Developmental Orthopedic Disease

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Developmental orthopedic diseases (DODs) are a common cause of lameness and pain in young dogs. A thorough knowledge of the patient’s signalment and history and a complete physical examination are necessary to localize the disease, establish a list of differential diagnoses, and develop a diagnostic plan. A thorough understanding of the disease etiology, pathophysiology, and progression is needed to recommend the appropriate medical and surgical treatments.

HYPERTROPHIC OSTEODYSTROPHY

Signalment
Hypertrophic osteodystrophy (HOD) is an idiopathic disease that affects rapidly growing large- and giant-breed dogs between 2 and 8 months of age [1–4]. Although there is no known sex predilection, male dogs are overrepresented in some reports [4–8]. Breeds found to have a higher incidence of HOD include German Shepherds, Irish Setters, Weimeraners, Great Danes, and Chesapeake Bay Retrievers [1–4].

Etiology and pathogenesis
The etiology of HOD is unknown. Reported potential causes include infection (canine distemper virus and Escherichia coli), hypovitaminosis C, oversupplementation with vitamins and minerals, vascular abnormalities, and genetics [4–8]. Lesions similar to those of HOD have been experimentally produced in dogs fed a free-choice diet high in protein, calcium, and calories [9].

Histologically, there is initially necrosis of the capillary loops that invade the cartilage model of the metaphyseal physis. Congestion and edema occur in the extraperiosteal soft tissues surrounding the metaphysis, followed by formation of a cuff of metaplastic cartilage and bone in the region [10]. Other histopathologic changes that may occur in dogs with HOD that die after a period of sustained high fever and systemic signs include interstitial pneumonia and mineralization of soft tissues (lung, spleen, kidney, aorta, and

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endocardium) [10]. In some dogs, viral inclusions resembling those of canine distemper virus have been observed in macrophages in the suppurative physeal lesions [10].

**Clinical signs**
Clinical signs of HOD include the acute onset of lethargy, reluctance to walk, mild to severe lameness, and generalized pain. The metaphyseal areas of affected long bones are swollen, firm, painful, and warm to the touch. These signs are most commonly observed in the distal radius, ulna, and tibia, although the ribs, mandible, scapula, and metacarpal bones may also be affected [3,6,11,12]. The lesions are usually bilateral and symmetric [13]. Systemic signs can include severe anorexia, weight loss, fever, and depression [9]. Death occurs in rare cases, usually caused by prolonged hyperthermia, euthanasia attributable to pain, long-term recumbency, anorexia, or general morbidity associated with the disease [14,15].

**Diagnosis**
The diagnosis of HOD is based on physical examination and radiographic findings. Lesions are most commonly seen in the distal radius, ulna, and tibia and have been reported in the femur. Dogs affected with HOD typically present with lameness, fever, and pain in the metaphyseal region of the long bones. Depression and anorexia are common. Radiographically, a radiolucent region (Fig. 1) is observed in the metaphysis parallel to the physis and is often referred to as a “double physeal line” [1–4]. Metaphyseal sclerosis, irregular widening of the physis, subperiosteal and extraperiosteal new bone formation may also be evident radiographically [9].

![Fig. 1. Craniocaudal radiographic view of the distal radius and ulna of a dog with HOD. Arrows identify radiolucent regions in the metaphyses parallel to the growth plates (“double physeal line”).](image-url)
Treatment of HOD consists of supportive care to maintain hydration, prevent decubital ulcers, and control pain. Nonsteroidal anti-inflammatory drugs (NSAIDs) are used to control pain in most cases, although corticosteroids have been used in unresponsive patients. A decrease in caloric intake may also be helpful in fast-growing dogs. Vitamin C and D supplementation has been described, but most reports indicate that such supplementation is not beneficial and may increase the risk of dystrophic calcification. Severe cases of HOD may require blood cultures to rule out septicemia. Broad-spectrum antibiotics are indicated with positive culture results [16].

Prognosis
HOD is usually a self-limiting disease with a good prognosis in uncomplicated cases. Permanent bony changes or growth plate abnormalities may occur in some cases, however. In severe cases, the prognosis is guarded, because systemic metabolic disease or secondary bacteremia causing tissue infections can lead to death or euthanasia. Specific vaccination protocols have been recommended (particularly for use in Weimaraners) using separate vaccines for canine distemper virus, parovirus, and adenovirus or using killed vaccines in place of modified-live vaccines [17,18]. These protocols are thought to avoid the possible cause of vaccine-induced HOD.

PANOSTEITIS
Signalment
Panosteitis is an acquired self-limiting inflammatory condition of undetermined cause that affects the diaphyseal and metaphyseal regions of the long bones of large-breed dogs from 5 to 18 months of age. It is rarely seen in older animals. German Shepherds, Doberman Pinschers, Golden Retrievers, Saint Bernards, Labrador Retrievers, and Basset Hounds are among the overrepresented breeds affected with panosteitis, although it may occur in other breeds [9]. The disease affects male dogs more frequently than female dogs [9,19,20].

Etiology and pathogenesis
The etiology of panosteitis is unknown. Histologically, there is increased osteoblastic and fibroblastic activity affecting the endosteum, periosteum, and marrow of affected sites, resulting in fibrosis and connective tissue replacement of the normal medullary cavity. Leakage of protein-rich fluid from congested medullary vessels and secondary formation of haphazard trabecular systems occur. Pain is likely caused by medullary hypertension and congestion or stimulation of pain receptors in the periosteum. There is no evidence of inflammatory cell exudates, necrosis, or neoplasia [21].

Clinical signs
Clinical signs of panosteitis include acute lameness, with or without a history of trauma. Dogs are typically presented for an acute shifting leg lameness, lethargy, and pain that is cyclic and recurrent. Anorexia and fever may also be
present. Pain is palpated along the diaphysis of long bones, especially the humerus, femur, and proximal radius and ulna [19,22]. Signs often resolve after several days or in 1 to 2 weeks; however, recurrence is common up to 18 months of age [9].

Diagnosis
The diagnosis of panosteitis is based on physical examination and radiographic findings. Early radiographic evidence of panosteitis includes an increased opacity of the medullary canal of long bones, usually near the nutrient foramen [13,23]. Blurring of the trabecular pattern and increased opacity of the endosteal surface of the medullary cavity are observed [19]. As the disease progresses, the medullary opacities become more delineated and begin to coalesce (Fig. 2). In 15% to 25% of cases, a smooth periosteal reaction occurs, giving the cortex a thicker appearance. Interestingly, radiographic signs may not always correlate with clinical lameness and may not be observed in early or mild cases of panosteitis. Radiographic lesions may occur in multiple bones simultaneously. Nuclear scintigraphy may aid in the diagnosis of cases of panosteitis in which radiographic signs are absent.

Fig. 2. Lateral radiographic view of the radius and ulna of a dog with panosteitis. Arrows identify areas of increased medullary opacity within the radius.
**Treatment**
Panosteitis is a self-limiting disease, and therapy consists of exercise restriction, weight reduction, and NSAIDs for pain management [13].

**Prognosis**
The prognosis for patients with panosteitis is excellent, and signs typically resolve by 18 to 20 months of age [24,25]. Secondary complications are rare [21,22]. In some dogs, intermittent lameness may occur for 6 to 18 months or longer and may shift to other limbs [21,22].

**OSTEOCHONDROSIS**

**Signalment**
Osteochondrosis (OC) is commonly observed in rapidly growing, large- and giant-breed, male dogs, typically between 5 and 10 months of age [26–31]. Puppies from predisposed breeds are typically large, have rapid growth rates, and are often on high planes of nutrition. The disease is most common in dogs that reach an adult weight of greater than 20 kg and tends to occur during periods of rapid growth. Puppies from small breeds of dogs are rarely affected with OC [27,32]. Male dogs are more commonly affected than female dogs [27,30,31]. The incidence of bilateral disease is reported to range from 20% to 85%, depending on the joint involved [26–33]. Right and left limbs are equally affected.

**Etiology and pathogenesis**
OC is a disturbance in the process of endochondral ossification in a focal area of a developing articular surface centered at the osteochondral junction. The cartilage in the affected site fails to undergo physiologic calcification and replacement by bone, leaving a thickened focal area of degenerative cartilage. This area of necrotic cartilage and fibrous tissue is vulnerable to shearing forces encountered during normal weight bearing and may become dislodged from the underlying bone, forming a flap (Fig. 3). This lesion is referred to as osteochondritis dissecans (OCD). When a flap forms, cartilage degradation products reach the synovial fluid, causing synovitis, effusion, joint pain, and lameness. The resultant flap of cartilage may remain within the defect or may become dislodged. Cartilage flaps that remain in the defect may reattach to the underlying subchondral bone, or the flap may break free, forming joint mice. Joint mice may be resorbed in synovial recesses or remain in the joint, causing synovitis and osteoarthritis (OA). The cause of OC has not been determined, but a multifactorial complex of factors, including genetics, rapid growth, overnutrition and excess dietary calcium, trauma, ischemia, and hormonal influences, has been implicated [34–36].

The shoulder (caudal humeral head) is the most frequently involved joint in dogs; however, the disease is also seen in the elbow (medial portion of the humeral condyle), stifle (lateral condyle of the femur), and hock (plantar aspect of the medial trochlear ridge). OC has also been reported in the medial femoral condyle, dorsal aspect of the lateral trochlear ridge and head of the femur,
dorsal rim of the acetabulum, scapular glenoid cavity, and cervical vertebrae [35,37,38].

Clinical signs
The most common clinical sign is mild to moderate unilateral lameness, even with bilateral disease. The lameness is usually gradual in onset, and it improves with rest and worsens with exercise. Pain is elicited on palpation of affected joints. Stiffness and reduced range of motion may be present. Joint effusion may be palpable in some joints affected with OC. Muscle atrophy may be present, particularly in more chronic cases.

Diagnosis
A presumptive diagnosis of OC is based on the history and physical examination findings, but radiographs are necessary to confirm the diagnosis and should be taken bilaterally. OC typically appears radiographically as subchondral bone radiolucency or flattening [30,39,40]. Joint effusion or an increase in joint space may also be observed. If mineralized, cartilage flaps or joint mice may be visible radiographically. The most useful radiographic views for the diagnosis of OC depend on the joint involved. Other diagnostic techniques useful in the diagnosis of OC include arthroscopy, CT, and contrast arthrography.

Scapulohumeral joint
OC lesions are most commonly located on the caudal central aspect of the humeral head and visualized on mediolateral radiographs [30,39,40].

Fig. 3. Microscopic view of an OC lesion in the dog. Note the area of thickened articular cartilage separating the lesion from the underlying subchondral bone.
Cubital joint
OC lesions are typically located on the medial aspect of the humeral condyle and visualized on mediolateral, flexed mediolateral, and craniocaudal (elbow flexed 90° plus slight medial rotation) radiographic views [41–44].

Stifle joint
OC lesions usually appear on craniocaudal and lateral radiographic projections as a flattening of the subchondral bone of the lateral (96%) or medial (4%) femoral condyle. The medial aspect of the lateral femoral condyle is the most common location reported [45–47].

Tibiotarsal joint
Most (79%) tarsal OCD lesions involve the medial trochlear ridge of the talus [48]. Eighty percent of medial ridge lesions occur on the plantar aspect of the ridge. Twenty-one percent of tarsal OCD lesions involve the lateral trochlear ridge (70% of these lesions are on the dorsal aspect of the ridge) [40,49,50]. Approximately 90% of the dogs with lateral trochlear ridge lesions are Rottweilers [51]. Radiographic evaluation should include standard lateral, flexed lateral, and dorsoplantar views of both tarsi. Additional views are also helpful, including a craniocaudal view of the proximal trochlear ridges, a dorsolateral-plantomedial oblique view (D45°L-PLMO), and a dorsomedial-plantolateral oblique view (D45°M-PLLO) [52–54].

Treatment
Conservative therapy and surgery have been advocated for treatment of OC in dogs. Recommended treatment varies according to the joint affected, severity and chronicity of the lesion, clinician’s experience, and financial restraints of the client.

Shoulder
Conservative treatment of shoulder OC may be warranted for dogs less than 7 months of age with mild lesions radiographically and no clinical pain or joint mice. Conservative therapy consists of strict rest for up to 6 weeks, restricted diet, NSAIDS, OA disease-modifying agents, and analgesics. Alterations in the diet include decreasing caloric intake, and stopping calcium supplementation may also be indicated [55]. If lameness persists for more than 4 to 6 weeks, surgery should be performed [56].

Surgery is recommended in dogs if a flap is present, the dog has been lame for more than 6 weeks, the dog is older than 8 months of age, a joint mouse is evident on radiographs, or the lesion is large. Surgical treatment provides a more rapid return to function and minimizes the development of OA. Arthroscopy is the preferred surgical treatment because it is less invasive and allows a more rapid return to limb function [29,57,58]. Whether an arthrotomy or arthroscopy is performed, the goal of surgery is to remove the cartilage flap or joint mice, remove cartilage in the periphery of the lesion that is not adhering to the underlying tissue, and stimulate defect healing. Healing of the defect requires bleeding from the subchondral bone to bring in mesenchymal
cells and a fibrin clot [59]. Fibrocartilage eventually fills the defect. This bleeding can be induced by curettage, forage, or abrasion arthroplasty.

**Elbow**

Treatment of elbow OCD may include medical management or surgery. Surgical therapy involves removal of the cartilaginous flap with or without curettage of the defect bed. Several surgical techniques exist, but most surgeons prefer a medial approach and arthrotomy. Arthroscopic techniques are becoming more popular and allow exploration of the entire joint, removal of the cartilage flap, and forage or curettage of the defect [59,60]. Whenever possible, arthroscopic treatment is preferred.

**Stifle**

Medical management of stifle OCD is most successful in patients with mild lameness and only a small subchondral lesion evident radiographically. Surgical treatment is preferred in patients with persistent lameness, joint mice, or larger radiographic lesions. Arthrotomy or arthroscopy may be used to explore the joint, excise the cartilage flap and joint mice, and promote healing of the defect [61–64].

**Hock**

Medical management for tarsal OCD is recommended in older dogs with severe degenerative changes. Although most reports suggest that surgical intervention is preferred for treating tarsal OC, two recent studies found no significant differences in long-term outcome between joints treated medically and those treated surgically [64,65]. This is likely to be particularly true in dogs with chronic disease and significant OA. Surgical exploration and removal of the cartilage flap or osteochondral fragment can allow ingrowth of fibrocartilage from the underlying subchondral bone. Early intervention with a minimally invasive approach is preferred. Once the lesion is exposed, the cartilage flap or osteochondral fragment is excised. Overall function is better with minimal curettage. Arthroscopy of the tarsus has also been described for evaluation and diagnosis of tarsal diseases [66–68]. Arthroscopic removal of OCD fragments is possible in the dorsal aspect but can be difficult on the plantar aspect of the talus [67].

**Prognosis**

The prognosis after treatment of OC depends on the affected joint and whether medical or surgical treatment is used.

**Shoulder**

The prognosis after surgical treatment of shoulder OCD is good to excellent. Mild OA often develops in the operated shoulder over time, although lameness typically resolves and limb function is good [27]. Older dogs with chronic lameness and OA have a more guarded prognosis; however, most dogs return to normal function within 4 to 8 weeks after surgery.
**Elbow**
The prognosis for medical or surgical treatment of elbow OCD is guarded. Progression of secondary degenerative joint disease is common after medical management. Early surgical treatment of the OCD lesion does decrease lameness but may not prevent the progression of OA.

**Stifle**
The prognosis for dogs with stifle OC is guarded to fair. Progression of OA is common even after surgery. The severity of OA present before surgery, the size and location of the defect, and the quality of postoperative physical therapy all affect the long-term prognosis.

**Hock**
The prognosis for OC of the tarsus after conservative therapy is guarded. Most dogs have intermittent lameness and moderate progression of OA. After surgery, OA is likely and often requires medical therapy to control pain and lameness. Despite the progression of OA noted radiographically after surgery, many dogs are clinically improved. Nevertheless, the prognosis remains guarded, because joint pain and lameness may recur as the OA progresses. Recovery is faster in dogs with lesions involving the non-weight-bearing dorsal aspect of the lateral trochlear ridge [69]. Several factors may influence the success of medical and surgical treatment, including the age of the dog, presence of OA, size of the osteochondral defect, presence of joint instability, site of the lesion, and whether the lesions are unilateral or bilateral.

**LEGG-CALVÉ-PERTHES DISEASE**

**Signalment**
Legg-Calvé-Perthes Disease (LCPD), or avascular necrosis of the femoral head, is a developmental condition that occurs in primarily toy- and miniature-breed dogs [70–72]. Most patients are 4 to 11 months of age, and male and female dogs are equally represented. LCPD is reportedly bilateral in 12% to 16% of cases [70–73].

**Etiology and pathogenesis**
The etiology of femoral head necrosis in patients with LCPD is unknown. Numerous suspected causes have been investigated, including infection, trauma, metabolic and hormonal imbalances, vascular abnormalities, and genetics. The normal femoral head receives its blood supply from epiphyseal vessels that enter the epiphysis near the joint capsule insertion. Compromise of these vessels may cause ischemic insult to the epiphyseal spongiosa and its marrow elements. Synovitis or a sustained abnormal limb position may cause sufficient increases in intra-articular pressure to collapse fragile veins and deprive the femoral head of blood flow, resulting in regional or generalized necrosis [70,71]. Initially, the necrotic bone remains mechanically sound and continues to support the articular cartilage. Over time, however, the subchondral bony plate and overlying cartilage collapse, leading to a loss of
normal contour of the femoral head and secondary OA [70–73]. In cases of LCPD, transient or temporary vascular compromise rather than a permanent vascular insult is suspected, because reparative fibrosis and osteoblastic activity can be seen histologically after the initial ischemic phase of the disease [74]. In human beings, LCPD is thought to be inherited [74]. LCPD is also an inherited condition in Manchester Terriers [75].

Clinical signs
Clinical signs of LCPD are typically seen between 4 and 11 months of age. Most dogs have an acute onset of non–weight-bearing lameness or an intermittent subtle lameness [70–73]. Recent trauma may be reported by the owners. Other signs of LCPD include hip pain, crepitus of the hip during palpation, and muscle atrophy.

Diagnosis
The diagnosis of LCPD is based on history, physical examination, and radiographic findings. Early radiographic changes include increased radio-pacity of the lateral epiphyseal area of the femoral head and focal bony lysis as resorption of the bony trabeculae progresses [74]. Later, flattening and a mottled appearance of the femoral head, collapse and thickening of the femoral neck, and potential femoral neck fractures can be seen on radiographs (Fig. 4) [9]. Degenerative joint disease and atrophy of the thigh muscles also become increasingly apparent later in the disease.

Treatment
Conservative and surgical treatment options have been reported for LCPD. Conservative therapy consists of rest, limited exercise, appropriate nutrition, and NSAIDs for analgesia. Published reports indicate that lameness resolves in less than 25% of affected dogs managed conservatively [72,73,76]. The preferred treatment for LCPD is femoral head and neck excisional arthroplasty. Surgery alleviates pain and lameness in 84% to 100% of patients regardless of age and progression of the disease [77].

Prognosis
The prognosis for a return to normal function is good to excellent in dogs that have surgery to remove the femoral head and neck. After surgery, passive range-of-motion exercises and controlled active exercise are encouraged to promote the creation of functional pseudoarthrosis. The degree of muscle atrophy present in the limb before surgery can affect the prognosis for limb function. Continue lameness is expected for patients that do not have surgery or if surgery is inadequate. Fracture of the femoral neck may occur in untreated patients. Proper technique in removing the femoral head and neck is critical for long-term success.

HIP DYSPLASIA
Signalment
Canine hip dysplasia (HD) most commonly affects large-breed dogs but is also seen in many small-breed dogs and cats [78–80]. Most dogs show clinical signs
of hip pain and lameness between 4 and 10 months of age. Many dogs are presented for treatment of HD after reaching skeletal maturity because of OA, however. Retrievers, Rottweilers, German Shepherds, and Saint Bernards are among the commonly affected breeds [78–80].

**Etiology and pathogenesis**

HD is a common skeletal developmental defect produced by a genetic predisposition to subluxation of the immature hip joint. Joint laxity is the initiating cause of dysplasia and leads to subluxation and poor congruence between the femoral head and the acetabulum. Abnormal forces develop across the joint that interfere with normal development and overload areas of articular cartilage. Over time, degeneration of the joint occurs. Numerous factors influence the development and progression of HD, including genetics, rapid weight gain in growing animals, a high nutrition level, and pelvic muscle mass.
Clinical signs
HD usually affects both hips, although one hip may appear more severely affected than the other. In many cases, dogs are presented for lameness in only one hind limb. Clinical signs of HD vary and may include decreased activity, difficulty in rising, reluctance to run or climb stairs, intermittent hind limb lameness, “bunny hopping,” swaying gait, narrow stance, hip pain, atrophy of the thigh muscles, hypertrophy of the shoulder muscles, crepitus, and reduced hip joint motion. Joint laxity (detected clinically by a positive Ortolani sign) is characteristic of early HD; however, joint laxity may no longer be present in chronic cases because of periarticular fibrosis. In many cases, early signs are overlooked by owners and the dogs may not be presented for veterinary care until late in the disease when OA is severe.

Diagnosis
The diagnosis of HD is based on physical examination and radiographic findings. Physical examination typically reveals hip pain and a reduced range of motion. Most dogs are in pain on extension and abduction of the hip joint. Gait abnormalities are often observed and usually worsen with exercise.

Radiographic findings consistent with HD include hip joint laxity (subluxation) or secondary morphometric and degenerative changes within the joint [81]. Early in the disease, the shape of the acetabulum and femoral head is normal and the primary radiographic finding is joint incongruity. Identification of this joint laxity is essential to the early diagnosis of HD. Joint subluxation may be observed subjectively on a ventrodorsal hip-extended radiographic view of the pelvis [82,83]. The degree of subluxation can be quantified by measuring the Norberg angle (angle formed by a line drawn from the center of the femoral head to the cranial acetabular rim and a line drawn between the centers of the two femoral heads) or by calculating the percentage coverage of the femoral head by the acetabulum. Unfortunately, the magnitude of joint subluxation observed on ventrodorsal hip-extended radiographs is positionally dependent and can vary significantly in sequential radiographs obtained in the same dog. Additionally, the twisting of the joint capsule that occurs when the dog is placed in this position can mask the presence of joint laxity [84].

Distraction radiography provides a more sensitive means of identifying and measuring hip joint laxity. With the PennHip radiographic technique, the dog is positioned in dorsal recumbency with the femurs in a neutral position [85,86]. Distraction is created by placing a wedge between the dog’s thighs and applying a mediadly directed force to the stifles. The distance the femoral head moves out of the acetabulum is measured to obtain a “distraction index,” which is a measure of the passive laxity present in the joint. Measurement of the distraction index has been shown to be a reliable predictor of the eventual development of OA in lax hip joints. Another distraction radiographic technique is the dorsolateral subluxation (DLS) test. The dog is positioned in sternal recumbency with the knees flexed and adducted so that the femurs are perpendicular to the table surface [87,88]. With the hind limbs in this kneeling
position, weight bearing is simulated and the femoral head subluxates dorsolaterally when laxity is present.

In more chronic cases of HD, radiographic changes indicative of joint degeneration and remodeling can be seen [81]. As abnormal forces continue to act on the lax hip joint, the acetabulum becomes shallow and the femoral head begins to flatten. Osteophytes form at the joint margins and are apparent radiographically as new bone production. The acetabular margin becomes irregular, and the femoral neck becomes thicker as the osteophytes are formed. Sclerosis of the subchondral bone develops and is often most apparent on the craniodorsal acetabular rim. Fibrosis and increased density of the periarticular soft tissues are also apparent radiographically. Because of periarticular fibrosis, joint laxity may no longer be a major component of the disease in chronic HD.

**Treatment**

Medical and surgical treatments have been used for HD in young dogs (Table 1). Medical treatment typically includes weight control or weight loss, physical therapy and exercise control, NSAIDs, and OA disease–modifying agents [89–99]. Medical therapy is quite successful in many cases.

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<tr>
<th>Table 1</th>
<th>Considerations when selecting treatment for hip dysplasia</th>
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<tr>
<td><strong>Treatment</strong></td>
<td><strong>Patient age</strong></td>
</tr>
<tr>
<td>Medical therapy</td>
<td>Any age</td>
</tr>
<tr>
<td>JPS</td>
<td>3–4 months</td>
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<tr>
<td>TPO</td>
<td>Less than 10 months</td>
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<tr>
<td>THA</td>
<td>After skeletal maturity (older than 10–12 months)</td>
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<td>FHNE</td>
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*Abbreviations: FHNE, femoral head and neck excision; JPS, juvenile pubic symphysiodesis; OA, osteoarthritis; THA, total hip arthroplasty; TPO, triple pelvic osteotomy.*
Surgical techniques used to treat HD depend on the size and age of the dog, the amount of OA present, cost, and clinician preference. In young dogs with hip joint laxity and no OA, a triple pelvic osteotomy (TPO) is often recommended [100–103]. A TPO is a corrective surgical procedure that reorients the acetabulum to establish congruity between the femoral head and acetabulum. It increases acetabular coverage of the femoral head to eliminate subluxation and improve joint stability. To be successful, the procedure must be performed early in the disease process, before OA changes develop and while the remodeling capability exists to allow development of a more congruent joint [101,102]. Ideally, a TPO should be performed in dogs less than 10 months of age. A TPO is contraindicated once OA is present. Whether a TPO is indicated in asymptomatic dogs with radiographic evidence of hip laxity remains controversial; however, many clinicians believe the potential success of conservative medical therapy precludes the use of a TPO unless significant clinical signs are present.

Another surgical procedure recommended for young dogs without evidence of OA is juvenile pubic symphysiodesis (JPS). JPS is a relatively simple surgical procedure used to close the pubic symphysis prematurely. Premature closure of the pubic symphysis results in ventrolateral rotation of the acetabulum, which increases acetabular ventroversion and the acetabular angle to improve coverage of the femoral head [104]. A ventral approach to the pubis is performed, and electrocautery is applied every 2 to 3 mm along the symphysis at 40 W for 12 to 30 seconds [105]. Improvement in hip conformation is greater when the procedure is performed in dogs between 3 and 4 months of age [106]. When JPS was performed in dogs at 15 weeks of age, the acetabular angle was increased by 16°. When JPS was performed in dogs at 20 weeks of age, the increase in acetabular angle was only 8°, however. Dogs older than 6 months of age do not benefit from JPS surgery [107,108]. Few complications are reported with JPS, although narrowing of the pelvic canal does occur.

In dysplastic dogs with OA that are unresponsive to medical therapy, total hip arthroplasty (THA) is recommended [109]. The procedure to implant the prosthetic hip components requires specialized equipment and training but yields excellent results in most cases. Generally, THA is performed after the dog reaches skeletal maturity. If possible, the dysplastic dogs are managed medically until they are at least 10 to 12 months of age before performing THA. Alternatively, femoral head and neck excision (FHNE) arthroplasty can be performed [110,111]. The goal is to eliminate hip pain by removing the femoral head and neck and initiating the development of a fibrous pseudoarthrosis that permits ambulation. The procedure can be performed in dogs of all sizes; however, results are usually better in smaller and lighter dogs (< 20 kg). FHNE arthroplasty is the current surgery of choice for smaller dogs with HD that are unresponsive to medical treatment. FHNE arthroplasty can also be used with success in large dogs.
Prognosis
The prognosis for dogs with HD is variable. With proper medical management, many dogs maintain a good quality of life without surgical intervention. The prognosis after surgical treatment is good with proper patient selection, sound surgical technique, and proper postoperative management.

ELBOW DYSPLASIA
Canine elbow dysplasia (CED) is a term used to describe all developmental conditions resulting in elbow arthrosis, regardless of the underlying cause. Currently, CED is typically used to describe a complex of developmental abnormalities of the elbow, including ununited anconeal process (UAP), fragmented medial cornoid process (FMCP) (Fig. 5), OC of the medial portion of the humeral condyle, and elbow incongruity [112–118].

Signalment
CED typically affects large- and giant-breed dogs that are rapidly growing. CED may also affect medium-sized and chondrodystrophic dogs [114,116,119–121]. No sex predilection has been observed in dogs with UAP and OC. Male dogs are more commonly affected with FMCP [122]. Bilateral joint involvement is common, and the right and left limbs are equally represented.

Etiology and pathogenesis
The etiology and pathogenesis of CED remain poorly understood, although genetics, nutritional excesses or deficiencies, growth disturbances, OC, and trauma are proposed causes. Pathologic mechanisms proposed to explain the

Fig. 5. Elbow specimen (cadaver) from a dog with an FMCP (arrow). (Courtesy of Roy R. Pool, DVM, PhD, Texas A&M College of Veterinary Medicine, College Station, Texas.)
development of the primary lesions of CED are OC, trochlear notch dysplasia, and asynchronous growth of the radius and ulna.

Clinical signs
Forelimb lameness is usually present for several months beginning at 4 to 12 months of age. Younger dogs and dogs as old as 8 years of age have been diagnosed, however. The lameness is gradual and progressive and usually worse after exercise. Other signs of CED include short striding and difficulty in rising or lying down. Most dogs with CED sit or stand with the elbow adducted and the carpus abducted. Palpation of the elbows often reveals soft tissue swelling, muscle atrophy, pain, and crepitus. Pain in the elbow is noted on flexion or when the antibrachium is pronated or supinated. A reduced range of motion may also be noted in some dogs.

Diagnosis
A thorough physical examination with lameness evaluation at a walk, trot, and circling figure-of-eight pattern should be performed. Although dogs normally place 60% of their body weight on their forelimbs, dogs with CED often place only 40% to 50%. Both elbows are radiographed to identify bilateral disease and to allow comparison between joints. Several radiographic views are recommended, including a craniocaudal, mediolateral, flexed mediolateral, and craniocaudal medial-to-lateral oblique with the elbow maximally extended and supinated 15°; a mediodistal-to-lateroproximal 30° oblique view is also helpful in some cases [114,123]. Positive-contrast arthrography may be used if OC is suspected to determine the size of subchondral defects, the presence of a radiolucent flap, and the presence of unmineralized free joint bodies [124]. CT and MRI are extremely helpful in the diagnosis of elbow diseases. Nuclear scintigraphy and arthroscopy may also be helpful in confirming the presence of CED.

Fragmented medial coronoid process
Treatment
Medical therapy for FMCP includes weight control, activity restrictions, and medications for pain and OA. Surgical treatment involves the removal of loose or free-floating cartilage or bone fragments and correction of articular incongruence [124,125]. Surgery is performed by medial elbow arthrotomy or arthroscopy (Fig. 6). In most cases, surgery is recommended for dogs with clinical or radiographic signs of FMCP that are less than 12 months of age and for older dogs with large lesions [126]. Dogs with severe radiographic signs of OA may be poor candidates for surgery and are often managed conservatively. Two published studies found that surgical intervention had little advantage over conservative medial therapy in dogs with FMCP [127,128]. Many surgeons find that clinical function improves after surgical removal of the fragmented coronoid process, however, although lameness and pain often recur as OA progresses.
Prognosis

The prognosis for dogs with FMCP varies and may depend primarily on the severity of clinical signs, progression of OA, and treatment used. Early diagnosis and treatment with surgery may provide the best clinical outcome. The increasing use of arthroscopy may also improve the prognosis in those patients undergoing surgery. Surgery is not curative, however, and secondary OA often develops, necessitating chronic medical therapy to control pain and lameness. Factors that do not seem to affect long-term outcome prognosis include the dog’s age at the time of surgery and the surgical approach used [129].

Ununited anconeal process

Treatment

Although the condition varies among breeds, the anconeal process remains ununited if it is not attached by 20 weeks of age (Fig. 7). Surgery is recommended for treatment of UAP. Surgical options include removal of the UAP, surgical reattachment, and osteotomy or ostectomy of the ulna with or without surgical fixation of the anconeal process [113,114,130]. Medical management alone is usually less successful than surgery, resulting in rapid progression of OA [131]. Surgical reattachment using a lag screw is usually attempted before 24 weeks of age. After 24 weeks of age, surgical removal of the UAP is usually recommended. Removal of the UAP may also be warranted after osteotomy of the ulna if fusion does not occur within 12 to 18 weeks after surgery.

Prognosis

Long-term evaluations have found that dogs treated with excision of the UAP have a favorable prognosis. A study of 10 dogs in which the UAP was
surgically reattached found encouraging results, though long-term studies are still needed [132]. Osteotomy of the ulna with or without lag screw fixation has produced good clinical outcomes in the long-term studies, but 30% of the patients developed signs of progressive OA [133].

Osteochondrosis dissecans

Treatment

Medical therapy is used primarily for small lesions and consists of rest, weight control, and medication consisting of NSAIDS and OA disease-modifying drugs. Surgery is typically performed by medial arthrotomy or arthroscopy. Arthroscopy provides a less invasive alternative to arthrotomy and is the preferred method of treatment [134,135].

Prognosis

The prognosis after medical or surgical treatment of elbow OCD is guarded. Progression of OA is common and may require chronic medical therapy to control clinical signs. Early surgical treatment of the OCD lesion often decreases lameness but may not prevent the progression of OA.

Elbow incongruity

Treatment

Elbow incongruity is likely caused by asynchronous growth of the radius and ulna and is treated surgically. Radioulnar bowing and rotation that clinically affect the elbow or carpal joint should be addressed early to avoid dysfunction and the potential for severe OA [13]. Addressing the disease with corrective ulnar or radial ostectomy or osteotomy can provide more synchronous growth and less stress on the elbow joint. In more severe cases, treatment options may

**Fig. 7.** Lateral radiographic view of the elbow joint from a dog with a UAP (arrow).
include medical therapy, arthrodesis, elbow replacement, corrective osteotomy, or limb amputation.

Prognosis
The prognosis for elbow incongruity varies and may depend on the patient’s age, severity of clinical signs, degree of incongruity, and progression of OA. Early surgical intervention may prevent or reduce angular limb deformities and progression of secondary OA.

**PES VARUS**

Signalment
Pes varus is characterized by a medial bowing of the distal tibia, resulting in deviation of the tarsus and phalanges toward midline (varus deformity). This DOD has been documented in dachshunds and is seen unilaterally or bilaterally [136].

Etiology and pathogenesis
Although there are no studies to validate the claim, this condition is thought to be genetic. Trauma to the medial distal tibial growth plate may cause a similar deformity, but most dogs presented for pes varus have no history of trauma.

Clinical signs
Muscle atrophy or lameness may be present on the clinically affected leg, or the patient may be asymptomatic despite the deformity.

Diagnosis
Physical examination and a thorough history to rule out trauma are usually sufficient to make this diagnosis. Radiographic evidence includes shortening of the medial aspect of the tibia in relation to the lateral cortex and thus a medial bowing of the distal tibia. Osteophyte formation can also be seen on the cranial aspect of the distal tibia [136].

Treatment
Clinical signs of lameness and muscle atrophy should be used to dictate the need for surgical intervention. An open wedge osteotomy and external fixation have been used [137].

Prognosis
Surgical intervention yields excellent results, and dogs without clinical signs do not seem to develop OA of the talocrural joint [137].

**SUMMARY**

DODs are a common cause of pain and lameness in dogs. Although the etiology of these diseases is not always known, the clinical and radiographic findings associated with each disease are well documented. A thorough history and careful physical examination often help to localize the abnormality; however, radiographic evaluation is usually required to confirm the diagnosis. Treatment varies depending on the type of developmental disease present.
References


