

Canine Osteochondrosis

Chapter 84

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Within the last several years, a developmental disease process termed osteochondrosis has been diagnosed with increasing frequency both in the human and veterinary medical fields. A generalized metabolic disease process disrupting the normal sequence of cartilage calcification and eventual ossification within endochondral growth areas, osteochondrosis has become a problem of worldwide economic significance in both the poultry and swine industries. It has likewise become a major problem in many breeds of dogs and has been described in horses and cattle. Osteochondrosis can affect multiple joints and metaphyseal growth areas within a single animal, with the clinical symptomatology related directly to the location and degree of affection.

Mechanisms of Normal Epiphyseal and Metaphyseal Growth

While both the neonate and the adult are capable of forming bone directly from a loose form of connective tissue without an intervening stage of cartilage formation (i.e., by a process termed intramembranous ossification), such a phenomenon is generally reserved for the formation of the flat bones of the body (e.g., those of the skull and face) and for the modification of the structure of preexistent bone tissue (remodeling). In embryonic life, most of the fetal skeleton is comprised of cartilage, which is destined eventually to become resorbed and replaced by bone through a process termed endochondral ossification. Its normal sequence of maturation generally includes the transformation of a cartilaginous anlage into a structure comprised of one or multiple proximal and distal secondary centers of ossification (epiphyseal) united by a single primary center of ossification (diaphyseal). Ossification continues to incorporate eventually the

entire diaphysis and epiphysis, the centers ultimately contacting each other in the region of the cartilaginous metaphyseal growth plates (physes). This phenomenon begins prenatally and continues throughout the postnatal period until growth is complete, at which time the metaphyseal

growth plate disappears, bone ceases to grow, and a continuous bone marrow cavity traverses the entire length of the bone. Thus, early in neonatal life a great majority of the long bones of the dog include three major centers of ossification (two epiphyseal and one diaphyseal) with a few secondary epiphyseal centers forming specific articulating structures or major bone prominences of tendon insertion (e.g., anconeal process of ulna, medial epicondyle of humerus, and traction apophyses of ulnar olecranon and tibial tuberosity). The epiphyseal centers of ossification and growth ultimately regulate the growth in length and breadth of the bony epiphysis and determine the form it takes in relation to the opposing joint surface with which it is in intimate contact; the metaphyseal centers of ossification and growth are responsible for metaphyseal funnelization and an increase in length of the diaphyseal portion of the bone. The process of endochondral ossification cannot occur, however, without the orderly production of a cartilage model upon which to develop, that is, without a normal growth zone of the diaphysis (metaphyseal growth plate) and growth zone of the epiphysis (joint cartilage of the immature animal). (72)

The articular cartilage in the young animal serves the dual function of both articulating surface and growth zone. It consists of two layers of hyaline cartilage that differ from each other morphologically as well as physiologically; they are often referred to as the articular-epiphyseal complex(59) (Figs. 84-1 and 84-2). The articular layer extends from an eosinophilic line within the cartilage to the joint surface. Chondrocyte organization in this region is similar to that of the adult articular cartilage, consisting of superficial (tangential), middle, and deep (radiate) layers. Chondrocytes here are smaller than those found on the epiphyseal layer, and there are no cartilage canals. This articular cartilage stains more positively for glycosaminoglycans, while the deep eosinophilic line stains positive for collagen with histochemistry. In contrast, the epiphyseal layer of juvenile articular cartilage contains chondrocytes that are larger and more randomly arranged than those of the articular layer. Moving toward the secondary center of ossification this pattern changes, with regular short columns of hypertrophied chondrocytes undergoing cellular differentiation, cartilage canal formation and endochondral ossification (Fig. 84-2). It is this layer of juvenile articular cartilage that serves as the growth zone of the epiphysis and has as its counterpart the metaphyseal growth plate.

FIG. 84-1 The articular-epiphyseal cartilage complex of the scapula (above) and the humeral head (below) of a 4-month-old canine shoulder. The thicker articular-epiphyseal cartilage of the humeral head is the caudal articular surface, while the thinner articular-epiphyseal complex on the right approaches the dorsal plateau. Note that the light-staining surface layer (articular cartilage) is of uniform thickness, whereas the underlying epiphyseal cartilage varies in

thickness, indicating a variance in ossification. This is a normal occurrence. (H&E x 4.5)

FIG. 84-2 An enlargement of an articular-epiphyseal cartilage complex. The darker matrix in the center is an eosinophilic line (area of calcified cartilage of adult articular cartilage). Note the arrangement of chondrocytes above and below the line, the relative chondrocytic size, and short columns of hypertrophied chondrocytes adjacent to the bone marrow of the secondary center of ossification. (H&E x 387)

In the normal animal the rate of cartilage cell proliferation, maturation, and calcification is in harmony with the concomitant process of chondrocyte death, disintegration, and calcification. In the area of the primary spongiosum, cellular calcified cartilage bars extend from the cartilage portion of the plate into the ossification center. A region of low oxygen tension, it is this area into which the capillary loops penetrate through the transverse noncalcified septa. Osteogenic cells follow to lay down osteoid upon these calcified cartilage bars, with mineralization immediately following. This new fiber (woven) bone forms the secondary spongiosum, interdigitating closely with the primary spongiosum above, and together with it forms the initial supportive bony trabeculae. Disturbance in any one of these steps can result in either joint or metaphyseal deformity or disruption.

Pathophysiology of Osteochondrosis

In osteochondrosis, a disturbance in the normal differentiation of cartilage cells occurs. Early on, the germinative zone of cartilage displays an irregular arrangement of cells that lack nuclei and cleave. Proliferation, differentiation, vesiculation, degeneration, and calcification of the growth cartilage do not take place in the normal fashion.⁽⁵⁴⁾ As a result, the transverse partitions between cells in each column fail to dissolve, preventing the resorption channels from penetrating the cartilage model. The normal continuum between calcifying chondrocytes and continuously developing longitudinal bony trabeculae with cartilage cores (primary and secondary spongiosa) is then disrupted. Endochondral ossification ceases and the cartilage model is retained, with affected areas of the growth plate becoming distinctly thickened ([Fig 84-3](#)). An osteochondritic lesion in the metaphyseal growth plate of a dog may minimally disturb long-bone growth, may moderately affect long-bone growth, resulting in an altered shape or angulation of the affected bone or its closely aligned neighbor, or severely affect it, paving the way for epiphysiolysis (slipped epiphysis). Alternatively, a lesion affecting the epiphyseal layer of juvenile articular cartilage may be clinically insignificant, may be sufficiently severe to result in mild lameness but heal spontaneously, or may manifest itself as the full-blown clinical syndrome termed osteochondritis dissecans.

In osteochondritis dissecans, normal joint stresses, focal trauma, necrosis, or some other unknown mechanism causes cracks and fissures to develop in the zone of hypertrophied chondrocytes of the epiphyseal cartilage layer and extend toward the articular surface (Figs. [84-](#)

[4 through 84-6](#)). Propagation of these fissures with ultimate extension into the joint results in the release of cartilaginous breakdown products into the joint fluid with a resultant synovitis, inflammation of subchondral bone and cartilage (osteochondritis), and creation of a flap of cartilage that further dissects away from its underlying subchondral attachments (dissecans). The cartilaginous flap often gives rise to superficial erosions of the cartilage of the opposing joint surface (kissing lesion), [\(68\)](#) which contributes further to the developing inflammatory arthritis. The dissection of synovial fluid into subchondral bone results in a focal necrosis of the bone and bone marrow under the flap. If the flap is extensive and the dog is of sufficient weight (e.g., Great Dane), a large area of avascular necrosis of the bone and bone marrow results, followed by entrapment of synovial fluid and subsequent cyst formation. [\(16\)](#) If the limb is immobilized (either by a veterinarian or the dog protecting it), vascularity can be reestablished in the fibrotic bone marrow and the flap may reattach to the secondary center of ossification by bony trabeculae derived intramembraneously within the fibrotic bone marrow. Following flap immobilization, epiphyseal chondrocytes adjacent to the vascularized fibrotic bone marrow of the secondary center of ossification begin to hypertrophy, marrow capillaries invade, and endochondral ossification is resumed. The only long-term lesion may be a transverse dimple in the articular surface.

In most dogs, fragmentation and separation at the base of the affected cartilage eventually result in the formation of a free flap, which may be resorbed, [\(49\)](#) remain a free body, fragment, or grow in size, nurtured by the synovial fluid in which it is bathed. Such a free joint mouse may cause lameness either by intermittently becoming interposed between articulating surfaces or by becoming lodged in tendon sheaths confluent with the joint structure in which it has been released (e.g., biceps tendon sheath of shoulder). Alternatively, it may become sequestered (and relatively quiescent clinically) in some outpouching of the joint, developing synovial attachments and possibly becoming ossified.

Osteochondritis dissecans has in the past been thought to result from osteonecrosis or to have originated as an osteochondral fracture, since the free flap or loose body in both humans and horses often contains both bone and cartilage. [\(7,74\)](#) In osteochondritis dissecans of the humeral head, medial humeral condyle, and femoral condyle of the dog, however, ossification of the flap does not occur. [\(49\)](#) While calcification of a free flap is possible, ossification can be seen only occasionally, and then only if the flap has adhered to the synovial membrane and become vascularized. In contrast to osteochondritis dissecans of the above-mentioned sites is osteochondritis dissecans of the medial trochlear ridge of the tibial tarsal bone in the dog, the flap of which is often ossified, as are the flaps occurring in ununited anconeal and coronoid processes. In osteochondritis dissecans, as long as the flap of cartilage remains situated in its bony bed, healing of the osteochondral defect does not occur and lameness persists. Once the flap has become detached from its pedicle and subchondral attachments, the defect becomes filled by an ingrowth of vascular connective tissue arising from the primitive mesenchymal cells of the underlying bone if the subchondral marrow space has been exposed. [\(14\)](#) The fibroblasts

soon differentiate into chondrocytes and eventually establish a new variant of hyaline cartilage. While morphologic studies demonstrate that this cartilaginous tissue can retain its hyaline appearance for at least several months, it has been reported that degenerative lesions and a more fibrous texture develop by one year in surgically created lesions.⁽³²⁾ This fibrocartilage eventually fibrillates, setting the stage for the development of subsequent osteoarthritis.⁽⁴³⁾ It would seem that the rapidity and degree of healing and the eventual development of secondary degenerative joint disease are dependent on many factors, not the least important of which are the location of the original lesion (weight-bearing versus non-weight-bearing area) and the initial size of the defect.⁽¹⁴⁾ At any rate, clinically affected animals exhibit marked improvement in function for prolonged periods of time once the cartilage flap has been removed. Few good clinical studies with long-term radiographic and clinical follow-up have chronicled the eventual fate of affected joints treated either medically or surgically. Osteochondritis dissecans of the shoulder is the notable exception.

FIG. 84-5. (A) Microscopic cartilage fracture at the area between the epiphyseal chondrocytes and the hypertrophying chondrocytes. (B) Magnification of the fracture in (A). Cells appear to be hypertrophying on each side of the cleft. If a complete separation does not occur, the lower area will undergo endochondral ossification followed by re-establishment of another ossification front on the upper cartilage, hence spontaneous healing and re-establishment of endochondral ossification. (H&E x 363)

FIG. 84-6 Fracture extending from area shown in Figure 84-5 to the articular surface. The chondrocytes bordering the fracture have been compressed and are aligned parallel to the cleft. This separation provides a tract for synovial fluid to enter the bone marrow of the secondary center and in the giant breeds results in subchondral cyst formation. (H&E x 363)

Osteochondrosis has been studied closely in many species of animals, particularly the pig, in which osteochondritis dissecans occurs in almost all joints and in which osteochondrosis occurs with a frequency of about 80%.⁽⁴¹⁾ In this species it has been demonstrated experimentally that the incidence and severity of osteochondrosis are related directly to rapidity of growth.⁽⁶⁰⁾ This finding has been reinforced by studies in poultry, while in the dog it is a disease limited to rapidly growing animals of the larger breeds. Among dogs, males have generally been shown to be affected more frequently than females. Nutrition plays a role in the development of osteochondrosis also, with high caloric intake the main factor related to its occurrence in swine⁽⁶⁰⁾ and probably in the dog. There most likely exists a hereditary predisposition for osteochondrosis in the dog also,⁽⁴⁹⁾ a fact not surprising in light of the findings in pigs⁽²⁵⁾ and in horses.⁽⁶¹⁾ Several factors suggest that mechanical forces also play an important role in lesion development, including the predilection of the disease for specific joints and metaphyseal growth plates and the apparent parallelism of epiphyseal and physeal lesions in the pig. In the dog, the *in vivo* motion of the glenoid cavity of the scapula relative to the articular surface of the humeral head as well as the area of contact between the two has been examined.^(35,67) It

was found that the lesion of the humeral head develops in an area located at the interface between contact and noncontact surfaces. It was also postulated that the lesion may result because of differing forces acting at the interface of these two areas. Other workers have proposed similar mechanisms. (16) This difference in contact versus noncontact areas appears to have an effect on the rate of ossification of the proximal humeral secondary center of ossification (the rate of ossification is slower caudally, on the non-weight-bearing surface; and faster laterally, on the weight-bearing surface).(11) A difference in the amounts of chondroitin sulfate (higher in the dorsal plateau, an area of greater usage) and keratin sulfate (higher in the caudal area, an area of little or no usage) in the articular cartilage has also been found.(34) Thus there is evidence that biomechanical forces may play a role in the rate of endochondral ossification; therefore, it is reasonable to predict that if the normal forces are exceeded, pathology will result.

Osteochondrosis affecting the immature joint cartilage (growth zone of the epiphysis) has been proven responsible for osteochondritis dissecans of the shoulder, distal humerus, distal femur, and tibial tarsal bone in the dog; it is believed to affect the distal radius and cervical intervertebral joints and has been implicated in the etiopathogenesis of hip dysplasia.(49) If similar changes take place in a metaphyseal growth zone, the normal shape of the bone may be changed because growth has been disturbed. Disruption of endochondral ossification in the metaphyseal growth zone (physis) due to osteochondrosis is thought to be responsible for such disease conditions as retained endochondral cores of the distal ulna and ununited anconeal process and possibly ununited (fragmented) coronoid process, and has been implicated in the etiopathogenesis of epiphysiolysis of the femoral head in dogs.(50)

While osteochondrosis is a generalized disease process affecting multiple areas of endochondral ossification, its most important clinical expressions seem restricted to certain predisposed areas specific to each species. Its degree of joint specificity and focalization to one small portion of an articular surface remains uncannily reproducible. A systemic problem of variable expression within areas of rapid osteochondral growth and remodeling, its changes may be so subtle as to merely result in an idiopathic osteoarthritis in the joint of a mature animal secondary to intra-articular lesion remodeling or subtle angular limb deformities. Alternatively, it may be as blatant and readily diagnosed as a painful osteochondritic lesion in a major weight-bearing joint of a young animal. The disease manifests itself most readily in areas where growth rate is accentuated (e.g., in the distal ulnar metaphyseal growth plate), where ossification occurs late relative to adjoining structures (anconeal process, coronoid process), and where joint cartilage remains thickened up until an age of 4 to 5 months (caudal humeral head, medial humeral condyle and femoral condyles). Although there seems little doubt that trauma, in many instances, may be the triggering mechanism for the clinical manifestation of an otherwise covert metabolic defect, the underlying etiology of that endochondral defect remains an enigma.

Osteochondritis Dissecans of the Shoulder

Osteochondritis dissecans of the canine shoulder was first recognized in the veterinary literature in the middle 1950s(8), although loose bodies associated with an epiphyseal defect had been known to afflict humans since the late 1800s.(28) Once defined, the disease was diagnosed with increasing frequency, proving to be a not uncommon orthopaedic condition afflicting the dog.(7,10,16,21,44,57,73) Classically osteochondritis dissecans of the shoulder has been described to occur in breeds that mature at greater than 60 lb body weight(15,16,76) and has been reported to affect the golden retriever, Labrador retriever, English pointer, German shorthaired pointer, Bernese mountain dog, Great Pyrenees, mastiff, Saint Bernard, Newfoundland, Great Dane, Doberman, Rhodesian ridgeback, Old English sheepdog, standard poodle, greyhound, vizsla, Siberian husky, boxer, Afghan hound, rottweiler, German shepherd, Dalmatian, English setter, wirehaired griffon, Irish setter, Irish wolfhound, and Samoyed.(13,15,23,44,54,70,73,74) In contrast to these larger breeds, however, it has also been reported in the cocker spaniel, springer spaniel, Scotch terrier, whippet, border collie, beagle and even a miniature poodle.(20,23,32,36,54,73) By its very nature osteochondrosis of the shoulder may remain subclinical in one animal while expressing itself as bilateral osteochondritis dissecans within the shoulders of another.(15) It is generally believed that afflicted animals experience pain only when those small fissures separating the zone of calcified cartilage and subchondral bone become so extensive as to dissect transchondrally, resulting in the creation of an active osteochondritic lesion that exposes the cleft and subchondral bone to synovial fluid.(38) Affected animals generally present with a lameness, which may be sudden or insidious in onset, but which initially is slight to moderate in degree.(49,74) The lameness generally diminishes with rest and becomes exacerbated with exercise, and in the majority of dogs limb dysfunction increases over a subsequent period of 3 to 4 weeks.(5,74) Very seldom is an affected animal presented immediately for diagnosis; the interval between onset of lameness and consultation ranges from weeks to months.(7,13,15,70,74) While both forelegs may be affected, most owners are aware only of unilateral symptomatology, the limb most severely affected representing the major cause of owner complaint.

While the conversion of an osteochondrosis to an osteochondritis generally heralds the onset of clinical signs, it is apparently the size of the osteochondritic lesion that determines the ultimate degree of lameness an animal manifests clinically.(7,16,23) Affected animals have been described as having an abduction stance and showing a reluctance to hyperextend the shoulder when gaited.(5,23,36,74) Upon physical examination, these animals usually enjoy a full range of motion, although obvious pain is readily manifested upon hyperextension or extreme flexion of the shoulder. Palpation of the shoulder is generally nonrewarding for evaluation of capsular effusion, a function of the amount of overlying musculature. Muscle atrophy may be marked over the supraspinatus, infraspinatus, and brachial areas, particularly if the clinical signs have been severe and the animal has experienced a long course of disease(2) (Fig. 84-7). Several authors have described the presence of a click upon hyperextension of an affected shoulder, probably the result of concomitant degenerative changes within the joint.(23,64,74) No predilection for right or left limb has been noted.(70)

The onset of disease occurs between 5 months and 10 months of age in a large majority of cases, with males affected three times more frequently than females. (28) In approximately 50% of the cases, animals are affected bilaterally (as determined radiographically), although clinical signs of pain and lameness may occur only unilaterally (Table 84-1). While presumptive diagnosis may be made upon assessment of breed and clinical examination, radiography is necessary for definitive diagnosis. A medial-lateral radiograph of the shoulder is taken with the animal lying on the suspect side. The affected limb is pulled cranioventrally while the head and neck are dorsiflexed and the upper leg and chest rotated away (supinated) from the table surface. (28,64) This position projects the limb away from superimposed body and soft tissue structures, revealing a defect in the subchondral bone of the caudal-central aspect of the humeral head. Supplemental films with the humerus in internal and external rotation may be necessary to tangentially outline an occasional lesion lying more medially or laterally. (5)

TABLE 84-1 Osteochondritis Dissecans of the Humeral Head: Literature Review

FIG. 84-7 Lateral photograph of a dog with chronic osteochondritis dissecans of the shoulder. Note the severe muscle atrophy of the supraspinatus and infraspinatus muscles. (Courtesy of Dr. W.H. Riser)

The most common radiographic finding of osteochondritis dissecans in the shoulder is an irregular radiolucent subchondral defect (Fig. 84-8), usually involving the caudal aspect of the humeral head. Additional radiographic findings that may be present include subchondral sclerosis, a calcified linear flap of cartilage (Fig. 84-9), joint mouse, and osteoarthritis (Fig. 84-10). Similar radiographic findings described for the humeral head can also involve the glenoid cavity of the scapula.

Shoulder arthrography, a contrast study, can be helpful in further demonstrating subchondral and articular defects, some of which are not visible on survey radiographs.

FIG. 84-8 Osteochondrosis of the caudal humeral head is seen on a survey lateral radiograph of a shoulder. A mineralized flap of cartilage is visible within the radiolucent subchondral defect.

FIG. 84-9 Osteochondrosis of the shoulder is characterized by a flattened and sclerotic margin to the caudal aspect of the humeral head. A curvilinear mineralized flap of cartilage is present on the caudal margin of the joint.

FIG. 84-10 on a survey radiograph of a shoulder, multiple mineralized joint mice are present, particularly on the caudal aspect of the joint. The caudal margin of the humeral head is irregular and sclerotic.

In osteochondritis dissecans of the canine shoulder the lesion invariably remains localized to the central portion of the caudal aspect of the humeral head, at the approximate junction of the caudal and middle thirds of the articular surface(16) (Fig. 84-11). In full flexion this portion of the humeral head is cradled nicely in the glenoid cavity (Fig. 84-12A); upon full extension, however, the joint surfaces become quite noncongruous (Fig. 84-12, B), with the caudal rim of the glenoid bearing directly on that portion of the head in which osteochondritis dissecans develops.(16,36) This is likewise an area where the humeral articular cartilage remains significantly thicker up until an age of 4 to 5 months, and it is the last region to experience conversion of primary spongiosa to subchondral plate.(49) The findings described above coupled with the fact that the giant breeds complete full development of the subchondral plate at older ages than do smaller breeds has led multiple authors to imply trauma as an initiating factor in the site predilection and clinical manifestations of the disease.(15,16,49,73) Numerous other primary causes of osteochondritis dissecans of the shoulder have been offered in the past, including ischemic necrosis(7), hormonal imbalance,(29,52,54) and nutritional factors.(29,55)

FIG. 84-11 Gross photograph of the humeral heads from a Great Dane with unilateral osteochondritis dissecans. Note the large articular cartilage flap present on the humeral head on the left of the photograph. (Courtesy of Dr. W.H Riser)

FIG. 84-12 Sagittal sections of a normal canine shoulder. In full flexion (A) the glenoid and humeral head are a good fit. In extension (B) the surfaces are incongruent, and the caudal glenoid comes into contact with the caudal central humeral head. (Courtesy of Dr. W.H. Riser)

While it would now appear that osteochondrosis is indeed the primary etiology of osteochondritis dissecans of the shoulder, it would seem that some of the other problems cited above (nutrition, trauma) may indeed be contributing to the manifestation of the clinical syndrome.(47,48,49) Other conditions should be considered in the differential diagnosis of forelimb lameness in a young, large breed dog, particularly lameness involving the shoulder joint and flexing and extending the elbow. Panosteitis, ununited coronoid process, ununited anconeal process, and osteochondritis dissecans of the distal humerus should always remain of concern until ruled out. Indeed, it is not uncommon for animals to be affected with two or three of these diseases simultaneously, which can lead to a perplexing diagnostic challenge. Often making a diagnosis of osteochondritis dissecans of the shoulder joint is much easier than determining the ultimate form of therapy (i.e., medical or surgical) to be used.

In the early veterinary literature, dogs with osteochondritis dissecans of the shoulder were uniformly treated conservatively with variable periods of rest and analgesics.[\(7,10,23,70,74\)](#) In animals afflicted more severely lameness tended to improve slowly, with a period of 9 to 12 months elapsing before animals remained sound after vigorous exercise (with lameness persisting 1 to 2 years in bilaterally affected animals). on long-term follow-up (3-5 years), these animals, although generally free of lameness, tended to exhibit abnormal posturing of the affected limb and radiographically exhibited large joint mice with obvious signs of secondary degenerative joint disease (osteophytosis of the caudal glenoid, caudal humeral head and neck, and bicipital groove; [\(Fig. 84-13\)](#)); moreover, most manifested a history of occasional osteoarthritic lameness or stiffness.[\(7,23,74\)](#) While variable responses to intra-articular injections of corticosteroids were reported,[\(7,44,74\)](#) in general they seemed not to shorten recovery time and should be considered to be contraindicated, particularly in light of current knowledge.[\(69\)](#) Those animals with minimal lesions treated nonsurgically generally improved rapidly, were usually free of lameness within 2 to 3 months, and exhibited much less secondary degenerative joint disease upon radiographic and clinical follow-up years later.[\(23\)](#) Thus, there seems to exist a definite population of animals in whom conservative therapy was and still is indicated, a fact reinforced by the low incidence of bilaterally affected animals requiring surgery on both shoulders (only 20% in one report).[\(13,70\)](#)

There seems little doubt, however, that surgery is both indicated and beneficial in the more severely affected animal. Animals so treated become uniformly free of lameness by the second postoperative month and consistently manifest less radiographic and clinical evidence of secondary degenerative joint disease on long-term follow-up.[\(7,13,23,70\)](#) Criteria for determining the surgical candidate have varied, but most authors seem to agree that those animals who are lame and who have a radiographic lesion greater than 1 cm in diameter and 3 mm deep, free joint bodies, bilateral disease, or a history of failure with medical management should be treated surgically.[\(5,23,28,38,70\)](#) There is some controversy concerning the management of the young (14 to 15-month-old) dog who initially presents with chronic lameness relating to an old osteochondritis dissecans lesion with superimposed secondary osteoarthritis changes.[\(23,38,70\)](#) Older animals presented for acute exacerbations and manifesting radiographic changes compatible with osteochondritis dissecans almost uniformly respond to medical management. Surgery apparently hastens the natural progression of the disease through early removal of the cartilaginous flap, which then allows for the normal ingrowth of granulation tissue into the osteochondral defect with its subsequent transformation into cartilage. Removal of the joint mouse further prevents mechanical lameness resulting from interposition between joint surfaces or within tendon sheaths (biceps brachii).[\(72\)](#) It is important to note that surgery significantly diminishes the rapidity of onset of chronic secondary degenerative joint disease.[\(7,31,64\)](#) Multiple surgical approaches have been described with apparently little variation in morbidity.[\(7,13,17,28,31,38,56,64\)](#) Removal of the flap, whether in situ or as a free joint body, is mandatory [\(Fig. 84-14\)](#). Curettage of the osteochondral defect is often unnecessary if a healthy bed of granulation tissue and islands of cartilage are already

present. However, the edges of the cartilage defect should be made perpendicular if possible, and only areas of subchondral eburnation should be curetted or drilled down to healthy, bleeding bone. Seroma is seemingly the major complication, probably relating to early vigorous postoperative activity and not to suture material, capsular closure, surgical approach, or other facets of the procedure.⁽⁶⁸⁾ Animals generally walk on the affected limb within 5 to 7 days and are seemingly normal within 4 to 6 weeks.^(7,13,64,70) Factors occasionally responsible for surgical difficulties include inadequate surgical approach, a thickened joint capsule limiting joint manipulation, broad-based defects, medially situated defects, fragmentation of the free flap, and sequestration of the flap in inaccessible portions of the joint (biceps tendon sheath).⁽¹²⁾ Postoperative care should include routine wound management, with close confinement for one week followed by leash activity for one month.^(5,70)

Osteochondritis Dissecans of the Medial Humeral Condyle

It would appear that osteochondrosis may manifest itself as one of three distinct disease entities occurring within the canine elbow, each of which may ultimately result in a significant amount of osteoarthritis and functional impairment.⁽⁵⁰⁾ Not surprisingly, one or more of these developmental anomalies may occur either alone or in combination within one or both elbows of an affected animal. Because all three disease processes ultimately result in joint instability by disrupting intra-articular structures, and because of the consistent fashion in which joint tissues respond to insult, it is not surprising that all three entities incite similar clinical manifestations, particularly in the early stages of the disease process. At 4 to 6 months of age, affected animals tend to exhibit a stiff forelimb gait seen initially after periods of rest and easily warmed out of. Persistent lameness (either unilateral or bilateral) generally develops by 6 to 8 months of age, with radiography providing the only key to early differential diagnosis. Ununited anconeal process can be diagnosed after a dog has reached 20 weeks of age; a discussion of its pathogenesis and treatment is found in Chapter 85. Dogs afflicted with ununited (fragmented) coronoid process and osteochondritis dissecans of the medial humeral condyle (elbow osteochondritis dissecans) do not generally manifest radiographic changes until 28 to 32 weeks of age, although clinical signs have already been present for many months.⁽⁴⁹⁾ If initial radiography proves unrewarding at 5 months of age, animals suspected of having these latter two diseases should be returned at a later date for further radiographic screening. Unfortunately, radiologic differentiation between the two lesions (ununited coronoid process and elbow osteochondritis dissecans) is often difficult at best, with surgical exploration ultimately providing the definitive diagnosis.^(19,25) Ununited coronoid process, which is discussed thoroughly in Chapter 85, will be referred to frequently in this discussion of osteochondritis dissecans of the medial humeral condyle owing to the close association of the two disease entities in both clinical and radiologic presentation.

Osteochondritis dissecans of the medial humeral condyle of the dog was first reported in the veterinary literature in 1974 by Olsson,⁽⁴⁶⁾ in a paper that likewise described the pathologic

entity now commonly referred to as ununited (fragmented) coronoid process. A condition perhaps not as commonly diagnosed as ununited coronoid process, [\(24,25,48\)](#) osteochondritis dissecans of the medial humeral condyle (elbow osteochondritis dissecans) also affects large and giant breeds of dogs, having been specifically described in the Labrador retriever, rottweiler, Newfoundland, golden retriever, chow, German shepherd, and bearded collie [\(19,24,25,39,63,75,76\)](#). Whereas for years ununited anconeal process was considered to be the main cause of secondary osteoarthritis in the elbow of young dogs, in fact osteochondritis dissecans of the medial humeral condyle and ununited coronoid process together are now considered to be the most common causes of canine elbow arthrosis as reported by several authors in various countries. [\(25,48,49,71\)](#)

While there seems to be a definite predisposition for males to develop either elbow osteochondritis dissecans or ununited coronoid process, a sexual predisposition in males for osteochondritis dissecans of the humeral condyle alone as yet remains speculative ([Table 84-2](#)). [\(25\)](#) Affected animals are usually presented at 8 to 10 months of age for forelimb lameness, having progressed from a stiffness that initially was present only after rest to an intermittent lameness that is exacerbated by exercise. The animals have a tendency to place the affected limb in both abduction and supination. Generally joint effusion is not present, although animals with a well-developed arthrosis tend to exhibit an increase in joint diameter with a decreased range of motion (particularly in flexion). [\(24\)](#) Pain can often be elicited upon hyperflexion/extension of the elbow. Not all animals are symptomatic at an early age; older animals often present with an acute episode of elbow lameness and upon radiologic examination manifest severe secondary degenerative arthrosis, not surprising in a disease of variable expression with long-term sequelae. In such cases definitive radiographic diagnosis is virtually impossible owing to the severity of secondary degenerative changes obscuring the inciting problem. Bilateral radiographic lesions occur in approximately 50% of the animals presented for unilateral symptomatology.

**TABLE 84-2 Osteochondritis Dissecans of the Medial Humeral Condyle:
Literature Review**

While osteochondritis dissecans of the medial humeral condyle remains the sole cause of elbow osteoarthritis in many canine elbows, it often occurs simultaneously with ununited coronoid process and may be difficult to distinguish from the so-called kissing lesions that occur on the medial humeral condyle in response to the latter. [\(6\)](#) Radiographically, the presence of a triangular area of subchondral radiolucency on the weight-bearing surface of the medial humeral condyle is reported to be diagnostic for osteochondritis dissecans of the elbow [\(24,48,65,75\)](#) ([Fig. 84-15](#)). Subchondral sclerosis surrounding the defect and calcification of the articular cartilage flap may further solidify the radiographic diagnosis and are best visualized in the craniocaudal and craniocaudal-medial oblique projections. [\(65\)](#) In older dogs with advanced lesions, large calcified round bodies (joint mice) have been reported to occur at

various locations within the joint. Those animals presenting early and with such classic radiology changes are the minority; most animals manifest either subtle degrees of generalized secondary osteoarthritis (more pronounced in the medial joint compartment) or changes in the medial humeral condyle accompanied by changes in the area of the coronoid process of the ulna significant enough to prevent the diagnosis of a singular osteochondritis dissecans lesion. [\(6,19,48,75\)](#) In even older dogs the signs of osteoarthritis may be so severe as to preclude a determination of inciting cause. In general, the extent of the radiographic changes shows no correlation with the age of onset or duration and severity of clinical signs, not unlike osteochondritis dissecans afflicting other parts of the body.

FIG. 84-15 Lateral (A), oblique (B.), and cranial-caudal (C) radiographic views of a canine elbow demonstrate osteochondritis dissecans of the medial humeral condyle.

Since there is much less tendency toward spontaneous healing of osteochondritis dissecans of the elbow than in the shoulder, for example, and in light of the difficulty in differentiating it clinically from ununited coronoid process, surgery has assumed a major role in the diagnosis and treatment of this disease. Numerous surgical approaches to the elbow have been offered, none of which completely affords direct visualization of all components of this composite joint. [\(6,19,25,46,56,63,72\)](#) Complete visualization of the medial humeral condyle is necessary, both for removal of the cartilaginous flap and for possible curettage of the resultant defect in the weight-bearing surface. Thorough inspection of both the caudomedial [\(65\)](#) and cranial [\(48\)](#) joint compartments may be necessary for retrieval of a flap that is loose or that has adhered to the synovium. Occasionally it may be necessary to make a separate lateral incision to enter the caudolateral joint compartment to remove a joint mouse lodged proximally in that location. [\(48\)](#) Finally, a thorough inspection of the medial coronoid process should always be made to evaluate its integrity and the role that structure is playing in the overall arthritic process.

While initial reports implied a guarded prognosis in those animals treated surgically, [\(49\)](#) more recently authors have reported the postoperative duration of lameness to average 5 weeks with an overall good functional outcome. [\(19,25\)](#) One author, in reviewing 25 affected animals, has offered the prognosis for dogs operated on for elbow osteochondritis dissecans to be better than that for ununited coronoid process, [\(19,25\)](#) with a return to soundness of approximately 70% in contrast to earlier reports. [\(19,49\)](#) It would seem that if surgery is to be performed, it should be done so early in the course of disease, since animals with severe secondary osteoarthritis changes do not fare as well postoperatively as their less severely affected counterparts. [\(19,25\)](#) The need for (or effect of) bilateral surgery in those animals so affected as yet remains speculative. Further elucidation of the characteristics of this disease entity in the dog awaits more documented, detailed reports of case series.

Osteochondritis Dissecans of the Stifle

It has recently been stated that approximately 10% to 15% of all cases of osteoarthritis of the knee seen in large dogs for which no other obvious cause of arthrosis is apparent are the result of old osteochondritic lesions.^(49,50) A much more common entity in dogs of larger breeds than previously assumed, osteochondritis dissecans may be somewhat difficult to diagnose clinically because there is often no obvious lameness; affected animals may manifest only a disturbed gait pattern in the hindlegs reminiscent of hip dysplasia. The disease has been documented to affect the German shepherd, bull mastiff, Samoyed, German wirehaired pointer, wolfhound, Labrador, standard poodle, greyhound, Great Dane, Saint Bernard, boxer, chow, collie, giant schnauzer, Doberman, border collie, and Staffordshire bull terrier.^(2,5,18,37,58) Most animals present at 6 months of age with a lameness gradual in onset and exacerbated with exercise.^(2,58) In cases of longer duration, lameness appears to be more intermittent in nature. Bilaterally affected animals often exhibit a shifting hindleg lameness; alternatively, they may manifest difficulty only upon rising and hesitancy in moving from the sitting position; when standing, they may tend to adopt a crouched position. Upon physical examination affected limbs often have limited extension, with passive range of motion producing discomfort and crepitus. Males are seemingly affected more frequently than females, the lateral condyle more so than the medial, and bilaterally affected animals outnumber those with solitary lesions ([Table 84-3](#)). Interestingly, in those animals affected bilaterally, the defect invariably occurs in the same condyle of both femora.^(2,18,36,62)

TABLE 84-3 Osteochondritis Dissecans of the Femoral Condyles	
Author	Year
Lalbas	1958
Rubens	1970
Shaw	1971
Smith	1971
Dennis	1982

TABLE 84-3 Osteochondritis Dissecans of the Femoral Condyles

Radiography is essential to early diagnosis ([Fig. 84-16](#)). Standard cranial-caudal and lateral views should be taken; occasionally cranial-caudal obliques are necessary to outline the defect. In affected animals the lateral view initially demonstrates an irregularity in the contour of the femoral condyle, with an eventual flattening of the cranial to middle third of the articular margin with or without an accompanying radiolucency of underlying subchondral bone. On the cranial-caudal view, lesions in the lateral condyles generally appear as medially situated spheroid areas of lucency often exhibiting sclerotic margins, while the medial condylar defects generally present as convex deformities of the articular margin. Associated signs of degenerative osteoarthritis may or may not accompany these routine findings.



FIG. 84-16 Lateral (A) and cranial-caudal (B) radiographic views of a canine stifle demonstrate osteochondritis dissecans of the lateral femoral condyle.

Free mineralized fragments (joint mice, corpora libera) occur not infrequently and are best visualized on the lateral radiograph. Often found in multiples within an affected joint, they

generally range in size from 1 mm to 8 mm and are cartilaginous in nature. They have been found to localize in the suprapatellar pouch, cranial and caudal joint regions, as well as in the recess of the origin of the extensor digitorum longus and the recess proximal to the sesamoids of the gastrocnemius.⁽²⁾ Radiography is generally unrewarding prior to 6 months of age, after which time screening films taken at monthly intervals in suspect animals may prove rewarding.

Many cases of osteochondritis dissecans of the stifle remain undetected and heal spontaneously, leaving only a scar on the affected condyle;⁽⁴⁹⁾ others are manifest by an acute lameness accompanied by joint effusion and obvious radiographic changes compatible with osteochondritis dissecans. Finally, in older, untreated animals severe osteoarthritis is most likely a sequel to larger lesions that have resulted in a change of shape and size of the affected femoral condyle with the concomitant development of abnormal joint biomechanics. Surgical treatment is seemingly indicated in the acutely lame animal in hopes of reducing the duration of lameness, or in those animals who manifest a free cartilaginous flap, which may result in a mechanical lameness whenever it becomes interposed between joint surfaces^(2,18) (Fig. 84-17). In one report of five dogs bilaterally affected, only one animal required bilateral arthrotomy. Thus it would appear that in many respects osteochondritis dissecans of the stifle behaves in a manner to osteochondritis dissecans affecting the shoulder joint, although this must yet be borne out by more reports on affected animals.

FIG. 84-17 (A) Intraoperative photograph of the osteochondritis dissecans lesion of the lateral femoral condyle that is shown radiographically in Figure 84-16,A. The articular cartilage flap (B) was removed.

Differential diagnosis should include any cause of stifle osteoarthritis (cranial cruciate ligament rupture, avulsion of the origin of the long digital extensor, fracture of the lateral condyle, as well as avulsion of the popliteus muscle or head of the gastrocnemius). Prognosis in those animals treated surgically has been good, with lameness resolving in 4 to 6 weeks.^(18,36,37) No long-term clinical trials of animals treated conservatively have been published, thus, no definite conclusions as to those factors determining good clinical outcome can as yet be made.

Osteochondritis Dissecans of the Talocrural Joint

Osteochondritis dissecans affecting the medial ridge of the trochlea of the tibial tarsal bone in the dog was first mentioned in the veterinary literature in 1975.⁽⁴⁸⁾ It has not been until more recently, however, that detailed case reports and series have been accessible for review (Table 84-4).^(33,42,45,66) The disease has been described to affect the Labrador retriever, golden retriever, rottweiler, Queensland cattle dog, Irish setter, bull terrier, and Australian cattle dog. Males are apparently affected more frequently than females. The number of animals with unilateral lesions approximates those afflicted bilaterally. Site predilection is believed to relate to biomechanical forces.⁽⁶⁶⁾ Clinical signs are generally manifested around 6 months of age,

the presenting complaint ranging from straight hindleg conformation to poor hindlimb movement (reminiscent of hip dysplasia) and lameness of a mild to moderate degree. (33,49) Affected animals manifest a shortened stride and generally become more lame with exercise, those animals affected bilaterally often develop a subtle lameness on the opposite limb. (45)

Upon physical examination, range of motion in the tarsocrural joint, although usually painless, (33) is decreased, the amount of restriction evident primarily in flexion and dependent on the degree of secondary osteoarthritic changes. (49) Crepitation may be evident upon Sexton and extension, while pain may be elicited upon forced flexion or by direct palpation of the caudomedial surface of the tibial tarsal bone. (45,66) Periarticular soft tissue thickening is often present, generally in proportion to the chronicity of the lesion, (42,45) with capsular distension appearing in both younger and older animals alike. (33)

Diagnosis is readily made with radiography; dorsoplantar and lateral radiographs of the talocrural joint (hock) should be taken in both full extension and flexion. A dorsoplantar view with the hock under a valgus stress may be of some benefit in those animals exhibiting obvious joint laxity on physical examination. Both limbs should be radiographed. Changes indicative of osteochondritis dissecans include an increased medial talocrural joint space, flattening of the medial trochlear ridge with or without the presence of an osteochondral flap, existence of radiopaque joint mice, lysis or fracture of the medial (tibial) malleolus, and other changes compatible with chronic osteoarthrosis throughout the joint (33,42,48) (Fig. 84-18). The osteochondritic lesion is generally confined to the proximal third of the medial ridge of the trochlea but sometimes extends into the trochlear groove. (33) The free flaps are frequently ossified with attachments to the synovial membrane, collateral ligament, or both. (45,49) Kissing lesions on the medial cochlea of the distal tibial articulating surface have been reported. The apparent laxity of the medial collateral ligaments with valgus deviation seen in some animals may relate to a combination of cartilage erosion, displacement of the osteochondral fragment, and continued stress with the burden of weight-bearing. Progressive osteoarthrosis is seemingly a hallmark of the disease, with or without surgical intervention. (66) Several surgical approaches to the medial talocrural joint have been suggested; the goal of the approaches is removal of the osteochondral fragment and curettage of the defect. (42,45,56,66) While some believe surgical intervention to be of questionable long-term benefit in affected animals, (66) others have stated that conservative treatment has no role in the management of talocrural osteochondritis dissecans and early diagnosis and surgical intervention are essential to the prevention of chronic lameness. (33,40,42,48)

FIG. 84-18 Dorsoplantar radiograph of a canine talocrural joint demonstrates osteochondritis dissecans of the medial ridge of the trochlea of the tibial tarsal bone.

Osteochondrosis of the Cervical Intervertebral Joints

In the early veterinary literature, the term spinal or vertebral osteochondrosis related to a disease process distinct from osteochondrosis as it is understood in the current context. Scheuermann first described in children a condition in which calcification and ossification of the ring apophysis of the vertebral body was interrupted, resulting in an irregular radiographic appearance of the vertebral epiphyses and often kyphotic deformities.[\(72\)](#) He classified it as an osteochondrosis, probably the result of aseptic necrosis. Confronted by immature animals manifesting pain referable to the spine and radiographic lesions affecting the vertebral bodies and end-plate of undiagnosed etiologies, many in the veterinary field believed these animals to be afflicted with a disease similar to that described by Scheuermann. Probably those cases of spinal osteochondrosis reported in the veterinary literature were of infectious or other etiologies and not truly a result of abnormalities in epiphyseal endochondral ossification.[\(14\)](#)

Cervical vertebral instability (cervical spondylolisthesis, wobblers disease) affecting young Great Danes and older Doberman pinschers is a disease commonly diagnosed in veterinary medicine. Its underlying etiopathogenesis has remained obscure. Olsson has encountered a few cases in Great Danes in whom the instability was believed to relate to a lesion affecting the articular surfaces of the facet joints in one or two pairs of cervical vertebrae. The lesions grossly resembled those of healed osteochondrosis and were similar to those lesions seen in cervical osteochondrosis in swine.[\(49,50\)](#) Further exploration into this as a possible cause of cervical vertebral instability will hopefully be forthcoming. Current treatments are all aimed at surgical stabilization and produce variable results.

Osteochondritis Dissecans of the Distal Radius

There is one report of osteochondritis dissecans occurring in the distal radius of a 9-month-old Dalmatian weighing 35 lb. The animal had experienced a 2-month history of lameness, and radiography revealed two small fragments of bone between the radial carpal bone and the distal radial epiphysis. Antebrachiocarpal arthrotomy was performed and the two ossifies were removed, one of which had to be freed from its partial capsular attachments. No mention of a defect on the weight-bearing surface was made. The animal made an uneventful recovery.[\(9\)](#)

Miscellaneous Diseases Possibly Osteochondritic in Nature

As a systemic disease, osteochondrosis exhibits an array of clinical manifestations. Retention of cartilage may occur in any growth zone, the size of the lesion and its susceptibility to trauma determining to a large extent its clinical significance. Osteochondrosis has been implicated in the etiology of retained endochondral cartilage cores of the distal ulna, a disease that may cause only a subtle, short-lived valgus deviation of the carpus or in its more

severe form may result in severe radius curvus and elbow subluxation.[\(48-50\)](#) Similarly, disturbances in endochondral ossification affecting the distal femur and proximal tibia have been implicated as a cause of genu valgum. [\(50\)](#) Slipped femoral capital epiphysis is a common manifestation of osteochondrosis in swine, and the possibility exists that it may cause a similar clinical entity in the dog.[\(30,49,50\)](#) Finally, it is well known that hip dysplasia is a developmental Condition of multifactorial etiology, with a heritability of approximately 0.3%, in which overnutrition plays a significant role. It would appear that osteochondritis dissecans of the dorsolateral rim of the acetabulum occurs in some dysplastic animals who, upon necropsy, have manifested osteochondrosis in other growth zones.[\(49\)](#)

References

1. Alexander JE, Pettit GD: Spinal osteochondrosis in a dog. *can Vet J* 8:47, 1967
2. Arbesser E: Osteochondrosis dissecans der Femurkondylen beim Hund. *Wien Tierarzt Mschr* 61:303, 1974
3. Arbesser E: Die Lage freier Gelenkkörper bei Osteochondrosis dissecans des Humeruskopfes. *Wien Tierarzt Mschr* 62:437, 1975
4. Bartels JE, Dorn AS, Britt AL: Osteochondrosis of the vertebrae in a dog. *can Vet J* 11:47, 1970
5. Berzon JL: Osteochondritis dissecans in the dog: Diagnosis and therapy. *J Am Vet Med Assoc* 175:796, 1979
6. Berzon JL, Quick CB: Fragmented coronoid process: Anatomical, clinical and radiographic considerations with case analysis. *J Am Anim Hosp Assoc* 16:241, 1980
7. Birkeland R: Osteochondritis dissecans in the humeral head of the dog. *Nord Vet Med* 19:294, 1967
8. Brass W: Osteochondritis in the dog. *Tierärztliche Umschau* 2:200, 1956
9. Butler HC, Wallace LJ, Ladds PW: Osteochondritis dissecans of the distal end of the radius in a dog. *J Am Anim Hosp Assoc* 7:81, 1971
10. Campbell JR: Shoulder lameness in the dog. *J Small Anim Pract* 9: 189, 1968

11. Carrig CB, Morgan JB: Microcirculation of the humeral head of the immature dog. *J Am Vet Radiol Soc* 15:23, 1974
12. Caywood DD, Schenk MP, Wilsman NJ et al: Craniomedial approach to the canine scapulohumeral joint. *ACVS Journal* 9:74, 1980
13. Clayton Jones DG, Vaughan LC: The surgical treatment of osteochondritis dissecans of the humeral head in dogs. *J Small Anim Pract* 11:803, 1970
14. Convery FR, Akeson WH, Keown GH: The repair of large osteochondral defects (an experimental study in horses). *Clin Orthop Rel Res* 82:253, 1972
15. Cordy DR, Wind A: Transverse fracture of the proximal humeral articular cartilage. *Pathol Vet* 6:424, 1969
16. Craig PH, Riser WH: Osteochondritis dissecans in the proximal humerus of the dog. *J Am Vet Radiol Soc* 6:40, 1965
17. Dee J: A simplified approach to the canine shoulder for the treatment of Osteochondritis dissecans. *J Am Anim. Hosp Assoc* 8:111, 1972
18. Denny HR, Gibbs C: Osteochondritis dissecans of the canine stifle joint. *J Small Anim Pract* 21:317, 1980
19. Denny HR, Gibbs C: The surgical treatment of Osteochondritis dissecans and ununited coronoid process in the canine elbow joint. *J Small Anim Pract* 21:323, 1980
20. Dingwall JL, Staples JW, Pennock PW: Osteochondritis dissecans in a miniature poodle. *MVP* 53:47, 1972
21. Evans PJ: Shoulder dysplasia in a Labrador. *J Small Anim Pract* 9:55, 1968
22. Furukawa T, Eyre DR, Koide S et al: Biochemical studies on repair cartilage resurfacing experimental defects in the rabbit knee. *J Bone Joint Surg* 62:79, 1980
23. Griffiths RC: Osteochondritis dissecans of the canine shoulder. *J Am Vet Med Assoc* 153:1733, 1968
24. Grondalen J: Arthrosis with special reference to the elbow joint of young rapidly growing dogs: I and II. *Nord Vet Med* 31:62, 1979
25. Grondalen J: Arthrosis with special reference to the elbow joint of young rapidly growing dogs: III. *Nord Vet Med* 31:520, 1979

26. Grondalen T, Vangen O: Osteochondrosis and arthrosis in pigs: V. A comparison of the incidence in 3 different lines of the Norwegian Landrace breed. *Acta Vet Scand* 15:16, 1974
27. Harrison JW: Osteochondritis dissecans. *Proceedings of the American Animal Hospital Association* 42:423, 1975
28. Harrison JW, Hohn RB: Osteochondritis dissecans. In Bojrab MJ (ed): *Current Techniques in Small Animal Surgery*, vol 1. Philadelphia, Lea & Febiger, 1975
29. Hedhammer A, Wu FM, Krook L et al: Overnutrition and skeletal disease: An experimental study in growing Great Dane dogs. *Cornell Vet* 64: 1, 1974
30. Herron M: Osteochondritis of the femoral head. *Southwest Vet* 23:9, 1969
31. Hohn RB: Osteochondritis dissecans of the humeral head. *J Am Vet Med Assoc* 163:69, 1973
32. Johnson KA, Dennis KA: Osteochondritis dissecans in a beagle. *Aust Vet J* 54:364, 1978
33. Johnson KA, Howlett CR, Pettit GD: Osteochondrosis in the hock joints in dogs. *J Am Anim Hosp Assoc* 16:103, 1980
34. Kincaid SA, VanSickle DC: Regional histochemical and thickness variations of adult canine articular cartilage. *Am J Vet Res* 42:428, 1981
35. Kinzel GL, VanSickle DC, Hillberry BM et al: Preliminary study of the in-vivo motion in the canine shoulder. *Am J Vet Res* 37: 1505, 1976
36. Knecht CD: Osteochondrosis of the shoulder and stifle in 3 of 5 border collie littermates. *J Am Vet Med Assoc* 170:58, 1977
37. Lafeber TJ, Beckwith J, Strombeck DR: What is your diagnosis? *J Am Vet Med Assoc* 134:267, 1959
38. Leighton RL: Osteochondritis dissecans of the shoulder joint of the dog. *Vet Clin North Am* 1:39 1, 1971
39. Leighton RL: Osteochondritis dissecans of the elbow in a dog. *Vet Med Sm An Clin* 311, 1978
40. Leighton RL: Osteochondritis dissecans of the hock in a dog. *Vet Med/Small Anim Clin* 325, 1979

41. Ljunggren G, Reiland S: Osteochondrosis in adolescent animals: An endocrine disorder? *Calcif Tissue Res* 4(suppl): 150, 1970
42. Mason TA, Lavelle RB: Osteochondritis dissecans of the tibial tarsal bone in dogs. *J Small Anim Pract* 20:423, 1979
43. Mitchell N, Shepard N: Healing of articular cartilage in intra-articular fractures in rabbits. *J Bone Joint Surg* 62A:628, 1980
44. Mostosky U: Osteochondritis dissecans of the canine shoulder. Proceedings of the 13th Gaines Veterinary Symposium, Athens, GA, 1964, pp 16 19
45. Olson NC, Mostosky UV, Flo GL et al: Osteochondritis dissecans of tarsocrural joint in 3 canine siblings. *J Am Vet Med Assoc* 176:635, 1980
46. Olsson SE: En ny typ av armbagsledsdysplasi hos hund? *Svensk Vet Tidn* 26:152, 1974
47. Olsson SE: Osteochondritis dissecans in the dog. Proceedings of the American Animal Hospital Association 42:362, 1975
48. Olsson SE: Lameness in the dog: A review of lesions causing osteoarthritis of the shoulder, elbow, hip, stifle, and hock joints. Proceedings of the American Animal Hospital Association 42:363, 1975
49. Olsson SE: Osteochondrosis: A growing problem to dog breeders. *Gaines Progress*, 1-11, 1976
50. Olsson SE: Osteochondrosis in the dog. In Kirk RW (ed): *Current Veterinary Therapy VI*. Philadelphia, WB Saunders, 1980.
51. Olsson SE, Reiland S: Osteochondrosis. *Acta Radiol (suppl)* 358:299A, 1978
52. Paatsama: Aetiological factors in osteochondritis dissecans. *Acta Orthop Scand* 46:906, 1975
53. Paatsama S, Rokkanen P, Jussila J et al: Somatotropin, thyrotropin and corticotropin hormone induced changes in the cartilages and bones of the shoulder and knee joint in young dogs. *J Small Anim Pract* 12:595, 1971
54. Paatsama S, Rokkanen P, Jussila J et al: A study of osteochondritis dissecans of canine humeral head using histological, OTC bone labeling, microradiographic and microangiographic methods. *J Small Anim Pract* 12:603, 1971

55. Palmer CS: Osteochondritis dissecans in Great Danes. *Vet Med/Small Anim Clin* 65:994, 1970
56. Piermattei D, Greeley R: *An Atlas of Surgical Approaches to the Bones of the Dog and Cat*, 2nd ed. Philadelphia, WB Saunders, 1979
57. Pobisch R: Aseptische Nekrose des Humeruskopfeseine Lahmheitsursache bei Junghunden. *Wien Tierarztl Mschr* 49:571, 1962
58. Punzet G, Walde I, Arbesser E: Zur Osteochondrosis dissecans genu des Hundes. *Kleintierpraxis* 20:73, 1975
59. Rang M: *Children's Fractures*. Philadelphia, JB Lippincott, 1974
60. Reiland S: Osteochondrosis in the pig. Reprint *Acta Radiol* 1 - 118, 1975
61. Rejno S, Stromberg B: Osteochondrosis in the horse:II. Pathology reprint. *Acta Radiol* 1-28, 1976
62. Robins GM: Osteochondritis dissecans in the stifle joints in a bitch. *J Small Anim Pract* 11:813, 1970
63. Robins GM: Osteoarthrosis of the elbow joint in dogs. *Aust Vet J* 52:485, 1976
64. Robins GM: Osteochondritis dissecans in the dog. *Aust Vet J* 54:272, 1978
65. Robins GM: Some aspects of the radiographical examination of the canine elbow joint. *J Small Anim Pract* 21:4 17, 1980
66. Rosenblum GP, Robins GM, Carlisle CH: Osteochondritis dissecans of the tibiotarsal joint in the dog. *J Small Anim Pract* 19:759, 1978
67. Scherrer PK, Hillberry BM, VanSickle DC: Determining the in vivo areas of contact in the canine shoulder. *J Biomech Eng* 101:271, 1979
68. Schiller AG, Smith CW, Seaman WJ: Surgical treatment of osteochondritis dissecans without suturing the joint capsule. *Vet Med/Small Anim Clin* p 50, 1978
69. Short CR, Beadle RE: Pharmacology of antiarthritic drugs. *Vet Clin North Am* 8:401, 1978
70. Smith CW, Stowater JL: Osteochondritis dissecans of the canine shoulder joint: A review of 35 cases. *J Am Anim Hosp Assoc* 11 :658, 1975

71. Tirgari M: Clinical, radiographical, and pathological aspects of arthritis of the elbow in dogs. *J Small Anim Pract* 15:671, 1974
72. Turek SL: *Orthopedic Principles and Their Application*, 3rd ed. Philadelphia, JB Lippincott 1977
73. Van Sickle DC: Selected orthopedic problems in the growing dog. *Am Anim Hosp Assoc* p 1, 1975
74. Vaughan LC, Jones DGC: Osteochondritis dissecans of the head of the humerus in dogs. *J Small Anim Pract* 9:283, 1968
75. Wissler J, Sumner-Smith G: Osteochondrosis in the elbow joint in the dog. *J Am Anim Hosp Assoc* 13:349, 1977
76. Wood AKW, Bath ML, Mason TA: Osteochondritis dissecans of the distal humerus in a dog. *Vet Rec* p 489, 1975