

Musculoskeletal Diseases

Greg L.G. Harasen and Susan E. Little

OUTLINE

Fractures, 704
 Arthritides, 709
 Conditions of the Front Limb, 717
 Conditions of the Hind Limb, 718
 Neoplasia, 724

Osteomyelitis, 725
 Dysostoses, 725
 Miscellaneous Musculoskeletal Conditions, 726
 Myopathies, 728

Conditions of the musculoskeletal system in the cat have received comparatively little attention in the literature. Much of what has been published assumes similarities with dogs and humans that may not be accurate. Developmental diseases, especially genetically determined conditions, are much less common in the cat than in the dog. Even those that are seen, including patellar luxation and hip dysplasia, are relatively uncommon. Trauma is the major source of musculoskeletal abnormality in the cat, and thus the entire patient must be evaluated.

The feline patient also presents challenges in examination and observation of abnormal gaits because many cats are uncooperative at best and fractious at worst when exposed to the clinic environment. To appreciate subtle gait disturbances, the clinician must frequently rely on patience when examining the cat, as well as on owner observations or videotapes taken in the home environment.

The cat has a number of anatomic and physiologic differences compared with the dog. Some are mere curiosities, whereas others can be extremely significant from a diagnostic perspective.⁹⁶ The presence of a free-floating clavicle in the cranial shoulder region falls into the category of curiosity, but it is sometimes mistaken for a fracture or foreign body (Figure 26-1). The median nerve and brachial artery pass through the supracondylar foramen on the medial side of the distal humerus in the cat, whereas the same structures lie medial to the humerus in the dog (Figure 26-2). The presence of these vital structures within the humeral metaphysis of the cat restricts the placement of orthopedic hardware in this region. In the condylar region of the distal humerus,

there is no suprartrochlear foramen in the cat as there is in the dog. This is one of the main reasons that humeral condylar fractures are relatively less common in cats.⁶⁵ Approximately 40% of cats have a sesamoid bone in the tendon of origin of the supinator muscle on the dorsal surface of the proximal radius (Figure 26-3). This structure may be visible on lateral radiographic projections of the elbow and should not be mistaken for a chip fracture. The round ligament of the femoral head provides significant vascular supply to the femoral head in the cat, which is not the case in the dog. This may be one reason that aseptic necrosis of the femoral head is not described in the cat. The cranial cruciate ligament is larger and thicker than the caudal cruciate ligament in cats, which is the reverse of what is found in the dog. This may be an important factor explaining why rupture of the cruciate ligament is much less common in the cat. The range of motion in the feline shoulder and hip is greater than in the dog, but in the feline carpus and stifle range of motion is less than in the dog. However, supination of the carpus and paw is much greater in the cat and is important in grooming behavior.^{13,65,96,103}

FRACTURES

Fractures make up a large percentage of musculoskeletal problems in the cat, with the distribution of fractures being somewhat unique to this species. Although both dogs and cats suffer a majority of their fractures in the hind limb or pelvis, this percentage exceeds 70% of all fractures seen in the cat.⁴⁸ When the 11% to 23% of feline



FIGURE 26-1 The clavicle is located cranial to the proximal humerus.



FIGURE 26-3 Approximately 40% of cats have a sesamoid bone located in the origin of the supinator muscle. It can be seen on a lateral radiographic projection of the elbow.

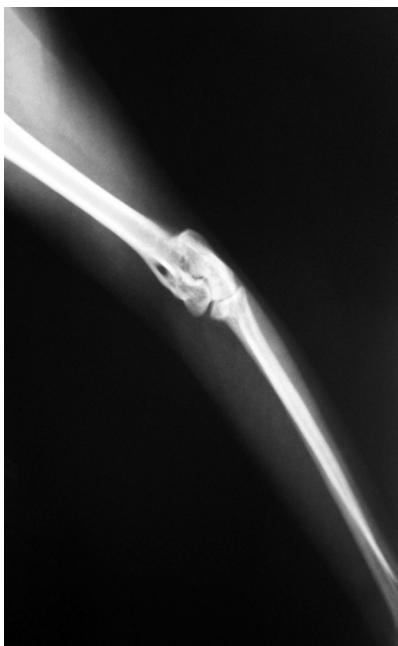


FIGURE 26-2 The supracondylar foramen is a unique anatomic feature of the medial aspect of the distal humerus in the cat through which pass the brachial artery and median nerve.

fractures that involve the maxillae, mandible, or facial bones are included, these two regions account for the overwhelming majority of fractures in the cat.

Because most fractures are associated with significant trauma, a thorough evaluation of the entire cat beyond the fracture is essential. Published estimates suggest that as many as 40% of fracture patients also have thoracic

trauma, which may affect not only the treatment plan but also the patient's very survival.¹³

Much commonality exists between dogs and cats in the fractures that are seen and the repair techniques that can be used successfully. Certain fractures with unique considerations in the cat bear special consideration.

Mandibular and Maxillary Fractures

Fractures involving the mandible or maxillae of cats are unique if for no other reason than that they are at least tenfold more common in the cat than in the dog.⁸⁶ Vehicular trauma and high-rise syndrome are the most common causes of these fractures, which occur when the cat absorbs a face-first impact. Not surprisingly, such trauma is frequently associated with additional injuries, including broken teeth, thoracic injury, head trauma, and forelimb fractures. Fracture of the mandibular symphysis accounts for nearly three quarters of mandibular and maxillary injuries.^{47,86} Circumferential wiring of the symphysis combined with 3 to 4 weeks of a soft diet followed by wire removal is usually successful (Figure 26-4). At the time of wire removal, some residual mobility may still be present at the symphysis, but this is due in part to the fact that the joint is cartilaginous and is not rigid, even in the normal state. Most patients do well clinically, regardless of the mobility.

Midsagittal splits of the hard palate are another common consequence of frontal facial impact trauma in the cat. Minor splits of 1 to 2 mm usually require no specific repair; however, wider splits should be compressed. This can be accomplished by running surgical wire in a figure-of-eight pattern across the split along the oral surface of the hard palate and around the base of a tooth on each side of the maxillae. Alternatively, a Kirschner wire can be driven across the maxillae between teeth so that the ends of the wire are exposed through the gingiva on either side of the maxillae, just dorsal to

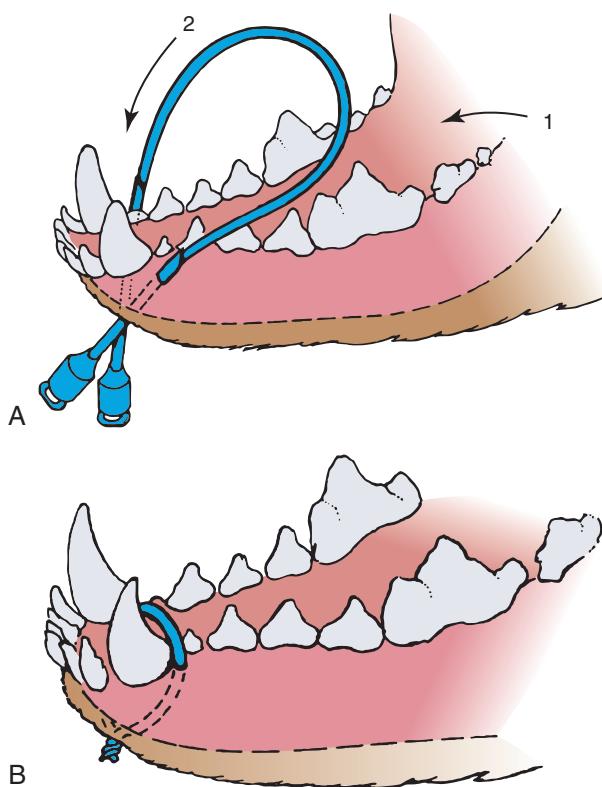


FIGURE 26-4 Mandibular symphyseal fractures can be stabilized with a loop of surgical wire passed around the cranial mandible, caudal to the canine teeth. (Reprinted with permission from Piermattei D, Flo G, DeCamp C: Brinker, Piermattei, and Flo's handbook of small animal orthopedics and fracture repair, ed 4, St Louis, 2006, Saunders Elsevier.)

the level of the hard palate. A figure-of-eight tension band wire is then placed around each end of the wire and tightened to achieve compression of the palatine split. The Kirschner wire ends can be bent over to prevent trauma to the lips. The hardware is removed after 4 weeks.⁴⁷

Treatment of more complex mandibular or maxillary fractures can involve many of the same techniques used in dogs, including interdental wiring, intraoral splints, bone plates, and external skeletal fixators. Regardless of the technique, the primary goal is to restore perfect dental occlusion. It bears remembering that the most beautiful surgical repair is of little consequence if the teeth do not fit. An adhesive tape muzzle can be used effectively in the cat despite its short, conical muzzle. A length of tape is wrapped around the muzzle with the adhesive side facing out, caudal to the level of the canine teeth. Care is taken to ensure that the canine teeth interdigitate properly but that there is enough space between the incisor teeth to allow the cat to lap liquid diets and water. Performing the procedure under general anesthesia with an endotracheal tube in place usually provides the proper amount of space. A length of tape is then passed behind the ears and stuck to the adhesive surface

of the muzzle wrap on the each side of the muzzle. Another wrap of tape is placed around the muzzle to hold the second length of tape in place. A final length of tape is placed from the head strap on both sides and beneath the cat's throat to prevent the head strap from pulling up over the ears.^{47,96} In cases in which fracture comminution precludes surgical reconstruction and stabilization, when financial constraints eliminate a surgical option, or in young kittens whose soft bone and erupting teeth make the use of surgical hardware difficult, a tape muzzle permits the maintenance of normal dental occlusion and often produces surprisingly good results.^{47,86,96}

Patellar Fractures

Traumatic fractures of the patella can occur in cats. If fracture fragments are sufficiently large, they can be stabilized with a pin and tension band wire. Small fragments may be removed. In either case the integrity of the quadriceps muscle and patellar tendon mechanism must be maintained.⁵³ Some cats may be born with bipartite patellas. The radiographic appearance reveals smooth edges to the patellar fragments and frequently a similar appearance bilaterally. The condition is usually an incidental finding but can cause diagnostic confusion in a lame cat.

An additional subset of young adult cats develops fractures of one or both patellae with no history or evidence of trauma. Lameness is acute but usually mild to moderate. Although the etiology is unclear, these have been characterized as stress fractures.⁶² Evidence for this pathogenesis includes the lack of known trauma in most cases and the presence of radiographic sclerosis of the fracture fragments and often the contralateral patella if it is intact. The fractures are simple transverse, involve the proximal one third of the patella, and are bilateral about half the time. In one survey about half of the contralateral patellae subsequently fractured at a mean time of 3 months.⁶² Attempts at surgical repair by pin and tension band wire have met with almost universal failure, characterized by iatrogenic fracture of the remaining fragments; hardware failure; or, most often, nonunion. However, most cats regained reasonable function of the limb, with stiffness or intermittent lameness in about half the cases.⁶² Some of the cats were found to have retained deciduous teeth or delayed dental eruption (Figure 26-5, A). In humans there is a connection between dentinogenesis imperfecta, which involves a number of dental abnormalities, and osteogenesis imperfecta, a condition involving brittle, easily fractured bones that is also seen in cats (discussed later).

Of 34 cats with apparently atraumatic patellar fractures, 10 also had a history of previous, concurrent, or subsequent fracture of other bones (see Figure 26-5, B).⁶² It may be that some of these cats with patellar fractures

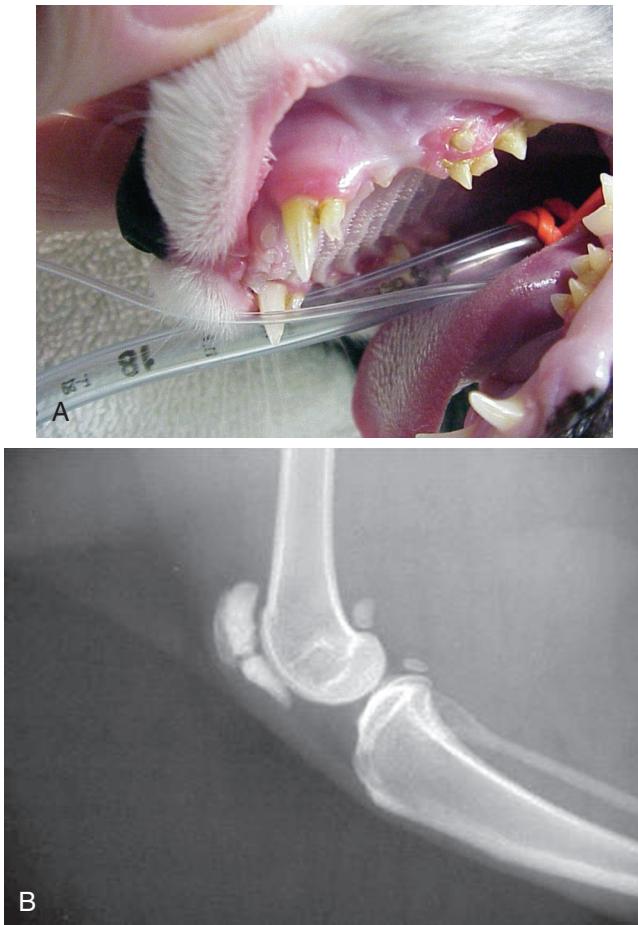


FIGURE 26-5 **A**, Persistent deciduous teeth are evidence of dentinogenesis imperfecta in this cat with a patellar fracture. **B**, A patellar fracture in a cat without history of trauma. (A courtesy Dr. Steven Bailey.)

have a form of osteogenesis imperfecta. Conservative treatment of these patellar fractures would appear to be the most prudent course, especially if distraction of fracture fragments is mild to moderate. If fragments are significantly distracted, then a circumferential wire may be preferred over an attempt to pass a pin through sclerotic bone. Alternatively, partial patellectomy may be performed. Complete patellectomy does not usually produce satisfactory function.⁶²

Radial and Ulnar Fractures

Fractures of the radius and ulna are relatively uncommon in cats, composing between 5% and 13.8% of feline fractures.⁸⁴ Further, surgical repair of these fractures, especially when comminuted or open, is associated with a high complication rate.¹⁰³ This would appear to be primarily due to the cat's ability to pronate the front limb and paw to a much greater degree than the dog. This increased mobility between the bones means that the standard surgical approach in the dog of stabilizing only the radius, when both bones are fractured, may not



FIGURE 26-6 Unilateral slipped capital femoral epiphysis.

confer enough stability to produce consistently good results in the cat. Adding an intramedullary pin to the ulna in addition to the radial repair has been associated with more reliable surgical outcomes in this species.¹⁰³ In addition, the ulna, especially its proximal portion, has been identified as a common site of nonunion in the cat, which may also be a significant contributory factor to surgical complication statistics for these fractures.⁸⁴

Capital Femoral Physeal Fractures

Capital femoral physeal fracture is a common traumatic injury in cats, but it appears as though just as many cases arise without a traumatic episode (Figure 26-6). Affected cats usually present with acute hind limb lameness, although the lameness may be mild and chronic in some instances. Most of these cases are seen in overweight, neutered males between 4 and 24 months of age.^{11,19,45,79} One report found a preponderance of domestic shorthairs,⁷⁹ whereas another found a large number of Siamese cats.¹⁹

The first report of this problem described it as metaphyseal osteopathy of the femoral neck, which was thought to result from an aseptic necrosis, not unlike Legg-Calve-Perthes disease in the dog.⁸⁸ However, further examination of serial radiography and histopathologic specimens suggests that the changes in the femoral neck are more likely to be resorptive and remodeling changes secondary to the Salter-Harris I physeal fracture rather than a vascular impairment as is seen in Legg-Calve-Perthes disease in the dog (Figure 26-7).^{19,45,79} The etiopathogenesis of this condition seems to revolve around abnormalities of the physis. Radiographically and histologically, these cats have abnormally wide physes that remain open long after they would be expected to have closed.



FIGURE 26-7 An acute slipped capital femoral epiphysis on the right side of the figure and a chronic example of the same injury on the left side. Note the resorption of the femoral neck and extensive remodeling.



FIGURE 26-8 A kitten with multiple pelvic fractures. Note the significant decrease in pelvic canal diameter.

Histologically, these physes are characterized by an irregular arrangement of chondrocytes rather than the normal columnar appearance, which has resulted in the use of the term *physeal dysplasia* to describe the process.

Although genetic factors may certainly be involved in the development of the physeal dysplasia, endocrine factors may also play a role. Neutering at an early age has been shown to delay physeal closure times in the cat,⁵³ and it has been suggested that neutering before 6 months of age may be the endocrine factor contributing to the physeal dysplasia and slipped femoral capital physis.⁷⁰ A similar syndrome occurs in young, overweight adolescent humans, especially those that are hypothyroid, are receiving growth hormone supplementation, or have hypogonadism.⁷² It may be that early neutering is one factor in the development of a physeal dysplasia, a wide physis, and a physis that remains open longer than normal, particularly in individuals that may be predisposed. If the cat becomes overweight, the stresses on the abnormal capital femoral physis may cause it to "slip," producing the characteristic Salter-Harris I fracture. Published reports suggest that between 24% and 38% of affected cats will develop bilateral fractures.^{19,45,79,88} The condition is best treated with femoral head and neck excision (FHNE), which will produce a return to normal function in the majority of cases.^{45,79} Primary repair with Kirschner wires has been described^{20,29} but has much greater potential for complications than FHNE, with few, if any, demonstrated advantages.

Pelvic Fractures

Pelvic fractures are extremely common in the cat, especially after vehicular trauma, and make up at least 22% of all fractures seen (Figure 26-8).⁶⁴ Most pelvic fractures are multiple, unstable, and displaced, at least to some degree. Regardless, most will heal with conservative therapy. However, the issue is not whether they will heal but rather the severity and consequences of the malunion that almost invariably results. Although surgery can be considered to hasten pain relief and return to function, in most practical applications there are two primary indications for surgery:

Displaced acetabular fractures: Approximately 17.5% of feline pelvic fractures involve the acetabulum. Any degree of fracture malunion in the coxofemoral joint will lead to degenerative joint disease (DJD) and pain. Such a fracture can be addressed near the time of the initial trauma by primary fixation methods that include plates, screws, and tension band wires.⁶⁴ Traditionally, caudal acetabular fractures have often been treated conservatively because this area of the acetabulum was not considered to be weight bearing. However, recent research has suggested that the central and caudal portions of the acetabulum are actually the major weight-bearing regions within the coxofemoral joint of the cat.⁴ Alternatively, femoral head and neck ostectomy can be performed several days or weeks later, once the patient's condition has stabilized, and where there is evidence of ongoing disability.

Pelvic canal narrowing: Ilial and acetabular fractures commonly displace axially, producing a narrowing of the pelvic canal. This can have immediate traumatic

effects on bladder and bowels, but the greater concern is the prospect of producing obstipation and megacolon in the longer term. These problems are extremely frustrating to treat and are much better prevented. Thus pelvic fractures that produce more than 25% to 30% narrowing of the pelvic canal are best treated surgically. This can be efficiently done, in most cases utilizing bone plates, within 5 days of the initial trauma. After that time it becomes more difficult to break down fibrous tissue and surgically reduce these fractures. Pelvic canal-widening procedures such as pelvic symphyseal osteotomy can be performed if constipation has been present for less than 6 months. If constipation has been present for more than 6 months, the colon is often beyond reclamation and subtotal colectomy should be performed.¹⁷

Nonunion Fractures

Cats may be characterized as the “perfect orthopedic patient” in many ways because their straight bones, lightweight frames, and legendary healing abilities have resulted in many amazing outcomes in fracture cases. However, the old adage that fractured feline bones will heal if placed in the same room is not always true. Feline fracture nonunions do occur, at a rate of 4.3% according to one report.⁸⁴ The tibia and proximal ulna were identified as the most common sites for nonunions. Increasing age and body weight, as well as open and comminuted fractures, were identified as risk factors.⁸⁴

ARTHRIDITIES

Degenerative Joint Disease

The slowly progressive degeneration of articular cartilage with osteophyte production, usually associated with acute or chronic joint trauma, is the most common form of joint disease seen in the cat and is variously described as DJD, osteoarthritis, or osteoarthrosis. It is only relatively recently that the common occurrence of DJD has been recognized in the cat. Knowledge about DJD in cats—prevalence, impact on lifestyle, efficacy of therapy—is less well developed than for the dog. Because cats have a small body size and are light and agile, they compensate for orthopedic diseases better than dogs. Cats are also notorious for hiding signs of illness, especially if onset is insidious, and it is more difficult to interpret signs of pain or discomfort in this species.¹⁰⁰

Twenty-two percent of cats in a general population older than 1 year of age³⁷ and 90% of cats older 12 years of age⁵¹ were found to have radiographic evidence of DJD. The elbow was the most frequently affected joint in the older population. The coxofemoral joint may also be affected, and most cats have bilateral involvement.¹⁵ Relatively few of these cats had clinical signs associated

BOX 26-1

Clinical Signs Associated with Degenerative Joint Disease in the Cat^{3,15,37,50}

1. Pain
2. Reduced activity, difficulty with jumping or stairs
3. Anorexia, weight loss
4. Irritability, aggression
5. Inappropriate urination, constipation
6. Decreased grooming
7. Lameness or stiff gait
8. Alopecia over affected joints

BOX 26-2

Physical Examination Findings Associated with Degenerative Joint Disease in Cats³

1. Pain on joint manipulation
2. Soft tissue swelling
3. Periarticular thickening
4. Joint effusion
5. Restricted range of movement
6. Muscle atrophy
7. Crepitus
8. Heat

with the radiographic findings, or, perhaps more accurately, clinical signs were infrequently recognized by owners and veterinarians.¹⁶ This may be because the most common clinical sign associated with DJD in the dog is lameness. Owing to the cat’s lightweight frame and behavioral differences, it appears as though there may be other, more significant clinical signs of DJD that need to be recognized (Box 26-1). Physical examination findings for DJD in cats are also different than those for dogs (Box 26-2).

It is assumed that the etiopathogenesis of DJD is the same for cats and dogs, although little evidence currently exists to support this assumption. Suspected causes include primary degeneration (wear and tear), joint dysplasia, joint injury, fractures, luxations and dislocations, congenital malformations, cranial cruciate ligament rupture, infection, and neoplasia. The clinical presentation of DJD in cats is different from dogs, and the radiographic signs differ slightly. Joint injuries are less common in cats than dogs, but hip dysplasia is probably underestimated in cats.

The hallmark of DJD is the progressive and permanent damage of articular cartilage.⁸² Injury of the chondrocytes leads to the production of inflammatory mediators such as cytokines (especially interleukin-1 [IL-1]) and tumor necrosis factor-alpha. IL-1 stimulates

production of degradative enzymes, inhibits production of proteoglycans, and stimulates fibroplasia of the joint capsule. The thickened joint capsule contributes to stiffness and decreased range of movement.

The degradative enzymes set in motion a process that damages collagen and causes it to swell. The abnormal cartilage cannot bear loads normally, causing increased load on certain areas of the joint and leading to further cartilage damage. The underlying subchondral bone is stressed, and pain receptors are stimulated. Osteophytes are bony proliferations formed at the conjunction of the synovium, perichondrium, and periosteum. They are believed to be caused by mechanical instability of the joint and joint inflammation. They may contribute to joint pain.

Even though the clinical signs of DJD may wax and wane, the changes to the joint are permanent, with limited ability to repair the articular surface or joint capsule. The vicious cycle of inflammation, degeneration, and mechanical dysfunction leads to progressive disease.

The diagnostic approach to joint disease in cats is similar to that in dogs. A medical history, physical examination, and radiographs are most commonly employed. Further diagnostic steps might include joint fluid analysis and culture, arthroscopy, myelography, or advanced imaging such as magnetic resonance imaging (MRI) or computed tomography (CT).

When taking a medical history, especially for senior cats, the veterinarian should focus the questions on changes in activity and behavior rather than solely on lameness. Many signs of chronic pain are not obvious to owners or may be misinterpreted as due to aging.¹⁰⁰ The degree of impairment caused by chronic pain may not be apparent to some owners until improvements occur after treatment.

Unfortunately, gait analysis is rarely helpful in cats, and orthopedic examinations are limited by the lack of data on normal ranges of motion for feline joints and the difficulty of detecting small changes associated with joint disease. In the examination room it may be possible to evaluate gait by allowing the cat to walk around the room. In addition, the cat can be encouraged to jump off a chair or jump up to get into its carrier. Cats with lumbosacral joint disease may be reluctant to jump and may exhibit signs of being in pain when the lower back is petted or examined. Cats with hip dysplasia may have no clinical signs at all, although cats with more advanced disease may have lameness and pain.

Radiographic signs of DJD in cats are variable.^{1,16,36} Radiographs are best at demonstrating bony changes, and changes in the cartilage and synovium are not well demonstrated on plain radiographs. Joint effusions and joint capsule thickening are rarely evident. Typical bony changes include osteophyte development, subchondral sclerosis, perichondral bone erosion, and change in

congruity of articular surfaces. Soft tissue swelling around the joint may be present. Lumbosacral DJD may be indicated by collapse of the L7-S1 disk space, sclerosis of the L7-S1 endplates, and spondylosis deformans.

Infectious Arthritides

Bacterial

Septic bacterial arthritis is most commonly associated with bite wounds sustained in cat fights. Hematogenous spread of bacteria from other sites in the body appears to be relatively rare in the cat, although it may occasionally be seen in kittens. Septic arthritis may develop secondary to orthopedic surgical procedures. Clinical signs include pain and swelling of the affected joint. Pyrexia and leukocytosis are usual but not invariable.⁵⁶ Radiographic signs in the early stages will be confined to joint effusion and soft tissue swelling. As the condition progresses, and depending on the infective organism, there may be evidence of a periosteal reaction, bony sclerosis, and varying degrees of bone lysis at the periosteum and in the subchondral bone. Diagnosis is based on clinical signs and the results of arthrocentesis. Cytology, as well as aerobic and anaerobic culture combined with bacterial sensitivity testing, is essential, although a negative culture result is not uncommon. Therapy with bactericidal antibiotics (based on sensitivity testing) is indicated for 4 to 6 weeks. In the absence of a positive culture, while awaiting results, or where empirical treatment is desired, cephalosporin or amoxicillin-clavulanate antibiotics are reasonable choices, with metronidazole a useful addition in confirmed or suspected anaerobic infections.⁵⁶ All of these antibiotic choices, and others besides, commonly cause vomiting or inappetence in cats, which may necessitate changes in therapy. Surgical drainage or flushing of infected joints is rarely necessary except in the most severe cases. Analgesics and other supportive care may be indicated.

Although up to 15% of cats have been found to be seropositive for *Borrelia burgdorferi*, they seem to be resistant to clinical disease, and no reports of arthritis associated with Lyme disease have been documented in this species.⁵⁶

Mycoplasma

Rare cases of polyarthritis and tenosynovitis associated with *Mycoplasma gateae* and *Mycoplasma felis* have been reported in the literature.^{56,107} Hematogenous spread from areas of active or latent infection in respiratory mucous membranes or the urogenital tract, most often in otherwise debilitated or immunocompromised individuals, appears to be the pathogenesis of the arthritis. There is potential for diagnostic confusion with immune-mediated arthritides because *Mycoplasma* arthritis may appear similar. Radiographically, there is the potential for erosive lesions. Synovial fluid analysis and a

negative aerobic culture may suggest immune-mediated arthritis. The organisms may be detected on a synovial fluid smear stained with Wright, Leishman, or Giemsa stains, or it may be grown on anaerobic culture from synovial fluid or synovium.^{56,107} The clinician should have an index of suspicion for *Mycoplasma* arthritis when dealing with debilitated individuals that appear to have immune-mediated arthritis. Tylosin, erythromycin, and gentamicin have traditionally been the most recommended therapies. However, fluoroquinolones represent a more recent, readily available alternative that is effective, convenient, and safe in cats.¹⁰⁷

Viral

A short-term, self-limiting polyarthritis associated with calicivirus has been described in kittens younger than 6 months of age.^{7,21} The arthritis may be caused by infective intraarticular live virus or by the deposition of immune complexes within the synovium. The condition may be seen in association with the typical respiratory infection, or it may be seen 5 to 7 days after vaccination with modified-live calicivirus vaccine. Vaccine-associated arthritis is now uncommon because vaccine manufacturers have for the most part discontinued the use of virus strains associated with the problem. Diagnosis is made largely on the basis of history and clinical signs, and therapy is supportive, including analgesia, because the condition is self-limiting.^{7,21}

Autoimmune Arthritides

Autoimmune arthritides are characterized in the first instance by inflammation of the synovium. They may be subdivided as "erosive" when there are deforming, lytic lesions to cartilage and subchondral bone, and "non-erosive" when no such lesions are found and inflammation is confined to the synovium. Arthrocentesis produces similar results in most cases. Increased amounts of a watery, turbid joint fluid with a poor mucin clot are commonly encountered. White cell counts in the joint fluid are increased, and the majority of cells are nondegenerate polymorphonuclear cells. Bacteria are not seen, and culture is negative. All types are similar clinically, presenting with stiffness and swollen, painful joints and typically resulting in an irritable cat. Some cases may have fever and inappetence or anorexia.

Erosive

PERIOSTEAL PROLIFERATIVE POLYARTHRITIS

Periosteal proliferative polyarthritis (PPP) is the most common erosive form of arthritis seen in the cat. It most often affects the hocks and carpi of young adult male cats. Clinical signs begin acutely with fever, depression, stiffness, joint effusion, and pain. Over the course of a few weeks, the disease enters a chronic phase in which extensive periosteal new bone forms around the hocks

BOX 26-3

Criteria Proposed for the Diagnosis of Periosteal Proliferative Polyarthritis⁷

1. Erosive polyarthritis
2. Periosteal new bone formation at affected joints
3. Negative for rheumatoid factor in the blood
4. Enthesopathies
5. Primarily the hocks and carpi are involved

NOTE: The first three criteria must be met to make the diagnosis; however, the last two are variable.

and carpi and at the attachments of ligaments and tendons. New bone production may be extensive enough to produce ankylosis of joints. Erosive lesions may also be seen in subchondral bone and at tendinous attachments.^{21,56} Erosive lesions or periosteal new bone formation at attachment points of ligaments, tendons, or fascia are referred to as *enthesopathies*.⁷ The criteria proposed by Bennett and Nash for diagnosing PPP are found in Box 26-3.⁷

An etiologic link has been proposed between feline syncytium-forming virus (FeSFV) and PPP insofar as all cats with PPP appear to have FeSFV. However, attempts to experimentally induce PPP by inoculating cats with the virus have failed, and FeSFV has been found as a normal inhabitant in the joints of many asymptomatic cats.²¹ A role for feline leukemia virus (FeLV) and feline immunodeficiency virus (FIV) has also been proposed in producing immunosuppression that allows proliferation of FeSFV; however, FeLV and FIV are frequently not found in cats with PPP.^{5,21} Occasionally symptoms of immune-mediated arthritis may be found in cats immunocompromised for other reasons, including chemotherapy and hyperadrenocorticism.

PPP bears some similarities to Reiter's disease, which is seen most commonly in men. Urethritis and diarrhea can be seen, in addition to the arthritic lesions in humans, and there is at least one report of hematuria associated with polyarthritis in a cat.⁵ Conjunctivitis and lesions of skin and mucous membranes, which are common in humans, have been found in some cats. In light of these types of lesions, a link between Reiter's disease and *Chlamydia* infection has been explored, and the organism has been implicated in humans.⁷ The prognosis for cats with PPP is guarded to poor. Few will experience anything more than marginal improvement on therapy, and many will end up being euthanized. However, decisions of that sort should be based on assessments of the patient's function and comfort level.

RHEUMATOID ARTHRITIS

By all accounts, rheumatoid arthritis (RA) is much less common in the cat than in the dog. RA is a synovitis

BOX 26-4**Diagnostic Criteria for Establishing a Diagnosis of Rheumatoid Arthritis in the Cat⁷**

1. Stiffness after rest
2. Pain on manipulation of at least one joint
3. Swelling of at least one joint
4. Swelling of at least one other joint within a 2-month period
5. Symmetric joint swelling
6. Subcutaneous nodules
7. Erosive radiographic changes
8. Positive rheumatoid factor blood test
9. Abnormal synovial fluid (poor mucin clot, predominance of polymorphonuclear cells)
10. Characteristic histologic changes in the synovium (villous hypertrophy)
11. Characteristic histologic changes in the subcutaneous nodules

Note: Criteria 1 to 5 must be present for at least 6 weeks, and at least two of criteria 7, 8, and 10 must be present to establish the diagnosis.

caused by the production of autoantibodies against immunoglobulin G (rheumatoid factor). The deposition of immune complexes within the synovium leads to an erosive, deforming arthritis. As with other immune-mediated arthritides, RA causes generalized stiffness, pain, and swelling of joints. In contrast to PPP and idiopathic immune-mediated arthritides, there is seldom malaise or inappetence, and the course tends to be more gradual in onset. Joint deformity to the point of subluxation and luxation is often a prominent feature. Bennett^{7,21} has described 11 diagnostic criteria, patterned after those used in man and the dog, for establishing a diagnosis of RA in the cat (Box 26-4). The criteria recognize the fact that a positive blood test for rheumatoid factor is not always present, and a positive test is not necessarily specific for the disease. The presence of subcutaneous nodules is common in humans but has not been described in the cat.⁷

One report of 12 cases of RA in the cat described an average age of 5.9 years, with Siamese cats overrepresented.⁴⁴ With aggressive forms of antirheumatic therapy, discussed at the end of this section, the prognosis for RA seems to be much better than PPP, with 58% showing a marked improvement.⁴⁴

Non-Erosive**SYSTEMIC LUPUS ERYTHEMATOSUS**

Systemic lupus erythematosus (SLE) is an uncommon cause of arthritis in cats. It is characterized by multisystemic involvement with polyarthritis and usually one of the following: autoimmune hematologic disease (thrombocytopenia, hemolytic anemia, and leukopenia),

BOX 26-5**Idiopathic Polyarthritis Subgroups⁷**

Type I: idiopathic

Type II: idiopathic associated with other infections.

These may be found in the respiratory or urogenital tracts, skin, or oral cavity.

Type III: idiopathic associated with gastrointestinal disease

Type IV: idiopathic associated with neoplasia. In the cat this is most often myeloproliferative neoplasia, which may be FeLV or FIV.

dermatitis, glomerulonephritis, or meningitis.⁷ There are no destructive or deforming lesions on radiography or gross examination. The disease results from an autoimmune reaction to nucleic acid, which manifests as the hemolytic anemia, leukopenia, or thrombocytopenia. Deposition of the resulting immune complexes produces synovitis, glomerulonephritis, meningitis, dermatitis, or occasionally polymyositis. Onset of symptoms is usually acute and includes pain, especially on handling, and joint swelling.

Clinical pathology is essential to diagnose SLE. Anti-nuclear antibody (ANA) blood tests are invariably positive at a level of 1:40 or greater in the cat.⁷ Although a diagnosis of SLE cannot be established without such a positive ANA value, the test is not specific and may be elevated in the acute phase of other disease conditions, including FeLV, FIV, and feline infectious peritonitis (FIP). Moreover, lower titers of ANA can be found in otherwise healthy cats.⁷ Hematologic abnormalities (anemia, thrombocytopenia, and leukopenia) are common, as is proteinuria.⁷ The diagnostic criteria to establish SLE are as follows:

1. Multisystem involvement, which most often includes polyarthritis and one other body system
2. ANA titre of 1:40 or greater
3. Presence of antibodies to blood cells or immune complexes in histopathology of affected tissues

Criteria 1 and 2 are essential for the diagnosis, which cannot be made solely on the basis of an elevated ANA titer.⁷ Reports of treatment for SLE in cats are rare in the literature, but the prognosis appears to be guarded.

IDIOPATHIC POLYARTHRITIS

Polyarthridities that do not fit into any of the other classifications end up in the idiopathic category. There are four subgroups of idiopathic polyarthritis (Box 26-5).⁷

Idiopathic arthritis may present at any age, but most affected cats are young adults that develop symptoms acutely or subacutely. The pathogenesis is undoubtedly a synovitis arising from the deposition of immune

complexes. Clinical signs are acute in all but type IV cases, which have a more chronic course. Most cases of all types exhibit signs of stiffness and pain, with occasional lameness. Joint and soft tissue swelling is usually present. These changes, along with the clinical signs, tend to be bilaterally symmetric. Pyrexia and inappetence are common. Type II idiopathic polyarthritis in the cat is most often associated with respiratory symptoms, including increased lung sounds, congestion, and conjunctivitis. Cats with type III frequently have diarrhea and dysentery, with occasional vomiting.⁷ Toxoplasmosis has been identified as one possible cause.⁷ Radiography is normal, except for joint effusion in some cases. The prognosis with treatment is generally good, especially in type I cases or in type II and III cases in which the underlying systemic illness can be identified and treated. Relapses, especially in types I and IV, are common.

Scottish Fold Arthropathy

Scottish Fold cats have an autosomal dominant trait that impairs enchondral ossification and produces abnormal cartilage maturation called *Scottish Fold arthropathy* (also known as *osteochondrodysplasia* and *osteodystrophy*). Heterozygotes for this trait have the characteristic “folded ear” appearance for the breed and may have mild signs of arthropathy. Homozygotes develop a progressive ankylosing arthropathy with radiographic lesions evident as early as 7 weeks of age. The condition is characterized by the production of new bone that bridges joints throughout the body but most prominently in the paws, spine, tail, and distal hind limbs (Figure 26-9). Affected individuals have a short, squat appearance that has been described as a form of dwarfism¹⁴ and experience decreased range of motion in affected joints. Clinical signs include stiffness, lameness, and inability to jump. The condition is progressive and can produce dramatic periarticular new bone, most prominently on the plantar aspect of the calcaneal–tarsal–metatarsal articulation. Exostoses in this area can produce ulceration of overlying skin. Surgical excision of the exostoses or radiation therapy have been used as successful palliative therapies for extended periods.^{14,54,78} Nonsteroidal anti-inflammatory drugs (NSAIDs), pentosan polysulfate, and oral glycosaminoglycans have been reported to provide symptomatic relief.^{14,74} One report has described the use of pantarsal arthrodesis to improve function.⁷⁸

Arthritis Therapy

It has been argued that no pharmaceutical therapy has a greater impact on the arthritic patient than weight control in the obese patient. The owner may also be able to make adjustments in the cat’s environment, such as placing litter boxes within easy access of the arthritic cat



FIGURE 26-9 Scottish Fold arthropathy. Note the ankylosing arthropathy characterized by new bone production in the hock, stifle, and vertebral (especially coccygeal) joints.

or making it easier for the cat to attain its customary perch in several small steps instead of one big jump.

Therapy for Degenerative Joint Disease

The goals of treatment for cats with DJD include reduction of pain and inflammation, improvement in joint function, and slowing the disease process, if possible. Treatments fall into four broad categories:

1. Weight loss
2. Drug therapy
3. Chondroprotectants
4. Nutraceuticals

Weight loss is indicated if the patient is overweight or obese and will reduce forces on the joint surfaces. Weight loss may also lead to decreased dose or frequency of drug administration.

Drug therapy is indicated to control inflammation, provide pain relief, and improve function. The development of several NSAIDs specifically for the small animal veterinary market is arguably one of the most significant therapeutic advances in veterinary medicine during the last 50 years. Despite this, relatively little information exists regarding the safety and efficacy of these drugs in the cat, and virtually nothing is known about the long-term use of these drugs in this species for chronic pain and inflammation, such as is seen in DJD. What is known is that the cat has a significantly decreased ability to metabolize most of these drugs through hepatic glucuronidation. The result is a much longer half-life for most NSAIDs in the cat and thus a greater potential for

accumulation and toxicity. Whereas gastrointestinal upset characterized by vomiting, inappetence, and diarrhea is the most common toxic effect, renal insufficiency is the most serious potential adverse reaction. Both of these adverse reactions arise from the antiprostaglandin effect of NSAIDs, specifically on the cyclooxygenase 1 (COX-1) isomer in the inflammatory pathway. Prostaglandins have a protective effect on gastric mucosa, presumably through increased secretion of mucus in the gastrointestinal tract. Inhibition of this secretion, combined with the acidic nature of most NSAIDs, produces gastric inflammation and ulceration. In the kidney, prostaglandins play a role in maintaining renal blood flow, particularly in the face of dehydration or hypotension. Inhibition of this effect has been associated with catastrophic renal failure in some cats.^{13,67,91} The potential for these adverse reactions undoubtedly explains some of the reluctance on the part of pharmaceutical manufacturers to pursue the use of NSAIDs in this species. Nevertheless, limited research has resulted in label recommendations in some parts of the world for feline use of carprofen, ketoprofen, tolafenamic acid, and meloxicam.^{13,43,67,80,91} Only meloxicam has a label for extended duration of administration in some countries, but cautious off-label use of various NSAIDs has been successful for much longer periods.^{43,91,99a}

Carprofen is labeled for use in the cat in the United Kingdom, Europe, Australia, and New Zealand; however, the indication is for one-time postsurgical subcutaneous administration at a dose of 4 mg/kg.^{67,80,91} No information exists on long-term use other than an individual report of toxicity after oral administration.⁶⁷ The drug has an extremely variable half-life (9 to 49 hours) in the cat, which increases the likelihood for toxicity with repeated use in some cats. Consequently, carprofen cannot be recommended as a safe option for management of DJD.

Ketoprofen is approved for use in the cat in Europe, Australia, and Canada at a dose of 1 mg/kg orally, once daily for up to 5 days, and 2 mg/kg subcutaneously, once daily for up to 3 days. Although the drug is eliminated by hepatic glucuronidation in the dog, the half-lives in both dogs and cats are similar. This, combined with other evidence, suggests the possibility of another excretory pathway for ketoprofen in the cat, which may imply a higher degree of safety; however, no long-term data corroborate this.⁶⁷ The drug is known to have a relatively greater activity against COX-1, which increases its inhibitory effect on platelet aggregation and may be significant in terms of renal or gastrointestinal side effects with long-term use.⁶⁷

Tolafenamic acid is licensed for use in cats in Canada, Australia, New Zealand, and most of Europe. The recommended dose is 4 mg/kg orally or subcutaneously, once daily for 3 to 5 days. The product is labeled for the treatment of fever and upper respiratory disease,

although its use as an analgesic and antiinflammatory is established in the literature.⁶⁷ Information provided by the manufacturer claimed no significant toxic effects when up to twice the recommended dose of the oral tablet or the injectable solution were given for up to 10 days to two groups of 12 cats. Increases characterized as "mild" were noted in the alanine transaminase (ALT) and aspartate aminotransferase (AST) of treated cats, and a positive test for fecal occult blood was noted in two cats from each group. There are anecdotal reports of DJD treatment in cats with tolafenamic acid for 3 to 5 consecutive days each week for extended periods, but little information in the literature supports this recommendation.

Most of what little information exists regarding the chronic use of NSAIDs for DJD in the cat concerns meloxicam. In contrast to the metabolism of most other NSAIDs in the cat, meloxicam is excreted by oxidative enzymes and thus has a similar half-life in both the cat and dog.⁶⁷ One recent study showed a COX-1-sparing effect for meloxicam in cats.³⁸ Consequently, the drug lends itself more readily to longer-term use in this species. In North America meloxicam is supplied in a 1.5 mg/mL honey-flavored oral formulation that is well tolerated by cats. In many countries it is supplied in a cat-specific formulation of 0.5 mg/mL. The same strength is available in the United States as a small dog formulation. It is labeled for feline use in the United States, Europe, Australia and New Zealand at a dose of 0.3 mg/kg for one-time subcutaneous use. Further, chronic use of the oral solution at 0.05 mg/kg per day is described on the package insert in many countries.⁹¹ Several other recommendations for oral dosing appear in the literature (Box 26-6).

Administration of 1 drop of the 1.5 mg/mL oral liquid preparation on the cat's daily food was tolerated without significant adverse effects compared with controls in 46 cats with a mean age of 12.9 years that were given the drug for a mean duration of 5.8 months.⁴³ Good to excellent results were reported by at least 80% of the owners and veterinarians surveyed.⁴³ Palatability was not reported to be a problem with meloxicam. The most commonly encountered side effect appears to be gastrointestinal upset.⁹¹

The currently available information, although far from overwhelming, would suggest that meloxicam is a viable option for long-term treatment of DJD in cats. Before starting NSAID therapy, a complete blood cell count, serum chemistries, and a urinalysis should be performed. Ideally, NSAIDs should be prescribed only to normotensive, normovolemic adult cats with no history of renal, hepatic, or gastrointestinal disease. However, for some cats the benefits of NSAID therapy outweigh the risks, insofar as the quality of life may be more important than the length of life. NSAIDs are not recommended in patients taking certain medications,

BOX 26-6**Published Doses for Meloxicam in the Cat**

Source	Dose
Label: US, Australia, New Zealand, EU	0.3 mg/kg subcutaneously, once
Label: Australia, New Zealand, EU	0.1 mg/kg on day one, followed by 0.05 mg/kg per day, orally
Wallace 2003, ^{103a} Carroll & Simonson 2005 ^{12a}	0.2 mg/kg on day 1, followed by 0.1 mg/kg/day for 2 days, then 0.025 mg/kg/day or 0.1 mg/cat 2-3 times/week
Lascelles 2007 ⁶⁷	0.1 mg/kg on day 1, followed by 0.05 mg/kg for 1 to 4 days, then reduction to the lowest effective dose (0.025 mg/kg every 24-48 hours)
Robertson 2008 ⁹¹	0.05 mg/kg given once, followed by 0.025 mg/kg or less once daily
Gunew 2008 ⁴³	0.1 mg/kg for four days, followed by 0.1 mg/cat once daily

such as diuretics and corticosteroids. Blood tests should be monitored periodically for cats on long-term therapy, and owners should be cautioned to monitor for vomiting, diarrhea, anorexia, increased thirst, increased urination, and lethargy. One author suggests re-evaluation every 8 to 12 weeks.⁹⁰

Renal values and urine protein levels should be monitored; hepatic sensitivity to NSAIDs seems to be primarily a canine problem.^{13,67,91} It may be helpful to recommend that cats taking NSAIDs be fed only or primarily canned foods because this increases water consumption by up to 50% and may help prevent subclinical dehydration in predisposed patients.

Glucocorticoids are controversial drugs for treatment of DJD. These drugs can reduce inflammation through various mechanisms, but chronic use has been found to delay healing and even damage cartilage.³ Furthermore, the potential adverse effects of glucocorticoids in cats are well known. Use of these drugs should be limited to cases in which all other therapies have failed and for short periods only.

Analgesics can be very useful in the treatment of feline DJD and may be added to other therapies. Commonly used analgesics include buprenorphine (0.01 to 0.03 mg/kg every 8 to 12 hours, by way of buccal mucosa), gabapentin (3 to 5 mg/kg orally, every 8 to 12 hours), and tramadol (2 mg/kg orally, every 12 hours).

Although the use of oral and injectable chondroprotectives is reported for arthritic conditions of various kinds in the cat,^{14,74} little objective evidence exists as to their effectiveness. Oral preparations include several combinations in which chondroitin sulfate and either hydrochloride or sulfate salts of glucosamine predominate. Little is known about the differences, if any, among various formulations; what dose rates and durations of therapy are best; and which patients, if any, will benefit. Virtually all of the currently available research has been done in the dog. Injectable chondroprotectants include polysulfated glycosaminoglycan (Adequan, Novartis)

and pentosan polysulfate (Cartrophen, Biopharm, Australia or Pentosan, Naturevet, Australia), which are labeled for use in the dog but have been used with apparent benefit and without adverse effect in the cat.^{14,78} Pentosan polysulfate is given at a dose of 3 mg/kg subcutaneously, once weekly for four consecutive treatments. Polysulfated glycosaminoglycan is given at a dose of 4 mg/kg intramuscularly, twice weekly for 4 weeks. Both chondroprotectants are given as needed after the first course of therapy. Oral or parenteral chondroprotectants have their beneficial effect in three primary ways:

1. By stimulating and enhancing the metabolism of chondrocytes and synoviocytes, including the provision of substrate for the production of cartilage matrix and synovial fluid
2. By inhibiting degradative enzymes and other entities, including prostaglandins, complement, and free radicals that play a role in the osteoarthritic destruction of the joint
3. By inhibiting the production of thrombi in the microvasculature of the synovium and subchondral bone, which have been shown to play a role in osteoarthritis⁴⁶

Omega-3 fatty acids have been shown to have an inhibitory effect on arachidonic acid, which is a key player in the inflammatory pathway in arthritic joints. Supplements of omega-3 fatty acids or diets containing these compounds have been used with apparent success in dogs. Diets for joint health are relatively new for cats. The first products on the market (Medi-Cal Royal Canin Mobility Support, Hill's Prescription Diet Feline j/d) contain omega-3 fatty acids as well as glucosamine and chondroitin sulfate.

Therapy for Immune-Mediated Arthritis

Prednisolone at immunosuppressive doses is the first-line treatment for immune-mediated arthritis in the cat.

Prednisone and prednisolone are interchangeable in the dog, but many cats are unable to adequately absorb prednisone and convert it to the active metabolite, prednisolone, within the liver.⁴⁰ The cat with immune-mediated arthritis should be started on a total daily dose of 2 to 4 mg/kg in two divided doses. This dose is continued for 2 weeks and then gradually tapered over a further 6 to 8 weeks.^{7,28,56,80} The short- and long-term side effects of corticosteroid therapy are well known, but most cats seem to be relatively more resistant to these effects compared with dogs or humans.

The determination of when to begin tapering the dose and how quickly that should be done is based on improvement of clinical signs and the results of follow-up joint taps starting at 2 weeks after the initiation of therapy. Most immune-mediated arthritides have synovial fluid profiles with high numbers of nondegenerate polymorphonuclear cells. Follow-up samples should show significant declines in absolute white cell counts (preferably below 4000/ μ L), but equally important is a shift to a predominantly mononuclear cell population. Results satisfying those criteria would suggest a favorable prognosis and would also be an indication to begin tapering the corticosteroid.^{7,56}

If, however, follow-up joint taps do not show favorable changes in cell numbers and populations, then the addition of a cytotoxic drug, most often cyclophosphamide in the cat, should be considered. Similarly, if clinical signs cannot be kept in remission or doses of prednisolone become high and side effects are problematic, cyclophosphamide should be added. Some have advised that both prednisolone and cyclophosphamide should be used together from the outset of therapy, especially in the erosive arthritides, for which the prognosis is much less favorable.⁷ Cyclophosphamide is given at a dose of 2.5 mg/kg orally, once daily for 4 consecutive days a week in combination with prednisolone at an antiinflammatory dose of 1 mg/kg orally, divided twice daily. This regimen should be continued for 4 weeks after remission of clinical signs.⁷

The introduction of cytotoxic drugs mandates a higher level of monitoring because of their potential for adverse effects. Hematuria is one such side effect and should be monitored with urinalysis every 2 weeks during therapy. Every 1 to 2 weeks, complete blood counts (CBCs) should also be done to detect any significant decrease in white cell or platelet counts. Neutrophil counts below 1000/ μ L or platelet counts below 50,000/ μ L should result in a decrease in cyclophosphamide dose by 25%. If neutropenia (<1000/ μ L) is found on the next CBC, a further reduction in drug dose is warranted.³²

Azathioprine, which is a popular cytotoxic drug in the dog, should not be used in cats because they seem particularly sensitive to its myelosuppressive effects. Tapering of medication is unique to every case of feline polyarthritis in that some will rapidly respond and can

be quickly tapered without recurrence. At the other end of the spectrum are cats that respond poorly and can be managed only with continuous therapy. Many variations of drug combinations and doses, as well as frequency and duration of treatment, have been used in the cat, so the clinician must find what works best in each case.

An alternative therapy for RA in the cat using methotrexate and leflunomide produced encouraging results in 12 cases.⁴⁴ Methotrexate inhibits inflammation and joint destruction through the promotion of adenosine release within the joint. Leflunomide has similar effects through inhibition of T lymphocytes.⁴⁴ Methotrexate was given on the same day each week at an oral dose of 2.5 mg at 0, 12, and 24 hours, for a total dose of 7.5 mg during the 24-hour period. Leflunomide was given orally at a dose of 10 mg/cat daily. When clinical signs were deemed to have significantly improved, the dose of methotrexate was reduced to 2.5 mg once weekly, and 10 mg of the leflunomide was given twice weekly.⁴⁴ The cats in the test group were treated for periods ranging from 2 to 6 months. Two of the twelve cats were determined to have had no beneficial response, and two were deemed to be in remission. More than 80% of the cats had a moderate or marked beneficial response in clinical signs. Rheumatoid factor was retested in only three cats but was decreased by at least 50% in all. Hepatotoxicity has been encountered with the use of these drugs, especially in humans, but was not encountered in these cats.⁴⁴ Severe cases of erosive arthritides may require surgical arthrodesis, especially of carpal and tarsal joints, to improve function and decrease pain in affected individuals.

The results of RA treatment with methotrexate and leflunomide notwithstanding, the expected outcomes with treatment are generally much poorer in erosive versus idiopathic immune-mediated arthritides. Type I idiopathic arthritis has the best prognosis. It is common for response to treatment to be rapid and for the condition to resolve without relapse. Types II and III idiopathic arthritis have a similar expected outcome, provided the mitigating medical factors can be identified and corrected. Type IV idiopathic arthritis obviously carries a much poorer expected response to treatment given the grave nature of most of the myeloproliferative diseases associated with it. For this reason the recommendation has been made that all cats with polyarthritis should have a bone marrow aspirate as well as FeLV and FIV tests included in the diagnostic workup.⁷ Euthanasia is a common eventual outcome in cases of erosive immune-mediated feline polyarthritides. Cats with PPP do not go into remission but may be able to maintain a reasonably comfortable lifestyle on treatment. SLE arthritis may be controllable, but disease processes in other organ systems may be more challenging to manage.

CONDITIONS OF THE FRONT LIMB

Dorsal Scapular Luxation

The scapula is attached to the thoracic body wall by the serratus ventralis, rhomboideus, and trapezius muscles. Trauma incurred by falling from a height can cause partial or complete rupture of these muscles, which will displace the scapula, especially during weight-bearing activities. If these cats are presented acutely there will be a non-weight-bearing lameness accompanied by pain and swelling between the dorsal scapula and the body wall. After a few days, this inflammation will subside and the cat will begin bearing weight. This produces an unusual gait because the scapula is dorsally displaced with each step. Most cats will regain a reasonable degree of mobility, and if the cat is not especially active, no further treatment need be undertaken. The cat will, of course, retain the distinctive gait abnormality. If the owner is unsettled by the gait or feels that it represents a disability for the cat, surgical stabilization can be undertaken. This consists of exploration of the dorsal aspect of the scapula and primary suturing of the torn muscles, where possible. In addition, two holes are drilled in the dorsocaudal area of the scapula and the scapula is attached in a normal position to an adjacent rib with nonabsorbable suture or surgical wire. Care must be taken that the thoracic cavity is not entered while passing the wire. The limb is then bandaged against the chest so it cannot be used for 2 weeks.⁴⁸

Carpal Hyperextension Injuries

Another injury commonly associated with falling from a height is hyperextension of the carpus. Alternatively, the carpus may become hyperextended on account of the degenerative process of an immune-mediated arthritis. Stress radiography will delineate the level at which the hyperextension has occurred, with antebrachial-carpal and carpometacarpal hyperextensions being most common (Figure 26-10, A). In some cases the hyperextension may involve more than one joint level, or it may be unclear which levels are affected.

The most common treatment for carpal hyperextension injuries in the cat is pancarpal arthrodesis (see Figure 26-10, B). Although a number of methods have been described, including cross pins, external skeletal fixators, and circular external skeletal fixation, the most commonly used and consistently effective technique involves the dorsal placement of a bone plate. After the articular cartilage is débrided from the joint surfaces and the gaps are packed with cancellous bone graft (usually from the ipsilateral proximal humerus), a plate is placed over the dorsal surface of the distal radius, carpus, and third metacarpal bone. Two types of plates work well in this application. A carpal arthrodesis plate (CAP) is



FIGURE 26-10 A, Stressed radiographic view demonstrating hyperextension at the carpometacarpal joint level. B, Postoperative radiograph from the cat in Figure 26-16 after pancarpal arthrodesis with a length of veterinary cuttable plate and 2-mm screws.

designed to place larger-diameter screws in the distal radius and smaller-diameter screws in the carpal and metacarpal bones. In addition, the CAP is manufactured such that it tapers in thickness from the radial to metacarpal portions and so that it places the arthrodesed carpus into a slightly extended standing angle of about 5 degrees. The plate can be contoured to alter this angle, but that is seldom necessary. CAPs come in an assortment of sizes, with the two smallest sizes being applicable to cats. The majority of cats will do well with the CAP that accommodates 2-mm diameter screws in the radius and 1.5-mm screws in the third metacarpal bone. Some large cats may fit a 2.7/2.0 CAP with 2.7-mm screws proximally and 2-mm screws distally. The average feline third metacarpal bone is 3.15 to 4.13 mm in diameter, so a 1.5-mm screw would occupy less than 50% of the bone's diameter, lessening the chance of iatrogenic fracture, a reasonably common complication. Thus the 2.0/1.5 CAP is the best fit for the average cat.¹² The CAP

is also engineered to cover more than 50% of the length of the third metacarpal, which has also been found to decrease the chance of iatrogenic fracture in dogs.

A cost-effective alternative to the CAP is the veterinary cuttable plate (VCP). The plate is sold in 50-hole lengths of two sizes. The screw holes in the small VCP accommodate 1.5-mm or 2-mm screws. The larger VCP accommodates 2-mm and 2.7-mm screws. An 8- or 9-hole length of the 1.5/2.0 VCP is usually the best choice when using VCPs. This allows one 2-mm screw to be placed first in the radial carpal bone, four 2-mm screws to be placed in the distal radius, and three to four 1.5- or 2-mm screws in the distal carpal bones and third metacarpal bone. The biggest advantage of the VCP is its cost. An 8- or 9-hole length of 1.5/2.0 VCP would be approximately 25% to 30% of the cost of a CAP. The plate can be used for many orthopedic applications, making it a versatile addition to a clinic inventory, whereas the CAP has few applications beyond carpal arthrodesis. The disadvantages of the VCP is that it is not as strong as the CAP; however, a second length of VCP can be stacked on top of the first length to increase its bending strength if this is deemed necessary in a very heavy cat. With limited soft tissues to close over the plate, this can sometimes increase tension on the incision but is usually not a significant problem.

The VCP also has only round holes, whereas the CAP has dynamic compression holes. It is common to load the most distal radial and most proximal metacarpal hole in compression; however, the importance of this in the cat is unknown. The principles of carpal arthrodesis have largely been extrapolated from experience with the dog, and data in the cat are scarce.¹² Because the dorsal application of a plate is not on the "tension" side of the bone, it has been deemed to be in a mechanically inferior position to withstand cyclic loading. Thus in dogs it is routine to augment the plate arthrodesis, usually with a cast or splint for at least the first 4 to 6 weeks. Support of the plate has also been attempted with one or two additional Kirschner wires angled across the carpal joint. These have commonly been found to migrate. Good results have been achieved with limited or no additional support for the plate, a fact that leads to speculation as to whether additional support is necessary in the cat.¹²

Complications of pancarpal arthrodesis primarily seem to involve a decrease in jumping and climbing behavior, based on the results of an owner survey.¹² Given the degree to which cats pronate and supinate the antebrachium and given the degree to which this would be affected by carpal arthrodesis, it has always been postulated that pancarpal arthrodesis would dramatically affect behaviors involving pronation and supination, such as grooming and batting of objects with the paw. Owners of cats that underwent pancarpal arthrodesis did not feel that this was a significant issue.¹²

When it can be clearly demonstrated that hyperextension involves the carpometacarpal joint level, partial carpal arthrodesis is an option in the cat.¹² T-plates (1.5 or 2.0 mm) allow fusion of the distal carpal and carpometacarpal joint levels while preserving function at the radial-carpal level. In dogs it has been shown that partial carpal arthrodesis results in the progression of DJD in adjacent joint levels that are not arthrodesed. Whether this also happens in cats and its potential clinical significance is not known.

CONDITIONS OF THE HIND LIMB

Hip Dysplasia

Hip dysplasia is a less common condition in cats than dogs; however, the exact incidence is unclear. One survey identified 6.6% of a population of 684 cats as being radiographically dysplastic.⁵⁸ The frequency of clinical signs attributable to hip dysplasia is another matter, however. Although reports of hind limb stiffness, weakness, or pain are few, there is an increasing realization that the signs associated with DJD in the cat are underdiagnosed. There is an increased incidence in female and purebred cats, with the Maine Coon, Siamese, Persian, and Himalayan breeds overrepresented.^{58,96} The radiographic signs of hip dysplasia in the cat differ somewhat from those seen in the dog. The primary finding is a shallow acetabulum, even given the fact that the normal acetabulum is shallower in the cat than the dog (Figure 26-11). Hip joint laxity, as measured by distraction index, is greater in dysplastic cats, although coxofemoral subluxation on the hip extended radiographic view is much less commonly seen in the cat than in the dog. Distraction indices have been reported as 0.6 in dysplastic cats and 0.49 or less in normal cats.⁶¹ The mean Norberg angle in a random group of cats was found to be 84 degrees among those considered dysplastic and 92.4 degrees among those considered normal, compared with 103 degrees in normal dogs.⁶¹ Degenerative changes seen in the hips of dysplastic cats also differed from the most common signs seen in dogs in that the craniodorsal acetabulum showed the most changes, whereas the femoral neck was relatively unaffected.⁵⁸ Treatment for dysplastic cats seldom seems necessary, but some may benefit from the previously discussed protocol for treating DJD, including weight control, modifications to the cat's environment, and NSAIDs. Conceivably, surgical salvage procedures such as femoral head and neck ostectomy could be used. Surgical prostheses are now available so that total hip replacement can be performed on cats; however, it is currently unclear whether this procedure offers any functional advantages over femoral head and neck ostectomy.⁷⁰



FIGURE 26-11 The primary finding in feline hip dysplasia is a shallow acetabulum; changes associated with degenerative joint disease are also common findings.

Patellar Luxation

Although patellar luxation occurs in the cat, it is much less common than in the dog, at least when it comes to the development of clinical signs. Many cats have some degree of mobility in their patellas. One may even go so far as to say that the classic description of a "grade 1" patellar luxation in the dog, wherein the patella can be luxated with moderate force but immediately returns to its normal position, may be a normal state of affairs for some cats. Certainly, such a state rarely would be associated with any clinical lameness in the cat. Grade 2 luxations are described as being easily achieved, but the patella spends most of its time in the normal position. Grade 3 luxations are characterized by a patella that is usually found luxated but can be reduced manually. Grade 4 luxations involve a continuously luxated patella that cannot be manually reduced. It should be pointed out that the previously described rating system, while useful in communicating what has been detected on physical examination, does not imply prognosis, nor does it correlate well with clinical signs.

As in the dog, most feline patellar luxations are medial, usually congenital, and bilateral. A small percentage of cases are lateral luxations, and a few cats seem to have the curious ability to luxate their patellas in either direction. Traumatic injury, especially malunions of femoral fractures, account for a small number of patellar luxation cases. Devon Rex, Abyssinian, and domestic shorthair cats have been identified as having a high incidence.^{60,73,96} Although patellar luxation can be found at

any age, most cats that develop clinical signs will do so within the first 1 to 2 years of life. Clinical signs in cats differ somewhat from those seen in dogs in that the "skipping lameness" characteristic of a poodle with medial patellar luxation is uncommon in the cat. A locking of the stifle with extension of the hind limb is a common presentation. Many cat owners will describe an associated painful response from the cat. There may be some degree of lameness or a crouched gait in some cases. The clinical signs may be described as acute in some cases and more intermittent and chronic in others.^{73,96}

Although much more is known about the pathogenesis of congenital patellar luxation in dogs, it does appear as though the developmental abnormalities of the hip (coxa vara), femoral bowing, and tibial torsion that are instrumental in the canine condition are not commonly encountered in the cat. More often, the problem arises from a shallow trochlear groove and an underdeveloped medial trochlear ridge. Occasionally, some medial deviation of the tibial tuberosity may be present but this is much less common than in dogs.^{60,73,96} An association has been noted between hip dysplasia and medial patellar luxation in that in one study cats were three times more likely to have both conditions together than either alone.⁹⁹ The clinical significance of this finding is unknown. The majority of cats with patellar luxation will never need treatment because they will never show clinical signs; some evidence suggests that even cats with clinical signs should initially be treated conservatively.⁷³ Rest and NSAIDs may be sufficient to manage intermittent episodes of lameness.

Radiographically, signs of DJD are slow to develop in cats with patellar luxation, and attempting to prevent such DJD is not a justification for surgery. Similarly, the decision to go to surgery should not be based on the grade of luxation but rather on the clinical signs and the degree of disability they cause the cat. In cats with more severe signs for which medical management has not been successful, surgery is associated with a high success rate. In dogs tibial tuberosity transposition is the most important procedure linked with a successful outcome. In cats that is usually not the case. If medial (or lateral) deviation of the tibial tuberosity is encountered, then transposition should be done. The keys to successfully performing this procedure are to make a sufficiently large osteotomy that will accommodate at least two Kirschner wires of 0.045 or 0.062 mm diameter. The osteotomy can be performed with a power-driven saw or an osteotome and mallet and should be at least 0.5 cm in thickness and depth and 1 cm in length. The veterinarian should always err on the side of making the osteotomy larger than necessary, for as long as the cut is positioned cranial to the insertion of the cranial cruciate ligament, no harm will arise. However, an inadequately small osteotomy will split

when implants are driven into it or will not stand up to weight bearing, resulting in a surgical complication that is very difficult to deal with. The osteotomy should be continued distally only far enough so that the tuberosity can be lateralized (or serialized). There is no need to completely detach the tuberosity. In most cases if a tibial tuberosity transposition is required at all, it will only be necessary to move the tuberosity a very few millimeters, sometimes only one or two. The most frequent complications in tibial tuberosity transposition are fracture of the osteotomized fragment or implant failure if the fragment is too small, lateral (or medial) patellar luxation if transposition is over exuberant or inadequate, and pin migration.

The most common surgical procedures to stabilize a patellar luxation in cats are deepening of the trochlear groove (trochleoplasty) or sulcus (sulcoplasty) and soft tissue procedures involving medial and lateral joint capsule and fascia. Trochleoplasty or sulcoplasty involves deepening the trochlear groove so that the patella will tend to remain in place. Originally, trochlear sulcoplasty simply involved taking a bone rasp to the groove to abrade cartilage and subchondral bone until a sufficient depth was achieved. The procedure destroys the articular cartilage and results in its replacement by fibrocartilage.⁵⁹ While many animals have done well with this procedure, many others have developed very extensive and clinically-significant DJD. As a result, the procedure has largely fallen out of favor in the wake of procedures that spare the articular cartilage.

The first of these was the wedge recession trochleoplasty, in which a lengthwise elliptical wedge is cut from the trochlear groove. A second cut is usually performed a millimeter or less from the edge of the original saw cut, and then the wedge is replaced in the deepened sulcus. It is not necessary to fix the wedge in place because the pressure of the patella maintains it in position.^{59,96} Usually, the cuts are made with a handsaw because this gives the surgeon more control over what is a reasonably delicate cut. Because the most common problem seen in the cat is an underdeveloped medial trochlear ridge, the surgeon may find it helpful to angle the cuts somewhat steeper medially so that the resulting sulcus will be deeper on the medial side. If the wedge does not sit solidly, the bottom surface can be trimmed with a scalpel blade until it is stable. A variation of the wedge recession is the block recession trochleoplasty in which a rectangular block of articular cartilage and subchondral bone is removed from the trochlear groove utilizing a bone saw and osteotome. Once the block is removed, additional bone is removed from the underlying defect and from the underside of the block. This allows the replaced block to be recessed below the original surface, producing a deepened groove. The advantage of this procedure is that a greater area of the sulcus is involved in the recession, especially in the proximal portion. The

proximal aspect of the groove may be the most critical area, especially when the stifle is in extension.^{59,96}

Recently, a rotating dome trochleoplasty technique, in which the trochlear groove is osteotomized and rotated medially, has been described. The procedure was performed on feline cadavers but showed some potential advantages over the currently used trochleoplasty techniques, particularly in increasing the size of the medial trochlear ridge. The ultimate utility of the procedure awaits clinical trials.

Soft tissue surgical corrections for patellar luxation include joint capsule and fascial imbrication and desmotomy. Essentially, one tightens the tissues on the side away from the luxation and loosens them on the side of the luxation. In the most usual case of a medial luxation, the surgeon would start with a medial parapatellar arthrotomy. This would allow inspection of the joint and completion of the trochleoplasty, if desired. A corresponding incision in the lateral joint capsule would allow placement of an osteotome to perform a tibial tuberosity transposition if that is deemed necessary. The medial incision can, in some cases, be continued as much as one third of the way up the distal femur if this relieves medial pressure from the patella and lessens the tendency for luxation. At surgical closure there is no need to do anything on the medial side. Joint capsule, muscle, and fascia may all be left open in what can be called a medial desmotomy. On the lateral side, the same incised tissues can be closed in one layer with mattress sutures, vest-over-pants sutures, or simply large bits of tissue in a simple interrupted pattern. The principle is to "gather or tighten up," otherwise referred to as imbricating, the tissues on the lateral side. Although some have described the use of nonabsorbable sutures for this procedure, the author has not found these to be necessary. Most absorbable sutures are present for an adequate period to allow healing of the tissues. Nonabsorbable sutures cannot be expected to hold the tissues in an unaltered state for an indefinite period.

Although soft tissue procedures can be very helpful in surgically stabilizing a luxating patella, they are rarely sufficient by themselves. "Bony procedures," such as trochleoplasty or tibial tuberosity transposition are the key components to surgical success. In those rare cats that luxate their patellas both laterally and medially and have clinical lameness, surgical correction with a wedge or block recession trochleoplasty and bilateral soft tissue imbrication is the most effective course.

Although the majority of cats with patellar luxation will have the problem bilaterally, not all will show clinical signs bilaterally. As previously mentioned, the decision to do surgery must be based on clinical signs, not the finding of the problem on a physical examination. If a cat is clinically affected bilaterally, it will easily tolerate bilateral surgery, and the surgeon need not be afraid to correct both stifles at the same surgery session.

Postoperative care of patellar luxation patients involves nothing more complex than adequate analgesia for a few days and exercise restriction for 4 to 6 weeks.

Stifle Ligamentous Injury

Cats suffer ligamentous injury to the stifle because of either trauma, where there are often multiple injuries to supporting structures, or as the result of a degenerative process that culminates in rupture of the cranial cruciate ligament. In the former case the most common trauma is a “falling” injury which can result in ligamentous and meniscal injury to the extent of complete stifle disruption or luxation. On physical examination, these cats have profound stifle instability in most planes. Radiographically, the most common appearance is of the tibia luxated cranially due to the almost invariable rupture of both the cranial and caudal cruciate ligaments. The medial collateral ligament is the next most likely structure to be compromised, followed by meniscal tearing in 50% of cases, with the lateral meniscus being most common.^{18,49} An assessment of the ligamentous injuries can usually be deduced through radiography, stress radiography, and a thorough physical examination, under general anesthesia. Ultimately, surgical exploration of the joint will reveal all.

Stifle disruption can be treated surgically in two potential ways. The initial step in both procedures is exploratory arthrotomy to identify the pathology and to débride any meniscal tears. In many cases, damage to one or both menisci will be severe enough to require complete meniscectomy. Ligamentous damage can be addressed either by reconstruction or by transarticular pinning. Occasionally it may be possible to suture or plicate stretched collateral ligaments with absorbable suture, but in most cases reconstructive techniques will be necessary to replace torn ligaments or reinforce suture repairs.

The most straightforward approach to collateral and cruciate ligament reconstruction in the cat begins with a conventional lateral extracapsular cranial cruciate ligament procedure. Although there are dozens of variations to extracapsular cruciate ligament procedures, most involve the placement of a nonabsorbable suture from behind the fabellae, behind the distal patellar tendon, and through a hole in the proximal tibia. Monofilament nylon leader material of 40 pound test strength is a popular choice in the cat. The ends can be fastened at the starting point with a metal crimp tube or any of several knots. Lateral placement of this extracapsular suture replaces the stability provided by the cranial cruciate and lateral collateral ligaments. Care must be taken not to overtighten the suture if the medial collateral ligament is compromised because a valgus deformity at the stifle can be created. Varus or valgus deformity can alternatively be prevented by the temporary placement of a

transarticular pin, which is removed after ligamentous reconstruction.

On the medial side of the stifle, if caudal cruciate or medial collateral ligaments are torn, reconstruction is best accomplished by placing 2.7-mm screws and washers or bone anchors in the distal femur and proximal tibia. In the proximal and distal planes, these anchor points should be placed at the mid-level of the femoral condyle and about 1 cm distal to the level of the tibial plateau. The final femoral anchor point is then slightly caudal to the midcondyle and the tibial anchor point slightly cranial of a line drawn distally from the intercondylar eminences of the tibia. A figure-of-eight suture between the anchor points then provides reconstruction of the medial collateral ligament, but the slightly caudo-proximal to craniodistal course also mimics the course of the caudal cruciate ligament and provides stability in the absence of that ligament. Again, monofilament nylon leader material is a popular choice.

Another way to stabilize a disrupted stifle after resection of damaged meniscal tissue is to place a transarticular pin. A 3- to 3.5-mm Steinmann pin can be introduced at the intercondylar eminences on the tibial plateau and driven in a distocranial direction to exit at the distal extent of the tibial tuberosity. The chuck is then attached to the distal extent of the pin, and it is drawn flush to the surface of the tibial plateau. The stifle joint is then placed in a standing angle of approximately 30 to 40 degrees of flexion, and the pin is driven proximally through the intercondylar fossa of the femur until the pin is lodged in the cranial cortex of the distal femur. The joint capsule and remaining soft tissues are then closed routinely.^{18,49} Distally, the pin is cut with enough room to allow its removal in 4 weeks. External support, especially in larger cats, in the form of a lateral splint can be helpful because the pin very frequently bends and occasionally breaks. Although stifle range of motion is ultimately reduced to some degree, owners and clinicians are generally amazed at the level of function and the range of motion that returns in these limbs.^{18,49} The procedure is relatively simple and requires little in the way of specialized equipment, but it usually allows a very serious situation to be salvaged.

Cats also rupture their cranial cruciate ligaments without any apparent trauma. There is epidemiologic and histologic evidence that they suffer from the same type of degenerative cruciate disease as dogs.^{49,95} Histologic disorganization of collagen fibers interspersed with acellular or hypocellular areas, chondroid metaplasia, and mineralization are found in degenerative cruciate ligaments along with granulation tissue formed in an attempt to repair the damage.⁴⁹

Affected cats tend to be older and overweight, with a mean age of 8.5 years and a mean weight of 6.5 kg identified in one survey.⁴⁹ They are presented with rear limb lameness, reluctance to jump, and generally decreased

activity. Diagnosis of cranial cruciate ligament (CCL) rupture is fairly straightforward on physical examination insofar as cranial drawer motion is usually present and quite easy to demonstrate, even in the nonsedated cat on the examination table. However, in some chronic cases or in the occasional case of early degeneration or partial ligament tear, the detectable stifle instability may be minor. Although the diagnostic challenge of partial CCL tears and mild inflammation of the ligament without grossly detectable stifle instability is commonly encountered in the dog, this seems to be relatively rare in the cat. The author (GH) has encountered some cats with cruciate ligament disease in which little stifle instability is present.

The more common obstacle to accurate diagnosis of CCL rupture in the cat is often the absence of an index of suspicion on the part of the clinician. Radiographically, cats with CCL rupture will usually have some joint effusion, although the amount is seldom as impressive as in the dog. Degenerative osteoarthritic changes are common and increase with chronicity (Figure 26-12). Cats develop a prominent osteoarthritic spur on the distal aspect of the patella in most cases. Distal displacement of the popliteal sesamoid bone is a variable radiographic finding.⁴⁹ A unique radiographic feature in the cat that is commonly encountered in CCL rupture is the development of mineralization within the stifle joint. Radiodense mineralized bodies are most often seen in the cranial joint space but can also be seen in the caudal joint space of the stifle. In most cases this mineralization forms as dystrophic calcification at the insertion point of the CCL on the cranial tibial plateau. Occasionally, the mineralization originates in the cranial horn of the medial or lateral meniscus.⁸⁹ Although the pathogenesis of calcification at the insertion of the ruptured ligament would seem to involve resolution at a site of inflammation and hemorrhage, the reasons for the development

of calcified meniscal cartilages are less clear. Both processes are associated with CCL rupture in the overwhelming majority of cases, and it appears that excision of the calcified tissue, along with extracapsular stabilization of the stifle, is curative.^{49,89} Although the presence of a degenerative process partially explains the rupture of many CCLs in the cat, little else is known about the pathogenesis. For example, a clear genetic predisposition has been demonstrated in the dog but remains a subject of speculation in the cat. An increased tibial plateau angle also appears to be an important contributing factor in some dogs and has recently been shown to be of potential importance in the cat.⁹⁵ A comparison of 21 cats with CCL rupture to a control group of 34 cats showed that the CCL group had a significantly higher tibial plateau angle (24.7 versus 21.6 degrees).⁹⁵ Still, the tibial plateau angle in the CCL group could not be considered "steep," at least by canine standards, in which angles of over 30 or 35 degrees are common, so the significance of this finding is unclear.

Treatment of CCL rupture remains somewhat controversial. If there can be said to be any dogma on this topic, it would be that isolated unilateral CCL rupture should be treated conservatively with NSAIDs and exercise restriction. However, as is so often the case in veterinary science, this recommendation emanates from one paper that is nearly 25 years old and involves only 18 cats.⁹⁴ Although it is true that many cats seemingly do well without surgical repair, the advancement of DJD in these joints is undeniable. In light of the underrecognized clinical signs, prevalence, and lifestyle impact of DJD in the cat, one must wonder whether cats are always best served by conservative management. Some clinicians adopt the approach of starting with conservative management but opting for surgery if lameness persists past 4 weeks. Extracapsular repair techniques reliably return cats to weight bearing, often in less than 1 week; offer an opportunity to débride intraarticular calcification or meniscal pathology; and will provide normal or near-normal function in nearly all cats.⁴⁹ This is the approach advocated by the author (GH) in light of seemingly increasing numbers of older, overweight cats that do not seem to do well with conservative therapy.

A curious addition to the limited published information on CCL rupture in the cat proposes a link with hypertrophic cardiomyopathy. In a report of three cats that died shortly after CCL surgery, as a result of apparent heart failure, the presence of hypertrophic cardiomyopathy was confirmed at necropsy in two of the cases.⁵⁵ No other reports have been published to corroborate such a connection; however, two of eight cats in another report that underwent CCL surgery were diagnosed with hyperthyroidism within 2 years of their surgeries.⁴⁹ Apart from being interesting, this information probably only reinforces the advice to do a thorough diagnostic workup on all cats in which surgery is contemplated, a



FIGURE 26-12 Dystrophic mineralization in the stifle of a cat with cranial cruciate ligament rupture.

workup that may include echocardiogram and thoracic radiography in some cases.

Common Calcaneal Tendon Ruptures

The common calcaneal tendon (CCT) consists of the combined tendons of the gracilis, semitendinosus, and bicep femoris muscles that insert on the medial aspect of the calcaneus; the superficial digital flexor muscle that passes over the top of the calcaneus and fans out to insert on the plantar surface of the second phalanges; and the calcaneal tendon, which is the major part of the CCT and ends on the lateral side of the calcaneus as the tendon of insertion of the gastrocnemius and soleus muscles. Equally important is the proximal end of this muscle group, which originates as paired heads of the gastrocnemius muscle on the caudal cortex of the distal femur, just proximal to the femoral condyles. The CCT group acts together with the quadriceps group of the cranial thigh region to flex the stifle and extend the hock.

Cats can be presented with acute or chronic histories of being “down in the hock,” or in a plantigrade stance in which the plantar surface of the hock is in contact with the ground (Figure 26-13). Some animals will exhibit a partial plantigrade stance if the CCT tendon components are torn except for the superficial digital flexor tendon. These incomplete ruptures of the CCT are also characterized by hyperflexion of the digits, especially if the stifle is extended and the hock is flexed. This manipulation stretches the superficial digital flexor tendon, which is the sole remaining component of the CCT, thus flexing the digits. Incomplete plantigrade stances may also be related to tibial neuropathies caused by diabetes mellitus, trauma, or degenerative conditions. Complete plantigrade stance presentations may be unilateral or bilateral.

Laceration of the CCT is a common cause, as is calcaneal fracture or tendon avulsion resulting from falling

injuries. In dogs tendinopathy of the CCT or degenerative rupture is well known, especially in older animals. There does appear to be a similar degenerative condition in older cats that can lead to CCT rupture, often bilaterally, without any history of trauma. There are also anecdotal reports of degenerative CCT ruptures that may be associated with the administration of fluoroquinolone antibiotics in the cat. Fluoroquinolones have been shown to have an inhibitory effect, in canine and human cell culture, on fibroblast, chondrocyte, and tendon cell proliferation, as well as collagen and proteoglycan synthesis.^{69,97,105} These effects have been linked to Achilles tendon ruptures in humans and caused the United States Food and Drug Administration to issue a warning to physicians in July 2008.⁸⁷

Diagnosis of CCT tendon ruptures are based on the history of plantigrade stance and the findings of a physical examination. In most cases a palpable defect in the distal one third of the tendon is present, but in some cases the gastrocnemius tendon avulses from the calcaneus. A unique presentation of CCT failure that seems to occur mostly in cats involves a plantigrade stance but no detectable defect in the distal tendon. Avulsion of the paired heads of the gastrocnemius muscle at their origin on the caudal cortex of the distal femur will produce the same clinical signs but can be more of a diagnostic challenge. Careful palpation of the area caudal and proximal to the stifle will generate a painful response in the acute phase but will often reveal a defect in the muscle. The integrity of the CCT can also be tested by placing the stifle in extension and flexing the hock joint. In the normal hock joint, it is abnormal to be able to flex the paw much below an angle of 140 degrees. The hock joint in cats with CCT rupture can often be flexed to 90 degrees or less.⁸¹ Geriatric cats may have more range of motion on hock flexion when tested in this manner but may still be normal. This is due to decreased muscle mass, especially in the hind limbs. Comparison should be made to the normal limb so that the test is not misinterpreted.

Treatment of CCT ruptures is usually surgical. Débridement of fibrotic tendon tissue followed by suturing with a three-loop pulley or similar tension-distributing suture is completed. Nonabsorbable sutures such as polydioxanone (2/0 or 0) work well for this purpose because these sutures maintain their integrity for more than the time necessary for healing. The hock should be placed in more extension than the standing angle to relieve tension on the repair, and then the hock joint is immobilized with a splint, cast, or transarticular external skeletal fixator for 4 weeks. Occasionally, if sufficient fibrotic tendon is débrided, the hock may have to be placed in nearly complete extension. This is not associated with any serious consequences for the limited time that the hock is immobilized. If the defect in the tendon is even greater, then



FIGURE 26-13 Cat demonstrating plantigrade stance after rupture of the common calcaneal tendon.

the gap can be bridged or the repair can be reinforced with a length of fascia lata harvested from the thigh region of the ipsilateral limb.

Recently, evidence produced in human and canine patients suggests that early controlled flexion of the hock joint may help align fibroblasts in a linear and stronger arrangement, thus making for faster, stronger healing. This can be accomplished with gentle passive range-of-motion exercises beginning 3 weeks after the operation. A bivalved cast or splint that can be removed as needed or an external skeletal fixator with hinges that can be loosened will facilitate this sort of physiotherapy. The prognosis for healing and return to function in isolated traumatic ruptures is good. In older cats with degenerative ruptures, the prognosis is somewhat more guarded. Some cases, especially more chronic ruptures in older, less active cats, have been treated conservatively with an acceptable outcome. The hock is placed in a steeper angle of extension than the normal standing angle, and a splint or cast is placed for 6 weeks, followed by another month of controlled exercise.⁸¹ Although these patients are likely to retain a more plantigrade stance than normal, they will be free of pain and usually have satisfactory function. In cats with bilateral rupture, especially where primary repair procedures have failed, arthrodesis of the tarsocrural joint is a treatment option. The development of a tarsal arthrodesis plate that can be applied to the medial surface of the hock and permits placement of 2.7-mm screws in the tibia and 2-mm screws in the tarsus and metatarsus has greatly simplified tarsocrural arthrodesis in the cat (Figure 26-14). The major potential complication of this procedure, however, is breakdown of the incision over the plate. This complication may require soft tissue care until the plate can be removed, which is at least 4 months postoperatively.



FIGURE 26-14 Tarsal arthrodesis plate that allows placement of 2.7-mm screws in the tibia and 2-mm screws in the tarsal and metatarsal bones.

NEOPLASIA

Primary musculoskeletal neoplasia is uncommon in cats. Although osteosarcoma (OSA) accounts for approximately 80% of primary malignant bone tumors, these cases account for fewer than 1% of all feline malignancies. OSA is a disease of older cats, with most cases being older than 9 years of age. Some reports have described a preponderance of female, domestic shorthair cats, with the hind limbs being involved most commonly. OSA can be divided into cases that involve the appendicular skeleton, primarily the distal femur or proximal tibia, but also the proximal humerus, and the less common axial skeletal cases involving the skull, pelvis, ribs, and vertebrae (Figure 26-15).⁸ Clinical signs include lameness and pain, which can be chronically progressive or acute in the case of a pathologic fracture. Local swelling may be noted at the tumor site and in axial OSA of the vertebrae; the initial signs may be neurologic because of swelling or vertebral collapse around spinal cord and nerves. Diagnosis initially requires radiography, which will show a primarily lytic lesion of the affected bone. New bone production may be present but tends to be less prominent than in the dog. Biopsy of the lesion is the only way to confirm a diagnosis. This is best accomplished using a Jamshidi needle. Taking at least three to four samples is the best way to maximize the likelihood of obtaining a definitive diagnosis.

The prognosis with appendicular OSA in the cat is much more positive than in the dog. The tumor seems to be less aggressive in the cat and seldom metastasizes in the early stages. That means that limb amputation may be curative, and adjunctive chemotherapy is seldom



FIGURE 26-15 Osteosarcoma of the proximal humerus. Note the periosteal reaction in the proximal humeral cortices.

necessary. Median survival times after amputation were 49.2 months in one study⁸ and 11.8 months in another.⁵² Although a curious discrepancy exists between those studies in survival times for appendicular OSA, the authors largely agree on the much graver prognosis for survival with axial OSA, citing 5.5 months in the first study and 6.07 months in the second. Some of the difference in reported survival times for appendicular OSA may be related to histologic grade and mitotic index of specific tumors, which has been directly linked to prognosis.²²

Chemotherapy of axial OSA using doxorubicin, carboplatin, or both after surgical excision has been described. Radiation therapy may also be of use in axial OSA. Some tumors histologically classified as OSA are “extraskeletal,” arising in assorted soft tissues, most often previous injection sites, but also orbital, oral, intestinal, hepatic, or mammary gland-associated sites.⁵² One report labeled 38% of feline OSA tumors as being extraskeletal and found affected cats to have a median survival time similar to appendicular cases.⁵²

OSTEOMYELITIS

Osteomyelitis occurs uncommonly in the cat under three different circumstances. First, it can arise secondary to deep bite wounds from other cats that involve bone in the septic process. Second, postsurgical osteomyelitis can develop in patients that have undergone surgical repair of traumatic fractures. Third, metaphyseal osteomyelitis can be seen, presumably by way of hematogenous infection, in young kittens. The latter condition seems to be much less common in the cat than in other species. *Staphylococcus* spp. are the most common bacteria involved and will produce mixed lytic and proliferative radiographic lesions in the metaphyseal region of affected long bones; will commonly produce systemic signs such as pain, swelling, pyrexia, and anorexia; and will usually respond to appropriate antibiotic therapy.¹⁰

Staphylococcus spp. are also the most common bacteria isolated in postsurgical osteomyelitis. Methicillin-resistant *Staphylococcus aureus* does not presently seem to be a common problem in cats; when it is isolated, the source seems to be from humans.⁶ Cats with osteomyelitis involving an orthopedic surgery site may show systemic signs, including pain, swelling, and lameness in the affected limb; pyrexia; and anorexia. Radiographically, elements of a proliferative periosteal or lytic reaction may be seen. Occasionally, a sequestrum of necrotic bone may be identified on radiographs. Soft tissue swelling is also prominent, and some cases may have draining fistulous tracts.

Treatment of this problem follows all the rules of managing a soft tissue infection: supportive care of the patient should be provided as needed, bacteriologic

samples for culture and antibiotic sensitivity are invaluable, drainage and lavage of areas with purulent exudate must be established, and appropriate bactericidal antibiotic therapy must be initiated. Antibiotic sensitivity testing will guide the selection of an appropriate drug, but pending those results an amoxicillin-clavulanate combination or a cephalosporin antibiotic is a good empirical choice. Both drugs attain good levels in bone and have a broad spectrum of activity against most common bacterial pathogens; however, some cats will become anorexic or will vomit after taking these drugs. Fluoroquinolones and clindamycin are other less desirable choices, in the former case because of poor levels in bone and in the latter case because of increasing instances of bacterial resistance. Orthopedic hardware such as pins and plates should be maintained if the fracture repair is stable, insofar as fractures will heal in the face of osteomyelitis. However, once the fracture is healed, it is often necessary to remove orthopedic hardware to ultimately resolve recurring osteomyelitis episodes. Stainless-steel hardware becomes coated in a protein calyx, which harbors bacteria and shields it from the effects of antimicrobials. Removal of all hardware will usually resolve the problem.

Treatment of osteomyelitis related to bite wounds follows the same principals of drainage, lavage as necessary, and antimicrobial therapy. The propensity for cat bite wounds to become infected is well known, with as many as 80% of cat bites to humans becoming infected, according to some surveys.³¹ *Pasteurella multocida* is the most commonly cultured bacterium from these wounds, and surveys have suggested up to 90% of the cat population harbor this organism in their mouths.³¹ In excess of 95% of isolates were found to be sensitive to benzylpenicillin, amoxicillin-clavulanate, cefazolin, and erythromycin, which would give some guidance for empirical therapy of affected cats pending specific microbiologic testing results. Osteomyelitis is best treated with antimicrobials for at least 4 to 6 weeks.

DYSOSTOSES

Dysostoses are congenital bone deformities involving individual bones or portions thereof. There are two of clinical significance in the cat.

Polydactyly

Many cats are presented with extra digits, and aside from occasional minor problems caused by lack of attention to regular nail trimming, the condition, known as polydactyly, is primarily a curiosity. It is the result of an autosomal dominant trait with variable expression.¹⁰¹

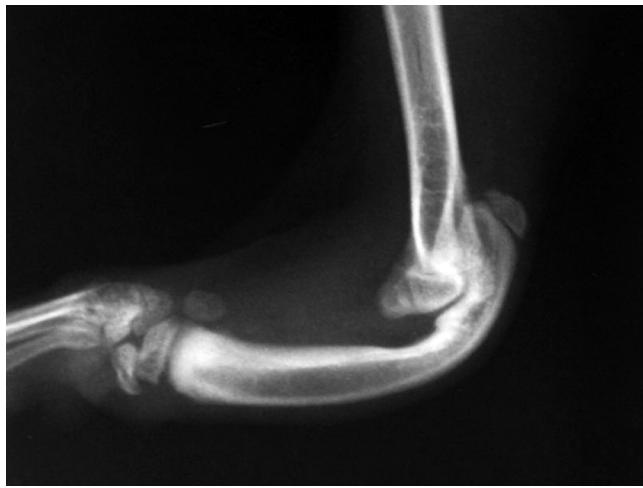


FIGURE 26-16 Forelimb radiograph of a cat with radial hemimelia. The only remnant of a radius in this limb is the small, oval density dorsal to the distal ulnar physis.



FIGURE 26-17 Synovial osteochondromatosis. This is a benign process, but it can affect joint range of motion.

Radial Hemimelia

Radial hemimelia involves complete or partial absence of the radius (Figure 26-16). As the limb develops without a radius, the action of flexor and extensor tendons deforms the ulna. A varus flexed deformity of the antebrachium produces a “flipperlike” appearance and leaves the front limbs largely nonfunctional. Although the condition can be unilateral and has been described as hereditary in Siamese and domestic shorthair cats, the practical significance of radial hemimelia in the cat is that it is a trait that is selected for by unscrupulous breeders. “Twisty cats” or “kangaroo cats” are marketed as uniquely desirable pets and get their names from the deformity of their front limbs that causes them to hop along on their back legs like rabbits or kangaroos. Occasionally, these cats may be presented to a veterinary clinic as the suspected victims of abuse. Although their deformity is not painful and usually cannot be significantly improved surgically, their lack of mobility may make them prone to attack from dogs or other animals.^{71,101}

MISCELLANEOUS MUSCULOSKELETAL CONDITIONS

Extraneous Skeletal Mineralization

The terminology surrounding extraneous mineralization of the cat skeleton is highly confusing. *Osteochondroma*, *osteochondromatosis*, *multiple cartilaginous exostoses*, *synovial osteochondromatosis*, and *synovial chondrometaplasia* are similar terms used to describe sometimes vastly different things. There are two primary syndromes that are seen with some regularity in the cat and must be distinguished. Osteochondromatosis involves the

production of exostoses of cartilage and subchondral bone at multiple sites that most often involve the skull, ribs, pelvis, scapula, and vertebrae. The lesions get progressively larger and can become aggressive, transforming to malignant neoplasms typed as OSA or chondrosarcoma. Affected cats are young adults that are nearly always test positive for FeLV. The exostoses may cause clinical signs because they grow by impinging on other structures. In this way neurologic signs are common when vertebrae are involved. Surgical debulking of the masses may be of some temporary benefit, but the progressive nature of the condition and the leukemia virus infection usually results in euthanasia.^{22,39,68}

More commonly seen is synovial osteochondromatosis, which is a much more benign condition that has been previously described in dogs (Figure 26-17).^{30,41} Cats have a propensity for producing mineralization in and around their joints, especially in the face of chronic inflammation. Radiographically visible mineralization at the CCL insertion or of meniscal cartilage is commonly seen in cats with cruciate ligament rupture. Synovial osteochondromatosis is an excessively exuberant variation of that tendency in which impressive amounts of intraarticular and extraarticular calcification forms. Chronic inflammatory stimulation of synoviocytes results in metaplasia, such that mineralized bodies are produced in association with synovial membranes. These “joint mice” may produce pain and lameness and are frequently large enough to restrict joint range of motion. Affected cats can be of any age but are generally middle-aged or older. There is no connection with FeLV, and although it is chronically progressive, the condition follows a much more benign course and does not necessitate euthanasia. Treatment can involve surgical excision but should also address the source of the chronic



FIGURE 26-18 Tendon contracture involving the digit of an adult cat that had undergone an amputation of P1 and P2 after a traumatic injury.

inflammation (e.g., CCL rupture). Recurrence after surgery is common but does not always result in the return of clinical signs.^{22,30,39,41,68}

Tendon Contracture

Cats have a tendency to develop contracture of their tendons in two distinct instances: as newborns and as adults. Newborn kittens can develop a tendon contracture condition that produces deformity of the distal limb and paw, primarily in the hind limbs (see Chapter 41). This condition seems to resolve as the kittens become ambulatory, and the cause is unknown. The significant point to remember is that these kittens may be brought to the veterinarian for euthanasia because of what the owner mistakenly believes is a hopeless congenital deformity.

Adults can develop tendon contracture, which may be associated with trauma. The most common source of such trauma is onychectomy. Remnants of the flexor process on the third phalanx may still have the deep digital flexor tendon attached. As the tissue heals, there may be contraction of the flexor tendon, producing flexion of the toes into a "claw grip" (Figure 26-18). This produces pain and lameness for the cat. Alternatively, cats may develop progressive flexor tendon contracture involving the front limbs without any history of trauma. The flexor carpi radialis and flexor carpi ulnaris tendons are preferentially affected, producing deformity and disability. The cause of the contracture is unknown, but a parallel has been drawn to Dupuytren's contracture in humans, which is a genetic condition. However, Dupuytren's contracture involves the palmar fascia of the hands, whereas the problem in cats seems to involve the tendons. Tendon contracture, of whatever cause, can be treated with stretching exercises and intermittent

splinting if the contracture is minor. In advanced cases tendon transection may be required.⁴⁸

Osteogenesis Imperfecta

Osteogenesis imperfecta is a genetic mutation of a gene coding for type I collagen. The result is a syndrome of fragile bones that develop pathologic fractures with minimal trauma. The condition has been described in humans, cattle, and dogs and infrequently in cats.²⁶ The fractures usually begin to appear between 10 and 18 weeks of age. In addition to the history of multiple fractures with minimal trauma (e.g., jumping from a low height), radiology will reveal "folding fractures," which are typical of pathologic etiology, as well as thin long bone cortices with decreased density, and there may be evidence of other healed fractures. Primary and secondary hyperparathyroidism are differential diagnoses; however, these can usually be ruled out by normal serum chemistry and parathyroid hormone levels. In cats there also appears to be a link between osteogenesis imperfecta and dentinogenesis imperfecta. Because dentin is composed of type I collagen, cats with osteogenesis imperfecta commonly have abnormal dentin development characterized by pink discoloration of the teeth and tooth fractures. Cats with patellar fractures, and often with evidence of other healing fractures, have been noted to have persistent deciduous teeth, which are thought to be another manifestation of the osteogenesis imperfecta-dentinogenesis imperfecta link.

Definitive diagnosis of osteogenesis imperfecta involves analysis of type I collagen cultured from dermal fibroblasts; however, this test is not readily available. Alternatively, bone biopsy, which may be obtained during surgical fracture repair, will reveal decreased cortical and trabecular bone, reduced numbers of osteons with porous lamellar bone interspersed with loose connective tissue.²⁶ The prognosis for these cats is guarded to poor because even the most patient owner is likely to find it difficult to manage the recurrent fractures. Therapy with vitamin C has been suggested because it plays a role in collagen formation and tissue repair. In addition, bisphosphonate therapy, specifically alendronate at 3 mg/kg orally, every 12 hours, has been suggested, although little information exists regarding its efficacy for this condition in cats. Bisphosphonates act by inhibiting osteoclastic bone resorption and have been credited with reducing pathologic fractures in osteoporotic menopausal women and in children with osteogenesis imperfecta, where these drugs also appear to increase bone density.²⁶

Transitional Vertebrae

Transitional vertebrae are defined as abnormal vertebrae that occur in the areas between segments of the vertebral

TABLE 26-1 Congenital Myopathies in the Cat

Disease	Affected Breeds and Geographic Provenance	Mode of Inheritance	Underlying Defect	Clinical Signs	Prognosis
Congenital myotonia	DSH (NZ, US)	Autosomal recessive	Probable defect in chloride channels	Stiff gait; hyperactivity of the selected muscle groups when startled; percussion dimple	Fair to good (nonprogressive condition; cats enjoy normal quality of life)
Devon Rex myopathy	Devon Rex (AUS, GB)	Autosomal recessive	Unknown	Cervical ventroflexion; generalized muscle weakness; abnormal gait; megaloesophagus	Poor (many cats die of asphyxiation)
Dystrophin-deficient myopathy	DSH (US, NL, CH)	X-linked recessive	Dystrophin deficiency	Skeletal muscle hypertrophy with possible complications; sensitivity to stress; stiff gait	Guarded to fair (cats can have almost normal quality of life but may require more frequent veterinary visits)
Glycogen storage disease type IV	Norwegian forest cats (US, Europe)	Autosomal recessive	Glycogen branching enzyme deficiency	Stillbirth; muscle tremor; muscle atrophy; cardiomyopathy	Poor (all cats eventually die)
Hypokalemic myopathy	Burmese (AUS, NZ, GB, NL)	Probably autosomal recessive	Unknown	Transient, paroxysmal clinical signs with generalized muscle weakness, cervical ventroflexion	Good response to potassium supplementation
Malignant hyperthermia	DSH	Unknown	Unknown	Severe hyperthermia during anesthesia (halothane)	Poor (the two reported cats died)
Merosin-deficient myopathy	DSH, Siamese (US)	Unknown	Merosin (laminin alpha ₂) deficiency	Hindlimb weakness from 6 months old, worsening to muscle atrophy and contractures at 1 year old	Poor (both cats were euthanized before 2 years of age)
Myasthenia gravis (MG)	DSH	Unknown	Lack of acetylcholine receptors	Generalized muscle weakness	Fair, generally good response to therapy
Nemaline myopathy	DSH (US)	Possibly autosomal recessive	Unknown	Progressive weakness (6-18 months); rapid, choppy, hyperthemic gait; tremor, exercise intolerance	Poor (all five reported cats died or were euthanized)

DSH, Domestic shorthair; NZ, New Zealand; US, United States; AUS, Australia; GB, Great Britain; NL, Netherlands; CH, Switzerland.

Gaschen FP, Jones BR: Feline myopathies. In Ettinger SJ, Feldman EC, editors: *Textbook of veterinary internal medicine—diseases of the dog and cat*, ed 6, St Louis, 2005, Elsevier Saunders, p 907.

column. They may be thoracolumbar, lumbosacral or sacrococcygeal transitional vertebrae. Transitional vertebrae may display characteristics of the vertebrae types on either side; they may display abnormalities of length, spinous or transverse process formation; and they may be asymmetrical in shape in either the dorsoventral or left and right planes. For the most part, transitional vertebrae do not produce clinical signs, and their significance is as a unique feature rarely encountered on radiographs. In dogs lumbosacral transitional vertebrae have been linked to an increased incidence of hip dysplasia and lumbosacral stenosis. There may be a similar connection in the cat.⁸³

MYOPATHIES

Myopathies are uncommon in the cat, but several congenital (Table 26-1)³⁵ and acquired diseases (Box 26-7) have been described in the literature. This chapter covers only the most common of a rare group of diseases. For more information on neuromuscular diseases, see Chapter 27. Common clinical signs seen in cats with myopathies include weakness and a stiff, stilted gait. Weak cats often show neck ventroflexion (Figure 26-19). The diagnosis of myopathies in the cat includes a minimum database (CBC, serum chemistries and electrolytes, urinalysis, +/− total T₄) and may also include

BOX 26-7**The Most Common Acquired Myopathies in the Cat****Inflammatory**

Infectious: *Toxoplasma*, *Clostridium*, feline immunodeficiency virus
Idiopathic/immune mediated

Metabolic

Hypokalemia
Hyperthyroidism

Ischemic

Thromboembolic disease associated with congestive heart failure



FIGURE 26-19 Cats with hypokalemia may exhibit profound muscle weakness.

infectious disease testing (e.g., *Toxoplasma*, FeLV, FIV) and special investigative techniques (e.g., electromyography, motor nerve conduction studies, muscle biopsy with histologic analysis, and immunohistochemical staining). Clinicians should consult the diagnostic laboratory before performing muscle biopsies for requirements regarding sampling, handling, and shipping. Generally, fresh or flash-frozen samples are preferred.³⁵ Fixing biopsy samples in formaldehyde does not allow for a comprehensive examination.

Because the half-life of creatine kinase (CK) is thought to be short in the cat, as it is in other species, an elevated serum CK level indicates recent changes. However, many factors other than myopathies can cause an elevated CK level in cats, such as prolonged anorexia.²⁷ Serum concentrations of AST and ALT may also be increased in cats with myopathic disorders.³⁵ Referral to a specialized facility may be necessary for diagnosis of some myopathies and neuromuscular diseases.

Infectious Polymyositis

The most common cause of infectious polymyositis in cats is toxoplasmosis. Affected cats are generally young, and clinical signs include weakness, reluctance to move, and muscle hyperesthesia.²⁴ Systemic signs of infection include anterior uveitis; chorioretinitis; and central nervous system, respiratory tract, and gastrointestinal tract signs. Most cats have fever and weight loss. Hematology and serum chemistry abnormalities include non-regenerative anemia, neutrophilia, lymphocytosis, eosinophilia, hyperglobulinemia, and increases in serum bilirubin and liver enzymes.⁹³ Serum immunoglobulin G (IgG) and immunoglobulin M (IgM) titers are generally positive, although some cats do not develop IgG titers either in the acute or convalescent stages of infection.⁹³

Diagnosis of *Toxoplasma* polymyositis is based on history, clinical signs, minimum laboratory database results, serology, and response to therapy. A definitive diagnosis may be reached with identification of parasites in muscle biopsy samples. The recommended treatment for toxoplasmosis in cats is clindamycin (10 mg/kg orally, every 8 hours for at least 4 weeks).²⁴

Burmese Hypokalemic Polymyopathy

Hypokalemia is a well-known cause of muscle weakness in the cat. Hypokalemia may be caused by systemic loss, reduced intake, or a shift of potassium from the extracellular to the intracellular space. The most common cause of hypokalemia, especially in older cats, is chronic kidney disease.²³ Other common causes of hypokalemia include chronic vomiting or diarrhea, hyperthyroidism, and administration of diuretics.

A hereditary disease with a probable autosomal recessive mode of inheritance causing hypokalemia and weakness has been reported in Burmese cats, primarily in the United Kingdom, the Netherlands, Australia, and New Zealand.* Signs are episodic and include acute onset of neck ventroflexion, head nodding, stiff gait, exercise intolerance, and weakness. Severely affected cats are reluctant to move and have myalgia. Exercise and stressors may induce clinical signs. Death resulting from cardiac arrest or respiratory paralysis has been reported.⁷⁷ Affected cats are typically from 2 to 12 months of age (mean age, 7.4 months) and are usually normal between episodes. There is no gender predisposition.

The molecular basis of the defect has not been determined but is most likely a channelopathy similar to hypokalemic periodic paralysis in humans.⁴² Electromyography and muscle biopsies are normal in affected cats.⁴² During episodes of clinical signs, serum potassium is decreased (<3.0 mEq/L) and serum CK is

*References 9, 25, 42, 57, 66, 77.



FIGURE 26-20 The prominent shoulder blades and neck ventroflexion characteristic of Devon Rex myopathy.

increased (often >100,000 IU/L). Clinical signs respond to potassium supplementation (potassium gluconate, typically 2 to 4 mmol/cat/day, orally but higher doses are required in some cats). Some affected cats appear to improve spontaneously.

Devon Rex and Sphynx Myopathy

A hereditary myopathy with presumed autosomal recessive inheritance has been reported in Devon Rex and Sphynx cats.^{75,76,92,98} The condition came to light in the 1970s and was first called "spasticity." Affected cats have been reported in the United Kingdom, Australia, New Zealand, the United States, and other countries. Clinical signs typically develop between 4 to 7 weeks of age but may not appear until 3 months or later. Signs often fluctuate in severity. The condition does not appear to be painful. Affected kittens have a peculiar high-stepping gait, with the shoulder blades held high and the neck ventroflexed, often with the head tucked into the sternum (Figure 26-20). When resting, the head is laid to one side. Generalized weakness and exercise intolerance are seen in moderately to severely affected cats and may be provoked by exertion, stress, concurrent disease, or excitement. A characteristic "dog-begging" or "chipmunk" position is adopted where the front legs are rested on an object and the head is held erect (Figure 26-21). The head may be rested on elevated objects for support when sitting or lying down. Some cats have difficulty prehending food, because of both oropharyngeal weakness and the abnormal head position. Severely affected cats may develop megaesophagus. The condition eventually stabilizes as the kitten matures and learns to cope with the disability.⁷⁵

Diagnosis is suspected on signalment and history and ruling out of other common causes of weakness, such as hypokalemia. The results of common laboratory tests, including serum CK concentrations, are usually within normal limits. Electrodiagnostic studies may be within normal limits, and examination of the central and



FIGURE 26-21 The characteristic "dog-begging" or "chipmunk" position adopted by Devon Rex cats with myopathy is shown in the cat on the *left*, compared to the normal littermate on the *right*.

peripheral nervous system is normal. Definitive diagnosis is established by examination of muscle biopsies histopathologically and with histochemistry and immunofluorescence staining.⁷⁶ Sites recommended for biopsy are the triceps brachii and dorsal cervical muscles.⁷⁵ Histopathologic examination of specimens shows changes consistent with a dystrophy (e.g., variation in muscle fiber size, internal nuclei, myofiber degeneration and regeneration, muscle atrophy and hypertrophy, and fibrosis).^{75,93} Recent studies have characterized this disease as a novel dystroglycanopathy, with loss of natively glycosylated alpha-dystroglycan.⁷⁶ A causative genetic mutation has not yet been identified.

There is no specific treatment for this disease. Feeding affected cats by hand or from an elevated position is recommended because the major cause of death is aspiration pneumonia or obstruction of the pharynx or larynx with food. Environmental modifications and avoidance of stressors and exertion may also be helpful.

Glycogen Storage Disease Type IV in Norwegian Forest Cats

A myopathy was first recognized in Norwegian Forest cats in 1992 and identified as glycogen storage disease (GSD) type IV; it is inherited in an autosomal recessive manner.^{33,34,35} There are many types of GSDs reported in the literature, most affecting humans. The disease in

Norwegian Forest cats is due to a deficiency of glycogen branching enzyme (GBE) and characterized by accumulation of abnormal glycogen in several tissues, including skeletal muscle, hepatocytes, and neurons.

The prognosis is very poor for affected cats because there is no effective treatment. Many kittens are stillborn or die within the first few days of life. Those that survive will generally develop clinical signs between 5 and 7 months of age, including persistently elevated body temperature, generalized muscle tremors, intermittent lethargy, and a "bunny hopping" type of movement.³⁵ Muscle weakness and muscle atrophy progress rapidly, resulting in the inability to chew, contractures of certain joints, and quadriplegia. Death typically occurs before 1 year of age.

Routine laboratory testing is not helpful in diagnosis, although increases in serum CK and ALT are commonly seen. Further diagnostic studies include nerve conduction velocities (typically normal) and electromyography. Samples obtained through biopsy or during necropsy show cytoplasmic inclusions containing periodic acid-Schiff and toluidine blue positive material. Nervous tissue and skeletal and cardiac muscle are most affected. A genetic test is available using buccal swabs or whole blood from PennGen Laboratories, at the University of Pennsylvania.

When an affected kitten is identified, it means that both parents are carriers of the defective gene, which has implications for future breeding. Ideally, carrier cats would be removed from breeding programs. In a recent report, 402 privately owned Norwegian Forest cats were tested, with 58 carriers and four affected cats identified.³⁴

Myositis Ossificans

Myositis ossificans (fibrodysplasia ossificans) is a rare disorder generally reported in young cats.^{2,85,102,104,106} The disease is characterized by ossification of skeletal muscle-associated connective tissue and adjacent skeletal muscle. Similar disorders are reported in humans and pigs. The disease appears to be inherited in an autosomal dominant manner in those species, but the etiology in cats is unknown. Typically, disease progression is rapid (over a few weeks to a few months), and most affected cats are euthanized. Clinical signs include muscle enlargement and limb stiffness that progresses and becomes painful with decreased range of motion. Fever and lymphadenopathy have also been reported.¹⁰² The results of routine laboratory tests are within normal limits; serum CK may be normal or elevated. Diagnosis is based on clinical signs, radiographic findings (mineralization of skeletal muscles), and histopathologic examination of muscle biopsy specimens (fibrosis, ossification without inflammation). No effective treatment for myositis ossificans in cats has been identified; therapy with

vitamin E, selenium, prednisone, and etretinate have all proved ineffective.

References

1. Allan GS: Radiographic features of feline joint diseases, *Vet Clin North Am Small Anim Pract* 30:281, 2000.
2. Asano K, Sakata A, Shibuya H et al: Fibrodysplasia ossificans progressiva-like condition in a cat, *J Vet Med Sci* 68:1003, 2006.
3. Beale BS: Orthopedic problems in geriatric dogs and cats, *Vet Clin North Am Small Anim Pract* 35:655, 2005.
4. Beck AL, Pead MJ, Draper E: Regional load bearing of the feline acetabulum, *J Biomech* 38:427, 2005.
5. Becker K, Brown N, Denardo G: Polyarthropathy in a cat seropositive for feline syncytial-forming virus and feline immunodeficiency virus, *J Am Anim Hosp Assoc* 30:225, 1994.
6. Bender JB, Torres SM, Gilbert SM et al: Isolation of methicillin-resistant *Staphylococcus aureus* from a non-healing abscess in a cat, *Vet Rec* 157:388, 2005.
7. Bennett D, Nash AS: Feline immune-based polyarthritis: a study of thirty-one cases, *J Small Anim Pract* 29:501, 1988.
8. Bitetto WV, Patnaik AK, Schrader SC et al: Osteosarcoma in cats: 22 cases (1974-1984), *J Am Vet Med Assoc* 190:91, 1987.
9. Blaxter A, Lievesley P, Gruffydd-Jones T et al: Periodic muscle weakness in Burmese kittens, *Vet Rec* 118:619, 1986.
10. Bradley WA: Metaphyseal osteomyelitis in an immature Abyssinian cat, *Aust Vet J* 81:608, 2003.
11. Burke J: Physeal dysplasia with slipped capital femoral epiphysis in a cat, *Can Vet J* 44:238, 2003.
12. Calvo I, Farrell M, Chase D et al: Carpal arthrodesis in cats. Long-term functional outcome, *Vet Comp Orthop Traumatol* 22:498, 2009.
- 12a. Carroll GL, Simonson SM: Recent developments in nonsteroidal antiinflammatory drugs in cats, *J Am Anim Hosp Assoc* 41:347, 2005.
13. Chandler JC, Beale BS: Feline orthopedics, *Clin Tech Small Anim Pract* 17:190, 2002.
14. Chang J, Jung J, Oh S et al: Osteochondrodysplasia in three Scottish Fold cats, *J Vet Sci* 8:307, 2007.
15. Clarke SP, Bennett D: Feline osteoarthritis: a prospective study of 28 cases, *J Small Anim Pract* 47:439, 2006.
16. Clarke SP, Mellor D, Clements DN et al: Prevalence of radiographic signs of degenerative joint disease in a hospital population of cats, *Vet Rec* 157:793, 2005.
17. Colopy-Poulsen S, Danova N, Hardie R et al: Managing feline obstipation secondary to pelvic fractures, *Comp Cont Edu Pract Vet* 27:662, 2005.
18. Connery NA, Rackard S: The surgical treatment of traumatic stifle disruption in a cat, *Vet Comp Orthop Traumatol* 13:208, 2000.
19. Craig L: Physeal dysplasia with slipped capital femoral epiphysis in 13 cats, *Vet Pathol* 38:92, 2001.
20. Culvenor JA, Black AP, Lorkin KF et al: Repair of femoral capital physeal injuries in cats: 14 cases, *Vet Comp Orthop Traumatol* 9:182, 1996.
21. Dawson S, Bennett D, Carter SD et al: Acute arthritis of cats associated with feline calicivirus infection, *Res Vet Sci* 56:133, 1994.
22. Dimopoulos M, Kirpenseijn J, Moens H et al: Histologic prognosticators in feline osteosarcoma: a comparison with phenotypically similar canine osteosarcoma, *Vet Surg* 37:466, 2008.
23. Dow S, Fettman M, Curtis C et al: Hypokalemia in cats: 186 cases (1984-1987), *J Am Vet Med Assoc* 194:1604, 1989.
24. Dubey JP, Lappin MR: Toxoplasmosis and neosporosis. In Greene CE, editor: *Infectious diseases of the dog and cat*, ed 3, St Louis, 2006, Saunders Elsevier, p 754.
25. Edwards CM, Belford CJ: Hypokalemic polymyopathy in Burmese cats, *Aust Vet Pract* 25:58, 1995.

26. Evasion MD, Taylor SM, Bebchuk TN: Suspect osteogenesis imperfecta in a male kitten, *Can Vet J* 48:296, 2007.
27. Fascetti A, Mauldin G, Mauldin G: Correlation between serum creatinine kinase activities and anorexia in cats, *J Vet Intern Med* 11:9, 1997.
28. Feldman D: Glucocorticoid-responsive, idiopathic, nonerosive polyarthritis in a cat, *J Am Anim Hosp Assoc* 30:42, 1994.
29. Fischer HR, Norton J, Kobluk CN et al: Surgical reduction and stabilization for repair of femoral capital physeal fractures in cats: 13 cases (1998-2002), *J Am Vet Med Assoc* 224:1478, 2004.
30. Flo GL, Stickle RL, Dunstan RW: Synovial chondrometaplasia in five dogs, *J Am Vet Med Assoc* 191:1417, 1987.
31. Freshwater A: Why your housecat's trite little bite could cause you quite a fright: a study of domestic felines on the occurrence and antibiotic susceptibility of *Pasteurella multocida*, *Zoonoses Public Health* 55:507, 2008.
32. Frimberger A: Principles of chemotherapy. In Ettinger S, Feldman EC, editors: *Textbook of veterinary internal medicine*, ed 6, St Louis, 2005, Saunders Elsevier, p 708.
33. Fyfe JC, Giger U, Van Winkle TJ et al: Glycogen storage disease type IV: inherited deficiency of branching enzyme activity in cats, *Pediatr Res* 32:719, 1992.
34. Fyfe JC, Kurzhals RL, Hawkins MG et al: A complex rearrangement in GBE1 causes both perinatal hypoglycemic collapse and late-juvenile-onset neuromuscular degeneration in glycogen storage disease type IV of Norwegian forest cats, *Mol Genet Metab* 90:383, 2007.
35. Gaschen F, Jaggy A, Jones B: Congenital diseases of feline muscle and neuromuscular junction, *J Feline Med Surg* 6:355, 2004.
36. Godfrey DR: Osteoarthritis (OA) in cats: a retrospective series of 31 cases, *J Small Anim Pract* 43:260, 2002.
37. Godfrey DR: Osteoarthritis in cats: a retrospective series of 31 cases, *J Small Anim Pract* 43:260, 2002.
38. Goodman LA, et al: In vivo effects of firocoxib, meloxicam and tepoxalin administration on eicosanoid production in target tissues of normal cats (abstract), *J Vet Intern Med* 23:767, 2009.
39. Gradner G, Weissenbock H, Kneissl S et al: Use of latissimus dorsi and abdominal external oblique muscle for reconstruction of a thoracic wall defect in a cat with feline osteochondromatosis, *J Feline Med Surg* 10:88, 2008.
40. Graham-Mize CA, Rosser EJ, Hauptman J: Absorption, bioavailability and activity of prednisone and prednisolone in cats. In Hillier A, Foster A, Bertola G, editors: *Advances in veterinary dermatology*, Ames, Iowa, 2005, Blackwell Publishing, p 152.
41. Gregory SP, Pearson GR: Synovial osteochondromatosis in a Labrador retriever bitch, *J Small Anim Pract* 31:580, 1990.
42. Gruffydd-Jones T, Sparkes AH, Caney SA et al: Hypokalaemic episodic weakness in Burmese kittens (abstract), *J Vet Intern Med* 10:175, 1996.
43. Gunew MN, Menrath VH, Marshall RD: Long-term safety, efficacy and palatability of oral meloxicam at 0.01-0.03 mg/kg for treatment of osteoarthritic pain in cats, *J Feline Med Surg* 10:235, 2008.
44. Hanna FY: Disease modifying treatment for feline rheumatoid arthritis, *Vet Comp Orthop Traumatol* 18:94, 2005.
45. Harasen G: Atraumatic proximal femoral physeal fractures in cats, *Can Vet J* 45:359, 2004.
46. Harasen G: Good stuff for joints!, *Can Vet J* 46:933, 2005.
47. Harasen G: Maxillary and mandibular fractures, *Can Vet J* 49:819, 2008.
48. Harasen G: Feline orthopedics, *Can Vet J* 50:669, 2009.
49. Harasen GL: Feline cranial cruciate rupture: 17 cases and a review of the literature, *Vet Comp Orthop Traumatol* 18:254, 2005.
50. Hardie EM: Management of osteoarthritis in cats, *Vet Clin North Am Small Anim Pract* 27:945, 1997.
51. Hardie EM, Roe SC, Martin FR: Radiographic evidence of degenerative joint disease in geriatric cats: 100 cases (1994-1997), *J Am Vet Med Assoc* 220:628, 2002.
52. Heldmann E, Anderson M, Wagner-Mann C: Feline osteosarcoma: 145 cases (1990-1995), *J Am Anim Hosp Assoc* 36:518, 2000.
53. Houlton J, McGlennon N: Castration and physeal closure in the cat, *Vet Rec* 131:466, 1992.
54. Hubler M, Volkert M, Kaser-Hotz B et al: Palliative irradiation of Scottish Fold osteochondrodysplasia, *Vet Radiol Ultrasound* 45:582, 2004.
55. Janssens LAA, Janssens GO, Janssens DL: Anterior cruciate ligament rupture associated with cardiomyopathy in three cats, *Vet Comp Orthop Traumatol* 4:35, 1991.
56. Johnson KA, Watson ADJ: Skeletal diseases. In Ettinger S, editor: *Textbook of veterinary internal medicine*, ed 6, St Louis, 2005, Saunders Elsevier, p 1958.
57. Jones BR, Swinney GW, Alley MR: Hypokalaemic myopathy in Burmese kittens, *NZ Vet J* 36:150, 1988.
58. Keller GG, Reed AL, Lattimer JC et al: Hip dysplasia: a feline population study, *Vet Radiol Ultrasound* 40:460, 1999.
59. L'Eplattenier H, Montavon P: Patellar luxation in dogs and cats: management and prevention, *Comp Contin Edu Pract Vet* 24:292, 2002.
60. L'Eplattenier H, Montavon P: Patellar luxation in dogs and cats: pathogenesis and diagnosis, *Comp Contin Edu Pract Vet* 24:234, 2002.
61. Langenbach A, Giger U, Green P et al: Relationship between degenerative joint disease and hip joint laxity by use of distraction index and Norberg angle measurement in a group of cats, *J Am Vet Med Assoc* 213:1439, 1998.
62. Langley-Hobbs SJ: Survey of 52 fractures of the patella in 34 cats, *Vet Rec* 164:80, 2009.
63. Langley-Hobbs SJ, Brown G, Matis U: Traumatic fracture of the patella in 11 cats, *Vet Comp Orthop Traumatol* 21:427, 2008.
64. Langley-Hobbs SJ, Sissener TR, Shales CJ: Tension band stabilisation of acetabular physeal fractures in four kittens, *J Feline Med Surg* 9:177, 2007.
65. Langley-Hobbs SJ, Straw M: The feline humerus: an anatomical study with relevance to external skeletal fixator and intramedullary pin placement, *Vet Comp Orthop Traumatol* 18:1, 2005.
66. Lantinga E, Kooistra HS, van Nes JJ: [Periodic muscle weakness and cervical ventroflexion caused by hypokalemia in a Burmese cat], *Tijdschr Diergeneesk* 123:435, 1998.
67. Lascelles BD, Court MH, Hardie EM et al: Nonsteroidal anti-inflammatory drugs in cats: a review, *Vet Anaesth Analg* 34:228, 2007.
68. Levitin B, Aroch I, Aizenberg I et al: Linear osteochondromatosis in a cat, *Vet Radiol Ultrasound* 44:660, 2003.
69. Lim S, Hossain MA, Park J et al: The effects of enrofloxacin on canine tendon cells and chondrocytes proliferation in vitro, *Vet Res Commun* 32:243, 2008.
70. Liska WD, Doyle N, Marcellin-Little DJ et al: Total hip replacement in three cats: surgical technique, short-term outcome and comparison to femoral head ostectomy, *Vet Comp Orthop Traumatol* 22:505, 2009.
71. Lockwood A, Montgomery R, McEwen V: Bilateral radial hemimelia, polydactyly and cardiomegaly in two cats, *Vet Comp Orthop Traumatol* 22:511, 2009.
72. Loder RT, Aronsson DD, Dobbs MB et al: Slipped capital femoral epiphysis, *J Bone Joint Surg Am* 82:1170, 2000.
73. Loughlin C, Kerwin S, Hosgood G et al: Clinical signs and results of treatment in cats with patellar luxation: 42 cases (1992-2002), *J Am Vet Med Assoc* 228:1370, 2006.
74. Malik R, Allan G, Howlett C et al: Osteochondrodysplasia in Scottish Fold cats, *Aust Vet J* 77:85, 1999.
75. Malik R, Mepstead K, Yang F et al: Hereditary myopathy of Devon Rex cats, *J Sm Anim Pract* 34:539, 1993.

76. Martin PT, Shelton GD, Dickinson PJ et al: Muscular dystrophy associated with [alpha]-dystroglycan deficiency in Sphynx and Devon Rex cats, *Neuromuscular Disord* 18:942, 2008.
77. Mason K: A hereditary disease in Burmese cats manifested as an episodic weakness with head nodding and neck ventroflexion, *J Am Anim Hosp Assoc* 24:147, 1988.
78. Mathews KG, Koblik PD, Knoeckel MJ et al: Resolution of lameness associated with Scottish fold osteodystrophy following bilateral ostectomy and pantarsal arthrodeses: a case report, *J Am Anim Hosp Assoc* 31:280, 1995.
79. McNicholas WT, Jr, Wilkens BE, Blevins WE et al: Spontaneous femoral capital physeal fractures in adult cats: 26 cases (1996–2001), *J Am Vet Med Assoc* 221:1731, 2002.
80. Mollenhoff A, Nolte I, Kramer S: Anti-nociceptive efficacy of carprofen, levomethadone and buprenorphine for pain relief in cats following major orthopedic surgery, *J Vet Med Series A* 52:186, 2005.
81. Muqhamnam A, Reinke J: Avulsion of the gastrocnemius tendon in three cats, *J Am Anim Hosp Assoc* 30:550, 1994.
82. Neil K, Caron J, Orth M: The role of glucosamine and chondroitin sulfate in treatment for and prevention of osteoarthritis in animals, *J Am Vet Med Assoc* 226:1079, 2005.
83. Newitt ALM, German AJ, Barr FJ: Lumbosacral transitional vertebrae in cats and their effects on morphology of adjacent joints, *J Feline Med Surg* 11:941, 2009.
84. Nolte D, Fusco J, Peterson M: Incidence of and predisposing factors for nonunion of fractures involving the appendicular skeleton in cats: 18 cases (1998–2002), *J Am Vet Med Assoc* 226:77, 2005.
85. Norris A, Pallett L, Wilcock B: Generalized myositis ossificans in a cat, *J Amer Anim Hosp Assoc* 16:659, 1980.
86. Owen MR, Langley-Hobbs SJ, Moores AP et al: Mandibular fracture repair in dogs and cats using epoxy resin and acrylic external skeletal fixation, *Vet Comp Orthop Traumatol* 17:189, 2004.
87. Ozaras R, Mert A, Tahan V et al: Ciprofloxacin and Achilles' tendon rupture: a causal relationship, *Clin Rheumatol* 22:500, 2003.
88. Queen J, Bennett D, Carmichael S et al: Femoral neck metaphyseal osteopathy in the cat, *Vet Rec* 142:159, 1998.
89. Reinke J, Muqhamnam A: Meniscal calcification and ossification in six cats and two dogs, *J Am Anim Hosp Assoc* 30:145, 1994.
90. Robertson S, Taylor P: Pain management in cats—past, present and future. Part 2. Treatment of pain—clinical pharmacology, *J Fel Med Surg* 6:321, 2004.
91. Robertson SA: Managing pain in feline patients, *Vet Clin North Am Sm Anim Pract* 38:1267, 2008.
92. Robinson R: "Spasticity" in the Devon rex cat, *Vet Rec* 130:302, 1992.
93. Ruehlman D: Myopathic disorders. In August J, editor: *Consultations in feline internal medicine*, ed 6, St Louis, 2010, Saunders Elsevier, p 602.
94. Scavelli T, Schrader S: Nonsurgical management of rupture of the cranial cruciate ligament in 18 cats, *J Am Anim Hosp Assoc* 23:337, 1987.
95. Schnabl E, Reese S, Lorinson K et al: Measurement of the tibial plateau angle in cats with and without cranial cruciate ligament rupture, *Vet Comp Orthop Traumatol* 22:83, 2009.
96. Scott H, McLaughlin R: *Feline orthopedics*, London, 2007, Manson Publishing Ltd.
97. Shakibaei M, de Souza P, van Sickle D et al: Biochemical changes in Achilles tendon from juvenile dogs after treatment with ciprofloxacin or feeding a magnesium-deficient diet, *Arch Toxicol* 75:369, 2001.
98. Shelton DG, Sturges BK, Lyons LA et al: Myopathy with tubulin-reactive inclusions in two cats, *Acta Neuropathol* 114:537, 2007.
99. Smith G, Langenbach A, Green P et al: Evaluation of the association between medial patellar luxation and hip dysplasia in cats, *J Am Vet Med Assoc* 215:40, 1999.
- 99a. Sparkes AH, Heiene R, Lascelles BDX et al: ISFM and AAFF consensus guidelines: long-term use of NSAIDs in cats, *J Feline Med Surg* 12:521, 2010.
100. Taylor P, Robertson S: Pain management in cats—past, present and future. Part 1. The cat is unique, *J Fel Med Surg* 6:313, 2004.
101. Towle H, Breur G: Dysostoses of the canine and feline appendicular skeleton, *J Am Vet Med Assoc* 225:1685, 2004.
102. Valentine B, George C, Randolph J et al: Fibrodysplasia ossificans progressive in the cat: a case report, *J Vet Intern Med* 6:335, 1992.
103. Wallace AM, De La Puerta B, Trayhorn D et al: Feline combined diaphyseal radial and ulnar fractures. A retrospective study of 28 cases, *Vet Comp Orthop Traumatol* 22:38, 2009.
- 103a. Wallace J: Meloxicam, *Comp Contin Edu Pract Vet* 25:64, 2003.
104. Warren H, Carpenter J: Fibrodysplasia ossificans in three cats, *Vet Pathol* 21:495, 1984.
105. Williams RJ 3rd, Attia E, Wickiewicz TL et al: The effect of ciprofloxacin on tendon, paratenon, and capsular fibroblast metabolism, *Am J Sports Med* 28:364, 2000.
106. Yabuzoe A, Yokoi S, Sekiguchi M et al: Fibrodysplasia ossificans progressiva in a Maine Coon cat with prominent ossification in dorsal muscle, *J Vet Med Sci* 71:1649, 2009.
107. Zeugswetter F, Hittmair KM, de Arepacochaga AG et al: Erosive polyarthritis associated with *Mycoplasma gateae* in a cat, *J Feline Med Surg* 9:226, 2007.