Canine Skeletal Development & Soundness

Presented at
The North American
Veterinary Conference
Orlando, Florida
January 13, 1998

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Asynchronous Growth of the Radius and Ulna in the Dog

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At birth, the long bones of dogs are composed of one primary ossification center and one or more secondary ossification centers. Secondary ossification centers are separated from the primary center by a cartilaginous plate, allowing for growth in length. The epiphysis is also covered with this growth-plate cartilage, allowing for proportional growth of the bone end. The proportional growth of the diaphyses takes place by appositional growth underneath the periosteum. The growth in length and proportional growth of the epiphysis take place via the process of endochondral ossification (Figure 1). Growth in length and development and maturation of secondary ossification centers depend on the breed of the dog[1,2] and are retarded in cases of high calcium intake.[3]

The growth of paired bones is so highly orchestrated that both bones continue to form congruent joint surfaces at both ends. In the tibia and fibula this is less remarkable because 50% of the growth in both bones occurs in the proximal and distal growth plate, respectively.[4] However, there is a noteworthy discrepancy in growth rate in the radius and ulna in the antebrachium. In the radius, 60% to 75% of bone growth occurs at its distal growth plate and 25% to 40% at its proximal growth plate, whereas the distal growth plate of the ulna accounts for approximately 80% to 90% of its growth in length (Table 1). During the growth period, the radius and ulna shift in relation to each other, but the elbow joint surface remains congruent. The distal radius and ulna keep in line with the carpal bones to form a normal antebrachiocarpal joint.[1,2,5]

Various causes result in different growth lengths of the antebrachium. In some cases this abnormality causes lameness and/or abnormal leg configuration. Owners of such dogs often contact veterinarians for information on the causes and prevention of this condition and possible therapeutic measures.

This article discusses various disturbances in asynchronous growth of the radius and ulna in dogs, including etiology, clinical signs, prevention, and treatment.

Growth in length and development and maturation of secondary ossification centers depend on the breed of the dog and are retarded in cases of high calcium intake.

INHERITED-RELATED INCONGRUITY

Basset Hounds, Sky Terriers, Dachshunds, and other chondrodystrophic breeds may have an inherited form of asynchronous growth of the radius and ulna. Very active cartilage cell mitosis and hypertrophy occur at the distal growth plate of the ulna, which has a typical V-shape in dogs. In chondrodystrophic breeds, the growth plate has an oblique shape (Figure 2) on a mediolateral radiograph. The decreased growth in length, which is seen in all long bones, is most pronounced in the ulna. This causes the longer radius to extend proximally beyond the joint level of the ulna, causing an elbow incongruity. The radius "pushes" the humerus in a proximal direction, against the anconeal process.[6] In young Basset Hounds up to 6 months of age, this may cause a discontinuity in the cartilaginous attachment of the anconeal process to the proximal ulna, starting at its distal end. Eventually, this may cause an ununited anconeal process (UAP) that, together with the joint incongruity, results in lameness and osteoarthrosis of the affected elbow (Figure 2). In more mature dogs (older than 6 months of age), the anconeal process has united with the ulna and will not be "pushed off" its origin. However, the compression of the

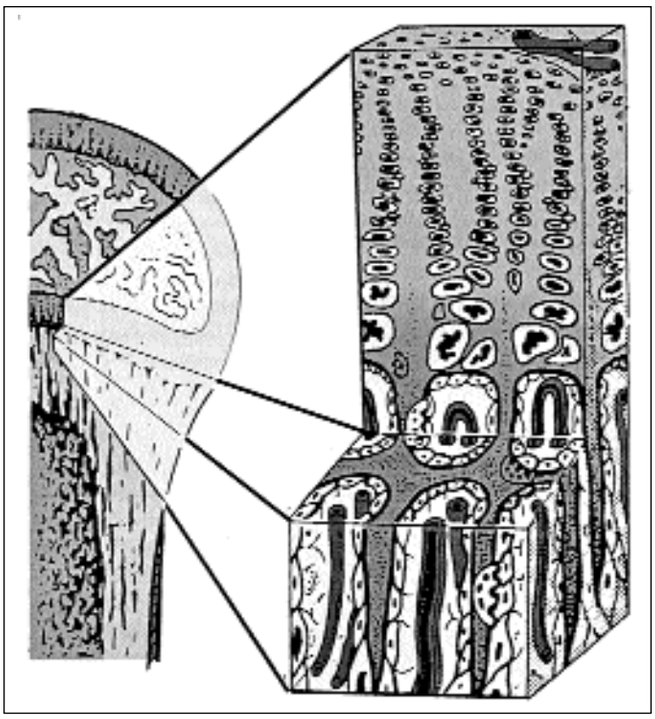

Figure 1—*Schematic representation of the proximal end of a long bone. Endochondral ossification takes place in the growth plate cartilage and in cartilage covering the epiphysis. The inset shows the process of endochondral ossification: chondrocytes lying in rows while dividing and enlarging. The intercellular substance mineralizes, and cell death occurs in the distal cartilage. Blood vessels grow into the empty lacunae and introduce osteoblasts, which lay down osteoid on the mineralized cartilage cones. Multinucleated osteoclasts both remodel and remove cancellous bone. (Modified from Nap RC, Hazewinkel HAW: Growth and skeletal development in the dog in relation to nutrition: A review.* Vet Q *16:50–59, 1994.)*

humerus against the anconeal process is very painful for the dog (Figure 3). This so-called "distractio cubiti" is most painful in dogs between 6 and 12 months of age. Some Basset Hounds of this age may simultaneously experience unilateral lameness due to severe incongruity in one elbow and UAP in the other. When the UAP is still partially attached by cartilage, the tension can be released by performing an ulnotomy. In complete and more chronic cases, UAP can be treated by removal after arthrotomy (see pp 31–32). Distractio cubiti can be treated conservatively with the aid of nonsteroidal antiinflammatory drugs (NSAIDs), especially when the incongruity is small. When severe incongruity and pain are still present at the end of the growing period, dogs can be treated successfully with

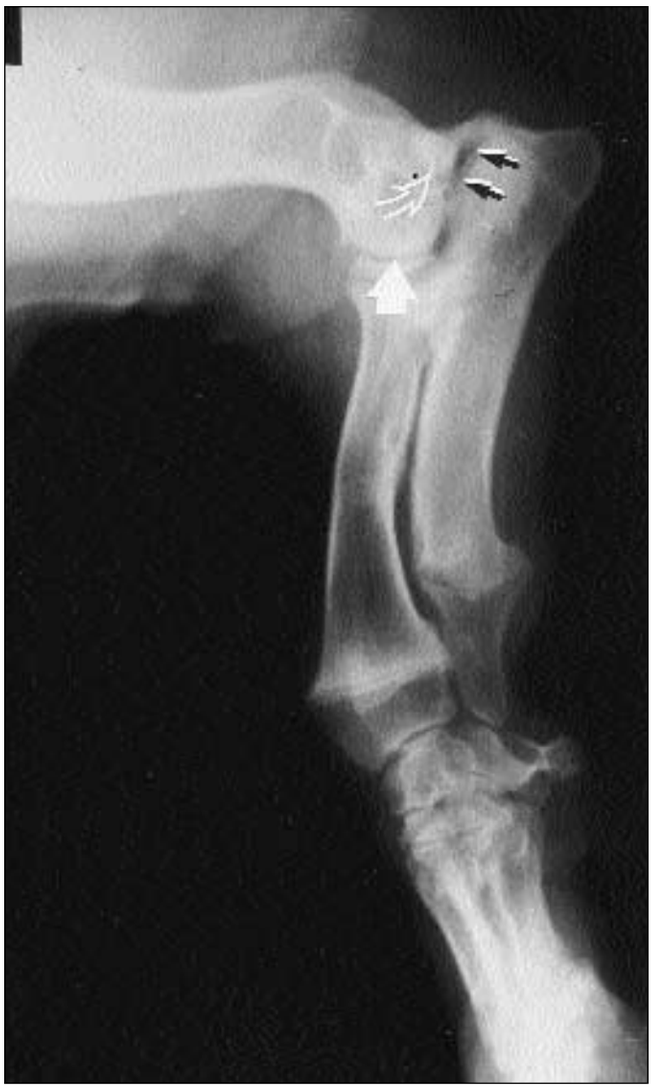

Figure 2—*Radiograph of a 6-month-old Basset Hound with severe right front leg lameness due to elbow incongruity. The radius pushes the humerus against the anconeal process* (white arrows)*, causing an ununited anconeal process* (black arrows).

TABLE 1
Skeletal Growth in Dogs[1–3]

Area of Growth	Radiologic Appearance of Secondary Center of Ossification (days)	Closure of Growth Plate (weeks)	Contribution of the Growth in Radius or Ulna
Distal ulna	28–64	24–47	80%–90%
Proximal ulna	35–64	29–37	10%–20%
Distal radius	14–28	35–47	60%–75%
Proximal radius	21–35	35–52	25%–40%

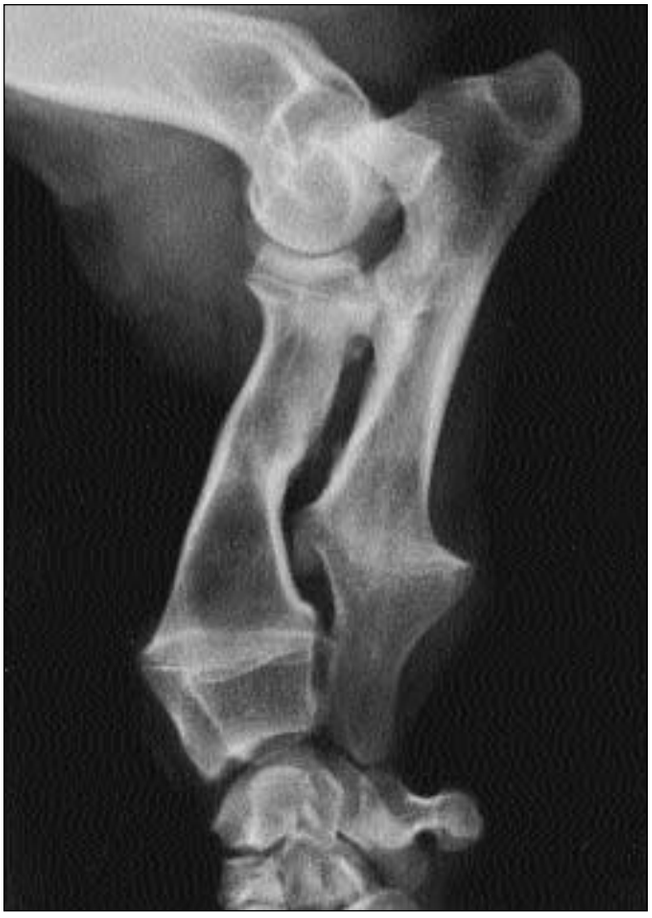

Figure 3A

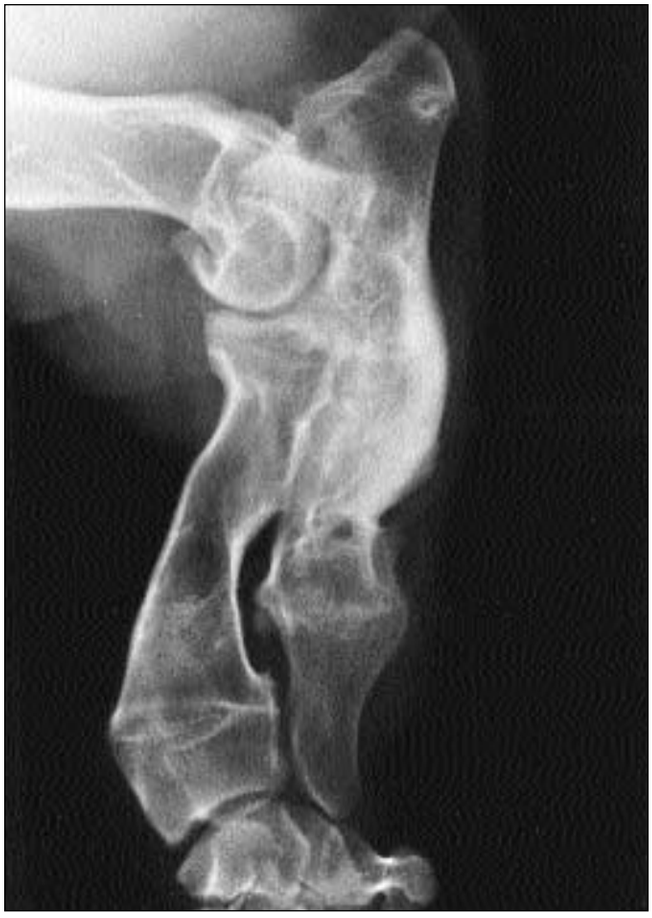

Figure 3C

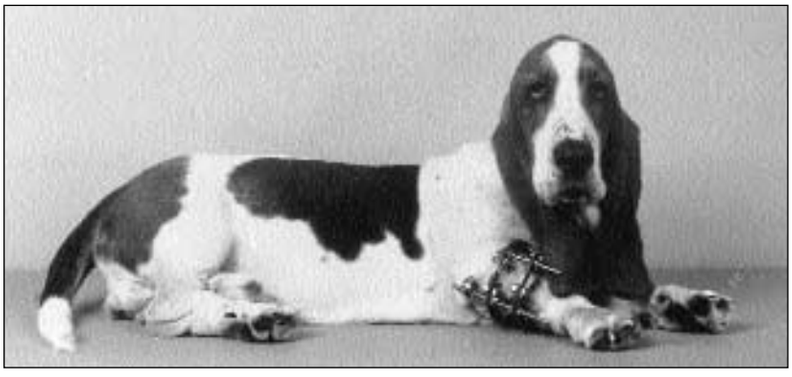

Figure 3B

Figure 3—*Lameness of the right front leg was revealed in a Basset Hound from the age of 7 months onward. **(A)** Severe incongruity due to elbow incongruity is evident on this radiograph. Since the anconeal process had been united with the olecranon at 7 months of age, distractio cubiti developed. **(B)** Ulnectomy and elongation of the ulna with an Ilizarov external ring fixator caused clinical and radiologic improvement. **(C)** As seen on this radiograph, healing of the distraction side occurred in 4 weeks.*

ulnotomy or a partial ulnectomy (Figure 3). Henschel and Grull described desmotomy of the radioulnar ligament, just proximal to the dorsolateral aspect of the carpal joint to successfully treat distractio cubiti and reported that locomotion improved in most cases after 2 to 10 weeks.[7] They claim that desmotomy of this ligament decreases the shearing force in the elbow joint without being detrimental to carpal stability. The concomitant incongruity at the distal end of the radius and ulna causes carpal laxity and valgus deformation that is typical in these breeds. Although this may cause osteoarthrosis of the carpal joint at a later age, it usually does not cause significant clinical problems.

Diminished growth in the length of long bones may be seen in isolated cases or in litters of nonchondrodystrophic breeds. This type of chondrodysplasia has been described in Alaskan Malamutes,[8] Labrador Retrievers,[9] Newfoundlands, and other popular breeds. Radiographically, the distal growth plate abnormality can be appreciated only for a limited period (i.e., in dogs younger than 6 months of age). Comparison with normal littermates or dogs of the same breed and age can make

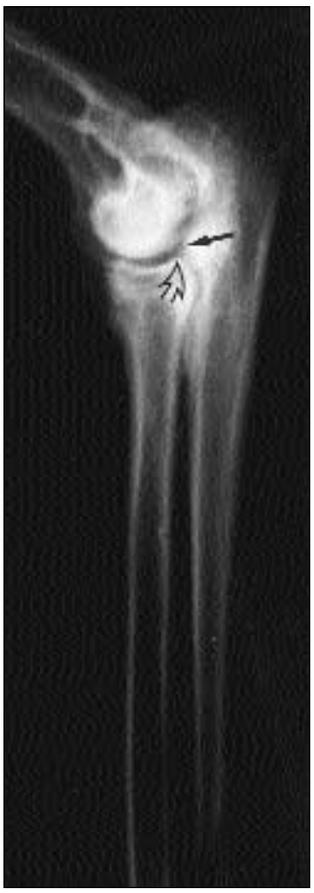

Figure 4A

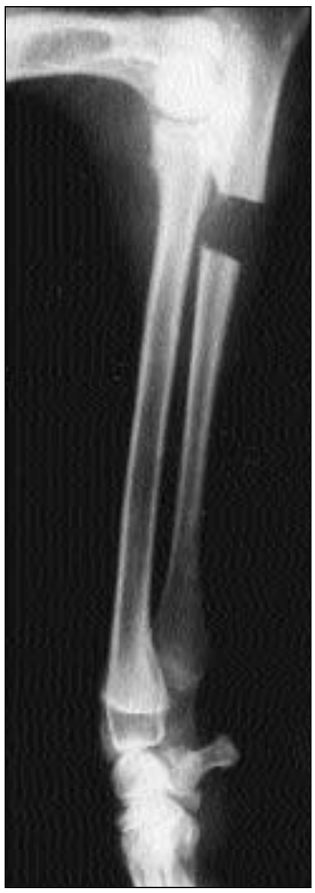

Figure 4B

Figure 4—*(A) Elbow incongruity in a 10-month-old Bernese Mountain Dog due to a short radius. The lateral coronoid process* (black solid arrow) *is more proximal than the radial head* (black curved arrow) *on the mediolateral extended view. (**B**) With partial ulnectomy, congruity of the elbow joint was reached spontaneously.*

abnormal growth in length evident on radiographs of the antebrachium. In some cases, chondrodysplasia can be seen with arthrosis in the bordering elbow and carpal joints as well as with other hematologic[8] and ophthalmologic[9] abnormalities, but these are not present in all cases.

ASYNCHRONOUS GROWTH IN RELATION TO FRAGMENTED CORONOID PROCESS

Elbow incongruity (EI) due to a short radius is frequently seen in Bernese Mountain Dogs, but other breeds such as Retrievers and Neapolitan Mastiffs may be affected. A random study in the Dutch Bernese Mountain Dog population revealed that 72% of the dogs had elbow incongruity. A longitudinal study by Bienz demonstrated that this incongruity may resolve spontaneously and is most likely genetic.[10] The joint surface supporting the humerus is decreased due to the short radius. This leads to an increased pressure on the remaining joint surface (the lateral and medial coronoid process) and may be the cause of the fragmentation of the coronoid process. This hypothesis is supported by the finding that elbow incongruity is seen in 72.6% of Bernese Mountain Dogs with a fragmented coronoid process (FCP), whereas only 6% had a FCP without elbow incongruity.[11] Based on the findings of Bienz,[10] we assumed that dogs with FCP but without elbow incongruity included dogs in which elbow incongruity resolved spontaneously. Elbow joint incongruity does not occur with FCP in all cases; in 12% of dogs, elbow incongruity was diagnosed without evidence of FCP.[11] Based on an owners' survey, we found that lameness was not present in all Bernese Mountain Dogs with either FCP or elbow incongruity but did affect all dogs with both disorders. In dogs with lameness due to FCP, the coronoid is removed. In cases with lameness due to FCP and severe incongruity, congruity is restored with partial ulnectomy in young dogs (Figure 4) and ulnotomy and elongation in mature dogs.

ASYNCHRONOUS GROWTH DUE TO TRAUMA

Trauma of the growth plate (i.e., Salter Harris type V) may cause a permanent or temporary decreased growth in length, either due to damage to the germinal cells in the growth plate of the distal ulna or to its vascular supply. The history may include trauma as the result of a fall or traffic accident (Figure 5). When the growth plates of the radius are not damaged, a growth discrepancy of the ulna may cause the radius curvus syndrome. This syndrome includes the following findings (Figures 5A and 5B):

- The ulnar styloid process is proximal to the accessory bone on mediolateral radiographs
- The lateral coronoid process is distal to the joint surface of the radial head
- The radius is curved on mediolateral radiographs, with a palmar cortex thicker and a dorsal cortex thinner than normal
- In more severe cases, a craniopalmar and valgus curving of the radius is present
- A valgus deformity of the distal extremity
- Abnormal development of the antebrachiocarpal joint

Various surgical techniques have been described (Table 2), depending on the expected growth of the animal, the severity of the curvature of the radius and the ability for spontaneous or surgical straightening, and the desire to restore or lose some of the length of the antebrachium. In growing dogs with radius curvus syndrome without (severe) curvature of the radius, a partial ulnectomy just proximal to the distal growth plate will remove the "bowstring effect" on the radius and allow it to straighten spontaneously, leading to clinical improvement. Correction of the alignment of the radius can be achieved by partial ulnectomy and an open or closed-wedge technique of the

Figure 5A

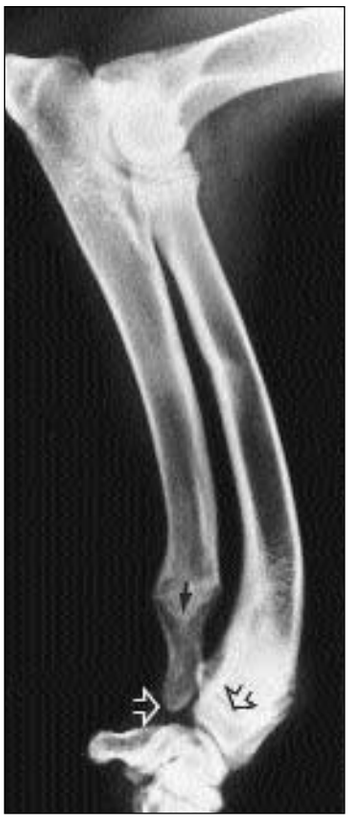

Figure 5B

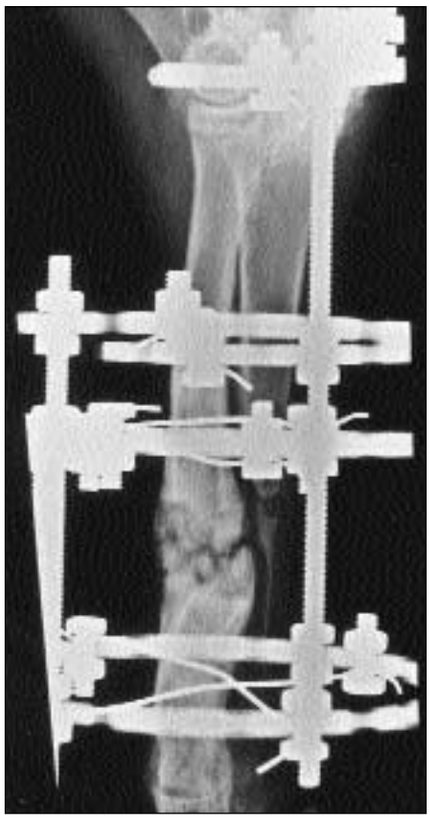

Figure 5C

Figure 5D

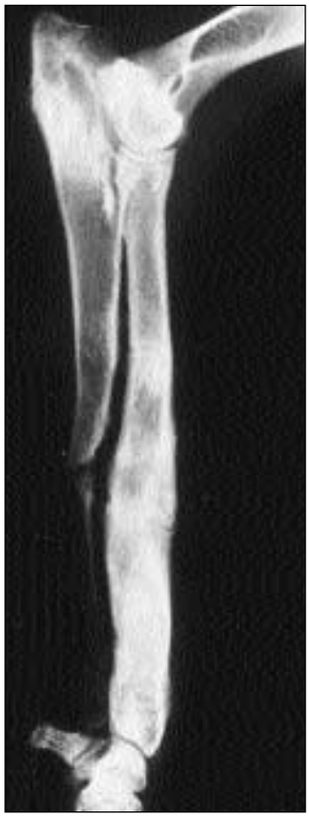

Figure 5E

Figure 5—*(**A**) A 5-month-old Retriever was presented because of right front leg lameness after jumping out of a car. Gradually, the right front leg revealed valgus deviation and a bowing of the radius. (**B**) The first radiograph was taken 4 weeks after the accident. Notice the V-shape of the growth plate of the distal ulna* (black solid arrow), *which is almost closed, in comparison with the open growth plate at the distal radius* (black open arrow). *Radiologically, signs of radius curvus syndrome can be noticed: thickening of the palmar cortex of the radius, very proximal location of the ulnar styloid process* (white open arrow) *when compared with the location of the accessory bone, abnormal radiocarpal joint, and elbow incongruity. (**C**) After partial ulnectomy and radiotomy, correction of the alignment was performed with an Ilizarov external ring fixator. (**D** and **E**) Eight weeks after surgical correction, a considerable clinical and radiologic improvement was seen, with only slight lameness of the right front leg.*

TABLE 2
Treatment Protocols for Radius Curvus Syndrome

Degree of Curvature	Surgical Technique	Stabilization Method
Slight curvus in young dog (+)	Ulnotomy	No stabilization / fixation (Robert Jones bandage for 5–7 days)
		Configuration of the osteotomy line
	Partial ulnectomy	No fixation (Robert Jones bandage for 5–7 days)
Mediolateral curvus (++)	Ulnotomy plus open wedge	Bone plate
		Kirschner–Ehmer splint or transfixation
		Ilizarov fixation
Three-dimensional curvature in growing dog (+++)	High ulnotomy plus corticotomy of radius	Ilizarov with "flag" on ulna; gradual lengthening
Three-dimensional curvature in mature dog (+++)	Ulnectomy and oblique radiotomy	Preoperative lengthening and fixation with external fixator

radius with fixation by bone plate or external fixator. More severe cases can be corrected with osteotomy of radius and ulna, followed by transfixation of the radius. The use of the Ilizarov external ring fixator (IERF) is preferred to correct the alignment of the radius as well as to restore the length of the ulna by applying an additional "flag" to the ulna.[12] If the growth in length of the radius is also disturbed, the IERF allows for distraction osteogenesis of the radius (Figure 5C). Distraction rates of 1 mm / day can be applied, allowing nerves, vessels, and muscles to keep pace with this distraction. The IERF is removed when the radiograph shows bony union of the distraction gap (Figure 5D).

In more advanced cases, the incongruity of the elbow joint may become so severe that the humerus is pushing against the anconeal process, which then ununites or leads to distractio cubiti. The UAP should be removed in chronic cases but may unite spontaneously in early cases in which the bow-string effect disappears by ulnotomy or partial ulnectomy. In cases of severe osteoarthrosis of the elbow joint or malformation of the radiocarpal joint, the prognosis for a complete restoration of locomotion is questionable.

ASYNCHRONOUS GROWTH DUE TO OVERSUPPLEMENTATION OR UNDERSUPPLEMENTATION

Three nutrition-related causes are clinically significant in the disturbed growth in length of the antebrachium. The occurrence of one or more of these depends on the region or country.

Excessive Calcium Intake

Various studies have proven that the disturbance in endochondral ossification characterized by impaired maturation of cartilaginous cells is related to high calcium intake. Radiographs of the distal ulna reveal a cartilage cone in the metaphyseal area in these cases. Longitudinal studies in Great Danes raised on a diet with 1.1% calcium on a dry matter basis (DM basis) demonstrated that a flattening or indentation of the distal ulnar metaphysis becomes visible on radiographs at the age of 15 to 21 weeks. Cones that do not exceed 20 to 25 mm in length will not cause clinical radius curvus syndrome and will spontaneously disappear at 6 months of age.[13] Longer cones seen in Great Danes raised on a diet with a calcium content of 3.3% on a DM basis coincide with severe, disturbed growth in length of the ulna, resulting in the development of the radius curvus syndrome (Figure 6).[3] The disturbed endochondral ossification may be due to direct or indirect (e.g., hormonal) action of calcium on these cartilage cells or possibly a disturbance of the vascular supply. Periosteal stripping and ligation of the ulnar nutrient artery[14] may cause a very similar disease. In cases of excessive calcium intake, occlusion of blood vessels may cause edema formation inside the medulla and between the periosteum and cortex, which on palpation results in severe lameness and a painful reaction. This shifting lameness (also known as panosteitis eosinophilia or enostosis) is often seen in conjunction with high calcium intake and retained cartilage cones.[15] Although small, retained cartilage cones are not of clinical significance, they are not considered normal. It has been debated whether 1.1% cal-

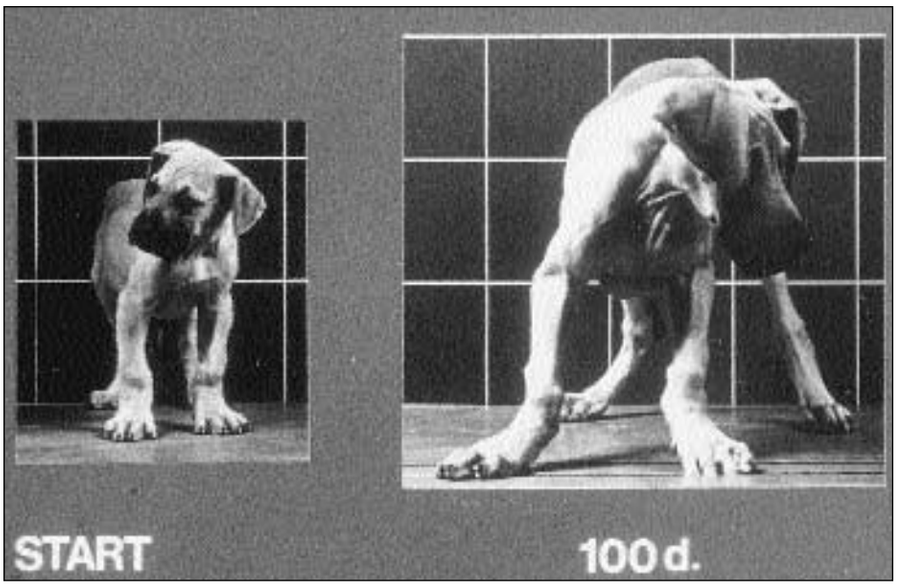

Figure 6A

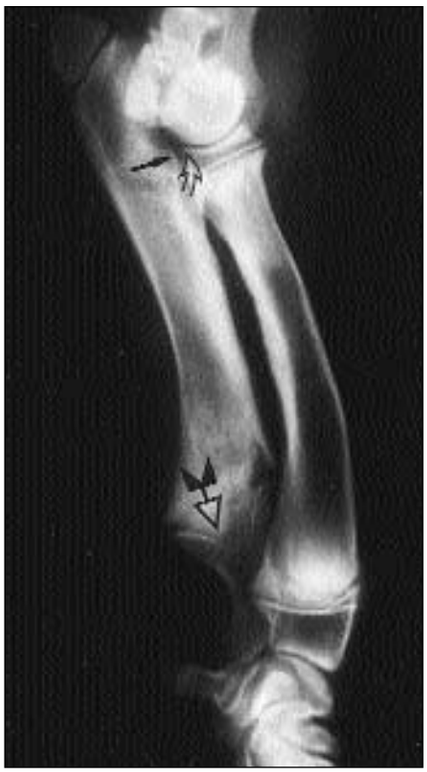

Figure 6B

Figure 6—*(A) This Great Dane was raised on a high-calcium diet (3.3% Ca on a DM basis) from the age of 6 weeks. (**B**) At the age of 4.5 months, this radiograph of the radius and ulna shows a normal V-shape of the epiphyseal side of the distal ulnar growth plate* (large black arrow) *but severe irregularities at the metaphyseal side, due to retained cartilage cones. This causes a disturbance of the growth in length of the ulna, and thus radius curvus syndrome with valgus deformation, a curved radius, and incongruity of the elbow joint* (small black arrows)*, eventually resulting in an ununited anconeal process in young dogs.*

cium on a DM basis in the daily ration is too high for large-breed dogs during growth.[13]

Excessive Nutrient Intake

The caloric requirements of growing puppies are three times higher than those of adult dogs until they reach 50% of the adult weight. Thereafter, the caloric requirements are two times as high until maturity. The discrepancy between puppy weight and adult weight is more evident in young dogs of giant breeds than in young dogs of miniature breeds. Giant-breed puppies also need additional calories for a longer period than do puppies of miniature breeds and will therefore eat extra food to obtain these required calories. As a consequence, they also "take in" extra calcium. Studies have demonstrated that young dogs of giant breeds are unable not to absorb this extra calcium effectively; calcium intake of three times the normal requirements causes almost a 2.5 times higher absorption of calcium.[16,17] In addition to enostosis and radius curvus syndromes, more frequent and severe cases of joint cartilage osteochondrosis were seen in groups of dogs with high food intake [16] and high calcium intake.[17] Radius curvus syndrome due to high calcium intake is seen especially in young dogs of large breeds (Table 3). As a result, commercially available canine diets with a lower calcium:energy ratio have been formulated for young large-breed dogs.

Excessive calcium intake may be seen in puppies that are overfed a balanced diet with a standard calcium content (i.e., a calcium content of 1.2%–1.5% on a DM basis) and/or fed a diet supplemented with bone meal or calcium salts. In such cases, an excessive amount of calcium is absorbed and routed to the skeleton, causing a variety of skeletal abnormalities such as the radius curvus syn-

TABLE 3

Characteristics of Dogs with Dietary-Related Radius Curvus Syndrome

Breed
- Bouvier des Flandres
- Briard
- Great Dane
- Greyhound
- Irish Wolfhound
- Great Pyrenees

Gender
- Predominantly in males

Age
- ≥4 months (average = 5.5 months)

Weight at Referral
- 26 kg (adult weight >35 kg)

Bilateral Growth Disturbance
- All cases

TABLE 4

Characteristics of Dogs with Hypertrophic Osteodystrophy

- Fast-growing breeds (Great Danes, Collies, Retrievers, German Shepherd Dogs, Saint Bernards)
- 2 to 8 months of age (most common in dogs 3 to 4 months of age)
- History of gastrointestinal or respiratory disease
- Reluctance to stand
- Extreme pain on bone palpation
- Biphasic fever
- Swollen metaphyseal areas
- Hematologic and biochemical findings are not conclusive
- Radiopaque line seen at 2 to 3 mm from growth plates on radiographs
- mRNA of distemper virus in bone cells in radiopaque area
- Death, remission, or complete recovery

drome (Figure 6), enostosis, and osteochondrosis of joint cartilage. When these dogs have an uncoordinated gait as well, a thorough neurologic investigation should precede corrective surgery, since cervical compression of the spinal cord may occur concomitantly (i.e., the wobbler syndrome in young giant-breed dogs).[16,17]

In most cases, corrective surgery of the radius curvus syndrome involves both front legs because this generalized disease causes bilateral growth disturbance. If osteochondritis dissecans, abnormal rear leg alignment, and/or wobbler syndrome are present, owners may hesitate to have all these diseases treated.

Vitamin D Deficiency

In cases of hypovitaminosis D, endochondral ossification will be disturbed by a lack of matrix mineralization, resulting in postponed cell death (Figure 1). As a consequence, growth plates will be abnormally wide on radiographs. Research in dogs and cats revealed that these carnivores are unable to synthesize vitamin D in their skin by sunlight radiation, counter to what occurs in herbivores like horses, cows, and sheep and in omnivores such as humans, rats, and pigs.[18] Thus dogs and cats depend on dietary intake of vitamin D, which is present in animal fat and is abundant in commercial pet food diets.

In many cases of rickets (hypovitaminosis D), the history reveals that the owner fed the animal only lean meat (and thus a diet free of calcium, fat, and vitamin D).

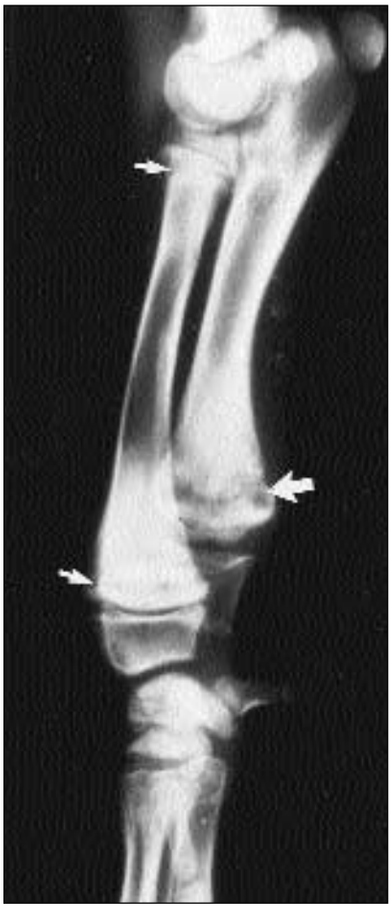

Figure 7A

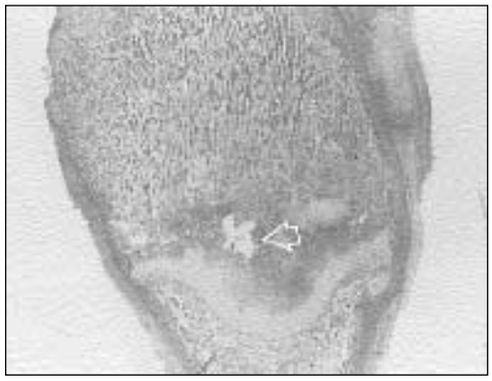

Figure 7B

Figure 7—*(**A**) Mediolateral radiograph of a 4-month-old Retriever that was presented with severe bone pain, high body temperature, and reluctance to stand. This radiograph of the antebrachium shows radiopaque lines* (arrows) *parallel to and 2 to 3 mm from the growth plates, which are pathognomonic for hypertrophic osteodystrophy. (**B**) Pathology of the distal ulna shows a zone with debris, inflammation, and bone cells* (arrow) *at some distance from the growth plate. (Courtesy of Dr. I Van der Gaag, Utrecht University.) (**C**) At a later stage, thickening due to periosteal new bone formation* (arrow) *can be seen on this radiograph and can coincide with signs of radius curvus syndrome.*

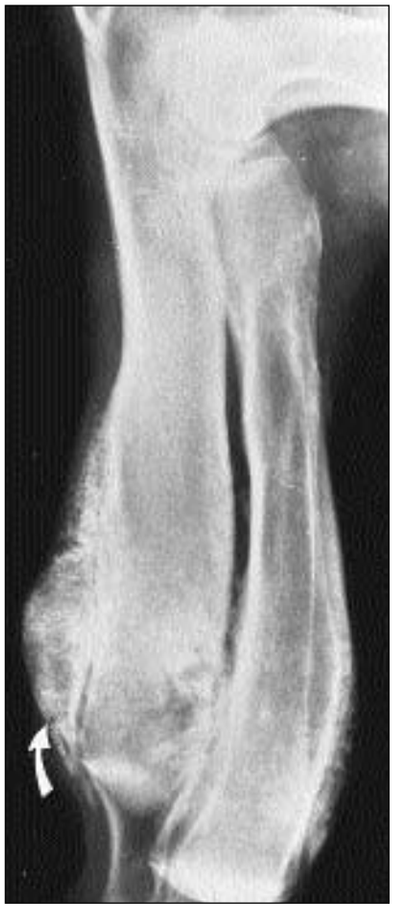

Figure 7C

Research dogs fed a diet meeting nutritional requirements but low in vitamin D have been found to develop radiologic, histologic, and clinical signs of rickets, including increased width of growth plates. Varus deformation can be more pronounced than valgus deformation, due to bowing of the weakened long bones. The thickening in the metaphyseal area of the distal radius due to abundant soft tissue formation rather than bone can be very pronounced. The abnormalities begin to heal after 3 weeks of feeding a complete and balanced commercial canine diet. Thus commercial dog food can resolve and also prevent hypovitaminosis D in dogs.[19]

HYPERTROPHIC OSTEODYSTROPHY

Hypertrophic osteodystrophy (HOD, metaphyseal osteopathy, or metaphyseal dysplasia) is a disease that may lead to valgus deformation of both front feet. Although also known as Möller–Barlow's disease (or canine scurvy), this disease is not caused by hypovitaminosis C. Vitamin C is of no benefit in the prevention or treatment of this disorder. In addition, the collagen degeneration in tendons and blood vessels seen in guinea pigs with hypovitaminosis C is not present in dogs with HOD. The growth in length of all long bones is disturbed; growth plates with the largest growth potential are most commonly affected. The disease is seen particularly in large-breed dogs, but HOD in miniature dogs has been reported.[20] HOD is described in studies of Great Danes fed a high-calcium diet,[16,17] but its association with dietary abnormalities is inconsistent.[21]

Clinical signs include depression and extreme pain on palpation of the swollen metaphyseal areas (Table 4). Littermates may also be affected. Although the dog may have been vaccinated against distemper, the history often includes periods of diarrhea and/or respiratory tract disease. High fever, often characterized as biphasic, is present in the acute phase of the disease (Figure 7). On radiographs (Figure 7A), a radiopaque line parallel to and at 2 to 3 mm from the growth plates (Figure 7B) can be seen as a pathognomonic sign in the subacute phase.[15]

Mee and colleagues[22] have demonstrated the presence of RNA and mRNA of canine distemper virus (CDV) in osteoblasts, osteocytes, and bone marrow cells of dogs infected with this virus. Osteoblasts and osteoclasts within the affected metaphyses of dogs with HOD also contained viral mRNA,[22] and recently a paramyxovirus was isolated from one of our patients that died in the course of HOD. These findings support those of Grondalen in which the blood of dogs with HOD caused clinical CDV infection (but not HOD) in recipient dogs.[23] It should be noted that some authors describe a similar disease (i.e., Paget's disease) in humans with distemper-like viral occlusions (not measles) in the bone cells; however, the existence of zoonotic infection has not been proven to date.[24, 25]

Some dogs will die of shock (pain or viremia?); others will survive and show remission of clinical signs. An adequate supportive and NSAID regimen may result in complete recovery. Based on the possible viral etiology, corticosteroids may be contraindicated. These animals need intensive care, including physical support during urination and defecation, frequent repositioning to prevent decubitus, and a balanced recovery diet rich in energy. Spontaneous remission of clinical signs or relapses may occur. Diaphyseal deformities can resolve after several weeks, although long bones may remain thickened due to periosteal new bone formation[15,21] (Figure 7C).

REFERENCES

1. Riser WH, Shirer JF: Normal and abnormal growth of the distal foreleg in large and giant dogs. *J Am Vet Radiol Soc* 6:50–64, 1965.
2. Yonamine H, Ogi N, Ishikawa T, Ichiki Y: Radiographic studies on skeletal growth of the pectoral limb of the beagle. *Jpn J Vet Sci* 422:417–425, 1980.
3. Voorhout G, Hazewinkel HAW: A radiographic study on the development of the antebrachium in Great Dane pups on different calcium intakes. *Vet Radiol* 28:152–157, 1987.
4. Henschel E: Zum Längenwachstum von Humerus, Os Femoris and Tibia des Hundes. *Kleintierpraxis* 18:100–103, 1973.
5. Olson NC, Carrig CB, Brinker WO: Asynchronous growth of the canine radius and ulna: Effects of retardation of longitudinal growth of the radius. *Am J Vet Res* 40:351, 1979.
6. Carrig CB, Morgan JP, Pool RR: Effects of asynchronous growth of the radius and ulna of the canine elbow joint following experimental retardation of longitudinal growth of the ulna. *JAAHA* 9:496–567, 1975.
7. Henschel E, Grull F: Zur Therapie der Distractio cubiti beim Bassethound. *Kleintierpraxis* 20:267–271, 1975.
8. Fletch SM, Smart ME, Pennock PW, Subden RE: Clinical and pathological features of chondrodysplasia (dwarfism) in the Alaskan Malamute. *JAAHA* 162:357–361, 1973.
9. Carrig CB, MacMillan A, Brundage S, et al.: Retinal dysplasia associated with skeletal abnormalities in Labrador Retrievers. *JAVMA* 170:49–57, 1977.
10. Bienz HA: Klinische und Radiologische Untersuchungen über den Fragmentierten Processus Coronoideus Medialis Im Ellenbogengelenk des Berner Sennenhundes und der andere Sennenhunde Rassen. Inaug Dissertation Zürich (Switzerland) 1985.
11. Hazewinkel HAW, Meij BP, Nap RC, et al: Radiographic views for elbow dysplasia screening in Bernese Mountain Dogs. Annual Meeting International Elbow Working Group, Constance, Germany, 1995, pp 29–32.
12. Latte Y: Application de la méthode d'Ilizarov en chirurgie orthopédique vétérinaire. *Prat Med Chirurg Anim Compag* 29:7–32, 1994.
13. Voorhout G, Nap RC, Hazewinkel HAW: A radiographic study on the development of the antebrachium in Great Dane pups, raised under standardized conditions. *Vet Radiol Ultrasound* 35:271–276, 1994.
14. Alvarado MS: The effects of nutrient artery ligation on the development of the distal ulnar metaphysis of the dog. Thesis in comparative medical sciences, University of Pennsylvania, 1976.
15. Hazewinkel HAW: Nutrition in orthopedics, in Bojrab MJ (ed): *Disease Mechanisms in Small Animal Surgery*, ed 2. Philadelphia Lea & Febiger, 1993, pp 1119–1128.
16. Hedhammar A, Wu FM, Krook L, et al: Overnutrition and skeletal disease: An experimental study in growing Great Danes. *Cornell Vet* 64(Suppl 5):11–160, 1974.
17. Hazewinkel HAW, Goedegebuure SA, Poulos PWE, Wolvekamp WTC: Influences of chronic calcium excess on the skeletal development of growing Great Danes. *JAAHA* 21:377–391, 1985.

18. How KL, Hazewinkel HAW, Mol JA: Dietary vitamin D dependence of cat and dog due to inadequate cutaneous synthesis of vitamin D. *Gen Comp Endocrinol* 96:12–18, 1994.
19. Hazewinkel HAW, How KL, Bosch R, et al: Inadequate photosynthesis of vitamin D in dogs, in Edney ATB (ed): *Nutrition, Malnutrition and Dietetics in the Dog and Cat.* UK, Waltham Centre Press, 1987, pp 66–68.
20. Appeldoorn AM, Schrauwen EMJ: Hypertrophic osteodystrophy in a toy breed. *Vet Comp Orthop Trauma* 8:210–212, 1995.
21. Bellah JR: Hypertrophic osteodystrophy, in Bojrab MJ (ed): *Disease Mechanisms in Small Animal Surgery,* ed 2. Philadelphia, Lea & Febiger, 1993, pp 858–864.
22. Mee AP, Gorden MT, May C, et al: Canine distemper virus transcripts detected in the bone cells of dogs with metaphyseal osteopathy. *Bone* 14:59–67, 1993.
23. Grondalen J: Letter to the editor. *J Small Anim Pract* 20:124, 1979.
24. Barker DJP, Detheridge FM: Dogs and Paget's disease. *Lancet* 2:1245, 1985.
25. Gorden MT, Bell SC, Mee AP, et al: Prevalence of canine distemper antibodies in the pagetic population. *J Med Virol* 40:313–317, 1993.

Nutritional Influences on Skeletal Growth of the Large-Breed Puppy

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NUTRITION AND SKELETAL DISEASE

One of the truly remarkable characteristics of the canine is the variation of mature body weights across breeds. The official American Kennel Club breed descriptions indicate mature body weights ranging from less than 6 lb for the Chihuahua and Pomeranian to greater than 150 lb for the Newfoundland,[1] a 25-fold difference. In practice, however, the range in mature size often greatly exceeds these reference boundaries. Application of genetic selection pressure favoring a large mature body size within specific canine breeds concurrently selects genotypes with a remarkable ability for a rapid rate of growth. For example, 20 years ago a typical Great Dane grew to approximately 130 lb mature body weight by 12 to 18 months of age. Today, mature body weights of 180 lb or more during that same 12- to 18-month growth period are often achievable. Unfortunately, it is often not fully appreciated that the genetic propensity for rapid growth rate, co-selected with mature body size, is associated with potentially negative consequences. It is well documented that the incidence of skeletal disease, including osteochondroses, hypertrophic osteodystrophy and hip dysplasia, is markedly increased if management practices are such that this maximal genetic potential for growth rate is realized. Of particular importance is the influence of nutritional management on growth rate and skeletal disease. Three dietary components—the dietary concentrations of energy, protein, and calcium (and phosphorus)—have been implicated as primary contributors to an increased incidence of skeletal disease in the growing large-breed puppy.

Hedhammer and colleagues[2] investigated the issue of dietary energy intake by feeding growing Great Dane puppies either ad libitum or 66% of ad libitum intake and observed a dramatic increase in the incidence of skeletal pathology in puppies consuming the higher level of intake. Apparently, the high plane of nutrition effectively supported the genetic potential for rapid growth and predisposed the large-breed puppy to the development of skeletal disease. Dammrich[3] provided further support for this growth rate response by feeding Great Danes either ad libitum or restricted (60% to 70% of ad libitum) intake from weaning through 6 months of age. Maximal growth (ad libitum intake) resulted in subchondral spongiosa, which was less dense and weaker per unit area. The resulting osteopenia and biomechanically weak subchondral bone could not adequately support the articular cartilage of the joint. In addition, the increased growth rate of ad libitum feeding more rapidly subjected the joint surface to stresses due to increased body mass. Clearly, a high level of energy intake promotes an excessive rate of growth in the large-breed puppy and increases the potential for skeletal disease.

Three dietary components—the dietary concentrations of energy, protein, and calcium (and phosphorus)—have been implicated as primary contributors to an increased incidence of skeletal disease in the growing large-breed puppy.

Dietary protein level has also been implicated as influencing the incidence and severity of skeletal disease in the growing large-breed puppy. Controlled research, however, does not support the hypothesis of an association between high dietary protein intake and skeletal abnormalities. Nap and colleagues[4] fed Great Dane puppies isocaloric diets that provided a broad range of dietary protein (31.6%, 23.1%, or 14.6% protein) from weaning for 18 weeks and observed no treatment effect on calcium metabolism or skeletal development.

In contrast to dietary protein, calcium concentration has been demonstrated to have a significant effect on

development, morphology, and pathology of the skeleton in the large-breed puppy. Hazewinkel and colleagues[5] and Goedegebuure and Hazewinkel[6] evaluated the effect of feeding either a typical calcium diet (1.10% Ca/0.90% P) or a high-calcium diet (3.30% Ca/0.90% P) to Great Dane puppies from weaning through 6 months of age. The effect of the high-calcium diet on endocrine status (hypercalcemia, hypophosphatemia, less active parathyroid glands, increased activity of thyroid C cells), skeletal development (increased osteoblasts, decreased osteoclasts, decreased osteoclast activity, more retained cartilage cones, increased bone mineral mass, delayed bone remodeling), and skeletal disease (increased radiographic irregularities, more osteochondritic lesions) clearly demonstrated the negative impact of excess dietary calcium on skeletal health of the large-breed puppy. This conclusion was further supported by the finding that the large-breed puppy was ineffective in reducing intestinal calcium absorption when provided a high-calcium diet and was therefore unable to protect itself from a chronic high dietary calcium intake.[7] In contrast, intestinal calcium absorption was increased to greater than 90% of calcium intake when a low-calcium diet was consumed.

In contrast to dietary protein, calcium concentration has been demonstrated to have a significant effect on development, morphology, and pathology of the skeleton in the large-breed puppy.

CURRENT RESEARCH

Research published to date clearly documents that (1) maximal growth rate supported by elevated energy intake increases the incidence of skeletal disease, (2) dietary protein level has little influence on the incidence of skeletal disease, and (3) high dietary calcium concentration interferes with normal skeletal development and promotes skeletal pathology in the large-breed puppy. Nevertheless, data on which to base specific dietary recommendations for dietary energy and calcium concentrations for the large-breed puppy are lacking. A comprehensive research effort is underway to provide this essential information. In brief, a total of 36 Great Dane puppies were assigned to three treatment diets differing in calcium and phosphorus concentration: 0.48% Ca/0.40% P (LC), 0.80% Ca/0.67% P (MC), and 2.70% Ca/2.20% P (HC). These levels of calcium and phosphorus were selected to allow evaluation of the hypothesis that a calcium and phosphorus concentration lower than found in typical premium puppy diets (e.g., 1.20% Ca/1.00% P) would enhance skeletal health when provided in a dietary matrix of reduced energy density designed to manage growth rate and decrease the slope of the growth curve. To achieve the reduced energy density, all diets were formulated to contain 14% fat, compared to 20% to 21% fat in the typical

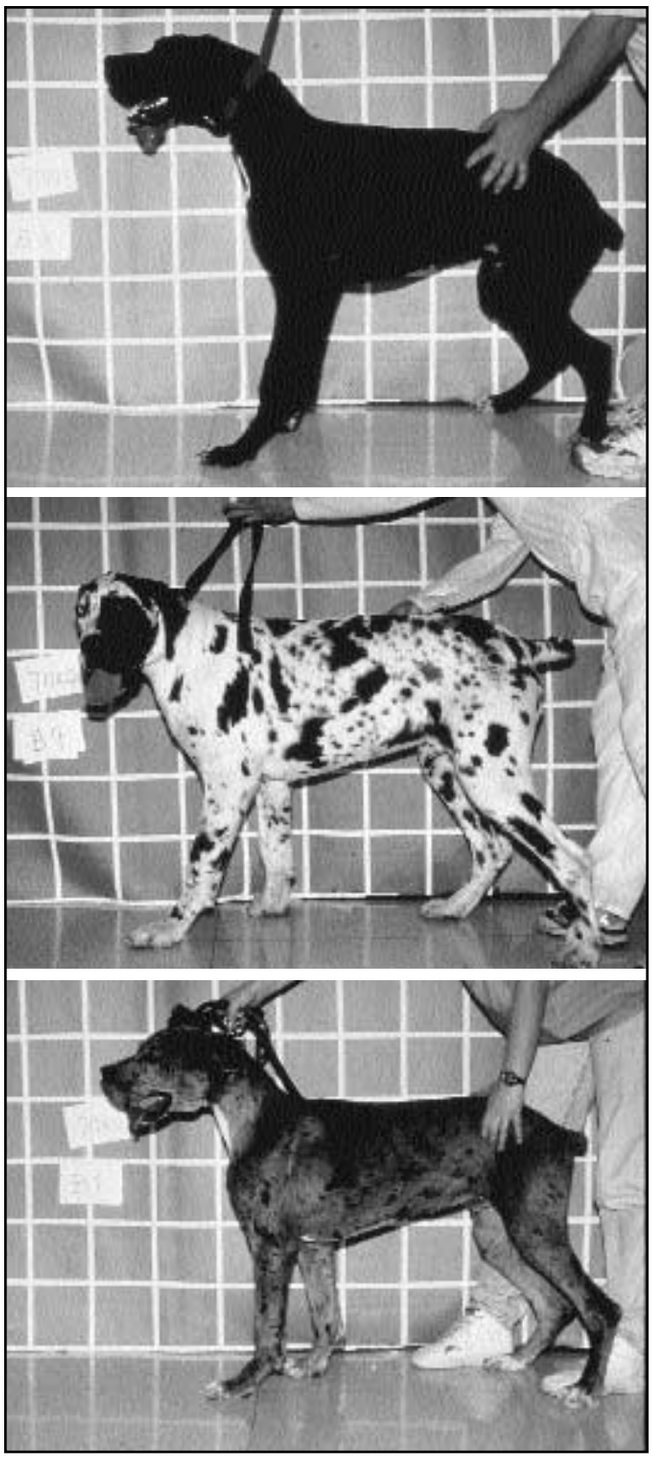

Figure 1—*Relative body size of Great Danes consuming a diet containing either high calcium (HC;* top*), medium calcium (MC;* middle*) or low calcium (LC;* bottom*).*

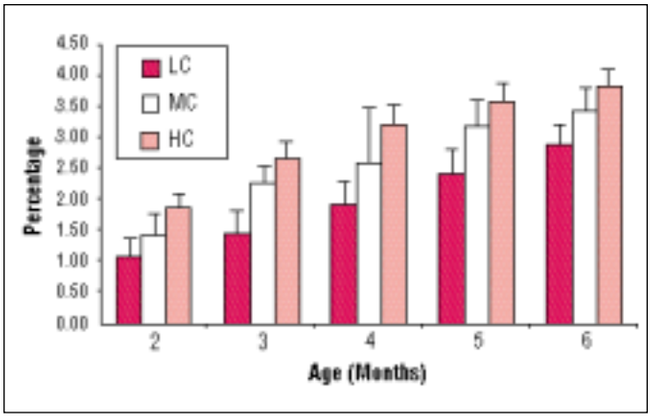

Figure 2—*Percentage of bone mineral content of Great Danes consuming a diet containing high calcium (HC), medium calcium (MC), or low calcium (LC).*

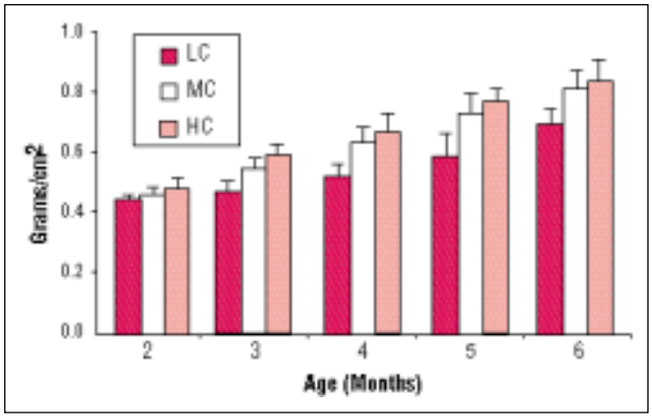

Figure 3—*Bone mineral density of Great Danes consuming a diet containing high calcium (HC), medium calcium (MC), or low calcium (LC).*

premium puppy food. Dietary protein concentration was also lowered to 26% to maintain an appropriate dietary protein:energy ratio. The three diets were fed to growing Great Danes from preweaning through 18 months of age. Briefly reported here are the results published to date for body weight, body composition, bone mineral density, and skeletal integrity (based on radiographic and orthopedic examinations).

Puppies consuming the MC diet grew more rapidly and had increased body weights relative to puppies fed the HC and LC diets (Figure 1).[8] Nevertheless, no differences in bone lengths were apparent across treatment groups by 6 months of age. Lean body mass and fat tissue were equal across treatments at weaning, but by 4 months of age, puppies fed the MC diet had a higher percentage of fat tissue and lower lean body mass as compared to the other two treatment diets.[9] Differences in bone mineral content (BMC), as measured by dual energy x-ray absorptiometry (DEXA), were evident by 8 weeks of age and continued to diverge through 6 months of age.[9] BMC, as a percentage of body weight, was 1.07% (LC), 1.42% (MC), and 1.85% (HC) at 2 months of age and increased to 2.91% (LC), 3.45% (MC), and 3.84% (HC) by 6 months of age (Figure 2). Furthermore, bone mineral density (BMD) values were likewise different by 8 weeks of age: 0.442 g/cm^2 (LC), 0.456 g/cm^2 (MC), and 0.482 g/cm^2 (HC; Figure 3).[10] Differences in BMD across treatment groups continued through 6 months of age, after which time the magnitude of the response began to decrease. By 12 months of age, BMD values were 0.976 g/cm^2 (LC), 1.046 g/cm^2 (MC), and 1.054 g/cm^2 (HC).[10] Orthopedic examination revealed that 6 of the 15 dogs consuming the HC diet exhibited clinical signs of lameness through the first 6 months of the study.[11] Three of these dogs exhibited clinical signs of hypertrophic osteodystrophy. Conformation of dogs fed the high mineral diet was considered inferior to those receiving the other diets.

PRACTICAL IMPLICATIONS

The results of this ongoing research clearly document that the unique nutritional demands of the large-breed puppy are best provided by a dietary matrix containing 26% protein (high quality, animal-based source), 14% fat, 0.80% calcium, and 0.67% phosphorus. Support for this recommendation is based on the following research observations:

The results of ongoing research clearly document that the unique nutritional demands of the large-breed puppy are best provided by a dietary matrix containing 26% protein, 14% fat, 0.80% calcium, and 0.67% phosphorus.

- A reduced dietary energy density, which is typically found in puppy foods, provides for easier management of growth rate and will moderately restrict the growth rate relative to the genetic potential. Mature body size is genetically determined and will simply be attained at a slightly older age with a more gradual growth rate.
- The altered body composition of puppies fed the MC diet may reflect that 0.80% calcium is a more appropriate level of supplementation, supporting the modestly restricted growth velocity resulting from a lower energy density. Puppies fed the MC diet may therefore be placed on a relatively more advanced position on the growth curve compared to puppies fed the HC and LC diets, while maintaining a more gradual growth curve relative to the genetic potential.
- BMC and BMD were lower in puppies fed the LC diet

(0.48% calcium), indicating an inadequate level of supplementation in this dietary matrix. Furthermore, BMC and BMD responses occurred very early in the growth phase, indicating the need to address the nutritional requirements of the large-breed puppy immediately after weaning.

- Orthopedic examination revealed a higher incidence of abnormalities in the puppies consuming the HC diet (2.70% calcium), indicative of calcium oversupplementation in this dietary matrix containing a reduced energy density.

REFERENCES

1. American Kennel Club: *The Complete Dog Book: Official Publication of the American Kennel Club*, ed 18. New York, Macmillan, 1992.
2. Hedhammer A, Wu F, Krook L, et al: Overnutrition and skeletal disease. An experimental study in Great Dane dogs. *Cornell Vet* 64(Suppl 1):1–160, 1974.
3. Dammrich K: Relationship between nutrition and bone growth in large and giant dogs. *J Nutr* 121:S114–S121, 1991.
4. Nap RC, Hazewinkel HAW, Voorhout G, et al: Growth and skeletal development in Great Dane pups fed different levels of protein intake. *J Nutr* 121:S107–S113, 1991.
5. Hazewinkel HAW, Goedegebuure SA, Poulos PW, Wolvekamp WThC: Influences of chronic calcium excess on the skeletal development of growing Great Danes. *JAVMA* 21:377–391, 1985.
6. Goedegebuure SA, Hazewinkel HAW: Morphological findings in young dogs chronically fed a diet containing excess calcium. *Vet Pathol* 23:594–605, 1986.
7. Hazewinkel HAW, Van Den Brom WE, Van 'T Klooster ATh, et al: Calcium metabolism in Great Dane dogs fed diets with various calcium and phosphorus levels. *J Nutr* 121:S99–S106, 1991.
8. Goodman SA, Montgomery RD, Lauten SD, et al: Orthopedic observations in Great Dane puppies fed diets varying in calcium and phosphorus content—A preliminary report. *Vet Comp Orthopaed Trauma* 10:75, 1997.
9. Lauten SD, Brawner WR, Goodman SA, et al: Changes in body composition of growing Great Danes fed diets varying in Ca and P level evaluated by dual energy x-ray absorptiometry. ACVR Proceedings, 1-8 (Abstract), 1996.
10. Lauten SD, Brawner WR, Goodman SA, et al: Dual energy x-ray absorptiometry measurement of body composition and skeletal development in giant breed dogs fed diets differing in calcium and phosphorus. *FASEB J* 11:A388, 1997.
11. Brawner WR, Hathcock JT, Goodman SA, et al: Clinical and radiographic observations of growing Great Danes fed diets varying in mineral content: A preliminary report. ACVR Proceedings, 1-9 (Abstract), 1996.

Diagnostic Imaging of Skeletal Growth and Disorders

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Dogs experience rapid growth and development from birth to skeletal maturity in the first 12 to 18 months of life. There is a tremendous range of size and conformation among breeds of dogs. The process of skeletal development and factors affecting that process have been studied in only a few breeds, and even in those breeds, many questions remain unanswered. Giant-breed dogs have been studied most often because they seem particularly susceptible to developmental and orthopedic abnormalities such as osteochondrosis, hypertrophic osteodystrophy, hip dysplasia, and others.[1–4] Developmental skeletal diseases are relatively uncommon in small breeds, common in large breeds, and even more common in the giant breeds.

Rapid growth of giant-breed dogs should intuitively place stress on the development of the skeletal system, and it is interesting to compare the development of giant-breed dogs to humans. A person born at 5 to 10 pounds develops to skeletal maturity at a weight of 100 to 150 pounds in 16 to 18 years. A giant-breed dog born at less than 2 pounds develops to skeletal maturity at a weight of 100 to 150 pounds in 12 to 18 months. Consequently, it should not be surprising that giant-breed dogs are susceptible to skeletal abnormalities and that the adverse effects of poor nutrition, metabolic disease, or other illnesses are magnified in the skeletal development of these dogs and even in the smaller breeds. Because of the rapid growth of dogs and the potential deleterious effects of abnormal skeletal development, continued effort to more fully understand the process of canine growth and the effects of diet, husbandry, and health maintenance on that process is critical.

The investigation of growth and development in dogs requires study of the animal over a period of months.[5] Physical examination and observation of size, conformation, and locomotion are often complemented by biochemical analysis of circulating enzymes, electrolytes, and hormones.[6] Ultimately, the bones themselves must be studied. Detailed assessment of cellular growth patterns can be obtained through histopathologic examination, but diagnostic material usually can be obtained only from postmortem specimens.[1,2] Modern diagnostic imaging methods provide the opportunity for noninvasive evaluation of the skeletal system with no effect on growth and development.

A giant-breed dog born at less than 2 pounds develops to skeletal maturity at a weight of 100 to 150 pounds in 12 to 18 months.

Diagnostic imaging techniques provide a number of distinct advantages in the study of growth and development that require multiple examinations over a period of months. Diagnostic imaging techniques typically do not affect the growth process in any way and may be repeated many times in the same animal. Although the information generated by these techniques does not allow assessment of growth at the cellular or molecular level, the array of methods now available does allow portrayal with excellent resolution of the morphology of musculoskeletal structures. A list of currently available diagnostic imaging systems is presented in Table 1. Many of these systems are expensive to purchase and maintain and therefore may be available only at universities, research centers, or large specialty practices. It is important to realize that no system produces images that provide optimal or inclusive information in every situation. Each imaging system has its own unique characteristics, and it is important to be aware of the relative strengths and weaknesses of each in order to select the most appropriate system or combination of systems in a given clinical or research setting. Diagnostic imaging techniques should not be compared on an either/or basis but more appropriately with regard to the

TABLE 1
Imaging Systems Available for Evaluation of the Skeletal System

Imaging System	Comments
Conventional x-ray machines	Direct x-ray film imaging, including contrast radiography such as myelography and arthrography
Digital x-ray systems	Conventional x-ray tube with digital image receptors
Cabinet x-ray systems	Specimen radiography and microangiography of excised thin sections only
Computed tomography (CT) systems	Cross-section x-ray imaging,[10,11] including quantitative CT (QCT)[16] for assessment of bone density
Magnetic resonance imaging (MRI) systems	Cross-section imaging using a strong magnetic field and radiofrequency waves[12]
Gamma scintillation cameras	Scintigraphy (radionuclide imaging) using injected radiopharmaceuticals[14,15]
Diagnostic ultrasound machines	Ultrasonography useful for soft tissues and surface features of bones; infrequently employed in orthopedics
Dual energy x-ray absorptiometers (DEXA)	Quantitative assessment of bone mineral density and body composition (fat, lean tissue, bone mineral)[13]

additional information that can be gained by their complementary strengths.[7,8]

RADIOGRAPHY

Conventional x-ray machines and x-ray film remain the most useful and cost-effective imaging system in most applications. X-ray film images provide excellent resolution of bone detail and usually allow visualization of an entire bone or group of bones on one image. Survey radiographs allow excellent assessment of the size, shape, contour, and opacity of bone and the relationship of adjacent and articulating bones.[7–9] Limitations include the fact that images are two-dimensional and often superimposition of structures inhibits clear visualization. For this reason, it is essential that at least two radiographic projections (made at 90˚ angles) be made as part of every examination. Oblique radiographic projections and other special techniques also can be used to overcome the difficulties of superimposition. Figure 1 is an example of a survey (noncontrast) radiograph of the scapulohumeral joint in a dog with osteochondrosis of the humeral head.

COMPUTED TOMOGRAPHY

Computed tomography (CT) is a cross-sectional imaging technique based on x-ray absorption. Narrow, "pencil" beams of x-ray are passed through the patient in multiple directions, and x-ray detectors on the opposite side of the patient determine the percentage of the absorbed x-ray beam. The information from these detectors is manipulated by a computer in a process known as *back projection*, which results in formation of a video cross-section image.[10,11] Multiple sequential images ("slices") are made through the anatomic region of interest. A major advantage of CT is that the cross-section images allow visualization of anatomic structures without the superimposition found in planar radiographic images. Figure 2 is an example of a CT image made through the elbow of a dog with a fragmented coronoid process.

MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging (MRI) is a cross-sectional imaging system developed since the advent of CT. The patient is placed in a strong magnetic field to align the magnetic vectors of the atoms in the body. Radiofrequency waves are then passed through the body to deflect the magnetic vectors. Depending on the characteristics of the atoms in a given tissue, the vectors return to alignment with the magnetic field at different rates. As the vectors realign, they omit radiofrequency waves that are detected by a receiving coil. This information is relayed to computers that use the data to generate cross-sectional images.[12] A major advantage of MRI is its ability to differentiate soft tissue structures. In orthopedics, this allows visualization of cartilage, ligaments, tendons, and subchondral bone. Dense bone generates almost no signal and appears as a signal void or negative image on magnetic resonance

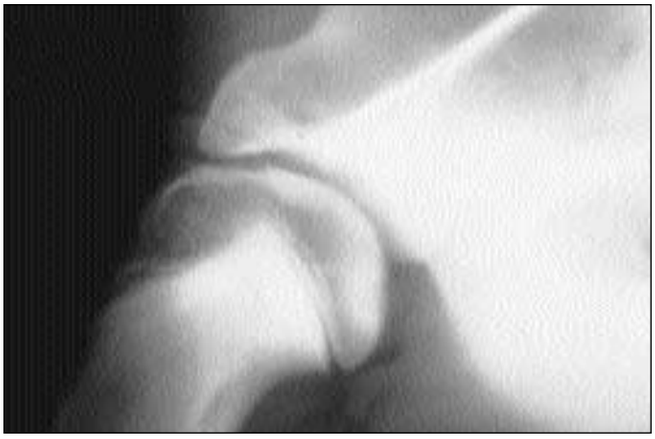

Figure 1—*Conventional survey radiograph. Mediolateral projection of the right shoulder of a 6-month-old Great Dane showing radiographic signs characteristic of osteochondrosis of the humeral head. Note sclerosis and flattening of subchondral bone at the caudal aspect of the articular surface.*

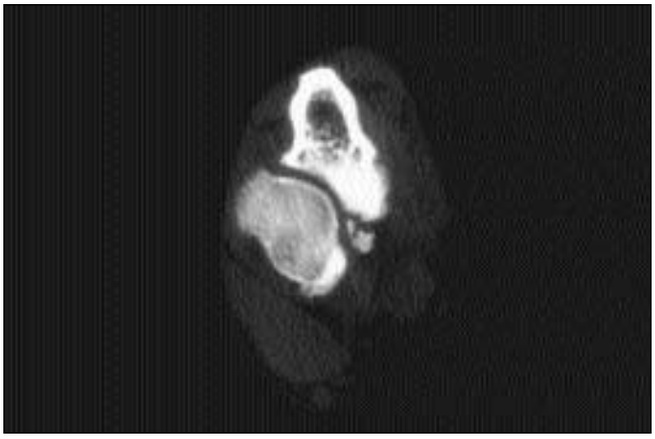

Figure 2—*Computed tomography image. Transverse image of the elbow at the level of the radial head in an 8-month-old Labrador retriever with fragmented coronoid process of the ulna. Note the small fragment of bone seen on this image that would not be visible on radiographs of the elbow.*

images. Computed tomography is superior to MRI for visualization of osseous structures. Figure 3 is a magnetic resonance image of a dog with osteochondrosis of the humeral head.

DUAL ENERGY X-RAY ABSORPTIOMETRY

Dual energy x-ray absorptiometry (DEXA) machines use a pencil-thin, scanning x-ray beam with receptors that measure absorption of the beam by the patient. Dual energy beams allow differentiation of bone, fat, and lean tissue so that body composition analysis can be made.[13] Early experience with DEXA in dogs indicates that it is a reliable method for noninvasive assessment of bone mineral density as well as for total bone mineral and body composition. This technology offers a remarkable tool for study of skeletal growth and development in relation to diet and other extrinsic factors because repeat studies can be made on the same animal without the necessity of harvesting tissues for analysis.

APPLICATION OF DIAGNOSTIC IMAGING TECHNIQUES

Too often, radiography and other diagnostic imaging techniques are considered only as a method to make a diagnosis of a clinical abnormality. In many cases, diagnostic imaging does not allow a specific etiologic diagnosis but does provide valuable information about the site, extent, and nature of an abnormality. It is also important to realize that skeletal growth and development is a dynamic process and that diagnostic images represent the state of tissues only at a single point in time, a "snapshot" of an ongoing process. Serial radiographs made over weeks or months can provide much more comprehensive information than any single examination. It is also evident that diagnostic imaging techniques can be used to collect information not

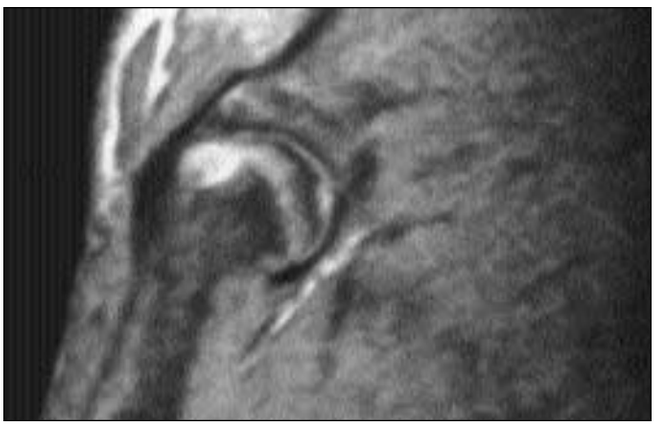

Figure 3—*Magnetic resonance image. Sagittal image of the scapulohumeral joint of a 6-month-old Great Dane showing abnormalities characteristic of osteochondrosis of the humeral head. Note the irregular cartilage at the caudal aspect of the articular surface and the decreased signal in the subchondral bone, characteristic of sclerosis.*

only from dogs with clinical signs of skeletal abnormality but also from normal growing dogs to further document the process of skeletal development[5] and to assess for subclinical abnormalities in dogs that do not show overt clinical signs.[14] An extended list of the possible applications of diagnostic imaging techniques for skeletal assessment of growing dogs is presented in Table 2.

It certainly is not practical to employ the full array of diagnostic imaging techniques on any given animal. Clinicians and researchers alike must choose the appropriate imaging method or methods to most efficiently and effectively arrive at a diagnosis and treatment plan or to collect sufficient data to prove or disprove a hypothesis. It is the challenge of imaging specialists to determine the appropriate applications of the many imaging techniques available and to develop algorithms for their appropriate use.[7] Radiography remains the most inexpensive method

TABLE 2

Applications of Diagnostic Imaging Techniques in Skeletal Evaluation of Growing Dogs

Examination of Dogs with Clinical Signs of Lameness and/or Skeletal Repair or Deformity

- Provide information to aid in determination of an accurate diagnosis
- Provide information to aid in determination of appropriate medical, surgical, and/or nutritional treatment
- Determine the anatomic location of the abnormality
- Determine the morphologic nature of the osseous abnormality (i.e., lysis or osseous proliferation)
- Determine the extent and severity of the abnormality
- Determine whether the abnormality is unilateral, bilateral, or multifocal
- Determine (by serial examinations) the progression, resolution, or response to treatment of the abnormality

Examination of Growing Dogs with No Clinical Signs of Skeletal Abnormality

- Initial screening examinations for early signs of developmental abnormality to assess
  - —Joint conformation (i.e., coxofemoral joints and elbows)
  - —For known heritable diseases such as chondrodysplasia or dwarfism
  - —Physeal closure and skeletal maturity
  - —Bone density and body composition
- Serial screening examinations to collect research data on normal skeletal development and incidence of subclinical abnormalities to document
  - —Appearance and development of secondary ossification centers
  - —Longitudinal and oppositional bone growth
  - —Shape, curvature, and alignment of bones
  - —Appearance and progression or resolution of subclinical skeletal abnormalities
  - —Physeal closures
  - —Progressive changes in body composition and bone mineral density

of skeletal imaging and is appropriate for initial survey of most developmental processes and possible developmental disorders. The radiographic appearance of the various juvenile bone diseases has been well documented.[3,4,6,9,20] Determination of standard protocols for skeletal imaging and interpretation with CT, MRI, and DEXA are ongoing in veterinary medicine but must be considered in their infancy at this time. A list of developmental disorders is presented in Table 3 along with imaging techniques that are useful or may potentially be useful in assessment of those disorders. In most cases, further investigation will be necessary to determine which imaging techniques other than radiography are most appropriate for diagnosis or screening of these diseases.

CURRENT RESEARCH

At Auburn University, a number of faculty members are currently completing a long-term cooperative project to evaluate the effects of various dietary levels of calcium and phosphorus on the growth and development of Great Dane puppies.[14,17–19] Thirty-two puppies were divided into three groups and fed different diets described below:

- **Low-mineral diet—**0.48% Ca/0.40% P; Ca:P ratio, 1.2:1.0
- **Medium-mineral diet—**0.80% Ca/0.67% P; Ca:P ratio, 1.2:1.0
- **High-mineral diet—**2.70% Ca/2.20% P; Ca:P ratio, 1.2:1.0

All three diets were isocaloric, and the nutrient composition was constant other than the percentage of calcium and phosphorus. The puppies were bred specifically for this study and were randomly assigned to the three groups at birth. They were maintained on the three diets for 18 months and were periodically evaluated by orthopedic examination; DEXA; radiographs of the cervical spine, scapulohumeral joints, antebrachium, stifle joints, and pelvis; and MRI of the carpus and scapulohumeral joint.

The results of the study showed differences in bone mineral density, with puppies fed the high-mineral diet exhibiting significantly higher bone mineral density. Puppies fed the medium-mineral diet were somewhat heavier and taller than others at 4 to 6 months of age, but by 12 to 18 months there was little difference in the size of the puppies fed the three different diets. Although many puppies did not exhibit clinical signs of lameness, a number of developmental bone abnormalities were seen on radiographs and magnetic resonance images.[14] The abnormalities identified included osteochondrosis of the humeral head, retained cartilage core, hypertrophic osteodystrophy, cranial mandibular osteopathy, cervical spinal abnormality, hip dysplasia, and panosteitis.

By 6 months of age, 7 of 32 puppies had shown evidence of clinical lameness. Six of these seven puppies that exhibited clinical lameness were fed the high-mineral diet. Preliminary assessment of the results of this study indicates that puppies fed the high-mineral diet did not grow faster or taller than puppies fed the low-mineral diet and that puppies on the high-mineral diet seemed to have poorer conformation and a higher incidence of clinical lameness than puppies on the medium- or low-mineral diets. The differences observed by radiography, MRI, and DEXA were most prominent during the first 6 months of life in these puppies. Clinical lameness and conforma-

TABLE 3
Developmental Disorders Affecting the Bones and Joints of Growing Dogs

Skeletal Disorders	Useful Imaging Techniques	Potentially Useful Imaging Techniques
Osteochondrosis (OD)	RAD, CT, MRI, ARTH	SCINT
Elbow dysplasia		
Ununited anconeal process (UAP)	RAD	CT
Fragmented coronoid process (FCP)	RAD, CT	SCINT, ARTH
OD of medial humeral condyle	RAD, CT	MRI, SCINT
Elbow incongruity	RAD	CT, ARTH
Retained cartilage core	RAD	MRI
Hypertrophic osteodystrophy (HOD; metaphyseal osteodystrophy)	RAD	MRI
Panosteitis (enostosis)	RAD, SCINT	MRI
Canine hip dysplasia	RAD	
Asynchronous growth of radius and ulna	RAD	
Patellar luxation	RAD	
Cervical vertebral malformation/malarticulation (CVMM; wobbler syndrome)	RAD, MYELO, CT, MRI	DEXA
Generalized increase or decrease in skeletal mineralization	RAD, DEXA	QCT
Primary hyperparathyroidism	DEXA	QCT
Secondary nutritional or renal hyperparathyroidism (fibrous osteodystrophy)	RAD, DEXA	QCT, MRI (spine)
Rickets	RAD, DEXA	QCT
Avascular necrosis of the femoral head (Legg–Perthes)	RAD, CT	MRI
Craniomandibular osteopathy (CMO)	RAD	CT, DEXA
Multiple cartilaginous exostosis (MCE)	RAD	SCINT
Multiple epiphyseal dysplasia (MED)	RAD	MRI
Chondrodysplasia/dwarfism	RAD	

RAD = radiology, CT = computed tomography, MRI = magnetic resonance imaging, ARTH = arthrography, SCINT = scintigraphy, QCT = quantitative computed tomography, DEXA = dual energy x-ray absorptiometry, MYELO = myelography.

tional abnormalities were also much more apparent in puppies younger than 6 months of age than in older, maturing puppies.[19] These observations suggest that excess mineral content in the diet and extra mineral supplementation should be avoided in rapid-growing giant-breed dogs, especially during the first 6 months of life.

Our experience also indicates that DEXA scanning techniques can be adapted to provide accurate and reproducible information regarding bone mineral density and body composition in dogs.[17] Reliable scanning requires general anesthesia of the dog, with careful attention to symmetric and reproducible positioning on the

scanning bed. Evaluation of the serial radiographs showed a high incidence of subclinical skeletal abnormality in these young Great Danes. The magnetic resonance images are currently being evaluated to determine how well they correlate with the radiographic images.

REFERENCES

1. Dammrich K: Relationship between nutrition and bone growth in large and giant dogs. *J Nutr* 121:S114–S121, l991.
2. Goedegebuure SA, Hazewinkel HAW: Morphological findings in young dogs chronically fed a diet containing excess calcium. *Vet Pathol* 23:594–605, 1986.
3. Hedhammer A: Nutrition related orthopaedic diseases, in Kelly N, Wills J (eds): *Manual of Companion Animal Nutrition & Feeding*, ed.1. Ames, IA, Iowa State University Press, 1996, pp 198–206.
4. Kallfelz FA: Skeletal and neuromuscular diseases, in Lewis LD, Morris ML, Hand M (eds): *Small Animal Clinical Nutrition III*. Topeka, KS, Mark Morris Associates, 1990, pp 12.1–12.15.
5. Voorhout G, Hazewinkel HAW: A radiographic study on the development of the antebrachium in Great Dane pups on different calcium intakes. *Vet Radiol* 28(4):152–157, 1987.
6. Hazewinkel HAW: Calciotropic hormones and bone metabolism, in Rijnberk A (ed): *Clinical Endocrinology of Dogs and Cats*. The Netherlands, Kluwer Acad Publishers, 1996, pp 177–195.
7. Fitch RB, Wilson ER, Hathcock JR, Montgomery RD: Radiographic, computed tomographic and magnetic resonance imaging evaluation of a chronic long digital extensor tendon avulsion in a dog. *Vet Radiol Ultrasound* 38(3):177–181, 1997.
8. Widmer WR, Buckwalter KA, Braunstein EM, et al: Radiographic and magnetic resonance imaging of the stifle joint in experimental osteoarthritis of dogs. *Vet Radiol Ultrasound* 35(5):371–383, 1994.
9. Riser WH: Radiographic differential diagnosis of skeletal diseases of young dogs. *J Am Vet Radiol Soc* 5:15–27, 1964.
10. Hathcock JT, Stickle RL: Principles and concepts of computed tomography. *Vet Clin North Am Small Anim Pract* 23(2):399–435, 1993.
11. Stickle RL, Hathcock JT: Interpretation of computed tomographic images. *Vet Clin North Am Small Anim Pract* 23(2):417–435, 1993.
12. Shores A: Magnetic resonance imaging. *Vet Clin North Am Small Anim Pract* 23(2):437–459, 1993.
13. Grier SJ, Turner AS, Alvis MR: The use of dual-energy x-ray absorptiometry in animals. *Invest Radiol* 31:50–62, 1996.
14. Brawner WR Jr, Hathcock JT, Goodman SA, et al: Radiographic lesions observed in growing Great Dane puppies fed diets varying in mineral content: A preliminary report. Proceedings of the American College of Veterinary Radiology 1.9, 1996.
15. Chambers MD: Bone imaging: The diphosphonates. *Handbook of Veterinary Nuclear Medicine*. Raleigh, NC, North Carolina State University, 1996, pp 49–59.
16. Markel MD, Morin RL, Wikenheiser MR, et al: Multiplanar quantitative computed tomography for bone mineral analysis in dogs. *Am J Vet Res* 52(9):1479–1483, 1991.
17. Lauten SD, Brawner WR Jr, Goodman SA, et al: Body composition of growing Great Dane puppies fed diets varying in calcium and phosphorus concentration evaluated by dual energy x-ray absorptiometry. Proceedings of the American College of Veterinary Radiology 1.8, 1996.
18. Brawner WR Jr, Hathcock JT, Lauten SD, et al: Effect of dietary mineral content on the occurrence of juvenile bone disease in Great Danes. Proceedings of the Auburn University Annual Conference for Veterinarians, 1997.
19. Goodman SA, Lauten SD: Feeding large and giant breed puppies: Are we feeding too much calcium? Proceedings of the Auburn University Annual Conference for Veterinarians, 1997.
20. Wind AP: Elbow dysplasia, in Slatter D (ed): *Textbook of Small Animal Surgery*, ed 2, vol II. Philadelphia, WB Saunders, 1993, pp 1966–1977.

Medical Management of Skeletal Disease in Dogs

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DEGENERATIVE JOINT DISEASE

Osteoarthritis, or degenerative joint disease (DJD), is the most common orthopedic diagnosis requiring medical management. In fact, DJD is the most common orthopedic diagnosis in dogs. Analysis of the Veterinary Medical Data Base (VMDB) survey from 1984 to 1994 revealed that 141,760 (23%) of 613,380 dogs had one or more orthopedic diagnoses. DJD was the most common, accounting for 11.5% of all orthopedic diagnoses. Similarly, a VMDB survey from 1980 to 1989 identified that 112,826 (24%) of 471,690 dogs had one or more orthopedic diagnoses.[1] DJD (41%) was also the primary diagnosis of all appendicular diagnoses in this report.[1]

The first step in management of DJD is client education. Clients should be informed that arthritis can *not* be cured and that medication can only reduce or improve the clinical signs, not stop the progression or cure DJD. However, in my opinion, medication is the least important aspect of DJD management. Conversely, the most important considerations are (1) exercise to maintain muscle mass and (2) correction or prevention of obesity.

All of us, clients and patients included, wish that correcting a problem were as easy as taking a pill. However, common sense tells us that if two individuals (dog or human) have ostensibly the same arthritis, the athlete will have less morbidity than the "couch potato." A good example is hip dysplasia, the most common cause of DJD in dogs. Of the weight-bearing forces in the normal hip region, approximately 50% go through the coxofemoral joint and the other 50% through the surrounding musculature. Muscle atrophy associated with DJD causes a greater percentage of the weight-bearing to go through the diseased joint. More load through the diseased joint causes the limb to be used less, which causes more muscle atrophy, and the cycle continues. The end result is progressively worsening muscle atrophy and lameness. The cycle can be broken by forced exercise to minimize muscle atrophy.

Exercise to Maintain Muscle Mass

Exercise is the most important aspect of conservative therapy. The goal is to build or maintain muscle mass, and it is much easier to maintain muscle mass than to try and rebuild it. Obviously, low-impact exercise such as leash walks or swimming is far better than high-impact exercise (e.g., Frisbee® competition) that places high-load shocks through the joint. However, clients should be informed that allowing the dog to run loose in the back yard and assuming it is exercising is *not* sufficient. Ad lib exercise will likely be too little exercise and in a few cases may be too high-impact for short periods of time.

Clients should be informed that arthritis can *not* be cured and that medication can only reduce or improve the clinical signs, not stop the progression or cure DJD.

I recommend a program of leash walks or swimming on Monday, Wednesday, and Friday. This program is analogous to "getting in shape" or any other exercise program. It is difficult at first, but as the muscle mass improves, the rewards are well worth the effort. Owners should decide how long to exercise their pet but with the understanding that they are to "push the envelope" and make the exercise time progressively longer.

The dog will experience some discomfort the day after exercise, especially during the beginning of the program. Therefore, on Tuesday and Thursday the dog should exercise ad lib (i.e., rest) and be given medications such as nonsteroidal antiinflammatory drugs (NSAIDs) *only* if necessary for the dog to exercise the next day. Limiting NSAID administration to every other day (and then only if necessary) reduces the risk of side effects. NSAIDs frequently become unnecessary or rarely necessary within a few weeks or months into the program.

This conservative management exercise program

should be considered a lifelong treatment since DJD is a lifelong disease. It requires commitment by both the dog and owner, and not every owner will comply. However, in 12 years of recommending this program, I am convinced that frequent low-impact exercise, not rest, is the best treatment. Owners should ultimately understand that failure of this conservative treatment program (with NSAIDs or other medications) is an indication for surgical intervention. Corticosteroids are not recommended since DJD is a chronic disease, and chronic use of corticosteroids is unsafe.

TABLE 1
Classes of Nonsteroidal Antiinflammatory Drugs

Chemical Classification	Generic Name (example)	Brand Name (example)
Salicylates (acetylated)	Aspirin	Numerous
Salicylates (nonacetylated)	Salsalate	Disalcid™ (3M)
Fenamates	Meclofenamic acid	Meclomen® (Parke-Davis)
Acetic Acids	Indomethacin	Numerous
Propionic Acids	Carprofen	Rimadyl® (Pfizer Animal Health)
	Ibuprofen	Motrin® (McNeil)
	Ketoprofen	Orudis® (Wyeth-Ayerst)
Pyrazolones	Phenylbutazone	Butazolidin® (Novartis)
Oxicam	Piroxicam	Feldene® (Pfizer Inc)
Nonacidic	Nabumetone	Relafen® (SmithKline Beecham)

Obesity Correction and Prevention

Obesity is an important consideration with conservative management of DJD. Again, common sense tells us that an arthritic joint should not be made to bear any more weight than is necessary. Exercise, as described above, helps with obesity correction. Unfortunately, what many owners believe is an ideal weight for their dog is in fact somewhat overweight. Client education on determining an ideal weight includes rubbing the chest without pressure and barely being able to feel the ribs, presence of a waist, being able to easily palpate the wing of the ilium, etc. Despite the revenue generated from dietary gimmicks, the fact remains that intake calories should not substantially exceed the calories burned by body maintenance and exercise. Many formulas exist, but a simple formula[2] for desired Kcal/day is as follows:

$$\text{Kcal/day} = (30 \times \text{target weight [kg]}) + 70$$

This formula takes into account all the food (e.g., the table food, cat food, etc.) consumed by the dog. If owners are only given the number of Kcal/day required, few patients will lose weight. Clients should provide the practitioner with the number of Kcal/unit (cup, can, etc.) of the dog food being used. Based on this number, clients should be informed to feed x number of cups per day and notified that a change in dog food will change this number. Owners should be informed that weight reduction in dogs takes time. They should not become discouraged if immediate results are not seen in their dogs.

Hypothyroidism ("the great impersonator") should be a consideration in any weight reduction program. The Orthopedic Foundation for Animals has started a "Thyroid Registry." In my opinion, hypothyroidism should be checked by measurement of free T_4 (thyroxine) levels at the start of any weight loss program and periodically rechecked regardless of whether the patient is on thyroid supplementation. Untreated or inadequately treated hypothyroidism will negate any attempts at weight reduction.

Medical Management of Degenerative Joint Disease

Medications that are effective and relatively safe for treatment of DJD include various (but not all) NSAIDs, polysulfated glycosaminoglycan (Adequan® Canine, Luitpold), and possibly nutraceuticals. Nutraceuticals do not require FDA approval, and to my knowledge none have proven their efficacy beyond subjective assessment of clinical signs. On the other hand, to my knowledge, none have serious side effects. Medications that I do not recommend are some NSAIDs, corticosteroids, and flunixin meglumine.

Regarding NSAIDs, none are 100% safe and some cause serious complications after short-term use at multiple doses per day,[3–5] hence my recommendation of giving NSAIDs no more than every other day. The Georgia Animal Poison Information Center reported that 5.2% (240 calls) of their calls over a 19-month period were NSAID related.[5] Ibuprofen (n = 120), acetaminophen (n = 94), aspirin (n = 18), and indomethacin (n = 8) accounted for 98% of the NSAID-related inquiries.[6] The most common clinical signs were vomiting and diarrhea, central nervous system depression, and circulatory manifestations.[6]

DJD is a lifelong disease; therefore, medications that are as safe as possible for long-term use are necessary and should be titrated to the lowest effective dose and frequency. In my opinion, this premise eliminates daily use of any medication for treatment of DJD. Specific medications that should not be used long term (or at all) for DJD are corticosteroids, flunixin meglumine, and phenylbutazone. Carprofen (Rimadyl®, Pfizer Animal Health) is a relatively new veterinary medication for the treatment of DJD. A Pfizer Animal Health Advisory distributed in August of 1997 reported that about 150,000 dogs are on carprofen,

TABLE 2
Antiinflammatory:Ulcerogenic Potential of NSAIDs

NSAID	COX-2 (Antiinflammatory)	COX-1 (Ulcerogenic)	COX-2:COX-1 Ratio
Acetaminophen	133	17.9	7.4
Aspirin	278	1.67	166
Carprofen	11	10.9	1
Ibuprofen	73	4.85	15
Indomethacin	1.68	0.028	60
Sodium salicylate	725	254	2.8

and label instructions are for q12h doses.[5] A total of 750 adverse side effects were reported, with gastrointestinal side effects being the most common. Additional "suspected" side effects include renal, hematologic, neurologic, dermatologic, and hepatic side effects. Acute hepatopathy was reported 16 to 21 days after starting carprofen therapy, with initial clinical signs of vomiting and/or anorexia. Six Labrador Retrievers reported by Colorado State University all recovered from acute hepatopathy after discontinuing use of carprofen and receiving supportive therapy. Labrador Retrievers and other breeds have also been reported to be afflicted with acute hepatopathy.[5] Pfizer Animal Health reported that 14 side effects (of any type) occurred for every 10,000 dogs treated with Rimadyl®. Again, I would not recommend daily administration of any medication for treatment of DJD. In addition, enteric-coated aspirin (or other enteric-coated medications) have been reported to produce large fluctuations in plasma concentrations, and more predictable alternatives should be used.[7]

With regard to pain relief, effectiveness in a given individual is unpredictable (hence so many products). If a drug in human medicine is ineffective, the typical recommendation is to change to a different class of NSAID (Table 1). However, side effects in dogs for many of these products are unknown and therefore my recommendations are limited to NSAIDs with a track record in dogs. NSAIDs inhibit prostaglandins, with isoenzymes cyclooxygenase-1 (COX-1) affecting the stomach and cyclooxygenase-2 (COX-2) affecting prostaglandins released during inflammation. The ratio of COX-2 divided by COX-1 indicates the antiinflammatory:ulcerogenic potential (Table 2), with the lower number having the better benefit:risk ratio.[8]

My personal recommendations are aspirin, aspirin with Maalox® (Ascriptin®, Rhône-Poulenc Rorer), buffered aspirin, or Rimadyl®. However, it should be noted that these medications should not be given more often than every other day. I also recommend Adequan® Canine (polysulfated glycosaminoglycan) for the treatment of DJD. Adequan® Canine is directly antiinflammatory via decreased prostaglandin E_2 release, inhibition of lysosomal enzymes from leukocytes, and scavenging of free radicals. In addition, polysulfated glycosaminoglycan is a normal component of joint fluid and is potentially a "chondroprotective" agent. So there is a logical physiologic/biochemical reason for Adequan® Canine to be effective. Although most veterinarians use Adequan® Canine in advanced cases of DJD, the chondroprotective potential prompts some to use this agent in a preventative role. In my experience, Adequan® Canine is effective in more than 80% of the dogs with DJD for a period of 2 or more years. The disadvantages of Adequan® Canine are its cost and availability only as an intramuscular injection (possibly a factor in its high efficacy), which in turn requires office visits for administration. Adequan® Canine administered via intraarticular injection is *not* recommended because a synovitis often results. In addition, the synovial membrane is a poor filter; therefore, intraarticular and plasma concentrations equilibrate rapidly regardless of the route of injection.

Medications that are effective and relatively safe for treatment of DJD include various (but not all) NSAIDs, polysulfated glycosaminoglycan, and possibly nutraceuticals.

JUVENILE BONE AND JOINT DISEASE

Hypertrophic osteodystrophy, panosteitis, and cranial mandibular osteopathy are juvenile bone and joint diseases that cannot be treated by surgery but are, however, amenable to medical therapy. These conditions usually are not lifelong afflictions and therefore can be treated with aspirin, as well as corticosteroids or other drugs that are not recommended for long-term therapy.

Hypertrophic Osteodystrophy

Hypertrophic osteodystrophy is a juvenile bone disease of large and giant breeds.[9] Age of onset is usually 3 to 5 months of age but may extend to 8 months. The disease is limited to dogs with open physes at the affected sites. Hypertrophic osteodystrophy is characterized by subperiosteal hemorrhage at the metaphysis plus hemorrhage and necrosis in the metaphysis evident within a few millimeters of the physis, which is seen radiographically as

a pseudophyseal line (a pathognomonic characteristic). The etiology of the condition is unknown but is often associated with "overnutrition" in rapidly growing (hence the early age of onset) large and giant breeds. Vitamin C deficiency is not a cause, since all mammals except primates and guinea pigs make their own, but abnormal vitamin C metabolism has not been ruled out.

Clinical signs of hypertrophic osteodystrophy include lethargy, unwillingness to stand, weight loss, and substantial elevation of temperature (e.g., 104˚F). Some animals also have a history of diarrhea and/or respiratory disease. The distal radius–ulna is the most severely affected location, and palpation causes severe pain. In rare cases, permanent deformity of the long bones can result. More typically, treatment with short-acting corticosteroids and supportive therapy (fluids, tube feeding, well-padded bedding) result in a rapid and full recovery. Recurrent episodes occur infrequently.

Juvenile bone and joint diseases usually are not lifelong afflictions and therefore can be treated with aspirin, as well as corticosteroids or other drugs that are not recommended for long-term therapy.

Panosteitis

Panosteitis is inflammation of the adipose in the bone marrow followed by osteogenesis within the medullary canal of long bones of juvenile dogs. One case has been reported to occur at 5 years of age.[9] Although numerous efforts have been made to identify an etiology, the cause is unknown. Clinical signs typically include a mild lameness of the affected limb, although more severe lameness can occur. Deep palpation usually yields a painful response during the acute phase (typically 7 to 14 days), especially if deep palpation is near the nutrient foramen (junction of proximal third with distal two thirds of a long bone). The history often includes an intermittent "shifting leg lameness" and in more severe cases, anorexia, malaise, and reluctance to move. Although a single bone is rarely affected twice, the disease may occur in each long bone in the leg. Bones of the foreleg are more frequently affected than those of the hindleg (ulna 42%, radius 25%, humerus 14%, femur 11%, tibia 8%). There is a high incidence of panosteitis in German Shepherd Dogs, but the disease can occur in any large breed.

Radiographic changes begin as a radiolucency of the medullary cavity near the nutrient foramen, followed by marked radiopacity and subsequently a coarse trabecular pattern. A mild periosteal reaction may occur in some cases. The radiographic changes occur over approximately 90 days, which may or may not coincide with the presence or severity of clinical signs. Most dogs are mildly affected and do not require treatment. Slightly more affected dogs respond well to aspirin or corticosteroids given for about 1 week to 10 days. More severely affected dogs almost invariably respond very quickly to corticosteroids.

Cranial Mandibular Osteopathy

Cranial mandibular osteopathy is an osseous proliferation of the mandibles and occasionally is seen in the tympanic bulla, cranium, radius, and ulna. Association with hypertrophic osteodystrophy has been speculated. Cranial mandibular osteopathy occurs most commonly in the West Highland White Terrier, a breed reported to have an autosomal recessive mode of inheritance.[10] Other affected breeds include Cairn Terriers, Boston Terriers, Scottish Terriers, Doberman Pinschers, Labrador Retrievers, and Shetland Sheepdogs.[10,11] Inability to open the mouth to eat and severe pain frequently make cranial mandibular osteopathy a life-threatening disease due to euthanasia. If the puppy can be maintained until osseous maturity, the clinical signs regress as physis closure occurs. Frequent and occasionally high doses of corticosteroids are necessary to ameliorate clinical signs. Owners should pay special attention to feeding a diet that is able to fit through a slightly opened mouth and is easy to chew to ensure adequate nutrition.

REFERENCES

1. Johnson JA, Austin C, Breur GJ: Incidence of canine appendicular musculoskeletal disorders in 16 veterinary teaching hospitals from 1980 through 1989. *Vet Comp Orthop Trauma* 7:56–62, 1994.
2. Lewis L, Morris Jr. ML, Hand MS: *Small Animal Clinical Nutrition III.* Topeka, KS, Mark Morris Associates, 1987.
3. Wallace MS, Zawie DA, Garvey MS: Gastric ulceration in the dog secondary to the use of nonsteroidal antiinflammatory drugs. *JAAHA* 26:467–472, 1990.
4. Vonderhaar MA, Salisbury SK: Gastrointestinal ulceration associated with flunixin meglumine administration in three dogs. *JAVMA* 203:92–95, 1993.
5. Pfizer Animal Health Advisory, August 1997.
6. Jones RD, Baynes RE, Nimitz CT: Nonsteroidal anti-inflammatory drug toxicosis in dogs and cats: 240 cases (1989–1990). *JAVMA* 201:475–477, 1992.
7. Nap RC, Peters IOM, Willemsen A, de Bruyne JJ: Aspirin medication in dogs. *Vet Comp Orthop Trauma* 4:95–99, 1991.
8. Pfizer Animal Health: Technical Monograph on Rimadyl, 1997, pp 16–17.
9. Milton JL, Montgomery RD: Diagnosis of foreleg lameness, in. Ford RB (ed): *Clinical Signs and Diagnosis.* New York, Churchill Livingstone, 1988, pp 351–387.
10. Padgett GA, Mostosky UV: The mode of inheritance of craniomandibular osteopathy in West Highland White Terrier Dogs. *Am J Med Genetics* 25:9–13, 1986.
11. Taylor SM, Remedios A, Myers S: Craniomandibular osteopathy in a Shetland sheepdog. *Can Vet J* 36:437–439, 1995.

Surgical Treatment of Elbow Dysplasia

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Elbow dysplasia is recognized by veterinarians and breeders as a serious problem in certain populations. Depending on the specific subpopulation and method of investigation, elbow dysplasia is seen most often in the following breeds[1]:

- Rottweilers (46% to 50%)
- Bernese Mountain Dogs (36% to 70%)
- Newfoundlands (30%)
- Golden Retrievers (20%)
- German Shepherd Dogs (18% to 21%)
- Labrador Retrievers (12% to 14%)

Elbow dysplasia is also seen in the Great Dane, Saint Bernard, Irish Wolfhound, Great Pyrenees, Bloodhound, Bouviers des Flandres, and Chow Chow, as well as in chondrodystrophic breeds.[2,3]

The success rate of surgical treatment of elbow dysplasia depends on many factors, such as a complete diagnosis before surgery, correct surgical positioning, atraumatic surgical approach, and aftercare by the owner. Elbow dysplasia can be separated into different disease entities, including the ununited anconeal process (UAP), fragmented coronoid process (FCP), osteochondritis dissecans (OCD) of the medial humeral condyle, and incongruities of the elbow joint (IE). This manuscript discusses the clinical and radiologic protocols for diagnosis of UAP, FCP, and OCD; the lateral approach to the elbow joint for removal of the UAP and ulnectomy for fusion of the anconeal process; the medial approach to the elbow joint for removal of FCP and OCD; and new information on the prevention of elbow dysplasia.

CLINICAL AND RADIOLOGIC INVESTIGATION

Clinical investigation starts with registration of the breed (Table 1), age of the dog (lameness begins at 4 to 10 months of age), and inspection of the dog in a standing position. In almost 50% of the cases, the paw of the affected leg is externally rotated and slightly abducted (Figure 1). During walking and trotting, the head of the dog is lifted when the affected leg is weight bearing. When both front legs are affected, this occurs as the most painful leg is weight bearing. On palpation, the elbow is effused. Effusion is usually most pronounced in cases of UAP rather than in cases of FCP or OCD. Effusion of the elbow joint is felt at the side of the anconeal muscle; slight bulging of the muscle is physiologic. With the dog in lateral recumbency, the range of motion of the elbow joint is examined by placing the thumb on the anconeal muscle to register crepitation, noting when pain is evoked. In cases of UAP, crepitation and pain sensation most often occur during a firm hyperextension of the elbow joint. In cases of FCP and/or OCD, crepitation and a pain reaction can be evoked by prolonged hyperextension, particularly when the radius and ulna are simultaneously exorotated. This produces increased pressure at the medial region of the elbow joint and thus pain when pathology is present in that area.

The success rate of surgical treatment of elbow dysplasia depends on many factors, such as a complete diagnosis before surgery, correct surgical positioning, atraumatic surgical approach, and aftercare by the owner.

Diagnosis of elbow dysplasia is confirmed by radiography. A bony union between the anconeal process and the olecranon should be complete by the age of 16 to 20 weeks.[3] When a radiolucent area is present in dogs older

TABLE 1
Radiographic Appearance of Fragmented Coronoid Process (FCP)[5]

View	Registration of FCP (n = 108)
AP	57.4%
APMO	15.8%
ML *flexed*	82.4%
ML *extended*	93.5%
ML *extended* + AP	96.9%
ML *flexed* + ML *extended*	97.9%
ML *flexed* + ML *extended* + AP	97.9%
ML *extended* + APMO	100%
ML *flexed* + ML *extended* + AP + APMO	100% (gold standard)

than 20 weeks, it is suggestive of an anconeal process that is not bony united (i.e., UAP due to a partial or complete separation in the cartilage between the anconeal process and olecranon). On a mediolateral flexed (ML *flexed*) view, there is no superimposition of the humeral condyles over the fusion area between the anconeal process and olecranon; therefore, this view is preferred. Sclerosis at the fracture site and osteophytes at the margins of the joint can be visible at a later stage (Figure 2).

OCD of the medial humeral condyle is best assessed on anterior–posterior medial oblique (APMO) views; one third of the cases will be overlooked if an anterior–posterior (AP) view is used.[4] In a small number of cases, a calcified flap can be seen located near the indentation of the contour of the medial condyle (Figure 3). Various views are advocated in screening elbow joints for the presence of a FCP, including the AP, APMO, ML *flexed*, ML *extended*, and the ML view with the joint extended plus the 15° exorotation of the radius–ulna.[4]

We conducted a survey to determine the value and additional value of the AP, APMO, ML *flexed*, and ML *extended* views, using the complete set of the four views as the gold standard. The ML *flexed* and the APMO views were demonstrated to have limited value when used alone but great value as an additional view (Table 1). The false-negative results of almost 20% when the ML *flexed* view is used alone in a screening program for FCP may explain the differences in the percentage of positive Bernese Mountain Dogs among countries.[5,6] Osteophytes and sclerosis of the semilunar notch are also considered when making the diagnosis (Figure 4). Small osteophytes are visible on a combination of at least three of four of the mentioned views. UAP and FCP occur bilaterally in 30% and more than 50% of the cases, respectively; therefore, both elbow joints should be investigated, even in cases of unilateral lameness.

Figure 1—*Nine-month-old Bernese Mountain Dog with left front leg lameness and slight supination due to a fragmented coronoid process (FCP).*

When no radiographic abnormalities are visible, the clinical investigation then attempts to exclude differential diagnoses (i.e., OCD of the proximal humerus, panosteitis eosinophylica [enostosis], fractures of sesamoid bones), and the investigation is repeated after 3 to 6 weeks. Auxiliary techniques (computed tomography, bone scintigraphy, arthroscopy) may be of value (Figure 5).

The correlation between radiographic signs of elbow dysplasia and clinical signs depends on the following factors:

- Physical demands (working dogs versus companion animals)
- Severity of lesions (FCP plus incongruity is more severe than FCP or incongruity alone; low-grade arthrosis does not necessarily accompany lameness)
- The age of onset of complaints (lameness at a young age is more severe)
- Breed

Clinical signs due to a comparable coronoid lesion in retrievers are more severe than in Rottweilers. In a

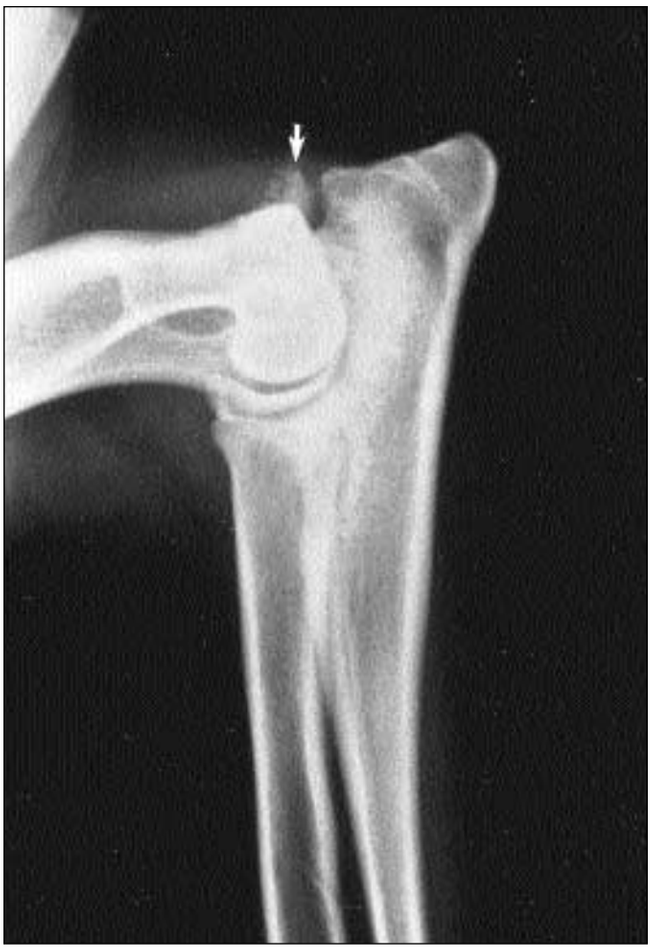

Figure 2—*Mediolateral flexed (ML* flexed*) view of the right elbow joint of a 14-month-old German Shepherd Dog with lameness of both front legs. An ununited anconeal process (UAP;* arrow*), in combination with a fragmented coronoid process (FCP), can be noticed together with sclerosis of the semilunar notch.*

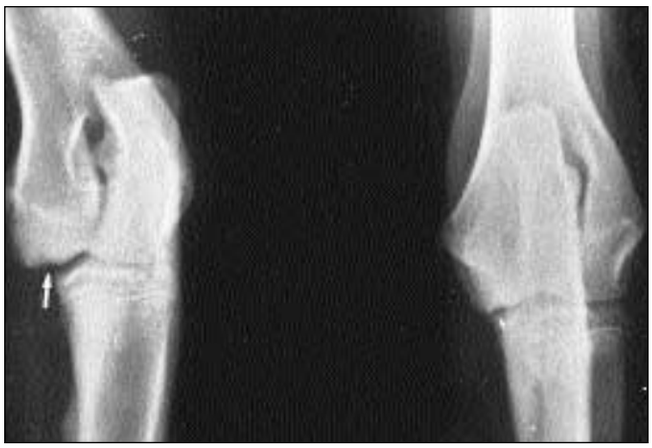

Figure 3A

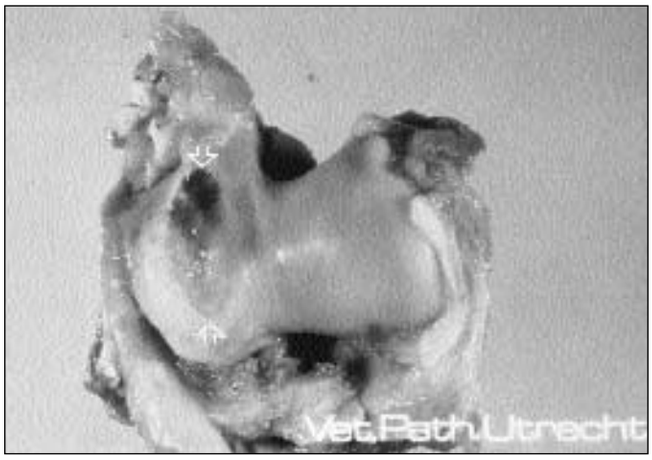

Figure 3B

Figure 3—*(A) An anterior–posterior (AP) view* (right) ***and anterior–posterior medial oblique (APMO) view*** (left) ***of the same elbow joint of a 10-month old Labrador Retriever with osteochondritis dissecans (OCD). An indentation in the medial humeral condyle*** (arrows) ***is especially visible on the APMO view, reflecting a (B) lesion of the joint cartilage and subchondral bone of the medial humeral condyle*** (arrows)***. (Courtesy of Dr. I Van der Gaag, Utrecht University.)***

prospective study of a group of 55 Rottweilers, Read and colleagues reported that 57% developed radiographic signs of FCP, but *only* 15% showed physical signs (e.g., joint effusion, pain, and crepitation) during examination, and 10% developed lameness.[7]

THERAPY

Ununited Anconeal Process (UAP)

Excision

The anesthetized dog is positioned in lateral recumbency with the lateral side of the affected elbow joint facing upward. The hair is clipped around the elbow joint, at least 10 cm proximal and distal to the lateral epicondyle. Joint effusion causes a swelling of the lateral aspect of the elbow joint, with the anconeal muscle bulging laterally. An imaginary line is drawn between the lateral epicondyle and the cranial aspect of the olecranon, just cranial to the insertion of the triceps muscle, and the distance is divided into three equal parts.[8] Counting from cranial, the incision is made between the first and second part through the skin, subcutis, fascia of the anconeal muscle, and the anconeal muscle. Care is taken not to undermine the skin. Synovial fluid is removed by suction, and hemorrhages are cauterized. The incised muscle is retracted in opposite directions with either two retractors or a Gelpi retractor. The elbow joint is flexed, and the anconeal process is freed from the supratrochlear foramen with blunt forceps and removed and fixed with pointed bone forceps. In more chronic cases, the UAP may have migrated to the proximal portion of the joint. The soft tissue attachment is cut to completely remove the anconeal process. The joint is inspected for free bodies, the fracture line may be curetted, and the joint is flushed with saline. Fascia and muscle are closed with interrupted absorbable sutures, the subcutis is closed carefully without leaving dead spaces (which may develop into a seroma), and the skin is closed routinely.

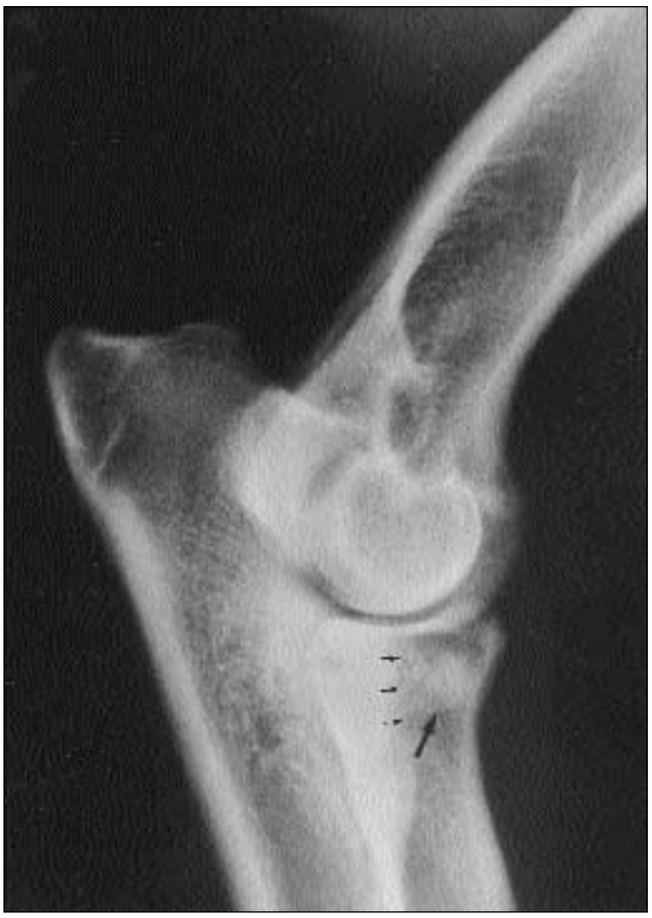

Figure 4—*Mediolateral extended (ML* extended*) view of the left elbow joint of a 12-month-old Rottweiler with a 15˚ exorotation of the radius–ulna (supination). This allows the dorsal margin of the ulna to be followed* (small arrows)*, facilitating identification of the radiolucent FCP* (large arrow)*. In addition, sclerosis of the semilunar notch can be noticed without obvious appearance of osteophytes.*

An elastic bandage is applied starting from the toes and up to and including the elbow joint. A small cut can be made in the bandage in the biceps tendon area to prevent pressure. The bandage should remain in place for 3 days, and sutures are removed after 8 to 10 days. Activity is restricted for 3 weeks; thereafter, leash activity is allowed for 3 weeks. If necessary, surgery on the other elbow may be performed after 6 weeks. Arthrosis formation will continue to develop, but it will most likely be slower than that in an irritating UAP allowed to remain in place.[9]

Reattachment

In cases of a partial separation of the anconeal process due to elbow incongruity, an osteotomy of the ulna (ulnotomy) is indicated to allow reattachment of the anconeal process. The spontaneous restoration of elbow congruity after ulnotomy may be expected in dogs younger than 12 months of age; however, in older dogs, the interosseus muscle becomes firm, thus preventing the ulna to shift proximally.

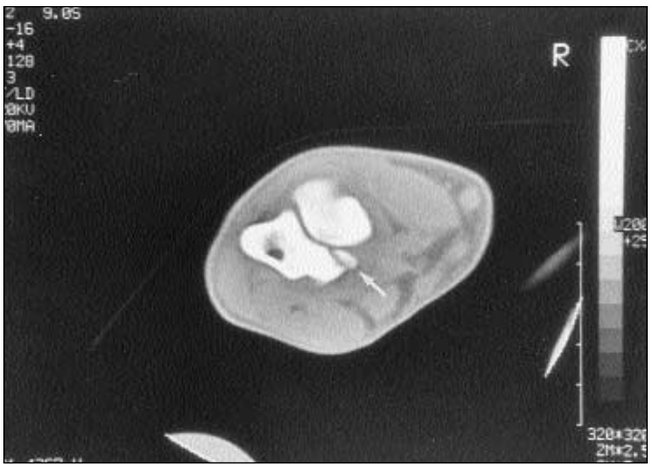

Figure 5—*Computed tomography of a 5-month-old Labrador and Golden Retriever mixed breed with unilateral front leg lameness demonstrating a fragmented medial coronoid process* (arrow).

The procedure is done with the dog in lateral recumbency. The affected elbow is facing upward, and the hair is shaved from proximal the elbow joint to half way down the length of the ulna. The skin and muscle fascia are incised at the caudolateral border of the ulna, which is located at the level of the proximal and middle one third of the ulna. The ulna is then exposed with Hohmann retractors.[10] With an oscillating saw or an osteotome, the ulnotomy is performed perpendicular or slightly oblique (i.e., 45˚) to the axis of the ulna. The free movement of the ulna is controlled with the aid of two osteotomes. Compared with the perpendicular cut, the oblique cut allows for less forward tilting of the proximal ulna by triceps pull and early healing of the osteotomy cap.[3] Forward tilting can be prevented by an intramedullary pin, which is especially advocated in chondrodystrophic breeds with a physiologic flexed elbow joint.[11] Early healing can be prevented by an ostectomy of 0.5 cm and/or by placement of autologous fat in the osteotomy gap.[3] Fascia, subcutis, and skin are closed in a routine fashion. An elastic bandage or Robert Jones bandage is applied to remain in place for 3 days. The dog is allowed to bear weight on the operated leg, and analgesics are administered because joint congruity is restored by frequent flexion and extension of the joint. Bony union may be expected within 4 to 14 weeks.[3]

Fragmented Coronoid Process (FCP) and Osteochondritis Dissecans (OCD)

The dog is positioned with the affected leg on the surgical table with the medial side of the elbow joint facing upward and placed at the edge of the table (Figure 6). After routine preparation and draping, the incision is made starting from the medial epicondyle, continuing 7 cm in the direction of the first phalanx. This incision is deepened through subcutis and fascia. The white aponeurosis between pronator teres and flexor carpi radialis (or

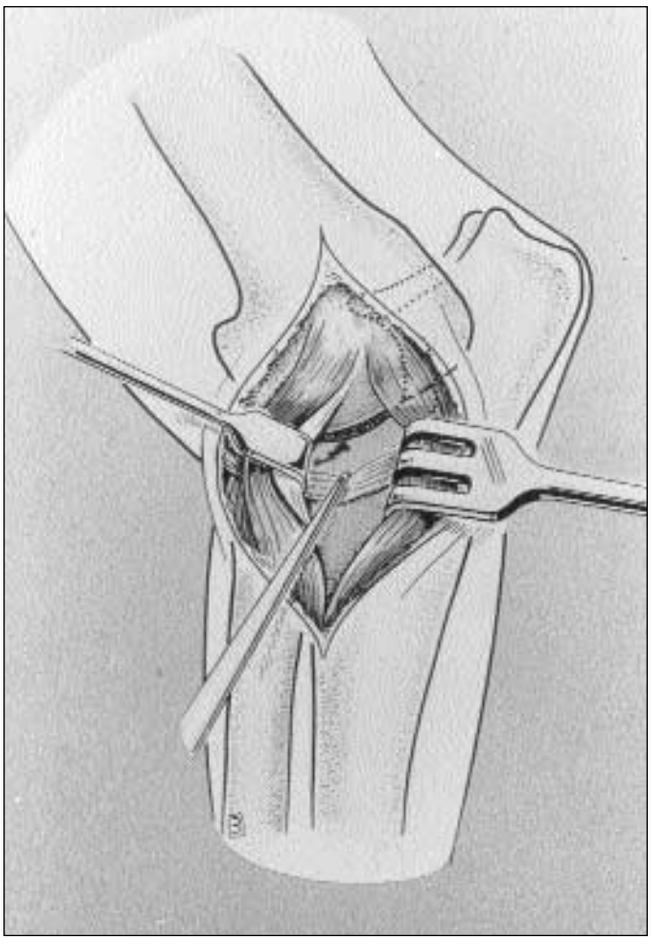

Figure 6—*Approach of the medial aspect of the elbow joint includes separation of the pronator teres and the flexor carpi radialis without desmotomy of the medial collateral ligament (retracted cranially with a Senn retractor) or of the annular ligament. In addition, endorotation and abduction of the radius–ulna should be performed to allow for inspection and surgery of a FCP or OCD lesion.*

the more subtle separation between flexor carpi radialis muscle and the humeral head of the deep digital flexor muscle) is separated by blunt dissection (Figure 6). Hemorrhages are cauterized. Using a curved mosquito forceps, the joint capsule is palpated starting at the medial epicondyle and eventually penetrated while the distal end of the leg is abducted. When the joint is opened, synovial fluid will escape. With a #11 scalpel blade, the opening is elongated proximally toward the medial epicondyle and distally toward the annular ligament, both caudal and parallel to the medial collateral ligament. The muscular branches of the median nerve are identified and protected. By opening the joint in this way, the medial collateral ligament should remain intact. Accidental incision or rupture of the medial collateral ligament is considered a serious complication and must be prevented at all times. Two small Senn retractors are used to expose the joint; the cranial retractor is positioned caudal to the medial collateral ligament, taking great care not to rupture the ligament.

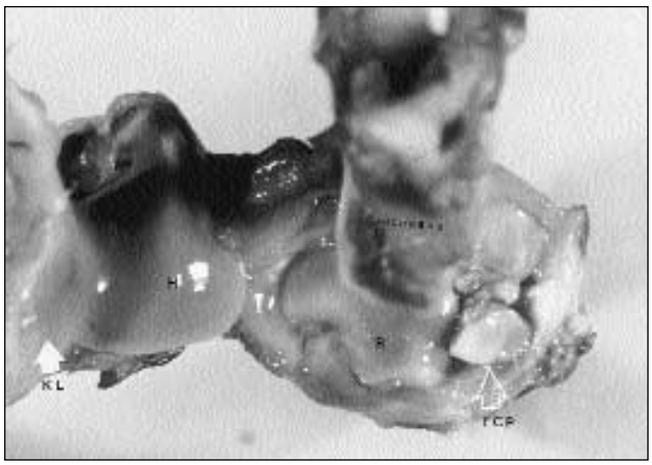

Figure 7—*Elbow joint of a 7-month-old Labrador Retriever (with lateral collateral ligament intact) with a fragmented medial coronoid process (FCP) and a kissing lesion (KL) on the opposite humeral condyle (H), which can be felt as roughening during surgery. Anconeus = anconeal process, R = radius.*

The antebrachium is now endorotated (pronation) by an assistant, thus exposing the joint space between humerus and ulna and allowing inspection of the medial coronoid process caudal to the medial collateral ligament. Using a curved mosquito forceps, the medial humeral condyle is palpated. Roughening is caused by a FCP (a kissing lesion; Figure 7), cartilage is unattached in cases of OCD, and a completely smooth surface makes the presence of a FCP questionable. The cartilage flap is removed when present and the lesion curetted carefully. When kissing lesions are present, the coronoid area of the ulna is inspected for fragmentation, abnormal coloring of the cartilage, or small blood-stained fissure / fracture lines indicating a fragmented coronoid process or chondromalacia of the joint cartilage. After 10 minutes of endorotation and abduction of the antebrachium plus retraction of soft tissues, the joint will open sufficiently.[2] If the apex of the coronoid process is fractured, it is removed and the edges smoothed with a small curette. When the FCP is "sandwiched" between the ulna and the medial aspect of the radial head, the intact medial aspect of the coronoid should be removed, which will subsequently facilitate removal of this type of FCP. Removal of the medial aspect is performed with a curette or a small (2 to 5 mm) osteotome, taking great care not to damage humeral condyle or radius cartilage. Removal of only the sandwiched FCP may cause a fractured, undermined tip in a later stage. When fissures are present in the apex of the coronoid, the apex should also be removed. The joint is frequently flushed with saline to improve the surgical view and remove the debris.

If the fractured coronoid is "lost" after the joint is prepared, it has usually moved in a cranial direction. In some instances, it is also necessary to retract the annular ligament distally. If a partially incised medial collateral lig-

TABLE 2
Success of Surgical Excision of FCP in 64 Dogs[12]

Age at Surgery	Number of Elbows with FCP	Number of Unimproved Elbows	Number of Sound Elbows (%)
2 years	51	11	40 (78%)
2 to 5 years	10	4	6 (60%)
>5 years	7	3	4 (57%)
Total	**68**	**18**	**50 (73%)**

TABLE 3
Breed Predisposition of Combined Elbow Dysplasia Entities in the Same Joint[2]

UAP and FCP	FCP and OCD
■ German Shepherd Dog	■ Labrador Retriever
■ Belgian Shepherd	■ Golden Retriever
■ Bernese Mountain Dog	■ Newfoundland
■ Doberman Pinscher	■ Bouvier des Flandres
	■ Neapolitan Mastiff

ament ruptures during forced endorotation of the radius and ulna, healing may be facilitated by Bunnell sutures using PDS or nonabsorbable suture material and a Robert Jones bandage for at least 3 weeks. However, severe lameness will usually develop because of elbow instability.

The joint capsule, muscle bellies, muscle fascia, subcutis, and skin are closed in separate layers with interrupted sutures. An elastic bandage is applied to include the surgery wound but not the olecranon. This will allow free movement and prevent postoperative swelling. Exercise is restricted for 3 weeks, followed by 3 weeks leash restriction. Lameness may resolve immediately to 6 months after surgery (mean = 6 weeks).[12]

The frequency and severity of the occurrence of osteochondrosis can be decreased by dietary management, including a food with a lowered calcium:energy ratio and quantitative restriction of food intake.

A 78% success rate was demonstrated in a follow-up study of 64 Retrievers (67.8% males) that underwent surgery at a young age (range = 0.5 to 8 years; mean = 2.7 years; Table 2). Only 33% of the conservatively treated dogs with a FCP (i.e., low bodyweight and controlled activity but no surgery) were not lame.[12] In a group of 60 Bernese Mountain Dogs (68% female), 60% had successful outcomes, 35% were satisfactory, and 5% were unsatisfactory.[13] These findings stress the importance of early diagnosis and surgical treatment.

MANAGEMENT OF ELBOW DYSPLASIA IN AFFECTED POPULATIONS

In some breeds, a combination of UAP and FCP or FCP and OCD is seen (Table 3). The combination UAP and FCP may be explained by the smaller diameter of the joint surfaces of the ulna (i.e., the ulnar trochlear notch) than of the humeral trochlea, which forces both bony protuberances from their origin.[14]

A combination of FCP and OCD has been explained by Olsson[15] as a disturbance of endochondral ossification and as such expressions of the same disease. Osteochondrosis is seen more frequently in certain breeds and subpopulations and can be aggravated by high food intake and excessive calcium intake.[16] The frequency and severity of the occurrence of osteochondrosis can thus be decreased by dietary management, including a food with a lowered calcium:energy ratio and quantitative restriction of food intake. The radiologic findings included in a dendrogram of a Labrador population revealed that FCP and OCD occurred in two different groups of closely related dogs; however, in one related subgroup, both entities were present.[17] This indicates that two different diseases may occur in the same animal. Because there is strong evidence that FCP in Retrievers is a recessive autosomal inherited disease and hence dogs that are closely related to an affected animal can have a normal phenotype, the family history should be taken into account before including dogs in the breeding stock.[17]

In 1989, a group of veterinary radiologists, geneticists, and clinicians founded the International Elbow Working Group (IEWG). Annual meetings were held in the United States and Europe. The IEWG introduced a scoring system for the evaluation of radiographs based on the proposal of Audell[18] that has been adopted by the Federation International Cynologique, the World Small Animal Veterinary Association, and different national kennel clubs. The protocol and additional information are available on the IEWG website at www.vetmed.ucdavis.edu/iewg/iewg.html or through its offices in California (fax: 650-941-7848). According to IEWG guidelines, the scoring of arthrosis can be performed on the ML *flexed* view alone, which underestimates the occurrence of the primary cause of elbow dysplasia by 12% to 25%.[5,6] The protocol, however, can be helpful to breeders for screening of elbow joints of their stock. By making the results of screening

available to all breeders, a significant improvement in elbow status can be achieved, which has been demonstrated in Norway and Sweden in Bernese Mountain Dogs, Rottweilers, Labrador Retrievers, and German Shepherd Dogs.[3,19]

REFERENCES

1. Swenson L, Audell L, Hedhammar Å: Prevalence and inheritance of and selection for elbow arthrosis in Bernese Mountain Dogs and Rottweilers in Sweden and benefit:cost analysis of a screening and control program. *JAVMA* 210:215–221, 1997.
2. Hazewinkel HAW, Kantor A, Meij BP, Voorhout G: Fragmented coronoid process and osteochondritis dissecans of the medial humeral condyle. *Tijdschr Diergeneeskd* 113:41S–46S, 1988.
3. Sjöström L, Kasström H, Källberg M: Ununited anconeal process in the dog. Pathogenesis and treatment by osteotomy of the ulnas. *Vet Comp Orthop Traumat* 8:170–176,1995.
4. Voorhout G, Hazewinkel HAW: Radiographic evaluation of the canine elbow joint with special reference to the medial humeral condyle and the medial coronoid process. *Vet Radiol* 5:158–165, 1987.
5. Hazewinkel HAW, Meij BP, Nap RC, et al: Radiographic views for elbow dysplasia screening in Bernese Mountain Dogs. Annual Meeting of the International Elbow Working Group, Constance, Germany, 1995, pp 29–32.
6. Busato A, Lang J: Comparison of two classification protocols in the evaluation of elbow dysplasia in the dog. Proceedings of the International Elbow Working Group, Birmingham, UK, 1997, p 11.
7. Read RA, Armstrong SJ, Black AP, et al: Elbow dysplasia in Rottweilers: Correlation between lameness, physical signs, and radiographic score in growing dogs. Proceedings of the International Elbow Working Group, Birmingham, UK, 1997, pp 7–8.
8. Hazewinkel HAW, Kantor A, Meij BP: Loose anconeal process. *Tijdschr Diergeneeskd* 113:47S–49S, 1988.
9. Roy RG, Wallace LJ, Johnston GR: A retrospective long-term evaluation of ununited anconeal process excision on the canine elbow. *Vet Comp Orthop Traumat* 7:94–97, 1994.
10. Piermattei D, Greely RG: *An Atlas of Surgical Approaches to the Bones of the Dog and Cat*. Philadelphia, WB Saunders, 1979.
11. Matis U: Treatment of ununited anconeal process. Proceedings of the 6th Annual Congress of the European Society of Veterinary Orthopedica and Traumatology, Roma, Italy, 1992, p 16.
12. Meij BP, Geertsen KMK, Hazewinkel HAW: Results of FCP treatment in retrievers: A follow-up study at the Utrecht University small animal clinic. Annual Meeting of the International Elbow Working Group, Constance, Germany, 1995, pp 24–26.
13. Brunnberg L, Allgoewer I: Age-related results of the treatment of elbow dysplasia (FCP) in the Bernese Mountain Dog. Annual Meeting of the International Elbow Working Group, Constance, Germany, 1995, pp 27–28.
14. Wind WP: Elbow incongruity and developmental elbow diseases in the dog: Part I. *JAAHA* 22:711–724, 1986.
15. Olsson SE: Pathophysiology, morphology, and clinical signs of osteochondrosis in the dog, in Bojrab MS (ed): *Disease Mechanisms in Small Animal Surgery*, ed 2. Philadelphia, Lea & Febiger, 1993, pp 777– 796.
16. Hazewinkel HAW: Nutrition in orthopedics, in Bojrab MS (ed): *Disease Mechanisms in Small Animal Surgery*, ed 2. Philadelphia, Lea & Febiger, 1993, pp 1117–1128.
17. Ubbink GJ, Hazewinkel HAW, Wolvekamp WTC, et al: Preliminary results of the genetic analysis of the ED program of the Dutch seeing eyes dogs. Annual Meeting of the International Elbow Working Group, Constance, Germany, 1995, pp 43–45.
18. Audell L: Heredity of elbow dysplasia: Can elbow dysplasia be controlled by judicious breeding? Proceedings of the AAHA Meeting, 1990, pp 730–733.
19. Grondalen J: Occurrence and genetic aspects of elbow dysplasia. Annual Meeting of the International Elbow Working Group, Constance, Germany, 1995, pp 12–17.

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