

# Canine Elbow Dysplasia

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**Introduction:**

Canine elbow dysplasia is a common orthopedic disease in large and giant breed dogs. The disease was first defined by the Elbow Working Group in 1993, encompassing a fragmented medial coronoid process, osteochondrosis, ununited anconeal process, and elbow joint incongruity. This condition is classified as a developmental orthopedic disease, with typical age of presentation of less than 1 year. While elbow dysplasia is an inherited disease there has not been a specific gene identified. Diagnosis is key to determining treatment of choice which ranges from medical management to salvage procedures. Prognosis is dependent on the underlying pathology and degree of secondary osteoarthritis at time of presentation.<sup>3,10,11,12</sup>

**History and presentation:**

Cooper, an 11 month old, intact male St. Bernard, was presented to MSU-CVM Small Animal Surgery service on November 13, 2017 for forelimb lameness. Cooper was purchased from a breeder when he was approximately 10 weeks old and had been apparently healthy prior to this incident. About 3 months prior to presentation, he was observed falling while he was playing. He yelped out and appeared suddenly lame upon standing. At this time, he presented to his referring veterinarian where radiographs of his right forelimb and pelvis were performed. According to the referring veterinarian, no significant findings were noted on either study. Cooper was prescribed carprofen and sent home with instructions to rest. His lameness did not improve on NSAIDs and rest, thus he was taken to a different veterinarian for a second opinion. Radiographs were again taken and no significant findings were observed, according to his owner. He was again prescribed carprofen and sent home to rest. Cooper's owner then consulted the internet and diagnosed him with shoulder pain, ordering a bilateral shoulder brace which he wore for 2 weeks. While wearing this brace, Cooper appeared uncomfortable, frequently licking at his

forelimbs. Upon further investigation, his owner noticed a laceration and significant irritation in his axillary region where the brace contacted his skin, and feared that a ligament was exposed. Cooper was again taken to his referring veterinarian on November 10<sup>th</sup>, 2017 due to a malodorous discharge that was now coming from the wound. The wound was cleaned and cefpodoxime and a “wound spray” were prescribed. Due to Cooper’s persistent lameness and his owner’s increasing concern, he was then referred to MSU CVM for further work up.

On presentation at MSU CVM, Cooper was bright alert and responsive. His vitals were within normal limits with a temperature of 102.0 F, pulse of 96 beats per minute, and respiration of 40 breaths per minute. There was moderate skin irritation in his cranial axillary region bilaterally, presumptively from the brace he had been wearing. He had a grade 2/4 lameness on his right forelimb and rotated the paw laterally when he walked. On orthopedic exam he was painful on palpation of the coronoid processes of the right elbow, but appeared to have a full range of motion in the elbow joint. There was mild effusion on the medial and lateral side of the right elbow.

### **Pathophysiology:**

Elbow dysplasia is a common orthopedic disease typically seen in large and giant breed dogs, but can affect chondrodystrophic breeds as well. It has been reported in the Netherlands that approximately 17% of Labrador Retrievers and 70% of Bernese Mountain Dogs show signs of elbow dysplasia, with 35% of those affected having bilateral disease.<sup>10</sup> There is a 50% correlation in pure-bred dogs who develop elbow dysplasia will also likely to develop hip dysplasia.<sup>7</sup> There is a sex predilection for the disease, with males being 1.6 to 2 times as likely to develop elbow dysplasia compared to females. The typical age of presentation is less than one year of age, however dogs can present later in life due to trauma or progression of disease.<sup>10</sup>

Genetics play a large role in the development of elbow dysplasia, however a specific gene has not yet been identified in the pathophysiology of disease formation. Further work is needed to identify the heritable components of elbow dysplasia. It has been proposed that elbow dysplasia may have a multifactorial gene expression, with each component of the disease being coded for by its own set of genes. Environmental influences such as diet and exercise can play a role in speeding up or slowing down the progression of the disease. High energy diets in young, large breed dogs have been found to predispose them to develop osteochondrosis.<sup>12</sup> The four main pathologies of elbow dysplasia are described as fragmented medial coronoid process, ununited anconeal process, osteochondrosis of the humeral condyle, and joint incongruity. In general elbow dysplasia is caused by abnormalities within the joint.<sup>3,11,10</sup>

Fragmented medial coronoid process is the most common form of elbow dysplasia, and is caused either by abnormal forces on the joint or abnormal endochondral ossification during growth. A growing consensus in the literature is moving towards calling fragmented medial coronoid process “medial compartment disease”, due to the fact that there are typically additional pathologies of the joint that occur with a fragmented medial coronoid process. Medial compartment disease includes the previously recognized fragmented medial coronoid process as well as sclerosis, coronoid microfracture, coronoid fragmentation, and/or cartilage damage. The medial and lateral coronoid processes hold 50% of the weight during weight bearing, while the radial head supports the other 50%. During development in some dogs, there is a weakness in the trabecular bone of the coronoid process as it is forming. Over time, force fatigues the trabecular subchondral bone and microdamage occurs. This damage to the developing bone leads to abnormal forces on the coronoid process, which culminates in a fracture of the coronoid process. Sclerosis of the subchondral bone is considered one of the first signs of medial

coronoid disease and can lead to fissures which weaken the bone and contribute to fracture occurrence. Additionally, normal tension forces applied to the elbow from the annular ligament can result in avulsion fractures off the medial coronoid process due to abnormal weakness in the trabecular bone. Other forces may play a role in medial coronoid fracture or medial compartment disease when there is incongruency in the elbow. These forces include: pressure from the ulnar trochlear notch, shearing from incongruent radio-ulnar articulations, overload from an abnormally short radius, and/or pressure from an abnormally long radius. Once the medial coronoid process has fractured the fragment moved around the joint creating a kissing lesion and ultimately leads to osteoarthritis.<sup>12</sup>

An ununited anconeal process is diagnosed when the anconeal process has not fused or has only partially fused around 20-24 weeks of age. This will be seen as a lucent line separating the anconeal process from the ulna on radiographs. Two theories for the development of an ununited anconeal process predominate. These include failure of fusion from the secondary center of ossification and unequal growth of the radius and ulna. Some breeds that are predisposed to elbow dysplasia, ununited anconeal process to be specific, do not have secondary centers of ossification, leading some researchers to believe that the unequal growth of the radius and ulna is a more feasible explanation for the pathology. Unequal growth of the radius and ulna also leads to incongruity and a step defect in the joint.<sup>10</sup> One study found that 16% of German Shephard dogs with ununited anconeal process also had concurrent fragmented medial coronoid process.<sup>9</sup> This study provided further evidence that elbow dysplasia is a multifactorial disease.

The elbow is made up of three joints: the radioulnar joint, the humeroradial joint, and the humeroulnar joint. Incongruency of any of these joints can result in pathology that contributes to elbow dysplasia. Incongruencies of the elbow are seen as radioulnar mismatch, humeroulnar

incongruity, and/or radioulnar incisure incongruity. Radioulnar mismatch occurs when the radius and the ulna do not grow to the same length. A shorter radius will lead to medial coronoid disease and a shorter ulna will lead to an ununited anconeal process. Humeroulnar incongruity occurs when the curvature of the ulnar notch is less than the curvature of the humeral trochlea, or when the radius is too long and causes subluxation of the joint. Radioulnar incisure incongruity has only been reported in three cases and involves crushing of the coronoid process by abnormal forces. Each of these incongruities in the elbow lead to mild to severe step defects, which lead to abnormal forces on the components of each joint.<sup>5,12</sup>

Osteochondrosis occurs due to a failure of appropriate endochondral ossification. There is a failure of the cartilage matrix calcification which leads to cartilage retention and a thickening of the articular epiphyseal cartilage. At the thickened area there is a lack of nutrient diffusion and an area of necrosis forms. The normal and abnormal area separate and form a flap known as osteochondritis dessicans or OCD. The flap can remain attached or float loose within the joint causing joint irritation.<sup>3, 13</sup> OCD leads to cartilage injury within the elbow, which becomes more painful as the cartilage erodes.<sup>10</sup> Osteochondrosis normally develops in fast growing large breed dogs at 4 to 7 months of age.<sup>13</sup> The predilection site for OCD in dogs with elbow dysplasia is the medial humeral condyle. Studies have shown that 50% of dogs with medial coronoid disease also have osteochondrosis lesions on the humeral condyle.<sup>4,8</sup> Overall, elbow dysplasia occurs as any single or combination of the pathologies listed above, and results in chronic osteoarthritis which can lead to mild to debilitating pain and lameness in affected dogs.<sup>10</sup>

**Diagnostic approach/ consideration:**

A complete history, physical exam, and thorough orthopedic exam should be performed on all patients with a suspected forelimb lameness. Historical clues such as age of onset and

breed can help in the development of a differential list. Clinical signs in addition to forelimb lameness may include mild effusion in the elbow, thickening of the joint capsule, and a decreased range of motion of the elbow joint with pain on palpation and/or extreme flexion of the joint. The lameness observed typically consist of an offloading of the weight from the forelimbs on to the hindlimbs, and lateral rotation of the paw. Both of these maneuvers relieve pressure off the medial compartment of the elbow joint. Digital pressure applied over the area of the coronoid processes can elicit a painful response which can be suggestive of medial compartment disease.<sup>13</sup>

Diagnostic imaging is relied on to definitively diagnose elbow dysplasia. Radiographs can be useful, but are not as sensitive as other imaging modalities. Detecting pathology based on radiographs is highly dependent on appropriate patient positioning, however, even in the appropriately positioned patient, mild elbow dysplasia may be undetectable on radiographs. Radiography is more diagnostic in cases of osteochondritis dessicans, or an ununited anconeal process, but diagnosing an abnormal medial coronoid process may be challenging due to superimposition of other osseous structures over the medial coronoid process.<sup>10</sup> Changes seen on radiographs in medial coronoid disease include sclerosis at the semi-lunar notch and blunting of the medial coronoid process. Sometimes a fragment can be seen when there is medial coronoid disease. Oblique views are needed to isolate the coronoid processes.<sup>13</sup> Computed Tomography has a 71-88% sensitivity and 85% specificity in locating osseous fragments, and is particularly helpful in diagnosing a fractured medial coronoid process.<sup>2,10</sup> The gold standard for diagnosis of elbow dysplasia is arthroscopy, because it allows visualization of the entire joint, facilitating assessment of both the bony and cartilaginous changes. Interestingly, one study found that Computed Tomography and arthroscopy identified different lesions within the joint, suggesting

that utilizing both modalities may increase the likelihood of diagnosing all joint pathology.<sup>1</sup>

Other imaging modalities that may be used to evaluate the elbow include magnetic resonance and nuclear scintigraphy. A new, noninvasive technology has been described that uses medical infrared imaging to evaluate the elbow joint, however it lacks specificity in its current form.<sup>8</sup>

### **Treatment/ management:**

The ideally with treatment of elbow dysplasia is to correct the underlying problem before significant damage to the cartilage has occurred, however dogs rarely present before damage has occurred. Treatment is aimed at managing the disease. This can be difficult, due to the multifactorial etiologies of elbow dysplasia. Treatment options include a combination of medical management, and surgical procedures.<sup>9,12,13</sup>

Medical management frequently employs the use of both anti-inflammatories and rehabilitation. NSAIDs, chondroprotectants (ex. chondroitin sulfate), omega-3 fatty acids, and routine polysulfated glycosaminoglycan (Adequan<sup>®</sup>) injections may help dogs with mild disease. These therapies may also be helpful when used after surgery to delay the progression of osteoarthritis. Physical rehabilitation modalities including hydrotherapy, laser therapy, and joint exercises (e.g. passive range of motion) have shown promising results in increasing comfort of animals with mild disease in many case studies. While some of these methods have not been scientifically proven to be beneficial, they represent very low risk and have no known negative side effects. In patients with moderate to severe pain, surgical intervention may provide relief quicker than medical management and should be utilized in patients that fail to respond to an appropriate course of medical therapy.<sup>9,12,13</sup>

Procedures to treat medial compartment disease include removal of the fragment through arthroscopy or arthrotomy with subsequent cartilage removal, subtotal coronoidectomy, cartilage

debridement alone, proximal ulnar osteotomy, and biceps brachii release. Numerous studies have demonstrated long term outcome does not differ when comparing arthrotomy, arthroscopy, and medical management. However, removing the medial coronoid fragment has shown a decreased morbidity in patients because there is no longer a fragment floating in the joint and causing disruption to the cartilage. A subtotal coronoidectomy is thought to be more effective in comparison to fragment removal alone, because it addresses some of the additional pathology within the joint. However, there is no difference in long term outcome as the joints still become arthritic and painful over time.<sup>10,12,13</sup>

In the case of ununited anconeal processes, surgery has been shown to improve outcome in comparison to medical management. Three procedures are described in the literature including; proximal ulnar osteotomy, anconeal process removal, and anconeal process reattachment. There is no definitive evidence as to which procedure has a more desirable outcome, thus the decision on which procedure to pursue should be made based on the circumstances of each case and surgeon preference.<sup>10,13</sup>

There are two main treatment options for osteochondrosis. Both surgical options, the area of affected cartilage is first removed down to the subchondral bone. Then, the cartilage is either allowed to either heal over, or the defect is replaced by a plug of subchondral bone and cartilage. With both options, long term development of osteoarthritis is still likely.<sup>10,13</sup> Studies using adipose mesenchymal stem cells to help regenerate cartilage have been described, however more research is needed to determine the efficacy of this method.<sup>6</sup>

Salvage procedures are recommended when joint disease is severe. These include decreasing the load off the medial compartment of the elbow, elbow arthroplasty, elbow arthrodesis, and elbow denervation, all in conjunction with the use of anti-inflammatories for

pain control. Decreasing the load off the medial compartment can be achieved by either a sliding humeral osteotomy or a proximal abducting ulnar osteotomy. In the sliding humeral osteotomy the weight is transferred to the lateral side of the elbow by transecting midhumerus and moving the distal portion of the humerus medially. A locking plate is then used for fixation. One study evaluating this approach found that 65% of dogs were sound post operatively, but long term outcome was not reported. The proximal abducting ulnar osteotomy is suspected to remove the load from the medial compartment by creating new load bearing surfaces on the humerus and ulna, but there is little clinical data to support this. Elbow arthroplasty or total elbow replacement is a surgical option when end stage disease is present. It is not commonly used due to surgical complexity, high complication rate, and cost. Patient selection is very important in a total elbow replacement. Patients should not have lameness associated with other joints on the limb undergoing the procedure as well as three other healthy limbs.<sup>13</sup> Elbow arthrodesis results in severe functional lameness by fusing the elbow joint. It should only be used as a last resort with specific case selection.<sup>10</sup> Zamprognio et al. attempted a procedure to transect the nerves supplying the elbow joint and subsequently decrease pain associated with elbow dysplasia. They were successful in transecting the appropriate nerves however, the effects on osteoarthritis pain were unclear.<sup>14</sup> Overall prognosis with elbow dysplasia is better when early diagnosis and treatment delay the progression of disease. Arthroscopy should be considered first when available and lifelong joint management should be considered in all patients.<sup>13</sup>

**Case outcome:**

Following discussion of surgical options, Cooper was sedated (5 mcg/kg of dexmedtomidine and 0.2mg/kg of butorphanol) and right elbow radiographs were performed.

Radiographs revealed sclerosis along the proximal ulna, a fragmented medial coronoid process and grade 1 arthritis along the caudal portion of the anconeal process.

To further evaluate both elbow joints, computed tomography of both elbows was performed. Computed tomography revealed fragmentation of the medial coronoid process of the right elbow and a small fissure line in the medial coronoid process of the left elbow. Although pathology was found in the left elbow as well as the right, the decision was made to surgically treat the right elbow only, as Cooper was not yet displaying signs of lameness in the left forelimb. Preanesthetic blood work was performed revealing no significant abnormalities and surgery was planned for the following day.

On November 14<sup>th</sup>, 2017, Cooper underwent general anesthesia, brachial plexus nerve block, and right elbow arthroscopy. Mild chondromalacia was noted on the medial condyle of the humerus, as well as a large fragmented medial coronoid process. The fragment was determined to be too large to remove through the scope, thus the decision to perform an arthrotomy was made. A 4 cm curvilinear incision coursing from the medial epicondyle of the humerus to the proximal aspect of the medial ulna was made. The musculature of the elbow was then separated using blunt dissection and retracted to improve visualization. The medial collateral ligament of the elbow was also retracted. The joint capsule was excised to meet the size of the skin incision, and the radius and ulna were pronated to provide adequate visualization of the cranial medial aspect of the elbow joint. The fragmented coronoid process was removed using ronguers, hemostats, and arthroscopic forceps. The joint was probed to check for any stray bone or cartilage and then thoroughly flushed with sterile saline. Each layer was closed separately and the incisions were covered with a suresite bandage. Recovery from surgery and anesthesia was uneventful. Cooper was managed on hydromorphone and NSAIDS for post-operative pain in

ICU. He was having difficulty walking the next morning which is likely due to neuropraxia from the nerve block. At the time of discharge he was bearing full weight and had a moderate lameness which is expected after surgery. He was sent home with specific rehabilitation instructions to slowly increase activity over the next 4 weeks and to return for a recheck in one month. Cooper was lost to follow up however, his owner reported that he appeared to be less lame at home one week after surgery.

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