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CHAPTER 80

Diseases of Joints and Ligaments

Michael P. Kowaleski

CONGENITAL/DEVELOPMENTAL DISORDERS

Avascular Necrosis of the Femoral Head (Legg-Calvé-Perthes Disease)

Definition and Causes

I. It is defined as noninflammatory, aseptic necrosis of the femoral head that occurs predominantly in adolescent, small breed dogs.
II. Interruption of blood flow to the femoral head occurs for an unknown reason.
III. Possible causes include hereditary factors, increased intra-articular pressure, infarction of the femoral head, hormonal factors, and anatomic conformation.
IV. A recessive mode of inheritance has been proposed in the Manchester terrier (Vasseur et al., 1989).

Pathophysiology

I. Initial change is osteonecrosis of the proximal femoral epiphysis, with viable articular cartilage.
II. Continued weight bearing results in microfractures of the necrotic trabeculae, subchondral bone collapse, subchondral cleft formation, and ischemic damage to the physis.
III. Normal metaphyseal bone responds and revascularization of the femoral head occurs; however, the femoral epiphysis is mechanically weakened and susceptible to collapse and fragmentation under normal physiologic loads.
IV. Resulting deformity of the femoral head leads to joint incongruity and osteoarthritis of the hip joint.

Clinical Signs

I. Initial sign is progressive pelvic limb lameness unassociated with trauma in a young, small-breed dog.
   A. Lameness slowly worsens over 6 to 8 weeks and may progress to a nonweight bearing.
   B. Acute-onset lameness is presumably related to a previously undiagnosed mild lameness that acutely progresses from sudden collapse of the epiphysis.
II. Average age at onset is 5 to 8 months (range of 3 to 13 months) (Gambardella, 1993).
III. Bilateral in 12% to 17% of cases (Gambardella, 1993).

Diagnosis

I. Physical examination reveals discomfort with flexion, extension, rotation, and abduction of the hip joint.
II. Radiography is essential to establish a diagnosis.
   A. Incongruency and widening of the coxofemoral joint space progressing to subluxation
   B. Flattening of the femoral head
   C. Foci of decreased density of the femoral head and neck (moth-eaten appearance)
   D. Irregular indentation of the subchondral bone of the femoral head, periarticular osteophyte formation
III. Histopathologic examination of the resected femoral head and neck is confirmatory.

Differential Diagnosis

I. Medial patellar luxation
II. Proximal femoral physeal fracture
III. Traumatic hip luxation
IV. Canine hip dysplasia

Treatment

I. Femoral head and neck excision is the treatment of choice.
II. Total hip replacement can be considered, depending on the size of the dog.
III. In bilateral cases the worst hip (clinically) is operated on first, followed by the second hip (generally in 4 to 6 weeks).

Monitoring of Animal

I. Postoperatively, low-impact activities are initiated that encourage use of the limb (e.g., leash walking, swimming).
II. Passive range-of-motion exercises may be needed if weight bearing does not begin within 10 to 14 days.
III. Radiographs are repeated if lameness develops in the opposite pelvic limb.

Patellar Luxation

Definition and Causes

I. Patellar luxation is intermittent or permanent displacement of the patella from the femoral trochlear sulcus.
II. It is associated with a malalignment of the quadriceps muscles, patella, and patellar tendon, as well as femoral and/or tibial deformities.

Pathophysiology

I. Medial malalignment of the quadriceps mechanism in growing dogs with medial patellar luxation can cause sufficient pressure on the medial distal femoral growth plate to retard growth, whereas reduced pressure laterally allows accelerated growth.
II. Deformities associated with medial luxation include distal femoral varus, external femoral torsion, and medial displacement of the tibial tuberosity.
III. Patellar luxation causes reduced pressure on the trochlear sulcus of the femur, allowing excessive growth of the articular cartilage and underlying bone, leading to a shallow or absent trochlear groove.
IV. Lateral luxation is associated with coxa valga (excessive angle of inclination of the femoral neck) and excessive anteversion angle (external rotation of the proximal femur with respect to the distal femur).

Clinical Signs

I. Medial luxation is a developmental disorder in small-breed dogs, may occur secondary to trauma in any breed, and is increasing in frequency in large-breed dogs.
II. Lateral luxation is seen most commonly in large-breed dogs.
III. Most animals have intermittent weight-bearing lameness and occasionally hold the limb in a flexed position for several steps.

Diagnosis

I. Grade I
   A. Patella may be luxated manually with the joint in extension, but reduction occurs when digital pressure is released.
   B. It is usually an incidental finding but may be associated with occasional lameness.
II. Grade II
   A. Patella can be luxated manually with the joint in extension, especially with rotation of the foot in the direction of luxation.
   B. Reduction occurs with flexion and rotation of the foot opposite to the direction of luxation.
   C. Patella luxates spontaneously and intermittently, and the gait ranges from "skipping" to non–weight-bearing.
III. Grade III
   A. Patella is permanently luxated; manual reduction is possible, but reluxation occurs when pressure is released or the stifle is flexed.
   B. Lameness ranges from weight-bearing to non–weight-bearing.
IV. Grade IV
   A. Patella is permanently luxated and cannot be manually reduced.
   B. Limb is non–weight-bearing if unilateral or the animal moves in a crouched posture if bilateral.

Differential Diagnosis

I. Avascular necrosis of the femoral head
II. Coxofemoral luxation
III. Cranial cruciate ligament rupture
IV. Distal femoral physeal fracture

Treatment

I. Grade I patellar luxations do not require surgical treatment unless lameness is frequent or progressive.
II. Grade II luxations commonly require surgery if episodes of lameness are frequent or severe.
   A. A combination of surgical procedures is performed to address each abnormality present.
   B. Deepening of the femoral trochlear sulcus is accomplished using trochlear wedge recession, trochlear block recession, trochleoplasty, or trochlear chondroplasty (Slocum and Slocum, 1993; Johnson et al., 2001).
   C. Tibial tuberosity transposition is done to align the quadriceps mechanism.
   D. Joint capsule imbrication is performed on the side opposite the luxation.
   E. Retinacular and joint capsule–releasing incision is made on the side of the luxation.
III. Grade III luxations require a combination of the previously discussed procedures and possibly femoral and/or tibial corrective osteotomy to address angular or torsional deformities.
IV. Grade IV luxations often also require corrective osteotomy of the femur and/or tibia.
   A. Prognosis for return of pet to a functional level is fair to good.
   B. Stifle joint arthrodesis may be required with poor return to function.

Monitoring of Animal

I. Postoperatively, low-impact activities are initiated that encourage use of the limb (e.g., leash walking, swimming).
II. Passive range-of-motion exercises may be needed if weight bearing does not begin within 10 to 14 days.
III. Physical examinations are repeated at 2 and 6 weeks to assess limb function and ensure the patella is stable.
IV. Radiographs are obtained to assess bony healing at 6 to 8 weeks.

Shoulder and Elbow Luxation

Definition and Causes

I. Congenital shoulder and elbow luxation are likely hereditary in origin.
II. Luxation of the shoulder or elbow can occur secondary to trauma.
III. Congenital medial shoulder luxation occurs most frequently in small and miniature breed dogs (e.g., Shetland sheepdog, toy poodle).
IV. Congenital elbow luxation has been reported in the Boston terrier, Yorkshire terrier, English bulldog, miniature poodle, Pomeranian, and pug.

Pathophysiology

I. The cause of congenital luxations is not known.
II. Congenital or developmental joint laxity may lead to luxation and hypoplastic joint surfaces.

Clinical Signs

I. Medial shoulder luxation: congenital or traumatic
   A. Affected limb is carried in flexion with foot rotated outward.
   B. Greater tubercle is palpable medial to the normal position.
II. Lateral shoulder luxation
   A. It is generally traumatic; large breeds are affected most.
   B. Affected leg is carried in flexion with foot rotated internally.
   C. Greater tubercle is palpable lateral to the normal position.
III. Elbow luxation
   A. Congenital elbow luxation results in lateral rotation of the proximal ulna, as well as subluxation or luxation of the humeroulnar joint.
   B. Most traumatic elbow luxations are lateral because of the large medial epicondylar ridge.
   C. Limb is abducted, externally rotated, and held in slight flexion.
IV. Congenital forms: possibly bilateral

Diagnosis

I. Malarticulation and displacement of normal bony landmarks are classic findings.
II. Diagnosis is confirmed radiographically.
III. Additional radiographic findings are flattening of the joint surfaces and angular or torsional deformities of the humerus, ulna, or radius.

Differential Diagnosis

I. Fractures of elbow or shoulder
II. Physeal trauma: fracture or growth deformity

Treatment

I. Congenital shoulder and elbow luxations
   A. Closed reduction and coaptation are generally unsuccessful.
   B. Surgical reduction and stabilization may result in acceptable joint function.
   C. Closed reduction and percutaneous fixation have been described in dogs (Dassler and Vasseur, 2003).
   D. Arthrodesis may be required if joint function is unacceptable.
II. Traumatic medial shoulder luxation
   A. Conservative management
      1. Closed reduction and cage rest for 2 to 3 weeks
      2. Reduction and placement of the limb in a Velpeau sling for 2 to 3 weeks to distract the humeral head laterally
   B. Surgical management: medial transfer of biceps brachii tendon and joint capsule imbrication
   C. Salvage procedures: excision arthroplasty of the humeral head, resection of the glenoid, shoulder arthrodesis
III. Traumatic lateral shoulder luxation
   A. Conservative management
      1. Closed reduction and cage rest for 2 to 3 weeks
      2. Closed reduction and placement of the limb in a spica splint or sling for 2 to 3 weeks
   B. Surgical management
      1. Lateral transfer of biceps brachii tendon and joint capsule imbrication
      2. Prosthetic glenohumeral ligament reconstruction using nonabsorbable suture material and bone anchors or tunnels in the scapular and humeral neck
   C. Salvage procedures: excision arthroplasty of humeral head, resection of glenoid, shoulder arthrodesis
IV. Traumatic elbow luxation
   A. Conservative management
      1. Closed reduction under general anesthesia
      2. Application of spica splint for 10 to 14 days
   B. Surgical management
      1. Open reduction is indicated if instability is present after closed reduction or for chronic or irreducible elbow luxations.
      2. Torn collateral ligaments may be primarily repaired, and a screw, washer, and heavy-gauge suture may be used to create a prosthetic collateral ligament.
      3. Spica splint is applied for 10 to 14 days.

Monitoring of Animal

I. Passive range-of-motion exercises are initiated, followed by low-impact activity once the coaptation has been removed.
II. Radiographs are repeated in 4 to 6 weeks to assess status of joint reduction and stability of implants.

Canine Elbow Dysplasia

Definition and Causes

I. Canine elbow dysplasia is a group of developmental diseases that researchers believe is caused by incongruity between the humerus, radius, and ulna.
II. Manifestations include fragmented medial coronoid process (FMCP) of the ulna, ununited anconeal process (UAP) of the ulna, and osteochondritis dissecans (OCD) of the medial portion of the humeral condyle.
III. In the past, decreased radius of curvature of the ulnar trochlear notch was the suggested cause.
IV. Recently, elbow incongruity from asynchronous growth between the radius and ulna has been implicated as the cause (Schultz and Krotscheck, 2003).

Pathophysiology

I. Lagging radial growth and a relatively long ulna may stress the medial coronoid process and humeral condyle, resulting in FMCP and, potentially, OCD of the humeral condyle.
II. A relatively long radius may increase the load on the anconeal process, preventing normal fusion of this center of ossification.
III. Dogs with unilateral UAP have a slightly longer radius on the affected side (Sjöström et al., 1995).

Clinical Signs

I. FMCP and OCD
  A. Thoracic limb lameness, initially evident at 5 to 8 months of age, is seen in retrievers, Bernese mountain dogs, and rottweilers.
  B. Dogs may not be presented until 1 to 2 years of age, after development of osteoarthritis.
  C. Lameness is worse after exercise, and joint effusion and periarticular fibrosis may be palpable.
    1. Lameness is usually unilateral, even if both joints are affected.
    2. Stiff or stilted gait may be observed if bilateral lameness is present (shortened stride).
  D. Pain is evident with supination or pronation of the pes, simultaneous flexion and extension of the joint, and palpation of the medial coronoid process.

II. UAP
  A. The anconeal process normally fuses with the proximal ulna by 20 to 24 weeks of age, so diagnosis of UAP before this may be premature.
  B. Large-breed dogs, especially the German shepherd dog, basset hound, and St. Bernard are affected.
  C. Signs of thoracic limb lameness, elbow abduction, and external rotation of the foot become apparent at 5 to 8 months of age.
  D. Crepitus, joint effusion, and periarticular thickening of the elbow may be noted.

Diagnosis

I. FMCP
  A. Presumptive diagnosis is based on typical history and clinical signs.
  B. Radiographs of both elbows are taken, because the disease is commonly bilateral.
    1. Radiographic findings are nonspecific; presence of osteoarthritis is suggestive of FMCP.
    2. Osteophytosis and superimposition of the radial head on the coronoid process make identification of the FMCP difficult.
    3. Obtain lateral and craniocaudal views; a flexed lateral view exposes the anconeal process to assess for osteophytosis.
    4. The craniolateral-caudomedial −15-degree oblique view (with 30 degrees of flexion) may help visualize the medial coronoid.
    5. Blunting of the coronoid, visible fragments, and osteophytes of coronoid process, anconeal process, or radial head may be visible.
    6. The earliest radiographic changes are osteophytes on the anconeal process.
    7. Later signs include subchondral bone sclerosis, articular and periarticular osteophytosis, joint space narrowing, joint effusion, and periarticular soft-tissue thickening; these signs indicate secondary degenerative joint disease (DJD).
  C. Definitive diagnosis can be made with arthroscopy, computed tomography, magnetic resonance imaging, or arthrotomy.

II. OCD
  A. An oval to triangular subchondral bone defect is evident in the medial portion of the humeral condyle in the craniocaudal radiographic projection.
  B. Earliest radiographic changes are similar to those of FMCP.

III. UAP
  A. Clinical signs, age, and breed allow for a tentative diagnosis.
  B. Flexed, lateral radiograph reveals an irregular radiolucent line between the anconeal process and the olecranon.
  C. Radiographs of the contralateral elbow may be useful for comparison.
  D. Bilateral UAP occurs in 11% to 47% of cases (Schultz and Krotscheck, 2003).

Differential Diagnosis

I. These conditions produce similar signs and can be differentiated radiographically.
II. Combinations of OCD, FMCP, and UAP may occur.
III. Humeroulnar subluxation resulting from premature physeal closure can be differentiated radiographically.
IV. Other diseases of the thoracic limb in young dogs are OCD of the shoulder and panosteitis.

Treatment

I. FMCP and OCD
  A. Medical management
    1. Asymptomatic dogs or those with severe DJD may be treated medically.
    2. Weight control, exercise moderation, nonsteroidal antiinflammatory drugs (NSAIDs), chondroprotective agents, and nutritional management are the cornerstones of treatment.
    3. Rest (2 to 3 weeks) and NSAIDs (Table 80-1) are used during episodes of lameness, followed by return to moderate regular exercise.
  B. Surgical management

TABLE 80-1

Nonsteroidal Antiinflammatory Drugs Approved for the Treatment of Osteoarthritis in Dogs

DRUG (BRAND NAME)	DOSE AND FREQUENCY
Carprofen *(Rimadyl)*	2.2 mg/kg PO BID
Deracoxib *(Deramaxx)*	1 to 2 mg/kg PO SID
Etodolac *(EtoGesic)*	10 to 15 mg/kg PO SID
Firocoxib *(Previcox)*	5 mg/kg PO SID
Meloxicam *(Metacam)*	0.1 mg/kg PO SID
Tepoxalin *(Zubrin)*	10 mg/kg PO SID

1. Indications include persistent lameness and mild DJD.
2. Some dogs with moderate or severe DJD may also benefit from loose fragment removal.
3. The joint is explored either arthroscopically or via arthrotomy; loose fragments of the FMCP or OCD lesion are removed, followed by debridement of the subchondral bone bed.
4. Surgical treatment does not halt the progression of osteoarthritis; therefore continued medical therapy is indicated.

II. UAP
   A. Removal of the anconeal process is an acceptable method of treatment but may lead to elbow instability and osteoarthritis.
   B. Surgical reconstruction and procedures that enhance fusion of the anconeal process may be preferred.
      1. Dynamic proximal ulnar osteotomy may allow fusion of the anconeal process in young (6 to 12 month) dogs.
      2. Dynamic proximal ulnar osteotomy and lag screw fixation may be required in older (>1 year) dogs and in dogs with loose attachment of the anconeal process to the olecranon (Schultz and Krotscheck, 2003).

Monitoring of Animal

I. FMCP and OCD
   A. Exercise is restricted for 4 to 6 weeks, followed by gradual return to normal activity.
   B. Prognosis for full function is guarded because of progressive DJD regardless of treatment.
   C. Most dogs are functional pets and have intermittent lameness.
   D. Osteoarthritis requires lifelong medical management.

II. UAP
   A. A soft, padded bandage is applied for 3 to 5 days from the digits to the middiaphysis proximal to the incision to minimize postoperative swelling.
   B. The dog is confined to leash walks for 6 weeks, then gradually returned to normal activity over the next 6 weeks.
   C. Radiographs are repeated in 4 to 6 weeks to assess fusion of the anconeal process and healing of the ulnar osteotomy (if performed).

Canine Hip Dysplasia

Definition and Causes

I. Canine hip dysplasia is an abnormal development of the hip joint with varying degrees of joint laxity that permits subluxation of the femoral head early in life.

II. Researchers agree that hip dysplasia is genetically mediated; however, the exact mechanism is unknown.

III. Environmental factors, such as increased energy and calcium intake, also play an important role (Todhunter and Lust, 2003).

Pathophysiology

I. If muscle development and rate of growth lag behind the development of skeletal structures, then the limit of the supporting structures is exceeded and joint laxity occurs.

II. Severity depends on the degree of overloading of the joint during its development.

III. Changes in cartilage, supporting soft tissue, and muscles cause alterations in bony architecture.

IV. Coxofemoral joint laxity leads to subluxation of the femoral head, which causes cartilage damage, release of degradative enzymes, and loss of cartilage matrix.

V. The end result is osteoarthritis (DJD).

Clinical Signs

I. Pelvic limb lameness is worse after exercise and varies from mild and intermittent to non–weight-bearing.

II. Decreased flexion and extension of the hip during a walk or trot and a "bunny-hopping gait" while running are common findings.

III. Pelvic limb muscle atrophy, joint laxity, and pain during range-of-motion examination of the coxofemoral joint are typical findings.
   A. Ortolani sign is a palpable click elicited as the subluxated hip reduces with abduction.
   B. Ortolani sign is often positive in young dogs with mild to moderate degenerative changes and is consistent with hip laxity and dysplasia.
   C. As the degeneration progresses, the acetabulum fills with new bone and the Ortolani sign becomes negative.
   D. A negative Ortolani sign can be a normal finding or indicative of advanced DJD.

Diagnosis

I. Radiographic changes range from mild subluxation of the femoral head to severe DJD.
   A. In young dogs (<10 months), subluxation is often the main abnormality.
   B. In dogs >10 months, evidence of DJD is typically present.
      1. Flattening of the femoral head
      2. Shallow acetabulum
      3. Osteophytosis of the femoral neck, femoral attachment of the joint capsule and acetabular margins
      4. Narrowing of the joint space
      5. Subchondral sclerosis of the femoral head and acetabulum

II. Stress radiographic techniques (e.g., PennHIP method) are useful to demonstrate laxity of the hip joint, which is a predictor of future DJD (Smith et al., 1990).

Differential Diagnosis

I. Cranial cruciate ligament rupture

II. Traumatic luxation

III. Degenerative myelopathy (older dogs)

IV. Femoral head, neck, or proximal femoral physeal fracture (younger dogs)

V. Infectious or inflammatory arthritis

Treatment

I. Medical management involves the following:
   A. Weight loss is important because it decreases loading of joints and muscles; the goal is a thin and athletic frame (body condition score of 2.5 to 3.5).
   B. Daily low-impact activity aids in weight loss and helps improve muscle mass, joint range of motion, and exercise tolerance.
   C. NSAIDs are administered as needed (see Table 80-1) (Bergh and Budsberg, 2005).
      1. Never administer NSAIDs with other NSAIDs (including aspirin) or corticosteroids.
      2. When switching from one NSAID (particularly aspirin) to another, wait a few days (according to the manufacturer's recommendations).
   D. Chondroprotective agents may mitigate inflammation and enhance reparative processes.
      1. Glucosamine hydrochloride 22 mg/kg PO SID, *with*
      2. Chondroitin sulfate 8.8 mg/kg PO SID
   E. Diets containing high levels of omega-3 fatty acids and eicosapentaenoic acid may help improve clinical signs associated with DJD.

II. Once the animal is refractory to medical management or is disabled by the condition, surgical therapy is indicated.
   A. An exception is the triple pelvic osteotomy (TPO), in which delay may preclude the animal from being a good candidate.
   B. Surgical management is divided into treatments that aim to diminish the progression of DJD and salvage procedures that remove the original hip joint.

III. Juvenile pubis symphysiodesis is electrocautery of the pubic symphysis to induce its premature closure (Dueland et al., 2001).
   A. The resulting asymmetrical closure of the pelvic symphysis (pubic is closed, ischial is not) and continued growth of the sacrum and ilium result in acetabular ventroversion, which enhances femoral head capture.
   B. Procedure is experimentally effective but must be performed at an early age (12 to 16 weeks).
   C. It is difficult to identify which dogs will benefit at such an early age.

IV. TPO increases the acetabular coverage of the femoral head.
   A. The ideal candidate is young (5 to 12 months), has minimal or no radiographic evidence of DJD, and has adequate femoral head capture.
   B. Femoral head capture is subjective assessment of dorsal acetabular rim wear, and is assessed by performing the Ortolani maneuver and determining the angle at which the femoral head reduces into the acetabulum (angle of reduction), as well as the angle at which it subluxates (angle of subluxation).
   C. The quality of femoral head capture may also help determine if the dorsal acetabular rim is excessively worn or able to maintain the hip in reduction postoperatively.
   D. Arthroscopic examination of the hip joint allows a more thorough evaluation of the joint before surgery.
   E. TPO entails osteotomy of the pelvis at the pubis, ischium, and ilium; external rotation (ventroversion) of the free acetabular segment; and stabilization of the ilial osteotomy with plate fixation, to provide more dorsal coverage to the femoral head and preventing continued subluxation.

V. Femoral head ostectomy (FHO) is a salvage procedure in which the femoral head and neck are removed and a scar tissue "joint" subsequently forms.
   A. Animals treated with this procedure have a limited range of motion, a mildly abnormal gait, and persistent muscle atrophy.
   B. Although range of motion is diminished, most animals <20 kg function well; results in animals ≥20 kg are less reliable.

VI. Total hip arthroplasty (THA) provides the animal with a prosthetic femoral component and acetabular cup.
   A. Currently, component systems are either cemented in place or are cementless in design.
   B. THA results in normal or near normal gait, muscle mass, and range of motion.
   C. THA is a sophisticated procedure that demands strict asepsis and precise surgical technique if a successful result is to be obtained.

Monitoring of Animal

I. Animals treated medically are monitored to assess efficacy of therapy and limb function.
   A. Initially, reevaluation is performed at monthly intervals.
   B. If the response to treatment is favorable, then the dog is reevaluated every 3 to 4 months and at times of disease flare-up.

II. Surgically treated animals undergo exercise restriction and are monitored for common complications as follows:
   A. Juvenile pubis symphysiodesis: progression of DJD
   B. TPO
      1. Exercise is restricted for 4 to 6 weeks.
      2. Radiography is conducted at 4 to 6 weeks postoperatively to assess bony healing.
      3. If clinically indicated, surgery may be performed on the contralateral side once bony healing is adequate on the initial side (typically 4 to 6 weeks).
      4. Long-term function is good despite progression of DJD (Rasmussen et al., 1998).
   C. FHO
      1. Initiate low-impact activities (e.g., leash walking, swimming) after 2 weeks.
      2. Passive range-of-motion exercises may be needed if weight bearing does not begin within 10 to 14 days.
      3. Gradual return to normal activities is encouraged during weeks 2 to 8.
   D. THA
      1. Walk the animal outside on a leash (for eliminations only) for the first 4 weeks.
      2. Duration of leash walks is increased progressively during weeks 5 to 8.
      3. Short episodes of off-leash activity are allowed during weeks 9 to 12.

4. Radiographs are taken at week 12 to assess implants, then yearly.
5. Most (92% to 95%) animals have good or excellent function postoperatively (Olmstead, 1987).

Osteochondrosis and Osteochondritis Desiccans

Definition and Causes

I. Osteochondrosis (OC) is failure of endochondral ossification and refers to the disease in general.
II. OCD is a combination of dissecting lesions of articular cartilage, communication of synovial fluid into subchondral bone, and synovitis (Schultz and Krotscheck, 2003).
III. Factors that have been implicated include nutrition (e.g., excessive nutrition, dietary calcium, protein), genetics, exercise, environmental factors, and trauma (e.g., excessive mechanical loading).

Pathophysiology

I. A defect in endochondral ossification results in a focal area of abnormal subchondral bone formation.
II. The overlying cartilage fails to ossify, resulting in focal retention of cartilage.
III. The thickened region of cartilage becomes necrotic and weak, and breaks down under normal loading conditions or secondary to trauma.
   A. Early in the disease process, OC affects only the epiphyseal cartilage and the animal is asymptomatic.
   B. Once a fissure occurs in the thickened cartilage, it extends through the necrotic cartilage to the subchondral bone, allowing access of the synovial fluid to the subchondral bone.
   C. This latter stage seems to correspond to the onset of clinical signs, at which time the disease is termed *OCD.*
IV. OCD results in two distinct joint abnormalities.
   A. Joint incongruity secondary to malformation of cartilage and subchondral bone
   B. Joint mouse formation
V. Cartilage flaps that remain attached may ossify, with the resulting bone remaining viable as long as the flap is attached.
VI. Detached cartilage flaps may survive in the joint fluid, grow in size, and mineralize or ossify if they attach to the joint capsule.

Clinical Signs

I. Common sites are the caudomedial humeral head (shoulder), distal humerus (elbow), trochlear ridge of the talus (hock), and femoral condyle (stifle).
II. Shoulder lesions often occur in large breed dogs, 4 to 8 months of age.
   A. Males are affected more commonly than females.
   B. Many dogs are clinically affected in only one limb, but lesions are bilateral in 43% to 65% of cases (Bloomberg and Lewis, 1998).
   C. Thoracic limb lameness may be noted before or after exercise.
   D. Pain is elicited with shoulder extension; crepitus, deltoid and spinatus muscle atrophy may be found.
III. Hock lesions typically occur in young (5 to 8 months) large-breed dogs, especially the rottweiler, Labrador retriever, and bullmastiff.
   A. OCD of the medial trochlear ridge is most common, whereas the lateral trochlear ridge OCD often occurs in rottweilers.
   B. Pelvic limb lameness is characterized by a shortened stride and hyperextension of the tarsocrural joint.
   C. Thickening of the tarsus occurs, especially if the medial trochlear ridge of the talus is involved.
IV. Stifle lesions occur in large-breed dogs that are 5 to 7 months of age.
   A. Lameness varies from mild to severe.
   B. Joint effusion, muscle atrophy, and crepitus may be evident.

Diagnosis

I. Normal cartilage is not visible radiographically unless significant dystrophic calcification has occurred.
II. Radiographically, an OCD lesion is a flattened or saucer-like "divot" in the subchondral bone.
III. Shoulder radiographs reveal a flattening of the humeral head in a properly positioned lateral view.
IV. An arthrogram of the shoulder may be needed if the lesion is not evident or if cartilage has migrated into the biceps tendon sheath.
V. Radiographic identification of hock lesions may be difficult because of the location of the lesion on the trochlear ridge.
   A. An extended dorsoplantar projection may reveal the defect in the trochlear ridge.
   B. A dorsolateral-plantar-medial 45-degree oblique projection in full extension outlines the medial trochlear ridge of the talus.
   C. A skyline view of the talus may identify a lesion on the center of the trochlear ridge of the talus.
VI. Slight flattening and sclerosis of the subchondral bone of the femoral condyle is evident in a caudocranial view of the stifle.
VII. Do not confuse the extensor fossa of the femur for an OCD lesion.

Differential Diagnosis

I. Panosteitis
II. Traumatic joint injury
III. Septic or inflammatory arthritis
IV. Other causes of thoracic limb lameness: elbow dysplasia
V. Other causes of pelvic limb lameness: cranial cruciate ligament rupture, hip dysplasia

Treatment

I. As the relative size of the osteochondral defect increases, the resulting joint incongruity also increases; therefore a small defect in a large joint (shoulder) has less of an affect than in a small (hock) or complex (elbow or stifle) joint.

A. Surgical treatment can result in normal or near-normal function in large joints with relatively small lesions (shoulder).
B. Function is improved, but clinical signs are not always alleviated in complex or small joints (elbow, hock, stifle).

II. Goals of surgery are to surgically debride the osteochondral defect, with minimal damage to the joint.
A. Arthrotomy or arthroscopy can be used.
B. Debride the lesion to the level of subchondral bleeding bone, using a curette, hand bur, or motorized shaver.
C. Debride the edge of the lesion peripherally until normal cartilage is reached, ensuring the edges are perpendicular to the subchondral bed.
D. Microfracture can be used to create vascular access channels from the lesion to the underlying subchondral bone.

III. Long-term medical management with weight control, exercise moderation, NSAIDs, chondroprotective agents, and nutritional changes are also helpful (see Osteoarthritis).

IV. Rest (2 to 3 weeks) and NSAIDs are used for recurrent episodes of lameness, followed by return to moderate regular exercise.

Monitoring of Animal

I. In the case of hock or stifle OCD, a soft, padded bandage is applied for 3 to 5 days from the digits to the middiaphysis proximal to the incision to minimize postoperative swelling.

II. The dog is confined to leash walks for 6 weeks, then gradually returned to normal activity over the next 6 weeks.

III. Degree of DJD in the joint before surgery has an inverse effect on long-term function.

IV. Dogs with OCD of the hock may fare well without surgery, because of the relatively large size of typical hock lesions and the tendency for significant preoperative DJD.

DEGENERATIVE DISORDERS

Osteoarthritis

Definition and Causes

I. Osteoarthritis (i.e., DJD) is a syndrome of pathologic changes in diarthrodial or synovial joints accompanied by signs of pain and disability.

II. It develops secondary to trauma, or from application of normal forces on abnormal joints, such as with hip dysplasia or cranial cruciate ligament disease.

III. Other less common causes include sepsis, prolonged joint immobilization, inflammatory joint disease, or developmental diseases (e.g., OCD).

Pathophysiology

I. All joint tissues are involved, including articular cartilage, joint capsule, subchondral bone, ligaments, and muscle.

II. Initially, degradative enzymes are released from chondrocytes, synoviocytes, and inflammatory cells.

III. The earliest form of articular cartilage damage is fibrillation or roughening of the cartilage surface.

IV. Once the superficial layer of cartilage loses its integrity, the deeper layers are exposed to progressively higher loads, leading to fissure formation.

V. Cartilage destruction leads to altered biomechanical function of the joint, which perpetuates the degradative process and worsens functional impairment and discomfort (Todhunter and Johnston, 2003).

Clinical Signs

I. Signs include slowly progressive, episodic or persistent lameness, pain, and disability.
A. Stiffness is often noted after periods of rest.
B. Stiffness and lameness partially or fully resolve with activity in most cases.
C. Lameness is exacerbated by strenuous activity, particularly if the activity is followed by a period of rest.

II. Discomfort is noted during range-of-motion examination of the joints.

III. Periarticular fibrosis, bony crepitus, joint effusion, and muscle atrophy are common findings.

Diagnosis

I. History and clinical findings allow a presumptive diagnosis.

II. Radiography reveals joint effusion, periarticular osteophytosis, muscle atrophy, and subchondral bone sclerosis.
A. Joint space narrowing may occur from cartilage thinning; however, most radiographs are not obtained under weight-bearing conditions, so this finding must be interpreted with caution.
B. Joint subluxation and luxation may also be seen.

III. Synovial fluid analysis is consistent with DJD (Table 80-2).

Differential Diagnosis

I. Trauma

II. Developmental joint disease

III. Inflammatory joint disease

TABLE 80-2

Interpretation of Synovial Fluid Analysis

CONDITION	TOTAL CELL COUNT ($\times 10^3$/L)	MONONUCLEAR CELLS (%)	NEUTROPHILS (%)
Normal	0.0-3.0	90-100	0-10
Nonsuppurative Inflammation			
Degenerative osteoarthritis	0.0-3.5	90-100	0-10
Suppurative Inflammation			
Nonerosive arthritis	4.4-370	5-85	15-95
Erosive arthritis	3.0-38	20-80	20-80
Bacterial arthritis	110-267	1-10	90-99

Treatment

I. Prevention of osteoarthritis is preferable, but the condition is usually well established before diagnosis.
II. Medical management of osteoarthritis involves five basic treatments.
  A. Weight loss decreases loading of joints and muscles, and the goal is a thin and athletic frame.
  B. Daily low-impact activity aids in weight loss and helps improve muscle mass, joint range of motion, and exercise tolerance.
  C. NSAIDs are administered as needed (see Table 80-1) (Bergh and Budsberg, 2005).
  D. Chondroprotective agents may mitigate inflammation and enhance reparative processes.
    1. Glucosamine hydrochloride 22 mg/kg PO SID, *with*
    2. Chondroitin sulfate 8.8 mg/kg PO SID
  E. Diets containing high levels of omega-3 fatty acids and eicosapentaenoic acid may help improve the clinical signs.
III. Surgical management is indicated in selected cases.
  A. If joint instability exists (e.g., cranial cruciate ligament rupture), then surgical stabilization mitigates clinical signs.
  B. Joint replacement is very effective in alleviating clinical signs but is routinely performed only in the hip (THA).
  C. Total elbow and knee arthroplasties may be performed.
  D. Excision arthroplasty may be performed in selected joints.
    1. FHO
    2. Resection of the glenoid excision arthroplasty of the humeral head: data on outcome lacking
  E. Arthrodesis may be performed in selected joints to salvage limb function.
    1. Partial or total carpal and or tarsal arthrodeses are well tolerated and result in acceptable limb function.
    2. Shoulder, stifle, and elbow arthrodesis result in significant mechanical lameness but may diminish clinical discomfort.

Monitoring of Animal

I. NSAIDs are administered only as needed at the lowest effective dose.
II. Reexamine the animal every 1 to 4 months to assess the efficacy of therapy and to monitor for any complications of treatment (Bergh and Budsberg, 2005).
  A. Perform a complete blood count and biochemical profile before initiating therapy.
  B. Laboratory tests are repeated every few months while the animal is on NSAIDs to monitor for hematological, hepatic, and renal abnormalities.
III. Perform follow-up radiographs 8 to 12 weeks after surgery in animals treated with surgical procedures.

Cranial Cruciate Ligament Rupture

Definition and Causes

I. Disruption of the cranial cruciate ligament (CCL) results in stifle instability and secondary DJD.
II. An acute mechanical overload from trauma can cause disruption of a normal CCL.
III. More commonly, a progressive deterioration of the ligament coupled with a normally high level of mechanical stress results in rupture.

Pathophysiology

I. The CCL ligament limits cranial tibial subluxation, internal rotation of the tibia, and stifle hyperextension.
II. Rupture results in stifle joint instability, which causes DJD and meniscal damage.
III. Meniscal injury occurs in approximately 20% to 80% of cases (Vasseur, 2003).
  A. The lateral meniscus is seldom damaged because of its mobility.
  B. The medial meniscus is commonly damaged because the structure is relatively immobile.
    1. Medial meniscal injury results from either crushing or tearing.
    2. Meniscal injury leads to further instability and synovitis.

Clinical Signs

I. Lameness can vary from mild and intermittent (partial tear) to non–weight-bearing (complete rupture and/or meniscal tear).
II. Non–weight-bearing lameness is often noted at the time of ligament rupture, followed by partial weight bearing within 48 to 72 hours.
III. Pain is elicited with palpation, manipulation, and full extension of the stifle joint.
IV. Stifle effusion, periarticular fibrosis (medial buttress formation), positive cranial drawer test, positive tibial compression test, bony crepitus, and muscle atrophy may be noted.
V. A meniscal click may be evident with flexion of the stifle, if the meniscus is torn.
VI. Bilateral CCL rupture may result in difficulty, inability, or unwillingness to rise and can be confused with neurological disease or myopathy.

Diagnosis

I. Stifle joint palpation is the most reliable noninvasive test.
II. Positive cranial drawer and/or tibial compression tests are diagnostic.
  A. To perform the cranial drawer test, position the animal in lateral recumbency.
    1. Place the thumb of the proximal hand behind the lateral fabella and the index finger on the patella.
    2. Place the thumb of the distal hand behind the fibular head, and place the index finger on the tibial tuberosity.
    3. Check the tibia for cranial movement with respect to the femur throughout a range of motion (from 30 degrees short of full extension to 90 degrees of flexion).
    4. In the adult, no cranial drawer is present.

5. In the juvenile, 3 to 4 mm of cranial drawer may be present; it ends with an abrupt stop and is symmetrical in both stifles.
6. Cranial drawer in flexion is consistent with a partial CCL rupture.

B. To perform the tibial compression test, position the animal in lateral recumbency.
1. Grasps the metatarsus with the distal hand.
2. Extend the index finger of the proximal hand down the straight patellar tendon, with the fingertip on the tibial tuberosity.
3. Monitor for cranial tibial translation as the hock is flexed.

III. Radiographic findings are consistent with DJD.
A. Effacement of the infrapatellar fat pad, with soft tissue opacity in the lateral view, is consistent with stifle joint effusion.
B. Periarticular or peritrochlear osteophytosis may be detected.
C. Osteophytosis of the base or apex of the patella, distal aspect of the fabella, and cranial intercondyloid area of the tibia may be noted.
D. Cranial tibial subluxation may be seen.
E. Medial buttress formation may be seen on the caudocranial view.

IV. Stifle arthrotomy or arthroscopy may be used to confirm the diagnosis.

Differential Diagnosis

I. Patellar luxation
II. Stifle OCD
III. Avulsion of the long digital extensor tendon
IV. Caudal cruciate ligament rupture
V. Hip dysplasia

Treatment

I. Very small dogs (<5 kg) and most cats may only require medical management (see Osteoarthritis).
II. Larger dogs, obese cats, and active small dogs benefit from stifle stabilization.
III. Extracapsular techniques use elements outside the joint to provide stability.
A. Lateral suture (lateral retinacular imbrication)
B. Fibular head transposition
IV. Intracapsular techniques use an intraarticular graft (patellar tendon or biceps fascia) to stabilize the joint.
A. Under-and-over fascial technique
B. Over-the-top procedure
V. Osteotomy procedures alter joint biomechanics to lessen or eliminate the cranial tibial thrust force, thereby eliminating the need for the cranial cruciate ligament.
A. Tibial plateau–leveling osteotomy
B. Tibial-closing wedge osteotomy
C. Tibial tuberosity advancement
VI. In young dogs, avulsion of the femoral origin or (more commonly) the tibial insertion of the CCL may occur; if the avulsed fragment is large enough, then it may be reduced and stabilized with internal fixation.
VII. If medial meniscal injury is present, the damaged portion (partial meniscectomy) or the entire meniscus (medial meniscectomy) is removed.

Monitoring of Animal

I. Institute medical management and monitoring for osteoarthritis in all animals.
II. A bandage may be placed after an arthrotomy to minimize postoperative swelling and edema.
III. Evaluate limb function and stifle stability 6 to 8 weeks postoperatively.
IV. Radiographically evaluate bony healing 6 to 8 weeks after osteotomy procedures.
V. Begin low-impact activity (e.g., leash walking) at weeks 2 to 4 and continue until lameness resolves (typically by weeks 6 to 8).
VI. The risk for contralateral CCL rupture in cases without a known traumatic event is high (Vasseur, 2003).
VII. Most procedures result in 90% of affected animals returning to good (occasional lameness) or excellent (no discernable lameness) function (Vasseur, 2003).
VIII. Deterioration of limb function after stifle stabilization may be the result of latent meniscal injury, which requires arthroscopic or open meniscectomy (partial or complete).

INFECTIOUS ARTHRITIS

Bacterial Arthritis

Definition and Causes

I. Bacterial joint infection results in arthritis.
II. Hematogenous spread of bacteria may occur from the respiratory, urinary, and digestive tracts; umbilicus; and endocardium, primarily in young or immunocompromised animals.
III. Exogenous sources are the most common and include trauma, surgical procedures, and intraarticular injections.
IV. Common organisms include staphylococci, streptococci, and coliforms.

Pathophysiology

I. Bacterial contamination of the synovium results in edema, hyperemia, and infiltration of neutrophils.
II. Inflammation of the synovium leads to capillary rupture and local areas of necrosis.
III. Lysosomal enzyme and enzyme by-products released by the synovial cells and neutrophils cause degradation of the cartilage matrix and collagen.

Clinical Signs

I. Joint effusion, periarticular swelling, and discomfort are found in one or more joints.
A. Hematogenous spread typically involves multiple joints.
B. Exogenous infections usually involve one joint.
II. Severe, weight-bearing or non–weight-bearing lameness occurs.

III. Pyrexia may be present.
   A. Systemic signs are often present in hematogenous cases.
   B. Systemic signs are uncommon in exogenous cases.

Diagnosis

I. Tentative diagnosis is based on clinical findings, particularly a history of recent joint surgery.
II. Definitive diagnosis is based on results of joint fluid analysis (see Table 80-2).
   A. Reduced viscosity
   B. Increased white blood cell (WBC) count
   C. Predominance of neutrophils
III. Joint fluid culture is commonly negative; however, inoculation of blood culture medium facilitates growth of the causative organism (Montgomery et al., 1989).

Differential Diagnosis

I. Trauma
   A. Ligamentous injury
   B. Intraarticular fracture
   C. Periarticular or physeal fracture
   D. Periarticular or intraarticular neoplasia
II. Other infectious arthritides
   A. Calicivirus infection of cats (Dawson et al., 1994)
   B. Mycoplasmosis
   C. Coronavirus
   D. Fungal infections: rare, usually associated with adjacent fungal osteomyelitis or immunocompromise

Treatment

I. Systemic antimicrobial therapy is initiated immediately after samples are obtained for fluid analysis and culture.
II. A broad-spectrum, bactericidal agent is started pending the results of culture, followed by long-term (6 to 8 weeks) oral administration.
   A. Cefazolin 20 to 25 mg/kg IV, IM TID to QID
   B. Cephalexin 11 to 33 mg/kg PO TID
   C. Enrofloxacin 5 to 20 mg/kg PO SID or 2.5 to 10 mg/kg PO BID (mature dogs)
   D. Tetracycline 15 to 20 mg/kg PO TID for 4 weeks or doxycycline 5 to 10 mg/kg PO BID for 4 weeks for *Borrelia* spp., rickettsiae, *Mycoplasma* spp., bacterial L-forms
III. Joint lavage is essential to decompress the joint and remove cellular and enzymatic agents that exacerbate cartilage damage.
   A. Fine-needle aspiration or ingress-egress drainage is rarely sufficient.
   B. Arthrotomy or arthroscopy, surgical debridement, and copious (5 L) lavage is recommended in the following cases (Bubenik and Smith, 2003):
      1. Postoperative infection
      2. Septic joints left untreated for >72 hours
      3. Septic joints that have not responded to antibiotics alone for 72 hours
      4. Penetrating wounds
IV. Severe infections may necessitate management of the joint as an open wound, with daily sterile bandage changes until the wound is fully granulated.

Monitoring of Animal

I. Parenteral (ideally) or oral antibiotic therapy, based on the results of culture and sensitivity, is continued for a minimum of 4 weeks or at least 2 weeks beyond resolution of clinical signs.
II. Residual lameness may be treated as outlined previously for Osteoarthritis.

Borreliosis (Lyme Disease)

See Chapter 113.

Ehrlichiosis

See Chapter 115.

Rocky Mountain Spotted Fever

See Chapter 115.

IMMUNE-MEDIATED ARTHRITIS

Nonerosive Arthritides

Definition and Causes

I. These inflammatory diseases are characterized by periarticular inflammation, without radiographic or histological evidence of joint destruction in their early stages.
II. The most common nonerosive immune-mediated arthropathies are idiopathic polyarthritis, systemic lupus erythematosus (SLE), polyarthritis of chronic disease, and drug-induced polyarthritis (Davidson, 2003).

Pathophysiology

I. Immune complex deposition in the synovial membrane results in synovitis.
II. Mononuclear cell infiltration occurs in the synovium.
III. Neutrophils migrate from synovial capillaries into the synovium.
IV. The inflammatory process results in weakening of the intraarticular and periarticular ligamentous structures and leads to joint instability.
V. In these cases, osteoarthritis may arise from chronic joint instability rather than the initial inflammatory process.

Clinical Signs

I. Lameness affects one or more limbs.
   A. The distal limb joints (carpus, tarsus, interphalangeal joints) are most often affected.
   B. Monoarticular disease is most commonly seen in the elbow.
II. Pain, joint effusion, periarticular fibrosis, periarticular soft tissue swelling, and palpable joint hyperthermia may occur.
III. Clinical signs may be intermittent or persistent and shift from limb to limb.
IV. SLE is often accompanied by abnormalities in other organs (see Chapters 76, 91, and 104).

Diagnosis

I. Radiographic findings are limited to joint effusion and periarticular fibrosis in the early stages.
II. Radiographic findings in late stages are similar to those of erosive polyarthritis (periarticular osteophytosis, joint space collapse).
III. Synovial fluid analysis is consistent with suppurative inflammation (see Table 80-2).
IV. Lupus erythematosus cell assays may be positive.
V. Serological testing for antinuclear antibody (ANA) may be positive.
VI. Serological testing for rheumatoid factor and infectious agents is negative.

Differential Diagnosis

I. Erosive arthritides
II. Infectious arthritides
III. Osteoarthritis

Treatment and Monitoring

I. Prednisone (1 to 2 mg/kg PO BID for 10 to 14 days) is used as the initial treatment.
II. In mild cases chronic administration of NSAIDs may control clinical signs (see Table 80-2).
III. If synovial fluid WBC counts drop to <4000 cells/μL, then the prednisone dose is slowly tapered over several weeks to 1 mg/kg PO QOD.
IV. If remission continues on reduced doses for 1 to 3 months, then elimination of prednisone can be considered.
V. In refractory canine cases, a cytotoxic drug may be added to the prednisone.
   A. Cyclophosphamide 1.5 to 2.5 mg/kg PO SID for 4 consecutive days each week (discontinued 1 month after remission, after 4 months of therapy, or if hemorrhagic cystitis develops)
   B. Azathioprine 2 mg/kg PO SID for 14 to 21 days, then 1 to 2 mg/kg PO QOD for 1 month beyond remission
   C. Methotrexate 2.5 mg/m^2 PO SID
VI. Arthrodesis may be considered for irreversibly damaged joints.

Erosive Arthritides

Definition and Causes

I. These arthritides are characterized by periarticular inflammation, with destruction of articular cartilage and bone.
II. They include rheumatoid arthritis (RA), erosive polyarthritis of greyhounds (EPG), and feline chronic progressive polyarthritis.

Pathophysiology

I. Specific causes have not been identified.
II. In RA, host immunoglobulin (Ig) G becomes antigenic and IgM (rheumatoid factor) is formed in response.
III. Immune complex deposition in the synovium causes synovitis.
IV. Destruction of the articular cartilage and bone results from the ensuing inflammatory process.

Clinical Signs

I. RA usually affects adult, small-breed dogs.
II. EPG affects greyhounds 3 to 30 months of age.
III. Feline chronic progressive polyarthritis affects cats 1 to 5 years of age.
IV. Lameness may occur in one or more limbs.
V. The most commonly affected joints are the distal limb joints, such as the carpus, tarsus, and interphalangeal joints.
VI. Pain, joint effusion, periarticular fibrosis, periarticular soft tissue swelling, and palpable joint hyperthermia may be noted.
VII. Clinical signs may be intermittent or persistent and shift from limb to limb.

Diagnosis

I. Radiographic findings include joint space collapse, periarticular fibrosis, periarticular soft tissue swelling, subchondral bone cysts, and periarticular osteophytosis.
II. In chronic cases, joint subluxation or luxation may be present.
III. Synovial fluid analysis is consistent with suppurative inflammation (see Table 80-2).
IV. Rheumatoid factor assays are positive in 25% to 75% of cases (Davidson, 2003).
V. ANA analysis must be negative for a diagnosis of RA.
VI. Animals with a positive ANA analysis probably have SLE.

Differential Diagnosis

I. Nonerosive arthritides
II. Infectious arthritides
III. Osteoarthritis

Treatment

I. Prednisone (1 to 2 mg/kg PO BID for 10 to 14 days) is used as the initial treatment.
II. In mild cases chronic administration of NSAIDs may control clinical signs (see Table 80-2).
III. In refractory cases a cytotoxic drug may be added to the prednisone (see Nonerosive Arthritis).
IV. In dogs, weekly injections of sodium aurothioglucose (1 mg/kg IM) have been successful for RA.
V. Arthrodesis may be considered in irreversibly damaged joints.

Monitoring of Animal

I. Complete resolution is rare, but remission may be achieved with 3 to 6 months of therapy.
II. CBC and platelet counts are monitored every 1 to 3 weeks in animals on cytotoxic therapy, depending on the dosage.
III. If synovial fluid WBC counts drop to <4000 cells/μL and clinical remission occurs, then dosages are slowly tapered over several weeks.
IV. Reexamination is recommended at 1- to 6-month intervals to assess for recurrence.
V. Treatment of EPG has been unrewarding, and the prognosis is poor.

VI. Cats are monitored at yearly intervals for infection with feline leukemia virus if treated with cytotoxic agents.

NEOPLASIA

Synovial Sarcoma

Definition and Causes

I. Synovial sarcoma is a malignant neoplasm that arises from primitive, undifferentiated, mesenchymal tissue adjacent to synovial membranes (Fox et al., 2002).
II. The etiology is unknown.
III. Presence and growth of the intraarticular tumor results in an inflammatory arthropathy and joint destruction.

Clinical Signs

I. Progressive lameness in a single limb is a classic sign.
II. Large-breed dogs are most commonly affected.
III. Joint effusion, periarticular soft tissue swelling, edema of the limb, and discomfort with manipulation of the limb are common.

Diagnosis

I. Radiographic findings may include intraarticular soft tissue opacity, subchondral and epiphyseal bone lysis, periarticular osteophytosis, soft tissue calcification, and an irregular periosteal reaction.
II. Definitive diagnosis requires arthroscopic or open biopsy, as well as histological examination.
III. Reliability of differentiating synovial sarcoma from other soft tissue sarcomas or round-cell neoplasia via histopathologic examination or immunohistochemistry is questionable (Fox et al., 2002).

Treatment

I. Limb amputation: local excision not possible
II. Chemotherapy: efficacy not well established

Monitoring of Animal

I. Monitor for local recurrence and distant metastasis.
II. The metastatic rate for synovial sarcoma is 40% to 50% (Vail et al., 1994).

TRAUMATIC DISORDERS

Caudal Cruciate Ligament Rupture

Definition and Causes

I. Trauma is the typical cause of this rare condition.
II. Force applied to the tibia in the caudal direction may cause isolated rupture because it prevents caudal subluxation of the tibia.
III. More commonly, this structure is damaged in association with collateral ligament rupture or stifle luxation.

Clinical Signs

I. Acute-onset lameness is seen after known or possible trauma (Johnson and Olmstead, 1987).
II. Stifle pain and joint effusion may be seen.
III. Caudal drawer sign, tibial sag, and an avulsion fragment (radiographically) may be evident in some cases.
   A. Caudal drawer sign must be carefully differentiated from a cranial drawer sign.
   B. Caudal drawer sign is present if the tibia moves from a neutral position to a position of caudal subluxation.

Diagnosis

I. Caudal drawer sign
II. Confirmed surgically by arthrotomy or arthroscopy

Differential Diagnosis

I. Cranial cruciate ligament rupture
II. Stifle collateral ligament rupture
III. Stifle luxation
IV. Meniscal injury
V. Patellar luxation

Treatment

I. Clinical signs may resolve with conservative treatment with cage rest and NSAIDs for 8 weeks.
II. In large dogs or cases of persistent lameness, extracapsular stabilization is recommended.
   A. Extracapsular suture stabilization
   B. Popliteal tenodesis

Monitoring of Animal

I. After surgery, restrict exercise for 6 to 8 weeks, then gradually return the animal to normal activity during weeks 8 to 16.
II. Prognosis for isolated rupture of the caudal cruciate ligament is good.

Stifle Luxation (Deranged Stifle)

Definition and Causes

I. Stifle subluxation is a complete or partial rupture of the stifle collateral ligaments or a combination of rupture of the stifle collateral ligaments and the cruciate ligaments (stifle luxation).
II. Severe trauma to the stifle joint is the cause.

Pathophysiology

I. Ligament injuries (sprains) can be mild (first degree), more severe with stretching and rupture of ligament fibers (second degree), or result in tearing or avulsion of the ligament (third degree).
II. Only third-degree and some second-degree injuries require surgical therapy.
III. Isolated rupture of the collateral ligaments is rare in small animals (Vasseur, 2003).
IV. Most injuries involve rupture of the medial and/or lateral collateral ligaments and the cranial and/or caudal cruciate ligaments.
   A. Most commonly affected ligaments are the medial collateral cranial cruciate ligament and the caudal cruciate ligament.

B. Lateral collateral ligament becomes lax with stifle joint flexion, which may spare it from damage.

Clinical Signs

I. Stifle effusion, pain, and angular deviation may be evident.
II. Damage to the medial collateral ligament results in a valgus instability.
III. Damage to the lateral collateral ligament results in a varus instability.
IV. Damage to the cruciate ligaments results in cranial and/or caudal drawer.

Diagnosis

I. Meticulous physical examination under anesthesia allows determination of which ligaments are involved.
II. Results of palpation may be confusing, and joint exploration is frequently required to confirm individual ligament involvement.

Differential Diagnosis

I. Cranial cruciate ligament rupture
II. Bacterial arthritis
III. Intraarticular or periarticular fracture

Treatment

I. Reconstruction of each of the involved ligaments
   A. The collateral ligaments can be primarily repaired, or a prosthetic ligament can be created from bone anchors or screws and heavy-gauge monofilament suture.
   B. The stifle can be stabilized as for cranial and/or caudal cruciate ligament rupture.
II. Transarticular stabilization
   A. Transarticular pin placement for small dogs or cats
   B. Rigid transarticular fixator application
   C. Hinged transarticular fixator placement
   D. External coaptation
   E. Arthrodesis

Monitoring of Animal

I. Institute medical management and monitoring for DJD.
II. A bandage may be placed to minimize postoperative swelling and edema after arthrotomy.
III. Evaluate limb function and stifle stability 6 to 8 weeks postoperatively.
IV. Low-impact activity is recommended, beginning at week 2 to 4 and continuing until lameness has resolved—typically by week 6 to 8.
   A. In animals treated with external skeletal fixation, remove the fixation device 4 to 6 weeks postoperatively.
   B. Institute low-impact activity after fixator removal.

Traumatic Joint Luxation

Definition and Causes

I. Traumatic joint luxation is the complete separation of two articulating joint surfaces.
II. Severe trauma is the typical cause of joint luxation.
III. Traumatic episode results in the disruption of the normal stabilizers of the joint, including the joint capsule, collateral ligaments, and other associated ligaments.
IV. Commonly affected joints include the shoulder, elbow, carpus, hip, stifle, tarsus, metacarpophalangeal and tarsophalangeal joints, and interphalangeal joints.

Clinical Signs and Diagnosis

I. Lameness of the affected limb can vary from mild to non–weight-bearing.
II. Clinical signs and history of trauma allow a presumptive diagnosis.
III. Physical examination findings are confirmatory in many cases.
   A. Discomfort with manipulation
   B. Excessive mobility
   C. Soft tissue swelling
   D. Abnormal limb length, with affected limb shorter
IV. Radiography confirms the luxation.

Differential Diagnosis

I. Intraarticular or periarticular fracture
II. Bacterial arthritis
III. Neoplasia

Treatment

I. Closed reduction and coaptation
II. Open reduction
III. Carpus
   A. Isolated collateral ligament injuries may be treated with primary repair or placement of a prosthetic ligament.
   B. Most injuries require partial carpal arthrodesis or pancarpal arthrodesis.
IV. Hip
   A. Closed reduction
      1. It is indicated for acute luxation (<3 to 5 days) in animals with normal hip conformation and no intraarticular fractures.
      2. Maintain reduction with an Ehmer sling for 3 to 4 weeks.
   B. Open reduction
      1. It is indicated when the femoral head does not seat well in acetabulum, the hip has reluxated after closed reduction, the hip has been chronically luxated, and intraarticular fractures or hip dysplasia are present.
      2. Open reduction techniques include toggle pin, extracapsular prosthesis, and iliofemoral suture.
   C. Salvage techniques
      1. FHO
      2. THA
V. Tarsus
   A. Isolated collateral ligament injuries may be treated with primary repair or placement of a prosthetic ligament.
   B. Many injuries require partial or total tarsal arthrodesis.
VI. Metacarpal, metatorsal, tarsophalangeal, and interphalangeal joints
   A. Closed reduction and external coaptation
   B. Primary ligamentous repair

C. Arthrodesis
D. Digit amputation

VII. Shoulder and elbow (see Congenital and Degenerative Disorders)

Monitoring of Animal

I. Instruct the owner to monitor bandages and slings SID-BID.
II. Restrict exercise for 4 to 8 weeks after bandage or sling removal to allow remodeling of ligamentous structures.
III. In surgical cases, obtain radiographs 6 to 8 weeks post-operatively to assess reduction, progression of osteoarthritis, and implant stability.

Physeal Injury

See Chapter 81.

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