

ELBOW DYSPLASIA

Daniel D. Lewis, DVM, Diplomate ACVS
Professor Small Animal Surgery
Department of Small Animal Clinical Sciences
University of Florida
Gainesville, Florida

The term "*elbow dysplasia*" has been coined to describe three developmental conditions, osteochondrosis/ osteochondritis dissecans (OC/OCD) of the humeral condyle, ununited anconeal process (UAP), and fragmented coronoid process (FCP), that commonly affect the elbow of many large and giant breeds of dogs. All three conditions share many clinical similarities. Lesions are often bilateral and multiple disease processes can occur concurrently in the same dogs as well as in the same joint. Most commonly osteochondrosis/ osteochondritis dissecans and fragmented coronoid process are recognized concurrently.

The history, signalment, and clinical signs are similar with all three conditions. Clinical abnormalities result from acute joint inflammation and progressive degenerative joint disease of the elbow. Affected animals may exhibit lameness as early as four months of age. The lameness is intermittent and may be exacerbated by exercise or when the dog first ambulates following prolonged rest. Affected dogs may stand or sit with the carpus and paw held in an externally rotational position and with the elbow abducted. The dog may circumduct the antebrachium during the swing phase of the stride. Full flexion and particularly extension of the elbow may elicit a pain response. In dogs with ununited anconeal process it has been stated that a pain response also can be elicited by applying direct pressure to the anconeal process through the anconeus muscle as the dog's elbow is held in flexion. Crepitus, joint capsule thickening, and decreased range of motion are present in advanced cases. Although synovial effusion is considered a classic sign of ununited anconeal process, synovial effusion can also be present with osteochondrosis of the humeral condyle and fragmented coronoid process.

These conditions can be differentiated by high quality craniocaudal and lateral radiographic views of the elbow joint. Additional oblique and flexed lateral views of the elbow may also be warranted. In all three conditions the prognosis for return to working or athletic performance, irrespective of whether or not surgical intervention has been done, is guarded. This guarded prognosis is based on the progression of degenerative joint disease with all three of these conditions irrespective of whether or not surgery is performed. In addition, an exploratory arthrotomy is not a benign procedure, especially if the collateral ligaments are incised, or an osteotomy of the medial epicondyle is performed. Although the apparent recovery following surgery is rapid, some morbidity is expected. Also, elbow function is dependent on a normal anatomic relationship of the radioulnar, humeroulnar and radiohumeral articulations and abnormal elbow function is believed to be an integral component in the pathogenesis of these diseases.

The heritability of fragmented coronoid process and osteochondrosis of the humeral condyle are well established and a heritable basis is also suspected in ununited anconeal process. Thus, consideration should be given to neutering affected dogs thus eliminating them from breeding programs.

Ununited Anconeal Process

Ununited anconeal process occurs primarily in large and giant-breed dogs, most notably German shepherds. The condition is also recognized in chondrodystrophic breeds such as Basset hounds and Bulldogs, as a result of retarded ulnar growth resulting in elbow incongruency. Male dogs are affected approximately twice as frequently as female dogs. The condition is bilateral in 11-30% of affected dogs.

Diagnosis of ununited anconeal process is confirmed by a flexed lateral radiograph of the elbow. In breeds that are predisposed to ununited anconeal process, the anconeal process develops as a distinct center of ossification separate from the ulna. In German shepherd dogs, the anconeal process begins to mineralize at approximately 12 weeks of age and fuses with the ulna at 16 to 24 weeks of age. A definitive diagnosis of ununited anconeal process thus should not be made before 24 weeks of age. Congruency of the elbow must be assessed as many chondrodystrophic and non-chondrodystrophic dogs with ununited anconeal process have proximal humeroradial subluxation. Secondary degenerative changes of the elbow may be present. Dogs with ununited anconeal process typically develop an intermittent, subtle to severe lameness of gradual onset between five and nine months of age. It should be noted, however, that ununited anconeal process can be a serendipitous radiographic finding in mature and middle-aged dogs with no prior history of forelimb lameness or can be a cause of acute forelimb lameness in mature or middle-aged dogs which may not have had any previous history of lameness.

Excision of the ununited anconeal process is still the most widely accepted treatment for this condition. If the anconeal process is to be excised, it is removed via a caudolateral approach to the elbow. The anconeal process is generally not freely moveable because of numerous fibrous adhesions. These adhesions must be broken down with a periosteal elevator to remove the anconeal process. Following surgery the limb is placed in a soft padded bandage for several days to limit swelling. Exercise is restricted for 21 days; however, passive flexion and extension of the elbow should be performed during this time.

Although the prognosis for working and athletic dogs with ununited anconeal process is guarded, many dogs obtain surprising good limb function following process excision. A recent retrospective study evaluated the long-term (mean follow-up 65 months following surgery) clinical results in six dogs which had ununited anconeal processes (one bilateral) excised between five and 12 months of age. Although degenerative joint disease progressed and range of motion was decreased in all affected elbows, limb use was considered good to excellent in six of the seven operated limbs. Others have reported similar results, and we have had dogs with ununited anconeal process return to hunting, obedience and field trial work after process excision. Some lameness after vigorous exercise, however, should be expected.

Stabilization of the ununited anconeal process utilizing lag screw fixation have been suggested, but this procedure has met with technical difficulties and complications and long-term results establishing the efficacy of lag screw fixation are lacking. One report described radiographic union of six of 10 ununited anconeal processes stabilized by lag screw fixation. Detail information describing limb function in these dogs, unfortunately, was not available.

Recent reports describe union of the ununited anconeal process with the olecranon following proximal diaphyseal ulnar osteotomy in young dogs with proximal humeroradial subluxation. The proximal diaphyseal ulnar osteotomy is performed to improve elbow joint congruency, and relieve pressure on the anconeal process. The anconeal process fuses to the ulna in a slightly abnormal position. The long-term functional results associated with proximal diaphyseal ulnar osteotomy appear to be superior in comparison to process excision. The value of performing osteotomies to improve joint congruency at the time of anconeal process excision in dogs with proximal humeroradial subluxation has not been established. Empirically it would seem advantageous to restore normal joint congruency, particular in dogs with marked subluxation; however, it is not known whether improving joint congruency results in improved limb function any more than fragment excision alone.

Osteochondrosis of the Humeral Condyle

In our practice we recognize dogs with osteochondrosis/osteochondritis dissecans of the humeral condyle infrequently in comparison to dogs with fragmented coronoid process and ununited anconeal process. Reports from Europe would suggest that osteochondrosis/osteochondritis dissecans of the humeral condyle occurs more frequently. This may reflect genetic differences in the populations. While osteochondrosis/osteochondritis dissecans of the humeral condyle occurs in many large and giant breeds of dogs, Golden and Labrador retrievers seem particularly prone to develop this condition.

Osteochondrosis is a developmental orthopedic condition characterized by a disturbance in the normal process of endochondral ossification. Endochondral ossification is the process responsible for long bone growth and involves the orderly formation of bone from cartilage. In the pathogenesis of osteochondrosis the normal process of cartilage resorption and subsequent calcification process is disrupted and affected articular or physeal cartilage becomes grossly thickened. Cartilage, which is avascular, is dependent on diffusion of synovial fluid for its metabolic needs. Chondrocytes in the deeper zones of abnormally thickened cartilage are deprived of nutritional support because of the increased distance synovial fluid must diffuse. The result is abnormal chondrocyte metabolism and dysfunction. Cartilage in these deeper layers may become necrotic and develop cracks and fissures. If a crack or fissure extends to the surface of the cartilage, synovial fluid dissects beneath the cartilage flap and debris and inflammatory mediators are released from the necrotic cartilage resulting in inflammation of the synovial tissues. When a cartilage flap or osteochondral fragment are present the condition is more appropriately described as osteochondritis dissecans.

The presence of an articular cartilage flap is the classic lesion of osteochondritis dissecans. The inflammation associated with osteochondritis dissecans lesions produces observable clinical signs such as pain and lameness. As the cartilage flap or osteochondral fragment continues to separate from the subchondral bone a number of different sequelae can develop. Cartilage flaps may remain attached and calcify causing lameness and osteoarthritis. Cartilage flaps and osteochondral fragments often give rise to a superficial erosive (“kissing”) lesion of the apposing articular surface. Cartilage flaps may detach and be resorbed or develop into an attached or free floating “joint mice”. The remaining articular cartilage defect will eventually fill in with a fibrous repair tissue resembling fibrocartilage.

Osteochondrosis is thought to have a multifactorial etiology. Genetic factors that affect weight gain and growth, behavior, sexual development and conformation undoubtedly are involved in the etiology of osteochondrosis. Osteochondrosis occurs primarily in medium to large and giant breed dogs. Rapidly growing representatives of these breeds seem to be predisposed. The genetic capacity for rapid growth and overfeeding may be influential during the dog's period of rapid growth. This is an important aspect in athletic dogs as often it is the largest, most rapidly growing dog that is desired. In addition the selective breeding of performance animals may be based on performance data, conformation, and other desired qualities without knowledge of the presence of articular disease that may be subclinical in nature or affected littermates.

Biomechanical forces are another important etiologic factor in the development of osteochondrosis. Osteochondrosis lesions develop in areas of articular cartilage that are subject to increased loads. Normal joint stresses and focal trauma likely are inciting perpetuating factors involved in the pathogenesis of this condition. The strenuous activity often imposed on performance dogs at very young ages may partially account for the high incidence of osteochondrosis in many breeds of working and racing dogs.

Overnutrition in the form of excessive amounts of food and/or nutritional supplements has been incriminated as another important etiologic factor. This is evidenced by higher caloric intake in rapidly growing animals resulting in greater incidence of osteochondrosis. Excessive calcium supplementation has been shown to increase the development of osteochondrosis. Hormonal disturbances have also been incriminated through experimental production of osteochondrotic lesions following the administration of thyrotropin, growth hormone, estrogens and androgens. All of the aforementioned etiologies may enter into the development of osteochondrosis as breeders, trainers and owners search for the combination of the biggest, fastest, best looking representatives of a breed, not infrequently placing the individual dog or breed at risk.

Osteochondrosis/osteochondritis dissecans lesions of the humeral condyle can be observed on the craniocaudal view radiograph of the elbow as a subchondral bone defect that affects the trochlea (the medial portion) of the humeral condyle. These lesions can be subtle and may not be identified unless the radiographs are of good quality and evaluated carefully. The pronated oblique, craniocaudal view is often of value in identifying lesions that may not be apparent on nonoblique craniocaudal view radiographs. If the lesion is large, an irregular subchondral bone defect or flattening of the articular surface of the medial condyle may be visible on the lateral view radiograph. Secondary, degenerative changes are usually present in dogs seven months of age and older. As previously stated osteochondrosis/osteochondritis dissecans of the humeral condyle can and often occurs concurrently with fragmented coronoid process. True osteochondrosis/osteochondritis dissecans lesions of the humeral condyle can sometimes be difficult to distinguish radiographically from erosive lesions of the trochlea of the humeral condyle induced by fragmented coronoid process particularly in older dogs.

Treatment involves excision of the cartilage flap and curettage of the subjacent subchondral bed. Some veterinary surgeons are now treating these lesions with arthroscopy. Arthroscopy allows a greater extent of the joint to be evaluated with the advantage of magnification. While the immediate postoperative morbidity is less with arthroscopy than arthrotomy, the long-term functional results are probably similar.

Several surgical approaches have been described for approaching the medial compartment of the elbow. A recent study performed using cadaver limbs evaluated articular cartilage exposure and immediate postoperative stability afforded by three described approaches to the medial compartment of the elbow, an osteotomy of the medial epicondyle, a longitudinal myotomy of the flexor carpi radialis muscle, and a desmotomy of the medial collateral ligament which included a tenotomy of the pronator teres muscle. The approach using an osteotomy of the medial epicondyle provided significantly greater exposures of the humeral articular cartilage (22%) than either of the other two approaches and the approach using a desmotomy of the medial collateral ligament provided significantly greater exposure of the humeral articular cartilage (16.5%) than the approach using a longitudinal myotomy of the flexor carpi radialis muscle (6.6%). The immediate postoperative stability of the approach using an osteotomy of the medial epicondyle and the approach using a longitudinal myotomy of the flexor carpi radialis muscle were significantly greater than that of the approach using a desmotomy of the medial collateral ligament. It must be noted, however, that testing of the limb was performed with both the elbow and carpus in 90° of flexion to accentuate the soft tissue contributions to valgus stability of the elbow. At lesser angles of elbow flexion, interlocking of the anconeal process in the trochlea and olecranon fossa provided the valgus stability of the elbow. This locking mechanism of the anconeal process in the trochlea and olecranon fossa probably negates much of the morbidity of any surgical approach to the medial compartment of the elbow and accounts for the lack of reported complications associated with the use of the medial desmotomies in clinical cases.

We and others have experienced implant complications in clinical cases in which we used an osteotomy of the medial epicondyle to approach the medial compartment of the elbow and feel exposure of the trochlea of the humeral condyle is sufficient to excise cartilage flaps and curette the lesion's bed using a muscle separating approach between the flexor carpi radialis muscle and the pronator teres muscle. In some instances exposure is sufficient by retracting the medial collateral ligament but in many instances a medial desmotomy is required. A myotenyotomy of the pronator teres muscle can be performed if additional exposure is required; however, this is seldom necessary and should be avoided in performance dogs.

Osteochondritis desiccans lesions of the humeral condyle may not always be obvious at surgery. If a lesion is suspected based on the pre-operative radiographs and is not readily apparent at arthrotomy, the articular surface of the trochlea of the humeral condyle should be probed with a Freer periosteal elevator. In these instances the malacic cartilage will readily separate from the adjacent unaffected cartilage. All diseased cartilage should be excised and the subchondral bed curettaged. Osteochondritis dissecans lesions of the humeral condyle should be differentiated from erosive or "kissing" lesions of the trochlea of the humeral condyle which frequently occur in response to fragmented coronoid process.

Postoperatively the limb is placed in a soft padded bandage for 2 to 4 days following surgery to limit swelling and exercise is restricted for 3-4 weeks. The prognosis for return to function is again somewhat guarded for dogs with osteochondrosis of the humeral condyle. The prognosis seems to be somewhat dependent on the size of the lesion and the extent of degenerative joint disease present at the time of surgery. Young dogs with small lesions and minimal degenerative joint disease are stated to have a more favorable prognosis than older dogs with larger lesions and more advanced degenerative joint disease. Although degenerative joint disease progresses irrespective of surgical intervention, some dogs may have acceptable limb function to return to hunting and other working activities.

Fragmented Coronoid Process

Fragmented coronoid process is the third developmental condition that affects the elbows of large and giant breed dogs, particularly retrievers, Rottweilers, mastiffs, Bernese Mountain dogs, and German shepherd dogs. The etiopathogenesis of this condition is controversial. Fragmented coronoid process was initially believed to be a manifestation of the osteochondrosis complex; however, pathoanatomic studies have not fully supported this contention. Fissures or fragmentation may result from abnormal stresses placed on the developing coronoid process secondary to conformational abnormalities of the elbow. The medial coronoid process is most often involved. The disease is more common in male dogs and is often bilateral. Injury to the distal radial physis in skeletally immature dogs may also result in distal radiohumeral subluxation as the ulna continues to grow with subsequent fragmentation of the coronoid process.

Clinical signs are rarely noted before five months of age. Subtle weight-bearing lameness, exacerbated by prolonged rest or exercise, is typical. The onset of lameness is insidious. As lameness persists, it may increase in severity. Affected dogs often place the carpus in an exaggerated valgus position when sitting or standing and circumduct the antebrachium during the swing phase of the stride. A pain response is usually not elicited unless the elbow is fully extended. Some investigators suggest that the carpus should be placed in a flexed, externally rotated position while the elbow is extended. Joint effusion may be detected as a fluctuant swelling beneath the lateral epicondyle of the humerus.

The fragmented coronoid process is rarely identified radiographically because of superimposition of the medial coronoid process and the head of the radius. The mediolateral (extended and supinated) view made with the elbow maximally extended and supinated 15° reportedly is a superior radiographic projection for demonstrating pathology of the medial coronoid process; however, we have not found this view to be useful for specific identification of coronoid pathology. We have found the pronated oblique, craniocaudal view to be more useful for specific identification of medial coronoid pathology, but the percentage of cases in which a specific fragment can be identified is still limited.

When fragmentation of the coronoid process can not be identified radiographically, a clinical diagnosis of fragmented coronoid process is supported by the presence of degenerative changes in the elbow in the absence of an ununited anconeal process or osteochondrosis of the humeral condyle. Osteophyte development on the anconeal process and increased density of the ulna subjacent to the coronoid process and the trochlear notch are early radiographic degenerative changes associated with fragmented coronoid process. Degenerative changes are usually not radiographically evident before seven months of age. Distal humeroradial subluxation, a purported etiology for fragmentation of the coronoid process, may be apparent in some dogs before degenerative changes are apparent.

Definitive diagnosis of fragmented medial coronoid process is made at exploratory arthrotomy or via arthroscopy. Again some veterinary surgeons are successful diagnosing and removing fragmented coronoid processes using arthroscopy. If a standard surgical arthrotomy is done, the medial coronoid process is exposed via the previously described muscle separating approach. Experience and adequate light facilitate definition of the pathology. In some dogs, a free fragment exists and is readily identified and removed. In other dogs, only fissuring of the articular cartilage is present. These fissures can extend variable depths into the subchondral bone. These lesions can be difficult to recognize and confusing. Probing the articular surface of the medial coronoid process with a Freer periosteal elevator can help identify fissures that are not readily apparent. Although optimal treatment of these fissures has not been determined, we generally excise the affected region as if it were a fragment.

Postoperative care depends upon the approach that was used at surgery. A soft padded bandage is applied to the affected limb for three to five days following surgery. If an osteotomy of the medial condyle or a desmotomy of the medial collateral ligament was performed, strict kennel confinement and limited activity on a leash is recommended for 4 to 6 weeks. If the medial collateral ligament was simply retracted and an osteotomy or desmotomy was not performed, 2-3 weeks of confinement and leash activity is sufficient. Gradual return to activity is recommended over the subsequent 4 weeks.

The prognosis for dogs with fragmented coronoid process is again guarded. The benefits and efficacy of surgery, and for that matter arthroscopy, for dogs with fragmented coronoid process is a contentious issue. Although degenerative joint disease progresses irrespective of whether or not surgical intervention is done, most dogs will eventually become sound with a slightly stiff, stilted gait. Although studies disagree regarding the benefits of surgery, we feel that surgical excision of fragmented coronoid process in most young dogs results in a more rapid improvement of lameness. We rarely, however, perform surgery on dogs older than 18 months. Many of these dogs can return hunting, obedience and field trail work; however, lameness should be expected with vigorous activity.

REFERENCES:

1. Wind A. Elbow incongruity and developmental elbow diseases in the dog: Part I. *J Am Anim Hosp Assoc* 1986;22:711 -724.
2. Fox SM, Bloomberg MS, Bright RM. Developmental anomalies of the canine elbow. *J Am Anim Hosp Assoc* 1983;19:605-614.
3. Grondalen J. Arthrosis with special reference to the elbow joint of rapidly growing dogs. II. Occurrence, clinical and radiographic findings. *Nord Vet Med* 1979;31:69-75.
4. Henry WB. Radiographic diagnosis and surgical management of fragmented medial coronoid process in dogs. *J Am Vet Med Assoc* 1984;184:799-805.
5. Berzon JL, Quick CB. Fragmented coronoid process: Anatomical, clinical and radiographic considerations with case analysis. *J Am Anim Hosp Assoc* 1980; 16 :241 -251.
6. Bennett D, Duff SRI, Kene RO, et al. Osteochondritis dissecans and fragmentation of the coronoid process in the elbow joint of the dog. *Vet Rec* 1981;109:329-336.

7. Voorhout G, Hazewinkle HAW. Radiographic evaluation of the canine elbow joint with specific reference to the medial humeral condyle and the medial coronoid process. *Vet Radiol* 1987;28:158-165.
8. Denny HR, Gibbs C. The surgical treatment of osteochondritis dissecans and ununited coronoid process in the canine elbow joint. *J Small Anim Pract* 1980;21: 595-608.
9. Olsson S-E. The early diagnosis of fragmented coronoid process and osteochondritis dissecans of the canine elbow. *J Am Anim Hosp Assoc* 1983;19:616-626.
10. Lewis DD, Parker RB, Hager DA. Fragmented medial coronoid process of the canine elbow. *Compend Contin Educ Pract Vet (Small Anim)* 1989;11:703-715.
11. Grondalen J, Lingaas F. Arthrosis in the elbow joint of young, rapidly growing dogs: A genetic investigation. *J Small Anim Pract* 1991;32:460-464.
12. Grondalen J. Arthrosis in the elbow joint of young rapidly growing dogs. II. Occurrence in the Rottweiler breed. *Nordisk Veterinaermedicin* 1982;34:76-82.
13. Tirgari M. Clinical, radiographical and pathological aspects of ununited medial coronoid process of the elbow joint in dogs. *J Small Anim Pract* 1980;21:595-608.
14. Wind AP. Incidence and radiographic appearance of fragmented coronoid process. *California Vet* 1982;6:19-25.
15. Grondalen J. Arthrosis with special reference to the elbow joint of rapidly growing dogs. III. Ununited medial coronoid process of the ulna and osteochondritis dissecans of the humeral condyle. Surgical procedure for correction and postoperative investigation. *Nord Vet Med* 1979;31:520-527.
16. Mason T, Lavelle R, Skipper S, et al. Osteochondrosis of the elbow joint in young dogs. *J Small Anim Pract* 1980;21:641-656.
17. Olsson S-E. Osteochondrosis - a growing problem to dog breeders. *Gaines Progress* 1976; 1-11.
18. Olsson S-E. Osteochondrosis in the dog. In: Kirk RW (ed). *Current Veterinary Therapy VI*. Philadelphia, WB Saunders Co, 1977, pp 880-886.
19. Olsson S-E. Lameness in the dog: A review of lesions causing osteoarthritis of the shoulder, elbow, hip, stifle and hock joints. *J Am Anim Hosp Assoc* 1975;1:363.
20. Grondalen J, Grondalen T. Arthrosis in the elbow joint of young rapidly growing dogs. V. A pathoanatomical investigation. *Nord Vet Med* 1981;33:1-16.
21. Macpherson GC, Lewis DD, Johnson KA, et al. Fragmented coronoid process associated with premature distal radial physeal closure in four dogs. *VCOT* 1992;5:93-99.

22. Wind AP, Packard ME. Elbow incongruity and developmental elbow diseases in the dog: Part II. *J Am Anim Hosp Assoc* 1986;22:724.
23. Wind AP. Elbow dysplasia. In: Slatter D (ed). *Textbook of Small Animal Surgery*. Philadelphia, WB Saunders Co, 1993, pp 1966-77.
24. Suess RP, Trotter EJ, Konieczynski D, et al. Exposure and postoperative stability of three medial surgical approaches to the canine elbow. *Vet Surg* 1994;23:87-93.
25. Boudrieau RJ, Hohn RB, Bardet JF. Osteochondritis dissecans of the elbow in the dog. *J Am Anim Hosp Assoc* 1983;19:627-635.
26. Studdert VP, Lavelle RB, Beilharz RG, et al. Clinical features and heritability of osteochondrosis of the elbow in labrador retrievers. *J Small Anim Pract* 1991;32:557-563.
27. Guthrie S. Use of radiographic scoring technique for the assessment of dogs with elbow osteochondrosis. *J Small Anim Pract* 1989;30:639-644.
28. Wissler J, Sumner-Smith G. Osteochondrosis in the elbow joint in the dog. *J Am Anim Hosp Assoc* 1977;13:349-354.
29. Berzon JL. Diagnosis and surgical management of osteochondrosis dissecans of the elbow joint. *Compend Contin Educ Pract Vet* 1983;5:285-288.
30. Sinibaldi KR, Arnoczky SP. Surgical removal of the ununited anconeal process in the dog. *J Am Anim Hosp Assoc* 1975;11:192-198.
31. Grøndalen J, Rørvik AM. Arthrosis in the elbow joint of young rapidly growing dogs. IV. Ununited anconeal process. *Nord Vet Med* 1980;32:212-218.
32. Roy RG, Wallace LJ, Johnston GR. A retrospective long-term evaluation of ununited anconeal process excision on the canine elbow. *Vet Compar Ortho Trauma* 1994;7:94-97.
33. Pritchard DL. Anconeal process pseudoarthrosis: Treated by lag-screw fixation. *Canine Pract* 1976;18-23.
34. Guthrie S. Some radiographic and clinical aspects of ununited anconeal process. *Vet Record* 1989;124:662-663.
35. Olsson S-E. Pathophysiology, morphology and clinical signs associated with osteochondrosis in the dog. In: Bojrab MJ (ed). *Disease Mechanisms in Small Animal Surgery*, ed 3. Philadelphia, Lea & Febiger, 1993, pp 777-796.
36. Hazewinkel HAW, Kantor A, Meij B. Loose anconeal process. *Tijdschrift voor Diergeneeslevnde* 1988;113 (Suppl 1):57-59.
37. Herron MR. Ununited anconeal process - a new approach to surgical repair. *Mod Vet Pract* 1970;51:30-34.

38. Guthrie S, Plummer JM, Vaughan LC. Aetiopathogenesis of canine elbow osteochondrosis: A study of loose fragments removed at arthrotomy. *Res Vet Sci* 1992;52:284-291.
39. Read RA. Arthrosis in young dogs: Incidence, diagnosis and management. *Waltham Internation Focus* 1993;3(2):2-10.
40. Lenehan TM, van Sickle DC. Canine osteochondrosis. In: Newton CD, Nunamaker CM. Textbook of Small Animal Orthopedics. Philadelphia, JB Lippincott Co, 1985, pp 981-997.
41. Lenehan TM, van Sickle DC. Ununited anconeal process, ununited medial coronoid process, ununited medial epicondyle, patella cubiti, and sesamoidal fragments of the elbow. In: Newton CD, Nunamaker CM. Textbook of Small Animal Orthopedics. Philadelphia, JB Lippincott Co, 1985, pp 999-1012.
42. Cawley AJ, Archibald J. Ununited anconeal process of the dog. *J Am Vet Med Assoc* 1959;134:454-458.
43. Van Sickle DC. The relationship of ossification to canine elbow dysplasia. *J Am Anim Hosp Assoc* 1966;2:24-31.
44. Probst CW, Johnston SA. Osteochondrosis. In: Slatter DH (ed). Textbook of Small Animal Surgery. Vol II, 2nd ed, Philadelphia, WB Saunders, 1993, pp 1944-1966.
45. Guthrie S, Pidduck HG. Heritability of elbow osteochondrosis within a closed population of dogs. *J Small Anim Pract* 1990;31:93-96.
46. Tobias TA, Miyabayashi T, Olmstead ML, et al. Surgical removal of fragmented medial coronoid process in the dog: Comparative effects of surgical approach and age at time of surgery. *J Am Anim Hosp Assoc* 1994;30:360-368.
47. Read RA, Armstrong SJ, O'Keef D, et al. Fragmentation of the medial coronoid process of the ulna in dogs: A study of 109 cases. *J Small Anim Pract* 1990;31:330-334.
48. Huibregtse BA, Johnson AL, Muhlbauer MC, et al. The effect of treatment of fragmented coronoid process on the development of osteoarthritis of the elbow. *J Am Anim Hosp* 1994;30:190-195.
49. Bouck GR, Miller CW, Taves CL. A comparison of surgical and medical treatment of fragmented coronoid process and osteochondritis dissecans of the canine elbow. *Vet Compar Orthor Trauma* 1995;8:177-183.
50. Fox SM, Burbidge HM, Bray JC, et al. Ununited anconeal process: Lag screw fixation. *J Am Anim Hosp Assoc* 1996;32:52-56.
51. Arbesser E. Osteochondrosis dissecans der femor-kondylen beim hund. *Wien Tierarztl Monatschr* 1974;61:303-313.
52. Ponzet G, Walde I, Arbesser E. Zur osteochondrosis dissecans genu des hundes. *Kleintier-Praxis* 1975;20:88-98.

53. Losonsky JM, Kneller SK. Misdiagnosis in normal radiographic anatomy: Eight structural configurations simulating disease entities in dogs and cats. *J Am Vet Med Assoc* 1987; 191:109-114.
54. Lewis DD, McCarthy RJ, Pechman RD. Diagnosis of common development orthopedic conditions in canine pediatric patients. *Compend Contin Educ Pract Vet (Small Anim)* 1992;14:287-301.
55. Hazewinkel HAW, Goedegebuure SA, Poulos PW, et al. Influences of chronic calcium excess on the skeletal development of growing Great Danes. *J Am Anim Hosp Assoc* 1985;21:337-391.
56. Guthrie S, Plummer JM, Vaughan LC. Post natal development of the canine elbow joint: A light and electron microscopical study. *Res Vet Sci* 1992;52:67-71.
57. Houlton JEF. Osteochondrosis of the shoulder and elbow joints in dogs. *J Small Anim Pract* 1984;25:399-413.
58. Olson NC, Brinker WO, Carrig CB, et al. Asynchronous growth of the canine radius and ulna: Surgical correction following experimental premature closure of the distal radial physis. *Vet Surg* 1981;10:125-131.
59. Vandewater A, Olmstead ML. Premature closure of the distal radial physis in the dog. A review of eleven cases. *Vet Surg* 1983;12:7-12.
60. Poulos PW. Canine osteochondrosis. *Vet Clin N Am (Small Anim Pract)* 1982;12:313-328.
61. Hayes HM, Selby LA, Wilson GP, et al. Epidemiologic observations of canine elbow disease (emphasis on dysplasia). *J Am Anim Hosp Assoc* 1979;15:449-453.
62. Grøndalen J, Lingaas F. Arthrosis in the elbow joint of young rapidly growing dogs: A genetic investigation. *J Small Anim Pract* 1991;32:460-464.
63. Studdert VP, Lavelle RB, Beilharz RG, et al. Clinical features and heritability of osteochondrosis of the elbow in labrador retrievers. *J Small Anim Pract* 1991;32:557-563.
64. Hazewinkel HAW. Nutrition in relation to skeletal growth deformities. *J Small Anim Pract* 1989;30:625-630.
65. Carlson CS, Hilley HD, Meuten DJ, et al. Effect of reduced growth rate on the prevalence and severity of osteochondrosis in gilts. *Am J Vet Res* 1988;49:396-402.
66. Grøndalen T. Osteochondrosis and arthrosis in pigs. VI. Relationship to feed level and calcium, phosphorus and protein levels in the ration. *Acta Vet Scand* 1974;15:147-169.
67. Slater MR, Scarlett JM, Kaderly RE, et al. Breed, gender, and age as risk factors for canine osteochondritis dissecans. *VCOT* 1991;4:100-106.
68. Sjöström L, Kasström H, Källberg M. Ununited anconeal process in the dog. Pathogenesis and treatment by osteotomy of the ulna. *VCOT* 1995;8:170-176.

