened so that vertebral alignment is maintained (Fig. 22.5).

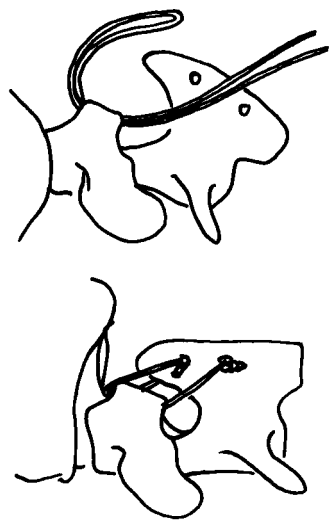

Fig. 22.5 Dorsal stabilisation of the atlantoaxial joint with wire sutures to replace the atlantoaxial ligament.

The disadvantages of these techniques includes their requirement for flexion of the atlantoaxial joint during placement of the prosthesis through the atlas. This is associated with a high incidence of iatrogenic cord damage and cardiorespiratory arrest. In addition they rely, long term, on the integrity of the prosthesis for maintenance of stability and fatigue failure of the implant or cheese-wiring through the dorsal spine of the axis may lead to recurrence of the clinical signs. *As a result of the complications inherent to these techniques they have been largely superseded by those aimed at ventral fusion of the vertebrae.* However, a case

report by Jeffery (1996) described how dorsal stabilisation, using crossed pins, could be supplemented by the promotion of fusion between the dorsal spine of the axis and the dorsal arch of the atlas via placement of cancellous bone between the two (Fig. 22.6). Such fusion would certainly help to avoid the long-term problems associated with the dorsal techniques.

Postoperative care

External support in the form of a neck brace may be of use although this may add to the respiratory stridor evident. If surgical stability is considered satisfactory it may be preferable not to add external support. Strict rest should be enforced for 4 weeks after surgery.

Prognosis

To a large extent this is related to the cause and severity of the clinical signs. Those dogs showing only neck pain and/or mild paresis have a fairly good prognosis whilst those that are non-ambulatory with congenital deformity of the joint have a guarded outlook. In cases with additional congenital problems (e.g. hydrocephalus) then the prognosis is extremely guarded and euthanasia should be considered. With respect to surgical technique, the difference in success rates for the two types of procedure varies between authors but there is a marked tendency for the results of the ventral fusion techniques to be more favourable than those with dorsal fixation. Comparisons of the two surgical strategies were made by Denny *et al.* (1988), where 8 out of 13 treated by dorsal fixation improved compared to 9 out of 10 dogs treated by ventral fusion, and by Thomas *et al.* (1991) where improvement was seen in only 2 of 11 treated by dorsal fixation compared to 8 of 18 treated by ventral fusion. The case report by Jeffery (1996) provides a better option than previous dorsal techniques and certainly warrants further evaluation. This technique may have advantages over those applied ventrally, particularly in smaller dogs (say less than 2kg bodyweight) where satisfactory placement of bone screws across the articular facets can prove problematic.

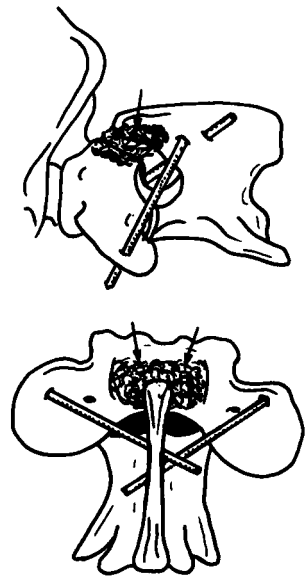

Fig. 22.6 Dorsal stabilisation of the atlantoaxial joint by placement of crossed pins and a cancellous bone graft (arrows).

Atlantoaxial subluxation in cats

This condition has been reported in the cat (Jaggy *et al.*, 1992). The clinical and radiographic signs were similar to those recorded in the dog and successful treatment may be achieved by ventral cross-pinning.

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Chapter 23
Cervical Disc Disease

Cervical disc disease is a common cause of neck pain in the dog. Any breed can be affected but the Spaniel family appear to be overrepresented. The pain caused by disc extrusion can be very severe and may be accompanied by neurological deficits. The clinical diagnosis is easily confirmed radiographically and appropriate treatment has a very good success rate except, perhaps, in those cases showing very severe neurological dysfunction. The aetiology and pathogenesis of this disease is detailed in Chapter 5 (p. 35).

History

Cervical disc extrusion

Almost any breed may be affected although the small breeds and the Spaniel family tend to be overrepresented. The condition is very rarely seen in dogs less than 1, or even 2, year(s) of age and uncommon in those greater than 11 years of age. The owners may report anything from the dog crying when it turns its head sharply, through persistent pain with a low head carriage and possibly carrying one forelimb, to complete collapse (usually after a head-on collision).

Cervical disc protrusion

This is more commonly seen in the larger breeds, especially the Dobermann Pinscher, in association with cervical spondylopathy. The history is usually one of progressive hindlimb incoordination but there may also be reports of signs related to neck pain, reluctance to feed from ground level, or forelimb lameness/hypermetria.

The diagnosis and management of cervical disc protrusions is dealt with elsewhere (see Chapter 24, p. 231). The problem of disc extrusions will be discussed here.

Clinical signs

The signs caused by a disc extrusion (type I prolapse) will be related not only to the site involved but also the amount of material extruded, the force of the impact on the spinal cord, and whether there is involvement of spinal nerve roots. The signs may develop suddenly or worsen progressively over a few days. Neck pain is the most frequent presenting sign with the head carried in a typically low posture and increased tone with or without fasciculations in the neck musculature. Neurological dysfunctions are not seen as often as with thoracolumbar disc protrusion since the vertebral canal is relatively wide in the cervical region and thus the spinal cord is less prone to compression. When seen they usually take the form of paresis or lameness affecting one forelimb. This may result from impingement of one of the nerves contributing to the brachial outflow and one of the more caudal cervical discs should be suspected. More severe signs, and even quadriplegia, may be seen very occasionally. The presenting signs of cases with cervical disc extrusion seen at Bristol University Veterinary School over a 5-year period are detailed in Table 23.1.

Differential diagnosis

Causes of neck pain, with or without neurological deficits, include:

- Intervertebral disc extrusion/protrusion
- Neoplasia
- Inflammatory central nervous system (CNS) disease (e.g. granulomatous meningoencephalomyelitis)
- Fracture/luxation
- Atlantoaxial subluxation
- Discospondylitis
- Spinal arteritis

Diagnosis

Definitive diagnosis depends on radiography and, in most cases, diagnostic radiographs of the cervical spine can only be achieved under general anaesthesia. The latter is mandatory for myelography. Lateral views are the most useful but ventrodorsal views may be beneficial and should be carried out if myelography is to be performed, to allow evaluation of the significance of any abnormalities noted.

Radiographic changes include (Fig. 23.1):

- Calcification within the intervertebral disc spaces
- Narrowing of the intervertebral disc spaces

Table 23.1 Clinical presentation of 47 dogs with cervical disc extrusions seen at Bristol University Veterinary School between 1985 and 1989.

Clinical sign	No.
Neck pain only	32
Neck pain and mild paresis in one or two limbs	9
Neck pain and quadriparesis	5
Neck pain and quadriplegia	1
Total	47

- Evidence of a dorsal protrusion
  - a dorsal tail
  - opacification of the intervertebral foramen

Myelography may help to further identify a disc extrusion. This may be necessary to reach a diagnosis if the plain films are normal, since not all disc problems are evident on standard radiographs, or if it is not clear exactly which disc space is involved or on which side the extruded material is present within the vertebral canal, which may be important depending on the planned treatment. Further elucidation of a cervical disc prolapse with myelography is best carried out by cisternal puncture, except perhaps when atlantoaxial subluxation has not been ruled out, when lumbar puncture might be considered safer.

Myelographic changes include (Fig. 23.2):

- Elevation of the ventral column
- Narrowing/loss of the dorsal column
- Lateralisation of the extrusion

Treatment and prognosis

The methods of treatment fall, broadly, into three categories: conservative, fenestration and decompression by means of ventral slotting. Each of these will be looked at in turn in terms of their aim, what they involve and their success rates. This information will then be brought together to provide guidelines as how best to manage these cases.

Conservative management

This comprises strict confinement or 'cage rest' for 4–6 weeks whilst the annulus fibrosus heals and the inflammatory response associated with the

Fig. 23.1 A lateral radiograph of the cervical spine of a 4-year-old German Shepherd Dog exhibiting neck pain and mild paresis. The C2–3 and C4–5 discs are both mineralised and the C3–4 disc space appears narrowed. From this radiograph a diagnosis of degenerative disc disease can be made. The presence of a disc prolapse causing the clinical signs is likely but not proven by this radiograph.

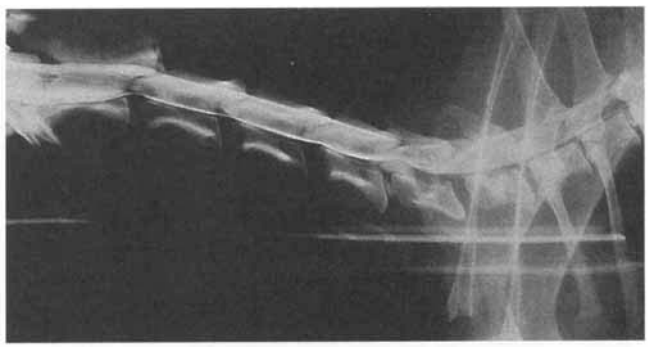


Fig. 23.2 A post-myelogram, lateral radiograph of the cervical spine of a 6-year-old Cocker Spaniel exhibiting neck pain and marked hemiparesis. The C5–6 disc is mineralised and the myelogram clearly delineates a mass of mineralised material within the vertebral canal, causing compression of the spinal cord, at the level of C5–6. From this study a definitive diagnosis of degenerative disc disease with extrusion at C5–6 can be made.

disc protrusion settles down. Non-steroidal anti-inflammatory drugs (NSAIDs) may be used to allow comfortable rest (refer to Table 7.1). If the dog is showing moderate to severe neurological signs then corticosteroids may be useful in the initial stages. They are, however, best avoided for simply controlling pain as they tend to slow down the healing of the annulus fibrosus, unless of course the NSAIDs do not provide satisfactory relief from pain. The use of methylprednisolone sodium succinate is discussed elsewhere (see Chapter 21, p. 207).

Reports of large numbers of cases treated conservatively are few in number. The majority of cases with pain with or without mild neurological signs will improve (possibly 70%). Those with severe pain and/or neurological signs have a much lower chance of recovery. In addition, the recurrence rate of clinical signs following conservative management over a 2-year period is in the order of 36%.

Fenestration

The standard technique involves a midline ventral approach to the cervical vertebral column (Fig. 23.3) and the creation of a window in the ventral annulus which allows the removal of any remaining nucleus pulposus from the disc (Fig. 23.4). In so doing the aims are to:

- Reduce the degree of pain by 'decompression' of the disc itself
- Eliminate the 'dynamic effect' described by Olsson (1958) of nuclear material being gradually forced dorsally resulting in the persistence of clinical signs, or their worsening, over a prolonged period of time
- Prevent recurrence by removing any remaining nucleus pulposus from not only the disc causing the current problem but also from the other 'high-risk' discs

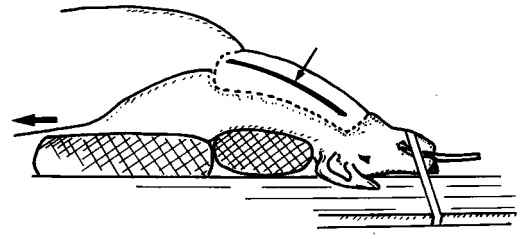
With respect to which discs should be treated simultaneously there is some variation between authors. Looking at the relative incidence of disc extrusions in the cervical region, shown in Table 23.2, it is seen that in 98% of cervical disc extrusions it is one of the first five discs that is involved. Since the C7–T1 disc space can be difficult to fenestrate, it is generally recommended that this one is omitted unless this is the site of extrusion or there is evidence of degenerative change radiographically.

In cases with neck pain as the only clinical sign, or when pain is accompanied by mild neurological signs, then fenestration can be expected to allow a complete recovery in over 90% of cases within 2–6 weeks of surgery. In those cases with marked neurological deficits, i.e. quadriparesis or quadriplegia, the success rate is worse with only 40–50% of cases making a satisfactory outcome within 10–12 weeks. The recurrence rate in one study (Russell & Griffiths, 1968) fell from 36.3% following conservative management to 5.6% after fenestration of at least the first four cervical discs.

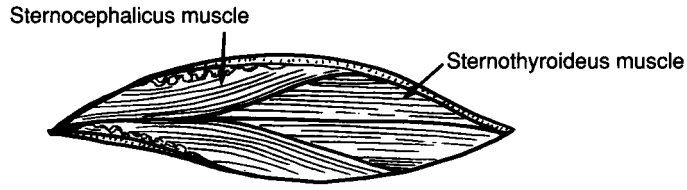
Ventral slot

With this procedure the same approach to the vertebral column is made as for fenestration (Fig. 23.3) but in this case a window is made through

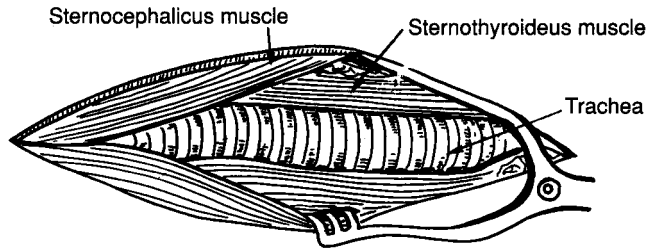
(a) The dog is placed in dorsal recumbency with the neck extended over a sandbag and the forelimbs drawn caudally alongside the thorax.



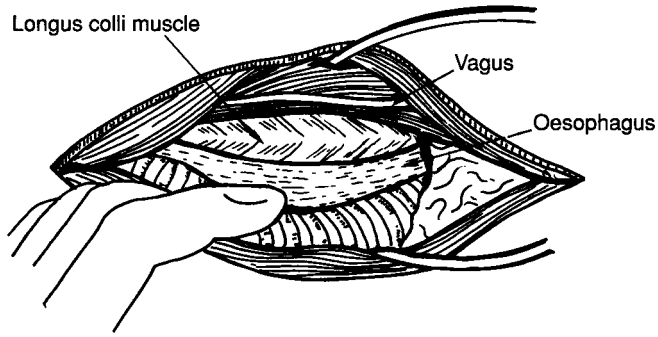
(b) A ventral, midline incision is made extending from the level of the atlas to the manubrium sternum, exposing the sternothyroideus muscles.



(c) The paired bellies of the sternocephalicus and sternothyroideus muscles are identified, separated and retracted to expose the trachea.



(d) The trachea and oesophagus are retracted to the left side of the neck and the right vagus and carotid artery displaced to the right to expose the longus colli muscle. The ventral processes at the caudal end of each vertebra can be palpated through this muscle. The process on C2 lies just behind the level of the most prominent points of the wings of the atlas and the ventral process of C5 lies between the cranial limits of the transverse processes of C6. One or both of these landmarks is/are used to orientate the relevant disc spaces.



(e) The attachment of the longus colli on each ventral process is cut and retraction of the muscle fibres allows exposure of the ventral annulus of the disc space just caudal to each ventral process. Closure involves apposition of the separated parts of the longus colli muscle with an interrupted or continuous suture pattern, coaptation of the sternothyroideus muscle and then the sternocephalicus muscle with continuous suture patterns, and then the subcutaneous, subcuticular and skin layers in a routine fashion.

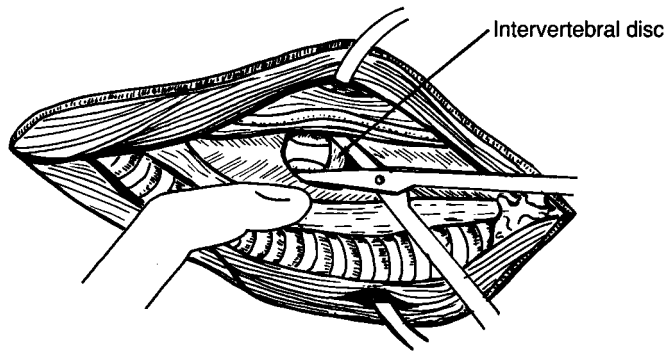


Fig. 23.3 Ventral approach to the cervical spine.

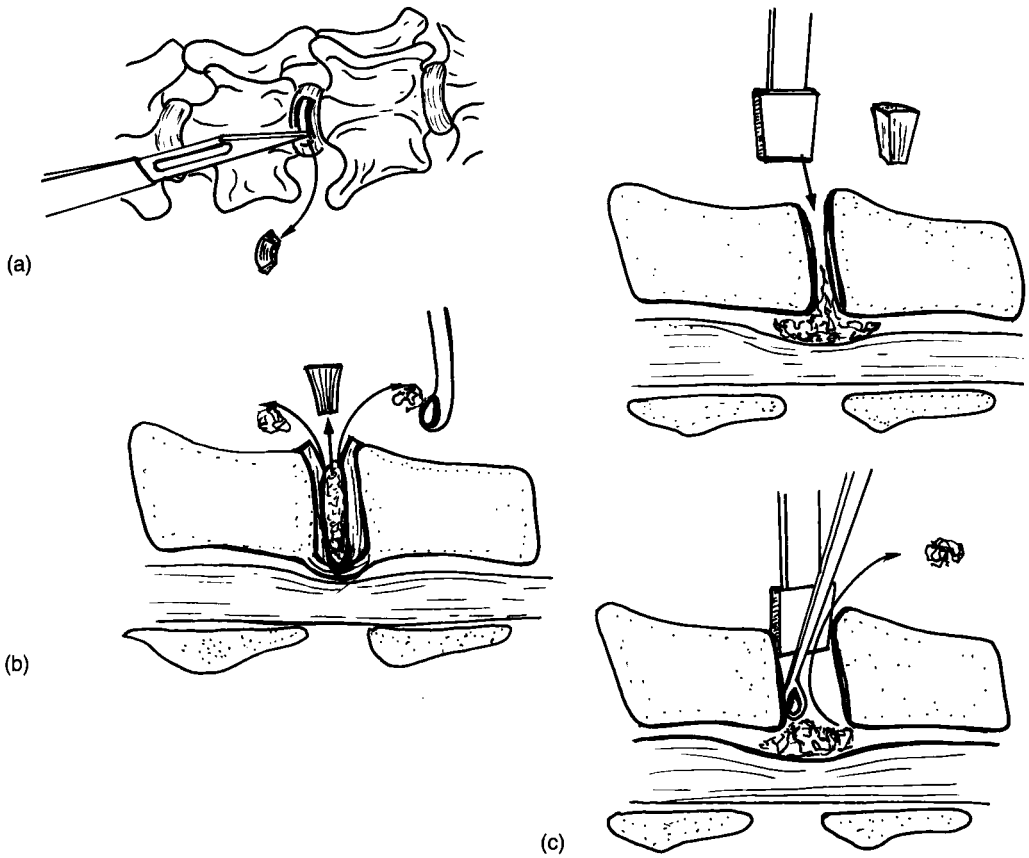


Fig. 23.4 Fenestration of each disc space is achieved by removal of a 'window' of ventral annulus using a no. 15 or 11 scalpel blade and then evacuating the nucleus pulposus using a dental tartar scraper (kept specifically for this purpose!), a small curette or scalpel blade. (a) The appearance of this during surgery (continuing on from Fig. 23.3e). (b) A schematic, 'cut away', lateral view of what is achieved by fenestration. Note that the extruded material remains within the vertebral canal. (c) In some cases the amount of disc material that can be removed by fenestration can be improved by using a vertebral distractor in the disc space to gain better access to the dorsal aspect of the disc. The blade of the distractor (Veterinary Instrumentation) is inserted into the vertebral disc space after fenestration and then rotated to separate the vertebral bodies as shown.

Table 23.2 Relative incidence of disc extrusions in the cervical spine.

Reference	No. of cases	Percentage (%) incidence at each disc space					
		C2–3	C3–4	C4–5	C5–6	C6–7	C7–T1
Olsson & Hansen (1952)	63	24	14	13	21	22	6
Denny (1978)	40	27	35	13	9	16	0
Fry *et al.* (1991)	111	42	20	19	12	6	1
Dallman *et al.* (1992)	105	29	24	21	15	9	2
Total/average	319	31	23	17	14	13	2

the disc and adjacent vertebral end plates into the vertebral canal. In general, the defect created should be no more than one-third of the width and one-quarter to one-third of the length of the vertebral bodies (Fig. 23.5). This procedure will effectively remove any remaining nucleus pulposus from the disc space, either as part of the process or else by fenestration immediately prior to it, and also allow removal of the extruded material from the vertebral canal. In so doing the aims are to:

- Reduce the degree of pain by decompression of the spinal cord as well as any benefit incurred by 'decompression' of the disc
- Improve the likelihood of neurological recovery by decompression of the spinal cord
- Eliminate the 'dynamic effect' discussed in Chapter 5
- Prevent recurrence by removing any remaining nucleus pulposus from the disc space (+/– other high-risk discs if these are fenestrated concurrently)

In cases with neck pain as the only clinical sign, or when pain is accompanied by mild neurological signs, then ventral slotting can be expected to allow a complete recovery in over 90% of cases within 2–3 weeks of surgery. In those cases with marked neurological deficits, e.g. non-ambulatory but with voluntary movement still present, the success rate is in the order of 70–80%, while the prognosis for those with quadriplegia remains guarded with perhaps only 30–40% regaining a reasonable ability to walk.

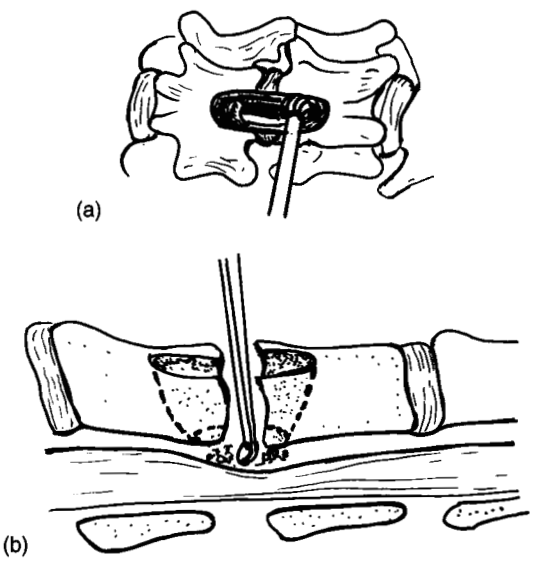


Fig. 23.5 Creation of a ventral slot involves removal of bone from adjacent vertebral bodies so that access to the vertebral canal is gained, allowing retrieval of the extruded nucleus pulposus. (a) The appearance of this during surgery (continuing on from Fig. 23.3e). More exposure of the vertebral bodies is required than for fenestration. The slot is centred just cranial to the disc space because the disc lies at an angle, sloping dorsocranially (see b) and the defect created involves no more than one-third the width and one-quarter to one-third the length of each vertebral body. (b) A schematic, 'cut away', lateral view of what is achieved by a ventral slot. Note that the extruded material is removed from within the vertebral canal.

Fenestration versus ventral slot

A direct comparison of these two techniques has been published by Fry *et al.* (1991) in which 41 dogs were treated by means of fenestration and 73 by means of a ventral slot. *All* the cases in this series were ambulatory at the time of presentation. The criteria determining which technique was used were not stated but the fact that this was a multicentre study might indicate that the choice was very much surgeon orientated. Details of the intraoperative and postoperative complications and results, as determined from clinical examination as well as owner opinion, were recorded.

No intraoperative complications were seen during any of the fenestrations while 24% of the slots had complications such as haemorrhage, respiratory acidosis and cardiac arrhythmias. A range of postoperative complications were seen in 68% of fenestrations and 37% of slots. However, when swelling at the surgical site was taken out of this list the postoperative complication rate became 12% for fenestrations and 30% for slots. Of those that had ventral slots, one died and three were put to sleep postoperatively due to a deterioration in their neurological status. The cases treated by ventral slotting had a significantly longer period of postoperative hospitalisation. The results with respect to the neurological improvement, as determined by clinical examination and the

Table 23.3 Results of fenestration and ventral slotting in the management of cervical disc extrusions (from Fry *et al.*, 1991).

	Treatment	
	Fenestration	Ventral slot
Neurological examination (%)		
Normal	47	65
Improved	19	20
Owner's comments (%)		
Normal	72	76
Improved	16	19
Successful outcome (%)	88	95
Time to reach best (average) (weeks)	9.4	4.1

owner's impression as to the degree of return to 'normal', are given in Table 23.3.

From these results certain general conclusions can be made regarding the relative advantages of these two techniques and guidelines for the management of cases with cervical disc extrusions can be drawn.

Advantages of fenestration:

- Less specialised instrumentation is required
- Myelography is often unnecessary since the specific disc space involved does not have to be identified, and thus the risk of this procedure may be avoided
- The surgery is unlikely to cause a worsening of the clinical signs
- As high a success rate as with ventral slotting can be expected in cases with mild/moderate pain +/– mild neurological signs
- Treatment of all the 'high-risk' discs simultaneously dramatically reduces the likelihood of recurrence

Disadvantages of fenestration:

- On average, it takes longer for a dog to reach its best postoperatively
- Although postoperative deterioration is uncommon, Tomlinson (1985) reported two cases in which the dogs became tetraparetic after fenestration and, in the authors' experience, the degree of pain suffered by some dogs may be worse immediately after this procedure
- A lower success rate is seen in cases with marked neurological signs

Advantages of ventral slotting:

- On average, the recovery rate is much quicker
- A higher success rate is seen in cases with marked neurological deficits

Disadvantages of ventral slotting:

- More specialised instrumentation is required
- Myelography is often necessary to allow accurate localisation of the lesion
- There are greater surgical risks with respect to iatrogenic damage to the spinal cord or excessive haemorrhage from the the internal vertebral sinuses
- Despite an overall improvement in speed of recovery, it is the authors' experience that some patients may exhibit worse neck pain starting 1–3 days postoperatively and lasting for several days

From these relative advantages and disadvantages the guidelines for case management of dogs with cervical disc extrusions can be summarised as detailed below:

Mild to moderate pain/paresis:

- First episode = conservative
- Persistent or recurrent = fenestrate

Moderate to severe pain/paresis:

- Ventral slot
- Fenestrate (may take longer to improve and perhaps less likely to do so in severe cases)

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Hamish R. Denny,Steven J. Butterworth

Chapter 24
Cervical Spondylopathy

Aetiopathogenesis

The term cervical spondylopathy describes a 'syndrome' with a complex aetiology which has led to it also being referred to variously as cervical vertebral instability, cervical spondylolisthesis, cervical vertebral stenosis, caudal cervical vertebral malformation-malarticulation and caudal cervical spondylomyelopathy as well as 'Wobbler syndrome'. The syndrome is caused by vertebral malformation or instability, with or without an associated soft tissue hypertrophy or hyperplasia, which results in compression of the spinal cord. Classification of the underlying pathology has been described by Seim & Withrow (1982) and is illustrated in Fig. 24.1.

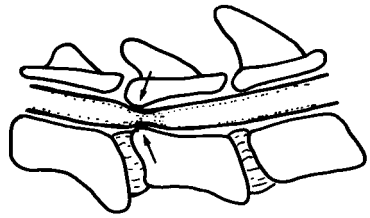
(1) *Vertebral malformation* (Fig. 24.1a) – congenital malformation and malarticulation of the vertebral bodies, the vertebral arches and the articular facets cause stenosis of the vertebral canal which worsens, relatively, as the puppy grows. Such stenosis resulting in clinical signs usually affects the C3–6 region in Great Danes and the C5–7 region in the Dobermann Pinscher. There are also reports of vertebral canal stenosis in the Basset Hound at C2–4 but the incidence of clinical signs in this breed appears to be low.

(2) *Vertebral 'tipping' or 'tilting'* (Fig. 24.1b) – adjacent vertebral bodies may be malpositioned with respect to one another such that the cranial pole of the caudal vertebra extends dorsally further than the caudal pole of the cranial vertebra. Such tipping may be present when the cervical spine is in a neutral position and worsen as the neck is flexed or else only becomes apparent with flexion. This has been suggested as a cause of static or dynamic compression in immature Dobermann Pinschers.

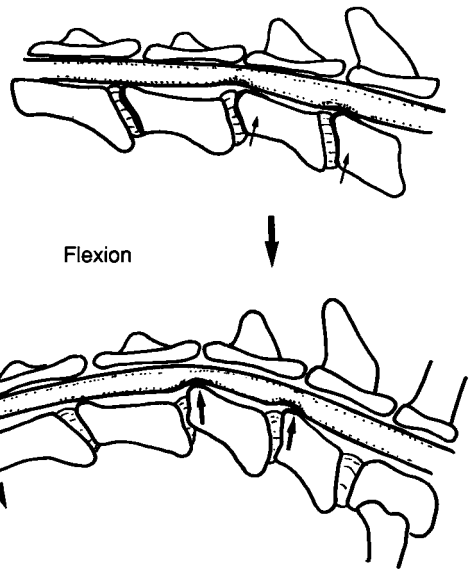
(3) *Ligamentum flavum disease* (Fig. 24.1c) – vertebral instability resulting from malformation may lead to secondary hypertrophy or hyperplasia of the ligamentum flavum which then causes dorsal compression of the spinal cord and has been reported in the Great Dane.

(4) *Intervertebral disc protrusion (Hansen type II)* (Fig. 24.1d) – chronic fibrinoid degeneration of the intervertebral disc together with hypertrophy or hyperplasia of the dorsal annulus fibrosus leads to disc protrusion and ventral compression of the spinal cord. Unlike the situation in man, the dorsal longitudinal ligament is not a major stabiliser of the cervical spine and is only passively involved in the resulting spinal cord compression. The compression is usually dynamic in that it is increased by hyperextension of the neck and reduced when the neck is flexed or put under traction. Whether vertebral malformation contributes to this degeneration by causing subclinical instability is unknown and it has been suggested that the disc degeneration may itself cause decreased stability of the ventral compartment of the cervical spine. This form of spondylopathy usually affects the caudal cervical spine and is seen in a number of breeds, most commonly the Dobermann Pinscher.

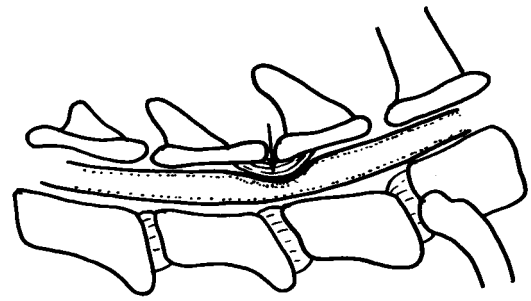
(5) *Hourglass compression* (Fig. 24.1e) – in some cases, usually young Great Danes, the compression is multidirectional with hypertrophy or hyperplasia of the ligamentum flavum (dorsal) and annulus fibrosus (ventral) together with malformation or osteoarthritis of the articular facets (lateral), causing an hourglass appearance during myelography.



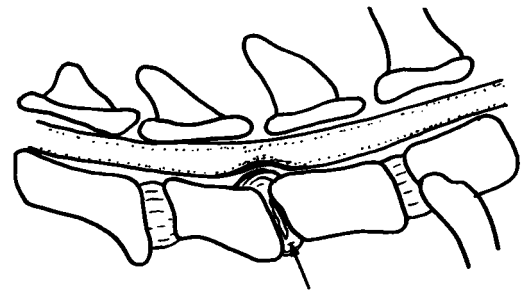
(a) Vertebral malformation causing osseous stenosis of the vertebral canal.



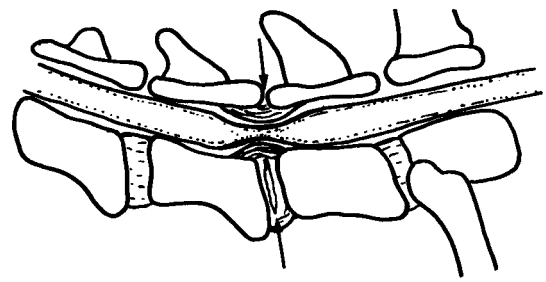
(b) Vertebral 'tipping' or 'tilting' creating malalignment of the vertebral canal.



(c) Ligamentum flavum hypertrophy caused by low-grade vertebral instability resulting from malformation.



(d) Intervertebral disc protrusion (Hansen type II) associated with low-grade vertebral instability resulting from malformation.



(e) Hourglass compression where the vertebral canal is compromised from all sides by a hypertrophied ligamentum flavum dorsally, an intervertebral disc protrusion ventrally and malformed articular facets laterally (diagram shows dorsal and ventral components only).

Fig. 24.1 Classification (Seim & Withrow, 1982) of cervical spondylopathy.

The aetiology of these pathological changes is considered to be multifactorial. Malformation of the vertebrae, causing stenosis or instability, could involve osteochondrosis lesions affecting the articular facets or vertebral end plates and thus genetic and nutritional factors may play a role. It has also been suggested that changes in breed characteristics in the Dobermann Pinscher over the last 10–15 years, with the goal of producing a heavier head on a longer neck, may have led to an

increasing incidence of the problem. Such abnormal forces acting on the immature skeleton could contribute to malformation/malarticulation.

When signs develop in the skeletally mature patient it is likely that the degree of malformation alone has been inadequate to produce clinically significant spinal cord compression. However, it may have led to long-term vertebral instability resulting in soft tissue hypertrophy and/or intervertebral disc degeneration and protrusion which then compounds the degree of stenosis and leads to progressive clinical signs.

History and clinical signs

Any age of dog may be affected and although various medium to large breeds of dog may develop signs, the vast majority will be Great Danes and Dobermann Pinschers. Other breeds sometimes seen with similar pathology to the Great Dane would include the Basset Hound, the Irish Wolfhound and some of the Mastiffs, whilst other breeds sometimes showing pathology more typical of the Dobermann Pinscher would include the Dalmation and the Bernese Mountain Dog.

The typical history depends on the site involved within the cervical spine. If the lesion involves the C2–4 region then the dogs will often develop a progressive forelimb paresis or knuckling which may show a degree of lateralisation. On the other hand, if the lesion involves the C5–7 region then the history is usually one of progressive hindlimb incoordination over a period of several months which is most often bilaterally symmetrical. Other features sometimes seen in association with such hindlimb incoordination are: difficulty in rising, the adoption of a wide-based stance, loss of balance when turning at speed and scuffing of the nails. Forelimb involvement in cases with caudal lesions usually presents as a bilateral hypermetria with short, choppy strides. The tendency for the forelimbs to hyperextend is thought to be a result of the compression causing lower motor neuron (LMN) deficits to the limb flexors and upper motor neuron (UMN) deficits to the extensors due to the median and ulnar nerves being involved directly whilst the radial nerves are relatively spared. In some cases involving either region the signs are more acute in onset with or without a previous history of a less severe, chronic problem.

On clinical examination the most common feature is that of a variable degree of paresis associated with proprioceptive deficits in the moderate to severe cases and involving predominantly fore or hindlimbs as discussed above. Forelimb lameness may be seen as a consequence of nerve root entrapment due to lateralisation of the compression. Neck pain may result from nerve root compression and is most commonly seen in dogs with an acute presentation or associated forelimb lameness. Neurogenic muscle atrophy may be present, particularly in cases with nerve root entrapment, with the spinatus muscles being most obviously affected. Urinary incontinence may be seen but is rarely associated with this condition.

Other conditions to be borne in mind, when dealing with Dobermann Pinschers, in particular, are hypothyroidism, gastrocnemius enthesiopathy/tendonopathy, cardiomyopathy and von Willebrand's disease.

Differential diagnosis

In cases with progressive onset paresis without LMN involvement, other possible causes would include:

- Intervertebral disc protrusion (Hansen type II) in the thoracolumbar region
- Congenital vertebral anomaly in the thoracolumbar region
- Discospondylitis
- Degenerative myelopathy
- Arachnoid cyst
- Neoplasia

Where there is an acute onset of clinical signs the differentials would include:

- Intervertebral disc extrusion (Hansen type I)
- Trauma
- Fibrocartilagenous embolism
- Spinal haemorrhage (spontaneous or secondary to neoplasia)
- Meningitis/myelitis
- Spinal arteritis

In dogs showing predominantly unilateral forelimb paresis or lameness, nerve sheath neoplasia should be considered also.

Diagnostic imaging

Radiography remains the principal method of investigation although computerised tomography (CT) and magnetic resonance imaging (MRI) have been reported to provide valuable additional information (Sharp *et al.*, 1992). General anaesthesia is a prerequisite for taking diagnostic radiographs. The following radiographic abnormalities associated with the condition in the Dobermann Pinscher have been listed by Lewis (1991) and are illustrated in Fig. 24.2:

- Malformation of vertebral bodies with elongation of the ventral processes and triangulation, particularly of C7, where the cranioventral border becomes lost to varying degrees
- Coning of the vertebral canal within one or more of the vertebrae such that in each one the cranial foramen is stenotic when compared to the caudal foramen
- Tilting of vertebral bodies causing malalignment of the vertebral canal floors
- Narrowing of an intervertebral disc space
- Ventral spondylosis deformans

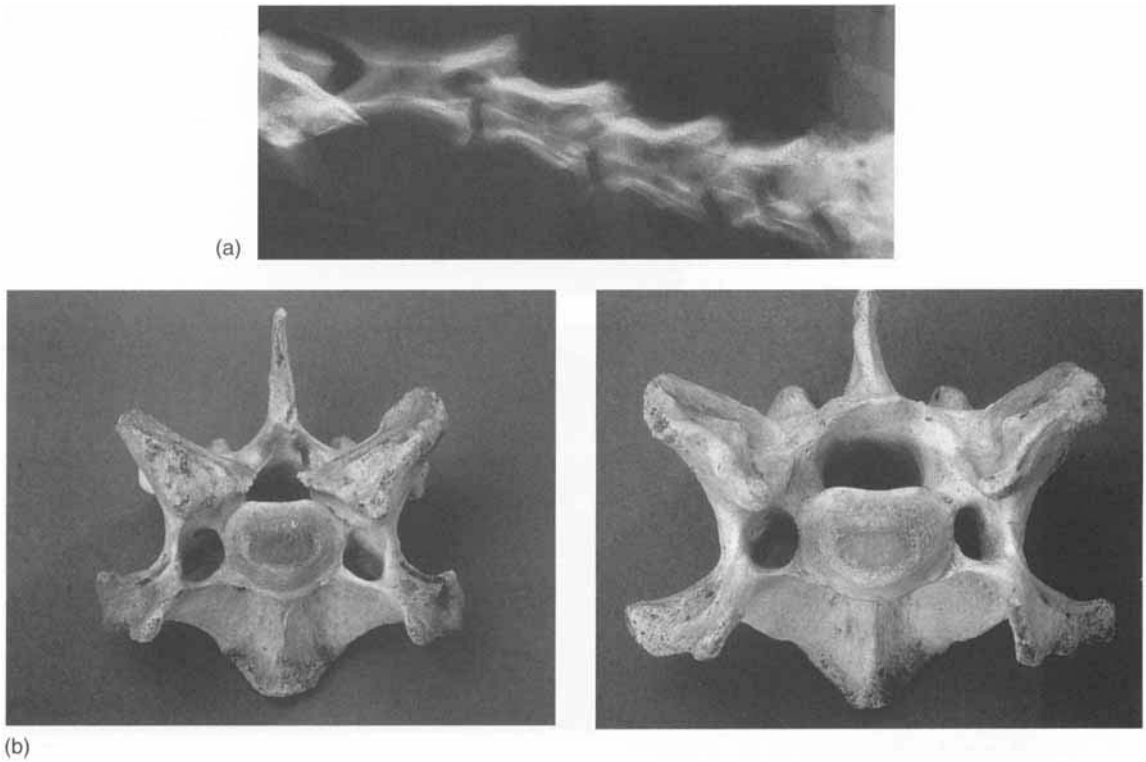
Flexion views may be useful to demonstrate instability by the appearance of, or increase in, the degree of vertebral tilting present, although it must be ensured that the cervical spine is not rotated at the same time as it is flexed, otherwise vertebral tilting may be produced artifactually. In the evaluation of clinical cases it is probably unnecessary to carry out these views during plain radiography since much more useful information would be gleaned, if they are indicated at all, following myelography.

Unfortunately, many clinically normal dogs of the breeds predisposed to the condition will show some or all of the aforementioned changes. Lewis (1991), however, considered the presence of the following changes as being significant in clinically normal, mature dogs:

- Stepping of adjacent vertebral canal floors of 3 mm or more
- Gross malformation of a vertebral body with rounding of the cranioventral border
- Cranial stenosis of the vertebral canal with a 3 mm or more difference between cranial and caudal sagittal diameters of a vertebra
- Ventral spondylosis deformans
- Intervertebral disc space narrowing

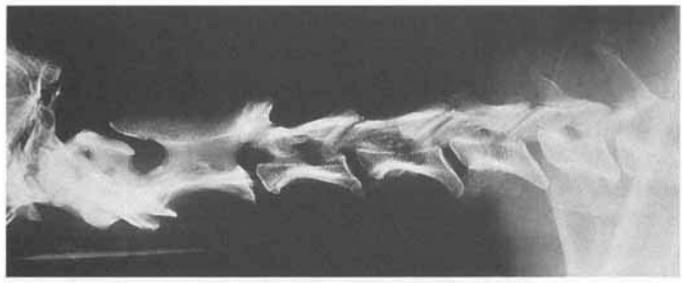
Of the Dobermann Pinschers with these features, 72% (20/28) developed clinical signs within 5 years whilst all those without these changes (87 dogs) remained normal during the same period. Such plain radiographic features, therefore, might be useful in screening dogs for breeding purposes although more evidence is required to demonstrate their value in this respect, since the ability to select which dogs will not develop the disease does not equate to the ability to select against dogs that may be carrying genes which may contribute towards the disease.

Myelography is essential, not only to evaluate the clinical significance of changes noted on plain radiographs but also to define the site(s) and direction(s) of spinal cord compression (Fig. 24.3). Lateral radiographs will demonstrate the dorsoventral compression most commonly associated with the clinical signs but ventrodorsal views may be necessary to evaluate the less common dorsal, lateral and hourglass compressions sometimes seen (Rendano & Smith, 1981). Occasionally the ventral contrast column appears to split as it lifts over a bulging dorsal annulus. This is taken to indicate lateralisation of the lesion and its presence often correlates with the presence of forelimb lameness (McKee & Butterworth, 1993). Dynamic studies may be undertaken during myelography to establish whether the degree of compression alters with positioning of the neck and whether it can be reduced or eliminated by distraction of the vertebrae. Flexion of the neck may demonstrate vertebral instability and associated cord compression, whilst views taken with the neck extended may show worsening of the compression or else additional lesions which might cause clinical signs at a later date. Such manipulations of the cervical spine should be undertaken with some care since there exists the possibility of causing iatrogenic damage to the spinal cord. Vertebral distraction is most easily achieved by applying linear traction to the neck. With the dog positioned on a slightly inclined table, the head can be 'drawn' up the table, so as to create cervical traction, and held there by a loop of bandage that is tied around the dog's nose, passing over the end of the table with sandbags suspended through it. In many



(a)

(b)



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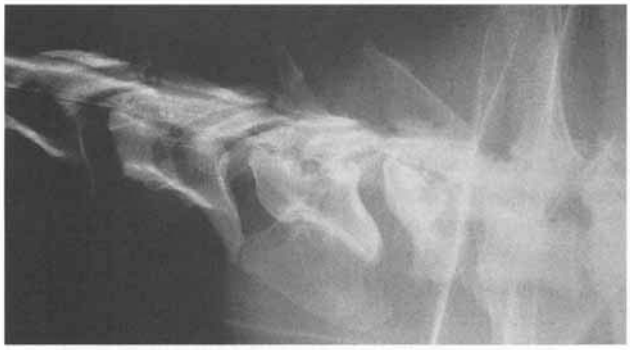

(d)

Fig. 24.2 (a) Lateral radiograph of an 11-month-old Great Dane's cervical spine. The dog was showing progressive hindlimb incoordination. The third and fourth cervical vertebrae demonstrate 'coning' of the vertebral canal. Myelography showed compression of the spinal cord at C4–5 in a ventrodorsal view.
(b) Post-mortem specimens from the same dog. Note the difference in vertebral canal dimensions when looking at the cranial end of the fifth cervical vertebra (right) compared to that of the fourth (left).
(c) Lateral radiograph of a 7-year-old Dobermann Pinscher's cervical spine. The typical signs associated with cervical spondylopathy are evident with elongation of the ventral processes, vertebral tilting/tipping at C4–5 and C5–6 and narrowing of the C6–7 disc space. However, these radiographic features would be found in many 'normal' dogs of this breed.
(d) Lateral radiograph of a 6-year-old Dobermann Pinscher's cervical spine. In this case there is marked deformity ('plough-shearing') of the seventh cervical vertebra.

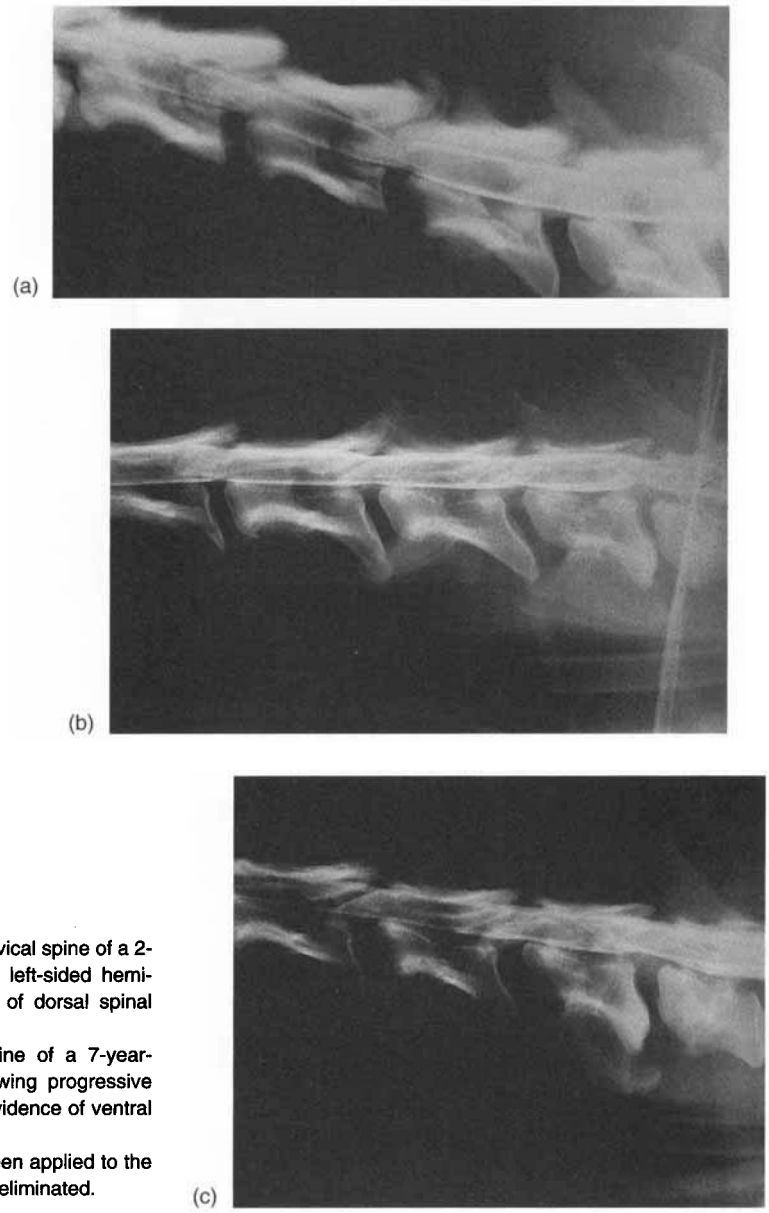

Fig. 24.3 (a) Lateral radiograph of the cervical spine of a 2-year-old Boxer-Cross which was showing left-sided hemiparesis. The myelogram shows evidence of dorsal spinal cord compression at C4–5.
(b) Lateral radiograph of the cervical spine of a 7-year-old Dobermann Pinscher which was showing progressive hindlimb paresis. The myelogram shows evidence of ventral spinal cord compression at C6–7.
(c) Same dog as in (b) after traction had been applied to the neck. The compression at C6–7 has been eliminated.

cases the best 'relaxed' view is achieved after traction has been applied and then released since more contrast will have passed caudal to the lesion, thus providing better delineation.

Treatment

Dogs with an acute onset of clinical signs, particularly when pain is the predominant feature, may respond to conservative measures involving rest and a course of prednisolone or NSAIDs in the short term, and using a harness rather than a collar and lead together with providing raised feeding bowls in the long term. However, in many acute cases, the degree of pain will improve but then progressive hindlimb incoordination will be seen.

In immature dogs showing paresis, it may be beneficial to reduce their rate of growth by decreasing the daily energy intake by up to as

much as 40%. This might allow gradual improvement in the degree of vertebral malformation and thus improvement or stabilisation of the clinical signs.

In general, unless the signs are mild and non-progressive, it is advisable to consider surgical management. In planning surgery for the treatment of a specific case it is important to define the type and number of lesions present since it is only then that an appropriate method may be chosen.

A lesion may be due to:

- Dorsal compression resulting from osseous stenosis +/– ligamentum flavum or joint capsular hypertrophy
- Ventral compression resulting from vertebral body malalignment and/or instability (i.e. static or dynamic)
- Ventral compression due to vertebral malalignment compounded by soft tissue hypertrophy and/or intervertebral disc prolapse which may be static or dynamic and may be reduced or eliminated by linear traction, i.e. be 'traction responsive'

The *surgical options* which may be tailored to eliminate such lesions include:

- Dorsal laminectomy
- Ventral slotting
- Vertebral fusion
- Vertebral distraction/fusion

In the past, poor clinical results have been achieved following ventral fenestration to treat this condition (Lincoln & Pettit, 1985) and this technique is therefore not generally employed as a therapeutic technique in the management of cervical spondylopathy.

Dorsal laminectomy (Fig. 24.4)

Dorsal laminectomy involves removal of the dorsal laminae over the site of compression. It is most appropriate in cases showing dorsal compression associated with osseous stenosis +/– hypertrophy of the ligamentum flavum but can be extended to include the articular facets if these are implicated as a source of compression. If several sites of compression exist then multiple dorsal laminectomies can be performed, even at several consecutive sites, leading to the development of a continuous dorsal laminectomy.

A major postoperative problem with this technique results from iatrogenic spinal cord damage and, in the long term, recurrence of signs may be seen due to the formation of a fibrous laminectomy membrane with subsequent stricture. The incidence of the latter can be reduced by use of a fat graft over the laminectomy site or else by creating a dorsal laminar elevation rather than laminectomy (McKee, 1988).

Ventral slotting (Fig. 24.5)

Ventral slotting allows complete removal of the disc including the entire dorsal annulus (Read *et al.*, 1983; Chambers *et al.*, 1986; Breucker *et al.*, 1989b). This procedure is most appropriate in cases with a static ventral compression but can be used to treat traction-responsive lesions or even cases with dynamic compression due to instability, since the slot will not only decompress the spinal cord but will also promote vertebral fusion. It is technically demanding in that success depends on adequate removal of the hypertrophied/hyperplastic dorsal annulus which may be complicated by haemorrhage from venous sinuses. The advantage of this technique is that once decompression is achieved it will persist but one of the disadvantages is that when two adjacent disc spaces are implicated simultaneously, slotting of these will lead to significant destabilisation of the vertebral column and is not recommended. Although the principle is the same as when this technique is used to treat cervical disc extrusions (see Chapter 23, p. 225) there is a far greater likelihood of perioperative complications when the technique is used to treat the condition being considered here. In order to reduce such complications the technique has been modified so as to create a defect in the vertebral bodies more akin to an inverted cone than a traditional slot (Goring *et al.*, 1991) but the potential advantages of this have not yet been confirmed in clinical trials.

The use of partial ventral slots to stimulate vertebral fusion may be a useful technique, particularly in dealing with secondary, subclinical sites at the same time as surgery is being performed at the primary site.

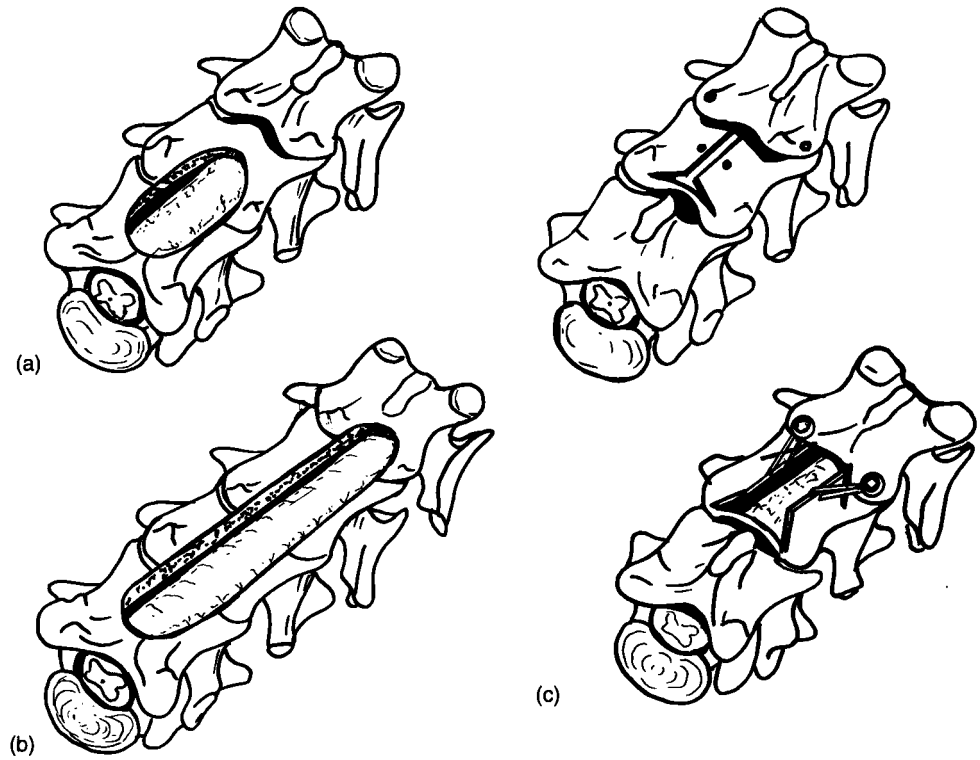

Fig. 24.4 Schematic illustrations of the principle of dorsal laminectomy.
(a) Dorsal laminectomy at a single site.
(b) Continuous dorsal laminectomy.
(c) Dorsal laminar elevation (after McKee, 1988).

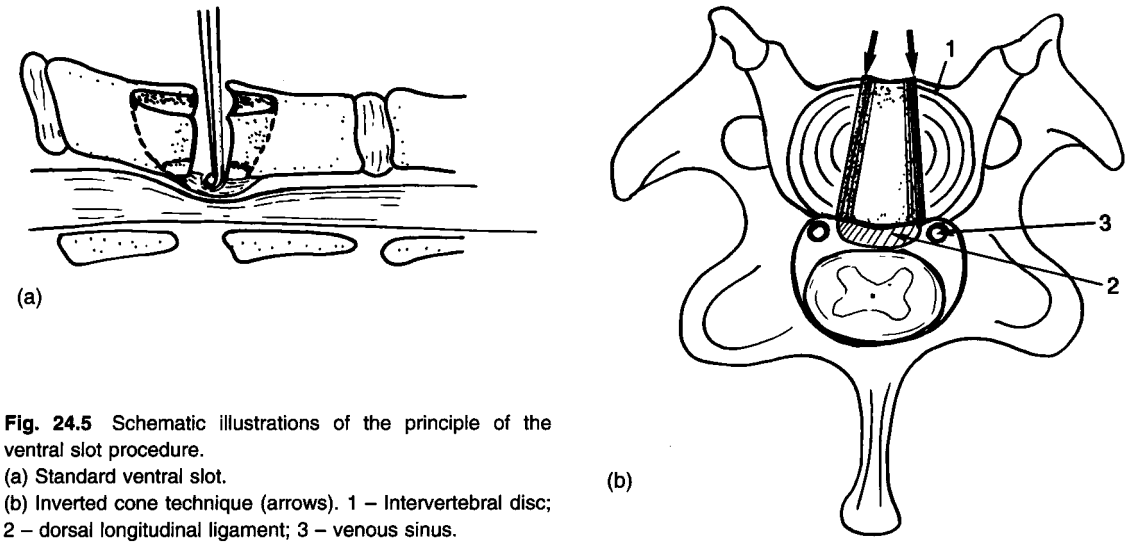

Fig. 24.5 Schematic illustrations of the principle of the ventral slot procedure.
(a) Standard ventral slot.
(b) Inverted cone technique (arrows). 1 – Intervertebral disc; 2 – dorsal longitudinal ligament; 3 – venous sinus.

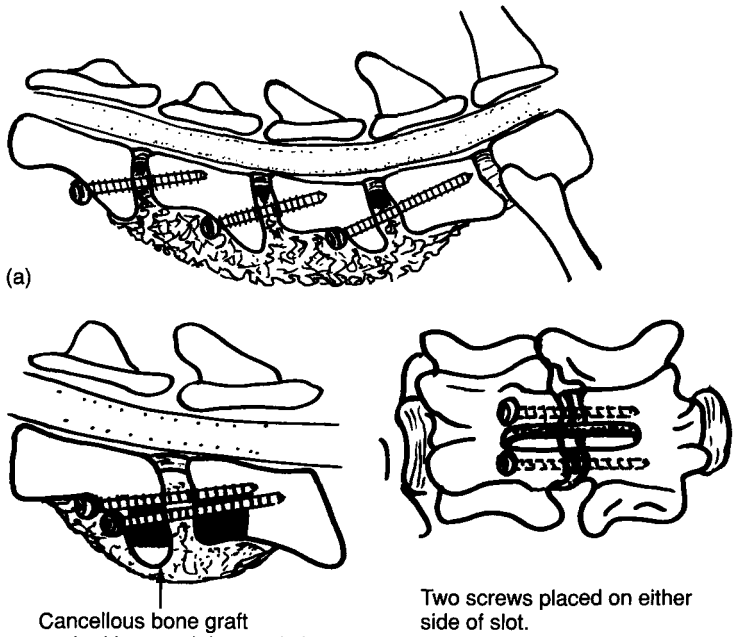

Fig. 24.6 Schematic illustrations of the principle of vertebral fusion.
(a) Using transvertebral screws and a cancellous bone graft.
(b) Using transvertebral bone screws, a partial ventral slot and a cancellous bone graft.

Vertebral fusion (Fig. 24.6)

In the past, vertebral fusion without distraction has been used to treat this condition (Denny *et al.*, 1977). The technique described involved placement of lagged, transvertebral bone screws at C4–5, C5–6 and C6–7, after fenestration of these disc spaces, along with placement of cancellous bone ventral to the disc spaces and vertebral bodies. Such a technique would be appropriate in cases with instability alone, but it has been largely superseded by attempts to create distraction as well as fusion since most cases with instability show a degree of traction-responsive soft tissue compression. However, in cases with mild signs, the creation of vertebral fusion alone may be sufficient since once stability is established there may be atrophy of the redundant soft tissues thus reducing the degree of compression on the spinal cord.

More recently, a technique of stabilising the adjacent vertebrae, without distraction of the disc space, using two positional bone screws and promoting vertebral fusion by creating a partial ventral slot and filling this with cancellous bone, has been described (Queen *et al.*, 1998), with similar results to those studies where intervertebral washers were used.

Distraction/fusion (Fig. 24.7)

The aim of all these techniques is to distract the vertebrae adjacent to the collapsed disc space and promote ventral fusion. They are appropriate only in dogs showing traction-responsive compressive lesions. Various techniques of holding the vertebrae apart have been described. Cortical grafts may be placed between the vertebrae with these being held in place using intervertebral bone screws or plastic (Lubra, Fort Collins, Co) plates (Breucker *et al.*, 1989b). The use of Harrington rods was reported by Walker (1990) and involves placement of hooks into the cranial end plate of C7 and the caudal end plate of C5 which are then wound apart along a threaded bar. The disadvantage of this technique is that two intervertebral spaces are distracted even if only one is collapsed. The intervertebral spacer most recently introduced has been a metal washer which is held in

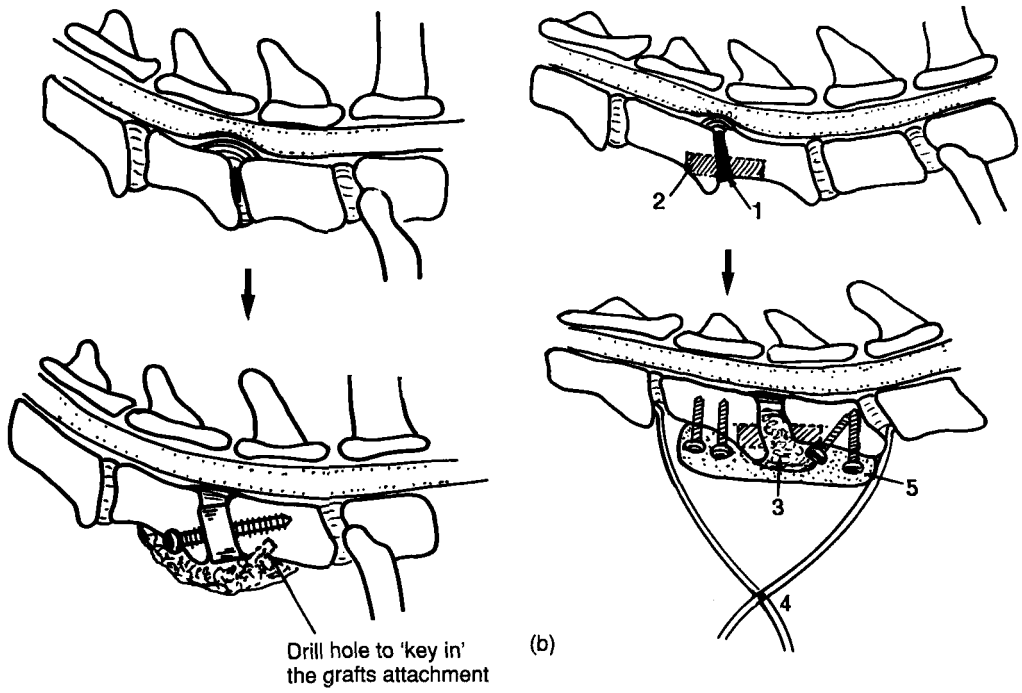

Fig. 24.7 Schematic illustrations of the principle of distraction–fusion techniques.
(a) Using an intervertebral metal washer, a transvertebral bone screw and a cancellous bone graft.
(b) Using bone screws or pins placed into the vertebral bodies and stabilised using polymethylmethacrylate after placement of a cancellous bone graft into a partial ventral slot. 1 – Disc fenestration; 2 – partial ventral slot; 3 – cancellous bone graft; 4 – retractors used to maintain distraction; 5 – polymethylmethacrylate.

this position using an intervertebral bone screw (McKee *et al.*, 1989; McKee *et al.*, 1990; McKee *et al.*, in press) (Fig. 24.7a). The advantage of this technique is that one or more disc spaces can be distracted simultaneously, allowing treatment of however many sites of compression are detected myelographically. Vertebral fusion is promoted by placement of a cancellous bone graft ventral to the vertebrae, bridging the disc space. Details of this surgical technique have been documented (Butterworth, 1995) and are illustrated in Fig. 24.8.

Alternatively, the vertebral distraction can be maintained by means of a combination of metal implants and polymethylmethacrylate (PMMA) bone cement (Fig. 24.7b). Either bone screws (Ellison *et al.*, 1988) or Steinmann pins (Breucker *et al.*, 1989a) are secured into the vertebrae and joined together ventrally using the PMMA. Fusion is achieved by creating a partial ventral slot, and packing it with cancellous bone graft, before placement of the implants and bone cement. The main disadvantage of this technique is that if more than one disc space is bridged there is a high incidence of implant failure.

These techniques may still be successful when the distraction is not maintained long term. This may be due to atrophy of the hypertrophied soft tissues once stability has been achieved (as discussed with vertebral fusion alone).

From these general principles a guide to which forms of treatment may be used for different types of compression can be drawn up and this is given in Table 24.1.

Prognosis

Although some cases with an acute onset of signs will respond to conservative measures, most will

Table 24.1 A guide to the use of various surgical options in the management of different types of cervical spondylopathy.

Type of compression	No. of sites	Surgical options*
Static dorsoventral stenosis	Single	Dorsal laminectomy Dorsal laminar elevation/laminoplasty
Static dorsoventral stenosis	Multiple	Continuous dorsal laminectomy Multiple dorsal laminar elevation/laminoplasty
Dynamic dorsoventral stenosis	Single or multiple	Vertebral fusion using transvertebral bone screw(s) +/– partial ventral slot
Traction responsive ventral compression (type II disc prolapse)	Single	i/v metal washer + transvertebral bone screw Bone screws or pins + PMMA Ventral slot
Traction responsive ventral compression (type II disc prolapse)	Multiple	i/v metal washers + transvertebral bone screws
Static ventral compression (type I and some type II disc prolapses)	Single	Ventral slot Vertebral fusion using transvertebral bone screws +/– partial ventral slot (best if signs are mild)
Static ventral compression (some type II disc prolapses)	Multiple	Dorsal laminectomy Vertebral fusion using transvertebral bone screws +/– partial ventral slot (best if signs are mild)

i/v = intervertebral; PMMA = polymethylmethacrylate.

* In most cases involving exposure of the vertebral bodies, cancellous bone may be used to promote vertebral fusion.

develop progressive clinical signs and will eventually require euthanasia. The success of surgical treatment is heavily dependent upon a number of factors, including:

- Severity and 'reversibility' of the clinical signs
- Age of the dog, with immature patients having a worse prognosis than middle-aged dogs
- Number of compressive lesions
- Surgical technique chosen together with surgeon's experience of that technique

The 'reversibility' of the spinal cord dysfunction is obviously out of the surgeon's control and it is possible that more advanced imaging techniques such as CT might allow a more accurate prognosis in this respect (Sharp *et al.*, 1992). The reported success rates for various surgical techniques is given in Table 24.2 but generally lies in the range of 75 to 90%. In general, of those cases that are considered to have successful outcomes about half will be normal and half will have mild incoordination which does not compromise the dog's quality of life.

Long-term follow-up of cases treated surgically has shown a varying incidence of recurrence at the disc space adjacent to the one treated previously, with some reports quoting up to a 26% incidence. This so-called 'domino effect' is thought to be due to increased stress across the disc space after the first one has fused and may occur any time from 6 months to several years after the initial surgery. A comparison of two techniques (ventral slotting and intervertebral washers) has been published by Rusbridge *et al.* (1998). Of 14 dogs that had ventral slots, 6 (43%) were euthanased for reasons directly related to the treatment (1) or consequent deterioration in neurological function due to a 'domino' lesion (5). In the case of 14 dogs treated with intervertebral washers, 8 (57%) were euthanased for the same reasons (2 and 6, respectively). However, in a study by McKee *et al.* (in press), of 76 dogs treated using intervertebral washers whose outcome was known, 9 were euthanased within 6 months due to complications relating to the treatment and a further 17 showed significant deterioration in neurological function

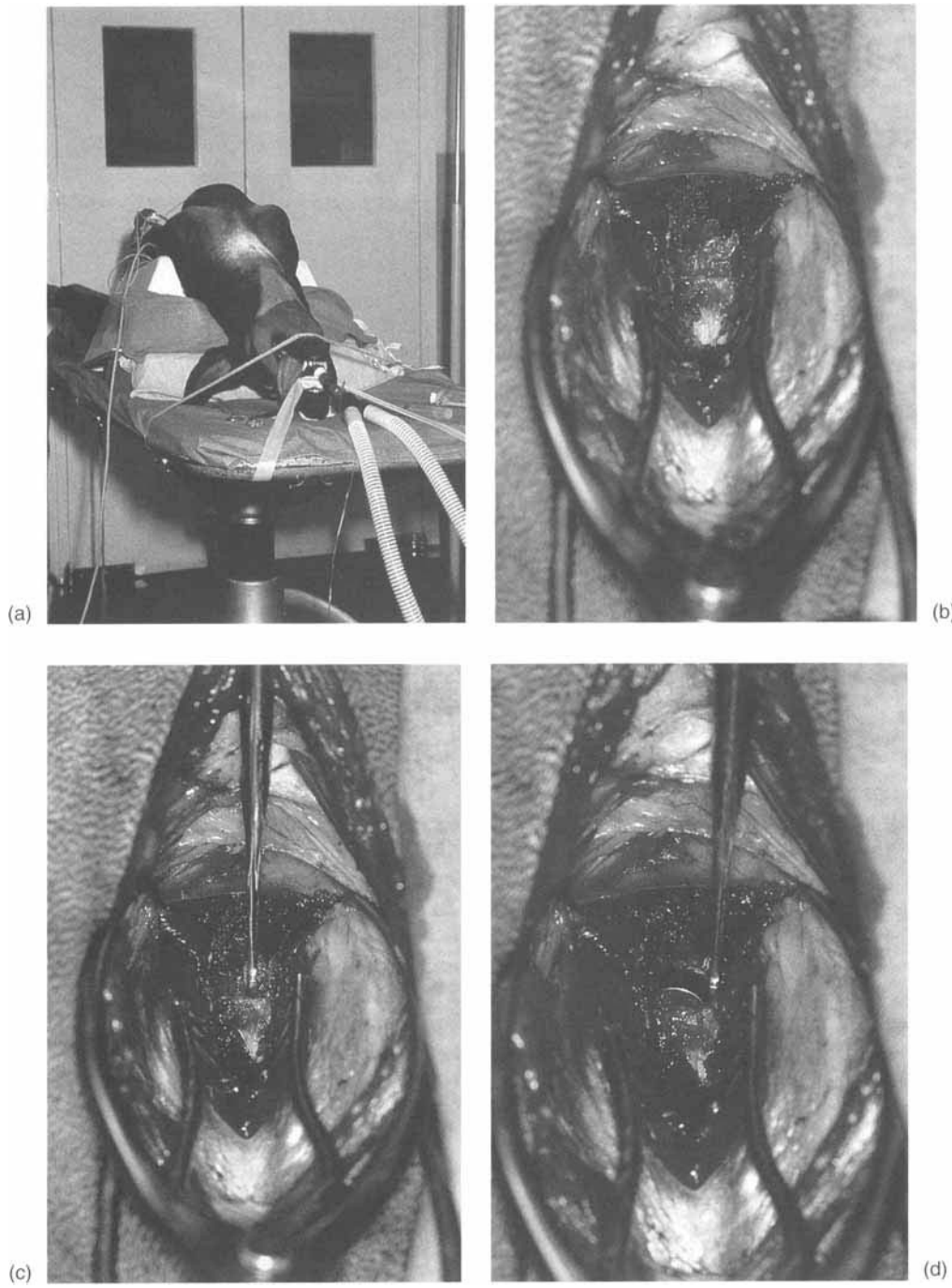

Fig. 24.8

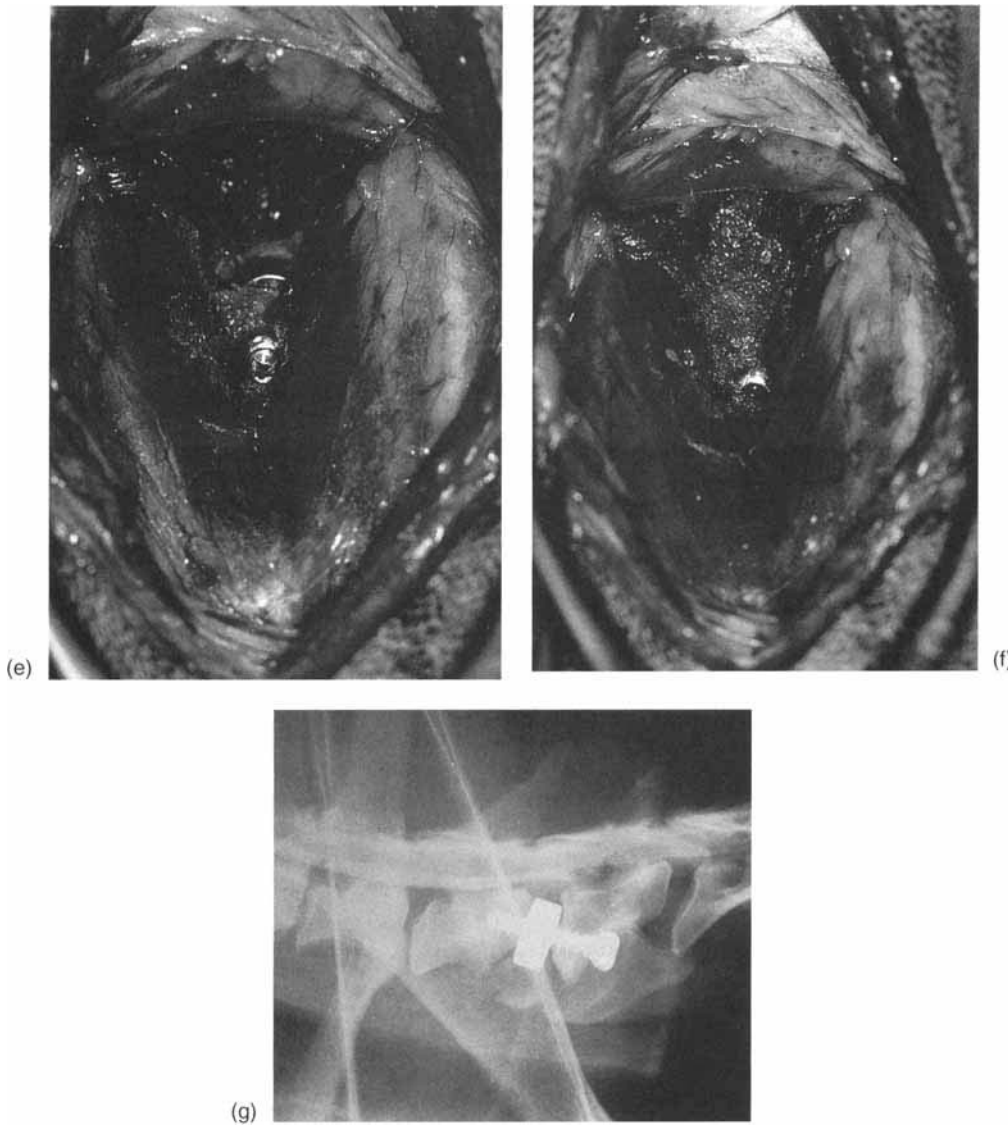

Fig. 24.8 Technique used for placement of intervertebral metal washer, transvertebral bone screw and cancellous bone graft to treat a traction-responsive disc protrusion at C6–7 secondary to cervical spondylopathy.
(a) The dog is placed in dorsal recumbency with its neck extended and under slight traction using tapes anchored on the upper canines.
(b) A standard ventral approach to the cervical spine is used to expose the C6–7 disc space, which is then fenestrated, and the ventral aspects of C6 and C7.
(c) A distractor (Veterinary Instrumentation) is then used to distract the vertebral bodies.
(d) An intervertebral washer is then pushed between the distracted vertebrae.
(e) A pilot hole is then drilled from a point just cranial to the ventral process, passing through the washer and the end plate of the caudal vertebra. In general, the tap needs only to just gain purchase in the caudal vertebra as the screw will then drive itself into the vertebra. A cortical bone screw is then placed in a positional fashion. If this fails to produce secure seating of the washer then the hole in the cranial vertebra may be enlarged to form a gliding hole so that the screw can be placed in a lagged fashion.
(f) An autogenous cancellous bone graft is harvested from the proximal humerus and packed ventral to the implants and vertebral bodies. A small hole can be drilled into the cranial part of C7 before this to encourage fusion.
(g) A postoperative radiograph of the same dog as in Fig. 24.3b and c showing vertebral distraction achieved by placement of the washer and the resulting decompression of the spine when compared to Fig. 24.3b.

Table 24.2 A comparison of reported results for various surgical techniques used in the management of cervical spondylopathy.

Technique	Reference	No. of cases*	Percentage (%) success
Ventral fenestration	Lincoln & Pettit (1985)	17	53
Dorsal laminectomy	Trotter *et al.* (1976)	18	78
Ventral slot	Read *et al.* (1983)	11	64
Ventral slot	Chambers *et al.* (1986)	27	67
Ventral slot	Breucker *et al.* (1989b)	20	78
i/v screw and cortical graft	Breucker *et al.* (1989b)	7	43
Plastic plate and cortical graft	Breucker *et al.* (1989b)	37	75
i/v screws	Denny (1989)	72	74
i/v screws + partial slot	Queen *et al.* (1998)	16	81
Harrington rods	Walker (1989)	not available	90
Screws and PMMA	Ellison *et al.* (1988)	10	80
Pins and PMMA	Breucker *et al.* (1989a)	41	90
i/v washers and screws	McKee *et al.* (1990)	20	85
i/v washers and screws	McKee *et al.* (in press)	76	88

i/v = intervertebral; PMMA = polymethylmethacrylate.

* No. of cases includes only those with adequate follow-up information.

(proven to be due to a 'domino' lesion in the 8 that had myelograms). These results would give a failure rate, as defined above, of 26/76 (34%).

Control programmes

Studies on the radiographic evaluation of 'normal' Dobermann Pinschers to allow identification of those likely to develop clinical signs might prove useful in screening dogs prior to breeding, as discussed earlier. However, it is unlikely that genotype is the sole influence on phenotype and although breeding programmes based on the findings of such studies might help reduce the incidence of the condition, they are unlikely to eliminate it.

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Chapter 25
Thoracolumbar Disc Disease

Thoracolumbar disc disease is an extremely common condition in dogs, predominantly affecting the chondrodystrophic breeds and, in particular, the Miniature Dachshund. The clinical signs associated with disc prolapse may vary from spinal pain, through paresis to paralysis and loss of conscious pain sensation (CPS). Although the signs can be very dramatic in onset and severity, the condition carries a relatively good prognosis, if managed correctly, as long as CPS is present. If this is absent then urgent treatment is required but even then the prognosis is guarded. The aetiology and pathogenesis of this disease is detailed in Chapter 5 (p. 35).

History

Thoracolumbar disc extrusion

In the main, it is the chondrodystrophic breeds that are affected, e.g. Miniature Dachshund, Jack Russell Terrier, Shih Tzu. In a survey of cases seen at Bristol University Veterinary School over a 5-year period (1985–1989), 56% were Miniature Dachshunds. Larger breeds may be seen with this condition also. It is very rare in dogs less than 1 year of age and uncommon in those over 11 years of age. The owners may report anything from the dog being reluctant to jump on furniture or to climb stairs or crying when he/she does so, to being weak or wobbly on his/her hindlimbs, or totally unable to rise at all on his/her hindlimbs.

Thoracolumbar disc protrusion

Any breed may be affected with this type of disc prolapse although it is most commonly seen in those breeds in which fibroid degeneration occurs (i.e. the larger breeds such as the German Shepherd Dog). The dog will most often be presented with a history of progressive hindlimb incoordination or reluctance to jump, due to low-grade 'back pain', although sudden deterioration in hindlimb function may occur.

Clinical signs (see also Chapter 17)

The signs caused by a disc prolapse will be related to not only the site involved and the type of prolapse but also, in the case of disc extrusions, to the amount of material extruded and the force of the impact on the spinal cord. The signs may develop suddenly or progressively worsen over a few days.

In cases suffering with disc extrusions, back pain is the commonest feature with arching of the back and a very stiff gait. Hindlimb weakness, or ataxia, may be present with the dog wobbling or knuckling on one or both hindlimbs. Alternatively, the dog may be so severely affected that he/she is unable to get up on his/her hindlimbs either due to severe weakness (paresis) or paralysis (termed paraplegia if it affects both hindlimbs). Beyond this, the signs may include urinary incontinence (take care to differentiate from 'bed-wetting' simply because he/she is unable to get off his/her bed, p. 181) and loss of CPS. The latter is a very significant sign to look for in a dog that is 'off' his or her back legs. If it is present, the dog will show *conscious pain* when pressure is applied to the toes (with fingers, artery forceps or pliers). This is *not* the same as a withdrawal reflex. If it is absent, then the dog has very severe cord damage which requires *urgent* attention (see later under 'Treatment and prognosis'). The local hindlimb reflexes

(patellar and withdrawal) are usually normal or slightly increased. The panniculus reflex often cuts out at the level of the protrusion in dogs that are 'off' their hindlimbs.

The possibility of ascending/descending myelomalacia must not be forgotten. Clinically, these dogs are very dull and obviously ill. Paralysis (often flaccid) will exist caudal to the lesion (with loss of lower motor neuron (LMN) function) and incontinence will be present. The level at which sensation is lost will be cranial to the level of disc prolapse. The Schiff–Sherrington phenomenon may be seen if cranial thoracic segments are involved. If the majority of the segments supplying the intercostal muscles are involved then paradoxical respiration may be seen. Opisthotonos is often a feature. If the malacia progresses cranially, it will eventually affect the phrenic outflow (C5 and C6) and respiratory arrest will occur. Some develop intermittent crying which does not appear to be associated with pain. These signs seem to develop within a few days of the start of the problem, are only seen in dogs that are quite severely affected neurologically and are not influenced by the choice of treatment (conservative or surgical). This myelomalacia, fortunately, is uncommon but is important since it makes the prognosis hopeless. Early evidence of this pathology may be detected on sequential neurological examinations. Significant changes would include loss of LMN reflexes in the hindlimbs and progressive cranial movement of the panniculus 'cut-off'.

In general, the clinical signs are graded according to the severity of neurological dysfunction as below:

- Grade 1: pain only
- Grade 2: hindlimb paresis/ataxia with or without pain
- Grade 3: an inability to stand unaided, voluntary movement still present
- Grade 4: an inability to stand unaided, voluntary movement absent (paraplegia)
- Grade 5: paraplegia + urinary retention with overflow (URO)
- Grade 6: paraplegia + URO + loss of conscious pain sensation (CPS)
- Grade 7: ascending/descending myelomalacia

The signs seen in the previously mentioned 5-year study are detailed in Table 25.1.

The clinical signs are *not always* bilaterally symmetrical, especially early on in the course of the disease. Such lateralisation is extremely important to note since it may aid decision making later if decompressive surgery is being contemplated. The signs may, at a later stage, become symmetrical and the myelogram may not demonstrate lateralisation. Although asymmetry has been reported in up to 80% of cases, the proportion of those cases showing asymmetry in which the disc extrusion is lateralised to the side of the worst neurological signs varies between reports. For example, the proportion was found to be only about 65% by Yovich *et al.* (1994) but about 80% by Schulz *et al.* (1998). However, if myelography (which is generally accepted as being more reliable than clinical signs) fails to show the direction of extrusion, and a hemilaminectomy is planned, then asymmetric clinical signs can be used as a guide to decide on which side to operate, though this is not fool-proof.

Table 25.1 Clinical presentation of 111 dogs with thoracolumbar disc extrusions seen at Bristol University Veterinary School between 1985 and 1989.

Clinical sign	No.
Pain only	14
Pain and hindlimb ataxia	53
Unable to stand (with or without voluntary movement)	29
Paraplegia + urinary retention and overflow (URO) (incontinence)	9
Paraplegia + URO + loss of conscious pain sensation (CPS)	6
Total	111

In cases with disc protrusions, which are far less common, pain is not such a prevalent feature and progressive hindlimb ataxia is the most frequently encountered clinical sign.

Differential diagnosis

Causes of thoracolumbar pain and/or neurological deficits in the hindlimbs include:

- Thoracolumbar disc extrusion/protrusion
- Fracture/luxation
- Discospondylitis
- Neoplasia
- Ischaemic myelopathy (fibrocartilagenous embolism)
- Chronic degenerative radiculomyelopathy (larger breeds)

Diagnosis

A diagnosis can usually be reached with good-quality plain radiographs but more specific information regarding the exact site and side on which the prolapse has occurred and the degree of compression present will require myelography. Although general anaesthesia makes patient positioning easier, diagnostic films often can be obtained with the dog sedated. General anaesthesia is mandatory for myelography. Lateral views are the most useful but ventrodorsal views may be beneficial and should be carried out if myelography is to be performed, to allow evaluation of the significance of any abnormalities noted.

Radiographic changes may include (Fig. 25.1):

- Calcification within the intervertebral disc spaces
- Narrowing of the intervertebral disc spaces
- Reduced size of the intervertebral foramen
- Narrowing of the space between the articular facets
- Evidence of a dorsal protrusion
  - a dorsal tail
  - opacification of the intervertebral foramen

In recent reports (Olby *et al.*, 1994; Yovich *et al.*, 1994; Schulz *et al.*, 1998) plain radiography allowed the extruded disc to be identified correctly in between 57 and 80% of cases. Accurate determination of the direction of extrusion may only be possible in about 10% of cases and in a similar proportion the site of disc extrusion may be positively identified incorrectly.

(a)

(b)

Fig. 25.1 (a) Lateral radiograph of the thoracolumbar spine of a 6-year-old Miniature Dachshund. Mineralisation within the T13–L1 disc space is indicative of degenerative disc disease but a degree of disc space narrowing and opacification of the intervertebral foramen at L1–2 is suggestive of mineralisation and extrusion of that disc.
(b) Lateral radiograph of the lumbar spine of a 5-year-old Jack Russell Terrier. There is subtle mineralisation but no obvious narrowing of the L2–3 disc space. However, the intervertebral foramen is smaller and more opaque than its neighbours suggesting a disc extrusion at this site.

Myelography may help to further identify a disc prolapse. It may be necessary in order to reach a diagnosis if the plain films are normal (not all disc problems are evident on standard radiographs) or if it is not clear at exactly which disc space, or on which side, the extruded material is present within the vertebral canal (the significance of this information may depend on the treatment being planned). In the case of thoracolumbar discs, if the contrast is introduced cisternally, it often stops in the mid-thoracic region. It is more useful to inject the contrast under pressure via a lumbar puncture whereby it will be forced past the area of interest and be more informative.

Myelographic changes may include (Fig. 25.2):

- Dorsal deviation of the ventral column in a lateral view
- Narrowing/loss of the dorsal column
- Lateral deviation of the columns in a ventrodorsal view
- Intramedullary swelling

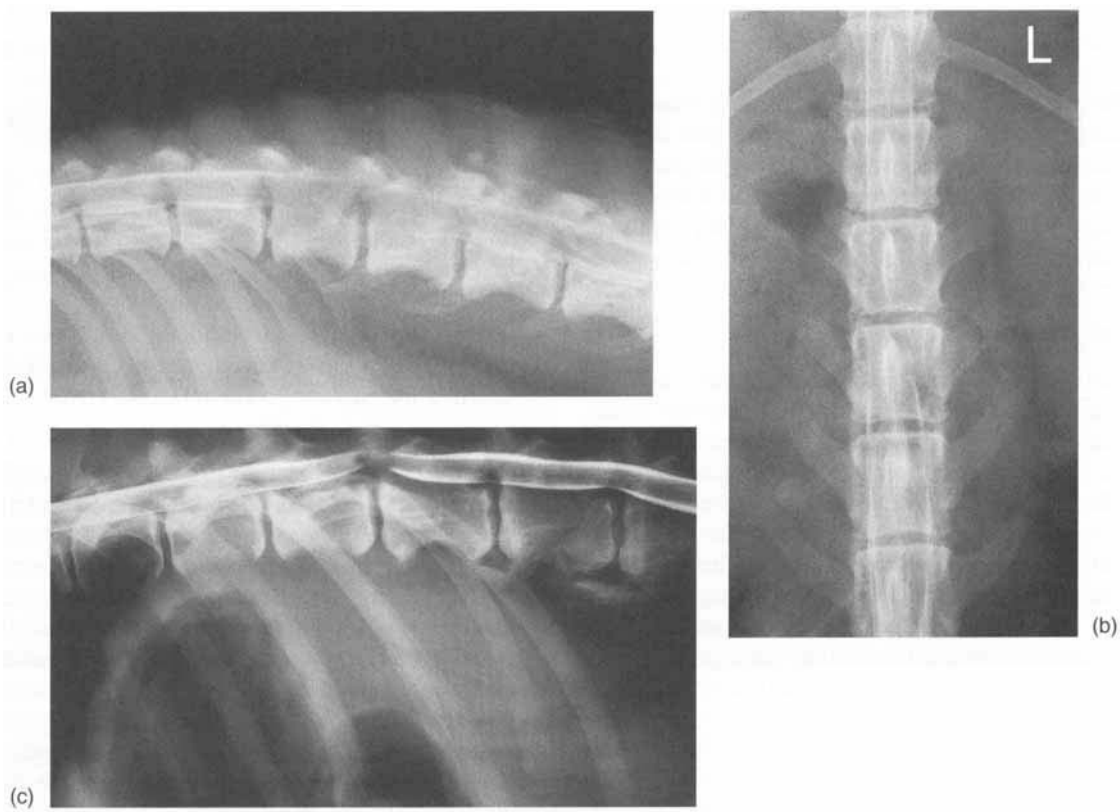


Fig. 25.2 (a) Lateral radiograph of the same dog as in Fig. 25.1b during lumbar myelography. The contrast columns between cranial L2 and mid L3 are poor suggesting that the L2–3 disc has extruded.
(b) Ventrodorsal radiograph of the same dog as in (a) showing deviation of the left contrast column to the right indicating that the L2–3 disc extrusion is predominantly to the left.
(c) Lateral radiograph of the thoracolumbar spine of a 10-year-old Old English Sheepdog taken during lumbar myelography. Ventral compression of the spinal cord is present over the T13–L1 disc space but there is no loss of the contrast columns suggesting that spinal cord swelling is minimal. Although this appearance makes interpretation relatively straightforward it is well to be aware that it may be associated with disc protrusions (type II prolapses) rather than extrusions (type I prolapses), especially when other chronic changes are present such as spondylosis. The significance of this difference becomes apparent when treatment and prognosis are considered.

Unfortunately not all disc protrusions will be evident in myelograms and, in many cases, there may be a loss of contrast over several disc spaces which can make it difficult to determine which disc, and which side, is affected. In recent reports (Olby *et al.*, 1994; Yovich *et al.*, 1994; Schulz *et al.*, 1998) myelography allowed accurate identification of the site of disc extrusion in 85–95% of cases and the direction of extrusion could be determined correctly in up to 82% of the dogs, although this proportion is reduced to about 35–45% if only ventrodorsal, and not oblique, views are used.

Treatment and prognosis

The methods of treatment fall, broadly, into three categories: conservative, fenestration and decompression (laminectomy) and the prognosis is determined by the degree of spinal cord injury, i.e. the severity of the neurological signs, together with the method of management chosen.

Conservative management

This comprises strict confinement or 'cage rest' for 4–6 weeks whilst the annulus fibrosus heals and the inflammatory response associated with the disc prolapse resolves. Non-steroidal anti-inflammatory drugs (NSAIDs) may be used to allow comfortable rest. If the dog is showing moderate to severe neurological signs then corticosteroids may be useful in the initial stages though this is controversial. They are, however, best avoided for simply controlling pain as they tend to slow the healing of the annulus fibrosus. The use of methylprednisolone sodium succinate is discussed elsewhere (see Chapter 21, p. 207).

There are few published reports of results from treating thoracolumbar disc cases conservatively and the numbers involved make it impossible to provide accurate figures. From these studies and their own experience, the authors would consider the following as a fair overview. The majority of cases showing pain with or without hindlimb ataxia will make a satisfactory improvement (probably about 80–85% of cases). Those that are non-ambulatory have a slightly lower chance of recovery unless they also show urinary incontinence, in which case about 60% will recover hindlimb function, or loss of CPS, in which case between 5 and 10% will regain reasonable hindlimb function. The recurrence rate of clinical signs following conservative management over a 2-year period is in the order of 30%.

Fenestration

Several approaches have been described but the the most commonly reported technique involves the lateral approach, as described by Flo & Brinker (1975) (Fig. 25.3). The principle of the technique is to create a window in the annulus allowing removal of any remaining nucleus pulposus from the disc (Fig. 25.4). The aims in doing so are to:

- Reduce the degree of pain by 'decompression' of the disc itself
- Eliminate the 'dynamic effect' described by Olsson (1958) of nucleus pulposus material being gradually forced dorsally resulting in the persistence of clinical signs, or their worsening, over a prolonged period of time
- Prevent recurrence by removing any remaining nucleus pulposus from not only the disc causing the current problem but also from the other 'high-risk' discs

The evidence for the first two of these aims is relatively anecdotal. One, or both, of these might serve to explain the fact that cases showing only pain that fail to respond to conservative measures often improve rapidly after fenestration. Since the procedure does not allow removal of the extruded material from the vertebral canal, it is reasonable to assume that the discomfort seen in some cases is due to either 'discogenic pain' or continued extrusion of disc material creating the 'dynamic effect' and that fenestration may effectively reduce the pain in these cases.

The evidence for the third aim is based on the recurrence rate noted after either conservative treatment or fenestration. Davies & Sharp (1983) reported on two groups of dogs with thoracolumbar disc disease that had been treated either conservatively or by fenestration of the last three thoracic and first three lumbar disc spaces. The follow-up period for the two groups had mean values of 23.7 and 28.6 months, respectively.

Recurrence of clinical signs was seen in 34% of those treated conservatively and 0% of those treated by fenestration. The low recurrence rate following this procedure has also been reported, more recently, in 2% of cases (Butterworth & Denny, 1991).

With respect to which discs ought to be fenestrated simultaneously, there is some variation between authors. Flo & Brinker (1975) suggested that up to eight could be fenestrated if there was radiographic evidence of degeneration, whilst Denny (1982) recommended fenestration of the last three thoracic and first three lumbar discs. On observing the relative incidence of disc extrusions in the thoracolumbar region (Table 25.2) it is apparent that in 89% of cases it is one of the last three thoracic or first three lumbar disc spaces that are involved. Owing to the relative increase in surgical risk of extending the fenestration cranially (proximity to the pleural cavity) or caudally (proximity to nerve roots of the lumbosacral plexus), it is often recommended that these six spaces be fenestrated routinely, although the procedure can be extended one space either way if radiographic evidence of disc degeneration is present.

The results, in relation to the grade of clinical signs shown by the patient, of this procedure have been reported on several occasions. Olsson (1951) reported an overall success rate of 85% in ambulatory and 80% in non-ambulatory patients compared to rates of 50% and 45%, respectively, in cases treated conservatively. A similar comparison between these two methods of management can be drawn from the series reported by Davies & Sharp (1983). If the dogs with no CPS are taken out of the conservative group, since no such cases were treated by fenestration making comparison of the overall success rates inappropriate, then the success rates for fenestration would be 100% in ambulatory and 81% in non-ambulatory dogs compared to 87% and 81%, respectively, in cases treated conservatively.

In reviewing the outcome in these cases the definition of a success has been 'considered an acceptable pet' or else 'a complete return to normal or only mild residual deficits that do not impair the dog from leading a normal life'. Using these definitions the results of thoracolumbar fenestration from several studies can be collated and are detailed in Table 25.3, in which the number of cases and success rates are recorded.

In cases showing only pain and/or ataxia, approximately 95% will recover within 4–6 weeks. Of the dogs that are non-ambulatory but have CPS, 80–90% will recover within 4–8 weeks. The number of cases reported that had no CPS are so few that giving results in terms of a percentage is very misleading but probably lies somewhere in the region of about 30%. The recurrence rate of clinical signs following fenestration is in the order of 1–2% of cases. From these results certain conclusions may be drawn as to the advantages of fenestration in relation to conservative management and by comparison with the results of decompressive surgery (see later) the relative merits of fenestration may be assessed.

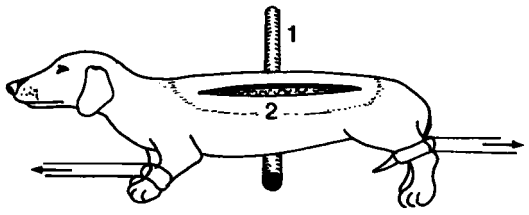
Advantages of fenestration:

- Less specialised instrumentation is required than for decompressive surgery
- Myelography may be unnecessary since the

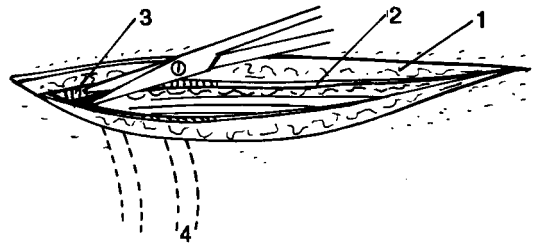
Table 25.2 Relative incidence of disc extrusions in the thoracolumbar spine.

Reference	No. of cases	Percentage (%) incidence at each disc space									
		T10–11	T11–12	T12–13	T13–L1	L1–2	L2–3	L3–4	L4–5	L5–6	L6–7
Knecht (1972)	184	2	11	17	23	16	11	9	6	4	1
Gage (1975)	654	1	8	31	22	14	9	6	6	2	1
McKee (1992)	60	2	8	33	25	17	5	5	5	–	–
Scott (1997)	50	6	4	32	20	8	4	4	2	–	–
Total/average	948	2	9	27	23	15	8	7	6	2	1

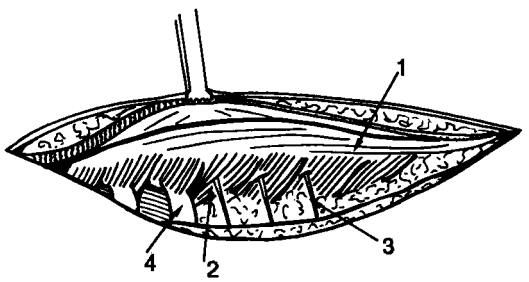
Disc extrusions at T11–12 to L3–4 inclusive represent 89% of the total in this region.



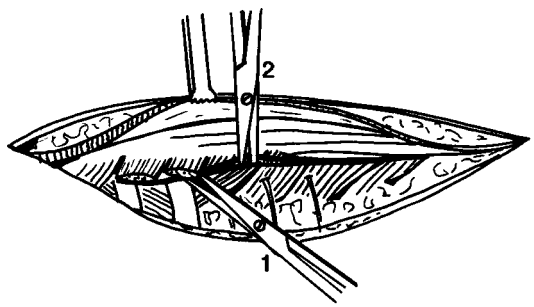
(a) The dog is placed on its right side with the thoracolumbar spine arched over a wooden pole (1). A lateral incision is made (2) from the ninth rib to the fifth lumbar vertebra.



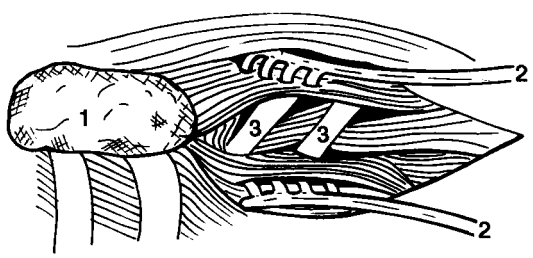
(b) The subcutaneous fat (1), lumbodorsal fascia (2), and the latissimus dorsi muscle (3) are incised. (4) The thirteenth rib.



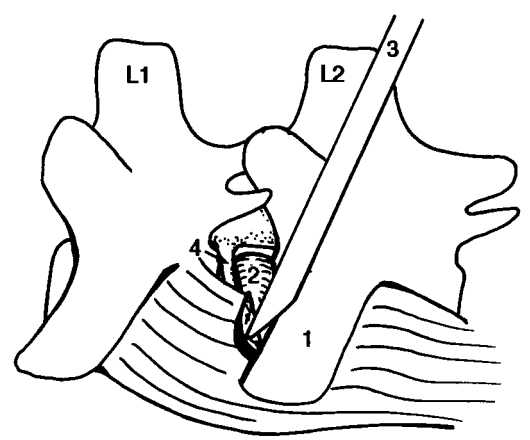
(c) The lumbodorsal fascia and the latissimus dorsi muscle are retracted to reveal the longissimus dorsi (1) and the iliocostalis lumborum muscle (2). The dorsal branches of the lumbar nerves (3) and the last two ribs (4) can also be seen.



(d) The iliocostalis lumborum muscle is detached and elevated from the last three ribs (1). The iliocostalis lumborum is then split longitudinally over the first four lumbar transverse processes (2).

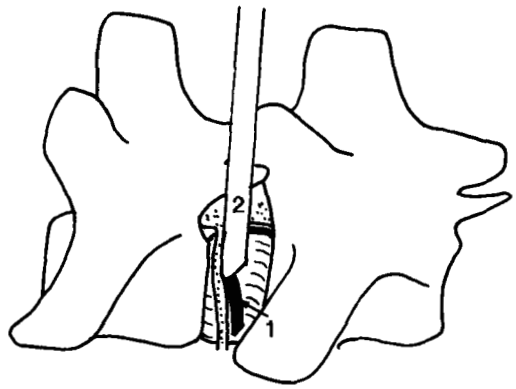


(e) Haemorrhage from the severed costal attachments of the iliocostalis lumborum muscle is controlled by packing the area with a swab (1). Self-retaining retractors (2) are used to retract the iliocostalis lumborum muscle in the lumbar region while the transverse processes (3) are being exposed.

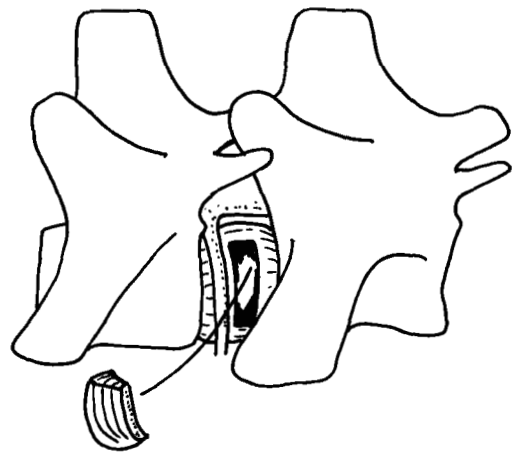


(f) The transverse process (1) of the second lumbar vertebra is cleared of muscle. The first lumbar vertebra disc (2) lies immediately cranial to the tranverse process. Fascia covering the lateral aspect of the disc is cleared in a cranial direction using a perisoteal elevator (3) initially. The ventral branch of the first lumbar nerve (4) crosses the cranial edge of the disc.

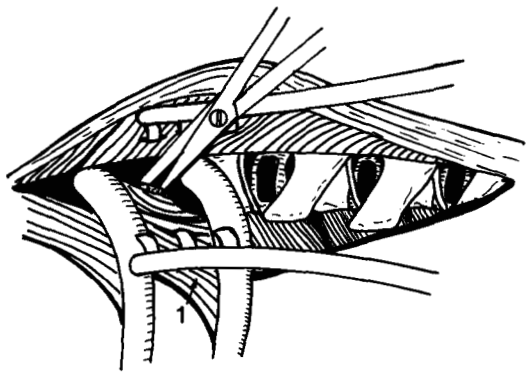
Fig. 25.3



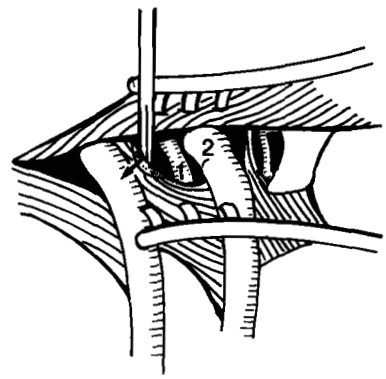
(g) A vein (1) which runs with the spinal nerve is sometimes accidentally ruptured. Haemorrhage can be controlled by applying pressure with an elevator (2) on the cranioventral corner of the disc.



(h) A window is cut through the lateral side of the annulus fibrosus and the nucleus pulposus is removed with a dental tartar scraper.



(i) Exposure of the twelfth thoracic disc. The origin of the levatores costorum muscle (1) is partially severed.



(j) Exposure of the twelfth thoracic disc. The levatores costorum muscle is retracted in a ventral direction to reveal the disc (1) lying just cranial to the head of the rib (2).

Fig. 25.3 *Contd.*

specific disc space involved does not have to be identified

- Once the 'dynamic effect' has been removed nursing of the patient becomes easier since absolute rest is not so important and there is less concern over moving him/her
- A high success rate can be expected in cases with pain +/– mild neurological signs
- Treatment of all the 'high-risk' discs simultaneously dramatically reduces the likelihood of recurrence

Laminectomy (decompression)

There are several methods of achieving spinal decompression. The principle involves removing parts of the vertebrae adjacent to the disc space so as to gain entry to the vertebral canal and remove the extruded disc material. The disc is usually fenestrated at the same time so as to prevent further extrusion. Some surgeons also fenestrate the other 'high-risk' discs during the same procedure whilst others do not.

Table 25.3 Results of lateral fenestration in the management of thoracolumbar disc extrusions.

Neurological grade*	Study						Overall	
	Denny (1978)		Davies & Sharp (1983)		Butterworth & Denny (1991)			
	No.	Recovered	No.	Recovered	No.	Recovered	No.	Recovered
		No. (%)		No. (%)		No. (%)		No. (%)
1	2	2 (100)	5	5 (100)	12	11 (92)	19	18 (95)
2	4	4 (100)	13	13 (100)	43	40 (93)	60	57 (95)
3	15**	15 (100)	11	8 (73)	26	22 (85)	37	30 (81)
4/5			5	5 (100)	8	7 (88)	13	12 (92)
6	9	5 (56)	–	–	6	2 (33)	15	7 (47)

* Neurological grades: 1 – pain only; 2 – ambulatory paresis; 3 – non-ambulatory paresis; 4 – paraplegia; 5 – paraplegia + urinary retention with overflow (URO); 6 – loss of conscious pain sensation (CPS).

** In the study by Denny (1978) it could not be determined how many dogs fitted into grades 3 and 4 (as defined here) and so these have not been used in calculating the overall averages.

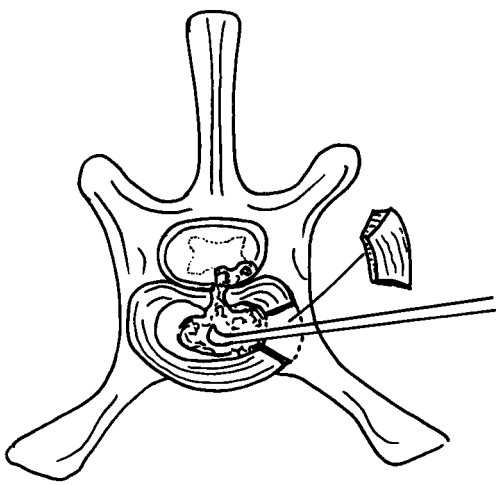


Fig. 25.4 Transverse section illustrating lateral disc fenestration. Note that it is not possible to remove disc material which has been extruded into the neural canal. A hemilaminectomy is needed to do this.

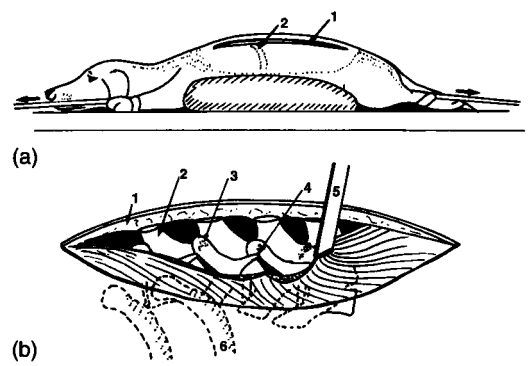


Fig. 25.5 Dorsolateral approach for hemilaminectomy. (a) The skin incision is marked by point 1 and the thirteenth rib by point 2. (b) Detail of the dorsolateral approach for hemilaminectomy showing lateral reflection of muscle attachments from the dorsal spinous processes and the articular facets. 1 – Lumbodorsal fascia; 2 – spinous processes; 3 – articular facets; 4 – accessory process; 5 – periosteal elevator; 6 – thirteenth rib.

Most commonly a dorsolateral approach is used based on that described by Yturraspe & Lumb (1973) (Fig. 25.5). The technique used to decompress thoracolumbar discs most often is a *hemilaminectomy* which involves removal of the articular facets and the surrounding pedicle bone (Fig. 25.6). Alternatively, the facets may be spared and the pedicle of bone ventral to them removed. The latter technique is often referred to as a *lateral laminectomy* or *mini-hemilaminectomy* and was first described by Braund *et al.* (1976) (Fig. 25.7). It is possible to use a lateral approach (Fig. 25.3) to create a mini-hemilaminectomy by elevating the epaxial muscles to expose the lateral wall of the vertebral canal (Fig. 25.8).

Dorsal laminectomy is used far less often than

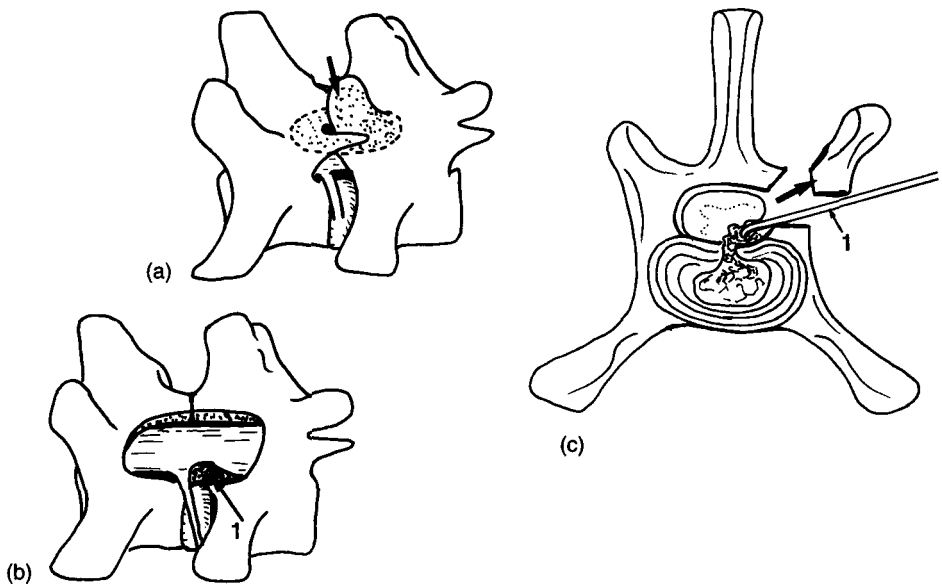


Fig. 25.6 Hemilaminectomy. (a) Articular facets are removed with rongeurs. (b) Bone is removed with a high-speed burr down to the inner cortical bone plate. This is then thinned to the thickness of an eggshell to allow removal with a curette or small rongeur. The laminectomy is extended ventrally with rongeurs to expose the spinal nerve and mass of extruded material (1) beneath the spinal cord. (c) Transverse section illustrating hemilaminectomy and removal of disc material from the floor of the neural canal (1).

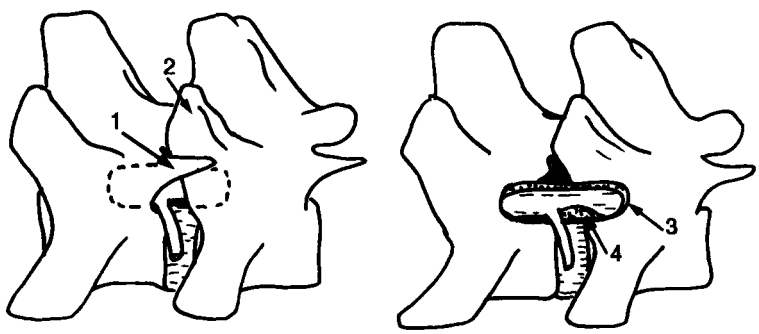


Fig. 25.7 Mini-hemilaminectomy (after Jeffery, 1988). 1 – Accessory process; 2 – articular facets; 3 – margins of laminectomy; 4 – extruded disc material.

in the past since there may be a requirement to manipulate the spinal cord in order to remove any ventrally positioned disc material. This is, perhaps, reflected by a higher incidence of immediate postoperative neurological worsening when compared to hemilaminectomy (Muir *et al.*, 1995), though the overall long-term results for the two techniques were not significantly different in this study. In addition, there may be problems with laminectomy membrane formation which may be more likely after dorsal laminectomy compared with hemilaminectomy. The latter is where fibrous tissue forms over the exposed spinal cord between the margins of the laminectomy. As this tissue contracts it may cause compression of the spinal cord with a return of clinical signs. There are several variations on the theme of dorsal laminectomy with respect to how much bone is removed (as reviewed by Hoerlein, 1978). In order to reduce the likelihood of problems due

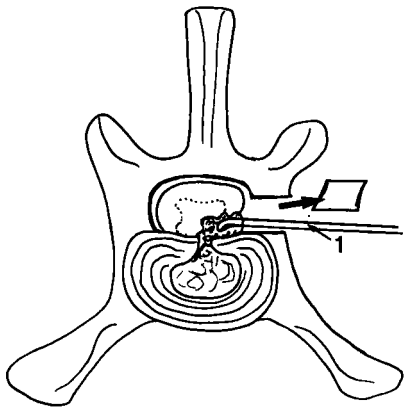


Fig. 25.8 Mini-hemilaminectomy. Transverse section showing removal of disc material with a curette (1).

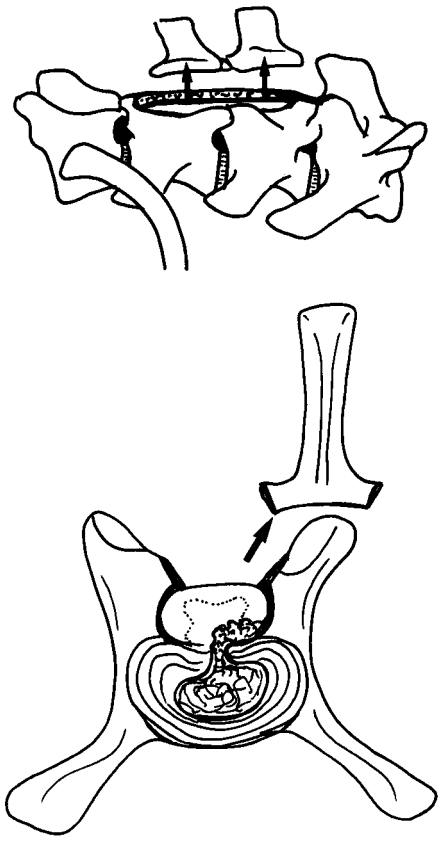


Fig. 25.9 Dorsal laminectomy. This procedure involves removal of the dorsal spines and laminae whilst leaving the articular facets in place. Although the procedure allows wide exposure of the spinal cord, access to disc material lying ventrally is limited (see transverse section in Fig. 25.8).

to laminectomy membrane formation, the articular facets are generally left in place as shown in Fig. 25.9 (i.e. a Funkquist type B laminectomy as described by Funkquist, 1962). Although still used by some surgeons to treat thoracolumbar disc extrusions at any level, many use it as the technique of choice only when the most caudal lumbar discs are involved (i.e. L6–7 and L7–S1). The approach used for this technique requires a dorsal midline incision as described by Redding (1951) and illustrated by Piermattei (1993).

There are several studies looking at the results of laminectomy but comparisons are made extremely difficult by differences that include: system of grading the clinical signs; duration of clinical signs; type and extent of surgery carried out; and definition of recovery time (time to walk versus time to reach best). The authors have tried to draw the results of several papers together to provide some idea of how successful this type of management is. Tables 25.4 and 25.5 provide the summary of these results for hemilaminectomy (including lateral laminectomy) and dorsal laminectomy, respectively.

Few cases with only pain or ambulatory paresis have been included in these studies but in those reported a success rate of 90–100% is expected within 1–2 weeks. Of the dogs showing non-ambulatory paresis a similarly high recovery rate of about 95% is expected within 2–3 weeks. When paraplegia is present the success rate may fall to about 90% and the time taken to recover be more in the order of 1 month. If CPS has been lost then only about 50–60% are likely to regain an ability to walk in an average time of about 6 weeks. In these cases it is generally considered that decompression is only helpful if performed within about 36–48 hours (preferably within 24 hours) of CPS being lost and even then only about 50% will recover reasonable use of their hindlimbs. One report (Schulman & Lippincott, 1987) recommends that dogs showing a loss of CPS should only be considered surgical candidates within 12 hours of such loss. In contrast, Anderson *et al.* (1991) consider the duration of signs to be less

Table 25.4 Results of hemilaminectomy and lateral laminectomy (*) in the management of thoracolumbar disc extrusions.

Neurological grade**	Study													
	Knecht (1972)		Schulman & Lippincott (1987)		Black (1988)*		Anderson *et al.* (1991)		McKee (1992)		Scott (1997)		Overall	
	No.	Recovered	No.	Recovered	No.	Recovered	No.	Recovered	No.	Recovered	No.	Recovered	No.	Recovered
		No. (%)		No. (%)		No. (%)		No. (%)		No. (%)		No. (%)		No. (%)
1	–	–	–	–	3	3 (100)	–	–	–	–	2	2 (100)	5	5 (100)
2	–	–	15	14 (93)	6	6 (100)	–	–	–	–	7	6 (86)	28	26 (93)
3	37	36 (97)	43	42 (98)	14	14 (100)	–	–	22	22 (100)	13	11 (85)	129	125 (97)
4/5	42	37 (88)	33	26 (79)	12	12 (100)	–	–	5	5 (100)	15	14 (93)	107	94 (88)
6	16	4 (25)	–	–	1	1 (100)	30	23 (77)	2	0 (0)	3	2 (67)	52	30 (58)

** Neurological grades: 1 – pain only; 2 – ambulatory paresis; 3 – non-ambulatory paresis; 4 – paraplegia; 5 – paraplegia + urinary retention with overflow (URO); 6 – loss of conscious pain sensation (CPS).

Table 25.5 Results of dorsal laminectomy in the management of thoracolumbar disc extrusions.

Neurological grade*	Study									
	Funkquist (1970)		Henry (1975)		Gambardella (1980)		McKee (1992)		Overall	
	No.	Recovered	No.	Recovered	No.	Recovered	No.	Recovered	No.	Recovered
		No. (%)		No. (%)		No. (%)		No. (%)		No. (%)
1	–	–	–	–	–	–	–	–	–	–
2	–	–	–	–	–	–	–	–	–	–
3	89	62 (70)	12	10 (83)	18	16 (89)	19	15 (79)	138	103 (75)
4/5	42	17 (40)	10	6 (60)	58	52 (90)	10	7 (70)	120	82 (68)
6	15	10 (67)	14	1 (7)	22	11 (50)	–	–	51	22 (43)

* Neurological grades: 1 – pain only; 2 – ambulatory paresis; 3 – non-ambulatory paresis; 4 – paraplegia; 5 – paraplegia + urinary retention with overflow (URO); 6 – loss of conscious pain sensation (CPS).

critical. In order to try and improve the success rate in the management of these cases, some surgeons are investigating the use of omental transplants in order to provide a more rapidly established blood supply and oxygen free radical scavenging system to the damaged area of spinal cord. Although this concept is of great interest, there are, as yet, no studies to show that it does improve the prognosis for these conscious pain-negative dogs.

The results appear to favour hemilaminectomy over dorsal laminectomy in the thoracolumbar region, and although a study by Muir *et al.* (1995) showed little difference in the long-term outcome, there appeared to be a lower incidence of post-operative deterioration with hemilaminectomy. The recurrence rate of clinical signs following laminectomy and fenestration of only the disc involved varies between reports but more recently has been cited as 13% (Scott, 1997).

Advantages of laminectomy:

- Allows a more definitive diagnosis to be reached
- Allows inspection of the spinal cord (may be most useful where malacia is suspected)

- By decompressing the spinal cord it leads to a higher rate of recovery and a more rapid rate of return to function
- It may be combined with fenestration of other 'high-risk' discs to reduce the likelihood of recurrence

Acupuncture

This treatment has been widely reported (Jansens & Rogers, 1989), though the authors have no experience of its use. The overall success rates are in line with other forms of treatment (75–95%) where CPS has not been lost and is similar to that following fenestration (20–30%) where CPS is absent. The rate of recurrence of clinical signs following recovery is in the order of 20–30% of cases. Overall, the results in ambulatory dogs are similar to those seen with conservative management or fenestration but the recurrence rate is higher than with the latter. In non-ambulatory dogs with CPS the results are less good than with laminectomy but approach those with fenestration. In those without CPS the results are less good than following laminectomy. There is no doubt that acupuncture can be used in cases with this disease but, although the results may be better than with standard conservative management, it does not confer the advantages of the two aforementioned types of surgery in terms of reducing the rate of recurrence of clinical signs or decompressing the spinal cord.

Guidelines to the management of thoracolumbar disc extrusions

From the above discussion conclusions need to be drawn regarding how best to manage such a case. The authors are not experienced in the use of acupuncture and so the discussion will be restricted to conservative management and surgery. Unfortunately, general conclusions are difficult to make as each case will be slightly different. Factors influencing the choice of management include:

- *Breed*
  - chondrodystophic breeds are more likely to have recurrence at a second site
  - larger breeds are less likely to have recurrence at a second site and fenestration is, practically, more difficult
- *Age*
  - young dogs are at higher risk of recurrence than older dogs
- *Severity*
  - the more severe the signs the more advantageous is surgery over conservative management and is laminectomy over fenestration
- *Radiographic findings*
  - evidence of multiple disc disease may suggest a higher likelihood of recurrence
  - ability to identify which disc has extruded is required for surgery (especially laminectomy)
  - degree of compression and extent of spinal cord swelling may influence choice of management (e.g. laminectomy versus fenestration)
- *Finance*
  - the costs involved with surgical treatment may not be affordable

Indications for conservative management

Conservative management may be considered as an option for all cases except, perhaps, those without CPS where the success rate is very low and possibly too poor to justify prolonging the situation for 4–6 weeks whilst it is established whether hindlimb function returns. However, the results are not as good as surgery at any level of neurological grade and so there is often an argument for recommending surgery over conservative management, except where the owner's financial constraints preclude this.

Indications for fenestration

This is an appropriate treatment in cases with thoracolumbar disc extrusions showing pain and/or mild neurological signs in that it produces a high success rate and reduces the likelihood of recurrence of clinical signs. In the presence of more severe signs, fenestration will still produce good results in the vast majority of dogs unless there is loss of CPS. When decompressive surgery is performed in dogs with multiple disc degeneration, it is advisable to concurrently fenestrate as many disc spaces as is convenient in order to prevent

recurrence of signs (McKee, 1992). Such fenestration can be achieved via the dorsolateral approach usually used for hemilaminectomy (Fig. 25.5) or else the lateral approach used for fenestration (Fig. 25.3) can be developed to expose the lateral aspect of the vertebrae, by elevation of the epaxial muscles, allowing mini-hemilaminectomy.

Indications for laminectomy

This may be considered appropriate for any case with a thoracolumbar disc extrusion, particularly when:

- The patient shows marked ataxia or paraplegia since it optimises their likelihood of recovery
- In larger breeds where a reduction in the time taken to become ambulatory post-surgery is, perhaps, of greater importance with respect to practical nursing care than with smaller breeds
- When CPS is absent, as it allows inspection of the spinal cord with a view to detecting malacia (although the ability to distinguish visually between a viable and non-viable spinal cord is questionable)

A summary of these conclusions is given in Table 25.6.

Treatment of disc protrusions (i.e. type II discs)

Treatment of this type of disc prolapse can, to say the least, prove problematic. If treated conservatively the protrusion may well progress, causing further cord damage and worsening clinical signs. However, the cord compression in these cases has been very insidious in onset and little, if any, of the dysfunction is due to contusion. As a result there is no 'reserve' of cord tissue with which the dog might compensate during recovery. Consequently, many of these cases will be made worse, at least initially, by surgical interference.

Fenestration may stop the potential for further protrusion. However, removal of the nucleus pulposus may cause collapse of the disc space with increased bulging of the dorsal annulus fibrosus into the vertebral canal and worsening of neurological signs. Hemilaminectomy will expose the protruding material but this is not loose and so cannot be removed easily. Attempts to remove the dorsal annulus from such an approach can easily lead to iatrogenic cord damage. A bilateral mini-hemilaminectomy may be performed so as to allow a scalpel to be passed along the floor of the vertebral canal from each side in the hope that this will allow withdrawal of the protruding annulus fibrosus. Although this may enable more complete removal of the protruding annulus it still carries a high risk of iatrogenic cord damage. The authors have found that removal of the protruding annulus via laminectomy is generally associated with a worsening of neurological status which may persist for weeks or months. Dorsal laminectomy has been suggested so as to remove pressure from the cord without attempting to remove the protruding material but the protrusion may continue and, with the concurrent formation of a laminectomy scar, the compression will recur.

As a result of these surgical complications, the possibility of injecting a proteolytic enzyme such as chymotrypsin has been suggested. However, it is imperative that no leakage of such material from the disc into the vertebral canal occurs and there is also concern over whether collapse of the disc space after such treatment could exacerbate the signs in the same way as suggested following fenestration.

It may be possible to consider treating these types of disc prolapse using a distraction/fusion technique, in a similar way to some forms of cervical spondylopathy and lumbosacral stenosis. However, proving that a thoracolumbar disc protrusion is 'dynamic' or 'traction-responsive' is problematic. One method of achieving some distraction, stability and fusion would be to plate the two vertebral bodies using a dynamic compression plate 'in reverse'. That is by positioning the drill holes for the screws at the end of the plate holes nearest the centre of the plate, tightening of the screws should then displace the vertebral bodies away from one another thus creating some traction. Radical fenestration of the disc space, forage of the end plates +/– placement of a bone graft into the disc space, should encourage vertebral fusion. Although this technique has been used with success in a few cases (M.G. Ness, pers. comm.), so far insufficient numbers are involved with adequate follow-up information to know whether this form of surgery can be advocated in some or all of these cases.

Table 25.6 Guidelines in the management of thoracolumbar disc extrusions.

Grade	Signs	Options	Success rate (%)	Indications
1	Pain	Conservative	85	Financial considerations First episode (controversial)
		Fenestration	95	Chondrodystrophic breed (recurrence more likely) and disc involved lies between T10–11 and L4–5
		Laminectomy	95–100	Non-chondrodystrophic breed (recurrence less likely) or disc involved is at or caudal to L4–5 in chondrodystrophic
2	Ambulatory paresis	Conservative	85	Financial considerations First episode (controversial)
		Fenestration	95	Chondrodystrophic breed (recurrence more likely) and disc involved lies between T10–11 and L4–5
		Laminectomy	95–100	Non-chondrodystrophic breed (recurrence less likely) or disc involved is at or caudal to L4–5 in chondrodystrophic
3	Non-ambulatory paresis	Conservative	80	Financial considerations
		Fenestration	80–90	Chondrodystrophic breed (recurrence more likely) and disc involved lies between T10–11 and L4–5 May be combined with laminectomy at the site of *extrusion*
		Laminectomy	95–100	Any breed but in chondrodystrophic dogs should consider combining this with fenestration of other 'high-risk' discs
4/5	Paraplegia +/– urinary retention and overflow (URO)	Conservative	70–80	Financial considerations
		Fenestration	80–90	Chondrodystrophic breed (recurrence more likely) and disc involved lies between T10–11 and L4–5 May be combined with laminectomy at the site of extrusion
		Laminectomy	90–95	Any breed but in chondrodystrophic dogs should consider combining this with fenestration of other 'high-risk' discs
6	No conscious pain sensation (CPS)	Conservative	5–10	Financial considerations (do the results justify welfare aspects?)
		Fenestration	30	Better than conservative management, especially in chondrodystrophic breeds (reduces chance of recurrence)
		Laminectomy	50	Any breed as allows inspection of the spinal cord Should be carried out within 24–48 hours (some controversy)
7	Malacia	Euthanasia	Not applicable	All cases where this identified as prognosis is hopeless

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Chapter 26
Lumbosacral Disease

Aetiopathogenesis

The term lumbosacral disease is often confused with that of cauda equina syndrome and the two are frequently used synonymously. In order to clarify the terminology it is necessary to consider what comprises the cauda equina, what disease processes contribute to cauda equina syndrome and how these relate to the lumbosacral junction specifically.

During canine embryonic development the vertebral column grows more rapidly, longitudinally, than the spinal cord. This results in the conus medullaris (termination of the spinal cord) lying within the vertebral canal of the sixth lumbar vertebra. The spinal cord segments become 'displaced' cranially in relation to their respective vertebrae such that the sacral segments usually lie within the fifth lumbar vertebra (most easily remembered as an 'S' looking not unlike a '5'), see Fig. 26.1. The spinal nerves emanate from their respective cord segments and pass caudally through the vertebral canal until reaching the relevant intervertebral foramen, i.e. the sixth lumbar nerve arises from the spinal cord within the fourth lumbar vertebra and passes caudally within the vertebral canal to the L6–7 intervertebral foramen. As a result, on moving caudally from the third lumbar vertebra, the vertebral canal contains a spinal cord of diminishing diameter and nerve roots of increasing number. The gross appearance of these nerve roots within the vertebral canal has led to the term 'cauda equina' (derived from 'horse's tail').

Any pathology within the vertebral canal caudal to the L4–5 disc space may cause pain and/or a cauda equina neuropathy or so-called 'cauda equina syndrome' which has been defined as a neurological condition caused by compression, displacement or destruction of the cauda equina. The characteristics of any neurological deficits resulting from such pathology will be lower motor neuron (LMN) in nature (e.g. weak or incomplete withdrawal reflex, reduced or pseudohyperreflexic patellar reflex) with the precise characteristics being determined by the site of the lesion (see Chapter 17, p. 180).

The types of pathology which may be responsible for cauda equina syndrome include:

- Vertebral malformation/malarticulation
- Intervertebral disc prolapse (extrusion or protrusion)
- Soft tissue hypertrophy (dorsal ligament, ligamentum flavum or joint capsule)
- Neoplasia (with or without vertebral involvement)
- Vertebral fractures
- Discospondylitis
- Fibrocartilagenous embolism
- Cauda equina neuritis

Vertebral malformation/malarticulation

Congenital malformation and malarticulation of the vertebral bodies, the vertebral arches and the articular facets may cause stenosis of the vertebral canal which worsens, relatively, as the puppies grow. Such stenosis resulting in clinical signs usually affects the vertebral canal at the level of L7–S1 in this region and is primarily recognised in miniature breeds (Tarvin & Prata, 1980).

The degree of malformation may be insufficient to cause nerve root compression but the associated malarticulation may promote secondary soft tissue hypertrophy which ultimately compounds the degree of stenosis, resulting in compression of the cauda equina. Such acquired or degenerative

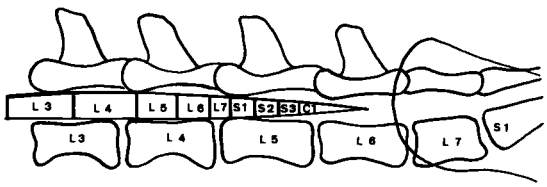


Fig. 26.1 Schematic illustration of the relationship between the spinal cord segments and vertebral bodies in the region of the canine cauda equina. Femoral nerve = L4–5; obturator nerve = L5–6; sciatic nerve = L6–7, S1; pudendal nerve = S1–2 (3).

stenosis is more commonly seen in medium-sized breeds, especially the German Shepherd Dog. There is some evidence to suggest that such malformation in the German Shepherd Dog may be related to osteochondrosis of the sacral end plate (Lang *et al.*, 1992) and similar changes have also been reported in a mastiff dog (Snaps *et al.*, 1998). There also appears to be a correlation between the incidence of cauda equina syndrome and the presence of a transitional vertebra, possibly by this contributing to chronic malarticulation and secondary soft tissue hypertrophy. In a study involving the German Shepherd Dog, Morgan *et al.* (1993) found that the frequency of transitional vertebrae in dogs with cauda equina syndrome was 38%, as opposed to 11% in dogs without cauda equina syndrome.

Intervertebral disc prolapse

Intervertebral disc extrusions may be seen in the caudal lumbar spine. They tend to be more painful than those in the thoracolumbar region and are more likely to be associated with carrying of a limb due to nerve root entrapment ('root signature') rather than neurological deficits, though mild proprioceptive deficits are not uncommon. Treatment options are as discussed in Chapter 25 (p. 246), but of the surgical options it is dorsal laminectomy that is used most often in this region. Such surgical intervention carries a high rate of success with less perioperative morbidity than may be seen with similar types of surgery in the thoracolumbar region.

Chronic fibrinoid degeneration of the intervertebral disc together with hypertrophy or hyperplasia of the dorsal annulus fibrosus may lead to disc protrusion and ventral compression of the cauda equina, most commonly at the lumbosacral junction. The compression is usually dynamic in that it is increased by hyperextension of the lumbosacral junction and reduced when the junction is flexed. Whether vertebral malformation contributes to this degeneration by causing subclinical instability is unknown, and it has been suggested that the disc degeneration may itself cause decreased stability of the ventral compartment of the vertebral column.

Soft tissue hypertrophy

Vertebral instability resulting from malformation may lead to secondary hypertrophy or hyperplasia of the soft tissues such as the interarcuate ligament, dorsal ligament or joint capsules. This hypertrophy compromises the vertebral canal and may eventually cause compression of the cauda equina.

Neoplasia

The caudal lumbar region is no more or less likely to suffer with this type of disease than other parts of the vertebral column but there may be more justification in exploratory surgery in that the cauda equina is more tolerant of surgical trauma than the spinal cord and so the postoperative morbidity is generally less. The types of neoplasia encountered and management options available are discussed further in Chapter 28 (p. 283).

Vertebral fractures

Fractures in this region might also be associated with damage to the cauda equina but such cases are usually presented with a more acute and traumatic history than those with other forms of cauda equina syndrome, except perhaps in the case of pathological fractures resulting from vertebral neoplasia or infection. Cats with sacrococcygeal luxations, resulting from traction on their tail, may present with not only paralysis of the tail but also urinary and faecal incontinence +/– hindlimb paresis. The presentation, management and prognosis in cases with such injuries are discussed in Chapter 21 (p. 215).

Discospondylitis

Intervertebral disc spaces predisposed to the establishment of infection appear to be those where the spinal column shows greatest movement and the lumbosacral region is one such site.

The features of this disease process are discussed fully in Chapter 27 (p. 278).

Fibrocartilagenous embolism
Fibrocartilagenous emboli arise from intervertebral discs and may affect the vertebral column at any level. If they occur in the caudal lumbar spine then ischaemia of the cauda equina may result. The hallmarks of this disease process in this region would include acute onset, asymmetric, non-progressive LMN signs. This subject is discussed further in Chapter 29 (p. 290).

Cauda equina neuritis
Inflammatory conditions affecting the cauda equina are rare. They may result from diseases such as distemper, feline infectious peritonitis or protozoal infections. An idiopathic polyradiculoneuritis presenting as a cauda equina problem has also been described in two dogs (Griffiths *et al.*, 1983).

Lumbosacral disease is, to all intents and purposes, a subdivision of cauda equina syndrome whereby the lesion affecting the cauda equina is at the level of the lumbosacral junction. The types of pathology encompassed by the term lumbosacral disease would include:

- Congenital lumbosacral stenosis
- Degenerative lumbosacral stenosis (+/– narrowing of the intervertebral forminae)
- Discospondylitis

Discospondylitis may be seen at the lumbosacral junction but its diagnosis and management is discussed in Chapter 27 (p. 278) and will not be repeated here. Congenital lumbosacral stenosis results from vertebral malformation/malarticulation, as discussed above (Fig. 26.2a). Degenerative lumbosacral stenosis involves secondary changes where any underlying malformation/malarticulation is insufficient to cause clinical signs on its own. These secondary changes are illustrated in Fig. 26.2b and include:

- Intervertebral disc protrusion (Hansen type II)
- Hypertrophy of the interarcuate ligament, dorsal ligament and/or joint capsule(s)
- Spondylosis deformans (may spread laterally and compromise the intervertebral formina(e))
- Osteophyte formation on the lumbosacral end plates and articular facets

The aetiology of the pathological changes leading to compression of the cauda equina is considered to be multifactorial. Malformation of the vertebrae, causing stenosis or instability, could involve osteochondrosis lesions affecting the articular facets or vertebral end plates and thus genetic and nutritional factors may play a role. When signs develop in the skeletally mature patient it is likely that the degree of malformation alone has been inadequate to produce clinically significant spinal cord compression. However, it may have led to long-term vertebral instability resulting in soft tissue hypertrophy and/or intervertebral disc degeneration and protrusion which then compounds the degree of stenosis and leads to progressive clinical signs.

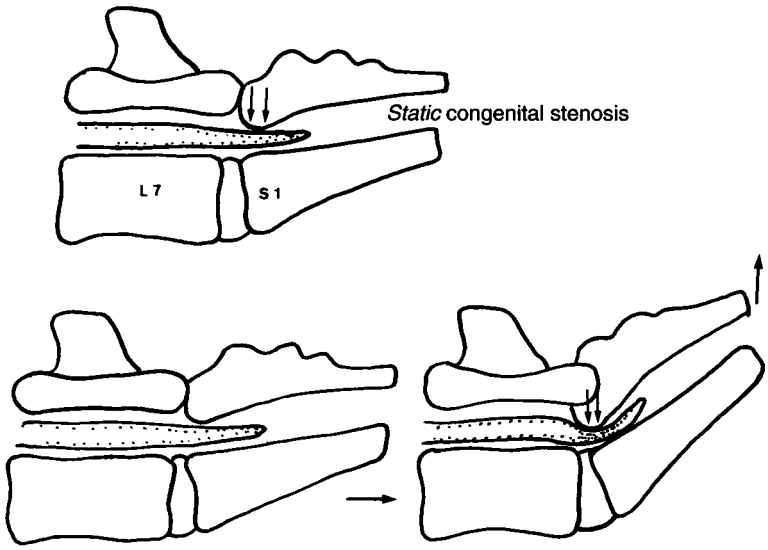
The remainder of this chapter will concern itself with canine lumbosacral stenosis specifically.

History and clinical signs

The most common feature of lumbosacral stenosis is that of 'lower back' pain. Affected dogs tend to stand with roached hindquarters and the owners will often report that they are slow to rise from a lying position, may be reluctant to sit and tend to avoid any manoeuvre that causes extension of the lumbosacral region (e.g. climbing stairs, jumping into the car, scrambling under fences, standing with their front paws on a raised level such as a window sill). Where a more persistent nerve root entrapment exists a pronounced hindlimb lameness may be present. In more long-standing cases the owners may report a progressive hindlimb weakness. Less common reasons for presentation would include self-mutilation, a low tail carriage or urinary/faecal incontinence.

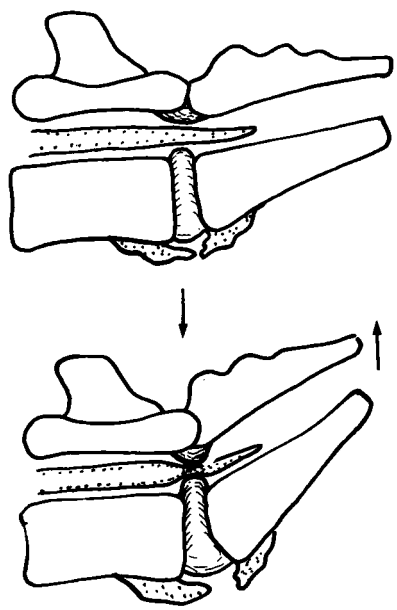
As with all cases a general clinical examination is mandatory so as to detect such indirect signs as prostatomegaly or pyrexia which might be a clue to the presence of a neoplastic or infectious condition. An orthopaedic examination is also required to rule out other diseases that may cause a similar clinical history (see under 'Differential diagnosis' below).

Observation of the patient at walk and trot should allow detection of stiffness, lameness or



No compression but extension causes compression = *dynamic congenital stenosis*

(a) Congenital lumbosacral stenosis showing osseous stenosis of the vertebral canal and mild pinching of the cauda equina which may be static or accentuated by extension of the lumbosacral junction.



Minimal compression but extension causes marked compression = *dynamic degenerative stenosis*

(b) Degenerative lumbosacral stenosis with soft tissue hypertrophy and an intervertebral disc protrusion compromising the cauda equina. A feature which is accentuated by extension of the lumbosacral junction.

Fig. 26.2 Schematic illustration of the types of lumbosacral stenosis.

scuffing of the nails. These patients will often show reluctance to climb stairs. Direct pressure applied over the lumbosacral junction may elicit pain or, in some cases, pain will only be noted when pressure is applied whilst both hindlimbs are held extended (the lordosis test). It must be remembered that extension of both hindlimbs will also produce pain in the presence of coxofemoral arthritis. Thus, if pain is elicited by the lordosis test then manipulation of each coxofemoral joint independently is required to eliminate these joints as potential sources of the pain (ideally with the patient in lateral recumbency as this allows hip manipulation without stressing the lumbosacral junction). Palpation of the hindlimbs may reveal muscle atrophy which, if present in cases with lumbosacral disease, should selectively affect the hamstring muscles due to sciatic nerve involvement.

Neurological function may be normal. If the nerve roots are compromised sufficiently to affect their function then the signs will depend on which nerve roots are compromised. When deficits are present they most commonly relate to sciatic nerve involvement. Proprioception may be reduced and the pedal withdrawal reflex may be reduced or incomplete (e.g. poor flexion of the hock despite good flexion of the stifle). Less commonly the pudendal, pelvic or caudal nerves may be affected. Involvement of the pudendal nerves causes a reduction in anal reflex, poor anal tone and reduced perianal skin sensation, whereas pelvic nerve involvement causes an easily expressed, atonic bladder and caudal nerve involvement causes reduced motor and sensory function in the tail. The patellar reflex should not be affected directly and will be either normal or else pseudohyperreflexic due to a reduction in hamstring muscle tone if sciatic nerve function is affected. A reduction in the patellar reflex may indicate more widespread pathology of the cauda equina or a more generalised neurological disease such as degenerative myelopathy.

Differential diagnosis

In cases showing 'lower back' pain or hindlimb lameness/weakness, alternatives to lumbosacral stenosis as possible causes would include:

- Other causes of cauda equina syndrome:
  - intervertebral disc extrusion (Hansen type I)
  - neoplasia
  - discospondylitis
  - fracture
  - fibrocartilagenous embolism
  - cauda equina neuritis
- Other neurological diseases:
  - degenerative myelopathy (chronic degenerative radiculomyelopathy (CDRM))
- Orthopaedic diseases:
  - coxofemoral osteoarthritis
  - cruciate disease

Diagnosis

Radiography remains the principal method of diagnostic imaging although more advanced modalities such as magnetic resonance imaging (MRI) appear to provide more reliable information due to the limitations of the contrast studies discussed below. Electrophysiological studies may be used to complement any such imaging.

Plain radiography

Plain radiographs are useful in helping to rule out other potential causes of the clinical signs such as vertebral neoplasia or discospondylitis. In cases of congenital stenosis there may be evidence of narrowing of the vertebral canal but such changes are non-definitive. In cases of degenerative stenosis plain radiographic abnormalities may be seen (Fig. 26.3), including:

- Spondylosis deformans
- Narrowing or wedging of the lumbosacral disc space
- Increased density of the adjacent vertebral end plates

However, although such radiographic abnormalities are undoubtedly indicative of degenerative pathology, they are often found in dogs that are not showing any related clinical signs and so, although such radiographic changes may be suggestive of a diagnosis, they are far from definitive. Flexion/extension views may be useful to demonstrate instability by the appearance of, or increase

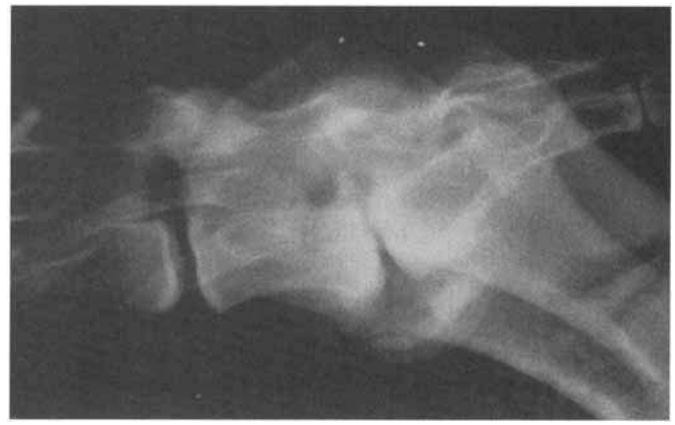


Fig. 26.3 Lateral radiograph of the lumbosacral spine of a 9-year-old Labrador Retriever showing clinical signs of pain. Lumbosacral spondylosis is present along with narrowing of the disc space. Although these features are suggestive of degenerative lumbosacral stenosis, they will be found in many clinically asymptomatic middle-aged dogs of medium- to large-sized breeds.

in, the degree of vertebral tilting present. They might also show a reduced range of motion in cases with degenerative stenosis (Schmid & Lang, 1993). In the evaluation of clinical cases it is probably unnecessary to carry out these views during plain radiography since much more useful information would be gleaned if they were performed during contrast studies.

Contrast radiography

Contrast radiography may involve myelography, epidurography or discography and preference for each technique varies between investigators. The authors prefer to begin such contrast studies with *cisternal myelography* (see Chapter 19, p. 189) and choose this route in preference to lumbar puncture as the latter may allow some contrast to be introduced into the epidural space which may lead to a confusing image (see later). After cisternal puncture, contrast can be followed from the cervical spine right down to the lumbosacral junction which allows certain other potential causes of the clinical signs to be ruled out (e.g. neoplasia). If the dural sac passes beyond the lumbosacral junction then myelography should demonstrate any stenosis, though flexion/extension studies may be required to show this conclusively (Fig. 26.4). In a significant proportion of cases (about 20% of medium-sized breeds) the dural sac terminates cranial to the lumbosacral junction and in these cases myelography alone is insufficient to confirm the diagnosis, though it still helps to rule out other causes. The situation may arise where the dural sac does pass beyond the lumbosacral junction but the compression is so severe as to prevent contrast from diffusing beyond this point. If the lumbosacral junction is held in flexion for a few minutes and then relaxed again it may be found that contrast has passed into the sacrum thus delineating the compressive lesion. In some cases the compressive lesion will only be noted, or accentuated, in views taken with the lumbosacral junction in hyperextension and may be reduced, or eliminated, in views taken with the lumbosacral junction in flexion.

If a myelogram fails to pass beyond the lumbosacral junction, despite manipulation, then alternative techniques may be required and these usually involve discography or epidurography. *Discography* involves the injection of contrast agent into the nucleus pulposus of an intervertebral disc. It is not possible to inject into a normal disc but a degenerate nucleus will accept 2 ml of contrast with little resistance. The same contrast agent and needles can be used as described under 'Myelography' (Chapter 19, p. 189). The needle is passed through the ligamentum flavum, in the midline, across the vertebral canal and through the dorsal ligament and dorsal annulus. Needle placement is between the two adjacent dorsal spinous processes in the midline and radiographs need to be taken in both planes if correct needle placement is to be ensured (Fig. 26.5). The dog may be placed in sternal recumbency with the hindlimbs drawn cranially, or in lateral recum-

bency with the lumbosacral junction fully flexed. If the signs are lateralised, and the procedure is performed with the dog in lateral recumbency, then the affected side should be uppermost so that any leakage from the disc in that direction cannot be attributed to gravity. Lateral and ventrodorsal (or dorsoventral) views are taken following injection, often with the needle still in place (Fig. 26.6). An ability to inject contrast into the nucleus pulposus is evidence of degeneration, leakage into the dorsal annulus suggests the presence of a disc protrusion, and contrast entering the epidural space demonstrates rupture of the annulus with or without extrusion of nucleus pulposus. If necessary, an epidurogram can then be produced by partially withdrawing the needle and injecting additional contrast agent.

Epidurography involves the introduction of contrast agent into the epidural space. The same contrast agents and needles can be used as described under 'Myelography' (Chapter 19, p. 189). The injection sites used are most commonly the lumbosacral junction or the cranial region of the coccygeal spine. Since cerebrospinal fluid (CSF) will not be obtained it is usual to inject a test dose of contrast agent to ensure correct needle placement. The final dose of agent required depends on the size of dog and the area of interest and may range from 1 to 8 or 10 ml. Films are examined for defects in the contrast. Wave patterns and cuffing around the nerve roots are normal findings. The interpretation of epidurograms can be problematic owing to their irregular normal appearance (Fig. 19.9). Although some radiologists use this as a preferred method of investigating clinical problems relating to this region (Selcer *et al.*, 1988), many investigators, including the authors, use it only as a last resort.

Magnetic resonance imaging (MRI)

Magnetic resonance imaging (MRI) has been reported to provide more useful information (deHaan *et al.*, 1993) and would be expected to produce less false positives and false negatives when compared to contrast radiography. Although such facilities are becoming more readily available for 'elective' veterinary patients, contrast radiography currently remains the diagnostic aid used most commonly and, in the majority of cases, will provide sufficient information on which to confirm the clinical diagnosis.

Electrophysiology

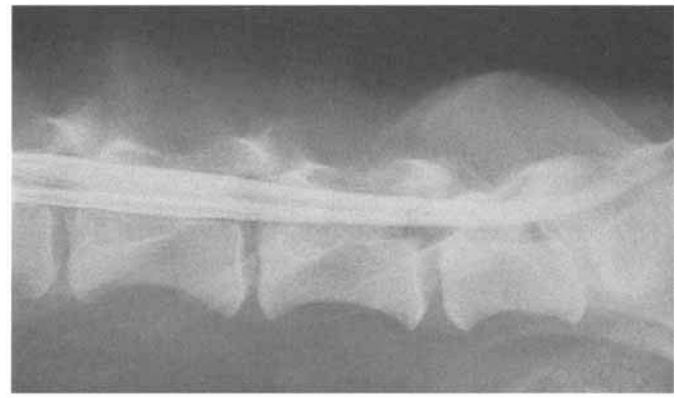
Electromyography of the limbs, tail and perineum may or may not reveal denervation potentials indicating lower motor neuron (LMN) involvement. Although the presence of such abnormalities supports a diagnosis of lumbosacral disease it does not help differentiate from other neurological disease and their absence would not preclude it. It has been suggested that abnormal radiographic contrast studies are more likely to be of significance when electromyographic abnormalities are also present (Sisson *et al.*, 1992).

Treatment

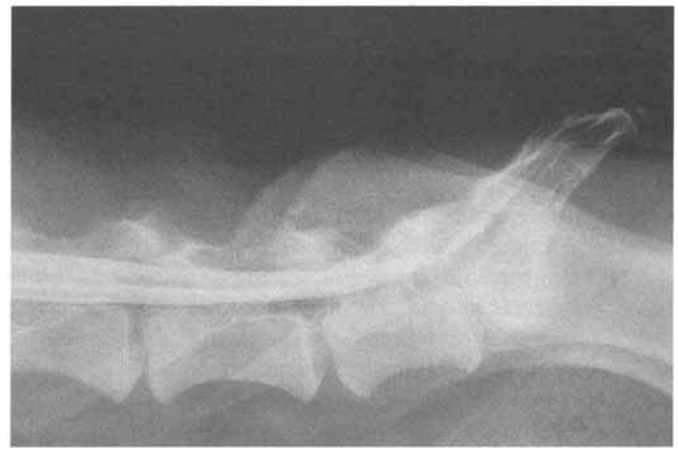
Dogs with an acute onset of clinical signs, particularly when pain is the predominant feature, may respond to conservative measures involving controlled exercise (lead walks/room confinement), weight loss (if appropriate) and a course of non-steroidal anti-inflammatory drugs (NSAIDs) (e.g. Metacam, Boehringer Ingelheim) for 1 month. If low-grade pain persists then the options would include management of the signs using long-term NSAID treatment or an alternative modality such as acupuncture, or turning to surgery. The option chosen in any given case would depend on the specific details of that patient, e.g. age, severity, function required (pet or working dog), presence of other significant disease.

In cases showing a poor response to such measures, early recurrence of clinical signs or when progressive hindlimb paresis becomes a feature, surgery should be considered. In planning surgery for the treatment of a specific case it is important to define the type of compressive lesion present since it is only then that an appropriate method may be chosen. Compression may be due to:

- Osseous stenosis due to vertebral malformation/malalignment, i.e. congenital stensosis, which may be static or dynamic
- Soft tissue hypertrophy and/or intervertebral disc prolapse, i.e. degenerative stenosis, which may be static or dynamic



(a)



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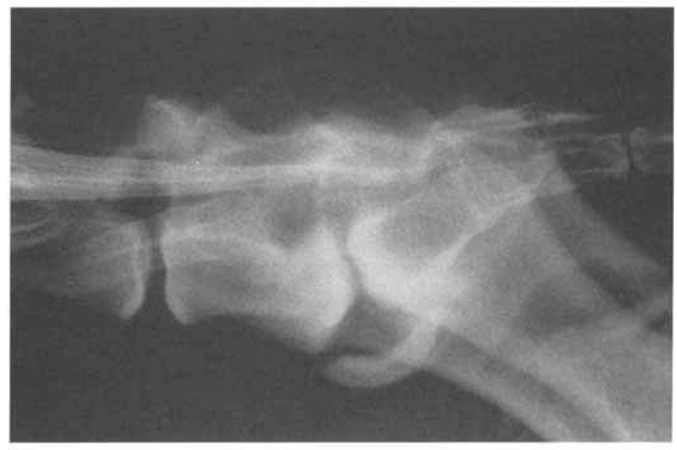
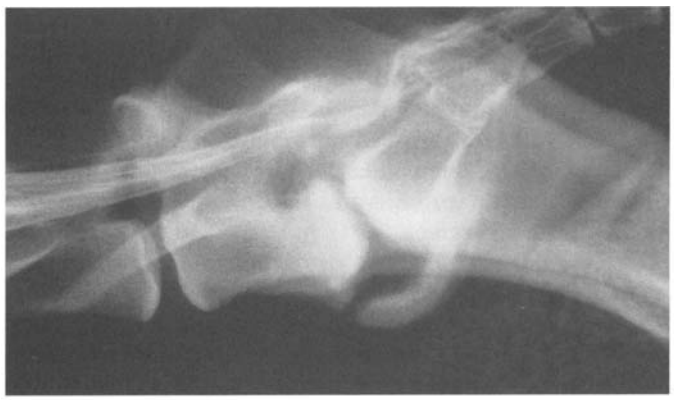


(c)

Fig. 26.4 (a) Lateral radiograph of a 2-year-old Dobermann Pinscher's lumbosacral junction after cisternal myelography. The appearance is normal.
(b) The same dog as in (a) but the lumbosacral junction is now being extended. There is now dorsal compression of the dural sac.
(c) The same dog as in Fig. 25.3 after cisternal myelography. Mild ventral compression of the dural sac is evident at the lumbosacral junction.
(d) *Opposite* The same dog as (c) but the lumbosacral junction is now being extended. The degree of compression has increased. In some cases this manipulation has the effect of 'squeezing' the visible termination of the dural sac in a cranial direction.

In those cases where a degree of stenosis exists, whatever the position of the lumbosacral junction (i.e. a static lesion), surgery will generally involve one or more of the following (as described below):

- Dorsal laminectomy
- Partial discectomy
- Curettage of osteophytes from the vertebral end plates



(d)

Fig. 26.4 *Contd.*

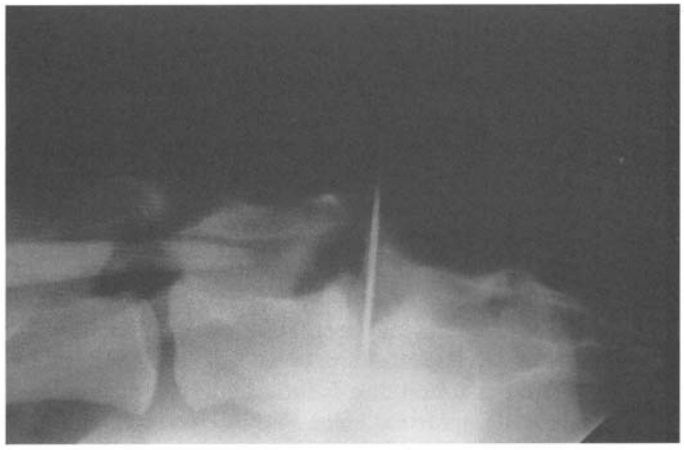


Fig. 26.5 Lateral radiograph of a 3-year-old Golden Retriever's lumbosacral junction taken after cisternal myelography. The dural sac terminates within the seventh lumbar vertebra. A spinal needle has been placed into the disc space. A dorsoventral view is also required to ensure correct needle placement.

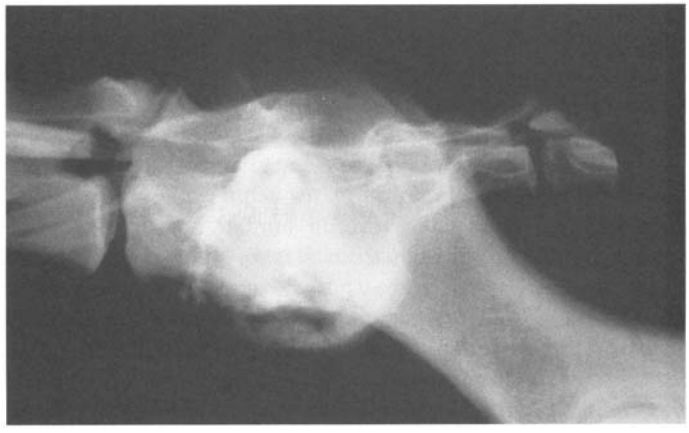


Fig. 26.6 The same dog as in Fig. 26.5 after injection of 2 ml of contrast agent. It is not possible to introduce contrast into a normal disc. In this case the contrast has diffused throughout the degenerate disc and shows evidence of a type II prolapse.

- Foramenotomy or facetectomy to relieve compression from the L7 nerve root

In the past, ventral fenestration has been described for the treatment of such degenerative stenosis but it might actually worsen the stenosis by allowing collapse of the disc space. It might be considered as an option in cases with discospondylitis to allow retrieval of material for culture. Exposure is via laparotomy.

In those cases where compression is present, when the lumbosacral junction is extended but absent when it is flexed or, better still, in a neutral position (i.e. a dynamic lesion), surgery may involve vertebral fusion with or without distraction. This may involve a dorsal approach, distraction of the vertebrae, stabilisation using transvertebral pins and the promotion of vertebral fusion by the application of a bone graft (Slocum & Devine, 1986), as described below. Alternatively, a ventral approach may be used to allow fenestration of the disc, curettage of the end plates to encourage fusion and stabilisation by placement of a transvertebral lagged bone screw (Jeffery, 1995). The latter technique has not been extensively reported and studies of large numbers of cases to evaluate results are not currently available.

Dorsal laminectomy +/– additional techniques (Fig. 26.7)

The patient is positioned in sternal recumbency with the lumbar spine in flexion (Fig. 26.7a). A midline skin incision is made over the lumbosacral junction extending from the dorsal spine of L5 to the tail base (Fig. 26.7b). Incision and retraction of the lumbodorsal fascia exposes the longissimus dorsi and multifidus lumborum muscles (Fig. 26.7c). These are divided in the midline and, after their fascial attachments to the dorsal spines have been severed, they are retracted bilaterally. The dorsal aspects of the last lumbar vertebra and first sacral segment are cleared of soft tissues which allows identification of the interarcuate ligament covering the junction between the two vertebrae. The interarcuate ligament is removed by sharp dissection to expose the epidural fat which covers the cauda equina (Fig. 26.7d). The dorsal spines of the last lumbar vertebra and the first sacral segment are removed with bone cutters or rongeurs. Dorsal laminectomy (Fig. 26.7e) may be carried out with rongeurs only or else by first reducing the dorsal laminae to eggshell thickness with a high-speed burr. The laminectomy should include the caudal half to two-thirds of the last lumbar vertebra and the cranial half of the first sacral segment.

In cases with a congenital, static stenosis this may be the limits of the required surgical treatment. However, in cases with degenerative pathology additional measures are almost always required.

The cauda equina can be retracted (Fig. 26.7f) to expose the underlying intervertebral disc since the nerve roots are relatively tolerant of manipulation, behaving more akin to peripheral nerve trunks (such as the sciatic nerve which can be retracted during fixation of, say, acetabular fractures with little fear of permanent damage) rather than to spinal cord, where minor manipulation may cause marked neurological deterioration. In many cases the dorsal annulus does appear to protrude into the vertebral canal but it may not do so even where a disc protrusion was suspected radiographically. This may be due to the lumbosacral junction having been positioned in flexion for surgery. Fenestration, or partial discectomy, of this intervertebral disc is indicated in any cases with radiographic evidence of a disc protrusion and is usually achieved in two stages. The cauda equina is first retracted to one side, a rectangular piece of dorsal annulus is then removed followed by curettage of the nucleus pulposus. Then it is retracted to the opposite side to allow extension of the fenestration across the floor of the vertebral canal and re-curettage of the disc space. At the same time it is possible to remove any osteophytes from the vertebral end plates using rongeurs.

Finally, the seventh lumbar nerve roots need to be examined to ensure that they are not entrapped as they pass through the relevant intervertebral foramen. Using a blunt retractor each nerve root can be mobilised and should move freely through 2–3 mm. It must be borne in mind, though, that during surgery the lumbosacral junction has been placed in flexion which should optimise the diameter of the foraminae. Thus, a mobile seventh lumbar nerve, as determined at surgery, might not be so when the dog is ambulatory. If

there is any suggestion that the nerve root might be trapped, either from the clinical presence of lameness or the radiographic presence of laterally positioned spondylosis, then it may be better to progress to surgery aimed at relieving any such entrapment. The simplest method of achieving this involves removal of the caudal articular facet of the last lumbar vertebra (facetectomy) (Fig. 26.7g). However, there is some concern that doing so may destabilise the lumbosacral junction further and make recurrence of clinical signs more likely, and it is strongly recommended by most authors that such a facetectomy is not carried out bilaterally. Instead, it may be preferable to remove the laminae from around the foramen whilst leaving the articular facets intact (foramenotomy) (Fig. 26.7h), which is less likely to reduce stability and may be performed bilaterally.

Closure is usually preceded by placement of a free fat graft over the site of laminectomy (+/– around the nerve root at the site of foramenotomy) to try and reduce the likelihood of recurrence of clinical signs due to laminectomy membrane formation. The dorsal musculature is then apposed using interrupted sutures of polydioxanone (PDS, Ethicon) followed by the lumbodorsal fascia using the same suture pattern and material. The remainder of the closure is routine. The likelihood of seroma formation can be markedly reduced if each tissue layer is 'tied down' to the previous one when the sutures are being placed.

Postoperatively, the dog should be restricted to controlled exercise (lead walks/room confinement) for 1 month with NSAIDs dispensed as required. Exercise may then be gradually increased to normal over the following 2 months.

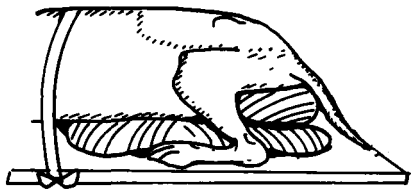
Vertebral distraction–fusion (Fig. 26.8)

The surgical approach is as detailed above to the point of removing the interarcuate ligament. The last lumbar vertebra and sacrum are then distracted by placement of retractors between the dorsal laminae of these vertebrae. Gelpi retractors may be used for this purpose but stifle joint distractors (Veterinary Instrumentation, Sheffield) are more robust. Crossed Steinmann pins are then driven across the articular facets and into the ilial wings, with or without first curetting the facets to encourage fusion. The fixation is more stable if the pins are first driven through the dorsal spine of the last lumbar vertebra but this may prove difficult to achieve, particularly for both pins, and is not an absolute necessity. These pins may have a tendency to migrate and one modification to the original technique (Slocum & Devine, 1986) is to use partially threaded pins (McKee *et al.*, 1990), though this does not stop migration completely. Bone screws have been used to stabilise the lumbosacral articular facets but, although there is a reduced risk of implant migration, there is likely to be a higher risk of postoperative fracture of the articular facets when compared to pins that pass through the dorsal spinous process. The cortical surface over the caudal aspect of the last lumbar vertebra and first sacral segment is removed with a high-speed burr. A bone graft is most conveniently harvested from one or both iliac wings and is applied dorsally to promote vertebral fusion which usually occurs within 3 months.

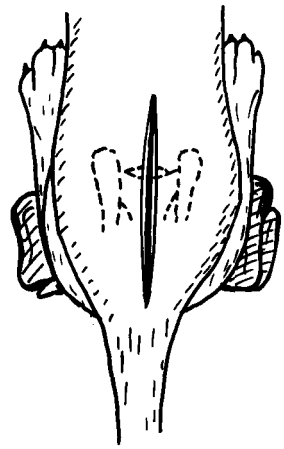
Postoperatively, the dog needs restricting to controlled exercise (lead walks/room confinement) until there is radiographic evidence of vertebral fusion (first assessed at 3 months postsurgery). Exercise may then be gradually increased to normal over the following month or two.

Prognosis

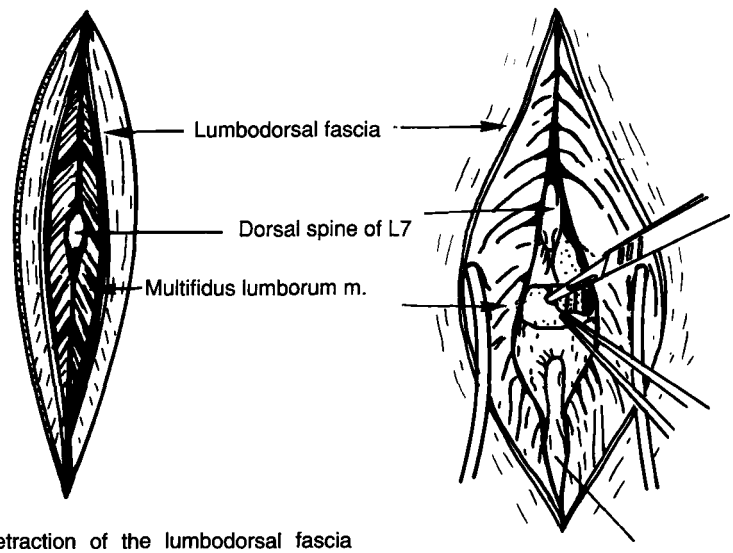
The prognosis depends on the nature of the lesion, the severity and the duration of the clinical signs. In cases with congenital stenosis causing clinical signs, there will often be no response to conservative management but a good outcome following decompressive surgery (Tarvin & Prata, 1980). Those patients with degenerative stenosis causing pain +/– mild proprioceptive deficits may respond well to conservative measures with a success rate of 8 out of 16 dogs (50%) being reported in one study (Ness, 1994). In such cases that fail to respond to conservative measures, surgery can be expected to provide a satisfactory outcome in a high proportion of cases with one report stating a success rate of 72% (Chambers *et al.*, 1988). Reasons for failure of surgery to alleviate the pain probably revolve around this being a degenerative condition involving numerous structures, not all of which are dealt with by the surgery



(a) The dog is placed in sternal recumbency with the lumbosacral junction in flexion.



(b) The midline, dorsal skin incision extends from the dorsal spine of L5 to the tail base.



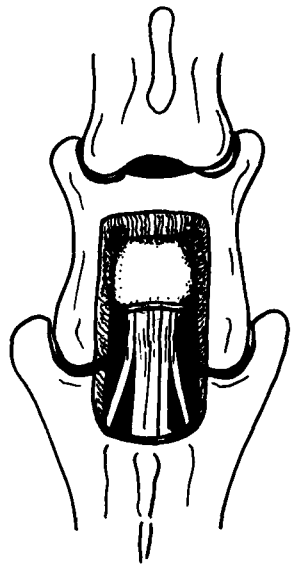
(c) Incision and retraction of the lumbodorsal fascia exposes the longissimus dorsi and multifidus lumborum muscles.

(d) Severing of the muscle attachments to the vertebrae allows their retraction, exposing the interarcuate ligament between L7 and S1 which is then removed by sharp dissection.

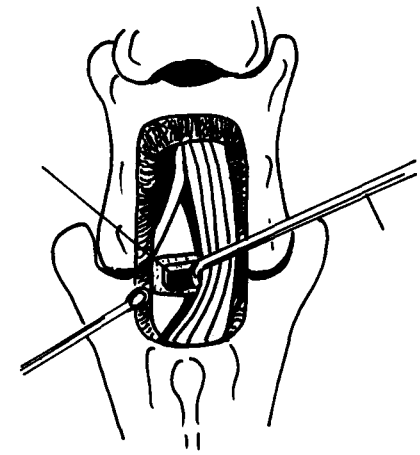
Fig. 26.7 Schematic illustrations of decompressive or excisional surgery in the management of lumbosacral stenosis.

described above (e.g. pain from osteoarthritic facet joints, pain from associated muscle spasm, etc.). Recurrence of signs may occur after decompressive surgery and is believed to be associated with formation of a laminectomy membrane. Implant failure following a distraction–fusion technique is also a possibility. The proportion of cases in which these complications may be expected has yet to be determined clinically.

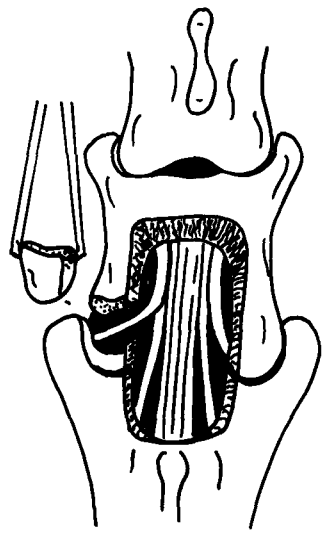
When dogs are showing more severe and long-standing neurological deficits the prognosis is less good. Conservative management is unlikely to be effective and surgery is likely to lead to a more prolonged and less complete recovery. In those



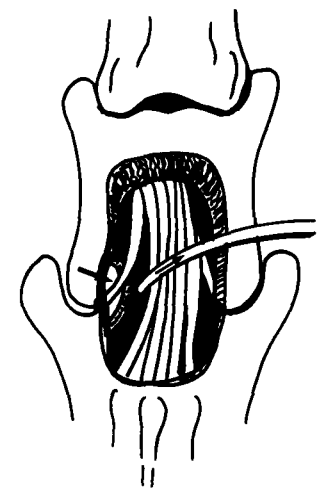
(e) After removal of the dorsal spines of L7 and S1, with rongeurs or bone cutters, a dorsal laminectomy is created using either rongeurs alone or else by first using a high-speed burr to reduce the laminae to eggshell thickness. The laminectomy should include the caudal two-thirds of L7 and cranial half of S1.



(f) In most cases the cauda equina is retracted to allow partial discectomy of the protruding intervertebral disc. This is done in two stages, first retracting the nerve roots to one side, allowing removal of a rectangular piece of dorsal annulus followed by curettage of the nucleus pulposus. Second, they are retracted to the opposite side to allow extension of the fenestration across the floor of the vertebral canal and re-curettage of the disc space. At the same time it is possible to remove any osteophytes from the vertebral end plates using rongeurs.



(g) If L7 nerve root entrapment is suspected then this can be relieved, most simply, by removal of the caudal articular facet of L7 on that side (facetectomy).



(h) However, facetectomy may destabilise the L–S junction and foramenotomy is arguably preferable.

Fig. 26.7 *Contd.*

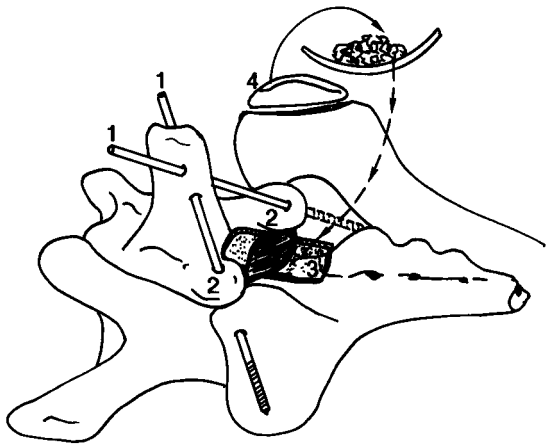


Fig. 26.8 Schematic illustration of the principles of distraction–fusion surgery in the management of lumbosacral stenosis. For clarity only the right wing of the ilium is shown. Threaded pins (1) are driven through the spine of L7 and down through the lumbosacral articular facets (2). A high-speed burr is used to remove the cortical surface (3) over the caudal aspect of the last lumbar vertebra and the cranial edge of S1 before application of a corticocancellous bone graft taken from the iliac crest (4).

patients showing urinary incontinence only one out of eight showed a satisfactory response to surgery (Chambers *et al.*, 1988).

Cauda equina syndrome in the cat

Although cauda equina syndrome or lumbosacral disease may be seen in the cat, except for, perhaps, lymphosarcoma infiltration of the vertebral canal (Northington & Juliana, 1978), it is a rare occurrence. When it is seen, the associated signs, methods of investigation and possible lines of treatment are similar to that outlined in the dog (Hurov, 1985) though the anatomy of the feline lumbosacral spinal cord is not quite the same as that of the canine (Kot *et al.*, 1994).

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Chapter 27
Discospondylitis

Discospondylitis is an inflammatory process involving an intervertebral disc together with its adjacent vertebral end plates. It is most commonly caused by bacterial infection and is seen predominantly in males of the larger breeds. Clinical signs most commonly involve lethargy, anorexia, pyrexia and stiffness due to spinal pain. In some cases there may be neurological deficits due to cord or nerve root compression. Any disc space may be involved but there are predilection sites. Most cases respond favourably to appropriate antibiotic treatment.

Aetiopathogenesis

Organisms involved

The cause of discospondylitis is usually bacterial infection and the most commonly reported isolate is *Staphylococcus intermedius (aureus)* (Kornegay, 1986). Other bacteria that have been implicated include *Streptococcus canis*, *Pasteurella* spp., *Proteus* spp., *Corynebacterium* spp., *Actinomyces* spp., *Nocardia* spp., *Bacteroides* spp., *Mycobacterium* spp. and *Brucella canis*. Mycotic discospondylitis may result from infection with *Aspergillus* spp. and although this is generally restricted to geographical areas with a warm, dry climate a case has been reported in the UK (Butterworth *et al.*, 1995).

Route of infection

The main route of infection is believed to be haematogenous but the source of the bacteraemia may be difficult to identify. Discospondylitis is often associated with lower urinary tract infection but other sources of bacteria might include periodontal disease, otitis externa, skin infections and perianal fistula. In the case of lower urinary tract infection it has been speculated that bacteria may gain access to the vertebral structures by way of the retrograde flow of blood through the internal vertebral plexus. Some cases are associated with tracking foreign bodies. These usually take the form of grass awns which may gain entry through the lungs or limbs. Normal soft tissue movement encourages their migration along fascial planes. They tend to come to rest in the tissues surrounding the cranial lumbar vertebral column (L2–4) where the crura of the diaphragm insert. Finally, discospondylitis may be seen as an iatrogenic complication of surgery involving fenestration. Fortunately this is not common.

Sites affected

Any disc space can be affected and, possibly due to the haematogenous origin of the infection, multiple disc spaces may be involved. However, there are certain predilection sites, namely the cervicothoracic, thoracolumbar and lumbosacral junctions. These sites are also where chronic instability may lead to degenerative changes and type II disc protrusion as seen in cervical spondylopathy and degenerative lumbosacral stenosis. It may be that this instability compromises the normal host defence mechanisms, thus increasing the likelihood of infection becoming established at these sites. Alternatively, or additionally, the nature of the tissue found in an area of degenerative change might itself make the environment more compatible with the establishment of infection.

History and clinical signs

The owner will rarely report any specific symptoms except when spinal pain is marked. The majority of the dogs suffering with this condition will be of medium- to large sized breeds and are usually middle-aged and male (male:female ratio of about 2:1). Most cases will have an insidious history of malaise and inappetance. Obviously a known history of foreign body penetration, of the foot or pharynx, or of recent spinal surgery would be important. In addition, any known site of recurrent infection, such as cystitis or otitis externa, might be of relevance. The presence of draining sinuses on the body wall should be taken as strong evidence of foreign body involvement.

Differential diagnosis

Possible causes of pain associated with the vertebral column include:

- Intervertebral disc prolapse
- Trauma
- Neoplasia
- Discospondylitis
- Inflammatory disease of the central nervous system
- Vertebral osteomyelitis
- Cervical spondylopathy
- Degenerative lumbosacral stenosis

In some cases the associated discomfort may manifest itself as abdominal pain, and discospondylitis should be borne in mind when considering causes of abdominal discomfort such as pancreatitis or in cases of pyrexia of unknown origin.

Diagnosis

Confirmation of the diagnosis relies on the detection of characteristic radiographic changes with or without culture of the organism from samples taken from the site or from potential sources of infection such as urine. A lateral radiograph of the suspected site of infection is of most value with ventrodorsal views adding little useful information.

Radiographic changes include (Fig. 27.1):

- Widening of the intervertebral disc space
- Lysis of the adjacent vertebral end plates
- Increased density within the vertebral body adjacent to the lesion
- Active (irregular) spondylitis
- Soft tissue swelling ventral to the vertebral column (foreign body)

These radiographic changes lag behind the clinical signs by up to 6 weeks. It is worth taking survey films of the entire vertebral column since these lesions are often multiple. Such a study will allow evaluation of the extent of the problem, is useful with respect to follow-up radiography and might allow a diagnosis to be made in cases where the 'active' lesion has not yet caused pathognomonic radiographic changes to develop. Scintigraphy may be of help in early cases, where radiographic changes have not yet developed, but is of limited practical application in this disease.

Once discospondylitis is suspected radiographically, blood and urine samples should be taken for aerobic and anaerobic culture as well as routine analysis. Serology can be useful for detecting infection with *Brucella canis* but, although this organism is important as a cause of discospondylitis in some parts of the world, it is rarely implicated in the UK. Samples for culture may be obtained by fine needle aspirate from the affected disc, possibly utilising ultrasound guidance, but most often disc material is retrieved if surgical curettage becomes indicated due to lack of response to treatment.

Treatment

Medical management

Most cases will respond to appropriate medication. Although the choice of drug will be influenced by the bacteriology results, antibiotic treatment should be instituted once a diagnosis has been made radiographically. Indeed, if the clinical signs fit but radiographic changes are not found, and no organisms are cultured, it might prove necessary to treat the dog empirically for 4 weeks with a review of the clinical and radiographic signs at that stage. In cases where

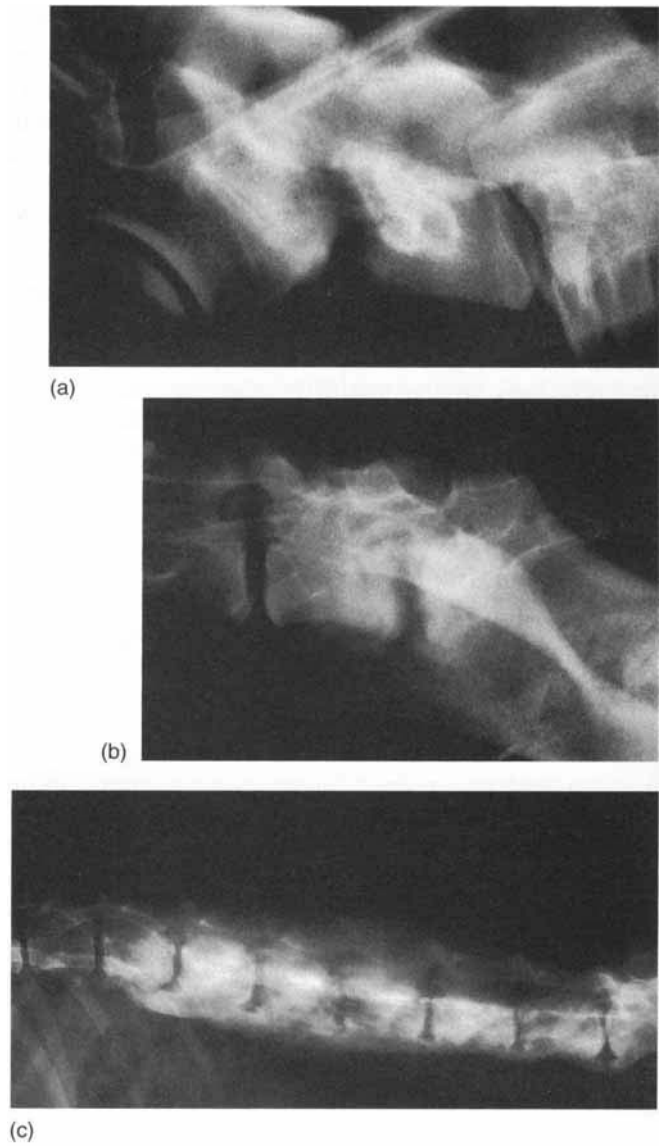

Fig. 27.1 Lateral radiograph showing discospondylitis at (a) C6–7 in a 3-year-old German Shepherd Dog that had been showing neck pain for several months and (b) L7–S1 in a 2-year-old Springer Spaniel that had been showing back pain for 4 weeks. There is evidence of end plate lysis, creating disc space widening and irregular vertebral margins, increased density of the adjacent bone and spondyle formation ventrally.
(c) Lateral radiograph of the lumbar spine of a 7-year-old Golden Retriever in which flank sinuses had been present intermittently for 2 years. All the lumbar vertebrae show an irregular density with bridging spondylosis and one area of lysis centred on the L3–4 disc space. A grass seed was suspected as the cause. No foreign body was found at surgery but radical soft tissue resection and curettage of that disc space led to an uneventful recovery.

bacteriology fails to isolate an organism it is reasonable to assume that the cause is *Staphylococcus intermedius* and to use an antibiotic to which this organism is unlikely to be resistant such as clavulanic acid-potentiated amoxycillin (Synulox, Pfizer), cephalexin (Ceporex, Schering-Plough Animal Health) or enrofloxacin (Baytril, Bayer, care re. skeletally immature patients). There should be clinical evidence of improvement within 7–10 days and if this is not the case then changing the antibiotic should be considered. Treatment with a suitable antibiotic (one on which the dog shows clinical improvement) should be continued for a minimum of 6 weeks and radiographic follow-up studies should be performed every 4–6 weeks until no further changes are noted. It must be remembered that radiographic changes lag behind clinical changes by 4–6 weeks and so it is common to see some radiographic deterioration on the first set of films taken after a suitable antibiotic has been used despite good clinical improvement during that period. Typical

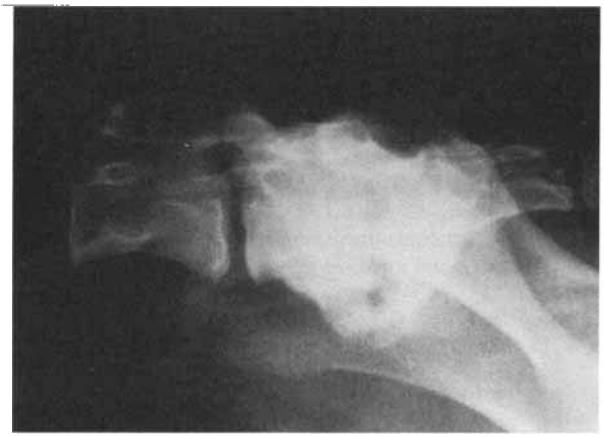

Fig. 27.2 Follow-up radiograph of the same dog as in Fig. 27.1b taken after 8 weeks. The dog had shown clinical improvement within 1 week of starting treatment with antibiotics. At 4 weeks the radiographic appearance was similar but in this film the lesion appears more productive than lytic with increased density and bridging spondylosis. Treatment was stopped at this stage and no recurrence of signs was seen.

changes seen in follow-up radiographs are shown in Fig. 27.2.

Surgical management

Surgical intervention is indicated when the clinical signs are severe or worsening, especially with respect to neurological deficits, or where there is lack of improvement with empirical treatment and tissue samples are required for culture.

The surgical techniques used in a particular case will not only depend on the surgeon's preferences but may also need to be tailored for that patient. There are several strategies to consider, and they may be used in combination. Surgical management of discospondylitis may be aimed at:

- Curettage of the infected site
- Stabilisation of the adjacent vertebrae
- Decompression of the spinal cord or nerve roots

Curettage of the disc space

Fenestration of the affected disc, allowing curettage of the site, can be performed relatively easily at most levels of the vertebral column. Such a procedure not only allows retrieval of material for bacteriology but also results in 'debulking' of the infected site and promotion of granulation tissue production with its neovascular component. The latter is important in improving access to the site for the host defence mechanisms as well as antibiotic penetration.

Stabilisation of the vertebrae

Since instability at a site has been suggested as having a role in the pathogenesis of this disease, it has been reasoned that stabilisation of the two adjacent vertebrae should be of benefit. Once the instability that compromises the normal healing and host defence mechanisms has been resolved the infection should be brought under control. These techniques generally involve placement of implants (lagged bone screws or pins) across the articular facets or else plating of the vertebral bodies.

Decompression of the spinal cord or nerve roots

If the neurological deficits are marked then decompressive surgery might have to be considered. This is most likely to involve dorsal laminectomy although hemilaminectomy might be appropriate in cases where a single nerve root is believed to be trapped. The danger of such techniques is that they will destabilise the site further and, in most instances, will have to be combined with some form of stabilisation technique. Alternatively, a distraction–fusion technique might be considered appropriate in some circumstanes, for example at the lumbosacral junction (see Chapter 26, p. 263).

A final consideration, with respect to surgery, is that of *sinus tracts*. The presence of these indicates the need for radical surgical debridement since a foreign body is usually present and the problem

will not resolve with antibiotic treatment alone. All necrotic tissue must be removed and the dead space closed meticulously or, failing that, drains may have to be left in place to avoid seroma formation. During surgery it is usually possible to curette the disc space and remove any obvious foreign material. The latter will not always be found, especially in long-standing cases, but so long as all necrotic tissue is removed a successful outcome is usually seen.

Prognosis

The prognosis for cases of discospondylitis in the UK is generally favourable with most cases responding well to antibiotic treatment, even when it has not proved possible to isolate the organism and establish its antibiotic sensitivity. Some cases will require surgical intervention and a few will fail to respond despite all possible efforts at treatment. The reason for the good prognosis is related to most cases being associated with *Staphylococcus intermedius* infection. Those cases with discospondylitis associated with *Brucella canis* infection or caused by fungal infection have a guarded and grave prognosis, respectively. Although the latter may be treated with antifungal agents such as ketoconazole in combination with amphotericin B, most cases are refractory and systemic disease is either present or becomes established leading to further deterioration.

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Chapter 28
Neoplasia of the Vertebral Column

Spinal tumours are relatively uncommon but they are a significant cause of spinal disease and should *always* be considered in the differential diagnosis (see Chapter 18), but especially in the older animal presented with a gradual onset of spinal pain and progressive neurological deficits. They may be classified according to where they are located, namely extradural, intradural–extramedullary, or intramedullary.

Extradural tumours

These arise outside the dura mater and account for some 50% of vertebral tumours in the dog (Prata, 1977). In the cat the most common extradural tumour is lymphoma (Wheeler, 1989; Spodnick *et al.*, 1992). Types of extradural tumour encountered in the dog are listed below:

- *Primary malignant vertebral tumours*
  - Osteosarcoma – commonest
  - Chondrosarcoma
  - Fibrosarcoma
  - Haemangiosarcoma
  - Multiple myeloma

  For further details of these bone tumours see Chapter 51.
- *Benign vertebral tumours*
  Osteochondroma is seen most frequently. This can be multiple and may cause cord compression.
- *Vertebral metastases*
  These are rare in animals although they may be seen in cases where, for example, an appendicular osteosarcoma has been removed followed by chemotherapy as metastases to bone appear more resistant to the effects of chemotherapy than metastases to soft tissues.

Intradural–extramedullary tumours

These lie within the dura mater but outside the spinal cord parenchyma. They account for approximately 35% of spinal tumours (Prata, 1977). Common primary tumour types include:

- Meningioma
- Neurofibroma
- Neurofibrosarcoma
- Lymphoma

Metastatic tumours occasionally arise in this site.

Intramedullary tumours

Intramedullary tumours arise within the substance of the spinal cord and are the least common of all spinal tumours, accounting for some 15% of cases (Prata, 1977). Primary tumour types encountered include:

- Astrocytoma
- Ependymoma

Metastatic tumours occasionally arise in this site.

Diagnosis

Some spinal tumours, particularly primary bone tumours affecting vertebrae, may be confirmed on plain radiographs. However, tumours of the spinal cord, meninges and nerve roots rarely cause bony changes and myelography is essential to confirm the diagnosis (Wright *et al.*, 1979). The myelographic appearance of extradural, intradural–

extramedullary and intramedullary tumours are illustrated in Fig. 28.1 (see also Chapter 19, p. 194). Spinal tumours seldom metastasise to the lungs but nevertheless thoracic radiographs should be taken to investigate this possibility. The final stage in the investigation may be exploratory surgery to confirm the presence of a tumour and/or biopsy or to remove the mass. This is generally done on the understanding that should the tumour prove inoperable/untreatable then euthanasia should be carried out at that stage.

Surgical treatment

Cases most suitable for surgery are those in which a discrete tumour lies in the extradural space or is intradural but extramedullary. Intramedullary tumours and vertebral body tumours can seldom be removed surgically.

A dorsal laminectomy is the preferred approach. This decompresses the cord and gives wide exposure for removal of the tumour. Even if tumour removal is possible the prognosis is very guarded. There may well be a worsening of neurological status post-surgery which may be transient or permanent. Spinal tumours have a tendency to recur locally, probably because of incomplete removal (Wheeler, 1991) and post-surgical radiation therapy or chemotherapy may improve the prognosis depending on the histological type of the tumour. Long remissions have been reported in some dogs after resection of meningiomas (Fingeroth *et al.*, 1987).

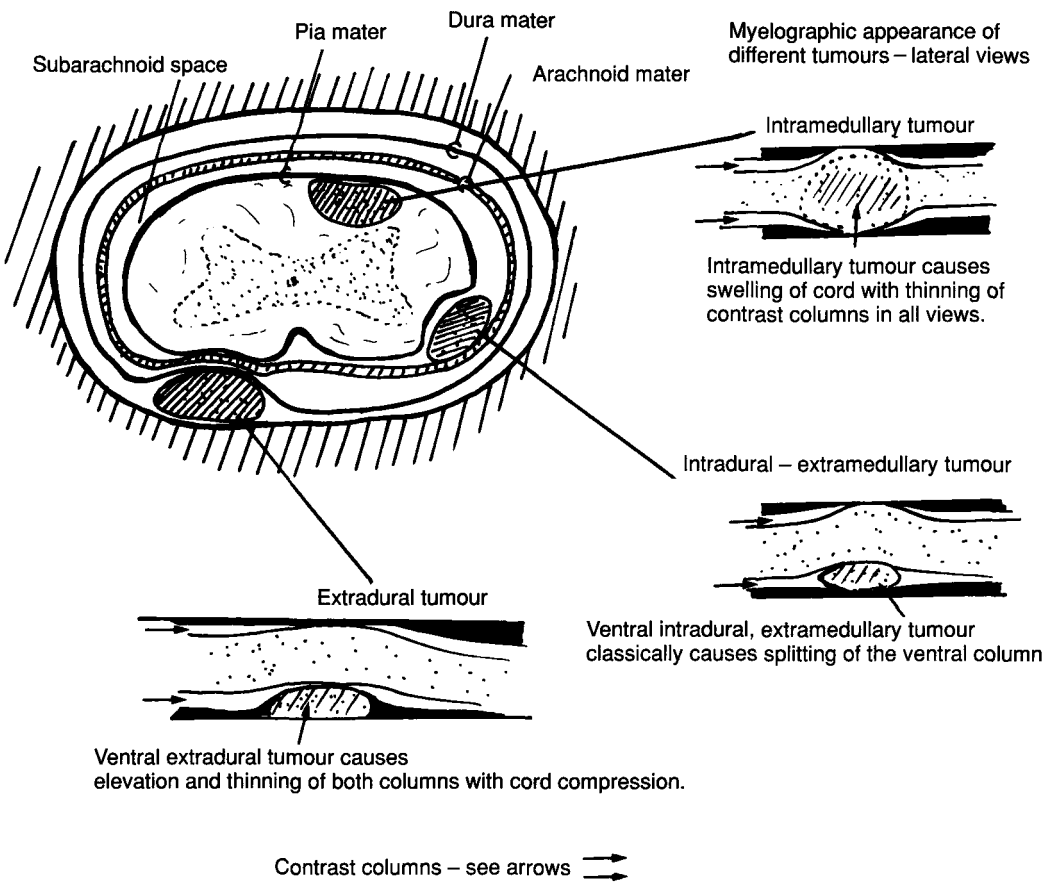

Fig. 28.1 Transverse section of the spinal cord showing the meninges and types of spinal tumour.

Feline spinal tumours

Spinal tumours account for over 50% of all cases of feline spinal disease (Wheeler, 1989). Lymphoma is the most common tumour type. The vast majority of cats with spinal lymphoma are FeLV positive (Lane & Kornegay, 1991; Spodnick *et al.*, 1992). Treatment by surgical debulking followed by chemotherapy, or chemotherapy alone (Cotter, 1986), gives an initial improvement but the long-term prognosis is poor because there tends to be multifocal involvement.

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A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny,Steven J. Butterworth

Chapter 29
Miscellaneous Conditions of the Spine

The subjects to be covered in this chapter are:

- Congenital anomalies
- Degenerative myelopathy (DM)
- Ischaemic myelopathy (fibrocartilagenous embolism [FCE])
- Inflammatory disease of the central nervous system (CNS)
- Spinal haemorrhage
- Leucoencephalomalacia
- Neuroaxonal dystrophy
- Spondylosis deformans

Congenital anomalies

A variety of congenital anomalies may affect the spine. These include: transitional vertebrae, hemivertebrae, block vertebrae, spina bifida and subarachnoid cysts. Many spinal deformities are asymptomatic and detected only as incidental radiographic findings.

Morgan (1968) reported an incidence of 47% for such deformities in a series of 145 canine spines examined. The incidental deformities found in this study included:

- Fusion of vertebral bodies
- Cervical ribs
- Change in position of anticlinal vertebra
- Hemivertebrae
- Lumbarisation of T13
- Sacralisation of L7
- Lumbarisation of S1
- Fusion of S3 and Co1
- Incomplete fusion of S2 and S3

Congenital deformities which are likely to cause clinical signs include:

- Spina bifida
- Hemivertebrae
- Atlantoaxial subluxation (see Chapter 22)
- Cervical spondylopathy (see Chapter 24)
- Spinal 'arachnoid' cyst
- Dermoid cyst
- Lysosomal storage diseases
- Spinal dysraphism
- Syringomyelia and hydromyelia

Spina bifida

This is a rare condition in dogs and cats. Affected animals tend to present with hindlimb paresis. There is failure of fusion of the left and right sides of one or more vertebral segments. Lateral radiographs show poor development or absence of neural arches and spines. Ventrodorsal projections may show vertical fissures in affected vertebrae.

Hemivertebrae

This condition is seen most often in screw-tailed breeds of dog (e.g. English Bulldog, French Bulldog, Boston Terrier). The defect usually involves a number of thoracic vertebral bodies which have a wedge-shaped deformity. This produces crowding of the ribs, scoliosis and kyphosis (Fig. 29.1). Many dogs with such vertebrae will remain clinically asymptomatic. In severe cases the resultant deviation causes spinal cord compression and hindleg paresis/ataxia at 3–4 months of age. The prognosis is poor and motor dysfunction is likely to become worse. However, some pups seem to compensate for the deformity and stabilise. In the early stages steroid therapy may help to alleviate the signs. Some dogs with hemivertebrae may not show signs until middle-

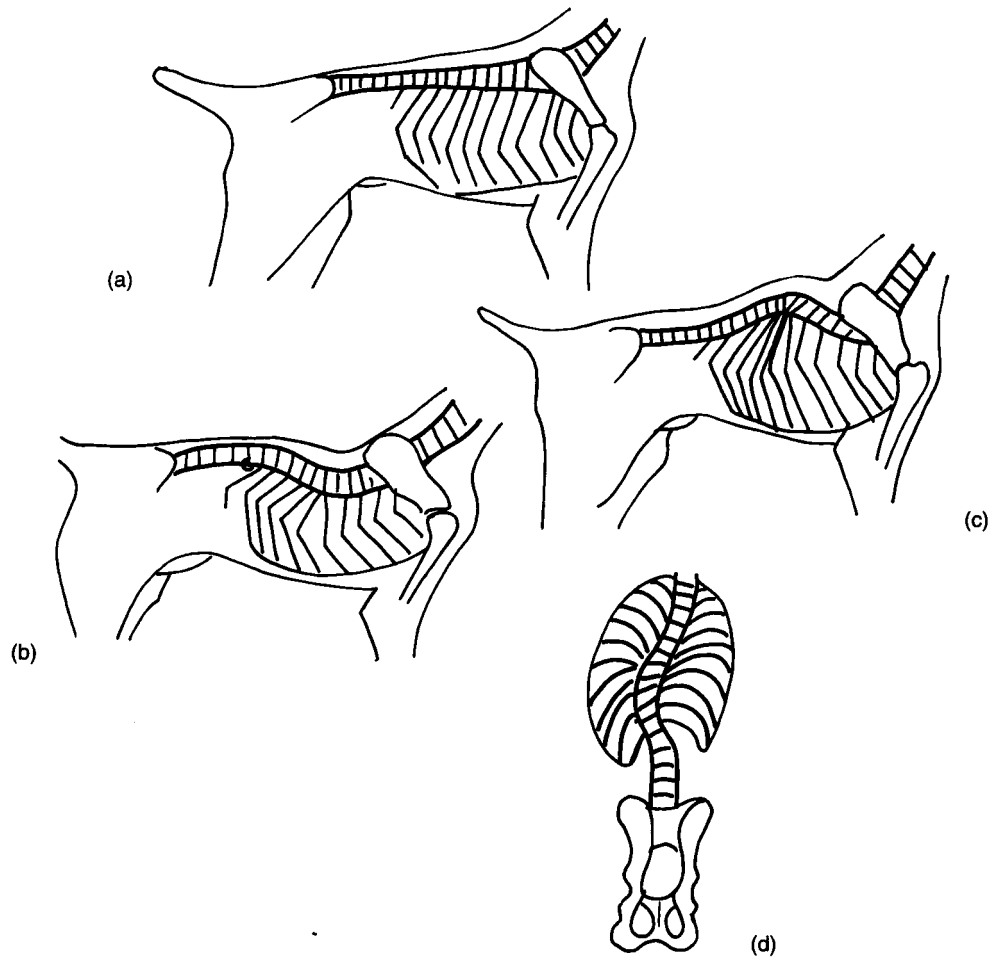

Fig. 29.1 (a) Normal spine, (b) lordosis – ventral curvature, (c) kyphosis – dorsal curvature, (d) scoliosis – lateral curvature.

age. The cause of spinal cord compression in these cases usually involves some secondary pathology, e.g. a disc protrusion, which compounds the long-standing stenosis. If decompressive laminectomy is to be undertaken it is important to carry out myelography to ensure that the hemivertebrae are the cause of cord compression and that there are no other spinal lesions present.

Spinal 'arachnoid' cysts in the dog

The arachnoid cyst is an uncommon cause of neurological dysfunction (Dyce *et al.*, 1991). The lesion is invariably focal and consists of a dorsal subarachnoid dilatation which gives compression of the spinal cord. A familial tendency may be suggested by the reporting of almost identical spinal cysts in two Shih Tzu littermates (Ness, 1998). Myelographic studies reveal a characteristic dilatation in the subarachnoid space (Fig. 29.2). Treatment involves dorsal laminectomy and durectomy, +/– marsupialisation, over the cystic area and carries a good prognosis for recovery.

Dermoid cyst (pilonidal sinus; epidermoid cyst)

These are most frequently encountered in Rhodesian Ridgebacks and Shih Tzus and are caused by an infolding of the skin in the dorsal midline. In

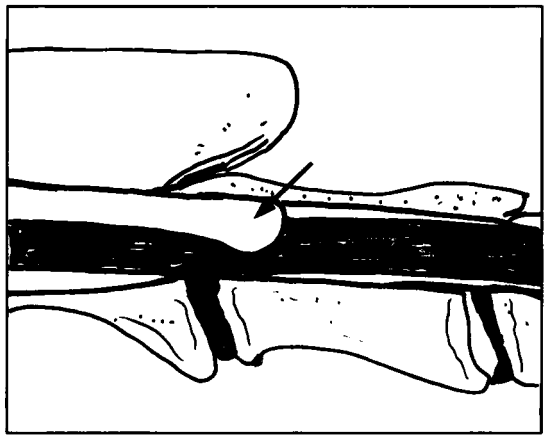

Fig. 29.2 Myelographic study demonstrating a subarachnoid cyst (arrowed) at C2–3.

some cases these 'tracts' communicate with the dura mater and thus provide an entry point for infection which may result in meningitis and myelitis. Treatment involves antibiotic therapy and surgical excision. In those cases with dural involvement it may be necessary to perform a laminectomy to excise the entire tract and doing so is not without its risks.

Lysosomal storage diseases

These result from a congenital defect in metabolism usually involving dysfunction of a specific enzyme which causes the intracellular accumulation of metabolites. Most are inherited as an autosomal recessive characteristic and they can affect both cats and dogs. Neurological signs develop early in the animal's life and are progressive. There is no treatment and the prognosis is hopeless. Definitive diagnosis depends on biopsies of specific tissues according to the nature of the particular disease involved. For further details the reader is referred to standard neurology texts.

Spinal dysraphism

This involves malformation of the spinal cord itself, most frequently in the T3–L3 region. It may be seen in many breeds of dog but the Weimaraner seems to be overrepresented (Van den Broek *et al.*, 1991) and in this breed the condition is believed to be inherited. Affected puppies develop a 'bunny hopping' hindlimb gait. The signs may or may not progress and although there is no treatment some animals may make functional pets for several years.

Syringomyelia and hydromyelia

The term *syringomyelia* describes the presence of fluid-filled cavities within the spinal cord whereas *hydromyelia* refers to the presence of cavitations within the central canal. They occur in association with other abnormalities such as dysraphism and may also be related to 'external' abnormalities of the vertebral column such as torticollis or scoliosis (Child *et al.*, 1986). Affected dogs may present with non-specific signs of pain or hyper-sensitivity evidenced by indiscriminate scratching (e.g. an 'Arnold-Chiari'-like syndrome recognised in Cavalier King Charles Spaniels (G.C. Skerritt, pers. comm.). The neurological signs resulting from such lesions depend on the region of spinal cord affected. Definitive diagnosis cannot be made with standard radiological techniques and magnetic resonance imaging (MRI) is the best aid to diagnosis. Treatment by the placement of shunts may be appropriate in some cases (G.C. Skerritt, pers. comm.).

Degenerative myelopathy (DM)

Degenerative myelopathy (DM) is a disorder characterised by the loss of myelin and axons starting in the thoracolumbar region and gradually becoming more widespread. In the past it has been referred to as chronic degenerative radiculomyelopathy (CDRM) but since the onset of signs may be fairly acute and involvement of nerve roots is seen in only the minority of cases, this term has been replaced by that of DM.

Clinical signs

The disease affects mainly medium–large breed dogs, and particularly the German Shepherd Dog, with the onset of clinical signs usually being at 5–7 years of age. Progressive hindlimb incoordination is seen with dragging of the nails, stumbling, knuckling the paws or loss of balance on cornering. Initially, the spinal reflexes of the hindlimbs are normal unless the nerve roots are affected when there will be reduction or loss of these (see

below). With time they may become exaggerated in line with the disease creating an upper motor neuron (UMN) lesion in the T3–L3 region of the spinal cord. Urinary and faecal continence are only affected late in the course of the disease, by which time the animal will be virtually non-ambulatory on its hindlimbs. Conscious pain sensation (CPS) is usually spared and the disease does not cause spinal hyperaesthesia. Ultimately, dogs showing hindlimb dysfunction will develop forelimb dysfunction and then brain stem involvement which is terminal.

In cases showing radiculopathy (less than about 10% of cases) there will be loss of lower motor neuron (LMN) function, most often seen as a loss of the patellar reflex, which is usually asymmetrical and not associated with electromyographic abnormalities.

Although the disease process is naturally progressive, fluctuations in the rate of progression are often seen with there being periods of relative stability. This undulating pattern in the natural course of the disease makes evaluation of potential treatments difficult.

Differential diagnosis

The clinical signs described above could easily be caused by other conditions such as intervertebral disc protrusion (i.e. Hansen type II) (see Chapter 25, p. 259) or neoplasia (see Chapter 28, p. 283), although both of these are likely to produce a degree of spinal pain. The disease might also be confused with degenerative lumbosacral disease or even hip dysplasia (to both of which the German Shepherd Dog is also predisposed), although careful clinical evaluation usually allows these to be differentiated (see Chapters 26 and 40).

Diagnosis

If the clinical signs are as detailed above and radiographic examination (including myelography) reveals no evidence of a structural spinal cord disease, then DM should be suspected. Cerebrospinal fluid (CSF) taken by lumbar puncture will usually show raised protein levels. Absolute confirmation of the disease in the living patient is not readily possible. Recent research, reviewed by Clemmons (1992), suggests there may be an immune-mediated pathology and that testing affected animals' cell-mediated immune responses to certain mitogens *in vitro* has revealed a depression in response that characterises the disease. Affected dogs have three to ten times the levels of circulating immune-complexes compared to normal dogs. Investigation of these immune-complexes revealed the presence of a specific protein of 85 kD molecular weight and this has raised hopes that it might be possible to develop serum markers for the disease.

Treatment

Many treatments have been suggested for this disease, but evaluation of their efficacy has been problematic due to doubts over the definitive diagnosis in many cases and the fact that the signs may stabilise naturally for a period of time. Currently, a treatment regime that has been reported (Clemmons, 1992) to significantly reduce the rate of progression of the disease, involves a combination of an active exercise programme, vitamin supplementation and the administration of aminocaproic acid (not readily available in the UK). Exercise should involve active walking, running or swimming – strolling or wandering around the garden is inadequate – on an alternate-day basis. Vitamin supplementation may include giving: high-potency vitamin B complex tablets twice daily; vitamin E at 2000 IU/day; if aminocaproic acid can be obtained then the details for its use are documented in the reference given. Corticosteroids may be helpful during periods when the clinical signs become exacerbated (prednisolone at 1 mg/kg/day in divided doses for 3 days, then 0.33 mg/kg bid for 2 days, then 0.5 mg/kg every other day for 2 weeks) but should not be used continuously as they may increase the rate of muscle wasting as a result of their catabolic activity.

Some owners have their dogs fitted with carts once hindlimb function is inadequate. The cart will allow the dog to be more active for a further 1–2 years before the spinal cord changes begin to affect the forelimbs.

Prognosis

This condition carries a poor prognosis with a natural course of 6–12 months from the appearance of clinical signs (Griffiths & Duncan,

1975) to the point where euthanasia is considered due to immobility. The treatment outlined by Clemmons (1992) is believed to slow down the rate of progression significantly, but only if treatment is started in the early stages and it certainly serves little purpose in dogs that are already markedly disabled. Those animals showing radiculopathy generally have a worse prognosis and do not appear to improve with any treatment yet described.

Ischaemic myelopathy (fibrocartilagenous embolism [FCE])

Essentially this is a syndrome involving acute infarction of part of the spinal cord. The spinal cord receives its blood supply from spinal branches of the vertebral artery which enter the vertebral canal through intervertebral foraminae (Fig. 29.3). These divide into the dorsal and ventral radicular arteries. The ventral radicular artery anastomoses with the unpaired ventral spinal artery which runs the length of the cord along the ventral median fissure. The dorsal radicular arteries join the unpaired dorsal spinal artery which may not form a continuous trunk along the length of the cord. Although the spinal cord is protected from ischaemia resulting from injury to vessels outside the cord itself by the presence of extensive anastomoses, the intrinsic vessels of the cord form an end arterial system, meaning that any obstruction may lead to significant ischaemic damage. The source of the emboli which cause this infarction is generally accepted as being the nucleus pulposus of an intervertebral disc because the histochemistry of the emboli is identical to that of the fibrocartilage within the nucleus pulposus. The mechanism by which this material gains entry to the spinal vasculature remains undetermined but several suggestions have been made and have been listed by Neer (1992) as:

- Persistence of embryonal arteries of the annulus fibrosis and herniation of nucleus pulposus material into these
- Direct arterial penetration of disc material into the ventral branch of the radicular artery or the ventral spinal artery
- Entrance of nucleus pulposus material into new arterioles forming within degenerate annulus fibrosus which could lead to the emboli reaching the ventral radicular artery
- Extrusion of nucleus pulposus material into anomalous vasculature leading to occlusion
- Nucleus pulposus material entering the venous system and then passing to the arterial

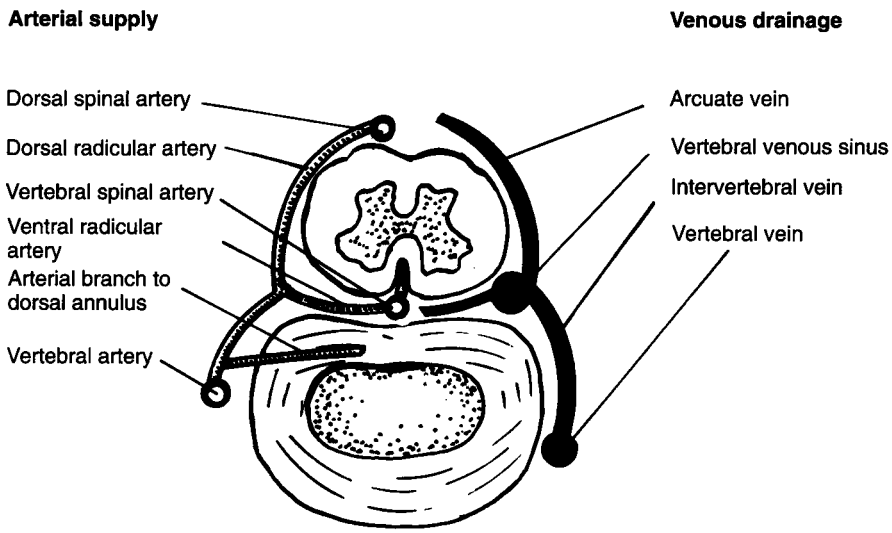

Fig. 29.3 Diagram illustrating the arterial supply and venous drainage of the spinal cord and the intervertebral disc.

side via either arteriovenous shunts or traumatically induced communications between the two sides of the vascular system
- Herniation of disc material through the vertebral end plate into the vertebral body (forming what are known as Schmorl's nodules in humans) with retrograde entrance into the venous sinuses and transfer to the arterial side as mentioned above

None of these suggestions have any scientific evidence to support them. Whichever, if any, is the case, it is generally believed that a rise in intradiscal pressure causes the material to enter the vascular system. Although this may require trauma or vigorous exercise, such a pressure rise can be created by nothing more than an awkward 'twist'. In humans it is believed that a severe coughing bout is sufficient to cause embolism.

The condition has also been reported in a cat (Scott & O'Leary, 1996).

Clinical signs

The condition tends to affect middle-aged (generally 3–7 years of age) dogs of medium to large breeds, although the authors have suspected the problem in some cases involving smaller breeds. A typical history is of a peracute onset of non-progressive neurological dysfunction. In the first instance the dog may appear to show pain but this has usually resolved by the time of examination by a veterinary surgeon, though some suspected cases have seemed to show pain for up to 24 hours. The neurological deficits will be determined by the level of spinal cord involved but one of the hallmarks of this condition is a distinct lateralisation of signs. If the cervical spine is involved then lateralising quadriparesis or hemiparesis will be seen. If the thoracolumbar spine is involved then lateralising paraparesis or monoparesis will be seen. Continence and CPS are rarely affected. It is important to recognise any LMN dysfunction (reduced withdrawal or patellar reflexes) as this influences the prognosis (see below).

Differential diagnosis

Other causes of the signs described that must be considered in such cases include:

- Intervertebral disc prolapse
- Neoplasia
- (Discospondylitis)

Diagnosis

A definitive diagnosis cannot be reached without histological examination of the dog's spinal cord and so a clinical diagnosis has to be based on the features described above and excluding other likely diagnoses by further investigation. A high index of suspicion is created by a history of a peracute onset, lateralising spinal cord lesion, without any associated pain, in a medium to large breed of dog. This suspicion is raised further by normal plain radiographic findings. Myelography should be used to rule out the differential diagnoses listed above and, in cases with ischaemic myelopathy, such contrast studies will be either normal or show mild intramedullary swelling as a result of oedema secondary to the ischaemia.

Treatment

There is no convincing evidence that any treatment is of value other than that of general nursing care in recumbent patients. The use of corticosteroids in such cases is widespread but, in the authors' opinion, of doubtful benefit. If such drugs are to be used then the same protocol as described for acute spinal trauma associated with vertebral fractures should be employed. Methylprednisolone sodium succinate (Solumedrone V, Pharmacia & Upjohn) should be given by slow intravenous injection *within* 8 hours of injury. The initial dose is 30 mg/kg, followed by 15 mg/kg every 4–6 hours with treatment not exceeding 24 hours.

Prognosis

Those cases showing signs of UMN involvement only have a reasonable prognosis with 10 out of 18 cases reported by Dyce & Houlton (1993) regaining satisfactory function. The least affected side will often recover normal function within a matter of a few days to a few weeks whilst the more severely affected side will improve significantly over a few weeks or months. Although some proprioceptive deficits may remain

long-term on the worst affected side, the dog's quality of life will become perfectly acceptable in about 60% of cases.

Lower motor neuron deficits rarely improve and cases showing these have a more guarded prognosis.

Inflammatory diseases of the central nervous system (CNS)

Inflammation of the brain is referred to as *encephalitis*, whilst involvement of the spinal cord and supporting structures is referred to as *myelitis*, and inflammation of the meninges is referred to as *meningitis*. Myelitis can be subdivided according to whether it involves the grey matter (poliomyelitis) or white matter (leucomyelitis). Most inflammatory diseases of the central nervous system (CNS) involve, to a greater or lesser extent, all of these structures and so are referred to as *meningoencephalomyelitis*. The aetiology of such disease includes viral, bacterial, rickettsial, fungal, spirochaetal and protozoal infections. A number of these diseases, such as granulomatous meningoencephalomyelitis, are idiopathic.

Clinical signs

Young to middle-aged animals are most frequently affected without there being any general sex or breed predisposition. The disease is usually acute in onset and progressive in nature. The most common sign relates to pain causing paraspinal muscle rigidity and 'stiffness' of the vertebral column. The inflammatory process can affect the CNS at any level, though the cervical region is most commonly involved. Neurological deficits may be encountered, usually in the form of reduced or absent postural reactions, and which limbs are involved depends on the region of spinal cord involved.

Differential diagnosis

Other common causes of vertebral column pain +/– neurological deficits include:

- Intervertebral disc prolapse
- Neoplasia
- Discospondylitis
- Vertebral fractures/luxations

Specific features of some of the more common causes of inflammatory disease of the CNS seen in the UK will be considered below.

Aseptic meningitis (steroid-responsive meningitis)

This disease affects mainly medium to large breeds of dog early in life with almost all cases presenting before 2 years of age, although in one study (Tipold & Jaggy, 1994) middle-aged dogs were also seen, the eldest being 7 years of age. The clinical signs include pyrexia, spinal pain (especially of the neck) and a stiff gait. Neurological signs are seen rarely. The onset of signs is usually acute but the severity of the signs may then undulate. Haematology may reveal an elevated white blood cell count with a neutrophilia whilst CSF analysis will show a markedly raised white cell count with neutrophils being predominant. These changes will be most pronounced when the signs are most severe and the CSF may appear normal during periods of remission. Organisms will not be seen in the CSF, culture will be negative and antibody titres, to the various organisms that might cause similar signs, will be insignificant. Treatment involves the use of prednisolone, starting with immunosuppressive doses of 1–2 mg/kg twice daily. A favourable response is usually seen within a week and the dose is then gradually reduced to once daily, then every other day and then by reducing the amount in each dose, over a period of about 6 weeks. Relapse may be seen in some dogs, in which case the treatment is repeated, preferably after re-establishing that there is no other explanation for the disease. The signs will not usually recur after the animal reaches 2 or 3 years of age.

In certain breeds, including Beagles, German Shorthaired Pointers and Bernese Mountain Dogs, a more aggressive meningitis with associated myelitis, encephalitis and arteritis has been recognised. The general signs described above apply to these cases also but as the disease progresses there may be neurological signs including

paresis, spinal reflex abnormalities, blindness and seizures. Laboratory findings are as outlined above but the values are generally very high. Treatment involves the use of prednisolone, as described above, but the response is less favourable and many of these dogs will carry a hopeless prognosis, especially those developing severe signs soon after the clinical onset.

Granulomatous meningoencephalomyelitis (GME; 'reticulosis')

Granulomatous meningoencephalomyelitis (GME) is characterised, histologically, by accumulations of mononuclear cells forming a whorling perivascular pattern which can be focal or diffuse within the CNS. Any breed, age and sex of dog can be affected but the onset of signs is most commonly seen between 3 and 7 years of age. The clinical signs are as described previously, though neurological deficits or intracranial signs may be seen also. They are usually chronic in onset (often indicating the focal form) but in some cases (usually those with the disseminated form of the disease) the onset may be acute with rapid deterioration. Cerebrospinal fluid analysis will usually show a moderate, predominantly mononuclear pleocytosis and raised protein levels. In an acute phase there may be a high proportion of neutrophils in the differential count. Myelography may show no abnormalities or else produce a 'patchy' appearance suggestive of irregular focal lesions within the subarachnoid space.

Treatment involves the administration of prednisolone starting with immunosuppressive doses of 1–2 mg/kg twice daily. Once the signs are in remission then the dose may be gradually reduced aiming for alternate-day therapy using a maintenance dose. Although there is a temptation to withdraw the drug altogether, there is always the risk of recurrence of signs. If this does occur then there is often a poor response to simply increasing the dose again, even back up to the original immunosuppressive levels.

The prognosis in these cases is guarded. Although it may be possible to control the signs for a period of time using prednisolone it is unusual for this to be longer than for a few weeks or months.

Canine distemper virus (CDV) infection

Canine distemper virus (CDV) is the most common infectious cause of CNS disease in the dog. Involvement of the CNS in neonates usually results in a rapidly fatal polioencephalomyelopathy whereas older dogs develop a leucoencepahlomyelopathy with lower mortality. Those showing a CDV-induced myelitis or meningitis tend to be mature and exhibit not only typical signs of spinal pain +/– paresis but also, in many cases, intracranial signs such as vestibular dysfunction or blindness. These nervous system signs may or may not be preceded by other systemic signs of illness associated with canine distemper (i.e. respiratory or gastrointestinal signs). Confirmation of the diagnosis antemortem is difficult with CSF analysis offering the best indication, although the analysis is generally normal in those cases with polioencephalomyelitis (i.e. the very young dogs). In those with leucoencephalomyelopathy the CSF will show a lymphocytic pleocytosis, and increased protein and gamma globulin levels. The presence of CDV antibodies in CSF is very indicative of the disease process although false positives can occur if serum antibodies have gained entry to the CSF either iatrogenically or through a breakdown in the blood–brain barrier.

Treatment is symptomatic and generally involves the use of prednisolone. An initial dose of 0.5–1 mg/kg twice daily is then halved every 4 or 5 days to determine the lowest maintenance dose, using alternate-day therapy if possible.

The prognosis for dogs developing neurological signs associated with CDV infection is poor. Although the signs might be controlled for some time with corticosteroid therapy, the disease is generally progressive and will become non-responsive even to increased doses. Furthermore, seizures may develop which would require additional anticonvulsant therapy.

Feline infectious peritonitis (FIP) infection

Feline infectious peritonitis (FIP) is a disease which results from coronavirus infection and affects multiple body systems in either an effusive ('wet') or non-effusive ('dry') form. The non-effusive form may be associated with the development of multifocal pyogranulomatous

meningoencephalomyelitis. Purebred cats are more commonly affected, especially when they are young and those coming from multiple cat households. The signs usually include one or more of the following: seizures, ataxia/paresis, vestibular disease and spinal pain. Other related signs include an anterior uveitis, malaise, anorexia and pyrexia. Most commonly the onset is acute and the signs progressive.

Other diseases, such as toxoplasmosis, feline leukaemia virus (FeLV) infection, feline influenza virus (FIV) infection and congenital storage diseases, may also cause similar signs and there is no definitive antemortem test for FIP. Although serum antibody titres to coronavirus can be measured, false positives are common due to cross-reactivity with antibodies to benign enteric coronaviruses. Cerebrospinal fluid analysis offers the best indication of the disease. Typically, the protein levels are extremely high (greater than 200 mg/dl) and the white cell count is raised with neutrophils being the predominant cell type.

There is no effective therapy for FIP and the prognosis in these cases is poor, especially when the fact that these cats often have concurrent infections with FeLV and/or FIV is taken into account.

Protozoal meningitis

Toxoplasma gondii and *Neosporum caninum* may infect animals and cause signs relating to many body systems, including those produced by a disseminated meningoencephalomyelitis. The clinical signs include spinal pain and muscle rigidity. Animals affected with these diseases also show pelvic limb hyperextension, reduced spinal reflexes and muscle pain/atrophy, and the presence of such signs leads to a high index of suspicion for this type of infection. Most cases are less than 4 months of age at onset. Confirmation of the diagnosis can be difficult. Assaying for antibodies to both these organisms in serum or CSF may provide evidence of infection. A fourfold rise in IgG antibody titres in samples taken 2 weeks apart or a single raised IgM titre are considered supportive of the clinical diagnosis. If the organism responsible can be identified in muscle biopsies, using electromyography to determine which muscles should be sampled, then the diagnosis is confirmed. The prognosis for animals showing neurological signs associated with either of these infections is poor. In those cases showing only myositis treatment with clindamycin (Antirobe, Pharmacia & Upjohn) at a dose of 40 mg/kg bodyweight/day in divided doses for 4–6 weeks is reasonably successful.

Bacterial meningitis

This is very uncommon but bacteria may enter the CSF by haematogenous spread from other sites of infection or iatrogenically (e.g. via spinal needles). The signs are as described above but other signs relating to the origin of the infection should also be noted. Cerebrospinal fluid analysis and culture are required to confirm the diagnosis, though negative cultures from CSF in cases with bacterial meningitis are not uncommon. Cerebrospinal fluid analysis should show raised protein levels and a high neutrophil count with cells showing degenerative changes and possibly evidence of phagocytosed bacteria. Whenever possible, treatment is based on culture and sensitivity results. If nothing is cultured then a broad-spectrum, bacteriocidal antibiotic that can cross the blood–brain barrier should be used. Favoured drugs for this role include chloramphenicol, trimethoprim-potentiated sulphonamides and cephalosporins. Other antibiotics, such as the penicillins, can be used in the early stages since they will cross the blood–brain barrier in the presence of inflammation. Treatment should continue for 2–4 weeks beyond the resolution of clinical signs. The prognosis is variable and depends on the severity and duration of the signs coupled with whether the organism responsible can be isolated and whether appropriate antibiotic treatment is given.

Spinal haemorrhage

Haemorrhage within the spinal cord, subarachnoid space or epidural space may occur as a result of trauma (fracture, disc extrusion), a clotting disorder, infections (e.g. toxoplasmosis or migrating parasitic disorders – one of the authors was presented with a dog showing acute onset hindlimb paralysis associated with extensive spinal cord

damage caused by infection with *Angiostrongylus vasorum* which was included in a series reported by Patteson *et al.* (1993)) or in association with neoplasia. If red blood cells are found on CSF analysis then it is likely they represent contamination from needle puncture unless there is also xanthochromia and/or evidence of phagocytosed erythrocytes. Plain radiographs will show no abnormalities and myelography may be normal or show various changes according to the location of the haemorrhage. Treatment involves attention to any underlying problem and the prognosis depends, to a large extent, on the cause and severity of the neurological signs.

Leucoencephalomalacia

This is a degenerative disease of unknown aetiology that affects only Rottweilers and has been reported in the USA, the Netherlands and Australia. Signs may become evident between the ages of 18 months and 4 years (reports thus far). A progressive hindlimb paresis and forelimb hypermetria develop over several months which is not dissimilar to the signs shown by middle-aged Dobermann Pinschers with cervical spondylopathy. The disease is non-painful. Cerebrospinal fluid analysis, plain radiography and myelography are all normal but help to rule out other causes of such clinical signs. Pathologically there is evidence of demyelination and cavitation, particularly in the cervical spine.

There is no treatment and the signs are progressive, making the prognosis hopeless. Most dogs with this disease will be destroyed within 1 year of showing signs. This outlook is different to that for neuroaxonal dystrophy (also affecting this breed) and so it is important to differentiate the two. Clinically they can be separated since leucoencephalomalacia causes proprioceptive deficits and neuroaxonal dystrophy does not, whereas the latter causes tremor when the former does not.

Neuroaxonal dystrophy

This is an idiopathic, degenerative condition of Rottweilers which is thought to have an autosomal recessive mode of inheritance. It has also been reported in cats. The dystrophy affects both the spinal cord and brain, including the cerebellum. Affected dogs show signs from a young age but these may not be severe enough for the owner to be concerned until the animal is adult. Hindlimb incoordination, without weakness or proprioceptive deficits, and forelimb hypermetria are seen coupled with signs related to the cerebellum (tremor, head incoordination, nystagmus) which may develop at a later stage. The condition is non-painful. Cerebrospinal fluid analysis, plain radiography and myelography are all normal but help to rule out other causes of such clinical signs.

There is no specific treatment and the prognosis has to be guarded although affected animals may make functional pets for several years.

Spondylosis deformans

This is a very common radiographic finding in older dogs, especially of certain breeds such as the Boxer, and is seen mainly in the thoracolumbar and lumbar regions. Spondyles develop ventral and lateral to the vertebral bodies and may eventually form a complete bridge between adjacent vertebrae, or even a continuous chain if more than one pair of vertebrae are involved (Fig. 29.4). It is thought that the spondyles form in response to stretching of Sharpey's fibres which arise from the periosteum of the vertebral bodies and blend with the annulus fibrosus of the intervertebral disc. Although such stretching might be indicative of degenerative pathology within the disc, the presence of spondylosis deformans alone is not diagnostic for clinical disease of the vertebral column. It is frequently noted in radiographs of dogs showing no signs relating to the spine, i.e. is clinically insignificant, no matter how dramatic the radiographic changes might appear. In very rare cases the spondyles might encroach on the intervertebral foraminae causing nerve root entrapment and clinical signs, but it cannot be overstressed that these cases are the exception rather than the rule. *In general, the finding of spondylosis deformans should be considered of no direct significance.*

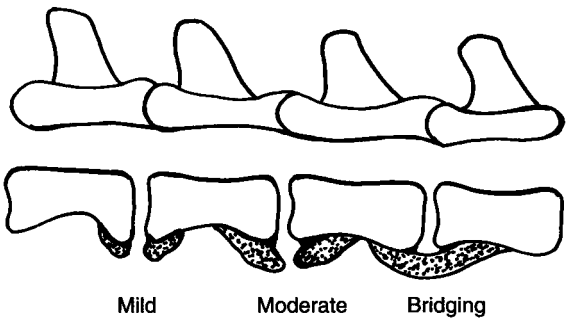

Fig. 29.4 Diagram illustrating the various forms of spondylosis deformans.

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Section 5
The Forelimb

Chapter 30
Examination and Differential Diagnosis of Forelimb Lameness

The examination of animals with lameness has been described in Chapter 6 (p. 41) and the conditions affecting each joint or bone of the forelimb are listed early in the relevant chapter of this section. Table 30.1 outlines the differential diagnosis of forelimb lameness according to whether the patient is skeletally immature or mature and whether the onset of lameness is acute or insidious. Details of each condition mentioned are to be found either in this section or elsewhere in the book. An overview of non-traumatic causes of forelimb lameness in the growing dog has been given by Scott (1998).

In addition to lameness, forelimb 'weakness' may result from other diseases and should be kept in mind, particularly if any neurological deficits are detected on examination. These would include:

- Conditions of the vertebral column (see Section 4), especially:
  - trauma
  - atlantoaxial subluxation
  - cervical disc disease
  - cervical spondylopathy
  - neoplasia

Table 30.1 Differential diagnosis of forelimb lameness.

Age of animal	Acute onset	Insidious onset

Skeletally immature (<1 year of age)	• Fractures (especially physeal and particularly lateral humeral condylar)
	• Panosteitis
	• Metaphyseal osteopathy
	• Injury to the foot
	• Traumatic luxation (uncommon as more likely to fracture physis)
	• Septic arthritis
		• Congenital luxation (especially elbow and shoulder)
		• Subluxation (especially elbow, or carpus, associated with antebrachial growth deformity)
		• Osteochondrosis:
		  ◦ shoulder – OCD
		  ◦ elbow – 'dysplasia' (OCD, FCP, UAP, UME)
		  ◦ sesamoid disease (?)
		• Immune-mediated polyarthritis

Skeletally mature	• Fractures • Traumatic luxation (carpus > elbow > shoulder) • Neoplasia (especially bone tumour with pathological fracture) • Injury to the foot • Intervertebral disc prolapse (causing nerve root entrapment) • Brachial plexus avulsion	• Osteoarthritis (elbow > carpus > shoulder) • Neoplasia (bone, periarticular soft tissues, brachial plexus) • Soft tissue injuries (spinatus contracture, rotator cuff tendon problems such as bicipital tenosynovitis) • Immune-mediated polyarthritis

OCD – osteochondritis dissecans; FCP – fragmentated/fissuing coronoid process; UAP – ununited anconeal process; UME – ununited medial epicondyle.

  - congenital anomalies (e.g. subarachnoid cysts)
  - fibrocartilagenous emboli
- Nutritional bone disease (see Chapter 47)
- Myopathies (see Chapter 49)
- Neuropathies (see Chapter 50)

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A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny,Steven J. Butterworth

Chapter 31
The Shoulder

Anatomy

The anatomy of the shoulder joint is illustrated in Figs 33.1–31.3. The scapulohumeral joint is an enarthrodial joint formed by the articulation of the scapular glenoid with the humeral head. The joint capsule is attached to both the rim of the glenoid and the humeral head but has several out-pouchings. The most significant of these is the caudal pouch and its cranial extension which forms a tendon sheath around the tendon of the biceps brachii, as it passes through the intertubercular groove (under the intertubercular ligament), before forming a pouch distal to this groove. Within the joint capsule are lateral and medial thickenings which make up the glenohumeral ligaments. These ligaments are relatively weak and the joint is also supported by several 'cuff' muscles. The supraspinatus and infraspinatus muscles arise from the scapular blade, cranial and caudal to the scapular spine respectively, and insert on the cranial and lateral aspects of the greater tubercle respectively. They provide cranial and lateral support for the joint. Medial support is provided by the subscapularis muscle which arises from the scapular blade and inserts on the lesser tubercle. The biceps brachii muscle arises from the scapular tuberosity and passes through the intertubercular groove, underneath the intertubercular ligament, thus providing craniomedial support. The tendons of all these muscles have very close associations with the joint capsule. In addition, there are several other muscles (e.g. the deltoids, teres minor and major, and coracobrachialis) which lend some support to the joint without such intimate contact with the capsule. An *in vitro* analysis of shoulder stability (Vasseur *et al.*, 1982) showed that removal of all the 'cuff' muscles had little effect on the static stability of the joint, although these were thought to serve an important function *in vivo*, and that it was necessary to damage the capsule itself before increased laxity could be detected.

Neurovascular structures to be borne in mind when contemplating surgery in the shoulder region are few but comprise:

- The omobrachial vein (a branch of the cephalic vein) which passes across the craniolateral aspect of the joint
- The caudal circumflex artery and vein and the axillary nerve which cross the caudal aspect of the joint capsule
- The suprascapular nerve which courses from cranial to caudal under the tendons of insertion of the suprascapular muscles and the acromion of the scapula

In feline patients it is important to bear in mind the differences, compared to the canine, in their osteology. The metacromion is located on the distal scapular spine and extends caudally, the coracoid process forms a prominent extension from the rim of the glenoid craniomedially and a clavicle is present (Fig. 31.4).

Conditions of the shoulder joint

Conditions of the shoulder can be divided into two groups:

- Developmental
  - Osteochondrosis
  - Congenital luxation of the scapulohumeral joint
  - Shoulder dysplasia

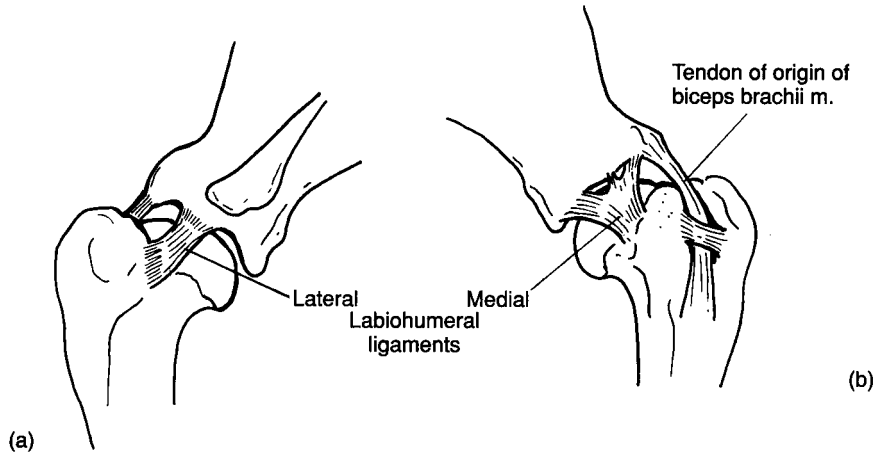


Fig. 31.1 Shoulder joint. (a) Lateral view, (b) medial view.

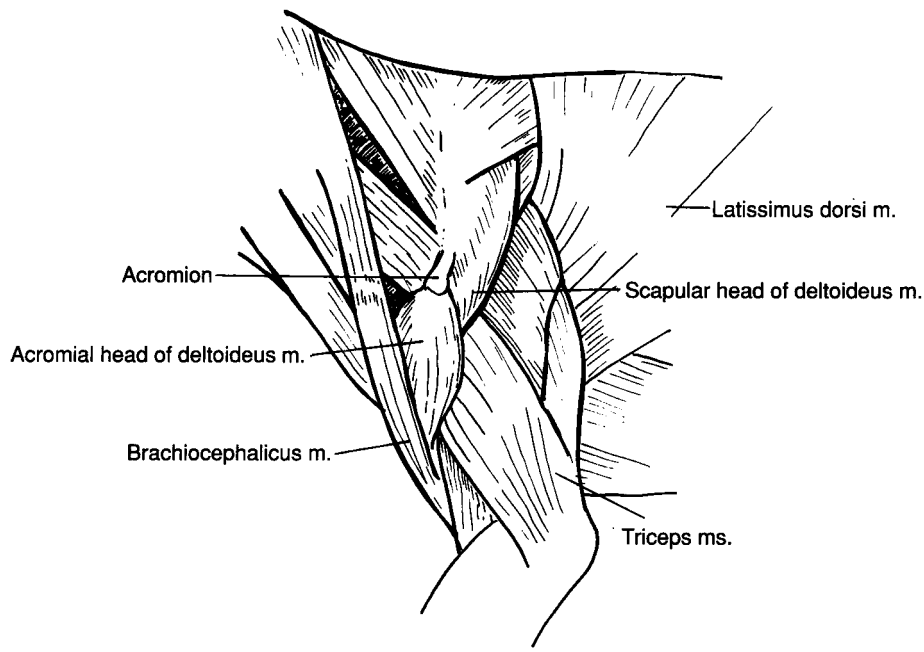


Fig. 31.2 Superficial muscles of the lateral shoulder.

- Acquired
  - Fractures of the scapula
  - Fractures of the proximal humerus (see Chapter 32)
  - Traumatic luxation of the scapulohumeral joint
  - Dorsal luxation of the scapula
  - Contracture of the infraspinatus (or supraspinatus) muscle
  - Bicipital tenosynovitis
  - Displacement of the biceps brachii tendon
  - Rupture of the biceps brachii tendon

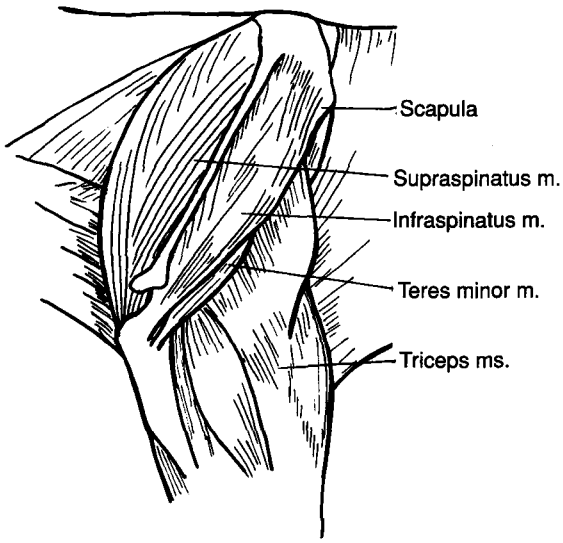


Fig. 31.3 Deep muscles of the lateral shoulder.

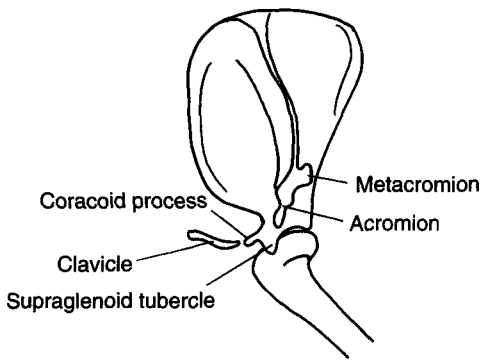


Fig. 31.4 Shoulder joint of the cat – lateral view.

- Osteoarthritis
- Neoplasia (osteosarcoma; synovial sarcoma; chondrosarcoma; fibrosarcoma; brachial plexus tumours)

Osteochondrosis

Osteochondrosis is an idiopathic condition in which there is a disturbance in normal enchondral ossification (see Chapter 4). In the shoulder joint it is associated with the development of thickened articular cartilage which is particularly prone to trauma with chondromalacia of the deeper layers and cleft formation between the cartilage and subchondral bone. Subsequently, the under-run cartilage may split vertically causing flap formation whereupon the term osteochondritis dissecans (OCD) becomes appropriate. In general it is the articular cartilage covering the medial aspect of the caudal third of the humeral head which is involved although, uncommonly, the caudal rim of the glenoid may be implicated. If such a flap is formed it may become mineralised and thus visible radiographically. In addition, it may break free and form a 'joint mouse' which can absorb nutrients from the synovial fluid and grow. It may also become mineralised. Once the flap has become detached the defect may fill in with granulation tissue which is then converted into fibrocartilage.

The condition shows a breed predisposition with the giant breeds, such as the Great Dane, the Pyrenean Mountain Dog and the Irish Wolfhound, being overrepresented. However, medium-sized breeds, such as the Labrador Retriever, the Golden Retriever and the Burmese Mountain Dog, are also commonly seen with the problem and smaller breeds, such as the Border Collie, are sometimes affected. Although the ratio of males to females varies between reports there is a general consensus that more males are affected clinically, with one large retrospective study (Rudd *et al.*, 1990) giving a male:female ratio of 2.24:1. In clinical cases the condition is found to be radiographically bilateral in just over 50% of cases.

History

The lameness usually begins when the dog is between 4 and 7 months of age, although the owners may not present the patient to a veterinary surgeon for a period of time because the onset is somewhat insidious. A number of cases, estimated as 36% in the study by Rudd *et al.* (1990), may be over 1 year of age at the time of diagnosis and so the condition should not be excluded from the list of differential diagnoses for shoulder lameness simply because a dog is over 1 year old. The degree of lameness varies between cases but is usually mild to moderate. On

questioning, the owner will usually report that the lameness deteriorates with exercise and that there is stiffness after rest, particularly following exercise. Restriction of exercise tends to improve the lameness.

Clinical signs

Observation of the dog at the walk and trot will tend to show a weight-bearing lameness. If both shoulders are clinically affected then the dog may show more of a stiff, shuffling forelimb gait rather than an overt lameness. On palpation there may be evidence of disuse atrophy, especially of the spinatus muscles which makes the scapular spine more prominent. Manipulation of the shoulder should produce a pain response, particularly on extension of the joint. A full clinical examination is required to detect any concurrent problems, which might affect the prognosis, or bilateral shoulder involvement.

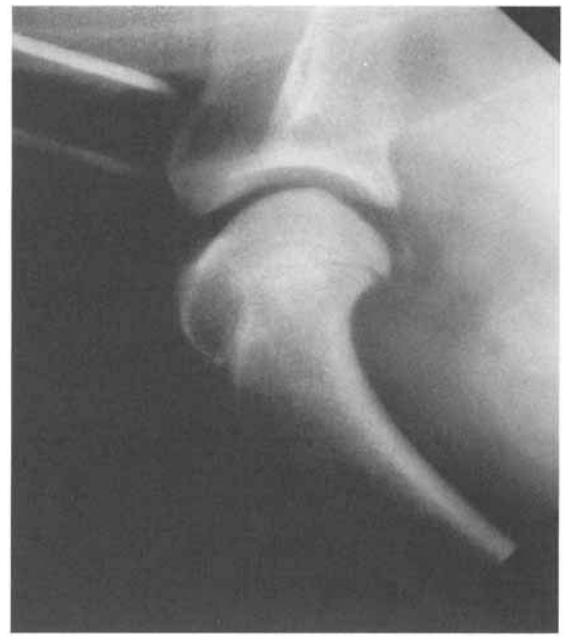


Fig. 31.5 Mediolateral radiograph of an 11-month-old Border Collie's shoulder. The caudal humeral head appears flattened and a mineralised cartilage flap is evident in the caudal joint space (not always seen). (Reproduced from *The Manual of Small Animal Arthrology* with permission from BSAVA Publications.)

Diagnosis

A definitive diagnosis is generally reached by means of radiography. A mediolateral projection of the extended shoulder will usually suffice but occasionally inwardly and/or outwardly rotated views may prove necessary to skyline the lesion. The changes that may be observed on plain radiographs in cases involving the humeral head include (Fig. 31.5):

- A subchondral defect with flattening of the caudal humeral head
- A sclerotic margin to any such defect
- The presence of a cartilage flap (only visible if mineralised)
- The presence of 'joint mice' (only visible if mineralised), most commonly found in the caudal recess of the joint
- Secondary degenerative joint disease, most often seen as osteophyte formation on the caudal borders of the glenoid and/or the humeral head

It is always worth radiographing both shoulders as the problem is often bilateral. This may be helpful in cases that have suffered subclinical osteochondrosis in one shoulder and then developed a clinical lameness due to the same problem in the second limb. The changes in the latter may not yet have developed or be quite subtle, and finding changes in the 'normal' limb helps to reinforce the clinical picture with respect to diagnosis.

In addition to these changes, van Bree (1992) has described the 'vacuum phenomenon' in shoulder osteochondrosis. This refers to the appearance of gaseous collections in the articular space creating an image resembling a negative arthrogram. This change was noted in 20 of 100 radiographs of shoulders affected by osteochondrosis and never in 30 radiographically normal contralateral joints. Nor could it be induced in 36 normal shoulder joints radiographed in full extension. The phenomenon is found in addition to other radiographic changes and so should be added to the list of other possible findings rather than being considered as a separate entity.

Positive contrast arthrography, using a low volume (1.5–2.0 ml) of a 25% w/v solution of meglumine and sodium diatrizoate (Urografin, Schering-Plough Animal Health) or iopamidol

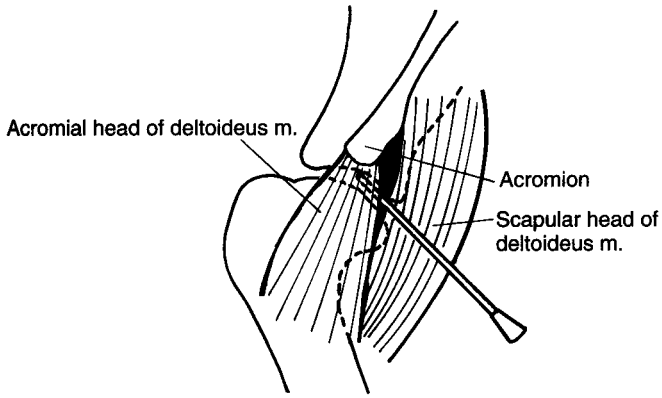


Fig. 31.6 Diagram showing placement of a needle into the shoulder for arthrography. (Source: BSAVA Publications, Cheltenham.)

at 300mg I_2/ml (Omnipaque, Nycomed) diluted 50:50 with sterile water for injection, may help to assess whether a non-mineralised cartilage flap or joint mouse is present. van Bree (1992) reported that using iopromide (Ultravist, Schering-Plough Animal Health) gave better arthrographic quality when compared to meglumine-sodium diatrizoate.

The technique is fairly simple and involves placement of a 1–2″ × 19g or 20g needle into the joint under strict asepsis. The needle enters the joint just caudal and 1cm distal to the acromion at a point where finger pressure can often detect the division between the acromial and spinous heads of the deltoideus muscle (Fig. 31.6). The needle is advanced into the joint whereupon synovial fluid may be aspirated although the volume varies from a few drops to a few millilitres. It should always be possible to obtain sufficient fluid to be convinced that the needle has been placed correctly. The contrast agent is then introduced, the needle withdrawn and the shoulder manipulated to encourage even distribution of the contrast through the joint (Fig. 31.7).

Although arthrography may provide useful information with respect to location of joint mice, the diagnosis can almost always be made from the plain radiographs. Some reports have suggested that the findings might help to determine whether a case should be treated conservatively or surgically. To date there are no clear data to suggest that arthrography can predict the outcome in a joint causing clinical problems.

In the case of bilateral osteochondrosis arthrography has been shown (van Bree, 1992) to be of some use in predicting the likely outcome for the shoulder showing radiographic signs but no clinical lameness at the time of initial

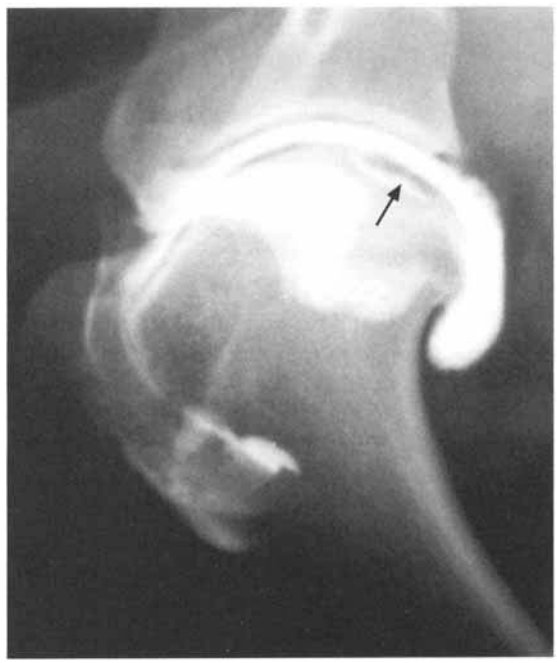


Fig. 31.7 Mediolateral arthrogram of an 11-month-old Great Dane's shoulder. A low dose of contrast is required otherwise the articular surface of interest becomes obliterated. A line of contrast is visible (arrow), below the surface of the articular cartilage and confirming the presence of an osteochondritis dissecans (OCD) flap.

diagnosis following surgical treatment of the lame shoulder. Of those dogs with a detectable, loose cartilage flap in the contralateral joint, 50% became lame at a later date and required surgery on that limb. Of those with no cartilage flap or else one that had detached and fallen into the caudal pouch of the joint, none required surgery on the contralateral limb. It is still questionable whether this information helps very much since a 'wait-and-see' policy with respect to the second limb would produce the same long-term results! Finally, arthroscopy may be used to visualise the articular surface and may detect fissures in the cartilage that are not evident on arthrography.

Treatment and prognosis

Conservative management

In some cases the lameness will resolve with 6–8 weeks of controlled (lead) exercise and so this may be well worth trying in the first instance. If such measures are continued then the majority of cases will eventually become sound but this may take several months and the resulting secondary osteoarthritis may be more pronounced. Whilst these dogs are being rested it may be necessary to prescribe a non-steroidal anti-inflammatory drug (NSAID) to prevent excessive discomfort. Some authors advocate a regime of vigorous exercise for these animals with the aim of promoting detachment of the cartilage flap and resolution of the lameness once the subchondral defect has filled in with fibrocartilage. The results following conservative measures are poorly documented but in one report by Vaughan & Jones (1968) of 22 dogs treated in this way only 12 became sound. The use of polysulphated glycosaminoglycans preparations such a sodium pentosan polysulphate (Cartrophen-Vet, Arthropharm, Australia) has not been documented as leading to an improvement in results over and above restricted exercise alone.

Surgical management

If a positive diagnosis has been reached radiographically and 6–8 weeks of conservative management have failed to allow resolution of the clinical signs then surgery is indicated. This is to be advocated over further conservative management because the success rate is high and the period of convalescence relatively short. Clayton Jones & Vaughan (1970) reported that, following surgery, 28 of 29 dogs recovered full use of the limb with no lameness whilst Rudd *et al.* (1990) found that 30 out of 40 dogs treated surgically became sound.

A caudal approach to the joint provides good exposure of the affected portion of the humeral head (Fig. 31.8). Using the scapular spine, the acromion process and the greater tubercle as landmarks a skin incision is made (Fig. 31.8a) from halfway along the scapular spine, caudal to the acromion, curving distally to end about one-third of the way down the humerus. After dissection of the subcutaneous tissue the two heads of the deltoideus muscle are identified (Fig. 31.8b), separated by blunt dissection and retracted. The teres minor muscle is identified along with a neurovascular bundle just caudal to it (made up of the cephalic vein, muscular branch of the axillary nerve and the caudal circumflex humeral vessels) (Fig. 31.8c). The muscle is retracted cranially and the nerves and vessels carefully dissected off the joint capsule and then pushed, or retracted, caudally. An incision is made through the joint capsule to expose the caudal humeral head (Fig. 31.8d).

There are several variations regarding this approach to the joint. Having separated the deltoids it is possible to gain access to the joint by blunt dissection between the teres minor and infraspinatus muscles (and thus avoid being close to the nerves and vessels mentioned above). Alternatively, exposure of the joint capsule may be achieved by separating the spinous head of the deltoids and the lateral head of the triceps. These both give satisfactory exposure but the first is slightly more limited and the second creates a 'deeper' approach requiring retraction of larger muscle masses.

Exposure is improved by inward rotation of the joint and by the use of Hohmann retractors. The latter must be introduced with some care to avoid damage to the normal articular cartilage and also so as not to push the partially detached flap of cartilage into the medial part of the joint space, from which retrieval can prove difficult. The cartilage flap (Fig. 31.9) is detached and removed, with the

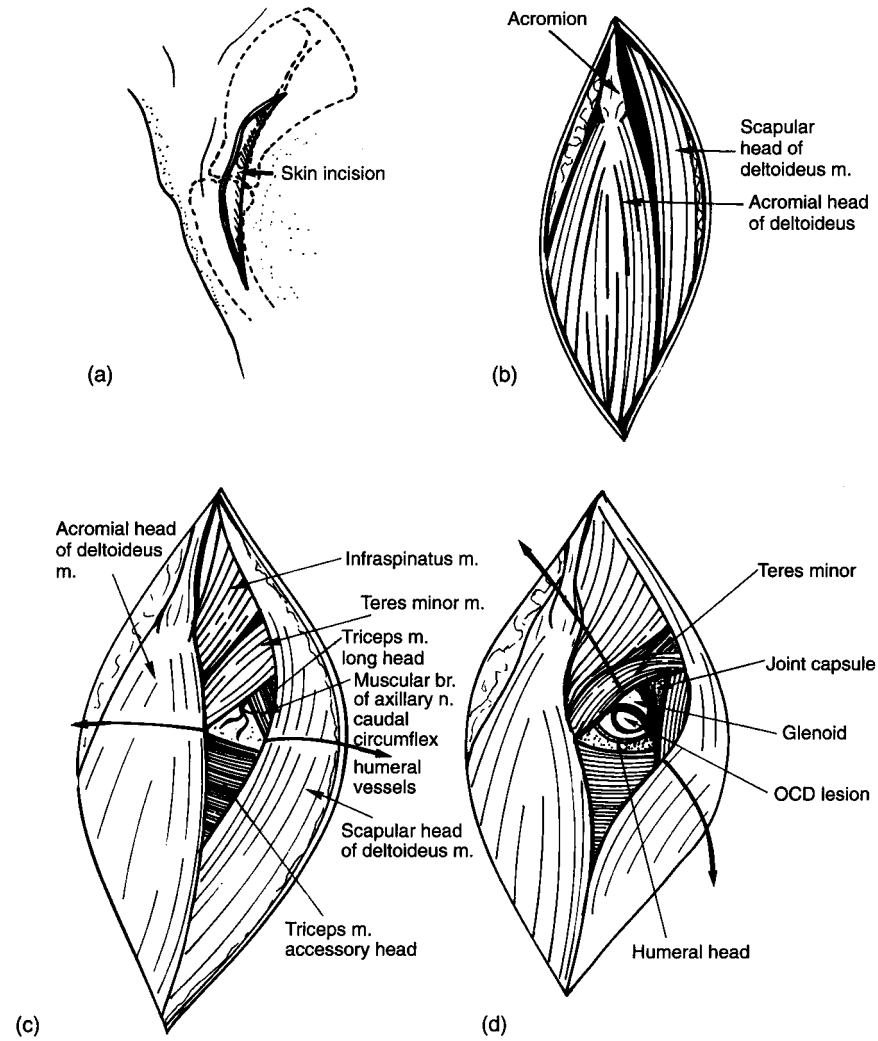


Fig. 31.8 Caudal approach to the shoulder joint. See text for details. (Source: BSAVA Publications, Cheltenham.)

help of forceps and/or an OCD curette, and the edges of the lesion curetted to remove any loose cartilage. Prior to closure the joint is flushed thoroughly to remove any remaining debris. A single suture, of absorbable material (Monocryl or Vicryl, Ethicon) may be placed in the joint capsule. The heads of the deltoids are re-apposed with the same material and the remainder of the closure is routine.

Arthroscopy has been advocated in some reports as being less invasive than arthrotomy and thus resulting in less postoperative lameness whilst achieving the same long-term results (Van Ryssen *et al.*, 1993). The disadvantages include the need to invest in the required instrumentation, to gain experience in the technique and also that if the cartilage flap remains within the joint it may form a joint mouse and cause further, intermittent lameness in the future. For these reasons, surgical intervention in most centres involves a caudolateral arthrotomy.

A figure-of-eight bandage may be placed over the wound to provide some comfort and also reduce the likelihood of seroma formation. The

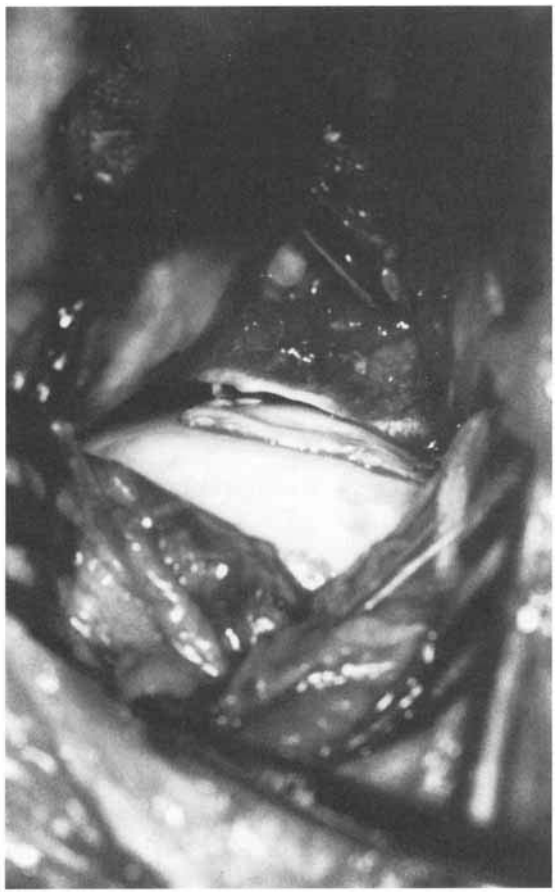


Fig. 31.9 Intraoperative view of an osteochondritis dissecans (OCD) flap sitting on the caudal humeral head. (Reproduced from *The Manual of Small Animal Arthrology* with permission from BSAVA Publications.)

latter can also be avoided by attention to closure of the tissue layers without leaving 'dead space', in which case it is possible to leave the site unbandaged. Any such bandage is removed after 5–7 days and the skin sutures 10 days postoperatively. The dog should be rested (restricted to one room at any one time and, when taken out for walks, should be restricted by a leash, even if in the garden) for 4–6 weeks. After this his/her exercise may be gradually increased if progress is satisfactory.

The majority of cases will be sound by 6–8 weeks after surgery but some, seemingly those with very large lesions, have been known to take up to 4 or 5 months to become sound.

If the problem is causing a bilateral lameness then the second limb may be treated surgically 6–8 weeks after the first although in many instances the contralateral lameness appears to resolve during the period of convalescence that follows the initial surgery, possibly because there is increased weight-bearing on the limb which may encourage detachment of the cartilage flap.

Congenital luxation of the scapulohumeral joint

Although it must be remembered that this condition is rare, there does appear to be a breed predisposition with Toy Poodles and Shetland Sheepdogs being overrepresented alongside some of the other toy breeds.

History and clinical signs

The dogs will usually be presented for recurrent lameness which develops at between 3 and 10 months of age. Occasionally these cases will present after reaching skeletal maturity following minor trauma. They characteristically adopt a begging posture with the joint held partially flexed. In some cases the problem is bilateral and these dogs may be found to have much better developed hindquarters or even a tendency to try and walk upright on their hindlimbs. The direction of luxation is invariably medial and this can easily be appreciated on clinical manipulation and palpation, when the acromion is much more prominent owing to its lateral displacement.

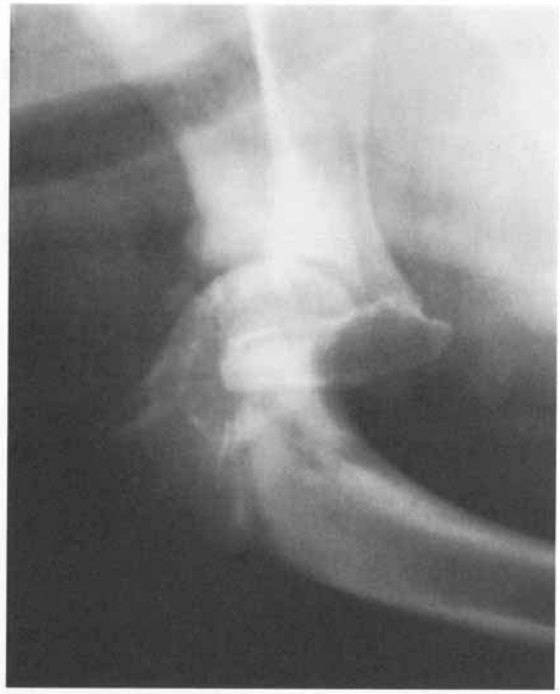
Diagnosis

Mediolateral and caudocranial radiographs should serve to show changes that are characteristic of this condition (Fig. 31.10), namely:

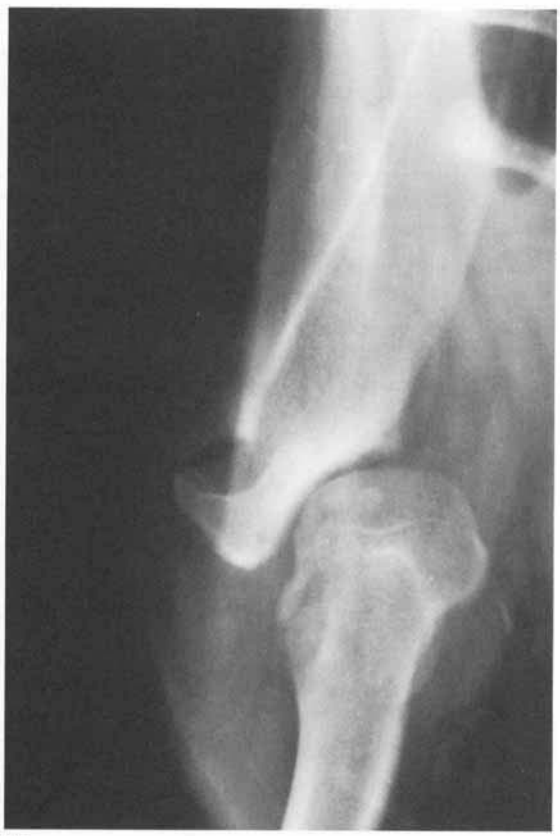
- Medial luxation
- A flattened or convex glenoid
- A relatively large and flattened humeral head

Treatment and prognosis

This type of luxation is not amenable to reduction because of the misshapen epiphyses. Attempts to



(a)



(b)

try and stabilise the joint are prone to failure due to this inherent incongruity. As a result, conservative measures are advisable. The joint tends to stabilise and the degree of lameness/incapacity improves as the dog matures. In general, it may be anticipated that, by 1 year of age, these dogs will be somewhat disabled but pain free and able to lead normal lives. If lameness persists then salvage procedures such as excision arthroplasty or arthrodesis might need to be considered.

Shoulder dysplasia

Shoulder dysplasia is a condition described as excessive joint laxity and has been documented as a cause of lameness in two single case reports, one involving a 3.5-year-old Collie and the other a 10-month-old Labrador. From such scant reports it is to be expected that, as a condition, it is quite rare. However, the authors have seen several dogs of chondrodystrophic breeds, most notably Basset Hounds, presented for non-specific forelimb lameness with mild discomfort on shoulder extension. Radiographically they were found to have shallow glenoids and flattened humeral heads, as expected for the breed (Fig. 31.11). No other abnormality could be found clinically or radiographically. The lameness was successfully treated by conservative means and, as in the case of immature dogs, improvement seemed to occur as they reached skeletal maturity. Obviously this observation cannot establish shoulder dysplasia as a cause of forelimb lameness in these dogs but all other likely diagnoses were ruled out as far as possible. Excessive laxity in the shoulder which in the immature patients improved as the surrounding musculature matured (as in many cases showing hindlimb lameness due to hip dysplasia) might well explain the clinical signs in such cases.

Fig. 31.10 Mediolateral (a) and caudocranial (b) radiographs of the shoulder of a 9-month-old Keeshound showing medial congenital luxation. There is gross deformity of the articular surfaces making the joint inherently unstable. (Courtesy of C. Gibbs.)

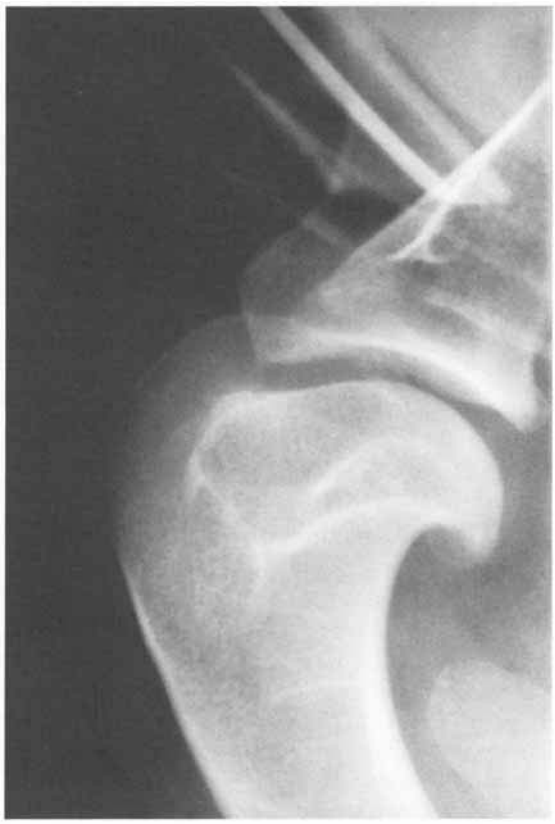


Fig. 31.11 Mediolateral radiograph of the left shoulder of a 3-year-old Basset Hound which showed intermittent lameness associated with shoulder pain. The glenoid appears shallow and the humeral head flattened. (Reproduced from *The Manual of Small Animal Arthrology* with permission from BSAVA Publications.)

Fractures of the scapula

Fractures of the scapula, and proximal humerus, are almost invariably a result of severe trauma, usually in the form of a road traffic accident. Several reports of the relationship between thoracic trauma and appendicular fractures have shown no correlation with whether the pectoral or pelvic limbs were involved or whether the fractures affected proximal or distal parts of the limbs. However, there has been some evidence to suggest a high incidence of thoracic injury in cases with scapular fractures (Spackman *et al.*, 1984) and in two recent reviews of scapular fractures (Harari & Dunning, 1993; Johnston, 1993) 7/12 and 8/26 cases were found to have concurrent thoracic injuries. As a result it is of paramount importance (as with all fractures) that the possibility of thoracic injury is investigated prior to contemplating treatment of the fracture itself.

In many cases of scapular fracture the fragments are not grossly displaced because of the splinting effect of the surrounding muscle masses and the rib cage (Fig. 31.12). In general they may be treated conservatively unless there is:

- A loss of joint congruity
- A distinct change in the angle of the joint articulation

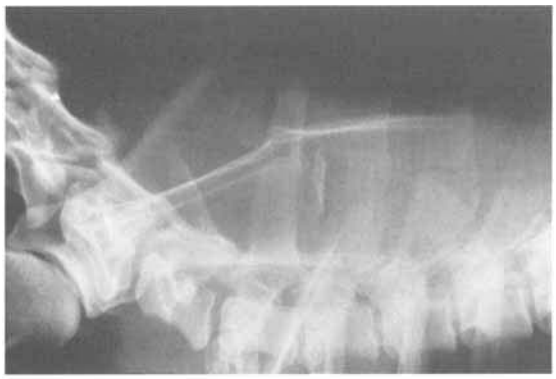
Conservative management

Conservative management may comprise nothing more than strict rest, with or without the tactical use of NSAIDs, if there is severe discomfort, for 3–6 weeks. Supporting the limb in a Velpeau or non-weight-bearing sling (see under 'Traumatic luxation of the scapulohumeral joint', p. 320) during this period may provide greater comfort for the patient.

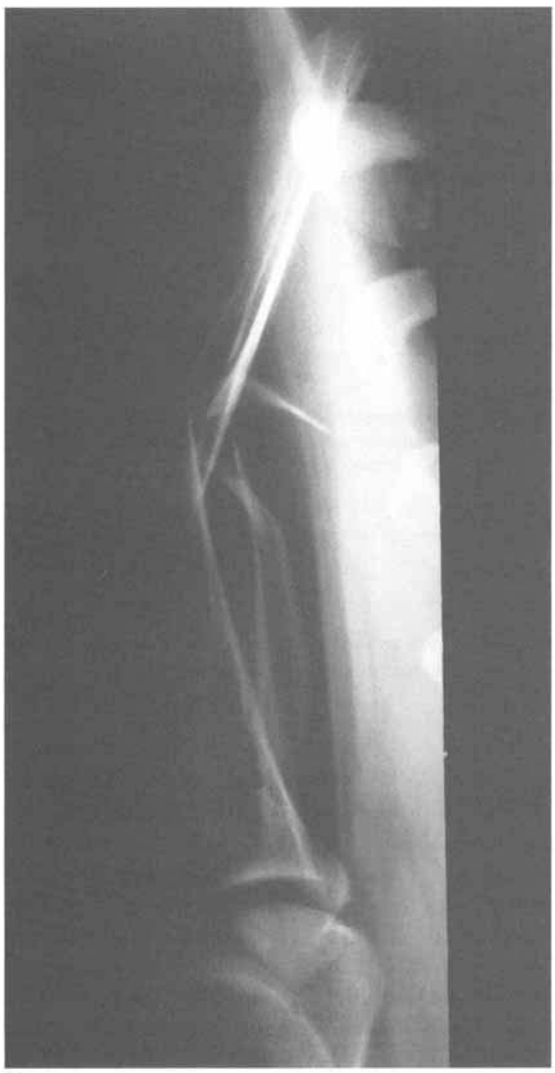
Surgical management

Fractures of the scapular blade or spine

Surgical management of such fractures requires a lateral approach (Fig. 31.13a). The skin incision is made directly over the scapular spine, extending distally over the acromial process and continuing distally along the craniolateral aspect of the humerus. The deep fascia is incised over the scapular spine, which releases the insertions of the omotransversarius and trapezius muscles cranially and the origin of the scapular head of the deltoideus muscle caudally, thus exposing the supraspinatus and infraspinatus muscles. This incision is continued distally, towards the omobrachial vein, to expose the acromial head of the deltoideus muscle. Exposure of the scapular spine, blade and neck can now be achieved by appropriate elevation of the spinatus muscles. In the case of the scapular neck, exposure can be improved by either tenotomy of the acromial head of the deltoideus muscle or osteotomy of the



(a)



(b)

acromion. During closure, repair would require tendon sutures, in the case of tenotomy, or reattachment of the acromion using either Kirschner wires and a tension band or else wire sutures, in the case of osteotomy. Exposure of the scapular neck could also be improved by tenotomy of the insertion of the infraspinatus muscle.

Stabilisation may be achieved using wire sutures, particularly in vertical fractures, or plates, particularly in cases with horizontal fractures (Fig. 31.14). In the case of wire sutures it is best to preplace the sutures before tightening them in turn, otherwise placement becomes progressively more difficult. In the case of plates, the use of a semitubular plate (Synthes) with its convex surface facing into the angle between the scapular spine and blade helps to provide the bone screws with better purchase.

In some cases the fracture line is found to extend into the joint itself. The surgical approach required is lateral but exposure may be improved by osteotomy of the acromion (Fig. 31.13b) and/or the greater tubercle. During reduction of the fragments it is important to pay attention to re-establishing joint congruity. In many cases it is possible to use a bone screw, in a lagged fashion, close to the joint to create compression and maintain correct anatomical alignment (Fig. 31.15).

Fractures of the acromion

These fractures involve the origin of the acromial head of the deltoideus muscle and, if the fragment is large enough to support the placement of implants, surgery is indicated otherwise healing may be impaired by the constant traction at the fracture site. A direct, lateral approach is required

Fig. 31.12 Scapular fracture in an 18-month-old Dobermann Pinscher. Mediolateral (a) and caudocranial (b) radiographs of the right scapula following a road traffic accident. A comminuted fracture of the blade is present but without articular involvement and only minor displacement. Treatment comprised strict rest for 6 weeks by which time the dog was sound, having been using the limb well since 7 days after the accident. (Source: BSAVA Publications, Cheltenham.)

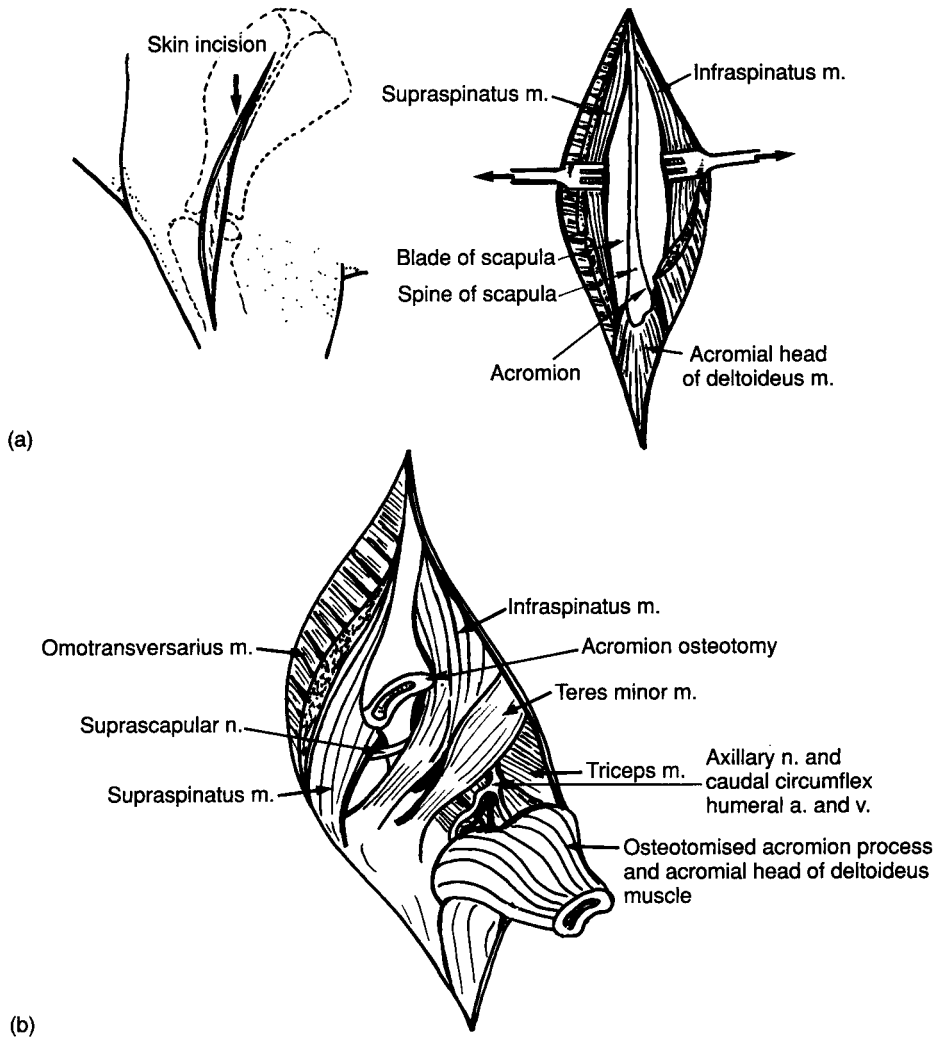


Fig. 31.13 Lateral approach to the shoulder. (a) Exposure of the scapular blade and spine. (b) Exposure of the scapular neck (osteotomy of the acromion, or tenotomy of the acromial head of the deltoideus muscle, may be necessary and is shown here for clarity).

to expose the fracture. Some authors advocate the use of a tension band technique to stabilise these fractures but placement of such implants can be quite difficult and the use of one or two wire sutures is adequate (Fig. 31.16).

Fractures of the scapular neck

The approach required to expose these fractures may involve osteotomy of the acromion (Fig. 31.13b and details given under 'Fractures of the scapular blade or spine', above). Care should be taken to preserve the suprascapular nerve. In some cases the fracture may be found to take the form of a 'T' with involvement of the articular surface. In such cases the two halves of the glenoid should be stabilised, preferably using a bone screw in a lagged fashion, before the scapular neck itself is stabilised using a bone plate (Fig. 31.17).

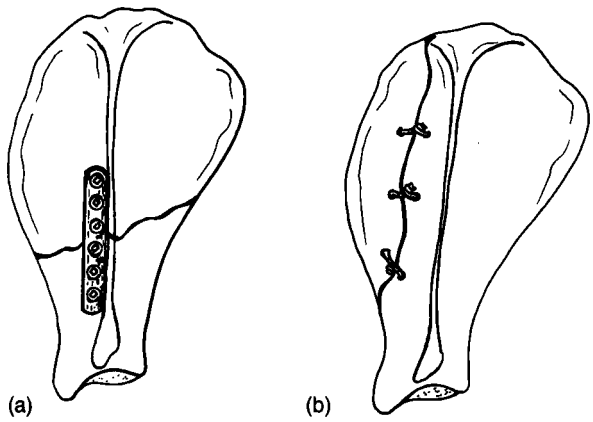


Fig. 31.14 Diagram illustrating the use of (a) a plate to stabilise a horizontal fracture of the scapular blade and (b) wires to stabilise a vertical fracture of the scapular blade.

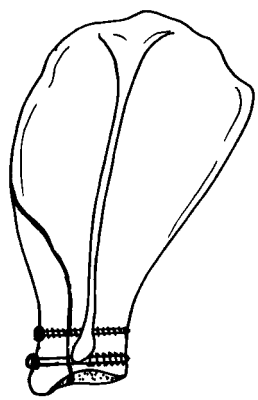


Fig. 31.15 Diagram illustrating the use of lagged bone screws to stabilise scapular fractures that extend to the joint surface.

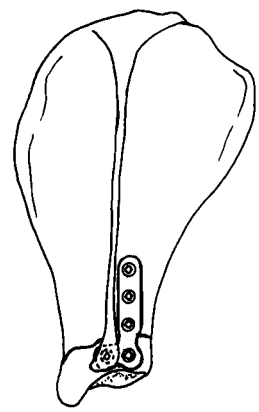


Fig. 31.17 Diagram illustrating the use of an oblique 'L'-plate to stabilise a fracture of the scapular neck.

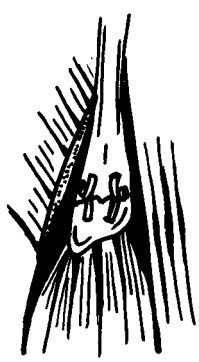


Fig. 31.16 Diagram illustrating the use of wires to stabilise an acromial fracture or osteotomy.

Fracture of the scapular tuberosity

This tuberosity is the site of origin of the biceps brachii muscle and develops as a separate centre of ossification, normally fusing on to the scapula by 5 months of age. Although fracture is uncommon, it usually occurs in immature patients with separation through the physis. Healing may be complicated by the distractive forces of the biceps muscle. If the fragment is large enough to accommodate implants then surgery is to be recommended. If it is not, then conservative measures may be adopted (refer to 'Rupture/avulsion of the biceps brachii tendon of origin' later in this chapter, p. 330). If such measures fail to allow

resolution of the lameness, then surgery may be carried out to remove the fragment and origin of the biceps after securing the biceps tendon to the proximal humerus as described later in this chapter under 'Bicipital tenosynovitis' (p. 327). Similar measures may need to be employed in cases with long-standing fracture of the tuberosity when open reduction may prove impossible due to biceps contracture.

Open reduction and stabilisation requires a cranial approach to the shoulder (Fig. 31.18). With the dog in dorsal recumbency and the limb drawn caudally, a skin incision is made from a point just medial and caudal to the acromion, passing distally over the craniomedial aspect of the humerus (Fig. 31.18a,b). Dissection through, and retraction of, the subcutaneous tissue exposes the brachiocephalicus muscle. The fascia along its lateral border is incised (+/– ligation of the omobrachial vein) to allow retraction of the muscle and exposure of the superficial pectoral muscle. The latter's insertion is freed from the humerus, exposing the insertion of the deep pectoral muscle which is elevated in a similar manner. The biceps brachii

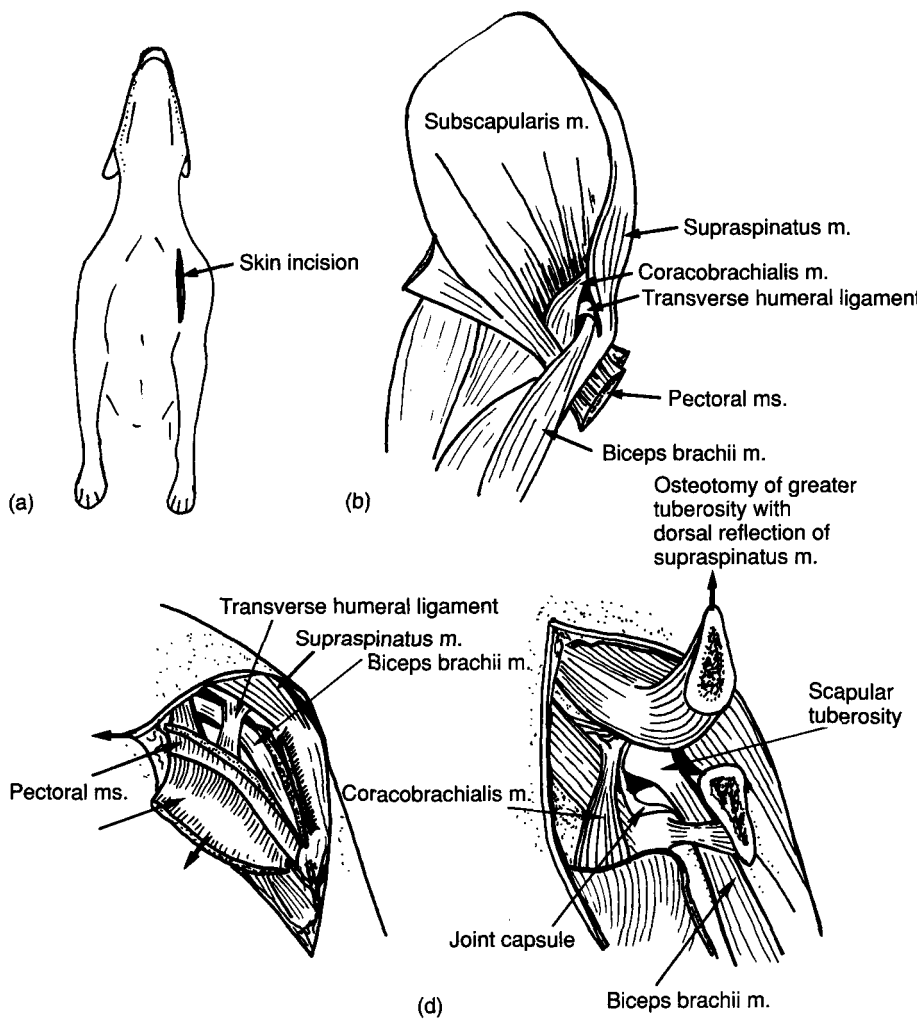


Fig. 31.18 Cranial approach to the shoulder. See text for detail.

muscle and tendon of origin will be exposed (Fig. 31.18c) and this can be followed proximally to the transverse humeral ligament and the scapular tuberosity. Exposure of the latter can be improved by osteotomy of the greater tubercle, allowing reflection of the supraspinatus muscle, followed by an incision through the joint capsule (Fig. 31.18d). The greater tubercle must be reattached, during closure, using crossed Kirschner wires, a lagged bone screw or pins and a tension band wire. Stabilisation of the fracture may be achieved using a pin and tension band wire but the use of a lagged bone screw is often simpler and just as effective with retrograde drilling of the tuberosity allowing more accurate placement of the implant (Figs 31.19 and 31.20).

'Chip fractures'

In some cases, small fragments originating from the rim of the glenoid will be seen radiographically. In general these may be treated conservatively, as described above, but if lameness persists beyond 6–8 weeks post-trauma then surgical removal of the fragment should be considered.

Postoperative care for fractures of the scapula

A pressure bandage (figure-of-eight) may be used to help reduce postoperative swelling or, particularly in cases with scapular blade or neck fractures, some benefit may be gained from placing the limb in a sling. Such dressings are left in place for between 5 and 7 days. The patient should be strictly rested for between 4 and 6 weeks, depending on progress, and then their exercise gradually increased. Unless there are good reasons for not doing so, the implants are left *in situ*.

Prognosis

Most scapular fractures show minimal displacement and do not interfere with scapulohumeral joint function. The majority of such cases will regain normal limb function with conservative management. In those where surgery is indicated because of joint involvement, the outlook is more guarded. Despite most dogs regaining limb function after prolonged recovery periods a significant degree of lameness will be noted in about half of these (Johnston, 1993).

Fractures of the proximal humerus

These fractures are discussed in Chapter 32.

Traumatic luxation of the scapulohumeral joint

Luxation of the shoulder is an *uncommon* condition that may be overdiagnosed because, in looking for an explanation for a forelimb lameness, an audible click may be detected on manipulation of the normal shoulder joint followed

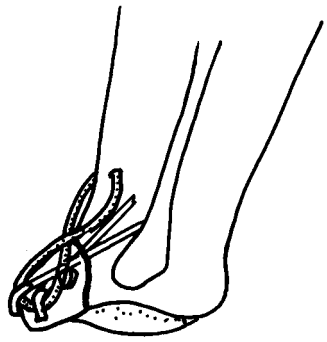


Fig. 31.19 Diagram illustrating the use of Kirschner wires and a tension band wire to stabilise a fracture of the scapular tuberosity.

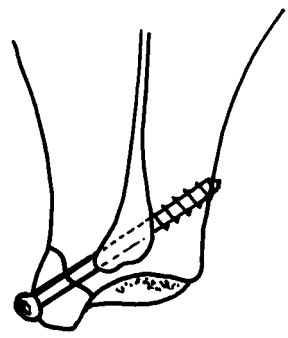


Fig. 31.20 Diagram illustrating the use of a lagged bone screw to stabilise a fracture of the scapular tuberosity.

by the declaration that 'his/her shoulder was out but it's just popped back in now!'. Scapulohumeral luxation may have a congenital or traumatic aetiology and causes signs which apply to all luxations:

- Loss of joint function
- A decreased range of joint movement
- Pain on manipulation which decreases as the condition becomes chronic

Except in cases where a congenital problem predisposes to luxation as a result of minor trauma, acquired luxations are usually associated with a fall or knock, particularly when this occurs whilst the dog is turning at speed. The direction of luxation is generally lateral or medial. It is often said that the smaller breeds tend to suffer medial luxation whilst lateral luxations are seen more in the larger breeds. Taking all the reported series of cases as a whole there is little to put between the incidence of luxation in these two directions and it is probably safer to take each case on its own merit. If a definite direction of luxation cannot be established clinically or radiographically it is better to treat the problem as if it could be either rather than guessing. Cranial and caudal luxations are rarely encountered. In the case of the former there may be an association with rupture of the transverse humeral ligament allowing displacement of the biceps brachii tendon and loss of cranial support (see p. 329)

History and clinical signs

Affected dogs will be presented with a history of acute-onset, severe (usually non-weight-bearing) forelimb lameness as a consequence of moderate to severe trauma. Patient *observation* will show the elbow to be held flexed and adducted with the distal limb held abducted in cases of medial luxation and adducted if the luxation is in a lateral direction. On *palpation*, the acromion and greater tubercle may be positioned abnormally relative to one another when compared to the contralateral limb. In more long-standing cases, with recurrent luxation that reduces spontaneously causing an intermittent lameness, muscle atrophy may be noted. *Manipulation* will reveal a reduced range of movement and pain on extension or flexion. In some cases the luxation will reduce spontaneously on manipulation of the joint and this is particularly true when the patient is positioned for radiography. It is, therefore, important not to rely on radiography for a definitive diagnosis but rather to evaluate joint stability by manipulation. This is done by holding the scapula in a fixed position whilst the joint is flexed, extended, rotated, abducted and adducted. It is imperative that any suspected abnormality is evaluated by comparison with the contralateral limb as the shoulder is a fairly lax joint and instability can easily be misdiagnosed, particularly when the dog is sedated or anaesthetised for radiography.

The possibility of concurrent injuries should be considered and attention paid to any signs of crepitation, which might suggest a fracture, or neurological deficits, which might indicate brachial plexus involvement (particularly in lateral luxations).

Diagnosis

Standard mediolateral and caudocranial radiographs (Fig. 31.21) are used to assess:

- The direction of luxation
- The contour of the joint surfaces
- Osteophyte formation
- Concurrent fractures

The direction of luxation is important in determining methods used in the management of the problem. If the luxation is long-standing then the joint surfaces may show remodelling, in which case stability may be more difficult to establish, or osteophytes may be present indicating secondary osteoarthritis. Both these will tend to make the prognosis worse. If concurrent fractures (for example, involving the scapular tuberosity or greater tubercle) are present they may interfere with reduction or inherent stability and so might be an indication for open reduction/fixation. As has already been mentioned, shoulder luxations have a tendency to reduce spontaneously during positioning for radiography and so plain radiographs may show no abnormality. If the instability is clearly evident clinically then the radiographs might simply be used to screen the joint for complicating factors. If, however, they are required to demonstrate the instability then further views may be required. Stressed views can

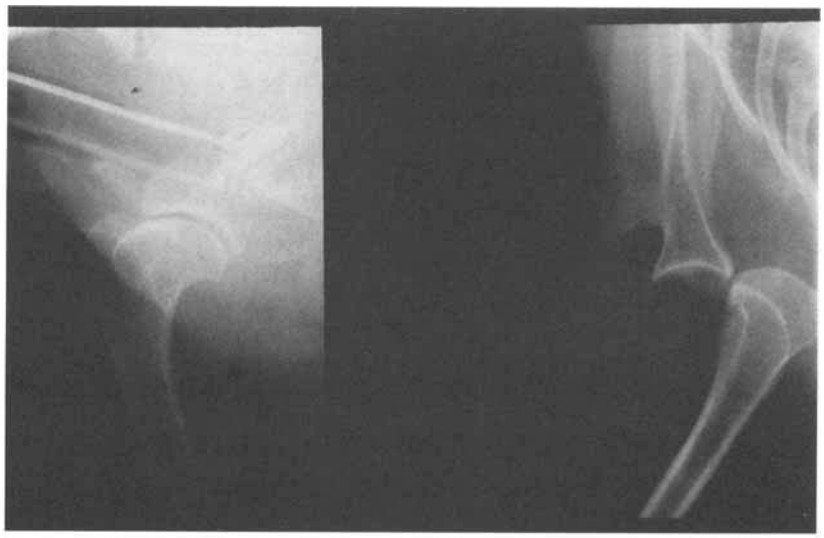


Fig. 31.21 Mediolateral and craniocaudal radiographs of a 9-year-old Toy Poodle's shoulder showing medial luxation resulting from the dog being 'extracted' from under an armchair. Note the 'normality' of the joint in the mediolateral view. (Reproduced from *The Manual of Small Animal Arthrology* with permission from BSAVA Publications.)

be used but are difficult to obtain and must always be taken of both limbs for comparison as the range of 'normal' appearance is great. Alternatively, arthrography might be employed to establish whether leakage of contrast helps to determine the site of joint capsular tearing. The technique is described under 'Osteochondrosis' earlier in this chapter (p. 307), but a volume of 5 or 6 ml would be more appropriate. Some care should be taken in the interpretation of such films as there are normal capsular extensions under the tendons of the 'cuff muscles' and some of the contrast often leaks back along the needle. It must be stressed that the most reliable test for instability is clinical manipulation and radiography should only be used, in this respect, as a last resort.

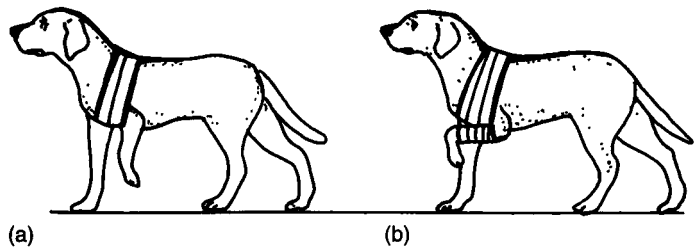
Treatment

Conservative management

In cases where luxation has not previously occurred and there are no concurrent fractures, it may be possible to achieve closed reduction, if this has not occurred spontaneously, and support the joint externally. Slings are particularly useful in this respect. A Velpeau sling (Fig. 31.22) may be employed in medial luxations as this creates pressure on the humeral head in a lateral direction, thus maintaining the reduction. A non-weight-bearing sling (Fig. 31.23) is used for lateral luxations or in cases where the direction is uncertain. Alternatively, a body cast may be used (Fig. 31.24) but this is best fitted in the conscious dog with the limb in a weight-bearing position so that a close-fitting cast can be applied, otherwise it may not serve its purpose and/or be uncomfortable for the dog. Whichever of these is used the joint should always be reradiographed following application of the sling or cast to ensure that reduction has been achieved and maintained. The external support is maintained for 2–6 weeks to allow the soft tissues to heal and exercise restricted for a further 2–4 weeks after removal of the support.

Surgical management

If the luxation is recurrent or surgery is required to remove or reattach a fracture fragment then surgical stabilisation is indicated. There are several techniques described in the literature all of which are aimed at re-establishing the collateral support for the joint. Three techniques will be discussed here but the reader is referred to



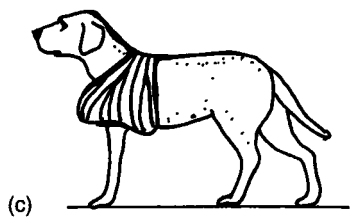
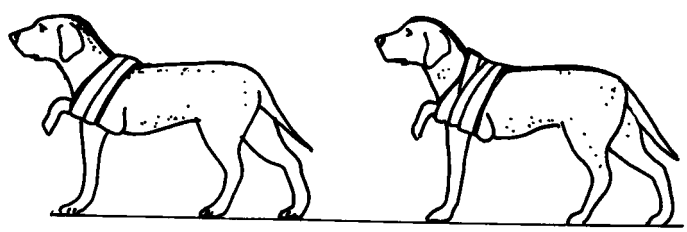


Fig. 31.22 Application of a Velpeau sling. For treatment of *medial* shoulder luxations: (a) the bandage is used to hold the distal humerus adducted against the chest wall; (b) cotton wool is placed around the antebrachium for padding; and (c) the entire limb is enclosed in the bandage, including the cranial aspect or the dog will step out of it. (Source: BSAVA Publications, Cheltenham.)



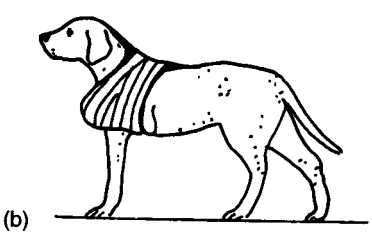


Fig. 31.23 Application of a non-weight bearing sling. For treatment of *lateral* shoulder luxations: (a) the distal limb is flexed and the antebrachium bandaged to the body and (b) the entire limb is enclosed, including the cranial aspect so that the dog does not step out of the sling.

other standard texts if further information is required.

(1) *Figure-of-eight prosthesis to provide lateral support* (Vaughan, 1967) (Fig. 31.25) A lateral approach is used to expose the distal part of the scapular spine and the greater tubercle of the humerus. Tunnels are drilled in a caudal to cranial direction through the distal scapular spine and the greater tubercle. A prosthesis made of braided nylon (in medium-sized dogs a double strand of 7 metric braided nylon [Ethibond, Ethicon] would be appropriate) or terylene is then passed through these tunnels and tied laterally after suturing any identifiable, torn joint capsule.

Since this technique reconstructs the lateral collateral support for the joint, it is primarily intended for use in medial luxations. However, successful use of this has been reported for lateral luxations as well.

(2) *Prosthetic replacement of the lateral and medial collateral support* (Campbell, 1968) (Fig. 31.26) A lateral approach is used with reflection

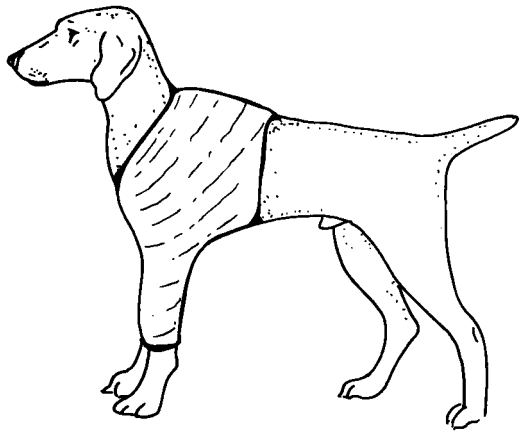


Fig. 31.24 Illustration of a 'body cast' which may be used to stabilise the shoulder whilst allowing some limb function.

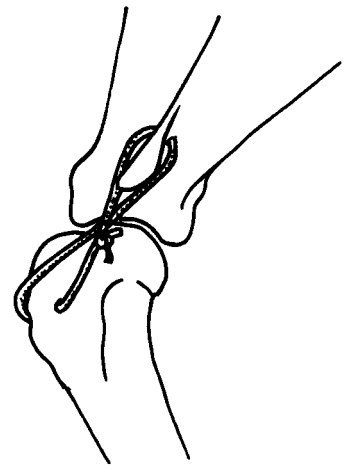


Fig. 31.25 Surgical stabilisation of a shoulder luxation using a figure-of-eight prosthesis. This is most appropriate for medial luxations but may also be used for lateral luxations.

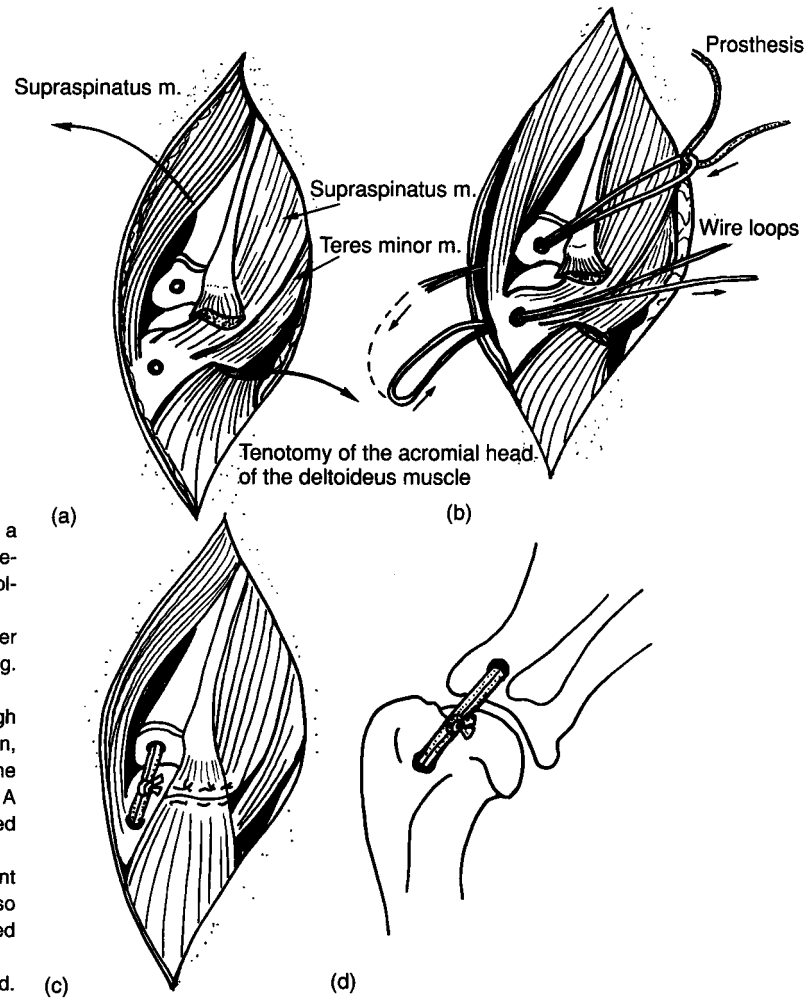


Fig. 31.26 Surgical stabilisation of a shoulder luxation by prosthetic replacement of both the medial and lateral collateral support.
(a) The scapular neck and greater tubercle are exposed as shown in Fig. 31.13.
(b) Transverse holes are drilled through the neck, just cranial to the acromion, and the greater tubercle, close to the insertion of the teres minor muscle. A prosthesis of braided nylon is passed through these.
(c) After repair of any available joint capsule the prosthesis is tightened, so that the joint is stable but not restricted in normal movement, and tied.
(d) Position of the prosthesis illustrated.

of the acromial head of the deltoideus muscle, either by transection of the tendon of origin about 1 cm from the acromion or else by osteotomy of the acromion itself. In addition, greater elevation of the supraspinatus is required so that the medial aspect of the joint can be approached. Transverse tunnels are drilled through the scapular neck, starting just cranial to the distal-most point of the scapular spine, and the proximal humerus, starting close to the point of insertion of the infraspinatus muscle. The prosthesis of nylon (a double strand of braided nylon, 7 metric braided nylon [Ethibond, Ethicon], is suitable for a medium-sized dog) or terylene is then passed from lateral to medial through the scapular tunnel and then from medial to lateral through the humeral tunnel using wire loops. After closure of the joint capsule the prosthesis is tied. This technique is more difficult than (1) because the ends of the tunnels are less accessible, but it does have the advantage of reconstructing medial, as well as lateral, collateral support and so is theoretically more appropriate in cases of lateral luxation.

(3) *Transposition of the tendon of origin of the biceps brachii muscle* (Hohn *et al.*, 1971) (Fig. 31.27) The principle of this technique is to transpose the biceps tendon such that it provides support and physically prevents luxation of the humeral head. By way of a craniomedial approach the biceps tendon is freed from the intertubercular groove by sectioning of the transverse humeral ligament. It may provide support medially, in cases of medial luxation, by being secured in position on the lesser tubercle by means of either a screw and spiked washer, or else by placement under a partial osteotomy of a portion of the tubercle reattached with Kirschner wires. In cases

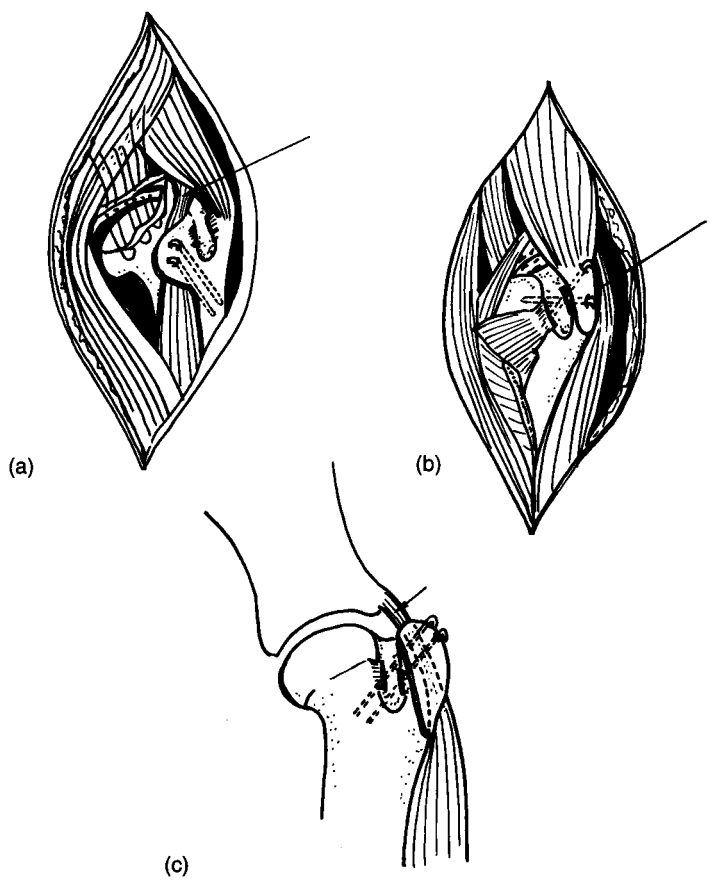


Fig. 31.27 Surgical stabilisation of a shoulder luxation by transposition of the biceps brachii tendon of origin (arrowed). (a) Transposed medially and secured under a bone flap on the lesser tubercle to prevent medial luxation. (b) Transposed laterally, requiring osteotomy of the greater tubercle, to prevent lateral luxation. (c) Transposed cranially, to lie in a groove created under an osteotomy of the greater tubercle, to prevent cranial luxation.

of lateral luxation the tendon may be transposed laterally by performing osteotomy of the greater tubercle which is then reattached using a lagged bone screw or Kirschner wires after the tendon has been repositioned. The technique may also be used in cranial luxations when the biceps tendon is relocated underneath the osteotomised greater tubercle after a groove has been created in the cancellous surface to accommodate it.

Some concern has been expressed over the possibility that repositioning the biceps tendon in this way might adversely affect the normal joint alignment and lead to secondary degenerative change in the long term. There is some experimental evidence to suggest that this is the case, particularly with medial transposition, and it has also been noted that lateral transposition leads to a temporary inward rotation of the limb (Vasseur *et al.*, 1983).

The use of biceps tendon transposition in treating shoulder luxations is widely reported in the North American literature but has only been utilised by one of the authors in one clinical case where a collateral prosthesis failed to maintain reduction. After lateral transposition of the biceps tendon the dog appeared to have difficulty weight-bearing with pain evident on full extension of the elbow and shoulder. Owing to such changes in the biomechanics of the tendon following such transposition, this technique is not routinely used by the authors. For further details of these techniques the reader is referred to the other standard texts listed.

Following surgical stabilisation, the joint may be supported in a sling, as with conservative management, for 7–14 days or else a figure-of-eight bandage may be applied simply for support and to help prevent seroma formation. The dog should be rested for between 4 and 6 weeks.

Prognosis

Maintaining reduction of an acquired shoulder luxation may prove problematic. If conservative measures fail then surgery should be undertaken but the prognosis remains only fair to good for a return to complete normality, particularly in cases with medial luxation. In general it is often found that if surgery has failed to maintain reduction then revision surgery is also prone to failure, unless a definite reason for failure has been identified, and it may be more appropriate to consider a salvage procedure such as arthrodesis or excision arthroplasty.

Dorsal luxation of the scapula

This is an uncommon condition which is invariably a result of trauma causing rupture of the serratus ventralis muscle and possibly additional tearing of the trapezius, rhomboideus, teres major and latissimus dorsi muscles. Diagnosis is based on the clinical appearance of dorsal displacement of the scapula which is clearly evident on weight-bearing. Treatment requires a caudolateral approach to allow reapposition of the torn muscles and reattachment of any avulsed muscles to the scapula using sutures of stainless steel wire or nylon by way of bone tunnels. Further support is usually required and the caudal edge of the scapula may be attached to one or more of the underlying ribs using stainless steel wire. Postoperatively, the limb is supported in flexion for 2–3 weeks and exercise restricted for 6 weeks. The prognosis appears to be reasonably good.

Contracture of the infraspinatus (or supraspinatus) muscle

History

Although any breed may be affected, this uncommon condition is most often seen in working dogs or particularly active pets of any age and usually of medium size. A traumatic aetiology is suspected since, on histological examination, the affected muscles show evidence of haemorrhage, degeneration, atrophy and fibrosis. On careful questioning the owner will often reveal that the dog had a previous lameness with an acute onset, usually starting at or soon after exercise, that improved over the next few days. After a period of a few weeks the current, progressive lameness then developed. The dog will not be reported to have shown discomfort and will still be keen to exercise.

Fig. 31.28 Infraspinatus contracture. Cases with contracture of the infraspinatus muscle may adopt this typical posture when sitting. The elbow is held adducted with the distal limb externally rotated.

Clinical signs

When the dog is standing the limb may be positioned normally but in chronic cases may be held slightly adducted at the elbow with abduction of the foot (Fig. 31.28). At the walk and trot the problem is characterised by obvious circumduction of the limb on protraction and a flip-like extension of the paw as the limb moves forwards. *Manipulation* of the joint causes no pain but flexion is reduced and if the whole limb is flexed up the antebrachium tends to deviate laterally from the body instead of remaining in a straight line. *Palpation* will normally reveal atrophy of the suprascapular muscles and possibly similar changes in other shoulder muscles. The condition may be bilateral.

Diagnosis

Mediolateral and caudocranial radiographs may show changes but it is often useful to take views of both limbs so that they may be compared. In the mediolateral view there may be a relative reduction in the width of the caudal joint space, whereas in the caudocranial view the abnormality looked for pertains to a reduction in the distance between the acromion or the rim of the glenoid and the greater tubercle of the humerus (Fig. 31.29).

Treatment

Surgery is indicated since tenotomy appears to be a successful way of restoring a normal forelimb action. A lateral approach to the greater tubercle provides good exposure of the tendon. The affected tendon appears scarred and fibrotic. It is sectioned at its insertion but elevation from the joint capsule may require the breakdown of adhesions. Following this, normal movement of the joint should be restored immediately.

Of 15 cases from the literature, reviewed by Vaughan (1979), 14 involved the infraspinatus and only one the supraspinatus. In one case dealt with by one of the authors a decision could not be made as to which of the spinatus muscles was involved. As a result, based on the aforementioned report, the infraspinatus tendon was sectioned, but this had no effect on the range of joint movement and so the supraspinatus tendon was also sectioned. A normal range of limb motion returned immediately and the tenotomy of both suprascapular muscles appeared to have no ill-effect.

Postoperatively, the dog should be rested until the skin sutures are removed and then a rapid return to a normal exercise regime should prevent the formation of adhesions.

Prognosis

Following surgery, the prognosis is favourable with all reported cases returning to normal and no recorded incidence of recurrence.

Note

A similar condition has been reported affecting the teres minor muscle (Bruce *et al.*, 1997). The resulting lameness resolved completely after resection of the muscle.

Bicipital tenosynovitis

The aetiology of this condition is poorly understood but it is thought to be related to trauma, often of a repetitive nature. Another possibility would be as a manifestation of osteoarthritis of the shoulder whereby adhesions form between

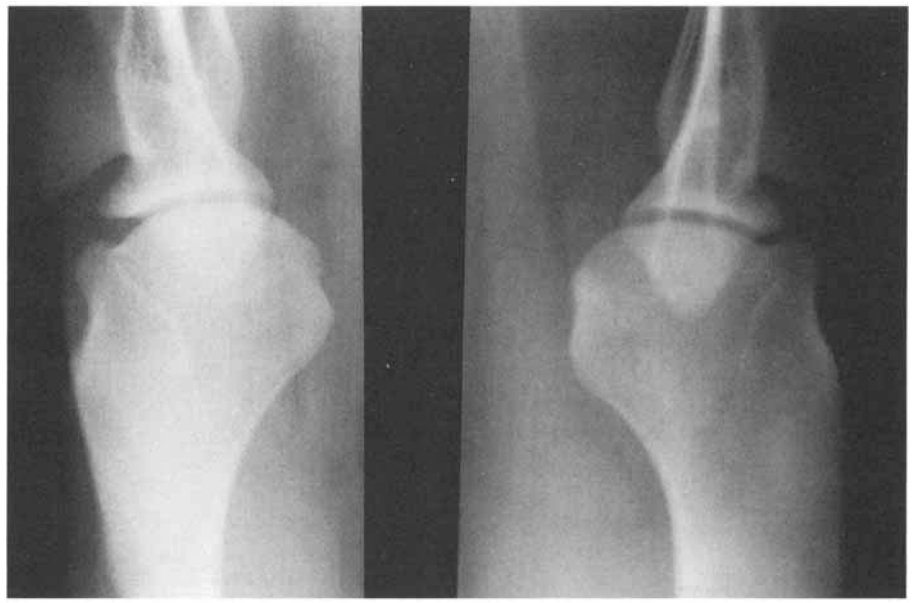


Fig. 31.29 Spinatus contracture in a 5-year-old Labrador Retriever. Caudocranial radiographs of both shoulders showing a reduction in the distance between the greater tubercle and the rim of the glenoid in the affected left shoulder (arrow). (Reproduced from *The Manual of Small Animal Arthrology* with permission from BSAVA Publications.)

the tendon and the joint capsule in the region of the intertubercular groove as a part of the overall arthropathy.

History

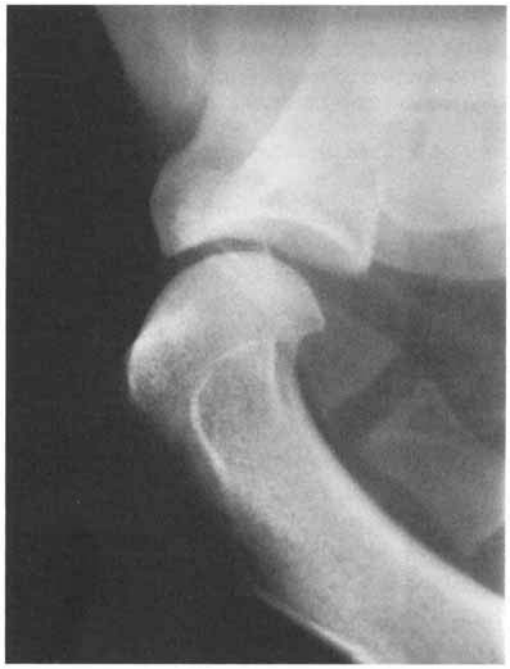
It is generally seen in the older, medium to large breed dogs although smaller breeds may be affected. The onset of lameness may be acute, following a particularly active period of exercise, or be more chronic and insidious. The dogs affected often lead active lives and may have a habit of digging or scrambling up canal banks, for example. The owners will often report a lameness that improves with rest, although there may be some stiffness for a few minutes on rising, and deteriorates with excessive exercise.

Clinical signs

The degree of lameness may be anything from mild to quite severe and pain will be evident on manipulation of the shoulder. Flexion of the joint should stress the tendon most, especially if the elbow is kept extended, and this may cause a pain response. Full extension of the joint also seems to cause discomfort, but this may be because of the pressure applied to the cranial aspect of the proximal humerus when such a manipulation is performed. Direct pressure applied to the tendon, which lies just medial to the cranial edge of the greater tubercle, should cause a response, and again this may be more obvious if the limb is held with the shoulder flexed and the elbow extended at the same time. Patients do vary in their 'normal' reactions to such manipulations and a comparison of the response seen on performing these in both forelimbs should always be made.

Radiology

Mediolateral views are most useful although caudocranial or skyline views (of the intertubercular groove) may add some information. In plain, mediolateral radiographs there may be evidence of new bone deposition superimposed on the greater tubercle which may be located within the intertubercular groove (Fig. 31.30a). In some instances minor lucencies may be observed superimposed on the greater tubercle. The significance



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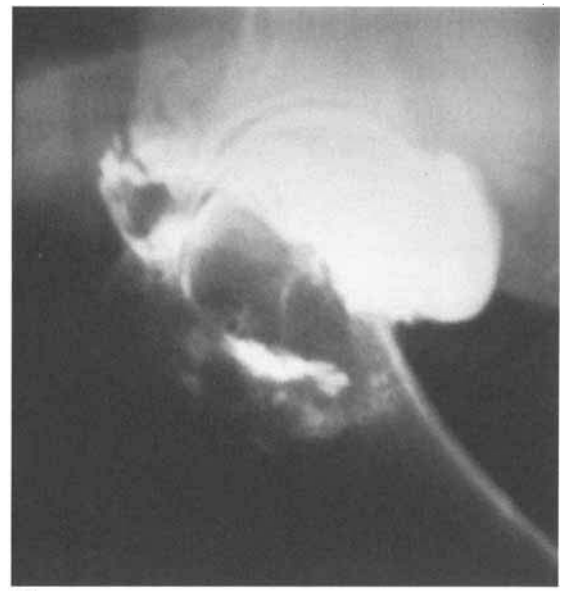

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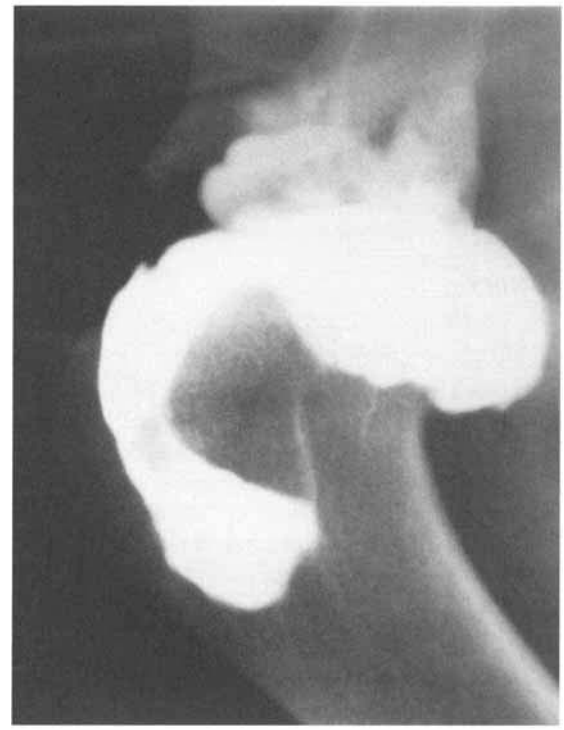

(c)

Fig. 31.30 Bicipital tenosynovitis. (a) Mediolateral radiograph of a 9-year-old German Shepherd Dog with forelimb lameness associated with pain on shoulder extension or direct pressure applied over the biceps tendon. The radiograph shows new bone deposition superimposed on the greater tubercle, possibly in the region of the intertubercular groove.
(b) Mediolateral arthrogram (post-injection of 6 ml of contrast) of a 7-year-old Airedale's shoulder which was showing similar signs to the dog in (a). There is poor filling of the bicipital tendon sheath.
(c) A normal arthrogram for comparison.
(Reproduced from *The Manual of Small Animal Arthrology* with permission from BSAVA Publications.)

of these changes has been brought into question by the fact that many normal dogs, without clinical lameness, show similar changes, and thus the findings must be correlated with clinical signs. Another point of interest is that new bone deposition along the cranial edge of the greater tubercle often represents mineralisation within the tendon of insertion of the supraspinatus muscle. This may be seen in many clinically normal dogs but has been reported as a cause of forelimb lameness, with some cases improving after surgical removal of the mineralised tissue (Flo & Middleton, 1990).

Arthrography may help in making a diagnosis. About 6 ml of a suitable contrast agent should be injected, as described under shoulder osteochondrosis (p. 306), and radiographs taken within about 5 minutes. There may be poor filling of the tendon sheath due to adhesions and this provides convincing evidence of the diagnosis (refer to Fig. 31.30b,c).

Unfortunately, it is quite possible that, as in human patients with this condition, radiographs, including arthrograms, may appear normal. This is not surprising when one considers that it is secondary changes that are being observed rather than the primary problem.

In an effort to improve diagnostic capabilities, ultrasonography has been looked at but has been reported by Rivers *et al.* (1992) to be less sensitive than arthrography. This modality was used, both before and immediately after arthrography, in a series of cases suspected of having this problem seen by one of the authors. Evidence of fluid accumulation around the tendon and the presence of adhesions was looked for. The results showed it to be of little clinical value in these patients, thus agreeing with the findings of other authors. Arthroscopy has also been suggested as a means of evaluating the biceps tendon (Person, 1986) and this is now being evaluated in clinical cases investigated at some centres.

Unfortunately, in many cases suspected of having this problem a definitive diagnosis cannot be reached since there will be no convincing evidence radiographically but the dog will respond to conservative measures (outlined below) and so material for histological examination will not be obtained. *Although it is a difficult diagnosis to* make, and care should be taken not to use it as a 'dustbin diagnosis', it does represent a possible explanation for many undiagnosed upper forelimb lamenesses. It is considered a fairly common condition in man and we have no reason not to think that this may also be the case in dogs. In cases with evidence of shoulder pain, and where no other explanation can be found, a successful outcome may be achieved if a presumptive diagnosis of bicipital tenosynovitis is made and appropriate treatment undertaken.

Treatment

Conservative management

Strict rest for between 4 and 6 weeks may allow the problem to settle down by reducing movement of the tendon in the intertubercular groove. Systemic NSAIDs may help during this period. If such measures fail then the injection of 40–60 mg of methylprednisolone (Depo-Medrone V, Pharmacia & Upjohn) either around the tendon itself or into the joint (as described for arthrography under 'Osteochondrosis', p. 307) under strict asepsis, followed by 4 weeks of controlled exercise and then a gradual return to normal exercise may alleviate the signs. The response to such an injection is usually quite rapid although the patient may be more uncomfortable for a few days following investigation and injection. In about 50% of cases the improvement seems to be permanent, or at least persists for a prolonged period before there is any recurrence. In the remaining cases the improvement is temporary, although the response tends to confirm the diagnosis and in half of these the result can be made permanent by repeating the injection a further one or two times. It has also been noted by the authors that some dogs respond better to peritendinous injection of the methylprednisolone whilst others respond more favourably when it is administered intra-articularly.

Surgical management

If conservative measures fail to provide a satisfactory improvement, surgery may be considered. The tendon is exposed by way of a cranial approach (Fig. 31.18 and details under treatment of 'Fracture of the scapular tuberosity', p. 315).

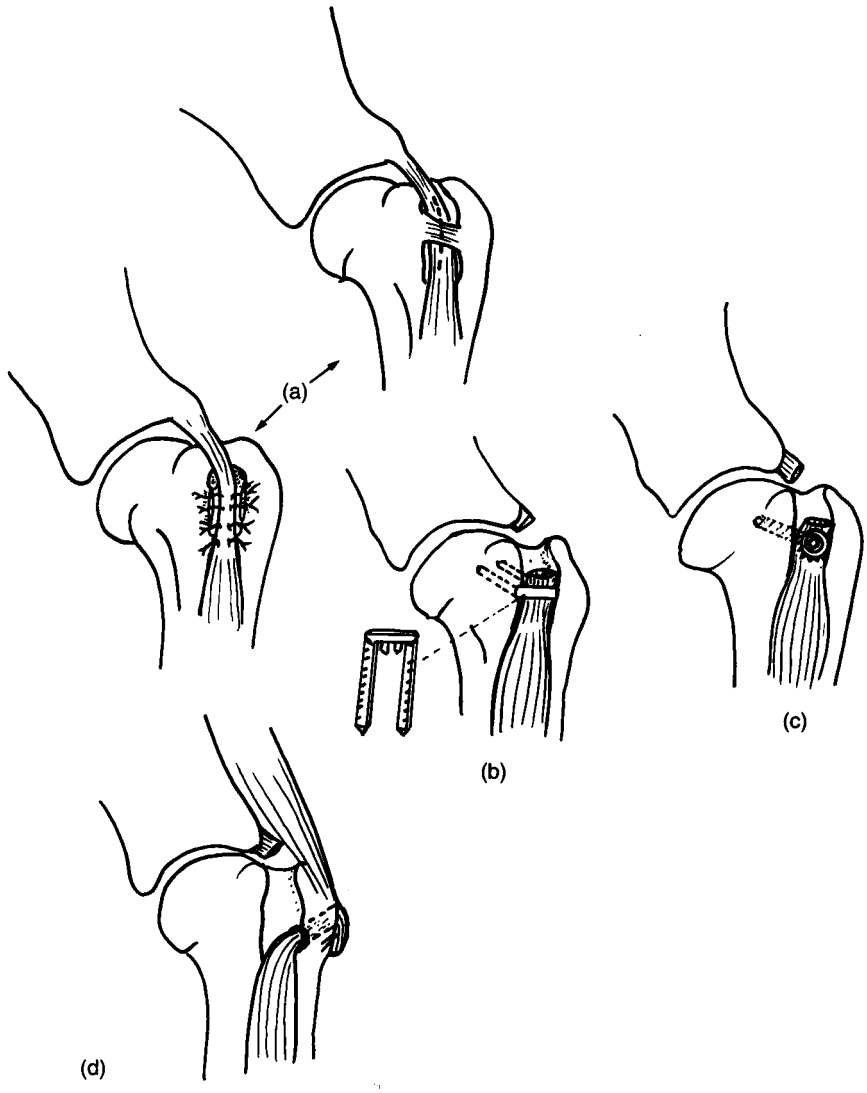

Fig. 31.31 Options in the surgical management of bicipital tenosynovitis.
(a) Transection of the transverse humeral ligament +/– suturing of the tendon to the periosteum.
(b) Sectioning of the tendon from the scapular tuberosity and then using a ligament staple to anchor the tendon in the intertubercular groove.
(c) Sectioning of the tendon from the scapular tuberosity with reattachment to the proximal humerus using a bone screw and spiked washer.
(d) Sectioning of the tendon from the scapular tuberosity and, after passage through a bone tunnel in the greater tubercle, attachment to the supraspinatus tendon laterally.
(Source: BSAVA Publications, Cheltenham.)

Several methods of treating the condition surgically exist (Fig. 31.31) and there are insufficient numbers of cases recorded to establish which of the techniques, if any, is most appropriate. The pressure may be removed from the tendon simply by sectioning the transverse humeral ligament. One concern over this technique is that it may allow medial displacement of the tendon from the intertubercular groove (a known cause of lameness in its own right, see later). In order to avoid

this two or three simple interrupted sutures of polydioxanone (PDS II, Ethicon) may be used to anchor the tendon to the periosteum, laterally, prior to cutting the ligament. This may, in fact, be producing the same effect as the stapling technique described next in causing permanent fixation of the tendon in the intertubercular groove and thus preventing the movement that causes the pain. Alternatively, after sectioning of the ligament, the tendon may be elevated from the groove to allow removal of any osteophytes, the tendon may be anchored in the intertubercular groove with a ligament staple (Veterinary Instrumentation) which is placed with the shoulder flexed and the elbow extended. This prevents the movement of the tendon that causes pain but maintains the normal cranial stability of the scapulohumeral joint afforded by the biceps tendon. Finally, some authors advocate the removal of the tendon from the intertubercular groove so that painful movement of the tendon through the groove is no longer possible. This involves sectioning of the tendon from the scapular tuberosity and its attachment to the proximal humerus by way of a screw and spiked washer, a ligament staple or else by passing it through a tunnel made in the greater tubercle and suturing the tendon to the insertion of the supraspinatus muscle. The dog should be rested for 4–6 weeks after surgery.

Prognosis

The prognosis in these cases is fair to moderate. The majority will respond to conservative measures, although recurrence at a later date is a possibility. There has been little recorded follow-up of cases treated surgically but in the authors' experience many of them appear to return to normal or are greatly improved.

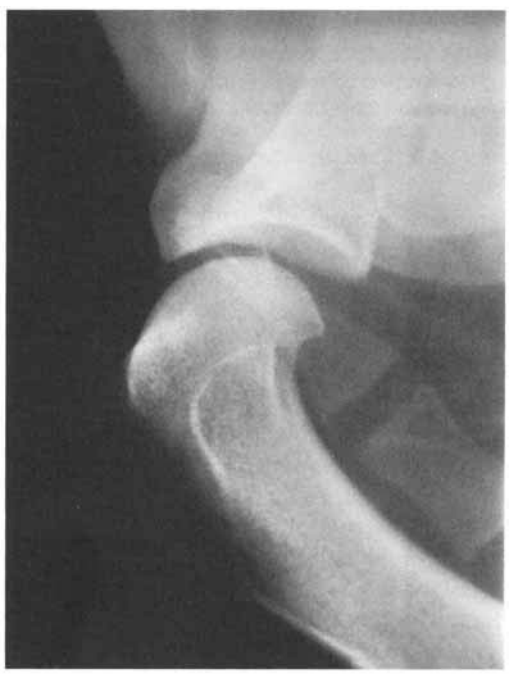

Fig. 31.32 Mediolateral radiograph of a 3-year-old Border Collie's shoulder after he had collided with another dog whilst working. Cranial displacement of the humerus (cranial subluxation) is evident and was associated with medial displacement of the biceps brachii tendon. (Reproduced from *The Manual of Small Animal Arthrology* with permission from BSAVA Publications.)

Medial displacement of the biceps brachii tendon

This is an uncommon cause of lameness that has been reported in racing Greyhounds, a German Shepherd Dog, an Afghan Hound and a Border Collie (Bennett & Campbell, 1979; Goring *et al.*, 1984; Fox & Bray, 1992; Boemo & Eaton-Wells, 1995). The onset of lameness appears to be chronic with gradual deterioration in severity and aggravation by exercise. On examination there may be evidence of pain and/or crepitus on shoulder manipulation. As the shoulder is flexed the tendon may be felt to 'pop' out of the intertubercular groove in a medial direction and then return to its normal position on extension of the joint. There may be an associated luxation of the shoulder joint due to loss of cranial support (Fig. 31.32).

This displacement of the biceps tendon is a result of rupture of the transverse humeral ligament and treatment is aimed at relocating the tendon in the intertubercular groove. A cranial approach is used (Fig. 31.18). It is usually not possible to reconstruct the torn ligament and the tendon is retained in the groove by either placing a mattress suture of wire between the greater and lesser tubercles (Fig. 31.33) or else the use of

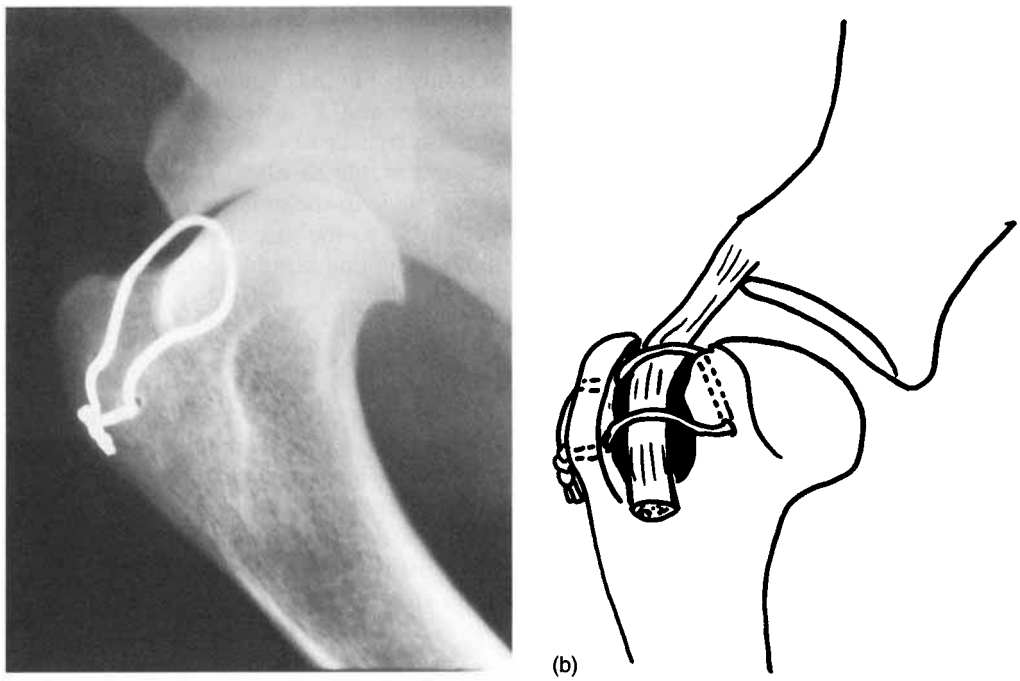

Fig. 31.33 (a) Postoperative radiograph of the same dog as in Fig. 31.32, following relocation of the tendon in the intertubercular groove and the use of orthopaedic wire to replace the restraining transverse humeral ligament. (b) Line drawing showing the relationship of the wire to the biceps tendon in this case. (Part (a) reproduced from *The Manual of Small Animal Arthrology* with permission from BSAVA Publications.)

staples. In either case it is important to allow sufficient room for the tendon to move freely under the prosthesis so that shoulder movement is pain-free and normal in range, or else no movement at all so that the tendon does not 'rub' in the implant (i.e. stabilising the tendon as with stapling in the treatment of bicipital tenosynovitis).

Prognosis

The prognosis for a return to full work has to be guarded but the Border Collie returned to working sheep and some of the Greyhounds returned to racing.

Rupture/avulsion of the biceps brachii tendon of origin

This is an uncommon injury but has been recorded in cases with shoulder luxation, particularly those occurring in a craniomedial direction (Bennett & Campbell, 1979). In such cases the possibility of biceps tendon injury should be investigated at surgery since failure to recognise and repair such damage may lead to reluxation postoperatively. Clinically there is generally swelling and pain over the cranial aspect of the shoulder. Plain radiography is of limited use unless part of the scapular tuberosity has been avulsed (Fig. 31.34). Arthrography may demonstrate disruption of the tendon sheath at the site of rupture. If the shoulder is stable then conservative measures may be employed in treating the patient. Should this fail, or if joint instability is present then the tendon may be exposed using a cranial approach. Wherever possible the tendon should be re-attached to its origin (Fig. 31.35). In some cases this might prove impossible, requiring the tendon's origin to be relocated to the proximal humerus (as described under 'Bicipital tenosynovitis', Fig. 31.31c,d). Such relocation would obviously remove the support that the biceps tendon contributes to shoulder stability and this would

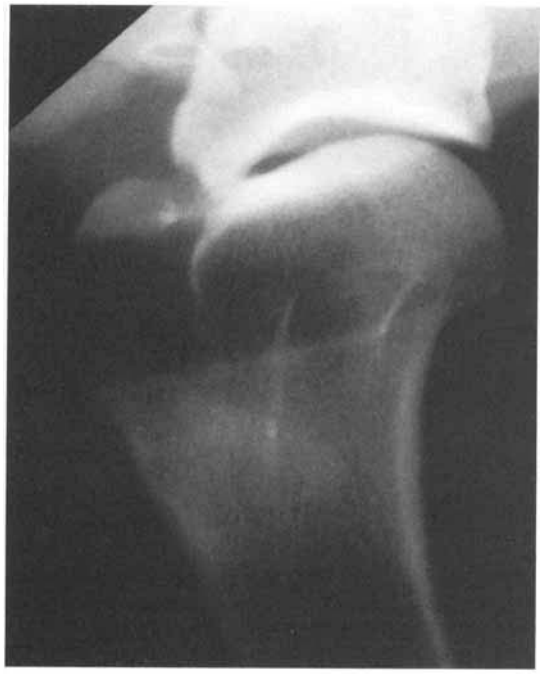

Fig. 31.34 Avulsion of the biceps brachii tendon. Mediolateral radiograph of a 4-year-old Irish Setter's shoulder which developed an acute onset lameness. The biceps brachii tendon had avulsed from the scapular tuberosity, along with a small fragment of bone. (Reproduced from *The Manual of Small Animal Arthrology* with permission from BSAVA Publications.)

have to be taken into account in choosing an appropriate method for surgically stabilising the joint.

Osteoarthritis (OA)

The scapulohumeral joint, as is the case for any articulation, may develop degenerative joint disease or osteoarthritis (OA). This subject is covered elsewhere and the reader is refered to Chapter 7 for further information. With specific reference to the shoulder, a study of the incidence and clinicopathological aspects of the disease was carried out by Tirgari & Vaughan (1973).

History

If this condition is associated with clinical signs the owners will normally report, as in the case of other joints with the same problem, that the dog shows an intermittent or gradually deteriorating lameness, possibly with sporadic worsening, that is aggravated by exercise and appears to be associated with stiffness after rest. The problem is usually secondary to an earlier shoulder problem such as osteochondrosis, fracture or luxation but may be associated with a more long-standing problem elsewhere in the limb, such as elbow osteochondrosis, presumably by this causing altered weight-bearing through the limb, with compensation at other joints leading to degenerative changes in the long term (H. Rudorf, pers. comm., 1992).

Clinical signs

The degree of lameness may vary but is most often of mild to moderate severity. Palpation of the area may reveal disuse atrophy, particularly of the suprascapular muscles whilst manipulation of the joint will reveal pain, possibly on flexion but most notably on extension. Also, there may be evidence of pain when pressure is applied directly over the biceps tendon, as discussed under 'Bicipital tenosynovitis'. This may be explained by the concept that if degenerative changes are present throughout the joint then the biceps tendon sheath will be involved and may be a cause of pain for the same reasons as in cases of tenosynovitis.

Diagnosis

A mediolateral radiograph is of most use (Fig. 31.36) but a caudocranial view may provide additional information. The changes that are evident relate to new bone formation and this is most often seen as a variably sized osteophyte on the caudal edge of the humeral head. A similar osteophyte may also be seen on the caudal aspect of the glenoid. Irregular, radiodense lines may be present across the humeral head indicating circumferential deposition around the edge of the articular surface. Similar lines, or a more general increase in density, may be observed superimposed on the greater tubercle in the vicinity of the intertubercular groove and it is in these cases that involvement of the biceps tendon sheath may be more likely.

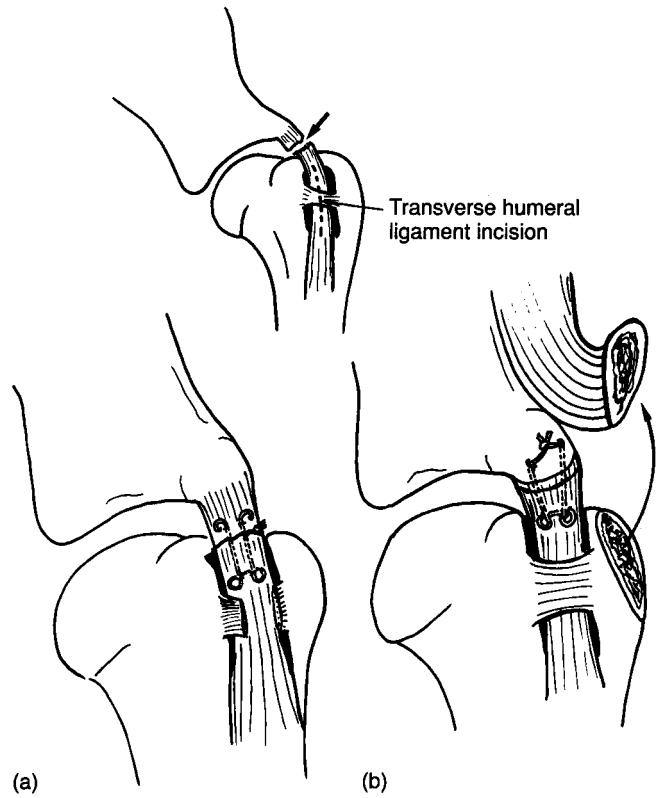

Fig. 31.35 Surgical treatment of a ruptured/avulsed biceps brachii tendon.
(a) Repair of a 'mid-tendon' rupture using appropriate sutures (locking loop in this illustration).
(b) If the tendon has avulsed from the scapula it may be necessary to anchor the sutures through holes drilled in the tuberosity. (Source: BSAVA Publications, Cheltenham.)

It is important to realise that these changes are found in a large number of middle- to old-aged, clinically normal dogs and so the significance of such findings has to be determined by clinical evaluation.

Other methods of further investigation, e.g. synovial fluid analysis, etc., are discussed in Chapters 6 and 7.

Treatment and prognosis

Many of these cases can be managed conservatively as discussed in Chapter 7 and mentioned previously. In cases that are refractory to such measures the authors have found the intra-articular injection of 40–60 mg methylprednisolone (Depo-Medrone, Pharmacia & Upjohn), followed by 4–6 weeks of restricted exercise, to reduce the degenerative effect of the corticosteroid on the articular cartilage, to be of value. With the advent of licensed polyglycosaminoglycans preparations (Cartrophen-VET, Arthropharm, Australia), it may be worth considering the intra-articular use of these, if systemic administration has failed, but there is no data, as yet, to indicate that clinical improvement should be expected.

If there is a clinical suspicion that the biceps tendon sheath is a cause of pain then treatment along the lines described under 'Bicipital tenosynovitis' might prove to be of benefit. Although these cases do have a general shoulder arthropathy it may be that the lameness is associated with the changes in the vicinity of the biceps tendon and appropriate treatment in this direction will alleviate the clinical signs.

The vast majority of cases can be managed adequately, such that they lead a normal life for most of the time, using conservative measures. As a last resort, if an intractable lameness develops that fails to respond to the measures discussed thus far, it may be necessary to consider the possibility of

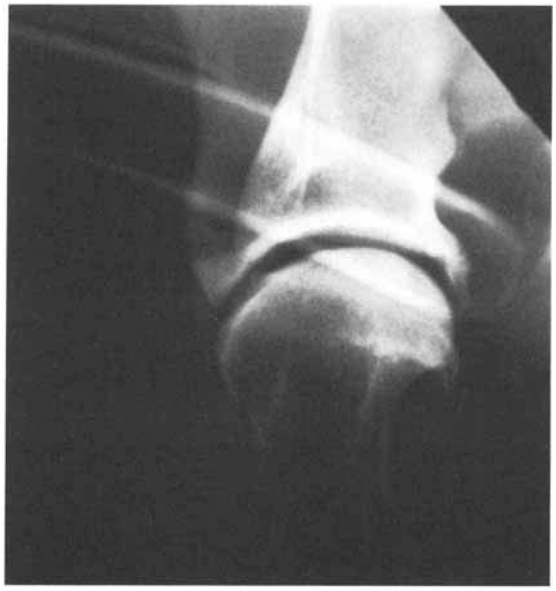

Fig. 31.36 Shoulder osteoarthritis (OA). Mediolateral radiograph of a 10-year-old Labrador Retriever's shoulder. There is radiographic evidence of OA with periarticular osteophyte formation, most notably on the caudal margins of the glenoid and humeral heads. There are also deposits of new bone along the margins of capsular attachment and superimposed on the greater tubercle.

carrying out a shoulder arthrodesis (discussed at the end of this chapter).

Neoplasia

Shoulder lameness as a result of neoplasia will be seen not infrequently in dogs. Although most cases will be middle-aged or older, some cases may be only in their second year of life. Any breed may be affected but it is the medium to large breeds that are seen most often for such a lameness. *Osteosarcoma* is by far and away the most common form of neoplasia to affect the shoulder, with the proximal humeral metaphysis being a predilection site for this condition. In one review of osteosarcomata affecting the appendicular skeleton in 74 dogs (Gibbs *et al.*, 1984) the lesion was located in the proximal humerus in 20% of the cases. Besides this, other sarcomata, such as *fibrosarcoma* and *chondrosarcoma* may affect the proximal humerus or scapular blade. The scapulohumeral joint itself may become involved with a neoplastic process, perhaps the most likely form being *synovial sarcoma*. Finally, *brachial plexus tumours* may also cause an intractable lameness associated with shoulder pain.

More information about these various tumour types will be found in Chapter 51 and, for the purpose of discussion of shoulder lameness associated with neoplasia, osteosarcoma, synovial sarcoma and brachial plexus, tumours will be considered here.

Osteosarcoma

History

This problem is usually encountered in large to giant breeds of dog and may affect any dog over 1 year of age. The owner will normally report a chronic, progressive lameness which is often not made particularly worse with exercise and not associated, to any great extent, with stiffness after rest. Occasionally, such a lameness may be described but with a recent, acute deterioration. The owner may also describe general weight loss, associated with an increased or decreased appetite and in some cases the dog will show general malaise due to either the chronic pain or else metabolic effects that the tumour may cause.

Clinical signs

On observing the patient, the weight-bearing lameness may be anything from very mild to very severe. Manipulation of the shoulder will reveal pain, particularly on extension, and palpation will often reveal muscle wastage. In the later stages of the disease the limb often feels wasted with 'relative sparing' of the area around the proximal humerus due to growth of the tumour which may feel hot and swollen. Although auscultation of the thorax reveals no abnormalities in most cases, even in the presence of pulmonary metastases, it is important to include this in the general examination of the patient.

Radiology

Mediolateral views are the most useful but additional information may be gleened from

caudocranial projections. The process may be typed, according to the radiographic findings, as predominantly *lytic*, predominantly *productive*, or *mixed*. Changes which may be observed include those listed below (Fig. 31.37):

- Destruction of the normal trabecular architecture with or without adjacent sclerosis
- Thinning of the cortical bone
- Breakout through the cortical margins with expansion into surrounding soft tissues
- Pathological fracture
- Periosteal reaction with new bone formation (sometimes in the 'sunburst pattern')
- Elevation of the periosteum with new bone formation (Codman's triangle)
- Surrounding soft tissue swelling

In general, the subchondral bone is spared and the lesion seldom crosses the joint. The severity of the radiographic changes correlates poorly with the duration or severity of the clinical lameness although those with an acute onset or deterioration of signs will often have a pathological fracture evident. Lateral views of the chest should be taken with the patient in right and left lateral recumbency, and the lungs inflated, to look for pulmonary metastases.

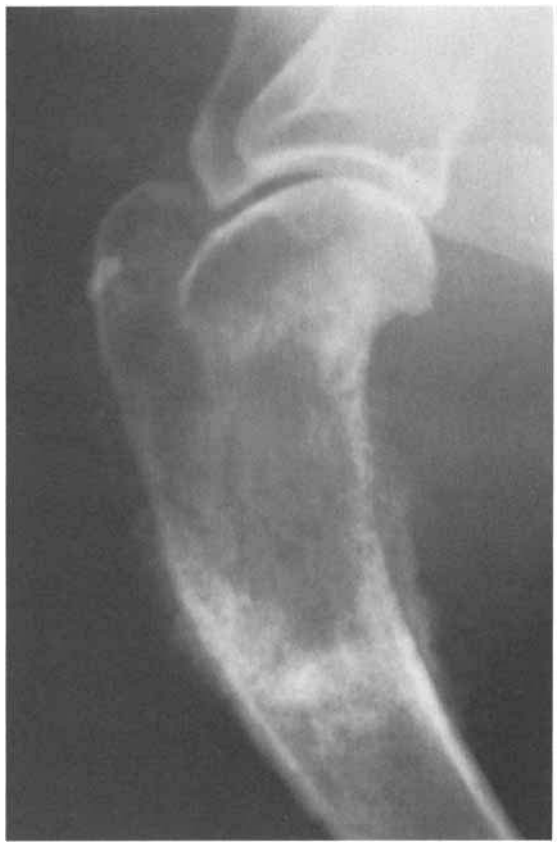

Fig. 31.37 Osteosarcoma of the proximal humerus. Mediolateral radiograph of a 6-year-old Rottweiler's shoulder. Lameness had been present for 8 weeks. The majority of the changes listed in the text, associated with osteosarcoma, are evident in this film.

Further investigation

A fairly accurate, provisional diagnosis can usually be made from the history, clinical signs and radiological findings. In order to make a definitive diagnosis as to the disease process, differentiating it from the possiblity of osteomyelitis and establishing the exact tumour type, a biopsy is required. It is important to obtain a 'core' biopsy from the margin of the lesion, otherwise reactive or necrotic tissue will be obtained that will tend not to provide a definitive diagnosis.

Scintigraphy is becoming more widely available and may provide some useful information (see Chapter 51, p. 619), particularly with respect to delineating tumour margins in cases where limb-sparing surgery is being considered.

Treatment and prognosis

Treatment and prognosis for cases with osteosarcoma are discussed in Chapter 51 (p. 619). Briefly, the outlook is poor. Treatment may be symptomatic, may involve removal of the primary lesion by forequarter amputation or limb salvage (by resection of the proximal humerus, replacement of this with a cortical bone graft, supplemented with a cancellous graft, combined with shoulder arthrodesis) and might include adjunctive chemotherapy after either of these surgical procedures. The average survival times for each treatment modality is given in Chapter 51 (p. 619). A recent report by Kuntz *et al.* (1998) showed that only 2 out of 17 dogs treated for osteosarcoma of the proximal humerus by limb salvage and adjunctive chemotherapy had good or excellent outcomes. The average survival time for the dogs in that

study was 172 days, compared to 204 days after forequarter amputation and chemotherapy. Their conclusions were that, currently, limb salvage surgery for osteosarcoma at this site is difficult to justify.

Synovial sarcoma

This is an infrequent cause of shoulder lameness in middle- to old-aged dogs with chronic, progressive clinical signs. Pain is evident on shoulder manipulation and soft tissue swelling is usually palpable. Enlargement of the axillary lymph node may be present possibly indicating lymphatic metastasis. Radiographs may show the soft tissue swelling and erosion of bone at the site of capsular attachment may be present. The latter may take the form of a localised erosion, with 'apple-coring' of the caudal humeral head (Fig. 31.38), or more diffuse erosions on both sides of the joint. Some of these tumours show a rapid rate of metastasis and thoracic radiographs are obligatory to look for pulmonary metastases.

Treatment may take the form of forequarter amputation but the prognosis is very guarded due to the incidence of pulmonary metastasis even when adjunct chemotherapy with cisplatin or doxorubicin is used.

Further information on synovial sarcoma is to be found in Chapter 10 (p. 77).

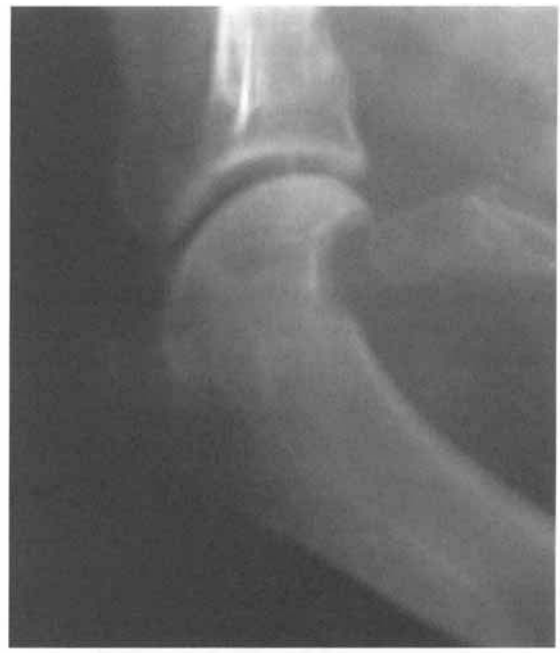

Fig. 31.38 Synovial sarcoma affecting the shoulder joint. Mediolateral radiograph of a 7-year-old Flat Coated Retriever's shoulder. The dog had shown a progressive forelimb lameness over a period of about 3 months and a palpable mass was present caudal to the shoulder, extending towards the axilla. A lytic lesion is evident in the caudal humeral head.

Neoplasia of the brachial plexus

History

The condition is usually seen in medium to large breeds of dog but the smaller breeds cannot be discounted. Most are middle-aged or older, but cases as young as 2 years of age have been reported. The owners will usually report that the dog has shown a chronic intractable lameness for several weeks or months (Sharp, 1989).

Clinical signs

A weight-bearing and/or swinging limb lameness will be evident with pain detectable on shoulder manipulation, especially extension, or axillary palpation. In some cases a mass may be detected, especially when the dog is anaesthetised, but lack of such a mass does not make the diagnosis any less likely. Taking hold of the proximal humerus and scapula simultaneously and moving the limb forwards and backwards, relative to the body, may also cause pain due to stretching of the nerve roots, so-called 'root signature'. If the lesion extends into the vertebral column then neck pain may be evident on manipulation. Muscle atrophy is usually very obvious, particularly of the suprascapular muscles. This is neurogenic atrophy and may be confirmed by means of electromyography. Neurological deficits such as poor proprioceptive and withdrawal reflexes may be noted. Although tendon reflexes and skin sensation may be tested, the results are often equivocal, and any apparent reduction or absence of these should only be considered significant if there is a definite difference when compared to the contralateral limb. Ipsilateral loss of the panniculus reflex and partial or

complete Horner's syndrome are other neurological signs that are sometimes apparent. If the lesion is causing spinal cord compression then neurological deficits may be present in the hindlimbs. The neurological signs in a series of such cases was reported by Targett *et al.* (1993).

Radiology

In the vast majority of cases, plain radiographs show no abnormality and serve only to rule out other potential causes of such a lameness. Occasionally, where the lesion involves the nerve root as it passes out of the vertebral column, there will be evidence of enlargement of the corresponding intervertebral foramen. If vertebral canal involvement is suspected then myelography might show the presence of an intradural, extramedullary lesion. Although pulmonary metastases are rare in these cases, thoracic radiographs should be taken to evaluate such a possibility.

Further investigation

If further confirmation of the diagnosis is required then surgical exploration of the brachial plexus, with or without biopsy, is possible by way of a medial approach described by Knecht & Greene (1977) and illustrated in Fig. 31.39, or using a craniolateral approach described by Sharp (1988). If

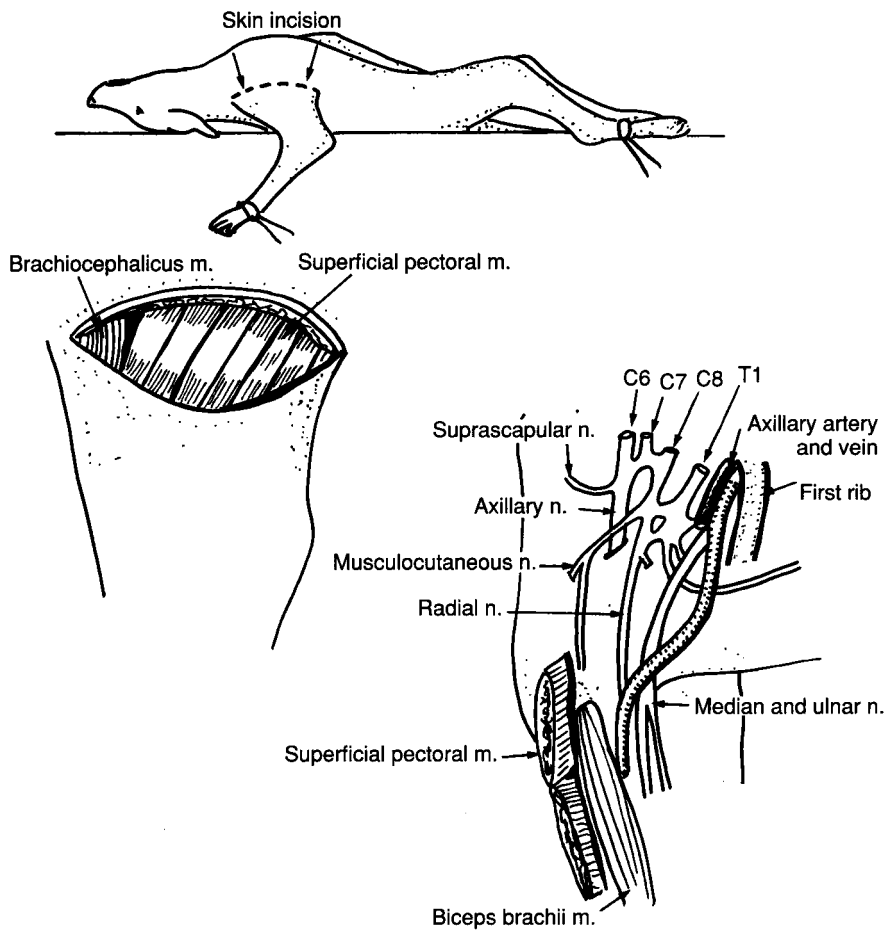

Fig. 31.39 Surgical exploration of the brachial plexus through a medial approach.

inspection of the vertebral canal is necessary then laminectomy is required.

Treatment and prognosis

Resection of the lesion with adequate margins of safety would damage the nerve supply to the forelimb to such an extent that it would be of little use to the dog. Owing to this, the only practicable means of treatment involves forequarter amputation (Harvey, 1974). In cases where the lesion has spread to involve the vertebral canal, forequarter amputation and removal of the intradural component via laminectomy has been advocated.

The prognosis in these cases is very guarded since although the rate of distant metastasis is quite low, the tumours are locally invasive and complete removal may prove very difficult or impossible since the extent of the lesion may not be detectable on gross examination. Unfortunately, some cases will require euthanasia at surgery due to the extensive nature of the lesion making resection impossible, whilst others will be put to sleep within a few months of surgery due to recurrence of clinical signs in the form of neck pain or neurological involvement of the hindlimbs.

Arthrodesis

Arthrodesis of the scapulohumeral joint is technically possible but should be considered a salvage procedure and resorted to when more appropriate methods of treatment (surgical or conservative) have failed or do not exist. The indications for this procedure include:

- Irreparable fractures of the glenoid or humeral head
- Recurrent luxation with resulting erosion of the glenoid rendering surgical stabilisation impossible
- Chronic pain associated with severe osteoarthritis

The principles of the technique are outlined in Fig. 31.40 and an example shown in Fig. 31.41. Further details relating to arthrodesis in general are given in Chapter 2 (p. 22). As in all cases of arthrodesis the aim is to remove the interposed articular cartilage and to stabilise the site at a functional angle (105–110°), preferably with compression between the opposing surfaces of cancellous bone, possibly using a cancellous bone graft to increase the rate of clinical union. Stability is provided by application of a well-contoured plate or Kirschner wires and a tension band wire in small dogs and cats. Although there is a resultant decrease in the range of

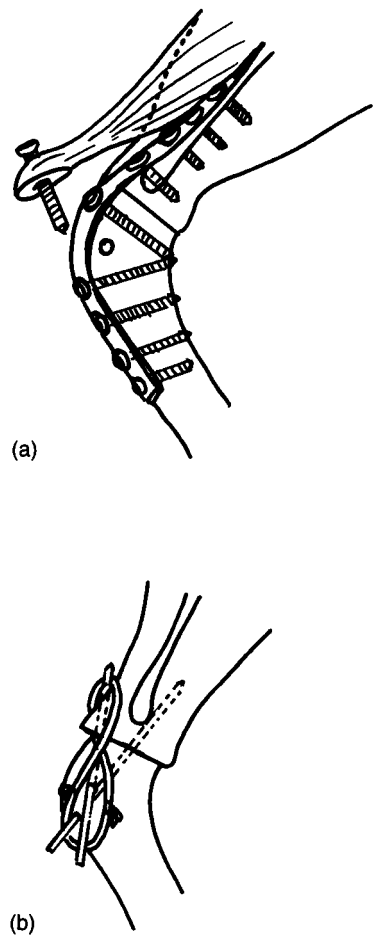

Fig. 31.40 Illustration of the principle of shoulder arthrodesis. (a) Using a bone plate to stabilise the site in medium to large breeds of dog. (b) Using Kirschner wires and a tension band wire to stabilise the site in small dogs and cats.

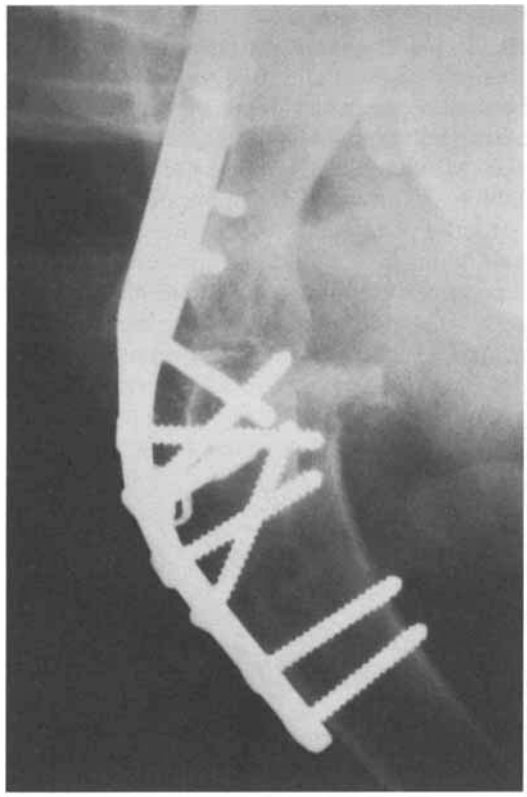

Fig. 31.41 Shoulder arthrodesis. A 12-year-old Shetland Sheepdog sustained a medial shoulder luxation as a result of fighting with another dog. At presentation 2 months later the luxation could not be reduced. Postoperative, mediolateral radiograph following resection of the articular surfaces and stabilisation with a bone plate.

movement in the limb and a persistent mechanical lameness, the clinical result is usually a significant improvement on the preoperative lameness with careful case selection. The procedure can be carried out on both shoulders in dogs with bilateral congenital shoulder luxation (8 weeks between surgeries) and again the functional end results have been reported as good (M. Herron, pers. comm., 1990).

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Chapter 32
The Humerus

The majority of fractures of the humerus, with the exception of condylar fractures, are caused in road traffic accidents. In a recent survey of the incidence of canine appendicular musculoskeletal disorders (Johnson *et al.*, 1994) fractures of the humerus accounted for 10% of all appendicular fractures. Most humeral fractures are treated by internal fixation because it is difficult to satisfy the main criteria for using external coaptation, in particular the immobilisation of the joint above and below the fracture. Chest injuries, particularly pneumothorax, are common complications of humeral fractures. Other possible thoracic injuries include intrapulmonary haemorrhage, diaphragmatic rupture, rib fractures and, occasionally, chylothorax. A careful clinical and radiological examination should be done to check for and, if necessary, treat chest injuries before embarking on fracture fixation.

The majority of humeral diaphyseal fractures follow the curvature of the musculospiral groove and tend to be spiral or oblique in nature. The radial nerve lies close to the fracture site and paresis is a common complication. Fortunately, the paresis is invariably transient and resolves within 2–3 weeks of fracture repair. Nevertheless, the nerve should be inspected during open reduction and carefully protected during the insertion of implants.

Fractures of the humerus can be broadly classified into three groups (Braden, 1975):

- Fractures involving the proximal epiphysis and metaphysis
- Fractures of the diaphysis
- Distal humeral fractures (supracondylar, condylar and intercondylar fractures)

The approximate distribution of fractures between these three groups has been quoted as 8, 40 and 52%, respectively (Braden, 1975). Methods of repair of humeral fractures have been described (Brinker, 1974; Braden, 1975). The surgical approaches used are those described by Piermattei & Greeley (1979).

Humeral fractures in the cat

Superficially the structure of the humerus is similar in the dog and cat, however there are some anatomical differences between the two species. The humeral diaphysis in the cat is straighter and the medullary cavity has a more uniform diameter than the dog. Consequently, intramedullary fixation provides a satisfactory method of treatment for many feline diaphyseal fractures. Another important anatomical difference between the dog and cat is the position of the median nerve. In the dog, the nerve lies just cranial to the medial epicondyle, while in the cat the medial epicondyle is pierced by the epicondyloid fossa which contains the median nerve (Fig. 32.1). The supratrochlear fossa is not completely penetrated in the cat.

Fractures of the proximal humerus

The proximal epiphysis (humeral head, greater and lesser tubercles) develops from one centre of ossification. Closure of the growth plate progresses from its centre, below the junction between the greater tubercle and the head, and is complete between 7.5 and 11 months of age in the dog (Sumner-Smith, 1966), and between 19 and 26

months of age in the cat (Smith, 1969). Approximately 8% of humeral fractures involve the proximal third. These can be divided into:

- Fractures involving the proximal growth plate
- Fractures of the head and/or tubercles
- Metaphyseal fractures

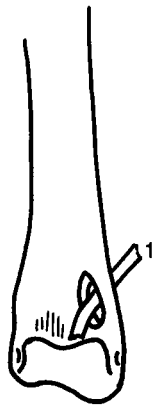

Fig. 32.1 Distal humerus of the cat. The median nerve (1) passes through the epicondyloid fossa in the medial epicondyle.

Fractures involving the proximal growth plate

Fractures involving the proximal growth plate are uncommon in the dog and cat. Salter Harris type I and II fractures (Fig. 32.2) have been recorded most frequently, while Dejardin *et al.* (1995) described a single case of a Salter Harris type III fracture (see below). Considerable trauma is necessary to separate the proximal humeral epiphysis and usually severe, caudomedial overriding of the fragments occurs. Open reduction is essential. In type I and II fractures, a longitudinal incision is made over the craniolateral aspect of the proximal humerus. The deep fascia is incised along the caudal border of the brachiocephalicus muscle and the muscle is reflected cranially to expose the fracture. Additional exposure of the proximal shaft can be achieved by incision of the perios-

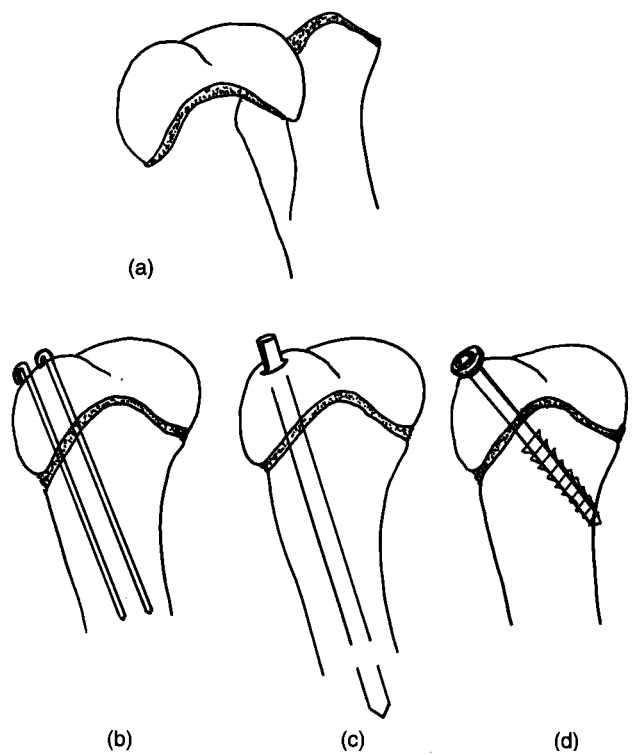

Fig. 32.2 (a) Salter Harris type 1 separation of proximal humeral epiphysis. Methods of fixation: (b) Kirschner wires, (c) intramedullary pin, (d) cancellous screw, partially threaded (only use in animals over 7 months of age).

teum between the cranial border of the deltoideus muscle and the superficial pectoral muscle.

The epiphysis is grasped with pointed AO reduction forceps and the proximal metaphysis with Kern bone-holding forceps. Reduction can be difficult but is achieved by a combination of steady, direct traction using the Kern forceps while a periosteal elevator is interposed between the fracture surfaces and used to lever the metaphysis forward until reduction is achieved. Stability is usually good and can be maintained in animals under 7 months of age by the insertion of two Kirschner wires or a Steinmann pin (Fig. 32.2b,c). The implants are introduced from the greater tubercle and driven down into the shaft of the humerus. A cancellous bone screw can be used for fixation in animals over 7 months of age (Fig. 32.2d). In growing animals, implants should be removed once fracture union is complete (4–8 weeks).

Fractures of the head and/or tubercles

In immature dogs and cats, fractures of the head and tubercles of the humerus occasionally occur as Salter Harris type III fractures involving the proximal humeral growth plate (Dejardin *et al.*, 1995). In this fracture (Fig. 32.3a) there is avulsion of the greater tubercle and complete separation of the humeral head. In addition, there may be fracture of the lesser tubercle. The greater tubercle tends to remain close to the glenoid but there is marked caudal displacement of the humeral head. As such fractures involve the articular surface, open reduction is essential to allow accurate anatomical reduction of the fragments. A craniolateral approach to the shoulder (see Fig. 31.13) is used to expose the fracture. Following reduction, the humeral head and greater tuberosity are retained in position using Kirschner wires (Figs 32.3b,c).

In adult animals, these fractures are rare. One reported case occurred in a 3-year-old Miniature Dachshund (Holt, 1990) and the fracture was stabilised using two Kirschner wires. An alternative method would have been the use of a Kirschner wire in combination with a lag screw (Fig. 32.4). For the surgical approach see 'Fractures involving the proximal growth plate' above. Implants are inserted from the craniolateral aspect of the greater tuberosity and are directed caudally into the humeral head.

Fracture of the proximal metaphyseal region

Fractures of the proximal metaphyseal region are uncommon. They tend to be transverse and impacted. Nutritional secondary hyperparathyroidism in pups or osteosarcoma formation in adults predispose to fracture in this region. In puppies or kittens, because of the inherent stability of the fracture, cage rest may be sufficient to allow healing to occur. However, if there is displacement then an intramedullary pin is used for fixation.

The surgical approach is as for 'Fractures involving the proximal growth plate' above. After fracture reduction, a Steinmann pin is inserted through the skin and bone just lateral to the ridge of the greater tuberosity and driven

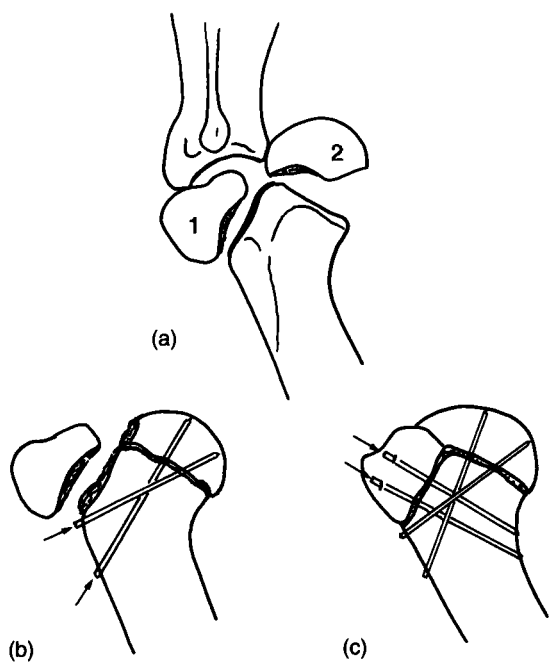

Fig. 32.3 (a) Salter Harris type III fracture of the proximal humeral epiphyses showing the greater tuberosity (1) and the humeral head (2). (b) Humeral head stabilised with two Kirschner wires. (c) Greater tuberosity reattached with two Kirschner wires.

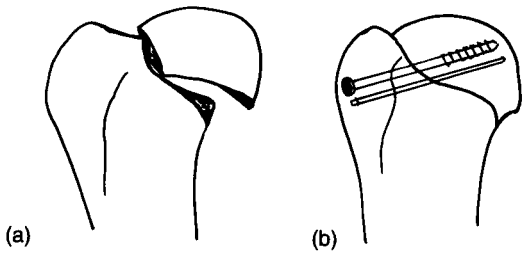

Fig. 32.4 (a) Fracture of the humeral head. (b) Fixation with lag screw and Kirschner wire.

well down into the distal shaft of the humerus. Postoperative care involves checking that the diet is adequate and supplementation with calcium if necessary. Exercise is restricted for 4 weeks and the pin is removed once healing is complete (4–6 weeks).

Fractures of the diaphysis

In the dog, the medullary cavity of the humerus is wide proximally and gradually decreases in size towards the supratrochlear foramen. Consequently, although fractures do occur in the proximal shaft, the majority involve the distal two-thirds and, in particular, the distal third. Fractures of the proximal and mid-shaft regions tend to be transverse, while the more distal fractures follow the curvature of the musculospiral groove and are spiral or oblique in nature. Many are also comminuted. The method of fixation chosen will be influenced by the age and size of animal involved and the nature of the fracture itself.

Intramedullary fixation

Rotational stability using a single Steinmann pin for fixation tends to be poor because of the shape of the medullary cavity which is wide proximally and narrow distally. This method should be reserved for transverse or blunt oblique fractures in small dogs and cats. Radiographs are taken of both the fractured and the normal humerus. The normal is used as a guide to select a pin of the correct diameter to fit the medullary cavity as tightly as possible at its narrowest point. If necessary, the length of pin required can be assessed at

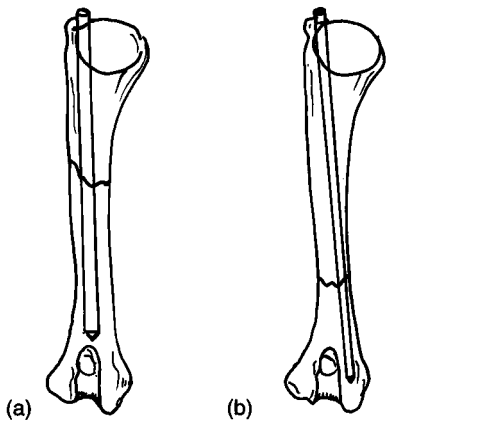

Fig. 32.5 Intramedullary pin. (a) For mid-shaft fracture. (b) For fracture of the distal third of the shaft, the pin is driven down into the medial epicondyle.

this stage. The pin may be partially transected with a hacksaw (cut around the entire outer circumference) so that the surgeon is then able to break the pin off flush with the surface of the greater tuberosity after insertion. The position of the fracture influences the length of pin required. For fractures involving the proximal or mid-shaft region, the pin is driven down the shaft to a point just proximal to the supratrochlear foramen (Fig. 32.5a). For fractures involving the distal third, a smaller diameter pin is used which should be directed towards the medial side of the shaft so the tip bypasses the supratrochlear foramen and is embedded in the medial epicondyle (Fig. 32.5b).

Surgical approach

A standard, craniolateral approach is used (see 'Plate fixation' below) and the radial nerve should be identified and protected throughout the surgery. After exposing the fracture, self-locking bone-holding forceps are applied to the bone just proximal and distal to the fracture site, whilst protecting the radial nerve. The bone ends are toggled against each other until reduction is achieved. The method of introduction of the intramedullary pin is a matter of personal preference. Using a Jacob's chuck the pin can be introduced from the proximal end of the humerus. The correct point of entry is just lateral to the ridge of the greater

tuberosity. The pin is then directed to glide down the medial cortex of the humerus towards the fracture site. Reduction of the fracture is most easily maintained by an assistant using the bone-holding forceps. Once the tip of the pin can be felt approaching the fracture, the bone fragments should be bowed slightly medially to help direct the tip of the pin down into the medial part of the condyle.

The alternative method of pin introduction involves retrograde pinning. The pin is driven up the shaft from the fracture site, keeping the shoulder flexed, and the pin directed towards the lateral side of the greater tuberosity. Once the tip of the pin has emerged, it is grasped with the Jacob's chuck and drawn up the shaft sufficiently to permit reduction of the fracture. Reduction is maintained with the bone-holding forceps while the pin is driven into the distal shaft. When it has been inserted to the correct depth, the pin is broken off flush with the bone or, if it has not already been pre-cut, the pin is cut with pin-cutters or a hacksaw just proximal to the tuberosity.

Methods of improving fracture stability when intramedullary fixation is used in humeral diaphyseal fractures include application of:

- An external fixator
- Cerclage wire
- Stacked or multiple intramedullary pins
- An interlocking nail

External fixator A two-pin unilateral, uniplanar external fixator (Fig. 32.6) can be used to supplement the intramedullary pin to minimise rotation and the risk of non-union. The device is applied to the craniolateral surface of the bone with one pin just distal to the greater tuberosity and the other just proximal to the supratrochlear foramen. The pins should penetrate both cortices of the humerus; the proximal pin can be safely driven through a stab incision in the skin into the humerus but care should be taken to avoid the radial nerve during insertion of the distal pin. The pin is introduced through a separate stab incision close to the distal end of the main surgical incision. It can then be guided between the muscle bellies, avoiding the radial nerve, and driven into the bone. Alternatively, the distal pin can be placed as a transcondylar pin through a stab incision made over the condyle laterally. Once the pins have been placed they are clamped to the external connecting bar and routine wound closure is undertaken.

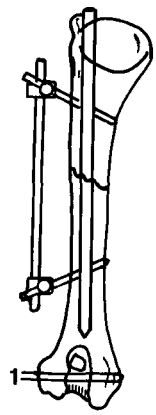

Fig. 32.6 External fixator used to minimise rotation following intramedullary pinning. Point 1 indicates alternative position for distal pin.

Cerclage wire In oblique fractures cerclage, or preferably hemicerclage, wires may be used. They will generally only control rotation adequately if two or more wires can be placed across the fracture line.

Stacked or multiple intramedullary pins In transverse fractures using two or three intramedullary pins, the pins will help to prevent rotation by creating multiple points of fixation. Stacking of intramedullary pins almost completely fills the canal and prevents rotation by creating friction between the endosteal surface and the implants.

Interlocking nail This method of fixation may be useful for comminuted mid-shaft fractures of the humerus, particularly in large dogs.

Postoperative care following intramedullary fixation

Exercise is restricted until fracture union is complete. If an external fixator has been used to supplement an intramedullary pin then it is only required to maximise stability during the initial

healing phase. The fixator is removed after about 3 weeks, before problems with soft tissues are encountered (see below) and by this time rotational stability is usually afforded by the healing callus. The intramedullary pin can then be removed once fracture healing is complete. Some surgeons remove all intramedullary pins and cut the pin proud above the tuberosity to allow easy removal. Others pre-cut pins so that they can be broken off flush with the tuberosity and do not interfere with adjacent soft tissues during fracture healing. Pre-cut pins in cats often remain *in situ* but, in dogs, the pins have a tendency to eventually migrate dorsally because of the looser pin fit. If this happens the pin is easily removed.

The external fixator

The external fixator can be used to stabilise most types of diaphyseal fracture of the humerus. However, it is used on its own most often for comminuted fractures and open fractures. The humerus is surrounded by muscle and, with the exception of two small areas on the proximal and distal ends of the bone (Fig. 32.7a) there are no safe corridors for the introduction of the fixation pins (Marti & Miller, 1994). Transfixion of large muscle masses by pins results in pain and stiffness due to muscle fibrosis. In addition, pin tract infections are more likely to occur. These problems can be minimised by using a standard craniolateral approach to the fracture (see 'Plate fixation' below). Once the fracture is aligned, the pins can be introduced either through the main incision or, preferably, through stab incisions close to the main incision. The pins are then directed between muscle bellies, avoiding the radial nerve, and driven into the bone, penetrating both cortices.

In comminuted fractures of the diaphysis, the intact shaft on either side of the comminuted area is grasped with self-locking bone-holding forceps and traction is exerted until satisfactory length and alignment of the bone is achieved. The fracture site is disturbed as little as possible, the fragments being left *in situ*. A unilateral external fixator is applied to the craniolateral surface of the bone, generally with three pins in the proximal fragment and two in the distal fragment. The distal pin is placed first, and if the distal segment is very short then the pin is placed in the transcondylar position through a stab incision over the condyle. If the distal segment is long enough, the pin can be placed just proximal to the supratrochlear foramen but this should be done as an open approach (see above). The most proximal pin is placed just distal to the greater tuberosity (stab incision). Five clamps are placed on a connecting bar. The proximal and distal clamps are attached to the pins, fracture alignment is rechecked and once it is satisfactory the clamps are tightened so as to maintain reduction. The three centre pins are then driven into the humerus using their clamps as guides (Fig. 32.7b). These pins may be introduced through the main incision as this allows the pins to be guided safely between muscle bellies into the bone. Final adjustment of the clamps is made and the wound closed. Alternatively, the pins may be placed through stab incisions adjacent to the incision, in which case it is often easier to close the wound first to ensure the stab incisions do not create tension in the skin if closure is carried out later. In grade 2 or 3 open, potentially infected fractures, after thorough wound debridement and application of the fixator, the wound is only partially closed to allow drainage.

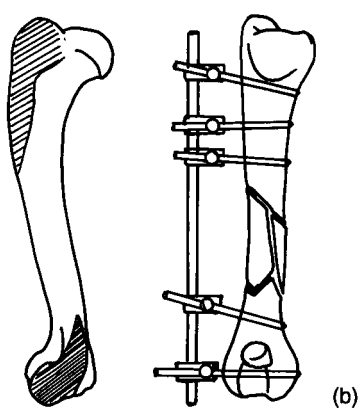

Fig. 32.7 (a) External fixator. Safe areas for pin introduction are hatched (after Marti & Miller, 1994). (b) External fixator used in open and comminuted fractures of the humerus.

Postoperative care of the external fixator

The frame is checked at weekly intervals to ensure that the clamps and/or pins have not loosened and

that pin tract infection has not occurred. A loose pin is accompanied by an increase in pin tract discharge and lameness. If necessary the pin is replaced or removed. Radiographs are taken at 4 and 8 weeks. The fixator is removed once healing is complete.

Plate fixation

Plate fixation is the preferred method of treatment for fractures of the humeral shaft in adult dogs. Ideally, the plate should be placed on the cranial surface of the bone as this is the tension side (Fig. 32.8a). However, the plate may also be placed on the lateral or the medial side of the bone. Choice of site is dependent on the type and position of the fracture and careful preoperative planning is essential. Lag screws are used for initial fixation of oblique or comminuted fractures and the position of these screws in relation to the plate must be considered in the choice of surgical approach. The prognosis following plate fixation is generally good but fracture reduction and insertion of implants, especially in comminuted fractures, is not easy.

Craniolateral approach to the humerus for application of a plate to the cranial aspect of the diaphysis (Fig. 32.8)

A craniolateral skin incision is made from the greater tuberosity to the condyle (Fig. 32.8b). The cephalic vein is identified and ligated (Fig. 32.8c). The brachiocephalicus and brachialis muscles are separated to expose the shaft of the humerus. The radial nerve can be easily identified in the mid-shaft region by separating the brachialis muscle from the lateral head of the triceps muscle (Fig. 32.8d). Once the nerve has been identified between these two muscles it can be followed distally as it runs around the caudal border of the brachialis muscle to emerge on its lateral aspect at the level of the extensor carpi radialis muscle.

During exposure of the cranial surface of the distal shaft, the brachialis muscle is retacted caudally and used to protect the radial nerve. Exposure of the proximal humeral surface is achieved by subperiosteal elevation between the pectoral and deltoideus muscles. Exposure of the cranial aspect of the humeral shaft is maintained with self-retaining retractors or Hohmann retractors while the fracture is reduced. A plate is then contoured and applied to the cranial surface of the humerus (Fig. 32.8e).

Craniolateral approach for application of a plate to the lateral surface of the humeral diaphysis (Fig. 32.9)

Exposure of the humeral diaphysis is as described above but, in addition, the brachialis muscle and the radial nerve are mobilised so that the plate can be slid beneath them on the lateral side of the humerus. The origin of the extensor carpi radialis muscle is freed from the lateral aspect of the condyle to complete exposure of the distal humerus.

Medial approach to the distal third of the humerus (Fig. 32.10)

The medial surface of the humerus is flat and provides an ideal surface for the application of a plate. The medial approach is used in preference to the lateral for dealing with fractures of the distal third of the humeral shaft. A skin incision is made over the medial aspect of the humerus from mid-shaft to the condyle (Fig. 32.10a). The median nerve and biceps muscle are retracted cranially while the ulnar nerve and the medial head of the triceps muscle are retracted caudally to expose the humeral shaft (Fig. 32.10b). Branches of the brachial artery and vein accompany the nerves and all these vital structures should be protected. It is important to note that in the cat the median nerve runs through the supracondylar foramen of the humerus. Exposure of the more proximal regions of the humerus is possible by mobilising the vessels and by subperiosteal elevation of the superficial pectoral and brachiocephalicus muscles.

Case studies:

- Fig. 32.11: comminuted mid-shaft fracture of the humerus
- Fig. 32.12: comminuted fracture of the distal third of the humeral diaphysis
- Fig. 32.13: oblique fracture of the distal third of the humeral diaphysis

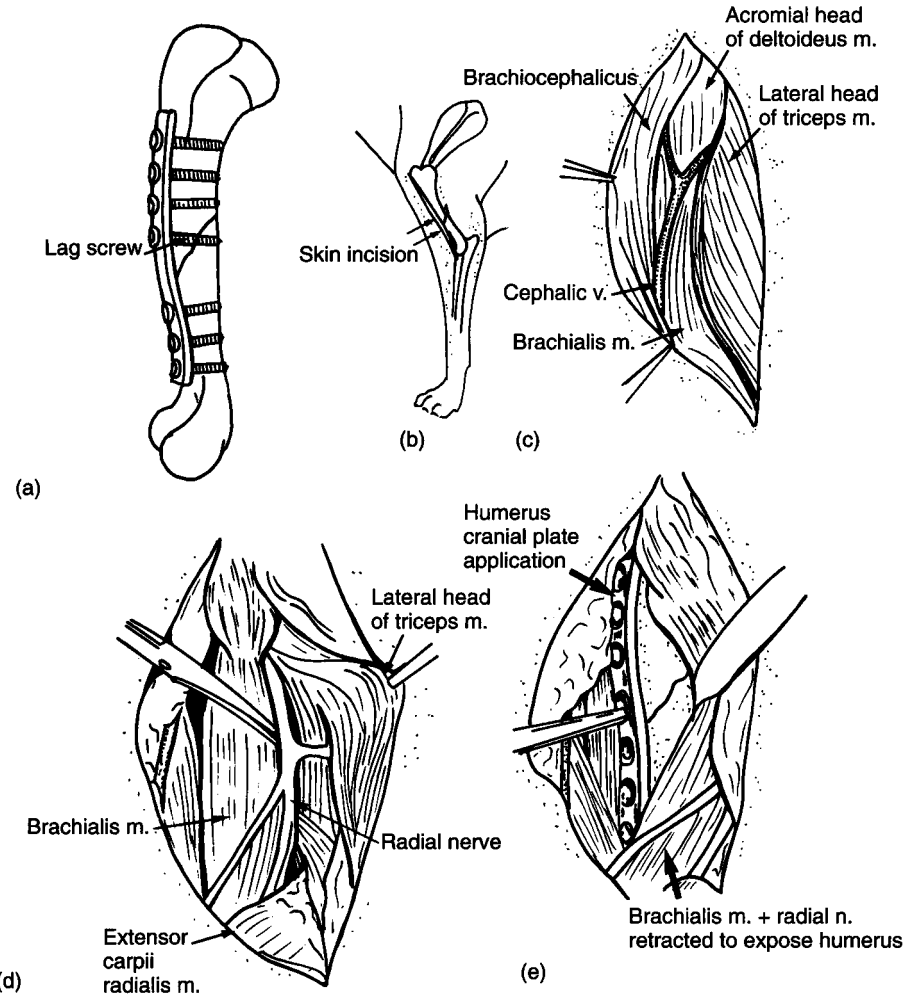

Fig. 32.8 Craniolateral approach for the application of a plate to the cranial aspect of the humeral shaft. See text for detail.

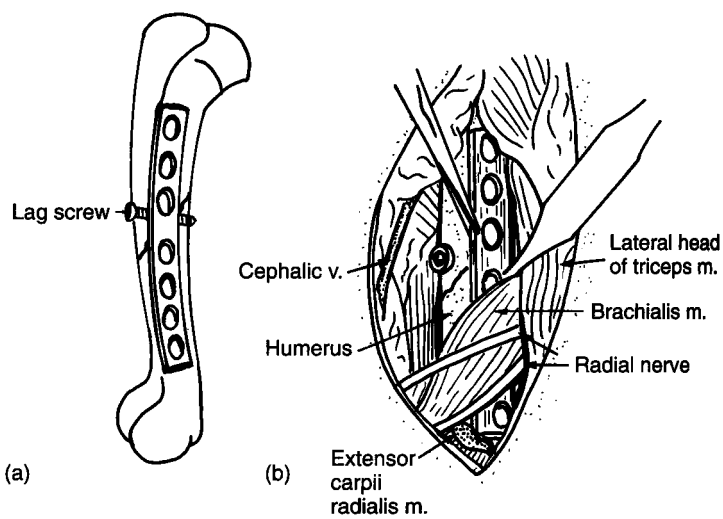

Fig. 32.9 Cranioloateral approach for the application of a plate to the lateral side of the humerus. Initial approach as in Fig. 32.8. Initial fixation is with a lag screw (a), and the plate is contoured to the lateral side of the humerus. Extensor carpii radialis muscle is reflected from the lateral condyle, and the distal end of the plate is slid beneath the brachialis muscle (b).

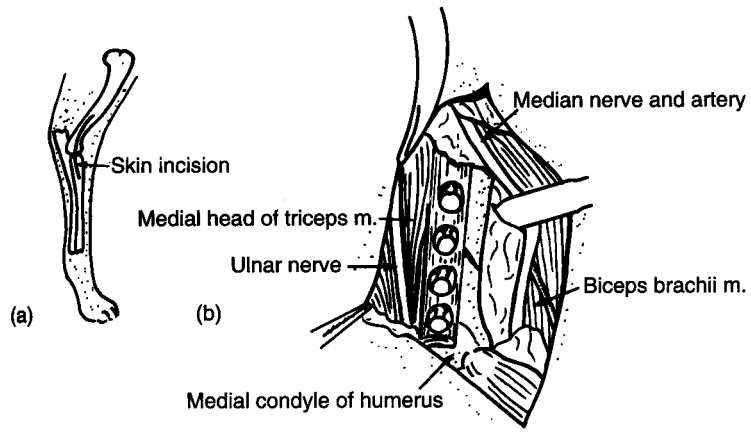

Fig. 32.10 Medial approach to the humerus for application of a plate. (a) Skin incision over the medial condyle. (b) Exposure of the distal third of the humerus in the triangle bounded by the median nerve and artery cranially and the ulnar nerve caudally.

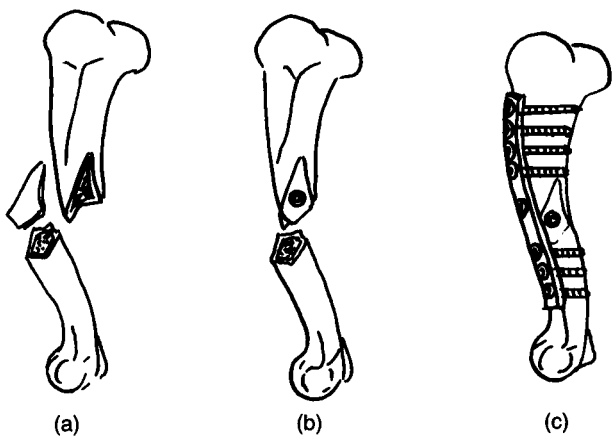

Fig. 32.11 Labrador with comminuted mid-shaft fracture. (a) Tracing of preoperative radiograph. (b) Craniolateral approach, fragment reduced, lag screw fixation with a 2.7 mm cortex screw. (c) Cranial application of a plate, 8 hole 3.5 DCP (dynamic compression plate).

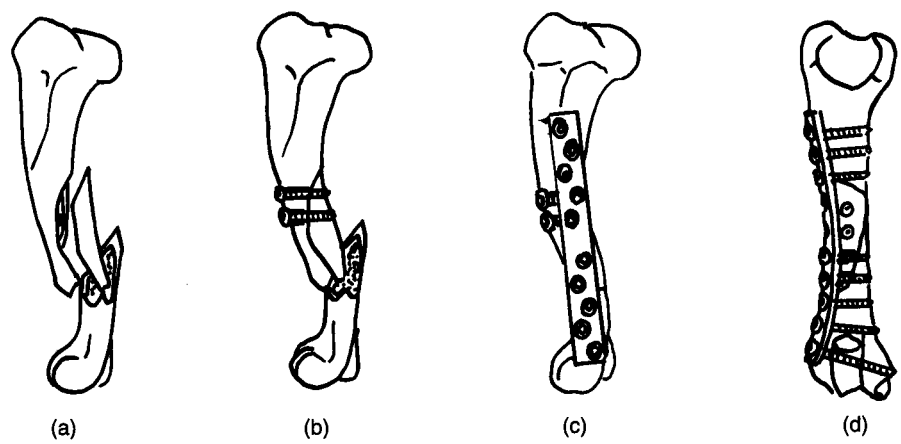

Fig. 32.12 Newfoundland with comminuted fracture involving the distal third of the shaft. (a) Tracing of preoperative radiograph. (b) Craniolateral approach, reconstruction of shaft using two lag screws (3.5 mm cortex screws). (c) Application of a plate to the lateral side of the humerus (Broad 4.5 DCP [dynamic compression plate]). (d) Screws 4 and 5 cross a fracture line and are placed as lag screws.

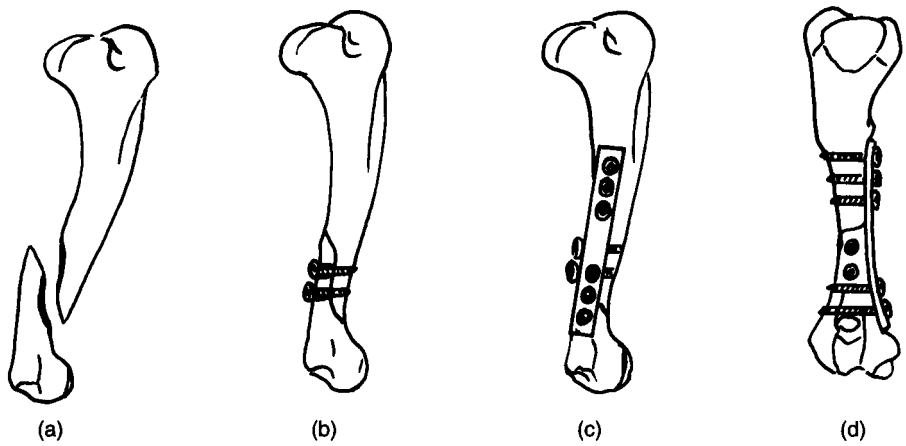

Fig. 32.13 Chow oblique fracture involving distal third of the shaft. (a) Tracing of preoperative lateral radiograph. (b) Medial approach, reconstruction of shaft using two lag screws (2.7 mm cortex screws). (c) Application of a plate to the medial surface of the humerus (6 hole 3.5 DCP [dynamic decompression plate]). (d) Craniocaudal view showing medial application of the plate.

Postoperative care following plate fixation

Exercise is restricted for 6–8 weeks or until fracture healing is radiographically complete. Plates on the humerus are generally left *in situ*. It is often difficult to identify the radial nerve in scar tissue resulting from the initial surgery and, therefore, plate removal carries a risk of iatrogenic damage to this nerve.

Supracondylar fractures

In supracondylar fractures, the fracture line passes through the supratrochlear foramen. These fractures tend to be transverse or oblique in nature. In immature dogs the fracture takes the form of a Salter Harris type II fracture separation of the growth plate. Supracondylar fractures should be accurately reduced and rigidly stabilised because of their close proximity to the elbow joint. An intramedullary pin, used in conjunction with a Kirschner wire to prevent rotation, is the simplest method of fixation (Brinker, 1974). Alternatively, in large dogs a plate can be applied to the medial supracondylar ridge of the humerus (Braden, 1975).

Surgical approach

The dog is positioned in dorsal recumbency with the affected leg pulled forward (see later in Fig. 32.25a). A medial approach is used to expose the fracture (see above under 'Plate fixation' of diaphyseal fractures) whilst protecting the median and ulnar nerves! The skin incision should be made towards the caudal aspect of the elbow to allow skin to be reflected from both sides of the joint.

The humeral condyle is grasped with pointed AO reduction forceps, the distal humeral shaft is held with self-locking bone-holding forceps. The bone fragments are tilted caudally, toggled against each other and then pushed cranially until reduction is achieved. If there is a medial oblique supracondylar fragment then small pointed reduction forceps can be used to hold this fragment in reduction with the shaft. Having checked that reduction is possible, the fracture site is hinged open to expose the medullary cavity of the medial supracondylar ridge. An intramedullary pin is introduced retrogradely into the cavity and directed laterally up the humeral shaft to emerge on the lateral side of the greater tuberosity. The Jacob's chuck is then attached to the proximal end of the pin which is pulled up until just the tip is

visible at the fracture surface. The fracture is reduced and then, with the elbow extended, the pin is driven down into the medial condyle. Finally, a Kirschner wire is driven up through the lateral aspect of the condyle, obliquely across the fracture and into the medial cortex of the humerus (Figs 32.14 and 32.15a). This second point of fixation prevents rotation. Both pins are cut close to the bone after insertion, with or without bending of the Kirschner wire to reduce the likelihood of migration.

Postoperatively, exercise is restricted until fracture healing is radiographically complete (4–6 weeks) and the pins can then be removed.

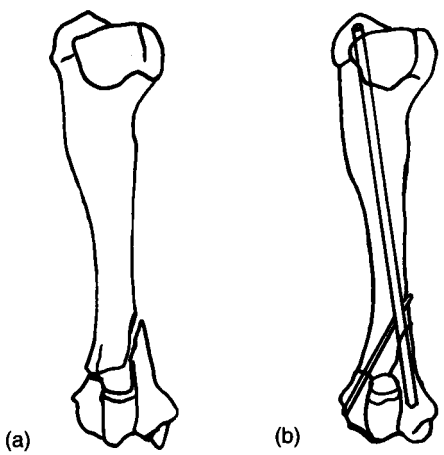

Fig. 32.14 (a) Supracondylar fracture. (b) Fixation with an intramedullary pin and Kirschner wire.

Other treatment options in supracondylar fractures

Supracondylar fractures which involve the growth plate in immature dogs generally take the form of Salter Harris type II injuries. The medial cortex is usually fractured obliquely and this component of the fracture can be readily stabilised with a Kirschner wire or a lag screw placed transversely from medial to lateral, proximal to the growth plate. After this initial fixation, Kirschner wires are driven up from the medial and the lateral aspects of the condyle and across the fracture site in a cruciate pattern (Fig. 32.15b).

In large dogs, lag screw fixation or plate fixation can be used for supracondylar fractures (Fig. 32.15c,d).

Condylar fractures

Condylar fractures can be broadly classified as lateral, medial or intercondylar. The relative incidence of these catagories, given below, is taken from reviews of 133 cases (Denny, 1983; illustrated in Fig. 32.16) and of 39 cases (Butterworth, 1992; detailed in Table 32.1), respectively. Condylar fractures usually result from a violent upward stress transmitted through the head of the radius onto the humeral trochlea. The lateral component of the humeral condyle appears to have the weakest attachment to the humeral shaft and fractures most frequently (56 and 57% of cases, respectively). In the face of even greater stress the

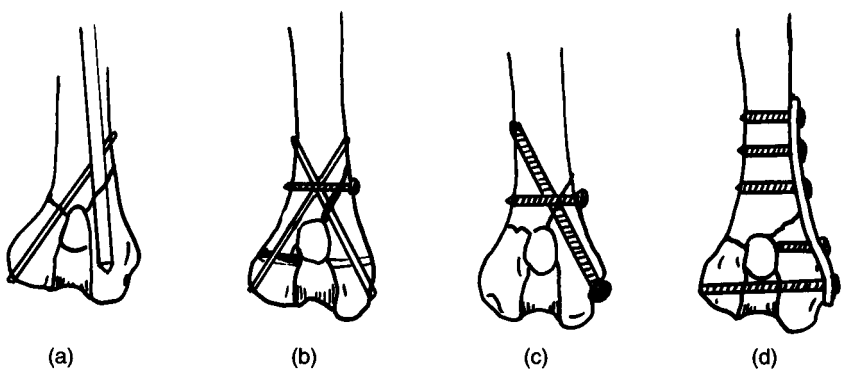

Fig. 32.15 Methods of fixation for a supracondylar fracture. (a) Intramedullary pin and Kirschner wire, (b) lag screws plus crossed Kirschner wires, (c) lag screws, (d) plate fixation.

Table 32.1 Relative incidence of the various catagories of 42 humeral condylar fractures and the age distribution of the dogs involved (Butterworth, 1992).

Age	Lateral condylar	Medial condylar	Intercondylar	Total
<4 months	7	–	–	7
4–6 months	5	1	1	7
6–8 months	1	–	3	4
8–10 months	–	–	1	1
10–12 months	–	–	1	1
1–3 years	2	–	1	3
3–5 years	2	–	4	6
5–7 years	4	–	5	9
7–9 years	2	–	1	3
9–10 years	1	–	–	1
Total	24	1	17	42

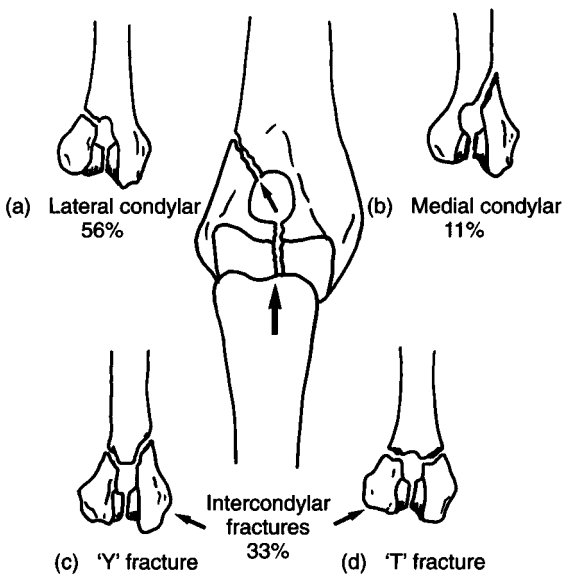

Fig. 32.16 Condylar fracture of the humerus – relative incidence based on 133 cases (Denny, 1983).

medial part of the condyle is also sheared off giving rise to an intercondylar ('Y' or 'T') fracture (33 and 40% of cases, respectively). Solitary fractures of the medial condyle occurred far less frequently (11 and 3% of cases, respectively). Spaniel breeds of dog appear to be more prone to condylar fractures. Incomplete ossification of the humeral condyle, predisposing to fracture, has been demonstrated in Cocker Spaniels and Brittany Spaniels and it has been suggested in the Cocker Spaniel that incomplete ossification of the humeral condyle may be a genetic disease with a recessive mode of inheritance (Marcellin-Little *et al.*, 1994). In the UK, such condylar fissures have been noted in association with elbow lameness, with the most frequently observed breed seeming to be the Springer Spaniel (authors' unpublished observations). Falls, jumping and sudden turns at exercise are the most common causes of lateral or medial condylar fractures. Intercondylar fractures are more likely to be caused in road traffic accidents. Lateral and medial condylar fractures affect predominantly immature dogs (peak age incidence at 4 months) whilst intercondylar fractures tend to be more evenly distributed between skeletally mature and immature dogs (Table 32.1). Condylar fractures are articular fractures and as such require surgical treatment with accurate anatomical reduction and stable internal fixation if normal joint function is to be restored.

Lateral condylar fractures

In the radiographic assessment of condylar fractures it is essential to take both mediolateral and craniocaudal views of the elbow. A lateral condylar fracture can be missed on a mediolateral view but should be obvious on the craniocaudal view. Lateral condylar fractures are stabilised with a transcondylar lag screw. A Kirschner wire is used

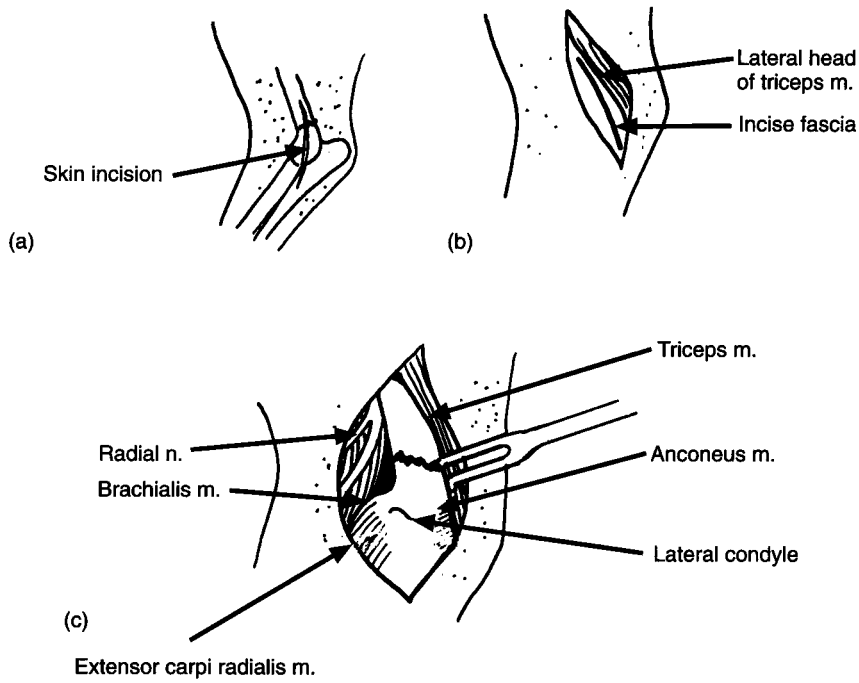

Fig. 32.17 Surgical approach for exposure in lateral condylar fractures. See text for details.

for initial fixation and this may be left in place to provide a second point of fixation, minimising rotation of the fragment (see later in Fig. 32.19). Failure to treat the fracture surgically results in medial luxation of the elbow because lateral support for the joint is lost. Malunion or non-union of lateral condylar fractures cause permanent joint deformity. The range of elbow movement remains limited and varying degrees of lameness persist. The prognosis is obviously better in immature dogs because of their ability to remodel the malunion and in these cases the functional end results can be surprisingly good despite permanent joint deformity, though this is not to say the dogs would not have been far better with successful surgical treatment.

Operative technique

Closed reduction using a condyle clamp and a single transcondylar lag screw placed through a stab incision is possible if the animal is presented within a few hours of injury. However, in the majority of cases an open surgical reduction is carried out (Fig. 32.17). A skin incision is made directly over the condyle laterally (Fig. 32.17a). The lateral head of the triceps muscle is exposed and the deep fascia along its cranial border is incised (Fig. 32.17b). The muscle is retracted to expose the condyle (Fig. 32.17c). The radial nerve emerges between the lateral head of the triceps and the brachialis muscle just proximal to the incision, but provided dissection is limited to the soft tissues over the condyle and the supracondylar ridge, there should be little risk of nerve damage during exposure. Using a periosteal elevator, any remaining muscle attachments are cleared from the adjacent surfaces of the fractured supracondylar ridge. The condylar fragment is then rotated laterally to allow removal of haematoma and granulation tissue from the fracture site. Methods of maintaining reduction of lateral condylar fractures after reduction are shown in Fig. 32.18. There are two methods of preparation of the drill hole for the transcondylar lag screw:

(1) After reduction of the condyle the drill hole for the transcondylar lag screw is commenced from a point immediately below and just in front of the most prominent point on the lateral aspect of the condyle and is directed towards the corresponding spot on the medial aspect of the condyle (Fig. 32.19a). A cortex screw is usually used for fixation. With this type of screw, the hole in the lateral fragment must be overdrilled (Fig. 32.19b) to the same diameter as the screw to ensure that the lag effect is achieved as the screw is tightened, giving compression of the fracture site (Fig. 32.19c). In young patients the pilot hole may be only partially tapped when non-self-tapping screws are used, as cortical screws will usually force their way through soft cancellous bone. In very young puppies with soft bone, a partially threaded cancellous screw may be used for fixation, in which case only a transcondylar pilot hole is drilled. Provided *all* the threads of the screw grip in the medial fragment the lag effect will be achieved (Fig. 32.19d). Also in very young patients with soft bone it may be advisable to place the screw through a washer to help prevent the head sinking too far into the condyle.

(2) The most accurate way of placing the lag screw is using the 'inside out', or retrograde, method (Fig. 32.20). After exposure of the fracture site, the lateral fragment is completely rotated out of the incision on its collateral ligament to allow exposure of the fractured trochlea surface (Fig. 32.20b). The glide hole for the screw can then be accurately drilled retrogradely starting in the centre of the fractured trochlea and drilling from this point to the lateral surface of the lateral fragment (Fig. 32.20c). The appropriate sized drill sleeve is introduced into the glide hole from the lateral side. The fragment is rotated back into its normal position and held in reduction.

Provided the fracture of the supracondylar ridge is accurately reduced it can be assumed that reduction of the intra-articular fracture is also accurate. The simplest method of reducing the fracture is to exert pressure on the condyle with

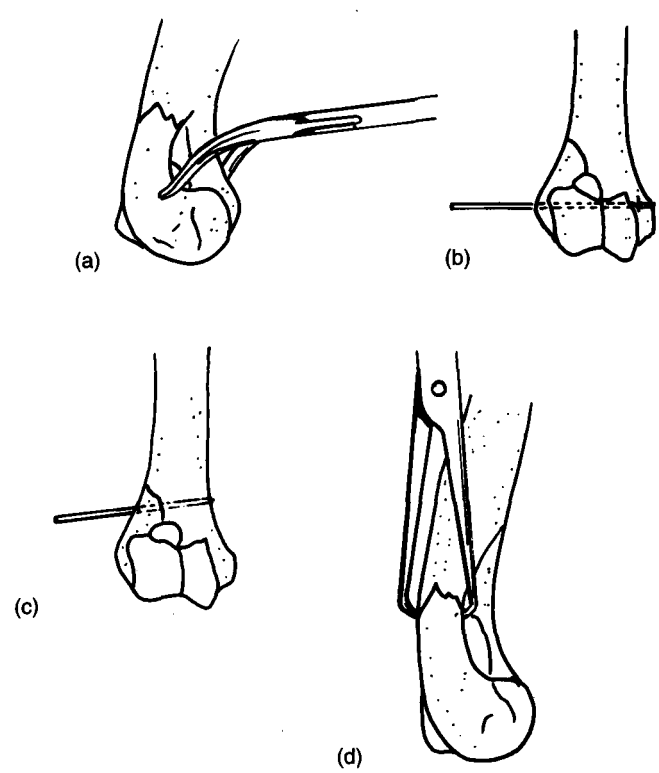

Fig. 32.18 Methods of maintaining reduction of lateral condylar fracture during insertion of the transcondylar lag screw. (a) AO pointed reduction forceps, (b) transcondylar Kirschner wire, (c) supracondylar Kirschner wire, (d) Allis tissue forceps.

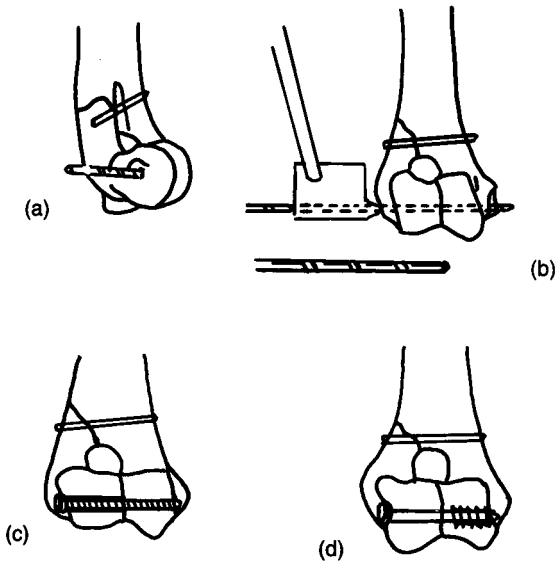

Fig. 32.19 Lag screw fixation of lateral condylar fractures.
(a) Initial fixation with Kirschner wire across supracondylar fracture site. The drill hole for the screw is commenced just below and just in front of the most prominent point on the lateral condyle. The hole should emerge at the corresponding spot on the medial condyle.
(b) If a cortical screw or Sherman screw is used for fixation, overdrill the lateral condyle to produce the lag effect.
(c) Transcondylar cortical screw used as a lag screw. A second lag screw may be placed across the supracondylar fracture line instead of a Kirschner wire.
(d) If a partially threaded cancellous screw is used as a lag screw there is no need to overdrill the lateral condyle provided that all the screw threads grip in the medial condyle.

finger and thumb and then maintain reduction with pointed AO reduction forceps. The lateral fragment does have a tendency to rotate caudally, especially in the case of the left humerus, due to the clockwise rotation of the drill and/or bone screw. Application of Allis tissue forceps across the fractured supracondylar ridge helps to prevent this. A Kirschner wire may now be placed across the supracondylar fracture to maintain reduction and prevent rotation while the lag screw is inserted. The glide hole has already been prepared in the lateral fragment and the drill sleeve is *in situ*. Next, the smaller drill bit is passed through the sleeve and used to drill the pilot hole in the medial part of the condyle. The length of screw is assessed with a depth gauge, a thread is cut in the pilot hole with a tap (unless a self-tapping screw is used or the patient is very young – see above under (1)) and then an appropriately sized screw is inserted. Unless the Kirschner wire used is parallel to the screw then it should be removed to allow final tightening of the latter since a non-parallel wire will prevent the screw from compressing the fracture fragments. The Kirschner wire in the supracondylar portion of the fracture is cut off flush with the bone, with or without first being bent over to reduce the likelihood of subsequent migration. Alternatively, it may be replaced by a second lag screw (in any dog larger than a toy breed) as this may provide better fixation of the supracondylar fracture (Fig. 32.19c). It is important to have a second point of fixation to prevent rotation of the condylar fragment on the transcondylar lag screw.

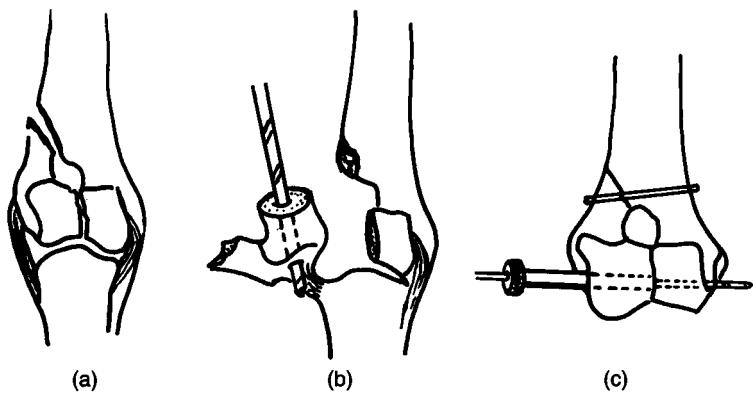

Fig. 32.20 'Inside out' method of preparing a transcondylar screw hole. (a) Preoperative view. (b) Condyle rotated out laterally on collateral ligament, and glide hole drilled from medial to lateral. (c) Reduce and stabilise the fracture with a Kirschner wire. The drill sleeve is inserted into the glide hole to allow accurate placement of the pilot hole through the medial condyle.

Postoperative care

A support bandage may be applied for 5 days, particularly if the region is swollen. Movement of the joint is important following repair of any articular fracture to minimise stiffness and encourage nutrition and healing of the articular cartilage. Gentle passive flexion and extension of the joint and controlled exercise should be recommended. Exercise should be restricted to walks on a lead for 4–6 weeks. Implants are generally left *in situ*. Lateral condylar fractures carry a good prognosis provided they are correctly reduced and stabilised. Some 77% go on to regain full limb function with an average recovery time of 4 weeks (range, 2 to 8 weeks) (Denny, 1983).

Non-union lateral condylar fractures

These tend to be seen in mature dogs. The usual causes are:

- Failure to treat the fracture by internal fixation
- Failure to provide adequate internal fixation
- Failure to control exercise during fracture healing
- Occasionally infection is the cause of non-union

Lateral condylar fractures should be stabilised with a transcondylar lag screw used in conjunction with a Kirschner wire or second lag screw to prevent rotation of the fragment. If a single lag screw is used alone, the condylar fragment may rotate on the screw and consequently the implant may loosen or fracture before bone union occurs. Dogs treated in this way may have reasonable limb function for several weeks following surgery. Lameness then becomes worse with pain and crepitus evident on palpation of the elbow. Radiographs show non-union of the condylar fragment and often bone lysis around the screw indicating movement or low-grade infection. In some cases the screw starts to 'back out' of the bone. Fracture of the screw is seen in chronic non-unions (illustrated in Fig. 32.21a).

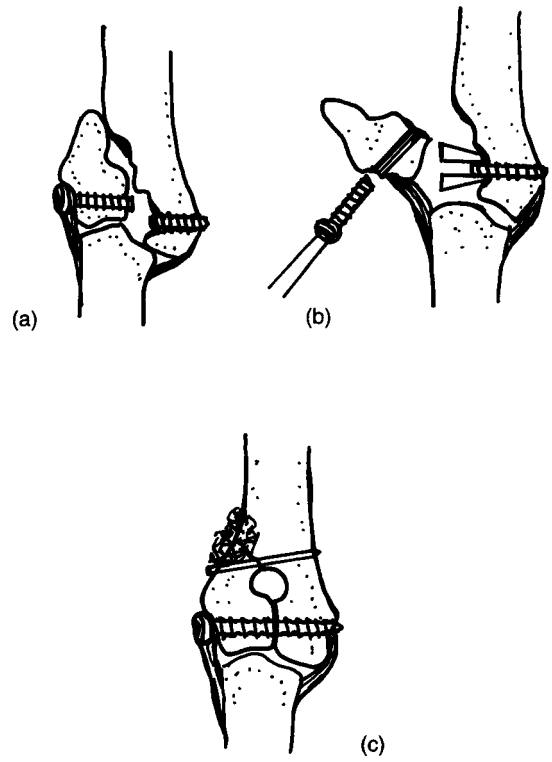

Fig. 32.21 Treatment of a non-union lateral condylar fracture and removal of a broken screw. (a) Non-union lateral condylar fracture with broken screw. (b) Removal of broken halves of screw: proximal half removed with screwdriver, lateral condyle rotated to expose intercondylar fracture site and medial portion of screw. End of screw gripped with rongeurs and twisted out. (c) Fixation of transcondylar lag screw plus Kirschner wire or second lag screw. Autogenous cancellous bone graft packed around supracondylar fracture site.

Treatment options

(1) If the screw is loose, it is either replaced by a larger-diameter screw or redirected into the medial part of the condyle. A transcondylar Kirschner wire and/or supracondylar lag screw should also be placed to prevent rotation.

(2) If the screw has broken then an attempt should be made to remove the broken halves (Fig. 32.21b). Removal of the lateral condylar half of the screw should present no problem but retrieval of the tip of the screw can be awkward. If the latter is protruding into the soft tissues on the medial side of the condyle then a small stab incision is made over it, the tip of the screw is grasped with rongeurs or orthopaedic pliers, and the broken end of the screw is gradually twisted out of the bone. If this is not possible because all the

screw threads are buried in the condyle then a lateral approach is used to remove both halves of the screw. Once the shank of the screw has been removed, the lateral fragment is freed from its dorsal soft tissue attachments and rotated out of the incision to allow inspection of the medial fracture surface from which a few threads of broken screw usually protrude. The broken end of the screw is grasped with rongeurs and removed (Fig. 32.21b). Once the broken screw has been removed from the condyle, the intercondylar fracture surfaces are 'freshened up' and reduced. Fixation is then achieved with another transcondylar lag screw and a supracondylar Kirschner wire or lag screw (Fig. 32.21c). In large breed dogs the application of a bone plate to the lateral supracondylar ridge may be appropriate.

In long-standing, non-union lateral condylar fractures associated with displacement it is preferable to use the transolecranon approach to the elbow (see under 'Intercondylar ("Y" or "T") fractures') to permit accurate reduction of the fracture. Often much fibrocartilaginous callus, as well as the broken implants, must be removed before reduction can be achieved. A cancellous bone graft, taken from the proximal humerus, is packed around the supracondylar fracture site to speed up fracture healing (Fig. 32.21c).

Medial condylar fractures

The same principles of treatment apply as for lateral condylar fractures. However, the medial condylar fragment is often large enough to accept at least two lag screws placed from medial to lateral, one transcondylar and the remainder placed proximal to the supratrochlear foramen (Fig. 32.22). Positioning and exposure has already been described above under 'Plate fixation' of distal diaphyseal fractures (p. 347). Postoperative care and prognosis are the same as for lateral condylar fractures.

Ununited medial epicondyle (UME) and fracture of the medial epicondyle

Failure of the medial epicondyle to unite with the humerus is occasionally encountered as a fusion defect in immature dogs of the larger breeds. Labrador Retrievers are affected most frequently.

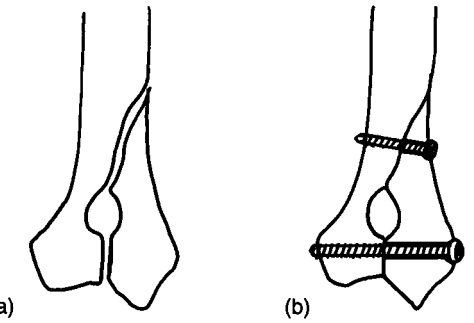

Fig. 32.22 (a) Medial condylar fracture. (b) Fracture stabilised with two lag screws.

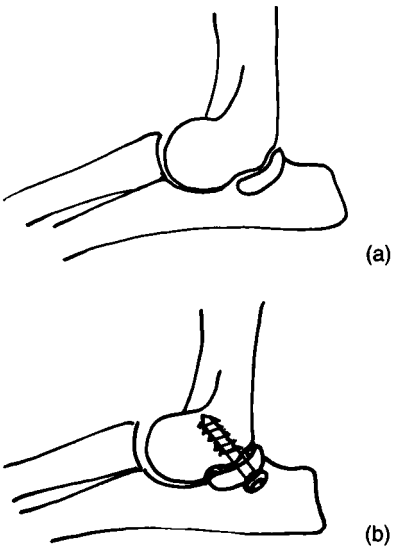

Fig. 32.23 (a) Ununited medial epicondyle (UME). (b) Postoperative radiograph showing lag screw fixation with a cancellous screw.

The condition is often bilateral and several members of the same litter may be affected. History and clinical signs are similar to other forms of elbow osteochondrosis (see Chapter 33). Radiographic examination shows the ununited epicondyle as a discrete fragment on the caudal aspect of the medial condyle (illustrated in Fig. 32.23a).

Fracture of the medial epicondyle is also occasionally encountered in immature dogs but here the onset of lameness is acute. In both the

developmental and traumatic lesions the epicondylar fragment tends to be distracted by the attached antebrachial flexor muscles. If the bone fragment is large enough, it is lagged into place with a bone screw (Fig. 32.23b). Smaller fragments causing persistent lameness are removed and the muscles reattached to the adjacent fascia.

Intercondylar ('Y' or 'T') fractures

The humeral intercondylar fracture is often referred to as a 'Y' fracture if the supracondylar ridges are fractured obliquely, and a 'T' fracture if the ridges are fractured transversely. Although intercondylar fractures are traditionally divided into these two broad groups, they can be further divided into five types (Fig. 32.24, Bardet *et al.*, 1983). Successful treatment of these fractures can be difficult and may present a real challenge even to the most experienced orthopaedic surgeon. Good exposure of the fracture is essential to achieve accurate anatomical reduction of the fragments. Most frequently, a caudal approach with osteotomy of the olecranon and dorsal reflection of the triceps muscle mass is used. There is some concern over the level of soft tissue trauma caused by this approach and its affect on long-term range of motion in the joint. Therefore, in selected cases it may be possible to accomplish accurate fracture reconstruction using a combination of medial and lateral approaches, thus avoiding the reduction in elbow flexion often seen following a caudal approach. Reconstruction and fixation of the condylar fragments is achieved with a transcondylar lag screw used in combination with a Kirschner wire. The condyle is then attached to the shaft with a plate applied to the medial supracondylar ridge of the humerus (see later in Fig. 32.26). Occasionally, particularly in very large dogs, a second plate is applied to the lateral supracondylar ridge. In young puppies, pins or Kirschner wires can be used to attach the condyle to the shaft but plate fixation gives the best results, particularly in older patients. If olecranon osteotomy has been used, it is repaired with a screw or Kirschner wires used in combination with a wire tension band.

Surgical technique

If a combination of medial and lateral approaches is used then the techinques involved are as de-

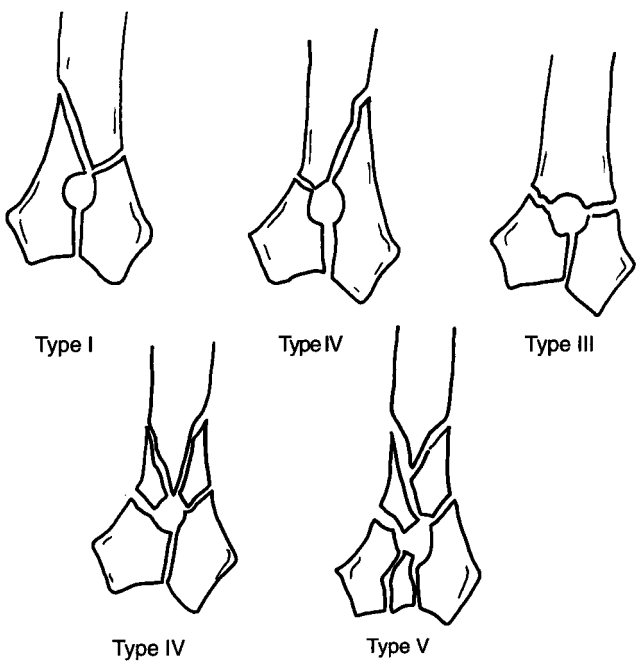

Fig. 32.24 Classification of intercondylar humeral fractures (after Bardet *et al.*, 1983).

scribed previously (Figs 32.9 and 32.10). For a caudal approach the dog is placed in dorsal recumbency with the affected leg pulled cranially (Fig. 32.25a). A skin incision is made over the caudomedial aspect of the elbow, the subcutaneous fat and fascia are incised and undermined to allow reflection of skin from both sides of the elbow. The fascia along the cranial border of the medial head of the triceps is incised, and the ulnar nerve is identified and retracted from the olecranon. The cranial margin of the lateral head of the triceps is also freed from its fascial attachments. The proximal shaft of the ulna is exposed by separating the flexor carpi ulnaris and extensor carpi ulnaris muscles. A hole is drilled transversely through the ulna with a 2mm drill just distal to the elbow. A length of orthopaedic wire (18 or 20 gauge) is passed through the hole and fashioned into a loop. This wire will be used later as a tension band but at this stage it makes a useful handle for an assistant to exert traction on the ulna during exposure and reduction of the humeral condyle. If a screw is to be used to repair the olecranon osteotomy, the screw hole should be prepared and tapped prior to osteotomy. Transverse osteotomy of the olecranon is performed with a saw or Gigli wire distal to the tendon of insertion of the triceps on the olecranon and proximal to the anconeal process (Fig. 32.25b). The ulnar nerve should be protected during this procedure. The olecranon is reflected dorsally with the attached triceps muscle mass and remnants of the anconeus and joint

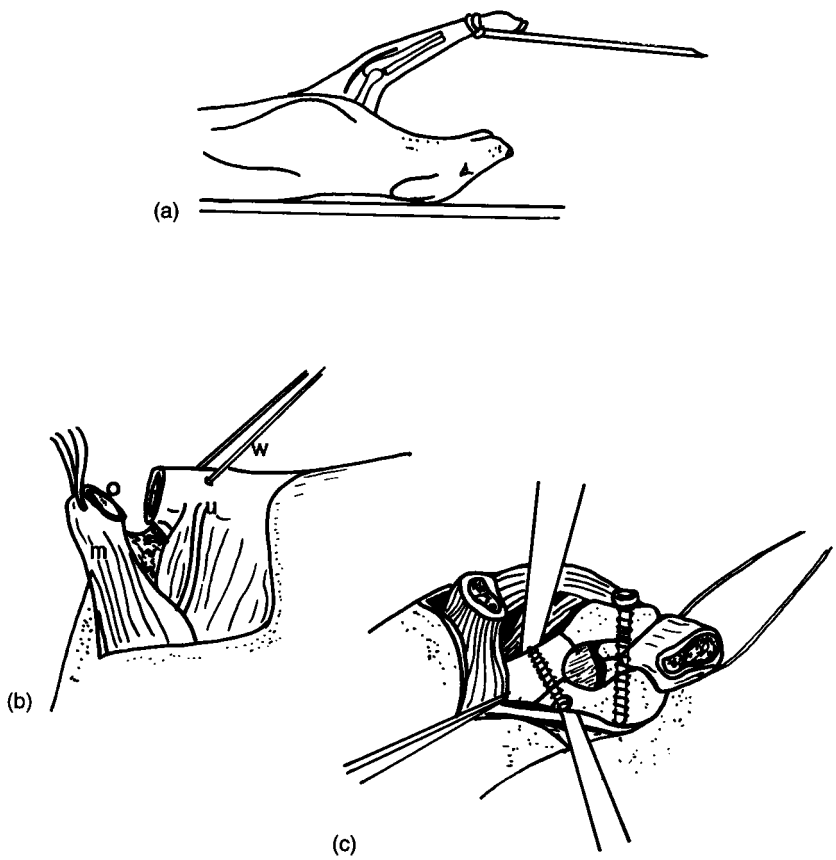

Fig. 32.25 Transolecranon approach for reduction and fixation of intercondylar fractures. (a) Caudomedial skin incision over olecranon. (b) Olecranon osteotomy (o), wire placed in ulna (w), triceps muscle (m) reflected and ulnar nerve (u). (c) Exposure of humeral condyle and reconstruction of distal humerus with lag screws.

capsule are reflected from the caudal aspect of the elbow to complete the exposure of the condylar fragments.

The condylar fragments are reduced, ensuring accurate reconstruction of the articular surface. In order to achieve this an assistant may exert traction on the ulnar wire to steady the elbow and draw the fragments away from the humeral shaft which often lies between them. The medial condylar fragment is held with small bone-holding forceps while the lateral fragment is aligned in its normal position. The fragments are then held in reduction with pointed AO reduction forceps. The proximal ends of the condylar fragments are transfixed with a Kirschner wire or, in the case of a 'Y' fracture, a lag screw (Figs 32.25c and 32.26b–d). Once this area has been stabilised the articular margins of the fracture can be checked again and final adjustments in reduction made before inserting a transcondylar lag screw from lateral to medial (Fig. 32.26c, see also lateral condylar fracture repair, p. 353).

The distal shaft of the humerus is grasped with self-locking bone-holding forceps while the condyle is grasped with pointed reduction forceps. Reduction of the supracondylar portion of the fracture is achieved by a combination of direct traction and toggling the fracture surfaces against each other. If possible the condyle is temporarily attached to the shaft at this stage with a Kirschner wire placed obliquely across the supracondylar fracture line (Fig. 32.26d). A plate (3.5 or 2.7 DCP [dynamic compression plate]) is then applied to the caudal aspect of the medial supracondylar

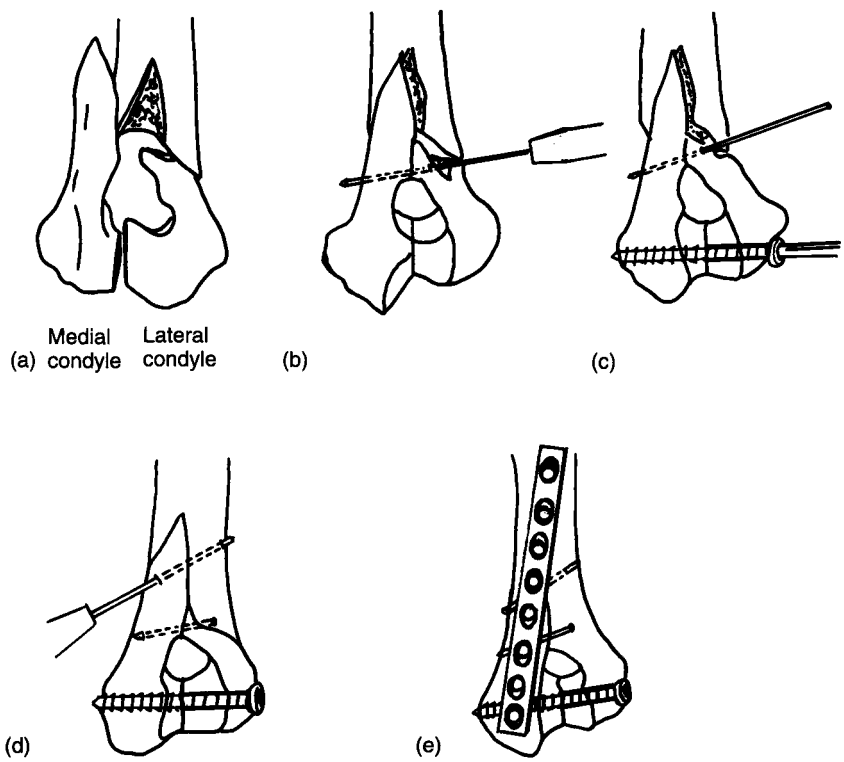

Fig. 32.26 Stages in reduction and fixation of a 'Y' fracture using Kirschner wires, transcondylar lag screw and plate applied to medial supracondylar ridge of the humerus. (a) 'Y' fracture. (b) Initial fixation with a Kirschner wire. (c) Transcondylar lag screw placed from lateral to medial. (d) Condyles fixed to shaft with Kirschner wire or lag screw. (e) Application of a plate to the medial supracondylar ridge of the humerus.

ridge. This is a flat surface and the plate should require little or no contouring to the surface of the bone (Fig. 32.26e). Alternatively, the plate may be applied to the medial aspect of the distal humerus. Although this is more awkward to achieve, a larger plate can usually be accommodated, providing increased support for the fracture. If a Kirschner wire is not used then, having ensured that it is possible to reduce the supracondylar fracture site, the fragments are disengaged and the medial fragment rotated laterally so that the medial supracondylar ridge is easily visible. The distal end of the plate is attached to the caudal (or medial) aspect of the medial condylar ridge. It is usually possible to place three screws distally, especially if a 2.7 DCP is used for fixation, but care should be taken to ensure that the most distal screws do not penetrate the articular surface. Having attached the plate distally, the free, proximal end can be used as a lever arm to complete reduction of the fracture site. The plate is held against the bone with self-locking bone-holding forceps while the first two screws are inserted proximal to the fracture site. Once stability is achieved the forceps can be removed and the rest of the screws placed. In large dogs a second smaller plate can be applied to the lateral supracondylar ridge to improve stability.

During wound closure the olecranon osteotomy is repaired with a lag screw or two Kirschner wires used in combination with the pre-placed ulna wire which is used as a tension band (see Chapter 33, Fig. 33.28). The lag screw is preferred as it causes less soft tissue interference and can usually be left *in situ*. The Kirschner wires, in contrast, may loosen causing local soft tissue problems leading to a requirement for their removal. The triceps fascia is repaired on both lateral and medial sides (care should be taken to avoid the ulnar nerve in sutures on the medial side). The rest of the wound closure is routine.

Postoperative care

A Robert Jones bandage may be applied for 5 days postoperatively to provide support and control postoperative swelling. Otherwise management is as described under 'Lateral condylar fractures' (p. 356). Implants are generally left *in situ* unless loosening causes soft tissue problems. The proximal ends of Kirschner wires used for repair of olecranon osteotomy are the most common cause of problems. In such cases any associated lameness tends to resolve once these wires have been removed.

Prognosis

Prognosis is favourable for return to reasonable function in the majority of animals (64–70%) provided accurate anatomical reduction and good stability is ensured allowing early pain-free elbow mobility (Denny, 1983; Anderson *et al.*, 1990).

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Chapter 33
The Elbow

Anatomy

The anatomy of the elbow is illustrated in Figs 33.1–33.5. The elbow is a composite joint in which the humeral condyle articulates with the head of the radius, the humeroradial joint, and also with the trochlear or semilunar notch of the ulna, the humeroulnar joint. The humeroradial joint transmits most of the weight-bearing load through the elbow while the humeroulnar part maintains joint stability in the sagittal plain. The elbow is a ginglymus (hinge) joint capable of flexion, extension and a limited amount of rotation. Strong collateral ligaments and the anconeal process, which fits deep into the olecranon fossa of the humerus, prevent medial and lateral movement of the elbow. Soft tissue structures of surgical importance are illustrated in Figs 33.4 and 33.5, while the normal radiographic anatomy of the elbow in the immature dog is shown in Figs 33.6 and 33.7. The growth plates of the distal humerus, proximal radius and ulna all close at approximately 7 months (range 5 to 8 months) (Sumner-Smith, 1966).

Conditions of the elbow joint

Conditions of the elbow can be divided into two groups:

- *Developmental*
  - 'Congenital' luxation
  - Osteochondritis dissecans (OCD)
  - Fragmented medial coronoid process (FCP)
  - Ununited anconeal process (UAP)
  - Ununited medial epicondyle (UME)
  - Luxation/subluxation secondary to growth disturbances (see Chapter 34)
- *Acquired*
  - Traumatic luxation
  - Fractures of the distal humerus (see Chapter 32)
  - Ulnar fracture with radial head dislocation (Monteggia fracture)
  - Luxation of the radial head in cats
  - Fractures of the proximal ulna and olecranon
  - Fracture of the anconeal process
  - Fractures of the radial head
  - Avulsion of the triceps tendon of insertion

Congenital luxation of the elbow

Congenital luxation of the elbow is uncommon. Two types of deformity are recognised (Campbell, 1979):

- Type 1 – lateral rotation of the proximal ulna
- Type 2 – caudolateral luxation of the radial head

Type 1 – lateral rotation of the proximal ulna

This type of congenital elbow luxation is recognised at, or within a few weeks of, birth. Small breeds of dog are affected and the condition has been recorded in the Sheltie, Pekingnese, Cocker Spaniel, Yorkshire Terrier, Miniature Poodle and Miniature Pinscher. Lateral or outward rotation of the proximal ulna results in loss of contact between the trochlear notch and the humeral condyle. There is rotation and lateral deviation of the antebrachium and the range of elbow extension is limited.

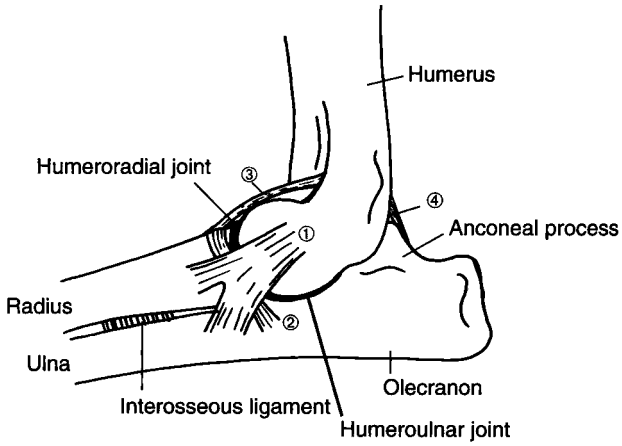


Fig. 33.1 Lateral aspect of the left elbow. 1 – Lateral collateral ligament; 2 – annular ligament; 3 – oblique ligament; 4 – olecranon ligament.

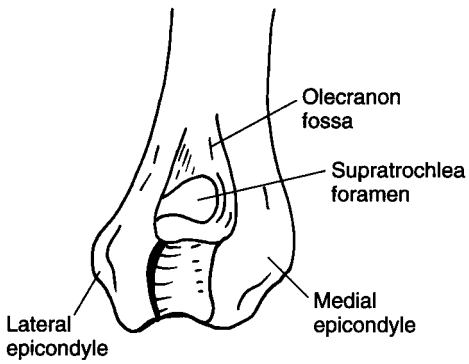


Fig. 33.2 Caudal aspect of the left humerus.

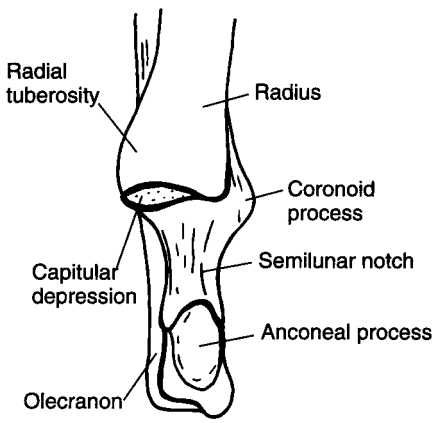


Fig. 33.3 Cranial aspect of the left radius and ulna.

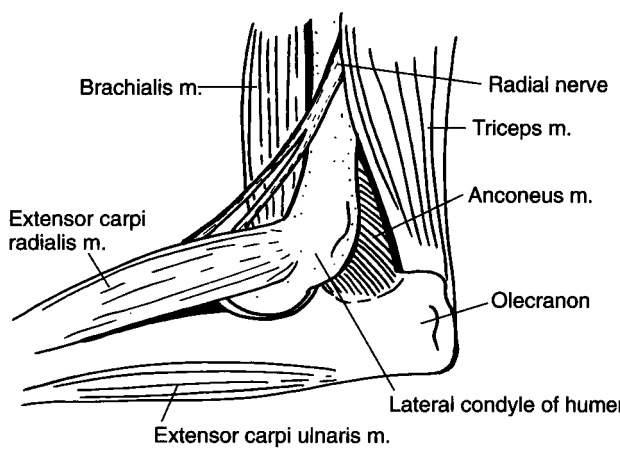


Fig. 33.4 Lateral aspect of the left elbow – soft tissue structures of surgical importance.

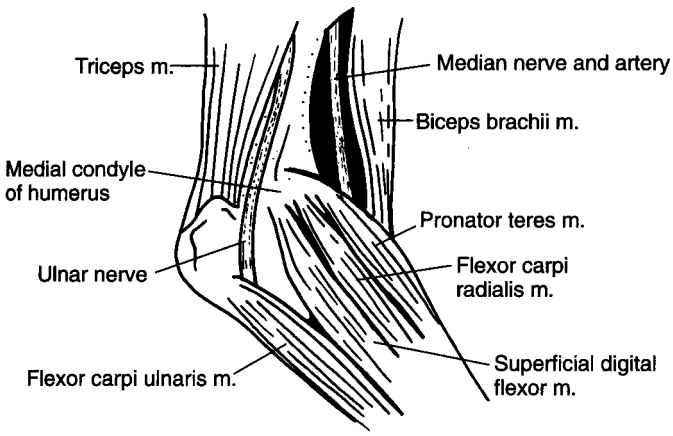
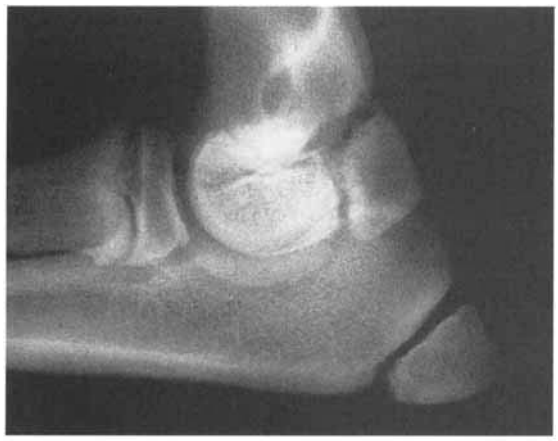
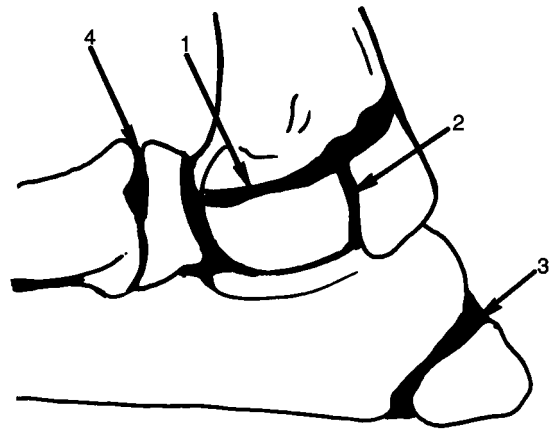


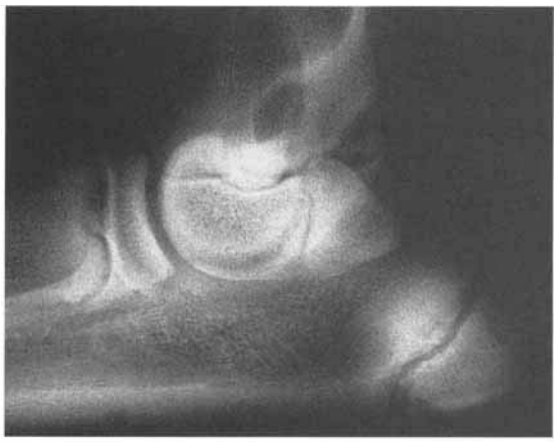
Fig. 33.5 Medial aspects of the left elbow – soft tissue structures of surgical importance.



(a) Lateral radiograph of elbow in a 2.5-month-old Rhodesian Ridgeback puppy.

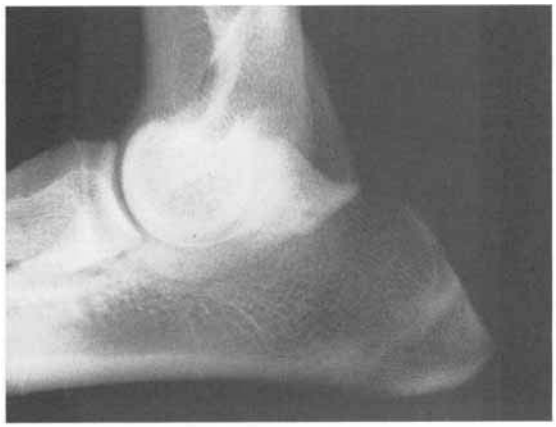


(b) Tracing of part (a). 1 – growth plate of medial humeral condyle; 2 – growth plate of medial humeral epicondyle; 3 – proximal ulnar growth plate; 4 – proximal radial growth plate.

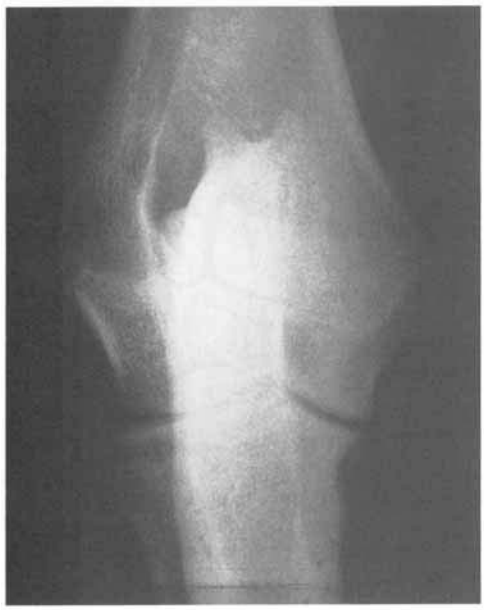


(c) Four-and-a-half-month-old Rhodesian Ridgeback puppy. Lateral radiograph of a normal elbow.

Fig. 33.6 (Photographs reproduced from *British Veterinary Journal* with permission from W.B. Saunders Company Ltd.)



(a)



(b)

Fig. 33.7 Nine-month-old Rhodesian Ridgeback puppy. Normal elbow, lateral view (a) and craniocaudal view (b). (Reproduced from *British Veterinary Journal* with permission from W.B. Saunders Company Ltd.)

Radiographic examination should include lateral and craniocaudal views of the elbow. The degree of deformity varies. Initially, the humeroradial joint will appear normal but the proximal ulna will be rotated, lying lateral to the humeral condyle (Fig. 33.8). Later, there is complete luxation of both the humeroradial and the humeroulnar joints (Fig. 33.9a).

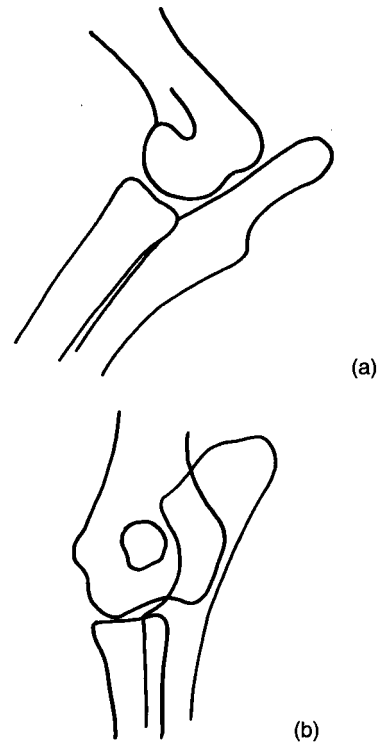
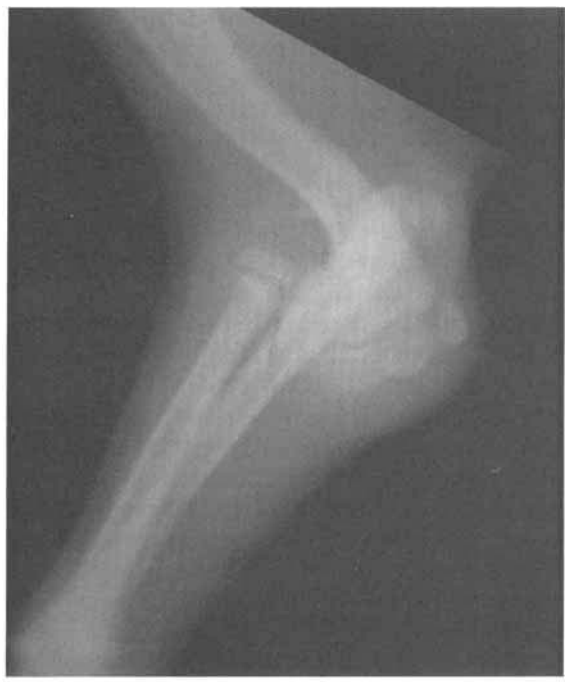


Fig. 33.8 Congenital luxation of the elbow – type 1. (a) Lateral rotation of the proximal ulna, lateral view. (b) Craniocaudal view of the elbow.

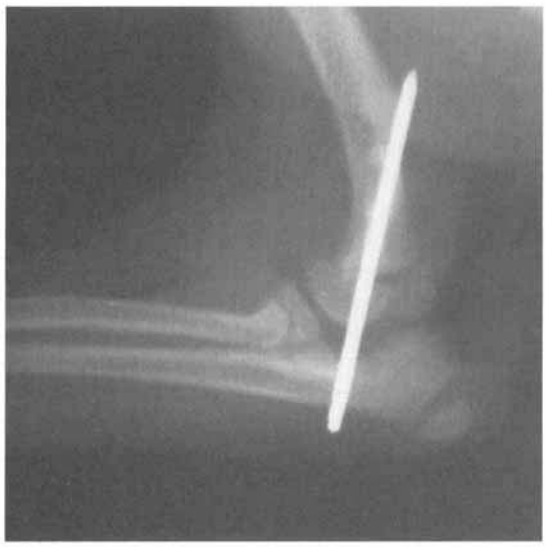
Closed reduction should be undertaken as soon as possible. Joint stability is maintained with a transarticular pin or Kirschner wire which is removed after 3–4 weeks. Good results have been reported with this approach (Campbell, 1979; Read, 1992). The technique is illustrated in Fig. 33.9b. If closed reduction is not possible then open reduction can be attempted, often involving osteotomy of the proximal ulnar diaphysis, but the results tend to be poor especially if treatment has been delayed too long resulting in deformity of the articular surfaces and contracture of the triceps muscle.

Type 2 – caudolateral luxation of the radial head

In type 2 elbow luxation the proximal radius is displaced in a caudolateral direction relative to the humeral condyle (Fig. 33.10) but the

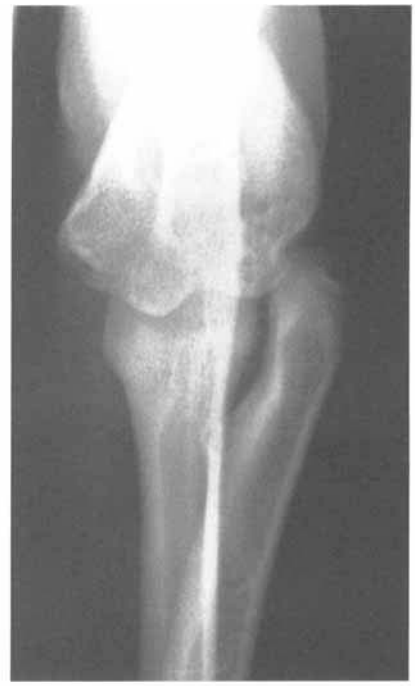


(a)

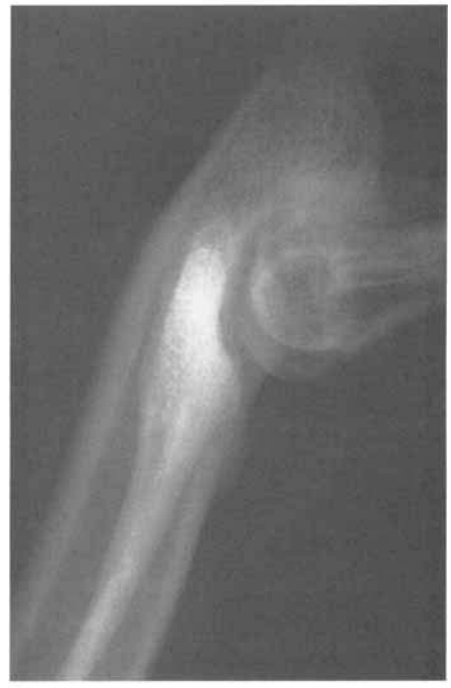


(b)

Fig. 33.9 (a) Congenital luxation of the elbow in a 3-month-old Sheltie showing type 1 complete luxation of both the humeroulnar and the humeroradial joints. (b) Postoperative radiograph of the case shown in (a). Reduction is maintained with a transarticular pin.



(a)



(b)

Fig. 33.10 (a) Congenital luxation of the elbow in an 8-month-old Labrador Retriever, showing type 2 caudolateral luxation of the radial head. (b) Lateral view of the elbow shown in (a). The humeroradial joint cannot be seen in this view. (Reproduced from *British Veterinary Journal* with permission from W.B. Saunders Company Ltd.)

humeroulnar joint is normal. The condition has been recorded in a wide variety of breeds, including: Pekingese, Yorkshire Terrier, Pomeranian, Staffordshire Bull Terrier, English Bulldog, Boxer, Bull Mastiff, Rough Collie, Old English Sheepdog and Labrador Retriever. Elbow deformity is usually first noticed at 4–5 months of age. Owners are often more concerned about the shape of the leg than the lameness. There is lateral bowing of the elbow and the radial head can be palpated lateral to the joint. The range of elbow movement is limited but there is usually little pain. The characteristic radiographic changes are shown in Fig. 33.10.

Although surgical correction by osteotomy and lag screw fixation of the radius to the ulna has been described (Campbell, 1979; Gurevitch & Hohn, 1980), the majority of affected animals manage surprisingly well despite the deformity, showing no evidence of lameness or elbow pain. Since most cases have good limb function despite the elbow deformity there is a good argument for treating cases conservatively with exercise restriction until skeletal maturity is reached. The main indications for surgical intervention are where normal joint congruency can be regained or limb function is deteriorating. If reduction is unsuccessful in resolving an intractable lameness then the options of radial head excision arthroplasty or arthrodesis may be considered.

Osteochondrosis ('elbow dysplasia')

Manifestations of osteochondrosis affecting the elbow are a common cause of lameness in young, rapidly growing medium to large breeds of dog. The conditions are listed below and can be grouped together under the general heading of 'elbow dysplasia'. Fragmentation of the medial coronoid process occurs most frequently (53% of cases). The incidence quoted is based on a review of 253 cases (Denny, 1995).

Elbow dysplasia includes:

- Osteochondritis dissecans (OCD) (25% of cases)
- Fragmented medial coronoid process ('coronoid disease') (FCP) (53% of cases)
- Osteochondritis dissecans + fragmented coronoid process (12% of cases)
- Ununited anconeal process (UAP) (7% of cases)
- Ununited medial humeral epicondyle (UME) (3% of cases)

Osteochondritis dissecans (OCD), ununited anconeal process (UAP) and ununited medial humeral epicondyle (UME) are all types of osteochondrosis which result from abnormal endochondral ossification of either the articular cartilage in OCD, or of growth plate cartilage in UAP and UME (Olsson, 1975). For further detail see Chapter 4. There is no evidence that the medial coronoid process develops as a separate centre of ossification (Guthrie & Vaughan, 1992) and the term 'ununited' coronoid process has been replaced by 'fragmented' coronoid process (FCP).

Elbow incongruency may have a role in the aetiology of these lesions, UME excepted, but even if this is the case then the cause of the incongruency must be related to abnormal activity within growth plates, and so the problem can still be correctly referred to as osteochondrosis. It is thought that FCP occurs secondary to elbow incongruency associated with a short radius or relative overgrowth of the ulna, (or too small a radius of curvature in the semilunar notch), which leaves the medial coronoid process more exposed to trauma and fragmentation because of increased weight-bearing (Wind, 1986). It is possible that OCD, which affects the medial part of the humeral condyle, could be an alternative manifestation of a similar joint incongruency. Conversely, it is thought that UAP occurs secondary to elbow incongruency associated with a short ulna or relative overgrowth of the radius which pushes the humeral condyle caudally onto the anconeal process so creating increased stresses within the process as the elbow joint extends (Sjostrom *et al.*, 1995).

Osteochondritis dissecans, fragmented coronoid process and ununited anconeal process cause chronic lameness in immature dogs and although the clinical features of each are similar, different breeds are affected in each case; UAP is seen most frequently in German Shepherd Dogs, Irish Wolfhounds and Basset Hounds; OCD is seen most often in Labrador Retrievers; and FCP is seen mainly in Labradors Retrievers, Golden

Retrievers, Rottweilers and Bernese Mountain Dogs. It is not unusual to find both OCD and FCP lesions in the same joint.

All three conditions cause a gradual onset of lameness at 4–7 months of age. Lameness tends to be intermittent in nature and is often most obvious after exercise or when the dog gets up from rest. There is pain on extension of the elbow and, in both OCD and FCP, the degree of pain is increased by supinating the forearm during elbow extension. There is increased synovial fluid production which may result in a fluctuating swelling on the lateral aspect of the joint, just caudal to the humeral condyle. The lesions cause secondary osteoarthritis which is detected clinically as periarticular thickening, possibly crepitation and a decreasing range of motion as the arthritis proceeds.

Ununited anconeal process (UAP)

Ununited anconeal process (UAP) is a well-recognised problem in the German Shepherd Dog. The condition occasionally occurs in other large breeds and sometimes in the Basset Hound. UAP accounts for some 7% of cases of elbow dysplasia. The anconeal process develops as a separate ossification centre and should unite with the ulna by the time a dog is 5 months old. Non-union is associated with elbow incongruency/instability and secondary osteoarthritis. The Basset Hound is an exception since although UAP is often diagnosed in this breed, it would be more accurate to describe this as delayed union because in the majority of affected Bassets the anconeus will eventually unite with the ulna by 10–11 months of age.

A flexed, mediolateral radiograph of the elbow will confirm the diagnosis of UAP (Fig. 33.11) but craniocaudal views should not be omitted as they may show additional joint pathology. The condition is bilateral in 40% of cases. Consequently, both elbows should be radiographed.

Treatment

In cases with mild lameness associated with UAP, *conservative management* may be advocated to see whether signs will settle over a 4–6 week period. This approach is particularly applicable in the Basset Hound (see above). In more severe cases, or those that fail to respond, *surgical treatment* should be considered. The ideal method of treatment should promote union of the anconeus and maintain elbow congruency. Dynamic ulnar osteotomy is the most promising method available at present. Other methods are briefly considered below.

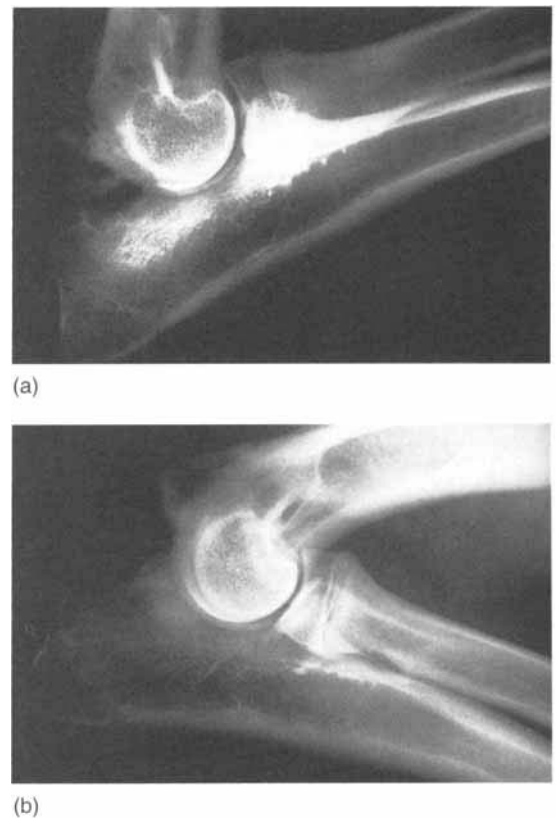


Fig. 33.11 A 6-month-old German Shepherd Dog showing ununited anconeal process (UAP). (a) Flexed lateral view of the right elbow showing UAP. (b) Flexed lateral view of the normal left elbow for comparison. (Reproduced from *British Veterinary Journal* with permission from W.B. Saunders Company Ltd.)

Surgical removal of the anconeal process through a caudolateral arthrotomy has been used most often in the past, particularly in dogs over 6 months of age. The prognosis is guarded and only 50–60% become sound after surgery (Table 33.1). The anconeal process normally contributes to the stability of the elbow joint. Instability after removal may predispose the dog to intermittent elbow sprains and osteoarthritis. If the cause is

Table 33.1 Results (%) of surgical treatment in a series of 253 cases of elbow dysplasia seen between 1987 and 1994 (Denny, 1995).

	Lesion				
	OCD[a]	FCP[a]	OCD + FCP	UAP[b]	UME[c]
No. of cases	63	134	30	18	8
Percentage (%)					
Sound	60	61	67	50	75
Occasionally lame	24	20	13	12.5	12.5
Slightly lame	3	6	13	25	–
Moderately lame	10	11	7	12.5	12.5
Severely lame	3	2	–	–	–
Success[d] (%)	84	81	80	62.5	87.5

[a] Osteochondritis dissecans (OCD) and fragmented coronoid process (FCP) lesions were treated by removal of loose fragments through a medial arthrotomy.

[b] Ununited anconeal process (UAP) lesions were treated by removal of the process through a caudolateral arthrotomy.

[c] Ununited medial epicondyle (UME) lesions were treated by reattachment of the fragment by means of a lagged bone screw (four cases) or excision of the fragment and suturing of its soft tissue attachments (four cases).

[d] Success was defined as those cases showing no or only occasional lameness.

now considered to be incongruency of the elbow joint then removal of the anconeal process is treating a result rather than the cause of the problem, which may account for the poor results. The main indications for this procedure are where dynamic ulnar osteotomy has failed to improve the lameness or is considered to be unlikely to do so because of significant osteoarthritic change already being present radiographically.

A skin incision is made along the caudolateral border of the lateral humeral epicondyle (Fig. 33.12a). The triceps fascia is incised and the cranial edge of the lateral head of the triceps muscle is retracted caudally to expose the anconeus muscle. The anconeus muscle is incised, together with the underlying joint capsule to which it is closely attached (Fig. 33.12b) just caudal to the lateral epicondyle (leaving sufficient muscle on the condylar side to be sutured). Haemorrhage from the muscle may be a slight problem but this can be controlled by using self-retaining retractors to retract the muscle edges and joint capsule and expose the caudal aspect of the elbow joint. The elbow is flexed to reveal the anconeal process (Fig. 33.12c) which is prised away from its caudal soft tissue attachments to the ulna using a periosteal elevator. After removal of the process, any remaining fragments within the joint are flushed out with saline solution. The anconeus muscle and joint capsule are closed together with a continuous suture of absorbable material (e.g. Vicryl or Monocryl, Ethicon). Subsequent wound closure is routine. A support bandage may be applied for a week postoperatively and exercise is restricted for 6 weeks. If the lesion is bilateral then a 4- to 6-week interval is left between operations.

Screw fixation using a lagged bone screw to stabilise the anconeal process has been described (Bohmer *et al.*, 1987). Implant failure and persistent non-union occur quite frequently and consequently the technique is not used by the authors.

Oblique osteotomy of the ulnar shaft just distal to the annular ligament (Figs 33.13 and 33.14) is the most promising treatment for UAP (Sjostrom *et al.*, 1995; Turner *et al.*, 1998). This is best done early, i.e. 5–6 months of age, as later there may be secondary osteoarthritis, which might compromise the technique's usefulness, and the speed of healing of the osteotomy is related to the dog's age. The osteotomy relieves tension on the

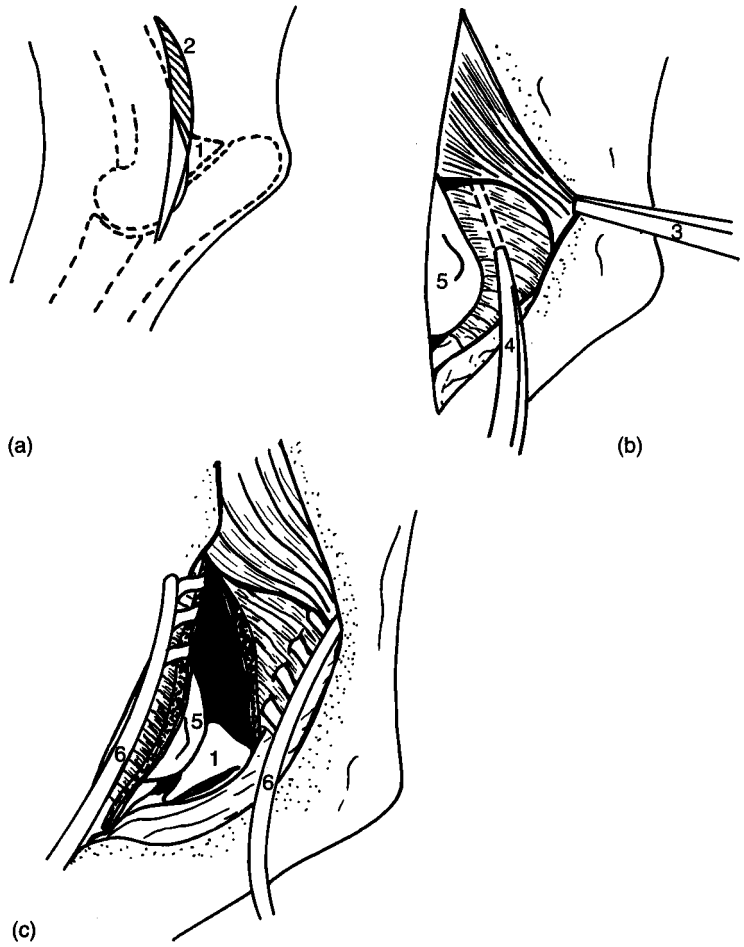


Fig. 33.12 Caudolateral view of elbow arthrotomy. Surgical approach for removal of an ununited anconeal process (UAP). (a) This shows the position of the underlying anconeal process (1) and the site of skin incision (2). (b) The lateral head of the triceps muscle is retracted (3) and the anconeus muscle and the joint capsule incised (4). The lateral humeral condyle is revealed (5). (c) The anconeus muscle and joint capsule are retracted (6) to expose the lateral humeral condyle (5) and the anconeal process (1).

anconeal process, by allowing the pull of the triceps to 'draw' the olecranon proximally and/or rotate it slightly, thus improving joint congruency and encouraging fusion of the anconeal process with the ulna. Dogs can be lame for up to 4 months postoperatively while the osteotomy heals and remodels. In some cases the anconeal process remains ununited despite improvement in lameness, possibly indicating that it is joint incongruency rather than UAP that caused pain in the first place.

After routine surgical preparation, the dog is placed in dorsal recumbency with the limb drawn forwards. A skin incision is made over the proximal ulnar diaphysis and exposure is completed by elevation of the flexor and extensor carpi ulnaris from each side of the ulnar shaft (similar to Fig. 33.29 but without exposing the olecranon as well). An oblique or transverse osteotomy is carried out to exit the ulna just distal to the annular ligament. If the cut is made too close to the elbow it will destabilise the joint, but if it is too far distal then it is less likely the movement required of the proximal ulna postoperatively will occur. There is no requirement for a piece of ulna to be removed although doing so (as shown in Fig. 33.13) may make it easier to ensure that the osteotomy is complete. The osteotomy is usually left 'unstable'. Although an intramedullary pin can be used to reduce the instability without interfering with the movement required for the technique to work, the authors' experience is that many of these pins will work loose, requiring removal at a later date, and so such osteotomies are now left without any

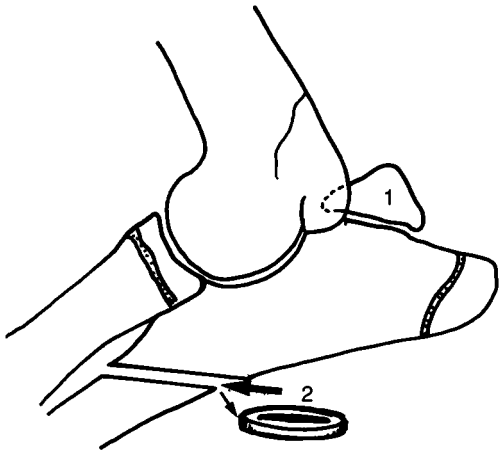
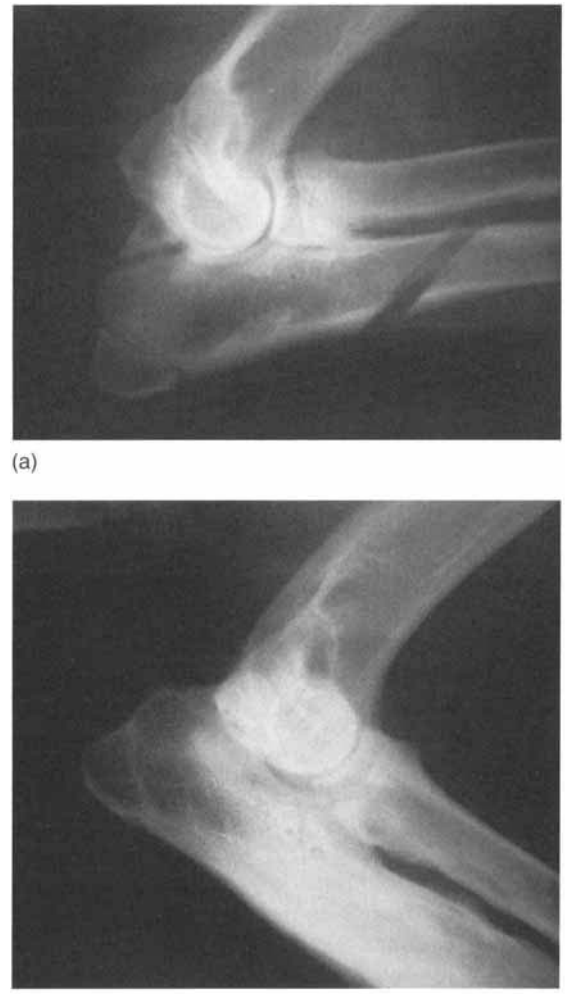


Fig. 33.13 Oblique dynamic ulnar osteotomy for ununited anconeal process (UAP) (1). A section of ulna, 3 mm in length, has been removed (2).

internal support. The muscle bellies/fascia are coapted with an absorbable suture material. The rest of the wound closure is carried out in routine fashion. A support bandage is used for 5 days and exercise is restricted to lead exercise for 2–4 months following surgery.

The mature dog

Dogs with an ununited anconeal process may reach maturity without serious lameness problems. They may then present in later life with a sudden onset of lameness. The elbow is painful and radiographs demonstrate advanced osteoarthritis and a UAP. What should be done in this situation? The osteoarthritis has usually been aggravated by an elbow sprain and lameness should resolve with rest and the administration of non-steroidal anti-inflammatory drugs (NSAIDs). Splinting the joint in extension with a Robert Jones bandage for 2 weeks may be of use, if the sprain is severe, as part of the conservative management. Surgical removal of the anconeus should only be considered at this late stage if lameness shows no sign of resolving with conservative measures, or the process has become displaced.



(a)

(b)

Fig. 33.14 Five-month-old German Shepherd Dog with an ununited anconeal process (UAP). (a) Postoperative radiograph showing oblique ulnar osteotomy. (b) Two-month follow-up radiograph showing that the anconeus has fused with the ulna and the osteotomy has healed.

Osteochondritis dissecans (OCD) and fragmented medial coronoid process (FCP)

Osteochondritis dissecans (OCD) of the medial part of the humeral condyle accounts for some 25% of cases of elbow lameness resulting from dysplasia and is most often seen in the Labrador Retriever. Fragmented medial coronoid process

(FCP) is the most common form of elbow dysplasia and accounts for 53% of cases. This condition affects the Labrador Retriever, Golden Retriever, Rottweiler and Bernese Mountain Dog most frequently. Clinical features have been described already.

Diagnosis

The radiographic diagnosis is not easy in the early stages. The following projections should be taken of *both* elbows:

- Extended mediolateral view
- Flexed mediolateral view
- Craniocaudal view (+/– craniolateral–caudomedial oblique views to skyline the medial coronoid process)

Osteochondritis dissecans and fragmented coronoid process both cause the development of osteoarthritis in the elbow. The first radiographic indication of this is osteophyte formation on the caudal aspect of the anconeal process (Fig. 33.15). Later, similar changes develop on the medial aspect of the elbow and on the head of the radius (Figs 33.15 and 33.16). The craniocaudal view of the elbow may demonstrate the OCD lesion as an erosion or area of flattening in the subchondral bone of the medial part of the humeral condyle (Figs 33.16 and 33.17). A mineralised cartilage flap may be seen (Figs 33.16 and 33.18).

Problems arise with the definitive diagnosis of FCP using conventional radiographic techniques because there is invariably superimposition of other bony structures. However, some features may be present which indicate FCP as the likely cause of lameness. In the mediolateral views of the elbows look for evidence of joint incongruency (Fig. 33.19), periarticular osteophytes or apparent sclerosis of the coronoid process. In an oblique craniocaudal view, with slight inward rotation of the antebrachium, the coronoid area is highlighted and a fragment may be visible, particularly if it is large or displaced (Fig. 33.20).

If the initial radiographic examination of the elbow is normal then exercise is restricted for 6 weeks and follow-up radiographs are taken. If there is an OCD or FCP lesion in the elbow then some evidence of secondary osteoarthritic change should be visible at that stage. Alternatively, arthroscopic diagnosis (van Bree & Van Ryssen, 1992; Bardet, 1997) is now routinely used in some centres and is particularly useful in suspected cases of OCD/FCP in which radiographic findings are inconclusive.

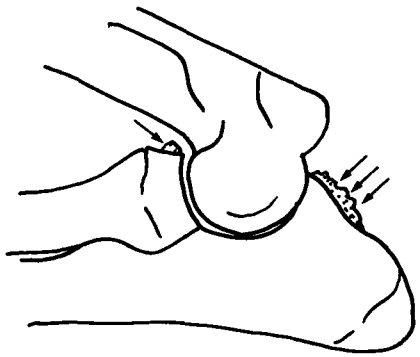


Fig. 33.15 Elbow, flexed lateral view. Osteophytes are seen on the caudal border of the anconeus and on the head of the radius (arrows).

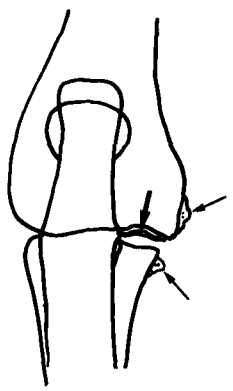


Fig. 33.16 Elbow, craniocaudal view. Osteophytes are shown on the medial aspect of the joint (small arrows) and an osteochondritis dissecans (OCD) lesion in the medial humeral condyle (large arrow).

Management

The cause of lameness in these cases is most likely to be a combination of one or more of the following:

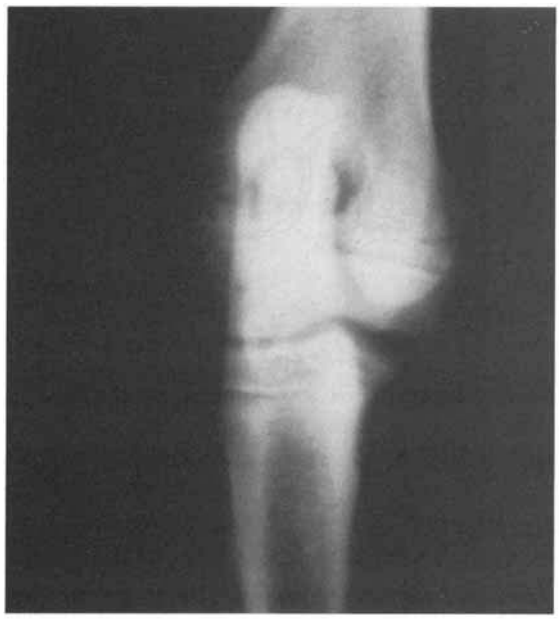


Fig. 33.17 Craniocaudal view of the elbow of a 6-month-old Labrador showing erosion of the medial humeral condyle and an increase in the joint space as a result of osteochondritis dissecans (OCD).

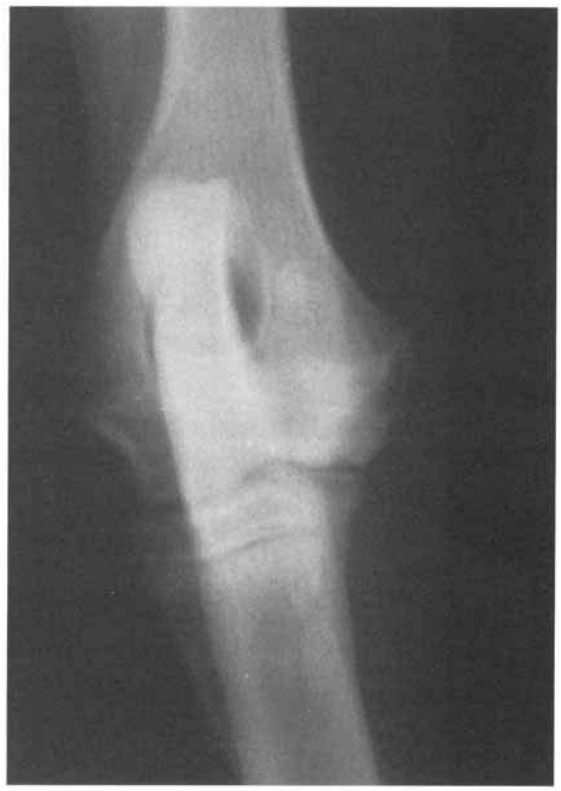
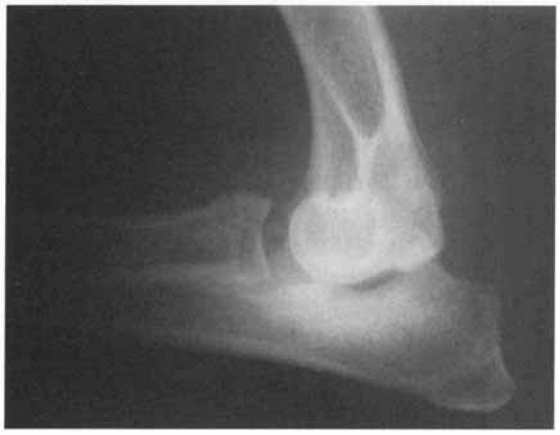
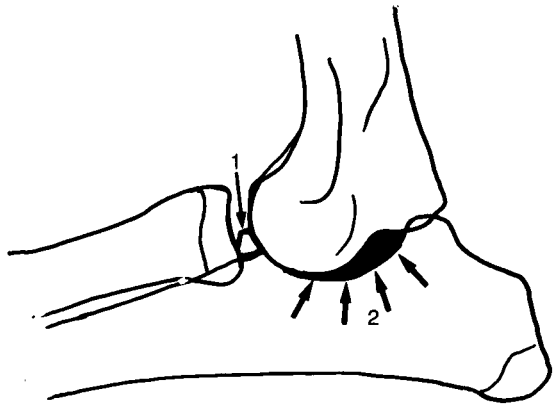


Fig. 33.18 Craniocaudal view of the elbow of a 7-month-old Labrador showing erosion of the medial humeral condyle and a mineralised cartilaginous flap as a result of osteochondritis dissecans (OCD).



(a)



(b)

Fig. 33.19 (a) Lateral radiograph of the elbow of a 6-month-old German Shepherd Dog demonstrating incongruity and fragmented medial coronoid process (FCP). (b) Tracing of the radiograph shown in (a). 1 – Increase in humeroradial joint space and coronoid fragment; 2 – incongruity in the humeroulnar joint space.

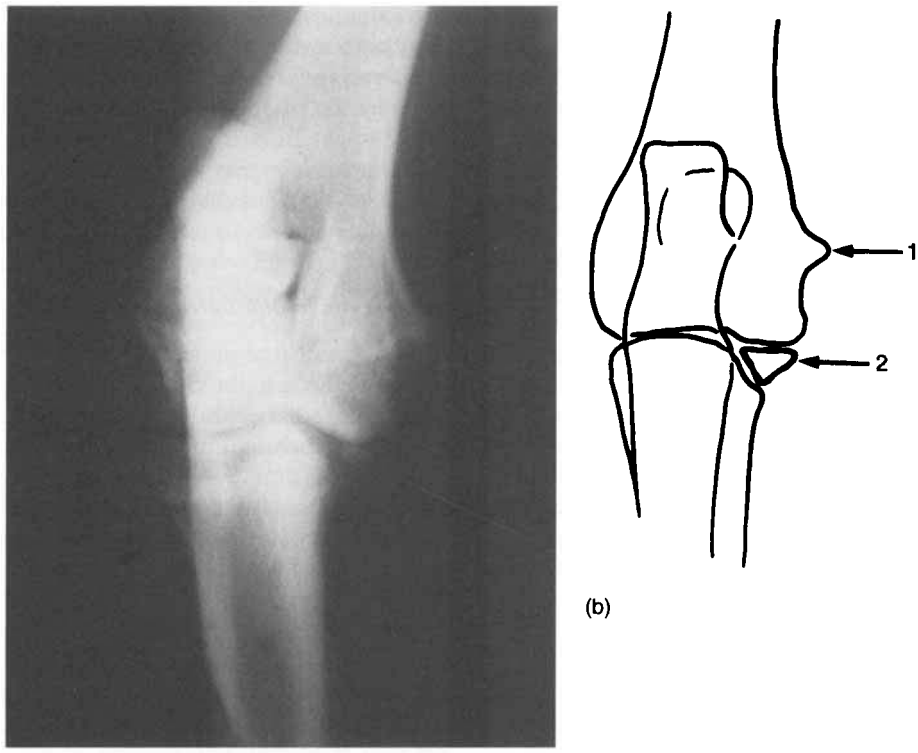


Fig. 33.20 (a) Radiograph of a 7-month-old Bernese Mountain Dog showing craniocaudal view of the elbow, indicating fragmented medial coronoid process (FCP). (b) Tracing of the radiograph shown in (a). 1 – Osteophytes on medial humeral condyle; 2 – fragmented medial coronoid process.

- Joint incongruency
- The presence of loose fragments within the joint (flap of cartilage in OCD or fragment of coronoid in FCP)
- Secondary osteoarthritic change

Joint incongruency may improve with time. The relative shortening of the radius, believed to underly the articular lesions, may disappear as the animal continues to grow, but any such 'self-correction' can only occur before the relevant growth plates close (i.e. before 9–10 months of age). Alternatively, joint incongruency may be improved by creating a 'dynamic' ulnar osteotomy which allows either the ulna to shorten slightly (bringing it back in line with the radius) and/or rotate slightly (so that the coronoid process 'sinks' to the level of the radial head) as depicted in Fig. 33.21.

Loose fragments may become detached and resorb (especially OCD flaps) or fibrose and remodel (in the case of coronoid process fragments). If they fail to do so and are thought to be irritating the joint then they can be removed surgically, preferably before significant radiographic evidence of osteoarthritis has developed.

Osteoarthritic change can really only be influenced rather than corrected. By attending to the first two contributors to lameness it is hoped that the degree of secondary osteoarthritic change will be minimised, though it is recognised that many dogs will go on to show clinical evidence of osteoarthritis in later life, even when the underlying elbow dysplasia did not cause clinical lameness during early life.

General guidelines

In cases with OCD, conservative management with restricted exercise for 4–6 weeks may allow

the cartilage flap to detach and the defect to fill in with fibrocartilage (see Chapter 4). If progress is unsatisfactory after that time then a medial arthrotomy or arthroscopy is advisable to remove the flap of cartilage and any loose edges around the defect.

In cases with FCP the decision-making is far less clear cut. The difference between groups of dogs managed surgically or conservatively is essentially in the degree of lameness rather than the presence of lameness. In cases with mild or intermittent signs, conservative management will lead to as good a result as surgery. If a more severe ($\frac{3}{10}$+), persistent lameness is present, and 4–6 weeks of conservative management have failed to improve matters, then surgical removal of the fragmented coronoid should be undertaken. If a mild case persists beyond 12 months of age then again surgery should be considered. During surgery the articular surfaces of the radius and ulna should be examined for evidence of incongruity, though the value and reliability of this observation has yet to be established since the appearance of the joint surfaces during arthrotomy might bear little resemblance to the situation/arthroscopy in the weight-bearing joint. If progress following surgery is unsatisfactory then dynamic ulnar osteotomy may be considered.

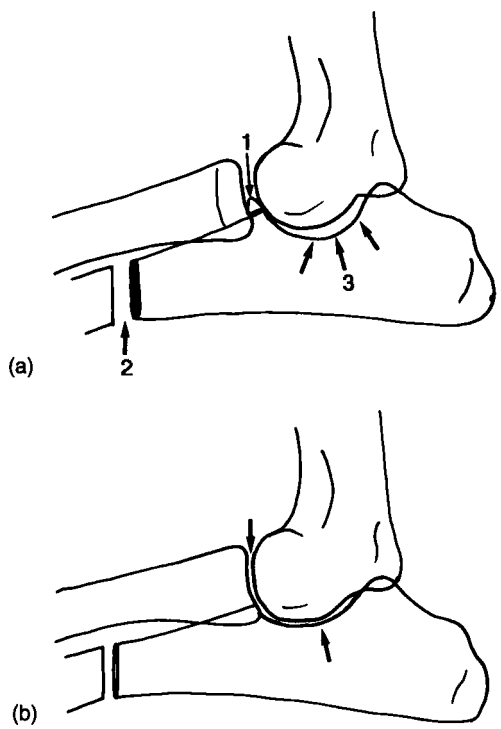


Fig. 33.21 Treatment of fragmented medial coronoid process (FCP) (according to Ness (1998)). 1 – Remove coronoid fragments; 2 – ulnar osteotomy to correct elbow incongruity (3). (b) Incongruity of humeroradial and humeroulnar joints corrected by ulnar osteotomy.

Conservative management

This is indicated in cases with suspect elbow OCD/FCP where the clinical signs are mild ($<\frac{3}{10}$ or intermittent lameness) or radiographic examination is inconclusive in the early stages. The aim is to allow time for resorption/remodelling of loose fragments and/or 'self-correction' of joint incongruency (see above). Exercise is restricted to lead walks, of a duration that does not aggravate the lameness or cause excessive stiffness after rest, and can be repeated regularly (three or more times daily), for 6 weeks. A 4-week course of pentosan polysulphate sodium (Cartrophen-VET, Arthropharm, Australia) may also be given (Bouck *et al.*, 1995).

Medial elbow arthrotomy

Arthrotomy (or arthroscopy) is indicated in cases with radiographically confirmed OCD that has not responded to conservative management, in suspect cases of FCP showing a persistent lameness that is moderate to severe ($>\frac{3}{10}$) and has not responded to conservative management, or in cases with FCP showing mild signs that are managed conservatively but the problem persists beyond 10–12 months of age.

The elbow is a 'tight' joint with little room for loose fragments within the joint space. Their pressence may cause lameness and the development of secondary osteoarthritic change. Removal of such fragments may reduce the level of lameness directly and may also reduce the rate of development of osteoarthritic change. If there are bilateral lesions both elbows are operated on, either together or with a 4- to 6-week interval between surgeries. Arthroscopic treatment is done at some centres but conventional arthrotomy is still widely practised. The medial approach gives access to both OCD and FCP lesions.

After routine surgical preparation, the dog is placed on its side and a bolster is placed beneath the elbow. A skin incision is made over the medial humeral epicondyle (Fig. 33.22a). The pronator teres and flexor carpi radialis muscles (Fig. 33.22b) are separated close to their origin on the medial epicondyle and retracted with a West's retractor. A vertical incision is made in the joint capsule over the humeral condyle only (Fig. 33.22c,d). The blades of a pair of straight scissors are then introduced between the articular surfaces of the joint and spread laterally to complete the exposure (Fig. 33.22e). The West's retractor is repositioned to include the cut edges of the joint capsule. This method of arthrotomy minimises the risk of damage to the median nerve and artery which cross the cranial and distal margins of the joint. The articular surfaces are separated with the aid of a Hohmann retractor (Fig. 33.22f) and exposure is improved by hinging the elbow open over the bolster and/or flexing the carpus and rotating the paw laterally.

The medial articular surface of the humeral condyle is inspected for an OCD lesion. If a cartilaginous flap is present, it is removed and the underlying erosion in the subchondral bone is curetted to remove any loose edges. In some cases the condylar surface may appear chondromalacic rather than showing a true OCD lesion. This so-called 'kissing lesion' is thought to be a result of an FCP rubbing on the condylar surface (further evidence of an underlying joint incongruity?). In some cases the lesion takes the form of fissures in the cartilage, in which case the area is curetted. The coronoid process is then inspected. Any fragments are freed from any remaining cartilaginous or fibrous attachments and removed. An affected coronoid process will be found either as a discrete triangular fragment of bone and cartilage or it may be in the form of two or three fragments. Exposure of the coronoid can be improved by cutting the caudal margin of the medial collateral ligament (Fig. 33.22g), however complete sectioning of the ligament should not be necessary. It is not unusual to find both OCD and FCP lesions in the same elbow.

After the lesion has been dealt with, the joint is flushed out with saline. The joint capsule may be closed with an absorbable material (e.g. Vicryl or Monocryl, Ethicon). The muscle bellies are coapted with a continuous suture of the same material, which effectively seals the joint capsule if this has not been closed. The rest of the wound closure is carried out in routine fashion. A support bandage may be used for 5 days and exercise is restricted to lead exercise for 6 weeks following surgery.

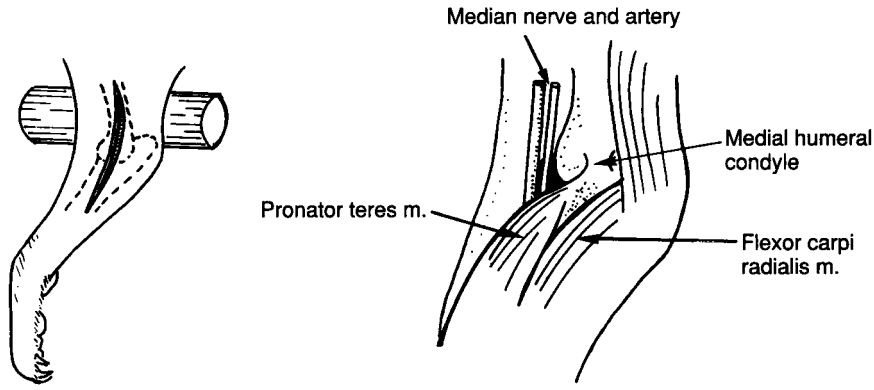
The results of treating OCD and/or FCP by arthrotomy are given in Table 33.1. Most cases will continue to develop osteoarthritic change despite removal of the cartilage flap or fragment, presumably because of elbow incongruity. However, this is not necessarily associated with clinical lameness.

The arthroscopic removal of such fragments has been described (Bardet, 1997). Although postoperative morbidity appears to be reduced, when compared to arthrotomy, the overall results are similar.

'Dynamic' ulnar osteotomy

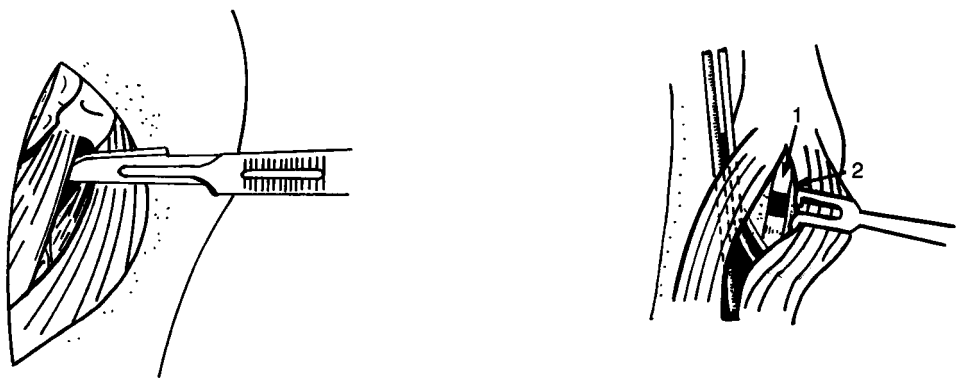
This is indicated in cases with convincing evidence of joint incongruency (either radiographically or at arthrotomy), in which case the surgery is usually combined with arthrotomy to remove any loose fragments, or in cases that have failed to improve significantly after such an arthrotomy. The aim is either to allow the ulna to shorten slightly (bringing it back in line with the radius) and/or to rotate slightly (so that the coronoid process 'sinks' to the level of the radial head) as depicted in Fig. 33.21.

After routine surgical preparation, the dog is placed in dorsal recumbency with the limb drawn forwards. A skin incision is made over the proximal ulnar diaphysis and exposure is completed by elevation of the flexor and extensor carpi ulnaris from each side of the ulnar shaft (similar to Fig. 33.29 but without exposing the olecranon as well). An oblique or transverse osteotomy is carried out to exit the ulna just distal to the annular ligament. If the cut is made too close to the elbow it will destabilise the joint, but if it is too far distal then it is less likely the movement required of the proximal ulna postoperatively will occur. Since one aim of this technique is to create ulnar shortening, it is better to remove about 3 mm of diaphysis by creating two parallel osteotomies (technically a partial ulnar ostectomy). A larger gap than this is



(a) A bolster is placed beneath the elbow and a skin incision made over the medial humeral condyle.

(b) The pronator teres and flexor carpi radialis muscles are separated.



(c) Vertical arthrotomy over the medial humeral condyle.

(d) The medial humeral condyle (1) and joint capsule (2) are exposed. Note position of the median nerve and artery.

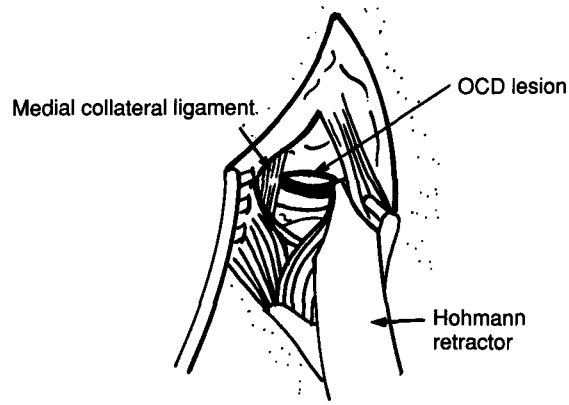
Fig. 33.22 Medial elbow arthrotomy for osteochondritis dissecans (OCD) and fragmented medial coronoid process (FCP).

unnecessary and might slow down the healing process. The osteotomy is usually left 'unstable'. Although an intramedullary pin can be used to reduce the instability without interfering with the movement required for the technique to work, the authors' experience is that many of these pins will work loose, requiring removal at a later date, and so such osteotomies are now left without any internal support. The muscle bellies/fascia are coapted with an absorbable suture material. The rest of the wound closure is carried out in routine fashion. A support bandage is used for 5 days and exercise is restricted to lead exercise for 2–4 months following surgery.

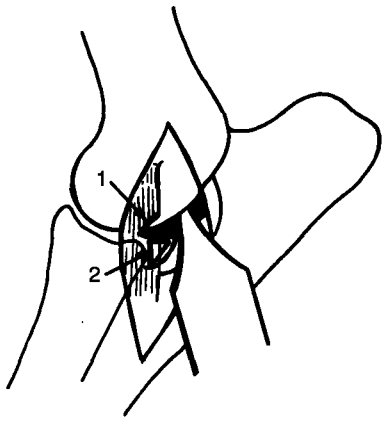
Prognosis

In the past the prognosis for these articular forms of elbow osteochondrosis have generally been given as fair to guarded. It appears that the prognosis for OCD is better than for FCP but it has to be remembered that the latter by far outweighs the former in terms of incidence. In cases with OCD, probably about 75% will become sound with

(e) The arthrotomy incision is enlarged using scissors.



(f) Exposure is maintained with West's retractors. Joint surfaces are separated with a small Hohmann retractor. The humeral condyle is inspected for OCD.



(g) Exposure of the fragmented coronoid process (2) can be improved by section of the caudal border of the medial collateral ligament (1).

Fig. 33.22 *Contd.*

conservative management or following surgical removal of the cartilage flap if conservative measures fail. The outlook is probably better if surgery is performed before significant radiographic osteoarthritic change has become evident.

In cases with FCP, the surgical removal of the fragments does not decrease the incidence of lameness although it may reduce the degree of clinical signs and allow the dog a greater level of activity. In one prospective study it was found that 75% of owners with surgically treated dogs were satisfied with the outcome compared to 60% with conservatively managed dogs (Read *et al.*, 1990). It may be that the reason for poorer results in surgical management of FCP has been that an underlying problem, such as joint incongruency, has been left unattended. There is already encouraging work to suggest that dynamic ulnar osteotomy may significantly improve the lameness seen in many dogs which fail to respond to coronoid process removal via arthrotomy (Bardet, 1992). Good results were also obtained in another series

of 10 dogs treated by medial arthrotomy combined with ulnar osteotomy (Ness, 1998), and of particular interest in that study was the observation that follow-up radiographs showed no further development of osteoarthritic change.

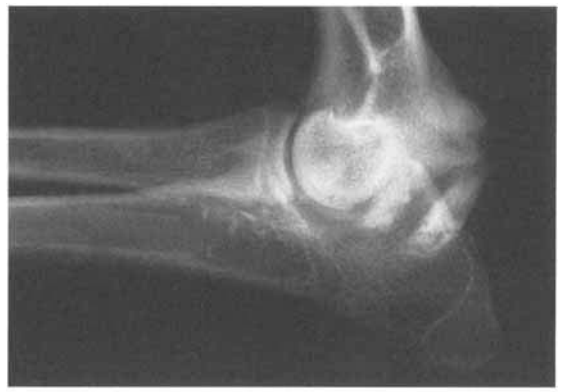
Treatment of older dogs with 'OCD/FCP'

Older dogs which have coped for years with elbow osteoarthritis secondary to OCD/FCP may suddenly develop an acute 'flare-up' of lameness in an elbow. This is usually the result of a sprain and should settle with rest and NSAIDs (see Chapter 7, p. 59). If lameness persists and radiographs indicate free fragments within the joint, then surgical removal of the fragments may make the dog more comfortable and reduce the severity of the lameness. When there is severe pain the possibility of secondary infection and septic arthritis should be considered.

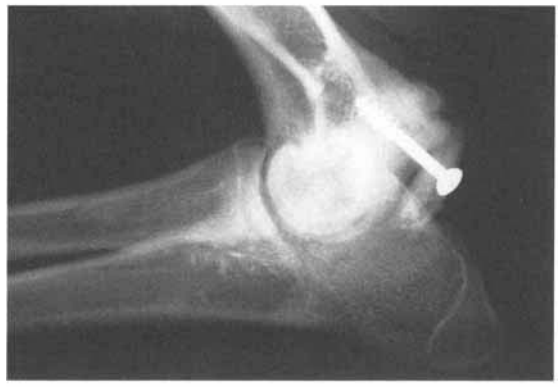
Ununited medial epicondyle (UME)

The distal humerus develops from three centres of ossification: one for the lateral part of the condyle, one for the medial part of the condyle, and one for the medial epicondyle (Hare, 1961). The medial epicondyle is the point of origin of several carpal and digital flexor muscles. This epiphysis normally fuses with the distal humeral epiphysis and metaphysis by 6 months of age.

Failure of the medial epicondyle to unite with the humerus is occasionally encountered as a fusion defect (Vaughan, 1979; Denny, 1983) and accounts for some 3% of elbow dysplasia cases. The Labrador Retriever is affected most frequently. History and clinical signs are similar to other forms of elbow dysplasia. Radiographic examination shows the ununited epicondyle as a discrete fragment on the caudomedial side of the elbow (Fig. 33.23a). There are numerous muscle attachments to the fragment. If it is large, then it should be reattached to the humerus with a lagged bone screw (Fig. 33.23b). If the fragment is small, lameness will generally resolve by the time the dog reaches 1 year of age provided exercise is restricted. If lameness persists, however, then the aim is to reattach the flexor tendon origins to the epicondyle using a screw and spiked washer, with or without first removing the small fragment.



(a)



(b)

Fig. 33.23 (a) A 1-year-old Golden Retriever with an ununited medial epicondyle (UME). (b) Postoperative radiograph. Fixation of the medial epicondylar fragment is achieved with a 4 mm cancellous screw. (Reproduced from *British Veterinary Journal* with permission from W.B. Saunders Company Ltd.)

Prognosis is good, particularly as UME is seldom associated with the severe secondary osteoarthritic change which occurs with other forms of elbow dysplasia. Indeed, if such secondary changes are noted then it is likely the UME is not the only manifestation of dysplasia present.

Prevention of elbow dysplasia

It is now clear that OCD and FCP carry a high degree of hereditability (Guthrie & Pidduck, 1990; Grondalen & Lingaas, 1991). Owners and breeders should be advised not to breed from affected animals. Routine elbow radiographic screening programmes have been in use in Scandinavia for many years to reduce the incidence of

elbow dysplasia. The British Veterinary Association (BVA) elbow screening scheme was introduced in the UK in 1998. It is aimed at screening potential breeding animals for radiographic evidence of elbow dysplasia which has not necessarily caused clinical signs. Both primary lesions and secondary osteoarthritic changes are looked for and scored. The value of this scheme has been much debated and its ability to reduce the incidence of elbow dysplasia in the UK remains to be seen, though such schemes appear to have improved the situation in Scandinavia.

Traumatic elbow luxation

Traumatic luxation (or dislocation) of the elbow is usually seen in dogs and cats over 1 year of age. The injury results from involvement in road traffic accidents or when the animal catches its leg in a fence and is suspended by the limb.

Mechanics of luxation/dislocation

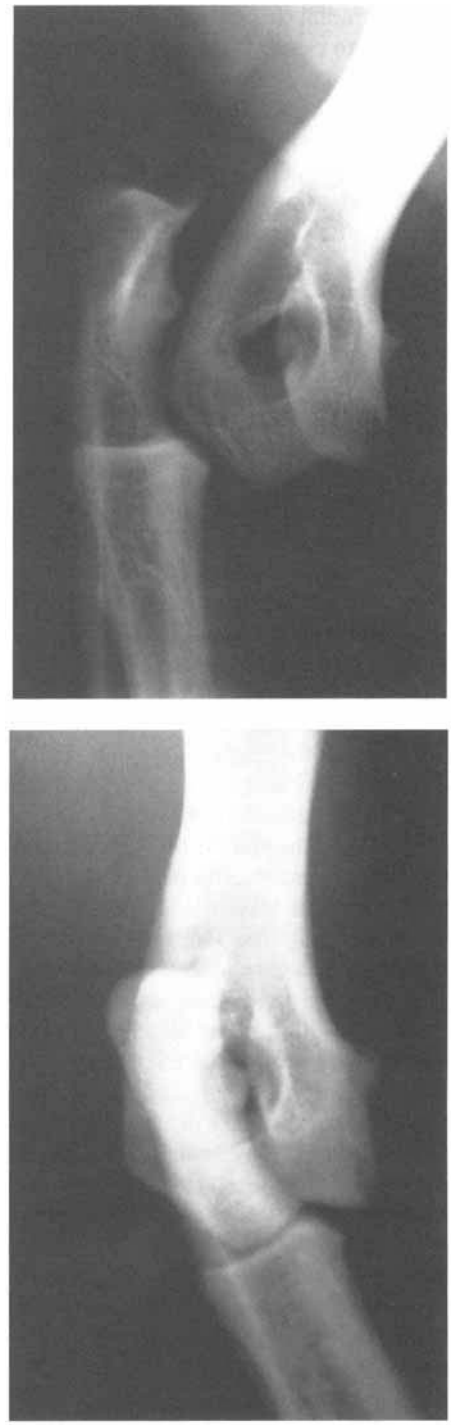
The radial head dislocates laterally and this can only occur if the anconeal process has become disengaged from its fossa between the humeral epicondyles. Therefore, the elbow must be flexed through more than 45° in the accident and twisted laterally for dislocation to occur. Provided this concept is understood then a rational method of reduction can be employed (see below).

Clinical features

After dislocation of the elbow the leg is held forward in semiflexion, with the lower limb abducted and supinated. The elbow joint will be obviously deformed and painful. Flexion and extension are limited. The diagnosis is confirmed radiographically. Both mediolateral and craniocaudal views should be taken (Fig. 33.24a,b) noting any bone fragments which may be associated with collateral ligament avulsion.

Treatment

Reduction is achieved under general anaesthesia. The elbow is fully flexed and then the radius and ulna are rotated medially. The elbow joint is slowly extended until the anconeal process is re-engaged in its normal position between the humeral epicondyles. If the manipulation is suc-



(a)

(b)

Fig. 33.24 (a) A 4-year-old Cocker Spaniel with traumatic elbow luxation. (b) Radiograph after closed reduction. (Reproduced from *British Veterinary Journal* with permission from W.B. Saunders Company Ltd.)

cessful the radial head and anconeal process 'snap' back into place, a full range of elbow movement is restored and the elbow should feel stable. A Robert Jones bandage may be applied for 7–10 days and exercise should be restricted for 4 weeks. Many cases make a satisfactory recovery following closed reduction but, in the long term, secondary osteoarthritic change is common (Billings *et al.*, 1992).

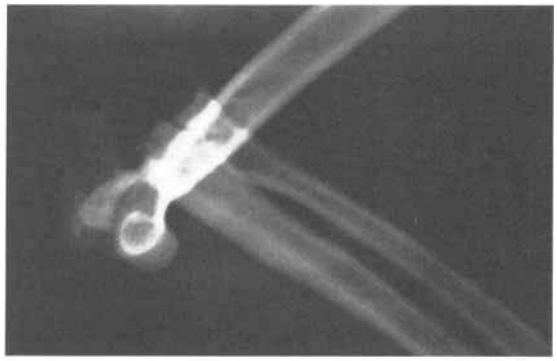
Following closed reduction, instability of the elbow may be present due to stretching or rupture of the collateral ligaments. Each collateral ligament can be tested grossly using the following manipulation. The carpus and elbow are held at 90° of flexion and the paw is then rotated medially and laterally. Normally, in a lateral direction, the paw will rotate through about 45° but when the medial collateral ligament of the elbow is ruptured this increases to about 90°. In a medial direction, the paw will normally rotate through about 70° but when the lateral collateral ligament of the elbow is ruptured this increases to about 140°. In these cases surgical stabilisation of the elbow is important. If the collateral ligament has avulsed from the humerus it can be reattached using a bone screw and spiked washer. If the ligament has ruptured then primary repair should be attempted (see Chapter 3, p. 24). Alternatively, stability can be restored by replacing the medial collateral ligament with a figure-of-eight heavy gauge braided polyester suture (7 metric Ethibond, Ethicon) anchored by two bone screws, one placed in the medial aspect of the humeral condyle and the other in the ulna. However, placement of the bone screws in exactly the right position (at the point of origin and insertion of the collateral ligament) is very difficult and not doing so will compromise joint function and predispose to osteoarthritis through abnormal articulation. Wherever possible, therefore, it is better to reattach or repair the natural ligament.

In three cases treated by one of the authors it has been noted in radiographs taken after closed reduction that the reduction is incomplete such that the radial head and ulna are 'subluxated' laterally, though the joint shows a good range of motion and appears stable using the test described above. A medial approach to these joints revealed avulsion of the medial collateral ligament from the humerus and 'entrapment' of the ligament within the joint space during reduction. The ligament was teased out of the joint and reattached to the humerus with a bone screw and spiked washer. In one case the lateral collateral ligament was also causing a similar problem and was removed from the joint space and reattached to the humerus in a similar fashion. All three cases returned to completely normal function, including a return to sheep dog trialling in one case. Long-term follow-up is not yet available on such cases, with respect to the development of osteoarthritic change. However, it is perhaps appropriate to consider that elbow luxation cannot occur without failure of at least the medial collateral ligament and that a more rapid return to normal activity and better long-term function is likely to be seen if the collateral ligament injury is treated in the acute phase of the injury, i.e. closed reduction alone may not be the optimal treatment in the majority of cases. After such surgery the joint may be bandaged for a few days and exercise is restricted to room rest and lead walks for 6 weeks.

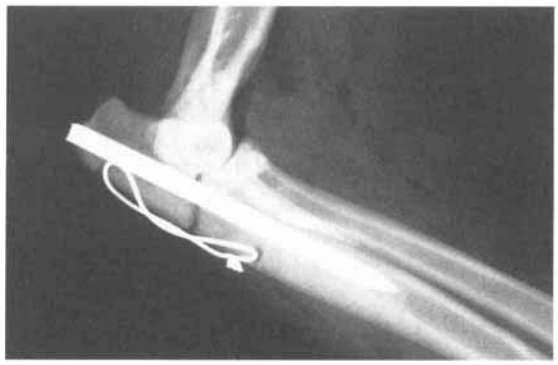
In long-standing, neglected dislocations open reduction must be carried out but this can be difficult. The operation is carried out in two stages. First, reduction is achieved through a lateral approach to the elbow. Fibrous adhesions are broken down and the radial head and anconeal process are levered back into their normal position with a periosteal elevator inserted into the joint space. The lateral wound is closed, the dog is turned over and the medial aspect of the joint is exposed. A polyester medial collateral ligament prosthesis is inserted (see above) to prevent redislocation. Following open reduction of elbow luxation a Robert Jones bandage is applied for 7–10 days and exercise is restricted for 6 weeks.

Monteggia fracture/luxation

Fracture of the ulna with cranial luxation of the radial head is known as a 'Monteggia' fracture (Boyd & Boals, 1969; Schwartz & Schrader, 1984). Cranial luxation of the radial head occurs when the annular ligament, which normally binds the radial head to the ulna, ruptures and the ulnar shaft is fractured just distal to the elbow. The ulnar shaft is firmly attached to the radius by the interosseous ligament and consequently moves



(a)



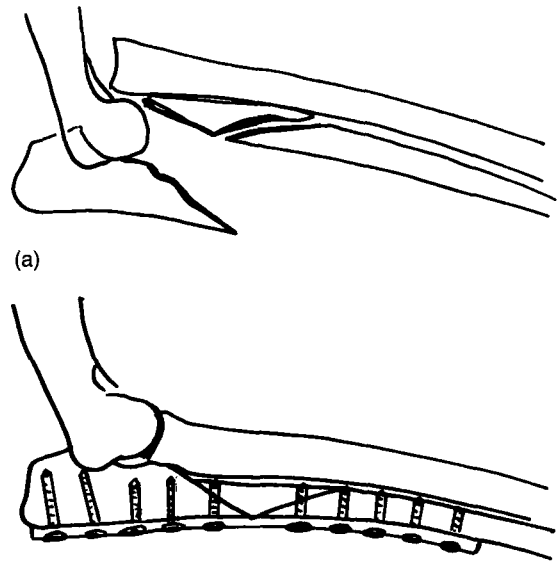
(b)

Fig. 33.25 (a) Cat with Monteggia fracture luxation. (b) Postoperative radiograph. Fixation is achieved using an intramedullary pin and tension band wire. (Reproduced from *British Veterinary Journal* with permission from W.B. Saunders Company Ltd.)

with the radius in a cranial direction (Fig. 33.25a). Provided the injury is recent, the luxation of the radial head can be reduced by manipulation and, because of the strong interosseous attachments between the radius and ulna, reduction of the luxation can be maintained simply by stabilising the ulnar fracture. This is achieved with an intramedullary pin and tension band wire (Fig. 33.25b), or a plate if the fracture is comminuted (Fig. 33.26).

Cranial luxation of the radial head in cats

Cranial luxation of the radial head, associated with rupture of the annular ligament, is occasionally seen in cats (Fig. 33.27a). Open reduction is performed and the radial head fixed to the ulna with a lagged bone screw (Fig. 33.27b). Although there might be concern over this treatment restricting the normal rotation between these two bones in this species, normal function does appear to be regained without requiring the screw to be removed.



(a)

(b)

Fig. 33.26 (a) Comminuted Monteggia fracture luxation. (b) Open reduction and fixation with a plate applied to the caudal (tension) side of the ulna.

Fractures of the proximal ulna and olecranon

Fractures of the proximal ulna can be divided into articular fractures, involving the trochlear notch of the ulna, or avulsion fractures involving the olecranon process (Muir & Johnson, 1996). In both fracture types the olecranon process is distracted by the strong pull of the triceps group of muscles. Internal fixation using the tension band principle (Chapter 12, p. 125) is essential. If an intramedullary pin or screw is used as the sole method of fixation then the articular margin of the fracture serves as a fulcrum which, combined with the pull of the triceps, subjects the intramedullary implant to excessive bending forces and may well lead to

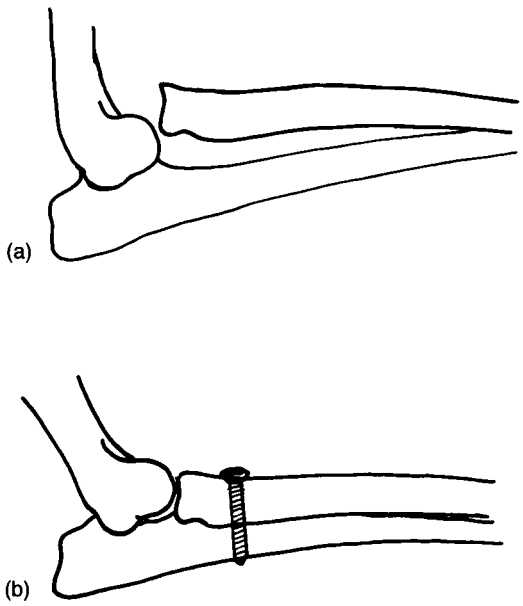


Fig. 33.27 (a) Traumatic luxation of the radial head of a cat. (b) Lag screw fixation of the radial head.

implant failure before healing is complete (Fig. 33.28a). This problem can be overcome, and the tensile forces of the triceps used to advantage in providing functional compression of the fracture, by using a tension band wire placed over the caudal aspect (tension side) of the ulna for fixation (Fig. 33.28b–d). In comminuted fractures of the olecranon (Fig. 33.26a) a plate is used for fixation. Ideally it is placed on the caudal aspect of the ulna (Fig. 33.26b) but if this is not possible then the lateral side is used.

Small avulsion fractures of the proximal olecranon are treated by lag screw fixation or wiring techniques.

Exposure of the olecranon and proximal ulna shaft is achieved through a curved caudolateral skin incision made directly over the olecranon. The extensor carpi ulnaris muscle and the flexor carpi ulnaris muscle are separated and retracted to reveal the shaft of the ulna (Fig. 33.29).

Fracture of the anconeal process

Fracture of the anconeal process, not to be confused with ununited anconeal process, is encountered occasionally (McCartney, 1993). Ideally, these cases are treated by lag screw fixation. Alternatively, excision of the anconeal process can be used (as described under treatment of ununited anconeal process earlier in this chapter).

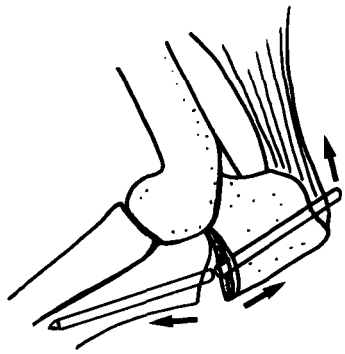
Anconeal fracture can also occur as a complication of comminuted fractures of the olecranon.

Avulsion of the tendon of insertion of the triceps muscles

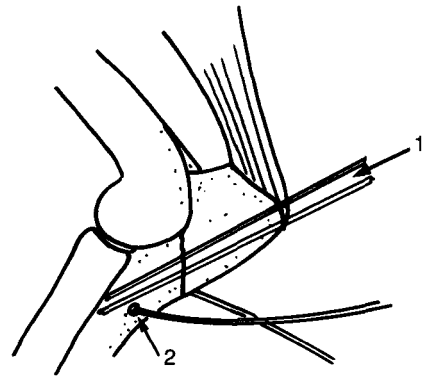
Avulsion of the triceps tendon of insertion is occasionally encountered in the dog. Although trauma is the usual cause, the condition has also been reported as a complication of local steroid injection (Davies & Clayton Jones, 1982). Following rupture or avulsion, the leg is carried in semi-flexion, the animal is unable to extend the elbow and there is a painful swelling over the point of the olecranon. The avulsed tendon can be re-attached using Kirschner wires and a tension band wire (Fig. 33.28d), while ruptures are repaired with Bunnell or Pennington-type locking-loop sutures of monofilament nylon (see Chapter 3, p. 26). If insufficient tendon remains to suture then a band of filamentous carbon fibre or polyester can be passed through the musculotendinous portion of the triceps. The band is fashioned into a figure-of-eight, a tunnel is drilled transversely through the proximal olecranon, one end of the band is passed through the tunnel and the ends are tied, thus securing the triceps to the olecranon. Postoperatively, the leg is kept in full extension with a full-length Robert Jones bandage for 4–6 weeks (the bandage is changed at weekly intervals).

Fractures of the radial head

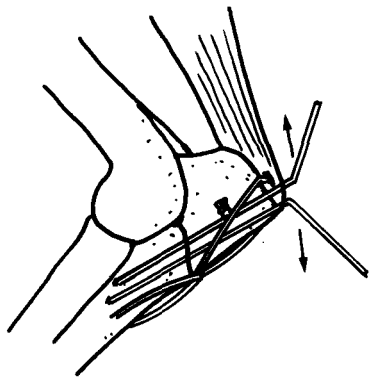
Fractures of the radial head are rare. Management of these fractures has been described by Neal (1975). Open reduction is indicated to restore the integrity of the joint surface and fixation is achieved with a lagged bone screw or Kirschner wire, depending on the size of the fragment. If the fracture is comminuted and cannot be stabilised,



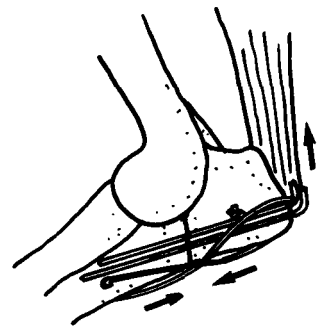
(a) Implant failure due to pull of the triceps muscle.



(b) Initial fixation with two Kirschner wires (1). A wire at point 2 is placed through the ulna distal to the fracture site.



(c) The wire is fashioned into a figure-of-eight tension band and the ends of the Kirschner wires are bent over.



(d) The proximal ends of the Kirschner wires are rotated to fit against the triceps tendon. The tension band wire counteracts the pull of the triceps resulting in functional compression of the fracture.

Fig. 33.28 Olecranon fracture.

then excision of the radial head can be performed as a salvage procedure.

Arthrodesis of the elbow

Arthrodesis of the elbow is only occasionally necessary and is used most often for the relief of chronic pain associated with osteoarthritis. The articular surfaces of the humeroulnar joint are exposed using a caudal approach to the elbow with a transolecranon osteotomy (Fig. 33.30a). The lateral joint capsule is incised to allow exposure of the humeroradial joint. Articular cartilage is removed from all articular surfaces using a high-speed burr or osteotome. Cancellous bone can be collected from the olecranon osteotomy site and packed into the joint. The elbow should be fused at a functional angle of 130–140° (de Haan *et al.*, 1996) and temporary fixation of the joint at this angle is maintained with a Kirschner wire while a dynamic compression plate (DCP) is contoured and applied to the caudal surface of the humerus and ulna (Fig. 33.30b). The olecranon process is attached to the medial epicondyle with a lagged bone screw.

Arthrodesis of the elbow relieves pain but it causes a change in gait. Initially the animal advances the leg by circumduction and tends to drag the toes. Within 3–6 weeks compensatory

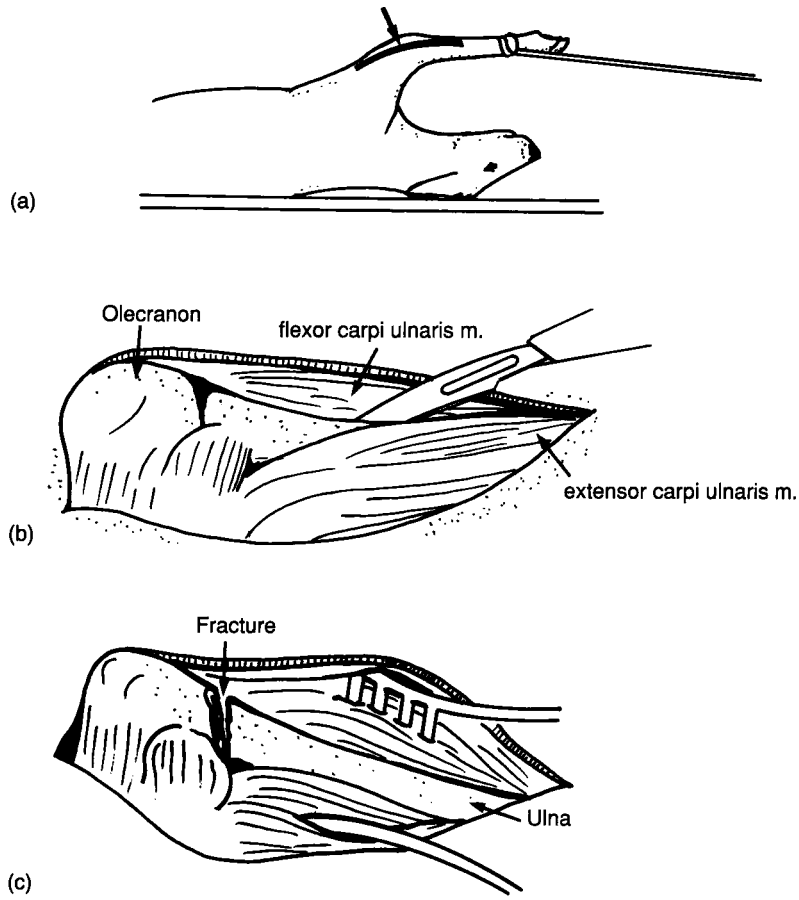


Fig. 33.29 Exposure of the olecranon and proximal ulna. (a) Caudolateral skin incision. (b) Separation of the extensor carpi and flexor carpi ulnaris muscles to expose the ulna. (c) Exposure of the ulna maintained with self-retaining retractors.

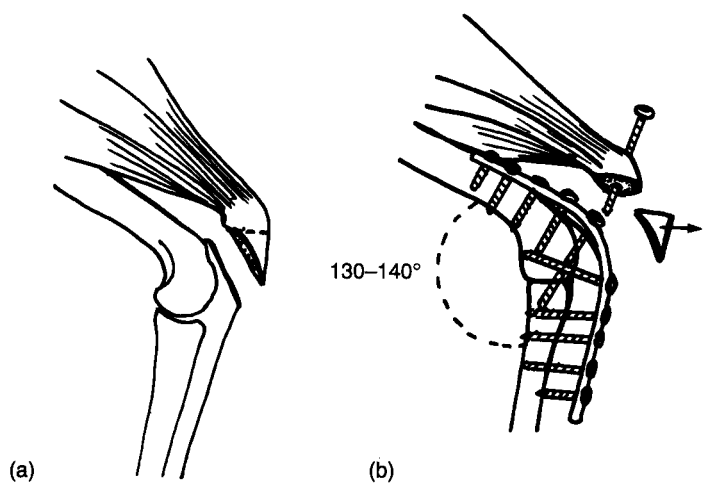


Fig. 33.30 Elbow arthrodesis. (a) Transolecranon osteotomy. (b) Caudal application of a dynamic compression plate (DCP) after removal of part of the olecranon (arrow). The proximal olecranon is reattached to the medial humeral condyle with a lag screw.

movement of the adjacent joints allows the dog to place the foot and a useful degree of limb function is regained. Use of the limb may improve gradually over several months.

Arthrodesis of the elbow creates a longer lever arm than normal. Stress concentration at the distal end of the plate can result in radius and ulna fracture. Consequently, the distal plate screw should penetrate one cortex only to reduce the risk of this complication.

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Chapter 34
The Radius and Ulna

Conditions of the radius and ulna

Conditions of the radius and ulna can be divided into two groups:

- *Acquired*
  - Proximal fractures (see Chapter 33, p. 383)
  - Diaphyseal fractures
  - Distal fractures
  - Osteosarcoma (see Chapter 51, p. 618)
- *Developmental*
  - Growth deformities caused by early distal ulnar growth plate closure
  - Growth deformities caused by early distal radial growth plate closure
  - Growth deformities caused by early distal radial and ulnar growth plate closure
  - Metaphyseal osteopathy (see Chapter 48, p. 607)
  - Panosteitis (see Chapter 48, p. 607)

Diaphyseal fractures of the radius and ulna

These are common in dogs and cats. In the majority of cases the fracture occurs through the middle or distal third of the radius and ulna. Usually both bones are fractured but occasionally a solitary fracture of either the radius or the ulna is encountered (Ness & Armstrong, 1995).

Methods of fixation include:

- External coaptation using casts or splints
- Bone plating
- External skeletal fixation

External coaptation

Although certain radius and ulna fractures can be satisfactorily treated by closed reduction and application of a cast or splint, the method does have its limitations. External coaptation is indicated for greenstick fractures, undisplaced fractures and following reduction of displaced transverse fractures provided at least 50% of the fracture surfaces can be brought into contact. External coaption is not generally recommended for distal radius and ulna fractures in toy and miniature breeds as there is a high incidence of delayed union or non-union in these breeds. The method is also unsuitable for overriding, unstable shaft fractures in adult dogs, where a plate or external fixator would be more appropriate.

If a cast is used to immobilise a radius and ulna fracture, it should extend from the foot to above the elbow. The pads are left exposed so that they can be checked for warmth and swelling, and also to allow some weight-bearing through the bone. In young, rapidly growing puppies, the cast will need to be changed at 10- to 14-day intervals, while in mature dogs changes are made every 3–4 weeks once the initial swelling associated with the fracture has subsided. For further details of application and management of casts see Chapter 12, p. 93.

Plate fixation

It is generally accepted that plate fixation gives consistently good results in the treatment of fractures of the radius and ulna. The method is recommended particularly for mature dogs with overriding transverse fractures, oblique or comminuted fractures. Plate fixation is also

recommended as the primary treatment in toy and miniature breeds of dog (see 'Delayed union and non-union in toy and miniature breeds of dog' below). The plate is usually applied to the cranial aspect of the radius. However, in fractures of the distal radius the plate can be applied to the

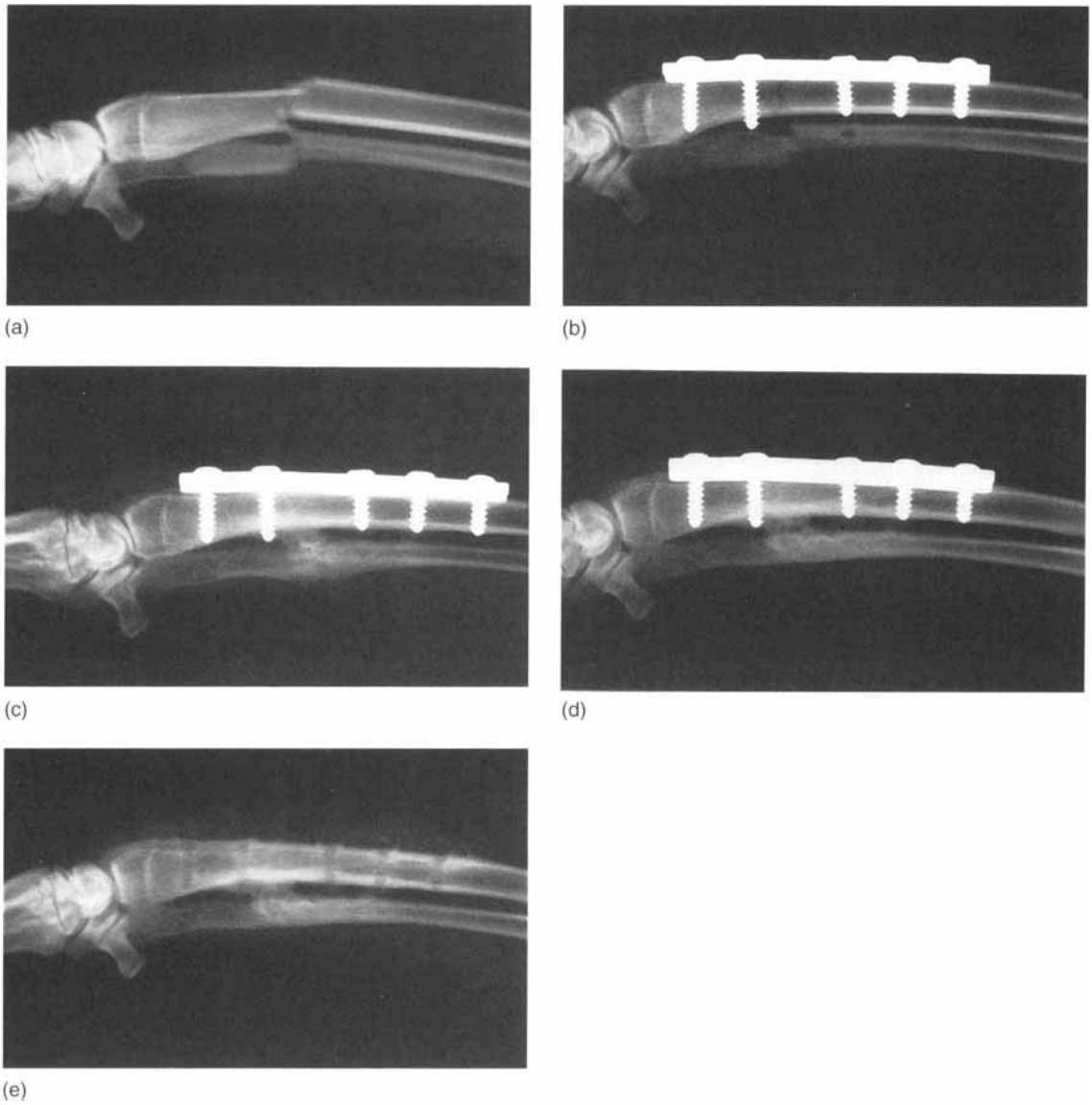

Fig. 34.1 Reproduced from Denny (1990) originally published in Whittick, R.L. (1990) *Canine Orthopedics*, 2nd edn. Lea & Febiger, Philadelphia, PA.
(a) Lateral radiograph of the right radius and ulna of a 1-year-old Saluki with a simple transverse fracture of the distal third of the radius and ulna.
(b) Fracture stabilised with a compression plate (ASIF narrow 4.5 DCP [dynamic compression plate]) applied to the cranial surface of the radius.
(c) Eight-week follow-up radiograph. There is primary bone union at the radial fracture site. However, callus is visible bridging the ulnar fracture site.
(d) Sixteen-week follow-up radiograph. Remodelling of the ulnar callus is complete.
(e) Postoperative radiograph following plate removal at 16 weeks.

medial aspect (Wallace *et al.*, 1992). Generally only the radius is plated (Fig. 34.1). The ulna requires no fixation because the radius is the main weight-bearing bone in the forearm and tends to act as a splint for the ulna because of the interosseous attachments between the two bones. The exceptions to this rule are the large and giant breeds of dog in which plate fixation of both the radius and ulna is recommended (Lappin *et al.*, 1983).

External skeletal fixation

The external fixator is a useful way of dealing with radius and ulna fractures, particularly open and comminuted fractures. A unilateral, uniplanar (type I) fixator is used most often and is applied to the medial or craniomedial surface of the radius. The fixator can be applied following closed reduction or a limited open reduction. The animal can be positioned in dorsal recumbency with the limb suspended from a ceiling hook or drip stand. This creates traction on the fracture using the patient's bodyweight and regains good antebrachial length and orientation of the carpus with respect to the elbow. It also allows good access to all aspects of the forearm making the application of the fixator easier. If open reduction is carried out then initial fixation can be achieved using lag screws, Kirschner wires or cerclage wire before application of the fixator. The distal two-thirds of the medial radius is palpable subcutaneously and offers a safe corridor for the closed insertion of fixator pins (Marti & Miller, 1994). For the more proximal radius, open pin placement is recommended. Two or three pins are usually placed in each of the proximal and distal fragments (Fig. 34.2). In large dogs, or those with a short distal radial fragment, bilateral, uniplanar (type II) frames can be used to provide more stability. For further details of the use of external skeletal fixators (ESFs) see Chapter 12, p. 97.

In miniature and toy breeds of dog, although plate fixation is the preferred method of treatment, an effective alternative is to use transfixion pins (Kirschner wires) and acrylic cement (Fig. 34.3) to form the connecting bars (Eger, 1990; Tomlinson & Constantinescu, 1991).

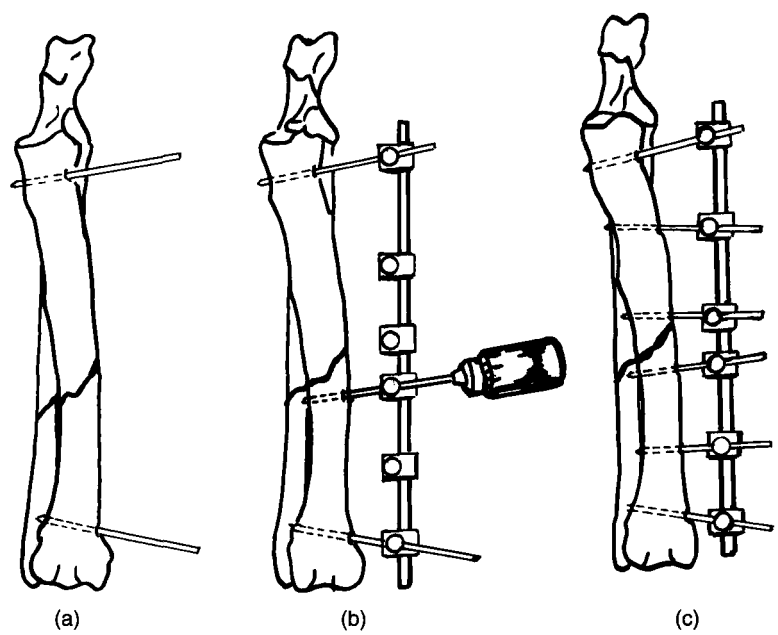

Fig. 34.2 External fixator (unilateral, uniplanar) used for fixation of a mid-diaphyseal radius and ulnar fracture. (a) Placement of the proximal and distal pins. (b) Clamps are placed on the connecting bar, the end clamps are tightened and used to maintain reduction. Insertion of the more central pins use clamps as guides. (c) Six-pin fixator in place.

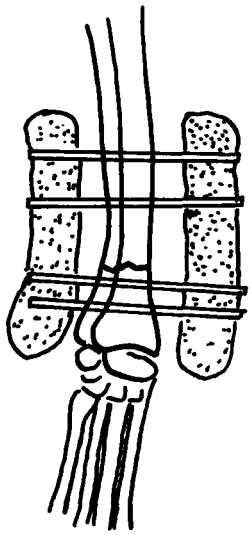

Fig. 34.3 External fixator for use in miniature and toy breeds with radius and ulna fractures. Kirschner wires used as transfixion pins with acrylic to form the connecting bars.

Fractures of the radius and ulna in cats

Radius and ulna fractures in cats tend to be simple transverse fractures involving the distal third of the shaft. It should be remembered that the cat has a much greater range of supination and pronation of the forearm than the dog and this rotatory movement can lead to non-union if the fracture is managed by external coaptation. For best results, a plate (2.7 or 0.2 mm DCP [dynamic compression plate] or mini-veterinary cuttable plate, Stratec) should be used for fixation. The plate is applied to the cranial aspect of the radius. Alternatively an external fixator can be used.

Exposure of radial shaft fractures

The animal is positioned in dorsal recumbency with the fractured forelimb pulled caudally by an assistant. In this position it is easy to manipulate the leg during reduction. Both sides of the leg are accessible and it is a comfortable position for the surgeon to work in, particularly if a plate is to be applied.

If the fracture involves the distal third of the radius, a skin incision is made over the craniomedial aspect of the radius from mid-shaft to carpus (Fig. 34.4a). Care is taken to avoid the cephalic vein (Fig. 34.4b). The extensor carpi radialis muscle is retracted laterally to expose the cranial aspect of the radius (Fig. 34.4b,c). Fractures involving the proximal two-thirds of the radius and ulna are exposed through a skin incision made over the craniomedial aspect of the radius from the elbow to just above the carpus (Fig. 34.5a). The deep antebrachial fascia is incised between the extensor carpi radialis muscle and the flexor carpi radialis muscle to expose the radial shaft (Fig. 34.5b). Proximally, the bone is covered by the supinator muscle. This is elevated to complete the exposure. The radial nerve lies deep to the supinator muscle and must be protected. Distally, the shaft of the radius is covered by the abductor pollicis longus muscle which can be incised and reflected to allow application of the plate (Fig. 34.5c).

Complications of diaphyseal fractures of the radius and ulna

These include:

- Soft tissue problems associated with incorrect use of external coaptation (see Chapters 12 and 13)
- Premature closure of the distal ulnar growth plate
- Delayed union and non-union (especially toy and miniature breeds of dog)
- Malunion (especially of distal fractures)

Premature closure of the distal ulnar growth plate

Although fractures of the distal third of the radius and ulna in immature dogs generally heal rapidly in a cast, a serious potential complication of such fractures is premature closure of the distal ulnar growth plate. Owners should be warned of this possibility. It is always worth taking a radiograph at 3 weeks to check fracture healing and the state of the distal ulnar growth plate. If closure occurs, as noted radiographically or clinically by the development of a deformity, then a section of the ulna shaft must be removed to allow growth of the radius to continue unimpeded, otherwise bowing of the radius and carpal valgus deformity will be

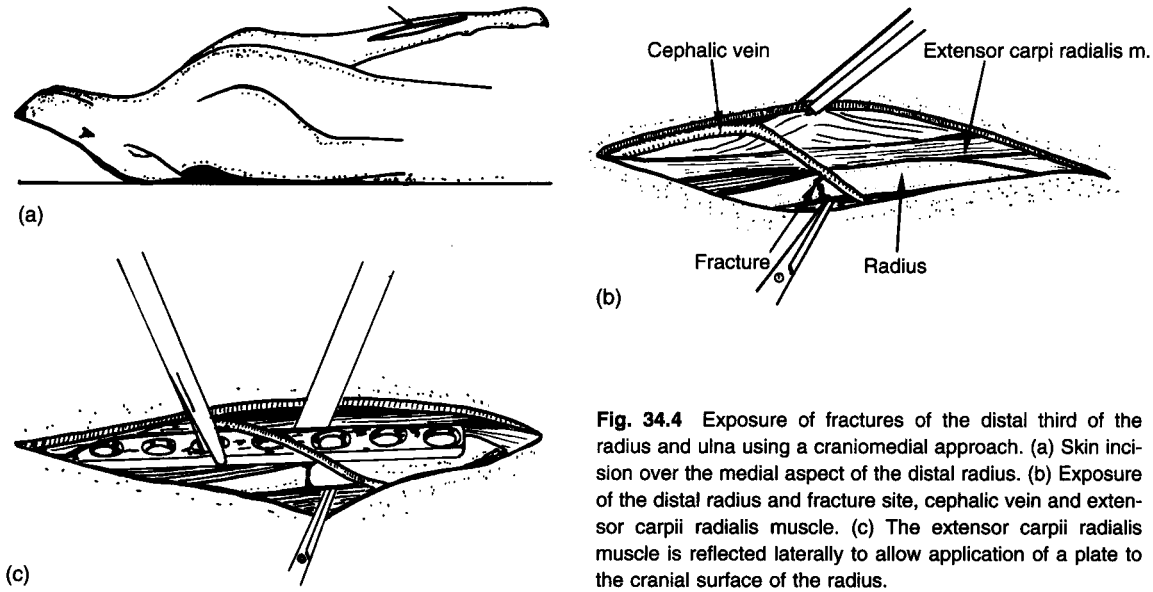

Fig. 34.4 Exposure of fractures of the distal third of the radius and ulna using a craniomedial approach. (a) Skin incision over the medial aspect of the distal radius. (b) Exposure of the distal radius and fracture site, cephalic vein and extensor carpii radialis muscle. (c) The extensor carpii radialis muscle is reflected laterally to allow application of a plate to the cranial surface of the radius.

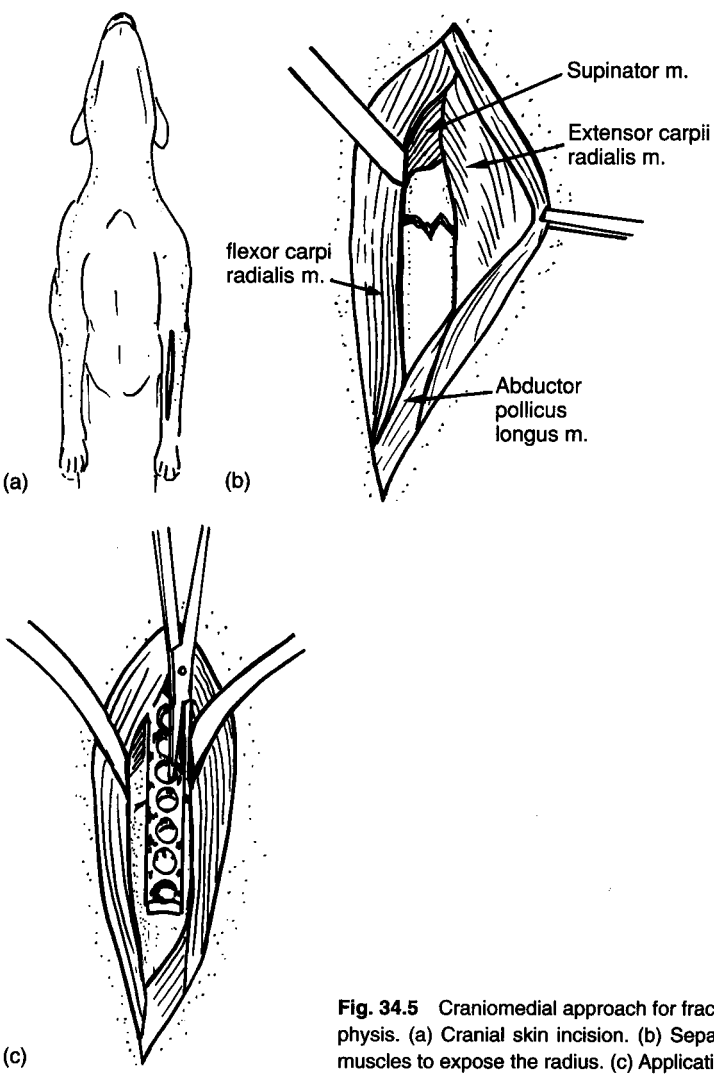

Fig. 34.5 Craniomedial approach for fractures of the proximal two-thirds of the radial diaphysis. (a) Cranial skin incision. (b) Separation of the extensor and flexor carpii radialis muscles to expose the radius. (c) Application of a plate to the cranial surface of the radius.

the end result. Although such deformities can be corrected (see growth distubances, p. 397 under the heading 'Early closure of the distal ulnar growth plate ('short ulna syndrome')'), their likelihood can be reduced by ulnar osteotomy.

Delayed union and non-union

Poor reduction and/or insufficient immobilisation of diaphyseal fractures in casts may lead to malunion, delayed union or non-union and approximately 80% of dogs referred to the authors for treatment of non-union radius/ulnar fractures have been managed by external coaptation initially. Subsequent open reduction and plate fixation gave a successful outcome in the majority of these cases which tends to confirm that most radius and ulnar diaphyseal fractures are best treated by primary plate fixation.

Delayed union and non-union in toy and miniature breeds of dog

Delayed union and non-union are common complications of distal shaft fractures of the radius and ulna in Toy Poodles and other miniature breeds of dog. The non-union is usually biologically inactive radiographically, with little or no callus formation and, in extreme cases, there is bone lysis. Contributory factors include:

(1) The small size of these dogs which makes satisfactory immobilisation of the fracture difficult, no matter whether external or internal fixation is used.
(2) The potential for iatrogenic damage to bone and soft tissues which is much greater in dogs of this size.
(3) In some of these dogs the intramedullary canal is virtually non-existent at this level of the radius resulting in a poor intra-osseous blood supply for fracture healing and, in this part of the forelimb of any dog, the extra-osseous blood supply is not extensive.

Mini-ASIF compression plates used with cortex screws (1.5mm or 2mm in diameter) give good results in the treatment of both fresh and non-union fractures in toy and miniature breeds. If non-union is present, fibrous and cartilaginous callus between the fragments is excised, the fracture surfaces are freshened up, the medullary canal opened and a cancellous bone graft, taken from the proximal humerus, is packed into the fracture site to stimulate osteogenesis. Fixation is achieved with a mini compression plate.

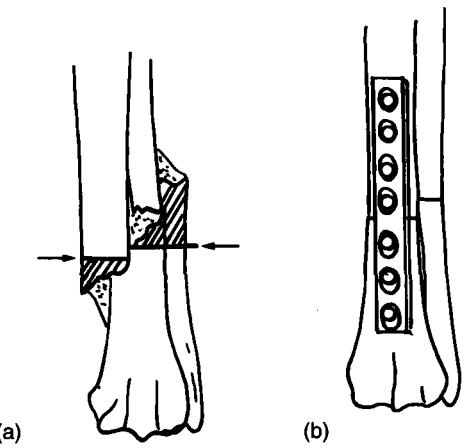

Fig. 34.6 Malunion fracture of the radius and ulna corrected by (a) transverse osteotomy and (b) plate fixation.

Malunion

Malunion results from inadequate reduction and/or immobilisation of the fracture during the healing process. Malunion resulting in angular deformity can be corrected by wedge osteotomy and plate fixation. The method of estimating the position and size of the wedge is illustrated later in Fig. 34.15. However, the majority of these cases are now treated by oblique osteotomy of the radius and ulna and application of an external fixator (see later in Figs 34.16 and 34.17). Transverse osteotomy and plate fixation is used for correction of overriding malunion fractures (Fig. 34.6).

Distal radial fractures

Fractures of the distal radial diaphysis with a short distal fragment

Although plate fixation is the preferred method of treatment for fractures in this region problems arise because:

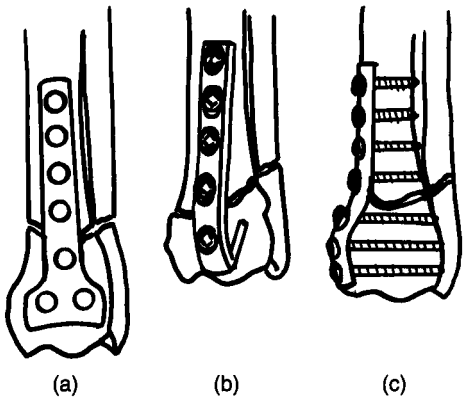

Fig. 34.7 Types of plates used for fractures of the distal radius. (a) 'T' plate, (b) hooked plate, (c) medial plate.

(1) The short length of the distal fragment may not allow placement of enough screws
(2) The cranial application of the plate may interfere with the overlying extensor tendons

Cranial application of a T-plate allows placement of two or three screws in the distal fragment (Fig. 34.7a), while a hooked plate provides stability with a screw and two hooks that are driven into the distal fragment (Fig. 34.7b). A good, third, alternative is to apply a plate to the medial side of the radius (Wallace *et al.*, 1992). This has the advantage of better screw purchase (Fig. 34.7c) than cranial plate application and also a smaller plate can be used (for example a 2.7 DCP instead of a 3.5 DCP in a 25 kg dog). A plate on the medial side of the radius does not interfere with the extensor tendons.

Fracture separation of the distal radial physis (Salter Harris type I or II injury of the distal radial growth plate)

This injury is seen in immature dogs and the distal radial epiphysis is displaced laterally causing carpal valgus deformity. Early closed reduction should be carried out and external support provided with a plaster cast for 3 weeks while healing occurs. Adequate reduction by closed means may prove impossible, especially if treatment is delayed for more than 48 hours. Open reduction is then required using a cranial approach and the epiphysis is stabilised with two

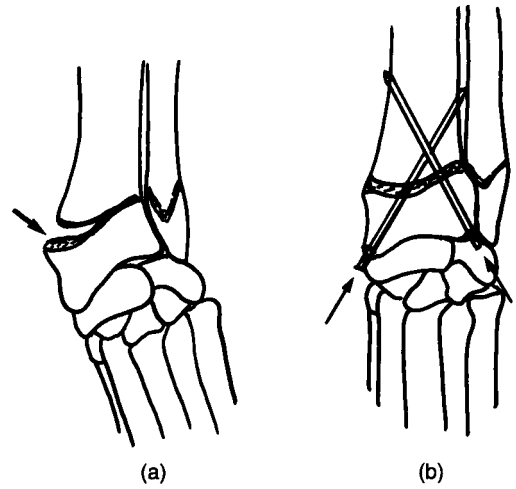

Fig. 34.8 (a) Separation of the distal radial epiphyses, (b) stabilised with two Kirschner wires.

Kirschner wires (Fig. 34.8). The wires are removed after 4–6 weeks. Premature closure of the distal radial growth plate is a potential complication of this injury.

The distal radial growth plate normally contributes approximately 70% of the final length of the radius. Premature closure of the distal growth plate may result in serious shortening of the forearm and an increase in the radiohumeral joint space with consequent elbow instability. The management of this complication is described on p. 404.

Fractures involving the distal articular surfaces of the radius and ulna

These are uncommon and usually seen in dogs that have fallen from a great height. The injury is often bilateral. General principles of dealing with intra-articular fractures apply. Open reduction is carried out to allow accurate anatomical reduction of the fragments. These are stabilised with lag screws or Kirschner wires and then the area is further supported with a neutralisation plate or cast (Fig. 34.9). Care should be taken to evaluate the radiographs for evidence of concurrent carpal injuries which might compromise the result of good internal fixation of the radial fracture.

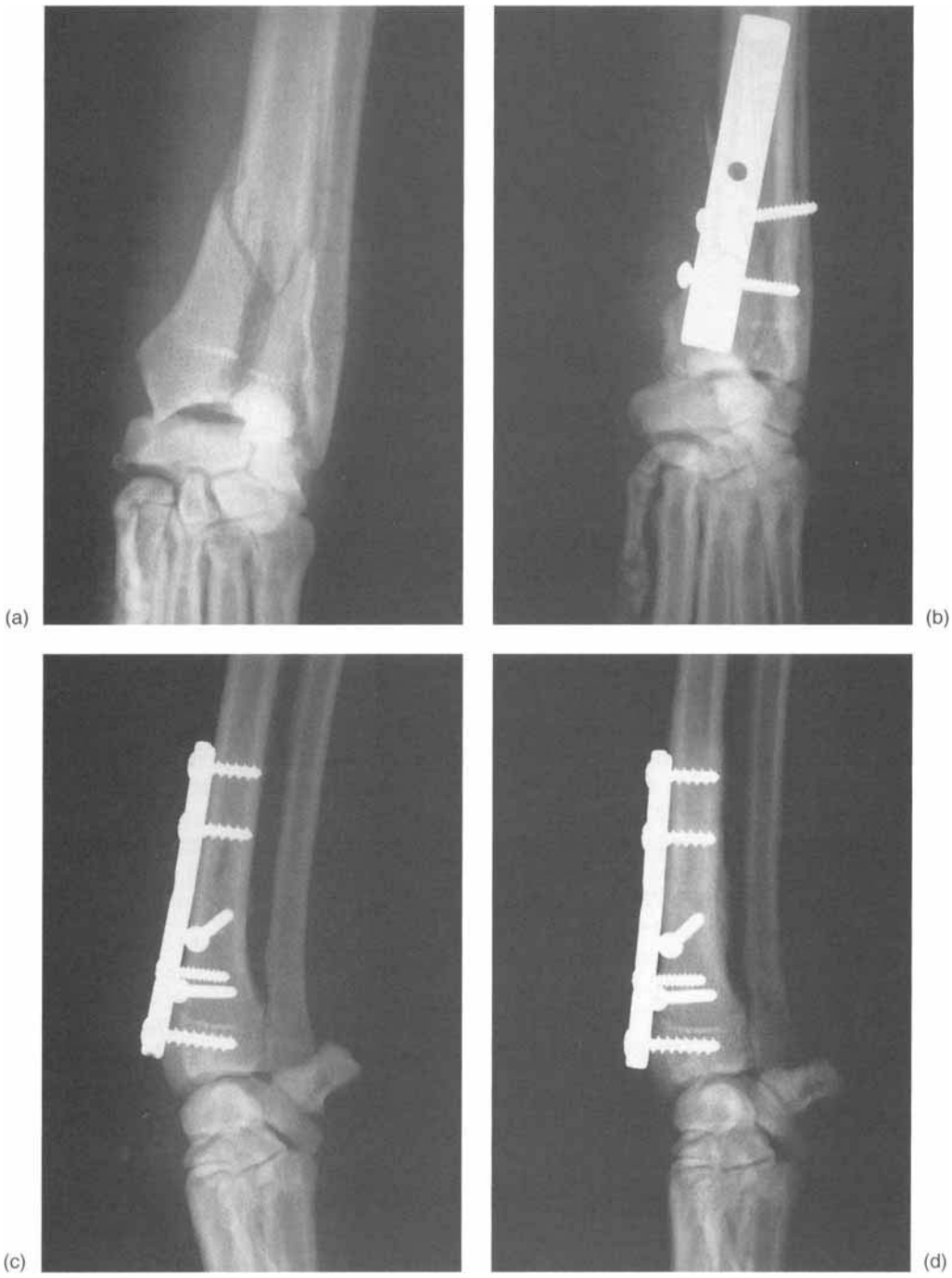

Fig. 34.9 (a) Craniocaudal view of the left carpus of a 2.5-year-old Labrador which had jumped from a church tower and sustained comminuted intra-articular fractures of the distal radius and ulna in both forelegs. (b) and (c) Postoperative radiographs showing reconstruction of the distal radius with lag screws and the application of a neutralisation plate. (d) Six-week follow-up radiograph, showing that fracture healing is complete. (Source: Lea and Febiger, Philadelphia, USA.)

In the presence of carpal injuries it may be prudent to combine fracture fixation with pancarpal arthrodesis.

Fracture of the styloid process of the radius

The medial collateral ligament of the carpus originates from this styloid process and fracture is, therefore, associated with carpal instability. If the fragment is large enough a lag screw is used for fixation. Smaller fragments are retained in position with a Kirschner wire used in combination with a tension band wire.

Fracture of the ulnar styloid process

Fractures of the ulnar styloid process are also associated with carpal instability because the lateral collateral ligament of the carpus originates on the process. Intramedullary fixation of the styloid is achieved with a Kirschner wire combined with a tension band wire (Fig. 34.10). Because of soft tissue injuries and carpal instability associated with styloid process fractures, it is important to provide external support with a gutter splint on the caudal aspect of the carpus for 4–6 weeks.

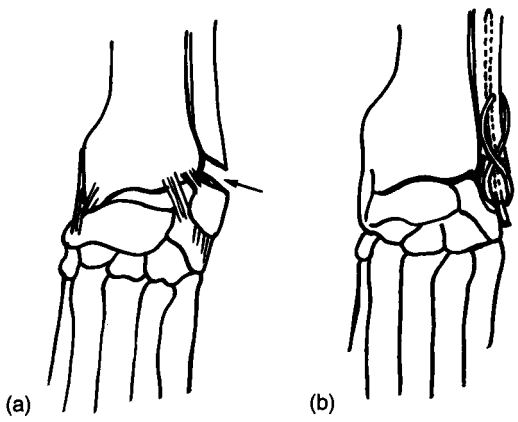

Fig. 34.10 (a) Fracture of the ulnar styloid process, (b) stabilised with Kirschner wire and tension band wire.

Growth deformities of the antebrachium

Local disturbances in bone growth form an important group of orthopaedic problems that occur in young animals. Premature closure of a growth plate can result in angular deformity, limb shortening or both. Causes of premature closure include:

(1) *Growth plate trauma*, particularly Salter Harris type V crush injury of the distal ulnar growth plate (Fox, 1984).
(2) *Abnormal nutrition*. Excessive, prolonged calcium intake in growing dogs results in disturbed endochondral ossification (Hazewinkel, 1989) and causes a variety of clinical conditions which include radius curvus syndrome (physeal osteochondrosis or retained cartilage core of the distal ulnar growth plate), genu valgum and osteochondritis dissecans (OCD).
(3) *Hereditary factors*. An inherited inability of the distal ulnar growth plate to grow as fast as the radial growth plate (*dysostosis enchondralis*) is recognised in chondrodystrophoid dogs especially Basset Hounds and Skye Terriers.

Growth deformities of the antebrachium are the commonest of those seen in dogs. They result from an asymmetrical or reduced rate of growth in one or more growth plates (Fig. 34.11). Since the proximal ulnar growth plate contributes only to development of the olecranon, it does not

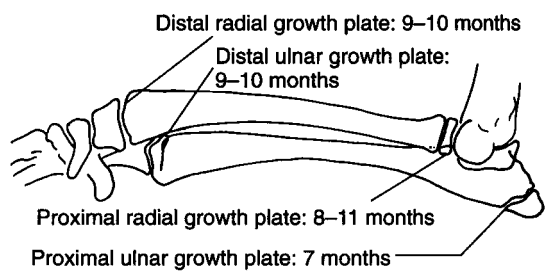

Fig. 34.11 Tracing of a lateral radiograph of the antebrachium in a 5-month-old Labrador showing the growth plates of the radius and ulna and their times of closure.

influence the section of ulna separating the elbow from the carpus. Thus, between the elbow and the carpal joints the ulna grows in length by virtue of activity at its distal growth plate only, whilst both the distal and proximal radial growth plates contribute to its length in relative proportions of about 70:30, respectively. Asymmetrical or reduced rate of growth in any of the radial or the distal ulnar growth plates will produce deformity. Clinical signs associated with such deformity will appear between 3 and 7 months of age. The causes of such alterations in growth rate have been discussed above. The distal ulnar growth plate is the one most commonly affected and this is presumed to be associated with its conical shape which predisposes it to 'crush'-type injuries rather than transverse fractures whatever the direction of insult (Fox, 1984).

Early closure of the distal ulnar growth plate ('short ulna syndrome')

The commonest growth disturbance recorded in the dog is early closure, or retarded growth, of the distal ulnar growth plate. This condition may be seen after other trauma (for example, fractures elsewhere in the bone or limb) or else may develop for no apparent reason. The latter cases are often bilateral and involve dogs of giant breeds especially Great Danes and Irish Wolfhounds. Following early closure of the distal ulnar growth plate, growth of the radius continues at the normal rate but the direction of growth is impeded by the 'bow string' effect of the ulna (Fig. 34.12). Consequently, there is first cranial then medial bowing of the radius (*radius curvus*) causing lateral deviation of the foot (*carpal valgus*). Later, subluxation of the elbow may occur as the head of the radius pushes the humeral condyle proximally. In the Basset Hound and other chondrodystrophoid breeds, elbow subluxation is often the main sequel to premature closure of the ulnar growth plate while carpal valgus may not be so obvious over and above that which is 'normal' for the breed.

Possible sequels to early closure, or retarded growth, of the distal ulnar growth plate include:

- Medial +/– cranial bowing of the radius
- Valgus deformity with lateral deviation +/– outward rotation of the foot
- Humeroulnar subluxation
- Lameness associated with abnormal stresses within the carpus or due to elbow subluxation

The degree of angular and rotational deformity should be evaluated in the weight-bearing patient as this is more reproducible and relevant than the radiographic appearance. In addition, the presence of pain or altered range of movement is

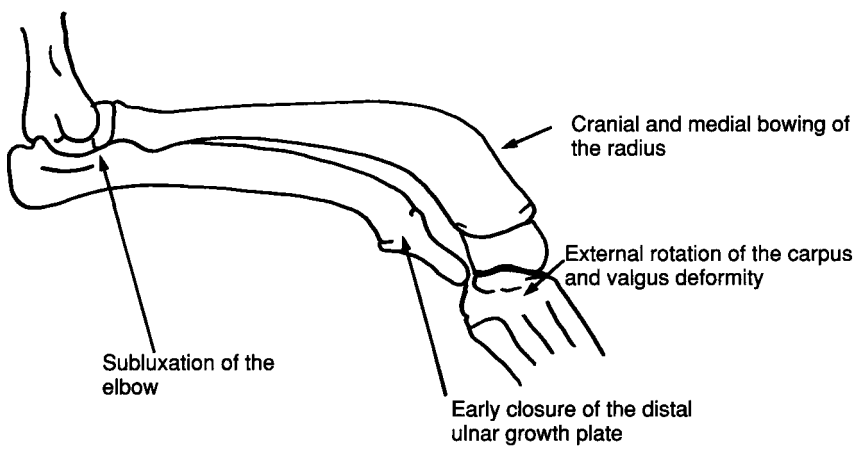

Fig. 34.12 The possible effects of early closure of the distal ulnar growth plate.

a more sensitive indicator of elbow involvement than the radiographic appearance.

Certain breeds 'normally' develop a valgus and rotational deformity of the foot without this being of clinical significance. The same retarded distal ulnar physeal growth rate that produces this may also result in a humeroulnar subluxation that is usually subclinical. However, in some dogs the degree of elbow subluxation, in particular, may be greater than that intended in the breed standards and lameness might then be seen. Such a situation might arise in dogs of any of the chondrodystrophic breeds but most of the cases seen and heard of by the authors involve Basset Hounds.

Radiographic features

Mediolateral and craniocaudal radiographs, which include both the elbow and carpus, are required for evaluation and, if elbow pathology is suspected, further views centred and collimated for this joint should be taken. The abnormalities observed may include:

- Reduced length of the ulna (compared to the contralateral limb – if the problem is unilateral)
- Premature closure of the distal ulnar growth plate (though the radiographic appearance does not correlate closely with remaining growth potential)
- Retained cartilage core (Fig. 34.13) in the distal ulnar metaphysis (significance unresolved)
- Craniomedial bowing of the radius
- Increased interosseous space
- Lateral deviation of the foot (far less important than evaluation of this clinically)
- Humeroulnar subluxation
- Secondary remodelling of the carpal bones or anconeal process
- Secondary carpal or elbow osteoarthritis (i.e. presence of periarticular osteophytes)

Management of angular limb deformity

Conservative management

Within certain physical limits an increase in growth plate compression (i.e. on the concave aspect of an angular deformity) will lead to increased growth. It is prudent to ensure that the dog is being fed a balanced diet and also reduce the rate of growth by decreasing their calorific intake by up to 20–40%.

Such management may be appropriate when:

- Lateral deviation is less than 10–15°
- History suggests non-traumatic aetiology and radiographic appearance suggests growth potential remains

Transphyseal stapling

Carpal valgus deformity can be corrected by temporary transphyseal bridging of the craniomedial aspect of the distal radial growth plate with either a staple or screws and a figure-of-eight wire. Placement of such implants will allow any potential growth on the lateral side to catch up. The staple may need to be removed if there is progression to overcorrection or if the implant loosens and causes irritation and/or lameness. The main disadvantage of this technique is that it reduces overall limb length.

This technique is most appropriate when there is mild to moderate deformity where sufficient ulnar growth remains to allow 'self-correction' (for example, in a 5-month-old giant breed dog with retarded ulnar growth).

The technique for stapling the medial side of the distal radial growth plate (Vaughan, 1976) is:

(1) The leg is prepared for surgery in routine fashion.
(2) A 19 gauge needle is inserted into the medial side of the distal radial growth plate (Fig. 34.13a).
(3) Craniocaudal and mediolateral radiographs are taken to check the position of the needle. The needle is then used as a landmark to decide the correct position for the staple.
(4) The dog is then taken into the operating theatre, the leg is preped again and draped, taking care not to dislodge the needle.
(5) A longitudinal skin incision is made directly over the medial side of the distal radial growth plate (there is no need to incise the periosteum).
(6) The staple is pushed into the bone making

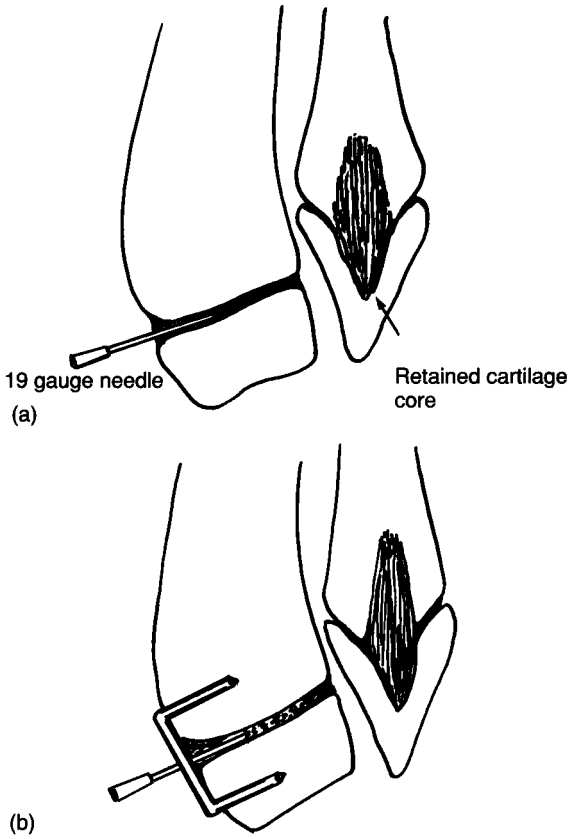

Fig. 34.13 Transphyseal stapling. (a) A marker needle is placed in the medial side of the distal radial growth plate. (b) A staple is used to bridge the medial side of the distal radial growth plate.

sure that the tines of the staple are on either side of the growth plate as judged from the position of the guide needle on the radiographs (Fig. 34.13b). The staple is driven into the bone with a hammer.

(7) The guide needle is removed and the skin is sutured.

(8) Radiographs are taken to ensure that the staple has been correctly placed.

(9) A support bandage is applied for 1 week.

The leg generally takes 4–6 weeks to straighten. The staple must then be removed (unless the dog is almost fully grown) otherwise there may be overcorrection with the foot beginning to deviate medially. Exercise is restricted to short walks while the staple is *in situ*.

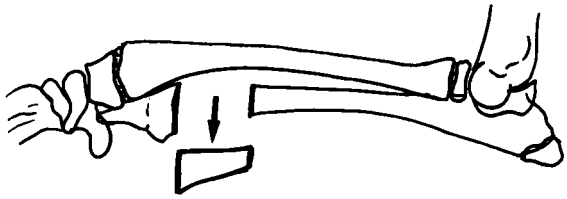

Fig. 34.14 Partial ulnar diaphyseal ostectomy.

Stapling can also be combined with partial distal ulnar diaphyseal ostectomy if the distal ulnar growth plate has closed. A 1–2 cm section of the distal ulnar shaft is removed to relieve the bow string effect of the ulna and allow growth of the radius to continue unimpeded (see below for further detail).

Partial ulnar diaphyseal ostectomy (distal)

Removal of a section of the distal ulnar diaphysis (Fig. 34.14) removes the 'bow string' effect of the ulna on the radius which is then allowed to continue its growth unrestricted and so correct the deformity (Newton, 1974). If this procedure is carried out in a young dog then preventing the ostectomy healing may be necessary to avoid recurrence of the problem. This may be achieved by use of a fat graft or suturing of the periosteum over the cut ends of the bone. However, healing may continue whatever precautions are taken and a second ostectomy may be necessary before the dog is fully grown. The radius is the main weight-bearing bone in the antebrachium and no form of external support is necessary following partial ulnar ostectomy.

This technique may be most appropriate when there is mild to moderate deformity that is expected to worsen due to a lack of ulnar growth potential whilst the radius retains sufficient growth potential to correct limb alignment (for example, in a 3-month-old, small-breed dog with complete distal ulnar growth plate closure following injury).

Corrective osteotomy

Ulnar ostectomy and stapling are designed to prevent further deformity and correct angulation of

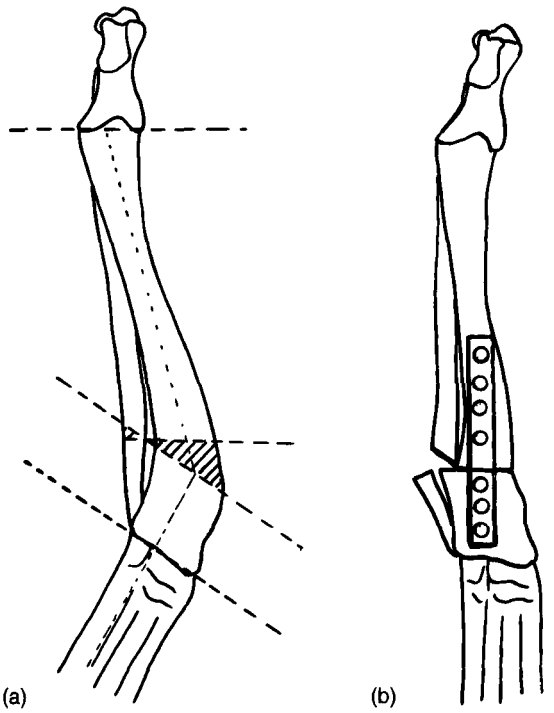

Fig. 34.15 (a) Planning a closing wedge osteotomy to correct an angular deformity. Trace the radiograph of the antebrachium, drawing lines through the radius and carpus (normally these two are parallel with each other). At the site of greatest curvature draw two more lines parallel with the elbow and carpus, respectively, giving the size of the radial wedge to be removed. (b) The osteotomy can be stabilised with a plate or external fixator.

the radius in the *growing* dog after early closure or retarded growth of the distal ulnar growth plate has occurred. Once the distal radial growth plate has closed then the only method of straightening the leg is by corrective osteotomy.

There are several techniques which allow restoration of limb alignment, namely:

- A closing wedge osteotomy – loses some limb length, inherently stable (Fig. 34.15)
- An oblique osteotomy – maintains limb length, inherently stable (Figs 34.16, 34.17)
- An open wedge osteotomy – improves functional limb length, potentially unstable
- A reverse wedge osteotomy – technically difficult, increases limb length, potentially unstable
- A dome osteotomy – technically difficult, maintains limb length, stable

In most cases, limb length is not a problem and a closing wedge or oblique osteotomy is used. Stabilisation can be achieved using a bone plate, in the case of a closing wedge osteotomy, or an external skeletal fixator (ESF) in either of these. Plate fixation is technically more demanding since further corrections postoperatively are not possible and it is difficult to remove any rotational deformity. Uni- or biplanar ESFs may be used but in oblique osteotomies the authors' experience has been that a single-bar unilateral, uniplanar fixator with four or five fixation pins is generally adequate (Fig. 34.17).

The site of osteotomy should be at the point of greatest curvature. This point may be too close to the carpal joint to allow satisfactory placement of implants in the distal fragment. Performing the osteotomy slightly more proximal will lead to the production of a 'double' or 'sigmoid' bend in the antebrachium. Although this is not ideal cosmetically it will usually produce a functional limb and may be safer in ensuring sufficient bone stock distally for secure implant placement. If the angulation is severe and very close to the joint then an alternative is to perform a closing wedge osteotomy and stabilise it with crossed pins. Postoperatively, such stabilisation requires external support with a cast for 4–8 weeks.

Corrective osteotomy is most appropriate when there is a moderate to severe deformity present and there is insufficient radial growth remaining to try and facilitate 'self-correction' – generally after 6 or 7 months of age.

Management of elbow subluxation

Elbow pain and lameness associated with subluxation of the humeroulnar joint secondary to early closure of the distal ulnar growth plate is encountered most often in the Basset Hound but can affect any breed with this type of growth disturbance. There is usually a gradual onset of unilateral or bilateral foreleg lameness at 6–8 months of age. There is pain on elbow manipulation. Diagnosis is confirmed from mediolateral radiographs of the elbows. In confirmed cases there will be an increase in the humeroulnar joint space and the

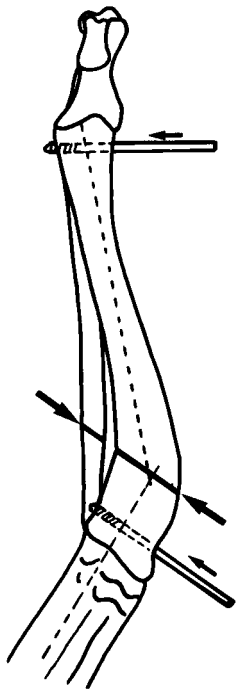

Fig. 34.16 Oblique osteotomy to correct angular deformity. Pins are placed through the proximal and distal radius parallel with the elbow and carpus. Oblique osteotomy is performed of the radius and ulna parallel with the distal pin.

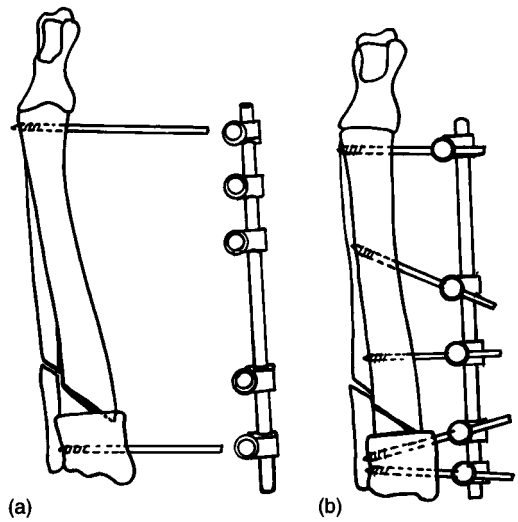

Fig. 34.17 (a) Clamps are placed on the connecting bar, angulation and rotation at the osteotomy are corrected and the alignment maintained by tightening the proximal and distal clamps. (b) The rest of the pins are placed and the clamps tightened.

head of the radius will lie proximal to the coronoid process (Fig. 34.18a).

Conservative management

In some cases, conservative management will allow the problem to either 'self-correct' or else become accommodated for, resulting in resolution of the clinical lameness as the dog approaches skeletal maturity.

Such management may be most appropriate when the degree of lameness and radiographic subluxation are both mild, there is a non-traumatic aetiology and a chondrodystrophic breed is involved (for example, a 6-month-old Basset Hound with mild lameness). If there is no improvement, or deterioration, during a 4- to 8-week period of conservative management then surgery should be reconsidered.

Dynamic proximal ulnar osteotomy

This technique involves creating an oblique or transverse osteotomy in the proximal ulnar diaphysis, close to the elbow joint, which then allows the pull of the triceps muscle to draw the olecranon proximally and reduce the subluxation in a dynamic fashion. In cases presenting at an early stage, i.e. less than 5 months of age, where continued growth might cause recurrence of subluxation once the osteotomy has healed, then a proximal partial ulnar diaphyseal ostectomy (Fig. 34.18b) is used instead. No form of fixation is usually necessary but exercise is restricted for 6 weeks. In the dog with bilateral elbow subluxations a 4- to 6-week interval should be left between operations on each leg.

This procedure may be most appropriate when there is radiographic evidence of an increased humeroulnar joint space in association with elbow pain on manipulation.

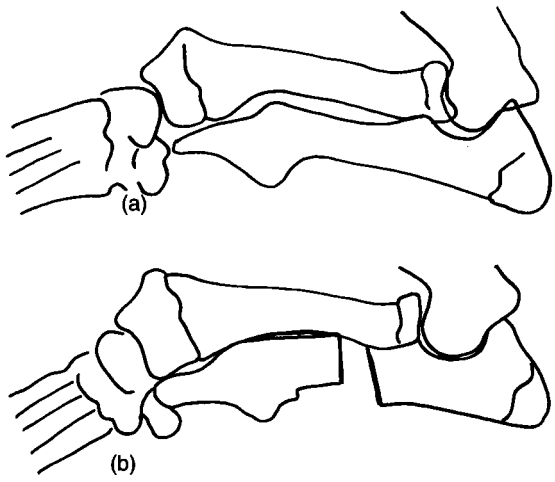

Fig. 34.18 (a) Basset Hound with proximal subluxation of the elbow caused by early closure of the distal ulnar growth plate. (b) Reduction of elbow subluxation by dynamic partial ulnar ostectomy.

A dynamic ulnar osteotomy (or ostectomy) can be combined with other techniques such as transphyseal stapling or corrective osteotomy. It is important to remember that the osteotomy needs to be done close to the elbow joint. Although a distal osteotomy or ostectomy is technically easier it will not allow elbow realignment. Conversely, a proximal osteotomy will not reduce the influence the ulna has on continued radial growth in the same way that a distal ostectomy will.

Prognosis following treatment of early distal ulnar growth plate closure

Prognosis is dependent on many factors, including:

- Age at presentation
- Severity of deformity
- Degree of elbow involvement, especially regarding any secondary change

Cases where a good outcome of normal limb function may be expected would be exemplified by a 5- to 6-month-old Great Dane with mild to moderate valgus deformity and no elbow involvement. Transphyseal stapling would usually produce a very satisfactory outcome. However, the same could not be said for a similar presentation in a St Bernard or Newfoundland where elbow involvement is likely and long-term lameness associated with osteoarthritis is common. Basset Hounds and other chondrodystrophoid breeds presented primarily with elbow subluxation secondary to early ulnar growth plate closure carry a good prognosis for recovery following appropriate management (conservative or surgical, depending on the merits of each case – see above).

Cases in which a guarded prognosis would be given from the outset would be those with established joint pathology or very young dogs (3 months or so) with apparent closure of the distal ulnar growth plate. These dogs have a long period of growth ahead of them and multiple surgical interventions (possibly including a limb lengthening procedure) might be required to achieve satisfactory long-term limb function.

Early closure of the proximal or distal radial growth plate ('short radius syndrome')

Early closure of the distal radial growth plate is seen most frequently and is usually the result of trauma but it may develop for no apparent reason. There is no specific breed predilection but smaller breeds tend to be overrepresented. The relative shortening of the radius caused by the growth disturbance results in:

- Humeroradial subluxation with pain on elbow flexion
- Lameness associated with the elbow subluxation or, occasionally, due to abnormal stresses within the carpus
- Varus (or valgus) deformity of the distal limb (uncommon)

Radiographic features

Diagnosis is confirmed from mediolateral and craniocaudal radiographs which include both the

elbow and the carpus (Figs 34.19 and 34.23) and the same projections, centred and collimated for the elbow, are also required. The radiographic features of short radius syndrome have been described by Clayton Jones & Vaughan (1970) and include:

- Reduced length of the radius (compared to the contralateral limb – if the problem is unilateral)
- Premature closure of the distal or proximal radial growth plate (though the radiographic appearance does not correlate closely with growth potential)
- Humeroradial subluxation with an increased joint space (Fig. 34.19)
- Secondary remodelling of the coronoid processes and/or the anconeal process (fragmentation of the medial coronoid may also occur)
- Straightening of the radius if the closure is symmetrical, or bowing if it is not
- Secondary elbow osteoarthritis (i.e. the presence of periarticular osteophytes)
- Deviation of the foot (only with asymmetric closure of the distal radial growth plate)

Management of 'short radius syndrome'

The aim of surgical treatment is to close the humeroradial joint space and stabilise the elbow. This can be done either by shortening the ulna (partial ulnar ostectomy) or by lengthening the radius. Ulnar shortening is the simplest technique.

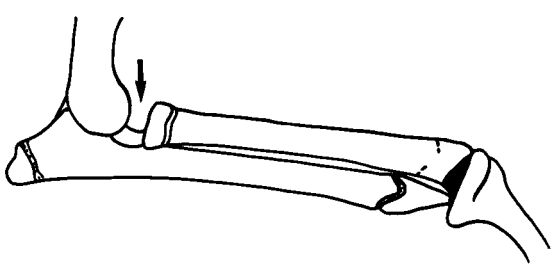

Fig. 34.19 Short radius syndrome. Note the increase in the humeroradial joint space.

Dynamic, partial ulnar ostectomy

Although this technique has the disadvantage of causing further limb shortening, it is quick and easy to carry out, and requires little postoperative care compared with the radial lengthening techniques described below. The caudal aspect of the proximal ulnar diaphysis is exposed. A section of ulna diaphysis (equal to the gap between the humeral condyle and the head of the radius) is removed. The interosseous ligament between the proximal radius and ulna may be sectioned. The ulnar osteotomy site may close, bringing about simultaneous reduction of the humeroradial joint space. The osteotomy may then be stabilised with an intramedullary pin and tension band wire (Barr & Denny, 1985) (Figs 34.20 and 34.21). More recently the procedure has been simplified by leaving the ulnar ostectomy site unstabilised (Fig. 34.22a). Postoperatively the dog is encouraged to bear weight on the leg by the administration of a non-steroidal anti-inflammatory drug (NSAID). The end result is that the ostectomy site in the ulna gradually closes and heals bringing about simultaneous closure of the humeroradial joint space (Fig. 34.22b). The results of both these techniques are the same but the recovery period is reduced if internal fixation is used.

This procedure is most appropriate when:

- The increase in the radiohumeral joint space is relatively small
- The patient is over 5–7 months of age

'Static' radial lengthening procedure (Clayton Jones & Vaughan, 1970)

A transverse proximal radial osteotomy is performed, the interosseous ligament between the radius and ulna is cut, and the osteotomy site is 'spread' pushing the proximal radial segment proximally until it engages the humeral condyle. A cancellous bone graft is packed in the osteotomy gap and fixation is maintained with a plate (Fig. 34.23). Unfortunately, the degree of joint realignment cannot be adjusted postoperatively. This problem can be overcome by combining the radial osteotomy with a dynamic ulnar ostectomy to allow 'fine tuning' postoperatively. Also, continued growth of the ulna may cause a further increase in the humeroradial joint space before

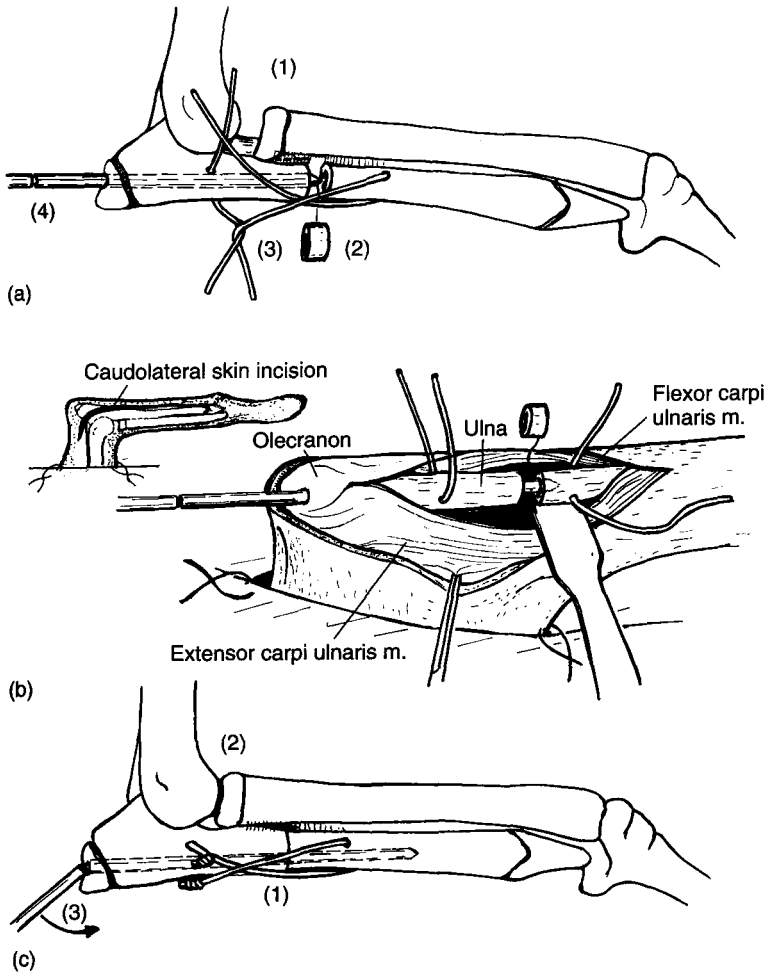

Fig. 34.20 Treatment of elbow instability associated with short radius syndrome by dynamic partial ulnar ostectomy. (a) Surgical technique. 1 – Increased humeroradial joint space; 2 – ulnar osteotomy; 3 – tension band wire; 4 – intramedullary pin. (b) Surgical approach. (c) 1 – Fixation of ulnar osteotomy using intramedullary pin and wire tension band; 2 – humeroradial joint space closed; 3 – intramedullary pin broken off flush with olecranon. (Reproduced from *British Veterinary Journal* with permission from W.B. Saunders Company Ltd.)

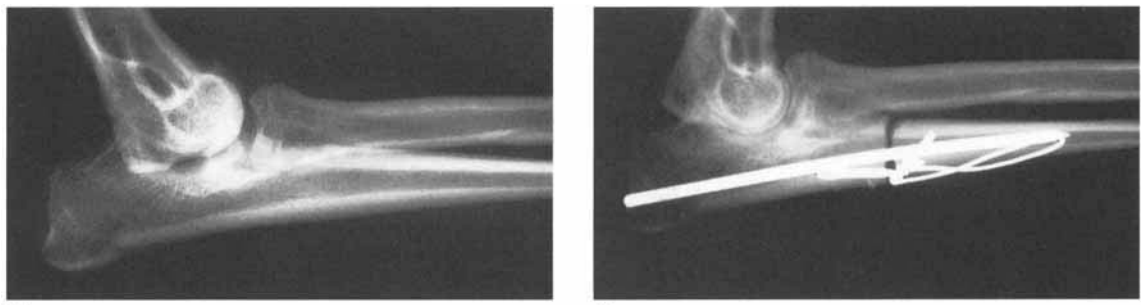

Fig. 34.21 (a) Lateral elbow radiograph of a 7-month-old German Shepherd Dog with short radius syndrome. There is a moderate increase in the humeroradial joint space. (b) Postoperative radiograph. Proximal ulnar ostectomy has been carried out resulting in closure of the humeroradial joint space. The ostectomy site has been stabilised with an intramedullary pin and tension band wire. (Source: *Journal of Small Animal Practice*, vol. **26**.)

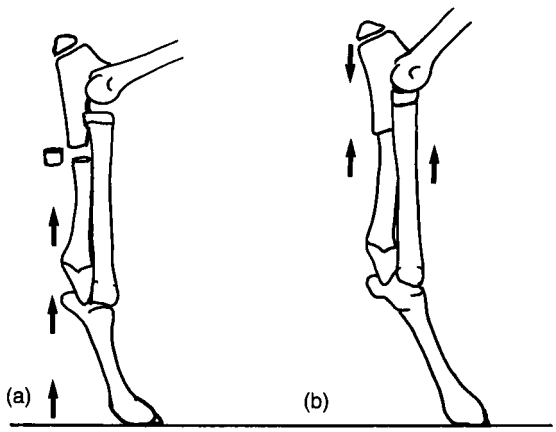

Fig. 34.22 (a) Dynamic partial ulnar ostectomy for short radius syndrome. (b) Weight-bearing results in closure of the ostectomy site and the humeroradial joint space.

the dog reaches maturity and then secondary degenerative change. If such a problem is anticipated then a 'dynamic' radial lengthening procedure might be more appropriate (see below).

This procedure is most appropriate when:

- The humeroradial subluxation is large
- The patient is over 5–6 months of age

'Dynamic' radial lengthening procedure using rubber bands (Mason & Baker, 1978)

Full fixation pins are placed through the proximal radius and either the distal humerus or, more commonly, through the olecranon. Transverse osteotomy of the radius is performed as for static radial lengthening. The pins are connected with strong rubber bands which gradually 'draw' the

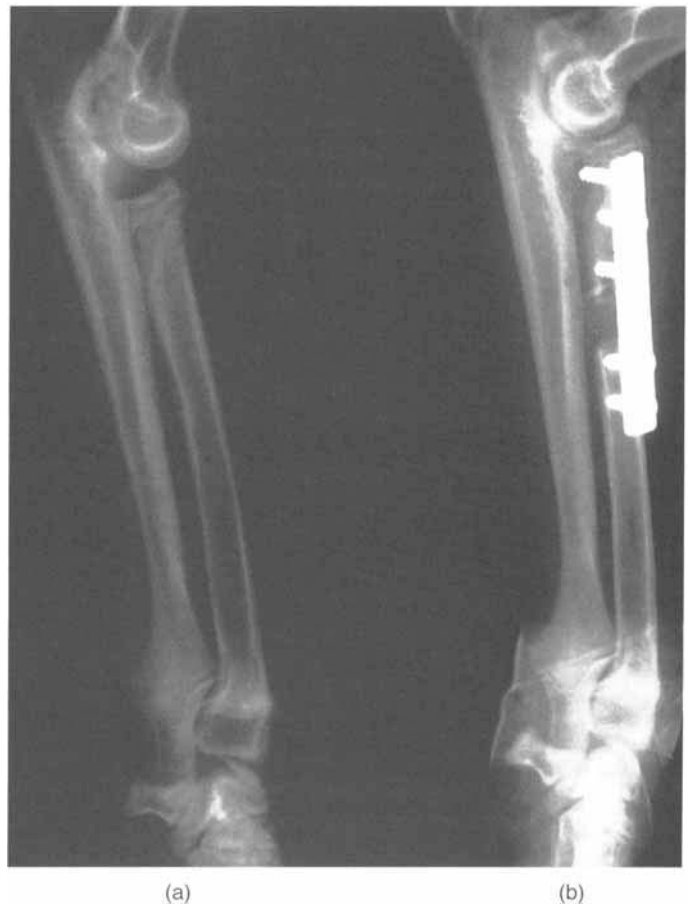

Fig. 34.23 (a) Preoperative radiograph of a 6-month-old crossbreed with short radius syndrome and a marked increase in the humeroradial joint space. (b) Postoperative radiograph. Closure of the humeroradial joint space by radial lengthening, gap at radial osteotomy filled with cancellous bone graft followed by plate fixation. (Source: Blackwell Science, Oxford.)

radial head proximally until it lies against the humeral condyle. This process normally takes 1–2 weeks. Once it has been achieved and confirmed radiographically, the rubber bands can be replaced with connecting bars that are maintained until the radial osteotomy has healed.

This is most appropriate when:

- The humeroradial subluxation is large
- The patient is 5 or 6 months of age

'Dynamic' radial lengthening procedure using external skeletal fixator

This procedure involves the creation of a radial osteotomy and the application of a dynamic external skeletal fixator (for example, an Ilizarov-type ring fixator). This will allow distraction of the radius and reduction of the humeroradial joint space but also continued lengthening of the radius to keep pace with ulnar growth during a period of about 3 weeks. If further distraction is required then a second osteotomy would need to be carried out.

This procedure is most appropriate when the patient is very young (<4 or 5 months), with considerable growth potential remaining.

Management of asymmetrical closure of the distal radial growth plate

- *Young patients, or those with only mild deformity*, may be treated by altering the balance of growth by such methods as:
  - transphyseal stapling on the side of greater growth
  - periosteal elevation to encourage growth on the side that is restricted
  - removal of the damaged portion of the growth plate using an air drill/burr and packing the space with a fat graft.
- In *older patients*, at the end of their growth phase, a definitive corrective osteotomy is more likely to be appropriate but the level of the osteotomy will often be very distal, requiring stabilisation using a technique such as cross-pinning.
- In *younger patients, with severe deformities*, it may be necessary to consider a corrective osteotomy to realign the limb and then subsequent radial and ulnar osteotomy with application of a dynamic fixator to allow distraction and maintenance of limb length.

Prognosis following treament of early distal radial growth plate closure

This varies from good, in say a 6-month-old dog with minor elbow subluxation and no secondary changes (where a single dynamic ulnar ostectomy is likely to suffice) to guarded in, say, a 3-month-old dog showing marked relative radial shortening and early coronoid remodelling (where a dynamic radial lengthening procedure extended over several months may need to be planned).

Reduced growth rate in both radius and ulna

Injury to the distal growth plates of both the radius and ulna will lead to overall antebrachial shortening, as well as angular deformity or joint subluxation if the involvement is asymmetrical. Although there may be some compensation for such shortening by using the limb with a greater degree of joint extension and/or compensatory increase in humeral length, it may be necessary to consider limb lengthening to regain normal function. This can be achieved by creating mid-diaphyseal osteotomies of the radius and ulna and applying a dynamic external fixator (for example, an Ilizarov-type ring fixator or modified bilateral, uniplanar fixator incorporating threaded connecting bars) to achieve distraction osteogenesis (Latte, 1998).

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A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny, Steven J. Butterworth

Chapter 35
The Carpus

Anatomy

The carpus is a compound, ginglymus joint, allowing flexion/extension and slight lateral angulation. The antebrachiocarpal joint accounts for 80–90% of carpal movement. The anatomy of the carpus is illustrated in Figs 35.1–35.3. There are seven carpal bones, three in the proximal row (radial, ulnar, accessory) and four in the distal row which are numbered, first, second, third and fourth. A small sesamoid bone (phacoid) lies in the tendon of the abductor pollicis longus muscle on the medial aspect of the intercarpal joint. There are also two small sesamoids which lie on the palmar aspect between the two rows of bones.

The bones of the carpus form three main joints: the proximal, the middle and the distal. The bony anatomy of the carpus affords little inherent stability which is dependent upon surrounding soft tissue support. Although the flexor and extensor tendons contribute some support, stability is primarily provided by the ligaments. Dorsal support of the joint is minimal due to this side being under compression and thus inherently stable. The most important ligaments are those providing collateral and palmar support.

There are no continuous collateral ligaments. Medially the short radial ligament, which is made up of straight and oblique parts, runs from the radial styloid process to the radial carpal bone, while laterally the short ulnar ligament runs from the ulnar styloid process to the ulnar carpal bone. On the palmar aspect, two ligaments extend from the accessory carpal bone to the fourth and fifth metacarpal bones. Another important supporting structure is the transverse palmar carpal ligament which not only supports the carpus but also forms the carpal canal enclosing the two flexor tendons and the main arteries, veins and nerves to the foot. There are several other ligaments linking the distal radius and ulna to the proximal carpal bones and there are short ligaments joining the carpal bones transversely in their two rows.

Radiographic examination of the carpus

Routinely, dorsopalmar and mediolateral views are required. In addition, a flexed mediolateral view is helpful when accessory carpal bone fracture is suspected, a stressed mediolateral view is essential for determining the degree and level of hyperextension injuries and stressed dorsopalmar views are useful in the assessment of ligament injuries and fractures of individual carpal bones.

Conditions of the carpus

Conditions of the carpus can be divided into four groups:

- *Congenital*
  - Ectrodactyly
- *Developmental*
  - Osteochondritis dissecans (OCD)
  - Flexural deformity
  - Hyperextension
  - Subluxation (see radial/ulnar growth disturbances covered in Chapter 34)
- *Traumatic*
  - Fractures of the accessory carpal bone
  - Fracture luxation of the radial carpal bone
  - Fracture of the ulnar carpal bone
  - Fracture of the numbered carpal bones
  - Carpal sprain
  - Dorsal radiocarpal ligament sprain

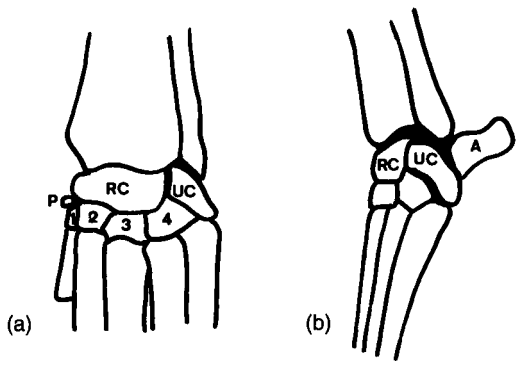

Fig. 35.1 Bones of the left carpus, (a) dorsal view and (b) lateral view. A – Accessory carpal bone; P – phacoid; RC – radial carpal bone; UC – ulnar carpal bone.

- Enthesiopathy of the short radial collateral ligaments
- Luxation of the antebrachiocarpal joint
- Open carpal luxations and shearing injuries
- Luxation of the middle carpal joint
- Luxation of the carpometacarpal joint

- *Acquired*
  - Osteoarthritis
  - Immune-mediated arthropathy (see inflammatory arthropathies covered in Chapter 8)
  - Osteosarcoma (see Chapters 34 and 51)

Congenital disorders

Ectrodactyly

This is a rare condition (Carrig *et al.*, 1981) resulting from abnormal cleavage of the embryological limb bud. The foot is split and there is absence of one or more of the carpal, metacarpal and phalangeal bones. Most animals compensate reasonably well for the deformity and require no treatment.

Developmental disorders

Osteochondritis dissecans (OCD)

Osteochondritis dissecans (OCD) is occasionally encountered in the carpus affecting either the distal radius (Butler *et al.*, 1971) or the radial carpal bone (Kralj & Leskovar, 1990). Principles of treatment are described in Chapter 4.

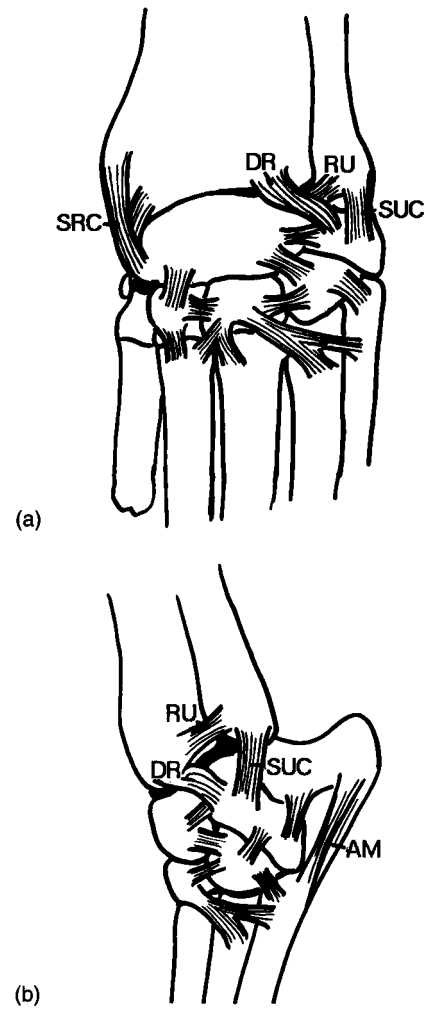

Fig. 35.2 Ligaments of the left carpus, (a) dorsal view and (b) lateral view. AM – Accessorometacarpal ligaments; DR – dorsal radiocarpal ligament; RU – radioulnar ligament; SRC – short radial collateral ligament; SUC – short ulnar collateral ligament.

Flexural deformity of the carpus in puppies

Flexural deformity of the carpus can occur as a unilateral or bilateral problem and is usually recognised at 6–12 weeks of age (Vaughan, 1992). In the series of cases reported by Vaughan there was a preponderance of Dobermann Pinschers

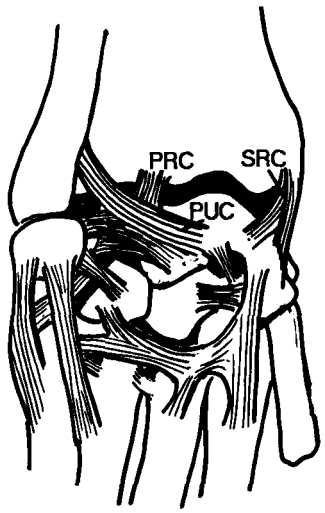

Fig. 35.3 Palmar ligaments of the left carpus. PRC – Palmar radiocarpal ligament; PUC – palmar ulnocarpal ligament; SRC – short radial collateral ligament.

(17 out of 21) but the condition has been recognised in several other breeds by the authors, and may follow a change in diet.

The condition is associated with excessive tension in the tendons of the flexor carpi ulnaris muscle. Clinically, there is knuckling of the carpus as the puppy stands and walks. In severe cases carpal hyperflexion and outward bowing of the carpus occurs and the puppy tends to stumble when walking.

Spontaneous recovery occurs in most cases and consequently a conservative approach to management is used in those cases with moderate signs and in which the carpus can still be fully extended. A regime of reduced activity is advised and, if necessary, a support bandage is used. It should be ensured that the puppy is being fed a balanced diet. Improvement should occur within 2–3 weeks with full recovery by 4 weeks after the onset (Vaughan, 1992). If the flexural deformity persists then correction of carpal posture can be attempted by sectioning of both tendons of insertion of the flexor carpii ulnaris muscle about 1 cm proximal to the accessory carpal bone.

Hyperextension of the carpus in puppies

Hyperextension of the carpii is a relatively common problem in puppies. Young German Shepherd Dogs are affected most frequently and the condition results in 'dropping' of the carpii and a palmigrade stance. The hyperextension is due to laxity of the carpal flexor tendons associated with poor muscle tone.

The aim of treatment is to improve muscle tone with a regime of short frequent walks (Shires *et al.*, 1985). Carpal posture tends to improve by the time the puppy reaches maturity. The use of casts or splints should be avoided in these cases as carpal posture becomes worse once external support is removed.

Traumatic conditions

Carpal sprains

Carpal sprains are common in racing Greyhounds and working dogs. Sprain of the palmar carpal ligaments as a result of carpal hyperextension during racing or working are common while sprains affecting the dorsal carpal ligaments are seen less frequently (Guilliard, 1997). It is usually the right carpus which is injured since this limb takes most of the bodyweight as the Greyhound corners counterclockwise at high speed. Carpal sprain injuries are often not accurately differentiated because carpal swelling and pain may mask more significant pathology and, as a result, appropriate treatment is not always given (Dee, 1987). By a process of careful palpation and radiographic examination it should be possible to make a fairly accurate assessment of which ligamentous structures have been damaged. Ligamentous sprain injuries can be acute or chronic and can be classified into three grades according to degree of injury, ranging from mild (grade 1) through to severe (grade 3) in which there is complete disruption or avulsion of the ligament (Roy & Dee, 1994).

Specific ligament injuries are discussed below under the various carpal luxations.

Dorsal radiocarpal ligament sprain

Sprain of the dorsal radiocarpal ligament (Fig. 35.2) has been described by Guilliard (1997). Diagnosis is based on the clinical signs which include a swelling over the dorsolateral aspect of the carpus and pain on carpal flexion. If there is

an avulsion of the origin of the ligament, then radiographs of the carpus (straight and flexed mediolateral views) may demonstrate the avulsed fragment of bone over the dorsal aspect of the distal radius. Prognosis for full recovery is good. Surgery is only indicated if there is an avulsion injury, in which case the bone fragment is removed, otherwise conservative management is recommended with the application of a carpal splint for 4 weeks followed by restricted exercise for a further 4 weeks.

Enthesiopathy of the short radial collateral ligaments

Calcification at a bone–tendon or ligament interface is known as *enthesopathy* or *enthesiopathy*. Enthesiopathy, affecting the origin of the short radial collateral ligaments which arise from an unnamed tubercle on the medial aspect of the distal radius just proximal to the styloid process (Fig. 35.4), has been reported in the racing Greyhound (Guilliard, 1998). In a radiographic survey of the carpii of 100 racing Greyhounds there was a 14% incidence of enthesiopathy of the short radial collateral ligaments. However, this was not identified as a clinical problem in any case, nor was there any evidence that the lesion had any adverse effect on performance. The enthesiopathy should be regarded as an inconsequential finding and not a cause of lameness unless clinical signs suggest otherwise.

Fracture of the accessory carpal bone

Fracture of the accessory carpal bone is a common injury in the racing Greyhound that is frequently overlooked (Hickman, 1975). The injury results from carpal hyperextension during racing. The right leg is usually involved and the fracture tends to occur when the dog is rounding a bend and suddenly changes direction (Bateman, 1960). Lameness becomes apparent during or soon after racing. The injury may be dismissed initially, by the trainer, as a carpal sprain and treated conservatively. However, intermittent lameness persists, especially after exercise, and there is pain on direct palpation of the accessory carpal bone and on carpal flexion.

Accessory carpal bone fractures have been classified into five types (Johnson, 1987) and these, along with the soft tissue attachments to the accessory carpal bone with which they are associated, are illustrated in Figs 35.5 and 35.6:

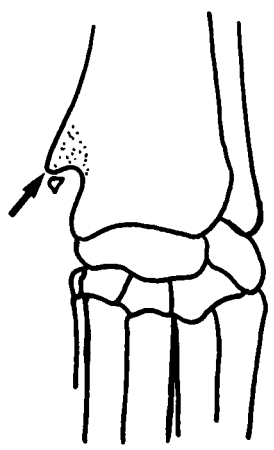

Fig. 35.4 Short radial collateral ligament enthesiopathy. Area of enthesiopathy indicated by arrow.

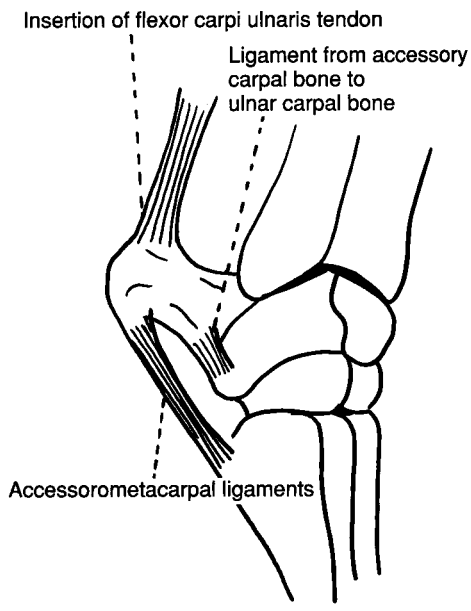

Fig. 35.5 Ligamentous attachments to the accessory carpal bone.

- *Type 1* fractures from the distal articular surface are the most common and accounted for 68% of cases reported by Johnson *et al.* (1988). The prognosis for a successful return to racing after surgical excision of the fragment is poor. However, as a general rule, small fragments are excised while larger fragments should be screwed back in position (Fig. 35.6). A carpal flexion cast should be applied for 6–8 weeks following surgery.

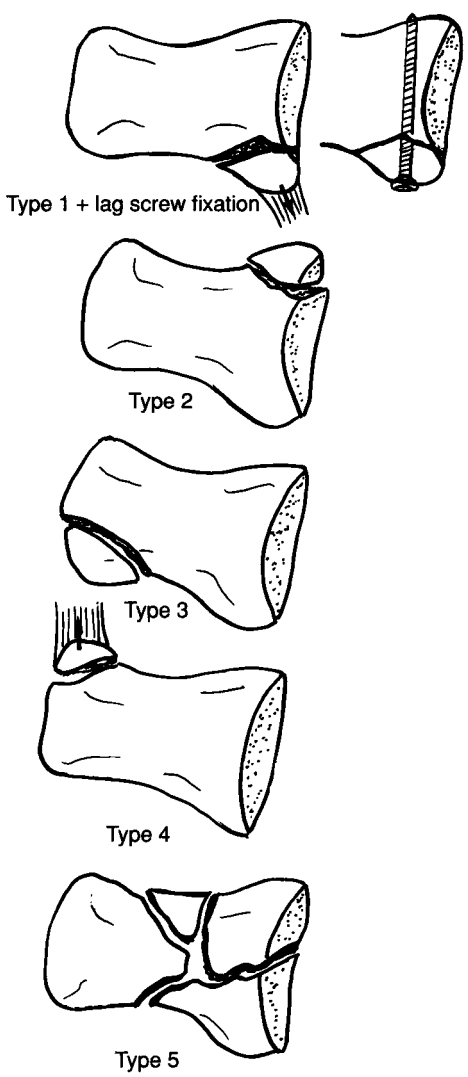

Fig. 35.6 Types of accessory carpal bone fracture (Johnson, 1987).

- Brinker *et al.* (1990) recommended lag screw fixation for *types I, II and III* injuries and claimed that 90% of dogs treated in this way return to racing. The discrepancy in reported results may lie in the fact that in Australia dogs are only allowed to return to racing at the same grading they were when injured, whereas in the USA they can return to racing at a lower grade.
- *Type IV* fractures are avulsion injuries involving the tendon of insertion of the flexor carpi ulnaris muscle and its attachment to the proximal surface of the caudal end of the accessory carpal bone. These fractures are best treated by surgical excision of the fragments with repair of tendinous attachments, followed by application of a cast for 6–8 weeks.
- *Type V* fractures are comminuted and are best treated by application of a flexion cast (Dee, 1991). These fractures carry a very guarded prognosis for a return to racing.

In all cases the cast is applied with the carpus in a moderate degree of palmar flexion initially. Then, with each cast change, the degree of flexion is gradually reduced until the normal standing position is reached.

Fracture of the radial carpal bone

Fractures of the radial carpal bone are rare. Sagittal fractures of the radial carpal bone have been encountered most often in the Boxer. The bone develops from three centres of ossification (Vaughan, 1985) and it is thought that the fracture may result from incomplete ossification of the cartilage precursor. Sagittal or dorsal slab fractures are treated ideally by lag screw fixation if the fragment is large enough (Fig. 35.7) or by surgical excision if the fragment is small. When lag screw fixation is used, care should be taken to ensure that the screw head does not interfere with carpal movement and, if necessary, the screw head should be countersunk.

Fracture of the ulnar carpal bone

This fracture is occasionally seen as a complication of medial luxation of the radial carpal bone

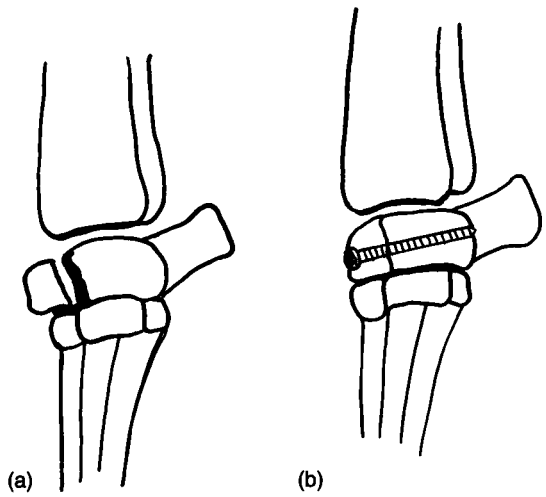

Fig. 35.7 (a) Fracture of the radial carpal bone. (b) Lag screw fixation.

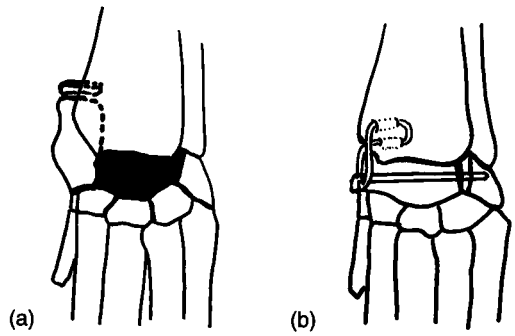

Fig. 35.8 (a) Luxation of the radial carpal bone associated with fracture of the ulnar carpal bone. (b) Stabilisation with Kirschner wire and medial collateral ligament replacement with prosthesis.

(Fig. 35.8a). Open reduction is performed and the fracture luxation stabilised with a Kirschner wire or lag screw placed transversely through the radial and ulnar carpal bones (Fig. 35.8b). Reconstruction of the medial collateral ligament is often also necessary (see 'Carpal luxations' below).

Fractures of the numbered carpal bones

Chip fractures of individual carpal bones are seen more frequently than fractures of the radial or ulnar carpal bones. As a general rule, these are treated conservatively or the fragments are removed to reduce any secondary carpitis.

Exposure of the carpus

A prerequisite for accurate surgery of the extremities is a bloodless field and this is best achieved by expressing the blood from the lower limb with a rubber Esmarch bandage followed by application of a tourniquet at the elbow. The Esmarch bandage is removed once the tourniquet is in place. An effective tourniquet should be released within *1 hour* of application and it is important that a support bandage is applied *before* removal of the tourniquet. There is always some delay between application of the tourniquet, final preparation of the limb and transfer of the animal to the operating theatre. This problem can be overcome, and time gained, by using a sterile Vetrap bandage (3M) as a combined Esmarch/tourniquet. Once the leg has been draped for surgery the sterile Vetrap bandage (3M) is tightly applied from foot to elbow exsanguinating the lower leg. On reaching the proximal end, a length of the bandage is twisted to form a 'rope' which is then wrapped around the limb three or four times to form the tourniquet. The carpal arthrotomy incision is then made directly through the Vetrap (3M) and the skin edges can be clipped or sutured to the edges of the bandage. This type of tourniquet tends to become less effective after an hour and the authors have had no complications resulting from keeping the Vetrap (3M) in place for up to about 90 minutes.

An axial skin incision is made over the dorsal aspect of the carpus. The deep antebrachial fascia is incised between the tendons of the extensor carpi radialis and the common digital extensor muscles. These tendons are retracted to allow exposure and incision of the joint capsule over the carpal bones. The synovial membrane adheres to the dorsal surface of individual carpal bones and must be dissected off the bones, as necessary, to achieve the required exposure. Following wound closure, a support bandage is applied, preferably before release of the tourniquet.

Carpal luxations

History and clinical signs

Carpal luxations are usually traumatic in origin. Although the injury can result from involvement in road traffic accidents, the more common history is that of twisting awkwardly while running, for example putting the foot down a hole whilst moving at speed, or else jumping down or falling from a reasonable height. Alternatively, in some dogs, particularly the collie-type breeds, the progressive development of a palmigrade stance may be reported. This is associated with degenerative changes, of unknown aetiology, within the palmar ligaments which progressively weaken and stretch allowing hyperextenion of the carpus during weight-bearing.

On examination of patients with traumatic carpal luxation there will be severe lameness associated with pain on carpal manipulation. If weight-bearing is possible then there may be hyperextension of the joint if the palmar ligaments are damaged, and medial or lateral deviation of the foot if collateral ligament injury is present. Manipulation of the joint may confirm these suspicions although the degree of pain may preclude definitive examination until the dog is anaesthetised. In the chronic, degenerative cases the pain, probably due to secondary osteoarthritis, will be less marked and the lameness will be far less severe.

Diagnosis

Although the history and clinical signs will be highly suggestive of carpal injury, radiography is essential for determining the extent of carpal damage. Mediolateral and dorsopalmar views will help to show the presence of fractures, including avulsion fractures associated with collateral instability, and obvious luxations. Stressed views may be required to demonstrate collateral or palmar instability. In recognising such instability it is necessary to try and establish which joints are involved as this may influence the choice of treatment. It is important also to scrutinise all films for the presence of chip fractures, as these may affect the prognosis following restoration of stability by influencing the development of secondary osteoarthritis.

Treatment

Although surgical repair or replacement of carpal ligaments have been described (Earley, 1978; Earley & Dee, 1980; Earley, 1990) and are detailed below, often the most reliable form of treatment is arthrodesis of all or part of the carpus and, as a general rule, arthrodesis should always be recommended for the treatment of hyperextension injuries.

Luxation of the antebrachiocarpal joint

Conservative management

In cases with only partial rupture of a collateral ligament, or an avulsion fracture of a styloid process which is amenable to closed reduction, the application of a cast for 6–8 weeks may allow satisfactory healing and a return to normal function. However, in cases with marked instability, or when palmar ligament injury is apparent, then surgery is indicated.

Surgical treatment

Surgical treatment of antebrachiocarpal joint luxations is indicated under the following circumstances:

(1) Where *avulsion fractures* of the medial and/or lateral styloid processes are responsible for instability and the fragment is considered large enough to accommodate implants. Surgical management may involve reattachment of the fragment using a lag screw or pin and tension band wire (Fig. 34.10 in Chapter 34).

(2) If the injury involves complete rupture of the collateral ligament, early treatment may enable the two ends to be *sutured* together using an appropriate material such as absorbable suture material or monofilament nylon. If the ligament has torn from its origin or insertion it may be possible to reattach it using a bone screw and spiked washer. Postoperatively, a cast or splint should be applied for 6–8 weeks.

(3) If the instability is marked and/or conservative management has failed then it may not be possible to reconstruct the ligaments due to the degree of tearing or fibrosis. In such cases it may be necessary to place *prosthetic collateral ligaments* of braided polyester (Ethibond, Ethicon) or wire. The medial collateral ligament is injured far more frequently than the lateral collateral and many techniques have been described to replace it whereby bone tunnels or bone screws have been used to anchor the prosthesis in place. The most anatomically correct method of placing a medial collateral ligament involves either two bone tunnels (Fig. 35.9a) or one tunnel and a bone screw in the distal radius and one tunnel or a bone screw in the radial carpal bone (Fig. 35.9b). These techniques allow placement of the prosthesis so as to recreate both the long and short components of the collateral ligament.

Alternatively, the tendon of insertion of the abductor pollicis longus muscle may be transected, keeping its attachment to the radial carpal bone while the cut end is attached to the distal radius using a bone screw and spiked washer.

Whichever method is used, the carpus should be supported with a cast for 4–8 weeks postoperatively.

(4) Open luxations and shearing injuries (Butterworth, 1991) should be considered emergencies. If the joint can be thoroughly debrided within the 'golden period' of 6–8 hours (possibly extended to 12–18 hours if antibiotics are given immediately) then it may be possible to prevent the contamination from becoming established as infection. After this it might be possible to consider ligament reconstruction or prosthetic replacement. However, it is often the case that much soft tissue has been lost and collateral ligament replacement would require a great deal of foreign material to be left in a potentially infected site. Instead it may be worth considering application of a *transarticular external skeletal fixator*. This will stabilise the joint whilst soft tissue healing takes place and at the same time the fixator allows access to the wounds for daily topical treatment. The fixator frame is most easily applied to the medial aspect of the limb (Fig. 35.10). In small

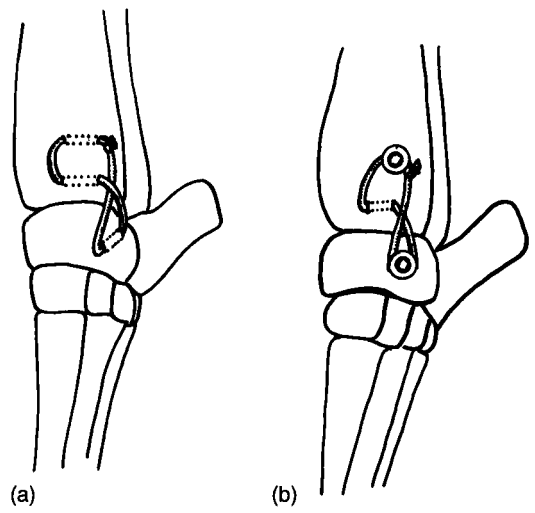

Fig. 35.9 Prosthetic replacement of the medial radiocarpal collateral ligaments. (a) Using bone tunnels, (b) using a combination of bone screws and a tunnel (shearing injuries).

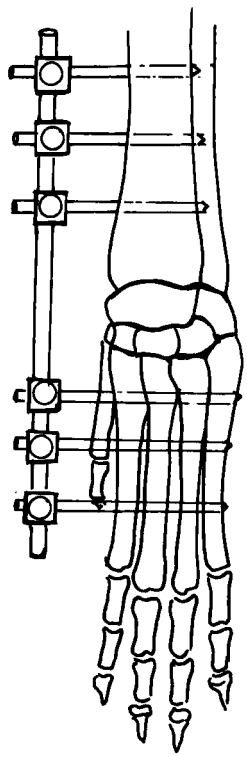

Fig. 35.10 External fixator used for stabilising open luxations of the carpus.

patients, placement of fixation pins into the metacarpi can be difficult and use of a Rudy boot may side-step such a problem. The principle behind the boot is to incorporate the distal pins into a cast applied to the manus rather than drilling them into the metacarpals (Fig. 35.11). The fixator is left in place for about 8 weeks and then, once soft tissue healing has taken place, surgery to correct any instability (as discussed above), or arthrodesis, can be considered.

(5) In cases where the techniques mentioned above have failed or where there is concurrent palmar instability or articular damage which would compromise function in the long term, it may be necessary to consider *pancarpal arthrodesis* as a salvage procedure.

Pancarpal arthrodesis

The indications and principles of arthrodesis are described in Chapter 2 (p. 22). The most common indication for pancarpal arthrodesis is a carpal hyperextension injury which results in chronic instability. The palmar ligaments are unable to heal satisfactorily following injury and hyperextension will persist if these cases are managed conservatively with a carpal splint. Surgical repair, replacement and/or reinforcement of the palmar ligaments is also generally unsuccessful and arthrodesis, either pancarpal or partial carpal, is the most satisfactory solution to the problem. The incidence and distribution of carpal hyperextension injuries is summarised in Table 35.1. Pancarpal arthodesis is the most widely used method of treatment for carpal hyperextension regardless of the level of joint involved. In a series of 45 cases described by Parker *et al.* (1981), pancarpal arthrodesis was performed using a plate applied to the dorsal aspect of the carpus for fixation. External support was provided for 6–8 weeks following surgery. In this series 74% of the dogs regained normal limb function. External support is essential to protect the plate from excessive bending forces until fusion of the carpus has occurred. Biomechanically it would be preferable to apply the plate to the palmar aspect of the carpus where it is subject to tensile forces only. The palmar approach for pancarpal arthrodesis was described by Chambers & Bjorling (1982).

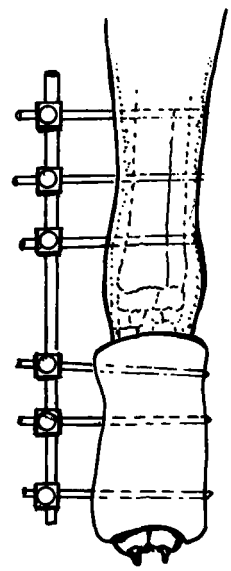

Fig. 35.11 In small dogs the distal pins can be incorporated in a cast (Rudy boot).

Table 35.1 Incidence and distribution of carpal hyperextension injuries.

Level of carpal hyperextension	Reported incidence (%)			
	Parker *et al.* (1981)	Piermattei (1990)	Earley (1990)	Denny & Barr (1991)
Antebrachiocarpal	31	10	5–10	56
Middle carpal	22	28*	65–70	10**
Carpometacarpal	47	46*	15–20	26**

* Combined middle and carpometacarpal hyperextension in 16% of cases.

** Combined middle and carpometacarpal hyperextension in 8% of cases.

The palmar approach is technically more demanding than the dorsal approach and the end results are the same, therefore the simpler dorsal approach tends to be used most often for pancarpal arthodesis.

Surgical technique

Pancarpal arthrodesis is performed using a technique similar to that described by Parker *et al.* (1981) but with some differences in positioning and approach. The lower leg is exsanguinated with an Esmarch bandage and a tourniquet is applied at the level of the elbow. Alternatively, a sterile Vetrap (3M) bandage may be used to create a bloodless field as described above under 'Exposure of the carpus' (p. 414). The dog is positioned for surgery in dorsal recumbency with the affected leg pulled caudally. A skin incision is made over the medial aspect of the carpus commencing at the distal third of the radius and ending in a mediolateral curve over the distal metacarpus. This skin flap is reflected laterally with the carpal and digital extensor tendons to expose the dorsal aspect of the carpus. The tendons of insertion of the extensor carpi radialis muscle on the proximal ends of the second and third metacarpal bones are severed and elevated to complete the exposure. The joint capsule is removed from the dorsal aspect of the carpus, the joint is flexed and articular cartilage is removed at all levels of the carpus using a high-speed burr or a small osteotome (Fig. 35.12a).

Although 3.5 mm (or occasionally 2.7 mm or 4.5 mm) dynamic compression plates (DCPs) have generally been used for fixation, there are now large, medium and small plates available specifically designed for carpal arthrodesis (Vet-

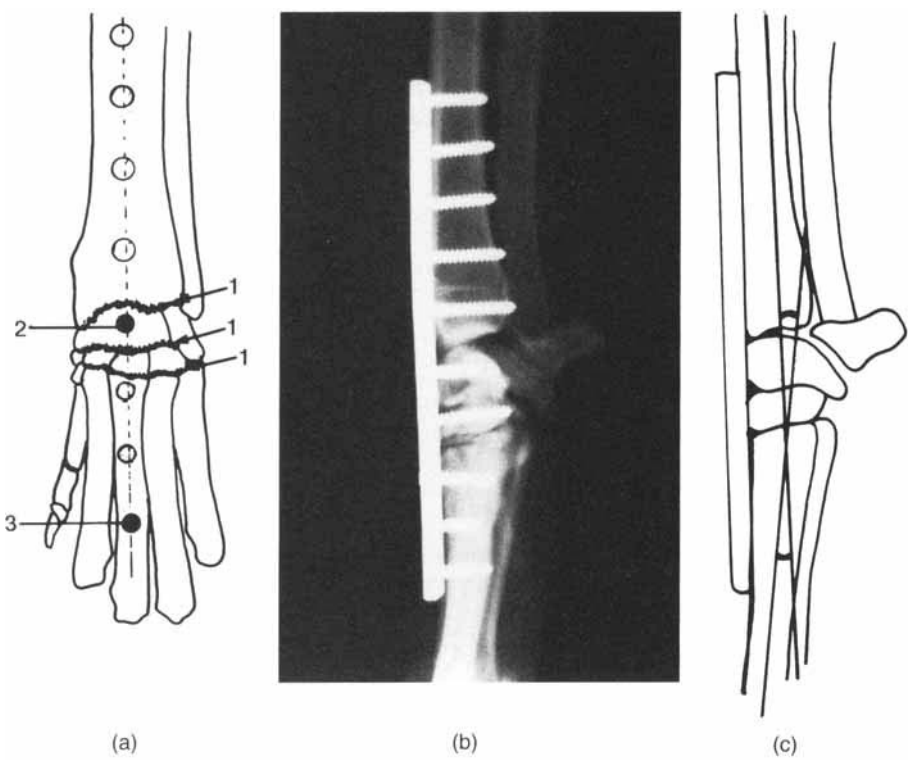

Fig. 35.12 Pancarpal arthrodesis. (a) Removal of articular cartilage (points labelled 1). Points 2 and 3 indicate positions of first two screw holes. (b) Postoperative lateral carpal radiograph. (c) Tracing of the radiograph in (b). Natural angulation of the metacarpus gives some 10° of carpal extension although a straight plate has been applied.

erinary Instrumentation). The medium-sized plate, which is used most frequently, takes 3.5 mm cortex screws in the proximal half of the plate, which is applied to the distal radius, while the distal half takes 2.7 mm cortex screws which are placed in the radial carpal bone and the third metacarpal bone. The distal end of the plate is tapered to fit the third metacarpal bone and also to produce 10° of carpal hyperextension (see below). The larger carpal arthrodesis plate takes 4.5 mm cortex screws proximally and 2.7 mm cortex screws distally, while the small plate takes 2.7 mm cortex screws proximally and 2 mm cortex screws distally.

Although it has been recommended that the carpus is fused in a normal standing position with 10° of extension (Parker *et al.*, 1981), in most cases little or no contouring of the plate is necessary because of the natural angulation of the metacarpus (Fig. 35.12b,c) and, if the special carpal arthrodesis plates are used, the tapered end ensures that 10° of hyperextension is achieved.

The plate is applied to the dorsal surface of the radius, radial carpal bone and third metacarpus with a minimum of seven screws; one placed in the radial carpal bone, three in the third metacarpal bone and at least three in the distal radius. The screw hole in the radial carpal bone is prepared first, followed by the distal screw hole in the third metacarpal bone (Fig. 35.12a). A cancellous bone graft is collected from the proximal humerus (as described in Chapter 2, p. 19) and packed into the carpal joint spaces. The graft is held in place by the plate using two screws at the prepared sites and a third screw in the distal radius. The remainder of the screws are placed in routine fashion. Following wound closure, the lower leg is bandaged, the tourniquet is released and the carpus is supported with a gutter splint for 6 weeks following surgery.

Results

The results of treatment by pancarpal arthrodesis in a series of 40 dogs was published by Denny & Barr (1991). Forty-three pancarpal arthrodeses were carried out as three dogs had bilateral arthrodeses. Seventy-four per cent of the dogs regained full limb function. The most common complication of pancarpal arthrodesis was loosening of one or more of the distal screws. This was invariably associated with lameness which rapidly resolved once the loose implant had been removed. Other recognised complications of the procedure include fracture of the third metacarpal bone at a later date (6/64 arthrodeses reported by Whitelock *et al.*, 1999). Following such fractures, some cases will show resolution of the associated lameness with conservative management, whilst in others removal of some or all of the implants will be required.

Plate removal was necessary in 8/43 arthrodeses reported by Denny & Barr (1991) and 11/64 reported by Whitelock & Houlton (1998). Soft tissue reaction over the plate as a result of low-grade infection and or implant loosening was the main indication for plate removal. A gutter splint should be used to support the carpus for 4–6 weeks after plate removal to minimise the risk of fracture at one of the levels of carpal arthrodesis. If there is any doubt about the completeness of the carpal fusion at the time of plate removal a further cancellous bone graft should be taken from the proximal humerus and packed over the dorsal aspect of the carpus after removal of the plate.

Carpal arthrodesis in the cat

The indications for carpal arthrodesis in the cat are the same as the dog. Crossed Kirschner wires (Fig. 35.13) can be used for fixation in most cases, but additional support should be provided with a gutter splint on the palmar aspect of the carpus for 6 weeks. The carpus should be fixed in about 10° of extension. Carpal arthrodesis is said to be useful in cats with low radial nerve paresis (M.R. Herron, 1990, pers. comm.). Here the carpus should be fixed with 20–30° of hyperextension to minimise trauma to the toes. If the carpus is to be hyperextended to this degree then the crossed Kirschner wire technique is no longer possible and under these circumstances an AO Veterinary Cuttable Plate (VCP, Stratec) is used with 1.5 mm or 2 mm diameter cortical bone screws. External support should be provided for 6 weeks following surgery.

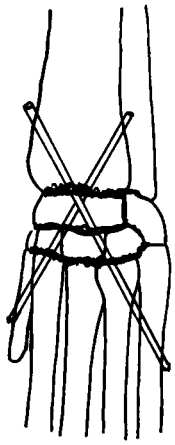

Fig. 35.13 Pancarpal arthrodesis using crossed Kirschner wires.

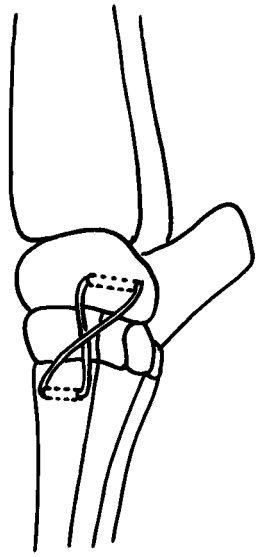

Fig. 35.14 Prosthetic replacement of the medial collateral support for the middle carpal joint and/or the carpometacarpal joint using a non-absorbable prosthesis placed through bone tunnels.

Luxation of the middle carpal joint

Conservative management

The application of external support for 4–8 weeks may allow healing of collateral ligament ruptures. It may also lead to a successful outcome in small dogs with a degree of palmar instability because it allows spontaneous ankylosis. However, if palmar ligament support is lacking, especially in medium- or large-breed dogs, then such management may fail, with the development of a palmigrade stance once the cast has been removed.

Surgical treatment

Surgery is indicated:

(1) In cases where a collateral ligament, most commonly the medial collateral, has been ruptured it may be possible to repair the soft tissue injury or, failing that, to replace it with a prosthetic ligament of braided polyester (Ethibond, Ethicon) or wire placed through tunnels drilled in the head of the appropriate metacarpal bone and through a tunnel (Fig. 35.14) or around a bone screw placed into the radial carpal bone. On the medial side it might be possible to use the tendon of the abductor pollicis longus muscle as a prosthetic ligament by maintaining its attachment to the radial carpal bone and securing it to the head of metacarpal 2 (Fig. 35.15). External support should be used for 4–6 weeks postoperatively.

(2) Where there is gross instability associated with palmar ligament injury, +/– concurrent collateral damage, then attempts to reconstruct the ligamentar support are likely to fail and a partial or pancarpal arthrodesis is the most appropriate form of management (see under 'Luxation of the carpometacarpal joint' below).

Luxation of the radial carpal bone

Luxation of the radial carpal bone is an uncommon injury in the dog (Pillet, 1957; Punzet, 1974; Earley & Dee, 1980; Vaughan, 1985; Miller *et al.*, 1990). The condition has also been described in a cat (Pitcher, 1996). The radial carpal bone pivots through 90° medially and in a dorsopalmar direction, making closed reduction difficult. Even if it can be achieved, surgery may still prove necessary due to damage to the medial collateral ligament.

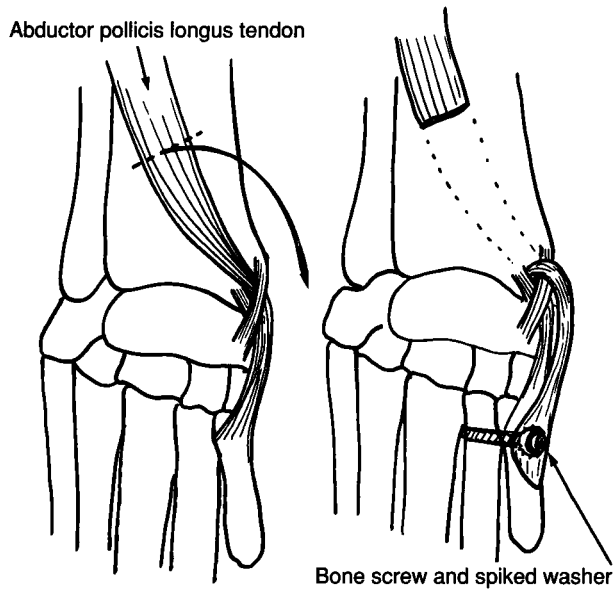

Fig. 35.15 Prosthetic replacement of the medial collateral support for the middle carpal joint and/or the carpometacarpal joint using a section of the abductor pollicis longus tendon which is reflected distally and secured to the second metacarpal bone.

Therefore, except in small breeds of dog, surgery is almost always justified.

At surgery the bone may be relocated after it has been derotated and it may be secured in position by placement of a Kirschner wire, or bone screw, through the radial carpal bone and into the ulnar carpal bone (Fig. 35.8b). Reconstruction of the medial collateral ligament may then be necessary but if this is not possible a prosthetic ligament may need to be created using the implant in the radial carpal bone as one of the anchor points (Fig. 35.9). Postoperatively, the joint is supported in a splint or cast for 6–8 weeks whilst soft tissue healing takes place.

Luxation of the carpometacarpal joint

Conservative management

The application of an external support for 4–8 weeks may allow healing of ligament ruptures or avulsion fractures. It may also lead to a successful outcome in small dogs with a degree of palmar instability because it may allow spontaneous ankylosis. However, if palmar ligament support is lacking, especially in medium or large breeds of dogs, then such management may fail, with the development of a palmigrade stance once the cast has been removed.

Surgical treatment

Surgery is indicated:

(1) In cases where a collateral ligament, most commonly the medial collateral ligament, has been ruptured it may be possible to repair the soft tisue injury or, failing that, to replace it with a prosthetic ligament of braided polyester (Ethibond, Ethicon) or wire placed through tunnels drilled through the head of the appropriate metacarpal bone and through a bone tunnel or around a bone screw in the proximal row of carpal bones (Fig. 35.14). On the medial aspect it may be possible to use the tendon of the abductor pollicis longus muscle as a prosthetic ligament by maintaining its attachment to the radial carpal bone and securing it to the head of metacarpal 2 (Fig. 35.15). External support should be provided for 4–6 weeks postoperatively.

(2) Where the instability is associated with an avulsion fracture of a collateral ligament's insertion on the relevant metacarpal bone,

it may be possible to reduce the fracture and stabilise it using bone screws. External support should be provided for 4–6 weeks postoperatively.

(3) Where there is gross instability associated with palmar ligament injury, +/– concurrent collateral damage, attempts to reconstruct the ligamentar support are likely to fail and a partial carpal (or pancarpal) arthrodesis is the most appropriate form of management (see prognosis section below discussing the choice between partial and pancarpal arthrodesis).

Partial carpal arthodesis – surgical technique

Preparation of the patient and provision of a bloodless field is exactly the same as described under 'Surgical technique' for 'Pancarpal arthrodesis' above (p. 418). A dorsal approach is made to the carpus and articular cartilage is removed from the middle carpal and carpometacarpal joints. Cancellous autogenous bone graft is taken from the proximal humerus and packed into the joint spaces. Fixation techniques include:

- 'T'-plate fixation (Earley, 1981; Brinker *et al.*, 1983; Smith & Spagnola, 1991) (Fig. 35.16a)
- Retrograde insertion of intramedullary pins up the middle two metacarpi and into the radial carpal bone (Slocum & Devine, 1982; Brinker *et al.*, 1983; Willer *et al.*, 1990) (Fig. 35.16b)

The plate is applied to the dorsal aspect with two screws placed in the radial carpal bone and three or four in the third metacarpal bone (Fig. 35.16a). In order to expose the latter, one part of the extensor carpi radialis muscle's tendon of insertion has to be elevated. Since this muscle is required to function postoperatively, it is necessary to suture the elevated tendon to that inserting on the second metacarpus.

However the joints are stabilised, it is important to utilise external support until there is radiographic evidence of fusion, which is usually in a matter of 6–10 weeks depending on the age of the dog. The implants are generally left *in situ* although there may be a need to remove plates

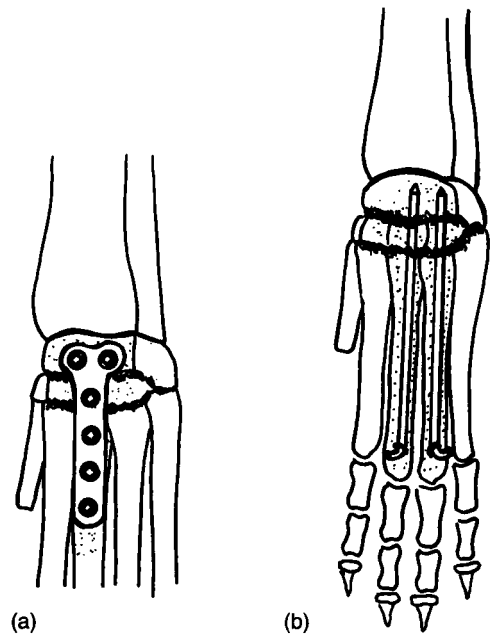

Fig. 35.16 Partial carpal arthrodesis. (a) Using a 'T'-plate. (b) Using intramedullary pins.

owing to local irritation due to lack of soft tissue cover in this area and the close proximity of their proximal border to the radiocarpal joint.

Prognosis following treatment of carpal luxations

Following the repair of collateral ligaments or their prosthetic replacement, many cases will return to very satisfactory, if not normal, function. Unsuccessful outcomes are most often seen as a result of not fully recognising the extent of ligamentar damage, leading to subsequent instability and osteoarthritis. Most often this occurs when the antebrachiocarpal joint is considered to have been spared from injury and a partial carpal arthrodesis is performed. Unrecognised injury to the 'spared' joint then leads to osteoarthritis or palmar collapse and further surgery may become necessary. *It has been noted that pancarpal arthrodesis does not affect gait significantly and if there is any doubt as to whether partial or pancarpal arthrodesis is most appropriate, it is always safer to perform the latter.* In addition, it has been

noted that when T-plates are used in partial carpal arthrodeses, they may be associated with a higher incidence of osteoarthritis in the antebrachiocarpal joint than when pins are used. For this reason it has been recommended that use of the latter may be preferable. In a series of 39 dogs with carpal hyperextension injuries treated by carpal arthrodesis using intramedullary pins for fixation, 70% regained full limb function while 12% had slight lameness after exercise (Willer *et al.*, 1990). T-plate fixation is best reserved for the larger dogs where the radial carpal bone is large enough to allow positioning of the plate without encroaching on the radiocarpal articulation.

Pancarpal arthrodesis carries a good prognosis with 74% of cases regaining full limb function (Parker *et al.*, 1981; Denny & Barr, 1991).

Acquired conditions

Osteoarthritis

Carpal osteoarthritis is a common secondary sequel to carpal injury, particularly if instability has resulted from the injury. The clinical features and changes associated with osteoarthritis are described in Chapter 7 (p. 56). If carpal pain associated with osteoarthritis fails to respond to medical management then arthrodesis is often the most satisfactory way of dealing with these cases. Postoperative splinting after such a procedure is not required to protect the plate (as described under 'Pancarpal arthrodesis' above) as the palmar ligament support is intact.

Immune-mediated arthropathy

The carpus tends to be one of the first joints in which an immune-mediated arthropathy may be noticed because joint swelling or instability is obvious. For further details refer to Chapter 8.

Neoplasia

The most common tumour diagnosed in the proximity of the carpus is *osteosarcoma* affecting the distal radius. The distal ulna is also an occasional site for the development of osteosarcoma. *Synovial sarcoma* is the most common primary joint tumour (for further details see Chapters 10 and 51). Benign lesions are uncommon but include *bone cysts*, which develop in the metaphyseal region of the distal radius (see Chapter 51, p. 624) and *synovial osteochondromatosis*, a condition in which numerous foci of cartilage develop in the synovial membrane of a joint (see Chapter 10).

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Chapter 36
The Manus

The manus consists of the metacarpal bones and the digits (Fig. 36.1). Conditions of the manus will be considered under the following headings:

- *Traumatic*
  - Fractures of the metacarpals
  - Fractures of the phalanges
  - Luxation of the metacarpophalangeal joint
  - Luxation of the proximal interphalangeal joint
  - Luxation of the distal interphalangeal joint
  - Dropped toe
  - Knocked up toe
  - Fracture of the proximal, palmar metacarpophalangeal sesamoids
  - Section of the digital flexor tendons
- *Developmental*
  - Sesamoid disease
- *Other foot problems*
  - Pad injuries
  - Split webs
  - Nail injuries
  - Tracking foreign bodies

Traumatic conditions

Fractures of the metacarpal bones

Metacarpal fractures are common. Fractures of the head of metacarpals II or V result in carpometacarpal joint instability and the management of this problem is described in Chapter 35 (p. 421). Shaft fractures of the metacarpal bones can be managed by external coaptation with a cast or splint if only one or two of the metacarpal bones are fractured as the remaining, intact, bones tend to act as internal splints. If all four metacarpals are fractured then internal fixation using pins or Kirchner wires is appropriate, with the two axial metacarpi +/– the abaxial metacarpi being stabilised (Whittick, 1974) (Fig. 36.2). Alternative methods of stabilisation include lag screw or plate fixation. The latter is used most often in the racing Greyhound. Small plates such as the AO Veterinary Cuttable Plate (Stratec) have proved particularly useful (Dee, 1991) in the racing Greyhound. The incidence and treatment of metacarpal fractures have been reviewed by Muir & Norris (1997).

Fractures of the phalanges

Fractures of the phalanges occur in the racing Greyhound due to rapid or incoordinate turns at speed placing torsional stress on the phalanx. In other breeds, the fracture is more commonly caused by a crush injury. External coaptation using a cast is a satisfactory form of treatment for most dogs with the exception of the racing Greyhound. If the Greyhound is to regain its form, then such fractures are best treated by open reduction and fixation with a lag screw(s) or wire sutures. If lameness persists after the fracture has healed, performance may improve following amputation of the distal phalanx to relieve pressure on the fracture site (see below).

Luxation of the metacarpophalangeal joint

Luxation is most common in the racing Greyhound but can affect any breed of dog. The injury occurs during exercise and on examination of the injured foot gross displacement of the metacarpophalangeal joint may be obvious, especially if

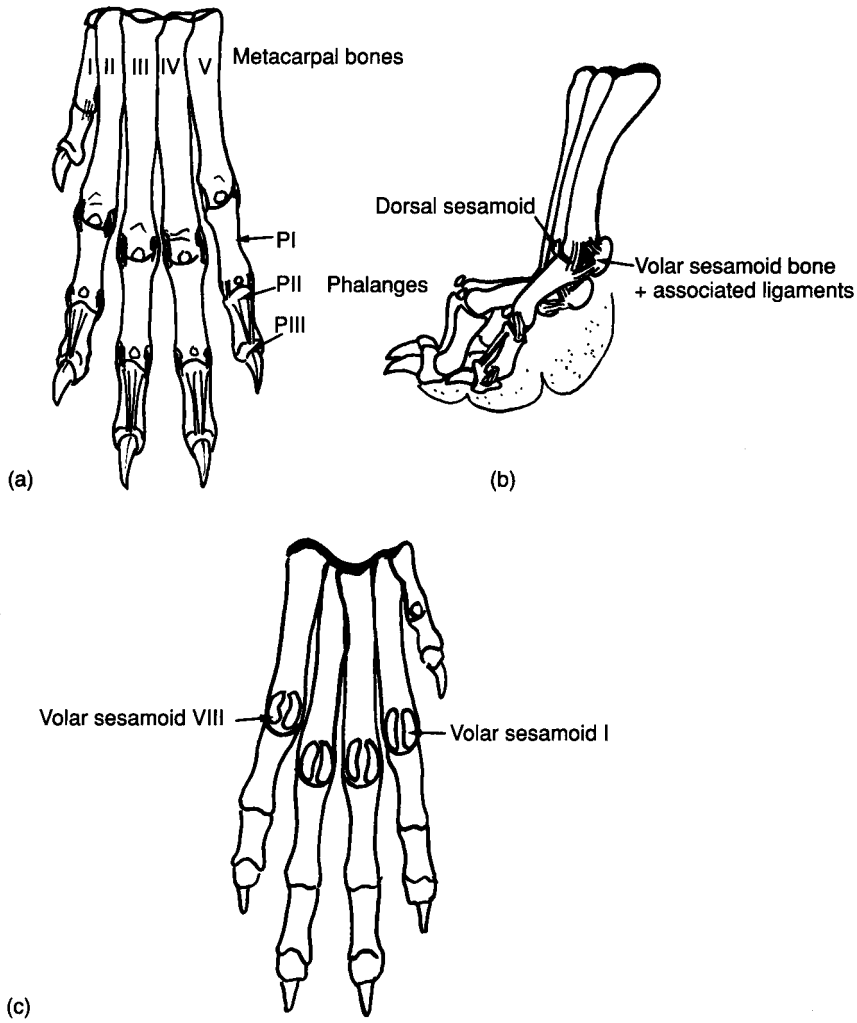

Fig. 36.1 Anatomy of the left manus. (a) Dorsal view, (b) lateral view and (c) volar view.

the axial collateral ligament has ruptured. Closed reduction is easily achieved and, in the pet animal, a support bandage or light cast applied for 2–3 weeks is the only treatment required, with a good prognosis for return to normal activity. In the racing Greyhound the aim of treatment should be primary surgical repair of the damaged collateral ligaments. However, because the injury is often complicated by involvement of the sesamoidean ligaments, sesamoid luxation and/or damage to articular cartilage, many treated dogs fail to return to racing and, as a result, amputation of the digit just proximal to the joint is often used as a salvage procedure.

Luxation of the interphalangeal joints ('sprung toe')

Interphalangeal joint luxation is a common injury in the racing Greyhound (Davies, 1958; Bateman, 1960; Hickman, 1975). The digits of the left manus, particularly digits II and V, are affected most fequently.

Fig. 36.2 Kirschner wires used to stabilise metacarpal fractures.

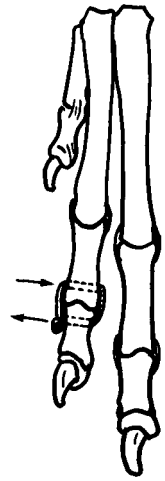

Fig. 36.3 Stabilisation of a proximal interphalangeal joint luxation using a wire suture passed through bone tunnels.

Luxation of the proximal interphalangeal joint

Luxation is associated with collateral ligament injury and disruption of the joint capsule. Surgical repair of the collateral ligament is achieved using two or three sutures of 4/0 polydioxanone (PDS, Ethicon) (Eaton-Wells, 1994). Following wound closure, the toenail should be cut short to prevent mechanical leverage on the proximal interphalangeal joint during the healing phase. In cases with severe instability, a wire suture can be placed through tunnels just proximal and distal to the joint (Fig. 36.3). This technique does reduce the range of joint motion, however, and there has been a trend towards treating severe interphalangeal joint luxations by arthrodesis (Dee *et al.*, 1990). Arthrodesis is achieved by removal of the articular cartilage (Fig. 36.4a) followed by fixation with a Kirschner wire and tension band wire (Fig. 36.4b) or by application of a plate (Fig. 36.4c). The nail is also removed to relieve stress on the joint.

If lameness persists, despite all these measures, then amputation of the third phalanx is carried out.

Luxation of the distal interphalangeal joint

The collateral ligament is repaired as described above for proximal interphalangeal luxations. The nail is amputated proximal to the ungual crest to reduce mechanical leverage on the repair as the dog weight-bears. To achieve this the nail is split longitudinally using bone or nail cutters, the two halves of the nail are pulled out exposing the bone of the ungual process which is then cut proximal to the ungual crest.

'Dropped' toe

Avulsion of the superficial digital flexor tendon from its attachment to the first phalanx results in a flat or 'dropped' toe. This is of little clinical significance.

'Knocked-up' toe

When the deep digital flexor tendon is avulsed from its extensor process on the third phalanx, or the tendon is severed, then the end of the toe tilts

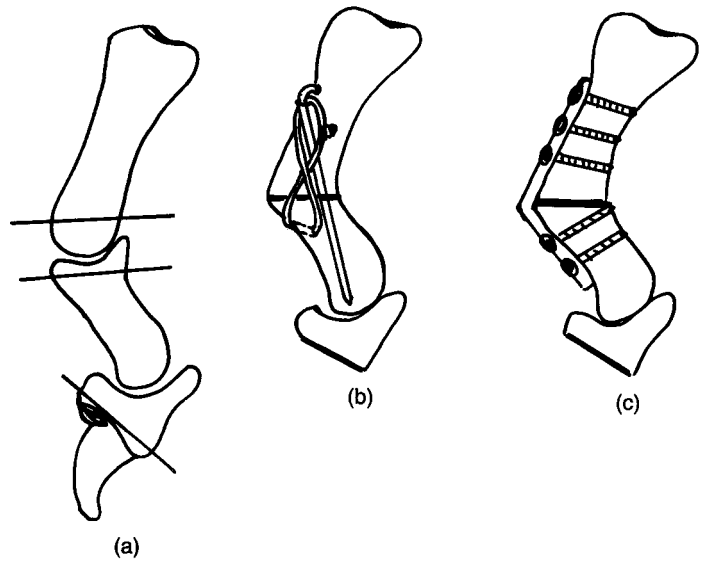

Fig. 36.4 Arthrodesis of interphalangeal joint. (a) Removal of articular surfaces and amputation of nail. (b) Arthrodesis with Kirschner wire and tension band wire. (c) Arthrodesis with a plate. The joint should be fused at an angle of 60°.

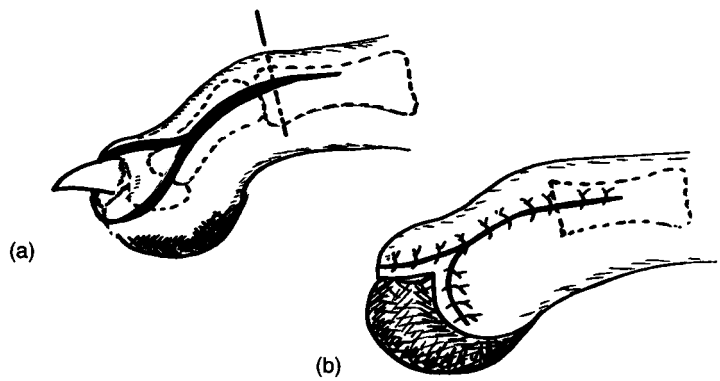

Fig. 36.5 Toe amputation. (a) Skin incision, (b) wound closure.

up and is referred to as a 'knocked-up' toe. This is not of clinical significance.

Amputation of the toe

The principles of amputation of the toe, whether it be through the metacarpophalangeal joint, the proximal interphalangeal joint or the distal interphalangeal joint, are the same (Fig. 36.5). A dorsal skin incision is made over the toe. Distally the incision encircles the nail. The digital pad is preserved in amputations through the proximal and distal interphalangeal joints but not in metacarpophalangeal amputations. The phalanges are 'filleted' out, preserving as much soft tissue as possible. Once the soft tissues have been dissected away from the bone, the toe is disarticulated at the required level. The condyles at the distal end of the remaining phalanx or metacarpal bone are removed with rongeurs. When the pad is preserved, skin sutures are placed to form an inverted Y-shaped incision which pulls the pad over the bone of the amputation stump. As with all surgery of the carpus and manus, use of a tourniquet expedites the procedure.

Sesamoid fractures

Fractures of the palmar metacarpophalangeal sesamoid bones are seen mainly in the racing

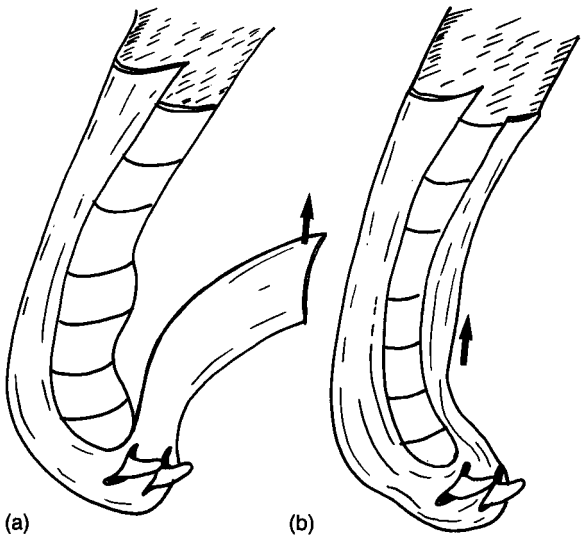

Fig. 36.6 Flexion bandage. (a) Elastoplast (Smith & Nephew) strip is used to keep toes in flexion. (b) Flexion bandage complete except for the final layer of Elastoplast (Smith & Nephew) which is applied in a wrap-around fashion.

Greyhound and usually involve sesamoids II or VII. For details of the clinical signs and treatment of this condition see 'Sesamoid disease (and fractures)' later in this chapter.

Section of the digital flexor tendons

Such injury is most frequently caused by the dog stepping on broken glass or sharp metal. The wound occurs between the digital pads on the caudal aspect of the metacarpus. There is profuse haemorrhage resulting in a natural tendency to control this with sutures and bandage while the important tendon injury remains unidentified. Section of the superficial digital tendon is of little significance, but if the deep digital flexor tendon is cut then flattening of one or more of the digits occurs. The severed tendon should be repaired as described in Chapter 3 (p. 26) and, postoperatively, a cast or flexion bandage is applied for 3–4 weeks so as to maintain the foot in a semi-flexed position and ease tension on the sutures while healing takes place.

Application of a flexion bandage

This is illustrated in Fig. 36.6. A soft conforming bandage, for example Soffban (Smith & Nephew), is applied first. Next, a layer of Elastoplast (Smith & Nephew) is applied leaving the central toes exposed. A strip of Elastoplast (Smith & Nephew) is taken, in which two holes have been cut which will form anchor points around the nails of digits III and IV. This adhesive strip is used to bend the foot into the flexed position so that tension is eased on the digital flexor tendon repair. A final layer of Elastoplast (Smith & Nephew) is applied to complete the bandage.

Developmental conditions

Sesamoid disease (and fractures)

The palmar metacarpophalangeal and plantar metatarsophalangeal (volar) sesamoid bones lie within the superficial digital flexor tendons of insertion. There are two sesamoids (axial and abaxial) relating to each digit and conventionally they are numbered from 1 to 8 starting medially (i.e. the abaxial sesamoid of digit II is numbered 1 and the abaxial sesamoid of digit V is numbered 8). They may be the cause of lameness as a result of abnormal development or fracture.

Aetiopathogenesis

Fractures of the volar sesamoids are usually seen in racing Greyhounds and numbers II or VII are

the most often affected. These two sesamoids have a different shape when compared to the other six. They have a groove to accommodate the deep digital flexor tendon whose path is more 'off centre' in digits II and V compared to III and IV. The forces generated by the more acute change in direction of the deep digital flexor tendon, together with the altered anatomy of these two sesamoids, may explain why these are fractured more often than the others.

Abnormal development of the volar sesamoids may be a form of osteochondrosis and results in deformation of, and irregular mineralisation within, the affected sesamoid. Exostoses within the soft tissue attachments may also develop. These attachments are:

- The axial and abaxial sesamoidean ligaments – from the respective sesamoids to the distal metacarpal (metatarsal) and to the proximal phalanx
- The intersesamoidean ligament
- The cruciate ligaments of the sesamoid bones – attached to the proximal phalanx

Such abnormal development is commonplace in certain breeds such as Rottweilers without necessarily being of clinical significance. In one survey (Vaughan & France, 1986) 44% of Rottweilers showed radiographic changes consistent with sesamoid disease without any having an associated clinical lameness. Sesamoids II and VII are most often affected, probably for the same reasons mentioned under fractures.

Pain results from movement of the abnormal sesamoid articular surface or else a 'sprain'-type injury of the abnormal sesamoid or its soft tissue attachments.

History

In the case of fracture, the dog will become lame at the end of a race or, in the uncommon event of a pet dog being affected, during a period of exercise. The developmental abnormalities will, if they cause any clinical signs, result in lameness of insidious onset in skeletally immature animals. The breeds affected are virtually all medium-sized with the Rottweiler and Labrador Retriever being reported most often. The degree of lameness is usually made worse by exercise and may be quite marked for the degree of pain found on examination. Because the problem lies within the foot, the severity of the lameness may vary according to the surface on which the dog is walking.

Examination

The lameness will be associated with swelling of the affected metacarpo(metatarso)phalangeal joint. Manipulation of the joint should be painful and there may be evidence of a synovial effusion or periarticular thickening. Restriction in the range of motion will be seen but this may also be present in long-standing and clinically insignificant sesamoid pathology. In examining immature patients it is important to remember that lameness in these breeds is more commonly caused by other problems, e.g. elbow osteochondrosis or hip dysplasia. The injection of local anaesthetic into the sesamoid may be a way of confirming this as the cause of the lameness but there are logistical problems in administering such an injection (which would usually require heavy sedation or anaesthesia) and then evaluating the effect in a fully conscious dog before the anaesthetic wears off.

Radiology

Dorsopalmar (plantar) views of the manus (pes) will show the sesamoids most clearly and mediolateral views will add little, if any, useful information. Taking the dorsopalmar view of the contralateral manus (pes) is useful for making comparisons, especially in acute cases. Radiography will establish the presence of any slab, mid-body, sagittal or chip fractures in acute cases. Abnormal development of the sesamoids is recognised radiographically as deformity of outline, irregularity of mineralisation or enthesiophyte formation. In long-standing cases, periarticular osteophyte formation may become evident. It is not uncommon to find more than one sesamoid affected radiographically in the lame foot and also similar changes in the contralateral foot.

It must be reiterated that such developmental abnormalities are often totally asymptomatic and, as always, radiographic changes must be used to confirm the clinically suspected diagnosis rather than to make a diagnosis.

Treatment

Conservative management Resting the dog for 4–6 weeks (+/– support bandaging for the first 2–4 weeks) is an appropriate means of management in pets with fractures or immature dogs with developmental abnormalities. In some cases that are responding poorly, there may be an indication for the local administration of methylprednisolone (Depo-Medrone, Pharmacia & Upjohn). In the authors' experience, an injection of 10–20mg of this preparation into an affected sesamoid may lead to improvement in cases that are showing a poor response to rest alone.

Surgical treatment (Bennett & Kelly, 1985) Excision of the affected sesamoid is appropriate in racing Greyhounds with fractured sesamoids since it gives a more predictable outcome in terms of speed of return to training after a 4- to 6-week postoperative period of convalescence. It is also appropriate in other cases that are non-responsive to the conservative management outlined above. The bone is removed by careful dissection whilst retaining the integrity of the superficial digital flexor (SDF) tendon. Sesamoids IV and V are more difficult to remove because of the presence of the metacarpal pad. Removal of one sesamoid will have no effect on a digit. Removal of both sesamoids will not affect function but is more likely to create flattening of the digit due to stretching of the SDF tendon of insertion.

Prognosis

The prognosis is generally good. Most racing dogs will return to form after removal of the fractured sesamoid. Most pets will respond to conservative management after fracture or abnormal development of a sesamoid and in those cases that do not, surgical removal will lead to resolution of the lameness in almost all cases.

Other foot problems

Although the foot problems described below tend to be seen most often in racing Greyhounds, they can affect any dog.

Pad injuries

Cut pads are caused by glass, metal or sharp stones. It is important to check for, and remove, any foreign body from the wound. Deep, penetrating cuts can sever the digital flexor tendon at the level of the third phalanx resulting in upward tilting of the nail. Most cut pads will heal by second intention but with deep cuts, healing time may be reduced by placement of vertical mattress sutures, or skin staples, to draw the edges together.

Split webs

Wounds involving the skinfold between two toes should be treated by careful suturing. If left unrepaired they result in a split web with loss of support for the toe (Fig. 36.7) which may cause some lameness.

Nail injuries

These include:

- *Split nail.* A longitudinal split in the horny nail extending proximally from the tip right up under the coronary band. Most of these splits occur during racing (Kidd, 1983).
- *Pulled nail.* The dew claws of the forefeet (or

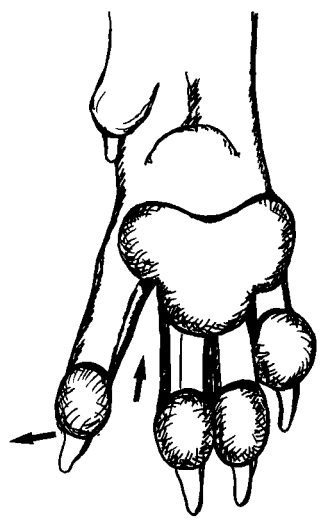

Fig. 36.7 Split web, resulting in loss of support for the toe.

the nails of the fifth digit in the hindlimb) are affected most often. The nail is stripped completely, exposing the sensitive lamina. This is a common racing injury.

- *Broken nails.* The nail and ungual process are fractured transversely (Kidd, 1983).
- *Bruised nail.* This is a partial separation of the nail from the nail bed which causes lameness and severe pain on palpation (Needham, 1978).
- *Deformed nail.* The nail grows at an abnormal angle as a result of some previous injury and may dig into an adjacent toe.

Amputation of the nail is used to treat these nail injuries.

Tracking foreign bodies

Grass seeds or splinters of wood which penetrate the skin between the toes cause swelling, pain and sinus formation. Most of these foreign bodies can be readily removed using crocodile forceps introduced up the sinus tract. However, in some cases the grass seed is not found and will continue to track proximally up the leg. Most of these will come to lie close to the palmar, or occasionally the dorsal, aspect of the metacarpus (metatarsus). There will be persistent lameness, soft tissue swelling, pain and sinus formation. Radiographic examination may show periosteal new bone formation on one or more of the metacarpal bones. There is often a clinical improvement following treatment with antibiotics but lameness returns once treatment is stopped and will not resolve until the foreign body is removed. It is essential to have a bloodless field to find the foreign body. This is achieved using an Esmarch bandage and a tourniquet applied proximal to the carpus (tarsus). A Vetrap bandage (3M) can be sterilised and used to exsanguinate the foot and create a tourniquet (as described in Chapter 35, p. 414). A longitudinal skin incision is made along the length of the metacarpus on the side closest to the area of sinus formation. Periosteal elevation of the interosseous muscles is carried out to allow exposure of the palmar aspect of the metacarpal bones. The foreign body (grass seed, piece of wood, etc.) will usually be found lying close to the bone and is removed.

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Chapter 37
Forelimb Amputation

The indications for amputation of the forelimb include:

- Gross trauma
- Gangrene
- Paralysis
- Osteomyelitis
- Neoplasia

The two basic techniques for amputation of the forelimb are:

(1) Amputation of the limb through the proximal third of the humerus (Hickman, 1964)
(2) Complete forequarter amputation with removal of the scapula (Harvey, 1974)

Forequarter amputation tends to be more popular for cosmetic reasons and also allows a wider margin of excision when the procedure is being used to manage neoplasms involving the proximal humerus.

Amputation through the proximal third of the humerus

A semicircular skin incision is made on the lateral aspect of the limb (Fig. 37.1a). The leg is lifted by an assistant and a similar incision is made on the medial side of the limb. The medial skin flap is reflected and the brachial artery and vein identified between the triceps and the biceps brachii muscles (Fig. 37.1b). Both vessels are ligated and severed. Early ligation of these vessels reduces the amount of haemorrhage during the rest of the operation.

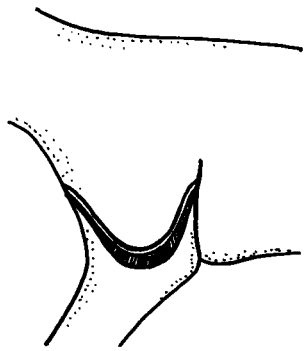
The limb is lowered, the lateral skin flap is elevated and the cephalic vein is ligated (Fig. 37.1c). The common tendon of insertion of the triceps is severed and the muscle mass reflected proximally to expose the brachialis muscle and radial nerve (Fig. 37.1d). The nerve and brachialis muscle are severed. The brachiocephalicus and biceps brachii muscles are also severed and reflected to complete the exposure of the distal humerus. The muscles are then bluntly pushed back from the shaft with a swab until the proximal third of the bone is exposed. Amputation is completed by sawing through the bone at this level (Fig. 37.1e).

Dead space between the muscle bellies is closed with a series of purse-string sutures started close to the cut ends of the bone and working towards the severed ends of the muscles (Fig. 37.1f). Finally, the ends of the muscles are sutured together with horizontal mattress sutures and the skin flaps closed in a similar manner.

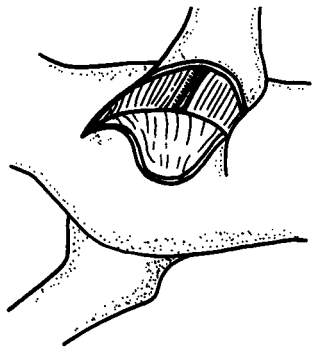
Complete forequarter amputation

An inverted, Y-shaped incision is made starting from the proximal end of the scapular spine, descending over the scapula and proximal humerus and then encircling the leg at the mid-humerus level (Fig. 37.2a). The superficial veins on the lateral aspect of the leg are ligated. These include the axillobrachial, the omobrachial and the cephalic veins (Fig. 37.2b). Cranially, the brachiocephalicus muscle is transected through its tendinous portion and the omotransversarius and trapezius muscles are severed and reflected from the edge of the scapular spine (Fig. 37.2b). Caudally, the latissimus dorsi muscle is severed close to its humeral attachment (Fig. 37.2c).

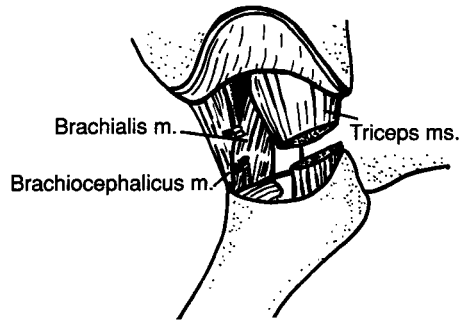
Next, the cranial edge of the scapula is rotated laterally to expose the insertions of the



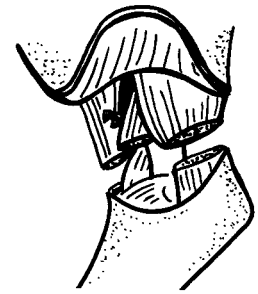
(a) Lateral skin incision.



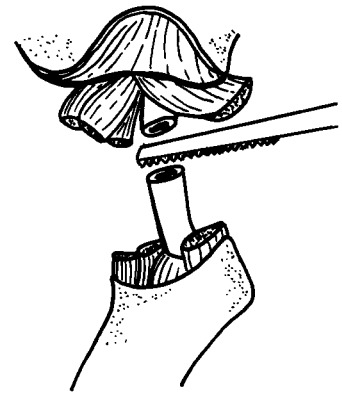
(b) Medial skin incision. The brachial artery and vein are ligated.



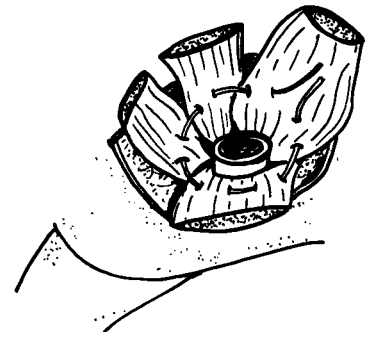
(c) The cephalic vein is ligated, the triceps muscle is severed and the brachialis muscle, radial nerve, brachiocephalis muscle and biceps brachii muscles exposed.



(d) The brachialis muscle, radial nerve, brachiocephalis muscle and biceps brachii muscles are severed.

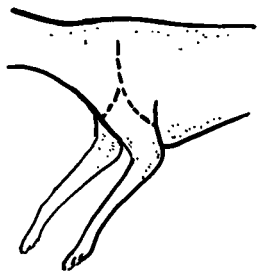


(e) Amputation in completed by cutting through the proximal third of the humerus with a saw.

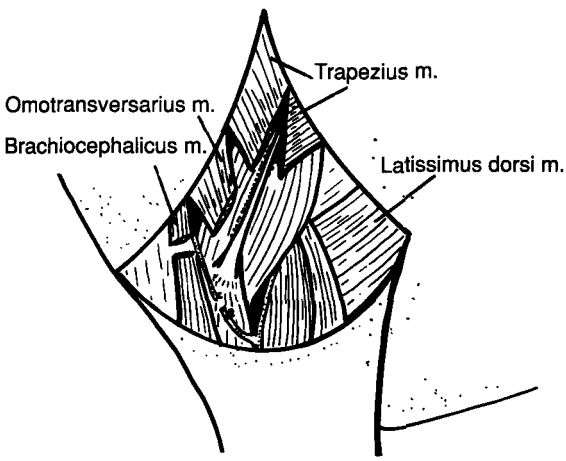


(f) Wound closure using purse-string sutures to close the dead space.

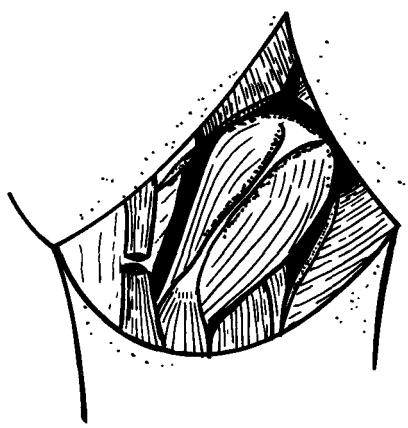
Fig. 37.1 Amputation of the forelimb through the proximal third of the humerus.



(a) Skin incision.

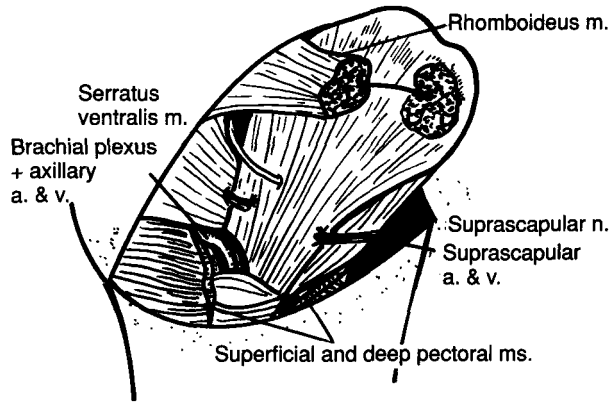


(b) Superficial lateral veins ligated. Lateral muscle attachments severed.

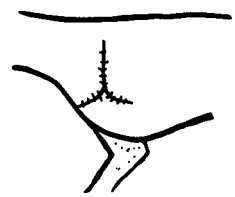


(c) Scapula freed from lateral muscle attachments.

Fig. 37.2 Complete forequarter amputation.



(d) Lateral rotation of scapula to allow section of medial muscle attachments, nerves and blood vessels. See text.



(e) Wound closure.

rhomboideus and serratus ventralis muscles. Both muscles are cut through their insertions (Fig. 37.2d). The suprascapular nerve is severed and the suprascapular artery and vein are ligated and cut. Both the superficial and deep pectoral muscles are cut close to their humeral insertions. The nerves of the brachial plexus are severed and the axillary artery, followed by the axillary vein, is ligated and cut, completing the amputation.

The cut ends of the muscle bellies are turned in and the lateral fascial sheaths of the latissimus dorsi, trapezius and omotransversarius muscles are sutured to the lateral fascial sheaths of the pectoral muscle. The rest of the wound closure is routine (Fig. 37.2e).

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Section 6
The Hindlimb

Chapter 38
Examination and Differential Diagnosis of Hindlimb Lameness

The examination of animals with lameness has been described in Chapter 6 (p. 41) and the conditions affecting each joint or bone of the hindlimb are listed early in the relevant chapters of this section. Table 38.1 outlines the differential diagnosis of hindlimb lameness according to whether the patient is skeletally immature or mature and the onset of lameness is acute or insidious. Details of each condition mentioned are to be found either in this section or elsewhere in the book.

In addition to lameness, hindlimb 'weakness' may result from other diseases and should be kept in mind, particularly if any neurological deficits are detected on examination. These would include:

Table 38.1 Differential diagnosis of hindlimb lameness.

Age of animal	Acute onset	Insidious onset
Skeletally immature (<1 year of age)	• Fractures (especially physeal and particularly proximal/distal femoral) • Avulsion injuries (especially tibial tuberosity and long digital extensor tendon origin) • Patellar luxation • Panosteitis • Metaphyseal osteopathy • Injury to the foot • Traumatic luxation (uncommon as more likely to fracture physis) • Septic arthritis	• Hip dysplasia • Associated with growth deformities (genu valgum, pes valgus or varus) • Patellar luxation • Osteochondrosis: ◦ hip – (OCD), Legg Perthes' disease (?) ◦ stifle – OCD ◦ hock – OCD ◦ sesamoid disease (?) • Immune-mediated polyarthritis
Skeletally mature	• Fractures • Traumatic luxation (hip > tarsus > stifle or patella) • Cranial cruciate ligament failure • Neoplasia (especially bone tumour with pathological fracture) • Injury to the foot • Intervertebral disc prolapse (causing nerve root entrapment)	• Cranial cruciate ligament failure • Osteoarthritis (hip = stifle > hock) • Lumbosacral disease • Patellar luxation • Neoplasia (bone, periarticular soft tissues, lumbosacral plexus) • Soft tissue 'injuries' (gracilis contracture, gastrocnemius enthesiopathy) • Immune-mediated polyarthritis

OCD – osteochondritis dissecans.

- Conditions of the vertebral column (see Section 4), especially:
  - trauma
  - atlantoaxial subluxation
  - cervical disc disease
  - cervical spondylopathy
  - thoracolumbar disc disease
  - lumbosacral disease
  - neoplasia
  - congenital anomalies (e.g. hemivertebrae)
  - fibrocartilaginous emboli
  - degenerative myelopathy
- Nutritional bone disease (see Chapter 47)
- Myopathies (see Chapter 49)
- Neuropathies (see Chapter 50)

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Chapter 39
The Pelvis

Although the bones of the pelvis may be affected by conditions such as neoplasia, the most common problems encountered, by far, are those of fractures and it is these that will be dealt with in this chapter. Pelvic fractures are a common injury in the dog and cat. Most pelvic fractures are as a result of road traffic accidents (Denny, 1978; Betts, 1993). Other causes include falls, kicks and crushing injuries. Stress fractures occur in racing Greyhounds and pathological fractures, secondary to neoplasia, are seen occasionally.

In many cases, pelvic fractures resulting from road traffic accidents will be complicated by other injuries, particularly to the chest and abdomen. There may well be other orthopaedic or spinal injuries present. Specific complications relating to pelvic fractures include:

- Ruptured/perforated bladder
- Ruptured/perforated/avulsed urethra
- Perforated rectum
- Peripheral nerve injury
- Prepubic tendon rupture
- Perineal lacerations

An approach to the examination and management of dogs or cats presented with pelvic fractures was published by Innes & Butterworth (1996). A careful clinical examination should be carried out, paying particular attention to the cardiovascular and respiratory status of the animal. Appropriate intravenous fluids should be administered in all cases initially. Chest radiographs should be taken in the early stages of case management as thoracic injury is common in fracture patients. The provision of analgesia is also important. Animals with pelvic fractures may be hypovolaemic and/or have renal dysfunction. Therefore, non-steroidal anti-inflammatory drugs (NSAIDs) should generally be avoided although carprofen (Rimadyl, Pfizer), being a prostaglandin-sparing agent, can be used more safely. In many cases, opioids such as morphine or pethidine will be the analgesics of choice.

The urinary tract is at particular risk in cases of pelvic trauma. A full bladder at the time of impact can result in rupture. The urethra may also rupture, particularly in male dogs. The ability of the patient to urinate does not preclude serious injury to the lower urinary tract. A plain lateral abdominal radiograph may allow an intact urinary bladder to be identified but a positive contrast retrograde urethrocystogram should be performed if there is any doubt about the integrity of the bladder or the urethra. Injury to other abdominal organs may be apparent on abdominal radiographs and ultrasonography may be helpful in assessing injury to liver, spleen or kidneys and for detecting intraperitoneal haemorrhage or other fluid in the abdomen. Rectal examination can be useful (see below).

Peripheral nerve injury, e.g. sciatic nerve paresis, is common following pelvic fracture but fortunately this is usually only a transient problem. A thorough neurological examination should be performed in all pelvic fracture cases, it being particularly important to identify any concurrent spinal injury. The presence of severe pain may make interpretation of tests such as proprioception and the withdrawal reflex difficult. However, it should be possible to assess conscious pain sensation (CPS) in the hindlimb digits by pinching them or, if necessary, by the application of forceps. Loss of CPS indicates severe neurological injury and spinal radiographs should be taken to check for fracture, luxation or disc lesions. Indeed survey radiographs of the

vertebral column may be indicated in any patient where immobility or pain resulting from pelvic injury precludes complete evaluation of spinal function.

Clinical assessment

Animals with unilateral pelvic fractures will generally present with unilateral lameness. If the fractures are bilateral, and particularly if they involve weight-bearing areas, then the patient may not be able to stand or walk initially. Gentle palpation of the pelvis will usually reveal swelling and asymmetry resulting from fracture. The palpable landmarks of the pelvis are the iliac crest and the ischial tuberosity; asymmetry is assessed by comparing the relationship of these structures with the greater trochanter on both sides of the pelvis (Fig. 39.1a). Rectal examination can give information about displacement of pelvic fragments and the appearance of blood on the finger may indicate rectal damage which will require further evaluation. Although rectal perforation is uncommon it is imperative that it is diagnosed early as a delay in treatment will make the prognosis hopeless (Lewis *et al.*, 1992).

Radiographic assessment

Lateral and ventrodorsal radiographs are necessary to evaluate pelvic fractures and to assess treatment options. Because of the box-like structure of the pelvis, a solitary fracture of the pelvis is unusual. If one part of the pelvis fractures then invariably there will be other pelvic fracture sites (Fig. 39.1b). Numerous combinations of fracture site are possible (Denny, 1978). However, some combinations are seen more frequently than others, for example fracture of the ipsilateral ilium, pubis and ischium, or sacroiliac separation with fracture of the contralateral ilium, pubis and ischium. On radiographs of the pelvis it is important to also look for complicating injuries, e.g. a sacral fracture which might be associated with neurological damage, particularly the lower motor neuron (LMN) supply to the bladder.

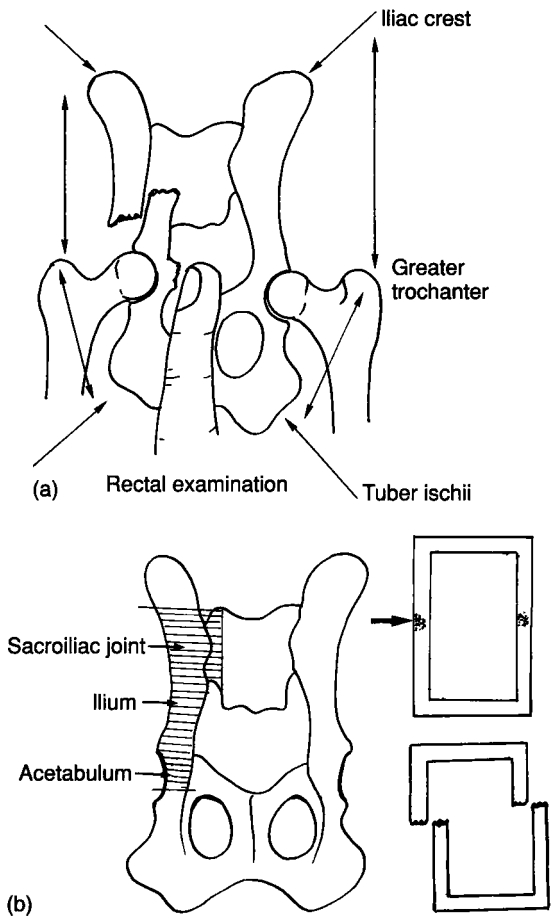


Fig. 39.1 (a) Palpation of the fractured pelvis. (b) Radiographic examination. Look for more than one pelvic fracture. Hatched area is the weight-bearing area of the pelvis.

Treatment options

The majority of animals (about 75%) would eventually recover with conservative treatment (Denny, 1978). However, in some cases malalignment and/or instability of the fragments may result in a prolonged recovery period, narrowing of the pelvic canal or a limited range of hip movement. Fractures involving the weight-bearing areas (or axes) of the pelvis are the ones that are subjected to internal fixation most often. These areas include (Fig. 39.1b):

- The sacroiliac joint
- The iliac shaft
- The acetabulum

The ideal fractures for conservative treatment (see below) are those involving non-weight-bearing areas of the pelvis, provided the fractures are not causing extreme narrowing of the pelvic canal which might later result in obstipation. The non-weight-bearing areas of the pelvis are:

- The wing of the ilium
- The pubis
- The ischium
- The caudal acetabulum

The type of patient also influences treatment selection. The small, well-muscled dog, like the Jack Russell Terrier, will often respond better to conservative managment than larger, thinner breeds of dog. The width of the pelvic canal is an important factor in breeding bitches or queens and medial displacement of fragments in these animals is best corrected surgically to avoid possible dystocia during future parturition. It may be preferable to treat performance or working animals surgically. In the case of stress fracture of the acetabulum in racing Greyhounds, surgery seems to be the treatment of choice (Wendelberg *et al.*, 1988).

Concomitant injuries may delay or reverse a decision to treat pelvic fractures surgically. Chest and abdominal injuries will obviously take priority over pelvic fractures. Timing of surgery is also important in pelvic fractures. Surgical treatment is not easy and should, ideally, be performed within 48 hours, or as soon as possible after stabilising the patient. If more than 5–7 days have elapsed since the accident, it can become very difficult to reduce the fractures due to muscle contraction, and after 10 days reduction becomes almost impossible without risk of damage to adjacent soft tissues.

Conservative management

Conservative management involves cage rest for an average of 4 weeks (range from 2 to 8 weeks). If the dog is unable to take weight on the hindlegs a foam rubber mattress should be provided for the animal to lie on. Gentle massage and regular turning should be encouraged to prevent the development of decubitus ulcers. Analgesia should be provided as appropriate. The animal's ability to urinate and defecate should be carefully monitored and assisted when necessary, this may simply involve helping the dog to stand using an abdominal sling, but in some cases the bladder may require manual expression or catheterisation. Movement should be kept to a minimum during the first 2–3 weeks, but as healing proceeds the dog can be encouraged to stand and walk. If necessary, it can be helped to do so by using an abdominal sling. In patients with symphyseal fractures, hobbles can be used to prevent hindlimb abduction.

Surgical treatment of pelvic fractures

The specific indications for surgery include:

- Displaced fractures of the acetabulum, particularly those involving the cranial half of the acetabulum
- Fractures of the ipsilateral ilium, pubis and ischium, resulting in gross instability of the acetabular segment
- Fractures of weight-bearing areas, particularly if displaced and causing stenosis of the pelvic canal
- Bilateral pelvic fractures involving weight-bearing areas and multiple pelvic fractures complicated by other limb bone fractures

If a fracture of the weight-bearing area of the pelvis is reduced and stabilised, this will bring about simultaneous reduction of the more caudal fractures involving non-weight-bearing areas of the pelvis, particularly if the surgery is carried out within 48 hours of the accident.

Sacroiliac separations and fractures

Sacroiliac separations/fractures may be unilateral or bilateral. Unilateral injuries are encountered most often and tend to be associated with fractures of the ipsilateral or contralateral ilium, ischium and/or pubis. Intense pain is often a feature of sacroiliac separations/fractures and is probably the result of nerve root damage in the lumbosacral plexus. Most cases would recover with conservative treatment (average recovery time 6 weeks), however, surgical stabilisation of the sacroiliac joint is indicated if there is:

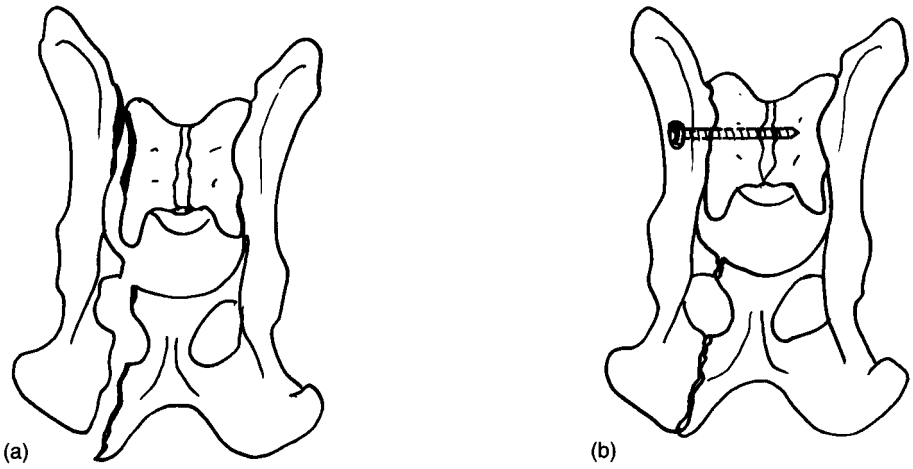


Fig. 39.2 (a) Sacroiliac separation with fracture of ipsilateral pubis and ischium. (b) Lag screw fixation. Notice the simultaneous reduction of caudal fracture sites.

- Severe and persistent pain
- Marked displacement
- Contralateral pelvic or hindlimb injuries (surgical stabilisation in these cases will permit an earlier return to weight-bearing and protect the contralateral repair)

Fixation techniques include the use of lagged bone screws (DeCamp & Braden, 1985a,b) or transilial pinning (Leighton, 1968; Whittick, 1974). Screw fixation is preferred. Traditionally, sacroiliac separations were stabilised with two short lag screws passed through the wing of the ilium into the sacrum. Short screws were used to avoid inadvertent penetration of the neural canal. There is a tendency for short screws to loosen and successful repair is best achieved by placing a *single, long screw* ventral to the neural canal into the sacral body. The screw should penetrate at least 60% of the sacral width (DeCamp & Braden, 1985a,b) (see below) (Fig. 39.2a,b).

The transilial pin

This method is used when there is a sagittal fracture of the sacrum in which insufficient bone remains medially to permit the use of a lag screw. Following open or closed reduction of the sacroiliac fracture/separation, a Steinmann pin is driven through the wing of the ilium on the unstable side. It passes over the dorsal aspect of the seventh lumbar vertebra, passing through or caudal to its dorsal spine, and then on into the contralateral iliac wing (Fig. 39.3).

Lag screw fixation

A dorsal approach is used to expose the sacroiliac joint (Piermattei, 1993). A skin incision is made directly over the iliac crest (Fig. 39.4a). The cutaneous trunci muscle and the gluteal fascia are incised to reveal the middle gluteal muscle and the iliac crest. Laterally the wing of the ilium is exposed by elevating the origin of the middle gluteal muscle (Fig. 39.4b). The muscles which cover the sacroiliac joint and insert on the medial surface of the crest and wing of the ilium (iliocostalis and longissimus groups) are usually severely damaged and little elevation is required to expose the sacroiliac joint. Retraction of the iliac wing ventrally, using a Hohmann retractor, exposes the lateral aspect of the sacral wing (Fig. 39.4c). A pilot hole is drilled in the sacral body first (the hole should penetrate at least 60% of the sacral width). Landmarks for estimating the correct position for the pilot hole in the sacral body are shown in Fig. 39.4d. The pilot hole is measured with a depth gauge and a thread is cut with the appropriately sized tap. In many cases, especially young animals, the tap needs only to be started in the sacrum as even non-self-tapping

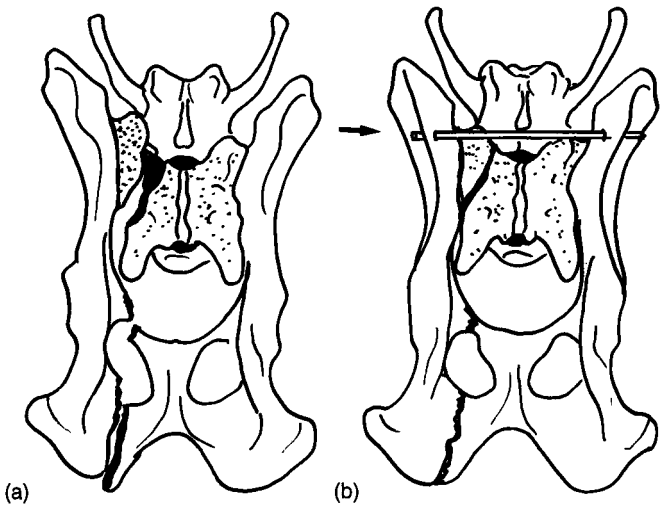


Fig. 39.3 (a) Fracture separation of the sacroiliac joint. (b) Transilial pin used for fixation.

screws will drive themselves into the cancellous bone of the sacral body. A glide hole is then drilled through the wing of the ilium, to emerge through the centre of the raised medial articular surface (Fig. 39.4c). The screw selected is introduced through the glide hole and the tip directed into the pilot hole as the sacroiliac separation is reduced (Fig. 39.4e) (this can be difficult!). If the pilot hole does not exit the sacral body then this must be taken into account when selecting the screw length – the use of a screw that is too long will result in incomplete reduction of the sacroiliac junction, or else stripping of the threads. The screw is slowly tightened, completing reduction and stabilising the sacroiliac joint (Fig. 39.4f).

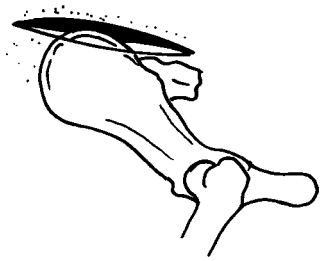
Iliac shaft fractures

Stable fractures, or those with relatively little displacement, may be treated conservatively, but most iliac shaft fractures will benefit from internal fixation. Plate fixation (Robins *et al.*, 1973; Brown & Biggart, 1975) is the most versatile method of dealing with these fractures (Fig. 39.5). In long, oblique fractures of the shaft, lag screws can be placed in a ventrodorsal direction (Brinker, 1974) and used either as the sole method of fixation (Fig. 39.6a) or in conjunction with a neutralisation plate. Cats have a relatively straight iliac shaft and intramedullary pins or Kirschner wires can be used to stabilise transverse or blunt, oblique fractures in this region (Fig. 39.6b).

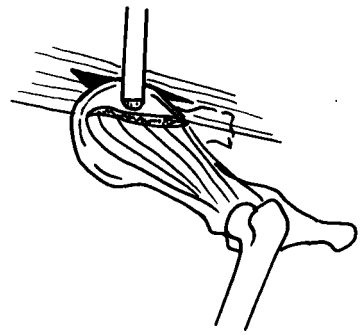
Exposure

Exposure of the shaft of the ilium is illustrated in Fig. 39.7. A lateral approach, with dorsal reflection of the gluteal muscles, is used to expose the ilium (Brinker, 1974). A skin incision is made from the iliac crest and extended caudally over the greater trochanter (Fig. 39.7a). The incision is continued through the subcutaneous fat and gluteal fascia to expose the aponeurosis between the middle gluteal muscle and the tensor fascia lata (Fig. 39.7b). The two muscles are separated and the lateral surface of the shaft and the wing of the ilium is exposed by dorsal reflection of the middle and deep gluteal muscles (Fig. 39.7c). Exposure is maintained with a Hohmann retractor placed dorsally with its tip on the medial side of the ilium. Care should be taken when applying bone-holding forceps to the iliac shaft so as not to damage the sciatic nerve which runs close to the medial aspect of the ilium.

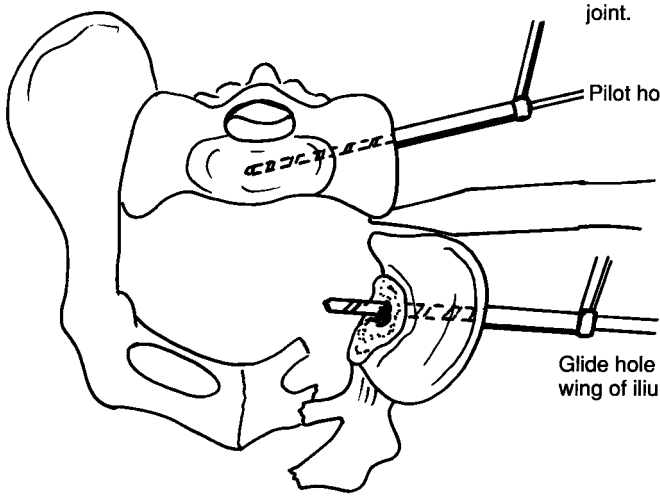
If a plate is to be used for fixation (Fig. 39.7d) it should be carefully contoured to the shape of the ilium, using the radiograph of the contralateral intact ilium as a guide (Fig. 39.5a). This is important because fracture of the ilium is frequently associated with fracture of the ipsilateral pubis and ischium. However, as the iliac fracture is reduced and its normal contour restored with the plate, simultaneous reduction of the other more caudal fractures usually occurs (Figs 39.5b and 39.8). A practical tip is to attach the plate to the caudal ilium first, reduce the fracture as much



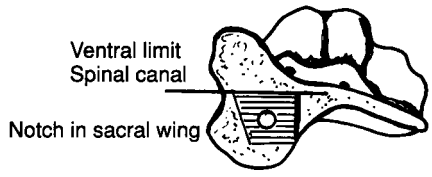
(a) Surgical approach with skin incision over the iliac crest.



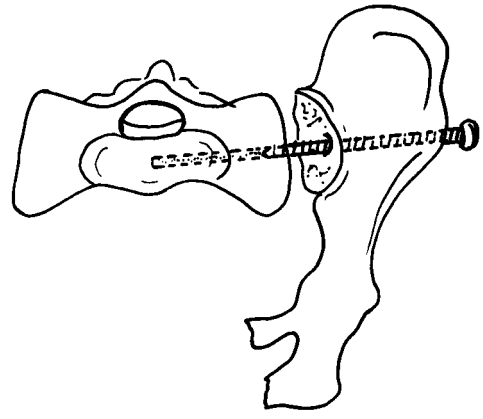
(b) Laterally the middle gluteal muscle is reflected. Medially the soft tissues covering the sacroiliac joint are usually torn already and require little dissection to expose the joint.



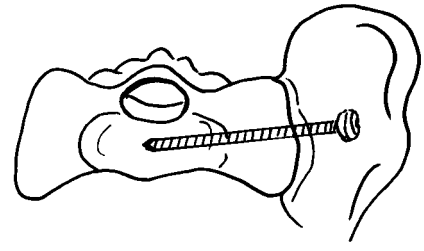
(c) Ventral reflection of the ilium using a Hohmann retractor.



(d) Lateral view of the sacrum. Hatched area represents a suitable area for screw placement (DeCamp & Braden, 1985a,b).



(e) Reduction commenced, lag screw placed through glide hole and engaged in pilot hole.



(f) Lag screw tightened, completing the reduction.

Fig. 39.4 Lag crew fixation.

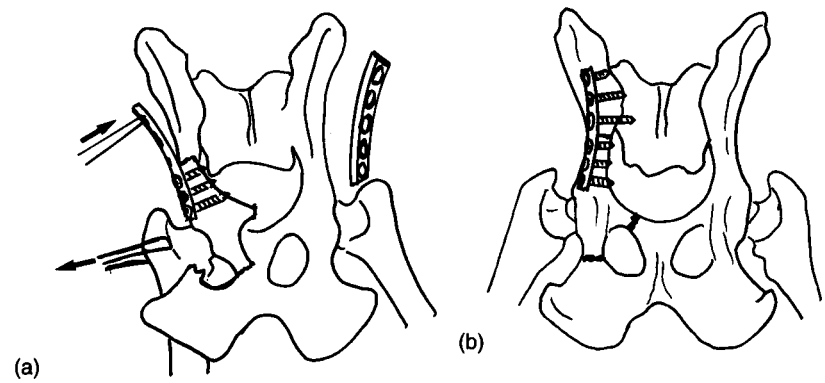


Fig. 39.5 (a) Plate fixation of iliac shaft fracture. A plate is contoured using a radiograph of the intact ilium as a guide. The plate is attached to the caudal fragment and the cranial end of the plate is used as a lever to complete the reduction. (b) Cranial screws placed completing the reduction of the iliac fracture and caudal fracture sites.

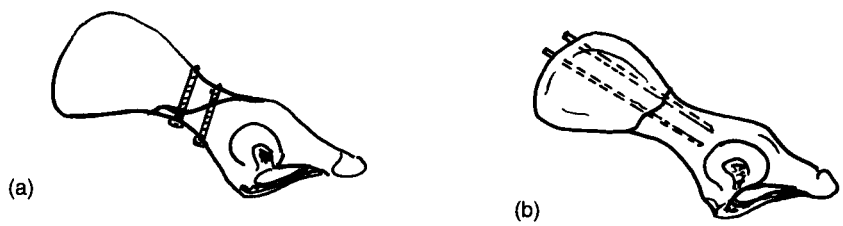


Fig. 39.6 Iliac fracture, (a) with lag screw fixation and (b) with a Kirschner wire fixation.

as possible using bone-holding forceps and then use the free cranial end of the plate as a lever arm to complete reduction.

Prognosis

Prognosis following plate fixation of iliac shaft fractures is excellent and some 90% of treated cases should make good recoveries (Brown & Biggart, 1975). Plate fixation also gives a much shorter recovery period, average of 3 weeks, compared with an average recovery period of 8 weeks in conservatively managed cases (Denny, 1978).

Acetabular fractures

The craniodorsal two-thirds of the acetabulum is the main weight-bearing area of the hip. Fractures involving this area, even if only moderately displaced, should be treated surgically; so should severely displaced fractures of any part of the acetabulum (Fig. 39.9a). Undisplaced fractures, particularly if they involve the caudal third of the acetabulum, can be successfully managed with conservative treatment (Fig. 39.9a). The same may apply to moderately displaced fractures of the caudal acetabulum (Butterworth *et al.*, 1994) though other reports recommend against this (Boudrieau & Kleine, 1988). Some acetabular fractures may be too severely comminuted to allow surgical reconstruction and these cases are managed conservatively with the options of excision arthroplasty or total hip replacement at a later date if hip pain persists and the patient is a suitable candidate (total hip replacement may not be an option in all dogs or healed acetabular fractures).

The aim of treatment of acetabular fractures is early, accurate anatomical reduction of the fracture and rigid fixation to promote primary bone union. Application of a plate to the dorsal rim of the acetabulum is currently the most widely

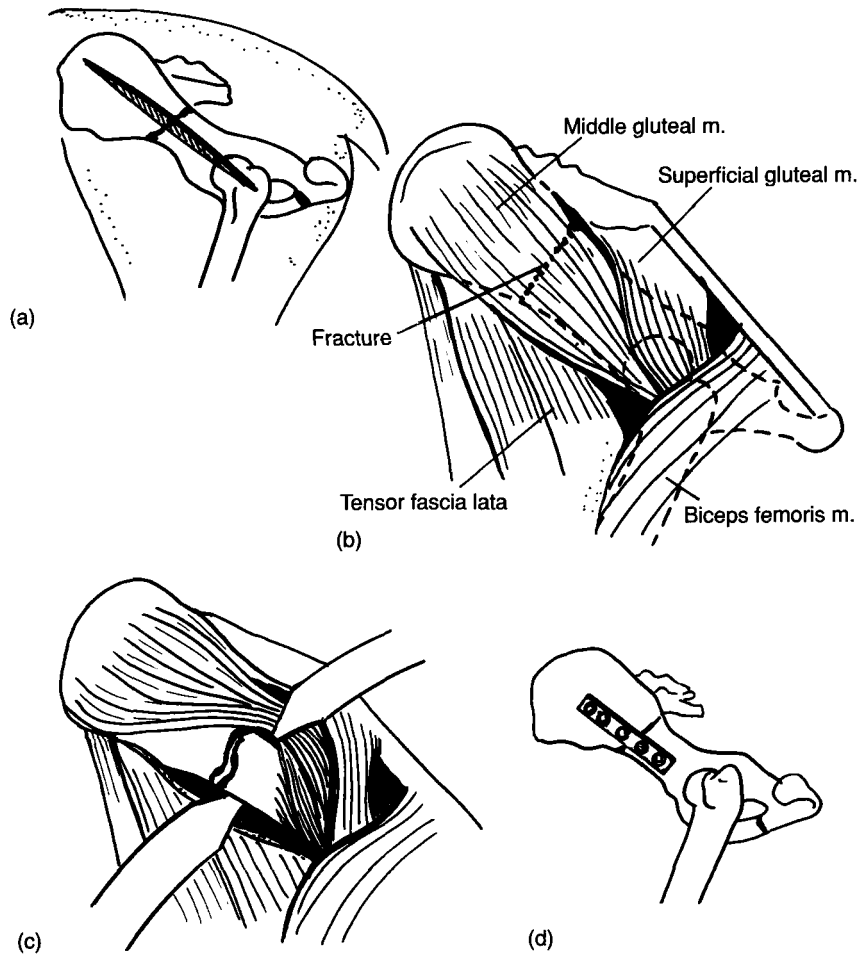


Fig. 39.7 Exposure of the ilium. (a) Position of skin incision. (b) Surgical anatomy. (c) The gluteal fascia is incised, the tensor fascia lata muscle is reflected ventrally, and the middle and deep gluteal muscles are rolled up dorsally to expose the iliac shaft and fracture. (d) A plate is applied.

preferred method of treatment. Curved acetabular plates (Fig. 39.9b,c) are the easiest to use as they do not usually require any further contouring (AO/ASIF acetabular plate, Stratec; or curved, round-hole acetabular plates, Veterinary Instrumentation). Reconstruction plates (Stratec) are useful in comminuted acetabular fractures or combined iliac and acetabular fractures (Dyce & Houlton, 1993). If a reconstruction plate or a dynamic compression plate is to be used to stabilise an acetabular fracture then operating time can be reduced by precontouring the plate to the shape of the dorsal acetabulum using a similar-sized pelvis before commencing surgery. A recent report has described the use of bone screws and polymethylmethacrylate to create an 'internal fixator' for stabilising acetabular fractures (Lewis *et al.*, 1997; Stubbs *et al.*, 1998) (Fig. 39.10a).

Oblique fractures of the caudal ilium which extend into the cranial acetabulum can be stabilised with lag screws (Fig. 39.11). In small dogs and cats an alternative to plate fixation is to use two screws or two Kirschner wires and a tension band wire (Fig. 39.10b,c).

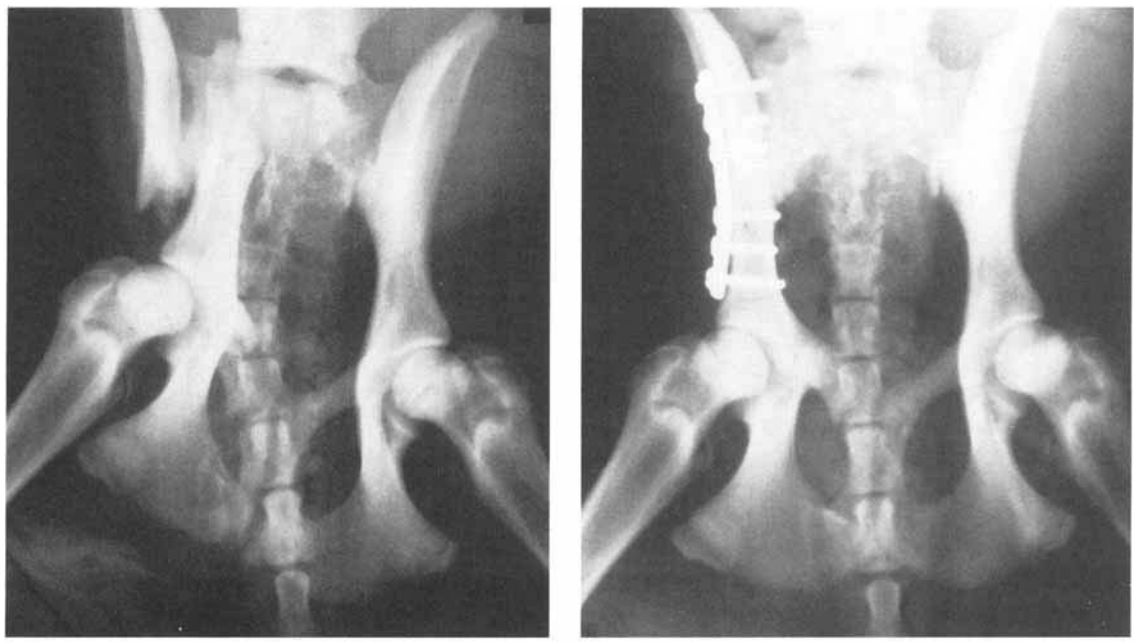


Fig. 39.8 (a) *Preoperative pelvic radiograph of an 8-month-old Golden Retriever showing fractures of the ipsilateral ilium, pubis* and ischium. (b) Postoperative radiograph showing that the iliac shaft fracture has been reduced and stabilised with a plate (2.7 DCP [dynamic compression plate]). Notice that accurate reduction of the iliac fracture has resulted in simultaneous reduction of the pubis and ischial fractures.

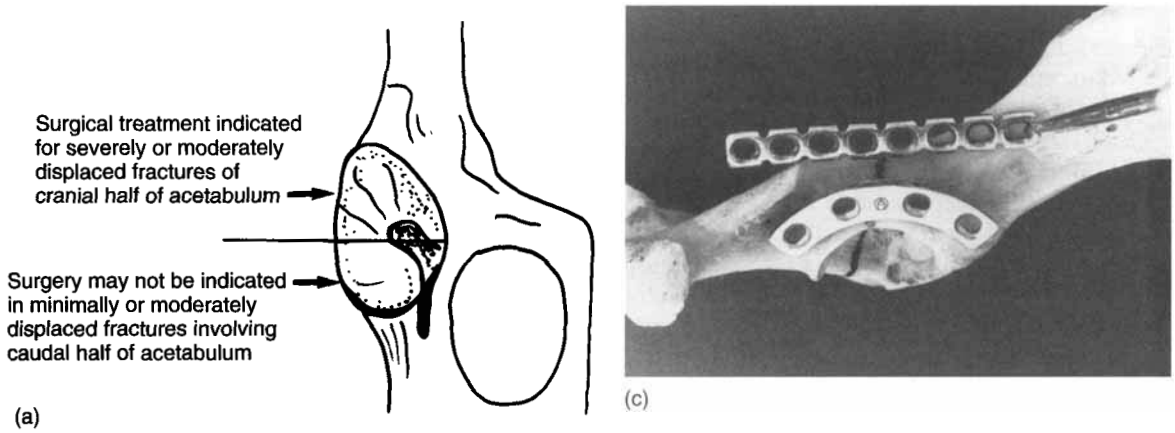


Fig. 39.9 Plate fixation of acetabular fractures. (a) The choice of surgical or conservative treatment. (b) Placement of an acetabular plate (Veterinary Instrumentation). Plates are most commonly used for fixation. Alternative methods are shown in Figs 39.10 and 39.11. (c) Types of plates used for fixation. Top – reconstruction plate (Stratec); bottom – AO/ASIF acetabular plate (Stratec). (Reproduced from Denny, H.R. (1991) *In Practice*, July, 139. With perminion from BVA Publications.)

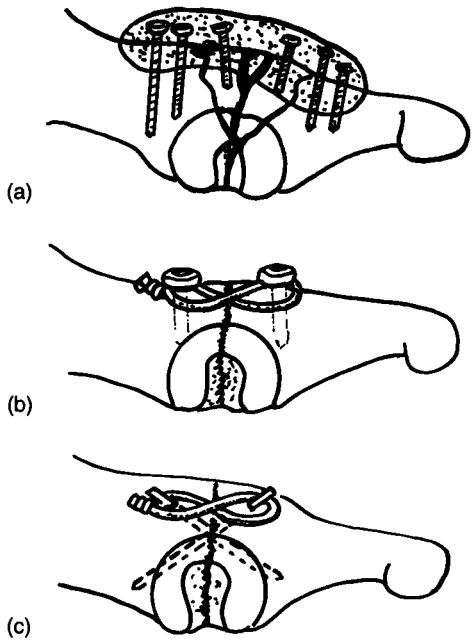


Fig. 39.10 Alternatives to plate fixation for acetabular fractures in small dogs and cats. (a) Comminuted fractures of the acetabulum can be stabilised using polymethylmethacrylate (PMMA). Small screws or Kirschner wires are placed in the fragments and used as anchor points for the PMMA. (b) Two lag screws and a tension band wire. (c) Crossed Kirschner wires and a tension band wire.

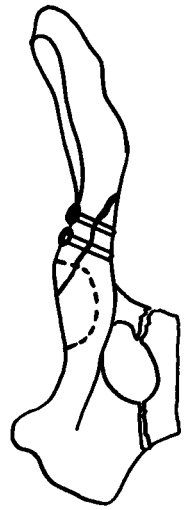


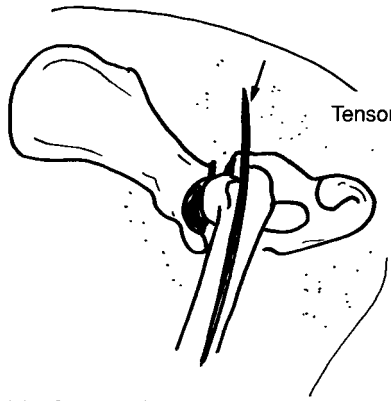
Fig. 39.11 Oblique fracture of the caudal ilium extending into the cranial acetabulum. Lag screws are used for fixation.

Dorsal approach to the hip

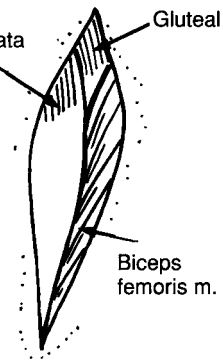
A dorsal approach to the hip (Piermattei, 1993) is used for most reconstructive surgery including the repair of acetabular fractures. A skin incision is made directly over the greater trochanter (Fig. 39.12a). The tensor fascia lata is incised just cranial to the biceps femoris muscle. The fascial incision is continued proximally over the greater trochanter separating the tensor fascia lata muscle from the biceps femoris. The biceps femoris muscle is retracted caudally to expose the greater trochanter and the gluteal muscle mass (Fig. 39.12b). Caudal to the hip, the sciatic nerve is identified and protected. Tenotomy of the tendon of insertion of the superficial gluteal muscle is performed (Fig. 39.12c). Transverse osteotomy of the greater trochanter or serial transection of the gluteal muscles' tendons is then carried out (Fig. 39.12c). Osteotomy is performed using a saw or osteotome just distal to the insertion of the middle gluteal muscle. If a lag screw is to be used to repair the trochanteric osteotomy then the screw hole should be drilled before the osteotomy is performed. The gluteals are reflected dorsally to expose the joint capsule (Fig. 39.12d). The joint capsule is opened to allow inspection of the articular surfaces. Soft tissue is elevated from the dorsal rim of the acetabulum. The caudal muscles of the hip, the obturator and the gemelli, are cut close to their insertion on the femur and are then reflected as a sling to protect the sciatic nerve during exposure of the caudal acetabulum and body of the ischium (Fig. 39.12d).

Pointed AO reduction forceps are useful for grasping acetabular fragments and also for maintaining reduction during application of the plate. Other aids to reduction are to exert traction on the caudal acetabular fragment by temporarily transfixing the tuber ischium with a Steinmann pin (Fig. 39.13a) or by direct traction on the ischium with Kern bone-holding forceps applied through a separate small skin incision over the ischium (Fig. 39.13b). In addition, it is helpful to attach the plate to the caudal acetabular fragment first and then use the cranial free end of the plate as a lever arm to complete the reduction.

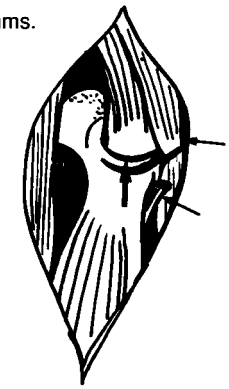
Closure involves suturing the joint capsule and then reattaching the gluteals using either tendon



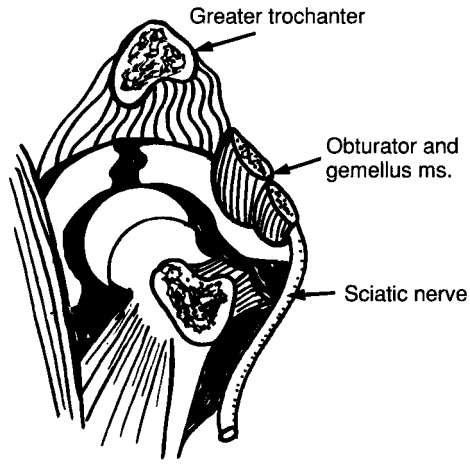
(a) Skin incision.



(b) Superficial muscles of the hip are exposed.

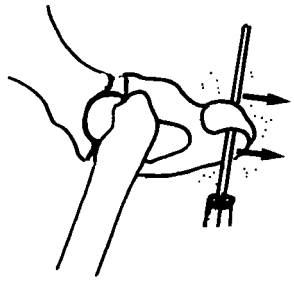


(c) Tenotomy of the superficial gluteal muscle (small arrows), osteotomy of the greater trochanter (large arrow).

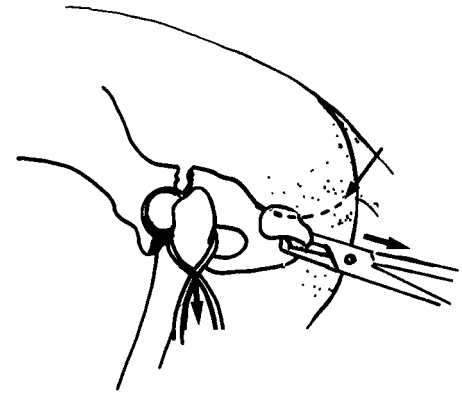


(d) Trochanter and gluteal muscles reflected dorsally. Tenotomy of the obturator and gemellus muscles. These are reflected and used as a sling to protect the sciatic nerve during exposure of the caudal acetabulum.

Fig. 39.12 Dorsal approach to the hip used to expose an acetabular fracture.



(a) The ischium is temporarily transfixed with a pin to allow traction.



(b) A small incision is made over the ischium (dashed line), and direct traction is applied on the ischium with reduction forceps.

Fig. 39.13 Methods of reduction of acetabular fractures.

sutures (in the case of tenotomy) or by stabilising the greater trochanter (in the case of osteotomy). The latter is achieved with a lag screw or two Kirschner wires and a tension band wire (see Chapter 41, Fig. 41.4). Exercise is restricted for 6 weeks following surgery.

Prognosis

Prognosis following internal fixation of acetabular fractures is generally good provided accurate anatomical reduction and stable fixation is achieved. If these criteria are met then 80% of treated cases should have an excellent or good outcome (Dyce & Houlton, 1993).

Stenosis of the pelvic canal

In general practice, the majority of pelvic fractures encountered in cats tend to be managed conservatively, the cat being given cage rest for 4–6 weeks. The functional end results tend to be good. However, a late complication is obstipation associated with stenosis of the pelvic canal. This seems to be a far more common problem in cats than in dogs.

Stenosis can be relieved by splitting the pelvic symphysis and spreading this open with an ulnar autograft (McKee & Wong, 1994), an allograft (Brinker *et al.*, 1983), or a steel insert (Ward, 1967; Leighton, 1969). The technique of symphyseal distraction–osteotomy using an ulnar autograft is illustrated in Fig. 39.14. An alternative is resection of the ventral pelvis (Fig. 39.15a). For this, a ventral midline approach is used to expose the pelvic symphysis. Having elevated soft tissues from the pubic symphysis, the prepubic tendon is freed from its attachment to the pubis taking care to avoid the inguinal vessels. The pubis and ventral ischium are resected with rongeurs on either side of the symphysis so that, in effect, the ventral floor of the pelvic canal is removed (Fig. 39.15b). During resection of the ventral ischium care should be taken to protect the obturator nerve which runs through the obturator foramen. During wound closure the prepubic tendon is sutured to the ventral musculature which is, in turn, coapted in the midline.

Resection of the ventral pelvis is a simple technique and has given good results with permanent relief from obstipation in a series of six cats treated by one of the authors. The procedure does not cause pelvic instability provided the sacroiliac joints are stable and the other pelvic fractures have healed. Some hindleg weakness may be noticed for a few days following surgery but this tends to be a transient problem.

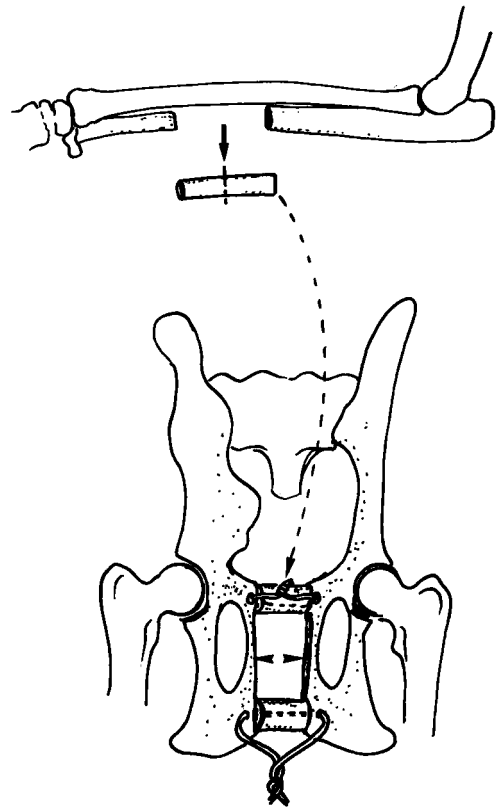


Fig. 39.14 Pelvic stenosis in the cat treated by symphyseal distraction–osteotomy using an ulnar autograft (McKee & Wong, 1994).

In some cases it may be more appropriate to create a partial hemipelvectomy in order to remove the segment of bone causing the obstruction. Usually this is an acetabular segment that has been displaced medially following fractures of the ipsilateral ilium, pubis and ischium. An alternative to such resection would be to create a triple pelvic osteotomy and apply a contoured plate (usually requires a fairly dramatic angulation) to the ilium so as to bring the displaced segment out of the pelvic canal (Ferguson, 1996).

Whatever technique is chosen for relief of

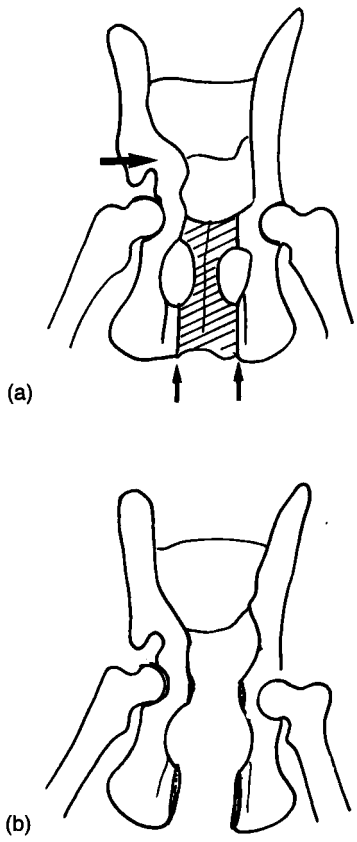


Fig. 39.15 Pelvic stenosis in the cat treated by pelvic symphyseal resection. (a) Hatched area shows area of pubis to be removed. (b) Completed resection.

pelvic stenosis, it is recommended that surgery is reserved only for those cats in which the injury creating the stenosis occurred less than 6 months ago (Matthiesen *et al.*, 1991; Schrader, 1992). In more long-standing cases chronic colonic distension may result in intramural myoneuronal damage and irreversible colonic dysfunction. Surgical relief of pelvic stenosis in these chronic cases is unlikely to be of any benefit.

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Further reading

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A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny,Steven J. Butterworth

Chapter 40
The Hip

Anatomy

The hip is a diarthrodial ('ball-and-socket') joint allowing a wide range of motion in extension and flexion and also abduction and adduction. There is no well-defined end-point to motion in any of these directions and it is the tension created in the soft tissues that limits the range of motion. The femoral neck is inclined at an angle of 130–145°, relative to the femoral shaft, and is rotated forwards (anteverted) through 10–40° (Fig. 40.1) The inherent stability of the 'ball-and-socket' configuration is augmented by the teres ligament (passing from the acetabular fossa to the fovea on the femoral head), the transacetabular ligament (on the ventral aspect of the joint), the joint capsule and the musculature originating on the pelvis and inserting on the femur. These anatomical features are illustrated in Figs 40.2–40.4.

Conditions of the hip joint

Conditions of the hip can be divided into three main catagories:

- *Traumatic*
  - Fractures of the acetabulum (see Chapter 39, p. 447)
  - Fractures of the femoral head and neck (see Chapter 41)
  - Coxofemoral luxation
- *Developmental*
  - Legg Perthes' disease
  - Hip dysplasia
- *Acquired*
  - Osteoarthritis
  - von Willebrand-associated heterotopic osteochondrofibrosis of Dobermanns (VW HOOD)
  - Neoplasia (see Chapters 10 and 51)
  - Immune-mediated arthropathy (see Chapter 8)

Coxofemoral luxation

History

Luxation of the hip is the commonest luxation seen in small animals. Any breed may be affected but most cases involve animals over 1 year of age. The injury which causes luxation in the mature animal is more likely to cause fracture of the proximal femoral physis in the skeletally immature patient. Luxation of the joint generally results from major injury, such as involvement in a road traffic accident. However, in some cases it may result from twisting awkwardly at exercise or by traction and rotation of the limb if the foot is caught in a fence or bitten by another animal.

Clinical signs

The femoral head luxates in a craniodorsal direction in 85–90% of cases. This results in a non-weight-bearing lameness with the limb held semi-flexed and adducted under the body creating outward rotation of the stifle. If both hindlimbs are held in extension then the affected limb will appear shorter (this is also true in cases with fracture of the proximal femur). With the animal standing, the triangle created by the greater trochanter, the iliac crest and the tuber ischii on each side is evaluated. This triangle will be altered

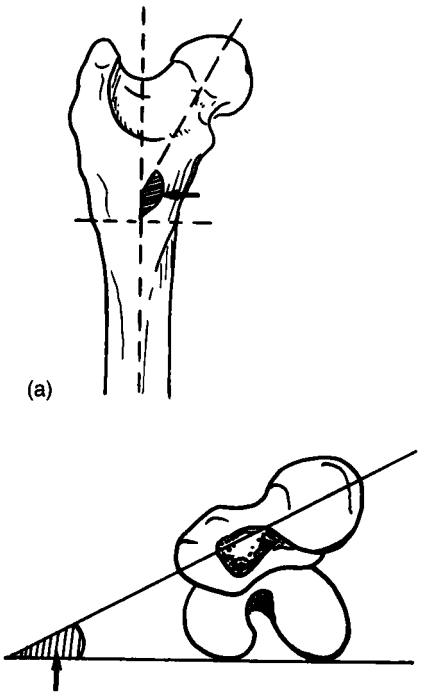


Fig. 40.1 Diagram illustrating (a) inclination and (b) anteversion angles of the femoral neck.

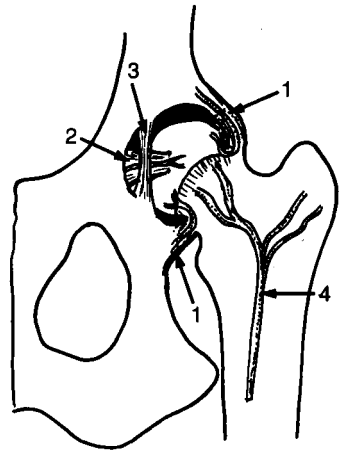


Fig. 40.2 Ventral view of the left hip showing: 1 – the joint capsule and epiphyseal blood vessels; 2 – the teres ligament; 3 – the transacetabular ligament; 4 – the metaphyseal blood vessels.

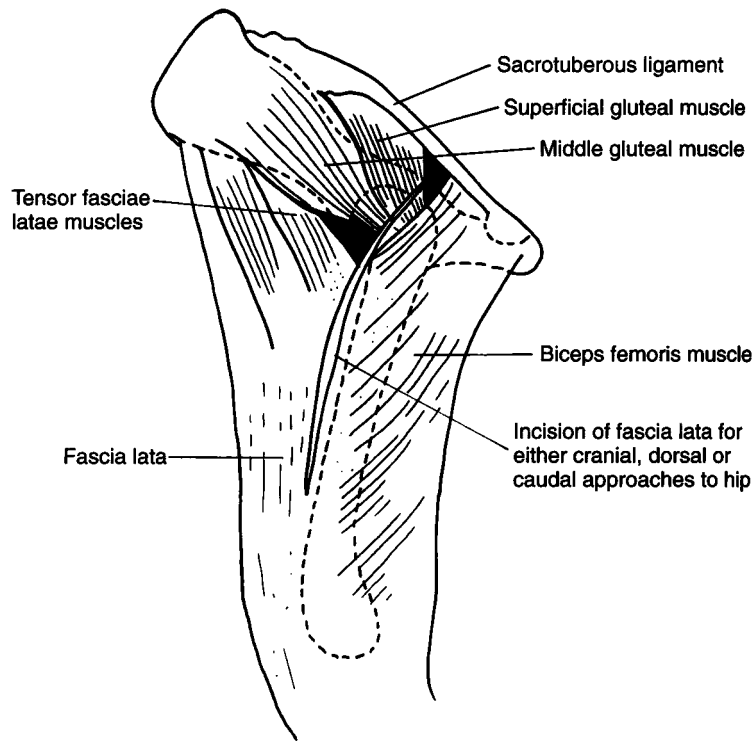


Fig. 40.3 Diagram illustrating the super-ficial musculature of the left hip from the lateral aspect.

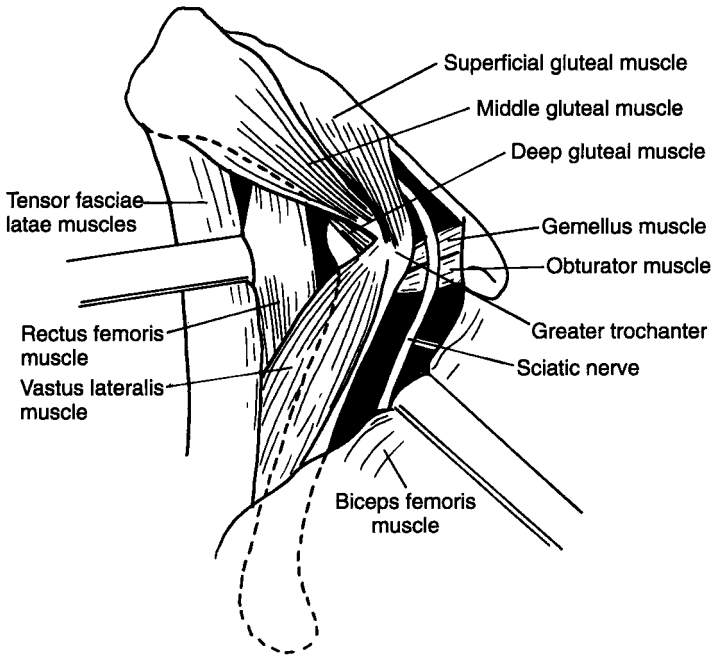


Fig. 40.4 Diagram illustrating the deep musculature of the left hip from the lateral aspect.

on the affected side such that the distance from the tuber ischii to the greater trochanter will be increased.

With the animal lying on its side the hip may be subjected to the *thumb displacement test.* The thumb of one hand is pressed firmly into the soft tissue depression caudal to the greater trochanter. The other hand then grasps the stifle and outwardly rotates the femur. In the case of a normal hip joint, the greater trochanter will move caudally, pushing the operator's thumb out of the soft tissue recess. In the case of luxation, the femoral head rotates cranially rather than the greater trochanter rotating caudally, and thus the operator's thumb is not displaced. Manipulation of the luxated hip usually demonstrates crepitation, pain and restriction in range of motion.

In the case of caudodorsal luxation, the limb is held abducted with the stifle inwardly rotated, the limb should appear slightly longer on extension and the distance between the greater trochanter and the tuber ischii will be reduced. Ventral luxation of the hip causes the limb to be carried in extension with slight abduction. The limb should again appear longer than the contralateral limb and the greater trochanter is difficult to palpate. Ventral luxations cause the greatest degree of disability and can cause considerable pain, which may be due to pressure on the obturator nerve as the femoral head comes to rest in the obturator foramen.

Radiology

Despite the fact that the clinical signs may be considered 'pathognomonic' for luxation of the hip, lateral and ventrodorsal radiographs (Fig. 40.5) of the pelvis must be taken to:

- Confirm the diagnosis (since fractures of the proximal femur may cause very similar signs)
- Determine the direction of luxation
- Establish that there is no avulsion of the teres ligament insertion (since the presence of a bone fragment in the joint space precludes any attempt to manage the luxation by closed reduction)
- Ensure there are no complicating fractures of the acetabulum (especially dorsal rim), iliac shaft and greater trochanter, or separation of the sacroiliac joint, all of which might contribute to coxofemoral instability

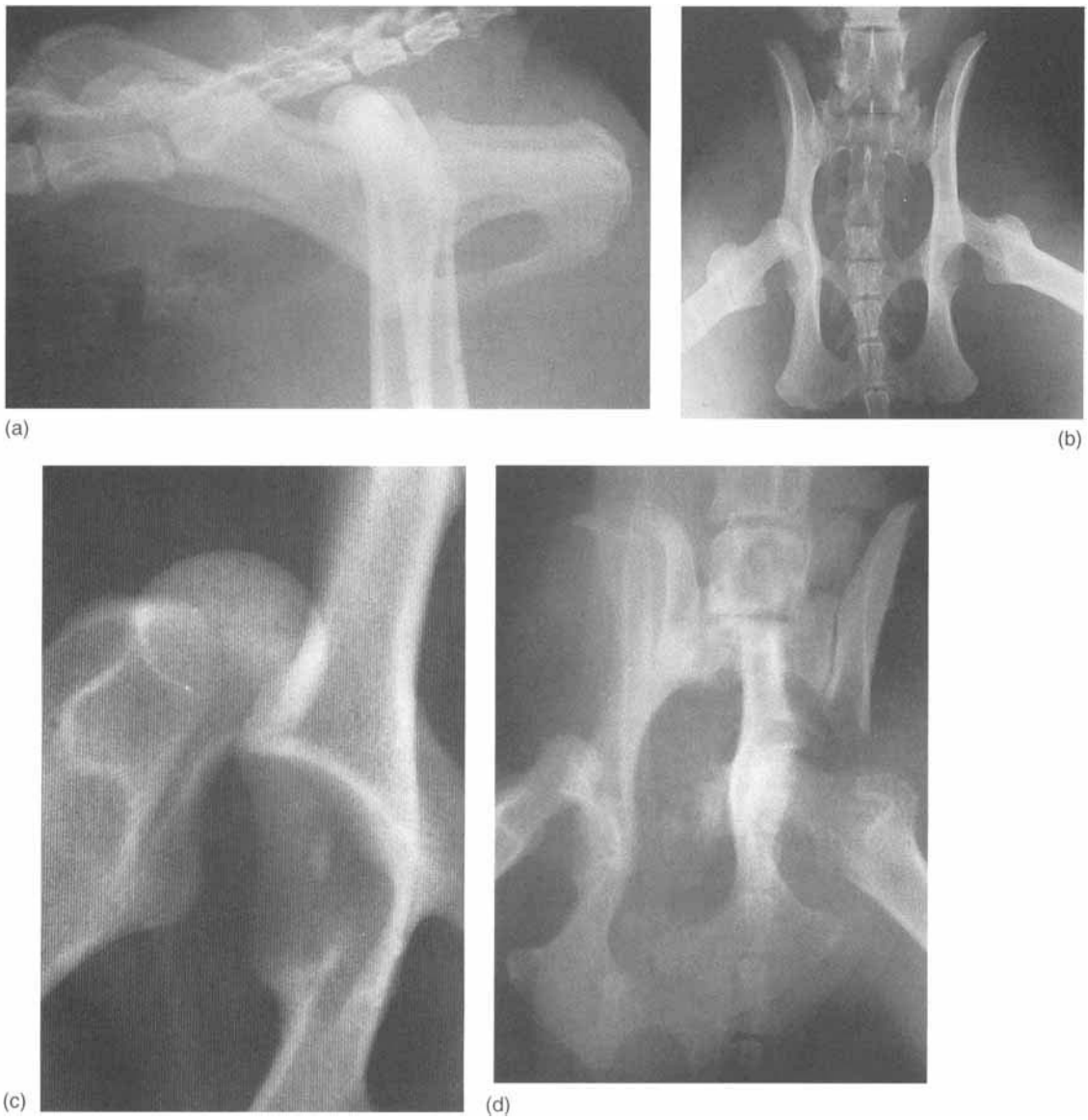


Fig. 40.5 (a) Lateral and (b) ventrodorsal radiographs of a 2-year-old cat showing craniodorsal luxation of the both hips. (c) Ventrodorsal radiograph showing a luxated hip with a small fragment of bone in the acetabulum indicating avulsion of the teres ligament insertion which is an indication for open reduction.
(d) Ventrodorsal radiograph of a 2-year-old Collie's pelvis after involvement in a road traffic accident. Besides a hip luxation there are fractures of the contralateral hemipelvis. Attempts at closed reduction of the luxation had, not surprisingly, been unsuccessful and treatment required plating of the iliac shaft and then open reduction and stabilisation of the hip joint.

Treatment

Dogs and, to a lesser extent, cats with hip luxations that are managed conservatively will always show a degree of disability in that limb and the only grounds for not pursuing closed or open reduction of the joint would be that of cost. The options available in managing acute, recurrent or chronic hip luxations include:

- Closed reduction +/–:
  - Ehmer sling

- DeVita pin
- 'dynamic', transarticular external skeletal fixator (ESF)

- Open reduction and suturing of the joint capsule +/–:
  - anchor suture
  - toggle fixation
  - transarticular pin
  - dorsal capsulorrhaphy
  - transposition of the greater trochanter

Closed reduction

This is best attempted within 48 hours of the luxation but is contraindicated in the presence of complications, most commonly where there is avulsion of the teres ligament insertion since closed reduction will leave the fragment of bone in the joint space. This fragment can be expected to cause reluxation through the femoral head not being properly 'seated' or a rapid onset of osteoarthritis due to it damaging the articular surfaces. The following description relates to a craniodorsal luxation and the procedure should be modified accordingly for the rare luxations in other directions.

The dog is anaesthetised and positioned in lateral recumbency with the affected leg uppermost. A strap of towelling is placed under the leg and secured to the edge of the table, or held by an assistant, to act as a 'groin sling' against which traction can be applied (similar to the way in which a rope is secured beneath the thigh prior to surgery, see later in Fig. 40.9). Traction is exerted in a ventrocaudal direction with the limb slightly abducted. This may allow the femoral head to be lifted over the acetabular rim and a 'clunking' sensation can usually be appreciated as the femoral head engages in the acetabulum. If traction alone does not cause this then thumb pressure can be applied to the greater trochanter to assist matters. Pressure is then applied over the greater trochanter whilst the joint is flexed and extended to express any haematoma from the joint space.

If the joint appears stable then radiographs are taken to ensure reduction has been achieved. *It is erroneous to assume that the hip has been reduced because it 'felt right' and not take post-reduction radiographs.* However, an accurately interpreted lateral view of the pelvis will usually suffice if there is concern over positioning for a ventrodorsal view causing reluxation (though a frog-legged ventrodorsal view is unlikely to cause reluxation). The animal may then be allowed to recover from anaesthesia or an Ehmer sling may first be applied for added security (Fig. 40.6). An Elastoplast (Smith & Nephew) bandage is taken and first of all wrapped around the foot. The leg is flexed and the bandage is then passed around the medial aspect of the stifle, brought over the cranial aspect of the distal femur and then taken down the medial side of the tibia to the hock. This is repeated several times and, if necessary, loops of bandage may then pass from below the hock over the dog's back to try and prevent attempts to extend the limb from causing the sling to slip off the thigh. The effect of this sling is to abduct the femur and rotate the femoral head into the acetabulum. Such a sling may be left in place for 3–10 days.

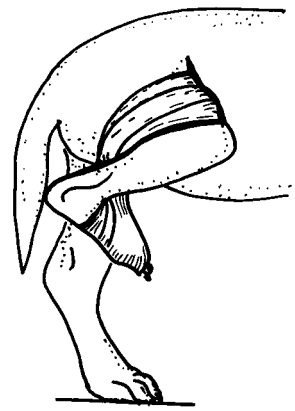


Fig. 40.6 Illustration of the application of an Ehmer sling.

Causes of reluxation following closed reduction, that has been confirmed radiographically, include:

- Poor seating of the femoral head within the acetabulum due to:
  - interposed, torn joint capsule
  - fragments of bone (especially avulsion of the teres ligament insertion)
- Inherent instability of the hip joint caused by:
  - hip dysplasia

- widespread damage to the supporting musculature
- concurrent ipsilateral fractures of the hemipelvis causing the acetabular 'segment' to be unstable

If the joint tends to reluxate on manipulation then the options include:

- Application of an Ehmer sling
- Placement of a DeVita pin
- Application of a 'dynamic' transarticular ESF
- Open reduction and stabilisation as listed above

If the reduction is unstable then it is unlikely an Ehmer sling will suffice and reluxation will occur either with the sling in place or else soon after it has been removed. Such a sling should not be applied and left in place for several weeks as the hip will probably reluxate and soft tissue contractures may develop which are more difficult to treat (sometimes impossible) than the luxation itself.

A DeVita pin is illustrated in Fig. 40.7. A Steinmann pin is introduced ventrally to the tuber ischium and driven forward over the neck of the femur and on into the wing of the ilium. The pin is left *in situ* for 3 weeks. Complications include pin migration (although using a threaded pin to gain better purchase in the ilium has been reported to reduce this), damage to the sciatic nerve during pin insertion and a relatively high rate of reluxation. In addition, the technique is difficult to apply in cats owing to their ilia being so straight, making placement of the pin into the iliac wing problematic.

A 'dynamic', transarticular ESF kit is now available for the management of animals with hip luxations (Veterinary Instrumentation). Fixation pins are placed into the greater trochanter, the iliac shaft and the ischium. These are then joined with connecting bars that incorporate a 'swivel' clamp which allows flexion and extension of the hip but prevents abduction/adduction and also lateral displacement of the femoral head, thus precluding reluxation (Fig. 40.8). A similar technique has been described by McLaughlin & Tillson (1994) using two Ellis pins joined by a flexible band to create a flexible external fixator.

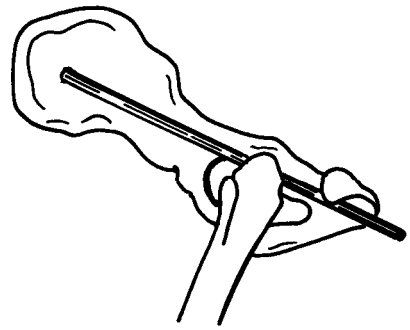


Fig. 40.7 Illustration of the positioning for a DeVita pin.

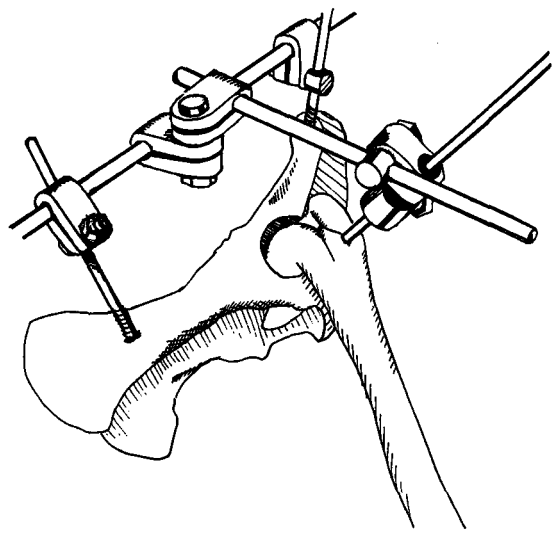


Fig. 40.8 Illustration of a 'dynamic', transarticular external skeletal fixator (ESF) used to stabilise a hip luxation (Veterinary Instrumentation).

Open reduction and suturing of the capsule

The animal is positioned in lateral recumbency with the affected limb uppermost. Before final preparation of the surgical field a 'groin sling', made of a calving or lambing rope, is positioned which allows traction to be applied to the limb during surgery (Fig. 40.9). A craniolateral approach is used (Fig. 40.10) to expose the joint. A skin incision is made just cranial to the greater trochanter if the hip has been reduced, or over it if not, extending halfway down the femur. The fascia lata is incised close to its attachment to the biceps femoris and the incision is extended prox-

imally through the gluteal fascia. The middle gluteal muscle is elevated to expose the tendon of insertion of the deep gluteal muscle and the cranial half of this is incised. Dorsal retraction of the gluteals and caudal retraction of the vastus lateralis exposes the torn joint capsule and femoral head. The acetabulum is located ventrally and is most easily exposed by caudal retraction of the proximal femur. Haematoma and remnants of teres ligament (especially if there is an avulsed fragment of bone attached to its free end) are removed from the acetabulum. More often than not, in cases with recent luxation, a healthy mass of dorsal joint capsule, which has retained its attachments to the acetabular rim, is found lying in the acetabulum and should be preserved. It is likely that the very presence of this capsule in the acetabulum is the cause of many closed reductions being unstable. The femoral head is most easily reduced into the acetabulum by grasping the greater trochanter with pointed reduction forceps and using these as a 'handle'. The preserved joint capsule can then be held retracted whilst the femoral head is reduced. After manipulation of the joint, to express any remaining haematoma, the capsular remnants are drawn over the femoral head and sutured to either capsular remnants on the femoral head or, more commonly, the insertions of the gluteal muscles using horizontal mattress or cruciate sutures of polydioxanone (PDS, Ethicon).

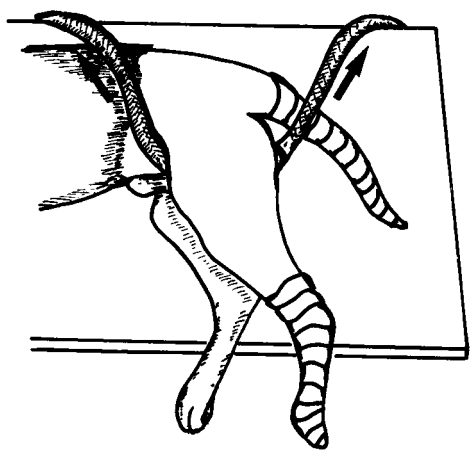


Fig. 40.9 Illustration of a 'groin sling' used when reducing a luxated hip, performing surgery on the hip joint, or during open reduction and fixation of a hindlimb long bone fracture, to allow traction to be applied to the limb.

If stability is good then wound closure may be carried out +/– the application of an Ehmer sling, followed by radiography to document satisfactory reduction. More often than not, however, if open reduction has been carried out then the surgeon will prefer to ensure joint stability during healing of the soft tissues by means of one of the techniques described below. Exercise is restricted for 1 month and then gradually returned to normal over the second month.

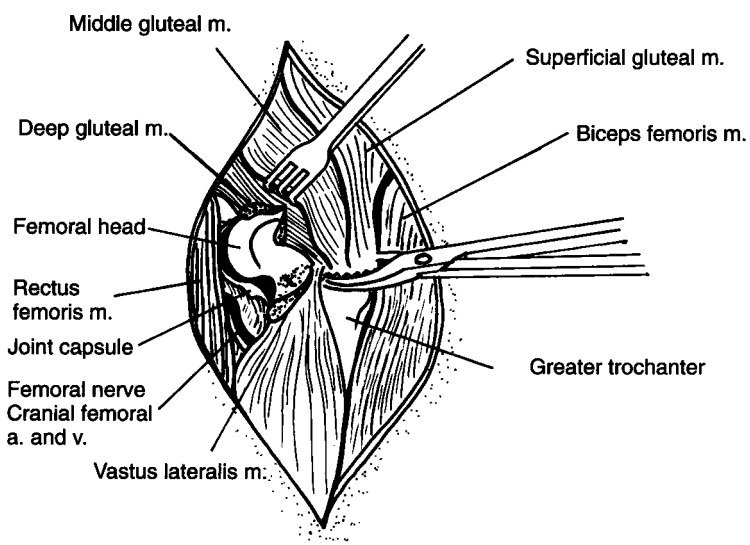


Fig. 40.10 Illustration of a craniolateral approach to the hip joint.

'Anchor' suture (prearticular or iliofemoral suture) (based on the technique described by Meij *et al.*, 1992)

A craniolateral approach to the hip is used to reduce the luxation and to suture any salvageable joint capsule as described above. Tunnels are then drilled through the caudoventral part of the iliac shaft, just in front of the acetabulum, and through the greater trochanter in a craniocaudal direction (Fig. 40.11). The 'anchor' suture is then placed through these tunnels and tied whilst the hip is inwardly rotated (Fig. 40.12 – for simplicity the suture is shown as a 'loop' but can also be placed as a figure-of-eight). Leader line or braided nylon (Ethibond, Ethicon) are suitable materials for the suture. In the case of Leader line a single strand of 70 lb line is used in dogs less than 15 kg bodyweight, a single strand of 80 lb line is used in dogs of 15–25 kg bodyweight and a double strand of 80 lb line is used in dogs of 25–40 kg bodyweight. In the case of a single-strand suture the ends are passed through the greater trochanteric tunnel in opposite directions and tied on its lateral aspect, and the knot may have to be sutured down to prevent local irritation to the overlying soft tissues. In the case of a double-strand suture, it is easiest to pass the free ends through the greater trochanteric tunnels and back under the gluteal insertions forming either a loop (as shown in the diagram) or a figure-of-eight. The free ends may then be tied through the looped end using a self-locking knot as described in Chapter 42 when considering lateral retinacular sutures for the treatment of cranial cruciate ligament failure (p. 540). The remainder of the closure is routine and no external support is used after radiography to ensure adequate reduction. Exercise is restricted for 1 month and then gradually returned to normal over the second month. Postoperatively the 'anchor' suture acts in a similar manner to an Ehmer sling and holds the hip inwardly rotated. The animals tend to show an awkward gait for a few weeks and then, often suddenly, their gait returns to normal (presumably when the suture breaks).

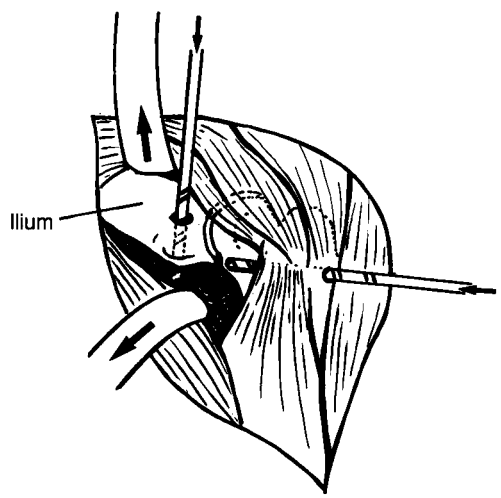


Fig. 40.11 Illustration of the exposure for the ilium and drilling of the bone tunnels required for placement of an 'anchor' suture to stabilise a luxated hip joint.

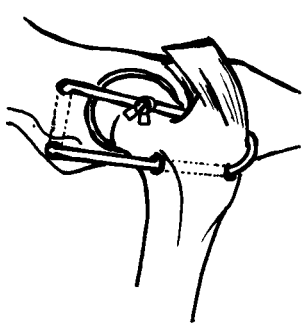


Fig. 40.12 Illustration of an 'anchor', prearticular or iliofemoral suture used to stabilise a luxated hip joint.

In small dogs or cats it is sufficient to place the suture around the tendon of origin of the iliopsoas muscle and through the tendon of insertion of the middle gluteal, as described by Mehl (1988).

Toggle fixation (based on the technique first described by Knowles *et al.*, 1953)

This technique involves replacement of the teres ligament with fascia (Knowles *et al.*, 1953), braided nylon (Lawson, 1965; Denny & Minter, 1973), stainless steel wire or a heavy plastic suture (Leonard, 1971) or skin (Zakiewicz, 1967). Some aspects of the biomechanics relating to toggle pin fixation have been investigated by Flynn *et al.* (1994) including the design and orientation of the toggle itself.

A caudodorsal approach is made and this is facilitated by the fact that the gemelli and obturator muscles, which insert on the caudal aspect of the femoral neck, are usually torn when the femoral head luxates. A skin incision is made directly over the greater trochanter and continued distally to halfway down the femur (Fig. 40.13a). The fascia lata is incised along its border with the biceps femoris and the latter is retracted caudally to reveal the greater trochanter. The sciatic nerve is identified caudal to the femoral shaft in the loose fascia between the biceps femoris and semimembranosus muscle (Fig. 40.13b). The nerve is carefully protected while exposure of the acetabulum is completed from the caudal aspect. The insertion of the superficial gluteal muscle is transected and the caudal muscles of the hip (gemelli and obturators), if not already ruptured, are transected close to their insertion on the proximal femur (Fig. 40.13c). Reflection of these muscles and cranial retraction of the femoral head will allow exposure of the acetabulum which is then cleared of haematoma and/or granulation tissue. A tunnel is drilled through the acetabular fossa using a $\frac{3}{16}''$ drill bit (Fig. 40.14a). The toggle to be used will have been made beforehand and, in its simplest form, comprises a piece of 1.6mm Kirschner wire, into which is fashioned a loop, and a double strand of 7 metric braided nylon (Ethibond, Ethicon) (Fig. 40.14b). (In small patients a 1.1mm K-wire and single strand of 7 metric Ethibond should suffice.) The toggle is guided into the acetabular tunnel with artery forceps and then pushed through into the pelvic canal using the blunt end of a smaller drill bit. Traction is applied to the toggle with the drill bit in place to begin with, so that it rotates and engages on the medial aspect of the acetabulum (Fig. 40.14c). The femoral head is rotated caudolaterally and a Hohmann retractor used to elevate it through the surgical incision. Remnants of teres ligament are removed and a tunnel is drilled retrogradely from the fovea and down the femoral neck, to exit the femur just ventral to the greater trochanter, using a $\frac{7}{64}''$ drill bit (Fig. 40.14d). A second tunnel is drilled transversely across the greater trochanter. A wire loop is used to draw the ends of the nylon through the tunnel in the femoral neck. Traction is maintained on the nylon whilst the femoral head is reduced. One end of the nylon is drawn

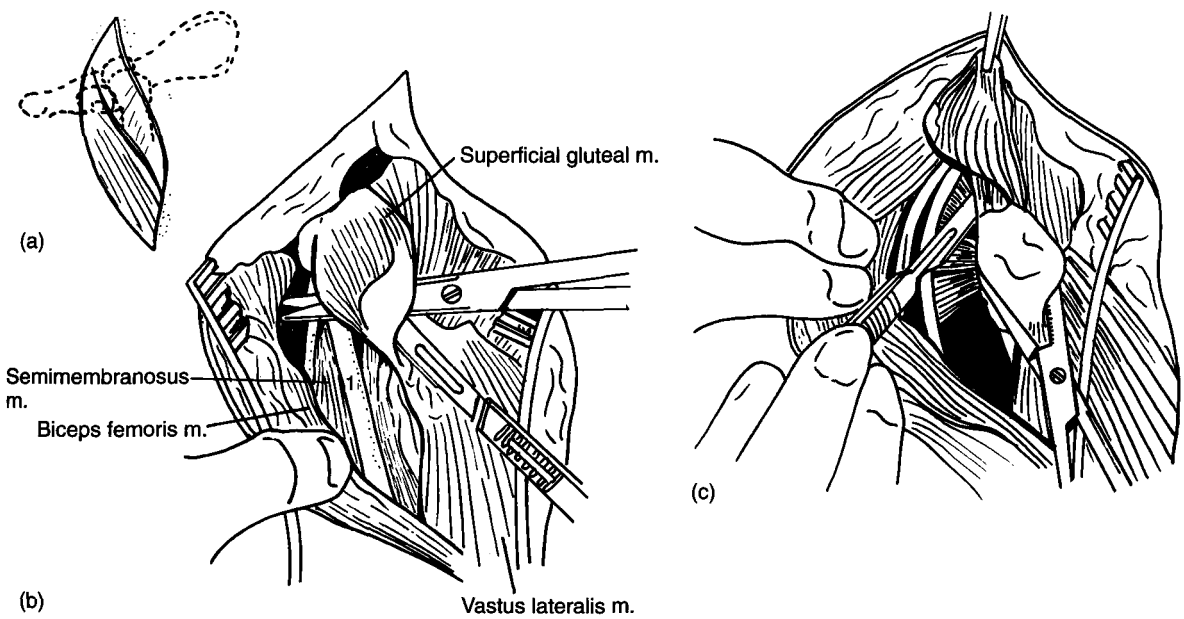


Fig. 40.13 Illustration of the caudodorsal approach to the hip used for toggle fixation of a hip luxation. (a) Skin incision. (b) Sciatic nerve (1) identified. (c) Transection of the superficial gluteal muscle +/– the caudal muscles of the hip.

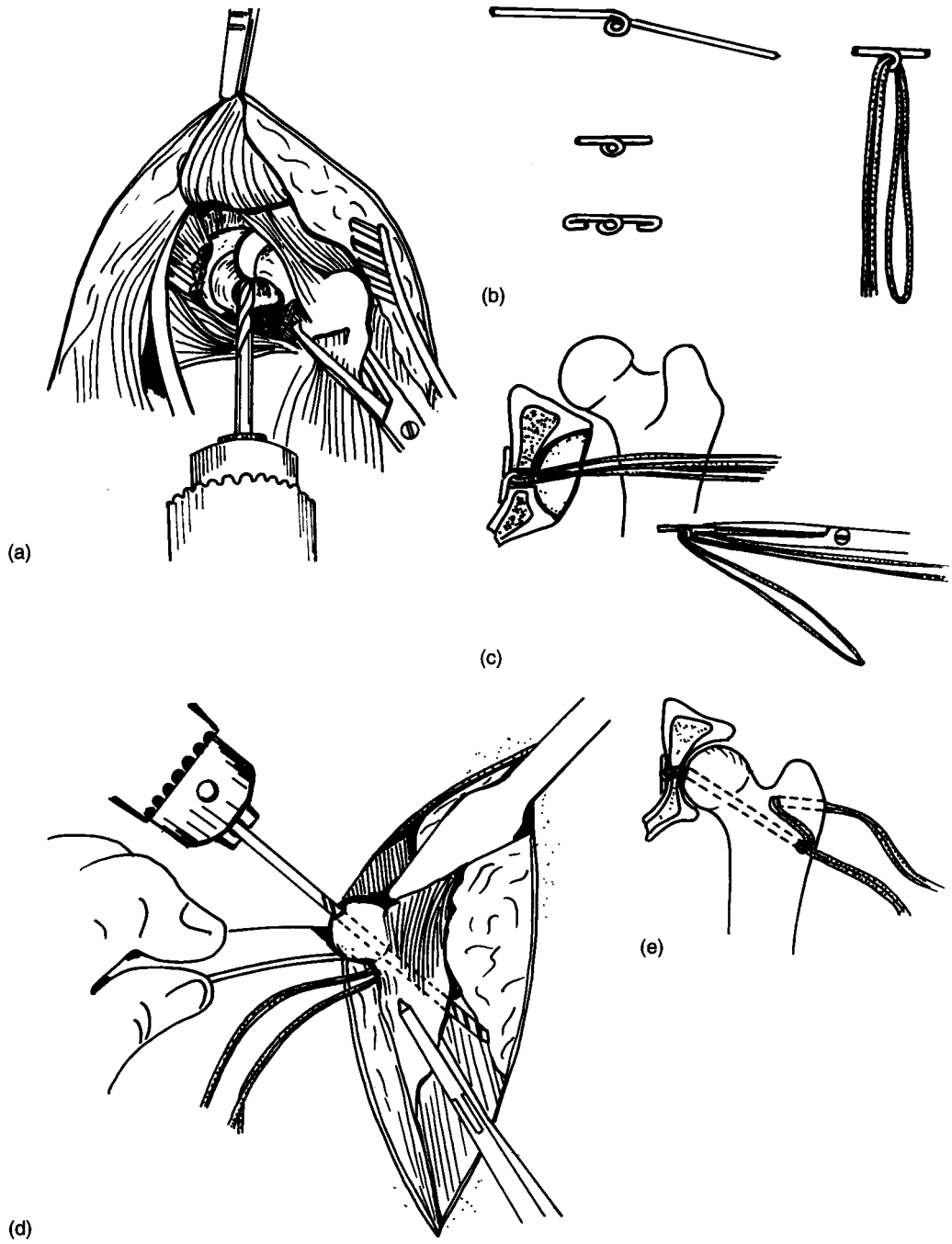


Fig. 40.14 Illustration of toggle fixation in the treatment of hip luxation. (a) A tunnel is drilled through the acetabular fossa. (b) A toggle can be made by fashioning a loop in a Kirschner wire (usually a 1.6 mm wire) and placing a double strand of 7 metric Ethibond (Ethicon) through this. (c) The toggle is pushed through the acetabular tunnel and then engages the medial wall of the pelvis as traction is applied. (d) A tunnel is drilled through the femoral head and neck. (e) After the nylon is drawn through the femoral tunnel with a wire loop, one end is passed through a second tunnel made through the greater trochanter and the ends are tied.

through the trochanteric tunnel, using a second wire loop, and the two ends are tied securely on the lateral aspect of the trochanter (Fig. 40.14e). Reduction and stability of the hip is evaluated by manipulation before the suture ends are cut. Closure is routine, starting with reattachment of the superficial gluteal muscle since the deeper muscles, caudal to the hip, are inaccessible. Radiography to confirm adequate reduction is carried out. An Ehmer sling is applied for 5 days and exercise restricted for 1 month, followed by a gradual return to normal over the second month.

Transarticular pin (Bennett & Duff, 1980; Hunt & Henry, 1985)

Placement of a Steinmann pin, or Kirschner wire in small patients, across the articular surfaces of the hip can be performed in a closed manner (i.e. through a stab incision after closed reduction) or, more accurately, in combination with open reduction. It is important that the pin exits the femoral head, at the point of insertion of the teres ligament (i.e. the fovea) and this is best ensured by use of a C-guide or by retrograde drilling of a pilot hole with a slightly smaller drill or pin than the one to be used. After reduction of the femoral head, the pin is driven across the joint space whilst the limb is held in the sagittal plane. The pin penetrates the acetabular fossa and is advanced until the whole of the trochar point and 2 or 3 mm of pin are protruding medially (most easily determined by use of a gloved finger of an unscrubbed assistant) (Fig. 40.15). The pin may then be cut close to the lateral aspect of the femur or bent and cut to produce a hooked end. The latter is more likely to prevent migration but has to be done with care to prevent the pin being drawn back as it is bent, resulting in loss of purchase in the acetabulum. The pin is removed after 4–6 weeks, once soft tissue healing has taken place. Whilst the pin is *in situ* the hip is able to flex and extend through a reasonable range but abduction/adduction is prevented. With time the range of motion increases, in all directions, presumably due to resorption of bone around the pin penetrating the acetabulum.

This technique can produce good results but it does require a second surgical procedure to remove the pin and complications arise:

- If the pin exits the femoral head other than at the fovea, as it will often still penetrate the acetabular fossa but in doing so creates a subluxation
- If the pin migrates, allowing reluxation
- If the pin breaks, making removal of the distal end very difficult, though this is often not necessary
- If the pin bends, making its removal very problematic

Dorsal capsulorrhaphy (Allen & Chambers, 1986)

A dorsal approach (see Chapter 39, p. 450), can be used to reduce the luxation and suture the dorsal capsule or create a capsulorrhaphy if the dorsal capsule is too severely torn to hold sutures adequately. Two bone screws with spiked washers are placed dorsal to the acetabulum at 10 and 1 o'clock, in the case of the left hip, or 11 and 2 o'clock, in the case of the right hip. A third screw and washer can be placed in the intertrochanteric fossa of the femur or, more usually, a bone tunnel is drilled in a craniocaudal direction through the dorsal part of the femoral neck. One or two sutures of 7 metric braided nylon (Ethibond, Ethicon) or 70lb Leader line are then placed through the femoral tunnel and around each of

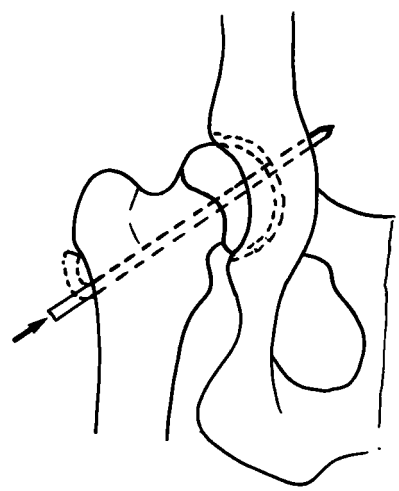


Fig. 40.15 Illustration of placement of a transarticular pin used to stabilise a hip luxation (see text for further details).

the screws and washers in a figure-of-eight pattern. The sutures are tied with the limb held in mild abduction after any remnants of joint capsule have been closed (Fig. 40.16). Closure is routine but if a trochanteric osteotomy has been used as part of the dorsal approach (rather than gluteal tenotomy) then the trochanter may be transposed during closure (see below). Postoperative care is as described above.

This technique generally produces good results. Its main drawbacks are the requirement for a dorsal approach to the joint and that if the femoral head were to reluxate then the presence of the screws dorsal to the acetabulum would be likely to cause significant damage to the femoral head in a fairly short space of time.

Transposition of the greater trochanter (DeAngelis & Prata, 1973)

If a dorsal approach, including osteotomy of the greater trochanter, is used to reduce a hip luxation then during closure the trochanter can be relocated 2–3 cm caudodistal to its original position (Fig. 40.17). This procedure places the gluteal muscles under increased tension, forcing the femoral head into the acetabulum and thus helps protect the repair during healing. This technique is, generally, not relied upon as a single-treatment entity, but if open reduction has involved trochanteric osteotomy then its transposition during closure would seem beneficial.

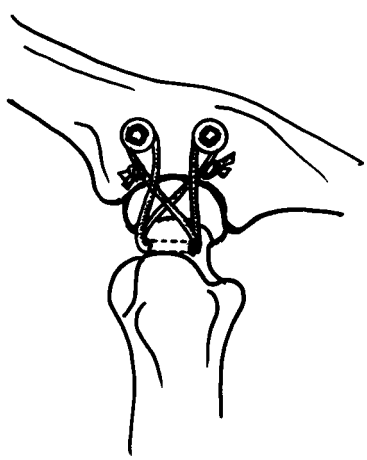


Fig. 40.16 Illustration of a dorsal capsulorrhaphy to treat a left hip luxation. Two screws with spiked washers are placed at 10 and 1 o'clock around the dorsal acetabular rim (11 and 2 o'clock for a right hip) and a tunnel is drilled through the femoral neck, dorsally. Figure-of-eight sutures are then placed through the tunnel and around each of the screws. The sutures are tied with the limb held slightly abducted. (See text for further details.)

Other techniques

Other surgical techniques sometimes used in the management of animals with hip luxation include:

- Triple pelvic osteotomy
- Total hip replacement
- Excision arthroplasty

Triple pelvic osteotomy This may be utilised in cases showing inherent instability due to hip

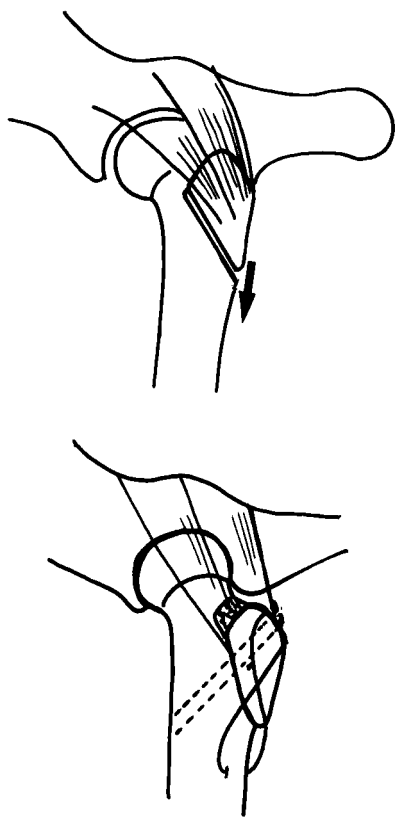


Fig. 40.17 Illustration of transposition of the greater trochanter used to stabilise a hip luxation.

dysplasia (Murphy *et al.*, 1997a) such that the standard techniques detailed above would not allow sufficient soft tissue healing to stabilise the joint. This procedure is discussed in more detail under 'Hip dysplasia' later in this chapter.

Total hip replacement This may be utilised in cases with recurrent luxation following standard methods of treatment (as detailed above) and significant evidence of secondary osteoarthritis that might preclude successful use of techniques such as triple pelvic osteotomy. This procedure is discussed in more detail under 'Osteoarthritis' later in this chapter.

Excision arthroplasty This may be utilised in cases with recurrent luxation following standard methods of treatment (as detailed above) and where triple pelvic osteotomy or total hip replacement cannot be considered. It may also be resorted to in cases where the luxation is associated with fracture of the femoral head and internal fixation is not considered feasible. This procedure is discussed in more detail under 'Legg Perthes' disease' below.

In the main, a dog will gain better hindlimb function after an excision arthroplasty compared to being left with an untreated hip luxation. However, due to a difference in the shape of their femur and pelvis, this is not necessarily true of the cat and there is some evidence to suggest that these patients will become as functional with a luxated hip as they will after an excision arthroplasty (Pérez-Aparicio & Fjeld, 1993). Thus, if closed reduction fails and internal stabilisation is not being considered, the clinician would be as well to leave the luxation and consider excision arthroplasty if limb function is unsatisfactory after 4–8 weeks rather than considering this as a first-line treatment.

Prognosis

With appropriate management the prognosis is generally good in uncomplicated cases, and although there will often be radiographic evidence of osteoarthritis at a later date, it is the exception rather than the rule that they should suffer clinically with this secondary pathology. The greatest problem is that of reluxation which appears to occur in about 40–50% of closed reductions and in up to 10–15% of cases treated with open reduction and internal stabilisation.

Legg Perthes' disease

Legg Perthes' disease is also referred to as Calvé Legg Perthes' disease, Perthes' disease, ischaemic/avascular necrosis of the femoral head, coxa plana or osteochondrosis juvenalis. It constitutes an aseptic necrosis of the femoral head and affects young dogs of mainly small breeds. The most effective treatment involves a femoral head and neck excision arthroplasty. In some breeds the disease has been shown to have an inherited basis, consistent with an autosomal recessive gene (Robinson, 1992) and so clinically affected dogs should not be used for breeding and, at the very least, the mating that produced an affected dog should not be repeated.

Aetiopathogenesis

Apart from there being evidence to support a genetic basis for the disease, the cause is largely unknown. Although various factors have been suggested, such as in imbalance of sex hormones, which may have some experimental evidence to support them, there are no studies confirming their relevance to the clinical disease. The fact that the femoral head necrosis appears to be avascular in nature and that the disease is seen almost exclusively in terrier breeds, has led to various hypotheses regarding an inadequate blood supply to the femoral head. It has been suggested that in the smaller breeds the vessels in the teres ligament or the joint capsule may be of a diameter that is inadequate to resist injury/thrombosis as a result of normal 'wear and tear'. It has also been proposed that in these breeds there are no vessels crossing the actual physis whereas in the larger breeds there may be some transphyseal vasculature.

Histologically, the femoral head and neck undergo ischaemic necrosis followed by trabecular collapse (Lee, 1970). This may lead to ineffective subchondral support causing the articular cartilage to implode which results in articular incongruency and joint widening. The latter

effectively reduces the joint's stability and leads to secondary osteoarthritis. The ischaemic bone is eventually replaced with granulation tissue by creeping substitution. Pain may result from the initial ischaemia but would be expected to become more marked if joint incongruency developed as a result of collapse of the articular cartilage.

History

Progressive hindlimb lameness is seen in dogs from about 5 months of age. If the problem is bilateral then a shifting lameness may be seen or else the dog may adopt a crouched stance and bunny-hopping gait. The breeds reported to have been affected include:

- Miniature Poodle (? Robinson, 1992)
- Toy Poodle (* Pidduck & Webbon, 1978)
- West Highland White Terrier (* Wallin, 1986; Robinson, 1992)
- Cairn Terrier
- Yorkshire Terrier (* Robinson, 1992)
- Manchester Terrier (* Vasseur *et al.*, 1989)
- Lakeland Terrier
- Miniature Pinscher
- Pug (? Robinson, 1992)

*indicates that heretability is proven for the condition in this breed and ? indicates that evidence is strongly suggestive of the condition being heritable in this breed.

Clinical examination

The lameness is associated with pain on manipulation of the hip, particularly extension or abduction. The presence of crepitation is usually evidence of joint incongruency and a reduced range of motion indicative of periarticular fibrosis. Wastage of the gluteal muscles may become apparent as the disease becomes chronic. This may cause the greater trochanter to become more prominant, possibly leading to a misdiagnosis of luxation.

When examining these patients, it is important to rule out other orthopaedic diseases that may cause similar signs, such as slipping of the proximal femoral epiphysis or patellar luxation.

Radiology

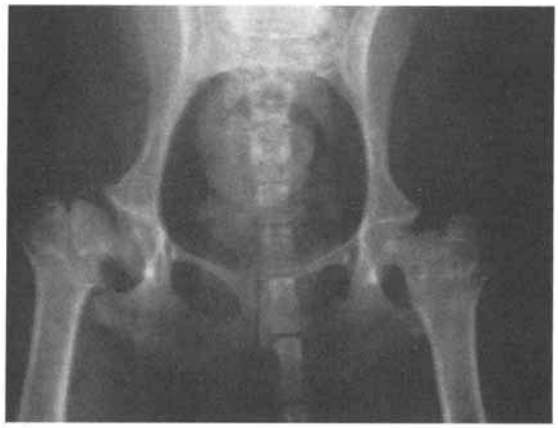
A ventrodorsal view of the pelvis should normally suffice to confirm the clinical diagnosis. Initially, foci of reduced radiodensity within the femoral head and neck result from the ischaemic necrosis. As trabecular collapse occurs, areas of increased density may appear and, with collapse of the articular cartilage, the outline of the femoral head will alter causing widening of the joint space (Fig. 40.18). Such joint incongruency may then become associated with secondary changes such as periarticular osteophytes on the femoral neck and acetabular rim and in-filling of the acetabular fossa. A restriction in extension of the hip may create foreshortening of the femur in the affected limb.

The rate at which these changes will appear is unpredictable as is when the dog will first start to show signs. If radiographs are taken at the time of first presentation then the changes seen may vary from virtually imperceptible to very advanced depending on the vigilance of the owner and what changes actually cause the joint to become painful to that individual.

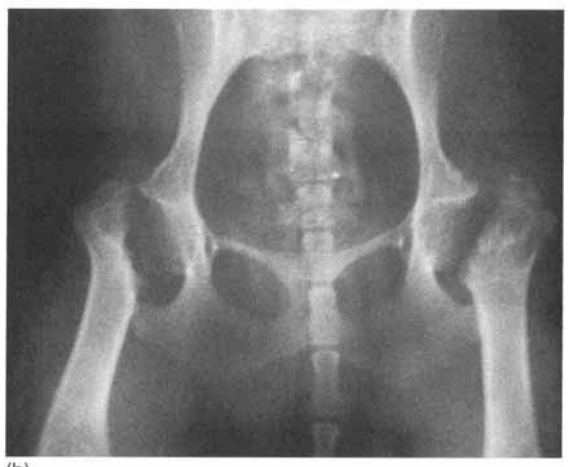
Treatment

Conservative management

Resting the dog for 4–8 weeks may allow the ischaemic areas of bone to be replaced with granulation tissue and the surrounding areas to remodel in order to accommodate the altered stresses through the femoral head. In the majority of cases the disease process will progress and the resulting articular incongruency cause continued lameness. This is reflected in the results of the study by Ljunggren (1967) in which only 15/62 (24%) dogs managed conservatively regained satisfactory joint function. As a result, conservative management cannot be recommended in any cases showing lameness associated with clinical crepitation, reduced range of motion, muscle wastage or radiographic evidence of articular collapse. It may be considered in early cases showing lameness but retaining a good range of joint motion and articular congruity. However, the progress of such cases should be monitored closely as it is likely that many will become surgical candidates.



(a)



(b)

Fig. 40.18 (a) Ventrodorsal radiograph of a 14-month-old West Highland White Terrier's pelvis showing deformity of both femoral heads, widening of the joint spaces and in-filling of the acetabula. The dog had shown lameness in the right hind as a puppy and was now intermittently lame on both hindlimbs. These radiographic features are considered pathognomonic for Legg Perthes' disease.
(b) Ventrodorsal radiograph of the same pelvis 6 weeks after excision arthroplasty on the left hip and immediately post-surgery on the right hip. Resection of the femoral head and neck is adequate bilaterally.

Surgical management

Femoral head and neck excision (FHNE) arthropasty gives the most rapid and predictable return to function. In the study by Ljunggren (1967), 33/39 (85%) cases were considered to respond favourably. Surgery should, therefore, be considered the management of choice in most cases. Femoral head and neck excision is best performed via a craniolateral approach (Fig. 40.10) as this maintains the hips' gluteal support and reduces the chance of iatrogenic femoral shaft fracture, whereas use of a dorsal approach carries both these disadvantages. The femoral neck is sectioned distally taking a line that is *not* perpendicular to the femoral neck but more aligned with the femoral shaft, either including or excluding the lesser trochanter (Fig. 40.19). It is important to also remember that the femoral neck is wider caudally than cranially and so the line of section must be more towards the sagittal plane than across the femoral neck, otherwise a caudal spur of bone will remain (Fig. 40.20). An osteotome or oscillating saw are the preferred instruments for the osteotomy. Although bone-cutting forceps or a Gigli wire can be used they both tend to cut through the femoral neck at its narrowest point, failing to follow the guidelines detailed in Figs 40.19 and 40.20 and thus requiring further resection using, for example, rongeurs.

Postoperative radiography to ensure satisfactory resection of the femoral head and neck has been achieved is mandatory.

Postoperative rehabilitation is very important and although rest is required until suture removal, thereafter the dog should be encouraged to use the limb. If good joint function is not returning by 4–6 weeks then stiffness will lead to reduced function which will lead to further fibrosis in a viscious, and negative, cycle. After 2 weeks the dog's

Fig. 40.19 Illustration of the line of osteotomy for resection of the femoral head and neck (excision arthroplasty) – cranial aspect.

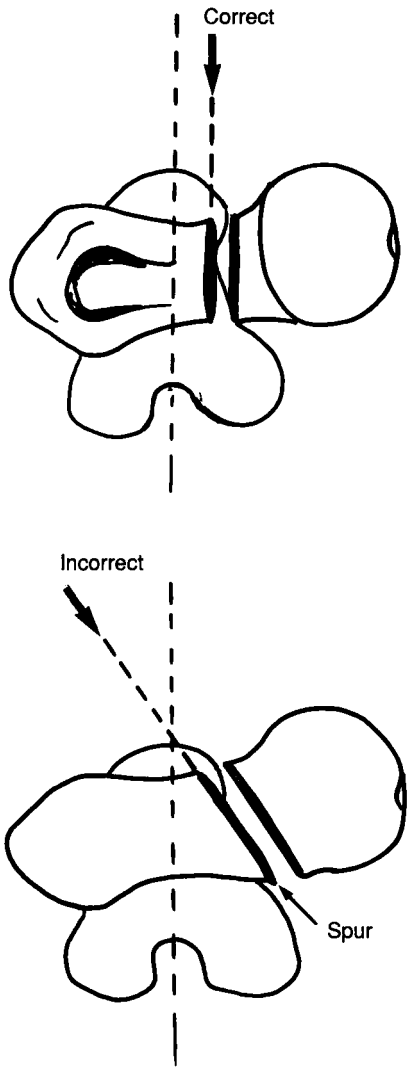


Fig. 40.20 Illustration of the line of osteotomy for resection of the femoral head and neck (excision arthroplasty) – dorsal aspect.

exercise should be increased but still restricted to lead walks as all four limbs are more likely to be used at the walk than when running. Repeatedly ascending flights of steps should be encouraged during exercise, again on the lead, and swimming should also be included where appropriate. Non-steroidal anti-inflammatory drugs (NSAIDs) may be administered to encourage limb use during this period whilst the pseudoarthrosis is developing.

If the problem is bilateral then there are arguments both for performing both procedures under the same anaesthetic and for separating the two by 4–8 weeks. Such a decision is down to the surgeon's preference and experience but even then each case merits consideration of both options. In general the authors tend to separate the two procedures.

Prognosis

The prognosis is generally good. Although most cases showing clinical lameness will require surgery to optimise their recovery, a good return to limb function can be expected following FHNE in dogs of these small breeds.

Hip dysplasia

Aetiopathogenesis

Hip dysplasia is a laxity of the joint which is seen in many breeds of dog. The larger breeds appear more susceptible to the development of clinical signs although even in these breeds, many dogs with radiographic signs will remain asymptomatic. The condition has also been noted in the cat, with pedigree animals, such as the Devon Rex, being more susceptible. Its presence may be an incidental finding but occasionally it will cause clinical signs.

There is good evidence that heritable factors predispose to the condition. The mode of inheritance is thought to be a polygenic dominant trait with incomplete penetrance. Few genes affect the osseous skeleton primarily. The changes in bone merely reflect changes that occur in the soft tissues (cartilage, connective tissue and muscle). There is little doubt that the environment acts in conjunction with the genotype in producing a phenotype. As a result factors such as nutrition, growth rate and trauma/exercise may influence the development of dysplasia.

Recently, there have been suggestions that the 'genetic flaw' may lie in the constitution of the synovial fluid. It has been hypothesised that a reduced concentration of hyaluronic acid results in decreased viscosity of the fluid. This, in turn, will

decrease the natural cohesion between articular surfaces and result in a lax joint. There is some evidence to support this claim but its significance is as yet unproven.

Affected animals have a normal hip at birth. During the first year of life, the skeleton grows more rapidly than the supporting tissue masses. As a result, the soft tissues do not have sufficient strength to maintain congruity between the articular surfaces of the femoral head and the acetabulum. Because of this, the femoral head and acetabulum pull apart and trigger a series of events that end in hip dysplasia and osteoarthritis.

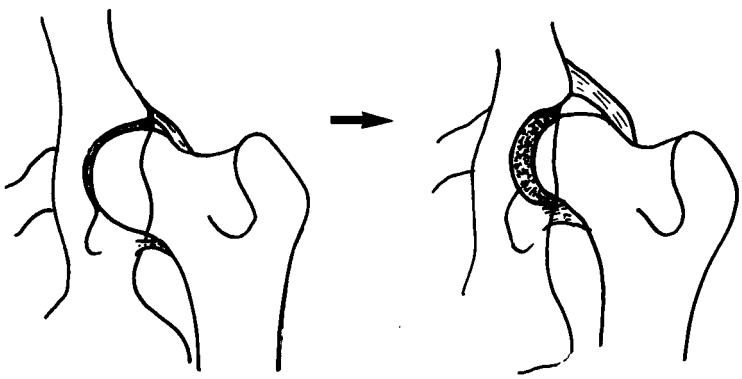
The pathology has been studied in detail by Riser (1973) who recorded the changes seen during the first year of life (Fig. 40.21):

0–2 months:

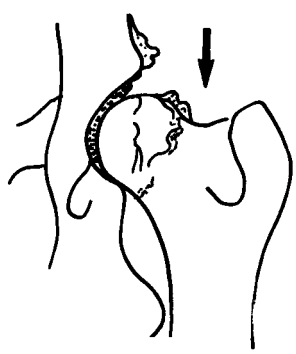
- The teres ligament and joint capsule stretches
- The craniodorsal acetabular rim becomes less sharp
- The femoral head subluxates

2–5 months:

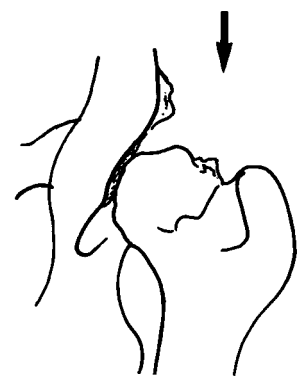
- The femoral head and neck become more subluxated
- The teres ligament and joint capsule becomes thickened
- The craniodorsal acetabular rim becomes more rounded
- Chondromalacia, due to abnormal stresses, becomes evident on the dorsal surface of the femoral head and acetabular rim



(a) Inflammatory phase. Laxity of the hip joint causes abnormal stresses in the supporting structures which leads to inflammtory changes (synovitis) causing an increased production of synovial fluid and thickening of the joint capsule.



(b) Remodelling phase. This includes erosion of the acetabular borders and new bone formation (osteophytosis) within the acetabular fossa, on the acetabular borders and around the femoral neck.



(c) Stabilisation phase. By 12–15 months of age the joint will usually be stable by virtue of: capsular thickening, acetabular and femoral head remodelling (i.e. osteoarthritic change), and improved muscular support.

Fig. 40.21 Illustration of the pathogenesis of hip dysplasia.

- The greater trochanter becomes bent over medially as a result of abnormal forces from the gluteals caused by the femoral head resting on the dorsal acetabular rim which causes lateral displacement of the femoral head
- Remodelling of the femoral head and acetabulum:
  - the excessive overloading of the acetabular rim stretches the plastic cartilage beyond its fatigue limit, causing a rounded rim
  - the rest of the acetabulum is bone and the change in loading results in new bone being deposited on the dorsal acetabular surface and resorbed from the ventral surface of the acetabular cavity
  - the femoral epiphysis changes its position on the physis – termed 'drift' this is achieved by two mechanisms:
    (1) *plastic bending* of the physis to try and keep the lines of force through it parallel to the long axis of the endochondral cartilage columns
    (2) *osseous drift* of the femoral head due to compression being unequal over the femoral head. More load is applied dorsally and as a result bone is laid down on the dorsal aspect of the femoral neck and resorbed from the ventral aspect. This stage involves remodelling secondary to abnormal loading and causes the femoral head and neck to bend downwards

5–9 months:

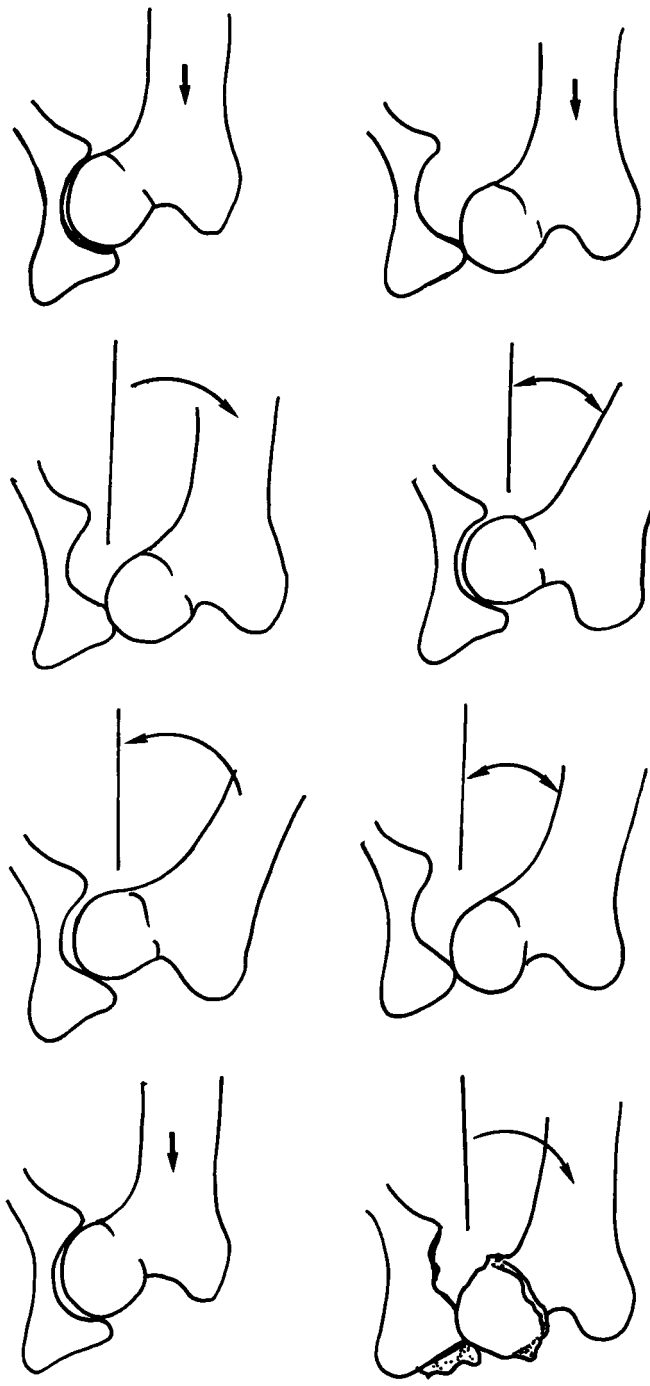
- The acetabular rim becomes eburnated (following chondromalacia and microfractures) due to continued overloading
- The joint capsule becomes grossly thickened with new bone at the points of attachment (which would probably restrict the range of movement in the joint)
- The acetabular fossa becomes filled with new bone thus becoming shallower
- Marginal lipping of the femoral head with new bone occurs, i.e. osteoarthritis begins to establish itself at this stage

History and clinical signs

Dogs presented with clinical problems tend to fall into one of two distinct groups, those that are suffering with hip dysplasia per se and those that are suffering with coxofemoral osteoarthritis secondary to dysplasia. The latter will be discussed later in this chapter under 'Osteoarthritis'. Dogs showing clinical signs associated with hip dysplasia will present between 4 and 10 months of age with a history of stiffness on rising, exercise intolerance and/or lameness that is made worse by exercise, with stiffness after rest following exercise. The owner may report that the dog cries out when turning sharply.

On examination, lameness of varying severity or a stiff hindlimb gait may be noted and, if the problem is bilateral, the dog may have a tendency to bunny-hop at faster paces. Poor hindquarter muscling is usually present and this may reflect a combination of poor development and atrophy. Pain is present on manipulation of the hip(s) and a reduced range of motion will be noted in most due to pain or, in chronic cases, capsular fibrosis.

Hip joint laxity can be assessed by one of several methods but the one most commonly used is the *Ortolani test* (Fig. 40.22). This involves placing the dog in dorsal recumbency (some clinicians prefer to have the patient in lateral recumbency, but the principle is the same) with the hips and stifles flexed such that the femoral shafts are perpendicular to the surface on which the dog is lying. Downward pressure is then applied to the stifles so as to subluxate the hips. The femurs are then gradually abducted. If the femoral head is suddenly felt to relocate in the acetabulum then the test is positive. The angle at which this occurs is termed the Ortolani angle, or the angle of reduction. If the femur is then gradually adducted whilst downward pressure is maintained then the femoral head will be felt to disengage from the acetabulum, back into its subluxated position. The angle at which this occurs is referred to as Barlow's angle, or the angle of reluxation. An Ortolani-negative patient has little laxity in the joints. This may be because they have good hip conformation or else they have dysplasia with marked secondary changes such that reduction of the joint(s) is no longer possible. The character of the Ortolani test is also of importance since it relays information about the quality of the dorsal acetabular rim. If the reduction is accompanied by a 'click' or 'clunking' sensation then the integrity



(a) With the dog in dorsal recumbancy and the femurs held perpendicular to the table downward pressure is applied to both stifles to try and subluxate the femoral head. This downward pressure is maintained throughout the test.

(b) The femur is then abducted until the femoral head is felt to reduce into the acetabulum and the 'angle of reduction' noted.

(c) The femur is then adducted until the femoral head resubluxates and the angle of subluxation noted.

(d) The Ortolani test is negative when the hip is stable, either because it is normal or because there are advanced secondary changes affecting the joint, or when the joint is severely dysplastic and cannot be reduced.

Fig. 40.22 Illustration of the Ortolani test (showing left hip in the test when conducted with the dog in dorsal recumbency).

of the dorsal rim is probably good, whilst if it feels as if the femoral head 'slides' into the acetabulum then the rim has probably undergone marked deformation and remodelling. This can be useful information when it comes to planning treatment.

Radiology

The changes observed radiographically can be divided into those that are associated with the dysplasia itself and those that result from secondary changes. The most useful single view is a ventrodorsal projection with the hindlimbs fully extended, the femurs parallel to one another and the patellae lying dorsally (i.e. the position used for the BVA/KC Hip Dysplasia Scoring Scheme discussed later, p. 482) (Fig. 40.23). Additional information may be gleaned from:

- A ventrodorsal, frog-legged view, as this provides an idea of how well the femoral heads can reduce into the acetabula (Fig. 40.24)
- A dorsal acetabular rim view (taken as a dorsoventral projection with the dog in sternal recumbency, the hindlimbs drawn forwards with the hocks raised off the table – Figs 40.25 and 40.26)
- A lateral view – especially if surgical treatment is being planned

The *primary changes* noted include:

- The centre of the femoral head lying on, or lateral to, the dorsal acetabular rim
- The joint space is wide with medial divergence of the cranial joint space

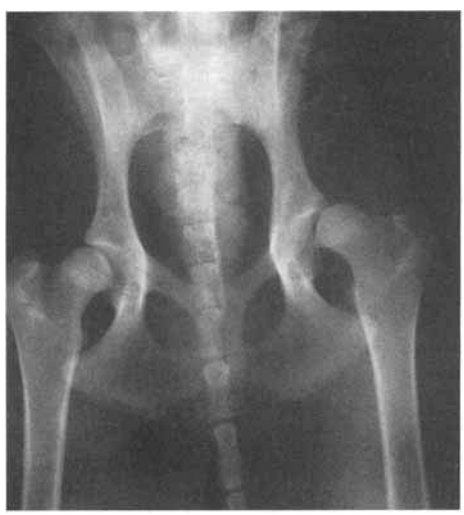


Fig. 40.23 A ventrodorsal radiograph of an 8-month-old Labrador Retriever's pelvis taken with the hindlimbs fully extended. Marked hip dysplasia is present bilaterally and the dog showed bilateral hindlimb lameness which was worst on the left (right hand side of picture).

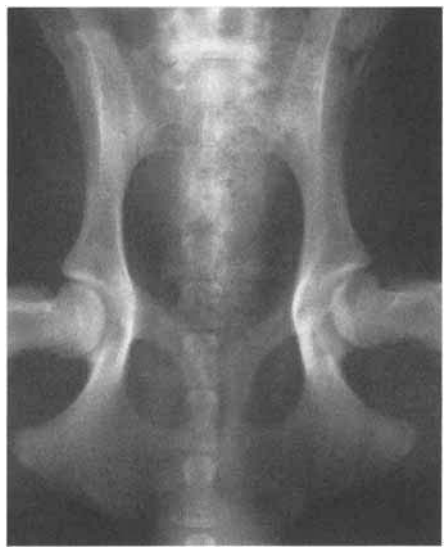


Fig. 40.24 Frog-legged, ventrodorsal views of the same dog as shown in Fig. 40.23 showing good reduction of the femoral head on the right and poor reduction on the left (right hand side of picture).

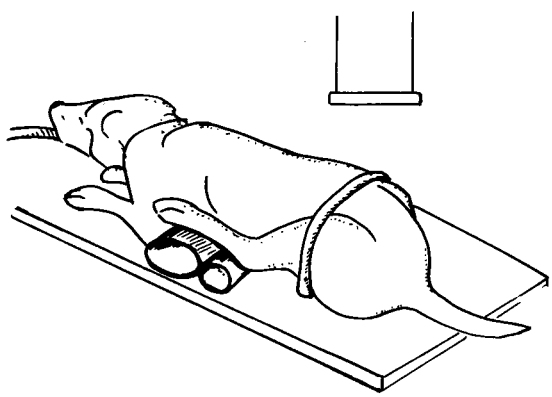


Fig. 40.25 Illustration of a dog positioned for radiography to produce an image of the dorsal acetabular rim.

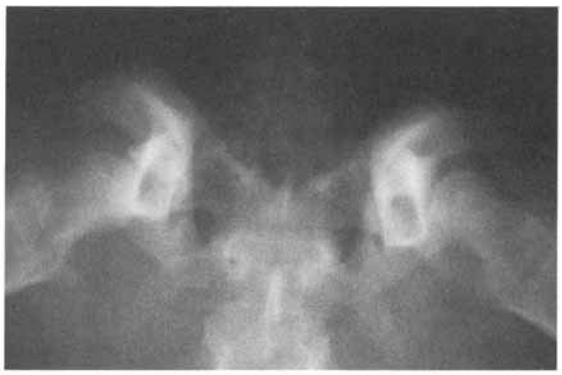


Fig. 40.26 Dorsal acetabular rim radiograph of the same dog as in Figs 40.23 and 40.24 showing mild remodelling of the rim on the right and severe remodelling on the left (right hand side of picture).

- Coxa valga (angle of greater than about 150° between the femoral neck and shaft)

The *secondary changes* include (see Fig. 40.33):

- New bone deposition around the femoral neck
- New bone deposition within the acetabular fossa
- Remodelling of the acetabular rim (especially the effective cranial acetabular rim) resulting in bilabiation and/or facet formation
- Osteophytosis along any of the acetabular rims

Based on these changes the degree of dysplasia can be classified into four groups:

(1) The centre of the femoral head lies on or just outside the dorsal acetabular rim with no, or minimal, secondary change
(2) The centre lies outside the dorsal acetabular rim, with between about 25 and 40% of the femoral head lying within the acetabulum, and minor secondary changes being present
(3) The centre lies well outside the dorsal acetabular rim, with less than 25% of the femoral head lying within the acetabulum, and/or moderate secondary changes being present
(4) Minimal or no contact being present between the femoral head and acetabulum and marked secondary changes being present

It must always be remembered that the radiographic appearance relates to the pathology present, to some extent, but does not necessarily correspond to the severity of the clinical signs noted.

Differential diagnosis

The differential diagnosis in a skeletally immature dog suspected of having signs associated with hip dysplasia would include:

- Metabolic disorders:
  - osteochondrosis (especially of the stifle or hock)
  - Legg Perthes' disease (smaller breeds)
  - panosteitis (especially German Shepherd Dogs)
  - metaphyseal osteopathy
  - sesamoid disease (plantar metatarsophalangeal)
- Traumatic injuries:
  - fractures of the proximal femoral physis
  - luxation

The differential diagnosis in the older patient is discussed later in this chapter under 'Osteoarthritis'.

Diagnosis

The diagnosis is based on the clinical history and clinical signs, with attention being paid to ruling out other causes, and is confirmed by radiography. It has to be stressed, however, that a diagnosis cannot be made from radiography alone. *Many dogs have radiographic changes consistent with hip dysplasia without showing any related clinical signs.*

Treatment

The methods of management available depend on whether the patient is being treated for hip dysplasia per se or osteoarthritis as a sequel to dysplasia. Again this tends to tie in with the dog being skeletally immature or mature, respectively. The management of hip osteoarthritis will be discussed later in this chapter.

Conservative management

The aim of this is to rest the dog so that awkward twisting and turning movements are avoided, which would lead to further distraction of the femoral head and acetabulum. As skeletal maturity is reached, muscle development will lend support to the hips. In addition, bone remodelling will slow down as the abnormal stresses become accommodated and the joint capsule will become thickened through fibrosis. As a result, there will be an increase in joint stability and a concurrent decrease in bone remodelling which will often lead to an improvement in clinical signs, though not radiographic changes.

The dog is restricted to lead exercise at all times (even when in the garden), and the length of walks should be determined by what does not aggravate the signs nor cause excessive stiffness after rest. The aim should be for the dog to have the same amount of exercise each day and not to overdo it on any one day (e.g. weekends!). This regime is continued until the dog is 12–15 months of age depending on progress. NSAIDs can be dispensed, if necessary, to allow comfortable rest, but should not be used to try and return the dog to 'normal' function.

The indications for conservative management are:

- When corrective osteotomies are not indicated
- When cost is a major factor

The results of such management have been reported as satisfactory in about 76% of cases followed up beyond 15 months of age (Barr *et al.*, 1987). Although not all of these were considered to have completely normal function or range of joint movement, and some showed pain on hip manipulation, the owners considered that the problem did not affect the dogs' quality of life.

Triple pelvic osteotomy (TPO)

This involves isolation of the acetabular segment of the pelvis by creating osteotomies in the pubis, ischium and ilium and, prior to fixation, rotating this segment so as to increase the joint's stability (Slocum & Devine, 1986). Rotating the acetabular segment in this way reduces the magnitude of the distractive forces acting on the hip joint (DeJardin *et al.*, 1996) and rotation of the segment through angles of up to 20° will increase the articular contact area (DeJardin *et al.*, 1998), though greater degrees of rotation appears not to further increase the area of contact. There are several variations on performing the osteotomies with some authors advocating removal of a portion of the pubis in order to avoid having a bony prominence projecting into the pelvic canal (Sukhiani *et al.*, 1994) and others suggesting osteotomy of the ischial body just caudal to the acetabulum so as to preserve the integrity of the sacrotuberous ligament (DàVid & Kasper, 1991). The shape, or angle, of the ileal osteotomy is also a debatable point. Overall, though, the aim is to free up the acetabular segment and rotate it over the femoral head. The degree of rotation is determined by the Ortolani and Barlow's angles. It must exceed the latter otherwise stable reduction will not be achieved and the nearer it is to the former the more stable the joint will be. It has been noted that it is not necessary to reach an angle of rotation close to the Ortolani angle and one that is somewhere between the two is normally sufficient, with an angle of 30° being most commonly used. During fixation of the ilium, the caudal fragment can be lateralised relative to the cranial fragment and this will cause anteversion of the acetabulum, thus increasing the articular contact. Fixation of the ilium can be achieved using a twisted dynamic compression plate (DCP) or a TPO plate (Veterinary Instrumentation) but if anteversion is to be achieved then a specially designed plate such as a Slocum Canine Osteotomy Plate (Slocum Enterprises, Oregon) is necessary (Fig. 40.27). In addition, some surgeons advocate stabilising the ilial osteotomy with orthopaedic wire. Although this does not appear to be mandatory it may lead to a quicker recovery.

The indications for corrective osteotomy include:

- Patients between 5 and 9 months of age (not exclusively)
- Clinical lameness (?)
- Little, or preferably no, secondary radiographic osteoarthritis

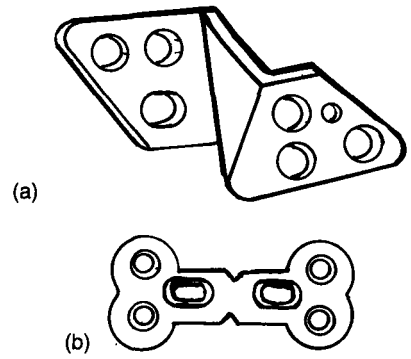


Fig. 40.27 Illustration of plates that may be used in triple pelvic osteotomy (TPO).
(a) Slocum Canine Pelvic Osteotomy Plate (Slocum Enterprises, Oregon). These are manufactured to create 20, 30 or 40° of rotation and also anteversion of the acetabulum (by transposing the caudal iliac shaft laterally). The plates are made to suit either the right or left hemipelvis.
(b) TPO plate (Veterinary Instrumentation). These plates are twisted before or during the surgery, according to what angle the clinician feels is required. They produce axial rotation but no anteversion of the acetabulum, are produced in two sizes and can be used on the left or right hemipelvis.

- Good integrity of the dorsal acetabular rim as assessed from the Ortolani test +/– a dorsal acetabular rim radiograph

Technique (Fig. 40.28) The first stage of this procedure is to create a *pubic osteotomy*. The dog is placed in lateral recumbency and the leg is raised by an assistant to allow the surgeon to make a standard approach to the pectineus muscle and pubic area of the pubis (Fig. 40.28a). This approach can also be made with the dog in dorsal recumbency which avoids the need for an assistant but does require that the dog be repositioned to allow the approaches for the other two osteotomies later in the procedure. The pectineus muscle is severed close to its origin on the iliopectineal eminence. The adductor muscle and a large branch of the deep femoral vein are retracted away from the pubic ramus and exposure is maintained with Hohmann retractors placed on either side of the ramus. The pubis is cut using an oscillating saw, a high-speed drill/burr, bone-cutting forceps or an osteotome either close to the acetabulum or else so as to remove a 1 cm section (so that a large spur of bone is not left projecting into the pelvic canal when the acetabular segment is rotated) (Fig. 40.28b). Care should be taken to protect the obturator nerve with a retractor while the osteotomy is performed. Closure of the wound includes reapposition of the fascia over the pectineus muscle, followed by the subcutaneous, subcuticular and cutaneous layers.

The second stage of the procedure is the creation of an *ischial osteotomy* by way of a caudal incision made directly over the tuber ischium. The ischial table is exposed by subperiosteal elevation of the internal and external obturator muscles (Fig. 40.28c). The osteotomy is most easily created by using a hacksaw blade starting dorsally as this requires minimal elevation of the muscles. Alternatively, a Gigli wire or embryotomy wire can be passed around the ischium via the obturator foramen and this can be used to cut the bone from cranial to caudal. Some surgeons prefer to stabilise the ischial osteotomy at the end of the procedure. If this is to be done then holes need to be placed on either side of the osteotomy and a piece of 18 gauge wire is preplaced for twisting after the iliac osteotomy has been created and stabilised. If such a wire is to be used then the ischial wound is covered with a moist swab. Ultimately the ischial wound is closed by apposition of the fascia over the tuber ischium followed by the subcuticular and cutaneous layers.

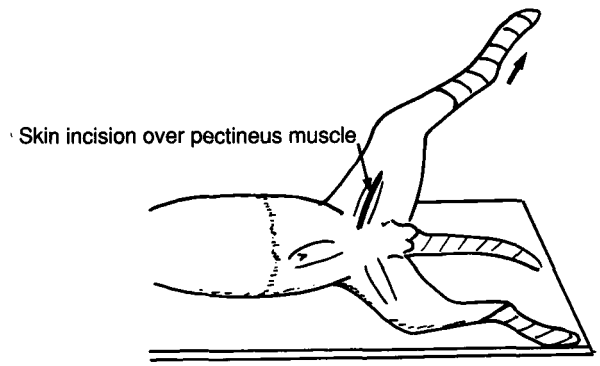
The third stage of the procedure is the creation of an *iliac osteotomy* via a lateral approach to the iliac shaft. A curved incision is made over the ilium and proximal femur (Fig. 40.28d). The tensor fascia lata is reflected ventrally and the middle and deep gluteal muscles are retracted dorsally, with the aid of a periosteal elevator and Hohmann retractors, to expose the iliac shaft (Fig. 40.28e). An oscillating saw is used to create a vertical osteotomy through the iliac shaft at the caudal margin of the sacrum, if a Slocum plate (Slocum Enterprises, Oregon) is to be used (Fig. 40.28f).

When alternative plates are used then the line of osteotomy is more perpendicular to the axis of the iliac shaft than vertical. A Hohmann retractor placed on the medial aspect of the ilium is used to protect the sciatic nerve from the saw. Before the osteotomy is complete it is helpful to prepare one of the screw holes in the caudal fragment as this is easier whilst the fragment is stable. The caudal part of the iliac shaft is then rotated with the aid

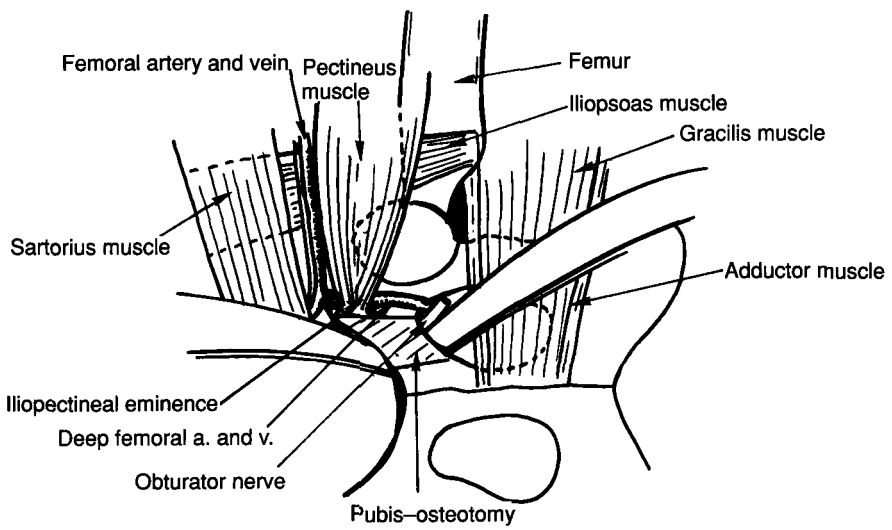
of bone-holding forceps. Particularly when a vertical osteotomy has been created, the dorsal edge of the caudal fragment forms a spur which projects into the gluteal musculature and at some stage this 'spike' is removed with the oscillating saw (and may be morcelised and packed around the osteotomy site as a bone graft). An appropriate plate (as discussed above) is then taken and first secured to the caudal fragment using the screw hole already prepared. If a Slocum plate (Slocum Enterprises, Oregon) is used then the next screw is placed in the cranial fragment through the plate hole that allows compression of the osteotomy to be achieved.

At this stage the hip joint stability can be evaluated by performing the Ortolani test. If, by chance, this is unsatisfactory then it may be possible to remove the plate at this stage and replace it with one incorporating a greater angle of rotation. The remaining screws are then placed with at least one of those cranial to the osteotomy passing into the sacral body for added security (Fig. 40.28g). The Slocum plate (Slocum Enterprises, Oregon) also has a hole caudally for placement of a cerclage wire through the plate and around the iliac shaft. Most surgeons do not make use of this feature, except perhaps in very large breeds of dog. Closure involves reapposition of the gluteal muscles with the tensor fascia lata followed by subcutaneous, subcuticular and cutaneous layers. If a wire has been preplaced in the ischium then this is now tightened and the caudal incision closed.

Postoperative care involves restriction to lead exercise and room rest for a period of about 8 weeks at which time follow-up radiography should be used to ensure healing of the osteotomies (in particular the iliac osteotomy)

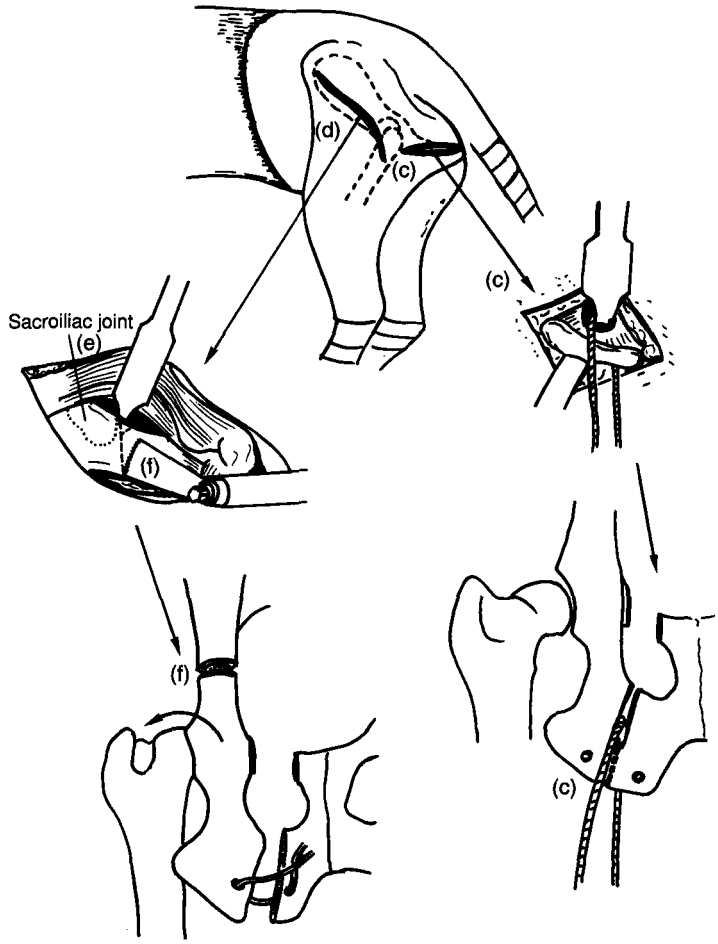


(a) The pectineus muscle is exposed by way of a ventral approach.

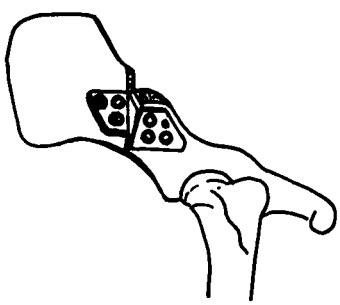


(b) The pubic ramus is either sectioned close to the acetabulum or else a segment is removed, to prevent creating a spike of bone in the pelvic canal once the acetabular segment is rotated.

Fig. 40.28 *Illustration of the steps involved in triple pelvic osteotomy (TPO). (See text for further details.)*



(c) The ischial table is exposed by a caudal skin incision and elevation of the obturator muscles. This is then cut using a saw or Gigli wire. A wire suture may be used to improve postoperative stability.
(d) A curved skin incision is made over the ilium and proximal femur.
(e) Dorsal elevation of the gluteal muscles exposes the iliac shaft.
(f) An oscillating saw is used to create an iliac osteotomy. This is made vertical and just caudal to the sacrum if a Slocum plate (Slocum Enterprises, Oregon) is being used, but more transversely for a TPO plate (Veterinary Instrumentation). A small triangle of bone may need to be removed to avoid a spike pressing into the gluteal muscles.



(g) A Slocum plate (Slocum Enterprises, Oregon) creates both axial rotation of the acetabular segment (according to the manufactured angle) and anteversion of the acetabulum by transposing the iliac shaft laterally.

Fig. 40.28 *Contd.*

before exercise is increased. In cases with bilateral clinical signs there is an argument for considering surgery on both sides close together. However, this creates a higher complication rate with respect to implant failure (presumably because the operated side cannot be protected from weight-bearing). As a result, most surgeons consider it appropriate to wait for 6–8 weeks before considering surgery on the contralateral limb, despite the fact that by then the hip may no longer be a candidate for such a procedure.

Complications Complications of TPO include:

- Unsatisfactory outcome due to poor patient selection – especially with respect to there already being too much secondary remodelling
- Implant failure – most commonly seen as pull-out of the cranial plate screws, the incidence of which is reduced by placement of at least one bone screw into the sacral body
- Outward rotation of the stifle during limb protraction – especially where rotations of 40° or more are used – but this may improve with time and seems not to affect limb function
- Reduction in range of hip joint extension – a mechanical impediment which does not seem to affect hindlimb function
- Narrowing of the pelvic canal – especially if the procedure is performed bilaterally; minimised by resection of part of the pubis or making the pubic osteotomy close to the acetabulum; and only likely to be of clinical significance in a whelping bitch, a situation which should not arise since an animal clinically affected by hip dysplasia should not be used for breeding
- Iatrogenic damage to the sciatic nerve when making the iliac osteotomy – rare and avoidable with due care and attention

Results Published results for this technique, including its variations, tend to suggest that between 80 and 90% of cases will regain satisfactory function within 3 months of surgery. Some authors advocate the use of TPO in clinically silent cases of hip dysplasia in order to prevent the future development of osteoarthritis. Although this has not been proven by long-term clinical follow-up, it may be the case since the question as to whether a total hip replacement can be performed on a hip that has had a TPO has not yet needed to be answered to the authors' knowledge!

Intertrochanteric osteotomy

Femoral osteotomy involves removal of a medially based intertrochanteric wedge which allows the angle of inclination to be reduced, thus pushing the femoral head further into the acetabulum. This technique is most appropriate when coxa valga is present with an angle of inclination in excess of 150° (Fig. 40.29). At the same time the anteversion angle can also be reduced. Normally this is about 10° but in cases with dysplasia it is often about 30°. Before stabilising the osteotomy site, the distal femur can be outwardly rotated so that, after fixation, when the stifle returns to its normal position, the femoral head becomes inwardly rotated. Thus, during the procedure, *varisation* causes downward turning of the femoral head (increasing the articular contact in a vertical plane) whilst *retroversion* causes inward turning of the femoral head (increasing articular contact in a horizontal plane). Following reduction the site can be stabilised using a special hook plate (Stratec) or a well-contoured DCP (Fig. 40.30a,b) so as to achieve three-point fixation in the proximal femur. During fixation it may be possible to

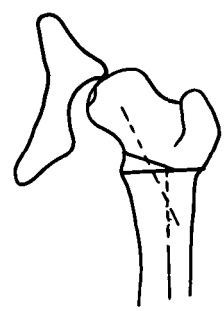


Fig. 40.29 Intertrochanteric osteotomy for treating hip dysplasia, indicated when the coxa valga (femoral neck angle in excess of 150°) is found in association with hip dysplasia. Removal of a medially based wedge will reduce the angle of inclination of the femoral neck and retroversion can also be achieved by rotating the proximal segment inwards prior to fixation.

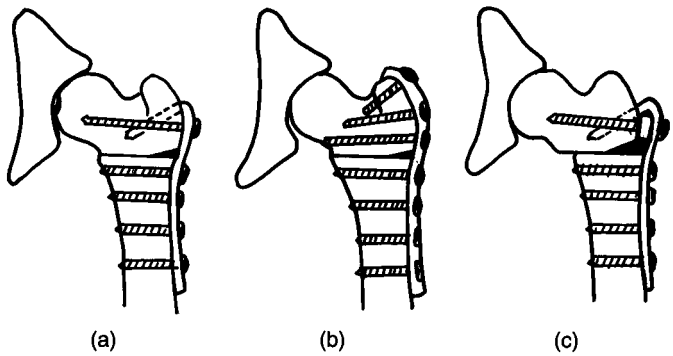


Fig. 40.30 Methods of stabilising an intertrochanteric osteotomy. (a) A hook plate (Stratec). (b) A well-contoured dynamic compression plate. (c) Placement of spacers (washers) under the plate proximal to the osteotomy will lateralise the femoral shaft, effectively lengthening the femoral neck.

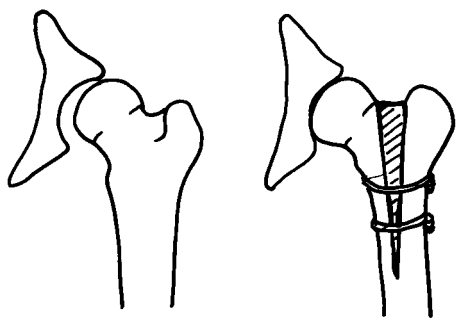


Fig. 40.31 Illustration of the principle underlying femoral neck lengthening procedures (see text for details).

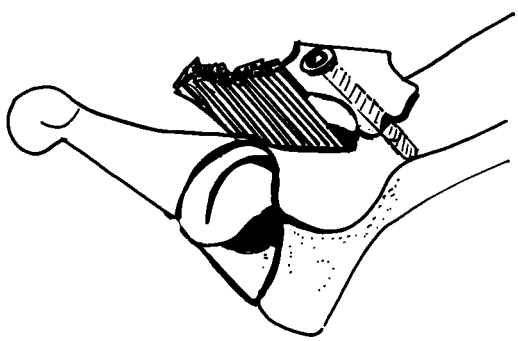


Fig. 40.32 Illustration of the principle underlying the Sertl (BOP) shelf arthroplasty (see text for details).

lateralise the femoral shaft relative to the proximal fragment, thus effectively lengthening the femoral neck (Fig. 40.30c). The technique is demanding, with respect to planning and execution (Prieur, 1987) and is performed less and less as TPO techniques have gained favour. The results of this procedure were reviewed by Evers *et al.* (1997).

Femoral neck lengthening procedures

The aim of these is similar to that of intertrochanteric osteotomy but improved femoral contact is achieved by creating a vertical cut between the femoral head and the greater trochanter and then displacing the femoral head medially by driving wedges down into the osteotomy (Fig. 40.31).

Sertl shelf arthroplasty

The aim of this technique is to extend the dorsal acetabular rim to cover the femoral head by stimulating bone deposition upon a scaffold of material termed BOP (biocompatible osteoinductive polymer) which is attached/implanted into the dorsal acetabulum (Fig. 40.32). Although this does help stabilise the joint, it may be simply promoting the osteophytosis that is seen with osteoarthritis and will certainly not improve stability by increasing articular contact. For these reasons the technique has not been widely accepted as a method of management. A similar principle of creating a dorsal shelf of bone is employed in the dorsal acetabular rim arthroplasty (DARthroplasty) described by Slocum & Slocum (1998a,b).

Pectineal myectomy

This is a technique which appears to reduce pain from a dysplastic, or osteoarthritic, hip joint but

does not have any influence on the further development of radiographic signs (Bowen *et al.*, 1972). The reason for the procedure leading to a reduction in pain is unknown but certain hypotheses have been suggested:

(1) The tight pectineus may create excessive hip adduction resulting in increased subluxation of the femoral head. After myectomy the hip may abduct, increasing the articular contact, decreasing the stresses on the weight-bearing articular surface and thus reducing pain resulting from remodelling in response to these stresses. In the osteoarthritic hip the alteration in femoral head position may allow relatively normal articular cartilage to contact the acetabulum thus reducing the pain caused by weight-bearing on eburnated cartilage.
(2) Electromyography readings from the muscle in an anaesthetised dog suffering with hip dysplasia show a great increase in evoked potentials when the hip is abducted. Therefore, the hyperactive stretch reflex within the muscle may contribute to the pain noted in these cases and myectomy would remove such a source of pain (Bowen, 1974).

Whatever the reason there is no doubt that this procedure can relieve pain seen with this condition in both the immature and mature patient and will be mentioned again under the management of osteoarthritis. The surgical approach is described above under 'Triple pelvic osteotomy' (Fig. 40.28a). It is important that the muscle belly is removed from the tendon of origin to the tendon of insertion otherwise the muscle will fibrose back together and, after contraction of the scar tissue, the preoperative problem might recur.

Its indications in immature patients are as an adjunct to conservative treatment when osteotomy is not indicated but the pain is difficult to control. In one study (Vaughan *et al.*, 1975) the results of the procedure were considered to be good with significant improvement in the clinical signs in 78% of cases. There are instances where the hip joint has been said to become more lax after this procedure, with deterioration in the clinical signs, and, therefore, unless the signs are marked, it may be worth avoiding and instead relying on conservative management alone in these cases.

Excision arthoplasty

This must always be considered a salvage procedure, particularly in large breeds of dog. Although a good return of function may be seen in small dogs and cats with hip pain due to dysplasia or osteoarthritis, the results in larger breeds are less encouraging. The use of muscle sling techniques is discussed later under 'Osteoarthritis'.

Therefore, this technique should be reserved for those cases where the signs are severe, osteotomies are inappropriate due to secondary changes or cost, total hip replacement cannot be considered due to the dog's age or financial constraints and euthanasia is being considered as the only alternative. It is *rare* for this procedure to require consideration in the management of hip dysplasia as seen in the skeletally immature animal.

Control of hip dysplasia

In the UK to date, the attempts to control this disease have revolved around the BVA/KC Hip Dysplasia Scoring Scheme. Unfortunately, the genetics of the problem are not simple and the trait is thought to be polygenic with incomplete penetrance. It is probable that environmental factors will influence the phenotype, including the radiographic appearance. With this in mind, in terms of choosing breeding stock, a scheme needs to identify animals that are free of disease. The current scheme scores the animal's phenotype and the general advice is to breed from animals with a score lower than their breed average. This may lead to a reduction in the breed's average score, with time, but will it have any influence on the incidence of clinical hip dysplasia since dogs with the disease are still being used for breeding and clinical signs show a poor correlation with radiographic appearance? It may, therefore, be unlikely that the current scheme will make much further impact on the disease.

It would, perhaps, be more appropriate to assess hip laxity itself and a scheme has been developed by Gail Smith at the Pennsylvania State University Veterinary School. The scheme measures the

position of the femoral head with the hip joints compressed and distracted. The difference is expressed as a proportion of the femoral head radius and this provides a measure of joint laxity. The results he reported to a specialist group meeting at the BSAVA Congress 1994 were very encouraging in that a dog's subluxation index correlated well with its likelihood of developing clinical signs. (These findings have been detailed in a number of references: Popovitch *et al.*, 1995; Smith *et al.*, 1990, 1993, 1995.) Having followed several generations of dogs in one breeding line he has shown that choosing breeding stock using this scheme can dramatically reduce both the radiographic hip subluxation and its clinical expression in the progeny. Unfortunately, at the moment, the radiographic technique requires manual restraint and could not be employed in the UK due to the 1985 Ionising Regulations.

A further alternative might evolve if the genes responsible for the disease could be identified since it might then be possible to 'score' a dog's potential to pass on hip dysplasia from analysis of the cells in, say, a routine blood sample.

Hip dysplasia in cats

The clinical and radiological features of feline hip dysplasia resemble those found in the dog but the occurrence of this as a clinical problem in the cat is rare. That said, it may be more commonly seen in the Siamese cat. The literature on feline hip dysplasia was reviewed by Holt (1978) who described the successful treatment of the condition in a Persian cat. The animal was initially treated by bilateral pectineal myectomy but this only caused a temporary improvement in fuction. Subsequently, bilateral excision arthroplasty was carried out, after which there was a complete remission of lameness. The condition has also been reported more recently in a series of three cats (Patsikas *et al.*, 1998).

Osteoarthritis (OA)

These dogs will present as adults, usually in middle to old age but sometimes as young as 12–18 months. The history is most commonly one of stiffness on rising, exercise intolerance and/or lameness that is made worse by exercise, with stiffness after rest following exercise. The severity may vary from day to day and be affected by such things as the weather and the degree of exercise.

On examination the signs most commonly include lameness of varying severity or a stiff hindlimb gait and, if the problem is bilateral, the dog may have a tendency to bunny-hop at faster paces. Atrophy, particularly of the gluteals is often apparent and manipulation of the hip(s) results in pain and reveals a reduced range of motion. Joint laxity, as such, is not generally a feature in cases at this stage of the disease.

Radiology

In the skeletally mature animals with signs attributable to osteoarthritic hip joints the radiological findings will generally relate to the secondary changes already outlined above (Fig. 40.33), namely:

- Osteophyte formation around the femoral neck
- Osteophyte formation causing 'in-filling' of the acetabular fossa

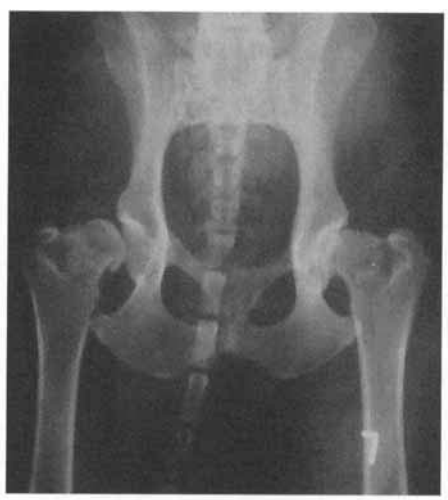


Fig. 40.33 Ventrodorsal radiograph of the pelvis of a 9-year-old Labrador Retriever showing marked periarticular osteophytosis indicative of osteoarthritis (OA). However, a dog showing these radiographic changes need not be lame (see text and also Chapter 7).

- Remodelling of the acetabular rim (especially the effective cranial acetabular rim) resulting in bilabiation and/or facet formation
- Osteophyte formation on any of the acetabular rims

Again *it must always be remembered that the radiographic appearance relates to the pathology present, to some extent, but does not necessarily correspond to the severity of the clinical signs noted.*

Differential diagnosis

In the mature patient, other common differential diagnoses include:

- Other bilateral orthopaedic conditions such as cruciate disease
- Lumbosacral disease (especially degenerative compression syndrome)

Diagnosis

The diagnosis is based on the clinical history and clinical signs, with attention being paid to ruling out other causes, and confirmed by radiography. It has to be stressed, however, that a diagnosis cannot be made from radiography alone. *Many dogs have radiographic changes consistent with hip OA without showing any related clinical signs.*

Treatment

Conservative management

The conservative management of OA is considered elsewhere (see Chapter 7, p. 59).

Pectineal myectomy

This procedure, its indications and results have been considered above. In mature patients the results are a little unpredictable but may lead to clinical improvement for periods ranging from several months to several years. It is well worth trying in dogs failing to respond to conservative measures where more advanced techniques are not appropriate.

Metaphyseal forage

This technique was reported by Svalastoga & Madsen (1992). It involves drilling a hole from the lateral aspect of the femur, up the femoral neck and into the femoral head stopping a few millimetres short of the articular surface. The principle behind this is similar to that of metaphyseal osteotomy, used to treat osteoarthritic joints in human patients, which is thought to relieve venous congestion which may contribute to joint pain. In the aforementioned study seven dogs with hip OA secondary to hip dysplasia were treated in this way and all showed a marked improvement in their clinical signs. Further studies are required to evaluate its use in treating hip OA but it may prove a useful adjunct to conservative management.

Total hip replacement (THR)

This procedure is becoming well established in veterinary orthopaedics. Several variations regarding technique and implants exist but most involve the replacement of the acetabulum with an ultra-high molecular weight polyethylene cup (Fig. 40.34a) and of the femoral head with a cobalt chrome ball and stem (Fig. 40.34b), which are secured in position using polymethylmethacrylate. The use of uncemented prostheses has been investigated (DeYoung *et al.*, 1992) but these have yet to gain acceptance in clinical practice, mainly because of the much higher cost of the implants.

Olmstead *et al.* (1981) described the technique for THR in the dog using a Richards type II Canine Total Hip Prosthesis and reported satisfactory results in over 90% of cases. More recently a modular prosthesis has been developed by a number of manufacturers in collusion with canine orthopaedic surgeons. These include: Biomedtrix Inc., Ohio, USA; La Biomécanique Intégrée, Brétigny-Sur-Orge, France; and Veterinary Instrumentation, Sheffield, UK. Each of these systems presents the surgeon with a choice of several sizes of acetabular cup and femoral stem which can be matched to the radiographs of the individual dog (and finally checked during surgery). The femoral head forms the third component and for each femoral stem there will be a choice of two or three

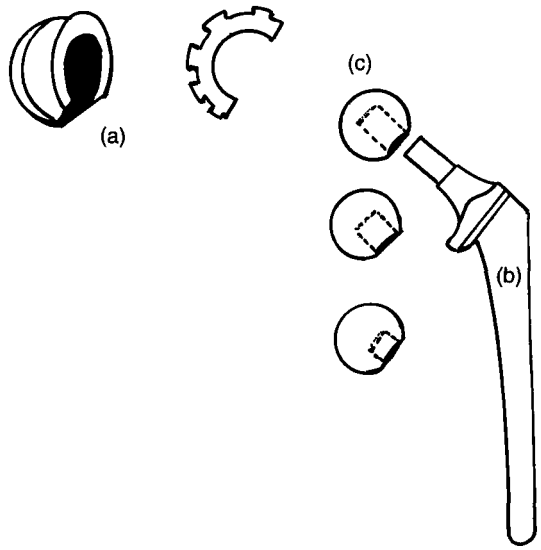


Fig. 40.34 Illustration of a generic 'modular' total hip prosthesis. (a) Ultra-high molecular weight polyethylene cup, (b) cobalt chrome femoral stem, (c) cobalt chrome femoral head (with differing depths of recess to create different functional lengths of femoral neck).

femoral heads (depending on the system used and the size of femoral stem in question). The femoral head forms a taper-cut junction with the femoral neck and the depth of the hole in the femoral head varies between the choices, thus creating options in effective length of femoral neck (Fig. 40.34c). The systems come complete with specialist instrumentation required for the procedure, though how extensive this instrumentation is varies between systems. The results of using these modular systems are similar to those using the original Richards prosthesis and their main advantages are, arguably, ease of use and range of sizes of patients in which they can be used. The use of cemented THRs was reviewed by Olmstead (1995).

The procedure is technically demanding and should only be carried out by a surgeon who is regularly performing surgical procedures on canine hip joints (preferably THR itself) in an environment where the highest standards of asepsis can be achieved and where appropriate instrumentation and surgical assistance is available.

Indications for THR Although OA is the most common reason for considering THR it is not the only indication. A more complete list would include:

- Osteoarthritis secondary to hip dysplasia
- Osteoarthritis secondary to healed fractures of the acetabulum or proximal femur
- Primary treatment of non-reconstructable fractures of the femoral head
- Recurrent coxofemoral luxation

The possibility of converting an excision arthroplasty into a THR because of poor limb function is often enquired about. Such surgery is difficult because the line of osteotomy across the femoral neck is different for THR (where a medial buttress is preserved) and the risk of postoperative infection around the prosthesis is greatly increased by operating in an area comprising considerable scar tissue. Indeed, if any surgery is performed on the hip and there is a possibility of needing to consider THR at a later date (e.g. acetabular fracture repair), then the first surgical procedure needs to be carried out with the same attention to asepsis as would be the case for a THR so as to minimise the possibility of residual bacteria lying dormant in the scar tissue just waiting for the second procedure! In the authors' opinion the option of THR should be discussed before an excision arthroplasty is performed and once such a salvage procedure has been decided upon then a THR is, in practical terms, contraindicated.

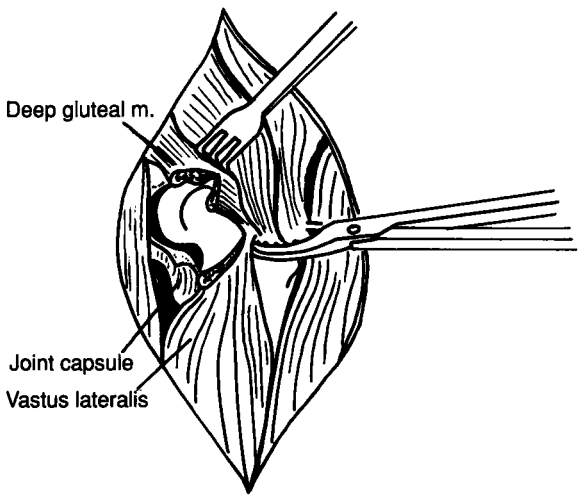
In addition there are certain other criteria which must be met for an animal to be a suitable candidate. These include:

- Showing clinical lameness that is non-responsive to conservative management (i.e. the decision to perform a THR is not based on the radiographic appearance of a hip)
- The animal must be of a size that is sufficient to accommodate at least the smallest prosthesis (this varies between the systems in use but the lower bodyweight limit is generally somewhere between 20 and 30 kg)
- There must not be evidence of other disease that might compromise the value of a THR (e.g. progressive neurological problems such as degenerative myelopathy, or neoplasitc conditions elsewhere in the body)

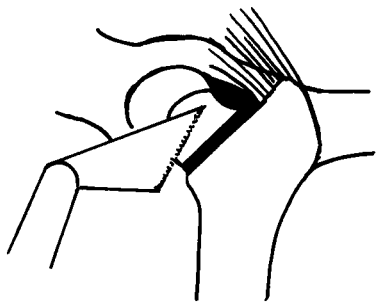
- There must be no established source of bacteraemia which might promote infection around the prosthesis (e.g. periodontal disease, anal furunculosis)

Technique for THR (Fig. 40.35) The procedure is technically demanding and requires strict asepsis. In order to maintain good hindlimb function, the prosthesis must remain in place for life which is often not the case for implants such as plates used to stabilise fractures where, if the plate loosens at a later date, the implants can be removed once the fracture has healed. The dog is bathed (+/– the limb clipped) the day before surgery with an antibacterial shampoo and a course of cephalosporin antibiotic is started preoperatively and continued for 5 days after surgery.

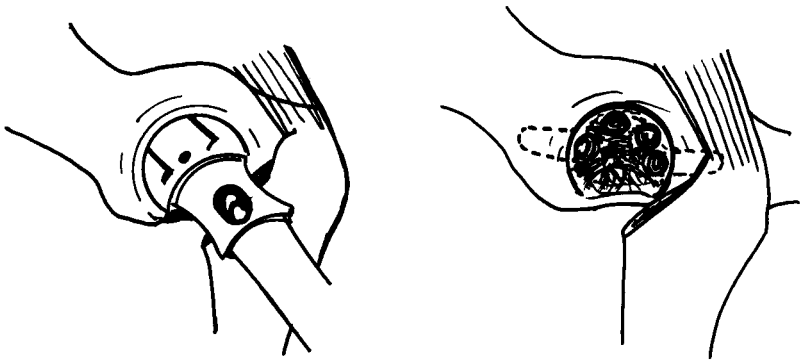
The following description is generally applicable, whatever system is used, but there are some differences in instrumentation with which surgeons must acquaint themselves before using any specific prosthesis. A craniolateral approach is usually sufficient to expose the hip joint adequately (Fig. 40.10). Some surgeons prefer a dorsal approach to achieve better exposure (involving either trochanteric osteotomy or gluteal tenotomy). Large bone-holding forceps



(a) A craniolateral (or dorsal) approach is used to expose the femoral head and neck.

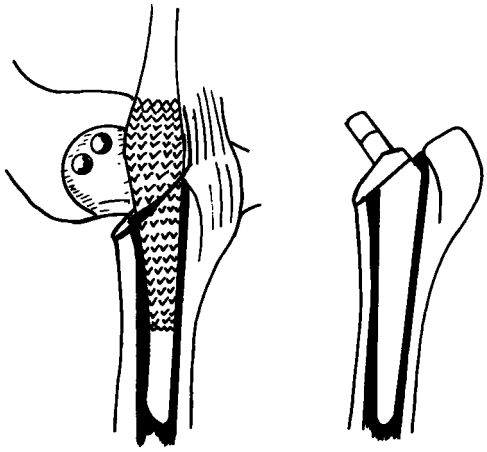


(b) An oscillating saw is used to create a femoral neck osteotomy after a template or guide has been used to establish the line of section required.

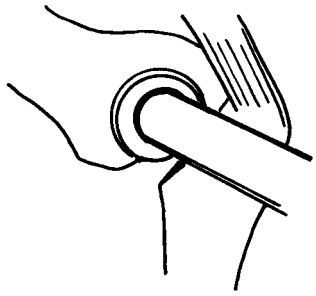


(c) The acetabulum is reamed until the medial cortex is just visible centrally and key holes are drilled into the exposed cancellous bone.

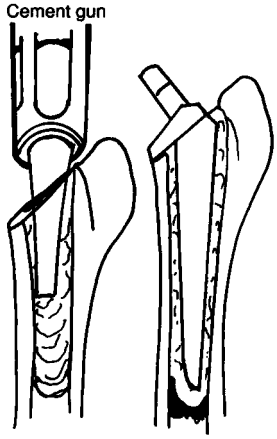
Fig. 40.35 Illustration of the steps involved in total hip replacement (THR). See text for further details.



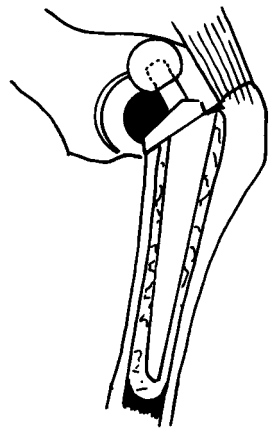
(d) The femoral medullary cavity is reamed until the chosen template can be accommodated with ease and rests evenly on the remnant of femoral neck.



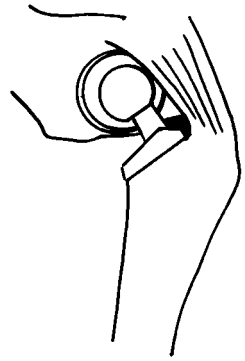
(e) Bone cement is pushed into the acetabulum and then the prosthetic cup is positioned with the aid of an acetabular 'pusher' or 'positioner'.



(f) Bone cement is packed into the femoral medullary cavity and the prosthetic femoral stem pushed in until its collar rests on the femoral neck in a neutral position.



(g) Using the trial femoral heads, the one providing the best femoral neck length is chosen and 'tapped' into position on the femoral neck.



(h) The prosthetic femoral head is then reduced into the polyethylene cup.

Fig. 40.35 *Contd.*

are required to grip the proximal femur and allow this to be outwardly rotated to expose the femoral head and neck. The joint capsule is incised transversely and the teres ligament sectioned to allow luxation of the femoral head. A periosteal elevator is used to remove all soft tissue attachments to the cranial aspect of the femoral neck (Fig. 40.35a). The femoral prosthesis, its template or an osteotomy guide is lined up with the femur and the angle of section of the femoral neck planned. An oscillating saw is used to create this osteotomy (Fig. 40.35b).

The femur is retracted caudally to expose the acetabulum. An acetabular reamer, of a size corresponding to the acetabular cup chosen for the particular dog, is then used to ream through the articular surface of the acetabulum and then through the subchondral cancellous bone until the medial cortex begins to show centrally. A drill is then taken and used to create holes in the cancellous bone extending cranially into the ilium, caudally into the ischium and two or three around the periphery of the acetabulum 'under' the dorsal rim of bone (Fig. 40.35c). The holes may be linked together by creating channels with a curette. These holes are essential to key in the bone cement and should not penetrate the medial cortex of the acetabulum otherwise cement may leak into the pelvic canal and the heat generated as it sets may injure the nearby sciatic nerve. The acetabulum is then packed with saline-soaked swabs to reduce haemorrhage from the bone. Using saline that has been cooled in a fridge beforehand is advantageous with respect to haemostasis.

The proximal femur is then reamed out using drills, reamers, a rasp or a broach until the stem of the prosthesis (or its template) can be slid easily into the medullary cavity such that the collar of the prosthesis rests evenly against the remnant of the femoral neck, especially with respect to the medial calcar (Fig. 40.35d). The medullary cavity is then flushed with cooled saline.

The appropriate size of acetabular cup is selected and a trial insertion carried out. The cup should be seated in the normal longitudinal axis of the acetabulum with about 10° of lateral tilt of the dorsal edge of the cup. Having checked that the cup can be easily inserted and the correct position assessed, the cup is removed and the bone cement mixed according to the manufacturer's instructions. The most commonly used bone cement in the UK is CMW3 (DePuy) which is a gentamicin-impregnated polymethylmethacrylate, although CMW2 (DePuy) is more rapid setting and so can be used for the acetabular component. A soft bolus of this is pushed into the dry acetabulum and then the acetabular cup is slowly pushed into the cement and orientated using the acetabular 'pusher' or 'positioner aid' (Fig. 40.35e). Excess cement is removed. The cup is held firmly and still whilst the cement goes hard as movements made late in the curing process tend to break the cohesion between the implant and cement, and this may result in the prosthesis being unstable. Once the cement has set hard, the final position and stability of the acetabular cup is checked and any loose tags of bone cement removed.

The appropriate size of femoral stem is selected and a trial insertion carried out. A second pack of bone cement is mixed and again CMW3 (DePuy) is suitable. This is most suited to 'hand packing' into the femur but can be introduced via a syringe if preferred. Alternatively, a lower viscosity cement, CMW1 (DePuy), may be used which is more amenable to being introduced into the femur with a syringe. To facilitate introduction of cement, a urinary catheter can be placed so that its tip reaches the bottom of the reamed medullary cavity. This allows air to escape from the cavity as cement is forced in. After removal of the urinary catheter, the femoral stem is inserted and seated in a neutral position (Fig. 40.35f). Extruded cement is removed and may be used to fill any defects between the collar of the prosthesis and the neck of the femur (though the aim is always not to have any such defects). Once the cement has set then the remaining choice relates to the femoral head and what length of femoral neck is required. In the authors' experience it is more common to require the longer femoral necks but this is tested in each patient by using the trial femoral heads, reducing the hip and evaluating stability. If the joint can be reduced easily then a longer neck is usually required. Once the appropriate femoral head has been chosen it is 'tapped' onto the femoral neck after being covered with a swab to protect its surface from damage (Fig. 40.35g). The femoral head is then reduced into the

acetabular cup and hip joint stability and range of motion tested (Fig. 40.35h). Swabs may be taken at this stage for bacterial culture to try and ensure that the operative site has remained aseptic. Closure is routine including suturing of the remaining joint capsule to the gluteal tendons of insertion. If a trochanteric osteotomy has been utilised then it may be transposed caudodistally to further improve joint stability (see under 'Coxofemoral luxation' earlier in this chapter).

Postoperatively an oblique lateral radiograph of the pelvis will suffice if there is concern over positioning for a ventrodorsal view being likely to cause luxation of the prosthesis. A light bandage may be applied over the hip or else it may be left unbandaged. The dog's exercise is restricted to lead walks on level ground and room rest for 2 months. Unrestricted access to stairs and jumping in and out of the car at will are examples of activities which should be avoided.

Follow-up radiographs are taken after 2–3 months to ensure all is well before exercise is returned to normal. If the procedure is being considered bilaterally then the operations are usually separated by at least 3 months. However, most pets will regain satisfactory function with surgery on only one painful hip and in one series described by Olmstead *et al.* (1983) only about one in five dogs with bilaterally painful hips required bilateral surgery to achieve satisfactory function. There is an argument for taking radiographs of these cases each year after surgery in case any early signs of complications might be detected. However, convincing owners that this should be done may prove difficult, especially when an anaesthetic has to be administered each time.

Results and complications of THR Success rates of 85–95% have been reported with less complications being seen as surgeons gain more experience. However, in those cases that are unsuccessful the reason usually relates to a serious complication which may develop soon after surgery or several months/years later and usually involves:

- Luxation of the prosthesis
- Loosening of the acetabular cup
- Infection
- Fatigue fracture of the femoral prosthesis
- Fracture of the femur
- 'Implant-related' osteosarcoma

If *luxation of the prosthesis* does occur then it is usually within the first 6 weeks and is most commonly due to an incorrect angle of placement of the acetabular cup. Closed reduction can usually be achieved by caudoventral traction on the leg, after which the hip may be strapped up as discussed above and the dog rested for a further 6 weeks. If the luxation recurs then treatments to be considered would include removal and replacement of the acetabular cup (see below), triple pelvic osteotomy or removal of the prosthesis, leaving the dog with an (expensive) excision arthroplasty.

Aseptic *loosening of the acetabular cup* may be seen within 4 months of surgery and is probably related to technical faults with its implantation, e.g. not seated deeply enough into the acetabulum, incorrect angle, insufficient cement. It is possible to revise the surgery by removing the acetabular cup and cement, re-preparing the site and implanting a new acetabular cup. This is more difficult than during the primary surgery as the femoral prosthesis obstructs exposure of the acetabulum.

Infection may gain access to the implants at the time of surgery or else through a bacteraemia at a later date. The dog will gradually stop using the leg and radiographs will show lysis at the bone–cement interface. Prolonged antibiotic treatment may improve the situation but, ultimately, removal of the prosthesis is required along with all the bone cement. The excision arthroplasty created is never as functional a pseudoarthrosis as following a primary femoral head and neck resection, probably because of increased scarring in the region from the two procedures and also the presence of infection.

Fatigue fracture of the femoral prosthesis may occur through its 'neck'. Although replacement of the femoral component is technically possible, it is a very demanding piece of surgery, unless the stem has also come loose, because removing the stem may involve accessing the cement through a longitudinal channel created in the femoral shaft. The cement then has to be removed to release the femoral stem.

Alternatively, the proximal end of the femoral component may be removed along with the acetabular cup to leave an excision arthroplasty with the femoral stem left *in situ*.

Fracture of the femur may be seen at the distal end of the femoral prosthesis or the cement mantle. Although the presence of the rigid stem/cement within the medullary cavity may create a 'stress riser' at the point where they stop, it may only serve to influence the way in which the bone fractures rather than being the cause of the fracture per se. Such fractures can be stabilised by plate fixation but obviously there is some difficulty in securing the plate to the proximal femur (the greater trochanter is utilised) and some concern over the medullary blood supply to that region of the bone.

There have been a few reports of *osteosarcomata* developing in the proximal femur subsequent to THR (Murphy *et al.*, 1997b; Roe *et al.*, 1996). As in the case of fracture-related sarcomata, the presence of implants has been suggested as the cause. However, the common denominator in the fracture cases is usually problematic healing which may indicate that the source of neoplasia in these patients was a chronic inflammatory process at the site. In the case of THR there may be chronic micromovement at the bone–cement interface which might also produce a low-grade, chronic inflammatory process. Alternatively, there has been some concern in human patients that 'wear debris' from the artificial hip may accumulate in the local soft tissues (and also further afield in the lymphatic system) and might provoke a chronic inflammatory response which might predispose, eventually, to malignant pathology. Such wear debris has been reported in the soft tissues surrounding a total hip prosthesis removed from a dog, though not one in which osteosarcoma was diagnosed (Day *et al.*, 1998).

Excision arthroplasty

This has already been discussed previously as a salvage procedure and the technique is described under 'Coxofemoral luxation' and 'Legg Perthes' disease' (Figs 40.10 and 40.19). It should be reserved for cases that fail to respond to conservative measures and/or pectineal myectomy, when THR is inappropriate and euthanasia would be the only practicable alternative. Suggestions have been made that the results can be improved by use of a biceps femoris muscle sling technique to keep the osseous surfaces apart, but in an experimental study of the results of the technique with and without a sling, Mann *et al.* (1987) reported that none of the dogs regained normal function and there was no significant difference in the clinical outcome of the two techniques. Variations in this 'sling' technique have also been reported as providing no better results than a standard excision arthroplasty (Remedios *et al.*, 1994; Dueland *et al.*, 1997) and sciatic nerve entrapment, as a complication of these techniques, has been reported in two dogs (Jeffery, 1993).

Prognosis

Most animals with clinical signs related to hip OA can retain good mobility with conservative measures alone. Those in which this is not the case are candidates for surgical intervention with THR providing a return to normal or near-normal function in about 90% of cases. If excision arthroplasty has to be considered as a salvage procedure, then the results may improve the animal's mobility but in medium-sized dogs the results are more variable and generally less satisfactory than when this procedure is performed in smaller (or giant) breeds.

VW HOOD (von Willebrand associated heterotopic osteochondrofibrosis of Dobermanns)

von Willebrand's disease is the commonest inherited bleeding disorder of man and the dog (Littlewood *et al.*, 1987). The condition has been reported in many breeds of dog but in the Dobermann Pinscher it has also been associated with hindlimb lameness. Typically, affected dogs present with a moderate to severe, progressive lameness associated with muscle atrophy, a markedly reduced range of motion in the hip joint and pain on hip manipulation, especially on extension. Radiographs may show no abnormalities in the early stage, or else the only abnormality noted is that of subluxation leading to a provisional

(mis)diagnosis of lameness associated with hip dysplasia. With time, a periosteal reaction develops on the ischium and/or proximal femur and a mass containing calcified tissue develops close to the hip. This mass usually involves the muscles caudal to the joint (gemelli, internal and external obturator, quadratus femoris) but may also affect the pectineus and iliopsoas muscles and can easily be misdiagnosed as neoplastic. The condition probably results from trauma with haemorrhage and an initial fibrous reaction progressing to a chondro-osseous response. Affected dogs should test positive for von Willebrand's disease.

Treatment with systemic prednisolone may lead to a satisfactory improvement in hindlimb function but in others it may prove necessary to resect the mass of osteochondrofibrotic tissue and to section the affected muscles, in order to restore a more normal range of hip joint movement. The response to such surgery has been reported as good (Dueland *et al.*, 1989) with four cases making complete recoveries.

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A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny,Steven J. Butterworth

Chapter 41
The Femur

Conditions of the femur

Conditions that may affect the femur may be divided into three groups and include:

- *Traumatic*
  - Fractures of the proximal femur:
    - capital fractures
    - capital (proximal) femoral growth plate
    - femoral neck
    - greater trochanter
  - Diaphyseal fractures
  - Fractures of the distal femur:
    - involving the distal femoral growth plate
    - condylar fractures

- *Developmental*
  - Coxa valga (may be relevant to hip dysplasia – see Chapter 40, p. 470)
  - Bowing distally may be related to genu valgum or patellar luxation (see Chapter 42)
  - Panosteitis (see Chapter 48)
  - Metaphyseal osteopathy (see Chapter 48)

- *Acquired*
  - Osteosarcoma (see Chapter 51)

Other than the fractures listed above, these conditions are discussed elsewhere and so it is the traumatic femoral conditions that will be considered below. Femoral fractures, especially those that involve the diaphysis, are commonly encountered in dogs and cats and are usually the result of road traffic accidents.

Fractures of the proximal femur

Some 90% of fractures which involve the femoral head or neck occur in animals between 4 and 6 months of age. In the skeletally immature dog, the main blood supply to the femoral head is derived from the epiphyseal vessels associated with the joint capsule while a small amount is derived from vessels running through the teres ligament. It is only after closure of the capital femoral growth plate at 8–11 months of age that the femoral head receives an additional blood supply from the metaphyseal vessels (see Chapter 40, Fig. 40.2). Traditionally, fractures of the femoral head and neck have been divided into two groups:

- Intracapsular fractures – those which occur within the joint capsule
- Extracapsular fractures – those occurring outside the joint capsule

If the blood supply to the femoral head is considered it would seem reasonable to assume that a potential complication of intracapsular fractures in immature animals would be disruption of the blood supply. Although ischaemic necrosis of the femoral head is a common complication of intracapsular fractures in children, this does not appear to be a significant problem in dogs and cats provided stable internal fixation is carried out soon after the accident, preferably within 48 hours and certainly within a week.

Capital fractures

Capital fractures occur most often as a complication of coxofemoral luxation (Fig. 41.1a). A bone fragment involving the insertion of the teres

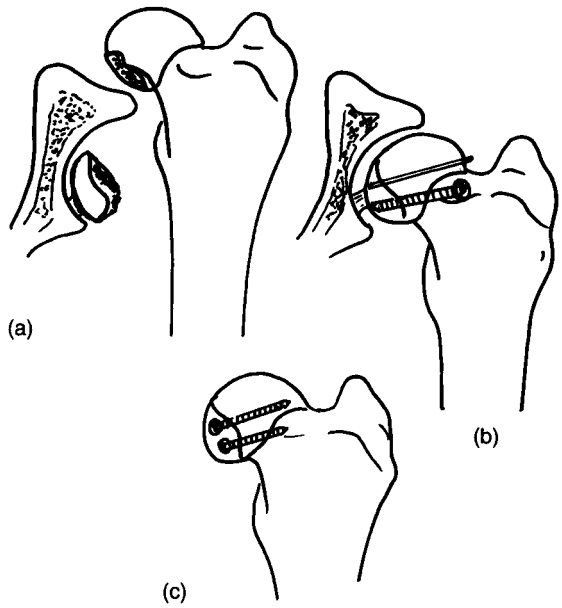


Fig. 41.1 (a) Fracture of the capital femoral epiphysis with luxation of the femoral head. (b) Fixation using Kirschner wire and a lag screw introduced from the lateral side of the fracture. (c) Fixation using countersunk screws introduced from the articular surface.

ligament is avulsed from the femoral head. Surgery is required to treat these and the options available include:

- Excision of small fragments during open reduction of the coxofemoral luxation.
- Lag screw fixation of larger fragments using countersunk screws (2.0mm) inserted in a retrograde manner from the articular surface (Kuzma *et al.*, 1989; Tillson *et al.*, 1994) (Fig. 41.1b,c). However, the prognosis is guarded and lameness may well persist (Miller & Anderson, 1993) in which case excision arthroplasty may be necessary as a salvage procedure.
- Primary excision arthroplasty which is particularly appropriate in small dogs and cats.

The surgical technique involved, the postoperative care and the prognosis are as for fractures through the proximal growth plate or femoral neck (discussed below).

Fractures through the capital femoral growth plate and fractures of the femoral neck

Again, these fractures require surgical management and two methods of fixation are commonly employed:

- Two or three diverging Kirschner wires. This is used for separations or fracture separations of the capital femoral epiphysis (Fig. 41.2) (Jeffery, 1989; Culvenor *et al.*, 1996).
- Lag screw fixation in conjunction with an anti-rotational Kirschner wire, used particularly in femoral neck fractures (Fig. 41.3) (Nunamaker, 1973; Hulse *et al.*, 1974).

An *in vitro* study, comparing the mechanical performances of four methods of internal fixation of femoral neck fractures in dogs, has been published by Lambrechts *et al.* (1993). The methods applied were:

- Two parallel Kirschner wires (2.0mm)
- Two divergent Kirschner wires (2.0mm)
- Three parallel Kirschner wires (2.0mm)
- One centrally placed 4.0mm cancellous lag screw + parallel Kirschner wire

The study showed that the latter two techniques were the most resistant to failure during loading.

Surgical technique

A craniolateral or a dorsal approach to the hip is used to expose the fracture of the femoral head or neck. The dorsal approach (see Chapter 39, Fig. 39.12) gives excellent exposure, allows accurate reduction of the fracture and makes placement of the implants relatively simple. Although the craniolateral approach gives more restricted exposure, its advantage is in maintaining soft tissue support for the repair and not causing further vascular injury (see Chapter 40, Fig. 40.10). A ventral approach has also been advocated for avulsion fractures (L'Eplattenier & Montavon, 1997). With a dorsal approach, after reflection of the greater trochanter and gluteal muscles, the dorsal joint capsule is incised to reveal the fracture (Figs 41.2a and 41.3a). The proximal femoral shaft is grasped with Kern bone-holding forceps, and a small Hohmann retractor is introduced into the joint

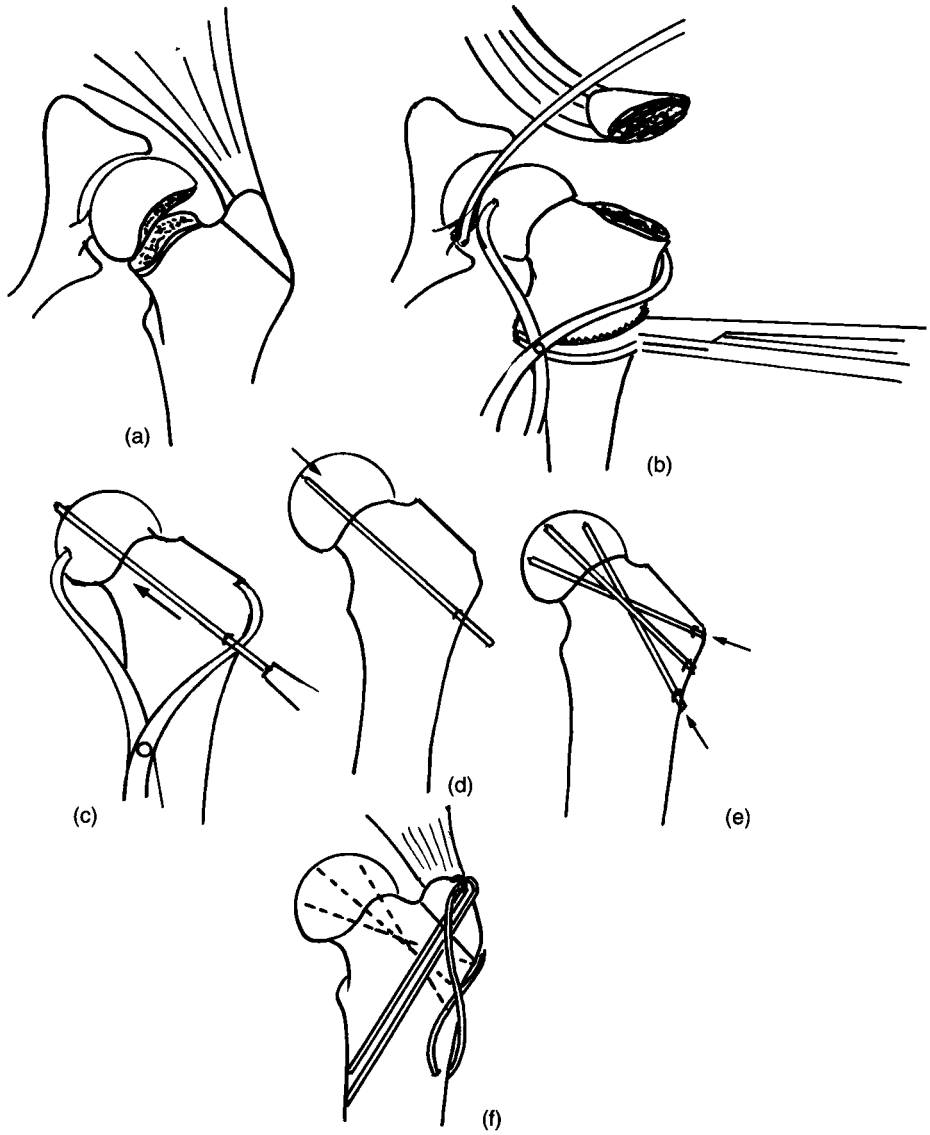


Fig. 41.2 Separation of the capital femoral epiphysis, reduction and fixation using Kirschner wires. (a) Dorsal approach to the hip with trochanteric osteotomy. (b) Dorsal reflection of the trochanter and gluteal muscles to expose the hip. Reduction of the epiphysis with the aid of a Hohmann retractor in joint space. The reduction is maintained with pointed reduction forceps. (c) First Kirschner wire is introduced until the tip just penetrates the articular cartilage. (d) The Kirschner wire is withdrawn until the tip lies just below the articular surface. (e) Two more Kirschner wires are introduced the same way as the first. (f) The trochanter is reattached with two Kirschner wires and a tension band wire.

space and used to rotate and/or lever the femoral head into its normal position (Fig. 41.2b). Reduction can then be maintained with small, pointed AO reduction forceps (Fig. 41.2c). Alternatively, by exerting pressure towards the acetabulum with the Kern forceps, it is usually possible to hold the fracture in reduction by pressure against the acetabulum.

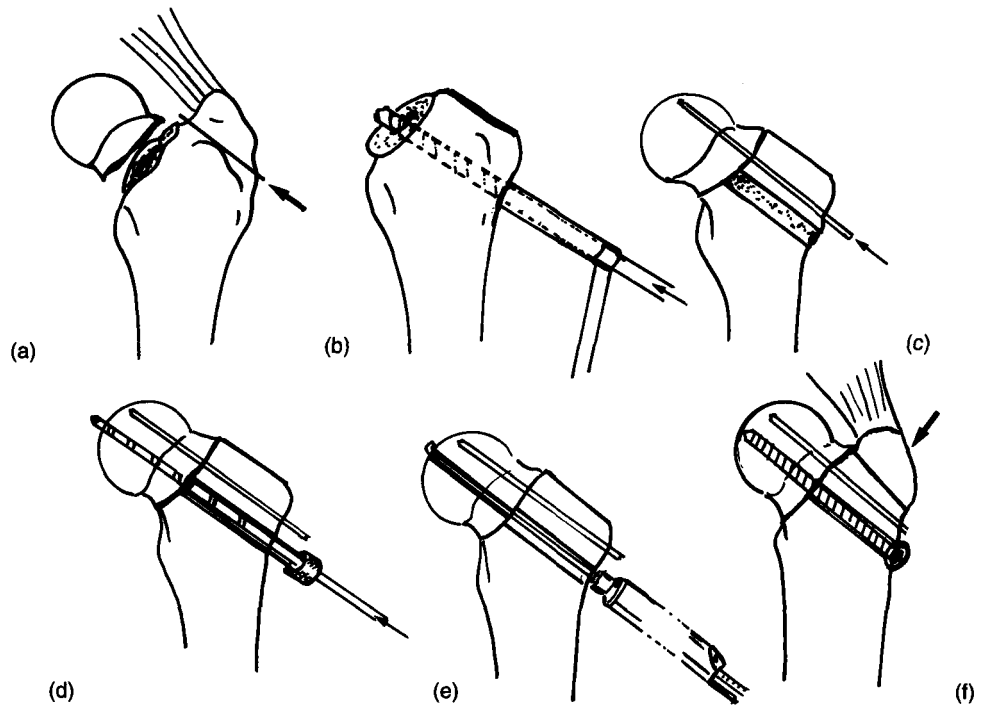


Fig. 41.3 Femoral neck fracture, and fixation using a lag screw and Kirschner wire. (a) Dorsal approach to the hip with trochanteric osteotomy. (b) A glide hole is drilled from the base of the trochanter to emerge in the centre of the femoral neck. (c) The fracture is reduced and fixation is maintained with a Kirschner wire. (d) The drill sleeve is placed in the glide hole and a pilot hole is drilled through the femoral head until the tip of the drill just emerges from the articular surface. (e) The glide hole is countersunk and the length of screw required is measured using a depth gauge. Select a screw 2 mm shorter than indicated so that the screw tip does not penetrate the articular surface once the screw is tightened. (f) Reattach the greater trochanter with tension band wire or a lag screw (see Fig. 41.4).

Kirschner wire fixation If Kirschner wires are to be used for fixation, the first wire is introduced from the base of the greater trochanter and directed towards the fovea of the femoral head. The wire is advanced until the tip just penetrates the articular surface (Fig. 41.2c). It is then withdrawn about 2 mm so that the tip lies just below the articular cartilage (Fig. 41.2d). Reduction of the fracture is checked before two more Kirschner wires are introduced in a similar fashion. These diverge from the first (Fig. 41.2e). The wires are then cut close to the base of the trochanter. Bending the wires over first reduces the likelihood of migration but care must be taken not to withdraw them a little way as they are bent because the length of wire gaining purchase in the epiphysis is small to begin with. For this reason some surgeons prefer to cut them flush with the bone without bending them.

Lag screw and anti-rotational Kirschner wire If a lag screw and anti-rotational Kirschner wire are to be used for fixation, the glide hole for the screw is prepared first. The hole is drilled from the base of the greater trochanter to emerge in the centre of the femoral neck (Fig. 41.3b). The fracture is reduced and initial fixation is achieved with a single Kirschner wire which is advanced parallel with the glide hole until its tip just emerges through the articular surface of the femoral head (Fig. 41.3c). The Kirschner wire is then pulled back about 2 mm so that the tip lies beneath the articular cartilage. Reduction of the fracture is checked. Using a drill sleeve in the glide hole, a

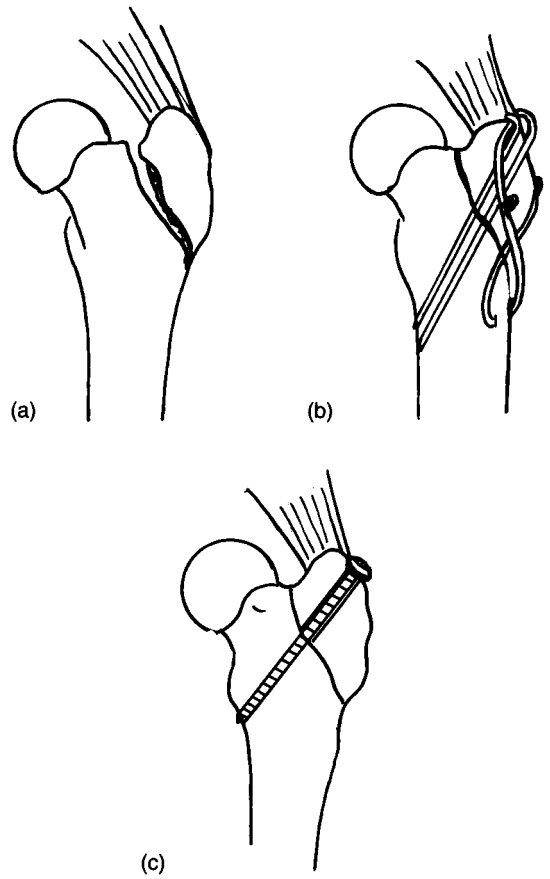


Fig. 41.4 (a) Fracture of the greater trochanter. (b) Fixation with Kirschner wire and tension band wire. (c) Lag screw fixation.

pilot hole is then drilled through the femoral neck and head. The drill bit is advanced until it just penetrates the articular cartilage (Fig. 41.3d). The drill sleeve is removed and then a depth gauge is used to accurately measure the length of the hole from the trochanter to the articular surface (Fig. 41.3e). A screw is chosen which is about 2–4 mm shorter than indicated to ensure that the tip does not penetrate the articular surface once the screw has been tightened (Fig. 41.3f). After stabilisation of the fracture, if a dorsal approach has been used, the greater trochanter is reattached using a lag screw or two Kirschner wires and a tension band wire (see Fig. 41.4).

Postoperative care

Exercise is restricted for 6 weeks following surgery. Implants are left *in situ* unless they loosen and cause soft tissue problems. In young dogs with fractures of the femoral head or neck, lysis of the dorsal aspect of the femoral neck ('apple coring') is often seen on follow-up radiographs taken 4–6 weeks after surgery. This is probably due to a local disturbance in blood supply. The zone subsequently revascularises and remodels and the radiographic finding is seldom of clinical significance.

Prognosis

Prognosis is dependent on early surgery, accurate reduction and stable fixation. If these criteria are met then many cases will make a satisfactory recovery. However, some will develop osteoarthritic changes which may be clinically significant. This may be a result of joint laxity following the injury and surgical treatment.

Long-standing or neglected fractures of the femoral head and neck

Intracapsular fractures in the dog, if untreated, invariably result in non-union with osteolysis of the femoral neck. The condition causes severe hip pain and lameness. Excision arthroplasty is the best treatment option in most cases, although the option of total hip replacement (THR) may be worth consideration in selected cases. The same fracture in the cat will also result in non-union but the functional end results are often surprisingly good, with the cat showing very little evidence of hip pain or lameness 6–8 weeks after the accident (see below).

Femoral neck fractures and capital femoral epiphyseal separations in cats

Based on a study of 62 cats over a 12-year period (Perez-Aparicio & Fjeld, 1993) it was shown that, if left untreated, an acceptable clinical result can be obtained in both femoral neck fractures and capital epiphyseal separations, although the best functional results are achieved following femoral neck fractures. All cats with untreated capital

epiphyseal separations developed hypertrophic pseudoarthrosis. Based on this study there could be an argument for treating these fractures conservatively (cage rest and analgesics) initially with the option of treatment by excision arthroplasty later if hip pain and lameness persist. The results would certainly not support the use of excision arthroplasty as a first line of treatment in these patients. Other authors prefer a more positive approach to treatment. In two series, one of 15 cats (Jeffery, 1989) and another of 13 cats (Culvenor *et al.*, 1996), excellent results were achieved following fixation of femoral head or neck fractures using two or three diverging Kirschner wires (0.8 and 1 mm wires).

Femoral neck metaphyseal osteopathy in the cat

Femoral neck metaphyseal osteopathy is a relatively new disease (at least in terms of being recognised) in cats and is characterised by primary bone resorption and secondary pathological fracture of the femoral neck (Queen *et al.*, 1998). The condition has some similarities with Legg-Calvé-Perthes' disease, traumatic fracture of the femoral neck, canine metaphyseal osteopathy, bacterial osteomyelitis and viral osteomyelitis but the aetiopathogenesis has still to be established. In the series reported by Queen *et al.* (1998) age at onset ranged from 5 months to 2 years and lameness tended to be gradual in onset with no reports of trauma. All cases were treated by excision arthroplasty and were sound within 3 months.

Fracture of the greater trochanter

Solitary fractures of the greater trochanter are uncommon. In the immature animal, the fracture tends to occur through the trochanteric growth plate and is often accompanied by fracture separation of the capital femoral epiphysis (Denny, 1971). In the mature animal, the trochanteric fracture is usually associated with fracture of the femoral neck or dislocation of the hip. Tension band wiring, or lag screw fixation are the best methods of repairing the trochanteric fracture (Fig. 41.4).

Comminuted fractures involving the subtrochanteric region and the femoral neck

These fractures are challenging and are usually treated by plate fixation (Fig. 41.5). Initial fixation of the femoral neck fracture is achieved with a Kirschner wire. A lag screw is then placed parallel with the Kirschner wire. This screw is often

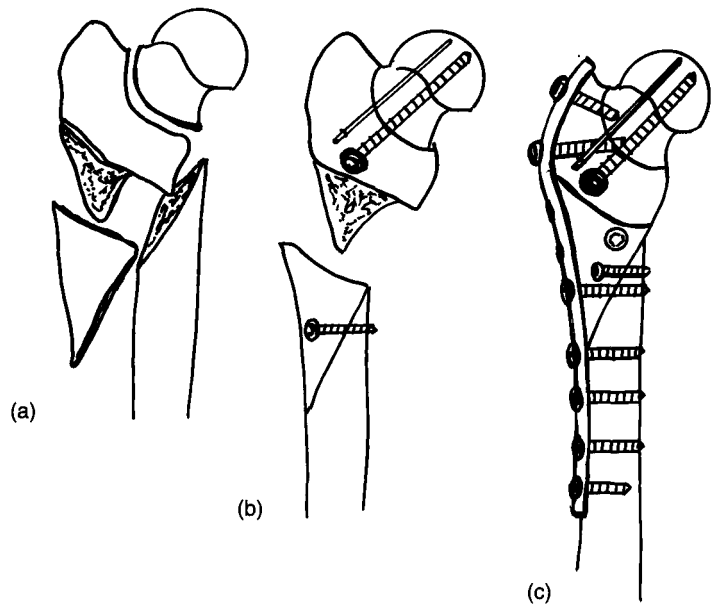


Fig. 41.5 (a) Comminuted fracture of the femoral neck and subtrochanteric region of the femur. (b) Initial reconstruction using Kirschner wire and lag screws. (c) Application of a neutralisation plate.

placed through the plate. Any other fragments in the subtrochanteric region are held in place with lag screws and/or Kirschner wires and the repair protected with a neutralisation plate. If the fragments are too small for reconstruction, they are left *in situ* and the plate is then used as a buttress plate to maintain bone length and joint alignment.

Diaphyseal fractures

The options and principles of managing diaphyseal fractures are outlined and discussed in Chapter 12 of this book and the reader is advised to read the relevant parts of that chapter in conjunction with the notes given here. Particular points with respect to the femur are detailed below.

Intramedullary fixation

Simple transverse fractures of the diaphysis in cats and young dogs are best treated by intramedullary fixation using a Steinmann pin(s) (Fig. 41.6) (see Chapter 12, Fig. 12.13). If necessary this can be combined with a unilateral external fixator to prevent rotational instability (see Fig. 12.14). Pins may also be used to treat comminuted, reconstructable fractures when combined with cerclage wires, particularly in small dogs and cats (see Fig. 12.15). They are best avoided in the treatment of comminuted, non-reconstructable fractures, which require buttressing, and also in the treatment of simpler fractures in medium- to large-breed, adult dogs since they are associated with a relatively high incidence of fracture complications in such cases. The pin may be introduced retrogradely through a lateral approach (described below under 'Plate fixation') of directly placing the pin just medial to the greater trochanter and driving it distally.

If an intramedullary pin is used for fixation in young puppies (4–5 months of age), open reduction must be undertaken with a minimum of soft tissue trauma as a common complication of diaphyseal fracture in dogs of this age is the formation of adhesions between the quadriceps and the femur resulting in *quadriceps contracture* and rigid extension of the stifle (see below). It should also be remembered that if a precut pin is used to treat a femoral diaphyseal fracture in a growing animal, rapid longitudinal growth of the bone often results in the pin becoming sealed within the medullary cavity which makes retrieval impossible. Consequently, if removal of an intramedullary pin is contemplated it is always important

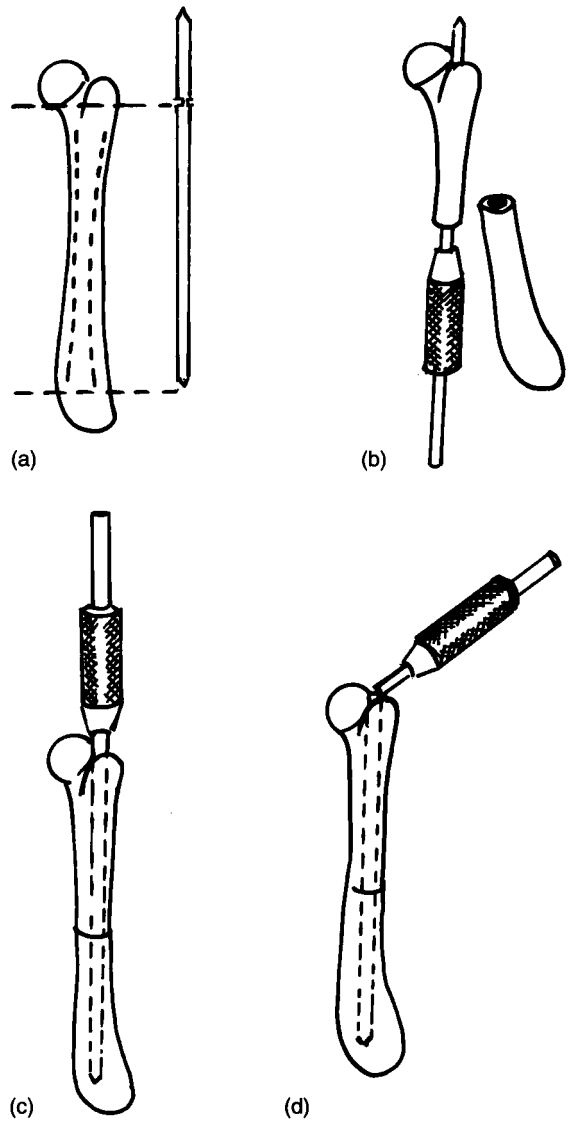


Fig. 41.6 The use of a Steinmann pin for fixation of a mid-diaphyseal fracture of the femur. (a) The pin is measured against the radiograph of a normal femur and precut. (b) Retrograde introduction of the pin up the proximal femoral shaft using a chuck so that the pin emerges in the trochanteric fossa. (c) The position of the chuck is reversed, the fracture is reduced and the pin is driven down into the distal shaft of the femur. (d) The pin is broken off flush in the trochanteric fossa.

to take radiographs to check the position of the pin and that fracture healing is complete.

The interlocking nail is a useful method of intramedullary fixation in the femur (Dueland *et al.*, 1996; Durall & Diaz, 1996) and can be used to prevent rotational instability and also to maintain bone length and alignment (i.e. buttress the fracture) in comminuted fractures (see Chapter 12, Fig. 12.18).

Plate fixation

Plate and screw fixation is used for comminuted and oblique fractures especially in medium to large breeds of dog. The plate (compression, neutralisation or buttress) is usually applied to the lateral aspect of the femur.

Surgical approach to the femoral diaphysis

The femur is exposed by a lateral skin incision extending from the greater trochanter to the stifle. The fascia lata is incised just cranial to the biceps femoris and blunt dissection between the vastus lateralis and the biceps femoris muscles will reveal the femoral diaphysis (Fig. 41.7a). The incision can be extended distally into the joint capsule of the stifle to complete exposure of the distal femur if necessary. Exposure of the proximal femur is achieved by subperiosteal elevation and cranial reflection of the origin of the vastus lateralis muscle (Fig. 41.7b).

Repair of comminuted femoral diaphyseal fracture using lag screws and a neutralisation plate

The stages in reconstruction of the femoral diaphysis following fracture are shown in Fig. 41.8. Soft tissue attachments to fragments are retained where possible. Ideally, lag screws are inserted in a craniocaudal or caudocranial direction so that they do not interfere with application of the plate to the lateral surface of the femur. However, due to the plane of the fracture, it is sometimes necessary to place lag screws in a lateromedial direction. Under these circumstances temporary reduction and fixation of the fragments can be accomplished with a cerclage wire (Fig. 41.9). The plate is applied and screws inserted through the

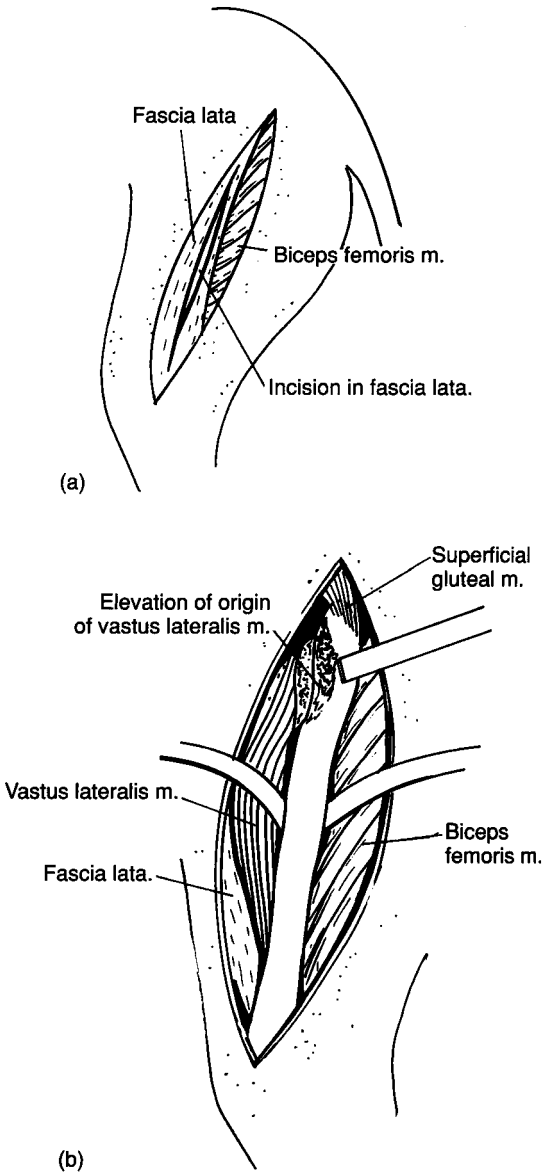


Fig. 41.7 Exposure of the femur. (a) Skin incision and incision of the fascia lata. (b) The biceps femoris muscle is retracted caudally and the vastus lateralis is retracted cranially to expose the femoral shaft. Further exposure of the proximal femur is achieved by subperiosteal elevation of the origin of the vastus lateralis muscle. Exposure of the distal femur is achieved by extending the incision in the fascia lata into the joint capsule.

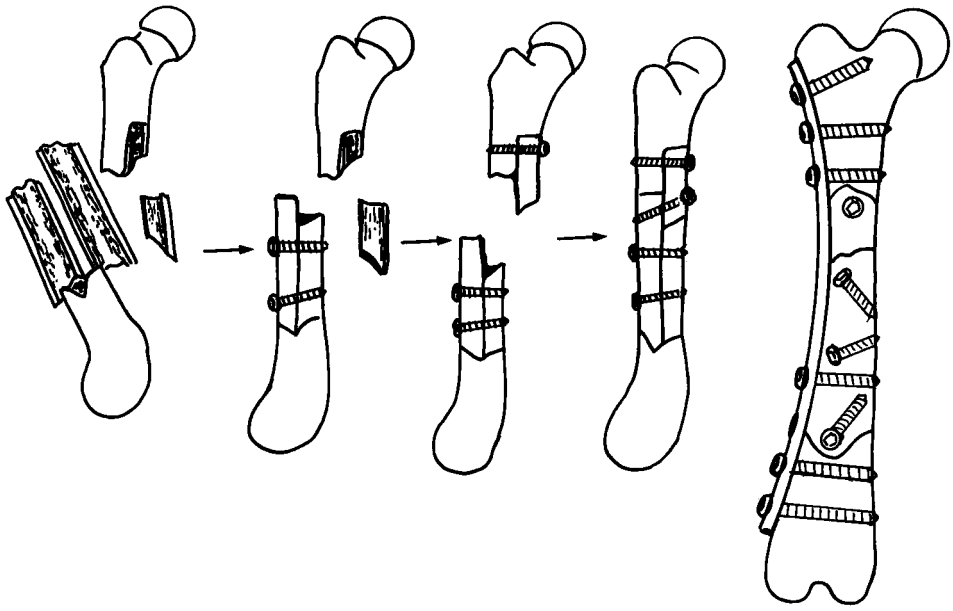


Fig. 41.8 Stages in the reconstruction of a comminuted fracture of the femur using lag screws and a neutralisation plate.

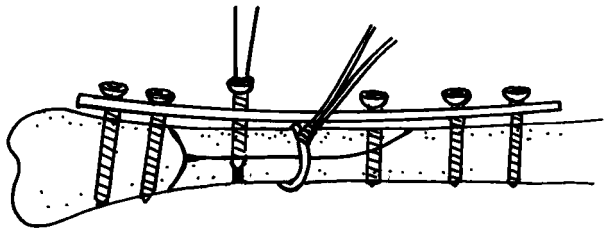


Fig. 41.9 Comminuted fracture of the femur. Fragment reduction is maintained using a temporary or permanent cerclage wire to allow placement of a lag screw through the plate and the fragments.

plate are used to lag fragments together. The cerclage wire can be removed before the screws are finally tightened or it can be left *in situ* to provide extra stability.

Segmental fractures of the femur are sometimes encountered. Although these may be stabilised with an intramedullary pin, there is a risk of rotation at one or more of the fracture sites. This problem can be overcome by the use of the interlocking nail or by the use of a dynamic

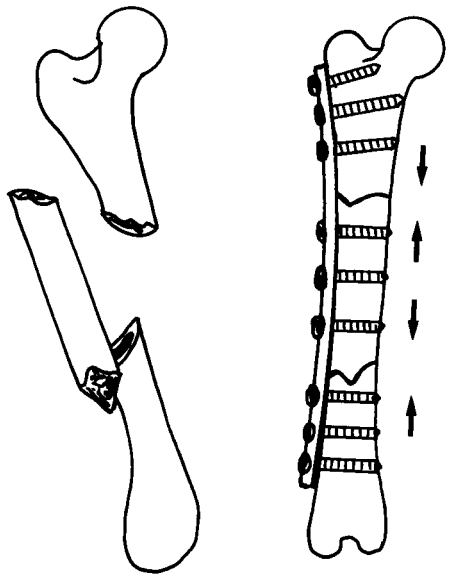


Fig. 41.10 Segmental fracture of the femur. Axial compression can be achieved at both fracture sites using a dynamic compression plate (DCP).

compression plate (DCP). The DCP is an ideal method of fixation and permits axial compression at both fractures sites (Fig. 41.10).

Plate-rod system

This method can be adapted to comminuted fractures in both dogs and cats (see Chapter 12, Fig. 12.41).

External skeletal fixator (ESF)

If an external skeletal fixator (ESF) is used as the only method of fixation it should be reserved for femoral diaphyseal fractures in young small breeds of dog and cats (Brinker *et al.*, 1990). Contrary to the usual principles of application, the fixator pins should be placed in the femur using an open approach to avoid transfixion of large muscles. In medium-sized patients stability can be improved by combining the ESF with an intramedullary pin, in which case the latter can be bent over and 'tied in' to the external connecting bar (see Chapter 12, Fig. 12.12). The external fixator can only be applied to the lateral side of the femur. However, the distal pin can sometimes be incorporated into a modified, bilateral frame.

Femoral diaphyseal fractures in cats

As a general rule, in cats, virtually all types of fracture, even severely comminuted fractures of the diaphysis, can be successfully treated by the use of intramedullary fixation used in conjunction with cerclage wires as necessary (Fig. 41.11). If the fragments are too small for reconstruction using cerclage wires then they are left *in situ* and a buttress plate (2.7 mm DCP, Stratec) is applied to maintain length and alignment (Fig. 41.12). Alternatively, an external fixator can be used in the same manner.

Contracture of the quadriceps femoris muscles (Vaughan, 1979)

Quadriceps contracture can occur as a congenital deformity or as a complication of femoral shaft fractures in puppies. The latter is seen most frequently. Splints (Thomas extension) or casts, which fit tightly round the mid-thigh, may cause muscle ischaemia leading to contracture. Alternatively, internal fixation of femoral diaphyseal fractures may be followed by adhesion of the quadriceps to the fracture site.

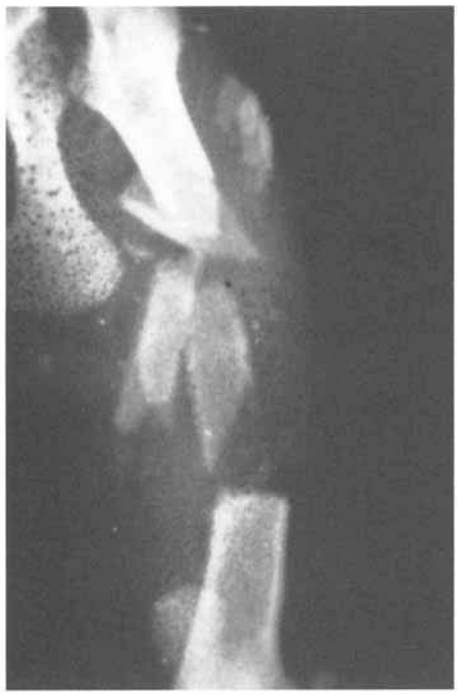
The clinical features of quadriceps contracture are:

- Rigid extension of the stifle
- Hyperextension of the hock
- The foot tends to be dragged, giving rise to excoriation of the dorsum
- The quadriceps become fibrous and taut

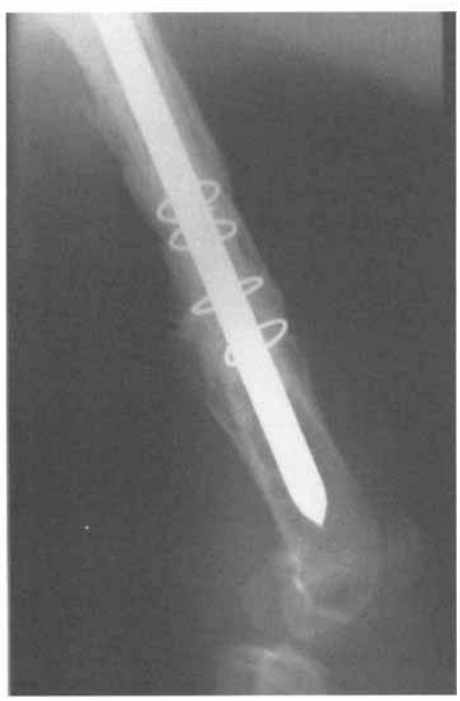
A lateral radiograph of the stifle shows the patella riding much further proximal to the trochlear than normal. Congenital contracture leads to genu recurvatum (see Chapter 42, p. 516).

Treatment

If adhesions have formed between the quadriceps and the femur then surgical release and vigorous physiotherapy may improve the range of stifle movement. Prognosis is very guarded once contracture has developed. Treatment involves



(a)



(b)

Fig. 41.11 (a) A severely comminuted femur fracture in a 4-year-old cat. (b) Follow-up radiograph 3 months after fracture repair using an intramedullary pin and cerclage wires. The fracture has healed. (Reproduced from Denny, H.R. (1991) *In Practice*, July, 141. With permission from BVA Publications.)

Fig. 41.12 Comminuted fracture in a cat. Buttress plate fixation with fragments left *in situ*.

quadricepsplasty, i.e. complete section of the muscles just proximal to the patella and then the application of an ESF to maintain the stifle and hock in flexion. The fixator is removed after 3–4 weeks and then vigorous physiotherpy is commenced to improve the range of stifle movement. Arthrodesis of the stifle, fixing the joint at a functional angle of 140° (see Chapter 42, p. 549), can also be used as a salvage procedure to improve limb function.

Fractures of the distal femur

Fractures involving the distal femoral growth plate – supracondylar fractures

Supracondylar fractures of the femur are common. The injury tends to be seen in puppies between 3 and 10 months of age and typically trauma causes separation or fracture–separation through the distal femoral growth plate (Salter Harris type I or II injury) with caudal displacement of the distal femoral epiphysis. There is pain and crepitus on manipulation but gross instability is not always a feature and such fractures can sometimes be missed on a cursory examination. The fracture should be obvious on radiographic examination (Fig. 41.13).

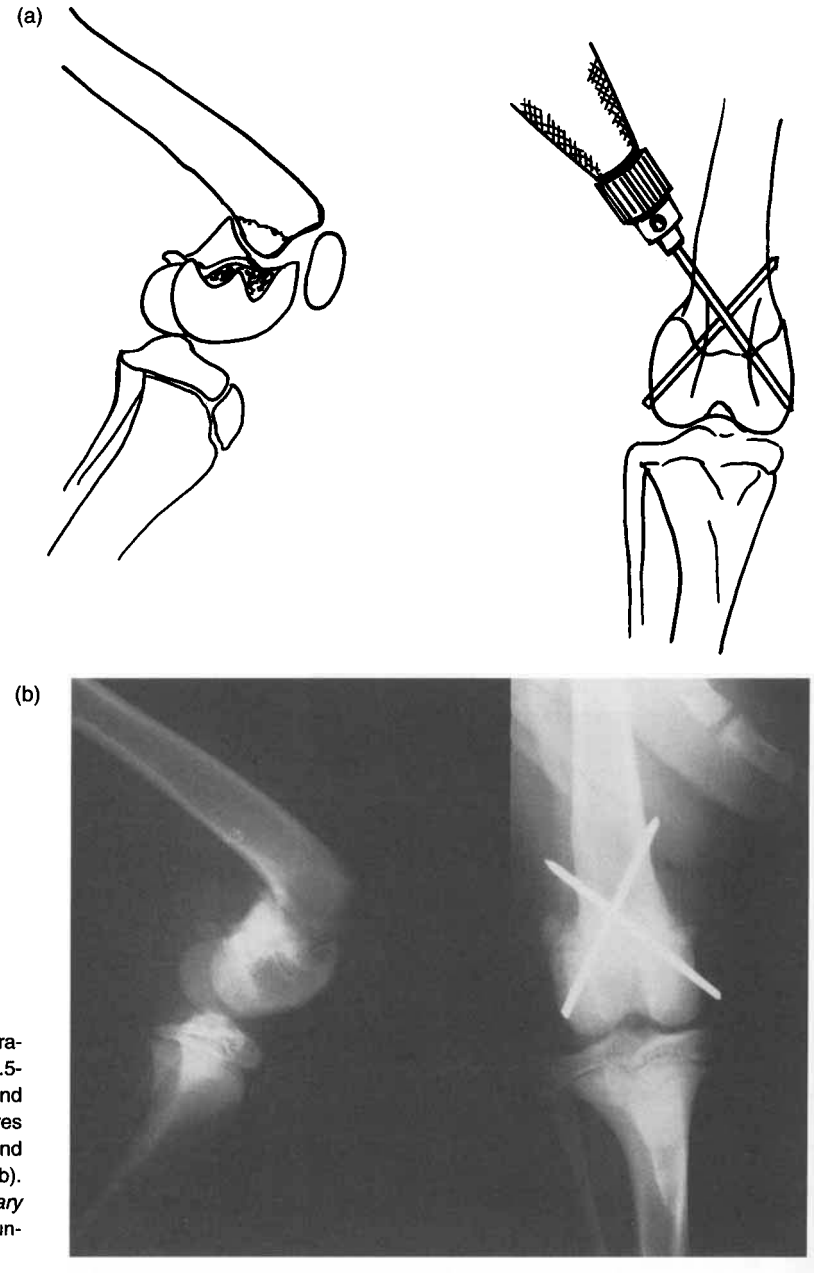


Fig. 41.13 Salter Harris type 2 (supracondylar) fracture of the femur in a 4.5-month-old Springer Spaniel. Fracture and fixation using crossed Kirschner wires are illustrated by line drawings (a) and pre- and postoperative radiographs (b). (Reproduced from *British Veterinary Journal* with permission from W.B. Saunders Company Ltd.)

Open reduction is essential to prevent caudal rotation of the femoral condyles and malunion. In puppies and kittens under 6 months of age crossed Kirschner wires are used for fixation (Sumner-Smith & Dingwall, 1973) (Fig. 41.13). This method should have minimal effect on longitudinal growth of the bone and has been shown to be biomechanically superior to other documented techniques *ex vivo* (Sukhiani & Holmberg, 1997). Care should be taken during reduction to protect the germinal cells of the growth plate by avoiding leverage on the epiphyseal side of the fracture.

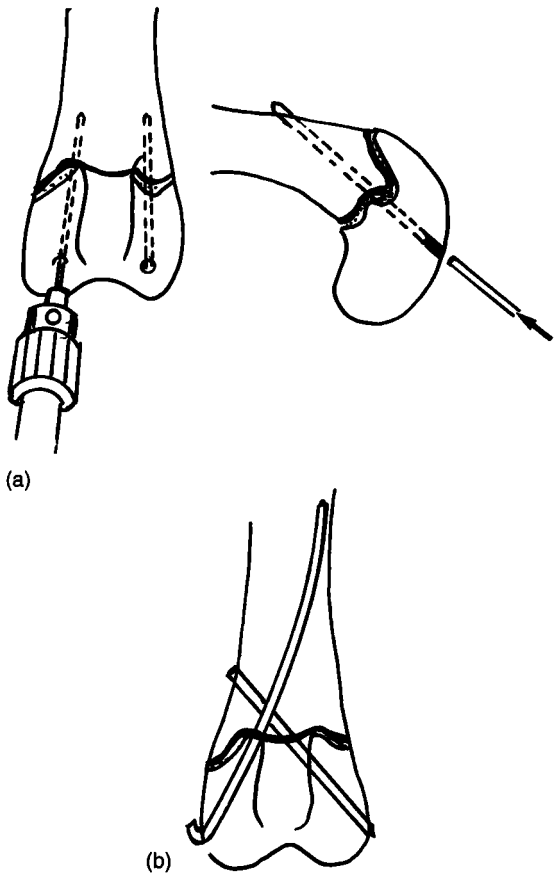


Fig. 41.14 Alternative methods of fixation using Kirschner wires in supracondylar fractures of the femur. (a) Parallel Kirschner wires introduced from the articular surfaces and then countersunk. (b) First Kirschner wire introduced in 'Rush pin' fashion, and the second Kirschner wire placed as a crossed pin.

Two alternative methods of placing Kirschner wires are illustrated in Fig. 41.14. These are aimed at further reducing the influence of the wires on longitudinal growth by avoiding each pin from being transfixed in two cortices (as is the case with crossed wires). The technique illustrated in Fig. 41.14b also requires only lateral exposure of the femur and leaves only one pin end within the joint capsule. In dogs over 7 months of age, with limited growth potential left, a single lag screw placed obliquely across the fracture site (Fig. 41.15) provides optimal stability (Knight, 1956; Hinko, 1974). The method should not be used in younger dogs as it will cause premature closure of the growth plate. However, closure of the physis may occur despite the surgeon's best efforts and whatever technique is used (see Chapter 15), although this is rarely of clinical significance.

Exposure

The fracture is exposed through a lateral parapatellar arthrotomy incision and the patella, if it is not already displaced, is reflected to reveal the fracture site. Reduction is not always easy. Pointed AO reduction forceps are ideal for gripping the femoral condyles to exert traction (Fig. 41.16). They are also used to maintain reduction of the fracture while the implants are inserted. A small hole is drilled in the cranial cortex of the femur just proximal to the trochlea, one point of the reduction forceps is inserted into the hole and the other point is inserted in the intercondylar fossa (Fig. 41.16). The forceps are locked, maintaining reduction.

Failure to treat a supracondylar fracture by internal fixation usually results in ankylosis of the stifle because the condyles rotate caudally and the distal shaft of the femur is displaced cranially, becoming incorporated in a mass of callus at the fracture site.

Single condylar fractures of the distal femur

These fractures are relatively uncommon in dogs and cats. Carmichael *et al.* (1989) described a series of nine cases in dogs. The medial condyle fractured in eight of these. The fracture is caused by trauma. Seven of the cases described had been caught and suspended by the affected leg and then struggled to free themselves. Diagnosis is confirmed by radiography with the lateral view being the most useful to demonstrate caudal displacement of the fractured condyle. Early open reduction and fixation of the condyle using a combination of a lag screw plus Kirschner wire is recommended (Fig. 41.17). The prognosis is good provided accurate reduction and rigid fixation is achieved. The same technique is used to repair single condylar fractures in the cat.

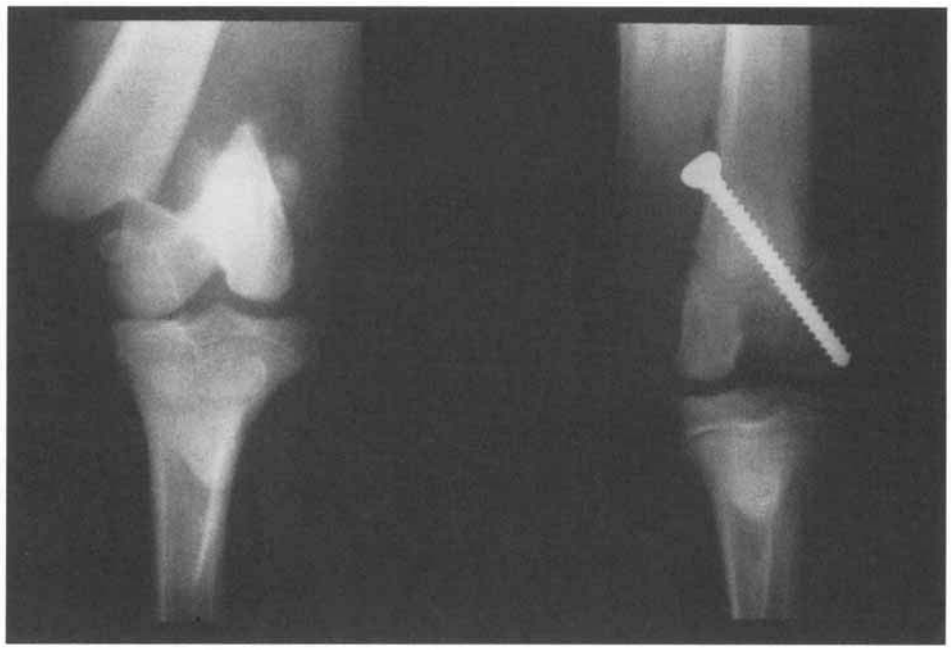


Fig. 41.15 Supracondylar fracture of the femur in a 7-month-old Border Collie. Pre- and postoperative radiographs showing lag screw fixation. This method should not be used for fixation of this type of fracture in dogs under 6 months of age. (Reproduced from *British Veterinary Journal* with permission from W.B. Saunders Company Ltd.)

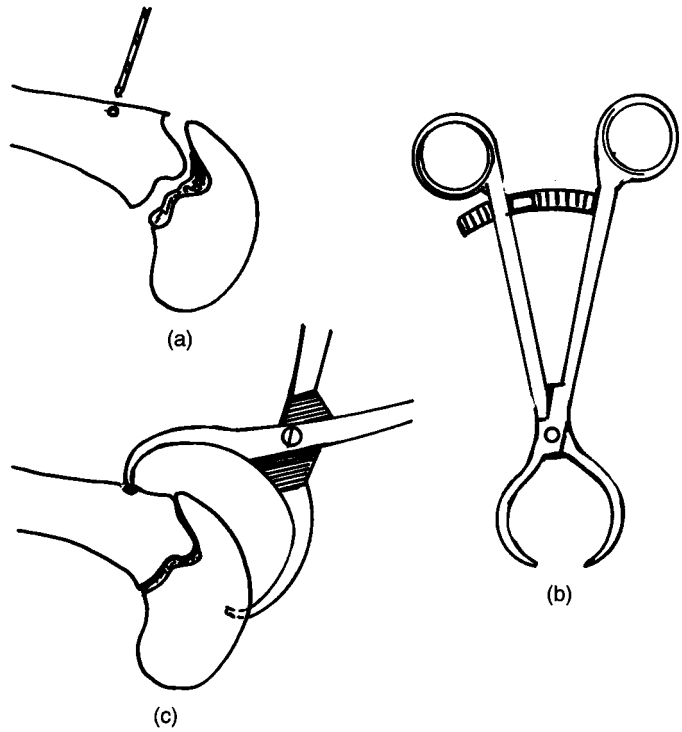


Fig. 41.16 Application of reduction pointed forceps in supracondylar fractures. (a) A small hole is drilled in the cranial cortex of the femur to improve purchase of the forceps. (b) Pointed reduction forceps. (c) Reduction is maintained by applying the forceps with one point in the hole in the cranial cortex of the femur and the other point in the intercondylar fossa. (Source: *British Veterinary Journal*, vol. 141.)

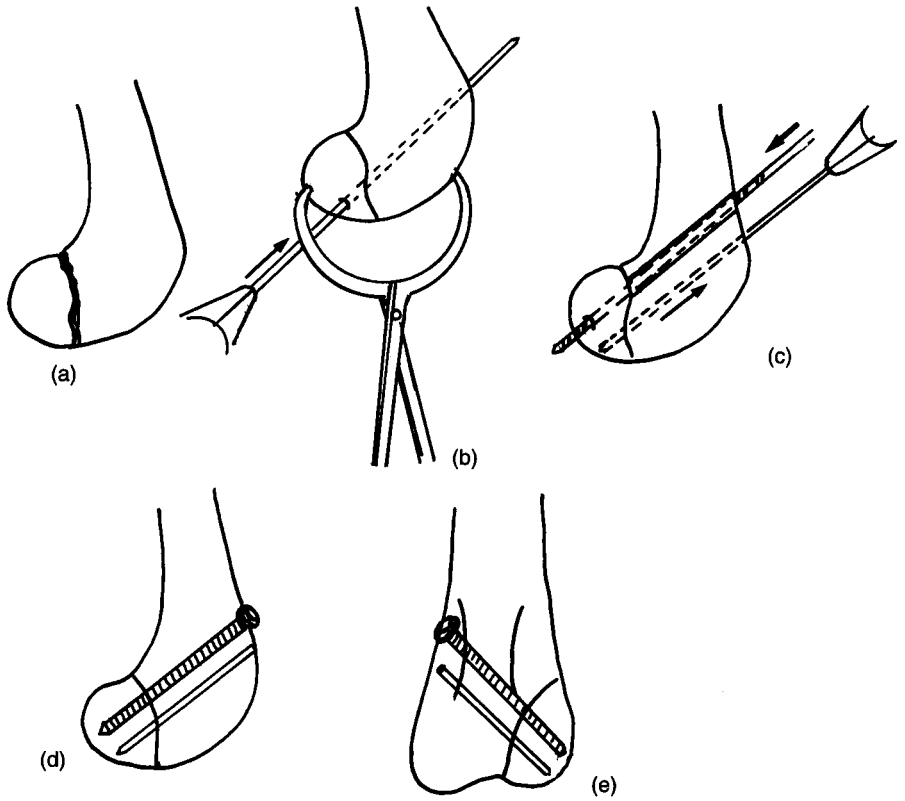


Fig. 41.17 Fixation of a fracture of the medial femoral condyle. (a) Fracture site. (b) Medial approach to expose the fracture. Following reduction a Kirschner wire is driven from the articular surface of the condyle until it emerges on the craniolateral side of the femur (e). (c) The drill is reversed to withdraw the Kirschner wire until the tip of the wire lies just below the surface of the articular surface of the medial condyle. A pilot hole is drilled for the lag screw from the femoral condyle, parallel with the Kirschner wire. The cranial section of the hole is overdrilled to produce the lag effect. A screw should be selected that is 2 mm shorter than indicated by the depth gauge. (d) The lag screw is inserted and tightened. (e) Craniocaudal view of the femur showing the position of the implants.

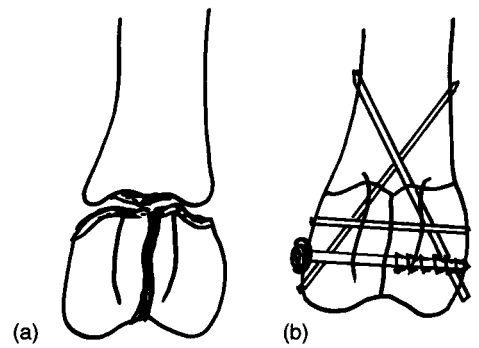


Fig. 41.18 (a) 'T' fracture or intercondylar fracture of the distal femur. (b) Fixation of the femoral condyles with transcondylar Kirschner wire and lag screw. Condyles are attached to the shaft with crossed Kirschner wires.

Fractures of both femoral condyles – intercondylar, or 'T', fracture

This is an uncommon injury in both the dog and the cat. The condyles are lagged together with a screw used in conjunction with a Kirschner wire to prevent rotation. The condyles are attached to the femoral shaft with crossed Kirschner wires (Figs 41.18 and 41.19) or a plate. Overall prognosis for dogs and cats with supracondylar or condylar fractures of the distal femur is good with appropriate treatment.

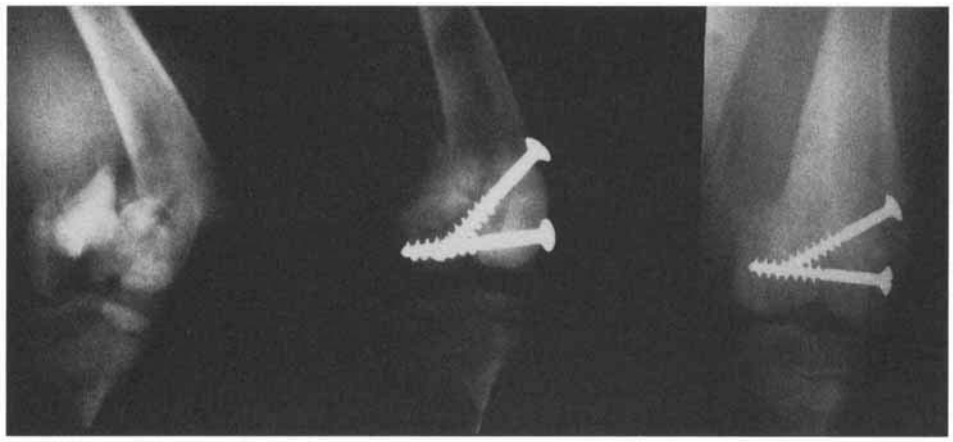


Fig. 41.19 (Left) 'T' fracture of the distal femur in a 4-month-old Labrador. (Centre) Reconstruction using lag screws. (Right) Fracture healing is complete at 4 weeks but notice that the placement of a screw across the growth plate has resulted in premature closure. The condyles should have been attached to the shaft using Kirschner wires to avoid this complication. (Reproduced from *British Veterinary Journal* with permission from W.B. Saunders Company Ltd.)

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Chapter 42
The Stifle

Anatomy

The stifle is a complex joint both anatomically (Fig. 42.1) and functionally. Although its primary motion is hinge-like, the menisci allow the femoral condyles to glide during movement so that the axis of rotation of the femur relative to the tibia varies according to the degree of flexion (Arnoczky & Marshall, 1977). Medial and lateral rotation of the tibia are also possible. The stifle consists of three interrelated joints: the femorotibial, the femoropatellar and the proximal tibiofibular joints. There are four sesamoid bones: the patella, medial and lateral fabellae, and the popliteal sesamoid. Primary ligamentous support for the stifle is provided by the medial and lateral collateral ligaments and the intra-articular cranial and caudal cruciate ligaments. Interposed between the femoral condyles and the tibial plateau are the medial and lateral menisci. The anatomical features of these various structures will be considered in more detail later in the chapter.

Conditions of the stifle joint

Congenital, or developmental, medial luxation of the patella and rupture of the cranial cruciate ligament are the main indications for surgery of the stifle but a variety of less common conditions can also cause lameness. These conditions are divided into four groups.

- *Developmental*
  - Osteochondrosis
  - Growth disturbances:
    - genu valgum
    - genu recurvatum
    - sloping tibial plateau
  - Patellar luxation (medial or lateral) and subluxation
  - Bipartite patella
  - Fabella abnormalities:
    - bipartite or multipartite
    - absent
    - displaced
- *Traumatic*
  - Fractures of the patella
  - Ruptured straight patellar ligament (tendon)
  - Fractures of the fabellae
  - Avulsion of the lateral head of the gastrocnemius muscle
  - Avulsion of the popliteus muscle
  - Avulsion of the long digital extensor tendon of origin
  - Displacement of the long digital extensor tendon
  - Avulsed tibial tuberosity (see Chapter 43)
  - Ruptured cranial cruciate ligament (+/– meniscal injury)
  - Ruptured caudal cruciate ligament
  - Ruptured collateral ligament
  - Multiple ligament injury
- *Acquired*
  - Cruciate disease (including meniscal injury)
  - Osteoarthritis (see Chapter 7)
  - Immune-mediated arthropathy (see Chapter 8)
  - Osteosarcoma (distal femur or proximal tibia, see Chapter 51)
- *Miscellaneous*
  - Gracilis rupture
  - Gracilis contracture

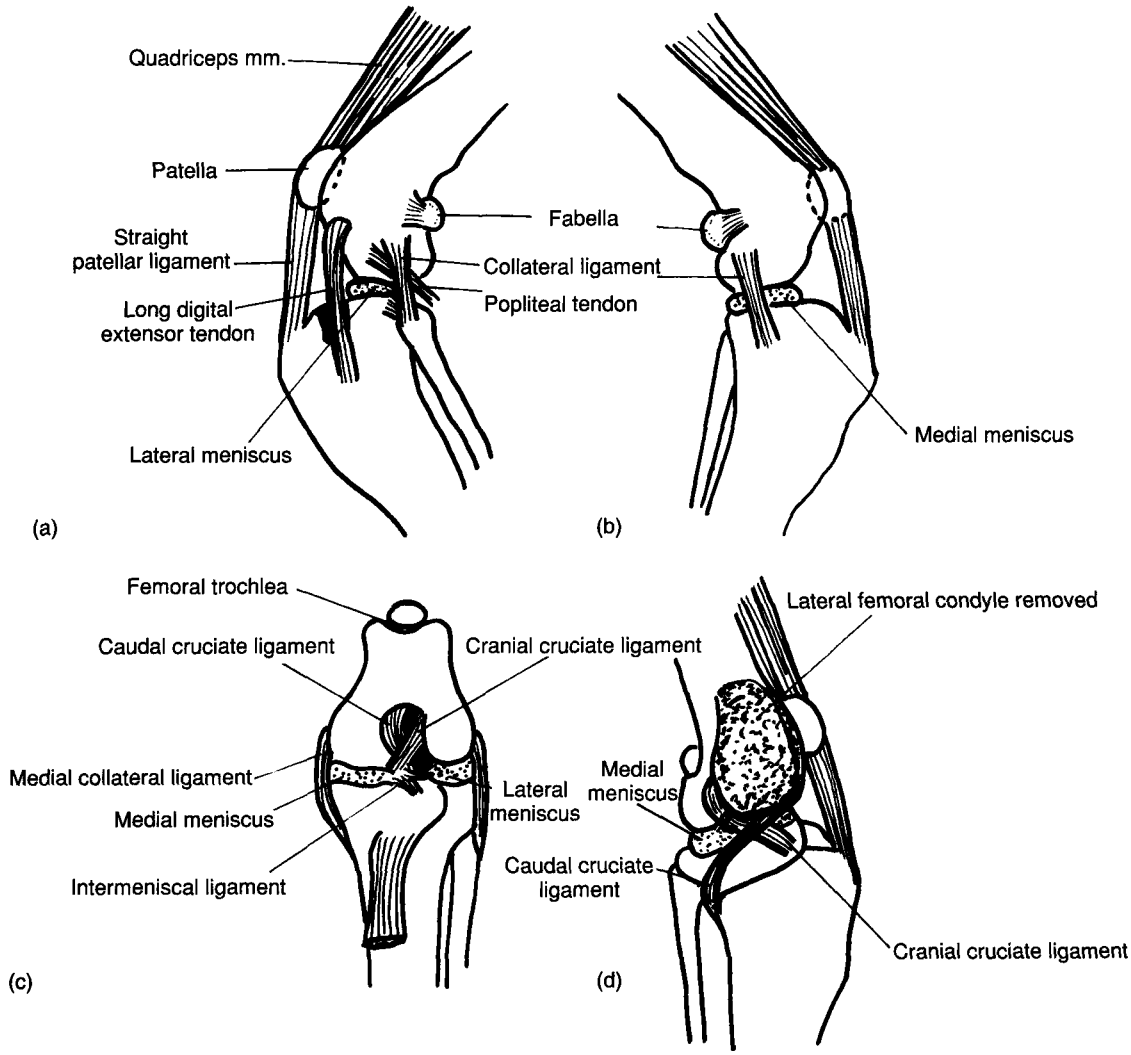


Fig. 42.1 Anatomy of the left stifle. (a) Lateral view, (b) medial view, (c) cranial view, (d) sagittal section.

Osteochondrosis

Osteochondritis dissecans (OCD) is a well-recognised, but not particularly common, cause of stifle lameness (Denny & Gibbs, 1980). The lesion is most often found in the axial articular surface of the lateral femoral condyle. Reported distribution of lesions between lateral and medial femoral condyles varies considerably, for example 96% of 141 cases reported by Montgomery *et al.* (1989) had lesions in the lateral condyle while in the series of 40 cases reported by Denny & Gibbs (1980) the distribution was more equal with the lateral condyle affected in 55% of cases and the medial condyle in 45%. Very occasionally an OCD lesion will be found in either the lateral femoral (Strom *et al.*, 1989) or the medial femoral trochlear ridge.

The condition is encountered most often in the Labrador Retriever and the Irish Wolfhound, but occurs in a variety of other breeds including the Staffordshire Bull Terrier, German Shepherd

Dog, Golden Retriever, Standard Poodle and Chow. Male dogs are more frequently affected than females. There is a gradual onset of lameness at about 5 months of age. Cases with bilateral lesions have a crouching gait and difficulty rising. There is discomfort and crepitus on manipulation of the stifle but no instability. Osteochondritis dissecans is generally associated with synovial effusion, and joint swelling may be appreciated on palpation, especially alongside the patellar ligament.

In the radiographic examination, two views (mediolateral and craniocaudal) should be taken of both stifles. A slightly oblique mediolateral projection (Fig. 42.2) is useful as it separates the femoral condyles and overcomes the problem of superimposition allowing comparison of the condylar outlines for evidence of flattening or an erosion. Which condylar outline belongs to the lateral condyle can usually be recognised by the presence of the notch from which the long digital extensor tendon arises. In the lateral view there may be evidence of synovial effusion with disruption of the outline of the infrapatellar fat pad. Calcified fragments ('joint mice') may be seen within the joint. Occasionally a fragment will find its way into the proximal femoral trochlear pouch, beneath the patellar tendon, and in this position the fragment can cause intermittent bouts of pain and lameness. The craniocaudal view of the stifle tends to be the most useful for demonstrating the OCD lesion and for determining which condyle is affected (Fig. 42.3).

Surgical treatment is recommended in most cases showing persistent lameness, allowing removal of the cartilaginous flap or 'joint mouse' and curettage of the underlying erosion. A standard, lateral parapatellar arthrotomy is used most often to expose and remove the detached cartilage. Limited stifle arthrotomy (incision from proximal patella to tibial tuberosity) has also been described (Shealy & Moilton, 1991) and is said to be quicker and less traumatic than standard arthrotomy. Arthroscopic surgical removal of stifle OCD has also been described (McLaughlin *et al.*, 1989). It should be noted, however, that the stifle, like the shoulder, has a large joint space to accommodate loose fragments of cartilage. Dogs with OCD affecting these joints can recover with conservative management although lameness may persist for several months. In a series of 40 cases of stifle OCD treated by one of the authors 20 out of 23 surgically treated cases became sound, while of the 17 cases treated conservatively

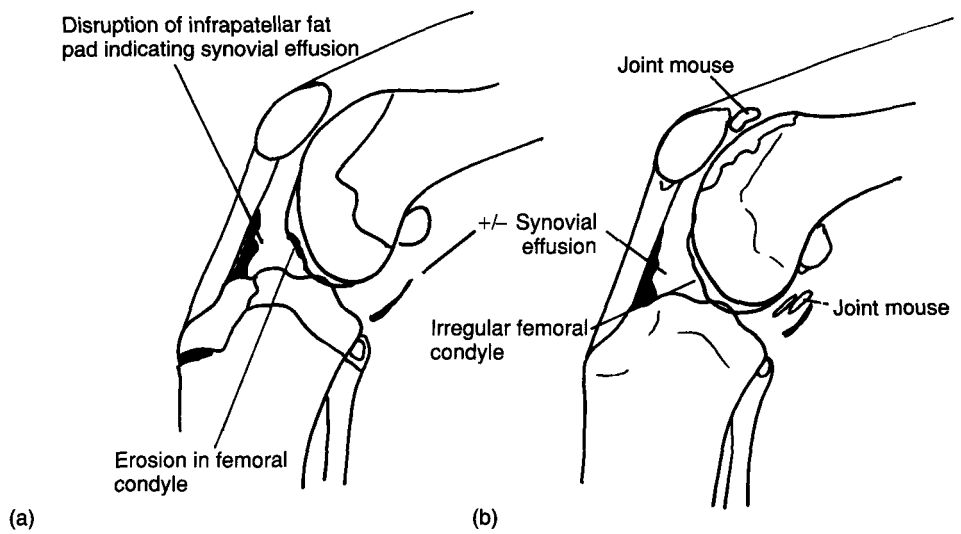
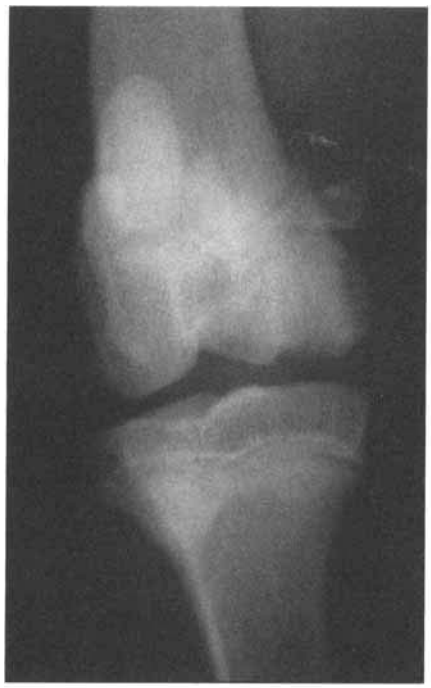
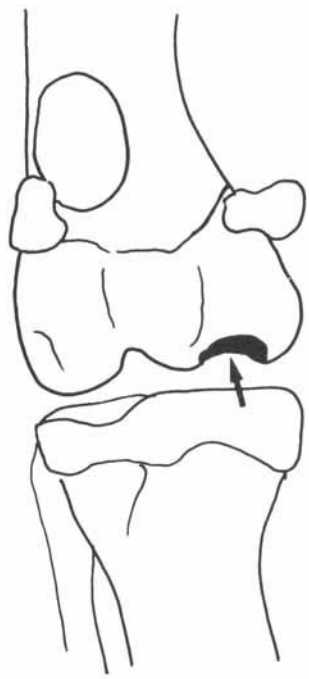


Fig. 42.2 Mediolateral projection of radiographic features of stifle osteochondritis dissecans (OCD). (a) Early features. (b) Late features.



(a)



(b)

Fig. 42.3 (a) Craniocaudal radiograph of the stifle of a 6-month-old Labrador with osteochondritis dissecans (OCD) of the medial femoral condyle. (Source: *British Veterinary Journal* vol. 141.) (b) Tracing of the radiograph showing position of the OCD lesion.

(restricted exercise and non-steroidal anti-inflammatory drugs [NSAIDs]), 12 became sound and 5 remained lame.

Growth disturbances

Genu valgum

The commonest growth disturbance affecting the distal femur and proximal tibia is known as *genu valgum*. The condition is seen in giant breeds of dog, especially Great Danes, Irish Wolfhounds, English Mastiffs and St Bernards. Average age at onset is 5 months and the condition is often bilateral. There is medial bowing of the distal femur so the stifles tend to knock together and the lower leg is turned out. Such dogs are often referred to as having 'knock knees' or 'cow hocks'. There is sometimes a tendency for the patella to luxate laterally. Genu valgum is usually the result of a distal femoral growth plate disturbance. The medial side grows more rapidly than the lateral producing medial bowing of the distal femur (Fig. 42.4a). The proximal tibial growth plate can also be involved and in some cases the main site of deformity appears to be the proximal tibia. The deformity can be corrected by stapling (Fig. 42.4b) provided the pup still has plenty of growth potential left. The staple is positioned to bridge the medial side of the growth plate. To aid in positioning the staple, the leg is first prepared for surgery and two or three 19 gauge needles are placed on the medial side of the stifle (Fig. 42.4c) followed by radiography. These needles serve as landmarks to locate the growth plate while the staple is being inserted. The staple temporarily impedes growth on the medial side of the growth plate while continued growth on the lateral side gradually straightens the leg in 4–6 weeks. The staple(s) is/are removed as soon as the leg is straight.

A method of correcting angular limb deformities which is used very successfully in foals and has limited application in the dog is the periosteal strip procedure (Auer *et al.*, 1982). Unlike stapling, which temporarily impedes growth, the periosteal strip procedure stimulates bone growth. Consequently, it is done on the lateral or

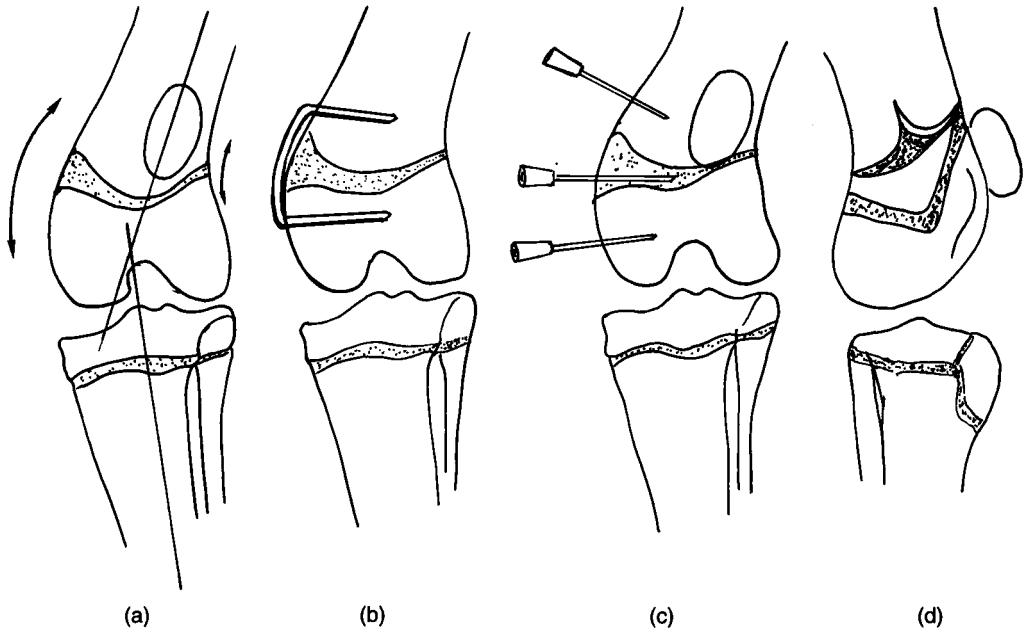


Fig. 42.4 Genu valgum. (a) Genu valgum is associated with a distal femoral growth plate disturbance. (b) A staple is used to bridge the medial side of the distal femoral growth plate. (c) Marker needles are placed on the medial side of the femur. (d) The periosteal strip procedure used on the lateral side of the femur to promote bone growth.

concave side. A transverse, inverted T-shaped incision is made through the periosteum just proximal to the growth plate and the periosteum is elevated (Fig. 42.4d). The leg should straighten within 4–8 weeks.

Genu recurvatum

Genu recurvatum (Vaughan, 1979) is a stifle deformity which results from contracture of the quadriceps muscles (Fig. 42.5). The condition can occur as a congenital deformity or as a complication of femoral diaphyseal fractures in puppies (see Chapter 41). The clinical features include:

- Rigid extension of the stifle
- Hyperextension of the hock
- The foot tends to be dragged giving excoriation of the dorsum
- The quadriceps become fibrous and taut

The prognosis is poor and the response to physiotherapy, surgical release of adhesions or section of the quadriceps is generally disappointing. Section of the quadriceps tendon with immobilisation of the stifle and hock in maximum possible flexion using an external fixator for 3 weeks, followed by physiotherapy after removal of the fixator, gives the best chance of improving the range of stifle flexion.

Degenerative joint disease of the stifle associated with a proximal tibial growth plate disturbance

In 1982, Read & Robins described several cases of degenerative joint disease affecting the stifle associated with a proximal tibial growth disturbance. In the normal dog, the tibial angle formed between the proximal tibial plateau and the long axis of the tibial shaft is 69.5° (Fig. 42.6). If the tibial angle is less than 69° then cranial tibial thrust on the cranial cruciate ligament is increased causing early breakdown of the ligament and degenerative joint disease in the stifle. An abnormal tibial angle is associated with overgrowth of the cranial half of the tibial growth plate and pre-

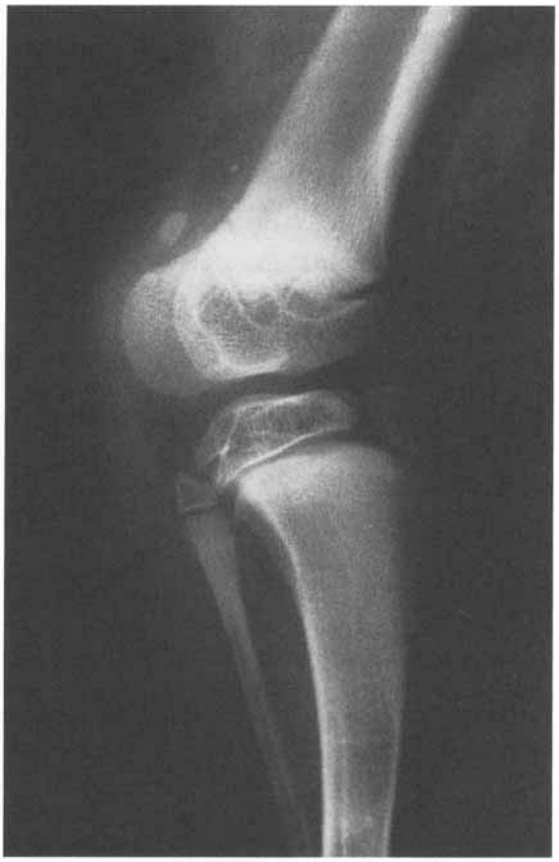


Fig. 42.5 Lateral radiograph of the stifle of a 14-week-old Afghan Hound with congenital genu recurvatum. (Reproduced from *British Veterinary Journal* with permission from W.B. Saunders Company Ltd.)

mature closure of the caudal half of the tibial growth plate. The deformity is probably traumatic in origin although it has been hypothesised that it may be an inherent phenotype that contributes to cruciate ligament disease in some dogs (Slocum & Devine, 1984). Uneven closure causes cranial bowing of the tibia, cranial cruciate ligament rupture and degenerative joint disease. Surgical treatment of the cruciate ligament rupture gives poor results in these cases. However, if a wedge osteotomy is performed to correct the abnormal tibial angle (Fig. 42.7) then an improvement in the dog's limb function occurs within 3 months and follow-up radiographs show remodelling of periarticular osteophytes (which may or may not suggest less active disease).

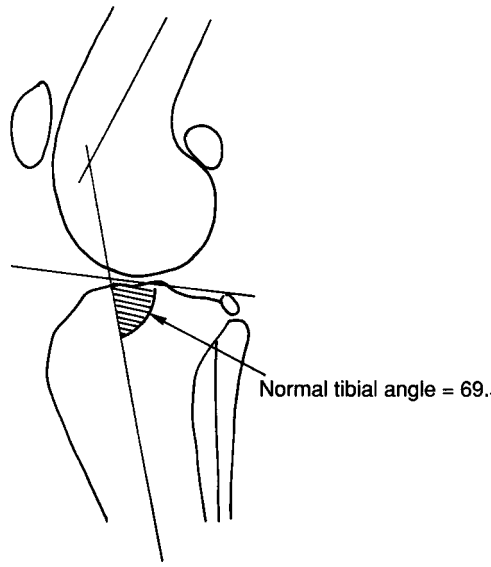


Fig. 42.6 Lateral view of the stifle showing the normal angle of the tibial plateau.

Patellar luxation

Aetiopathogenesis

Luxation of the patella may be congenital/developmental or traumatic in origin. The latter can affect any breed and usually follows a blow to the lateral aspect of the stifle causing damage to the lateral retinaculum, resulting in instability and medial luxation of the patella. The cause of congenital patellar luxation has not been definitively established. It is most commonly seen in small breeds, such as Miniature and Toy Poodles, Cavalier King Charles Spaniels, Yorkshire Terriers, Chihuahuas and Griffons. However, it may also be seen in larger breeds, such as Boxers, Flat Coated Retrievers and Labrador Retrievers, and also giant breeds, such as the St Bernard, where a lateral luxation may be associated with genu valgum. Cats may also be afflicted with this condition, either as a congenital problem, when it is frequently asymptomatic, or else in association with coxofemoral luxation. In dogs with longstanding, asymptomatic patellar luxation, lameness may develop due to cruciate injury which may be predisposed to by the lack of cranial

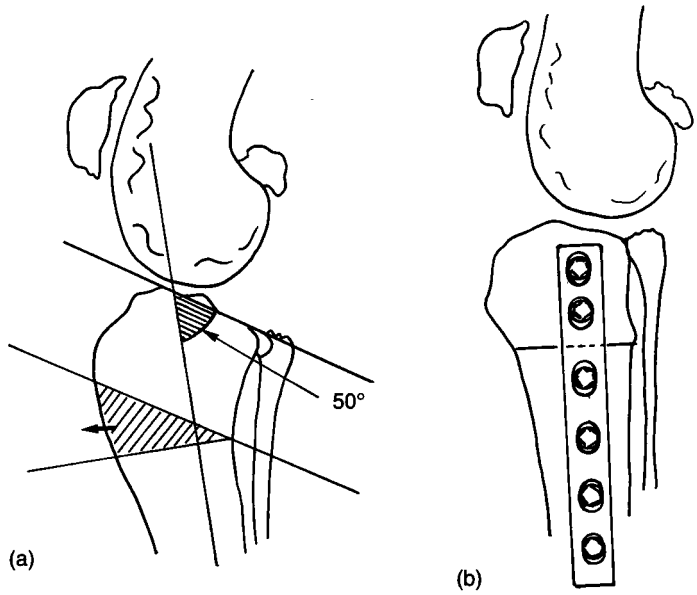


Fig. 42.7 Degenerative joint disease associated with abnormal tibial plateau angle (Read & Robins, 1982). (a) Tracing of a preoperative lateral radiograph of the stifle in a Greyhound. The tibial angle is 50° and there is secondary arthritis. (b) Tracing of a postoperative radiograph illustrating corrective osteotomy.

support which the quadriceps complex may offer the normal stifle joint (though because the quadriceps is a stifle extensor it is more likely to create hyperextension, which may damage the cranial cruciate ligament, than to protect against it). In treating such cases the patellar luxation may be more significant than the secondary cruciate injury, depending on the size of the dog.

Patellar subluxation may be recognised in some dogs, particularly the bull terrier breeds. In these cases the patella cannot be displaced from the trochlear groove and no lameness is seen until adulthood. Malalignment of the quadriceps complex causes the patella to ride up on the medial trochlear ridge. With time the articular cartilage on the ridge, and/or the patella, becomes worn away and subchondral bone is exposed leading to pain and resulting in lameness.

There can be little doubt that the congenital form of patellar luxation is related to developmental abnormalities in the limb that create malalignment of the quadriceps complex (quadriceps muscle + patella + patella ligament/tendon). Underlying anatomical abnormalities of the hip and/or stifle joints, seen in association with congenital medial patellar luxation, have been proposed and include the following:

- Retroversion of the femoral head and neck
- Alteration of the angle of inclination (coxa vara)
- Lateral rotation and bowing of the distal femur
- A shallow trochlear groove with a poorly developed medial ridge
- Dysplasia of the distal femoral epiphysis
- Rotation and lateral laxity of the femorotibial joint
- Medial bowing and rotation of the proximal tibia
- Medial deviation of the tibial tuberosity
- Outward rotation of the hock
- Reduced growth of the limb

In the case of congenital, lateral luxation the changes would be similar but in the opposite sense, possibly with physeal abnormalities in the cases with genu valgum. The radiographic abnormalities noted in the hip have been brought into question by the suggestion that they are artifacts resulting from an inability to correctly position a patient, with respect to the coxofemoral joints, in those with abnormalities causing patellar luxation.

The abnormalities seen in a particular case are

not static and the number may increase with time. For example, a laterally bowed distal femur might allow medial patellar luxation. As this repeatedly luxates, the medial trochlear ridge may become worn down, thereby increasing the frequency of luxation. With the patella in the trochlear groove for a reduced amount of time, this will not develop properly and will become shallow. With the patella positioned medially it will create medial traction on the tibial tuberosity which will then begin to deviate medially as it develops. Thus, an abnormality causing an intermittent luxation may lead on to further deformity and permanent luxation.

There can be little doubt that this condition has a heritable component, since the majority of conformational abnormalities are genetically programmed, although it is likely to be a polygenic, multifactorial condition. With respect to advice regarding breeding, there is probably sufficient evidence to say that an affected animal should not be bred from, nor should the mating that produced that animal be repeated.

History and clinical signs

On presenting patients with congenital patellar luxation the owners may report an intermittent, 'skipping' lameness which 'switches on and off' several times during exercise and does not seem to trouble the dog unduly. With more permanent luxations the dog will tend to hold the limb in a semiflexed position unless the problem is bilateral, in which case a crouching stance and bunny-hopping gait are often adopted. Lateral congenital luxation appears to be more disabling than medial luxation. Patellar subluxation may result in a progressive stifle lameness which may worsen with exercise and cause stiffness after rest. In traumatic cases the lameness is acute in onset and tends to be non-weight-bearing initially with some improvement over the first few days. In some cases a long-standing, mild patellar instability may be exacerbated by trauma later in life, in which case quite major surgical correction may prove necessary after what appears to be a minor injury.

Clinical examination should allow exclusion of other possible causes of lameness, such as Legg-Perthes' disease, and any complicating factors, such as coxofemoral luxation or cruciate ligament failure. Manipulation of the stifle is often non-painful, except when erosions are present in the articular surface, e.g. with patellar subluxation, or when the soft tissues are being stretched during attempts to reduce a permanent luxation. There may be a decreased range of extension in the joint and this may affect the prognosis since, if this is marked, it may be associated with contracture of the soft tissues caudal to the joint or, in the immature patient particularly, with irreversible epiphyseal malformation. Such changes will affect the range of mobility achieved post-surgery and may compromise the result significantly.

Traditionally, Singleton's (1969) classification has been used to assess the degree of deformity associated with luxation and the type of treatment required. He divided animals into four grades, only the anatomical features are given here; the clinical features are essentially the same as Putnam's classification below:

- **Grade 1**: Intermittent patellar luxation. There is minimal stifle deformity or medial deviation of the tibial tuberosity.
- **Grade 2**: Frequent luxation of the patella associated with 15–30° medial deviation of the tibial tuberosity.
- **Grade 3**: Permanent medial luxation of the patella associated with 30–60° medial deviation of the tibial tuberosity. The femoral trochlear groove is usually shallow.
- **Grade 4**: Permanent medial luxation of the patella associated with 60–90° medial deviation of the tibial tuberosity. The femoral trochlear groove is absent or convex.

However, evaluation of the degree of rotation of the tibial tuberosity is very subjective and it may be considered more reliable to use the more straightforward descriptions of the grades provided by Putnam (1968):

- **Grade 1**: Intermittent patellar luxation with manual dislocation on full extension and spontaneous reduction on release.
- **Grade 2**: Frequent patellar luxation on flexion of the joint or digital

pressure where spontaneous reduction is not always immediate.

- **Grade 3**: Permanent patellar luxation where manual reduction is possible but spontaneous reluxation occurs on release.
- **Grade 4**: Permanent patellar luxation where manual reduction is not possible.

Although such grades may not correspond to the clinical signs, they may be of some use in monitoring progression in a young, asymptomatic patient, or else in planning what surgery is likely to be required in patients that are lame. Although the techniques required are often determined at surgery, the clinical grade can be of some help. For example: grade 1 luxations may be treated conservatively, if asymptomatic, or by reinforcement of the lateral retinaculum; a grade 1 or 2 luxation will not usually require medial capsular release; grade 2–4 luxations will require lateral transposition of the tibial tuberosity; grades 3 and 4 luxations will require medial retinacular release and, probably, deepening of the femoral trochlear groove.

Radiology

Radiography may help to confirm the clinical diagnosis by ruling out other causes of stifle lameness, showing the luxated patella, in the more severe cases, and to demonstrate the bony deformities present. A tangential view of the flexed stifle skylines the trochlear groove and allows assessment of its depth (see later in Fig. 42.12, relating to patellar fractures). Other features which may be observed include periarticular osteophytes indicative of osteoarthritic change, although this is often not present in small breeds of dog even when the problem is long-standing. However, much of this information can be gained from the clinical examination and final decisions regarding the corrective procedures necessary can be made at the time of surgery.

Treatment

Conservative management can be advocated only when instability of the patella is not associated with any clinical signs or else when lameness is seen infrequently. Exercise is to be encouraged in order to build up and maintain tone in the quadriceps muscle.

If patellar luxation is associated with recurrent or persistent clinical signs then surgery is indicated. The aim is to restore normal alignment of the quadriceps mechanism and there is no point in delaying surgery since this may lead to further bone deformity, necessitating more extensive surgery. In some cases, the patients present at a very early age and it may be worth waiting until they reach 5–6 months of age so that tissue handling is improved. Whether this is possible will, to some extent, depend on the severity of the deformity.

In considering the surgical techniques available in the management of medial patellar luxation, they may be grouped according to which of the following effects they are trying to create:

- Reinforcement of the lateral retinaculum
- Release of the medial retinaculum
- Deepening of the trochlear groove
- Transposition of the tibial tuberosity
- Corrective osteotomy

Often a combination of these procedures is required to correct the patellar luxation, and deformities should be corrected as they are found. For example, if the femoral trochlear groove is shallow then it is deepened but if it is normal then leave well alone. Not every dog which requires a lateral transposition of the tibial tuberosity will require the femoral trochlear groove to be deepened.

Reinforcement of the lateral retinaculum

Suturing of the torn lateral retinaculum may be all that is required to restore stability to a traumatic medial patellar luxation in the absence of any predisposing bony deformity. In congenital cases, various means of imbricating the lateral retinaculum by means of capsulectomy or capsular overlap (Fig. 42.8), or augmenting the lateral support by means of a fascia lata graft extending from the patella and around the lateral fabella, have been described. The use of sutures placed around the patella and lateral fabella, or else from the latter to the tibial crest, have also been advocated. In addition to tightening the lateral retinaculum, the

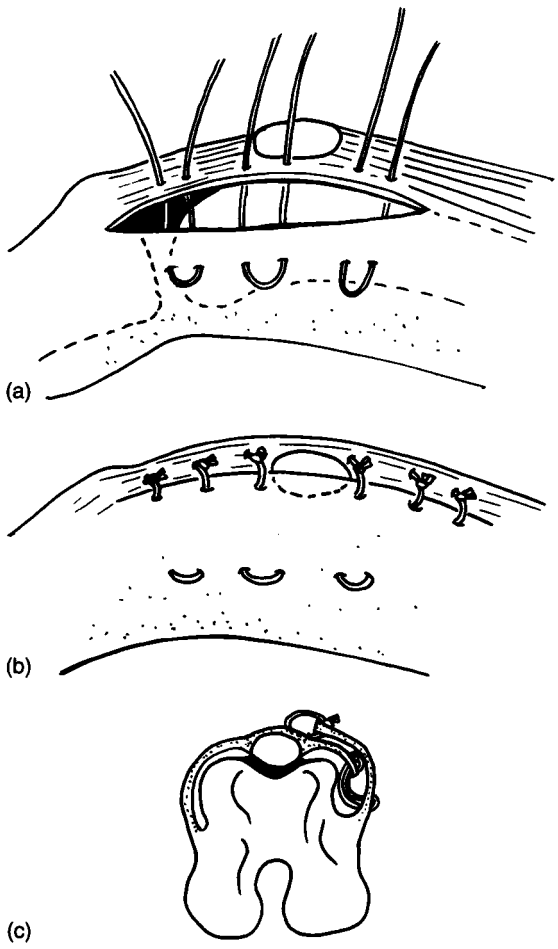


Fig. 42.8 Lateral capsular overlap in the treatment of medial luxation of the patella. (a) Lateral parapatellar arthrotomy, with the first row of sutures preplaced as horizontal or mayo mattress sutures. (b) The second row of sutures in overlap placed as simple interrupted sutures. (c) Transverse section through the stifle illustrating lateral capsular overlap.

alignment of the quadriceps pull can be altered by extending the lateral incision more proximally and suturing the edge of the biceps femoris further cranially on the quadriceps thus tending to pull the muscle belly further laterally and reducing any medially directed forces applied to the patella.

The problem with all these techniques is that they are directed at improving soft tissue support which all too often has developed as a consequence of underlying bony deformity. If this is the case then the imbricated tissues will stretch again, any prosthetic sutures will pull through the tissues or else break, and the luxation is liable to recur. Thus, such techniques can only be used alone successfully when no bony deformity is present. However, capsulorrhaphy is often used in combination with other techniques to accommodate the redundant soft tissues once the patellar luxation has been corrected.

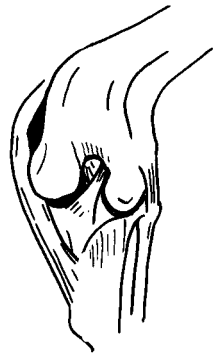
Release of the medial retinaculum

This technique is generally not used alone since it does not address the underlying problem. However, it may be necessary in order to allow replacement of the patella in the trochlea in cases of permanent luxation. In some patients it is necessary to develop extensive medial release involving sectioning of the insertion of the cranial sartorius muscle and continuing proximally along the border of the vastus medialis.

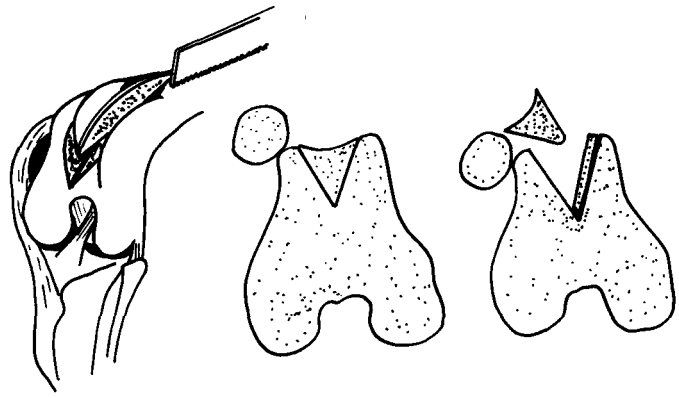
Deepening of the trochlear groove

A shallow (Fig. 42.9a) or absent trochlear groove, or a relatively poorly developed medial trochlear ridge may indicate a need to deepen the groove. Attempts to increase the height of or extend the medial ridge using non-absorbable implants have not generally found favour and most techniques revolve around the removal of tissue from the distal femur. Trochlear sulcoplasty (Fig. 42.9b) involves the removal of articular cartilage and underlying subchondral bone so as to create a sulcus that will become lined by fibrocartilage. Preservation of the articular cartilage may be achieved in dogs less than about 6–8 months of age by creating a flap of cartilage, which is left attached distally, removing subchondral bone from underneath, and then replacing the layer of articular cartilage. This technique is referred to as trochlear chondroplasty.

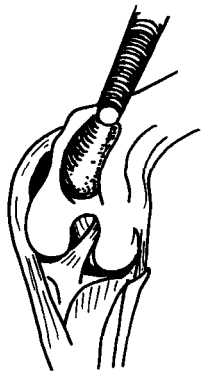
Latterly the technique of recession V-sulcoplasty (Fig. 42.9c) has been advocated (Boone *et al.*, 1983; Slocum & Devine, 1985). A V-shaped wedge, with the sulcus forming the base, is removed from the distal femur as an autogenous osteochondral graft. The V-shaped defect in the distal femur is deepened either by making a



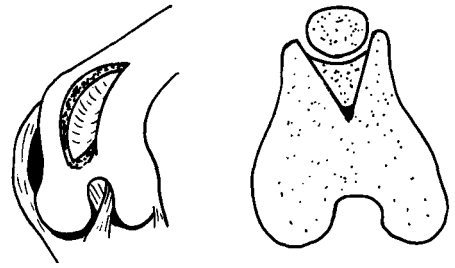
(a) Shallow trochlea.



(c) Recession V-sulcoplasty, wedge cut (Boone *et al.*, 1983).



(b) Trochlear sulcoplasty using a rasp. It heals by fibrocartilage formation.



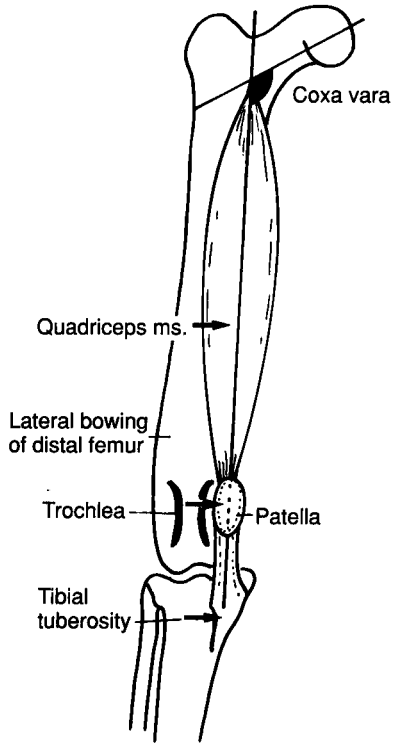
(d) The trochlear wedge lined by hyaline cartilage is seated more deeply and held in position by the patella.

Fig. 42.9 Deepening the femoral trochlear groove.

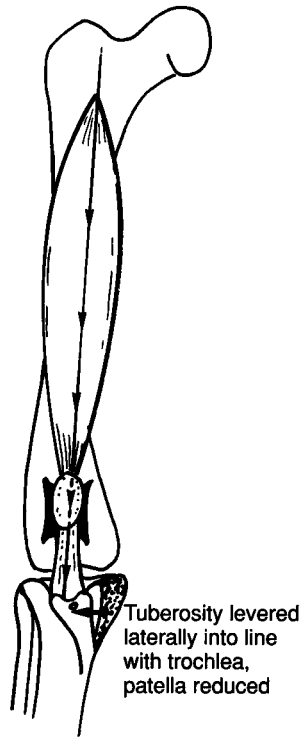
second wedge-shaped cut with sides parallel to the first but with a wider base, or, in the case of small breeds with medial luxation, removing a slice of bone from only the lateral wall of the V. The original wedge should fit into the defect, since they are similar triangles, with a degree of recession. Sometimes the wedge tends to 'rock' in the V because the cuts are not symmetrical and this is most easily resolved by cutting off the angle of the wedge made up of bone only (i.e. its apex). The sulcus so formed will ultimately have a hyaline cartilage floor and fibrocartilage walls. The wedge does not need to be held in place since it is retained in position by the compression created by the patella (Fig. 42.9d), and because the friction between the subchondral bone trabeculae is greater than the friction between the wedge and the patella. Because this technique is not limited by the size or age of the patient, and maintains a surface of hyaline cartilage on which the patella can move, it is, perhaps, the most appropriate technique to use in situations where the trochlear groove is insufficiently deep to allow patellar stability.

Transposition of the tibial tuberosity

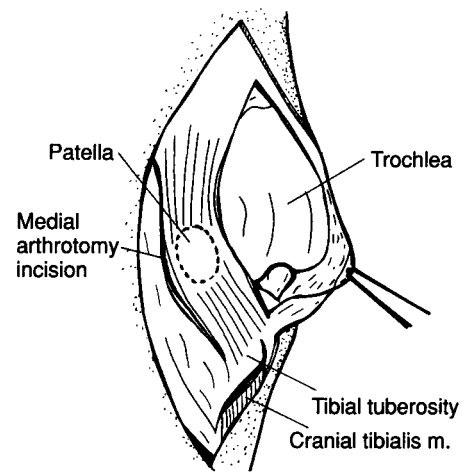
If medial deviation of the tibial tuberosity is present, then transposition (Fig. 42.10) to a more



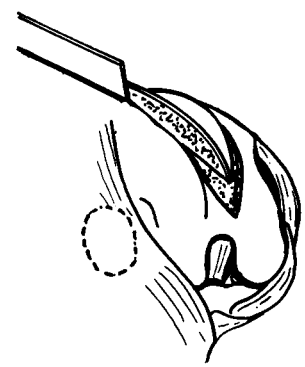
(a) Medial luxation of the patella is associated with medial rotation of the tibial tuberosity, caused by the medially directed quadriceps mechanism. Predisposing factors may include coxa vara and lateral bowing of the femur.



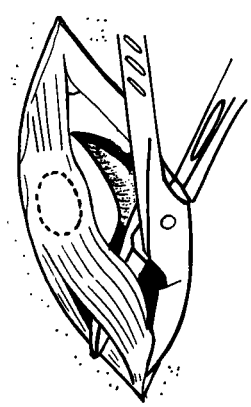
(b) The aim of lateral transposition of the tibial tuberosity is to restore the normal pull of the quadriceps extensor mechanism to the midline.



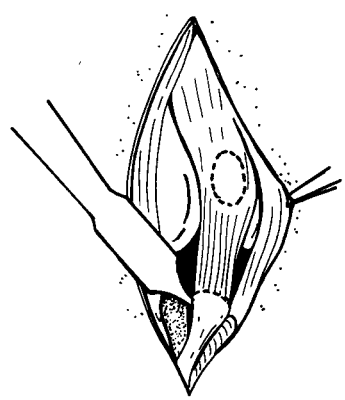
(c) Trochlea exposed by lateral parapatellar arthrotomy.



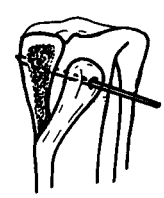
(d) Trochlea deepened by wedge recession technique.



(e) Tibial tuberosity freed proximally but periosteal attachment retained distally.



(f) Tuberosity levered laterally into line with trochlea, and patella reduced.



(g) Tuberosity fixed with Kirschner wire.

Fig. 42.10 Transposition of the tibial tuberosity in the treatment of medial luxation of the patella (DeAngelis & Hohn, 1970).

lateral position (DeAngelis & Hohn, 1970) allows correction of the quadriceps complex alignment such that the patella falls in line with the distal femur rather than medial to it. Although this can be assessed clinically, it is far more easily seen at surgery when any medial deviation in the patellar ligament can be observed once the patella has been returned to its normal position, with or without the need for medial retinacular release and/or recession V-sulcoplasty. It is also possible to observe the deviation through a full range of extension and flexion of the joint as well as during medial rotation of the tibia. In most cases, sufficient lateral transposition may be gained without separation of the periosteal attachments distally and the tibial crest may be fixed in its new position using a Kirschner wire or wire suture. If lateral transposition of more than about 1 cm is required, then the tibial tuberosity may need to be completely separated from its origin. In these cases a wire suture or pin and tension band technique would be an appropriate means of securing the fragment in its new position.

Corrective osteotomy

In cases where severe bowing of the distal femur and/or proximal tibia is present, the aforementioned techniques may be unable to restore normal alignment of the quadriceps complex. Instead it may be necessary to consider corrective osteotomies of the femur and/or tibia. Such techniques are rarely indicated and should only be considered in the most severe of cases.

Patellectomy

This may need to be considered if marked erosions are present on the articular surface of the patella since these may cause persistent lameness after its successful relocation. However, the observation of erosions does not necessarily mean that corrective surgery alone will not allow healing and a satisfactory outcome. In general, it may be better just to note the presence of the erosion and then continue with surgery as normal. If postoperative progress is unsatisfactory then it may be worth considering the option of patellectomy. Patellectomy cannot be successfully used as an alternative to correction of the alignment of the quadriceps complex, although it may improve joint function in cases with chronic, irreducible patellar subluxation by removing the painful apposition of exposed subchondral bone.

Postoperative care

A support bandage may be applied for a week postoperatively. Exercise is restricted for 4–6 weeks and then gradually increased. In dogs with bilateral patellar luxation, an interval of 6–8 weeks is left between operations on each stifle.

Complications

- Reluxation of the patella may result from:
  - Failure to bring the tibial tuberosity into normal alignment with the trochlea
  - Failure to adequately immobilise the tibial tuberosity in its new position
  - Failure to provide a trochlear groove of sufficient depth.
- Inability to fully extend the stifle joint. This complication is generally seen in dogs with grade 4 medial luxation when surgical correction has been attempted towards the end of growth or after 1 year of age. Ideally, surgical correction should be undertaken at 4–5 months of age, before contracture of the caudal muscles of the stifle has resulted in permanent joint deformity with inability to extend the stifle.

Prognosis

The prognosis in the majority of cases is very good with about 90–95% of cases regaining normal or close to normal function within about 8–12 weeks of surgery. The cases with the worst prognosis are those with very severe deformities at a very young age and middle-aged dogs with long-standing patellar luxations. In the latter, there is contraction of the soft tissues caudal to the joint, subsequent to the long period for which the stifle has been held in a semiflexed position. Owing to this, there is often residual lameness due to an inability to extend the joint fully even after successful relocation of the patella.

Lateral luxation of the patella in adult Toy Poodles also carries a poor prognosis. Unilateral or bilateral luxation occurs spontaneously at 8–9 years of age and is probably the result of a collagen disorder. Most cases are treated by medial capsular overlap only as the alignment of the tibial tuberosity and the depth of the femoral trochlear groove is normal. Although patella stability tends to be good initially, re-luxation frequently occurs within a few weeks. The risk of recurrence can be reduced by immobilising the stifle in a cast for 4 weeks following surgery.

Congenital patellar luxation in cats

There are few references to patellar luxation in the cat (Flecknell & Gruffyd-Jones, 1979; Davies & Gill, 1987; Houlton & Meynink, 1989). The condition has been recorded in British Shorthaired cats, Devon Rex, Siamese and a variety of crossbreeds of cat. Only about 30% of cats with patellar luxation exhibit lameness and, consequently, the true incidence of the condition is probably higher than is generally thought. The principles of treating both congenital and acquired traumatic luxation of the patella are the same as in the dog. The prognosis for a return of normal limb function following corrective surgery is usually good.

Bipartite patella

Bipartite patella is an uncommon congenital malformation. The condition may be unilateral or bilateral and is usually asymptomatic. The area of division in the patella is thought to be associated with poor vascularity. Cases have been reported in the Greyhound (Robins, 1990) and the authors have seen the condition in the cat and, sporadically, in various breeds of dog.

Fabella abnormalities

Abnormalities of one or both fabellae may be seen as incidental radiographic findings in the normal dog. A fabella may be bipartite, multipartite, displaced or absent (Park, 1979).

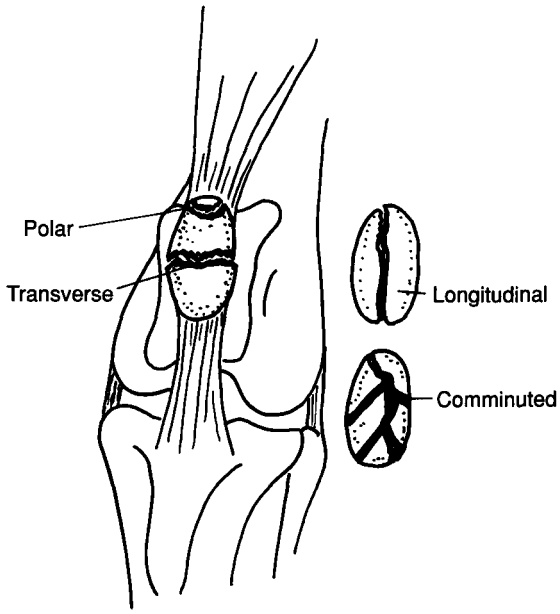


Fig. 42.11 Fractures of the patella.

Fractures of the patella

Fracture of the patella is an uncommon injury (Denny, 1975; Betts & Walker, 1975; Abercromby, 1998). The injury is caused by direct trauma. Fractures can be classified as transverse, longitudinal, polar or comminuted (Fig. 42.11). Transverse fractures are the most common. Most animals are unable to bear weight on the leg or extend the stifle following fracture. The exception are those cases with undisplaced longitudinal fractures which often show only mild to moderate degrees of lameness.

Radiographic examination

Caudocranial and flexed, mediolateral radiographs of the stifle are used to demonstrate the fracture. A tangential, or skyline, radiograph of the patella (Fig. 42.12) can be useful to demonstrate undisplaced longitudinal fractures or the

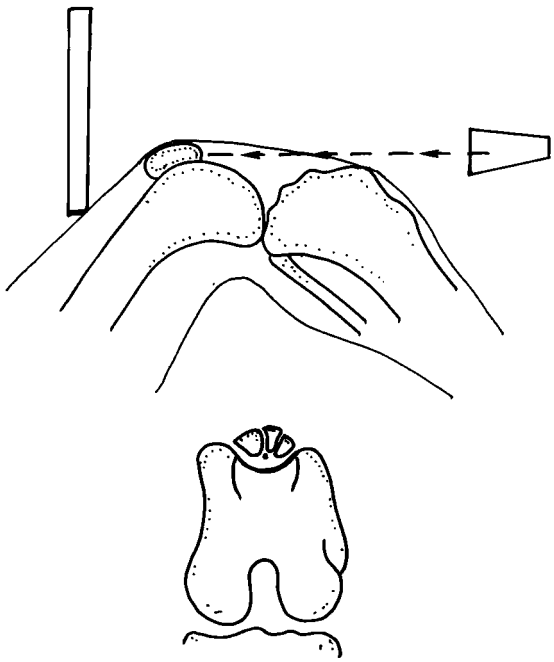


Fig. 42.12 Radiographic diagnosis. Tangential or skyline projection of the patella.

number of fragments present in comminuted fractures.

Treatment

Transverse fractures are repaired using a Kirschner wire and tension band wire technique (Fig. 42.13) (Weber *et al.*, 1980). The repair can be protected from excessive distractive forces by placing a loop of heavy orthopaedic wire through the insertion of the patellar tendon and, distally, through the tibial tuberosity (see later in Fig. 42.18). This second wire does have a tendency to break eventually and is usually removed 6 weeks after surgery.

Longitudinal fractures (Fig. 42.14a), if undisplaced, tend to be stable causing minimal lameness and will heal satisfactorily with conservative management. If the fracture is displaced then lag screw fixation (Fig. 42.14b) is used (Betts & Walker, 1975) or a pin and tension band (Fig. 42.14c) (Abercromby, 1998).

Polar fractures often produce fragments that are too small for internal fixation. These small fragments have little effect on the extensor apparatus or femoropatellar articulation. Conservative management usually gives satisfactory functional results although non-union of the fragment often persists. Alternatively, the fragment can be removed.

Comminuted fractures (Fig. 42.15a) require management that is tailored to their specific configurations. The options include:

- Reconstruction using lag screws, Kirschner wires, wire sutures or a combination of these
- Partial reconstruction with partial patellectomy (Fig. 42.15b)
- Partial patellectomy
- Patellectomy
- Replacement of the patella using a frozen allograft of patella, patellar tendon and ligament (Fig. 42.16) (Vaughan & Formston, 1973)

Patellar fracture in the cat

Principles of treating patellar fractures are the same as in the dog. It should be noted, however, that cats will be occasionally encountered which have obviously sustained a transverse or polar patellar fracture at some stage, the fracture has gone untreated and a non-union has developed but, despite this the cat has good or normal hindlimb function (Arnbjerg & Bindseil, 1994).

Straight patellar ligament (tendon) injuries

Rupture of the straight patellar ligament is a rare injury (Brunnberg *et al.*, 1992). The extensor mechanism of the stifle is disrupted and a severe mechanical lameness results. Palpation of the stifle soon after injury may reveal the defect in the patellar ligament but later this is obscured by soft tissue swelling. A flexed, mediolateral radiograph of the stifle will show the patella lying much further proximally than normal in relation to the femoral trochlea. Mid-section ruptures are reported most often but ruptures can also occur proximally, close to the patella, or distally, at the attachment with the tibial tuberosity (Fig. 42.17a–c). A Bunnell suture pattern using polydioxanone (PDS, Ethicon) or monofilament nylon

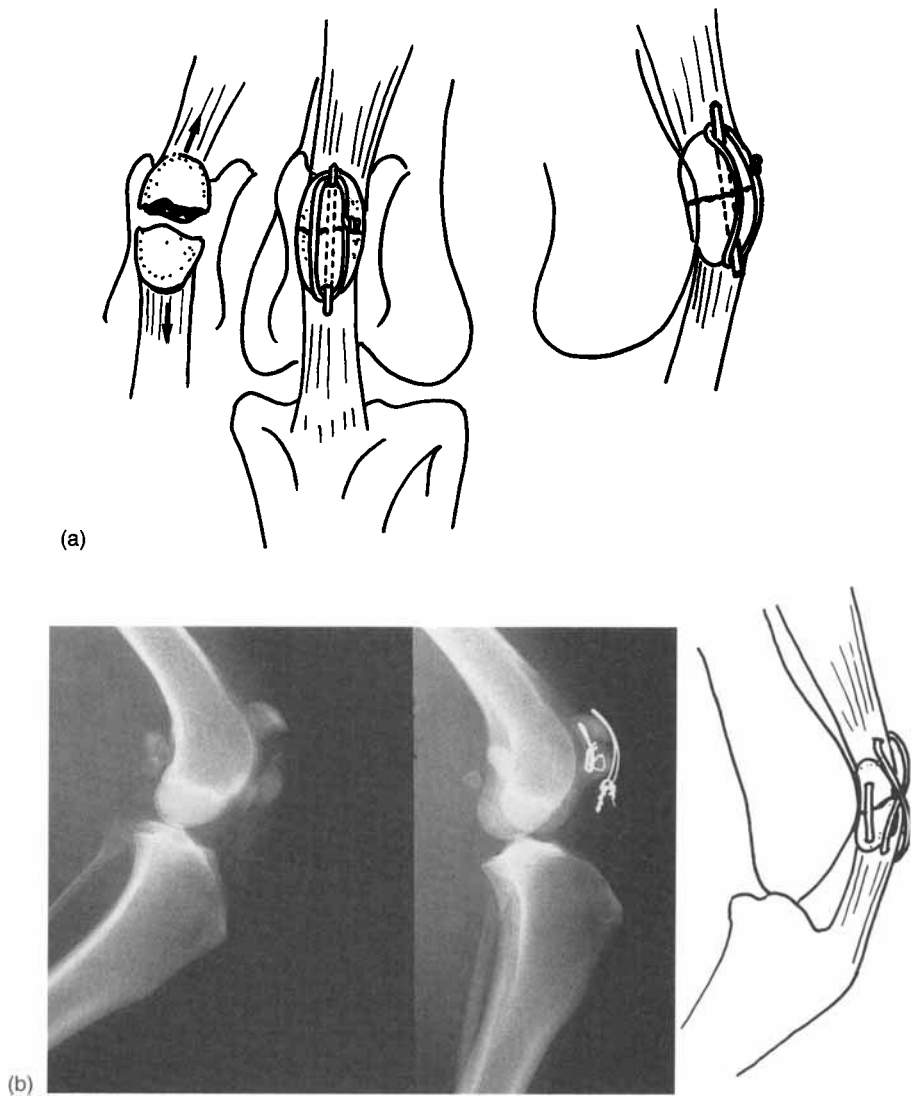


Fig. 42.13 Transverse fractures of the patella. (a) Repair using Kirschner wire and tension band wire. (b) Transverse fracture in a Whippet. Pre- and postoperative radiographs and postoperative tracing. Fracture repaired with a wire suture and tension band wire. (Source: *British Veterinary Journal*, vol. 141.)

is used to appose the tendon ends in mid-section rutures. If rupture has occurred close to the patella or the tibial tuberosity, then a Bunnell suture is placed in the ligament and the ends of the suture material are taken through a tunnel drilled transverely through the distal patella or tibial tuberosity, respectively, before being tied. If there is a defect in the patellar ligament (Fig. 42.17d) then this can be reinforced with a fascia lata strip (Culvenor, 1988; Brunnberg *et al.*, 1992). It is important to protect the patellar ligament repair with a wire loop (1 mm or 0.8 mm wire depending on the size of the animal) placed between the patella and the tibial tuberosity. The wire is usually taken through tunnels drilled transversely though the patella and tibial tuberosity (Fig. 42.18). An alternative to the patellar tunnel is to anchor the wire loop through the tough

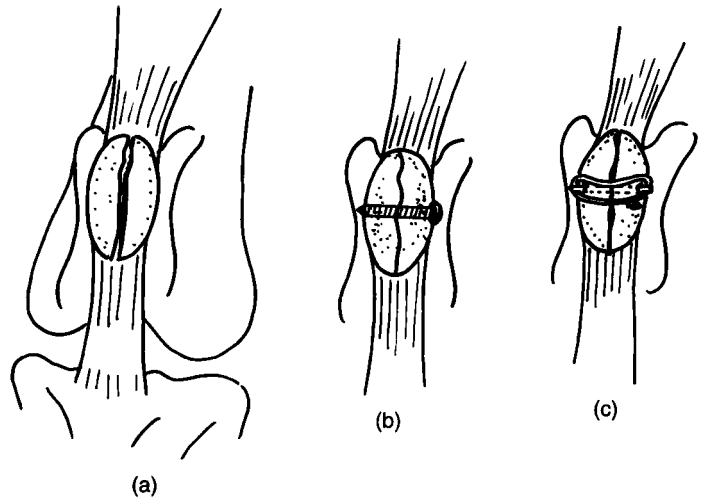


Fig. 42.14 (a) Longitudinal patellar fracture. (b) Fixation with lag screw. (c) Fixation with Kirschner wire and tension band wire.

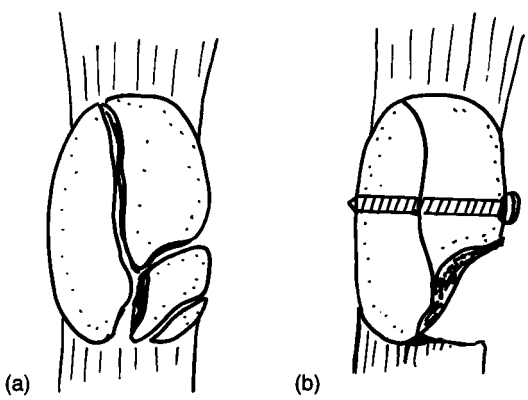


Fig. 42.15 (a) Comminuted patellar fracture. (b) Partial reconstruction and partial patellectomy.

tendinous tissue just proximal to the patella. The ends of a screw passed transversely though the tibial tuberosity can be used for anchorage of the wire distally, instead of a tunnel (Brunnberg *et al.*, 1992). The wire should not be crossed, like a tension band, otherwise the central portion will interfere with the ligament repair.

The stifle may be supported with a splint for 4 weeks following surgery. Wire loops have a tendency to break and may need removal if they cause soft tissue problems. Prognosis following repair of the straight patellar ligament is generally good provided adequate support is provided during the healing process.

Fracture of the fabellae

Fractures of the lateral fabella have been recorded in the dog (Houlton & Ness, 1993). Fabella fractures are rare and should be distinguished from the more common congenital bipartite, or multipartite, fabella. Fractures appear to be spontaneous and unassociated with any other form of trauma. Pain can be localised to the lateral fabella on deep palpation. Mediolateral and craniocaudal radiographs of the stifle will demonstrate the fracture. The prognosis is excellent with either conservative treatment (restricted exercise, 10–15 minute walks on a leash twice daily) or with surgical excision of the fragments. Recovery time with conservative treatment is 10–12 weeks, with surgery giving a more rapid return to normal function.

Avulsion of the lateral head of the gastrocnemius muscle

Avulsion of either the lateral or medial head of the gastrocnemius muscle is an occasional cause of stifle lameness (Chaffee & Knecht, 1975; Vaughan, 1979; Reinke *et al.*, 1982; Muir & Dueland, 1994; Prior, 1994). The lateral and medial heads of the gastrocnemius muscle arise on the corresponding caudal tuberosity of the femur and a sesamoid bone, the fabella, lies in

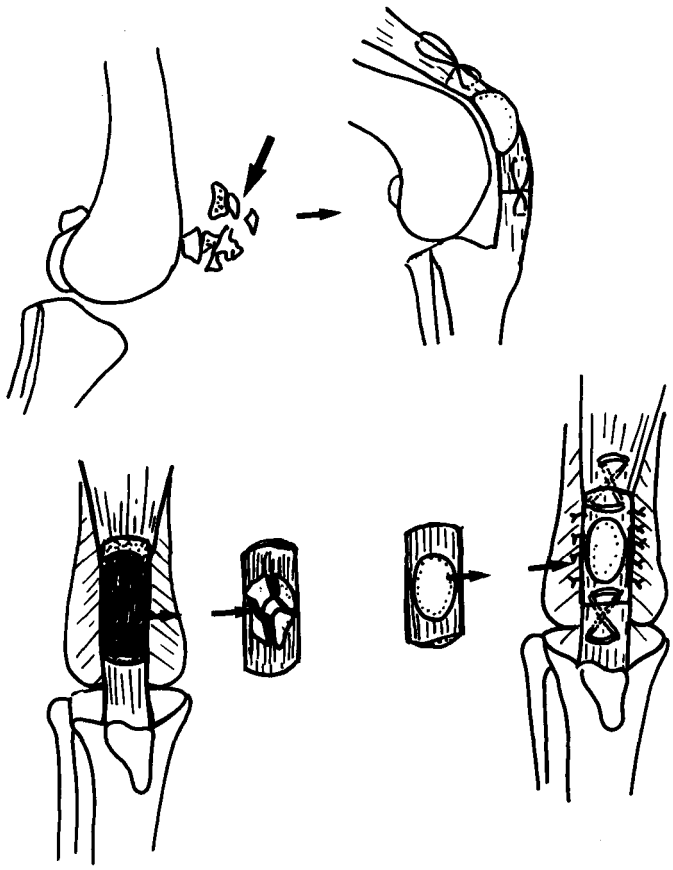


Fig. 42.16 Comminuted fracture of the patella replaced with a frozen allograft of patella, patellar tendon and ligament (Vaughan & Formston, 1973).

each tendon of origin. The superficial digital flexor muscle is firmly attached to the deep surface of the lateral head of the gastrocnemius muscle and also shares attachments to the lateral fabella.

Avulsion of either head of the gastrocnemius muscle is associated with swelling and pain over the caudal aspect of the stifle. Postural defects may also occur with hyperflexion of the hock and a plantigrade stance when the lateral head is affected, but were not observed with medial head involvement (Muir & Dueland, 1994). Diagnosis is confirmed radiographically by demonstrating ventral displacement and possibly fracture of the associated fabella.

Definite guidelines for treatment have not been fully established. There are reports of recovery with both conservative and surgical management. Cases with avulsion of the medial head of the gastrocnemius have been successfully managed with anti-inflammatory drugs, physiotherapy and swimming activity. If the lateral head is involved, and particularly if hyperflexion of the hock is occurring, then the head of the gastrocnemius muscle should be reattached to the femur using a wire suture if the dog is to regain normal limb posture. The stifle is immobilised with a Robert Jones bandage for 2 weeks following surgery. The average recovery period is 3 months.

Avulsion of the popliteus muscle

Single case reports of this injury have been described (Pond & Losowsky, 1976; Eaton-Wells & Plummer, 1978; Tanno *et al.*, 1996). The tendon of origin of the popliteus muscle arises from the

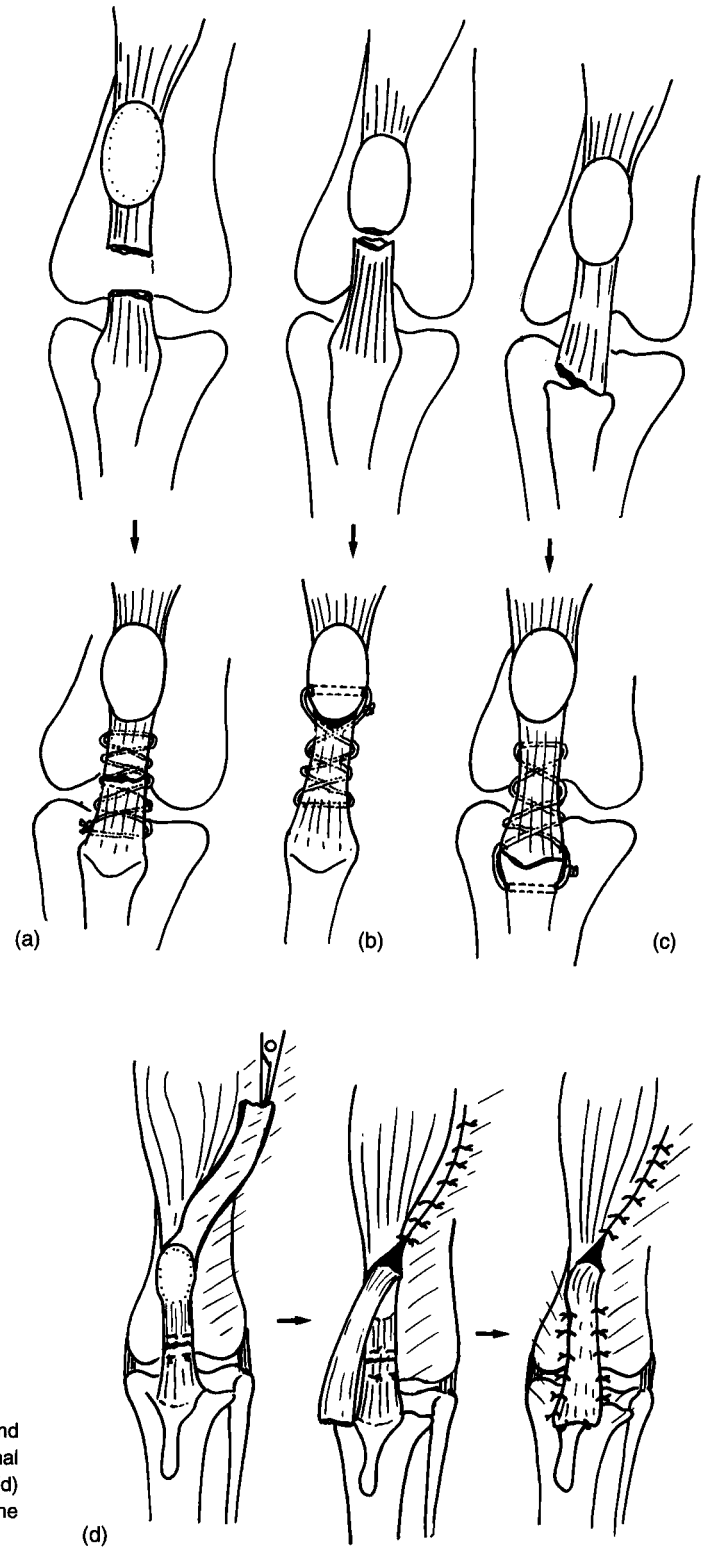


Fig. 42.17 Straight patellar ligament injuries and their repair. (a) Mid-tendon rupture. (b) Proximal rupture or avulsion. (c) Distal rupture or avulsion. (d) Use of a fascia lata strip to reinforce repair of the straight patellar ligament (Culvenor, 1988).

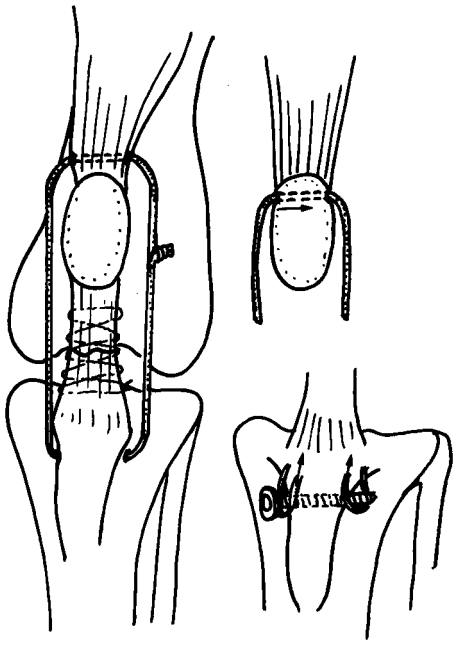


Fig. 42.18 Protecting straight patellar ligament repairs with a wire loop. Proximally, wire can be anchored through the patellar tendon or patella. Distally, the wire is taken through a tunnel in the tibial tuberosity or around the ends of a screw placed transversely through the tibial tuberosity.

lateral femoral condyle and runs caudally, passing between the lateral collateral ligament and the abaxial border of the lateral meniscus. The musculotendinous junction lies caudodistal to the tibial plateau. The popliteal sesamoid lies in the caudal part of the tendon of origin. The muscle inserts on the caudal aspect of the proximal tibia. The popliteus muscle is a stifle flexor and causes inward rotation of the tibia.

Avulsion of the popliteus muscle results in a sudden onset of lameness at exercise with stifle swelling and pain but no instability. A mediolateral radiograph shows a defect at the site of origin of the popliteus with bone fragments and also caudoventral displacement of the popliteal sesamoid bone.

Surgical treatment is indicated. The avulsed tendon is exposed though a lateral parapatellar arthrotomy incision. If the bone fragment is large enough, it can be reattached to the femoral condyle with a lag screw. Alternatively, the tendon can be sutured to the long digital extensor tendon.

Avulsion of the long digital extensor tendon (Denny & Minter, 1973; Pond, 1973; Lammerding *et al.*, 1976; Vaughan, 1979; Eaton-Wells & Plummer, 1978; Butterworth, 1994)

The tendon of origin of the long digital extensor muscle is an intra-articular structure which arises from the lateral condyle of the femur, crosses the joint and emerges on the lateral side of the tibia in the muscular groove. An extension of the joint capsule acts as a synovial sheath for the tendon in the muscular groove. Avulsion of the tendon occasionally occurs in young dogs of the larger breeds; the average age of recorded cases is 6 months. There is a sudden onset of lameness, the leg is carried and there is swelling and pain but no instability on palpation of the stifle. After the acute phase has subsided, mild lameness persists with slight discomfort on palpation. A mediolateral radiograph of the stifle should reveal the avulsed fragment of bone and an erosion in the lateral femoral condyle (Fig. 42.19a).

Surgical treatment is indicated. A lateral parapatellar arthrotomy provides good exposure of the defect in the lateral femoral condyle and the avulsed fragment, which is often covered in fibrous tissue. Wherever possible, reattachment of the tendon is carried out using a lag screw (Fig. 42.19b) if the fragment is large enough. Alternatively, when the fragment is too friable to allow this, then the tendon is sutured to the lateral collateral ligament or the connective tissue in the region of the sulcus muscularis.

The prognosis is good and most cases should regain normal limb function within 2–3 months of surgery.

Displacement of the long digital extensor tendon

Single case reports of caudal displacement of the tendon from the tibial sulcus muscularis have been described (Addis, 1971; Bennett & Campbell, 1979). One of the authors has seen the condition in a 2-year-old racing Whippet. The dog was only lame at the canter and the tendon could be felt slipping in and out of the sulcus muscularis as the stifle was flexed and extended. There was no

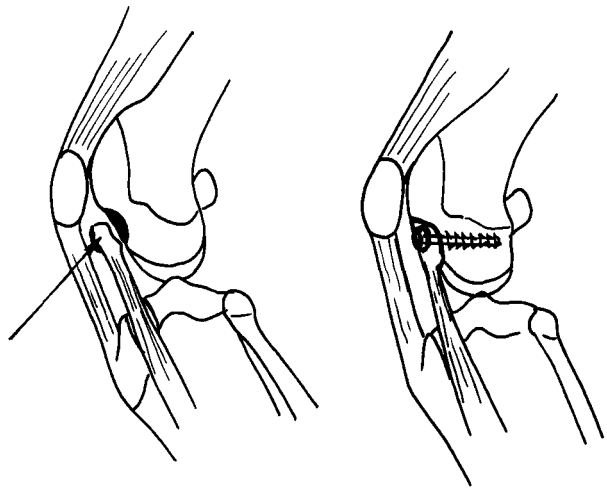


Fig. 42.19 (a) Avulsion of the tendon of origin of the long digital extensor muscle. (b) Lag screw fixation.

obvious pain. The tendon was retained in the sulcus by repairing the torn retinaculum overlying the tendon with sutures of polydioxanone (PDS, Ethicon). Recovery was uneventful, the dog was sound within 3 months and continued to race successfully.

Cranial cruciate ligament (CrCL) deficiency

Cranial cruciate ligament (CrCL) deficiency is the most common condition to affect the canine stifle (Bennett, 1990; Anderson, 1994) and inevitably results in the development of osteoarthritis (OA). The CrCL arises on the caudomedial aspect of the lateral femoral condyle and inserts on the cranial intercondyloid area of the tibia. It has three basic biomechanical functions in preventing:

- The cranial drawer movement which is cranial displacement (translation) of the tibia relative to the femur
- Overextension of the stifle
- Excessive internal rotation of the tibia with respect to the femur

The CrCL is made up of two distinct functional bands: a craniomedial band and a caudolateral band. The craniomedial band is taut when the joint is in flexion and extension while the caudolateral band is taut in extension only (see partial ruptures of the CrCL below, p. 543).

Aetiology

Dogs with CrCL deficiency, or disease, can be divided into four clinical groups depending on the cause:

(1) *CrCL rupture as a result of trauma.* This is probably the least common form of CrCL deficiency. There is a sudden onset of lameness at exercise or through the dog catching its leg in a fence, or other obstacle, resulting in overextension of the stifle or excessive internal rotation.

(2) *Degeneration of the CrCL in older dogs.* Degeneration of the CrCL leading to rupture occurs in dogs between 5 and 7 years of age and is probably the most common form of CrCL disease seen. Lameness is often insidious in onset and suddenly becomes worse as a partial rupture proceeds to complete rupture of the ligament. This often occurs after only minor trauma which may occur during normal exercise. At the time of radiography or arthrotomy, osteoarthritic change is often already well established. Overweight Labrador and Golden Retrievers are particularly prone to this form of CrCL disease.

(3) *Rupture of the CrCL in young large breed dogs* (Bennett *et al.*, 1988). In large breeds of dog, especially the Rottweiler, Bull Mastiff, English Mastiff, Labrador Retriever, Goldern Retriever, St Bernard, Newfoundland and Boxer, cruciate ligament disease characterised by partial tearing of the CrCL and chronic osteoarthritic change in the joint occurs in young dogs aged between 6 months and 3 years. This 'early' degeneration of the ligament *may* be related to stifle or hindlimb conformation.

(4) *Rupture of the CrCL associated with inflammatory arthropathies.* Immune-mediated or infective inflammatory arthropathies can cause pathological changes in the CrCL resulting in rupture.

Other factors which may predispose to degeneration of the CrCL include:

- *Excessive caudal tilting of the tibial plateau* by increasing cranial tibial thrust and causing increased stress in the CrCL (Slocum & Devine, 1984). See 'Growth disturbances' of the stifle earlier in the chapter (p. 516) and (3) above.
- *Stenosis of the intercondylar notch of the femur* may cause impingement of the medial aspect of the lateral femoral condyle on the CrCL. This is thought to contribute to anterior cruciate ligament rupture in humans. A recent study by Aiken *et al.* (1994) has shown that the stifles of dogs with CrCL injuries had significantly smaller notch indices compared to normal stifles. Based on this study, they recommended intercondylar notchplasty if an intra-articular replacement of the CrCL is planned.

Clinical signs of CrCL rupture

- Sudden onset of lameness (note lameness can be insidious in onset – see above).
- Leg carried with stifle slightly flexed.
- After 7–10 days the dog uses the leg when walking, but at rest stands, with toe just touching the ground.
- 'Clicking' noises may be heard when the dog is walking due to the femoral condyles slipping in and out of their normal position on the menisci. Although this is often considered as indicative of meniscal injury, there is no evidence to substantiate this and in the authors' opinion the presence of clicking is probably indicative only of functional instability.
- The quadriceps muscles atrophy, assessed by comparison with the contralateral leg (unless the dog has bilateral CrCL deficiency).
- Stifle effusion is detected by palpating the straight patellar ligament which is normally taut and well defined. If there is effusion present, definition of the patellar ligament is lost and thickening or fluctuating swellings can be felt on either side of it.
- A *cranial drawer movement* may be detected. Normally, the CrCL prevents cranial displacement of the tibia on the femur. Abnormal movement in this direction is diagnostic for CrCL rupture and is known as the cranial drawer sign (Fig. 42.20). The *tibial compression test* is useful for detecting complete ruptures of the CrCL in larger dogs. The dog is positioned in lateral recumbency with the affected leg uppermost. The leg is extended, the metatarsus grasped in one hand and the stifle is fixed

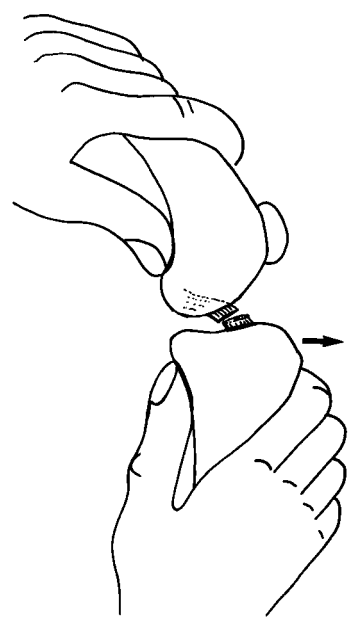


Fig. 42.20 Rupture of the cranial cruciate ligament (CrCL), the cranial drawer movement.

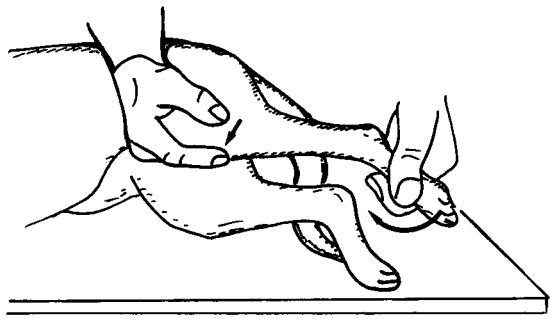


Fig. 42.21 Tibial compression test for cranial cruciate ligament (CrCL) deficiency.

with the other. If there is complete rupture of the CrCL then attempts to flex the hock with the stifle extended will result in cranial movement of the tibia and tibial tuberosity in relation to the femur. This movement can be detected by placing the index finger of the hand supporting the stifle over the tibial tuberosity (Fig. 42.21).

- Joint instability is associated with the development of OA. In the larger breeds of dog, weighing 15 kg or more, this is characterised by the development of periarticular osteophytes. In the smaller breeds of dog, minimal osteophytic reaction occurs.
- The stifle gradually restabilises by periarticular fibrosis which is particularly marked over the medial side of the joint.
- Restabilisation becomes optimal within 6–8 weeks and lameness may resolve in this period, especially in small breeds of dog. In larger breeds, varying degrees of lameness tend to persist because of the osteoarthritic changes which have occurred during restabilisation, together with any meniscal injury that might have developed during the period of instability.

Radiographic examination

It should be stressed that diagnosis of CrCL rupture is based on clinical examination and the radiographic changes are non-specific. Nevertheless, it is useful to take mediolateral and craniocaudal radiographs of both stifles to detect the presence, or absence, of osteoarthritic change, the degree of change present and to ensure there are no other obvious causes of the lameness. The mediolateral projection gives the most information about the joint. If there is synovial effusion present then there will be disruption of the outline of the infrapatellar fat pad and distension of the joint capsule which is most readily observed caudally. Periarticular osteophyte/enthesiophyte (new bone around a sesamoid) formation tends to be seen first around the proximal margins of the trochlea and poles of the patella and, later, around the fabellae and the edges of the tibial plateau (Fig. 7.5). Sclerosis of subchondral bone and areas of soft tissue mineralisation are seen in more advanced cases of OA. Intra-articular bone fragments may be seen in avulsion injuries of the CrCL.

Treatment

Small breeds, under 15 kg bodyweight, with CrCL rupture can be managed conservatively by restricting exercise to short (10 minute) walks on a leash only for 6–8 weeks and some 85% of these dogs will regain satisfactory hindlimb function (Vasseur, 1984). If lameness persists then surgical stabilisation of the joint should be recommended. In larger breeds of dog, early surgical stabilisation of the stifle is recommended to try and minimise the development of OA. The second purpose of surgery is to examine the menisci and treat any associated injury to either of these structures (see later under 'Meniscal injuries', p. 544). In dogs with bilateral CrCL ruptures the most unstable stifle, or the one which is causing the worst lameness, should be operated on first. Surgery is carried out on the second stifle 6–12 weeks later if that joint is not already stabilising satisfactorily by periarticular fibrosis. The fact that numerous techniques have been described since Paatsama's original work on CrCL rupture in 1952 indicates that no single technique is entirely satisfactory. Surgical techniques can be broadly divided into:

- *Intracapsular techniques* which aim to restore stability by replacing the ligament with some type of graft.

- *Extracapsular techniques* which restore stability by the use of sutures or by using soft tissues as a sling.
- *Periarticular techniques* like the tibial plateau levelling procedure and fibular head transposition which restore stability by altering local anatomy.

The two most commonly used techniques in the UK are the modified 'over-the-top' procedure, which is an intracapsular procedure, and the lateral retinacular (fabellotibial) suture, which is an extracapsular procedure. Both give similar results and, for most surgeons, choice of technique is a matter of personal preference. No matter what technique is used, it is periarticular fibrosis which ultimately tends to stabilise the joint.

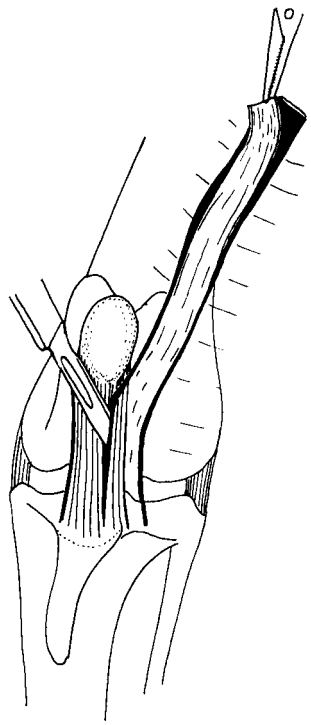
Intracapsular procedures

The original 'over-the-top' (OTT) procedure was described by Arnoczky *et al.* (1979) and his work represented a major advance in our understanding of the management of CrCL rupture. A graft consisting of the medial third of the straight patellar ligament, a wedge of patella, patellar tendon and fascia lata is passed through the stifle along the path of the original CrCL. The free end of the graft is pulled 'over the top' of the lateral femoral condyle where it is sutured to the periosteum. The graft directly overlies the origin of the CrCL and at no time during flexion and extension of the stifle is it subjected to excessive tension. In most other intra-articular procedures, particularly those in which a prosthesis or graft is passed through bone tunnels, it is difficult to replicate the origin of the CrCL accurately. This leads to excessive tension on the replacement ligament as the stifle is flexed and results in excessive wear and early breakdown. The 'OTT' technique offers a distinct advantage in this respect. It has been demonstrated, in experimental dogs, that the patellar ligament graft is revascularised within 20 weeks and, at 1 year, the vascular and histological appearance of the graft resembles that of a normal CrCL (Arnoczky *et al.*, 1982). Biomechanically, the 'OTT' technique has many advantages over other methods of CrCL ligament replacement and gives good results, particularly in large and giant breeds of dog. However, the operation in its original form presents some technical difficulties, particularly in preparation of the patellar segment of the graft and passage of the graft through the joint.

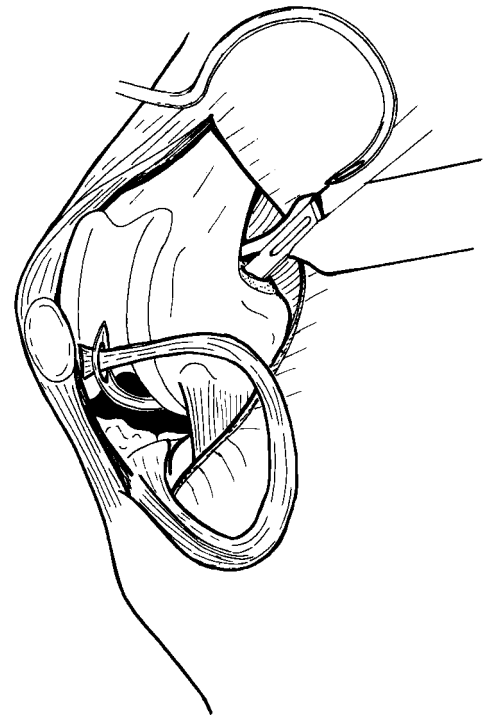
A much simpler 'OTT' technique was described by Hulse *et al.* (1980) in which the lateral third of the patellar ligament and fascia lata was used as a graft (Fig. 42.22). A biomechanical analysis of the method was subsequently reported (Butler *et al.*, 1983; Hulse *et al.*, 1983). The results were encouraging with a gradual reduction in joint instability and an increase in the stiffness and strength of the graft. However, even at 26 weeks following surgery, the material properties of the graft were considerably weaker than those of a normal CrCL. Nevertheless, no mechanical failures of the graft were seen in *in vivo* laboratory tests and in a series of 38 operations performed in clinical cases, 93% of animals were considered to have regained normal limb function (Shires *et al.*, 1984). Similar results in clinical cases have been reported by Denny & Barr (1984, 1987). Various modifications of this technique exist, each attempting to mimic the anatomy of the CrCL more closely (Bennett & May, 1991a), but none has been shown to offer any practical advantages in terms of success rate over this simple 'OTT' technique. Overall, satisfactory outcomes are reported in 80–90% of cases, though only about two-thirds of these will be truly sound and the remainder will have a mild or intermittent lameness that does not affect their quality of life.

'Over-the-top' procedure using the lateral third of the straight patellar ligament and fascia lata as a graft

The fascia lata, patella and straight patellar ligament are exposed through a lateral parapatellar skin incision. A graft consisting of the lateral third of the straight patellar ligament and fascia lata is prepared (Fig. 42.22a). Starting from the tibial tuberosity, the straight patellar ligament is split with a scalpel longitudinally through the lateral third. The incision is curved laterally and caudally, just before reaching the patella, and continued around the lateral margin of the patella (leaving sufficient soft tissue for closure) before being directed back towards the midline and extended proximally into the fascia lata. A parallel incision



(a) A graft is prepared consisting of the lateral third of the straight patellar ligament and fascia lata. The length of the graft should be 3 to 4 times the length of the patellar ligament.

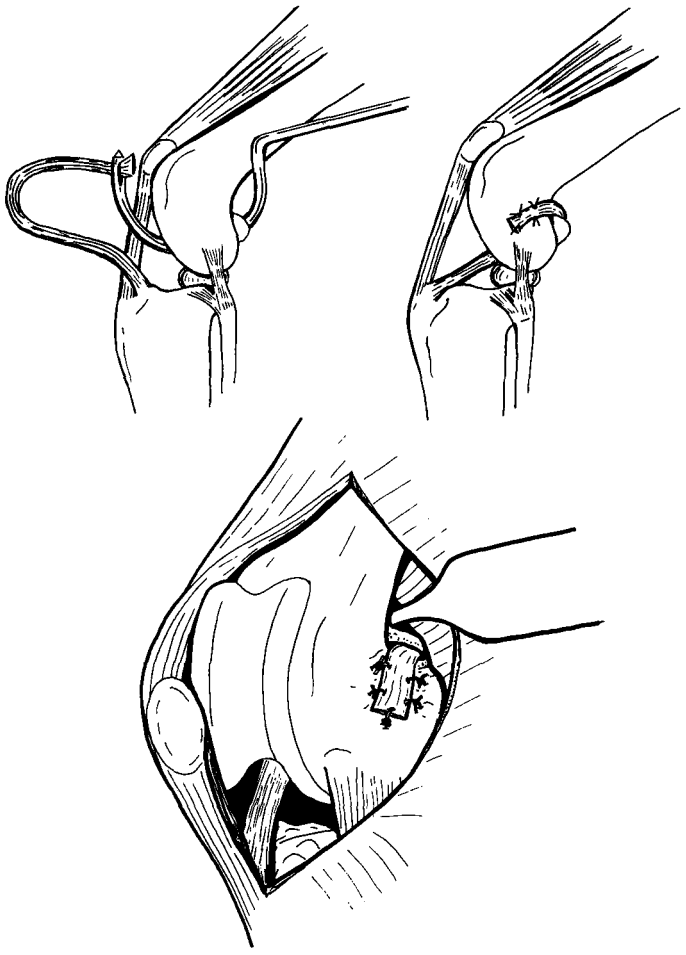


(b) A lateral parapatellar arthrotomy is made and the patella displaced medially. The medial meniscus is inspected (see 'Meniscal injuries'), and torn remnants of cruciate ligament and osteophytes are removed. The lateral joint capsule is retracted with a Hohmann retractor, the femorofabellar ligament is incised and a graft passer in inserted through the incision and directed through the joint. The graft is passed through the 'eye' of the graft passer.

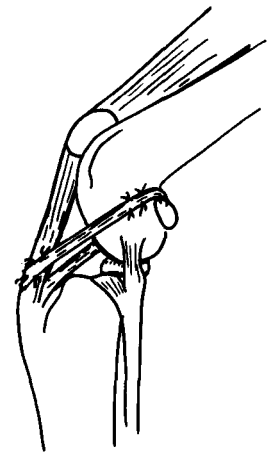
Fig. 42.22 The 'over-the-top' procedure for replacement of the cranial cruciate ligament (CrCL) using the lateral third of the straight patellar ligament and fascia lata as a graft (Hulse *et al.*, 1980).

is made in the fascia lata, caudal to the first, and a strip of patellar ligament and fascia lata 1–1.5 cm in width is prepared. The proximal fascial attachments are cut and the strip is reflected distally. The attachments to the tibial tuberosity are retained. A lateral parapatellar arthrotomy incision is made and the patella is reflected medially out of the femoral trochlear groove. The patella can be completely 'flipped over' to expose its articular surface which gives a wide exposure of the joint, places the soft tissues under tension, controlling haemorrhage, and makes subsequent inspection of the medial meniscus easier. The stifle is inspected and torn remnants of the CrCL are removed. The medial meniscus is carefully checked for injury (see 'Meniscal injuries' later in the chapter) and, if necessary, partial or total medial meniscectomy is performed. Periarticular osteophytes between moving parts of the stifle, i.e. in the proximal femoral trochlea or on the poles of the patella, are removed.

The lateral joint capsule is reflected with a Hohmann retractor to reveal the lateral femoral condyle and the fabella (Fig. 42.22b). In positioning the Hohmann, the tip of the retractor is hooked under the caudal aspect of the femur just proximal to the lateral condyle. The retractor protects the soft tissues caudal to the stifle and,



(c) The graft is pulled through the joint and sutured to the femorofabellar fascia, and the patella is reduced.



(d) For extra stability, and if the graft is of sufficient length, the free end of the graft can be sutured to the straight patellar ligament. The fascia lata, joint capsule, subcutis and skin are closed in separate layers.

Fig. 42.22 *Contd.*

provided all sharp dissection is done cranial to it, there should be no risk of iatrogenic damage to the popliteal artery. A small, vertical incision is made through the femorofabellar ligament into the caudal compartment of the joint. The stifle is flexed, a graft passer (Veterinary Instrumentation) is inserted through this incision into the intercondylar fossa and this is directed into the cranial aspect of the joint (Fig. 42.22b). The graft is threaded through the 'eye' of the graft passer and the instrument is used to draw the graft through the stifle.

The graft is pulled tight over the lateral femoral condyle and sutured to the femorofabellar fascia and periosteum using polydioxanone (PDS, Ethicon) (Fig. 42.22c). If the graft is long enough then the free end is sutured to the straight patellar ligament to provide additional stability (Fig. 42.22d) (Coetzee, 1993). The patella is replaced in the trochlea and the joint capsule and fascia lata are closed with two layers of polydioxanone sutures. The rest of the wound closure is routine. A support bandage is applied for 5 days postoperatively. Exercise is restricted to short

(10-minute) walks on a leash only for 12 weeks and is then gradually increased. Dogs start to weight-bear 10–14 days after surgery, they should be using the leg reasonably well by 6 weeks, with full recovery by 12–16 weeks.

Extracapsular procedures

The aim of extracapsular techniques is to stabilise the joint by tightening or reinforcing the lateral retinacular tissues. DeAngelis, or retinacular, sutures are used most widely. In their simplest form these involve the placement of sutures from around the fabella to the point of insertion of the patellar ligament (DeAngelis & Lau, 1970). The fact that both ends of the suture are essentially anchored in soft tissue structures gave rise to concern over premature loosening and so techniques were adopted whereby the sutures were anchored distally by passing either through two transverse tunnels in the tibial crest (Fig. 42.23a) or through one tunnel and back under the patellar ligament. Both of these sutures may be referred to as fabellotibial sutures. One modification of the technique involved placement of sutures both laterally and medially (Flo, 1975) but clinical experience showed that placement of only a lateral suture was equally effective. Permanent materials are generally used, which may be monofilament wire (Olmstead, 1993), braided nylon (Ethibond, Ethicon), or heavy gauge monofilament nylon such as Leader line (Caporn & Roe, 1996) (used more commonly for sea fishing!).

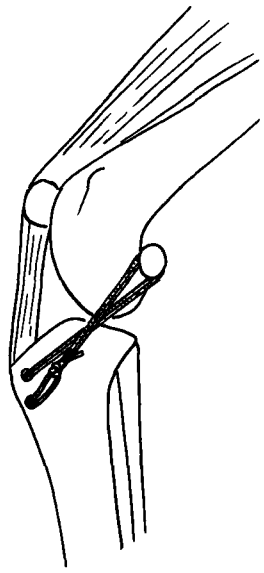
Although such techniques do not attempt to replace the 'lost' ligament, and the sutures involved will eventually stretch or break, they do stop any cranial drawer movement during the healing phase, thereby protecting the menisci from injury (though not absolutely – see under 'Complications' later in this chapter) and may enhance useful periarticular fibrosis along the line of the suture which, ultimately, is what will stabilise the joint. The success rates with such techniques is comparable to the 'OTT' procedure with satisfactory outcomes reported in 80–90% of cases, though only about two-thirds of these will be truly sound and the remainder will have a mild or intermittent lameness that does not affect their quality of life. The reasons for failure are very similar to those seen with 'OTT' techniques (see below), apart from the additional one of sepsis in the presence of an effective foreign body if surgery is not performed with good attention to aseptic technique.

Stabilisation of a cruciate deficient stifle using lateral fabellotibial sutures of monofilament nylon (Leader line)

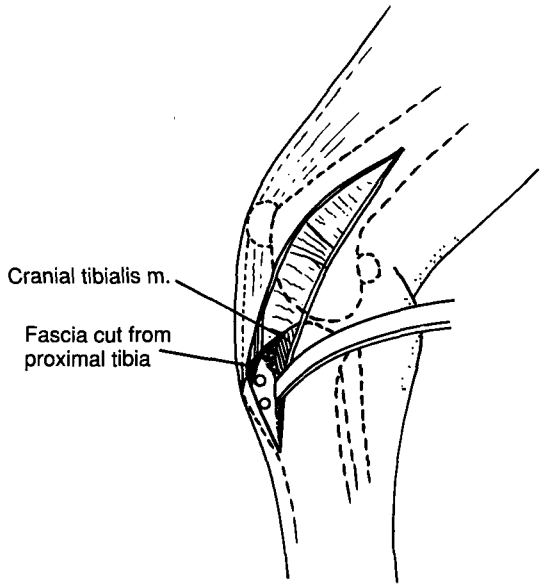
A lateral parapatellar arthrotomy is used which is similar to that described above for the 'OTT' technique except that a graft is not harvested and the fascial incision does not involve the patellar ligament. In a medium-sized breed of dog the skin incision begins about 2 cm below the point of insertion of the patellar ligament and ends about 2 cm above the patella. The fascial incision includes elevation of the cranial tibialis muscle from the lateral aspect of the tibial crest and release of the fascia from the proximal tibia. A small area of periosteum is elevated from the medial aspect of the tibial crest and two transverse tunnels are drilled through the crest such that the drill bit emerges medially in the area exposed (Fig. 42.23b).

The joint capsule is then incised to allow evaluation of the intra-articular structures. The arthrotomy is more limited than that described above for the 'OTT' technique and the patella cannot be 'flipped' over. Adequate exposure of the joint can easily be achieved by placement of a Gelpi retractor in a mediolateral direction and using a stifle joint distractor to separate the femoral condyles from the tibial plateau (see later under 'Meniscal injuries'). Any remnants of the CrCL are resected and the menisci are inspected. Damaged portions of the menisci are treated by partial meniscectomy. Any osteophytes that are interfering with joint function are removed but the vast majority are left untouched. The joint is flushed and the capsule closed with cruciate/crossed mattress sutures of polydioxanone (PDS, Ethicon).

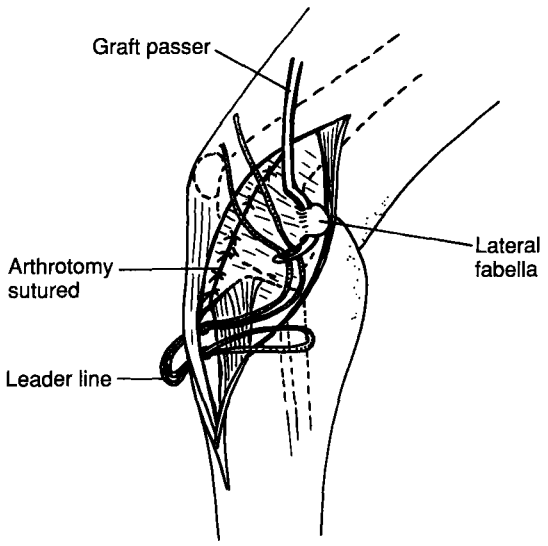
The lateral fascia is retracted sufficiently to allow palpation of the lateral fabella and a small graft passer (as used for the 'OTT' technique) (Veterinary Instrumentation) is introduced just proximal to the fabella and passed around it so as to reappear through the soft tissues lateral to the joint (Fig. 42.23c). Each fabellotibial suture is



(a) Stabilisation of a cranial cruciate ligament (CrCL) deficient stifle using lateral fabellotibial sutures of monofilament nylon (Leader line).

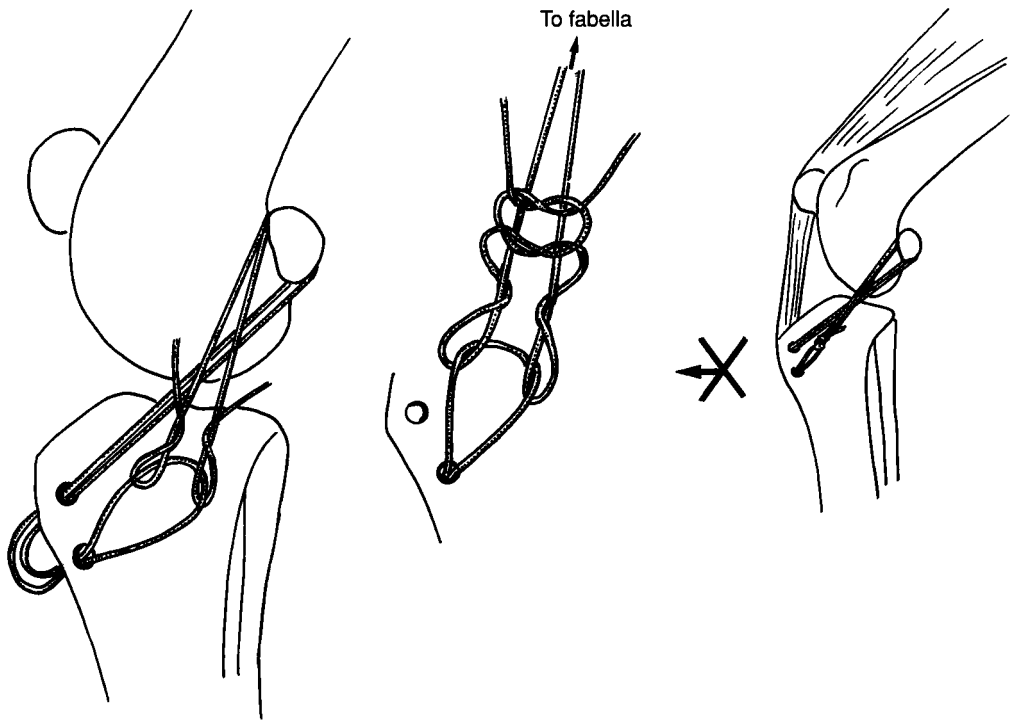


(b) To stabilise a cranial cruciate deficient stifle using a lateral fabellotibial suture requires a lateral parapatellar skin incision. The fascial incision extends distally to expose the tibial crest by elevation of the cranial tibialis muscle, and proximally beyond the patella so that the fascia can be sufficiently retracted caudally to allow palpation of the lateral fabella. Two tunnels are drilled through the tibial crest (see text for further details).



(c) After an arthrotomy has been performed to resect the remnants of the ligament and any damaged portions of meniscus, the joint capsule is closed. The fascia is retracted caudally to allow placement of a graft passer around the lateral fabella. The free ends of a double strand of Leader line are then placed through the distal tunnel, back through the proximal tunnel, and through the eye of the graft passer.

Fig. 42.23



(d) After the graft passer has pulled the Leader line around the fabella the free ends are placed through the loop to form a self-locking knot. The suture is then drawn tight.

(e) Once the suture is tight the two ends are tied together using five or six single throws.

(f) The overall effect is to create a double stranded lateral fabellotibial suture which stabilises the joint effectively.

Fig. 42.23 *Contd.*

made up of a single or double strand of Leader line with a breaking strain of either 70 or 80 lb. In the case of a double strand, a piece of line is taken and folded to create a loop and two free ends. The free ends are passed through the distal tibial tunnel, from lateral to medial, and then back through the proximal tunnel. The ends are pulled through until about 1 cm of the looped end remains. The free ends are then placed through the eye of the graft passer which is then withdrawn, so placing the two strands around the lateral fabella. If a single strand of line has been used then the two ends are tied with a square knot and five or six throws. Holding the first throw tight whilst the second is applied can be problematic which is one reason for using a double strand as this allows the use of a self-locking knot. If, however, a single strand has been used then tension can be released from the suture by having an assistant externally rotate the tibia. In the case of a double strand of line, the looped end is used to create a self-locking knot, based on that described by McKee & Miller (1999) (Fig. 42.23d). Once the knot has been drawn adequately tight, the two ends are brought together over the top of the knot and tied using five or six single throws (Fig. 42.23e). With respect to how many sutures and what size of material should be used in a particular case, views remain subjective rather than objective. One of the authors, who uses this technique almost exclusively in the management of cruciate disease, currently favours the use of:

- One single strand suture of 70 lb Leader line in patients <10 kg bodyweight

- One double strand suture of 70lb Leader line in patients 10–15kg bodyweight
- One double strand suture of 80lb Leader line in patients 15–25kg bodyweight
- Two double strand sutures, one of 70lb and one of 80lb Leader line, in patients 25–40kg bodyweight
- Two double strand sutures, both 80lb Leader line, in patients >40kg bodyweight

A figure-of-eight suture is created in this way with the knot lying on the lateral aspect of the joint (Fig. 42.23f). The knots should, whenever possible, be 'tied down' with polydioxanone (PDS, Ethicon) sutures so that the ends do not stand up and irritate the soft tissues. The periosteal flap on the medial aspect of the tibia is then reattached so as to cover the strands of nylon. The fascia of the cranial tibialis muscle is reattached with cruciate/crossed mattress sutures of the polydioxanone. The lateral fascia is closed using PDS (Ethicon) and an overlapping pattern achieved by placement of a row of Mayo mattress sutures followed by a simple, continuous suture along the remaining free edge. The remainder of closure is routine.

Postoperatively, there is no need to use a bandage and the dog's exercise should be restricted to lead walks and room rest for 12 weeks. The dog should be toe touching within a few days, taking reasonable weight on the limb by 4–6 weeks and, by 12 weeks, only mild lameness, if any, should remain. From that point on, exercise should be gradually increased, still predominantly lead exercise, whilst the dog regains muscle strength and confidence in the limb. In most cases, a satisfactory outcome is apparent within 12 weeks of surgery but it often takes up to 4 or 6 months for optimal limb function to be achieved.

Combination of intracapsular and extracapsular techniques

Based on a study in humans by Noyes & Barber (1991) it was considered that a combination of such techniques might be advantageous in the dog. However, a series of studies (Butterworth & Innes, 1994; Coetzee, 1994; Houlton & Dyce, 1994), which were presented at the 1994 British Small Animal Veterinary Association Congress in Birmingham, failed to show any advantage to combining such techniques in clinical cases. However, this option may be worth considering in giant breeds (Miller, 1996).

Complications following surgical treatment of CrCL deficiency using intracapsular or extracapsular techniques

Failure to use the leg

If the dog is still carrying the leg after 4 weeks and the stifle is painful, then the most likely explanation would be that of a low-grade infection. Options in management include empirical treatment with a broad-spectrum antibiotic, e.g. a cephalosporin (Ceporex, Schering-Plough Animal Health), and symptomatic administration of an NSAID such as carprofen (Rimadyl, Pfizer) for 3–6 weeks may resolve the problem, though there would be a degree of concern regarding persistence of infection, especially if permanent suture material had been left *in situ*. Alternatively, the joint could be managed more aggressively by taking joint fluid for analysis and culture, flushing of the joint to reduce bacterial numbers and then administering antibiotics according to the laboratory test results.

One other cause of acute, persistent limb dysfunction is that of peroneal nerve injury or entrapment. The nerve runs caudolateral to the distal femur and could be encroached upon when placing the graft passer either through the joint or behind the fabella.

Prolonged recovery time

Although the average time before maximum limb function is regained following surgery is 3–4 months (Denny & Barr, 1987), some dogs, particularly those of large and giant breeds, show lameness that persists beyond this time but do, eventually have a satisfactory outcome, i.e. do not show any of the identifiable problems discussed below.

Persistence of lameness beyond the usual recovery period

This can be associated with several potential causes:

- *Atrophy of the quadriceps muscle group and loss of proprioception.* Wasting of the quadriceps muscles occurs very soon after injury to the stifle and the CrCL plays a role in proprioception as well as serving a mechanical function. Loss of both of these can create a weakness or lack of confidence in the limb, resulting in lameness without this being associated with pain. In human orthopaedics it is this aspect of cruciate ligament rupture at which most effort is directed in an attempt to regain good knee joint function (Barratt, 1991) and it is quite possible that herein lies the explanation for why some of our patients fail to recover normal limb function. The aim of treatment is to try and improve function and build up the quadriceps muscles. NSAIDs may make the dog more comfortable and boost its confidence to use the leg. Short, frequent walks should be given, preferably on a leash, so that the animal tends to pull and, at the same time, puts weight on the leg. Hydrotherapy and swimming exercise, if readily available, are also useful. If stifle pain persists and a meniscal injury is suspected then further surgery should be undertaken with partial or total medial meniscectomy if necessary and restabilisation of the joint if required.
- Failure to recognise and treat a *meniscal injury*. Possibly the most likely explanation for a persistent moderate to severe lameness.
- *Established osteoarthritis* at the time of surgery. Although this does not preclude a successful outcome, the presence of advanced osteoarthritic change might well result in a persistent lameness, tending to be mild to moderate in degree.
- *Low-grade infection.* This may be associated with persistent lameness, especially where permanent suture materials have been implanted. The most common situations in which this problem is seen is when braided nylon has been used to replace the CrCL (e.g. when placed through tunnels using the Paatsama technique) or has been used to create lateral retinacular sutures (often present with low-grade lameness and sinus formation; Dulisch, 1981). Such problems are best avoided by strict attention to asepsis. Management of such cases is as described above under 'Failure to use the leg' but must also include removal of all the non-absorbable material.
- *Persistent instability*. Although this may be considered undesirable, given the objectives of surgery, instability of the joint is a common finding in dogs examined several months post-surgery (whatever technique is used) and there is no correlation between this finding and the degree of lameness, if any, present. On its own it should not be considered a definitive explanation for persistent lameness.

Recurrence of lameness

A dog may do well following surgery, initially, and then become lame again due to a meniscal injury which develops later (so-called 'late meniscal injury'). The occurrence of this complication is well recognised in an average of 14% of cases (range between 8 and 19% depending on surgical technique) (Metelman *et al.*, 1995). Similarly, a dog which has apparently recovered following surgery may suddenly go lame again as a result of a stifle sprain. At examination there may be a degree of stifle instability present but the significance of this is questionable (see 'Persistent instability' above). Most of these cases respond to a further period of exercise restriction, 10-minute walks on a leash only for 4–6 weeks. In addition, a 4-week course of sodium pentosan polysulphate (Cartrophen-Vet, Arthropharm, Australia) often seems to help dampen down the lameness. Alternatively, a single intra-articular injection of methyl-prednisolone (Depo-Medrone, Pharmacia & Upjohn), 20 mg in a Labrador-sized dog, may also produce the same effect. Fortunately repeat operations are not often necessary and generally, if required, a medial meniscal injury will be identified. In dogs that have long-standing cruciate disease, which has been managed conservatively or surgically, that then require surgery because of lameness that does not respond to conservative measures, it is often found that the joint is stable by virtue of the degree of periarticular fibrosis. In such cases arthrotomy allows inspection of the menisci and resection of any injured portions. Before closure of the joint it may be worth removing any significant osteophytes and drilling two or three tunnels (using a 3.2 or 3.5 mm drill bit) into the femoral condyles from each side.

Such metaphyseal forage may reduce the pain caused by subchondral vascular congestion in an osteoarthritic joint. Although there is no scientific evidence to support such a procedure the authors routinely use it as part of the surgical approach to chronic stifle OA in those patients that do not respond favourably to conservative measures.

Periarticular procedures

Two techniques, tibial plateau-levelling osteotomy (TPLO) (Slocum & Slocum, 1993; Slocum & Devine, 1984) and fibular head transposition (FHT) (Smith & Torg, 1985, Dupuis *et al.*, 1994) have also been advocated for treatment of the cruciate deficient stifle. In the case of TPLO, it has been proposed that caudal sloping of the tibial plateau causes increased stress on the CrCL leading to early failure (Slocum & Devine, 1984). Osteotomy of the tibia allows rotation of the plateau and fixation is then achieved by application of a plate. This neutralises the tibial thrust but does not remove cranial drawer motion and, if it is intact in the first place, release of the medial meniscus is required to avoid injury at a later date. In the case of FHT, the fibular head is released from its normal position, transposed cranially and secured to the tibial crest with a pin and tension band wire. This causes a degree of external rotation of the tibia and changes the alignment of the lateral collateral ligament (attached to the fibular head) such that it passes from caudal to cranial, so acting along a similar line to the original CrCL and providing a cranial restraint for the tibia. These are both considerably more complex than either the 'OTT' or the lateral retinacular (fabellotibial) suture techniques described above and seem to provide no proven advantages. Since neither of the authors currently use either of these techniques, they will not be described further here. However, the relevant references are provided at the end of the chapter.

Partial rupture of the cranial cruciate ligament (CrCL)

The clinical features of partial rupture of the CrCL were described by Strom (1990). The CrCL consists of two bands:

(1) *Caudolateral band* (CLB) – which is taut in extension but loose in flexion. No cranial drawer movement results from rupture of this band.
(2) *Craniomedial band* (CMB) – which is taut in both flexion and extension. With rupture of this band it is possible to elicit a cranial drawer movement when the stifle is in the *flexed* position.

In dogs with partial rupture of the CrCL, it is rupture of the CMB that is seen most often. One of the problems with diagnosing a partial rupture of the CrCL is the lack of a cranial drawer movement, and yet the dog presents with a typical history and signs suggestive of cruciate ligament injury, i.e. a sudden onset of lameness, stifle pain and synovial effusion. It is only possible to elicit a cranial drawer movement with the stifle in flexion, if the medial band of the CrCL is ruptured, and if only the lateral band is ruptured, then the joint will still feel stable in all positions.

Despite the lack of instability, partial cruciate ligament ruptures do cause pain and persistent lameness. There may be concurrent damage to the medial meniscus and untreated cases often progress to complete rupture of the CrCL.

Initially, it is difficult to differentiate a partial CrCL deficiency from a non-specific stifle sprain. Treatment at this stage tends to be 6 weeks of rest and administration of NSAIDs. If lameness does not resolve during this period, and radiographs show evidence of early OA (loss of outline of the infrapatellar fat pad, indicating synovial effusion, and periarticular osteophyte formation) then arthrotomy should be recommended to confirm the diagnosis of partial CrCL rupture. There is a difference of subjective opinion as to whether the intact band of ligament should be left in place or not. Some surgeons claim that it helps to maintain joint stability and contributes towards proprioception within the joint, whilst others claim that it will continue to degenerate, promoting continued inflammatory changes within the joint. Neither of these opinions has any objective results to support it. An overriding factor may be that sacrifice of the remaining band of ligament can be necessary in order to allow adequate examination of the menisci and resection of any damaged portions of these structures. The stifle is then stabilised using

the surgeon's preferred technique. The prognosis is good, with a successful outcome in about 80% of cases.

Meniscal injuries

Anatomy

The stifle contains two menisci located laterally and medially in the femorotibial joint space (Fig. 42.24). The meniscal cartilages are C-shaped and composed of fibrocartilage. The menisci are anchored to the tibia and the femur by five meniscal ligaments and to one another by a single ligament. These are:

- The cranial tibial ligaments of the lateral and medial meniscus
- The caudal tibial ligaments of the lateral and medial meniscus
- The femoral ligament of the lateral meniscus
- The intermeniscal ligament

In addition to the ligamentous attachments, each meniscus is secured peripherally to the joint capsule and the medial meniscus also has an attachment to the medial collateral ligament so it is much less mobile than the lateral meniscus. Only the outer third of each meniscus is directly supplied by blood vessels which are derived from the joint capsule. The inner two-thirds are considered to be avascular.

Injury

The medial meniscus is frequently injured in association with rupture of the CrCL. Flo & De Young (1978) recorded an incidence of 53% in a series of 113 dogs. Similarly, Bennett & May (1991b) recorded meniscal injuries in 49% of a series of 87 dogs with cruciate disease. The medial meniscus is more frequently injured than the lateral because of its more rigid ligamentous attachments. Lateral meniscal injuries are rare and one of the authors has seen an isolated lateral meniscal injury in only one dog.

In the main, meniscal injuries are a problem of the large and giant breeds of dog. The injury is less frequent in small dogs following CrCL rupture, presumably because the leg tends to be carried. In the dog, medial meniscal injury is invariably associated with partial or complete rupture of the CrCL. Unlike the case in man, meniscal damage without CrCL injury is extremely rare in dogs.

During the cranial drawer movement the medial meniscus moves forward with the tibia and the femoral condyle crushes its caudal horn. Repeated crushing can cause meniscal tearing and detachment which in turn causes erosion of the femoral condyle.

Classification of meniscal injuries (Fig. 42.25)

Meniscal injuries can be classified into seven types (Bennett & May, 1991b):

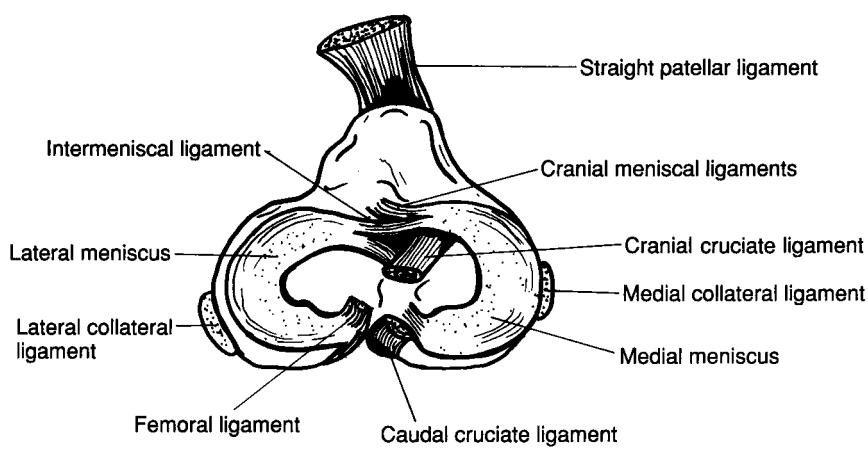


Fig. 42.24 Anatomy of the menisci.

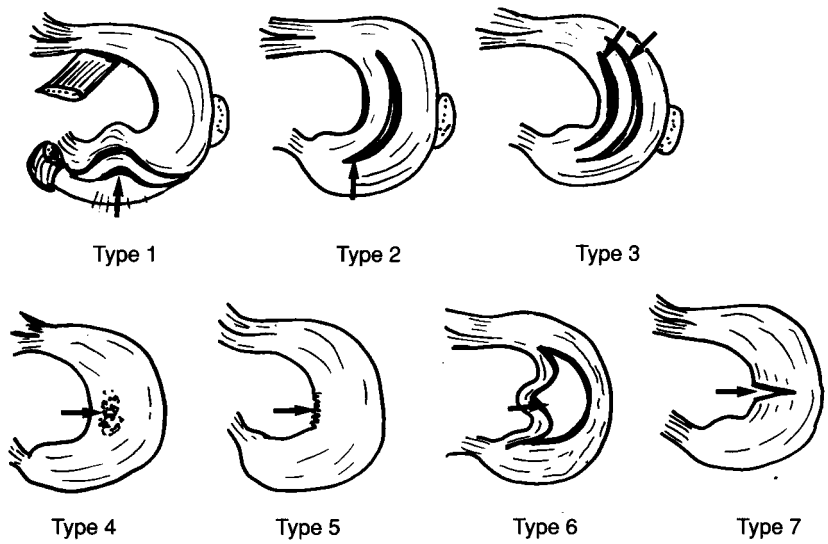


Fig. 42.25 Classification of meniscal injuries (medial meniscus) (Bennett & May, 1991). See text for details.

- Type 1: Caudal detachment with folded caudal horn
- Type 2: Longitudinal tear
- Type 3: Multiple longitudinal tears
- Type 4: Fibrillation/tearing of the surface
- Type 5: Axial fringe tear
- Type 6: Bucket handle tear (similar to type 2 or 3 but inner concave portion, or portion between two longitudinal tears, of meniscus becomes lax and displaced from tibial surface giving bucket handle appearance)
- Type 7: Transverse tear

Types 1, 2 and 6 are seen most frequently. The meniscal parenchyma has poor healing properties (Pearson, 1971) and this has led to the development of techniques for partial or total medial meniscectomy. Experimentally, it has been shown that complete medial meniscectomy results in the development of OA in 3–6 months (Cox *et al.*, 1975). Therefore, only the damaged portions of the medial meniscus should be removed and complete menisctomy should be avoided if possible. Visualisation of the medial meniscus can be difficult. The stifle should be fully flexed and the patella reflected or retracted (see details relating to cruciate ligament surgery above). The stifle distractor (Veterinary Instrumentaion) has now become an essential instrument to allow inspection of the meniscus. It functions like a large Gelpi retractor and separates the femoral condyles from the tibial plateau (Fig. 42.26). Alternatively, the tip of a small Hohmann retractor can be introduced through the femoral intercondylar fossa to engage on the caudal aspect of the proximal tibia (Fig. 42.27). The Hohmann is then used to lever the tibial plateau forward to view the meniscus. A rake retractor can be used at the same time to pull the infrapatellar fat pad cranially in order to improve exposure. Small mosquito forceps and a scalpel fitted with a number 11 blade are usually adequate for partial meniscectomy, though meniscectomy instruments can be useful, particularly when caudal attachments of the caudal horn require sectioning.

Rupture of the cranial cruciate ligament (CrCL) in cats

Rupture of the cranial cruciate ligament (CrCL) in cats is usually an acute traumatic injury associated with gross stifle instability. There is often concurrent damage to the caudal cruciate ligament (CauCL) (Matis & Kostlin, 1978). Uncomplicated

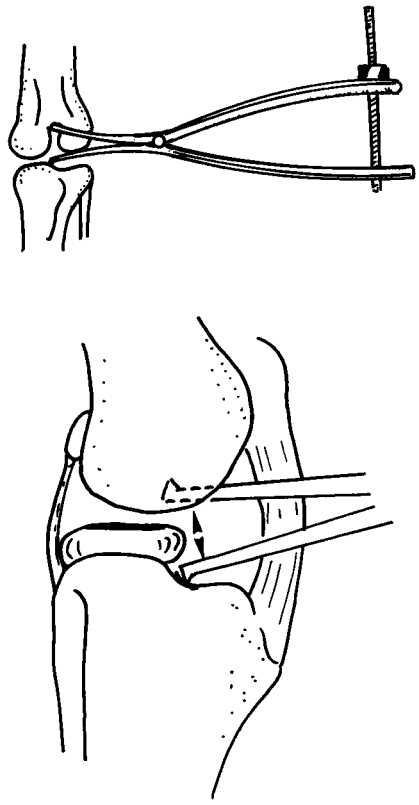


Fig. 42.26 Exposure of the medial meniscus using a stifle distractor (Veterinary Instrumentation).

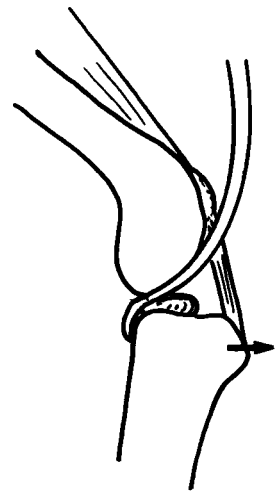


Fig. 42.27 Exposure of the medial meniscus using a small Hohmann retractor to push the tibia cranially.

CrCL ruptures carry a good prognosis with conservative treatment (cage rest). However, stabilisation by extracapsular suturing techniques will give a more rapid and certain recovery.

Rupture of the caudal cruciate ligament (CauCL) in dogs

The caudal cruciate ligament (CauCL) stabilises the stifle by limiting caudal movement and internal rotation of the tibia. The CauCL is well protected by the adjacent ligaments and joint structures. Rupture is uncommon and usually occurs with a concurrent rupture of the CrCL. Nevertheless, solitary ruptures of the CauCL do occur and a series of 14 dogs with this injury was described by Johnson & Olmstead (1987). The condition is seen primarily in large, young, adult dogs and is the result of trauma. Stifle instability with a caudal drawer sign is the main diagnostic feature of CauCL rupture. However, cases are often misdiagnosed, initially, as CrCL ruptures and the correct diagnosis is only confirmed at surgery. None of the cases described by Johnson & Olmstead (1987) had meniscal damage. Extracapsular imbrication techniques to stabilise the joint are favoured by most surgeons and the results are good.

There must be some doubt about the necessity of stabilising the stifle following CauCL injury based on experimental transection of the ligament in dogs (Harari *et al.*, 1987). Six months after transection and partial excision of the ligament, none of the dogs were lame although there was still a positive caudal drawer movement present. None of the dogs developed OA. It was concluded from this work that, in the dog, loss of the CauCL has very little effect on gait compared with loss of the CrCL.

Rupture of the collateral ligaments

Rupture of a collateral ligament may occur alone or in combination with rupture of the CrCL. Manipulation of the stifle reveals abnormal movement in a medial or lateral direction. If the medial

collateral ligament has ruptured, the tibia can be displaced laterally in relation to the femur and the medial side of the stifle can be hinged open. The reverse applies if the lateral collateral ligament is ruptured. Stressed radiographs may be used to document the instability but comparable films of the contralateral limb should be taken to ensure that a widened joint space is not simply a positional artefact.

Treatment

Surgical reconstruction or replacement of the collateral ligament is advisable within a matter of days. Waiting longer than this will reduce the likelihood of identifying the torn ligament and its anatomical origin and insertion, making repair and/or accurate replacement with a prosthesis more difficult.

Exposure of the *lateral collateral ligament* involves a lateral parapatellar skin incision cranial to the lateral collateral ligament. The fascia lata is incised and separated from the joint capsule. Caudal retraction of the fascia lata and biceps femoris muscle exposes the lateral collateral ligament.

Exposure of the *medial collateral ligament* involves a medial parapatellar skin incision cranial to the medial collateral ligament. The deep fascia of the thigh is incised along the caudal edge of the cranial sartorius muscle. The caudal belly of the sartorius is retracted caudally to expose the medial collateral ligament.

Where possible, primary repair of collateral ligament tears should be achieved using 3 metric polydioxanone (PDS, Ethicon), or monofilament nylon, placed in a locking-loop suture pattern (Aron, 1981). Additional, simple horizontal mattress sutures can also be used to appose the edges of the ligament. The repair is protected by using a ligament prosthesis, which is anchored by two screws and washers, one pair placed at the origin and the other at the insertion of the ligament. A figure-of-eight suture is placed around the two screws (Rudy, 1974) (Fig. 42.28). Although wire can be used as the prosthesis, a double strand of 5 metric braided polyester (Ethibond, Ethicon) is preferred by the authors. Suture anchors (e.g. Bone Biter Anchors, Innovative Animal Products, supplied in the UK through Veterinary Instrumentation, Sheffield) can be used in place of the screws/washers to secure the prosthetic material in the bone. However, apart from being slightly quicker to apply and more 'tidy', these offer no significant advantage and are considerably more expensive than the option of using a screw/washer combination.

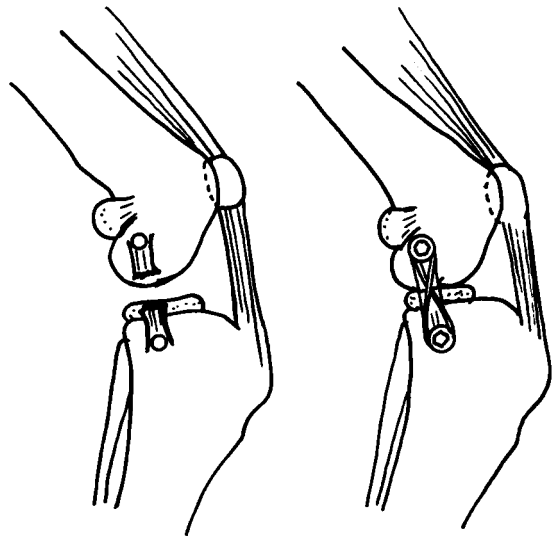


Fig. 42.28 Replacement or reinforcement of medial collateral ligament using two screws and braided polyester.

On the medial side, the collateral ligament is often avulsed from its origin on the femur. If this is treated early (see above) then the ligament can be reattached using a spiked washer and screw, or a ligament staple, and this avoids the need for a prosthetic ligament.

Although prosthetic reinforcement or replacement of the collateral ligament using two screws and a figure-of-eight suture on the medial side is relatively straightforward, on the lateral side the shape of the proximal tibia and the head of the fibula makes it more difficult to place a screw accurately at the insertion of the collateral ligament. This problem can be overcome by anchoring the prosthesis under the head of the fibula or by placing it through a tunnel drilled through the head of the fibula (Fig. 42.29).

If rupture of the CrCL is complicated by rupture of the lateral collateral ligament then the 'OTT' cruciate ligament replacement can be

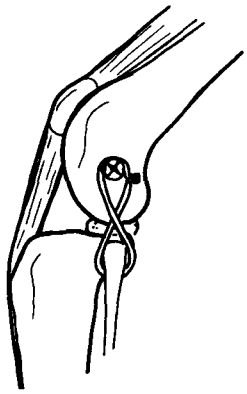


Fig. 42.29 Replacement or reinforcement of lateral collateral ligament using braided polyester anchored by a screw proximally and passed though a tunnel in the fibular head distally.

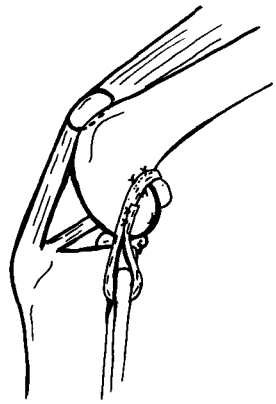


Fig. 42.30 Replacement of the cranial cruciate ligament (CrCL) and the lateral collateral ligament using a fascia lata strip. See text for details.

modified to include replacement of the collateral ligament. During the 'OTT' procedure, a much longer strip of fascia lata is prepared than usual. The CrCL is replaced first and, having secured the fascia lata graft to the lateral femoral condyle, the free end of the graft is passed under the head of the fibula and sutured to itself to create a lateral collateral ligament (Fig. 42.30).

Multiple ligament injuries

Multiple ligament injuries resulting in stifle subluxation or luxation are uncommon in dogs and cats (Denny & Minter, 1973; Phillips, 1982; Aron, 1988; Bruce, 1998). In the dog, stifle luxation is most often seen in adult, working or sporting dogs which have caught the leg in a gate or fence. Rupture of the cranial cruciate, caudal cruciate and lateral collateral ligaments is the most common combination of ligamentous damage observed (Bruce, 1998). There is gross stifle instability, diagnosis of the ligaments involved can usually be determined by careful clinical examination and palpation. Stress radiography is also helpful, particularly in confirming medial or lateral instability and the presence or absence of avulsed fragments of bone. Peripheral tears in the menisci can be sutured using a horizontal pattern of 3 metric polydioxanone (PDS, Ethicon) whereas axial damage is best managed by partial (or total, if absolutely necessary) meniscectomy. It is not usually necessary to replace the CauCL. Reconstruction of the CrCL, the collateral ligament(s) and careful repair of the damaged menisci and joint capsule will result in good to excellent return of function in most cases (Bruce, 1998). However, an injury of this nature should not be underestimated and it is a common mistake to assume the disruption is only slightly more difficult to manage than a joint with cranial cruciate deficiency. The injury requires much more in terms of evaluation and preoperative planning if a similar prognosis is to be achieved.

Surgical 'repair' of the ligaments requires a combination of the techniques detailed earlier in this section. It is easiest to inspect and treat any meniscal injuries before ligament reconstruction is carried out. The order of ligament 'repair' varies with surgeon and with which ligaments are involved. For example, if both cruciates and the medial collateral ligament were ruptured then a medial approach could be used to inspect/repair the menisci, followed by treatment of the medial collateral injury and then stabilisation of the joint with respect to the CrCL instability (the instability caused by the CauCL failure does not require attention). If both cruciates and the lateral collateral ligament were ruptured then a lateral parapatellar approach could be used to inspect/treat the menisci, followed by treatment of the lateral collateral and cranial cruciate instability by whatever method the surgeon deemed appropriate (though fibular head transposition would obvi-

ously not be an option and again the instability caused by the CauCL failure does not require attention).

Stifle luxation in the cat

The principles of treating stifle luxations in the cat are the same as the dog. Surgical replacement of the CrCL alone (+/– meniscectomy), and with no external support, gave good results in a series of nine cats described by Phillips (1982). In the cat, stifle luxations can also be treated by closed reduction, and stabilisation with a transarticular pin (Nunamaker, 1985; Welches & Scavelli, 1990). The pin is introduced through the lateral distal femur just above the lateral trochlear ridge. It crosses the joint space and exits through the medial aspect of the tibia just distal to the tibial plateau. Alternatively, the pin can be introduced in the opposite direction. The pin is removed after 4 weeks, when sufficient soft tissue healing should have occurred to stabilise the joint.

Rupture of the gracilis muscle

(Bateman, 1960; Sanders, 1962; Bateman, 1964; Davis, 1967; Vaughan, 1969; Hickman, 1975; Eaton-Wells, 1992)

The gracilis muscle arises from the pelvic symphysis and inserts on the tibial crest with a second point of insertion, together with the semitendinosus muscle, on the tuber calcis. The gracilis muscle adducts the limb, flexes the stifle and extends the hip and hock joints. Rupture of the gracilis muscle is the most common major muscle injury in the hindlimb of the racing Greyhound (Eaton-Wells, 1992). The right hindleg is most frequently affected and injury to the muscle is usually accompanied by haematoma formation. Tears of the muscle occur in a number of positions; through the belly of the muscle, at the musculotendinous junction and at the conjoined insertion with the sartorius and semitendinosus muscles. The caudal border of the muscle is most frequently involved.

Hickman (1975) recommended that, in new or recent cases, treatment should be directed towards a radical surgical repair rather than conservative treatment (i.e. cold applications, pressure bandages and aspiration to limit the size of the haematoma). Surgical exposure of the medial aspect of the thigh allows inspection of the damaged tissues. The haematoma can be drained, haemorrhage controlled and, more importantly, accurate anatomical reconstruction of the torn muscle can be carried out using a series of mattress sutures. Repair of the torn muscles should allow healing to occur with minimum fibrous tissue formation and give the dog the best chance of racing again. Conservative treatment invariably leads to excessive fibrous tissue formation which may require surgical release (Bateman, 1964) to improve the dog's action.

Contracture of the gracilis muscle in the German Shepherd Dog

(Vaughan, 1979; Capello *et al.*, 1991)

Contracture of the gracilis muscle in the German Shepherd Dog causes a characteristic alteration in gait. The condition is recorded in dogs between 3 and 7 years of age and affected animals have usually led extremely active lives. Gait changes suddenly and deteriorates over a period of 6 weeks before remaining relatively static. When walking, the affected leg is raised in a jerky fashion with the hock hyperflexed and rotated outwards while the foot is rotated inwards. The gracilis muscle can be palpated as a taut band on the medial aspect of the thigh. The condition may become bilateral.

Owners should be advised that lameness is mechanical, there is no pain and affected animals can continue to lead an active life despite the condition. Surgical treatment is hard to justify because although section of the gracilis tendon of insertion immediately restores a normal action, the gait abnormality almost inevitably recurs within 3–5 months of surgery. This recurrence is also seen if the entire muscle belly is removed because the semitendinosus becomes affected.

Stifle arthrodesis

Stifle arthrodesis is only occasionally necessary and is used most frequently for the relief of pain

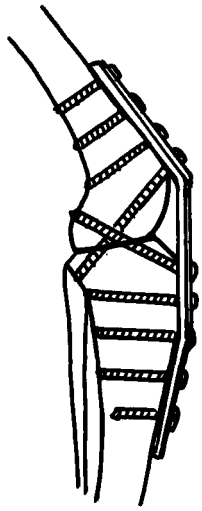


Fig. 42.31 Stifle arthrodesis using a plate.

associated with severe degenerative joint disease. The stifle is exposed through a craniolateral skin incision. Osteotomy of the tibial crest with dorsal reflection of the quadriceps allows a wide exposure of the joint. Ostectomies of the distal articular surface of the femur and proximal tibia are planned to allow the stifle to be fixed at an angle of 140° (Fig. 42.31). A saw or osteotome is used to remove the articular surfaces together with the intra-articular structures. The stifle is stabilised temporarily with crossed Kirschner wires while a dynamic compression plate (DCP) is contoured and applied to the cranial aspect of the distal femur and proximal tibia. The tibial crest is reattached on the medial side of the tibia using a lag screw.

Dogs and cats accommodate arthrodesis well (Cofone *et al.*, 1992) although there may be a tendency to knuckle over on the digits for the first 3–4 weeks. Exercise should be restricted for at least 8 weeks following surgery. Plate removal 6–9 months after surgery is recommended to minimise the risk of tibial fracture at the distal end of the plate.

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Chapter 43
The Tibia and Fibula

Conditions of the tibia and fibula

Conditions affecting the tibia and fibula may be listed as:

- *Traumatic*
  - Proximal fractures
    - avulsion of the tibial tuberosity
    - fracture of the proximal physes
    - fracture of the fibular head
  - Diaphyseal fractures
  - Distal fractures
    - fractures of the distal physes
    - fractures of the malleoli (see Chapter 44, p. 585)
- *Developmental*
  - Asynchronous growth of the proximal tibial physis causing angulation of the tibial plateau (see Chapter 42, p. 516)
  - Asynchronous growth of the distal tibial physis causing pes valgus or varus
  - Synchronous but retarded physeal growth causing crural shortening (rarely requires treatment)
- *Acquired*
  - Osteosarcoma of the proximal or distal tibia (see Chapter 51)

Tibial/fibular fractures

Fractures of the tibia and fibula are fairly commonplace injuries in small animal practice. In one study (Phillips, 1979) they represented 14.8% of 284 canine fractures and 5.4% of 298 feline fractures, whilst in a more recent study by Ness *et al.* (1996) they represented 15.6% of 160 canine fractures and 18.5% of 124 feline fractures. Such injuries are usually a result of involvement in a road traffic accident, but other causes such as dog fights or trapping a foot whilst moving at speed are also reported. Fracture of the fibula occurs rarely without concurrent fracture of the tibia and repair of the latter is, in most cases, sufficient. The exceptions may be fractures at each end of the fibula where involvement of sites of ligamentar attachment may demand specific attention. The anatomy of the crus makes it feasible to employ a number of methods to stabilise tibial shaft fractures, namely casts, intramedullary pins, external skeletal fixators (ESFs), and bone plates and screws. The large choice of methods may lead to confusion in decision-making and one of the aims of this chapter will be to try and help the reader to make an appropriate choice in any given situation.

Fractures of the proximal tibia and fibula

In the vast majority of cases these involve physes and so are seen in skeletally immature patients. The most commonly seen fractures in this region are illustrated in Fig. 43.1. Special considerations apply to such patients (see Chapter 15) and these revolve around not only trying to re-establish normal joint congruity but also to prevent, or at least not accentuate, any potential for future growth deformity.

Avulsion of the tibial tuberosity

This injury is almost exclusively seen in animals less than about 10 months of age and the Grey-

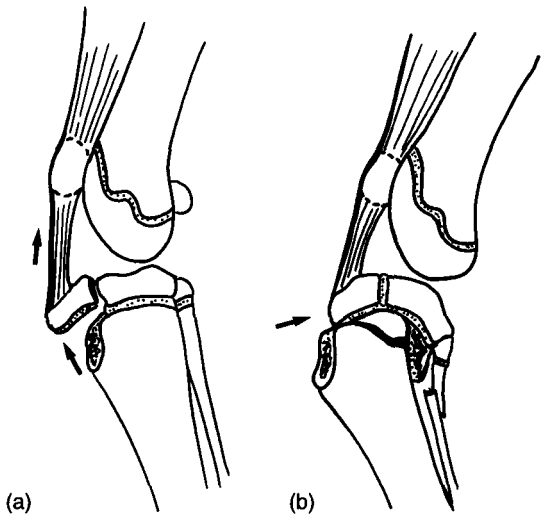

Fig. 43.1 Fractures of the proximal tibia. (a) Avulsion of the tibial tuberosity. (b) Salter Harris type 2 fracture of the proximal tibial growth plate with caudal rotation of the epiphysis.

hound and Terrier breeds seem to be overrepresented. The tibial tubercle forms as a separate centre of ossification and serves as the point of insertion for the straight patellar ligament. Avulsion of the tubercle renders the dog unable to fix the stifle during weight-bearing. Swelling will be present on the cranial aspect of the joint, the tubercle may be palpated proximal to its normal position and the patella will be positioned proximally in the trochlear groove. Radiography provides a definitive diagnosis (Fig. 43.2).

Management

If the only clinical signs are lameness associated with swelling, and radiography shows only a partial avulsion, then the patient may be treated conservatively. Casting or splinting is unlikely to be effective and management should comprise strict cage rest and monitoring in case the avulsion should become complete. In all cases where complete avulsion has occurred open reduction and internal fixation are required to re-establish the integrity of the quadriceps complex. A dilemma exists between the requirements of an avulsion fracture and involvement of a physis. Ideally, avulsion fractures should be treated,

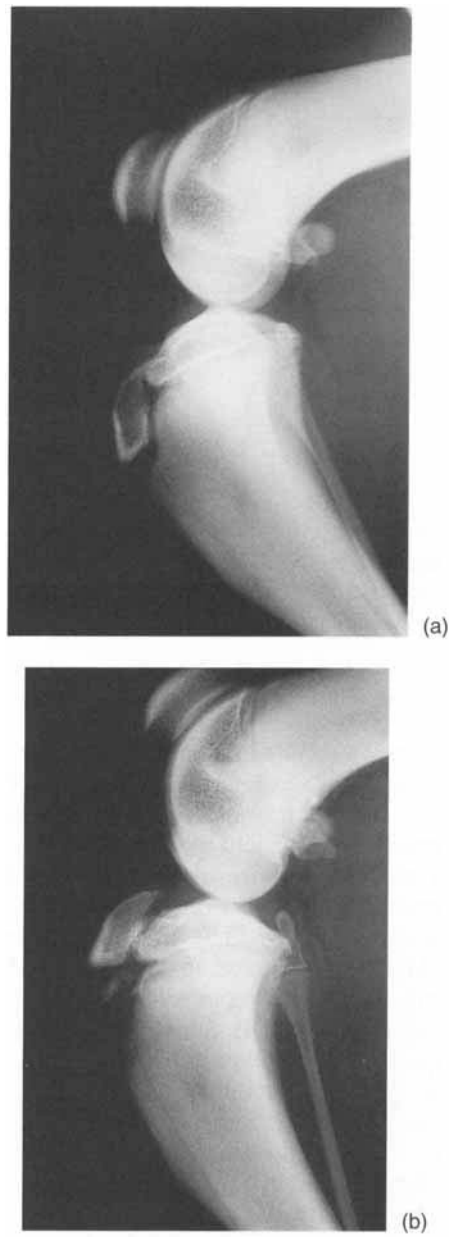

Fig. 43.2 (a) Mediolateral radiograph of the normal stifle of a 6-month-old Greyhound. (b) Mediolateral radiograph of the same dog's contralateral stifle showing avulsion of the tibial tuberosity. (Source: BSAVA Publications, Cheltenham.)

wherever possible, using the tension band principle. However, the compression this creates at the fracture site may lead to early closure of the physis and subsequent deformity.

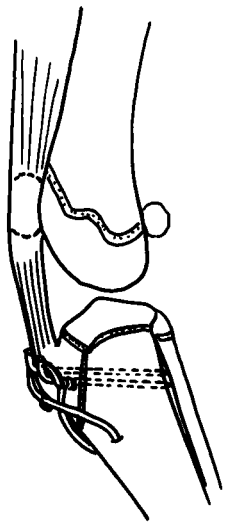

Fig. 43.3 Surgical treatment of an avulsion fracture of the tibial tuberosity. Fixation using Kirschner wires and a wire tension band.

The recommended method of internal fixation involves the placement of two Kirschner wires through the tubercle and into the proximal tibia, to prevent rotation, and then securing a figure-of-eight tension band wire around these and through a transverse tunnel drilled more distally in the tibial crest (Fig. 43.3). In some patients it is difficult, due to their size, to pass two pins through the tubercle. A single pin will suffice in such circumstances (Fig. 43.4). Some surgeons prefer to secure the tubercle in position with a lagged bone screw, +/– an anti-rotational Kirschner wire and/or tension band wire. Since such a technique will create static compression of the physis, it can only be recommended in patients already approaching skeletal maturity.

In patients approaching skeletal maturity, by the time the fracture has healed, i.e. 8–10 months of age, the implants may be left in place, but in much younger patients they should be removed after about 5 weeks to try and prevent early closure of the physis and subsequent drifting of the tubercle distally relative to the tibial shaft. Alternatively, in these young patients, absorbable implants may be used. Polylactic acid pins have now become available and could be used to

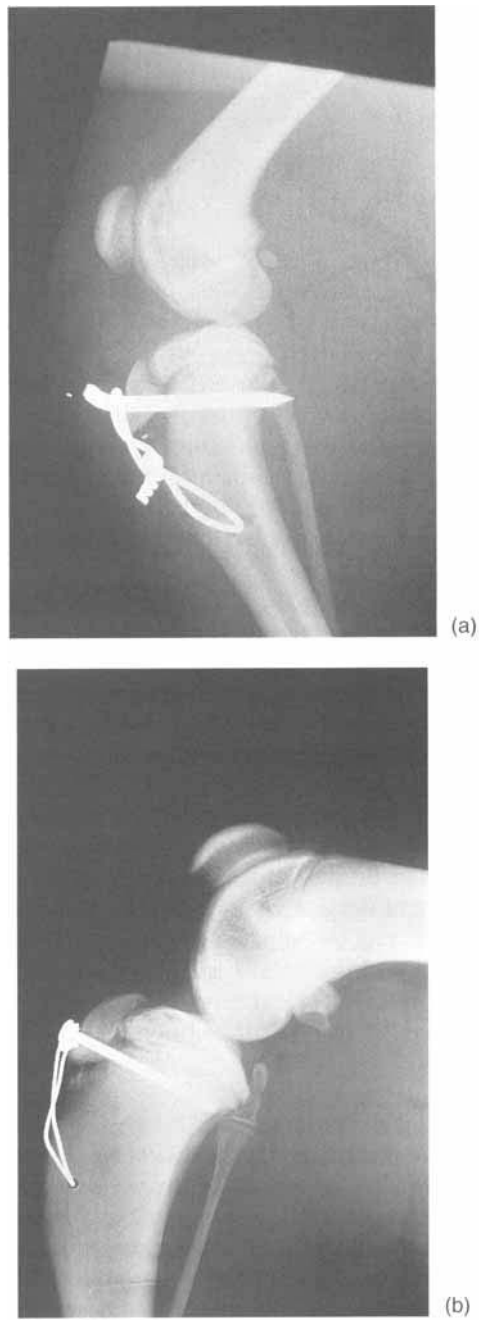

Fig. 43.4 (a) Mediolateral radiograph of a 7-month-old Border Terrier's stifle after a tibial tuberosity avulsion has been stabilised using a single Kirschner wire and wire tension band. (Source: BSAVA Publications, Cheltenham.)
(b) Mediolateral radiograph of the same stifle as in Fig. 43.2b taken after the tibial tuberosity avulsion had been stabilised using a lagged bone screw and wire tension band.

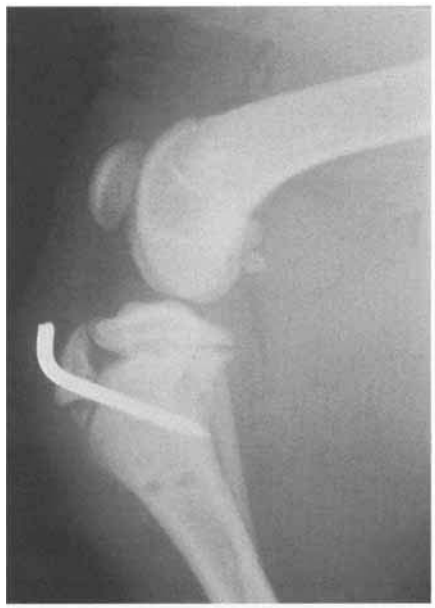

Fig. 43.5 Mediolateral radiograph of a 6-month-old Tibetan Terrier's stifle after a tibial tuberosity avulsion had been stabilised using a Kirschner wire and polydioxanone suture.

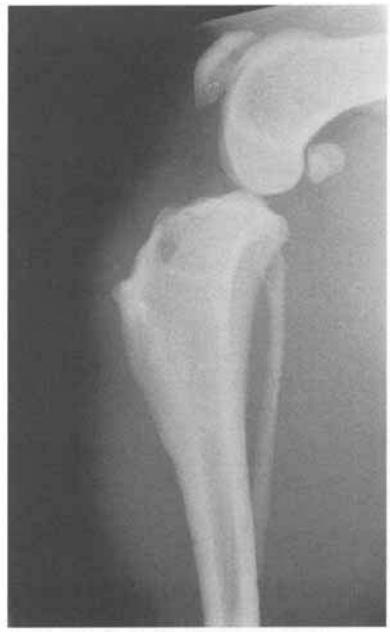

Fig. 43.6 Mediolateral radiograph of the same stifle as in Fig. 43.4a taken 8 weeks later. Despite the implants being removed 4 weeks after fixation the tibial tuberosity has failed to develop normally leading to the deformity seen. (Source: BSAVA Publications, Cheltenham.)

reattach the tubercle and a figure-of-eight PDS (Ethicon) suture can be used to replace the wire tension band (Fig. 43.5). It must be remembered, though, that premature closure of the physis may result from the injury itself and deformity can be seen whatever treatment method is chosen, even if a tension band is avoided or removed early (Fig. 43.6).

In cases where only a small part of the tubercle has become avulsed then reattachment of the patellar ligament to the tibia is best achieved by placement of tendon sutures through the ligament and through transverse bone tunnels in the tibial tuberosity/crest (Fig. 43.7) (see Chapter 42, p. 526).

Surgical technique

The patient should be positioned in dorsal recumbency with the affected limb extended caudally. Free limb draping is most appropriate to allow manipulation of the stifle joint during open reduction. The surgical approach involves a

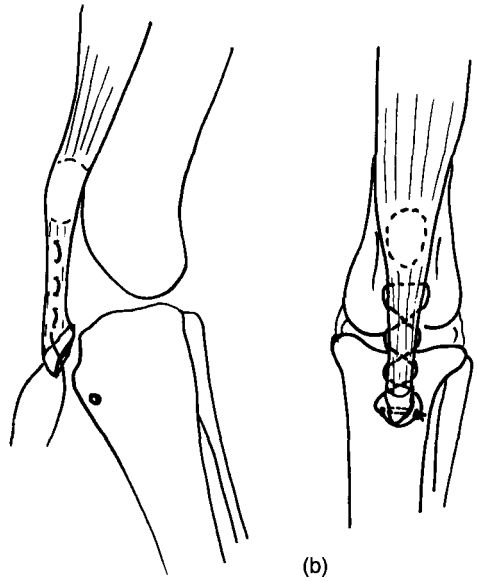

Fig. 43.7 (a) Avulsion of the straight patellar ligament with a small fragment of the tibial tuberosity. A Bunnell suture is placed in the ligament. A hole is drilled transversely through the tibial tuberosity. (b) Ligament plus avulsed fragment reattached to the tibia with a suture.

craniolateral incision extending from just below the level of the patella to about two-thirds of the way down the tibial crest. Soft tissue dissection should allow identification of the tibial tubercle and removal of any organising haematoma should expose the fracture surface on the tibial crest. If a tension band wire is to be applied then reflection of the cranial tibialis muscle, from the lateral aspect of the tibia, is required to expose the site for drilling of the transverse tibial tunnel. Reduction of the fracture is most easily achieved with the stifle extended but maintaining reduction whilst implants are placed can be difficult since the fragment is often too small to be held with forceps. The traction created by the quadriceps muscle can be counteracted by the application of pointed forceps to the patella or Allis tissue forceps to the patellar ligament and using these to draw the fragment distally.

Placement of implants through the tubercle may be preceded by predrilling if the fragment is very small. Pins should be bent through 90° close to the bone to reduce the likelihood of migration. Tightening of the figure-of-eight tension band should, ideally, be achieved through the placement of 'knots' on both the lateral and medial arms of the figure-of-eight but in small dogs a single 'knot' will usually suffice, and placing this laterally makes burying the wire ends easier. Closure should include reattachment of the fascia of the cranial tibialis muscle to the cranial aspect of the tibia.

Postoperatively the joint may be supported in a padded dressing for 5–10 days and the patient should be rested until fracture healing has taken place, usually by 4–6 weeks. Implant removal may have to be considered as discussed above.

Separation of the proximal tibial physis

This is an uncommon injury seen only in immature patients. It is associated with caudal rotation of the tibial plateau and craniomedial displacement of the proximal tibial metaphysis, most easily noted radiographically (Fig. 43.8). Such rotational deformity is severely disabling since the stifle cannot be fully extended. Marked lameness will be seen, associated with pain and swelling around the stifle.

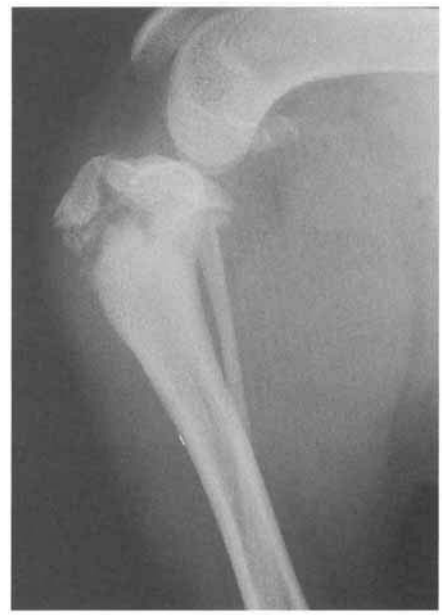

Fig. 43.8 Mediolateral radiograph of a 6-month-old Shetland Sheepdog's stifle showing a displaced fracture of the proximal tibial growth plate. Note that the tibial tuberosity has remained attached to the plateau. (Source: BSAVA Publications, Cheltenham.)

Management

If the only clinical signs are lameness associated with swelling and radiography shows only minimal displacement then the patient may be treated conservatively. Casting or splinting may be beneficial but management should also include strict cage rest. In all cases where caudal displacement of the plateau has occurred, early open reduction and internal fixation are required to re-establish joint congruity. The plateau may be secured in place using crossed Kirschner wires (Fig. 43.9a) or a single intramedullary pin in larger patients. However, all such implants sit close to the articular margins and may interfere with normal joint function making it necessary for them to be removed. In most cases the tibial tubercle remains attached to the plateau and, following open reduction, stability may be achieved by placement of a figure-of-eight wire anchored under the insertion of the patellar ligament and through a transverse tunnel in the tibial crest, and/or placement of a Kirschner wire through the

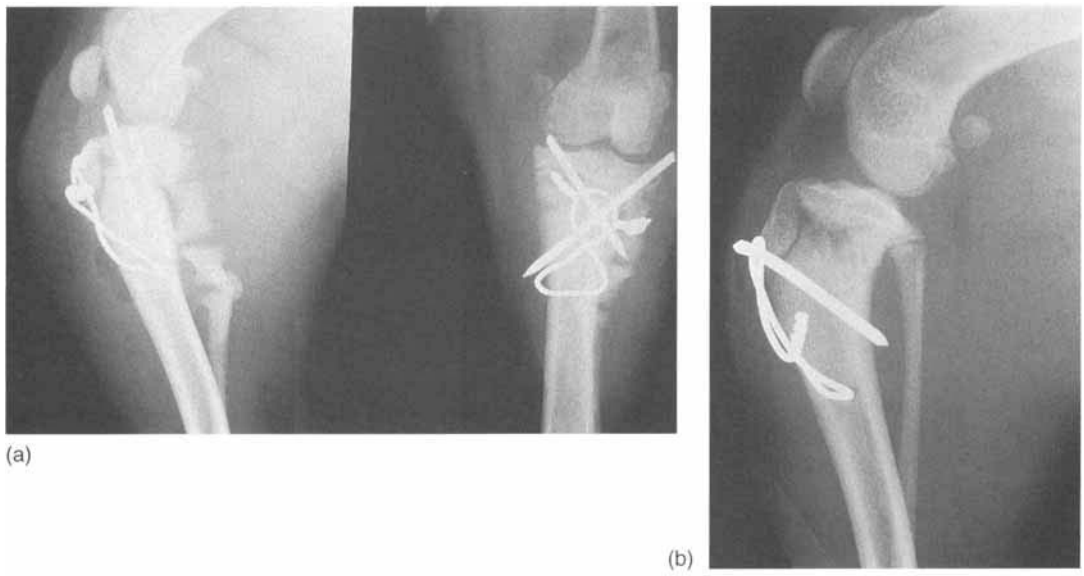

Fig. 43.9 (a) Mediolateral and craniocaudal radiographs of an 6-month-old West Highland White Terrier's stifle taken after open reduction of a proximal tibial growth plate fracture and fixation using crossed Kirschner wires and a wire tension band from the patellar ligament. Note the degree of callus formation around the fibular fractures. The injury had been presented for treatment 3 weeks after the onset of lameness.
(b) Mediolateral radiograph of the same stifle as in Fig. 43.8 taken after open reduction and stabilisation using a Kirschner wire placed through the tuberosity and a wire tension band. (Source: BSAVA Publications, Cheltenham.)

tubercle and into the tibia (Fig. 43.9b). If the tubercle is used to create stability then the points discussed above under 'Avulsion of the tibial tuberosity' would be applicable.

Surgical technique

The patient should be positioned and draped as described under 'Avulsion of the tibial tuberosity' above. The surgical approach involves a craniomedial incision extending from just below the level of the patella to about two-thirds of the way down the tibial crest. Soft tissue dissection should allow identification of the tibial plateau and removal of any organising haematoma should expose the fracture surfaces. If a tension band wire is to be applied then reflection of the cranial tibialis muscle from the lateral aspect of the tibia is required to expose the site for drilling of the transverse tibial tunnel. Reduction of the fracture is most easily achieved with the stifle extended but care should be taken with the tibial plateau since it may split if too much leverage is applied. In most cases reduction can be achieved by holding the stifle in extension and placing a small Hohmann retractor into the fracture space from the caudomedial aspect and gently levering the plateau forwards. Digital pressure is usually the most practicable way of holding the plateau in reduction whilst the implants are placed. If at all possible the pins should be bent over to avoid implant migration. Placement of implants and tension band wires in the region of the tibial tubercle is described above.

Postoperatively the joint may be supported in a padded dressing for 5–10 days and the patient should be rested until fracture healing has taken place, usually by 4–6 weeks. Implant removal may have to be considered as discussed above.

Fractures of the fibular head

These fractures occur rarely in isolation. If they do result from a lateral blow then there may be pain

and/or swelling on the lateral aspect of the stifle, and pain on joint manipulation. The majority of these rare events are not associated with separation of the fibular head from the tibia and can be treated conservatively. If such separation were found then this could result in lateral instability of the stifle due to weakening of the insertion of the lateral collateral ligament. In such a case it would be advisable to reattach the fibular head to the tibia using either a lagged bone screw or a pin and tension band wire.

Fractures of the tibial and fibular diaphyses

Such injuries usually occur in combination, as a result of a road traffic accident or trapping the distal limb whilst moving at speed or turning, and it is the tibial fracture that is the more important. The fibula bears little weight and shaft fractures of this bone alone may be treated conservatively. In cases where both are fractured, reduction and stabilisation of the tibia will amply realign and protect the fibula during fracture healing. Where the fibula remains intact in the face of a tibial fracture the support offered by the intact bone will greatly support the tibial repair. The sparcity of soft tissue cover in the mid and distal diaphysis results in an increase in the likelihood of such fractures being open, makes the surgical exposure of the fracture relatively straightforward but may also lead to a reduced rate of fracture healing.

As was mentioned at the beginning of the chapter, the anatomy of this region makes it plausible to utilise a multitude of techniques in the management of fractures. It is, therefore, intended that this section should discuss each of these possibilities in turn and assist the reader to understand in which situations each of these is most appropriate. Owing to the natural twist in the tibia, fractures tend to spiral along the shaft and hairline fissures that extend beyond the radiographically visible fracture lines are not uncommon.

Surgical approach to the tibial diaphysis (Fig. 43.10)

To some extent the approach varies with the method of fixation chosen. Preparation of the patient is the same for all except external casting. The surgical exposure will be described for the purpose of applying a bone plate, but if limited exposure is required for intramedullary pinning, or introduction of lagged bone screws that are to be protected with an ESF, then the description should be modified accordingly.

The entire limb from just proximal to the pes is clipped and prepared. The patient may be positioned in lateral recumbency with the affected limb down, to allow access to the medial aspect, but with allowance to lift the limb off the table in case full pins are to be used. The contralateral limb is drawn out of the surgical field and a rope or bandage sling is secured to the table passing

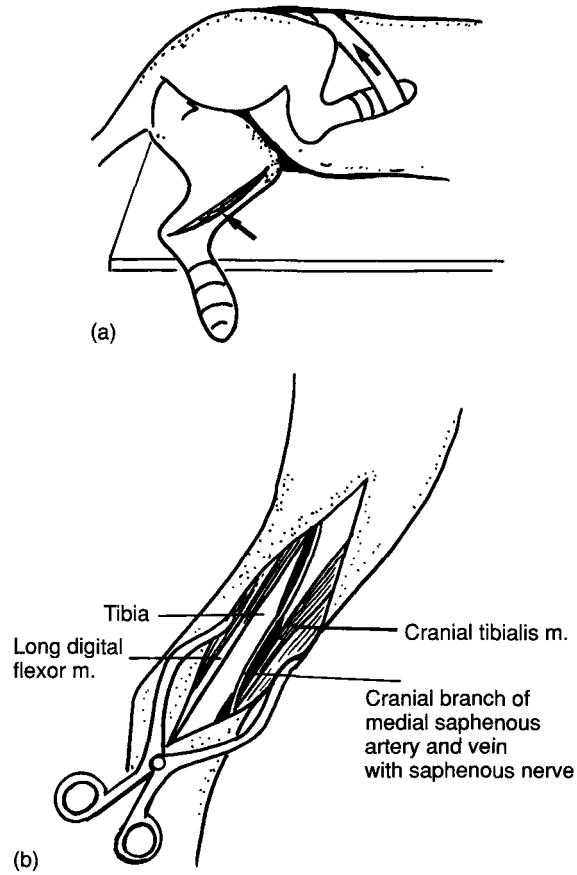

Fig. 43.10 Surgical approach to the medial aspect of tibial diaphysis. (a) Craniomedial skin incision. (b) Skin reflected to expose diaphysis.

medial to the affected limb proximally so that tension at the fracture site can be achieved by drawing on the pes without this causing movement of the patient. Alternatively, the limb may be suspended from a ceiling hook, or drip stand, which allows 360° access to the crus. Free limb draping is necessary to allow adequate exposure and manipulation. Where intramedullary pins or a bilateral external fixator are to be used then access to the limb can be improved by placing the patient more in dorsolateral recumbency with the limb supported on a sandbag so that it can be lifted, allowing manipulation of the stifle, for intramedullary pinning, or placement of full fixation pins, in the case of a bilateral external fixator.

A craniomedial skin incision is made along most, if not all, of the tibial length. If the incision is made too medially then the closure will lie directly over the plate and make problems with wound healing more likely. Dissection through the subcutaneous fascia will expose the tibial shaft easily with the cranial tibial muscle forming the cranial margin and the long digital flexor muscle the caudal margin. The only complicating structures are those of the cranial branch of the medial saphenous artery and vein which run alongside the saphenous nerve. All three structures cross the medial aspect of the tibia in a caudoproximal to craniodistal direction about halfway along the diaphysis. Although it is preferable to try and preserve these structures they can be ligated and sectioned, in order to reduce operating time, without causing serious complications. Closure is achieved by apposition of the subcutaneous and/or subcuticular fascia and then the skin.

Conservative management

In certain cases the most appropriate management may involve nothing more than strict rest with or without a support bandage or external cast, depending on the inherent stability of the fracture following closed reduction, for 4–6 weeks.

Such management is most appropriate when most or all of the following criteria are fulfilled (Fig. 43.11):

- The fracture lines are hairline or minimally displaced
- The patient is young, preferably skeletally immature
- Manipulation of the crus reveals good inherent fracture stability especially with respect to rotational and axial forces

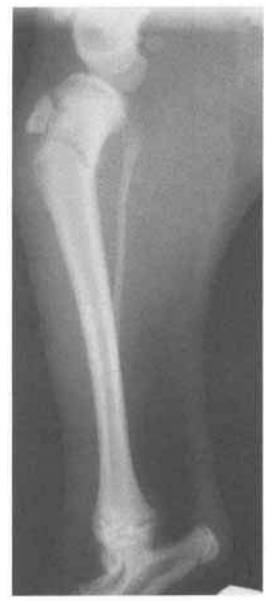

Fig. 43.11 Mediolateral radiograph of a 3-month-old Yorkshire Terrier's tibia showing a stable, transverse fracture of the proximal diaphysis/metaphysis.

Conversely such management should not be employed when:

- The fracture line is simple but rotationally unstable after reduction
- The fracture is comminuted, creating poor stability in all senses
- The patient is middle- or old-aged
- The fracture is open

The method of application of an appropriate cast is discussed in Chapter 12, p. 93. The main problems are those of 'cast sores' and fracture disease, which are usually reversible once the cast is removed, and the more serious complications of rotational deformity, usually involving lateral rotation of the pes, or non-union resulting from rotational instability of the fracture site. Such

deformity can only be rectified by refracturing the bone and non-union requires surgical intervention which always exceeds the effort that would have been required to treat such a rotationally unstable fracture more appropriately in the first place. The management of such severe complications is discussed elsewhere (Chapter 13).

Intramedullary (IM) fixation

Intramedullary (IM) pins afford good protection against angular forces acting at a fracture site but provide little resistance to rotational or axial forces. As a result, the use of IM pins alone is most appropriate when:

- The fracture line is transverse, i.e. axially stable, and interdigitation of the two fragments creates rotational stability (Fig. 43.12)
- The patient is young, preferably skeletally immature, so that early callus formation will create rotational stability to counteract the instability resulting from resorption of the fracture ends

In addition, IM pins may be combined with other implants which provide auxiliary stabilisation in situations where:

- Transverse fractures can be afforded rotational stability by application of a unilateral, uniplanar ESF, often requiring only one proximal and one distal half-pin and to be *in situ* for only 2–3 weeks, by which time the callus will provide adequate rotational stability (Fig. 43.13a)
- Reconstructible, spiral or comminuted fractures can be afforded rotational and axial stability by accurate reduction and compression of the fracture surfaces using cerclage wires (Fig. 43.13b)
- Non-reconstructible, comminuted fractures can be afforded rotational and axial stability by application of a 'load-sparing' external fixator with a unilateral or bilateral, uniplanar configuration and four or more fixation pins

Intramedullary pinning, with or without auxiliary methods of fixation, is probably best avoided when:

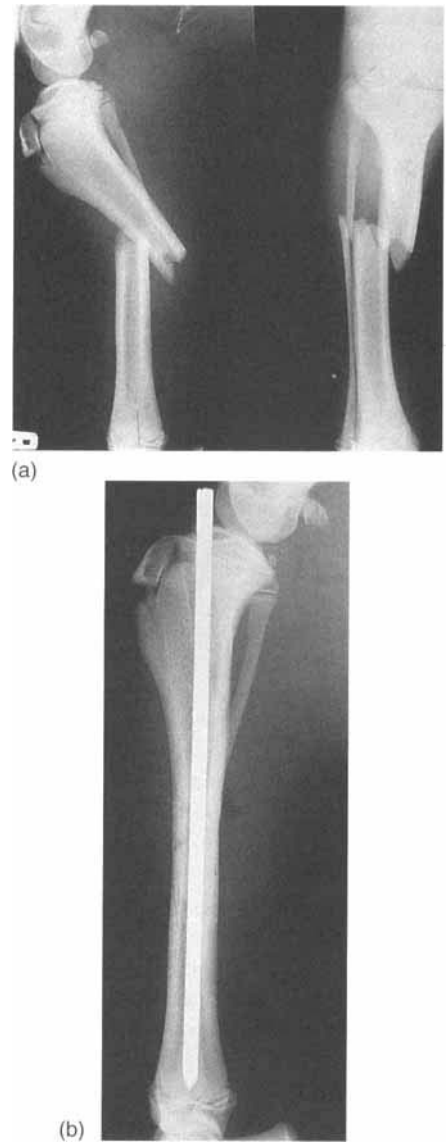

Fig. 43.12 (a) Mediolateral and craniocaudal radiograph of a 6-month-old crossbreed's tibia showing a transverse, mid-diaphyseal fracture. (b) Same tibia postoperatively showing stabilisation using an intramedullary (IM) pin.

- The tibial conformation is such that placement of a straight IM pin would not regain a semblance of normal anatomy, e.g. in some of the chondrodystrophoid breeds, where this might cause delayed healing and/or clinically significant malunion

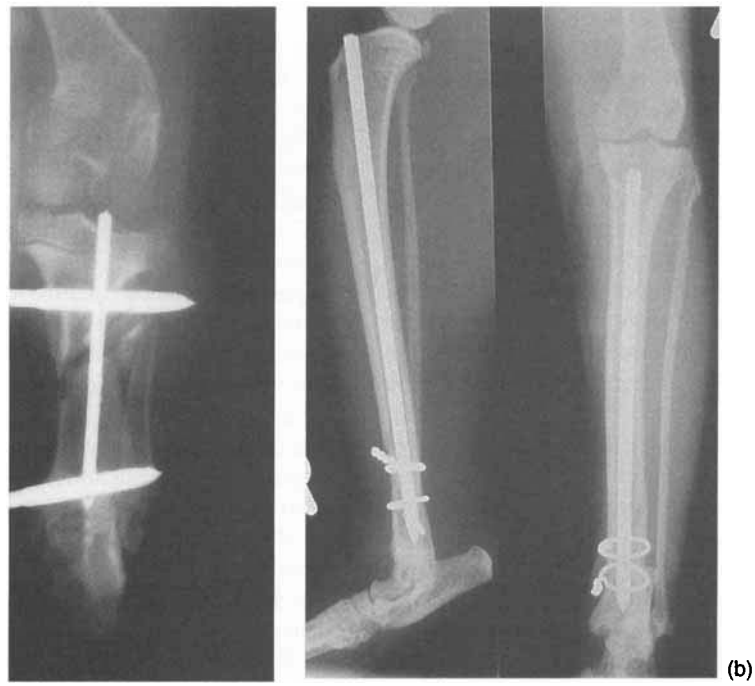

Fig. 43.13 (a) Craniocaudal radiograph of a 4-month-old Miniature Dachshund's tibia showing the use of an intramedullary (IM) pin and two-pin fixator to stabilise a transverse fracture. (b) Mediolateral and craniocaudal radiographs of an 8-year-old cat's tibia showing the use of an intramedullary (IM) pin and cerclage wires to stabilise an oblique/spiral fracture of the distal diaphysis.

- The fracture is open and rather than interfere further with the fragments and possibly assist spread of the contamination within the IM canal it would be more appropriate to utilise an ESF alone to stabilise the fracture
- The age and/or size of the patient, together with the configuration of the fracture, makes it likely that auxiliary fixation may fail or need repeated adjustments before healing is adequate, and use of a bone plate and screws might be considered more appropriate in reducing postoperative management and complications

Intramedullary, interlocking nails have now been developed (Innovative Animal Products, supplied in the UK through Veterinary Instrumentation, Sheffield) which have transverse holes into which can be secured bone screws passing through one or both cortices. Such interlocking, IM nails afford rotational and axial, as well as angular, stability and can be used in many situations where an IM pin would require augmentation with cerclage wires or an ESF, or indeed as an alternative to a buttress plate or external fixator which might be used to bridge a severely comminuted fracture. Neither of the authors has any experience with these implants but their use in the tibia has been reported (Muir *et al.*, 1993) and will, no doubt, become more widespread with time.

Surgical technique

It is advisable to include a range of IM pins in the surgical pack, in case the first choice has to be abandoned, and appropriate orthopaedic wire for cerclage wiring, together with wire twisters and cutters, even if use of these has not been planned, since fissure lines may be found at surgery that were not evident radiographically. Choice of the

implant is based on the principle of it occupying 65–75% of the medullary canal's diameter and this can be evaluated preoperatively from radiographs of the fractured bone or, better still as there is less distortion, of the contralateral limb. A slightly smaller pin may be required if an external fixator is to be applied as well. The use of plain Steinmann pins is to be advocated and the use of partially threaded pins discouraged (as discussed in Chapter 12). Precutting of the IM pin is advisable wherever possible since it markedly reduces surgical time, makes it easier to ensure the pin protrudes minimally at its proximal end and avoids iron filings being left in the soft tissues when a suitably sized pin-cutter is not available and a hacksaw is used. The required length of pin is estimated from the preoperative radiographs of the unaffected limb since evaluating the required length from the fractured bone usually leads to placement of too short a pin. In order to ensure that the pin can be broken intraoperatively, without excessive manipulation, it is necessary to cut away about 50% of the cross-sectional area of the pin. Whether this is done on one side or circumferentially is a matter of surgeon's preference but the authors tend to use the latter. In skeletally immature patients it may be preferable to remove the IM pin once healing has taken place. If this is the case then precutting of the pin is not necessary since enough will have to remain proud of the tibia to allow retrieval. When cutting the implant intraoperatively it is preferable to use pin-cutters, rather than a hacksaw, for the reasons mentioned above.

The fracture site is exposed using a limited craniomedial approach. In the case of reconstructible, comminuted fractures the fragments are reduced and compressed into position using cerclage wires until a two-piece fracture is achieved. When applying these it must be ensured that the fibula is not included since this will make it impossible to achieve adequate tension in the wire. The proximal part of the tibial diaphysis is wedge-shaped and to prevent slipping of the wire it may be necessary to create a notch in the surface of the bone or apply the wire in a hemicerclage fashion. If the wire is tightened by twisting the two ends around one another, then it is usually necessary to bend the ends over as there is inadequate soft tissue cover to consider the option of leaving them standing at right angles to the bone surface. It is inappropriate to place the pin first and then try to reconstruct the fragments as some bone length will have been lost and accurate anatomical alignment will not be possible. The resulting fracture gaps will create extra strain on the wires leading to their loosening and loss of the auxiliary stability they were intended to achieve.

Once a two-piece fracture has been created and fissure lines have been protected with cerclage wires, the IM pin can be introduced. This may be achieved in a normograde or retrograde fashion. The latter can be used but it is imperative that the stifle joint is avoided by careful attention to where the pin exits the tibia and keeping the stifle flexed. In order to avoid the joint there is a tendency to aim the pin cranially.

Problems may arise if the fracture is very proximal, when the pin may be driven from the caudal cortical surface, heading cranially and resulting in excessive caudal rotation of the tibial plateau when the pin is introduced into the distal diaphysis. Conversely, in more distal fractures the pin may glide off the cranial cortex, in a Rush pin fashion, and head more caudally than intended. Therefore, although retrograde pinning is possible, with care, it is generally believed that normograde pinning is most appropriate for tibial fractures. The pin is introduced alongside the medial border of the straight patellar ligament through a keyhole incision with the stifle held flexed. It enters the bone at the base of the tibial crest, cranial to the intermeniscal ligament (Fig. 43.14). Slippage of the pin off the proximal tibia can be a problem, whatever means of application is used, and it may be useful to predrill the proximal hole into the medullary canal with a smaller pin (better than a drill as it causes less soft tissue damage). Whenever possible the notch in a precut pin is protected by being kept within the chuck in order to prevent premature breakage. Once the pin appears at the fracture site and then on into the distal fragment after final reduction, resistance should be met as it embeds in the distal metaphysis. As the pin approaches the distal metaphysis it is perhaps better to abandon a power drill, if one has been used, in favour of a Jacob's chuck, which provides a better feel for what the pin is passing through and makes it far less likely

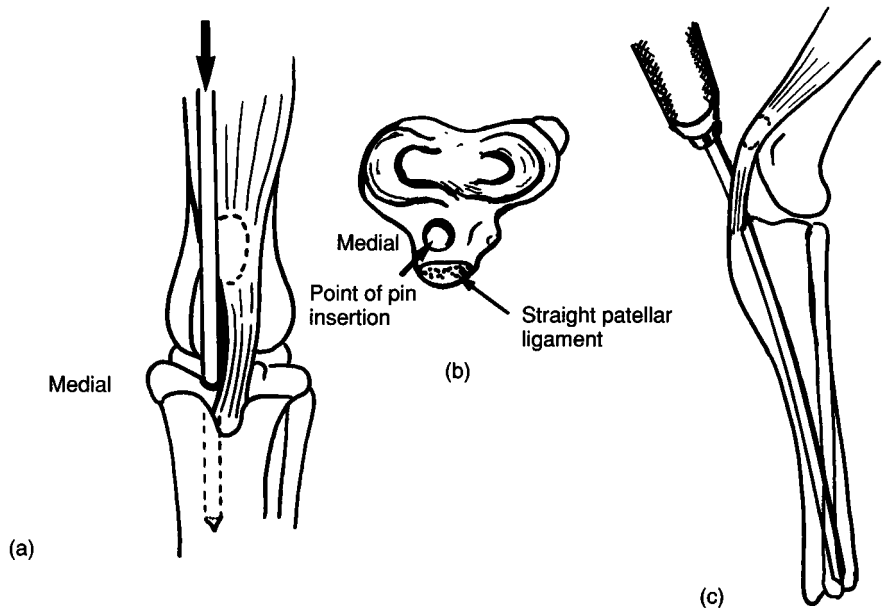

Fig. 43.14 (a) Cranial, (b) dorsal and (c) lateral views of the tibia to illustrate correct position for normograde placement of a tibial intramedullary (IM) pin.

that the hock joint will be entered. After application of any remaining cerclage wires the pin is broken or cut off as discussed above.

Closure is routine with the addition of a single suture in the skin at the site of pin placement.

Postoperative considerations

As a result of surgical intervention being minimal and the fracture often being relatively simple, implying low-grade soft tissue injury, there is often no need to apply a postoperative dressing, especially in smaller patients. If one is to be applied then a Robert Jones bandage for 3–7 days would be appropriate. Exercise restriction should be implemented until radiographic healing of the fracture is apparent, usually 4–8 weeks depending on the nature of the fracture and age of the patient. The need to remove implants is a controversial issue. Generally, it may be preferable to remove the pin from patients that are still growing in case it becomes totally enclosed within the bone as the latter grows, making removal very difficult if problems become apparent. Otherwise the pin is left *in situ* unless it causes problems by protruding too far into the stifle or becoming loose. In most cases cerclage wires are left in place.

External skeletal fixation

External skeletal fixators (ESFs), applied to the tibia, can be designed to protect the fracture site against some or all of the forces which result from weight-bearing. As a result they may be used to treat virtually any fracture of the tibia/fibula either alone or in combination with other implants. There are virtually no contraindications to their use but other methods may be chosen because they are adequate for fracture fixation or achieve equal stability to an ESF and involve less expense in terms of application and/or postoperative management. They are most commonly used as the primary method of stabilisation in the following situations (Fig. 43.15):

- Minimally displaced or stable fractures, particularly in skeletally immature patients, when an external cast might be insufficient or difficult to maintain
- To protect implants used to create compression at the fracture surfaces, e.g. cerclage wires

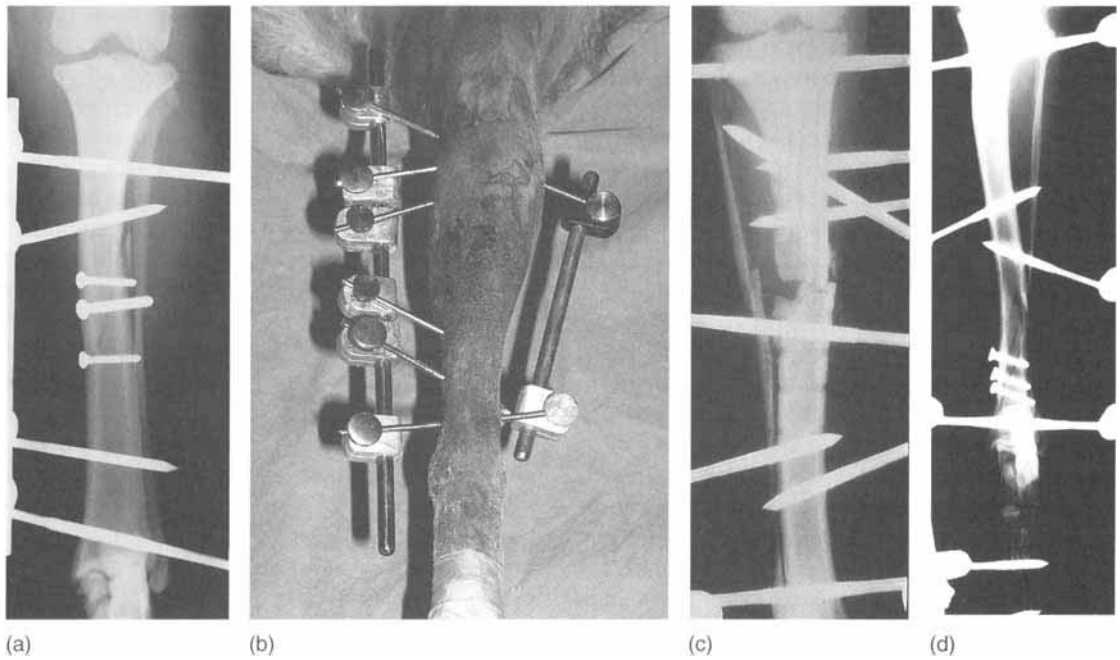

Fig. 43.15 (a) Craniocaudal radiograph of an 18-month-old English Bull Terrier's tibia showing the use of a bilateral, uniplanar fixator to protect an oblique, mid-diaphyseal fracture that has been reconstructed using three lagged bone screws. (b) Craniocaudal view of a 6-year-old Lurcher's tibia after the closed application of a bilateral, uniplanar fixator to stabilise a grade 1 open, comminuted fracture. (c) Craniocaudal radiograph of a 5-year-old German Shepherd Dog's tibia showing stabilisation of a comminuted, mid-diaphyseal fracture using a 'bridging' bilateral, uniplanar fixator. A cancellous bone graft was packed into the fracture gap. At the time of application the fracture was just over 2 weeks old and application of a bone plate to stabilise the fracture had failed on two occasions. Healing was satisfactory, allowing removal of the lateral connecting bar after 6 weeks and removal of the remaining implants after a further 2 weeks. (d) Craniocaudal radiograph of a 2-year-old Lurcher's tibia showing reconstruction of a spiralling fracture of the distal diaphysis using lag screws. These have been protected by application of a bilateral, uniplanar fixator but because of lack of purchase distally, the frame has been extended distally to create a transarticular component.

or lagged bone screws (Fig. 43.15a), especially in chondrodystrophic breeds where the medullary canal is not straight, making the use of an IM pin inappropriate, and the contour of the bone's surface would make contouring of a plate difficult

- To stabilise an open fracture, where avoiding further trauma at the fracture site would minimise iatrogenic enhancement of the contamination and the lack of implants at the fracture site would make control of the contamination easier (Fig. 43.15b)
- In severely comminuted, non-reconstructible fractures an ESF can be used to 'buttress' the fracture site and allow 'biological healing' with the opportunity to gradually 'disassemble' the frame as healing progresses, thus exposing the callus to increasing stresses, which, within certain limits, is believed to accelerate the rate of healing (Fig. 43.15c)
- In very proximal or distal, comminuted, non-reconstructible diaphyseal fractures where the option of bridging the adjacent joint with the frame can be utilised when there is insufficient

bone stock adjacent to the joint to allow adequate stabilisation to be achieved using other methods (Fig. 43.15d)

In addition, an ESF can be used to provide ancillary stability for the primary method of fixation, for example:

- To provide rotational stability in axially stable fractures where an IM pin is being used for angular stability (Fig. 43.13a)
- To provide rotational and axial stability in non-reconstructible fractures where an IM pin is being used for angular stability (since placement of sufficient fixation pins is difficult in the presence of an IM pin, stabilisation of such fractures often involves application of a bilateral external fixator on its own)

Surgical technique

The appropriate choice of implants and technique in application of an ESF to the tibia is described in Chapter 12, p. 97. The application of two half-pins will create a unilateral, uniplanar (type I) fixator that is adequate to control rotational forces around an IM pin, whereas four to six half-pins would be sufficient to stabilise relatively simple fractures either alone or in combination with cerclage wires or lagged bone screws. Full pins, used to create a bilateral, uniplanar (type II) frame, are generally only required when there is axial instability due to comminution where fragments have not been or cannot be reconstructed. Bilateral, biplanar (type III) frames are rarely required in cases treated in the UK. They are most often used in situations where much bone stock has been lost and the fracture is open, i.e. where healing is expected to be slow, and this situation is most commonly associated with gunshot injuries. The frame configurations most often used in managing tibial fractures are illustrated in Figs 43.13a and 43.15.

Postoperative management

The postoperative considerations for tibial ESFs are the same as those applied to other long bones and these are discussed in Chapter 12, p. 106.

Once fracture healing is adequate the ESF may be 'disassembled' or removed completely. Where it is being used to provide only rotational stability this may be possible after only 2 or 3 weeks. In many cases where an ESF has been used to provide most or all the stability, it is possible to detect strong clinical union, by manipulation after removal of the connecting bar(s), before radiographic union is complete. If there is any doubt then there is no harm in simply 'disassembling' or 'staging down' the ESF by reducing the number of connecting bars, reducing the number of fixation pins or moving a connecting bar further away from the bone. It is wise to restrict the patient's exercise until radiographic union is complete or until 3–4 weeks after frame removal. The removal of any adjunctive IM pins has already been discussed and any cerclage wires or lagged bone screws, used to reduce the fracture fragments, are generally left *in situ*.

Plates and screws

Bone plates and screws can be used to protect the fracture site against some or all of the forces caused by weight-bearing. As a result they could be used to treat virtually any fracture of the tibia/fibula but are most suited to the following situations (Fig. 43.16):

- In medium to large breed, skeletally mature dogs with transverse or short oblique fractures when the plate can be used to create compression at the fracture site (Fig. 43.16a)
- In medium to large breed dogs with oblique or reconstructible, comminuted fractures where interfragmentary compression can be created with lagged bone screws and these can be protected from adverse weight-bearing forces by application of a plate in a neutralisation role (Fig. 43.16b)
- In any size of patient with a non-reconstructible, comminuted fracture where the plate can be applied in a 'buttress' fashion, bridging the fracture site and protecting it from all weight-bearing forces whilst biological healing takes place (Fig. 43.16c)
- In some cases with fractures of the proximal or distal diaphysis where specially designed

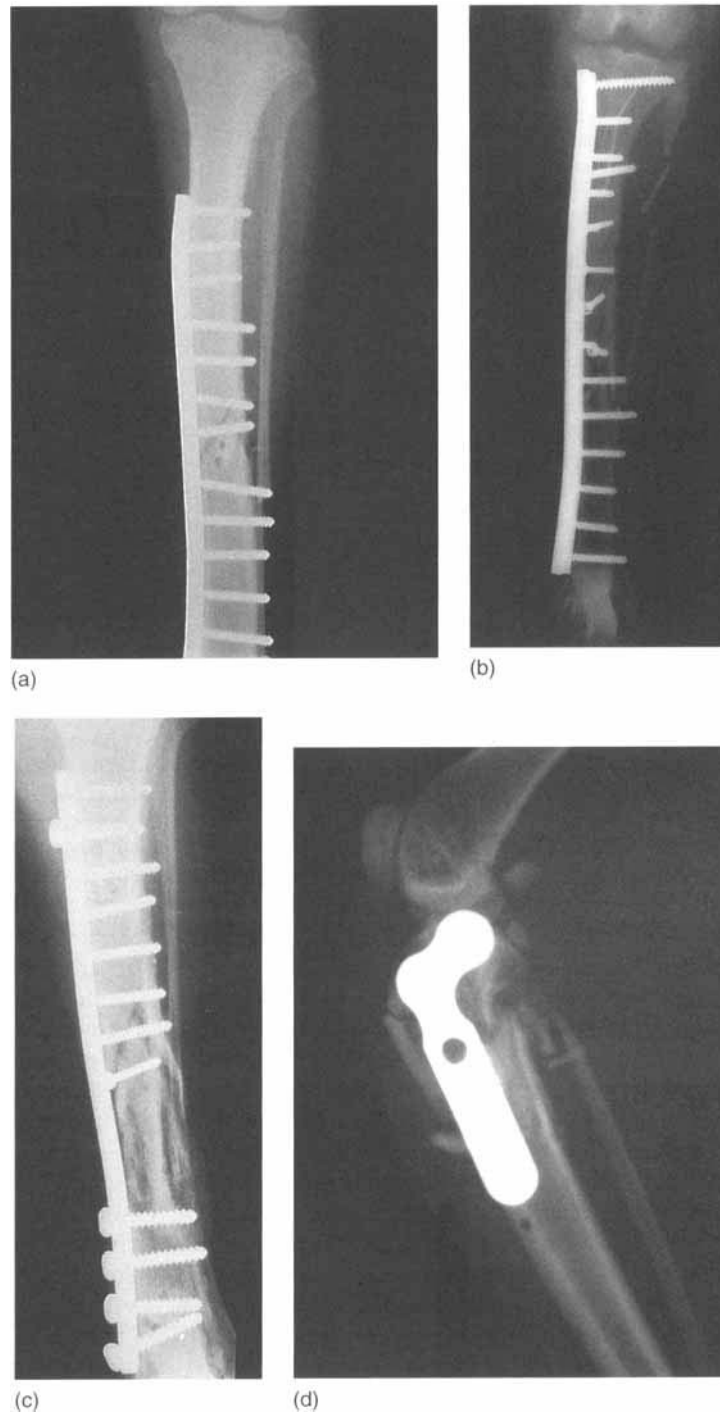

Fig. 43.16 (a) Craniocaudal radiograph of a 1-year-old Great Dane's tibia after stabilisation of a short, oblique, mid-diaphyseal fracture using a compression plate. After compression of the fragments an unsuccessful attempt was made to place a lag screw across the fracture line. (Source: BSAVA Publications, Cheltenham.) (b) Craniocaudal radiograph of an 8-year-old German Shepherd Dog × Rough Collie's tibia after stabilisation of a comminuted, mid-diaphyseal fracture using a number of lag screws which have then been protected by application of a dynamic compression plate (DCP) as a neutralisation plate. (c) Craniocaudal radiograph of a 10-year-old German Short-Haired Pointer's tibia 4 months after a DCP had been applied as a buttress plate to bridge a severely comminuted, mid-diaphyseal fracture.
(d) Mediolateral radiograph of a 12-year-old cat's tibia showing an oblique L-plate being used to buttress the tibial plateau in the treatment of a comminuted, proximal diaphyseal fracture.

plates, e.g. T-plates, might allow adequate stability in a situation with low bone stock and avoid the need for alternatives such as a transarticular ESF (Fig. 43.16d)

The only contraindications to their use are situations where alternative methods might provide perfectly adequate stability and should be chosen because they avoid unnecessary iatrogenic soft tissue damage, expense of implants and possibly the leaving of large metal implants *in situ*. In situations other than those listed above, the application of a plate might be considered 'overkill'. In non-reconstructible, comminuted or open fractures the alternatives of applying an ESF or interlocking nail exist and the choice between these options may come down to surgeon's preference. The authors would tend to favour the use of bone plates, with minimal fragmentary interference (the so-called 'open but do not touch' approach), in the severely comminuted fractures and ESFs in cases with open fractures or in those patients with tibial conformation that makes plate application difficult.

Surgical technique

The choice of implants is generally governed by the appropriate size of bone screws used as plate screws and this is discussed in Chapter 12, p. 123. Bone plates are generally applied to the medial aspect of the tibia and exposure of the bone is relatively straightforward (Fig. 43.10). Common mistakes in plate application include not exposing the tibia proximally enough, where it is easy to believe the exposure must be close to the stifle when there is still one-third of the tibia proximally, especially in obese patients, and not extending the plate distally enough for fear of compromising the tarsocrural joint. Proximal exposure is assisted by use of the groin sling and having an assistant to apply traction to the limb. If the bone stock is poor proximally then T- or L-plates may enable adequate screw 'grouping' and thus implant purchase (Fig. 43.16d), although these tend to be available in only limited lengths and are often inadequate in comminuted fractures. As long as the plate does not extend beyond the origin of the medial collateral ligament distally and the distal-most screw is angled slightly proximally, then there is little chance of interfering with hock joint function.

Wherever possible fragments should be assembled and stabilised with lagged bone screws, or cerclage wires, until only a two-piece fracture remains or else the shaft of bone has been completely reconstructed using only these implants, although this is often not possible in comminuted fractures without one of the lagged bone screws interfering with plate application. After careful contouring of the plate, it is applied to the medial aspect of the tibial diaphysis to act as either a *compression* or a *neutralisation* plate, depending on whether interfragmentary compression has already been achieved. In some cases lagged bone screws will be placed through the plate holes instead of, or as well as, in a different plane to the plate screws. In cases where the plate is being applied in a buttress fashion, the fragments are not reconstructed anatomically and the required contouring of the plate may have to be assessed from radiographs of the contralateral limb or a bone specimen from a similar sized patient.

Postoperative considerations

In most cases it is preferable to apply a Robert Jones bandage for 3–7 days. Exercise restriction should be implemented until radiographic healing of the fracture is apparent, usually 4–8 weeks depending on the nature of the fracture and age of the patient. The need to remove implants is a controversial issue. Generally, the authors prefer to leave the implants *in situ* unless they cause problems. The most common reasons for removing the plate are caused by lack of soft tissue cover in this region. The subcuticular implant may cause irritation, leading to lick granulomas (Fig. 43.17), or lameness due to cooling in low environmental temperatures leading to differential shortening of the plate and bone causing stresses within the bone and pain (so-called 'cold' or 'thermal' lameness). If any such problems are noted then the implants are removed. Following removal of a plate, a Robert Jones dressing should be applied for 7–10 days and the patient rested for about 6 weeks whilst bone remodelling accommodates for any 'stress protection' afforded by the plate.

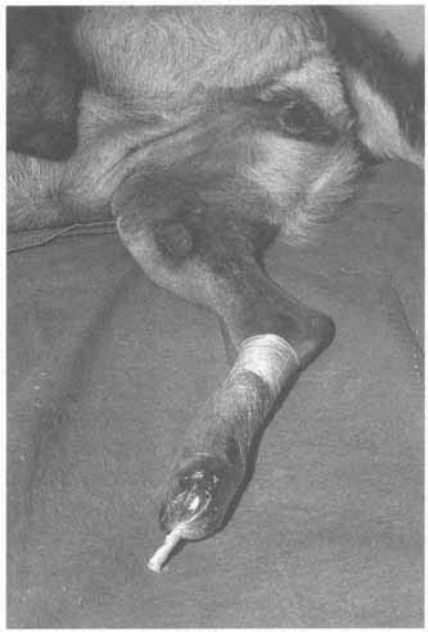

Fig. 43.17 Slide showing the medial aspect of a 7-year-old German Shepherd Dog's tibia 5 months after a plate had been applied to stabilise a comminuted fracture. The lick granuloma present was resected at the same time as the plate was removed. Healing was uneventful. (Source: BSAVA Publications, Cheltenham.)

Fractures of the distal tibia and fibula

In skeletally immature patients the 'weak points' in this region are the distal physes, whereas in the older patient it is more likely that trauma will result in avulsion of the medial and/or lateral malleolus which are the points of origin for the tarsocrural collateral ligaments (see Chapter 44, p. 585). The importance of these fractures revolves around their influence on tarsocrural joint alignment and stability.

Distal physeal separation

This injury is seen in skeletally immature patients and most often results from a medially directed blow to the lateral aspect of the distal crus which causes medial displacement of the distal tibial metaphysis and valgus deformity of the pes (Fig. 43.18a). Abrasions may be present or the distal tibial metaphysis might have actually broken through the skin.

Management

Reduction is of paramount importance to regain limb alignment. If closed reduction is possible, and the fracture then feels relatively stable, conservative management may be employed with casting of the limb as far proximal as the stifle. If reduction cannot be achieved, which is likely, or the site is considered unstable, then open reduction and internal fixation is mandatory. The latter usually involves cross-pinning from the medial and lateral malleoli (Fig. 43.18b). Alternatively an IM pin may be used, although this gains very little purchase in the distal epiphysis and if it is passed across the tarsocrural joint to improve security it restricts articular function.

Surgical technique

The dog is placed in lateral recumbency with the affected limb down and the contralateral limb drawn cranially. Free limb draping is necessary for satisfactory access to both aspects of the hock. A medial approach is made to the distal tibia and reduction is achieved by toggling the fragments. This may be assisted by use of a Hohmann retractor as a lever. Once reduced the fracture will remain stable as long as the foot is not allowed to displace laterally. One or two Kirschner wires or small Steinmann pins are then placed diagonally, at an angle of about 30–40° to the longitudinal axis, through the medial malleolus and distal tibia. After placement of *each* pin, movement of the tarsocrural joint should be checked so that if an implant has compromised joint function it can be removed and relocated. The ends should then be bent over to try and prevent migration. Whether the pins are placed through the transcortex or the Rush pin principle is used is a matter of personal preference. Although theoretically the Rush pin principle is superior, in practical terms crossed pins are easier to apply and produce satisfactory results. Increased stability is then achieved by suturing torn soft tissues and placement of a Kirschner wire, in a similar fashion, through a key-hole incision over the lateral malleolus.

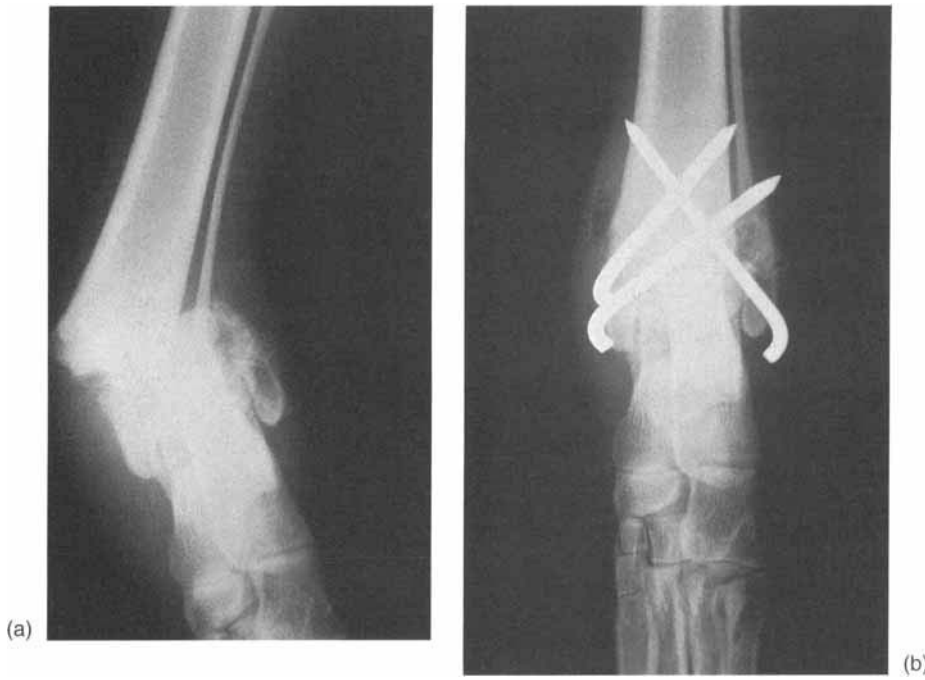

Fig. 43.18 (a) Craniocaudal radiograph of a 5-month-old Dobermann Pinscher's tibia showing fracture–separation of the distal physis with lateral displacement of the pes. (Source: BSAVA Publications, Cheltenham.) (b) After open reduction of the fracture, stabilisation was achieved using crossed Kirschner wires.

Postoperatively the repair may need protection with a cast but if adequate stability has been achieved then it should be possible to avoid casting and allow early return to controlled joint function. In general, the application of a Robert Jones bandage for 2 weeks, when the skin sutures may also be removed, is sufficient as long as the patient's exercise is restricted to cage/room rest and short lead walks for 6 weeks after surgery.

Fracture of the lateral and/or medial malleolus See Chapter 44, p. 585

Pes valgus or varus

Asynchronous growth of the distal tibial/fibular physes results in hindlimb deformity but is an uncommon clinical presentation. Retarded growth laterally will result in outward turning of the foot (pes valgus) whilst reduced growth medially will cause inward turning of the foot (pes varus). Although traumatic injury might influence physeal activity, valgus or varus deformity in the hindlimb is seen far less often after, say, tibial fractures than it is seen in the forelimb after antebrachial fractures. The reason for this may be the anatomical shape of the distal ulnar physis which predisposes it to injury (see Chapter 34). In fact, most cases do not have a history of previous trauma and so the conditions may then be considered truly developmental and, accordingly, they appear to show breed predispositions. Pes valgus is most often seen in Rough Collies and Shetland Sheepdogs whilst pes varus is overrepresented in the Miniature Dachshund and, possibly, the giant breeds such as Mastiffs.

Clinical signs

The main clinical sign is the deformity itself +/– a degree of lameness caused by a mechanical influence of the deformity on gait and/or pain

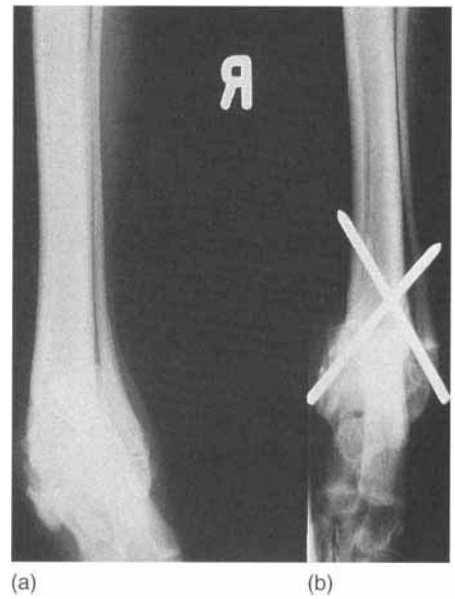

Fig. 43.19 (a) Craniocaudal radiograph of a 7-month-old Rough Collie's tibia showing bowing of the distal crus (pes valgus) due to asynchronous closure of the distal tibial physis.
(b) Postoperative radiograph after a closed medial wedge osteotomy and fixation using crossed Kirschner wires. The joint was cast for six weeks, healing was uneventful and the wires subsequently migrated out through the skin. The contralateral limb developed a similar problem and was treated successfully in the same way.

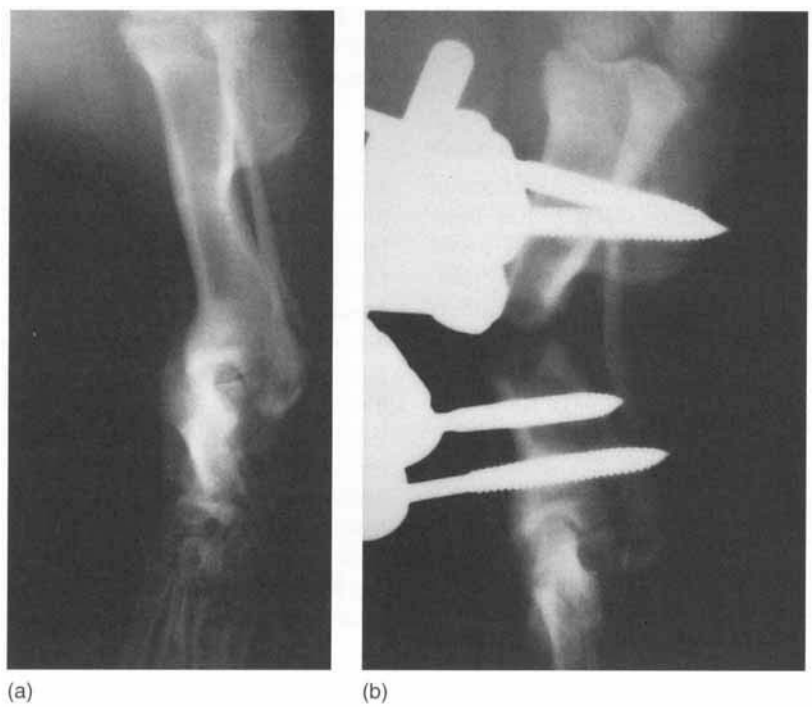

Fig. 43.20 (a) Craniocaudal radiograph of a 6-month-old Miniature Dachshund's tibia showing bowing of the distal crus (pes varus) due to asynchronous closure of the distal tibial physis.
(b) Postoperative radiograph after an open medial wedge osteotomy and stabilisation using a unilateral, uniplanar fixator. Healing was uneventful and the fixator was removed after 6 weeks.

through increased stresses through the tarsocrural joint. The problem may be, or become, bilateral, though the severity in each limb may not be the same.

Diagnosis

This is generally made on the clinical appearance but radiography is required to show that the deformity arises from the distal tibia (Figs 43.19a and 43.20a). In the giant breeds the deformity tends to arise from a general bowing of the tibia rather than angulation at a specific site and so radiography can prove more frustrating in terms of confirming the diagnosis.

Management

Although techniques such as transphyseal stapling or periosteal elevation may be considered, these are technically more difficult and less successful than when used in the distal antebrachial deformities. Furthermore, patients with distal crural deformity tend to present at an age when techniques such as transphyseal stapling would be inappropriate because insufficient growth potential remains for 'self-correction'. As a result, most cases are treated by corrective osteotomy. In the case of pes valgus an oblique or closing wedge osteotomy is used to allow realignment. The site is then stabilised using either crossed pins (Fig. 43.19b), with external casting to protect the repair, a bone plate or an external fixator (McCarthy, 1998). In the case of pes varus an open wedge osteotomy may be used (possibly leaving the fibula intact) so as not to lose limb length. Stabilisation may then be achieved using one of several methods including plate or external skeletal fixation (Fig. 43.20b) (Johnson *et al.*, 1989).

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A Guide to Canine and Feline Orthopaedic Surgery
Hamish R. Denny,Steven J. Butterworth

Chapter 44
The Tarsus

Anatomy

The tarsus, or hock, is a composite joint consisting of seven tarsal bones and their related soft tissues. The tarsal bones are arranged in three irregular rows (Fig. 44.1). The specific joints in the tarsus are:

- The tarsocrural joint
- The proximal intertarsal joint
- The centrodistal joint (distal intertarsal joint)
- The tarsometatarsal joint

The tarsocrural joint

This is a ginglymus joint formed between the tibia, fibula and talus (tibial tarsal bone). This joint is responsible for most of the movement which occurs in the hock as a whole. The range of movement in a dorsoplantar direction is limited by the hock flexors and extensors, as well as the dorsal and plantar ligaments within the joint capsule, whilst mediolateral stability is provided primarily by the collateral ligaments (Fig. 44.2). The medial collateral ligament arises from the medial malleolus and has long and short components. The long component inserts on the first tarsal bone and, to a lesser extent, on the first and second metatarsal bones. Part of the short component inserts on the talus whilst the other part runs parallel to the long component and has a similar insertion. Medial support is also provided by tendons of the long digital flexor, hallucis longus and caudal tibial muscles, which cross this area in close association with the joint capsule. On the lateral side of the joint the collateral ligament arises on the lateral malleolus and again has a long and short component. The long component inserts on the head of the fifth metatarsal bone after attaching to both the calcaneus and fourth tarsal bone. The short component also divides into two bands, one inserts on the calcaneus whilst the other passes more dorsally to insert on the talus. Additional lateral support is provided by tendons which lie in close association with the joint capsule. These are the tendons of the peroneus longus, peroneus brevis and lateral digital extensor muscles. On both sides of the joint, the long component of the collateral ligament is responsible for stability when the hock is extended whilst the short component affords stability when it is flexed, and this is of significance when stability is assessed clinically.

The *common calcaneal tendon* (Achilles tendon) inserts on the tuber calcaneus. It consists of the gastrocnemius tendon and the common tendon of the gracilis, biceps femoris and semitendinosus (Evans, 1993). The superficial digital flexor tendon (SDFT) passes distally over the tuber calcaneus. A bursa is interposed between the tendon and the calcaneus.

The intertarsal joints and tarsometatarsal joints listed below are arthrodia and, normally, movement between them is minimal.

The proximal intertarsal joint

This consists of the calcaneoquartal and talocalcaneocentral joints (Fig. 44.1). The *calcaneoquartal joint* is the articulation between the calcaneus and the fourth tarsal bone. The *talocalcaneocentral joint* is essentially the articulation between the talus and central tarsal bone, however, the joint capsule is continuous with the calcaneus.

Plantar support for the proximal intertarsal joint (PITJ) is provided by ligaments (Fig. 44.2)

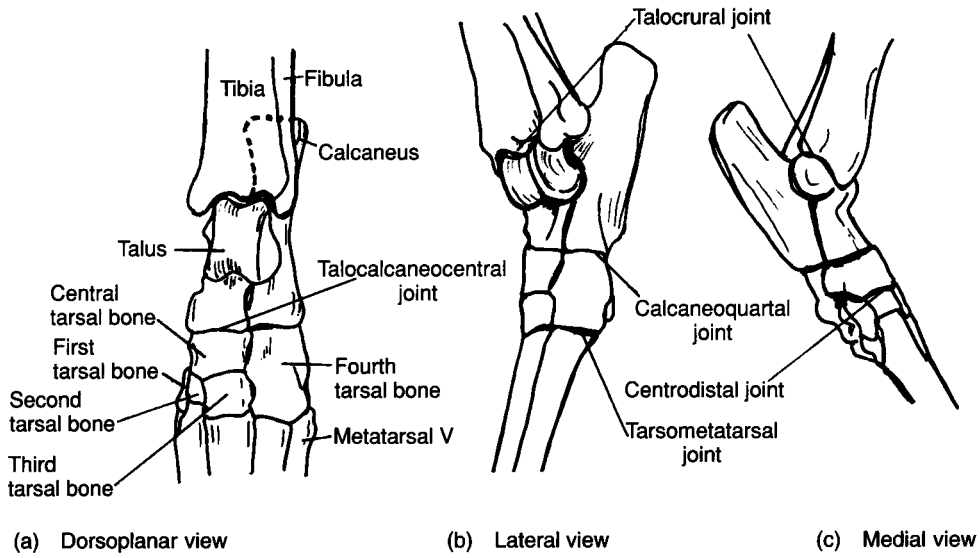

Fig. 44.1 Anatomy, bones and joints of the left tarsus.

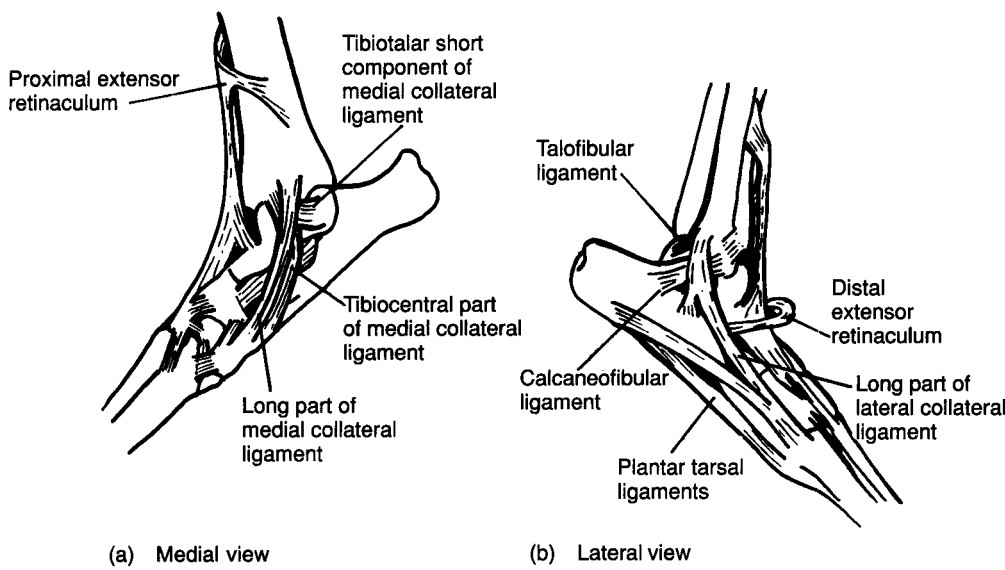

Fig. 44.2 Anatomy and ligaments of the right tarsus.

arising from the plantar aspect of the calcaneus and sustentaculum and inserting on the same aspect of the fourth and central tarsal bones, respectively, before going on to form the tarsal fibrocartilage and inserting on the heads of the metatarsi. There is also a ligament arising from the plantarolateral aspect of the calcaneus and inserting on the head of metatarsal V. This probably affords some plantar, and possibly lateral, support. Lateral stability is maintained by the long part of the lateral collateral ligament, arising from the lateral malleolus and inserting on the head

of the fifth metatarsal. On the medial aspect, support is provided by the long and short parts of the medial collateral ligament which arise from the medial malleolus and insert, at least in part, on the first tarsal and, to a lesser extent, the first and second metatarsi. Dorsal ligaments are also present but are of little clinical significance and, if damaged, will heal without assistance as they lie on the compression side of the joint and thus normal weight-bearing will not disrupt them.

The centrodistal joint (distal intertarsal joint)

This is the joint between the central tarsal bone and the distal numbered tarsal bones.

The tarsometatarsal joint

This comprises the joints between the distal tarsal and the metatarsal bones. The stability of this joint is dependent on its collateral and plantar ligaments (Fig. 44.2). Dorsal ligaments are also present but are of minor importance. The lateral collateral ligaments arise from the lateral malleolus and plantarolateral aspect of the calcaneus and insert on the head of the fifth metatarsal bone. The medial collateral ligaments arise from the medial malleolus, the central tarsal and the second tarsal bones, and insert on the head of the second metatarsal bone. Plantar support is provided by the calcaneal and sustentacular plantar ligaments arising from the proximal row of tarsal bones and inserting on the heads of the metatarsi, via the tarsal fibrocartilage, having attached to the plantar aspects of the fourth and central tarsal bones respectively.

Conditions of the hock

Conditions of the hock can be divided into three groups:

- *Developmental*
  - Osteochondrosis
  - Tarsal bone anomalies often causing outward turning of foot and/or 'cow hocks'
- *Traumatic*
  - Calcaneus (fracture)
  - Talus (fracture of condyle or neck; luxation of head)
  - Central tarsal bone (fractures; luxation)
  - Numbered tarsal bones (fractures; luxations)
  - Tarsocrural instability (including shearing injury)
  - Proximal intertarsal instability
  - Distal intertarsal instability
  - Tarsometatarsal instability
  - Achilles tendon injury
  - Slipped superfical digital flexor tendon
- *Acquired*
  - Failure of plantar ligaments (see under proximal intertarsal and tarsometatarsal instability)
  - Gastrocnemius enthesiopathy (discussed in conjunction with Achilles tendon injury)
  - Osteoarthritis (see Chapter 7)
  - Immune-mediated arthropathy (see Chapter 8)

Osteochondrosis

Osteochondritis dissecans (OCD) of the hock is now a well-recognised cause of hindleg lameness in young, large-breed dogs, especially Labrador Retrievers, Golden Retrievers, Rottweilers, Irish Wolfhounds and Bull Terrier breeds (Olsson, 1975; Mason & Lavelle, 1979; Johnson *et al.*, 1980; Denny, 1981; Beale *et al.*, 1991). The OCD lesion is usually found in the medial trochlear ridge of the talus (see later in Fig. 44.4a). However, lesions are occasionally found in the lateral trochlear ridge (Robins *et al.*, 1983; Wisner *et al.*, 1990).

History and clinical signs

A gradual onset of unilateral or bilateral hindleg lameness occurs at about 5 months of age. Initially, there is a normal range of tarsocrural joint flexion, but as the condition progresses hock conformation appears to become increasingly 'straight' (Fig. 44.3) and the range of joint flexion becomes restricted. Synovial effusion may be

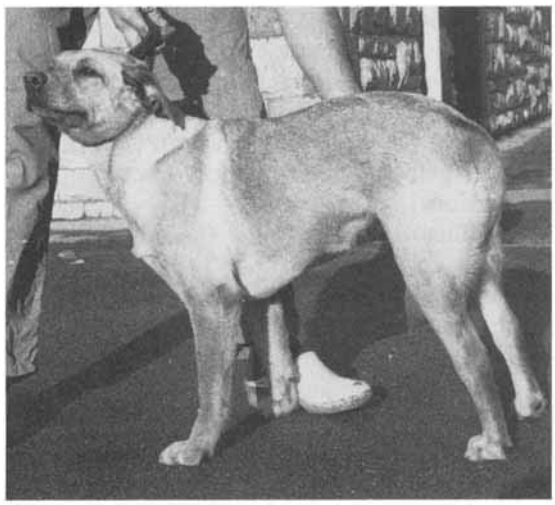

Fig. 44.3 A 7-month-old Labrador with bilateral osteochondritis dissecans (OCD) of the hocks. Notice straight hock conformation.

apparent, resulting in a fluctuating swelling just caudal to the tarsocrural joint. In chronic cases, periarticular osteophytes and fibrosis result in thickening, particularly over the medial side of the hock.

Radiographic findings

Standard mediolateral and dorsoplantar radiographic views are usually sufficient to diagnose OCD lesions in the medial trochlear ridge (Fig. 44.4a). The calcaneus overlies the lateral trochlear ridge and, consequently, an oblique dorsoplantar view or a flexed dorsoplantar skyline view is necessary to demonstrate a lateral OCD lesion.

The radiographic changes are the same whichever side is affected but will be described for lesions affecting the medial trochlear ridge as these are encountered most frequently. The dorsoplantar view usually gives most information (Fig. 44.4b) and changes include:

- An increase in the medial joint space of the tarsocrural joint
- Flattening of the medial trochlear ridge of the talus (also evident on the mediolateral view, Fig. 44.4c
- Mineralised fragment overlying the medial trochlear ridge which may displace and be seen on the caudal aspect of the joint as a 'joint mouse' in the mediolateral view
- Periarticular osteophytes, which develop rapidly

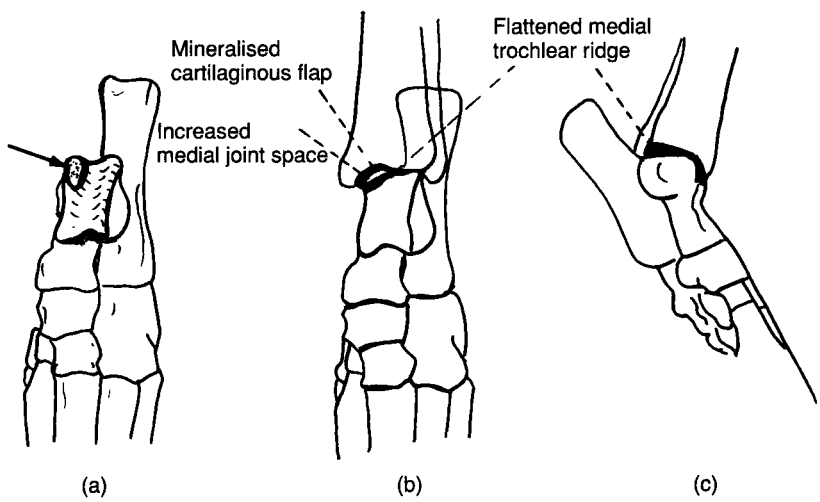

Fig. 44.4 (a) Osteochondritis dissecans (OCD) lesion in the medial trochlear ridge of the talus (arrow). Radiographic features of the hock, (b) dorsoplantar view and (c) mediolateral view.

Management

The general consensus of opinion is that early diagnosis and surgical treatment offers the best prognosis. Treatment involves a plantaromedial arthrotomy, in the case of a medial lesion, to remove loose osteochondral fragments from the tarsocrural joint and allow curettage of the margins of the erosion in the articular cartilage. Excessive curettage is to be avoided since it serves only to remove more of the trochlear ridge which may create more postoperative joint incongruency/instability. Bilateral arthrotomies can be performed during a single surgery. Details of a series of 35 cases treated by one of the authors are given in Table 44.1. Unilateral arthrotomies were carried out in 24 dogs and bilateral arthrotomies in 11. The average follow-up period was 4 years (range: 6 months to 6 years). The outcome of surgery was considered successful in 71% of cases (60% sound, 11% with occasional lameness after exercise). Of the remainder, 20% had slight lameness and 9% moderate to severe lameness. The results of this series indicate that provided surgery is undertaken early, before 9 months of age, and provided there is still a good range of hock flexion at the time of surgery, then there is about a 70% chance of a successful outcome. Nevertheless, progression of secondary osteoarthritis (OA) is not arrested by surgery, but this is not necessarily associated with lameness. Surgical treatment in chronic cases, with established tarsocrural OA, is not generally recommended although in those which become refractory to treatment with non-steroidal anti-inflammatory drugs (NSAIDs), surgical removal of any loose fragments within the joint will often make the dog more comfortable. In extreme cases, tarsocrural arthrodesis may be considered as a salvage procedure.

Table 44.1 Osteochondritis dissecans (OCD) of the medial trochlear ridge of the talus in 35 cases[a,b,c] recorded between 1987 and 1994.

Breeds affected:	No.	
Labrador Retriever	21	
Rottweiler	8	
Golden Retriever	4	
English Bull Terrier	1	
Bull Mastiff	1	
Results of surgical treatment:	No.	(%)
Sound	21	(60)
Occasional lameness	4	(11)
Slight, persistent lameness	7	(20)
Moderate to severe, persistent lameness	3	(9)
Total	35	

[a] Sex ratio = 15 males : 20 females.
[b] Uni- vs bilateral arthrotomy = 24 unilateral lesions : 11 bilateral lesions.
[c] Average follow-up period of 4 years (range: 6 months to 6 years).

Plantaromedial arthrotomy

The OCD lesion is exposed by a plantaromedial approach to the tarsocrural joint (Fig. 44.5a). Because a tourniquet tends to restrict hock flexion, which is required to expose the OCD lesion, many surgeons prefer not to use one for this procedure. However, a Vetrap (3M) tourniquet (as described under 'Exposure of the carpus' in Chapter 35, p. 414) can be used to good effect during this procedure. A curved skin incision is made just caudal to the medial malleolus of the tibia. The joint capsule is usually distended and thickened and is incised close to the caudal border of the tibia. Care should be taken to avoid the flexor hallucis longus tendon, the tibial nerve and plantar branches of the saphenous artery and vein which lie caudally (Fig. 44.5b). The joint is fully flexed to reveal the trochlea of the talus. Additional exposure can be achieved by cutting the joint capsule transversely towards the medial collateral ligament. The cartilaginous flap is easily identified on the medial trochlear ridge and once the flap has been removed the edges of the erosion in the subchondral bone are curetted (see above). The joint capsule is closed with simple interrupted sutures of polyglactin 910, poliglecaprone 25 or polydioxanone (Vicryl, Monocryl or PDS, Ethicon). The remainder of the closure is routine. A support bandage is applied. Surgery is then carried out on the other hock if the dog has bilateral, clinically active lesions. The support bandage is removed after 5 days and exercise is restricted to short walks on a leash only for 6 weeks.

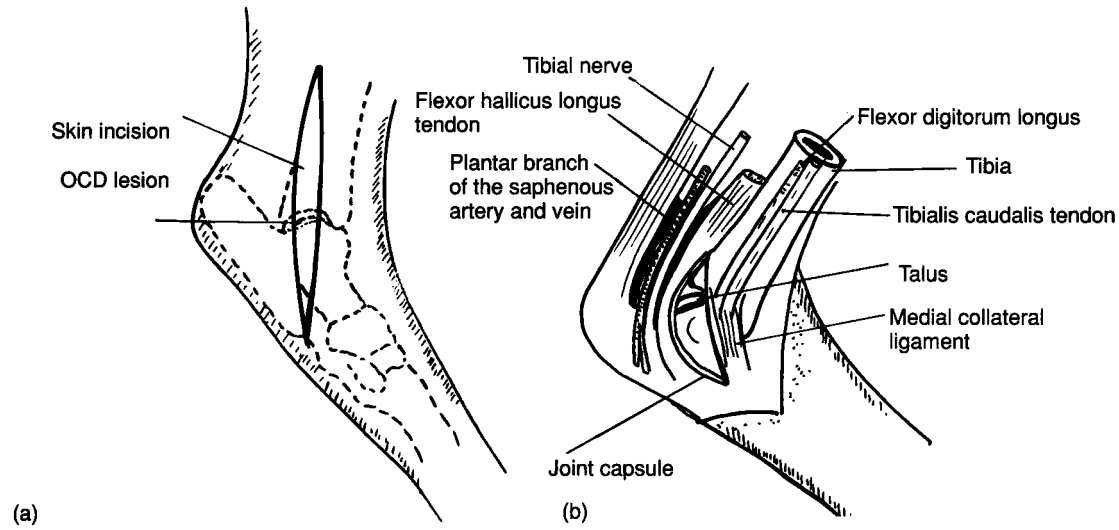

Fig. 44.5 Plantaromedial arthrotomy for removal of osteochondritis dissecans (OCD) lesions. (a) Skin incision. (b) Arthrotomy.

Tarsal bone anomalies

In the Pyrenean Mountain Dog, St Bernard and Rottweiler, an additional bony prominence may be seen on the medial aspect of the central tarsal bone (Vaughan, 1987). This is a congenital malformation and is of no clinical significance.

Poor conformation through the hock may cause outward turning of foot and/or 'cow hocks', and is seen in large breeds of dog such as the St Bernard, Rottweiler and Bernese Mountain Dog (Bennett, 1990). Lameness is not usually a problem and owners are more concerned about the cosmetic appearance of the hock. Radiographs will show tarsal bone abnormalities with respect to shape, fusion and alignment. There is no treatment.

Fractures of the calcaneus

In calcaneal fractures the proximal fragment is distracted by the gastrocnemius tendon resulting in hyperflexion of the hock and a plantigrade stance. Four types of fracture are recognised:

(1) *Salter Harris type 1 or 2 fracture* involving the proximal calcaneal growth plate. This is an avulsion injury seen in immature animals. The epiphysis is distracted by the gastrocnemius tendon. Kirschner wires and a figure-of-eight tension band wire are used for repair (Fig. 44.6).

(2) *Mid-body fractures*. These are a common injury in the racing Greyhound and it is

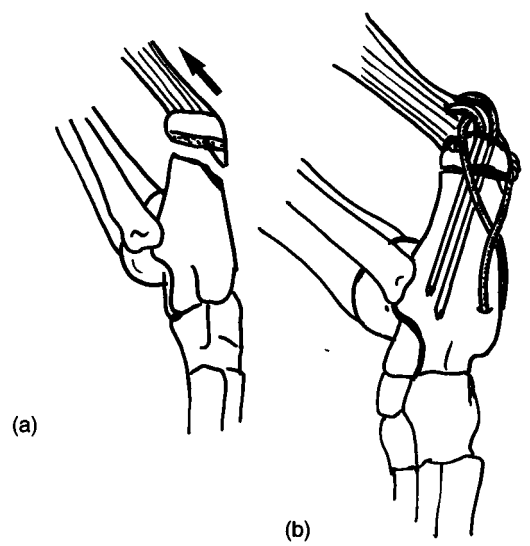

Fig. 44.6 (a) Salter Harris type 2 fracture of the proximal calcaneal growth plate. (b) Fixation with Kirschner wires and a tension band wire.

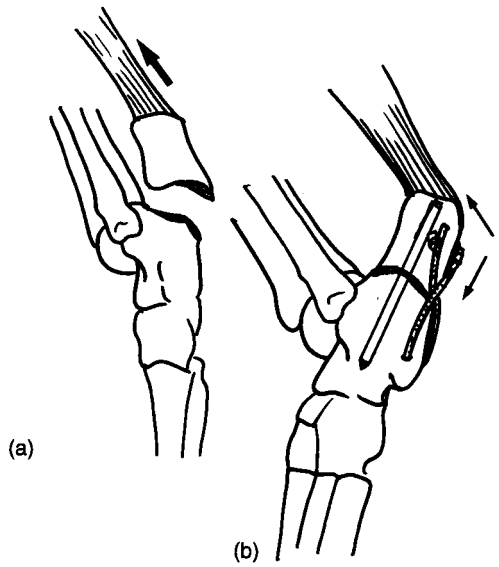

Fig. 44.7 (a) Mid-body fracture of the calcaneus. (b) Fixation with intramedullary (IM) pin (countersunk) and tension band wire.

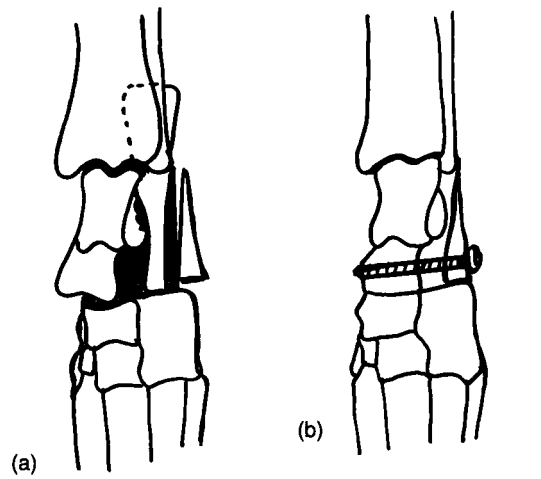

Fig. 44.8 (a) Slab fracture of distolateral calcaneus with luxation of the adjacent talus. (b) Lag screw fixation.

usually the right calcaneus that is fractured. The fracture tends to be associated either with fracture of the central tarsal bone, or subluxation of the proximal intertarsal joint if the central tarsal bone remains intact (Ost *et al.*, 1987). A countersunk intramedullary (IM) pin and tension band wire are used for fixation (Fig. 44.7). In comminuted fractures it may be necessary to use a laterally placed plate for fixation.

(3) *Slab fractures* of the distolateral or dorsomedial calcaneus are often complicated by other injuries, for example luxation of the adjacent talus or fracture of the central tarsal bone. Lag screw fixation is used for repair in most of these injuries (Fig. 44.8). If there is comminution then it may be necessary to combine the lag screws with a pin and tension band wire or plate fixation.

(4) *Fractures of the base.* The plantar ligaments originate on the base of the calcaneus. Avulsion fractures involving this area result in plantar instability and subluxation of the proximal intertarsal joint. Treatment is by arthrodesis (see later).

Prognosis

Prognosis following calcaneal fracture repair is generally good but, in the racing Greyhound, if the repair involves arthrodesis of the proximal intertarsal joint then the animal is unlikely to return to racing form.

Fractures of the talus (tibial tarsal bone)

Fractures of the talus are uncommon (Dee, 1988). They can be divided into two groups:

(1) *Intra-articular fractures*, which involve either the medial or lateral trochlear ridge. Small fragments are removed while larger fragments are fixed *in situ* with countersunk Kirschner wires or absorbable PDS pins (Johnson & Johnson) (Fig. 44.9). Exposure generally requires maleollar osteotomy. The osteotomy is repaired with a lag screw or Kirschner wire used in conjunction with a tension band wire.

(2) *Extra-articular fractures*, which involve the neck, body or base. Closed reduction of

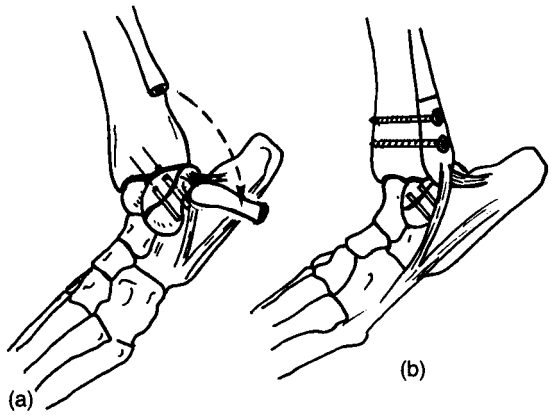

Fig. 44.9 (a) Intra-articular fracture of the lateral trochlear ridge of the talus, exposed by lateral malleolar osteotomy. (b) Fracture fixation with countersunk Kirschner wires. Screw fixation of the malleolar osteotomy.

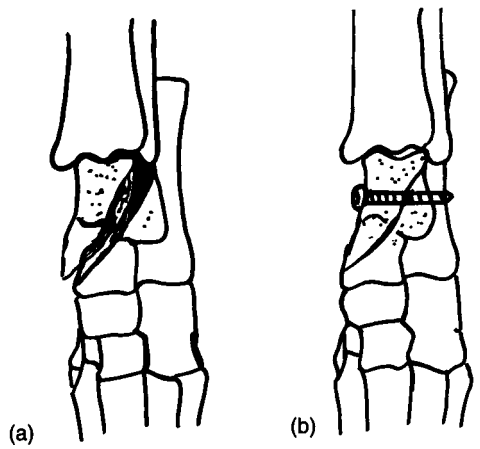

Fig. 44.10 (a) Slab fracture of the body of the talus. (b) Lag screw fixation.

these fractures is sometimes possible in cats and small dogs but generally open reduction and lag screw fixation is required. Fracture of the neck is usually associated with luxation of the body and fixation is achieved by placing a lag screw across the fracture between the body of the talus and the calcaneus (Fig. 44.10). There is an anatomical gap between the two bones known as the tarsal sinus and, consequently, the screw will be subjected to bending forces if early weight-bearing is allowed. Therefore, the repair should be protected with a splint for 4 weeks following surgery (Brinker *et al.*, 1990).

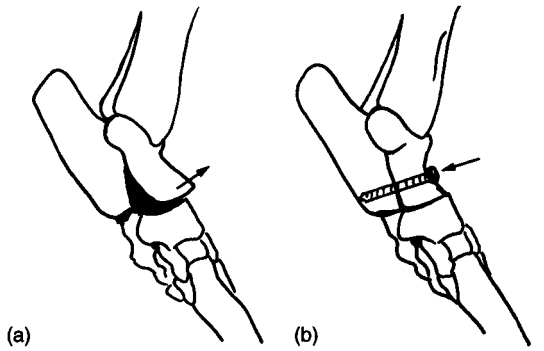

Fig. 44.11 (a) Luxation of the head of the talus. (b) Lag screw fixation.

In small dogs with fractures of the neck of the talus, the fragments may be too small to use internal fixation and, under these circumstances, a transarticular external fixator is used to maintain stability while healing occurs.

Luxation of the head of the talus (tibial tarsal bone)

This is an infrequently seen injury (Fig. 44.11) which causes a severe lameness due to medial collapse of the hock on weight-bearing. Open reduction is necessary and once the dorsally displaced bone has been relocated, internal fixation is required. This most commonly involves placement of a bone screw through the head of the talus and into the calcaneus. In small patients a Kirschner wire may suffice. The implant should be placed as far distally as possible to avoid crossing the tarsal sinus. Postoperatively a splint or cast should be applied for 4 weeks. In some cases, this injury has been associated with damage to the medial collateral ligament of the tarsocrural joint, and so it is as well to check for instability at that level once the more obvious luxation has been reduced. The prognosis in these cases appears to be good, presumably because the joints that have been disrupted are low-motion articulations.

Fractures of the central tarsal bone

Central tarsal bone fractures are a common injury in the racing Greyhound. It is invariably the right

hock that is involved. The reason for this is that the dog runs anticlockwise when racing. During cornering, the medial side of the right hock is under compression, with the central tarsal bone acting as a buttress which may fracture when subjected to extreme stresses. In managing central tarsal bone fractures, the aim is to maintain bone space and prevent collapse of the medial side of the hock. Dorsal and medial slab fractures are seen most often. Most of these are best managed by lag screw fixation. Fusion of the adjacent intertarsal joints is often seen during fracture healing but should not compromise the dog's chances of racing again as there is normally very little movement in these joints.

Treatment

The treatment of central tarsal bone fractures was described by Bateman in 1958. In a more recent publication (Dee *et al.*, 1976), a classification of fractures of the central tarsal bone was presented together with treatment and prognosis. The fractures were grouped into five types (Fig. 44.12):

- *Type 1*: A dorsal slab fracture with no displacement. These fractures are treated by external coaptation.
- *Type 2*: A dorsal slab fracture with dorsal and proximal displacement. These are treated by open reduction and fixation with a single lag screw.
- *Type 3*: A fracture in the sagittal plane with or without displacement of the medial fragment. Treated by open reduction and fixation with a single lag screw.
- *Type 4*: This fracture is a combination of types 2 and 3, in which there is dorsal displacement of a slab in conjunction with a larger medially displaced fragment. The fracture is treated by open reduction and fixation with two lag screws.
- *Type 5*: The central tarsal bone is severely comminuted and displaced. Treatment is by closed reduction and external coaptation.

Prognosis

The prognosis for type 1 and 2 fractures is good; for types 3 and 4 is fair to good; and for type 5 is poor (Boudrieau *et al.*, 1984). External support (a light cast for 4–6 weeks) is provided in all cases treated by internal fixation. Exercise is restricted for 12 weeks after surgery and then training can be gradually resumed.

Luxation of the central tarsal bone

This is an uncommon injury which again produces a severe lameness due to collapse of the medial side of the hock during weight-bearing. Reduction of the dorsomedially displaced bone requires an open approach and stabilisation using a bone screw, or Kirschner wire, passing through the central tarsal bone and into the fourth tarsal bone, in much the same way as in treating central tarsal bone fractures. Attention should be paid to any ligament injuries causing medial instability which could be dealt with at the same time. The joint should be supported for 4 weeks postoperatively. The prognosis in these cases appears to be good, presumably because the joints that have been disrupted are low-motion articulations.

Fractures/luxations of the numbered tarsal bones (T2, T3 and T4)

Fractures of the numbered tarsal bones invariably occur as a complication of central tarsal bone fractures in the racing Greyhound. Fracture of T4 occurs most frequently and is usually a compression fracture. Repair of the central tarsal bone fracture with a lag screw is, generally, the only treatment required (Dee, 1988). Dorsal slab fractures of T3 can occur without involvement of other tarsal bone fractures and these are managed by placing a lag screw in a dorsoplantar direction (Dee, 1988). Lag screw fixation is also used to maintain reduction of fracture luxations of T2 and T3 (Dee *et al.*, 1990).

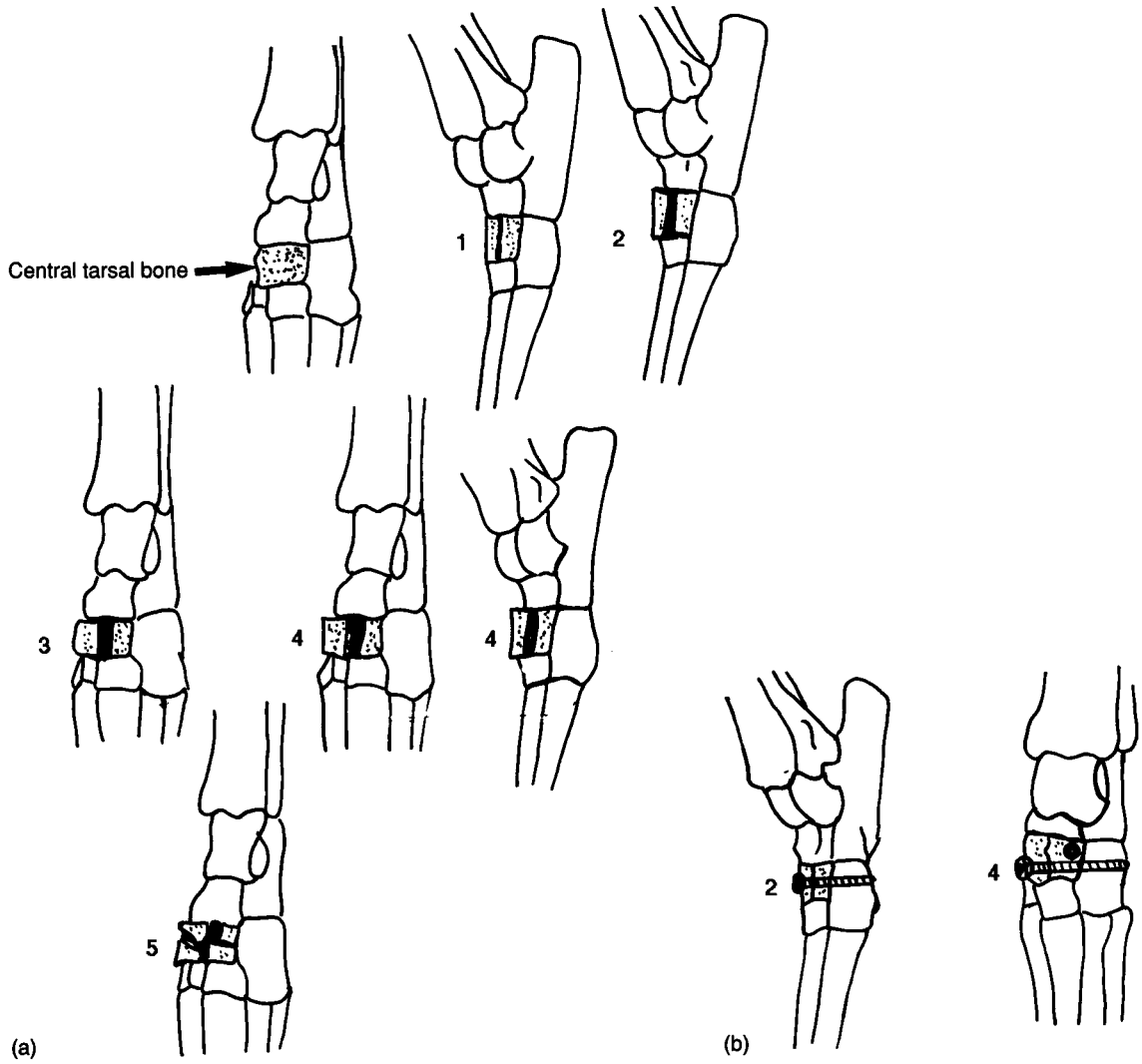

Fig. 44.12 (a) Classification of central tarsal bone fractures into five types. Lateral and dorsoplantar views of the left hock are shown. See text for further details of the fracture types. (b) Lag screw fixation of type 2 and type 4 fractures.

Tarsocrural instability (including shearing injuries and arthrodesis)

Aetiology

Instability of this joint is almost always a result of trauma and involves damage to the lateral and/or medial collateral ligaments with or without concurrent injury to the associated tendons listed above. Injury to the ligaments may involve a mid-substance rupture or else an avulsion fracture of the origin. The cause is usually an awkward fall or placement of the foot down a hole whilst running. Alternatively, the instability may be a result of a scraping injury sustained by involvement in a road traffic accident. It is believed that such injuries are caused by the hock becoming trapped between a wheel and the road and the soft tissues being planed off as the wheel slides along the road. It is most often the lateral side that is affected in such a scenario.

Diagnosis

These cases will present with an acute onset, severe lameness. Pain is noted on manipulation of the hock and soft tissue swelling or injury is seen in the vicinity of the tarsocrural joint. Instability may be detectable in the conscious patient but those cases with severe trauma, and particularly those with loss of soft tissues, might be better assessed at a later stage, under general anaesthesia. It is important to assess stability, by stressing the joint, in both flexion and extension since it is necessary to establish whether one or both components of each collateral ligament is/are injured.

Radiography may be helpful in showing the presence of avulsion fractures and joint space widening due to instability, which might require stressed views to become apparent. It may also show the extent of bone loss in 'shearing' injuries, although this is usually known after clinical examination.

Treatment

Conservative management

Conservative management is indicated in cases with only partial rupture of a collateral ligament or an avulsion fracture of a malleolus which is amenable to closed reduction. The application of a cast for 6–8 weeks may allow satisfactory healing and a return to normal function.

Surgical management

Surgical management is indicated in the following situations:

(1) Where avulsion fractures of the medial and/or lateral malleoli are responsible for the instability and the fragment is considered large enough to accommodate implants. Surgical management may involve reattachment of the fragment using a lagged bone screw (Fig. 44.13) (Holt, 1976) or pin and tension band wire (Fig. 44.14) (Denny, 1975).

(2) If the injury involves complete rupture of the collateral ligament, and it is treated early, it may

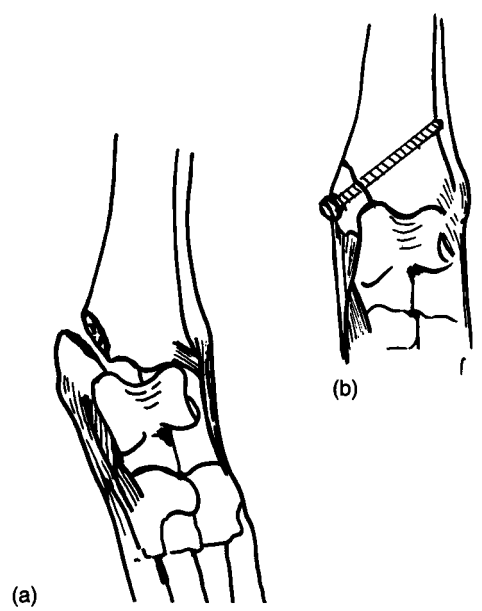

Fig. 44.13 (a) Medial malleolar fracture. (b) Lag screw fixation.

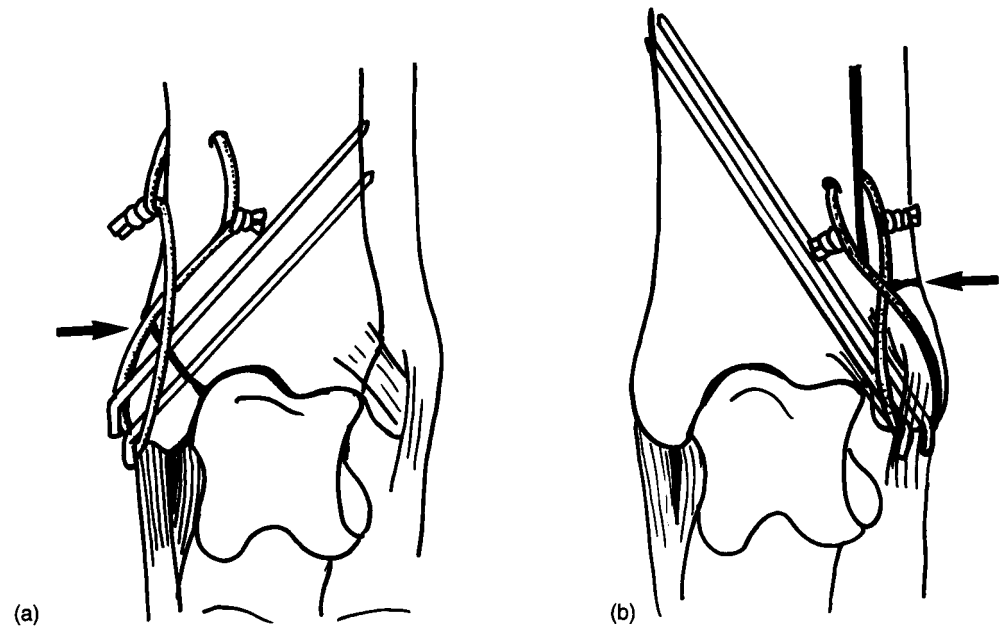

Fig. 44.14 (a) Medial malleolar fracture fixation with Kirschner wires and tension band wire. (b) Lateral malleolar fracture fixation with Kirschner wires and tension band wire.

be possible to suture the two ends together using an appropriate material such as monofilament nylon or polydioxanone (PDS, Ethicon). Injuries to the local tendons should also be evaluated and sutured appropriately. Postoperatively, a cast should be applied to the hock for 6–8 weeks.

(3) If the instability is marked and/or conservative management has failed then it may not be possible to reconstruct the ligaments due to the degree of tearing or fibrosis. In such cases it may be necessary to place prosthetic collateral ligaments of monofilament nylon, braided polyester (Ethibond, Ethicon) or wire. Many techniques have been described to achieve this (Holt, 1976; Holt, 1977), whereby bone tunnels or bone screws have been used to anchor the prosthesis in place. The earlier methods described did not really take into account the collateral ligaments having two components and thus may not have been able to stabilise the joint in both flexion and extension. A more recently developed method (Aron & Purinton, 1985a,b) addresses this problem and is probably the best one to use, particularly in patients that are large enough to allow placement of the

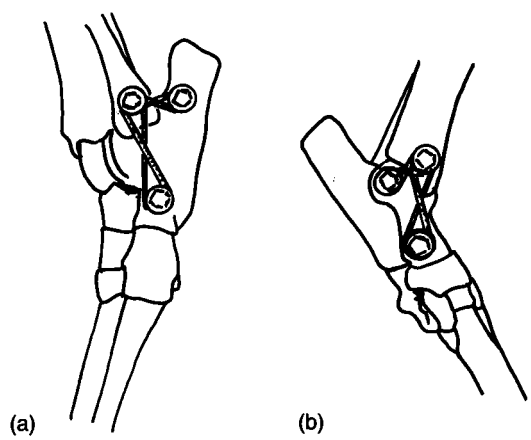

Fig. 44.15 (a) Replacement of the lateral collateral ligament. (b) Replacement of the medial collateral ligament (Aron & Purinton, 1985a,b.)

bone screws (Fig. 44.15). On the medial side, a screw is placed in the medial malleolus and then two are placed in the talus, one proximally and one distally, thus allowing placement of a long and

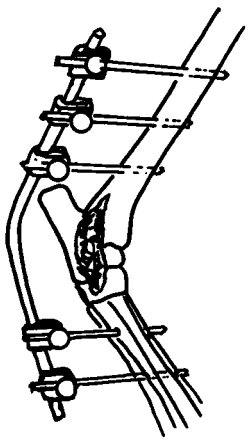

Fig. 44.16 Transarticular external fixator used for a shearing injury, resulting in tarsocrural instability.

a short collateral ligament. The technique is the same on the lateral aspect except that the screws are placed into the lateral malleolus and calcaneus in a similar fashion. Whichever method is used, the hock should be supported with a cast for 4–8 weeks postoperatively.

(4) Open luxations should be considered emergencies. If the joint can be thoroughly debrided within the 'golden period' of 6–8 hours (possibly extended to 12–18 hours if antibiotics are given immediately) then it may be possible to prevent the contamination from becoming established as infection. The presence of the latter in a joint, particularly one that is as tight-fitting as the tarsocrural joint, is likely to severely compromise the end result. After this it might be possible to consider ligament reconstruction or prosthetic replacement. However, it is often the case that much soft tissue has been lost and collateral replacement would require a great deal of foreign material to be left in a potentially infected site. Instead it may be worth considering the application of a transarticular external skeletal fixator (ESF) (Fig. 44.16). This will stabilise the joint whilst soft tissue healing takes place and at the same time will allow access to the wounds for daily topical treatment. Such a frame is most easily applied to the medial aspect which works quite well in most cases since the soft tissue loss is usually on the lateral aspect.

In small patients, placement of the fixation pins into the metatarsi can be difficult and the use of a Rudy boot (Gallagher & *et al.*, 1990) may sidestep such a problem (see Chapter 35, Fig. 35.11). The principle behind such a boot is to incorporate the distal pins into a cast applied to the pes rather than drilling them into the metatarsi. The fixator is left in place for about 8 weeks and then, once soft tissue healing has taken place, surgery to correct any instability (as discussed above) can be reconsidered. It is the experience of many surgeons that the soft tissue healing is sufficient to restore stability and that a second operation is often unnecessary.

(5) In cases where the techniques mentioned above have failed or where there is concurrent articular damage which would compromise function in the long term, it may be necessary to consider *tarsocrural arthrodesis* as a salvage procedure. Several techniques to achieve this have been described (Stoll *et al.*, 1975; Klause *et al.*, 1989; Sumner-Smith & Kuzma, 1989) but their differences revolve around the method of stabilisation.

In all cases, the articular cartilage is removed either by making parallel cuts with an oscillating saw across the distal tibia and trochlea of the talus or by burring the cartilage off the subchondral bone whilst maintaining the articular contours which then provide inherent postoperative stability. If an oscillating saw is used then attention has to be paid to the angle of the cuts since this will determine the fixed angulation of the joint. The aim is to produce a weight-bearing angle which is considered to be 135–145° in the dog and 115–125° in the cat. Stability may then be achieved using a transarticular bone screw or pin, placed through the talus and up into the tibial medullary cavity, with or without the addition of a figure-of-eight wire from the calcaneus to the tibia. Alternatively, several lagged bone screws can be placed across the joint in varying directions or a plate may be applied to the lateral aspect, after resection of the distal fibula, and contoured around the plantar aspect of the talus which then permits a bone screw to be placed through the plate and across the joint.

All these methods are directed at arthrodesis of the tarsocrural joint alone and each has its

advantages and disadvantages. However, there has been some concern over the incidence of lameness associated with OA of the more distal joints of the hock after successful tarsocrural arthrodesis (Doverspike & Vasseur, 1991; Gorse *et al.*, 1991). It has, therefore, been suggested that the optimum method of stabilising a tarsocrural arthrodesis would be to apply a plate to the dorsal aspect of the joint extending from the distal tibia right down to the proximal metatarsi (Klause *et al.*, 1989). This stabilises all the joints of the hock and should promote ankylosis of the distal joints as well as arthrodesis of the tarsocrural joint (Fig. 44.17). The main long-term complication of this technique is that of implant loosening (DeCamp *et al.*, 1993).

Any such arthrodesis requires external support (cast or splinting) postoperatively until radiographic fusion is evident. This may take 6–12 weeks depending on the patient's age.

Prognosis

Luxation of this joint carries the worst prognosis of all the hock luxations. There is little in the way of natural laxity in the joint but a good range of movement is essential for pain-free use of the hock. Unfortunately, periarticular fibrosis and posttraumatic OA may cause a persistent lameness even when stability has been restored. In dealing with such cases it must be remembered that they carry a guarded prognosis. However, the outlook is related, to some extent, to the degree of injury and it is often possible to return a dog to reasonable function, even if this requires arthrodesis.

In the cat it has been suggested that tarsocrural luxation has a poor long-term outlook (Schmökel *et al.*, 1994) and unless stable reconstruction can be achieved, to allow early return to function, then it may be better to consider arthrodesis from the outset.

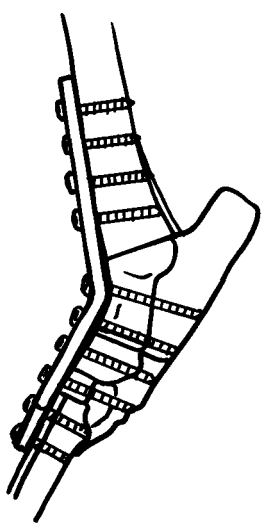

Fig. 44.17 Pantarsal arthrodesis using a plate.

Proximal intertarsal joint instability

(including calcaneoquartal arthrodesis)

Aetiology

Although proximal intertarsal joint (PITJ) instability may result from a traumatic injury, such as the dog putting its foot down a hole whilst travelling at speed or falling awkwardly, the most common presentation of luxation at this level within the hock is that of the chronic, progressive development of a plantigrade stance. The latter is thought to be associated with degenerative changes, of unknown aetiology, within the plantar ligaments which progressively weaken and stretch. With loss of its plantar support the PITJ will hyperextend causing 'hyperflexion' of the hock and a plantigrade stance. Such chronic, degenerative change is most commonly seen in the Shetland Sheepdog and collie-type breeds.

Diagnosis

In acute cases a non-weight-bearing lameness will be associated with pain and swelling in the region of the PITJ. It may be possible to appreciate instability but definitive manipulation might best be saved until the patient is anaesthetised for radiography. In the chronic cases, a mechanical lameness is evident with very little, if any, pain on palpation/manipulation. However, the dog is seen to weight-bear with the hock totally or partially dropped to the floor whilst the calcaneus can be palpated as being rotated so as to lie parallel to

the tibia (this allows differentiation from an Achilles tendon injury). It is important to check for collateral and dorsal instability as well as the often more obvious plantar instability, particularly in cases with an acute onset lameness. Additionally, it is well recognised that in the dogs with chronic onset changes, the owners may not present them until both legs are affected as they often cope reasonably well whilst the problem is unilateral. Thus, in such cases it is well worth checking both hocks for signs.

Radiography

This should provide a definitive diagnosis with mediolateral views of the hock in neutral and flexed positions showing plantar instability and stressed dorsoplantar views being necessary to demonstrate collateral instability. It is important to try and establish whether there is evidence of concurrent tarsometatarsal instability since this may influence the method of treatment chosen. It is often worth taking the same views of the contralateral hock as it is useful to know what is normal in the stressed dorsoplantar views and whether any early degenerative changes, such as enthesiophytes, are evident in the lateral views in chronic cases.

Treatment

In virtually all cases of instability at this level of the hock, there will be damage to the plantar support (Fig. 44.18a). When this has been lost, any attempts to manage the case conservatively or else reconstruct the plantar ligaments are doomed to failure. This is because the resulting plantar support will be inadequate to support the dog's weight in the long term. Instead, the only practicable method of regaining useful function in the hock is to arthrodese the calcaneoquartal joint.

Calcaneoquartal arthrodesis is achieved by first debriding any soft tissue, including remnants of plantar ligament, and articular cartilage from the joint surfaces by way of a plantarolateral approach. A cancellous bone graft may be packed into the joint space prior to stabilisation, although the need to do so is somewhat a matter of opinion. Several methods are available to stabilise the joint (Lawson, 1961; Campbell *et al.*, 1976; Brinker *et al.*, 1990; Allen *et al.*, 1993) and no studies are available to suggest which of these is optimal, either from a biomechanical or clinical results point of view. Essentially, the aim is to apply compression to the site and a list of methods would include: a pin and a figure-of-eight wire; a lagged bone screw +/– a figure-of-eight wire; or a compression plate applied laterally (Fig. 44.18). Some

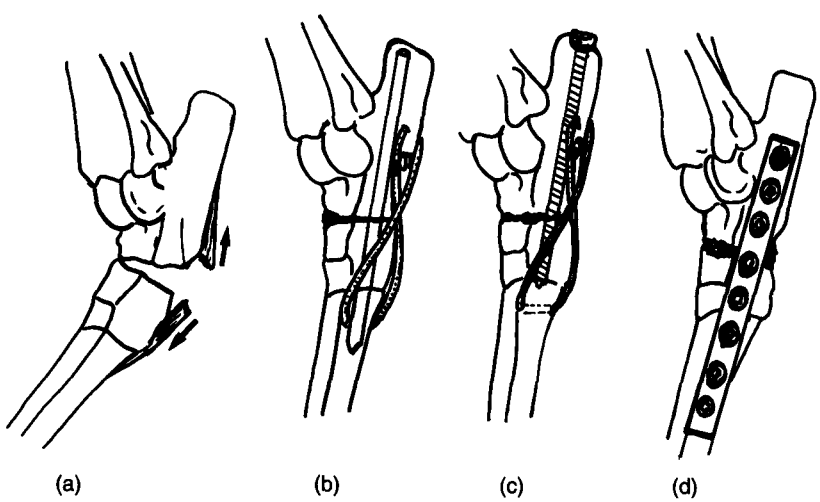

Fig. 44.18 (a) Calcaneoquartal (proximal intertarsal joint) instability associated with rupture of the plantar ligaments. (b) Arthrodesis using a pin and tension band wire. (c) Arthrodesis using a lag screw and tension band wire. (d) Calcaneoquartal arthrodesis using a laterally applied plate.

authors have suggested the use of crossed pins alone and although this technique might be adequate in smaller patients, there are no reports in the literature to support the notion that it is as good as the aforementioned techniques.

The most commonly reported method is that of placing a pin down the calcaneus, across the joint space and into the fourth tarsal bone together with a figure-of-eight wire placed from the calcaneus to the fourth tarsal bone (Fig. 44.18b). The aim of this combination is to create compression by way of a tension band effect but whether this will indeed be the case at this site is in some doubt, particularly when the joint is then put into a cast. It is perhaps more likely that the wire is acting as a prosthetic plantar ligament and thus will protect the pin, to an extent, during the early stages of healing without necessarily creating dynamic compression. A lagged bone screw, however, placed down the calcaneus and into the fourth tarsal bone will create static compression at the arthrodesis site. The addition of a figure-of-eight wire in this case might also help to protect the implant during healing (Fig. 44.18c). A plate may be used, particularly in older or larger patients or when the joint has been totally disrupted. Applied to the lateral aspect it may be secured to the calcaneus, the fourth tarsal bone and the heads of the metatarsi (Fig. 44.18d).

Whichever method of stabilisation is used, the hock should be supported in a cast until radiographic fusion is evident, which is usually at about 2 months. In most cases the implants are left *in situ* but laterally applied plates might need to be removed, owing to local irritation due to the lack of soft tissue cover in this area.

Prognosis

The prognosis for these cases is reasonably good. In one report of 39 hocks with calcaneoquartal instability, arthrodesis of the joint was successful in 33 (85%) after the initial surgery and 37 (95%) if success after revision surgery was included (Allen *et al.*, 1993). Not all the cases had injuries restricted to the PITJ but, overall, 76% of the dogs were sound when followed up for an average of 3 years. The most common complication of intertarsal arthrodesis is implant failure with loss of stability before fusion is complete. Incomplete removal of articular cartilage, failure to use a bone graft, strong tension band or external support all predispose to this complication.

Centrodistal joint (distal intertarsal joint) instability

Luxation of this joint is rarely seen but injury to the medial collateral support may lead to lameness through instability which will be demonstrable via stress radiography. External coaptation for 4–8 weeks may allow soft tissue healing and a restoration of stability. Otherwise a collateral prosthesis can be anchored around the heads of two small bone screws driven into the central and distal tarsal bones. Each screw passes into the fourth tarsal bone for increased purchase. The prognosis in these cases appears to be good, presumably because it is a low-motion articulation.

Tarsometatarsal instability

(including arthrodesis)

Aetiology

Luxation of the tarsometatarsal (T-M/T) joint is almost always a result of trauma, either in the form of a knock or else the dog putting its foot in a hole whilst running. Such injuries may involve rupture of the collateral and/or plantar ligaments. Since the collaterals insert on the heads of the second and fifth metatarsi, instability may also be caused by an avulsion fracture of the ligament's insertion. In addition, the chronic, progressive degeneration of the plantar ligament, as described under 'Proximal intertarsal joint instability' above, may also be seen. Again the Shetland Sheepdog and collie breeds appear to be overrepresented. Such degenerative breakdown in the plantar ligament may lead to subluxation of the PITJ and T-M/T joint simultaneously and this may alter the choice of management compared to when the PITJ alone is affected. A further complication may be when the T-M/T joint luxation does not develop until after the PITJ problem has been successfully treated!

Diagnosis

In acute cases a non-weight-bearing lameness will be associated with pain and swelling in the region of the T-M/T joint. Radiography should provide a definitive diagnosis although stressed views may be required, and fractures of the metatarsi should be looked for as well as widened joint spaces. It is important to evaluate both collateral and plantar support but this should be deferred until the patient is anaesthetised so that careful manipulation can be performed without causing the animal unnecessary pain.

Treatment

Conservative management

Conservative management involving the application of external support, for 4–8 weeks, to the joint may allow healing of ligament ruptures or avulsion fractures. However, if plantar ligament support is lacking then such management is likely to fail and most cases will develop a plantigrade stance once the cast has been removed.

Surgical management

Surgery should be considered in the following circumstances:

(1) In cases where a collateral ligament has been ruptured it may be possible to repair the soft tissue injury or, failing that, to replace it with a prosthetic ligament of monofilament nylon or a wire placed through tunnels drilled in the head of the appropriate metatarsal bone and around screws placed into the fourth or central tarsal bones.

(2) Where the instability is associated with an avulsion fracture of the collateral ligament's insertion, it may be possible to reduce the fracture and stabilise it using bone screws in either a positional or lagged fashion. External support should be used for 4–6 weeks postoperatively.

(3) Where there is gross instability associated with plantar ligament injury, +/– concurrent collateral damage, then attempts to reconstruct the ligamentar support are likely to fail and arthrodesis of the joint is the most appropriate form of management. After removal of the articular cartilage to allow apposition of bleeding bone surfaces, the joint is stabilised either with crossed pins or else a laterally applied dynamic compression plate (DCP) (Dyce *et al.*, 1998). The latter is secured, with bone screws, to the proximal metatarsals, the fourth tarsal bone and, if necessary, to the calcaneus. Some authors recommend the use of a pin and figure-of-eight wire, as described under 'Proximal intertarsal joint instability' except that the pin extends further, penetrating the head of metatarsal IV, and the wire passes from the calcaneus to the head of the fifth metatarsal bone. Placement of the pin can prove difficult as there is a tendency for it to pass down the plantar aspect of the metatarsi and afford no stability whatsoever. As a result this method has distinct limitations and is perhaps best avoided. Penwick & Clark (1988) described the use of a similar pin placed down the calcaneus, through the fourth tarsal bone and into the metatarsus, together with crossed pins directly traversing the T-M/T joint. Although they reported good results, even in dogs of 20–30kg bodyweight, the authors still prefer the application of a plate, particularly in dogs of this size.

However the joint is stabilised, it is advisable to utilise external support until radiographic fusion is evident (usually between 6 and 10 weeks, depending on the age of dog). The implants are usually left *in situ* although there may be a need to remove laterally applied plates owing to local irritation due to the lack of soft tissue cover in this area.

Prognosis

The prognosis for these cases is good and the major complications revolve around treatment of a collateral instability without recognition of concurrent plantar ligament damage which may then compromise the anticipated return to function.

Achilles tendon injury

The Achilles tendon (AT) (Fig. 44.19) or the common calcaneal tendon (CCT) inserts on the tuber calcaneus. It consists of the gastrocnemius tendon and the common tendon of the gracilis, biceps femoris and semitendinosus (Evans, 1993).

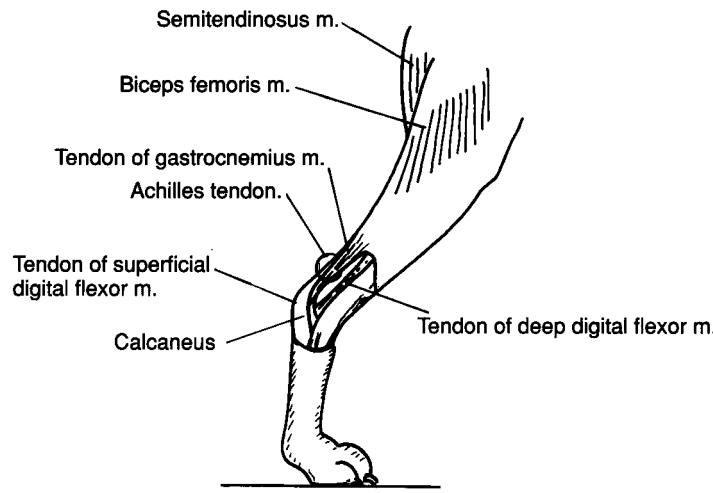

Fig. 44.19 Lateral view of the right hindleg showing the Achilles tendon (AT).

The superficial digital flexor tendon (SDFT) passes distally over the tuber calcaneus. A bursa is interposed between the tendon and the calcaneus.

Most lesions of the AT are traumatic in nature either due to overloading and/or direct trauma from the outside. Lesions of the AT have been classified into three types (Meutstege, 1993) and are listed below:

- *Type 1*: Complete AT rupture.
- *Type 2*: Lengthened AT system. Three subtypes are recognised:
  - (a) Musculotendinous rupture
  - (b) AT rupture with intact paratenon
  - (c) Gastrocnemius tendon avulsion with an intact SDFT.
- *Type 3*: Tendinosis and/or peritendinitis.

In addition, these injuries can be divided into acute or chronic lesions and types 2c and 3 also encompass the condition referred to as gastrocnemius enthesiopathy (see later).

Clinical features

The clinical features of these Achilles tendon lesions have also been described (Meutstege, 1993):

- *Type 1*: The combined tendon rupture results in a *plantigrade* stance if the dog attempts to weight-bear. A gap can be palpated between the tendon ends and there may be an external wound present. If the stifle is held in extension the tarsocrural joint can be completely flexed but there is *no* effect on flexor tension in the toes.
- *Type 2*: The stifle is extended and slight to moderate flexion or 'dropping' of the hock is seen during weight-bearing in all subtypes of type 2. Additional features are:
  - *Subtype 2a*: Varying degrees of swelling at the musculotendinous junction. When the stifle is fully extended, the hock can only be partially flexed. There is no effect on toe posture.
  - *Subtype 2b*: The findings are as in type 1. A gap can be felt between the tendon ends but in addition, when placed under tension, a string-like structure, the paratenon, can be felt along with the tendon ends. The effect on stifle and hock posture is the same as subtype 2a.
  - *Subtype 2c*: Is characterised by obvious thickening involving the insertion of the AT on the calcaneus. There is 'dropping' of the

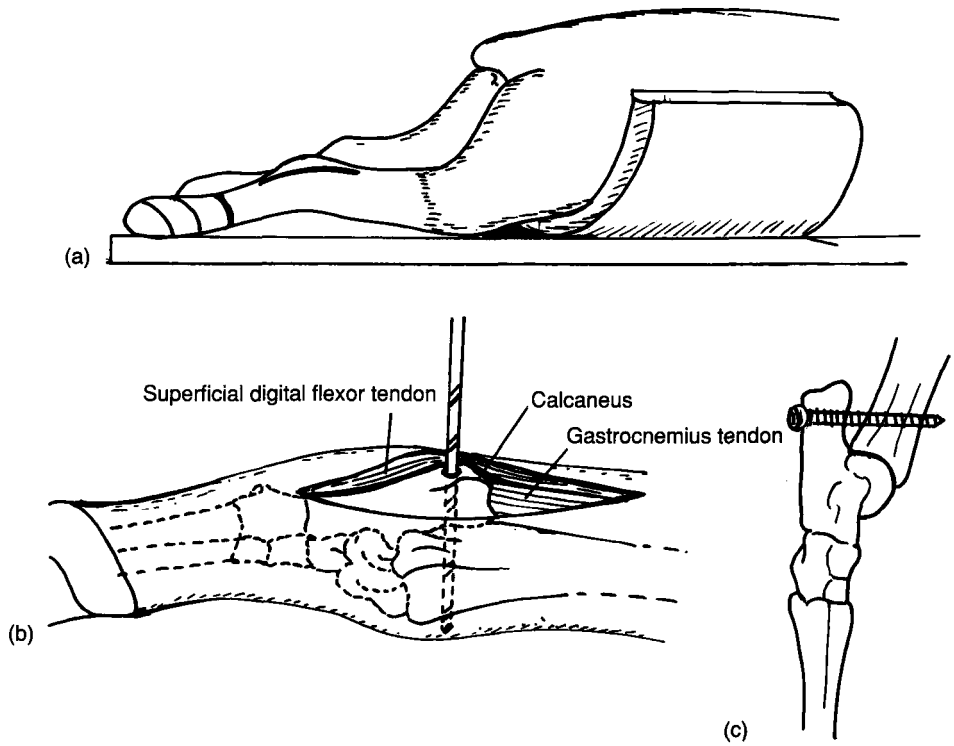

Fig. 44.20 Management of Achilles tendon (AT) injuries in the dog. Immobilisation of the hock in extension is achieved using a lag screw placed through the calcaneus and distal tibia. (a) The dog is positioned in sternal recumbency with the hindleg pulled caudally in full extension. A skin incision is made over the lateral aspect of the calcaneus. (b) The lateral retinacular attachments of superficial digital flexor tendon (SDFT) are incised and the tendon displaced medially to expose the caudal aspect of the calcaneus. A hole is drilled through the calcaneus and tibia. (c) Correct placement of the lag screw.

hock and 'clenching' of the toes due to increased tension in the SDFT.

- *Type 3*: The AT is thickened but affected dogs have a normal stance. When the stifle is extended it is not possible to flex the hock and there is no abnormal flexor tension in the toes.

Management of type 1 (complete) rupture of the Achilles tendon

Primary tendon repair is indicated (see Chapter 3, p. 25). However, before suturing the tendon, the hock should be fixed in extension using a lag screw placed through the calcaneus and distal tibia just proximal to the tarsocrural joint (Bloomberg *et al.*, 1976; Vaughan, 1979). This facilitates the repair and avoids undue tension on the sutures during the healing process.

The dog is positioned in sternal recumbency with the hindleg drawn out caudally in extension (Fig. 44.20a). This position will tend to keep the hock in extension and force the calcaneus close to the caudal aspect of the tibia, so preventing soft tissues becoming caught in the drill or tap. A caudolateral skin incision is made over the damaged area of the AT and is extended distally over the calcaneus (Fig. 44.20b). The SDFT is reflected medially after incising the lateral retinacular attachments, fully exposing the caudal aspect of the calcaneus. A lagged bone screw is placed through the calcaneus and into the distal tibia (Fig. 44.20c). In a Dobermann Pinscher a 4.5 mm cortical AO/ASIF type screw or a $\frac{5}{32}''$ Sherman screw would be suitable. It is important that the

screw is placed as close to a perpendicular to the long axis of calcaneus and tibia as possible to minimise the risk of fatigue failure or pull-out. It is generally advisable to use a screw that is longer than indicated on the depth gauge so that the tip of the screw protrudes through the cranial cortex of the tibia. This means that if the screw should break prematurely then the two halves of the broken screw can be removed through two, small, separate, stab incisions.

Once the hock has been fixed in extension then the severed ends of the gastrocnemius and SDFTs are identified and each tendon is repaired separately using three-loop-pulley, Bunnell and/or locking-loop sutures of 1 metric polydioxanone (PDS, Ethicon) or monofilament nylon.

Postoperatively a Robert Jones bandage is applied for 2 weeks and exercise is restricted for 6 weeks. The screw is removed through a stab incision at 6 weeks and exercise is restricted for a further 4 weeks before being gradually increased to normal levels.

Management of long-standing type 1 Achilles tendon ruptures

The principles of treatment are the same as for recent injuries, however, muscle contraction will have occurred which often prevents complete apposition of the tendon ends. If a defect exists, it is bridged with filamentous carbon fibre or polyester (Johnson & Johnson) which acts as a scaffold for collagen formation across the defect (Jenkins *et al.*, 1977). Alternatively, the deficit may be spanned by a fascia lata graft (Braden, 1976).

Management of type 2 rupture of the Achilles tendon

Subtypes 2a (musculotendinous rupture) and *2b* (AT rupture with intact paratenon) are treated by primary repair and the hock is immobilised in extension with a lagged bone screw for 6 weeks. Although *subtype 2c* (gastrocnemius tendon avulsion with an intact SDFT) can be seen as an acute injury, a chronic form is seen most often and is called *gastrocnemius enthesiopathy* (or tendonopathy). Treatment of both the acute and chronic forms is the same and is discussed below under 'Gastrocnemius enthesiopathy'.

Management of type 3 Achilles tendon lesions

Conservative management with restricted exercise for 6 weeks and administration of NSAIDs is usually sufficient, otherwise they may be treated as described under 'Gastrocnemius enthesiopathy'.

Gastrocnemius enthesiopathy

(Bonneau *et al.*, 1982; Butterworth; 1995)

The vast majority of cases affected with this condition are middle-aged Dobermann Pinschers. Owners will generally report a hindleg lameness of gradual onset or one which followed fairly minor trauma and then deteriorated gradually. The degree of lameness may vary from mild to severe with the dog only touching its toes to the ground. Typically the hock is partially 'dropped' (hyperflexed) during weight-bearing and this is associated with 'clenching' (hyperflexion) of the toes due to increased tension in the SDFT. Palpation of the gastrocnemius tendon reveals soft tissue swelling around its point of insertion on the calcaneus.

Radiography

A mediolateral radiograph provides most information about this condition but a dorsoplantar view is required for full evaluation. In chronic cases, as well as the soft tissue swelling of the gastrocnemius tendon detected clinically, there may be mineralisation within this tissue and the margins of the calcaneus invariably appear irregular in outline.

Management

In acute cases it may be worth considering 6–8 weeks of conservative management, involving room rest and short lead walks only, as some cases will show gradual improvement following such an acute 'flare-up'.

If lameness is long-standing, deteriorating or is associated with dropping of the hock then surgical treatment is advisable. In order to protect the injured tendon from the normal, disruptive

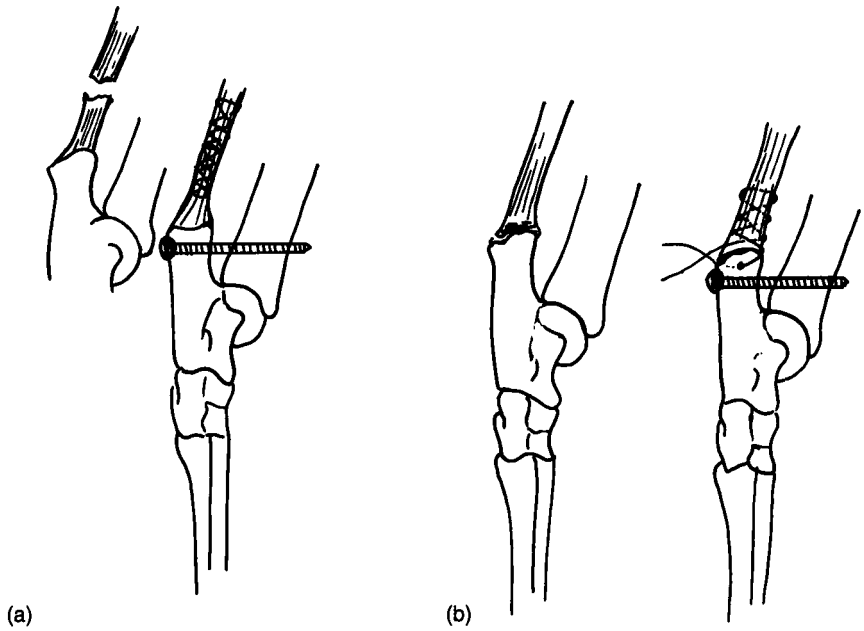

Fig. 44.21 (a) Rupture of the gastrocnemius tendon and repair. (b) Avulsion of the gastrocnemius tendon and repair.

stresses incurred by weight-bearing the tarsocrural joint should be fixed in hyperextension by placing a lagged bone screw through the calcaneus and then into the tibia (Fig. 44.21a). This should allow the tendon to heal and the screw is removed after 6–8 weeks. In addition, the tendon's attachment to the bone can be reinforced by placing sutures in the distal portion of the tendon and through the calcaneus.

Surgical approach and placement of the lagged bone screw is as described for ruptures of the AT. Once the screw has been inserted, the damaged area of tendon is identified and Bunnell and/or locking-loop sutures of 1 metric polydioxanone (PDS, Ethicon) or monofilament nylon are then placed in the distal portion of the gastrocnemius tendon, through a transverse tunnel in the calcaneus (Fig. 44.21b) and tied. Closure is routine with particular attention being paid to secure suturing of the lateral retinaculum to retain the SDFT in its normal position on the calcaneus.

Postoperatively a Robert Jones bandage is applied for 2 weeks and exercise is restricted for 6 weeks. The screw is removed through a stab incision at 6 weeks and exercise is restricted for a further 4 weeks before being gradually increased to normal levels. In cases that are treated for a bilateral problem, it is advisable to protect the bone screws by supporting the joints in light casts for the 6 weeks while the screws are *in situ*.

Prognosis

Although the period of convalescence can be in the order of 3 months the prognosis is good with the vast majority of dogs returning to normal activity. In cases with bilateral pathology the prognosis is more guarded since neither limb is given the chance to heal well and so recovery may not be complete, particularly if the patient is overweight and/or there is evidence of a plantigrade stance at the time of presentation.

Displacement of the tendon of the superfical digital flexor muscle

(Bernard, 1977; Bennett & Campbell, 1979; Vaughan, 1979)

Displacement of the tendon of the superficial digital flexor muscle occurs spontaneously or

through direct trauma. Rupture of the medial retinacular attachments allows the tendon to displace off the calcaneus in a lateral direction in most cases. The condition is seen most often in Shetland Sheepdogs and racing Greyhounds (Vaughan, 1987). There is a sudden onset of lameness, swelling and pain over the point of the hock and the tendon can be felt slipping in and out of its normal position. In untreated cases, extensive fibrosis and tenosynovitis occur. However, lameness may resolve despite the tendon remaining displaced.

Treatment

A curved incision is made over the medial side of the calcaneus, the tendon is reduced and the torn medial retinacular attachments are repaired with interrupted sutures of polydioxanone (PDS, Ethicon). In chronic cases, the stretched and fibrosed retinaculum is incised parallel with the tendon and overlapped with two layers of sutures in a similar way to the capsular overlap procedure described for patellar luxation on p. 517. Postoperatively, the hock is immobilised with a Robert Jones bandage for 2 weeks and exercise is restricted for 6 weeks.

Prognosis

Prognosis is normally good and even if primary surgical repair is unsuccessful, revision surgery is worthwhile. If necessary, the medial retinaculum can be reinforced with polypropylene mesh (Houlton & Dyce, 1983).

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Chapter 45
The Pes

The pes consists of the metatarsal bones and the digits.

Conditions of the pes

Conditions of the pes include:

- *Traumatic*
  - Fractures of the metatarsals
  - Fractures of the phalanges
  - Luxation of the metatarsophalangeal joint
  - Luxation of the proximal interphalangeal joint
  - Luxation of the distal interphalangeal joint
  - Dropped toe
  - Knocked-up toe
  - Fracture of the proximal, plantar metatarsophalangeal sesamoids
  - Section of the digital flexor tendons
- *Developmental*
  - Sesamoid disease
- *Other foot problems*
  - Pad injuries
  - Split webs
  - Nail injuries
  - Tracking foreign bodies

The diagnosis, management and prognosis for each of these conditions is the same as in the manus and the reader is referred to Chapter 36 for further details.

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Chapter 46
Hindlimb Amputation

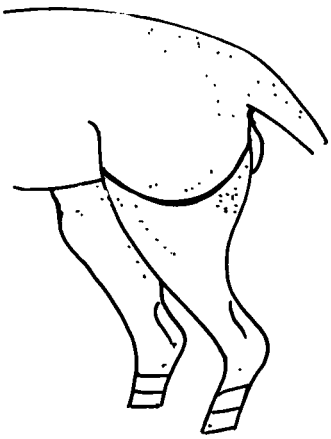
The indications for amputation of the hindlimb include:

- Neoplasia
- Gross trauma
- Gangrene
- Paralysis
- Osteomyelitis

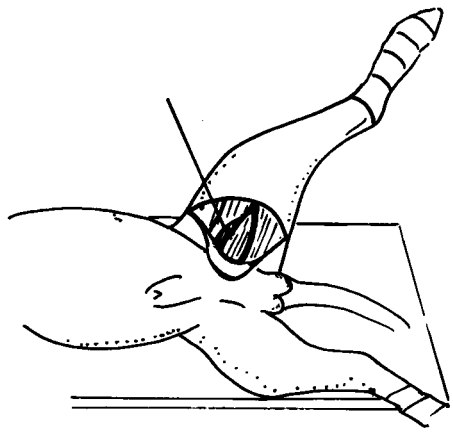
Basic technique for hindlimb amputation

A semicircular skin incision is made on the lateral aspect of the leg extending from the distal third of the thigh to the stifle joint (Fig. 46.1a). The leg is lifted by an assistant and a similar skin incision is made on the medial aspect of the thigh. The medial skin flap is reflected and the femoral artery and vein identified just cranial to the pectineus muscle (Fig. 46.1b). The artery and then the vein are ligated and severed. Early ligation of these vessels reduces haemorrhage during the subsequent amputation. The limb is then lowered and the lateral skin flap is reflected. Caudal to the femur, the biceps femoris, semimembranosus, semitendinosus and gracilis muscles are sectioned just proximal to the stifle together with the sciatic nerve and the distal femoral artery (Fig. 46.1c). Cranially, the tendon of the quadriceps is sectioned just proximal to the patella and then the sartorius muscle is severed to expose the distal femur. The muscle bellies are bluntly reflected from the femur with a swab and the adductor muscle is elevated to expose the proximal shaft. The amputation is completed by section of the femur through the proximal third of the shaft with a saw (Fig. 46.1d). The quadriceps muscle mass is folded caudally over the remaining stump of the femoral shaft and is sutured to the hamstring muscles followed by routine skin closure (Fig. 46.1e).

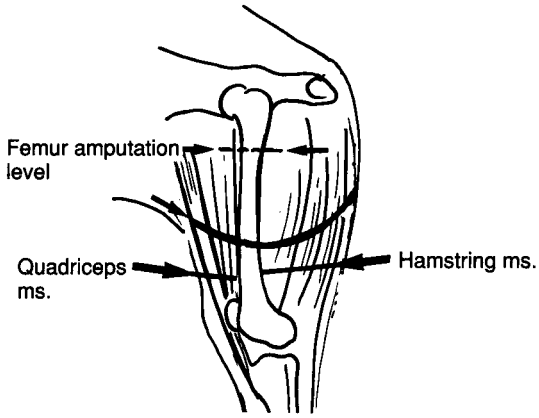
In certain cases it may prove necessary to consider amputation through the hip joint (e.g. neoplasia involving the stifle) but this technique is not used routinely as it is technically more difficult and is less cosmetic, leaving little protection for the genitalia.



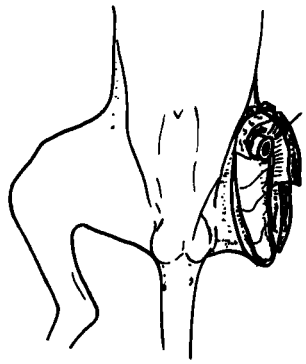
(a) Lateral skin incision.



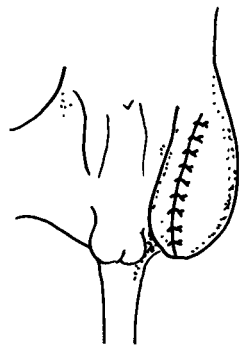
(b) Medial skin incision. The femoral artery and vein are identified cranial to the pectineus muscle and ligated.



(c) The lateral skin flap is reflected caudally, and the hamstring muscles and sciatic nerve are severed. Cranially the quadriceps muscles and sartorius muscles are sectioned just proximal to the patella. The muscles are pushed back from the shaft of the femur and the bone cut through the proximal third.



(d) Muscles are reflected over the cut end of the femur and sutured.



(e) Closure of the amputation stump is completed.

Fig. 46.1 Hindleg amputation.

Section 7
Miscellaneous Orthopaedic Conditions

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Chapter 47
Nutritional Bone Disease

An adequate dietary intake of calcium, phosphorus and vitamin D is necessary for the development and maintenance of normal bone. The requirements for dogs have been estimated (Krook *et al.*, 1971) as:

- 265 mg/kg bodyweight/day calcium
- 220 mg/kg bodyweight/day phosphorus
- 7 IU/kg bodyweight/day vitamin D

These figures apply to adult dogs; puppies require twice this intake. It is important to feed the correct ratio of calcium to phosphorus, otherwise a relative deficiency of one or the other occurs, and this ratio should be 1:1. In many diets there tends to be an excess of phosphorus, for example in meat the ratio of phosphorus to calcium is 20:1. A diet of meat and water will quickly lead to calcium deficiency but carnivores in the wild avoid this problem by also eating the bones of their victims which helps to redress the balance. The growing puppy will need about 50% meat in the diet initially which is gradually reduced to 30–40%. The rest of the diet is made up of carbohydrate and vegetable. If the puppy is receiving a balanced diet then a natural way of supplementing calcium and phosphorus is to give milk and bone meal (the latter is given at a rate of 15–20 g/kg dry weight of food (Abrams, 1962)). It must be stressed, however, that bone meal, because it is balanced, will not correct an abnormal dietary calcium to phophorus ratio. If a puppy is calcium deficient, then calcium lactate is given (available in 300 mg tablets [Evans]). Vitamin D is needed for the absorption of calcium from the bowel. Natural sources of the vitamin are meat and dairy products and supplements can be given in the form of cod liver oil capsules (5 ml cod liver oil/10 kg bodyweight/day for growth).

The cat is a carnivore and, as such, its dietary requirements differ from those of a dog, which is an omnivore. The specialised carnivorous metabolism of the cat has resulted in some nutrients which are derived from animal tissue being essential for cats but not for dogs. The cat has an insignificant, or limited, ability to synthesise nutrients such as vitamin A, arachidonic acid, arginine, taurine and niacin, but its metabolism allows the consumption of high-protein meals at all times and, consequently, a higher protein intake is required than in dogs. The cat is born with small stores of calcium and requires 200–400 mg calcium/day (6–8 g/kg dry weight of diet) for normal bone development. The ratio of calcium: phosphorus is important for both absorption and utilisation of these minerals and needs to be in the range 0.9–1.1:1. In the growing kitten, dietary concentrations of 8 g calcium and 6 g phosphorus/kg dry weight are to be recommended. Vitamin D is involved in the metabolism of calcium and phophorus and, for kittens, the minimal requirement should be provided by including 500 IU of vitamin D/kg dry weight of diet.

During the 1990s commercially prepared diets for dogs and cats that are growing, adult, lactating and geriatric have been developed. Owners can be well advised to use such diets, especially in the growing animal, to ensure that the right balance, as well as absolute quantities, of these nutritional requirements is achieved. If supplements are used, which should not generally be necessary, then it is important to ensure the balance is maintained since adding just calcium or just vitamin D will serve only to unbalance a scientifically balanced diet!

The clinical conditions which can be directly related to diet include:

- Nutritional secondary hyperparathyroidism
- Rickets
- Hypervitaminosis A
- Hypervitaminosis D

Although 'over-feeding', in terms of carbohydrate and/or protein intake, or 'over-supplementation', in terms of calcium, of young, rapidly growing dogs has been implicated experimentally in the aetiology of several developmental disorders (Hazewinkel *et al.*, 1985; Nap *et al.*, 1993), the importance of these aspects in the aetiology of the natural disease processes is not understood. Excessive calcium may lead to a state of hypercalcitoninism which reduces bone resorption (affects bone remodelling) and influences cartilage maturation and may, therefore, contribute to such disorders as cervical spondylopathy, osteochondrosis and retained cartilage cores. Excessive energy intake would be most likely to exert its influence by causing obesity which may play a role in the development of conditions such as hip dysplasia. Since the role of nutrition in these diseases is unclear they will not be considered further here.

Nutritional secondary hyperparathyroidism

Also known as 'all meat syndrome', 'butcher's dog disease', juvenile osteoporosis and osteogenesis imperfecta, this syndrome is usually a consequence of feeding a meat-rich diet to puppies or kittens. Because of such a diet's high phophorus: calcium ratio a relative calcium deficiency occurs. Other causes of hyperparathyroidism include an inability to absorb or utilise dietary calcium (true osteogenesis imperfecta) which is seen in some lines of German Shepherd Dogs, or reduced availability of dietary calcium as in vitamin D deficiency or renal insufficiency. The resulting hypocalcaemia causes release of parathyroid hormone which promotes release of calcium from the skeleton in order to maintain normal serum levels (9–12 mg calcium/100 ml serum).

Affected animals may show lameness or difficulty in standing due to bone pain or pathological fractures. It tends to affect the hindlimbs or vertebral column most frequently. They may also show joint laxity, due to poor soft tissue support, or paresis/paralysis, due to vertebral fracture. Radiographic examination shows poor skeletal mineralisation, thin cortices and pathological fractures (usually 'folding' or 'compression' fractures) (Fig. 47.1).

Treatment involves ensuring the animal receives a balanced diet +/– supplementation with bone meal (as discussed above), but vitamin D supplementation should be avoided as it will only make matters worse. Management of fractures is made difficult by the fact that they are often multiple and the bone quality is not good in terms of the application of implants. Initially, at least, it is usually best to provide cage rest whilst the diet is corrected. Once bone strength is improved it may be possible to consider any necessary stabilisation of fractures or, more likely, corrective surgery on any non-functional malunions. In those cases that are paraplegic due to vertebral fracture, or are severely affected with multiple fractures, euthanasia should be considered on humane grounds.

Nutritional rickets

This form of rickets is caused by a deficiency of vitamin D but the term is often used loosely and incorrectly. It is now a very rare clinical disease.

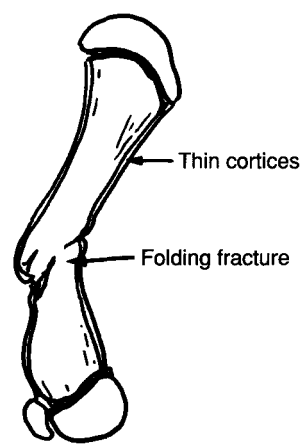


Fig. 47.1 Line drawing illustrating a folding fracture of a humerus in a 3-month-old puppy with secondary hyperparathyroidism.

Hazewinkel (1989) showed that dogs are dependent on dietary vitamin D and cannot synthesise it in their dermis under the influence of sunlight in the same way as some species can. It has been suggested that vitamin D deficiency makes cartilage matrix highly stable such that it does not mineralise, resulting in thickened growth plates. However, in one study (Campbell, 1964) puppies that were fed a diet devoid of vitamin D failed to develop rickets.

Affected animals show 'flaring' of metaphyses and possibly lameness because of bone pain or pathological fractures. Radiography will show wide growth plates, flared metaphyses and poor skeletal mineralisation (Fig. 47.2). Laboratory findings may include hypocalcaemia, hypophosphataemia and increased levels of parathyroid hormone, which is attempting to raise the serum ion levels. Treatment involves correction of the dietary inadequacy and resting the animal until skeletal strength is regained.

See also 'Vitamin D-dependent rickets' in Chapter 48, p. 611.

Hypervitaminosis A

This is a disease that is peculiar to cats and results from an excessive dietary intake of vitamin A, historically involving a 'liver-rich' diet. For reasons that are not understood, this excess causes periosteal bone formation, particularly on the vertebrae and around the major joints. Clinically, it usually appears in cats aged between 2 and 4 years and they demonstrate stiffness of the cervical spine (Seawright *et al.*, 1967) and/or lameness associated with joint pain/stiffness. In immature animals, growth may be retarded. Radiography, in typical cases, shows extensive new bone formation along the cervical and thoracic regions of the vertebral column (Clarke *et al.*, 1970), often leading to fusion of the vertebrae and spinous processes (Fig. 47.3a), and around one or more joints (Fig. 47.3b). Treatment involves correction of the dietary intake of vitamin A which will usually halt the progression of the disease, although the new bone formation will not regress. Any lameness or stiffness may need to be managed long-term with a non-steroidal anti-inflammatory drug (NSAID) (e.g. Metacam, Boehringer Ingelheim) or steroid therapy.

Hypervitaminosis D

Excessive intake of vitamin D is a rare occurrence but may result from oversupplementation with cod liver oil capsules or (one case seen by one of the authors) a dog gaining access to its owner's concentrated vitamin D supplement, a problem that has also been reported by the Veterinary Poisons Information Service in a number of dogs (Campbell, 1997). In the case of oversupplementation, there is a generalised loss of mineral from the skeleton and soft tissue mineralisation. In the case of the single, massive overdose, the dog showed severe, life-threatening, loss of control over serum calcium levels which required several periods of forced diuresis. In the more long term the Great Dane involved developed bowing of both femurs with secondary patellar luxations. Bilateral corrective surgery was required.

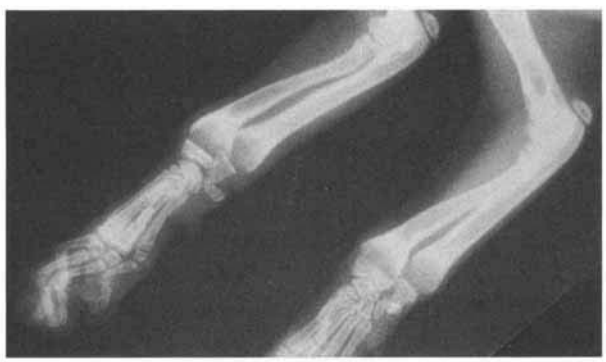
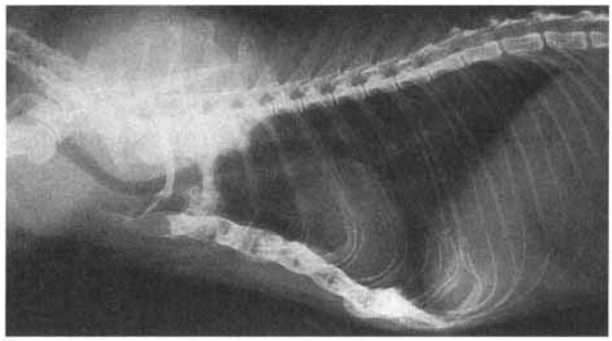
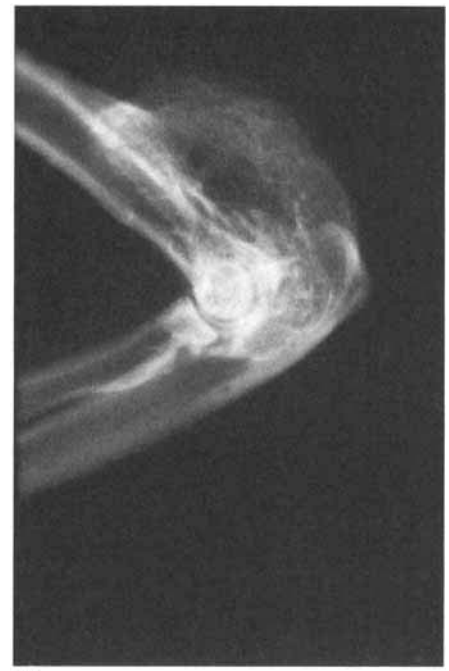


Fig. 47.2 Carpal radiographs of a 7-month-old Bengal cat illustrating rickets. The growth plates are wide and the metaphyses show increased density and flaring. There is bowing of the radius and ulna, and the cortices are thin.



(a)



(b)

Fig. 47.3 (a) Lateral view of a cat's thorax showing signs typical of hypervitaminosis A. Fusion of the sternabrae and cranical thoracic vertebrae is apparent.
(b) Mediolateral view of a cat's elbow showing signs typical of hypervitaminosis A. In this case the periarticular exostoses are creating a bridge between the olecranon and the distal humerus.

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Hamish R. Denny, Steven J. Butterworth

Chapter 48
Non-nutritional Bone Disease

A number of bone diseases exist in which the aetiology may or may not be understood but where diet is not considered to have a role, including:

- Panosteitis
- Metaphyseal osteopathy
- Hypertrophic osteopathy
- Craniomandibular osteopathy (see Chapter 16, p. 172)
- Chondrodysplasia
- Renal secondary hyperparathyroidism
- Vitamin D-dependent rickets
- Osteopetrosis

Panosteitis

This condition is a reasonably common cause of lameness in young dogs of medium to large breeds, especially German Shepherd Dogs, but its cause remains undetermined. The subject has been reviewed by Bohning *et al.* (1970), Tandy (1977) and Muir *et al.* (1996). The pathology of the condition involves replacement of adipose tissue within the medullary cavities of affected long bones by fibrovascular tissue that then becomes mineralised. In some cases the periosteal envelope of the bone is also involved, giving rise to periosteal new bone formation. In those cases where there is no periosteal involvement the condition may be more appropriately referred to as endosteitis.

Affected puppies usually present at 5–12 months of age with a history of acute onset or gradually worsening lameness that may wax and wane or shift from one leg to another. Males are affected more frequently than females (ratio in the region of 4–6:1). Systemic signs of illness (anorexia, pyrexia) may be associated with the lameness. Pain is observed when digital pressure is applied to an affected area of bone (especially if there is periosteal involvement) or if an adjacent joint is manipulated in such a way as to cause traction in a tendon originating or inserting on an affected area of bone.

Radiography of an affected bone may show increased lucency within the medullary cavity in the early stages, such that the bone has a 'reamed' appearance but later on the changes are those of a granular or sclerotic appearance to the medullary cavity (Fig. 48.1(a)) with or without evidence of periosteal new bone formation. The changes are most commonly detected in the areas surrounding the bone's nutrient foramen. Other radiographic findings may confuse the picture in terms of cause of lameness, for example, as in Fig. 48.1b where elbow lameness in a German Shepherd Dog was associated with both ulnar endosteitis and an ununited anconeal process.

Treatment is symptomatic, involving rest and the administration of non-steroidal anti-inflammatory drugs (NSAIDs). In most cases the lameness resolves within a few days to a few weeks but recurrence in the same or another limb is not uncommon up until the dog reaches 18 months to 2 years of age.

Metaphyseal osteopathy

Also referred to as hypertrophic osteodystrophy, Barlow's disease and skeletal scurvy, this disease is seen predominantly in medium to large breeds of dog (especially Great Danes) between 4 and 6 months of age. The aetiology remains obscure. In the past nutritional imbalances have been

suggested, without any substantiating evidence, and the possibility of vitamin C deficiency was driven by the similarity of the radiographic changes in this disease compared to scurvy in man, but again this is unproven and doubtful. More recently, Mee *et al.* (1993) detected distemper virus antigen in the cells of affected metaphyses in clinical cases and this led to the suggestion of the disease being a manifestation of canine distemper. However, although many dogs with the disease do show signs of systemic illness, not all do so and the significance of this finding in relation to distemper virus being a causative agent remains unproven. Even the source of the distemper virus antigen is unclear since many puppies of this age will have been recently vaccinated against the disease.

Affected dogs tend to show multiple lameness associated with painful swellings just proximal to the carpi and/or tarsi, but although the distal radial, ulnar and tibial physes are affected most commonly, any physis can be involved (see later regarding involvement of the proximal femoral physis). When multiple limbs are involved the dog may be unable to stand. They may also show systemic signs such as anorexia, pyrexia and general malaise. The severity of such signs may vary from very mild to *very* severe. In the long term, metaphyseal osteopathy is seldom a cause of premature growth plate closure, but the possibility should be kept in mind.

The radiographic changes around the affected metaphyses are usually pathognomonic (Fig. 48.2). The metaphyses show areas of sclerosis and

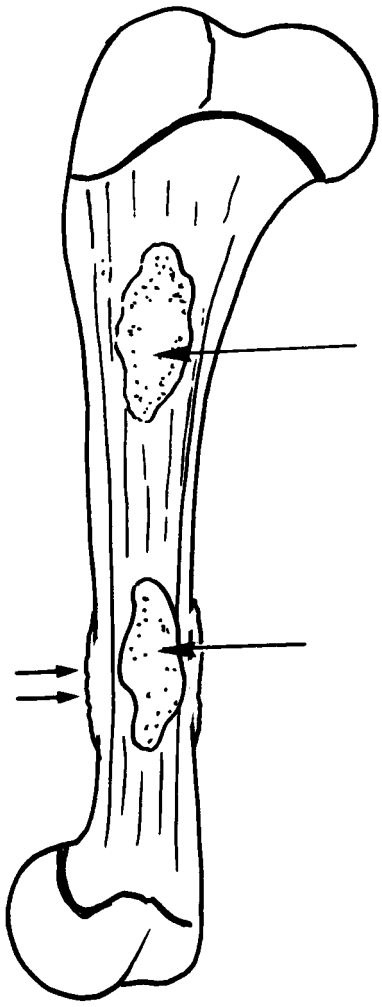

Fig. 48.1 (a) Illustration of the radiographic appearance of panosteitis affecting a humerus showing granular deposits of bone in the medullary cavity (large arrows) and, distally, periosteal new bone (small arrows). (b) Mediolateral radiograph of a 5-month-old German Shepherd Dog's elbow. A granular, increased density within the proximal ulnar diaphysis is indicative of panosteitis (or, since there is no periosteal reaction, endosteitis) but the elbow lameness detected clinically might also be associated with the ununited anconeal process (UAP) seen better in a flexed view.

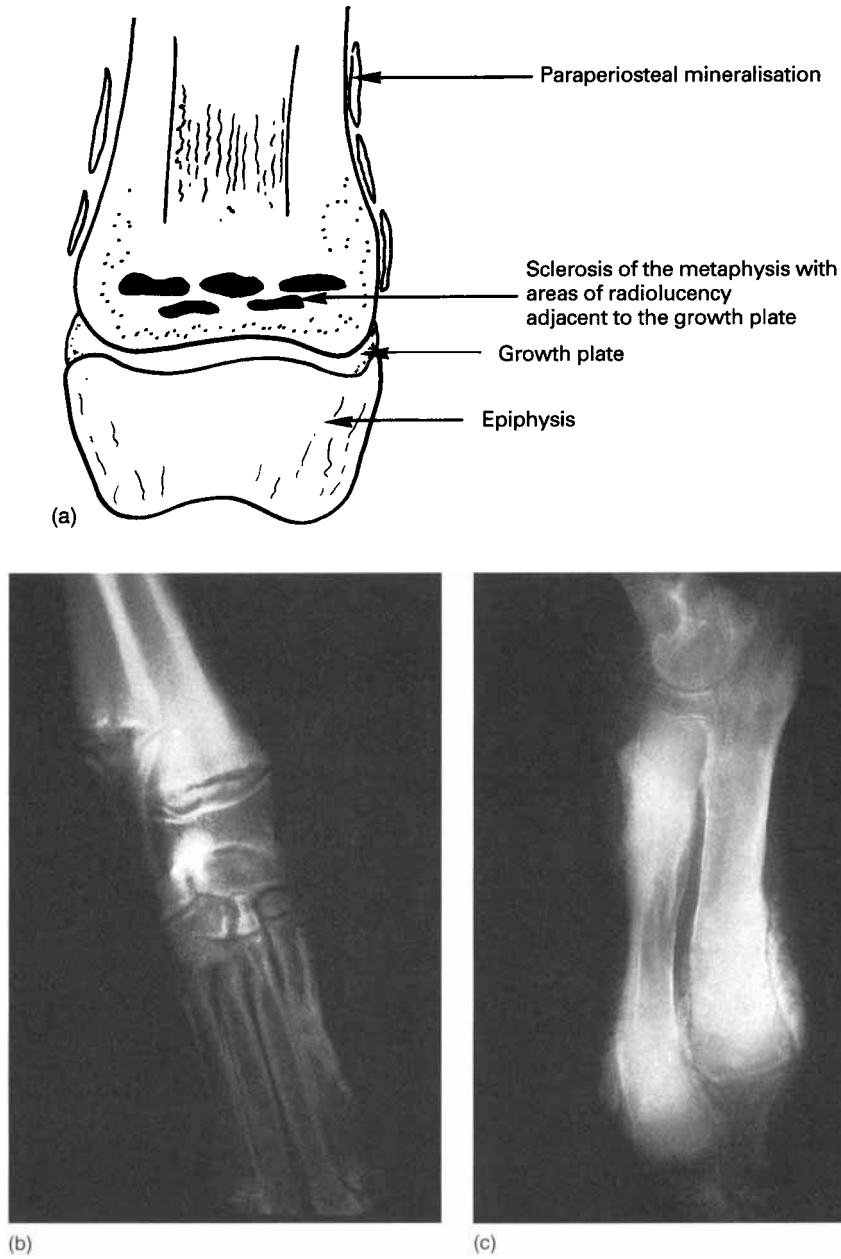

Fig. 48.2 (a) Illustration of the radiographic appearance of metaphyseal osteopathy affecting the distal antebrachium.
(b) Craniocaudal radiograph of a 5-month-old Border Collie's antebrachium showing relatively mild changes associated with metaphyseal osteopathy. A clear lucent line is present in both distal metaphyses, parallel to the physes.
(c) Mediolateral radiograph of a 5-month-old Great Dane's antebrachium showing severe changes associated with metaphyseal osteopathy affecting not only the distal radial and ulnar metaphyses but also that of the proximal radius. The metaphyseal regions are irregular in density and a mineralised 'collar' has formed around the bones in these regions.

bands of lucency which are often parallel to the physis itself. In addition, there may be a 'collar' of mineralised material adjacent to the bone at this level. Although the latter is often described as periosteal new bone formation, the density is actually paraperiosteal and more likely to be mineralisation of organising haemorrhage around the periosteum.

There is no specific treatment for the condition and management has to be supportive in nature. Ensuring that a balanced diet is being fed would be prudent, but although the administration of vitamin C has been advocated (Campbell (1964) suggested 0.5–1 g/day) there is little evidence to support such supplementation. In most cases, NSAIDs and rest are all that is required, but in those cases that show marked systemic signs intravenous fluid therapy will be necessary, and possibly intravenous feeding until the signs are brought under control.

Most animals make a complete recovery within a few weeks, although relapses do occur occasionally during the remainder of their growth period. However, in those where the systemic signs are severe the condition can become life-threatening or require that euthanasia be considered.

A condition characterised by lameness associated with radiolucency in the femoral necks of cats (often bilateral) has been reported (Queen *et al.*, 1998) and the pathology involved was considered to resemble metaphyseal osteopathy fairly closely. Treatment of such cases generally involves excision arthroplasty with satisfactory results. A few similar cases have been seen by the authors in dogs, usually giant breeds, that have been presented with acute onset lameness, associated with hip pain, following minor trauma. Fractures of the femoral neck were found but with radiographic changes that appeared to 'pre-date' the recent injury and, in one case, radiolucent patches were found in the contralateral femoral neck (though clinical signs did not develop). Again, excision arthroplasty was used with satisfactory results.

Hypertrophic osteopathy

Also referred to as hypertrophic pulmonary osteoarthropathy or Marie's disease, this condition is usually associated with some form of intra-thoracic or intra-abdominal space-occupying lesion (Alexander *et al.*, 1965). Its name has been shortened to hypertrophic osteopathy since the condition is not always associated with pulmonary pathology nor does it always affect joints. It is characterised by periosteal proliferation, predominantly affecting the bones of the distal limbs. The affected regions are swollen and painful, causing lameness. Radiographic findings are considered pathognomonic and comprise pallisading, periosteal new bone formation beginning on the distal phalanges, metacarpal and metatarsal bones (Fig. 48.3). Other bones may become affected in due course. If the primary lesion can be successfully removed then the new bone will remodel and the clinical signs associated with the osseous changes will usually improve. The aetiological link between the primary disease and secondary bone involvement remains obscure.

Chondrodysplasia

There are a number of rare disorders that are loosely connected by the term chondrodysplasia. Radiographically they resemble rickets, with

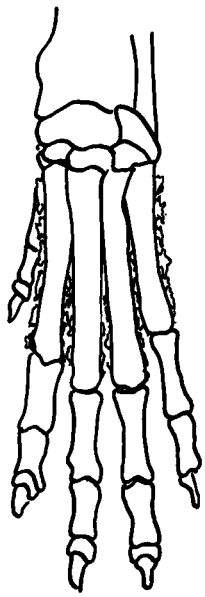

Fig. 48.3 Illustration of the pallisading periosteal new bone formation seen radiographically in cases with hypertrophic osteopathy.

widening of the growth plates, but there are no abnormalities with respect to calcium, phosphorus or vitamin D homeostasis. They are believed to be genetically controlled and have been reported predominantly in the Miniature Poodle but also in the Alaskan Malamute, Beagle, Bull Terrier and English Pointer. In the latter the condition is described as an enchondrodysplasia with changes in both physeal cartilage, causing dwarfism, and articular cartilage, causing lameness, and in this breed a homozygous, recessive mode of inheritance has been reported (Whitbread *et al.*, 1983).

A similar problem has also been reported in two unrelated kittens (Gunn-Moore *et al.*, 1996). Initially their clinical and radiographic features were thought to be a result of vitamin D-dependent rickets (see below) but laboratory investigation failed to show this was the case and a diagnosis of metaphyseal chondrodysplasia was made.

Renal secondary hyperparathyroidism

Renal insufficiency with a reduction in glomerular filtration rate may cause retention of phosphorus and, since the product of the serum concentrations of phosphorus and calcium must remain constant, this causes a fall in serum calcium levels. This, in turn, causes hyperparathyroidism and the increased production of parathyroid hormone results in a net increase in osteoclastic activity so as to raise the serum calcium levels. This process of demineralisation affects all bones but those of the skull, especially the mandible and maxilla, are affected first. The mandible may feel soft, leading to the term 'rubber jaw', and teeth may become loose. Excess salivation may be noted and softening of the maxilla may cause respiratory obstruction. Radiographic examination of the skull shows evidence of demineralisation whereby the teeth, which are unaffected, appear prominent and almost 'floating'. Treatment has to aim at correcting renal function.

Vitamin D-dependent rickets

Cases of rickets have been identified in the dog whereby there is no dietary deficiency of vitamin D and the animals will not respond to supplementation (Johnson *et al.*, 1988). Other than their lack of a response to treatment, there is no clinical difference between these animals and those suffering with nutritional rickets (see Chapter 47). It is hypothesised that the aetiology of this problem is an inability to metabolise the vitamin D_3 (cholecalciferol) from the diet, by serial hydroxylation, into its active form, 1,25-dihydroxycholecalciferol. Such cases may respond to treatment with dihydrotachysterol which is metabolised by the liver into an active analogue of 1,25-dihydroxycholecalciferol.

Osteopetrosis

Osteopetrosis, or 'marble bone', is a term used to describe a generalised increase in skeletal mass (Marks, 1984). In osteopetrotic bone there is marked endosteal thickening of the cortex with irregular dense structures within the medullary cavity. The subchondral bone is extremely dense and cancellous bone structures are no longer visible. Congenital osteopetrosis has been described in dogs and is reported to cause anaemia (Riser & Frankhauser, 1970; Lees & Sautter, 1979). Two cases have been described in adult cats (Kramers *et al.*, 1988) and one of these developed anaemia. Feline leukaemia virus-induced medullary sclerosis has also been reported in experimental kittens (Hoover & Kociba, 1974).

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Chapter 49
Myopathies

Myopathies can be classified into two broad groups, *degenerative* and *inflammatory*. Degenerative myopathies can be further divided into *hereditary* and *acquired* myopathies. A review of the various myopathies encountered in dogs was published by Blot & Fuhrer (1995).

Hereditary or breed-specific myopathies

Myotonia in Chow Chows (Farrow & Malik, 1981)

Myotonia describes the state in which active contraction of a muscle persists after cessation of voluntary effort or stimulation. Signs become apparent from 2 to 3 months of age and include stiff gait, bunny-hopping and difficulty in rising or climbing stairs. The pup's action improves with exercise but becomes worse with excitement or cold weather. If muscles are percussed with artery forceps a dimple is formed which persists for several seconds. The myotonic dimple can be produced both in the conscious and anaesthetised animal. Electromyography produces characteristic 'dive bombing' sounds. Procainamide may reduce the signs of myotonia in affected dogs.

Hereditary myopathy in Labrador Retrievers (Kramer *et al.*, 1981; McKerrell & Braund, 1987)

In the UK, this condition appears to be confined to Labrador Retrievers from working strains. Clinical signs include weakness that becomes worse with exercise, limb stiffness and a hopping gait. The condition is made worse by excitement or cold weather. Megalo-oesophagus has been reported as a complication in one case (McKerrell & Braund, 1987). Rest and the administration of diazepam helps to relieve the signs. The condition stabilises at about 6 months of age, however some muscle atrophy persists and there is poor exercise tolerance which makes affected animals unsuitable for work but they can be kept as house pets.

Golden Retriever myopathy

This myopathy affects male animals from 6 to 8 weeks of age and is characterised by severe stiffness and weakness. There is progressive muscle atrophy and the jaw muscles may be affected as well as the limbs (De Lahunta, 1983).

Irish Terrier myopathy

A myopathy affecting male Irish Terriers from 8 weeks of age has been described (Wentick *et al.*, 1972).

Myopathy of Devon Rex cats

A primary myopathy has been identified in a series of closely related Devon Rex cats (Malik *et al.*, 1993).

Nemaline myopathy in cats (Cooper *et al.*, 1986)

Nemaline, or rod, myopathy is a congenital muscle disorder which has only rarely been reported in cats. Clinical signs become apparent between 6

and 18 months with muscle weakness and, later, trembling. The cat has difficulty in moving and the gait becomes hypermetric.

Acquired myopathies

Atrophic myopathy

Examples are disuse atrophy or neurogenic muscle atrophy following peripheral nerve injury. The latter produces characteristic denervation potentials in electromyography studies.

Ischaemic myopathy

An example is bacterial endocarditis, or cardiomyopathy, leading to aortic thromboembolism and vascular occlusion in the cat. For further details see 'Ischaemic neuropathy' in Chapter 50.

Nutritional myopathy

A myopathy associated with diets deficient in vitamin E or selenium has been produced experimentally in dogs but is unlikely to occur naturally.

Metabolic myopathy

Muscle atrophy and weakness are well-recognised features in dogs with hyperadrenocorticalism but some cases have more obvious abnormalities indicative of myopathy, i.e. stiff gait with increased resistance to passive limb flexion.

Exercise-induced myopathy

Exercise-induced myopathy is seen most often in the racing Greyhound. Ischaemic changes occur in muscle with consequent rhabdomyolysis and haemoglobinuria. The dog is distressed and affected muscles are swollen and painful. Severely affected dogs may collapse and die while others can die 2–3 days later because of renal failure associated with myoglobinuria. Intravenous fluids and sodium bicarbonate are essential to correct the hypovolaemia and metabolic disturbance.

Inflammatory myopathies

Myositis of the masticatory muscles (temporal muscle myositis, eosinophilic myositis)

Myositis of the masticatory muscles is a relatively common form of myositis encountered in the dog. It appears to be an autoimmune disease and the German Shepherd Dog is affected most frequently but the myopathy can occur in dogs of any breed, age or sex. In the acute phase there is swelling of the jaw muscles with pain and reluctance to open the mouth. The acute phase is followed by a chronic form with progressive muscle atrophy and difficulty in opening the mouth. Early treatment with immunosuppressive doses of corticosteroids usually gives satisfactory results.

Idiopathic polymyositis

Idiopathic polmyositis is probably the commonest polymyopathy diagnosed in dogs (Christman & Averill, 1983; Smith, 1989). Large breed, adult dogs are usually affected. Clinical signs include muscle weakness, muscle pain, stiffness, pyrexia and megalo-oesophagus. Prognosis is guarded. Immunosuppressive doses of corticosteroids are used for treatment and in refractory cases a combination of prednisolone and azathioprine is used (see also polyarthritis/polymyositis syndrome in Chapter 8, p. 71).

Infectious bacterial myositis

Focal traumatic infectious myositis is common and occurs secondary to wounds and bites. In the dog, *Staphyloccus* spp. and *Streptococcus* spp. are usually involved while in the cat *Pasteurella multocida* is more frequently encountered. Clostridial myositis (*Clostridium welchii/perfringens*) is occasionally encountered in dogs and has been reported as a complication of open fractures (Denny *et al.*, 1974). Clostridial myositis results in extensive muscle destruction with marked soft tissue swelling and severe pain. Emphysematous crackling is evident on palpation due to the production of pockets of gas in the tissues. There is marked elevation in body temperature (up to 106°F). Treatment involves debridement of

infected tissue, drainage of any abscess that may be present and treatment with the appropriate antibiotic as determined by culture and sensitivity testing. Clindamycin (Antirobe, Pharmacia and Upjohn) is recommended for treatment of anaerobic bacterial myositis (Blot & Fuhrer, 1995).

Parasitic myositis

Toxoplasma gondii and *Neospora caninum* are closely related protozoan parasites. Both can cause polyradiculoneuritis and polymyositis in puppies. The main clinical feature is a progressive paralysis of the hindlimbs resulting in a characteristic spastic hyperextension of the legs. For many years cases of neosporosis were wrongly diagnosed as toxoplasmosis because the morphology of the lesions is similar (Dubey *et al.*, 1988). Diagnosis of neospora infection is based on the magnitude of the indirect immunofluorescent antibody test (IFAT). A titre greater than 1:800 is considered positive. Muscle biopsy can also be used to confirm the diagnosis. Treatment with sulphonamides, pyrimethamine and/or clindamycin allows a functional recovery in 60% of cases.

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Chapter 50
Peripheral Neuropathies

Peripheral neuropathies can be divided into inherited and acquired neuropathies. For a comprehensive review of these the reader is referred to Duncan (1989).

Inherited canine neuropathies

The inherited neuropathies in dogs are classed as autosomal recessive and include the following.

Giant axonal neuropathy (Duncan & Griffiths, 1981)

German Shepherd Dogs of 14–15 months of age are affected. There is hindleg weakness and ataxia. Megalo-oesophagus appears to be a cardinal feature of the disease.

Progressive axonopathy of Boxer Dogs
(Griffiths *et al.*, 1980)

The disease is inherited as an autosomal recessive trait. Age at the onset of signs is usually between 3 and 6 months. The main presenting sign is hindlimb ataxia with decreased muscle tone and absence of patellar reflexes but preservation of the pedal reflexes, good conscious pain sensation (CPS) and virtual absence of muscle atrophy. The signs are symmetrical. Nerve roots and, to a lesser extent, peripheral nerves show changes characteristic of demyelination/remyelination.

Sensory neuropathy in Dachshunds
Sensory neuropathy in English Pointers
Hypertrophic neuropathy in Tibetan Mastiffs
Globoid cell leucodystrophy in West Highland White Terriers

Acquired canine neuropathies

Distal denervating disease
(Griffiths & Duncan, 1979)

This is a degenerative neuropathy of the distal motor axon in dogs. Affected animals present with quadriparesis, local reflex activity is depressed and there is muscle atrophy. Spontaneous recoveries have been recorded.

Feline inherited neuropathies

Neuropathy of inherited hyperchylomicroanaemia
Neuropathy of Niemann–Pick disease (lysosomal storage disease)

Feline acquired neuropathy

Ischaemic neuropathy

The most important acquired feline neuropathy is ischaemic neuropathy due to thromboembolism. Occlusion of the aortic trifurcation with emboli produced as a sequel to cardiomyopathy causes an acute onset of paresis or paraplegia with pain, loss

of femoral pulses, cold hindlimbs and pale foot pads. It is the release of serotonin (5-HT) from the platelets, not just loss of blood supply, which is the main cause of muscle and nerve ischaemia. Treatment is aimed at the underlying cardiac disease. Analgesics are used as necessary and aspirin therapy is recommended for the rest of the cat's life to inhibit platelet function (25 mg/kg every 3 days – Flanders, 1986). A very guarded prognosis is given in all cases because although the cat may recover from the neuropathy, further relapses are likely if the cardiomyopathy is not controlled.

Neuromuscular transmission disorders

Myaesthenia gravis

(Hertage & McKerrell, 1989)

Myaesthenia gravis is a disorder of the neuromuscular junction (i.e. a junctionopathy). The condition has been reported in both dogs and cats. Congenital myaesthenia gravis is seen in the Jack Russell Terrier, Springer Spaniel and Smooth-Haired Fox Terrier. It is associated with signs of weakness at 6–8 weeks of age. Acquired myaesthenia is seen most often in the German Shepherd Dog but can affect other adult large breeds of dog. The signs are severe muscle weakness and fatigue on exercise which improves with rest. Diagnosis is based on clinical signs and response to the Tensilon test (intravenously administered edrophonium chloride). In positive cases there is a dramatic improvement following injection which lasts for a few minutes. Treatment involves the administration of oral anticholinesterase drugs, e.g. pyridostigmine bromide (Mestinon, Roche).

Swimmers

Occasionally one or more puppies in a litter is affected by the *swimmer syndrome*. Affected puppies are unable to stand and they make swimming movements with their limbs in abduction as they attempt to move. They often have flat chests and, if allowed to survive, may develop permanent joint deformity. The pups are strong, alert and no neurological deficits are usually apparent. The pathogenesis of the condition is not understood and it is described as a musculoskeletal growth abnormality. Breeders who recognise the condition can sometimes improve or correct the deformity by hobbling the legs, splinting the chest and keeping the pups on soft litter.

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Chapter 51
Bone Neoplasia

Bone tumours can be classified into four groups:

- Benign
- Malignant
- Allied malignant
- Metastatic

The majority of bone tumours encountered in the dog are malignant and *osteosarcomata* account for 80% of tumours of the skeletal system. Benign bone tumours and metastatic tumours are uncommon in animals.

- *Benign tumours* include:
  - Osteoma
  - Osteochondroma
  - Enchondroma
  - Benign chondroblastoma
  - Osteoclastoma
  - Bone cyst
  - Non-ossifying fibroma
  - (Calcinosis circumscripta)
- *Malignant bone tumours* include:
  - Osteosarcoma
  - Chondrosarcoma
  - Fibrosarcoma
  - Malignant giant cell tumour
- *Allied malignant lesions* of bone include:
  - Reticulum cell sarcoma
  - Multiple myeloma
  - Haemangiosarcoma
  - Haemopericytoma

Osteosarcoma

Osteosarcomata occur in the large and giant breeds of dog and it is generally the older animal that is affected, but cases have been recorded in dogs as young as 1 year of age. The primary lesion is usually found in the *metaphyseal region* of a long bone and the predeliction sites are the proximal humerus, the distal radius, the distal femur and the proximal tibia, but any part of the skeleton can be affected. The tumours present as hard, often painful, swellings.

Radiology (Morgan, 1972; Gibbs *et al.*, 1984)

The radiographic changes associated with osteosarcoma are illustrated in Figs 51.1, 51.2a and 31.37 and include:

- Destruction of cortical bone
- Growth of the tumour beyond the original confines of the bone
- The development of a tumour mass that may become ossified or contains calcified material.

Radiographically, three types of osteosarcoma are recognised:

(1) *Osteoblastic* or productive.
(2) *Osteoclastic* or lytic, slightly more common than (1).
(3) A *mixture* of osteoblastic and osteoclastic.

Changes which may be observed include:

- Destruction of the normal trabecular architecture with or without adjacent sclerosis
- Thinning of cortical bone
- Breakout through the cortical margins with expansion into surrounding soft tissues
- Pathological fracture
- Periosteal reaction with new bone formation, approximately 50% of osteosarcomata give rise to a 'sun-burst' effect which is a reaction

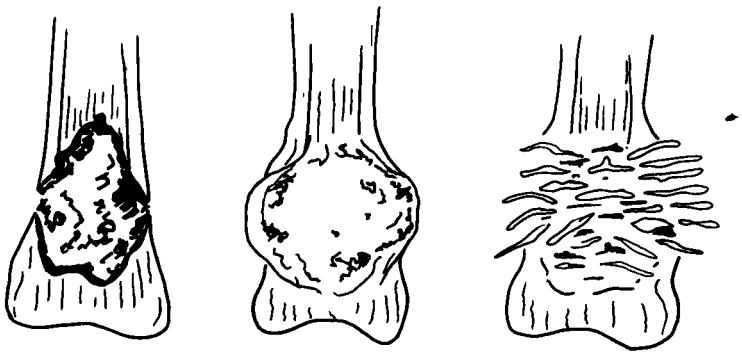

Fig. 51.1 Radiographic features of an osteosarcoma. (a) Destruction of cortical bone. (b) Growth of the tumour beyond the original confines of the bone. (c) Development of a tumour mass that may become ossified or may contain calcified material.

of the periosteum to produce radiating spikules of new bone
- Elevation of periosteum with new bone formation (Codman's triangle)
- Surrounding soft tissue swelling

In general, the subchondral bone is spared and osteosarcomata *seldom invade joint spaces* or adjacent bones. However, changes due to pressure deformity and periosteal reaction may be seen on radiographs. The severity of radiographic changes correlates poorly with the duration or severity of clinical lameness, although those animals with an acute deterioration of signs will often have a pathological fracture present. Lateral views of the chest should be taken with the patient in right and left lateral recumbency to look for pulmonary metastases. Early diagnosis of osteosarcoma may not be easy. If there is doubt then a simple, practical procedure is to repeat the radiographs 4 weeks later when typical changes should be present in positive cases. Benign changes in the distal radius (Figs 51.2b,c), such as those associated with enthesiopathy of the medial collateral ligament (see Chapter 35, Fig. 35.4) are often mistaken for tumours. Benign lesions are fairly localised, show no lysis and are often non-painful, the owner noticing a 'lump' rather than lameness (see Fig. 51.2c).

Further investigation

A fairly accurate provisional diagnosis can usually be made from the history, clinical signs and radiological findings. In order to make a definite diagnosis as to the disease process, differentiating it from the possibility of osteomyelitis, or to determine the exact tumour type, a biopsy is required. It is important to obtain a 'core' biopsy from the margin of the lesion, otherwise reactive or necrotic tissue will be obtained that will not provide a definitive diagnosis.

Scintigraphy is becoming more widely available and may provide some useful information (Lamb, 1987; Lamb *et al.*, 1990). The procedure rarely helps to make a diagnosis of the primary lesion over and above radiography and, in fact, may give false negatives as some bone tumours have only a mild uptake of the tracer (^{99m}technetium methylene diphosphonate). It may be useful, however, in detecting other sites of involvement (i.e. in cases with multicentric osteosarcoma) prior to treatment being considered. It has also been considered to be more accurate than radiography in delineating the tumour margins. Although it appears to overestimate the degree of diaphyseal involvement, this may be safer when considering limb-sparing procedures. It may also be useful in following up cases that have been treated, since bone metastases are not uncommon and scintigraphy is more sensitive in detecting these than radiography.

Treatment and prognosis

Prognosis is usually very poor as osteosarcomata are highly malignant tumours which tend to metastasise to the lungs at an early stage. If these cases are left untreated and the pain is controlled with steroidal or non-steroidal anti-inflammatory

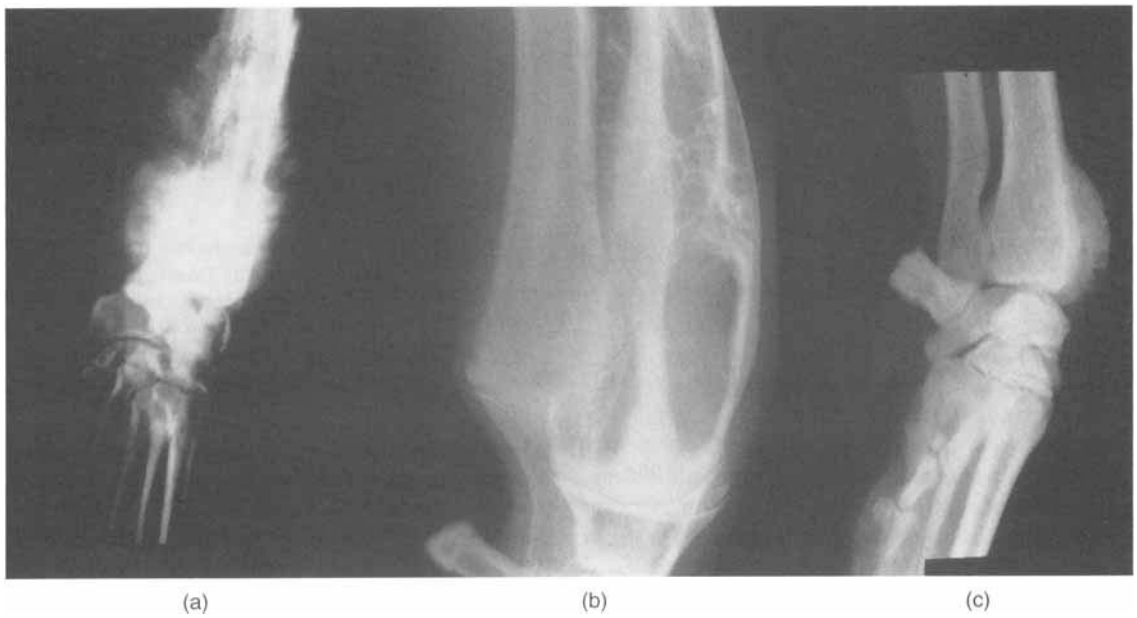

Fig. 51.2 It is important to differentiate osteosarcoma from benign lesions of bone. Radiographs of the distal radius in three separate dogs illustrate: (a) osteosarcoma, (b) benign bone cyst and (c) benign exostosis.

drugs (NSAIDs), then the average time before euthanasia is carried out on humane grounds is usually only a matter of a few weeks to a few months. Chemotherapy, on its own, seems to help control pain but does not improve the survival time. Radiotherapy also relieves pain and the patient may be improved for a period of time before the tumour regrows or metastases develop. Following this form of treatment few dogs live beyond a year.

Amputation

This may be used to remove the lesion in cases without evidence of pulmonary metastases. In a long-term follow-up study of cases with osteosarcomata treated by amputation alone (Spodnick *et al.*, 1992), the average survival time was 19–20 weeks with 11.5% and 2% of the dogs surviving to 1 and 2 years, respectively. Attempts to improve the results of amputation have revolved around adjunctive chemotherapy. Prophylactic radiotherapy of the lung fields appears to confer no advantage with respect to the development of pulmonary metastases.

Chemotherapy

Following limb removal, this does appear to improve survival times and postoperative administration of cisplatin or doxorubicin is currently receiving most attention (Kraegel *et al.*, 1991; Berg *et al.*, 1992; Thompson & Fugent, 1992). Following such protocols the average survival time appears to be about 11–13 months with 1-year survival rates being between 33 and 62%, and 21% surviving to 2 years.

Limb-sparing techniques

In some circumstances it is possible to carry out a block resection of the osteosarcoma but yet preserve the limb. The basic indications for such a procedure are, firstly, that there should be no, or minimal, spread of the tumour from the bone into the surrounding soft tissues. Secondly, there should be no radiographic evidence of lung metastases and, thirdly, it should be possible to retain a functional limb by filling the bone defect with a cortical allograft. Most osteosarcomata arise in the metaphyseal region of long bones and, fol-

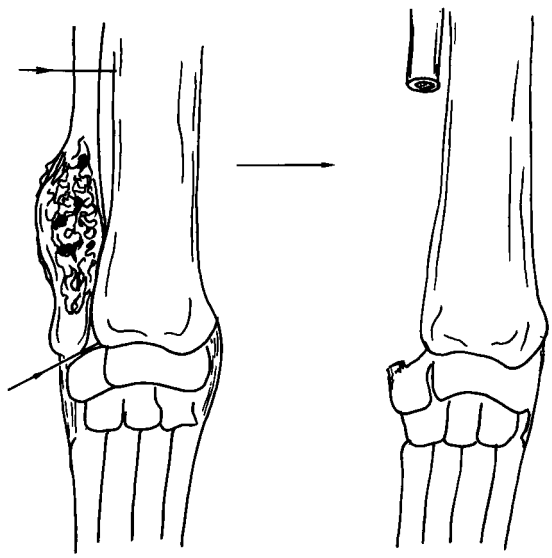

Fig. 51.3 Block resection of an osteosarcoma involving the distal ulna.

lowing block resection, arthrodesis of the adjacent joint is generally necessary. In some locations a graft is not needed following tumour resection, for example osteosarcoma of the distal ulna (see below). In general, the success rate of 'limb-sparing' procedures, in terms of average survival time and 1-year survival rates, is roughly the same as that following amputation, but can be improved by adjunctive chemotherapy, to the same degree as that seen with amputation.

Limb-sparing technique for osteosarcoma involving the distal ulna Occasionally, an osteosarcoma develops in the distal ulna (Fig. 51.3) and block resection of the tumour mass is possible with no effect on limb function. The ulna is transected through healthy bone proximal to the tumour mass and the entire distal ulna is dissected out with the tumour. The procedure does not affect carpal stability or function. Although the procedure is simple and in most operated cases the tumour mass has apparently been completely excised, local recurrence rates are high and in a series of six cases, reported by one of the authors in the previous edition of this book, there was recurrence of the tumour within 6 months in all cases. However, none of these had received adjunctive chemotherapy and so the survival times were similar to those following amputation.

Limb-sparing technique for osteosarcoma involving the distal radius The basic stages of block resection, bone grafting and carpal arthrodesis which are used following removal of a distal radial osteosarcoma can be used in other sites, for example the proximal humerus with shoulder arthrodesis or the distal tibia with arthrodesis of the tibiotarsal joint. The procedure for distal radial osteosarcomata is used most frequently and the technique is illustrated in Fig. 51.4.

Intramedullary cisplatin chemotherapy Some dogs with advanced stage appendicular osteosarcoma may be unable to withstand an amputation or limb-sparing surgery. Intramedullary administration of cisplatin has been proposed as an alternative form of treatment for such cases and in a report on four dogs with advanced stage osteosarcoma the chemotherapy provided effective localised control of the tumour in two of them (Hahn *et al.*, 1996).

Osteosarcoma in the cat

(Turrell & Pool, 1982; Bitetto *et al.*, 1987)

Osteosarcoma in the cat carries a much better prognosis than in the dog. The tumour tends to be locally malignant with little tendency to metastasise. Amputation is the treatment of choice.

Giant cell tumours of bone (osteoclastoma)

Giant cell tumours are very rare in domestic animals. Occasional cases have been described in the cat and dog (Bennett & Duff, 1983). The radiographic appearance of the tumour could be confused with a bone cyst or expanding, non-osteogenic osteosarcoma. The tumour presents as an expansile, well-circumscribed, osteolytic area with thinning of the overlying cortices and no periosteal new bone formation. The osteoclastoma is usually slow growing but the degree of malignancy is variable. Secondary spread to the lungs and kidneys has been reported. Successful treatment of a tumour involving the distal ulna in

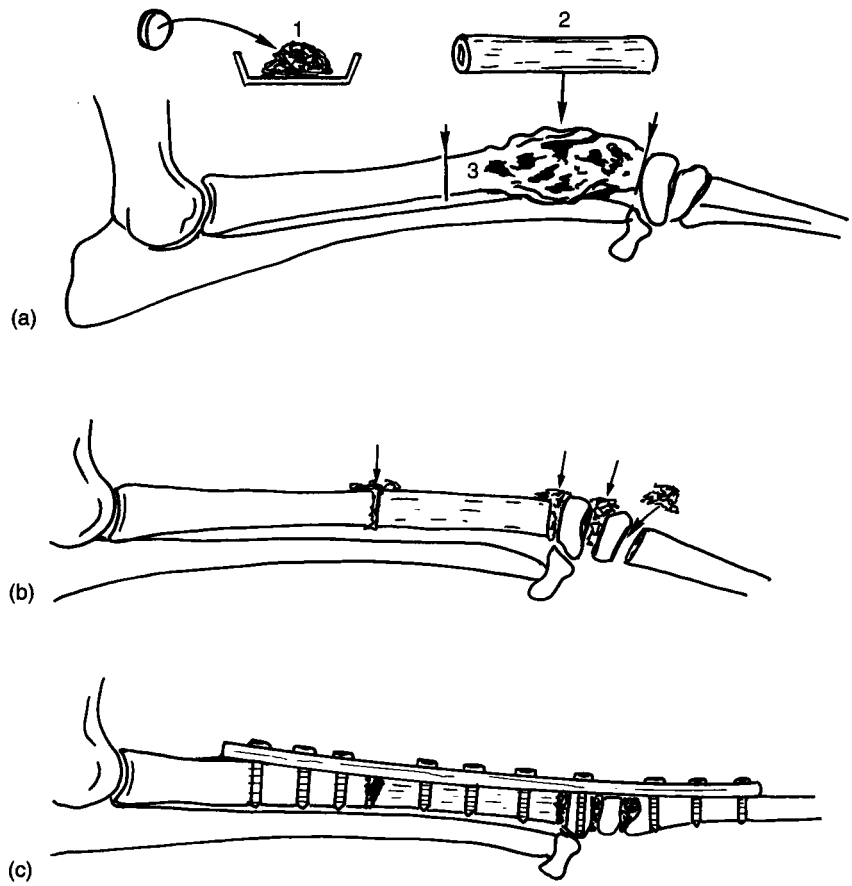

Fig. 51.4 (a) Block resection of an osteosarcoma involving the distal radius: 1 – a cancellous bone graft is taken from the proximal humerus; 2 – allograft of cortical bone; 3 – tumour resection. (b) Articular cartilage is removed from carpal joints. Cancellous bone graft is packed into the joint spaces and around the junction with the cortical allograft. (c) A dynamic compression plate (DCP) is used for fixation.

a cat, by block resection, was reported by Bennett & Duff (1983).

Chondrosarcoma

Chondrosarcomata tend to involve the flat bones in the dog. The major sites of origin have been described as the ribs (29%), nasal cavity (26%) and pelvis (14%) by Brodey *et al.* (1974). Chondrosarcomata do not metastasise as rapidly as osteosarcomata and some cases have been cured following early block resection or radical excision of rib tumours. The radiographic features of chondrosarcomata are illustrated in Fig. 51.5.

Allied malignant lesions of bone

Non-osteogenic malignant tumours of bone

Although osteosarcoma is by far the commonest malignant tumour to affect bone of the appendicular skeleton, a wide variety of neoplasms of other histological types, both primary and secondary, have also been reported. In a series of 34 dogs described by Gibbs *et al.* (1985), the commonest tumour types were fibrosarcoma (9), metastatic tumours (8) and haemangiosarcomata (5). These tumours are termed non-osteogenic because although they may stimulate the production of new bone, it is not formed specifically by neoplastic

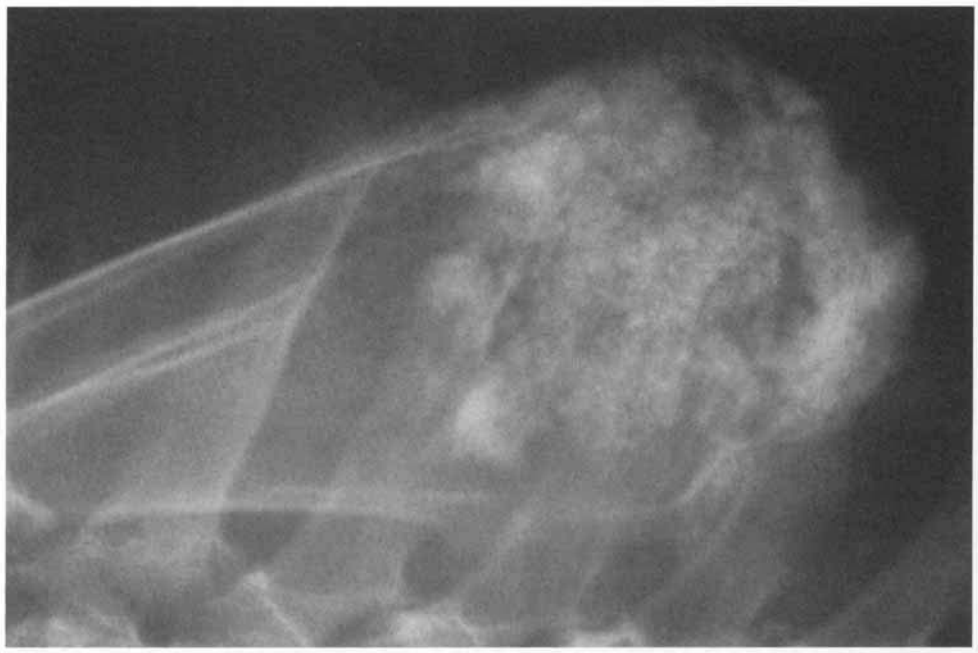

Fig. 51.5 A 12-year-old crossbreed showing chondrosarcoma involving the proximal scapula. The proximal third of the scapula has been destroyed and replaced by dense floccular mineralised tissue. (Reproduced from Gilts, C., Denny, H.R. & Lucke, V.M. (1985) *Journal of Small Animal Practice* **26**, 537–53. With permission from BVA Publications.)

cells. No distinctive radiological features could be related to histological type, but only one demonstrated radiological signs indistinguishable from the characteristic changes of osteosarcoma. Lung metastases were detected radiographically in 15% of the cases. Eight dogs were treated by amputation or surgical excision of the tumour mass and three of these dogs are known to have survived, without recurrence, for more than 2 years.

Fibrosarcoma and haemangiosarcoma

A fibrosarcoma or haemangiosarcoma which arises in the appendicular skeleton is usually treated by amputation. Fibrosarcomata carry a better prognosis than osteosarcomata, but the prognosis for haemangiosarcomata is very guarded because of their potential to metastasise.

Squamous cell carcinoma
(Theilen & Madwell, 1987)

These often arise around the nail bed. There is swelling, ulceration and usually secondary infection. Radiographic examination shows lysis of the third, and sometimes the second, phalanx. They are often misdiagnosed as simple nail bed infections and are treated with antibiotics initially. Radical amputation of the digit is the treatment of choice.

Synovial sarcomata See Chapter 10.

Multiple myeloma

Multiple myeloma (plasma cell myeloma) is a neoplastic proliferation of plasma cells (B lymphocytes), predominantly in the bone marrow (Dobson, 1998). Animals can present with a variety of non-specific clinical signs which are the result of hypergammaglobulinaemia, infiltration of bone marrow with plasma cells and osteolysis of bone. The hypergammaglobulinaemia may also be associated with raised protein levels (so-called Bence-Jones protein) in the animal's urine.

The main radiographic features of bone lesions associated with multiple myeloma are punched out areas of osteolysis in bone resulting in a

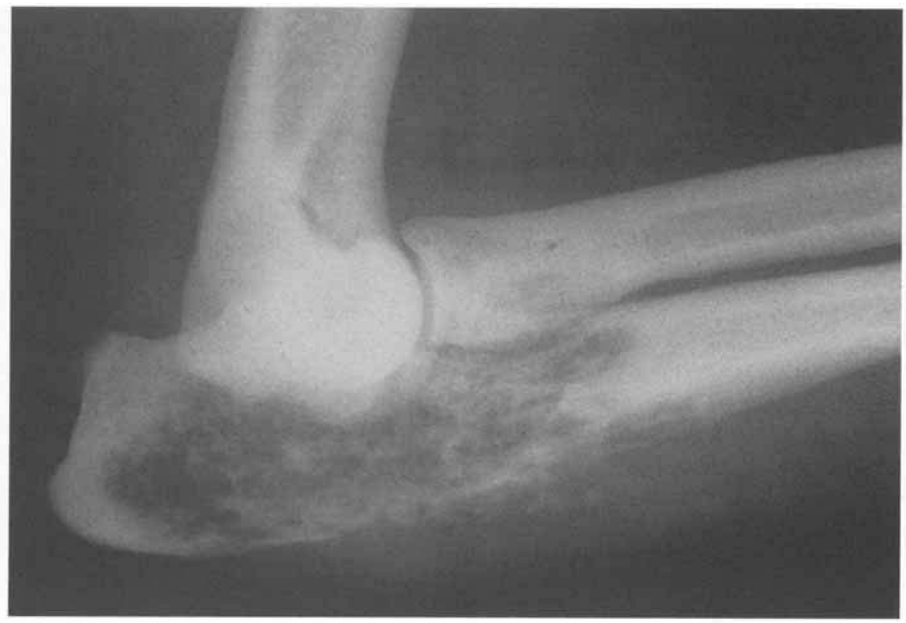

Fig. 51.6 An elbow radiograph of a 6-year-old Collie cross showing extensive punched out areas of bone destruction in the proximal ulna caused by a multiple myeloma.

'moth-eaten' appearance at normal sites of haematopoiesis (Fig. 51.6). Lesions can be either focal or multifocal and can affect any part of the axial or appendicular skeleton. Pain becomes a feature as bone lysis progresses and, eventually, pathological fracture may occur. The clinical effects of the latter will depend on the area of the skeleton affected.

Diagnosis is based on clinical signs, the presence of monoclonal hypergammaglobulinaemia and by the demonstration of malignant plasma cells in the bone marrow. Chemotherapy (immunosuppressive doses of prednisolone +/– additional cytotoxic agents) is the treatment of choice. Over 75% of cases respond to treatment with median survival times of between 12 and 18 months (Matus *et al.*, 1986).

Benign bone tumours

Osteochondroma

Osteochondromata are cartilage-capped exostoses arising from any bone that develops from cartilage. They can be solitary or multiple and, if multiple, the condition is often called osteochondromatosis or multiple cartilaginous exostoses. Multiple cartilaginous exostoses are said to be an inherited defect in the dog (Pool, 1978). Radiographically, lesions appear as dense areas of bone interspersed with radiolucent areas of cartilage. These can be seen in the metaphyseal regions of long bones but can also affect vertebrae, ribs, scapula and pelvis. If lesions cause pain or mechanical interference then surgical removal is indicated, but in most cases this is unnecessary because growth of the tumour stops once the growth plates close.

In the cat, solitary osteochondromata are rare and affect the axial skeleton only (Turrell & Pool, 1982). Multiple lesions are more common and tend to be seen in adult cats arising mainly on the perichondrium of flat bones and occasionally on the appendicular skeleton.

Bone cysts

Bone cysts are occasionally encountered in the dog and the literature on these lesions has been

Fig. 51.7 Bone cyst.

reviewed by Carrig *et al.* (1975). The cyst is generally found in the metaphyseal region of either the distal radius or ulna, femur or tibia. Young dogs of the large breeds are affected. A painless, bony swelling develops and radiographs show a radiolucent lesion with marked thinning of the overlying cortices (Figs 51.2b and 51.7). There is no evidence of an actively destructive process or of any reactive periosteal new bone. Trabeculation within the cysts is minimal. The cyst should be drained, the cavity curetted and then packed with an autogenous cancellous bone graft. The response to surgery is usually good and the cyst decreases in size while the bone cortices thicken.

Calcinosis circumscripta

Calcinosis circumscripta is the name given to a granulomatous lesion which consists of chalky/putty-like masses embedded in fibrous tissue. The condition is seen most often in the German Shepherd Dog but can affect other young large breeds of dog such as the Irish Wolfhound and Afghan Hound. Affected animals are usually under 1 year of age. Lesions may be single or multiple and can be found in a variety of sites, such as the footpad, closely attached to the cervical vertebrae, the thorax, elbow, ischium and even under the tongue. If a calcinosis circumscripta lesion occurs close to a joint then it may restrict movement and could be confused with a malignant bone tumour on radiographic examination. The lesions are benign and tend to stop growing as the dog reaches skeletal maturity. If the lesion is causing lameness, is creating compression of the spinal cord or has become very large then surgical excision is recommended. However, complete excision can be difficult because of the firm soft tissue attachments to the mass. In some sites surgery should be avoided if possible, for example a calcinosis circumscripta lesion in the lower neck where the lesion tends to be firmly adherent to one or more of the caudal cervical vertebrae, and surgical excision does carry risk of damage to the nerve roots of the brachial plexus. The aetiology of calcinosis circumscripta is unknown but it has been described as an inherited, local, metabolic defect of connective tissue (Seawright & Grono, 1961).

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