Canine Elbow Dysplasia

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Canine elbow dysplasia (CED) is a general term for several developmental abnormalities that involve the canine elbow (cubital) joint. The term elbow dysplasia was implemented by the International Elbow Working Group to describe all conditions resulting in elbow joint arthrosis, regardless of the underlying etiology. Dogs diagnosed with elbow dysplasia have pain and lameness due to one or more of the following conditions: ununited anconeal process (UAP) or fragmented medial coronoid process (FCP) of the ulna, osteochondrosis (OC) of the medial humeral condyle, or elbow incongruity. These conditions primarily affect fast-growing, large- and giant-breed dogs younger than 1 year, although small, medium-sized, and chondrodystrophic canine breeds may also develop CED. CED frequently involves both elbow joints, and OC and FCP can occur together in the same joint. There is no sex predilection in dogs with OC, but FCP and UAP have been observed more frequently in males than in females.

Young, large- and giant-breed dogs with forelimb lameness should be evaluated for CED. Early diagnosis and treatment are recommended to improve comfort and function as well as decrease the progression of osteoarthritis (OA). All dogs with FCP, osteochondritis dissecans (OCD), and UAP should be eliminated from breeding.

Anatomy and Pathogenesis
The elbow joint comprises the humeral, radial, and ulnar bones and their supporting ligaments. The elbow joint has three separate articulations: the humeroradial, humeroulnar, and proximal radioulnar joints, all of which are contained within a common joint capsule.

The major pathogenetic mechanisms of CED are OC (e.g., OCD), trochlear notch dysplasia (e.g., FCP, UAP, OCD), and asynchronous growth of the radius and ulna (e.g., FCP, UAP, elbow incongruity). Numerous factors have been implicated in the pathogenesis of CED, including genetics, nutritional imbalances (excesses or deficiencies), growth disturbances, hormonal influences, and trauma.

Diagnosis
Clinical signs of CED are usually first noted at 4 to 7 months of age, although they may not appear until the dog is older and develops signs of OA. An orthopedic examination may reveal elbow joint swelling, crepitus, and pain during elbow flexion or extension; reduced range of motion; and muscle atrophy. The degree of lameness may vary and is frequently most pronounced after exercise or rising. Dogs normally bear 60% of their body weight on the forelimbs, but painful dogs with CED may place only 40% to 50% of their weight on the forelimbs. Affected dogs may ambulate in short strides and often sit or stand with the affected elbow close to their bodies (adduction) while rotating the affected foot outward (abduction).

Diagnosis is confirmed by radiography or computed tomography (CT). Radiography can be used to diagnose UAP, most OCD lesions, and, often, elbow incongruity, but it is less effective for diagnosing FCP. Radiography is also useful for detecting advanced stages of OA secondary to CED and for ruling out other conditions, such as fractures or dislocations. However, CT is one of the best modalities for visualizing FCP lesions. CT allows three-dimensional visual...
Canine Elbow Dysplasia

Canine Elbow Dysplasia (CED) is a hereditary disorder that affects the elbow joint of dogs. It is characterized by the development of osteoarthritis (OA) and is typically diagnosed in young, large-breed dogs. CED can lead to pain, lameness, and decreased mobility in affected dogs.

CT imaging is more sensitive than traditional radiography for diagnosing CED. It provides a three-dimensional view of the elbow joint, allowing for a thorough evaluation of specific regions. CT can detect early changes in the elbow joint that may not be visible on radiographs, leading to earlier intervention and improved prognosis in affected dogs.

Other diagnostic imaging options include positive-contrast arthrography, magnetic resonance imaging (MRI), nuclear scintigraphy, and arthroscopy. Arthroscopy is considered the gold standard for treating CED because it can also be used for diagnosis.

Treatment
Medical and surgical options are available for treating CED. Conservative therapy of CED is analogous to treating OA and may consist of rest, weight control, judicious exercise, and administration of analgesic medications (including NSAIDs). Young dogs diagnosed before the onset of degenerative joint disease may have a better prognosis after surgery.

Arthroscopy is considered the gold standard for treating CED because it can also be used for diagnosis. This procedure allows for the diagnosis and treatment of CED by directly visualizing the joint and performing necessary procedures such as repair or removal of the medial coronoid process (FCP).

Reconstruction of the joint for thorough evaluation of specific regions of the elbow. Therefore, CT is more sensitive for diagnosing CED, providing fewer false-negative results than traditional radiography.

(FIGURE 3). Additionally, dogs with forelimb pain or lameness but apparently “normal” elbow radiographs have been diagnosed with CED using CT, thereby leading to earlier intervention and improved prognosis in these patients.

Other diagnostic imaging options include positive-contrast arthrography (for OC), magnetic resonance imaging, nuclear scintigraphy, and arthroscopy.

**FIGURE 2.** (A) Normal anatomy of the canine left elbow joint (lateral aspect). (B) Normal anatomy of the canine left elbow joint (cranial view). (C) Normal anatomy of the canine left elbow joint (medial aspect).

**FIGURE 3.** Fragmented medial coronoid process (FCP) of a dog’s left elbow (arrows). (A) A CT scan showing the FCP lesion. (B) A CT scan showing the entire FCP lesion after three-dimensional reconstruction of the left elbow joint. (Reprinted with permission from Anthony J. Fischetti, DVM, MS, DACVR; Department of Diagnostic Imaging, The Animal Medical Center, New York, NY; phone: 212-838-8100)

**FIGURE 4.** Diagnostic arthroscopy is considered the gold standard for treating CED because it can also be used for diagnosis.
Canine Elbow Dysplasia

Open arthroscopy, minimally invasive arthroscopy has many advantages, including smaller incisions requiring little postoperative wound care, the ability to perform copious flushing, superior joint surface visualization due to magnification and illumination, a decreased risk of infection, a low rate of complications, a rapid recovery, and earlier return to function, resulting in decreased patient morbidity and the ability to treat multiple joints during the same procedure. Additional, arthroscopy facilitates removal of abnormal tissue and allows evaluation of elbow incongruity.

Introduced in 2004, the TATE Elbow System (BioMedtrix, Boonton, NJ; Figure 5) has had a success rate of ~80% in select cases but is considered unproven by many.

Osteochondritis Dissecans

OC has been implicated in the development of OCD. OC is a disturbance of cartilage and bone formation during growth that leads to the thickening of the joint cartilage, which often progresses to separation of the cartilage (fissuring) and formation of a flap. OC has been associated with the shoulder, stifle, and tarsus but affects the medial humeral condyle most frequently. OC that results in cartilage fissuring and a subsequent flap is known as OCD (Figure 6). OCD results in pain, swelling, inflammation, and, eventually, OA. A loose cartilage flap that becomes completely detached may be referred to as a joint mouse.

Most affected dogs belong to rapid-growing, large or giant breeds such as the Newfoundland, Labrador retriever, and golden retriever. Other affected breeds include the Bernese mountain dog, chow chow, German shepherd, mastiff, Old English sheepdog, rottweiler, and standard poodle.

Surgical treatment involves removal of the cartilage flap and abrasion arthroplasty (curettage of subchondral bone until bleeding is observed) or forage (drilling small holes into the bone) of the defect bed to promote healing of the defect with fibrocartilage.

The prognosis after medical or surgical treatment of OCD is good but is limited by the progression of secondary joint disease.

Fragmented Medial Coronoid Process

FCP is the most frequently encountered manifestation of elbow dysplasia. The coronoid process of the ulna forms the distal extent of the trochlear notch, where most of the humeroulnar joint surface comes into contact with the humerus and ulna. An FCP lesion develops when fragmentation or fissuring of the cranialateral aspect of the medial portion of the coronoid process leads to separation from the proximal ulnar bone (Figure 7). Delayed ossification of the coronoid process may also be a contributing factor to developing FCP.

FCP is also prevalent in dogs with ulnae that are proportionately longer than their radii (i.e., asynchronous growth of the radius and ulna). This abnormality results in overloading and failure of the medial coronoid process. Most asynchronous growth of the radius and ulna occurs before 6 months of age and may be most notable in achondroplastic dwarfs (i.e., dogs with chondrodysplasia), chondrodystrophic breeds, large-breed dogs with a retained ulnar cartilaginous core, and normal dogs with physeal trauma.

Elbow incongruity due to trochlear notch dysplasia was proposed by Wind et al during a study involving Bernese mountain dogs. The trochlear notch of the ulna articulates with the trochlea of the humerus.
humerus. Slight misshaping in the curvature of the trochlear notch puts excessive pressure on the coronoid and anconeal processes of the ulna and the medial humeral condyle. Trochlear notch dysplasia in the Bernese mountain dogs was determined to result in failure of the coronoid process due to overload, fatigue, and fracture.1

FCP commonly affects large- and giant-breed dogs such as Labrador retrievers, rottweilers, Bernese mountain dogs, Newfoundlands, golden retrievers, and chow chows. Clinical signs, including pain, lameness, swelling, and OA, become apparent at 5 to 7 months of age.4

Surgical treatment consists of correcting articular incongruity and removing loose or free-floating cartilage or bone fragments via arthroscopy or arthrotomy.3,4 Diagnosis and treatment before the development of severe clinical signs have been associated with better prognoses. However, dogs with evidence of severe OA may not respond as well and may require long-term, conservative treatment.3

**Ununited Anconeal Process**

UAP is a failure of the anconeal process to fuse to the ulna by 5 months of age and can often be diagnosed from a lateral radiograph of a fully flexed elbow joint. (Reprinted with permission from Dr. Nick Parker, DACVS, Alta Vista Animal Hospital, Ottawa, Ontario)

Figure 8. UAP (arrow) is a failure of the anconeal process to fuse to the ulna by 5 months of age. UAP can often be diagnosed from a lateral radiograph of a fully flexed elbow joint. (Reprinted with permission from Dr. Nick Parker, DACVS, Alta Vista Animal Hospital, Ottawa, Ontario)

Figure 9. Premature closure of the distal radial or ulnar growth plate may result in elbow incongruity. (Reprinted with permission from Fossum TW. Management of specific fractures. Small Animal Surgery. St Louis, MO: Elsevier; 2002:955. © Elsevier)

Elbow Incongruity typically occurs in chondrodystrophic breeds with a genetic predisposition to premature closure of a growth plate, but it can occur either without apparent growth plate injury or secondary to trauma.4 Although elbow incongruity can be caused by premature partial or complete closure of the distal radial physis, trauma to the distal ulnar physis is the most common cause of growth deformities in immature, nonchondrodystrophic dogs. Due to the location and cone shape of the distal ulnar growth plate, this plate is most susceptible to injury and subsequent early
Canine Elbow Dysplasia

Closure, resulting in decreased growth of the ulna. Elbow incongruity may be evidenced by radial bowing and external rotation of the elbow or carpus joint in the affected limb as well as by partial elbow dislocation.3,4 (FIGURE 9). Elbow incongruity can result in lameness, swelling, pain, and OA.

Premature closure of the distal ulnar or radial growth plates is not amenable to medical management; thus, early surgical intervention to restore congruity and alignment in the elbow joint is imperative to minimize the potential for the development of severe OA.3,4 Depending on the cause, treatment of elbow incongruity generally involves either ostectomy (FIGURE 10A; FIGURE 10B) or corrective osteotomy (FIGURE 11). Dogs diagnosed with elbow incongruity that have growth potential (i.e., radiographic evidence of open physes) may be treated with ostectomies and fat grafting, while corrective osteotomies are indicated in dogs with evidence of closed physes (i.e., in mature dogs).4

**Prognosis**

The prognosis for patients with CED depends on the severity of preexisting arthritic changes at the time of treatment. Many patients have clinical improvement despite residual OA and its progression, but all patients with CED demonstrate progression of OA and may display intermittent lameness or stiffness. These patients generally respond to administration of NSAIDs, chondroprotectants, or agents designed to slow cartilage degradation; weight management; judicious exercise; and diets containing a high concentration of omega-3 fatty acids, which can have anti-inflammatory effects that ease the clinical signs of OA.

**References**

1. The most frequently encountered manifestation of elbow dysplasia is
   a. OCD.
   b. FCP.
   c. UAP.
   d. elbow incongruity.

2. A good modality for diagnosing UAP is
   a. CT.
   b. magnetic resonance imaging.
   c. arthroscopy.
   d. radiography of a fully flexed elbow in a lateral view.

3. OC most frequently affects the
   a. medial humeral condyle.
   b. proximal humerus.
   c. stifle joint.
   d. tarsus.

4. The most common cause of growth deformities in immature, nonchondrodystrophic dogs is
   a. partial closure of the distal radial physis.
   b. complete closure of the distal radial physis.
   c. trauma to the distal ulnar physis.
   d. a retained ulnar cartilaginous core.

5. There may be a genetic predisposition to UAP in some
   a. Newfoundlands.
   b. German shepherds.
   c. Great Pyrenees.
   d. basset hounds.

6. The gold standard for treating CED is
   a. total elbow arthroplasty.
   b. arthrotomy.
   c. arthroscopy.
   d. corrective osteotomy.

7. Which manifestation(s) of CED does/do not have a sex predilection?
   a. OC
   b. FCP
   c. UAP
   d. a and c

8. Elbow incongruity due to trochlear notch dysplasia was identified in
   a. Labrador retrievers.
   b. Newfoundlands.
   c. golden retrievers.
   d. Bernese mountain dogs.

9. The major mechanisms implicated in the pathogenesis of CED include
   a. genetics, nutritional imbalances, and trauma.
   b. OC, trochlear notch dysplasia, and asynchronous growth of the radius and ulna.
   c. OCD, UAP, and FCP.
   d. delayed ossification of the coronoid process, UAP, and distal ulnar physis.

10. Treatment of elbow incongruity usually includes
    a. arthroscopy.
    b. screw-and-pin fixation.
    c. ostectomy or corrective osteotomy.
    d. abrasion arthroplasty.