FELINE ORTHOPAEDICS

SECOND EDITION









HARRY SCOTT, JUAN MARTI AND PHILIP WITTE

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Harry Scott
Juan Marti
Philip Witte

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Preface

More than a decade has passed since the publication of the first edition of this book. At the time the first edition was conceived, the authors felt that there was a growing need for an orthopaedic text devoted specifically to cats. Interest and knowledge in the field of feline orthopaedics has continued apace, and as time passed, it became clear that a second edition was called for, to incorporate the many advances that have been made.

The second edition has two additional authors, Philip Witte and Juan Marti, who bring with them enthusiasm for the discipline and a wealth of experience gained in academia and in private referral practice. Ron McLaughlin has bowed out of his role as co-author because of other commitments. We are very grateful to Ron, without whom publication of the first edition would not have been possible.

The second edition has a similar format to the first but has been updated and expanded, to give both a comprehensive overview of the subject matter, and practical advice when faced with a challenging case. According to the adage, a picture is worth a thousand words; in this vein, many new images have been added, and previous illustrations have been revised and redrawn to improve clarity.

Neuromuscular and spinal disorders have been included, as before, along with other neurological disorders of relevance to the orthopaedic clinician and there is a new chapter, written by guest author Donna Scott, discussing physiotherapy and rehabilitation. We are grateful to the publishers, CRC Press, for the opportunity to write this second edition of the book, especially our Senior Editor, Alice Oven, and our Production Editor, Kyle Meyer.

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1 Introduction to Feline Orthopaedic Surgery

(Chandler & Beale 2002, McCartney & MacDonald 2006, Nolte et al. 2005, Richardson & Thacher 1993, Scott 2005, Shively 1977, Toombs et al. 1985, Toombs & Wallace 1985).

The popularity of cats as pets has increased in recent years and population numbers of owned cats now exceed those of dogs in many developed countries with recent estimates suggesting there are more than 200 million cats in the United States, Canada and Europe. Despite an increasing trend towards keeping cats indoors vehicular trauma remains common particularly in young cats under 2 years of age. In older patients, a combination of increased longevity and greater client expectation has led to the recognition and description of a number of degenerative orthopaedic disorders that were previously overlooked or considered to be of little clinical significance.

It is often stated that cats make good orthopaedic patients because of their small size, undemanding lifestyle and their ability to redistribute weight and protect an injured limb. There is a common misconception that feline fracture repair is simple to perform and healing is not prone to complications. The facetious remark that two cat bone fragments will unite if placed together in the same room is familiar to all orthopaedic surgeons. This casual attitude is not borne out by clinical and experimental studies, which show that the cat is subject to the same range of complications of fracture repair as the dog. The notion that feline orthopaedics is straightforward is all too often testimony to the ability of cats to compensate for or conceal impaired function.

The obvious drawback of small size is the difficulty that this creates for the surgeon in terms of visualization and manipulation of small bones and bone fragments. The small bones of the distal extremities, in particular, are challenging to manipulate and repair surgically. According to the familiar proverb a bad workman blames his tools, but even the most dexterous of surgeons will be frustrated without the correct instruments and implants. Fortunately, the armamentarium of the modern feline orthopaedic surgeon comprises a range of equipment designed for small bone fracture repair that is ideally suited for cats. Historically, there has been a trend towards conservative treatment of many feline orthopaedic injuries, the canine equivalent of which would be treated surgically. While conservative treatment may be appropriate, treatment should always be tailored to the individual case. The goal of the feline orthopaedic surgeon should be to return the patient to optimum function as soon as possible using the treatment with the most predictable outcome and the least morbidity for the patient.

EVALUATION OF THE ORTHOPAEDIC PATIENT:

(Garosi 2012, Kerwin 2012, Moody et al. 2018, Perry 2014).

For the veterinarian who is more familiar with the canine orthopaedic patient successful feline orthopaedic examination requires a change of mindset that incorporates an understanding of the unique qualities of feline behaviour. Feline orthopaedic examination is facilitated by appropriate handling and examination techniques, and modifications to the environment. The goal should be to minimize stress for the patient whilst achieving accurate localization to allow formulation of an appropriate diagnostic plan. Cats are solitary predators with a well-developed flight or fight reaction in response to a perceived threat. The veterinarian has a degree of control over many environmental factors that determine whether a cat will want to run from, or confront, its handler. Desirable environmental modifications for cats include reduction of noise levels, gentle handling, absence of dogs and absence of odours that are perceived as unpleasant. Allocation of a secure consulting room that is used exclusively for cats is strongly recommended. Synthetic facial pheromone analogue should be provided in all areas frequented by cats, whereas the smell of dogs should be eliminated as far as possible. Ideally there should be a separate entrance and waiting area for cats as this will help to reduce stress and anxiety prior to examination. In the examination room provision of equipment such as a non-slip rubber mat and a blanket or thick towel, catnip, treats and a laser pointer may help to make the cat more relaxed.

In comparison with dogs where gait analysis is an important component of the orthopaedic examination most cats cannot be made to walk on a lead and reliance has to made on indirect methods of assessment. The aim should be to gain as much information as possible using indirect techniques and minimal restraint. Gait analysis in cats is challenging because when a cat is fearful or anxious it will stay as close to the ground as possible and will either refuse to move or will attempt to hide. If possible, a cat presented for lameness examination should be allowed to leave its carrier voluntarily and should initially be observed moving around the consulting room as it explores its surroundings. A number of techniques can be used to encourage ambulation including the use of laser pointers, carrying the cat's basket around the room or placing it on the opposite side of the room. Movement can often be assessed by placing the cat on a low stool or chair as most cats will jump down and walk away. A useful way to supplement this examination if a cat is uncooperative is to suggest that the client makes a video of the cat in the home environment.

Hands on examination should always be performed last, and any potentially painful areas or uncomfortable manoeuvres should always be left until the end of the examination. Handling and restraint should be performed using a 'less is more' approach with the cat held in a position of its choosing, still able to move its head, body and limbs, if it chooses to do so. When a cat is placed in a stressful situation its coping mechanism is avoidance of, or retreat from, the source of stress. The handler should use light restraint to allow the cat to feel that it still has the option of retreat otherwise stress levels will escalate and an aggressive response will be elicited as a last resort. For fractious cats, acceptable restraint techniques include a thick towel or blanket and an Elizabethan collar. Gauntlets should be worn to remove a cat from its cage if necessary to avoid injury to the handler. Heavy restraint methods such as scruffing, increase fear, stress and anxiety and are therefore counterproductive as they lead to an increase in the likelihood of defensive aggression. In contrast to dogs where an apparently uncomfortable manoeuvre can usually be repeated to show consistency this approach is poorly tolerated by cats. Once the cooperation of the patient has been lost and an aggressive response has been elicited, such as attempting to bite or scratch the examiner, then the examination should either be abandoned or the cat should be allowed a respite.

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COMPARATIVE FEATURES:

(Conzemius et al. 2003, Corbee et al. 2014, Hill 1977, Hudson & Hamilton 2010, Jaeger et al. 2007, Johnson 2013, Langley-Hobbs 2012, Lund et al. 1999, Mahoney 2012, Maritato et al. 2020, Ness et al. 1996, Newton 1985, Rochlitz 2003a, Rochlitz 2003b, Rochlitz 2004, Schnabl & Bockstahler 2015, Schnabl et al. 2017, Stadig & Bergh 2015, Thrall & Robertson 2016, Wilson et al. 2017).

The feline musculoskeletal system has some unique anatomical and biomechanical features that will be discussed in this section. There are also fundamental behavioural differences between dogs and cats that demand a feline-specific approach to many aspects of diagnosis, treatment and post-operative management of orthopaedic disease. Furthermore, feline physiology places some limitations on post-operative analgesic regimens that are discussed in chapter 3. Optimal outcomes for feline patients can only be achieved by adopting a species-specific approach that is expressed by the familiar adage 'cats are not small dogs.'

In comparison with the dog, cats are less likely to be presented to veterinarians for lameness or intervertebral disc disease. Feline developmental orthopaedic disease is rarely encountered and many of the conditions that are commonly seen in immature dogs, such as elbow dysplasia or aseptic necrosis of the femoral head, have not been reported in the cat. A number of developmental disorders, such as hip dysplasia and patellar luxation, are recognized, although their clinical significance in the cat may differ from that in the dog. In addition, there are some unusual and rare causes of lameness that are either exclusive to the cat such as plasma cell pododermatitis or ulnar neuropathy associated with medial humeral epicondylitis, are breed-specific such as Scottish fold osteochondrodysplasia, or are more commonly reported in the cat than the dog such as hypervitaminosis A.

Traumatic injury, primarily as a result of vehicular trauma, is a common occurrence and musculoskeletal trauma accounts for a large proportion of the disorders seen in the cat. A recent study of a cohort of over a thousand cats up to 12 months of age that were allowed access to the outdoors found that 3.9% had suffered vehicular trauma. In this study the proportion of accidents that were known to have occurred in the dark and the number known to have occurred in daylight were very similar and perhaps counterintuitively did not suggest a trend for accidents to be more likely to occur at night. The same study found that sex and neuter status did not have any association with the risk of vehicular trauma, which contradicts the results of a previous study, in which significantly more males (entire and neutered) than female cats (entire and neutered) were involved. The latter study found that cats are at greatest risk between 7 months and 2 years of age. In this study, which included cats of all ages, 12% of owners reported that their cat had been hit by a vehicle at least once.

Orthopaedic disorders of the feline hindlimb are more prevalent than those of the forelimb with 73% of all fractures involving the hindlimb or pelvis and sacrum in one survey of over 100 cats. Knowledge of the comparative surgical anatomy and physiology of the feline musculoskeletal system is clearly of importance for the feline orthopaedic surgeon. When performing an orthopaedic examination or viewing radiographs, the clinician should be aware of aspects that are unique to the cat. There are many minor and a few major differences between the musculoskeletal system of the cat and the dog. Feline musculoskeletal anatomy predominantly reflects the size, mobility and diet of the cat. For example, the cat's retractile claw mechanism is ideally suited for catching and holding small prey. The feline locomotor system is adapted for agility and for great bursts of speed over short distances. However, cats are poor endurance athletes, tiring quickly and rarely standing for long periods of time without moving. In comparison with the dog, the feline musculoskeletal system tends to be more flexible, skeletal muscles are more strap-like, and intervening connective tissues are looser. Feline limb muscles have a large proportion of type II fast twitch muscle fibres that are adapted for glycolytic metabolism. Fast twitch fibres are associated with the generation of speed and power during muscle contraction but have poor fatigue resistance in comparison with type I fibres and are not well suited to the maintenance of posture. Hence, a palmigrade and plantigrade stance and dorsal protrusion of the scapulae with weight bearing are common signs of muscle weakness in cats.

Cats are relatively small and lightly built and have a higher body surface area to weight ratio than dogs. The corollary of this is that the cat's skeleton is of a lightweight design. Long bones have a large medullary cavity and thin cortices and flat bones, such as the scapula and pelvis, are only a few millimetres thick. Additionally, the long bones are relatively straight and tubular in comparison with those of the dog. Differences in skeletal morphology between breeds are relatively minor and it is, therefore, potentially easier to standardize surgical techniques, instruments and implants for the cat than for the dog.

Other notable features are the greater range of motion in the spine and all of the major limb joints, particularly the shoulder, hip, stifle and tarsus, primarily as a result of an increase in the ability to flex these joints in comparison with the dog. There is also a different distribution of weight bearing between the fore- and hindlimbs when walking. A recent study of cats using pressure platform analysis showed that, as in the dog, limb load forces when walking are greater in the forelimbs than in the hindlimbs. However, in comparison with the dog, the distribution of load at a walk is different, with the hindlimbs of the cat sharing more of the load. The differences in range of motion of the major joints of the appendicular skeleton have been well documented (Table 1.1).

Familiarity with normal feline osteology is essential for the correct interpretation of radiographs. It is often helpful to radiograph the contralateral normal limb and to have feline bones or a skeleton and a set of normal feline radiographs for comparison. An awareness of the differences between the dog and the cat in the arrangement of muscles and nerves, especially around the hip and elbow, is of importance when making a surgical approach to these joints. In addition to specific anatomic differences between the dog and the cat, the relative sizes and dimensions of bones and muscles may differ. For example, the feline ulna and fibula are more substantial than their canine counterparts, the lumbar vertebrae, in particular, are longer and more slender, and the scapula is broader and shorter.

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Table 1.1: Angle Measurements of the Major Joints of the Appendicular Skeleton

Joint		Cat (Degrees)	Dog (Degrees)
Shoulder	Flexion	32	60
	Extension	163	165
Elbow	Flexion	22	35
	Extension	163	165
Carpus	Flexion	22	30
	Extension	198	195
Hip	Flexion	33	50
	Extension	164	160
Stifle	Flexion	24	40
	Extension	164	160
Tarsus	Flexion	21	40
	Extension	167	165

SPECIFIC ANATOMICAL FEATURES AND THEIR SIGNIFICANCE:

Shoulder/Brachium

(Cohen et al. 2012, Langley-Hobbs & Straw 2005).

- The feline scapula is broader craniocaudally than that of the dog (Figure 1.1). The acromion has both hamate and suprahamate processes. The hamate process projects from the distal aspect of the acromion. The suprahamate process (metacromion) is a flat protuberance extending caudally from the distal spine of the scapula 10–20 mm proximal to the acromion. The suprahamate process slightly overhangs the infraspinatus and the surgeon should be aware of this structure when elevating this muscle from the spine of the scapula during surgical approaches to the distal portion of the bone.
- There is a beak-like bony projection called the coracoid process extending craniomedially from the rim of the glenoid. The coracoid process is sufficiently large to be at risk of fracture and should not be confused with an osteophyte or enthesophyte.
- A small ossicle representing the clavicle is consistently present from birth and is usually palpable clinically. It is

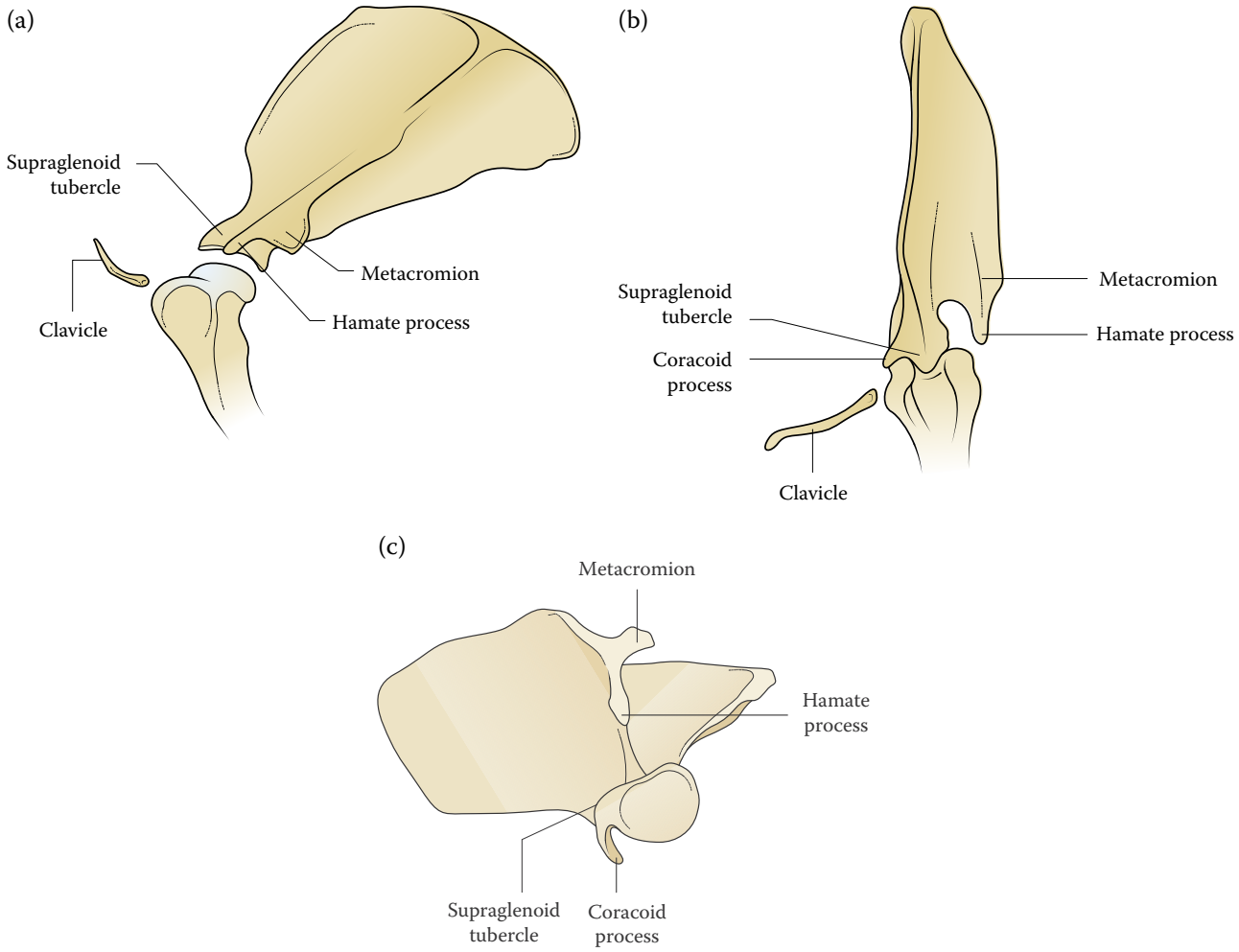


Figure 1.1 Bony anatomy of the scapulohumeral joint. **(a)** Lateral view of the left scapulohumeral joint showing the suprahamate process of the acromion (metacromion), hamate process of the acromion, supraglenoid tubercle and clavicle. **(b)** Cranial view of the left scapulohumeral joint. **(c)** Distal view of the left scapula.

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Figure 1.2 Craniolateral-caudomedial radiographic view of the left distal humerus showing the supracondylar foramen.

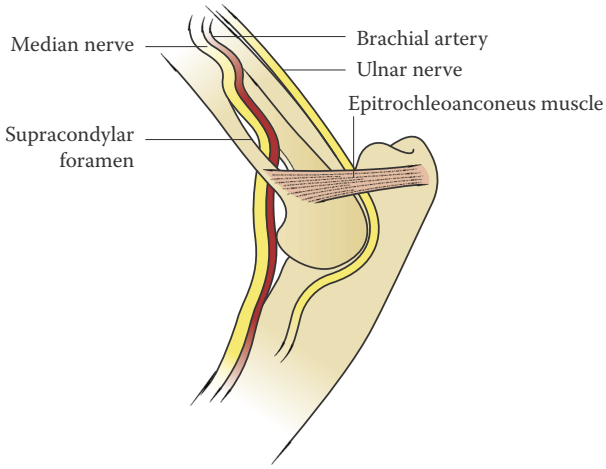


Figure 1.3 Medial aspect of the elbow joint showing the passage of the median nerve and brachial artery through the supracondylar foramen in a caudocranial direction. Note the position of the ulnar nerve deep to the epitrochleoanconeus muscle.

seen radiographically on both lateral and ventrodorsal views as a slender curved cylindrical bone, at least 20 mm long, just cranial to the shoulder joint, and can easily be misdiagnosed as a fracture of the scapula or an oesophageal foreign body. It is a vestigial structure with no bony connections and has no clinical significance.

- The humerus is straighter and more uniform in diameter throughout its length than that of the dog and has an indistinct deltoid tuberosity on the lateral aspect. The humeral diaphysis is penetrated by an oval supracondylar (epicondylar) foramen on the distomedial aspect just proximal to the medial epicondyle (Figure 1.2). The brachial artery and median nerve (but not vein) pass through the foramen from caudal to cranial as they course distally. The surgical approach to this area is complicated by the presence of this foramen and great care should be taken not to damage the neurovascular structures when elevating and retracting the soft tissues about the elbow.
- Similarly, when there is a humeral supracondylar fracture there may be impingement of bone fragments on the median nerve and brachial artery. In this situation, the medial wall of the foramen can be removed using rongeurs to allow the nerve to course in the adjacent soft tissues.
- The nutrient canal of the humerus is present at the junction of the middle and distal third of the diaphysis as in the dog but is medial rather than lateral and therefore is not usually visible radiographically.
- Intramedullary pin insertion for humeral fracture repair is complicated by the presence of the supracondylar foramen. Pins placed distally in the humerus can enter the foramen, damaging the median nerve and brachial artery and penetrating the elbow joint. When possible, intramedullary pins should be positioned 7–9 mm proximal to the medial epicondyle. For distal fractures the pin should be placed normograde from the distal aspect of the medial epicondyle. Normograde pinning using a 1.5 mm bevelled pin avoids damage to the supracondylar foramen whilst maximizing pin purchase in the distal fragment. Retrograde pinning of the distal fragment of humeral fractures even with small pin sizes may damage the articular cartilage of the humeral condyle and is not recommended.
- As in the dog the triceps brachii muscle actually has four heads rather than three (long, lateral, medial and accessory). However, in the cat the lateral head of the triceps is relatively larger than that of the dog and overlies the lateral epicondyle of the humerus. This is of relevance when making a lateral surgical approach to the lateral aspect of the distal humerus and elbow joint.
- The ulnar nerve lies deep to the epitrochleoanconeus muscle on the medial aspect of the distal humerus (Figure 1.3). This muscle has its origin on the medial epicondylar crest and inserts along the caudomedial edge of the olecranon. This is of relevance when making a surgical approach to the medial aspect of the distal humerus and elbow joint. Additionally in cats with medial humeral epicondylitis the ulnar nerve may be compressed between the epitrochleoanconeus muscle and new bone formation within the humeral head of the flexor carpi ulnaris muscle. Medial humeral epicondylitis is discussed in chapter 9.

Elbow/Antebrachium

(Engelke et al. 2010, Preston et al. 2015, Preston et al. 2016, Wallace et al. 2009, Wood et al. 1995).

- Unlike the dog the olecranon fossa is not perforated by a supratrochlear foramen. The distal humeral epiphysis is less prone to fracture because of the absence of this foramen. Consequently, humeral intracondylar Y and T fractures, and lateral and medial condylar fractures are rare in the cat.
- The straighter conformation of the distal humerus and the humeral condyle, and the relatively wide and thick epicondylar crests, in comparison with the dog, may also

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help protect this region of the skeleton from fracture. Furthermore, humeral intracondylar fissure, which is recognized as a predisposing cause of fracture in some breeds of dog, has not been reported in the cat.

- The olecranon ligament, which passes between the craniomedial aspect of the olecranon and the medial border of the olecranon fossa, is well developed in the cat. The ligament is twice the width and a third of the length of its canine counterpart. This may be an evolutionary adaptation to increased pronation and supination.
- A mineralized sesamoid bone in the tendon of origin of the supinator muscle is a normal anatomic variant present in approximately 40% of cats. A non-mineralized sesamoid is generally present in all elbows but will not be visible radiographically. The sesamoid bone is rarely visible on the craniocaudal view of the elbow joint but may be seen on mediolateral radiographs where the sesamoid articulates with the craniolateral aspect of the radial head. It should not be confused with an avulsion fragment, osteophyte or joint mouse. However, the sesamoid bone does become more obvious in arthritic elbow joints, as a result of mineralization, and is often increased in size.
- The radius and the ulna are of a similar diameter and the strong interosseous ligament between the bones that is seen in the dog is lacking in the cat. In contrast to the dog the radius of the cat exhibits marked torsion such that the cranial surface is directed craniolaterally in the proximal part and craniomedially in the distal part. This is of importance when placing a plate on the radius particularly when performing minimally invasive plate osteosynthesis. The proximal approach to the radius is located in a more lateral position than that described for the dog.
- The ulna is able to rotate around the radial head allowing considerable pronation and supination (115°). The shape and orientation of the medial aspect of the humeral condyle (the trochlea) is different from that of the dog leading to medial movement of the radius and ulna during flexion of the elbow joint. Because there is greater mobility between the two bones in comparison with the dog it is important to attempt to preserve motion between the radius and ulna when undertaking orthopaedic procedures in this area. A consequence of this mobility, and the fact that the radius and ulna are of a similar diameter, is that treatment of fractures of the antebrachium by fixation of the radius or ulna alone is less likely to result in adequate stability of the adjacent bone. When managing fractures of the radius and ulna by plating the radius the authors advise supplementary internal fixation of the ulna either using an additional plate or an intramedullary pin.
- The distal ulnar diaphysis, metaphysis, and the styloid process are larger than that of the dog. At the level of the carpus the ulna is more rounded and often has a small radiolucent region. This is a normal variant and should not be confused with an osteolytic lesion.
- The medial and lateral collateral ligaments of the elbow each comprise two parts (ulnar and radial) as in the dog, but there are differences in their course and relative strength which may influence the frequency and type of elbow luxation seen in cats. The ulnar parts are broader than the radial parts and are taut throughout the full range of motion, whereas the radial parts are lax in flexion allowing rotation of the radial head.

Carpus

(Basa et al. 2018, Basa & Johnson 2019).

- The bony anatomy of the carpus is similar to that of the dog with seven carpal bones comprising intermedioradial, ulnar, and accessory carpal bones in the proximal row and numbered carpal bones 1–4 in the distal row. In some cats a sesamoid bone is found on the medial aspect in the tendon of the abductor pollicis longus muscle. This should not be confused with a chip fracture or joint mouse.
- Unlike dogs, cats only have a single short radial (medial) collateral ligament which originates from the medial aspect of the styloid process and inserts on the palmarolateral aspect of the radial carpal bone. Because the ligament runs obliquely in a proximomedial to distolateral direction it not only counteracts valgus stress but also resists palmar luxation of the radial carpal bone. Rupture of the short radial collateral ligament and dorsomedial joint capsule alone can result in antebrachiocarpal subluxation.
- The feline carpus is prevented from overextension by the palmar carpal ligaments as in the dog. Carpal injuries are less common in the cat than the dog and are usually caused by falls from a height resulting in carpal hyperextension and a palmigrade stance or antebrachiocarpal luxation or subluxation. Partial carpal hyperextension is occasionally seen in cats when there is a loss of integrity of the ligaments secondary to chronic overloading as a result of neuromuscular disease such as diabetic neuropathy. Combinations of carpal fractures and ligamentous injury are most common, whereas isolated ligament injury or fracture is unusual.

Pelvis

- The pelvis is more rectangular in shape than that of the dog and the ilium is proportionally longer with respect to the ischium. The pelvic floor is V shaped in the transverse plane.

Hip/Thigh

(Ablin & Gambardella 1991, Beck et al. 2005, Culvenor et al. 1996, Gibson et al. 2008).

- In contrast to the dog, the ligament of the head of the femur (round ligament) is purported to provide a significant proportion of the blood supply to the femoral capital epiphysis. This may explain why aseptic necrosis of the femoral head (Legg–Calvé–Perthes disease), commonly seen in small breed dogs, has not been reported in the cat. The blood supply via the round ligament will also help protect the epiphysis from vascular compromise after capital physeal separation. Although these fractures should be treated promptly, the prognosis in cats is generally good, even in cases in which repair is delayed.
- In common with other feline long bones the femur is straighter than that of the dog. The morphological features are similar to those described in the dog except for the fact that the lesser trochanter is more prominent and the greater trochanter is distal to the femoral head. The biomechanical properties of the feline femur have been studied.
- The arrangement of the muscles around the hip is similar to that of the dog, but there are some differences. The gluteal muscles are relatively larger in the cat and the caudofemoralis muscle, which is not present in the dog, is inter-

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posed between the superficial gluteal and biceps femoris muscles. The tensor fascia lata and the vastus lateralis muscles are broader in cats. The craniolateral approach to the feline hip is most commonly used. When making this approach a longer incision is necessary through the insertion of the tensor fascia lata muscle than in the dog. Similarly, the vastus lateralis muscle requires more subperiosteal elevation to gain adequate exposure of the femoral neck. The caudofemoralis muscle should not be mistaken for the superficial gluteal muscle. The articularis coxae muscle is well developed and covers the joint capsule in the cat.

- The sartorius muscle is undivided in the cat, unlike in the dog where it has cranial and caudal bellies. This muscle may be encountered when making a lateral approach to the femoral shaft.
- The sacrotuberous ligament, which in the dog extends between the caudolateral angle of the sacrum and the lateral part of the tuber ischium, is absent in the cat.
- A kinematic study has shown that the principal load-bearing areas of the feline acetabulum are the central and caudal thirds rather than the cranial third. This is of relevance to the treatment of acetabular fractures. The frequent recommendation that simple caudal acetabular fractures should be treated conservatively should be reconsidered in the cat.

Stifle/Crus

(Arnbjerg & Heje 1993, Freire et al. 2010, Leijon et al. 2017, McCarthy & Wood 1989, Scavelli & Schrader 1987, Voss et al. 2017)

- In the dog the gastrocnemius tendon inserts on the dorsoproximal surface of the tuber calcis. In the cat three caudal crus muscles converge to form the equivalent tendon. These are the gastrocnemius, the plantaris which is sandwiched between the two heads of the gastrocnemius and the soleus which arises from the proximal fibula. The combined action of these muscles accounts, in part, for the cat's great leaping ability. The gastrocnemius and soleus muscles together are sometimes referred to as the triceps surae.
- The patella is elongated and tapered distally in comparison with that of the dog. Radiographically this should not be confused with a distal pole enthesophyte.
- Small mineralized densities are commonly seen in the cranial aspect of the stifle joint on lateral radiographs. In one study radiographic meniscal mineralization was identified in 37% of the stifles of client-owned cats. In the same study 60% of 57 cadaver stifles were found to have meniscal mineralization which was always located in the cranial horn of the medial meniscus. The clinical significance is uncertain as mineralization may be either a cause or a consequence of osteoarthritis.
- There is variable mineralization of the medial fabella and the popliteal sesamoid bone. Unmineralized sesamoid bones will not be visualized on radiographs.
- In contrast to that in the dog, the cranial cruciate ligament is larger than the caudal cruciate ligament. This may be one of the factors contributing to the relative infrequency of cranial cruciate ligament rupture in the cat.
- The fibula is relatively larger than that of the dog and there is a wider interosseous space between the fibula and tibia.

Tarsus

(Kulendra & Arthurs 2014; Nicholson et al. 2012).

- The medial and lateral collateral ligaments of the talocrural joint are each comprised of two short ligaments; there is no long component spanning the whole tarsus as seen in dogs. The medial collateral ligament comprises oblique tibiotalar and straight talocentral parts, and the lateral collateral ligament is comprised of calcaneofibular and talofibular components. In addition to the ligaments the tendons of the fibularis brevis and tibialis caudalis muscles also provide some passive support as they each have a firm attachment to their respective malleolus as they course to their insertion distally. This is of relevance when performing prosthetic collateral ligament replacement for the treatment of talocrural luxation or subluxation. Additionally when performing a lateral approach to the calcaneus it is especially important to avoid disrupting the short part of the lateral collateral ligament as this is the primary lateral stabilizer of the joint.
- The feline fibula articulates more distally with the lateral tibial condyle than in the dog and the lateral malleolus has a smooth lateral protuberance not present in the dog.

Manus and Pes

(Hamelin et al. 2017).

- Digit 1 (dewclaw) is always absent in the hindlimb.
- Polydactyly is more common in the cat than the dog (Figure 1.4). The supernumerary digits may be just the first digit on the hindlimbs or may be multiple extra digits on all four limbs. The additional vestigial digits are usually preaxial (medial). Claws of these digits may need clipping if they grow too long and impinge on the pads, but the condition is otherwise asymptomatic.



Figure 1.4 Forepaw of a cat with polydactyly. The cat hadsupernumerary digits on all four limbs.

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- There is a smooth protuberance on the proximal aspect of the fifth metatarsal bone that can be seen on dorsoplamar radiographs of the tarsus that is not present in dogs.
- The distal ends of the middle phalanges of digits 2 through 5 of both the manus and the pes are oblique and bevelled so that the distal phalanx can pivot on the middle phalanx. At rest the distal interphalangeal joint is maintained in hyperextension by the paired dorsal elastic ligaments that attach to the middle phalanx. Protracting the claws is an active process that requires contraction of the deep digital flexor muscles to overcome the pull of the dorsal elastic ligaments. Injuries to the distal forelimb can disrupt this mechanism.

Spine

(Burger et al. 2004, Kot et al. 1994, Singh 2017, Shkorbatova et al. 2019).

- The nuchal ligament, which in the dog extends from the spinous process of the axis to the tip of the first thoracic spinous process, is absent in the cat. The surgeon should be aware of this when making a dorsal approach to the cervical spine. The lack of ligamentous support accounts for the propensity for neck ventroflexion with neuromuscular disorders in the cat.
- There are no unique anatomical features to the vertebrae of the cat. In comparison with the dog feline cervical vertebrae are more rectangular and the trabecular bone pattern is coarser. The transverse processes of the caudal cervical vertebrae are smaller and the transverse processes of C6 are angled more laterally rather than projecting ventrally as in the dog. In the thoracic spine, typically the ribs do not extend as far dorsally as they do in the dog. The latter feature allows for better visualization of the intervertebral foramina and the accessory processes of the caudal thoracic vertebrae. The vertebral bodies of the lumbar spine are elongated in comparison with those of the dog and the transverse processes are longer.
- Unlike dogs, which have a notch in the cranial edge of the sacral wing as a consistent anatomic feature, the notch is only present in 34% of cats and its location is variable. This is of importance when performing lag screw fixation of sacroiliac luxation. In contrast to dogs the sacral wing notch in cats is not a useful landmark for screw placement.
- In a proportion of cats, a smooth region of bone is seen on the caudoventral aspect of the sacrum as a normal anatomical variant. This should not be misinterpreted as callus from a previous fracture.
- The spine of the cat is more flexible than that of the dog. Part of this increased flexibility can be accounted for by the fact that the intervertebral discs contribute a larger proportion to the total length of the vertebral column (20% in the cat and 15%–17% in the dog). The intervertebral discs of the cat are prone to degeneration in the same way as in non-chondrodystrophoid breeds of dog. Affected cats are frequently asymptomatic; the reasons for this have not yet been determined.
- The spinal cord is longer relative to the vertebral canal in the cat than in the dog. In the past, there has been some uncertainty about the exact termination of the spinal cord. Various levels from the caudal border of the seventh lumbar vertebra (L7) to the caudal border of the sacrum have been given by different authors. It is likely that the cranial limit is accurate for adults and the more caudal one for young kittens. In adult cats there is some individual and breed variation in the termination of the spinal cord, but typically the rootlet areas of segments L6, L7 and S1 are located within or below the L5 vertebral body and the segments S2 and S3 are located within or caudal to the L6 vertebral body. There is closer correlation between spinal cord segments and vertebrae in the cat than the dog, and a focal lesion will therefore tend to affect fewer segments in the cat.

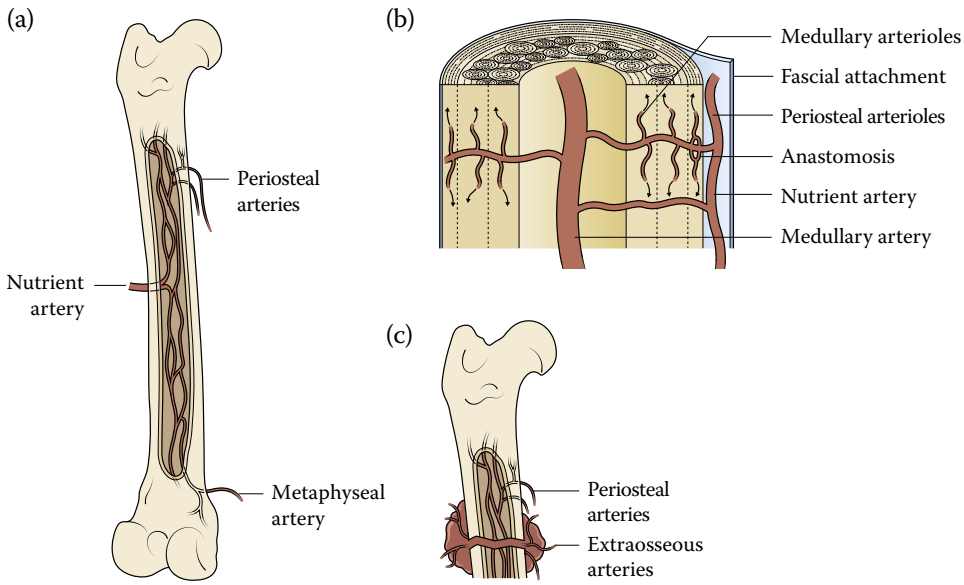


Figure 1.5 Arterial blood supply to bone. **(a)** Normal mature bone. **(b)** A section of the diaphysis of normal mature bone. **(c)** Fractured bone showing the development of an extraosseous blood supply.

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BLOOD SUPPLY TO BONE:

(Emery & Murakami 1965, Rhinelander 1968, Rhinelander 1974, Wilson 1991, Wilson 2002).

Arterial blood supply to a typical long bone is provided by an afferent vascular system, comprised primarily of the nutrient artery and the proximal and distal metaphyseal arteries (Figure 1.5). Small periosteal arterioles also provide blood to the outer cortical layer in the region of fascial attachments and muscle insertions. The intermediate vascular system of compact bone comprises Haversian canals, Volkmann canals and minute canaliculi creating a vascular lattice to provide nutrients to the osteocytes. Venous drainage occurs via the efferent vascular system from the periosteal surface of the bone. This centrifugal blood flow from the medullary cavity to the periosteal surface is disrupted to varying degrees when fractures occur. The afferent blood vessels then undergo hypertrophy and increase in number to reestablish the medullary blood supply quickly. Additionally, a new and temporary extraosseous blood supply develops from the surrounding soft tissues to vascularize the developing periosteal callus, bone fragments and cortex. Revascularization and bone healing are delayed by severe trauma to the bone and soft tissues, overzealous soft tissue dissection and improper tissue handling during surgery, poor reduction of bone fragments, inadequate stabilization of the fracture and infection.

BONE GROWTH:

The bones of the appendicular skeleton grow in length by endochondral ossification and in width by intramembranous ossification. Endochondral ossification is characterized by osteoblastic conversion of a cartilaginous model to bone. Longitudinal bone growth occurs at the physes, which remain open until bone growth has ceased. Functional physeal closure occurs when there is anatomic continuity of osseous tissue between the epiphysis and the diaphysis. Closure of the physis and cessation of bone growth occur before fusion can be documented radiographically.

Growth Plates

(Smith 1969).

In contrast to those in the dog, the growth plates of kittens are simple and flat. Cats reach puberty between the age of 8.5 and 10 months with weight and season being determining factors. Physeal closure occurs between 4 and 9 months of age with the majority being closed by 9 months of age. However, open physes may still be present in entire cats up to 2 years of age with the physes that close last most prone to delayed closure.

Physeal closure times have been documented in entire cats based on the radiographic examination of 37 kittens and anatomic dissection of eight kittens. There are individual differences in closure times and there is no precise sequence of physeal closure. However, physes can be classified on the basis of closure times into first, middle and last groups (Figure 1.6 and Table 1.2).

Growth Plate Disturbances and Angular Limb Deformity

(De Lima Dantas et al. 2016, Swanson et al. 2012, Voss & Lieskovsky 2006).

Traumatic injury may cause disturbance in the growth of the physis leading to a reduction or cessation of growth and bone shortening. Disturbances of physeal growth may also be associated with angular limb deformity if there is asynchronous growth in paired bones, such as the radius and ulna, or if the disturbance in a single physis is asymmetric (Figure 1.7). In comparison with the dog,

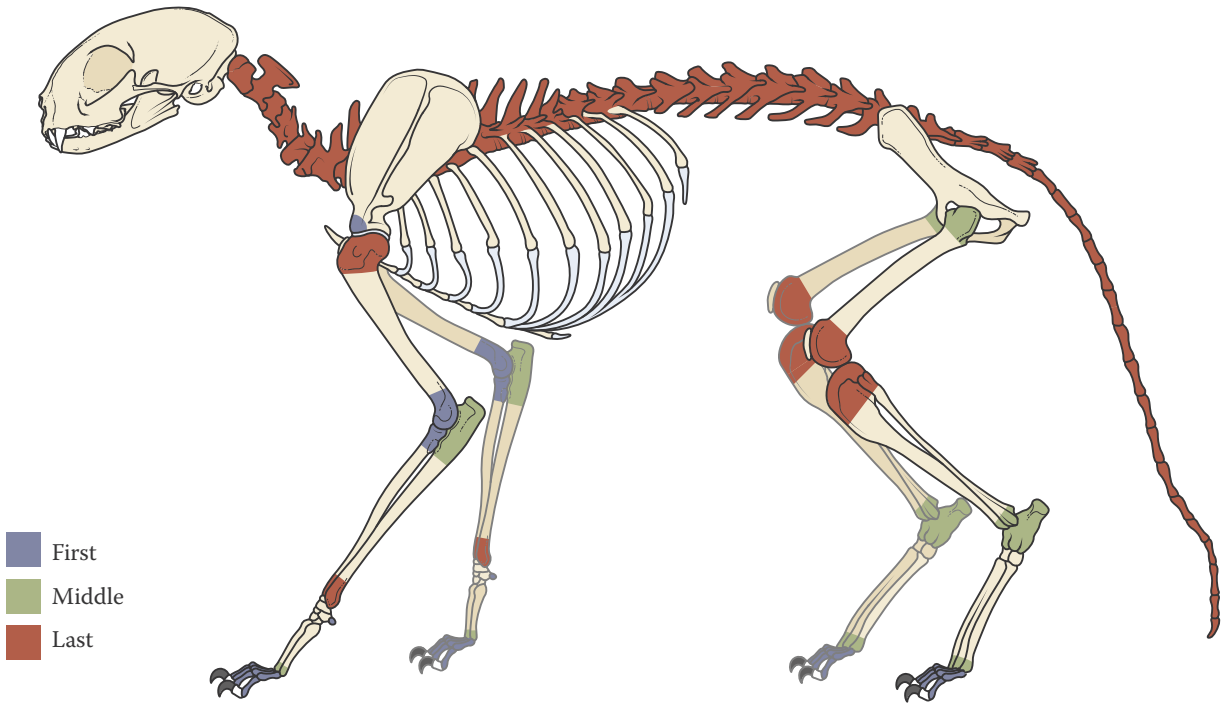


Figure 1.6 Cat skeleton showing closure times of the growth plates of clinical significance. Physeal closure times are classified as first, middle and last.

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Table 1.2: Physeal Closure Times in the Cat

First (4–8 Months)	Middle (8–14 Months)	Last (14–24 Months)
Scapular tuberosity	Proximal ulna	Vertebrae
Distal humerus	Metacarpals	Proximal humerus
Proximal radius	Proximal femur	Distal radius
Accessory carpal bone	Distal tibia	Distal ulna
Phalanges I & II	Distal fibula	Distal femur
	Fibular tarsal bone	Proximal tibia
	Metatarsals	Proximal fibula

angular limb deformity is uncommon in the cat, partly because of the smaller stature and reduced overall growth potential. An important additional factor is the linear shape of the distal ulnar physis of the cat in contrast to the conical ulnar physis of the dog. The shape of the canine physis increases its surface area, facilitating rapid growth, but it also predisposes the germinal cells to compression injury. In young cats the physes represent significant structural weak links in the skeleton and Salter–Harris fractures are a common occurrence in this age group.

Angular limb deformity may also be the result of malunion fractures, congenital patellar luxation and other congenital malformations. Congenital medial patellar luxation displaces the quadriceps mechanism medially leading to abnormal loading of the distal femoral and proximal tibial physes. Deformities associated with the condition include distal femoral varus and proximal tibial valgus. Radiographic methods for measurement of normal anatomical radial angles and femoral and tibial joint reference angles have recently been described. Mean joint reference and limb alignment angles can be used both for diagnosing angular limb deformities and for planning corrective osteotomies.



Figure 1.7 Proximal tibial and distal femoral growth plate disturbance. CT 3D volume rendering of the pelvis and hindlimbs of a cat with left medial patellar luxation associated with mild distal femoral varus and moderate to severe proximal tibial valgus deformity.

The Effect of Neutering on Growth Plates

(Houlton & McGlennon 1992, May et al. 1991, Perry et al. 2014, Root et al. 1997).

A number of studies have reported on the effect of neutering on physeal closure times in cats, but the results of some studies are contradictory. It is generally accepted that physeal closure in neutered male cats is delayed compared with that in entire males owing to the effect of gonadal steroids. In one study the relationship between castration at about the time of puberty (28 weeks of age) and the timing of physeal closure was studied in male cats. In another study the time of closure was compared in neutered and entire male and female cats. In both of these studies, physeal closure was found to be delayed in castrated male cats with some individuals having open physes beyond 4 years of age.

One of these studies looked at the effect of neutering male and female cats at 7 weeks and 7 months of age on the proximal radial (first group), and distal radial (last group) physes and eventual radial length. This study found that castration had no effect on the timing of proximal radial physeal closure. Castrated cats, however, had delayed closure of the distal radial growth plate irrespective of the age at castration. In female cats, closure of the proximal radial physis was delayed in cats neutered at 7 weeks of age but not at 7 months of age, whereas closure of the distal radial physis was delayed in both groups of neutered female cats. Final radial length was found to be significantly increased in both groups of neutered male and female cats compared with entire cats. A major limitation in this study was the small numbers in each group.

In a more recent study pelvic and femoral radiographs of 808 cats were examined for the effect of neutering on physeal closure. Physeal closure was found to occur later in neutered male cats than entire male cats for the greater trochanter, distal femur and tibial tuberosity. No effect was shown for the proximal tibial or proximal femoral physes, nor for any physis when comparing entire female with neutered female cats which contradicts the findings of the previous study.

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In conclusion there is no doubt that neutering of immature male cats will delay physeal closure and predispose to Salter–Harris fractures, and those physes that close after puberty (those in the middle and last groups) are more at risk of injury than those closing before puberty.

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2 Examination and Diagnostic Tools

INTRODUCTION:

(Perry 2015a).

Lameness is the predominant clinical sign in cats with orthopaedic disorders, but because of the cat's independent lifestyle and ability to compensate, this is less readily observed in comparison with the dog. Therefore, investigation of lameness in the cat has much in common with the dog, but there are important differences. Many of the conditions encountered are similar, but their significance may differ and there are also causes of lameness that are rarely or never seen in dogs. Also, the feline gait is generally more difficult to evaluate than in dogs, since they are not used to being walked on the lead and do not reliably respond to commands to facilitate the orthopaedic exam. As usual, the goal of the examination should be to formulate and prioritize a list of differential diagnoses; these differential diagnoses are then investigated with the aim of achieving a specific diagnosis. The more common musculoskeletal causes of lameness are listed in Table 2.1. Generally speaking, a very severe or a non-weight bearing lameness in a cat warrants a suspicion of a fracture, septic arthritis, abscess, or a neoplastic process (Figure 2.1).

Lameness or limb weakness may also be caused by a variety of conditions unrelated to the limb bones and joints. These include conditions of the spine and peripheral nerves, such as vertebral fracture and, rarely, brachial plexus avulsion or neoplasia. These conditions should be suspected if evidence of neurologic dysfunction is detected on examination or if no orthopaedic abnormalities are found to justify the lameness. Degenerative joint disease, the most common joint pathology, and cause of lameness and decrease of quality of life in the cat can be challenging to diagnose.

SIGNALMENT:

(Ness et al. 1996, Rochlitz 2003, Scarlett & Donoghue 1998).

The age, sex, breed and weight of the cat may give an indication of the type of conditions most likely to be encountered. For example, male (entire and neutered) cats less than 2 years of age are more likely to suffer from fractures and luxations as a result of traumatic incidents. Degenerative conditions such as osteoarthritis or cruciate disease are relatively less common and are typically seen in older cats. Joint and bone neoplasia is also more common in the older cat, although lymphoma is an important exception. There are a few nontraumatic conditions that have a sex predisposition, such as periosteal proliferative polyarthritis, but gender is generally not a major factor. Cat bites and traumatic conditions are more common in young cats, probably because of their territorial aggression and lack of experience of natural and man-made hazards.

With the exception of certain inherited and congenital disorders, there are few conditions that are regularly seen more frequently in specific breeds. However, breed predispositions can also be an important indicator of the existing pathology in the cat, since specific pathologies are especially prevalent in certain breeds (such as sacrococcygeal hypoplasia in Manx cats, capital physeal fractures in young Maine Coon cats, different skeletal abnormalities in the Scottish folds, etc). Non-pedigree cats are, generally speaking, affected much less by non-traumatic orthopaedic conditions.

The morphology of pedigree cats also differs very little from non-pedigree cats so, in contrast to dogs, conformation rarely plays a significant role in musculoskeletal disorders. Body weight may predispose cats to clinical signs of musculoskeletal disease in general and osteoarthritis in particular. One study found that overweight cats were 2.8 times more likely and obese cats were 5.4 times more likely to suffer from lameness visible to owners.

HISTORY:

History taking is the component of the investigation that is most likely to be neglected in practice because it is perceived as time-consuming. However, the history often provides relevant information concerning the cause of musculoskeletal disease and should always precede physical examination of the patient. The astute feline clinician should be

Table 2.1: Differential Diagnosis of Lameness

Diagnosis	Skeletally immature <2 years	Skeletally mature >2 years
Cat bites	++	+
Fractures	++ (often physeal)	+
Traumatic luxations	+	++ (hip>hock>stifle>elbow>carpus>shoulder)
Avulsion injuries (tibial tuberosity, etc.)	++	–
Septic arthritis	++	+
Calicivirus arthritis	++	–
Periosteal proliferative polyarthritis	++	++
Other immune-mediated polyarthritides		++
Neoplasia (bone, periarticular soft tissue)	+	++
Osteoarthritis	–	++ (elbow & shoulder most common)
Hip dysplasia	++	+
Patellar luxation	++	+
Cranial cruciate ligament rupture	–	++

– to ++ indicates an increasing relative frequency of occurrence

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Figure 2.1 Neoplastic conditions are more likely causes of lameness in older cats. This radiograph shows a soft tissue sarcoma in the hindlimb of a 14-year-old domestic short hair (Courtesy of M. Ruiz).

wary of a number of pitfalls. Cats are small and light and have an independent lifestyle, so they are very good at disguising mild to moderate musculoskeletal impairment. It is not unusual for the duration of clinical signs to be underestimated by the client because early signs have been overlooked. An apparently acute condition may, therefore, already be chronic at the time of first presentation. Another difficulty is determining whether conditions that are identified during subsequent investigations are incidental when there has been no history of clinical signs, for example, an osteoarthritic joint identified radiographically. Additional problems are commonly encountered with cats that have suffered unobserved traumatic incidents. Cats are frequently presented, having been missing for several days, with an apparent orthopaedic disorder and no history to guide the clinician as to the cause. It should be noted that some nontraumatic conditions, such as ischaemic neuromyopathy, might mimic unobserved traumatic injuries, such as pelvic fracture or spinal fracture/luxation.

PHYSICAL EXAMINATION:

A general physical examination should precede the specific orthopaedic component of the examination. The examination may detect evidence of concurrent or intercurrent disease. This may provide clues to the orthopaedic disorder; for example, cats with certain types of immune-mediated polyarthritis may have disease of other body systems. Intercurrent disease may influence the prognosis and have a bearing on the administration of anaesthetics and intravenous fluids; a degree of renal insufficiency is not uncommon in geriatric cats, for example. Cats with unobserved trauma that have been missing are generally cachectic and the immediate concern is fluid and caloric resuscitation. Cats with traumatic orthopaedic injuries frequently have concurrent injuries involving other body systems, for example, thoracic injuries (diaphragmatic rupture, pneumothorax, rib fractures, etc.) and abdominal injuries (urinary bladder rupture, etc.). The investigation and treatment of the trauma patient are discussed more fully in Chapter 3.

ORTHOPAEDIC EXAMINATION:

(Jaeger 2007, Langley-Hobbs, 2012, Perry 2014a, 2014b, Perry 2015a, 2015b, 2015c).

The specific orthopaedic examination is performed in a standard fashion regardless of species. The main difficulties are the lack of patient compliance for gait analysis and potential difficulties with the restraint of a fractious and painful cat.

Observation of the gait should be made by allowing the patient to walk unrestrained around an examination room. This can sometimes only be achieved by allowing the cat to settle first in a quiet environment or by observation through a window from outside the room. It is sometimes helpful to ask the owner to video the cat at home. Some cats will tolerate, if used to it at home, to be walked by the owner using a lead connected to a chest harness, either inside the hospital or in a quiet area outside the office. The cat's posture and stance should also be noted at this stage of the examination. An initial evaluation is generally performed with the cat in a standing position and using minimal restraint. Careful and methodical palpation of the muscles and joints of the limbs should be performed first, usually starting proximally and working distally. It is helpful to compare left and right limbs for overall symmetry, evidence of pain or tenderness, muscle atrophy and joint thickening or swelling. Muscle atrophy is most easily detected where there are large muscle masses adjacent to bony prominences, such as the blade of the scapula and the greater trochanter. Joint thickening and swelling cannot usually be appreciated in the hip and shoulder because of overlying muscle masses. Synovial effusion is easier to detect with the joint bearing weight, but, in general, palpable joint effusion is less common in comparison with the dog.

A more focused orthopaedic evaluation is generally performed with the cat gently restrained in lateral recumbency by an assistant or standing if better tolerated. It is preferable to commence the examination with the healthy limbs, in order to accustom the cat to being handled and to minimize the chances of the patient becoming fractious, by leaving manipulation of the potential painful areas to the last phase of the exam. A good orthopaedic examination should include careful palpation and manipulation of the joints of all of the limbs, and this can be done by beginning distally starting with the phalanges and working proximally or from proximal to distal, depending on the examiner's preference. The range of joint motion is assessed by putting the joints through a full range of flexion/extension and abduction/adduction, as appropriate, while observing the cat for any evidence of discomfort. Joint angles and their ranges of motion can also be assessed using goniometry, in both sedated and conscious patients.

Joint stability is assessed at this time in lateral to medial and cranial to caudal planes. Any evidence of joint effusion,

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periarticular thickening and crepitus is noted. Specific manipulations such as the cranial draw sign and tibial compression tests are identical to those performed in the dog and are applicable to the cat. Others, such as the Ortolani sign, are less frequently performed but can also be performed as part of coxofemoral hip evaluation. These manoeuvres are best performed under sedation or general anaesthesia and are discussed more fully with the relevant disorders. Long bones and the overlying soft tissue structures are also palpated along their length for irregularity or discomfort. A manoeuvre that provokes an apparent pain response should be repeatable to ensure its significance, and, if pain is expected, this manoeuvre should be reserved for the final part of the examination so as to ensure a cooperative patient. Therefore, a comprehensive examination should be performed on all four limbs and spine before focusing attention on the affected limb.

The spine should also be examined thoroughly in every patient, especially if there are no obvious findings in the orthopaedic exam or there is any indication of weakness or neurological disease. The spine of the cat is surprisingly flexible and the examiner is encouraged to get accustomed to the normal feline spine to avoid misdiagnosis. Evaluation of the cervical spine should begin by testing the patient's neck dorsally (extension), ventrally (flexion) and lateral excursions to both sides to assess flexibility and possible discomfort. Direct palpation of the spine, by applying pressure to the dorsal spinous processes or, more laterally, to the epaxial musculature to the thoracic spine, thoracolumbar area and lumbar spine can elicit a painful reaction if there is spinal pathology. The thoracolumbar junction and lumbar spine can also be extended by lifting the patient's hind quarters and applying direct pressure to the area of interest. Further caudally, pain in the lumbosacral junction can be tested by direct palpation in a dorsoventral direction or producing its extension by simultaneous lifting of the pelvis.

DIAGNOSTIC TOOLS:

Following the orthopaedic examination a decision is made about treatment or further investigation. Further investigations should be performed to confirm a tentative diagnosis or to differentiate between the different conditions in a short list of differential diagnoses. In many cases, the diagnosis may be obvious and is easily achieved with standard diagnostic modalities such as radiographs in fracture cases, without the need for further work up; however, other modalities such as ultrasound, computed tomography, magnetic resonance, or others may be necessary to achieve a final diagnosis in more complicated or perhaps cases with subtle or unusual presentations. In other cases, where the condition is mild or where there are financial constraints, it may be preferable to start a therapeutic medical trial and reassess in the light of the response to treatment.

Radiography

Radiography is still the most commonly used ancillary diagnostic tool in the investigation of orthopaedic disease, especially in first opinion practice. The use of radiography as an aid in the investigation of joint disease is discussed in Chapter 7. Radiographs should be interpreted in the light of the clinical findings and should not be used as a substitute for a correctly performed clinical examination. Radiography is often used to confirm a suspected clinical diagnosis and occasionally to differentiate between two or more diseases with similar clinical signs. Radiography is used in traumatic injuries to assess the location and extent of fractures and to assist with the planning of surgical repair. For diaphyseal fractures, especially if there is comminution, radiographs of the contralateral bone are obtained to assist further with fracture planning and bone repair. Radiographs of the thorax and abdomen are warranted for cats that have suffered severe trauma, irrespective of whether there are clinical signs of a respiratory or abdominal disorder. Feline radiography is best performed with the patient heavily sedated or under a general anaesthetic to eliminate movement and allow optimum positioning, and, indeed, this may be a legal requirement in some countries. Gentle manual restraint, with or without light sedation, may be possible in compromised patients, in countries where this is allowed.

Changes are often subtle and the bones are small, so high-quality radiographs are essential. If traditional radiography is still used, fine detail screens should be used and human mammography film/screen combinations are preferred for the distal extremities. Radiographs should be correctly collimated to minimize scatter and should be obtained in two orthogonal planes (i.e. at right angles to each other). The addition of a radiographic ball marker of known size, placed at the same height as the affected bone, is useful to obtain reliable measurements of the true size of bone. This is particularly useful when deciding the type and size of implants to use, for fracture repair, for example, and helps in pre-operative decision-making. With digital radiography being more commonplace in veterinary clinics, the speed of image acquisition and availability, their detail and resolution, and the ability to manipulate the images to appreciate their subtleties have been greatly enhanced when compared to traditional, screen-film radiography. Radiographic interpretation should be thorough and complete so that each structure is assessed fully (i.e. changes in number, position, size, shape, margin, opacity, internal architecture and function). In selected cases, where interpretation of radiographic changes may be difficult, the opinion of a diagnostic imaging specialist will prove very useful. The pattern of changes for individual conditions is discussed in the relevant chapters.

Synoviocentesis and Joint Fluid Analysis

(Pacchiana et al. 2003, Pacchiana et al. 2004).

Synoviocentesis and analysis of synovial fluid is an underutilized procedure in feline medicine. Analysis is particularly useful for differentiating between degenerative and inflammatory causes of arthritis and in determining whether an inflammatory arthritis is infective or immune mediated. General anaesthesia or heavy sedation is required for synoviocentesis in the cat. It is usually convenient to perform radiography first and then immediately tap affected joints in the light of the radiographic interpretation. This also prevents the unlikely introduction of air in the joint from complicating the initial interpretation of the radiograph and missing a slight synovial effusion due to a previous arthrocentesis. A small area should be clipped and aseptically prepared at the site of the puncture (Figure 2.2), but it is not necessary to use surgical drapes or gloves, although hand cleaning with antiseptics is advised. If direct palpation of the bony landmarks around puncture sites is necessary due to the small size of these joints, associated swelling and so forth, sterile gloves are then recommended. A 3-ml syringe and a 25-gauge hypodermic needle of sufficient length to penetrate the soft tissues (usually up to 25 mm) are used to aspirate fluid

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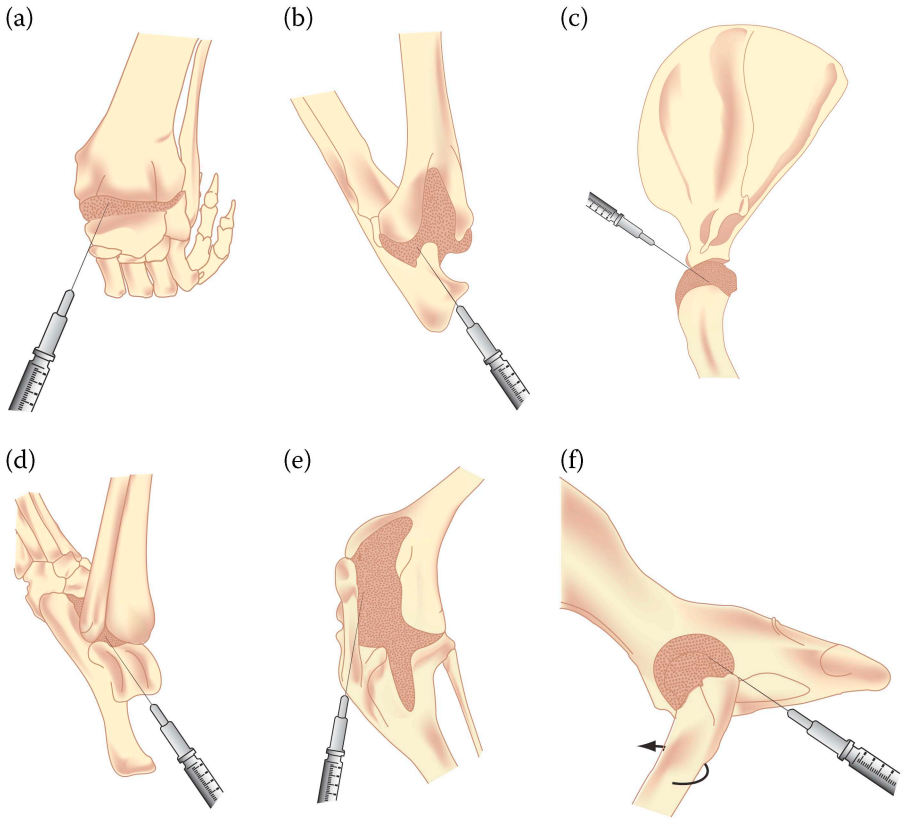


Figure 2.2 Six sites for synoviocentesis. **(a)** Carpus. The antebrachio-carpal joint is located with thumbnail pressure with the joint flexed. The needle is introduced slightly to one side of the median plane. **(b)** Elbow. The joint is held in flexion and the needle is introduced from the caudolateral aspect between the lateral humeral epicondyle and the olecranon. **(c)** Shoulder. Gentle traction is placed on the limb to open up the joint space. The needle is introduced into the scapulohumeral joint from the craniolateral aspect just distal to the acromion process. **(d)** Hock. The joint is held in flexion and the needle is introduced into the tarsocrural joint from the caudolateral aspect between the lateral malleolus and the lateral trochlear ridge of the talus. **(e)** Stifle. The joint is held partially flexed and the needle is introduced just medial or lateral to the mid-portion of the straight patellar ligament and directed proximally. **(f)** Hip. The femur is held partially abducted and rotated outwards. The needle is introduced into the coxofemoral joint just caudal to the greater trochanter and directed cranially at an angle of approximately 45°.

from the joint. The volume of fluid obtained from normal joints is typically less than 0.25 ml for the shoulder, elbow, stifle and hip joints and less than 0.1 ml from the carpal and tarsal joints. Difficulty may be encountered introducing the needle into the hip joint and multiple attempts are sometimes necessary (ultrasound guidance may be advisable to increase accuracy and limit needle-related damage). Synovial fluid from cats with inflammatory arthritis may clot on standing (a positive fibrinogen clot test). Clotting is prevented by placing samples that are to be submitted for cytologic examination in blood tubes containing ethylenediamine tetra-acetic acid (EDTA) as an anticoagulant. Cells in synovial fluid degenerate rapidly, and cytologic examination should preferably be performed within 4 hours. For submission to an external laboratory, cooling to 4°C (39°F) provides preservation for up to 24 hours.

Samples for bacterial culture should not be placed in EDTA because it inhibits bacterial growth. Since the volume of synovial fluid samples obtained from cats is small, proper sample handling and correct choice of tests are critical (Table 2.2)

The features of synovial fluid that are most frequently evaluated are as follows:

- Physical characteristics (volume, colour, turbidity and viscosity)
- Cytologic examination (total nucleated cell count and differential cell count)
- Bacterial culture.

Physical Characteristics

The physical characteristics of the fluid are noted at the time of collection. Normal synovial fluid should be of low volume, should appear clear and colourless or straw-coloured, and should have a high viscosity. Aspiration of more than 0.25 ml of fluid, even from the larger joints, is abnormal and indicates joint effusion. Turbidity indicates increased white or red cell counts and a yellow discoloration, or xanthochromia, is almost always due to the presence of haem pigments derived from previous haemorrhage. Red discoloration may indicate recent haemorrhage but is more likely due to contamination during sampling, which can be seen as a swirl of blood entering the syringe during the procedure. A sample iatrogenically contaminated with blood will render cell counts that are

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Table 2.2: Synovial Fluid Analysis

	Normal joint	Osteoarthritis	Immune-mediated arthritis	Bacterial infective arthritis
Volume (ml)	0–0.25	0–0.50	0–2.0	0.25–1.0
Colour	Clear/pale yellow	Yellow	Yellow-white +/− blood tinged	White +/− blood tinged
Clarity	Transparent	Transparent	Transparent or opaque	Opaque
Viscosity	Very high	High	Low/very low	Very low
Mucin clot	Good	Good–fair	Fair–poor	Poor
Spontaneous clot	None	Sometimes	Often	Often
White cells (cells/μl)	<1000	1000–5000	5000–9000	5000–100000
Neutrophils	<5%	<10%	10–95%	>95%
Mononuclear cells	>95%	>90%	5–90%	<5%
Protein (g/l [g/dl])	20.0–25.0 [2.0–2.5]	20.0–30.0 [2.0–3.0]	25.0–50.0 [2.5–5.0]	>40.0 [>4.0]

not representative of the existing joint pathology so, if possible, the contaminated portion of the sample should not be allowed to enter the syringe or should be discarded before submission. A subjective assessment of viscosity is made by performing the string test, whereby synovial fluid is allowed to drip slowly from the syringe (onto a slide) or a small amount (a drop or less) is placed between the finger and thumb, which are then slowly drawn apart. Normal fluid will produce a string that is at least 20–40 mm in length. Care needs to be exercised so that not too much sample is used for this, if this prevents the clinician from doing other more important tests. Viscosity is a measure of the hyaluronic acid content and is reduced in inflammatory arthropathies. The viscosity will also be reduced owing to a dilution effect if there is a substantial joint effusion, irrespective of the aetiology.

Cytologic Examination

If sample size is limited, cytologic examination should take priority. A haemocytometer is used because fluid volume is generally insufficient for Coulter counter evaluation. Cytology provides estimates of the total nucleated and differential cell counts, thus allowing the important distinction to be made between inflammatory and noninflammatory disease. Normal feline synovial fluid is less cellular than canine synovial fluid. If necessary, examination can be performed on a stained smear prepared from a single drop of synovial fluid. A smear is made on a glass microscope slide at the time of collection, air-dried and then stained with a Romanovsky's stain, such as Wright's, before examination. Many commercial laboratories request submission of a freshly prepared unstained air-dried smear in addition to a sample of fluid in EDTA.

Bacterial Culture

(Montgomery et al. 1989, Proot et al. 2015, Scharf et al. 2015).

This is used to isolate the causative organism and guide antibiotic therapy in suspected cases of septic arthritis and should include both aerobic and anaerobic culture. Isolation of bacteria from synovial fluid is inconsistent and a negative culture does not rule out septic arthritis. The likelihood of a positive culture can be significantly increased either by submission of a sample of synovial membrane or by inoculation of the synovial fluid into sterile blood culture medium before submission to the laboratory. Liquid blood culture medium reduces the in vitro phagocytosis of bacteria by leucocytes and has a dilution effect on bacterial inhibitors. Polymerase chain reaction (PCR) as an additional test to diagnose septic arthritis has not been reported in the cat, as it has in the dog.

Other Tests

Numerous other tests have been used to analyse synovial fluid in humans, but their value has not been demonstrated in cats. Common additional tests include measurement of glucose, which is decreased in inflammatory disease, and protein, which is elevated in inflammatory disease. Both of these tests require concurrent submission of serum so that the two levels can be compared. Measurement of an elevated lactate acid in synovial fluid from septic joints has not been reported for clinical use in the cat, as it has in the dog (Proot JL, personal communication).

Pressure Platform Gait Analysis

(Addison & Clements 2017, Conzemius et al. 2003, Corbee et al. 2014, Gruen et al. 2017, Lascelles et al. 2007, Schnabl & Bockstahler 2015, Schnabl-Feichter et al. 2017, Stadig & Bergh 2015, Stadig et al. 2016).

Despite the inherent difficulties of walking cats in a guided, controlled, repeatable manner, pressure platform gait analysis offers an objective noninvasive method for evaluating limb function in cats. Given the limitations of gait evaluation in cats, this test will provide the only means of objective measurement of limb function and is very useful for evaluation of treatment outcomes in a non-biased manner. The technique has been described experimentally in normal cats and used to describe different aspects of the feline gait.

Pressure platform gait analysis has been used to evaluate long-term limb function after onychectomy, for assessment and comparison of the efficacy of analgesic drugs after onychectomy and for cats with cranial cruciate ligament-deficient stifles. Also, kinetic gait analysis based on a force plate has been used to evaluate somatosensory abnormalities associated with increased sensory sensitivity in untrained cats with osteoarthritis.

Fluoroscopy

Although not generally available, fluoroscopy has been used to aid in the intraoperative placement of implants,

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such as pins and screws, especially when located close to joints and to assess fracture alignment. The use of more portable, intraoperative C-arms has become more widespread in larger referral centres. Other uses of fluoroscopy include contrast studies (myelography, arthrography, etc) and location of radiopaque foreign bodies, although other imaging modalities, such as ultrasound or computed tomography, may prove more useful in these situations.

Scintigraphy

(Morandi 2016).

Bone scintigraphy is a functional imaging technique which involves the injection of a radioisotope (Technetium-99, in two variations of labelled diphosphonates: 99 m Tc-MDP or technetium methylene diphosphonate and 99 m Tc-HDP, or technetium hydroxydiphosphonate), which emits gamma radiation. This radioisotope is tagged to a pharmaceutical and the combination (called a radiopharmaceutical) mimics physiological process in the patient, concentrating in specific areas where the radioisotope activity can be detected from the outside. The agent is injected intravenously and an image is created using a gamma camera showing areas of increased bone turnover. Therefore, this technique is useful to detect areas of increased bony activity, typically in investigation of an obscure lameness, but also in staging of bone neoplasia by detecting other bone lesions somewhere in the body, in determining the size and extent of a bony lesion, in fracture healing, detection of stress or occult fractures or in aiding differentiation of the extent of bony versus soft tissue lesions.

The study is typically divided into an initial vascular phase, a soft tissue (blood pool) phase in which the radiopharmaceutical equilibrates with the vascular spaces, and a bone phase. Non-specific accumulations of the compound in affected soft tissues with increased blood supply or expansion of the extracellular fluid spaces will help diagnose acute soft tissue lesions.

Scintigraphy is rarely performed in cats because of concerns about radiation safety, expense and lack of availability. The technique is a very sensitive indicator of bone lesions but is nonspecific and, therefore, an area of increased activity can be due to many different normal and disease processes. In cases of investigation of an obscure lameness, a more specific bone imaging technique, such as radiographs or computed tomography would follow, to further characterize the bone lesion.

A variation of this technique, extremity lymphoscintigraphy, can be used to confirm the diagnosis of lymphoedema, by using an inert colloid compound called 99 m Tc-SC (sulphur colloid). This compound is injected in the toe webs and enters the lymphatic channels, moving proximally aided by muscle contractions and osmotic gradients, also accumulating in local lymph nodes. The rate of absorption of the compound from the injection site, its time-dependent movement along the limb, degree of visualization of lymphatic vessels and nodes etc, can be used to diagnose lymphatic drainage-related conditions. The spatial resolution of musculoskeletal scintigraphy is poor, so its anatomical localization capabilities are limited using planar gamma cameras. The use of SPECT (Single-photon emission computed tomography) with several cameras can provide 3D reconstruction images and, therefore, improve the image-acquisition capabilities. This technology is rarely used in veterinary medicine.

Sonography

(Anson et al. 2013, Gnudi & Bertoni 2001, Kramer et al. 1997, Kramer et al. 2001, Kramer 2011, Mahoney 2012, Risselada et al. 2005, Samii et al. 1999, Scharf et al. 2004).

Sonography is a complementary technique to radiology, in the examination of the musculo-skeletal system. It has limited application for the investigation of orthopaedic disease because of the inability of ultrasound to penetrate osseous tissues but it has become more widely used in small animals in the past few years, as an aid to investigate soft tissue injuries, such as tendon, ligament or muscle and the surface of the bone. Specifically, it can be used to evaluate any soft tissue swelling around joints and some intraarticular structures, around bones, in or around muscle, tendon or ligamentous structures, any thickening or swelling of tendon sheaths, defect in muscles/tendons, adhesions between these structures, etc. Peripheral nervous structures can also be evaluated with sonography.

Although CT and MRI overcome the limitations of ultrasound to penetrate bone tissue, these are more expensive tests which usually require sedation or anaesthesia. Moreover, ultrasound is capable of providing a more dynamic examination of the existing pathology, as the diseased structures can be manipulated during the study. This is particularly useful in limb pathology. The evaluation of tissue morphology can be complemented with Doppler ultrasonography, which provides more functional information regarding vascularity. The lack of radiation exposure to the patient, operator and assistants, its low cost, portability and short examination times in a typically conscious patient make it a very desirable tool in small animal orthopaedics and rehabilitation clinics.

In general, ultrasonography is limited in the evaluation of joints due to the strong acoustic shadowing produced by bone, although the subchondral bone and superficial bony features such as osteophytes can be assessed. Hyaline cartilage is anechoic and cannot be imaged with this modality. Sonography is useful for assessment of soft tissue structures such as synovial membrane and joint capsule, in addition to some intraarticular structures, such as tendons (biceps brachii tendon in the shoulder, long digital extensor in the stifle) and associated sheaths. Deeper intraarticular structures, such as menisci or cruciate ligaments can also be evaluated, as it has been reported in the dog. Additionally, this modality can be used to image tendons not surrounded by bone, such as the common calcaneal (Achilles tendon) or the patellar tendon.

Sonographic evaluation of muscles and musculotendinous junctions is readily achieved. Muscle imaging can diagnose conditions such as traumatic ruptures, muscle fibrosis/atrophy, presence of intramuscular abscesses or neoplasia, tendon ruptures (complete or partial), tendon sheath effusion or thickening (Figure 2.3).

Sonographic assessment of secondary fracture healing of the long bones has been described and a preliminary report described the use of ultrasound-guided needle biopsy for the diagnosis of bone lesions. Also, power Doppler has been

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Figure 2.3 Sonography of musculo-skeletal structures is a readily available and inexpensive diagnostic modality, both for muscular and tendinous lesions, as in this case with a suspected gastrocnemius tendon lesion.



Figure 2.4 Computed tomography, an excellent diagnostic tool for bone disease, has become very available in clinical practice in recent years.

used to reveal the neo-vascularisation at the formation of the callus.

Scanning of musculoskeletal soft tissue structures, specifically muscle, ligament and tendons relies on thorough knowledge of their normal anatomy, which in turn depends on the familiarity of the operator with these structures, in different species. Examining the uninjured contralateral region or limb also provides a very useful comparison, particularly when evaluating structures that may be relatively unfamiliar. Injuries of these soft tissues can then be identified and their progression in time, as the tissues heal or fail to do so, can be assessed. The skills to reliably diagnose soft tissue lesions using ultrasonography are very specialized and imaging professionals require a reasonable caseload to become proficient at distinguishing normal from diseased structures and at interpreting the significance of their findings. Also, the small size of some of these structures, such as some ligaments and tendons, complicates their sonographic evaluation and may require specialized transducers. This is likely to restrict the use of musculoskeletal ultrasound to referral centres with a reasonable caseload or to radiologists with a special interest in this discipline.

Advanced Imaging Techniques

Magnetic Resonance Imaging (MRI)

(Banfield & Morrison 2000, Benamou et al. 2012, De Rycke et al. 2015, Sage & Gavin 2016).

The use of magnetic resonance imaging (MR) and computed tomography (CT) has been restricted in the past by their expense and lack of availability (Figure 2.4). They are being used increasingly in the investigation of spinal and joint disorders in dogs, but there are few reports of their use in cats. The small size of cats means that imaging of feline structures requires the use of sophisticated, expensive equipment that is capable of obtaining very high definition images. Magnetic resonance imaging is most useful for lesions involving the spinal cord and nerve roots and for

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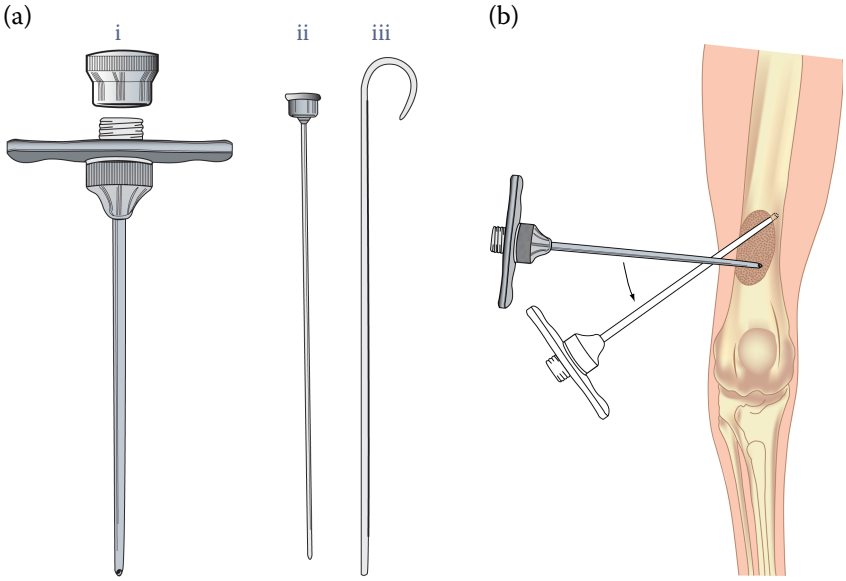


Figure 2.5 Jamshidi-type bone marrow biopsy needle. **(a)** (i) Cannula and screw-on cap. The cannula has a tapered point to retain the specimen in the needle. (ii) Pointed stylet for use when the needle is advanced through the soft tissues. (iii) Probe used to expel the specimen from the base of the needle. **(b)** After the stylet has been removed, the needle is advanced through the bone using a twisting motion until the opposite cortex is reached. The needle is then withdrawn and the specimen expelled. The procedure is repeated using the same skin incision, with the cannula directed towards the periphery of the lesion.

soft tissue structures of joints, whereas CT is most useful for osseous lesions.

Musculoskeletal soft tissue disorders can be investigated with magnetic resonance. Different muscle pathologies can be distinguished by their image pattern, such as oedema, infiltration of fat, mass lesions and so forth with significant overlap possible between patterns. Muscle atrophy and volume can also be assessed. Problems in larger ligaments and tendons can also be diagnosed and distinguished from normal structures, with their smooth margins, uniform diameter and low signal. Acute, more vascular responses (oedema, inflammation) in recent tendon injuries or disruption of the tendon tissue itself, scarring, fat replacement or calcification can be identified in more chronic cases. Inflammation or haemorrhage can be seen at the periosteal level if the injury occurs at the tendon insertion (enthesis). Changes in smaller tendons and ligaments are more difficult to appreciate given the limits of the image resolution. For the investigation of intra and periarticular joint structures, magnetic resonance will provide superior resolution for soft tissues when compared to CT although bony detail will be better assessed on CT. The use of MRI-arthrography, the use of intraarticular contrast to enhance the study, is also possible and has been described in dogs but not in cats.

Still, the main use of the MRI technology in the feline patient is the investigation of intracranial disease and, secondly, spinal disease. The main disadvantages of this modality include its expense, limited availability of equipment sophisticated enough to produce good images in feline patients, and the need for anaesthesia.

Computed Tomography

(Tivers 2008).

Computed tomography is much more sensitive in evaluation of bone than soft tissue structures so, when used for assessment of the musculoskeletal system, this modality is excellent for detection of early or inconclusive bony changes that may be too subtle or not visible at all on radiographs, (e.g., bony lysis in neoplastic disease or non-displaced fractures or fissures not seen on radiographs). Also, this modality is very useful in evaluating the extent of destruction in a bony lesion, such as neoplastic process. This is usually aided by the ability to perform three-dimensional reconstructions and on screen, 'virtual' disarticulations of the affected joint. This spatial resolution provides another interesting use of CT, namely, the planning of fracture fixation (long bones, pelvis, spine) so that the surgeon can evaluate the fracture in a three-dimensional setting and the appropriate surgical implants can be chosen.

Unfortunately, evaluation of soft tissue structures such as muscle, tendon, ligament, synovium is limited even with the most sophisticated equipment. As in MRI, the use of CT-arthrography can be used to help delineate the intraarticular soft tissue structures.

Bone Biopsy

(Ehrhart et al. 1998).

Bone biopsy is indicated in the definitive diagnosis of proliferative and lytic bone lesions. Different types of bone tumours and osteomyelitis caused by bacteria or fungi may have a similar radiographic appearance and can only be

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differentiated on the basis of histopathologic examination. Biopsy is performed less frequently in the cat than the dog because of the relative infrequency of osteosarcoma and other types of bone neoplasia. The Jamshidi-type bone marrow biopsy needle is suitable for obtaining bone biopsies from cats (Figure 2.5). Use of the needle only requires one small incision and the size of the sample obtained is small, thus minimizing the risk of iatrogenic fracture. Jamshidi needles are available in single-use or reusable versions. The single-use needle can be reused a number of times but requires ethylene oxide sterilization, whereas the reusable needle can be autoclaved. An accurate histopathologic diagnosis can be obtained in approximately 90% of cases if an experienced histopathologist examines the sample. The technique has been described in detail. Briefly, the site is clipped and prepared, and a small incision is made in the skin over the centre of the lesion. The biopsy tract may be contaminated with tumour cells and should be positioned so that it can be included in the excised tissue if definitive surgical treatment is to be performed subsequently. The needle is advanced through the soft tissues, with the stylet in place, until bone cortex is contacted. The stylet is removed, and the needle is advanced until it contacts the transcortex. The needle is removed and the specimen is pushed out using a probe, which is inserted into the opening at the tip of the needle. The process is repeated through the same incision, so that at least two samples are obtained, one from the centre and the other in the transition zone. Diagnostic accuracy can be increased if the bone is radiographed post-operatively to confirm correct selection of the biopsy site.

Additional Tests

The extent of routine preoperative blood testing is controversial but a biochemistry panel, haematology, electrolytes and urinalysis are recommended for any cat >5 years old undergoing a lengthy orthopaedic procedure (>60–90 minutes). Blood testing is particularly indicated for geriatric cats, and for cats that have a preexisting medical condition or evidence of intercurrent disease on clinical examination. Young cats (<5 years old) presented for elective orthopaedic procedures require only a packed cell volume (PCV), total protein and urine-specific gravity. Feline leukaemia virus (FeLV) and feline immunodeficiency virus (FIV) tests may also be performed routinely or if they are specifically indicated.

Other tests performed on the basis of the clinical signs and other findings include coronavirus, antinuclear antibody, rheumatoid factor, creatine kinase, toxoplasma serology and measurement of acetylcholine receptor antibody and molecular techniques. The significance of these tests and the indications for them are discussed in the relevant chapters.

Definitive diagnosis of many spinal and neuromuscular disorders requires specialized testing such as cerebrospinal fluid analysis or histopathological examination of muscle and nerve tissue. These procedures and the processing of specimens are discussed in Chapters 15 and 17. For histopathological examination the laboratory should be consulted beforehand for advice about the correct selection and processing of samples.

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3 Management of The Orthopaedic Trauma Patient

INTRODUCTION:

(Griffon et al. 1994, Kolata et al. 1974, Kraje et al. 2000, Lux et al. 2018, Vnuk et al. 2004).

A logical approach to the assessment and management of the traumatized cat will help maximize survival and increase the success rate of subsequent surgery. The major cause of feline orthopaedic injury is vehicular trauma; other aetiologies include falls from heights, animal fights and gunshot wounds. Multiple orthopaedic injuries are not uncommon, and there are frequently injuries to multiple body systems (Table 3.1)

Thoracic injuries, in particular, are often seen in cats with fractures and represent a common cause of death following trauma. Cats falling from heights (feline high-rise syndrome) can suffer severe thoracic trauma in association with fractures. In one study, thoracic trauma was diagnosed in 33% of cases, with a higher incidence of severe injuries and thoracic trauma in falls from the seventh or higher stories.

In several studies of cats with traumatic fractures, a large percentage of patients had radiographic evidence of thoracic trauma, the most common injuries being lung contusion and pneumothorax. Pleural effusion, diaphragmatic, sternal fracture, rib fractures, lung lobe atelectasis and pneumomediastinum need to also be considered.

Traumatic rib fractures in cats, although usually of little clinical significance in their own right, should alert the clinician to the likely presence of other more severe intrathoracic and orthopaedic injuries. It is essential that the patient is adequately stabilized and properly evaluated for concurrent injury before performing orthopaedic surgical intervention. With the exceptions of fractures involving the skull and spine and open fractures, there are few instances where orthopaedic injuries constitute an emergency. Since many concurrent injuries produce minimal clinical signs on first presentation, they can easily be overlooked in favour of the more obvious orthopaedic injuries. Diagnosis and treatment of thoracic injury is especially important prior to anaesthetic administration. In one study, 40% of cats showed no clinical signs suggestive of thoracic injury despite having abnormalities detected radiographically.

Table 3.1: Common Thoracic and Abdominal Injuries Seen in Cats with Traumatic Fractures

Thoracic injuries	Abdominal injuries
Diaphragmatic rupture	Abdominal rupture
Pulmonary contusion	Bladder rupture or avulsion
Pneumothorax	Ureteral avulsion
Pneumomediastinum	Perforation/tear of colon or rectum
Hemothorax	Hepatic laceration
Pericardial tamponade	Gall bladder rupture
Posttraumatic arrhythmia	Splenic laceration
Rib fractures	Kidney laceration or avulsion
Thoracic wall injuries	Penetrating abdominal wounds

PHYSICAL EXAMINATION:

(Kraje et al. 2000).

Examination of the orthopaedic trauma patient can be divided into three phases. As with all trauma victims, triage of all of the injuries is very important. At the time of first presentation, evaluation of the respiratory and cardiovascular systems should receive priority, in order to identify potentially life-threatening injuries. Orthopaedic injuries are not immediately life-threatening, whereas mortality due to thoracic trauma or peritoneal haemorrhage in traumatized cats is not uncommon. An initial rapid evaluation of the trauma patient is performed by checking respiratory rate and character, mucous membrane colour, capillary refill time and pulse rate and quality. Careful observation of the cat's breathing pattern can provide an indication of the location of the problem without immediate resort to stressful diagnostic procedures. The lungs and heart should be auscultated, checking for evidence of pulmonary or pleural pathology and cardiac murmurs or arrhythmias, respectively. During the initial examination, attention should also be paid to the cat's level of consciousness and ability to ambulate. The integrity of the urinary bladder can be checked by gentle abdominal palpation, although this does not completely rule out trauma to the bladder itself, ureters, or urethra. Following stabilization of the patient and treatment of life-threatening injuries, a more thorough physical and orthopaedic examination can be performed. This should encompass all body systems and include, at least, a cursory neurologic examination. In those patients suspected of having injury to the nervous system, a complete neurologic examination is then performed. The third and final phase of the examination involves ongoing assessment including re-evaluation for progression of clinical signs and the response to treatment.

DIAGNOSTIC TESTS:

The most important part of the assessment of the trauma patient is the physical examination; however, some ancillary testing is frequently indicated. All cats that have sustained major trauma should have thoracic and abdominal imaging once they are stable, either using good quality thoracic radiography and abdominal ultrasound or, whole-body computed tomography. Computed tomography is also useful in head trauma cases, to evaluate skull fractures. (Figure 3.1)

In cases in which urinary tract trauma is suspected, abdominal ultrasonography and/or radiographic/computed tomographic contrast studies for more distal structures such as the urethra are required to determine the location and nature of the lesion. Laboratory investigations, such as blood gas analysis or a coagulation profile, may be indicated in specific cases. Cats with arrhythmias or tachycardia may have traumatic myocarditis secondary to blunt thoracic trauma and associated myocardial trauma and should have electrocardiographic evaluation, especially if anaesthetic administration is contemplated. The onset of this typically self-limiting posttraumatic arrhythmia is delayed 1–5 days after the traumatic episode and is characterized by ventricular premature contractions or, in severe cases, ventricular tachycardia. Treatment is typically only necessary if the

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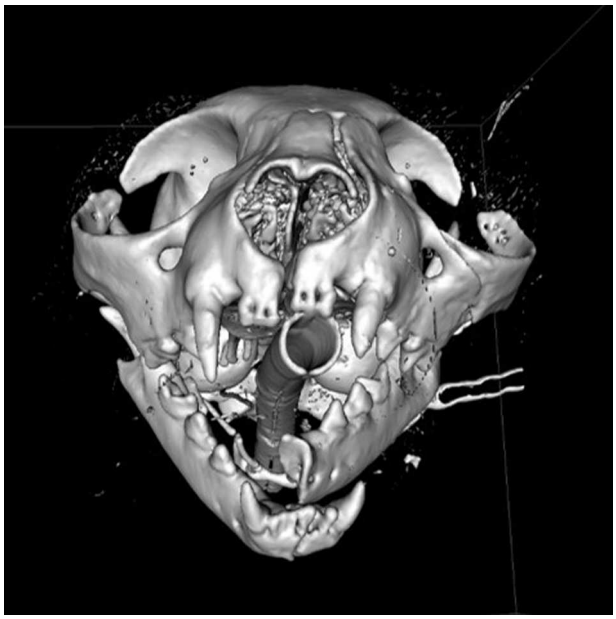


Figure 3.1 Computed tomography is a very useful diagnostic modality for suspected skull and mandibular fractures.

arrhythmias are hemodynamically relevant and may compromise the cardiovascular stability of the patient under anaesthesia.

RESUSCITATION AND STABILIZATION OF THE TRAUMA PATIENT

Oxygen Therapy

Cats that appear to be in respiratory distress following trauma should receive immediate oxygen therapy. Supplementary oxygen can be given during the initial examination using a facemask or flow-by. Struggling and excessive manipulation at this stage may be life threatening and should be avoided. Following this cursory examination, cats with dyspnoea should be placed in an oxygen tent or cage while being stabilized. Longer term administration of oxygen is best performed via a nasal cannula or a hood. A mild sedative, such as butorphanol at a dosage of 0.2–0.4 mg/kg, may be required when these methods are employed.

Fluid Therapy

Cats that have suffered major trauma may have hypovolemic shock, as evidenced by pallor of the mucous membranes, delayed or absent capillary refill, hypothermia, weak or nonpalpable peripheral pulses, and mental depression. The tachycardia typically displayed by dogs is not a typical feature in the cat and the heart rate is most often normal or slow. In addition, cats that have suffered unobserved trauma and been missing for several days will be cachectic, hypothermic and dehydrated. The priority in all cases is to restore hemodynamic stability by restoration of the circulating blood volume. External haemorrhage will be readily apparent, but internal haemorrhage is less easy to detect. The fracture haematoma alone may account for substantial blood loss, especially when the femur or pelvis is involved or where there are fractures of multiple bones. Unfortunately, measurement of the packed cell volume (PCV) in acute trauma cases will not reflect the severity of blood loss, since there is a lag period of up to 24 hours while fluid moves into the circulation. Although overinfusion of fluids must be avoided, one of the greatest causes of mortality in cats requiring intensive care is underestimation and underinfusion of intravenous fluids. Cats presenting with hypovolemic shock should have their circulating plasma volume restored as quickly as possible. Crystalloids should be given routinely at shock rates, and body temperature, urine output, peripheral perfusion and blood pressure (if available) should be monitored. The crystalloids of choice for acute hypovolemia are normal (physiologic) 0.9% saline or Hartmann's (lactated Ringer's) solution. Attempted resuscitation with crystalloids alone carries a high risk of significant pulmonary oedema and accumulation of pleural fluid. Crystalloids are therefore usually used in combination with colloid solutions. Colloid solutions contain large molecules (e.g. dextrans, starch, or albumin), which exert an osmotic effect that holds water within the plasma. Colloids therefore provide more effective and prolonged expansion of the circulating volume than a similar quantity of a crystalloid solution. Rapid infusion rates of colloids are less well tolerated in cats than in dogs, and excessive rates can cause seizures and other adverse reactions. In severe cases of hypovolemic shock, a loading dose of fluid can be given over a period of 5–10 minutes (10–20 ml/kg isotonic crystalloid and 5 ml/kg of colloid). Infusion rates of crystalloid of 40–60 ml/kg/hour are acceptable for a short time to counter life-threatening hypovolemia. The same dose of colloid can be repeated as necessary every 5–10 minutes if systemic arterial blood pressure remains low, and then maintained at 10–40 ml/kg/day. Accurate administration of these fluids requires the use of modern infusion pumps. The cat should be monitored closely and systemic arterial blood pressure, if available, should be checked regularly to prevent volume overload. Should pulmonary edema develop as a result of overinfusion, the infusion of crystalloid should be reduced, the colloid should be stopped, and furosemide should be given at 2–4 mg/kg IV. After the first 24 hours, hypovolemia and fluid deficits should have been corrected. If the cat is unwilling or unable to take oral fluids, then sufficient fluid to fulfil normal maintenance requirements will be needed, together with fluid to replace any ongoing abnormal losses, for example, those incurred as a result of fracture surgery. The maintenance fluid requirement in a healthy cat is 40–60 ml/kg/day.

Blood Transfusion

(Bovens & Gruffydd-Jones 2012, Giger & Bucheler 1991, Knottenblet 2002, Knottenbelt et al. 1999a, Knottenbelt et al. 1999b, Weingart 2008).

Blood transfusion is indicated in the management of acute trauma if the patient has hypovolemic shock and cannot be stabilized by administration of crystalloids and colloids alone. This may occur if the blood loss has been severe or if there is ongoing haemorrhage. In the latter case, blood transfusion is performed in conjunction with surgical intervention to identify the source of haemorrhage and prevent further blood loss. The other common indication for blood transfusion is in the stabilized patient that requires surgery and is moderately anaemic (PCV of 15–20% or less) as a result of previous blood loss. Although cats appear to be more tolerant of anaemia than dogs, transfusion is indicated, especially if further blood loss is anticipated during surgery.

Feline blood transfusion is routinely performed in practice nowadays. Minimal equipment is necessary for

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collection and a dedicated collection system for cats is not needed. Most practices keep a cat or two as possible donors or will keep a list of possible donors belonging to the staff. Due to concerns about transfusion reactions, blood typing must be done in every case prior to blood administration.

Three blood types or groups have been identified in cats (A, B and AB). The blood group is determined by the nature of the blood group antigens on the erythrocyte cell membranes. In worldwide studies type A has consistently been found to be the most common type, whereas type AB cats are consistently uncommon. The proportion of type B cats shows considerable geographical and breed variation. Unlike dogs, in addition to blood group antigens, cats also have naturally occurring serum antibodies to red cell antigens known as alloantibodies. These alloantibodies can result in severe transfusion reactions in blood that has not been cross-matched. Type A cats generally have low anti-B antibody titre and, if given type B blood, may develop mild clinically inapparent transfusion reaction with reduced red cell lifespan. All type B cats have high anti-A titres and, if given even a single transfusion of a small volume of type A blood, develop a severe transfusion reaction. Type AB cats have no naturally occurring anti-A or anti-B antibodies. It is, therefore, vital that donor–recipient compatibility is established prior to first transfusion of all cats.

Ideally, compatibility should be ensured by both blood typing and cross-matching. A desktop blood typing kit has been developed, which requires only 0.25 ml of blood in EDTA and is easy to use (Rapid Vet-H Feline, DMS laboratories or Woodley Veterinary Diagnostics). The kit provides an accurate method by which feline blood can be routinely typed in house. Cross-matching is used to assess the effect that recipient serum antibodies will have on donor cells (major cross-match) and the effect that donor serum will have on recipient cells (minor cross-match). Cross-matching is available at commercial laboratories, although, in an emergency situation, crude cross-matching can be performed in house. The ideal blood donor should be healthy, have a lean body weight >5 kg, should be vaccinated, and should be negative on testing for FeLV, FIV, and Haemobartonella felis. The donor should have a PCV at the high end of normal, preferably >35% (normal range 24–45%) and the blood group should be known.

A cat's blood volume is estimated at 66 ml/kg. Between 10% and 15% of a donor's circulating blood volume may be safely removed, i.e. approximately 35–50 ml for a 5-kg cat. If the volume exceeds 15%, crystalloids should be given to the donor to prevent hypovolemia. The amount of blood required by the recipient can be calculated from the formula:

$$\text{Blood needed (ml)} = \text{Weight of recipient (kg)} \times \frac{\text{desired PCV} - \text{recipient's PCV}}{\text{Donor's PCV}} \times 66$$

As a rough guide, for every 2.2ml of blood/kg body weight transfused, the PCV of the recipient will increase by 1%. Blood is collected from the jugular vein of the donor using a 50-ml syringe with a butterfly extension set. The inside of the extension set and syringe should be coated with approximately 1.3 ml of acid citrate dextrose (ACD) or citrate phosphate dextrose (CPD) per 10 ml of blood to be collected (removed from a human blood transfusion pack). Sedation of the donor is routinely performed, for example, using ketamine combined with midazolam or diazepam (Table 3.6). Hypotensive agents such as acepromazine should be avoided. The blood can be given immediately, via either the intravenous or intraosseous routes, through a blood giving set or in-line filter to remove small clots. A maximum flow rate of 20 ml/kg/hour is normally recommended for hypovolemic patients, but much greater rates can be used in certain circumstances, such as when there is ongoing haemorrhage. Blood not required for immediate use can be stored for up to 4 weeks provided it is kept refrigerated. The recipient should be monitored closely for signs of an acute transfusion reaction, especially during the first 15 minutes of administration. Severe reactions are characterized by haemolysis and systemic anaphylaxis (tachycardia, hypotension, bradycardia, apnoea, vomiting and pyrexia).

The transfusion of canine blood, either whole blood or packed red cells, is not recommended as a routine procedure as it is much less beneficial than transfusion with matched feline whole blood or packed red blood cells. This xenotransfusion is possible if no other options are available, as a life-saving emergency procedure. Cats do not appear to have naturally occurring antibodies against canine red blood cell antigens so acute reactions are not expected from a first blood transfusion. Anaemic cats receiving canine blood are reported to improve clinically within hours. However, antibodies against canine red blood cells are produced rapidly and can be detected within 4–7 days of the transfusion, leading to the destruction of the transfused canine red cells in a delayed haemolytic reaction. The average lifespan of the transfused canine red cells is less than 4 days. A second transfusion with canine blood later than 4–6 days after the first transfusion is contraindicated due to severe anaphylaxis, which is frequently fatal.

Blood Substitutes

(Adamantos et al. 2005, Gibson et al. 2002, Walton 1996).

Because of the difficulties associated with blood transfusion in cats, the availability of a red blood cell substitute would be invaluable. A haemoglobin-based oxygen-carrying (HBOC) solution (oxyglobin) was licensed for use in dogs and has been used in cats both experimentally and clinically.

Administration of HBOC to clinical patients was documented in a retrospective study involving 72 cats. There was a high incidence of complications ranging from those that were mild and clinically insignificant through to severe cardiovascular and respiratory effects. Higher infusion rates were associated with an increased risk of complications. Unfortunately this compound was discontinued from the market and no similar product is currently available.

NUTRITION:

(Chandler et al. 2000, Crowe 1986, Devitt & Seim 1997, Fink et al. 2014, Green 1992, Seim & Willard 2002, von Wethern & Wess, 2001).

It is not uncommon for feline patients to show reluctance to eat or drink during a stay in hospital or following discharge after surgery. Many cats can be coaxed to eat by hand feeding or offering different types of food. Appetite stimulant drugs can be helpful in some cases but are usually not effective in cats that are completely anorexic. Currently, the most commonly used appetite stimulants are mirtazapine

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and cyproheptadine Less commonly nowadays, diazepam, megestrol acetate, prednisolone and oxazepam can also be used for this purpose in the cat.

Mirtazapine is a serotonin receptor antagonist that also antagonizes pre-synaptic α2-receptors, resulting in an increase of norepinephrine. It also antagonizes 5-HT2 receptors, which can result in appetite stimulation via nuclei within the hypothalamus. It produces a significant increase in food consumed after daily dosing in healthy young cats, with minimal side effects. Cats with chronic kidney disease have been shown to exhibit reduced mirtazapine clearance, necessitating an extended dosing regimen, every other day or even every third day. Its use in patients with hepatic insufficiency also requires dosing modification due to reduced hepatic clearance. Adverse effects may include sedation, vocalization and varied behavioural changes (e.g. increased affection). Mirtazapine should not be used in patients receiving monoamine oxidase inhibitors (e.g. selegiline) or tramadol due to an increased risk of serotonin syndrome.

Cyproheptadine is a serotonin (5-hydroxytryptamine, 5-HT) antagonist antihistamine that historically has been more commonly used as a longer term appetite stimulant in cats. It also shows reduced renal and hepatic clearance in patients with renal or hepatic insufficiency necessitating adjustment of the dosage. Cyproheptadine has been reported to cause fulminant liver failure and its use is therefore not recommended in patients with hepatic lipidosis. Side effects may include sedation, paradoxical hyperexcitability, or anticholinergic effects

The concurrent use of cyproheptadine and mirtazapine for appetite stimulation is not recommended since their effects may be negated by each other.

Provision of adequate nutrition is important, because undernutrition or malnutrition have a negative influence on morbidity and mortality in critically injured patients. Adverse consequences for the orthopaedic surgical patient include increased susceptibility to infection, delayed wound and fracture healing, muscle weakness, major organ failure and death. Protein and energy under-nutrition is a common problem in the traumatized cat. The cat's protein requirement is 50% higher for growth and more than 100% higher for maintenance compared with that in the dog. Unlike in the dog, the urea cycle and hepatic transaminases have a fixed high rate of activity and therefore the cat cannot adapt to the amount of protein consumed. Nutritional support is indicated when the intake of calories is reduced because a cat is unable or unwilling to eat and in situations where there is an excessive demand for protein and calories. Common indications for nutritional support in the feline orthopaedic patient include (Figures 3.2 to 3.4):

- Substantial haemorrhage (traumatic and/or surgical).
- Large open wounds.
- Fractures of the mandible or maxilla.
- Multiple orthopaedic injuries.
- Anorexia because of intercurrent medical conditions.
- Cats that have been missing for several days.
- Anorexia because of concurrent injury to other body systems or deterioration of other stable, chronic conditions such as chronic renal disease.

Nutritional support can either be provided by the parenteral route using a central venous catheter or peripheral vein, or by the enteral route, by force-feeding or, preferably, insertion of a feeding tube. The parenteral route is rarely used since it is expensive and has a high complication rate. Enteral feeding is more physiologic, is inexpensive, and is easy to perform with few complications when a feeding tube is properly applied. Additionally, owners can perform feeding by this route at home. Methods for delivery of enteral nutrients include placement of nasogastric, pharyngostomy, esophagostomy, gastrostomy, or jejunostomy feeding tubes. Nasogastric or esophagostomy feeding tubes



Figure 3.2 Oral trauma can negatively affect the patient's ability to eat and nutritional support may be needed.



Figure 3.3 Radiograph of a palatine fracture with moderate displacement along the midline.

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Figure 3.4 A lower lip avulsion, a soft tissue injury, may or may not be associated with other orthopaedic injuries.

are used most commonly for feline orthopaedic patients requiring nutritional support. Gastrostomy tube feeding may be indicated where it is anticipated that longer term nutritional support will be required (weeks to months) but is rarely used for orthopaedic patients. Methods of placement of these tubes have been described.

Nasogastric tube feeding is well tolerated by cats for short-term enteral feeding (up to 10 days). The main advantages of nasogastric tubes are that they are simple to place and placement does not require the administration of a general anaesthetic. The disadvantages are that feeding is restricted to liquid diets because of the small bore of the tube and an Elizabethan collar is almost always required to prevent premature removal.

Esophagostomy tubes are generally preferred for longer term use. Because of the larger bore of an oesophageal tube, blended commercial canned food can be used in addition to commercial liquid diets. Long-term nutritional support using oesophageal tube feeding is well tolerated by cats and has been associated with minimal complications. The surgeon should not hesitate to place a feeding tube at the time of surgery if it is anticipated that there will not be a rapid return to oral feeding post-operatively. Tube feeding is started immediately after recovery from anaesthesia and continued until the patient is feeding voluntarily. It is possible for a cat to eat and drink normally with a feeding tube in place.

PRE- AND POST-OPERATIVE STABILIZATION OF FRACTURES AND LUXATIONS:

(Aron & Crowe 1987, Earley et al. 2018, Fox 1988, Liehmann & Lorinson 2006, Meeson et al. 2011, Piermattei & Flo 1997).

Bandages and splints (Table 3.2 and Table 3.3) are frequently used preoperatively to provide temporary immobilization and reduce swelling for fractures at or distal to the elbow and stifle.

Bandages, splints, slings and casts can be used to provide adjunctive fixation post-operatively for fractures or after reduction of joint luxations. In general, however, external coaptation is not as well tolerated in the cat when compared to the dog and maintaining bandages or splints in place for more than a just a few days can be challenging in cats due to the increased mobility of their skin and their limb flexibility. Soft tissue complications such as pressure sores have been well described and can vary in severity, but they can certainly become major problems with longer term coaptation. The benefits of external coaptation must be weighed against the risk of complications by the veterinary surgeon for each specific case.

Generally, surgeons should strive to repair fractures with a strong and secure fixation method and avoid depending on external coaptation for additional support. Certainly, the tendency in the past decade is to repair most feline fractures, thanks to the greater availability of smaller implants suitable for the feline patient, so most fractures which would have been treated conservatively are now repaired.

In some cases external coaptation may be used as the sole method of fracture fixation (see Chapter 6). The principles and techniques for application are the same as those for the dog and have been well described elsewhere. For fractures

Table 3.2: Effect of Bandages, Slings, Splints, and Casts on Joint Motion and Weight Bearing

Region	Weight bearing, joint still mobile	Weight bearing, joint immobilized	Nonweight-bearing, joint still mobile	Nonweight-bearing, joint immobilized
Shoulder	-	Spica splint		Velpeau sling
Elbow	Robert Jones/soft-padded bandage	Spica splint, cast/splinted bandage	-	Velpeau sling
Carpus	Robert Jones/soft padded bandage	Cast/metasplint	-	-
Hip	-	-	Ehmer sling, 90/90 sling	-
Stifle	Robert Jones/soft padded bandage	Cast/splinted bandage	-	90/90 sling
Hock	Robert Jones/soft padded bandage	Cast/splinted bandage	-	-
Digits	Robert Jones/soft padded bandage	Cast/metasplint	-	-

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Table 3.3: Bandages, Splints, Slings, and Casts

Type	Indications	Complications/Comments
Robert Jones bandage	Pre- (and post-) operative for fractures or luxations at or distal to the elbow or stifle	Circulatory impairment digits
Soft padded bandage	Post- (and pre-) operative for fractures or luxations at or distal to the elbow or stifle	Circulatory impairment digits
Cast	Postoperative immobilization distal to the elbow or stifle (and primary treatment of selected fractures)	Circulatory impairment digits, pressure sores
Spica splint	Pre- and postoperative for scapular, shoulder, and humeral fractures, lateral shoulder luxation, elbow luxation	Circulatory impairment digits, not tolerated by some cats
Velpeau sling	Postoperative for surgery proximal to the elbow, medial shoulder luxation	Not well tolerated by most cats
Ehmer sling	Craniodorsal hip luxation	Circulatory impairment distal to the sling, not well tolerated by some cats
90/90 sling	Postoperative for surgery proximal to the stifle, femoral fractures to help prevent quadriceps contracture	Circulatory impairment distal to the sling, not well tolerated by some cats
Mason metasplint	Pre- and postoperative immobilization of distal extremities	Circulatory impairment digits

distal to the elbow and stifle joints, a soft, padded bandage can be applied post-operatively to the limb to limit swelling. Some surgeons prefer to avoid these bandages completely so that very early physiotherapy modalities can be applied immediately, such as cold therapy, gentle massage, laser therapy, etc. If applied, this bandage is, in most cases, removed 1–2 days after surgery to allow early joint mobility. This also allows careful examination of the limb for signs of neurologic dysfunction or vascular impairment and the initiation of physical rehabilitation techniques. If the bandage is left in place for a longer period of time, or when external coaptation is used for fracture fixation, the cat's toes are observed for signs that the bandage is too tight.

In cats with external fixators, incorporating the frame and limb in a bandage will help reduce initial post-operative swelling. After the initial swelling subsides, this bandage can prevent the frame from catching on objects in the environment, and padding the frame and sharp points of the pins limits self-trauma. In most cases, the pin–skin interface is left exposed and monitored daily for drainage or signs of inflammation. In general, no cleaning is necessary. However, if drainage develops, cleaning the pin–skin interface daily with a mild soap or chlorhexidine solution may be beneficial. If the frame is bandaged, the foam portion from surgical scrub brushes can be dried and packed around the pins beneath the bandage.

Robert Jones Bandage

A Robert Jones bandage is a heavily padded, bulky bandage often used preoperatively to provide temporary stability and reduce swelling in fractures at or distal to the elbow and stifle joints (Figure 3.5).

Tape stirrups are placed on the limb to apply traction while the bandage is placed and to prevent the bandage from slipping. Roll cotton or orthopaedic wadding is applied to the limb, beginning distally and progressing proximally. In cats orthopaedic wadding is often easier to apply than roll cotton. Each wrap should overlap the previous one by approximately 50%. A large quantity of padding is used to provide bulk and is wrapped tightly around the limb. The bandage must extend beyond the joints proximally and distally to the fracture, but the distal aspect of the digits is left exposed to monitor swelling. Elastic gauze is then tightly wrapped over the padding (distally to proximally) to provide compression. The tape stirrups are reflected proximally and stuck to the outer layer of gauze. Tightly wrapped cohesive bandaging tape is then applied to cover the bandage. If additional support is desired, a metal rod or thermomoldable splint material (X-Lite, AOA Kirschner Medical Corp.) can be incorporated into the bandage before the Vetrap is applied. The splint material is generally placed on the lateral aspect of the forelimb and hindlimb for fractures distal to the elbows and stifle or the palmar/plantar aspect of the forelimb/hindlimb, for injuries distal to the carpus/tarsus.

Soft Padded Bandage

A soft padded bandage, or modified Robert Jones bandage, is similar to a Robert Jones bandage but uses less padding. Because it is less bulky than the Robert Jones bandage it provides less immobilization of the limb. It is used primarily for support and control of post-operative swelling at or distal to the elbow or stifle joints. The procedure for application of the bandage is the same as the Robert Jones bandage except that less orthopaedic wadding or roll cotton is used. Splint materials can be added as described earlier.

Spica Splint

A Spica splint can be applied if immobilization of the scapula, shoulder, humerus, or elbow is desired, incorporating the entire forelimb and torso (Figure 3.6).

Spica splints may be used to immobilize fractures prior to definitive repair, as ancillary support after internal fixation, or to stabilize luxations of the shoulder or elbow. They are occasionally used as a primary means of fracture fixation. Unfortunately cats tend not to tolerate external coaptation as well as dogs and can become agitated or otherwise unwilling to move at all. Also, the elasticity of their skin and marked limb flexibility makes secure, long-term application of splints, casts, etc. difficult. For a Spica splint, stirrups are applied to the distal limb to provide traction while the splint is applied. The elbow and shoulder joints should be slightly flexed and the limb placed in a normal standing angle. Several layers of cast padding are applied to the limb from

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Figure 3.5 Application of a Robert Jones bandage. **(a)** Application of stirrups and cotton roll. **(b)** Application of conforming bandage. The stirrups are folded back before application of the final layer. **(c)** Application of Vetrap bandage.

the digits to the axillary region. Application is continued around the torso, wrapping both cranially and caudally to the opposite shoulder in a modified figure-of-eight pattern. Thermomoldable splint material (products such as X-Lite, AOA Kirschner Medical Corp.; Veterinary Thermoplastic (VTP), IMEX Veterinary Inc., Orthoplast, Johnson & Johnson or Vet-Lite casting material) is then placed on the lateral aspect of the limb from just proximal to the digits to the dorsal mid-line. Alternatively, fibreglass casting material can be used. Elastic gauze is then placed to anchor the splint and conform it to the limb and torso as it hardens. Cohesive tape or elastic tape is placed to cover the bandage.

Slings

Slings are almost never used for primary fracture fixation, but they may occasionally be used to prevent weight



Figure 3.6 A completed Spica splint.

bearing after fracture repair and to stabilize joint luxations temporarily, after closed or open reductions. Immobilizing a joint after surgery would allow a degree of fibrosis to develop, which may aid in secondary joint stabilization, but prolonged lack of use would induce excessive periarticular fibrosis, muscle wastage and cartilage changes. Their use would also prevent the clinician from implementing physiotherapy in these patients, while the sling is in place.

Velpeau Sling

Velpeau slings are indicated as a primary or adjunctive stabilization to maintain reduction of medial shoulder luxations and to immobilize fractures of the scapula (Figure 3.7).



Figure 3.7 Application of Velpeau sling. The forelimb is flexed and adducted against the body wall. Cast padding material is applied, followed by roll gauze and Vetrap or elastic adhesive tape. The bandage encircles the torso and the flexed forelimb, wrapping cranial and caudal to the opposite forelimb to prevent slippage. The material should not be placed too tightly.

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To apply the Velpeau sling, the forelimb is flexed and adducted against the thorax. The limb is incorporated into a bandage (cast padding, roll gauze, cohesive tape) that encircles the torso. The bandage is placed cranial and caudal to the opposite limb to prevent slippage. The bandage should allow the cat to breathe normally but must be snug to prevent movement of the affected limb. Velpeau slings are not well tolerated by most cats and very hard to keep in place. Joint stiffness (particularly in the carpus and elbow) is the most common complication, apart from sling slippage.

Ehmer Sling

Ehmer (figure-of-eight) slings are used primarily to prevent weight bearing on the hindlimb and to stabilize craniodorsal hip luxations after closed reduction (Figure 3.8).

A light bandage is placed on the paw over the metatarsus and phalanges (to protect soft tissues and reduce swelling of the toes). The hock and stifle joints are then flexed and adhesive tape is applied around the limb in a figure-of-eight pattern. The tape is placed from the bandage on the paw, medial to the stifle between the thigh and inguinal area, over the cranial and lateral aspects of the thigh, and then medial to the tarsus to the bandage on the paw. Several passes are needed to maintain the limb in a flexed position. Adhesive tape sticks to the cat's hair and reduces slippage of the sling. Gauze may be used instead and is easier to remove but is more likely to slip prematurely. In addition to maintaining flexion of the hindlimb, an Ehmer sling also rotates the femur internally to stabilize the hip joint. If greater abduction of the limb is required to prevent reluxation of the hip, tape from the sling can be extended over the dorsal mid-line to a band of tape placed around the caudal abdomen. This is not necessary when prevention of weight bearing is the goal and is often poorly tolerated by cats. The most common problem with the sling is that it is prone to slip off the cranial aspect of the stifle because of the mobility of the skin in this region. Other complications are discomfort, soft tissue trauma to the metatarsal or thigh region, and swelling of the toes. The sling is not generally applied for longer than 10 days.



Figure 3.8 Ehmer sling application. A small padded bandage is applied to the metatarsus and phalanges. The hock and stifle joints are flexed and adhesive tape is applied in a figure-of-eight pattern (the adhesive tape must not encircle either the thigh or the distal limb). The tape is applied from the bandaged foot, medial to the stifle joint, and over the cranial and lateral aspects of the thigh, medial to the tarsus, back to the foot.

The 90°/90° flexion sling is a variation on the Ehmer sling. It prevents weight bearing on the hindlimb by maintaining the stifle and hock flexed at right angles.

It was used primarily on cats at high risk of quadriceps contracture after repair of femoral fractures, but its use is no longer recommended, the emphasis being now on rigid internal fixation and early physiotherapy to avoid quadriceps contracture.

Metasplints

Metasplints (Mason metasplints/spoon splints) are plastic splints of variable rigidity and thickness, with a sometimes padded, concave surface to accommodate the limb. They have a distal, wider area to fit the paw in and provide a surface for weight bearing. They are typically used to immobilize the extremities distal to the mid-antebrachium in the forelimb and distal to the hock in the hindlimb. These splints are not suitable for immobilization of the proximal radius and ulna, the elbow joint, or the talocrural joint. Their application is similar to a soft padded, splinted bandage as mentioned earlier.

INITIAL TREATMENT OF FRACTURES:

Closed Fractures

(Aron 1988, Aron & Crowe 1987, Ben-Amotz et al. 2007, Demaria et al. 2011, Dirschl & Wilson 1991, Dow et al. 1986, Fox 1988, Gustilo & Anderson 1976, Gustilo et al. 1990, Harari & Lincoln 1989, Hedlund 1997, Nolff et al. 2017, Oakley 1999, Parker & Waldron 1980, Patzakis & Wilkins 1989, Rosin 1993, Rosin et al. 1993, Sanchez et al. 1988, Stevenson et al. 1986, Tillson, 1995, Wilkins & Patzakis 1991, Wiebe & Hamilton 2002, Withrow & Moore 1981).

Fractures proximal to the elbow and stifle joints are typically not immobilized prior to definitive repair. Scapular, humeral, pelvic and femoral fractures are generally closed because of the greater soft tissue coverage. After analgesia is provided, the cat is simply confined (and sedated if necessary) to limit activity, reduce pain and prevent closed fractures from becoming open. If preoperative immobilization of a proximal forelimb fracture is required, a Spica splint may be used. Closed fractures distal to the elbow and stifle joints are placed in a Robert Jones bandage to reduce swelling and pain, minimize further soft tissue injury, and to prevent penetration of the skin by bone fragments. A splint may be incorporated into the bandage for additional support if required.

Open Fractures

For information on the classification of open fractures see Chapter 4. Early, aggressive treatment of open fractures is begun immediately to prevent contamination of the site becoming a deep infection of the bone and soft tissues. Cleaning and bandaging also prevent further contamination of the fracture from the environment (Figure 3.9).

1. A sterile bandage or dressing is applied to protect the wound from further contamination and reduce the likelihood of nosocomial infections. Sterile gloves should be worn at all times when treating the wound. A temporary splint can be applied rapidly, if necessary, to prevent further damage especially in fractious patients.

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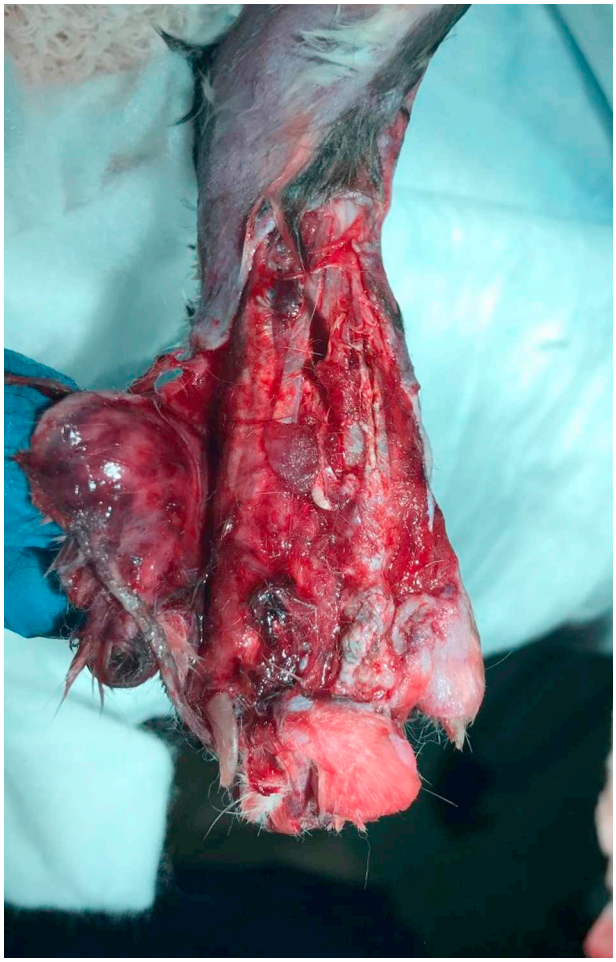


Figure 3.9 This severe degloving injury, associated with metatarsal fractures, requires extensive attention.

2. The cat is examined thoroughly and treated appropriately for shock and any concurrent injuries. Particular attention should be paid to thoracic trauma (pneumothorax, hemothorax, pulmonary contusions), neurologic injuries (cranial trauma, spinal fractures/luxations), abdominal injuries (haemorrhage, bladder ruptures, hernias) and concurrent orthopaedic injures (fractures, luxations).
3. The fracture site is radiographed to document the severity of the orthopaedic injury and aid selection of the appropriate fracture fixation method. These radiographs can be obtained with the splint in place, if needed, for an initial evaluation of the fracture.
4. When the cat is stable, the initial evaluation of the wound is begun. Sedation or short-acting anaesthesia may be required to allow proper treatment. Under aseptic conditions, the bandage is removed and a swab is taken from the wound for bacterial identification and sensitivity testing. Both aerobic and anaerobic cultures are indicated.
5. Broad-spectrum antimicrobial drugs are administered intravenously. It is important to begin antimicrobial therapy soon after the injury to reduce the likelihood of subsequent bone infections. Intravenous administration of fluoroquinolones is contraindicated because they may induce retinal degeneration and these drugs should be used with caution in cats with impaired renal function. Several antibiotics are appropriate for the initial treatment of open fractures, including cephalosporins (cefazolin at 22 mg/kg IV every 8 hours) and clindamycin (11 mg/kg IV every 12 hours). A combination of a cephalosporin and an aminoglycoside may also be used to increase efficacy against Gram-negative organisms. The use of gentamicin (6.6 mg/kg IV or SC every 24 hours) has been recommended, but aminoglycoside therapy should be used with caution in traumatized cats to avoid renal complications. In grade IIIb and IIIc fractures, penicillins, third-generation cephalosporins or metronidazole may also be given to improve efficacy against anaerobic organisms. Antibiotic therapy is continued for 3–5 days and altered, based on the results of sensitivity testing. In many cases, bacteria isolated from open fractures are nosocomial strains from the hospital where the limb is being treated.
6. The wound is filled with sterile lubricating jelly and the surrounding hair is shaved. The wound is lavaged copiously to remove hair and debris; 1–2 l of fluid are recommended for most wounds, although more lavage may be necessary in severely contaminated wounds. A pulse-jet lavage system may be used if available. Alternatively, a lavage system comprised of a 1 l bag of fluid, a sterile intravenous administration line, a sterile three-way stopcock, and a 60-ml syringe attached to a 19 gauge needle can be used (Figure 3.10).

   By adjusting the stopcock, the lavage solution is drawn into the syringe from the fluid bag and then injected through the needle to lavage the wound. This allows the clinician to maintain sterility, lavage the wound with adequate pressure, and avoid excessive force that can drive debris deeper into the wound. Sterile saline or lactated Ringer's are most commonly used for wound lavage. The addition of antibiotics to the lavage solution is controversial and probably provides negligible benefit.



Figure 3.10 Lavage system used for treatment of open wounds. The system consists of a 1 l bag of lactated Ringer's solution, a sterile IV administration line, a sterile three-way stopcock, and a sterile 60 ml syringe attached to a 19 gauge needle. A 0.05% chlorhexidine solution may be used to reduce contamination and improve wound healing: 12 ml of chlorhexidine solution (2%) is added to each 500 ml of lactated Ringer's solution.

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However, the use of a 0.05% chlorhexidine solution has been shown to reduce contamination and improve wound healing. It is prepared by adding 12 ml of chlorhexidine solution (2%) to 500 ml of sterile water, saline, or lactated Ringer's. A precipitate will form when chlorhexidine is mixed with saline. More concentrated lavage solutions are avoided since they may damage tissues and inhibit granulation tissue. A 1% or 0.1% povidone–iodine solution (10% solution diluted 1:10 or 1:100 in saline) may also be used for wound lavage and has broad-spectrum antimicrobial activity. Many cats have contact hypersensitivity to povidone–iodine and stronger solutions can interfere with wound healing. Tap water should not be used as a lavage solution because its hypotonicity may cause additional cellular trauma.

7. All debris and devitalized tissues are removed from the wound (taking care to avoid damaging vessels and nerves) with forceps, scissors and the mechanical action of the lavage solution. Little debridement is usually necessary in grade I open fractures; however, extensive debridement may be necessary in grade III open fractures. Where possible, tissues that are grossly contaminated, discoloured, fail to bleed when cut, or are obviously dried or devitalized are removed. Fat and subcutaneous tissues are excised with minimal consequence in most wounds. Excessive use of electrocautery is avoided to minimize the amount of devitalized tissue in the wound. Muscle is assessed by colour, consistency, circulation and contractility to subjectively determine its viability. If the viability of a tissue is in question, it may be left in situ and removed later if it becomes devitalized. In severely traumatized or contaminated wounds, progressive debridement over several days may be required to achieve a clean wound. In gunshot injuries, accessible bullet fragments are removed; however, healthy tissue should not be traumatized in an effort to remove deeper bullet fragments. The decision to remove or leave cortical bone fragments can be difficult. Fragments that are devoid of soft tissue attachments and not essential to fixation of the fracture are removed so that they do not become sequestra. Articular fragments and those essential to fracture stabilization may be left in place and lavaged. The wound is lavaged again after the debridement is completed.
8. The fracture is stabilized surgically as soon as possible. Early stabilization preserves existing blood supply, promotes ingrowth of new vasculature and helps prevent infection. In many cases, the fracture is stabilized at the time of initial debridement. If the fracture cannot be immediately stabilized, a dressing is applied to the wound and a sterile bandage is placed over it until fixation can be performed. A splint may be incorporated into the bandage to provide support and improve comfort. Wet-to-dry dressings changed daily can be used to aid in debridement of the wound during bandaging.

Although wet-to-dry and dry-to-dry bandages have been extensively used in open-wound treatment, these bandages produce mechanical debridement of the wound surface but also promote evaporation of the wound exudate through the dressing, which leads to the loss of growth factors and cells during therapy. Cats show poorer wound healing capacities than dogs, with slower production of granulation tissue, which can lead to impaired and slow healing. Especially in cases with extensive loss of soft tissue (such as shearing injuries, etc.), effective and rapid establishment of granulation is critical for a successful outcome. Negative pressure wound therapy (NPWT) can therefore be used to greatly aid in debridement of the infected wound and has become the standard of care in human medicine. Its effectiveness in the treatment of canine wounds has been extensively established. This method of wound treatment relies on vacuum to provide constant drainage of the wound bed, promote the formation of granulation tissue by increasing blood flow within the tissues and reduction of interstitial oedema, all leading to a more rapid wound closure and effective infection control, also potentially decreasing the introduction of nosocomial infections. Although not yet reported, its use could be combined with external coaptation during the initial phase of wound treatment in open fractures and with external skeletal fixation or, potentially, with internal fixation after fracture stabilization.

PRE- AND PERIOPERATIVE ANALGESIA:

(Cambridge et al. 2000, Hansen & Hardie 1993, Hellyer 1999, Hellyer & Gaynor 1998, Lamont 2002, Scherk-Nixon 1996, Taylor 1999).

The assessment of pain in cats is notoriously difficult because overt signs may be subtle. In one survey, only one of 15 cats was given any analgesia after surgery. Assessment of pain is best made subjectively, by a trained observer, on the basis of observation and interaction. Cats that are in pain typically become inactive, sit at the back of their cage, and avoid human interaction. They tend to resent or avoid handling and may show signs of aggression. Occasionally a cat will thrash around the cage violently, a reaction that is seen more commonly in young animals. Analgesic drugs should be administered to increase comfort and reduce stress in all cats with fractures. The cat's cardiovascular status should be carefully monitored when analgesics are used. Preemptive analgesia is preferred to control pain in cats scheduled for surgery. It prevents so-called 'wind-up', which is sensitization of the central nervous system from afferent nociceptive stimuli. If possible, balanced (multimodal) analgesia is used. This involves the simultaneous administration of two or more analgesic techniques to achieve a synergistic effect, reduce drug dosages and minimize complications. Many drugs can be administered systemically to provide analgesia in cats, including opioid drugs, α_2-adrenergic agonist drugs, and nonsteroidal antiinflammatory drugs (NSAIDs) (Table 3.4).

Multimodal analgesia is usually achieved by the administration of an opioid and an NSAID, although consideration should also be given to the use of local and epidural analgesia.

Opioids are suitable for moderate to severe pain control. They have a rapid onset of action and block central sensitization and autonomic responses to surgery but are short acting with the exception of buprenorphine. Fractious cats may be given buprenorphine orally by squirting the injectable solution into the mouth so that it is absorbed through the oral mucosa. NSAIDs are suitable for mild-to-moderate pain control. They have a slower onset of action than opioids, are long acting, reduce peripheral pain sensitization and have long-lasting antiinflammatory effects at the surgery site. Gabapentin is a medication licensed for the treatment of a number of conditions in human medicine including focal seizures and neuropathic pain. Indications include neuropathic pain, pain associated with malignancy and arthritic pain. In North America it is the most commonly prescribed medication for the treatment of chronic musculoskeletal pain in cats. Gabapentin is also being used increasingly to treat anxiety and fear associated with stress

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Table 3.4: Analgesics

Drug	Dose (mg/kg)	Routes	Duration (hours)	Comments
Buprenorphine	0.01–0.02	IV, IM, SC	4–8	For mild to moderate pain
Butorphanol	0.1–0.3	IV	0.5–1	For mild to moderate pain
	0.1–0.6	IM, SC	2–3	
Carprofen	2.0–4.0	IV, SC	24	Only one treatment, for mild to moderate pain
Fentanyl	0.002–0.005	IV bolus	<0.5	Short duration unless given as constant infusion
	0.002–0.004 kg/hour	Constant rate infusion	During infusion + 0.5	For moderate to severe pain
	0.025 (25 µg) patch	Transdermal	72	Not recommended for cats <2.0 kg (<4.4lb)
Hydromorphone	0.05–0.1	IV, IM, SC	2–4	For moderate to severe pain
Ketoprofen	1.0–2.0	IV, IM, SC	24	Maximum 3 days; contraindicated with renal disease, hypovolemia, coagulopathies. For mild to moderate pain
	1.0	PO once daily		Maximum 5 days
Medetomidine	0.001–0.01	IV, IM, SC	0.5–2	Monitor for cardiac depression, reversible
Meloxicam	0.3	SC	24	Only one treatment, for mild to moderate pain
	0.1 (1 drop/kg) for 5 days followed by 0.02–0.04 (1–2 drops/cat)	PO once daily	24	Liquid palatable
Morphine	0.1–0.3	IM, SC	3–4	Moderate to severe pain
	0.06–0.12 kg/hour	Constant rate infusion	During infusion	
Oxymorphone	0.02–0.05	IV	3–4	For moderate to severe pain
	0.05–0.1	IM, SC	2–6	
Xylazine	0.1–1.0	IV, IM, SC	0.5–2	Monitor for cardiac depression, reversible

in cats and has been used as an appetite stimulant after surgery. The dose of gabapentin when used for analgesia is 5-10mg/kg twice daily.

Regional Analgesia

(Duke et al. 1994, Hansen 2000, Torske & Dyson 2000, Troncy et al. 2002, Wertz et al. 1994).

Epidural and local administration of analgesic agents can also be used to provide regional analgesia in cats.

Local infusion

Local infusion of bupivacaine (1–2 mg/kg) at the surgery site can be used to provide 2–4 hours of analgesia. The total dose of bupivacaine for a healthy cat should not exceed 2 mg/kg (1.6 ml of a 0.5% solution for a 4 kg cat).

Epidural Analgesia

Epidural administration is usually reserved for cats that are already anaesthetized in preparation for an orthopaedic procedure. Epidural administration of bupivacaine alone provides 4–6 hours of pain relief (Table 3.5).

A single epidural injection of preservative-free morphine can provide effective analgesia for procedures on the hindquarters for up to 24 hours. Bupivacaine may also be combined with morphine for epidural injections. A recent study compared the analgesic effects of epidural morphine with a combination of morphine and bupivacaine in cats undergoing onychectomy. The combination of drugs allowed a significant reduction in the quantity of inhalation agent required to maintain general anaesthesia compared with morphine alone and gave a longer duration of analgesia. The authors concluded that preemptive epidural administration of morphine with or without bupivacaine provided better control of postoperative pain than repeated injections of oxymorphone and ketoprofen. The analgesic effects of epidural fentanyl and medetomidine have also been described.

Oxymorphone is an opioid drug with similar properties to morphine when used for epidural analgesia but with a shorter duration of action. The analgesic effects of drugs administered by epidural injection are both volume- and dose-dependent. A volume of 1 ml/5 kg calculated on the basis of lean or ideal body weight will generally provide analgesia to the level of the first lumbar vertebra. The analgesic agent is diluted with normal saline to achieve the desired volume for injection. Contraindications to epidural analgesia include thoracolumbar and lumbosacral fractures and luxations. The reported incidence of complications after epidural administration of morphine in a series of dogs and cats was 0.75%. For a more detailed description of the technique of epidural analgesia the reader is referred elsewhere. Generally a single injection is made with a 40 mm 22 gauge spinal needle between L7 and S1 (Figure 3.11).

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Table 3.5: Drugs Used for Epidural Analgesia

Drug	Dose (mg/kg)	Dose (ml/kg)*	Onset of analgesia (minutes)	Duration of analgesia (hours)
Bupivacaine (0.5%)+	0.5–0.75	0.1–0.15	10–15	3–6
Morphine (10 mg/ml)+	0.1–0.3	0.01–0.03	20–60	10–24
Morphine (10 mg/ml)+	0.1	0.01	10–15	24
+Bupivacaine (0.5%)+	0.1–0.2	0.02–0.04	-	-
Oxymorphone (1 mg/ml)	0.05–0.1	0.05–0.1	20–40	7–10
Fentanyl (0.05 mg/ml)	0.004	0.08	5–30	2–5
Medetomidine (1 mg/ml)	0.01	0.01	5–10	1–8

* The drugs are diluted with normal saline to achieve a total volume of approximately 0.2ml/kg.
+ Preservative-free morphine and Bupivacaine are preferred. For intrathecal injection it is essential to use preservative-free bupivacaine without epinephrine.

The cat is placed in lateral recumbency and the area is clipped and prepared. The injection is made in the mid-line at the centre of the lumbosacral depression. The dorsal spinal processes of L7 and S1 are palpated and the injection is made between these two points. The spine is flexed at the lumbosacral junction by drawing the hindlimbs cranially. The needle is advanced perpendicular to the skin through the ligamentum flavum. If bone is encountered the needle is withdrawn slightly and walked cranially or caudally along the bone as appropriate to find the space. Slight resistance is encountered as the ligamentum flavum is penetrated and the needle is advanced a further 2 mm until there is loss of resistance. The stylet is removed and the needle hub is observed for blood or cerebrospinal fluid (CSF). If blood is encountered, the needle is removed and the procedure is repeated. It is not uncommon for the needle to enter the subarachnoid space in the cat. If CSF flows from the needle the injection can be made intrathecally, but the calculated dose is reduced by 40–50%. A number of methods have been described for confirming the location of the needle in the epidural space. The simplest method involves the injection of 0.5 ml of air. If the needle is correctly located, the injection will proceed with no resistance and no visible leakage of air into the subcutaneous tissues.

Transdermal Patches

(Davidson et al. 2004, Franks et al. 2000, Gellasch et al. 2002, Glerum et al. 2001, Hofmeister & Egger 2004, Scherk-Nixon 1996).

Transdermal fentanyl patches are suitable for use in cats over 2.0 kg body weight. The application of a 25 µg/hour patch provides relatively consistent analgesia for up to 72 hours after application. Plasma concentrations reach effective levels 7 hours after application. To apply the patch, the skin over the dorsal or lateral thorax is clipped and gently cleaned. Scrub solutions and alcohol are avoided. The patch is handled by the edge or gloves are worn to avoid contact with the membrane. The adhesive backing on the patch is placed directly on the skin and held in place for 1–2 minutes. A bandage may be placed to cover the patch and prevent inadvertent removal; however, this is not always required. Transdermal fentanyl patches can provide a steady-state opioid concentration within the therapeutic range without the need for repeated injections or oral drug administration. However, fentanyl concentrations may vary significantly in individual patients and cats should be monitored for signs of insufficient analgesia or adverse reactions, such as respiratory depression.

PRE- AND PERIOPERATIVE SEDATION:

(Hellyer & Gaynor 1998, Hulse 1997).

Sedative drugs are usually administered as premedication before general anaesthesia (Table 3.6).

Sedative drugs may be administered to stable cats that are refractory to handling or intolerant of confinement or bandages. Sedation is often required during radiography and bandaging procedures. Acepromazine alone is not very

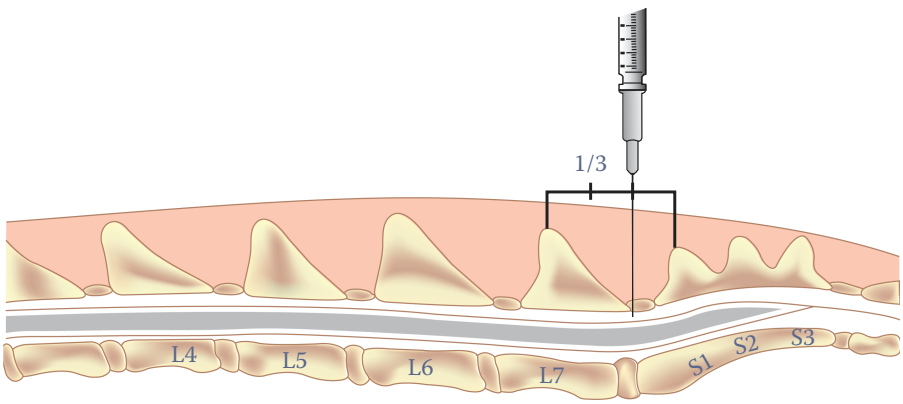


Figure 3.11 Diagram showing the site for epidural injection. The dorsal spinous process of the first sacral vertebra (S1) is more prominent in the cat than the dog. Using L7 and S1 dorsal spinal processes as palpable landmarks the injection site is approximately in the caudal third.

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Table 3.6: Sedatives

Drug	Dose (mg/kg)	Routes	Duration (hours)	Comments
Acepromazine	0.01–0.03	IV	1–2	Avoid if history of seizures
	0.05–0.1	IM, SC	2–6	
Acepromazine	0.05			Avoid if history of seizures
+Butorphanol or	0.1–0.3	IM	2–5	
+Buprenorphine or	0.005–0.01	IM	4–8	
+Morphine	0.1–0.2	IM, SC	2–6	
Medetomidine	0.05–0.08	IV, IM, SC	0.5–2	Monitor for cardiac depression, reversible with atipamezole
Medetomidine	0.025–0.05			
+Butorphanol or	0.1–0.2	IV, IM	2–3	
+Buprenorphine	0.005–0.02	IV, IM	2–4	
Xylazine	0.1–1.0	IV, IM, SC	0.5–2	
Xylazine	0.2			
+Butorphanol or	0.1–0.2	IV, IM	1–3	
+Buprenorphine	0.005–0.02	IV, IM		
Ketamine	2.5			Profound sedation for 12–20 minutes
+Diazepam or midazolam	0.25	IV		
Ketamine	2.5–7.5			Lower doses of acepromazine and ketamine are used in ill cats
+Midazolam and	0.25			
+Acepromazine	0.05–0.1	IM		
Diazepam	0.1–0.5	IV	0.5–1	

effective and combinations of drugs are usually preferred. Acepromazine combined with an opioid will provide mild-to-moderate sedation, and this combination is frequently used for premedication. Alpha 2 agonists, such as medetomidine and xylazine, used alone have adverse effects on cardiopulmonary function and are only used for healthy patients. The addition of an opioid agent to the alpha 2 agonist may reduce some of these adverse effects. Benzodiazepines, such as diazepam or midazolam, given alone can provide satisfactory sedation in cats that are very ill and these agents may also be used in combination with opioids given at the same dosages that are recommended for use with alpha 2 agonists. Ketamine, in combination with acepromazine and diazepam or midazolam, provides effective sedation and low doses of this combination are suitable for very ill cats. Inclusion of both the acepromazine and the benzodiazepine components of the mixture increases its efficacy with no increase in the adverse effects. Atropine (0.04 mg/kg) or glycopyrrolate (0.005–0.01 mg/kg) may also be administered to control bradycardia during sedation.

POST-OPERATIVE CARE:

(Gentry & Mann 1993, Schwarz et al. 2001).

Post-operative management is an important component of treatment of the orthopaedic trauma patient. Immediate concerns include monitoring and supporting respiratory and cardiovascular systems during the anaesthetic recovery period. Fatal pulmonary fat embolism has been reported as a complication of fracture repair and may be a cause of unexplained chronic progressive post-operative respiratory failure. Cats lose body heat rapidly in comparison with dogs because of their high surface area to volume ratio. Attention must be paid to thermoregulation, especially in cats that have received sedative or anaesthetic drugs. Analgesia is routinely continued into the post-operative period to alleviate pain and reduce stress. Other aspects of post-operative management include radiography, antibiotic therapy, bandaging, nutritional support and physical therapy. The importance of good nursing care should not be underestimated.

Radiography

Radiographs are obtained immediately after surgery to confirm reduction and implant positioning. Complications, such as the placement of implants in joints, severe malalignment, or evidence of inadequate stabilization may necessitate an immediate return to the operating theatre to correct the problem. If available, intraoperative radiographs are extremely helpful to monitor implant placement during the procedure. Follow-up radiographs are typically obtained 6–8 weeks after the surgery, depending on factors such as the age of the patient, fracture type and fixation method. Depending on the case, further radiographs may be needed later to monitor healing, check for signs of infection and observe for implant complications. Radiographs can be obtained more frequently if complications develop and additional monitoring is indicated.

Analgesia

(Slingsby & Waterman-Pearson 2000).

Analgesic drugs are administered during recovery from anaesthesia, and the cat is carefully observed for signs of pain (Table 3.4). Long-acting agents are preferred for post-operative analgesia. For moderate-to-severe pain control in the immediate post-operative period a continuous intravenous infusion of an opioid or a transdermal fentanyl patch can be used. Analgesics are continued as necessary during fracture healing, although many cats are quite com-

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fortable after a few days and medications can be reduced or discontinued. NSAIDs should be used with caution in cats. Carprofen, ketoprofen, meloxicam and tolfenamic acid have been shown to provide effective short-term analgesia when administered perioperatively to cats.

Antibiotics

Antibiotics are not administered routinely after closed fracture repair or after open stabilization under aseptic conditions for procedures lasting less than 2 hours (unless other factors dictate their use). The prophylactic antibiotics administered during surgery are usually adequate. However, for longer procedures, or if contamination is present (or occurs during surgery), therapeutic administration of antibiotics may be necessary after surgery. If possible, the choice of antibiotics is based on the results of culture and sensitivity testing.

Restricted Activity

The patient's activity is restricted while the fracture is healing. Although early return to limb and joint function is beneficial, excessive loads should be avoided. Sedation is rarely needed to limit activity in cats. Full restriction is usually achieved by confining the cat to a large dog crate so there is enough space for the patient to walk around, change positions, stretch and so forth. The cat can be taken outside of the crate twice a day, for several minutes at a time and under supervision so it can socialize and, if tolerated, walk around the house on a lead/harness. Restriction to a small room in the house, with good non-slippery footing, is also possible if some exercise can be allowed, but complete avoidance of jumping would be impossible unless the room is empty. The transition period from indoor to outdoor exercise needs to be done carefully so unrestricted access to exercise outside is not gained suddenly.

Cats with external fixators are confined until implant removal is complete to ensure the fixator frame does not become caught on furniture, fences, or other objects in the environment.

POSTTRAUMATIC/POST SURGICAL OSTEOMYELITIS:

(Albarellos et al. 2016, Dow et al. 1986, Johnson 1994, Muir & Johnson 1992, Slunsky et al. 2017, Weese 2008).

Posttraumatic bacterial osteomyelitis is uncommon in cats and usually occurs after open reduction of fractures, treatment of open fractures, or bite wounds. In comparison with dogs, local extension of soft tissue wound infections involving the distal extremities is a more frequent cause of osteomyelitis. *Staphylococcus sp.*, *Streptococcus sp.* and *Escherichia coli* are common isolates from infected bones. *Proteus sp.*, *Klebsiella sp.*, *Pseudomonas sp.*, *Pasteurella sp.*, and anaerobes, such as *Actinomyces sp.*, *Clostridium sp.*, *Bacteroides sp.* and *Fusobacterium sp.*, are frequently isolated from bone infections caused by bite wounds. Multiple organisms may be present. Young male cats are overrepresented, reflecting the disproportionate incidence of fractures and bite wounds in this section of the cat population.

It has been well established that bacterial contamination of surgical sites is very common, but it does not necessarily lead to post-surgical infection. After internal fixation of fractures, surface colonization of metal implants must be distinguished from a deeper infection into the local soft tissues and/or bone. Very often, bacteria can be cultured from implants removed routinely, in healthy patients in which an infection is not suspected. The presence of metal implants in general increases the chances of a surgical site infection or persistent low grade contamination due the ability of bacteria to produce a glycocalyx on the surface of the metal.

Diagnosis

The diagnosis of bacterial osteomyelitis is based on clinical signs, characteristic radiographic findings, culture of samples collected from the site by aspirate or biopsy, and, in some cases, blood culture.

Clinical signs of osteomyelitis may include pain, swelling, erythema and poor limb function. Initially, these signs may be difficult to distinguish from early recovery from fracture repair or an incision complication, although an acute onset of more severe lameness or failure of the patient to improve as expected after fracture repair may be an indication of infection. A persistent fever may be present in some cats. With chronic infections, muscle atrophy and muscle fibrosis may be observed. Eventually, draining tracts may develop in the skin over the infected bone. Radiographic signs of osteomyelitis depend on the chronicity and severity of the infection (Figure 3.12).

Radiographs obtained early in the disease process may be inconclusive, as signs may be difficult to distinguish from the normal healing process. However, as the infection progresses, loss of the normal trabecular pattern, bone lysis, sclerosis and a periosteal response separate from the healing callus may be appreciated. With the passage of time, coalescence of adjacent foci of lysis leads to larger irregular areas of bone loss. Bone resorption is most marked around implants and at the fracture site. Eventually, sequestration of bone may occur. Sequestered bone maintains its increased density and sharp borders and thus appears denser than the surrounding bone. A more radiolucent region comprised of infected exudate usually surrounds the sequestrum. Surrounding this is the involucrum, comprised of new bone and fibrous tissue laid down as the body attempts to isolate the infection. Unfortunately, radiography alone is neither highly sensitive nor specific in the diagnosis of osteomyelitis. Nuclear scintigraphy, although not specific, is very sensitive and can be helpful. If infection is suspected, confirmation should always be sought by culture of the wound. Culture and sensitivity testing is performed on samples collected by deep aspirates of the infected site or biopsy samples of bone or periosteum collected during surgical debridement. Aspiration of the site is performed using a 20–21-gauge needle following aseptic surgical preparation of the skin. The sample is placed in blood culture medium for culture and sensitivity testing. The efficacy of this technique is supported by a study in dogs in which its use resulted in bacterial growth in 86% of cases. The samples should be examined for aerobic and anaerobic bacteria. Cultures from draining tracts usually isolate contaminant or opportunistic bacteria and provide little information about the organism affecting the bone. Blood cultures are rarely needed but can be helpful in identifying the pathogenic organism in cats with osteomyelitis.

Prevention

Perioperative antimicrobial prophylaxis is recommended for all surgical procedures to prevent surgical site infections. The ideal antimicrobial agent for prophylaxis should be bactericidal, non-toxic and achieve high enough serum concentrations against the typical pathogens involved in surgical site infection. Intravenous administration of

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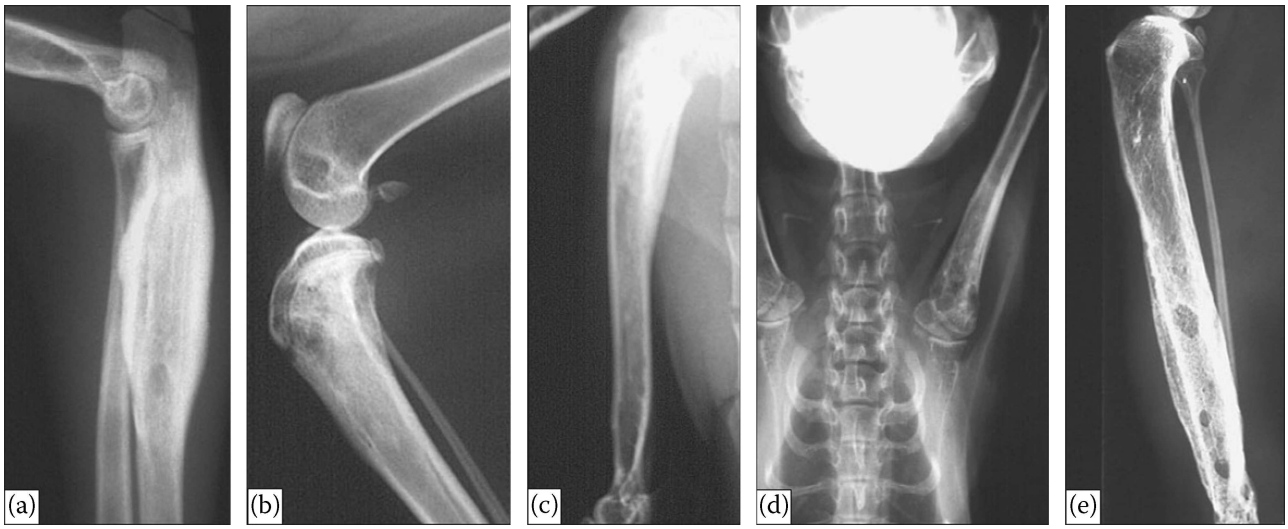


Figure 3.12 Osteomyelitis of the long bones. **(a)** Lateral radiographic view of an ulna with osteomyelitis secondary to a bite wound. Sclerosis and a bony sequestrum are visible. **(b)** Lateral radiographic view of a stifle joint with osteomyelitis affecting the tibial tuberosity. **(c)** Lateral radiographic view of a humerus with osteomyelitis of the proximal metaphysis and diaphysis. Note the bony sclerosis and periosteal proliferation. **(d)** Craniocaudal radiographic view of a humerus with pyogranulomatous osteomyelitis. Note the bony lysis and loss of the normal trabecular pattern in the proximal humerus. **(e)** Lateral radiographic view of a tibia, with osteomyelitis secondary to a bite wound. Lysis, periosteal reaction and sclerosis are evident in the diaphysis and distal metaphysis.

cefazolin is the usual choice for prophylaxis in the perioperative period in both human and veterinary medicine. A recent study showed that a single cefazolin intravenous administration of 20 mg/kg provides adequate plasma and tissue concentrations to guarantee antimicrobial coverage for most common pathogens that colonize surgical sites in cats. Therefore, this should be done for all clean orthopaedic surgeries for prophylactic antibiotic coverage.

Prevention of bacterial osteomyelitis is better than cure and all bite wounds and abscesses should be treated aggressively, especially in locations where there is underlying bone or in open fractures. Further soft tissue trauma during fracture repair should be minimized by appropriate debridement and the use of an atraumatic surgical technique. The vascularity of bone fragments should be preserved by avoidance of periosteal stripping and by leaving muscle attachments to bone fragments undisturbed. Intraoperative inoculation by bacteria occurs when there is a breach in aseptic surgical technique. Prolonged operating time, associated with attempted anatomic reconstruction of comminuted fractures, increases the extent of bacterial contamination of wounds and also has a detrimental effect on the vitality of the soft tissues around the fracture. Metallic implants and suture material should be used judiciously where there is wound contamination or infection. External fixators are useful for the treatment of open fractures and shearing injuries, especially those distal to the elbow and stifle joints. Removal of clearly avascular bone fragments, devoid of any soft tissue attachments, should be considered (removed unless they make a significant contribution to the stability of the reconstructed fracture) during fracture reconstructions, unless they can be rigidly secured to the parent bone. However, as less invasive fixation techniques and better implants have been developed and more refined understanding of bone healing has been achieved, comminuted fractures are now routinely treated with a more biological approach, leaving most if not all fracture fragments to be incorporated in the bone healing. This reduces the risk of iatrogenic osteomyelitis since it limits bone exposure and further damage to the fracture's soft tissue envelope.

As part of a more modern approach to infection control in animal hospitals, development of a proper infection control programme is highly recommended for every practice. Unfortunately, this is an area that is still often overlooked in veterinary medicine, despite the increasing concerns with bacterial antimicrobial resistance and nosocomial infections. An infection control program requires developing protocols, ensuring compliance and surveillance. Providing a resource for answering infection control questions and ensuring training to new staff are some of the tasks required to make this an effective tool. Although formal training of the responsible person on infection control is recommended, it is not mandatory, and it is likely to become more available as concerns with antibiotic resistance grow in the future. Veterinary nurses and technicians can become a very useful resource for this necessary role in modern veterinary practice.

Treatment

(Caylor & Cassimatis 2001, Wiebe & Hamilton 2002).

Treatment of osteomyelitis requires long-term systemic antibiotic therapy using appropriate drugs at adequate dosages, debridement and lavage of the infected site (including removal of dead bone), and rigid stabilization of the fracture (including the placement of cancellous graft in bone deficits).

Antimicrobial Therapy

Systemic antibiotic therapy is indicated in all cats with bacterial osteomyelitis. The selection of drugs is based on culture and sensitivity testing, ideally from deep samples obtained from the surface of implants or periosseous area and not superficial samples from draining sinuses on the skin.

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- Gram-positive organisms cause the majority of infections and cefazolin or amoxicillin/clavulanate are often used pending sensitivity results.
- Metronidazole or clindamycin may be administered in cases where anaerobic infection is identified or suspected. Long-term or high-dose therapy with metronidazole should be restricted to refractory cases because of potential neurotoxicity.
- Aminoglycoside or fluoroquinolone antibiotics should only be used for refractory infections caused by Gram-negative organisms, because of the potential for toxicity.

Fluoroquinolones are chondrotoxic in immature cats and long-term administration should be avoided in geriatric cats or cats with impaired renal function because of potential retinal toxicity. Systemic antibiotic therapy is continued for 5–7 weeks or until clinical and radiographic signs of active infection have resolved. Rarely, the infected site is re-cultured after several weeks of treatment to identify resistant organisms that may have developed during prolonged antibiotic therapy, particularly if radiographic and clinical signs of infection are not improving or worsen during the course of therapy. Cats should be monitored carefully for complications associated with long-term antibiotic treatment.

In cases of severe chronic osteomyelitis, local antibiotic therapy may be done by placing gentamicin- impregnated collagen sponges or GICS (Collatamp G, Schering-Plough, or similar products) or other antibiotics into the infected site, in isolation or in conjunction with systemic treatment.

The usage of PMMA beads is no longer favoured because of the need for a second surgery to remove them a later date. GICS results in prolonged antimicrobial release without the need for future implant retrieval. Furthermore, the sponge is simple to insert and does not interfere with joint motion if placed intraarticularly. GICS are absorbed and the release of gentamicin depends on the rate of collagen breakdown. The relatively rapid rate of gentamicin release prevents sub-inhibitory local antibiotic concentrations, which can occur using PMMA beads, and which contribute to bacterial resistance problems. To the best of the author's knowledge, their use has not been reported in the cat.

Debridement and Lavage

All necrotic tissues are surgically excised. In chronic osteomyelitis, dead (avascular and infected) fragments of bone (sequestra) may be present and are often surrounded by a pocket of infected fluid and a layer of fibrous tissue and new bone (involucrum). All sequestra are removed and the involucrum is debrided to healthy, bleeding bone. Previously placed orthopaedic implants that do not contribute significantly to fracture stability are also removed. Stable implants may be left in place until healing is complete. The wound is lavaged with sterile saline or Ringer's solution.

Fracture Stabilization

Infected fractures will generally heal if they are stable and have adequate blood supply. If the fracture has not been previously stabilized (or if previously placed implants were removed during debridement), rigid fixation is applied. In open fractures, external skeletal fixation may be considered since it can be applied with minimal disruption to the fracture site's blood supply by percutaneous insertion of the fixator pins away from the fracture area, especially in the tibia and radius/ulna. This is particularly useful in situations with extensive soft tissue damage, such as degloving or shearing injuries. Judicious placement of positive-profile threaded pins in safe corridors, in combination with stiff clamps and connecting bars can help build a strong and comfortable configuration frame that can provide durable enough fixation to ensure fracture healing in most situations. Circular fixation or hybrid (combination of circular and linear fixation) may also be used, depending on surgeon's preference and fracture configuration. Plates are occasionally used because they provide rigid fixation for the long period of time required for infected bone to heal. However, the extensive soft tissue dissection required to position the plate properly can be a disadvantage when stabilizing infected fractures. Therefore, a MIPO (minimally invasive plate osteosynthesis)-type of approach is favoured, when plates are used.

Intramedullary pins and cerclage wires are not recommended for stabilizing infected fractures since they are unlikely to provide enough stability to the fracture. Additionally, there is a theoretical concern with intramedullary pins spreading the infection further by dragging bacteria along the medullary canal. Once the fracture is stabilized, cancellous bone graft is placed in all bone defects. Alternatively, grafting can be delayed until the initial inflammation and suppuration is resolved. The wound or incision made to place implants can be closed primarily (after thorough debridement and lavage) in most cases. In more severe cases, open wound management with wet-to dry bandages or closure of the incision over a drain may be preferred. Perhaps a better option nowadays for these severe cases, consideration to negative pressure wound therapy (NSWT) may be given as an alternative to treat the infected wound and encourage early granulation tissue before final closure, even in the presence of orthopaedic implants. The orthopaedic implants are removed once the fracture is healed, since they may act as a nidus for infection, by harbouring bacteria, and prevent complete resolution of the osteomyelitis. Culturing from the implants at the time of removal may help determine whether there is a need for longer antimicrobial therapy.

Prognosis

The prognosis for osteomyelitis is variable depending on the stage and extent of the infection. Low-grade osteomyelitis may be resolved with appropriate antibiotic therapy and delayed removal of implants. More severe and extensive infection carries a much more guarded prognosis and may require prolonged and expensive treatment with an uncertain outcome. Such cases may require multiple surgical procedures for removal of sequestra, fracture stabilization, cancellous bone grafting and eventual removal of all metallic implants. Even if the infection can be resolved, the legacy of infection and chronic disuse may be poor limb function associated with muscle atrophy and fibrosis, soft tissue adhesions and joint stiffness. Ensuring adequate physiotherapy during bone healing is important to avoid these complications (see Chapter 16).

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4 Fracture Classification, Decision-Making and Bone Healing

FRACTURE CLASSIFICATION:

(Clayton-Jones 2016, Piermattei & Flo 1997).

Numerous systems have been devised to classify fractures, including cause, anatomic location, severity, fracture configuration and contamination. Fracture classification and standardization of nomenclature are useful because they help communication amongst surgeons and clarity in the description of specific fractures. The discussion and planning of fixation methods and ideal treatment plans, even across hospitals and countries, can therefore be facilitated. It also allows direct comparisons of outcomes for a specific fracture type and fixation method and could help facilitate ordering of implants based on prevalence and frequency of specific fracture types. Additionally, comparison between treatment methods, outcomes and so forth could be established between human and veterinary surgery if a similar classification system could be agreed upon.

Accurate and complete, concise but systematic description of a fracture provides useful information when selecting repair options and facilitates communication among clinicians. In most cases, more than one classification system is used to describe fully a given fracture, since the veterinary community has not yet agreed on any specific classification system of fractures in small animal patients. Alphanumerical systems, such as the AO/ASIF classification in human and equine orthopaedic surgery, use the affected bone, location and pattern of the fracture to produce a code, based on previously agreed definitions of the descriptive terms. These systems attempt to improve on the mere verbal descriptions used in the past, when there was no universally agreed definition of the terms being used, leading to confusion between communicating clinicians.

Cause

Traumatic (Direct or Indirect)

(Cantatore & Clements 2015).

Direct trauma to a bone is the most common cause of fractures and frequently occurs when the cat is hit by an automobile or falls from a significant height. Indirect trauma to bone may lead to fractures by transmitting force through bones or surrounding muscles, tendons and ligaments. Femoral neck fractures and avulsion fractures of the calcaneus are examples of fractures often caused by indirect trauma. Fractures caused by repeated low-grade trauma (stress fractures) have been reported in cats, but their pathogenesis is not completely clear since they may be the result of a bone malformation such as a mild form of osteogenesis imperfecta, versus a true fatigue fracture, as reported in other species.

Pathological

(Brockley et al. 2012, Simon et al. 2000).

Pathological fractures occur when a disease process weakens the bone, whether this process is a localized primary bone pathology (primary osteosarcoma), it secondarily invades the bone locally (soft tissue sarcoma invading nearby bone) or the skeletal system becomes involved as part of a systemic disease. Causes may therefore include neoplasia, infection and nutritional or metabolic bone diseases (Figure 4.1).

Anatomic Location

(McNicholas et al. 2002, Salter & Harris 1963).

The location of a fracture in a long bone may be described as diaphyseal, metaphyseal, or epiphyseal. Other anatomic nomenclature is often used to describe specific fractures more completely, including terms such as capital, condylar, supracondylar, subtrochanteric and articular. Physeal fractures involve the growth plate in immature animals and are described using the Salter–Harris classification system (Figure 4.2), which is an old human system based on a radiographic description of the orientation of the fracture line and its relationship to the growth plate.

Distal femoral growth plate fractures are common in cats (typically Salter-Harris type II). Femoral capital physeal fractures (typically type I), can occur as a result of trauma or spontaneously in young, male obese cats, often bilaterally.

Other locations, such as distal tibial, proximal tibial, proximal humeral growth plates are much less common.



Figure 4.1 Proximal tibial osteosarcoma producing a pathological fracture.

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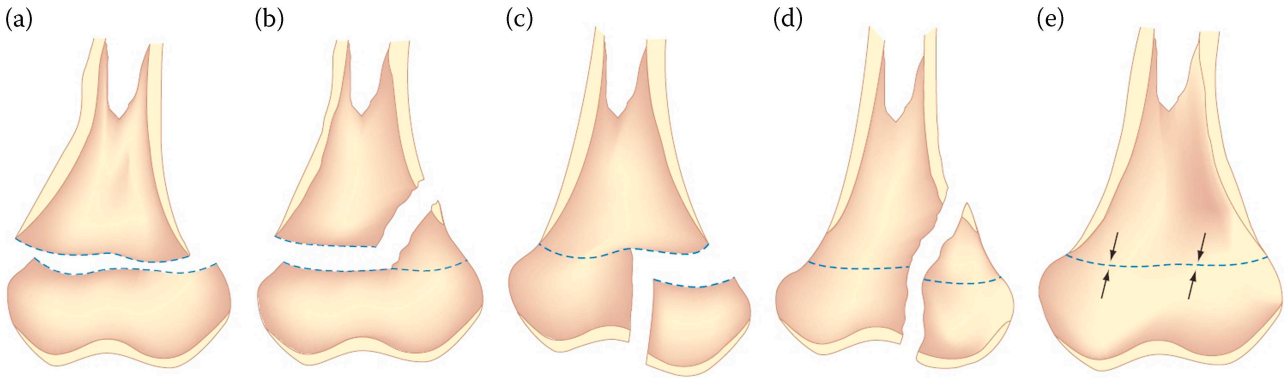


Figure 4.2 Salter–Harris classification of growth plate injuries. **(a)** Type I involves the growth plate only. **(b)** Type II involves the growth plate and the metaphysis. **(c)** Type III involves the growth plate and the epiphysis. **(d)** Type IV involves the growth plate, metaphysis and epiphysis. **(e)** Type V is a compression injury of the growth plate.

Severity of Fracture

Incomplete

The fracture involves only one cortex of the bone and displacement is minimal (e.g. fissure fractures that disrupt a single cortex and often course longitudinally in a bone) (4.3 a). Greenstick fractures are incomplete fractures of immature bones in which there is a 'folding' of the bone rather than a true fracture.

Complete

The fracture disrupts the entire circumference of the bone and may lead to significant displacement. Displacement of a fracture, as in a joint luxation, is described based on how the distal fragment moves relative to the proximal fragment. The direction of displacement of the main fracture fragments is usually predictable and is due to the muscle pull of the surrounding muscle groups. Once the integrity of the bone is lost due to the fracture, the inherent tension of the muscles inserting on the fragments is able to move them in specific directions. For examples, in diaphyseal femoral fractures, the proximal fragment is always displaced cranially mainly due to the muscle pull of the iliopsoas muscle and the distal fragment is displaced caudally due to the action of the hamstring group and gastrocnemius muscles

Complete fractures can be classified into:

- Simple: One fracture line produces two main fragments (see later for different configurations)
- Comminuted: Multiple fragments are present, in addition to the main proximal and distal fragments. Comminuted fractures have more than two fragments and severely comminuted have 5 or more fragments. Comminuted fractures can be further characterized into reconstructible or not reconstructible, depending on whether or not the comminution can be repaired to restore the original anatomy, as a bony column between the main proximal and distal fragments.

Segmental fractures are a variation of comminuted fracture in which there are two or more separate fractures in the bone and the fractures lines do not connect. In diaphyseal fractures, the fracture lines in segmental fractures can be very separate from each other, leaving a centre, cylinder-like fragment (Figures 4.3 and 4.4).

Fracture Configuration

Simple fractures can be classified according to direction of the fracture line (Figures 4.5). The basic configurations are:

Transverse

The length of the fracture line is approximately equal to the diameter of the bone (4.5 a) and the fracture line is roughly perpendicular to the diaphyseal axis.

Short Oblique

The length of the fracture line is less than two times the diameter of the bone (4.5 b). The fracture line angle is greater than 30° to the long axis of the bone.

Long Oblique

The length of the fracture line is greater than two times the diameter of the bone (4.5 c). The fracture line angle is less than 30° to the long axis of the bone.

Spiral

This is a long oblique fracture in which the fracture line curves around the diaphysis (4.5 d). These fractures can be considered oblique fractures that involve different planes as they progress along the bone and are usually the result of torsional forces.

Other Configurations and Descriptive Terms

Impacted

The fracture fragments are compressed together.

Depressed

A fracture which invades an underlying cavity, typically the skull.

Avulsion

The origin or insertion site (apophysis) of a tendon or ligament is pulled from its parent bone (4.5 e) by an overwhelming muscle pull. The greater trochanter, tibial tuberosity, tuber calcis of the calcaneus and olecranon are common sites where avulsion fractures occur in cats due to an acute muscle pull. These fractures are more common in immature cats due to the presence of growth plates, which represent a weak area in the bone structure. A typical avulsion fracture in cats due to a ligament pull are tibial and fibular malleolar fractures due to the pull of the

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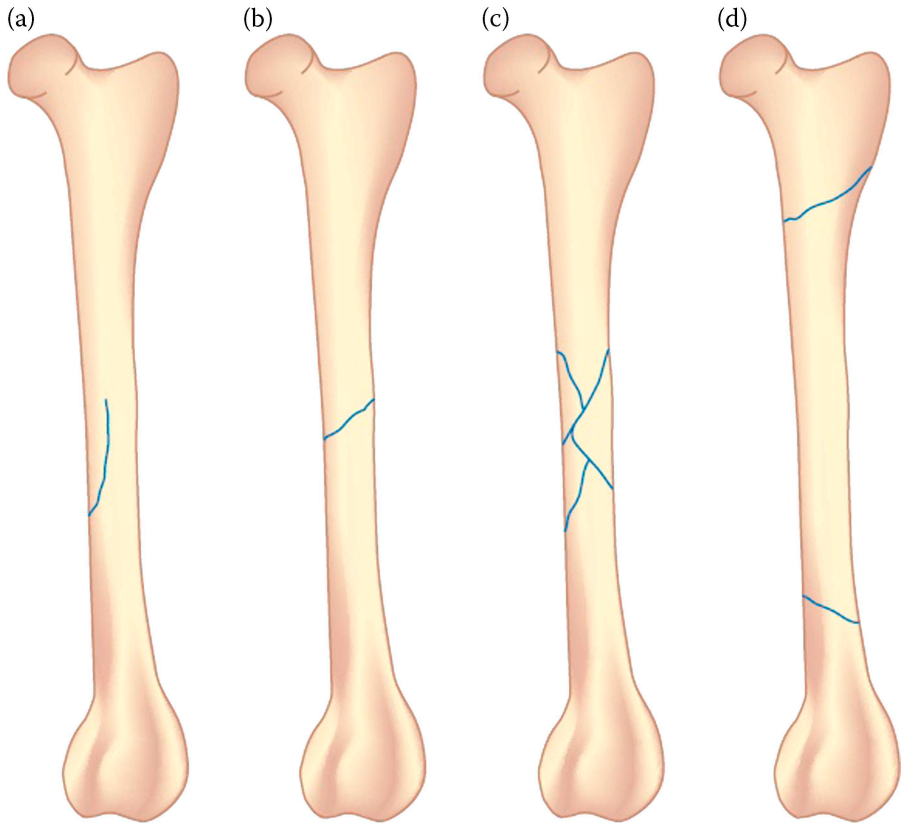


Figure 4.3 Fracture classification based on the severity of bone disruption. **(a)** Incomplete. **(b)** Complete. **(c)** Comminuted. **(d)** Segmental.

tibiotarsal collateral ligaments in severe sprain injuries of the hock.

Chip Fracture

These are small fragments that typically develop from the edge of articular surfaces

Slab

A fragment of articular bones, usually larger than a chip fracture, which involve the underlying cancellous bone and one or two articular surfaces.

Contamination

Closed Fracture

No communication exists between the exterior and the fracture site. The skin is intact, although contusion and varying degrees of bruising may be present. These are the most common type of fractures.

Open Fracture

(Gustilo & Anderson 1976, Gustilo et al. 1984, Millard & Weng 2014, Wang et al. 2011).

This is a relatively common presentations in cats, estimated as 29.2% of all fractures in one report. Particularly at risk for an open fracture are, statistically speaking, young intact male cats suffering from trauma to the tarsal area or a comminuted fracture at any location Figures 4.6 and 4.7.

A communication exists between the exterior and the fracture site. Open fractures can be classified by the degree of soft tissue injury associated with the fracture. This is based on the Gustilo and Anderson classification of human open fractures

- Grade I: A small puncture (<1 cm) is present in the skin. The puncture occurs from the inside when a sharp fragment of bone briefly penetrates the surrounding soft tissues and skin. There is usually a clean wound, limited soft tissue damage, no bone loss and the bone exposure is typically not permanent.
- Grade II: A wound (>1 cm) is present and communicates with the fracture site. The wound is a result of external trauma (such as an animal bite or a low energy projectile such as an airgun pellet). Contusion of the surrounding soft tissues is common and can be severe, but there is no clear loss of soft tissue or bone.
- Grade III: An extensive wound is present and is caused by high-energy external trauma, such as gunshot injury. Soft tissues surrounding the fracture site are severely damaged or absent. Also, fractures that remain open for longer than 8 hours are considered Grade III.

These lesions can be further classified into

- Grade IIIa: Significant soft tissue injury is present, but adequate tissue remains to allow closure of the wound.
- Grade IIIb: Extensive soft tissue injury with insufficient skin and soft tissue remaining to allow primary wound closure (i.e. degloving or shearing injuries).

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Figure 4.4 Lateral and craniocaudal views of a severe comminuted, segmental, diaphyseal humeral fracture in a 3-year-old cat after vehicular trauma.

- Grade IIIc: Extensive soft tissue injury with compromised vascular supply to the distal extremity. Loss of the limb may occur without immediate intervention.

A different classification, SMABC, proposed by the Orthopedic Trauma Association, consists of five factors: S (skin), M (muscle injury), A (arterial injury), B (bone loss) and C (contamination). There is a grading system for every factor, as mild, moderate and severe. The use of this classification system in the veterinary literature has not been reported.

Fracture Classification Systems

There are two systems for long bone fracture classification described for small animal orthopaedic surgery, adapted from the AO/ASIF system for computerized documentation of human fractures. Neither of them has been universally accepted for use in veterinary surgery.

The Unger Classification System

(Unger et al. 1990).

This is a classification of fractured long bones in dogs and cats, adapted from the AO/ASIF classification of human fractures, which allows the coding and computerized filing of these injuries. The localization and morphology of fractures are characterized with defined conventional terms, in order to assign an alpha-numeric code to each fracture. With this classification system, the fractures are ranked in increasing severity and complexity for the various anatomical locations.

The long bones are referred to by number:

- Humerus = 1
- Radius/ulna = 2
- Femur = 3
- Tibia/fibula = 4

The fracture location is also referred to by number:

- Proximal = 1
- Shaft = 2
- Distal = 3

Fracture severity is classified as follows:

- A = simple
- B = wedges
- C = complex

Each further graded from 1 to 3 based on the degree of fragmentation.

This classification system is used in some veterinary literature to classify fractures and in reported data on fracture treatment methods.

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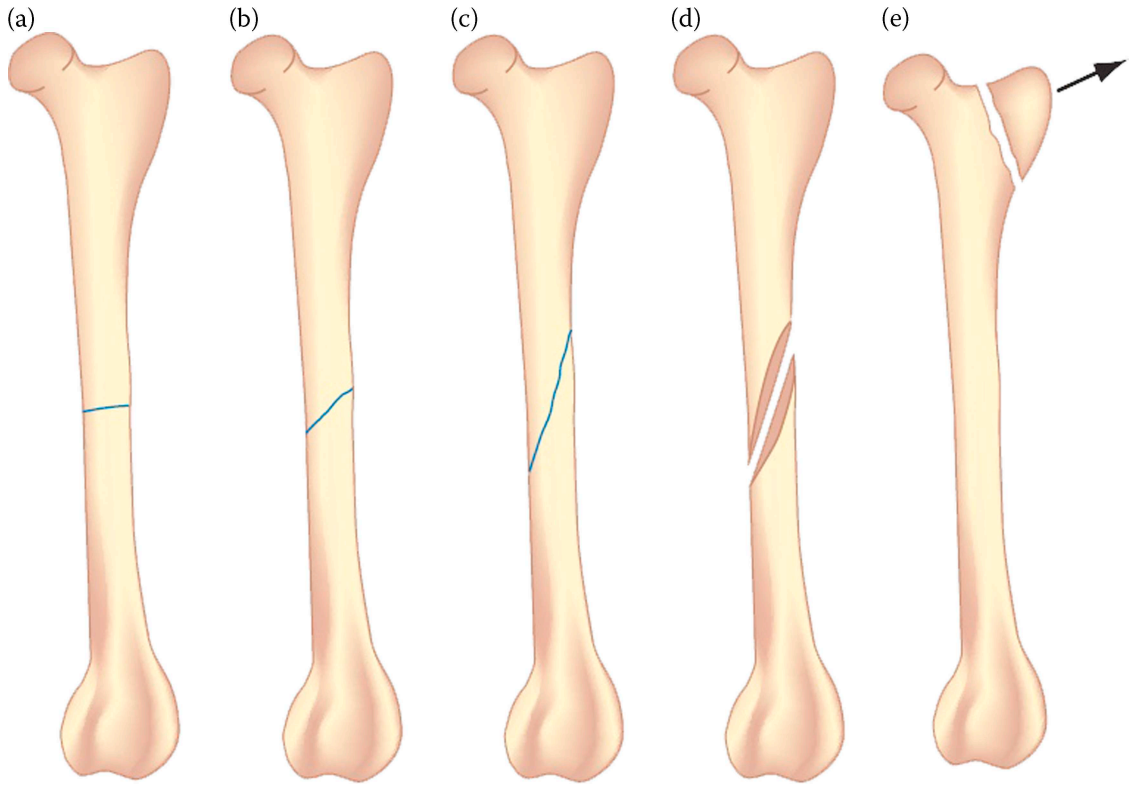


Figure 4.5 Common fracture configurations. **(a)** Transverse. **(b)** Short oblique. **(c)** Long oblique. **(d)** Spiral. **(e)** Avulsion.

The Prieur Classification System

(Anderson & Coughlan 1997, Cook et al. 1997, Messmer & Montavon 2004, Prieur et al. 1990).

In this system, the affected bones, location of fractures, fracture area and fragment number are coded similarly to the Unger system, so that each fracture is described using four numbers. The first two numbers define the fracture location (first number determines the affected bone and the second number, the location within the bone), the last two numbers the fracture type (third number defines the extent of the fracture and the fourth number, the number of fragments).

Thus, a fractured defined as 3.2.1.2 is fracture of the femoral (3) shaft (2), only involving less than 5% of the bone length (1), producing only two fragments (2).

A classification of pelvic fractures, using a more elaborate coding system, has also been reported for small animals. More specific to particular bones, canine scapular fractures have been classified according to biomechanical principles, a non-numerical classification of sacral fractures has been described for dogs and cats and, also, systems have been described for the canine accessory carpal bone, canine central tarsal bone and so forth. Still, there is no general fracture classification specific for cats and clinicians still rely on descriptive terminology for their descriptions of fractures.

FRACTURE DECISION-MAKING:

(Cockshutt 1995, Payne 1995, Radash 1999, Roush & McLaughlin 1998, Schrader 1994).

Fractures are diagnosed based on the cat's history, physical examination findings and radiographs of the affected limb. In many cases, fracture stabilization is delayed to allow treatment of trauma-related injuries, such as shock, hypothermia, cachexia, external wounds or haemorrhage, thoracic injuries, abdominal injuries, neurologic injuries and concurrent orthopaedic injuries (see Chapter 3). Two orthogonal radiographic views of the fractured limb are obtained to document the type and location of the fracture and gather information on feasible repair options. Sedation or short-duration general anaesthesia may be required to obtain radiographs without causing pain, and this may be necessary in non-cooperative patients since manual restraint to obtain radiographs is not legal in some countries.

If necessary, radiography should be delayed until the cat is stable. In some instances, a single radiographic view of the fracture site can be obtained without sedation and can be used to confirm the fracture location, help determine appropriate repair options and provide the owner with an estimated cost for treatment. The second view is then obtained prior to treatment. Oblique views are occasionally helpful to identify the presence of articular fractures or to further delineate fracture configuration. In complicated fractures, especially in articular or juxta-articular locations, the improved images provided by a computed tomography study may prove very valuable to determine the extent, configuration and severity of the lesion.

Fractures occur when intrinsic or extrinsic forces are applied that exceed the material and structural properties of the bone. The configuration of the fracture depends on the magnitude, speed and direction of the force applied. The bone's inherent strength and any loads applied to the bone at the time of injury also influence the fracture configuration. Compressive, tensile, bending, torsional and shear forces may act alone or in

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Figure 4.6 Lateral radiograph of the tarsal area of a cat involved in an RTA, showing a Grade III open, comminuted fracture-luxation of the tibiotarsal joint, with a distal fibular fracture and substantial loss of talar bone.

combination to disrupt the support function of the bone (i.e. cause fracturing) and may cause instability once the fracture is repaired (Figure 4.8). Successful fracture repair requires knowledge of regional anatomy (including blood supply), an understanding of basic bone healing physiology, information on the fracture type and the forces acting at the fracture site, and familiarization with the advantages and limitations of all available repair methods. Each repair method chosen must provide adequate stabilization while preserving blood supply and soft tissue attachments to the bone. When selecting the most appropriate repair method for a given fracture, the surgeon must understand the ability of each available fixation technique to neutralize disruptive forces (Table 4.1). Bone plates, external fixators and interlocking nails can counteract all disruptive forces when properly applied. Intramedullary pins do not adequately control rotational forces when used alone, so cerclage wires or an external fixator should be added to provide adequate stabilization.

External coaptation devices may reduce forces applied to the fracture site (depending on the fracture location and configuration) but may not completely neutralize disruptive forces and may produce substantial problems associated with excessive pressure to soft tissues and bony prominences, vascular compromise to distal limbs and joint stiffness due to prolonged immobilization. External coaptation in cats is particularly problematic due to the elasticity of the feline skin and subcutaneous tissues, making splints, etc. difficult to maintain in position for extended periods of time. In comminuted fractures, disruptive forces acting at the fracture site may be quite large, particularly if weight-bearing load is not shared between the bone and the implants after fracture reduction. Fracture configuration, available implants and equipment, surgeon preference and experience, expense and owner compliance with post-operative care procedures are also considered when selecting an appropriate repair method.

FRACTURE HEALING (ALSO SEE CHAPTER 6):

(Harari 2002, Rahan 2002).

Bone healing is affected by mechanical factors (reduction, stability, post-operative activity) and biological factors



Figure 4.7 Photograph of the open fracture-luxation, after initial shaving, cleaning and debridement.

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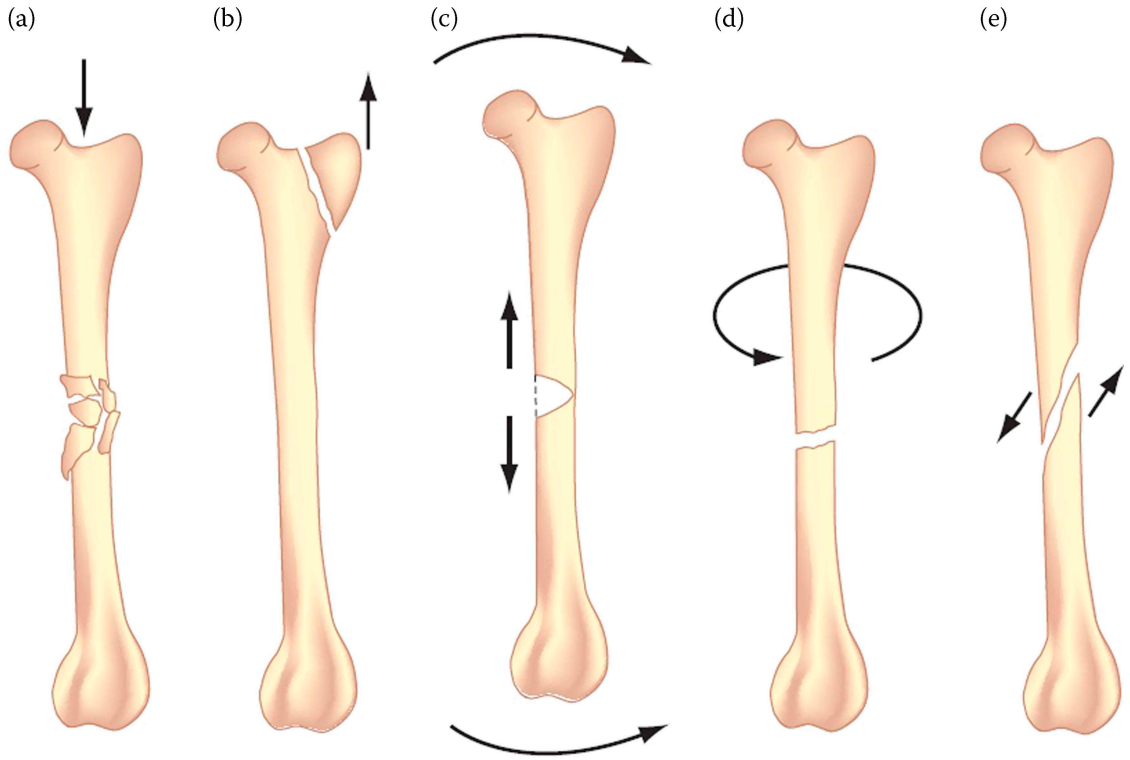


Figure 4.8 Forces acting on bone. **(a)** Compression (collapse and overriding). **(b)** Tension (distraction). **(c)** Bending (angulation). **(d)** Torsion (rotation). **(e)** Shear (collapse and overriding).

(blood supply, fracture location, degree of soft tissue injury). Bone healing can be classified into a number of types.

Indirect Bone Healing

(Huluth 1989, Perren & Cordey 1980, Radash 1999, Rahan Sumner-Smith 2002).

Indirect healing (secondary healing) of cortical bone is by callus formation and occurs when small gaps or instability exist at the fracture site (Figure 4.9). The bridging callus exists as medullary callus, intercortical callus and periosteal callus. Typically, indirect bone healing occurs when fractures are immobilized by external coaptation. The fracture gap is first filled with a haematoma. Pluripotential mesenchymal cells in the region then proliferate to form a 'fracture callus', comprised sequentially of granulation tissue, fibrous connective tissue, cartilage, mineralized cartilage, woven bone, lamellar bone and, finally, cortical bone. This process is regulated by the amount of interfragmentary strain placed on tissues in the fracture gap and by numerous cellular mediators, chemoattractants and growth factors. Repair tissues within the fracture gap are subjected to interfragmentary strain when motion occurs at the fracture site. Strain is defined as the ratio between the change in gap width to the total gap width during this motion. Various tissue types can withstand differing levels of strain: fibrous tissue tolerates up to 20% strain, cartilage tolerates up to 10% strain and bone tolerates up to 2% strain. Thus, the level of interfragmentary strain determines the tissue types that may form within the gap. Strain-tolerant tissues, like granulation tissue and fibrous tissue, will survive in a gap under conditions of high strain, while bone will not. Thus, strain-tolerant tissues initially form in fracture gaps where motion exists and strain is high. As interfragmentary strain is reduced by increasing callus size or strength (or by fracture fixation), less strain-tolerant tissues, such as cartilage and bone, begin to form. Clinically, when a fracture is well reduced (small gap), it is essential to provide rigid stabilization to prevent motion and high strain levels that may prevent bone formation in the gap. Excessive strain can lead to tissue rupture and delayed healing. When larger gaps are present between fragments, strain is distributed over a larger area, thus reducing the strain on individual cells. If rigid fixation is impossible due to the fracture configuration, a bigger fracture gap is preferred to distribute strain over a larger area and reduce strain on each cell within the gap. The resorption of fragment ends that occurs in fractures that are reduced, but not completely stable, has the effect of reducing interfragmentary strain. As the callus matures, there is gradual replacement of each tissue type by progressively stiffer and stronger tissue until bony union is completed. In unstable fractures, a large callus forms to provide early stability to the fracture site. The potential drawback of indirect bone healing is the slow return to function that may be associated with instability of the fragments during the healing process; this predisposes to the development of fracture disease (muscle atrophy, joint stiffness, disuse osteopenia, soft tissue adhesions).

Direct Bone Healing

Direct healing (primary healing) of cortical bone occurs when bone ends are in contact (<0.1 mm gap) and under compression, as may be achieved with bone plating (Figure 4.10). Internal remodelling of the Haversian systems unites the apposed cortical ends (rather than callus

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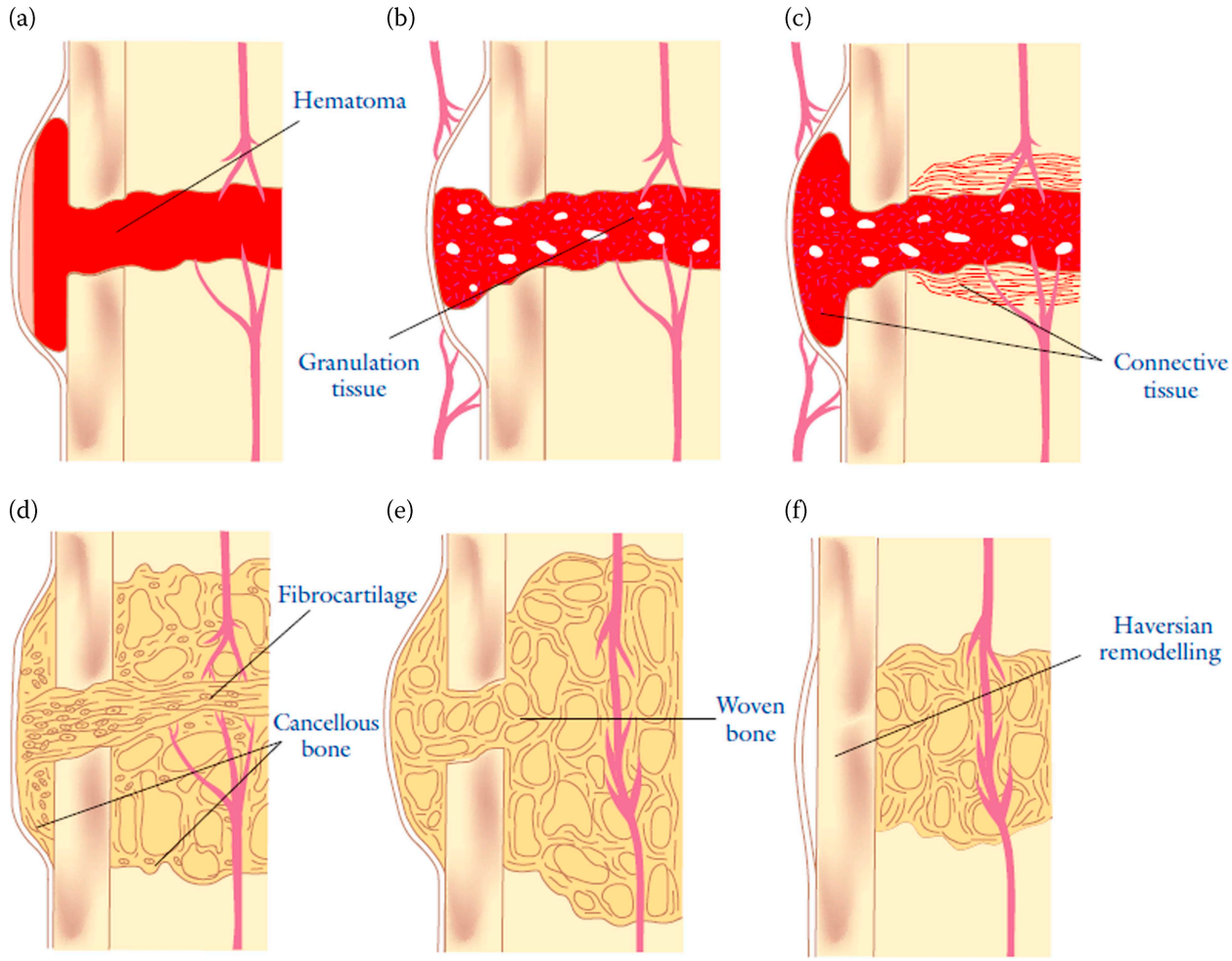


Figure 4.9 Indirect bone healing. Fracture healing occurs by progressive bridging of the fracture gap by more strain-tolerant tissues. **(a)** Haematoma. **(b)** Granulation tissue. **(c)** Connective fibrous tissue. **(d)** Fibrocartilage, which mineralizes to form cancellous bone. **(e)** Woven bone. **(f)** Cortical bone forms by Haversian remodelling and the callus is eliminated.

formation). Metaphyseal fractures involving cancellous bone heal by increased osteoblastic activity and the deposition of new bone on existing bony trabeculae. Gap healing is a type of direct healing that occurs under similar prerequisite conditions to contact healing but where the fractured bone fragments are separated (<1.0 mm gap). The gaps are filled with lamellar bone oriented perpendicular to the longitudinal axis, which is then integrated into the normal architecture of the bone during the process of remodelling.

Table 4.1: Fracture Repair Methods and Control of Disruptive Forces

	Disruptive forces acting on the fracture site				
Fixation method	**Bending**	**Shear**	**Torsion**	**Compression**	**Tension**
External coaptation (casts/splints)	+	–	+/–	–	–
Single intramedullary pin	+	+/–	–	–	–
Multiple intramedullary pins (stacked pins)	+	+/–	+/–	–	–
Full cerclage wire alone	–	+	+	–	–
Hemicerclage wire alone	–	+	+	–	+/–
External fixators (linear and circular)	+	+	+	+	+
Bone plates	+	+	+	+	+
Lag screws alone	–	–	–	–	–
Plate rod fixation	+	+	+	+	+
Interlocking nail	+	+	+	+	+

+ = good control of the disruptive force; +/– = partial control; – = poor control

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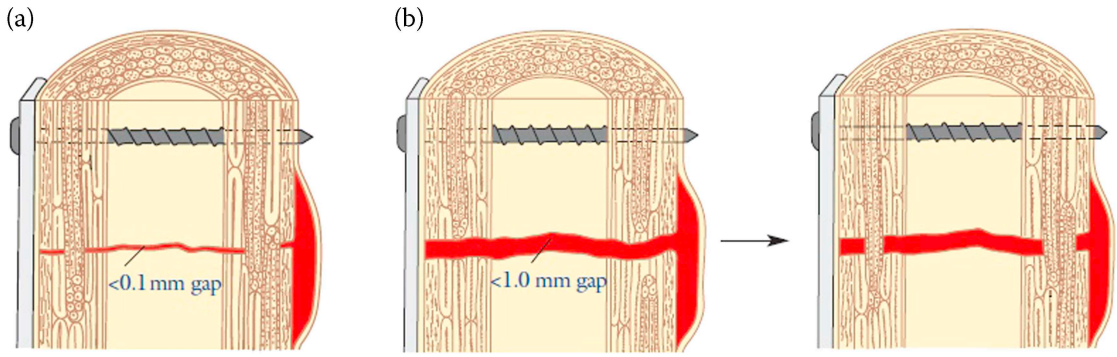


Figure 4.10 Direct bone healing. **(a)** Contact healing occurs when the bone ends are compressed together and rigidly fixed (<0.1 mm gap). Healing occurs by Haversian remodelling. **(b)** Gap healing occurs when the bone ends are apposed with a <1.0 mm gap. The gap fills with lamellar bone oriented perpendicular to the longitudinal axis, which then undergoes Haversian remodelling to restore normal bone architecture.

Although direct bone union was once considered to be the ultimate goal of fracture repair, it is now recognized that it is not a panacea. There are, in fact, a number of disadvantages associated with this approach. Rigid stabilization and compression of fractures requires an invasive surgical procedure for the application of a plate, with a consequent disruption of the blood supply and soft tissue envelope. Bone strength is regained very slowly with direct healing, so implants need to be left in place for a long time and the rigid stability they provide may protect the bone from stress and lead to osteopenia. The main advantage lies in an earlier return to function since the bone fragments are stable in compression. The limb can therefore be loaded while the bone is healing, thus fracture disease is avoided. Direct bone healing should be the goal when dealing with intraarticular fractures and simple transverse fractures of long bones where compression can be achieved readily by means of a plate.

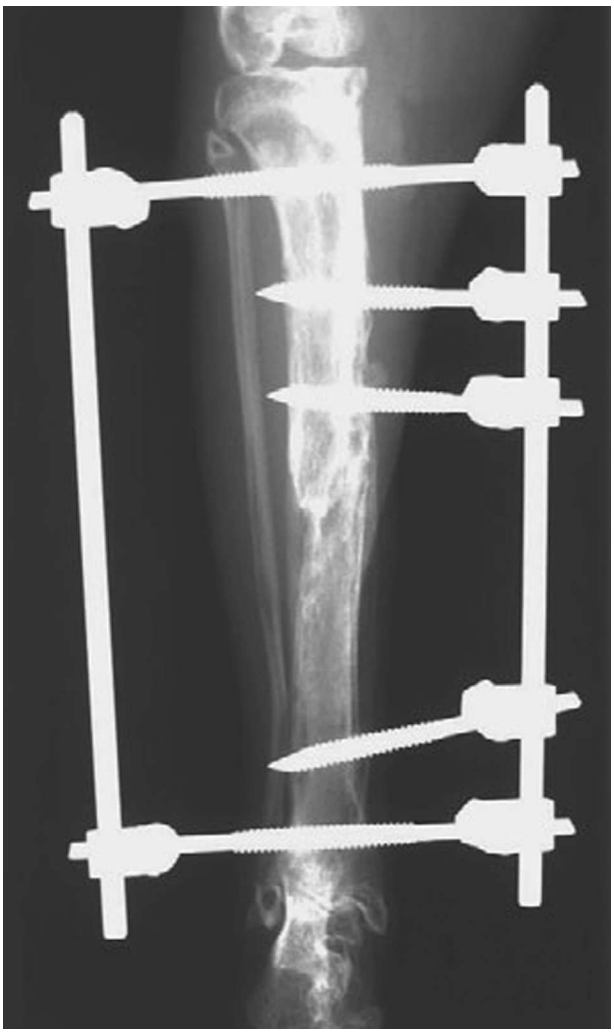


Figure 4.11 Craniocaudal radiograph of a tibial fracture stabilized with an external fixator to achieve biological fixation and bridging osteosynthesis.

Bridging Osteosynthesis

(Aron et al. 1995).

The concept of bridging osteosynthesis has arisen in an attempt to harness the rapid bone healing that occurs with indirect bone healing whilst reducing the risk of fracture disease (Figure 4.11). Bridging osteosynthesis is most applicable to comminuted mid-diaphyseal long bone fractures where there are multiple small fragments that are not amenable to reconstruction. The two ends of a fractured bone are immobilized in their correct anatomic positions relative to one another, without stabilization of the intervening fragments. Implants are applied with minimal disturbance to the fracture haematoma, the soft tissue envelope and the blood supply to the bone. Thus, interfragmentary strain is reduced and the fracture is encouraged to heal in an environment of relative stability where there is sufficient micromotion to stimulate bone callus formation. Stability can be achieved by application of an external fixator in a closed fashion or by using a minimally invasive (or biological) "open-but-do-not-touch (OBDNT) approach" in which the fracture site is inspected for reduction of the main fragments without interfering with the intermediate fragments. A locking plate (with or without an intramedullary pin) or an interlocking nail can also be applied using the same principles or even used following the principles of minimally invasive osteosynthesis (MIPO). Plates specifically designed for this purpose (bridging) generally have a central solid section to avoid placing empty screw holes over the fracture site (leg lengthening plates). The

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more recent development of locking plates has greatly facilitated the management of comminuted fractures with a more biological approach, by being able to support a fracture gap more effectively without any attempts to reconstruct the bony column.

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5 Instruments and Implants

The armamentarium of the feline orthopaedic surgeon should include instruments and implants suitable for use on small bones to optimize reduction and fixation and avoid iatrogenic injury. Manipulation of small bones necessitates delicate instruments. Fixation elements (pins, wires, screws, plates etc) are also necessarily small. The small size of fixation elements compromises the area moment of inertia and therefore stiffness and resistance to cyclical loading, causing difficulty for the feline orthopaedic surgeon. A versatile range of instruments and implants is available for orthopaedic disease in cats.

INSTRUMENTS:

Instrumentation should include those required for the surgical approach and maintenance of sufficient exposure; those required to maintain temporary fracture reduction; and those required for application of fixation elements.

Instruments for Exposure (Figure 5.1)

Scalpels, rat-toothed or Adson-Brown forceps, and Metzenbaum scissors are used for dissection of tissues. Mini periosteal elevators, such as the Freer, are used for gentle blunt separation of tissues. Small retractors are useful in the surgical approaches to bones and joints. Assistant-held mini Hohman retractors can be useful in certain circumstances to maintain exposure. Self-retaining function, such as that of the Gelpi retractor, is invaluable, even where an assistant is available, to minimize the number of hands present within limited work-space.

Instruments for Fragment Manipulation (Figure 5.2)

Feline long bone cortex is relatively thin in comparison with canine cortex and careful manipulation is indicated to prevent iatrogenic fracture/fissure. Bone holding forceps should be the correct size and should grip without crushing. The authors prefer the use of small serrated bone holding forceps. Small pointed fragment or reduction forceps, and mini Kern bone-holding forceps are also of use for the manipulation of feline long bones and small bone fragments.

Instruments for Bone Cutting

Bone-cutting instruments (rongeurs) should be sharp to avoid inadvertent crushing of bone. Many of the instruments designed specifically for dogs or adapted from human surgery are too large and cumbersome. Bone cutting using rongeurs is rarely necessary and the authors prefer to use hand-held saws (such as mini hacksaws), or air-powered or electrical sagittal or reciprocating saws to minimize bone trauma.

Instruments for Placing Screws

A power drill is required to optimize accuracy and minimize wobble when drilling holes in bone. Air-powered, electrical and battery-powered drills are available. The drill



Figure 5.1 Instruments for exposure. Adson-Brown thumb forceps (top left), scalpel (top middle) and Metzenbaum scissors (top right) for dissection. Freer periosteal elevator (bottom left) for blunt separation of tissues. Retractors include the Hohman (bottom middle, usually used in pairs either side of a bone, retracting tissue and held in position by an assistant) and self-retaining mini Gelpi distractors (bottom right).

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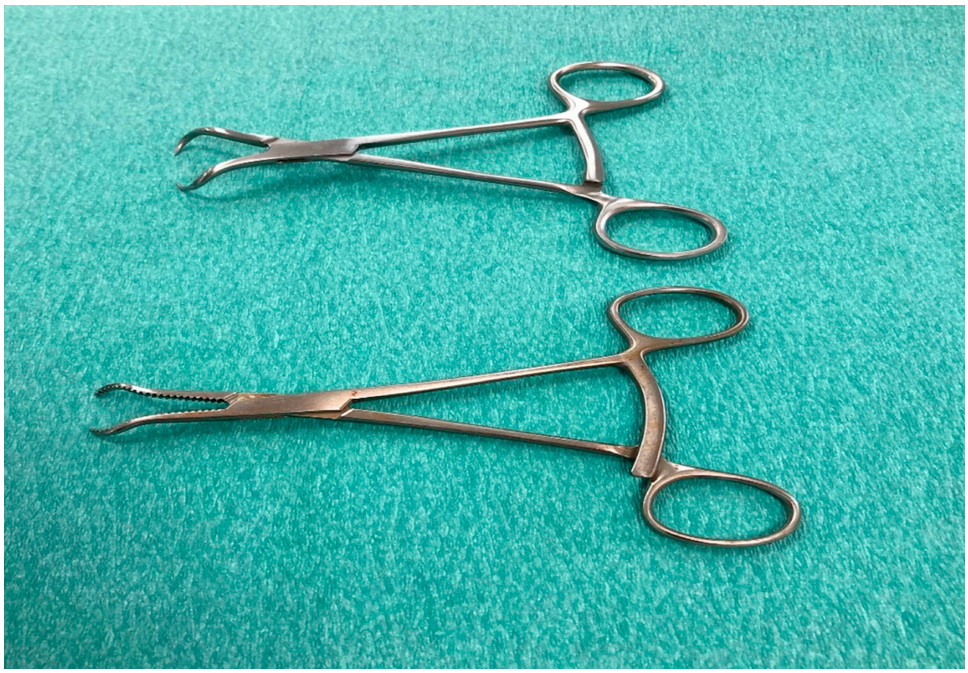


Figure 5.2 Instruments for fragment manipulation. Pointed or point-to-point reduction forceps (top) and small bone holding forceps (bottom) are useful for manipulation of bone, fracture reduction and maintenance of reduction during application of fixation.

is sterilized using steam (autoclave) or gas (ethylene oxide), or it may be placed into a sterile shroud to prevent contamination of the surgical site. Drill bits, drill guides, cortical taps, depth gauges and a screw driver may also be required. Many of these instruments are specific for the size of screw and plate being used.

Instruments for Passing and Securing Prosthetic Ligatures (Figure 5.3)

Sutures may be placed as prosthetic ligaments, for example, to augment or replace collateral ligaments of the shoulder, stifle, or hock. Such ligatures may be anchored using screws (by twisting the suture around the screw head and tying tight), bone anchors/suture screws, or through bone tunnels.

Bone anchors/suture screws of appropriate size exist for anchoring suture in cats (suture anchors of 2 to 2.5 mm diameter typically have a suture hole of around 1 mm which should be sufficient for most requirements in cats). Bone anchors may abrade suture, resulting in early failure.

Screws can be difficult to use in this situation and suture may slip off the screw head. Use of a washer may help to reduce the likelihood of slippage. After placing the suture, the screws are fully tightened, compressing the suture against bone. This should act to fix the suture in place but will also abrade the suture, potentiating early failure.

Bone tunnels allow accurate location of origins and insertions. Placing sutures through bone tunnels requires no specific equipment (although plastic or metal buttons for tying sutures may aid their placement). Where bone tunnels are utilized, passage of the suture through the tunnel is greatly facilitated by a suture passer. These simple devices consist of narrow semi rigid rods with a small semi rigid loop at one end. The rod is passed through the tunnel with the suture to be placed passed through the loop. The suture is then pulled through the tunnel quickly and simply.

Specific Instrument Sets

It is recommended to establish instrument sets for the application of certain implants. For example, intramedullary pin placement requires a kit containing a selection of pin sizes, a hand chuck and a pin cutter; cerclage wire placement requires a selection of wire sizes, a wire passer, instruments for wire twisting and wire cutters; and various plate and screw combinations may be combined into sets alongside appropriate drill guides, taps (if non-self tapping screws), drill bits and screws of various lengths (usually



Figure 5.3 Instruments for securing prosthetic ligaments. Suture screws and bone anchors are available in increasingly sophisticated design. In the absence of specific bone anchors, smalls screws can be placed and the prosthetic ligament wrapped around the somewhat prominent screw head. Bone tunnels are a further alternative.

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ascending in 2 mm intervals). Sets of specific fixation systems (plates and screws, interlocking nails, external skeletal fixators) often come from their manufacturers with a complete instrument set (and box).

IMPLANTS:

A variety of fixation implants is available for use in cats. Implants are commonly made of stainless steel or titanium/ titanium alloy. Given the array of fixation systems, particularly plating systems, now available for the cat-sized animal the authors would recommend that the surgeon has a good understanding of the requirements of his/her caseload and becomes familiar with the options prior to investing in one particular system. There follows a basic summary of implants available for feline orthopaedic surgeons.

Kirschner Wires, Arthrodesis Wires and Steinman Pins

Kirschner wires and arthrodesis wires are narrow gauge smooth stiffened stainless steel pins of circular cross-section. They are typically 120 mm long and range in diameter from 0.6 mm to 2.0 mm. Some texts refer to pins greater than 1.6 mm diameter as Steinman pins (see section on Intramedullary fixation, below), which are frequently used as intramedullary implants and are available in incremental increases in diameter to beyond 6.0 mm, with a diameter of 3.2 mm typically being the maximum for the medullary canal of most cats. Kirschner and arthrodesis wires may have bayonet or trocar points at one or both ends. Larger pins (1.4 mm to 3.2 mm) may be used as intramedullary pins for the majority of cats, and smaller wires may be used for skewering small bone fragments, sometimes in combination with tension band wires.

Orthopaedic Wire

Orthopaedic wire is available as a spool or with a preformed loop at one end in a wide range of diameters. Wire resists tension extremely well but is otherwise of limited strength. Therefore, it is always used under tension, for cerclage and hemicerclage, for interfragmentary fixation, and as a tension band. A range of diameters between 0.5 mm and 1.0 mm (24–18 American wire gauge) should be available. Gauge sizes are not universal since there are two different systems of measurement in common usage.

Implants for Intramedullary Fixation (Figure 5.4)

(Altunatmaz et al. 2012, Brueckner et al. 2016, Dallman et al. 1990, Duhautois 2003, Hach 2000, Oszoy 2004, Roush & McLaughlin 1999).

Intramedullary Pins

Steinmann pins have a smooth shaft and trochar points at both ends. Pins may be placed normograde or retrograde, depending on the bone and fracture configuration. Crushing of cancellous bone within the metaphyses provides frictional resistance to pin migration. Single smooth intramedullary pins are located in the neutral axis of the bone and therefore primarily subjected to compression forces, rather than bending forces (compare with plates which are placed remote to the neutral axis of the bone). Used alone, intramedullary pin fixation is poor at resisting axial and rotational forces. Three implant modifications are available to address this failing of intramedullary fixation: trilam nail, fully-threaded pin and interlocking nail.

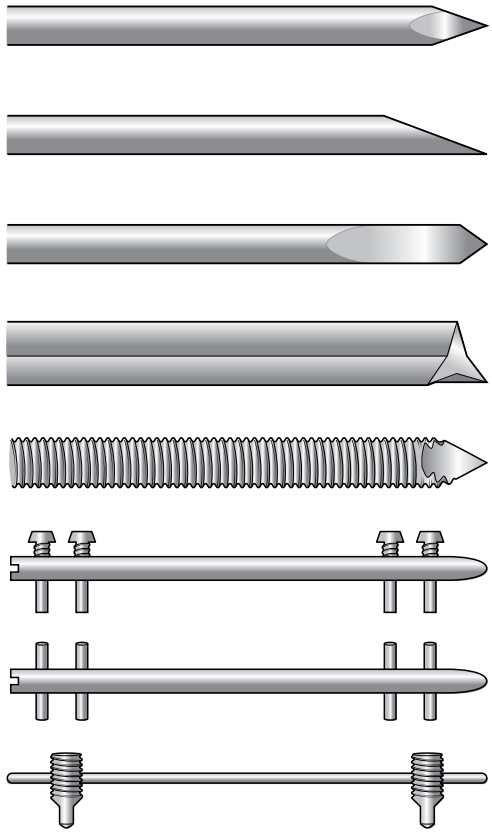


Figure 5.4 Implants for intramedullary fixation. From top to bottom: smooth pin with trochar tip; smooth pin with bayonet tip; smooth pin with bayonet tip (rotated 90° to above image); Trilam nail; fully threaded intramedullary pin; bolt-in-nail interlocking nail (IAP); bolt-in-nail interlocking nail (BioMedtrix); nail-in-bolt interlocking nail (BBraun).

Trilam Nail

The trilam nail is an intra-medullary pin modified by the addition of three longitudinal fins which impinge on the endosteal surface of the bone and therefore increase rotational stability. It is manufactured by Rotec Medizintechnik. A series of fracture cases in various species (including 62 cats) with trilam nail fixation recorded a major complication (pin migration) in a single case. Despite this promising report, no further studies have been published of the biomechanical evaluation or clinical use of this implant.

Fully Threaded Pin

Use of a fully threaded intramedullary pin may increase axial and rotational force resistance compared with smooth intramedullary pin fixation. Such a pin is manufactured by Serbay Ortopedi, Turkey. The cannulated nail is screwed into place normograde over a Kirschner guide wire and then screwed into the distal segment to fill the medullary canal. Two studies have been published documenting use of fully threaded intramedullary pins. The more recent study described results of 96 feline humeral, tibial and femoral fractures (and 79 canine fractures), with no major complications among the cats. Elective pin removal was performed in the majority of cases following fracture union.

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Interlocking Nail

Significant reduction in axial and rotational forces associated with intramedullary pinning may be achieved with interlocking nailing. In these systems bolts are placed proximally and distally to engage cortical bone either side of the intramedullary implant, as well the implant itself. Interlocking nails for cats are available from at least three different companies (Targon nail, B-Braun; I-Loc, BioMedtrix; Small Interlocking Nail System, Innovative Animal Products). Two systems involve a large diameter intramedullary pin through which the bolts pass (bolt-in-nail systems, in which intra-operative location of the bolt holes relies on an aiming guide), while the Targon nail involves placement of the bolts initially, with a narrow-diameter intramedullary pin passing through cannulations in the bolts, to which it is then rigidly locked with fixation screws (nail-in-bolt system). Results of interlocking nailing in cats have been published for the B-Braun (17.6% major complications in 51 cats and dogs, Brueckner et al. 2016) and Innovative Animal Products (8% major complications in 121 cats and dogs, Duhautois 2003) systems. Information regarding the BioMedtrix interlocking nail in cats remains limited at this time; however, several studies documenting biomechanical performance of the larger version of the same system have been published.

Bone Plates and Screws

(Bichot et al. 2011, Fruchter 1991, Rose et al. 2009, Suber & Basinger 2008).

Bone plates are attached with screws to the side of a bone to cross and stabilize fractures. Bone screws are made of stainless steel, titanium, or titanium alloy and consist of a head, a shank and threads. Screws may be self-tapping or non-self tapping. The difference refers to the method by which the thread within the bone (into which the thread of the screw interdigitates) is produced. Self-tapping screws cut a thread into the bone as they are placed through a drill hole while non-self tapping screws require a thread to be tapped using an instrument called a tap. Historically the technology required to manufacture self-tapping screws which cut a thread as consistently as a tap was lacking. However, modern-day self-tapping screws are reliable and invariably are the screw of choice for the authors. The thread is prepared during screw insertion by cutting flutes in the tip of the screw itself. This speeds surgery by removing the need to tap a bone tunnel, but the screw design is such that bone-purchase of the screw tip is limited; therefore, self-tapping screws should be placed with 2–4 mm protruding from the trans cortex. Self-drilling, self-tapping screws also exist but are not in frequent use in veterinary orthopaedics. Screws are inserted and tightened through torque applied with a screwdriver. Screw heads may couple with screwdrivers in a number of ways and the surgeon should ensure that the appropriate screwdriver is available (e.g., a hexagonal head screwdriver will not tighten a torx or star screw).

Screws may also be defined as cortical or cancellous. Cortical screws are designed to maximize holding in rigid cortical bone and have a thick shank with many threads per unit length. Cancellous screws have a wider thread pitch giving a more robust thread-bone interface in (weaker) trabecular bone.

Screws may be used to fix plates to bones but may also be used as stand-alone implants in positional or lag fashion. Lag screws are used to apply compression across oblique fractures. Partially threaded screws are readily placed in lag fashion because they have threads on only the distal end of the screw. Fully threaded screws may also be placed in lag fashion to achieve interfragmentary compression. Positional screws are placed across fractures to maintain reduction but without generating interfragmentary compression (Figure 5.5).

Plates and screws are broadly divided into locking and non-locking. Non-locking implants are the conventional implant in which rigid stability is created through the frictional forces achieved by compression of the plate against the bone as screws are tightened. The strength of the plate-bone interface is dependent on the torque applied during screw application. Loose screws will not contribute to friction between the bone and the plate. Therefore,

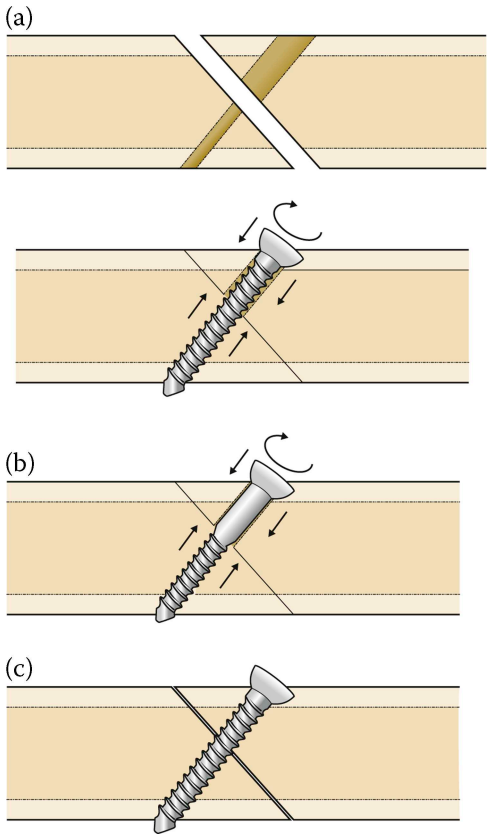


Figure 5.5 Use of screws to cross an oblique fracture. Fully threaded screws may be placed in lag fashion **(a)** the hole is drilled perpendicular to the fracture line to generate maximal interfragmentary compression (note that the optimal orientation for resistance to axial loading is perpendicular to the long axis of the bone). The cis cortical hole is drilled to a diameter equal to the outer diameter of the screw threads ('glide hole'). The trans cortex is drilled to a diameter equal to the shaft diameter of the screw ('thread hole'). As the screw is tightened the threads engage the cis cortex and generate compression of the fracture. **(b)** Partially threaded screws may also be used to achieve lag function. **(c)** a fully threaded screw placed through a hole in which the cis and trans cortex are of the diameter of the screw shaft is placed in positional fashion. The bone segments will be fixed in position (albeit with rotational instability about the axis of the screw) but not compressed.

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bicortical screws are recommended, since monocortical screws may toggle within the cis cortex and loosen. In locking implants the screw head and plate hole are locked to one another following tightening of the screw, such that the screws and plate can be considered a single unit. In this circumstance compression of the plate to the bone is not critical so the periosteal blood supply is preserved, accurate contouring is of less importance and monocortical screws are unlikely to toggle and loosen. Furthermore, there is reduced reliance on the bone-screw interface to maintain construct strength so this form of plate fixation is superior in the situation of poor bone quality.

Non-locking and locking plates are available in stainless steel or titanium/titanium alloy and come in a variety of shapes and sizes. Sizes from 1.0 mm to 2.7 mm are most useful for cats. The measurement refers to the diameter of the screws accommodated by the holes in the plate. Selection of screw diameter requires consideration of two studies demonstrating significant reductions in torsional and bending strength of bone following production of a circular defect measuring greater than 30% of the bone's diameter (Edgerton et al. 1990, McBroom et al. 1988). The diameter of a screw should be approximately 30% of the diameter of the bone to which it is applied. The shape of the hole in a plate has an impact on the function of the bone-plate-screw construct (see Box 5.1).

Conventional Plates

Dynamic Compression Plates

Dynamic compression plates (DCPs) create compression across a fracture or an osteotomy through tightening of the screw placed eccentrically (the 'load' or 'loaded' position) within an oval screw hole. 'Dynamic' compression should promote primary bone healing by abolishing interfragmentary gaps. In addition, compression increases friction between the bone ends, improving construct stability. DCPs may also be utilized with screws placed in the neutral (central) position of the screw hole, giving no interfragmentary compression (for example in a comminuted fracture where compression would not be achieved).

The limited contact dynamic compression plate (LC-DCP) has semi-circular notches in the underside between the screw holes to reduce stress concentration at the screw holes (reducing the likelihood of fracture through empty plate holes and easing contouring) and to reduce the 'footprint' of the implant, theoretically reducing compromise of the periosteal vasculature. This implant may be used as an alternative to the standard DCP.

Tubular Plates

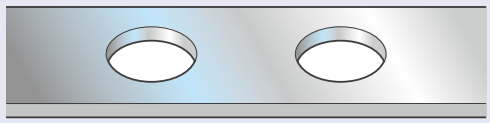
One-third tubular plates are useful in the repair of cat long bone fractures. Tubular plates have a low profile because they are relatively thin, and they fit closely to the contour of the bone surface, which facilitates closure of the soft tissues over the plate. Torsional and bending strengths are still relatively high because of their partial tubular shape.

Veterinary Cuttable Plates

Veterinary cuttable plates (VCPs) are a unique and versatile plate indicated for fixation of long bone fractures in cats. They are custom cut to the appropriate length at the surgery table. Sizes are variable and often the holes in a plate will accommodate a number of different screw sizes (e.g., some 2.4 mm VCPs may be used with 2.0, 2.4 and 2.7 mm screws). The VCP has a high density of screw holes per unit length of plate, allowing the placement of multiple screws in small bone fragments. The low profile of VCPs eases skin closure. Disadvantages of VCPs are low stiffness and strength. To

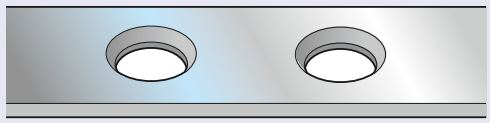
BOX 5.1 PLATE HOLES AND THEIR INFLUENCE ON BONE-PLATE-SCREW CONSTRUCT

- Round hole



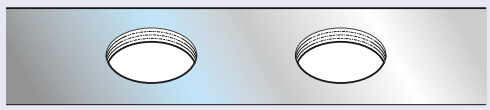
The bone hole is drilled in the centre of the round hole. Tightening of the screw achieves compression of the plate to the bone.

- Dynamic compression hole



The bone hole is drilled eccentrically such that the screw purchases bone and then translates it axially as the head of the screw engages the sloping side of the hole, potentially achieving compression in two ways: the plate is compressed to the bone and the bone slides axially with respect to the other fragment, resulting in interfragmentary compression.

- Locking plate hole



There are a number of designs of locking mechanisms such that the screw head engages the plate itself, while the screw shank threads engage the bone. When the screw is tightened, it becomes a single unit with the plate. The plate is not compressed to the bone. Often the screw head has a thread which matches a thread within the plate hole; however, other mechanisms exist and are increasingly varied and popular.

- Combination dynamic compression and locking holes.

The benefits of compression and locking may be achieved in a single hole, with various designs to achieve this.

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increase stiffness and strength VCPs can be 'stacked' i.e. applied one on top of the other.

The cuttable malleable plate is a notched version of the VCP that is similar to the reconstruction plate in that it is readily contoured to irregularly shaped bones. In addition the reduced plate to bone contact theoretically reduces the impact on periosteal vasculature.

Locking Plates

Locking plates appropriate for use in cats are available from various manufacturers with screw sizes ranging from 1.0 mm to 2.7 mm. Variation comes in the form of the locking mechanism, plate profile, plate and screw material and plate design. Locking mechanisms include:

1. Threaded screw head and matching threaded screw hole,
2. Threaded screw head which cuts a thread in the screw hole
3. Conical coupling or Morse taper (the screw head has a taper slightly less than the screw hole, such that tightening results in a frictional force between the two).
4. A single system in which the screw thread adjacent to the head engages part of the screw hole and the head itself then undergoes a frictional coupling with the hole.

Some angulation of the screw is possible with the second design, while the angle between the plate and the screw is dictated by a specific guide necessary to ensure proper locking for the other mechanisms. The only way for the surgeon to direct screws in these systems is through contouring of the plate such that the screw holes are appropriately directed.

Pancarpal and Pantarsal Arthrodesis Plates

Pancarpal and pantarsal arthrodesis plates appropriate for use in cats exist. Conventional and locking implants exist, though interfragmentary compression is considered extremely important in arthrodesis, so locking implants typically also contain some holes which accept conventional screws with a dynamic compression function. Arthrodesis plates typically consist of hybrid plates, accommodating two different screw sizes in the same plate, the smaller screws typically going into the metacarpal(s) or metatarsal(s) and the larger screws in the radius or tibia. Pancarpal arthrodesis plates range from 1.0/1.5 mm to 2.0/2.7 mm (the latter plate also accommodating 2.4 mm screws rather than 2.7 mm if required). Partial tarsal (or intertarsal) arthrodesis plates range from 1.5/2.0 to 2.0/2.7 mm. Pan tarsal arthrodesis plates consist of medial and dorsal designs, and range from 1.5/2.0 mm to 2.0/2.7 mm. Pancarpal arthrodesis in cats is typically performed with a joint angle of approximately 170° (i.e. 10° extension), while pantarsal arthrodesis is usually performed with a flexion angle of 110°–120°.

Reconstruction Versus Straight Plates

Reconstruction plates have small notches on each side of the plate between the oval screw holes. As a result they may be contoured in three planes and thus can be made to conform to irregular bone surfaces. Whilst this improves their versatility, they are relatively less stiff than a straight plate in which these notches are not present.

Selecting the Appropriate Plate and Screw Size

(Edgerton et al. 1990, McBroom et al. 1988, Muir et al. 1995).

Area moment of inertia is essentially a measure of stiffness of an object of homogeneous material. For objects with a circular cross-section (screw, Kirschner wire or Steinman pin) the area moment of inertia is proportional to the radius to the fourth power (area moment of inertia = $\pi r^4/4$). For objects with a rectangular cross-section (plates) the area moment of inertia is proportional to the base (thickness parallel to the bone surface) multiplied by the height (thickness off the bone) to the third power (area moment of inertia = $bh^3/12$). Increasing screw diameter or plate thickness will dramatically increase the area moment of inertia, and therefore the stiffness of the implants. While larger implants are desirable for their stiffness and strength, there are limits. There is some evidence that excessively stiff fixation may either suppress fracture healing (by removing strain altogether) or increase the strain at the bone-screw interface resulting in screw pull-out or failure of the bone-screw interface. The appropriate screw size is selected based on the size of the bone. Screws should be selected such that the diameter of the shank approximates to 20%–30% of the bone diameter, since studies have shown a dramatic reduction in bone strength in bending and torsion with circular cortical defects greater than 30% of the bone diameter. Plate size is determined by screw size (i.e. a 2.4 mm plate accepts 2.4 mm screws). The 20%–30% rule also applies for other implants engaging bone, such as the transfixation pins of external skeletal fixation.

External Skeletal Fixators

(Anderson et al. 1993, Anderson et al. 1996, Anderson et al. 1997, Aron et al. 1986, Egger et al. 1986, Griffin et al. 2011, Jaeger et al. 2005).

Transfixation pins engage the bone, penetrate adjacent soft tissues and are secured to an external frame which consists of one or more connecting bar(s). Smooth pins, end-threaded pins and centrally threaded pins are available (Figure 5.6).

Smooth transfixation pins (Kirschner or arthrodesis wires) are to be avoided, since they permit translation of bone segments along their length, a potential contributor to instability, which is avoided with threaded pins. Threaded pins may have either a negative or a positive profile. Negative profile pins (Ellis pins) are those in which the threads are cut into the pin surface. The junction between the smooth and threaded portions of pin is a stress riser susceptible to failure. Therefore, when using negative profile pins the pin is placed such that the junction between the smooth and threaded portions of the pin is protected within the medullary cavity. Nonetheless, this technique does not always protect pins from failure. Positive profile pins are those in which the threads are added to the shank, eliminating the stress riser at the interface between the threaded and non-threaded portions of the pin. Positive profile pins also fail through premature pin loosening less frequently than negative profile pins. A relatively recent innovation is a negative profile pin in which the transition from threaded to non-threaded pin is gradual (tapered run-out) reducing the stress riser present normally at this transition.

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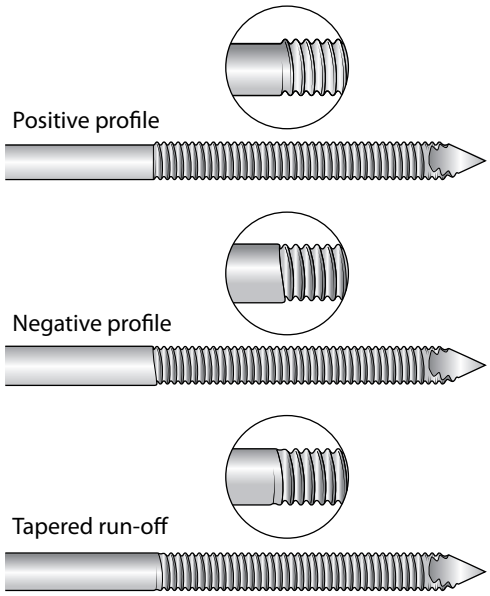


Figure 5.6 Transfixation pins. Pins may have smooth shafts, though these are rarely used except for very small bones, such as the metacarpals or metatarsals. More frequently transfixation pins are threaded.

Transarticular external skeletal fixation is the temporary bridging of a joint using an external frame. The addition of a hinge at the level of the joint to form a hinged transarticular external skeletal fixator may avoid many of the detrimental effects of prolonged joint immobilization by allowing joint motion (albeit in a single plane) and early weight bearing.

The diameter of transfixation pins used for linear external skeletal fixation is limited by the small size of the bones. It is generally accepted that there is a risk of iatrogenic fracture if the diameter of the pin exceeds 20%–30% of the diameter of the bone. Small diameter pins reduce the risk of iatrogenic fracture, but there is a trade-off in terms of reduced stiffness and strength of the individual pins and the frame overall (stiffness is greater with larger diameter transfixation pins). The concern of lost stiffness with small diameter fixation elements is of particular relevance for external fixation because the working length of transfixation pins (the distance from the point of bone purchase to the point of frame engagement) is greater than, for example, a screw in a plate. Pins 0.9–2.4 mm in diameter are most often used in cats. The metaphyseal regions of some long bones (the proximal humerus and tibia and the proximal and distal femur) may accommodate somewhat larger diameter pins. Pin diameters should always be assessed from preoperative radiographs and size selection adjusted accordingly. Larger diameter pins may also be used, with less risk of fracture where the pins are introduced into the metabones because the stress is distributed through multiple bones. The metatarsals are larger than the metacarpals and can, therefore, accommodate larger pins. Smaller transfixation pins are used if external skeletal fixation is used in combination with an intramedullary pin (in long bone fractures) to allow space for the pin.

Circular external skeletal fixators involve circular, semicircular, or horse-shoe-shaped rings around the bone. Narrow-gauge wires cross the rings from one side to the other, purchasing the bone in between. The rings are connected to one another using special connecting bars. There are typically two wires per ring and these should be placed to cross at right-angles where possible. Circular ESF systems for cats may involve rings of 35 mm diameter, accommodating 0.9 mm and 1.1 mm Kirschner or arthrodesis wires. 'Olive wires' have a single fixed bead in the centre of the pin which is placed deep to the tissues, adjacent to the bone, to prevent translation of the bone along the wire (in the direction of the bead). Circular ESF can be combined with linear components to form a hybrid device. Circular frames are very stiff under shear and bending loads, but they allow axial micromotion at the fracture site, which encourages healing. Circular fixators can be used to stabilize very small fragments and they are extremely adjustable after application.

TOTAL HIP REPLACEMENT:

(Kalis et al. 2012, Liska et al. 2009, Marino et al. 2012, Witte et al. 2010).

Modular cemented hip prosthesis systems suitable for use in cats are available (BioMedtrix cemented Micro and Nano Hip Systems). Instrumentation for implantation of the prostheses is purchased as a kit. Four sizes of acetabular component (10 mm, 12 mm, 14 mm and 16 mm in diameter), and three sizes of femoral prosthesis (#1, #2, #3) are selected from according to measurements made on radiographs. Results using this implant have been documented.

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6 Fracture Fixation Methods: Principles and Techniques

- The term 'reduction' refers to attempts to restore the following features of a fractured bone:
  - Continuity of the cortices,
  - Bone length,
  - Orientation of the proximal and distal joint surfaces.
- The term 'fixation' refers to the use of biomaterials (most commonly metallic) to maintain reduction.

The fixation system which is selected is required to maintain reduction until bone healing has progressed to a point where load is borne primarily by the healed bone. Thus, the fixation system may be considered temporary, although in most cases internally applied fixation elements are left in situ. Failure of implants in feline orthopaedics is unlikely to result from acute failure with a single load (i.e. acute load to failure), rather it is more typically a result of the use of fixation systems with insufficient durability (resistance to cyclical loading) to maintain integrity until bone healing has occurred. Note that if fracture healing does not occur, implants will eventually fail, either through failure of the implant material or at the implant-bone interface(s).

FRACTURE REDUCTION:

(Schmokel et al. 2007, Von Pfeil et al. 2017).

Slow, steady, constant pressure is the most effective means of moving bone segments against the resisting forces of muscle contracture and early fibrosis whilst minimizing iatrogenic trauma. The formation of a fibrous callus from the initial haematoma may be present within a few days of the fracture, depending on the age of the cat (young = fibrosis sooner) and the region of the fracture (well vascularized such as pelvic fracture = fibrosis sooner). Muscle relaxants may be helpful but are rarely needed in cats.

The surgeon should aim to preserve soft tissue attachments to all bone fragments during reduction wherever possible to optimize the biological health of the developing callus.

Fracture reduction techniques include traction, countertraction, ligamentotaxis and toggling (Box 6.1).

A decision must be made whether to utilize an open or closed technique (i.e. with or without a surgical approach), which will influence how the bone is to be manipulated.

Open Fracture Reduction

An open approach and direct manipulation will improve visualization and aid accurate reduction but will be of detriment to fracture healing (see Chapter 4 for a discussion of the importance of the early fracture haematoma). Direct manipulation of the fracture haematoma may be avoided despite an open approach, by employing so-called open-but-do-not-touch (OBDNT) technique.

The only absolute indication for open technique is articular fracture in which anatomic reduction is essential and callus formation to be avoided (to preserve the articular surface contour). Open reduction may also be necessary in older fractures that cannot be adequately reduced closed as a result of muscle contracture or early fibrosis. The bone segments are manipulated into alignment using bone forceps and digital pressure. Levering fragments with a periosteal elevator is also helpful, although care is needed to prevent further fracturing of the bone.

Closed Fracture Reduction

Increasingly, fracture reduction is performed remotely (i.e. by manipulation of the bone ends further from the fracture). This may involve short approaches at the proximal and distal extents of the bone rather than a single long approach exposing the fracture and is termed minimally invasive percutaneous osteosynthesis (MIPO).

With closed reduction, perfect alignment of the fragments at the level of the fracture is sacrificed in exchange for preservation of the fracture haematoma (see Chapter 4 for a discussion of the importance of the early fracture haematoma). The goal is to restore rotational alignment in all three planes (dorsal, sagittal and transverse) and bone length. The better the reduction of primary bone segments is, the greater the load sharing with the fixation system is assumed to be; however, 50% overlap of the primary bone fragments is often quoted as sufficient to limit bending forces (reducing the strain environment for the healing fracture) and avoid deformity (which may impact on adjacent joint function).

Fracture reduction in closed surgery may be verified by:

1. Palpation of the bone percutaneously;
2. Visualization of anatomy remote to the fracture itself (requiring a degree of radiographic planning and familiarity with anatomy); or
3. Use of intra-operative imaging such as fluoroscopy.

Closed reduction is often performed for avulsion fragments in skeletally immature cats. Manipulation of an avulsion fragment with a Kirschner wire placed percutaneously has been described. Once the fragment is correctly located (determined by palpation or intra-operative imaging), the Kirschner wire is driven into the main bone fragment. This minimally invasive technique has been termed 'spiking' and was reported in three cats with distal tibial physeal fractures and concurrent distal fibular fractures (a major complication was seen in one case in which removal of one of two pins was necessary following irritation to the overlying skin). This technique does not allow for bending pins to avoid their impingement on the overlying skin, so appropriate case selection is required.

Temporary Maintenance of Reduction

Reduction is maintained initially using a number of techniques, depending on the fracture configuration, surgeon preference and type of approach. Techniques primarily involve bone holding forceps of various conformations. However, temporary simple external skeletal fixator frames may be considered to assist during surgery. Also, intra-medullary pins, cerclage wires and lag screws may be considered devices for temporary maintenance of fracture reduction, although if it is considered that they are contributing to construct stiffness overall they may remain place. Permanent fixation is then applied.

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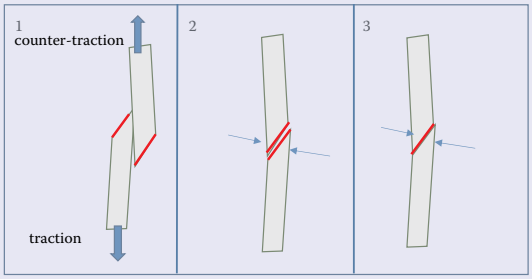
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BOX 6.1 FRACTURE REDUCTION TECHNIQUES

- **Traction** refers to the application of axial tension to the distal segment of bone.
- **Counter traction** refers to tension applied to the proximal segment of bone.

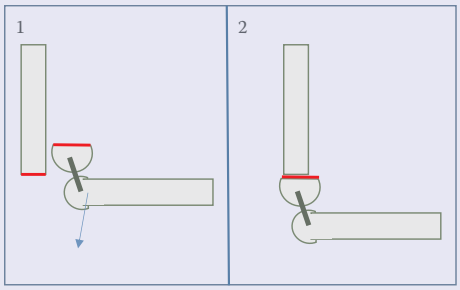
During application of traction to the limb, tension within the soft tissue envelope surrounding the fracture can guide fragments toward an anatomic position, helping with reduction. Traction/counter-traction may be achieved using gravity: the distal aspect of the limb is tied to the ceiling or an infusion stand and the table is lowered such that the patient is essentially suspended by the limb. Muscles are usually fatigued in 15–30 minutes so this technique may be employed during the surgical preparation, if surgery is to be performed.

Traction/counter-traction



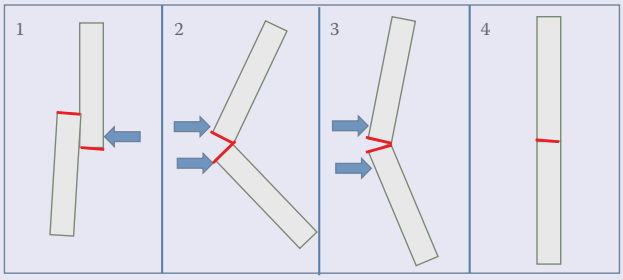
- **Ligamentotaxis** refers to manipulation of bone segments through traction applied across a joint to an adjacent bone (e.g. traction applied to the tibia in the situation of a distal femoral fracture). The applied tension is transmitted to the fracture site via soft tissues crossing the joint.

Ligamentotaxis



- **Toggling** refers to bending the bone at the level of the fracture (which reduces soft tissue tension) followed by alignment of the bone ends and then gradual straightening of the bone to reduce the fracture. Fractures must be non-comminuted and preferably transverse to minimize the shear force.

Toggling



FRACTURE FIXATION METHODS:

Fixation following closed reduction may include: external coaptation, MIPO and external skeletal fixation. Fixation following open fracture reduction may involve lag screw/cerclage wire (so-called secondary fixation devices), plates and screws, intramedullary pins/rods/interlocking nails (ILNs), Kirschner wires and external skeletal fixators (see Chapter 5 for a discussion of the available implants).

EXTERNAL COAPTATION:

(Aron & Crowe 1987, Fox 1988, Hulse et al. 1997, Meeson et al. 2011, Oakley 1999, Richardson & Thacher 1993, Tomlinson 1991).

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External coaptation refers to bandages, splints and casts and has been reported for feline fractures of the tibia, radius/ulna, and distal limb. Casts and splints should prevent rotation, bending and axial collapse of the fracture and minimize fracture strain during healing. To achieve this they must control motion of the joints proximal and distal to the fracture site.

Minimally displaced fractures respond well to external coaptation, as do fractures in younger animals, particularly greenstick fractures, in which some periosteum remains intact (but note the warning regarding physeal growth in Box 6.2). The location and configuration of a fracture dictate how effective external coaptation will be at providing stability (Box 6.2).

BOX 6.2 DECISION-MAKING REGARDING SUITABILITY OF A FRACTURE FOR MANAGEMENT WITH EXTERNAL COAPTATION

1. Location
   Limited soft tissue coverage is required such that the cast can influence bone without significant soft tissue cushioning. Therefore distal to the elbow or stifle are considered suitable locations.
2. Configuration
   Mid shaft diaphyseal fractures in which there is plenty of bone stock distal and proximal to the joint being immobilized are best, such that motion at the fracture site can be controlled.
3. Care in skeletally immature cats.
   Warning: external coaptation must bridge joints. In the growing animal this inevitably means bridging physes. Thus external coaptation can affect growth and should be changed frequently (as frequently as every second day in rapidly growing cats) and discontinued as soon as possible (or an alternative fixation method considered).

Advantages of external coaptation:

- The fracture envelope and surrounding soft tissues are not disturbed by surgical manipulation, maximally preserving the biology of the healing fracture.
- It is inexpensive (provided frequent changes are not required).
- Readily available in general practice.

Disadvantages of external coaptation:

- Potentially inadequate control of forces (with instability implicated in pain, limb disuse and delayed healing).
- Potential to interfere with physeal growth in skeletally immature cats.
- 63% of animals had soft tissue complications associated with external coaptation in one study.
- Prolonged immobilization of joints is implicated in atrophy of articular cartilage, muscle contracture and peri-articular fibrosis, with permanent joint changes being documented in some studies following immobilization for any more than 4 weeks.
- Some cats are intolerant of casts, bandages, splints and slings. They may refuse to walk for a period of time or may roll and struggle to remove the coaptation.

Casts

Full-limb casts may act as primary fixation after closed fracture reduction or as ancillary stabilization following open reduction and internal fixation. Fibreglass resin-impregnated casting tape is lightweight, easy to apply and resistant to impact. Fibreglass casts are also radiolucent, so healing can be monitored radiographically.

For crural or antebrachial fractures the cast extends from the mid-femur or mid-humerus to just distal to the metatarsophalangeal/metacarpophalangeal joints, with digits 3 and 4 exposed (to improve ambulation and allow monitoring of digital swelling or cast slippage).

Cast Care

Clients are educated to keep the cast clean and dry. It should be monitored daily for swelling of the digits, slipping of the cast and for odour. The cat should be strictly confined and must not be permitted to lick or chew the cast.

The cast should remain in place until radiographic healing is documented. However, if it becomes loose or causes irritation to the skin it may need to be removed prematurely and replaced. The cat is sedated and the cast is cut medially and laterally (or cranially and caudally) with an oscillating saw (termed 'bivalving', this technique is sometimes performed as routine, in the absence of any complications). The two halves of the cast and the padding material can then be removed and the skin treated appropriately. Padding is applied and the bivalved cast is replaced, held in position with tape. If fracture healing is almost complete and the clinician wishes to increase strain on the bone, or if skin concerns prevent replacement of both halves of the cast, only one half of the bivalved cast may be reapplied and this serves as a splint.

Splints

Various types of splint are available and may be used to stabilize fractures distal to the elbow and stifle joints. Splints are placed on the caudal/palmar aspect of the forelimb for fractures distal to the elbow. Various plastic (and less frequently metal) splints are commercially available to fit various regions of the feline forelimb and hindlimb. Alternatively, thermomoldable plastics can be used to customize a splint for each individual injury. Splints are typically placed on the lateral aspect of the hindlimb for fractures distal to the stifle. Fibreglass casting tape or thermomoldable plastics may be used to create a splint that conforms to the limb.

Care of Splints

Care of splints is identical to that recommended for casts. Splints are easier to change than casts and are particularly useful for fractures of the metacarpals, metatarsals and phalanges and when wounds are present that necessitate frequent bandage changes.

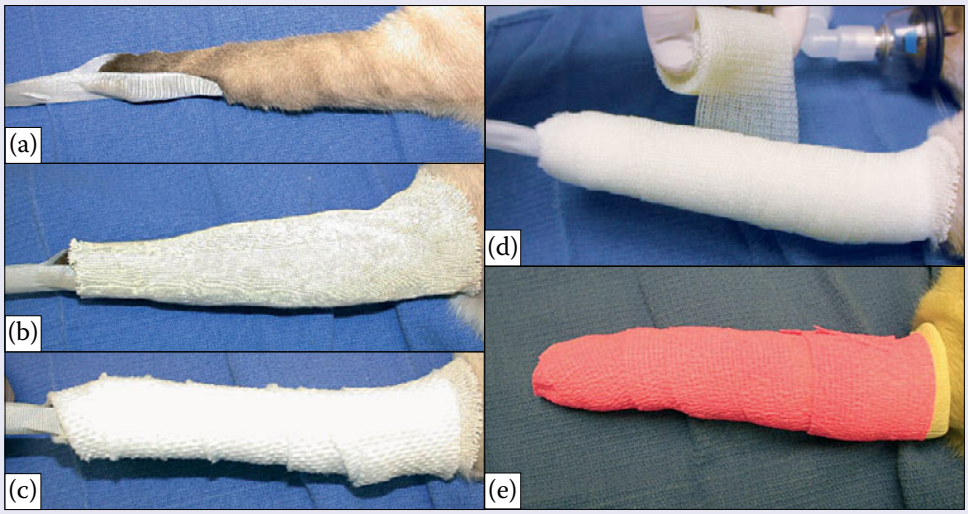
INTRAMEDULLARY FIXATION:

(Cohen et al. 2012, Forterre et al. 2007, Newman & Milton 1989, Palmer et al. 1988, Payne et al. 2005).

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BOX 6.3 APPLICATION OF A CAST

1. Long hair is removed if necessary.
2. Tape stirrups apply traction on the limb and reduce the risk of cast slipping (a).
3. A small stockinette is placed over the limb extending from the toes to the proximal extent of the cast. Folds in the stockinette may cause pressure on the underlying skin and should be avoided (b).
4. Cast padding is then wrapped around the limb from distal to proximal with each wrap overlapping by approximately 50%. Padding should protect the limb and prevent cast sores particularly over bony prominences. Excessive padding will prevent the cast material from conforming to the limb and may cause fracture instability and/or skin excoriation (c).
5. Roll gauze may be applied over the cast padding (optional).
6. The fibreglass casting material is immersed in water and squeezed to remove excess water. The cast material will harden more slowly if cold water is used versus warm or hot water. The cast material is gently wrapped circumferentially around the limb, beginning just distal to the metacarpophalangeal/metatarsophalangeal joints and progressing proximally, overlapping each wrap by 50%. Two or three layers of casting material may be used to ensure the cast is sufficiently strong. Cast application should be quick enough for the layers to interconnect for added strength. Care is taken not to squeeze the cast as it is hardening since depressions in the cast may place focal pressure on the underlying skin (d).
7. The stockinette is folded over proximally and distally to cover the ends of the cast and the stirrups are reflected and stuck to the outside of cast.
8. Elastic adhesive tape or cohesive bandage may be placed over the cast (e).



Intramedullary Pinning

Intramedullary pins are often used to stabilize long bone fractures in cats. The aim of intramedullary pinning is to engage cancellous bone in the proximal and distal metaphyses to crush trabeculae in these regions and achieve stiffness through friction. In this way, the risk of pin migration is minimized and the stiffness of the fixation is maximized. Placing an intramedullary pin in the radius is classically considered inappropriate because of the oval shape of the bone, the limited medullary cavity and the likelihood of damaging the elbow or radiocarpal joints during insertion.

Intramedullary implants are typically of circular cross-section, such that their resistance to deformation is greater than a rectangular cross-sectional implant (see Chapter 5 for a brief discussion of area moment of inertia, but remember that AMI for a circular cross-sectional object is proportional to the radius to the fourth power, while area moment of inertia for a rectangular cross-sectional object is proportional to the width to the third power).

Selection of the appropriate pin depends on:

- The size of the intramedullary cavity at its narrowest point (isthmus),
- Whether ancillary methods of fixation are to be used.

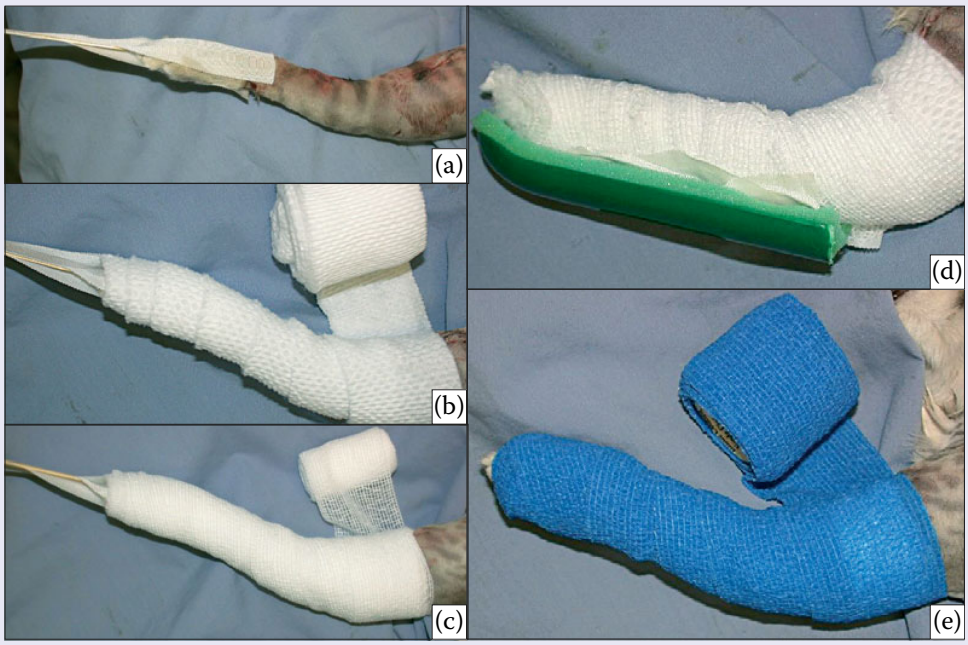
Where a single intramedullary pin is to be placed alone, it should fill 60–70% of the medullary cavity at its narrowest point. Larger pins provide greater resistance to bending forces and are preferred in straighter bones.

Modifications to intramedullary pinning techniques have been described. These modifications were intended to engage more bone to address the failure of intramedullary pins to resist torsional and axial forces. Two important techniques are: stack pinning and dynamic (Rush) pinning; and there are three specific implants: the trilam nail, fully

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BOX 6.4 APPLICATION OF A SPLINT

1. Long hair is removed if necessary.
2. Tape stirrups apply traction on the limb and reduce the risk of cast slipping (a).
3. A small stockinette is placed over the limb extending from the toes to the proximal extent of the cast. Folds in the stockinette may cause pressure on the underlying skin and should be avoided.
4. Cast padding is then wrapped around the limb from distal to proximal with each wrap overlapping by approximately 50%. Padding should protect the limb and prevent cast sores particularly over bony prominences. Excessive padding will prevent the cast material from conforming to the limb and may cause fracture instability and/or skin excoriation (b).
5. Roll gauze may be applied over the cast padding (optional) (c).
6. Commercial or custom-made splint (d) is positioned and secured to the limb with elastic adhesive tape or cohesive bandage (e).
7. Stirrups are folded up and stuck to the outer wrap.



threaded pin (which were mentioned in Chapter 5 and are not covered in any further detail here) and ILN. Plate-rod technique and the combination of an intramedullary pin and external skeletal fixation (with the pin tied into the frame or not) also aim to address the failing of intramedullary fixation performed in isolation.

Stack Pinning

(Dallman et al. 1990, McLaughlin 1999, Suber & Basinger 2008).

The technique of stack pinning involves placement of more than one smooth pin into the medullary canal, aiming to increase resistance to bending and rotational forces. Three studies suggest that stack pinning is not a reliable method of improving construct stability, so this is rarely considered by the authors.

Dynamic Intramedullary Pinning (Rush Pinning)

(Roush & McLaughlin 1998, Whitney & Schrader 1987).

Dynamic intramedullary pinning or Rush pinning is a modification of the intramedullary pinning technique. This technique is used primarily for fractures in the vicinity of physes. Rush pins have a hook on one end and are flat and skid-like on the opposite end. They are always placed in pairs. Each pin should cross the fracture line, glance off the endosteal surface of the trans cortex and bend back toward the cis cortex. In this way each Rush pin engages (though does not necessarily penetrate) three cortices, one distal to the fracture and two proximal to the fracture. The stability provided by pins placed in Rush fashion depends on the elasticity of the pins and friction of the bent pin against the inner cortex of the bone. Theoretically, the Rush pin technique should permit continued symmetrical physeal growth, despite crossing the physis. Kirschner wires can be inserted in 'Rush fashion'. It is useful to blunt the end, or create a ski-tip and to bend the pin a little prior to insertion to ensure that the pin seats correctly.

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BOX 6.5 PLACING AN INTRAMEDULLARY PIN

1. **Closed or open approach?**

Intramedullary pins are inserted using closed or open technique. An open approach eases optimal reduction and aids visualization of pin placement.

2. **Normograde or retrograde?**

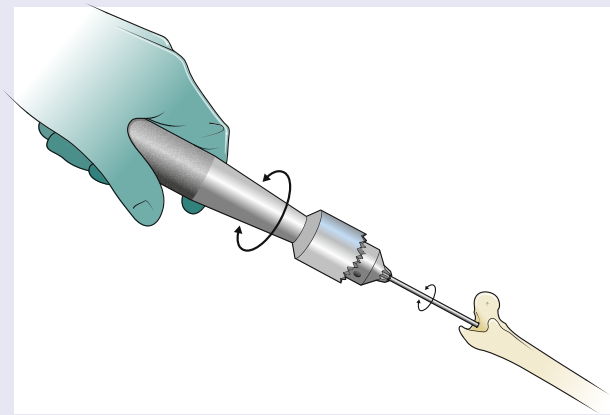
For normograde insertion, the pin is inserted from one end of the bone, across the fracture and into the opposite bone segment. For retrograde insertion, the pin is started at the fracture site and directed out of the proximal cortex of the bone. The fracture is reduced and the pin is then driven in the opposite direction and seated into the distal segment of bone. It is considered to be safer in most bones to place intramedullary pins normograde whenever possible (avoidance of sciatic nerve trauma in the femur, avoidance of ulnar nerve and articular surface trauma in the distal humerus, avoidance of patellar tendon trauma in the tibia).

3. **Pin placement**

Intramedullary pins are typically inserted with a hand-chuck, using a smooth internal and external rotation of the wrist and forearm. 'Wobble' during pin insertion will result in a bone tunnel of greater diameter than the pin itself, lost friction at the bone-pin interface and an increased risk of pin migration. Intramedullary pins may also be inserted with a power drill, but care is needed to prevent thermal necrosis of the bone and, inadvertent penetration of the cortex or into a joint. Removal of the tip (perhaps at the level of the fracture) reduces the risk of penetration of the distal joint during placement.

The pin is inserted until it engages metaphyseal cancellous bone. The depth of the pin can be estimated by pre-measuring on radiographs (perhaps of the contralateral bone) or placing a pin of the same length along the outside of the bone. Joints proximal and distal to the stabilized fracture should be manipulated through a full range of motion and evaluated for crepitus indicative of pin penetration into the joint.

Once the pin is in place, the protruding end is cut as short as possible. Alternatively, the pin can be partially precut with a saw at the appropriate length, placed in the bone and snapped off flush with the bone surface to minimize any protruding pin and reduce the risk of soft tissue impingement.



ILN fixation

(Brueckner et al. 2016, Dé Jardin et al. 2014, Dueland et al. 1996, Dueland & Johnson 1993, Dueland et al. 1997, Duhautois 2003, Endo et al. 1998, Larin et al. 2001, Lorinson et al. 2000, Macedo et al. 2017, Nabholz et al. 2019).

ILNs consist of an intramedullary pin engaged by (or engaging) bolts placed perpendicular to the bone axis, themselves purchasing cortices adjacent to the pins. The ILN resists torsional and axial forces in a way that a smooth intramedullary pin cannot. The circular cross-section and location within the medullary canal along the neutral axis of the bone result in a construct of high stiffness in comparison with plates and screws of equivalent size. ILNs can be inserted through limited approaches, minimizing disturbance of the fracture haematoma and injury to local soft tissues. The relatively long, straight bones of the cat are well suited for fracture repair using intramedullary fixation.

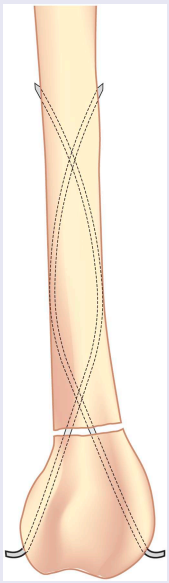
Depending on the system the cannulated nails are placed into the bone and using an insertion guide bolts are directed through the bone and nail (bolt-in-nail system), or the cannulated bolts are placed first and the nail inserted into the bone through the bolts (nail-in-bolt system). The ILNs are available in various lengths, or in the nail-in-bolt system the nail can be cut to the appropriate length.

When used for fixation of comminuted fractures, ILNs function well in bridging mode and may be placed with minimally invasive technique with no interference with the fracture haematoma. Alternatively, fracture fragments can be anatomically reduced with cerclage wires and the ILN inserted to function in neutralization mode. Typically, it is

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BOX 6.6 DYNAMIC (RUSH) PINNING TECHNIQUE

1. Drill pilot holes. Pilot holes are drilled medially and laterally near the end of the bone. In the case of the distal femur, the pilot holes are positioned on the distal-most aspect of the trochlear ridges just abaxial to the articular cartilage of the trochlear groove. The pilot holes are drilled such that they will direct the pins at approximately 20° to the long axis of the bone such that the pins will run obliquely within the medullary canal and strike the trans cortex at least half way up the bone.
2. Introduce the pins into the distal segment.
3. Reduce the fracture.
4. Advance the pins alternately into the distal segment and then across the fracture line. Pin advancement may be performed by tapping rather than twisting, to avoid penetration of the trans cortex. The pins are further advanced after glancing off the trans cortex. To achieve three points of fixation with each pin, they can be advanced until they engage the cis cortex and/or are buried within cancellous bone at the other metaphysis of the fractured bone.



preferable to avoid using cerclage wires since they are unlikely to contribute significantly to construct stiffness and their placement is costly in terms of time and local soft tissue trauma.

Destabilization of ILNs by removal of bolts from one end of the nail has been reported to increase strain by allowing dynamic loading of the fracture site. This may encourage healing.

Complications of ILNs

Complications reported with ILN fixation include breakage of the nail at the screw holes, breakage of the screws, missing the screw holes during screw/bolt placement and delayed healing. Specific concerns regarding torsional stiffness have been recognized for one of the bolt-in-nail systems (torsional instability termed 'slack' has been related to bolts moving within the cannulations in the nail) and the nail-in-bolt system in which fixation screws may allow slipping of the nail within the fixation bolts at higher strain. It is possible that reports of delayed union relate to the recognized torsional instability in one of the bolt-in-nail ILN systems. Appropriate consideration of the biological and mechanical requirements of fracture healing should help to minimize this concern with this system. The other bolt-in-nail system employs a tighter fit between the bolts and the nail, such that slack is essentially eliminated. Fracture repair using this system has shown increased rates of callus formation and remodelling in a canine tibial fracture model in comparison with the other bolt-in-nail system. There are no comparisons between the bolt-in-nail and nail-in-bolt systems.

SMALL WIRE FRACTURE FIXATION:

(Roe 1997).

Kirschner wires and arthrodesis wires are small, somewhat flexible pins shaped much like Steinmann pins. They may be applied to purchase bone fragments and fix them to other bone fragments. Cerclage wire is pliable. Its strength lies in its ability to resist tension. Therefore, it is typically applied to encircle reconstructed oblique fractures, with tension in the wire applying compression across the fracture.

Kirschner/arthrodesis wires and cerclage wire are often used in combination in fracture fixation; therefore, they are covered together in this section. The most common techniques are cross pinning and cerclage wiring. These are discussed initially, followed by combinations of these techniques for specific circumstances.

Cross Pinning

(Milton 1980, Sukhiani & Holmberg 1997).

The cross-pinning technique is commonly used to stabilize metaphyseal and physeal fractures (fractures similar to those for which dynamic (Rush) pinning might be employed). The distal femur, proximal tibia, distal tibia and distal radius are sites where cross pinning is often employed. Two Kirschner wires are inserted across the fracture line from the epiphysis into the metaphysis. The joint surface is avoided when starting the pins. The Kirschner wires should cross on the metaphyseal side of the fracture line and not directly in the fracture. In comparison with dynamic (Rush) pinning, this technique may provide a more stiff fixation, but it may interfere with any remaining physeal growth.

Cerclage Wiring

(Blass et al. 1986, McLaughlin 1999, Roush & McLaughlin 1998, Roe 1997, Wilson et al. 1985).

When applied properly, cerclage wires neutralize specific forces at the fracture site and should not compromise bone healing. Used alone, cerclage wires are unable to withstand load-bearing and muscle-generated forces. Therefore, they are typically applied in conjunction with other fixation methods.

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BOX 6.7 ILN TECHNIQUE

Planning:

Radiographs of the contralateral, unfractured bone may be used to select the appropriate diameter and length of nail. The diameter of the nail should be such that endosteal blood supply is preserved and iatrogenic trauma to the bone during insertion is minimal. Long nails should be selected so that metaphyseal bone is purchased proximally and distally to improve stability. For fractures near the ends of the long bone, bolt-in-nail ILNs with a single bolt may be used (the nail-in-bolt system typically involves only a single bolt either side of the fracture anyway).

Technique:

1. The fracture may be reduced closed or open.
2. The proximal (or distal) cortex is approached and the medullary canal is opened using the nail itself or a Steinmann pin of the same diameter.

	Nail-in-bolt system	Bolt-in-nail system
3.	Bolts are placed according to pre-operative measurements perpendicular to the long-axis of the bone. One bolt is placed proximal to the fracture and one is placed distal to the fracture. The bolts are tightened until the cannulation is central within the medullary canal.	The ILN is passed across the fracture line and seated distally without penetrating the articular surface. Pre-operative measurements or another nail of the same length will aid proper pin seating distally. As with intramedullary pinning the proximal end of the nail should be seated below the proximal cortex.
4.	The ILN is passed into the medullary canal, through the cannulation in the first bolt, across the fracture (which is now reduced), through the cannulation in the second bolt and seated distally without penetrating the articular surface.	A jig guide is attached to the nail which will direct drilling of bone tunnels through the cannulations in the bolt

5. Proper alignment in terms of torsion and bone length is confirmed

6.	Fixation screws are placed into the fixation bolts, to secure the bolts to the nail.	The fixation bolts are placed purchasing the cortices and the cannulations in the nail.

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Cerclage wires may be used alone only in specific situations, such as in the repair of mandibular symphyseal fracture/separation. The most common wire used in cat fracture repair is 0.8 mm, although 0.6 mm and 0.5 mm wires are occasionally needed. Cerclage wire is available as a spool of wire or with preformed loops on one end (loop cerclage).

Full Cerclage Wires

Full cerclage wires encircle long bones. They are used to stabilize long oblique and spiral fissures/fractures. If a full cerclage wire is placed on a short oblique fracture, shear forces will cause the wire to loosen and slide into the fracture line where it will interfere with healing. Full cerclage wires are not used on transverse fractures. A correctly applied full cerclage wire creates interfragmentary compression. There are two methods of securing a cerclage wire: twist-knot, loop-knot (which may be single or double).

Twist-Knot Cerclage

The two ends of the wire are twisted together, commonly using wire holders or a strong pair of needle holders. Traction is applied away from the bone surface as the twist is created producing equal tension to both strands of wire such that each strand of wire wraps around the other. After completing the twist, the wire is cut leaving two to three twists in place (cutting the twist reduces wire tension by up to 21%, but bending the twist over reduces tension by up to 70%). If a twist wire must be bent because of minimal soft tissue coverage at the fracture site, it is best to continue to twist the wire as the bending motion is applied. Wires secured using the twist-knot method provide only 40–60% of the static tension created when using a loop cerclage.

Loop Knot Cerclage

Loop cerclage wires are tightened by placing the free end of the wire through a preformed loop ('eye') created in the opposite end. A specially designed tightening device is available that creates maximum tension on the wire around the bone. Once the wire is tight, the free end of the wire is bent 180° to lock it in place and the excess is removed. Loop cerclage wires can be placed more tightly than twist type wires.

Double loop cerclage is similar, except that the loop is created by folding a wire in two, prior to passing around the bone. The two free ends of wire are passed through the loop and are bent over. Principles for application of cerclage wire are given in Box 6.8.

Cerclage wires applied to a bone that is conical in shape, may tend to shift toward the narrower portion of the bone, resulting in a loose wire. Placing a small Kirschner wire through the bone at the level of the cerclage wire can prevent slippage. Alternatively, a small notch may be cut into the cortical bone and the cerclage wire placed in the notch as it is tightened. If cerclage wires are used to stabilize fragments prior to application of a bone plate, the knot in the wire should be placed such that it does not interfere with proper plate positioning.

Hemicerclage Wires

Hemicerclage wires are placed through holes drilled in the bone and are tightened to create compression. Overtightening may damage the bone where the wire exits the drill hole. These interfragmentary wires should be placed perpendicular to the fracture line to maximize compression between the fragments and prevent wire movement and loosening. In most cases, hemicerclage wires provide little rotational stability and their use is limited to specific situations, such as stabilization of non-weight-bearing bones, such as the mandible or maxilla.

Complications of Cerclage/Hemi Cerclage

Complications from the use of cerclage wires generally result from the use of undersized wire, use of a single wire on a fracture, or from technical errors in application. Most technical errors result in loose wires that can migrate into the fracture lines, disrupting vascular ingrowth and delaying the healing process. Wires can become loose if they are not tightened sufficiently, are not placed perpendicular to the long axis of the bone, or if the underlying bone fragments are not reduced adequately. The wire knots should not be placed where they may damage vessels and nerves.

BOX 6.8 RULES FOR CERCLAGE WIRE APPLICATION

Cerclage wire has been implicated in fracture non-union when used incorrectly. Rules of cerclage wire use are as follows:

1. The fracture length should be at least 2.5x the diameter of the bone.
2. A minimum of two cerclage wires should be placed (except in specific circumstances, e.g. mandibular symphysis)
3. Wires should be spaced apart by half the bone diameter.
4. Complete reduction of the bone is imperative (loss of 1% of the bone diameter following placement of a cerclage wire results in a dramatic reduction in tension in the wire, rendering it non-functional).
5. Place wires perpendicular to the long axis of the bone.
6. Soft tissues surrounding the fracture must be protected and entrapment of soft tissues is to be avoided.
7. Check loops frequently. It is not uncommon for one loop to loosen while another is placed.

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Figure-of-Eight Skewer Technique (Figure 6.1)

A Kirschner wire is placed across the fracture line with the ends of the Kirschner wire protruding approximately 2 mm from each cortex. The Kirschner wire is positioned either perpendicular to the fracture line or perpendicular to the long axis of the bone, whichever will provide the greatest fracture stability. A cerclage wire is then placed in a figure-of-eight fashion around the tips of the Kirschner wire, against the outer cortex of the bone. The wire is tightened, with care taken to keep even tension on the two wire ends. The protruding ends of the Kirschner wire can be bent to help retain the cerclage wire and prevent slippage.

Pin and Tension Band Wire Fixation (Box 6.9)

Pin and tension band wiring (PTBW) is used to stabilize avulsion fractures or osteotomies of the olecranon, greater tubercle of the humerus, deltoid tuberosity of the scapula, greater trochanter of the femur, tuber calcaneus, tibial tuberosity and the medial and lateral malleoli. Distractive forces applied to the bone by tendon or ligament attachments are converted to compressive forces by the tension band fixation.

BONE PLATES AND SCREWS:

(Conzemius & Swainson 1999, Emmerson & Muir 1999, McLaughlin & Roush 1999).

Early return to limb use is expected and encouraged following fracture fixation with plates and screws. Early limb function minimizes complications such as muscle atrophy, joint stiffness and muscle contracture. It is also important in cats with multiple injuries when early use of the fractured limb is essential.

Plates and screws are available in a variety of sizes and shapes (Chapter 5). They can control all of the forces acting on the fracture site (Chapter 4). Plate and screw selection depends on the size of the bone and the fracture configuration. Specific fracture configurations may require the use of specialized plates or plating techniques. Knowledge of the basic techniques and when to use special implants or methods are the keys to successful bone plating.

Plate application requires exposure of the fractured bone, which can disrupt fracture haematoma and soft tissue attachments. Relatively recent innovations include open-but-do-not-touch (OBDNT) and MIPO to minimize the negative influence of the approach on fracture healing. Plates and screws can be applied to almost all fracture types, but successful repair still requires the use of proper surgical technique and strict attention to the principles of plate/screw fixation. In most cases, plates are not removed after healing unless complications develop.

Plating Principles

- Aseptic technique is imperative. Implant-associated infection can lead to morbidity, delayed union, nonunion, draining tracts and pain and may necessitate implant removal to address bacteria in biofilm/glycocalyx.
- The disruption of soft tissue attachments to the bone should be minimized.
- Appropriate sizes and shapes of implants should be used (the size is determined by the screw size, the length and

BOX 6.9 PIN AND TENSION BAND WIRING TECHNIQUE

1. The avulsed fragment is reduced.
2. One (or preferably two) Kirschner wires are inserted across the fracture line to maintain alignment and neutralize shear forces.

   Tips: If the pins are parallel to each other then theoretically greater compression within the fracture gap can be achieved.

   The pins should be driven until they have penetrated the trans cortex and then stopped immediately. Driving pins beyond the trans cortex and then withdrawing them to reduce the amount of protruding pin will theoretically loosen their purchase within the cortices.
3. A bone tunnel is drilled transversely across the main segment of bone.
4. Orthopaedic wire is placed in a figure-of-eight pattern through the bone tunnel and around the protruding ends of the Kirschner wires and twisted to tighten.

   Tips: it is recommended that tightening of the figure-of-eight tension band wire should involve twisting on both sides of the construct, to avoid a differential between the tension achieved (which is inevitable if a single piece of wire is used and it is twisted on either the medial or lateral side). Even bilateral twists are achieved using two pieces of wire or a single piece of wire into which a twist is developed on the side opposite the free ends.
5. The protruding ends of the Kirschner wires are bent over, cut and rotated to prevent soft tissue irritation.

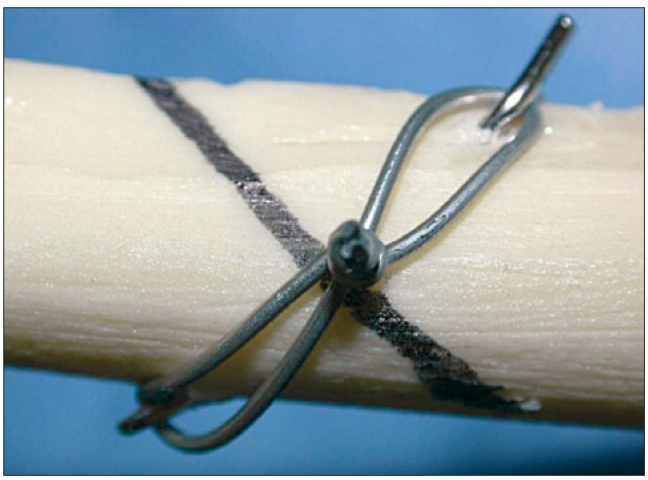


Figure 6.1 Figure-of-eight skewer technique. A Kirschner wire is placed perpendicularly across the fracture (black line). Wire is placed in a figure-of-eight fashion around the protruding ends of the Kirschner wire and tightened. The ends of the Kirschner wire are bent to prevent slippage and minimize soft tissue trauma.

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shape of the plate depends on fracture location and configuration).

- A minimum of three screws (five to six cortices) should be placed though the plate on each side of the fracture line to ensure stable fixation. Longer plates distribute strain over more of the bone and avoid placing stress riser of the end of the plate in the isthmus of the bone.

Screws

Screws are usually inserted by first drilling a hole in the bone of the same size as (or just smaller than, creating 'radial preload') the shaft of the screw. The depth of the hole is measured using a depth gauge to determine the proper screw length.

Plate Screws

Plate screws secure the plate to the bone surface. The screw passes through a hole in the plate, compressing the bone to the plate. Cortical screws are used in most cases, but cancellous screws may also be used as plate screws. A minimum of three screws on each side of the fracture lines is preferred to ensure adequate fixation. (Note: the recommendation for three screws appears to have no true evidence base. The reasoning is probably along the following lines: two screws are required to give rotational stability and a third, extra, screw is advocated in case of a complication with one of the other two).

Lag Screws

Screws placed in lag fashion compress two fragments together and provide the stability needed for healing (Chapter 5). They are commonly used to stabilize articular fractures and secure butterfly fragments in long bone fractures. Lag screws are occasionally placed through a bone plate hole (if this overlies an oblique fracture). The threads engage the trans cortex while the screw head engages the plate hole. As for any lag screw hole, the cis cortex hole is drilled to the diameter of the screw threads such that the threads of the screw only engage the trans cortex. If the screw is correctly oriented, as the screw is tightened, the two fragments are compressed. Apophyseal or avulsion fractures can also be stabilized with lag screws. The screw will compress avulsion fractures and counteract the distractive forces created by tendon or ligament attachments. Lag effect can be achieved with a fully threaded screw (the cis cortex is drilled with a drill bit matching the outer diameter of the screw threads while the trans cortex is drilled with a drill bit matching the shank diameter) or with specifically manufactured lag screws, in which through variable screw pitch in the screw threads compression is achieved as the screw is tightened.

Position Screws

(Cheng et al. 2018).

Position screws are used to maintain the relative position of two bone fragments. In isolation, position screws do not prevent rotation of fragments about the shaft. The fracture is reduced and a thread hole is drilled across both fragments. When the screw is tightened, the fragments are fixed in position by the threads engaging both fragments. Interfragmentary compression may be achieved by placing a positional screw following application of reduction forceps. Using this technique, compression may be similar to that which may be achieved with a lag screw, and it has been demonstrated that the compression may be more durable, perhaps as a result of a greater surface area of purchased bone.

Plates

(Emmerson & Muir 1999, Voss et al. 2009).

Bone plates are used in bridging, neutralization and buttressing modes.

Plate Functions (Figure 6.2)

Bridging function

Plates applied to cross defects in a long bone are used in bridging mode. When a gap exists at the fracture site such that bone fragments do not share in load bearing, the plate maintains normal limb length and joint orientation by preventing collapse at the fracture site. The plate bears the entire functional load applied through the bone until healing occurs. Plates placed in bridging fashion are often used to repair severely comminuted fractures when reconstruction of the fragments is impossible, would be too time consuming, or would significantly disrupt the blood supply to the fracture site. When this technique is employed, normal bone length is maintained, less time is spent reducing the fragments and disruption of the fracture site and its blood supply is minimized.

Screw holes over the fracture gap may be stress risers resulting in early failure of the plate, so direct superimposition of a plate hole over the fracture is avoided.

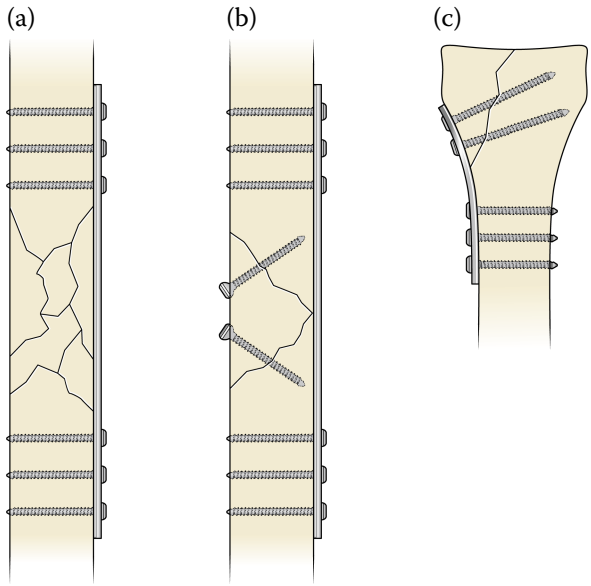


Figure 6.2 Plate functions. **(a)** in bridging mode the plate is applied to the non-reconstructed bone. As such the plate acts to conduct all load from the proximal segment of the bone to the distal segment. This is used often in the case of non-reconstructable fractures. **(b)** in neutralization mode the fracture has been reconstructed and stabilized using lag screws (pin and/or wire techniques are alternatives to lag screws) and the plate has been applied to augment the initial fixation, neutralizing all forces on the healing bone. **(c)** plates are used in buttress mode in the specific situation of an oblique (often articular or juxta-articular) fracture in which shear is resisted by the location of the plate itself.

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Specially-designed lengthening plates are also available for use in buttress fashion. The central portion of the plate is devoid of holes to prevent implant failure through empty plate holes. Overall, plates used in bridging mode must have stiffness and durability to resist strain and tolerate fatigue to which they will be subjected.

When applied in this role plates may undergo significant strain as there is no load sharing between the reconstructed bone column and the implant. As such, failure of the implants may be more likely to occur. Cuttable plates may be stacked to increase stiffness when used in bridging fashion.

Neutralization function

Plates applied to cross a fracture which has been anatomically reconstructed (e.g. using lag screws and/or cerclage wires) are used in neutralization mode. In this role the plate acts to neutralize forces which were not wholly addressed by the initial implants.

Buttress plating

A plate serves as a 'buttress plate' when used to resist shear of bone fragments in specific oblique articular fractures. The plate supports the metaphyseal portion of the bone and maintains proper joint alignment during fracture healing.

Plate and Screw Application Techniques

Dynamic Compression of a Transverse Diaphyseal Fracture

1. The fracture is reduced, avoiding disruption of the soft tissues and vascular supply (consider OBDNT and MIPO technique).
2. A plate is applied with screws proximal and distal. Consider compression to increase stiffness and minimize the fracture gap (promoting primary bone healing). The stability provided by such rigid internal fixation allows early return of limb function.

Options for Comminuted Fractures

In comminuted fractures, several options exist for plate stabilization:

- The fragments are reduced and stabilized with lag screws or cerclage wires and a neutralization plate is applied.
- If the comminuted area constitutes a small area of the overall bone length, the primary fracture ends can simply be apposed, compressed and plated, shortening the limb but not to a significant degree. Small, comminuted fragments are discarded or packed around the fracture site as bone graft.
- If the area of comminution is too great to allow shortening of the bone and too severe to permit reconstruction, a plate is applied in bridging fashion (consider OBDNT/MIPO). Fragments are left in place if they have a blood supply but may become sequestra if devoid of soft tissue attachment and left in situ following disturbance of the fracture haematoma. Fragments can be gently moved closer to the centre of a widely displaced fracture envelope using absorbable suture material. Alternatively, they can be morselized using a rongeur and used as graft to fill the defect.

Specialized Plates

L and T plates are used for fixation of metaphyseal fractures, when only a small fragment of bone exists between the fracture site and the joint. The plate allows several screws to be placed in the small fragment of bone. Holes are typically round so compression cannot be achieved. Secondary fixation (i.e. lag screw/cerclage) is complicated by the location of such fractures, so plates are typically employed in bridging mode. However, strain is low due to the juxta-articular location so plate strength is of limited concern.

For feline fractures involving small metaphyseal fragments conversion of the veterinary cuttable plate into a hook plate has been described by cutting the plate through a plate hole and bending the resulting prongs through 90°.

Hybrid 1.5/2.0 mm and 2.0/2.4 mm plates exist specifically designed for fixation of pan carpal and pan tarsal arthrodesis.

Techniques for Increasing Stiffness in Plated Fractures

(Alza Salvatierra et al. 2018, Beirer et al. 2014, Bichot et al. 2011, Craig et al. 2018, Gutbrod et al. 2017, Hulse et al. 1997, Hulse et al. 2000, Reems et al. 2003).

Plate–rod fixation (Figure 6.3)

A combination of an intramedullary pin and a bone plate has been reported and the mechanical benefits investigated. In comparison to a plate/screw repair alone, the additional intramedullary pin increases construct stiffness, reduces plate strain and increases the fatigue life of the plate.

The intramedullary pin is first inserted to restore and maintain bone length and alignment while the plate is applied. A pin that fills 35–40% of the medullary cavity is used allowing room to insert the plate screws. A bone plate is then contoured to the tension surface of the bone and applied. The most proximal and distal screws are inserted to engage both cortices of the bone (bicortical). As many as possible of the remaining screws should be bicortical, but monocortical screws may be sufficient if the pin obstructs passage of the screws. A minimum of five cortices should be engaged on each side of the fracture.

Plate-rod technique was classically reported using non-locking plates and screws, and the capacity to direct screws around the intramedullary pin is of benefit; however, plate-rod using locking plates has been investigated and is routinely employed for feline fracture management.

Plate-rod technique using a polyetheretherketone (PEEK) intramedullary pin (in which the plate screws also purchased the intramedullary pin) was reported in a biomechanical study in 2014. Although the material is less stiff than a standard stainless steel intramedullary pin, the plate-rod construct overall was stiffer because a larger pin could be placed. This technique may be useful in cats, since the narrow medullary canal in this species necessitates relatively smaller intramedullary pins.

Stacked plating

The area moment of inertia of a rectangular object is proportional to the thickness to the third power (area moment of inertia = base x height^3). Therefore, the stiffness of a construct in which two plates are stacked on top of one another is more than double that of a single plate. This technique has been described for veterinary cuttable plates. Various combinations of plate thickness, size (i.e. screw size)

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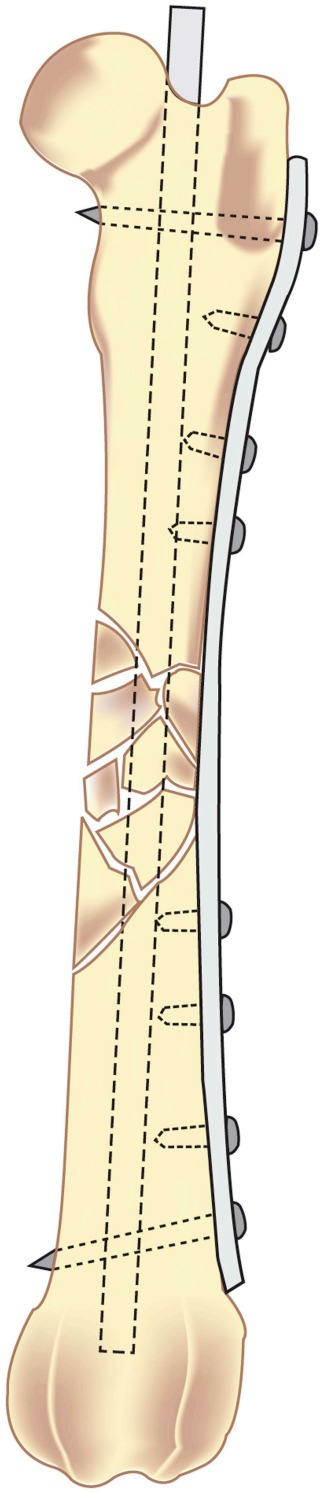


Figure 6.3 Plate-rod fixation of a femoral fracture. An intramedullary pin is combined with a plate and screws, reducing the strain on the plate itself.

and length may be employed, with the thinner, smaller, shorter plate always on top. This method gradually transmits load from the implants to the bone at the plate ends. No algorithms exist to aid in plate selection, and selection of a single thicker plate, rather than stacking two thin plates, is probably superior, since there is no additional interface between the two plates.

Orthogonal plating (Figure 6.4)

Orthogonal plate application is an extremely effective technique for enhancing the strength and stiffness of a fracture fixation. Plates are weakest in bending in the plane perpendicular to the plate height; however, they provide excellent resistance to bending in the plane of the plate width. By applying two plates orthogonal to one another the surgeon creates a construct which maximises the stiffness in all planes. Application of orthogonal plates necessitates greater disruption of soft tissues; however, this form of plating has been described via MIPO technique in cats.

EXTERNAL SKELETAL FIXATORS:

(Aron et al. 1995, Aron & Dewey 1992, Egger 1992, Farese et al. 2002, Harari et al. 1996, Johnson et al. 1989, Johnson & DeCamp 1999, Marcillin-Little 1999, Marti & Miller 1994a, b, McLaughlin & Roush 1999, Witte et al. 2014).

External skeletal fixators (ESF) are a versatile method of fracture repair. Linear, circular and hybrid fixation systems are available for use in the cat. They are easily customized into a variety of configurations to stabilize almost any type of fracture. They are well tolerated by most cats. Since they involve application of transfixation pins remote to the fracture and are eventually removed, they are often said to be ideal for stabilization of comminuted, infected and non-union diaphyseal fractures. ESF may be used as a primary means of stabilization or in conjunction with internal fixation methods. They may be applied in a closed manner to minimize disruption of the fracture haematoma and soft tissues local to the fracture. An open approach to the fracture site may be performed to facilitate reduction and alignment. Typical components of an external fixator system include the transfixation pins, clamps and connecting bars.

Transfixation Pins

(Anderson et al. 1993, Anderson et al. 1996, Anderson et al. 1997, Aron et al. 1986, Aron et al. 1991, Bennett et al. 1987, Clary & Roe 1995, Dernell et al. 1993, Egger et al. 1986, Griffin et al. 2011, Harari 2002, Langley-Hobbs et al. 1996, Langley-Hobbs et al. 1997, Palmer et al. 1992, Silva et al. 2012).

Transfixation pins penetrate the soft tissues and bone, stabilize fragments and attach to the connecting bar(s). Pins 0.6–2.5 mm in diameter may be used in cats depending on the bone's diameter. They are inserted through small stab incisions in the skin and must penetrate both cortices of the bone or they will loosen prematurely. Pins that penetrate the skin on only one side of the limb are termed 'half pins'. Pins that penetrate the skin on both sides of the limb are termed 'full pins'. A minimum of two transfixation pins are inserted on each side of the fracture line, although three (or four) pins are preferred if space allows. Knowledge of the regional anatomy and careful palpation are important to avoid neurovascular structures (hazardous corridors) or soft tissue structures (unsafe corridors). Wherever possible pins should be placed in safe corridors. When placed after open fracture reduction, pins are inserted through separate stab incisions rather than through the surgical incision. Transfixation pins 25–30% of the diameter of a bone can be inserted without significantly weakening the bone.

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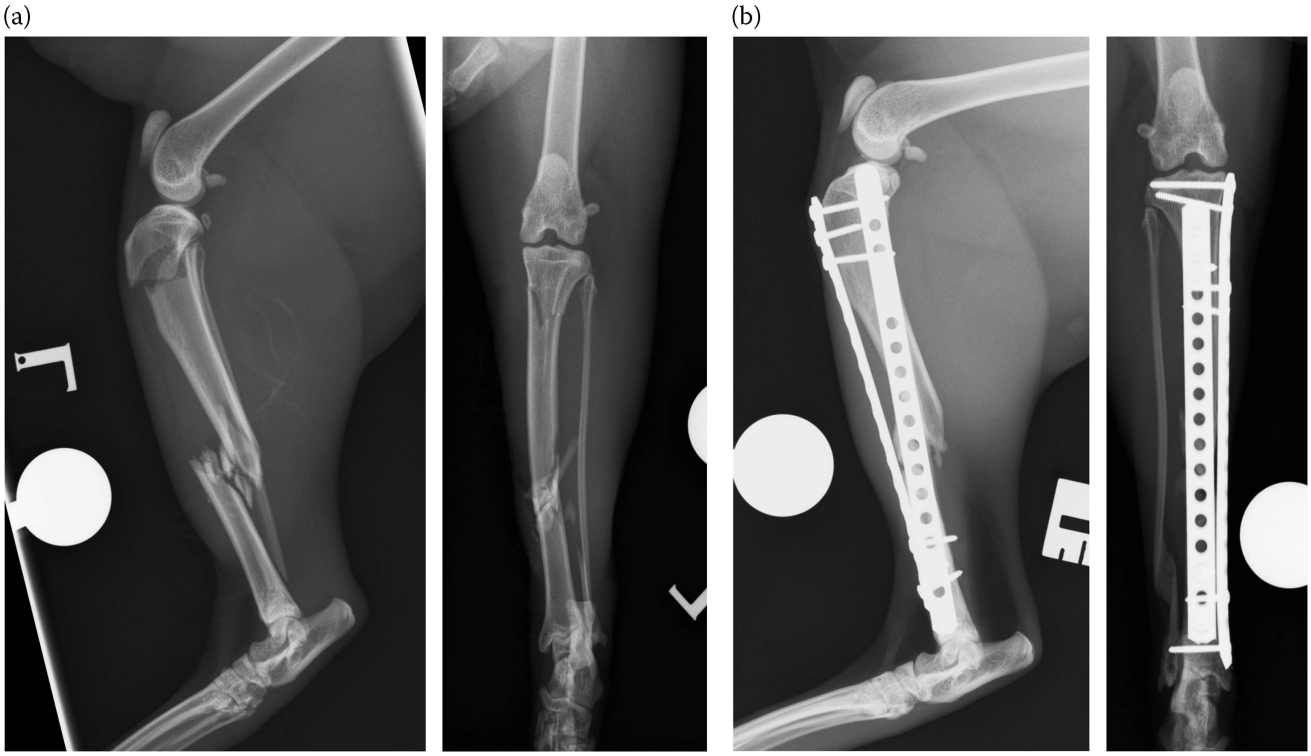


Figure 6.4 Segmental feline tibial fracture (and distal fibular fracture) consisting of proximal metaphyseal and mid-shaft diaphyseal comminuted fractures stabilized using two long orthogonal veterinary cuttable plates. **(a)** pre-operative mediolateral and caudocranial radiographic projections. **(b)** Three months following surgery involving orthogonal plate application with plate screws in the proximal metaphysis, central section of bone and distal diaphysis/metaphysis, revealing healing of tibial and fibular fractures with imperfect reduction of the mid-diaphyseal fracture in the sagittal plane (functional malunion).

A threaded pin is recommended to increase the surface area of contact with the cortex in which it is placed. Negative profile pins have an innate weakness because the transition from non-threaded to threaded portions of the pin is a stress riser. Positive profile threaded pins are recommended to avoid the stress riser; however, these must be selected with a narrower diameter (weakening the construct stiffness external to the bone) than the equivalent negative profile pin, to avoid over-sized holes in the bone. To address these concerns, an alternative transfixation pin using a tapered thread-run-out is available. This is essentially a negative profile with a gradual transition from non-threaded to threaded portions to avoid the stress riser effect.

Transfixation pins should be inserted using slow-speed power insertion (<150 rpm) with a variable speed power drill. High-speed power insertion results in thermal necrosis of the bone and premature pin loosening. Predrilling with a drill bit will reduce the risk of iatrogenic fracture and heat generation resulting in thermal necrosis, during pin placement. This will improve the health of the bone-implant interface. It is recommended to use a drill bit a size smaller than the pin diameter, such that radial tension is exerted on the bone from the shank of the implant (so-called 'radial preload'), providing increased stiffness as a result of the friction at the bone-implant interface. In certain situations predrilling may not be necessary for examples, in (relatively soft) metaphyseal bone and where fixation pins are of small diameter (i.e. less than 1.5 mm diameter). A hand-chuck may also be used to insert the pins, but the slight wobble that occurs when pins are inserted in this manner can lead to premature pin loosening. 'Backing-up' the pin once it is inserted should be avoided, as this can also cause pin loosening.

Linear Fixator Systems

(Aron et al. 1991, Beck & Simpson 1999, Bouvy et al. 1993, Brinker et al. 1985, Egger 1983, Egger et al. 1985, Foland & Egger 1991, Foland et al. 1991, Keller et al. 1994, Martinez et al. 1997, McPherron et al. 1992, Peirone et al. 2002, Roe & Thearayouk 1997, Silva et al. 2012, Tomlinson & Constantinescu 1991, Toombs et al. 1989, Willer et al. 1991, Williams et al. 1997, Witte et al. 2014, Worth 2007).

Various systems are available. A 3.2 mm diameter steel or carbon fibre connecting bar is typically used. Clamps are used to attach the transfixation pins to the connecting bar. Some clamps can be disassembled and added onto the connecting bar at any level, while some need to be slid onto the bar from the ends (necessitating a degree of forward planning regarding the number of clamps likely to be required).

Frame Configurations

Various frame configurations can be used when applying a linear fixator:

- Type Ia: A unilateral frame in which half pins are placed in a single plane.

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- Type Ib: A unilateral frame in which half pins are placed in two planes (two type Ia frames placed 60–90° to each other). The two frames can be connected to increase stiffness.
- Type II: A bilateral frame (connecting bars used on both sides of the limb) in which all of the transfixation pins are full pins (penetrating the skin on both sides of the bone).
- Modified type II (also known as type IIb): A bilateral frame in which both full pins and half pins are used.
- Type III: Bilateral and biplanar frame (the most rigid of the frame configurations).
- Tie-in configuration: A connecting bar is used to attach the external fixator frame to an intramedullary pin. This has been reported in femoral and humeral fracture repair.

Frame Stiffnes

Stiffer ESFs reduce micromotion at the fracture site. They may also experience lower rates of fixation pin loosening and pin-tract discharge/sepsis. Type III frames are stiffer than both type I and II frames. Type II frames are approximately twice as stiff as type I frames. Type I and type II frames are commonly used in cats, while type III frames are rarely required. Techniques to increase the stiffness of any fixator configuration include:

- Increasing the number of transfixation pins in each fragment (three to four pins is ideal).
- Using larger diameter transfixation pins (up to 30% of bone diameter).
- Placing the innermost transfixation pins close to the fracture site and the outermost pins near the bone ends.
- Increasing the number and size of the connecting bars.
- Placing the connecting bars closer to skin. The connecting bar is typically placed approximately 1–2 cm from the skin surface to allow for postoperative swelling. In some situations, the connecting bar is slightly contoured to fit the patient better.
- Reducing the fracture so that the fixator frame shares load with the bone segments.

Fixator Application Techniques

Preparation

A hanging-limb technique is used for preparing and draping the limb. This will fatigue the muscles, provide distraction and facilitate fracture reduction. The limb may be left hanging during the initial steps of the application process.

Type Ia Frame

1. Predrill proximal and distal bone tunnels through stab incisions (pins are not placed through an approach to the fracture) for placement of the transfixation pins.
2. Place the proximal and distal transfixation pins (half pins). Placing these pins parallel to the joint surfaces will help ensure proper alignment.
3. Empty clamps are placed on the connecting bar (to accommodate the expected number of additional pins to be placed) and the connecting bar is loosely attached to the proximal and distal pins.
4. Reduce the fracture and tighten the clamps on the proximal and distal pins. This will hold the fracture in reduction while additional transfixation pins are inserted through the empty clamps on the connecting bar (minimum of two pins per bone segment).
5. Ensure satisfactory alignment and reduction of the fracture using radiography prior to full tightening of the clamps. If necessary, minor adjustments in fracture reduction can be made by loosening the clamps and manipulating the fragments.

Type Ia ESF and Intramedullary Pin

1. Reduce the fracture and place an intramedullary pin (closed or open). A pin less than 70% of the diameter of the medullary cavity is used to allow room to insert the transfixation pins.
2. The intramedullary pin is cut as previously described to prevent impingement on soft tissue (see Box 6.5).
3. The type Ia external fixator is applied as described.

Type Ia ESF and Intramedullary Pin, Tie-in Configuration

1. Reduce the fracture and place an intramedullary pin (closed or open). A pin less than 70% of the diameter of the medullary cavity is used.
2. Leave the proximal end of the intramedullary pin is left exposed (2–3 cm) above the skin surface.
3. Place the type Ia external fixator frame as described.
4. Attach the protruding IM pin to the connecting bar using clamps (either by bending the IM pin or using an additional short section of connecting bar).

Type Ib ESF

1. Place a type Ia frame as described.
2. Place a second type Ia frame approximately 60° to 90° to the first. The two frames can be attached using short connecting bars to increase stiffness.

Type II ESF

1. Predrill proximal and distal bone tunnels through stab incisions (pins are not placed through an approach to the fracture) for placement of the transfixation pins.
2. Place the proximal and distal full pins parallel to the joint surfaces to facilitate alignment. Centrally threaded pins are used.
3. The connecting bars (with the necessary allotment of empty clamps) are clamped to the two transfixation pins on each side of the limb.
4. Reduce the fracture and tighten the four clamps. This rectangular frame will maintain reduction while further pins are placed.
5. The remaining full pins are inserted through stab incisions in the skin. To ensure the full pins contact the connecting bars bilaterally:

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a. Use an aiming device to direct the pins. Devices may attach to the fixator frame and serve as a guide to allow placement of pilot holes in the bone, or

b. Place an additional (temporary) connecting bar with empty clamps on the proximal and distal pins, forming a 'double bar' on one side of the limb. Since the three connecting bars are in the same plane, transfixation pins inserted through the empty clamps on the double bar (and then through the bone) will connect with the empty clamps on the opposite side of the limb.

6. Place at least two full pins in each bone segment (3–4 pins are preferred).
7. Tighten the clamps and remove the additional connecting bar (or it may be left in place as a double bar if additional stiffness is required).

Modified Type II ESF

1. Predrill proximal and distal bone tunnels through stab incisions (pins are not placed through an approach to the fracture) for placement of the transfixation pins.
2. Place the proximal and distal full pins parallel to the joint surfaces to facilitate alignment. Centrally threaded pins are used.
3. The connecting bars (with the necessary allotment of empty clamps) are clamped to the two transfixation pins on each side of the limb.
4. Reduce the fracture and tighten the four clamps. This rectangular frame will maintain reduction while further pins are placed.
5. Place half pins (rather than full pins) through the empty clamps on the two connecting bars.

Type III Frames

Type III frames (bilateral and biplanar) are the most rigid of the frame configurations and are rarely required in cats. However, they may be used to stabilize severe or infected fractures of the distal extremities (particularly the tibia) when delayed healing is expected. The fracture is reduced and a type II frame is placed followed by a second type Ia frame approximately perpendicular to the type II frame. Clamps and connecting bars are used to connect the two frames together.

Freeform ESF

Connecting bars may be manufactured from acrylic or epoxy resin or polymethylmethacrylate (PMMA) at the time of surgery, rather than using those of metal or carbon fibre. Freeform ESF are particularly useful in cats because they are lightweight, and they can be contoured to any shape bone or fracture configuration. Transfixation pins can be inserted anywhere along the bone and still incorporated into the connecting bar which can be of any shape. Pins of various sizes can also be used in the same frame.

The external portion of the pin is often bent or slightly notched with a pin cutter so that the rod material can grip the pin more firmly. Specially designed pins are available with a roughened surface to strengthen the interface between the pin and the rod material.

BOX 6.10 TECHNIQUE FOR PLACEMENT OF FREEFORM ESF

1. Inserted transfixation pins into the bone at locations desired for maximum stability and minimal soft tissue injury.
2. Reduce the fracture and maintain either with a temporary connecting bar or otherwise.
3. Impale flexible tubing of the appropriate diameter onto the transfixation pins to contain the acrylic as is hardens. Thin-walled, flexible tubing is preferred and rubber or polyvinyl medical tubing, paediatric anaesthetic tubing, or silastic tubing is often used. The appropriate diameter of the tubing depends on the bone being repaired, fracture configuration, frame configuration and the size of the cat. A 2-cm diameter acrylic bar is stronger than the medium sized metal bar.
4. Fill the tubing with an appropriate polymer which hardens from a liquid to a solid within a reasonable time period. Products include dental-grade nonsterile methylmethacrylates and dental acrylics, methylmethacrylates used for prosthesis implantation or hoof repair, and various epoxy putties. The material can be injected into the tubing in liquid phase using a catheter-tipped syringe. Alternatively, the material may be allowed to reach a doughy stage and is then moulded to the transfixation pins in which case tubing is not required.

Acrylics undergo an exothermic reaction during curing which can cause thermal injury to soft tissues and bone by conduction along the transfixation pins. Placing the bar at least 1 cm from the skin surface and lavaging the pin/acrylic interface with saline during the exothermic reaction reduces thermal injury.

Commercially available acrylic fixator kits are available in sterile packaging for use during surgery. When using nonsterile acrylic or polymethylmethacrylate (PMMA), care must be taken to avoid contamination of the fracture site.

Transarticular ESF

Transarticular fixators are used for joint arthrodesis and to stabilize juxta-articular fractures where insufficient bone length is available for standard fixation. When transarticular ESF are used to stabilize articular fractures, the fracture involving the articular surface is reduced and internal fixation performed, followed by application of the ESF to minimize loading of the fracture site and protect the internal repair. Triangular frame configurations can be used or the connecting bars (metal or acrylic) can be bent to immobilize the joint in a weight-bearing position. The transarticular fixator is removed as soon as possible and physical therapy is initiated to restore joint motion and minimize the impact of immobilization.

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Ideally, the joint is immobilized for a maximum of 6 weeks, though 4 weeks is preferable, to minimize permanent joint injury. Use of a hinge on trans-articular frames has been described for cats and may be useful for reducing the impact of joint immobilization by permitting a continued limited range of motion in one plane or at least allowing frequent physiotherapy sessions (when the hinge is loosened and the joint manipulated in a passive range of motion exercise before the hinge is tightened again).

Circular ESF

(Farese et al. 2002, Ferretti 1991, Lewis et al. 1998, Marcellin-Little, 1999, Witte et al. 2014).

The basic circular frame consists of metal rings, partial rings, or arches interconnected by threaded rods and nuts.

BOX 6.11 TECHNIQUE FOR APPLICATION OF CIRCULAR ESF

1. The limb is prepared.
2. One or two rings (with two wires per ring) are placed on each side of the fracture. They are selected to fit around the appropriate part of the limb with a few millimetres to spare all around, to account for soft tissue swelling.
3. Wires (1.0 mm wires are preferred in cats) are placed through the bone and attached to the rings with slotted or cannulated bolts. Tensioning of fixation wires is not required in feline circular ESF.

Many variations of the basic frame are possible, and they are readily adjusted after application, making circular external fixator systems very useful in stabilizing complex fractures, transporting bone segments to fill defects, or correcting angular and rotational deformities.

Hybrid ESF

Hybrid ESF consists of a frame in which components of a circular ESF on one side of a fracture are combined with those of a linear ESF on the other side. These hybrid frames are particularly useful when stabilizing fractures with short segments near joints or repairing fractures proximal to the elbow or stifle.

Complications of External Fixators

(Beever et al. 2017, Harari 1992, Könning et al. 2013).

Complications are common with the use of external skeletal fixation but are easily avoided or managed with proper care. Transfixation pins should not penetrate large muscles. Restriction of the patient's activity after surgery and strict owner compliance are important to prevent premature loosening at the pin–bone interface and to ensure the frame is not damaged in the patient's environment. Drainage from the pin tracts is often a sign of pin loosening. Nonunion or delayed union of the fracture may occur if appropriate provision for the mechanical and biological environment for fracture healing are not made. Delayed healing has been suggested to be a consequence of excessive frame stiffness. Staged removal of the frame can gradually transmit load to the bone and encourage healing.

BONE GRAFTING:

(Dorea et al. 2005, Heiple et al. 1987, Martinez & Walker 1999, McLaughlin & Roush 1998, Ragetly & Griffon 2011, Toombs & Wallace 1985).

Cortical bone grafts are used to fill cortical defects while cancellous bone grafts are placed around the fracture site, providing little or no structural support but acting to promote healing. Grafts may be collected from the same patient (autogenous), in which case they have the advantage of early revascularization and early osteoinduction or from another patient of the same species (allograft), which are commercially available. Cancellous grafts promote bone healing by osteogenesis, osteoinduction and osteoconduction. Osteogenesis refers to the delivery of viable osteoblasts and osteoprogenitor cells capable of producing bone. A small percentage (approximately 10%, depending on the method of collection and care of the graft material) of the transplanted bone cells survive the transfer and produce new bone in the recipient site. Osteoinduction refers to the delivery of cytokines including bone morphogenic proteins (BMPs) capable of stimulating undifferentiated cells at the fracture site to become osteoblasts and produce bone. Osteoconduction refers to the graft forming a scaffold for ingrowth of capillaries and osteoprogenitor cells from the surrounding bone and soft tissues.

Indications

Cancellous bone grafts are used where biology is shown to be or is predicted to be inferior, for example, comminuted fractures (indicating high energy injuries likely to have disrupted local soft tissues), delayed union fractures and nonunion fractures and to promote arthrodesis of joints. In fracture repair, autogenous bone graft is indicated to fill gaps at the fracture site, replace missing bone fragments, or promote healing by being placed around the fracture site. Unlike cortical bone grafts, cancellous grafts can be placed in contaminated fracture sites without concern for sequestration. Autogenous cancellous bone graft should only be placed where soft tissue coverage of the grafted region can be ensured.

Autogenous Graft Collection

An autogenous bone graft must be collected properly to maximize its effectiveness and prevent complications. The donor site(s) must be selected and prepared (clipped and scrubbed) before surgery. Several sites may be harvested if a large quantity of graft is needed. The sites selected may depend on the amount of graft required, their accessibility when the patient is positioned for fracture repair, and any morbidity associated with the donor site. In most cases, the graft is collected just before it is needed. However, if the recipient site is infected or contains neoplastic cells, the graft can be collected first to prevent contamination of the donor site. A separate surgical pack may be used to collect the graft.

In cats, autogenous cancellous graft may be collected from the greater tubercle of the humerus, the proximal tibia, the proximal femur and the iliac crest (Figure 6.5). The proximal humeral site provides more graft than the tibia and has been found to heal more quickly after graft collection.

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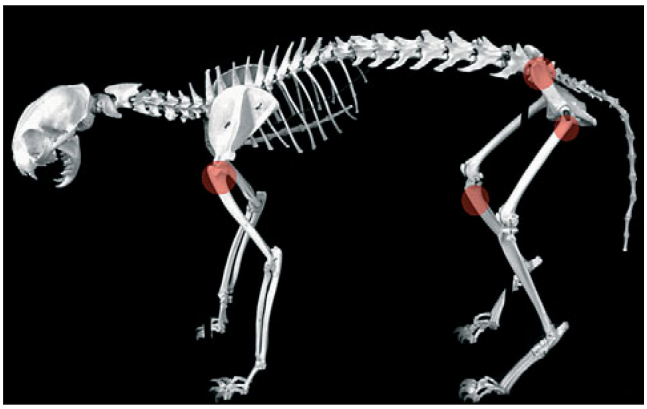


Figure 6.5 Cat skeleton showing the reported sites for harvesting of autogenous cancellous bone graft: greater tubercle of humerus, proximal tibial metaphysis (medial approach), proximal femur (lateral approach to greater trochanter) and iliac crest (approached from dorsal and offering cortico-cancellous graft which should be morselized to form small fragments).

A small incision is made in the skin and subcutaneous tissues over the donor site. A 2.5 mm drill bit is used to create a hole in the near cortex of the bone. When collecting graft from the iliac crest, a saw, rongeur, or osteotome may be used to remove the dorsal cortex and permit access to the medullary cavity (Figure 6.6). A bone curette is used to remove the cancellous bone from the medullary cavity. If harvesting from the iliac crest, the cortical bone is removed dorsally to gain access to the medullary cavity. A total hip replacement acetabular reamer (if available) can be used to acquire corticocancellous graft from the lateral aspect of the ilial wing. In some cases, small bone fragments from the fracture site can be used as corticocancellous graft.

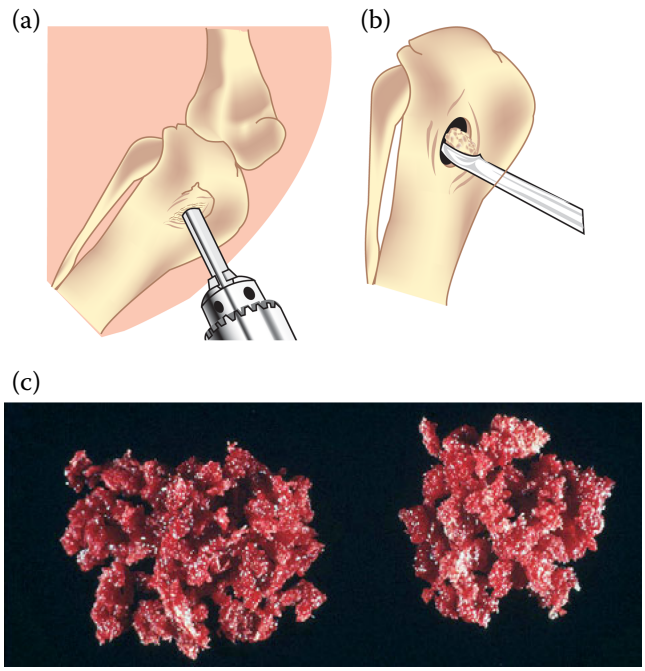


Figure 6.6 Technique for collection of autogenous cancellous bone graft (in this case from the proximal tibia). **(a)** use a drill bit to make a hole in the cortex at the chosen site. **(b)** use a bone curette to remove the cancellous bone from the medullary canal. **(c)** the normal appearance of cancellous bone.

Graft Placement

The autogenous bone graft is immediately placed in the recipient site or stored briefly by wrapping it in a moist sponge. A sponge moistened with blood is ideal. Some surgeons prefer to use the hub of a syringe. The graft should not be allowed to desiccate and should not be immersed in fluid. It is gently packed into the recipient site and the surrounding soft tissues help hold the graft in place. Once a sufficient quantity of graft is collected, the incision over the donor site is closed routinely. Allografts or graft substitutes are used as extenders for autografts in cats.

Complications

Complications after collection of autogenous cancellous graft are rare. Infection of the donor site, seroma formation, or fracture of the donor bone are potential risks but are not reported in cats. Collection of the graft may also increase operative time if a separate surgical team is not available.

AUTOGENOUS OMENTAL GRAFTING:

(Baltzer et al. 2015, Dugat et al. 2011, Ree et al. 2016, Ree et al. 2018, Saifzadeh et al. 2009).

The technique of using free omental grafts to augment bone healing in fracture management and arthrodesis has not been reported in the cat but has been reported in the dog. The technique involves a small coeliotomy to harvest a small section of greater omentum which is placed directly over a reduced fracture or arthrodesis following fixation with a plate and screws, prior to closure of the approach. In particular it was shown that healing of distal radial fractures in toy breed dogs was comparable, or superior, when omental grafting was performed in comparison with autogenous cancellous bone grafting. The situation of the distal radius in toy breed dogs may be somewhat similar to that of distal tibial fractures in cats, in that the biological environment is compromised as a result of limited surrounding soft tissue and limited internal blood supply. Therefore, omental grafting may prove useful in this situation in cats and may be considered as a useful adjunct in treatment of nonunion fractures.

IMPLANT REMOVAL:

External Skeletal Fixator

Staged removal of external fixator pins may be used to destabilize the frame gradually, thus increasing the load on the bone and theoretically preventing stress protection. This is particularly helpful in cats where rigid frame configurations have been suggested to slow healing. The purpose is to stimulate healing by gradually increasing loading of the fracture callus. More typically the entire frame is removed when fracture healing is radiographically complete. In most cases, the fixator can be removed with the patient under sedation. The clamps are removed to disconnect the connecting bar from the transfixation pins and the pins are removed individually, or, alternatively, the transfixation pins may be cut close to the connecting bar, left long enough to allow then to be reversed out of the bone. During fracture healing, loose pins may need to be removed if they cause pain and tissue reaction.

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Internal Implants

Plates, screws, intramedullary pins and wires may be removed when healing is complete, but this requires additional surgery and added expense, so is usually avoided. Complications that may warrant implant removal include migration of intramedullary pins, infection (where infection is thought to be associated with biofilm/glycocalyx), stress protection of the bone by the rigid implants, implant failure and lameness that occurs in cold weather when plates are applied to the distal extremities. Sciatic irritation after intramedullary pinning or ILN fixation of the femur necessitates implant removal. Destabilization of plated fracture repairs by removal, for example, of screws closest to the fracture, has been documented, with the aim of gradually increasing strain applied to the healing callus, stimulating healing. In the experience of the authors this is rarely necessary.

DELAYED AND NONUNION FRACTURES:

(Dugat et al. 2011, Nolte et al. 2005, Weber & Cech 1976).

Delayed unions are (somewhat vaguely) defined as those fractures that have failed to unite in the expected period of time. Nonunion fractures are those in which the bone ends have failed to unite and staged radiography confirms no progression of healing. Where nonunions represent failure of fracture healing as a result of an inadequate biological and/or mechanical environment, delayed unions are those in which the environment is only just adequate for the slow continuation of healing. The difference between the two situations is not clear-cut; however, a delayed union can be expected to heal to completion, provided the durability of the supporting fixation is sufficient, while a nonunion will not. Implant failure is the occasional result of delayed union, in which case revision surgery is required. For this reason, delayed union is to be avoided where possible. Fortunately the reported incidence of delayed/ nonunion in cats is rare, ranging 2–4.3%. Nonunions occur most often in fractures of the tibia and proximal ulna. They are more likely to occur in older cats and heavier cats. The surgeon should be aware of the potential for delayed/nonunion and efforts should be made to avoid these situations.

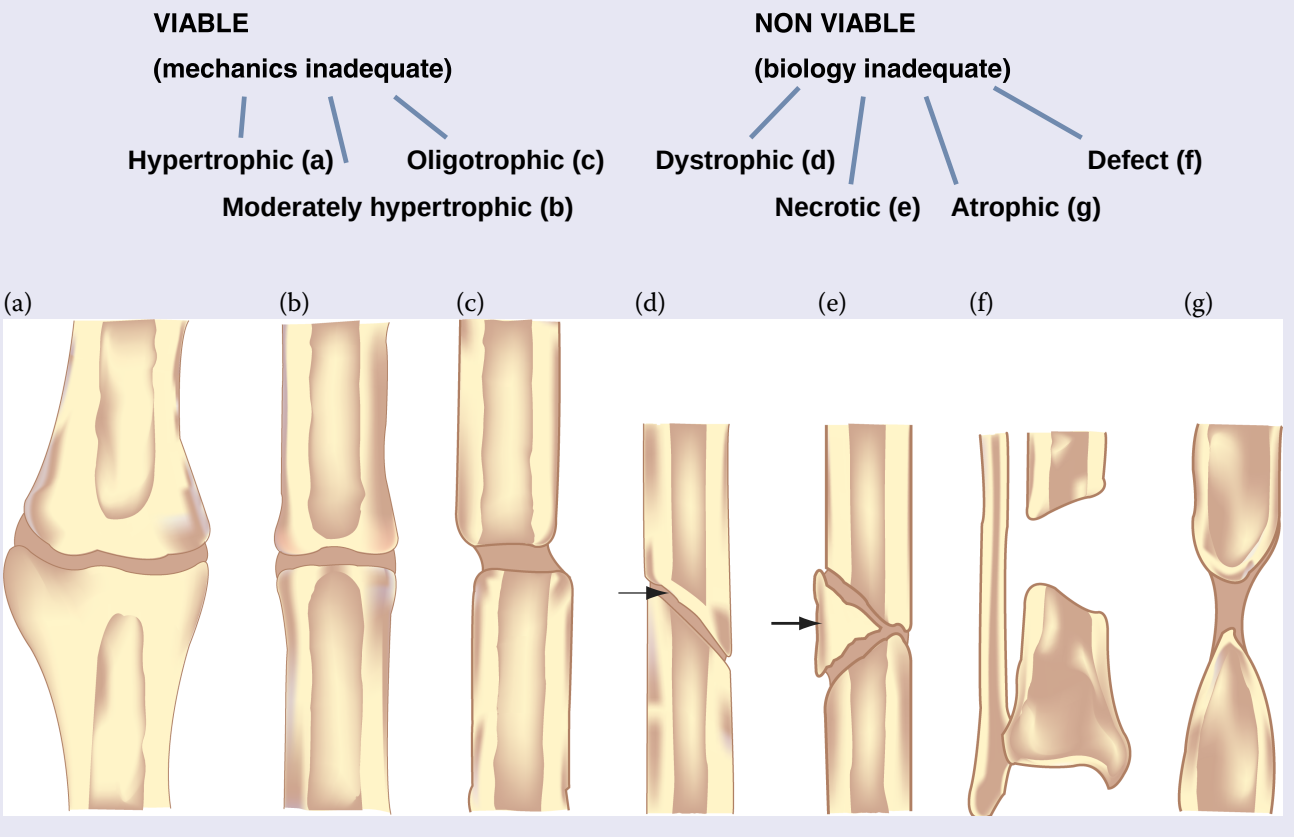
Nonunion fractures:

When a non union is recognized it is important to define the cause of the failure of healing (see Box 6.12).

Viable (Biologically Active)

- Hypertrophic (a) – generous callus is present, but a fracture gap persists. This is caused by excessive motion at the fracture site, such that strain exceeds that required for mineralization and remodelling of callus.
- Moderately hypertrophic (b) – a callus is present but is not overly large (this form of nonunion is difficult to distinguish from a hypertrophic nonunion). Instability is generally a result of selecting an inadequate fixation

BOX 6.12 CLASSIFICATION OF NONUNION FRACTURES



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method or implant selection. Premature loosening of orthopaedic implants may also lead to instability.

- Oligotrophic (c) – the fracture callus is minimal or absent, and yet the biological environment is such that healing could progress. Typically this situation is seen where unstable implants are close to the fracture site (most commonly cerclage wire). Removal of the offending implant may be enough to allow healing. Oligotrophic nonunions are distinguished radiographically from nonviable nonunions by the irregular, hazy appearance of the bone ends indicative of vascularity. This form of non union is extremely uncommon.

Non-Viable (Biologically Inactive)

- Dystrophic (d) – fragments fail to heal because of a loss of blood supply. Devascularization may be caused by the initial trauma or iatrogenic trauma during surgery, e.g. long term or excessive soft tissue retraction, desiccation of soft tissues and bone and liberal use of electrocautery.
- Necrotic (e) – fragments fail to heal because of infection or sequestration. Bone fragments will heal in the presence of infection; however, in this situation the surgeon should aim for stiff fixation, since instability is thought to hamper neovascularization, impeding the host defences.
- Defect (f) – fragments fail to heal because of an excessive fracture gap. Repair tissues are unable to bridge large fracture gaps caused by bone loss, surgical resection, or distraction. The resultant gap fills with fibrous tissue or cartilage and fails to mineralize.
- Atrophic (g) – as nonviable nonunions progress, they may become osteoporotic and lose blood supply and osteogenic activity. Resorption of the bone ends occurs.

Other Causes of Non Union

Other less common causes of delayed and nonunion fractures in cats include osteopenia, neoplasia, radiation therapy, glucocorticoid medication, metabolic abnormalities and nutritional deficiencies. Patellar fractures (Figure 6.7) have been reported in cats and may with some frequency be associated with further fractures in a syndrome referred to as KATS (knee and teeth syndrome) because of an association between proximal tibial/patellar fractures and retained deciduous teeth. The patellar fractures which occur in cats with this condition have not been reported to progress to union.

Diagnosis

Delayed union and nonunion fractures are diagnosed by radiography. On physical examination, disuse muscle atrophy, lameness, pain and reluctance to use the limb may be apparent, though in the absence of implant failure convalescence may superficially appear to be progressing uneventfully. Drainage from the fracture site may occur if necrotic nonviable nonunion is present. Radiographic findings include persistence of a fracture gap, although some signs of early healing (i.e. some mineralized callus formation) may be evident. Further radiographic features of nonviable nonunion fractures include sclerotic fracture ends, the presence of a gap between fragments, and smooth, well-defined fracture surfaces. In nonviable nonunions, the bone ends may appear atrophied and osteopenic. Varying degrees of callus are present at the fracture site in viable nonunions. Nuclear scintigraphy has been reported to determine whether bone ends are vascularized, although it is not widely utilized for this purpose.



Figure 6.7 Mediolateral radiograph of the tibia and femur of a cat with lameness ascribed to a transverse patellar fracture. Note the extent of displacement, despite extension of this stifle in the image, as a consequence of traction on the proximal fragment from quadriceps muscle contraction.

Treatment of Delayed Union

In many cases, delayed unions are managed by simply allowing more time for healing to occur. The cat's activity is restricted to delay implant failure and the fracture is monitored radiographically. If instability at the fracture site is contributing to delayed healing, additional support may be provided using an external skeletal fixator (or adding additional fixation pins and/or a second connecting bar to an existing external skeletal fixator) or external coaptation device. However, if instability is severe, an entirely new fixation method may be indicated. Autogenous cancellous bone graft can also be placed to stimulate more rapid healing by providing cytokines and growth factors such as bone morphogenic proteins (BMP) and vascular endothelial growth factor (VEGF). Infection is controlled by antibiotic therapy (based on culture and sensitivity testing) if indicated.

Treatment of Nonunion Fractures

Successful treatment of a fracture nonunion is contingent upon correcting the biomechanical and biological factors that influence bone healing. An understanding of the cause

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of a specific nonunion is vital, so radiographs should be critically assessed for the suitability of implants and for the appearance of the callus. Classifying a nonunion according to the classification system shown in Box 6.12 is of use in deciding on appropriate treatment.

Electrical stimulation has been used to promote healing of nonunions, since bone growth is associated with electronegativity and an electrical current may encourage osteoblast activity. However, the lack of clinical trials, and the additional expense, has limited the use of electrostimulation to treat nonunions in cats.

Viable Nonunions

Viable nonunions typically result from instability (oligotrophic non union is rare). Treatment therefore involves stabilization by rigid fixation. The existing fixation method may be enhanced or it may be removed and replaced with another system. Rigid internal fixation (i.e. plates and screws + - intramedullary pin) is often recommended, although external skeletal fixation can also be considered. Debridement of the fibrous pseudoarthrosis between the bone ends and opening the medullary cavity is performed if it is required to achieve adequate reduction, create freshly sectioned bleeding bone surfaces (which will create an environment of growth factors similar to that of the fracture envelope), and allow restoration of an intramedullary blood supply. In some cases, the bone ends are simply removed with a bone saw and the resultant transverse fracture is plated under compression. Sufficient callus is removed to position the plate properly and compression is applied to maximize stiffness. Stable, rigid fixation should be achieved even if compression is not possible.

Nonviable Nonunions

Treatment of nonviable nonunions requires promotion of the biological healing process. Surgery is often required. The surrounding soft tissues must be handled with care during surgery to protect what remains of the tissues from which the periosteal blood supply is derived. Following surgery, early return to function and proper physical therapy help to prevent muscle contracture/tie-down and adjacent joint stiffness. Some patients will require multiple surgeries to provide stability, place additional cancellous grafts, and remove implants.

For dystrophic nonunions establishment of the intramedullary vasculature is enhanced by opening the medullary cavity with a Steinmann pin or drill bit. Additional steps include placing autogenous cancellous bone graft at the fracture site, and drilling holes in dense cortical bone to allow vascular ingrowth.

In infected (necrotic) nonunions, rigid fixation and preservation of blood supply are very important. Initially any implants, which may be potentiating the survival of infecting bacteria through adherence and biofilm/glycoclayx formation, should be removed. Similarly, all dead bones (sequestra) must be removed using rongeurs and curettes. Infected soft tissues, draining tracts and fistulas are debrided and aerobic and anaerobic samples are collected for culture and sensitivity testing. Rigid fixation is applied. This may involve internal fixation, although in the presence of multi-resistant bacteria external skeletal fixation may be preferable, to facilitate (likely) eventual removal.

Appropriate antibiotics are administered systemically. Local therapy with antibiotic-impregnated methylmethacrylate beads or collagen sponges should be considered. The client should be aware that removal of the surgical implants may be required when the fracture heals to definitively eradicate any biofilm-associated infecting bacteria.

ARTICULAR FRACTURES:

Articular fractures are considered to have specific requirements of fixation. By their nature they offer little bone stock for implant fixation on one side of the fracture. In addition, perfect reduction and rigid fixation to minimize callus formation are indicated to avoid disruption of the articular surface(s). Therefore, open reduction is required (to achieve proper alignment), and internal fixation is recommended (to optimize the mechanical environment). Malarticulation, osteoarthritis, joint pain and limited function may result if articular fractures heal without perfect restoration of the articular surface(s). Adequate surgical exposure is essential to visualize the joint and the fractures fully. Small cartilage fragments devoid of subchondral bone must be removed. Articular fragments with attached subchondral bone are manipulated into reduction, taking care not to damage the articular cartilage. Large gaps or steps in the articular surface are prevented by accurate reduction. Fragments are stabilized using one of several methods (Figure 6.8):

- Screws in lag (or positional fashion following compression by application of forceps) provide interfragmentary compression and counteract shearing forces at the fracture site. 1.5, 2.0, 2.4 or 2.7 mm screws are used, depending on fragment size, to avoid splitting the fragment. Screws are placed through subchondral bone where possible, avoiding damage to the articular surface. If necessary, screws can be inserted through the articular cartilage and the heads countersunk below the surface.
- Kirschner wires may be used to maintain reduction of very small fragments, but they do not provide compression. If Kirschner wires are inserted through the articular cartilage, they must be countersunk below the surface to avoid trauma to the opposing cartilage. Multiple Kirschner wires should be placed, preferably converging or diverging to maximize fixation.
- If necessary, a plate or external fixator may be used to support fragments which would undergo shear during joint loading (buttress fixation). Transarticular fixators may be applied if needed but are partially or fully disassembled once initial healing has occurred to allow joint motion, preferably prior to 4 weeks, but at least prior to 6 weeks.

Joint motion is encouraged soon after surgery to preserve the health of the cartilage and peri-articular soft tissues. Early joint function without disruption of the fracture site obviously requires adequate internal fixation of osteochondral fragments and restoration of normal ligamentous structures and joint capsule. Passive motion exercises should be performed frequently from the time of surgery. If fracture fixation is inadequate to permit early joint function, the joint is immobilized with external coaptation or an external skeletal fixator (perhaps applied transarticular) to protect the repair until partial healing has

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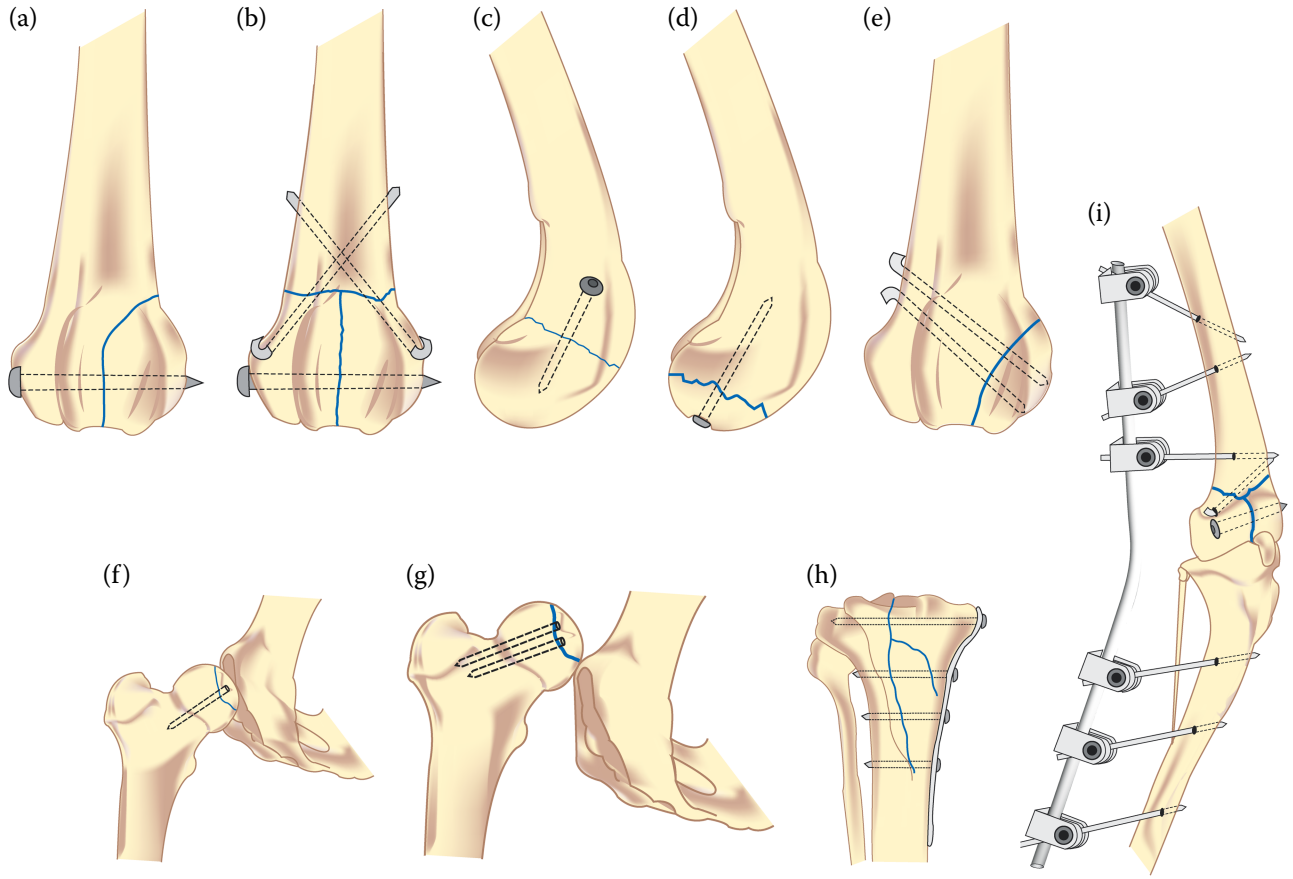


Figure 6.8 Repair of intraarticular fractures. **(a)** A transcondylar lag screw is inserted to compress the intraarticular fracture. Accurate anatomic reduction is required to prevent gaps or steps in articular surface. **(b)** A transcondylar lag screw is inserted to stabilize the intraarticular fracture. The supracondylar portion of the fracture is stabilized with cross pins. **(c)** A lag screw is inserted from the metaphysis into the articular fragment. The screw does not penetrate the articular surface. **(d)** A lag screw is inserted from the articular surface. The screw head must be countersunk below the cartilage surface. **(e)** Kirschner wires are inserted from the metaphysis into the articular fragment. Interfragmentary compression is not achieved with pin fixation. **(f)** A single Kirschner wire inserted through the femoral neck and into the osteochondral fragment to stabilize a fracture of the femoral head. **(g)** Two Kirschner wires inserted from the articular surface. The wires are countersunk below the cartilage surface to reduce trauma to the opposing cartilage. **(h)** A plate is applied to the metaphysis of the bone to buttress the articular surface. A plate screw may be placed in lag fashion through the plate to compress the fracture line. **(i)** A transarticular external fixator is applied to protect the internal joint fixation during healing.

occurred. Typically, the external support can be removed in 3–4 weeks.

Complications following articular fracture fixation include implant failure, since implants are necessarily small; infection, which is of increased risk where instability remains at any level of the fracture fixation; and the more chronic impact on joint health including reduced range of motion and cartilage loss (secondary osteoarthritis). Osteoarthritis is more severe in cases with malalignment of the cartilage surface but is also frequently a result of compressive trauma to the articular cartilage. The degree of trauma to the cartilage cannot be assessed from pre-operative imaging or the intra-operative appearance and this fact may account for a degree of unpredictable variability in post-operative function following articular fracture fixation. Fortunately, the progression of osteoarthritis is often slow and most cats function well, with limited range of motion.

Joint Arthrodesis

Joint arthrodesis (fusion) is rarely required in cats. It may be an alternative to limb amputation in some cases to preserve function in cats with complicated intraarticular fractures or joint luxations, painful joint instability, severe osteoarthritis unresponsive to medical therapy, or postural abnormalities caused by neurologic dysfunction. Various techniques used for arthrodesis of specific joints are discussed in the pertinent chapters. Arthrodesis takes place slowly, and implants are typically acting in bridging mode, necessitating robust and fatigue-resistant implants.

The steps to achieve arthrodesis of any joint include:

- **Removal of the articular cartilage** with curettes, rongeurs, power driven burrs, or a bone saw to expose the subchondral bone. Sclerotic subchondral bone is removed to expose bleeding cancellous bone.

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- **Placement of autogenous cancellous bone** graft at the arthrodesis site to promote bony union across the joint.
- **Application of rigid fixation**. Bone plates, lag screws, or Kirschner wires and external fixators may be used in various combinations as necessary. Compression must be applied between the exposed subchondral bone surfaces to enhance healing. The joint should be fused in a weight-bearing position. The appropriate angle is derived from published normal standing angles or by measuring the standing angle of the contralateral limb (provided it is not affected by orthopaedic disease).

POST-OPERATIVE CARE FOLLOWING FELINE ORTHOPAEDIC SURGERY:

The majority of cats undergoing orthopaedic surgery undergo a period of approximately 3 months of exercise restriction post-operatively. This typically involves the following:

1. Strict exercise restriction = confinement to a large (dog-size) crate for 2–4 weeks. The crate should be sufficiently large to accommodate a litter tray, bed area, food and water bowls without the latter being too close the litter tray (which may result in inappetence). The cat should be in the crate when not supervised but may come out of the crate under supervision to walk in a single room or enjoy time with the owner.
2. Moderate confinement = confinement to a single room in the house for 4 weeks. This room should not be too large and jumps up onto surfaces such as tables, window sills and so forth should be broken down into smaller steps (steps made of boxes are an example).
3. House confinement = the cat is permitted free access to the entire house, but should not go outdoors for 4 weeks. Following orthopaedic injury/disease, orthopaedic surgery and a period of exercise restriction, cats will need time for muscle strength to return. The concern with outdoor activity in this species, particularly with male cats, is that territorial behaviour will begin immediately upon being released. Therefore, cats must be 'fighting fit' before being allowed outdoors (to avoid re-injury soon afterwards). Confinement to the house allows cats to exercise and generate muscle mass prior to dodging cars on the roads.

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7 Arthrology

Feline arthrology has been an overlooked subject in the past with most reviews of joint disease in small animals focusing on the dog. However, cats are now known to suffer from many different types of joint disease and, although there are many similarities with the dog, there are also many features that are unique to the feline patient.

CLASSIFICATION OF JOINT DISEASE:

The terms arthritis and arthropathy literally mean joint inflammation and joint disease, respectively. Although these terms are used interchangeably in this chapter, to describe a number of well-defined joint diseases characterized by a combination of inflammatory and degenerative changes, the nomenclature is still confusing in the veterinary literature. The terms degenerative joint disease (DJD) and osteoarthritis (OA) are also often used synonymously. In this chapter, DJD is used as a general descriptive term to encompass degenerative changes in any joint in the axial or appendicular skeleton, including synovial, cartilaginous and fibrous types. When DJD affects the fibrocartilaginous intervertebral joints of the spine, it is known as spondylosis deformans. Therefore, the term OA is reserved for the specific type of DJD that affects diarthrodial synovial articulations. The terms arthrosis and OA are more commonly used in the human literature, to define non-inflammatory, degenerative joint conditions.

Diseases of synovial joints can conveniently be divided into degenerative arthritis and inflammatory arthritis on the basis of the predominant pathologic process (Table 7.1). Degenerative arthropathies are the most common types and include traumatic arthritis and OA. Inflammatory arthropathies are less common than degenerative arthropathies and have either an infective or immune-mediated aetiology. Infective arthritis caused by bacterial infection (septic arthritis) is the commonest type of inflammatory arthritis in the cat. Septic arthritis is classed as an erosive type of arthritis because there is destruction of articular cartilage in joints infected by bacteria. Immune-mediated arthropathies can be subdivided into both erosive and nonerosive forms. Differentiation between the infective and immune-mediated forms of inflammatory arthritis is essential since the therapeutic approaches to these conditions are diametrically opposed. Inappropriate treatment of an infective arthritis with immunosuppressive drugs, without appropriate antibiotic therapy, will have disastrous results.

INVESTIGATION OF JOINT DISEASE:

Diagnostic Imaging

(Keller et al. 1999).

Radiography is still the most frequently used ancillary diagnostic aid in the investigation of joint disease. Radiographs should be interpreted in the light of the clinical findings and not used as a substitute for a correctly performed clinical examination. Radiography is usually used to confirm a suspected clinical diagnosis or to differentiate between two or more joint diseases that have similar clinical characteristics. However, radiographic surveys of multiple joints might be indicated to evaluate the overall status of the patient in conditions that are frequently polyarticular, such as OA or immune-mediated arthritis. Radiographs or more advanced imaging of other body regions may be indicated; for example, in cats with joint trauma or neoplasia or some of the immune-mediated arthritides to check for evidence of lesions elsewhere. Feline joints are small so it is essential to obtain high-quality radiographs if they are to be of diagnostic value. Radiographs of joints should be correctly collimated to minimize scatter and obtained using fine detail screens in two orthogonal planes (i.e. at right angles to each other). When using traditional radiography, human mammography film/screen combinations are especially useful for radiographs of the distal extremities. Nowadays, with the widespread use of digital radiography, image resolution and the ability to manipulate the images are greatly enhanced so that appreciation of subtle changes in bone structure or soft tissue details is easier to achieve. Additional stress views may be performed in the evaluation of joint instability. Oblique projections and special views to outline certain structures are performed less frequently in the cat than in the dog. It is often useful to radiograph the contralateral joint to provide a normal radiograph for comparison or because some conditions occur bilaterally.

Table 7.1: Classification of Joint Disease

Degenerative arthritides		**Inflammatory arthritides**		**Miscellaneous joint disorders**
		Infective arthritides	*Immune-mediated arthritides*	Synovial sarcoma
	Osteoarthritis			Secondary neoplasia
Traumatic arthritis	Primary	Bacterial (septic)	Systemic lupus erythematosus	Hypervitmainosis A
	Secondary	Bacterial L-form	Polyarthritis/meningitis	Osteochondroma
		Mycoplasmal	Idiopathic	Synovial osteochondromatosis
		Calicivirus	Periosteal proliferative	Synovial cysts
		Lyme disease	Rheumatoid	Patellar malformation
		Tubercular		Meniscal calcification
		Fungal		Osteochondritis dissecans
				Osteochondrodysplasia in the Scottish Fold cat

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When bilateral involvement is suspected, consultation with an atlas of normal radiographic appearance of feline joints may be required to determine what is normal or abnormal for a specific patient.

General anaesthesia or heavy sedation is usually required for optimal positioning and may be a legal requirement in some countries. Radiographic interpretation must be thorough and complete so that each structure is assessed according to its Roentgen signs. Radiographic lesions in feline joint disease are often subtle and the use of magnification and brightness/contrast manipulation capabilities, etc, afforded by modern digital systems of radiography is extremely useful. Their interpretation should always be based on radiographs obtained in standard, consistent and reproducible projections and angles, to standardize the radiographic views. This is particularly important when subtle differences are diagnosed in serial radiographs of a specific joint or bone, which could potentially be an artefact of sub-optimal positioning.

The clinician should be familiar with normal feline osteology and with the radiographic appearance of normal feline joints. Accessory centres of ossification and sesamoid bones may be present in or around joints and these ossicles should not be confused with chip fractures. For example, an accessory centre of ossification may be present as a normal anatomic variant in the shoulder joint on the caudal or medial rim of the glenoid. Methodical examination is essential; the most common error is to find the expected abnormality and then to stop interpreting the radiograph before the whole film has been assessed.

All three components of the joint should be evaluated radiographically: the bones, the soft tissues and the joint space. The specific features that should be examined are the bony anatomic relationships, subchondral bone plates and subchondral bone of the epiphyses, the width and contents of the joint space, the articular margins and periarticular regions, and the periarticular soft tissues. The radiographic appearance of some disorders that are familiar to the canine clinician may differ in the cat. For example, the predominant radiographic signs of feline hip dysplasia are a shallow acetabulum with remodelling of the craniodorsal margin but with minimal remodelling of the femoral neck (Figures 7.1 and 7.2).

Computed tomography (CT) is a very useful modality to image the skeleton, although less indicated to evaluate soft tissue changes in great detail. Unlike radiographs, CT will provide a three-dimensional view of the skeleton, which is especially useful when evaluating the spine, skull and overlapping bones of the carpus and tarsus. Although more expensive than radiography, the slightly increased cost over several radiographic exposures needed for some complex cases can be justified by the increased resolution and quality of the study (Figure 7.3).

Points to Remember

(Holloway & McConnell 2014, Kirberger & McEvoy 2016, Mahoney & Lamb 1996, Thrall and Robertson 2015).

- The anatomic relationships of the bones making up the joint may be altered with joint luxation, subluxation and intraarticular fracture. Reference to a radiographic atlas, a file of normal cat radiographs, or a radiograph of the contralateral joint may be helpful, especially for the smaller more complex joints of the distal extremities. Of course, it is possible that the contralateral joint may also

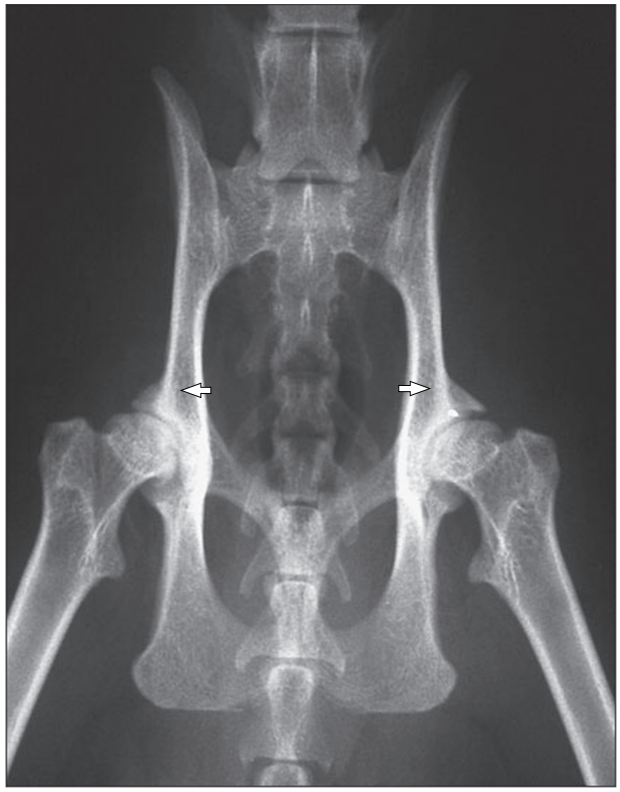


Figure 7.1 Ventrodorsal radiograph of the pelvis of a cat with coxofemoral osteoarthritis secondary to hip dysplasia. Note the remodelling of the cranial margins of the acetabula.



Figure 7.2 Degenerative joint disease is a very common, often underdiagnosed, cause of lameness and chronic pain in the cat. This radiograph shows a very advanced stage of hip osteoarthritis. (Courtesy of M. Ruiz)

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be abnormal. Knowledge of common variants of normal anatomy is also necessary.

- The subchondral bone plate should appear as a thin radiopaque line parallel to and adjacent to the joint space. Erosion of the subchondral plate occurs with erosive immune-mediated or infective arthritides, osteomyelitis and joint neoplasia. Localized areas of sclerosis and remodelling of the subchondral plate may be seen at the margins of destructive lesions, such as osteomyelitis. However, sclerosis is more commonly seen as a generalized change in chronic osteoarthritic joints where there is loss of articular cartilage and eburnation of the opposing joint surfaces, changing the loading pattern of the subchondral bone and inducing sclerosis.
- The subchondral bone of the epiphysis subadjacent to the subchondral plate should have a homogenous appearance. In comparison with the dog, the cancellous bone of cats has a coarse trabecular pattern. Osteomyelitis, neoplasia and articular fracture will cause an alteration in the internal architecture of the trabecular bone. Subchondral cysts are occasionally seen in osteoarthritic joints in cats as discrete radiolucent defects in the subchondral bone.
- Joint space width may be increased with joint subluxation or luxation. Narrowing of the joint space is indicative of loss of articular cartilage but cannot be appreciated on normal non-weight-bearing radiographs and, for this reason, it should be cautiously interpreted.
- Joint space structures are contained between the subchondral bone plates of the opposing bones that comprise the joint. This space, therefore, includes the articular cartilage, synovial fluid and intracapsular fat pads (and intraarticular ligaments, menisci and synovial effusion when present). Intraarticular structures are only visible when they become mineralized or when increased joint mass results in distension of periarticular soft tissues or alteration of other intraarticular structures. An example of the latter would be a reduction in size of the infrapatellar fat pad in the stifle, associated with a synovial effusion. Differentiation between increased joint mass associated with the presence of a soft tissue mass or fluid cannot readily be made radiographically and requires further imaging and synoviocentesis.
- The articular margins and the periarticular areas where ligaments and tendons attach should have a regular, smooth cortical outline. Osteophytes develop at the articular margins, along the insertion lines of the synovial membrane into the bone, where they appear as characteristic bony outgrowths. Osteophytes are most commonly associated with OA. Bony outgrowths that develop at the site of bony attachment of ligaments or tendon insertions are known as enthesophytes.
- Periarticular soft tissues are assessed for swelling, thickening and mineralization. Swelling most commonly occurs as a result of acute trauma, for example, joint sprain, whereas thickening is a chronic change most often associated with joint instability, such as chronic cranial cruciate ligament (CrCL) rupture. It is not possible to differentiate between thickening and swelling radiographically. Mineralization of periarticular tissue is a rare occurrence. It is occasionally seen as a sequela to chronic inflammation, for example, bacterial infective arthritis, and is also a feature of synovial osteochondromatosis.

Contrast Arthrography

(Seeburg et al. 1992).

Contrast arthrography is rarely performed in the cat due to the small size of joints, general lack of reproducible results, and reduced clinical applicability. Positive contrast arthrography involves the injection of a contrast agent into a joint space so that it mixes with the synovial fluid and delineates the internal margins of the joint. Therefore, proper knowledge of the synoviocentesis technique for every joint is necessary (for details of synoviocentesis and analysis of synovial fluid see Chapter 2)

The contrast agent of choice is iohexol (Omnipaque 300; Nycomed Imaging SA), which is diluted 50:50 with sterile water to give a concentration of 150 mg of iodine per ml. The technique is perhaps most useful for investigation of shoulder joint pathology. The volume of contrast agent required for suspected articular cartilage lesions (0.5–1.0 ml) is less than that required to detect capsular defects or to outline the biceps tendon of origin (2.0–3.0 ml). The technique for contrast arthrography has been described for the shoulder, elbow and carpus in the cat.

Sonography

(Mahoney 2012).

Ultrasonography has potential value for the investigation of joint disease, but there are few reports of its use in cats. The evaluation of bone is limited because of the inability of ultrasound to penetrate osseous tissues. However, sonography may be useful in the identification of joint effusion, joint thickening, articular or bone destruction, and joint instability using a dynamic examination. Periarticular soft tissue structures that may be examined with ultrasound include muscles, tendons, abscesses, cysts, foreign bodies and tumours. Use of the technique is likely to remain restricted to specialized centres because of the small size of the structures under investigation and the difficulty in the interpretation of the images obtained.

Arthroscopy

(Bardet 1998, Beale 2009, Bright 2010, Cusack & Johnson 2013, Chandler & Beale 2002, Ridge 2009, Staiger & Beale 2005).

Arthroscopy is less commonly used in the investigation and treatment of feline joint disease, as compared to the dog, likely due to the small size of these joints and the associated technical difficulty of the procedure. Also, the current price and technical limitations of small scopes may be a limiting factor for many clinicians (Figure 7.3).

Examination of the shoulder and stifle joints were described first, but the arthroscopic evaluation of hip and elbow joints has also been described. There is one report of the successful evaluation and debridement of the elbow joints of a cat with intraarticular osteochondral fragments. Application of arthroscopy to other feline joints, such as the carpus and tarsus, is likely to remain limited because of their small size, although continuing improvements in technology and reducing size of scopes will make these procedures more common in the future.

Technically, the diameter of the scopes used for feline arthroscopy range from 1.9 mm (for shoulder, elbow and hip joints) to 2.7 mm (for stifle joint in larger cats) and, other than differences in size, the technique is similar to that used in the dog. Specialized motorized shavers, electrocautery and radiofrequency can also be used to treat specific conditions.

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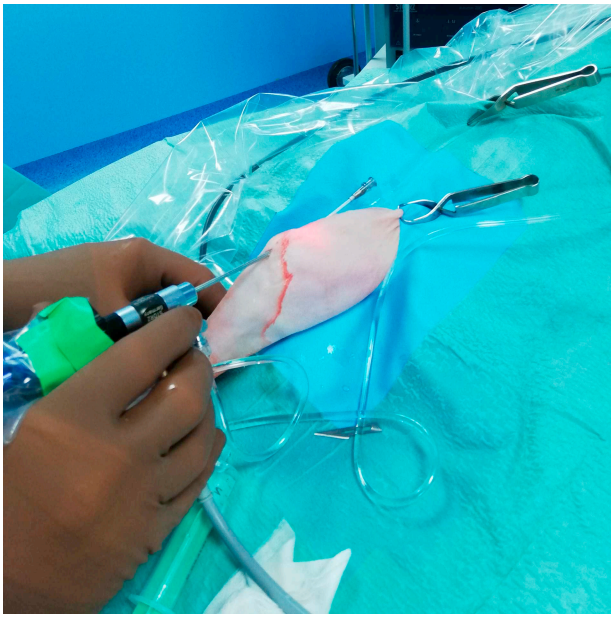


Figure 7.3 Arthroscopy of the stifle joint in a cat with a CCL rupture, using a craniolateral portal (Courtesy of J. Rial).

Shoulder Joint

Routine portal sites are very similar to the dog and the indications for shoulder arthroscopy in the cat include diagnosis and treatment of ligamentous injury, septic arthritis or characterization of inflammatory conditions with the help of synovial membrane biopsy.

Elbow Joint

The most common indication for arthroscopy in the feline elbow is diagnosis and management of elbow dysplasia, including fragmentation of the medial coronoid process, cartilage defects in the medial humeral condyle, removal of loose cartilage bodies and overall evaluation of the joint. Arthroscopic debridement of osteoarthritic elbow joints has also been described, through lavage, fragment removal, abrasion arthroplasty, or microfracture.

Hip Joint

Again, the portal sites are similar to the ones used for canine hip arthroscopy. The limited indications for a hip arthroscopy in the cat include evaluation of cartilage and ligamentous damage after a hip dislocation and assisting in the diagnosis and management of DJD and inflammatory joint diseases.

Stifle Joint

This is one of the most common joints evaluated arthroscopically in the cat and the indications for this exam include diagnosis and management of cruciate ligament disease, meniscal tears or mineralization, OCD, other ligamentous injuries and inflammatory arthropathies.

Laboratory Tests

A minimum database for investigation of a suspected inflammatory arthropathy would include a biochemistry and haematology panel and tests for feline leukaemia virus (FeLV) and feline immunodeficiency virus (FIV). This may help to identify cats that are immunosuppressed or those that have involvement of other body systems, such as may be seen with some immune-mediated arthritides (systemic lupus erythematosus and idiopathic polyarthritis types II, III and IV). Further investigation of joint disease requires synoviocentesis and synovial fluid analysis (see Chapter 2). Individual tests, such as measurement of titre for rheumatoid factor, antinuclear antibody, or antibodies to Borrelia burgdorferi, may be indicated in appropriate circumstances. Synovial fluid culture is necessary for the diagnosis of a septic arthritis. These tests are discussed more fully with the relevant joint disorders.

Synovial Membrane Biopsy

(Cazzini et al. 2015, Hardy & Wallace 1974, Pacchiana et al. 2003).

Biopsy of the synovial membrane or synovium is rarely performed in the cat, although the technique has been well described. The procedure can provide an invaluable insight into the pathology affecting a joint and is the only way of achieving a definitive diagnosis in some cases, mainly to distinguish neoplastic disease from degenerative or inflammatory conditions (relatively common canine conditions such as villonodular synovitis, leishmania-induced synovitis etc have not been described in the cat). Samples are usually collected by means of an arthrotomy or arthroscopy and should be obtained from several different sites to ensure that the changes seen are representative of the pathologic process in the entire joint. The histologic appearance of normal feline and canine synovial membrane is similar. Histopathologic examination can provide precise information about the extent and type of cellular infiltrate, allowing differentiation between degenerative and inflammatory joint disease. Although there is considerable overlap between diseases, the nature of an inflammatory cell infiltrate may help in determining the type of inflammatory arthritis.

Histopathology is usually the only way of achieving a definitive histological diagnosis of joint neoplasia, since joint neoplasms do not typically exfoliate and, therefore, identification of tumoral cells is rare during analysis of synovial fluid. Also, given the small size of feline joints, obtaining a good sample of joint fluid can be problematic. Samples of the synovium can also be submitted for bacterial culture and may be more likely to yield organisms than synovial fluid.

DEGENERATIVE ARTHRITIDES:

Traumatic Arthritis

Cause and Pathogenesis

Traumatic arthritis follows a single acute joint injury caused, for example, by vehicular trauma, an awkward fall, gunshot wounds, or fights with other animals. The least severe form of injury is a joint sprain, in which there is variable stretching or mild tearing of ligaments and joint capsule. Joint luxation involves a more severe disruption of peri- and intraarticular structures, with tearing of ligaments and joint capsule and disruption of articular cartilage. Intraarticular osseous and/or cartilage fractures are associated with the more severe traumatic injuries and may occur in isolation or combined with joint luxation or subluxation. Following a traumatic incident there will be haemorrhage within the joint (hemarthrosis) and synovitis, which may be localized to the site of injury.

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Clinical Signs

Clinical signs may include lameness, joint swelling, joint deformity and pain on manipulation. Crepitus will be present if there is fracture or luxation.

Diagnosis

If fracture or luxation is suspected, radiography should be performed. Joint instability and ligamentous disruption are evaluated by manipulation under general anaesthesia or deep sedation and stress radiography. Injury to individual ligaments is most commonly encountered in the stifle and tarsocrural joints. Joint sprains are classified according to the severity of the ligamentous injury as first, second and third degree.

Treatment and Prognosis

A more detailed discussion of the treatment of traumatic disorders relating to specific joints can be found in the relevant chapters. In general, treatment of joint trauma depends on the severity of the injury.

- First-degree sprains are mild and only require rest and/or anti-inflammatory medication.
- Second- and third-degree sprains and more severe types of joint injury such as intraarticular fracture usually require external coaptation or surgical repair. OA will occur as a sequela to traumatic arthritis if there are repeated mild to moderate episodes or if a single episode is severe enough to cause significant damage to the joint.

Osteoarthritis

(Clarke & Bennett 2006, Crawley et al. 2000, Freire et al. 2010, Freire et al. 2014, Hardie 1997, Hardie et al. 2002, Godfrey 2005, Lascelles 2010, Lascelles et al. 2010, Moskowitz & Goldberg 1987, Peterson et al. 1990).

OA is a type of DJD that is defined as a disorder of diarthrodial synovial articulations characterized by degeneration of articular cartilage, bone remodelling, pathologic changes in periarticular tissues, low-grade nonpurulent inflammation and the formation of new bone at the articular margins. OA may be classified into primary and secondary forms. Primary OA, most commonly accepted as part of normal aging, is thought to occur as a consequence of an inherent defect in the articular cartilage so that it is unable to cope with normal joint forces, whereas secondary OA is secondary to some other joint disorder, such as trauma, instability, previous fracture and so forth. This type of joint disorder was poorly studied until recently, but it has been recognized in the past few years as the most prevalent musculoskeletal disorder in the cat. The realization that OA is a common clinical problem in the aging cat is therefore a relatively recent phenomenon.

Most OA, however, is thought to be secondary in the cat. In a retrospective study of cats over 1 year of age radiographed for any reason, 22% showed evidence of OA of an appendicular synovial joint, although this was not clinically apparent in 67% of cases. In another study of 100 cats over 12 years of age, radiographic evidence of DJD was found in 90% of cats and severe OA was found in 17% of elbow joints.

Cause and Pathogenesis

OA as a cause of chronic pain is common in the geriatric cat, but it may be seen in any cat with joint abnormality or following injury. It is postulated that the inciting cause is repeated joint trauma associated with the cat's agile lifestyle and ability to jump. In accordance with this theory, some studies have found that the shoulder and elbow joints of geriatric cats are preferentially affected. OA may also be secondary to gross trauma, such as joint fractures, luxations and ligamentous disruption. Developmental diseases, such as hip dysplasia or patellar luxation, may lead to OA. OA has been reported in multiple joints as part of feline mucopolysaccharidosis VI and associated with acromegaly in middle-aged cats caused by a pituitary adenoma. The pathogenesis of acromegalic arthropathy is not fully understood, but it is thought that cartilage hypertrophy may interfere with cartilage metabolism, thereby causing cartilage degeneration.

Despite multiple aetiologies, the pathologic changes of OA share a final common pathway that serves to perpetuate the degenerative processes. Attention usually focuses on the articular cartilage but, typically, all of the joint structures are involved. Cartilage becomes fibrillated and ultimately may be completely lost, exposing the underlying subchondral bone, which responds by becoming thickened. The other main feature of OA is the production of osteophytes, which initially develop outside the epiphysis of the joint but eventually become incorporated into the joint, so that the joint assumes a different shape. This process is known as remodelling and is thought to be a mechanism that allows the joint to cope better with the altered stresses placed on it. Osteophytes are usually thought of as a chronic change in OA, but they have been shown to start to develop within 1 week of experimental induction of joint instability. Enthesophytes are soft tissue mineralizations that develop when there is pathology of the entheses, which are osseous insertion sites of ligaments and tendons. Additional soft tissue mineralization may occur as a result of capsular dystrophic calcification or synovial metaplasia. Degenerative enthesopathy and intraarticular or periarticular soft tissue mineralization often accompany OA but may also occur independently. Other pathologic changes include thickening of the joint capsule, an increase in vascularity with hypertrophy and hyperplasia of the lining layer of the synovial membrane, and degeneration of intraarticular menisci. The degeneration of articular cartilage that occurs in OA may be associated with synovial osteochondromatosis in which there is formation of osteocartilaginous bodies that remain free in the joint, disappear, or become embedded in the synovium.

Subchondral cysts are an occasional feature of osteoarthritic joints in cats. A cyst develops when there is replacement of subchondral bony trabeculae by mixed connective tissue. Mineralization of soft tissue structures within the stifle joint, such as meniscus, synovial membrane and fat, is also a common occurrence of osteoarthritic joints.

Clinical Signs and Scoring Systems

(Brondani et al. 2011, Brondani et al. 2013, Benito et al. 2012, Benito et al. 2012, Evangelista et al. 2019, Gruen et al. 2015, Gruen et al. 2017, Holden et al. 2014, Reid et al. 2017, Reid et al. 2017, Sano et al. 2012, Zamprogno et al. 2010).

The veterinary profession has been slow at recognizing the high prevalence of degenerative joint disease in older cats and its impact on the comfort and welfare of these patients. OA has long been recognized radiographically in cats, but, because the disease may be asymptomatic, the

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changes were often considered incidental. Typically, the clinical signs can go undiagnosed in cats, who are able to mask the symptoms and hide the physical manifestation of this disorder. The lack of attention to feline OA may be explained by a tendency to extrapolate information from the dog where the cardinal sign of OA is lameness. In one study of feline DJD, ability to jump and decrease of the height of the jump were lifestyle changes related to the musculoskeletal system that were more common clinical features than overt lameness. However, it is now believed that behavioural changes reflecting chronic pain are more relevant to the diagnosis of OA in the cat.

Different scoring systems for evaluating pain levels in cats have been proposed. Pain scoring systems for cats have been recently validated for both acute pain and chronic pain.

More recently, a 'grimace scale' has been developed for assessment of clinical acute pain in cats. A further refinement of this scoring system (CMPS-Feline, or Glasgow Feline Composite Measure Pain Scale), incorporating facial changes such as muzzle shape and ear position to a behavioural scale, increases its sensitivity and clinical usefulness. However, evaluating more chronic pain such as musculoskeletal pain due to OA is a much more complex situation for any species, perhaps even more challenging in cats than in others. Recent research aims to evaluate the chronic pain experience involve trying to measure not pain per se but rather its effect and impact on the patient's health-related quality of life (HRQL), scoring parameters such as vitality, comfort and emotional well-being. This is an attempt to include emotional and physical well-being issues and how the animal 'feels' about its circumstances (what is called 'the affective component of the pain experience'), instead of only focusing on physical and functional limitations of the patient. Clinical metrology instruments based only on functional limitations have been developed for cats, such as the Feline Musculoskeletal Pain Index. A significant caregiver placebo effect is possible when evaluating an analgesic effect, making demonstration of efficacy for an analgesic drug above a placebo difficult.

However, an instrument to measure feline HRQL using a scoring algorithm based on an owner's questionnaire would help owners and practitioners to assess the therapeutic benefits of any intervention, medication, or any other treatment modalities to treat chronic pain and its impact on overall quality of life. Currently, there is only one instrument validated for this approach, for use in dogs (VetMetrica HRQL for dogs) and its validation and possible use in cats are being investigated, as part of this emerging field.

Behavioural changes reported by owners of cats with OA are nonspecific and include those associated with attitude (hides away, resents handling, bad tempered, less playful, etc) and those associated with disability (reduced grooming, cannot jump, inactive, etc.). A behaviour-based pain assessment instrument has also been validated. Because cats are small, light and agile they compensate well for musculoskeletal disease and are able to adapt by redistributing weight-bearing forces to other limbs. Additionally, because the cat's lifestyle prevents critical analysis of the gait by the owner, mild lameness or joint stiffness usually passes unnoticed. Lameness and stiffness associated with OA typically have an insidious onset and are chronic and progressive in nature. As in other species, signs are worse on rising after a period of rest following exercise and initially wear off after the animal warms up. The signs may tend to wax and wane and are usually exacerbated by cold and damp weather. Acute flare-ups of the signs of established OA may occur as part of the natural history of the disease or be associated with joint sprain or sepsis. Osteoarthritic joints are susceptible to trauma and are also at increased risk of infection spread haematogenously from a septic focus elsewhere in the body.

Diagnosis

In advanced cases the diagnosis of OA may be obvious, especially if there is overt lameness. Affected joints will be thickened on palpation owing to bony remodelling, joint capsule fibrosis and synovial effusion. It is not usually possible to appreciate this in the shoulder and hip joints because of thickness of the surrounding soft tissues. Manipulation of a joint with OA will usually be resented and there may be crepitus and a reduced range of joint motion due to periarticular fibrosis. The subtler behavioural changes associated with OA may be caused by many other diseases, and careful assessment of the geriatric cat is required to establish their significance. This may include radiographic and laboratory evaluation to confirm the presence of OA and rule out intercurrent disease. The radiographic changes of OA are similar to those of the dog (Figure 7.4). The most striking feature is the presence of osteophytes. These are visualized as roughening of the joint margins, as obvious bony masses (spurs or exostoses) projecting beyond the normal bony outline, or as irregular bony densities when superimposed on the normal osseous architecture. There will be secondary soft tissue swelling with thickening of the joint capsule and there may be an increase in joint mass owing to the presence of excessive joint fluid (synovial effusion), more readily recognized in the stifle joint. Although enthesophytes and/or soft tissue mineralizations are commonly seen in osteoarthritic joints, these features alone are not indicative of OA. With advanced OA there will be changes in bone shape due to remodelling, accompanied by sclerosis of subchondral bone and, occasionally, subchondral bone cysts visualized as discrete radiolucent defects. CT images of the joints provide a more comprehensive, three-dimensional view of the osseous structures of the joint, and features such as osteophytosis/enthesophytosis, bone cysts etc are much more easily recognizable, when compared to radiography. Also, more subjective and difficult to interpret features such as degree of subchondral sclerosis and soft tissue swelling become easier to appreciate. Analysis of the synovial fluid from affected joints can be performed to rule out inflammatory joint disease. The fluid from joints with OA is normal or has only a slight reduction in viscosity and a minimal increase in cell numbers, with macrophages and lymphocytes predominating.

Treatment and Prognosis

(Addison & Clements 2017, Boothe 1990, Gunew et al. 2008, Giraudel et al. 2009, Gowan et al. 2012, Jones et al. 1992, King et al. 2016, Knazovicky et al. 2016, Lascelles & Sheilah 2010, Mathews 2000, Runk et al. 1999, Schmid et al. 2010, Speranza et al. 2015).

The approach to the management of OA in the cat is similar to that for dogs. Options include exercise modification, weight control, the use of medication and surgical

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Figure 7.4 Osteoarthritis. **(a)** Plantarodorsal radiograph of a cat with osteoarthritis of the hock joint secondary to previous trauma. **(b)** Plantarodorsal view of the contralateral normal hock joint. **(c)** Mediolateral radiograph of a cat with osteoarthritis of the elbow. **(d)** Craniocaudal view of the joint in **(c)**.

intervention. Potential problems with the management of OA include lack of familiarity with the disease in cats, difficulties with lifestyle modification, and the toxicity of available drug therapies, particularly long term. Despite this, the prognosis for OA is generally better than that for the dog because cats are smaller and more athletic. Modification of a cat's exercise regimen may be difficult but some control is possible; for example, by altering the environment or by only allowing the cat out at set times each day or restricting jumping. Strategies used in the dog, such as restricted leash exercise and aquatic therapy, are sometimes appropriate for the cat. Lameness associated with flare-ups of OA should be managed by enforcing strict confinement for a few days. Weight loss is essential if the cat is obese and use of a commercial low-calorie diet is recommended. Conversely, geriatric cats that are underweight should be fed a calorie-dense diet, since OA will worsen if they stop exercising as energy levels decline.

Pain control in cats with OA has been a problem in the past because of a lack of drugs specifically licensed for long-term use in the cat (Table 7.2). Other factors, such as the central sensitization of chronic painful states such as OA, are due to the sustained activity of nociceptors, increasing the excitability of neurons within the central nervous system. This activity-dependent synaptic plasticity leads to increases in synapse efficacy and reductions in inhibition, causing somatosensory abnormalities such as allodynia, hyperalgesia, or thermal hypersensitivity at local and remote sites from the affected joint. Central sensitization can have important implications for the diagnosis and effective treatment of pain. Its associated somatosensory abnormalities have been studied in dogs with OA in which an increased sensory sensitivity has been demonstrated when compared to normal dogs This increased thermal sensitivity in limbs with OA was also demonstrated in a study involving force plate analysis in cats.

Cats are more susceptible than other species to the adverse effects of nonsteroidal anti-inflammatory drugs (NSAIDs) because of a deficiency of glucuronide conjugation. Slower drug metabolism and a longer half-life lead to accumulation of toxic amounts much faster in cats than other species.

Development of newer NSAIDS has recently expanded the choices for treatment of DJD in the cat and, mainly due to this reason, opioids are not really a good option for long-term management of this condition. Currently, several NSAIDS have been extensively studied and adapted for use in the feline patient and, although they may be commercially available, they may or may not be licensed for use in the cat, depending on the country. With the availability of these more modern drugs, the use of older, unlicensed anti-inflammatories, such as aspirin, phenylbutazone, ibuprofen,

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Table 7.2: Orally Administered Drugs and Nutraceuticals in the Management of Osteoarthritis (Licensing, Product Name and Availability May Vary in Different Countries. List is not comprehensive)

Drug	Dose	Comments
Meloxicam (Metacam, Loxicom etc)	INJECTABLE: 0.3 mg/kg SC, single injection, OR, 0.2 mg/kg SC, if followed by oral suspensionORAL: 0.1 mg/kg, q 24 h followed by 0.05 mg/kg q 24 h	A popular COX-2 selective inhibitor, available in palatable, liquid form for oral administration
Robenacoxib (Onsior)	INJECTABLE: 2 mg/kg, SC, Q 24 h, up to three injectionsORAL: 1–2 mg/kg, q 24 h, up to three doses	Newer, COX-2 selective Nsaid.
Carprofen (Rimadyl, etc.)	INJECTABLE: 4 mg/kg SC/IV, single dose	Oral administration is not an option for cats
Ketoprofen (Ketofen, etc.)	Injectable: 2 mg/kg, SC, q 24 h, up to three dosesORAL: 1 mg/kg, q 24 h, up to 5 days	This COX-1 inhibitor is much less used nowadays
Tramadol	3 mg/kg, po, q 12 h	Not very well studied
Gabapentin	5–10 mg/kg, po, q 12 h	May produce ataxia and sedation
Glucosamine/Chondroitin Sulphate (Cosequin)	1 capsule a day	Also contains manganese
Glucosamine/Chondroitin sulphate/ Avocado-soybean unsaponifiables (ASU) (Dasuquin)	1 sprinkle capsule a day for 4–6 weeks and EOD for maintenance	
Green lipped mussel, Omega 3 fatty acids, etc (YUMOVE)	1 capsule a day	

piroxicam, naproxen, flunixin meglumine and so forth, is not justified. Other NSAIDS, such as ketoprofen or tolfenamic acid are COX-1 inhibitors or have uncertain COX inhibition patterns and, therefore, are more likely to produce side effects in the cat and should be used with caution only when safer options are not available. Even with these drugs it is essential that dosing is performed accurately if toxic effects are to be avoided.

Carprofen, a COX-2 selective compound, is available for use in cats, but only licensed as a single injection of 4 mg/kg (0.24 ml/3 kg) bodyweight by subcutaneous or intravenous injection, given pre-operatively at the time of induction of anaesthesia, for surgical analgesia. Oral administration is not an option for cats and makes this drug unsuitable for long-term treatment of DJD.

Meloxicam, a COX-2 selective inhibitor, is currently licensed for reduction of post-operative pain and inflammation, at a dose of 0.3 mg/kg SC if a single injection is given or at a dose of 0.2 mg/kg SC if then followed by the use of the oral suspension. Also, for the treatment of more chronic pain, the initial oral dose of 0.1 mg/kg po q 24 h is then followed by a maintenance dose of 0.05 mg/kg po q 24 h., discontinuing after 14 days if no clinical improvement is seen. The clinical efficacy of the syrup has been demonstrated for cats with acute and chronic painful locomotor disorders. The authors have found the oral form of the drug to be both safe and effective when administered at a loading dose of 0.1 mg/kg (1drop/kg) once daily for 5 days, followed by a maintenance dose of 0.02–0.04 mg/kg (1–2 drops/cat). This dosage can be used safely long term, but, like all NSAIDs, meloxicam should preferably be used strategically for a few weeks at a time. In cases where medication is required on a permanent basis, the lowest effective dose is titrated against the clinical signs, if necessary, by reducing the frequency of administration. Long-term administration has been shown to not affect longevity in the cat.

Robenacoxib is a newer, COX-2 selective NSAID with a short half-life in plasma (approximately 2 hours) and licensed for the alleviation of pain and inflammation in both acute and chronic musculoskeletal disorders, both marketed in injection form and palatable tablets. The dose for injection is 2 mg/kg SC q 24 h, for a maximum of three doses and 1–2 mg/kg po q 24 h for the oral form, for up to 3 days of treatment. Its use for the treatment of feline DJD has been reported.

Treatment for up for 9 days was tolerated and produced no side effects and another study showed no side effects after 30 days of treatment, even in patients with chronic kidney disease.

Ketoprofen, a COX-1 inhibitor, is indicated for use in cats for the treatment, as an antipyretic, analgesic, and anti-inflammatory, for acute pain from musculoskeletal disorders. As an injection, it can be given at a 2 mg/kg dose, sc q24 h, repeated up to 3 days. In tablet form, the dose of 1 mg/kg po q24h can be given up to 5 days. Its clinical efficacy and safety compare well with other, more COX-selective NSAIDS.

Tolfenamic acid is a COX-1 selective NSAID, rarely used nowadays, as a single injection or for 3 days of oral use (4 mg/kg dose).

The use of glucocorticoids is controversial in the treatment of OA and there is evidence that high doses cause deterioration by promoting cartilage degeneration. However, glucocorticoids are well tolerated by the cat and obvious clinical improvement can often be produced by the use of relatively low doses for short periods of time. Prednisolone or prednisone at a dose rate of 0.1–0.5 mg/kg

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once or twice daily is the drug of choice for severe OA unresponsive to NSAIDs or for acute flare-ups when infection has been ruled out. Considering the safer, more selective and available drug choices for long-term administration in cats, the chronic use of steroid for control of musculoskeletal disease rarely becomes necessary. The combined administration of glucocorticoids and NSAIDs confers no therapeutic advantage and is contraindicated because of the greatly increased risk of side-effects. When NSAIDs are used after a course of steroids, or conversely, a flushing period of 2–3 days between the end of one course and the beginning of the other is probably the safest course of action.

Other Analgesics

(Adrian et al. 2019, KuKanich 2013, Lorenz et al. 2012, Monteiro et al. 2016, Monteiro et al. 2017, Robertson & Lascelles 2010, Siao et al. 2010).

Gabapentin, an antiepileptic medication, has been used extensively in the treatment of persistent, chronic neuropathic pain in humans. It works by binding to the N-type voltage calcium channels in the dorsal root ganglion neurons, reducing the release of excitatory neurotransmitters, such as glutamate and substance P. The use of gabapentin for treatment of feline OA has been recommended, and, in a recent survey, it was the most commonly prescribed medication for treatment of chronic musculoskeletal pain in cats. Its use in cats (at a dosage of 6–7 mg/kg BID) as part of a long-term pain management strategy has also been described. Gabapentin shows good bioavailability in cats when given orally, with a reported 88.7% with a half-life of 2.8 h., although food was likely to change its absorption. Most commonly observed adverse effects in cats include ataxia and sedation. At this point, its use is not licensed for cats in the UK or the US.

Tramadol has also been used to treat OA in cats. This drug is, also, not currently licensed for use in cats in the UK.

Intraarticular Injections

(Bogers 2018).

The use of intraarticular medication for treatment of feline DJD has not been reported. Anti-inflammatory steroids in various forms, lubricants (mainly hyaluronic acid) and other more biological compounds with a more disease-modifying approach, such as platelet rich plasma (PRP) and stem cell therapy have all been used to treat osteoarthritic joints in dogs, both in experimental and clinical situations. These treatment modalities may become more widespread in the future, especially the use of blood-derived biological therapy, as we continue deepening our understanding of their mechanisms of action.

Others

(Bennet & May 1995, Boothe 1997, Corbee et al. 2013, De Haan et al. 1994, Gück 2009, Lascelles et al. 2010, Vandeweerd et al. 2012, Vandeweerd et al. 2012).

The use of the so-called matrix supplements or visco-supplements is still very poorly documented in the cat. The injectable drugs, pentosan polysulphate and polysulphated glycosaminoglycans, are unlicensed for use in the cat. Pentosan polysulphate is administered at a dose rate of 3 mg/kg subcutaneously (SC) on four occasions 5–7 days apart. These drugs should not be combined with NSAIDs since they have anticoagulant properties that are potentiated by NSAIDs. In one trial no short-term problems were identified when polysulphated glycosaminoglycan was administered to healthy cats.

Their mode of action is not fully understood, but products such as Cosequin or Dasuquin (Nutramax Laboratories Inc.) may have some benefits in the treatment of cats with chronic pain due to OA. Although their efficacy is not proven, they appear to be safe. They can be used in combination with NSAIDs when they may have the effect of reducing the minimum effective dose of the NSAID.

There is also a number of nutritional supplements available for oral administration that are recommended for use in the cat for the treatment of OA. These preparations are classed as nutraceuticals and are not subject to the same rigorous licensing procedures applied to pharmaceutical drugs. Most nutraceutical products marketed for OA contain glucosamine and chondroitin sulphate as the main ingredients, but they can also contain many other related compounds. The term 'nutraceutical' was coined from 'nutrition' and 'pharmaceutical' in 1989 and was defined as 'a food or part of a food that provides medical or health benefits, including the prevention and/or treatment of a disease.' Other available products, classified as 'dietary supplement', are defined as products taken by mouth that contains a dietary ingredient intended to supplement the diet. These typically include vitamins, herbs, minerals, amino acids, enzymes and so forth and can be marketed in several presentations. The definition of a nutraceutical, according to the North American Veterinary Nutraceutical Council (NAVNC), is "a substance produced in purified or extracted form which, when administered orally to patients, aims to provide them the necessary elements for their structure and normal function to better their health and well-being", but this definition has no regulatory meaning. In Europe, no specific indications can be legally recognized for nutraceuticals as no official evidence of efficacy is required to market them, and manufacturers do not have to conduct the extensive studies that are necessary to obtain official approval for marketing medicinal products such as antibiotics or anti-inflammatories.

Unfortunately, studies typically found in the veterinary literature have the usual limitations, such as the lack of rigorous randomized controlled trials and the limited numbers of patients, the lack of objective outcome measures, the risk of conflict of interest, the lack of standardization of dosages and duration of treatments and so forth. A relatively recent systematic literature review reveals that the only dietary supplement that has been studied with enough rigour in veterinary medicine is omega-3 fatty acids in dogs.

The effects of omega-3 fatty acid supplements on OA have been studied in different species and omega-3 fatty acids have been proposed to have a beneficial effect on OA in cats.

A study of the effect of a diet supplemented with eicosapentaenoic acid (EPA), docosahexaenoic acid (DHA), green-lipped mussel extract and glucosamine/chondroitin sulphate in cats with painful, mobility-impairing DJD showed an increase in objectives measures of mobility.

Adjunctive Therapy

(Benjamin et al. 2005, Fu et al. 2009).

Joints that are poorly responsive to medical therapy may benefit from irrigation with sterile normal saline or lactated Ringer's solution. The procedure can be combined with synoviocentesis and is performed by insertion of an intravenous cannula into the joint under general anaesthesia

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followed by lavage with up to 200 ml of fluid. This may be beneficial because of the temporary removal of joint debris and inflammatory mediators. Although this approach to osteoarthritic joint treatment has been studied in humans, with conflicting results, it has not been evaluated in small animal veterinary trials. So, although the theoretical basis of large-volume joint lavage as a method for removal of degradative products, inflammatory cytokines and matrix-degrading enzymes is understood, there is still no clear evidence it provides long-term relief over a placebo, sham treatment in humans. In an experimental meniscectomy model rabbit study, joint lavage produced a reduction in joint degeneration and expression of pro-inflammatory cytokines in synovial fluid.

Theoretically, joint lavage can also be done in combination with an arthroscopic inspection and debridement of osteoarthritic joints.

The goal of surgical intervention should be the correction of the underlying causes of OA, such as cruciate rupture or intraarticular fracture, in order to prevent or slow the progression of the disease. Joints that are chronically painful and nonfunctional may be salvaged by arthrodesis, excision arthroplasty in the case of the hip and mandibular condyle, or joint replacement for the hip joint. These procedures are discussed in detail in the relevant chapters.

INFECTIVE INFLAMMATORY ARTHRITIDES:

(Lemetayer and Taylor, 2014).

An infective arthritis is a type of inflammatory arthropathy caused by a living microbial agent, which can usually be isolated from the joint. The microbial agent in cats may be a bacterium, mycoplasma, virus, fungus, or spirochaete. These conditions have been recently reviewed

Bacterial (Septic) Arthritis

(Bennett 2010, Lascelles et al. 2010, Lund et al. 1999, May 2005, Mielke et al. 2018, Palerme et al. 2016, Taylor & Scott-Moncrieff 2013).

Bacterial or septic arthritis is the most common type of infective arthritis. This condition is typically monoarticular and the bacteria involved are those commonly found in the oral cavity, skin and so forth (*Pasteurella multocida*, *Streptococcus* spp., *Staphylococcus* spp., *coliforms*, *anaerobes*).

Cause and Pathogenesis

Septic arthritis is usually the result of a bite wound sustained in a fight with another cat. Cat bites are a common occurrence contributing to 4.7% of all feline consultations in one North American study. Other causes include traumatic injuries, such as vehicular trauma, extension of infection from adjacent soft tissues, hematogenous spread, or iatrogenic following joint surgery. Intervertebral disc joints are rarely affected causing discospondylitis. In line with the aetiology the joints of the distal extremities, particularly the carpus and the hock, are most commonly affected. Hematogenous spread from an infected focus elsewhere in the body is uncommon.

This type of septic arthritis is usually mono-articular and infection is more likely to localize to a joint that is already damaged. Bacterial arthritis may thus be a complication of traumatic joint injury, such as a sprain, and osteoarthritic joints are predisposed to infection, as it occurs commonly in dogs, perhaps due to a persistent, chronic synovitis.

Multiple joint involvement may occur secondary to bacterial endocarditis, although this is very rare in the cat and the definitive confirmation of septic arthritis versus reactive joint inflammation has not been established. Young kittens occasionally suffer from polyarticular septic arthritis as a result of hematogenous spread from an infected umbilicus (omphalophlebitis) or associated with postparturient metritis or mastitis

Clinical Signs

The main presenting sign is lameness of a single limb, which is usually acute and severe. If septic arthritis follows a cat bite, initial lameness may fail to resolve or there may be relapse after initial improvement. In some cases the primary injury may have passed unnoticed by the owner and evidence of a bite wound may only be discovered on closer inspection. The affected joint will be hot, swollen and painful, with a reduced range of motion. In advanced cases there may be crepitus indicating extensive damage to articular cartilage and bone. The cat is often, but not invariably, systemically ill and pyrexic. In chronic cases there may be secondary ligament rupture and disuse atrophy of the muscles of the limb, and joint ankylosis may eventually develop.

Diagnosis

Diagnosis is based on the history and clinical findings, radiography and synovial fluid examination. Routine haematology can be done if a septic process is suspected, but this is not a reliable indicator of septic arthritis, since leucocytosis is an inconsistent finding. Radiography in the early stages shows nonspecific changes associated with the accumulation of exudate, soft tissue swelling and joint distension. Osseous changes become evident within a few weeks. Loss of articular cartilage may be evident as a loss of joint space, although changes in joint space are difficult to assess in non-weight-bearing radiographs. Periarticular osteophytes develop and these may be extensive. In more chronic cases there is destruction of bone, with erosion of the subchondral plate and, eventually, complete loss of bone with an irregular widening of the joint space. Osteomyelitis may extend into the metaphyses of the adjacent bones, with a mixed pattern of lysis and sclerosis as attempts are made to wall off the infection (Figures 7.5–7.8). Bony ankylosis of the joint may eventually develop due to extensive periarticular fibrosis and lack of use of the affected limb.

Synovial fluid should be obtained from any joint when infection is suspected. Suppurative fluid will be increased in quantity, turbid and watery, and may be haemorrhagic. White blood cell numbers will be moderately to greatly increased (sometimes as high or higher than 75,000 cells/mm^3), and these will predominantly degenerate neutrophils (as high as 90%–98%). Culture of synovial fluid may reveal the organism, although organisms cannot be grown in some cases. The likelihood of a positive culture is much greater if the sample is inoculated into blood culture medium and if prior antibiotic therapy has not been given. The culture should always include both aerobic, anaerobic and Mycoplasma species.

Culture of the synovial membrane may be necessary if the synovial fluid culture is negative in the face of clinical indicators of infection. It is more indicated in cases in which the septic arthritis is managed via an open approach (for debridement, implant removal, drainage, etc.) and, in these cases, a sample of synovial membrane should always be obtained for histopathology.

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Figure 7.5 Lateral projection of an elbow suffering from a septic arthritis. Observe the marked soft tissue swelling and the early periosteal reaction around the distal humerus and olecranon. (Courtesy of M. Ruiz)



Figure 7.7 Lateral view of the same joint, left untreated for 2 months. The advanced degree of joint destruction is apparent. (Courtesy of M. Ruiz)



Figure 7.6 Craneocaudal radiograph of the same joint. (Courtesy of M. Ruiz)



Figure 7.8 Craniocaudal view of the same joint. Observe the extensive bone destruction and periosteal reaction. (Courtesy of M. Ruiz)

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Treatment and Prognosis

Cat bites involving a joint or adjacent to a joint should be treated aggressively, using lavage and drainage, if necessary, to prevent infection becoming established in the joint and to flush fibrin and inflammatory enzymatic debris from the joint. Bacterial cultures of deep tissues should be performed to determine appropriate antibiotic therapy. Joints that are already septic should be lavaged copiously under anaesthesia with normal saline or Ringer's solution, either percutaneously, arthroscopically, or via a mini arthrotomy. If infection is more chronic or in case of postoperative infections involving implants that may need removal, an arthrotomy may be necessary. However, the additional tissue trauma associated with this approach must be weighed up against achieving adequate treatment without surgery.

Antibiotic therapy, preferably guided by culture and sensitivity, is continued for a minimum of 4 weeks or for at least 2 weeks after resolution of clinical signs. Treatment should be initiated as soon as samples have been obtained for culture. To avoid clinical deterioration, antibiotic therapy should be started before culture results are available, and inevitably, the initial choice of antibiotics is based on the likelihood of the presence of common staphylococcal pathogens. A good initial choice is a bactericidal β-lactamase-resistant antibiotic with activity against both Gram-positive and Gram-negative organisms and, due to the possible presence of anaerobes, inclusion of an appropriate second antibiotic is indicated. The authors' initial choice may typically include clavulanate-potentiated amoxicillin and metronidazole or clindamycin, and then changing to a more long-term antibiotic therapy choice when the culture and sensitivity results become available.

Other commonly used antibiotics for definitive therapy include cephalosporins, clindamycin, fluoroquinolones, metronidazole, sulphonamides and so forth. Aminoglycosides (amikacin, gentamicin, etc.) and other, more toxic antibiotics, should be reserved for treating difficult infections caused by bacteria resistant to other antibiotics.

Special care needs to be exercised when using fluoroquinolones in cats due to concern with retinal blindness when high doses are given but both marbofloxacin and pradofloxacin can be safely used in medium dosages. Also, these drugs have a potential to produce cartilage damage in younger cats so their use is not recommended in cats less than 6–16 weeks of age. Analgesia and anti-inflammatory medication may also be advisable to treat the clinical signs of joint pain and stiffness. Rest and appropriate physiotherapy are recommended to facilitate full recovery.

Initially, resting affected joints will be important and bandaging to immobilize them should be considered. After the acute phase, gradually increasing activity will be important to re-establish mobility and adequate muscle mass.

The prognosis is dependent on the degree of permanent damage to the joint. If there has been considerable destruction of articular cartilage, joint function will be poor and excision arthroplasty, arthrodesis, or even amputation may be necessary in extreme cases. If a joint replacement is a possibility (in a coxofemoral joint infection, e.g.), repeated negative cultures of synovial fluid after long-term antibiotics would be indicated before surgery is considered.

Persistent low-grade lameness may be a sequela because of OA or a likely immune- mediated synovitis caused by the presence of nonviable bacterial antigens. In the latter case, glucocorticoids (oral, low-dose prednisolone or similar) may be beneficial provided bacterial infection has been completely eliminated. These drugs also help to relieve joint stiffness associated with soft tissue swelling and fibrosis. There may also be a possible role of steroids, in combination with antibiotic therapy in the acute management of joint infections, to reduce the negative effects of joint inflammation. More studies are necessary before this approach is clinically warranted.

Bacterial L-Form Arthritis

(Greene & Chalker 2012, Lemetayer & Taylor 2014).

Bacterial L-forms are cell-wall deficient, similar to Mycoplasma. They are a rare cause of inflammatory polyarthritis in cats. A number of reasons for the loss of the cell wall have been proposed, including therapy with certain antibiotics and interaction between the host's immune system and the bacterium. Subcutaneous abscesses and arthritis associated with a probable bacterial L-form have been described. Clinical signs were seen in three cats from one household and included pyogenic subcutaneous abscesses, inflammatory joint disease, pyrexia and systemic illness. The infection starts from direct inoculation (typically from a bite wound), spread locally and hematogenously to involve other joints and subcutaneous sites. The exudate is characterized by pyogranulomatous inflammation.

Radiography of affected joints revealed destructive changes, including periarticular soft tissue swelling and periosteal proliferation progressing to damaged articular cartilage and subchondral bone, consistent with erosive joint disease. Bacterial L-forms are bacteria that lack a cell wall and are resistant to many broad-spectrum antibiotics, although they are susceptible to doxycycline or quinolones. Because of the lack of cell wall the bacterial L-forms cannot be identified using routine light microscopy. The organisms are very difficult to grow in the laboratory and routine cultures of joints and abscesses will be negative. Treatment is recommended to continue for 4–6 weeks or for at least 1 week beyond the healing of abscesses.

Mycoplasmal Arthritis

(Liehmann et al. 2006, Moise et al. 1983, Zeugswetter 2007).

Mycoplasmas are found naturally in the upper respiratory and urogenital tracts of healthy cats. There are a number of reports of the organisms causing polyarthritis and tenosynovitis, usually in debilitated or immunosuppressed individuals. Mycoplasmal arthritis has also been reported in immunocompetent cats. In a case report, it has been associated with an erosive polyarthritis Affected cats are presented with lameness, swollen limbs, painful joints and pyrexia. Mycoplasma polyarthritis is primarily non-erosive based on radiographs, but erosions can occur, perhaps more readily visible when using computed tomography. Subchondral bone and cartilage erosions have been described at necropsy, in cases with no radiographic changes. The synovial fluid is watery and turbid, and cytology reveals large numbers of white blood cells with nondegenerate neutrophils predominating. Direct visualization of the organism is rare. Culture of the organism from the synovium or the synovial fluid may be possible on special media or strictly anaerobic conditions. A tentative

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diagnosis is possible if the organism can be visualized by light microscopy after staining with Wright–Leishmann or Giemsa. Treatment with doxycycline, enrofloxacin, or pradofloxacin should result in rapid clinical improvement. Long-term treatment (6–8 weeks) may be required to avoid relapse.

Calicivirus arthritis

(Bennett et al. 1989, Dawson et al. 1993, Dawson et al. 1994, Lascelles & Sheilah 2010, Levy & Marsh 1992, Pedersen et al. 1983).

Cause and Pathogenesis

Calicivirus is a common pathogen of cats, which produces fever, conjunctivitis, sneezing, oronasal ulceration and nasal discharge. It is also a cause of acute arthritis in cats, either following natural infection or live virus vaccination, producing lameness alone or in combination with upper respiratory signs. The role of calicivirus in chronic arthritis is unknown. The arthritis may be infective, associated with intraarticular live virus, or immune-mediated, associated with intraarticular nonviable virus antigens. Disease is most commonly seen in cattery kittens infected with field strains of calicivirus. It can occur in spite of vaccination since immunity to at least one strain of calicivirus is not conferred by the more commonly used vaccine strains. Lameness has been reproduced experimentally using two separate strains of calicivirus in young kittens. Viraemia was demonstrated but no lesions were found in muscles, joints, or the central nervous system; increased numbers of macrophages were found in the synovial fluid. In the past, vaccine-associated calicivirus arthritis was commonly seen associated with certain strains of calicivirus included in live vaccines. This is now a rare cause of disease since manufacturers have removed these strains from their vaccines.

Clinical Signs

Infective arthritis associated with certain field strains of calicivirus is seen mainly as a transient lameness and pyrexia in kittens between the ages of 6 and 12 weeks. Affected kittens are usually ill for 1–2 days and demonstrate a stiff gait with pain on manipulation of affected muscles and joints. Less than half of the kittens have, or subsequently develop, ulceration of the tongue or hard palate. Kittens experimentally infected displayed generalized hyperaesthesia with evidence of joint pain on manipulation and, in some cases, cutaneous erythema over the tarsal joints. Immune-mediated arthritis is seen mainly as a polyarthropathy 5–7 days after vaccination with live vaccines containing feline calicivirus. Affected cats have a shifting lameness, a stiff gait and pyrexia. Signs generally occur after the first vaccination and most affected cats are less than 6 months of age.

Diagnosis is generally attempted on the basis of clinical signs and, in the case of the vaccine-associated form of arthritis, the history of recent inoculation. Serology is generally unhelpful because of the widespread presence of antibody as a result of vaccination. Cytology of synovial fluid from affected joints may reveal increased white cell counts (neutrophils and small mononuclear cells), with a greatly increased number of macrophages, many of which contain phagocytosed neutrophils. If possible, synovial fluid culture to rule out septic and Mycoplasma arthritis should also be done.

Treatment and Prognosis

The prognosis for infective calicivirus arthritis is good since the disease is self-limiting and only symptomatic therapy is indicated. The usual clinical signs of fever and lameness only last for 3–5 days and resolve by themselves, so in cases with a typical history or already improving, an aggressive diagnostic approach may not be warranted. Immune-mediated calicivirus arthritis is also usually transient, although some cats develop a more protracted inflammatory polyarthropathy. Disease is usually self-limiting, but a short course of analgesics may be indicated and administration of glucocorticoids may be indicated for persistent cases. This form of polyarthritis is similar to type II idiopathic immune-mediated polyarthritis.

Rickettsial Polyarthritis

(Breitschwerdt et al. 2002, Lappin & Breitschwerdt 2012).

Non-erosive polyarthritis secondary to many tick-borne rickettsial infections have been reported in the dog but have been only rarely described in cats. These feline pathogens include some Ehrlichia and some Anaplasma species.

Ehrlichia species

Ehrlichia infection in cats have not been extensively studied and which species can produce natural infection is not clear.

Hyperaesthesia, joint pain, fever, inappetence and weight loss are typical clinical signs, suggesting a polyarthritic condition. There are several reported cases in which feline monotropic ehrlichiosis was suspected based on finding morulae in mononuclear cells and positive Ehrlichia canis serology or a positive response to anti-ehrlichial drugs, but isolation of E canis-like DNA is rare. Infected cats may show a neutrophilic, culture-negative inflammatory picture in their synovial fluid examination. Serology is not a reliable test for diagnosis so confirmation of Ehrlichia species infection using PCR is recommended but only possible through a research laboratory at this time. The current recommendations are that cats with a suspected infection could be treated with doxycycline for a minimum of 28 days.

Anaplasma phagocytophilum

(Lappin & Breitschwerdt 2012, Lappin et al. 2004, Little 2010).

This organism (previously known as Ehrlichia equi) has been reported to affect horses, dog, cats, ruminants and people. After transmission, the organism infects blood neutrophils forming intracellular inclusions known as morulae (Latin translation: mulberry, which describes their appearance). Naturally occurring feline granulocytotropic anaplasmosis has been reported in several countries, including the United States, Poland, Finland, Sweden, Denmark, Ireland, Italy and the UK.

In experimentally produced infections with Anaplasma phagocytophilum, affected cats develop mild pyrexia and a neutrophilia and although the most common clinical signs described are fever, lethargy, anorexia, lymphadenomegaly, epistaxis, weight loss, conjunctivitis and so forth, some infected cats have exhibited fever, swollen painful joints and lameness. Microscopic detection of morulae within

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neutrophils in a clinically ill cat is highly suggestive of the diagnosis, but they can be difficult to find. Serology may be necessary to confirm the diagnosis. Antibodies detected by indirect immunofluorescence assay can be measured, but results are often negative during acute infection, cross-reactivity with Ehrlichia species occasionally occurs, and a single positive titre cannot differentiate active disease from previous infection. Recommended treatment of cats with suspected anaplasmosis includes supportive care, analgesia and administration of doxycycline for 3–4 weeks.

Lyme disease

(Burgess 1992, Gibson et al. 1993, Gibson et al. 1995, Greene et al. 2012, Jacobson & Saidla 1990, Krupka & Straubinger 2010, Lemetayer & Taylor 2014, Levy et al. 2003, Littman et al. 2018, Magnarelli et al. 1990, May et al. 1994. Shaw et al. 2005).

Cause and Pathogenesis

Lyme disease is a multisystemic inflammatory disorder of humans and domestic animals caused by infection with the tick-borne spirochaete Borrelia burgdorferi, transmitted by Ixodes ticks. Lyme disease was first recognized in the US, where it is endemic in certain areas but has since been described worldwide. Cats appear to be resistant to the development of clinical signs of Lyme borreliosis. Although they can become infected and develop high levels of serum anti-borrelia antibodies, the natural disease has not been described as a distinct clinical entity in the cat.

Polyarticular lameness, synovitis, meningitis and evidence of systemic inflammation have been described in cats inoculated with organisms harvested directly from infected ticks.

Seroconversion is a recognized feature of this disease, both in naturally exposed and experimentally infested cats suggesting that infection occurs, although it is possible that cats fail to develop clinical borreliosis because they remove infected ticks faster than dogs. At this time, there is no convincing evidence indicating direct causation between borreliosis and clinical illness in cats.

Clinical Signs

Clinical signs were not observed in experimental cats inoculated by various routes with laboratory-derived strains of Borrelia. In contrast, when cats were inoculated with the organism directly from ticks they developed polyarthropathy and had joint, lymphoid, pulmonary and central nervous system inflammation on postmortem examination. In acute infections in dogs, clinical lameness is frequently localized to one or more joints, although chronic nonerosive polyarthritis is the main finding in more prolonged infections. The course of disease may be intermittent and spontaneous resolution may occur. There is no information on the clinical manifestations of natural infection in cats, but clinical signs are likely to be similar to those described in the dog.

Diagnosis

The clinical signs are often vague and infection can be asymptomatic, so accurate diagnosis of Lyme disease is difficult. Serology can be done to detect antibodies against the C6 peptide of B. burgdorferi (4DX plus SNAP IDDEX)

A positive serologic test merely signifies exposure to the organism and is not proof that the clinical signs are caused by the spirochaete. In two North American studies serum samples taken from cats that had been infected with ticks were positive for antibodies to the organism in 14% and 15% of cases. The proportion of positive results was the same irrespective of lameness. In the UK 4.8% of cats, screened as clinical patients, were seropositive. None of the seropositive cats in the latter study had clinical signs of lameness. PCR can be used for definitive detection of these organisms from several sources such as synovial membrane, joint fluid, CSF fluid or blood

Treatment and Prognosis

Therapy for Lyme disease is given empirically because of the difficulty in making a definitive diagnosis. The organism is susceptible to a number of antibiotics but doxycycline (10 mg/kg twice daily for 4 weeks) is still currently the drug of first choice for adult cats. Clinical improvement should always be viewed with suspicion because the clinical signs may be intermittent or may resolve spontaneously. Based on the information available from other species, prompt improvement in clinical signs is to be expected with appropriate antibiotic therapy. Treatment is more likely to be successful if antibiotics are given in the initial acute phase of the infection.

Tubercular Arthritis

(Bennett et al. 2011, Gunn-Moore 2010, Gunn-Moore et al. 2011, Lalor et al. 2017, Lo et al. 2012, Major et al. 2015).

Tubercular arthritis is an occasional manifestation of tuberculosis, an uncommon disease in cats. In the UK, 34% of their mycobacterial infections are caused by members of the *Mycobacterium tuberculosis* complex, 19% of cases are caused by *Mycobacterium microti* and 15% by *Mycobacterium bovis*. Another, non-tuberculous mycobacterium infection has been reported to produce a septic arthritis in a hip joint in an immunocompromised patient, as an opportunistic infection in a renal transplant patient.

Primary pulmonary tuberculosis is very rare in cats, much more typically having cutaneous disease, in the form of single or multiple cutaneous lesions affecting the 'fight and bite sites', such as the head and legs, and are believed to result most commonly from being bitten when playing with infected prey, such as infected small rodents. Local lymph node involvement, typically affecting the mandibular nodes, is very common in affected patients. Pulmonary or systemic signs are seen less frequently in the cat, but are usually seen in chronic infections, following haematogenous dissemination.

Joint involvement in feline mycobacterial infections is uncommon, as it is in humans. In a retrospective study describing radiographic findings in cats with mycobacterial infections, patients showed skeletal abnormalities in the distal antebrachium, pes, maxilla, scapula, ventral spine, manus, femur and tarsus. The most common changes were osteolytic lesions, although osteoproliferative changes have also been described.

These osteolytic lesions have also been seen in CT examinations of affected bones, involving the nasal bridge

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and the limbs, with cortical lysis or erosive lesions at joint surfaces. Again, a mixture of osteolytic and osteoproliferative patterns were found.

True articular involvement has been described in a recent series of four cats diagnosed with tuberculosis, patients were 2–6 years old and immune competent, with both monoarticular and polyarticular presentations. *Mycobacterium bovis* was isolated in two cases and *Mycobacterium microti* in the other two.

In a previous case series of feline tuberculosis, one cat presented with involvement of an elbow joint. Radiological findings included osteolysis, periosteal reaction and associated soft tissue swelling.

Diagnosis can be obtained with joint biopsies since analysis of joint fluid appears to be of little use. Histopathology will typically show pyogranulomatous inflammation and acid-fast stains can be applied to cytology and histopathology samples to screen for acid-fast bacilli. A special mycobacterial culture, mycobacterial PCR tests on fresh or fixed tissue, or a blood test for interferon gamma release assay (IGRA) could all be helpful in the diagnosis.

Prognosis is uncertain due to the small number of reported cases but a treatment regimen based on rifampicin (10–15 mg/kg PO q24h), azithromycin (5–15 mg/kg PO q24h) and pradofloxacin (3.5–5 mg/kg PO q24h) for 3–6 months has been recommended, depending on the extent of the disease and always for at least 2 months following complete resolution of the lesions.

Amputation of the affected limb has also been recommended in severe cases or if the medical therapy fails to improve clinical and radiological signs.

Fungal arthritis

(Lioret et al. 2013, Tisdall et al. 2007).

Histoplasma capsulatum, Crytococcus neoformans, Coccidioides and Blastomyces are rare causes of hematogenously disseminated infective arthritis. There is usually multisystemic involvement and multiple joints may be affected. As a result of the fungal infection, feline patients can develop an immune mediated polyarthritis, due to deposition of complexes in the joints. Since fungal arthritis is likely to be another manifestation of a systemic fungal infection, investigation of other organ systems is indicated (thoracic radiographs, nasal, ocular and sinus investigation, abdominal ultrasound). Fungal organisms can be identified cytologically in synovial fluid, but a low number of organisms may be a limitation. Serological diagnosis is possible for coccidiodomycosis and cryptococcus. The diagnosis of blastomycosis can be achieved in a urine antigen test.

Systemic fungal disease and fungal arthritis are treated with systemic antifungal drugs, such as fluconazole, itraconazole or clotrimazole. As can be expected, the extent of the systemic involvement in various organ systems will determine the prognosis for affected cats.

IMMUNE-MEDIATED INFLAMMATORY ARTHRITIDES:

(Carro 1994, Letemayer 2014, Perry 2015).

Immune-based arthritides are uncommon causes of lameness in the cat and their diagnosis and management have been recently reviewed. There are a number of different types, but they are all characterized by chronic synovitis affecting multiple joints in a bilaterally symmetrical fashion. Although the underlying cause of these disorders is unknown and they are classed as noninfective, persistent intraarticular microbial antigens may play a role in their etiopathogenesis. It is useful to subdivide them into erosive and nonerosive types based on the degree of damage to bone and articular cartilage. Joint inflammation in nonerosive immune-mediated polyarthritis is thought to be due to the deposition of immune complexes of antibody, antigen and complement in the synovial membrane. Treatment of the various types of immune-mediated polyarthritis is similar and is discussed at the end of this section.

Nonerosive types

(Abrams-Ogg et al. 2018, Bennett & Nash 1988, Lemetayer & Taylor 2014, Taylor & Scott-Moncrieff 2013).

Systemic lupus erythematosus (SLE) and idiopathic polyarthritis are both more common in young adult cats. The clinical signs of cats with nonerosive immune-based polyarthritis are very similar irrespective of type and generally have a rapid onset. Affected cats typically appear very stiff and uncomfortable and may have a history of shifting lameness. They are usually reluctant to exercise, resent handling and may be uncooperative and bad tempered. There is usually inappetence, malaise and pyrexia unresponsive to antimicrobial therapy. Affected joints are painful on manipulation and distal limb joints may be obviously thickened and swollen. In some cases it may be more difficult to appreciate synovial effusion or periarticular thickening. Cats with SLE and idiopathic polyarthritis may show other signs of immune complex disease reflecting involvement of other body systems. Idiopathic type II cats may show signs of infection elsewhere in the body, type III cats will have gastrointestinal signs, and type IV cats may have signs relating to an underlying neoplasm. Cats with type IV idiopathic polyarthritis usually have myeloproliferative disease, which can be diagnosed by bone marrow biopsy. A proportion of these cases will be positive for FeLV.

Systemic Lupus Erythematosus (SLE)

Systemic lupus erythematosus is a multisystemic disease in which nonerosive polyarthritis may be a feature. The antigen involved in the pathogenesis of the polyarthritis, dermatitis and glomerulonephritis that may be seen with SLE is believed to be nucleic acid. In one study, definite SLE was diagnosed in only 2 of 31 cats with polyarthritis, and it is a rare cause of joint disease.

Three criteria must be satisfied to diagnose definite SLE. Criteria 1 and 2 must always be satisfied; if criterion 3 is not fulfilled a diagnosis of probable SLE is made.

1. There should be multisystemic involvement, although it is rare to have more than two body systems affected at any one time. Common manifestations include polyarthritis, thrombocytopenia, haemolytic anaemia, leucopaenia, pyrexia, dermatitis, glomerulonephritis and meningitis.
2. There should be a positive antinuclear antibody (ANA) titre. A 1:40 titre is considered positive in cats. The ANA

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test is very sensitive but has poor specificity and it can be positive in many inflammatory disorders other than SLE such as ehrlichiosis, FeLV and FIV infections, as well as drug-induced in cats being treated with propylthiouracil and methimazole.

The background level of ANA is relatively high in the cat and false positive results may occur during the acute phase of infectious diseases such as FeLV, FIV and feline infectious peritonitis (FIP). On the other hand, in a recent study of the prevalence of antinuclear antibodies in cats, it was noted that a strong false positive is unlikely in healthy cats.

It should be noted that a diagnosis of SLE cannot be made by positive ANA alone, regardless of titre.

3. The immunopathologic features should be consistent with the clinical involvement. For example, antibodies against red blood cells or platelets should be demonstrable in cats with anaemia or thrombocytopenia; immune complex deposition should be shown in tissue biopsies of the synovial membrane or kidney in cats with polyarthritis or glomerulonephritis respectively.

Because of the overlap in clinical presentation and lack of serological specificity, any cat with a non-erosive polyarthritis should have testing performed to eliminate an infectious or secondary (reactive polyarthritis) cause. A 2-week course of empirical doxycycline may also be considered and the response to this medication assessed. After ruling out these initial diagnoses, the treatment for SLE is identical to other immune-mediated joint condition protocols, involving anti-inflammatory steroids (prednisone/prednisolone), alone or in combination with immune-suppresors (leflunomide, chlorambucil, methotrexate, etc). Decision-making about ongoing medication is based on clinical progression and possibly, on serial synovial fluid analysis. The prognosis for resolution of SLE polyarthritis should be good, but other systemic manifestations may be more problematic.

Polyarthritis/meningitis syndrome (PMS) is a rare condition with clinical manifestations similar to SLE. However, there is joint and central nervous system involvement only and affected cats test negative for serum ANA. The typical presentation is an immature or young adult cat with neck pain, depression, pyrexia and stiffness in addition to polyarthritis. Cerebrospinal fluid (CSF) analysis reveals increased protein and pleocytosis.

Idiopathic Polyarthritis

(Bennett & Nash 1988, Feldman 1994, Taylor & Scott-Moncrieff 2013, Wilkinson & Robins 1979).

Cats with polyarticular, noninfective inflammatory joint disease that does not satisfy the diagnostic criteria for other types of polyarthritis are included in this broad classification. Idiopathic polyarthritis is the commonest of the immune-mediated arthritides in dogs but is rarely reported in the cat. Idiopathic polyarthritis can be subdivided into four types based on the presence of concurrent disease:

- Type I: Uncomplicated idiopathic polyarthritis.
- Type II: Reactive idiopathic polyarthritis associated with infection elsewhere in the body; for example, urinary or respiratory tract infection (chlamydial conjunctivitis, coronavirus infection associated polyarthritis, etc), drug-induced or vaccination-induced.
- Type III: Enteropathic idiopathic polyarthritis associated with gastrointestinal disease.
- Type IV: Neoplasia-related idiopathic polyarthritis associated with neoplastic disease.

All types are believed to be the result of immune complex hypersensitivity reactions. Therefore, the term 'reactive arthritis' has been used in the literature to describe a non-erosive polyarthritis which occurs secondarily to antigenic stimulation from chronic infection, neoplasia, or drug administration, therefore encompassing several types of the traditional classification of idiopathic polyarthritis.

The different associations in types II through IV may represent the antigenic source for the immune complexes or may be coincidental. Multisystemic involvement occurs in some cats; these cases may resemble SLE, but they are consistently negative for ANA. Some cases of type I, and occasionally type II, may eventually progress to an erosive rheumatoid or periosteal proliferative polyarthritis.

Erosive Types

(Bennett 1994, Moise & Crissman 1982, Pedersen et al. 1980).

Erosive immune-mediated arthritis in the cat include chronic progressive polyarthritis and rheumatoid-like arthritis.

These are two types of disease: a more common periosteal proliferative type and an uncommon deforming type with many of the features of human and canine rheumatoid arthritis. With both types of diseases the clinical signs have a tendency to wax and wane, but their clinical course is generally progressive.

Periosteal Proliferative Polyarthritis

(Becker et al. 1994. Magne 2000, Oohashi et al. 2010, Pedersen et al. 1980, Voss & Langely-Hobbs 2009).

Periosteal proliferative polyarthritis (PPP) is seen in young entire and neutered male cats less than 5 years of age, but female cats can also be affected

Cause and Pathogenesis

The aetiology is uncertain, but there may be links with FeLV, FIV and feline syncytia forming virus (FeSFV) infections. It has been suggested that FeLV- (or FIV-) induced immunosuppression allows multiplication of FeSFV in the joints of predisposed individuals, which causes the disease. However, an increased incidence of FeLV or FIV virus in infected cats has not been demonstrated. It has also been reported that all cats with PPP are infected with FeSFV. It has not, however, been possible to reproduce the disease experimentally and the virus can be cultured from the joints of many normal cats. It is possible that the presence of FeSFV is incidental, since an already inflamed joint with many actively dividing cells is an ideal environment for the virus to multiply.

Clinical Signs

There is typically an acute onset of pyrexia and malaise with a stiff gait, joint effusion and joint pain, particularly in the carpi and hocks. There may also be generalized muscle atrophy and generalized or localized lymphadenopathy. Initially, the radiographic signs may only include soft tissue

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Figure 7.9 **(a)** Mediolateral radiograph of the hock joint of a cat with early changes of PPP. Note the erosive changes on the tuber calcis and the periarticular periosteal new bone formation on the plantar aspect. (Radiograph courtesy of Davies Veterinary Specialists.). **(b)** Mediolateral radiograph of the elbow joint of the cat in **(a)** showing erosive changes in the olecranon. (Radiograph courtesy of Davies Veterinary Specialists.) (**c** and **d**) Dorsopalmar view of carpal joint and a lateral view of a stifle joint of a different patient, showing extensive periosteal reactions around the joints (Courtesy of N. Calvo)

swelling and mild periosteal bony reaction. Within several weeks of this early acute phase the disease enters a more chronic phase in which there is extensive periosteal new bone formation, especially around the hocks and carpi (Figure 7.9). If these bony changes become severe, it can lead to joint ankylosis. Osteophytosis, enthesophytes, subchondral cysts and periarticular erosions are also late radiographic features of this condition.

Rheumatoid Arthritis

(Bennett & Nash 1988, Hanna 2005).

Rheumatoid-like arthritis is a rare chronic, progressive, irreversibly destructive polyarthritis occurring in middle-aged and older cats, with a possible predisposition in Siamese cats. This is likely the same disease previously described as the destructive form of feline chronic progressive polyarthritis.

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Cause and Pathogenesis

The pathogenesis of the synovitis involves the production of autoantibodies against immunoglobulin G (rheumatoid factor). The synovitis is caused by the formation of immune complexes, including rheumatoid factor and immunoglobulin, within the joint. However, there is not always serologic evidence of rheumatoid factor and its presence is not specific for rheumatoid arthritis.

Clinical Signs

Cats with rheumatoid arthritis have generalized stiffness, progressive lameness and debility of increasing severity over a period of many months. The onset is generally gradual, and, unlike PPP, there is no malaise and no significant synovial effusion. Palpation reveals joints that are thickened and painful, with crepitus and reduced range of motion on manipulation. Typically, multiple distal joints are affected; however, it is not uncommon to diagnose a lameness in these patients if one limb is more severely affected than the rest.

Diagnosis

The criteria used to diagnose rheumatoid arthritis in cats are based on those used in human rheumatology: 1. Stiffness after rest. 2. Pain or tenderness on motion of at least one joint. 3. Swelling of at least one joint. 4. Swelling of one other joint within a period of 2 months. 5. Symmetrical joint swelling. 6. Radiographic changes suggestive of rheumatoid arthritis. 7. Serologic evidence of rheumatoid factor. 8. Abnormal synovial fluid. 9. Characteristic histologic changes in the synovial membrane.

Characteristic radiographic changes include severe, progressive subchondral bone lysis, joint deformity, soft tissue swelling, and joint subluxation or luxation, with no significant periosteal reaction. Periarticular soft tissue calcification is also possible. Subcutaneous nodules seen in humans with rheumatoid arthritis have not yet been reported in the cat. Seven criteria must be satisfied to make a diagnosis of classical rheumatoid arthritis and five to make a diagnosis of definite rheumatoid arthritis. In addition, two of criteria 6, 7 and 9 should be fulfilled and criteria 1–5 should have been present for at least 6 weeks.

Diagnosis of Immune-Mediated Arthritides

Radiography

All clinically affected joints should be radiographed. In cats with suspected multisystemic involvement, radiography of other body regions may be indicated; for example, the thorax for evidence of respiratory infection in idiopathic type II polyarthritis. The radiographic changes seen in the joints of cats with the different types of immune-mediated arthritis are very similar, especially in the early stages of disease. Common features of both erosive and nonerosive arthritis are periarticular soft tissue swelling, distension of the joint capsule and loss of intraarticular fat shadow in the stifle joint. With chronicity, the erosive forms show a more characteristic combination of destructive and proliferative changes. In rheumatoid arthritis the destructive changes predominate. There is erosion of the joint margin visualized as an increased irregular joint space or an overall loss of mineralization of the bones forming the joint. In comparison with the dog, the coarse trabecular pattern of feline bones may make it more difficult to appreciate the destructive changes in the early stages.

In PPP there are also destructive changes, but the proliferative changes predominate. PPP is characterized by periarticular periosteal new bone formation, which is so extensive in some cats that affected joints become partially or sometimes completely ankylosed. There are also erosive changes in the joints and bony deposits and erosions at the point of attachment of ligaments or tendons (enthesopathies). Serial radiography is useful to monitor the progression of the arthritis. For example, some nonerosive forms may progress to erosive disease and secondary OA may become apparent as joints become more damaged and less stable.

Laboratory Features

In cats with multisystem involvement the laboratory features will be consistent with the underlying nature of the immune complex disease, such as anaemia and thrombocytopenia. There is generally a nonspecific absolute or relative increase in serum globulin levels. Synovial fluid should be obtained from multiple joints to confirm that there is an inflammatory polyarthropathy. There is typically an increased volume of turbid synovial fluid, with reduced viscosity and a positive fibrinogen clot test. The fluid has an increased white cell count, the majority of which will be nondegenerate polymorphonuclear cells and is negative on culture (see Chapter 2). If there is still doubt about the diagnosis, multiple synovial membrane biopsies from different parts of one or more joints should be submitted for histopathologic examination. However, the histologic changes can be variable and are not specific for one particular type of inflammatory arthropathy.

Serologic tests are performed for suspected cases of rheumatoid arthritis and SLE. Rheumatoid factor may be present in the blood of cats with rheumatoid arthritis (although this is not in itself sufficient or essential for diagnosis), but specific tests for measuring RF in cats are not commercially available. Therefore, modifications of the canine Rose-Waaler or ELISA tests have been used to measure RF in cats.

ANA must be present in the blood of cats before SLE can be diagnosed. Cats with SLE may also show red cell autoantibodies or autoantibodies against leucocytes or platelets. It is important to check with the diagnostic laboratory that species-specific reagents are available before submitting samples for immunologic testing.

Treatment and Prognosis for Immune-Mediated Arthritides

(Hanna 2005).

Nonspecific treatment of cats with immune-mediated arthritis is the same as that for cats with OA (exercise restriction or moderation, weight control, analgesia, etc.). Specific treatment is directed towards the underlying cause in cats with idiopathic polyarthritis types II, III and IV. Successful treatment of the infective, gastrointestinal, or neoplastic process may resolve the arthritis without the need for additional immunosuppressive therapy.

Cats with chronic PPP or rheumatoid-like arthritis are usually treated with anti-inflammatory doses of prednisolone for short periods when there is a flare-up of the clinical signs. Strong anti-inflammatory/immunosuppressive therapy with glucocorticoids is the mainstay of treatment for all other types of immune-mediated arthritis. The drug of choice is prednisolone

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administered at a starting dose of 4 mg/kg divided twice daily for acute cases. This dose is continued for 2 weeks and then gradually reduced over a period of 8 weeks, provided the disease stays in clinical remission. In some cats the drug can be discontinued but in others a continuous low dose is required to maintain remission. The maintenance dose should preferably be given on an alternate day basis to minimize side-effects. If remission cannot be achieved or can only be maintained with excessively high doses, a combination of prednisolone and a cytotoxic/immunosuppressive drug should be used.

Most cats have only a minimal response to treatment with non-steroidal anti-inflammatory drugs or even low dose glucocorticoids by themselves. In addition to steroids, other drugs, such as cyclosporine, chlorambucil, leflunomide, or methotrexate, should be considered, either in combination with low-dose steroids, as a single agent or in a dual-agent therapy, provided the steroids can be discontinued without exacerbation of the clinical signs. The safe use of these drugs has been reported in other feline conditions, and it is accepted they could also be used for treatment of these arthropathies. Azathioprine is not recommended in cats and cyclophosphamide is no longer recommended by many clinicians. Methotrexate in combination with leflunomide produced clinical improvement in 58% of cats, with no serious toxicity in one report. Analgesics, such as tramadol, gabapentin and so forth, should also be prescribed to help alleviate the clinical signs and reduce the dosage of anti-inflammatories and other drugs used.

Patients that are immunosuppressed should be regularly checked for and, as far as possible, protected from secondary infections, especially of the urinary and respiratory tract. Vaccination is advisable but should not be given until the immunosuppressive drugs have been stopped or suspended. The prognosis is variable and differentiation between types of immune-mediated arthritis is essential when attempting to predict the likely outcome of treatment. The idiopathic polyarthritides carry the best prognosis, with the exception of type IV, which is secondary to a neoplastic process. The prognosis tends to be worse for erosive than for nonerosive forms, especially rheumatoid arthritis. However, SLE often carries a poor prognosis because of the involvement of other body systems.

In addition, the destruction of the cartilage, and so forth in joints affected with erosive forms may produce mechanical collapse and necessitate arthrodesis, typically of the carpus or tarsus.

MISCELLANEOUS DISORDERS OF JOINTS:

Osteochondroma (Also see Chapter 19)

(Perry 2015, Rosa & Kirberger 2012, Schmidt & Langham 1967, Turrel & Pool 1982, Voss & Langely-Hobbs 2009).

Solitary osteochondromas (also known as osteocartilaginous exostosis) are ectopic mineralization disorders that have been reported as a rare cause of lameness in domestic cats, and especially in the Burmese breed. They are slow-growing tumours with a cartilaginous cover, typically seen as a cause of lameness in older cats. Treatment involves local excision, but local regrowth and malignant transformation have both been reported. In the multiple form (feline osteochondromatosis or multiple osteocartilagenous exostosis) these bony growths arise in ribs, scapula, skull, vertebrae and pelvis of young mature cats, possibly with a Burmese breed over-representation. They are more aggressive than osteochondromas and have been compared to parosteal sarcomas. There is no effective treatment although radiation therapy and surgery have been attempted, in a small number of cases and with limited success. A link to FeLV infections has been proposed.

Synovial Osteochondromatosis (Also see Chapter 19)

(Cotter 1998, Langley-Hobbs 2001, Levitin et al. 2003, Perazzi et al. 2013, Perry 2014, Poole et al. 1972, Perazzi 2013, Straw 2000, Tan et al. 2010, Tas et al. 2013, Villacin et al. 1979).

Synovial osteochondromatosis (also known as synovial chondrometaplasia) is a rare condition characterized by the formation of chondral or osteochondral nodules within the synovial tissue of a joint, tendon sheath, or bursa. Lesions resemble synovial osteochondromatosis but the calcifications do not seem to be attached to the bone. It has been reported in the stifle, shoulder and elbow joints of cats (Figure 7.10). Also, synovial osteochondromatosis has been reported affecting the elbow joint of a Bengal tiger. The condition is typically monoarticular, but it can occur in several joints concurrently and is thought to occur as a primary idiopathic abnormality but its relationship to the ongoing pathological changes in degenerative joint disease is unclear. There is chronic progressive lameness and joint stiffness. The radiographic appearance varies according to the stage of the condition. Initially there may be only soft tissue swelling as a result of the cartilaginous nodules and joint effusion. As the condition progresses, calcified deposits become visible in and around the joint and there will be evidence of OA. Treatment may be palliative using NSAIDs or surgical treatment may be attempted by total synovectomy with removal of all loose bodies. In advanced cases excision arthroplasty, arthrodesis, or in selected joints such as the hip, total joint replacement may be performed.

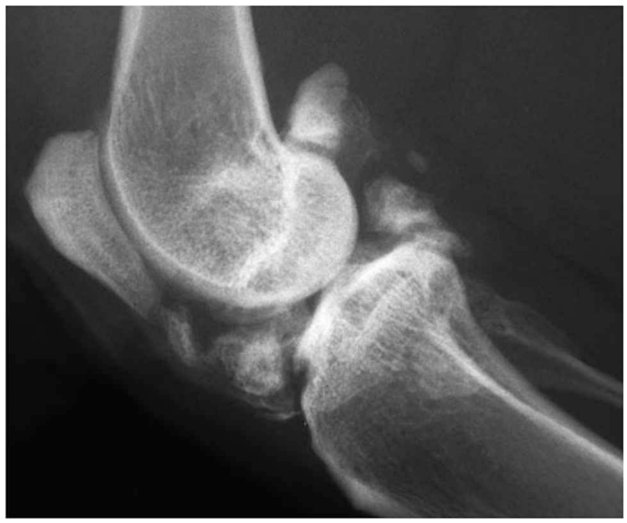


Figure 7.10 Mediolateral radiograph of the stifle joint of a cat with synovial osteochondromatosis. Note the extensive intraarticular and periarticular mineralized deposits. (Radiograph courtesy of Davies Veterinary Specialists.)

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Synovial Cysts

(Hittmair et al. 2010, Kligman et al. 2009, Prymak & Goldschmidt 1991, Stead et al. 1995, White et al. 2004).

Synovial cysts have been rarely reported in the cat, commonly affecting the elbow joint, although not exclusively.

The etiopathogenesis of the condition in the cat is unknown, although in humans it has been theorized that synovial cysts are related to herniations of the joint capsule, inflammation, or OA. All of the cats with elbow involvement presented with fluid-filled swellings centred on the medial aspect of a single elbow joint, typically in joints showing radiographic evidence of osteoarthritis. The osteoarthritis may have been the cause of the lameness in these cases. Plain radiography is inconclusive but contrast arthrography or ultrasonography will outline the cystic extensions of the synovial joint capsule. Confirmation of the diagnosis requires aspiration and analysis of the fluid, to confirm its synovial origin (based on viscosity, low cell counts, negative culture, etc) and biopsy of synovial membrane. Treatment options include periodic aspiration of the cyst and management of the underlying degenerative joint disease or surgical excision of the cystic cavities. Surgery can be curative but, local recurrence is likely, based on the reported cases.

Patellar Malformation

Acute traumatic fractures of the patella are discussed in Chapter 10. Malformation is occasionally encountered, usually as an incidental finding on stifle radiographs. The bone is divided into two (bipartite), three (tripartite), or more (multipartite) parts, and the abnormality may be unilateral or bilateral. In bilateral cases it would seem most likely that the aetiology is congenital. The lack of patella continuity is thought to be associated with an area of reduced vascularity. In some unilateral cases the lesion may represent chronic nonunion patella fracture.

Soft Tissue Calcification

(Chandler & Beale 2002, Freire et al. 2010, Ganey et al. 1994, Mahoney & Lamb 1996, Reinke & Mughannam 1994, Voss et al. 2017, Whiting & Pool 1985).

Calcification of the feline meniscus and other soft tissue structures within the stifle joint, such as synovial membrane and fat pad, is a common incidental radiographic finding in the stifle joint of cats and has also been reported in the tiger. Meniscal mineralizations are variable in size, shape, location and microscopic appearance, commonly affecting the cranial pole of the medial meniscus but also other areas within the meniscus. Other mineralizations and ossifications of soft tissues tend to be found in the tissues cranial to the menisci, particularly osteoarthritic stifles and in joints with CrCL pathology.

It is thought to occur either as a normal anatomic variant or secondary to joint trauma. The radiographic appearance, visualized on the mediolateral view, is of single or multiple foci of calcification in the cranial compartment of the joint. The condition may be unilateral but is frequently bilateral; it is sometimes associated with lameness or may be seen in combination with other disorders of the stifle, such as CrCL rupture or as incidental findings. Therefore, the clinical significance of meniscal calcification in the cat is still unknown, since no difference in pain scores were found between radiographically normal stifles and stifles with just meniscal mineralization in one study. In the absence of any other cause of stifle lameness, medial meniscectomy has been advocated and may be curative. Although the best possible treatment for cats with CrCL rupture is still debatable, stabilization of the stifle joint is likely to produce the best long-term results. Meniscectomy has been advocated to treat meniscal calcifications but this approach has to be carefully considered, given its negative mechanical consequences on the stifle joint and the likelihood that these mineralizations are incidental findings.

Osteochondritis Dissecans

(Bright 2010, Butcher & Beasley 1986, Hanna 2013, Palierne et al. 2010, Peterson 1984, Ralphs 2005).

There are two reports in the English language literature of cats with lesions resembling osteochondritis dissecans (OCD) of the proximal humerus, as commonly seen in dogs. There are also reports of OCD lesions of the lateral femoral condyle of the stifle joint and the patella. All the reported cases were immature cats that presented with lameness, which resolved after surgical treatment. Lumbosacral OCD has also been described in the cat.

Osteochondrodysplasia in the Scottish Fold Cat

(Malik 2001, Malik et al. 1999, Mathews et al. 1995, Simon 2000, Takanosu et al. 2008).

Osteochondrodysplasia is a type of skeletal dysplasia, determined by an inherited defect in the Scottish Fold cat caused by a simple autosomal dominant gene. This breed originated in Scotland by crossing naturally occurring spontaneously mutated farm cats with folded ears with British and American Shorthair cats and, currently, their breeding is discouraged or banned. An autosomal incomplete dominant pattern of inheritance has been demonstrated for Scottish Fold cats. The characteristic folding of the ears is due to an abnormality of the aural cartilage and the typical short inflexible tail is a characteristic feature. Heterozygous Scottish Fold cats invariably become afflicted by a progressive arthritis and homozygous cats for the Fold gene develop crippling arthritis at an early age, whereas heterozygous Folds develop arthritis but more slowly. The kittens affected by osteochondrodysplasia develop an arthropathy of the hindlimbs with extensive exostosis formation around the tarsal and metatarsal joints (Figures 7.11 and 7.12)

Radiographic examinations of affected individuals suggest defective endochondral ossification. The metatarsal and metacarpal bones show reduced length and abnormal shape, accompanied by accelerated degenerative joint disease and progressive periarticular new bone formation.

The resulting lameness can be treated surgically either by excision of the exostoses or by performing bilateral pantarsal arthrodesis. Palliative radiation therapy has also been used to resolve the clinical signs of the disorder. Since this breed is a genetic mutant, all animals will invariably be affected sooner or later so the only real solution, from a moral point of view, is to stop breeding these animals.

TENDONS AND LIGAMENTS:

In the musculoskeletal system ligaments predominantly function to join bones and provide support for joints. Ligaments may also perform other functions of importance to the orthopaedic surgeon, such as the ligaments that are

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Figure 7.11 Osteochondrodysplasia. Radiographs of an affected hock. There is extensive exostosis around the tarsal bones, particularly around the calcaneus.

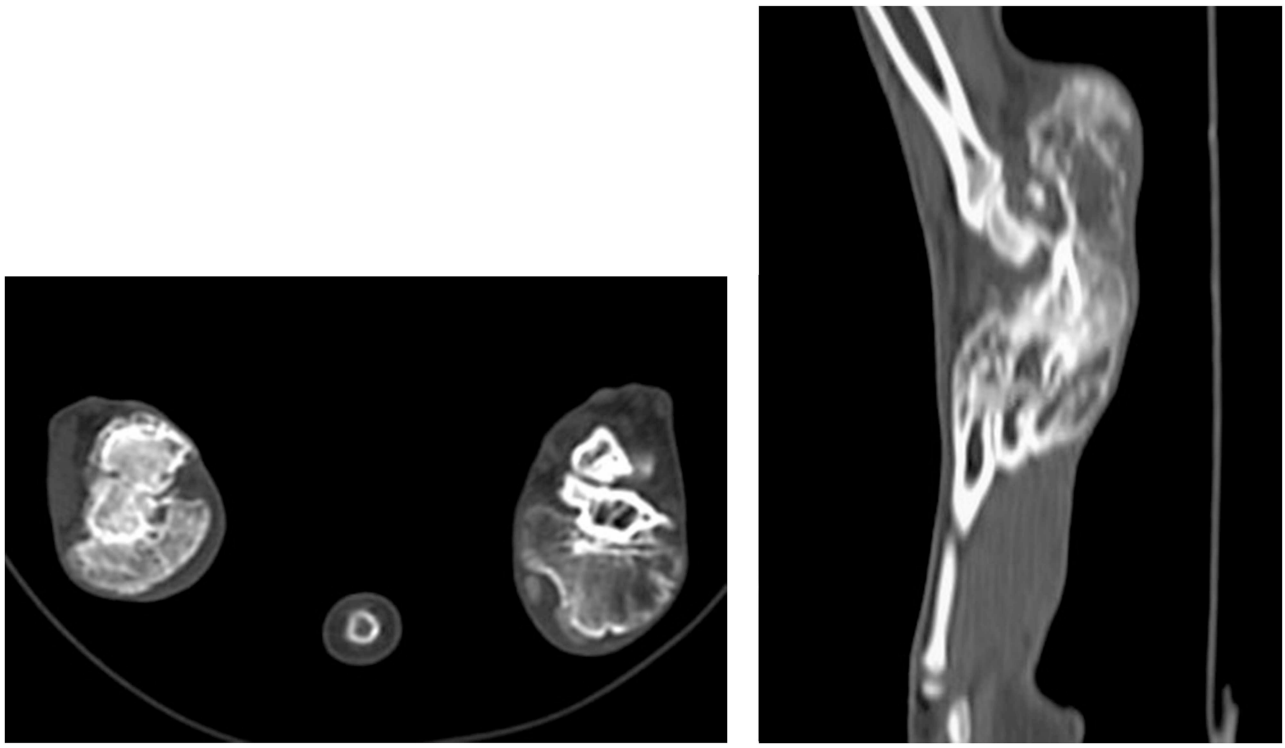


Figure 7.12 Osteochondrodysplasia. Transverse and sagittal tomographic views of the affected hocks. Observe the periosteal reaction affecting these hocks, bilaterally, especially marked on the plantar aspect of the calcaneus. (Courtesy of J. Font).

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associated with the menisci in the stifle joint. Ligaments that unite bones are strong bands consisting of longitudinally oriented bundles of collagen fibres. The collagen fibres undergo a transition to fibrocartilage where they attach to bone and are known as Sharpey's fibres. Ligaments supporting a joint may be intraarticular, as in the case of the cruciate ligaments, or, more commonly, are found as cord like thickenings incorporated into the joint capsule or separated from it by extensions of the synovial lining, termed bursae. Injuries to ligaments are known as sprains. When a ligament is avulsed from its attachment site to bone with a small fragment of bone this is known as a sprain-avulsion fracture. Ligaments and tendons heal in a similar fashion, which is described later. Tendons function to attach muscles to bone and may also provide ancillary support for joints. Although the terms are not synonymous, some parts of tendons are also known as ligaments. For example, the portion of the tendon of the quadriceps femoris muscle between the patella and the tibial tuberosity is commonly known as the patellar ligament. A muscle tendon unit (MTU) comprises a muscle belly, its origin and insertion, and the musculotendinous junction. Injury to any component of the MTU is termed a strain. Tendons consist of bundles of crimped collagen fibres and parallel rows of fibroblasts. The crimping of the fibres confers a spring-like function so that deformation occurs in a nonlinear fashion when the tendon is loaded under tension. A sac containing synovial fluid, known as a tendon sheath, surrounds the tendon where it crosses a joint. Nonsheathed tendons are enclosed in loose connective tissue known as the paratenon, which allows the tendon to glide.

Healing of Tendons and Ligaments

Strains and sprains are associated with rupture of collagen fibres and haemorrhage from torn blood vessels. There is an inflammatory response with neovascularization and invasion of fibroblasts, which lay down new collagen fibres. These fibres are at first randomly oriented but become organized within about 12 weeks of the injury. The collagen fibres regain their longitudinal orientation in response to the direction of tension within the healing tissues. Optimum healing of tendons and ligaments is promoted by complete immobilization during the initial stages of fibroplasia (4–6 weeks) and progressive loading of the tendon during the process of maturation and organization of the healing tissue (6–8 weeks). The gliding function of severely injured tendons is often compromised by excessive formation of fibrous tissue. Loss of function can be minimized by early repair, gentle tissue handling, control of infection and appropriate rehabilitation. Any delay in surgical repair will increase the likelihood of complications.

Repair of Tendons

(Bardet 1999, Das et al. 2015, Scharf et al. 2004).

Tendon disorders are relatively uncommon in the cat with the exception of injury to the tendons of the distal extremities by sharp objects. In contrast to the dog, disorders of the biceps tendon have rarely been reported. Patellar tendon injuries are rare but have been reported.

Severed tendons are repaired by suturing using fine monofilament suture materials and this primary anastomosis may require protection with external coaptation, transarticular fixation or, in the case of patellar tendon injuries, by the addition of a fascia lata graft or circumpatellar-tibia suture loops. The tendons that most commonly require suturing are the digital flexor tendons over the palmar and plantar aspects of the metacarpus and metatarsus, respectively. Severance of digital flexor tendons can occur in association with small wounds and it is important to check the integrity of the tendons at the time of first presentation. Severance or avulsion of the common calcanean tendon from its attachment on the tuber calcis is occasionally seen as a result of trauma and may be associated with a tendinopathy in older cats.

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8 Forelimb Fractures

Part 1 Fractures of the Scapula

SCAPULAR FRACTURES:

(Cockshutt 1995, Perry et al. 2012, Schmiedt et al. 2003, Tamas 1985).

Scapular fractures are rare in cats and occur as a result of trauma. The majority of cats with traumatic injuries of the scapula have concurrent, trauma-related injuries, which may include pulmonary contusions, rib fractures, pneumothorax, haemothorax and diaphragmatic rupture. Injuries to the brachial plexus and suprascapular nerve may also occur, and testing of sensation in the distal limb is recommended to confirm nerve integrity where possible (testing of suprascapular nerve function is difficult, since this nerve supplies supraspinatus and infraspinatus muscles and provides sensory function only in the region of the cranial shoulder). Feline scapular fractures may be encountered in association with scapular avulsion (see Chapter 9), a concurrent injury which should not be overlooked.

CLINICAL SIGNS:

Forelimb lameness, pain, swelling and crepitus in the scapular region.

DIAGNOSIS:

Palpation and radiography (caudocranial and mediolateral views).

Treatment and prognosis

(Bruse et al. 1989, Cook et al. 1997, Piermattei & Flo 1997, Schrader 1994).

Scapular fractures in dogs are categorized as follows:

- Scapular body: Stable
- Scapular body: Unstable
- Glenoid.

This classification system may also be applied to feline scapular fracture. Although other systems have been employed, separating fractures of the scapular body from those of the scapular neck and scapular spine (since the surgical approaches differ), the system given above is useful since it dictates whether or not surgical management is indicated. Only stable fractures of the scapular body may be managed conservatively. The other two groups of scapular fracture require surgical intervention. Most fractures of the scapular body are stable, with cats presenting with minimal lameness. Unstable fractures of the scapular body are typically of the distal body or scapular neck. Glenoid fractures are articular.

SCAPULAR BODY: Stable

Stable fractures of the body and spine of the scapula may be managed conservatively. The surrounding musculature (subscapularis, suprapinatus and infraspinatus muscles) prevents severe displacement and provides ample periosteal blood supply. Additionally, healing of the cancellous bone may be expected to be rapid. Confinement to a crate for 4 weeks is recommended. Immobilization in a Velpeau sling for 2 weeks may be considered if pain is not controlled by NSAIDs. Within 4 weeks of the injury clinical function would be expected to have improved significantly. Radiography may indicate bone healing at that stage, although a functional fibrous union may be sufficient to allow normal limb function.

SCAPULAR BODY: Unstable

Internal fixation is indicated where displacement is significant or instability palpable. Fractures of the scapular neck should always be considered unstable.

Fixation may be performed by interfragmentary wiring or plate/screw application (the latter being preferable in terms of feasibility and stiffness). Application of implants to the scapular body in the infraspinatus or supraspinatus fossae is avoided, since the bone is thin and fragile. Interfragmentary wiring involves passing loops of orthopaedic wire (0.6 mm) or monofilament suture material (size 0) through holes drilled either side of the fractures (Figure 8.1). Tightening the wire (or suture) will reduce fracture gaps, although compression is unlikely to be achieved, and overtightening will result in tearing through the thin bone (this is an exercise in alignment, not interfragmentary compression). Plates should only be applied parallel to, and at the base of, the scapular spine (cranially or caudally), where the bone is thicker, and screws should be angled to maximize bone

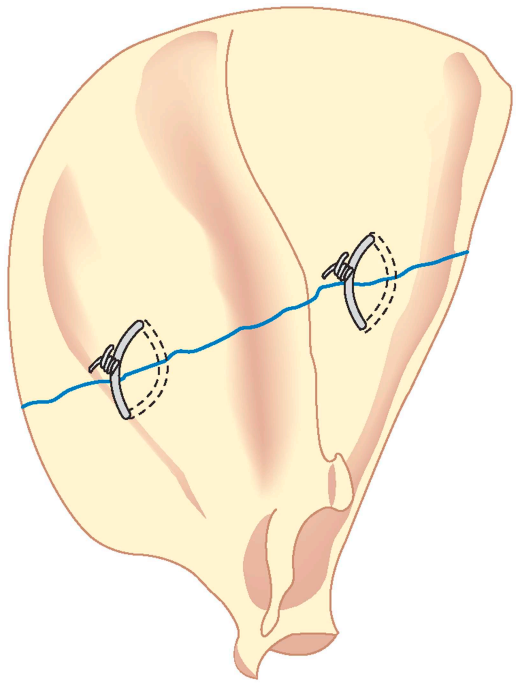


Figure 8.1 Transverse scapula body fractures may be reduced using narrow gauge wire or slowly absorbable suture material passed through holes drilled either side of the fracture line. Typically similar fractures are stabilized sufficiently by the overlying muscles, but where significant tearing of musculature is present, it may be preferable to realign bone fragments, likely resulting in a functional nonunion.

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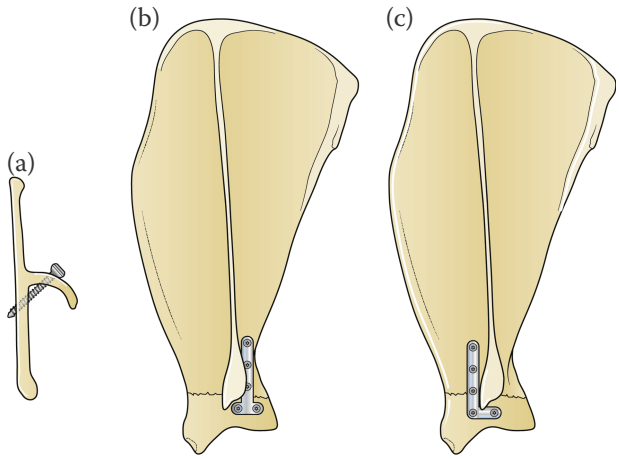


Figure 8.2 Diagrams of options for fixation of scapular neck fractures using plates and screws. Plates are placed such that screws may be inserted to maximize purchase in the thicker bone at the base of the scapular spine **(a)**. In the feline scapula the caudally projecting metacromion (or supra-hamate process) interferes with placement of a plate caudal to the scapular spine **(b)**, so cranial placement **(c)** is more commonly employed. T-plates or L-plates are often utilized such that at least two screws are placed in the scapular neck.

purchase (Figure 8.2). Care is taken to avoid penetrating the thoracic wall with the drill bit.

The acromion in the cat has hamate and supra-hamate (metacromion) processes (see Chapter 1). These may occasionally fracture independently. The fractured acromion is typically distracted distally by the acromial head of the deltoid muscle. Surgery is indicated to prevent entrapment of the suprascapular nerve within callus. This nerve courses from cranial to caudal on the lateral aspect of the scapular neck distal to the acromion process. Large fragments of the acromion process may be stabilized using pin and tension band wire fixation (to oppose the tension from the acromial head of the deltoid muscle), and this is also the technique employed to repair acromial osteotomies (Figure 8.3). A single 0.9–1.4 mm Kirschner wire (or preferably, two) is/are placed from the avulsed fragment into the spine of the scapula. The tension band wire (0.6–1.4 mm) is placed in a figure-of-eight pattern around the Kirschner wire and through a bone tunnel drilled approximately 1–2 cm proximally in the scapular spine. Small fragments of the acromial process may be reattached using orthopaedic wire alone (0.6 mm) or monofilament suture material (2–3.5 metric). The limb may be immobilized in a Velpeau sling or Spica splint for 2 weeks following surgery to protect the repair.

Fractures of the scapular neck are often transverse and displaced medially (Figure 8.4). Examination will reveal swelling and a prominent acromion process. The suprascapular nerve may be traumatized. Internal fixation is indicated to prevent nonunion, restore shoulder function and prevent entrapment of the suprascapular nerve.

A craniolateral approach is made involving ostectomy of the acromion process to reflect the acromial head of the deltoid muscle distally. Tenotomy of this muscle may also be employed but healing will be by fibrosis and its function may not be restored. The suprascapular nerve is protected

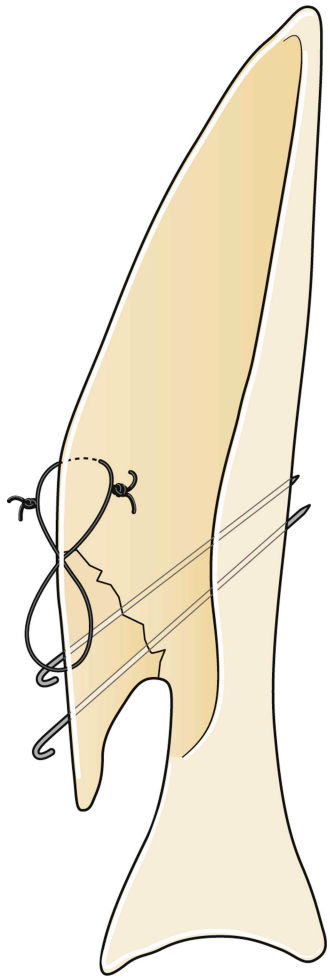


Figure 8.3 Fractures of the acromion process are stabilized using a pin and tension band wire technique when fragments are sufficiently large. One or two 1.1 mm Kirschner wires are placed to fix the acromial process into position, and compression is achieved through a tension band wire placed in a figure-of-eight pattern through a hole in the scapular spine proximal to the fracture.

during the procedure. Fixation of scapular neck fractures may involve cross pinning or plate application. Cross pinning is usually achieved using Kirschner wires placed from the glenoid margin cranially and caudally directed to engage the thin bone of the scapular body proximal to the fracture. Ideally, the Kirschner wires should cross proximal to the fracture line. Plate fixation is the authors' preference and may involve 1.5, 2.0 or 2.4 mm T or L plates or similarly sized veterinary cuttable plates (Figure 8.2). Since the distal fragment is often too small for bicortical application of more than two screws, locking plate fixation may be preferable, or conventional plates can be applied cranial and caudal to the scapular spine, essentially resulting in orthogonal plating.

Where acromial osteotomy is employed in the approach, closure includes reduction and fixation of this osteotomy with a pin and tension band wire technique or interfragmentary wire. Where tenotomy of the acromial head of the deltoid muscle is employed in the approach, repair should involve a modified locking-loop or Bunnell–Meyer suture

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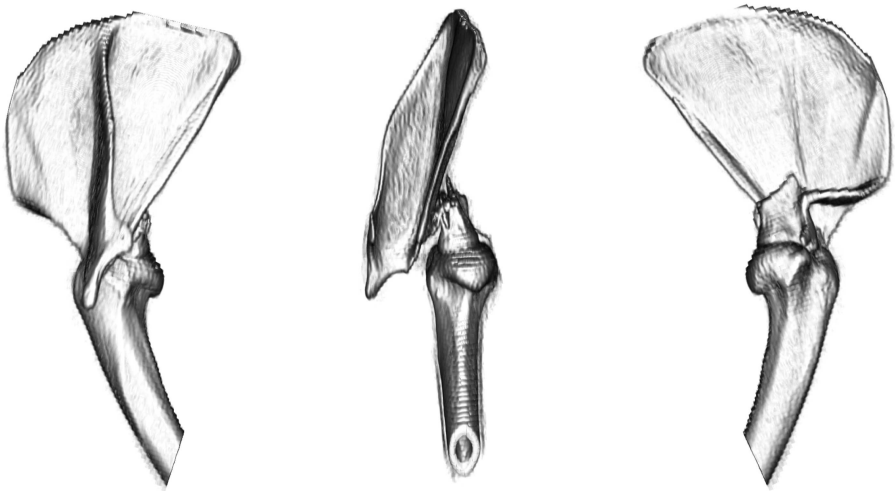


Figure 8.4 Computed tomographic 3-dimensional volume rendering reconstructions of a shoulder and scapula with a medially and proximally displaced scapular neck fracture viewed from lateral, caudal and medial. Note folding of cranial margin of the scapular body.

pattern between the acromial head of the deltoid and bone tunnels drilled in the scapular spine.

GLENOID (AND SUPRAGLENOID TUBERCLE):

(Clarke & Findji 2012, Franczuski & Parkes 1988, Johnston 1985, Mclaughlin 1998, Plesman et al. 2011, Preston & Hosgood 2015, Schrader 1994).

Glenoid fractures are articular and therefore necessitate accurate reduction and rigid fixation to maximize post-operative joint function and minimize callus formation. Small fragments may simply be removed, indeed, in one report a sizeable volume of bone including the caudal 30% of the glenoid was removed, apparently with minimal effect on joint function over 3 years. However, where possible, fragments large enough to accept implants should be stabilized using screws (1.5 mm or 2.0 mm) placed in lag fashion or multiple Kirschner wires. Anatomic reduction is important and care is taken to avoid placing implants in the glenoid cavity.

Fractures of the supraglenoid tubercle are rare in cats. They may be displaced craniodistally by the tendon of origin of the biceps brachii muscle. Anatomic reduction is required to prevent gaps or steps in the articular surface. A cranial approach is performed and the tubercle is reattached using a screw (1.0 mm to 2.0 mm), or, more commonly a Kirschner wire (or two) is placed from the tubercle into the scapular neck, oriented parallel to the biceps tendon of origin to reduce bending loads. A figure-of-eight tension band wire technique should also be employed passed around the Kirschner wire(s) and through a hole drilled in the cranial border of the scapular body.

After fixation, the limb may be immobilized in a Velpeau sling for 2 weeks.

Where articular comminution is severe and irreparable, or if pain persists after fixation, excision arthroplasty or shoulder arthrodesis may be indicated.

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Part 2 Fractures of the Humerus

(Bardet et al. 1983, Tamas et al. 1985, Vannini et al. 1988).

Humeral fractures in cats generally result from trauma and most occur in the distal portion of the bone. Open reduction and internal fixation are preferred in most cases of humeral fractures, since immobilization of the humerus using external coaptation is difficult and cats are unlikely to tolerate spica splints. The approach to the humerus may be medial or lateral (or both). Minimally invasive approaches can be made laterally and medially; however, the proximal approach to the medial aspect is hampered by the insertion of the pectoralis muscles. Fractures may be categorized according to the location on the humerus (proximal, diaphyseal, supracondylar and condylar).

DIAGNOSIS:

The diagnosis is confirmed radiographically. The cat must be carefully evaluated for concurrent injury to the thorax and nervous system (brachial plexus, radial nerve, median nerve).

Proximal humeral fractures

(Prieur 1989).

Proximal Physeal/Apophyseal Fractures

Proximal humeral fractures are rare in the cat and are typically physeal fractures in skeletally immature cats. They may involve the greater tubercle alone and/or the humeral head, which develop as separate centres of ossification. Technically the greater tubercle is an apophysis and is the insertion point of supraspinatus and infraspinatus muscles, while the humeral head is a true epiphysis. The anatomy is similar in this way, to that of the proximal tibia (the tibial tuberosity is an apophysis at which the patellar ligament inserts and the tibial plateau is the proximal tibial epiphysis). Fractures of these portions of the proximal humerus in the skeletally immature cat should be referred to according to standard nomenclature, for example, where the humeral head and greater tubercle are both involved and remain connected to one another, fractures should be described as greater tubercular avulsion fracture in combination with a Salter-Harris type I fracture of the proximal humeral physis. Sometimes proximal humeral physeal fractures involve a small additional proximal diaphyseal fracture caudally (in which case they are type II fractures). A true Salter Harris type III fracture in this region involves articular fracture of the humeral head, although this term is often used to describe fractures in which the greater tubercle (apophysis) and humeral head (epiphysis) are both separated from one another and from the humeral diaphysis.

Proximal humeral physeal/apophyseal fractures are rare in cats. Where displacement is minimal, the limb can be immobilized in a Velpeau sling for 2–4 weeks. When displacement is more severe, internal fixation is preferred to achieve anatomic reduction and fixation. Reduction may be aided by extension of the shoulder. Fixation typically involves one or, preferably, multiple Kirschner wires placed normograde through apophyseal and/or epiphyseal fragments into the humeral diaphysis (Figure 8.5). Trauma to articular surfaces and musculotendinous structures crossing the joint should be avoided. Placement of wires may be performed using a closed technique particularly where intra-operative imaging is available. Pins are bent over and cut short to reduce irritation. In larger or adult cats an additional tension band may be considered in certain circumstances, for example, following greater tubercle avulsion repair, to resist traction during shoulder flexion.

Humeral Neck Fractures

In immature cats smooth Kirschner wires are placed for fixation of reduced fractures of the humeral neck, while in mature cats, these may be stabilized with a lag screw (2.0–2.7 mm) and a single (anti-rotational) Kirschner wire (Figure 8.6). Implants may be placed retrograde (inserted from the fracture surface to emerge cranially from the greater tubercle) then driven a few millimetres into the humeral head following fracture reduction. It is important to avoid penetration of the shoulder joint, and pre-operative measurements from radiographs allow the surgeon to plan how far implants may be driven.

Articular Fractures of the Humeral Head

(Pond 1975).

Fractures involving the articular surface of the humeral head are rare. Fragments are reduced and stabilized using Kirschner wires and/or a lag screw driven from distal to proximal from the humeral shaft or greater tubercle into the base of the fragment. If the articular fragment is very small, the Kirschner wires can be inserted from the articular surface into the subchondral bone and countersunk below the articular cartilage (Figure 8.7). This technique injures the articular surface but improves the accuracy of implant positioning in small fragments. It should be used only when necessary.

Diaphyseal humeral fractures

(Cohen et al. 2012, Fearnside et al. 2002, Harari 2002, Langley-Hobbs & Straw 2005, McLaughlin 1999, Montgomery et al. 1988, Piermattei 1992, Peirone et al. 2002, Piermattei & Flo 1997, Roush & McLaughlin 1998, Schrader 1994, Wallace Berg 1991).

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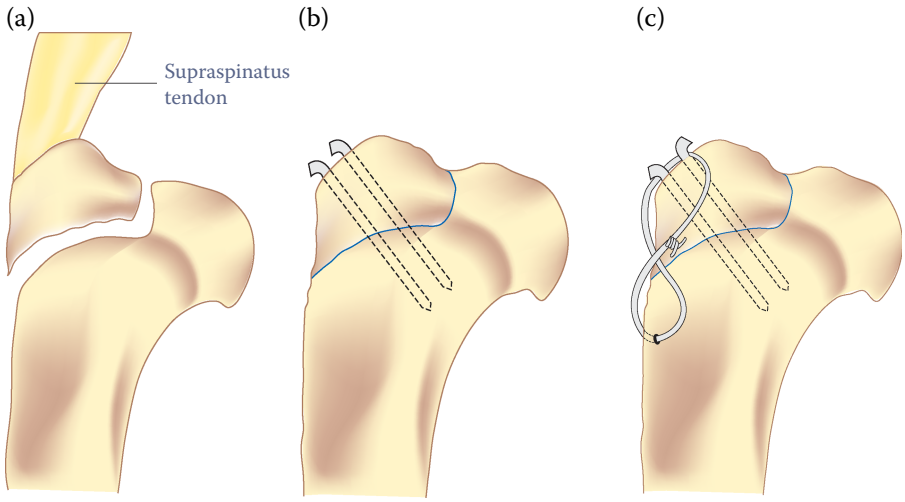


Figure 8.5 Fixation of fractures of the greater tubercle. **(a)** avulsion fracture of the greater tubercle, which will rotate about the supraspinatus tendon of insertion. **(b)** Fixation of the greater tubercle avulsion fracture using two parallel Kirschner wires driven from cranio-medial in a proximal to distal direction to avoid penetration of the shoulder joint and engage the caudal cortex of the proximal humeral diaphysis. **(c)** Tension band fixation may be combined with the Kirschner wire fixation where stability is considered inadequate, particularly in large and adult cats.

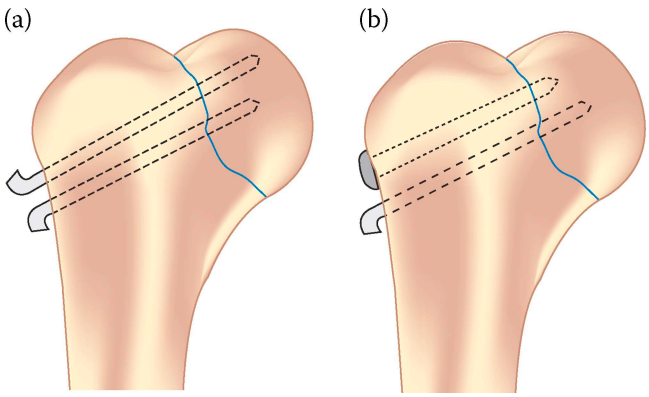


Figure 8.6 Fixation of fractures of the humeral neck (resulting in a free humeral head). **(a)** In immature cats this should involve two parallel Kirschner wires placed from craniomedial toward the articular surface. **(b)** In mature cats a lag screw may be used to achieve interfragmentary compression if sufficient space is available. An additional anti-rotations Kirschner wire is recommended.

Diaphyseal humeral fractures in the cat are typically transverse or oblique, often with mild comminution. The humeral diaphysis is exposed through either a craniolateral or medial approach. The radial nerve should be identified and protected during the lateral approach to the bone. The median nerve and brachial artery pass through the medially located supracondylar foramen from caudal to cranial. The ulnar nerve, located beneath the short part of the medial head of the triceps brachii muscle, must also be protected. Stabilization of diaphyseal humeral fractures can be achieved using several methods.

Techniques for Management of Diaphyseal Humeral Fractures:

EXTERNAL COAPTATION:

External coaptation may be considered for stabilization of nondisplaced fractures in immature cats. A Spica splint or Velpeau sling is applied until the fracture is healed.

INTRAMEDULLARY PINNING:

Intramedullary Pinning of the Humerus from Proximal

The relatively straight humerus in cats allows for intramedullary placement of a Steinmann pin (2.0 mm to 3.2 mm diameter), typically placed normograde from the greater tubercle. Intramedullary pins are placed from proximal if the fracture is in the mid-diaphysis or more proximal. Two important anatomic features of the feline humerus must be considered when placing an intramedullary pin from proximal:

- Narrow medullary cavity distally – a pin is selected that will completely fill the distal medullary cavity proximal to the condyle (this will commonly be a pin of diameter 2 mm to 2.5 mm), but attempts to seat the pin into the medial epicondylar crest are unlikely to be successful.
- Supracondylar foramen – attempts to drive pins distally into the medial aspect of the condyle should not be made, since they may exit the bone and enter the supracondylar foramen potentially resulting in iatrogenic neurovascular injury. Pins should terminate 5–10 mm proximal to the medial epicondyle.

For retrograde insertion of intramedullary pins in the feline humerus, the pin is directed proximally from the fracture site along the cranial surface of the medullary cavity to exit at the tip of the greater tubercle. The pin is inserted until its distal end is just proximal to the fracture line. The fracture is then reduced and stabilized with a bone clamp. The pin is driven distally and seated into the distal humerus just

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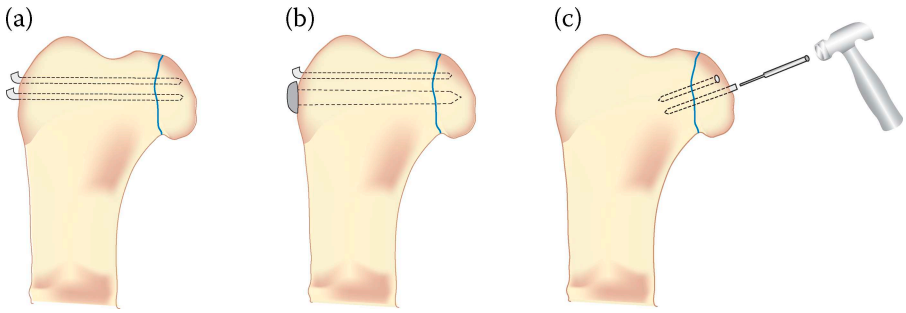


Figure 8.7 Fixation of fractures of the articular surface may require visualization by arthrotomy. Kirschner wires may be driven normograde from the greater tubercle or retrograde from the fracture surface to engage the small articular fracture fragment **(a)**. Use of a lag screw should be considered, but the fragment may be too small to permit engagement of many threads, in which case Kirschner wires alone may be adequate **(b)**. Small Kirschner wires may be placed from the articular surface if arthrotomy permits access. **(c)** Wires should be countersunk beneath the articular surface.

proximal to the supracondylar foramen. Proper insertion is confirmed and the pin is cut short.

For normograde insertion of intramedullary pins in the feline humerus, the pin is started at the proximolateral aspect of the greater tubercle (proximal to the insertion of infraspinatus tendon) and driven distally across the fracture and into the distal humerus (terminating just proximal to the supracondylar foramen). Redundant steps to prevent malpositioning include use of a second pin of identical length to determine how far distally the pin has been placed, and manipulation of the elbow to ensure the pin has not penetrated the joint (unlikely). The pin is then cut flush with the bone surface at the greater tubercle to minimize impingement on overlying tissues. Closed insertion of the intramedullary pin may be possible if the fracture is minimally displaced or if adequate reduction of the fragments is achieved by manipulation.

Intramedullary Pinning of the Humerus from Distal

For distal diaphyseal humeral fractures normograde pin placement can be performed from distal to the medial humeral epicondyle, provided a medullary canal is present in the medial epicondyle).

Intramedullary pins may rarely be used alone if reduction is good and interdigitation of the fragments provides rotational stability. In most cases, however, ancillary fixation is encouraged, consisting of an external skeletal fixator (which may be tied-in) or bone plate and screws (plate-rod construct). Cerclage wires, hemicerclage wires and lag screws may also be considered, in an attempt to control rotational and compressive forces at the fracture site. Stack pinning (placing several pins into the medullary cavity) does not completely prevent rotation but has been reported for humeral fractures in cats.

DYNAMIC INTRAMEDULLARY CROSS-PINNING (RUSH PINNING):

Kirschner wires inserted with the dynamic intramedullary cross-pinning technique achieve three-point fixation that provides rotational and axial stability. Rush pinning may be used to stabilize fractures of the proximal or distal diaphysis and metaphysis in cats. Two Kirschner wires are inserted into the medullary cavity (Figure 8.8). For proximal fractures, the Kirschner wires are started at the greater tubercle medially and laterally and driven distally to reflect off the inner cortical wall distal to the fracture. For distal fractures, the wires are started just cranial to the medial and lateral epicondyles and driven to reflect off the inner cortical wall proximal to the fracture (since the lateral and medial epicondyles are narrow or possess no medullary cavity at all, placing these pins can be difficult). Pins should glance off the inner cortical wall and continue in the medullary cavity ideally until they engage the

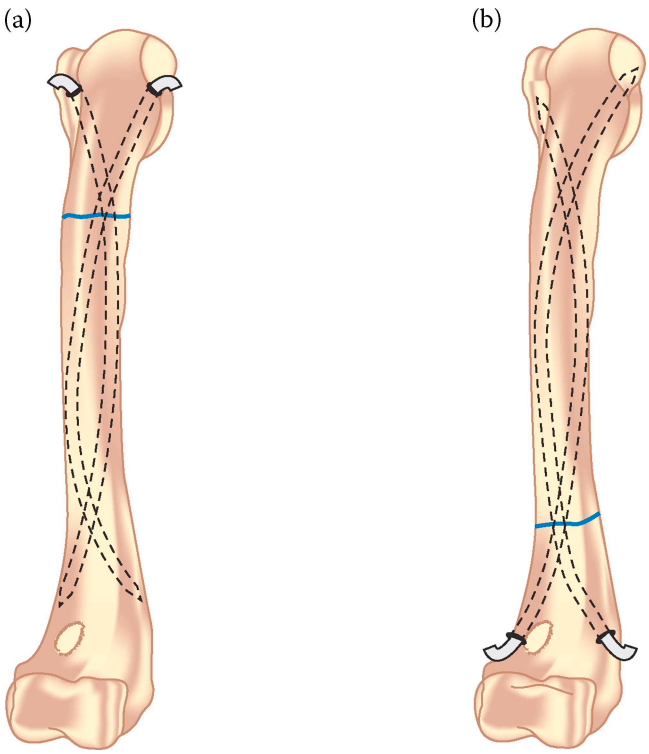


Figure 8.8 Dynamic intramedullary cross-pinning for fixation of proximal and distal diaphyseal fractures of the humerus (craniocaudal views are shown). **(a)** Proximal humeral fracture. Pins are inserted from the proximal aspect of the greater tubercle medially and laterally. They are driven distally aiming to cross over and contact the endosteal surface distal to the fracture such that they glance off the inner cortical wall. They may cross over again before engaging the opposite cortical wall. **(b)** Distal humeral fracture. Pins are inserted cranial and proximal to the epicondyles. As for the proximal fracture technique the pins should cross over, glance off the inner cortical wall and engage the cortical wall on the side of the bone from which they started.

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BOX 8.1 TYPE I ESF APPLICATION TO THE HUMERUS

Transcondylar fixation pins are typically driven from lateral to medial and they should start just distal and cranial to the lateral epicondyle. The pin is driven parallel to the joint surface to emerge just cranial and distal to the medial epicondyle. Pins up to 2.5 mm in diameter are recommended for transcondylar use. A second half pin is inserted in the proximal humerus (the greater tubercle is readily palpated). Accommodating more half pins in the proximal humerus is uncomplicated, but more consideration to the local anatomy must be made when attempting to place more half pins in the distal humerus. The epicondyle provides insufficient bone for secure fixation and pin placement here risks penetration of the anconeal fossa, interfering with anconeal excursion during elbow extension. Therefore, a second pin is placed proximal to the condyle. The radial nerve is of concern laterally but can be visualized through the surgical approach. The supracondylar foramen is present medially, and digital palpation of the emerging tip of the fixation pin on the medial aspect of the humerus can inform the surgeon of the location of the pin and the likelihood of iatrogenic neurovascular trauma.

cortex on the side that they started. The wires are advanced alternately either by tapping them with a small mallet or with a hand-chuck. In order to ensure that the wires pass up the medullary cavity and do not penetrate the humeral cortex, the tips of the wires should be blunt, or may be filed to have a ski-shaped tip. Predrilling of the insertion sites for each wire is recommended.

EXTERNAL SKELETAL FIXATION:

External skeletal fixation (ESF) may be applied to the humerus in a closed manner or after open reduction (pins are placed through stab incisions adjacent to the primary approach). A type I external fixator can be applied to the craniolateral aspect of the humerus. This may be combined with an intramedullary pin. The intramedullary pin may be tied-in to the frame if it is allowed to penetrate the skin proximally. A further modification to type I ESF involves placing a full (or centrally threaded) pin across the humeral condyle with the standard half pins on a lateral connecting bar and a second connecting bar bent to connect the medial aspect of the transcondylar full pin to the proximal end of the lateral connecting bar. This type of frame is often referred to as a hybrid type I–II external fixator (Box 8.1).

BONE PLATE AND SCREW FIXATION:

(Bruse et al. 1989, Cohen et al. 2012, Gentry et al. 1993, McLaughlin & Roush 1999, McLaughlin et al. 1992, Montgomery et al. 1988, Piermattei 1992, Piermattei & Flo 1997, Schrader 1994, Wallace & Berg 1991).

Bone plates and screws may be used for diaphyseal humeral fracture repair in cats. Dynamic compression plates (1.5 mm–2.7 mm) and veterinary cuttable plates are commonly used (Figure 8.9). Where anatomy is complicated (e.g. the humeral condyle), locking plates may be used to reduce the requirement for accurate plate contouring. Plates may be applied cranially for proximal fractures (to avoid the pectoral muscles medially and intrinsic muscles of the shoulder laterally), while they may be applied laterally, medially, or cranially for mid-diaphyseal fractures. Lateral plating requires that the plate be positioned beneath the radial nerve. Medial plating requires less contouring (it presents a flatter surface), but the median nerve and brachial artery must be protected as they pass through the supracondylar foramen. The supracondylar foramen may be removed using rongeurs and the artery and nerve retracted to avoid inadvertent iatrogenic trauma. For fractures involving the distal third of the humerus, plates may be applied either laterally or medially, or both. When plates and screws are being applied to the humerus, the surgeon should remember that the condyle offers significantly better bone stock than the epicondylar ridges. It is a worthy aim to achieve a transcondylar screw through the distal-most plate hole where possible.

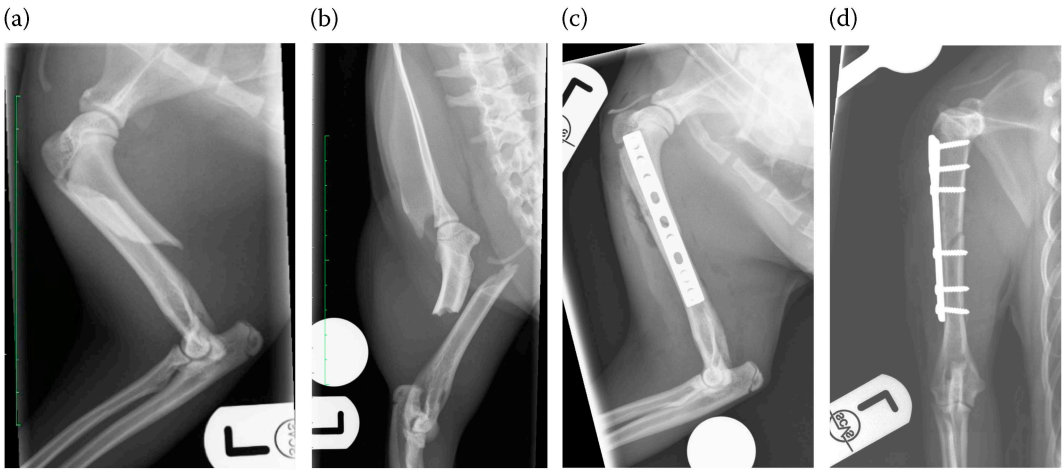


Figure 8.9 Pre-operative mediolateral **(a)** and craniocaudal **(b)** views of a humerus with a mid diaphyseal short oblique fracture. The fracture has been reduced and fixation is seen in the post-operative radiographic views **(c** and **d)** involving a 2.7 mm dynamic compression plate applied laterally (necessitating minimal contouring) with three bicortical screws in the proximal and distal fragments.

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BOX 8.2 PLATE APPLICATION TO THE HUMERUS

For non-locking plates ensure six cortices per fracture fragment. Screw purchase is poor in the distal humerus, except in the condyle, so always aim to have one transcondylar screw. Plates may be used in neutralization, or bridging fashion, and interfragmentary compression may be appropriate, depending on the fracture configuration.

- Transverse fractures – the fracture is reduced and temporarily stabilized with a bone clamp. The plate is contoured to the lateral or medial cortex, ensuring that at least three screws can be placed proximal and distal to the fracture. A dynamic compression plates may be used to compress the fracture, enhancing stability and promoting primary bone healing (if that is considered desirable).
- Oblique fractures – the fracture is reduced and temporarily stabilized with a bone clamp. A plate may be applied in bridging mode as described for transverse fractures, although any attempt to induce axial interfragmentary compression (with a dynamic compression plate) will result in shear and loss of fracture reduction. Lag screws or cerclage wires can be placed to compress the fracture and maintain reduction while a (neutralization) plate is applied. If the orientation of the oblique fracture line permits, one of the plate screws can be placed in lag fashion across the fracture line.
- Comminuted fractures – there are a few options for management of comminuted humeral diaphyseal fractures:

Controlled collapse – if the area of comminution is small, the primary fragments are apposed, as in a transverse fracture, and the comminuted fragments are used as autogenous graft. This results in minor shortening of the limb which is unlikely to affect clinical function.

Fragment reconstruction – if the bone column may be reconstructed by reduction of a few interposing fragments then cerclage wires or lag screws may be applied followed by a plate and screws in neutralization mode.

Bridging plating – the plate is applied to bridge an area of comminution with screws applied to the proximal and distal bone segments and no attempt to reconstruct the fragments, leaving the fracture envelope undisturbed (good for biology, but with increased loading on the implants and the implant-bone interface). Requires stiff and durable fixation.

Plate-rod fixation – a combination of an intramedullary pin and a bone plate reduces the strain on the plate and improves the resistance to torsional and compressive forces not addressed by an IM pin alone. According to studies performed on canine cadaveric femora the pin should fill approximately 35–40% of the medullary cavity. The pin is placed, restoring alignment and bone length. Torsional alignment is restored. The plate is contoured and applied to the medial or lateral aspect of the humerus.

INTERLOCKING NAIL FIXATION:

(Duhautois 2003).

The straight feline humerus is well suited for interlocking nails (ILN). However, the anconeal and radial fossae reduce the usefulness of ILNs in humeral fracture repair for anything other than proximal to mid-diaphyseal fractures. The ILN is inserted normograde into the medullary cavity through the greater tubercle. There are three widely available ILNs suitable for use in cats. Placing the wider diameter systems in the feline humerus is difficult, while the smaller diameter system in which the nail passes through the bolts is more easily positioned within the medullary canal.

Supracondylar humeral fractures

(Matthiesen & Walter 1984, Milton et al. 1980, Vannini et al. 1988).

Supracondylar fractures are relatively common in cats. They often involve the supracondylar foramen and are frequently comminuted. Medial and lateral approaches may be combined to allow reduction and proper implant placement. The supracondylar foramen is protected medially to avoid neurovascular injury or the medial wall of the supracondylar foramen may be excised with a rongeur to release the median nerve and brachial artery. Several fixation techniques have been reported for feline supracondylar fractures.

CROSS-PIN FIXATION:

Kirschner wires (1.1–1.6 mm) are placed in cross-pin fashion from the lateral and medial aspects of the condyle. The wires are inserted from the nonarticular portion of the condyle, just caudal to the epicondyles, and advanced across the fracture line, exiting the metaphyseal cortex proximal to the fracture line. The anconeal fossa must be considered during pin placement. Pin diameter is severely limited by that which can be accommodated in the lateral and medial epicondyles, so this technique is rarely employed.

DYNAMIC INTRAMEDULLARY CROSS-PINNING:

The technique for Rush pinning has been described earlier and in Chapter 6. The small diameter of the lateral and medial epicondyles and the presence of the anconeal fossa complicate use of this technique and it is rarely employed.

INTRAMEDULLARY PIN PLACEMENT:

A small Steinmann pin or Kirschner wire is driven into the medullary cavity of the humerus from the medial aspect of the condyle. In most cats, the pin must be relatively small (less than 1.6 mm) to avoid entering the supracondylar foramen. If possible, the pin is advanced up the medullary cavity to the greater tubercle. It is then recessed below the surface of the condyle to prevent injury to the ulnar nerve. A study comparing retrograde and normograde placement of intramedullary pins in feline supracondylar humeral fractures was published in 2012. The cadaveric study demonstrated that intramedullary pins placed retrograde from the fracture to emerge from the medial epicondyle resulted in trauma to the articular cartilage of the trochlea in 7/16, ulnar nerve entrapment in 2/16 and trauma to the flexor muscles in 13/16. On the other hand,

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normograde placement from a point located caudodistal to the medial epicondyle avoided any iatrogenic trauma to these structures. It has been reported that only 7/17 cats possess a medullary canal in the medial epicondyle at the level of the supracondylar foramen such that 10/17 cats in that study could not be penetrated by an intramedullary pin (Langley-Hobbs & Straw 2005). Nonetheless, in those cats in which placement of an intramedullary pin is possible, this may form a useful adjunct (though not stand-alone) fixation method.

EXTERNAL SKELETAL FIXATOR:

Type I may be applied to the humerus as shown earlier. Locating a half-pin in the supracondylar bone is limited by the presence of comminution; therefore, if this method is to be employed, a tied-in intramedullary pin (provided such a pin can be placed to cross the fracture) would be considered a useful method of increasing construct stiffness. Alternatively, a hybrid type I–II ESF (in which the distal-most fixation pin is bilateral, i.e. a fully threaded pin exiting the skin medially and laterally) provides greater stiffness.

BONE PLATES:

Use of biaxial plating for comminuted supracondylar humeral fractures has been reported (Dean & Witte 2016). In that report of two cases long lateral plates were applied via minimally invasive approaches laterally and shorter plates were placed via a medial approach. Application of biaxial plates gives rigid fixation and is useful in the situation of a non-reconstructable supracondylar fracture. Distal screws in both plates purchased the humeral condyle. An alternative to medial and lateral plate fixation is to nest two narrow plates side-by-side on the lateral aspect of the humerus as shown in Figure 8.10.

Condylar Fractures

(Harari 2002, Matthiesen & Walter 1984, Silva et al. 2012, Vannini et al. 1988).

The feline humeral condyle is relatively uniform and lacks the supratrochlear foramen. In addition, the common predisposing factor to humeral condylar fracture in dogs (humeral intracondylar fissure) has not been reported in the cat. Thus, fractures of the humeral condyle are uncommon in cats. Where such injuries have been reported, they are often bicondylar fractures associated with supracondylar comminution, although fractures of the lateral part of the condyle, the medial part of the condyle and non-comminuted bicondylar fractures are also possible. Open reduction and internal fixation are required to reconstruct the articular surface accurately and stabilize the fracture.

MEDIAL OR LATERAL CONDYLAR FRACTURE (FIGURE 8.11):

A lateral or medial approach is made. The fractured aspect of the condyle is reduced and held with a bone clamp (small pointed reduction forceps or vulsellum forceps). A transcondylar screw may be placed from medial or lateral. Drilling from outside-in, as has been reported in dogs to permit lag function of the screw, may be considered; however, a positional screw may be placed following accurate reduction of the condyle and compression using forceps, to avoid removing too much bone from a relatively small diameter condyle. The transcondylar screw may be 2.0 mm, 2.4 mm or 2.7 mm (the largest screw the surgeon can confidently place should be selected).

BICONDYLAR FRACTURE:

These fractures are often referred to as T or Y (or Y-T) fractures. In cats they are rare and are often associated with high energy trauma; therefore, supracondylar comminution is not uncommon. Surgical management involves combining medial and lateral approaches to the condyle. Where there is supracondylar comminution reduction of the condyle should be the first stage of the repair. However, where there is minimal or no comminution, it is possible to reduce one part of the condyle to the distal humeral diaphysis, applying fixation with lag screws and/or an epicondylar plate, followed by an approach to the contralateral side and reduction of the second part of the condyle, applying similar fixation again. As part of the fixation a transcondylar screw is recommended.

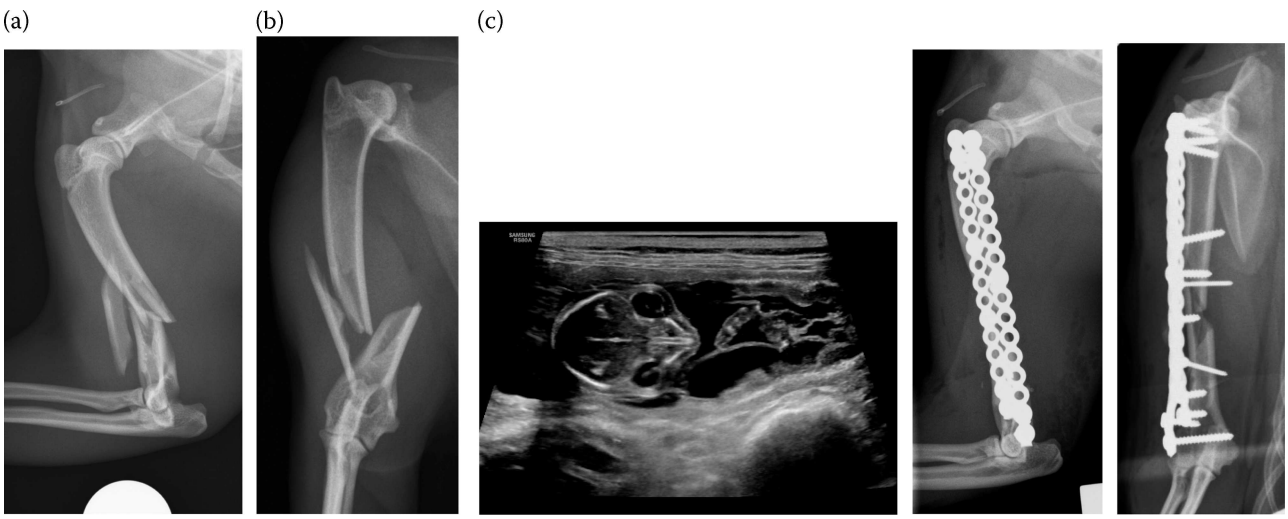


Figure 8.10 Pre-operative mediolateral **(a)** and craniocaudal **(b)** views of the humerus in a cat with a supracondylar fracture with a large butterfly fragment and fissuring extending distally toward the condyle (see **b**). This cat was heavily pregnant **(c)** and underwent minimally invasive surgery using two nested laterally-positioned cuttable malleable plates with screws placed through three short incisions at the levels of the greater tubercle, mid diaphysis and condyle.

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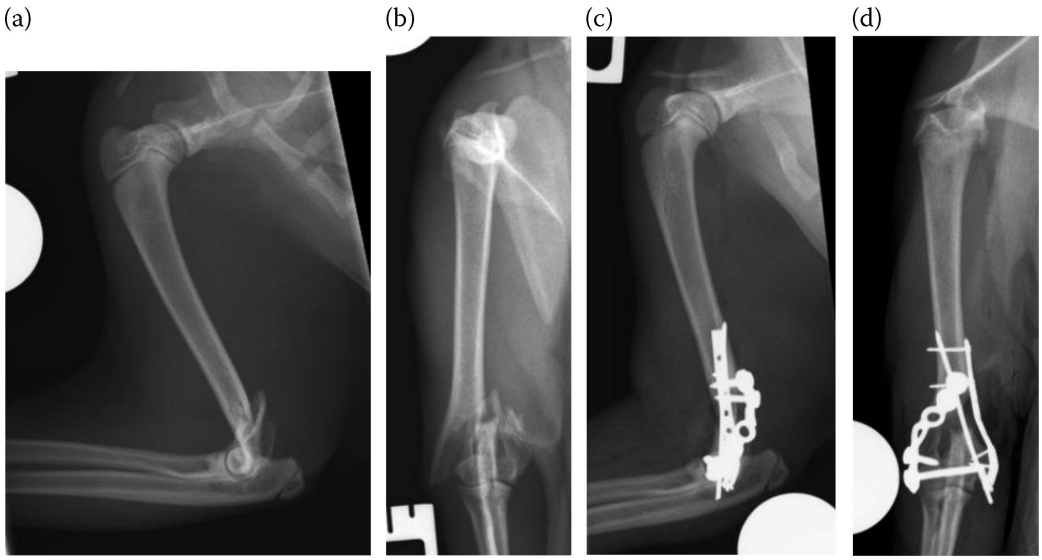


Figure 8.11 Unusual distal humeral fracture in which a combination of techniques has been applied. The fracture **(a** and **b)** is essentially supracondylar, with comminution on the medial aspect and extends obliquely to terminate very distally on the lateral aspect. Fixation **(c** and **d)** involved bilateral plating with a 1.1 mm Kirschner wire driven from the medial epicondyle to engage the lateral cortex of the proximal segment augmented with a 1.0 mm plate with three short screws distally and two in the humeral diaphysis; the lateral portion has been bridged using a 2.0 mm cuttable malleable plate contoured such that screws in the diaphyseal segment are directed from caudal to cranial and with one screw in the distal segment positioned transcondylar to maximize purchase within this useful volume of bone.

BOX 8.3 TRANSCONDYLAR SCREW PLACEMENT IN HUMERAL CONDYLAR FRACTURE REPAIR FOR CATS

The screw is placed from lateral to medial or medial to lateral using one of two methods:

- Outside-out. The condyle is reduced and temporarily stabilized with a bone holding forceps. A drill bit is used to drill a screw hole, starting and terminating just cranial and distal to the epicondyles. The bone tunnel should be orientated parallel to the joint surface. The hole is measured (and tapped if necessary) and the appropriate length screw is inserted. The screw will maintain the interfragmentary compression created by the forceps and these can be removed prior to adjunctive fixation, for example, with plates and screws or an external skeletal fixator. In larger cats, the screw can be inserted in lag fashion by 'overdrilling' the hole in the lateral condyle to the same size as the outer diameter of the screw (glide hole).
- Inside-out. The intracondylar fracture line is exposed by reflecting the fractured part of the humeral condyle craniodistally about the collateral ligaments. A bone tunnel is drilled from the fracture line to a point just cranial and distal to the epicondyle. The tunnel should be centred in the condyle and parallel to the articular surface. The hole may be drilled to the same size as the outer diameter of the screw if a lag effect is desired. The fracture is then reduced and temporarily stabilized with bone holding forceps. The drill bit is then passed through the hole drilled in the first part of the humeral condyle (using a drill sleeve if overdrilled), and this is used as a guide for drilling the contralateral part of the condyle. The hole is measured, tapped if necessary and the appropriate length screw is inserted.

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Part 3 Fractures of the Radius and Ulna

(Harari 2002, Preston et al. 2016, Schrader 1994, Wallace et al. 2009).

CAUSE AND PATHOGENESIS:

Fractures of the radius and ulna usually occur together as a result of trauma. Most occur in the distal one-half of the diaphysis. Open fractures occur infrequently.

TREATMENT AND PROGNOSIS:

Cats are able to pronate and supinate the antebrachium and forepaw to a greater degree than dogs, and stabilization of only the radius, as is commonly performed in dogs, may be insufficient. Fixation of both the radius and ulna is recommended in most cases of feline antebrachial fracture and was shown in one study to result in a lower rate of complications requiring revision.

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Proximal Radial Fractures

(Milton et al. 1980, Prieur 1989).

Fractures of the proximal radius may involve the radial metaphysis, proximal radial growth plate and the radial head. The articular surface is involved in some cases. Proximal radial fractures can be stabilized with Kirschner wires placed in cross-pin fashion. The wires are inserted just medial and lateral to the articular surface of the radial head and driven into the opposite cortex distal to the fracture site. If a portion of the articular surface is involved, small screws (1.0 mm to 2.0 mm) may be placed in lag fashion or small fragments may be best removed. Where the epiphyseal fragment is large enough, small T or l plates may be placed. The surrounding soft tissues and collateral ligaments are sutured to improve joint stability, since the approach is likely to be traumatic to stabilizing structures. The limb may be immobilized in a soft padded bandage or Spica splint for 2 weeks following surgery for additional support; however, early joint motion is encouraged if fracture stability permits.

Diaphyseal Radial Fractures

(Bruse et al. 1989, Gentry et al. 1993, Harari 2002, Harari et al. 1996, McLaughlin & Roush 1999, McLaughlin et al. 1992, Piermattei 1992, Piermattei & Flo 1997, Roush & McLaughlin 1998, Schrader 1994, Sardinas & Montavon 1997, Yu et al. 2011).

Diaphyseal fractures of the radius and ulna can be stabilized with external coaptation, external fixators or a bone plate and screws.

External Coaptation

External coaptation of radial and ulnar fractures is indicated if the fracture can be adequately reduced closed and the reduction is stable. This is true of some minimally displaced simple fractures, particularly those in the distal half of the bone and the rare fractures in which the ulna remains intact. A cast or splint is placed from the paw to the mid-humerus, immobilizing both the carpus and elbow joints. Placing the splint on the lateral aspect of the limb will allow immobilization of the elbow joint at a functional angle.

External Skeletal Fixation

External fixators can be used to stabilize most fractures of the radial diaphysis. They are particularly useful for repair of open and comminuted fractures since they are easily removed following healing.

Bone Plate and Screw Fixation

Bone plates and screws are the preferred method for fixation of antebrachial fractures in cats. Plates are applied to the flat cranial surface of the radius (craniomedial approach) and the flat lateral surgery of the ulna (caudolateral approach). Application of a bone plate to the medial surface of the radius has also been described. As the plate is edge-loaded in this position, the fixation is somewhat stiffer. In addition, a plate may be placed medially on the radius where a wound overlies the cranial aspect of the bone, to avoid a surgical incision through a potentially contaminated site. Low profile plates facilitate closure of the soft tissues after plate application.

Intramedullary Pinning

Occasionally an intramedullary pin is placed in the ulna (in combination with a plate on the radius, this may be described as a plate-rod technique, Figure 8.12) to provide additional support. The pin may be inserted normograde from the olecranon process or retrograde from the fracture site, or, for distal ulnar fractures, narrow intramedullary pins may be placed retrograde from distal (i.e. driven into the distal fragment from the fracture and driven back following fracture reduction to be flush with the styloid process).

Intramedullary pinning is not recommended for repair of radial fractures. Insertion of the pin is difficult without damaging the carpal or elbow soft tissues and would inevitably traumatize the articular cartilage. However, a 'dowel' pinning technique for placing short length intramedullary pins in the radius from the fracture has been shown to improve radial fracture reduction in small breed dogs when combined with external skeletal fixation, and this may be considered for the cat if reduction is particularly difficult to achieve or unstable.

Box 8.4 TYPE 1 EXTERNAL SKELETAL FIXATOR IN THE FELINE RADIUS

Type I ESF (unilateral frame/half pins) is easy to apply. Because the feline radius is relatively flat in the sagittal plane, placement of transfixation pins from medial to lateral can be challenging and pin insertion from craniomedial to caudolateral is preferred. The proximal half pin is inserted parallel to the radiohumeral joint through a stab incision in the skin over the proximal, craniomedial aspect of the radius. The distal half pin is inserted parallel to the radiocarpal joint through a stab incision over the distal, craniomedial aspect of the radius. These two pins are used to ensure proper rotational alignment of the fracture and may be used to provide distraction if needed. They are connected to a connecting bar. Additional half pins are then inserted.

If an acrylic connecting bar is to be used, the half pins are inserted and connected with flexible tubing. Special pins designed for use with acrylic fixators exist (with roughened surface on the non-threaded shaft to improve the durability of the acrylic-pin interface). The fracture is reduced and the tubing is filled with acrylic.

A type IIb fixator (bilateral frame with full pins and half pins) may be applied if comminution of the fracture necessitates additional stiffness. The proximal full pin is inserted through the radius parallel to the radiohumeral joint. The distal full pin is inserted through the radius parallel to the radiocarpal joint. Connecting bars are attached to the transfixation pins on each side of the limb. The fracture is reduced, the clamps are tightened and additional half pins are inserted as necessary.

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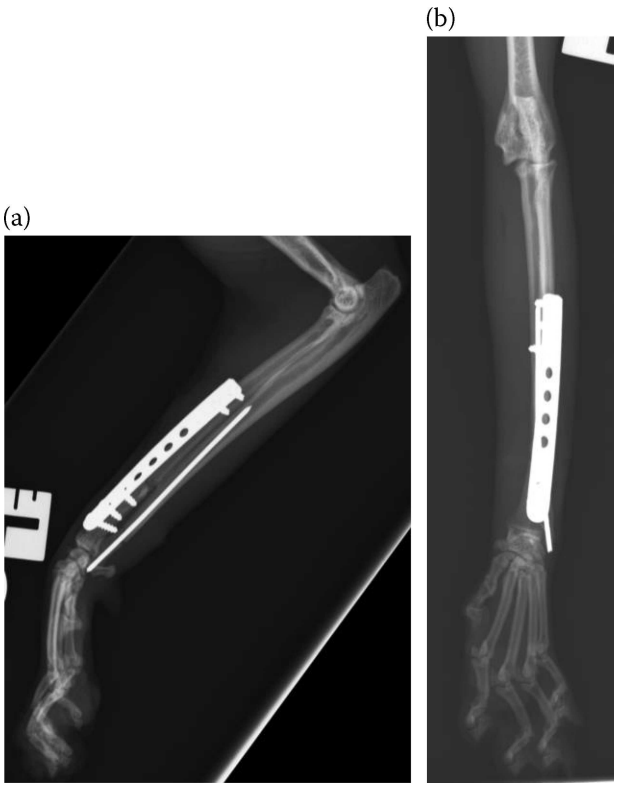


Figure 8.12 Plate-rod fixation of feline antebrachial fracture mediolateral **(a)** and craniocaudal **(b)** post-operative images demonstrating craniomedial position of radial plate with three screws proximally and distally and combination with intramedullary pin in the ulna placed retrograde from distal.

DISTAL RADIAL FRACTURES:

(McLaughlin 1995, Milton et al. 1980, Prieur 1989, Roush & McLaughlin 1998, Sardinas & Montavon 1997).

Fractures of the distal radius include Salter-Harris fractures of the distal radial growth plate, metaphyseal fractures, epiphyseal fractures and articular fractures. Pancarpal arthrodesis may be indicated for irreparable, severely comminuted articular fractures of the distal radius.

External Coaptation

External coaptation is ill-advised if stability is unlikely to be achieved in juxta-articular fractures. External coaptation may be indicated for fractures that can be reduced closed and are relatively stable after reduction. Distal radial fractures in immature cats and fractures in which the ulna is intact may be amenable to cast or splint fixation.

Cross-Pin Fixation

Distal metaphyseal fractures and fractures of the distal radial growth plate may be stabilized using two Kirschner wires (0.9–1.6 mm) placed in cross-pin fashion. The wires are inserted from the medial and lateral aspects of the distal radius, avoiding the articular surface. Removal of the implants may be necessary if pin migration or soft tissue irritation occurs. Cross-pin fixation should be considered for distal radial physeal fractures, and implants should be removed within 3–4 weeks in cats under 5 months of age to mitigate the influence of physeal bridging. Limb deformities associated with cessation of growth in the distal radius are rare in cats, but clients should be aware of the potential for their development.

External Skeletal Fixation

A type Ia external skeletal fixator can be applied to the craniomedial aspect of the distal radius if the distal fragment will accommodate at least two transfixation pin. A lightweight acrylic/putty bar (or even just an irregular mass of acrylic/putty) is an alternative, to connect transfixation pins driven from irregular angles into the bone distal and proximal to the fracture.

Bone Plate and Screw Fixation

Distal radial fractures in cats are best stabilized using a bone plate and screws. The plate may be applied to the cranial cortex, in which case a T-plate may be considered to maximize the number of screws it is possible to place in the distal fragment. Medial plate application should also be considered, since the plate is favourably edge-loaded and screw lengths will be greater than with a cranial plate. An intramedullary pin in a distal ulnar fracture accompanying a plated distal radial fracture should make for robust fixation, even where only two screws (four cortices) are placed in the distal fragment. In addition to the use of T-plates or similar, locking plates should be considered in young cats (in which screw-holding strength is poor since the bone is soft) and where the size of the fragment limits the number of cortices which may be purchased.

Lag Screw Fixation

Bone screws (1.0 mm to 2.0 mm) may be placed in lag fashion to compress fracture lines, particularly for stabilization of oblique articular fractures. Augmentation of the repair may involve an additional bone plate and screws, ESF (acrylic free-form type) or external coaptation.

ULNAR FRACTURES:

(Piermattei & Flo, 1997, Preston et al. 2016, Roush & McLaughlin 1998, Wallace et al. 2009).

Stabilization of ulnar diaphyseal fractures concurrent to stabilization of radial fractures is considered more important in cats than in dogs. Stabilization of both bones in the antebrachium has been termed dual bone fixation and may involve a plate-rod technique (Figure 8.12) or double plate technique (Figure 8.13). Fixation of a fractured ulna is also considered mandatory in the following situations:

- Where the ulnar fracture is associated with a comminuted radial fracture.
- Where the ulnar fracture involves the olecranon process or trochlear notch (in which case a radial fracture is frequently not present).
- Where the fracture involves the ulnar styloid process and carpal instability exists.

Olecranon Process Fractures

The fractured olecranon process is typically displaced proximally by the triceps muscles. This distractive force must be controlled if strain on the healing fracture is to be minimized. Reduction is facilitated by extension of the elbow joint. Tension band wire fixation has been described. Two Kirschner wires (0.9–1.1 mm) are inserted from the tip of the olecranon, across the fracture site, and into the medullary cavity of the

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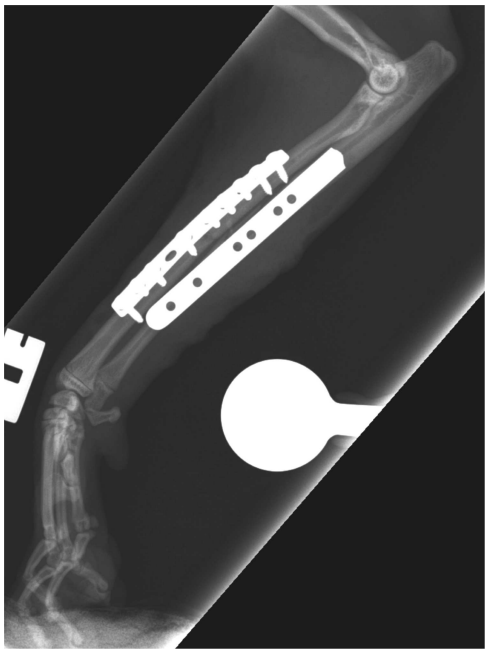


Figure 8.13 A mediolateral radiographic view following dual bone fixation for mid-diaphyseal radius and ulna fractures using veterinary cuttable plates on the cranial surface of the radius and the lateral surface of the ulna.

ulna. In smaller cats it may be difficult to position two Kirschner wires in the narrow olecranon. A single pin may be placed instead, but it will not control rotational forces, depending on the fracture configurations. A figure-of-eight tension band wire is placed through a transverse bone tunnel in the ulna distal to the fracture and around the proximal ends of the intramedullary Kirschner wires.

Bone plates may be applied to the lateral or caudal surface of the ulna as an alternative to a pin and tension band wire technique. This is preferable where reconstruction of the bone is not possible, as a result of comminution. The important feature of the olecranon is that it can resist the proximal pull of the triceps muscles following reconstruction.

Trochlear Notch Fractures

Fractures of the ulna which involve the trochlear notch are articular in nature; therefore, the principles of accurate reconstruction and interfragmentary compression are advocated. The caudal aspect of the ulna is the tension surface. Fixation of such fractures can be with a caudal tension band wire (passed through bone tunnels proximal and distal to the fracture) or, preferably, a bone plate and screws (Figure 8.14). Comminuted fractures of the trochlear notch may be reconstructed with Kirschner wires and augmented with robust internal fixation; however, accurate reconstruction may not be possible in every case due to the small size of fragments.

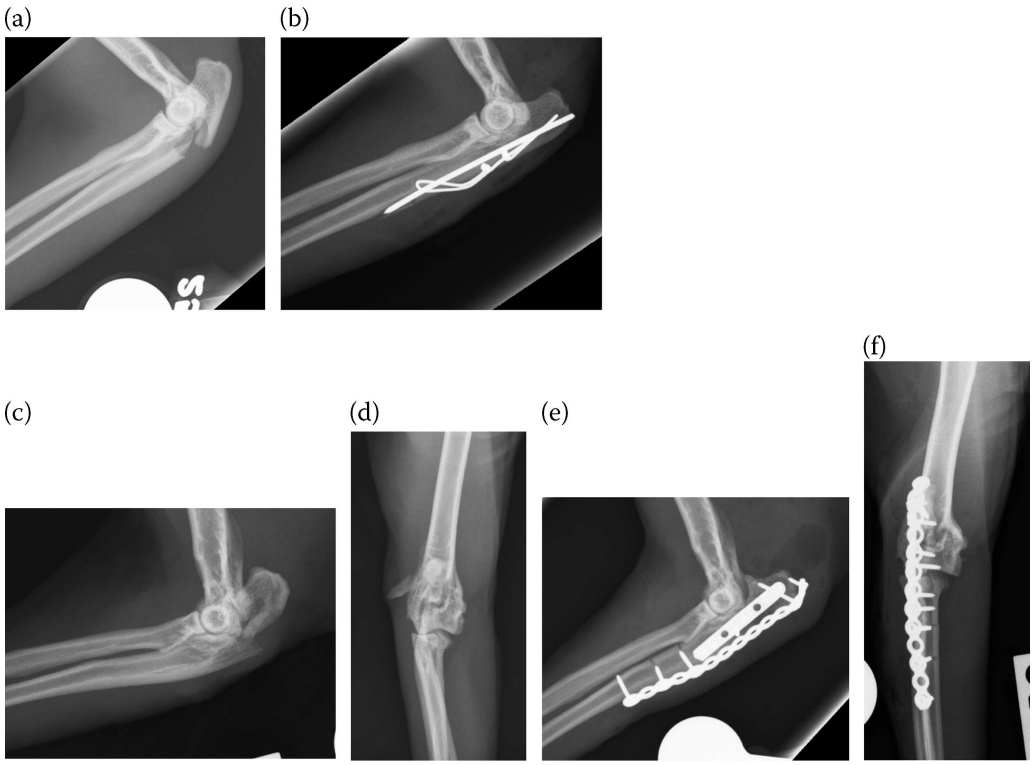


Figure 8.14 Ulnar fractures through the trochlear notch. **(a** and **b)** demonstrate mediolateral radiographic views of the elbow of a cat with a simple fracture of the trochlear notch which was reduced and underwent fixation using a Kirschner wire placed normograde from proximal and figure-of-eight orthopaedic tension band wire placed through bone tunnels in the ulna cranial to the pin. Drilling a bone tunnel for proximal fixation may be preferable to looping the wire around the protruding end of the pin since this avoids the risk of iatrogenic trauma to the triceps tendons of insertion. **(c** and **d)** show a similar fracture of the trochlear notch with additional comminution. Fixation was necessarily robust involving two orthogonal veterinary cuttable plates **(e** and **f)** since full reconstruction of the fracture was not possible. A caudal plate acts as a tension band wire to resist the distracting force exerted by the triceps muscles.

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Styloid Process Fractures

The ulnar styloid process is the origin of the lateral antebrachiocarpal collateral ligament. Fracture of the distal styloid process may be associated with joint instability or luxation. Minimally displaced fractures, particularly in immature cats, are immobilized in a caudal splint for 4–6 weeks until healing is complete. Styloid fractures may also be stabilized internally by inserting a small Kirschner wire placed normograde from the tip of the styloid process into the medullary cavity of the distal ulna. The carpus should be immobilized using external coaptation for up to 4 weeks. Carpal varus instability associated with severely comminuted styloid process fractures may be addressed by attaching the distal styloid process to the distal radius with a small lag screw. Pancarpal arthrodesis may be required for irreparable styloid process fractures accompanied by severe distal radial fracture.

Monteggia Fractures

(Bush & Owen 2009, Irubetagoyena et al. 2011, Koch 2017, Piermattei & Flo, 1997, Schwarz & Schrader 1984, Vallone & Schulz 2011).

Fractures of the ulna with concurrent radiohumeral luxation are termed Monteggia fractures (Figure 8.15). In cats, they usually result from a fall. The ulnar fracture may be simple or comminuted. Typically the radioulnar joint remains intact. Partial or complete rupture of the annular ligament and lateral collateral ligament may result in concurrent radioulnar luxation. Internal fixation is recommended to stabilize the ulnar fracture and restore joint congruence. Monteggia fractures are classified according to the direction of radial head luxation (type I cranial, type 2 = caudal, type 3 = lateral). Type IV Monteggia fractures include a fracture of the radius, and this injury has been described in two case reports in cats, suggesting that it may be somewhat more prevalent in this species than in dogs.

The ulnar fracture must be stabilized. Techniques include an intramedullary pin placed normograde or retrograde alongside a figure-of-eight tension band wire through bone tunnels drilled proximal and distal to the fracture. Plate and screw fixation is preferable, since this produces a stiffer construct (Figure 8.15). Proximal ulnar fractures may be stabilized using caudally or laterally positioned plates.

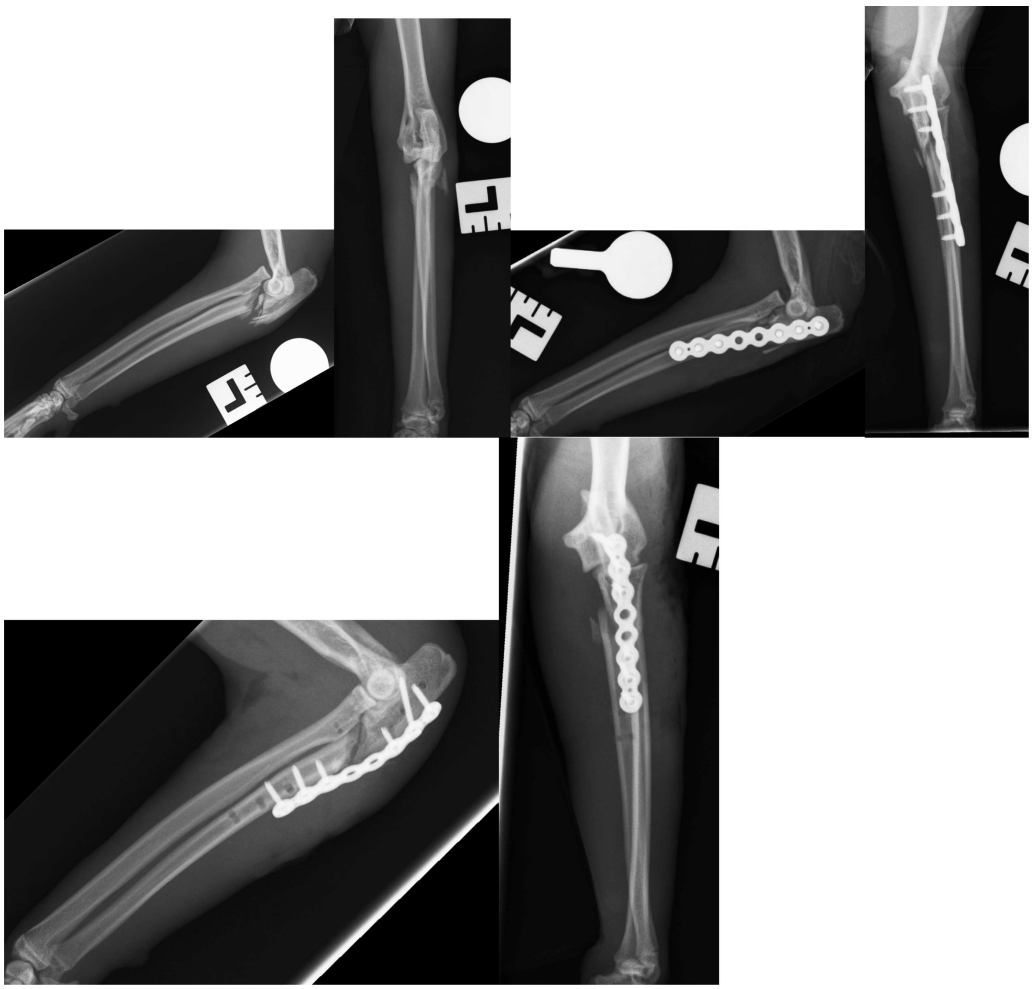


Figure 8.15 Orthogonal radiographic projections of the antebrachium of a cat demonstrating a type 1 Monteggia fracture with comminution of the ulnar component **(a** and **b).** Fixation was initially performed with a lateral plate on the ulna. Immediate post-operative radiographs **(c** and **d)** revealed persistence of cranial luxation of the radiohumeral joint. Revision necessitated removal of the implants as reduction of the radiohumeral joint was not possible with the plate and screws in place. **(e** and **f)** The repair was revised with a caudal plate (to avoid placing screws through the same bone holes) and a loop of 4 M polydioxanone suture was passed through a proximal radial bone tunnel and around the caudal aspect of the ulna at the same level, to maintain radioulnar reduction temporarily during healing.

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Ulnar fracture reduction may maintain reduction of the radiohumeral joint, provided the periarticular soft tissues of the radioulnar joint (in particular the annular ligament) remain intact. If the radioulnar joint is luxated, and fixation of the ulnar fracture does not result in a reduced radiohumeral joint then surgery to repair the torn annular ligament and lateral collateral ligament has been suggested. More typically, placing a positional screw through the ulna into the proximal radius to stabilize the radioulnar joint is recommended (temporary synostosis with the screw being removed after 2–4 weeks) to allow healing by peri-articular fibrosis. This resists pronation and supination and the screw is often loose by two weeks following surgery. Another technique involves using suture to maintain reduction of the radioulnar and radiohumeral joints, either using a Tightrope® fixation system or sutures passed through bone tunnels in the lateral aspect of the radial head and lateral aspect of the ulna. This has been hypothesized to be a superior technique for reducing the radiohumeral joint in cats, since their increased reliance on pronation and supination may increase the risk of early implant failure where more rigid radio-ulnar fixation is performed.

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Part 4 Paw

METACARPAL AND METATARSAL FRACTURES:

(Anderson et al. 1993, Benloch-Gonzales et al. 2014, Kapatkin et al. 2000, Kornmayer et al. 2014a).

Data regarding treatment of metacarpal fractures are typically combined with metatarsal fractures. Therefore this chapter refers to metabone fractures, to cover the forepaw and hind paw simultaneously. In cats the

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anatomical differences between metacarpals (narrow, short and often lacking an intramedullary canal) and metatarsals (long and slender) is notable. Nonetheless, fixation options for the two types of bone are identical. It is worth noting that the majority of these bones have greater dorso-palmar (or dorso-plantar) dimensions in comparison with their mediolateral dimensions. Fixation devices may therefore be larger when applied to the medial or lateral surfaces, rather than the dorsal surfaces; however, this is only feasible for the second (medial surface) and fifth (lateral surface) metabones. The palmar/plantar surfaces are the tension aspects of these bones, so dorsal fixation is mechanically inferior but is performed routinely as the approach is uncomplicated.

Management of metacarpal fractures has been divided into conservative and surgical. The literature gives no evidence of superiority of either approach for dogs and cats. However, none of the publications regarding management of metabone fractures have been prospective, resulting in bias in the data. To clarify, surgically managed metabone fractures have not been shown in the published literature to have a better outcome than non-surgically managed cases. However, in the few studies available, surgical management has been performed for the more severe injuries (those more prone to complications) while the conservatively managed fractures have been the less severe injuries (more likely to progress to a favourable outcome, regardless of the treatment choice). In conclusion, the literature remains unclear regarding the overall benefits of conservative versus surgical management of metabone fractures.

Severity of metacarpal fractures is judged according to various parameters:

- *Comminution* – comminution indicates more instability and greater trauma to the periosteal blood supply
- *Open/closed* – it is relatively common for metabone fractures to be open due to their minimal dorsal soft tissue coverage. Contamination means internal fixation is less desirable.
- *Multiple bones affected* – intact metabones will act as an internal splint for the other metabones.
- *Third and fourth metabones affected* – these are considered most important for loading, so while fractures involving the second and/or fifth metabones might be considered for conservative management, fractures of the third and/or fourth might not be.

CONSERVATIVE MANAGEMENT:

External coaptation may be applied for up to 6 weeks. If all four of the metabones are fractured, care must be taken to avoid angular deviation of the paw within a dressing.

SURGICAL MANAGEMENT:

(Benedetti et al. 1986, Degasperi et al. 2007, De La Puerta et al. 2008, Okumura et al. 2000, Zahn et al. 2007).

A number of techniques have been described. These include intramedullary pinning, ESF (various methods) and plate and screw application.

Intramedullary Pinning

Intramedullary Kirschner wires may be placed into the feline metatarsals; however, in a proportion of cats (2/12 in one study), the medullary cavity is too small or entirely absent, preventing pin insertion. Kirschner wires may be placed in the following three ways:

1. **Retrograde**, although this is difficult and in most cases tends to traumatize the metacarpo/metatarso-phalangeal articulation.
2. **Via a small slot or hole** drilled or burred in the dorsal surface of the metacarpal bone (at the head or base) to access the medullary cavity. A small, flexible Kirschner wire is inserted normograde from the hole, across the fracture line and into the proximal metaphysis.
3. By **dowel pinning** (Figure 8.16) in which a Kirschner wire is inserted from the fracture into one end of the bone, the pin is cut short, leaving approximately 5–10 mm exposed and the other part of the fractured bone is then distracted and placed over the end of the pin. This technique is relatively easy to employ and may be used for a single fractured metacarpal or multiple fractures alike. This technique does not lend itself to fixation of

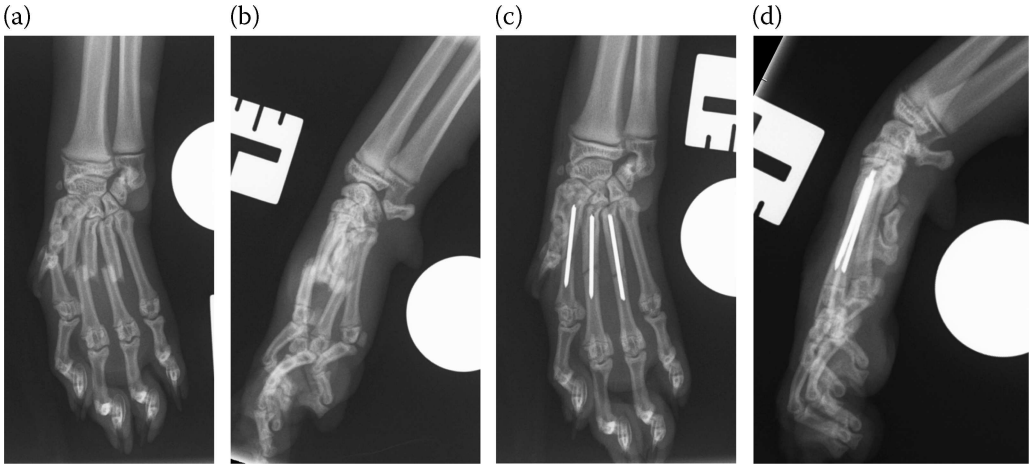


Figure 8.16 **(a** and **b)** Orthogonal radiographic projections of mid diaphyseal fractures of the second, third and fourth metacarpal bones in a skeletally immature cat. **(c** and **d)** Immediate post-operative radiographs demonstrating intramedullary pin fixation by dowel pinning. Alignment is good, fractures are well reduced and the intramedullary pins are large enough to be filling the majority of the medullary canal in these narrow bones.

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severely comminuted bones, although provided one or two (particularly of the third or fourth metabones) are intact or undergoes sufficiently robust fixation, realigning a comminuted metabone using this technique is not unreasonable.

External Skeletal Fixation

(Anderson et al. 1993, Benloch-Gonzales et al. 2014, Okumura et al. 2000).

Two different methods of application of fixation pins have been described for the metacarpus or metatarsus (Figure 8.17):

1. Pins are placed intramedullary, crossing the fracture and exiting the bone distally through a dorsal slot where they emerge from the skin to be fixed into the frame
2. Pins are placed transverse from medial to lateral (or lateral to medial).

Connecting bars may be linear (i.e. type 2 where pins are passed transversely) or freeform (more common). The described bone tunnels for placing pins transversely are small, around 1.5 to 2.0 mm in diameter. Therefore, the technique in which pins are placed intramedullary may be considered preferable; however placing pins through a dorsal slot in a distal feline metabone is difficult and may risk further fracturing of the bone.

It should be noted that the metacarpals and metatarsals are not arranged in a single sagittal plane throughout their lengths. Proximally the bases of the metacarpals and metatarsals are essentially at the same dorsopalmar/plantar level, however, distally the third and fourth metacarpals and metatarsals assume a more dorsal position in comparison with the second and fifth. Thus, it is possible to purchase all four main metabones at the level of the bases with a single transverse pin; however, it is not possible to purchase

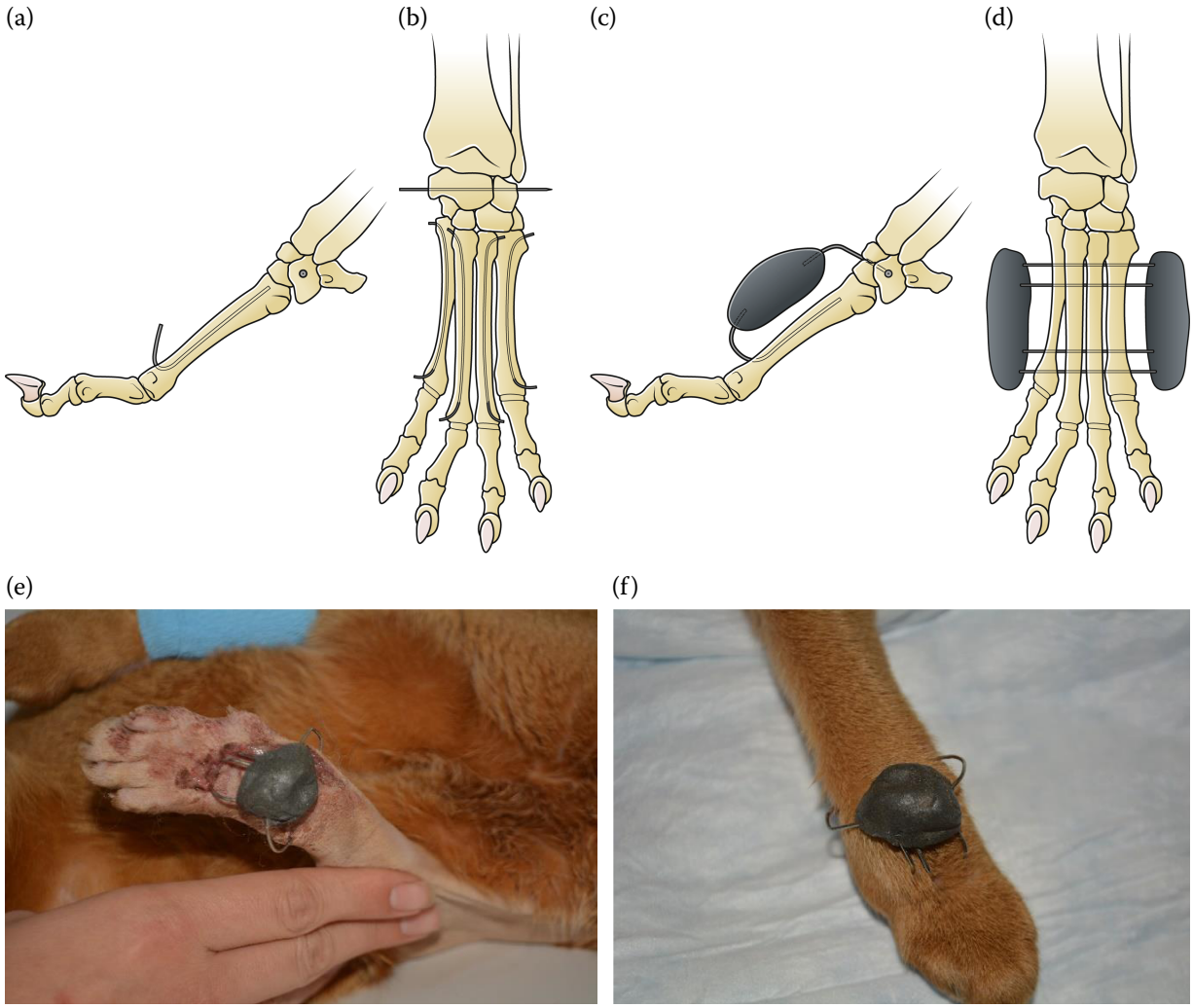


Figure 8.17 External skeletal fixators may be placed in the metabones in two ways: Intramedullary pins emerging from a slot in the dorsal surface distally **(a)**. These pins are bent backwards of the dorsal aspect of the paw. An additional pin is placed from medial to lateral across the proximal row of carpal bones or across the fourth and central tarsal bones emerging on both sides **(b)**. The ends of the pin are also bent upwards and the five pins are connected using a mass of epoxy putty or similar connecting material **(c)**. The appearance immediately following surgery and the appearance after 8 weeks, prior to frame removal, are shown **(e** and **f)**. Alternatively, a modified type 2 external skeletal fixator may be applied **(d)** with full pins proximally where the metabones are arranged in a single plane and half pins used to engage the second and third (from medial) and fourth and fifth (from lateral) bones. Epoxy putty connecting bars are placed on each side of the paw to connect the protruding pin ends.

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all four bones at the level of the mid-diaphyses or heads with a single pin. A type II frame is therefore often modified to include half pins placed obliquely to purchase the second and third (from medial) and the fourth and fifth (from lateral) metabones.

Plates and Screws

Where comminution is high or a high level of stiffness is required in the fixation (e.g. if only one or two of four fractured metabones are to undergo fixation) bone plates are a good option. The plates are applied dorsally on metacarpals three and four and abaxially for the second and fifth. Plates should be 1.0 to 2.0 mm in size.

Combinations

It is appropriate to view each fractured metabone in a single paw as an individual. Where plate/screw fixation may be appropriate for one metabone, an intramedullary pin placed through a slot or by dowel pinning may be more appropriate for the neighbouring bone. Combinations of techniques are often applicable and may be very effective.

PHALANGEAL FRACTURES:

Fractures involving the phalanges are usually managed by closed reduction and splinting for 4–6 weeks. Spoon splints or medial/lateral splints may be applied, with dressing changes (sometimes necessitating sedation, depending on the nature of the cat) on a weekly basis. Where dressings are not tolerated, confinement alone may be sufficient to achieve a functional (mal) union.

If severe trauma to the phalanges has occurred, the loss of soft tissues (including skin, tendons, ligaments and joint capsule) may complicate repair.

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9 Other Disorders of the Forelimb

Part 1 Scapula and Shoulder Joint

Other than proximal humeral and scapular fracture, trauma to the shoulder region in a cat may result in two conditions which cause severe lameness: dorsal displacement of the scapula and shoulder luxation.

DORSAL DISPLACEMENT OF THE SCAPULA (FLYING SCAPULA, SCAPULAR LUXATION):

(Kano et al. 2013, Leighton 1977).

Cause and Pathogenesis

Traumatic rupture of the serratus ventralis, rhomboideus and trapezius muscles can result in dorsal displacement of the scapula (Figure 9.1). The muscles usually tear near their insertions on the cranial angle and dorsal border of the scapula.

Clinical Signs and Diagnosis

Severe lameness is seen initially with this condition; however, despite a gait abnormality resulting from dorsal displacement of the scapula during loading, lameness reduces over a few weeks following the injury. Dorsal displacement of the scapula is visible during loading of the limb, and this is pathognomonic for this condition. Where cats are non-weight bearing (in which case the dorsal displacement may not be recognized) plain radiography is also unlikely to be diagnostic. Therefore, examination of cats with undiagnosed forelimb lameness and a suspected history of trauma, should involve an attempt to dorsally displace the scapula. Comparison of the scapular excursion over the thoracic wall with that of the contralateral limb should be diagnostic. Anecdotally folding fractures of the dorsal scapular body may be associated with dorsal displacement of the scapula.

Treatment and Prognosis

In a case report of a single cat with chronic left scapular luxation and right forelimb amputation, force plate analysis revealed force distribution similar to that expected of a right forelimb amputee. The authors concluded that scapular luxation was not an impediment to weight-bearing. Thus conservative therapy consisting of restricted activity for 2–4 weeks, may be attempted, however, dorsal displacement of the scapula persists. Surgical intervention is typically recommended to normalize limb use.

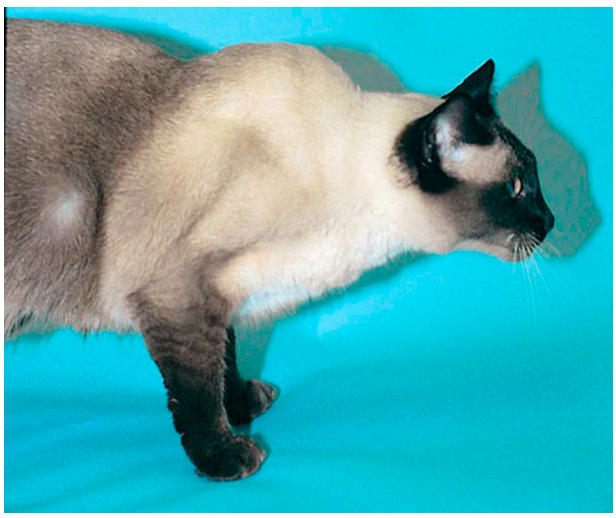


Figure 9.1 Dorsal displacement of the scapula.

Surgery involves an approach to the scapula to allow its lateral rotation, hinging about the shoulder to expose the damaged muscles. Primary repair of the muscles is performed by suturing, either repairing damaged muscles or fixing them to the scapula via bone tunnels drilled into the scapular body. Following repositioning of the scapula into its normal location, a wire or thick suture is placed through holes drilled in the caudal or dorsal scapular body and around an adjacent rib. Care is taken to avoid causing pneumothorax by damaging lung tissue. Post-operative imaging should be acquired to confirm satisfactory implant positioning, but also to determine the extent of any pneumothorax and to facilitate treatment as necessary. Exercise is strictly restricted for 2 weeks, then gradually reintroduced over a further 4 weeks.

SHOULDER LUXATION:

(Bardet 1998, Bedford 1969, Carwardine & Langley-Hobbs 2019, Vaughan 1967).

Cause and Pathogenesis

Shoulder luxation results from trauma and is uncommon in the cat. The evidence base regarding shoulder luxation in the cat is almost exclusively theoretical or extrapolated from the canine literature. The humeral head is cranially, laterally or medially displaced from the glenoid cavity. The level of instability following reduction of the joint depends on the extent of trauma to stabilizing structures such as the medial and lateral glenohumeral ligaments, joint capsule and muscles including supraspinatus, infraspinatus, teres minor (laterally) and subscapularis (medially). Where the joint capsule, glenohumeral ligament and muscular injuries are medial the humeral head luxates medially, whereas when the injuries are lateral, the luxation is to the lateral side (this is most common).

Clinical Signs and Diagnosis

Clinical signs may include non-weight-bearing lameness and resentment to examination of the shoulder. Palpation of a prominent acromion process may be diagnostic of medial shoulder luxation while absence of a palpable acromion process may indicate lateral shoulder luxation. Diagnosis of shoulder luxation is confirmed radiographically (Figure 9.2). Concurrent scapular and humeral fractures are possible, as are brachial plexus or other nerve injuries.

Treatment and Prognosis

Closed Reduction

Closed reduction is attempted and may be successful for the treatment of shoulder luxation in cats. The joint is immobilized by external coaptation for 2–3 weeks, and the cat's activity is restricted for 4–6 weeks. For medial

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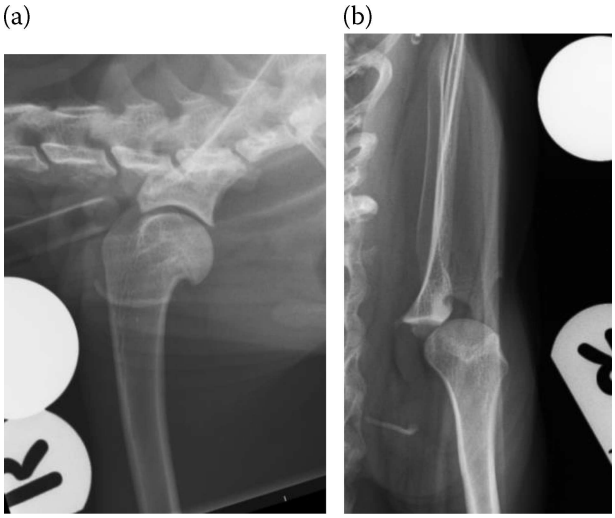


Figure 9.2 Mediolateral **(a)** and caudocranial **(b)** radiographic projections of a cat's shoulder demonstrating lateral luxation of the shoulder. (Note that the mediolateral view does not appear particularly abnormal).

shoulder luxation, immobilization involves a Velpeau sling. The sling distracts the humeral head laterally to maintain joint reduction. For lateral shoulder luxation, the limb is immobilized in a Spica splint. The humerus is positioned in slight abduction within the splint to maintain reduction of the shoulder.

Surgical Repair (Figure 9.2)

(Bardet 1998, Carwardine & Langley-Hobbs 2019, Fitch et al. 2001, Postredny et al. 1993, Puglisi 1986, Ringwood et al. 2001, Vasseur 1983).

Surgical repair is indicated if the joint is markedly unstable after reduction, or remains unstable after external coaptation, or if external coaptation is poorly tolerated. Surgery should always involve suturing of the joint capsule and this repair should be augmented through temporary trans-articular pinning or prosthetic glenohumeral ligament replacement.

Repair/Imbrication of the Joint Capsule

Torn joint capsule is sutured where possible with monofilament, absorbable suture material in a simple interrupted or continuous mattress pattern. Where possible, imbrication of the joint capsule by overlapping the torn edges may improve the holding power of the sutures. The glenohumeral ligaments are in essence linear thickenings of joint capsule, so no attempt to identify these individual structures is made. Repair/imbrication of joint capsule should typically be combined with another method of fixation.

Transarticular Pinning

Following reduction and repair of the joint capsule, the shoulder joint is temporarily immobilized at a weight-bearing angle of approximately 110° using a small Steinmann pin. The pin is inserted normograde from the distal aspect of the greater tubercle (through a separate stab incision), across the joint and into the scapular neck. The pin is cut short to minimize skin impingement. A Spica splint may be applied to augment the immobilization and minimize trauma to the articular surface. Exercise is strictly restricted while the splint and pin remain in place for 3 weeks. Moderate activity restriction should be maintained for a further 3 weeks following re-mobilization. Transarticular pinning is relatively simple but should be avoided where possible due to the trauma caused to the articular surface.

Prosthetic Glenohumeral Ligament Placement (Figure 9.3)

(Carwardine & Langley-Hobbs 2019, Fitch et al. 2001, Ringwood et al. 2001).

Techniques employing bone tunnels for fixation of sutures (3.5 M or 4 M polydioxanone, synthetic multi-filament or nylon may be considered) have been described:

1. Single suture loop. In two canine studies, one treating shoulder luxation in 10 dogs and the other an ex vivo study, bone tunnels were drilled from lateral to medial through the centre of the scapular neck (protecting the suprascapular nerve) and the proximal humerus, between the humeral head and greater tubercle. Suture material was passed laterally to medially through the hole in the scapular neck and medially to laterally through the hole in the proximal humerus. The suture was tied under moderate tension on the lateral aspect.
2. Double suture loop. In a study performed in cadaveric feline shoulders medial, lateral and bilateral (i.e. medial and lateral) instabilities were induced by sectioning various supporting soft tissue structures and treated using a technique involving a single central medial-to-lateral bone tunnel in the proximal humerus and two parallel lateral-to-medial bone tunnels in the scapular neck (the caudal tunnel originating 5 mm cranial to the caudal-most aspect of the glenoid and 5 mm proximal to the distal aspect of the glenoid and the cranial tunnel originating 5 mm caudal to the cranial-most aspect of the glenoid and 5 mm proximal to the distal aspect of the glenoid).

Techniques employing bone tunnels encircling the humeral head and scapular neck appear to be able to address medial, lateral and bilateral luxation in dogs and cats. Whether two-bone tunnels in the scapular neck are superior to a single-bone tunnel in the scapular neck remains to be determined. In the absence of evidence, the authors would opt for a single central bone tunnel within the scapular neck, to simplify the procedure and minimize the risk of trauma to the suprascapular nerve.

An alternative technique for replacement of the lateral glenohumeral ligament involves use of suture anchors rather than bone tunnels to secure suture material connecting two points: the scapular neck at the origin of the glenohumeral ligament and the humeral head just distal to the insertion of the glenohumeral ligament (Figure 9.3a & b).

Post-Operative Care

Post-operatively, the limb may be supported in a Velpeau sling for 2 weeks, though there is no evidence that this improves outcome. If irreparable articular fractures are present, or instability persists after repair, excision arthroplasty or arthrodesis of the scapulohumeral joint may be required.

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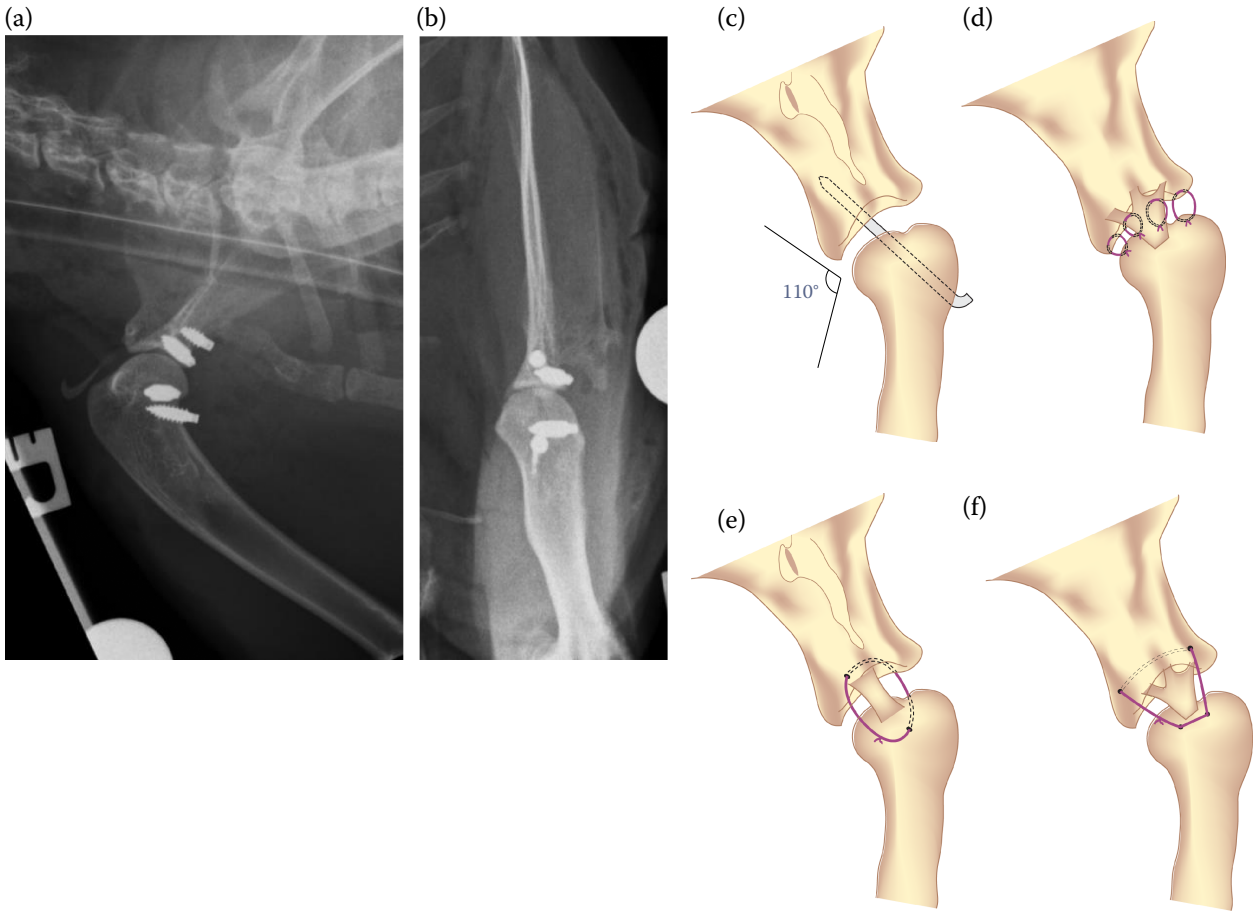


Figure 9.3 Techniques for stabilizing shoulder joint luxation. (**a** and **b**) Mediolateral and caudocranial radiographic postoperative views of a shoulder following fixation of lateral luxation with small bone anchors in the scapular neck and humeral head for securing prosthetic collateral support. **(c)** Transarticular pinning. The joint is placed at an angle of 110° prior to pin placement. **(d)** Imbrication of the joint capsule and glenohumeral ligament with monofilament absorbable suture material placed in an interrupted mattress pattern (medial view). **(e)** Collateral support. Suture material is placed through holes drilled in the scapular neck and proximal humerus and tied on the lateral side. **(f)** Prosthetic medial glenohumeral ligament replacement (medial view). Suture material is attached to the proximal humerus just distal to the insertion of the glenohumeral ligament using a bone anchor or bone tunnel. Bone tunnels are drilled through the scapular neck at the cranial and caudal origins of the glenohumeral ligament. The suture is passed from medial to lateral through the caudal bone tunnel, beneath the infraspinatus and supraspinatus muscles, and then from lateral to medial through the cranial bone tunnel. The suture is tied medially.

EXCISION ARTHROPLASTY OF THE SHOULDER (FIGURE 9.4):

(Franczuski & Parkes 1988).

Excision arthroplasty is a salvage procedure for the shoulder which preserves the shoulder range of motion. It may be somewhat easier to perform and less prone to complications than shoulder arthrodesis. However, outcomes for this procedure are poorly reported in the dogs, and not at all in cats.

Surgery involves a craniolateral approach employing tenotomies of:

1. The infrapinatus tendon of insertion
2. Origin of the acromial head of the deltoid muscle (an acromial osteotomy is an unreported alternative for this muscle)
3. Origin of the biceps brachii on the supraglenoid tubercle.

The joint capsule is incised transversely close to the glenoid rim. The suprascapular nerve and caudal circumflex humeral artery are preserved. An ostectomy of the glenoid is performed, using an oscillating saw or osteotome at an angle such that the lateral edge is slightly longer than the medial edge. If desired, the proximal portion of the humeral head can also be removed with a saw or osteotome, though whether this improves outcome is not known. Soft tissue closure involves suturing of the infraspinatus tendon of insertion. Teres minor may be used to close dead space by suturing to the biceps tendon and medial joint capsule. The acromial head of the deltoid muscle is reattached to the scapular spine using standard tendon suturing techniques (or if an acromial osteotomy was performed the bone fragment may be reattached using pins and tension band wire technique). Early use of the limb is encouraged after surgery to promote pseudoarthrosis.

SHOULDER ARTHRODESIS:

(Fowler et al. 1987, McLaughlin 1998, Vasseur 1990).

Shoulder arthrodesis is a salvage procedure for the shoulder in which the joint range of motion is not preserved.

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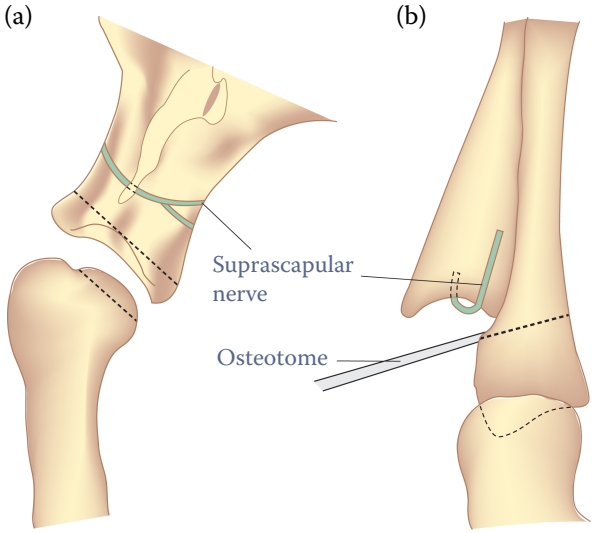


Figure 9.4 Excision arthroplasty of the shoulder joint. **(a)** Ostectomy sites for excision of the glenoid and proximal humerus. **(b)** The proper angle for ostectomy of the glenoid. The lateral edge should be slightly longer than the medial edge. The suprascapular nerve and caudal circumflex humeral artery are preserved.

Following shoulder arthrodesis the scapula moves through a greater excursion across the thoracic wall, restoring what appears to be near-normal limb function. The angle of fusion should allow advancement of the limb and weight bearing without overextension of the elbow. Arthrodesis of the shoulder joint in cats is usually performed at an angle of 110°; however, the correct angle is best determined by evaluating the cat preoperatively in a normal standing position.

Surgery involves a combined cranial and craniolateral approach, including:

1. Ostectomy of the acromion process (elevating the acromial head of deltoid muscle)
2. Ostectomy of the greater tubercle (elevating supraspinatus)
3. Tenotomy of the biceps brachii, infraspinatus and deltoideus muscles.

The suprascapular nerve is protected. The articular surfaces of the humeral head and glenoid are removed with a power burr or an oscillating saw at the correct angles and avoiding lateral or medial angulation of the ostectomies (Figure 9.5). The distal scapula and proximal humerus are apposed and temporary fixation with Kirschner wires is applied. Autogenous cancellous bone graft, collected from the ostectomized humeral head and glenoid, is placed around the ostectomy site.

Permanent fixation of the arthrodesis may involve the following techniques:

1. *Lag screw fixation*: A lag screw may be placed to bridge the joint. The glide hole is drilled from the cranial aspect of the scapular neck to end at the ostectomized surface and the lag hole continues across the ostectomy gap into the proximal humerus to engage the caudal humeral cortex. A 3.5 mm cortical screw or a 4.0 mm cancellous screw may be placed in most cats.
2. *Cross-pins and tension band wire fixation*: Two or three Kirschner wires are inserted as cross-pins to bridge the joint space. Orthopaedic wire (0.8 mm) is placed in a figure-of-eight pattern through holes drilled in the cranial border of the scapular neck and greater tubercle. Tightening the wire acts to compress the arthrodesis gap.
3. *Bone plating* (Figure 9.6): Plates are applied to the cranial or lateral aspect of the proximal humerus and the cranial or caudal junction of the spine and body of the scapula (with screws placed obliquely to maximize bone purchase). If possible one or two of the screws may be placed in lag fashion through the plate and across the joint. Because this is the technique most likely to afford rigid and durable fixation, the use of plate and screw fixation should be considered the technique of choice for shoulder arthrodesis.

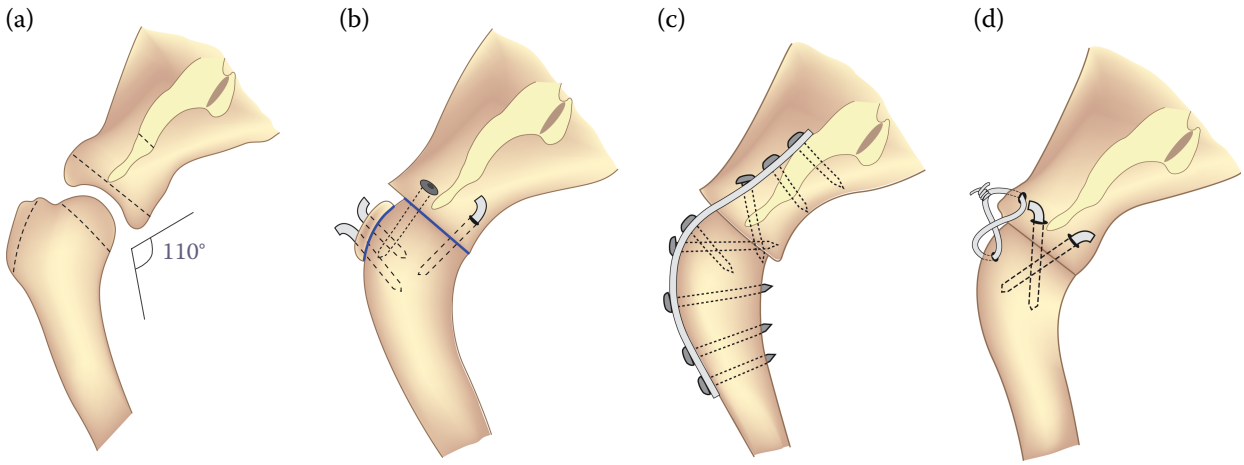


Figure 9.5 Techniques for shoulder arthrodesis. **(a)** The proper ostectomy sites on the distal scapula and proximal humerus. The arthrodesis angle is approximately 110° in cats. Osteotomies of the greater tubercle and acromion process are shown (dotted lines). **(b)** Shoulder arthrodesis using a lag screw and a Kirschner wire. The ostectomized greater tubercle is reattached with Kirschner wires or a small screw. **(c)** Shoulder arthrodesis by plate fixation. Two of the plate screws are placed in lag fashion across the ostectomy site. **(d)** Shoulder arthrodesis using cross-pins and a tension band wire fixation. The tension band wire is placed through bone tunnels on the cranial surfaces of the scapula and humerus.

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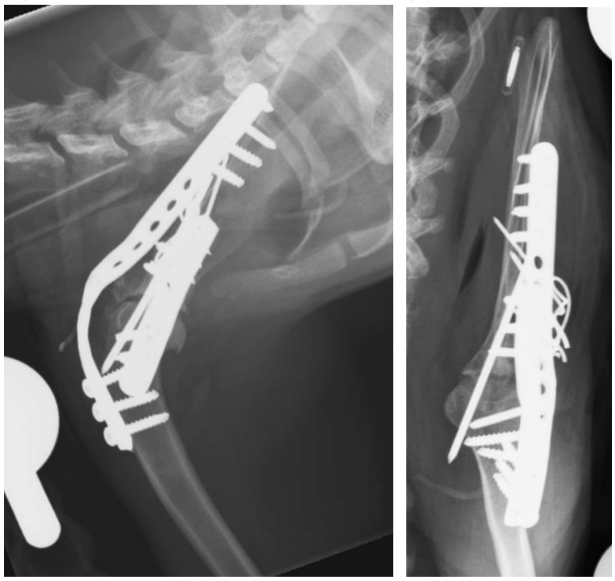


Figure 9.6 Mediolateral **(a)** and caudocranial **(b)** radiographic projections following shoulder arthrodesis to treat chronic instability and osteoarthritis. Articular cartilage was removed by power reaming, leaving rounded surfaces on the glenoid and humeral head. Access to the joint included an osteotomy of the acromion to elevate the acromial head of the deltoid muscle, therefore the implants present include a pin and tension band wire repair of this osteotomized segment of bone (apparent in figure b). Fixation of the arthrodesis itself involves a trans-articular Kirschner wire and two plates- one cranial on the humerus and craniolateral on the scapular spine, the other caudal to the scapular spine with fixation on the lateral aspect of the humerus.

Closure involves reattachment of the ostectomized greater tubercle (Kirshner wires plus a distal tension band wire to counter the pull from supraspinatus, or a small lag screw). The ostectomized acromion process is reattached with Kirschner wires and a tension band wire to counter the pull from the acromial head of deltoid). A Spica splint may be applied for up to 6 weeks following surgery (not vital if fixation is satisfactory) and exercise should be restricted until union has occurred.

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Part 2 Humerus and Elbow Joint

MEDIAL HUMERAL EPICONDYLITIS:

(Streubel et al. 2012, Streubel et al. 2015).

Mineralization within the origin of the flexor carpi ulnaris muscle distal to the medial epicondyle has been reported in dogs and in cats. In dogs it has been termed 'flexor enthesopathy' and may be present in elbows without radiographic evidence of elbow osteoarthritis (in which case it is termed 'primary flexor enthesopathy', and may be a cause of lameness) or in dogs with elbow osteoarthritis (in which case the term is 'concurrent flexor enthesopathy' and the alternative causes of lameness merit investigation). In cats similar enthesopathy of the insertion of the humeral head of the flexor carpi ulnaris muscle has been termed 'medial

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humeral epicondylitis' The two conditions have similarities, including the radiographic appearance, but their aetiologies have not been fully elucidated, so it is not correct to call them analogous.

This is a common disease. In a cadaveric study 6 of 60 adult European shorthair cats had radiographic evidence of bilateral mineralization, shown by histopathology to be within the humeral head of the flexor carpi ulnaris. These cats had reportedly shown no lameness. Among 17 cats demonstrating associated forelimb lameness, findings on clinical examination included resentment to pressure applied in the region of the mineralization and to pronation/supination with the elbow and carpus flexed. Compression of the ulnar nerve between the thickened tendon and the overlying epitrochleoanconeus muscle seems to be a cause of significant morbidity.

Treatment

Surgical intervention (Figure 9.7) involves a medial approach to the epicondyle and identification of the thickened tendon of origin of the humeral head of flexor carpi ulnaris. Abnormal tendinous material is debrided and neurolysis may be considered. Neurolysis may be external (release of the nerve from surrounding fibrous/fibrocartilaginous material) or internal (splitting of the epineurium to expose nerve fibres) depending on the individual case. The prognosis for dramatically reducing lameness with surgery is good, while the prognosis for conservative management is guarded. Ulnar neuropathy associated with medial humeral epicondylitis is also discussed in Chapter 17.

ELBOW OSTEOARTHRITIS:

(Freire et al. 2014, Freire-Gonzales et al. 2014, Perry 2017, Staiger & Beale 2005).

Intra-articular osteochondral fragments may be found in the elbow joints of adult cats in the presence of wear of the cartilage particularly within the medial compartment of the joint. This finding has been likened to medial coronoid process fragmentation seen as part of the elbow dysplasia complex in dogs, and arthroscopic treatment for debridement of intra-articular osteochondral fragments has been reported to carry a good prognosis for cats with 2-year follow-up.

However, more recently it has been recognized that osteochondral fragments may be present within the feline elbow in the absence of any evidence for fragmentation of the medial coronoid process or for osteochondrosis. In these cats there is significant erosion of articular cartilage in the medial compartment, while the articular cartilage of the lateral humeroradial articulation remains healthy. Such medial compartment disease is common in adult cats. In a cadaveric study the finding of elbow osteoarthritis (visible cartilage erosion) was present in 73% of adult cats (39/60 joints) and free osteochondral fragments were present in 10 of the 39 joints. The free osteochondral fragments were found to be consistent with osteochondromatosis. Therefore, elbow osteoarthritis in cats should be considered a specific entity unlike elbow dysplasia in dogs. It is possible that osteochondromatosis could be a significant driver of feline osteoarthritis throughout the body; however, at this stage this is not clear.

Treatment

Treatment to address the presence of multiple osteochondral fragments within an elbow joint may involve arthrotomy or arthroscopy. With synovial osteochondromatosis osteochondral fragments may be free-floating or attached to the synovial membrane. This has been reported in a single case report of one cat affected with bilateral osteochondromatosis and additional medial epicondylitis. Greater numbers of cats with this condition treated by fragment removal have not been reported as yet, and conservative management remains a viable option.

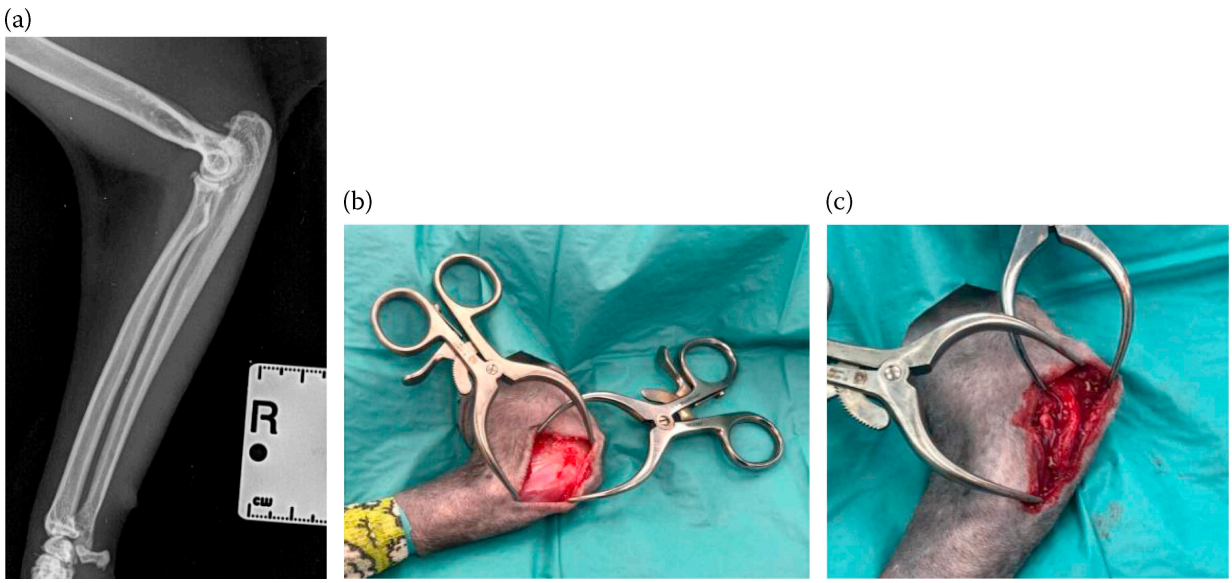


Figure 9.7 Medial humeral epicondylitis. Radiography confirms the presence of peri-articular new bone formation affecting the elbow and in the region of the medial epicondyle **(a)**. Surgery is performed via a short approach to the medial aspect of the elbow **(b)**. The mineralized and thickened humeral head of the flexor carpi ulnaris muscle is identified deep to the ulnar nerve. It is transected and the abnormal material removed. It is not possible to reattach the shortened muscle back to the epicondyle, so it is sutured to fascia adjacent to the ulnar head of the same muscle. The ulnar nerve moves freely with no adhesions to the underlying tissues at the end of the surgery **(c)**.

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RADIAL HEAD LUXATION:

Luxation of the radial head is rare in cats. It is associated with traumatic rupture of the annular ligament. The diagnosis is confirmed by radiography. Treatment for radial head luxation is open reduction and internal stabilization. Suturing of the torn annular ligament and lateral collateral ligament is complicated by the shear size of these structures. Augmentation using prosthetic sutures may be considered. Once reduced, the radius is attached to the ulna with a 2.0 mm positional screw (placed from the caudal aspect of the ulna) or a suture/toggle system. A screw will impede pronation and supination, may loosen/migrate and will require removal after 4–6 weeks to restore normal range of motion. A suture/toggle system will preserve some pronation and supination and will inevitably stretch or snap, thus may not necessitate removal.

TRAUMATIC ELBOW LUXATION:

(Farrell et al. 2007, Mitchell 2011, Schrader 1994).

Cause and Pathogenesis

Elbow luxation is generally a result of significant trauma. Lateral luxation is most common (the radioulnar joint may remain intact, and is located lateral to the distal humerus). The medial epicondyle is larger than the lateral epicondyle and the size and shape of the medial epicondyle prevents medial movement of the ulna. Caudal luxation also occurs in cats. One study suggested that luxation of the cadaveric feline elbow was only possible when both the medial and lateral collateral ligaments were sectioned (grade 3 ligament injury), however, stretching of ligaments (i.e. grades 1 or 2 ligament injury) is possible without complete loss of integrity and may be sufficient to allow luxation.

Clinical Signs

Cats with elbow luxation may have a history of recent trauma. Most are acutely non-weight-bearing and hold the limb with the elbow slightly flexed and adducted. The antebrachium is abducted and externally rotated. Palpation of the elbow region reveals a bony mass on the lateral aspect of the distal humerus. Manipulation reveals reduced range of motion and pain.

Diagnosis

Lateral and craniocaudal radiographic views of the elbow are obtained to confirm the presence of a luxation (Figure 9.8), determine the direction of the luxation and to evaluate for additional fractures of the humerus, radius or ulna (which may include small intra-articular or juxta-articular fractures of the articular surface or avulsion fragments).

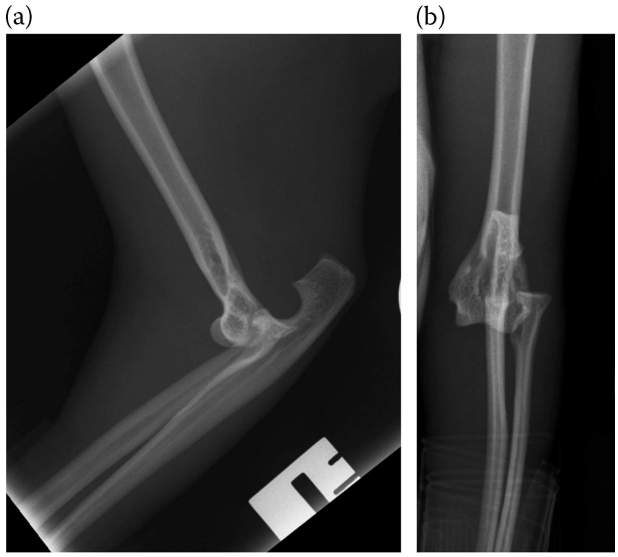


Figure 9.8 Radiographic views of an elbow luxation **(a)** mediolateral view and **(b)** craniocaudal view. Luxation is lateral in this case.

Treatment and Prognosis

Elbow Reduction

In acute injuries, closed reduction is attempted first and is usually successful in cats. In fact in one study of nine cats with elbow luxation (all lateral), closed reduction was reportedly successful in all cases. General anaesthesia is recommended. Partially suspending the cat by the limb for 5–10 minutes may fatigue the muscles and aid in reduction of the elbow.

Lateral luxation (Figure 9.9)

Flex the elbow and pronate and abduct the antebrachium (with the aim of directing the anconeal process into the olecranon fossa). Apply medial pressure to the radial head and proximal ulna. Extension of the elbow should engage the anconeal process fully such that the anconeal process acts as fulcrum to lever the radial head into position. The joint should now be reduced. Flexion and extension are performed to release trapped shreds of torn collateral ligament/joint capsule. If external coaptation is to be employed, then maintaining the elbow in extension should prevent re-luxation by keeping the anconeal process within the olecranon fossa. External coaptation (Spica splint) is applied for 2–3 weeks.

Caudal Luxation

Suspension by the affected limb may be sufficient to reduce the elbow in the case of a caudal luxation. Otherwise, further traction is applied to the antebrachium until the humeral condyle is aligned with the ulnar notch. Flexion of the elbow should then seat the condyle fully. Maintaining moderate flexion of the elbow should theoretically prevent re-luxation. If external coaptation is to be employed, then maintaining the elbow in moderate flexion is achieved through placement of a Velpeau sling. External coaptation is maintained for 2–3 weeks.

Surgical Management of Elbow Luxation

After reduction of the luxation, the stability of the joint is assessed. The collateral ligaments are evaluated by flexing both the carpus and elbow to 90° (Campbell's test). The foot is then internally and externally rotated and the amount of rotation compared with the contralateral normal limb (Table 9.1).

Joints that are reasonably stable after closed reduction are managed with external coaptation. Joints that cannot be reduced closed, easily reluxate after closed reduction or have a rotational range of motion within the ranges indicating complete rupture of one or both collateral ligaments (see table) should be considered candidates for surgery, since they are at risk of re-luxation with conservative treatment.

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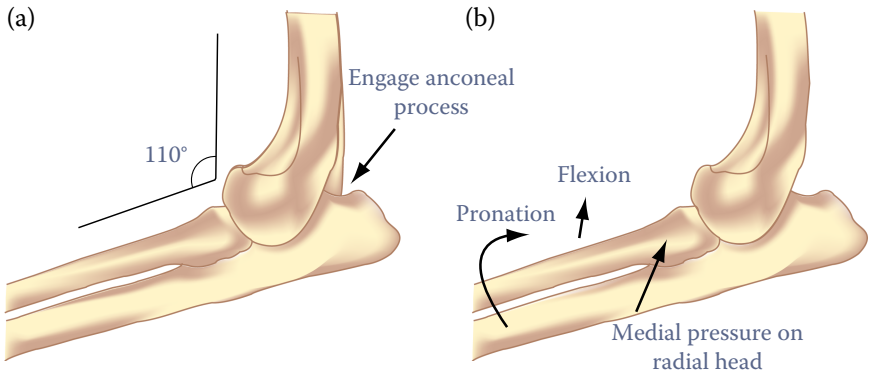


Figure 9.9 Technique for closed reduction of lateral elbow luxation. **(a)** the elbow is extended somewhat (to approximately 110 degrees) while valgus force is applied to direct the anconeal process into the supratrochlear foramen. **(b)** with the anconeal process engaged in the supratrochlear foramen the joint is flexed while the antebrachium is pronated and valgus force is maintained. Medial pressure is applied to the laterally-located radial head.

Table 9.1: Ranges of Rotational Motion from a Neutral Resting Position in Cadaveric Feline Elbows with the Elbow and Carpus Flexed to 90°

	Normal ROM in Flexion and Extension	Which Collateral Transected?	ROM with Collateral Transected
Internal rotation/ pronation	30–70	Medial	80–110
External rotation/ supination	110–150	Lateral	160–180

Note: Comparison is made between the ranges of motion in the intact elbow and following transection of collateral ligaments (note that medial collateral sectioning affects pronation, while lateral collateral sectioning affects supination).
Source: Adapted from Farrell et al. 2007.

Internal Fixation

A number of techniques for open reduction and fixation of traumatic elbow luxation in cats have been reported, unfortunately, each with only a small number of cases. The joint is typically approached through a lateral incision with access to the joint caudal to extensor carpi radialis or the common digital extensor tendon of origin. Haematoma, torn remnants of the joint capsule and muscle are removed from the joint and the articular surfaces examined. The elbow is reduced as described for the closed method. An olecranon ostectomy (followed by reattachment using a tension band fixation) has been suggested to facilitate reduction but is rarely required and carries a relatively high risk of complications post-op.

Soft Tissue Reconstruction

Following reduction of the joint, torn joint capsule and collateral ligaments are reconstructed where they are identifiable. Whilst it is often difficult to identify the individual structures, this reconstruction should be attempted, since the feline elbow joint collateral ligaments have specific roles and it is not certain that peri-articular fibrosis alone can restore satisfactory joint function. Following soft tissue reconstruction augmentation of the repair is performed either by placing prosthetic ligaments or by temporary joint immobilization.

Prosthetic Ligaments

With this technique an approximation of the normal function of the collateral ligaments is made with suture material.

Bone anchors or screws and washers

Appropriately sized bone anchors may be placed to affix the suture in the proper anatomic location. Alternatively, 1.5 mm or 2.0 mm screws may be placed + - washers and the suture material placed to encircle the screw heads. Nonabsorbable suture material (2–0) in a figure-of-eight pattern is used.

Circumferential suture prosthesis

(Farrell et al. 2007, Farrell et al. 2009, Perry 2017).

A technique was devised in 2007 to replace the radio-humeral and ulnaro-humeral components of the medial and lateral collateral ligaments in a single manoeuvre. A lateral approach is made to the elbow and 2.0 mm bone tunnels are made at three sites:

1. Transcondylar at the origins of the collateral ligaments immediately craniodistal to the epicondyles.
2. Transradial at the radial head insertion of the lateral collateral ligament.
3. Transulnar at the middle of the trochlear notch.

A double loop of suture (the study recommended 4 m polydioxanone) is threaded onto a straight needle which is passed lateral to medial through the transcondylar tunnel. A stab incision is made medially to retrieve the suture material and needle. The needle is then passed eye-first lateral to medial through the radial bone tunnel to retrieve both suture ends. Both strands are pulled back through the radial head bone tunnel and a self-locking knot tied laterally (the humero-radial components of both collateral ligaments are now augmented or replaced with a double loop of polydioxanone). The process is repeated for the transcondylar and ulnar bone tunnels; however, only a single strand is required (the humero-ulnar components of the collaterals are now augmented or replaced with a single loop of polydioxanone).

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The circumferential suture prosthesis technique has also been reported for stabilization of humero-radial subluxation and instability of iatrogenic origin following a medial elbow arthrotomy and excision of mineral bodies in the joint and within flexor carpi ulnaris (synovial osteochondromatosis and medial epicondylitis, respectively).

Temporary Maintenance of Joint Reduction

By maintaining joint reduction for 2–3 weeks these techniques allow healing of the repaired ligaments and periarticular fibrosis, such that once implants are removed after a few weeks the joint remains stable. Note that these techniques may be performed using minimally invasive technique following closed reduction in cases in which there is questionable post-reduction stability.

Transarticular pin

A transarticular Kirschner wire (1.1–1.6 mm) may be inserted to temporarily immobilize the elbow joint at a physiological standing angle (110°–120°). Kirschner wires are inserted either from the caudal aspect of the ulna into the humeral condyle, or from the humeral condyle (caudo-cranially) into the radial head. The Kirschner wire is cut but left long enough to ease retrieval after 2 weeks. A Spica splint should also be placed to protect the Kirschner wire from migrating during attempts to move the elbow. Activity is restricted for a further 4 weeks following removal of the splint and pin.

Trans-articular external skeletal fixator (Figure 9.10)

Hazardous corridors may be avoided by placing fixation pins in the lateral aspect of the humerus proximally and distally (an understanding of the location of the radial nerve and the supracondylar foramen on the medial aspect of the humerus is important when choosing the location of these fixation pins) and on the craniolateral aspect of the radius. The frame remains in position for 2–4 weeks (Figure 9.10).

Elastic trans-articular external fixator construct

(Schwartz & Griffon 2008, Vedrine 2017).

A relatively simple external frame has been described in dogs and cats for placement following closed reduction of a luxated elbow. The frame involves two 2.0 mm pins placed perpendicular to the long axis of the humerus, one passing from medial to lateral in the distal quarter of the humerus and one passing from medial to lateral (parallel to the first) in the centre of the olecranon, toward its proximal aspect. Tight elastic bands are placed connecting the bars on the medial and lateral aspects of the joint. This frame maintains the joint in extension (reducing the likelihood of re-luxation) but still allows some range of motion of the joint. Excellent outcomes have been reported in the only two cats in which this technique has been reported.

Post-Operative Management

Regardless of the technique employed, the limb will often be immobilized in a Spica splint for 2 weeks after surgery. The cat's activity is restricted for 4 weeks after splint removal. Passive range-of-motion exercises are used to restore full mobility to the joint.

Prognosis

The prognosis after elbow luxation depends on the severity of cartilage and periarticular soft tissue injuries, and the accuracy with which the collateral ligaments in this species heal, to restore normal elbow function. The prognosis with elbow luxation is good to excellent if treated early and physical therapy is begun soon after reduction. Potential complications include re-luxation, reduced range of motion, osteoarthritis and infection. In cases of severe articular cartilage and periarticular soft tissue injury, elbow arthrodesis may be considered.

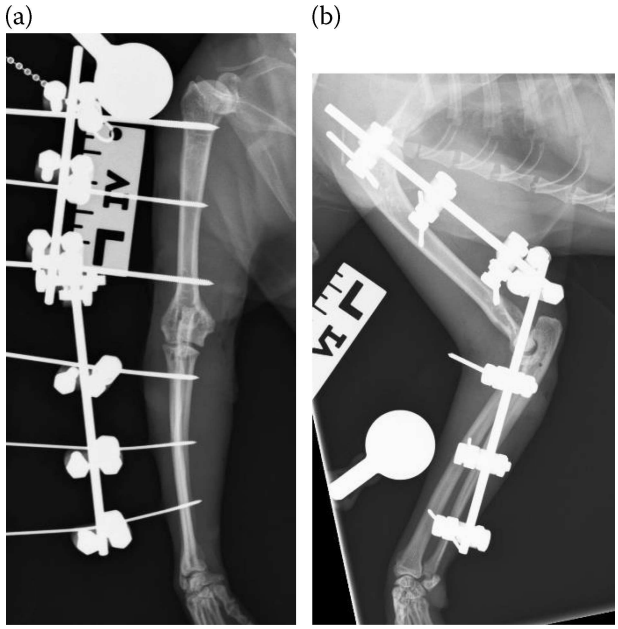


Figure 9.10 Craniocaudal **(a)** and mediolateral **(b)** radiographic projections of a feline forelimb demonstrating transarticular external skeletal fixation applied to maintain reduction of a luxated elbow.

CONGENITAL ELBOW LUXATION:

(Valastro et al. 2005).

Congenital elbow luxation is relatively rare in dogs and may be associated with a disparity in the growth rates of the radius and ulna. In dogs congenital elbow luxations are of three types: type 1 or lateral (sub)luxation of the radial head from the humero-radial joint, type 2 or rotational (sub)luxation of the ulna from the humero-ulnar joint and type 3, a combination of the radial and ulnar (sub)luxations. A single case report of bilateral type 1 congenital elbow luxation exists in the veterinary literature. Treatment may be conservative provided clinical function is acceptable. Surgical options have been reported for dogs with congenital elbow luxation, often in small numbers and with limited outcome data. Radial head excision may be a reasonable option, with loading transmitted to the often hypertrophied coronoid processes.

ELBOW ARTHRODESIS:

(Moak et al. 2000).

Arthrodesis is considered for cases of severely comminuted intra-articular fractures, chronic luxation unresponsive to surgical stabilization and severe degenerative joint disease. Anecdotally, limb function following elbow arthrodesis is unpredictable. Whilst successful arthrodesis should abolish pain associated with the joint, the limb is circumducted during the swing phase of each stride and in some cases placing of the paw may be only intermittent.

The elbow is usually fused at 110°–120°, but the angle should be individualized for each cat based on preoperative evaluation of the standing angle of the contralateral limb.

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The joint is exposed through a lateral approach, which may be combined with a caudal approach including ostectomy of the olecranon process (though this is commonly not necessary). Transection of the ulnaris lateralis, lateral collateral ligaments and the joint capsule allows removal of the articular cartilage from the radial head, humeral condyle and the trochlear notch of the ulna using bone curettes or a power burr. The joint space is filled with autogenous cancellous bone graft or demineralized bone matrix to promote fusion. Rigid fixation of the elbow for arthrodesis is achieved with one of three techniques:

Lag screw fixation

Lag screws may be inserted from the lateral epicondyle into the radial head, from the olecranon process into the condyle and from the ulna to the medial epicondyle. An additional screw can be inserted from the ulna to the humerus if sufficient room exists. Stability is unlikely to be sufficient to achieve arthrodesis using this technique and it is not recommended.

Plate fixation (the authors' preference)

A bone plate and screws are applied to the caudal aspects (tension surface) of the distal humerus and proximal ulna after ostectomy of the olecranon process, which is fixed back in position using a pin and tension band technique. An additional plate may be applied to the lateral aspects of the humerus and onto the radius (this necessitates a plate contourable in two planes). Where possible, screws may be placed in lag fashion through the plate to achieve compression of the joint space. Separate lag screws may also be placed outside the plate.

Transarticular external fixator

It is possible to insert a Kirschner wire (1.6–2.0 mm) from the caudal aspect of the ulna into the humeral medullary cavity and drive it proximally to exit at the greater tubercle. Both ends of this Kirschner wire are left protruding from the skin. Transfixation half pins are inserted into the radius, ulna and humerus. Flexible tubing is placed to connect the exposed ends of the various pins and filled with acrylic. The connecting bar and pins are removed when fusion is complete.

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Part 3 Antebrachium and Carpus

RADIAL HEMIMELIA:

(Bezhentseva et al. 2018, Lockwood et al. 2009, Pisoni et al. 2012, Swalley & Swalley (1978), Towle & Breur 2004, Winterbotham et al. 1985).

Hemimelia refers to an absence of one or more long bones. It may be transverse (all bones across the limb's width are absent) or longitudinal in which all bones along the medial (preaxial) or lateral (postaxial) aspect of the limb are absent.

Radial hemimelia (also called radial agenesis) is one of the most common dysostoses in the cat. It is characterized by the absence or severe hypoplasia of the radius and variable absence of the medial carpal bones and medial metacarpal bones and digits (Figure 9.11). The aetiology for a partial failure of development of the limb bud in utero is uncertain, though various metabolic, toxic and genetic suggestions have been made. In one study, a second breeding of the parents of kittens with hemimelia produced another litter with affected kittens.

Most kittens have an obvious limb deformity at birth, characterized by shortening and varus deformity at the level of the carpus. One or both forelimbs may be affected. Kittens are lame and have muscle atrophy, but the condition itself is not considered to be painful. Additional malformations including polydactyly, abnormal numbers of ribs and vertebrae and cardiomegaly have been reported in cats with radial hemimelia.

Some kittens will use affected limbs to ambulate. Long-term splinting, amputation and euthanasia are options. One case of surgical management exists in the literature in which fusion of the abnormal antebrachiocarpal joints was performed bilaterally (staged 6 weeks apart). The hypertrophic ulnae underwent distal ostectomy and following debridement of articular cartilage in the remaining carpal joints, fixation was with 2.0 mm locking compression plates applied cranially to the ulnae and dorsally to the central one of three digits. Depending on the conformation of the deformity, such an approach is a reasonable option; however, the potential for discomfort

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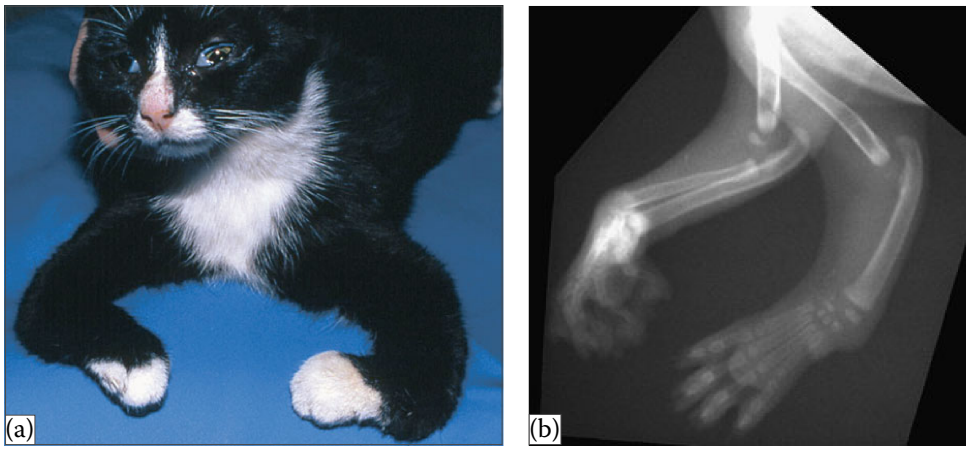


Figure 9.11 **(a)** Cat with radial hemimelia. **(b)** Radiograph of forelimbs from a cat with radial hemimelia.

associated with degenerative joint disease affecting the elbow remains in the medium to long term.

Neutering is advised because of the potential genetic component of the disease.

CARPAL JOINT INJURIES:

(Basa & Johnson 2019, Greef et al. 2014, Nakladal et al. 2013, Voss et al. 2003).

The seven carpal bones are arranged in two rows to form a composite joint. The radial and ulnar carpal bones articulate with the distal radius and styloid process of the ulna to form the **antebrachiocarpal joint**. The accessory carpal bone articulates with the palmar surface of the ulnar carpal bone and serves as the insertion point for the flexor carpi ulnaris muscle. The numbered carpal bones (I through IV) articulate proximally with the radial and ulnar carpal bones to form the **middle carpal joint**, and distally with the metacarpal bones to form the **carpometacarpal joint**. The spaces between each of the carpal bones on a given level are called intercarpal joints. Numerous ligaments maintain stability yet permit joint motion. The joint capsule, palmar carpal fibrocartilage and medial and lateral collateral ligaments support the joint while bearing weight. Most movement in the sagittal plane is at the antebrachiocarpal joint, while the middle carpal joint has a small range of motion and the carpometacarpal joint has little or no movement.

Carpal joint injuries in the cat were analysed in an epidemiological study. Injuries were found secondary to falls in 72.6%. They occurred at all three joint levels including fracture/luxation at the antebrachiocarpal joint (50.8%), at the middle carpal joint (12.3%), at the carpometacarpal joint (27.7%), at both the antebrachiocarpal and middle carpal joints (7.7%) and at the ulnaro-accessory carpal joint in a single case (1.5%).

Swelling and joint instability may be palpated in the carpal region. Radiographs are indicated to identify fractures and joint luxations. Stress radiographs are often necessary to identify the level of instability and should include flexion and extension (hyperextension injuries are relatively common) and varus and valgus stress.

Hyperextension Injuries of the Carpus

Cause and Pathogenesis

Hyperextension injuries to the feline carpus are uncommon. The trauma causes tearing of the palmar carpal fibrocartilage and palmar ligaments. Injury to any or all of the antebrachiocarpal, middle carpal or carpometacarpal joints can occur.

Clinical Signs

Hyperextension of the carpus is usually palpable and the cat may walk with a palmigrade stance.

Diagnosis

Stress radiographs are used to exclude concurrent fractures and to identify the level of the hyperextension injury.

Treatment and Prognosis

Although this is not the case for the canine hyperextension injury, anecdotally, feline carpal hyperextension injuries may respond to conservative treatment. The limb is bandaged with the antebrachiocarpal and carpometacarpal joints in flexion for 4 weeks. The carpal flexion bandage prevents weight bearing and allows healing of palmar fibrocartilage. Following bandage removal, the cat's activity is restricted for an additional 4–6 weeks. Mild physical therapy exercises (including stretching and passive range-of-motion exercises) may be instituted to restore normal joint motion. If hyperextension recurs after bandage removal, or if the dressing cannot be maintained, pan or partial carpal arthrodesis may be indicated. Note that this is anecdotal, and it is imperative that clients understand arthrodesis may remain necessary following a period of unsuccessful conservative management.

ANTEBRACHIOCARPAL LUXATION/ RADIAL CARPAL BONE LUXATION:

(Miller et al. 1990, Pitcher 1996, Voss et al. 2003).

The terminology of antebrachiocarpal luxation is somewhat unclear. In cats the most common cause of antebrachiocarpal luxation is palmar luxation of the radial carpal bone. In dogs radial carpal bone luxation is a very rare occurrence, with the bone essentially swivelling between the distal radius and middle carpal bones such that the proximal articular surface faces palmar and medial. In cats the middle carpal joint remains intact, so radial carpal bone luxation takes the entire paw with it, to lie palmar to the distal radius (Figure 9.12).

Cause and Pathogenesis

Trauma to the medial (radial) collateral ligament may be the most common injury associated with antebrachiocarpal

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joint subluxation. The radial carpal bone (and with it the rest of the paw) luxates in a palmar direction (Figure 9.12). There is only one (oblique) medial collateral in the cat, with no short part being present. The medial collateral ligament originates on the cranio-medial aspect of the distal radius and courses obliquely (at 80° to the long axis of the radius) in a palmarodistal direction to insert on the radial carpal bone. Rupture of the medial collateral ligament results in medial joint instability and palmar dislocation of the radial carpal bone.

Diagnosis

Pain and crepitus may be elicited on palpation of the carpal region. Medial instability is palpable if rupture of the collateral ligament is complete. One study reports manipulations involving palmar luxation and dorsal reduction of the antebrachiocarpal joint similar to 'cranial drawer' in the cranial cruciate ligament deficient stifle.

Treatment and Prognosis

1. Closed reduction and fixation

The antebrachiocarpal joint may be reduced in a closed fashion. Reduction is achieved by first rotating the radial carpal bone (and paw) about its dorsopalmar axis and then about its mediolateral axis. It is then repositioned by applying digital pressure from the palmar aspect of the joint while extending the carpus. The limb is then supported in splinted external coaptation from the digits to the elbow. The dressing remains in place for 4–6 weeks. Alternatively, a small transarticular Kirschner wire can be placed percutaneously through the radial and ulnar carpal bones from medial to lateral to maintain reduction of the radial carpal bone. The wire may be removed after 4–6 weeks.



Figure 9.12 Antebrachiocarpal luxation and palmar displacement of the paw with respect to the radius. Note additional small fracture fragments potentially precluding restoration of joint function with replication of the medial collateral support.

2. Open reduction and fixation

A dorsal approach to the distal radius and carpus is performed. The medial collateral ligament is sutured if possible. Collateral ligament replacement involves drilling bone tunnels in the medial prominence of the radial carpal bone and distal radius through which suture is passed. Alternatively, bone anchors or 1.5 mm screws may be placed at the origin and insertion of the collateral ligament to attach the suture (Figure 9.13). The joint capsule is closed. A palmar splint dressing may be considered for a few weeks.

MIDDLE CARPAL AND CARPOMETACARPAL JOINT (SUB)LUXATION:

(Greef et al. 2014, Nakladal et al. 2013).

While luxation of the middle carpal joint is rare in cats, carpometacarpal luxation accounts for approximately one quarter of hyperextension injuries in cats. One case report describes dorsal luxation of the second carpal bone, allowing collapse of the dorsal surface of the carpus, which gives the appearance of a carpal hyperextension injury. The injury was managed by reduction of the second carpal bone and temporary immobilization of the collapsed middle carpal and carpo-metacarpal joints using intramedullary pins and a trans-articular external skeletal fixator.

Treatment and Prognosis

Closed reduction and immobilization using a palmar splint for 4 weeks with the carpus at 15°–20° of flexion should be considered. If the joint is markedly unstable after closed reduction, internal fixation is indicated. Internal fixation involves drilling bone tunnels in the medial prominence of the radial carpal bone and the medial base of metacarpal II through which suture is passed and tightened in a figure-of-

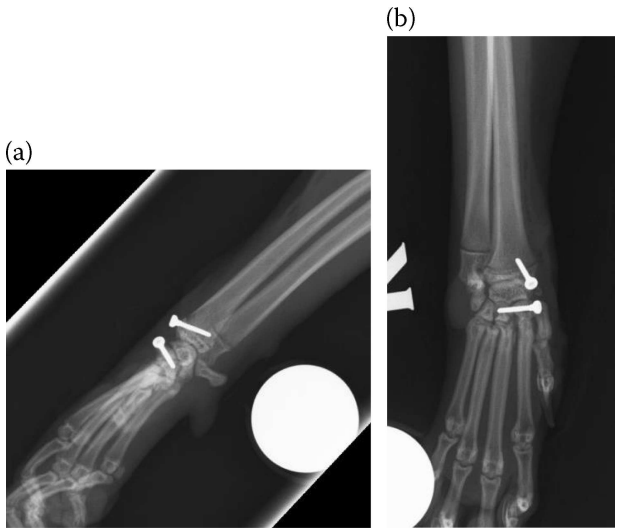


Figure 9.13 Open reduction and prosthetic medial collateral ligament placement is demonstrated in mediolateral **(a)** and dorsopalmar **(b)** radiographic projections of a feline carpus. A 2 mm screw has been placed in the distal radius and a 1.5 mm screw has been placed in the numbered carpal bones (typically the recommended location would be the radial carpal bone, however, there was a suspicion of laxity within the medial aspect of the middle carpal joint in addition to the antebrachiocarpal joint in this case). Suture is tied between the screws in a figure-of-eight pattern and tightened.

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eight pattern. Alternatively, bone anchors or 1.5 mm screws may be placed at the same sites to attach the suture. The joint capsule is closed. A palmar splint dressing may be considered for a few weeks. If pain and instability persist after closed or open stabilization, partial carpal arthrodesis is considered.

ACCESSORIO-ULNAR LUXATION:

(Harris et al. 2013).

A single case of a cat with luxation of the accessory-ulnar carpal joint has been reported. Lameness and resentment to carpal manipulation were present. Mild hyperextension may be apparent during loading of the limb as a result of failure of the flexor carpi ulnaris muscle which inserts on the accessory carpal bone. Treatment involved accessoioulnar arthrodesis. Arthrodesis of the joint between the accessory and ulnar carpal bones may be achieved through debridement of the joint followed by placement of a single Kirschner wire from the palmar surface of the accessory carpal bone, driven along its length, across the accessionulnar joint and into the ulnar carpal bone.

Carpal Shearing Injuries

(Beardsley & Schrader 1995, Corr 2009).

Shearing (degloving) injuries occur less frequently on the forelimb than on the hindlimb. Most occur when the cat is hit by a car.

Clinical Signs

Typically the medial aspect of the carpus and metacarpus is abraded by the pavement, removing variable amounts of skin, muscle, tendon, ligament and bone. One or more of the carpal joints may be open. Inevitably these injuries are heavily contaminated. See Chapter 11 for a more detailed description of the approach to the shearing injury.

Pancarpal Arthrodesis

(Calvo et al. 2009, Guerrero & Montavon 2005, Okin 1982).

Pancarpal arthrodesis involves fusion of the antebrachiocarpal, middle carpal and carpometacarpal joints. Indications include luxation, hyperextension, debilitating osteoarthritis of the antebrachiocarpal joint, shearing injuries with significant loss of articular cartilage and postural deformity resulting from radial nerve paralysis. Literature regarding pan carpal arthrodesis in cats is scarce. There is a concern that loss of pronation/supination may have a detrimental effect on quality of life in cats, however, although one retrospective study revealed a reduction in the jump-height and a reduced tolerance of exercise in cats with pan (and partial) carpal arthrodesis, quality of life following these procedures was still considered good.

A hanging-limb preparation is used. An Esmarch or similar tourniquet technique may be considered to limit haemorrhage, but is not imperative. A dorsal approach is made to identify the three joint levels. The articular cartilage is removed with a small bone curette or burr. The joint is reduced in a weight-bearing position (10°–12° of carpal extension). Fixation may involve various combinations of bone plates and intramedullary pins (Figure 9.14). The bone plate is applied on the cranial surface of the radius and on the dorsal surface of the radial carpal bone, and metacarpal III. Three screws are placed in the

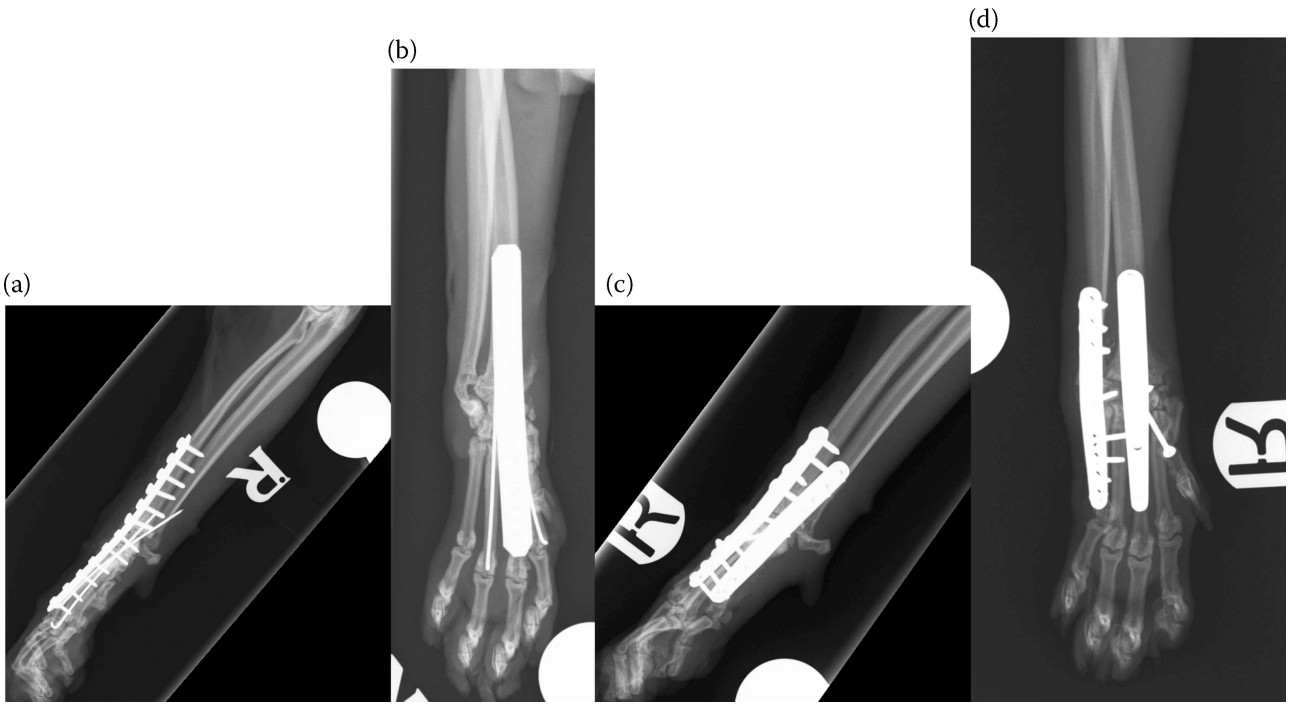


Figure 9.14 Pan carpal arthrodesis in cats has been described using various combinations of plates and pins. (a + b) Mediolateral and dorsopalmar projections of a carpus following arthrodesis using a plate-rod technique with a 2.0 mm veterinary cuttable plate on the cranial aspect of the radius and dorsal aspect of the radial carpal bone and third metacarpal and intramedullary Kirschner wires driven from the distal end of the second and fourth metacarpals to cross the carpus. (c + d) Mediolateral and dorsopalmar projections of a carpus following arthrodesis using an oblique lag screw to bridge the second metacarpal base and radial carpal bone, a 2.0 × 1.5 mm hybrid pan carpal arthrodesis plate on the cranial radius and dorsal radial carpal bone and third metacarpal bone and an additional lateral plate on the ulna extending onto the lateral aspect of the fifth metacarpal bone.

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radius, one in the radial carpal bone, and three in metacarpal III. Cuttable plates (1.5/2.0 mm, 7–8 holes) are sufficiently small for carpal arthrodesis in cats, accommodate several sizes of small screws, and can be cut to the desired length at the surgery table. Where possible, compression should be applied across the joint spaces. The feline metacarpal bones are narrow, and applying fixation to a single metacarpal may be considered somewhat tenuous. Medial plate application has been described for dogs and may be an appropriate technique in cats. Care is taken during insertion of screws to avoid fracturing the small metacarpal bones. Autogenous cancellous bone graft is placed in the joint spaces. Alternatively, fixation can involve a modified type II external skeletal fixator with freeform connecting bars instead of a plate and screws. However, this is not recommended, since premature pin loosening and fractures of the metacarpal bones can occur.

Partial Carpal Arthrodesis

(Haburjak et al. 2003).

Partial carpal arthrodesis involves fusion of the middle and carpometacarpal joints. Indications are as for pan carpal arthrodesis, but this procedure preserves a normal antebrachiocarpal joint. Fixation can be with a medial plate and screws, cross pins or intramedullary Kirschner wires driven from the heads of the metacarpals II and III (via dorsal slots) across the carpo-metacarpal and middle carpal joints. The latter technique is extremely difficult because of the small size of the metacarpal bones. With the cross-pin technique the medial Kirschner wire is placed from the medial-dorsal aspect of metacarpal II into the ulnar carpal bone and the lateral Kirschner wire is placed through the radial carpal bone and through the lateral-dorsal aspect of metacarpal V. The tip of the wire is countersunk below the articular surface of the radial carpal bone. Autogenous cancellous bone graft should be applied.

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Part 4 Metacarpal and Phalangeal Injuries

(De Rooster et al. 2007).

Luxation of the metacarpophalangeal/metatarsophalangeal and interphalangeal joints is uncommon in cats. Reduction and stabilization are the preferred options by collateral ligament replacement. However, particularly for digits two and five, conservative management, resulting in pseudoarthrosis formation, is an option. Salvage procedures may be considered if healing is incomplete and pain persists after 2–4 weeks of crate confinement +/- external coaptation. Salvage procedures following failed management of metatarsophalangeal or interphalangeal luxation may involve excision arthroplasty, arthrodesis or (partial or complete) digit amputation.

COLLATERAL LIGAMENT REPLACEMENT:

Primary surgical management involves replacement of the medial and lateral collateral ligaments through a dorsal approach to the affected joint (see Figure 9.15 for this repair in a metatarsophalangeal joint). 0.9–1.5 mm bone tunnels are drilled transversely through the head and base of the two bones involved. Suture material (2–3 M slowly absorbable) is passed through the tunnels and tied such that the knots are adjacent to one or other of the bone tunnels (i.e. not likely to irritate the joint during movement). Outcomes for this technique can be excellent.

METACARPOPHALANGEAL/ INTERPHALANGEAL EXCISION ARTHROPLASTY:

This technique has been reported in dogs but not in cats. It is indicated for salvage of the joint and preserves a digital pad, which is variably functional, depending on the level of the excision arthroplasty. A dorsal approach is made to the affected joint preserving the dorsally located common digital extensor tendon which widens over the dorsal surface of the joint to blend with interosseous tendons and the various ligamentous structures which stabilize these small joints. The tendon may be elevated medially or laterally. Using a small sagittal saw, osteotome or rongeurs the head and base of the bones of the affected joint are ostectomized. The fascia and skin are closed routinely.

METACARPOPHALANGEAL/ INTERPHALANGEAL ARTHRODESIS:

Arthrodesis of the metacarpophalangeal joints in cats is possible. Kirschner wires (0.6–1.1 mm) are driven from the mid-shaft or base of the metacarpal bone in question. The metacarpophalangeal joint is extended and the Kirschner wire driven distally into the proximal phalanx. The joint is bent with a steady gentle application of force into a standing angle. Bone graft may be applied. A dressing should be applied to protect the healing site.

SALVAGE PROCEDURES FOR SEVERE DIGITAL TRAUMA:

With severe trauma to the phalanges, the loss of soft tissues (including skin, tendons, ligaments and joint capsule) may complicate repair. Fusion podoplasty may be performed as a salvage procedure in the treatment of degloving injuries of the digits. Digit amputation may be indicated as treatment for comminuted fractures, nonunions, intraarticular fractures, irreparable luxation, shearing injuries, neoplasia or chronic infections of the digit. In some cases of severe phalangeal injuries, the digits are amputated at the metatarsophalangeal joint and the metatarsal pad is advanced distally to cover the distal plantar aspect of the foot, thus preventing the need for a total limb amputation. A splint is applied to the limb for 4 weeks to allow complete healing and reduce the likelihood of pad injury caused by shearing motion between the pad and the metatarsal bones during weight bearing.

Degloving Injuries of the Digits

(Swaim & Pope 1988).

Options for management of degloving injuries of the digits include wound management to allow healing by granulation followed (hopefully) by re-epithelialization; primary closure where possible; secondary closure and consideration of grafting technique. Fusion podoplasty may be considered another treatment option for degloving injuries of the digits. The traumatized soft tissues are debrided between the digits, and between the digital and carpal pads which are preserved where possible. The digital and carpal pads are apposed on the palmar surface of the paw with interrupted sutures. The skin is apposed on the dorsal surface of the paw.

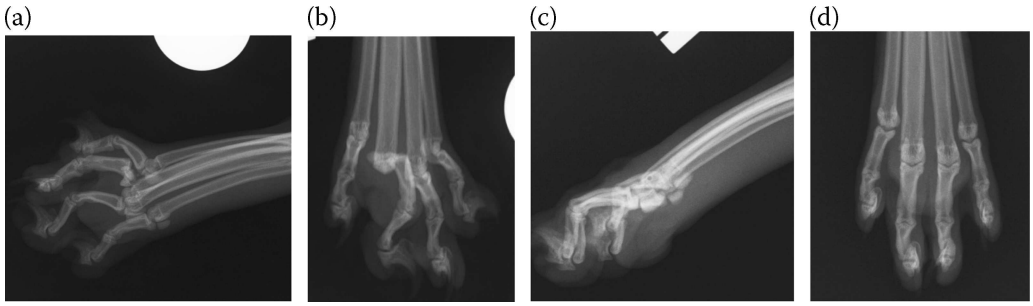


Figure 9.15 Mediolateral **(a)** and dorsoplantar **(b)** views of feline hind paw with luxation of the fourth metatarsophalangeal joint. A dorsal approach was made and bone tunnels were drilled across the head of the metatarsal and the base of the first phalanx. 3 m polydioxanone was passed through the bone tunnels in a loop and tied. Post-operative views **(c** and **d)** show good reduction.

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DIGIT AMPUTATION:

Digit amputation (Figures 9.16 and 9.17) may be indicated as treatment for comminuted fractures, fracture nonunion, intraarticular fracture, irreparable luxation, shearing injuries, neoplasia, or chronic infections of the digit. Amputation of the digits is usually performed by osteotomy of the head of the associated metacarpal/metatarsal, for the third and fourth digit, the osteotomy is transverse, while for the second and fifth metacarpal/metatarsal the osteotomy may be made oblique to create a smooth contour to the paw. Amputation at the distal interphalangeal joint is as described for onychectomy (see Onychectomy).

Most cats function very well after amputation of a single digit. Chronic lameness may occur after amputation of more than two digits, particularly if the third or fourth digit is removed. Preservation of the digital pad will improve postoperative function.

Technique for digit amputation:

1. The foot is clipped and aseptically prepared for surgery. Care is taken to clean the claws, pads and skin between the digits.
2. A tourniquet is considered to control haemorrhage.
3. An elliptical skin incision is made around the digit at the level of the amputation. The ellipse begins proximodorsally and ends distally on the palmar/plantar surface. The digital pad is preserved if the level of the amputation is distal enough. Vessel ligation and electrocautery are used to control haemorrhage.
4. The head of the bone proximal to the desired level of amputation is osteotomized using a sagittal saw, gigli wire or osteotome.
5. The subcutaneous tissues and skin are closed. If the digital pad remains, it is sutured to the skin to close the defect.
6. The foot is bandaged for 10 days and activity is restricted to allow healing.

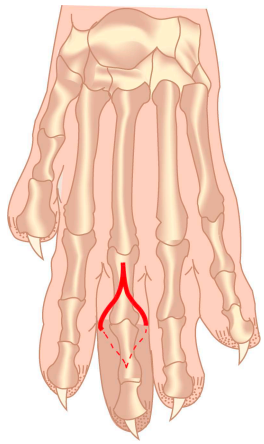


Figure 9.17 Digit amputation at the proximal interphalangeal joint. Diagram of bones of the foot depicting the location of the elliptical skin incision made for amputation of the third digit at the proximal interphalangeal joint. The joint is disarticulated to remove the second and third phalanges. The distal condyle of the first phalanx is removed with a rongeur. The digital pad is preserved if possible and sutured to the skin to close the defect.

In some cases of severe phalangeal injuries, the digits may be amputated at the metacarpophalangeal joint and the metacarpal pad is advanced distally to cover the distal palmar aspect of the foot, thus preventing the need for a total limb amputation (Figure 9.18). A splinted dressing is applied to the limb and changed frequently for 4 weeks to allow complete healing and reduce the likelihood of a pad injury caused by shearing motion between the pad and the metacarpal bone during weight bearing.

Onychectomy (Declawing)

(Bennett et al. 1988, Carroll et al. 1998, Jankowski et al. 1994, Landsberg 1994, Lin et al. 1993, Martinez et al. 1993, Mison

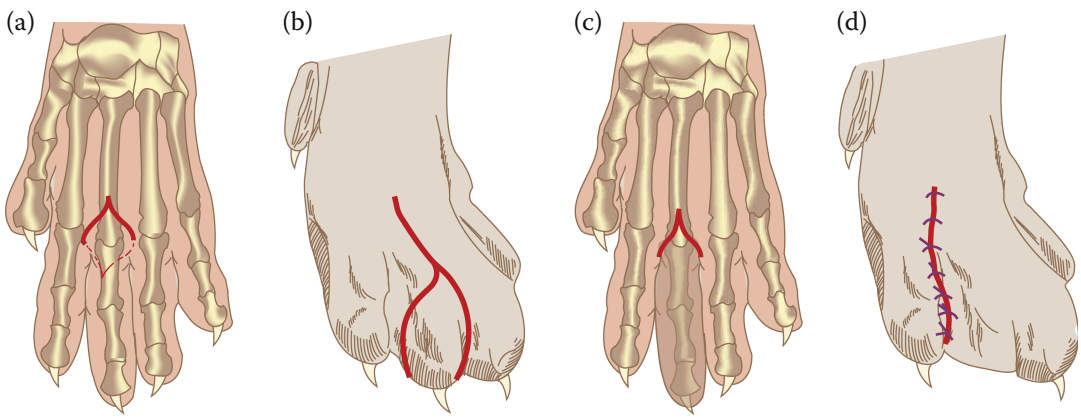


Figure 9.16 Digit amputation at the metacarpo-/metatarsophalangeal joint. **(a)** Diagram of bones of the foot depicting the location of the elliptical skin incision made for amputation of the third digit at the metacarpophalangeal or metatarsophalangeal joint. **(b)** Diagram of a cat's foot depicting incision location. The elliptical incision begins proximodorsally and ends distally on the palmar/plantar surface of the foot. **(c)** The metacarpo-/metatarsophalangeal joint is disarticulated to remove the phalanges. The distal condyle of the metatarsal/metacarpal bone is excised with a rongeur. **(d)** Subcutaneous tissues and skin are closed.

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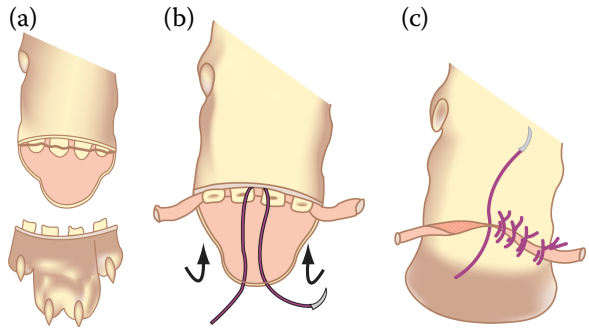


Figure 9.18 Metacarpophalangeal amputation and distal transfer of the metacarpal pad for treatment of irreparable phalangeal trauma. **(a)** The phalanges are amputated at the metacapophalangeal joint, preserving the carpal pad. The heads of the metacarpal bones are excised. **(b)** A surgical drain may be placed and a subcuticular suture pattern is placed to rotate the carpal pad distally to cover the ends of the metacarpal bones. **(c)** The remainder of the incision is closed with mattress sutures in the skin. The limb is splinted for 4 weeks.

et al. 2002, Morgan & Houpt 1989, Patronek 2001, Patronek et al. 1993, Rife 1998, Romans et al. 2005, Romans et al. 2004, Tobias 1994, Yeon et al. 2001).

Declawing procedures are commonly performed in domestic cats in some countries to prevent damage or injury to furniture, other pets, the owners and the cat itself. The procedure is usually performed on indoor cats between 3 and 12 months of age. In most cases only the front claws are removed. Thousands of cats are declawed each year in North America.

The humane considerations of onychectomy are controversial. Proponents consider it a justifiable option to prevent scratching, particularly if the cat cannot be retrained and is in danger of being removed from the home. Opponents condemn onychectomy on ethical grounds as a needless procedure (performed strictly for owners' convenience) that renders the cat unable to engage in species-specific behaviours, such as scratching, grooming and self-defense.

Onychectomy may be performed as a treatment in cases of fracture of the distal phalanx (P3).

Surgical Techniques

Onychectomy is the removal of the distal phalanx including the claw. Growth and development of the claw is derived from the basal germinal cell layer (stratum germinativum) located on the ungual crest of the distal phalanx (Figure 9.19). It is essential to remove the entire ungual crest (and all germinal cells) during surgery to prevent regrowth of the claw. The procedure is performed with the cat under general anaesthesia. Analgesic drugs are essential to minimize intraoperative and post-operative pain.

A tourniquet may be considered to minimize haemorrhage, though there is evidence that touniquets placed around the elbow may damage nerves. The limb is draped and the distal phalanx, including the claw, is excised from each of the front digits using one of several described techniques.

Amputation of the distal phalanx with a guillotine-type nail trimmer

The ring of a sterile nail trimmer is placed over the claw into the dorsal interphalangeal space between the second and third phalanx (Figure 9.20). The claw is grasped and lifted dorsally with a towel clamp or forceps and traction is applied to open the joint space. The nail trimmer is closed to complete the amputation after ensuring that the ring is positioned in the interphalangeal space dorsally and the digital pad is protected. The blade must be sharp to avoid crushing the tissues. Cutting the pad or the distal aspect of the middle phalanx will lead to greater post-operative discomfort and lameness. With the distal phalanx removed, the wound is inspected for complete excision of the ungual crest. The distal articular surface of the middle phalanx should be visible. In most cases, a small remnant of the distal phalanx (the flexor tubercle) remains in the wound and is visible just palmar to the articular surface of the middle phalanx. This remnant can be left *in situ* without concern for claw regrowth. However, if a large piece of the distal phalanx remains, it may contain germinal cells of the ungual crest and should be excised with a scalpel blade.

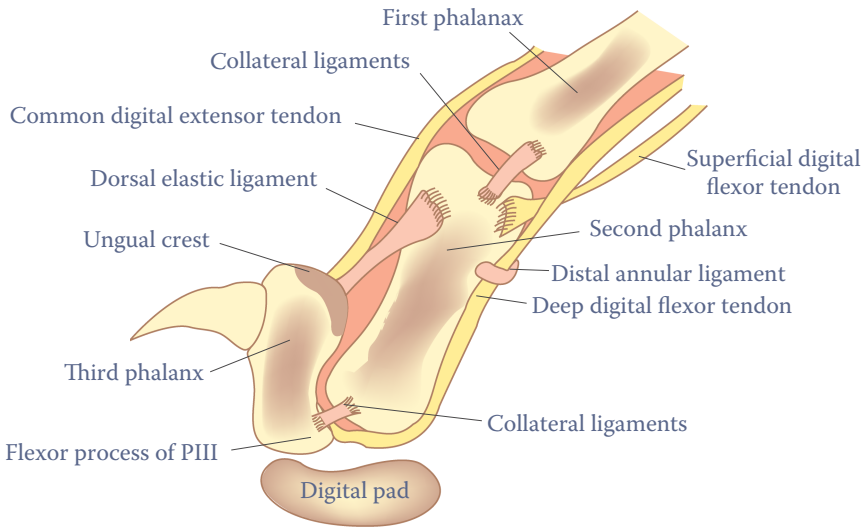


Figure 9.19 Feline phalangeal anatomy (lateral view).

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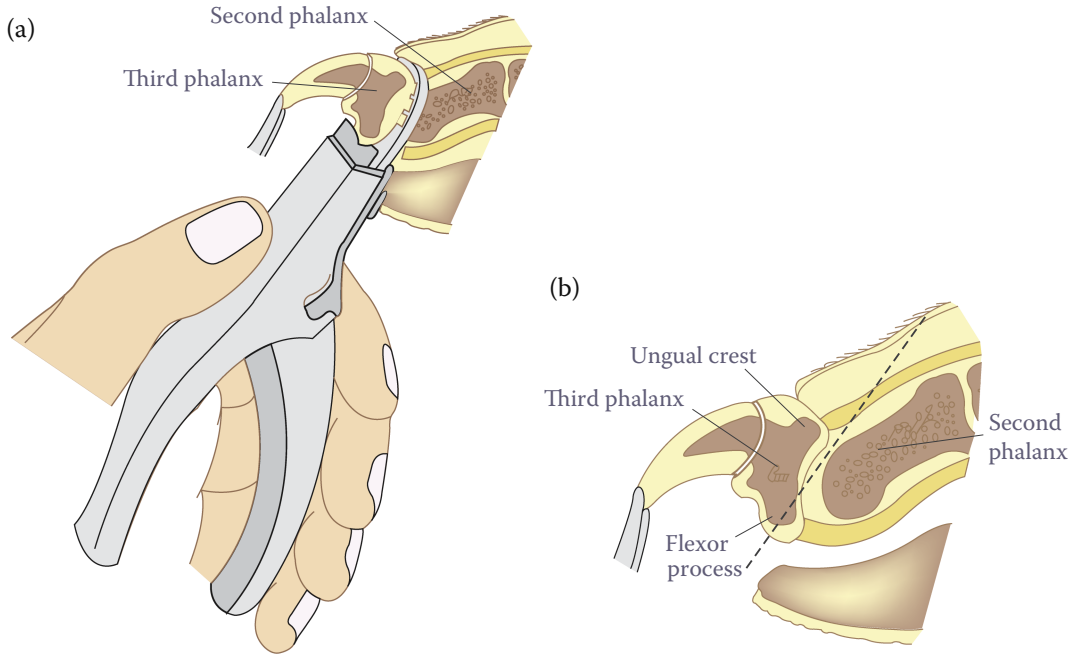


Figure 9.20 Onychectomy performed with a guillotine-type nail trimmer. **(a)** The ring of the nail trimmer is placed over the claw into the dorsal interphalangeal space between the second and third phalanx. The blade of the nail trimmer is positioned ventral to the distal phalanx, distal to the digital pad. Before performing the amputation, the claw is lifted dorsally to rotate the flexor process of the distal phalanx ventrally while the blade reflects the digital pad proximally. **(b)** With a proper amputation (dashed line), the entire ungual crest is removed to prevent regrowth of the claw. Inspection of the incision will reveal the distal articular surface of the second phalanx. A small remnant of the flexor process of the distal phalanx is seen palmar to the articular surface of the second phalanx.

Disarticulation of the distal phalanx

Removal of the distal phalanx by disarticulation of the distal interphalangeal joint is achieved using a scalpel blade or surgical laser.

Scalpel method

The claw is grasped with a towel clamp or forceps. Pushing up on the digital pad will also help extrude the claw. A circumferential incision is made in the hairless, cuticle-like skin along the dorsal aspect of the claw. The skin is retracted proximally away from the claw. The scalpel blade is then used to transect the common digital extensor tendon and dorsal elastic ligaments between the second and third phalanges (Figure 9.21). The incision is continued along both sides of the digit cutting the skin, joint capsule and palmar collateral ligaments. The dissection continues around the flexor tubercle of the distal phalanx to transect the deep digital flexor tendon. The distal phalanx is then dissected from the digital pad and other soft tissue attachments. Cutting the digital pad is avoided.

Laser method

Laser disarticulation of the distal interphalangeal joint is performed like the scalpel method, except that the incisions are made with a carbon dioxide (CO_2) laser (Luxar Corporation). Tourniquets are often not required during laser onychectomy because blood vessels are sealed by heat. Nerve endings and lymphatic vessels are also sealed, reportedly resulting in less pain and swelling after surgery. All routine safety measures for laser usage are followed.

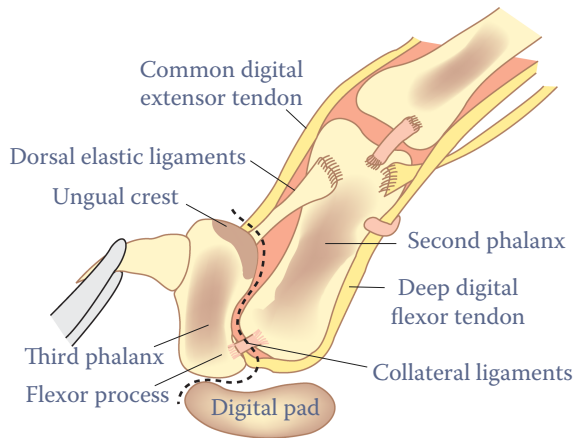


Figure 9.21 Onychectomy by disarticulation of the distal phalanx – scalpel method. The claw is grasped with forceps and a circumferential incision is made along the dorsal aspect of the claw with a scalpel blade (dashed line). The common digital extensor tendon and dorsal elastic ligaments are transected. The incision is continued along both sides of the digit, transecting the skin, joint capsule and collateral ligaments. The dissection continues in the palmar direction around the flexor process of the distal phalanx to transect the deep digital flexor tendon. The distal phalanx is then dissected from the digital pad and other soft tissue attachments. All of the distal phalanx, including the ungual crest and flexor tubercle, is removed.

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CLOSURE:

Closure of the onychectomy site may be by suturing or using a tissue adhesive. Suturing after onychectomy is not mandatory but may reduce the incidence of post-operative haemorrhage in adult and obese cats. A bandage is often applied after suturing. In most cases, the cat will remove the sutures within 2 weeks following surgery. Otherwise, sedation may be required to allow suture removal in some cats.

Post-Operative Care

Bandages may be applied to the front feet for 24 hours after onychectomy. The bandage should cover the foot and extend to the proximal antebrachium. Placing bandages too tightly can lead to swelling and necrosis of the foot. Mild bleeding can occur at bandage removal. If bleeding persists, the bandage is reapplied for an additional 24–48 hours.

Litter is replaced with shredded paper or commercially available paper litter for 2 weeks. Sutures are removed in 10–14 days if not already removed by the cat. Antibiotics are not routinely administered after onychectomy. In general, recovery to pain-free limb function is faster in young cats than in older or obese cats.

Analgesia

Analgesics are required to control pain after surgery. nonsteroidal anti-inflammatory drugs and oral opioids may be considered. Infiltration of a local anaesthetic agent (bupivacaine) in a 'ring block' pattern around the distal antebrachium or irrigation of the wounds with bupivicaine at the time of surgery may also reduce pain associated with onychectomy.

Complications

Complications are common after onychectomy and have been reported in up to 50% of cases. Early post-operative complications including haemorrhage and pain occur in approximately 24% of cats. Later complications including claw regrowth, chronic draining tracts, protrusion of the second phalanx, persistent lameness and behaviour changes occur in approximately 20% of cats. Radial neuropraxia or paralysis secondary to tourniquet use and tissue necrosis from improper bandaging have also been reported.

Alternatives to Onychectomy

Alternatives to declawing should be offered. These include deep digital flexor tenectomy, behaviour modification and capping.

DEEP DIGITAL FLEXOR TENECTOMY (FIGURE 9.22):

The deep digital flexor tendon inserts on the flexor tubercle of the distal phalanx. During extrusion of the claw from the sheath, the deep digital flexor muscle contracts, flexing the distal interphalangeal joint. Surgical excision of a portion of the deep digital flexor tendon prevents flexion of the distal interphalangeal joint and extrusion of the claw. Severing the tendon does not affect the cat's ability to walk normally. After tenectomy, the claws continue to grow and must be trimmed regularly to prevent them from growing into the digital pad.

Surgical Technique

A 3–5 mm skin incision is made over the palmar surface of each digit (beginning 3–5 mm proximal to digital pad) to expose the deep digital flexor tendon. The tendon lies just beneath the skin and is identified by its white, glistening appearance. The tendon is isolated by blunt dissection with a mosquito haemostat or curved iris scissors. A 5 mm long segment of the tendon is exteriorized and excised with a scalpel blade. Haemorrhage is usually minimal and can be controlled by direct pressure or electrocautery. The incision is closed.

Complications

Complications include haemorrhage, infection, pain, interphalangeal joint immobility and fibrosis, and excessive claw growth. Some cats can still use their claws after tenectomy, though the ability to scratch is limited. Owner satisfaction rates of 70–94% are reported after tenectomy.

BEHAVIOUR MODIFICATION:

Retraining or behaviour modification techniques may help prevent cats with claws from damaging furniture or scratching owners and other pets.

CAPPING:

An alternative to surgical declawing is to place a vinyl cap over the tip of each claw. The caps are attached with glue

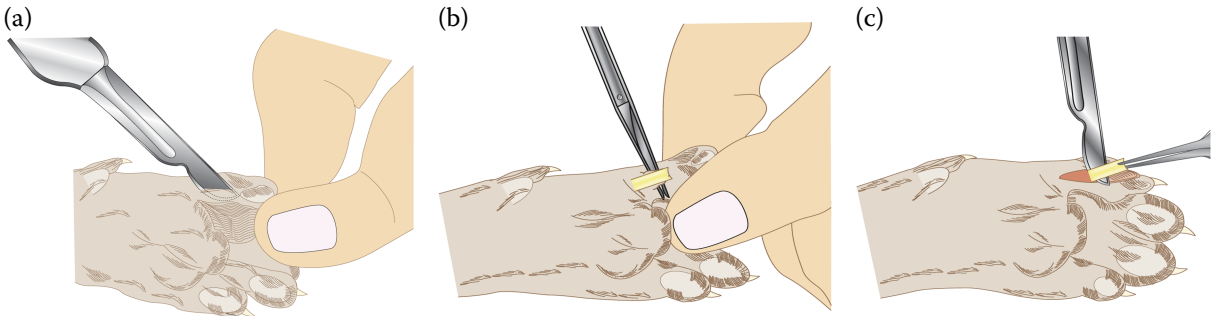


Figure 9.22 Deep digital flexor tenectomy. **(a)** A 3–5 mm skin incision is made over the palmar surface of each digit to expose the deep digital flexor tendon. **(b)** The deep digital flexor tendon lies just beneath the skin and is isolated with a mosquito haemostat or curved iris scissors. **(c)** A 5-mm long segment of the tendon is exteriorized and excised with a scalpel blade. Surgical excision of a portion of the deep digital flexor tendon prevents flexion of the distal interphalangeal joint and extrusion of the claw. After tenectomy, the claws continue to grow and frequent trimming is necessary.

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and must be replaced approximately every 6–8 weeks as the claws grow. Premature removal of the caps will also necessitate replacement.

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Part 5 Forelimb Amputation

Complications necessitating amputation may include severe injury, fracture nonunion, osteomyelitis, severe muscle contracture or joint dysfunction, and neurologic dysfunction. Neoplasia affecting the soft tissues or skeleton is also an indication for amputation. The cat should be carefully evaluated to ensure the remaining three limbs are healthy and can adequately support weight. Cats are typically able to ambulate well after amputation of one limb. Amputation of two limbs has been described in cats but is rarely an appropriate option.

FORELIMB AMPUTATION WITH SCAPULECTOMY:

Removal of the entire forelimb, including the scapula, provides excellent cosmetic results and is the preferred method. The entire forequarter is clipped and prepared for surgery. The skin incision begins at the dorsal border of the scapula and extends distally along the scapular spine to the shoulder joint. The incision is then extended circumferentially around the shoulder joint. Electrocautery may be used to control small bleeding vessels. Ligation is used to prevent haemorrhage prior to transection of larger vessels. The cephalic vein on the lateral aspect of the shoulder is ligated and transected. The omotransversarius muscle is sharply transected from the cranial border of the scapular spine. The incision is continued dorsally along the scapular spine, severing the cervical and thoracic portions of the trapezius muscle. The rhomboideus muscle is removed from the dorsomedial aspect of the scapula. The serratus ventralis muscle is elevated from the scapula. The scapula is then abducted from the body wall to expose the axillary space.

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The fascia is bluntly dissected to allow transection of the insertions of the latissimus dorsi, teres major and cutaneous trunci muscles near the humerus. The thoracodorsal artery and

vein are identified and separately ligated and divided. The thoracodorsal nerve is transected. The scapula is abducted further and rotated to expose the axillary artery. It is double ligated and transected. The lateral thoracic artery is ligated and transected. The brachial and axillary veins are separately ligated and transected. The nerves of the brachial plexus are then transected in a caudal to cranial direction. Branches of the superficial cervical artery are ligated. The scapula is further abducted to allow transection of the ventral musculature. The deep and superficial pectoral muscles are removed from their insertions on the humerus. The cleidobrachialis muscle is transected to complete the amputation. Bupivacaine can be injected into the nerves of the brachial plexus prior to sectioning. The fascia of the deep pectoral muscle is sutured to the scalenus and latissimus dorsi muscles. The fascia of the cleidobrachialis muscle is sutured to the fascia of the superficial pectoral muscle. Fascia of the trapezius and omotransversarius muscles is sutured to the dorsal aspect of the latissimus dorsi muscle. Subcutaneous tissue and skin are sutured. Dead space is reduced during closure to minimize seroma formation. The incision and body wall may be bandaged for 24–48 hours.

Forelimb Amputation Mid-Humerus

Removal of the forelimb at the mid-humerus is easier to perform but provides less satisfactory cosmetic results than amputation with scapulectomy because atrophy of the supra- and infraspinatus muscles results in a prominent scapular spine and humeral remnant.

A lateral skin incision is made from the cranial aspect of the brachium, distally to just proximal to the elbow joint, and then proximally to the caudal aspect of the brachium. The incision is continued in a straight line on the medial aspect of the limb to connect the cranial and caudal portions of the lateral incision. Electrocautery may be used to control small bleeding vessels. Ligation is used to prevent haemorrhage prior to transection of larger vessels. Medially, the brachial artery and vein are separately ligated and transected proximal to the collateral ulnar artery. The median, musculocutaneous and ulnar nerves are transected. The triceps tendon is transected near the olecranon. The brachialis and biceps brachii muscles are transected near their insertions distal to the elbow joint. The cephalic vein is ligated and divided. The radial nerve is cut. The brachiocephalicus muscle is elevated from the humeral crest to expose the humerus. The bone is cut at its mid-shaft using a power saw or wire saw. The distal aspect of the triceps is sutured to the brachial and biceps brachii muscles to cover the cut end of the humerus. The subcutaneous tissues and skin are sutured. A bandage may be applied to control swelling.

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10 Hindlimb Fractures

Part 1 Fractures of the Pelvis

The pelvis is comprised of two bilaterally symmetrical hemipelves, each consisting of the ilium, ischium, pubis and acetabular bones. Each ilium is attached to the spine at the sacrum, forming the sacroiliac joints. Weight-bearing loads are transmitted from the femoral head to the acetabulum, along the body of the ilium, and through the sacroiliac joint to the spine.

PELVIC FRACTURES:

(Bennett 1975, Bookbinder & Flanders 1992, Draffan et al. 2009, Hill 1977, Kolata 1980, Meeson & Geddes 2015, Muir 1988, Stieger-Vanegas et al. 2015a,b, Verstrate & Lambrechts 1992, Whitney & Mehlhaff 1987, Witte & Scott 2012).

Pelvic injuries are common in cats (20%–22% of all feline fractures), usually resulting from severe trauma. Concomitant injuries occur in up to three quarters of cases. Over half of cats with pelvic fractures have other musculoskeletal injuries (sacral fracture, coxofemoral luxation, femoral fractures, sacrocaudal luxation). A quarter of cats with pelvic fractures show concurrent thoracic or abdominal injuries (pneumothorax, haemothorax, urinary tract injury, gastrointestinal injury, abdominal hernia, diaphragmatic hernia, rectal perforation and prepubic tendon rupture). A fifth of cats with pelvic injuries show concurrent nervous system injuries (lumbosacral plexus, sciatic nerve, caudal nerves). These injuries should be assessed and addressed prior to consideration of intervention for pelvic injuries.

Because the pelvis is a rigid box, injuries usually occur at two or more sites, leading to displacement of the fragments and narrowing of the pelvic canal. The four most common pelvic fracture combinations are as follows:

- Ilial shaft fracture plus contralateral sacroiliac joint luxation, pelvic floor fractures;
- Ilial shaft fracture plus pelvic floor fractures;
- Sacroiliac luxation plus pelvic floor fractures;
- Bilateral sacroiliac luxation +/- pelvic floor fractures.

All four common fracture combinations routinely include fractures of the pelvic floor (pubis and ischium). Pelvic fractures are rarely open because the pelvis is completely surrounded by muscle and other soft tissues.

Cats with pelvic fractures are often unwilling to stand or ambulate initially, but may be ambulatory within a few days, particularly where injuries are primarily unilateral. Diagnosis is by physical examination and imaging. Bony landmarks (greater trochanter, ilial wing, ischium, etc.) may be asymmetrical. Digital rectal examination of the sedated or anaesthetized cat may help with assessing the integrity of the pelvic canal. Standard ventrodorsal (with hips extended and/or in frog-leg position) and lateral radiographic views of the pelvis are acquired. Oblique views reduce overlap of the two hemipelves and may be helpful. Computed tomography is a superior imaging modality for identifying sacral and some pelvic fractures (ischial and pubic fractures) in dogs, and this is further enhanced by the use of three-dimensional reconstruction over two-dimensional image analysis alone (Figure 10.1).

Conservative management involves crate confinement for 4–6 weeks. Urination and defecation are monitored and the cat is kept clean and dry to avoid decubital ulcers. Physical therapy including passive range-of-motion exercises is performed if tolerated by the cat. Analgesic medications should be used as required. Fracture healing is monitored radiographically to ensure healing is progressing. However, surgical intervention should be considered where:

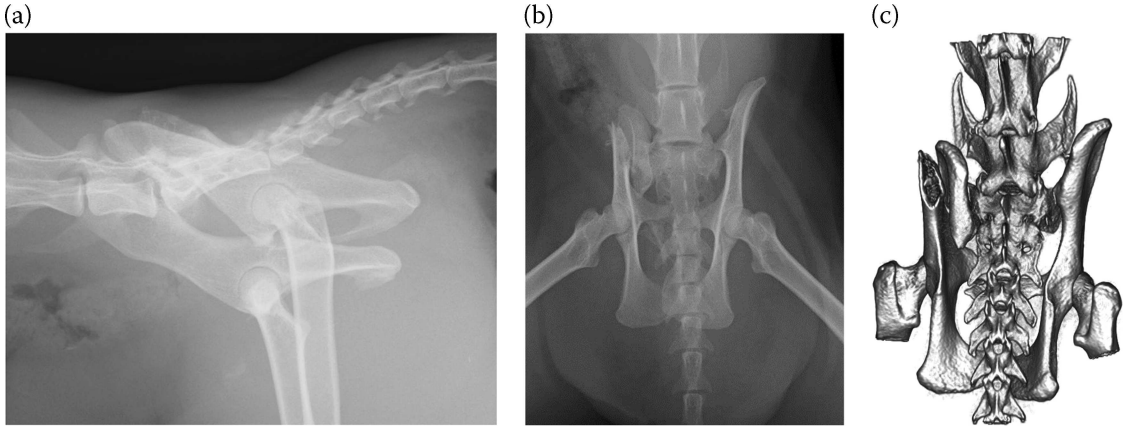


Figure 10.1 Radiography and computed tomography for imaging of a cat's pelvis with multiple injuries following a road traffic accident. **(a, b)** lateral and ventro-dorsal view of pelvis revealing left-sided sacroiliac luxation, pubic and ischial fractures and right-sided cranial ilial fracture. **(c)** Reconstructed 3-dimensional volume rendering view of the same pelvis permitting improved evaluation of the pubic and ischial injuries and identification of a fracture of a right L7 lateral process.

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- The fractures involve the weight-bearing axis of the pelvis (acetabulum, ilial body, sacroiliac joint).
- Significant narrowing of the pelvic canal is present.
- The fracture involves the articular surface of the acetabulum.
- Concurrent orthopaedic injuries require stabilization for the cat to ambulate.
- Extreme pain or neurologic deficits indicate nerve injury.

So surgical intervention is commonly performed for pelvic injuries. Nonetheless, one report documented a 22% complication rate following surgical management of pelvic fractures, most commonly sciatic neuropraxia (13%), which resolved spontaneously in the vast majority of cases. In 86% of cats with surgical management of pelvic fractures there was no long-term impairment to mobility. Similar studies of conservative management of pelvic fractures do not exist for comparison.

SACROILIAC LUXATION:

(Bookbinder & Flanders 1992, Borer et al. 2008a,b, Burger et al. 2004, Kaderly 1991, Lanz 2002, Montavon et al. 1985, Parslow & Simpson 2017, Philp et al. 2018, Pratesi et al. 2018, Raffan et al. 2002, Shales et al. 2009, Shales et al. 2010, Silveira et al. 2017, Tomlinson et al. 1999, Yap et al. 2014).

Sacroiliac luxation involves displacement of the ilial body from its attachment to the sacrum. Fractures of the pubis and ischium are often present and allow displacement of the hemipelvis, usually cranially and dorsally. Concurrent injury to the lumbosacral nerve trunk or sciatic nerve is not infrequent.

Conservative Therapy

Conservative therapy is considered in cases with minor displacement, when no neurologic deficits are present, or when the cat is ambulating well. Because cats are lightweight, many respond well to conservative treatment. However, stabilization of the sacroiliac joint may improve the cat's comfort, allows earlier return of limb function and reduces repeated trauma to the lumbosacral nerve trunk.

Surgical Therapy

Several techniques have been described for surgical stabilization of sacroiliac fracture/dislocation in cats, however, the mainstay treatment for the majority of cases should be an ilio-sacral lag screw. Where possible, the use of intra-operative imaging should be considered to facilitate reduction and implant positioning.

Placement of an Ilio-Sacral Lag Screw with an Open Approach

1. The cat is positioned in sternal recumbency with a bean bag or similar under the pelvis such that the hindlimbs are mildly abducted and the hips are in a neutral position (Figure 10.2,). Lateral recumbency has also been reported, however, in cases with bilateral sacroiliac luxation this positioning necessitates turning of the patient intra-operatively. Since this procedure requires familiarity with the anatomy, the authors advocate becoming familiar with one technique, and since sternal recumbency is appropriate for unilateral and bilateral sacroiliac luxations, this is the recommendation.
2. A dorsal surgical approach is made to the lateral aspect of the sacral midline. Unless there is severe displacement of the hemipelvis, this incision should be over the dorsal aspect of the ilial wing.
3. The origin of the superficial gluteal muscle is elevated from the lateral aspect of the ilium. The ilial wing is displaced ventrally to expose the lateral aspect of the

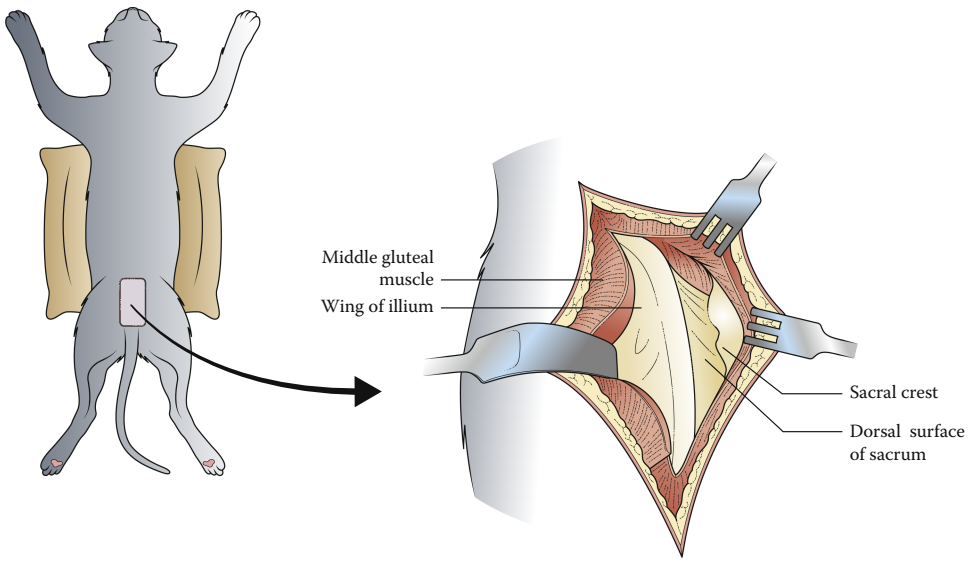


Figure 10.2 Positioning and approach for SI luxation surgery from dorsal approach. The dorsolateral approach to the sacroiliac joint. A. The cat is positioned in sternal recumbency on top of a wedge, sandbag or rolled towel placed to elevate the abdomen and thorax. The limbs hang either side with the hips in a neutral position with some abduction. B An incision is made over the dorsal iliac spine. The middle gluteal origin on the ilium is elevated. The ilial wing may be grasped, with pointed or fragment forceps, and displaced ventrally, to expose the lateral surface of the sacral wing.

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sacral wing. Care should be taken when placing a periosteal elevator or Hohman retractor ventral to the sacral wing to lever the ilium ventrally, to avoid iatrogenic trauma to ventral sacral nerves and blood vessels.

4. The sacral wing is visualized and the articular surface identified, sometimes necessitating .gentle scraping with a periosteal elevator. The articular surface of the sacral wing consists of a semilunar synovial portion (which is located caudoventrally and is covered with hyaline cartilage) and a fibrocartilaginous portion which is located craniodorsally.
5. The proper location to insert the screw into the sacral body (which is not visible to the surgeon and which has a cross-sectional area of approximately 0.5 cm^2) is approximately 1 mm dorsal to the geometric centre of the sacral wing, usually just cranial to the crescent shaped hyaline cartilage. A bone tunnel of appropriate size for the chosen screw (screw diameters of 2.4 mm or 2.7 mm are appropriate for all cats) is drilled into the sacrum. Care is taken to avoid ventral, dorsal or cranial exit from the sacral body, since screw purchase will be weakened and damage to the median sacral vessels and nerves, cauda equina and lumbosacral intervertebral disc is possible. Safe corridors for the feline sacral body have been described in all planes using plain radiographs and computed tomography (Figure 10.3,).
6. A glide hole is drilled in the ilial wing. The location of the glide hole is half way between the dorsal and ventral surfaces of the ilial wing at a level approximately two thirds caudally along the dorsal iliac spine.
7. Bone holding forceps are used to manipulate the ilium into position and the screw hole's depth is measured. The screw should be long enough to extend 60% of the distance across the sacrum. Premeasuring the distance on the ventrodorsal radiograph is helpful. The screw is placed and tightened, compressing the sacroiliac joint (Figure 10.4,). Some authors advocate use of a washer to prevent progression of the screw head into the ilial bone during tightening.

ALTERNATIVE/MODIFIED TECHNIQUES FOR FELINE SACROILIAC LUXATION FIXATION:

Ventrolateral Surgical Approach

A ventrolateral surgical approach may be used when repair of ilial fractures will be performed concurrently, since it is easily combined with a lateral approach to the ilium. Lateral recumbency is required and a skin incision is made from the centre of the iliac crest to the greater trochanter. The intermuscular septum, between the middle gluteal muscle and the tensor fascia lata, is incised from the ventral iliac spine to the cranial border of the biceps femoris muscle. Visualization of the sacral body is as poor as with the dorsal approach, and digital palpation is used to identify the ventral margin of the bone.

Ventral Surgical Approach

A ventral approach has been described through the abdomen, with lag screws placed from the ventral sacral wing directed dorsolaterally to purchase the ilial wing. The technique was described in cadavers and has been reported in clinical cases. This technique may be useful in cases in which abdominal surgery is indicated as a result of concurrent injuries.

Fluoroscopic Guidance

The use of fluoroscopy or intra-operative plain radiography was reported to improve the accuracy of open iliosacral lag screw positioning. Closed sacroiliac reduction and iliosacral lag screw placement have been reported in dogs, though not in cats. This technique reduces the iatrogenic trauma to the tissues.

Single Transsacral Screw or Pin for Bilateral Sacroiliac Luxation

Bilateral sacroiliac luxation can be stabilized using separate surgical procedures to place lag screws bilaterally (avoiding interference between the tips of the two screws if possible). Alternatively, a technique for placing a single, long transsacral screw to stabilize bilateral dislocations has been described.

1. Dorsal approaches are made to both sacroiliac joints as described for unilateral iliosacral screw placement.
2. A single drill hole is made through the entire width of the sacrum, starting and aiming to emerge, at the described optimal location (1 mm dorsal to the geometric centre of the sacral wing). With dorsolateral approaches made to both sacroiliac joints it is possible to visualize the drill bit emerging from the sacrum and to reposition if necessary. Intra-operative imaging is useful to achieve accurate drilling.
3. The ilial bone tunnels are drilled.
4. The first sacroiliac joint is reduced and a long screw is placed through the ilium into the sacral thread hold. Measurements from pre-operative radiographs may direct screw length choice, however, if an excessively long screw is selected, it can be easily cut with a bolt cutter as a final step prior to closure of the incisions. As the tip of the screw emerges from the sacrum, the second ilium is passed onto it, and the screw is advanced further, until compression is achieved at the first sacroiliac joint.
5. Compression is achieved at the second sacroiliac joint using one or two nuts passed over the tip of the screw and tightened.

A similar technique for bilateral sacroiliac luxation involves placing a single Kirschner wire through both ilia and the sacral body, via dorsal approaches. The tips of the Kirschner wire are bent cranially. This technique may be somewhat simpler technically, but does not produce compression at the sacroiliac joints, and may therefore have poorer resistance to rotation of the hemipelvis about the axis of the pin.

Trans-Ilial Techniques

A number of techniques have been described to reduce the risks innate to passing implants through the small feline sacral body. These may be used as stand-alone procedures in cases with relatively good stability (unilateral sacroiliac luxation with minimal displacement, e.g.) or to augment fixation with an iliosacral screw where additional stability is required.

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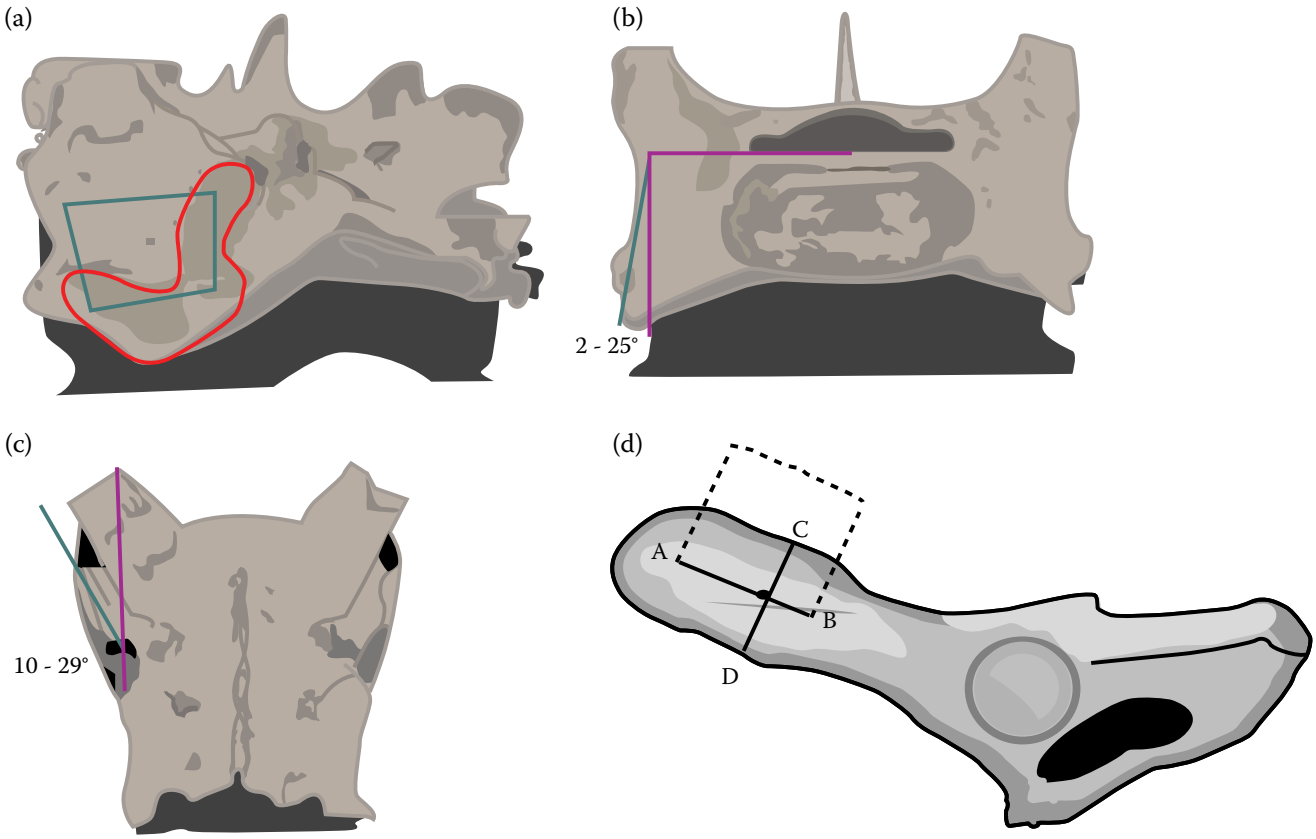


Figure 10.3 Corridors for drilling sacrum and ilium for sacroiliac lag screw placement. Line drawings of the feline sacrum: **(a)** lateral aspect, **(b)** cranial aspect, **(c)** dorsal aspect. The red outline shows the approximate location of the cartilaginous component of the sacroiliac joint, while the green box indicates the location of the first sacral vertebral body. The optimal site of screw insertion is shown (approximately 1 mm dorsal to the geometrical centre of the sacral wing). The dorsoventral inclination of the sacral wing is between 2 and 25°. The craniocaudal inclination of the sacral wing is between 10° and 29°. **(d)** the landmarks for the appropriate placement of the ilial wing glide hole include the cranial and caudal extents of the dorsal iliac spine and the dorsal and ventral margins of the ilial wing. The hole is drilled at the level of the caudal third of the iliac spine (ie between A and B marked in the diagram) and halfway from dorsal to ventral (ie between C and D).

Transilial implants that have been described include a strand of large monofilament nylon suture (tension band technique), Kirschner wires (Figure 10.5), lag screws and long bolts with nuts at either end. Implants are typically placed from lateral to medial through one iliac crest, passed dorsal to the dorsal lamina of the seventh lumbar vertebra, and driven from medial to lateral through the second iliac crest. Purchase of the dorsal spinous process of the seventh lumbar vertebra between the ilia has been proposed to improve stability, since without this there is no direct fixation of the ilia to the vertebral column; however, this is difficult in cats. A second transilial implant, parallel to the first, may be considered to give rotational stability. In combination with transiliosacral lag screw placement, a transilial pin may be considered to improve stability for very unstable luxations (Figure 10.6).

ILIAL FRACTURES:

(Bowlt & Shales 2010, Case et al. 2012, Guthrie & Kalff 2018, Hamilton et al. 2009, Langley-Hobbs et al. 2009, Schmierer et al. 2015, Scrimgeour et al. 2017).

Fractures of the ilium disrupt the weight-bearing axis of the pelvis, leading to significant dysfunction. They are usually accompanied by fractures of the pubis and ishium, allowing the caudal segment of the fracture to collapse medially into the pelvic canal. Ilial fractures in cats are typically long oblique, extending from a point cranioventral to a point caudodorsal, sometimes extending dorsal to the acetabulum. The sciatic nerve courses along the medial aspect of the ilial body and can be traumatized at the time of the trauma or at the time of surgical manipulation.

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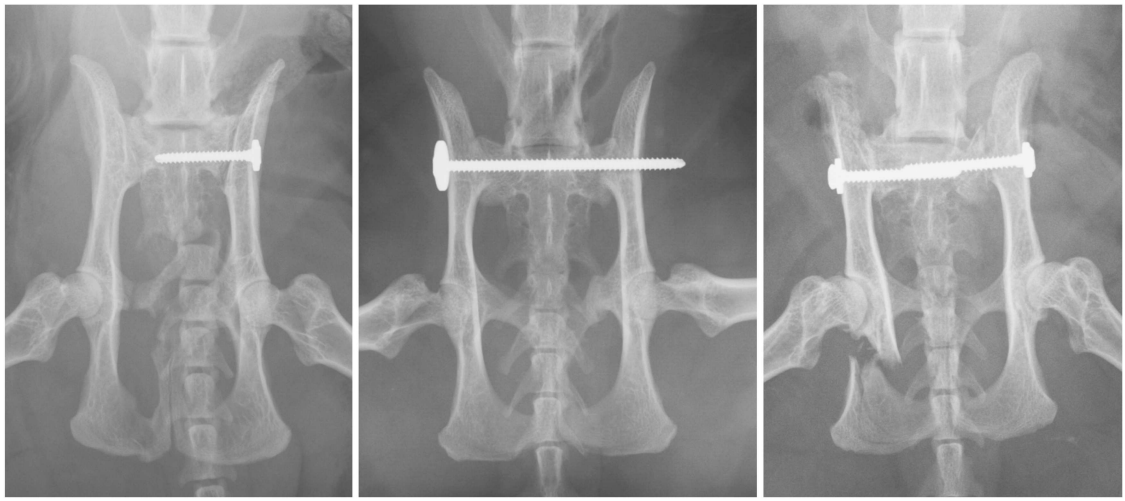


Figure 10.4 Post-op radiographs of single iliosacral screw, bilateral SI luxation with long screw, bilateral SI luxation two screws.

Reduction and fixation are recommended to restore limb function, prevent narrowing of the pelvic canal, and prevent injury to the sciatic nerve (through motion of the fragments or exuberant callus formation). Fixation involves a lateral approach and lateral plating of the ilium using locking implants.

1. The cat is positioned in lateral recumbency. A hanging-limb preparation allows the limb to remain in the surgical field for manipulation as needed during surgery.
2. A lateral approach involves a skin incision from the iliac crest to the greater trochanter (Figure 10.7). The middle gluteal and the tensor fascia lata muscles are separated. The deep and middle gluteal muscles are reflected dorsally. A Hohmann retractor is placed over the dorsal aspect of the ilium to maintain retraction of the gluteal muscles.
3. Reduction techniques include direct manipulation through bone holding forceps, levering the two fragments relative to one another with a periosteal elevator, application of bone holding forceps to the ischiatic tuberosity (provided the ischium is attached to the ilium) and manipulation of the hindlimb (which acts on the caudal fragment via the hip joint) (Figure 10.8)
4. Reduction may be maintained either temporarily or permanently using implants (1.4 mm to 2.0 mm Kirschner wires or 2.0 mm screws in positional or lag mode) placed across long oblique fractures from ventral to dorsal.
5. Bone plates may be contoured to the lateral surface of the ilium such that a minimum of two screws are placed cranial and caudal to the fracture site. Locking plates and recommended, and T-plates are useful, since there is often a paucity of bone stock either cranially or caudally. Screws are inserted (non-locking screws may be placed into one or two holes initially to pull the bone to the plate and ensure good reduction, before replacement with locking screws) and tightened.

A single study describes intramedullary pinning of the hemipelvis from the ischium to stabilize a unilateral transverse ilial fracture following femoral head and neck ostectomy. This technique should only be considered for cats with no functional hip joint and is not recommended.

The Importance of Locking Plates and Screws in Feline Ilial Fracture Fixation

Whilst lateral body plating is commonly advocated, presumably because the approach is simple and gives good visualization of the lateral surface of the ilium, screw loosening has been reported in up to 60% of feline cases. Loosening of screws has been theorized to be associated with the relatively thin feline cortices and the narrow space between the two cortices allowing toggling of non-locking screws. Techniques reported to reduce the frequency of screw loosening in ilial plating in cats include positioning a lateral plate so that at least one screw can be placed into the sacral body (increasing the holding power for that screw) and placing plates ventrally or dorsally on the ilium, such that screws purchase the much more widely spaced ventral and dorsal cortices, reducing their chances of toggling loose (Figure 10.9). However, the method of choice for reducing the frequency of screw loosening in feline ilial fracture repair is use of locking plates and screws (Figure 10.10). Several studies have demonstrated superiority of locking plates over non-locking plates in this particular situation.

ACETABULAR FRACTURES:

(Ablin & Gambardella 1991, Beck et al. 2005, Bookbinder & Flanders 1992).

Acetabular fractures are typically unilateral in cats, and most (77%) are two-piece fractures, making them favourable candidates for reduction and fixation with a successful

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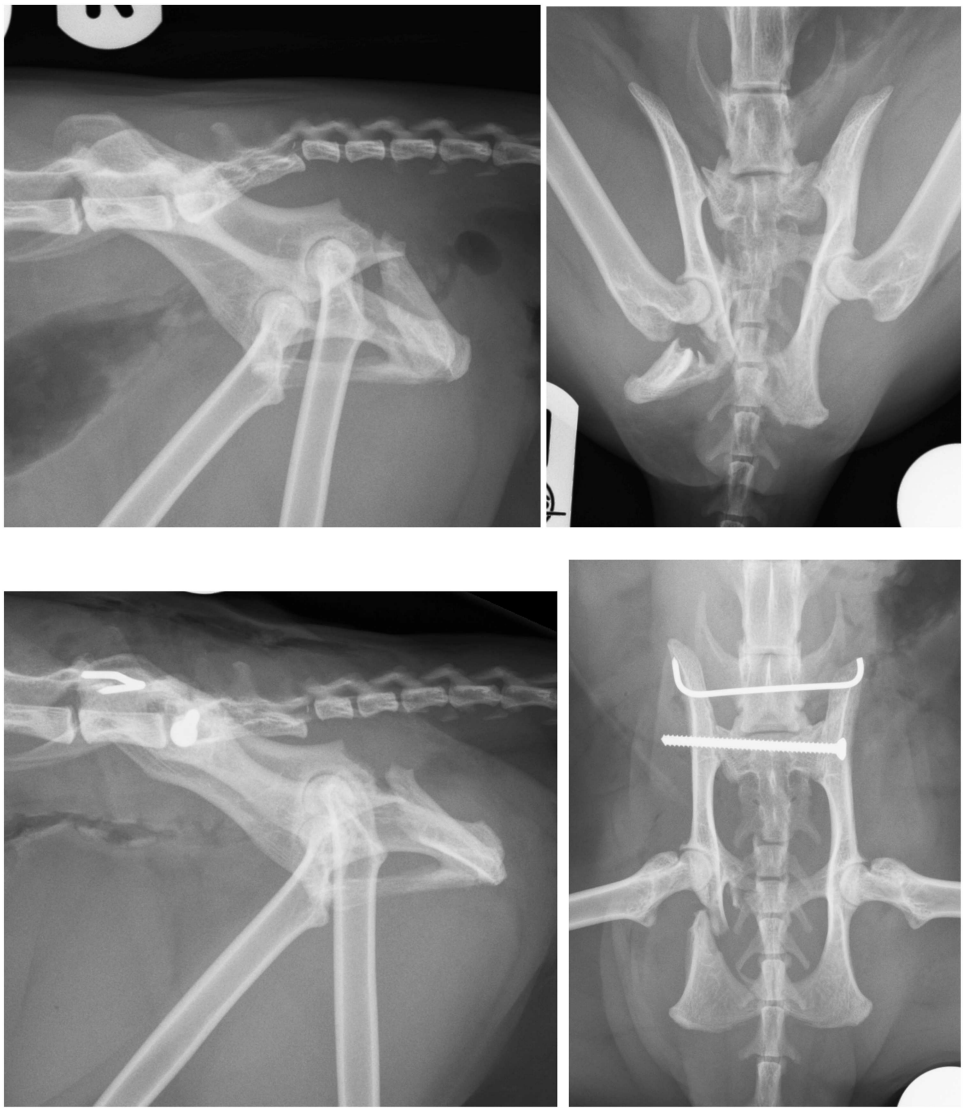


Figure 10.5 Pre-operative and post-operative views of the pelvis of a cat demonstrating bilateral sacroiliac luxation with severe cranial displacement and caudal pelvic canal narrowing (necessitating surgical intervention) with an ischial fracture on the right side caudal to the acetabulum, right-sided pubic fractures and sacrocaudal luxation. Fixation of the reduced sacroiliac joints was with a long 2.7 mm cortical screw placed in lag function from left to right with a plastic nut applied over the screw tip to give compression on the right sacroiliac joint. A transilial 1.4 mm Kirschner wire was applied (including purchase of the L7 dorsal spinous process) and the tips were bent cranially to maximize stability and minimize the potential for narrowing of the caudal pelvic canal.

outcome. Where reduction and fixation are not options, conservative therapy may be considered, although femoral head and neck ostectomy or total hip replacement may carry a superior prognosis.

Conservative Management

Fractures involving the caudal third of the acetabulum may be treated conservatively, though this portion of the acetabulum is reportedly involved in weight-bearing in cats. Conservative management should be considered only when there is no apparent displacement of the fragments, no narrowing of the pelvic canal, and no palpable crepitus with manipulation of the hip joint. Restricting activity may allow healing and restoration of a functional joint. It may be preferable to avoid surgery in some cases; however, it can be assumed healing of such a fracture will involve greater callus formation and disruption of the articular surface than following reduction and fixation (and therefore more osteoarthritis in future). In most cases, the cat with a conservatively managed caudal acetabular fracture will begin using the limb within 10–14

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Figure 10.6 Lateral and ventrodorsal radiographic projections of the pelvis of a cat with pelvic injuries and surgical stabilization. **(a** and **b)**: pre-operative views demonstrate left-sided oblique cranial ilial fracture and right-sided sacroiliac luxation with minimal displacement. Note the location of the ilial body following medial and ventral displacement. Cats in which the ilium has displaced ventral to the sacrum are often in significant discomfort, presumably as a result of impingement on sacral nerves. In addition, retrieval of the ilial body from this location during surgery may be difficult, but must be performed with care to avoid further trauma to the sacral nerves and median sacral blood vessels. **(c** and **d)**: post-operative views demonstrate stabilization of the sacroiliac joints with a transilial 1.6 mm Kirschner wire bent cranially on the lateral aspect of the iliac wings. A lateral 2.4 mm locking T-plate (Securos Pax) has been applied to stabilize the ilial fracture. Note: caudal pelvic canal narrowing is apparent, presumably as a result of imperfect ilial fracture reduction, although undetected left sacroiliac luxation may also be present.

days. Salvage procedures such as femoral head and neck ostectomy and total hip replacement (Chapter 12) may be indicated if comfort does not improve. Figure 10.8 (a) Depending on the conformation of an ilial fracture the caudal section of ilium may be manipulated through the use of bone holding forceps applied at one of three locations: the ilium cranial to the acetabulum, the greater trochanter of the femur or, via a separate short approach, the caudal aspect of the ischium. In this way fracture reduction may be achieved. (b) a pointed reduction forceps may be applied from dorsal to ventral across the ilium to maintain fracture reduction during application of fixation.

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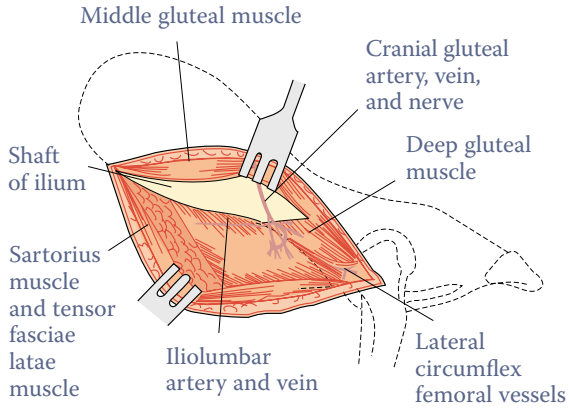


Figure 10.7 Lateral approach to the ilium. A skin incision is made from the iliac crest to the greater trochanter. Approach is continued between the tensor fascia lata and the middle gluteal muscles. The deep and middle gluteal muscles are reflected dorsally using a periosteal elevator to expose the ventral and lateral surfaces of the ilium. The lateral circumflex femoral vessels and the cranial gluteal vessels and nerve are preserved. The iliolumbar vessels course ventral to the ilial wing and these may be transected as the exposure is continued cranially. The ventral portion of the gluteal attachment on the cranial aspect of the ilial wing is cut to increase visualization of the cranial ilium.

Open Reduction and Internal Stabilization

Surgery should reconstruct the articular surface to improve comfort, encourage early limb use and minimize the development of osteoarthritis.

1. A dorsal approach to the hip including ostectomy of the greater trochanter is performed.
2. The fragments are reduced and temporarily stabilized using pointed reduction forceps or an oblique Kirscher wire. Reduction techniques include direct manipulation through bone holding forceps, levering the two fragments relative to one another with a periosteal elevator, application of bone holding forceps to the ischiatic tuberosity (provided the ischium is attached to the ilium) and manipulation of the hindlimb (which acts on the caudal fragment via the hip joint). The sciatic nerve must be identified and protected throughout the procedure.
3. Internal fixation is performed using a conventional (or preferably locking) plate with two screws cranial and caudal to the fracture (Figure 10.9). If a non-locking plate is applied it must be perfectly contoured to the bone to prevent disruption of fracture reduction during screw tightening.
4. Closure involves fixation of the greater trochanteric osteotomy with two Kirschner wires and a figure-of-eight tension band wire Figure 10.9 (Figures 10.11 and 10.12).

ISCHIAL AND PUBIC FRACTURES:

(Kipfer & Montavon 2011).

The ventral approach for stabilization of ischial and pubic fractures has been reported and may be considered for reducing pelvic canal narrowing in cats with intact ilia and acetabula, if a ventral approach is already being made, for example, to address abdominal disease. However, in general, ischial and pubic fractures (and pubic symphyseal separations) rarely require surgical intervention. Most occur in conjunction with other pelvic fractures, and reduction of

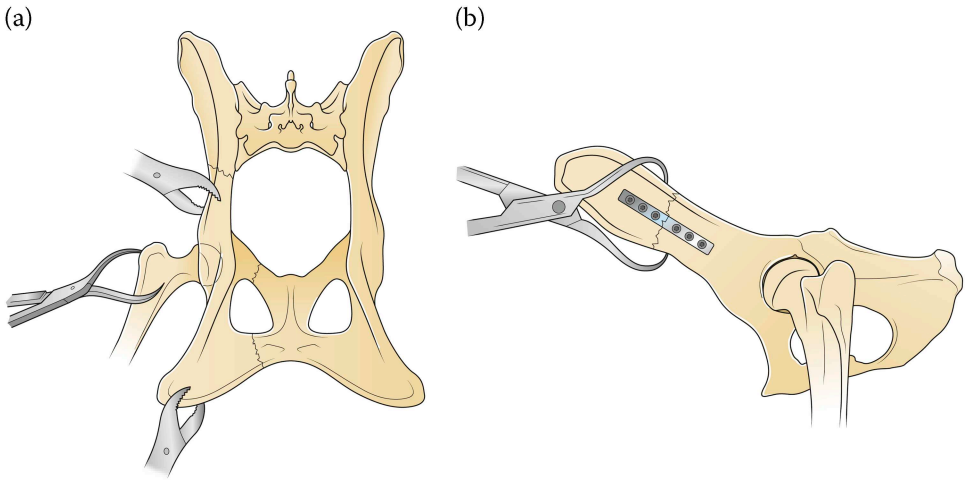


Figure 10.8 **(a)** Depending on the conformation of an ilial fracture the caudal section of ilium may be manipulated through the use of bone holding forceps applied at one of three locations: the ilium cranial to the acetabulum, the greater trochanter of the femur or, via a separate short approach, the caudal aspect of the ischium. In this way fracture reduction may be achieved. **(b)** a pointed reduction forceps may be applied from dorsal to ventral across the ilium to maintain fracture reduction during application of fixation.

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the concurrent injuries (sacroiliac luxation, ilial fracture, or acetabular fracture) usually aligns the ischial/pubic fractures as well. The ischial tuberosity is occasionally markedly displaced by the origins of the biceps femoris, semitendinosus and semimembranosus muscles and may be replaced using interfragmentary wires.

PELVIC MALUNION FRACTURES:

(DeGroot et al. 2016, Ferguson 1996, Matthiesen et al. 1991, McKee & Wong 1994, Schlicksup et al. 2013, Schrader 1992).

Malunion of the pubis and ischium often causes no clinical abnormalities. However, malunion of the acetabulum may lead to poor joint congruence and osteoarthritis, in which case salvage options (femoral head and neck ostectomy and total hip replacement) should be considered. Malunion of the ilium may be a cause of lameness but is more likely to be of detriment as a result of narrowing of the pelvic canal. A narrow pelvic canal may be a cause of obstipation, and secondary megacolon. Megacolon in cats is defined as a colonic diameter greater than 1.5 times the length of the seventh lumbar vertebral body. In some cases, stool softeners, proper diet and occasional enemas may resolve the clinical signs. However, if signs are persistent or recur frequently, surgical intervention to widen the pelvic canal is indicated. Widening the pelvic canal may not return defecation to normal in cats with neurologic deficits, idiopathic megacolon, or where signs of obstipation have been present for more than 6 months. In these cases, additional subtotal colectomy is recommended.

Splitting and separating the pubic symphysis, partial pelvectomy and corrective ostectomy procedures can widen



Figure 10.9 Pre-operative and post-operative radiographs of the pelvis of a cat with right ilial fracture and left (minimally displaced) sacroiliac luxation. Given the right-sided ilial fracture it was considered appropriate to stabilize the left sacroiliac joint using an ilio-sacral lag screw (and washer). A lag screw was placed from dorsomedial to ventrolateral to compress the fracture and definitive fixation was achieved with a conventional (veterinary cuttable) plate applied to the dorsal aspect of the ilium with three 2.0 mm screw placed cranial and caudal to the fracture. Note an empty screw hole cranially on the ilium.

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Figure 10.10 Pre-operative and post-operative radiographic projections of the pelvis of a cat with a right ilial fracture with medial displacement and mild comminution (pubic and ischial fractures were also apparent). Fixation of the reduced fracture was with a laterally-applied 2.4 mm locking T-plate (Securos Pax) with three screws caudal to the fracture and three screws cranial to the fracture.

the pelvic canal in cats with obstipation secondary to pelvic malunion. In many cases, continued medical therapy is required after surgery.

Pubic Symphysiotomy

A ventral approach is performed and an osteotome or saw is used to separate the two halves of the pubis. Retractors are used carefully to spread the symphysis and widen the pelvic canal. The sacroiliac joints limit the amount of separation achieved by splitting the pubic symphysis. In canine cadavers a third of cadavers had sacroiliac luxation when the pubis was separated by 50% of the transverse width of the sacrum, so this distance should be measured on pre-operative radiographs and separation should not exceed this width. A cortical graft from the ilium or ulna or a piece of sterile high-density polyethylene can be wired into place between the two halves of the pubic symphysis to maintain separation.

Internal Hemipelvectomy

Portions of the pubis and ischium may be resected, leaving the acetabula, ilia and sacroiliac joints intact. Bilateral resection may be performed if necessary. Alternative procedures (symphysiotomy and pelvic ostectomy) should be considered if repair of the collapsed pelvis requires removal of the acetabulum and ilium, since resection of these structures would necessitate concurrent limb amputation.

Pelvic Ostectomy

In a procedure similar to triple pelvic osteotomy, ilial, pubic and ischial osteotomies are made. The resultant free acetabular segment is displaced laterally and stabilized with a bone plate.

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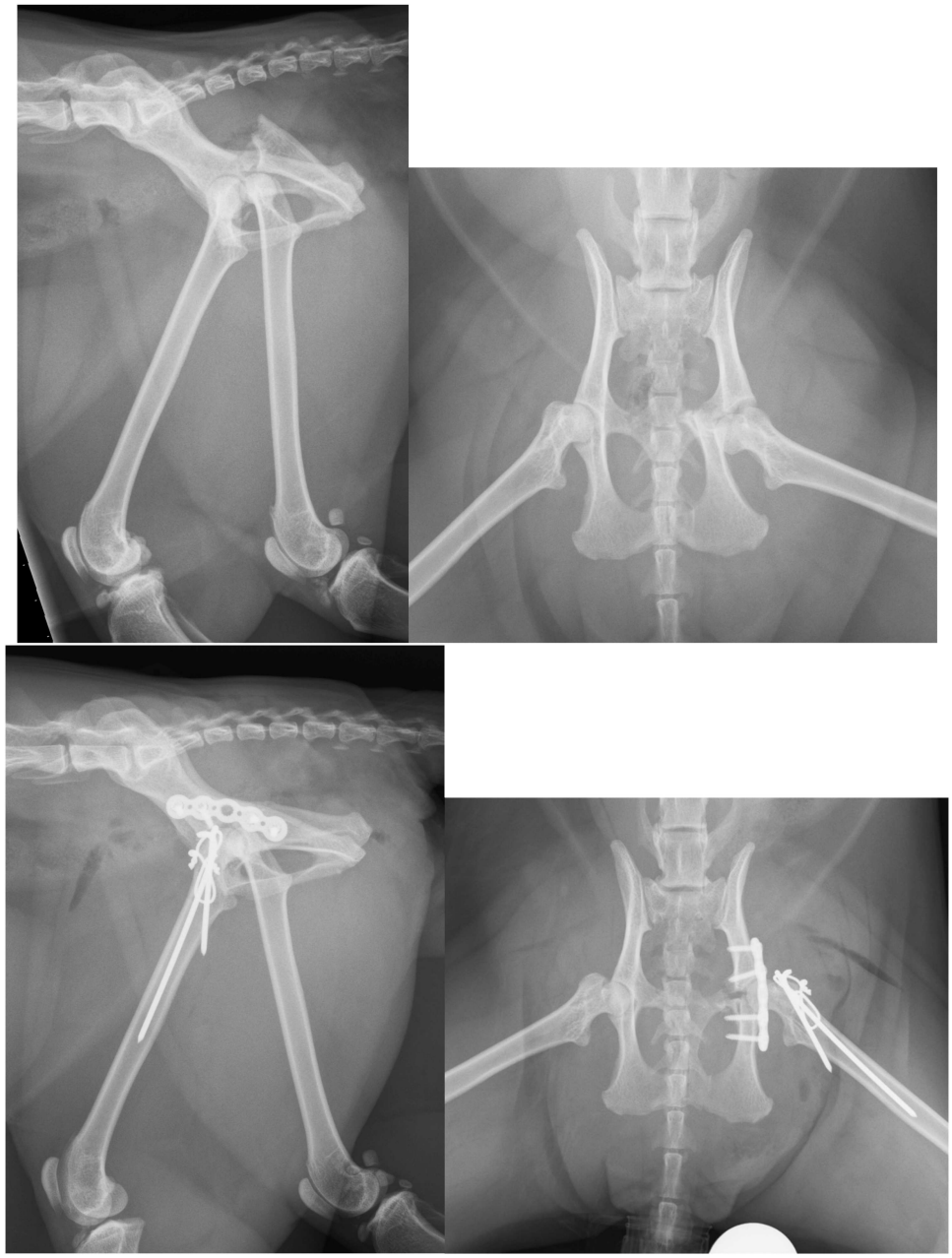


Figure 10.11 Pre-operative and post-operative radiographic projections of a cat with a left acetabular fracture. Approach to the fracture involved a greater trochanteric osteotomy. Fixation of the acetabulum involved a locking reconstruction plate (2.4 mm Securos Pax) contoured to the dorsal acetabulum and applied with two screws cranial and caudal to the fracture. Fixation of the reduced greater trochanter was with two Kirchner wires and a figure-of-eight tension band wire.

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Part 2 Fractures of the Femur

(Aron & Crowe 1987, Fries et al. 1988, Liptak & Simpson 2000).

Femoral fractures occur commonly in cats and are readily diagnosed by palpation and radiography. In almost all cases, surgical intervention is indicated to reduce the fracture properly and provide stability.

Following femoral fracture repair early passive range of motion exercises are encouraged since the stifle, in particular, is prone to post-operative complications. Complications of femoral fracture repair include malunion, delayed union, infection, implant failure, stifle or hip joint stiffness, quadriceps contracture and sciatic nerve injury. Quadriceps contracture is a rare condition which develops after fracture fixation in young cats, in which traumatized muscles become incorporated into an exuberant fracture callus and undergo rapid fibrosis. Successful treatment with a dynamic flexion apparatus in a cat has been reported, but this complication is best avoided through minimally traumatic technique and encouragement of early limb use. Osteoarthritis may develop after surgical repair of articular fractures, although it is rarely severe in cats and is minimized by good surgical technique, anatomic reduction, rigid fixation and early post-operative joint motion.

PROXIMAL FEMORAL FRACTURES:

(Borak et al. 2015, Culvenor et al. 1996, Fischer et al. 2004, Forrest et al. 1999, Jeffery 1989, McNicholas et al. 2002, Queen et al. 1998, Witte et al. 2010).

Femoral Head Fractures

Fractures of the femoral head in cats are rare. Avulsion fractures of the ligament of the femoral head may be recognized as a small fragment of bone in the region of the hip joint alongside coxofemoral luxation (see Chapter 11). Reattachment of avulsion fractures within the acetabulum is not possible, although if the fragment is large it may be prudent to remove it from the ligament and attempt its reduction. Otherwise treatment should involve excision of the ligament of the femoral head alongside the bone fragment and reduction of the hip (typically using a toggle system).

Reduction and Fixation

Large fracture fragments of the articular surface of the femoral head may be reduced and reattached with Kirschner wires (0.6–0.9 mm) which are countersunk beneath the articular surface. If the fragment is not an avulsion fragment and the ligament of the femoral head remains in place, anecdotally Kirschner wires may be placed from the greater trochanter, through the femoral neck to purchase femoral head fragments, however, this is unlikely to be successful with all but the largest fragments, and assessing whether pin tips are protruding into the joint space may be impossible. Luxation of the hip by incising the ligament of the femoral head allows the surgeon exposure to the fractured articular surface but necessitates a coxofemoral reduction technique during closure.

Fragment Removal

Given the small size of some fragments in cats, they may not be reducible/reparable and they are frequently discarded. Depending on the size of the remaining defect, salvage options (femoral head and neck ostectomy and total hip replacement) should be considered, since discomfort associated with the defect in the articular margin may be limiting in the short term and osteoarthritis is inevitable. There are no guidelines to inform us how great a defect in the articular surface may be tolerated and if there is uncertainty, the surgeon should allow 4–6 weeks after fragment excision, when decisions regarding salvage options may be made according to clinical function.

Femoral Capital Physeal Fracture

Femoral capital physeal fractures are common in cats. They may be traumatic or non-traumatic in which case they are often termed 'spontaneous femoral capital physeal fracture'. The latter is more common than the former, so cats often present without a history of a traumatic event, with no concurrent injuries and an insidious onset of lameness. The cause of the femoral capital physeal fracture may be of secondary importance in terms of management, to the radiographic appearance of the remaining femoral neck. Where spontaneous femoral capital physeal fracture is diagnosed, the contralateral hip should also be imaged and monitored over the following months.

Traumatic Femoral Capital Physeal Fracture

Most traumatic femoral capital physeal fractures are Salter–Harris type 1 or type 2 injuries. In chronic cases (i.e. those for which diagnosis has been delayed by a few weeks or months), the radiographic appearance reveals atrophy of the femoral neck, although the femoral head, which remains within the acetabulum, is preserved. The ligament of the femoral

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head has been suggested to contain blood vessels which contribute to the blood supply to the femoral head; however, in dogs, in which there are no such blood vessels within the ligament of the femoral head, atrophy of the femoral head within the acetabulum is not seen in this condition. Where atrophy of the femoral neck is not apparent, traumatic femoral capital physeal fractures respond very well to reduction and internal fixation.

Kirschner wire fixation (Figures 10.13 and 10.14)

1. A craniolateral approach is made to the hip joint. External rotation of the limb allows visualization of the femoral head and femoral neck physeal surfaces.
2. The fracture is reduced and gentle pressure is applied from the lateral aspect of the proximal femur to compress the fracture. The femoral shaft is manipulated to move the hip through a small range of flexion and extension, whilst monitoring the femoral head. If there is sufficient remaining femoral neck, the physeal surfaces will interdigitate such that manipulation of the femur moves the femoral head within the acetabulum. Provided reduction can be performed acceptably, the surgeon proceeds to perform fixation. Otherwise a salvage procedure (femoral head and neck ostectomy or total hip replacement) is considered.
3. Three Kirschner wires are inserted retrograde from the fractured surface of the femoral neck aiming to emerge

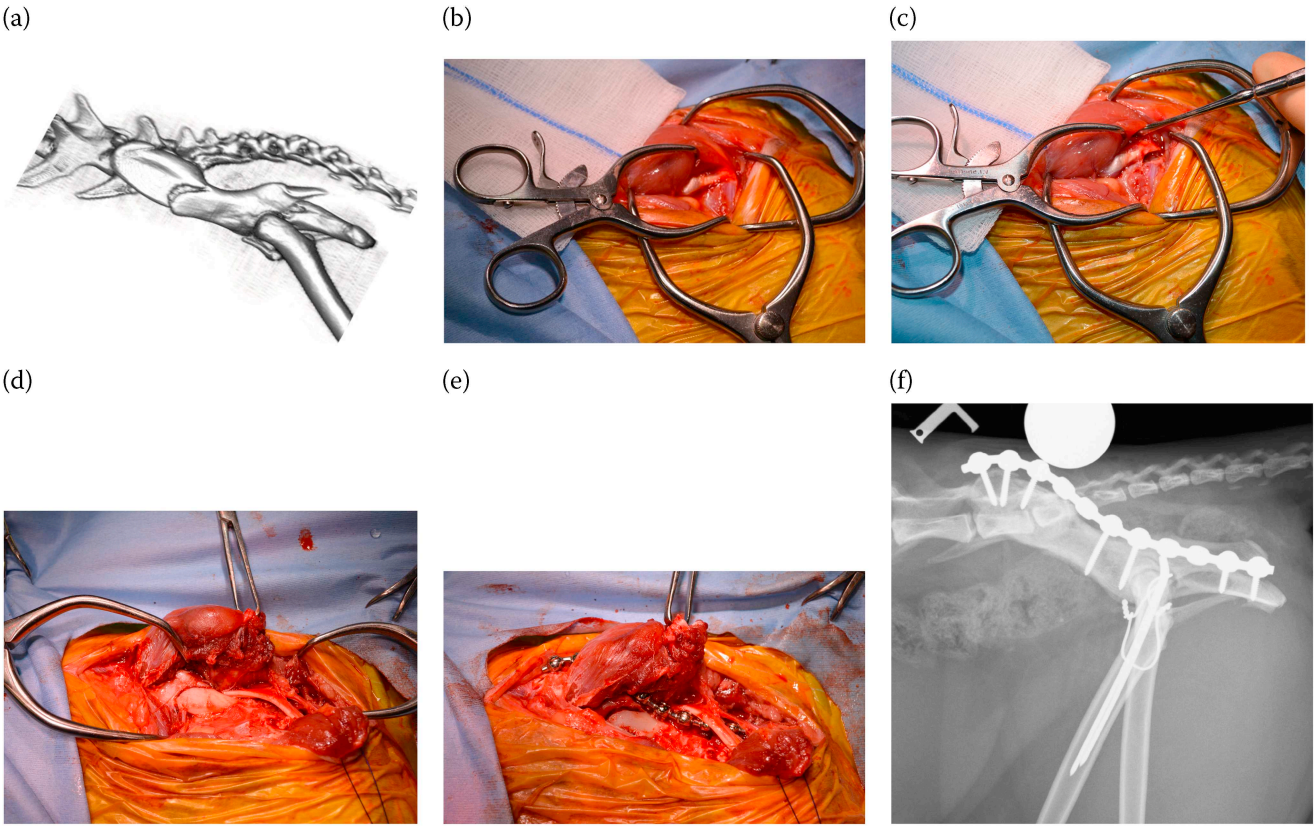


Figure 10.12 Intra-operative photos of an approach to a segmental fracture of the feline pelvis (ipsilateral ilial and acetabular fractures). In the images caudal is to the left, cranial to the right, dorsal to the top. The approach involved a greater trochanteric osteotomy. **(a)** pre-operative CT 3d reconstruction showing fractures of the right ilium and right ischium/caudal acetabulum. **(b)** a transverse incision has been made in the skin parallel to the ilium, starting just caudal to the greater trochanter. The superficial and middle gluteal muscles s are being distracted by the Gelpi distractors to reveal the white tendon of insertion of the deep gluteal muscle (inserting on the greater trochanter). **(b)** An osteotomy has been made across the greater trochanter between the insertion of the deep gluteal tendon and the origin of the vastus lateralis. **(c)** An Alice tissue forceps has been used to apply dorsal traction on the osteotomized greater trochanteric fragment, elevating the gluteal muscles to reveal the ilium and dorsal acetabular rim. Note the sciatic nerve running caudally, which is protected to some extent by the elevated gluteal muscles. The internal obturator and gemelli muscles and tendons may also be used to protect the sciatic nerve more caudally, following elevation from the trochanteric fossa, though they simply been elevated in this case. **(d)** Through further elevation of the middle gluteal the lateral surface of the ilium has been exposed. Caudally the origin of biceps femoris has been elevated to expose the ischium caudal to the acetabulum. **(e)** A malleable locking plate (String-of-pearls, Orthomed) has been contoured to the dorsal surface of the ilium and ischium to bridge both fractures. **(f)** A post-operative lateral radiographic view of the pelvis demonstrates adequate implant-positioning on the ilium and ischium and to apply fixation to the reduced greater trochanter. (Two Kirschner wires and a figure-of-eight tension band wire passed through a bone tunnel in the lateral femoral cortex)

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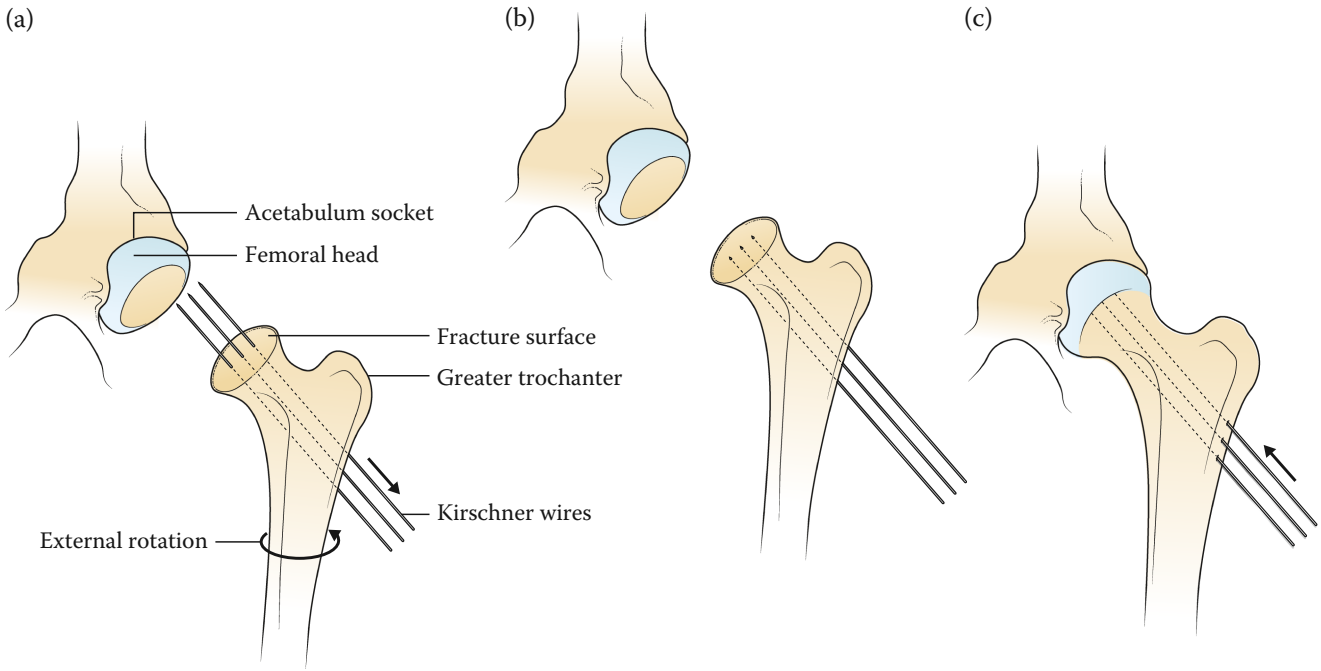


Figure 10.13 Stabilization of femoral neck fractures. **(a)** The femur is externally rotated to allow visualization of the fractured surface of the femoral neck. Two or three Kirschner wires are introduced into the femoral neck to emerge from the lateral cortex of the femur at the level of the third trochanter. **(b)** The Kirschner wires are withdrawn from the lateral aspect of the femur until their tips are flush with the fracture surface. **(c)** The fracture is reduced and the Kirschner wires are driven a short distance into the femoral head. (Without penetrating the articular surface)

laterally just distal to the greater trochanter. They are pulled backwards until their tips are flush with the fractured surface. (alternatively, pins may be advanced normograde from just distal to the greater trochanter, however, aiming must be accurate).

4. The fracture is reduced again and the femur manipulated to ensure adequate reduction.
5. The Kirschner wires are advanced into the femoral capital epiphysis (a few mm). The joint is manipulated through a range of motion and the articular surface is palpated with a smooth periosteal elevator to ensure the wires do not penetrate the cartilage of the epiphysis.
6. The Kirschner wires are bent against the lateral femoral cortex to reduce the risk of soft tissue trauma and migration.

Whether Kirschner wires should be placed parallel or converging/diverging remains controversial. While parallel Kirschner wires theoretically permit interfragmentary compression resulting from contraction of the gluteal muscles, converging/diverging Kirschner wires may provide stiffer fixation across the fracture. Provided three Kirschner wires are placed bridging the fracture, their orientation with respect to one another is probably of little consequence.

The prognosis following internal fixation is good, although occasionally the femoral neck continues to undergo atrophy in which case lameness may remain, necessitating revision to femoral head and neck ostectomy or total hip replacement.

Spontaneous Femoral Capital Physeal Fracture

This condition has also referred to as slipped capital epiphysis and capital physeal dysplasia syndrome. The existence of a condition termed 'femoral neck metaphyseal osteopathy' remains controversial, since the described appearance is similar to chronic femoral capital physeal fractures, and it probably represents a particular stage within the disease process. Spontaneous femoral capital physeal fractures are typically seen in heavier, neutered males, although any cat up to the age of approximately 3 years may be a candidate for this condition, particularly if delayed physeal closure is apparent at other sites. Maine coons may be overrepresented. In one study 7/17 cats were affected bilaterally.

The diagnosis can be difficult, since cats do not necessarily resent hip manipulation. Radiographs may reveal minimal or no displacement at the physis so radiography should include views of the pelvis with hips in frog-legged and extended positions, in addition to caudo-cranial and mediolateral views to avoid missing the fracture.

Treatment of spontaneous femoral growth plate fractures depends on the radiographic appearance. Provided sufficient femoral neck remains at the time of diagnosis, internal fixation as described for traumatic femoral capital physeal fractures, should be considered. Where femoral neck atrophy is significant, femoral head and neck ostectomy and total hip replacement should be considered. There is no published evidence for the natural progression of conservatively managed femoral capital physeal fractures; however, anecdotally, cats may attain function levels similar to

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Figure 10.14 Mediolateral and caudocranial views of the left femur of a cat with a traumatic femoral neck fracture. Fixation was performed using three parallel Kirschner wires placed retrograde to bridge the fracture. Pins were bent over at the level of the third trochanter to avoid irritation of the soft tissues.

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that which might be expected from femoral head and neck ostectomy, presumably once sufficient femoral neck atrophy has occurred. However, this approach is not recommended, since lameness may continue for several months, or indefinitely.

Femoral Neck Fractures

Cause and Pathogenesis

Fractures of the femoral neck are rare in cats. They are treated as for femoral capital physeal fractures, with Kirchner wire internal fixation or salvage (femoral head and neck ostectomy or total hip replacement).

Greater Trochanteric Fractures

Cause and Pathogenesis

Avulsion fracture of the greater trochanter may occur in isolation but is frequently found concurrent with coxofemoral luxation and/or traumatic femoral capital physeal fracture (Figure 10.15). Ipsilateral greater trochanteric fracture is seen in approximately one fifth of cats with traumatic femoral neck fracture. The greater trochanter is the site of deep and middle gluteal muscle insertion, and should not be excised. The condition may be difficult to identify radiographically, since the greater trochanteric fragment often displaces only mildly to overlap the proximal femur, but it may have serious consequences for limb use and should be recognized and addressed where possible.

Treatment of greater trochanteric avulsion includes open reduction and internal stabilization. Since this is not an articular fracture, the prognosis is excellent.

1. A craniolateral approach is made to the greater trochanter. (The hip joint is not exposed unless open reduction of coxofemoral luxation or fixation of a femoral capital physeal fracture is required).
2. The greater trochanter is reduced and temporarily stabilized with reduction forceps.
3. Fixation involves directing two Kirschner wires from the proximo-lateral aspect of the greater trochanter distally into the medullary cavity of the femur. The Kirschner wires may engage the femoral cortex, or may be driven distally until they engage the bone of the distal femoral metaphysis.
4. A bone tunnel is drilled in the lateral femoral cortex from cranial to caudal (protecting the sciatic nerve) distal to the third trochanter.
5. Orthopaedic wire is passed through the bone tunnel and looped in a figure-of-eight pattern over one or both of the Kirschner wires. The wire is tightened. This wire counters the distracting force of gluteal muscle contraction acting on the greater trochanter. The Kirschner wires are bent over flush with the bone and cut short.

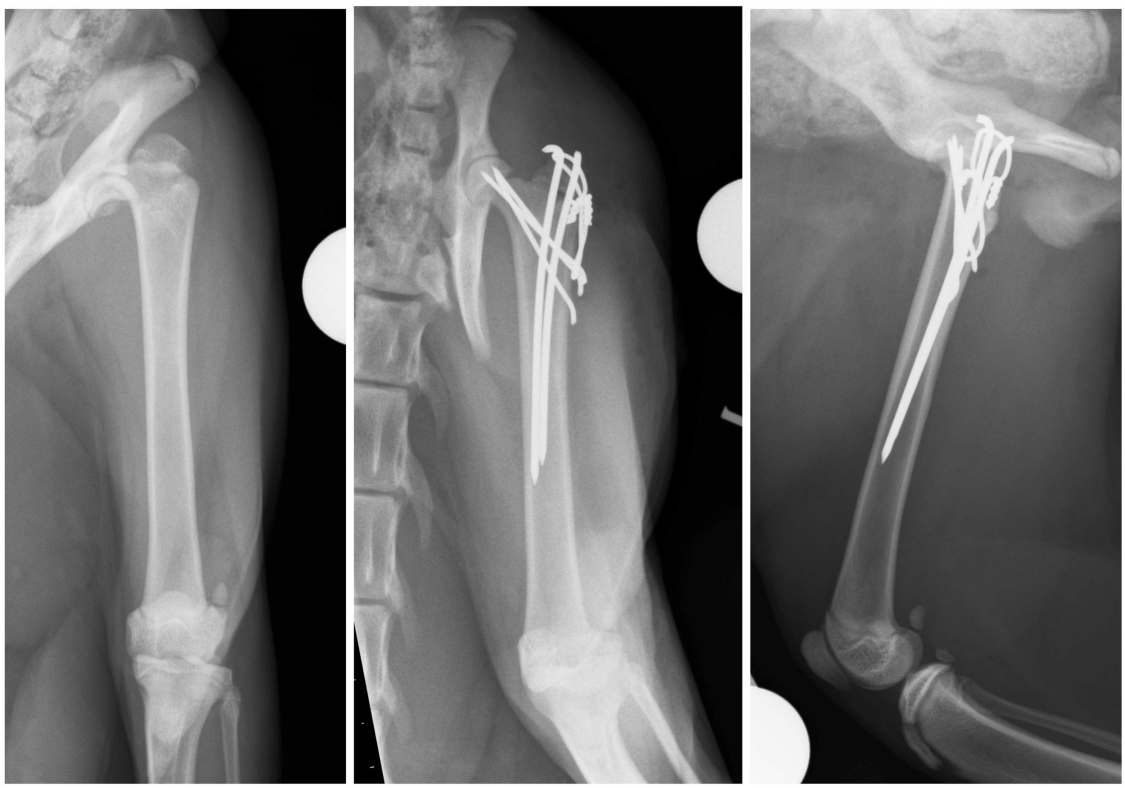


Figure 10.15 Traumatic femoral capital physeal fractures and greater trochanteric avulsion fracture are commonly associated with one another. Caudocranial pre-operative and post-operative views and mediolateral view of the femur with this combination of injuries are shown. Fixation of the femoral capital physeal fracture was with three Kirschner wires. The greater trochanteric avulsion was reduced and fixation was with two Kirschner wires and a figure-of-eight tension band wire. (Looped proximally over one of the pins bridging the greater trochanteric fracture and distally over one of the pins maintaining the femoral neck fracture)

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DIAPHYSEAL FEMORAL FRACTURES:

(Bruckner et al. 2014, Dallman & Martin 1990, Diaz-Bertrana et al. 2005, Duhautois 2003, Gibson & van Ee 1991, Harari 2002, Hulse et al. 1997, Larin et al. 2001, McLaughlin et al. 1992, McLaughlin & Roush 1999b, 1999a, Nabholz et al. 2019, Newman & Milton 1989, Palmer et al. 1988, Peirone et al. 2002, Reems et al. 2003, Scotti et al. 2007, Vassuer et al. 1984).

Repair of fractures of the femoral diaphysis with open or closed reduction and rigid stabilization is recommended. Malunion and non-union frequently follow management of femoral fractures by external coaptation due to the proximity of the body and the amount of muscle surrounding the bone.

A lateral surgical approach is relatively simple. A craniolateral skin/fascia incision is made from the greater trochanter to the patella. The fascia lata is incised along the cranial edges of the biceps femoris muscle and the biceps is retracted caudally exposing the femur. Vastus lateralis muscle is retracted cranially. Care is taken to preserve the soft tissue attachments to the femur and bone fragments wherever possible during reduction. The femur also lends itself very well to minimally invasive percutaneous osteosynthesis.

Intramedullary pins and cerclage wires, external skeletal fixators, bone plates and screws, interlocking nails (ILNs), or combinations of these methods can be applied for femoral diaphyseal fracture management in cats. A brief description of each method as it applies to the femoral diaphysis is given, followed by a discussion of options for various common femoral fracture configurations.

Intramedullary Pinning

Intramedullary pin placement should not be considered a stand-alone technique for fracture fixation, since rotational forces are not controlled, and axial forces are only resisted by the friction that may be generated proximally in cortical bone and distally in cancellous bone. Intramedullary pins may be combined with cerclage wires (in the case of oblique fractures), external skeletal fixators and plates and screws to control the remaining forces on a fracture femoral diaphysis. ILNs are a form of intramedullary pin in which the pin is attached to the bone's cortex at various levels.

Where the pin is to be combined with cerclage wires it should be of a diameter to fill 60–70% of the medullary cavity at its narrowest point (isthmus). Otherwise it should fill 30–40% of the medullary cavity, leaving space for application of other trans-cortical implants (such as plate screws).

Normograde pin insertion is preferred because it ensures proper placement of the pin in the trochanteric fossa and may reduce the possibility of injury to the sciatic nerve (Figure 10.16).

1. An approach is made to the greater trochanter and extended dorsally. The gluteal musculature is immediately apparent. Efforts may be made to retract the superficial and middle gluteal muscles caudally and to incise the tendon of insertion of the deep gluteal muscle; however, pin placement directly through these muscles may be less traumatic and is quite reliable with experience.
2. The pin is started at the medial aspect of the greater trochanter in the trochanteric fossa. Bone holding forceps applied from lateral to the proximal femur just distal to the greater trochanter will assist appropriate directing of the pin as it penetrates the proximal cortex.
3. The pin is driven distally to the fracture. If an open approach has been made to the fracture the pin can be observed entering the distal fragment. If no approach has been made then insertion of the pin into the distal fragment is confirmed by palpation of using intraoperative imaging (fluoroscopy).
4. The pin is driven distally until it is seated in the distal fragment without entering the stifle joint. The stifle joint is manipulated through a range of motion to ensure the pin has not penetrated the joint. A second pin of identical length can be used to measure how far distally the pin has been inserted. It should be seated as distally as possible to crush the metaphyseal cancellous trabeculae and maximize the friction with which the pin is purchased in the distal fragment.

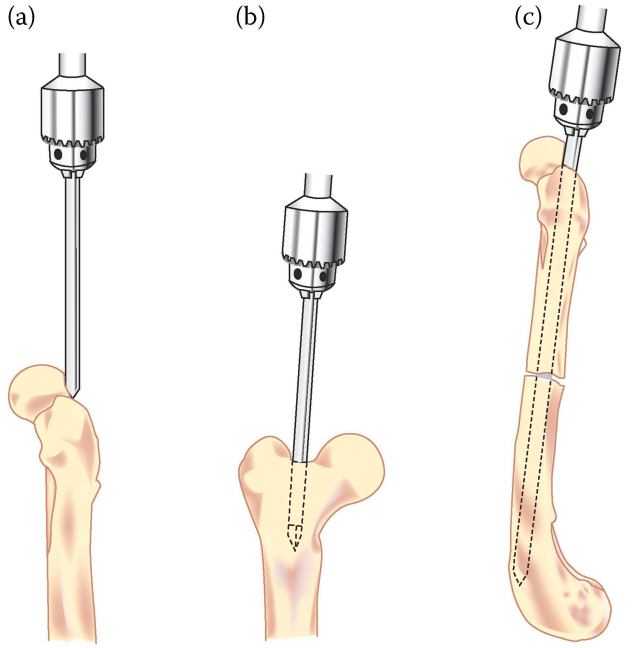


Figure 10.16 Normograde intramedullary pin placement in the feline femur. A pin large enough to fill 60–70% of the medullary cavity is selected. **(a, b)** Pin insertion begins in the trochanteric fossa. **(c)** The pin is driven to the level of the fracture, the fracture is reduced and the pin is then driven further into the distal segment of the bone. The pin should be advanced into the metaphyseal bone of the distal femur, but penetration of the articular surface should be avoided. Retrograde pin insertion is to be avoided, as a result of the risk of sciatic nerve trauma during the initial insertion of the pin. If retrograde femoral intramedullary pinning is to be considered, the hip should extended to protect the sciatic nerve.

Intramedullary pins may also be placed retrograde, although this should be avoided to limit the risk of iatrogenic sciatic nerve trauma. The intramedullary pin is directed proximally from the fracture site, emerging proximally until the distal tip is flush with the fracture. The hip should be extended somewhat and the femur adducted to minimize the risk of impingement of the sciatic nerve as the pin emerges. The fracture is reduced and the pin is pushed distally to seat in the distal metaphysis.

Stack pinning (placing several pins into the medullary cavity) does not completely prevent rotational forces and should be avoided, although it has been used with success in cats.

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Bone Plates and Screws

Bone plates are commonly used to stabilize femoral fractures in cats. They may be applied to the lateral (tension) surface of the femur through a lateral approach. The medial surface and cranial surface (proximally to avoid interference with the function of the patella) also lend themselves to plate application, if these are preferable. Long plates are generally selected such that they do not terminate in the diaphysis (where they will act as stress risers and may precipitate a new fracture) (Figure 10.17).

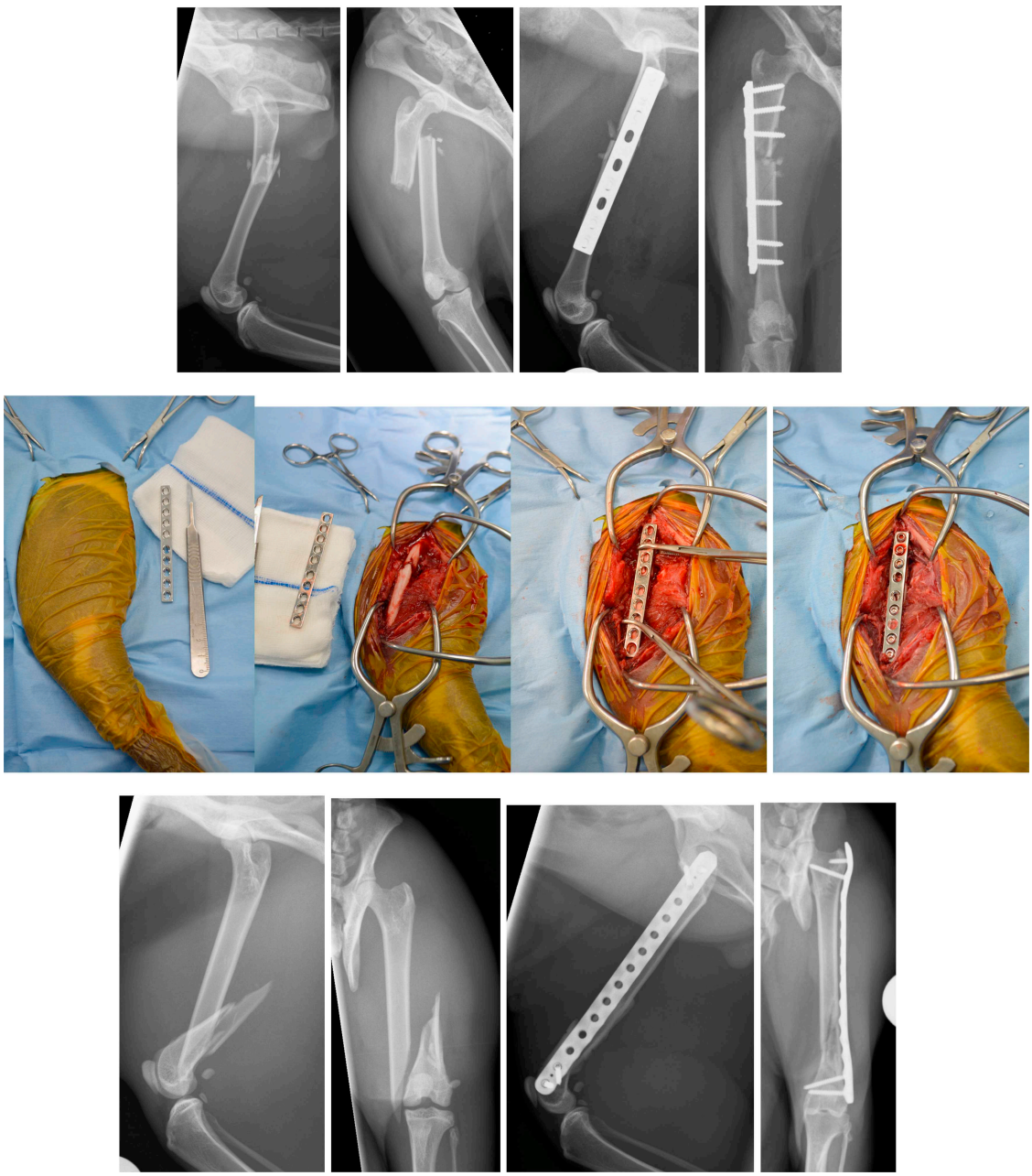


Figure 10.17 Examples of femoral fracture repair using plates and screws. Top: conventional screws in a dynamic compression plate to give fixation to a mildly comminuted transverse proximal femoral diaphyseal fracture. Comminution resulted in a failure to reconstruct the bone accurately, and interfragmentary compression was not possible (in addition a fissure is apparent extending from the fracture into the distal femoral fragment, increasing the risk of applying compression using the dynamic compression function of the plate). The plate and screws are large (2.7 mm). Middle: intra-operative photographs of left femoral fracture open reduction and fixation using a 2.7 mm dynamic compression plate (same case as in top row) From left to right: the limb is clipped and prepared for surgery, an iodine impregnated adhesive drape is applied and the draping isolates the limb to include the proximal femur such that the entire limb can be manipulated. A lateral approach is made to the femur caudal to the vastus lateralis muscle and the fracture is reduced. A dynamic compression plate is contoured and applied to the lateral aspect of the femur using mini bone holding forceps proximally and distally. Screws are placed as appropriate (in this case of a conventional plate, three bicortical screws proximal and distal to the fracture are appropriate). Bottom: a distal third femoral fracture with comminution was stabilized with a long 2.4 mm locking straight plate (Securos, Pax) with screws placed proximally and distally at the metaphyses of the bone. Surgery was performed with minimally invasive technique, with fracture reduction confirmed by percutaneous palpation.

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External Skeletal Fixators

A type I external skeletal fixator may be applied to the lateral aspect of the femur. Transfixation pins should be placed proximally in the region of the palpable greater and third trochanters (avoiding directing pins from caudal to minimize the risk to the sciatic nerve) and distally where the lateral femoral condyle and distal femur are relatively superficial. The external skeletal fixator frame can incorporate the proximal portion of an intramedullary pin which is bent laterally where it emerges from the trochanteric fossa (so-called 'tie-in' configuration).

Some cats will be reluctant to use the limb for a time after application of an external fixator to the femur, presumably because of the pins penetrating large muscles.

Interlocking Nails

The straight feline femur lends itself well to fracture fixation using ILNs. The nail is typically placed normograde from proximal, although placement of an ILN retrograde from the intercondylar notch has been described (with the risk of sciatic nerve trauma innate to retrograde intramedullary pinning of the femur). ILNs are excellent at providing bridging function but cannot provide interfragmentary compression (Figure 10.18).

Reconstructable Fractures

Reconstructable fractures are typically two-piece fractures with no comminution. They may be transverse or oblique. Since the bony column can be accurately reconstructed in these cases, a load sharing construct with the implants may be achievable. If interfragmentary compression is not achieved, a narrow fracture gap will be present, where, unless very stiff fixation is applied, fracture strain will be high, resulting in delayed bone healing. Therefore, it may be appropriate to perform open reduction, with the aim of generating interfragmentary compression and promoting direct bone union.

Since direct bone healing necessitates stiff fixation, a good option is the use of plates and screws. Dynamic compression plates of 2.0 mm, 2.4 mm or 2.7 mm size are appropriate for transverse fractures. In oblique fractures it may be possible to place one of the plate screws obliquely across the fracture, to act as a lag screw.

It is possible to combine plate and screw fixation with intramedullary pins and/or lag screws. Figure 10.17, Figure 10.18

The combination of a plate and an intramedullary pin is known as plate-rod fixation (Figure 10.19). The purpose of the intramedullary pin in a plate-rod combination is to



Figure 10.18 Interlocking nails used to address feline femoral fractures. **Top row:** transverse fracture of the distal third of the femur, fixation with nail-in-bolt interlocking nail (Targon Nail, BBraun) and fixation with bolt-in-nail interlocking nail (Innovative Animal Products). **Bottom row:** moderately comminuted fracture of the proximal third of the femur. Note the capacity to direct bolts medio-lateral or cranio-caudal as necessary. (The proximal bolt was placed from caudal to cranial to purchase healthier bone unaffected by fissuring)

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Figure 10.19 Plate-rod fixation of comminuted femoral fractures using a combination of an intramedullary pin which fills approximately 30–40% of the medullary cavity and a 2.4 mm reconstruction polyaxial locking plate (Securos Pax). In comminuted fractures reconstruction of the bone is not possible, so the implant-bone construct will not be load-sharing. Implants are acting in bridging mode. The addition of the intramedullary pin reduces strain on the plate and screws. In addition, in severely comminuted femoral fractures the insertion of the intramedullary pin restores bone length and alignment of the proximal and distal bone segments. The torsional alignment is adjusted prior to placement of the plate. **Top row:** Central segments of bone may be purchased by fixation with screws from the plate. **Bottom row:** Plate-rod technique lends itself to minimally invasive technique, with screws only placed at the proximal and distal extents of the bone.

reduce the strain on the plate and screws and increase the stiffness of the construct overall. By placing the intramedullary pin first the surgeon can restore the bone's length and long axis and need only consider the torsional alignment of the bone before applying the plate and screws.

In oblique reconstructable fractures lag screws may be combined with plate and screw fixation. The fracture is reduced and one or two lag screws are placed to apply compression. An attempt should be made to avoid placing screws where the plate is intended to be placed. The plate in this situation is in neutralization mode.

Fixation of reconstructable fractures using a lateral type 1 external skeletal fixator or ILNs are reasonable options, though primary bone healing will not be achieved except where an open approach is made to apply cerclage wire or lag screws to oblique fractures. As such, fracture strain in the narrow fracture gap will be high, and healing will be delayed. Non-union has been reported in simple transverse femoral fractures stabilized using ILNs.

Non-Reconstructable Fractures

Where comminution is severe, reconstruction of the bony column is not attempted. Fixation may be applied via an open approach, but minimally invasive percutaneous osteosynthesis should be considered. Intramedullary fixation is useful to lengthen the bone to its appropriate length, as

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measured from radiographs of the intact contralateral bone. This can be performed with a closed technique. The axial location of the intramedullary pin is also beneficial, since the implant is placed in the neutral axis of the construct, where forces are minimal. This is of benefit in the case of a non-reconstructed fracture where the implants are to take all the force of loading until bone healing has occurred. Adjunctive fixation is required to address torsional malalignment and forces. This may involve external skeletal fixation (including a tied-in intramedullary pin) or plate application by minimally invasive technique (plate-rod technique). Short proximal and distal approaches are made to the lateral aspect of the bone connected by an epiperiosteal bone tunnel through which the plate is slid. Screws are applied through these short approaches or through additional stab incisions.

An ILN is a good alternative to an intramedullary pin, sharing all the benefits of intramedullary pinning but also addressing torsional forces acting on to the bone.

Where reduction of the fracture is not present to guide assessment of torsional alignment, this can be assessed in a number of ways in the femur:

1. The linea aspera on the caudolateral aspect of the femur can be a useful landmark;
2. If short approaches to the stifle and femoral neck are performed, the trochlear ridges may be visualized to confirm a true lateral-to-medial view of the distal femur and the femoral head/neck may be visualized projecting somewhat cranial to the proximal femoral shaft to confirm a true lateral-to-medial view of the proximal femur;
3. A correctly reduced femur should demonstrate approximately 90° of external rotation at the hip and 45° of internal rotation.

DISTAL FEMORAL FRACTURES:

(Berg et al. 1984, Carmichael et al. 1989, Chico et al. 2001, Francis & Johnson 1990, Franczuszki et al. 1986, Frydman et al. 2014, Graver et al. 1981, Hardie & Chambers 1984, Lewis et al. 1993, Milton et al. 1980, Mize 1989, Parker & Bloomberg 1984, Prieur 1989, Sumner-Smith & Dingwall 1973, Whitney & Schrader 1987).

Distal femoral fractures may be of the distal femoral physis, supracondylar, trochlear or condylar.

External coaptation is ineffective in stabilizing most distal femoral fractures because the distal fragment is typically displaced caudally by muscular attachments. Open reduction and internal fixation are imperative for fractures affecting the articular surfaces and are recommended for most peri-articular fractures to avoid the production of callus, which may interfere with normal joint function. If an articular fracture is irreparable or severe complications develop, limb amputation or stifle arthrodesis may be indicated.

With distal femoral fractures, the cruciate and collateral ligaments (and other soft tissue support structures of the joint) may be damaged as well, requiring additional treatment to re-establish joint function.

Fractures of the distal femoral growth plate

Salter-Harris types 1 or 2 distal femoral physeal fractures are common in immature cats. They also occur with some frequency in young adult cats and may result from late closure of the growth plate after early neutering. Treatment involves open reduction and fixation with cross pins (Figure 10.20) or rush pins.

Cross Pinning

1. A lateral parapatellar approach is made to the stifle and distal femur
2. The patella is reflected medially to visualize the distal femur (the cranial margin of the fracture lies at the osteochondral junction of the trochlea and femoral diaphysis).
3. The epiphyseal segment, which is typically displaced caudally and proximally, is pulled distally to achieve reduction. A periosteal elevator placed cranially into the fracture gap may be used to lever the fragment into place. The most common intra-operative complication is under-reduction. The physeal surfaces should interdigitate well when reduction is appropriate.

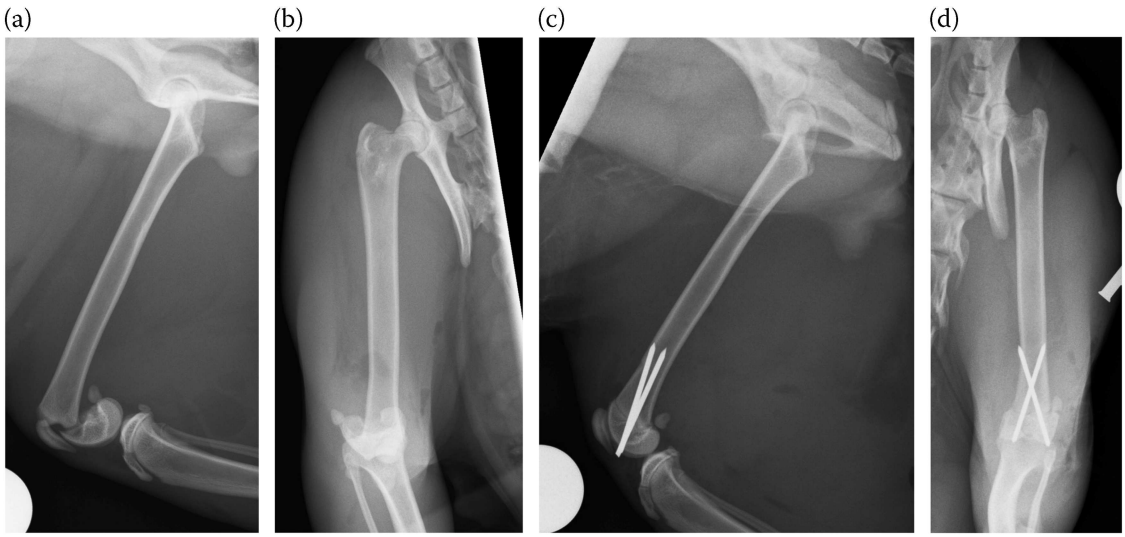


Figure 10.20 Cross pinning to address a Salter-Harris type 1 distal femoral physeal fracture. **(a** and **b)** pre-operative mediolateral and caudocranial views, **(c** and **d)** post-operative mediolateral and caudocranial views.

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4. A cis cortex hole is drilled in the cranial cortex of the femoral diaphysis proximal to the fracture to allow placement of a pointed reduction forceps with one point in the hole and the other in the intercondylar notch, maintaining reduction.
5. Smooth pins are inserted in a cross-pin fashion from the medial and lateral points of intersection between the condyles and trochlear ridges. The pins should be directed to exit the femur approximately 2 cm proximal to the fracture.

Studies have found that mild growth disturbance is common after repair of Salter–Harris type I and II fractures of the distal femur. However, clinical signs of functional impairment are rare, and this injury carries a very good prognosis with surgery involving good reduction of the fracture. Complications such as malalignment, implant failure, nonunion and patellar luxation have been reported.

Where comminution is present, such that reduction is poor, or where injuries affect the other limbs and greater stiffness is required in the repair, distal femoral physeal fractures may be bridged with short lateral plates. In particular, reconstruction-type plates permit contouring to fit the cranial bowing of the condyle such that at least two screws may be placed in the epiphyseal fragment (Figure 10.21).

Rush Pinning

Dynamic intramedullary cross-pinning is discussed frequently and may be used in very young animals with the potential for significant further growth to address distal femoral physeal fractures. Kirschner wires inserted in the dynamic intramedullary cross-pinning technique achieve three-point fixation that provides rotational and axial stability.

1. A lateral parapatellar approach is made to the stifle and distal femur
2. The patella may be reflected laterally to visualize the distal femur (the cranial margin of the fracture lies at the osteo-chondral junction of the trochlea and femoral diaphysis).
3. The epiphyseal segment, which is typically displaced caudally and proximally, is pulled distally to achieve reduction. A periosteal elevator placed cranially into the fracture gap may be used to lever the fragment into place. The most common intra-operative complication is under-reduction. The physeal surfaces should interdigitate well when reduction is appropriate.
4. A cis cortex hole is drilled in the cranial cortex of the femoral diaphysis proximal to the fracture to allow placement of a pointed reduction forceps with one point in the hole and the other in the intercondylar notch, maintaining reduction.
5. Smooth pins are inserted from the medial and lateral points of intersection between the condyles and trochlear ridges. The pins are directed at a 10°–15° angle to the median plane of the femur and alternately pushed up the femoral shaft, directing them toward the greater trochanter, glancing off the femoral cortex, rather than penetrating it. During insertion, the hand-chuck is only minimally rotated to prevent the pins from penetrating the cranial femoral cortex. If desired, the pins can be alternately tapped into place with a small mallet rather than inserted with the hand-chuck. Another means of assuring the pins pass up the medullary cavity and do not penetrate the femoral cortex is to blunt their tips prior to insertion. The pin tracts in the epiphyseal

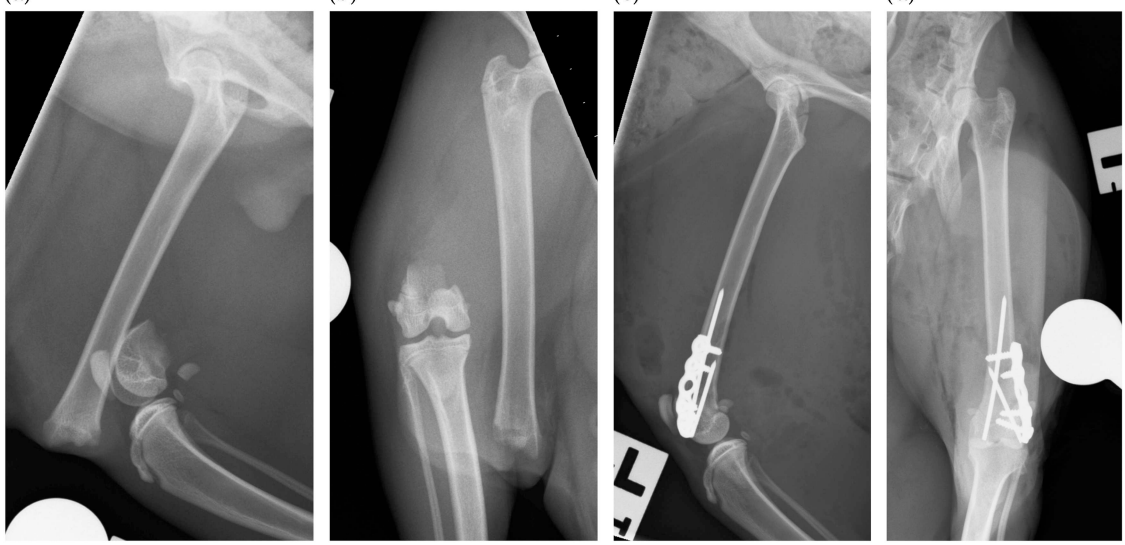


Figure 10.21 Cross pinning may be augmented with a short lateral plate (preferably with a low profile to avoid significant interference with peri-articular soft tissues). **(a, b)** pre-operative mediolateral and caudocranial views of a Salter-Harris type 2 distal femoral physeal fracture in a cat with concurrent contralateral forelimb injuries (indicating increased strength and stiffness in the repair of the femoral fracture). **(c, d)** post-operative mediolateral and caudocranial views of the femur revealing initial reduction and fixation with crossed pins and augmentation of the repair using a lateral 5-hole reconstruction-style veterinary cuttable plate with two screws in the diaphysis and two screws in the distal metaphysis.

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segment may be predrilled using a small drill bit. The pins are seated into the proximal femoral metaphysis.

Supracondylar Fractures

Supracondylar fractures in adult cats are typically transverse or short oblique and do not involve the articular surface. Fixation can involve the pinning techniques described for Salter–Harris fractures, or if sufficient bone stock is available, a bone plate and screws is placed through a lateral approach to the femur combined with a para-patellar approach to the stifle. Veterinary cuttable plates, dynamic compression plates and reconstruction-type plates have been used for repair of distal femoral fractures in cats. Locking implants may be superior in the soft distal epiphyseal and metaphyseal bone of kittens, particularly where space only allows for placement of two screws.

Condylar and Trochlear Fractures

Condylar and trochlear fractures involve the articular surface of the distal femur. Fractures involving a single condyle are termed unicondylar. The medial condyle is most commonly fractured, becoming separated from both the femoral shaft and the lateral condyle. Bicondylar fractures occur when the medial and lateral condyles separate and each is fractured from the femoral shaft. These injuries have been termed 'Y' or 'T' fractures. Concurrent damage to the cruciate ligaments or menisci is not uncommon, though they often remain attached to their relevant sites of origin and insertion. Fractures of the femoral trochlea (the articulating surface for the patella) may occur in conjunction with condylar fractures, in which case they may be longitudinal intercondylar fractures, or they can be transverse or short oblique.

Diagnosis of these injuries can be through plain radiography alone; however, given their potential for complexity and comminution, computed tomography may prove useful for planning.

Internal reduction and stiff fixation are imperative to restore the articular surface and achieve direct bone healing, avoiding callus formation which may disrupt joint function. A parapatellar incision is used and can be extended into a lateral approach to the femur to improve exposure of the supracondylar region. An arthrotomy allows visualization of the intraarticular fractures.

Screws (1.0–2.7 mm) and Kirschner wires are placed to stabilize the fracture fragments. Good anatomic alignment is essential and gaps or steps in the articular surface should be avoided. In a study of experimentally induced unicondylar fractures, lack of interfragmentary compression was implicated in fracture instability and delayed union, osteophyte formation and cartilage erosion.

Post-operatively, passive range-of-motion exercises are encouraged to maintain flexibility. In a study of seven cats operated on for repair of trochlear fractures, 85% had good functional recovery.

Unicondylar Fractures

Lag screws may be placed obliquely or transversely from the fractured condyle into the femoral metaphysis. An anti-rotational Kirschner wire should be placed in combination with the lag screw (Figure 10.22). Lateral or medial plates may be applied in neutralization mode following application of other implants or in buttress mode (Figure 10.21).

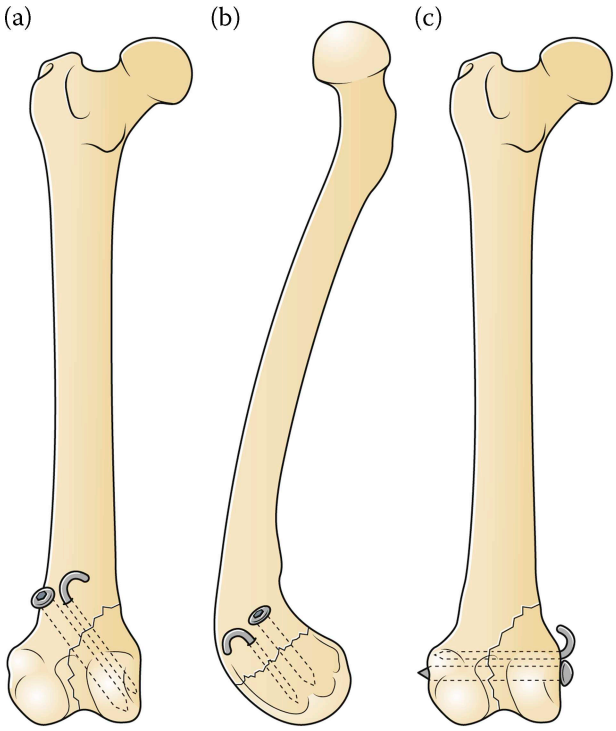


Figure 10.22 Stabilization of unicondylar fractures of the distal femur. Diagrams of femur viewed from cranial **(a)** and from lateral **(b)** showing a lag screw placed to cross a unicondylar fracture with a parallel Kirschner wire placed to prevent rotation of the condylar fragment around the screw. **(c)** view from cranial showing a combination of lag screw and anti-rotational Kirschner wire placed transverse, rather than obliquely.

Bicondylar Fractures

Surgical treatment of bicondylar fractures requires repair of both the intercondylar fracture and the supracondylar portion of the femur. Depending on the conformation, the two condyles may be reduced and compressed using lag screw(s) and/or Kirschner wires, or one condyle (usually the larger fragment) may be reduced to the femoral diaphyseal fragment, prior to reduction and fixation of the other condyle. Accurate reduction of the joint surface is the most important feature of the reconstruction, to minimize future osteoarthritis. As for unicondylar fractures, neutralization or buttress plates should be considered (Figure 10.23).

Trochlear Fractures

Transverse or short oblique fractures of the trochlea are articular. Therefore, internal fixation is essential. Reduction should be as accurate as possible and small osteochondral fragments may be pinned with Kirschner wires to the underlying subchondral bone. The wires are countersunk below the articular surface. Those fragments that are too small to stabilize are discarded. As for condylar fractures, lag screws and Kirschner wires are used as necessary and neutralization or buttress plates applied to give stiffness to the repair.

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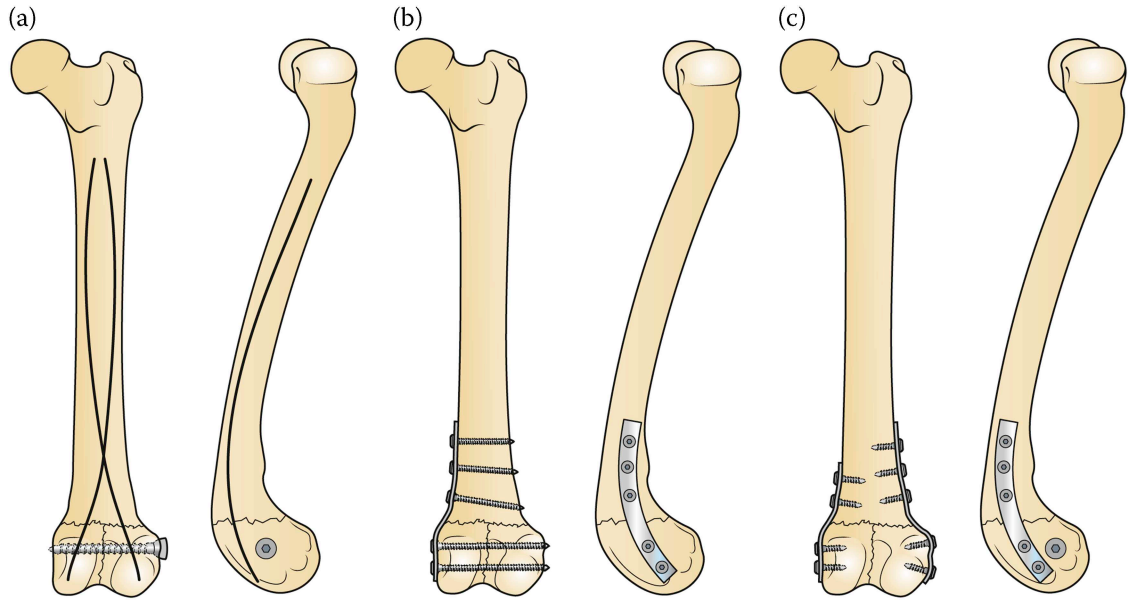


Figure 10.23 Bicondylar fractures of the distal femur necessitate intercondylar compression, which is typically achieved with a transcondylar lag screw. Additional fixation should be robust, since such fractures are innately unstable and often associated with supracondylar comminution. Diagrams of the femur viewed from cranial and lateral demonstrate methods of fixation for bicondylar fracture of the femoral condyle. **(a)** A transcondylar lag screw produces interfragmentary compression and Rush pins are used for fixation of the condyle to the diaphysis. **(b)** A lateral plate is contoured and applied with one or two of the plate screws applied in lag fashion. An alternative technique involves placing a transcondylar lag screw initially, followed by fixation of the reduced condyle to the diaphysis using a lateral plate. **(c)** Plates are applied medially and laterally to bridge both fractures. If locking plates are applied the screws may be unicondylar. It is appropriate to achieve intercondylar compression using a lag screw which may be placed through a plate, provided it is not locking.

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Part 3 Patellar Fractures

(Arnbjerg & Bindseil 1994, Gibert et al. 2016, Harari et al. 1990).

Patellar fractures are uncommon. They occur through trauma or as insufficiency fractures as part of a condition currently reported as 'knee and teeth syndrome' (KATS), or 'patella and dental anomaly syndrome' (PADS). Retention of immature dentition is commonly associated with this condition and the teeth should be checked in cats with patellar fractures (Figure 10.24). Trauma may involve application of direct force to the patella or extreme traction applied by the quadriceps mechanism with the stifle in extension during a fall, however, this is rare. Patellar fractures are typically closed and usually involve the articular surface of the patella. Comminuted, longitudinal and transverse fractures of the patella can occur and some are minimally displaced. Transverse fractures are the most common type.

CLINICAL SIGNS:

Cats with patellar fractures are initially non-weight-bearing. There may be a history of a trauma. Over the course of a few days the lameness reduces somewhat, but resolution of lameness is not likely. The patella is a sesamoid bone and should be considered a continuation of the quadriceps mechanism of insertion (in fact it is located in the rectus femoris tendon of insertion, with the other three bellies of the quadriceps group inserting onto the proximal pole of the patella). The patellar fracture should therefore be considered a gap in the quadriceps mechanism. If the gap opens during tension then the quadriceps mechanism is physically compromised. Even minimal widening of the fracture gap is expected to cause pain with each loading of the limb (Figure 10.25).

DIAGNOSIS:

The diagnosis of patellar fracture is confirmed radiographically. Mediolateral, craniocaudal and tangential views may be used to identify fracture type and exclude comminution.

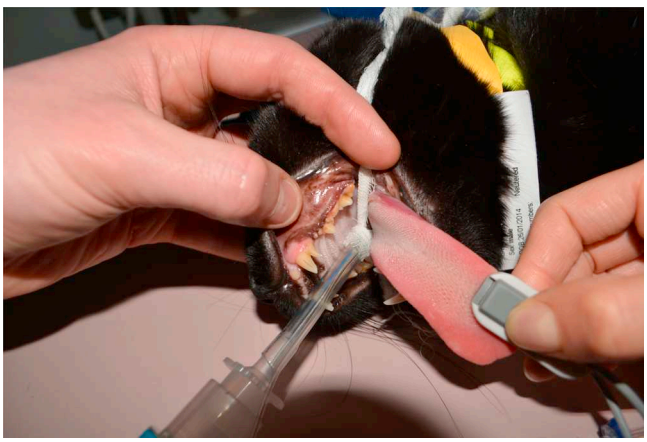


Figure 10.24 Multiple retained deciduous teeth in an adult cat presenting with patellar fracture, consistent with patellar and dental anomaly syndrome (PADS).

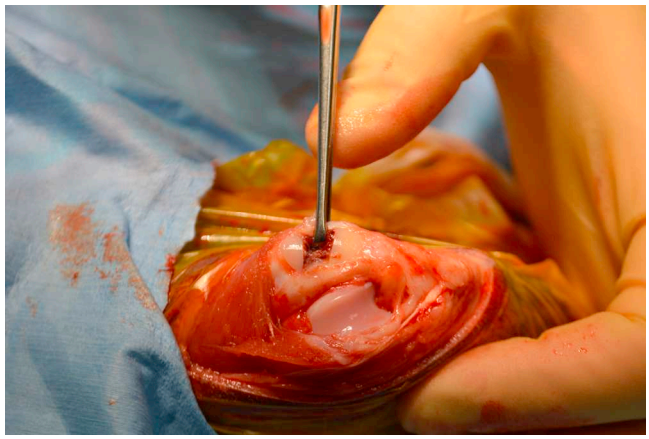


Figure 10.25 Intra-operative photograph of a transverse central patellar fracture (distal to the right, proximal to the left). The trochlear groove and caudal (articular) surface of the reflected patella are appreciated. An Alice tissue forceps has been used to reflect the patella, grasping the soft tissues interposed within the patellar fracture gap.

PADS/KATS:

(Howes et al. 2019, Reyes et al. 2019).

This condition is currently poorly defined. It appears to be a condition of insufficiency fractures (one type of stress fracture resulting from fracture of abnormal bone undergoing normal force); however, the aetiology of the primary bone abnormality is not known. Fractures may affect multiple bones over time, but most typically affect the patella. Persistence of deciduous teeth (Figure 10.24) alongside failure of eruption of adult teeth are common (42.8%). In a study encompassing 191 cats with suspected PADS/KATS 25% had concurrent acetabular fracture, 22% tibial fracture, 15.4% had tibial fracture and 13.7% had humeral condylar fracture. Therefore cats presenting with patellar fractures should be examined for evidence of dental abnormalities and healed fractures at other sites.

TREATMENT AND PROGNOSIS:

(Hermer et al. 2012).

Theoretically patellar fractures should be repaired to preserve the quadriceps extensor mechanism and re-establish continuity of the retropatellar articular cartilage surface to minimize the development of degenerative joint disease. Only two reports of healing of patellar fractures in cats exist in the literature, both in kittens (4 months and 5 months of age). In one case a minimally displaced fracture was treated conservatively and proceeded to healing, while in the other case circumferential wiring was performed and healing documented radiographically after 12 weeks. The majority of patellar fractures proceed to non-union for two reasons:

1. Patellar fractures are biologically disadvantaged (the patella possesses no medullary canal, and consists of dense cortical bone) and mechanically disadvantaged (it is continuously under traction as a result of quadriceps tension).
2. A proportion of patellar fractures in cats result from KATS/PADS rather than direct trauma, in which case the primary underlying abnormality remains, so healing is unlikely.

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Conservative Treatment

External coaptation may be used in some non-displaced fractures, maintaining the stifle in extension to prevent movement of the fragments caused by contraction of the quadriceps muscles. A bandage should remain in place for 4–6 weeks. Physical therapy is then needed to reestablish quadriceps muscle strength and joint mobility. Bone healing is unlikely to result; however, a functional quadriceps mechanism may be re-established if the fracture gap is stabilized by soft tissue alone.

Surgical Treatment

(Gibert et al. 2016).

To achieve healing of patellar fractures, the mechanical and biological concerns should be addressed. To date a method to address patellar fractures in cats with PADS/KATS has not been reported, but attempts to achieve healing in feline transverse patellar fracture are inevitably met with failure. A previously unreported alternative technique employing a fascia lata autograft is described later.

Subtotal/Total Patellectomy

(Carb 1975, DePalma & Flynn 1958).

Small chondral or osteochondral fragments are discarded if they cannot be incorporated into a repair. Where a transverse fracture affects the distal or proximal poles of the patella, the small polar fragment may be removed and discarded. It is important when doing this to ensure that the quadriceps mechanism is restored by suturing the distal quadriceps muscles and/or proximal patellar ligament (Figure 10.26).

Total patellectomy has been reported in the cat but is not recommended since the prognosis for improvement of stifle function is guarded.

Internal Fixation of Patellar Fractures

A lateral or medial stifle arthrotomy is performed to visualize the articular surface of the patella. Accurate reduction of the articular surface is important to reestablish joint congruity and this is maintained (and interfragmentary compression applied) using pointed reduction forceps.

Fixation is classically reported using cerclage wiring or pin and tension band wiring.

Cerclage Wire

Wire can be placed to encircle the patella. A large-bore hypodermic needle is used to pass cerclage wire through the quadriceps insertion onto the patella (proximally) and the patellar ligament origin (distally) to encircle the patella completely. The tension band wire is positioned on the cranial surface of the patella (tension side) to prevent

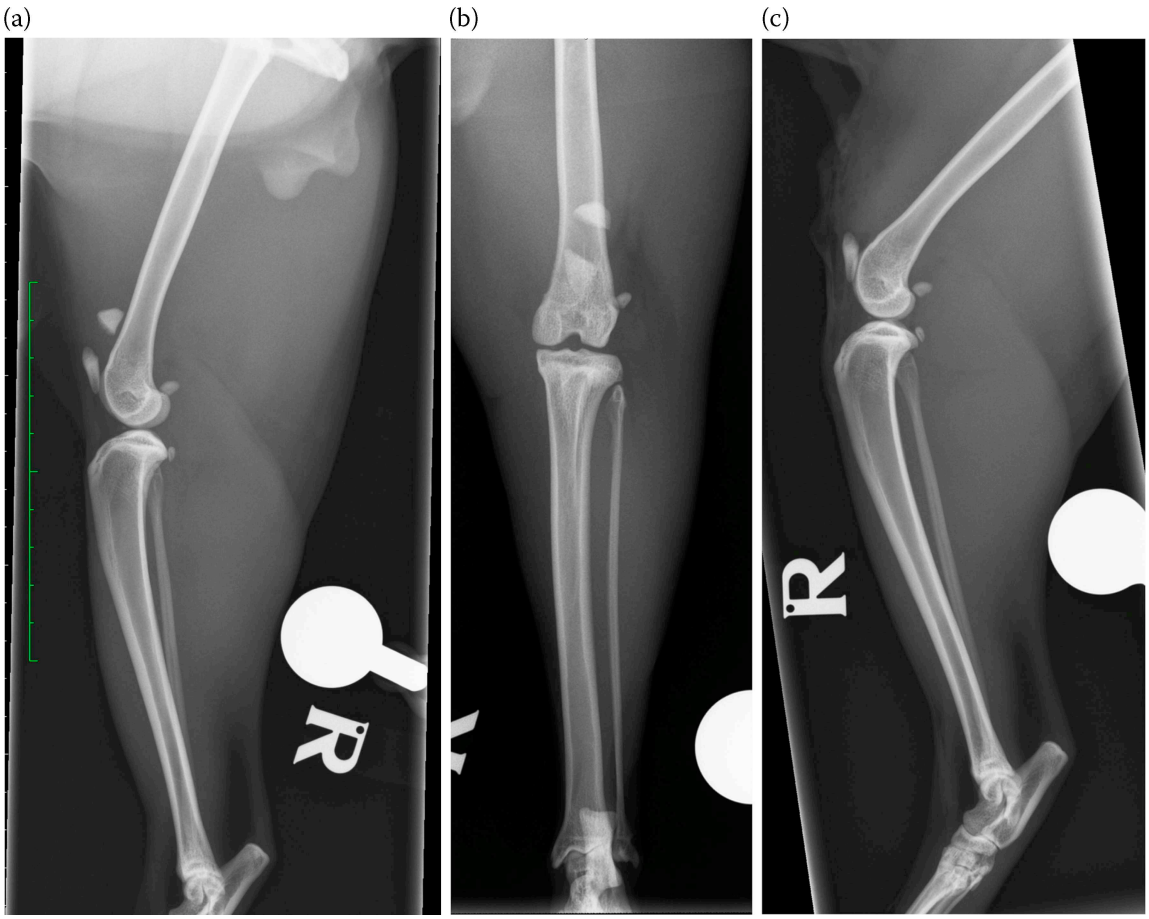


Figure 10.26 Mediolateral **(a)** and caudocranial **(b)** views of the tibia and stifle of a cat with transverse patellar fracture with moderate proximal displacement. **(c)** post-operative mediolateral view following removal of the proximal fragment (subtotal patellectomy) and suturing of the sectioned gluteal muscles to the remaining fragment and patellar ligament.

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displacement of the fracture line during weight bearing. Tightening the wire until the fracture is slightly over-reduced will create compression at the fracture when the stifle is flexed.

Pin and Tension Band Fixation

A Kirschner wire is inserted normograde or retrograde from proximal to distal through the patella. A tension band wire is placed in a figure-of-eight over the cranial surface of the patella looped over the protruding pin ends proximally and distally. The cerclage wire is tightened and the pins are cut short to minimize soft tissue trauma. The technique has not been reported in cats and is not recommended because of the likelihood of failure of the small dense patella during placement of the Kirschner wire. Predrilling with a 1.1 mm drill bit may reduce the risk of fracture during Kirschner wire placement. The Kirschner wire must not penetrate the caudal articular surface of the patella.

A pin and tension band wire technique was shown to be inferior to a prototype locking plate placed on the cranial surface of the patella in terms of fracture gap opening with cyclical loading in a canine cadaveric model. The plate (a polyethylene button) was attached to the cranial surface of the patella using fine Kirschner wires engaging the bone of the patella from cranial to caudal. The technique was reported in cadaveric canine patellae, and, due to the limited cranio-caudal dimensions of the feline patella, it may not be transferable to the feline patella.

Overall, the prognosis for achieving healing of a transverse patellar fracture, either of traumatic or insufficiency aetiology, is guarded.

Fascia Lata Autograft

One of the authors has treated a number of feline patellar fractures in cats with PADS/KATS (evidenced by history of previous or concurrent non-traumatic fractures) using a fascia lata autograft technique (Figure 10.27). Even where implants are placed to reduce the fracture, healing is likely to fail. Acceptance that healing may not be achieved using standard technique in these cats results in a paradigm shift in the philosophy of how these fractures may be addressed. The technique gives opportunity for bone healing but importantly also focuses on restoring quadriceps function. Where displacement of a patellar fracture has occurred, we can consider this a rupture of the rectus femoris tendon of insertion at the level of the fracture resulting in failure of the quadriceps mechanism. Restoration of this soft tissue connection between the quadriceps muscles proximal to the patella and the patellar ligament distal to the patella is critical to quadriceps function overall.

1. The fracture is reduced and an encircling non-absorbable suture or wire is placed to reduce the fracture gap.
2. A second suture (monofilament non-absorbable or slowly absorbable) is placed encircling the patella through the quadriceps insertion on the proximal patella and through a bone tunnel in the tibial tuberosity. In this

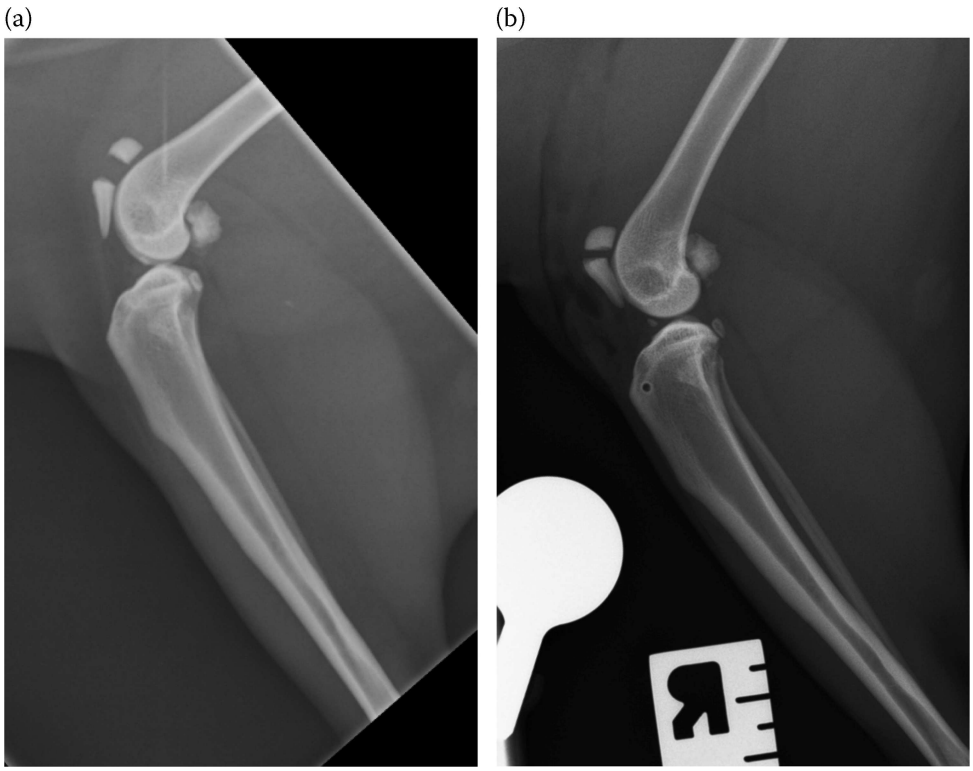


Figure 10.27 Mediolateral views of the stifle of a cat with a transverse patellar fracture. **(a)** pre-operative view demonstrating mild proximal displacement of the proximal patellar fragment and gap widening. **(b)** post-operative radiograph demonstrating closure (but not interfragmentary compression) of the fracture gap, restoring quadriceps function, though not eliminating a gap defect in the patellar articular surface. Surgery involved a suture (4 M polydioxanone) placed circumpatellar and through a bone tunnel in the tibial tuberosity to reduce strain on the patellar fracture and augmentation using a tensor fascia lata autograft with a distal base.

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way the distracting force of the quadriceps mechanism is neutralized (at least temporarily, depending on the longevity of the material).

3. A strip of tensor fascia lata is elevated from the lateral aspect of the limb with a base at the level of the proximal patella. The graft is passed just proximal to the patella from lateral to medial through the quadriceps insertion. The graft is placed overlying the patella and patellar ligament. The graft is sutured distally (simple interrupted or cruciate sutures) to the fascia overlying the lateral aspect of the tibial tuberosity, along the length of the patellar ligament and to the quadriceps musculature proximally (where a locking loop pattern may be considered to improve holding power in the linear myofibres of the distal quadriceps muscles).

A variable increase in the width of the fracture gap is observed in the few weeks following this technique, and patellar healing has not been documented. However, the fracture gap itself is not relevant in terms of restoring function of the quadriceps mechanism, and subjective success

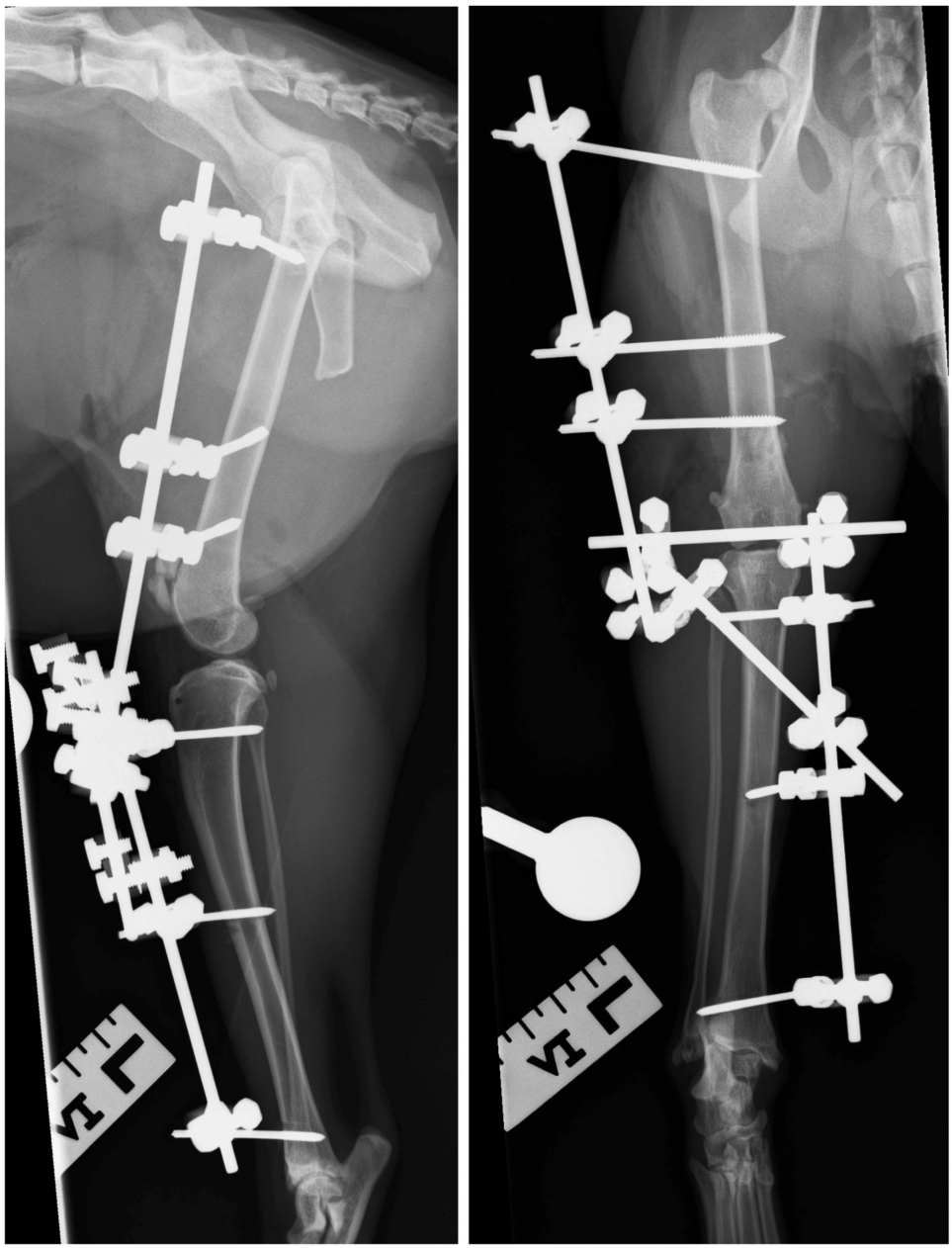


Figure 10.28 Mediolateral and caudocranial views of the femur and tibia following reduction of a patellar fracture (note poor patellar fracture reduction, which is sometimes the consequence of a circumpatellar suture/wire) and augmentation by fixation of the stifle in extension with a trans-articular external skeletal fixator. The external skeletal fixator consisted of three 2.5 mm positive profile fixation pins placed from lateral to medial and three 2.0 mm positive profile fixation pins placed from medial to lateral.

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has been reported. It is not known what relevance a persistent transverse patellar fracture (with or without a small gap) has on the future health of the stifle joint.

Augmentation with Trans-Stifle External Skeletal Fixator

(Jaeger et al. 2005, Lister et al. 2009).

A trans-articular external skeletal fixator may be placed to reduce the strain on the quadriceps mechanism during the post-operative period (Figure 10.28). A modified type 1 frame with three 2.5 mm positive profile half pins on the lateral aspect of the femur (one in the greater trochanter aimed distally to purchase the medial cortex distal to the femoral neck, two close to the distal femoral condyles), and three positive profile pins on the cranial or medial aspects of the tibia (spread evenly along the length of the bone) is recommended. A hinge may be considered at the level of the joint.

Trans-stifle external skeletal fixator placement is considered contra-indicated in cases in which PADS/KATS is suspected, given the potentially increased risk of fractures of the femur and/or tibia through pin tracts. Trans-articular fixation probably does not remove all force on the healing patella anyway and is not considered critical for a successful outcome.

Post-Operative Care

It is possible that the majority of healing relates to restoration of soft tissue structures, rather than fracture healing. Therefore, the time-frame for the convalescence is not the same as for most post-operative fracture cases. However, management is not dissimilar. Activity is strictly restricted for 4 weeks. Radiographs may be acquired at this stage to ensure minimal or no opening of the patellar fracture gap. Mild, passive flexion and extension of the stifle may be started five days following surgery. Room confinement is maintained for 4 weeks and house confinement for a further 6–8 weeks prior to a resumption of outdoor activity.

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Part 4 Fractures of the Tibia and Fibula

(Brunnberg 2003, Harari 2002, Nolte et al. 2005, Perry & Bruce 2015, Phillips 1979, Richardson & Thatcher 1993, Whitney & Mehlhaff 1987).

Tibial and fibular fractures are common in the cat, accounting for 5% to 19% of all fractures. Most result from various types of trauma, including automobile trauma, bite injuries, gunshot wounds, and falls (high rise syndrome). Because of the minimal soft tissue coverage on the medial aspect, tibial fractures are frequently open (17%–46%). Following various fixation techniques tibial fractures have been reported to carry a high risk of complications, including infection, delayed union and nonunion. The reported nonunion rate is up to 37.5%. Generally, fibular fractures are not treated with internal fixation and heal readily once the tibia is stabilized. However, fibular head fractures causing stifle instability and distal malleolar fractures causing tarsal instability are often surgically repaired.

PROXIMAL TIBIAL FRACTURES:

Fractures of the proximal tibia include avulsion fracture of the cranial cruciate ligament, tibial plateau fractures and proximal metaphyseal/diaphyseal fractures. In immature cats tibial tuberosity avulsion should be considered, sometimes in addition to proximal tibial physeal fracture/separation. Physical examination should also include a complete evaluation of the ligamentous structures of the stifle joint and stressed radiographs and/or computed tomography should be considered.

Tibial Tuberosity Avulsion Fractures with/without Proximal Tibial Physeal Fracture/Separation

(Milton et al. 1980, Prieur 1989).

The tibial tuberosity is the insertion point for the patellar ligament. Hyperflexion of the stifle joint alongside quadriceps contraction may cause separation (avulsion) of the growth plate of the tibial tuberosity in immature cats (usually those less than 10 months of age). This is a common condition in dogs but is rare in cats.

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Clinical Signs

Clinical signs include lameness, pain in the stifle region and a limited ability to extend the stifle joint.

Diagnosis

Lateral and caudocranial radiographs of the tibia centred on the stifle confirm the diagnosis. The tibial tuberosity is displaced proximally and the patella is positioned more proximally in the trochlear sulcus. Views should be inspected for concurrent displacement of the proximal tibial epiphysis (indicating proximal tibial physeal fracture/separation) and if this is present the caudocranial view is scrutinized for medial/lateral displacement of the proximal tibial epiphysis + - medial or lateral fractures of the metaphysis or epiphysis (Salter-Harris types 2 and 3, respectively).

Management

Conservative therapy is rarely recommended in dogs with this condition since proximal displacement of the cranial aspect of the proximal tibial epiphysis results in an increased tibial plateau angle, with potential repercussions on the cranial cruciate ligament. However, reports of this sequel in the cat are rare, with or without surgical treatment. Therefore, if displacement is minimal, 2–4 weeks of crate rest may be considered (a dressing which maintains the stifle in slight extension has anecdotally been suggested; however, such a dressing is unlikely to remain in place on a cat for the full period of exercise restriction). Repeat radiography should be considered after 7–14 days to ensure no further proximal migration of the tibial tuberosity + - proximal tibial epiphysis. If fracture displacement is severe in the first instance or if it continues over the course of 7–14 days then surgical management is indicated.

Open Reduction and Stabilization

Surgical repair is recommended for treatment of tibial tuberosity avulsion fractures with moderate or severe displacement (>3 mm) or where displacement continues over a week or two following the initial injury (Figure 10.29).

1. A short incision is made on the medial aspect of the patellar ligament and tibial tuberosity.
2. The distal aspect of the tibial tuberosity is identified at the level of the physeal fracture and reflected caudally and distally back into its normal location (stifle extension facilitates this reduction). This manoeuvre is typically expected to reduce any associated proximal tibial epiphyseal fracture/separation. If the fragment is small, it may be manipulated by driving a Kirschner wire from cranially. Once positioned appropriately, the Kirschner wire is driven further into the metaphysis to hold it in place and the positioning verified.
3. Fixation involves one or two Kirschner wires placed from the cranial aspect of the tibial tuberosity through the proximal tibial metaphysis to engage the caudal tibial cortex. If two Kirschner wires are placed, at least one should be placed just proximal to the point of the tibial tuberosity, penetrating the patellar ligament fibres, and reducing the risk of fracture of the tibial tuberosity through the pin tract. If only one Kirchner wire is to be placed, it should start just proximal to the point of the tibial tuberosity. The wires should be directed perpendicular to the patellar ligament in the sagittal plane.
4. If the wires are directed distally (to avoid penetrating the caudal aspect of the stifle joint) then a tension band wire is required to reduce the risk of pin migration. A tension band wire should also be considered for cases of tibial tuberosity avulsion with severe displacement and for cases in which a concurrent proximal tibial physeal fracture/separation is present. A bone tunnel is drilled in the cranial aspect of the tibia distal to the tibial tuberosity. Orthopaedic wire is passed through this bone tunnel, crossed over the cranial aspect of the tibial tuberosity and passed around the proximal-most of the Kirschner wires. The tension band wire counteracts the pull of the quadriceps muscles.
5. Any associated proximal tibial physeal fractures in which the proximal tibial epiphysis is medially or laterally

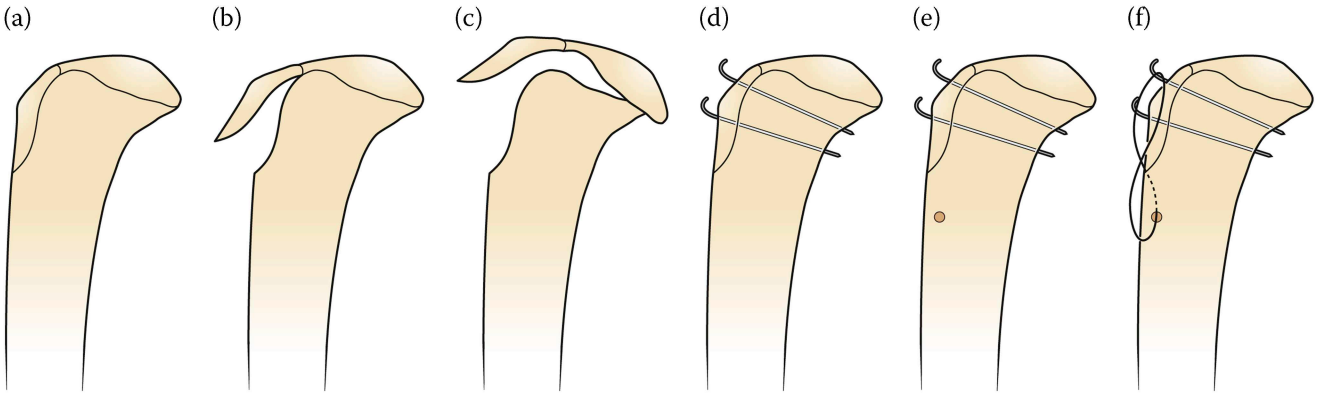


Figure 10.29 Diagrams of tibial tuberosity avulsion injury and fixation. **(a)** the normal tibia in the skeletally immature cat demonstrates a tibial tuberosity apophysis and a proximal tibial epiphysis. **(b)** the tibial tuberosity apophysis may be displaced proximally without displacement of the proximal tibial epiphysis or **(c)** in combination with the proximal tibial epiphysis which may undergo type 1 or type 2 Salter-Harris fracture. **(d)** Reduction is performed using traction applied to the tibial tuberosity and fixation is with one or two parallel Kirschner wires (at least one pin should penetrate the bone proximal to the insertion of the patellar ligament). **(e)** a bone tunnel is drilled caudal to the cranial cortex in the proximal tibial diaphysis. **(f)** a figure-of-eight tension band wire is placed through the bone tunnel and looped over the proximal-most Kirschner wire to apply traction to oppose the proximal pull from the quadriceps muscles.

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displaced should undergo fixation using Kirschner wires placed from the medial and/or lateral articular margin of the tibial plateau in cross-pin fashion to engage the contralateral cortex.

Post-operatively, the cat is strictly confined for 2–4 weeks at which point radiographs are acquired. In immature cats, implants are removed as soon as healing is complete to allow any further growth of the proximal tibia. The prognosis for limb function after repair of an avulsion fracture of the tibial tuberosity is good to excellent if the condition is treated early and anatomic reduction and rigid fixation are achieved.

Tibial Plateau Fractures

Cause and Pathogenesis

Fractures of the tibial plateau often involve the articular surface and may occur as an extension of oblique or longitudinal fractures of the tibial metaphysis.

Clinical Signs

The cat is typically non-weight bearing on the limb, and pain, swelling and crepitus are evident on palpation of the stifle joint.

Diagnosis

The diagnosis is confirmed by radiography. Oblique radiographic projections may help to reveal intra-articular fractures.

Treatment and Prognosis

If the fracture can be anatomically reduced in a closed manner, external coaptation may be applied including splints or full casts. However, in most cases, articular fractures are managed with open reduction and rigid internal fixation in order to minimize future osteoarthritis.

1. The approach may be craniomedial or craniolateral.
2. Fracture reduction requires delicate manipulation to prevent iatrogenic trauma to the articular surface.
3. Fixation is achieved using one or more combinations of intramedullary pinning plus fixation of small fragments with Kirschner wire(s) or small screw(s), preferably in lag fashion to generate interfragmentary compression and promote direct bone union.
4. Fixation of the fibular head is performed with a single Kirshner wire placed through the fibular head and driven across the proximal tibial metaphysis to provide additional stability and/or to restore lateral collateral ligament function.
5. A plate in buttress fashion on the proximal tibia (medial, cranial, difficult craniolaterally) to support the tibial plateau and stabilize metaphyseal portions of the fracture may be considered.

Cranial Cruciate Ligament Avulsion Fractures

Avulsion fractures of the cranial cruciate ligament occur very rarely in cats, and usually in conjunction with severe trauma to the stifle joint. A positive cranial draw motion, joint effusion and pain are detected on palpation. Radiographically, a small bony fragment may be visible within the joint. This should not be mistaken for mineralization within intra-articular soft tissues. In most cases, the bone fragment is too small to allow primary fixation and it is removed along with the cranial cruciate ligament. The cruciate-deficient stifle is then stabilized with one of the techniques outlined in Chapter 11. Note that traumatic cranial cruciate ligament rupture or avulsion is often associated in cats with rupture of other stifle supporting structures, such as the caudal cruciate ligament, collateral ligaments and menisci, and these injuries should be considered during planning.

Proximal Metaphyseal/Diaphyseal Fractures

(Newman & Milton 1989, Payne et al. 2005).

Fractures of the proximal tibial metaphysis are often transverse or short oblique (Figure 10.30). Longitudinal fractures may extend into the stifle joint. Occasionally a curvilinear fracture is observed beginning just distal to the tibial tuberosity and extending proximo-caudally before deviating distally to emerge from the caudal tibial cortex.

Closed Reduction and Stabilization

If the fracture can be reduced closed and is inherently stable, stabilization may involve external coaptation (potentially incorporating a splint, but immobilization of the stifle is not likely to be achieved), closed intramedullary pinning, or an external skeletal fixator.

Closed Intramedullary Pinning

1. A small stab incision is made medial to the insertion of the patellar ligament.
2. The stifle is flexed 90° and a Steinmann pin which fills 60–70% of the narrowest point of the tibial medullary canal is started at the medial aspect of the tibial plateau (approximately 5 mm caudal to the point of the tibial tuberosity).
3. The fracture is reduced and the pin is driven along the medial tibial cortex and seated into the distal metaphysis. The pin is then retracted 5 mm, cut short with a pin cutter, and reseated using a countersink and mallet to reduce trauma to the stifle.

Closed Application of an External Fixator

The medial aspect of the tibia lends itself to closed application of a type 1 frame. Half pins are driven into the proximal and distal bone fragments and the connecting bar applied. Further pins are added to the frame until three half pins are in place proximal and distal to the fracture, to maximize stability. A free-form frame may be considered to increase the versatility where multiple points of fixation are required in a small bone fragment.

Open Reduction and Stabilization

More rigid internal fixation is typically recommended for proximal metaphyseal fractures of the feline tibia. Methods of open fixation for proximal metaphyseal fractures include intramedullary pins, lag screw/Kirschner wire fixation, external skeletal fixators and small bone plates and screws.

Intramedullary pinning: A Steinmann pin is inserted normograde from the medial aspect of the tibial plateau. Retrograde insertion is also feasible, although the pin should be directed toward the craniomedial aspect of the tibial plateau to avoid injury to the patellar tendon or cruciate ligaments.

The proximal tibia does not lend itself to the application of cerclage wire.

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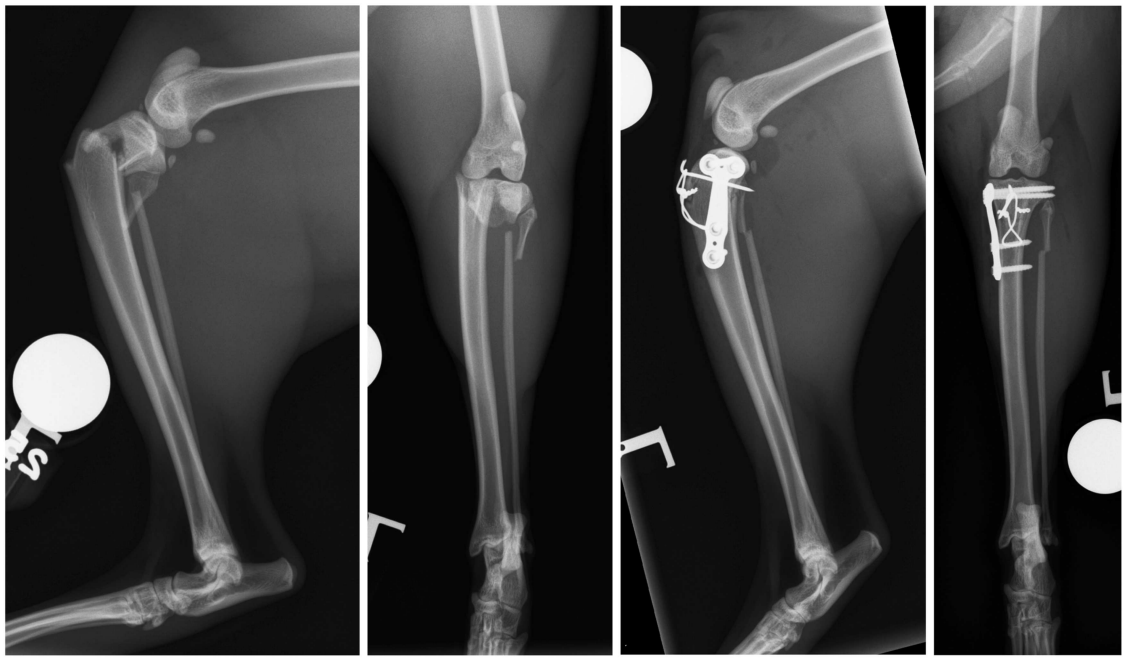


Figure 10.30 Pre-operative and post-operative radiographs of a proximal tibial metaphyseal/diaphyseal fracture associated with a proximal fibular fracture in an 11-month-old cat following a suspected road traffic accident. The small proximal fragment was reduced by traction applied to the tibial tuberosity and initial fixation was applied with a pin and tension band technique. Given the instability innate to this fracture a medial locking T-plate was applied in buttress fashion with two screws distal to the fracture and two screws in the proximal fragment.

Lag screw/Kirschner wire fixation: A single lag screw or two lag screws placed in a crossed-screw fashion can be used to stabilize some proximal metaphyseal fractures. Alternatively, Kirschner wires in cross-pin fashion may be used to stabilize proximal tibial fractures.

External skeletal fixator: A type I or freeform ESF frame may be applied following open reduction.

Bone plate and screws: Depending on the fracture conformation, plates may serve in neutralization or buttress modes and compression may be desirable. Small TPLO plates are intended to be used on the medial aspect of the proximal tibia and may be employed as primary fixation or in neutralization mode for proximal metaphyseal/diaphyseal fracture fixation (Figure 10.30).

DIAPHYSEAL TIBIAL FRACTURES:

(Boone et al. 1986a, Cabassu 2019, Craig et al. 2017, Gutbrod et al. 2017, Morris et al. 2016, Nicetto & Longo 2017, Payne et al. 2005, Reems et al. 2003, Schmökel et al. 2007).

Diaphyseal tibial fractures are common in cats. They are amenable to fixation using numerous methods. They are prone to complications, including non-union and the methods of reduction and fixation should reflect this, by protecting the biological environment and optimizing stiffness and durability.

External Coaptation

Splinting and casting may be used to stabilize tibial fractures with minimal displacement, particularly long oblique and/or greenstick fractures in young kittens. The presence of an intact fibula increases the chances of success with external coaptation, since the fibula prevents collapse at the fracture site and helps to control rotational and shearing forces. The splint or cast is applied with the stifle and hock joints at functional weight-bearing angles. When the coaptation device is removed, physical therapy may be required to restore full function of the stifle and hock joints. In young patients the presence of a stiff external coaptation can have deleterious effects on the physes and on bone density, so this approach is avoided where possible.

Intramedullary Pinning

Intramedullary pinning is a common technique for stabilization of tibial fractures in cats. An intramedullary pin controls bending forces and resists axial forces somewhat. However, when an intramedullary pin is placed in isolation, rotation is not controlled, and unless the pin fills the medullary canal at the level of the fracture, shear is also not controlled. The relatively straight tibia in the cat generally permits easy insertion. Pins should be placed normograde from proximal, since retrograde intramedullary pinning was shown in cadaveric studies to risk trauma to the patellar ligament, cruciate ligaments and articular cartilage.

Closed Pinning

Closed pinning is used if the fracture can be satisfactorily reduced with minimal trauma to the overlying soft tissues and if interdigitation of the fracture segments will provide rotational stability with a single intramedullary pin.

1. A small stab incision is made medial to the insertion of the patellar ligament.
2. The stifle is flexed 90° and a Steinmann pin which fills 60–70% of the narrowest point of the tibial medullary canal is started at the medial aspect of the tibial plateau

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(approximately 5 mm caudal to the point of the tibial tuberosity).

3. The fracture is reduced and the pin is driven along the medial tibial cortex and seated into the distal metaphysis. The pin is then retracted 5 mm, cut short with a pin cutter, and reseated using a countersink and mallet to reduce trauma to the stifle.

Open Reduction and Pinning

In some cases it may be preferable to perform an open approach to the fracture to visually assess reduction and/or to place ancillary fixation. An intramedullary pin may then be placed (normograde as described under 'closed pinning') to facilitate reduction and maintain the bone at the correct length. Once the pin is in place, ancillary fixation with cerclage wires, plate and screws (plate-rod fixation) or an external skeletal fixator can be applied. Open fixation also allows the placement of bone graft to promote healing.

External Skeletal Fixators

External fixators may be used for stabilization of most tibial fractures and are generally well tolerated by cats. They are particularly useful in the repair of open or infected fractures as the implants are placed remote to the contaminated site and are eventually removed, therefore will not harbour bacteria in the long term. External skeletal fixators are applied through stab incisions following closed or open reduction (the fixation pins are not placed through the incision made to approach the fractured bone).

Type I (unilateral) frames are easily applied to the medial or cranial aspects of the tibia. Type II frames (bilateral and uniplanar) may be used in severely comminuted fractures, but there is no safe corridor for fixation pin placement on the lateral aspect of the tibia. Circular-linear hybrid external skeletal fixators have been described for the stabilization of canine and feline tibial fractures.

A study comparing complication rates for feline tibial fracture fixation identified complications in 1/7 cats in the plate and screw group versus 22/44 in the external skeletal fixator group. Cats with external skeletal fixation had longer follow-up and higher numbers of re-examinations.

One study of a locking plate utilized as an external skeletal fixator in a feline tibial fracture has been reported. The authors claimed the benefit of the plate over a true external skeletal fixator was a lower profile. At this stage it is unclear whether this approach will become more familiar in future.

Bone Plates and Screws

Bone plates carry a lower risk of complications than external skeletal fixators in feline tibial fracture fixation. They are typically applied to the medial surface of the tibia and may be used in neutralization or bridging function, depending on the fracture configuration. If the fracture lends itself to interfragmentary compression then dynamic compression plates may be employed. The medial saphenous vein should be preserved during the medial approach. If a plate or plate-rod system is to be employed then minimally invasive percutaneous osteosynthesis may be considered with mini approaches made proximally and distally and the plate slid through an epiperiosteal bone tunnel such that two to three screws may be placed at each end of the plate.

Minimally Invasive Percutaneous Osteosynthesis

Locking plates are commonly recommended for minimally invasive percutaneous osteosynthesis. This is not as a result of improved strength or stiffness in locking systems, as is commonly suggested. Rather, the emphasis on locking implants for minimally invasive percutaneous osteosynthesis relates to the fact that a locking plate-screw construct does not rely on friction generation between the plate and bone, and therefore accurate contouring is not necessary. This is of benefit in the context of minimally invasive surgery because accurate contouring is difficult when the bone is not exposed. Whilst locking technology may be beneficial in minimally invasive percutaneous osteosynthesis, many locking systems are 'fixed angle', meaning that screws must be inserted into the plate at a specific angle to ensure locking between the plate and screw. A study investigated the feasibility of placing bicortical screws in a 2.4 mm locking compression plate (a fixed angle plate) applied to the medial aspect of the feline tibia in combination with various diameter intramedullary pins. The study is important because the sagittal plane width of the tibia is variable, being rather generous proximally but small distally. If the intramedullary pin interferes with screw placement in the locking plate then locking plate-rod technique may not be possible in this bone (unless one of the polyaxial locking systems are utilized). In the study bicortical screws could be placed in the proximal tibia even with intramedullary pins up to 50% of the diameter of the medullary canal; however, in the distal tibia placing bicortical screws was frequently not possible, although it was always possible to place monocortical screws. Where the intramedullary pin filled only 30% of the medullary canal more bicortical screws could be placed distally. The recommendation in plate-rod systems in which non-locking technology is applied is usually to fill 30–40% of the medullary canal with the intramedullary pin. This rule should also apply to plate-rod constructs with locking plates. (Figure 10.31)

A technique termed 'fracture reduction under the plate' has been described to facilitate minimally invasive percutaneous osteosynthesis where intra-operative imaging is not available to direct fracture reduction/bone realignment. The technique was reported retrospectively in canine and feline femoral and tibial fractures (eight feline tibial fractures). In this technique the plate is contoured accurately pre-operatively according to radiographs of the contralateral intact bone. The plate is positioned on the fractured bone using minimally invasive percutaneous osteosynthesis technique. Non-locking screws are placed through the proximal and distal approaches via the plate holes into pre-planned locations in the proximal and distal bone segments. Tightening the screws should draw the bone segments into correct alignment. Whilst this technique reduces the need for fluoroscopic guidance of fracture reduction, in the authors' experience percutaneous palpation is often sufficient to identify feline tibial fracture malreduction intra-operatively. The contour of the plate alone cannot be relied upon to achieve reduction, as was demonstrated in the study, with 5 of 41 cases requiring immediate revision due to implant malpositioning or fracture malalignment.

ILN Fixation

(Bruckner et al. 2014, Diaz-Bertranal et al. 2005, Duhautois 2003, Nabholz et al. 2019).

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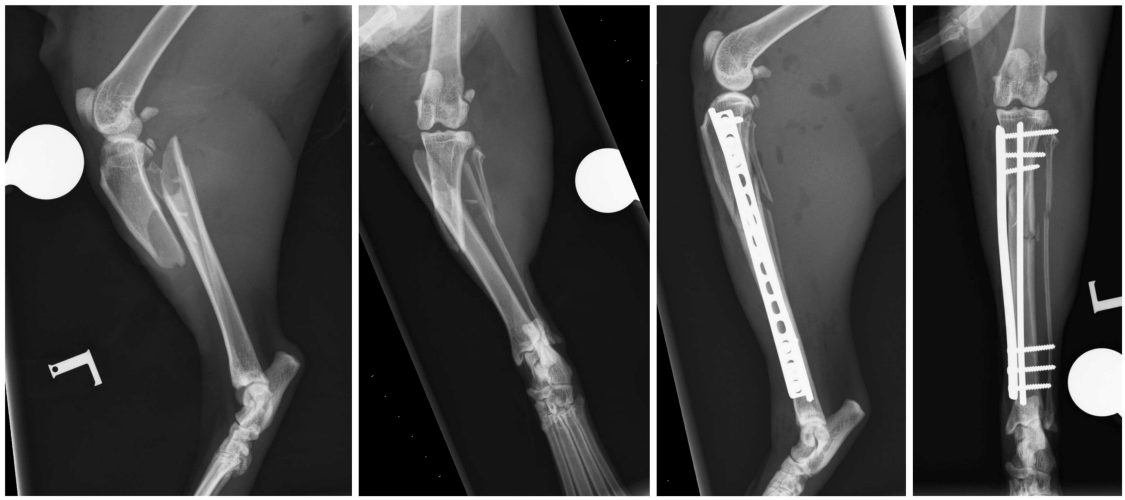


Figure 10.31 Pre-operative and post-operative mediolateral and caudocranial views of the left tibia in a cat with a mildly comminuted diaphyseal tibial fracture. Fixation was by application of a plate-rod construct. The intramedullary pin is placed normograde from proximal, restoring bone length and alignment to some extent. The plate is applied using minimally invasive technique. It is placed through a short incision to the medial aspect of the proximal tibial metaphysis and slid through an epiperiosteal bone tunnel to a short incision at the level of the distal tibia. Screws are applied at the proximal and distal extents of the plate via the short approaches. Note: the intramedullary pin has been cut at its distal end to remove a sharp point which might penetrate the talo-crural joint. In fact the intramedullary pin could have been placed more distally, increasing the purchase of trabeculae in the distal tibial metaphysis (further insertion of the pin may have been limited by contact with the caudal cortex). The screws in the distal fragment are longer than necessary. (Usually 2–4 mm protruding from the trans cortex is recommended)

Various systems of ILN exist for use in the cat and successful outcomes may be achieved with any of them. ILNs are placed according to the guidelines of the specific implant systems with one or two bolts on either side of the fracture. Surgeons performing ILN fixation of the tibia should note that the distal tibia is narrow and large diameter holes at this level may create stress risers, increasing the risk of iatrogenic fracture. However, a cadaveric study showed no complications with placement of an ILN system in the tibia, compared with multiple complications when the same system was placed into femora and humeri.

DISTAL TIBIAL FRACTURES:

Distal tibial fractures

(Boone et al. 1986b, Berzon 1980, Craig et al. 2018, Dugat et al. 2011, McLaughlin et al. 1992, Witte et al. 2014).

Distal tibial physeal fractures are often severely displaced and accompanied by distal fibular diaphyseal or physeal fractures. These fractures carry a good prognosis following accurate reduction and appropriate fixation. However, fractures in the region of the distal tibia of the adult cat are commonly affected by atrophic non-union. This is likely due to the biological environment of the feline distal tibia. There is limited soft tissue coverage at this level, such that the periosteal blood supply to a healing fracture is limited. In addition, the intraosseous vascular supply has been demonstrated to consist of one or two large diameter vessels which are prone to trauma and slower to be restored following trauma in comparison with the blood supply in the canine distal tibia in which there is diffuse arborization into multiple smaller vessels, an arrangement which may promote revascularization following trauma. Finally, in one study of external skeletal fixation of feline distal tibial fractures healing was uneventful in closed fractures, but non-unions were experienced in cases in which the fractures were open. Why open fractures are more likely to undergo non-union may be due to contamination/infection but is more likely associated with the greater soft tissue disruption innate to open fractures. Given concerns with the extra-osseous and intra-osseous blood supply and the fact that distal tibial fractures in cats are commonly open, these fractures pose a significant concern.

Distal Tibial Physeal Fractures

Cross-Pin Fixation

Two Kirschner wires are inserted from the lateral and medial malleoli across the fracture line, and into the tibia. The wires should cross proximal to the fracture line and exit the tibial cortex. The tibial sulci are deep so placement of these pins must be almost parallel to the long axis of the bone to avoid penetration of the joint. A temporary trans-articular external skeletal fixator or external coaptation may be applied for 2–4 weeks to augment the repair if this is considered tenuous (Figure 10.32).

Trans-Articular External Skeletal Fixator

A trans-articular external skeletal fixator may be applied following reduction of the physeal fractures, without any direct internal fixation of the fractures themselves. Immobilization of the joint prevents fracture displacement, and because they are physeal fractures they are expected to heal rapidly. The frame is maintained for approximately 4 weeks.

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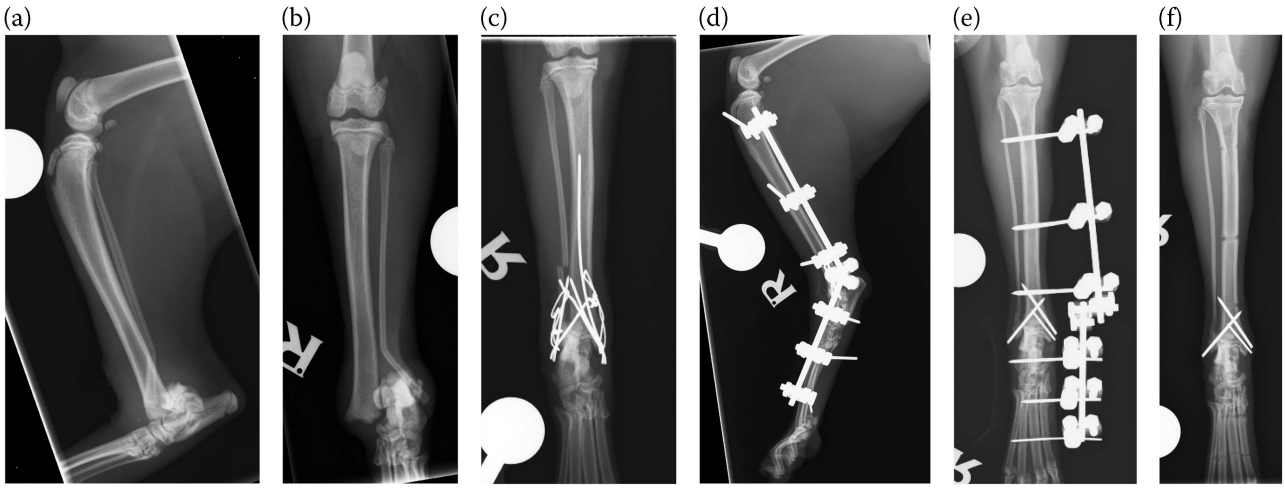


Figure 10.32 Distal tibial physeal fractures are commonly associated with distal diaphyseal fibular fractures and may be severely displaced **(a, b)**. Fixation following reduction may involve cross pins. The malleoli extend distally on either side of the talus, therefore placement of the pins requires a very acute angle to avoid interference with the joint. Cross pins may be combined with tension band wires, which are placed through bone tunnels drilled across the medial or lateral cortex of the distal tibia or may be hooked over the protruding points of Kirschner wires placed from the other side **(c)**. Note that multiple pins have been placed in this case with tension band wires on both sides. This is typically considered unnecessary, with a single pin + - tension band wire typically being sufficient. Note also the pin which has glanced off the lateral tibial cortex and continued proximally within the medullary canal, rather than engaging the bone. Where additional stability is required (if bone purchase is considered to be limited or where concurrent orthopaedic disease is present) then a trans-articular skeletal fixator may be applied to augment the repair through hock immobilization for 4–6 weeks **(d, e)**. The appearance following removal of the external frame is shown in **(f)**.

Distal Tibial Fractures in Adult Cats

Hybrid External Skeletal Fixation

A hybrid external skeletal fixator frame consisting of a proximal full pin and two medial half pins in the tibia and a split ring at the level of the distal tibia with fixation of the distal tibial fragment by 1.1 mm olive wires has been reported (Figure 10.33). In that study healing was complete within 3 months in the majority of cases of closed fractures. Three open tibial fractures failed to heal (although the fibulae healed in all three cases with large callus indicative of sufficient vascular supply to that bone), and they were revised by removal of the external fixator frames and replacement with short medial plates.



Figure 10.33 A cat with a hybrid external skeletal fixator on the tibia to stabilize a distal tibial fracture.

Double Plate Internal Fixation

Because of the short length of distal tibial bone stock present in distal tibial fracture in cats, and the narrow cranio-caudal width of the bone, fixation using a single plate (even a T-plate) rarely permits rigid fixation with sufficient points of bone purchase. However, a study of cats in which double plating was performed using minimally invasive percutaneous osteosynthesis reported good outcomes (Figure 10.34). The technique is as follows:

1. Mini (1–2 cm long) approaches are made craniomedially to the proximal and distal aspects of the fractured tibia.
2. Medial and cranial extra-periosteal bone tunnels are made with a small periosteal elevator to connect the proximal and distal approaches.
3. The reduction of the fracture is guided by percutaneous palpation or intra-operative radiographic imaging (fluoroscopy). If necessary it is often possible to visualize the fracture site through the distal incision.
4. A 2.0 mm or 2.4 mm veterinary cuttable plate is slid along the medial aspect of the bone between the two mini approaches and two screws are placed proximally and, if possible, distally.
5. A 1.5 mm or 2.0 mm veterinary cuttable plate is slid along the cranial aspect of the tibia within the extra-periosteal bone tunnel. Two screws are placed proximally and, if possible, distally.

The plates require minimal contouring, since the medial aspect of the bone is relatively flat and the cranial plate is typically sufficiently thin that it contours itself to the bone

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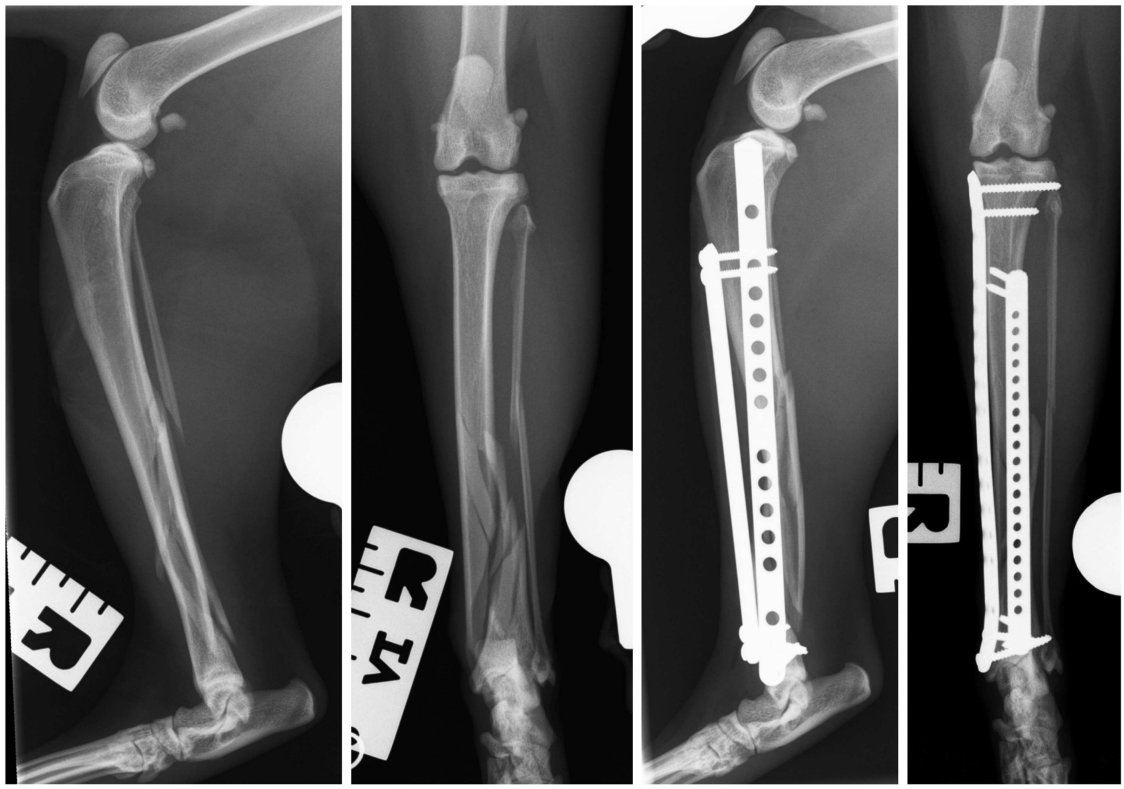


Figure 10.34 Mediolateral and caudocranial radiographic projections of the right tibia of a cat following a suspected road traffic accident revealing a comminuted distal tibial fracture in which fissuring extends almost to the level of the talo-crural joint. Fixation was with application of orthogonal veterinary cuttable plates with 2.4 mm screws in the medial plate and 2.0 mm screws in the cranial plate. The approach was minimally invasive, with a short craniomedial approach for placement of all four distal screws and a longer craniomedial approach to the proximal tibia including elevation of the insertion of the caudal part of Sartorius on the medial aspect of the tibial metaphysis. (Which was closed over the top of the proximal end of the plate as part of the closure)

during screw tightening. The cranial plate may be somewhat shorter than the medial plate (which spans the length of the bone) such that the proximal end of the plate fits distal to the tibial tuberosity. Double plating offers two significant advantages: screws and plates are placed at right-angles to one another, increasing the area moment of inertia of the construct dramatically. In addition, in the distal fragment, it is typical that two screws can be placed at least in one plate, such that a minimum of three screws can be placed, with six points of fixation.

Malleolar Fractures

(Kulendra & Arthurs 2014, Roch et al. 2009).

The lateral and medial malleoli represent the origins of the collateral ligaments of the talocrural joint and therefore their fractures lead to instability of the joint (Figure 10.35). Malleolar fractures are also often concurrent to partial or complete rupture of one or both of the ipsilateral and/or contralateral talocrural collateral ligaments (see Chapter 11). Stressed radiographs are used to demonstrate the severity of instability within the joint. If the malleolus is minimally displaced and instability is minimal, a period of external coaptation may be sufficient to restore joint function through a functional non-union of the malleolus and/or stabilization of the talocrural joint through peri-articular fibrosis.

Medial malleolar fractures occur alone in 1/6 of cases, lateral malleolar fractures occur alone in 1/3 of cases and both malleoli are fractured in 1/2 of cases.

As malleolar fractures affect the articular surface, the optimal approach involves rigid internal fixation. Stabilization may be achieved using one or more Kirschner wires (depending on the size of the fragment) and a tension band wire.

1. Short approaches are made directly over the malleoli, or just cranial or caudal if the skin overlying the malleolus in question appears traumatized.
2. Kirschner wires are inserted through the fractured malleolus (after reduction) and through both cortices of the tibia in a distal to proximal orientation.
3. Tension band wires are placed to counter the effect of loading causing tension on the joint space medially or laterally. Narrow gauge orthopaedic wire is looped around the wire at the level of the distal aspect of the malleolus and, in a figure-of-eight pattern, passed either through a bone tunnel or screw in the distal tibia, or hooked over the protruding end of a pin placed to secure the contralateral malleolus in the case of bilateral injuries.
4. Trans-articular external skeletal fixation is considered to augment the repair during healing and minimize the strain on small implants (which otherwise carry a risk of migration). Trans-articular external skeletal fixation is also of importance in this situation since collateral ligament rupture (partial or complete, ipsilateral or contralateral) may accompany malleolar

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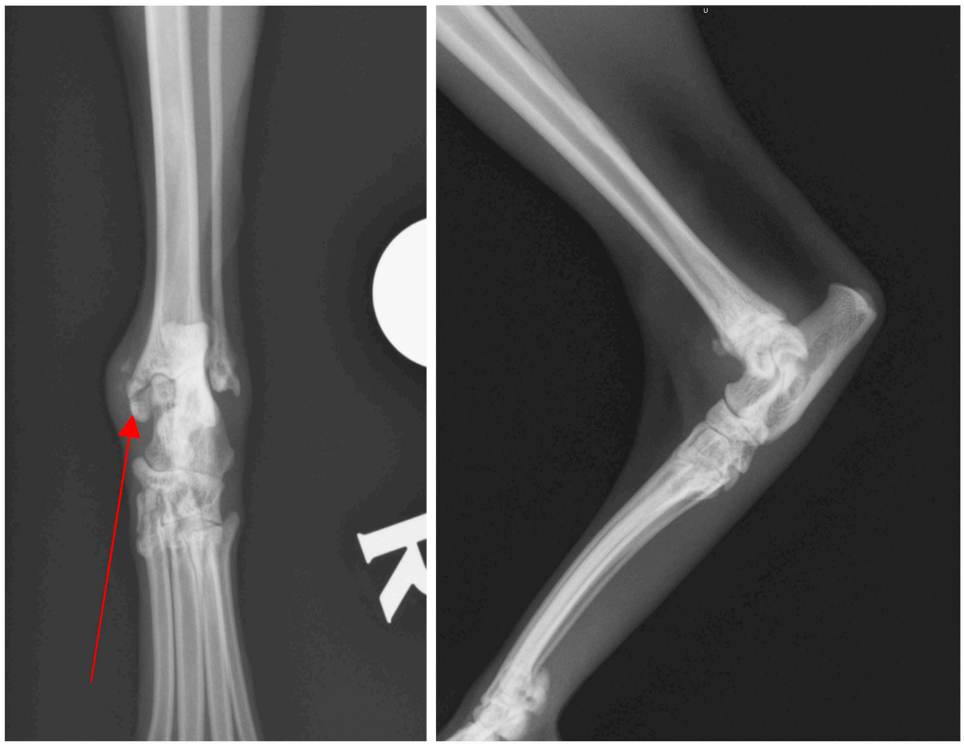


Figure 10.35 Orthogonal radiographic projections of the right hock of a cat demonstrating non-weight bearing lameness. The images reveal a medial malleolar fracture with minimal displacement and a small mineral density fragment within soft tissue swelling overlying the medial malleolus, which may also be apparent in the mediolateral projection, lying somewhat cranial to the talocrural joint. Hock effusion is well defined in the mediolateral projection. Note the angle at which a pin would need to be placed to purchase the medial malleolus and avoid penetration of the joint (red arrow).

fracture, and this is often difficult to determine as a result of gross talocrural instability. Temporary immobilization of the joint allows peri-articular fibrosis and healing of stretched peri-articular ligaments.

The prognosis is relatively good following appropriate management of malleolar fractures in cats, although implant-removal may be indicated if soft tissue irritation is present, persistent talocrural instability has been reported and the onset and progression of osteoarthritis is inevitable (though the influence on quality of life is unpredictable).

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Part 5 Fractures of the Tarsal Bones, Metatarsal Bones and Phalanges

(Kulendra & Arthurs 2014, Matthiesen 1983).

The tarsus consists of seven tarsal bones and four metatarsal bones. Fractures of the tarsus and/or metatarsus typically occur in combinations as a result of trauma, with small (often articular) chip fractures of multiple bones being common. It is rare to be faced with fractures of the individual bones, other than the calcaneus and treatment must aim to restore hock function (the most important joint being the talo-crural joint) and comfort. Radiographic assessment of hock injuries should be thorough, with stressed mediolateral views (flexion and extension) and caudocranial views (valgus and varus stress) revealing small chip fragments where they may otherwise go unnoticed. Computed tomography is of value in assessing fractures of the tarsus and metatarsus.

CALCANEAL FRACTURES:

Calcaneal fractures disrupt the tarsal extensor apparatus (Achilles tendon function). Therefore, cats may be non-weight bearing or walk with a plantigrade stance. Fractures occur more often near the base of the calcaneus than in the shaft or tuber.

The Achilles tendon displaces the proximal calcaneal fragment(s) proximally and internal fixation is needed properly to reduce and stabilize the fracture. Several methods of internal fixation can be used (Figure 10.36).

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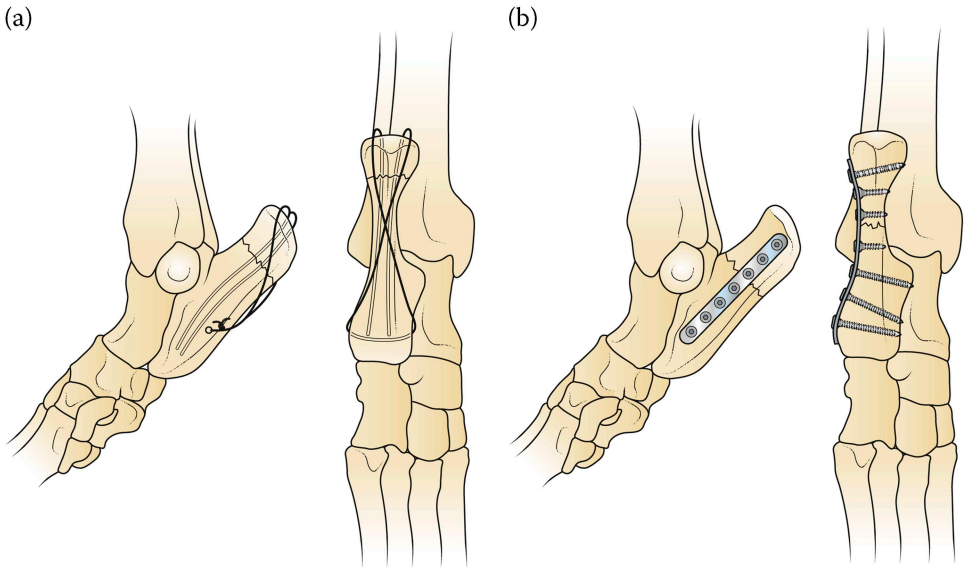


Figure 10.36 Stabilization of calcaneal fractures. **(a)** Lateral and plantar views of the hock with a calcaneal tuber fracture. Fixation is demonstrated with two parallel Kirschner wires and a tension band wire. The superficial digital flexor tendon must not be entrapped beneath the tension band wire as it passes over the proximal aspect of the calcaneus. The wire is crossed on the plantar aspect of the calcaneus and passed through a bone tunnel in the base of the calcaneus. The Kirschner wires are bent over at the proximal aspect of the calcaneus to stabilize the wire and prevent irritation of the superficial digital flexor tendon at that level. **(b)** Lateral and plantar views of the hock with a calcaneal body fracture. Mini plates may be applied to the lateral aspect of the calcaneus. Whilst the tension surface is the plantar aspect of the calcaneus, positioning a plate in this location causes interference with the overlying superficial digital flexor tendon. A laterally-applied plate is loaded sideways on, increasing the area moment of inertia, and is therefore biomechanically reliable.

Plate Fixation

The fracture(s) are reduced and stabilized using a bone plate applied to the lateral aspect of the calcaneus. In this location the plate undergoes 'edge-loading' and therefore has a high moment of inertia and resistance to bending.

1. A lateral approach is typically appropriate.
2. Mini plates and cuttable plates (with 1.0–2.4 mm screws) may be used, depending on the fracture conformation.
3. Small lag screws or Kirschner wires may be placed to stabilize comminuted fragments at the base of the calcaneus if necessary.
4. If the fracture is at the base of the calcaneus, the plate may bridge the calcaneoquartal joint to achieve increased bone stock for screw placement into the fourth tarsal bone. The joint space does not need to be debrided of articular cartilage since there is minimal movement at this level and ankylosis follows readily.

Tension Band Fixation

Tension band fixation converts the tensile force applied by the Achilles tendon into compressive force at the fracture. However, drilling Kirschner wires longitudinally through the feline calcaneus is complicated and accidental maldirection (and emergence of the pins in the wrong location) is not uncommon.

1. A plantarolateral approach is performed.
2. The superficial digital flexor tendon is reflected medially from the proximal end of the calcaneal tuberosity to allow normograde insertion of Kirschner wires into the calcaneus. Predrilling may be necessary, although the small feline calcaneus typically only accepts 1.1 mm diameter Kirschner wires, in which case predrilling may be unnecessary.
3. The fracture is reduced, and the Kirchner wires are driven distally across the fracture site and seated into the base of the calcaneus.
4. A hole is drilled from lateral to medial across the base of the calcaneus distal to the fracture.
5. Narrow gauge orthopaedic wire is placed in a figure-of-eight pattern through the hole and around the proximal ends of the Kirschner wires protruding from the calcaneal tuberosity (alternatively, the wire may be passed through a second bone tunnel drilled proximal to the fracture in which case the wire is likely to irritate the superficial digital flexor tendon). The wire is tightened and the twist is bent over and placed against the cortex of the bone to minimize irritation to the overlying soft tissues. The Kirschner wires are bent over to stabilize the wire and reduce irritation to the superficial digital flexor tendon.
6. If the fracture is at the base of the calcaneus, the Kirschner wires are inserted normograde from the tuber calcaneus, through the base of the calcaneus across the calcaneoquartal joint and into the fourth tarsal bone. The figure-of-eight wire is then placed through holes drilled in the calcaneal tuberosity and the fourth tarsal bone.

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Post-operative Care

Post-operatively, a soft padded bandage is recommended to minimize soft tissue swelling over two to five days. A lateral splint may be considered for up to 4 weeks to provide additional support for comminuted calcaneal fractures.

TALAR FRACTURES:

Fractures of the talus are relatively common in cats and may involve the neck, body, base or trochlear ridges. Comminuted fractures occur frequently. Fractures of the trochlear ridges affect the talocrural articular surface. The diagnosis is based on careful radiographic evaluation of the joint. Oblique views with the joint flexed and extended may help confirm the presence of a fracture. Since malleolar fractures and/or collateral ligament trauma commonly accompany talar ridge fractures, valgus and varus stress may also be employed to permit assessment of the talar ridges. However, computed tomography is invaluable in assessing talar fractures and is the modality of choice.

Treatment may involve hock immobilization, fragment removal and/or internal fixation. Where comminution is severe, arthrodesis should be considered.

Temporary Talocrural Immobilization

Where fragments are too small for internal fixation but are minimally displaced, immobilization of the hock for 4–6 weeks may permit healing, restoring comfortable function. A cast or lateral splint may be considered, however, the optimal approach to minimize movement in the joint is trans-articular external skeletal fixation. The joint should be immobilized in a functional, weight-bearing position. With intraarticular fractures, osteoarthritis is inevitable, although the clinical effect is unpredictable.

Fragment Removal

Small fragments of the proximal or distal trochlear ridges may be considered a source of discomfort and inflammatory mediator release within the joint. The health of the joint may be better served by their removal than any attempt to fix them in position (risking iatrogenic trauma to the joint and malreduction). The proximal talar ridges are approached via an incision caudal to the distal tibia with the hock flexed, while the distal talar ridges are approached via an incision cranial to the distal tibia with the hock extended.

Internal Fixation

If fracture fragments are of sufficient size, they may be stabilized using small Kirschner wires (0.6–0.9 mm) or bone screws (1.0–1.5 mm). Kirschner wires are inserted and countersunk below the articular surface.

Fractures of the talar neck are stabilized using Kirschner wires or a positional screw inserted from the head of the talus (which tends to displace dorsally) into the calcaneus (which is located plantar and lateral to the talar head).

OTHER TARSAL BONE FRACTURES:

Surgical fixation of fractures of the smaller tarsal bones is difficult in cats. Typically such fractures will be associated with malleolar, calcaneal and/or talar fractures, and they may be managed as part of the management of the more severe injuries through temporary immobilization (allowing complete fracture healing or development of a functional non-union) or inclusion within partial or pan tarsal arthrodesis (see disorders of the hind paw). External coaptation, temporary immobilization by external skeletal fixation (which may or may not bridge the talocrural joint and include the tibia) and partial tarsal arthrodesis are good options for fractures of the tarsal bones not involving the talocrural joint, depending on their severity.

METATARSAL AND PHALANGEAL FRACTURES:

Metatarsal and hind paw phalangeal fractures are managed in the same manner as the metacarpal and phalangeal injures in the forelimbs (see Chapter 8).

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11 Other Disorders of the Hindlimb

Part 1 Disorders of the Pelvis and Hips

COXOFEMORAL LUXATION:

(Ablin & Gambardella 1991, Ash et al. 2012, Basher et al. 1986, Bennet & Duff 1980, Braden & Johnson 1988, DeVita 1952, Ehmer 1934, Flynn et al. 1994, Fox 1991, Hunt & Henry 1985, Johnson & Braden 1987, Mehl 1988, Meij et al. 1992, Pratesi et al. 2012, Schlag et al. 2019, Sissener et al. 2009, Wildgoose 1983).

The coxofemoral joint is the most commonly luxated joint in cats (Figure 11.1). The majority of hip luxations are craniodorsal. Ventral and caudal luxations occur infrequently and may be accompanied by fracture of the greater trochanter (avulsion fracture involving the insertions of the middle and deep gluteal muscles). Femoral head avulsion fractures (of the insertion of the ligament of the femoral head) may also accompany hip luxation. Articular cartilage damage results from the initial trauma to the joint, from the abrasion of the femoral head against pelvic bone and from lack of lubrication and nourishment normally provided by the synovial fluid during loading. Osteoarthritis is therefore inevitable, although the severity and whether clinical signs follow is unpredictable.

The clinical findings in cats with coxofemoral luxation may include:

Lameness (usually non-weight-bearing initially).
Resentment to hip manipulation.
External rotation and adduction of the affected limb.
Palpable asymmetry of the greater trochanters.
Increased distance between the greater trochanter and ischiatic tuberosity on the affected side.
Apparent shortening of the affected limb with craniodorsal luxations.



Figure 11.1 Ventrodorsal radiographic view of the pelvis of a cat with hips extended showing luxation of the left coxofemoral joint.

A diagnostic examination exists: by placing a thumb in the ischiatic notch between the greater tochanter and ischial tuberosity. External rotation of the femur will not displace the thumb from the notch. This technique is also valuable for assessing the joint after closed reduction. With the femoral head seated in the acetabulum, the thumb will be displaced from the ischiatic notch with external rotation of the femur.

DIAGNOSIS:

Radiographic evaluation of the hip is required to confirm the luxation, determine the direction of the luxation and evaluate for other abnormalities (acetabular fractures, femoral neck fractures, slipped capital physis, hip dysplasia).

TREATMENT AND PROGNOSIS:

Conservative Treatment

Conservative treatment of coxofemoral luxation has been reported in cats. A pseudoarthrosis develops between the luxated femoral head and the caudal ilium, which allows limited function. Chronic lameness is likely to be present in the long term. This approach is not recommended and should not be a first-line approach; however, where no surgical alternatives are available and closed reduction has failed repeatedly, prior to consideration of any irreversible surgery (ie amputation) or euthanasia, it may be attempted.

Reduction and Stabilization

Hip reduction should be performed as soon as possible after injury to minimize cartilage trauma, re-establish cartilage loading and minimize muscle spasticity and fibrosis which hamper reduction.

Plain radiography and/or computed tomography aid decision-making regarding whether management will be closed or open. Closed reduction is not suitable where acetabular or femoral head fractures are present or in cases with advanced osteoarthritis, in which the prognosis for restoration of a comfortable joint is poor.

CLOSED REDUCTION:

Closed Reduction and Ehmer Sling

Closed reduction is generally thought to carry a high success rate, although true numbers are not available, since the rate at which this procedure is performed in general practice is unknown. In fact, studies of closed reduction of canine coxofemoral luxation performed at referral veterinary centres show re-luxation in 44–50% of dogs. In addition, it was reported that 50% of dogs had soft tissue injuries as a result of the sling, 40% of which were classified as severe. Closed reduction should be attempted, provided

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imaging does not reveal a genuine reason for likely failure; however, care should be taken when applying an Ehmer sling, and clients should be educated in monitoring of the sling for possible complications.

Procedure for reduction of craniodorsal coxofemoal luxation:

1. General anaesthesia is required to relax muscles and eliminate pain during hip manipulation.
2. The cat is positioned in lateral recumbency with the luxated limb uppermost.
3. A small towel or rope is passed around the base of the limb in the cat's inguinal area and held by an assistant to provide countertraction and stabilization of the pelvis.
4. The limb is externally rotated (grasp the stifle and hock) to disengage the femoral head from the dorsal acetabular rim.
5. The limb is pulled distally and caudally to align the femoral head over the acetabulum. Application of forces in a similar direction may be aided by simultaneous pressure through a thumb positioned on the greater trochanter.
6. The limb is then internally rotated and abducted to reduce the joint.
7. Digital pressure is applied to the greater trochanter in distal and medial directions to guide the femoral head into the acetabulum.
8. The reduced joint is manipulated through a full range of motion maintaining pressure on the greater trochanter. This stage is suggested to remove blood clots, joint capsule and other soft tissues from the acetabulum. The stability of the joint is also assessed during this manipulation. Reduction is confirmed by radiography.
9. A non-weight-bearing sling (Ehmer sling) may be applied to maintain internal rotation at the level of the hip temporarily. Radiographs are obtained after the sling is applied to ensure the joint is still reduced. There is no evidence that an Ehmer sling reduces the rate of re-luxation and successful maintenance of reduction of a luxated hip is possible without the sling.
10. The sling is removed after 10–14 days, at which stage the joint capsule and periarticular soft tissues are sufficiently healed to maintain reduction.

If the joint re-luxates during manipulation or application of external coaptation, then surgical options should be considered.

Closed Reduction and Ischio-Ilial Pinning

The insertion of an ischioilial pin (DeVita pin) has been reported for temporary maintenance of reduction following closed reduction, though it is difficult to place in the cat (because of a flat ilial wing) and risks trauma to the sciatic nerve and femoral head as a result of its 'blind' insertion technique. A 2.0 mm Steinmann pin is placed from ventral to the ischium, over the femoral head, and imbedded in the wing of the ilium. This procedure should be considered of historical interest and is not recommended.

Open Reduction

Open reduction allows exploration of the joint, removal of soft tissue entrapped in the acetabulum and internal stabilization. The success rate after open reduction and stabilization (85–100%) is significantly greater than after closed reduction. A craniolateral approach to the joint is usually sufficient. A trochanteric osteotomy may be considered to improve exposure, although is avoided if possible, since the gluteal muscles (cranially) and gemelli and obturator muscles (caudally) are important in stabilization of the hip, and elevating their insertion or transecting them, theoretically may influence the success of open reduction and stabilization. Techniques to maintain hip reduction include:

- **Capsulorrhaphy.** Torn joint capsule is sutured using permanent or relatively long-lasting suture material in a mattress or cruciate pattern. This should form part of the surgical management of every hip luxation, but, if clear edges are appreciated and the capsule can be reconstructed, it may be considered sufficient to provide stability. Since an open approach has been made to perform capsulorrhaphy it is reasonable to suggest that an additional stabilization technique should be considered to augment the repair. Additionally, it is rare for sufficient joint capsule to be available for a confidence-inspiring repair to be achieved.
- **Prosthetic capsule technique** (Figure 11.2). Two bone screws (2.0 mm, 2.4 mm or 2.7 mm) are placed in the dorsal acetabular rim at approximately the 10 o'clock and 1 o'clock positions for the left hip (or 11 o'clock and 2 o'clock for the right hip). The screws serve as origin points for sutures (3 M to 4 M polydioxanone or permanent multifilament braided suture or nylon). A hole is also drilled through the femoral neck for insertion of the sutures. The web of suture material prevents dorsal re-luxation. This technique is difficult to perform in small cats but has been successful in maintaining joint reduction.

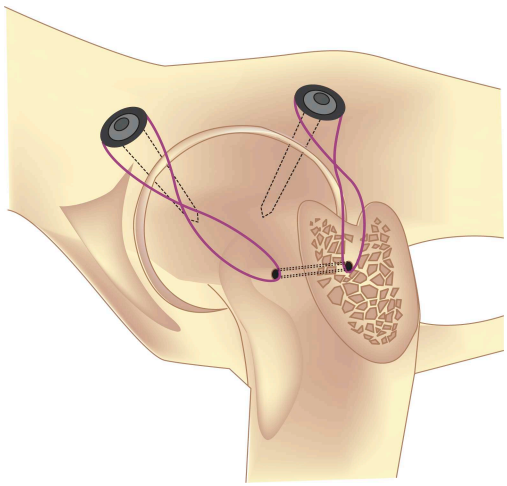


Figure 11.2 Prosthetic capsule replacement for stabilization of coxofemoral luxations. A craniolateral or dorsal approach to the hip joint is performed and two screws with washers are inserted into the dorsal acetabular rim at approximately the 10 o'clock and 1 o'clock positions for the left hip (or 11 o'clock and 2 o'clock for the right hip). A hole is drilled through the femoral neck and monofilament suture material is woven between the screw heads and the femoral neck to prevent reluxation.

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- **Extraarticular iliofemoral suture.** A hole is drilled from lateral to medial in the ventral ilium just cranial to the acetabulum. A second hole is drilled from caudal to cranial through the femur just distal to the greater trochanter. Suture material (3 M to 4 M polydioxanone or permanent multi-filament braided suture or nylon) is passed from lateral to medial through the hole in the ilium (grasping with a curved hemostat placed under the ventral edge of the ilial body helps) and then from cranial to caudal through the hole in the femur. The suture is tied with the joint reduced.
- **Transposition of the greater trochanter.** Figure 11.3 Fixation of a greater trochanteric osteotomy (performed to improve exposure to the dorsal acetabular rim) is performed using two parallel Kirschner wires and a figure-of-eight tension band wire. Distal transposition of the greater trochanter (followed by the same fixation method) has been reported to increase tension within the gluteal muscles, in turn increasing the medially-directed vector force between the femoral head and acetabulum. Osteotomy of the greater trochanter improves exposure to the joint for capsulorrhaphy or prosthetic capsule placement. During closure, the greater trochanter is simply transposed approximately 1 cm distally and caudally. Fixation is with two Kirschner wires and a figure-of-eight tension band wire anchored proximal to one or both Kirschner wires and through a bone tunnel at the level of the third trochanter (Figure 11.3). Transposition of the greater trochanter theoretically increases tension in the gluteal muscles with resulting compression of the femoral head into the acetabulum and internal rotation of the femur.

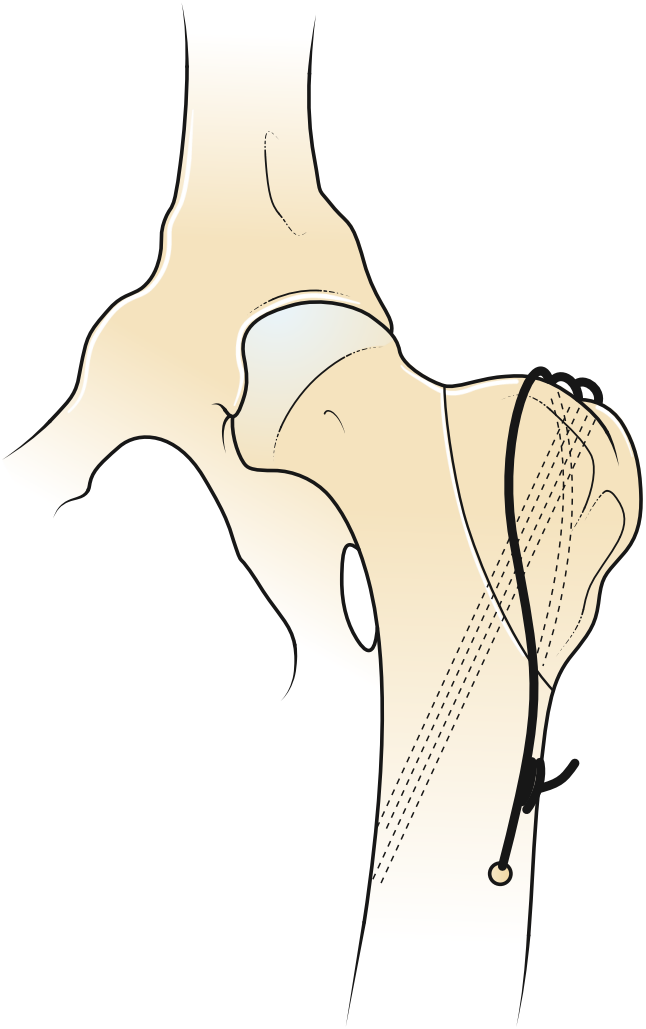


Figure 11.3 Fixation of a greater trochanteric osteotomy (performed to improve exposure to the dorsal acetabular rim) is performed using two parallel Kirschner wires and a figure-of-eight tension band wire. Distal transposition of the greater trochanter (followed by the same fixation method) has been reported to increase tension within the gluteal muscles, in turn increasing the medially-directed vector force between the femoral head and acetabulum.

- **Transarticular pinning** (Figure 11.4). A small Steinmann pin or Kirschner wire (1.6–2.0 mm diameter) is inserted through the femoral head and neck into the acetabulum to provide joint stability. The pin is inserted retrograde, starting at the fovea capitis and exiting near the third trochanter. The joint is then reduced while the pin is driven through the acetabular wall into the pelvic canal. An assistant evaluates pin depth by rectal examination. The lateral portion of the pin is then bent over and cut short. The pin remains in place for 2–6 weeks and then is removed via traction through a stab incision. This technique presumably traumatizes articular cartilage. A study documented 20 feline cases in which two had re-luxated 6 months following surgery.
- **Toggle rod fixation** (Figure 11.5). This procedure aims to replace the ruptured ligament of the femoral head with a prosthetic ligament. A hole is drilled retrograde (normograde has also been described anecdotally) through the femoral head and neck from the fovea capitis to the region of the third trochanter. A second hole is drilled in the centre of the acetabular fossa penetrating the medial acetabular wall. Toggle rods may be bought or made from a twisted narrow Kirschner wire (1.1 mm diameter). Suture material (3 M to 4 M polydioxanone or permanent multi-filament braided suture or nylon) is inserted through the hole in the centre of the toggle rod which is passed through the acetabular hole into the pelvic canal. Pulling the ends of the suture ensures that the toggle rod is positioned against the medial acetabular wall and will not pass back through the hole. The free ends of the suture are passed through the bone tunnel in the femoral head and neck, exiting near the third trochanter where they are secured, either through another bone hole drilled caudal to cranial through the lateral femoral cortex, or using a button. The sutures are tightened while the hip is held in a reduced position. Two studies in 2012 reported toggle rod stabilization of 18 feline cases between them. Two cases experienced re-luxation.

The preferred technique is currently a combination of toggle rod stabilization and capsulorrhaphy. An Ehmer sling may be considered following surgery but is not necessary for a successful outcome and may be of no benefit. The prognosis after coxofemoral luxation is good if reduction and stability are achieved soon after injury. Osteoarthritis is inevitable

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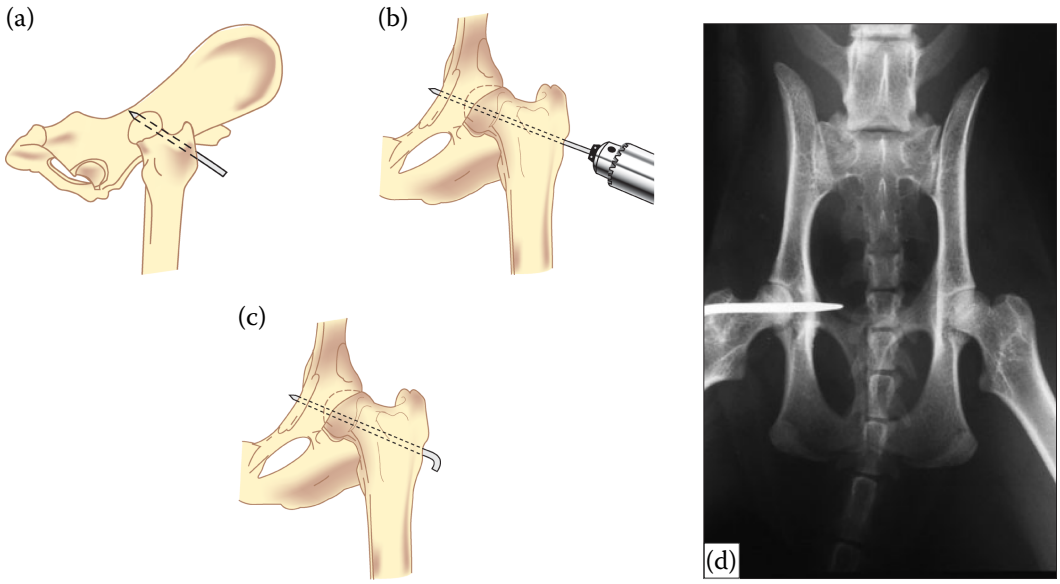


Figure 11.4 Transarticular pinning for stabilization of coxofemoral luxations. **(a)** A 1.6–2.0 mm diameter pin is inserted retrograde, starting at the fovea capitis and exiting near the third trochanter. The pin is advanced until the tip is at the level of the femoral head. **(b)** The joint is reduced and the limb is held in a weight-bearing position with slight abduction while the pin is driven through the acetabular wall into the pelvic canal. **(c)** The lateral end of the pin is bent over and cut short, leaving enough pin length to allow later removal. **(d)** Ventrodorsal radiographic view of the hip joint in a cat. The craniodorsal hip luxation is stabilized with a transarticular pin.

due to injury of the articular cartilage. Femoral head and neck ostectomy and total hip replacement should be considered if closed and open techniques are unsuccessful, or if severe femoral head damage is present.

HIP DYSPLASIA:

(Boileau et al. 2009, Davidson & Kerwin 2014, Freire et al. 2011, Hayes et al. 1979, Holt 1978, Keller et al. 1999, Koeppel & Ebner 1990, Langenbach et al. 1998, Lascelles et al. 2007, Lascelles et al. 2010, Lascelles & Robertson 2012, Patsikas et al. 1994, Robin et al. 1994, Slingerland et al. 2011, Smith et al. 1999).

Clinically relevant hip dysplasia has long been considered to be less common in cats than in dogs. The prevalence is reported at between 6.6% and 32%, and appears to be more common in large pedigree breeds (particularly Persian, Himalayan and Maine coon). It is an inherited disease (though heritability has not been determined) and occurs more commonly in females. It appears to have some association with patellar luxation, with both conditions being more common in combination than alone. In dogs, laxity is the consistent underlying cause of hip dysplasia, and this becomes apparent from about 12 weeks of age; however, in cats, laxity is less consistent, while a shallow acetabulum (which may be recognized earlier than 12 weeks of age) appears to be a more consistent feature.

Clinical Signs

Some cats with hip dysplasia show no clinical signs, and it may be noted as an incidental finding, for example, following a road traffic accident. However, increasingly it is becoming apparent that, while cats are able to mask lameness better than dogs, and cat owners tend to be less observant of their pets at exercise than dog owners (for obvious reasons), behavioural changes are a consistent feature of feline hip dysplasia, presumably indicating discomfort. Therefore, the clinical signs which are reported include inactivity, difficulty and reluctance to climb or jump, changed toileting behaviours (inappropriate defaecation). Clients may not recognize these features as indicators of hip dysplasia, or volunteer this information without prompting, so specific questioning should form a part of the diagnostic protocol.

Diagnosis

The diagnosis of hip dysplasia is based on an appropriate history with examination findings including resentment to manipulation of the hips and a reduced range of motion, with negative findings on further examination of the more easily palpated joints on the limb (stifle and tarsus) and of the spine. Remember that examination findings are not particularly reliable for the diagnosis of hip dysplasia in cats. In one study, only 36% of cats with radiographic hip osteoarthritis showed positive findings on examination.

Radiographic Findings (Figure 11.6)

Radiographic findings of hip dysplasia in cats include shallow acetabula. Even the normal cat has a shallower acetabulum than the dog. In dogs the depth of the normal acetabulum is often suggested to cover 50% of the femoral head by the dorsal acetabular rim. Such an assessment used to detect hip dysplasia in cats would be highly sensitive and poorly specific in cats (ie hip dysplasia would be overdiagnosed). Peri-articular new bone formation is certainly a feature of osteoarthritis secondary to hip dysplasia. However, laxity is a less reliable predictor of peri-articular new bone formation than it is in dogs, and cats seem tolerant of somewhat greater coxofemoral laxity. A study showed that no cats with a distraction

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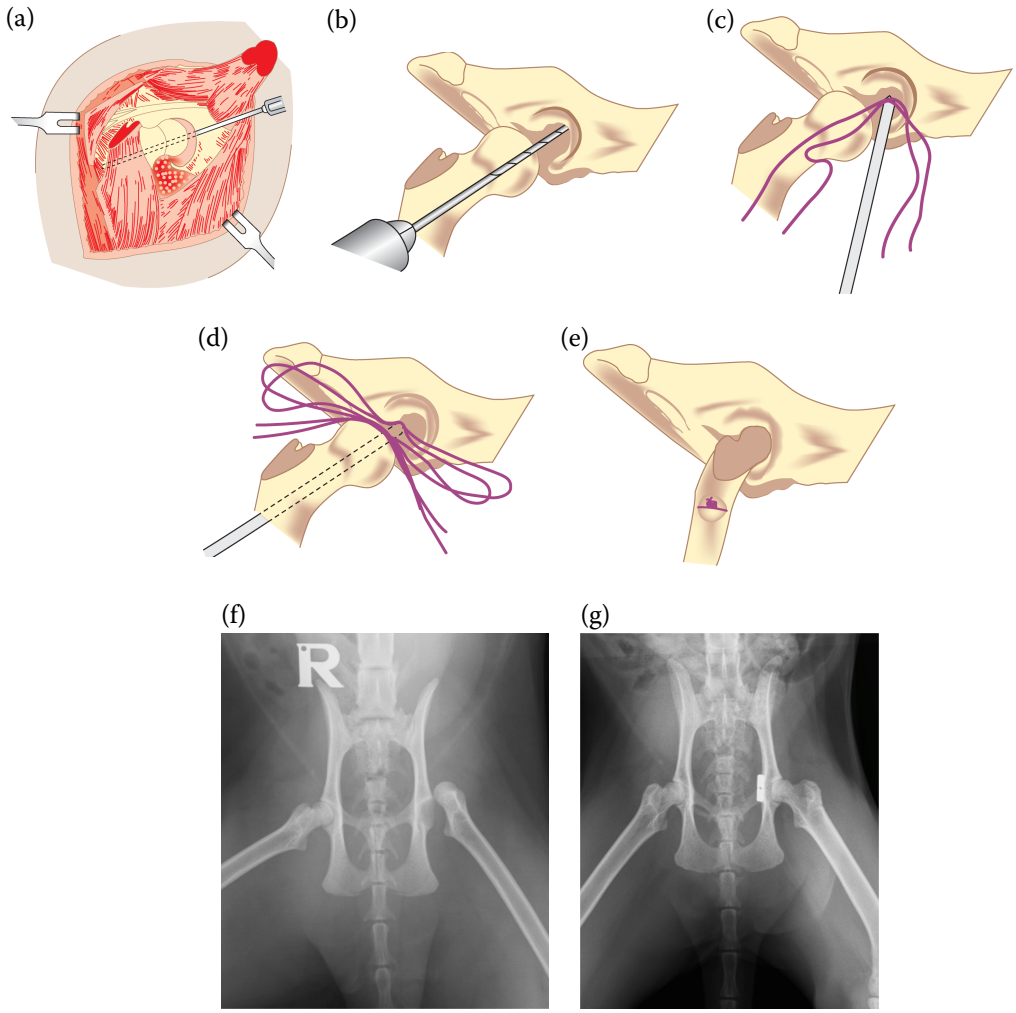


Figure 11.5 Toggle rod fixation for stabilization of coxofemoral luxations. **(a)** A hole is drilled through the femoral head and neck from the fovea capitis to the region of the third trochanter. **(b)** A hole is drilled in the centre of the acetabular fossa, penetrating the medial acetabular wall. **(c)** Suture material is attached to the toggle rod and it is inserted through the hole in the acetabulum. The toggle rod is positioned against the medial acetabular wall. **(d)** The suture is then passed through the bone tunnel in the femoral head and neck, exiting near the third trochanter. **(e)** The suture is secured on the lateral aspect of the femur either using a sterile button or by passing the suture through a transverse hole drilled in the femoral cortex and tying the suture ends together. Ventrodorsal radiographic projections of the pelvis and hips showing luxation of the left coxofemoral joint **(f)** and following reduction and fixation using a toggle rod method **(g)**. The metallic rod is observed on the medial aspect of the acetabulum and the bone tunnel through the femoral head, neck and proximal metaphysis, through which suture is passed, can be observed.

index (a measure of hip laxity) <0.4 demonstrated radiographic evidence of hip osteoarthritis, while for dogs this figure is 0.3. The mean Norberg angle in normal cats is 92.4°. It has been suggested that this difference reflects a generalized greater laxity in feline joints in comparison with canine joints. Remodelling of the femoral neck, often seen in dysplastic dogs, is uncommon in cats until osteoarthritis is relatively advanced. Overall, radiographic findings are poorly correlated with hip osteoarthritis in the cat, and 57% of cats with no radiographic change in one study demonstrated macroscopic cartilage damage.

Treatment

In most cases, conservative management of feline hip osteoarthritis is appropriate. For short-term flare-ups it is reasonable to trial restriction of activity for 2–4 weeks alongside medical management with a licensed non-steroidal antiinflammatory drug (NSAID). In this way acute flare-ups of the clinical signs may be managed; however, longer term management is likely to be more involved.

Environment Modification

For the longer term, management should include modification of the environment. Since access to litter trays is a problem for cats with hip osteoarthritis, low-sided litter trays should be considered. In addition, it should be recognized that outdoor access via a cat flap, and access to raised surfaces within the house may be avoided because climbing and jumping may cause discomfort. Modifications such that cats can step up onto surfaces, rather than needing to jump or climb, should be considered. It has also been suggested that improving access to areas of the house and outdoor environment may have a beneficial effect on the disease process itself, by encouraging

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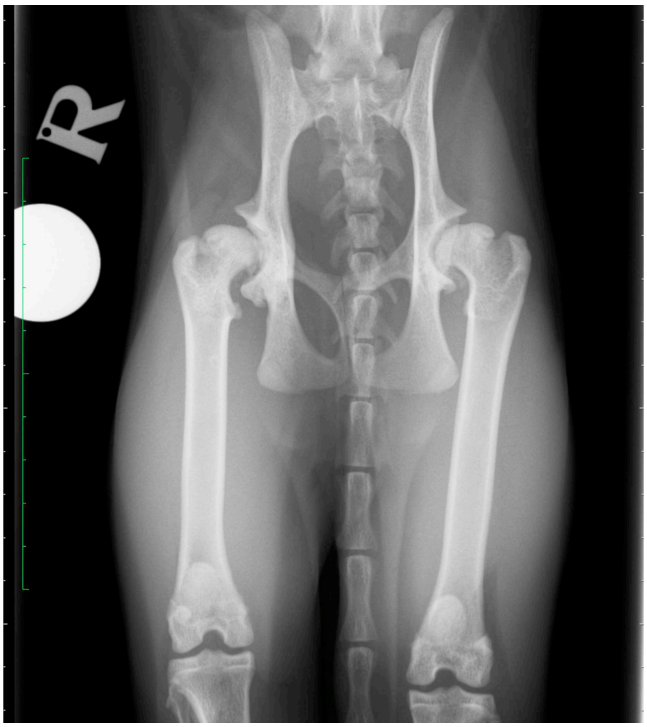


Figure 11.6 Ventrodorsal radiographic projection of the pelvis and hips of a cat demonstrating severe bilateral coxofemoral subluxation (very limited dorsal acetabular coverage of the femoral heads) and peri-articular new bone formation consistent with osteoarthritis secondary to hip dysplasia.

more movement, and thus preserving joint range of motion and hindlimb muscle mass.

Physiotherapy

There is limited evidence on which to base a physiotherapy plan for a cat with hip dysplasia (see Chapter 16). Additionally, many cats are intolerant of hydrotherapy. In short, both passive and active physiotherapy exercises would be expected to be of benefit to the feline patient with hip osteoarthritis by maintaining joint range of motion and reducing stiffness, maximizing muscle mass and encouraging joint loading, to improve articular cartilage metabolism and nutrient diffusion.

Diet and Nutraceuticals

A diet with a high omega-3 to omega-6 fatty acid ratio has been shown to improve activity levels in cats with hip dysplasia. Such a diet should be encouraged. In addition to the direct effects through modification of the prostaglandin concentrations within osteoarthritic joints, a diet high in omega-3 fatty acids may aid bodyweight reduction. Optimization of bodyweight may be the single most important facet of conservative management of feline hip osteoarthritis, particularly where the patient is overweight on initial presentation. There is little or no evidence that supplementation of glucosamine or chondroitin is likely to be of benefit for hip dysplasia in cats. Avocado soybean unsaponifiables (ASU) is available for cats as a further dietary supplement which may prove to be NSAID sparing in the management of osteoarthritis.

Surgical Options

The limited options available for the skeletally immature dog to improve hip conformation and slow the progression of osteoarthritis (namely juvenile pubic symphysiodesis and double/triple pelvic osteotomy) are not documented for the cat. Surgical options are limited to femoral head and neck ostectomy and total hip replacement.

Femoral Head and Neck Ostectomy

(Berzon et al. 1980, Gendreau & Cawley, 1977, Liska et al. 2009, Off & Matis 2010, Yap et al. 2015).

Indications for femoral head and neck ostectomy (FHNO, also known as femoral head and neck excision) include recurrent hip luxation, severe fractures or malunion of the acetabulum and/or femoral head and neck (including spontaneous femoral capital physeal fracture), and coxofemoral osteoarthritis.

The aim is to remove the femoral head and sufficient femoral neck to create a smooth surface which may glide over the lateral aspect of the acetabulum and caudal ilium Figure 11.7. Lateral radiographic projections of a cat with recurrent cranioventral coxofemoral luxation following trauma. (a) lateral view demonstrating cranioventral luxation. (b) lateral view following total hip replacement, demonstrating cement (polymethylmethacrylate) mantle surrounding femoral component within the femoral medullary cavity and metallic marker ring within the ultra-high molecular weight polyethylene acetabular component (itself invested with a cement mantle). The prognosis has been reported as satisfactory to good in some studies, but was inconsistent in another study, with the main concern in that study being dorsal displacement of the operated limb. The most recent study of functional outcome in cats with femoral head and neck ostectomy (FHNO) used a non-validated owner-completed questionnaire to evaluate client-perceived outcome in 18 cats. Results of that study were found to demonstrate good to excellent functional outcome in 17 of the 18 cats. Cats took between 1 and 2 months to return to normal function according to clients. Long-term analgesia was not provided for any cats.

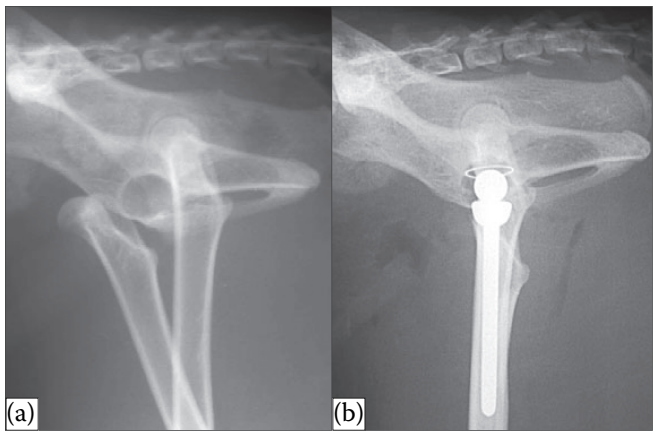


Figure 11.7 Lateral radiographic projections of a cat with recurrent cranioventral coxofemoral luxation following trauma. **(a)** lateral view demonstrating cranioventral luxation. **(b)** lateral view following total hip replacement, demonstrating cement (polymethylmethacrylate) mantle surrounding femoral component within the femoral medullary cavity and metallic marker ring within the ultra-high molecular weight polyethylene acetabular component (itself invested with a cement mantle).

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FHNO technique:

1. The cat is positioned in lateral recumbency
2. A skin incision is made over the cranial aspect of the hip joint, followed by an incision in the fascia lata along the cranial border of the biceps femoris muscle. The biceps femoris muscle is retracted caudally (protecting the sciatic nerve) and the superficial and middle gluteal muscles are retracted dorsally. The deep gluteal muscle is also retracted dorsally and its partial tenotomy is not typically required.
3. An incision is made in the joint capsule cranial to the femoral head to expose the entire femoral neck.
4. The femur is externally rotated and curved scissors are used to transect the ligament of the head of the femur.
5. The femur is then externally rotated 90° to luxate the femoral head from the acetabulum and allow visualization of the entire femoral head and neck. In this position, the patella is pointing straight up and the surgeon is seeing the cranial aspect of the femur through the incision. This exposure and positioning is important to ensure a proper ostectomy is performed.
6. The ostectomy is performed with a sagittal saw where possible. Otherwise a sharp osteotome and mallet may be used. The osteotomy follows a line from the lateral aspect of the intertrochanteric notch proximally to just proximal to the lesser trochanter distally. Care is taken not to perform the ostectomy along a line directed perpendicular to the axis of the femoral neck, since this location does not account for anteversion of the femoral head and will fail to remove a portion of the caudal femoral neck.
7. When the ostectomy is completed, the femoral head and neck are grasped with forceps and the remaining soft tissues are excised. The ostectomy site is palpated to ensure it is smooth. Any bone spurs are removed.

After femoral head and neck excision arthroplasty, a few days' rest should be encouraged while the soft tissues heal. Passive physiotherapy is commenced during this time. Thereafter indoor exercise and active physiotherapy promote development of a lax pseudoarthrosis.

Total Hip Replacement

(Borak et al. 2017, Fitzpatrick et al. 2012,Kalis et al. 2012, Liska 2010, Liska et al. 2009, Liska et al. 2010, Marino et al. 2012, Witte et al. 2010).

Indications for total hip replacement are as for femoral head and neck ostectomy. These include recurrent hip luxation (Figure 11.7), severe fractures or malunion of the acetabulum and/or femoral head and neck (including spontaneous femoral capital physeal fracture), and coxofemoral osteoarthritis.

Currently only one commercially available (cemented) implant system is available for cats; however, more may become available in future. Total hip replacement should theoretically restore a normal coxofemoral range of motion and does not rely on a soft tissue pseudoarthrosis. Therefore, normal or near-normal comfort should be achievable. Outcome for total hip replacement has been suggested to be superior to that following femoral head and neck ostectomy, though direct studies with reasonable numbers of cases do not exist.

Studies of feline total hip replacement have reported a total of 33 cases to date. Outcomes have been reported to be good or excellent, though outcome measures have been subjective. Only two complications have been reported: luxation and aseptic loosening of the acetabular component, both of which were addressed successfully through second surgeries. However, some studies have documented feline and canine total hip replacements together, reporting complications, but not specifying whether these were in canine or feline patients, so an overall complication rate cannot be given.

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Part 2 Femur and Stifle Disorders

The stifle comprises the femorotibal and femoropatellar articulations, a single common joint capsule and ligamentous support structures. The cruciate ligaments (cranial and caudal) and collateral ligaments (medial and lateral) provide stability to the stifle joint. Traumatic disruption of these ligaments and luxation of the patella (traumatic or congenital) are common causes of stifle pain and lameness in cats. Cranial cruciate ligament disease is a common cause of hindlimb lameness in the dog and is increasingly frequently reported in cats in the veterinary literature.

PATELLAR LUXATION:

(Arthurs & Langley-Hobbs 2006, Boone et al. 1983, Brioschi et al. 2020, Davies & Gill 1987, De Sousa et al. 2014, Engvall & Bushnell 1990, Flecknell & Gruffydd-Jones 1979, Houlton & Meynink 1989, Johnson 1986, Johnson et al. 2001, L'Eplattenier & Montavon 2002, Loughin et al. 2006, Prior 1985, Rutherford & Arthurs 2014, Rutherford et al. 2015, Smith et al. 1999, Talcott et al. 2000, Yasukawa et al. 2016).

Medial patellar luxation is not uncommon in cats, though it has been reported infrequently in the published literature. There is a greater degree of laxity in the normal feline patellofemoral joint than in the canine patellofemoral joint, and the trochlear groove is wider and flatter (relative to body size) with a wider patella. As with the dog, the terminology regarding whether patellar luxation should be considered congenital (ie present from birth) or developmental remains unclear. 95% of cases are medial and grades 2 and 3 are most common. 81% are bilateral. Two reviews of cats with patellar luxation reported that 66–100% demonstrated lameness ranging from very mild (and intermittent in some cases) to very severe, with a poor correlation between lameness severity and grade of luxation. Femoral and/or tibial deformities (distal femoral varus, proximal tibial valgus, etc.) have been rarely reported in association with feline patellar luxation, although tentative associations with hip dysplasia, previous femoral surgery and cranial cruciate ligament rupture have been variably reported.

Diagnosis

The diagnosis of patellar luxation is made by digital evaluation of patellar stability. Other grading systems for feline patellar luxation have been described; however the system utilized for dogs applies without modification to cats:

- Grade I: The patella may be manually luxated, but reduces spontaneously when pressure is released.
- Grade II: The patella may be manually luxated using digital pressure or with rotation of the tibia. The patella will remain luxated, but may spontaneously reduce during stifle manipulation.
- Grade III: The patella is luxated most of the time, but may be reduced manually. It will re-luxate spontaneously.
- Grade IV: The patella is permanently luxated and cannot be reduced with digital manipulation or rotation of the tibia.

Treatment may be conservative or surgical. Conservative therapy essentially consists of moderate activity restriction for 4–6 weeks alongside non-steroidal anti-inflammatory oral medications. The prognosis for a good quality of life and return to normal activity is relatively good for cats presenting with low grade lameness and low grade patellar luxation.

For more severely affected cats (in terms of lameness and/or grade) surgical management is appropriate. For surgical management of patellar luxation to be successful, planning should involve an investigation of the underlying cause(s) of patellar luxation, and these should be addressed individually. Surgical stabilization of the feline patella has been reported to involve combinations of:

- Medial parapatellar soft tissue release (for medial patellar luxation)
- Lateral parapatellar soft tissue imbrication (for medial patellar luxation)
- Anti-rotational/de-rotational suture placement
- Recession trochleoplasty (deepening of the trochlear groove)
- Tibial tuberosity transposition
- Partial patellectomy

Parapatellar Soft Tissue Procedures

In cases of medial patellar luxation the parapatellar soft tissues may be shortened and thus act to resist reduction of the patella (or to encourage its re-luxation following surgery). Medial release involves extending an incision in the joint capsule proximally. Since the Sartorius muscle does not divide distinctly into cranial and caudal parts as in the dog, this often involves an incision across the aponeurosis of this muscle's insertion caudally. The release should extend between the vastus medialis and its medial fascial attachments to the fascia overlying semimembranosus and the other hamstring muscles.

Imbrication of the loose lateral parapatellar soft tissues is often performed to apply laterally-directed force to the patella and help to maintain its reduction. An elliptical portion of joint capsule is excised and the edges are sutured, or the edges of joint capsule are overlapped using a vest-over-pants technique. The lateral joint capsule is imbricated to stabilize medial patellar luxations; the medial joint capsule is imbricated to stabilize lateral patellar luxations.

Anti-Rotational/De-Rotational Suture

In some dogs a rotational deformity at the level of the stifle (primarily internal rotation of the tibia with respect to the stifle in association with medial patellar luxation) may be implicated in patellar luxation. Rotational deformity may be present continuously (static rotational deformity) or may be present particularly during flexion of the stifle (dynamic rotational deformity). Such a deformity has been reported to be of greater importance than tibial tuberosity medialization in the aetiology of patellar luxation in dogs but has not been reported

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in cats. Therefore, use of an anti-rotational suture, to prevent internal rotation of the tibia, or a de-rotational suture, to realign the tibia with respect to the femur at the level of the stifle may be considered if appropriate. Evaluation of rotation of the tibia with respect to the femur is most accurately performed using computed tomography so this modality should be considered where this deformity is suspected.

The technique involves placing a suture around the lateral fabella and through a hole drilled in the tibial tuberosity. Monofilament, nonabsorbable suture material (3–4 metric) is appropriate. Such a suture is likely to stretch or snap since its positioning is not isometric (i.e. the separation of the origin and insertion points will alter through the stifle range of motion), and, the feline femoro-fabella ligament has been hypothesized to be relatively lax, so a taut suture placed in this way will not be maintained for the long term. As such, the importance of this procedure in helping to prevent re-luxation of the patella following surgery is questionable.

Fabello–patellar suture

A suture is passed in a figure-of-eight pattern from the fabella to the patella. The suture can be passed around the patella or placed through the patellar tendon. The suture is tightened just enough to stabilize the patella. This procedure is rarely employed, and should be considered adjunctive only. It is not a stand-alone technique.

Bony Reconstructive Procedures

Trochleoplasty

Trochleoplasty techniques are used to deepen the trochlear sulcus and increase the resistance to luxation of the patella. Several techniques have been described. They are performed following lateral (for medial patellar luxation) or medial (for lateral patellar luxation) arthrotomy and luxation of the patella to allow access to the trochlear groove.

- **Sulcoplasty** involves rasping the articular surface and subchondral bone of the trochlear groove, deepening the groove but also destroying the articular surface. This technique should be considered historical only.
- **Trochlear wedge recession** (Figure 11.8) involves use of a small saw to excise a wedge-shaped osteochondral fragment from the trochlea, excision of a small amount of bone deep to the resultant femoral defect and replacement of the osteochondral fragment. The replaced wedge is thus recessed, creating a deeper trochlear sulcus. Fixation of the wedge is not necessary.
- **Rectangular block recession trochleoplasty (**Figure 11.9) is similar to the wedge recession; however, three osteotomies are made: two parallel osteotomies just axial to the trochlear ridges and a third osteotomy made with an osteotome perpendicular to the first two. A small section of bone is removed from the resultant femoral defect (and from the base of the excised ostechondral block) and the fragment is replaced. Block recession trochleoplasty recesses a larger percentage of the articular surface than wedge recession. Given the broader patella of the cat relative to the dog, trochlear block recession trochleoplasty, which deepens the groove more efficiently at its proximal extent, should be considered the optimal approach to groove deepening.

Tibial Tuberosity Transposition

The purpose of transposition of the tibial tuberosity is to realign the quadriceps mechanism (within which the patella is located) such that it remains aligned over the cranial aspect of the distal hindlimb, encouraging the patella to remain located in the trochlear groove. Realigning the quadriceps mechanism should be considered the most important component of surgical management of patellar luxation.

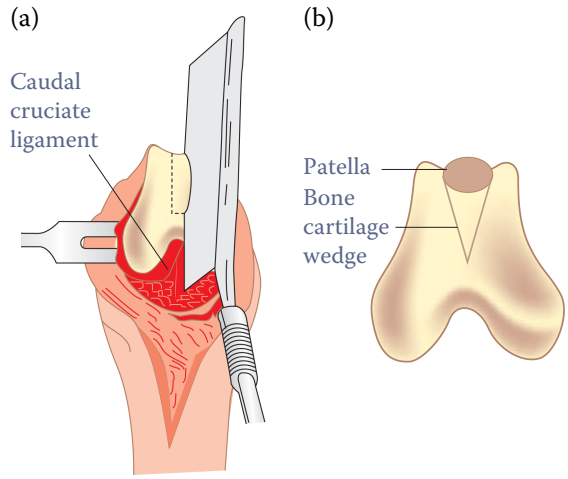


Figure 11.8 Trochlear wedge recession. **(a)** A saw is used to excise a wedge-shaped osteochondral fragment from the trochlea, preserving the articular surface. **(b)** Bone is removed from the wedge and the femoral defect and the osteochondral fragment is replaced (recessed), deepening the trochlear sulcus.

1. The tibial tuberosity is osteotomized with a finger saw or fine sagittal saw. The osteotomy begins just caudal to the insertion of the straight patellar ligament. Where possible the fascial attachment at the distal aspect of the tuberosity remains intact.
2. The tibial tuberosity is displaced laterally to correct medial patellar luxation (or medially to correct lateral patellar luxation). Alignment is confirmed by attempting to luxate the patella in either direction. Once the patella reduces spontaneously following attempts to luxate it, the tibial tuberosity has been displaced sufficiently. In most cases, the tuberosity is only displaced a few millimetres to achieve proper alignment.
3. Fixation of the tibial tuberosity involves two Kirschner wires (1.0 mm to 1.5 mm) driven through the tuberosity and into the proximal tibial metaphysis to exit its caudal cortex. Care is taken not to place the Kirschner wires into the stifle joint or into the soft tissues on the caudal aspect of the tibia. Kirschner wires are driven until a loss of resistance indicates emergence from the caudal tibial cortex and then are stopped immediately. Driving too far and then drawing the Kirschner wire back increase the risk of pin migration.
4. A figure-of-eight tension band wire (18 or 20 gauge) is applied over the top of the proximal Kirschner wire and through a bone tunnel in the cranial tibial cortex distal to the tibial tuberosity. The wire should be tightened medially and laterally to ensure even tension throughout its length.
5. With the tuberosity transposed, the tibia is internally and externally rotated and the stifle flexed and extended to confirm patellar stability.

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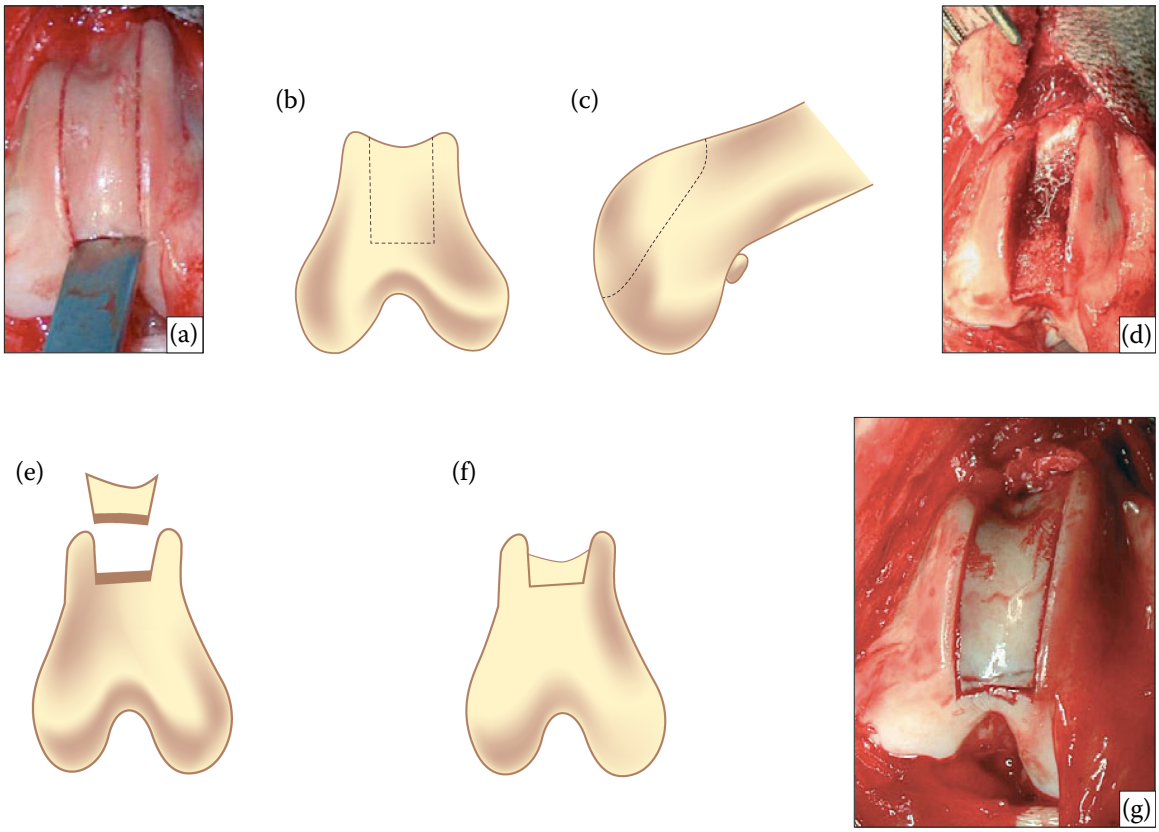


Figure 11.9 Groove deepening by rectangular block recession trochleoplasty (photographs show views of the distal femur of a cat during surgery for medial patellar luxation (the tibial tuberosity is in the foreground, with the patella luxated medially to the right of the images). **(a, b, c)** a saw and small osteotome are used to make osteotomies as shown such that a rectangular osteochondral fragment can be removed from the trochlear sulcus **(d)**. Bone is removed either from the base of the osteochondral block and/or from the defect in the sulcus. When the block is replaced into the defect it is recessed. **(e** and **f) (g)** following groove deepening by rectangular block recession trochleoplasty the femoral articular surface of the femoropatellar joint is recessed between the trochlear ridges.

In one study of feline patellar luxation surgery 28% of tibial tuberosity transposition cases had complications. However, in cases where tibial tuberosity transposition was not performed, the re-luxation rate appeared to be higher (this was not statistically confirmed, though has been reported to be statistically significant in dogs). The authors perform tibial tuberosity transposition using the above technique routinely for patellar luxation in cats.

Partial Patellectomy

Partial (para)sagittal patellectomy is a technique performed in cats with patellar luxation in response to what has been recognized as a wide patella relative to the width of the trochlear groove. First described in a case series of four cats, the procedure was recently assessed in a cadaveric study in which the fit of the patella into recessed trochlear grooves was compared between unaltered patellas and those which had undergone partial patellectomy (removal of a sagittal sliver of patella from the lateral aspect, medial aspect or both sides). The findings of that study were that patellar recession was no improved by groove deepening alone but partial patellectomy resulted in greater recession of the patella between the raised trochlear ridges. The conclusion of that study was that partial patellectomy should be considered a mainstay of patellar luxation treatment in cats. Cats do appear to have a relatively wide patella, so it is appropriate to confirm that the width of any groove deepening technique performed is sufficient to accommodate the patella and if not, partial patellectomy is an option for improving patellar recession into the deepened groove.

Prognosis Following Patellar Luxation Surgery

Complications for feline patellar luxation surgery were reported to occur in 26% of cases, the majority of which were major complications (including 5% patellar re-luxation). The prognosis following successful surgical management for patellar luxation in cats is good to excellent. Where groove deepening and tibial tuberosity transposition are performed management should involve the standard 4 weeks of crate confinement, 4 weeks of room confinement and 4 weeks of house confinement prior to a reintroduction of outdoor activity.

CRANIAL CRUCIATE LIGAMENT RUPTURE:

(Allan 2014, Boge et al. 2019, Bilmont et al. 2018, De Sousa et al. 2014, De Sousa et al. 2015, Harasen 2005, Hoots & Petersen 2005, Kneifel et al. 2018, McLaughlin 2002, Mindner et al. 2016, Retournard et al. 2016, Ruthrauff et al. 2011, Scavelli & Schrader 1987, Schnabl et al. 2009, Stadig et al. 2016, Voss et al. 2017).

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The feline cranial cruciate ligament (CCL) limits stifle hyperextension, internal tibial rotation with respect to the femur, and cranial tibial translation (or 'drawer'). Failure of the ligament permits joint instability. While the instability itself undoubtedly results in inflammation within the joint and abnormal cartilage wear (triggering osteoarthritis), there is good evidence in the dog and increasingly in the cat, that early degeneration of the ligament is associated with an arthropathy (which is primarily mononuclear) and lameness, prior to the presence of detectable stifle instability. The influence of hormones, genetics, joint inflammation and conformation on CCL rupture in cats has not been evaluated to the same extent as in dogs. However, cats with CCL rupture are often overweight and one study showed that cats with cranial cruciate ligament rupture had tibial plateau angles on average 3.1° greater than cats with no stifle pathology.

Trauma has also been reported to be a cause of CCL rupture in the cat, theoretically when the ligament undergoes supraphysiological loading, presumably through severe internal tibial rotation, stifle hyperextension, or cranial translation of the tibia with respect to the femur.

Clinical Signs

Signs of CCL rupture include a sudden onset lameness (may be non-weight bearing), stifle pain, and joint effusion. The lameness usually improves with rest, but may worsen after exercise.

Diagnosis

The diagnosis of CCL rupture relies on the findings of physical examination and radiography.

Physical examination reveals a positive cranial drawer sign, (the tibial compression test may be performed but is somewhat harder to interpret than in the dog, due to a greater overall laxity in the cat), stifle effusion and resentment to manipulation of the joint. With more chronic ruptures, periarticular thickening and crepitus may be apparent. Joint laxity usually persists even in chronic cases of CCL rupture.

Radiography of the stifle may reveal joint effusion and peri-articular ossification consistent with osteoarthritis (Figure 11.10). Unlike the dog, intra-articular mineralization is also common in the cat. Large mineralizations in the feline stifle are often present in association with osteoarthritis and



Figure 11.10 Radiographic inspection of the feline stifle joint. **(a)** Effusion is recognized in this mediolateral radiographic projection of the right tibia and stifle of a cat. The fat pad is visible as a narrow sliver of fat opacity between the patellar ligament and effusion (both of which are of soft tissue opacity). A small intra-articular mineralization is also apparent consistent with mineralization of the cranial aspect of the medial meniscus. **(b)** a much larger area of mineralization is apparent in this cat's stifle consistent with mineralization of the fat pad.

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cranial cruciate ligament rupture. They are typically located in the fat pad. Smaller mineralizations in the cranial aspect of the feline stifle may be within the cranial horn of the medial meniscus and may not necessarily be associated with cruciate ligament rupture (Figure 11.10). The incidence of meniscal injuries in 98 feline stifles with cranial cruciate ligament rupture was 67%. As in dogs, the majority of meniscal injuries affect the medial meniscus.

Treatment and Prognosis

Treatment of feline CCL rupture may be conservative or surgical.

Conservative Therapy of the Cranial Cruciate Ligament Deficient Stifle in Cats

Conservative therapy should be attempted for approximately 6 weeks. The cat is confined to a crate or small single room relatively strictly for the first 2–3 weeks, to limit activity. Where bodyweight is not optimal, a plan for weight loss is instituted. Normal limb function and pain-free range of motion were reported in one study to return within 4–5 weeks. However, many cats treated conservatively will have persistent joint laxity, mild muscle atrophy, thickening of the joint capsule and mild osteoarthritis.

In a study comparing cats following conservative management of cranial cruciate ligament rupture with cats with no stifle disease, assessed by pressure mat and an owner-completed questionnaire, the cats in the former group demonstrated a different gait pattern and owner-recognized limitations to activity. Whilst this does not necessarily constitute a significant welfare concern, if a superior surgical option is identified, it should be considered. However, only one study has compared outcomes for surgically versus conservatively managed cats with cruciate disease.

Surgical Stabilization of the Cranial Cruciate Ligament Deficient Stifle in Cats

Stabilization of the stifle after CCL rupture is recommended only in cats with persistent lameness unresponsive to conservative therapy. Obese cats may be reluctant to ambulate during conservative therapy and may benefit from early surgery.

As for the dog, intracapsular, extracapsular and tibial osteotomy techniques have been described for the cat with CCL deficient stifles.

Extracapsular stabilization (lateral stabilizing suture, fabello-tibial suture among other names) involves placing a suture, to tether the femur and tibia, at a caudo-proximal to craniodistal angle similar to the native CCL, to prevent cranial drawer motion and minimize internal rotation of the tibia. (Box 11.1)

1. A lateral mini or parapatellar arthrotomy is recommended to permit joint exploration. Torn remnants of the CCL and damaged portions of the menisci are excised.
2. Proximal attachment of the suture: the fascia of the biceps femoris muscle is elevated from the joint capsule and retracted laterally and caudally. The fabella is palpable just proximal to the femoral condyle. The suture (3–4 M non-absorbable material) is placed around the fabella through the broad and rather ill-defined femorofabellar ligament using a curved needle. Entrapment of the common fibular nerve in the suture should be avoided, by good visualization of the tissues being purchased. Once the suture is positioned around the fabella, proper suture placement is confirmed by placing tension on the suture and ensuring stability. Alternatively, the suture can be anchored to the lateral femoral condyle caudally, adjacent to the distant aspect of the lateral fabella, using a suture anchor. This method requires less exposure, reduces the risk of nerve entrapment and may permit more isometric suture positioning.
3. Distal attachment of the suture: cranial tibial muscle is elevated from the medial aspect of the tibial tuberosity. A bone tunnel is drilled from lateral to medial through the tibial tuberosity. One end of the suture is then passed from lateral to medial, caudal to the patellar tendon, and back through the bone tunnel from medial to the lateral side. The suture is tightened until no drawer motion can be detected and secured by tying or crimping. The knot or crimp is buried beneath the biceps fascia during closure.

BOX 11.1 ISOMETRIC/QUASI-ISOMETRIC EXTRA-CAPSULAR SUTURE PLACEMENT

Two studies of lateral fabello-tibial suture placement in cats involved the determination of quasi-isometric attachment sites on the femur and tibia. Because the stifle is not a true hinge joint it is recognized that the distance between two attachment sites (one on the femur/fabella) and one on the tibia will change through the joint range of flexion and extension. Therefore a suture placed to bridge the femoro-tibial joint will inevitably stretch or snap, or impede the range of motion. Isometry refers to attachment sites which maintain a constant separation through the joint's range of motion. True isometry does not exist in the feline stifle. Therefore, quasi-isometric attachment sites were determined. When compared in a biomechanical study, the lateral fabello-tibial suture and suture placed with quasi-isometric attachment sites performed similarly to restore joint function. Nonetheless, these attachment sites may be preferable for the longevity of the lateral extra-capsular suture, but no clinical studies of their use have been reported.

Intra-articular stabilization involves placement of a fascia lata graft which is passed through the stifle joint to tether the femur and tibia, in an approximate replacement of native CCL tissue. This technique was widely reported in dogs in the past and has increasingly fallen out of favour in this species. It is possible to perform this procedure in cats; however, reports are limited.

The stability of feline cadaveric stifles was evaluated following a fascia lata over-the-top or under-and-over technique (fascia lata graft passed through the joint to mimic the location of the native CrCL) or following a lateral fabello-tibial suture (40 lb nylon leader line) and comparisons made. The lateral fabello-tibial suture was found to be superior at resisting cranial tibial subluxation under loading. Therefore, intra-articular stabilization techniques at this stage appear to be inferior.

Tibial osteotomies include tibial plateau leveling osteotomy (TPLO, Figure 11.11), cranial closing wedge

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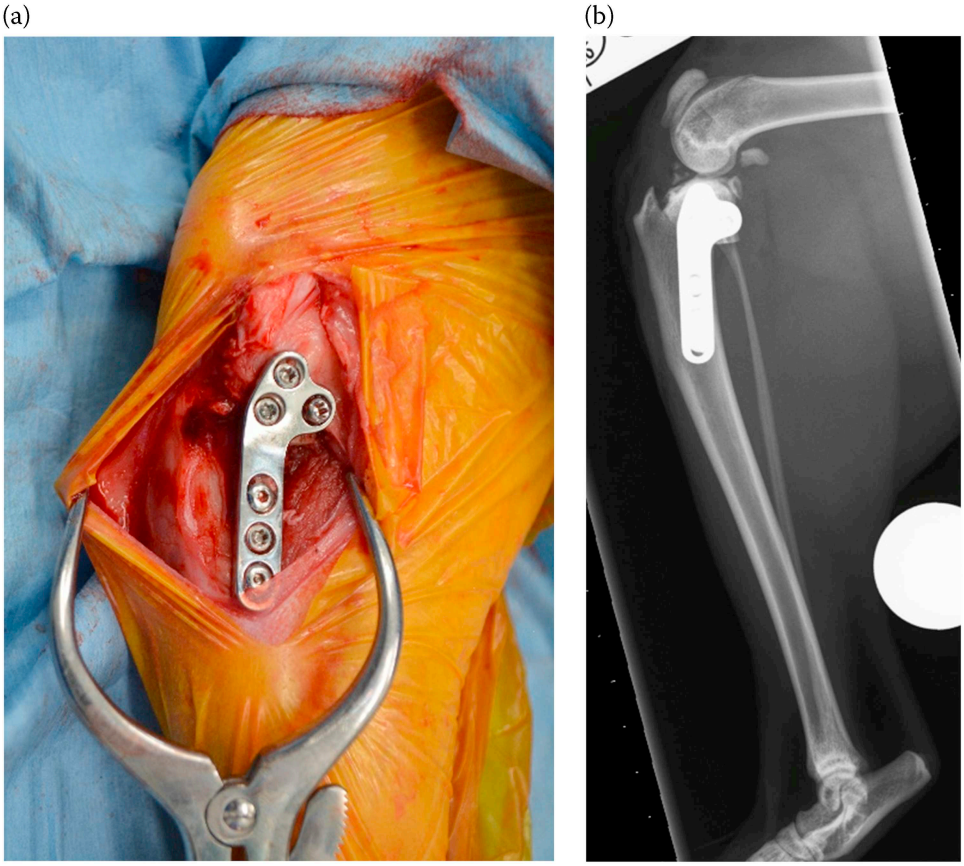


Figure 11.11 Tibial plateau levelling osteotomy has been reported in cats with successful outcomes. Nonetheless, the biomechanical benefits of this technique in this species are yet to be confirmed. **(a)** intra-operative photograph prior to closure following tibial plateau levelling osteotomy (TPLO) in a cat. **(b)** post-operative mediolateral radiograph of the same tibia following TPLO.

osteotomy (CCWO) and tibial tuberosity advancement (TTA). These procedures are well described and defined for dogs. They aim to stabilize the cranial cruciate ligament deficient stifle by abolishing cranial tibial subluxation during joint loading. TPLO and CCWO rotate the tibial condyles until the tibial joint surface (tibial plateau) is near perpendicular to the tibial long axis, such that load applied parallel to the tibial long axis does not induce a cranially directed vector force on the tibia. TTA advances the insertion of the patellar ligament until the ligament is orientated perpendicular to the tibial joint surface, such that tension in the patellar ligament, resulting from quadriceps contraction during loading, does not induce a cranially directed vector force on the tibia.

Cadaveric studies similar to those performed to evaluate the tibial osteotomies for dogs have been performed for cat stifles. Limb press models involve recording cranial tibial subluxation in stifles under load with the cranial cruciate ligament intact and then transected, followed by repeat measurements following a modification (such as TPLO). Cranial tibial subluxation inevitably increases dramatically following cranial cruciate ligament transection (often by around 9 mm). Both TPLO and TTA have been evaluated in this way, revealing that cranial tibial subluxation was unresolved following either of these procedures. Nonetheless, studies evaluating outcomes following these surgical interventions in cats with naturally occurring cranial cruciate ligament rupture have been published, with satisfactory outcomes, albeit with subjective measures of outcome.

Reported Outcomes for Cats with CCL Rupture

Cats that underwent conservative management were compared with cats that had lateral fabello-tibial suture stabilization in one study. Owners completed a validated questionnaire (feline musculo-skeletal pain index, FMPI) regarding their cats' behaviour. Cats that had conservative management of CCL rupture had lower FMPI scores (indicating less chronic pain) than cats with lateral fabello-tibial suture.

Clinical outcomes for 12 cats with tibial plateau levelling osteotomy have been reported in two studies (in one case cranial closing wedge ostectomy was also performed to lower an excessive tibial plateau angle) and outcomes for three cats with tibial tuberosity transposition have been reported in two studies. In these clinical reports, subjective outcome assessments have delivered good to excellent results. How good outcomes are achieved despite an apparent failure to resolve cranial tibial subluxation is unclear at this stage.

CAUDAL CRUCIATE LIGAMENT INJURY:

Isolated caudal cruciate ligament injuries are very rare in cats. They may cause lameness and stifle joint pain. The examination finding of caudal tibial subluxation

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differentiates this condition from cranial cruciate ligament rupture. Conservative management will usually result in return of normal function.

COLLATERAL LIGAMENT INJURY:

Injuries to the collateral ligaments of the stifle joint result from trauma. Isolated collateral ligament ruptures are extremely rare in cats. Even isolated collateral ligament ruptures require surgical attention. However, these injuries are typically associated with cranial + - caudal cruciate ligament ruptures and/or meniscal injuries as part of traumatic luxation of the stifle (see stifle luxation).

BOX 11.2 LIGAMENT INJURY

Proper treatment of ligament injuries depends on the severity of the ligament damage.

- **First-degree ligament sprain**. Haemorrhage and oedema are present within the parenchyma of the ligament and a few collagen fibres may be ruptured, but the ligament is essentially intact. The function of the ligament is preserved and the joint is stable. Spontaneous healing may be expected provided strain in the healing ligament is minimized (e.g. by restriction of activity).
- **Second-degree ligament sprain**. The ligament is grossly intact but its function is disrupted with some collagen fibre disruption and loss of cross-linking. Joint instability is present and surgical stabilization is indicated to restore joint function and allow healing.
- **Third-degree ligament sprain**. The parenchyma of the ligament is totally disrupted or it is avulsed from its point of origin or insertion on the bone. The joint is unstable and surgical stabilization is indicated.

STIFLE LUXATION:

(Addison & Conte 2019, Aron 1988, Bruce 1999, Connery & Rackard 2000, Elliott 2019, Hulse & Shires 1986, Jaeger et al. 2005, Keeley et al. 2007, Swanwick 1987, Welches & Scavelli 1990).

Although it is observed more frequently than in dogs, stifle luxation (stifle derangement, stifle dislocation, global stifle disruption) remains an infrequent injury in cats (Figure 11.12). It involves disruption of more than one of the four primary stabilizing ligaments (cranial and caudal cruciates, medial and lateral collaterals). In addition there is often trauma to the joint capsule, the medial and/or lateral meniscus and muscles/tendons that cross the joint. Bilateral luxation has been reported. In two published series of this injury the combination of rupture of the cranial cruciate, caudal cruciate and medial collateral ligaments was present in all cats (six cats in total). Serious vascular and neurologic damage to the limb concurrent to this injury is rare, and the injury is rarely open.

Diagnosis

Cats present with non-weight-bearing lameness and a history of trauma. Palpation alone is often enough to give a suspicion of stifle luxation. Obvious stifle instability is present, but there is often severe resentment to manipulation of the luxated stifle.

Manipulation under sedation or general anaesthesia gives some information regarding which structures are traumatized. To assess collateral ligament integrity, varus and valgus stress is applied to the stifle with the joint in flexion and extension. Normally, the medial collateral ligament is taut in stifle flexion and extension. The lateral collateral ligament is taut in stifle extension but slightly lax in stifle flexion. Loss of medial collateral ligament integrity results in valgus instability of the stifle and excessive internal rotation of the tibia when the joint is flexed. Loss of lateral collateral ligament integrity results in varus instability. Distinguishing between rupture of the cranial cruciate ligament, caudal cruciate ligament or both is difficult in very unstable joints.

Radiography and/or computed tomography may be required to exclude fractures, since these may have an influence on the likely outcome, particularly where they affect the articular surface. Small fragments of bone may be associated with avulsion injuries to the collateral ligaments. Stressed views may be useful in determining which of the ligaments are ruptured, although in cases of multi-ligamentous injuries, the tibia and femur move in unpredictable ways relative to one another, making interpretation of such images difficult. Surgical exploration may be required to confirm the extent of ligamentous trauma and damage to the menisci (Figure 11.13).

Treatment and Prognosis

Immobilization with external coaptation may reduce swelling and improve comfort while the patient is stabilized for 24–48 hours. The first stage of surgery is exploratory, to confirm which structures are traumatized.

1. Approach: A lateral or medial parapatellar approach is made to explore the joint.
2. Evaluation: Each of the following structures should be assessed individually (visually and by probing) for their integrity: cranial and caudal cruciate ligaments, medial and lateral collateral ligaments, long digital extensor tendon of origin, medial and lateral meniscus, patellar ligament, retropatellar articular surface, trochlear groove.
3. Primary treatment: Ruptured cruciate ligaments and damaged portions of the menisci are excised. Fully avulsed menisci (which often remain attached at their meniscotibial ligaments) may be sutured abaxially to the joint capsule. Ruptured or avulsed collateral ligaments should be sutured or reattached where possible. Nonabsorbable suture material is placed to approximate the traumatized ends of the ligament using a locking-loop, Bunnel–Meyer, or continuous cruciate suture pattern. Large avulsion fragments should be anchored to their original site with a lag screw or two divergent Kirschner wires. Most avulsion fragments are too small so the ligament is reattached to the bone using suture material anchored through the ligament and a bone tunnel drilled at the ligament's normal site of attachment. Luxated or traumatized long digital extensor tendons or patellar ligaments should be addressed appropriately (see below), though restoration of a normal relationship between the femur and tibia may normalize the position of these structures.
4. Definitive treatment: Once the extent of trauma is clarified a definitive treatment is established. Definitive treatment follows two algorithms:

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Figure 11.12 Severe cranial displacement of the tibia with respect to the femur is apparent in the mediolateral **(a)** and caudocranial **(b)** images of the tibia and stifle joint of a cat with stifle luxation.

1. Immobilization of the joint for 4–6 weeks.
2. Placement of extra-articular sutures to replace the function of traumatized ligaments.

A third approach exists (salvage by arthrodesis, or limb amputation), and should be offered if damage to the articular cartilage and joint support structures is severe and the prognosis for a return to adequate comfort considered unlikely.

IMMOBILIZATION OF THE STIFLE JOINT:

Immobilization of the traumatized stifle with the femur and tibia in appropriate orientation to one another permits periarticular fibrosis and healing of types 1 and 2 sprains of collateral ligaments, which may result in relatively good reestablishment of a stable femoro-tibial joint. The feline stifle joint should be immobilized at an angle of approximately 120°, although an assessment of the contralateral hindlimb femoro-tibial standing angle should be made and this angle may be matched during immobilization.

A period of immobilization is expected to have deleterious effects on the soft tissues of the joint, with atrophy of the articular cartilage and associated muscles, potentially worsening the long-term prognosis for joint health. Conversely, it has been reported that stifle immobilization following transection of the cranial cruciate ligament prevents the onset of radiographic evidence of osteoarthritis in the short term, so a period of immobilization following stifle

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Figure 11.13 Intra-articular examination of this luxated feline stifle demonstrates avulsion of both medial and lateral menisci. With little hope of preserving their function these menisci were excised. If the menisco-tibial ligaments remain intact suturing the menisci to the peripheral joint capsule may preserve them, however, this is unreported in the cat.

luxation may also theoretically be protective from osteoarthritis.

Immobilization may be performed using a transarticular pin or a transarticular external skeletal fixator (Figure 11.14). The use of a hinged transarticular external skeletal fixator has been documented in a single case with a good outcome. The benefit of the hinge was suggested to be the potential to gradually reintroduce movement, such that healing tissues may remodel according to the periodically increasing strain applied to them.

Immobilization of the Stifle by Transarticular Pinning

Temporary stifle immobilization with a transarticular pin has been described as a stand-alone treatment option (with the pin remaining in place for 4–6 weeks), or as an intra-operative technique to maintain femoro-tibial alignment while a transarticular external skeletal fixator is applied or while combinations of transarticular sutures are placed and tightened (see below). A 3.0 mm or 3.5 mm Steinmann pin is placed 'retrograde', passing initially into the intercondylar fossa of the femur to exit at the proximal extent of the trochlear articular cartilage with the stifle flexed, before the stifle is positioned appropriately and the pin driven back across the joint into the tibia, where it penetrates the non-articular intercondylar area cranial to the intercondylar

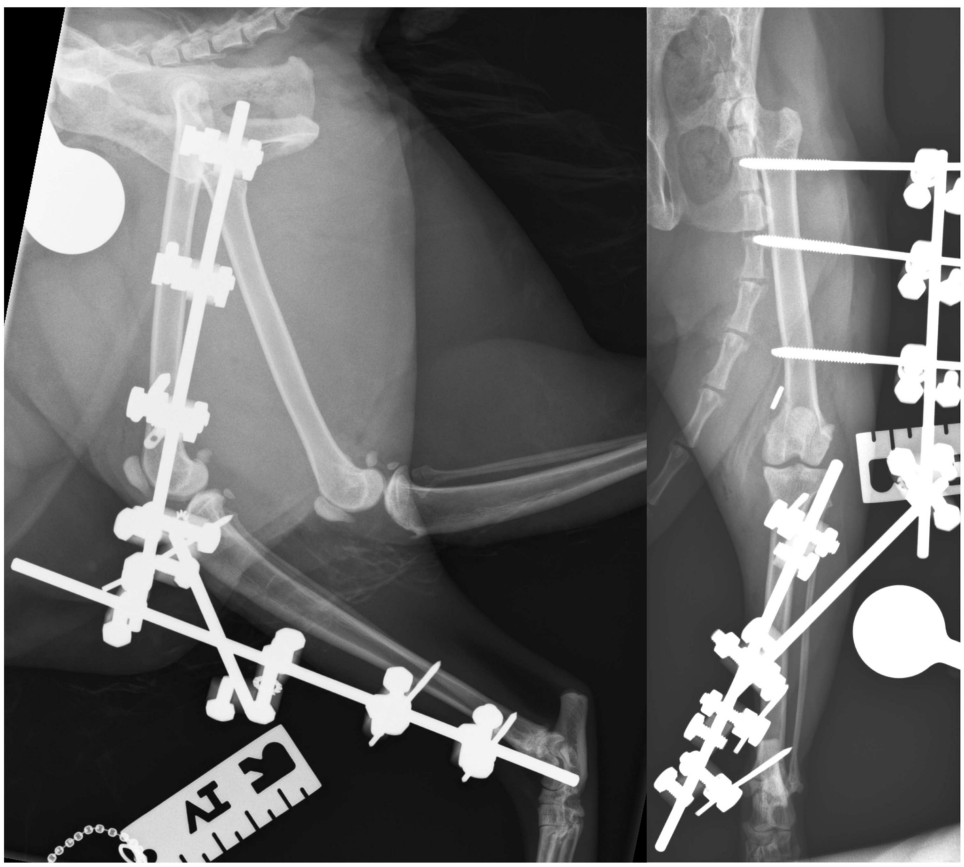


Figure 11.14 Mediolateral and craniocaudal radiographic projections of a feline right hindlimb following internal fixation of a luxated stifle (note a metallic toggle medially and a femoral bone tunnel through which suture passes as a lateral extracapsular suture). Fixation pins are placed to avoid hazardous corridors where possible, i.e. the lateral aspect of the femur and the craniomedial aspect of the tibia.

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eminence, to exit the cranial tibial cortex. Normograde placement of the pin from distal to proximal has been reported but is considered likely to increase the risk of traumatizing the femoral trochlear articular surface and/or patella so is not encouraged.

A two-pin technique has also been described, in which a second pin is placed normograde from the medial aspect of the tibial tuberosity into the lateral femoral condyle. Whilst it is assumed the additional pin may increase construct stiffness, there is no evidence it is appropriate, and placement of multiple intra-articular implants is to be avoided.

If the pin is to remain in position for 4–6 weeks then it is cut short (but not so short that later retrieval is not possible) at the points of exit from the tibia and femur, the joint capsule is imbricated with absorbable suture and the subcutaneous tissues and skin are closed routinely. Strict confinement is enforced until the transarticular pin is removed. Migration, bending and loosening of the pin have been reported. An end-threaded negative profile pin may reduce the likelihood of migration.

Immobilization of the Stifle by Transarticular External Skeletal Fixation

The authors prefer to use external skeletal fixation to avoid the risk of further articular trauma and implant-related complications of transarticular pin placement. Typically frames will consist of type Ia frames applied to the femur and tibia. The frame is applied laterally to the femur (three positive profile fixation pins placed in the greater trochanter, proximal femoral diaphysis and in the femoral condyles) and cranially or medially to the tibia (three positive profile fixation pins placed in the tibial condyles, distal diaphysis and distal metaphysis). It is simpler for overall frame construction to place the tibial part of the frame laterally, as was reported in two studies; however, the safer corridors for implant placement on the tibia are medial. Placing the femoral part of the frame laterally and the tibial part of the frame medially necessitates a more inventive connection between the two connecting bars. The two frames are connected using double clamps and one or two additional connecting bars extending between the femoral and tibial connecting bars. Use of a hinge at the level of the stifle has been reported, such that some controlled movement in a single plane may be permitted, potentially mitigating the concerns regarding joint health associated with chronic immobilization. Such a hinge must be placed at the correct level and plane of rotation of the joint such that any permitted flexion and extension does not result in opening or closing of the joint space, which will theoretically have deleterious effects on articular cartilage and healing ligaments.

EXTRAARTICULAR SUTURE STABILIZATION (FIGURE 11.15):

Large diameter slowly absorbing or non-absorbable sutures are placed outside of the joint capsule to replace the function of the various ruptured or sprained ligaments. Sutures may be placed to replicate the function of the specific traumatized ligaments or, alternatively, a suture prosthesis system is described which aims to stabilize the stifle in all directions, regardless of the combination of injuries.

1. Replicating the function of individual traumatized ligaments:
    - Cranial cruciate ligament – a suture is placed between the lateral fabella and a hole drilled in the

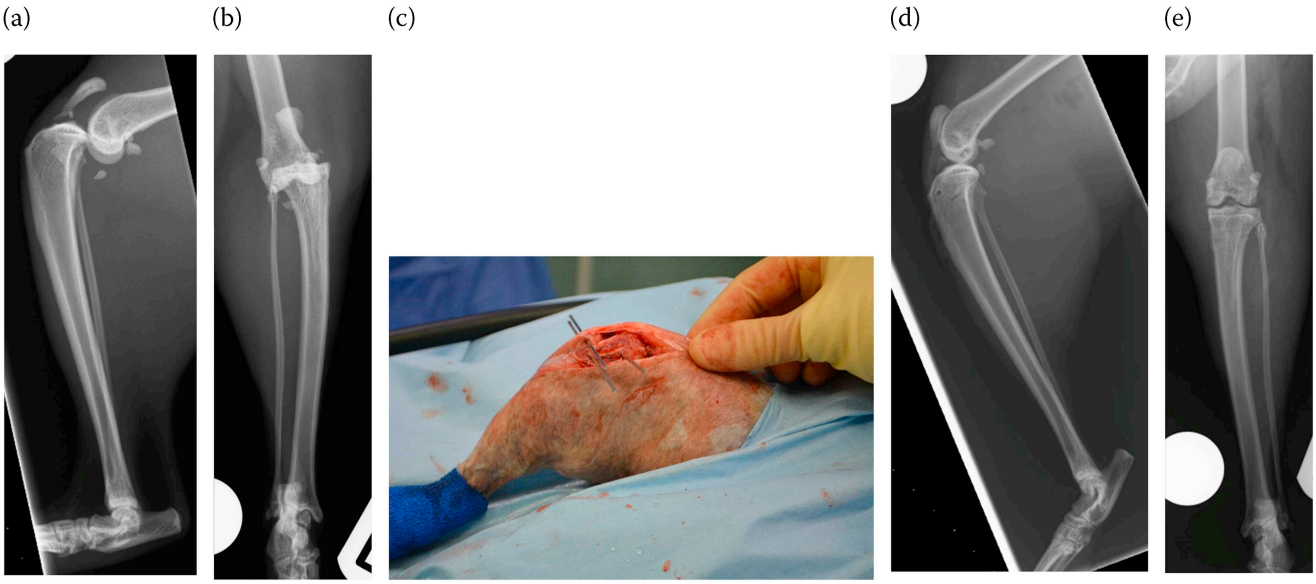


Figure 11.15 Stifle luxation fixation using extra-capsular suture technique. **(a** and **b)** mediolateral and caudocranial radiographic projections indicate severe cranial tibial luxation with a mineralization potentially consistent with avulsion of the tibial insertion of the cranial cruciate ligament (or other intra-articular or juxta-articular structure). **(c)** three bone tunnels are created: one in the femur connecting the origins of the medial and lateral collateral ligaments, one in the proximal tibia from the insertion of the medial collateral ligament to just cranial to the fibular head (or through the fibular head) and one in the tibial tuberosity. Two loops of suture are made: both pass through the femoral bone tunnel but one passes through the cranial tibial tunnel and one through the caudal tibial tunnel. Sutures are tightened (care with overtightening particularly if the caudal cruciate ligament is not intact, since iatrogenic caudal tibial subluxation is a possibility). **(d** and **e)** mediolateral and caudocranial post-operative radiographic projections demonstrating bone tunnel locations and normalized femoro-tibial alignment.

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tibial tuberosity. A bone anchor technique may also be considered (see Cranial Cruciate Ligament Rupture).

- Caudal cruciate ligament – one suture is placed between a tunnel drilled in the fibular head (or looped around the tibio-fibular ligament, taking care of the common fibular nerve) and the patellar ligament just distal to the patella.
- Collateral ligaments – suture material is placed between the distal femur and the tibial plateau (medially) or fibular head/tibio-fibular ligament (laterally), using the normal origin and insertion points of the relevant ligament as landmarks. The suture is anchored to the bone using bone tunnels, screws and washers, or bone anchors.

2. Ligament prosthesis through bone tunnels:

Bone tunnels are drilled medial to lateral or lateral to medial using a 1.5 mm or 2.0 mm drill bit as follows: 1. across the femur at the origins of the collateral ligaments, 2. across the tibia from the fibular head into the tibia to emerge in the insertion of the medial collateral, 3. across the tibial tuberosity just caudal to the cranial cortex. Two loops of suture are passed, one through tunnels 1 and 2 (replacing the collateral ligaments) and one through tunnels 1 and tunnel 3 (replacing the cranial cruciate ligament). Replacement of the caudal cruciate ligament is unnecessary.

Good clinical function is reported after use of extraarticular suture stabilization for treatment of stifle luxation in cats.

Prognosis and Outcome

The outcomes for cats with stifle luxation are not well reported. No comparison of outcomes, short-term or long-term, exists for stifle immobilization versus extra-articular suture stabilization in dogs or cats.

INTRA-ARTICULAR STIFLE MINERALIZATION:

(Freire et al. 2010, Leijon et al. 2017, Tas et al. 1994, Voss et al. 2017, Whiting & Pool 1985).

Intrameniscal mineralizations are common in cats and have been reported in the literature since 1985 (Figure 11.10). Their significance is controversial. The cranial horn of the medial meniscus is a common site for mineralization within the cat's stifle. At this site, small radiographic mineralizations may be considered incidental findings, since they may be unassociated with osteoarthritis. Mild osteoarthritis is frequently apparent in clinically normal cats (as detected by histopathology), while articular mineralizations may be present in about half of cats. It appears that a distinction may be made between small mineralizations of the cranial horn of the medial meniscus, and large mineralizations, which are found in the peri-articular tissues, in particular the joint capsule and fat pad. Large mineralizations are frequently associated with cranial cruciate ligament rupture and/or osteoarthritis, particularly medial compartment disease. Whether meniscal mineralization may be considered primary or secondary to osteoarthritis remains uncertain.

STIFLE SYNOVIAL OSTEOCHONDROMATOSIS:

(Allen 2000, Hubler et al. 1986, Tas et al. 2013).

Synovial osteochondromatosis involves the development of ostechondral nodules within joints. These nodules may detach from the synovium and become free-floating bodies within the joint. They can be present in large numbers and may be considered primary (where no other pathology affects the joint) or secondary (in the presence of additional pathology). Often synovial osteochondromatosis may be present in multiple joints, and it may (but may not) be clinically relevant. This condition should be differentiated from intra-articular mineralization and from the more severe forms of osteochondromatosis, in which FeLV is often implicated in the aetiology.

PATELLAR LIGAMENT RUPTURE:

(Das et al. 2015, Manca & Witte 2019, Witte 2014).

The patellar ligament is the portion of the quadriceps mechanism between the patella and the tibial tuberosity. It is rarely traumatized in cats. In fact, only two reports of the condition in cats exist, describing eight patellar ligament ruptures in seven cats. Diagnosis is typically by radiography (Figure 11.16).

Management options have been explored more thoroughly in dogs, and techniques for management of this condition in cats appear to be mostly extrapolated from dogs. Surgical management of patellar ligament rupture may involve a combination of three steps: primary ligament repair, circumpatellar augmentation and temporary stifle joint immobilization.

1. Primary ligament repair should be performed in all cases and might involve 3 or 3.5 M slowly absorbable or non-absorbable materials in locking loop, Mayer-Bunnell or modified mattress suture patterns. One case of feline

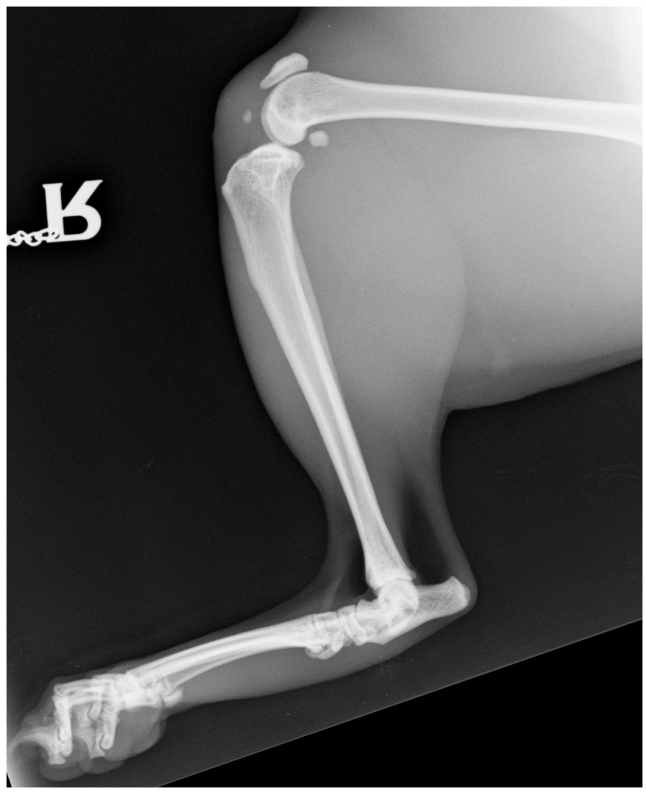


Figure 11.16 Mediolateral radiographic projection of right hindlimb of a cat. Soft tissue opacity obscures the articular structure of the stifle joint, a mineral opacity fragment is present in the centre of the joint and there is patella alta despite positioning in moderate flexion of the stifle. These signs are indicative of patellar ligament rupture.

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patellar ligament rupture has been reported in which no further augmentation was performed over and above the primary ligament repair, with no reported complication.

2. Circumpatellar augmentation is relatively simple to perform, and should reduce the strain applied to the primary ligament repair during quadriceps contraction. This may involve a circumpatellar loop of suture material passed through a tibial tuberosity bone tunnel, or a strip of fascia lata. A suture passed through the patella is not recommended, since drilling through the narrow patella risks transverse patellar fracture. 3.5 or 4 M slowly absorbable suture is used for circumpatellar suturing. The fascia lata graft technique has been described involving maintenance of a distal base, passage of the graft from lateral to medial proximal to the patella (through the quadriceps muscular insertions), and suturing of the graft to the medial and lateral aspects of the parapatellar tissues, and to the fascia overlying the cranial tibial muscle. It has been suggested that a fascia lata graft should be recommended in cases of more chronic rupture to address concerns with the viability of the patellar ligament in the region of the rupture, although the precise stage at which a patellar ligament rupture ceases being acute and therefore requires grafting is not clear. If the rupture has been present more than a week or the ligament ends cannot be reapposed intra-operatively then fascia lata graft should be considered. It is used routinely by one author for all cases of patellar ligament rupture.
3. Immobilization of the stifle aims to minimize tension within the healing patellar ligament. Transarticular external skeletal fixation was performed in three of eight cases reported in the literature (in addition to other techniques) and, although no direct complications were associated with use of this method, the remaining five cases appear to have progressed uneventfully; therefore, the requirement for stifle immobilization in cats is called into question.

Luxation and Rupture of the Long Digital Extensor Tendon of Origin

(De Rooster et al. 2004, Haaland & Sjostrom 2007, Hasiuk et al. 2017, Kalff et al. 2014).

Traumatic injuries of the long digital extensor are an infrequent cause of stifle effusion and hindlimb lameness in the dog. It should be remembered that cruciate disease is not the only cause of stifle effusion. As yet, luxation of the long digital extensor tendon of origin (typically a lateral luxation from the extensor fossa) and rupture of the ligament (typically secondary to lateral patellar luxation) have not been reported in cats.

Quadriceps Contracture

(Fries et al. 1988, Liptak & Simpson 2000, Tisdall & Rogowski 2017).

Quadriceps contracture relates to severe quadriceps muscle atrophy and adhesion to or incorporation into callus associated with a femoral fracture (alternative aetiologies include immune-mediated myositis, neuromuscular disease, neoplasia, ischaemia and protozoal infection. Stifle hyperextension and severe atrophy of the quadriceps muscles is commonly present. The process eventually results in fibrous metaplasia of the muscle tissue. It is most common in skeletally immature animals.

Treatment of quadriceps contracture should involve passive and active physiotherapy (see Chapter 17). More severe cases may involve removal of metallic implants and mobilization of adhesions between the femur and quadriceps muscles by blunt and/or sharp transection. A dynamic flexion apparatus has been described involving elastic bands connecting metallic implants in the ischium (three threaded external skeletal fixation pins) and the distal tibia (a circular external skeletal fixation ring block secured to the bone with two crossed wires). The apparatus was placed following surgical release of adhesions, such that a normal range of motion was present at the time of its application. Nonetheless, following removal of the device after 2–3 weeks, contracture returned consistently. Medication with prednisolone at 1.85 mg/kg twice daily for 3 days followed by gradual tapering over a period of 8 weeks prevented the worsening or return of contracture.

STIFLE ARTHRODESIS (FIGURE 11.17):

(Belch et al. 2012, Cofone et al. 1992, McLaughlin 1993).

Arthrodesis is the only salvage procedure readily available for the feline stifle. It is rarely performed but serves as an alternative to amputation. Indications include irreparable joint luxations/fracture, severe osteoarthritis and debilitating quadriceps contracture. Stifle arthrodesis does not restore normal limb function, but acceptable, pain-free function may be achieved. Results are best if the ipsilateral hip is normal and the stifle is fused at an angle (110°–125° for most cats) that allows the foot to contact the ground without causing excessive flexion or extension of the opposite limb. The ideal fusion angle is determined preoperatively by measuring the contralateral stifle joint in a normal standing position. To compensate for bone shortening caused by ostectomy of the femur and tibia, 5° is added to the measurement obtained.

1. Autogenous cancellous bone grafting may not be essential because of the large contact area created between the femur and tibia during arthrodesis.
2. A medial or lateral parapatellar approach is made. Exposure may be improved by ostectomy of the tibial tuberosity to allow elevation of the patellar ligament proximally.
3. The fat pad, cranial and caudal cruciate ligaments, medial and lateral menisci, and intermeniscal ligaments are excised. The collateral ligaments are preserved, if possible, to facilitate manipulation of the limb.
4. Using an oscillating bone saw, ostectomies of the distal femur and proximal tibia are performed to provide good contact between the two bones. The technique description usually involves placing Kirschner wires to ensure appropriate orientation of the ostectomies (such that the desirable fusion angle is achieved) and to avoid introducing rotation (Figure 11.17). The popliteal vessels and common fibular nerve must be avoided. An alternative to ostectomy is to burr the articular surfaces of the femur and tibia (and femoro-patellar joint) using a high-speed burr.
5. Kirschner wires are inserted in a cross-pin configuration to maintain reduction temporarily.
6. If it is to be added, autogenous cancellous bone graft may be harvested and placed at this stage.
7. Definitive fixation has been reported with a medially applied or cranially applied 2.7-mm dynamic compression plate and screws. Whilst cranial plating has been reported in dogs and one cat, it was theorized that medial plating may be preferable since the feline femur is wider in the dorsal than the sagittal plane so screw purchase may be superior, and; in addition, the patella

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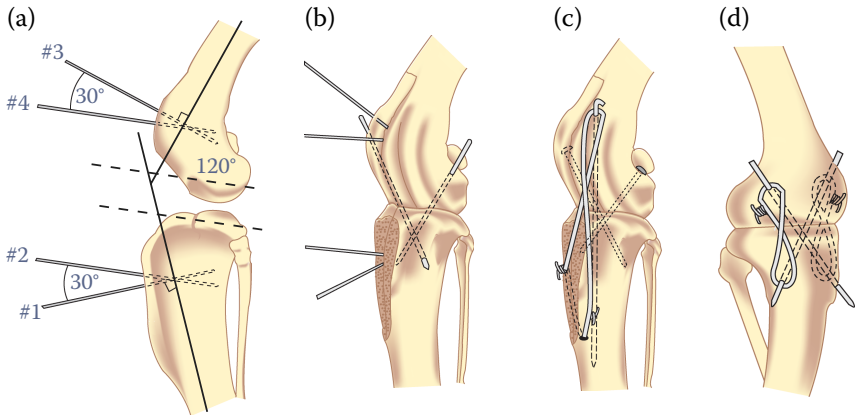


Figure 11.17 Stifle arthrodesis. **(a)** Kirschner wire #1 is inserted in the sagittal mid-line plane of the tibia perpendicular to the long axis of the bone. Wire #2 is inserted in the sagittal plane 30° from the first. Wire #3 is inserted in the sagittal plane of the femur perpendicular to the long axis. Wire #4 is inserted 30° from the third wire. Ostectomy of the proximal tibia is performed parallel to wire #2; ostectomy of the distal femur is performed parallel to wire #4 (ensuring a fusion angle of 120°). **(b)** The femur and tibia are apposed using the Kirschner wire markers to maintain rotational alignment. Temporary reduction is maintained with crossed Kirschner wires. Cancellous graft is placed. **(c)** Two screws are placed in lag fashion across the joint (one from the lateral femoral condyle to the cranial–medial aspect of the tibia, the other from the medial femoral condyle to the cranial–lateral aspect of the tibia). A small pin is placed from the proximal aspect of the trochlear sulcus into the proximal tibia. A transverse hole is drilled through the proximal tibial crest and a tension band wire is placed. **(d)** Alternatively, Kirschner wires may be inserted in cross-pin fashion across the joint. Cerclage wire is placed in a figureof- eight fashion around the ends of the Kirschner wires medially and laterally. Augmentation with a plate (medial or cranial) and screws is advised.

may be immobilized within the trochlear groove (which is not possible where the plate is placed cranially) which was theorized to preserve flexion of the hip joint.

Medium- to long-term outcomes following stifle arthrodesis have been reported for two cats. In both cases limb use was subjectively good, although circumduction was apparent in both cases.

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Part 3 Tibia, Fibula, Hock

The tarsus consists of numerous joints and their supporting ligaments. From proximal to distal, the joints of the pes include:

- Tarsocrural (hock) joint: articulation between the tibia and fibula and the talus and calcaneus.
- Talocrural joint – between the talus and tibia.
- Talocalcaneal joint – between the talus and the calcaneus.
- Intertarsal joints- articulations between the tarsal bones (including sagittal intertarsal joints which are rigid).
- Talocalcaneocentral joint – between central tarsal bone and the base of the talus and calcaneus.
- Calcaneoquartal joint – between the calcaneus and the fourth tarsal bone. The combined calcaneoquartal and talocalcaneocentral joints are often referred to as the proximal intertarsal joint.
- Centrodistal joint – between the central tarsal bone and tarsal bones I, II, and III. This is often referred to as the distal intertarsal joint.
- Tarsometatarsal joints – between the distal tarsal bones and metatarsal bones.
- Metatarsophalangeal joints – between the metatarsal bones and first phalanges.
- Interphalangeal joints – between phalanges.

TALOCRURAL LUXATION:

(Aron 1987, Gorse et al. 1990, Gorse et al. 1991, Kulendra & Arthurs 2014, Kulendra et al. 2011, Yardimci et al. 2016).

Traumatic luxation of the tarsocrural joint is associated with disruption of the medial and/or lateral collateral ligaments. This may result from malleolar fracture (see Chapter 10) or, less commonly, luxation occurs with rupture or avulsion of the collateral ligaments. In cats, the medial and lateral collateral ligaments are each comprised of two short ligaments; there is no long component as seen in dogs.

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Examination Findings

Clinical signs of tarsocrural luxation include hindlimb lameness (often non-weight bearing), swelling in the region of the hock, which may be severe, and palpable joint luxation and/or instability.

Investigation

Radiographs are obtained to evaluate the malleoli for fractures. Varus or valgus stress can be applied to the tarsocrural joint during radiography to demonstrate laxity. If concurrent fractures of the tibia, fibula, tarsus or metatarsus are suspected computed tomography is recommended to facilitate determination of a prognosis.

Treatment

Two algorithms exist for treatment of hock instability in the cat: reconstruction of collateral support versus temporary transarticular external skeletal fixation. Theoretically, reconstruction of the collateral support by (a) malleolar fracture repair, (b) collateral ligament repair and (c) collateral ligament augmentation should restore joint function and permit early loading of the joint and use of the limb. However, it involves open approaches to both sides of the joint and attempts to reconstruct fine structures. The potential influence of complications may be of greater significance than the potential benefits of restoration of joint function. Temporary immobilization allows healing by peri-articular fibrosis, which may restore a functional joint without any attempt to reconstruct malleolar fractures or sprained/ruptured collateral ligaments. No comparison has been made between these two approaches.

COLLATERAL LIGAMENT RECONSTRUCTION:

The joint is explored to evaluate the cartilage and remove haematoma and debris. Any articular fractures are stabilized with small Kirschner wires countersunk below the articular surface (this is rarely possible with cats and small fragments are better discarded). The joint is then reduced and stabilized using one of several methods.

Malleolar Fracture Repair

In many cases, collateral ligament function is restored by repairing the fractured malleolus using a pin with or without a tension band technique. Pins should be directed sufficiently proximally to avoid penetration of the talocrural joint and tension bands are typically applied through bone tunnels in the medial or lateral aspect of the tibia (Figure 11.18).

Collateral Ligament Repair

Primary tenorrhaphy of ruptured collateral ligaments is rarely an option due to their small size but should be attempted if appropriate. If the collateral ligament is avulsed from the malleolus, its function may be restored by reattaching the ligament to its origin. Unfortunately, in the cat, this is difficult to achieve using rigid fixation due to the small size. Therefore, suture material (fine gauge monofilament, non-absorbable) is used to place a locking-loop or Bunnel–Meyer suture in the ligament adjacent to the avulsion fragment and the suture is then passed through a hole drilled in the relevant malleolus.

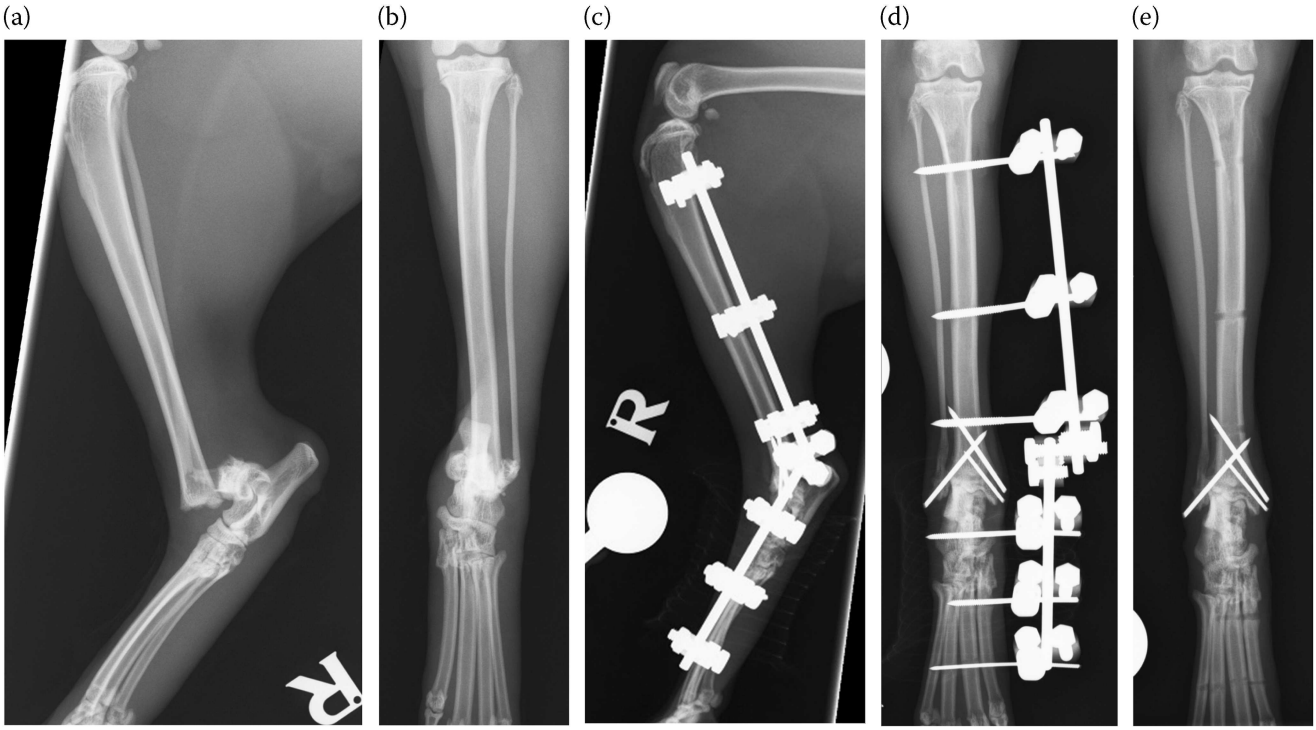


Figure 11.18 Radiography of the hock in this case of presumed talocrural luxation actually reveals caudo-proximal displacement of the pes in association with distal tibial (physeal) and distal fibular (malleolar) fractures. **(a, b)** Fractures are stabilized using Kirschner wires placed obliquely to avoid penetration of the hock joint and the repair is augmented with a transarticular external skeletal fixator for 4 weeks. **(c, d)** Following removal of the external skeletal fixator **(e)** the fixation pins can remain in place or they can be removed if they are prominent.

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Prosthetic Collateral Ligament Replacement

If the collateral ligament is irreparable, it is replaced with suture material to restore joint stability. This technique may also be performed to protect a repaired collateral ligament. Nonabsorbable, monofilament suture material is placed in a figure-of-eight pattern to replace both components of the collateral ligament.

In most cases, remnants of the damaged ligament help identify the anatomic sites of ligament attachment. Typically these are as follows:

Lateral collateral ligaments – the origin is the lateral malleolus.

The insertions are on the dorsal aspect of the base of the calcaneus and the coracoid process of the calcaneus.

Medial collateral ligaments – the origin is the medial malleolus.

The insertions are on the head and body of the talus.

Bone tunnels are drilled at the appropriate sites to serve as anchor points for the sutures. Alternatively, small screws with washers or bone anchors may be used to attach the suture. Sutures are placed, the hock is held in reduction and the sutures are tightened and tied.

TEMPORARY HOCK IMMOBILIZATION:

Transarticular Pin Fixation

A Steinmann pin is inserted through the base of the calcaneus, across the joint, and into the distal tibia. Typically this technique is combined with external coaptation (splint dressing). This technique of hock immobilization causes damage to the articular cartilage and should be avoided where possible.

Transarticular External Fixation (Figures 11.18 and 11.19)

A transarticular external fixator may be applied to stabilize tarsocrural luxations, either as a primary means of repair or in conjunction with collateral ligament reconstruction. Typically the frame involves a type 1a frame on the medial aspect of the tibia (three transfixation half pins 2.0–3.2 mm diameter) and medial aspect of the pes (three or four transfixation half pins 1.1–1.6 mm in the base of the calcaneus and in the metatarsals). A metal or acrylic connecting bar is used. A mini circular external skeletal fixator frame has also been reported for feline talocrural joint immobilization, with two half ring blocks on the medial aspect of the pes and two full rings on the tibia.

If the external fixator is the primary means of stabilization, it should remain in place for up to 6 weeks to allow healing of the periarticular soft tissues. Physical therapy is initiated after fixator removal to restore full joint motion.

Complications are not uncommon in cats with transarticular external skeletal fixation bridging the hock, being reported in 5/17 cases in one study and 13/32 cases in another. The majority of complications were mild (pin tract discharge and pin loosening); however, long-term function in some cases was limited by osteoarthritis and persistent instability.

Pan Tarsal Arthrodesis

Pan tarsal arthrodesis should be considered in cases of severely unstable tarsocrural luxation in cats, particularly where this is complicated by fractures of the articular surfaces.

TALOCALCANEAL LUXATION:

(Gorse 1990, Macias et al. 2000).

The talocalcaneal joint has three pairs of cartilage-covered facets and two talocalcaneal ligaments. The proximal ligament is located between the proximal and middle articulations. The distal ligament is located at the proximolateral aspect of the distal articulation. Traumatic rupture of the talocalcaneal ligaments permits luxation. The talus tends to displace dorsally, particularly if the talocentral joint also luxates. Luxation of the calcaneoquartal joint is also often associated with this injury, in which case the body of the calcaneus may displace dorsally or plantarly. Concurrent fractures of the talar neck and collateral ligament ruptures occur rarely.

Examination Findings

Clinical signs of talocalcaneal luxation include hindlimb lameness (often non-weight bearing), swelling in the region of the hock, which may be severe, and palpable joint luxation and/or instability.

Investigation

Radiographs are obtained to identify the luxation (Figure 11.20) and computed tomography should be considered if concurrent articular fractures are suspected.

Treatment and Prognosis

1. Closed reduction and stabilization with external coaptation may be possible in some cases.
2. Open reduction and internal stabilization is recommended. A dorsomedial approach to the tarsus is made via an incision from the medial malleolus to the base of the second metatarsal bone. Joint capsule and fascia overlying the talus is incised and retracted allowing access to the head and neck of the talus. Flexion of the calcaneoquartal and tarsometatarsal joints increases the space into which the talus is to be reduced. Digital pressure and/or pointed reduction forceps are used to reduce the luxation. Fixation involves a positional screw (2.0 mm is usually appropriate) from the head of the talus into the calcaneus. The screw is orientated from dorsomedial to plantaro-lateral (Figure 11.20). Immobilization of the talocrural joint has been recommended following this procedure, and may be achieved by external coaptation or transarticular external skeletal fixation.

INTERTARSAL AND TARSOMETATARSAL SUBLUXATION/LUXATION:

(Gunstraet al. 2019, Kornmayer et al. 2014, Kulendra & Arthurs 2014).

Luxation or subluxation of the transverse intertarsal joints or the tarsometatarsal joints results from hyperextension and tearing of the plantar ligaments and fibrocartilage. Such luxations are rare in cats. Luxation of the proximal intertarsal joint, common in the dog particularly in certain breeds, is not reported in the cat.

Examination Findings and Investigation

Non-weight-bearing lameness following trauma is expected. Diagnosis is based on palpation of joint laxity and stress radiography.

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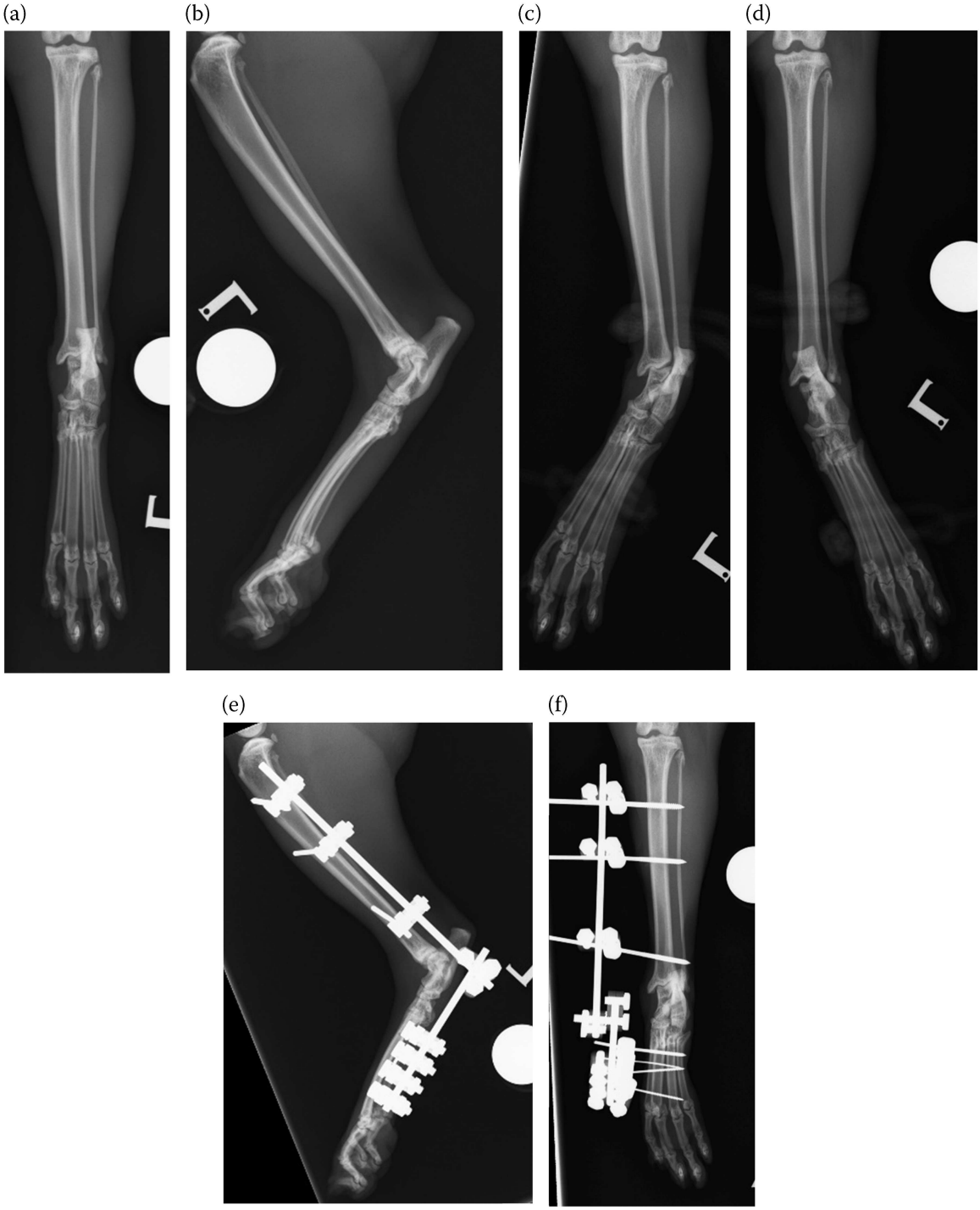


Figure 11.19 Investigation and treatment of feline talacrural luxation. Caudocranial **(a)** and mediolateral **(b)** views of the tibia and pes reveal no abnormalities other than soft tissue swelling in the region of the hock. Stressed radiography allows identification of lateral instability (varus stressed view, **c**) and medial instability (valgus stressed view, **(d)** Note the position of ropes on the distal tibia and distal metatarsus used to induce stress for radiography. In the absence of fractures of the medial or lateral malleoli, collateral ligament sprain is diagnosed. A transarticular external skeletal fixator is placed **(e** and **f)** with half pins placed to avoid hazardous corridors (medial on tibia and medial or lateral on pes).

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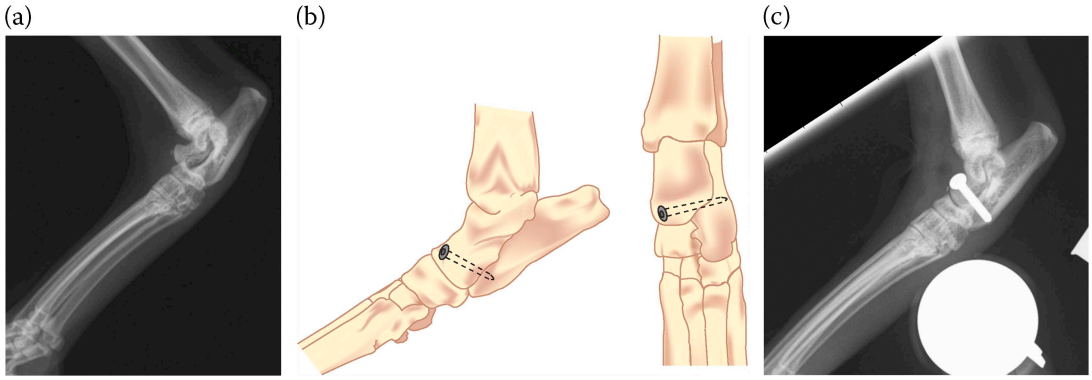


Figure 11.20 Talocalcaneal luxation stabilization. **(a)** Mediolateral radiographic projection of the hock of a cat with talocalcaneal (and talocentral) luxation. **(b)** diagrams of a talocalcaneal luxation stabilized with a 2.0 mm positional screw placed from dorsomedial in the talar head in a plantarolateral direction to engage the distal calcaneus. **(c)** mediolateral radiographic projection of the hock of the same cat in (a) following fixation of talocalcaneal luxation using a 2.0 mm positional screw.

Treatment and Prognosis

Two modes of treatment are described. Surgical treatment of similar injuries is preferred in dogs. In cats there is no evidence that non-surgical management is inferior to surgical stabilisation.

Non-surgical management

The affected joint is reduced and external coaptation involving a cast or lateral splint is placed, extending from the toes to the proximal tibia. External coaptation is maintained for up to 6 weeks to permit healing and stabilization by periarticular fibrosis. Particularly in cases where the plantar ligaments are disrupted, external coaptation may be ineffective, and this is not the authors' preferred approach.

Surgical management

Stabilization of transverse intertarsal and tarsometatarsal luxations involves ankylosis or arthrodesis of the affected joints by internal fixation. Anklyosis refers to the bridging of joints with implants, typically resulting in atrophy of interposed articular cartilage and some degree of eventual bony fusion, while arthrodesis refers to an attempt to remove articular cartilage, place some form of bone graft or synthetic substitute and bridge with implants, with the specific aim of achieving joint fusion. In the low motion transverse intertarsal and tarsometatarsal joints of the feline pes, ankylosis alone is typically sufficient to achieve long-term stability, although theoretically a successful arthrodesis will result in lower long-term implant strain.

Depending on the location of the instability tarsometatarsal or partial tarsal ankylosis/arthrodesis may be considered.

Tarsometatarsal Ankylosis/Arthrodesis (Figure 11.21)

The medullary canal of the metatarsal bones is typically significantly greater than that of the metacarpal bones in cats. Kirschner wires may be introduced through slots in the dorsal surface of the third and fourth metatarsal head. They are driven proximally within the medullary canal, often glancing off the plantar aspect of the bones until they penetrate at least the numbered tarsal bones. Usually they are driven further into the calcaneus and/or talus, though they are difficult to direct and penetration of the talocrural joint is to be avoided. Fluoroscopic guidance may be helpful, though frequent manipulation of the hock to ensure the absence of crepitus may be sufficient to detect articular penetration during pin placement.

Partial Tarsal Ankylosis/Arthrodesis (Figure 11.21)

Partial tarsal ankylosis/arthrodesis refers to fixation of all the joints of the pes except the talo-crural joint. Where a Steinman pin, driven from the proximal aspect of the calcaneus is used to provide proximal intertarsal ankylosis/arthrodesis, it may be driven further into the proximal metatarsus to achieve additional tarsometatarsal fixation if required (though directing it into one specific metatarsal base is not possible). Similarly, driving Kirschner wires placed from the metatarsal heads across the tarso-metatarsal and intertarsal joints will achieve partial tarsal ankylosis.

True arthrodesis is accomplished by removing the articular cartilage using a power burr, placing autogenous cancellous bone graft (or similar), and applying rigid fixation. Typically this involves a lateral bone plate with screws in the calcaneus, fourth tarsal bone and metatarsals. Fixation may also involve pin and tension band wire, Steinmann pin or cross-pin techniques as described earlier.

SHEARING INJURIES OF THE TARSUS:

Cause and Pathogenesis

(Diamond et al. 1999).

Shearing (degloving) injuries of the tarsus are theorized to result from being dragged behind or beneath a moving vehicle. The medial (and occasionally lateral) aspect of the tarsus is abraded by the surface of the road, destroying variable amounts of tissue, including skin, muscle, tendons, ligaments, and bone. In many cases, one or more of the tarsal joints are open and severely contaminated. Often the medial malleolus and collateral ligaments are destroyed.

Treatment and Prognosis

Shearing injuries of the tarsus are managed by

1. Careful cleaning and early debridement of the wound.
2. Proper open-wound management.
3. Early stabilization of the joints.

The wound is covered with a sterile bandage while the cat is stabilized and evaluated for other injuries. The cat is then

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anaesthetized and the wound is examined under sterile conditions. Initial management of the wound is as follows:

1. The wound is filled with sterile lubricating jelly and the surrounding hair is shaved.
2. Copious lavage, with sterile isotonic solution (or a 0.05% chlorhexidine solution), is performed.
3. Surgical debridement is performed to remove debris and devitalized tissues from the wound. Vessels and nerves must be preserved.
4. Following debridement and thorough lavage a sample is collected for culture and susceptibility testing and a broad-spectrum antimicrobial drug is administered. Cephalosporins (cefazolin/cefuroxime at 22 mg/kg IV every 8 hours) and clindamycin (11 mg/kg IV every 12 hours) have been recommended.

At this stage, it may be possible to convert the wound to 'clean' by lavage and debridement and primary closure may be considered, alongside suturing of damaged collateral tissue and placement of prosthetic ligatures if considered appropriate to stabilize the joint. However, in most cases insufficient tissue remains for primary closure, and it is undesirable to place implants where they cannot be covered with viable tissue, since they have the potential to harbour infection. These cases are managed as open wounds but immobilization should be considered early on, to promote neovascularization. A transarticular external skeletal fixator may be placed (hindlimb modified type 1 frame with half pins medially in the tibia and medially in the metatarsals, forelimb modified type 1 or type 2 frame with half pins craniomedially in the radius and half or full pins purchasing the metacarpal bones). The external skeletal fixator should be positioned such that it can remain in place for up to 4 weeks while wound management continues. A plantar/palmar splint can be used to immobilize the joint rather than an external skeletal fixator and this should be changed frequently to avoid complications.

Management of the wound may include wet-to-dry dressings or vacuum assisted closure to remove debris and encourage granulation. Once granulation tissue covers the wound, a dry nonadherent dressing is maintained with less frequent dressing changes. A splint may be incorporated in the dressings once the external skeletal fixator is removed. Wounds should heal completely by epithelialization and contraction in 10–12 weeks. If adequate soft tissue is present, secondary closure is considered over a healthy granulation bed. Rarely, a skin graft may be required.

Stabilization of Unstable Joints Following Shearing Injury

Temporary immobilization by external skeletal fixation alone may be sufficient to result in stabilization of traumatized joints through peri-articular fibrosis. This is more likely to be successful for the tarsus than for the carpus. Where instability remains (or returns) following removal of an external skeletal fixator (usually after 4–6 weeks), options include attempts to place prosthetic ligaments to replicate those which have been traumatized (non-absorbable suture material secured using bone tunnels, small bone anchors or screws) or arthrodesis.

Arthrodesis is also indicated in shearing injuries with severe trauma to the articular surfaces. In some cases, arthrodesis is performed only after primary treatment of the shearing injury has failed to restore adequate, pain-free function.

COMMON CALCANEAN TENDON RUPTURE:

(Cervi et al. 2010, Gelberman et al. 1999, Lister et al. 2009, Mughannam & Reinke 1994).

The common calcanean tendon consists of three parts:

1. Gastrocnemius tendon of insertion in combination with the soleus (a combination of three muscles located caudal and distal to the proximal tibia and known as the triceps surae) and plantaris tendons of insertion,
2. Combined tendons of insertion of gracilis, biceps femoris and semitendinosus,
3. Superficial digital flexor tendon (SDFT).

The triceps surae/plantaris and combined tendons of gracilis, biceps femoris and semitendinosus insert on the calcaneal tuberosity. The superficial digital flexor tendon courses over the calcaneal tuberosity and continues on the plantar aspect of the pes, dividing to insert on the plantar surfaces of the middle phalanges. The combined gastrocnemius and soleus tendons of insertion insert on the lateral aspect of the calcaneal tuberosity. The gastrocnemius and triceps surae musculotendinous units are important structures for maintenance of a digigrade posture in the hind paw. The common calcanean tendon may be lacerated through sharp trauma to the caudal aspect of the limb proximal to the calcaneal tuberosity. Complete and partial ruptures of the common calcanean tendon are uncommon injuries in cats.

Signs of Common Calcanean Tendon Laceration

The common calcanean tendon may be affected by sharp trauma (laceration), blunt trauma (rupture), and chronic tendinopathy. In the latter case cats may present with lameness associated with discomfort originating in a degenerating tendon or they may present with plantigrade (or somewhat plantigrade stance) resulting from complete or partial rupture of a degenerating tendon. Trauma to the common calcanean tendon involves sharp trauma to the skin and various components of the tendon itself. The aetiology of chronic tendinopathy of the common calcanean tendon is uncertain; however, this condition is not uncommon.

Complete rupture of the common calcanean tendon results in a plantigrade stance (the plantar surface of the tarsus contacts the ground), and must be differentiated from fracture of the calcaneus and instability in the intertarsal joints. One or more components of the tendon may remain intact. Where the gastrocnemius/triceps surae tendon is ruptured and the SDFT remains intact, a plantigrade stance is evident with hyperflexion of the digits.

Palpation typically helps identify the severity of laceration (i.e. whether any structures remain intact) or the location of tendon thickening. On palpation of the limb with the stifle fully extended, the tarsus will flex beyond 90°. As the tarsus is flexed, where the SDFT remains intact, the digits will also flex. Radiography excludes concurrent bone/joint injury but may identify mineralization within a thickened tendon indicative of a chronic disease process, i.e. chronic tendonopathy with acute tearing of some or all of the remaining structure. Ultrasonography may help with identification of which structures have been traumatized by acute sharp (or blunt) trauma; however, surgical exploration is often required.

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Conservative Treatment

Treatment for all forms of feline common calcanean tendon injuries may involve conservative management (exercise restriction, a period of 2–4 weeks' immobilization with external coaptation, appropriate analgesia, bodyweight optimization), and this has been reported with short-term success in two cats with avulsion of the common calcanean tendon. A lateral splint is applied from the toes to the proximal tibia. The tarsal angle should be approximately 120°. The splint is removed after 6 weeks, and activity is gradually reintroduced over a further 4–6 weeks.

Surgical Treatment Following Laceration

Surgical repair of the lacerated tendons is indicated. Initial management should involve lavage and broad spectrum antibiotic therapy.

1. An incision is made over the common calcanean tendon and extended as required to identify the tendon ends. Devitalized tissue is debrided.
2. Individual tendons are identified and purchase with a loop of suture may facilitate their manipulation. Extension of the tarsocrural joint will reduce tension and aid apposition of the tendon ends.
3. The tendons are sutured separately in a locking-loop pattern using monofilament, (2 or 3 M) nonabsorbable suture material. Where tendons are avulsed from the tuber calcis (or the laceration is near the tuber), they are reattached via bone tunnels (1.5 mm) drilled from medial to lateral through the dorsal surface of the proximal calcaneus. If an avulsion fragment can be identified and is sufficiently large, it may be reattached to the calcaneus using a pin and tension band wire technique.
4. To protect the repair during healing, the tarsocrural joint is immobilized in extension. This is achieved using a lateral splint, external skeletal fixator (modified type 2 with pins in the distal tibia and metatarsals), or by placing a 2.7 mm screw calcaneo-tibial screw (which may be protected by application of a lateral splint dressing).
5. Implants (external skeletal fixator or lag screw) should be removed after 4 weeks and a lateral splint maintained for a further 4 weeks. Where no implants are placed the lateral splint should be maintained for a total of 8 weeks.

Surgical Treatment of Chronic Tendonopathy

The approach to rupture of a common calcanean tendon in which chronic degeneration is apparent depends to some extent on the severity of the change. Surgery may involve a similar approach to the acute rupture/laceration. Where possible abnormal tendon tissue should be identified and sparingly debrided. Provided tension is not too great, the tendon ends can be apposed and sutured. Maintenance of hock extension for a period of 2–4 weeks following surgery is imperative to minimize the tension on the repair associated with limb use, and the use of a calcaneotibial screw or external skeletal fixator is recommended in this situation to maintain rigid tarsal extension.

Where tension is likely to be too great for uneventful healing to occur, or apposition of the tendon ends is not possible, pan tarsal arthrodesis may be considered and will give a more predictable outcome.

LUXATION OF THE SUPERFICIAL DIGITAL FLEXOR TENDON:

(McNicholas et al. 2000).

Lateral luxation of the superficial digital tendon has been reported in the cat. Clinical signs include intermittent lameness and swelling over the tuber calcis. Palpating the superficial digital flexor tendon while internally and externally rotating the tarsus may confirm the luxation. Treatment is achieved by suturing the retinaculum near the tuber calcis using monofilament, nonabsorbable suture material. The medial retinaculum is repaired to stabilize a lateral luxation.

PARTIAL TARSAL ARTHRODESIS (FIGURE 11.21):

(Barnes et al. 2013, Benlloch-Gonzalez et al. 2014, Theoret & Moens 2007).

Partial tarsal arthrodesis involves fusion of the proximal intertarsal, distal intertarsal, and tar-sometatarsal joints.

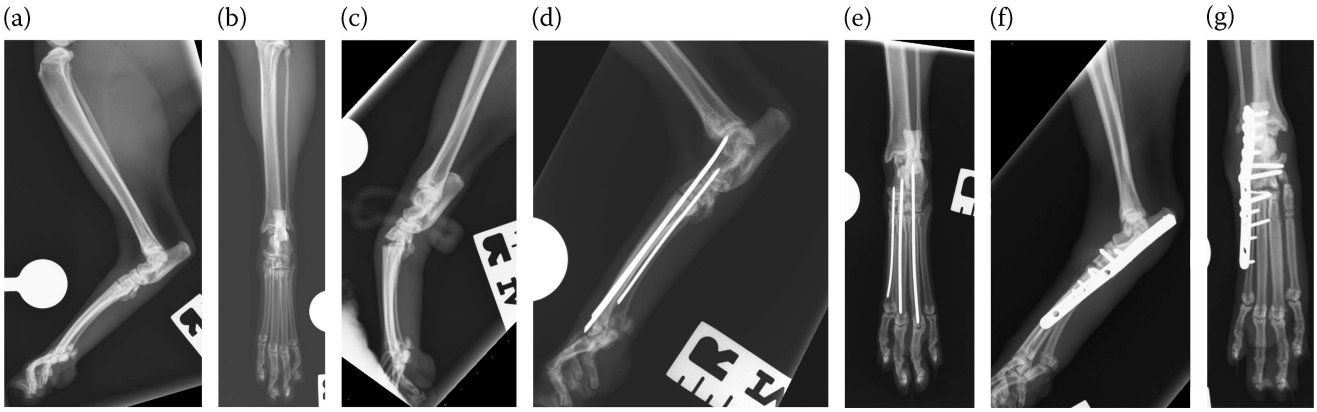


Figure 11.21 Treatment of dorsal instability at the level of the tarsometatarsal joint. Neutral mediolateral and caudocranial views demonstrate increased soft tissue opacity in the region of the distal hock joint **(a, b)**. Application of stress reveals dorsal instability at the tarsometatarsal joint **(c)**. Fixation may be simple, with transarticular Kirschner wires entering the metatarsals through distal dorsal slots **(d, e)** note the proximity to the hock joint of one of the pins unintentionally driven too far. Another Kirschner wire of the same length held on the outside of the pes will act as a guide to avoid driving Kirschner wires too far. An alternative if there is greater instability (particularly is this involves plantar instability, since the plantar aspect of the pes is the tension surface) is to perform plated partial tarsal arthrodesis **(f, g)**.

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Indications include hyperextension injuries and subluxation of the intertarsal and tarsometatarsal joints, comminuted intraarticular fractures of the tarsal bones, shearing wounds and severe osteoarthritis. The tarsocrural joint is spared, so limb function after partial tarsal arthrodesis is good or excellent.

The calcaneo-quartal and lateral tarsometatarsal joints are exposed via lateral or plantar approaches (depending on the fixation method) and the articular cartilage is removed with a curette or small power burr. The joints are aligned and autogenous cancellous bone graft is placed in the joint spaces. Stabilization is achieved using one of several techniques.

Pin and/or cross-pin fixation

A Steinmann pin is inserted normograde or retrograde (from the calcaneo-quartal joint) to bridge from the calcaneus through the fourth tarsal bone and into the fourth metatarsal base. The tip of the pin should be countersunk into the calcaneal tuberosity to avoid trauma to the overlying superficial digital flexor tendon. Two Kirchner wires are then placed in cross-pin fashion across the tarsus, one inserted through the fourth tarsal bone and driven medially and distally into the second metatarsal bone and the other started at the head of the fifth metatarsal bone and driven proximally and medially into the central tarsal bone. Compression is not achieved with this technique and stability may be compromised if pin engagement in bone is imperfect.

Pin and tension band wire fixation

A Steinmann pin is inserted normograde or retrograde as above. Holes are drilled from lateral to medial through the calcaneus and through the plantar tubercle of the fourth tarsal bone (or the plantar aspect of the base of the fourth metatarsal bone). Orthopaedic wire (0.8 mm) is passed through the holes to cross in a figure-of-eight fashion plantar to the tarsus and tightened to provide interfragmentary compression.

Bone plate and screws

A bone plate is placed along the lateral surface of the tarsus from the calcaneus to the fifth metatarsal bone. Veterinary cuttable plates have been reported in this role, although hybrid plates typically used for pan carpal arthrodesis are also useful in this situation. Screw sizes may be up to 2.7 mm in the calcaneus and tarsus and 1.5 mm or 2.0 mm in the metatarsus. To improve contact between the plate and the bone, the base of the fifth metatarsal bone and the calcaneus may be leveled with a power burr. Typically, three screws are placed into the calcaneus (one also engages the talus, lying dorsomedial to the calcaneus) and one or two screws are inserted into the tarsal bones. The three distal screws are placed into the metatarsal bones (near the metatarsal bases it is possible to engage all four metatarsal bones as they lie in a single plane, however, more distally fewer metatarsals are typically purchased as they are arranged in an arc (the optimal insertion corridors for transverse implant placement in the feline tarsus and metatarsus have been reported). A padded dressing is applied for three to five days to minimize soft tissue swelling and then discontinued.

Lateral plating was associated with the lowest rate of post-operative complications in a study of arthrodesis of the calcaneoquartal joint in dogs. Therefore this is the recommended approach to treatment of this condition.

TALOCRURAL ARTHRODESIS:

(Gorse et al. 1991, Kirsch et al. 2005, Sumner-Smith 1989, Theoret & Moens 2007, Yardimci et al. 2018).

Talocrural arthrodesis has been described for severe shearing injury, comminuted intraarticular fractures, joint laxity or hyperextension, irreparable damage to the common calcanean tendon, and debilitating osteoarthritis. The joint may also be fused along with transposition of the long digital extensor tendon for injury to the sciatic nerve. Arthrodesis of the talocrural joint alone eliminates the majority of tarsal joint movement. Additional stress on the remaining distal tarsal joints may lead to the development of osteoarthritis. Therefore, pan tarsal arthrodesis is often recommended.

The technique involves an approach to the talocrural joint utilizing a tenotomy of the medial collateral ligament or a medial malleolar ostectomy. The joint surfaces are prepared by removing the cartilage with a curette or power-driven burr. The joint is aligned and initial fixation is achieved by placing two Kirschner wires across the joint from the plantar aspect of the calcaneus or talus. The angle of talocrual arthrodesis is 115–125° in the sagittal plane in cats. Additional internal fixation is achieved using combinations of lag screws, intramedullary pins placed from the plantar aspect of the calcaneus and driven into the tibia, external skeletal fixation and bone plates and screws.

PANTARSAL ARTHRODESIS:

(Alza Salvatierra et al. 2018, Fitzpatrick et al. 2013, Gorse et al. 1991, Kulendra et al. 2011, Yardimci et al. 2018).

Pantarsal arthrodesis involves fusion of the tibiotarsal, proximal and distal intertarsal, and tarsometatarsal joints. Indications include shearing injuries, osteoarthritis and painful unstable conditions unresponsive to reconstruction. A lateral incision is made from the distal one third of the tibia to the distal metatarsus. The joints are prepared by removing the articular cartilage and placing cancellous bone graft. The preferred angle of arthrodesis is 115°–125° in cats. Rigid fixation is provided with one of several techniques typically through the application of a medial and/or craniodorsal plate and screws.

Cranio-dorsal bone plate and screws

Application of a plate in this position is biomechanically inferior as the plate is subjected to bending forces. However, a feline-specific plate has been reported in 11 cats with good long-term outcomes and one (clinically irrelevant) plate failure. Alternatively a 2.7 mm dynamic compression plate or veterinary cuttable plate may be applied to the cranial surface of the tibia and dorsal surface of the tarsus and metatarsus. The third metatarsal bone is often significantly larger than the other three metatarsal bones, so screws should purchase this bone. A cast or lateral splint may be applied to the limb during the healing period to reduce the likelihood of implant failure.

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Medial bone plate and screws

A bone plate is applied to the medial aspect of the tibia, tarsus and metatarsus. Reconstruction plates are readily contoured to allow arthrodesis at an angle of 115°–125°. However, they are relatively weak at the internodes, with concerns for maintenance of rigid stability for long enough to achieve joint fusion. Feline pan tarsal arthrodesis plates are available for medial application. The plate should be long enough to allow insertion of three screws in the tibia and three screws in the metatarsal bones. A cast or lateral splint may be applied to the limb during the healing period to reduce the likelihood of implant failure.

Cranio-dorsal and medial bone plate and screws (Figure 11.22)

To address concerns with high profile plates interfering with the overlying soft tissues but to provide sufficient stiffness for healing to occur, one study reported the application of orthogonal veterinary cuttable plates (a straight plate cranio-dorsally and a reconstruction plate medially). Outcomes for seven procedures in six cats were good. Casts/lateral splints are considered unnecessary following fixation with orthogonal plating.

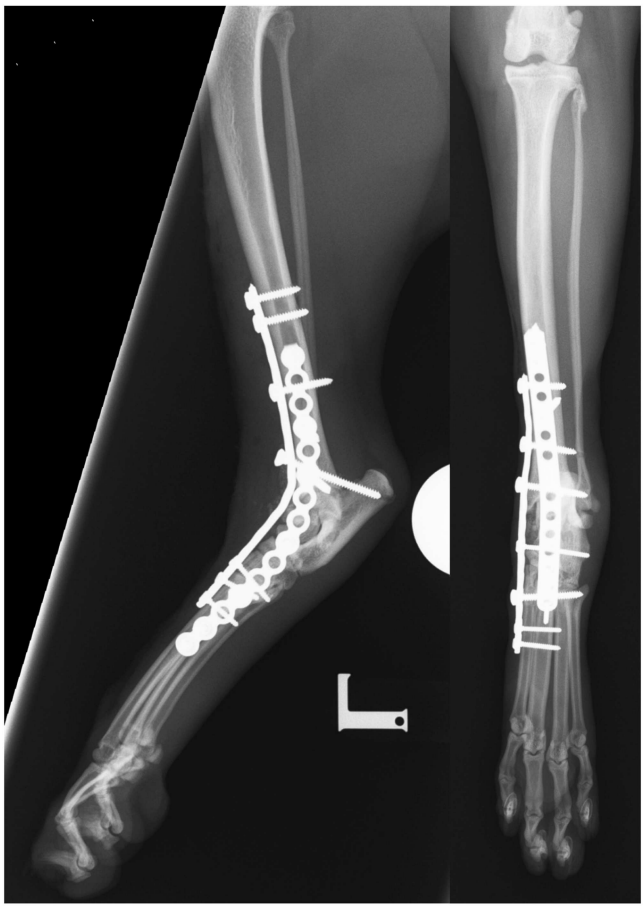


Figure 11.22 Pan tarsal arthrodesis performed using orthogonal plating. Veterinary cuttable plates are utilized. The plates should terminate at different levels on the distal tibia to avoid a stress riser. At least one screw in the cranial/dorsal plate should engage the calcaneus to improve stability.

External skeletal fixator

In cases of open fracture, where contamination is possible, external skeletal fixation may be employed to give temporary stabilization during healing of the arthrodesis. The articular cartilage is excised from the joints and bone graft is placed. The tarsus is positioned at an angle of 115°–125°. A modified type II or circular construct is applied with transfixation pins in the tibia (bilaterally in the distal tibia, medially in the proximal tibia) and bilaterally in the tarsus and proximal metatarsus. A potential complication of using external fixators is fracture of the metatarsal bones. Additionally, in the small bones of the cat there is a significant risk of premature fixation pin loosening necessitating implant removal prior to complete union of the arthrodesis.

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Part 4 Conditions of the Metatarsus and Phalanges

METATARSOPHALANGEAL AND INTERPHALANGEAL LUXATION:

Luxations of these small joints of the hind paw are managed as for metacarpophalangeal and interphalangeal luxation (see Chapter 9 part 4).

HINDLIMB AMPUTATION:

Amputation is a salvage procedure indicated for irreparable fractures or when severe complications occur after attempted fracture treatment. Neoplasia affecting the soft tissues or skeleton is also an indication for amputation. The cat should be carefully evaluated to ensure the remaining three limbs are healthy and can adequately support the bodyweight.

Amputation may be through the proximal one-third of the femur or by disarticulation of the hip (the latter is preferred where distal femoral neoplasia is the indication for amputation).

1. The limb is prepared from the dorsal mid-line to the ventral mid-line, caudally to the tail head and cranially to the last rib. A hanging-limb preparation may be used so the limb can be manipulated during surgery.
2. Asemicircular skin incision is made over the lateral surface of the thigh starting at the flank, extending over the distal third of the femur, and ending near the ischiatic tuberosity. A similar semicircular skin incision is made medially to complete the circular incision around the limb.
3. Medially, the sartorius and gracilis muscles are transected mid-belly. Electrocautery may be used to control small bleeding vessels. Ligation is used to prevent haemorrhage prior to transection of larger vessels. The femoral artery and vein and saphenous vein are separated, ligated, and transected.
4. The cranial belly of the sartorius muscle and the quadriceps muscles are transected proximal to the patella.
5. Laterally, the fascia lata and biceps femoris muscles are transected along the same line as the skin incision. Dorsal retraction of the biceps femoris muscle allows visualization and transection of the sciatic nerve at the level of the greater trochanter. The semitendinosus, semimembranosus and adductor muscles are then transected at mid-femur.
6. The femur is cut at the junction of its proximal and middle thirds using a bone saw or wire saw, or the gluteal muscles are transected from the greater trochanter and the hip joint disarticulated. The distal limb is removed.

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7. The remaining bone or exposed acetabulum is covered during closure by suturing the distal aspect of the quadriceps muscles to the adductor muscle and the biceps femoris to the gracilis. The caudal sartorius muscle is sutured to the fascia lata prior to suturing the subcutaneous tissues and skin. The longer skin flap on the lateral aspect of the limb provides a more cosmetic closure and places the thicker, hair-covered lateral skin over the end of the stump.

HEMIPELVECTOMY WITH LIMB AMPUTATION:

(Barbur et al. 2015)

Hemipelvectomy is occasionally indicated to achieve complete excision of neoplastic lesions of the proximal limb and/or pelvis. The technique utilized depends on the location and definitive diagnosis of the neoplasia. Options include internal and external approaches (removing the limb versus preserving the limb) and removal of cranial, central or caudal sections of pelvis.

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12 The Skull and Mandible – Fractures and Dislocations

SURGICAL ANATOMY:

(Hsuan et al. 2017).

The skull comprises the bones of the cranium or neurocranium and the bones of the face. The facial bones surround the eyes, and the respiratory and digestive passageways. The neurocranium is the neural part of the skull that surrounds and protects the brain. The dorsal portion of the neurocranium is known as the calvarium. The skull is a composite structure comprised of about 29 bones (depending on how a separate bone is defined). Many of these bones are bilaterally paired and are united by synarthroses (sutures), which are fibrous joints that are immobile in the adult. The major exceptions are the temporomandibular joint (TMJ) which is a diarthrodial or synovial joint and the symphysis of the mandible which is a synchondrosis or fibrocartilaginous joint that allows very limited movement between the left and right mandibles. The roots of the teeth of each superior arcade are embedded in the incisive bone (formerly premaxilla), and the maxilla and those of each inferior arcade are embedded in the body of the mandible. Most of the dorsal two-thirds of the body of the mandible are occupied by the tooth roots. The ventral third of the mandibular body includes the mandibular canal which contains the inferior alveolar artery, vein and nerve. The mandibular canal has one caudomedial opening, the mandibular foramen, and two rostrolateral openings, the mental foramina. The surgeon should be aware of the proximity of the inferior alveolar nerve and vessels, as they enter the mandibular foramen, when performing a medial surgical approach in this region, for example, for a unilateral mandibulectomy.

Adult cats have 30 teeth; the dental formula is 2(I3/I3, C1/C1, P3/P2, M1/M1). The superior jaw accommodates four cheek teeth (three premolars and one molar), while the inferior jaw bears three (two premolars and one molar). All of the incisors and canine teeth have one root, the superior 2nd premolar has one root, the 3rd premolar has two roots, and the 4th premolar has three roots while the superior 1st molar has two roots. The inferior cheek teeth (3rd and 4th premolars and 1st molars) all have two roots.

INTRODUCTION:

(Shields Henney et al. 1992, Soukup et al. 2013, Soukup & Snyder 2014).

When treating fractures of the skull and mandible the surgeon is confronted by a unique set of challenges that are not encountered when managing fractures elsewhere:

- Dental occlusion must be restored
- Dentoalveolar injuries are common
- Implants must not invade tooth roots
- Provision must be made for the patient to be able to ingest food and water
- Radiographic interpretation is complicated by the superimposition of bony anatomy
- Multiple fractures are common
- There is a high incidence of concurrent injuries

Approximately half of all cats presenting with fractures of the skull and mandible are likely to require medical or surgical management of their concurrent injuries in addition to treatment of their fractures. Concurrent injuries occur with greater frequency in cats with injuries caused by vehicular trauma. Careful assessment should be made of the eyes, the nervous system, and the thorax, in particular, before performing definitive fracture repair.

Traumatic dentoalveolar injuries involve the teeth or the supporting structures of the teeth (gingiva, periodontal ligament, alveolar bone), and have been reported to occur in up to 72.1% of dogs and cats with maxillofacial trauma. Injuries to the teeth are easily overlooked, but unaddressed they may be a source of chronic pain and may adversely affect oral function and the cat's quality of life. Fractures often occur through dental alveoli because the alveolus serves as a stress-riser and in some cases because of prior bone loss associated with periodontitis. Extraction of a tooth in the fracture line is only indicated if there is severe periodontitis or if the tooth root is fractured and cannot be stabilized. The tooth may need future endodontic treatment if the neurovascular supply was severed and should be monitored postoperatively, ideally with radiographs every 6 months for 1–2 years. The most frequent dentoalveolar injury is a crown (enamel-dentin-pulp) fracture that causes grossly visible pulp exposure. Most such fractures of the canine teeth are associated with pulp exposure because the distance between the pulp chamber and the surface enamel is small in cats. More severe fractures involving the crown and the root are not uncommon. In all instances of dentoalveolar trauma, rather than endodontic therapy being regarded as an alternative to extraction, extraction should be considered as a less desirable alternative to endodontic therapy when endodontic treatment is not possible for financial or other reasons. Severe dentoalveolar injuries may necessitate extraction, but in many cases teeth can be salvaged with appropriate dental treatment. For information about the management of traumatic dental injuries standard dentistry texts should be consulted or consideration should be given to referral to a veterinarian who specializes in veterinary dentistry.

DECISION-MAKING:

With regard to mandibular and maxillary fracture repair, restoration of dental occlusion is paramount. If the teeth are slightly displaced the fracture will heal but the patient may not be able to prehend and chew food correctly owing to the malocclusion. When choosing a method of surgical stabilization an important feature of this region is that the bones are non-weight bearing. A corollary of this is that fixation methods can be less robust than those required for equivalent fractures of the appendicular skeleton. Fracture management techniques can be classified according to four broad categories:

- Conservative/non-interventional
- Closed/minimally invasive
- Open/surgical
- Salvage

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Conservative treatment is indicated for selected fractures that are stable and non-displaced or minimally displaced with normal dental occlusion especially in immature cats. The anticipation is that the fracture will progress to bony union or that a fibrous union will provide an acceptable functional outcome. Conservative treatment entails a degree of confinement and the feeding of a soft diet for at least 4 weeks alongside appropriate antibiotic therapy and analgesia. Salvage procedures are generally used as a last resort and should be reserved for cats with very severe injuries or where standard methods have failed to achieve an acceptable outcome.

For many fractures the choice will be between closed/minimally invasive and open/surgical techniques, and the decision will be based on a number of factors including the number and configuration of the fractures, the expectations and finances of the client, the availability of referral services and the experience and technical ability of the clinician. An array of closed/minimally invasive procedures has been described and the choice of individual technique will depend to an extent on previous experience and personal preference. Open/surgical options are more limited and may be best performed by an experienced surgeon. Appropriate methods of management will be discussed for each fracture type with reference to these categories along with guidelines to assist the surgeon in the decision-making process.

DIAGNOSTIC IMAGING:

(Arredondo et al. 2012, Arzi et al. 2013, Bar-Am et al. 2008, Knight & Meeson 2018, Schwarz et al. 2002, Tundo et al. 2019, Winer et al. 2017).

Evaluation of radiographs of the head is challenging because of superimposition and the complexity of the bones of the skull. Correct positioning is important to avoid distortion, and provided injuries are unilateral, to take advantage of the bilateral symmetry of the skull. Radiological interpretation is made easier in the cat in comparison with the dog by the fact that there is much less anatomical variation between breeds. The authors find it helpful to have a skull and radiographs of a normal cat available when interpreting radiographs. Routine radiographic examination includes standard lateral and dorsoventral or ventrodorsal projections of the head (Figure 12.1). The mandible and temporomandibular joints can be evaluated on the lateral and dorsoventral projections, and if necessary, left and right 20° oblique lateral views can be obtained. The zygomatic arches, maxillae, and calvarium should also be evaluated for fractures.

Increased availability of CT together with technical advances in the images produced has been reflected in an increasing number of published studies of its use for cats with head injuries. CT is superior to conventional radiography for the identification and assessment of caudal mandibular, TMJ, and maxillary fractures, and to identify concomitant craniofacial injury (Figure 12.2). In a study that included 15 cats with traumatic maxillofacial injury, CT scans of the skull revealed twice the number of injuries in comparison with conventional radiographs, and the average number of injuries per cat identified by CT was 7.7. An additional benefit of CT is that the images can be used to generate 3D printed models to facilitate surgical planning for complex oral and maxillofacial surgeries such as defect non-union mandibular fractures.

Two recent papers have reported on the results of CT imaging of cats presented for treatment of head trauma. In the first involving 45 cats mandibular fractures were seen in 86.7% of cats. The median number of anatomical regions affected was 8 and fractures of the nasopharynx, orbit, nose, superior jaw, median palatine suture and zygomatic arch often occurred together and were frequently bilateral. There was also a high incidence of tooth fractures (64.4%) and fracture of one or both TMJs (56%). In the second study involving 75 cats, mandibular fractures, notably symphyseal



Figure 12.1 Radiographs of the skull showing fracture of the ramus of the right mandible. (**a)** Dorsoventral view. (**b)** Lateral view.

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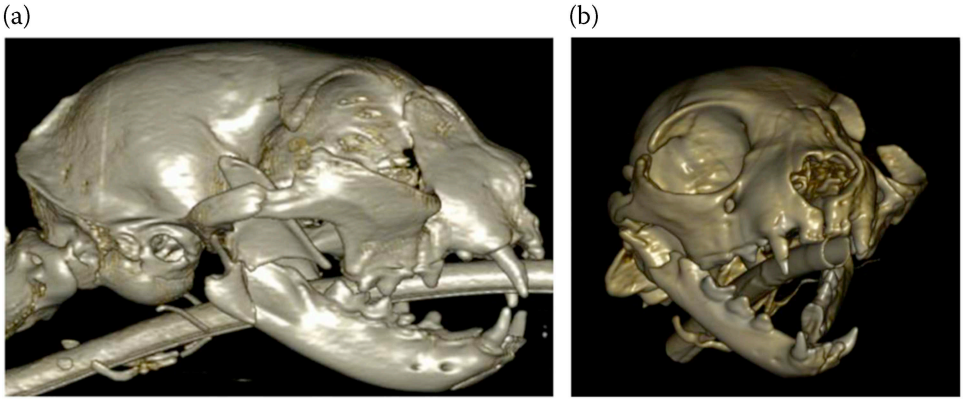


Figure 12.2 **(a)** CT 3D volume rendering of the skull of a cat with multiple maxillofacial and mandibular fractures showing fracture of the right mandibular ramus. **(b)** CT 3D volume rendering of the skull of the same cat. There are multiple craniofacial and mandibular fractures including a displaced fracture of the left mandibular body, symphyseal separation, left condylar fracture and left maxillary fracture.

separations, were the most common injuries seen. TMJ injury was usually unilateral and was seen in 56% of cats. In contrast, craniofacial and superior jaw fractures were most commonly bilateral. Multiple fractures were common occurring in 89% of the cats. Recognition of these multiple fractures may have a bearing on the management of the case, the prognosis given to the owner of the cat, and the final outcome.

MANDIBULAR FRACTURES:

Cause and pathogenesis

(Hill 1977, Mulherin et al. 2014, Phillips 1979, Scott 2015, Smith 2004, Umphlet & Johnson 1988, Woodbridge & Owen 2013, Zacher & Marretta 2013).

In comparison with dogs, cats suffering a traumatic episode are more likely to sustain maxillomandibular fractures relative to fractures elsewhere. This may partly be explained by their overall smaller size. Fractures of the mandibles in particular are common in cats and comprise approximately 11–23% of all fractures. Mandibular fractures may involve the mandibular body and/or the ramus of the mandible which is the caudal non-tooth-bearing, vertical part of the bone. The most common location is the mandibular symphysis, followed by the mandibular body and then the condyloid process and coronoid process. The most frequent causes are as a result of blunt trauma (often vehicular trauma), and less commonly an attack by another animal or injury caused by a metal projectile. Falls from a height, so-called feline high-rise syndrome, are an occasional cause of fracture (primarily of the mandibular symphysis). In comparison with dogs mandibular fracture caused by severe periodontal disease or neoplasia is rare. Mandibular body fractures are usually open.

Clinical Signs

Cats with mandibular fractures tend to hold the mouth partially open. Most caudal mandibular fractures result in overriding of the fracture segments and cause the rostral mandible to deviate towards the side of the injury. In bilateral fractures, the rostral aspect of the mandible will be displaced ventrally. Blood in the saliva and excessive salivation are common features. Pain and crepitus are elicited if attempts are made to palpate the mandible.

Diagnosis

The location and nature of mandibular fractures, particularly those of the mandibular body, can usually be determined on physical examination. Nevertheless, radiography or CT should be performed to rule out concomitant fractures, TMJ luxation and dentoalveolar trauma. Diagnostic imaging is also useful to identify fractures caudal to the molar teeth that cannot be seen on oral examination. Cats with mandibular fractures must be carefully evaluated for evidence of craniofacial injury. Other potential problems in cats with mandibular fractures include airway obstruction and inability or reluctance to eat and drink. Full assessment and stabilization of the patient should be performed before initiating treatment of the fracture.

Treatment and Prognosis

(Diez Bernal & Iff 2019, Soukup & Snyder 2015).

Anaesthesia is required to allow a complete oral examination and radiography or CT and to enable definitive repair of the fracture. Initially, the endotracheal tube is placed through the oral cavity. However, once repair of the fracture has begun, the endotracheal tube can be redirected and placed via a pharyngotomy or preferably a transmylohyoid incision to allow assessment of dental occlusion during the repair. In comparison with pharyngotomy intubation, transmylohyoid orotracheal intubation has the advantage of a simpler surgical approach and a lower risk of iatrogenic surgical complications. Alternatively, the endotracheal tube can be placed by performing a tracheotomy, but this is only needed when the airway is compromised and a tracheostomy will be required after surgery.

Antibiotics are indicated because mandibular fractures are usually open to the oral cavity. Fortunately, despite the open nature of most fractures, osteomyelitis is rare. Antibiotics directed at microorganisms common to the oral cavity are preferred, including ampicillin (25 mg/kg IV) or cefazolin (22 mg/kg IV). Amoxicillin/clavulanate, clindamycin, or metronidazole are good empirical choices of antibiotic for cats with severe periodontal disease. Targeted antibiotic therapy is rarely possible as culture of the fracture site will often reveal a wide variety of organisms, and determining which are likely to cause infection is difficult. In general, defects in the gingiva and other soft tissues of the

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mouth are left open to allow drainage and healing by second intention. If the defect is large, it may be sutured after copious lavage and debridement.

Several repair methods may be applicable for a given fracture configuration. The choice of technique will be based on a number of factors including the location and stability of the fracture, the age of the patient, concurrent injuries, financial considerations, the preferences and experience of the clinician, and the equipment available.

Biomechanics and Principles of Mandibular Fracture Repair

The goals of fracture repair are anatomic reduction and occlusive alignment, stable fixation, preservation of soft tissues, and avoidance of iatrogenic tooth damage. When choosing a method of fracture fixation it is important to have an understanding of the biomechanics of mastication. The dominant muscle pull on the mandible is from the muscles that close the jaw (temporalis, masseter, medial and lateral pterygoid muscles) and the primary force acting on the mandible during mastication is bending. Maximum tensile stress will be induced at the alveolar (oral) border and maximum compressive stress at the ventral (aboral) border of the mandible. If the tension band principle of fracture fixation were the only consideration when undertaking fracture repair, implants would always be placed on or near the alveolar border on the tension side of the bone.

As a result of muscle contraction the direction of a fracture line will influence inherent stability (Figure 12.3). For a fracture line that is perpendicular to the long axis of the mandibular body, the rostral segment will be displaced in a caudoventral direction, and the fracture will be distracted at its alveolar border. For simple oblique mandibular fractures the direction of the obliquity will affect inherent stability. A mandibular body fracture, with the fracture line running in a rostroventral direction, will be relatively stable because masticatory muscle forces hold the fracture segments in apposition (a so-called favourable fracture). A mandibular body fracture with the fracture line running in a caudoventral direction will be unstable because the muscular forces will lead to displacement of the fracture segments (a so-called unfavourable fracture).

In most cases, proper occlusion is achieved by providing adequate reduction of the fragments. Occlusion should not be sacrificed for better reduction, since fractures will heal even if slight gaps are present in the fracture line. However, seemingly minor malocclusion can lead to difficulty eating and pain post-operatively.

Stable teeth are generally not removed, even if the root is exposed. Leaving teeth prevents further damage at the fracture site and aids in occlusion, which helps stabilize the fracture. Early stabilization of the fracture supports compromised teeth and allows quicker healing of soft tissues. In the unlikely event of non-union, removal of the tooth at the fracture site may be indicated. Diseased teeth should be removed from the fracture site at the time of fixation.

Implants should be placed to avoid damaging the tooth roots, which occupy the dorsal half to two-thirds of the mandibular body. It is also preferable to avoid further injury to the neurovascular bundle in the mandibular canal, which occupies most of the ventral one-third of the bone. Hence, most corridors for implant placement are hazardous or unsafe, and there are few safe corridors.

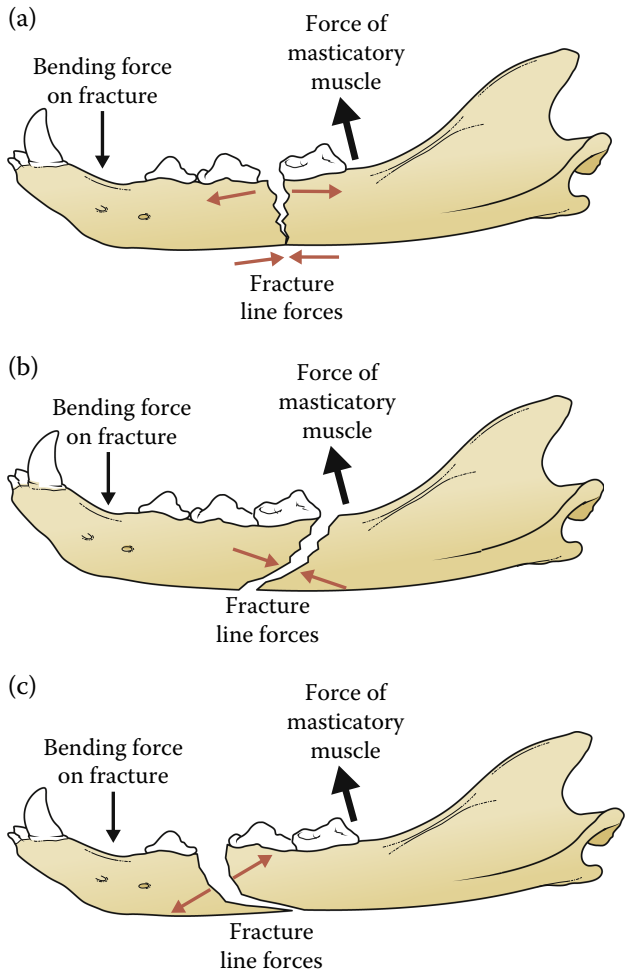


Figure 12.3 Biomechanics of mandibular body fractures. **(a)** A fracture line perpendicular to the long axis of the mandibular body will tend to open at its alveolar border. **(b–c)** For simple oblique fractures the direction of the obliquity will affect inherent stability. **(b)** A fracture line that runs from dorsocaudal to ventrorostral will be relatively stable because masticatory muscle forces will tend to hold the fracture segments in apposition. **(c)** A fracture line that is oriented from dorsorostral to ventrocaudal will be unstable because masticatory muscle forces will lead to displacement of the fracture segments.

Symphyseal Separations

(Mulherin et al. 2012, Saverino & Reiter 2018 , Vicari & Stepaniuk 2014).

Symphyseal separations are common, accounting for 73% of mandibular fractures in cats. Approximately 15% per cent of all feline fractures are mandibular symphyseal separations. The majority are separations of the mandibular syndesmosis caused by trauma. Symphyseal separation is usually readily identified on physical examination. However, radiographic or CT imaging of the head is still recommended as approximately a third of cats with symphyseal separation will have additional craniofacial injuries.

Symphyseal separations are readily stabilized with a single cerclage wire placed around the rostral mandibles (Figure 12.4). It is not uncommon for symphyseal separation to be accompanied by avulsion of the lower lip exposing the

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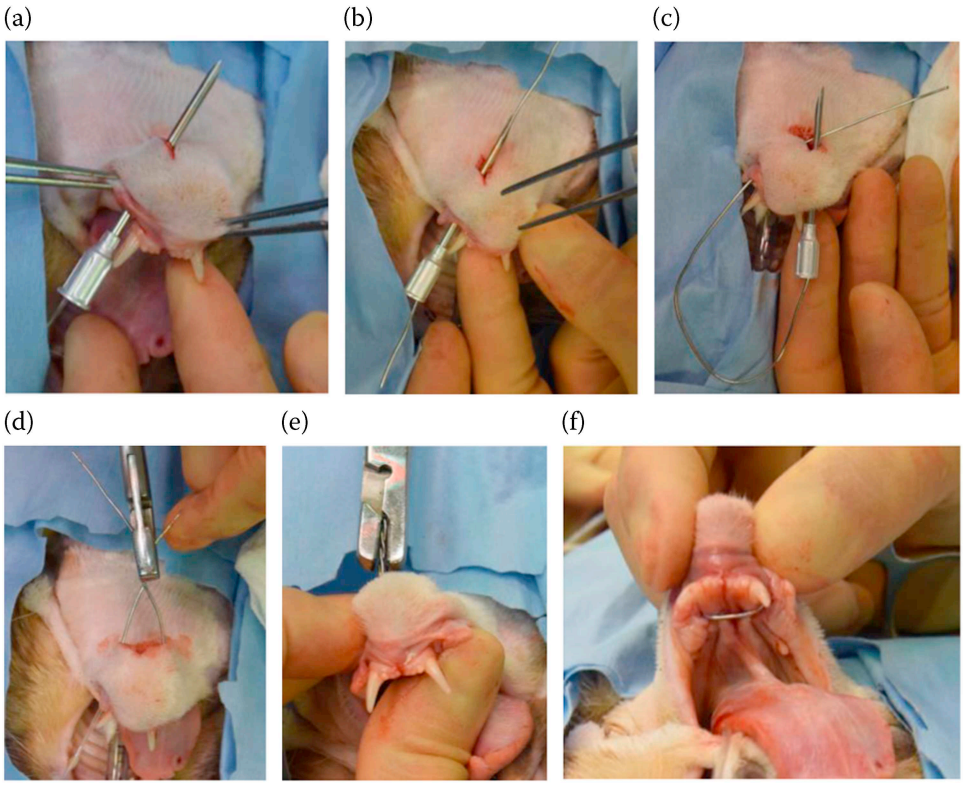


Figure 12.4 Placement of a cerclage wire in a cat with mandibular symphyseal separation. **(a)** A 16-gauge hypodermic needle is passed through the gingiva adjacent to the lateral aspect of the mandible just caudal to the ipsilateral inferior canine tooth. The needle emerges through a stab incision in the skin on the midline ventral to the mandibular symphysis. **(b)** Orthopaedic wire (0.8 mm/20 AWG) is passed through the needle and the needle is then withdrawn. **(c)** The procedure is repeated on the contralateral side of the mandibular symphysis. **(d)** The needle has been withdrawn and the wire is tightened by twisting. **(e)** The wire is tightened sufficiently to completely eliminate shearing forces at the symphysis. Overtightening is avoided as this may displace the canine teeth and result in malocclusion. **(f)** The tightened wire in situ. The twisted end of the wire is cut leaving 3–4 twists, bent over, and buried under the skin surface. One or 2 sutures are placed to close the skin incision.

bone of the rostral mandibles (Figure 12.5). Lip avulsion injuries should be treated at the time of fracture repair by wound debridement and lavage followed by appositional repair with simple interrupted and/or horizontal mattress monofilament absorbable sutures. Because cats lack the elasticity of the lips and cheeks seen in dogs the use of anchoring sutures around the crowns of adjacent teeth is usually necessary to provide additional support for the surgical repair. If teeth are absent, guide holes can be drilled through the alveolar margin to pass the suture material through.

To place a cerclage wire a 16-gauge hypodermic needle is used as a wire passer. The needle is inserted into the gingiva adjacent to the lateral aspect of the mandible just caudal to the ipsilateral inferior canine tooth and passed through the soft tissues to exit the skin in the midline ventral to the mandibular symphysis through a stab incision. Orthopaedic wire (0.8 mm [20 AWG]) is inserted through the needle from the skin end to exit in the mouth. The process is repeated on the opposite side with the other free end of the wire. The wire is then tightened by twisting ventral to the symphysis to compress the fracture.



Figure 12.5 Repair of right mandibular lip and gingival avulsion (courtesy of Matthew Oxford). **(a)** Preoperative photograph. This cat also has symphyseal separation. **(b)** Repair of the soft tissue injuries with interdental sutures and sling sutures around the teeth for additional support. The symphysis was subsequently repaired using an interdental wire and acrylic splint on the inferior canine teeth. **(c)** Photograph taken prior to interdental splint removal showing healing of the soft tissues.

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The wire should be tightened sufficiently to prevent shearing forces between the mandibles. Overtightening should be avoided as this may cause displacement of the canine teeth and malocclusion. The wire is bent over during final tightening to reduce soft tissue injury, cut leaving 3–4 twists, and buried under the skin surface. Alternatively the twisted end of the wire can be covered with a blob of dental composite resin and left protruding. The intraoral portion of the wire should be flattened against the intermandibular soft tissues. Irritation to the skin around the twisted end of the wire and mild drainage from the site is common. The wire is removed after 4–6 weeks when healing is complete. To remove the wire, the site of the previous stab incision is explored to find the twist, which is grasped with forceps. The intraoral portion of the wire is cut and straightened as the wire is pulled ventrally by the twist and out through the previous stab incision.

An alternative method involves placement of the wire such that the free ends are twisted intraorally caudal and buccal to one of the canine teeth. The twist is bent over during final tightening to minimize irritation to the tongue and other oral soft tissues. The technique simplifies wire cutting and removal which can then be performed entirely through an intraoral approach.

In some cases of symphyseal separation, for example, where there is comminution, extra stability and control over the angle of the crowns of the canine teeth is necessary. This can be achieved by placing a figure of eight of wire around the base of the canine teeth and adding a small amount of composite on the buccal surface of the canine teeth to cover the twisted end of the wire and prevent slippage. Stabilization of the symphysis using this latter technique alone but with the addition of composite resin to completely

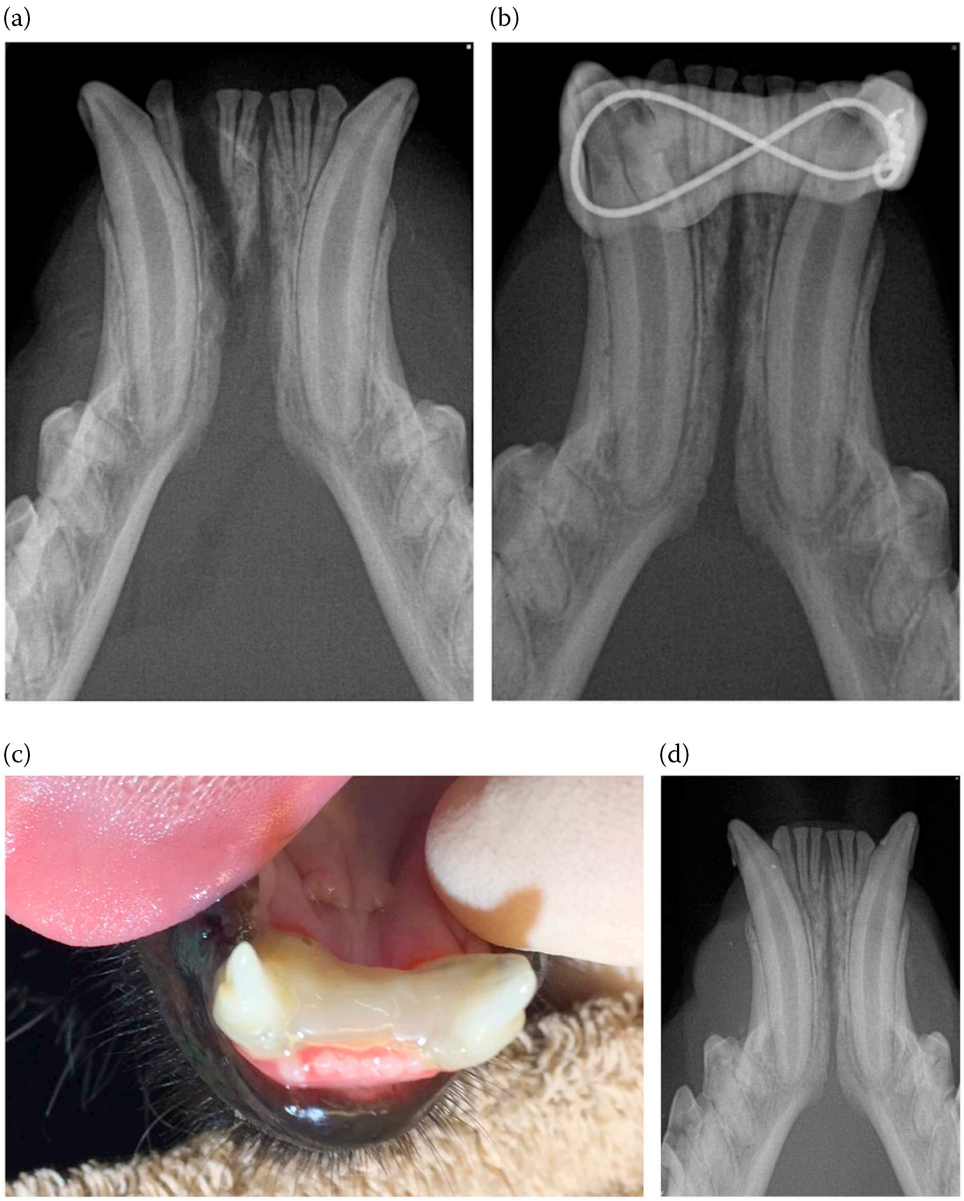


Figure 12.6 Placement of interdental wire and acrylic in a cat with mandibular symphyseal fracture (courtesy of Matthew Oxford). **(a)** Intraoral radiograph of the rostral mandibles of a cat with symphyseal separation and parasymphyseal fracture. **(b)** Repair using a figure of eight wire and a dental acrylic splint. **(c)** Photograph of the rostral mandibles showing the wire and acrylic splint. **(d)** Follow-up radiograph after removal of the splint showing healing of the fracture.

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encase the wire has been advocated as a minimally invasive method of repair (Figure 12.6). Whichever method is chosen implants should not be placed into the rostral mandibles as the roots of the inferior canine teeth occupy most of the width of the bone; approximately 70% of these teeth lie beneath the gingiva and are therefore not visible to the surgeon. As a result, inserting a pin or screw without hitting a tooth root is difficult. If the inferior canine teeth are absent a K wire can easily be placed across the symphysis to provide anchor points for the figure of eight wire.

Mandibular Body Fractures

Closed/Minimally Invasive Techniques

Closed or minimally invasive techniques for the management of mandibular fractures include muzzles, interdental fixation, and various methods of interarcuate fixation. Interarcuate techniques all involve maxillomandibular fixation in which the mouth is maintained in a closed or partially closed position and stability is conferred by interdigitation of the teeth. Maxillary-mandibular wiring, transarticular external skeletal fixation (TESF), intercanine bonding, the bi-gnathic encircling and retaining device (BEARD), and the modified labial reverse suture through buttons technique are all types of maxillomandibular fixation. The advantage of these techniques is that they are all relatively simple and inexpensive to perform. However, there are some disadvantages with all partial or complete mouth closure methods the most obvious being that the ability to prehend and masticate food is compromised for a prolonged period of time. There may also be an increased likelihood of nonunion or malunion as stability is likely to be much less than a correctly performed open reduction and fixation, and a prolonged period of immobilization may lead to soft tissue contracture and periarticular fibrosis with reduced TMJ range of motion. Mouth closure techniques are contraindicated in cats with vomiting or regurgitation because of the risk of aspiration pneumonia, and in cats with pharyngeal soft tissue swelling which prevents normal ventilation when the mouth is closed.

Muzzle Fixation

(Somrak & Maretta 2015).

Application of a tape muzzle is an inexpensive closed technique for stabilizing mandibular fractures. Unfortunately tape muzzles are often not well tolerated by cats and suitable candidates for muzzle application should be chosen carefully. A muzzle can be used to prevent movement of the fracture site temporarily and reduce further soft tissue injury while the cat is prepared for surgery. Alternatively, a muzzle can be used as a definitive form of stabilization or to provide additional support following internal fixation. When used alone, muzzles are applied to fractures that are relatively stable, minimally displaced, have satisfactory dental occlusion and where healing is expected to be rapid. Muzzles typically do not provide adequate stability for rostral fractures of the mandible. A muzzle may also be applied after closed reduction of TMJ luxation in cases where there is considered to be an increased risk of reluxation. Contraindications include brachycephalic breeds, vomiting or regurgitation, respiratory distress and cats with canine tooth fractures which prevent the teeth from remaining interdigitated. Clients should be instructed how to remove the tape muzzle quickly by cutting it if the cat vomits or becomes dyspnoeic.

Commercially available feline muzzles are often too large for use in fracture stabilization and may interfere with breathing or eating. The relatively short maxilla in cats can make maintaining a tape muzzle difficult. An Elizabethan collar is used to prevent the cat from removing the muzzle. The muzzle should be tight enough to hold the mouth shut while allowing approximately 5 mm of space for the tongue to protrude between the superior and inferior incisor teeth. The superior and inferior canine teeth should still interdigitate to prevent excessive lateral movement of the mandible. If the muzzle is too tight, the cat will be unable to open its mouth sufficiently to drink or eat a semi-liquid diet.

To make a customized three-strap tape muzzle one length of 25 mm wide semi-porous adhesive tape is placed ventral to the mandibles with the adhesive side away from the cat (Figure 12.7). The tape is pulled up and around the muzzle and crossed over as far rostral as possible over the bridge of the nose. A second length of tape, with the adhesive side towards the cat, is then placed ventral to the mandibles and crossed over the bridge of the nose so that it covers the first piece of tape with the two adhesive sides in contact with each other. At this stage there will be a gap ventrally and the eyes will be covered, but this is corrected in later steps. Without disconnecting the endotracheal tube the muzzle is pulled off the cat and cut with scissors in the ventral midline. The muzzle is replaced on the cat and the cut ends are brought together ventral to the mandibles to create a snug fit, with the tape conforming to the cat's muzzle rostrally on the bridge of the nose, without covering the nasal planum. A third short length of adhesive tape is placed to join the two free ends of the tape muzzle and cover the gap between them. Two lateral straps and a dorsal strap, made of tape folded on itself, are then attached to the tape muzzle. The lateral straps are placed approximately at the level of the zygomatic arches and are tied behind the head in the occipital region. The dorsal strap is placed in the midline and joins the lateral straps behind the head. The straps are secured to the tape muzzle using small pieces of tape which are folded over to the inside of the muzzle. With the muzzle fitted a marker pen is used to delineate the area of tape that needs to be removed so that the edges of the muzzle are not in contact with the eyes. The muzzle is then removed from the cat and surplus tape is trimmed away with scissors. The muzzle can be reinforced with additional tape as required. The muzzle is secured to the cat using the three straps. The dorsal strap is pulled caudally over the head. The side straps are pulled behind the head caudal to the ears and tied together using a single throw at the occipital region over the dorsal strap. The dorsal strap is flipped rostrally and a second throw is made with the side straps to trap the dorsal strap inside a square knot. The dorsal strap is now flipped caudally and a third throw of the knot is made to further secure it. Excess length is cut from all three straps. A least two muzzles should be fabricated whilst the cat is anaesthetized and then one is reapplied when the cat has been extubated and recovered from anaesthesia.

After application of a tape muzzle, fracture reduction may be imperfect. However, the fragments are usually guided into adequate alignment by occlusion of the teeth. If significant malocclusion develops, an alternative form of fixation should be chosen. This can occur if the fracture is inherently unstable or if the muzzle is too loose. Failure of the canine teeth to interdigitate may also allow the mandible

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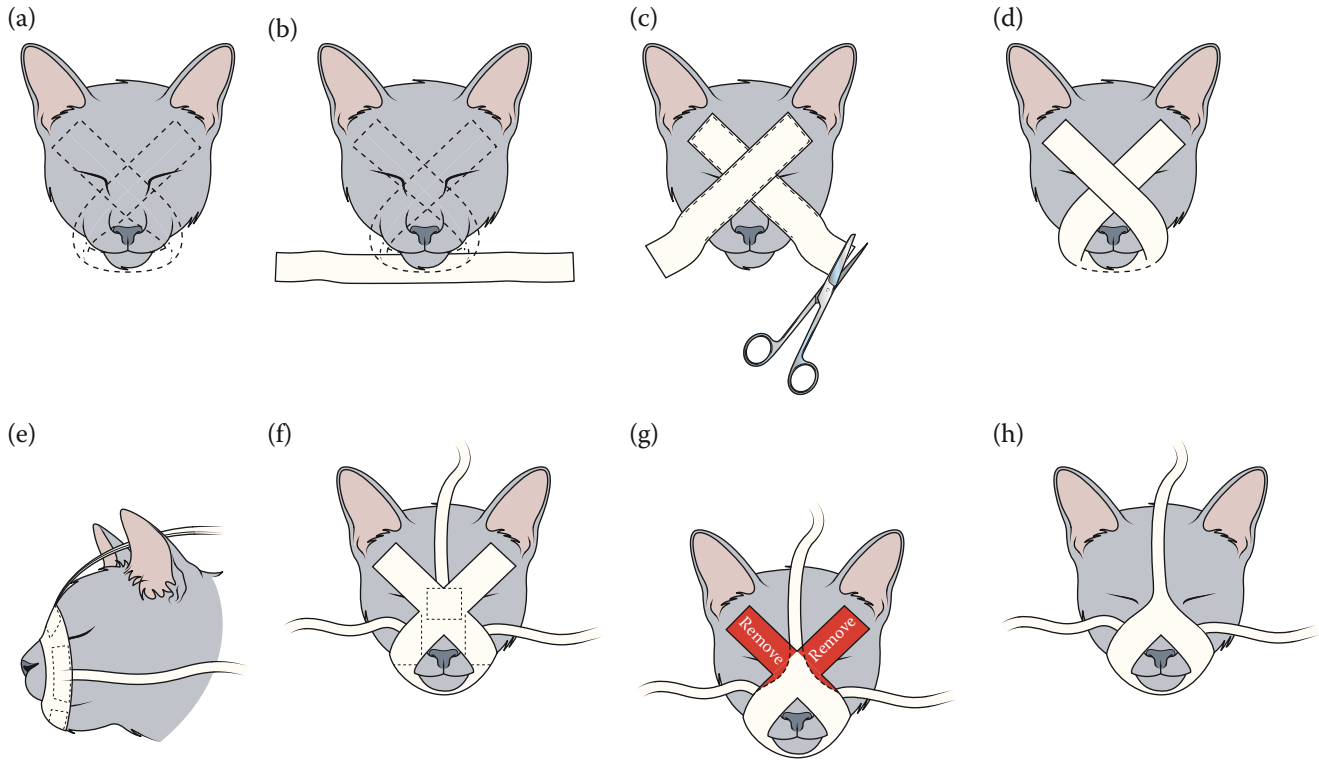


Figure 12.7 Tape muzzle application. **(a)** A length of 25 mm wide semi-porous adhesive tape is placed ventral to the mandibles with the adhesive side away from the cat. The tape is pulled up and around the muzzle and crossed over as far rostral as possible over the bridge of the nose. The dotted lines indicate that the adhesive side is facing away from the cat. **(b)** A second length of tape, with the adhesive side towards the cat, is then placed ventral to the mandibles. **(c)** The second tape is crossed over the bridge of the nose so that it covers the first piece of tape with the two adhesive sides in contact with each other. At this stage there will be a gap ventrally and the eyes will be covered, but this is corrected in later steps. Without disconnecting the endotracheal tube the muzzle is pulled off the cat and cut with scissors in the ventral midline. **(d)** The muzzle is replaced on the cat and the cut ends are brought together ventral to the mandibles to create a snug fit, with the tape conforming to the cat's muzzle rostrally on the bridge of the nose, without covering the nasal planum. A third short length of adhesive tape is placed to join the 2 free ends of the tape muzzle and cover the gap between them. **(e)** and **(f)** Two lateral straps and a dorsal strap, made of tape folded on itself, are then attached to the tape muzzle. The lateral straps are placed approximately at the level of the zygomatic arches and are tied behind the head in the occipital region. The dorsal strap is placed in the midline and joins the lateral straps behind the head. The straps are secured to the tape muzzle using small pieces of tape which are folded over to the inside of the muzzle. **(g)** With the muzzle fitted a marker pen is used to delineate the area of tape that needs to be removed so that the edges of the muzzle are not in contact with the eyes. The muzzle is then removed from the cat and surplus tape is trimmed away with scissors. The muzzle can be reinforced with additional tape as required. **(h)** The completed muzzle is secured to the cat using the 3 straps.

to displace laterally, leading to malocclusion. Complications with tape muzzles include malocclusion, aspiration secondary to vomiting or regurgitation, and skin irritation around the muzzle. Daily cleaning of the skin beneath the tape will reduce the severity of dermatitis.

Interdental Fixation

(Cetinkaya et al. 2011).

Interdental fixation involves the application of wires, or acrylic, or both, around the teeth to stabilize fractures of the mandible (Figure 12.8). An adequate number of healthy teeth must be present for interdental fixation to be effective. The technique avoids iatrogenic trauma to tooth roots and neurovascular structures in the mandibular canal, is minimally invasive, preserves blood supply and soft tissues, and restores occlusion. It is also mechanically strong since the tension side of the mandible is the alveolar border. A composite intraoral dental restorative splint (i.e. combining interdental wiring with acrylic) is stronger than either interdental wire or restorative alone. Interdental wiring used as the sole method of fixation is not advisable in the cat because of the large interdental spaces, the small size and conical shape of the crowns of the teeth, and the paucity of cheek teeth.

A modification of this technique has been described for the repair of unilateral and bilateral rostral mandibular body fractures in cats just caudal to the canine teeth. These fractures are challenging to repair because of the presence of the symphysis, which can easily be disrupted, and insufficient bone around the canine tooth roots for the placement of implants. The technique involves the placement of multiple cerclage wires around both mandibles to anchor a lingual arch bar that has been contoured to the shape of the lingual side of the alveolar margin. The circummandibular wires are placed using a hypodermic needle as a wire passer. An alternative method of wire placement involves passing the wires through

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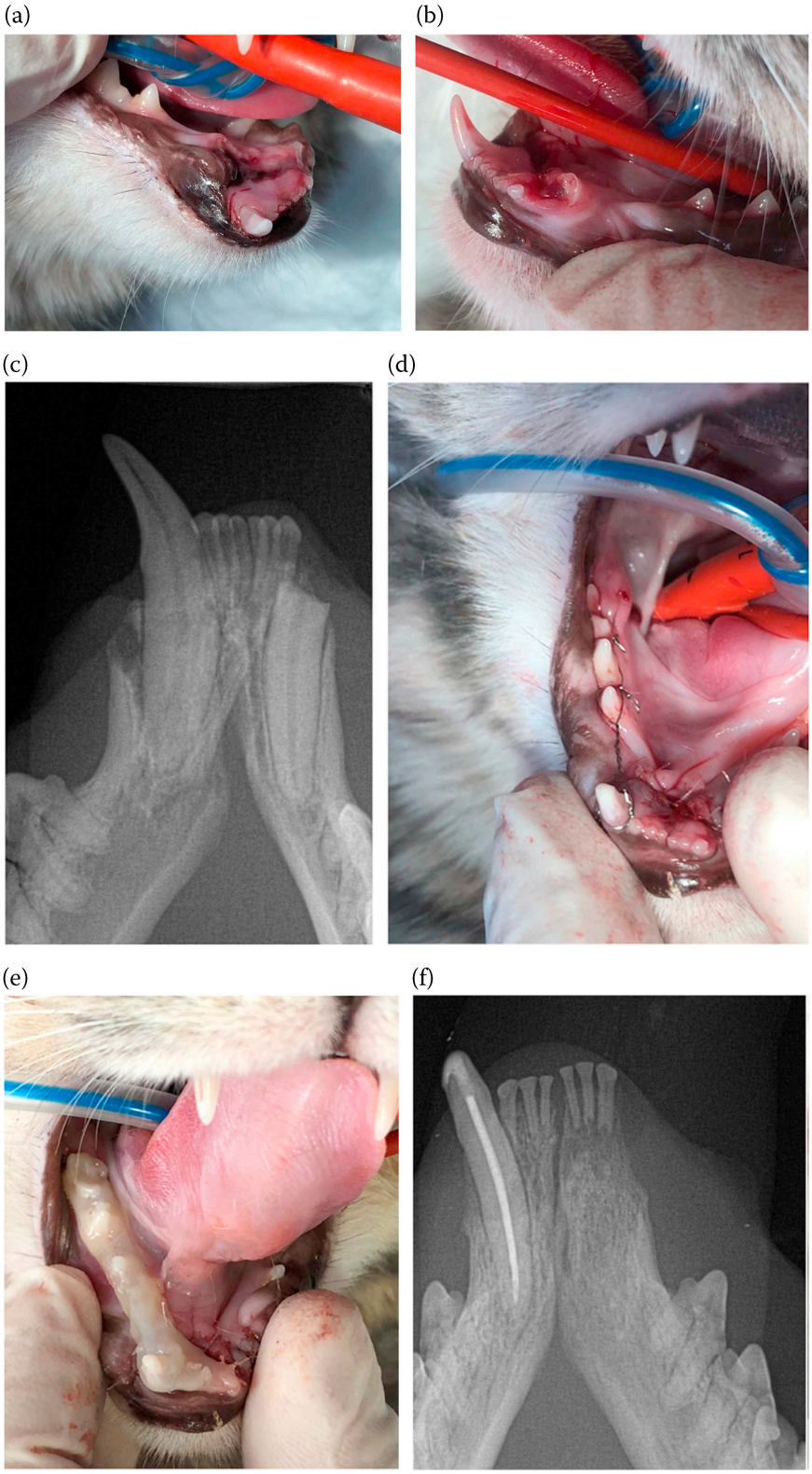


Figure 12.8 Interdental fixation of a mandibular body fracture (courtesy of Matthew Oxford). **(a)** Right rostral mandibular body fracture including alveolar fracture of the right inferior canine tooth. **(b)** The same cat showing a complicated fracture of the left inferior canine tooth. **(c)** Preoperative intraoral radiograph of the rostral mandibles. **(d)** Application of interdental wire to the right mandible. **(e)** Fracture fixation completed with application of acrylic to form an interdental splint. The left inferior canine tooth was extracted and root canal treatment was performed on the contralateral canine tooth owing to compromise of the apical vasculature. **(f)** Follow-up radiograph after removal of the interdental splint showing healing of the fracture.

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predrilled holes in between the tooth roots. The wires and arch bar are removed when fracture union is evident on clinical and radiographic examination.

Interarcuate (Maxillomandibular) Fixation

(Beckman & Smith 2009, Bennett et al. 1994, Goodman & Carmichael 2016, Hoffer et al. 2011, Lewis et al. 1991, Moores 2011, Nicholson et al. 2010).

This is a form of biological osteosynthesis that involves using wire, TESF, a suture or elastic or dental acrylic to fix the mandible and maxilla together to provide stability. The fixation is usually maintained for 3–4 weeks for TMJ dislocation and up to 6 weeks for mandibular fracture. If possible sufficient space (approximately 8 mm) should be left between the incisors to allow the cat to drink and eat gruel. Nevertheless, provision of nutrients via an oesophagostomy or gastrostomy tube is advisable. It is not unusual for a cat to require 24–48 hours to adapt to the device before it will eat, and it is better to have to remove a feeding tube prematurely, than it is to have to give a second general anaesthetic to place one. All of these closed techniques rely on anatomic interlocking of the dental arcades to achieve accurate restoration of dental occlusion. Cats with missing teeth, especially one or more canines, may not be suitable candidates for interarcuate fixation. Alternative techniques must also be used for very rostral mandibular, and of course symphyseal, fractures.

Maxillary-Mandibular Wiring

Wire (0.6–0.8 mm [22–20 AWG]) is passed through transverse holes drilled bilaterally in the alveolar bone of the mandibles and maxillae, being careful to avoid tooth roots. The wires are usually placed just caudal to the canine teeth. Wire is tightened by twisting until stable occlusal alignment is achieved. Maxillary-mandibular wiring has largely been superseded by other methods of mouth closure and is now rarely performed.

Transarticular External Skeletal Fixation (TESF)

Fixation pins or K wires are placed laterloaterally caudal to the superior and inferior canine tooth roots and connected using either elastic bands or epoxy putty connecting bars. Complications include trauma to tooth roots during pin insertion and pin tract infection (Figure 12.9).



Figure 12.9 Transarticular external skeletal fixation (maxillomandibular ESF). The mouth is left partially open to allow the cat to lap liquidized food.

Canine Interdental Bonding

The above techniques of interarcuate fixation have largely been replaced by bonding of the ipsilateral superior and inferior canine teeth (Figure 12.10). Canine acrylic bonding is particularly applicable if all four canine teeth are intact, but it may still be effective if there are two opposing canine teeth. Bonding has the same functional effect as external coaptation but, unlike the application of a tape muzzle, it is better suited to cats. Unfortunately, it is less suitable for brachycephalic breeds because of their jaw conformation and the small size of the canine teeth. The canine teeth are cleaned, pumiced, and acid etched to allow adherence of the restorative. Partial acid etching of the canine teeth has been recommended to permit easier removal of the composite resin without damage to the crown. In this case the acid etch is applied only to the apical 50% of the canine tooth surface (the portion of the crown closest to the gingiva). A thin layer of dental adhesive may then be applied and cured with an ultraviolet light. The fracture is reduced and the restorative material is applied to bridge the superior and inferior canine teeth on one side. The mouth remains open (approximately 1 cm between the mandibular and maxillary incisors) to allow the cat to eat and drink. If using light-cured resin several thin layers of restorative are applied and cured until fixation is adequate. The composite must be applied in increments of no greater than 2 mm to allow for penetration of the UV light. The procedure is then repeated to bridge the contralateral superior and inferior canine teeth. A modification of the technique involves placing a plastic cylinder (the flexible portion of a drinking straw) on the ipsilateral canine teeth and filling the cylinders with chemical-cured rather than light-cured restorative. Interarcuate canine acrylic bonding does not harm the teeth and avoids possible damage to the tooth roots associated with some other mandibular stabilization techniques. The restorative sometimes breaks or loosens before the end of the 4–6-week period but, by this time, healing is usually sufficiently advanced so that replacement is not required. The procedure carries the theoretical risk of electrolyte disturbances and acid-base imbalance secondary to excessive loss of saliva.

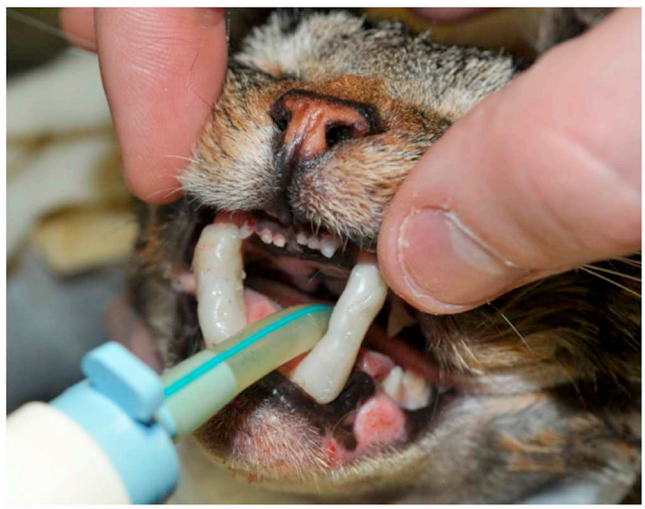


Figure 12.10 Interdental bonding of the canine teeth. The mouth is left partially open to allow the cat to lap liquidized food.

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(a)



(b)



Figure 12.11 Bignathic encircling and retaining device (BEARD). **(a)** Diagram showing BEARD application. **(b)** Photograph of a cat after BEARD application.

Bi-Gnathic Encircling and Retaining Device (BEARD)

This technique restricts maxillomandibular movement by the application of encircling nylon suture material (Figure 12.11). The suture (usually 18 kg test nylon leader line) is tunnelled subcutaneously using a large bore hypodermic needle (14 G) which has been pre-contoured into a 45° curve as a suture-passer. The needle is inserted through the skin overlying the dorsal midline of the maxillae to emerge within the oral cavity just caudal and lateral to a superior canine tooth. The process is repeated on the contralateral side using the same needle hole in the dorsal midline nasal skin to emerge at the same place within the mouth on the contralateral side. One of the free ends is then tunnelled through the gingiva just caudal to the inferior canine tooth lateral to the mandibular body to emerge through the skin over the ventral midline of the jaw. The process is then repeated on the contralateral side, resulting in a subcutaneous suture that encircles the nasal, incisive and maxillary bones and both mandibles with the 2 free ends of the suture exiting the skin at the ventral midline. The suture is then tightened sufficiently to achieve stable fixation by interdigitation of the canine teeth. Provided the reduced canine teeth remain interdigitated the suture can be tied loosely enough to allow the cat to eat semi-liquid food. One or two crimp clamps are then placed over the free ends of the suture and left uncrimped as spacers. Another clamp is then placed distally and crimped tightly.

The BEARD is a simple to perform and inexpensive, but unlike interdental bonding the mouth often has to be fully closed in order to achieve sufficiently stable fixation. An advantage over some other techniques is that the suture can be released easily by the owner using a pair of scissors if there is an emergency.

Modified Labial Reverse Suture through Buttons

This technique which has also been called 'labial locking with buttons' is similar in application to the BEARD insofar as it restricts maxillomandibular movement using suture material (Figure 12.12). The sutures are placed in the midline of the ventral chin then passed rostral to the canine teeth on both sides of the superior lip. The sutures are placed through buttons to distribute the pressure. In the original description of the technique two lines of suture were used, each connecting a ventral button to the left or

(a) (b)

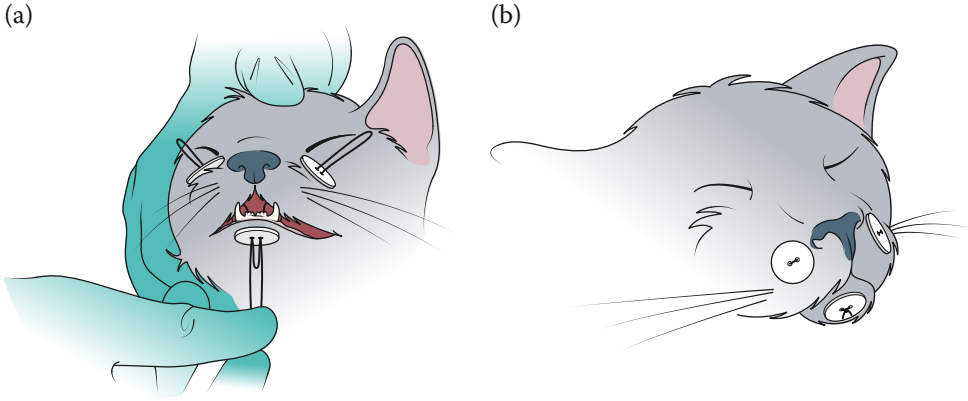


Figure 12.12 Modified labial reverse suture through buttons. **(a)** A single suture is placed to connect a button in the ventral midline with right and left superior labial buttons. **(b)** The suture is pulled to the appropriate tightness to maintain dental occlusion and is secured with a knot over the ventral chin button.

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right superior lip with knots tied over the labial buttons. In the modified technique a single suture is used to connect all three buttons with only one knot secured on the ventral chin button. The use of a single suture is more likely to maintain appropriate dental occlusion because equal tension will be applied to both mandibles. Provided the reduced canine teeth remain interdigitated the suture can be tied loosely enough to allow the cat to eat semi-liquid food.

The modified labial reverse suture through buttons technique is simple to perform and inexpensive but unlike interdental bonding the mouth often has to be fully closed in order to achieve sufficiently stable fixation. As with the BEARD an advantage of the technique is easy removal by the owner using a pair of scissors if there is an emergency.

Open/Surgical Techniques

Open or surgical techniques for management of mandibular fractures are by their nature more invasive and technically more challenging to perform than closed techniques. The benefit of surgery is immediate stabilization of the fracture and rapid return to normal function during the healing period. Open/surgical techniques include ESF, plates and screws, and interfragmentary wires.

External Skeletal Fixation

(Owen et al. 2004).

An external fixator can be applied to one or both mandibles to stabilize fractures (Figure 12.13). Indications include severely comminuted fractures, fractures that are grossly infected or contaminated, fractures where there is significant loss of soft tissue and fractures of atrophic edentulous jaws. The fixation pins are inserted through stab incisions in the skin over the mandible. It is important to ensure that the pins are placed at least 10 mm from the fracture line and do not damage tooth roots, the tongue and other oral soft tissues. The aim should be to place the pins in the interalveolar bone between the tooth roots. If the pin accidentally hits a tooth root, it may glance off and proceed through the bone, or the tooth enamel will prevent further insertion. The tooth may be damaged and the heat generated during the drilling can lead to premature pin loosening. Pins placed ventral to the tooth roots are likely to enter the mandibular canal and damage the neurovascular structures within. Placement of pins in the ramus of the mandible is suboptimal because pin purchase in the thin bone is usually inadequate and there is increased postoperative morbidity owing to penetration of the large masseter muscle. A minimum of two pins should be placed on each side of the fracture line. The mandible is quite thin with poorly defined cortices and threaded pins are recommended since they will enhance holding power and minimize premature loosening of the fixator. Fixation pins should be placed in a bicortical fashion but should not be placed across both mandibles as they may interfere with the function of the tongue.

When ESF is used for mandibular fracture repair a free-form acrylic or epoxy resin fixator is preferred since it is lightweight, can be conformed to the shape of the mandible and allows pins to be inserted in variable locations and still attach to the connecting bar. Small diameter pins are used to avoid damaging tooth roots and the structures in the mandibular canal. Once the transfixation pins are inserted, flexible tubing is placed over the protruding ends of the pins. The fracture is held in reduction (and the teeth are held in proper occlusion) while the tubing is filled with acrylic or polymethylmethacrylate, and it is allowed to harden. Alternatively, epoxy resin (or acrylic when it reaches a doughy consistency) is packed over the ends of the pins. Small K-wire size positive profile pins specially designed for use with acrylic frames (Miniature Interface fixation half pins, IMEX Veterinary Inc.) are preferred. These pins have a roughened area on the pin shaft to enhance the interface between the acrylic and the pin. Other types of fixation pin should be bent or notched to improve the interface between the pin and the connecting bar. The fixation pins are cooled using saline-soaked swabs during exothermic setting of the acrylic. The acrylic side bar should be 10–15 mm from the skin surface to help prevent thermal necrosis and allow for soft tissue swelling. Complications of external fixators used in mandibular fracture repair include premature pin loosening, infection and damage to the frame by objects in the environment. To remove the fixator, the pins are cut

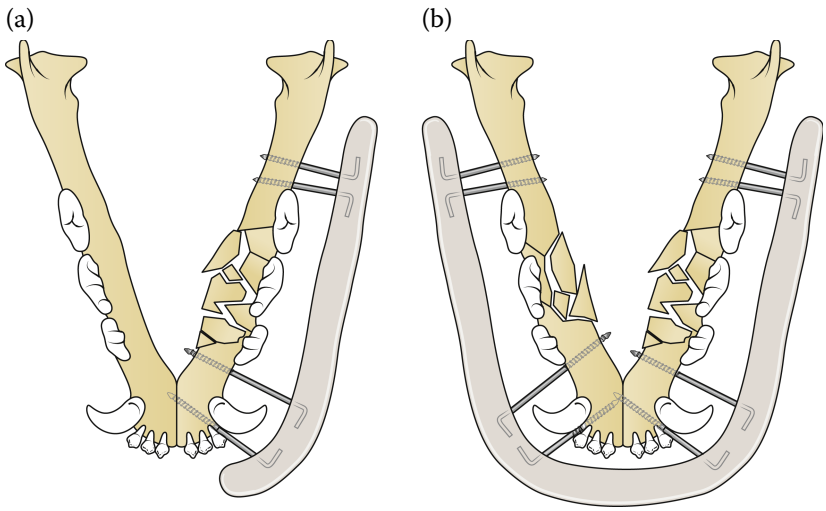


Figure 12.13 External skeletal fixation of comminuted mandibular body fractures. The rostral pins should be angled ventrally so that the acrylic side bar does not interfere with eating. **(a)** Unilateral acrylic external fixator. **(b)** Bilateral acrylic external fixator.

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between the mandible and the connecting bar, and then each pin is pulled out individually.

Plates and Screws

(Boudrieau & Kudisch 1996, Boudrieau 2012, Bilgili & Kurum 2003, Greiner et al. 2017, von Werthern & Bernasconi 2000).

The advent of locking plate technology has seen an increase in the use of plates and screws to stabilize mandibular fractures in cats. Plates and screws provide rigid fixation and a rapid return to normal function. A 1.5 mm cuttable malleable locking plate system is available (Veterinary Instrumentation, Sheffield) and a 1.5 mm veterinary locking compression plate system that accepts locking screws or cortex screws is available (DePuySynthes 1.5 mm LCP System). There is also a veterinary locking reconstruction plate system manufactured from titanium (DePuySynthes 2.0 mm Locking and Reconstruction Mini Plate System). These plates are low profile to avoid soft tissue irritation, can be cut to the appropriate length intraoperatively, and are easily contoured to the bone. Maxillofacial mini-plates designed for the treatment of humans with maxillofacial fractures have also been used for mandibular fractures in cats. Locking and non-locking 2.0 mm titanium plate constructs were recently evaluated in feline cadavers for repair of a transverse caudal mandibular osteotomy. The locking plate construct was mechanically stronger than the nonlocking construct although neither was as strong as the intact mandible.

Plating is performed using a ventral approach over the mandibular body. The plate should be placed on the ventral half of the lateral surface of the mandible. Plate application near the alveolar border on the tension side of the mandible would be most advantageous biomechanically but is not recommended because of the risk of screw placement in the tooth roots and the likelihood of gingival erosion over the implants. Screws must be placed carefully to avoid damage to the tooth roots and/or neurovascular structures in the mandibular canal. Locking screws can be placed in monocortical fashion if required. When placing a plate on the mandible, it is important to preserve dental occlusion. Unfortunately, because of plastic deformation of the bone at the time of injury, normal occlusion is not necessarily achieved even when the fracture is perfectly reduced. Therefore, the fracture may need to be stabilized with a small defect at the fracture site to ensure proper occlusion of the teeth. This requires visualization of the oral cavity during plate placement. If infection occurs, a second surgical procedure may be necessary to remove the implants once the fracture has healed

Interfragmentary wires

Interfragmentary wires can be used for stabilizing simple transverse or oblique mandibular body and ramus fractures, provided the contralateral mandible is intact (Figure 12.14). Ideally suited fractures are simple, unilateral and oblique with a fracture line that runs from dorsocaudal to ventrorostral so that there is already inherent stability when the mouth is closed. Interfragmentary wires are not recommended in comminuted fractures where fixation is difficult or when bone loss has occurred. The technique therefore requires careful case selection and is unforgiving of technical errors. The authors rarely use interfragmentary wires as the sole method of mandibular fracture repair.

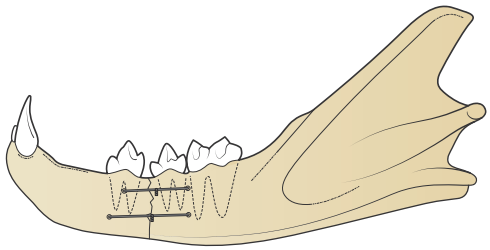


Figure 12.14 Interfragmentary wire fixation of a mandibular body fracture.

A ventral approach is made to the bone and the periosteum is reflected from the fracture site. The wire (0.6 mm [22 AWG]) is passed through oblique holes drilled through the mandible on each side of the fracture line (avoiding tooth roots). The drill holes should be 5–10 mm away from the fracture and should be angled towards the fracture line as this facilitates insertion of the wire medially and subsequent tightening. At least 2 wires must be placed across each fracture line to achieve stability. The first wire is placed close to the alveolar border (the tension surface) of the mandible as a tension-band wire. The second wire is placed as a stabilization wire near the ventral margin of the mandible. The tension-band wire must be placed perpendicular to the fracture line to provide interfragmentary compression and reduce shearing forces. The stabilization wire is often placed parallel to the mandibular body. It is important that the wires are tight, otherwise motion will soon cause cyclic failure of the implants. The wires are twisted together and bent over during final tightening away from the gingival margin, then cut short leaving 3–4 twists, to reduce soft tissue trauma. In most cases, interfragmentary wires are not removed unless complications develop.

Salvage Procedures

Salvage procedures, which include partial or complete mandibulectomy, are indicated where primary fracture repair is likely to fail or has already failed such as mandibular fractures with irreparable damage to the bone or soft tissues, osteomyelitis, sequestration, nonunion and severe bone loss. Cats that are unable to eat or drink as a consequence of failed primary fracture repair may be good candidates for mandibulectomy. Sufficient mandible is excised to remove infected bone and soft tissues and to permit tension-free closure of the oral mucosa.

Mandibulectomy

(Liptak et al. 2017, Northrup et al. 2006, Umphlet et al. 1988, Withrow & Holmberg 1983).

In contrast to dogs, partial unilateral, bilateral rostral, and complete unilateral mandibulectomy in cats are associated with a high complication rate. In a study of 42 cats undergoing a mandibulectomy procedure for treatment of oral tumours the majority of cats experienced significant short- and long-term complications from the surgery. Long-term (>4 weeks) adverse effects including dysphagia or inappetence, mandibular drift, palatine trauma secondary to malocclusion, and TMJ crepitus were seen in 42%, 37% and 18%, and 3% of cats, respectively. Mandibular drift occurs when the contralateral mandible is unstable and drifts towards the midline. Life-threatening

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morbidity was seen in 12% of cats which never returned to voluntary eating following surgery. Although the authors are not aware of any such studies in cats, histological evidence of temporomandibular osteoarthritis was found in dogs from 3 months after experimental unilateral rostral mandibulectomy. TMJ osteoarthritis is likely to be secondary to mandibular instability and abnormal loading of the joint. As an alternative to mandibulectomy, successful reconstruction of a mandibular segmental defect in a cat using a customized three-dimensional-printed titanium prosthesis has been described, and this may become more commonplace in the future.

Mandibular Ramus Fractures

(El-Warrak et al. 2011, Lantz et al. 1982, Salisbury & Cantwell 1989, Southerden & Barnes 2018).

The ramus is the caudal non-tooth-bearing vertical part of the mandible. The ramus has three prominent processes: the angular process caudoventrally, the coronoid process dorsally and the condyloid process caudally. The masseteric fossa is a prominent triangular depression on the lateral surface of the ramus where the masseter muscle inserts. Internal fixation of ramus fractures is problematic because the bone of the ramus is much thinner and weaker than that of the mandibular body, ranging from less than 1.5 mm thick in most of its central portion to 3.5 mm at its leading rostral edge. Additionally, implant placement involves considerable elevation of soft tissues. Because of the extensive musculature surrounding the ramus, displacement of bone fragments may be minimal. Conservative management using closed or minimally invasive techniques is appropriate for fractures where there is inherent stability and where normal occlusion can be achieved. Fortunately, dental malocclusion as a complication of fracture healing appears to be less common in this region of the mandible, and the presence of significant malocclusion at the time of presentation is often an indication that there is concomitant TMJ luxation. Fractures of the ramus dorsal to the condyloid process do not require repair.

Internal fixation of ramus fractures using plates and screws can provide a rapid return to normal function (Figures 12.15 and 12.16). An alternative to bone plating is the use of interfragmentary wiring. When using interfragmentary wire or a single bone plate, implants should be placed in the thicker bone surrounding the masseteric fossa along the condyloid crest (the ventral border of the ramus) and/or along the coronoid crest (the rostal border of the ramus), as there is less bone in the masseteric fossa for screw purchase and wire will pull through thin bone when tightened. The coronoid crest is the tension surface of the bone and is therefore the preferred location for plate placement. Double plate fixation of caudal mandibular fractures using stainless steel 1.0 mm mini-plates has recently been reported in two cats using three-dimensional models, with pre-contouring of bone plates and the use of a template to facilitate accurate plate positioning and anatomical fracture reduction. The longer of the two plates was placed first along the rostral surface of the ramus and then a

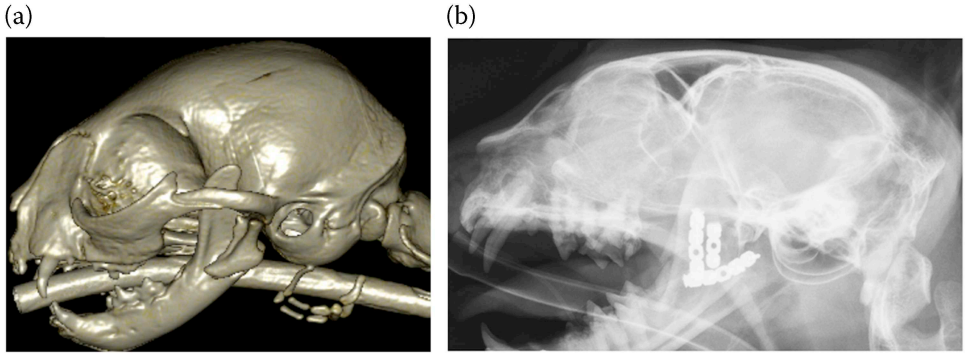


Figure 12.15 Plate fixation of a mandibular ramus fracture. **(a)** CT 3D volume rendering of a cat with a mildly comminuted fracture of the left mandibular ramus. **(b)** Postoperative lateral radiograph of the same cat showing fracture repair using three 1.0 mm plates and 9 screws.

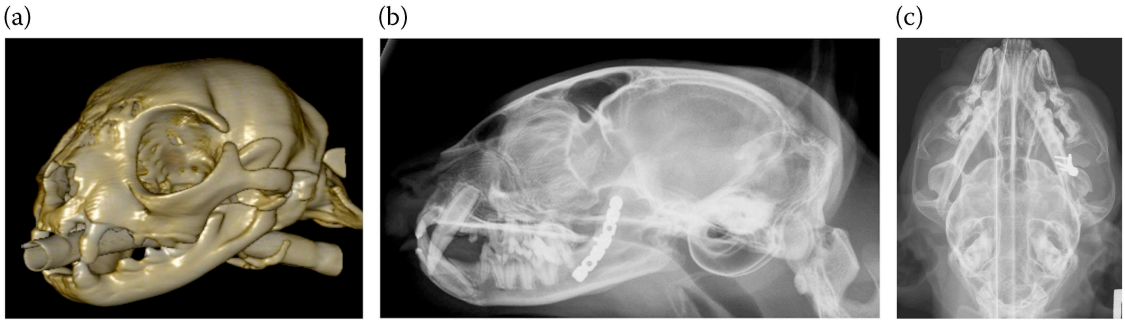


Figure 12.16 Plate fixation of a mandibular ramus fracture. **(a)** CT 3D volume rendering of a cat with a fracture of the left mandibular ramus. **(b)** Post-operative lateral radiograph following repair using a 1.0 mm plate and 5 screws. The plate has been placed along the coronoid crest. **(c)** Dorsoventral radiograph of the same cat.

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second shorter plate was placed parallel to this plate in the central part of the ramus. The fractures healed in both cats and normal jaw function and occlusion was achieved. The technique necessitates a CT scan, but compared with conventional plating with non-locking plates reduces surgical time and plate handling, and is likely to improve the accuracy of anatomical reduction of fractures. When using interfragmentary wiring a minimum of two wires should always be used as described for mandibular body fractures. The tension-band wire is placed first along the coronoid crest followed by a stabilization wire along the condylar crest. Accurate three-dimensional reconstruction with sufficient stability to withstand the forces of mastication using interfragmentary wire fixation alone is difficult to achieve; therefore, the authors prefer the use of two mini-plates. The surgical approach for placement of plates and interfragmentary wires is similar.

Fractures of the condyloid process are uncommon and when they do occur they are often associated with fractures of the symphysis or rostral mandibular body. Despite being an articular fracture, good results are normally achieved with conservative management and postsurgical periarticular fibrosis is avoided. If a functional outcome is not achieved following conservative management, condyloid excision can be performed. After excision arthroplasty a pseudoarthrosis develops as the area is filled with fibrous connective tissue. Potential complications include re-ankylosis, facial nerve paralysis and malocclusion.

The functional outcome after unilateral condylectomy is usually satisfactory despite the fact that the loss of one TMJ has been shown to lead to greater instability in cats than in dogs. In cats a degree of malocclusion is to be expected, as a result of displacement of the ipsilateral mandible, causing the jaw to deviate to the operated side. Sequelae of malocclusion include interference with mastication, painful mastication and secondary osteoarthritis of the unaffected TMJ. The effect of unilateral condylectomy on dental occlusion was reported in 12 feline cadavers, and it was found that there was a significant increase in latero-lateral amplitude of jaw movement which clinically would be expected to affect dental occlusion. This is in contrast to similar studies in dogs in which mandibular condylectomy appears to be a more benign procedure. The difference may be attributed to the anatomical differences between the two species, most notably the deeper mandibular fossa and the hinge-like motion of the normal TMJ in the cat.

Post-operative Management of Mandibular Fractures

(Clarke 2001).

After any form of fracture fixation, the oral cavity should be thoroughly lavaged. Most cats with mandibular fractures will eat soon after stabilization of the fractures. A liquid diet should be fed for the first 4–7 days after surgery and hard food should be avoided for at least 4 weeks.

If damage to the mouth and pharynx is severe, or if the fracture fixation technique prevents opening of the mouth, a nasopharyngeal feeding tube can be placed to allow tube alimentation before and after surgery. Nasopharyngeal feeding tubes should not be placed in cats that are vomiting, have a reduced level of consciousness or lack a gag reflex. They are also contraindicated in cats with fractures of the maxilla. Alternatively, if a nasopharyngeal tube is contraindicated or if it is anticipated that more long-term nutritional support will be required, an oesophagostomy or gastrostomy feeding tube should be placed while the cat is anaesthetized for radiographs or fracture repair. Although the goal is rapid return to enteral nutrition, tube feeding using liquidized tinned foods or commercial diets may be required to maintain the cat's caloric intake during the first few days or weeks after surgery. For further discussion of feeding tubes see Chapter 3.

All intraoral appliances will cause some trauma to soft tissues and will accumulate food debris and exudates from open fractures. In cats with oral wounds, or in cases where an intraoral appliance has been used for fracture stabilization, daily rinsing of the mouth with 0.12% chlorhexidine mouthwash or application of an oral antiseptic gel will reduce the severity of gingivitis and stomatitis. Compliance with such oral hygiene measures can be a problem and mouthwashes are often not tolerated. Cats seem to tolerate the taste of zinc ascorbate better than chlorhexidine, and this is available as an oral gel formulated for use in dogs and cats.

Complications of Mandibular Fracture

(Umphlet & Johnson 1988).

In the largest single study to date complications were common, affecting 24.5% of mandibular fractures in 62 cats, a figure that is higher than that reported for fractures of the appendicular skeleton. Complications include osteomyelitis, bone sequestration, delayed union, malunion, nonunion, TMJ osteoarthritis, true and false TMJ ankylosis and malocclusion. Additionally, the use any intraoral appliance for fracture repair will inevitably be associated with a degree of stomatitis and gingivitis, but this resolves rapidly once the appliance is removed. Treatment of delayed union, nonunion, bone sequestration and osteomyelitis should follow the same principles that have been discussed for the appendicular skeleton (see Chapter 6). Treatment of TMJ disorders is discussed in Chapter 13.

The most frequent complication is dental malocclusion secondary to malunion (Figure 12.17). Treatment for malocclusion is determined by the severity of the clinical signs. Treatment options include extraction of the malocclued teeth or immediate removal of the fixation device followed by accurate reduction and fixation. Crown height reduction combined with root canal treatment can be performed if there is malocclusion of the canine teeth and is preferable to extraction. Malocclusion associated with segmental bone defects can be corrected by bone grafting and plate fixation.

MAXILLOFACIAL FRACTURES:

(Buchet & Boudrieau 1999, Legendre 2005).

Introduction

Fractures affecting only the maxillofacial region are common in cats sustaining trauma to the head. In addition to the maxilla itself, which bears all of the premolar and molar teeth, fractures may involve the incisive (formerly premaxilla), palatine, nasal, zygomatic, lacrimal and frontal bones. The pattern of distribution of maxillofacial fractures is more predictable than that of mandibular fractures. As a rule fractures of this region are always multiple and are usually bilateral. Fractures in the nasopharynx, orbit, nose, superior jaw, median palatine suture and zygomatic arch region are likely to occur in combination. Fractures of the maxillofacial region often occur in concert with mandibular fracture and should not be overlooked.

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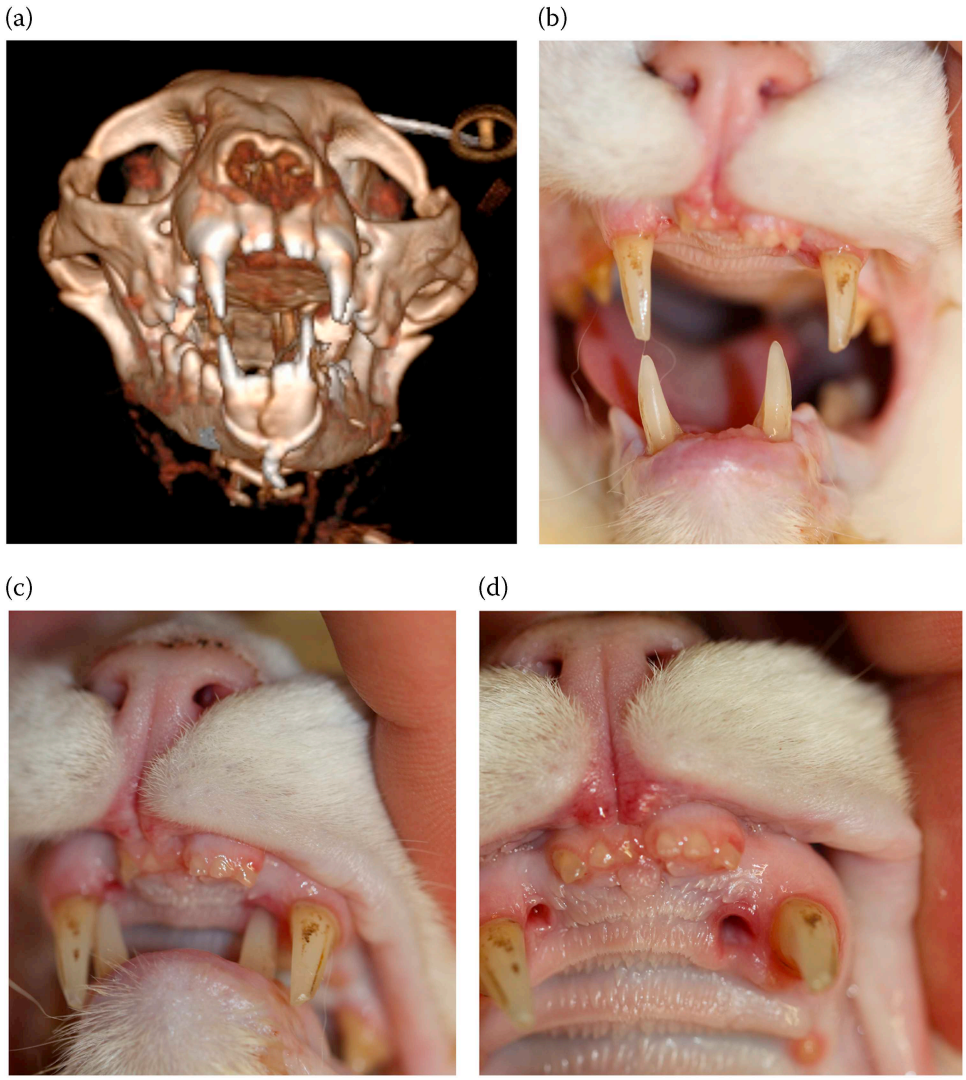


Figure 12.17 Malocclusion of the canine teeth (courtesy of Matthew Oxford). **(a)** CT 3D volume rendering following cerclage wiring for a symphyseal separation and previous BEARD application for the treatment of an unreduced temporomandibular joint . **(b)** Severe malocclusion of the canine teeth. **(c)** The inferior canine teeth impinge on the palate when the mouth is closed. **(d)** Palatal trauma caused by the inferior canine teeth.

Cause and Pathogenesis

Fractures of the maxillofacial bones and palate occur as a result of trauma. There is usually epistaxis associated with concurrent injury to the nasal turbinates which tends to be self-limiting.

Clinical Signs

Swelling, eye trauma, epistaxis and subcutaneous emphysema are common clinical signs. Dyspnoea and airway obstruction can occur due to intranasal haematomas and swelling, or if bone fragments are markedly displaced. Subcutaneous emphysema may be seen if fracture fragments penetrate the frontal sinuses.

Diagnosis

Cats with maxillofacial fractures should be carefully evaluated for concurrent injuries, particularly neurological deficits from cranial trauma. Physical examination is insufficient to assess the location and nature of the fractures owing to soft tissue swelling and the inaccessibility of the mid-face region. Radiographic examination is helpful to assess the extent of the injury to the skull. CT imaging has been shown to be superior to plain radiography to assess the full extent of the bony injuries (Figures 12.18 and 12.19).

Treatment and Prognosis

The majority of maxillofacial fractures are stable and minimally displaced and can be treated conservatively. The muscular forces on the maxilla are less than those on the mandible; therefore, less rigid fixation is needed and a fibrous union may be an acceptable outcome provided dental occlusion has been restored. The nares are kept clean to ease respiration and oxygen may be provided initially if needed. Anti-inflammatory drugs are administered to reduce swelling and pain. Most cats will drink and eat soft food, although nutritional support via an oesophagostomy or gastrostomy tube may be required in more severe cases. A tape muzzle can be used to stabilize the fracture site if needed.

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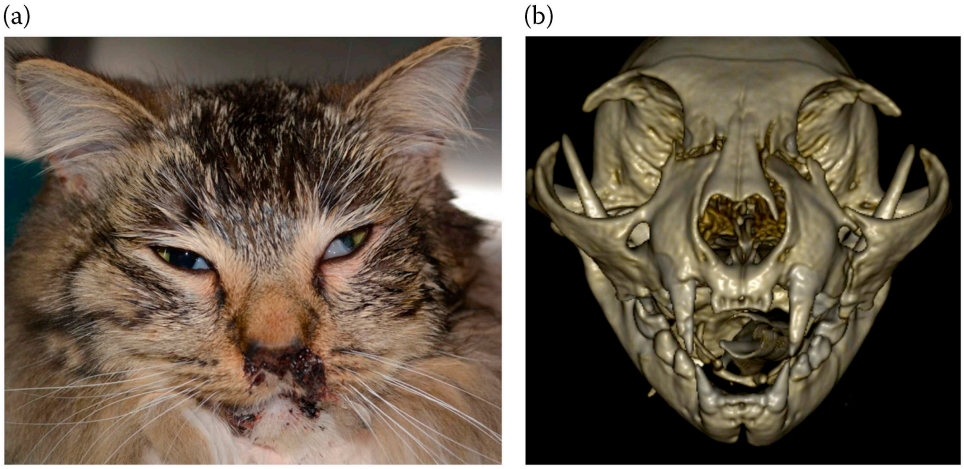


Figure 12.18 Maxillofacial trauma. **(a)** Cat with head trauma after being hit by a car. Note the left sided Horner syndrome. **(b)** CT 3D volume rendering of the same cat. The cat had multiple injuries including fracture of the right TMJ and fractures of the maxillofacial bones. A vertical fracture line through the left maxilla crossing the infraorbital canal is clearly visible.

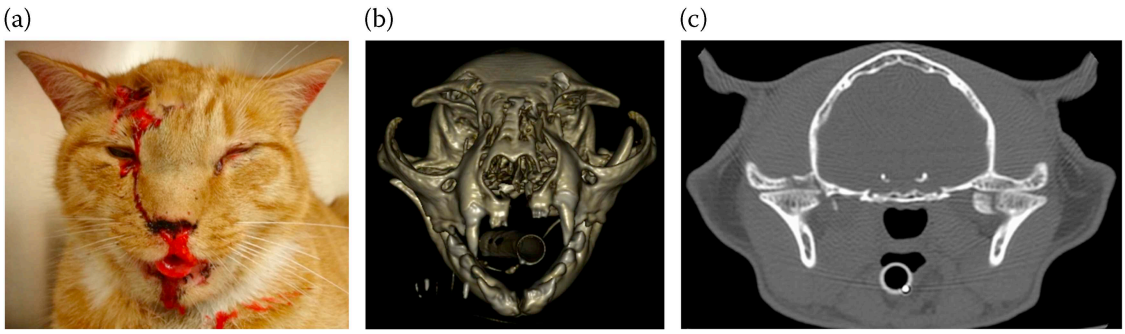


Figure 12.19 Maxillofacial trauma. **(a)** Cat with symphyseal separation and multiple maxillofacial fractures including median fracture of the hard palate after being hit by a car. **(b)** CT 3D volume rendering of the same cat. **(c)** Transverse CT image at the level of the temporomandibular joints showing fractures of both condyloid processes and the squamous part of the right temporal bone.

Surgical treatment of maxillofacial fractures is indicated when the nasal cavity is occluded, when marked displacement of bone fragments causes disfigurement, or when malocclusion with the inferior jaw is present. Surgery is performed once the initial swelling has resolved. If the fracture is reconstructable, bone fragments are reduced and stabilized with interfragmentary wires (0.5 mm [24 AWG]) or plates and screws. Nonreconstructable comminuted fractures are treated using interdental acrylic splinting or application of a bilateral acrylic external fixator. Rarely, a sequestrum will form at the fracture site and require excision. Avulsion fractures of the alveolar bone typically retain some mucosal or gingival attachments and are stabilized using Kirschner wires. If fragments must be removed, mucosal flaps are used to cover the defect and prevent oronasal fistula formation.

Correction of malocclusion secondary to stable fractures of the maxilla where there has been collapse and interdigitation of bone with slight deviation of the maxilla can be performed by realignment of the mandibular symphysis. The technique can be performed acutely at the time of the injury or after a malunion has developed and involves separation of the symphysis, if this is not already disrupted, and repositioning of the 2 mandibles so as to realign the teeth of the superior and inferior dental arcades. Mandibular realignment is obtained by moving one mandible in a lingual direction and the contralateral mandible in a labial direction, while simultaneously maintaining symphyseal contact, and thereby restoring occlusion to match any maxillary deviation. To prevent movement of the symphysis during subsequent placement and tightening of the cerclage wire a small K wire can be placed across the rostral mandible as either a temporary or permanent fixation device.

Palatine Fractures

(Bonner et al. 2012, Vnuk et al. 2003, Whitney & Mehlhaff 1987).

Fractures of the hard palate and nasal bones in the median plane are common in traumatized cats. The fracture is a component of the triad of injuries (thoracic trauma, orofacial trauma, and limb fractures) termed 'high rise syndrome'. Injury occurs when a cat jumps or falls from a height of two storeys or more and lands on its forelimbs and chin. Ventral trauma forces the mandibles dorsally, creating

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distraction forces between the maxillae, which results in fracture in the median plane often accompanied by a cleft palate. The fracture is characterized by a cleft in the soft tissue and bone of the hard palate and is readily diagnosed on oral examination. Cats with palatal fractures may have epistaxis and may have blood in the saliva. In one study of 84 cats with high-rise syndrome, midline hard palate fracture was seen in 20.5% of the cats.

The approach to treatment of acute palate fracture is controversial. In the opinion of the authors surgery is the preferred option for all acute fractures as soon as the cat is stable enough to undergo general anaesthesia. Conservative treatment of palatine fractures has been recommended, with a diet of soft food for at least 4 weeks. Small midline defects less than 2–3 mm in width may heal uneventfully, but there is a risk of the cat developing an oronasal fistula. Treatment of palatal fracture at the time of first presentation is much more straightforward than surgical correction of a chronic oronasal fistula. Furthermore, surgical repair will prevent food and water from entering the nasal cavity while the fracture is healing.

If the injury is recent, the defect can be reduced by approximating the bones using digital pressure or pointed reduction forceps on either side of the maxillae. For small defects and stable fractures the torn palatal soft tissues are closed with simple interrupted absorbable monofilament sutures. If the fracture is unstable and/or the defect is large osteosynthesis is performed using a wire and acrylic interdental splint attached to the superior canine teeth (Figure 12.20). If one or both superior canine teeth are missing a transfixation pin and wire technique is used instead. With the palate reduced, a K wire is driven across the bony palate rostral to the premolar teeth and perpendicular to the fracture line. The angle of the pin should be maintained parallel to the hard palate and carefully controlled to avoid penetration of the soft tissues. The free end of the wire is bent at right angles and cut to leave a small hook. The wire is then pulled back until the hook sits snugly against the maxilla. The opposite end of the K wire is then bent in a similar fashion so that it sits tightly against the contralateral maxilla and then cut. If further stability or compression is required an intraoral figure of eight cerclage wire is then placed around the hooked ends of the K wire. The wire can be left exposed in the oral cavity or can be embedded in dental acrylic. Alternatively, the figure of eight wire can be buried by passing it between the bone and the mucoperiosteum before looping it around the ends of the interfragmentary K wire. If the wire is left exposed the ends should be twisted together laterally to avoid trauma to the tongue and lips and then bent over during final tightening. If necessary, the oral mucosa overlying the palate is apposed with simple interrupted absorbable monofilament sutures.

Postoperatively there should be rapid relief from pain and cats are usually willing to eat the day after surgery. The patient should be fed soft food and observed for signs of rhinitis. If the intraoral wire is left exposed, the surgical site may be lavaged daily with water or 0.12% chlorhexidine mouthwash using a syringe or zinc ascorbate oral antiseptic gel can be applied. The implants are removed after 4 weeks or when healing is complete.

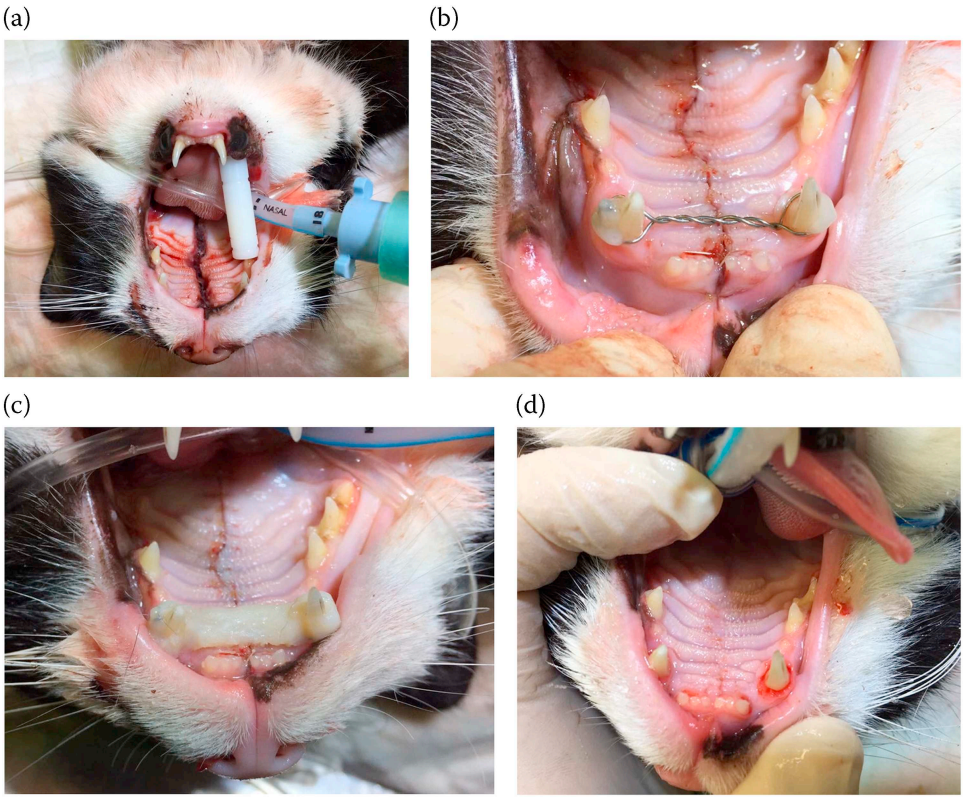


Figure 12.20 Palatine fracture repair. **(a)** Median palatine separation. **(b)** The same cat showing the first stages of repair following suturing of the soft tissues and interdental wiring of the superior canine teeth. **(c)** Repair completed with application of dental acrylic. (**d**) The same cat immediately after removal of the interdental splint. (Courtesy of Matthew Oxford).

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Zygomatic Arch Fractures

Fractures of the zygomatic arch cause pain and swelling of the periorbital region. In most cases, zygomatic arch fractures are managed conservatively, using cold compresses to reduce swelling. The facial nerve and maxillary artery are in close proximity to the caudal aspect of the zygomatic arch and surgical approaches to this region are usually best avoided.

If the cat is unable to blink properly, topical eye ointment must be applied to protect the cornea until periorbital swelling subsides. Medially displaced fragments can displace the globe resulting in exophthalmos and interfere with movement of the mandible. They should be reduced and stabilized using interfragmentary wires or, if comminution is severe, they can be removed. The arch is exposed via a lateral skin incision and elevation of the platysma muscle, avoiding the palpebral nerve and facial vessels.

FRACTURES AND LUXATIONS OF THE TEMPOROMANDIBULAR JOINT:

Surgical Anatomy

(Arredondo et al. 2012, Caporn 1995a).

The temporomandibular joints (TMJs) are complex bicondylar synovial joints that are oriented in a slightly ventrolateral to dorsomedial direction. The joint contains a thin fibrocartilaginous meniscus that has a 360° attachment to the joint capsule dividing the joint into completely separate dorsal (maxillary) and ventral (mandibular) compartments. Each TMJ is formed between the transversely elongated condylar process of the ramus of the mandible and the mandibular fossa of the zygomatic process of the squamous temporal bone. Rostrally bony support for the mandibular condyle within the mandibular fossa is provided by a tubercle that is more prominent in cats than dogs and is analogous to the articular eminence in humans. Caudally the retroarticular process, a ventral extension of the squamous temporal bone, forms part of the caudal mandibular fossa and helps prevents caudal condylar movement. The joint is stabilized by the joint capsule and is reinforced by lateral and caudal thickenings of the joint capsule.

As with other obligate carnivores jaw motion in the cat is almost exclusively restricted to flexion and extension and there is very little translational movement laterally and rostrally. The restriction to hinge-type movement in the cat is the result of the deeply recessed mandibular fossa, the conical shape of the condyloid process and the prominent retroarticular process and articular eminence. Although movement of the two mandibles occurs in unison, the synchondrosis at the symphysis, allows a small amount of independent movement that can permit luxation of the temporomandibular joint to occur without fracture. The temporal, masseter and medial pterygoid muscles are responsible for closing the jaw and the relatively weak digastricus muscle opens the jaw with the aid of gravity. There are a number of important structures in close proximity to the TMJ including the maxillary artery, the mandibular nerve and the middle ear. Unlike the dog, the maxillary arteries in the cat supply oxygenated blood to all of the brain except for the caudal part of the medulla.

Temporomandibular Joint Fracture

With the increased availability of computed tomography condyloid or subcondyloid fractures of the mandible are being recognized with increasing frequency (Figure 12.21).

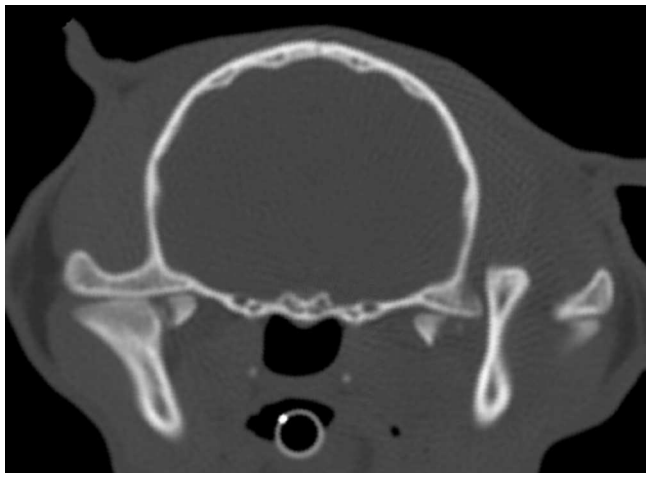


Figure 12.21 Temporomandibular joint fracture. Transverse CT at the level of the temporomandibular joints showing a comminuted fracture of the left condyloid process and the squamous part of the left temporal bone, and fracture of the right medial condyloid process.

In a CT study of 75 cats with head trauma TMJ fracture or fracture/luxation occurred in 43% of cats, the majority of which were unilateral. The principles of articular fracture repair dictate rigid internal fixation with accurate anatomic realignment, and an early return to normal joint function. However, fracture reconstruction is generally not feasible owing to the small size of the fragments and the inaccessibility of the joint. Conservative management is often sufficient in stable fractures with minimal displacement and normal occlusion. The cat is fed soft food for 3–4 weeks to allow healing. Unstable fractures or fracture luxations where there is malocclusion are treated with a tape muzzle or interarcuate fixation. Immobilization for 3–4 weeks is usually sufficient. It is prudent to warn the client that there is a risk of subsequent TMJ ankylosis in cats with damage to the articular cartilage and intra-articular bone fragments. If there is severe comminution or displacement of fragments or if adequate function does not return, condylectomy is performed. The condyle is removed using a bone bur, rongeurs or an osteotome at the base of the neck of the condyloid process to allow development of a functional pseudoarthrosis and prevent contact between the mandible and the temporal bone. The meniscus, if undamaged, is left in situ as it may help to prevent fibrous adhesions and preserve range of motion. The prognosis following condylectomy is discussed in Chapter 13.

Temporomandibular Joint Luxation

Cause and pathogenesis

(Baines & Langley-Hobbs 2001, Caporn 1995b, Klima 2007, Somrak & Maretta 2015, Thatcher 2017).

The TMJ is inherently stable, rostral condyloid displacement is prevented by a well-developed bony process analogous to the articular eminence and caudal displacement is prevented by the prominent retroarticular process. Luxation is presumed to occur when there is traumatic overextension of the TMJ. In most cases there is unilateral luxation of the mandibular condyloid process from the mandibular fossa in a rostrodorsal direction. TMJ luxation occurs more frequently in the cat than the dog, possibly because there is much less latero-lateral

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movement in the normal feline joint. Luxation commonly accompanies other maxillofacial injuries, bilateral luxation is occasionally seen in which case both mandibles are usually displaced rostrally.

Clinical Signs

Typically an affected cat is presented with an inability to close the mouth as a result of malocclusion associated with mandibular drift, and with the inferior jaw deviated away from the affected TMJ. Unilateral, caudoventral luxation is occasionally encountered, in which case the jaw will be deviated toward the side of the lesion. Luxations accompanied by other mandibular or maxillary fractures or symphyseal separation are more difficult to detect but should be suspected in any patient with head trauma where there is significant unexplained malocclusion.

Diagnosis

Intraoral examination may reveal swelling and contusion in the region of the TMJ and latero-lateral laxity can be palpated in most cases. Radiography (lateral, ventrodorsal, and oblique views) or CT is used to confirm the luxation and evaluate for concurrent fractures.

Right and left lateral oblique projections with the cat's head rotated dorsally by 20° will usually permit evaluation of both TMJs. To obtain these views the head is rotated along its median axis so that the TMJ nearest the film will be projected clear of the skull ventrally. The radiographs should be checked carefully for periarticular fracture since this will complicate treatment.

Treatment and Prognosis

Closed reduction of TMJ luxations under general anaesthesia is possible in most cases.

A tuberculin syringe or small diameter wooden dowel is placed transversely across the jaw as far caudally as possible (typically between the carnassial teeth) to act as a fulcrum (Figure 12.22). The rostral mandibles and maxillae are then squeezed together, partially closing the jaw and levering the caudal mandible against the dowel to disengage the luxated condyle. While maintaining pressure on the rostral mandibles, the mandible on the affected side is pushed in the appropriate direction to reduce the luxation. For caudoventral luxations, the rostral mandible is pushed away from the side of the luxation; for the more common rostrodorsal luxations, the rostral mandible is pushed toward the side of the luxation. Once the luxation is reduced, pressure on the mandibles is released. Reduction is confirmed by proper occlusion of the superior and inferior canine teeth and by radiography.

In an alternative reduction method, the head is grasped and stabilized with the non-dominant hand. The thumb of the dominant hand is then placed in the oral cavity and the ipsilateral mandibular body is grasped between the thumb and the first 2 fingers. For rostrodorsal luxations traction is applied to the mandible in a rostral direction to disengage the condyloid process from the bony eminence and manipulate it ventrally; the mandible is then guided caudally to allow the condyloid process to be pulled back into the mandibular fossa by the surrounding soft tissues. Manipulating the mandible with the mouth closed as much as possible will ease reduction.

In most cases, the TMJ will be stable after closed reduction. If there is instability, as indicated by mandibular drift, or if luxation recurs on manipulation of the jaw, additional support is provided by the application of a tape muzzle for 2

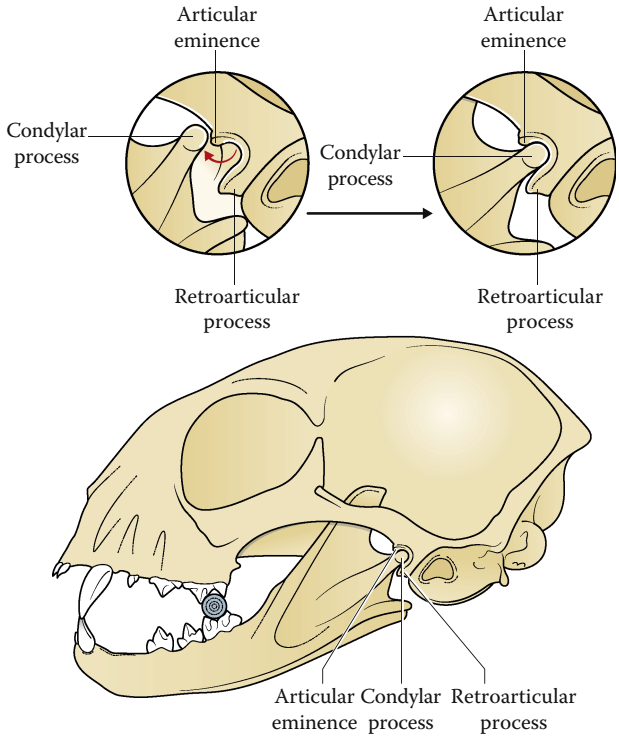


Figure 12.22 Technique for reduction of a rostrodorsal temporomandibular joint dislocation using a dowel placed between the carnassial teeth.

weeks or an interarcuate fixation technique is used. If the TMJ luxation is chronic, and closed reduction is not possible, open reduction and imbrication of the joint capsule or condylectomy is indicated; this is achieved through a lateral approach. A condylar tethering technique using polyester suture has been described and was successfully applied to a cat with recurrent luxation and fracture of the articular eminence.

The prognosis after uncomplicated TMJ luxation is good, although the development of TMJ post-traumatic osteoarthritis is likely. True and false TMJ ankylosis are recognized complications of TMJ luxation or fracture/luxation occurring in a small proportion of cats and are discussed in Chapter 13. Post-reduction physiotherapy may help to reduce the incidence of ankylosis after reduction of luxation. A case of transient iatrogenic Horner syndrome has been reported following reduction of bilateral temporomandibular luxation. The presumptive cause was trauma to the periorbita and postganglionic fibres by the coronoid process of the ramus.

FRACTURES OF THE CALVARIUM:

(Garosi & Adamantos 2011, Knight & Meeson 2019).

Fractures of the bones surrounding the brain (the neurocranium) represented 4.4% of craniomaxillofacial fractures in one CT study of 75 cats. Fractures of the base of the neurocranium are rare, most fractures involve the calvarium (the skullcap), which is the more exposed dorsal portion (Figure 12.23). The rarity of fractures may be partly explained by the fact that most cats are killed outright or die soon after severe head injury. It has also been postulated that the bones of the mid-face may act as an energy-absorbing region or "crumple zone" in cats, thereby reducing the

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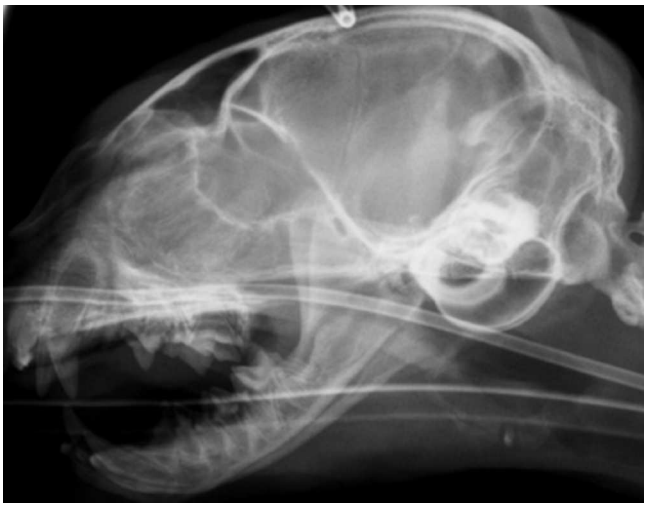


Figure 12.23 Lateral radiograph of the skull showing a fissure fracture of the calvarium.

impact on the intracranial contents, and protecting the neurocranium from fracture.

All cats with traumatic brain injury constitute a medical emergency, and surgical intervention may be indicated in a small proportion of cases. Mortality, even in treated cases, is high, with death typically resulting from a progressive increase in intracranial pressure. The goals of therapy are to alleviate brain swelling and prevent damage to vital brainstem structures. The most important consideration for cats with brain injury is maintenance of cerebral perfusion and oxygenation. Early surgical intervention to provide decompression may be indicated for treatment of fractures of the skull when they are open or depressed sufficiently to impinge on the underlying neurological structures. Cats have a remarkable ability to compensate for loss of cerebral tissue, and it is important not to make hasty decisions based on the initial appearance of a patient with brain trauma.

The initial approach to management is the same for any acutely traumatized cat and is directed at concurrent life-threatening injuries. Head injury should be graded as mild, moderate, or severe based on the level of consciousness. Hypoxia is addressed by maintaining a patent airway, and placing the patient in an oxygen-enriched environment. Despite concerns about exacerbation of cerebral oedema, hypovolaemia should be corrected by administration of intravenous fluids. In addition, specific medical therapy may be initiated, although there is considerable controversy surrounding the use of drugs for head injury. Mannitol is an osmotic diuretic that has been shown to reduce brain oedema and intracranial pressure. Theoretical concerns about exacerbation of intracranial haemorrhage by the osmotic action of mannitol are clinically unfounded and should not prevent its use. However, mannitol should be avoided if the patient is hypovolaemic. Once the patient is haemodynamically stable, mannitol can be administered for suspected increased intracranial pressure as a slow intravenous infusion over 10–20 minutes at a dose of 0.25–1.0 g/kg. Mannitol has a rapid onset of action, which persists for 2–5 hours. The drug may be repeated if necessary to a maximum of three boluses in a 24-hour period. Intravenous hypertonic saline (7%) should be used instead of mannitol in the face of hypovolaemia at a dose of 3–5 ml/kg over 15 minutes.

The use of glucocorticoids is contraindicated for head trauma patients because they exacerbate hyperglycaemia. Hyperglycaemia can potentiate neurological injury in patients with head injury, so iatrogenic hyperglycaemia should be avoided. High-dose methylprednisolone sodium succinate therapy may provide beneficial therapeutic effects solely because of its free-radical scavenging activity. To be effective as an adjunctive treatment methylprednisolone sodium succinate (MPSS) must be given within 8 hours of injury. Because of concerns about induction of hyperglycaemia, MPSS use should probably be restricted to nonhyperglycaemic cats that are not responding to fluid and oxygen therapy and mannitol administration.

Advanced imaging is recommended in any cat with moderate to severe neurological dysfunction that is either not showing a rapid response to appropriate medical therapy or is deteriorating despite medical therapy. Radiographs of the skull may be useful to identify fractures of the calvarium but otherwise are of limited value. CT is generally preferred to MRI as it is much quicker to perform and shows better resolution of acute haemorrhage and greater bone detail. The results of CT help to determine whether surgery is indicated for depressed skull fracture and/or haemorrhage.

Prediction of outcome is based on an assessment of the level of consciousness and brainstem reflexes and the results of serial neurologic examinations following treatment. Loss of consciousness, dilation of one or both pupils, and motor dysfunction, such as paresis or decerebrate rigidity, are bad prognostic indicators. Deterioration in the neurologic status despite medical therapy indicates progressive brain oedema and/or haemorrhage and, under these circumstances, surgical decompression should be considered. Decompressive craniectomy should be performed within 12 hours in cats with severe head injury that are either refractory to aggressive medical treatment or have severely depressed or open calvarial fractures or an extra-axial haematoma. The value of craniotomy and durotomy solely as decompressive procedures in cats with brain injury is unknown. For further details concerning the medical and neurosurgical management of head injury standard neurology texts should be consulted.

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13 The Skull and Mandible – Other Disorders

INTRODUCTION:

(Arzi et al. 2013, Beam et al. 2007, Schwarz et al. 2002).

Orthopaedic disorders of the skull and mandible other than fractures and dislocations mainly comprise conditions affecting the temporomandibular joints (TMJs) and are uncommon in the cat. Lack of familiarity with these conditions and difficulties with radiographic imaging of the bony anatomy of the head both present diagnostic challenges for the clinician. In the past reliance was made on radiography however, in recent years computed tomography (CT) has been used more widely to assess disorders in this region of the body. Although CT is able to furnish the clinician with detailed images in multiple planes their interpretation requires a greater knowledge of conventional and sectional anatomy. A recent study of the use of CT in the investigation of TMJ disorders at a dentistry and oral surgery referral practice included 17 cats. Nine of the cats had fractures, eight had osteoarthritis, four had luxation, two had ankylosis and one had neoplasia. The presence of two or more concurrent TMJ disorders was not unusual. Neoplasia of the skull and mandible is discussed in Chapter 19.

TEMPOROMANDIBULAR JOINT ANKYLOSIS:

(Cetinkaya 2012, Gemmill 2008, Lantz 1985, Larguier & Jamet 2015, Maas & Theyse 2007, Meomartino et al. 1999, Miller et al. 1975, Sullivan 1989, van Ee & Pechman 1987).

Cause and Pathogenesis

TMJ ankylosis can result from trauma, developmental abnormality, infection, or neoplasia. The majority of reported cases of ankylosis have occurred as a sequela to TMJ trauma. It is prudent to warn clients of all cats suffering craniofacial trauma of the risk of subsequent TMJ ankylosis. The ankylosis may be intraarticular (true ankylosis) or extraarticular (false ankylosis) but the presentation is the same. In kittens ankylosis of the TMJ can lead to malocclusion and craniofacial malformation. Ankylosis secondary to trauma typically occurs in young cats whereas neoplasia-related ankylosis occurs in older cats. Post-traumatic ankylosis may be unilateral or bilateral.

Clinical Signs

The primary clinical sign is the inability to open the mouth completely, leading to difficulty eating, weight loss, atrophy of the masticatory muscles and an unkempt staring coat caused by a lack of grooming. Typically there is a progressive reduction in the range of jaw motion, usually over a period of many months, which may pass unnoticed by the owner. If ankylosis occurs in an immature cat, altered development of the mandible may occur, causing facial asymmetry, mandibular brachygnathism or other abnormalities in facial appearance. It should be noted that the term *trismus* should only be used for the uncontrolled contraction of the masticatory muscles that occurs in tetanus and it is not the correct term where there is mechanical resistance to opening of the jaw such as TMJ ankylosis.

Diagnosis

Radiographic examination has limited diagnostic value in the investigation of complex disorders of the TMJ. Advanced imaging should be used when it is available. CT is the procedure of choice for bony lesions and it also allows three-dimensional reconstruction which is of great value for diagnosis and for surgical planning (Figure 13.1). Magnetic resonance imaging (MRI) will show the presence and extent of fibrous tissue proliferation in cats with false ankylosis. If advanced imaging is not available radiographic examination should be performed using a dorsoventral view of the skull and lateral oblique views of both TMJs. Radiographic evaluation of the feline TMJ for ankylosis is difficult and the changes may be missed or their extent may be underestimated. Expected radiographic changes include loss of joint space and periarticular new bone formation, and there may be evidence of previous trauma. When a tumour is suspected as the cause a biopsy should be taken to differentiate between periarticular new bone and fibrous tissue, and benign or malignant neoplasia.

Treatment and Prognosis

Treatment for TMJ ankylosis is by surgical resection of the ankylosing tissue in false ankylosis, and excision arthroplasty in true ankylosis, to allow a more normal range of jaw motion (Figure 13.2 and Figure 13.3). It may be necessary to excise the condyloid process, a portion of the zygomatic arch, periarticular osteophytes and in some cases part of the coronoid process. Treatment by stretching the jaws open under general anaesthesia (so-called *brisement forcée*) is contraindicated because it carries the risk of jaw fracture and is associated with a high frequency of recurrence. Parenteral or periarticular administration of glucocorticoids fails to prevent recurrence of ankylosis after this manoeuvre.

In some cases with a limited degree of jaw opening laryngeal intubation may be possible using a fibreoptic scope. In others, endotracheal intubation may not be possible and provision should be made for the maintenance of anaesthesia by tracheotomy. If it has not been possible to deter-

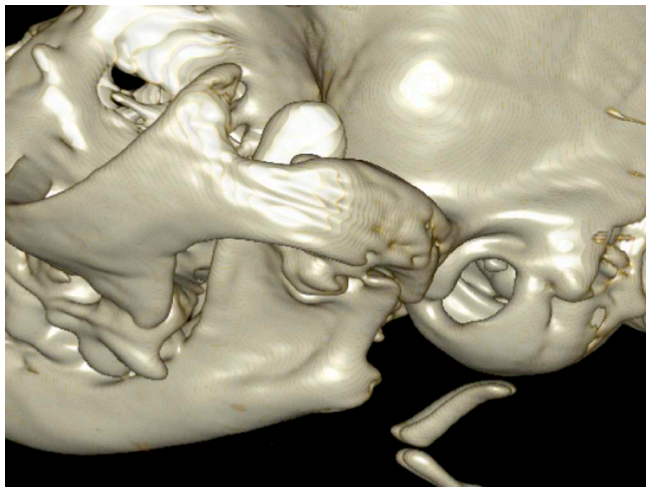


Figure 13.1 Preoperative CT 3D volume rendering showing false ankylosis of the left temporomandibular joint (Courtesy of Steven Baird).

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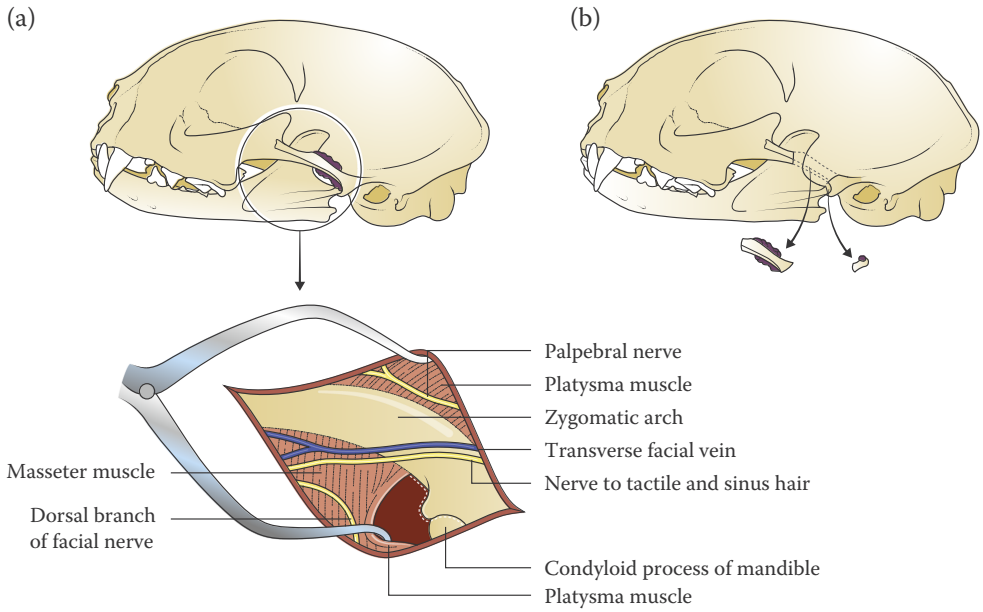


Figure 13.2 Surgery for temporomandibular (TMJ) ankylosis. **(a)** TMJ ankylosis showing the surgical approach (inset). **(b)** Treatment by excision of the condyloid process and a portion of the zygomatic arch.

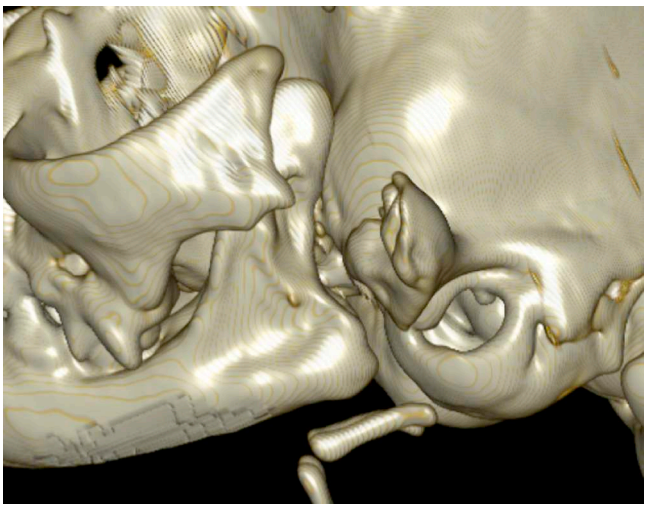


Figure 13.3 Postoperative CT 3D volume rendering of cat in Figure 13.1 following removal of the condyloid process and a portion of the zygomatic arch.

mine the affected side or whether the condition is unilateral or bilateral before surgery it may be necessary to separate the mandibular symphysis to allow independent manipulation of the mandibles. If ankylosis is bilateral, surgery is performed on both joints simultaneously.

Excision arthroplasty for cats with true ankylosis is performed as follows:

> With the patient in lateral recumbency, a curved skin incision is made along the ventral border of the zygomatic arch, crossing the TMJ caudally. The origin of the masseter muscle on the zygomatic arch is incised and subperiosteal reflection of the muscle is performed taking care to avoid the dorsal branch of the facial nerve.
>
> In most cases there is extensive bone formation around the caudal portion of the zygomatic arch, necessitating removal of its caudal portion and adjacent periarticular new bone to gain access to the TMJ. This exposes the lateral surface of the joint and the dorsal portion of the condyloid process. Proceeding in a ventromedial direction, further proliferative bone and fibrous tissue, the mandibular condyle, and as much of the dorsal portion of the ramus as necessary are excised in order to re-establish jaw movement.

After excision arthroplasty, a pseudoarthrosis develops as the area is filled with fibrous connective tissue. Potential complications include re-ankylosis, facial nerve paralysis and malocclusion. The functional outcome after unilateral condylectomy is usually satisfactory despite the fact that the loss of one TMJ has been shown to lead to greater instability in cats than in dogs. In cats a degree of malocclusion is to be expected, as a result of displacement of the ipsilateral mandible, causing the jaw to deviate to the operated side. Sequelae of malocclusion include interference with mastication, pain on mastication and secondary osteoarthritis of the unaffected TMJ. The effects of unilateral condylectomy on dental occlusion were reported in 12 feline cadavers and it was found that there was a significant increase in latero-lateral amplitude of jaw movement which might clinically affect dental occlusion. This is in contrast to similar studies in dogs in which mandibular condylectomy appears to be a more benign procedure. The difference may be attributed to the anatomical differences in the region between the two species most notably the deeper mandibular fossa in the cat. In those cases with restriction of jaw opening because of neoplasia resection of the mass may be indicated. In one report of five cats with TMJ ankylosis two of the cats had tumour-related ankylosis. Histopathological examination revealed benign bone proliferation consistent with osteoma. Resection of the osteoma in both cats carried a favourable prognosis for return of function with no long-term recurrence.

Postoperatively a soft diet is fed for 4 weeks and physical therapy is commenced after the first week to help improve range of motion and reduce the likelihood of recurrence of the ankylosis. Physical therapy for the jaw may be achieved by feeding frequent small meals and gently opening and closing the mouth several times a day.

INTERMITTENT OPEN MOUTH LOCKING:

(Gatineau et al. 2008, Hsuan et al. 2017, Kraus & Tomlinson 1988, Lantz 1987, Nutt et al. 2018, Oakes et al. 1990, Soukup et al. 2009).

Cause and Pathogenesis

This rare disorder is characterized by an inability to close the mouth. It is well recognized in certain breeds of dog and there is an increasing number of case reports of the condition in cats. Cats of all ages and skull types may be affected but brachycephalic breeds are over-represented. The condition may occur unilaterally or bilaterally.

Open mouth jaw locking in dogs is usually associated with TMJ dysplasia and joint laxity, which allows the coronoid process of the mandible to contact the ventral or lateral aspect of the ipsilateral zygomatic arch, preventing closure of the mouth. Mandibular symphyseal laxity may also contribute to excess lateral movement of the mandibles and the resulting impingement. Proposed aetiologies in the cat include TMJ dysplasia, symphyseal laxity, post-traumatic and abnormalities of skull conformation. In one reported case a deformity of the zygomatic arch that was either developmental or post-traumatic was presumed to be the cause of the impingement. TMJ dysplasia is difficult to confirm radiographically as the underlying cause but has been diagnosed definitively in two recent cases using CT.

Clinical Signs

All reported cases have had a history of repetitive open mouth jaw locking, especially after yawning. Clinical signs when the jaw is locked may include salivation, pawing at the mouth and apparent distress and discomfort. Palpation in the locked state reveals a unilateral protuberance (the coronoid process) along the ventrolateral aspect of the zygomatic arch on the affected side.

Diagnosis

Diagnosis is based on physical examination and radiographic findings, including displacement of the mandible, impingement of the coronoid process on the zygomatic arch, and, in some cases, bony dysplasia of the TMJ. A CT scan with the jaw locked in position is recommended both for diagnosis and for surgical planning. Using CT an accurate assessment of the degree of overlap of the coronoid process and the zygomatic arch can be made and the skull can be evaluated for other abnormalities such as fractures or TMJ luxation.

Treatment and Prognosis

If the cat is presented with the jaw locked, closure of the mouth is achieved by opening the mouth a little further to release the coronoid process from the ventrolateral aspect of the zygomatic arch and then pressing the protruding coronoid process medially. Application of a tape muzzle may be a temporary solution although this is poorly tolerated by many cats. Definitive treatment is by partial zygomatic arch ostectomy and/or partial coronoidectomy. Partial zygomectomy is performed by removal of an approximately 10 mm full-thickness section of the mid-portion of the zygomatic arch. Partial coronoidectomy involves removal of only the locking part of the coronoid process. In severe cases, it may be necessary to perform both procedures.

For partial zygomectomy the site of impingement is identified on pre-operative CT or by recreating jaw locking intraoperatively. An approach is made over the zygomatic arch at this point, using blunt dissection and periosteal elevation to expose the bone. A high-speed bur or rongeurs are used to remove a section of the arch. The mouth is opened again to confirm that it is not possible to induce locking. If locking still occurs approximately 5 mm of bone can be removed from the dorsal border of the coronoid process. Bilateral partial zygomectomy and bilateral partial coronoidectomy have been reported recently in a cat with bilateral open mouth jaw locking with a successful outcome.

TEMPOROMANDIBULAR OSTEOARTHRITIS:

(Arzi et al. 2013, Lorenz et al. 2012, Schwarz et al. 2002).

The prevalence of osteoarthritis (OA) of the TMJ in cats is unknown as the condition is often asymptomatic. Osteoarthritis may develop as a sequela to TMJ fracture and/or luxation, TMJ dysplasia and contralateral mandibulectomy. Severe OA may be associated with open-mouth jaw locking, and periarticular new bone is the main radiographic feature of true ankylosis. A CT study that included 42 cats that were presented to a dentistry and oral surgery referral service found that OA was the second most common TMJ disorder after fracture and frequently occurred in combination with other disorders. In this study, OA was found in 8 of the 17 cats with TMJ disorders and was always bilateral. Only half of the cats, in which OA was the only disorder detected, had a decreased range of motion of the jaw. Osteoarthritis was seen more frequently in cats with a mesaticephalic skull conformation than in cats with either a dolicocephalic or brachycephalic skull conformation. In contrast to OA of the appendicular joints no association was found between the presence and severity of OA and age, sex or body weight.

Cats with TMJ OA may present with signs of pain and difficulty opening and closing the jaw. Treatment involves feeding a soft diet and pain management. Gabapentin has been reported as a treatment for chronic orofacial pain in cats and may be a useful adjunct or alternative to NSAIDs in cats with TMJ OA. If medical treatment is not effective excision arthroplasty should be considered.

TEMPOROMANDIBULAR JOINT INFECTION:

Septic arthritis and osteomyelitis are rare disorders of the TMJ that are more common in cats than dogs. Infection is usually an extension of osteomyelitis from the ipsilateral tympanic bulla which anatomically is very close to the TMJ in the cat. Septic arthritis may also occur as a result of a local bite wound from another cat or a dog. Clinical signs include pain on opening and closing the mouth and periarticular soft tissue swelling.

Radiography shows mixed osteoproductive and osteolytic changes although the appearance may not differ significantly from those seen with OA. Unilateral changes and associated soft tissue swelling would raise the index of suspicion for infection rather than OA. Computed tomography, if available, will help in the differentiation between OA, septic arthritis and false anykylosis. If sepsis is suspected

synoviocentesis should be performed and samples should be submitted for cytology and bacteriology.

Septic arthritis should be treated by appropriate drainage, analgesia and a prolonged course of antibiotics based on culture and sensitivity testing. If changes are severe excision arthroplasty may be indicated.

TEMPOROMANDIBULAR JOINT NEOPLASIA:

(Arzi et al. 2013, Fiani et al. 2011, Hodik et al. 2012, Maas & Theyse 2007, Schwarz et al. 2002).

Neoplasia of the feline musculoskeletal system is discussed in Chapter 19. For information about neoplasia of the mandible, maxillofacial region and skull the reader should consult oncology and soft tissue surgery texts. Neoplasia affecting the TMJ is mentioned here because of its importance as a differential diagnosis in cats presenting with signs of pain and difficulty in opening and closing the mouth.

Neoplasia directly involving or impinging on the TMJ is rare in the cat. Initial clinical signs include discomfort on opening and closing the mouth which progresses to TMJ deformity and ankylosis. In a CT study that included 17 cats with TMJ disorders only one cat had a tumour. Tumour types in cats include osteosarcoma, multilobular tumour of bone, osteoma and chondroma. The typical radiographic appearance of malignant tumours is that they are poorly demarcated and are characterised by extensive lysis of bone and marked irregular periosteal reaction. In contrast benign tumours of bone, such as osteomas, appear as a well circumscribed very radiodense mass on the periosteal surface of otherwise normal bone. The prognosis for malignant neoplasia is guarded but surgical excision of osteomas can be curative.

MAXILLOAFACIAL OSTEONECROSIS:

(Larson et al. 2019).

Osteoradionecrosis is an uncommon condition in which bone is devitalized and becomes susceptible to pathologic fracture following cancericidal doses of radiation therapy. It can occur in any bone, typically years after treatment, but the mandible is most susceptible. The risk can be minimized by administering lower doses per fraction.

Medication-related osteonecrosis of the jaw (MRONJ) is a well-known adverse effect of bisphosphonate treatment in humans and has been reported in a single cat after long-term administration of alendronate. Bisphosphonates are inhibitors of osteoclastic bone resorption that have been used primarily to decrease the concentration of circulating ionized calcium in cats with idiopathic hypercalcaemia and occasionally for cats with granulomatous disease or malignancy-associated hypercalcaemia.

Treatment of osteonecrosis involves surgical debridement, long-term antibiotic therapy and analgesics. In severe cases, partial mandibulectomy or maxillectomy may be performed to remove all affected bone.

FELINE OROFACIAL PAIN SYNDROME:

(Rusbridge et al. 2010).

Feline orofacial pain syndrome (FOPS) is a suspected neuropathic pain disorder characterized by episodic signs of oral discomfort and self-mutilation. Discomfort seems to be confined to one side of the buccal cavity and lips and may be analogous to trigeminal neuralgia and/or glossodynia in humans. Affected cats are usually presented because of exaggerated chewing movements and pawing at the mouth. The predominance in the Burmese cat breed is consistent with an inherited disorder. The importance of the condition is as a differential diagnosis for cats with signs of orofacial pain.

MASTICATORY MYOSITIS:

(Blazejewski & Shelton 2018).

Masticatory myositis is well reported in dogs but has been considered either not to occur or to be extremely rare in cats. The condition has been reported recently in a cat that was presented with a 2-month history of inability to fully open the mouth when yawning, and difficulty prehending hard food. A canine ELISA demonstrated the presence of a positive serum antibody titre against masticatory muscle type 2M fibres, and subsequent histopathology confirmed end-stage masticatory myositis. Treatment with glucocorticoids was not successful. The condition may be underdiagnosed in cats and masticatory muscle myositis should be included in the differential diagnosis of cats with reduced jaw mobility, orofacial pain and masticatory muscle atrophy.

TRIGEMINAL NEURITIS:

Acute idiopathic bilateral inflammation of all motor branches of the trigeminal nerve is well documented in dogs but is very rare in cats. Affected cats present with a dropped jaw and have difficulty in prehending food or drinking water. Diagnosis is based on the history and clinical signs and the absence of any other demonstrable cause. Treatment is supportive including the use of a feeding tube if necessary in severely affected cases. Spontaneous improvement usually occurs in 2–3 weeks.

PATELLAR FRACTURE AND DENTAL ANOMALY SYNDROME:

(Howes et al. 2019).

Mandibular and maxillary abnormalities including multiple persistent deciduous teeth, gingivitis and osteomyelitis of the mandible may occur in a small proportion of cats affected by patellar fracture and dental anomaly syndrome (PADS) (see Chapter 18). Radiologically the jaw lesions have an aggressive appearance that may mimic neoplasia. Extraction of retained deciduous teeth and unerupted permanent teeth and debridement of proliferative and necrotic bone is indicated alongside prolonged antibiotic therapy guided by the results of culture and sensitivity testing.

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14 The Spine – Fractures and Dislocations

INTRODUCTION:

(Bali et al. 2009, Grasmueck & Steffen 2004, Hernon et al. 2018, Marioni-Henry et al. 2004, Marioni-Henry 2010, Zulauf et al. 2008a).

Traumatic injury is a common cause of spinal disease in cats. In a post mortem study of a referral population, 7% of cats with spinal disorders were found to have a vertebral fracture and/or luxation. The true figure is likely to be higher as many cats are not referred for acute spinal injury because of a perceived poor outcome or because of financial constraints. Unfortunately because of the extreme forces involved many traumatic spinal injuries in the cat result in severe spinal cord injury or complete spinal cord severance.

In comparison with dogs, there is a greater predisposition in young animals less than 12 months of age. Concomitant injuries are more common in cats than in larger breeds of dog which is probably a reflection of the protective effect of larger body size in dogs. Between 30–83% of cats with vertebral fracture/luxations have concurrent injuries some of which may be life-threatening. Cats with polytraumatic injuries are at a greater risk of mortality than cats with single injuries. In a study of 100 feline trauma patients, whole-body radiographs demonstrated spinal injury in 26% of cases. Cats with spinal injury were euthanased more frequently than cats with injury to any other body region. Road accidents are the most common cause of injury and also carry the highest risk of mortality compared with other traumatic events. In a study of 30 cats with traumatic injury to the thoracic and lumbar spine, the overall survival rate was 50%. In this study, none of the cats with paraplegia and absent deep pain perception recovered function.

In cats with thoracolumbar fractures and luxations there is a tendency for the caudal segment to displace dorsally whereas dogs most commonly show ventral displacement of the caudal segment (Figure 14.1). The difference may be explained in part by the more robust articular processes in larger dogs that are less prone to fracture and if intact will prevent dorsal displacement. The Schiff-Sherrington phenomenon of forelimb hypertonicity that is commonly seen in dogs associated with severe acute thoracolumbar spinal cord injury is rarely seen in the cat.

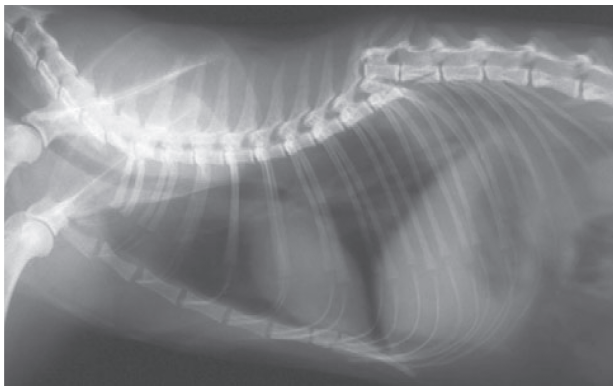


Figure 14.1 Mid-thoracic spinal fracture/luxation. Lateral radiograph of a fracture/luxation at T9–T10. This degree of displacement is inevitably associated with spinal cord transection. The cat had no deep pain perception caudal to the lesion. (Courtesy of Malcolm McKee.)

SURGICAL ANATOMY:

Vertebrae

The vertebral column is composed of a series of 48–50 vertebrae, most of which are joined by intervertebral discs, ligaments and the synovial joints between the articular processes. The vertebral formula for the cat is C7, T13, L7, S3, Cd 18–20. The basic structure of each vertebra is similar, although the morphology varies from one region of the spine to another. Each vertebra has a vertebral arch comprising two pedicles and a dorsal lamina. The vertebral canal is formed from the vertebral arch laterally and dorsally and the vertebral body ventrally. Most vertebrae have transverse processes projecting laterally from the vertebral body, a spinous process projecting dorsally from the lamina, and cranial and caudal articular processes on the vertebral arch. Other bony processes vary according to region. The synovial articulations lie dorsally except for those between the first and second cervical vertebrae (C1–C2), where they are ventral, and the sacrum, where they are fused. These are standard diarthrodial joints with articular cartilage, a joint capsule and synovial fluid. In the cervical spine, the articulations between cranial and caudal articular processes of adjacent vertebrae are close to the dorsal plane whereas in the lumbar spine the articulations are in the sagittal plane. Paired spinal nerves exit the vertebral canal between vertebrae through their respective intervertebral foramina, with the exception of the first cervical spinal nerves, which exit through the lateral vertebral foramina of the atlas.

The first two cervical vertebrae, the atlas and axis, have a distinct morphology that reflects their more specialized function. There is no intervertebral disc between the atlas and axis. With the exception of C7, the vertebral arteries and veins traverse the transverse foramina in the transverse processes of the cervical vertebrae. The transverse processes of the caudal cervical vertebrae are relatively smaller than those of the dog. The C6 transverse processes are larger than the other cervical vertebrae providing a useful surgical landmark when performing a ventral surgical approach, however, they are angled more laterally compared with those of the dog in which ventral projection is more pronounced. The axis has a very large dorsal spinous process that extends over the atlas and is connected to it by the atlantoaxial ligament. The dens arises embryologically from the atlas and has a separate growth plate. It projects cranially from the body of the axis and lies on the floor of the vertebral canal. The atlantooccipital joint allows for flexion and extension in the sagittal plane and is therefore known as the “yes joint” whereas the atlantoaxial joint allows for rotation in the transverse plane and is known as the “no joint”. The cat lacks a nuchal ligament, which in the dog extends from the caudal part of the spinous process of the axis to the tip of the spinous process of the first thoracic vertebra.

Thoracic vertebrae articulate with the ribs and have a small vertebral body and a large dorsal spinous process. Lumbar vertebrae have relatively long vertebral bodies in

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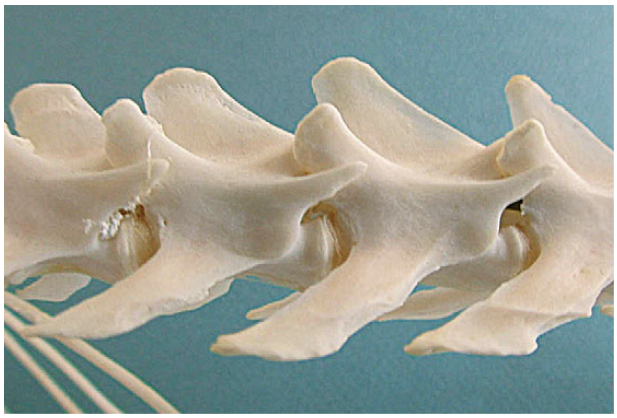


Figure 14.2 Articulated cranial lumbar vertebrae. The vertebral bodies of the lumbar spine are elongated in comparison with those of the dog and the transverse processes are longer.

comparison with the dog with short spinous processes angled cranially. The lumbar transverse processes are also longer than those of the dog and project cranioventrally, overlapping the preceding vertebra (Figure 14.2). The ventral deflection of these processes is more pronounced in the cat than the dog. The transverse processes are short on the first lumbar vertebra (L1) but become progressively narrower and longer on more caudal lumbar vertebrae. The ribs and the transverse processes of L1 at the thoracolumbar junction are important landmarks when performing surgery in this region of the spine. The three sacral vertebrae fuse postnatally to form one body, which articulates with the pelvis at the sacroiliac joints. The original sacral dorsal spinous processes remain individually distinct despite fusion of the bodies and transverse processes. The caudal vertebrae vary in number and gradually lose all processes until elongated vertebral bodies are all that remain towards the tip of the tail.

Spinal Cord

The spinal cord extends from the foramen magnum to L7 in the vertebral canal. The cord is surrounded by the meninges, which comprise three layers. The most superficial and substantial layer is the dura mater. Lying adjacent to the dura is the thin arachnoid mater and internal to this is the pia mater, which is closely apposed to the spinal cord. Cerebrospinal fluid (CSF) is contained intrathecally in the subarachnoid space between the pia and arachnoid maters. This space is traversed by the arachnoid trabeculae, which suspend the spinal cord in the CSF. The termination of the spinal cord is known as the conus medullaris and the meninges caudal to the conus medullaris form the filum terminale, which extends into the sacrocaudal vertebral canal.

The cord is divided into a central core of grey matter, comprising the cell bodies of lower motor neurons, and an external portion of white matter. The white matter is composed of ascending and descending nerve cell bundles that are organized functionally into tracts or fasciculi. The tracts that ascend (afferent) to the brain have a sensory function and those that descend (efferent) from the brain have a motor function. It is worth noting that, although the terms are sometimes used interchangeably, pathways and tracts are not synonymous. A tract is a bundle of axons in the central nervous system, with the same function, that originate together and terminate together, and do not synapse on the way. A neural pathway is a route through the nervous system composed of sequential tracts, separated by synapses, that are all involved in the same neural function.

The spinal cord is segmental (eight cervical, thirteen thoracic, seven lumbar, three sacral, five caudal) with each paired dorsal and ventral nerve root emanating from its respective segment and joining to form the paired segmental spinal nerves, which exit at the corresponding intervertebral foramina. The C1 spinal nerves exit through the lateral foramina in the atlas. The remaining cervical spinal nerves exit through the intervertebral foramina cranial to the vertebra of the same annotation, with the exception of the C8 nerves, which exit between the C7 and T1 vertebrae. Thus the thoracic and lumbar segmental spinal nerves exit through the intervertebral foramina caudal to the vertebra of the same annotation. Spinal cord segments C6–T2 and L4–S3 contain the cell bodies for the lower motor neurons forming the brachial and lumbosacral plexuses innervating the forelimb and hindlimb musculature, respectively. These two regions of the spinal cord are thicker and are known as intumescences. It is essential to understand the relationship between spinal cord segments and vertebrae in order to determine the anatomical location of a lesion; neurological lesion localization refers to spinal cord segments and not vertebrae.

In contrast to the dog, where the lumbosacral spinal cord is considerably foreshortened, the feline conus medullaris terminates in the sacrum in over 90% of adult cats (Figure 14.3). As a consequence of this there is a closer correlation between the vertebrae and their respective spinal cord segments, and a focal compressive lesion will tend to affect fewer spinal cord segments and nerve roots than a lesion in the same location in the dog.

CAUSE AND PATHOGENESIS:

(Eminaga et al. 2011, Olby 1999, Olby, 2010, Vnuk et al. 2016).

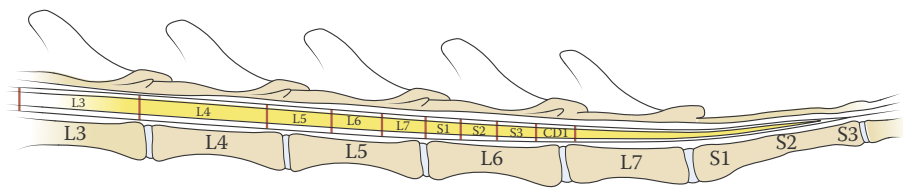


Figure 14.3 Lumbosacral spine showing the relationship between spinal cord segments and vertebrae. Segments L3 and L4 correspond to their respective vertebral bodies. Segment L5 lies dorsal to the L4–L5 intervertebral disc, segments L6 and L7 lie dorsal to the L5 vertebral body, and the three sacral segments lie dorsal to the L6 vertebral body.

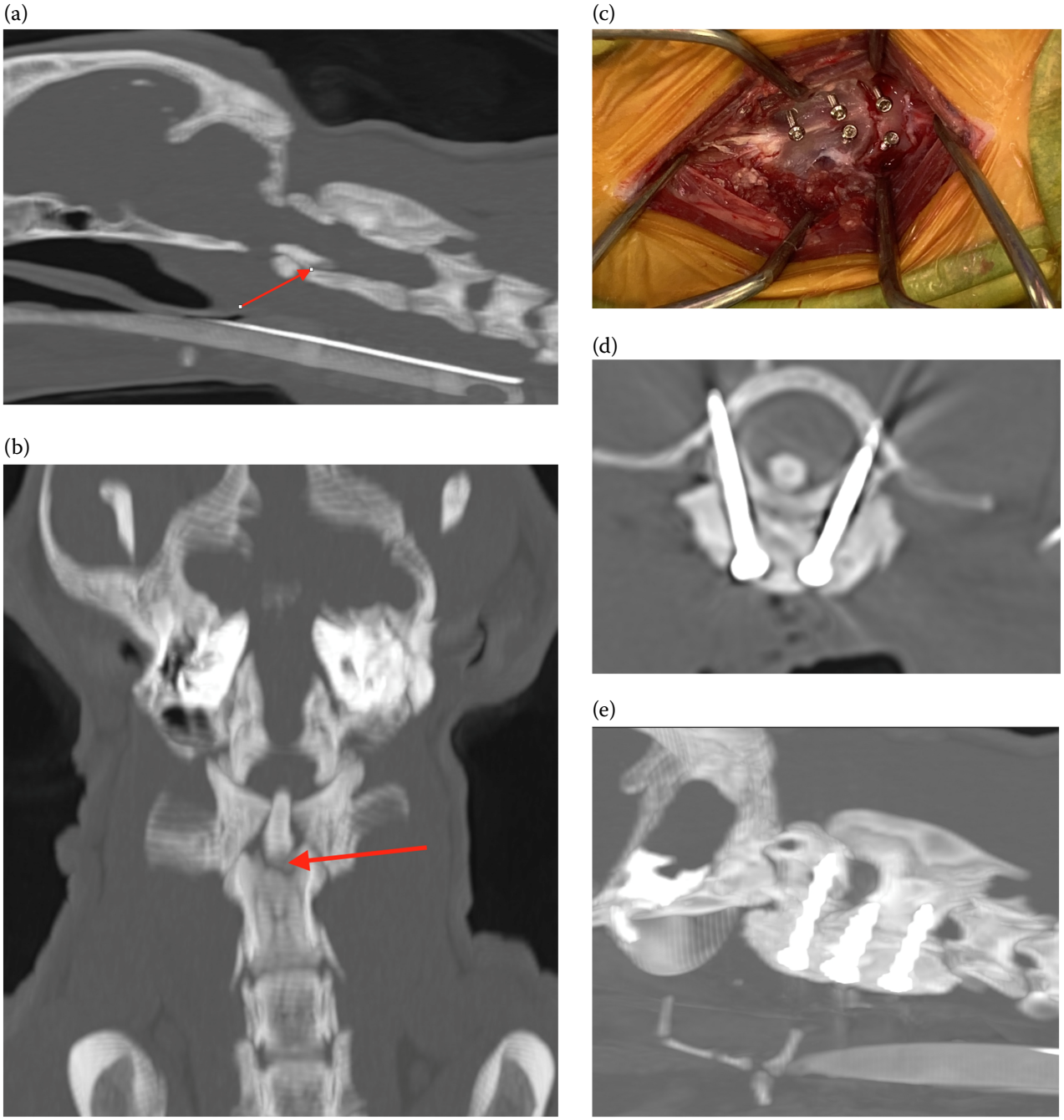


Figure 14.4 Screw and bone cement stabilisation of a fracture of the axis. **(a)** CT sagittal multiplanar reconstruction showing fracture of the axis. The cat was ambulatory tetraparetic. **(b)** CT dorsal multiplanar reconstruction. **(c)** Intraoperative photograph after placement of 2 screws in the atlas and 3 screws in the axis using a ventral approach. The head is on the right side of the image. **(d)** Postoperative transverse CT image showing positioning of screws in the lateral masses of the atlas. **(e)** Postoperative CT 3D reconstruction showing reduction and stabilisation of the fracture with screws and bone cement. (courtesy of Andrew Craig).

The usual cause of spinal fractures and dislocations is road traffic accidents, but spinal injury may also occur as a result of dog bites, metal projectiles, and jumps or falls from a great height. The primary tissue of concern following spinal trauma is nervous rather than osseous, in contrast to fractures and dislocations affecting the appendicular skeleton. There are two components to any acute or primary traumatic spinal cord injury. The first is the concussive effect of the forces applied at the time of the trauma and, to a lesser extent, the ongoing instability that follows disruption of the vertebral column. The second is the residual compressive effect of the tissues surrounding the spinal cord. Severe spinal cord compression is addressed by restoration of the normal anatomy of the vertebral column and, if necessary, by decompressive surgery. The pathophysiology of severe concussive injury involves a

cascade of events known as secondary or delayed injury mechanisms that exacerbate the original trauma and result in reduced spinal cord perfusion. These events include vascular changes, excitotoxicity, free radical production, ion disturbances and inflammation. The vascular changes impair the normal autoregulatory mechanisms of the spinal cord and leave it vulnerable to the effects of systemic hypotension.

BIOMECHANICS AND CLASSIFICATION:

(Bagley 2000, Bali 2011, Beslati et al. 2002, Jeffery 2010, Schmidli et al. 2019, Vedrine & Maurin 2017).

Spinal fractures and luxations generally occur at the junctions between a flexible and relatively inflexible region of the vertebral column, adjacent to the skull, thorax and pelvis. Normal forces on the vertebral column are flexion, extension, torsion, compression and traction. The type of injury will be determined by the interaction between the specific anatomy of the affected region of the spine and the size and direction of the forces involved. The thoracic region constitutes the most stable region of the spine and many injuries occur at the thoracolumbar junction, with approximately 50% of injuries occurring between T10 and L2. Other common locations for injury are the lumbar region and the junction of the sacrum and the caudal vertebrae. Cervical vertebral fractures are rare in the cat in comparison with the dog with three studies involving a total population of 98 cats reporting no cervical lesions and one study of 69 cats reporting only two cervical lesions. The reasons for the species difference are unclear but may relate to aetiology and body size. Fracture of the atlas and the axis has been reported rarely in cats and there are only a few reports of atlantoaxial instability (Figure 14.4). The cervical region normally permits a large range of movement in all planes whereas movement in the thoracic and lumbar regions is largely restricted to the sagittal plane which may partly explain the low frequency of cervical fractures. For the purposes of assessing the stability of the spine it is convenient to divide the vertebral column into three compartments (Figure 14.5).

1. The dorsal compartment comprises the spinous process, dorsal ligamentous structures (flaval, interspinous and supraspinous ligaments), pedicles, lamina, articular processes and joint capsules.
2. The middle compartment comprises the dorsal longitudinal ligament, dorsal anulus fibrosus, dorsal aspect of the vertebral body, transverse processes and intertransverse ligaments.
3. The ventral compartment comprises the remaining vertebral body and anulus fibrosus, the nucleus pulposus and the ventral longitudinal ligament.

Injuries to two or more of these compartments are generally considered unstable and surgical stabilization should be considered. The decision to perform surgical stabilization will also depend on the neurological status, the chronicity of the injury and the presence of concurrent injuries.

- Isolated dorsal compartment injuries with fracture of the articular facets usually result from hyperextension of the spine and are generally stable.
- Isolated ventral compartment injuries, with wedge compression fracture of the vertebral body usually result from hyperflexion of the spine and are generally stable.
- Minimally displaced combination dorsal, middle and ventral injuries with fracture of the articular facets, compression fracture of the vertebral body and extrusion of the intervertebral disc usually result from axial compression of the spine and are often unstable.
- Grossly displaced combination dorsal, middle and ventral compartment injuries with fracture of the articular facets and fracture of the vertebral body or complete disruption of the intervertebral disc usually result from a mixture of torsion and flexion of the spine and are extremely unstable.

The various types of traumatic spinal injury encountered include:

- Vertebral fracture.
- Vertebral luxation or subluxation.
- Intervertebral disc extrusion.
- Spinal cord concussion.
- Spinal cord haemorrhage and haematoma.

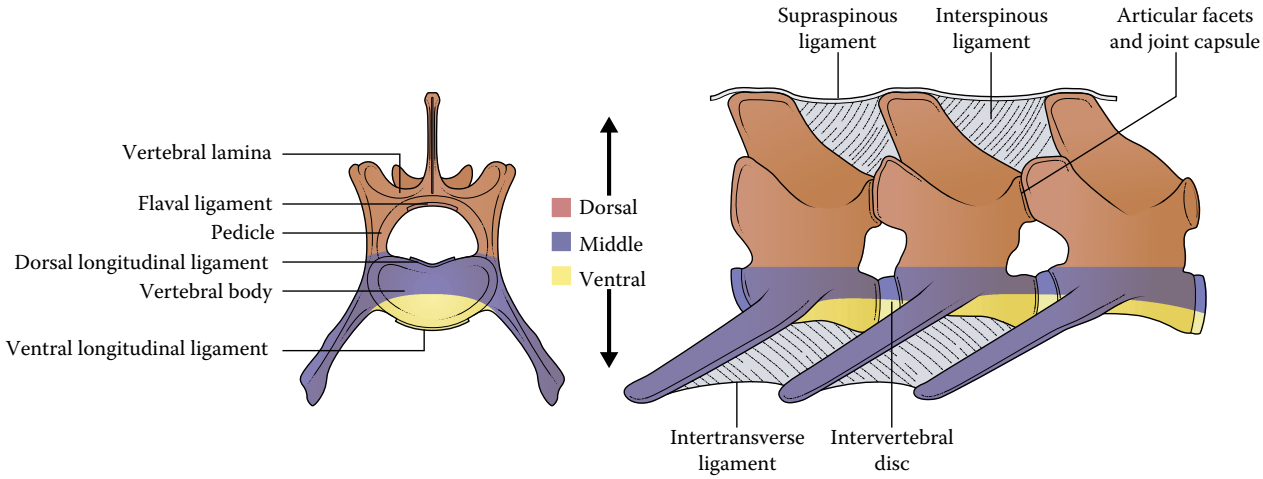


Figure 14.5 The three compartments of the vertebral column shown in three adjacent lumbar vertbrae.

FRACTURES AND DISLOCATIONS OF THE THORACOLUMBAR AND CERVICAL SPINE:

(Bagley 2000, Carberry et al. 1989, Eminaga et al. 2011, Feeney & Oliver 1980, Jeffery 2010, McKee 1990, Selcer et al. 1991).

Clinical Signs

Clinical signs vary from sudden onset spinal pain to paraplegia or tetraplegia, depending on the severity and the location of the injury. Sacrocaudal fractures and luxations may produce urinary incontinence and paralysis of the tail, with or without hindlimb paresis. It is important to recognize the possibility of vertebral fracture or luxation at an early stage of the examination to avoid manipulation that may cause further neural injury. This can be challenging in those cases that have spinal pain but are not showing any overt signs of neurological dysfunction and in those cats where there are concomitant injuries such as pelvic fracture that may mimic paraparesis. In cats with more than one spinal cord lesion, as a general rule lower motor neuron signs mask upper motor neuron signs, for example it may be difficult to recognize a T3–L3 lesion if there is a more caudal injury between L4 and S3 spinal cord segments, or similarly between C1–C5 if there is also a lesion between C6–T2. Localization may also be confounded in a number of cats with severe spinal cord injury because of the presence of spinal shock. Cats with spinal shock suffer a temporary loss of muscle tone and absent reflexes caudal to the level of the injury irrespective of the localization. In the authors' experience spinal shock is less common in cats than dogs; in both species signs disappear within the first 12–48 hours after the initial injury. In such cases, the cutaneous trunci reflex although not completely reliable in cats can be helpful if the lesion is between segments T3–L3. The presence of spinal shock should not dissuade the clinician from pursuing appropriate investigation and treatment.

Diagnosis and Prognosis

In cases where spinal fracture or luxation is suspected, handling of the patient should be minimized until stability of the spine can be assessed radiographically. If tolerated the cat can be secured to a flat board or stretcher pending an assessment of spinal stability. Concurrent injuries are common particularly in cats that have suffered vehicular trauma and it is important to recognize and prioritise them accordingly. A full neurological examination is contraindicated because of the risk of inflicting further injury on the spinal cord. It is possible to perform a modified examination involving testing of myotatic and flexor reflexes and deep pain perception to help determine the location and severity of the lesion. Patients should be assigned to a grade on the basis of the degree of neurological dysfunction as discussed in Chapter 15.

An initial radiographic assessment is performed on the conscious patient, if possible, but precautions must be taken to avoid struggling. The administration of sedation or general anaesthesia may be necessary if this is not contraindicated; for example, because of hypovolaemia or the presence of other injuries. If the initial radiographs show that the spinal injury is likely to be stable, further radiographs may be obtained under general anaesthesia. If the injury is deemed to be unstable anaesthesia may be best delayed until the time of surgical stabilization. In some cases the lesion may be subtle and good quality, well-positioned radiographs in two planes are essential to allow a proper evaluation (Figure 14.6). When radiography shows marked displacement of the vertebrae the spinal cord injury will be severe. If there is greater than 50% displacement of the vertebral canal on both orthogonal radiographs combined with an absence of deep pain perception, it is assumed that there is functional, if not anatomical, spinal cord severance and the prognosis is generally hopeless. Greater vertebral displacement can be tolerated in the lumbosacral spine because there is more space in the vertebral canal relative to the neural structures and the cauda equina is more tolerant of deformation than the spinal cord.

It is important to be aware of the limitations of spinal radiography; assessment of the prognosis is based on the degree of spinal cord injury and cannot be reliably predicted from the radiographic appearance of the vertebral column alone (Figure 14.7). Furthermore, radiographs do not show

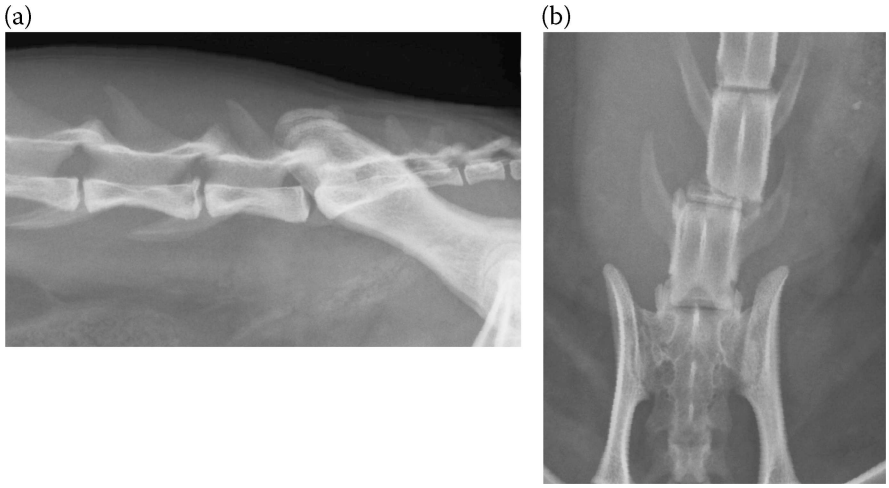


Figure 14.6 Well positioned orthogonal radiographs should be obtained to evaluate spinal fractures and luxations. **(a)** Lateral radiograph showing fracture of the L6 caudal vertebral endplate. The lesion appears to be relatively mild on this view. **(b)** Ventrodorsal radiograph of the fracture showing the degree of displacement of the vertebrae. There is disruption of all 3 compartments, gross displacement and instability.

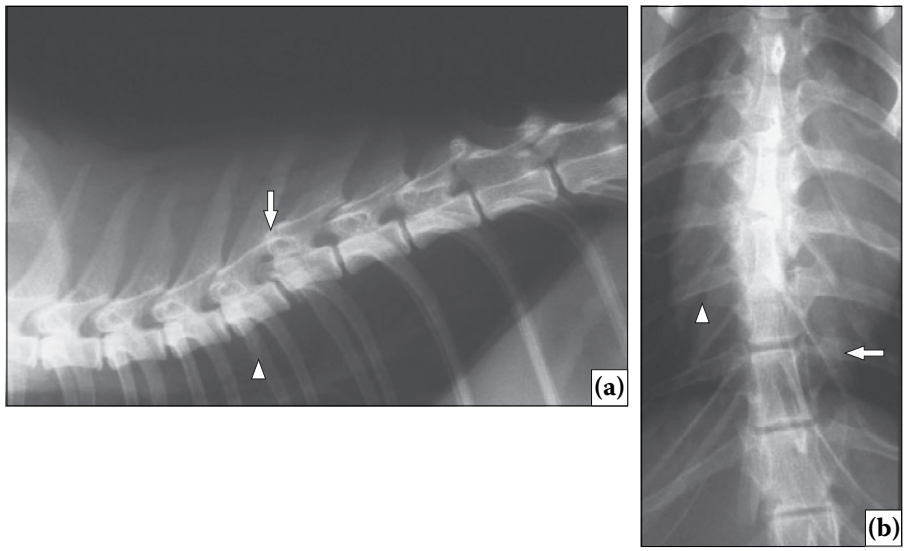


Figure 14.7 Spinal fracture/luxation. Despite the relatively mild degree of vertebral displacement, the cat had no deep pain perception caudal to the lesion. **(a)** Lateral radiograph of the thoracic spine showing spinal fracture/luxation at T7–T8 (arrow). Note the rib fracture just ventral to T7 (arrowhead). **(b)** Ventrodorsal radiograph the same cat. The spinal lesion is much less apparent on this view.

the maximum degree of displacement achieved at the time of the injury so it is possible to underestimate the extent of the spinal injury. Despite these reservations, radiography is an essential tool for assessing the extent of the injury and determining treatment options. It is advisable to radiograph the whole spinal column because multiple injuries are not uncommon; in one study, 20% of patients had a second spinal fracture/luxation. Advanced imaging, particularly computed tomography is becoming increasingly available and is specifically indicated either when the radiographs reveal no abnormalities or when the radiologic findings fail to correlate with the results of the neurological examination. Lesions that can only be identified by advanced imaging include some traumatic intervertebral disc extrusions, spinal haemorrhage and spinal cord concussion. Advanced imaging is indicated to facilitate lateralization of the lesion when cord decompression by hemilaminectomy is planned. Computed tomography may be helpful in those rare cases where further assessment of spinal instability is considered desirable by the use of stressed views. However, great care must be taken to avoid further injury to the spinal cord when performing the manipulations required for such studies.

Surgical Planning

Computed tomography is extremely helpful for surgical planning in all cats with spinal fracture/luxation (Figure 14.8). For non-urgent cases the authors recommend the use of commercially available patient-specific three-dimensional drill guides to improve the accuracy and speed of implant placement into the narrow safe corridors that are available in cat vertbrae (Vet 3D Cumbria, UK). The drill guides are designed using data derived from a CT scan.

Optimal safe corridors for bicortical implant placement in the cervical vertebrae of cats have recently been described based on CT imaging and are different from those of the dog. In the cervical spine the safe corridor insertion points for implants are located within the caudal third of the vertebral body of C2 and in the cranial third of the vertebral bodies from C3 to T1. Mean safe corridor widths were in the range of 1.0 mm for C2 to 2.3 mm for C7 and T1. The mean optimal angles of implantation for the cervical spine are shown in Table 14.1.

Optimal safe corridors for bicortical implant placement in the thoracolumbar spine (T10–L7) have also been reported. Insertion points were located at the level of the vertebrocostal joint or the base of the transverse process of the

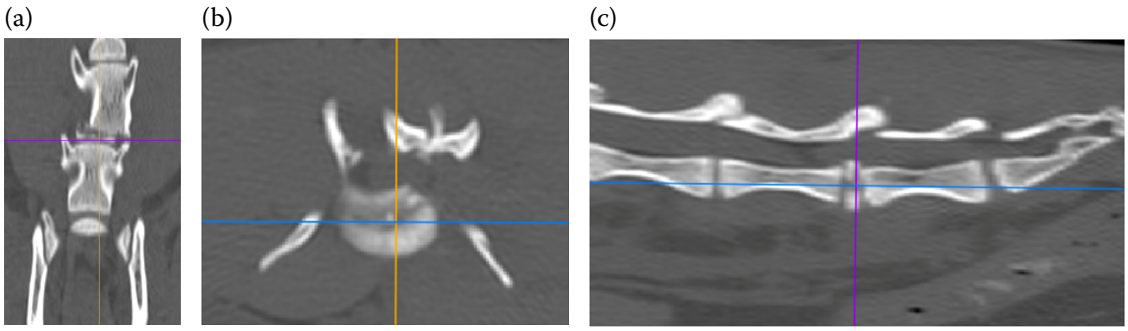


Figure 14.8 CT multiplanar reconstruction of the same fracture as Figure 14.5. **(a)** Dorsal image. **(b)** Transverse image at the level of the fracture. **(c)** Midline sagittal image.

Table 14.1: Optimal Insertion Angles for Implants Placed in Vertebrae C2–T1 with Respect to the Sagittal Plane

Vertebra	Angle°	Angle range°
C2	22	(13–36)
C3	46	(40–51)
C4	51	(47–57)
C5	54	(48–59)
C6	57	(49–64)
C7	64	(53–70)
T1	53	(49–59)

Source: Espadas et al. 2018.
Notes: Values are the mean values of the left and right sides. The insertion points for C3–C6 and T1 were located in the midline. The insertion point for C2 and C7 were located at an average of 3.1 mm and 1.4 mm off midline, respectively.

vertebral body for thoracic and lumbar vertebrae, respectively. The mean optimal angle of insertion was perpendicular to the vertebrae with a range of approximately 80–100°. Perpendicular implant placement is ideal but is not always possible because of surrounding soft tissues and a small deviation of up to 10° dorsally is acceptable. The implants used should not exceed 2.0 mm in diameter.

Treatment and Prognosis

There are relatively few reports of treatment in the literature and many of those cases that have been reported have been treated conservatively rather than surgically. The goals of treatment are to relieve spinal cord compression, maintain stability during healing, and mitigate the effects of secondary spinal cord injury. The choice of treatment is based on the duration, severity and progression of the neurological dysfunction and an assessment of the stability at the site of injury. Additional concerns include financial constraints, the experience of the veterinarian, and the availability of referral. If surgical intervention is chosen as the best course of action it should generally be performed as soon as the patient is sufficiently stable to undergo general anaesthesia safely.

The options available for management of spinal fracture/luxation include:

- Conservative treatment.
- External splinting or casting.
- Internal fixation.
- Internal fixation plus spinal cord decompression.

There is currently no satisfactory method of treatment for secondary injury despite the fact that it has been the focus of much research. Methylprednisolone sodium succinate (MPSS) is the only available drug that has been shown to have any neuroprotective effect, albeit in human clinical trials and only if given within 8 hours of the initial spinal injury. The potential benefits of this drug are because of its free radical scavenging ability and not because of its glucocorticoid action. Recent evidence of its use in dogs suggests that there is no improvement in neurological recovery. Side effects of high doses of glucocorticoids include gastric haemorrhage, colonic perforation, hyperglycaemia and increased likelihood of urinary tract infection. The use of MPSS and other glucocorticoids should therefore be avoided in cats with spinal injury.

Conservative Treatment

Provided deep pain perception is still present, many cats with spinal fractures and luxations will return to satisfactory function with time, strict cage rest and good nursing care. It should be borne in mind that the cost saving with conservative treatment may be less than expected. The expense of surgery should be weighed against the extra costs likely to be incurred, as a result of the increased duration of nursing care required, if the same patient is managed conservatively.

Conservative treatment is indicated if radiographic assessment shows that the injury is likely to be stable and there is strong voluntary movement in the limbs. Single dorsal or ventral compartment injuries are generally stable, whereas injuries involving two or more compartments are generally unstable. In cases where there is doubt the patient may be hospitalized for conservative treatment and observed carefully for progression of neurological signs. Worsening of the neurological status in a cat undergoing proper conservative treatment is generally a sign of spinal instability and is an indication for surgical intervention. If the patient has no voluntary movement, but deep pain perception is present and the spinal injury is stable with little displacement, there is a fair prognosis for return to function with conservative treatment. Surgical stabilization, and decompression if necessary, will likely improve the prognosis for a successful outcome however, and there will be a more rapid and complete return to function.

The prognosis for complete return of function in patients with an absence of deep pain perception as a result of trauma is very poor and euthanasia is usually recommended. Failure to regain deep pain perception does not always preclude recovery of hindlimb motor function (although urinary and faecal incontinence are likely to persist). Recovery of motor function can still occur if descending axons survive the injury. Survival of as few as 5–10% of axons is sufficient for restoration of basic locomotion in cats. In cats with complete spinal cord transection a degree of locomotor function may also return as a result of the activity of local spinal circuits (spinal reflex walking).

External Splinting or Casting

(Patterson & Smith 1992).

External coaptation can be used as the sole method of treatment or may be used to supplement internal fixation. To the authors' knowledge there are no descriptions of the use of external coaptation in a series of feline spinal fractures and luxations. Back splinting has been used with some success in the dog, where the main indication is for

thoracolumbar injuries. The cat's small stature and propensity to rest means that cage confinement may be sufficient for many of the injuries where splinting would be indicated for the equivalent canine injury. Nevertheless, splinting may occasionally be applicable particularly for cats with thoracolumbar injuries. The ideal candidate would be one where there is still voluntary movement in the hindlimbs and the injury is confined to the dorsal compartment of the vertebral column, with an intact vertebral body and intervertebral disc providing a ventral buttress.

The goal of splinting is to immobilize the spinal segments cranial and caudal to the site of injury. Various materials can be used but thermoplastic material is ideal since it can be moulded to fit the patient. The splint is conformed to the shape of the body along the dorsal spine, extending from the withers to the base of the tail. It is preferable to apply the splint with the patient sedated rather than anesthetized so that the protective muscle tone of the epaxial muscles is maintained. The splint is first held against the spine with the cat lying on its side restrained by two people. The cat is then gently rolled into dorsal recumbency synchronously with the splint so that the splint cradles and supports the spine. Application is completed by the addition of Elastoplast strips cranially and caudally, encircling the splint and incorporating the forelimbs and the hindlimbs, respectively. If the cat struggles and will not tolerate the splint, it should be removed.

Reduction and Internal Fixation

Internal fixation is the recommended method of treatment in unstable and/or displaced fractures and luxations of the spine, especially those cases where the injury involves more than one compartment. Many techniques have been described but the most commonly used options for internal fixation in the cat are pins and polymethylmethacrylate (PMMA), vertebral body plating, and modified segmental fixation using pins and wire. The goals of reduction and internal fixation are to eliminate ongoing neural compression by displaced anatomical structures and to provide stability during the healing process. Although perfect anatomical reduction is desirable it should not be at the expense of further injury to the spinal cord.

Reduction of cervical vertebral fracture/luxations can be facilitated by the use of a small Gelpi retractor placed in the intervertebral disc spaces cranial and caudal to the lesion after partial fenestration. Alternatively, a small hole can be drilled in the ventral vertebral body either side of the lesion to accommodate the points of small pointed reduction forceps. Reduction of fracture/luxations of the thoracic and lumbar spine can be achieved by placing traction on the spine using very small pointed fragment (reduction) forceps attached to the dorsal spinous processes or the vertebral bodies of the vertebrae either side of the lesion (Figure 14.9). Correct reduction is assessed by noting the position of the articular facets and by inspecting the floor of the vertebral canal if hemilaminectomy has been performed. Reduction can be maintained by continued traction on the bone holding forceps or by temporary insertion of a small K wire across the articular facet.

Pins or Screws and Bone Cement

(Bruce et al. 2008, Espadas et al. 2018, Garcia et al. 1994, Vallefuoco 2013, Vallefuoco et al. 2014).

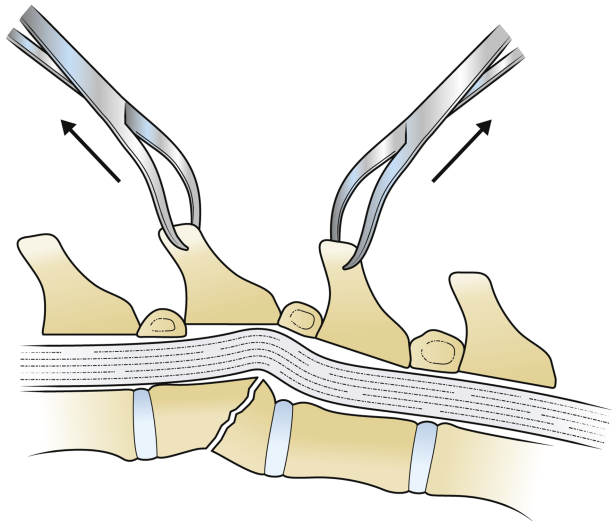


Figure 14.9 Reduction technique for displaced thoracolumbar spinal fracture/luxations. Traction is placed on the spinous processes or vertebral bodies either side of the lesion using small fragment forceps or towel clamps. To avoid fracture of the dorsal spinous processes the force used should not be excessive.

This is a versatile method of spinal stabilization first described in dogs and more recently in cats with vertebral fracture/luxations. It does not require a great deal of specialized equipment and can be performed in all regions of the spine. However, it does require a thorough knowledge of vertebral anatomy and strict attention to aseptic technique. There is little margin for error when placing the pins or screws and anatomic landmarks vary from one region of the spine to another, so it is helpful to have an anatomic specimen available for reference during the procedure. Accuracy of implant placement can be greatly improved by preoperative computed tomography. Additionally, the use of patient-specific three-dimensional drill guides, derived from the CT scan, is recommended for non-urgent cases (Vet 3D Cumbria, UK). Optimal safe corridors for implant placement have been described for the cervical and for the thoracolumbar spine in cats (Table 14.1 and Figure 14.10). In the thoracolumbar spine the greatest amount of bone purchase with safe margins for bicortical implant placement is obtained by insertion in the vertebral body either perpendicular to the median plane or with a small angle of deviation (<10°) from the perpendicular. In contrast to the dog the optimal safe implantation corridors for L7 are the same as those of other lumbar vertebrae. However, passage of implants through the iliac wing or partial ostectomy of the iliac wing may be necessary. Insertion points are situated at the level of the costovertebral joints for thoracic vertebrae or the base of the transverse process of the vertebral body for lumbar vertebrae. Nonetheless, safe corridors for implant placement in feline vertebrae are narrow, and it is therefore advisable to take account of individual variation between patients and even variation between right and left sides in the same patient. Postoperative CT has been shown to be more accurate than conventional radiography in identifying vertebral canal violation by implants and when available should always be

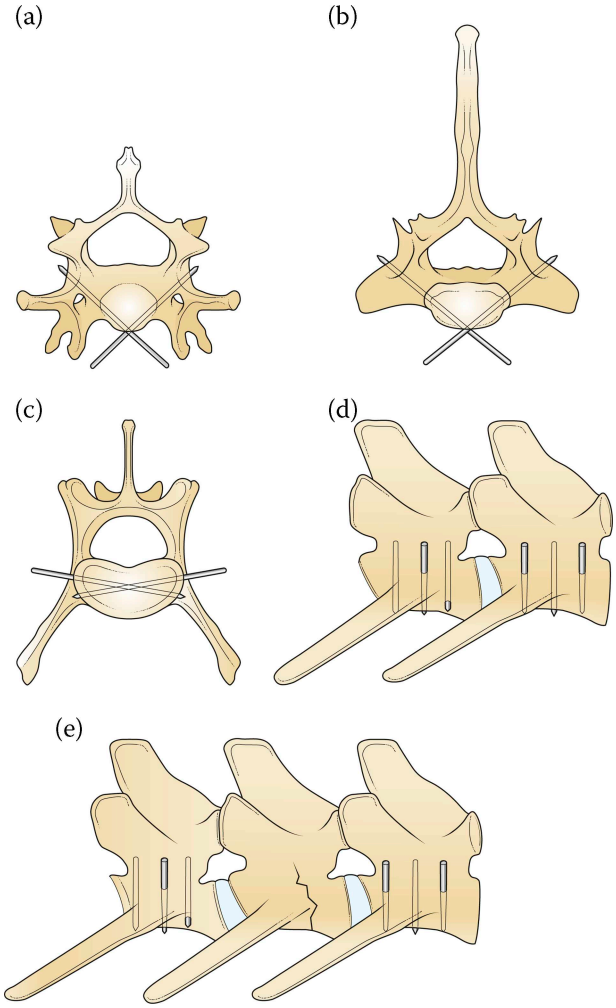


Figure 14.10 The use of pins and bone cement for repair of spinal fractures and luxations. **(a)** Optimal placement of pins in the cervical vertebrae. **(b)** Optimal placement of pins in the thoracic vertebrae. **(c)** Optimal placement of pins in the lumbar vertebrae. **(d)** Optimal placement of pins in two adjacent vertebrae. **(e)** If there is fracture of the vertebral body, the pins are placed in the vertebral bodies cranial and caudal to the lesion to span the lesion.

performed to assess the accuracy of implant positioning. A biomechanical study in canine cadavers of simulated lumbar fracture/luxation compared screws and bone cement with pins and bone cement and found that screw fixations failed by screw bending and were less rigid and weaker at failure than the corresponding pin configuration. However, these considerations are less likely to be of importance in cats because of their small body size.

A routine ventral approach is used for most cervical lesions whereas a dorsolateral or lateral approach is used for thoracic and lumbar lesions. For the dorsal approach, epaxial muscles are reflected from one or both sides of the spine to the level of the transverse process if the lumbar spine is involved. In the thoracic region, dissection is continued to the costal fovea taking care not to penetrate the pleural cavity. If decompressive surgery is to be performed, then this is done first. A minimum of two or preferably three pins or screws are then placed into the vertebrae either side of the fracture/luxation to span the lesion using a slow speed electric or air drill after predrilling. Pin and screw size should be 1.0–2.0 mm in diameter. Unilateral placement requires less soft tissue dissection and often provides sufficient stability for cats. If the lesion is mid-body (fracture), the pins are placed in the adjacent vertebral bodies. If the lesion is at the level of the intervertebral disc space (fracture/luxation), the pins are placed in the vertebrae either side of the disc. Positive profile threaded pins will maintain stability longer than smooth pins and are quicker and easier to place than bone screws. Small-diameter threaded pins that have a roughened area of pin shank to enhance the interface with the bone cement are available (Imex Miniature Interface™ fixation half pins). The pins are driven so that they emerge 2–3 mm from the ventral aspect of the vertebral body to engage both bone cortices. The pins are then cut just below the level of the dorsal spinous processes and bent over or notched with a pin cutter if they have a smooth shank. When bone screws are used the screw is left protruding to engage with the bone cement. The ratio of screw in bone to screw left protruding should be in the range of 1:1 to 1.5:1. A small pack (20 g) of PMMA bone cement is mixed and about half of the cement is applied to connect the implants. Once it has reached the doughy stage and does not stick to the surgeon's gloves, the cement is packed around the pins taking care to avoid the spinal cord and nerve roots. The amount of cement should not be so excessive that it precludes subsequent closure. The cement is lavaged with cool saline for five minutes to dissipate the heat produced as it polymerizes. For thoracolumbar lesions the lumbodorsal fascia is sutured over the cement mass with monofilament absorbable suture material. Occasionally it may be necessary to place releasing incisions in the lumbodorsal fascia to permit closure. The remainder of the wound is closed routinely. For cervical lesions a ventral approach is made and the longus colli muscles are reflected from the relevant vertebral bodies. The pins or screws are angled from the midline away from the vertebral canal to engage both cortices of the vertebral body. In order to achieve maximum bone purchase and avoid neurovascular structures implants placed in vertebrae C3–T1 should be located in the cranial third of the vertebral body. Pins are cut and notched, if necessary, and embedded in bone cement. The longus colli muscles are closed cranial and caudal to the cement and the remainder of the closure is performed routinely.

Vertebral Body Plating

(Nel et al. 2017, Sturges et al. 2016, Swaim 1971, Vallefuoco et al. 2013).

This is a very effective means of stabilization for lumbar vertebral body fractures or luxations and is the authors' preferred technique for these injuries. A recent biomechanical study compared the use of a unilateral locking plate with monocortical screws with bilateral bicortical positive profile pins and polymethylmethacrylate in the lumbar spine of canine cadavers. Not surprisingly the bilateral construct had greater vertebral stiffness however both constructs were found to be stronger than intact specimens. Application requires specialized equipment and as for the pins and bone cement technique a thorough knowledge of vertebral anatomy is essential. Pre and postoperative CT should be performed where available. A 2.0 mm or 2.4 mm plate or a 2.0 mm veterinary cuttable plate (VCP), depending on the configuration of the fracture/luxation, can be applied in most cases. Locking plates are preferred as they do not require

excessive plate contouring, they do not rely on friction between the plate and the bone, and although bicortical screw placement is preferable, the screws can be placed in monocortical fashion if required. Polyaxial locking plates that allow angulation of the screws facilitate accurate screw placement in the vertebral body (Figure 14.11). Approach to the thoracic vertebrae is restricted to the caudal thoracic region and requires rib resection to allow placement of the plate. Plating using locking plates can be performed caudal to the fourth lumbar vertebra but care should be taken to avoid the important nerve roots of the lumbosacral plexus. A dorsolateral approach is made to the spine, as described for hemilaminectomy, to the level of the transverse process of the lumbar vertebrae or the head of the rib in the case of the thoracic vertebrae. For unilateral plating the plate chosen should be of a sufficient length to allow placement of at least

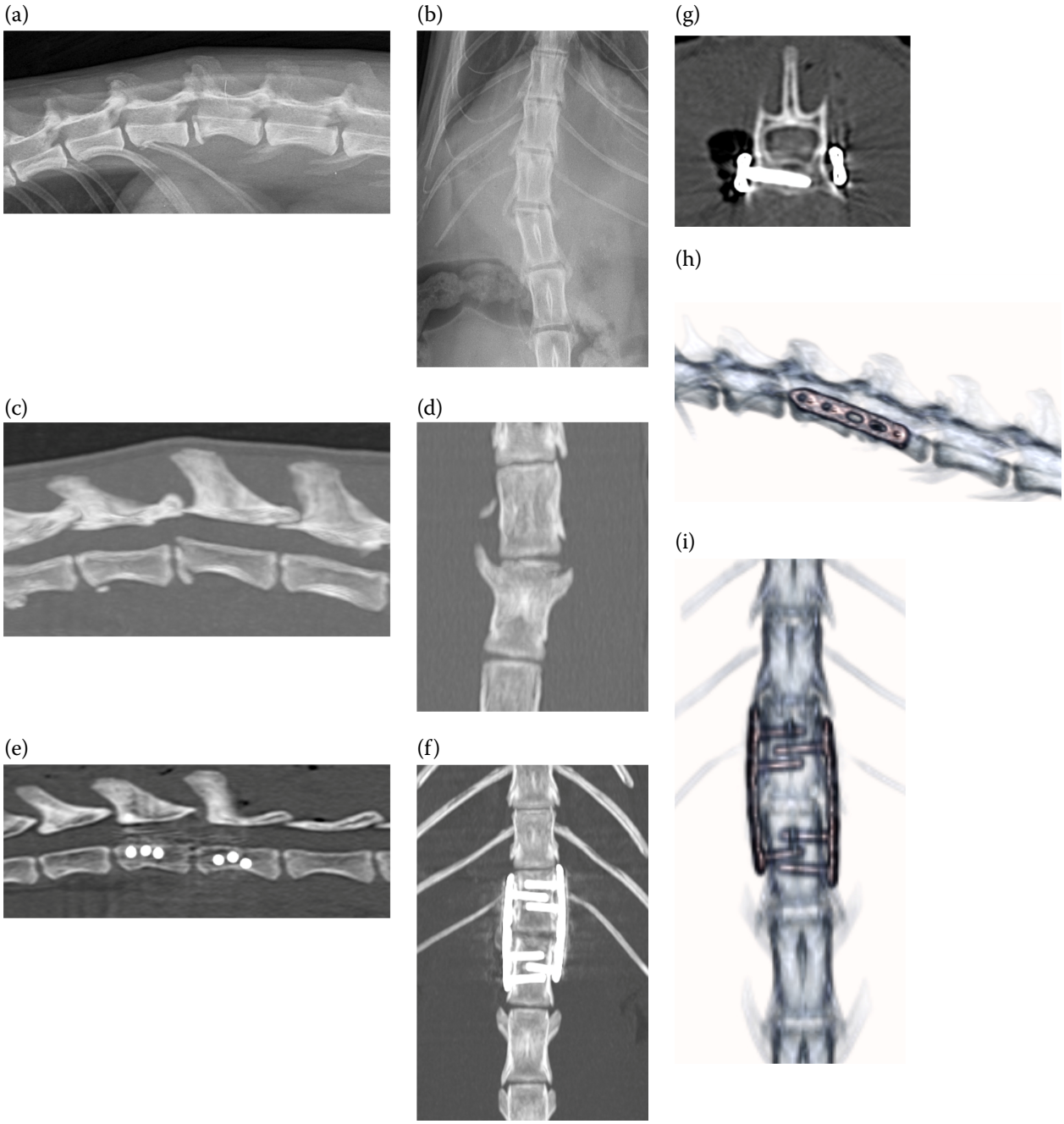


Figure 14.11 Bilateral vertebral body plating of a thoracolumbar spinal fracture or luxation. **(a)** Lateral radiograph of a fracture/luxation at T13-L1. The cat was paraplegic with intact nociception. **(b)** Ventrodorsal radiographic view. **(c)** CT multiplanar sagittal reconstruction. **(d)** CT multiplanar dorsal reconstruction. **(e)** Postoperative sagittal CT image showing placement of screws in the vertebral bodies of T13 and L1. **(f)**Postoperative dorsal CT image showing placement of bilateral locking plates and 6 screws. **(g)** Postoperative transverse CT image showing correct placement of a screw in the vertebral body of L1. **(h)** Postoperative CT 3D reconstruction sagittal view. **(i)** Postoperative CT 3D reconstruction dorsal view.

two screws in the vertebral body cranial and caudal to the lesion. If the lesion is mid-body (fracture), the screws are placed in the adjacent vertebral bodies. If the lesion is at the level of the intervertebral disc space (fracture/luxation), the screws are placed in the vertebrae either side of the disc. Safe corridors for optimal bicortical screw placement in the vertebral body are identical to those already described for pins or screws and bone cement. If a hemilaminectomy has been performed, screw holes are drilled using the anatomic location of the spinal cord as a reference point.

Spinal Stapling (Modified Segmental Spinal Fixation)

(McAnulty et al. 1986, Voss & Montavon 2004).

Spinal stapling involves wiring of a U-shaped pin to the dorsal spinous processes and the articular processes or transverse processes of the vertebrae (Figures 14.12 and 14.13). The technique can be used to stabilize lesions in all regions of the spine with the exception of the cervical region. It is relatively easy to perform and requires no specialized equipment. However, pin migration and loss of fixation are potential complications and it is therefore not the authors' preferred technique. Furthermore, application requires more extensive soft tissue dissection than other techniques, with exposure of 2–3 dorsal spinous processes and articular facets cranial and caudal to the lesion.

Following a bilateral dorsal approach, a K wire (or small intramedullary pin) 1.0–1.6 mm in diameter is passed through the skin and epaxial muscle and is inserted through the dorsal lamina of a vertebra adjacent to the lesion. The entire length of the wire is passed through the opposite side and then half withdrawn while retracting the epaxial musculature to free it from the wire. The wire is then bent to a U shape using bending pliers and carefully contoured to fit along the dorsal laminae between the spinous and articular processes. The wire is held in place with a hemicerclage wire (0.6 mm [22 AWG]) passed through a hole drilled in the dorsal lamina of the vertebra at its opposite end. The fracture/luxation is stabilized with a figure-of-eight wire passed through the dorsal laminae of the vertebrae at the site of the lesion and placed dorsal to the K wire to span the lesion. The technique is effective although care must be exercised when drilling the holes in the dorsal laminae not to penetrate the vertebral canal. The fixation can be strengthened by placing supplementary cerclage wires attaching the wire to the transverse processes or the ribs but this requires extra dissection and is rarely necessary.

Spinal Cord Decompression

Accurate anatomic realignment and stabilization of fracture/luxations will automatically result in a degree of

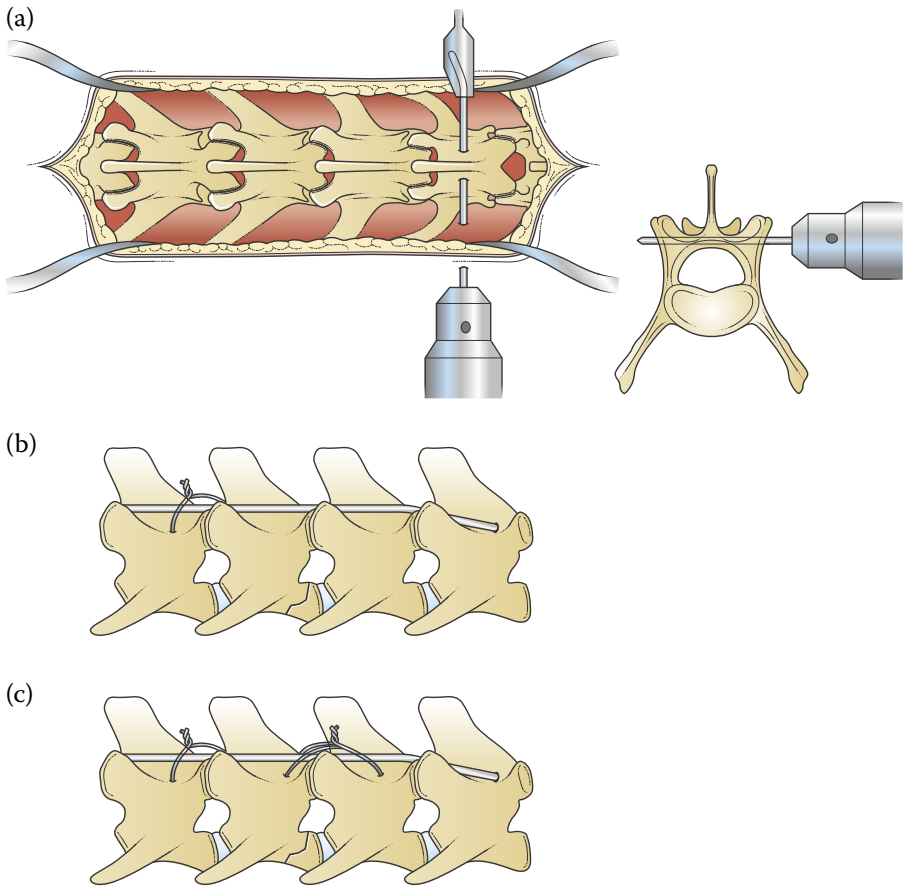


Figure 14.12 Spinal stapling. **(a)** A K wire is inserted through the skin and epaxial muscle into the dorsal lamina. **(b)** The K wire is bent into a U shape and contoured to fit over the dorsal laminae between the spinous processes and the articular facets. A hole is drilled in the dorsal lamina and a hemicerclage wire is placed to stabilize the free end of the Kirschner wire. **(c)** A figure of eight wire is placed through holes drilled in the dorsal laminae of the vertebrae at the site of the lesion.

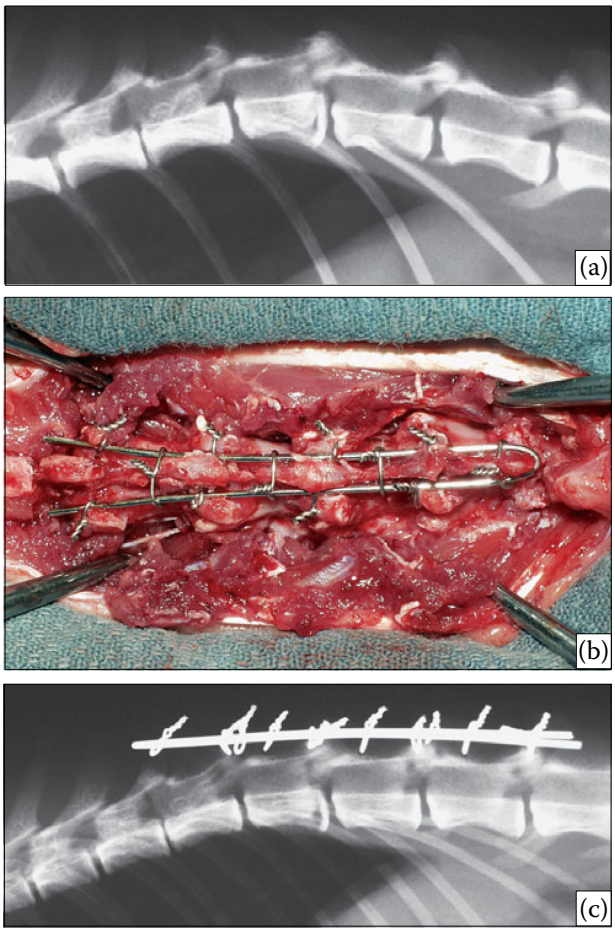


Figure 14.13 Spinal stapling of a thoracolumbar spinal fracture/luxation (Courtesy of Otto Lanz). **(a)** Lateral radiograph of the thoracolumbar spine showing a spinal fracture luxation at T12–T13. **(b)** Intraoperative photograph of the same cat showing placement of the spinal staple. **(c)** Lateral postoperative radiograph of the thoracolumbar spine of the same cat.

spinal cord decompression. However, additional decompression of the spinal cord should be considered when there is no voluntary movement in the affected limbs and/or there is evidence of bone fragments, haematoma, or extruded intervertebral disc in the vertebral canal on radiographs, CT or MRI. Decompressive surgery is indicated most frequently in the thoracolumbar region where the vertebral canal is at its narrowest relative to the diameter of the spinal cord. Cats with absent deep pain perception are usually euthanased, but if they are treated surgically, spinal cord decompression is essential to establish that the cord has not been transected. Thoracolumbar decompression should be performed by hemilaminectomy since this has less of a destabilizing effect than dorsal laminectomy. Hemilaminectomy allows visual assessment of the condition of the spinal cord and removal of compressive material. It is important to note that because spinal cord decompression causes iatrogenic spinal instability it is only ever undertaken as an adjunct to internal fixation.

FRACTURES AND LUXATIONS OF THE LUMBOSACRAL, SACRAL AND SACROCUADAL SPINE:

(Anderson & Coughlan 1997, Beck et al. 2007, Bernasconi et al. 2002, Caraty et al. 2018, Couper & De Decker 2020, Davies & Walmsley 2012, Hurov 1985, Kudnig & Fitch 2004, Mills 2009, Nel et al. 2017, Segal et al. 2018, Smeak & Olmstead 1985, Sunol et al. 2018, Tatton et al. 2009, Taylor 1981, Wilson 2015, Zulauf et al. 2008b).

These are common spinal injuries in the cat especially sacrocaudal fracture and luxation which is seen with some frequency in the cat but is rare in the dog. Fracture/luxations of this region of the spine are considered separately from other spinal injuries because of their unique features. The predominant clinical signs are the result of injury to the nerves of the cauda equina that are derived from the terminal spinal cord segments from L7 caudally. The neurological signs are the result of injury to the peripheral nervous system which in general is more resistant to trauma than the spinal cord.

Cause and Pathogenesis

The most frequent cause of injuries in this region of the spine is vehicular trauma. Traumatic lumbosacral luxation with fracture of the L7 articular facets has also been reported following dorsal laminectomy. Sacral fracture/luxations are more common in the cat than the dog and often occur in combination with sacroiliac luxation. In contrast, lumbosacral fracture/luxation occurs less frequently and has been more commonly reported in the dog than the cat. Sacral fractures in dogs and cats have been described and classified. In the same study concomitant pelvic injuries were common, with 65% of cats having unilateral or bilateral sacroiliac luxation. Sacroiliac luxation is discussed in Chapter 10.

Sacrocaudal luxation or fracture/luxation or so-called "tail-pull injury" is more common than sacral fracture (Figure 14.14). This region of the spine is vulnerable to injury as the articular facets are small and there is little muscle support. The reason for the injury is not always clear but it is thought to occur as a result of traction forces on this region of the spine when a cat, which is attempting to escape, has its tail trapped under the tyre of a moving vehicle.

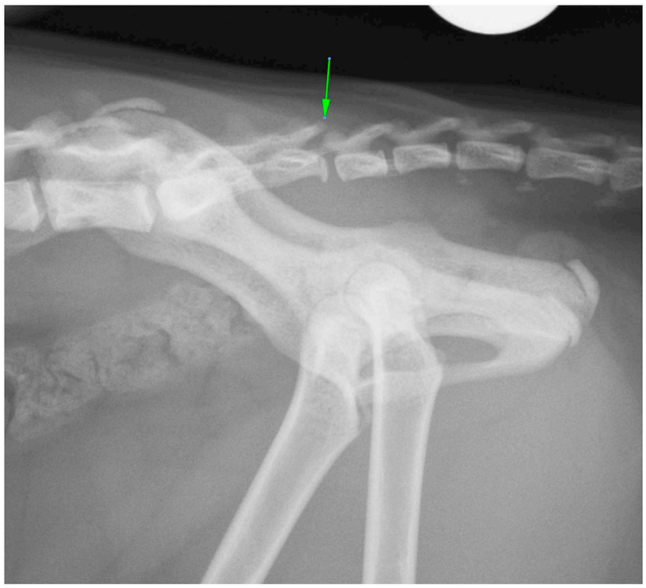
Clinical Signs

Clinical signs of sacrocaudal injuries include pain and swelling in the sacrocaudal region and tail paralysis. There may be severe neurological dysfunction associated with transverse or comminuted sacral or lumbosacral fracture/luxation or when there has been traction on the nerve roots of the cauda equina causing a variety of deficits in hindlimb, anal and bladder sphincter function.

Diagnosis

It is important to obtain radiographs in two orthogonal planes since the osseous injuries may be relatively mild and may be obscured on the ventrodorsal view by faecal material in the rectum. Traction on any of the more cranial nerve roots of the cauda equina (L6–S1) will cause paraparesis with lower motor neuron deficits involving the sciatic nerve. The L7 nerve root which exits at the lumbosacral junction makes a major contribution to the

(a)



(b)

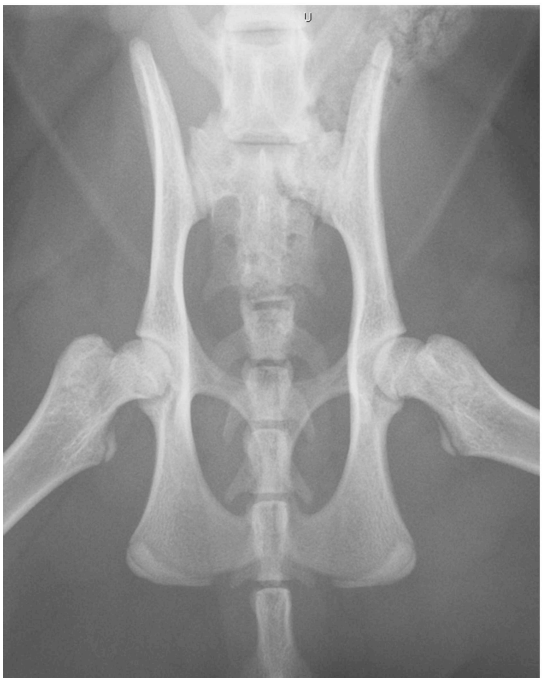


Figure 14.14 Sacrocaudal fracture/luxation **(a)** lateral radiograph **(b)** ventrodorsal radiograph.

sciatic nerve. The pudendal and pelvic nerves originate from the sacral nerve roots (S1–3) and innervate the bladder, rectum and colon. Injury to these nerve roots may cause urinary retention, loss of anal and urethral sphincter tone, and loss of perineal sensation. Injury to the caudal nerve roots Cd1–5 in isolation causes tail paralysis.

Lumbosacral Fracture/Luxation

These injuries are generally unstable with substantial displacement of the affected vertebrae and surgery is nearly always indicated. Dorsolateral locking plates or positive profile pins (or screws) and polymethylmethacrylate have both been shown to provide similar stability in canine cadaver spines and are both applicable to the cat. Application of bilateral 2.0 mm string-of-pearls locking plates has been reported in two cats with lumbosacral fracture/luxation. Successful treatment of traumatic luxation of the lumbosacral joint in two cats using a suture sling has also been reported. In both cases after reduction of the luxation a suture sling anchored between the spinous processes of L7 and S2 was used in order to prevent hyperflexion of the joint and reduce the risk of reluxation. Stabilization of the lumbosacral spine is discussed further in Chapter 15.

Sacral Fractures

Sacral fractures can be classified based on whether they are abaxial, lateral to the foramina, or axial, medial to the foramina. This is a useful distinction as axial fractures involve the nerve roots of the cauda equina and will generally be more painful and will have more severe neurological dysfunction. Sacral fractures are usually classified based on fracture configuration as type I – alar, type II – foraminal, type III – transverse, type IV – avulsion and type V – comminuted (Figure 14.15). In a study that included 17 cats with sacral fractures, type I and type III fractures were seen most frequently, and type IV and type V fractures were not identified in any of the cats. Type IV fractures in dogs are avulsion fractures of the origin of the sacrotuberous ligament which is not present in cats.

Stabilization of sacral fractures is indicated when there are neurological deficits, obvious displacement of bone fragments or instability likely to cause compression of nervous tissue. Minimally displaced type I (alar) fractures can be repaired by placement of a lag screw (Figure 14.16), or using a tension band technique in conjunction with a transilial pin, in a similar fashion to sacroiliac luxation (see Chapter 10). For foraminal or transverse fractures, comminuted fractures or fractures in which the landmarks for lag screw or pin placement are obscured the preferred method of fixation is the use of a transilial locking plate placed as a bridge between the iliac crests (Figure 14.17).

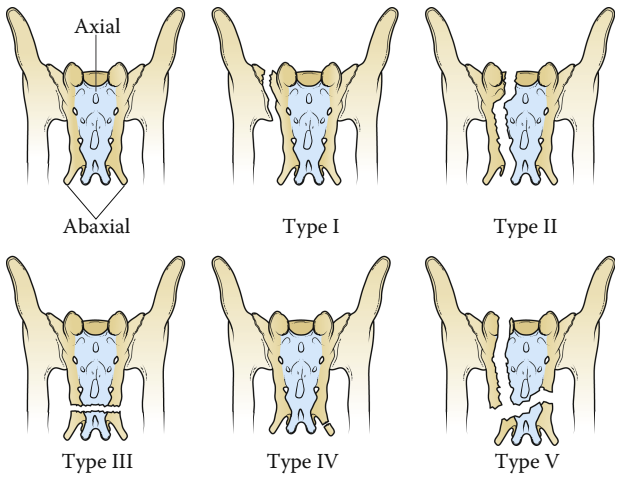


Figure 14.15 Classification of sacral fractures based on fracture configuration. Type 1 – alar. Type II – foraminal. Type III – transverse. Type IV – avulsion. Type V – comminuted. Fractures can also classified as axial or abaxial based on location medial or lateral to the sacral foramina, respectively.

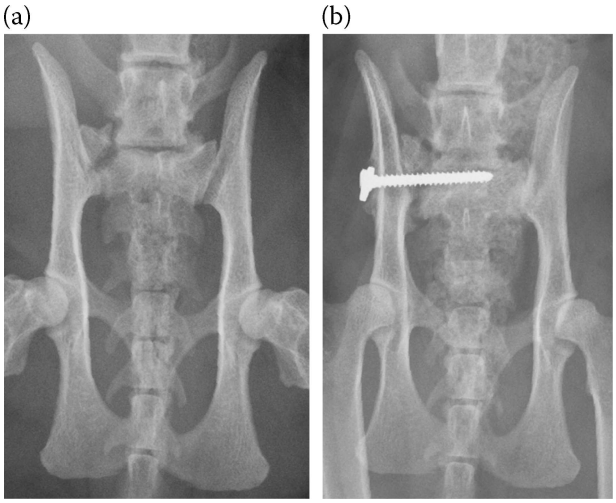


Figure 14.16 **(a)** Ventrodorsal radiographic view of the pelvis of a cat with right sacroiliac fracture/luxation showing a right type I (alar) fracture of the sacrum. **(b)** Follow up ventrodorsal radiograph showing partial healing of the fracture 4 weeks after repair using a 2.4 mm lag screw and washer.

Using locking plate fixation sacral fractures can be buttressed without compression and without the need to place implants near the fracture site provided at least one sacroiliac joint is intact. A technique using pins and bone cement has also been described in one cat with injuries that included a transverse sacral fracture and bilateral sacroiliac luxations. Transverse sacral fractures between the second and third sacral vertebrae (Figure 14.18) can be stabilized using a nonabsorbable suture or hemicerclage wire passed through holes drilled in the base of their respective spinous processes.

To place a locking plate the ilial wings and sacroiliac joints are approached using two parallel skin incisions. A string of pearls plate is used that has been precontoured into a "U" shape, based on radiographs, so that it is slightly narrower than the desired final gap between the iliac crests. The plate is inserted under the epaxial muscles and is first attached to the most stable iliac crest with at least two screws placed in a dorsoventral plane. The contralateral iliac crest is then manoeuvred into alignment and screws are similarly placed to complete the fixation.

Medical treatment of cats with sacrocaudal spinal injury involves pain relief and manual expression, intermittent or indwelling catheterization or placement of a cystostomy

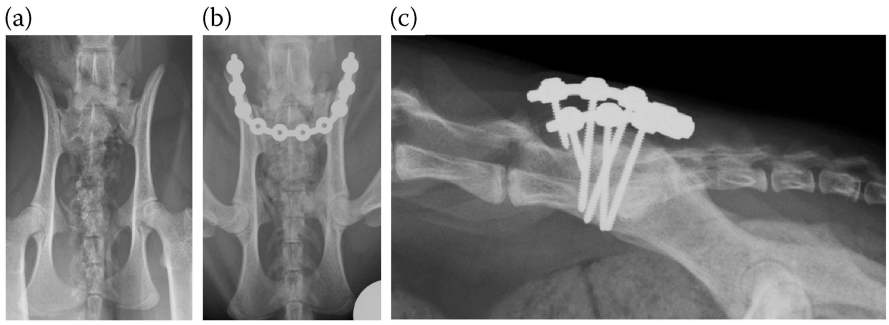


Figure 14.17 **(a)** Ventrodorsal radiographic view of the pelvis of a cat with a left type II (foraminal) sacral fracture. **(b)** Postoperative ventrodorsal radiograph showing fracture repair using a 10 hole 2.0 mm transilial string of pearls locking plate and 6 screws. **(c)** Lateral postoperative radiograph of the fracture repair.

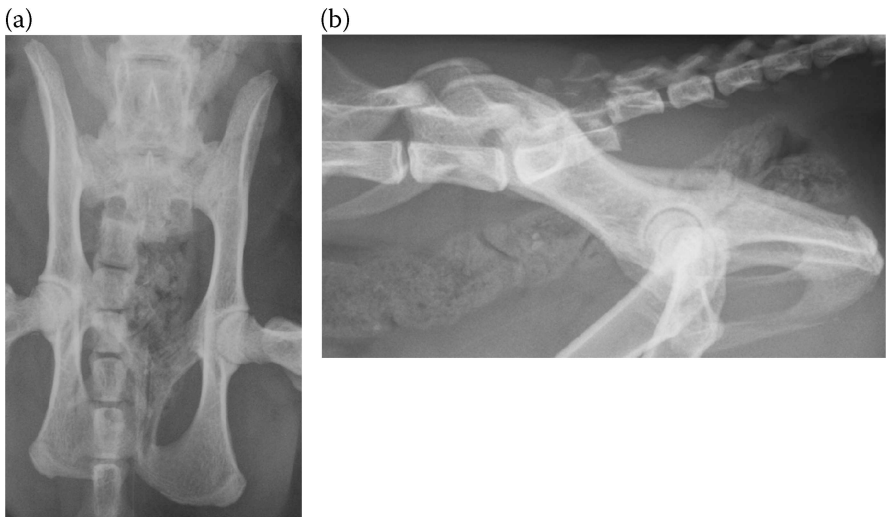


Figure 14.18 **(a)** Ventrodorsal radiograph of the pelvis of a cat with a type III (transverse) sacral fracture and right sacroiliac luxation. **(b)** Lateral radiograph of the same cat clearly demonstrating the type III sacral fracture.

tube to prevent excessive bladder distension, which may cause irreversible damage to the detrusor muscle.

Sacrocaudal Fracture/Luxation

Management of the bladder in cats with sacrocaudal injury is extremely important and although some cats will have a flaccid bladder that is easy to express this is not always the case. A proportion of cats will have an enlarged bladder that is difficult to express and other cats will be temperamentally unsuited to having their bladder manually evacuated. The bladder may be more difficult to express in cats than dogs because the internal sphincter, which is innervated by the hypogastric nerve, is more pronounced in the cat. Innervation of the internal sphincter is preserved because the hypogastric nerve originates from the lumbar spinal cord segments. Pharmacologic therapy in cats that are difficult to express involves the use of drugs such as phenoxybenzamine (0.25–0.5 mg/kg PO BID) or prazosin (0.25–0.5 mg PO BID) to reduce tone in the internal sphincter and bethanecol 1.25–2.5 mg/cat to promote detrusor muscle contraction.

Surgical treatment of cats with sacrocaudal fracture/luxation is controversial, with some authorities advocating immediate tail amputation or surgical repair to reduce pain and eliminate further traction on the nerve roots of the cauda equina. Surgical options include repair of the osseous injury using pins or wires or decompression of the cauda equina in conjunction with amputation or stabilization of the vertebrae. None of these has so far been conclusively demonstrated to influence the outcome, although stabilization will provide early pain relief. A simple method of surgical stabilization involves the use of an internal sling of polypropylene or PDS suture material placed using a dorsal approach (Figure 14.19). The suture (3.5 Metric) is passed through a hole drilled in the second dorsal spinous process of the sacrum and is then passed around the transverse processes of the luxated vertebra (usually Cd1).

Dorsal laminectomy, in an attempt to visualize and decompress the sacral nerve roots, has been advocated for treatment of fractures of the sacrocaudal region. Decompressive surgery allows visualization of the cauda equina,

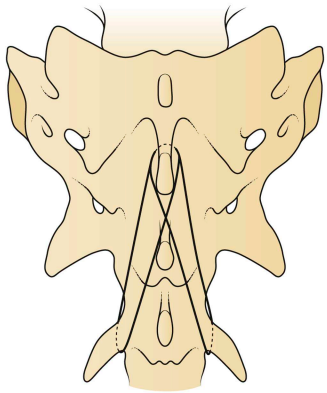


Figure 14.19 Repair of sacrocaudal fracture/luxation. Dorsal view showing internal fixation using polypropylene or PDS suture material as a sling. The suture is passed through a hole drilled in the base of the second dorsal spinous process of the sacrum and is then passed around the transverse processes of the luxated vertebra (Cd1 in this example).

which may help in assessing the prognosis but does not have any therapeutic benefit for cats with traction injury of the nerve roots.

Prognosis for sacrocaudal fracture/luxation

The most serious neurological deficit is urinary incontinence because failure to recover usually leads to euthanasia. As recovery of continence can be prolonged it would be extremely useful if there were a way of determining at an early stage which cats are likely to recover function. There are three studies that have attempted to correlate the clinical signs at the time of presentation with the eventual outcome.

In the first study, a series of 51 cases with fracture/luxation of the sacrocaudal region, cats were assessed on the basis of four different parameters (tail function, urethral tone, residual urine and anal tone/perineal sensation). The prognosis for return of urinary function in those cats with intact perineal sensation and anal tone was favourable (75–100% recovered). The recovery rate for return of urinary function within 30 days in the cats in this study that had more severe neurological deficits and lacked perineal sensation was around 50%. The average time taken for return of urinary function in this study was 13 days (range 2–30 days). It is noteworthy that tail motor function could take several months or much longer to show improvement. Cats in this series that did not recover urinary function within 4 weeks of the injury remained incontinent during a 2–36 month follow-up period. In light of this study the prognosis for cats that do not recover bladder function within 4 weeks would appear to be grave, notwithstanding the authors have seen cats that have recovered urinary function after several months. An insufficient number of cats were treated surgically to permit comparison between the results of surgical and medical treatment.

In a second study of 21 cats, presence or absence of tail base sensation was used as a prognostic indicator. In this study, all 11 cats that had intact tail base sensation showed control of urination within three days (mean 1.9 days). However, the same study showed that lack of tail base sensation does not preclude recovery with 60% (6 out of 10) of these cats regaining the ability to urinate within 7 days (mean 2.6 days).

In the most recent study of 71 cats, those with more severe neurological dysfunction on presentation had a decreased likelihood and longer duration of regaining urinary function. Cats had to be given at least 30 days to regain urinary function to be included in the study. Fifty-five of 61 cats (90%) that had urinary incontinence on presentation, regained voluntary urinary function. No significant associations were found between the following findings at the time of diagnosis and survival: urinary outcome and age, sex, anal tone, perineal sensation, tail base sensation, degree of craniocaudal or dorsoventral sacrocaudal displacement, concurrent orthopaedic injury, tail amputation and defaecatory function. Although almost 90% of the cats that regained urinary continence did so in the first 30 days, more than 10% did so between 31 and 52 days after diagnosis. Cats that did not regain faecal continence had shorter survival times than those that did not recover defecatory function.

Overall the prognosis for recovery of tail function for cats with sacrocaudal fracture/luxation is fair to good with conservative treatment, especially for cases with less neurological impairment, so immediate tail amputation is difficult to justify. However, there may be ongoing instability at the site of the injury and in one study up to 50% of cases had continuing hyperaesthesia at the tail base. In

the same study a comparison of surgical and medical management of the tail injury showed that surgical stabilization resulted in recovery of tail function in a greater proportion of cats with fewer cases having chronic tail base pain. The study did not show any improvement in urinary function between the two groups of cats. Amputation of the tail is indicated for those cats with permanent loss of motor and sensory function to prevent further injury to the tail. The authors advocate early tail amputation if there has been severe trauma to the tail itself or if there is gross displacement of the vertebrae on radiographs, on the grounds that complete avulsion of the caudal nerve roots is likely to have occurred. Hindlimb neurological dysfunction, sometimes seen in these cases, is usually the result of neuropraxia and generally resolves completely within 2–4 weeks.

In conclusion, for cats that have urinary incontinence at the time of presentation increasing severity of neurological dysfunction is associated with a longer duration of recovery of urinary function and poorer eventual outcome. The finding of intact deep pain perception predicts that recovery of control of urination within 30 days of the initial injury is very likely. For those cats with absent tail base sensation the advice given to owners should be to wait at least for 4 weeks for the return of bladder function as many of these cats will recover. Recovery of function after this period of time can occur, albeit less commonly and the authors prefer to wait longer if possible. The prognosis is very good if tail base sensation is present but the absence of tail base and/or perineal sensation for more than 2 weeks particularly when this is associated with a flaccid, areflexic anal sphincter is a poor prognostic indicator. Persistent faecal incontinence occurs in a proportion of cats and is associated with reduced survival time.

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15 The Spine – Other Disorders

INVESTIGATION OF SPINAL DISEASE:

(Bradshaw et al. 2004, Mella et al. 2019, Negrin et al. 2009).

Introduction

Investigation of spinal disease in any species represents a significant diagnostic challenge for the general practitioner. This is especially true of the cat, in which spinal disease in general is uncommon in comparison with the dog. Symptomatic intervertebral disc disease (IVDD) in particular is a relatively rare occurrence in this species. This may appear to be good news for the cat, but the consequences for the veterinarian are unfamiliarity with the procedures for diagnosis and investigation of spinal diseases when they do occur. There are few studies of the relative frequency of the different causes of spinal disease, and there is a lack of consensus on the optimum methods of management for many disorders. Spinal disease should be suspected in any cat presented with spinal hyperaesthesia and ataxia, paresis or paralysis. Neuromuscular diseases must also be considered if there is more generalized paresis or paralysis with all four limbs affected.

Signalment and History

The signalment is important in the initial assessment of any neurological disease but this information can be misleading and should be used with care. Breed predispositions are less important in the cat than the dog since pedigree cats are less common than their canine counterparts. In a recent study of 221 cats referred for spinal disease, purebred cats were significantly more likely to have a diagnosis of degenerative IVDD and thoracic vertebral canal stenosis whereas nonpurebred cats were more likely to have presumptive non-lymphoid neoplasia. In general, traumatic injury and infectious disorders such as FIP are more prevalent in young cats, whereas degenerative and neoplastic conditions tend to increase in frequency with increasing age. Older cats are more likely to have neoplasia, ischaemic myelopathy and lumbosacral stenosis. However, there are exceptions; for example, spinal lymphoma, which is more prevalent in young cats, and IVDD which is more likely to be seen in middle-aged cats.

A full history is an important prerequisite to the neurological examination and will provide information about the onset, duration and progression of the disease in addition to antecedent disease and the response to previous therapy. For example, cats with FIP virus myelitis, lymphoma and spinal empyema are more likely to have a history of concurrent clinical signs, such as lethargy, anorexia and weight loss, whereas cats with degenerative IVDD are less likely to have concurrent clinical signs. Peracute onset (<24 hours) of clinical signs is most likely to be associated with vertebral fracture and luxation, and ischaemic myelopathy whereas an acute to subacute onset (1–5 days) of signs is more consistent with degenerative IVDD. Progressive clinical signs are suggestive of a diagnosis of non-lymphoid neoplasia or degenerative IVDD, whereas static or improving clinical signs will be seen with ischaemic myelopathy.

Physical Examination

A carefully performed routine physical examination should provide information about relevant concurrent or intercurrent disease and will assist in ruling out a number of disorders that may mimic spinal disease. Cats with FIP virus myelitis, lymphoma and spinal empyema are more likely to have abnormal physical examination findings, such as pyrexia or lymphadenomegaly. In practice it is easier to perform a screening neurological examination as part of this routine physical examination. If there are concurrent signs referable to the head, such as altered consciousness, seizures, or cranial nerve deficits, then the disease is diffuse or multifocal in origin involving both the brain and the spinal cord.

Observation of gait, posture and general demeanour should precede the hands-on part of the examination. The patient should be allowed to relax and should be gently coaxed to move around the examination room, unless a traumatic spinal injury is suspected, in which case movement should be restricted. This part of the examination is particularly relevant to the cat, as latter parts of the examination may be more difficult to perform.

Gentle palpation of the vertebral column should be performed to detect the presence of swelling, atrophy, masses or spinal deformity. Palpation and manipulation of the vertebral column should not be performed on cats with suspected traumatic spinal injuries. If hyperaesthesia is detected on gentle palpation this part of the examination should be performed last to avoid losing the cooperation of the patient. The thoracolumbar spine should be palpated dorsally and the cervical spine laterally to check for hyperaesthesia. Palpation of the dorsal paraspinal musculature is performed by placing the index finger and the middle finger of the dominant hand on the epaxial muscle either side of the midline. If there is no response or an equivocal response to initial light palpation, firmer pressure is applied and the examination is repeated. Deep palpation should begin away from any potentially painful areas. Palpation is followed by manipulation of the cervical vertebral column and tail. These areas are slowly and gently put through a range of motion dorsally, ventrally and laterally checking for resistance or evidence of a response to pain from the cat. The lumbosacral spine can be placed in extension by lifting the hindquarters and gently extending both hindlimbs simultaneously.

Cats with spinal hyperaesthesia are more likely to have degenerative IVDD, spinal fracture and luxation, or thoracic vertebral canal stenosis. An absence of spinal hyperaesthesia is more consistent with a diagnosis of ischaemic myelopathy. It is also important to check the colour of the footpads and the quality of the femoral pulses in any cat with acute paraparesis or paraplegia to rule out ischaemic neuromyopathy.

Neurological Examination

(Garosi 2009, Taylor & Kerwin 2017).

If neurological abnormality is suspected during the initial physical examination, a more thorough neurological examination is performed with the goal of establishing whether spinal disease is present, and if it is, to determine its location and assess its severity. Performing a neurological examination on any patient can seem daunting for the novice. Additional difficulties include the potentially uncooperative nature of the feline patient and the fact that some of the tests

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used in the dog are either not applicable to the cat or must be interpreted with caution. It is essential that the clinician develops familiarity with the responses of normal cats by regular practice before drawing conclusions about the altered responses seen in clinical cases. Patients must not be sedated or anaesthetized prior to neurological examination since this will alter the responses seen to many of the stimuli. Descriptions of how to perform a full neurological examination are contained in standard neurology texts and only those aspects of the examination that are pertinent to feline spinal disorders will be discussed here. As most cats are not tolerant of prolonged examination as much information as possible should be gleaned from the hands-off part of the examination, and rather than performing a large barrage of tests that are likely to result in loss of patient compliance, the hands-on part of the examination should be targeted to a few key tests. If a methodical approach is followed this will make the clinical syndromes in cats with neurological dysfunction caused by spinal disease easier to recognize.

The hands-off part of the neurologic examination is an assessment of gait and posture. The three abnormalities that should be assessed during gait analysis are lameness, weakness or paresis/plegia, and incoordination or ataxia. Gait analysis in cats is challenging because when a cat is fearful or anxious it will stay as close to the ground as possible and will either refuse to move or will attempt to hide. The cat should preferably be allowed to leave its carrier voluntarily and should initially be observed moving around the consulting room as it explores its surroundings. A number of techniques can be used to encourage ambulation including the use of laser pointers, carrying the cat's basket around the room or placing it on the opposite side of the room. In selected cases movement can be assessed by placing the cat on a low stool or chair as most cats will jump down and walk away. A useful way to supplement this examination if a cat is uncooperative is to suggest that the client makes a video of the cat in the home environment.

The hands-on part of the examination should be performed with the patient initially in the upright position. Proprioceptive (paw) positioning is unreliable and difficult to interpret in the cat as most cats do not tolerate handling of their paws. The preferred postural reaction tests for cats are hopping, placing (visual/tactile), extensor postural thrust and if tolerated hemi-walking and wheelbarrowing. Cats are often reluctant to hop, in which case the patient should be held with three limbs restrained, and lowered suddenly to a non-slip surface with the limb to be tested extended. As soon as the paw strikes the surface the cat should be moved laterally to force it to hop on the limb. Cats should be hopped or hemi-walked away from their centre of gravity and the two sides compared as a finding of asymmetry is often significant. The cutaneous trunci (formerly panniculus) reflex should be elicited by lightly plucking guard hairs along the dorsum either side of the spine.

Once the cat has become more accustomed to being handled, the remainder of the tests can be performed with the patient in lateral recumbency if this is tolerated by the cat. Alternatively spinal reflex examination can be performed with the cat positioned in dorsal recumbency between the thighs of the examiner. The examiner should sit on the floor with their back against a wall and with the knees flexed. The cat is placed with its back resting on the thighs and facing towards the examiner. Most cats find this position comfortable, the stifles are naturally slightly flexed and opposite sides can be compared easily. The most reliable spinal reflexes in the cat are the withdrawal (flexor) reflex and the patellar reflex.

Neuroanatomical Localization of Spinal Cord Lesions

(Tudbury et al. 2017).

Spinal cord lesions display a range of signs based on their location, lateralization and severity. The key to lesion localization is to identify both the spinal cord functions that are disrupted and just as importantly those that are still normal. An attempt should be made to determine whether all of the clinical signs can be explained by a focal lesion at one site in the spine. Focal lesions are the commonest type of spinal disorder seen in the cat. If the clinical signs cannot be explained by a single focal lesion then the disorder may be diffuse or multifocal. Cats with ischaemic myelopathy are more likely to have a lesion localized to the caudal cervical spine, while cats with FIP are most likely to have a multifocal neuroanatomical localization with possible intracranial involvement. Lateralization of clinical signs occurs when a lesion predominantly affects one side of the spinal cord. Cats with ischaemic myelopathy or thoracic vertebral canal stenosis are more likely to have lateralization of their clinical signs.

For focal lesions the aim should be to localize the lesion to one of five functional regions of the spinal cord on the basis of the neurological examination. These regions are cervical (C1–C5), cervicothoracic (C6–T2), thoracolumbar (T3–L3), lumbosacral (L4–S3) and sacral (S1–S3) (Figure 15.1). The cervical and thoracolumbar regions of the spinal cord convey the upper motor neurons whereas those of the cervicothoracic and lumbosacral regions also contain the cell bodies of the lower motor neurons to the forelimbs and hindlimbs, respectively. Lesions in these four different regions will produce different permutations of neurological signs (Table 15.1). The presence of spinal hyperaesthesia

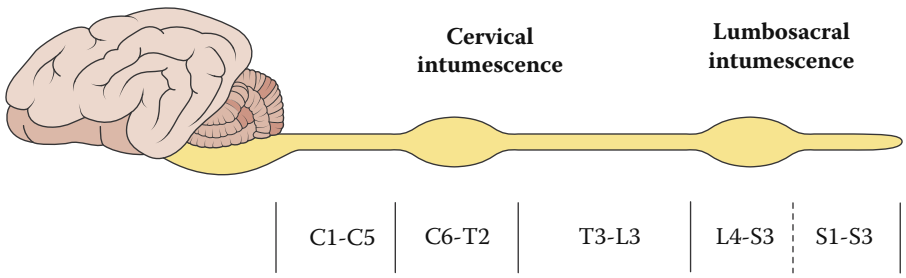


Figure 15.1 The four functional regions of the spinal cord: cervical, cervicothoracic, thoracolumbar and lumbosacral.

Table 15.1: The Expected Reflex Response to Spinal Cord Lesions

Lesion Location*	Forelimb	Hindlimb	Bladder, Anus and Tail
C1–C5	UMN	UMN	UMN
C6–T2	LMN	UMN	UMN
T3–L3	Normal	UMN	UMN
L4–S3	Normal	LMN	LMN
S1–S3	Normal	Normal	LMN

Notes
* Based on spinal cord segments rather than vertebrae
UMN = upper (central) motor neuron
LMN = lower (peripheral) motor neuron.

may help to confirm or refine anatomical localization based on the results of neurological examination, but hyperaesthesia on its own should not be relied on to localize a lesion in a cat with neurological dysfunction.

The tests used to localize neurological signs include postural reactions and assessment of reflexes (Table 15.2). Postural reactions are sensitive but relatively nonspecific indicators of neurological dysfunction. A positive test merely indicates loss of integrity of either the lower motor neurons of the local reflex arc or the upper motor neuron tracts to the brain. Assessment of withdrawal and myotatic (tendon stretch) reflexes is used to localize a lesion to the area of the spinal reflex tested. The segmental cutaneous trunci reflex is unreliable in the cat and it is therefore important not to ascribe too much significance to its absence since it cannot be elicited in some normal individuals. These segmental tests do not rely on the integrity of pathways to the higher centres and these reflexes will be intact even if portions of the spinal cord cranial to the reflex arc are completely destroyed.

The patellar reflex, which is used to assess the femoral nerve and spinal cord segments L4, L5 and L6, is the only reliable myotatic reflex, and can generally be assessed accurately in most cats. The cranial tibial and extensor carpi radialis reflexes should not be relied on for neurological examination, as they are independent of the reflex arc, and are therefore not strictly myotatic reflexes. Myotatic reflexes are graded on the basis of the strength of the motor response. Lesions that affect the lower motor neurons of the reflex arc cause depression or absence of the reflex tested. This type of neurological dysfunction is referred to as lower (or peripheral) motor neuron or flaccid paresis or paralysis since the tone in the affected muscles is reduced. It is important to note that the finding of lower motor neuron deficits in all four limbs is usually indicative of neuromuscular disease rather than spinal disease. Lesions cranial to the segmental site of the reflex arc spare the spinal reflex and the motor response may be increased because of the loss of the inhibitory influence of the upper motor neurons. The neurological dysfunction in these cases is referred to as upper (central) motor neuron, or spastic paresis or paralysis, since the tone in the affected muscles is increased. However, the distinction between hyperactive and normal responses is not clear-cut and caution should be exercised in the interpretation of an apparently hyperactive response.

The flexor reflex is elicited by pinching the interdigital skin using fingers or a haemostat and may be tested at the same time as nociception, recognizing that withdrawal of the limb and nociception are two different phenomena. A positive response is shown by withdrawal of the limb accompanied by flexion of all of the joints in the tested leg. The reflex indicates functional spinal cord segments at this level but does not require conscious perception of noxious stimuli. The crossed extensor reflex may also be observed when testing the flexor reflex. A positive response is seen when the contralateral limb rapidly extends as the tested limb flexes. This abnormal reflex is seen in most cats that have a severe lesion at a site cranial to the segments involved in the flexor reflex. Assessment of the perineal reflex is accomplished by stimulating the left and right perineum. The normal reaction is bilateral contraction of the anal sphincter and flexion of the tail. Bladder function should be assessed when urinary retention or incontinence is suspected by attempted manual evacuation. Increased urethral sphincter tone is associated with lesions involving the upper motor neuron supply to the bladder. There will be urinary retention with overflow and manual evacuation of the bladder will be difficult. Loss of urethral sphincter tone is associated with lower motor neuron lesions affecting the sacral spinal cord segments or nerve roots. The bladder will be large and flaccid, with little resistance to manual evacuation.

Table 15.2: Commonly Assessed Spinal Reflexes

Site	Reflex	Spinal Cord Segments
Forelimb	Flexor (withdrawal)	C6–T2
Hindlimb	Patellar	L4–L6
Hindlimb	Flexor (withdrawal)	L6–S1
Anus	Perineal	S1–Cd1
Bladder	Sphincter tone	S1–S3
Dorsum	Cutaneous trunci (panniculus)	T3–L1 (afferent) C8–T1 (efferent)

Assessment of Severity and Prognostic Indicators

(Gonçalves et al. 2009, Jeffery et al. 2016).

The severity of a lesion is inferred from the degree of neurological dysfunction and has a bearing on the diagnosis and prognosis. Cats that are non-ambulatory at the time of presentation are more likely to have a spinal fracture and luxation or acute non-compressive nucleus pulposus extrusion. Cases are assigned to a grade based on the results of the neurological examination. The specific criteria used to assign patients to a grade are spinal pain, unassisted walking ability, voluntary limb movement, and perception of pain or more correctly nociception. The grading system used by the authors is derived from the Frankel scale. Cats are assigned to a neurological grade based on the results of the neurological examination as follows:

- Grade 1 myelopathy: Spinal hyperaesthesia with no evidence of neurologic dysfunction.
- Grade 2 myelopathy: Ataxia and ambulatory paresis (monoparesis, paraparesis or tetraparesis).
- Grade 3 myelopathy: Non-ambulatory paresis (paraparesis or tetraparesis).
- Grade 4 myelopathy: Plegia (paraplegia or tetraplegia).
- Grade 5 myelopathy: Plegia (paraplegia) and absence of nociception (pain perception).
- Grade 6: Ascending/descending myelomalacia.

Nociception is dependent on functional ascending tracts in the spinal cord and a positive response is shown by a conscious reaction to the stimulus. Positive responses include turning the head in recognition of the noxious stimulus, attempting to scratch or bite the examiner, or an autonomic effect, such as dilatation of pupils or increased respiratory or heart rate. The small nonmyelinated nerve axons that carry nociception are more resistant to the effects of distortion than other nerve fibres. They are also numerous and diffusely distributed in multisynaptic tracts adjacent to the spinal cord grey matter. Therefore, nociception is the last modality to be lost and its absence is a bad prognostic sign. If there is a behavioural response to pinching the interdigital skin with the fingers then no further stimulus is required. However, if there is no response, because nociception is such a key prognostic indicator, it is important to apply a maximal stimulus such as application of haemostats to the distal limb (taking care not to crush the tissues), before consigning a patient to the grade 5 category. It is also important to assess the presence or absence of voluntary bladder function since this will affect the management of the case and may have an influence on the prognosis.

It is important to be aware of the phenomenon of spinal shock when assessing cats with acute severe spinal cord injury. Cats with spinal shock suffer a loss of muscle tone and reflexes caudal to the injury irrespective of the localization. Failure to recognize the discrepancy between spinal reflexes and lesion location could lead to erroneous clinical localization, inappropriate use of diagnostic tests and inaccurate assessment of the prognosis. Signs of spinal shock disappear within the first 12–48 hours after the initial injury. In such cases the cutaneous trunci reflex although not completely reliable in cats can be helpful if the lesion is between segments T3–L3. The presence of spinal shock should not dissuade the clinician from pursuing appropriate investigation and treatment.

In some cases with severe neurological signs where there is an absence of nociception, the prognosis may be so bad that further investigation to determine the aetiology may be deemed unnecessary. In dogs with intervertebral disc extrusion and absent nociception, some studies have shown that the prognosis is better following decompressive surgery if the onset of signs was gradual. Duration of loss of nociception prior to decompressive surgery as a prognostic factor is more controversial, and although it is commonly accepted that surgery should be performed as soon as possible, a recent prospective study in dogs found that the immediacy of surgical treatment had no apparent association with outcome. There is no specific information available in the cat either regarding the rate of onset of signs or the duration of loss of nociception prior to surgery, but the situation is likely to be similar to that of the dog.

In general, the prognosis worsens as the severity of neurological dysfunction increases and is worse for individuals displaying lower motor neuron rather than upper motor neuron deficits. The nature of the injury, and the duration and rate of onset of the neurological signs, are also factors that may have a bearing on the prognosis. In a study involving magnetic resonance imaging (MRI) of 92 cats with clinical signs of spinal disease it was found that cats with a peracute or chronic onset of signs had a better prognosis than cats with an acute onset of signs. This is difficult to explain but may be because the cats with an acute onset of signs were more likely to have conditions that were less amenable to successful treatment such as neoplasia. The group of cats with a peracute onset of signs were more likely to have traumatic or vascular aetiologies; pathologies that often have a better prognosis for recovery, whereas the cats with chronic signs were more likely to have slowly progressive or non-progressive disease that did not prompt the owner to request euthanasia.

Cervical and Cervicothoracic Lesions

Compressive lesions affecting spinal cord segments C1 through T2 cause neurological dysfunction in both the forelimbs and hindlimbs. Differentiation between cervical (C1–5) and cervicothoracic (C6–T2) lesions is attempted primarily on the basis of whether the deficits are lower motor neuron or upper motor neuron in nature. Unfortunately this differentiation is not always clear-cut. A lesion anywhere in this region that causes irritation of the meninges and dorsal nerve roots with minimal cord compression will cause neck pain. The lesion can sometimes be localized fairly accurately by determining the site of pain on palpation.

A focal compressive lesion of spinal cord segments C1–5 will produce signs affecting all four limbs, with the extent depending on the rate of onset and severity of the lesion. With most compressive lesions that affect the cervical spinal cord the hindlimbs will show more obvious clinical signs than the forelimbs because the afferent tracts from the hindlimbs are more superficial than those from the forelimbs. A unilateral lesion will produce signs of ipsilateral hemiparesis. Cranial cervical lesions that affect the first 2 or 3 cervical spinal segments occasionally cause clinical signs of vestibular disease, presumably due to brain stem and/or spinovestibular tract injury. The spinovestibular tracts carry proprioceptive information from the first 3 cervical spinal nerves that are involved in the orientation of the head and neck.

A cervicothoracic lesion may affect the nerve roots of the brachial plexus as well as the spinal cord so that the forelimbs may appear more severely affected than the hindlimbs. This distinction is not absolute, however, as occasionally a C1–C5 spinal cord lesion will cause more obvious clinical signs in the forelimbs than the hindlimbs, a condition known as central cord syndrome. This occurs when the lesion is closer to the grey matter and spares the more superficial tracts. A unilateral lesion that impinges predominantly on one of the nerve roots comprising the brachial plexus will cause forelimb lameness known as a 'root signature' that may mimic lameness associated with orthopaedic disease. A more severe unilateral lesion affecting these nerve roots will cause monoparesis.

Progressive paresis is noted with increasing severity of spinal cord injury, although voluntary movement is initially retained. The paresis may then be classed as ambulatory or nonambulatory according to the ability of the patient to stand and walk unassisted. Hemiparesis is seen with unilateral involvement and tetraparesis with bilateral lesions. Voluntary movement ceases with more severe impairment of neurological function but nociception is retained and the patient becomes hemiplegic or tetraplegic. Spinal causes of hemiplegia are very rare and usually result from a unilateral intraparenchymal cord lesion. The most severe lesions of the cervical and cervicothoracic spinal cord will affect urinary and respiratory function and are rarely seen. Lesions above C5 affect the upper motor neurons of the phrenic and intercostal nerves. At C5–7 the lower motor neurons of the phrenic nerve are also affected and caudal to C7 the upper and lower motor neurons of the intercostal muscles are affected. A cervical lesion severe enough to cause loss of nociception would also cause death from respiratory paralysis and such cases rarely survive long enough to receive veterinary attention.

Lesions of the cervical and cervicothoracic regions can be difficult to differentiate, particularly if they are mild. The vertebral canal, especially in the caudal cervical region, is more spacious relative to the diameter of the spinal cord than elsewhere; a compressive lesion must, therefore, be larger before it impinges on the cord. The myotatic reflexes in the forelimb are unreliable, making depressed responses difficult to interpret. The withdrawal reflex is easy to test for, and is reliable, but it has low sensitivity and specificity. It is derived from five spinal cord segments and nerve roots (C6–T2) extending over four vertebral bodies (C5–T1), so it takes a severe lesion spread over multiple vertebral bodies to depress or abolish this reflex.

Horner syndrome is caused by lesions affecting the sympathetic innervation of the orbit and eye. The second order axons exit the spinal cord with the T1 to T3 ventral nerve roots. Cats with Horner syndrome display a combination of miosis, ptosis, enophthalmus and protrusion of the third eyelid on the same side as the lesion. Horner syndrome, associated with spinal cord disorders, is usually only recognized with an acute asymmetrical cord lesion between C1 and T2 that is severe enough to cause tetraparesis or tetraplegia. The main causes in cats are ischaemic myelopathy and vertebral fractures and luxations. Horner syndrome is more commonly seen with a severe lesion of the brachial plexus, such as traumatic avulsion of the ventral roots of T1 and T2 adjacent to the spinal cord, or neoplasia involving the ventral roots in combination with ipsilateral monoparesis. A lesion involving the nerve roots C8 and T1 will cause loss of the ipsilateral cutaneous trunci reflex as these spinal cord segments innervate the cutaneous trunci muscle via the lateral thoracic nerve.

Thoracolumbar and Lumbosacral Lesions

(Kot et al. 1994, Shkorbatova et al. 2019).

Compressive lesions of spinal cord segments T3 through S3 will cause neurological dysfunction in the hindlimbs with normal forelimbs. Differentiation between thoracolumbar (T3–L3) and lumbosacral (L4–S3) lesions is attempted primarily on the basis of whether the deficits are upper motor neuron or lower motor neuron in nature. This is generally easy to accomplish by evaluating the hindlimbs reflexes and assessing bladder and bowel function. A mild focal compressive lesion in these regions, which only irritates the meninges and dorsal nerve roots with minimal spinal cord compression and/or gradual onset, will result in spinal hyperaesthesia. Localization is based on evidence of pain or increased sensitivity on paravertebral muscle palpation.

Mild spinal cord compression in the thoracolumbar region results in ataxia as a consequence of general proprioceptive deficits in the hindlimbs. There is progressive hindlimb weakness or paraparesis with increasingly severe and/or acute lesions and, eventually, there is difficulty in supporting body weight, although voluntary movement is retained. Paraparesis progresses to paraplegia with more severe lesions, as voluntary movement is abolished. Neurogenic urinary incontinence usually develops at this stage, as the bladder fills and then overflows. Palpation characteristically reveals a turgid bladder that is difficult to express because urethral sphincter tone is increased. Hindlimbs nociception is only abolished when the lesion is very severe and/or extensive.

The Schiff–Sherrington phenomenon, commonly seen in acute severe lesions of the thoracolumbar spine in the dog, is rare in the cat. In affected patients the forelimbs become rigidly extended when the patient is placed in lateral recumbency. The phenomenon is caused by disinhibition of the extensor motor neurons in the cervical intumescence as a result of a sudden interruption of a pathway that originates in the grey matter of the L1 to L5 spinal cord segments. Postural reactions and reflexes will be intact, despite the increased extensor tone in the forelimbs, although they may be hyperactive. Spinal cord integrity has been maintained if deep pain perception is present in the hindlimbs. The reason for the species difference in the frequency of this phenomenon are not understood.

Lesions involving the lumbosacral segments are localized by the alterations in the spinal reflexes seen on the neurological examination. The spinal cord is foreshortened relative to the vertebral column so that segments L4–Cd5 are housed in vertebrae L4–S1; but, as already mentioned, the disparity is less than that seen in the dog. There is some individual and breed variation but typically spinal cord segment L4 is located in the corresponding vertebral body, whereas segment L5 is positioned over the L4–L5 intervertebral disc, segments L6, L7 and S1 are within or caudal to the L5 vertebral body, and segments S2 and S3 are within or caudal to the L6 vertebral body. The nerve roots from these segments run caudally in the vertebral canal and some of them form the cauda equina before exiting caudal to the vertebra of the same annotation. There is no precise anatomical definition of the cauda equina but the commonly accepted view is that it includes the L7, sacral and caudal nerves. The nerve roots are vulnerable to injury at several

sites but there is more space in the lumbosacral region of the vertebral canal and nerve roots are more tolerant of deformation than the spinal cord. Nevertheless, recovery is unlikely if there is severe damage. Spinal cord segments and nerve roots S1–S3 contribute to the pelvic nerve, which supplies sensory and motor innervation to the bladder, descending colon and rectum. The same segments and nerve roots also contribute to the pudendal nerve, which transmits sensory information from the external urethral sphincter, anal sphincter and perineal region, and provides motor innervation to the external urethral sphincter and the anal sphincter.

Mild compressive lesions between L4–S3 may cause pain and depress reflexes in the hindlimbs, anus, or tail. More severe lesions affecting these segments may cause paraparesis or paraplegia, with a reduced or absent patellar reflex if segments L4–6 are involved. Tests for sacral spinal cord segments and their associated nerves rely on sensory stimuli applied to the various regions supplied by these nerves and motor responses originating in the sacral spinal cord. A lesion between segments S1–3 will spare the hindlimbs but there will be no reflex urination or defecation. The so-called lower motor neuron bladder is overdistended, flaccid and is generally easy to express manually. The anal sphincter will be dilated and the tail atonic. There will be some autonomous function because of the intrinsic ability of smooth muscle to contract but this is usually not effective in emptying the bladder. Caudal vertebral lesions may produce only an atonic tail with normal urination, hindlimb function and defaecation.

Differential Diagnosis

Following neuroanatomical localization to one of the four areas of the spinal cord on the basis of the neurological examination, a differential diagnosis list should be drawn up embracing all of the disease conditions that may be present at that location. It is helpful to formulate this checklist using the mnemonic VITAMIN D (Table 15.3). Key

Table 15.3: Differential Diagnosis of Spinal Cord Lesions

Disease Category		Differential Diagnosis (Examples)
V	Vascular	Fibrocartilaginous embolic myelopathy (FCEM)
		Ischaemic myelopathy of the cranial cervical spinal cord
I	Inflammatory/Infectious	Feline infectious peritonitis (FIP)
		Toxoplasmosis
		Cryptococcosis
		Discospondylitis
		Bacterial myelitis/meningomyelitis
		Bacterial epidural empyema
		Immune mediated (FIP? FIV? FeLV?)
T	Traumatic	Vertebral fracture/luxation
		Acute non-compressive nucleus pulposus extrusion (ANNPE)
A	Anomalous (congenital)	Atlantoaxial subluxation
		Sacrocaudal dysgenesis/agenesis
		Incomplete ossification of cervical vertebrae
		Spina bifida
		Miscellaneous vertebral malformation
		Arachnoid diverticulum
		Syringomyelia and hydromyelia
		Vertebral angiomatosis
		Dermoid sinus
		Thoracic vertebral canal stenosis
M	Metabolic	Lysosomal storage disease
I	Iatrogenic	Incorrect/contaminated spinal puncture!
	Idiopathic	The aetiology of many disorders is not fully understood
N	Neoplastic	Lymphoid spinal tumour
		Non-lymphoid spinal tumour
		Vertebral tumour
	Nutritional	Hypervitaminosis A
D	Degenerative	Intervertebral disc disease (IVDD)
		Thoracic vertebral canal stenosis
		Degenerative lumbosacral stenosis (DLSS)
		Spondylosis deformans
		Diffuse idiopathic skeletal hyperostosis (DISH)
		Dural ossification
		Degenerative myelopathy

Table 15.4: Differential Diagnosis Based on Rate of Onset and Age

Acute		Chronic	
Immature*	**Adult**	**Immature**	**Adult**
Fracture/luxation	Fracture/luxation	Lymphoma	Lymphoma
Bacterial myelitis	Bacterial myelitis	FIP	Nonlymphoid tumours
Epidural empyema	Epidural empyema	Toxoplasmosis	Toxoplasmosis
FCEM	FCEM	Discopsondylitis	Discospondylitis
ANNPE	ANNPE	FeLV/FIV	FeLV/FIV
Anomalous	IVDD	Anomalous	Spondylosis deformans
		Lysosomal storage	DLSS

Notes
* = <2 years of age
ANNPE = acute non-compressive nucleus pulposus extrusion
IVDD = intervertebral disc disease
FCEM = fibrocartilaginous embolic myelopathy
DLSS = degenerative lumbosacral stenosis

features from the signalment, the history and the findings from the physical and neurological examination can be used to make a short-list of differential diagnoses ranked in order of probability (Table 15.4). Once this has been done then both the clinician and the owner will be better placed to make decisions regarding further investigation and treatment and possible referral to a specialist centre.

Diagnostic Tests

The cause of some spinal disorders may be apparent on the basis of the signalment, history and the findings of the physical and neurological examinations but, in most cases, further investigation will be required to achieve an accurate diagnosis and prognosis. A minimum database would include a complete blood count, serum biochemistry profile, urinalysis and tests for feline leukaemia virus (FeLV) and feline immunodeficiency virus (FIV). With the exception of inflammatory disease, spinal cord disorders rarely cause alterations in biochemistry, haematology and urinalysis results. However, these tests may shed some light on the cause of the spinal cord dysfunction or identify significant intercurrent disease that may have a bearing on the management of the case. Hypergammaglobulinaemia and neutrophilia may be associated with infection or neoplasia. If an inflammatory disease is suspected, serology or polymerase chain reaction (PCR) testing should be performed for Toxoplasma, Cryptococcus spp. and feline coronavirus (FIP). Additional diagnostic investigations include radiography, CSF analysis and advanced imaging techniques. These latter investigations are generally performed if the cat is showing signs of moderate to severe or progressive neurological dysfunction or spinal pain that is not responding to conservative management.

Radiography

Conventional radiography may be diagnostic with some conditions, such as spinal trauma, vertebral neoplasia and discospondylitis but there are severe limitations in the investigation of many spinal disorders. For example in the case of intervertebral disc disease, advanced imaging will be required to assess the significance of changes seen on radiographs and to identify the site of the lesion prior to surgical intervention. Radiographic changes may be subtle and good quality radiographs should be obtained in two orthogonal planes to allow full assessment. This requires general anaesthesia, unless contraindicated because of concurrent injury in the trauma patient or because spinal fracture/luxation is suspected. Radiographs should be obtained of the area of interest following localization by neurological examination. In some cases it may be necessary to obtain survey radiographs of the complete spine to check for multiple lesions; for example, where the neurological signs indicate a multifocal disease or in cats with spinal trauma. In this case two or three radiographs should be obtained centred on different regions of the spine; for example, lumbosacral, thoracolumbar and cervicothoracic junctions. The temptation to obtain a single survey radiograph of the whole spine should be resisted.

Advanced imaging is often indicated after performing plain radiography for the following reasons:

- To identify a spinal lesion not visible on plain radiographs.
- To determine if spinal cord compression is present following identification of a lesion on plain radiographs.
- To determine the relative significance of multiple lesions identified on plain radiographs.
- To assist with planning the type of procedure to be performed.

In those cases that are likely to be referred for advanced multiplanar imaging it may be best not to obtain plain radiographs to avoid repeat general anaesthesia, reduce costs for the owner and prevent unnecessary delay in referral.

Myelography

(Nykamp & Scrivani 2001, Pardo & Morgan 1988, Suess et al. 1991, Wheeler et al. 1985).

Myelography is the injection of positive contrast medium into the subarachnoid space. In the past myelography was an essential tool in the investigation of many feline spinal diseases but is now rarely performed except in combination with computed tomography (CT). The procedure involves injection of a contrast medium either at the cisterna magna (cerebellomedullary cistern) or at L6–7 into the lumbar cistern, using a 22 gauge 38 mm spinal needle. Iohexol, at a concentration of 240–300 mg/ml of iodine (Omnipaque, GE Healthcare), is the contrast agent of choice, and is

administered at a dose rate of 0.2–0.5 ml/kg depending on the site of injection and the suspected location of the lesion. After myelography, patients should be recovered in sternal recumbency with the head raised to promote the flow of contrast away from the brain. Postmyelographic seizures are controlled by administration of midazolam.

In general, cisternal injection of contrast medium is performed for cervical and lumbosacral lesions and lumbar injection of contrast medium for thoracolumbar lesions. The advantages of cisternal puncture are that it is technically easier and it is more likely that a satisfactory CSF sample will be obtained. However, seizures are more likely to occur, the flow of contrast caudally may be problematic, and it will not be possible to outline an acute compressive lesion, particularly in the thoracolumbar region. In lumbar myelography the injection can be made under pressure, so the contrast can be made to outline an acute compressive lesion regardless of location. The disadvantages of lumbar injection are that it is technically more difficult to perform, it is frequently not possible to obtain a CSF sample, and epidural leakage of contrast is more likely to occur, making interpretation difficult. A degree of epidural leakage of contrast is inevitable and tends to make lumbar injection unsuitable for the investigation of suspected lumbosacral lesions. Injection of contrast into the subarachnoid space is contraindicated in cats where the risk of anaesthesia or spinal puncture is unacceptable and is inadvisable in cats suspected of having severe inflammatory spinal disease.

Computed Tomography

(Ricciardi et al. 2018).

Multidetector CT is increasingly becoming more available in larger general practices and is now an essential item of equipment in all specialist centres. CT uses X-rays generated by an anode rotating around the patient at high speed. Computer-generated images are produced that represent transverse sections through the patient at that level. CT is particularly useful for showing osseous rather than soft tissue detail and specific indications include spinal trauma and suspected bone neoplasia. CT is often used on its own or in combination with MRI for diagnosis and to assist with surgical planning.

The sensitivity of CT images for investigation of lesions affecting the spinal cord can be improved by myelographic enhancement. Myelography in conjunction with CT acquisition is indicated in cases where a compressive lesion is suspected but is not apparent on plain CT or CT after intravenous administration of contrast medium. Injection of contrast into the subarachnoid space is contraindicated in cats where the risk of anaesthesia or spinal puncture is unacceptable and is inadvisable in cats suspected of having severe inflammatory spinal disease.

Magnetic Resonance Imaging

(Gonçalves et al. 2009).

MRI is a computer-assisted technique that depends on the magnetic properties of atomic nuclei in body tissues. A variety of different image sequences can be used, but the principle ones are T1-weighted (T1w) and T2-weighted (T2w). Tissues with high water content, such as CSF, appear dark (hypointense) on T1w and bright (hyperintense) on T2w images. Images can be enhanced by the use of a paramagnetic contrast agent such as gadolinium. MRI is particularly useful for examining the spinal cord and nerve roots and the other non-osseous structures of the spine. High-field MRI has become an essential tool in the investigation of feline spinal disease and should be available in all specialist referral centres. MR provides superior images of the spinal cord parenchyma and nerve roots in comparison with CT and CT myelography, and has the major advantage of not requiring the administration of intrathecal contrast.

In comparison with dogs, a much larger proportion of cats with spinal disease will have no detectable abnormality on MRI. Therefore, it is especially important for clinicians and owners to understand that MRI will often not provide a definitive diagnosis on its own and is just part of the diagnostic work up for a cat with clinical signs of spinal disease. Low-field MR magnets (0.2–0.4 Tesla) have lower signal to noise ratio than high-field scanners resulting in poorer spatial resolution and longer acquisition time. When considering referral of a cat for MRI of the spine the clinician should be aware of the fact that it is difficult to obtain diagnostic studies using a low-field scanner.

A study of 92 cats with clinical signs of spinal disease identified two factors as being predictive of whether MRI findings would be abnormal; the severity of the clinical signs, and the presence of spinal pain on presentation. Cats with the most severe clinical signs were eight times more likely to have an abnormal MRI than cats with mild clinical signs, and cats with spinal pain at the time of presentation were four times more likely to have an abnormal MRI compared to cats with no spinal pain. In the same study lesions were identified in 60 cats on MRI, the majority of which were extradural, and the spinal cord segments most frequently affected were T3–L3. In 32 cats MRI was unremarkable and although a final diagnosis was achieved in three of these as a result of further diagnostic tests there was no presumptive diagnosis in the remaining 29 cats. The group of cats in which no abnormalities were detected on MRI had a better prognosis than those cats in which a lesion was found.

Cerebrospinal Fluid Analysis

(Bienzle et al. 2000, Bray et al. 2016, Chrisman 1992, Singh et al. 2005).

CSF examination is a useful diagnostic tool in the investigation of feline spinal disease but it does have limitations. Abnormal CSF is a strong indicator of spinal cord or nerve root inflammatory disease but no other type of spinal disease consistently produces any change in the CSF.

Indications and Contraindications

Indications for CSF analysis include cats with suspected meningomyelitis or with multifocal signs on neurological examination. CSF analysis should always be performed when advanced imaging has failed to demonstrate a lesion. Spinal puncture is an invasive procedure with a degree of risk and should only be performed when there is a definite indication. CSF collection is always performed with the patient anaesthetized and is, therefore, contraindicated if the risk of general anaesthesia is unacceptable. Other contraindications include cats with spinal injury or instability at the proposed site of collection and cats in which raised intracranial pressure may lead to caudal brain herniation following removal of CSF. Intracranial pressure is typically normal in cats with spinal disease, but may be elevated if there is concurrent head injury, severe multifocal inflammatory disease, or neoplasia. Concurrent intracranial

disease should be suspected when the signs include an altered state of consciousness, dilated pupils with reduced pupillary light reflexes, or respiratory depression. In such cases it is advisable to delay collection of CSF until after neuroimaging when an informed decision can be made based on assessment of the risks involved.

Collection Procedure

CSF is collected either by cisternal puncture at the cerebellomedullary cistern (cisterna magna) Figure 15.2 or by lumbar puncture at L6–L7 Figure 15.3. The choice between the two sites is largely the preference of the clinician. Cisternal puncture is technically easier to perform but carries the risk of injury to the medulla or first cervical spinal cord segment if performed incorrectly. If CSF has not been obtained after three attempts at this site it may be best to abandon the procedure and perform a lumbar puncture or omit CSF analysis from the investigation. One common reason for failure is obliteration of the cistern by the disease process. The caudal flow of CSF means that a lumbar sample is more likely to be representative of a disease localized to a site cranial to the puncture. This advantage may be outweighed by the greater difficulty in obtaining a sufficient quantity of CSF and the frequency of blood contamination at the lumbar site. In many cases the pathological changes are sufficiently generalized or are multifocal so that abnormalities are present throughout the CSF. When performing a myelogram or CT myelogram, CSF should always be collected prior to injection of contrast medium. The CSF can subsequently be submitted for analysis if imaging does not provide a diagnosis.

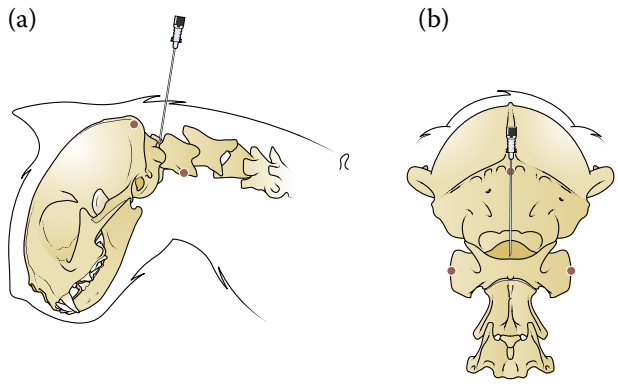


Figure 15.2 Technique for cisternal puncture. **(a)** Lateral view. **(b)** Dorsal view. The spinal needle is inserted in the dorsal midline at a point bisecting a line between the wings of the atlas, as shown by the lower dots, and the external occipital protuberance, as shown by the upper dot.

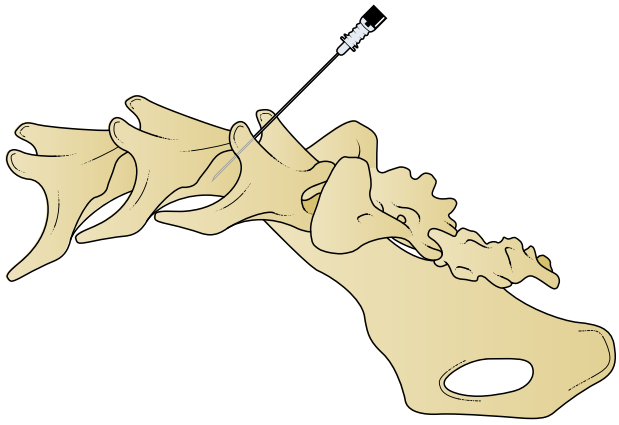


Figure 15.3 Technique for lumbar puncture. The spinal needle is inserted slightly off midline caudal to the L7 dorsal spinous process and directed cranioventrally towards the midline.

Cisternal puncture

The site is clipped and aseptically prepared for injection into the cerebellomedullary cistern. The cat should be in right lateral recumbency for the right-handed operator. The head is held by an assistant at an angle of 90° with the nose parallel to the table top. Maximal flexion is not necessary and is avoided since it may occlude the endotracheal tube. The site of injection is in the dorsal midline at a point bisecting a line joining the wings of the atlas and the occipital protuberance (15.2). The needle is inserted perpendicular to the neck and the stylet is removed once the skin has been penetrated. The needle is then slowly advanced until a slight pop is felt as it passes through the ligamentum flavum, the hub is observed for the appearance of CSF and samples are collected for laboratory examination.

Lumbar Puncture

The site is clipped and aseptically prepared for lumbar injection. The cat is positioned in lateral recumbency and the spine is hyperflexed by drawing the hindlimbs cranially between the forelimbs. The main landmark is the dorsal spinous process of L7, which lies between the wings of the ilium and is shorter than the other lumbar spinous processes. Placement of the spinal needle can be made in one of several ways. The authors' preferred method is to puncture the skin slightly off the mid-line caudal to the L7 dorsal spine and advance the needle cranioventrally towards the mid-line, sliding it down the spinous process (15.3). If bone is palpated, the needle is walked cranially off the vertebral lamina and advanced through the ligamentum flavum into the vertebral canal. A muscular twitch is felt and seen in the hindlimbs and tail as the needle penetrates the meninges. The needle is advanced to the floor of the vertebral canal. If CSF does not flow the needle may be withdrawn very slightly until CSF appears in the hub and samples are collected for laboratory examination.

Laboratory Analysis

CSF should be collected into EDTA for cytology and a plain container for biochemistry or culture. The examiner should observe the gross appearance of the CSF at the time of collection. Normal CSF is clear on gross examination unless there has been blood contamination during sampling. A yellowish discolouration is caused by the presence of free bilirubin as a result of previous haemorrhage and is known as xanthochromia. Turbidity is caused by an increase in the white cell count above approximately 500 cells/µl.

Cytology and measurement of protein levels are performed routinely and culture and sensitivity is performed if clinical signs suggest bacterial infection or if there is an increase in the number of neutrophils in the CSF. CSF contains low levels of protein so it is a hypotonic fluid with poor cellular stability. It is usually recommended that cytology

Table 15.5: Normal Values for Cerebrospinal Fluid Analysis

Colour	Colourless
Turbidity	Clear
Specific gravity	1.004–1.006
Urinary reagent strips	pH 8 +/– 1
	Protein trace to 30
	Glucose trace to +
	Blood negative
Cell number	<3/µl (cisternal)
Cell type	RBC 0
	Lymphocytes
	Monocytes
	Neutrophils (rare)
	Eosinophils (rare)
	Choroid plexus or ependymal cells (rare)
Total protein	10–27 mg/dl (cisternal)
	<45 mg/dl (lumbar)
Albumin	1–20 mg/dl

should be performed within 30 minutes of collection because of rapid cellular deterioration. Submission of freshly prepared air-dried smears of sediment is helpful in cases where laboratory examination is likely to be delayed. The addition of autologous serum and storage at 4°C was found to result in satisfactory preservation of cells for up to 48 hours in one study.

An elevation in the number of cells, termed pleocytosis, occurs when there is inflammatory disease; this is normally accompanied by a corresponding increase in the amount of protein. In general, neutrophils are indicative of bacterial infection. Mixed pleocytosis tends to be associated with fungal or protozoal disease. Eosinophilic pleocytosis defined as >10% of the total CSF leucocyte count or >10 eosinophils per microliter, is rare in cats. CSF eosinophilia is most commonly associated with parasitic infections, although it can be caused by other infections, and marked eosinophila has been reported in a cat associated with T-cell lymphoma. Vascular and traumatic diseases, and diseases resulting in degeneration of the spinal cord or nerve roots without inflammation will produce an elevation in protein with little or no pleocytosis, termed albuminocytological dissociation. Electrophoresis may be utilized to determine the levels of different protein fractions. An elevation of albumin suggests leakage into the CSF as a result of a disturbance of the blood–brain barrier, whereas elevated immunoglobulin indicates intrathecal synthesis. Glucocorticoid administration decreases the white blood cell count and/or total protein level, therefore evaluation of CSF in pre-treated cats may give false negative results. Normal values for CSF are given in Table 15.5. Interpretation of abnormal values is given in Table 15.6. Further tests such as antibody titres and PCR are performed if they are indicated.

Electrophysiologic Testing

The electromyogram (EMG) is a recording of the electrical activity of resting muscle in the anesthetized patient. Any disease process affecting the ventral horn cells in the spinal cord, the ventral nerve root, the peripheral nerve, the neuromuscular junction, or muscle can produce changes in electrical activity. Assessment of spontaneous electrical activity may be used to help differentiate between upper motor neuron and lower motor neuron deficits, but the technique is rarely used in the evaluation of feline spinal

Table 15.6: Abnormal values and their interpretation

Cell Type	<50 Cells/µl	50–500 Cells/µl	>500 Cells/µl
Neutrophils	Bacterial	Bacterial	Bacterial
	Neoplasia	Neoplasia	Neoplasia
	IVDD	FIP	Cryptococcosis
	Spinal cord trauma	Cryptococcosis	
Lymphocytes/lymphoblasts	Lymphoma	Lymphoma	Lymphoma
		Bacterial (after antibiotic therapy)	Bacterial (after antibiotic therapy)
		Toxoplasmosis	
Eosinophils	Toxoplasmosis	Toxoplasmosis	
		Cryptococcosis	
Mixed cell population	FIP	FIP	FIP
	Toxoplasmosis	Toxoplasmosis	Cryptococcosis
	Cryptococcosis	Cryptococcosis	Neoplasia
	Neoplasia	Neoplasia	
	IVDD		
	Spinal cord trauma		
	FCEM		
Erythorcytes (supernatant clear)	Iatrogenic contamination	Iatrogenic contamination	Iatrogenic contamination
Erythrocytes (erythrophagocytosis or crenated RBCs)	Intrathecal haemorrhage	Intrathecal haemorrhage	Intrathecal haemorrhage

IVDD = intervertebral disc disease
FCEM = fibrocartilaginous embolic myelopathy
FIP = feline infectious peritonitis

disease. Abnormalities in individual muscles can be used to localize pathology to specific nerves or nerve roots, which may be used in the investigation of neuromuscular disorders. These investigations are usually undertaken at specialist centres.

TREATMENT OF SPINAL DISORDERS:

The success of treatment for a spinal disorder is dependent on accurate diagnosis and is predicated on the nature of the underlying cause and the extent of the injury to the spinal cord and nerve roots. Some conditions, such as FIP and spinal lymphoma, carry a poor prognosis whereas others, such as spinal trauma and intervertebral disc disease, may carry a good prognosis for return to function with appropriate treatment.

Conservative Treatment

Conservative and/or medical treatment is indicated for a number of conditions, particularly in selected cases where neurologic dysfunction can be mild (trauma and intervertebral disc disease) or where the primary condition either cannot be addressed (fibrocartilaginous embolism) or can be addressed without resort to surgery (discospondylitis, lymphoma). Details of medical treatment are given in the appropriate section. Conservative treatment entails cage rest, nursing care, pain relief and appropriate physical therapy. Good conservative treatment of a paralyzed patient is demanding and should not be undertaken lightly. Analgesia can be provided by the use of opiates and/or nonsteroidal anti-inflammatory drugs (NSAIDs) but the latter must not be combined with glucocorticoids.

Surgical Treatment

Surgical intervention is indicated to restore the normal anatomy of the vertebral canal, to relieve compression of nervous tissue, and, in some cases, to confirm or establish a definitive diagnosis. Decompressive procedures performed on the spine are essentially the same as those for the dog and include dorsal laminectomy, hemilaminectomy, lateral corpectomy and ventral slot. With the exception of thoracolumbar hemilaminectomy, there are few indications for these procedures and this is the only technique that will be described in any detail. Surgical treatment of vertebral fracture/luxation is described in Chapter 14.

Dorsal Laminectomy and Hemilaminectomy

Dorsal laminectomy is the surgical removal of the dorsal spinous process and the dorsal lamina. Both sets of dorsal articular facets should be left intact, if possible, to preserve stability of the spine. Hemilaminectomy is the removal of one pair of articular facets and the surrounding bone of the vertebral pedicle to gain access to the vertebral canal (Figure 15.4). Laminectomy is performed to enable visualization of the contents of the vertebral canal for diagnostic purposes, to obtain biopsy samples, and for the removal of compressive lesions such as extruded disc material. Hemilaminectomy is usually preferred, especially if the lesion is ventral to the spinal cord, since manipulation of nervous tissue is avoided. Dorsal laminectomy is usually performed for dorsal or dorsolateral cervical and thoracic lesions and for dorsal or ventral lesions at the lumbosacral junction.

Hemilaminectomy is most easily performed in the thoracolumbar region, with the cat positioned in sternal recumbency and using a dorsal approach. The correct location

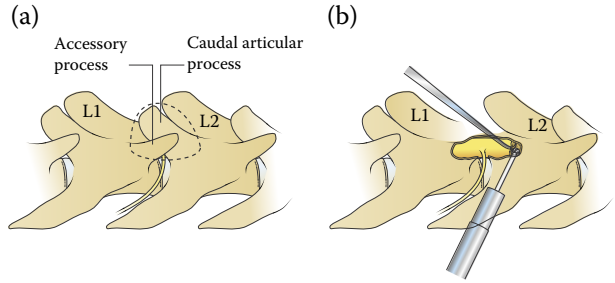


Figure 15.4 Thoracolumbar hemilaminectomy for intervertebral disc extrusion. **(a)** The articular facets are first removed with bone cutters or rongeurs. **(b)** Bone is removed using a high-speed burr down to periosteum. The remaining wafer-thin bone and periosteum are removed using fine instruments to expose the spinal nerve and extruded disc material on the floor of the vertebral canal.

is ascertained by reference to a lateral radiograph taken after placement of a marker such as the metal portion of a hypodermic needle in the dorsal spinous process of a vertebra in the region of interest. Alternatively the location can be determined by palpation of the last rib and the first lumbar transverse process. The skin and subcutaneous tissues are incised slightly to one side of the midline. The lumbodorsal fascia is incised on one side of the dorsal spinous processes and the epaxial muscle attachments are reflected from the vertebrae in the region of interest. The articular facets are removed using rongeurs or a bone cutter and the bone is removed from the pedicle using a high-speed burr to expose the spinal cord. If there is a compressive lesion, such as extruded disc material, this is removed using fine instruments and suction, taking care to touch the spinal cord or nerve root as little as possible. Particular care should be taken not to damage the nerve roots at or caudal to the L4–5 intervertebral disc, since these contribute to the nerves that innervate the hindlimbs. If the lesion is an intervertebral disc herniation, the procedure is completed by cranial retraction of the spinal nerve from the lateral aspect of the intervertebral disc and fenestration of the disc. A window is then created in the anulus fibrosus of the disc and the remaining nucleus pulposus is removed with a small dental tartar scraper or similar instrument. Wound closure is performed using simple interrupted or continuous monofilament absorbable sutures in the lumbodorsal fascia and the rest of the wound closure is routine.

Nursing and Physical Therapy

Irrespective of whether treatment is conservative or surgical, cats with severe spinal cord dysfunction should be hospitalized initially and their progress should be constantly monitored and re-evaluated by performing serial neurological examinations. Once it has been established that the cat is showing progressive neurological improvement, owners can be shown how to perform nursing and physical therapy at home.

Adequate nursing care of the paralyzed cat is crucial to achieve a successful outcome. The single most important aspect of nursing care is management of the urinary bladder. If voluntary movement in the limbs is weak or absent, it can be assumed that the bladder is paralyzed; this will require manual emptying three or four times daily or use of an indwelling catheter or tube cystostomy. If the bladder is not emptied, stretching, as a result of urinary retention, will

cause irreversible damage to the detrusor muscle so that it will not contract even when voluntary motor function returns. A lower motor neuron bladder is easier to manage than an upper motor neuron bladder since there will be less resistance to manual expression.

Other aspects of nursing care include keeping the cat clean and dry, grooming the coat, and ensuring adequate nutritional and fluid intake. Pressure sores should be prevented by the provision of soft absorbent bedding and by regular turning every 4–6 hours if the cat cannot move for itself or maintain sternal recumbency. It is imperative that cats that are being treated conservatively for spinal fracture/luxation or intervertebral disc disease should be confined in a cage and movement should be severely restricted for the first 4 weeks. Passive flexion–extension exercise and massage of paralyzed limbs should be performed with great care during this period. More aggressive rehabilitation can be started after 4 weeks but cats should be confined indoors for at least a further 8 weeks.

Physiotherapy and rehabilitation can be performed much earlier for cats that have undergone surgical stabilization following spinal trauma or decompressive surgery in the case of disc disease. Weight bearing and walking movements should be encouraged following recovery from anaesthesia by supporting the cat under the abdomen with the hands or by the use of a sling. Aquatic therapy or hydrotherapy can also be beneficial during recovery from spinal injury and is discussed in Chapter 18.

NEOPLASTIC SPINAL DISEASE:

(Goncalves et al. 2009, Marioni-Henry et al. 2004, Marioni-Henry et al. 2008, Marioni-Henry 2010, Moon et al. 1990, Troxel et al. 2003).

Introduction

Spinal cord and vertebral neoplasia is common , accounting for approximately 27% of all cases of feline spinal disease. In an MRI study of cats referred with spinal disorders, amongst the group of cats with abnormal imaging findings, neoplasia was the most frequent diagnosis. Many tumour types have been reported affecting the feline spinal cord and vertebrae, but lymphoma is the most common, with a reported prevalence between 28% and 40% of all spinal neoplasia. After lymphoma the most common tumours are osteosarcoma and other tumours of the vertebral column. Meningioma is the most common primary tumour of the central nervous system accounting for 58% of intracranial tumours in a study of 160 cases, but only 7% of tumours of the spinal cord in a study of 85 cats with histologically confirmed spinal and vertebral neoplasia, and 8% of tumours affecting the spinal cord in an MRI study of cats with spinal cord disease.

Cats with nonlymphoid tumours are typically significantly older, than cats with lymphoid tumours, and usually do not show signs of systemic illness unless there is significant metastatic disease. In a study of 85 cats with primary or metastatic spinal tumours or vertebral tumours causing spinal cord disease by local extension, the median age of cats with lymphoma was 4 years whereas the median age of cats with non-lymphoid tumours was 10 years. In this study 84.6% of the cats with spinal lymphoma also had tumour tissue at extraneural sites.

Spinal tumours can be classified according to location relative to the spinal cord and meninges as extradural-extraparenchymal, intradural-extraparenchymal and intraparenchymal. Extradural tumours such as osteosarcoma, and intra-
dural-extraparenchymal tumours, for example meningioma, are the most common. Lymphoma is either extradural or has combined extradural and intradural-extraparenchymal components. There are relatively few descriptions of intraparenchymal tumours in cats, the most frequent being astrocytoma, but oligodendroglioma, oligoastrocytoma and gliomatosis cerebri have also been reported. Intraparenchymal tumours appear to have a predilection for the caudal cervical spinal cord, and are not associated with spinal hyperaesthesia initially because the meninges are not affected.

Lymphoma

(Barr et al. 1993, Bray et al. 2016, Elmslie et al. 1991, Lane et al. 1994, Mandara et al. 2016, Mella et al. 2019, Meichner et al. 2012, Mooney et al. 1989, Shelton et al. 1990, Suess et al. 1990, Spodnick et al. 1992, Vail et al. 1998).

Clinical Signs

The clinical presentation of cats with spinal lymphoma is variable. The most frequent clinical signs are progressive paraparesis or tetraparesis. Cats with cervical spinal cord or nerve root involvement generally have subacute tetraparesis and may have lower motor neuron signs in the forelimbs. Signs typical of nerve root lesions (root signature), such as lameness and pain on shoulder or hip extension, are infrequently reported. Non-specific extraneural clinical abnormalities that often precede signs of neurological dysfunction are common, and include anorexia and weight loss, lethargy, respiratory infection, abnormal behaviour and peripheral lymph node enlargement. One study found that the duration of clinical signs before diagnosis was shorter for cats with lymphoma than for other forms of spinal neoplasia, with 93% of cats with lymphoma having a diagnosis within 2 months of the onset of clinical signs.

Cause and Pathogenesis

Lymphoma typically affects several spinal cord segments with a predilection for the thoracic and lumbosacral regions. An exclusively epidural location was thought to be most common in the past, but based on recent publications, lymphoma seems to have a combined extradural and intradural-extraparenchymal location in the majority of cases. In some cases tumour tissue may invade adjacent vertebrae and epaxial or hypaxial muscles or may extend into the spinal cord parenchyma or nerve roots.

Lymphoma can occur in the spinal cord as a primary tumour but is more commonly part of a multicentric process. The systemic clinical signs may be explained by involvement of other organ systems, which was seen in 84.6% of the cats in a post-mortem study of spinal disease, and additionally by immunosuppression in cats that are FeLV-positive. Although spinal lymphoma can occur at any age, there is a bimodal age distribution. In one study 50% of cats were less than 4 years old and 25% were more than 11 years old. The aetiological association of spinal lymphoma with FeLV probably explains the age bias in reported cases.

The combined results of two North American studies published in the early 1990s showed that 89% of 36 cats with spinal lymphoma tested positive for FeLV antigen. However, there may be geographical variations in the prevalence of FeLV, and there has been a noticeable decline in the

prevalence of FeLV-positive cats in developed countries during the past few decades, probably because of the widespread availability of an effective vaccine. A more recent study of cats in Europe with all forms of lymphoma found that the prevalence of FeLV infection between 1995 and 2009 was 13%, compared with 59% between 1980 and 1994, and a study of cats with spinal lymphoma in North America between 1980 and 2005, found that 56.5% were FeLV-positive. The proportion of cats with FeLV will have a bearing on the prognosis, since positive FeLV status has been shown to have a negative influence on the outcome of chemotherapy. Cats with feline immunodeficiency virus (FIV) infection are also predisposed to develop lymphoma. Cats with FeLV infection and cats with concurrent FeLV/FIV infection are 60 times and 80 times more likely to develop lymphoma than FeLV/FIV negative cats, respectively.

Diagnosis

Spinal lymphoma can present a diagnostic challenge. The presence of systemic clinical signs in a cat with spinal disease should alert the clinician to the possibility of lymphoma. A minimum database includes a complete blood count, serum biochemistry panel, testing for FeLV/FIV, urinalysis, and either thoracic radiographs and abdominal ultrasound or CT of the thorax and abdomen. Conventional radiography of the spine is often unhelpful as spinal lymphoma is rarely detectable even in cases where there has been local invasion of a vertebra. Lytic vertebral lesions identified radiographically are much more likely to be associated with other tumours such as osteosarcoma. In some cats radiography will reveal soft tissue masses in close proximity to the vertebrae or evidence of extraneural involvement. Myelography and CT will reveal evidence of spinal cord compression by a soft tissue mass or masses in the vertebral canal. CT is the most sensitive imaging modality for the detection of lytic osseous lesions. The imaging appearance of lymphoma is variable, and will reflect the morphology and location of the tumour tissue. On MRI, lymphoma typically has homogenous contrast enhancement and a mild to moderate mass effect, margins are usually indistinct, and there may be meningeal contrast enhancement (Figure 15.5). Unfortunately, all imaging techniques show poor sensitivity for differentiation of lymphoma from other spinal cord masses.

Cytology on blood smears is positive for lymphoma in 5–13% of cats, and neoplastic cells are seen in the CSF of 9–35% of cats. CSF often shows mild nonspecific increases in the white blood cell count and protein concentration. There is a single case report of a cat with progressive tetraparesis that had a marked eosinophilic pleocytosis associated with eosinophilic meningomyelitis and a T-cell lymphoma. Bone marrow aspiration cytology shows evidence of neoplastic infiltration in many cats with lymphoma but negative findings do not rule out the disease. Thorough staging to detect involvement of other organs especially the kidneys, liver, spleen and peripheral lymph nodes is a less invasive approach than direct sampling of neural tissue to obtain a diagnosis. Nevertheless, in some cases definitive diagnosis may require fine needle aspiration of the mass under fluoroscopic guidance or exploratory laminectomy and histopathological examination of a biopsy sample.

Treatment and Prognosis

The three main treatment modalities for spinal lymphoma, which can be used individually or in combination, are surgery, radiation therapy and chemotherapy. Few studies have reported the results of treating cats with spinal lymphoma so the number of cases is small. Radiotherapy can be effective when used to treat localized epidural spinal lymphoma. In one study of 10 cats with various forms of localized lymphoma treated with either radiotherapy alone or a combination of radiotherapy and chemotherapy, eight cats achieved complete remission with a median duration of over 2 years.

Remission of spinal lymphoma can also be achieved with a standard three drug COP combination chemotherapy protocol employing cyclophosphamide, vincristine (Oncovin), and prednisolone or a four drug CHOP protocol which also incorporates the drug doxorubicin. In one study the median duration of complete remission using a COP protocol in six cats was 14 weeks, despite the fact that all of these cats had severe neurological deficits at the time of treatment. In the same study a single cat treated by dorsal decompressive surgery and chemotherapy had a more prolonged remission of 62 weeks. The addition of doxorubicin increases the risk of gastrointestinal side-effects, predominantly inappetence, and there is currently a lack of consensus about whether there is a significant benefit in terms of overall survival. For further details concerning the treatment of lymphoma, standard oncology texts should be consulted.

Non-Lymphoid Spinal Tumours

(Asperio et al. 1999, Hammond et al. 2015, Hampel et al. 2016, Haynes & Leninger 1982, Levy et al. 1997, Nakata et al. 2017, O'Brien et al. 1980, Rossmeisl et al. 2006, Stigen et al. 2001, Shell et al. 1987, Smoliga et al. 2005).

Clinical Signs

Cats with non-lymphoid spinal or vertebral tumours are usually middle-aged to geriatric. In the largest study to date

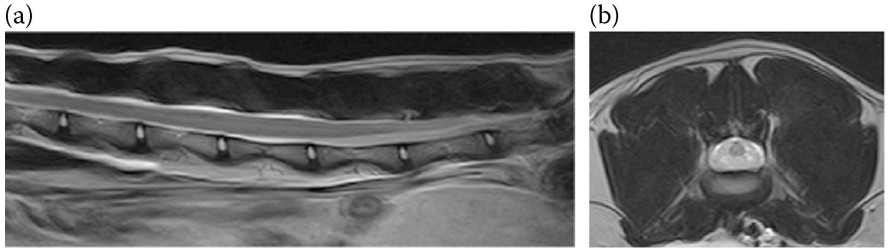


Figure 15.5 Spinal lymphoma. **(a)** Midline sagittal T2w MRI of the lumbosacral spine showing a mass-like lesion below the cauda equina. **(b)** Transverse T2w MRI of the same cat at the level of the cauda equina.

involving 26 cats with presumptive primary vertebral or spinal cord neoplasms the median age at diagnosis was 10 years with a range from 6 to 15 years. Typically there is a subacute or chronic progressive paraparesis or tetraparesis and spinal hyperaesthesia. There is usually an absence of systemic signs of illness unless there is significant intercurrent or metastatic disease. The median duration of clinical signs prior to referral in the aforementioned study was 50 days.

Cause and Pathogenesis

Osteosarcoma is the commonest malignant non-lymphoid tumour affecting the spinal cord in the cat (Figures 15.6 and 15.7). Osteosarcoma accounts for 80–90% of primary malignant bone tumours and most frequently involves the appendicular rather than the axial skeleton. In a retrospective study of feline tumours, 42% of the 19 cases of osteosarcoma involved the axial skeleton but only 11% affected vertebrae. Based on published cases the median age of cats with vertebral osteosarcoma is 8 years. Although osteosarcoma can involve any region of the spine the thoracic and lumbar regions appear to be preferentially affected. Osteosarcoma, and other malignant tumours of the vertebral column that secondarily affect the spinal cord, such as chondrosarcoma and other sarcomas are extradural in location and cause vertebral bone lysis.

Meningioma is the commonest benign spinal tumour, accounting for 16 of 18 benign feline spinal cord tumours in a study of 26 cats (Figure 15.8). All of the meningiomas in this study were intradural-extraparenchymal in location. Based on published cases the median age of cats with spinal meningiomas is 9 years. Although any region of the spine can be involved the thoracic spinal cord is preferentially affected.

Diagnosis

A minimum database includes a complete blood count, serum biochemistry panel, testing for FeLV/FIV, urinalysis, and either thoracic radiographs and abdominal ultrasound or CT of the thorax and abdomen. Thorough staging should be performed to detect evidence of metastatic disease, particularly if surgical treatment is contemplated. Survey radiography of the spine is most likely to be helpful for nonlymphoid vertebral tumours, such as osteosarcoma, where there is almost always evidence of an osseous lesion. In more advanced cases there may also be evidence of pathological fracture of the affected vertebra. Nerve sheath tumours whether benign or malignant often show radiographic evidence of enlargement of the affected intervertebral foramen as a result of pressure induced bone resorption. Malignant nerve sheath tumours may additionally show evidence of lysis of the adjacent pedicle. However, most spinal cord nonlymphoid tumours, such as meningioma, will not show any changes on radiography and in these cases advanced imaging is necessary for diagnosis. Myelography and CT will reveal evidence of spinal cord compression by a soft tissue mass or masses in the vertebral canal. CT is the most sensitive imaging modality for the detection of lytic osseous lesions. MRI is the preferred imaging modality for most spinal tumours. The imaging appearance is variable, and will reflect the morphology and location of the tumour.

Treatment and Prognosis

Few published reports have looked at the long-term outcome of a series of cases with nonlymphoid tumours undergoing surgical treatment. All spinal tumours should be critically evaluated in order to differentiate spinal lymphoma and nonlymphoid malignant tumours from benign tumours because the latter carry a better prognosis following treatment. In one study a series of 11 cats, with a range of different tumours, underwent surgical removal of tumour tissue via hemilaminectomy or dorsal laminectomy but without extensive bony resection. The median survival time following surgery alone was 180 days. In a more recent study involving 26 cats, treated by surgical removal of tumour tissue, clinical improvement was seen in 25 cats and the median survival time following surgery was 401 days. Clinical improvement postoperatively was rapid in most cats, and many cats with benign tumours had a sustained improvement in neurological function. The median survival time for the cats with benign tumours was 518 days and the median survival time for cats with malignant tumours was 110.5 days. In this study tumour phenotype was the only factor that had a significant effect on survival time. The authors concluded that surgery is successful at palliating clinical signs of spinal cord dysfunction, and prolonged survival is possible in cats with benign tumours. Successful surgical treatment of osteosarcoma of the first lumbar vertebra, by vertebrectomy and vertebral body replacement using a titanium spinal cage, has been reported. Improved management of feline vertebral and spinal neoplasia in the future is likely to require combinations of surgical, chemotherapeutic and radiotherapeutic treatments.

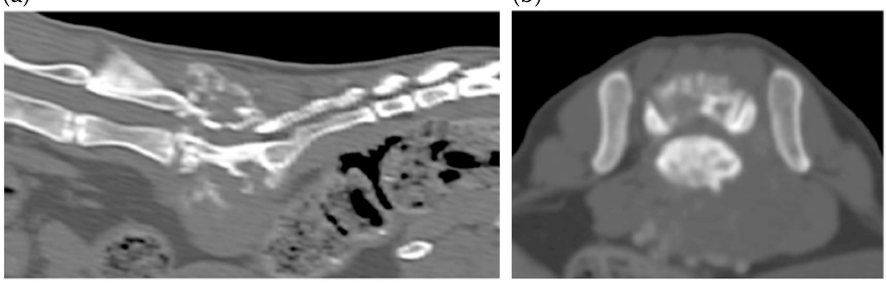


Figure 15.6 Vertebral osteosarcoma. **(a)** CT multiplanar reconstruction midline sagittal image of the lumbosacral junction of a 16-year-old domestic shorthair with progressive ambulatory paraparesis. There is extensive lysis and sclerosis of the L7 vertebra. There is evidence of a soft tissue mass extending into the vertebral canal and retroperitoneum. **(b)** Transverse CT image of the same cat at the level of the L7 vertebral body.

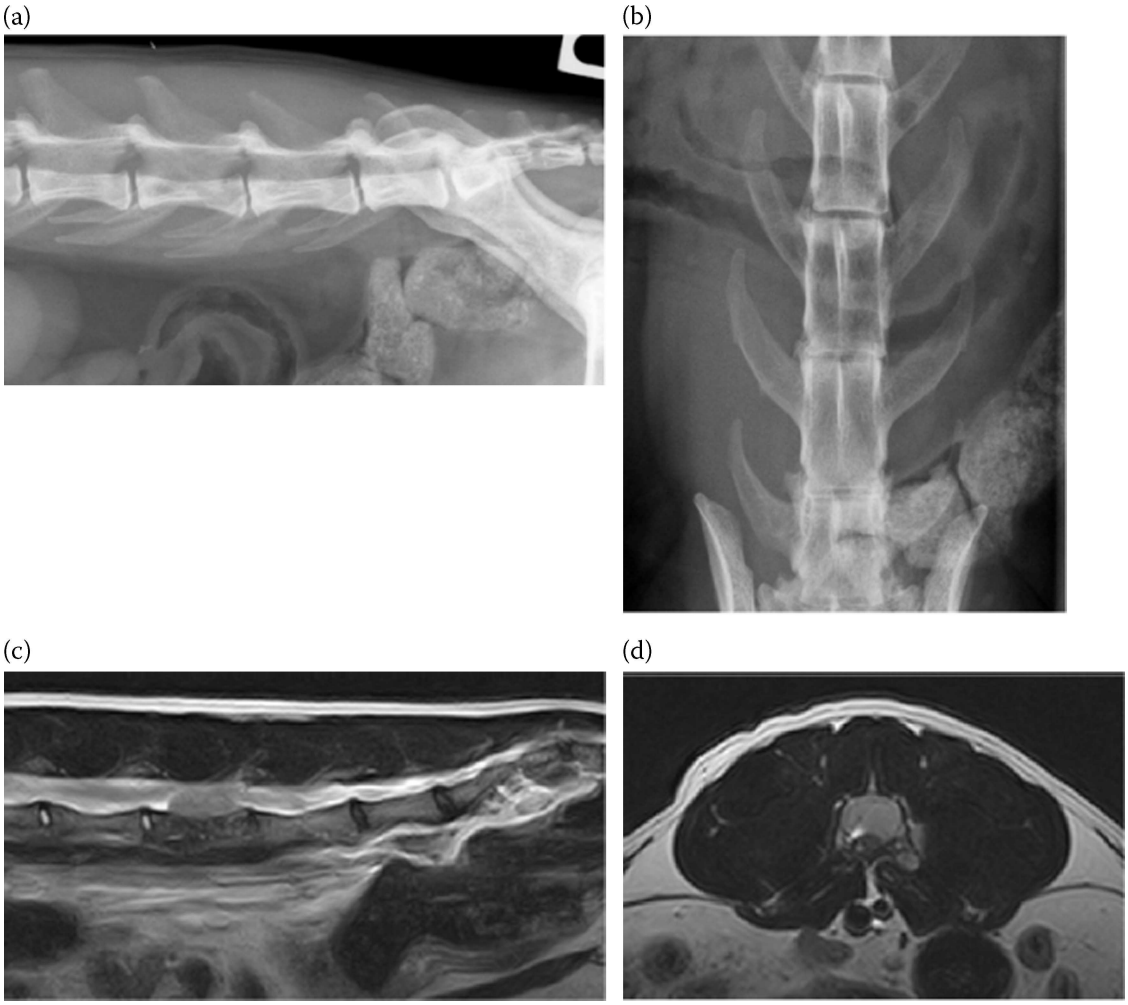


Figure 15.7 Vertebral osteosarcoma. **(a)** Lateral radiograph of an 11-year-old domestic shorthair with progressive ambulatory paraparesis. There is subtle lysis in the L5 vertebral body and enlargement of the vertebral canal of L5. **(b)** Ventrodorsal radiograph of the same cat. **(c)** Sagittal T2w MRI of the lumbosacral spine of the same cat. There is an L5 vertebral body lesion with extradural extension into the vertebral canal extending along most of the length of the L5 vertebral body. **(d)** Transverse T2w MRI at the level of L5 showing a left sided extradural mass with marked spinal cord compression.

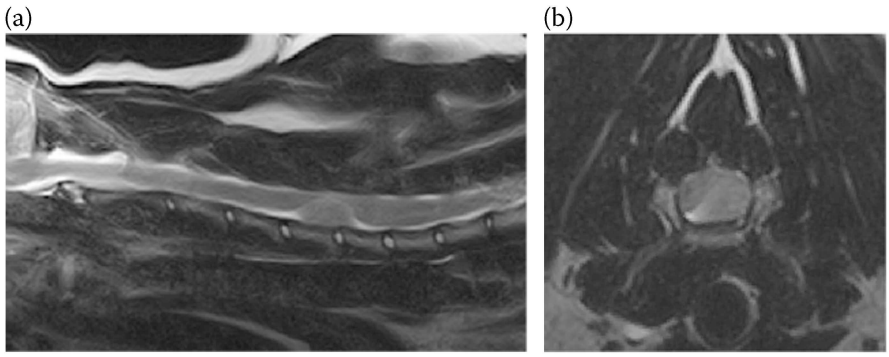


Figure 15.8 Spinal meningioma. **(a)** Sagittal T2w MRI of a 7-year-old Maine coon with progressive non-ambulatory tetraparesis. There is a well-defined intradural-extraparenchymal mass extending from the cranial endplate of C5 to the mid vertebral body of C6. **(b)** Transverse T2w MRI of the same cat showing the left sided intradural-extraparenchymal mass causing severe spinal cord compression.

INFLAMMATORY/INFECTIOUS SPINAL DISEASES:

Inflammatory Meningomyelitis

(Singh et al. 2005).

Inflammation of the meninges (meningitis) and the spinal cord (myelitis) is a common cause of feline spinal disease, accounting for approximately 32% of cases. Inflammatory disease should be suspected in a cat of any age presented with spinal hyperaesthesia, progressive paresis, hypergammaglobulinaemia, iritis, chorioretinitis and signs of systemic illness. The commonest aetiologic agent responsible for meningomyelitis is viral (FIP), followed by bacterial, protozoal (toxoplasmosis), fungal (cryptococcosis) and parasitic causes, the latter occurring only sporadically. Other possible causes include immune-mediated and idiopathic disorders of uncertain aetiology.

Diagnosis of inflammatory disease can be problematic because of the diversity of aetiologic agents. Since immunosuppression may play a role in infectious disease, all suspected cases should be tested for FeLV and FIV. CSF analysis is an essential part of the investigation for any cat with suspected inflammatory spinal disease. An inflammatory CSF is one with either an increased total nucleated cell count, or with a normal cell count, but an abnormal nucleated differential cell count. However, the results are often not specific for a particular inflammatory disorder, and in addition to primary inflammatory CNS disorders any disease that can induce tissue damage and necrosis will lead to similar changes in the CSF. A study of 62 cats with inflammatory CSF found that CSF analysis alone was useful only in the diagnosis of cats with FIP, Cryptococcus species infection, lymphoma and trauma. Overall the prognosis for cats with inflammatory CSF was poor with 77% of cats surviving for less than one year.

Feline Infectious Peritonitis

(Addie et al. 2009, Boettcher et al. 2007, Crawford et al. 2017, Dickinson et al. 2020, Doenges et al. 2016, Gruendi et al. 2017, Ives et al. 2013, Kennedy 2020, Kline et al. 1994, Legendre & Whiteneck 1975, Pedersen 2014a, Pedersen 2014b, Pedersen et al. 2019, Rissi 2018, Soma et al. 2018, Rohrer et al. 1993, Stranieri et al. 2018, Tasker 2018).

Cause and Pathogenesis

Feline infectious peritonitis (FIP) is an immune-mediated disorder associated with coronavirus infection that is invariably fatal. In most reports FIP is the commonest feline central nervous system disease, but FIP virus myelitis was only the fifth most common spinal disorder in a recent European study of 221 cats with spinal disease. In a North American study of 205 cats with histological confirmation of spinal disease, FIP accounted for 51% of all cases of inflammatory/infectious disease. Although the pathogenesis is complex and is still not fully understood the disease is caused by the FIP virus, which is a mutant of the ubiquitous feline enteric coronavirus. A marked inflammatory reaction is triggered by a population of virus-laden macrophages leading to multisystemic granulomatous or pyogranulomatous vasculitis. The severity of FIP seems to be determined by host susceptibility and immune responses, and the strain of the FIP virus.

Two forms of the disease are recognized, a progressive polyserositis (effusive or wet) form associated with pyogranulomas and serous exudates into the abdominal cavity, and a granulomatous (non-effusive or dry) form associated with granulomatous parenchymal lesions. The majority of young cats with neurological disease have the non-effusive or dry form of FIP. Approximately 38% of cats with the non-effusive form and 5% of the effusive form will have CNS signs. Involvement of the brain or brain and spinal cord together is well recognized whereas apparent involvement of the spinal cord alone is less commonly diagnosed. Cats that present with clinical signs of spinal cord involvement often have concurrent occult brain pathology and may progress to show neurological dysfunction consistent with forebrain or brainstem localization.

Clinical Signs

Cats of any age may be affected, but disease is most prevalent among cats less than 3 years of age, and especially between 4 and 16 months of age. Purebred cats and cats living in multicat environments are predisposed. Neuroanatomical localization is frequently multifocal although paraparesis as a result of a T3–L3 myelopathy is also a common manifestation of non-effusive FIP virus myelitis. When FIP causes a granuloma affecting a peripheral nerve or the spinal cord there may be lameness or progressive ataxia and paresis. Neurological dysfunction is usually accompanied by spinal hyperaesthesia and systemic signs such as anorexia, pyrexia and weight loss. Concurrent intraocular disease, usually manifesting as a uveitis, is also frequently seen in non-effusive FIP, although Toxoplasma spp., FeLV, FIV and systemic fungal infections may cause similar changes.

Diagnosis

Prompt diagnosis is important to avoid unrealistic client expectations, and prolonged efforts at unsuccessful treatment. Presumptive diagnosis is based on the characteristic signalment, history, clinical signs and physical examination, accompanied by supportive routine laboratory findings. FIP should be suspected in any young cat from a high-density environment with compatible clinical signs including a persistent but undulating pyrexia unresponsive to antibiotics. Routine laboratory test findings in cats with FIP are varied and generally non-specific. Haematologic changes may include lymphopaenia, neutrophilia, a left shift and mild to moderate normocytic, normochromic anaemia. Common serum biochemistry findings are hyperglobulinaemia and hypoalbuminaemia or low normal serum albumin. Positive serum antibody testing for feline coronavirus merely indicates that a cat has been infected with feline coronavirus and has seroconverted, which takes 2–3 weeks.

At present there is no single accurate diagnostic test for FIP. Serologic testing is nonspecific because many healthy cats and cats with conditions other than FIP may be seropositive. Cats with non-effusive FIP tend to have a higher antibody titre to feline coronavirus than non-infected cats, however there is considerable overlap and negative results have been reported in up to 57% of cases of neurological FIP, which severely limits the usefulness of the test for cats with suspected FIP myelitis.

CSF analysis reveals elevated protein (56–348 mg/dL) and pleocytosis with a mixed population of neutrophils, lymphocytes and macrophages. In some cases the CSF may not flow easily through the needle at the time of collection owing to the massive inflammatory cell accumulation. The coronavirus reverse transcription polymerase chain reaction (RT-PCR) test performed on blood, plasma or serum samples is not helpful in the diagnosis of FIP owing to the low sensitivity of the test. A recent paper described the use of the RT-PCR test on CSF samples from cats with neurological FIP and found that the specificity of the test for FIP was 100% but the sensitivity was only 42.1%. A positive result is therefore useful but a negative result does not exclude FIP. Failure to detect coronavirus in CSF may have been caused by an absence of CNS inflammation and coronavirus infected macrophages in the CSF.

A more certain diagnosis is based on RT-PCR or immunohistochemistry performed on tissue samples. Immunostaining can be performed on formalin-fixed tissues using immunohistochemistry (IHC), or on cytology samples using immunocytochemistry (ICC) or immunofluorescence (fluorescent antibody test). These techniques rely on the binding of antibodies to host cell-associated feline coronavirus antigens, which are then visualized by enzymatic reactions, either producing a colour change (immunocytochemistry) or fluorescence (immunofluorescence). A recent study evaluated the sensitivity and specificity of ICC testing of CSF in the diagnosis of cats with neurological FIP. ICC was found to be a sensitive test for the detection of neurological FIP. However, there were a number of false positive results so the test is not specific enough to confirm a diagnosis of FIP.

Definitive diagnosis has traditionally relied on histopathological examination of tissues. However, obtaining appropriate tissue samples in cats with neurological FIP is problematic. Samples are assessed for characteristic histopathological changes of FIP that when present are considered to be reliable to confirm a diagnosis. IHC is usually performed to confirm the histopathological diagnosis, and has been shown to be a more sensitive and reliable test than the fluorescent antibody test. Histopathology demonstrates lesions consisting of pyogranulomatous or lymphoplasmacytic infiltration of the meninges of the spinal cord and brainstem. Positive feline coronavirus RT-PCR or immunostaining of tissues will confirm a diagnosis of FIP but a negative result does not rule out FIP.

MRI is a sensitive means of diagnosing neurological FIP in cats with compatible clinical features. In a study that included the MRI findings in 24 cats with histopathologically confirmed neurological FIP, there were abnormalities within the brain or spinal cord or both in all of the cats. MRI of the brain should be performed in addition to the spine in cats with suspected FIP as concurrent brain pathology is common in cases presenting only with clinical signs of spinal cord disease. MRI of cats with spinal FIP typically shows meningeal contrast enhancement of the spinal cord, with or without evidence of brainstem involvement, that is bilateral and symmetrical. In the aforementioned study of 24 cats, the only imaging finding in 2 of the cats was meningeal contrast enhancement of the cervical spinal cord. Ventriculomegaly was a common finding and was detected in 20 of the 23 brains imaged. Syringomyelia was present in the cervical spinal cord in 17 of the cats and is presumed to result from obstruction of CSF flow along the central canal of the spinal cord by the extensive inflammatory cell infiltrates. One of the cats that presented with a T3–L3 myelopathy, and underwent imaging only of the spinal cord, was found to have lymphoplasmacytic meningoencephalomyelitis and ventriculomegaly on post mortem examination. All three of the cats that presented with a T3–L3 myelopathy were found to have histopathological lesions consisting of pyogranulomatous or lymphoplasmacytic infiltration into the meninges of the spinal cord and brainstem.

Treatment and Prognosis

Treatment of cats with FIP has traditionally been aimed at suppressing the immune response using appropriate drugs. Supportive therapy is also administered as required (for example analgesics, nutritional support, antimicrobials, entiemetics, appetite stimulants), based on the clinical condition of the cat. More recently treatment has focused on antiviral drugs that have the potential to effect a cure by targeting viral replication. Feline infectious peritonitis is a good candidate for antiviral drug development as there is no effective vaccine, preventative measures are difficult in multicat environments, and FIP kills 0.3-1.4% of cats worldwide.

The recently developed antiviral adenosine nucleoside analogue GS-441524 has been shown to be effective in the treatment of nonneurological FIP, although at the time of writing, data from neurological FIP cases are limited. A recent study of 4 cats, with naturally occurring FIP with central nervous system involvement, reported favourable results following treatment with GS-441524 for at least 3 months. Unfortunately legal formulations of this drug are not currently commercially available.

Remdesivir is a prodrug of GS-441524 with a broad spectrum of activity against RNA viruses. Anecdotally reported response rates to remdesivir are 80-95%. Cats with FIP with neurological signs require a higher dose of the drug than those with nonneurological FIP. Renal and hepatic toxicity has been reported in humans and may occur in cats on higher doses of the drug. Cats with concurrent FIV or FeLV infection or severe neurological dysfunction are likely to have a poor prognosis. Remdesivir (as the product Veklury) has been available in Australia via veterinary prescription for some time and is now legally available in the UK and a number of other countries from Gilead Sciences, which manufactures the drug for human use. At the time of writing remdesivir is not legally available to veterinarians in North America. Treatment recommendations are still evolving but based on current evidence, for cats with neurological signs of FIP, remdesivir should be used at a dose of 12-15mg/kg once daily for a minimum of 3 months. Neurological signs may worsen in the first few days of treatment and this may include the development of seizures which should be treated with an appropriate anticonvulsant (for example levetiracetam 20-30mg/kg every 8 hours).

Toxoplasmosis

(Alves et al. 2011, Dubey & Carpenter 1993, Hartmann et al. 2013, Lappin et al. 1992, Lindsay et al. 2010, Schatzberg et al. 2003).

Cause and Pathogenesis

Toxoplasma gondii is an obligate intracellular coccidian parasite that affects virtually all species of warm-blooded animals. Domestic cats and other Felidae are the definitive hosts for the organism and toxoplasmosis in cats is usually subclinical. Three infectious structures are recognized namely sporozoites found in oocysts, tachyzoites which are the actively multiplying stage, and bradyzoites which are the slowly multiplying stage enclosed in tissue cysts. Sporozoites are excreted in faeces, whereas tachyzoites and bradyzoites are found in tissues and milk.

Clinical signs usually only appear when bradyzoites in tissue cysts are reactivated and replicate rapidly in immunosuppressed cats. Clinical toxoplasmosis has been reported in some cats infected with FeLV or FIV. Disease is caused by cell necrosis and inflammation associated with intracellular growth of tachyzoites, and may affect multiple body tissues with diverse clinical signs. Although toxoplasmosis should always be included in the differential diagnosis of spinal disease, the nervous system was the predominant organ of involvement in only 7% of cases in 100 cats with histologically confirmed toxoplasmosis. A specific spinal localization of spinal meningomyelitis caused by Toxoplasma would appear to be very rare with only a small number of cases reported in the literature.

Clinical Signs

Reported cases of spinal toxoplasmosis have had a history of progressive paresis and ataxia with the signs reflecting the

neurolocalization. Non-specific clinical signs may include pyrexia, inappetence and lethargy. Clinical findings include paresis, ataxia, muscle pain and spinal hyperaesthesia.

Diagnosis

Antemortem diagnosis is based on clinical signs, serological examination, laboratory findings and the response to anti-toxoplasmic treatment. All infected cats develop IgG and IgA antibodies and about 80% also have IgM antibodies which can be detected using an immunofluorescence assay. Following infection IgG takes 4–6 weeks to appear with maximum antibody titre being achieved within 2–3 weeks of first appearance. Antibodies are commonly found in both affected and unaffected cats so their presence does not confirm a diagnosis of clinical toxoplasmosis. A tentative diagnosis of toxoplasmosis can be made based on a rising IgM titre, exclusion of other possible aetiologies and a favourable response to therapy. A definitive diagnosis is only confirmed when the organism is found in body fluids or tissue samples. During the acute phase of illness tachyzoites may occasionally be found in CSF, alternatively a PCR test can be performed on CSF. CSF cytology reveals a neutrophilic pleocytosis consistent with a bacterial, fungal or protozoal aetiology. In a recent report of a cat with toxoplasma meningomyelitis MRI revealed non-specific changes indicative of a focal destructive space-occupying intraparenchymal lesion.

Treatment and Prognosis

The drug of choice is clindamycin, which is administered orally at a dosage of 20 mg/kg body weight every 12 hours for 4-6 weeks. Transient vomiting is a common side-effect of clindamycin in cats. The response to treatment with clindamycin is fair to good if the cat is negative for FeLV and FIV and is treated before the development of paralysis. Systemic signs, if present, usually begin to resolve within the first 2–3 days of administration. Neurological signs generally improve more slowly and there may be residual neurological dysfunction as a sequela to nervous tissue inflammation.

Neosporosis

(Dubey et al. 1990).

Natural infection with Neospora caninum has not been documented in the cat. Experimental infection of prenatal and neonatal kittens produced lesions similar to those seen in the dog, i.e. encephalomyelitis, polymyositis and hepatitis. The disease was subclinical in adult cats.

Cryptococcosis

(Berthelin et al. 1994, Belluco et al. 2008, Bentley et al. 2018, Flatland et al. 1996, Jacobs et al. 1997).

Cause and Pathogenesis

Cryptococcus neoformans is the commonest cause of systemic mycotic infection in the cat. Cats with neurological involvement generally have diffuse meningoencephalitis, although a more localized granulomatous lesion may develop occasionally. Cryptococcosis confined to the spinal cord is rarely reported. It has been speculated that cats that are immunosuppressed as a result of FeLV or FIV infection may be more prone to cryptococcal infection. However, underlying diseases are often not detected in cats with clinical disease and factors that have predisposed to infection in the majority of reported cases remain elusive.

Clinical Signs

The clinical signs are variable according to the sites affected. Signs of spinal cord and nerve root infection are seen occasionally and include ataxia, paresis and paralysis. These may occur either alone or be accompanied by signs of involvement of other organs, typically the respiratory tract.

Diagnosis

Diagnosis is based on CSF analysis, serological testing, tissue biopsy and isolation of the organism by fungal culture. CSF cytology has revealed organisms in most dogs and almost all cats. CSF is preferred to serum for antigen testing of cats with neurological signs of Cryptococcus spp. and may be more sensitive than either CSF cytology or CSF culture. False-negative cytology and serology results may be seen and, in these cases, culture may be required to confirm a diagnosis. Unfortunately culture has limited clinical utility because there is a time delay of several weeks before the results are available.

In a recent case report a solitary spinal cryptococcocal granuloma (cryptococcoma) was diagnosed in an 11-year-old cat that presented with progressive paraparesis. The cat was found to be free from FIV, FeLV, FIP and toxoplasmosis. CSF analysis revealed a mild mixed pleocytosis with 2% eosinophils and increased total protein. C. neoformans antigen assay performed on CSF was negative. MRI findings of an irregular intraparenchymal spinal cord mass were consistent with a tumour or a granuloma with extensive secondary oedema, or gliosis and syringohydromyelia. The cat died and grossly at postmortem there was a focal malacic area in the thoracic spinal cord. Histopathological examination revealed severe granulomatous inflammation associated with a large number of encapsulated yeast cells which were identified as C. neoformans.

Treatment and Prognosis

Involvement of the brain or spinal cord is a negative prognostic indicator in cats with Cryptococcosis. Treatment is by the administration of antifungal antibiotics such as fluconazole or posaconazole that are able to cross the blood-brain barrier. Voriconazole is not suitable for cats because of neurotoxicity. Ketoconazole and itraconazole are poor choices unless the infection is extradural. Treatment should be prolonged for 2–3 months beyond the resolution of all clinical signs. Monitoring of the response to treatment by serial antigen measurement, MRI or CSF analysis is recommended. Seropositivity for FeLV or FIV was found to have a negative influence on the prognosis and such cases may require maintenance therapy.

Feline Leukaemia Virus and Feline Immunodeficiency Virus

(Carmichael et al. 2002, Meichner et al. 2012, Shelton 1991).

Tests for both of these viruses are frequently indicated as part of the investigation of spinal disease. Positive FeLV or FIV status may be associated with spinal cord disease or could be coincidental. A neurological syndrome was reported in 16 FeLV positive cats that appeared to be associated with chronic FeLV infection. Clinical signs included abnormal vocalization, hyperaesthesia, ataxia and paresis progressing to plegia. Postmortem findings suggested that FeLV has the potential to become cytopathic for cells in the central nervous system in chronically infected cats.

Neurological manifestations of FIV infection are not common but a wide variety of neurological signs have been reported, including ataxia and paresis. There is also a significant correlation between FIV infection and the development

of leukaemia and lymphoma. Furthermore, both viruses may cause immunosuppression and predispose to infection with opportunistic pathogens that may cause spinal disease, such as coronavirus, Toxoplasma spp., or Cryptococcus spp.

Bacterial Myelitis and Meningomyelitis

(Balk et al. 1974, Dow et al. 1988, Holloway et al. 2009).

Cause and Pathogenesis

Bacterial myelitis and meningomyelitis are rare but may occur occasionally when there is bacteraemia secondary to infection remote from the spine. Myelitis refers to an inflammatory process involving only the spinal cord, whereas meningomyelitis involves inflammation of the spinal cord and meninges. Clinically the distinction is based on the presence or absence of spinal hyperaesthesia which will be present in meningomyelitis but not in a cat with pure myelitis. Critically ill patients and cats that are immunocompromised as a result of FeLV or FIV infection may be predisposed. Other causes include iatrogenic (as a result of a contaminated spinal needle), or direct extension along the paraspinal muscles into the vertebral canal from an infected bite wound over the dorsum.

Clinical Signs

Affected cats show neurologic dysfunction predicated on the location and severity of the infection and will have evidence of spinal pain if the meninges are involved. There are frequently clinical signs caused by bacterial infection elsewhere in the body.

Diagnosis

CSF analysis may show neutrophilic pleocytosis and a positive bacterial culture. Neutrophils show degenerative changes and may contain phagocytosed bacteria. False-negative culture results are not uncommon. *Pasteurella* spp. and Staphylococcus spp. are the most likely isolates, although anaerobic bacteria may also be involved; definitive antimicrobial therapy should be based on the results of culture and sensitivity when available.

Treatment and Prognosis

A broad-spectrum bactericidal antibiotic that penetrates the CSF should be chosen initially and given intravenously for the first 3–5 days. If anaerobes are suspected, metronidazole may be added or a third generation cephalosporin can be used instead. Cefotaxime and ceftazidime are usually effective against anaerobes and are the drugs of choice for Gram-negative infections. However, these drugs are expensive and may have limited activity against Gram-positive cocci. Antimicrobial therapy should be continued for 2–4 weeks beyond the resolution of clinical signs. For localized infections antibiotics should be combined with drainage and debridement of affected tissues. The outcome of treatment will depend on the severity and duration of the clinical signs and the ability to isolate the organism responsible. The prognosis will be better for those cases where the underlying cause is identified and can be addressed.

Spinal Epidural Empyema

(Granger et al. 2007, Guo & Lu 2018, Kraus et al. 1989, Maeta et al. 2010).

Cause and Pathogenesis

Spinal epidural empyema or epidural abscess is a rare condition in which an accumulation of purulent material in the epidural space causes a severe myelopathy as a result of spinal cord compression and inflammation. Routes of bacterial infection include haematogenous, contiguous spread from adjacent tissues, direct inoculation (e.g. bite wound, spinal needle), aberrant parasites and migration of foreign bodies such as grass awns. Purulent epidural fluid accumulation can be extensive over several vertebral lengths or can be more focal or multifocal along the length of the spinal cord. Bacteria isolated from affected cats have included *Pasteurella multocida* and *Staphylococcus aureus*.

Clinical Signs

Clinical signs include spinal hyperaesthesia and rapidly progressive neurological dysfunction predicated on the location and extent of the lesion. Systemic signs of infection such as pyrexia, lethargy and inappetence are not invariably present.

Diagnosis

Early diagnosis is imperative as delayed diagnosis and treatment is more likely to result in permanent neurological dysfunction. A complete blood count may reveal a mild to moderate leukocytosis with a neutrophilia. CSF examination may reveal a neutrophilic pleocytosis with elevated protein concentration. Contrast enhanced MRI is the imaging modality of choice and diffusion weighted sequences are useful to confirm infection. If liquid purulent material is present there will be a region of T2w hyperintensity, with T1w hypointensity and a rim of T1w enhancement. CT myelography should be performed if MRI is not available as lesions may be missed on both myelography and non-enhanced CT.

Treatment and Prognosis

Aggressive treatment with surgical decompression in combination with broad spectrum bactericidal antibiotic therapy is most likely to be successful in cases with rapid progression of neurological dysfunction. In more mildly affected cases antimicrobial therapy alone may be successful preferably targeted on the basis of culture and sensitivity testing. In a recent report of four cats with spinal epidural empyema three of the cats were treated by surgical decompression and one cat was treated medically. All of the cats improved following treatment over a 3 month follow-up period.

Discospondylitis

(Gomes et al. 2019, Malik et al. 1990, Norsworthy 1979, Packer et al. 2005, Watson & Roberts 1993).

Cause and Pathogenesis

Discospondylitis is an inflammatory process involving the intervertebral disc space (discitis) and adjacent cartilaginous endplates of successive vertebral bodies (spondylitis). The condition is not uncommon in the dog but is rare in the cat. The largest study to date involved 13 cats from three referral institutions. In this study the mean age of affected cats was 9.1 years (range 1–14 years), and there were five females and eight males. The majority of cats were domestic shorthair. A single cat had two sites of infection and these were L1–L2 and L5–L6. There is a predilection for lumbar and lumbosacral intervertebral discs with thoracic disc involvement only reported twice (T12–T13 and T13–L1). In published reports the L7–S1 disc is the most commonly affected site, accounting for almost half of all cases.

In most cases discospondylitis originates by haematogenous spread of bacteria from a source of sepsis elsewhere

in the body, such as the urinary tract. The infection is thought to start as an osteomyelitis of the vertebral metaphysis and then spread to the disc and adjacent soft tissues after penetrating the vertebral endplate. The involvement of multiple sites in a few of the reported cases is consistent with haematogenous spread rather than extension from a local soft tissue infection.

Clinical Signs

Clinical signs include spinal hyperaesthesia, progressive ataxia, paresis, plegia, inappetence and pyrexia. Spinal hyperaesthesia with no neurological dysfunction has been reported.

Diagnosis

Radiographic features include erosion of adjacent vertebral endplates with variable degrees of spondylosis deformans and endplate sclerosis. Vertebral osteolysis, irregular new bone proliferation ventral to the affected disc, an increase in ventral soft tissue opacity and narrowing of the intervertebral disc space are variable features. CT is more sensitive for detecting osteolysis than radiography and offers a clearer depiction of bone lesions with the potential to achieve a diagnosis earlier in the course of the disease (Figure 15.9). In a recent study of 13 cats there were two affected sites where CT and radiography failed to reveal evidence of discospondylitis despite characteristic changes on MRI. The MRI features of discospondylitis in cats have recently been described in detail. Blood and urine should be cultured to identify the causative organism but have been reported to be negative in 40–75% of cases in dogs. Percutaneous disc aspiration may be more likely to yield a positive culture. Bacteria isolated from affected cases have included Escherichia coli, Enterococcus sp. Clostridium perfringens, Streptococcus canis and Actinomyces viscosus.

Treatment and Prognosis

Medical management is likely to be successful if neurological dysfunction is not severe. A broad-spectrum bactericidal antibiotic should be chosen, preferably based on the results of culture and sensitivity. If there is no clinical improvement within the first 7–10 days, the antibiotic should be changed or the diagnosis reviewed. Treatment with a suitable antibiotic is continued for a minimum of 6 weeks but may be required much longer. Follow-up radiographs should be obtained every 6 weeks to assess the efficacy of therapy. Antimicrobial therapy is continued until lesions become radiographically silent with resolution of lytic foci and their sclerotic margins, and bony bridging of affected vertebrae. Bony changes lag behind clinical improvement so worsening of the radiographic features may be seen on the first set of follow-up films.

The prognosis for cats with severe neurological dysfunction is likely to be guarded. These cases may require surgical management for curettage of affected disc spaces, to obtain samples for culture, and to decompress the spinal cord. Instability may play a role in the pathogenesis and perpetuation of infection, so concurrent surgical stabilization of affected vertebrae should be considered. Information in the literature suggests that spread of the infection from the disc to the meninges occurs early in the course of the disease when the radiographic changes are subtle. If this is a particular feature in cats, it is likely to have a negative influence on the prognosis.

DEGENERATIVE SPINAL DISEASE:

Intervertebral Disc Disease

(Bagley et al. 1995, Bottcher et al. 2008, Crawford et al. 2018, De Decker et al. 2017, Farrell & Fitzpatrick 2015, Gage 1975, Gilmore 1983, Hamilton-Bennett & Behr 2019, Jeffery et al. 2016, Kathmann et al. 2000, King et al. 1958, King & Smith 1960a, King & Smith 1960b, King & Smith 1964, Knipe et al. 2001, Littlewood et al. 1984, Lu et al. 2002, Magi et al. 2018, Malik et al. 2009, Maritato et al. 2007, McConnell & Garosi 2004, Macpherson & Ye 1988, Munana et al. 2001, Rayward 2002, Seim & Nafe 1981, Salisbury & Cook 1988, Sparkes & Skerry 1990, Scott & McKee 1999).

Introduction

Clinically significant degenerative intervertebral disc disease (IVDD) is considered to be a rare disorder of middle-aged to older cats. By contrast, intervertebral disc degeneration is a common incidental post-mortem finding in cats, especially in the cervical region. The reported prevalence of feline IVDD is 0.12–0.24% compared with a reported prevalence of 2.0% in the canine population. In two large retrospective studies of referred populations of cats with spinal cord disease, confirmed by histopathology in one, and MRI in the other, IVDD was diagnosed in 4% and 5.4% of cats, respectively. There have been a growing number of reports of feline IVDD in the literature in recent years, which may have several explanations. The disease may be showing a true increase in prevalence because of the increased life expectancy of cats or, more likely, feline spinal disease is being investigated and treated with greater frequency. It is important to include IVDD in the differential diagnosis for any cat with paresis or paralysis as with appropriate management the prognosis is often good, unlike many other feline spinal cord diseases.

Cause and Pathogenesis

Widespread intervertebral disc degeneration was discovered from about 10 years of age onwards in a series of groundbreaking post-mortem studies on the feline intervertebral disc

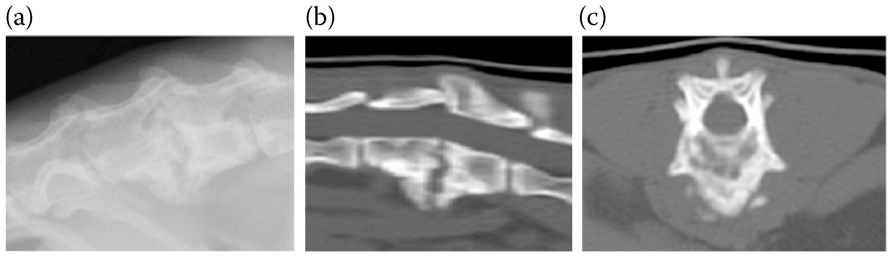


Figure 15.9 Discospondylitis. **(a)** Lateral radiograph showing lysis of the vertebral endplates of L1–L2 typical of chronic discospondylitis. **(b)** Midline sagittal CT multiplanar reconstruction of the same cat. **(c)** Transverse CT image at the level of the cranial endplate of L2.

performed by King et al. (1958 and 1960). In these studies Hansen type 1 disc extrusion and Hansen type 2 disc protrusion were identified, although the latter was much more common. In contrast to dogs, the thoracolumbar area was not affected and cervical disc protrusions were more common than lumbar disc protrusions. None of these cats was showing clinical signs of disc disease and it was postulated that intervertebral disc degeneration was of little clinical relevance in the feline. However, over the last few decades there has been growing awareness of feline IVDD as a significant clinical entity. In contrast to the post-mortem studies, the majority of cats that have presented with clinical signs of IVDD have had Hansen type 1 disc extrusion.

The reason that cats are rarely affected by IVDD compared with dogs is unclear. A preliminary investigation evaluating histopathological changes in normal and degenerative feline intervertebral discs found that there were feline-specific changes in the anulus fibrosus that may have a protective effect. Furthermore, chondrodystophic dwarfism, which is the basis for a number of common dog breeds such as the dachshund, predisposes to IVDD, and there are few chondrodystrophic cats. Chondrodystrophic breeds of cat include the munchkin or sausage cat and related breeds such as the minskin, Napoleon or minuet, and bambino. Although there appears to be an increased prevalence of lordosis in munchkins, there is currently no evidence of a predisposition for IVDD in any of these cat breeds.

The reported anatomical distribution of feline disc disease is different from that of the dog with a disproportionate localization to the lumbar region of the spine, followed by the thoracolumbar junction, with fewer reports of clinical disc disease in the cervical region. Clinically relevant cervical disc disease accounts for approximately 16% of cases in dogs and an estimated 6% of cases in cats. Sacrocaudal intervertebral disc protrusion, which is rare in dogs has also been reported in two cats. The reasons for the different anatomical distribution of feline disc disease is not fully understood but may reflect the greater flexibility of the vertebral column in the cat. Range of motion in the sagittal plane is similar across most thoracic and lumbar joints in the feline spine resulting in a more even distribution of forces, whereas in the dog there is a concentration of motion at the thoracolumbar and lumbosacral junctions.

The most frequently affected discs in the cat are those in the caudal lumbar region, in contrast to the dog where the intervertebral discs at and adjacent to the thoracolumbar junction are most at risk, especially T12–13 and T13–L1 in small and large breed dogs, respectively. The lumbosacral intervertebral disc is a common site of Hansen type 2 disc protrusion in the dog, and there is an emerging recognition of symptomatic feline lumbosacral disc protrusion, which has many similarities with its canine counterpart. Lumbosacral disc disease has some unique features that differentiate it from IVDD in other regions of the spine, and is discussed later in this chapter.

Clinical Signs

The clinical signs of IVDD are very similar to those in the dog. Cats with Hansen type 1 disc extrusion will typically have an acute onset of progressive clinical signs, and unlike lymphoma or inflammatory spinal diseases such as FIP, there are usually no concurrent systemic clinical signs or abnormalities. By contrast, cats with clinically significant Hansen type 2 disc protrusion will typically have a more insidious onset and a much slower progression of signs. Reported cases have varied in age but most cases have been middle-aged to older with a mean age ranging from 8 to 9.8 years. In a number of studies purebred cats have been overrepresented and it has been suggested that British shorthairs and Persian cats in particular may be predisposed to IVDD. It is not known whether this reflects a true breed predisposition or whether it is because purebred cats are more likely to be referred for investigation of spinal disease. There is no convincing sex predilection for thoracic and lumbar IVDD although male cats appear to be overrepresented in reports of lumbosacral disc disease.

The severity of clinical signs ranges from spinal hyperaesthesia with no neurological deficits (grade 1) to plegia with an absence of nociception in the hindlimbs (grade 5). Neurological signs of cats with cervical disc extrusion in reported cases has ranged from chronic mild ambulatory tetraparesis and proprioceptive ataxia to acute non-ambulatory tetraparesis. Patients with tetraplegia as a result of cervical disc disease are unlikely to present for treatment because respiratory arrest will intervene. Spinal cord compression at or caudal to the L5–6 intervertebral disc will cause lower motor neuron signs and hyporeflexia or areflexia as the nerve roots of the cauda equina are affected. Because of the propensity for caudal lumbar disc disease, a significant number of cats that present with IVDD will have lower motor neuron deficits and localize to spinal cord segments L4–S3.

Diagnosis

IVDD should be included in the differential diagnosis for any cat presented with spinal cord disease, especially where there is a history of an acute to subacute onset. In comparison with cats presented with spinal trauma or lymphoma, cats with IVDD tend to be older, are more likely to be kept indoors, and test negative for FeLV. Radiography has severe limitations in the diagnosis of IVDD but may provide some clues where there is no access to advanced imaging. However, radiographic evidence of disc degeneration is not an uncommon incidental finding in older cats, and conversely cats clinically affected by disc extrusion may show minimal changes on conventional radiographs. The radiographic changes seen with IVDD are similar to those described for dogs. Survey films may show mineralization of the nucleus pulposus of one or more discs in cats with disc degeneration. Cats with clinical disc disease may show narrowing of the extruded disc space, radiopacity in the affected intervertebral foramina, and a reduction in the width of the diarthrodial joint space.

Myelography is rapidly becoming obsolete, but if performed may show an extradural compressive lesion centred over the affected intervertebral disc space. Since most cats have an acute to subacute onset of thoracolumbar disc disease, the preferred site of contrast injection is L6–L7. Cisternal injection may be used if the lesion is chronic or has been localized to the cervical region by neurological examination. Left and right lateral oblique views are obtained as soon as possible after the injection as these are most useful for determining lateralization, which is important when performing a hemilaminectomy. The ventrodorsal and lateral views are then obtained.

High-field MRI is considered to be the imaging modality of choice for diagnosing intervertebral disc disease followed by CT (Figure 15.10). The MRI features of feline disc disease have been described in a number of reports. Typically in cats with an extruded disc there will be a variable volume of T2w hypointense extradural material contiguous with the

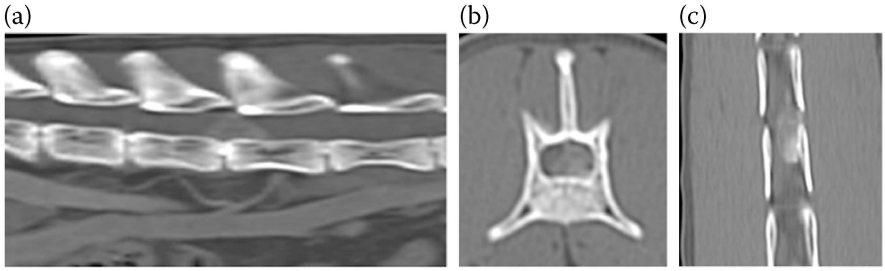


Figure 15.10 Lumbar disc extrusion. **(a)** Midline sagittal CT multiplanar reconstruction (MPR) in a 10-year-old domestic shorthair with acute paraplegia. There is a predominantly hyperdense extradural lesion causing severe spinal cord compression dorsal to the L3–L4 intervertebral disc space. **(b)** Transverse CT of the same cat at the level of the L3–L4 intervertebral disc space showing a large amount of disc material in the vertebral canal. **(C)** Dorsal CT MPR showing the disc material on the left side of the vertebral canal.

affected intervertebral disc causing ventral or ventrolateral compression of the spinal cord (Figure 15.11). Other features that can be identified on MRI are extradural haemorrhage and T2w intraparenchymal hyperintensity (Figure 15.12). Intraparenchymal (intramedullary) and intradural-extraparenchymal intervertebral disc extrusions are rare but have been recorded in a number of cats. Diagnosis of an intraparenchymal disc extrusion is based on the MRI finding of a linear disc trail extending from the intervertebral disc space into the spinal cord (Figure 15.13).

Treatment and Prognosis

The approach to treatment is based on a number of factors including the severity of the clinical signs, the degree of spinal cord compression, and the finances and wishes of the client. Conservative treatment should entail appropriate analgesia and strict cage rest for 4 weeks followed by house confinement for a further 8 weeks. Conservative treatment of the paralyzed patient is discussed earlier in this chapter. Surgical treatment involves decompressive surgery with or without fenestration of the affected intervertebral disc. Since

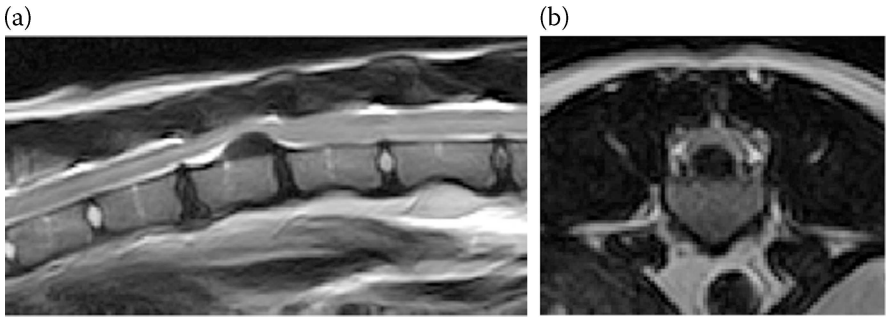


Figure 15.11 Thoracolumbar intervertebral disc extrusion. **(a)** Midline sagittal T2w MRI of an 8-year-old Siamese with acute ambulatory paraparesis showing a large disc extrusion at T13-L1. There is mild dilation of the central canal cranial to the level of the T13-L1 intervertebral disc space. **(b)** Transverse T2w MRI at the level of the disc extrusion. There is severe spinal cord compression which is slightly worse on the right side of the vertebral canal.

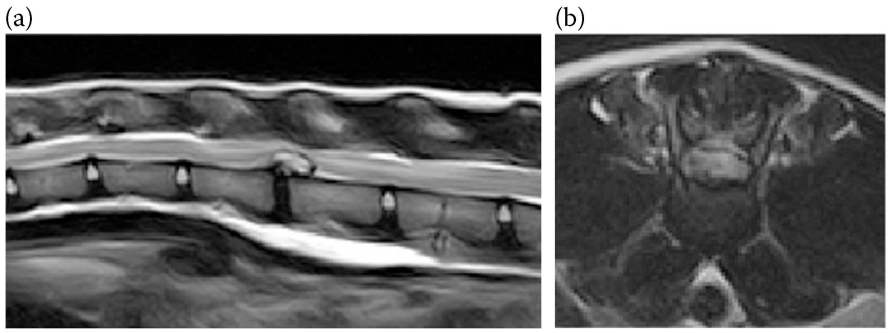


Figure 15.12 Thoracolumbar intervertebral disc extrusion and haemorrhage. **(a)** Midline sagittal T2w MRI of a 12-year-old Devon rex with acute paraplegia showing an intervertebral disc extrusion at L1–L2. There is a large amount of extradural material with mixed intensity ventral to the spinal cord resulting in severe spinal cord compression. The material is composed of disc material and haemorrhage. **(b)** Transverse T2w MRI of the same cat at the level of the disc extrusion. There is severe spinal cord compression which is slightly worse on the right side of the vertebral canal.

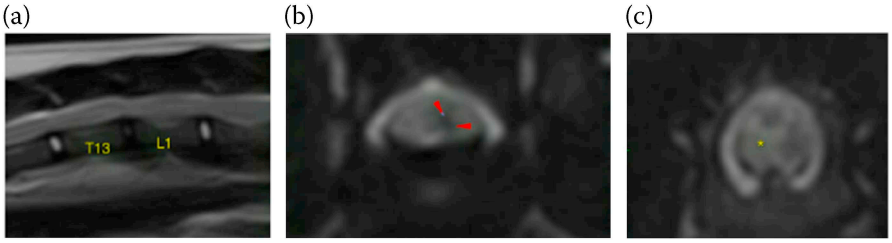


Figure 15.13 Intraparenchymal (intramedullary) intervertebral disc extrusion. **(a)** Midline sagittal T2w MRI of a 4-year-old domestic shorthair with peracute non-ambulatory paraparesis showing degeneration of the intervertebral disc at T13-L1. **(b)** Transverse T2w MRI at the level of the T13-L1 intervertebral space showing mild flattening of the spinal cord and a hypointense focus of disc material within the left ventral aspect of the spinal cord (red arrowheads). **(c)** Transverse T2w MRI at the level of caudal L1 showing oedema of the spinal cord white matter (yellow asterisk).

most clinical disc disease is located in the lumbar region, hemilaminectomy is the procedure of choice. For cases with ventral midline spinal cord compression associated with Hansen type 1 or 2 disc disease, or in chronic cases where adhesions are present between the disc material and the dura mater, decompression by hemilaminectomy may involve undue manipulation of the spinal cord. In order to gain access to compressive material and to limit iatrogenic trauma associated with its removal partial lateral corpectomy is the preferred technique for these cases. There are currently two reports of partial lateral corpectomy in the cat, one for a chronic midline intervertebral disc protrusion at L1–L2 and the other for a cat with a midline disc protrusion at T3–4. The only reported modification of the technique in the thoracic vertebrae in comparison with canine lateral corpectomy was increased slot height in order to achieve unconstrained access to the vertebral canal. The procedure was successful in resolving the clinical signs in both cases.

There are currently two reports of ventral slots performed on a total of four cats with cervical IVDD. In the one report the cat, which had a cervical disc extrusion at C2–C3, died in hospital 3 days postoperatively as a result of cardiorespiratory arrest. In the other report two cats with cervical IVDD had a single ventral slot and one cat had two ventral slots performed at adjacent sites. All three cats showed neurological improvement postoperatively. Although the number of reported cases is too low to draw definite conclusions about the optimum method of management, on the basis of this very small number of affected cats, it would appear that the prognosis following decompressive surgery for cervical IVDD is favourable.

There are a number of reports describing the treatment and clinical outcome of cats with clinically significant IVDD including a recent report describing the MRI features and outcome in six cats with lumbar intervertebral disc extrusion treated by hemilaminectomy. Nevertheless, the published evidence on feline intervertebral disc disease remains very limited compared with that for dogs. In the opinion of the authors decompressive surgery is indicated for cats with significant neurological dysfunction, cats failing to respond to conservative management and cats with moderate to severe spinal cord compression. In contrast to the situation in dogs, because other causes of spinal disease are more common in the cat, it is difficult to make even a presumptive diagnosis of IVDD without performing advanced imaging. The authors' protocol for treatment is therefore based primarily on the degree of spinal cord compression identified on advanced imaging.

The prognosis for all surgically treated cases that retain nociception is good to excellent. It should be noted that there are no reported cases of recurrent disc extrusion following successful surgical treatment in the cat. Multiple disc fenestration as frequently advocated in the dog, as a prophylactic measure, is not currently indicated in the cat. Adverse prognostic indicators include absence of nociception and T2w intraparenchymal hyperintensity on MRI. Increased MRI signal intensity in the spinal cord of paraplegic dogs with thoracolumbar intervertebral disc extrusion has been associated with a poor prognosis for recovery of function after decompressive surgery, and there is at least one report of a poor outcome in a cat with an extensive area of intraparenchymal T2w hyperintensity seen on MRI. There are reports of cats with paraplegia and absence of nociception having a good to excellent outcome after decompressive surgery, but the small number of cases precludes the formulation of any meaningful conclusions about the prognosis for these cats following surgical or nonsurgical management. In light of the current lack of evidence it is not unreasonable to extrapolate information from studies in dogs with paraplegia and an absence of nociception where the recovery rate following decompressive surgery based on a recent meta-analysis was 61%. In the same study the recovery rate following conservative treatment of such cases was 10%.

The authors' recommendations for treatment are as follows:

- Mildly affected cases (grade 1 or 2 with minimal to mild spinal cord compression) can initially be treated conservatively by cage rest and the use of analgesia if necessary. Clients should be warned that sudden deterioration of signs may occur, particularly if cage rest is not performed correctly. There is also a risk of recurrence of neurological signs after successful conservative treatment and these signs may be worse than the original episode. Recurrence is thought to occur in about 30% of dogs, the recurrence rate for cats is currently unknown.
- More severely affected cases (grades 2 through 4 with moderate to severe spinal cord compression) should be treated by hemilaminectomy and fenestration of the affected disc. Some of these cases would recover with conservative treatment but the recovery is less predictable, takes longer, and is less likely to be complete, especially in those cats with absent voluntary movement. There may be a risk of recurrence of signs following resumption of normal activity following conservative treatment.
- The most severely affected cases (grade 5 with moderate to severe spinal cord compression). The authors are not

aware of any reports of cats that have developed ascending/descending haemorrhagic myelomalacia following intervertebral disc extrusion (although the condition has been reported in a cat with systemic hypertension). Nonetheless, the possibility of this complication occurring should be discussed with the client before undertaking surgery on cats with signs of severe neurological dysfunction particularly where there is extensive spinal cord T2w hyperintensity, which has been identified as a risk factor in dogs.

- Cats that are not improving with conservative therapy within a period of 2 weeks and cats showing progression of neurological signs are also likely to be good surgical candidates but repeat imaging may be necessary to confirm worsening spinal cord compression.

Thoracic Vertebral Canal Stenosis

(Carletti et al. 2019, Gillespie & De Decker 2019, Gillespie & De Decker 2020, Mella et al. 2019).

In a study of a referral population of 221 cats with spinal disease a diagnosis of thoracic vertebral canal stenosis was made in 11 cats (5%). In a further study of nine of these cats, British shorthair and male neutered cats were overrepresented and the median age was 9 years. Clinical signs in the majority of cats were chronic progressive ambulatory paraparesis and hindlimb ataxia with thoracic spinal hyperaesthesia. Diagnosis was based on MRI findings of obvious dorsoventral flattening of the spinal cord and adjacent intraparenchymal signal intensity changes. The vertebral canal at T3–T4 and T11–T12 was most commonly affected followed by T4–T5 and T8–T9. Affected cats were found to have a significantly smaller vertebral canal diameter in the median plane at multiple levels compared to unaffected cats. It was proposed that pre-existing relative vertebral canal stenosis predisposes to spinal cord compression caused by degenerative changes in the vertebral column. Two of three cats treated surgically had a good long-term outcome whereas one cat deteriorated 8 months postoperatively after an initial improvement. Of five cats treated medically two improved and three deteriorated resulting in euthanasia.

Thoracic vertebral canal stenosis has also been reported in two cats associated with articular process hypertrophy. The first was a 9-year-old British shorthair and the other a 13 year old domestic shorthair cat. Both cats presented with a history of a chronic progressive myelopathy. MRI revealed bilateral enlargement of the articular process joints at T11–T12 in one case and at T3–T4 in the other causing dorsolateral extradural spinal cord compression. Surgical decompression, performed by hemilaminectomy and partial osteotomy of the spinous process via a unilateral approach, resulted in satisfactory outcomes in both cases.

Thoracic vertebral canal stenosis although uncommon should be included in the differential diagnosis for any adult cat with a chronic progressive, painful T3–L3 myelopathy. The index of suspicion should be higher for male neutered British shorthair cats.

Lumbosacral Disease

(Kot et al. 1994, Shkorbatova et al. 2019).

Synonyms for lumbosacral disease include lumbosacral syndrome, and cauda equina syndrome. Lumbosacral disease can be defined as any condition caused by compression, displacement, or destruction of the nerve roots of the cauda equina. The cauda equina is the collection of nerves that courses caudally beyond the conus medullaris to exit the vertebral canal at their respective intervertebral foramina. There is no precise anatomical definition of the cauda equina but the commonly accepted view is that it includes the L7, sacral and caudal nerves. The conus medullaris is the elongated cone shaped terminal portion of the spinal cord consisting of segments S2, S3 and Ca 1 to 5. There is some individual and breed variation in the termination of the spinal cord but typically the rootlet areas of segments L6, L7 and S1 are located within or below the L5 vertebral body and the segments S2 and S3 are located within or caudal to the L6 vertebral body. Therefore any pathology at or caudal to the L5–L6 disc space will cause cauda equina neuropathy.

In comparison with the dog, lumbosacral disease is infrequently diagnosed in cats, with the exception of spinal fracture/luxation. Causes include spinal and vertebral neoplasia, especially lymphoma, caudal lumbar or lumbosacral disc protrusion or extrusion, fibrocartilaginous embolism, cauda equina neuritis and discospondylitis. Cauda equina neuritis is caused by inflammatory disease such as FIP or toxoplasmosis. Spondylosis deformans is not an uncommon finding at the lumbosacral junction and sometimes appears to be associated with vague signs of spinal discomfort suggestive of mild nerve root compression.

The principles for the investigation of lumbosacral disease are the same as those for other regions of the spine. Delineation of the lumbosacral region on plain ventrodorsal radiographs can be improved by positioning the hindlimbs cranially, thereby flexing the lumbosacral spine. The clinician should be aware of the anatomy of the lumbosacral spinal cord in the cat. Prior to MRI and CT, myelography was used to investigate lumbosacral disease. Myelography is more useful in the cat than the dog because the dural sac invariably crosses the lumbosacral junction and continues well into or beyond the sacrum. Advanced imaging has now almost completely superseded myelography, and MRI is the preferred imaging modality for the investigation of lumbosacral disease.

Degenerative Lumbosacral Stenosis

(Cariou et al. 2008, Danielski et al. 2013, Farrell & Fitzpatrick 2015, Harris & Dhupa 2008, Harris et al. 2019, Hurov 1985, Jaeger et al. 2004, Newitt et al. 2009, Sunol et al. 2018).

Cause and Pathogenesis

Degenerative lumbosacral stenosis (DLSS) is a complex multifactorial condition in which there is narrowing of the vertebral canal and/or L7 neuroforamina at the level of the lumbosacral junction. Stenosis is caused by a varying combination of degenerative changes at the lumbosacral junction. These changes are well described in large breed dogs but can also occur in cats and include L7-S1 disc protrusion, hypertrophy of soft tissues (ligamentous and synovial structures), articular facet osteophytosis, lumbosacral spondylosis deformans and spondylolisthesis with cranioventral tipping of S1 relative to L7. Intervertebral disc protrusion or extrusion could occur at L7-S1 without any other evidence of degenerative joint disease but appears to be uncommon in the cat. Diagnosis and treatment would be the similar to that for DLSS.

Instability, which may be defined as increased or abnormal motion at the lumbosacral junction, has been proposed as an important factor in the pathogenesis of

DLSS in dogs. Although reduced range of joint motion has been reported in some dogs with DLSS, when the vertebral canal and L7 neuroforamina are stenotic, any degree of motion could cause dynamic compression of the cauda equina, and the L7 nerve roots in particular. Hindlimb neurological deficits are lower motor neuron in nature, and relate to the sciatic nerve, to which the L7 nerve root is a major contributor. Faecal and urinary incontinence with a lower motor neuron bladder occur when there is pelvic and pudendal nerve dysfunction. A recent study examined the relationship between lumbosacral transitional vertebrae and DLSS and found that transitional vertebrae were significantly more prevalent in the cats with DLSS (53.8%) compared with a control population of unaffected cats (5.9%). Lumbosacral transitional vertebrae are therefore a significant risk factor for the development of DLSS in cats presumably because degenerative changes at the lumbosacral junction are accelerated by the altered biomechanics.

Clinical Signs

In most reports there is a male predisposition to DLSS. The mean age of affected cats is 9.3 years. In contrast to dogs, there are relatively few reports of feline DLSS in the published literature. As with degenerative joint disease elsewhere it is likely that the condition is underdiagnosed because the clinical signs are relatively nonspecific and cats are able to adapt their lifestyle to compensate for the disability. Clinical signs are usually chronic and progressive although there may be acute exacerbation. Common clinical signs include difficulty jumping, reduced activity, unilateral or bilateral hindlimb lameness, low tail carriage, elimination outside the litter tray, difficulty posturing to defaecate, lumbosacral hyperaesthesia and spontaneous vocalization when exercising. In more severely affected cases there may be mild hindlimb paresis and ataxia and urinary and faecal incontinence.

Diagnosis

DLSS should be suspected in any middle aged or older cat with compatible clinical signs and with evidence of pain when direct digital pressure is applied over the lumbosacral region and when the hind limbs and/or tail are hyperextended. Postural reaction tests may be abnormal and the withdrawal reflex will be reduced if there is partial sciatic neuropathy secondary to compression of the ipsilateral L7 nerve root.

Conventional radiography may show degenerative changes at the lumbosacral junction including narrowing of the intervertebral disc space, end-plate sclerosis, ventral and lateral spondylosis deformans and articular facet joint hypertrophy. However, these findings may be incidental, and conversely symptomatic cats may have mild or minimal radiographic changes. Myelographic changes have been described but the procedure is now rarely performed, particularly for lumbosacral disease. CT will show the bony changes in great detail and is the preferred technique for surgical planning when implants are being used, but overall MRI is the diagnostic imaging modality of choice. The authors prefer to perform both CT and MRI in those cases where surgical treatment is contemplated as the two imaging modalities are complementary.

Treatment and Prognosis

There are sufficient similarities between canine and feline DLSS to suggest that similar treatment approaches should be adopted. Early detection and treatment of DLSS, before progression to permanent faecal and urinary incontinence, is paramount for successful surgery. Mild cases with evidence of spinal pain and no evidence of neurological dysfunction may be treated conservatively with analgesia, weight control and permanent lifestyle changes. Surgery may be indicated in more severely affected cases and in cats that are not responding to conservative treatment. Surgical treatment has been described in a number of reports and involves either decompression by dorsal laminectomy or stabilization with or without dorsal decompression. Currently there are no objective criteria to assist in decision-making between conservative and surgical treatment or to decide between decompressive surgery alone or decompression accompanied by stabilization.

Decompression of the L7 nerve roots by foraminotomy has currently not been described in cats. Dorsal laminectomy alone is not a good choice for cats with evidence of neurogenic lameness associated with compression of either the L7 or S1 nerve roots as this will not be alleviated by this procedure. Dorsal anulectomy or partial discectomy is controversial in dogs, and in some canine studies performing the procedure either did not result in an improved outcome or actually resulted in a worse outcome. Discectomy may increase putative instability and may cause further collapse of the disc space and significant narrowing of the L7 neuroforamina. The authors only perform discectomy at L7-S1 when there is obvious dorsolateral protrusion of the intervertebral disc into either of the L7 neuroforamina. Traumatic L7 articular process fracture and spondylolisthesis have been described in a cat as a complication of dorsal laminectomy, anulectomy and discectomy.

Stabilization is often preceded by dorsal laminectomy and if necessary partial discectomy. The joint is then stabilized using pins or screws and bone cement. In the largest series of cases to date, which involved five cats affected by cauda equina syndrome as a result of DLSS, stabilization was achieved using miniature positive-profile pins inserted into the vertebral body of L7 and the wings of S1 with the ends of the pins embedded in polymethylmethacrylate. The outcome in all of the cats 2 years postoperatively was good, with complete resolution of signs of pain, and return to normal activity.

Spondylosis Deformans and Diffuse Idiopathic Skeletal Hyperostosis

(Bossens et al. 2016, Kranenburg et al. 2012, Morgan 1967).

Spondylosis deformans is a non-inflammatory degenerative condition affecting the peripheral region of the vertebral endplates that results in new bone formation (enthesophytes). The enthesophytes vary in size from small bone spurs to complete bony bridges that cross the intervertebral disc space (Figure 15.14). Severe spondylosis deformans should not be confused with diffuse idiopathic skeletal hyperostosis (DISH) which is a systemic disease of the skeleton that results in ossification of soft tissues including the ventral longitudinal ligament of the spine. There is currently only one report of feline DISH in a 9-year- old cat that presented with clinical signs of spinal pain and a T3-L3 myelopathy. CT revealed contiguous smooth new bone formation ventral and lateral to the vertebral column extending from the cranial thoracic area to the lumbosacral junction. The cat also had marked dorsolateral stenosis of the vertebral canal at the level of T4–T5 as a result of degenerative changes affecting the facet joints.

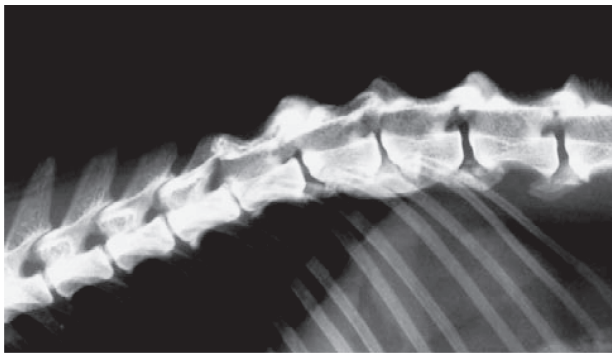


Figure 15.14 Spondylosis deformans. Lateral radiograph of the thoracolumbar spine showing extensive spondylosis deformans around the thoracolumbar junction. The radiographic changes were an incidental finding in this 11-year-old domestic shorthair.

Spondylosis deformans is a common incidental finding in cats, with a prevalence of 39.4% in a study of 402 cats aged one year and older. The aetiology is presumed to be the same as in dogs where it has been associated with degeneration of the anulus fibrosus of the intervertebral disc. However, the pathogenesis of spondylosis deformans does not include degeneration of the nucleus pulposus and disc extrusion and its presence at a disc space is therefore not proof of clinical disc disease at that site. Cats with neurological dysfunction and spondylosis deformans evident radiographically should be investigated thoroughly for other causes of spinal disease.

In the aforementioned study of 402 cats, the thoracic vertebrae T4 to T10 were most commonly affected by spondylosis, although the changes were most severe from T10 to S1. The cats with spondylosis were significantly older than those without radiographic evidence of spondylosis, and there was a female predisposition. No association was found between the severity of spondylosis and body weight. Cats with spondylosis of the lumbosacral region were not lame but there was a significant correlation with owner-perceived behavioural changes suggesting that they disliked being petted, which manifested as a reluctance to greet people and increased aggression. A proposed explanation for the behavioural changes is that a proportion of the cats had spinal pain associated with concurrent degenerative lumbosacral stenosis.

Degenerative Myelopathy

(Mesfin et al. 1980, Salvadori et al. 2003).

To the authors' knowledge there are only two reports of feline degenerative myelopathy. However, the condition may be more common than realized since definitive diagnosis requires histopathological examination of the spinal cord. In one report the history and clinical signs and the histopathological findings were similar to those described in German shepherd dog degenerative myelopathy, except that the pathology extended to the white matter of the medulla and the cerebellar peduncles. In the other reported case degenerative myelopathy was diagnosed in a cat with a clinical history of chronic pancreatitis. The suspected cause of the myelopathy was cobalamin deficiency secondary to pancreatic atrophy.

Spinal Dural Ossification

(Antila et al. 2013).

Spinal dural ossification, also termed ossifying pachymeningitis and osseous dural metaplasia, is a common finding in older dogs. The condition in dogs is usually incidental, but in rare cases has been associated with spinal hyperaesthesia and neurological dysfunction. There is a single reported case of the condition in a middle-aged ragdoll that presented with ambulatory tetraparesis and ataxia. MRI revealed cervical spinal cord compression by an intradural extraparenchymal lesion that was subsequently confirmed by histopathology as dural ossification. Surgical decompression resulted in a temporary improvement in clinical signs.

VASCULAR SPINAL DISEASE:

In a retrospective study of 205 cats with histologically confirmed spinal cord disease thrombosis or vasculopathy of unknown cause accounted for 9% of cases. In this study 15 of 19 older cats had ischaemic lesions (infarcts) or unexplained focal malacia affecting multiple areas of the spinal cord and in some cats the brain as well. Fibrocartilaginous embolic myelopathy is the most common cause of cause of ischaemic myelopathy, but embolization of spinal cord vasculature was not identified in any of the aforementioned cats. Other causes of ischaemic myelopathy include embolization of thrombi, parasitic or septic emboli, hypercoagulable states and vasculopathy. It is important to perform thorough investigation of all older cats with presumptive ischaemic myelopathy to identify and address underlying predisposing causes.

Fibrocartilaginous Embolic Myelopathy

(Abramson et al. 2002, De Risio 2015, MacKay et al. 2005, Mikszewski et al. 2006, Nakamoto et al. 2010, Scott & O'Leary 1996, Theobald et al. 2012, Turner et al. 1995, Zaki & Prata 1976).

Cause and Pathogenesis

Fibrocartilaginous embolic myelopathy (FCEM), a form of ischaemic myelopathy, is an uncommon spinal disease in the cat, the importance of which lies in its differentiation from other more frequent causes of peracute spinal cord injury. In an MRI study of 92 cats referred with clinical signs of spinal disease a presumptive diagnosis of FCEM was made in 6.5% of cases. The condition is not uncommon in the dog and has been reported in a number of other species, including humans. The pathogenesis involves infarction of the spinal cord as a result of embolization of spinal cord vasculature with fibrocartilaginous material histologically and histochemically identical to the nucleus pulposus of the intervertebral disc, resulting in ischaemic necrosis of dependent regions of spinal cord parenchyma. It is not known how the intervertebral fibrocartilage enters the spinal cord vasculature, although several theories have been proposed. The precise aetiology may differ between species or even between different individuals in the same species.

Clinical Signs

In published reports the domestic shorthair is the most frequently represented breed, there is a male to female ratio of 1.3:1, and the median age at diagnosis is 10 years (range 6

months to 17 years). The clinical presentation is of a peracute onset of neurological dysfunction referable to the site of embolization, ranging in severity from para or tetra paresis to plegia, dependent on the extent of spinal cord injury. Onset of neurological signs during physical activity such as running, jumping or playing is frequently reported. There is usually a complete absence of spinal hyperaesthesia and although any region of the spine may be affected, FCEM has been reported most frequently at the cervicothoracic intumescence (C6-T2). In the largest single study to date involving 19 cats lateralization of neurological signs was seen in 58% of cases.

Diagnosis

FCEM should be suspected in any cat irrespective of age that presents with a history of a non-progressive peracute onset of spinal disease where there is no clear history of trauma. Asymmetrical signs are common and in comparison with other causes of spinal disease, cats with FCEM are more likely to have a lesion localized to the C6-T2 spinal cord segments. FCEM can usually be easily differentiated from other more frequent spinal cord diseases such as neoplasia and inflammatory disease based on clinical and diagnostic findings.

Presumptive diagnosis is supported by the imaging features on high-field MRI (Figure 15.15), although distinguishing FCEM from peracute noncompressive intervertebral disc extrusion may be more problematic. Typically infarcted spinal cord appears as a focal, sharply demarcated and often lateralized intraparenchymal lesion that predominantly involves the grey matter and is hyperintense on T2w and isointense to hypointense on T1w images. MRI in dogs with FCEM performed 24–72 hours after the onset of neurological signs may reveal no signal intensity changes whereas MRI repeated more than 72 hours after onset may show a spinal cord intraparenchymal lesion. Diffusion-weighted MRI is more sensitive for the diagnosis of spinal cord infarction in the early stages of the disorder. A definitive diagnosis of FCEM can only be made post-mortem by histological examination of the affected spinal cord segments.

Treatment and Prognosis

Treatment entails good nursing care, bladder management and appropriate physical therapy as for any paretic or paralyzed patient. Restriction of physical activity beyond that required to ensure that the cat does not injure itself is not necessary. In dogs the prognosis is poor to good depending on the localization of the lesion and the extent of spinal cord injury. Absence of nociception and involvement of the cervical (C6-T2) or lumbosacral (L4-S3) intumescences are usually considered to be negative prognostic indicators in dogs. Prognostic indicators are less well defined in cats. Early reports of the disease involved individual cases, or small numbers of cats, many of which were euthanased soon after presentation because of a suspected poor prognosis. However, based on more recent studies the prognosis appears to be favourable in the majority of cats, even when plegia is present at the time of presentation.

In a study of six cats with FCEM all of the cats survived for at least 20 months following diagnosis, and improvement of clinical signs occurred in one cat with loss of nociception. In a more recent study of 19 cats a favourable outcome was achieved in 15 (79%) and all surviving cats with long-term follow up were reported to return to a normal quality of life. There is currently no evidence to suggest that involvement of either intumescence carries a worse prognosis in cats. In dogs the extent of intraparenchymal hyperintensity on sagittal and transverse T2w MR images is a prognostic indicator, and a similar association in cats is likely. Time to recovery of unassisted ambulation in cats with FCEM that were initially non-ambulatory has been reported to range from 2 to 27 days after diagnosis.

Ischaemic Myelopathy of the Cranial Cervical Spinal Cord

(Rylander et al. 2014, Simpson et al. 2014).

A subtype of ischaemic myelopathy with a predilection for the cranial cervical spinal cord has recently been reported in cats. In one study eight cats had a peracute or acute onset of symmetrical neurological signs with localization to the C1-T2 spinal cord segments, and an apparent predisposition of the ventral spinal artery at C2 or C3 to embolic occlusion. Neurological dysfunction ranged from ambulatory tetraparesis to tetraplegia with nociception present. A characteristic feature in six of the cats was marked ventroflexion of the neck. All of the cats were more than 12 years of age, and had at least one concurrent systemic disease including chronic renal disease, hypertrophic cardiomyopathy and hypertension.

In another study, five cats ranging in age from 11 years to 18 years had an initial acute onset of neurological signs of myelopathy and encephalopathy, three of which localized to the C1–C5 spinal cord segments. All of the cats improved initially but were eventually euthanased owing to recurrence of neurological signs. Histological examination revealed hyaline degeneration of the ventral spinal artery, basilar artery and meningeal arteries and infarction of the brain and spinal cord. It is not known how common

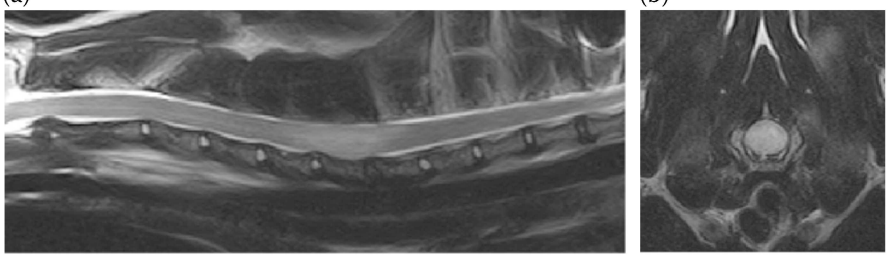


Figure 15.15 Fibrocartilaginous embolic myelopathy. **(a)** Sagittal T2 w MRI of an 8-year-old Bengal with peracute tetraparesis showing parenchymal (intramedullary) spinal cord injury at the level of the C6 vertebral body. **(b)** Transverse T2w MRI at the level of the lesion.

vascular hyaline degeneration is in older cats, but the condition should be considered as an underlying factor in older cats with ischaemic myelopathy.

The presentation of the cats in both reports is distinct from FCEM, which typically causes a single event with no concurrent medical conditions and has been reported in cats as young as 6 months of age. The short-term prognosis was favourable irrespective of neurological grade although a chronic relapsing course was seen in three of the cats presumably because the underlying medical conditions predisposed to recurrent ischaemic episodes.

TRAUMATIC SPINAL DISEASE:

Acute Non-Compressive Nucleus Pulposus Extrusion

(Chow et al. 2012, De Risio L 2015, Lu et al. 2002, Taylor-Brown & De Decker 2017).

This type of disc extrusion is now well described in dogs and has been reported in a number of cats. Synonyms for the condition include high-velocity-low volume disc extrusion, traumatic intervertebral disc extrusion, exercise-associated peractute thoracolumbar disc extrusion and Hansen type III intervertebral disc disease. The term acute non-compressive nucleus pulposus extrusion (ANNPE) is the preferred terminology and refers to the extrusion of hydrated non-degenerate nucleus pulposus due to a sudden increase in intradiscal pressure as a result of trauma or during vigorous activity. The hydrated nuclear material herniates through a tear in the dorsal anulus fibrosus, contuses the spinal cord and then dissipates within the epidural space causing a non-compressive myelopathy.

Dogs with presumptive ANNPE typically present with non-progressive peracute spinal cord dysfunction. Neurological signs may be lateralized and spinal hyperaesthesia is seen in approximately 50% of cases. The largest feline report of ANNPE to date involved 11 cats, two of which had asymmetrical neurological deficits. All of the cats had a peracute onset of signs and in almost 75% of cases there was either a confirmed traumatic episode or there was evidence of trauma based on clinical examination or imaging findings. The majority of cats presented with paraparesis or paraplegia and had a T3-L3 or L4-S3 neurolocalization. MRI findings compatible with ANNPE include focal T2w spinal cord hyperintensity overlying the affected intervertebral disc space, mild narrowing of the affected intervertebral disc space, and extraneous epidural material with absent or minimal spinal cord compression. The outcome with conservative treatment was good with almost 90% of the cats returning to ambulation, and faecal and urinary continence. The presentation of cats with ANNPE is similar to that of cats with fibrocartilaginous myelopathy (FCEM). ANNPE and FCEM should both be included in the differential diagnosis for any cat presenting with a peracute onset of spinal cord dysfunction. It is not currently known whether the prognosis differs significantly between the two conditions.

ANOMALOUS SPINAL DISEASE:

(Harris et al. 2019, Lee et al. 2014, Newitt et al. 2008, Watson et al. 1985).

Congenital anomalies of the vertebrae and spinal cord are uncommon causes of spinal disease in the cat. A variety of deformities resulting from a disturbance of the normal developmental processes may be found incidentally during routine radiographic examination. The range of anomalies is similar to those reported in the dog although they occur less frequently in the cat. The clinical significance of a vertebral anomaly depends on the nature and extent of the abnormality. Deformities that are generally asymptomatic include hemivertebra, butterfly vertebra, transitional vertebra (Figure 15.16), block vertebra (Figure 15.17) and abnormalities of the sacrum such as fusion of S3 and Cd1 or incomplete fusion of S2 and S3.



Figure 15.16 Transitional lumbosacral vertebra. Ventrodorsal radiograph of the pelvis showing a transitional L7 vertebra at the lumbosacral junction. There is unilateral sacralization with the right side of L7 fused to the ilium and sacrum. This was an incidental finding in this cat.

Transitional vertebrae occur at the junctions of the major divisions of the vertebral column and possess anatomical features from the two adjacent regions. Transitional

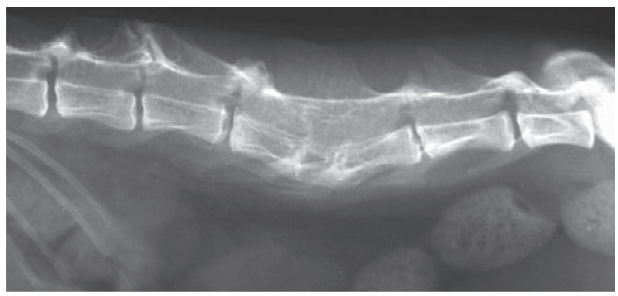


Figure 15.17 Block vertebra. Lateral radiograph of a lumbar block vertebra involving L4 and L5. (Courtesy of Malcom McKee.)

vertebrae at the cervicothoracic and thoracolumbar junctions are not associated with clinical signs whereas lumbosacral transitional vertebrae are a significant risk factor for degenerative lumbosacral stenosis. In cats where the lumbosacral transitional vertebra results in asymmetry or rotation of the sacroiliac joints there is an accentuation of degenerative changes in the lumbosacral disc, but in contrast to dogs, no association with hip dysplasia has so far been demonstrated. Hemivertebrae and block vertebrae rarely cause clinical signs in cats although there may be an increased risk of degeneration of the intervertebral discs adjacent to a block vertebra.

In a recent study, radiographs of 200 cats were reviewed to determine the prevalence of congenital vertebral anomalies. Congenital anomalies were identified in 46 cats. Transitional vertebrae were the most frequent abnormality and these were identified most at the sacrocaudal and thoracolumbar junctions. At the thoracolumbar junction thoracicization of L1 was more common than lumbarization of T13. Supernumerary ribs on L1 may be unilateral or bilateral, are often small and fragmented, and may be mistaken for rib fractures on radiographs. They can also cause confusion if the ribs are used for anatomical localization when performing surgical approaches to the spine at the thoracolumbar junction. Abnormalities at the lumbosacral junction were less common and included sacralization of L7 and less commonly lumbarization of S1. In all the cats with lumbosacral transitional abnormalities the angulation between the lumbar and sacral spine appeared increased and in those cats where ventrodorsal radiographs were available the transverse processes appeared to be asymmetrical.

Atlantoaxial Subluxation

(Dennis & De Risio 2009, Jaggy et al. 1992, Richter et al. 1983, Shelton et al. 1991, Thomson & Read 1996, Watson et al. 1985, Wessmann et al. 2015).

Craniocervical junction abnormalities such as subluxation of the atlantoaxial articulation are rarely reported in the cat. The few reported cases of atlantoaxial subluxation include two cats with occipitoatlantoaxial malformation and two cats with aplasia or hypoplasia of the dens. Suspected hyperflexion injury of the atlantoaxial joint in cats has also been reported.

Clinical signs of atlantoaxial subluxation are similar to those in the dog, ranging in severity from chronic neck pain to tetraparesis. The diagnosis of atlantoaxial subluxation is made radiographically based on the lateral view where there will be misalignment of, and an increased distance between, the dorsal arch of the atlas and the spinous process of the axis. An aplastic or hypoplastic dens may be detected on the ventrodorsal projection. Radiographic changes are absent with hyperflexion injury unless there is concomitant fracture, malformation or instability, and diagnosis is based on compatible MRI findings. Atlantoaxial subluxation can be treated using standard surgical techniques described in dogs, such as ventral stabilization with bone screws and polymethylmethacrylate. The condition should be included in the differential diagnosis for any cat with signs of a cranial cervical myelopathy.

Incomplete Ossification of Cervical Vertebrae

(Dean et al. 2019).

There is a single case report of a 21 week old kitten that presented with nonambulatory tetraparesis suspected to be caused by dynamic spinal cord compression secondary to multiple cervical vertebral malformations including incomplete ossification of cervical vertebrae. The cat returned to normal ambulation following 4 weeks of conservative management.

Spina Bifida

(Hall et al. 1988, Plummer et al. 1993, Ricci et al. 2011, Song et al. 2016, Wilson 1982).

Spina bifida is a type of neural tube defect in which there is incomplete closure or fusion of the dorsal vertebral arches over the spinal cord. There may be protrusion of the meninges (meningocele), or nervous tissue and meninges (meningomyelocele), through the vertebral defect. If the defect communicates with the skin surface, there will be leakage of CSF onto the skin and a risk of meningitis and electrolyte depletion. The terms occulta, in which there is no neural tissue involvement, and cystica, manifesta, or operta, in which there is an associated meningocele or meningomyelocele, are used to denote the amount of neural and soft tissue involvement. Concomitant anomalies include hydrocephalus and tethering of the spinal cord. Tethering of the spinal cord is a syndrome in which there is excessive stretching and tension on neural tissues resulting from abnormal attachments to the vertebrae or skin. Spina bifida is most common in the sacral region of the Manx cat but has been reported in the thoracic and lumbar vertebrae. Spina bifida not involving the spinal cord or cauda equina is not associated with any neurological signs.

The diagnosis of spina bifida is made radiographically. MRI or ultrasonography are required to confirm the presence of a meningocele or meningomyelocele. Surgery is indicated to close the meningocele and relieve spinal cord tethering, which may be of benefit to prevent worsening of neurological function including loss of urinary and faecal continence and progressive paraparesis. There is a report of successful surgical treatment of a Manx cat with a tethered spinal cord and an associated intradural extraparenchymal lipoma and meningocele.

Sacrocaudal Dysgenesis/Agenesis

(DeForest & Basrur 1979, Leipold et al. 1974, Ricci et al. 2011, Song et al. 2016).

Cause and Pathogenesis

The bobtail of Manx and Manx cross cats is an autosomal dominant trait that is also responsible for other vertebral and neural anomalies of the sacrocaudal region. The Cymric is a semi-long-haired variety of the Manx breed that suffers from the same anomalies. The homozygous trait is lethal; clinically affected cats are heterozygotes with variable expression. The so-called Manx cat is not a pure breed and the morphology of the sacrocaudal region in a litter of kittens may range from absent caudal vertebrae ('rumpies'), a tail stump with several caudal vertebrae ('rumpy risers'), a short tail with several mobile caudal vertebrae ('stumpies'), and a normal length tail ('longies').

The most frequent vertebral malformation, apart from caudal dysgenesis, is sacral dysgenesis but spina bifida may also occur (Figure 15.8). Spinal cord abnormalities include absence or partial development of sacral and caudal spinal cord segments or the cauda equina, myelodysplasia (disorganization of grey and white matter), myeloschisis (cleft within the spinal cord), diastematomyelia (duplication of the sacral segments), syringomyelia/hydromyelia in the



Figure 15.18 Sacrocaudal dysgenesis and spina bifida. Ventrodorsal radiograph of the pelvis of a Manx cat with sacrocaudal dysgenesis and sacral spina bifida. (Courtesy of Malcolm McKee.)

lumbar and sacral spinal cord segments, shortening of the spinal cord, tethered cord syndrome, meningocele, meningomyelocele and dermal sinus tract.

Clinical Signs

The spinal cord and cauda equina malformations are associated with a range of neurological deficits. Rumpies are most likely to display an abnormal gait and urinary and faecal incontinence and have the most severe underlying congenital anomalies. A bunny-hopping gait is considered a normal characteristic of the breed and even cats with no abnormality of the sacrum have a degree of myelodysplasia. Stumpies with deformed caudal vertebrae are prone to degenerative joint disease affecting the tail. Cats with sacrocaudal dysgenesis have more severe clinical signs reflecting abnormal development of the nerves of the cauda equina including urinary and faecal incontinence, and loss of tone, reflexes, and nociception in the anal and perineal region. In severe cases kittens may present at 3–6 weeks of age with paraparesis and urinary and faecal incontinence.

Diagnosis

Breeders of Manx cats recognize severely affected kittens early in life and euthanase them. The diagnosis of less severely affected kittens can be confirmed by radiographic examination, and MRI will reveal concurrent spinal cord abnormalities such as meningocele or tethered cord syndrome. The severity of the neurological signs does not always correlate with the apparent degree of spinal deformity.

Treatment and Prognosis

There is no specific treatment and the prognosis for cats with severe neurological impairment is poor. Meningocele and tethered cord syndrome should be corrected surgically in cats with minimal neurological dysfunction. Breeders should be urged to stop selecting for absence of the tail in Manx cats because of the unacceptable congenital malformations and neurological dysfunction that accompany this trait.

Syringomyelia and Hydromyelia

(Bone & Wilson 1982, Bagley et al. 2000, Okada et al. 2009).

Hydromyelia and syringomyelia are terms used to describe cystic fluid-containing cavities within the spinal cord. The fluid is similar, if not identical, to CSF. Hydromyelia is a dilation of the spinal cord central canal, which is lined by ependymal cells and often communicates with the ventricular system. The term syringomyelia refers to cystic cavities within the substance of the spinal cord. In practice the two may be difficult to differentiate and the terms syringohydromyelia or syringomyelia are often used to encompass both conditions. The clinical signs reflect spinal cord dysfunction and depend on the size of the lesion, the rate of onset and the region of spinal cord involved.

Syringohydromyelia may be primary, occurring in association with other congenital anomalies of the spinal cord, or may be secondary to compressive myelopathies or inflammatory disease such as feline infectious peritonitis. In dogs Chiari-like malformation is the most common cause of syringohydromyelia. Chiari-like malformation is rare in cats and is seen mainly in brachycephalic breeds such as the Persian. In a recent report two young domestic shorthair cats with Chiari-like malformation presented with neurological signs consistent with a spinal cord (C1–C5) neurolocalization. Neither of the cats had syringohydromyelia but based on the findings in this report Chiari-like malformation should be included in the differential diagnosis of any young cat presenting with clinical signs of a C1–C5 myelopathy.

Cervical syringohydromyelia has been reported in four cats secondary to intracranial pathology that resulted in either a disturbance in the flow of CSF or increased intracranial pressure. In the past hydromyelia was sometimes accidentally diagnosed during routine myelography when contrast medium entered a dilated central canal. In many cases, however, myelography was normal or showed only intraparenchymal enlargement of the cord. Definitive diagnosis is best made using MRI. Although rarely reported in the cat, these conditions are being recognized more frequently with the increased availability of MRI. Treatment of primary syringohydromyelia may be attempted by shunting or surgical drainage of the syrinx but the prognosis is often poor. Treatment of secondary syringohydromyelia should be directed at the underlying cause.

Spinal Arachnoid Diverticulum

(Adams et al. 2015, Galloway et al. 1999, Schmidt et al. 2007, Shamir et al. 1997, Vignoli et al. 1999).

Intradural spinal arachnoid diverticula are focal dilations within the meninges, containing an accumulation of cerebrospinal fluid, which can lead to a progressive compressive myelopathy. Synonyms for this condition include arachnoid cyst, subarachnoid cyst, meningeal cyst and leptomeningeal cyst. The condition is well known in humans and is increasingly being reported in dogs, but there are few reported cases in the cat. In dogs spinal arachnoid diverticula are equally distributed between the cervical and thoracolumbar regions of the spine. In contrast, with one exception, all of the diverticula reported in cats have been located in the thoracolumbar region.

The condition should be included in the differential diagnosis for any cat with progressive spinal cord dysfunction, especially if this localizes to the thoracolumbar region. Definitive diagnosis requires myelography or advanced imaging techniques. The MRI appearance is characterized by a bulbous expansion of the subarachnoid space usually in the dorsal mid-line with attenuation of the subadjacent spinal cord. The lesion is hyperintense to grey matter on T2w images and hypointense on T1w images. The pathogenesis of the condition is not fully understood but arachnoid diverticula may be broadly classified as either congenital or acquired. There is a report of an acquired diverticulum in the cervical spine of a cat apparently secondary to a previous acute non-compressive nucleus pulposus extrusion. Treatment of arachnoid diverticula by decompressive dorsal laminectomy and drainage of the contents with or without marsupialization is likely to be successful.

Vertebral Angiomatosis

(Frizzi et al. 2017, Hans et al. 2018, Kloc et al. 2001, Schur et al. 2010).

Angiomatosis is a rare developmental vascular malformation characterized by the formation of multiple non-neoplastic tumours that form from blood vessels (angiomas). The pathogenesis is not fully understood but the condition appears to be a solitary intraosseous congenital vascular malformation with arteriovenous shunts and without systemic involvement. The vascular lesion arises in a vertebra and extends into the vertebral canal, causing compression of the spinal cord. All cases have been less than 2 years of age and have presented with spinal hyperaesthesia and progressive ataxia and paresis with localization to the thoracolumbar spinal cord segments T3-L3. Most cases have involved the thoracic spine with only one reported case affecting a lumbar vertebra.

The radiographic appearance shows a poorly circumscribed mixed pattern of bone proliferation and bone lysis usually involving the pedicle of a single vertebra. The condition should be included in the differential diagnosis for any young cat with a T3-L3 myelopathy and an apparently aggressive thoracic vertebral bone lesion, especially if the vertebral pedicles are involved. A presumptive diagnosis can be achieved based on the signalment and advanced imaging. Definitive diagnosis requires histological examination which shows a poorly defined vascular proliferation extending through the bone of the vertebra.

The response to surgical resection and decompression alone can be favourable with no clinical evidence of recurrence in a recent report of two cases, one year and 1.5 years after surgery. In another recent report a cat underwent surgical decompression and adjuvant radiation therapy resulting in an excellent outcome, with no evidence of recurrence on MRI, 26 months after the initial diagnosis.

Spinal Dermoid Sinus

(Fleming et al. 2011, Henderson et al. 1993, Kiviranta et al. 2011, Song et al. 2016, Tong & Simpson 2009).

A spinal dermoid sinus, also termed a dermoid cyst or pilonidal sinus, arises because of failure of the skin to separate completely from the neural tube during embryonic development. This leads to the formation of a tubular tract of skin, of varying depth, extending from the subcutaneous tissue towards the vertebral canal. These sinuses may form a small opening to the exterior usually in the dorsal midline marked by a tuft of hair. Neurological signs have been reported either as a result of tethering of the spinal cord or when the sinus communicates with the subarachnoid space and becomes infected.

Dermoid sinuses occur most commonly in the Rhodesian ridgeback as a heritable condition, but the condition has also been reported in a small number of cats including three Burmese, one Balinese and a domestic shorthair. In four cases the sinus was in the cranial thoracic region and the cats had neurological signs referable to a tethered spinal cord. In the domestic shorthair the sinus was at the level of the atlas. Diagnosis is best made using MRI and treatment involves complete surgical excision of the sinus tract. In view of the apparent Burmese breed predisposition to the condition it may be worthwhile palpating young Burmese kittens, at the time of vaccination, for the presence of a dermoid sinus or raised dermal papule between the scapulae.

METABOLIC SPINAL DISEASE:

Lysosomal Storage Diseases

(Haskins et al. 1981, Lyons et al. 2016, Skelly & Franklin 2002).

Cause and Pathogenesis

Lysosomal storage diseases are a diverse group of metabolic diseases affecting multiple body systems linked by the common pathological feature of abnormal intracellular accumulation of storage material. They are rare congenital disorders caused by a deficiency of one or more enzymes necessary for the hydrolysis of proteins, polysaccharides and complex lipids. The enzymes are present in lysosomes, hence the collective term of lysosomal storage diseases. The lysosomes provide a suitable environment for enzyme-catalyzed hydrolysis, and keep other cellular structures protected from the deleterious effects of unconfined degradative enzymes. All of the lysosomal storage diseases of cats are inherited as autosomal recessive traits. They are classified into subgroups on the basis of the metabolic pathway affected and the type of storage found. The main subgroups are the glycoproteinoses, the sphingolipidoses, the mucopolysaccharidoses and the proteinoses.

Clinical Signs

The enzyme deficiency leads to accumulation of metabolite, causing a wide array of clinical signs. Neurological signs usually develop in the immature animal in the first few months of life and are progressive over the first year. However, delayed onset diseases have been reported, for example ceroid lipofuscinosis, and their prevalence is probably underestimated. Clinical signs include paraparesis or tetraparesis often with signs of cranial involvement such as seizures, intention tremor, and blindness. Sphingomyelinosis (Niemann–Pick disease) is associated with a

deficiency of the enzyme sphingomyelinase, which results in a demyelinating polyneuropathy (see Chapter 17).

Mucopolysaccharidosis VI (Maroteaux–Lamy disease) is due to a defect in the metabolism of mucopolysaccharides (glycoaminoglycans) and is seen predominantly in Siamese cats but has also been reported in the Birman, ragdoll and domestic shorthair. It is one of the most commonly diagnosed lysosomal storage diseases in cats. Different DNA mutations are associated with a varying severity of clinical signs. Cats present at 2–6 months of age with progressive paraparesis associated with spinal cord compression. Affected kittens are smaller than normal littermates and have characteristic physical deformities, including a broad face with small ears, shortened nose, corneal opacification and pectus excavatum. Skeletal abnormalities include fusion of vertebrae and bony proliferation, with protrusion into the thoracolumbar vertebral canal and the intervertebral foramina, causing spinal cord and nerve root compression.

Diagnosis

Lysosomal storage disease should be included in the differential diagnosis for any young cat with progressive multifocal neurological disease, particularly if inflammatory disease has been excluded. Diagnosis of mucopolysaccharidosis VI is based on a PCR assay coupled with DNA pyrosequencing. The assay can be used to identify affected and carrier cats and should enable breeders to eliminate the disease from purebred cats in the future.

Treatment and Prognosis

There is no effective treatment for any of the lysosomal storage diseases but progression of neuronal storage may be slow and some cats may survive months to years after diagnosis. The skeletal changes seen in mucopolysaccharidosis VI are nonprogressive after physeal closure at around 9 months of age and decompressive surgery may improve the neurological status of these cats.

NUTRITIONAL SPINAL DISEASE:

Hypervitaminosis A

(Polizopoulou et al. 2005).

Hypervitaminosis A is a rare nutritional disorder caused by an excessive intake of vitamin A. The condition frequently causes cervical spinal lesions and is discussed in Chapter 19.

MISCELLANEOUS SPINAL DISEASES:

Dorsal Spinous Process Impingement

(Guiterrez-Quintana et al. 2011).

There is a single report of a middle-aged Persian cat with dorsal spinous process impingement syndrome ('kissing spine'). The cat presented with a history of chronic progressive thoracolumbar spinal pain. Radiographs revealed close approximation of the dorsal spinous processes of the mid-thoracic vertebrae, accompanied by moderate spinous process sclerosis and new bone proliferation. Surgical resection performed to eliminate contact between the affected spinous processes was curative.

Epidural Dirofilariosis

(Favole et al. 2013).

There is a single report of a middle-aged cat presented with progressive nonambulatory parapresis and ataxia caused by aberrant epidural localization of an adult female heartworm (Dirofilaria immitis). MRI identified an epidural mass lesion that was successfully removed surgically.

Extraparenchymal Spinal Cysts

(Aikawa et al. 2014, Gutierrez-Quintana et al. 2014, Lowrie et al. 2014).

A cyst is defined as a closed sac or capsule lined by epithelium that contains a liquid, gaseous or semi-solid substance. Spinal cysts in dogs have been classified according to location with a sub-classification according to pathological and surgical findings. There are few reports of true spinal cysts in cats, and although arachnoid cysts have been reported a number of times in cats, the use of the term cyst is incorrect, they should be termed diverticula. Perineural or Tarlov cysts are a type of meningeal cyst involving the dorsal nerve roots that have currently not been reported in cats.

Intraspinal cysts are non-meningeal cysts that have been reported in cats; they have also been termed juxta-articular or juxta-facet cysts. These cysts arise in an extradural location from periarticular tissue, and less commonly the ligaments of the vertebral canal and intervertebral discs. Their pathogenesis is not fully understood but has been linked to degenerative disease and trauma. Two types of intraspinal cyst have been described based on pathological findings although it has been suggested that they represent the same entity at different stages of development. One has a synovial lining containing fluid and is known as a synovial cyst whereas the other contains myxoid material with no specific lining and is known as a ganglion cyst. The terms used for these cysts are descriptive and hold no clinical significance.

There are two reports of cats with intraspinal cysts arising from the composite occipito-atlanto-axial joint cavity. In one case the cat presented with a long history of episodes of ataxia and tetraparesis. MRI revealed a cystic lesion at the occipito-atlanto-axial region causing dynamic spinal cord compression on flexion of the neck. In the other report the cat presented with progressive ambulatory tetraparesis and ataxia. MRI revealed a cystic lesion at the occipito-atlanto-axial region causing dynamic spinal cord compression on extension of the neck. Histopathological findings in this case were consistent with a ganglion cyst.

DISORDERS OF THE TAIL:

(Batle et al. 2019, Pollard et al. 2015).

The normal tail is comprised of 18–20 caudal vertebrae. Several cat breeds are defined by morphological abnormalities of the tail, and either have no tail (anury), or have a tail comprised of fewer vertebrae that is shortened or bobbeb (brachyury). Other abnormalities include tail kink, which is a congenital deformity in which there is a kink, usually in the distal third of the tail. Tail kink was formerly common in the Siamese and Burmese but has largely been eliminated from the modern breeds. Surgical correction of the tail kink is not indicated and is fruitless if it is attempted.

Bobbed breeds include the Kurilian bobtail, American bobtail, Pixie bob, Highlander and Japanese bobtail. Vertebral morphology and genetics of the condition in the Japanese bobtail have been reported. In this breed the tail abnormalities have an autosomal dominant mode of inheritance with variable expression. Vertebral anomalies in the Japanese bobtail cat include a reduction in the number of caudal vertebrae, caudal hemivertebrae, transitional vertebrae and an abnormal vertebral formula with a reduced number of vertebrae. The range of tail abnormalities seen in

Manx, Cymric and Manx-cross cats is discussed in the section on sacrocaudal dysgenesis/agenesis. In the opinion of the authors the deliberate production of any tailless cat is a welfare issue as cats use their tails for balance and communication with other cats.

Self-trauma to the tail may be a component of feline hyperaesthesia syndrome. Clinical signs include tail chasing and mutilation of the tail, skin rippling and muscle spasms over the dorsal lumbar region, and biting or licking the lumbar and perianal regions and the flanks. Episodes may occur spontaneously or may be elicited by light touch. Other clinical signs such as episodic and apparently uncontrolled running and jumping and vocalization are frequently seen. Although current understanding of the condition is incomplete, there may be a favourable response to a number of different therapeutic approaches. In a study of seven cats with feline hyperaesthesia syndrome a putative diagnostic pathway for the condition was proposed by the authors.

Cat bite abscesses involving the tail are common and occasionally result in vascular compromise or osteomyelitis, which is generally best treated by partial amputation. Traumatic injury to the tail is also common. Causes include road traffic accidents, bites, metallic projectiles, malicious injury and entrapment in doors. Concurrent soft tissue injury is common and many fracture/luxations of the middle and distal thirds of the tail are open. Surgical reduction and stabilization of the osseous component of tail injuries are rarely indicated. In cases where there is instability, sufficient immobilization of the caudal vertebrae can generally be afforded by simple bandaging techniques or cage rest. For more severe injuries, in cases where there is vascular compromise or paralysis of the tail with marked displacement of caudal vertebrae, partial or total amputation is indicated. Surgical reduction and stabilization of the tail base either alone or in conjunction with more distal amputation may be indicated in cases where the proximal caudal vertebrae impinge on the rectum or anus. A careful assessment of bladder function and anal tone should be made in all cats with proximal caudal vertebral fracture/luxations. Sacrocaudal fracture luxation (tail-pull injury) is discussed in Chapter 15.

Tail Amputation

Partial or complete amputation of the tail is performed more frequently in cats than dogs. Indications include traumatic injury, neoplasia, loss of sensory or motor function, severe soft tissue and/or bone disease and degloving or vascular injuries to the tip of the tail. Total amputation will adversely affect fine balance until the cat learns to compensate for loss of the tail. To perform amputation the skin is manually retracted proximally over the desired site of amputation generally leaving a 20–30 mm margin of healthy tissue around traumatic, infected or neoplastic lesions. U shaped incisions are made in the skin dorsal and ventral to the amputation site leaving sufficient skin to cover the end of the amputated tail (Figure 15.19). The medial and lateral superficial caudal arteries are isolated and ligated or diathermy is used to achieve haemostasis. The soft tissues are incised and the tail is amputated at the most appropriate intervertebral disc space. Closure of soft tissues over the amputation site is performed routinely and the skin is sutured without any tension preferably using absorbable intradermal or skin sutures so that suture removal is not required. Bandaging is not necessary but an Elizabethan collar is usually required.

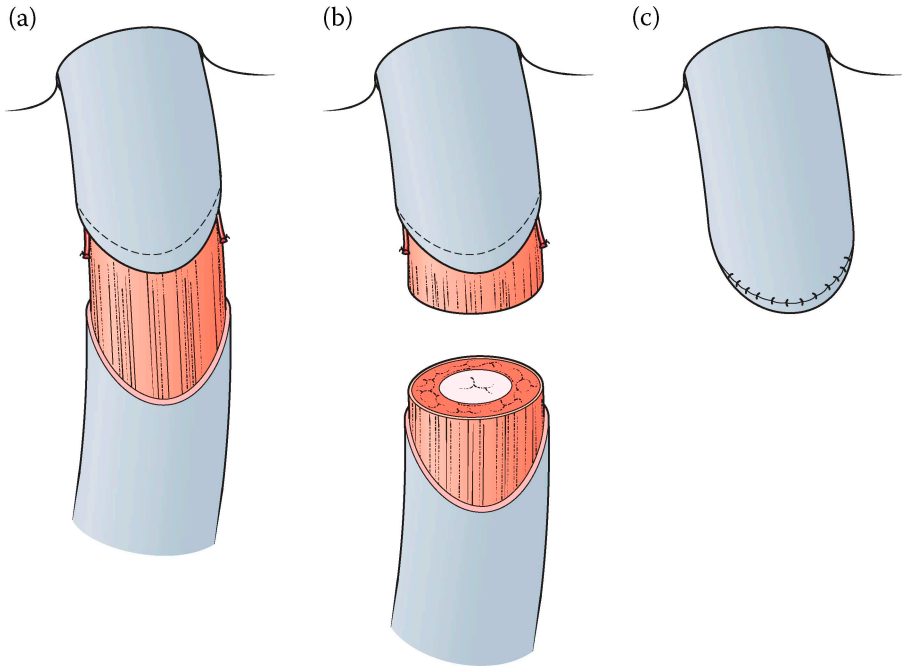


Figure 15.19 Tail amputation **(a)** The skin is manually retracted proximally over the desired site of amputation and U shaped incisions are made in the skin dorsal and ventral to the amputation site. The skin flaps should extend beyond the site of disarticulation. The medial and lateral superficial caudal arteries are isolated and ligated or diathermy is used to achieve haemostasis just proximal to the joint. **(b)** The soft tissues are incised and the caudal vertebrae are disarticulated at the appropriate intervertebral disc space. **(c)** The dorsal skin flap is pulled over the stump and sutured to the ventral flap avoiding any tension in the repair.

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16 Physiotherapy and Rehabilitation

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INTRODUCTION:

(Bennett & Morton 2009, Grant 2006, Lascelles et al. 2010).

Physiotherapy (or physical therapy) can be defined as the treatment of disease, injury, or deformity by physical methods such as massage, heat treatment, and exercise. The term 'physiotherapy' is derived from Greek and literally means 'natural treatment or healing'; the word 'rehabilitate' is derived from Latin and means 'make fit again'.

The goals of feline rehabilitation are to restore function and mobility, and to improve quality of life. This is achieved by stimulating the healing process, restoring the strength of injured tissues, and improving balance and coordination. When performed correctly physical therapy, and a number of the modalities used in feline rehabilitation, can help to alleviate pain and prevent permanent physical disabilities. Physiotherapy can be used as a component of immediate postoperative care, to promote early controlled weight-bearing, to help restore normal gait, for the management of acute and chronic pain, and for the conservative management of selected conditions.

Compared to dogs, cats tend to have fewer developmental orthopaedic disorders and are therefore more likely to require rehabilitation following traumatic injuries to the musculoskeletal system, and for neurological disorders. Nonetheless, cats resemble dogs in that they are commonly affected by osteoarthritis (OA) For a full discussion of feline OA see chapter 7. Studies have shown that cats in the age range from 6 months to 20 years have at least one joint with radiographic signs of OA. The condition in cats can often go unnoticed, which is in part due to cats not exhibiting the same clinical signs as dogs when in chronic pain. Rather than lameness it is often a change in the behaviour or lifestyle of the cat that arouses the owner's concern. It is very important to obtain a comprehensive history from the owner to ascertain the patient's normal behaviour and therefore be able to recognize any pain-related changes.

The independent nature of cats has inevitably meant they are not often considered for rehabilitation. Cats also differ from dogs in that they are not motivated by food rewards and as rehabilitation continues, cats can become more anxious and less compliant. Rehabilitation of this species is therefore challenging, however, if handling is adapted to the cat's disposition and physical therapy is presented in short episodes it can be very worthwhile (Figure 16.1).

BENEFITS OF REHABILITATION:

The guiding principle of any rehabilitation programme, is to return the patient to full potential where possible, while reducing the risk of further injury. When formulating a plan, consideration of how productive and safe it will be to administer, is key to a successful outcome. The modalities and exercises selected should be tailored to the individual patient being treated. The key areas of rehabilitation, centre on muscles, bones, and joints. Rehabilitation is aimed at developing strength, improving endurance, restoring range of motion and enhancing proprioception.

Rehabilitation can be divided into *passive* and *active* rehabilitation and *proprioceptive* training exercises. Passive treatments require that the patient is compliant to manual therapy; these include passive range of movement (PROM), stretching and massage, electro-physical and thermal treatments (thermotherapy). Active rehabilitation requires the cat to move in a specific way using a range of equipment. Active therapeutic exercises require that the patient actively participates in gait training, therapeutic exercises and aquatic therapy. Supplementary to active rehabilitation are proprioceptive therapeutic exercises that provide sensory feedback whilst performing static or active exercises.

PASSIVE REHABILITATION:

(Drum et al. 2015, Millard et al. 2013, Millis et al. 2005).

Passive rehabilitation techniques include thermotherapy (cold and heat), manual therapies and modalities. These include modalities to reduce heat, pain, swelling, and oedema from the effects of trauma to the tissues. Passive rehabilitation can be initiated in the immediate postoperative period.

Once the patient has overcome the immediate effects of surgery or trauma, a physical therapy plan can be employed. Careful consideration should be given to choosing the appropriate therapeutic modality to achieve the relevant effects based on an understanding of how the modality chosen may facilitate or hinder a positive outcome. Selection of the appropriate modality depends largely on an understanding of, and accurate assessment of, the stages (acute inflammatory, proliferative, tissue remodelling) of tissue healing.

Cold Therapy (Cryotherapy)

Cold therapy initiates vasoconstriction and therefore reduces haemorrhage after injury or surgery. Cold also reduces cell metabolism, decreases nerve conduction velocity, and helps to alleviate pain. It decreases impulse conduction velocity and stimulates cold receptors, thereby activating the gate control system via neural mechanisms. Application of cold therapy will therefore reduce blood flow to an area, reduce inflammation, facilitate the reduction of swelling, and alleviate moderate pain. Cryotherapy is most effective during the



Figure 16.1 Handling a cat using minimal restraint.

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acute inflammatory stages of tissue healing and is used in the acute stages during the first 72 hours postoperatively. It is also useful after exercise and for the treatment of acute flare-ups of OA as a form of analgesia.

Cryotherapy is a superficial treatment; skin temperature will show a 15° Celsius drop within 15 minutes after application of a cold pack. The depth of cold penetration depends on the amount of adipose tissue and local blood flow. Subcutaneous tissues will take over 30 minutes to show a similar temperature drop and muscle will take much longer.

Cats often resent cold application, particularly gel packs, which if used, should always be wrapped in a thin damp cloth. Ice cubes in water with the application of a cloth dipped into this cold solution (iced towels) is a more appropriate option. Once the cat has become accustomed to the iced towels, ice cubes can be wrapped in the cloth then applied to the target site (Figure 16.2).

Postoperatively cryotherapy can be applied repeatedly, the duration of the treatment time depending upon compliance of the patient. The optimal amount of time is 5–10 minutes to gain the benefits of cold application. When applying cold therapy, it is important to check the area being treated repeatedly, especially if the cat shows signs of discomfort. Contraindications for cryotherapy include anaesthesia, paraesthesia and circulatory disorders affecting the area being treated.

Contraindications for cryotherapy:

- Dermatitis
- Advanced cardiovascular disease
- Local areas of impaired circulation
- Tumours
- Open or infected wounds
- Local areas of absent, impaired or altered sensation

Heat Therapy

The beneficial physiological effects of heat are, increased circulation (vasodilation), pain relief, increased soft tissue extensibility and relaxation of muscle spasm. Heat increases the release of mediators, such as histamine, bradykinin and prostaglandins, resulting in the dilation of blood vessels and an increase in the metabolic rate of tissues. Heat can also be effective for increasing the extensibility of collagenous tissues, such as ligaments, tendons, joint capsule and scar tissue. Heat is mainly used to treat chronic conditions and to warm an area of the body before active and passive exercise or massage.



Figure 16.2 Cold therapy using ice cubes and a damp cloth.

Superficial heat therapy penetrates to a depth of approximately 10–20 mm. It is often applied for subacute and chronic conditions, to relieve pain and muscle spasm and to aid passive movements. Heat can help to improve flexibility of a joint with OA. Heat can also be used for an infected site or abscess to boost the suppurative process and promote drainage. Other benefits of superficial heat therapy may include reducing oedema and increasing patient comfort. However, heat should not be used during the acute inflammatory phase because oedema may be exacerbated.

Gel packs are commonly used wrapped in a damp cloth, applied for 10–15 minutes, several times daily. Heat should not be applied at a surgical incision site for at least 72 hours postoperatively. If the area around the incision remains hot and swollen it is better to avoid the application of heat. Cats in general, tolerate heat well for short periods of time. A swaddling technique is recommended, using a towel as a low-stress restraint technique, for hot pack application.

Contraindications for heat therapy:

- Acute inflammation
- Tumours
- Open or infected wounds
- Advanced cardiovascular disease
- Local areas of impaired circulation
- Open or infected wounds
- Devitalized tissue, e.g. after radiotherapy

Massage

There are special considerations when performing massage on cats. The techniques described in Table 16.1 will not be tolerated by all cats. Minimal restraint techniques are best to achieve low-stress sessions.

Therapeutic massage is the manipulation of the soft tissues of the body. Massage improves circulatory blood and lymphatic flow in the musculoskeletal and nervous systems. Massage provides relief from pain, reduces tension and helps to calm the nervous system. When a cat is confined post-surgery, massage can help to maintain mobility and prevent tissue adhesions. It also helps psychologically by maintaining contact between the cat and the owner. By encouraging blood flow, delivery of nutrients and maintenance of soft tissue extensibility, massage is also beneficial in the treatment of cats with OA.

The physiological and psychological effects of massage:

- Reduces anxiety, improves relaxation
- Provides afferent input - both sedative and stimulatory
- Pain control via the ‘pain gate’ mechanism
- Releases endorphins
- Improves circulatory systems (venous and lymphatic)
- Promotes removal of noxious chemicals

Table 16.1: Benefits of Different Massage Techniques

Technique	Major Benefits	Other Benefits
Stroking	Accustoms cat to touch Reduces tension and anxiety Lowers muscle tone	Useful for the beginning and end of a massage
Effleurage	Reduces swelling and oedema. Start from distal area move proximally using medium pressure	Moves toxins towards lymph nodes and aids lymphatic drainage
Petrissage	Increases circulation. Applies compression to underlying tissues	Relaxes and lowers muscle tension. Enhances sensory stimulation
Friction	Breaks down adhesions	Increases blood flow (hyperaemia)
Percussion	Stimulates circulation. Provokes muscle and tendon reflexes. Stimulates muscle tone. Clears airways (coupage)	
Compression	Compression can be used on large muscles	Restores mobility between tissue surfaces
Shaking	Increases circulation	
Vibrations	Loosens chest secretions, stimulates coughing	

Passive Movements

(Jaeger et al. 2002, Jaeger et al. 2007, Lascelles et al. 2012, Norkin & White 2009, Norkin & Levangie 1992, Sprague 2013).

Gross movement of bones at joints is termed osteokinematics and can occur in a number of different directions around the joint axis or centre of rotation. These include flexion, extension, abduction, adduction, internal rotation and external rotation Movement of joint surfaces with respect to one another is termed arthrokinematics and includes rolling, gliding and spinning. Joint motion can be passive or active.

Passive range of movement (PROM) exercise is used when a patient is incapable of moving a joint on its own because of a neurological or orthopaedic disorder. Reduced range of movement often occurs secondary to prolonged immobilization or inactivity. It is essential to start some gentle manipulation of a joint as soon as possible to maintain joint mobility, to avoid contractures and for neuromuscular re-education. Range of movement can be restricted by a number of structures and conditions. These include tissue swelling, muscle, tendon and ligament disorders, periarticular fibrosis, pain, meniscal injury, and joint mice. The source of limitation can often be determined by assessing end-feel at the end stage of joint movement.

Initial evaluations using a baseline of goniometric guidelines are used when assessing a cat for orthopaedic and neurological disorders. Table 16.2 illustrates the normal ROM of joints in the cat. Passive range of movement exercise (PROM) allows for evaluation of existing ROM and serial measurements are then made to monitor for improvement as a result of therapy over a period of time. Benefits of PROM include establishing joint stability and increasing soft tissue elasticity.

The term "end-feel" is the subjectively assessed sensation imparted to the examiner's hands at the end of the range of joint motion. An awareness of the end-feel of a joint provides additional information about the passive motion of a joint (Table 16.3). Assessment of end-feel is performed by slow application of moderate pressure at the end of the range of joint movement and noting the character of resistance. Some joints will be painful through their normal ROM, whereas with others pain may only be elicited by stressing the joint at the end of the normal range, either in flexion or extension. An equivocal pain response should be repeatable to ensure that it is genuine and comparison should be made with the contralateral joint and other normal joints. Potentially painful joints should always be examined last to avoid loss of patient compliance. Joints should also be tested for abnormal motion such as rotation, and medial and lateral instability, where these types of motion are not normally expected.

Passive exercises are performed without active muscle contraction by the patient. They help to maintain or improve ROM of joints, improve flexibility of muscles, tendons, and ligaments, and help enhance awareness of neuromuscular structure and function. Manual contact or hand placement is critical when performing PROM exercises. Ensure the distal bone is moved on a stabilized proximal bone. It is important

Table 16.2: Range of Motion (Degrees) of Various Joints Measured by Goniometry in Cats

Joint	Position	Mean
Carpus	Flexion	22
	Extension	198
	Valgus	10
	Varus	7
Elbow	Flexion	22
	Extension	163
Shoulder	Flexion	32
	Extension	163
Tarsus	Flexion	21
	Extension	167
	Valgus	7
	Varus	10
Stifle	Flexion	24
	Extension	164
Hip	Flexion	33
	Extension	164

Table 16.3: Interpretation of End-Feel

End-Feel	Sensation	Interpretation
Bony/hard/unyielding	Bone approximates bone resulting in an abrupt stop	Always pathological May be indicative of extensive periarticular oestophytosis
Capsular/firm	A firm but slightly yielding sensation associated with tension in the joint capsule	Normal for many joints e.g. carpal extension Abnormal if associated with reduced range of motion Pathological capsular end-feel is usually firmer than normal capsular end-feel
Soft tissue approximation	Motion is halted by compression of soft tissues or fluid resulting in a reduction in range of motion with soft end-feel	Normal for some joints e.g. stifle flexion Abnormal if it occurs too early in the range of motion or in a joint normally having a capsular end-feel May be seen in joints with substantial effusion or periarticular oedema
Empty/painful	Resistance is not felt because the patient resists full range of joint motion	Joint pain is always pathological

to look for any cues of patient discomfort. If the patient splints (actively contracts muscles), the movement is no longer passive.

Range of motion may be limited by pain or a mechanical problem such as contracted muscle, ligament or tendon restriction. It is important to measure the unaffected contralateral joint as a comparison when measuring the angles of any joint, although allowance should be made for bilateral disease. Sedation will make the measurements easier to perform but it also eliminates the possibility of being able to accurately assess the cat for a painful response to joint manipulation.

Passive range of movement maintains flexibility but will **not** reverse existing muscle atrophy or increase muscle strength. When treating a limb, it is better to start PROM of individual joints distally with approximately 10–20 repetitions, two to three times daily. Comfortable soft padding should be provided if the exercises are performed with the cat placed in lateral recumbency. Alternatively, some cats will prefer to stand in a comfortable position on a non-slip surface. It is important when performing range of movement to avoid causing pain with aggressive movements, which will result in reflex inhibition, delayed use of the limb, and ultimately more periarticular fibrosis.

A more natural gait movement is to move all of the joints of a limb through a ROM simultaneously, for example bicycling exercises. Cycling is a passive movement whereby multiple joints in the limb are moved in the sagittal plane. It is a motion that is particularly useful for neurological patients, to help restore gait patterns, and can be performed in either lateral recumbency or in a standing position.

Stretching

Stretches are passive movements that are often performed in conjunction with ROM exercises. Decreased flexibility or muscle tightness can occur passively or actively. Passive tightness may be the result of postural adaptation or scarring and active tightness may result from overuse, injury or muscle spasm. The goal of stretching is to restore or maintain flexibility by realigning soft tissues and collagen, **not** to rip or tear tissues. Stretching is often tolerated more by cats following massage, exercise, application of heat or therapeutic ultrasound, all of which increase the extensibility of collagen.

Acupuncture

(Grant 2006, Lindley 2019, Tacke & Henke 2019).

Acupuncture can be defined as the insertion of a solid needle into the body for the purpose of therapy and pain relief. The insertion of the needle stimulates afferent alpha-delta nerve fibres (in skin) and type II/III nerve fibres (in muscle). The evidence base for the use of acupuncture for the treatment of cats is limited.

Acupuncture is more usually thought of as a treatment for chronic, rather than acute pain. Typically when acupuncture or other complementary treatments are considered by an owner, it is because that they are looking for a non-pharmacological treatment because their cat's condition is either non-responsive or poorly responsive to conventional therapy or there are unacceptable drug-related side effects.

Simply put acupuncture is thought to fool the body into thinking that tissues have been damaged, so that it triggers potent analgesic mechanisms that are focused on the area that most needs the pain relief. This means that the body responds to a new or recurrent pain stimulus by rapidly providing its own pain relief. Acupuncture treatments can often be reduced to 4–6 weekly, once the pain is controlled.

Electroacupuncture is a form of acupuncture where a small electric current is passed between pairs of acupuncture needles. Electroacupuncture provides a stronger stimulus and can be used to augment the use of regular acupuncture if it is not producing the desired effect. It may be particularly useful for neurological disorders where there are sensory or motor deficits.

In summary acupuncture's analgesic effects may be mediated by:

- Activation of the endogenous pain inhibition system
- Distribution of neuro transmitters (e.g. ACTH, serotonin, norepinephrine, GABA, acetylcholine, substance P)
- Release of endogenous opioids, (e.g. B-endorphin, met-enkephalin, dynorphin)

- Activation of spinal segmental and heterosegmental inhibition processes
- Activation of supraspinal diffuse pain inhibition mechanisms

Modalities

(Johnson et al. 1997, Karu 1987, Melzack & Wall 1965, McClure et al. 2004, Millis et al. 2005).

There are several electrophysical modalities that can be used on animals. These include laser, therapeutic ultrasound, neuromuscular electrical stimulation (NMES), transcutaneous electrical nerve stimulation (TENS) and extracorporeal shock wave therapy (ESWT). Most cats will tolerate all or some of these modalities if they are administered in a relaxed quiet environment.

Neuromuscular Electrical Stimulation

Neuromuscular electrical stimulation (NMES) is a form of electrical stimulation whereby current is used to stimulate a motor nerve. NMES is used mainly for muscle strengthening by inducing muscles to contract. Muscle stimulation recruits fast twitch fibres first. The denervated muscle of a neurological patient with spinal cord injury requires direct stimulation of muscle fibres. This necessitates removal of hair in the affected area before the electrodes can be placed (Figure 16.3).

Benefits of NMES for cats that cannot exercise actively include:

- Increased blood flow
- Reduction of oedema
- Decreased spasticity and muscle spasm
- Improved sensory awareness
- Decreased joint contracture
- Muscle re-education and facilitation of muscle control
- Muscle strengthening and prevention of disuse atrophy

Transcutaneous Electrical Nerve Stimulation

Transcutaneous electrical nerve stimulation (TENS) is a method of pain relief involving the use of a mild electrical current. The primary pain relief mechanism of TENS was explained by Melzack and Wall in 1965 when they published their gate control theory of pain relief. TENS primarily aims to provide symptomatic pain relief by specifically exciting sensory nerves and thereby stimulating either the 'pain gate' mechanism and/or the endogenous opioid system. The two wave forms of TENS are interferential and premodulated. These frequencies interfere with the transmission of pain signals at the level of the spinal cord. For optimal effect the electrodes are usually placed close to the source of pain, for example:

- Either side of an incision
- Over a motor point
- Over a trigger point(s)
- At the level of the appropriate nerve root(s)

Contraindications to electrical nerve stimulation include:

- Patients with pacemakers
- An allergic response to the electrodes
- Dermatitis
- Over infected or open wounds
- Tumours
- Local areas of impaired circulation

Laser Therapy

The term 'laser' is an acronym for *light amplification by stimulated emission of radiation.* When light is used for therapeutic purposes it is called *phototherapy*. Laser light energy is absorbed in the superficial tissues so penetration is rarely greater than a few millimetres. Nevertheless, deeper effects can occur as a result of chemical mediators, whereby the cell membrane is the primary absorber of the energy, which then generates intracellular effects by means of a cascade response.

Phototherapy can be used in the management of OA, to promote healing of soft tissue injuries and wounds, and for pain relief. Indications for wound management include ulcers, pressure sores, burns, skin infections, and incision sites (Figure 16.4).

Therapeutic Ultrasound

Therapeutic ultrasound (TUS) uses sound energy at frequencies greater than 20,000 Hz. Sound waves are produced by applying an electrical current to a crystal in the trans-



Figure 16.3 A cat receiving neuromuscular electrical stimulation.

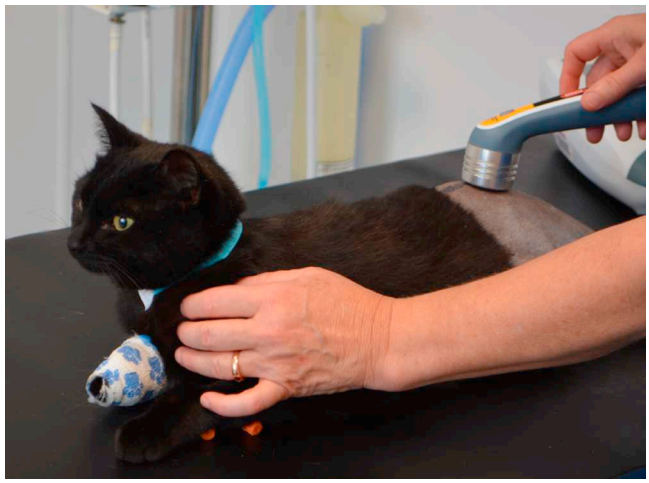


Figure 16.4 A cat receiving laser therapy.

ducer head, which causes the crystal to vibrate. The emitting pressure waves are absorbed by the tissue; the level of penetration being determined by the type of tissue being treated. As the sound waves move through the tissues, absorbed energy is converted into kinetic energy. Pulsed or continuous modes are available, with pulsed modes used for promoting healing, and continuous modes for their thermal effects. Sound energy does not transmit well through air so a coupling medium of water-soluble gel is required to allow energy delivery to the tissues. The area should be clipped before gel application and the transducer head must remain in contact with the gel throughout treatment.

The thermal effects are the same as those of superficial heating agents but at greater depths of up to 50 mm. Because of its ability to heat deeper tissues, TUS is often used when the target tissue is muscle, tendon or ligament.

The thermal mode of TUS is indicated in the treatment of chronic soft tissue injuries. Beneficial effects include remodelling of scar tissue, reduction of joint restriction associated with periarticular fibrosis, and reduction of muscle spasm. Non-thermal TUS is useful for promoting healing of acute soft tissue injuries and nerve injuries.

Extracorporeal Shockwave Therapy

Extracorporeal shockwaves are acoustic waves of high pressure and velocity produced outside the body. These pressure waves differ from ultrasound waves because of their lower frequency, minimal tissue absorption, and lack of a thermal effect. Extracorporeal shockwave therapy (ESWT) is capable of penetrating deeper than any other modality.

ESWT can be either radial or focused. Some units are capable of providing both types of shockwave therapy (Figure 16.5).

- Radial ESWT. The transmitter surface constitutes the highest pressure and highest energy density. The shockwave loses power the deeper it goes into the tissues of the body. Radial shockwaves are used for larger areas and for superficial conditions.
- Focused ESWT. The focusing mechanism directs each shock to a defined point within the body. At the site of actual wave generation the amount of energy dispersed is minimal. This limits any possible damage to the skin and to underlying soft tissues. The location within the body is adjusted by altering the energy output of the machine or by adjusting the focusing mechanism. A stand-off applicator can also be used to alter the depth of energy delivery. Focused ESWT is growing in popularity for veterinary patients, owing to the increased treatment options and the fact that there is less discomfort during treatment.

There are three different methods of pressure wave generation:

- Electrohydraulic. This method produces the highest peak pressure in the shortest time. This treatment method is the most uncomfortable to the patient so sedation is recommended.
- Electromagnetic. This produces changes in pressure similar to the effect of a loudspeaker.
- Piezoelectric. This creates ultrasonic vibrations and produces the smallest change in pressure. This method is the least uncomfortable and often does not require sedation of the patient.

ESWT may be beneficial as an ancillary treatment for OA as part of a multimodal approach to management of this condition. ESWT has also been considered an option for patients that are unable to tolerate the administration of NSAIDs. Many cats will tolerate focused electromagnetic or piezoelectric ESWT without sedation.

ESWT has been used in the treatment of a number of different conditions:

- Wounds
- Osteoarthritis
- Delayed and non-union fractures
- Ligament and tendon injuries
- Tendinopathies (Figure 16.6)



Figure 16.5 A shockwave machine suitable for use on veterinary species.



Figure 16.6 Extracorporeal shockwave therapy being used in the treatment of calcaneal tendinopathy.

- Desmitis

Contraindications for ESWT include:

- Use with caution over implants
- Acute inflammation
- Immune-mediated joint disease
- Neoplasia
- Skeletal immaturity
- Over open growth plates
- Unstable fractures
- Patients with neurological deficits
- Coagulopathies
- Lungs, brain, heart, major nerves and blood vessels
- Pregnancy

ACTIVE REHABILITATION:

(Burkholder 2001, Millis & Levine 2014, Millis et al. 2014, Page 2012, Sharp 2012).

Active rehabilitation involves exercises that require muscular activity by the patient and includes a variety of land-based therapeutic exercises and hydrotherapy either in a swimming pool or an underwater treadmill. Institution of active exercise must take account of the physical ability of the cat, the stage of tissue healing after surgery or injury and the desired outcome.

Therapeutic Exercises

It is important to have reasonable expectations and to set realistic goals at the beginning of a rehabilitation programme involving therapeutic exercises. The physiotherapist should determine and record the body condition of the cat as a baseline before starting rehabilitation. It is important to have a comprehensive understanding of the cat's condition, and the expectations, and the commitment of the owner to the rehabilitation programme from the outset, if a successful outcome is to be achieved.

An exercise therapy programme begins with short low impact activities and progresses to longer time periods with more strenuous activities. Cats do best with therapeutic exercises when these exercises are linked to natural activities such as hunting, playing or feeding.

Four areas should be targeted when considering a rehabilitation programme that incorporates land and water-based exercises:

- Strength
- Endurance
- Range of motion
- Balance and proprioception

Strength

Lack of use through injury, disease, and inactivity leads to disuse or reduced-use muscle atrophy and the deterioration of muscle strength. Neurogenic atrophy occurs when the motor nerve supplying a muscle is affected by disease or injury, and is the most rapid and severe type of muscle atrophy. Disuse atrophy is reversible whereas reversal of neurogenic muscle atrophy is possible only if nerve function returns.

Once an appropriate time has passed for healing to occur the goal of physiotherapy is to return strength to the affected tissues. In the early stages, the focus should be on the affected limb or limbs, taking care not to damage or overload healing tissues. The underwater treadmill is a safe environment for building strength in the affected limb. The water level can be at a height where full weight is not put through limb but there is some resistance from walking in water. Wheelbarrow and dancing exercises can be introduced when strength has increased to the point where it is no longer difficult for the cat to support its weight in the stationary position.

Endurance

Progression of rehabilitation moves from treatment of a localized area to overall body conditioning. Cats rely on stealth and rapid movements to catch prey and rarely if ever stand or run for long periods so endurance exercises are less relevant for this species.

Range of Motion

Maintaining ROM helps preserve a cat's flexibility. A home exercise programme should start with PROM, then once the cat has gained sufficient strength and use of the affected limb, active exercises can be given. Active exercises will provide an evaluation of how much ROM is already present, and observing the cat sit, rise, lie down, climb stairs or walk over poles will show how much the cat is able to flex and extend its joints. The patient should be lured with food, by calling, or can be allowed to walk over obstacles such as poles into a safe place. Having the cat walk in water is another way of generating an increased ROM of the limbs.

Balance and Proprioception

Proprioception can be affected through the ageing process; it can also be altered through injury, surgery or neurological disease. Weight-shifting is a good way of improving balance and proprioception, and can be introduced as soon as the cat is able to stand safely. A peanut roll can be used for support if the patient is unable to stand unaided (Figure 16.7). Adding an unstable surface requires the cat to provide an immediate readjustment of its equilibrium. Unstable surfaces include balance and rocker boards or standing the cat on a therapy ball. Proprioceptive exercises such as stepping



Figure 16.7 A cat on a peanut roll.

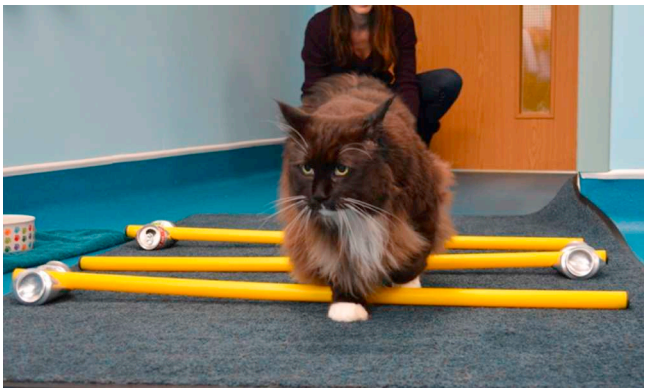


Figure 16.8 A cat walking over cavaletti rails.

over obstacles and walking on different terrains will help the cat to develop an awareness of where it is placing its limbs (Figure 16.8).

Cats prefer activities that involve minimal handling and restraint so when considering land-based exercises it is best to take advantage of a cat's natural hunting instincts. Sessions need to be kept short to maintain interest and prevent boredom. An example of hunting behaviour that can be elicited is chasing a laser light beam, which encourages stretching to reach the light. Playing with a toy mouse or any other toy involves movement of multiple joints and will improve dexterity. These activities can be used to encourage standing, crouching and moving in circles. It is important to match the level of activity to the cat's ability and stage of recovery.

Hydrotherapy

(Lindley et al. 2010, Mucha 2019).

The therapeutic properties of water include:

- Thermal – warming of submerged tissues
- Buoyancy – upward thrust of water on the immersed body
- Hydrostatic pressure – the pressure exerted by water on the immersed body
- Cohesion – water molecules adhere to one another creating resistance for the moving body
- Turbulence – whirlpools caused by body movement

Aquatic therapy is beneficial for soft-tissue injuries, OA, postoperative fracture care, muscle weakness, neurological impairment and geriatric care. Contraindications include, gastroenteritis, cardiorespiratory disease, pyoderma, coagulopathies, and severe peripheral vascular disease.

Although many cats tolerate aquatic therapy surprisingly well, to avoid injury, patient selection will determine whether aquatic-based therapy is appropriate for the individual. A life jacket will be necessary in a swimming pool whereas a small harness is adequate when walking a cat in an underwater treadmill (UWT) (Figure 16.9). In either case, the therapist will need to accompany the cat in the aquatic environment.

Both the pool and the UWT are excellent for non-weight-bearing exercise and generating limb movement. In contrast to the pool the degree of weight bearing in the UWT can be carefully controlled by altering the depth of the water. Therefore, an UWT has significant advantages over a pool in that it allows for a more controlled form of exercise and it also allows the handler to respond more quickly to changing situations.



Figure 16.9 A cat in an underwater treadmill.

Aquatic therapy in the UWT helps to make movement more comfortable, offering exercise that is functional, mechanically correct, and muscularly challenging without being painful. Use of the UWT also promotes limb loading, and challenges balance and coordination more safely than on land. Sutures or staples are usually removed prior to initiating hydrotherapy, however there are occasions where early intervention using hydrotherapy can be considered advantageous with the surgeon's consent.

Use of the UWT also improves proprioception and assists gait retraining and sequencing, especially in the neurological patient. A cat recovering from non-ambulatory paresis or plegia will usually walk in water before it is able to do so on land. The UWT can be employed safely sooner in the recovery process than the swimming pool. Postoperative spinal patients can generally be placed in the UWT as soon as the incision has healed, whereas use of a swimming pool is usually delayed for at least 4 weeks. For cats that are being managed conservatively for spinal injury or intervertebral disc disease use of the swimming pool should be delayed for at least 12 weeks.

When introducing a patient to the UWT for the first time most cats accept walking around in the underwater chamber, it is usually the movement of the track that they find unsettling. When the track is moved for the first time it should be slow and in short bursts of around 5–10 seconds to accustom the cat to the sensation. The height of the water can be varied in order to determine the level the cat will find acceptable. Bulky buoyancy jackets are unnecessary in the UWT and cats tend to become more reactive when they are donned. However, cats will generally tolerate a harness which is important as it enables the handler to have some control over the patient. Unfortunately, cats become bored or uncompliant to a particular exercise quickly, so once the track is started, the sessions need to be very short and the speed of the track should be slow.

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17 Neuromuscular Disorders

INTRODUCTION:

Neuromuscular disorders are a broad group of pathological conditions that impair the functioning of muscles causing weakness. The pathological changes affect either the muscle fibre directly and/or indirectly by impairing its nervous control. Neuromuscular disorders in the cat are relatively uncommon, but it is important to be able to recognize the signs when they do occur. With the exception of autonomic neuropathies, such as dysautonomia, all neuromuscular disorders manifest as muscular weakness. The expression of this weakness varies and may include paresis or plegia, gait abnormalities, exercise-related weakness, regurgitation, dysphagia, dysphonia and dyspnoea. In addition, there may be gross abnormalities of the musculoskeletal system such as muscle atrophy, muscle hypertrophy and skeletal deformities. It should be noted that paresis is a descriptor for muscle weakness or partial loss of voluntary movement and plegia or paralysis are descriptors for a complete absence of voluntary movement.

The motor system is divided into two main components – upper motor neurons (UMNs) and lower motor neurons (LMNs). The upper motor neurons are completely contained within the central nervous system and they can be thought of as 'central motor neurons'. Lower motor neurons have their cell body in the central nervous system either in the spinal cord or in the brainstem. Their axons project into the peripheral nervous system to innervate striated or smooth muscle at the neuromuscular junction and they can be thought of as 'peripheral motor neurons'. Lower motor neuron disorders in cats with neuromuscular disorders that are ambulatory are characterized by paresis in which there is a reduction in the ability to support weight resulting in a shortened stride. This is in contrast to the paresis seen with upper motor neuron disorders where the stride length is increased. The distribution of involvement of a neuromuscular disorder may be localized, regional or generalized.

CLASSIFICATION OF NEUROMUSCULAR DISORDERS:

Neuromuscular disorders can be classified into four types determined by the primary anatomic location of the lesion (Table 17.1)

- Neuropathies – disorders of the lower (peripheral) motor neuron and its axon in the nerve root and the peripheral nerve.
- Myopathies – disorders of muscle fibres.
- Junctionopathies – disorders of the neuromuscular junction.
- Neuromyopathies – disorders of both nerves and muscle fibres.

Diseases are further subdivided according to whether they are acquired or inherited. Inherited or congenital neuromuscular diseases are less common than acquired ones.

CLINICAL SIGNS:

The clinical signs of neuromuscular disease are summarized in Table 17.2. In contrast to dogs with neuromuscular disease, which usually present with episodic weakness often related to exercise, affected cats will limit their activity and tend to present with persistent weakness. This may manifest as neck ventroflexion or the cat may lie with the head resting on its paws appearing uncharacteristically relaxed despite being in the normally stressful consulting room environment. When made to walk, instead of being smooth and free flowing, limb movements are often stiff, stilted and choppy. As a result of the rather flexed position of the distal limb joints in the cat when weight bearing and the relative paucity of type 1 muscle fibres in postural limb muscles a palmigrade and/or plantigrade stance is a common manifestation of feline neuromuscular disease.

A common non-specific clinical sign in cats with many disorders causing generalized neuromuscular weakness is abnormal posture with ventral neck flexion (Figure 17.1). This occurs because cats do not possess a nuchal ligament. Affected cats usually rest their chin near the thoracic inlet, with the eyes positioned dorsally to maintain their gaze straight ahead. Other clinical manifestations of generalized weakness include a stiff forelimb gait and protrusion of the dorsal aspects of the scapulae when weight bearing. A crouched, wide-based stance is often seen in the hindlimbs. Causes of neck ventroflexion are numerous and include myasthenia gravis, hypokalaemic myopathy, thiamine responsive neuromuscular weakness, hyperthyroidism, hypocalcaemia, portosystemic encephalopathy, subacute or chronic organophosphate toxicity, ethylene glycol toxicity, ammonium chloride toxicity, polyneuropathy, hypernatraemic polymyopathy, hereditary myopathies (Burmese, Devon Rex) and immune-mediated polymyositis.

DIAGNOSIS:

The diagnosis of feline neuromuscular disorders can be challenging. Weakness is a relatively common clinical sign in cats and may be caused by a primary neuromuscular disease or the neuromuscular disease may be secondary to disease of another body system. Additionally, the presenting signs of severe systemic disease and some orthopaedic and cardiorespiratory disorders may mimic those of neuromuscular disease. Diagnosis of a neuromuscular disorder is made on the basis of signalment, physical and neurological examination, laboratory testing, electrodiagnostic testing and biopsy.

A minimum database includes a complete blood count, serum biochemistry panel, urinalysis, thoracic radiographs and abdominal ultrasound or computed tomography scanning of the thorax and abdomen. Electrophysiologic evaluation of nerve and muscle disorders is usually only performed at specialist centres. With the exception of neuromyopathies neurological examination should provide a clear distinction between most neuropathies and myopathies. Hyporeflexia or areflexia and in some cases impairment of sensory pain perception are distinguishing features of neuropathies and are not seen with myopathies or junctionopathies. The distinction between a myopathy and a junctionopathy may be more difficult to make. Myasthenia gravis, in particular, may produce signs that mimic those of a generalized myopathy, although it is a junctionopathy. The diagnosis of cats suspected of having neuromuscular disease is confounded by a narrow range of presenting clinical signs which are common to many disorders irrespective of cause.

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Table 17.1: Categorization of Feline Neuromuscular Disorders

	Inherited	Acquired
Neuropathies		
	Degenerative	*Degenerative*
	Birman cat distal polyneuropathy	Adult onset motor neuron disease
	Bengal cat recurrent polyneuropathy	
	Axonal neuropathy of Snowshoe cats	*Inflammatory/idiopathic*
	Spinal muscular atrophy	Acute polyradiculoneuritis
	Hypertrophic neuropathy	Chronic idiopathic polyneuropathy
		Brachial plexus neuritis/neuropathy
	Metabolic	
	Hyperoxaluria	*Infectious*
	Hyperchylomicronaemia	Bacterial
	Niemann-Pick disease type A	Protozoal
	Glycogenosis type IV	Viral
		Vascular
		Ischaemic neuromyopathy
		Traumatic ischaemic neuromyopathy
		Endocrine/metabolic
		Diabetic polyneuropathy
		Hypersomatotropism and acromegaly
		Toxic
		Organophospahate
		Salinomycin
		Vincristine
		Neoplastic and paraneoplastic
		Traumatic
		Brachial plexus avulsion
		Radial neuropathy
		Ischiatic/sciatic neuropathy
		Ulnar neuropathy
		Iatrogenic neuropathy
Myopathies		
	Degenerative	
	Nemaline myopathy	Myositis ossificans
	X-linked muscular dystrophy	Traumatic myopathy
	Muscular dystrophy with laminin alpha 2 deficiency	Semitendinosus myopathy
	Myotonia congenita	Brachialis contracture
		Digital flexor tendon contracture
		Quadriceps contracture
		Hypokalaemic myopathy
		Bacterial myopathy
		Protozoal myopathy
		Idiopathic polymyositis
		Paraneoplastic myopathy
		Focal Tetanus
Junctionopathies		
	Myasthenia gravis	Myasthenia gravis
	Congenital myasthenic syndrome	Organophosphate and carbamate toxicity
		Snake bite and tick paralysis

MUSCLE AND NERVE BIOPSY:

(Dickinson & LeCouteur 2004).

Muscle and nerve biopsy are underutilized techniques that provide an opportunity to evaluate the morphology of portions of the motor unit. Histological identification of pathological processes should allow differentiation between neuropathies, myopathies and junctionopathies and in some instances will provide a definitive diagnosis. Histological

Table 17.2: Summary of the Clinical Signs of Neuromuscular Disease

Neuropathies	Myopathies	Junctionopathies
Muscle atrophy	Muscle weakness or stiffness	Reduced exercise tolerance
Altered muscle tone	Variable muscle pain	Intermittent weakness
Paresis or paralysis	Muscle atrophy or hypertrophy	Flaccid paralysis
Proprioceptive deficits	Reduced exercise tolerance	
Hyporeflexia or areflexia	Stiff, stilted gait	
	Limited joint movement (contracture)	



Figure 17.1 A cat with neck ventroflexion (Courtesy of Rob Quinn).

examination of muscle in particular may also provide valuable information about the severity of disease and the expectations for recovery. Biopsies should follow neurological examination and electrodiagnostic testing. Needle insertion artefacts in muscle can be avoided by collecting biopsies from sites that are contralateral to the side where the majority of electrodiagnostics were performed. In acute disorders of muscle the most severely affected site should be chosen. However, in chronic cases where the muscle is fibrotic a less severely affected site is more likely to show underlying pathological changes. The associated motor nerve should also be biopsied if nerve conduction studies have revealed abnormalities. Techniques for obtaining muscle and nerve biopsies have been well described elsewhere. It is advisable to contact the laboratory in advance for specific advice about preservation and shipping. Samples should be taken on a day when their arrival will not fall at the weekend or on a public holiday.

NEUROPATHIES:

The clinical signs of neuropathies generally reflect failure of the lower motor neuron (neuronal cell bodies, axons and Schwann cells). Neuropathies can be divided into inherited and acquired types. Clinical signs of both include paresis or paralysis, ataxia, hypo- or areflexia, tremors, altered muscle tone, and muscle atrophy (Table 17.2). Impairment of sensation is variable and there may be anaesthesia, hyperaesthesia, or paraesthesia. The acquired types may have infectious, immunological, nutritional, metabolic, traumatic, vascular or toxic aetiologies or may be idiopathic. Inherited or congenital neuropathies generally have an early onset of clinical signs whereas acquired neuropathies can occur at any age.

Degenerative Inherited Polyneuropathies

There are few reports of inherited polyneuropathies in cats and these are mostly limited to specific breeds. However, an inherited polyneuropathy should be suspected in any breed or sex of cat less than 12 months of age with a history of slowly progressive tremors and paraparesis, or tetraparesis with depressed or absent spinal reflexes. Inherited neuropathies are degenerative conditions that are usually symmetrical and non-painful. Many congenital polyneuropathies are untreatable, progressive, and fatal but there are important exceptions.

Birman Cat Distal Polyneuropathy

(Chrisman 2000, Coates & O'Brien 2004, Moreau et al. 1991).

Affected cats show a degenerative neuropathy of the distal parts of the peripheral nervous system and several areas of the brain and spinal cord. The disease occurs in Birman kittens from 8-10 weeks of age presenting as a subtle hypermetria of all limbs, progressive hindlimb ataxia and plantigrade stance. Postmortem examination reveals a central and peripheral distal axonopathy. An autosomal recessive mode of inheritance is suspected. Diagnosis is based on the signalment, history, clinical findings and the results of diagnostic tests. Confirmation of the diagnosis is based on histopathology of the central and peripheral lesions. There is no known treatment and the prognosis is poor.

Bengal Cat Recurrent Polyneuropathy

(Aleman et al. 2014, Bensfield et al. 2011, Granger et al. 2008).

A chronic relapsing predominantly motor polyneuropathy with recurrent demyelination and remyelination is a relatively common disease in young Bengal cats. Occasional cases with only a single episode of weakness have also been described. Affected cats present with gait and postural abnormalities of all four limbs or just the hindlimbs including paresis and weakness, plantigrade stance, ataxia and reduced ability to jump. Severely affected cats may be non-ambulatory. Diffuse muscle atrophy or atrophy most pronounced in the hindlimbs is a common finding. Spinal reflexes are usually reduced. The cause is unknown although an inflammatory, possibly immune-mediated, aetiology is likely. Diagnosis is based on signalment, history, clinical findings and the results of diagnostic tests. Histopathology of nerve biopsies shows axonal changes with loss of nerve fibres. Bengal cats appear to be genetically predisposed to the condition, but similar recurrent motor polyneuropathies have been reported less frequently in other cat breeds. Even in cases where the clinical signs are dramatic the prognosis for functional recovery is good although there can be residual motor deficits.

Axonal Polyneuropathy of Snowshoe Cats

(Matiasek et al. 2009).

An early onset suspected inherited polyneuropathy has been described in related Snowshoe kittens that presented with hindlimb weakness at 4 and 6.5 months of age. Electrodiagnostic findings were consistent with an axonopathy and muscle and nerve biopsies confirmed axonal degeneration. In contrast to most axonal neuropathies there was a tendency for remission of the clinical signs over a follow up period of 2 years. Concurrent central nervous system abnormalities have been reported.

Spinal Muscular Atrophy

(Chrisman 2000, Iannaccone 2005, Shelton et al. 1998).

In this condition there is degeneration of motor neuron cell bodies and premature cell death primarily in the spinal cord. Typically kittens present in the first 6 months of life with a rapidly progressive polyneuropathy causing tetraparesis, hyporeflexia and muscle atrophy. The disorder is most commonly recognized in Maine coons where it is inherited as an autosomal recessive trait. These cats present with muscle weakness by 3–4 months of age and by 6 months of age are usually too weak to be able to jump and have an abnormal gait. A PCR-based genetic test is available to identify affected cats and carriers of the condition. The prognosis is fair as the condition does not appear to be painful and is not fatal. Affected cats can live a relatively normal life indoors.

Hypertrophic Neuropathy

(Chrisman 2000).

This is best known as a peripheral neuropathy of Tibetan mastiffs but has also been described in cats. There is gross enlargement of affected nerves with widespread demyelination and remyelination and hypertrophy of Schwann cells. Kittens present at 7–12 months of age with generalized tremors, hypermetria, hyporeflexia, plantigrade stance and decreased sensation in the distal extremities and facial region. Muscle atrophy and mild paresis of the limbs are also commonly observed. There is no treatment and the prognosis is poor because of the progressive nature of the condition.

Inherited Metabolic Disorders

Hyperoxaluria

(Chrisman 2000, De Lorenzi et al. 2005, McKerrell et al. 1989).

Affected cats have low concentrations of the enzyme D-glycerate dehydrogenase, resulting in an unexplained axonopathy with lower motor neuron dysfunction. Kittens usually present between 5 and 9 months of age with generalized weakness, proprioceptive deficits, hyporeflexia and decreased nociception. There is an intermittent oxaluria and affected cats usually die from renal failure due to deposition of oxalate crystals in the renal tubules before 1 year of age. Diagnosis is based on history and clinical signs, electrodiagnostic tests, muscle/nerve biopsy and identification of oxalate and/or L-glyceric acid in the urine.

Hyperchylomicronemia

(Coates & O'Brien 2004, Ginzinger et al. 1996, Jones et al. 1986).

Affected cats have low lipoprotein lipase activity and elevated plasma triglyceride and cholesterol. The condition is thought to be inherited as an autosomal recessive trait. Kittens first show neurological signs that include monoparesis, facial paralysis, and Horner syndrome between 1 and 8 months of age. Polyneuropathy results from compression of nerves and nerve roots by lipid granulomas (xanthomas) that probably develop subsequent to trauma over pressure points and at intervertebral foramina. Unilateral or aysmmetric neurological deficits are common. Affected cats have lipaemia retinalis and their blood has a 'cream of tomato soup' colouration associated with hyperlipaemia. Diagnosis is based on history and clinical findings, elevated serum triglyceride and cholesterol, electrodiagnostics and nerve/muscle biopsies. Feeding a low-fat diet can result in resolution of lipaemia and neurological dysfunction.

Lysosomal Storage Diseases

(Skelly & Franklin 2002).

These diseases occur when there is an intracellular accumulation of one of more products of an interrupted degradative metabolic pathway. The accumulated products adversely affect normal cellular function as a result of cellular swelling and/or toxicity. Affected cats are normal at birth but develop progressive neurological signs usually during the first year of life. Cerebellar dysfunction is a common early sign of many lysosomal storage diseases. The lysosomal storage diseases that cause polyneuropathy as part of the clinical syndrome in cats are Niemann-Pick disease type A and glycogen storage disease type IV. Siamese cats with Niemann-Pick disease may initially present purely as a result of a polyneuropathy.

Niemann–Pick Disease Type A (Sphingomyelinosis)

(Baker et al. 1987, Coates & O'Brien 2004, Cuddon et al. 1989, Wenger et al. 1980).

Sphingomyelinosis is a lysosomal storage disease associated with a deficiency of the enzyme sphingomyelinase, which results in a demyelinating polyneuropathy. The disease has been reported in Siamese and Balinese cats. Clinical signs include progressive lower motor neuron paresis starting in the hindlimbs, plantigrade stance, ataxia, tremor and hypermetria. Organomegaly (hepatomegaly and splenomegaly) develops due to accumulation of sphingomyelin, cholesterol and gangliosides in the cells of abdominal organs. Clinically signs are typically seen from 4 months of age with head bobbing and apparent blindness seen in the later stages. Affected kittens are usually euthanased during the first year of life.

Glycogen storage disease type IV (Glycogenosis, Andersen Disease)

(Fyfe et al. 1992).

Glycogen storage disease type IV is a disorder seen in the Norwegian forest breed resulting from a deficiency in a glycogen branching enzyme that leads to abnormal glycogen metabolism and deposition. Glycogen accumulation occurs in the liver, heart, neurons and muscles causing progressive organ, nerve and muscle dysfunction. The disease is inherited as an autosomal recessive trait. Affected kittens may die at or soon after birth but some individuals may remain clinically normal up to 5 months of age. These kittens present with paresis that progresses to persistent pyrexia, encephalopathy, generalized muscle atrophy and muscle tremors. A specific PCR test is available for diagnosis and to screen for carriers of the genetic mutation. Affected kittens are usually euthanased during the first year of life or may die of heart failure.

Acquired Degenerative Neuropathies

Adult Onset Motor Neuron Disease

(Rohdin et al. 2010, Shelton et al. 1998, Summers et al. 1995).

Acquired motor neuron cell death is a rarely described condition that predominantly affects adult cats of different breeds between the ages of 6 and 12 years. Clinical signs have included chronic progressive weakness, neck ventroflexion, generalized muscle atrophy, and eventually, hyporeflexia or areflexia. Diagnosis is based on the slowly progressive clinical signs, and electromyographic changes. There is no effective treatment but affected cats may survive for many years after the diagnosis. There is a single report of an atypical case of suspected acquired motor neuron degeneration that presented in a kitten at 12 weeks of age with paraplegia and severe hindlimb contractures consistent with a neurogenic form of arthrogryposis.

Inflammatory/Idiopathic Disorders

There are a few reports of a variety of inflammatory/idiopathic polyneuropathies that tend to be progressive and symmetrical and occasionally painful. These include an acute idiopathic polyneuritis, a chronic relapsing polyradiculoneuritis and an acute brachial plexus neuritis.

Acute Polyradiculoneuritis

(Gerritsen et al. 1996, Granger et al. 2008, Lane & DeLahunta 1984).

Acute-onset, areflexic, flaccid tetraparesis or tetraplegia similar to coonhound paralysis (acute canine polyradiculoneuritis) has been reported in cats. The age of onset has varied from 3 months up to 4 years. Clinical signs typically begin with an acute paraparesis progressing rapidly to tetraparesis within 72 hours. Respiratory depression occurs in severely affected cases, necessitating oxygen therapy or ventilation. Sensory function is usually well preserved and ambulatory patients are therefore paretic and not ataxic. Electromyography usually reveals evidence of denervation, in the form of fibrillation potentials and positive sharp waves 5–7 days after onset, in limb and paravertebral musculature. Loss of axons, demyelination and macrophage accumulation were found in the ventral (motor) nerve roots of cats with the condition that were euthanased. Nerve biopsy shows non-specific changes secondary to the nerve root pathology.

The prognosis is good unless respiratory failure intervenes. As with the analogous polyneuropathy in dogs, cats usually make a complete recovery with supportive care over a period of 2–6 weeks. Acute relapsing polyneuropathy has been reported with complete recovery between episodes.

Chronic Idiopathic Polyneuropathy

(Braund et al. 1996, Flecknell & Lucke 1978, Malik et al. 1991, Shores et al. 1987).

A chronic relapsing inflammatory demyelinating polyneuropathy has been described in adult cats with insidiously progressive tremors, ataxia, and weakness. Progression to severe flaccid lower motor neuron tetraparesis may occur. Nerve biopsy demonstrates inflammatory infiltrates, segmental demyelination and remyelination, and axonal loss. Electromyography reveals positive sharp waves and fibrillation potentials consistent with axonal and myelin disease. In some cats there has been apparent spontaneous improvement. The aetiology of chronic polyneuropathy is presumed to be immune-mediated and most cases improve with immunosuppressive doses of glucocorticoids. Prednisolone is administered at a dosage of 2–4 mg/kg divided twice daily for 2 weeks, followed by a reducing dosage over a period of 2–3 months. If glucocorticoids are discontinued too rapidly, there may be a recurrence of clinical signs, but their re-administration may again result in clinical improvement. The long-term prognosis is more guarded, however, as signs tend to recur and may eventually become refractory to treatment.

Brachial Plexus Neuritis/Neuropathy

(Freeman et al. 2009, Garosi et al. 2006).

Brachial plexus neuritis is a rare multiple mononeuropathy that presents as acute bilateral lower motor neuron paresis or plegia of the forelimbs. Other signs include bilateral atrophy of forelimb muscles and severe resentment to forelimb manipulation. Subclinical involvement of other motor and sensory nerves has been reported. An immune-mediated aetiology is suspected. Clinical signs may resolve over a period of several weeks with supportive care although relapses have been reported.

Infectious Disorders

(Dickinson & LeCouteur 2004, Dubey et al. 1990, Mari et al. 2016, Paulsen et al. 2000, Phillips et al. 1996).

A single case of pyogranulomatous neuritis affecting the sciatic nerve and its distal branches has been reported. A diagnosis of mycobacterial neuritis was made based on the finding of acid-fast bacilli within macrophages, nerve fibres and degenerate axons. Treatment with clofazimine and enrofloxacin for an extended period halted further disease progression but complete restoration of sciatic nerve function was not achieved.

Neospora caninum causes a protozoal polyradiculoneuritis predominantly affecting the lumbosacral nerve roots in puppies along with a meningoencephalomyelitis and myositis. Although the condition has not been reported in cats a distal polyneuropathy associated with toxoplasmosis has been reported in a six-year-old Birman cat. Experimental infection of cats with Neospora caninum may result in meningoencephalitis but there are no reports of naturally occurring cases.

Chronic feline leukaemia virus (FeLV) infection has been associated with progressive tetraparesis and generalized muscle atrophy as a result of a generalized polyneuropathy. Feline immunodeficiency virus (FIV) is known to be capable of causing subclinical myopathy and has been demonstrated experimentally to produce a generalized neuropathy.

Vascular Disorders

Ischaemic Neuromyopathy (Feline Cardiogenic Arterial Thromboembolism)

(Hogan et al. 2015, Horton & Renfroe 1978, Hogan 2017, Kettner & Kirberger 2006, Luis Fuentes V, 2012, Schoeman 1999, Smith et al. 2003, Whigham et al. 1998).

Cause and Pathogenesis

Ischaemic neuromyopathy is a common condition that results from an ischaemic insult usually associated with aortic thromboembolism secondary to myocardial disease. Occlusion of the aorta by a foreign body has been described



Figure 17.2 Forelimb monoparesis as a result of embolization in the right brachial artery (Courtesy of Dr John Tyler).



Figure 17.3 Footpads of the forelimbs and hindlimbs of a cat with embolization in the aortic trifuraction. The affected hindlimb footpads are on the right.

as a rare cause of ischaemic neuromyopathy. In cats with arterial thromboembolic disease, occlusion of the distal aortic trifurcation generally occurs when an embolus breaks free from a thrombus in the left auricular appendage and enters the peripheral circulation. The aorta is the commonest site, but emboli may also lodge in other vessels, most commonly the right brachial artery, causing ipsilateral forelimb monoparesis or monoplegia (Figure 17.2).

Ischaemia of muscles and nerves primarily distal to the stifles is produced by the vascular occlusion and associated vasoconstriction of collateral circulation induced by inflammatory mediators released from platelets in the clot.

Clinical Signs

Most cats do not have a previous history of cardiac disease. Clinical signs include acute or peracute hindlimb dysfunction with cold extremities, absent or weak femoral pulses and tachypnoea. Male cats are overrepresented which probably reflects the male predisposition to myocardial disease. The signs may be asymmetric but typically there is non-ambulatory paraparesis or paraplegia and absence of nociception in the distal hindlimbs including the digits and tarsus. Pain perception and voluntary movement are usually preserved in the stifle and hip regions but affected cats are unable to flex or extend the tarsi. Other signs that tend to develop within the first 24 hours include firm painful muscles, swelling of the limbs, and cyanosis of the claws and footpads (Figure 17.3).

Diagnosis

Diagnosis of ischaemic neuromyopathy is made on the basis of the clinical findings and the demonstration of underlying cardiac disease. The clinical signs in cats with traumatic spinal/pelvic and concurrent thoracic injury after an unobserved road traffic accident may be superficially similar to those in cats with ischaemic neuromyopathy. Differentiation can easily be made between the two conditions by checking the femoral pulses and comparing the colour of the forelimb and hindlimb footpads.

Treatment and Prognosis

No single treatment has been shown to improve the outcome of cats with this condition. Supportive care and analgesia are the main priorities in the short-term and this should include the use of opioids such as methadone or a fentanyl constant rate infusion. Congestive heart failure should be treated appropriately. Thrombolytic therapy is not recommended but antiplatelet or anticoagulant drugs should be administered to induce a hypocoagulable state. In a recent prospective trial comparing the antiplatelet drug clopidogrel with aspirin, clopidogrel was superior to aspirin both at reducing the likelihood of recurrence and increasing the time to recurrence.

The short-term prognosis is usually dependent on the severity of the underlying myocardial disease. If heart failure is controlled, many cats will recover partial or even complete function over a period of weeks to months after the initial episode. Ideally all cats should be treated for 72 hours to determine the acute clinical course. The prognosis is better for cats with only one limb affected, those that retain motor function, and for cats that are normothermic on presentation. However, because the embolus is usually only a portion of the intracardiac thrombus, or because additional thrombi form, recurrent episodes are likely. In a series of 250 cats managed in general practice in the UK, 153 cats were euthanased on presentation and only 6 cats survived for more than 12 months. No factors were identified that predicted euthanasia on presentation.

Traumatic Ischaemic Neuromyopathy (Bottom-Hung Window Syndrome)

(Gradner et al. 2017).

Ischaemic neuromyopathy can also occur when cats become trapped in such a way that the blood supply to the part of the body distal to the entrapment becomes obstructed. This can happen in various ways for example under a garage door or in a ventilation system. Entrapment when a cat tries to escape through the small V-shaped opening of a bottom-hung window, so-called 'bottom-hung window syndrome', is the most frequent cause in countries where this type of window is commonplace. Bottom-hung or hopper windows have bottom mounted hinges and open by tilting vertically, typically to the inside. When a cat becomes trapped any attempt to free itself will result in the entrapped region becoming lodged even more securely in the V-shaped opening. A recent study of cats in Austria

described the findings in 71 cats seen at one institution with bilateral hindlimb neurological deficits that had been trapped by the thoracolumbar and abdominal area between the last rib and the pelvis.

Clinical signs are the result of compressive ischaemia of the hind quarters and can include weak or absent femoral pulses, low rectal temperature, patellar hyporeflexia or areflexia, and paraparesis or paraplegia. The overall mortality rate in the 71 cats in the study was 35% with the majority developing terminal clinical signs of respiratory distress. The prognosis was influenced by the neurological grade on presentation with a higher mortality rate (55%) in cats with paraplegia and absence of nociception. The pathogenesis involves ischaemic injury and venous congestion as a consequence of partial or complete arterial and venous obstruction, direct compression of peripheral nerves and blood vessels and reperfusion injury with electrolyte disturbances and release of toxins.

Endocrine/Metabolic Disorders

Diabetic Polyneuropathy

(Kramek et al. 1984, Mizisin et al. 2002, Platt 2002, Wolff 1984).

A distal polyneuropathy affecting the hindlimbs is occasionally seen in cats with type 2 diabetes mellitus and may occur in advance of those signs more typically associated with the disease. The reported incidence is 8% of diabetic cats but the true figure may be much higher. Traditionally the polyneuropathy has been thought of as a distal axonopathy with secondary demyelination. Recent evidence suggests a more crucial role for Schwann cells and myelin with axonopathy being less important. However, there are a number of hypotheses to explain the polyneuropathy including microvascular disease, metabolic aberrations and immune-mediated disease.

Affected cats have a severe plantigrade posture, progressive paraparesis, hyporeflexia, and hindlimb muscle atrophy (Figure 17.4). Signs may progress to involve the forelimbs although this is less common than in the dog. In contrast to the dog, cats with forelimb involvement are likely to show a significant palmigrade posture. There is no specific proven treatment for improving the polyneuropathy. However, clinical signs often improve and may resolve within 6–12 months with strict glycaemic control. Diagnosis is based on confirming diabetes mellitus and ruling out other causes of polyneuropathy. Electrodiagnostic tests and nerve/muscle biopsy can be performed to support the diagnosis.



Figure 17.4 Diabetic cat with a plantigrade stance (Courtesy of Jon Wray).

Hypersomatotropism and Acromegaly

(Niessen et al. 2007, Niessen et al. 2013).

Hypersomatotropism is characterized by excessive production of growth hormone and acromegaly is the clinical syndrome that develops as a result of the excess growth hormone. Hypersomatotropism usually results in diabetes and has been reported in up to a third of insulin-treated cats presented for assessment of glycaemic control in first opinion practices in the UK. Hypersomatotropism is most commonly caused by a growth-hormone secreting pituitary adenoma. Acromegaly is an underdiagnosed disorder most commonly seen in older neutered male cats that are presented for insulin-resistant diabetes mellitus with concurrent weight gain rather than weight loss. Common physical changes in cats with acromegaly include polyuria/polydipsia, polyphagia, weight gain, organomegaly, systolic heart murmur, plantigrade posture and an unkempt coat. Protrusion of the mandible (prognathia inferior), increased interdental spacing, broad facial features, and enlarged paws are features generally only seen in the later stages of the disorder. The plantigrade posture is a manifestation of diabetic polyneuropathy, and usually resolves with improved glycaemic control.

Toxic Polyneuropathies

(Chrisman 2000, Dickinson & LeCouteur 2004, Zook et al. 1968).

Toxicity should be suspected in any cat with polyneuropathy and a history of exposure to a toxic substance or drug. Once the underlying toxic substance is removed most cats recover from the neuropathy with supportive care. Cats have been used as an experimental model for studying polyneuropathies associated with a number of toxic substances. These include thallous acetate, acrylamide, and organophosphates, which are all capable of disrupting axonal transport. Organophosphates and carbamates cause a nonselective reduction in the activity of acetylcholinesterase, allowing continuous cholinergic stimulation.

Delayed Organophosphate Toxicity

(Jaggy & Oliver 1990).

Polyneuropathy may occur as a delayed form of neurotoxicity after prolonged exposure to chlorpyrifos. Chronic toxicity is manifested as paresis, hyporeflexia, generalized hyperaesthesia and dilated pupils. The pathogenesis of the polyneuropathy is not understood. Diagnosis is based on the history of exposure, reduced serum cholinesterase activity and abnormal electrodiagnositics. Treatment with pralidoxime (2-PAM) and atropine has been successful.

Salinomycin Toxicity

(van Nes et al. 1997, van der Linde-Sipman et al. 1999, Wheeler 1996).

In 1995 there was an outbreak of neurological dysfunction in cats in the Netherlands and Switzerland associated with the accidental contamination of cat food with salinomycin, an ionophore coccidiostat used in poultry. The neurological dysfunction ranged from mild paresis to severe tetraparesis, dyspnoea, dysphagia, and autonomic signs. A distal axonopathy was found on histological examination of peripheral nerves. Most of the cats that were not euthanased recovered after withdrawal of the affected food and supportive care.

Vincristine

(Cho et al. 1983, Goldstein et al. 1981).

The alkaloid cytotoxic drug, vincristine, may induce peripheral neuropathy ranging from dysphonia to sensory and motor polyneuropathy and tetraparesis. The suspected mechanism is the drug's adverse effects on axonal transport. Diagnosis is based on the clinical signs and history of vincristine administration. Electrodiagnostic tests and nerve/muscle biopsy are supportive. Resolution of signs once the drug is discontinued distinguishes vincristine toxicity from a paraneoplastic neuropathy.

Neoplastic Neuropathy

(Cavana et al. 2009, Lane et al. 1994, Sakurai et al. 2016, Watrous et al. 1999).

Nerves can be compromised either by direct neoplastic involvement or remote paraneoplastic effects. Direct involvement may be the result of primary neoplasia, compression by adjacent neoplasms, or infiltration by neoplastic cells. Primary nerve sheath neoplasms arise from the myelin-producing cells (Schwannomas) or the connective tissue surrounding the nerve (neurofibromas, malignant peripheral nerve sheath tumours) and are rare in the cat. Lymphoma has been reported involving nerves and nerve roots, especially at the brachial intumescence and the sciatic nerve and a condition resembling neurolymphomatosis in humans has been reported in a nine-year-old cat. Neoplastic neuropathy may or may not be painful and often has an asymmetric distribution. Clinical signs include paresis, muscle atrophy, and paraesthesia, which may manifest as excessive licking at the site of the affected sensory dermatome.

There is a single report of suspected paraneoplastic polyneuropathy in a cat with renal lymphoma. The pathogenesis is uncertain, although an immunological mechanism is most likely. Paraneoplastic inflammatory demyelinating polyneuropathy is well documented in dogs but appears to be a rare occurrence in cats. Nevertheless, progressive symmetrical polyneuropathy may be a sentinel sign for a clinically silent malignancy and a diligent search for primary neoplasia should be included in the routine diagnostic work-up of these cases. In theory, successful treatment of the primary neoplasm should result in resolution of the associated polyneuropathy, although this may take several months. It is important to be aware of the fact that some antineoplastic drugs such as vincristine are capable of causing a toxic polyneuropathy.

Traumatic Neuropathies

(Bailey et al. 1992, Garosi 2012, Hudson & Hamilton 2010, Kitchell et al. 1982, Seddon 1942).

Traumatic neuropathies in cats are usually acute and typically asymmetrical and in the early stages are often painful. Depending on the severity of injury there is usually an improving clinical course although very rarely proliferating connective tissue or bony callus causes a worsening of signs as a result of progressive nerve impingement or entrapment.

Nerves consist of bundles of nerve fibres in an arrangement of connective tissue. Individual nerve fibres are surrounded by a myelin sheath. Traumatic injuries to nerves have been classified in increasing degrees of severity as neurapraxia, axonotmesis, and neurotmesis. Clinically it is often not possible to distinguish axonotmesis from neurotmesis.

- Neurapraxia refers to a transient loss of nerve function and occurs when there is ischaemia or a mild stretch or compression injury with little or no structural damage to the axons or their supporting connective tissue. There is temporary loss of function for a period of days to weeks with variable motor and proprioceptive deficits although nociception is usually preserved. Neurogenic muscle atrophy is usually not a feature although there will be disuse atrophy.
- Axonotmesis occurs when there is a severe compression or stretch injury with complete disruption of axons, but the supporting connective tissue elements remain intact. There is substantial loss of motor, proprioceptive and nociceptive function. The extent of loss of nociception will be dependent on the number of axons injured. Neurogenic muscle atrophy is common with this type of injury. Return to function requires regeneration of proximal axons distally along the connective tissue scaffold to the end organ. Regrowth of axons occurs at a rate of 1–4 mm per day and recovery, if it happens, takes many months.
- Neurotmesis occurs when there is an avulsion injury or severance of a nerve. There is complete disruption of the neural and connective tissue components of the nerve with total loss of motor, proprioceptive and nociceptive function. Neurogenic muscle atrophy will be severe with this type of injury. Unless the nerve ends are surgically reunited, the proximal axons lack direction and regrow into the surrounding tissues, creating a neuroma. After surgical repair (neurorrhaphy) up to 75% of the original function may be restored but this takes many months.

The hallmarks of nerve injury include lower motor neuron signs, such as flaccid paresis or paralysis, hypo- or areflexia, rapid neurogenic muscle atrophy (within 7–10 days of the injury), and sensory impairment. The prognosis depends on the severity of axonal disruption and is worst for nerve root avulsion injuries. Stretch and compression injuries may resolve but recovery can take from several weeks to several months. Serial neurological examinations should be performed to monitor regeneration after nerve injury.

Treatment and prognosis are dependent on localization of the injury and an assessment of the severity of the neurological impairment. Skin sensation is a useful clinical tool for localizing nerve lesions. In order to assess cutaneous sensory function it is necessary to understand dermatomes and cutaneous zones and to know the location of autonomous zones. A dermatome is the area of skin innervated by a spinal nerve and a cutaneous zone is the area of skin innervated by a specific named nerve which originates from 2 or more spinal nerves (e.g. the ulnar nerve). Adjacent dermatomes and cutaneous zones usually overlap. The area of skin innervated solely by one named nerve is called an autonomous zone. The musculature innervated by a single spinal nerve is called a myotome. Autonomous zones in the forelimb and hindlimb and the muscles innervated by named nerves are shown in Table 17.3/Figure 17.5 and Table 17.4/Figure 17.6, respectively.

There are minor differences in innervation between the cat and the dog. For example in the cat the cutaneous area comparable to that supplied by the brachiocephalic nerve in the dog is supplied by a branch of the suprascapular nerve. In the cat the cutaneous areas of the palmar branches of the median nerve and the palmar branches of the ulnar nerve do

Table 17.3: Autonomous Zones and Muscle Innervation and Function in the Forelimb

Nerve	Spinal Cord Segments	Autonomous Zone	Motor Function
Suprascapular	C6, C7	Distal 2/3 of the craniomedial brachium and medial proximal antebrachium	Shoulder stabilization (Supraspinatus, Infraspinatus)
Subscapular	C6, C7	None	Shoulder stabilization (Subscapularis)
Axillary	C7, C8	Lateral brachium and distal craniolateral brachium	Shoulder flexion (Deltoid, Teres major and minor)
Musculocutaneous	C6, C7, C8	Medial antebrachium	Elbow flexion (Biceps brachii, Brachialis, Coracobrachialis)
Radial	C7, C8, T1, (T2)	Lateral antebrachium and dorsal digits II, III and medial IV	Extension of the elbow, carpus and digits, and antebrachial supination (Triceps brachii, Anconeus, Tensor fasciae antebrachia, Brachioradialis, Extensor carpi radialis, Lateral digital extensor, Ulnaris lateralis, Supinator Abductor pollicis longus, Extensor pollicis longus)
Median	C7, C8, T1	Palmar digits II, III and medial IV	Flexion of carpus and digits, and antebrachial pronation (Flexor carpi radialis, Pronator teres, Pronator quadratus, Deep digital flexor [radial head], Superficial digital flexor, Interflexorii
Ulnar	C7, C8, T1	Medial distal brachium and caudal antebrachium	Flexion of the carpus and digits (Flexor carpi ulnaris, Deep digital flexor [ulnar and humeral heads]

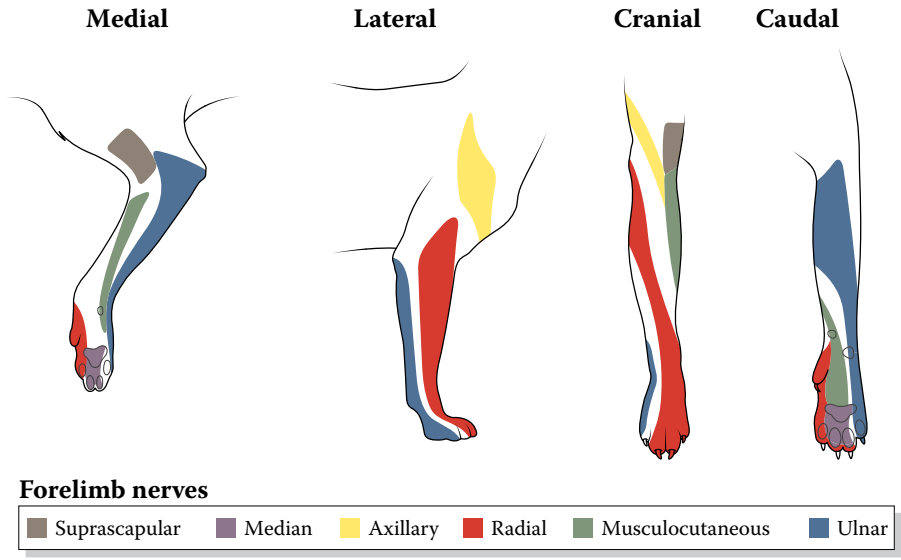


Figure 17.5 Cutaneous autonomous zones of the forelimb.

not completely overlap, therefore unlike the dog, the cat has an autonomous zone for the palmar branches of the median nerve. The cutaneous nerves and autonomous zones of the hindlimbs of the cat and dog are similar although the origin of the cutaneous nerves that arise from the lumbosacral plexus tends to be more caudal in the cat than the dog.

A variety of traumatic injuries may be associated with peripheral nerve dysfunction. Important traumatic neuropathies include brachial plexus avulsion, radial neuropathy, ischiatic and sciatic neuropathy, and sacrocaudal traction injuries. Traumatic neuropathies commonly occur in association with orthopaedic injuries such as pelvic, humeral, and femoral fractures. It is critical to evaluate these cases carefully for nerve injury that may affect the functional outcome before performing fracture repair. The particular nerve or nerve roots affected by an injury can be determined by neurological examination and can be further evaluated using electrophysiologic testing.

Table 17.4: Autonomous Zones and Muscle Innervation and Function in the Hindlimb

Nerve	Spinal Cord Segments	Autonomous Zone	Motor Function
Lateral cutaneous femoral	L3, L4, L5, L6	Craniolateral thigh and adjacent abdomen	Flexion of the lumbar spine (Psoas minor)
Genitofemoral	L3, L4, L5	Scrotum and prepuce or inguinal mammary gland, medial proximal thigh	
Caudal cutaneous femoral	L7, S1, S2, S3	Caudal thigh	
Femoral (saphenous)	L4, L5, L6	Medial thigh, crus and metatarsus	Hip flexion and stifle extension (Iliospoas [part], Sartorius, Quadriceps femoris)
Obturator	(L4), L5, L6	None	Limb adduction and lateral hip rotation (Pectineus, Gracilis, External obturator, Adductor longus)
Sciatic	L6, L7, S1, (S2)	See below	Hip extension, abduction and external rotation, stifle flexion (Gemelli, Internal obturator, Quadratus femoris, Biceps femoris, Caudal crural abductor Semitendinosus, Semimembranosus)
Common fibular	L6, L7	Lateral crus, dorsal tarsus, metatarsus and digits	Tarsal flexion and digital extension (Cranial tibial, Long digital extensor Lateral digital extensor, Peroneus longus Peroneus brevis, Short digital extensor)
Tibial	L6, L7, S1, (S2)	caudal crus, plantar tarsus, metatarsus and digits	Tarsal extension and digital flexion (Gastrocnemius, Soleus, Popliteus Superficial digital flexor, Caudal tibial Deep digital flexor, Interflexorii Short digital flexor, Quadratus plantae Interossei, Adductores digiti II and V Abductor digiti V)

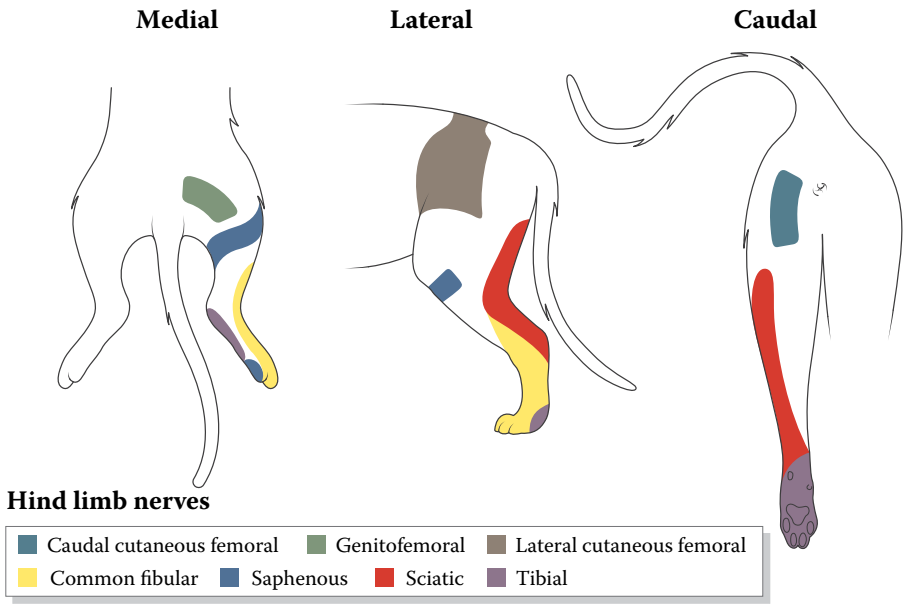


Figure 17.6 Cutaneous autonomous zones of the hindlimb.

Brachial Plexus Injury

(Bennett & Vaughan 1976, Hoffmann et al. 1996).

Avulsion injury of the brachial plexus is a well-recognized syndrome that is usually caused by vehicular trauma or falls from a height. Traction or abduction of the forelimb results in the nerve roots comprising the plexus being stretched and torn from the spinal cord. The root avulsions are typically intradural and are thought to occur at this location owing to the absence of a perineurium. The ventral nerve roots appear to be more vulnerable to injury than the dorsal roots leading to inconsistency in the pattern of sensory and motor deficits with a relatively greater degree of motor dysfunction.



Figure 17.7 Cat with left brachial plexus avulsion showing left sided Horner syndrome (Courtesy of Rob Quinn).

The brachial plexus is derived from the last three cervical and the first two thoracic nerve roots (C6, C7, C8, T1 and T2). Injuries may be partial or complete depending on the extent and the direction of the traction. Injury to the caudal nerve roots (C8–T2) or the entire plexus occurs most frequently, whereas selective cranial root (C6–7) injury is uncommon. Complete avulsion results in total paralysis of the affected limb, often with ipsilateral Horner syndrome and ipsilateral loss of the cutaneous trunci reflex because of injury to the T1-T3 and C8-T1 roots, respectively (Figure 17.7). Caudal avulsion will spare the flexors of the elbow and the limb will be carried with the elbow flexed, but the cat will be unable to extend the elbow to bear weight. Less common cranial plexus injury results in an inability to flex the elbow. The prognosis is poor unless the injury is confined to the cranial plexus in which case triceps function will be intact. Absence of nociception in the digits and absence of triceps function are poor prognostic indicators. If the nerve roots are not completely avulsed, there may be some recovery of neurological function over a 1–3-month period with supportive care and physiotherapy. Amputation is indicated if there is no improvement in limb function within a period of 3 months, there is self-mutilation, or there is abrasion of the dorsal surface of the paw. Muscle relocation techniques have been described for cases with partial neuropathy but the results are often unsatisfactory in the forelimb, probably because denervation is frequently more widespread than initially suspected. Experimentally there has been some success with reimplantation of ventral rootlets in the cervical spinal cord.

Radial Nerve Injury

(Evers 1968).

True radial neuropathy is rare and should not be confused with brachial plexus avulsion. Selective distal radial nerve injury is very rarely seen in association with humeral fractures. The entire radial nerve is sometimes injured in conjunction with fracture of the first rib. Selective distal injury is less serious since it spares the innervation to the triceps muscle and the elbow can still be extended to bear weight. Options for the treatment of permanent neurological impairment are arthrodesis, muscle relocation, and amputation. Amputation is indicated for proximal radial nerve injuries if there has been no recovery within a period of 3 months. Distal radial nerve paralysis may be managed by arthrodesis of the carpus in 20–30° of hyperextension to prevent trauma to the digits.

Ischiatic/Sciatic Neuropathy

(Bennett 1976, Bennett & Vaughan 1976, Bookbinder & Flanders 1992, Dayer et al. 2017, Jacobson & Schrader 1987, Meeson & Geddes 2017).

Ischiatic neuropathy is most commonly observed as a result of craniomedial displacement of iliac shaft fractures with impingement on the underlying ischiatic trunk. Sciatic nerve injury is rarely seen in association with femoral or acetabular fracture and hip luxation.

Partial sciatic nerve lesions in cats commonly manifest as tarsal overflexion and a plantigrade posture in the affected limb. This is rarely observed in dogs and it was thought that the explanation was that the tibial component of the nerve was more developed than the fibular component in cats and was positioned lateral to the fibular part of the nerve, under the sacral wing, making it more vulnerable to injury. However, a recent comparative cadaveric study of the surgical anatomy of the sciatic nerve in dogs and cats found no difference in the relative size or lateralization of the tibial compared to the fibular nerve between dogs and cats. The authors suggested that the difference between species might be explained by differences in the biomechanics of the caudal tibial musculature and the smaller tarsal angle in the cat when weight-bearing. Severe injury of the sciatic nerve or ischiatic trunk causes paralysis of the stifle flexor muscles and the tarsal flexors and extensors. The limb can still be protracted by flexion of the hip, and the limb will support weight as the stifle extensor muscles are unaffected, but the paw will be placed on its dorsal surface (Figure 17.8).

If the sciatic nerve has not been completely severed (neurapraxia or axonotmesis), recovery of partial or complete neurological function may occur over a period of 1–3 months. If it is known that the nerve has been transected (neurotmesis), for example if the injury is iatrogenic (see below), the ends should be surgically anastomosed. This is relatively easy for true sciatic nerve injury, but exposure is very difficult in the region where the ischiatic trunk lies medial to the pelvis.



Figure 17.8 A cat with traumatic sciatic nerve injury (Courtesy of Rob Quinn).

Muscle relocation techniques can only be used in selected cases where there is remaining functional muscle that can be transferred to replace the function of a paralyzed muscle. Injuries are more likely to involve a single nerve, or a component of a single nerve, in the hindlimb than the forelimb, so muscle relocation is more appropriate for restoration of hindlimb function. In cats with fibular nerve paralysis a side-to-side anastomosis is performed between the tendons of insertion of the functional long digital flexor and the nonfunctional long digital extensor muscles. The relocated muscle acts as a digital extensor and hock flexor. In cats with sciatic neuropathy affecting both the fibular and tibial components, use can be made of the functional femoral nerve by relocation of the tendon of origin of the nonfunctional long digital extensor muscle in combination with pantarsal arthrodesis. The tendon is relocated to the lateral aspect of the patellar retinaculum so that quadriceps contraction causes synchronous extension of the digits.

Ulnar Neuropathy

(Perry 2015, Streubel et al. 2012, Streubel et al. 2015).

Ulnar neuropathy has been reported in association with medial humeral epicondylitis (MHE). In this condition, there is a tendinopathy and enthesopathy of the flexor carpi ulnaris muscle with degenerative changes that include dystrophic mineralization in the tendon of origin. The cause of MHE is not fully understood, but is presumed to be single or repetitive trauma or overuse causing partial of complete avulsion of the tendon. Radiographic and histologic evidence of MHE was reported in 10% of cats in a postmortem study of 60 cats.

A proportion of cats with MHE develop lameness associated with caudal displacement of the ulnar nerve by the mineralized tissue. Nerve entrapment occurs between the new bone formation in the humeral head of the flexor carpi ulnaris muscle and the epitrochleoanconeus muscle. Lameness is generally moderate, gradual in onset and progressive although an acute onset is seen in some cats. Affected cats may be reluctant to jump down from heights or may land awkwardly when they do. A pain response can usually be elicited by digital palpation caudodistal to the medial epicondyle. For those cases that do not respond to conservative management surgical excision of abnormal tissue is usually curative. MHE is discussed further in Chapter 9.

Iatrogenic Neuropathy

(Autefage et al. 1990, Fanton et al. 1983, Forterre et al. 2007, Liska et al. 2010, Palmer et al. 1988, Withrow & Amis 1977).

Acute iatrogenic nerve injury most commonly involves the sciatic nerve as a result of inadvertent impalement of the nerve during retrograde intramedullary pinning of femoral fractures. Injections into the gluteal or caudal thigh muscles may cause acute sciatic injury either as a result of direct injury by the needle, the drug itself, and/or the vehicle injected. Injection of irritant substances may also cause a more chronic injury owing to progressive fibrosis involving the nerve. Acute injury may occur during closed reduction of hip luxation and procedures that involve a caudal approach to the hip joint, internal fixation of acetabular fractures, femoral head and neck ostectomy, total hip replacement and repair of sacroiliac luxation. Femoral fracture repair with a medially misplaced or excessively long intramedullary pin is the most common cause of chronic injury. When the pin lies in close proximity to the nerve pressure is placed on the nerve by the surrounding soft tissues as the limb moves. Additionally, fibrous tissue which forms around an excessively long pin may entrap and progressively compress the sciatic nerve.

The distal radial nerve may be injured when performing internal fixation of humeral fractures or during the application of an external fixator to the proximal forelimb.

Focal Tetanus

(Baker et al. 1986, Bianchi et al. 2013, De Risio L, Gelati A, 2003, Greene 2012, Jain et al. 1982, Lee & Jones 1996, Malik et al. 1989, Polizopoulou et al. 2002).

Technically tetanus is a spinal cord disorder, nonetheless it is included here because the clinical signs may resemble those of a myopathy.

Cause and Pathogenesis

Tetanus is caused by the obligate anaerobic spore-forming bacillus, Clostridium tetani, which produces the neurotoxin tetanospasmin. The toxin undergoes retrograde neuronal transport from the neuromuscular junctions adjacent to the site of infection to exert its effect on inhibitory neurotransmission in the central nervous system. The toxin interferes with the release of inhibitory neurotransmitters by interneurons (e.g. glycine and GABA) in the spinal cord ventral grey horn (Renshaw cells) and brainstem motor nuclei. This results in uncontrolled and persistent skeletal muscle contraction most evident in the extensor muscles. Cats are less susceptible to tetanus than most other domestic species, being 10 times less susceptible than dogs, because of an inability of this neurotoxin to penetrate the nervous system easily and attach to binding sites. Generalized tetanus is easily recognized, and the diagnosis is usually straightforward. Focal tetanus is a rare manifestation of tetanus that has been reported predominantly in the cat. The clinical signs result from the action of the neurotoxin on the inhibitory neurons in the spinal cord that innervate the local muscle groups adjacent to the site of the infection.

Clinical Signs

When a single limb is affected the condition presents as a cause of non-weight-bearing lameness with continuous involuntary contraction of the muscles innervated by the same or adjacent spinal nerves. Sensory perception is not impaired and affected cats are otherwise mentally alert and healthy. There is usually a history of a penetrating wound or surgery up to 3 weeks before the onset of signs, close to or distal to the spastic muscle groups (Figure 17.9). Focal



Figure 17.9 A cat with local tetanus of the hindlimbs and tail following castration.

tetanus occasionally progresses to the generalized form and there is also a single report of progression to severe spastic tetraparesis with no intracranial signs.

Diagnosis

The diagnosis is based on the history and clinical signs and may be confirmed by finding the organism in Gram-stained smears if a contaminated wound is still present. There will be characteristic abnormalities on electromyography and creatine kinase (CK) will be elevated. Electromyography will show non-specific spontaneous persistent post-needle motor unit insertion activity in the affected muscles during rest and under general anaesthesia. In a recent study of two cats, there were also significant changes in the F wave (increased F/M amplitude in both cats, and increased F wave duration in one cat). Haematology may show neutrophilic leucocytosis if there is an active infection. Serum concentrations of antitetanus antibody may or may not be elevated.

Treatment and Prognosis

Treatment is directed at controlling the infection, neutralizing the toxin, and relieving the spasticity. If a wound is found it should be treated appropriately by lavage and debridement. Irrespective of whether an active infection is identified antibiotics effective against the bacterium should be administered for 14 days. Penicillin G is traditionally the antibiotic of choice and can be given both intravenously in the aqueous form and intramuscularly as the procaine salt. Metronidazole may be more effective than penicillin G, although it carries a higher risk of toxicity. Other drugs with activity against the bacterium include tetracycline and clindamycin. Tetanus antitoxin can be administered early in the course of the disease to prevent further binding of circulating tetanospasmin toxin to the central nervous system, but it has no effect on toxin that is already bound to interneurons and there are concerns about anaphylaxis, so it is rarely used in cats. The prognosis is good, although resolution is gradual and may take several months.

MYOPATHIES:

Myopathies are rare in cats occurring less frequently than neuropathies and junctionopathies. Cats with polymyopathies present with generalized paresis. Other presenting signs include exercise intolerance, fatigue, stiff stilted gait, and often, neck ventroflexion. Muscle swelling, myalgia, or atrophy may also be present, although muscle hypertrophy may occur in some myopathies. In contrast to the polyneuropathies, because the lower motor neurons and sensory neurons are generally unaffected sensory pain perception, spinal reflexes, and proprioception are not impaired (Table 17.3). Certain disorders of the neuromuscular junction may mimic the signs observed in a generalized myopathy. An elevation of creatine kinase (CK) occurs when there is myonecrosis associated with an inflammatory myopathy but increased CK alone is not diagnostic of myositis. Differentiation of many of these disorders depends on the examination of a properly processed muscle biopsy specimen. Myopathies can be divided into inherited and acquired types. Idiopathic myopathy is an acquired myopathy where the underlying cause cannot be determined.

Inherited/Degenerative Polymyopathies

Nemaline Rod Myopathy

(Cooper et al. 1986, Kube et al. 2006).

Nemaline or rod myopathy is a rare inherited disorder characterized by rod-shaped nemaline inclusions within myofibers. Clinical signs, which appear at 6–18 months of age, include progressive paresis with reluctance to walk and a hypermetric gait. There is also muscle atrophy, tremors, and hyporeflexia. Electrophysiology is normal and CK is only marginally elevated, so diagnosis is based on muscle biopsy.

X-Linked (Dystrophin-Deficient) Muscular Dystrophy

(Carpenter et al. 1989, Gashen et al. 1994, Shelton & Engvall 2002).

Dystrophin is a cytoskeletal protein that is vital in maintaining the integrity of the basement membrane (sarcolemma) of the myofibre. Dystrophin-deficient muscular dystrophy is an inherited disorder characterized by progressive, mostly noninflammatory, degeneration of skeletal muscle typically in young male cats 5–24 months of age. There is generalized muscle hypertrophy including the tongue, larynx, diaphragm, and heart. Diagnosis is based on the signalment and clinical signs of marked symmetrical muscle hypertrophy, tongue enlargement, difficulty in walking, and a stiff bunny-hopping gait. A peculiarity of the condition is the presence of calcified nodules on the tongue. Serum CK levels are often dramatically elevated and there are severe generalized electromyographic changes with spontaneous activity characterized by complex repetitive discharges. Histopathological examination of muscle biopsies reveals muscle pallor and myofibre necrosis. Definitive diagnosis requires dystrophin immunostaining. The prognosis is poor, in particular, owing to the cardiac, diaphragmatic and lingual muscle hypertrophy.

Muscular Dystrophy with Laminin Alpha 2 Deficiency

(Awamura et al. 2008, O'Brien et al. 2001, Poncelet et al. 2003, Shelton & Engvall 2002).

Laminin alpha 2 (merosin) is a large glycoprotein component of the sarcolemma. The glycoprotein also forms an important component of the Schwann cell basement membrane. Dystrophic myopathy and demyelinating neuropathy associated with absence of laminin alpha 2 has been described in two unrelated cats in the USA. One cat was a young female Siamese with muscle atrophy and marked weakness and the other was a young female domestic shorthair with progressive spasticity beginning at 6 months of age. There was moderate elevation of serum CK in both cases. Laminin alpha 2 deficiency has also reported in a young European-bred Maine coon and in a cross-Persian cat. Myopathy in these cases was characterized by progressive weakness, muscle atrophy, and joint contracture. The diagnosis can be confirmed by demonstrating an absence of laminin alpha 2 on immunohistology of muscle biopsies.

Myositis Ossificans (Fibrodysplasia Ossificans Progressiva)

(Asano et al. 2006, Klang et al. 2013, Norris et al. 1980, Valentine et al. 1992, Warren & Carpenter 1984, Yabuzoe et al. 2009).

Myositis ossificans is a rare idiopathic condition characterized by progressive fibrosis and heterotopic ossification of the connective tissues associated with skeletal muscle.

The localized form results in firm enlargement of one muscle or a group of muscles and there is associated myalgia and muscle atrophy. Fibrovascular proliferation may also occur within the muscles themselves with secondary calcification and ossification.

The generalized progressive form typically affects young to middle-aged cats of either sex. Firm nodules develop anywhere on the body but especially on the back and neck. The gait becomes stiff with the hindlimbs often worst affected and may progress to difficulty walking. Proximal limb muscles become firm and painful on palpation and joint range of motion becomes restricted.

The aetiology is unknown although the localized form may be secondary to trauma, infection, ossifying haematoma or metaplasia of connective tissue and muscle to cartilage and bone. The progressive form is thought to be congenital or inherited and may be due to a disorder of fibroblasts in collagenous connective tissue.

Radiography reveals extensive soft tissue mineralization. The masses may be confused with extraskeletal osteosarcomas. Creatine kinase levels are usually elevated and muscle biopsy shows differentiated cartilage and bone within the muscle, fibrosis, myofibre necrosis and phagocytosis. The prognosis for the generalized form is poor with development of severe disability within a period of several weeks to several months. Focal lesions may regress or may be amenable to surgical excision. See Chapter 18 for further discussion of Myositis ossificans.

Myotonia Congenita

(Gandolfi et al. 2015, Hickford et al. 1998, Toll et al. 1998, Vitae 2002).

Myotonia is the continued active contraction of a muscle after the cessation of voluntary movement. Myotonia congenita is characterized by muscle hypertrophy, spasm, stiffness, and inability to initiate movement. The condition is a skeletal muscle channelopathy that is inherited as an autosomal recessive trait. Failure of muscle relaxation is associated with abnormal sarcolemmal chloride conductance which leads to hyperexcitability of the muscle membrane and sustained muscle contraction following initial depolarization. A genetic test is available to identify affected cats and carriers of the mutant gene.

Affected kittens walk with an awkward stiff gait that is worse after rest and in cold weather. If they walk on carpet the kittens tend to snag their claws. When startled they may fall into lateral recumbency in rigid hyperextension for approximately 10 seconds. Startling also results in bilateral protrusion of the nictitating membrane, blepharospasm, retraction of the lips and flattening of the ears. There may also be dysphonia manifested as a quiet purr and a hoarse meow.

Palpation reveals widespread muscle hypertrophy especially of the gastrocnemius muscles. Creatine kinase levels are often normal or only mildly elevated. Muscle biopsy changes are mild and non-specific. There is characteristic dimpling of the tongue and skeletal muscle under general anaesthesia. Electromyography shows spontaneous trains of repetitive waxing and waning discharges after insertion of the electrode, producing an audible dive-bomber or motorcycle sound.

Drugs used to control canine myotonia such as procainamide are unsuitable for use in cats because of the increased risk of toxicity in this species. Clinical signs tend to stabilize by 6–12 months of age and mildly affected cats may have a reasonable quality of life with environmental modification alone.

Acquired Myopathies

Traumatic Myopathy

Skeletal muscle is composed of an arrangement of striated muscle fibres or myofibrils and connective tissue sheaths. Injuries heal by a combination of fibrosis and regeneration of myofibrils. Excessive postoperative scar tissue formation compromises function and can be minimized by careful tissue handling, debridement of necrotic tissue, and accurate muscle apposition. Muscle should be sutured using an absorbable material in a horizontal mattress pattern placed through the holding layer, which is the external connective tissue sheath or epimysium. Traumatic injuries to muscles and tendons are called strains and are classified as first degree (mild), second degree (moderate), and third degree (severe). A strain can affect any of the components of the muscle–tendon unit, i.e. the tendon of origin or insertion, the musculotendinous junction, or the muscle belly itself. Such injuries are commonly seen in working or racing dogs but are rarely recognized in the cat.

Injury causes disruption of muscle and/or collagen fibres, haemorrhage, and inflammatory oedema. The goal of treatment in the acute phase is the reduction of haemorrhage and swelling in an attempt to limit subsequent scar tissue formation and muscle contracture. This is achieved by restriction of activity and application of cold to the site of injury. A nonsteroidal anti-inflammatory drug (NSAID) should be administered by injection to help reduce swelling and relieve pain. Third-degree strains frequently involve rupture of the tendon or musculotendinous junction and require surgical repair.

Fibrotic Myopathy of the Semitendinosus Muscle

(Lewis 1988).

Fibrotic myopathy is a condition in which muscle tissue is replaced by fibrous connective tissue. A case comparable to that affecting the semitendinosus muscle in dogs has been reported in a Himalayan cat. The cat presented with lameness, characterized by decreased abduction of the limb and marked flexion of the hip, stifle and hock as the limb was advanced. The limb could not be fully extended, and the foot was placed abruptly with intermittent knuckling of the paw. Surgical exploration revealed a firm pale semitendinosus muscle caused by fibrosis of unknown origin. A full range of normal movement was restored using a Z-plasty lengthening procedure. The lameness recurred within 2 months of surgery, but limb abduction was maintained.

Brachialis Contracture

(Taylor & Tangner 2007).

There is a single case report of a 13-year-old domestic shorthair cat with chronic non-painful weight bearing lameness of a forelimb and an inability to extend the right forelimb past a 90° angle. The only gait abnormality present was a shortened stride. There was no history of injury or trauma. A taut, non-painful muscle band was palpated over the craniolateral aspect of the brachium. A tentative diagnosis of brachialis muscle contracture was made, and the cat was treated surgically by sectioning the muscle and tendon of insertion. The brachialis muscle was normal in colour but smaller than normal and very firm. Surgery provided

immediate relief of the contracture and restored a normal range of motion. Histopathology revealed replacement of skeletal muscle with dense fibrous connective tissue.

An examination eight months later showed that the contracture had once again returned to the same degree at which it was first seen. A similar fibrotic band involving the brachialis muscle was detected on palpation. A second surgery was performed and release of the brachialis muscle and tendon once again provided immediate post-operative relief of the contracture.

A single injection of triamcinolone was given intramuscularly at 2.2 mg/kg to help prevent further adhesions followed by physiotherapy. At follow up five months later the cat was reported to be doing well with normal range of motion and was not lame. The authors suggest that the case most likely represents a muscle contracture secondary to trauma, chronic irritation, or fibrotic myopathy. The case is of particular interest as, at the time of writing, contracture of the brachialis muscle has not been reported in dogs.

Digital Flexor Tendon Contracture

(Cooper et al. 2005, Cabon et al. 2015, Thom et al. 2017).

Digital flexor tendon contracture has been described in three reports in a total of seven cases comprising three Devon Rex cats, three Siamese or Siamese cross and one domestic shorthair. All cats presented with abnormal flexion of the forelimb phalanges and a severe weight bearing forelimb lameness. In five cats there was a possible association with previous onychectomy, which is a commonly performed surgical procedure in some countries. In those cats that had undergone previous onychectomy this was between 6 weeks and 9 years prior to the onset of clinical signs. In two of the Devon Rex cats there was also musculotendinous contracture and flexural deformity of the carpi and in this breed the authors suggested an underlying immune-mediated myopathic process and a possible genetic predisposition to the contracture.

The deep and superficial digital flexor tendons are responsible for digital flexion. The deep digital flexor tendon is responsible for flexion of the distal interphalangeal joint between P2 and P3 and the superficial digital tendon enables flexion at the level of the proximal interphalangeal joint between P1 and P2. Following onychectomy, which involves surgical resection of P3, digital flexion is maintained by the superficial digital flexor tendons. Contracture results in hyperflexion of the phalanges such that the cat is forced to ambulate on the distal aspect of P2. Treatment by tenectomy or tenotomy of the affected flexor tendons in order to release the contracture has been curative.

Quadriceps Contracture

(Bardet 1987, Carberry & Flanders 1986, Fries et al. 1988, Leighton 1981, Liptak & Simpson 2000, Tisdall & Rogowski 2017).

Quadriceps contracture occurs as either a congenital abnormality or more commonly is acquired as a complication of mid diaphyseal and distal femoral fracture in immature cats (Figure 17.10). Quadriceps contracture is less common now than it used to be as a result of a better understanding of the aetiology and improved fracture repair techniques. The condition is characterized by progressive hindlimb lameness, rigid stifle hyperextension and atrophy of the quadriceps muscles. In kittens, severe

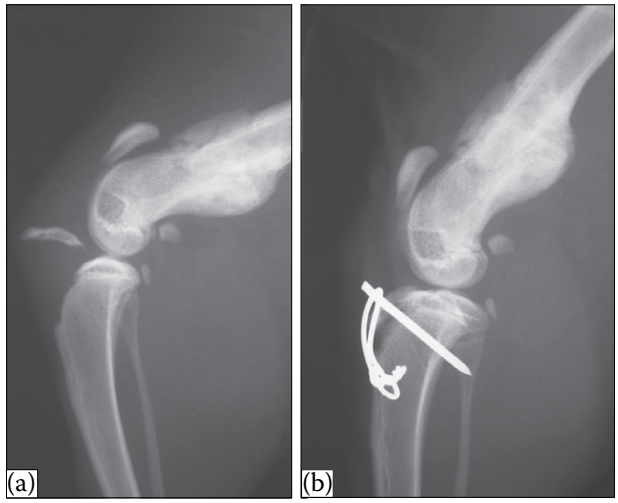


Figure 17.10 Quadriceps contracture. **(a)** Mediolateral radiograph of the stifle joint of an immature cat with quadriceps contracture following a distal femoral fracture, showing avulsion of the tibial tuberosity. **(b)** Tibial fracture repair using a Kirschner wire and figure of eight tension band wire.

contracture leads to the condition of genu recurvatum, in which the stifle is bent backwards.

The aetiology includes adhesion of the quadriceps muscle group to an exuberant femoral callus, delayed fracture repair, damage to muscles and nerves or vascular compromise associated with trauma or fracture repair, prolonged immobilization and osteomyelitis. There may be an increased risk of this complication when treating femoral fractures in young cats following biological principles of repair, because of poor fragment reduction and prolific callus formation associated with these techniques. Following contracture, degenerative changes occur in articular and peri-articular tissues, which become irreversible if not recognized and treated promptly. Other sequelae include proximal displacement of the patella, avulsion of the tibial tuberosity, disuse osteopaenia, stifle osteoarthritis, limb shortening and coxofemoral subluxation or luxation.

The prognosis for return of full function is poor, conservative and surgical treatments may be unrewarding, and the focus should be on preventing this condition. Surgical treatment options include release of adhesions, muscle-lengthening procedures, vastus intermedius excision, stifle arthrodesis, and limb amputation. Successful management has been described in two reports. In one report a 6-month-old cat that developed contracture following plating of a comminuted femoral fracture was managed by surgical release of adhesions between the quadriceps and the fracture callus, placement of an autogenous fat graft between the quadriceps and the femur and application of a dynamic flexion apparatus in combination with intensive physiotherapy. In the second report a 6-month old cat was treated successfully for bilateral quadriceps contracture which developed following surgical stabilization of bilateral femoral fractures. Initial treatment which included physical therapy, surgical mobilization, autogenous fat grafting and use of a dynamic flexion apparatus resulted in only temporary improvement. However, the addition of prednisolone to the treatment regimen at a reducing dosage over a

period of 8 weeks, coincided with immediate clinical improvement and long-term resolution of the contractures.

Hypokalaemic Myopathy

(Blaxter et al. 1986, Dow et al. 1987, Dow et al. 1989, DiBartola 2001, Dickinson & Lecouteur 2004, Edwards & Belford 1995, Malik et al. 2015, Nemzek et al. 1994).

Cause and Pathogenesis

Hypokalaemic myopathy is a generalized acute onset polymyopathy, associated with low extracellular potassium levels, that is relatively common in the cat. The condition occurs frequently in cats with chronic renal insufficiency because there is excessive loss of potassium in the urine. Other conditions associated with hypokalaemia include hyperthyroidism, diuretic therapy with potassium wasting diuretics, chronic gastrointestinal disease, inadequate dietary potassium, excessive administration of fluids and primary hyperaldosteronism (Conn syndrome). Potassium loss may be exacerbated by some diets that are acidified to reduce the development of crystalluria and urolithiasis.

Periodic hypokalaemic polymyopathy is a genetic disease of Burmese kittens in the first year of life. Affected kittens lack an enzyme that is present primarily in the distal nephron that is involved in the sodium/potassium exchange mechanisms in the kidney. Deficiency of the enzyme is thought to cause a potassium wasting nephropathy.

The most common presentation is of an older cat with renal dysfunction. The hypokalaemia further compromises renal function, thus establishing a vicious cycle of renal dysfunction and potassium depletion.

Hypokalaemia leads to hyperpolarization of the resting membrane potential of the sarcolemma so that it becomes refractory to depolarization and subsequent contraction. In comparison with dogs, cats appear to be particularly susceptible to electrolyte abnormalities, although the mechanisms to account for this are not fully understood.

Clinical Signs

Clinical signs are typically acute and include generalized muscle weakness, cervical ventroflexion, reluctance to move, stiff gait and myalgia. In some cases, reduced exercise tolerance and lethargy may be noticed before obvious weakness becomes apparent. In severe cases respiratory paralysis and rhabdomyolysis can occur. Clinical signs of renal disease may also be present. In Burmese kittens, clinical signs are similar, although weakness is usually periodic or episodic, occasionally it is continuous. Episodes are often acute and transient and may be precipitated by vigorous exercise or stress.

Diagnosis

Diagnosis is based on clinical signs, low serum potassium (<3.0 mmol/l), moderate to marked elevation of CK and assessment of renal function. Abdominal ultrasound should be performed for renal evaluation and to rule out underlying adrenal neoplasia supporting Conn syndrome. Muscle biopsy, if performed, may show either no changes or evidence of mild myonecrosis. Rapid resolution of clinical signs with potassium supplementation supports the diagnosis.

The diagnosis of Burmese hypokalaemia is based on a PCR test to identify affected homozygous cats as well as carriers of the genetic mutation. Elimination of the condition from the Burmese breed and other related breeds such as Tonkinese, Tiffanie and Bombay should be possible in the future.

Treatment and Prognosis

Muscle weakness improves within 24–72 hours of the commencement of treatment with oral potassium gluconate (5–8 mEq/kg/day divided twice daily). Maintenance therapy is 2–4 mEq/day divided twice daily after achieving normal serum potassium levels. In some Burmese cats with hypokalaemia signs of disease may resolve after 1–2 years of age without the need for further treatment.

Infectious Myopathy

(Mansfield et al. 1984, Poonacha et al. 1982, Podell et al. 1998).

Feline bacterial myositis is a common occurrence secondary to wounds and bites. Cats with deep abscesses or cellulitis of a limb typically present with lameness, depression, and pyrexia. *Pasteurella* multocida is the most frequently isolated organism. Clostridial myositis is rare and is characterized by palpable crepitus owing to accumulation of gas in the tissues. Clinical signs include severe focal or multifocal myalgia. There may be a history of a wound or recent surgery. Treatment involves aggressive surgical debridement and appropriate antibacterial therapy. Infectious myopathies caused by fungal or viral proliferation are very rare. Feline immunodeficiency virus has been shown to cause a subclinical myopathy in experimentally infected cats.

Protozoal Myopathy

(Dubey et al. 1990, Dubey & Carpenter 1993, Hartmann et al. 2013, Kirkpatrick et al. 1986, Troxel 2009, Zitzer et al. 2017).

Toxoplasma gondii is an obligate intracellular coccidian parasite that can produce an inflammatory polymyopathy often with respiratory, ocular, and central nervous system signs. Involvement of the liver and pancreas is less common. In a large series of histologically confirmed cases of toxoplasmosis the most frequent presentation was persistent pyrexia unresponsive to routine antibiotic therapy. Clinical signs include seizures, ataxia, muscle hyperaesthesia, dyspnoea, uveitis, diarrhoea, icterus, anorexia and weight loss. Because intraocular inflammation is a common finding, ophthalmic examination should be performed routinely in all cats with suspected toxoplasmosis.

Cats are the definitive host for this parasite and pass oocysts in the faeces. Infection may occur through ingestion of any of the three life stages of the organism or transplacentally. Toxoplasma gondii infection in cats is common but the clinical disease is rare. Up to 50% of cats have antibodies indicating previous infection and the presence of cystic stages in the tissues. However, infection is generally subclinical; clinical signs are most frequently associated with postnatal infection. Immunosuppression in cats infected with FIV or FeLV may allow reactivation of cystic stages. Commonly used doses of glucocorticoids do not cause reactivation of cysts. Administration of cyclosporin to cats with skin disease or with renal transplants has been associated with the onset of clinical disease.

Diagnosis of clinically active Toxoplasma infection is based on clinical signs, tissue biopsy, immunofluorescence assay (IFA), and response to therapy. All infected cats

develop IgG and IgA antibody isotypes and about 80% also have IgM antibodies. IgG antibodies take 4–6 weeks to appear and maximum antibody titres are reached within 2–3 weeks of first appearance. Antibodies are commonly found in both healthy and symptomatic cats thus their presence merely proves exposure and does not signify that the cat has clinical toxoplasmosis. Definitive antemortem diagnosis relies on PCR or microscopic examination of body fluids (e.g. CSF, aqueous humour, bronchoalveolar lavage fluid), tissues samples or fine needle aspirates of organs (e.g. lymph nodes), for distinctive pathological changes and the presence of the organism. A tentative diagnosis can be based on the presence of significant IgM antibodies to Toxoplasma especially if there is a rising titre on consecutive serum samples.

The drug of choice for treatment is clindamycin at a dose of 40 mg/kg orally in divided doses for 4–6 weeks. Transient vomiting is a common side-effect of clindamycin in cats. Some improvement in clinical signs is to be expected within the first 2–3 days of clindamycin administration.

Neospora caninum does not appear to affect cats naturally, although experimental infection of kittens has resulted in fatal encephalomyelitis and myositis. Sarcocystis species have been identified in feline skeletal muscle and Sarcocystis neurona-like parasitaemia has been demonstrated in a clinically ill cat.

Idiopathic Polymyositis

(Carpenter & Holzworth 1982, Dickinson & Lecouteur 2004, Ginman et al. 2009, Mayousse et al. 2017).

Polymyositis is an acquired diffuse inflammatory myopathy that is less commonly reported in cats than dogs. The cause is not always known although the response to immunosuppressive therapy in many cases suggests an immune-mediated pathogenesis. In some cases, immune-mediated polymyositis occurs as a paraneoplastic complication and there is an association with other immune-mediated disorders such as myasthenia gravis and polyneuritis. In one report of eight cats with suspected myasthenia gravis and concurrent polymyositis, six cats had confirmed acetylcholine receptor antibody seropositive myasthenia gravis and five cats had a cranial mediastinal mass (three of which were confirmed as thymoma).

Reported cases have been 6 months to 14 years of age and there is no identified breed predisposition. Clinical signs are acute and symmetrical and include pronounced neck ventroflexion, exercise intolerance, generalized weakness, and variable myalgia. There may be regurgitation and secondary aspiration pneumonia associated with megaoesophagus. The serum CK level is usually moderately or markedly elevated and there is EMG evidence of muscle disease. Muscle biopsies demonstrate myonecrosis and regeneration with mononuclear cell (lymphocytes and macrophages) inflammatory infiltrates. Toxoplasma gondii, FeLV and FIV should be ruled out and concurrent hypokalaemia if present should be treated before making a diagnosis of idiopathic polymyositis. The prognosis for cats without megaoesophagus is generally good; some cats recover spontaneously, and others respond to immunosuppressive doses of glucocorticoids (prednisolone 4–6 mg/kg daily in divided doses initially and then tapered over 8 weeks) although relapses may occur. Long-term administration of glucocorticoids may be necessary in some cats.

Paraneoplastic Myopathy

(Carpenter & Holzworth 1982).

A low-grade polymyositis is seen occasionally in cats with thymoma and other malignancies. In a series of 11 cats with thymoma, three cats had polymyositis. It has been postulated that the neoplasm causes T cell proliferation against an unidentified muscle antigen. Cats with histopathologically confirmed myositis that relapse or fail to respond to immunosuppressive glucocorticoid therapy should be screened for occult malignancy.

JUNCTIONOPATHIES:

Neuromuscular junction disorders generally manifest as progressive loss of muscle strength with exercise or complete flaccid paralysis (Table 17.2). Histopathological changes are usually absent or non-specific and diagnosis is usually based on the results of biochemical, immunological and electrodiagnostic testing. Junctionopathies are less common in the cat than the dog.

Myasthenia Gravis

(Bell et al. 2012, Day 1997, Ducote et al. 1999, Hague et al. 2015, Indreiri et al. 1983, Malik et al. 1997, Shelton 1998, Shelton et al. 2000, Shelton 2002).

Causes and Pathogenesis

Acquired myasthenia gravis is an immune-mediated disease associated with the production of antibodies directed against the acetylcholine receptor. The resultant depletion of functional acetylcholine receptors on the postsynaptic membrane of the neuromuscular junction reduces the sensitivity of the neuromuscular junction to acetylcholine and clinical signs of paresis occur because of decreased neurotransmission. The basis for the autoimmune disorder is unknown in many cases although there may a genetic predisposition. Congenital myasthenic syndromes are hereditary disorders in which there is a structural or functional defect at the neuromuscular junction that affects neuromuscular transmission.

Myasthenia gravis in cats shows distinct differences from the disorder in dogs both in the clinical presentation and the frequency of spontaneous remission. In cats, myasthenia gravis is more frequently associated with a thymic disorder (thymoma or non-neoplastic thymic disease) than it is in dogs with 52% of cats having a cranial mediastinal mass in a recent study of 235 cats. Myasthenia gravis has been reported 2–4 months after initiation of therapy with the antithyroid drug, methimazole because of the immunomodulatory properties of the medication, and should be suspected in any hyperthyroid cat that develops muscle weakness following drug therapy. All breeds may be affected, but Abyssinian and Somali cats are overrepresented. Congenital myasthenia gravis has been reported in kittens but is very rare. The condition is a hereditary developmental disorder that results in a deficiency of functional acetylcholine receptors on the postsynaptic membrane.

Clinical Signs

The spectrum of clinical signs is similar to those seen in dogs, but there are notable differences. The predominant clinical sign is generalized exercise-induced muscle weakness without megaoesophagus. However, in comparison with dogs, cats are difficult to exercise in the hospital

environment. Cats usually exhibit reluctance to move around, and there may be neck ventroflexion and muscle tremors. Clinical signs are typically less episodic than in the dog. Affected cats often move with a crouching gait and then either flop over onto their sides or lie with their head resting on their paws. The head and neck are often carried in a flexed position. Megaoesophagus and regurgitation are less common in cats than dogs because only the proximal portion of the oesophageal wall has striated muscle in the cat.

In a large series of cases over half the cats presented with either generalized weakness or generalized weakness associated with a cranial mediastinal mass. An acute fulminating form manifesting as severe appendicular muscle weakness and respiratory muscle paralysis has been recognized. Focal forms of myasthenia gravis affecting muscles innervated by cranial nerves are less common in cats than dogs accounting for about 15% of feline cases. Focal myasthenia gravis manifests as weakness of specific muscle groups such as the oesophagus, pharynx, larynx or facial muscles and this may occur singly or in combination.

Diagnosis

Diagnosis is based on clinical signs and the response to the intravenously administered ultra-short acting anticholinesterase edrophonium chloride at a dose of 0.25–0.5 mg/kg (Tensilon test). When performing the Tensilon test an intravenous catheter should be placed and the cat should be pretreated with atropine to prevent overstimulation of the acetylcholine receptor which causes a depolarizing blockade. In positive cases there is a dramatic improvement in muscle strength, which lasts for a few minutes. Circulating antibodies to acetylcholine receptors are usually found in the acquired form of the disease. Samples must be submitted to a specialist laboratory that uses a feline-specific antigen to measure acetylcholine receptor antibodies. There is a frequent association with thymoma, so cats with a cranial mediastinal mass should be tested for myasthenia gravis prior to surgical removal of the mass, even in the absence of muscle weakness. Conversely, all cats with myasthenia gravis should be radiographed to screen for thymoma even in the absence of clinical signs of oesophageal dysfunction or respiratory distress.

Treatment and Prognosis

In contrast to the condition in dogs, spontaneous remission is uncommon and cats with myasthenia gravis should be treated with the long-acting cholinesterase inhibitor, pyridostigmine bromide (Mestinon), at a dosage of 0.25 mg/kg orally every 8–12 hours. Immunosuppressive therapy may be more effective than treatment with anticholinesterase drugs. Cats are less prone to develop exacerbation of muscle weakness and are more resistant to the side-effects of high doses of glucocorticoids. Prednisolone is administered at a dose of 2–4 mg/kg divided twice daily for a minimum of 2 weeks, reducing to alternate day therapy once remission is achieved. Thymectomy should be considered for cats with thymoma or those that respond poorly to medical therapy. However, in a recent retrospective study of 235 cats the clinical outcome of cats with a cranial mediastinal mass was the same with medical or surgical management. The prognosis may be better for myasthenia gravis in cats than dogs, probably because of the lower incidence of megaoesophagus and aspiration pneumonia although in the same study 58% of cats were euthanased. The acute fulminating form carries a grave prognosis.

Devon Rex and Sphynx Congenital Myasthenic Syndrome (Hereditary Myopathy)

(Abitbol et al. 2015, Gandolfi et al. 2015, Malik et al. 1993, Martin et al. 2008, Robinson 1992).

Rapidly progressive muscle weakness with an autosomal recessive pattern of inheritance has been described in Devon Rex and Sphynx cats. Some cats may have reduced expression of alpha-dystroglycan which is involved in the linkage of the cytoskeleton to the extracellular matrix and helps to confer structural stability to the sarcolemma during contraction. Recently the disease has been shown to be due to a congenital myasthenic syndrome and the underlying genetic defect has been identified. Clinical signs, which appear from 3 weeks to 6 months of age, include generalized muscle weakness and tremor, head bobbing, and megaoesophagus. Affected kittens walk with a hypermetric gait with protrusion of the scapulae, develop ventroflexion of the head and neck, and at rest often adopt a characteristic 'dog-begging' or 'chipmunk' posture with the forepaws resting on a convenient raised surface. The neck often remains permanently flexed, causing difficulty in chewing and swallowing food. Serum CK is not elevated. Histopathological examination of muscle biopsies shows a variable myopathy with the cervical and proximal limb muscles preferentially affected. Affected cats deteriorate most rapidly up to 6–9 months of age but can have a reasonable quality of life if kept indoors. Premature death due to food-related asphyxiation or aspiration pneumonia by 2 years of age is not uncommon. A DNA test is available to identify carriers of the mutation and is recommended for the Devon Rex and Sphynx breeds.

Organophosphate and Carbamate Toxicity

(Blodgett et al. 1996, Dickinson & LeCouteur 2004, Jaggy & Oliver 1990, Nafe 1998, Wismer & Means 2011).

Toxicity associated with organophosphates and carbamates has become less common as these compounds are phased out in favour of newer insecticides with mechanisms of action that target insects more specifically and not mammals. Organophosphates and carbamates exert their effects by competitively inhibiting acetylcholinesterase activity throughout central and peripheral receptors. Cats are particularly susceptible to the acute and delayed toxic effects of organophosphates and, to a lesser extent, carbamates. Nevertheless, most poisoning has been associated with carbamate compounds. Toxicity is generally the result of accidental exposure, the inadvertent use of products intended for dogs, or an overzealous regimen of control for external and internal parasites. Acetylcholine acts as a neurotransmitter at parasympathetic muscarinic receptors, sympathetic nicotinic receptors, and at the neuromuscular junction (nicotinic receptors). Organophosphates are considered to be irreversible inhibitors of acetylcholinesterase whereas carbamates are slowly reversible inhibitors. Diagnosis is based on the history of exposure, clinical findings, and decreased serum acetylcholinesterase activity.

Accumulation of excess acetylcholine results in a variety of clinical signs associated with overstimulation of receptors. Skeletal muscle signs are associated with nicotinic receptors and are partly stimulatory and partly inhibitory, Signs include muscle weakness, muscle tremors, fasciculations, neck

ventroflexion and stiff gait. Muscarinic signs (salivation, lacrimation, dyspnoea, diarrhoea, vomiting, miosis), and central cholinergic signs (seizures, anxiety, ataxia, hyperactivity and coma), are usually seen within minutes to hours of exposure, but nicotinic signs may occasionally appear in isolation in cats treated with the product for several weeks. Delayed neuropathy resulting from distal axonal degeneration has been reported after prolonged exposure to organophosphate compounds but not carbamates. Clinical signs of a predominantly lower motor neuron paraparesis typically occur after a delay of one to four weeks, sometimes after even minimal exposure. In severe cases, the forelimbs are also involved. Signs may mimic those of myasthenia gravis but in contrast to myasthenia gravis the signs worsen after edrophonium chloride administration.

Mildly affected cats recover with symptomatic therapy. In known cases of acute organophosphate intoxication, atropine and the cholinesterase reactivator, pralidoxime (diluted to 20 mg/ml and given at a dose of 20–50 mg/kg slowly IV over 2 minutes at least), should be administered. Pralidoxime may alleviate nicotinic signs but is only effective if given within 24 hours of exposure and is usually only required for 24–36 hours. It is contraindicated for carbamate intoxication. Atropine is effective at a dosage of 0.2 mg/kg for relief of muscarinic signs and centrally mediated cholinergic signs of organophosphate toxicity but has no effect on nicotinic sympathetic signs. Muscle tremors can be alleviated using diphenhydramine (2–4 mg/kg orally every 8 hours) and seizures can usually be controlled with IV or IM midazolam (0.5 mg/kg).

Snake Bite and Tick Paralysis

(Dickinson & Lecouteur 2004, Malik & Farrow 1991, Schull et al. 2007).

Clinical signs of weakness and paralysis result from the effects of neurotoxic components of certain snake venoms (of the family Elapidae) and tick saliva on the neuromuscular junction. There is also a significant elevation of CK following snake envenomation.

The snakes most commonly incriminated are the Texas coral snake, Eastern coral snake and Sonoran coral snake in the United States and the brown snake and the tiger snake in Australia.

The ticks most commonly involved are Dermacentor variabilis and Dermacentor andersoni and Ixodes holocyclus in Australia. There may be a history of a venomous snake bite or exposure to ticks.

Appendix

Laboratories specializing in the investigation of neuromuscular diseases:

1. Prof. Dr. Kaspar Matiasek, Neuropathology Laboratory, Veterinarstr. 13, D-80539 Munich, Germany. Email: info@neuropathologie.de
2. Dr GD Shelton, Comparative Neuromuscular Laboratory, 9500 Gilman Drive, Biomedical Sciences Building, Room 2095, University of California, San Diego, La Jolla, CA 92093-0709, USA. Tel: (858) 534 1537 Fax: (858) 534 0391 Email: musclelab@ucsd.edu
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18 Miscellaneous Orthopaedic Disorders

METABOLIC BONE DISEASES AND DISORDERS OF CALCIUM METABOLISM:

(Bright 2010).

Calcium is vital for animal life and is present in various sites in the body, including body fluids and structural parts of the cell. However, 99% is present as a major component of the skeleton. Cats in the wild state obtain calcium and phosphorus by eating the whole body of their prey. Whole rats and mice contain approximately 2% calcium as a proportion of dry matter. Growing kittens have lower absolute requirements for calcium and phosphorus than puppies and can also tolerate a calcium:phosphorus ratio as low as 0.65:1 with no apparent ill effects. The calcium requirement for kittens is 0.5–2.0 g/400 kcal, which is more than twice that of the adult cat.

Bone metabolism and mineral homoeostasis are regulated primarily by the calciotropic hormones (parathyroid hormone [PTH], calcitonin, and vitamin D or calcitrol) under the influence of dietary calcium and phosphorus. A number of other hormones and dietary constituents have a lesser effect on bone growth and remodelling. Hormones either target bone cells specifically or have a more generalized influence on cell function in multiple tissue types. A metabolic bone disease occurs when there is a disruption in the natural turnover of bone, with an imbalance between osteoid production, mineralization, and bone resorption. This may have significant consequences for skeletal integrity, by increasing the risk of fracture as a result of minor trauma and reducing the rate of bone healing. Bone pain in the absence of fractures is a frequent feature of metabolic bone disease in humans and there is no reason to suppose that this does not occur in cats as well, manifested clinically as lameness, lethargy or unwillingness to exercise. Developmental orthopaedic disease, such as osteochondrosis, commonly seen in young, growing, large-breed dogs, has been occasionally reported but it is not recognized as a common clinical problem in cats.

In large-breed dogs these conditions may be associated with hypercalcitonism as a result of excessive consumption of calcium in the diet. It is likely that the skeleton is protected from these disorders because of the cat's smaller stature and slower growth rate, as with small breeds of dog. When orthopaedic disease does occur during development it is usually the result of a metabolic disorder, in which the cat has been fed a wholly inappropriate diet, or it is related to a genetic disorder, such as osteochondrodysplasia.

PRIMARY HYPERPARATHYROIDISM:

(Blunden et al. 1986, Bolliger et al. 2002, Feldman et al. 2005, Kallett et al. 1991, Savary et al. 2000, Singh et al. 2019).

Primary hyperparathyroidism (PHPT) is a rare endocrine disorder in middle-aged to older cats, with no reported breed predisposition, typically due to a parathyroid adenoma.

Like in the dog, this condition is due to the overproduction of PTH by the hyperplastic or neoplastic parathryroid gland or glands, producing hypercalcemia. Probable primary hyperparathyroidism has been described in a cat with polyostotic lesions. Lesions involve particularly the axial skeleton, including the cranial vault, mandible, and sternebrae. The appendicular skeleton was involved to a lesser extent. Cervical and thoracic intervertebral and diarthrodial joints had collapsed. Bone lesions appear to be uncommon, however, and have not been described in other cats with primary hyperparathyroidism.

A final diagnosis of PHPT requires simultaneous measurement of PTH ionised calcium (iCa) levels, although the interpretation of these tests can sometimes be challenging and a consultation with an internal medicine colleague may be advisable. A diagnosis of parathyroid hyperplasia or carcinoma is less likely and the measurement of parathyroid hormone-related peptide (PTHrP) may be necessary to rule out malignant hypercalcemia.

Ultrasonography of the thyroid and parathyroid glands is recommended as a non-invasive, easy to perform test to investigate the likely source of the hypercalcemia.

RENAL SECONDARY HYPERPARATHYROIDISM:

Cause and Pathogenesis

(Barber & Elliot 1998, Lucke 1968, Sherrard et al. 1993, Slatopolsky et al. 1980).

Renal secondary hyperparathyroidism (RSHP) is a common complication of chronic renal failure. In one study, 84% of all cats with chronic renal failure had elevated plasma PTH concentrations on first presentation. Hyperphosphatemia occurs as a consequence of reduced renal phosphorus excretion in cats with renal dysfunction. Reciprocal hypocalcemia, coupled with impaired renal vitamin D metabolism associated with reduced renal mass, leads to secondary hyperparathyroidism. Additionally, in chronic cases, there is a tendency for the parathyroid glands to function autonomously in spite of restoration of normal blood calcium (tertiary hyperparathyroidism).

Hyperphosphatemia predisposes to metastatic calcification of soft tissues, including the kidneys, thus further compromising renal function and completing a vicious circle. Persistent elevation of PTH has widespread adverse effects on glucose and lipid metabolism, neurologic function, and the immune system. This has led to the suggestion that PTH should be regarded as a uraemic toxin. In addition, PTH has well-established adverse effects on the skeleton. In humans, this includes painful and debilitating bone disease. In one study histologic evidence of osteopenia was found in the majority of cats with advanced renal failure examined at post-mortem.

Clinical Signs

(Gnudi et al. 2001, Jackson & Barber 1998).

Clinical signs relate primarily to the underlying renal disease. Rubber jaw, seen classically in the dog with juvenile nephropathy, has not been reported. Lesions described in a 5-month-old cat with glomerulonephritis included marked osteolysis in the mandible and calvarium with more severe changes in the appendicular skeleton. Histologically there was a massive substitution of bone by fibroconnective tissue. Clinical signs of bone disease in cats with renal disease usually represent a more generalized and subtle osteopenia and there is no predilection for the skull. Bone pain on palpation can be elicited in some cats with chronic renal failure and there may be more covert pathology, such as an increased risk of fracture and delayed union. Cutaneous metastatic calcification is an unusual manifestation of renal hyperparathyroidism that has been reported in a 10-year-

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old cat. The cat presented with multiple nodular calcifications of the footpads and interdigital spaces. Recognition of these secondary phenomena is likely to increase as the life expectancy of cats continues to rise.

Diagnosis

(Edelstyn et al. 1967, Lachman 1955, Lamb 1990).

The majority of cats with chronic renal failure can be assumed to have RSHP and a degree of osteopenia. A case of suspected RSHP has been reported in a cat with chronic renal insufficiency and radiographic evidence of spinal and femoral osteopenia. Osteopenia may not be readily apparent radiographically, because loss of 30–50% of bone mass must occur before it is detectable as decreased bone opacity. Clinically significant skeletal effects are most likely to be seen in those cases with long-standing and severe renal disease. All cats with evidence of bone pain, delayed healing, or pathologic fracture should be screened for renal disease by performing urea, creatinine and blood phosphate assays. Elevation of plasma phosphate and PTH are significantly correlated, whereas measurement of ionized calcium is unhelpful since significant hypocalcemia is only seen in cats with end-stage renal failure.

Treatment and Prognosis

(Barber et al. 1999, Nagode et al. 1996).

Treatment is palliative and the prognosis is determined by the severity of the renal disease. A commercially prepared phosphate and protein-restricted diet should be fed to all cats with chronic renal failure and is an effective means of normalizing plasma PTH in cats with RSH. An intestinal phosphate binder can also be used if the renal diet by itself is not enough after 2–3 months. The use of calcitriol in the treatment of this condition is still controversial due to variable clinical efficacy.



Figure 18.1 A case of nutritional secondary hyperparathyroidism in a Domestic Short Hair kitten, showing antebrachial bone deformities. (Courtesy of R. Quinn).

NUTRITIONAL SECONDARY HYPERPARATHYROIDISM:

(National Research Council Ad Hoc Committee on Dog and Cat Nutrition 2006).

Nutritional secondary hyperparathyroidism (NSHP) is also referred to as juvenile osteoporosis, nutritional osteoporosis, or all-meat syndrome. It is typically due to the consumption of a diet that is relatively or absolutely deficient in calcium. Historically this disease was reported occasionally in kittens before the widespread feeding of manufactured pet foods, now adequately formulated and well-balanced for minerals. It is possible that there may be a resurgence of the condition with the modern trend towards feeding organic, natural, or vegetarian foods to pets. Adult cats have lower absolute demands for calcium and greater reserves of calcium than kittens, so they are rarely affected. It is recommended that growing kittens eat a diet with a Ca:P ratio of approximately 1:1.

Cause and Pathogenesis

(Tomsa et al. 1999).

Traditionally the disease is caused by feeding a diet composed primarily of meat and offal with no bones; this diet is relatively high in phosphorus and low in calcium. In the only contemporary case series there were six cats. Three of the cats were fed an all-meat diet, one cat was fed a vegetarian diet, and two cats were fed a meat and rice diet. The common feature of all the diets was a severe deficiency of calcium and a moderate deficiency of phosphorus, resulting in an unfavourable calcium to phosphorus ratio (0.061:1–0.12:1). All the cats were less than 7 months old and were not allowed outdoors, which prevented them from supplementing their diet by hunting. Transient hypocalcemia, owing to low calcium intake, stimulates the release of PTH from the parathyroid glands and ionized calcium levels are restored. If calcium deficiency is sufficiently severe, ionized blood calcium may remain low. Formation of bone occurs normally but is outstripped by accelerated bone resorption under the influence of PTH. Increased PTH also promotes renal reabsorption of calcium and excretion of phosphorus and renal synthesis of active vitamin D (calcitrol). Calcitrol, in concert with PTH, stimulates further bone resorption.

Clinical Signs

Affected cats show lameness and pain, which may be severe. Bony deformity occurs due to pathologic fractures and joint deformity is seen due to ligament and tendon laxity (Figure 18.1). The hindlimbs tend to be more severely affected than the forelimbs and paresis or paraplegia may result from spinal cord compression following vertebral fracture. Four of the six cats in the aforementioned series also showed neurologic signs, including seizures, tetanic spasms, and muscle tremors and fasciculations consistent with hypocalcemia.

Diagnosis

(Craiger & Nachreiner 1993, Dimopoulou et al. 2010, Parker et al. 2015).

Diagnostic imaging of the skeleton shows a generalized loss of radiopacity and thinning of the cortices of the long bones with sparing of the physes (Figures 18.2 to 18.4). The

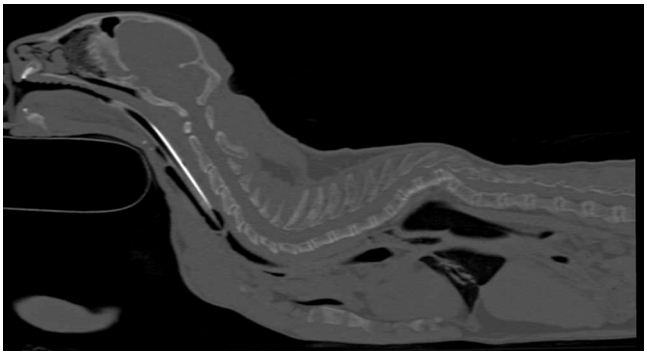


Figure 18.2 A sagittal tomographic view of the spine of this patient. Observe the generalized osteopenia and the folding, pathological fracture in the thoracolumbar area. (Courtesy of R. Quinn)

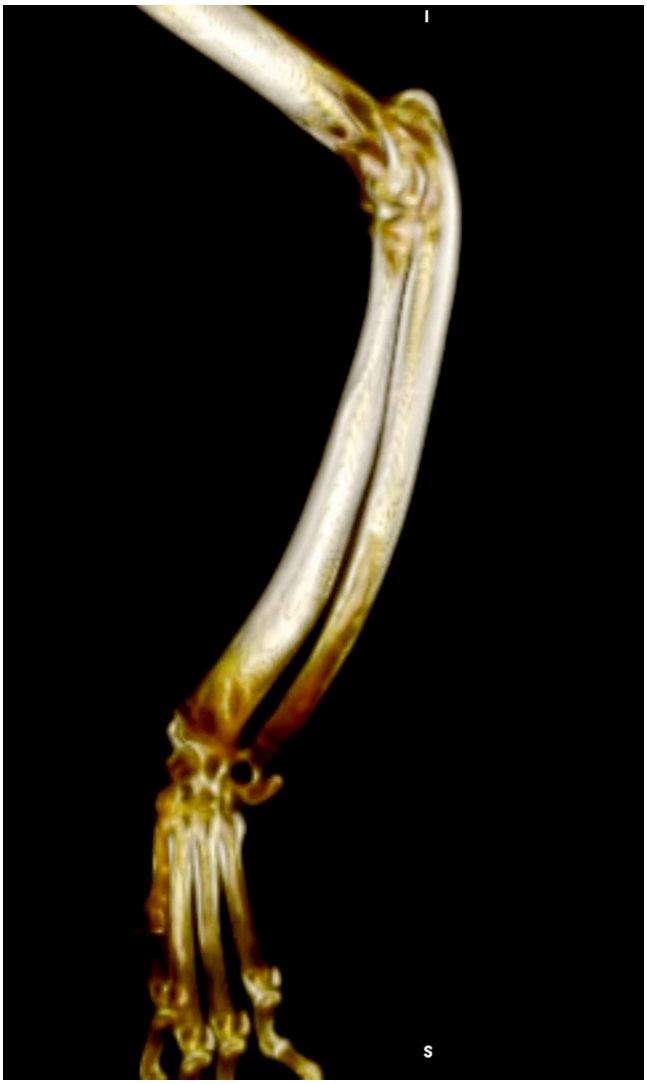


Figure 18.3 Lateral view of a 3D reconstruction of the left antebrachium, showing the bony deformities associated with this condition. (Courtesy of R. Quinn)

metaphyses appear broader than normal and there may be regions of relative radiopacity in the metaphyses adjacent to the growth plates where there is preferential mineralization. In severely affected cases there will be folding fractures of the long bones and compression fractures of the vertebrae. Bony protuberances, such as the olecranon and the calcaneus, become curved as a result of weight-bearing forces. Blood testing is not normally necessary for diagnosis but, if performed, may show normal to reduced ionized calcium, increased PTH, normal to increased phosphorus and normal to increased calcitrol. These biochemical changes are more marked in severely affected cases but once the cat is fed a balanced diet they are all reversed rapidly. Differential diagnoses for this condition include renal secondary hyperparathyroidism (RSHP), osteogenesis imperfecta and rickets.

Treatment and Prognosis

Treatment is by provision of a commercially prepared, balanced diet formulated for young cats. Cage rest is vital in the early stages to prevent further injury. Calcium gluconate should be given intravenously only to cats with clinical signs of hypocalcemia, with the appropriate precautions. There is a high risk of vertebral fracture in cats presenting with tetanic muscle contractions. Symptomatic

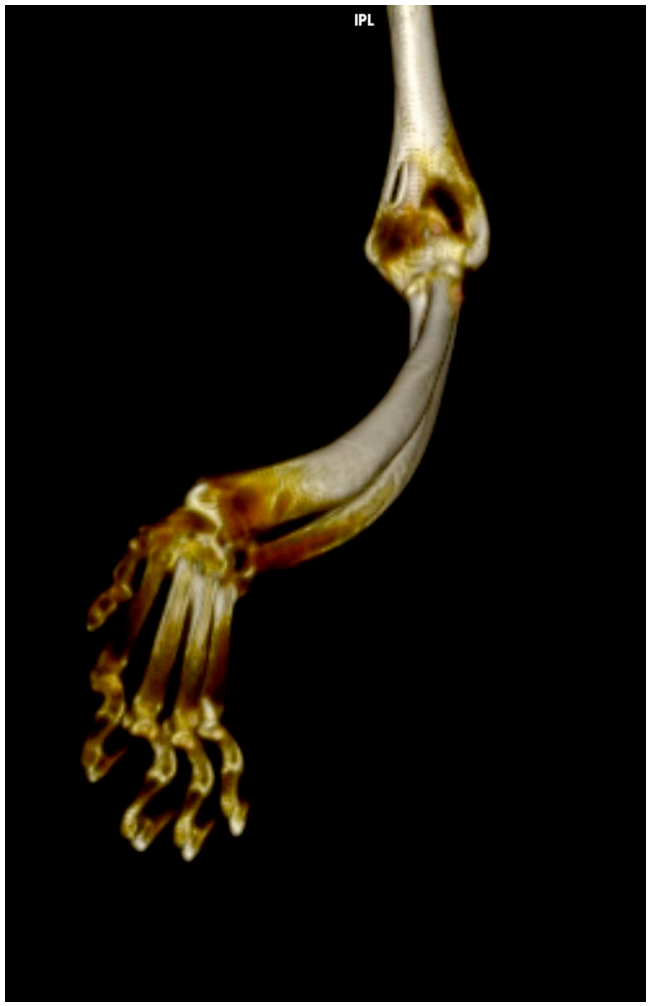


Figure 18.4 Craniocaudal 3D reconstruction of the same antebrachium. (Courtesy of R. Quinn)

therapy with nonsteroidal anti-inflammatory drugs (NSAIDs) or pure analgesics may be necessary for a few days to control pain.

Conventional fracture treatment is not possible because of the fragility of the bones and poor bone quality for the placement of implants. Early surgical intervention is inadvisable and coaptation with bandages, splints, or casts is contraindicated. Surgical correction of skeletal deformities should be postponed until the skeleton has become adequately mineralized. Medical treatment with glucocorticoids or calcitriol is also contraindicated. The response to treatment is rapid and follow-up radiographs obtained after 3 weeks on a balanced diet should show adequate mineralization of the skeleton. Osteogenesis imperfecta should be suspected if skeletal mineralization has not occurred within 3 weeks. The prognosis is generally good, although there is likely to be residual deformity and growth retardation. The prognosis for cats with vertebral fracture and severe neurologic dysfunction is poor.

HYPERTHYROIDISM AND HYPERPARATHYROIDISM:

(Williams 2012).

Approximately 57–77% of cats with hyperthyroidism also show hyperparathyroidism, although in these cases, ionised calcium concentration is usually normal or slightly decreased. Hyperphosphatemia and azotemia have been more frequently observed. It is hypothesized that this may be due to the effects of thyroid hormone on bone turnover and renal excretion, causing a compensatory increase in PTH secretion.

HYPERVITAMINOSIS A:

(Freytag et al. 2003, Guerra et al. 2014, Polizopoulou 2005).

This uncommon condition is caused by ingestion of a diet based primarily of liver. It produces abnormal bony growth along the spine and can also affect several joints, although the cervical spine and the elbow joints are most commonly involved (Figure 18.5). Also, a rare, chronic liver toxicity manifested as hepatic fibrosis has been described in cats, similar to the human form. Chronic administration of a diet with excessive vitamin A in queens can induce teratogenic effects in kittens, if continued through pregnancy, such as cleft palate, craniofacial and pelvic abnormalities, shallow ophthalmic orbits, and vascular abnormalities.

The predominant clinical sign in affected animals is lameness due to perarticular bone growth formation and encroachment of nerve roots along the spine. Also, signs related to overall discomfort, such as irritability, lack of grooming due a stiff spine and joints, malaise etc, can be found. Although removal of the liver in the diet will cease progression and clinical improvement is possible, substantial regression of the bony growth is very unlikely.

Cause and Pathogenesis

(Clark 1971, Corbee et al. 2014, Franch et al. 2000, Polizopoulou 2005, Seawright & English 1964).

Hypervitaminosis A is a metabolic disorder caused by a chronic excessive intake of vitamin A (retinol). Vitamin A stimulates osteoblastic activity and plays a role in skeletal growth. The condition has become rare since the widespread feeding of commercially prepared pet foods. Disease usually results from the consumption of a diet consisting principally of raw liver, which is also very rich in vitamin D. Vitamin A is thermolabile and is largely destroyed by cooking. Excess vitamin A cannot be excreted and accumulates in body fat. Kittens are rarely affected; most cases are the result of accumulation and occur between 2 and 9 years of age. Affected cats have usually been consuming liver regularly for at least 12 months at the time of presentation.



Figure 18.5 Anatomical specimen of a cat affected by hypervitaminosis A. Observe the extensive periarticular bone proliferation (courtesy of J. Font)

Vitamin A promotes the differentiation of mesenchymal stem cells into osteoblasts and osteoclasts and the maturation and mineralization of cartilage, which are further potentiated by vitamin D. The clinical form of the chronic, combined exposure of these two compounds in adult cats, mainly characterized by new bone formation, indicates a shift towards increased osteoblast activity and bone formation.

Microfractures occur at the site of attachment of ligaments, tendons, and the joint capsule of diarthrodial joints, leading to dystrophic calcification of the axial and appendicular skeleton. The chronic form of the disease was originally described as 'feline deforming cervical spondylosis' because extensive confluent exostoses of the cervicothoracic vertebrae are the most frequent pathologic finding. The osseous lesions are always accompanied by lipid infiltration of the liver, which may predispose to secondary disease.

Clinical Signs

(Clark et al. 1970, Kallfelz & Dzanis 1989, Seawright et al. 1967).

Affected kittens show reduced longitudinal growth of the appendicular skeleton, with flaring of the metaphyses and

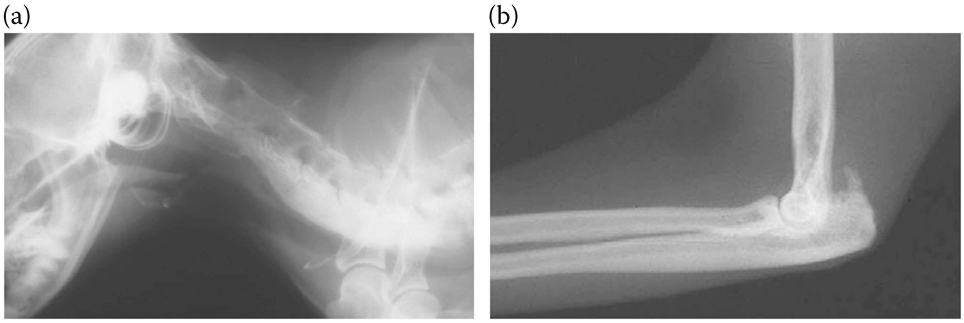


Figure 18.6 Hypervitaminosis A. (a) Lateral view of the cervical spine of a cat fed an all-liver diet. There are extensive confluent exostoses of the cervicothoracic spine. (Radiograph courtesy of Davies Veterinary Specialists.) (b) Lateral view of the elbow joint of a cat fed an all-liver diet. (Radiograph courtesy of Malcolm McKee.)

osteopenia. The clinical signs in the more common chronic cases relate mainly to ankylosis of the cervicothoracic vertebrae. In one of the few recent reports of the disease, however, there was ankylosis of the stifles and hips with no spinal involvement. Signs include anorexia, weight loss, depression, hyperaesthesia, constipation, joint pain and lameness, and an unkempt hair coat as a result of decreased grooming. In advanced cases, the neck becomes rigid due to ankylosis of the cervical vertebrae and there is forelimb lameness and pain owing to nerve root compression. Affected kittens are stunted and have limb deformity.

Diagnosis

Radiography shows exostoses affecting the cervical vertebrae, the sternebrae, and the periarticular regions of the long bones, particularly the distal humerus and proximal ulna (Figure 18.6). The ribs may also be affected in advanced cases. Plasma concentrations of vitamin A are increased to >16.67 µmol/l (500 µg/dl) in 80% of clinical cases, but measurement of this is not essential for diagnosis.

Treatment and Prognosis

Treatment is by removal of the source of vitamin A and D from the diet and substitution in the short term with a home-made low vitamin A diet. This should contain lean meat and a source of carbohydrate but exclude organ meat (heart, liver, and kidneys), eggs, and milk. It may be difficult to persuade some cats to eat anything else apart from the liver. In the long term a commercially prepared balanced diet can be fed. The pathology is irreversible, although clinical improvement is usually seen within 4 weeks. Limited remodelling of the osseous changes occurs with time following dietary correction. Glucocorticoids are contraindicated and will slow the reduction in plasma vitamin A concentration. Symptomatic therapy with NSAIDs may be used to control pain. The overall prevalence of this condition seems to have decreased in the past years due to the increased availability of more balanced, commercial diets.

RICKETS:

(Geisen et al. 2009, Godfrey et al. 2005, Henik et al. 1999, Langley-Hobbs 2012, MacKenzie et al. 2011, Phillips et al. 2011).

Nutritional rickets or hypovitaminosis D is an extremely rare metabolic bone disease of the young growing kitten producing defective bone mineralization. The term is sometimes erroneously used as a synonym for NSH. The disease is now so rare that there are very few recent clinical descriptions in naturally affected cats.

Cause and Pathogenesis

Rickets is a disorder of growing bones that occurs when normal mineralization is prevented by lack of calcitrol (1, 25-dihydroxycholecalciferol) activity. Cats, in common with dogs, do not synthesize vitamin D in their skin when irradiated with ultraviolet B light. Vitamin D (cholecalciferol) is obtained from the diet and is then metabolized in the liver before being converted to the active form (calcitrol) in the proximal kidney tubules. The primary feature of vitamin D deficiency rickets is a reduction in intestinal calcium absorption due to inadequate calcitriol concentrations in the face of an otherwise normal dietary calcium intake. Lack of vitamin D may also be associated with low dietary intake or secondary to gastrointestinal malabsorptive syndromes.

Hereditary, nonnutritional types of vitamin D-dependent rickets (also known as pseudo- vitamin D-deficiency rickets) also occur and four types have been recognized in cats:

1. Vitamin D-dependent rickets type 1 (VDDR-1), caused by a defect in the gene encoding the enzyme 25-hydroxyvitamin D-1α-hydroxylase.
2. Vitamin D-dependent rickets type 2 (VDDR-2), is caused by a defect in the vitamin D receptor gene.
3. Hypophosphataemic rickets, in which the defect is due to loss of phosphate at the level of the renal tubule.
4. Vitamin D-dependent rickets non-type 1, non-type 2. The cause has not been yet determined.

Hypovitaminosis D impairs gastrointestinal absorption of calcium leading to secondary hyperparathyroidism. In growing kittens, there is defective mineralization of the newly formed organic matrix of the skeleton. Osteomalacia is the adult equivalent of hypovitaminosis D and primarily affects bone remodelling, so that unmineralized matrix accumulates in all parts of the skeleton.

Clinical Signs

The clinical signs are similar to those of NSH and often include stiffness, reluctance to move and pain or signs that are consistent with hypocalcaemia, such as tremors, seizures or gastrointestinal disturbances, with the additional findings of bony swellings proximal to the carpi and tarsi, lateral bowing of the antebrachia, and muscle weakness.

Diagnosis

Radiographs show generalized loss of radiopacity and skeletal deformity. In kittens, additional changes include grossly widened, radiolucent physes and characteristic mushrooming or cupping of the enlarged metaphyses. The diagnosis of nutritional rickets can be confirmed by the demonstration of reduced circulating levels of the storage form of vitamin D (25-hydroxycholecalciferol) in the blood.

Treatment and Prognosis

Treatment of nutritional rickets is by substitution of a commercially prepared feline diet and cage rest in the early stages. Supplementation with vitamin D analogues is indicated but the levels of supplementation recommendable for a specific case is difficult to formulate since it depends on the type of rickets and its severity. Adjusting dosing may need close monitoring of blood levels and serial radiographs to assess the response to treatment. Typically, the effective dosage levels for supplements are often considerably higher than those recommended to be given in normal animals.

The prognosis is variable, depending on the type of rickets and its response to therapy, although there may be residual skeletal deformity. Improvement is rapid and follow-up radiographs taken after 3 weeks should show nearly normal skeletal mineralization. Failure to improve should prompt consideration of underlying renal pathology, an inborn error of vitamin D absorption or metabolism due to a genetic abnormality, or target organ resistance.

OSTEOPETROSIS:

(Fujita et al. 2007, Hanel et al. 2004, Hoover & Kociba 1974, Kramers et al. 1988, Van Hul et al. 2001, Wright et al. 2003).

Osteopetrosis, also known as osteosclerosis or "marble bones", is a type of sclerosing bone dysplasia, a term used to describe a rare group of genetic diseases in humans characterized by abnormal hardening of bone due to defective osteoclastic resorption of immature bone. This group of conditions are a type of inherited, heterogeneous osteochondrodysplasia.

A similar syndrome has been described in adult cats in which there is generalized increase in bone density, without alteration in bone shape. In the cat, the etiopathogenesis for the disorder is poorly understood but appears to be acquired and is probably more correctly known as generalized or diffuse osteosclerosis.

Radiographically there is a diffuse increase in medullary opacity with a marbled, densely homogenous appearance to the long bones and vertebrae. There is marked endosteal thickening of the diaphyses of long bones and dense subchondral bone with loss of the normal trabecular architecture. Usually the osseous abnormalities are detected incidentally in cats radiographed for other reasons. Similar bone lesions have been described in neonate cats, where they were induced experimentally by inoculation of FeLV. The lesions probably developed because of infection of hemopoietic precursor cells, from which osteoclasts arise. Osteosclerosis may cause non-regenerative anaemia if the medullary canal is totally compromised and in one cat there was inspiratory stertor and epiphora owing to obliteration of the nasal turbinates and nasolacrimal duct.

In adult cats the etiology and significance of the osseous lesions has not been established but most cases have been associated with concurrent disease, including lymphoblastic leukaemia, systemic lupus erythematosus, lymphoma, C-cell tumour, myeloproliferative disorders, chronic renal failure, and fibrosarcoma. The prognosis is variable and will depend on the nature and severity of the primary condition.

OSTEOGENESIS IMPERFECTA:

(Drogemuller et al. 2009, Evason et al. 2007, Gehring 1975, Haase et al. 2016, Hubler et al. 2009, Parker et al. 2015, Rauch & Glorieux 2004, Schutz et al. 2013, Won et al. 2017).

Osteogenesis imperfecta, also known as brittle bone disease, is a very rare inherited bone disease in the cat. It has been better studied in the canine than in the feline species, and determined to be a condition most often caused by mutations in either of two genes that code for Type 1 collagen (COL1A1 and COL1A2).

There are several types described in humans, with different biochemical defects and inheritance patterns. It is generally characterized by bone fragility, in which abnormal bone matrix and secondary osteopenia are associated with an increased susceptibility to fracture, caused by a defect in production of collagen type I, which constitutes 85% of the collagen present in bone.

The main significance of the condition is as a differential diagnosis for NSHP. Clinically, affected cats may show some lameness and pain in the pelvic limbs, avoiding heavy jumping and showing overall painful mobility or may suffer fractures with minimal or no apparent trauma. Deformities of the thorax or spine can also be seen, seriously affecting the patient. Radiographically there is generalized osteopenia with thin diaphyseal cortices and multiple fractures in various stages of union. Measurement of bone mineral density, a commonly done test in human medicine, is rarely available in veterinary medicine but its use has been reported in a suspected feline case.

Fractured bone forms callus and heals normally, often resulting in malunion. A typical presentation would be a kitten fed on a balanced diet with multiple fractures, apparently occurring spontaneously or as a result of minimal trauma. Their skin may be thin and soft tissues prone to herniation. Abnormal dentine development, characterized by severe thinning of the dentine layer, translucency and pink discoloration, may also be observed (dentinogenesis imperfecta). Histologically, bone mass and cortical density is reduced, with increased porosity. More common causes of osteopenia, such as NSHP and RSHP, should be ruled out first.

These animals should have normal blood calcium, phosphorus, vitamin D and PTH concentrations. In addition to genomic analysis of the affected individuals, definitive diagnosis can be achieved by laboratory culture of fibroblasts obtained from a skin biopsy, looking for collagen Type I abnormalities, although this is not yet a readily available test.

There is no proven treatment although long bones improve in their shape and density with puberty and increased age. In a case report, a kitten was treated with vitamin C but the outcome is unknown since it was euthanased. Bisphosphonates have also used with some efficacy in the treatment of children but their efficacy has not been proven in cats.

METAPHYSEAL OSTEOPATHY:

(Adagra et al. 2015, Pantaleo et al. 2016).

Metaphyseal osteopathy has recently been described in cats, sharing the typical features of the canine disease, showing the pathognomonic radiographic changes consisting of irregular radiolucent lines running parallel to the physis seen in the metaphyses of the long bones, typically affecting the distal radius and ulna. Macroscopic evaluation of bone with metaphyseal osteopathy reveals a transverse band of disrupted trabeculae situated immediately proximal to the growth plate and corresponding to the lucent zone observed radiographically. This band of diseased bone corresponds with an area of trabecular disruption and consists of haemorrhage and necrosis with an infiltrate of inflammatory cells. Osteoclastic resorption of the adjacent metaphyseal trabeculae is focally extensive, and there may be subperiosteal haemorrhage or reactive new bone formation. There is also irregular widening of the growth plate because of increased length of the columns of hypertrophied chondrocytes.

The aetiology of canine metaphyseal osteopathy remains obscure but numerous causes have been proposed including vitamin C deficiency, overnutrition, infection, hereditary factors and abnormal immunological response to vaccination. In the reported feline cases, there was a suspected infectious etiology, although this was not confirmed. Treatment in these few reported cases consisted of medical treatment until complete resolution of clinical signs.

HYPERTROPHIC OSTEOPATHY:

(Becker et al. 1999, Brandes & Heblinski 2013, Carr 1971, Cetinkaya et al. 2011, DeMelo Ocarino et al. 2006, Gram et al. 1990, Grierson et al. 2003, Langley-Hobbs 2012, Mills 2010, Nafe et al. 1981, Richards 1977, Roberg 1977, Rohr 2003, Salgüero et al. 2015).

Hypertrophic osteopathy is also known as pulmonary osteopathy, hypertrophic pulmonary osteoarthropathy or Marie's disease.

Cause and pathogenesis

Hypertrophic osteopathy is an extremely rare bone disease characterized by periosteal new bone formation along the limb bones, as a secondary manifestation of a primary disease process, either in the thoracic or, less commonly, the abdominal cavity. The inciting primary process is typically a space-occupying lesion, commonly neoplastic but it could also be an inflammatory process.

The pathogenesis is not fully understood, but vascular congestion of the distal extremities secondary to the underlying disease process is thought to play a major role in the new bone formation in dogs and humans. Neurogenic and humorally mediated mechanisms have been proposed as causes of the circulatory disturbance.

Most of the reported cases in cats have been associated with neoplasia, although in some cases, the cause was an inflammatory condition or it was not identified.

The primary condition included, amongst others; bronchiolar carcinoma, pulmonary sarcoma, thymoma, renal papillary adenoma, and adrenocortical carcinoma. Hypertrophic osteopathy has also been described in association with pneumonia caused by a migrating grass awn and in association with an injection-site sarcoma, in the absence of intrathoracic or intrabdominal disease.

Clinical signs

Clinically there is bilaterally symmetrical soft tissue swelling affecting the distal portions of the limbs (especially in the metacarpus/metatarsus) and this may progress proximally to affect all of the bones of the limb. Clinical signs related to the painful and swollen distal extremities often precede those arising from the primary lesion, such as coughing or dyspnea. Affected cats are usually depressed and reluctant to move around.

Diagnosis

Radiographs of the distal limbs show an irregularly marginated periosteal reaction, typically described as lamellated or pallisading. Radiographic and/or ultrasonographic examination or computed tomography of the thorax and abdomen may be required to reveal the primary lesion.

Treatment and prognosis

The prognosis is determined by how treatable the primary lesion is but it is generally poor, since this lesion is often neoplastic. Partial regression and remodelling of the bony changes may occur a few weeks following successful removal of the primary lesion. If surgery is not possible, pain control or any other medical treatment to improve quality of life is indicated, until euthanasia is necessary.

SPONTANEOUS FEMORAL CAPITAL PHYSEAL FRACTURES:

(Barr et al. 2010, Bennett 1994, Borak et al. 2017, Craig 2001, Forrest et al. 1999, Lafuente 2011, McNicholas et al. 2002, Perry 2014, Perez-Aparicio 1993).

Separation (Salter-Harris type I) of the femoral capital physis from the femoral neck in skeletally immature cats is commonly seen as a result of trauma (see Chapter 10), typically younger than 6 months of age. Perhaps less commonly, but increasingly recognized as a unique presentation, separation of the capital physis occurs in heavier, neutered male cats over 12 months of age in the absence of trauma. Lameness may have a variable duration of onset associated with progressive femoral neck osteolysis and pathologic fracture.

Cause and Pathogenesis

Metaphyseal bone necrosis has been produced experimentally in cats by the intravenous inoculation of feline herpes virus. However, no inclusion bodies typical of herpes virus have been found in cells of material removed at the time of surgery. Still, the inciting cause for this condition is unknown, although hormonal, infectious and genetic factors have been postulated.

The high incidence in neutered male cats of Siamese and Maine Coon cats, between 12 and 24 months of age suggests an association with delayed physeal closure seen after neutering. Other breeds, such as domestic short hair, Birman, British Blue etc, have also been reported.

The condition of physeal dysplasia has been described to explain these non-traumatic separations of the femoral capital epiphysis, drawing similarities with the same condition in humans, pigs and dogs. It seems likely that slipped femoral capital epiphysis, spontaneous femoral capital physeal fracture, femoral capital physeal dysplasia syndrome

and proximal femoral metaphyseal osteopathy merely represent different manifestations and etiopathologic interpretations of the same condition. It is important to recognize that these conditions do not feature bone loss in the femoral head, as in Legg-Calve-Perthes in the dog, and the epiphysis is not affected. However, the blood supply coming through the round ligament is substantial in the cat and this may preclude the femoral head from developing avascular changes.

Clinical Signs

Typically, affected cats have prodromal hindlimb lameness, which suddenly worsens when pathologic fracture occurs. Pain can be elicited on manipulation of the hip joint although this may be mild until fracture occurs. Over half the cases eventually show bilateral disease in which the main clinical sign is stiffness and reluctance to jump rather than an obvious limp. Less commonly, the contralateral hip can become affected at a later date.

Diagnosis

Radiographs of the pelvis show loss of bone within the femoral neck creating a narrowing effect and varying degrees of sclerosis, particularly in more chronic cases. The fracture may be apparent, although there is usually minimal displacement at the fracture site (Figure 18.7). Therefore, this fracture may not be visible on the standard extended ventrodorsal view due to a position-induced fracture reduction and a frog-leg position may also need to be obtained.



Figure 18.7 Spontaneous femoral capital physeal fracture. This ventrodorsal extended pelvic radiograph shows a bilateral, chronic fracture, with femoral neck necrosis, remodelling and sclerosis of the metaphyseal bone distal to the fracture.

Histologically, the affected femoral head may show a broad zone of hypertrophic, irregularly arranged, sometimes necrotic, cartilage at the site of the capital femoral physis. Chondrocytes are separated by an abundant matrix and arranged in clusters. The physis may be thickened and necrotic bone may be visible at the dorsal femoral neck region, with proliferating fibroblasts, granulation tissue and an inflammatory infiltrate and fibrin exudation. In traumatic physeal fractures, the fracture line can extend through multiple zones of cartilage but chondrocytes retain their columnar arrangement on both sides of the fracture.

Treatment and Prognosis

(Fischer et al. 2004, Kalis et al. 2012, Perry 2014).

Conservative management is usually not successful for the treatment of these fractures, likely due to ongoing fracture instability and the presence of necrotic tissue, osteolysis and local bone remodelling. Internal fixation of the capital femoral physis is usually precluded by the degree of femoral neck resorption, although it may be a reasonable option in mild, early cases with little bone loss. Total hip replacement is the preferred treatment option although femoral head and neck ostectomy will produce satisfactory results, especially when coupled with a physiotherapy programme. Femoral head and neck ostectomy may be performed on both hips simultaneously in bilaterally affected cats and, in these cases, professional physiotherapy is strongly recommended for best results.

OSTEOCHONDRODYSPLASIA:

(Gunn-Moore et al. 1996, Malik et al. 1999).

Osteochondrodysplasias or metaphyseal chondrodysplasias are a group of abnormalities that result from defects in endochondral bone formation with variable skeletal and systemic involvement. A number of dog breeds, such as the Dachshund, have osteochondrodysplastic features and selecting for this disorder has created new breeds of cat, such as the Munchkin (Sausage) cat, the Bambino or the Minuet cats, with very short limbs. This deformity is produced by an autosomal dominant mutation and only heterozygous embryos develop into viable individuals. Potential problems in the Munchkin are likely to be similar to those in the equivalent dog breeds, such as intervertebral disc disease and angular limb deformity.

Osteochondrodysplasia has been reported as an inherited disease in the Scottish Fold cat. There is also a report of two unrelated kittens with signs similar to nonnutritional vitamin D-dependent rickets that were diagnosed as having metaphyseal chondrodysplasia. Affected Scottish Fold kittens develop progressive severe hindlimb lameness and a broad-based, inflexible tail. Radiographic changes of metaphyseal chondrodysplasia include widening of the physes (Figure 18.8) and exostoses around the tarsal and metatarsal joints. The exostoses become palpable clinically and eventually lead to ankylosis of the joints.

OTHER SOFT TISSUE DISORDERS:

Plasma Cell Pododermatitis

(Bettenay et al. 2003, Dias Pereira & Faustino 2003, Drolet & Bernard 1984, Guaguere et al. 2004, Langley-Hobbs 2012,

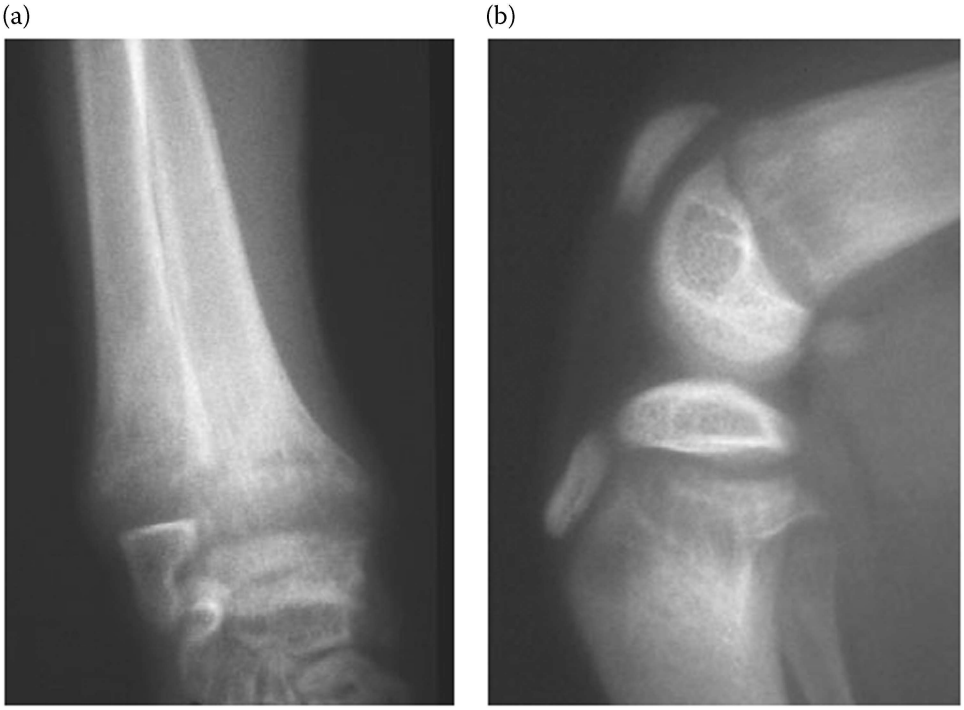


Figure 18.8 Osteochondrodysplasia in a kitten (metaphyseal chondrodysplasia). (Radiographs courtesy of Malcolm McKee.) **(a)** Craniocaudal radiograph of the distal radius and ulna showing the abnormal appearance of the physes. **(b)** Mediolateral radiograph of the stifle joint.

Medleau et al. 1982, Scarampella & Ordeix 2004, Taylor & Schmeitzel 1990, Yamamura 1998).

Plasma cell pododermatitis is a rare musculoskeletal disorder which often produces lameness in cats, due to pad tissue infiltration by plasma cells, with a possible immune-mediated or allergic etiology. In a retrospective study, neutered males of all ages were overrepresented, and the primary complaint was lameness in 22 out of 26 cats.

The affected pads may show a different colour (violet/mauve) and softer than normal consistency (Figure 18.9). Ulceration and chronic haemorrhage may occur due to the softness of the pad. In some animals there is a seasonal occurrence supporting the allergy supposition. Histologically, the dermis and subcutis are characterized by infiltration of plasma cells. Long-term glucocorticoid therapy is usually effective but surgical excision of focal lesions may also be necessary.

Successful treatment with doxycycline and gold salts has also been described, for their immunomodulator effect and suggesting an immune mediated pathogenesis.

OSSIFYING MUSCLE DISORDERS:

Fibrodysplasia Ossificans Progressiva (Myositis Ossificans)

This is a rare, progressive condition of muscle ossification that has received several names in the veterinary literature, including myositis ossificans and progressive ossifying myositis, athough the more accepted current nomenclature is fibrodysplasia ossificans progressiva.

Generalized form

(Asano et al. 2006, Klang et al. 2013, Langley-Hobbs 2012, Norris et al. 1980, Valentine et al. 1992, Warren & Carpenter 1982, Yabuzoe et al. 2009).

This condition has some features in common with fibrodysplasia ossificans progressiva in man, producing calcification in skeletal muscle and connective tissue. In young to middle-aged feline patients, it has been described to produce progressive stiffness, decreased limb movement, weakness and muscle pain due to the presence of calcified



Figure 18.9 A case of plasma cell pododermatitis, a rare cause of lameness. Observe the violet discoloration of the pads,which are often softer than normal. (Courtesy of Filippo DeBellis)

masses in muscles. This condition is a disorder of epimysial connective tissue that is characterized by hyperplasia and ossification causing atrophy of muscle tissue, entrapment of vessels and nerves, and progressive immobility. Radiographs or computed tomography reveal extensive soft tissue mineralization.

There is no effective treatment for the generalized form and the prognosis is poor.

Localized form

(Langley-Hobbs 2012, Lewis 1988).

The term myositis ossificans circumscripta continues to be used to describe nonhereditary forms of heterotopic ossification. This localized form more closely related to the human condition of myositis ossificans tends to occur in specific muscles such as the biceps femoris or semitendinosus bellies. Myositis ossificans may be related to local trauma or repetitive trauma. This form has a better prognosis than the generalized form in that the lesions stay localized and, presumably, they may be more amenable to curative surgical excision. Recurrence of the lesion, however, is likely after excision. Perhaps as a variation of this condition, there is one report of a fibrotic myopathy of the semitendinosus muscles.

PADS (PATELLAR FRACTURE AND DENTAL ANOMALY SYNDROME):

(Howes et al. 2018, Langley-Hobbs 2016, Langley-Hobbs 2009, Reyes et al. 2018).

This is a pathological disorder which involves a combination of patellar fractures and dental abnormalities such as non-eruption of permanent teeth and retained deciduous teeth. The current terminology is 'patellar fracture and dental anomaly syndrome' (PADS), although it can be found in older literature as 'Knees and Teeth syndrome' (KaTS).

Affected individuals suffer insufficiency patellar fractures (typically transverse, at the mid to proximal third of the patella) at a young age (median age of 28 months) and half the cases will develop a fracture on the contralateral patella within three months of the first fracture. The dental abnormalities can also lead to developmental bone abnormalities in the skull or mandibular problems such as abscessation or osteomyelitis.

Other long bone fractures are common in these cases. In a recent report, 48.2% of affected cats had dental abnormalities and 40.8% had fractures of other bones, such as pelvis, tibia, humerus, calcaneus, etc, described as 'insufficiency' or 'stress' fractures, often preceding the patellar fractures.

The best treatment for these fractures is still unknown and difficult to ascertain but surgery is unlikely to be successful in most cases, given the intrinsic abnormalities within the affected bone. Initial treatment of the dental abnormalities may include medical management with antibiotic and anti-inflammatory therapy but may also necessitate surgical extraction of persistent deciduous and unerupted teeth, including debridement of surrounding necrotic bone.

INFECTIONS OF BONE:

(Hess 2009).

Inflammation of periosteum, cortical bone and the medullary cavity is known as osteomyelitis. Most cases are caused by bacterial infection, which is discussed in Chapter 3. Infection of the intervertebral discs with secondary involvement of adjacent vertebral bodies is termed discospondylitis and is discussed in Chapter 15. Fungal osteomyelitis is occasionally encountered and is most commonly caused by Histoplasma capsulatum.

Histoplasmosis

(Ashbee et al. 2008, Boothe et al. 1997, Brömel & Sykes 2005, Clinkenbeard et al. 1987, Mavropoulou et al. 2010, Panackal et al. 2002).

Histoplasma capsulatum is a soil-borne fungal organism, especially prevalent on soil contaminated with bird and bat dropping, found in the southeastern and mid-western regions of North America but also in Central and South America, Africa, India and Southeast Asia. More recently, the disease has been recognized in Europe and some geographical areas of Italy, Germany and Turkey could be considered endemic for H. capsulatum.

Although disease is most prevalent in these areas, there are sporadic reports of fungal osteomyelitis in other parts of the world.

Infection occurs after inhalation of infective forms of the fungus. Osteomyelitis associated with histoplasmosis has been reported in cats of various ages. Cases typically present with clinical signs such as anaemia, weight loss, lethargy, fever, anorexia and interstitial lung disease, often present for several weeks before diagnosis is reached. They can also be presented with lameness, soft tissue swelling, and pain on palpation of the affected bone. Draining tracts over the osseous lesions were present in two cats. Systemic signs including anorexia, pyrexia, weight loss, lethargy, and chorioretinitis were present in some cases. Although the organism is contracted by inhalation of the fungal spores, respiratory signs were absent. Radiographic features included multifocal irregular areas of osteolysis and endosteal new bone formation around the metaphyses of affected long bones. The appendicular skeleton was most commonly affected, especially adjacent to the carpus and tarsus. More than one bone was affected in some cats and lesions were present in the skull in one case. Pathologic fractures occurred in two cats.

Diagnosis is made by bone biopsy or identification of the organism in the draining lymph nodes or in the bloodstream. Itraconazole is currently the drug of choice for the treatment of histoplasmosis. Studies in cats have shown significant variability in the absorption of the drug and the oral solution is more consistently absorbed than the capsules. The response to treatment is monitored clinically and radiographically. The drug is administered at 5–10 mg/kg divided twice daily for at least 3–6 months or until complete resolution of the infection.

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19 Oncology of the Skeletal System

NEOPLASTIC DISORDERS OF BONE:

(Dorn et al. 1968, Schneider 1978).

The skeletal system is not a common site for either primary or secondary neoplasia, with an incidence of 3.1–4.9/100,000 cats. The majority of bone tumours are malignant. Osteosarcoma (OSA) is the commonest tumour type, followed by fibrosarcoma and chondrosarcoma. Other primary tumours such as liposarcoma, anaplastic or undifferentiated sarcoma, giant cell tumour, multiple myeloma, plasma cell tumour and lymphoma are rarely observed and thus their true incidence is unknown.

PRIMARY MALIGNANT TUMOURS OF BONE:

Osteosarcoma

(Bitetto et al. 1987, Heldmann et al. 2000).

Primary malignant bone tumours occur much less commonly in the cat than the dog, representing less than 1% of all malignancies. OSA accounts for 70–80% of all feline primary bone tumours. Extraskeletal OSA has been reported in the cat; it arises from soft tissue and, accordingly, is not classed as a primary tumour of bone.

The incidence of OSA in the appendicular skeleton versus the axial skeleton varies with the studies. In one study, the incidence of appendicular OSA was almost double that of the axial skeleton but in another study, the incidence of appendicular OSA was approximately 50%.

Cause and Pathogenesis

(Almela et al. 2017, Banks 1971, Baum et al. 2018, Bennett et al. 1979, Dhaliwal et al. 2003, Fry & Jukes 1995, Gold et al. 2018, Sonnenschein et al. 2012, Spugnini et al. 2001, Stimson et al. 2000).

The etiology of OSA is not fully understood. Genetic predispositions exist in dogs with OSA and are the subject of current investigation but the situation in cats is unknown. The risk of tumour development may be increased at the site of prior local radiation treatment and at the site of previous fracture or orthopaedic intervention. In the reported cases that occurred following previous fracture, the tumour developed between 6 months and 17 years after repair of a femoral fracture. A true causative relationship between the previous surgery and the development of OSA is difficult to establish.

The majority of OSAs are of medullary or endosteal origin. Parosteal or juxtacortical OSA is a variant, which develops from the periosteum and thus is found on the outer cortex of the bone. Parosteal OSA has been reported in axial and appendicular sites and is seen more commonly in the cat than in the dog.

OSA in the cat is less aggressive than its canine counterpart, growing more slowly and metastasizing later. There are no pain fibres in the endosteum, so medullary OSA may be subclinical until there is cortical destruction and invasion into the periosteum and surrounding soft tissues.

The extraskeletal variation is relatively common, representing 56 out of 146 cases in one study, affecting typical vaccination sites as well as the eyes, oral cavity, mammary tissue, intestine, flank and liver.

Clinical Signs

(Bitetto et al. 1987, Heldmann et al. 2000, Leonard & Malik 1998, O'Brien et al. 1980, Schmidt et al. 2010).

OSA typically occurs in older cats, between 9 and 12 years of age but has been reported in cats as young as 1 year of age, so ruling out this neoplasia on the basis of the young age of the patient alone is unwise. This is in contrast to the dog, where the age distribution is bimodal, with rare exceptions. In one study, the average age at presentation of axial OSA was greater than that of appendicular OSA. There may be a slight predisposition in males but other studies show no gender difference. The predilection sites for appendicular OSA differ from those in the dog and preferentially affect the hindlimbs, especially the distal femur, proximal tibia and also the proximal humerus, although the tumours are not necessarily metaphyseal in location. Axial OSA originates most commonly in the skull (especially from



Figure 19.1 Lateral radiograph of a proximal humeral osteosarcoma.

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Figure 19.2 Lateral view of a proximal tibial osteosarcoma, producing a pathological fracture.



Figure 19.3 Distal tibial osteosarcoma.

the oral cavity) and pelvis but has also been reported in the ribs and vertebrae.

Clinical signs include progressive lameness, pain and local swelling. Neoplasia of the vertebrae usually causes neurologic dysfunction due to spinal cord compression. Lameness may sometimes be acute, following a minor traumatic incident, or peracute, if there is pathologic fracture with no antecedent signs (Figures 19.1–19.4).

Diagnosis

(Liu et al. 1974, Quigley & Leedale 1983).

The radiographic and tomographic features of OSA are variable. The classical picture is that of an aggressive, poorly delineated, monostotic lesion with a mixed pattern of destructive and productive bony change, cortical destruction and extension into the soft tissues. Lesions of the long bones are, however, often predominantly lytic in nature and a solitary osteolytic lesion of a long bone in an older cat would carry a high index of suspicion for OSA. The main radiographic differential diagnoses for OSA are osteomyelitis (bacterial or fungal) and, in the case of very lytic lesions, bone cysts should also be considered. Fungal osteomyelitis is seen mainly in specific geographic areas but there are sporadic reports in many parts of the world. It has been suggested that if the radiographic findings are equivocal, then radiographs should be repeated at a later date. In this situation, it is preferable either to submit the radiographs or CT to an experienced radiologist for interpretation or to proceed with immediate biopsy, since any delay will increase the risk of metastasis if the lesion is malignant. Reviews of the imaging study by a board-certified imager would be very helpful in doubtful cases.

Diagnosis should be confirmed by bone biopsy, preferably with a Jamshidi needle or by using a limited surgical

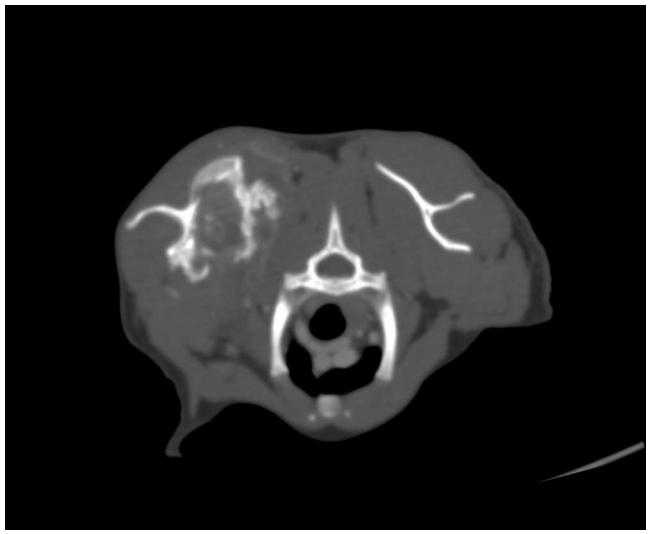


Figure 19.4 Transverse CT image of a scapular osteosarcoma.

approach. The authors prefer the use of the Jamshidi needle core biopsy instrument. When a minimum of at least two samples are taken (one central and one in the transition zone), the accuracy of diagnosing a known OSA by an experienced pathologist is probably in the order of 90% or higher. The use of the Jamshidi needle is described in Chapter 2. Diagnostic accuracy can be improved by verifying correct tissue sampling on a postoperative radiograph of the biopsy site. Fine-needle aspirates of the surrounding soft tissues (of tumour-invaded periosteum, for example) may also prove useful, if only to make an initial, fast diagnosis of a malignancy based on the identification of sarcoma cells.

Histologically, both canine and feline OSA share the same features, mainly mesenchymal cells embedded in a variable amount of cartilage and malignant osteoid. Feline OSA is also characterized by the presence of multinucleated giant cells. The surrounding bone is invaded by the neoplasia and the soft tissues may be infiltrated by it, or just compressed. The histologic types described resemble those of canine OSA and include chondroblastic, fibroblastic, giant cell and telangiectatic. Efforts to link histologic type and prognosis have not shown any correlation. A pathology report of reactive bone does not constitute a diagnosis and further confirmatory studies are needed. There is no evidence that performing a biopsy increases the metastatic rate.

Treatment and Prognosis

(Barbur et al. 2015, Dimopoulou et al. 2008, Ehrhart et al. 2013, Heldmann et al. 2000, Wolfesberger et al. 2016, Wypij et al. 2008).

Metastasis of feline OSA is less frequent than its canine counterpart, but three view, inflated thoracic radiographs or a thoracic computed tomography study should always be obtained prior to surgery. It has been estimated that the metastatic rate in cats is only 5–10% versus more than 90% in dogs and usual anatomic sites for metastasis include lung, kidney, brain and spleen.

It has been theorized that the stem cell growth factor receptor KIT may be implicated in the higher metastatic rate of canine OSA cells versus its feline counterpart. The mRNA expression of KIT was quantified by reverse transcription-quantitative polymerase chain reaction and was observed to be significantly higher in canine than in feline osteosarcoma samples in a study.

The treatment of choice for OSA of the appendicular skeleton continues to be amputation, which has relatively good results compared with the dog. An amputation that achieves complete surgical excision is important since it has been correlated with a longer survival time disease-free interval and recurrence-free interval. The authors prefer a complete forequarter amputation for OSA in any location in the front limb and an amputation through coxofemoral joint disarticulation for OSA of the distal femur or more distal. In cases of proximal femoral or pelvic involvement, a variation of hemipelvectomy has been described and is recommended to achieve proper surgical margins.

In one study of a relatively small number of cases the median survival time for cats with appendicular OSA treated by amputation was 64 months and the median survival time for cats with axial OSA was 5.5 months. The mean survival time in another larger series of cases was 11.8 months for appendicular OSA treated by excisional biopsy or amputation and 6.07 months for cats with axial OSA. The reason for the difference in the survival times for appendicular OSA between these two studies is not clear. Some histological features such as tumour vascular invasion, mitotic index, cell necrosis, etc have been correlated to a worse prognosis and, especifically, a high mitotic rate has been linked to a decreased recurrence-free interval.

Adjunctive chemotherapy is not normally given for appendicular OSA because of the success of surgery alone. OSA of the axial skeleton carries a less favourable prognosis because the site of the tumour usually precludes complete surgical removal. Aggressive excision and adjunctive chemotherapy, using carboplatin and/or doxorubicin, are likely to provide the best prognosis for these tumours. Cisplatin must not be used for adjunctive chemotherapy in cats because of its toxicity. A combination of modalities is likely to also contribute to a longer survival time but there are no definitive studies to confirm this point.

There is a small amount of evidence for the use of bisphosphonates in cats to help reduce bone resorption in oral squamous cell carcinoma (SCC) but this may not be applicable to OSA.

Chondrosarcoma

(Davidson 1995, Durham et al. 2008, Liu et al. 1974, Merchav et al. 2005, Mukaratirwa et al. 2001, Shell et al. 1987, Thompson et al. 2016, Turrel & Pool 1982, Vinayak et al. 2018).

Chondrosarcoma is reportedly the third most common primary bone tumour in cats and may be indistinguishable clinically and radiographically from other primary bone tumours (Figure 19.5–19.7). In a recent review, males were



Figure 19.5 Chondrosarcoma. Mediolateral radiograph of the stifle showing chondrosarcoma of the proximal tibia.



Figure 19.6 Scapular chondrosarcoma, in a patient ready for amputation.

much more commonly affected than females, and a mean age of 9.6 years was observed.

An uncommon, more aggressive variant of chondrosarcoma has been reported in one cat, in which a high-grade sarcomatous component is found adjacent to a low-grade cartilaginous component, referred to as a bimorphic pattern, to confirm the diagnosis of a dedifferentiated chondrosarcoma on histopathology.



Figure 19.7 Distal tibial chondrosarcoma. (Courtesy of M. Ruiz).

Chondrosarcoma occurs in both long bones and flat bones but reported numbers in the cat are too small to make valid conclusions about site predisposition. A periosteal origin has also been described.

Presenting signs are similar to those of OSA; they include pain and lameness associated with the presence of a mass, which may have been present for many months. There is one report of forelimb paralysis secondary to a chondrosarcoma that was compressing the brachial plexus. The growth rate of chondrosarcoma is generally slow initially. Pulmonary metastasis has been reported, but complete resection, usually by amputation for tumours of the long bones, is generally associated with a good prognosis.

Giant Cell Tumour

(Alexander et al. 1975, Bennett & Duff 1983, Ferreras et al. 2005, Howard & Kenyon 1967, Leonardo et al. 2013, Ogilvie & Moore 2001, Popp & Simpson 1976, Schmidt et al. 2010, Thornburg 1979, Walsh & Rhodes 1995).

Giant cell tumours of bone, previously cited as osteoclastoma in the literature, are considered rare in all domestic animals, although they seem to be more commonly diagnosed in the cat than in the dog. Giant cells may be found in both neoplastic and nonneoplastic bone lesions, therefore diagnosis of neoplasia is based on a combination of the clinical, radiographic and histologic features. Giant cell tumour of the bone is a very rare tumour with uncertain histogenesis. Of the reported cases, several cats were only one year of age.

The radiographic characteristic of giant cell tumours is typically an expansile, multiloculated appearance, causing significant osteolysis of the long bone diaphysis. They can be confused with bone cysts or any expanding nonosteogenic tumour. Treatment by resection of the diseased bone or by amputation has been successful.

Activity levels of acid and neutral proteases acting in feline giant cell tumour cells, which induce osteolysis by destroying extracellular matrix, have been studied and compared to human counterparts. The possible use of these molecules as diagnostic and prognostic tools in these tumours still has to be investigated.

Other Sarcomas

(Canapp et al. 2001, Cook et al. 1998, Quigley & Leedale 1983, Simon et al. 2000, Straw et al. 1992, Trout et al. 1995).

Other mesenchymal tumours, such as fibrosarcoma, hemangiosarcoma, rhabdomyosarcoma, liposarcoma and anaplastic sarcoma, occur sporadically.

Fibrosarcomas may arise from soft tissue or bone and account for up to 12% of all feline neoplasms, although they are much more common as a soft tissue sarcoma. Bone fibrosarcoma is the second most frequent primary bone neoplasia after OSA but still relatively rare. The primary cause in young cats is feline sarcoma virus (FeSV) and these neoplasms are usually multicentric, poorly differentiated and highly malignant. Solitary fibrosarcomas are typically less invasive, occur in geriatric cats and have no relationship to FeSV.

Fibrosarcomas of bone arise from medullary or periosteal connective tissue. The radiographic appearance is predominantly osteolytic with minimal reactive response; transarticular involvement has been reported (Figure 19.8).



Figure 19.8 A rare primary bone fibrosarcoma, affecting the distal tibia. (Courtesy of C. Macias).

Fibrosarcoma should be considered in the differential diagnosis of any osteolytic lesion in cats of all ages. Well-differentiated tumours usually do not metastasize and complete resection of diseased bone, usually by amputation, may be curative. There are reports of resection of fibrosarcoma involving the pelvis and scapula by total hemipelvectomy and partial scapulectomy, respectively. The metastatic potential and, therefore, the prognosis with other sarcomas is variable.

Hemangiosarcoma and anaplastic sarcoma may be more aggressive than osteosarcoma although much more rare. Limb amputation is usually the treatment of choice for all mesenchymal tumours of the long bones, although this may not be curative. Recurrence at the surgical site and pulmonary metastasis occurred within 4.5 months in a recent report of a rhabdomyosarcoma treated by amputation. It should be noted that not all bone sarcomas are primary bone tumours. A thorough search should always be made for other tumour deposits since a proportion will be metastases from mesenchymal tumours of soft tissue origin or from another skeletal site.

ROUND CELL TUMOURS:

Round cell tumours of the skeleton are uncommon in comparison with the sarcomas. Round cell tumours that commonly affect bone are plasmacytoma, multiple myeloma and lymphoma. Multiple myeloma is a plasma cell neoplasm that has its origin in the bone marrow.

Plasmacytoma is a plasma cell tumour arising from tissue other than bone marrow. Clinical signs relating specifically to skeletal involvement are not common but are seen most often with multiple myeloma.

Plasmacytoma

(Mellor 2007, Soutar et al. 2004).

Solitary plasmacytoma is a myeloma-related disorder (MRD), that occasionally affects either the axial or appendicular skeleton. Where a long bone is affected, wide surgical excision, usually by amputation, may be curative. Where this is not possible, radiation therapy may be considered as an alternative since these tumours are radiosensitive. Survival time of 4 years in one case has been reported with radiotherapy alone. Chemotherapy treatment with melphalan has also been reported, with a long survival time in a single case, of 6 years.

In humans, solitary plasmacytoma of bone commonly undergoes transformation into myeloma, with a median time to progression of 2–4 years. To the authors' knowledge, this has not been reported in the cat.

Multiple Myeloma

(Bienzle et al. 2000, Cannon et al. 2015, Eastman 1996, Erickson et al. 2018, Fikry 2005, Jacobs 1996, MacEwen 1977, Mellor et al. 2006, Mills et al. 1982, Osborne et al. 1968, Patel et al. 2005, Vail 2000, Weber & Tebeau 1998).

Cause and pathogenesis

Multiple myelomas are unusual bone neoplasms in cats, arising from plasma cells in the bone marrow and then involving other organs, while solitary plasma cell tumours in organs other than bone marrow are called plasmacytomas. In the limited literature available on the subject, this disease usually affects middle-aged cats and there is a male predisposition. Multiple myeloma accounts for less than 1% of feline haematopoietic neoplasms, plasmacytomas being even less common.

No link has been found between infections by feline leukaemia virus (FeLV) or feline immunodeficiency virus (FIV) and the development of myelomas.

Clinical signs

Presenting signs tend to be insidious in onset and non-specific, including depression, anorexia, polyuria, polydipsia and weight loss. Clinical signs related to bone involvement include lameness, bone pain, neurologic dysfunction due to extradural spinal cord compression by bone lesions in the spinal column, and pathologic fracture due to the osteolytic effect of the neoplastic cells. In a retrospective study of cats with multiple myeloma, 7 out of 12 cats evaluated radiographically showed solitary or multiple bone lesions.

In one study with nine cases, 11% of cats presented with lameness and 44% presented with paresis/paralysis with pain.



Figure 19.9 Lateral view of humerus, showing multiple lytic lesions due to multiple myeloma. (Courtesy of M. Ruiz).

Diagnosis

Laboratory findings include anaemia, thrombocytopenia, azotemia, hypercalcemia and elevated globulin with a monoclonal gammopathy seen on serum protein electrophoresis, leading to a hyperviscosity syndrome. Bence Jones protein (myeloma light-chain protein) may be present in the urine and because of their nephrotoxicity, they can induce renal insufficiency in these patients. Osteolytic lesions are typically well-circumscribed, 'punched-out' bone defects that can affect any part of the skeleton including the spine, although the distal extremities may be most commonly affected, in contrast to the dog (Figures 19.9 and 19.10). A CT bone study may show subtle lesions earlier than radiographs.

The diagnosis of multiple myelomas in the cat is based on the criteria previously developed for the dog and at least two of the following criteria need to be met:

1. Paraproteinaemia or monoclonal gammopathy, in blood or urine.
2. Radiographic evidence of osteolytic bone lesions.
3. Bence Jones proteinuria.
4. Greater than 5% neoplastic plasma cells in the bone marrow.

Histopathologic confirmation can be made from a bone marrow aspirate or a bone biopsy, showing infiltration of malignant plasma cells.

Treatment and prognosis

Multiple myeloma cases are usually treated with chemotherapy (Melphalan), in combination with immunosuppressive drugs. There is a need for administration of antibiotics at the beginning of therapy to prevent opportunistic bacterial infection since these patients are prone to infection. It is hypothesized that the main reason for this is that normal immunoglobulin levels are severely depressed.



Figure 19.10 Craniocaudal view of the same case. (Courtesy of M. Ruiz).

Radiotherapy has also been used to treat focal myeloma lesions and in one report, response to a combination of surgery, radiation and chemotherapy provided a long survival time of 2 years. However, the disease is invariably progressive and fatal, with an average survival time of 12–16 months.

It has been reported that chemotherapy with cyclophosphamide or melphalan combined with prednisolone increase survival time and radiotherapy may be applied palliatively and that the combination of cyclophosphamide and corticosteroids is usually well-tolerated and should be considered as the first-line therapy in cats with systemic myeloma disorders.

Lymphoma

(Brockley et al. 2012, Flatland et al. 2008, Liu et al. 1974, Pool 1990, Quigley & Leedale 1983, Suess et al. 1990, Thompson & Pool 2002).

Lymphoma occasionally presents with skeletal involvement typically, in the vertebrae of young cats. Neoplasms of lymphoreticular origin involving bone have not been reported. Lymphoma may occur as a primary bone neoplasm but most frequently occurs in association with multicentric lymphoma or leukaemia. Infiltration of bone by neoplastic cells will replace bone marrow but bone destruction is rarely recognized radiographically or as an initial cause of clinical signs. A more distal distribution of bony osseous lesions has been described in a case with multiple pathological fractures in a cat.

A thorough search should be made for tumours in other sites since lymphoma is seldom solitary. Local excision or radiotherapy may be appropriate, but combination chemotherapy is advisable, even in the absence of overt disease elsewhere.

JOINT NEOPLASIA:

(Walsh & Rhodes 1995).

Neoplasia involving joints is very rare in the cat. Primary joint neoplasms include synovial cell tumours arising from the synovial membrane which then secondarily invade adjacent bone. Periarticular and juxta-articular malignancies, such as osteosarcomata and fibrosarcomata, may arise from extraarticular tissues and invade joints secondarily. Transarticular involvement of fibrosarcoma has been reported. The presentation, diagnosis and treatment of these tumours are similar to synovial sarcoma.

Metastasis of malignant tumours to joints is very rare.

Synovial Cell Neoplasia

(Bennett & Duff 1983, Cazzini et al. 2015, Heldmann et al. 2000, Liptak et al. 2004, Merchav et al. 2005, Pinard et al. 2006, Shell et al. 1987, Turrel & Pool 1982).

Feline synovial sarcoma is a rare articular tumour.

Synovial sarcomata originate from mesenchymal synovioblastic cells in the joint capsule, tendon sheath, or bursa and invade the joint cavity secondarily. Clinical signs include chronic progressive lameness, with periarticular soft tissue thickening and swelling, pain on joint manipulation and reduced range of motion. All reported cases have involved the joints of the distal extremities or the elbow. Radiography typically reveals a poorly defined periosteal reaction and multiple punctate osteolytic lesions involving the epiphyses of more than one bone adjacent to the affected joint.

Diagnosis may be confirmed in some cases by cytologic examination of synovial fluid but otherwise requires incisional biopsy. The tumour should be staged before surgery by performing chest radiographs and needle aspiration of the regional lymph node. Biopsy may suggest that the tumour is a benign synovioma, but, in those cases where local excision was performed, the neoplasm recurred. In dogs, survival may depend more on the aggressiveness of treatment rather than on clinical staging or tumour grading and the recommended method of treatment is amputation. The metastatic potential of the tumour in cats is unknown, although in dogs 41–54% of cases have metastases at the time of diagnosis or will develop metastasis subsequently. Only two cases of metastatic synovial cell sarcoma to regional lymph nodes have been described in cats. The best treatment for synovial cell sarcoma in the cat is not known, since there is a very small number of cases in the literature. Amputation may be the best option in most cases, given the degree of articular bone destruction at the time of diagnosis and given the lack of data on effectiveness of palliative chemotherapy or radiotherapy. The two reported metastatic cases received adjuvant chemotherapy (doxorubicin), with survival times of 444 and 41 days.

Given the difficulty in the definitive diagnosis of this tumour and its similarities to other synovial neoplasia, such as histiocytic sarcoma and similar entities, a more in-depth characterization of the feline synovial cell sarcoma cell has been described in a case report, based on ultrastructural, immunohistochemisty and morphological features, in an attempt to further define this pathology.

SECONDARY MALIGNANT TUMOURS OF BONE:

(Dorn & Priester 1976, Fleming et al. 2019, Gardner & Dubielzig 1995, Martin et al. 2010, Rousset et al. 2013, Stebbins et al. 1989).

A number of soft tissue tumours have a propensity to invade bone locally, causing varying degrees of osteolysis and periosteal reaction. The most common of these is squamous cell carcinoma (SCC). Sarcomas, especially fibrosarcomas, anaplastic sarcomas, rhabdomyosarcoma and synovial cell sarcomas arising in soft tissue adjacent to bone, often also behave in this way. For example, injection-site sarcomas can secondarily invade the bone of adjacent vertebral spinous processes, although this finding is not a common one. In a report, 13 out of 19 cases with bone contact, none had evidence of local osteolysis. Also, in a recent study of 10 cats with injection-site sarcomas, none showed bony lysis of adjacent bony structures, when evaluated with CT angiography and MRI.

Also, local bone involvement has been reported in cats affected by feline inductive odontogenic tumour (FIOS), typically in the maxilla, producing tooth loss.

Squamous cell carcinoma of the gingiva is frequently associated with a marked bony reaction of the mandible or maxilla that may be mistaken for osteosarcoma radiographically. It is the most commonly diagnosed tumour of the oral cavity in cats.

Feline oral SCC arises most frequently from the gingiva and secondarily shows involvement of adjacent bone, characterized by osteoclastic bone resorption, formation of

new bone and direct apposition and adhesion of neoplastic cells to bone. The mechanisms that regulate the ability of some neoplasias to invade adjacent bone and produce osteolysis are not fully understood, but likely involve the activity of osteoclasts mediated by the expression of several possible regulators such as parathyroid hormone-related protein (PTHrP), interleukin 1α (IL-1α), tumour necrosis factor α (TNFα), receptor activator of nuclear factor κB ligand (RANKL), osteoprotegerin (OPG) and transforming growth factor β1 (TGFβ1). A relationship between the expression of various regulators of osteoclastic activity in feline oral SCC cells and osteoclastic bone resorption has been demonstrated. In a study involving 451 cases, it was determined that specimens with osteolysis had increased PTHrP expression and nuclear localization, compared to oral SCC without osteolysis. Tumour necrosis factor α expression was only present in a very small proportion of tumour cells. This suggests that PTHrP may play an important role in bone resorption and tumour invasion in cats with oral SCC.

Primary digital squamous cell carcinoma arises from the epithelium of the nail bed and invades the phalanx by direct extension. Treatment by digital amputation carries a good prognosis. The majority of feline digital carcinomas, however, are metastatic with a primary pulmonary origin.



Figure 19.11 Photograph showing swelling and ulceration of digits.

METASTATIC TUMOURS TO BONE:

Metastatic digital carcinoma or feline lung-digit syndrome

(Anderson et al. 2000, Bertone et al. 2003, Brown et al. 1985, Cohen 2001, Finotello et al. 2017, Gottfried et al. 2000, Hanselman & Hall 2004, Helm & Morris 2012, Malik et al. 2002, Malik et al. 2006, Reppas et al. 2015, Schoen et al. 2010, Scott-Moncrieff et al. 1989, Scott & Miller 1992, Thrift et al. 2017, Van der Linde-Sipman & van den Ingh 2000, Wobeser et al. 2007).

Cause and pathogenesis

Some feline tumours have a predilection for metastasis to bone, most notably primary pulmonary malignancies. Hematogenous metastasis of primary pulmonary malignancies to the distal limbs and digits, mainly affecting P2 and P3 in several digits often of different paws, has been the subject of a number of case reports. Digital metastasis of pulmonary carcinoma has not been reported in the dog and is uncommon in humans.

It has been suggested that the vascularity of the feline footpads required to facilitate heat loss may predispose to this pattern of metastasis in the cat. The dewclaw is not usually affected, perhaps supporting this theory. Other factors facilitating this type of metastasis are the angioinvasive properties of these lesions, facilitating their haematological spread. Tumour cells have been identified within the pulmonary and digital arteries in histopathological samples. Factors involving cell markers or chemical secretions may also play a role in this syndrome.

Clinical signs

The typical clinical signs include lameness, accompanied by swelling of the distal extremity of the digit, ulceration of the digital skin or claw bed, discharge, deviation or loss of the claw (exsheathment) (Figure 19.11). More vague systemic clinical signs, such as malaise, inappetence and pyrexia, can also be seen. Other, more rare, localizations for metastasis have been reported, such as skeletal muscle, skin or distal aorta.

In a 2007 clinical study of 85 feline digits submitted for histopathology, it was found that 63 showed evidence of neoplasia, with malignant disease affecting 95% of these cases. Significantly, 21% of the tumours identified were adenocarcinoma, presumed to represent metastatic spread from a primary pulmonary neoplasm.

There may be an association, as a paraneoplastic disease, between hypercalcaemia and malignancy of feline bronchogenic carcinoma.

In the only large series of cases, 36 cats were identified with digital metastases originating from a bronchogenic carcinoma. The mean age was 12.7 years with no breed or sex predilection. The 19 cats for which full records were available all presented with the primary complaint of lameness. None of the cats had respiratory signs, despite the presence of radiographically detectable pulmonary carcinoma.

Diagnosis

Radiographs of the digits show extensive osteolysis of the third phalanx, with invasion of the distal interphalangeal joint, possible osteolysis of P2 and severe soft tissue swelling (Figure 19.12). Occasionally, periosteal proliferation has been seen on all phalanges of the affected foot. Thoracic radiography may show the typical appearance of a primary pulmonary neoplasia as a solitary mass in the caudal lung lobes (Figure 19.12), but other presentations are possible, such as multiple circumscribed masses, lobar consolidation or in a diffuse pattern affecting one or more lobes. Associated pleural effusion may also be seen, often causing respiratory compromise. Enlargement of the tracheobronchial lymph nodes is possible, although thoracic computed tomography may be necessary to fully appreciate the thoracic structures.

Diagnosis can be obtained through aspiration cytology (FNA), analysis of an avulsed claw or a biopsy may be



Figure 19.12 Dorsoplantar radiograph showing an osteolytic lesion in the third phalanx of digits 2 and 3. (Courtesy of C. Macias).

required for histopathological confirmation, including the cases with unusual presentations such as muscle lesions. In the more classic presentation of multiple claw bed involvement, the clinical presentation is typically very suggestive of this condition. Amputation of an affected digit may also be necessary to provide a larger sample for a final histopathological diagnosis. In a recent report, the use of thyroid transcription factor-1 (TTF-1) along with the microscopic confirmation of ciliated epithelium has been reported for the diagnosis of this condition. TTF-1 is a nuclear protein known to play a crucial role in the differentiation of lung epithelial cells, which remains expressed in neoplastic tissue and therefore can be useful in confirming the primary pulmonary origin of lung tumours since non-pulmonary and non-thyroid neoplastic tissues do not express TTF-1.

Confirmation of pulmonary neoplasia can often be made based on thoracocentesis of pleural effusion, cytology of tracheal washes or bronchoscopic samples, or fine-needle aspiration of a peripherally located lung mass.

It is important to differentiate metastatic digital pulmonary carcinoma from primary digital squamous cell carcinoma. In comparison with metastatic carcinoma, primary digital neoplasia is rare, is unlikely to be multiple, and does not usually show pulmonary metastasis at the time of initial presentation. All geriatric cats presented with multiple digital swellings should have thoracic radiography or, with an increased sensitivity, thoracic CT, to rule out a primary pulmonary neoplasm. Abdominal ultrasonography is also recommended since extraskeletal metastases can also occur, such as to lymph nodes, lung, spleen, kidney, skeletal muscle and heart.

Multiple bacterial paronychia, nocardia infections, mycobacterium species (e.g. feline leprosy syndrome), fungal infections and immune-mediated disease are other differential diagnoses for nail bed disorders in cats.

Treatment and prognosis

Amputation of affected digits, given the typical multiple involvement, may be palliative in the short-term but does little to alter the clinical course and progression of the primary pulmonary tumour. The prognosis was poor with a mean survival time of 58 days from initial presentation in one study. If there is no evidence of metastasis, surgical resection of primary, solitary lung neoplasia is still recommended since the survival time can be favourable in well-differentiated tumours. Feline pulmonary adenocarcinoma is not considered to be a chemotherapy-responsive disease.

Mammary Tumours

The majority of feline mammary tumours exhibit malignant behaviour, with frequent early metastasis to the regional lymph nodes and lungs. However, skeletal metastases were found in only 4% of cases in one large study of cats with metastatic mammary adenocarcinoma. In contrast to the dog, the distal portions of the limbs were more likely to be affected, particularly the talus.

BENIGN TUMOURS OF BONE:

Benign bone tumours are occasionally reported in the cat, the commonest of which is the osteoma. Ossifying fibroma, chondroma and osteochondroma have been reported less frequently. Osteoma and ossifying fibroma are similar histologically and have not been adequately differentiated in the veterinary literature.

Osteoma

(Cunha et al. 2015, Darnell et al. 2007, Fiani et al. 2011, Jabara & Paton 1984, Lantz et al. 1982, Pool 1990, Quigley & Leedale 1983, Schmidt & Langham 1967).

Osteoma occurs as a solitary bony outgrowth with a sessile base that typically arises on the surface of an

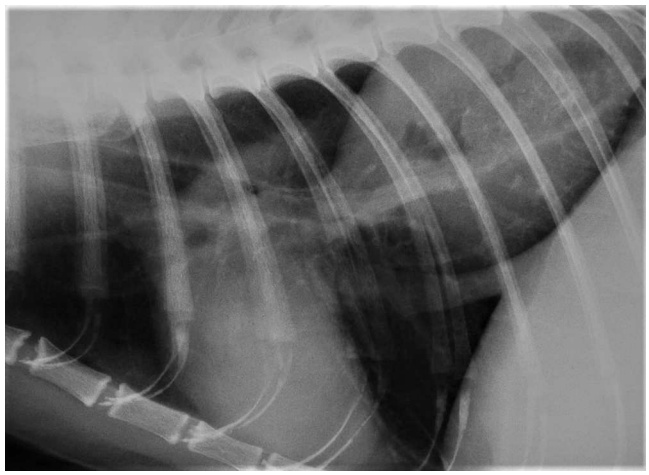


Figure 19.13 Lateral radiograph of the thorax showing a pulmonary bronchogenic carcinoma in the caudal lung field.



Figure 19.14 Osteomas are benign osseous tumours, occasionally seen on the surface of long bones.

intramembranous bone (Figure 19.14). The tumour is composed of mature compact or cancellous bone and grows slowly, with the underlying cortex either remaining intact or remodelling with the base of the lesion.

Osteomas arising on the surface of bone are referred to as periosteal osteomas, and if they develop within bone are more accurately termed to endosteal osteomas; extraskeletal osteomas have also been described.

Although the etiology is unknown, trauma, infection and developmental abnormalities have been suggested as contributing factors. In the cat, they have mainly been reported in the maxillofacial region but also a rib location has been described.

The diagnosis is usually made by clinical evaluation, diagnostic imaging and histologic testing. Osteoma bone is, however, histologically very similar to reactive bone.

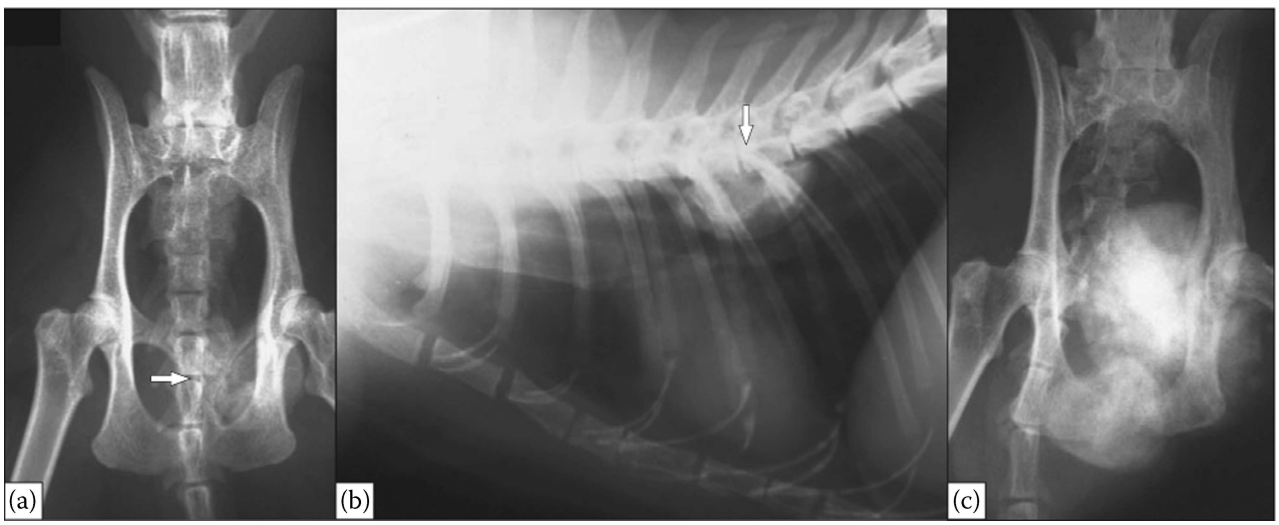


Figure 19.15 Osteochondromatosis. **(a)** Ventrodorsal radiograph of the pelvis showing an ossified mass attached to the pubis (arrow). The mass was surgically resected. **(b)** Lateral radiograph of the thorax of the cat in **a** showing an ossified mass projecting ventrally from the mid-thoracic vertebrae (arrow). **(c)** Ventrodorsal radiograph of the pelvis of the cat in **a** and **b** 1 year later. There are now multiple ossified masses arising from the pubis and ischium. Biopsy revealed malignant transformation to OSA.

Treatment by surgical excision from the surface of the bone carries a good prognosis.

Chondroma

(Hodik et al. 2012, Lichtenstein & Hall 1952, Sullivan 1960).

A chondroma is a benign, rare neoplasm of cartilage that has been reported in several domestic species, particularly dogs and sheep. They can arise from the medullary cavity of bone or from cartilage elsewhere in the skeleton, such as flat bones and the sternocostal area. In humans, a periosteal chondroma, developing under the periosteum has been reported.

A case report of a chondroma in the mandible of a cat has been described, as a relatively radiopaque mass with a mixed bony opacity probably due to matrix mineralization. Surgical removal provided good function and a recurrence-free period of 3 years.

Ossifying Fibroma

Ossifying fibroma is a tumour-like growth of fibro-osseous tissue that replaces alveolar or cortical bone in the mandible or maxilla (Figure 19.16). Resection of the affected portion of the jaw is necessary to prevent local recurrence. This benign neoplasm has not been specifically reported in the cat.

Solitary Osteochondroma

(Langley-Hobbs 2001, Rosa & Kirberger 2012, Schmidt & Langham 1967, Turrel & Pool 1982).

An osteochondroma is a solitary cartilage-capped exostosis that arises from the surface of any bone formed by endochondral ossification. The lesion is thought to originate as a result of an abnormality in the underlying periosteum, such as the separation of epiphyseal growth plate cells and their herniation through the periosteum adjacent to the growth plate. Also, these tumours may originate from separate secondary ossification centres in the spinal processes.

Osteochondromas are rare in cats. When the presentation involves multiple sites, this condition is named osteochondromatosis (see later in this chapter).

The presenting complaint is usually lameness and the typical radiographic appearance is of a single bony outgrowth affecting the appendicular skeleton and consisting of well-demarcated and well-organized new bone. Solitary osteochondromas have no clinical significance unless there is pressure on adjacent structures. Affected cats are typically middle-aged and Burmese cats are overrepresented. The commonest appendicular site is the distal humerus but the spine has also been reported, with secondary compression of neurological structures. Also, an extraskeletal location has been reported.

Surgical excision carries a good prognosis, although there is a tendency for local recurrence due to the cartilaginous cap blending into the surrounding soft tissues and making full surgical resection difficult. Differentiation from the multiple forms of the disease by radiographic evaluation of the skeleton and FeLV testing is advisable prior to surgical excision. The exact, possible role of the FeLV virus in the pathogenesis of solitary osteochondromas in the cat is still unknown.

Osteochondromatosis

(Cotter 1998, Langley-Hobbs 2001, Levitin et al. 2003, Perry 2014, Perazzi et al. 2013, Poole et al. 1972, Straw 2000, Tan et al. 2010, Tas et al. 2013, Villacin et al. 1979).

Osteochondromatosis is also known as multiple cartilaginous exostoses (MCE). It is a polyostotic disease with the same histologic features as solitary osteochondroma. The literature on MCE is confusing because of the numerous synonyms for the disorder. In humans, dogs and horses it is an inherited disease that develops in the immature skeleton and is considered to be a type of skeletal dysplasia rather than a true neoplasm. A similar disease occurs in cats but, in contrast to other species, the masses first appear after skeletal maturity, they undergo progressive enlargement, and they often increase in number until functional impairment necessitates euthanasia. The mean age of 10 cases reported in the literature was 2.5 years and all cats that were checked for the presence of FeLV tested positive.

The pathogenesis of feline osteochondroma is presumed to be related to FeLV infection of periosteal fibroblasts but the exact role is still unclear. Another virus that has been implicated is the atypical feline fibrosarcoma virus-feline oncornavirus. Although the Siamese breed was featured in an earlier report there is no apparent breed or sex predisposition.

Clinical signs occur due to impingement of the exostoses on normal anatomic structures.

The skull, scapula, pelvis, ribs and vertebrae are preferentially affected. Lesions in the limbs may produce obvious swelling and pain, and lameness may be present. Vertebral lesions cause paresis or paralysis if they impinge on the spinal cord. Synovial osteochondromas that arise without a known predisposing cause are termed primary synovial osteochondromas, while those arising from a diseased joint are referred to as secondary synovial osteochondromas, in which chronic osteoarthritis is very common.

Lesions appear radiographically as multiple eccentric, sessile or pedunculated protuberances of cancellous bone that merge into the substance of the parent bone. In long-standing cases, the lesions may take on a more aggressive appearance and malignant transformation to chondrosarcoma or OSA may occur (Figure 19.15). Survey radiographs of the skeleton will reveal the true extent of the disease. Biopsy of lesions is required for definitive diagnosis prior to treatment. Cats with MCE should be considered to be FeLV-positive until proven otherwise.

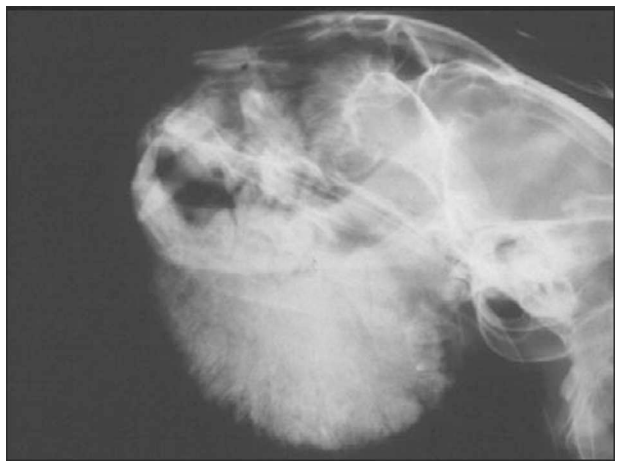


Figure 19.16 Lateral radiograph of the skull showing an ossifying fibroma arising from the mandible. (Radiograph courtesy of Malcolm McKee).

Treatment by surgical resection may provide temporary relief, but is palliative at best, because of the tendency for local recurrence and new lesion development at other sites. Full surgical resection of the lesions may prove very difficult, due to indiscrete margins, and relief is generally short-lived, due to frequent recurrences and emergence of new lesions.

The treatment of choice for synovial osteochondromatosis is total synovectomy with removal of all loose bodies but many of the fragments seen radiographically may be embedded in the synovium and not easy to locate. The medical management of these cases may include the judicious use of non-steroidal antiinflammatory medication and analgesics.

NEOPLASIA-LIKE DISORDERS OF BONE:

Benign Bone Cyst

(Berger et al. 2016, Capanna et al. 1984, Markovic & Cvijetic 1988, Miura et al. 2003, Yanagi et al. 2010).

Bone cysts are very rare, benign, unicameral fluid-filled structures of unknown etiology, both in human and veterinary medicine. Different theories about aetiology have been proposed including local infection, vascular intraosseous malformations, traumatic causes or altered bone metabolism They are most commonly monostotic but may occasionally be polyostotic.

The cysts are lined with a thin layer of fibrous connective tissue and filled with serous fluid. Bone cysts are usually located in the metaphyseal region at the distal end of long bones, sparing the growth plates and epiphyses. They are frequently asymptomatic unless they are large or cause pathologic fracture. Large cysts may cause local swelling and pain and the adjacent joint may have a reduced range of motion. Radiography shows a multilocular radiolucent lesion with expansion and thinning of the overlying cortical bone and little periosteal new bone proliferation. In kittens, the cyst may become more diaphyseal as the bone lengthens. Computed tomography and MRI are other imaging modalities that are routinely used in human medicine and can help distinguish benign solitary cysts, from aneurysmal bone cysts, telangiectatic osteosarcomas and other bone tumours.

Analysis of the fluid in the cystic cavity can reveal relative elevation of ALP and ACP (acid phosphatase) when compared to serum levels and this test is used in human medicine to diagnose bone cysts.

Treatment is not necessary if the cysts are subclinical, and spontaneous resolution occurs in some cases. In humans and dogs, a more aggressive approach such as surgical curettage and autogenous bone grafting have been used with good outcomes. However, complications may arise such as iatrogenic fracture, infection, growth plate injury, long-term immobilization, etc in humans so other techniques have been proposed such as drainage and local steroid therapy, bone marrow injections or even the packing of the cyst using hydroxyapatite or bone cement. In particular, intracavitary steroid injections have been reported to provide up to 90% success rate in humans.

This treatment method was used in a cat with a bone cyst in the distal ulna, showing increased ALP activity, with complete resolution with a follow-up of 14 months. Another recent report describes the use bone cement for treatment of a proximal humeral bone cyst in a 3-year-old Norwegian forest cat, which then developed malignant malformation into an osteosarcoma 17 months later.

Aneurysmal Bone Cyst

(Barnhart 2002, Benamou et al. 2012, Berger et al. 2016, Biller et al. 1987, Liu et al. 1974).

An aneurysmal bone cyst is a benign, vascular lesion of unknown etiology that results in considerable local bone destruction. They are typically blood-filled sponge-like lesions partially or completely destroying the parent cortical bone by expansion. They affect mainly the metaphyseal region of long bones but flat bones can also be affected. Their pathogenesis is not completely understood, but they may be initiated by an insult to the bone that disrupts bone marrow vasculature leading to arteriovenous shunting and bony reaction that forms a blood-filled cavity. For possible causative factors, an underlying neoplasia, trauma and fibrous bony dysplasia have been considered.

In one study, three cats each had a solitary lesion arising from the axial skeleton (sacrum, caudal vertebrae and pelvis). Two of the cats were 2 years old and the other was geriatric. Other bones, such as scapula and rib, have also been described as affected by this condition. Malignant transformation of an aneurysmal bone cyst into a chondrosarcoma has been described in a dog. Malignant transformation of a possible aneurysmal bone cyst has also been described in a cat.

Clinical signs typically would include lameness, swelling over the cysts and tenderness on palpation. Pain is caused by compression of soft tissues and periosteum, joint involvement, bone lysis or pathologic fracture. Radiography revealed a large expansile lesion with minimal soap bubble-like trabecular septation surrounded by a thin rim of mineralized soft tissue or bone. Differential diagnoses include primary and secondary bone tumours and unicameral bone cysts. Treatment by curettage and bone grafting, en bloc resection, or amputation may be successful, depending on the location of the lesion.

Fibrous Dysplasia

(Liu et al. 1974).

Fibrous dysplasia is a rare developmental abnormality of bone characterized by the formation of fibro-osseous cystic lesions. There is confusion in the older veterinary literature where the terms fibrous dysplasia, aneurysmal bone cyst and benign bone cyst are sometimes used synonymously. The lesion was reported long ago in two cats; one in the mandible and the other in the distal ulna, with signs of inability to open the jaw and lameness respectively. Radiographically the affected bone had a marked homogenous increase in radiopacity. The lesion in the ulna showed thinning of the cortices and irregular periosteal new bone formation. The lesions were resected from both cats and there was no recurrence 4 years later.

This condition has not been further reported in the cat.

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