The Epiphyseal Plate: Nutritional and Hormonal Influences; Hereditary and Other Disorders*

Nutritional, hormonal, and genetic factors play important roles in the growth of animals.1–15 For example, unbalanced or incomplete diets can result in growth abnormalities.1–15 Hormonal factors, such as prepubertal gonadectomy, have been associated with delayed closure of growth plates in cats, which increases the risk of slipped capital femoral epiphysiodesis (SCFE) and may also affect the distal radius and femur.1,2 Documented hereditary disorders affecting the epiphyseal plate include ocular-skeletal dysplasia, dwarfism, canine epiphyseal dysplasia, premature closure of the ulna in Skye terriers, multiple cartilaginous exostoses (MCE), and mucopolysaccharidosis (MPS).3–7 Osteochondrosis dissecans of the articular cartilage is a common disease in growing dogs, but diseases of the epiphyseal cartilage, including hypertrophic osteodystrophy, retained cartilaginous core, and ununited anconeal process, can also be observed.8,9 The etiology of these conditions appears to be multifactorial,16,17 and further research is warranted to better understand, prevent, and treat these diseases.

Nutrition
Musculoskeletal disorders are common in young dogs but uncommon in cats. The prevalence of musculoskeletal disorders is reported to be 22% in dogs younger than 1 year.12 Of these cases, 20% are thought to be nutrition related.12 Deficiencies in vitamin D or trace elements, excessive calcium or vitamin C, and high energy intake have been discussed as reasons for developmental growth abnormalities.12–15 Calcium and energy are two of the most important factors.16 Commercially prepared large-breed puppy diets that meet the nutritional guidelines of the Association of American Feed Control Officials (AAFCO) and are labeled “complete and balanced” should be fed to growing large-breed dogs until they reach approximately 80% of their mature size.18 Smaller dogs can be fed puppy diets until approximately 1 year of age and should then be switched to an adult diet.12,18–20 Recommended levels of key nutrients in a diet to prevent orthopedic disease in large-breed puppies are summarized in TABLE 1.

Calcium
Excess feeding (>3% of dry matter) of calcium increases the risk of slowing chondrocyte maturation, which can lead to the development of retained cartilaginous cores and, in some cases, angular limb deformity.16 Osteochondrosis dissecans

Table 1: Recommended Levels of Key Nutrients in a Diet to Prevent Orthopedic Disease in Large-Breed Puppies

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Recommended Level</th>
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<tbody>
<tr>
<td>Calcium</td>
<td>0.8–1.2% of dry matter</td>
</tr>
<tr>
<td>Protein</td>
<td>20–30% of dry matter</td>
</tr>
<tr>
<td>Fat</td>
<td>10–20% of dry matter</td>
</tr>
<tr>
<td>Carbohydrates</td>
<td>20–30% of dry matter</td>
</tr>
</tbody>
</table>

*Dr. Abood discloses that she has received financial support from Abbott Laboratories, Hill’s Pet Nutrition, and Nestlé Purina PetCare Company.

Abstract: This article reviews nutritional and hormonal influences, diseases with uncertain etiology, and hereditary disorders affecting the growth and development of the long bones in dogs and cats.
Disorders of the growth plate and diseases of young bone and cartilage can be affected by unbalanced nutrition.

QuickNotes

**Phosphorus**

Imbalances of dietary phosphorus intake can affect calcium homeostasis and may influence bone metabolism. Low dietary phosphorus is uncommon but has been reported to increase calcium and phosphorus absorption in the gut. Clinical signs can include poor weight gain and reduced growth rate. By contrast, excessively high phosphorus levels can stimulate hormone secretion (secondary hyperparathyroidism), which decreases calcium absorption. The result is poorly calcified, soft bones that are predisposed to pathologic fractures. It is therefore recommended to maintain a dietary calcium:phosphorus ratio between 1.1:1 and 2:1.

**Vitamin D**

Vitamin D and its metabolites are important in the regulatory mechanism of calcium metabolism and skeletal development in dogs and cats. Vitamin D deficiency leads to a decreased plasma concentration of calcium. Chronic hypocalcemia, especially during growth, causes inadequate mineralization of bone so that the cartilaginous matrix in the growth plate fails to calcify. The resultant disease, rickets, is characterized by soft bones, lameness, pain, angular limb deformation, and pathologic fractures. Radiographs typically show thickened physeal cartilage plates and “cupping” of the metaphyseal bone with a dense sclerotic margin (FIGURE 1). Histology shows severe enlargement of the chondrocytes in the growth plate. Rickets is extremely rare in dogs and cats being fed commercially avail-

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**TABLE 1** Recommended Ranges of Key Nutrients and Energy Content in a Diet for Large-Breed Puppies

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Percentage on Dry Matter Basis</th>
<th>Amount Fed as Canned</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein</td>
<td>29–34</td>
<td>6%–8%</td>
</tr>
<tr>
<td>Fat</td>
<td>11–16</td>
<td>2%–4%</td>
</tr>
<tr>
<td>Calcium</td>
<td>0.8–1.4</td>
<td>—</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>0.7–1.2</td>
<td>—</td>
</tr>
<tr>
<td>Fiber</td>
<td>2.4–5.6</td>
<td>1%</td>
</tr>
</tbody>
</table>

KJ/g = kilojoule per gram; DMB = dry matter basis

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**FIGURE 1**

**Rickets in a 5-year-old chimpanzee.** Note the extremely widened and radiolucent physes (arrows) of the pelvis, femora, and tibiae, typical of this disease. Excessive vitamin D deficiency may prevent the calcification process in the metaphyses. This disease is extremely rare in dogs and cats in developed countries because of the availability of well-balanced commercial veterinary diets. (Courtesy of Dr. Tobias Schwarz, University of Wisconsin-Madison)
Adipocytes have been shown to be related to obesity as well as secondary changes in joints and prolonged epiphyseal plate maturation. Adipocytes have been shown to be related to the development of arthritis by the release of adipokines, which are linked to immunity and inflammation. Feeding commercial dry diets with an energy density below 4.0 kcal/g can help minimize the negative effects of high calorie intake. Restricted total dietary intake has been associated with decreased development of hip dysplasia and arthritis in several joints, as well as with increased life span and delayed onset of chronic disease.

Other Nutrients
Copper, zinc, and manganese also affect epiphyseal growth. Although rare, copper deficiency can impair the metabolism of collagen and elastin. Zinc deficiencies can lead to impaired growth and skeletal development as well as skin problems and decreased immune function.

Experimentally, manganese deficiency has been shown to promote the development of thickened, short, disproportionate long bones; abnormal ossification of the skull; and otoliths in the inner ear. However, no clinical cases of manganese deficiency have been reported.

Hormonal Influences
As in people, congenital hypothyroidism has been documented to cause severe abnormalities in dogs. Congenital hypothyroid dwarfism that presented with tetraparesis due to vertebral physeal fracture. This dog also showed skeletal immaturity in the long bones. Greco et al reported congenital hypothyroid dwarfism in five giant schnauzers of the same family. When treated with levothyroxine before 4 months of age, puppies responded with complete remission. A similar report in toy fox terriers described the heritability of congenital hypothyroidism; a DNA-based carrier test has been developed and is used to prevent breeding of affected dogs.

Sex hormones influence the time of growth plate closure. Compared with animals that do not undergo gonadectomy, male and female kittens and puppies neutered at the age of 7 weeks can have significantly delayed physeal closure and greater radial length. Bone volume in these animals is also decreased, presumably due to decreased osteoblast function.

Slipped Capital Femoral Epiphysiolysis
The skeletal changes associated with prepubertal gonadectomy have been associated with an increased risk for developing SCFE in cats and, rarely, in dogs. Animals are lame in the affected hindlimb; the duration and severity of the lameness vary. Serial radiographs are usually used to make the diagnosis, and it has been demonstrated that dysplasia can occur in multiple physes in the same animal. The distal femoral and distal radial epiphyses can be affected concurrently with SCFE. Histologic changes in the growth plate of cats with SCFE include loss of normal columnar architecture of chondrocytes, chondrocyte clustering, granulation tissue within the growth plate, physeal clefts with necrosis, and wider growth plates (up to twice normal). These findings are similar to those in SCFE in people (obese, adolescent boys are most commonly affected). In a study of SCFE in cats, all
The Epiphyseal Plate: Influences and Disorders

QuickNotes

The recommended percentages, on a dry matter basis, of key nutrients in a diet for large-breed puppies to minimize the risk of orthopedic disease are as follows: protein, 29% to 34%; fat, 11% to 16%; mean energy, 3.4% to 4.1%; calcium, 0.8% to 1.4%; phosphorus, 0.7% to 1.2%; fiber, 2.4% to 5.6%.

Affected animals were 5 to 24 months old, 85% were male, 23% were Siamese, and 90% were obese. These cats were neutered between 4 and 8 months of age. Depending on the clinical signs, conservative treatment or surgery (e.g., femoral head and neck ostectomy) may be indicated.

Diseases With Uncertain Etiology

Femoral Neck Metaphyseal Osteopathy in Cats

Cats with metaphyseal osteopathy develop necrosis in the proximal metaphysis, which eventually results in a secondary, pathologic fracture of the femoral neck (Figure 4). The condition may be unilateral or bilateral. Adult male cats (neutered and intact) are overrepresented. Lameness originates from the coxofemoral joint. Unlike SCFE, radiographs show characteristic chronic degenerative changes in the femoral neck region, including areas of radiolucency, bone resorption, an “apple-core” appearance, or an irregular radiolucent fracture line. Several etiologies have been proposed: traumatic fracture with secondary bone resorption, avascular necrosis, osteomyelitis, feline herpesvirus, and changes secondary to SCFE. Histology does not show the typical features of SCFE but may include bone necrosis and microfracture, fibrosis, metaplasia, osteomyelitis, and synovitis of the surrounding connective tissue. Excision of the femoral head and neck carries a good prognosis.

Hypertrophic Osteodystrophy

Hypertrophic osteodystrophy is seen in growing large- or giant-breed dogs with open
epiphyseal plates. Affected animals present with intermittent lameness, fever, and extremely painful, swollen distal metaphyses of the long bones. A radiolucent line parallel to the growth plate (pseudophyseal line) is the hallmark of the disease. Possible causes of this line include increased leukocyte activity, bone lysis, and failure of ossification of the hypertrophic zone. Large, swollen, dense metaphyses and periosteal exostosis complete the classic radiographic appearance.

The etiology of hypertrophic osteodystrophy is unknown. Nutritional (vitamin C deficiency) and viral (distemper virus) factors have been suggested. However, these theories remain unproven. The disease is usually self-limiting, but affected dogs can be extremely ill. Treatment consists of supportive care. Permanent bone changes, angular limb deformities secondary to asymmetric or asynchronous growth or bridging of bone, and retardation of axial growth have been reported.

Osteochondrosis
Osteochondrosis is a multifocal disease with incomplete endochondral ossification of the articular-epiphyseal cartilage. It has also been reported to affect the cartilage of the epiphyseal growth plate. The etiology of osteochondrosis is not clear. Early in an animal’s development, blood vessels, nerves, and lymphatics supply the epiphyseal cartilage. As the animal ages and gains weight, the number of blood vessels decreases until the cartilage becomes avascular. Studies of pigs and chickens in which ischemia was induced have demonstrated that, at some point during growth, the viability of cartilage cells depends on the integrity of cartilage canal vessels. Aside from ischemia, other etiologies, such as hereditary factors, rapid growth, and nutritional imbalances, have been suggested to cause osteochondrosis.

Osteochondrosis results in failure of matrix calcification, with retention of cartilage rather than conversion to bone, leading to a thickened, weaker growth plate. Excessive caloric intake has been suggested to be a risk factor in the development of increased articular cartilage thickness, but high dietary protein alone is unlikely to be the cause of osteochondrosis in dogs. Damage to the cartilage structures, retained cartilaginous cores, and asymmetric growth may be consequences of this process. Histologic examination of the lesions may reveal areas of necrotic chondrocytes in the reserve zone, close to necrotic vascular channels.

When osteochondrosis affects the articular surface, lesions consist of necrotic cartilage with a cleft extending from the subchondral bone to the articular surface, resulting in synovitis, joint effusion, and clinical signs of lameness. This form of osteochondrosis is called osteochondrosis dissecans (FIGURE 6). Treatment consists of removal of loose cartilage and debridement of subchondral bone. The prognosis varies depending on the affected joint and advancement of arthritis.

Retained Cartilaginous Core
Retained cartilaginous cores are most commonly seen in the distal ulna of growing large- and giant-breed dogs (FIGURE 7). These cores are a developmental disorder of endochondral ossification in which physeal calcification is disturbed, resulting in decreased bone growth of the ulna. The etiology is uncertain; however, dietary imbalances and a form of osteochondrosis have been suggested. Depending on the severity, affected dogs present with variable degrees of lameness and multiple deformities, including valgus deformity of the carpus and cranial bowing of the radius (with or without lameness). A radiolucent cartilage core in the center of the distal ulnar physeal can be seen radiographically (FIGURE 7). Treatments include return to a com-
The Epiphyseal Plate: Influences and Disorders

Complete, balanced diet and cessation of excessive dietary supplements in growing dogs. If clinically indicated, corrective osteotomy should be performed. The prognosis varies according to the degree of deformity and lameness at initial presentation.

Ununited Anconeal Process
Unlike small-breed dogs, in large-breed dogs, the anconeal process develops from a separate center of ossification. Failure of this center to fuse with the proximal ulna by 5 months of age is characteristic of ununited anconeal process (Figure 8). Disturbance of endochondral ossification, resulting in osteochondrosis, is presumed to be the underlying etiology. However, joint incongruity, growth plate trauma, excess calcium, excess weight, and genetic and hormonal factors have also been discussed. This disease is hereditary in some German shepherd lines, in which three dominant genes have been suggested for this trait. Treatment options are controversial and include medical management, excision of the anconeal process, attachment with a lag screw, and ulnar osteotomy. Combination of the latter two methods possibly provides a better prognosis than other treatment options.

Hereditary Disorders Affecting the Epiphyseal Plate
Incomplete Ossification of the Humeral Condyle
Incomplete ossification of the humeral condyle (IOHC) was first described in 1990 by Drapé. Lateral radiograph of the distal radius and ulna in a 5-month-old Newfoundland with a retained cartilaginous core. Note the "flame-like" radiolucent area in the center of the ulnar growth plate enclosed by two radiodense lines (box), which is diagnostic for this condition.

QuickNotes
Gonadectomy at an early age can lead to significantly delayed physisal closure and slipped capital femoral epiphysiolysis (SCFE) in cats and, rarely, dogs. The distal femoral and distal radial epiphysis can be affected concurrent with SCFE.
Fusion of the two centers of ossification normally occurs at around 10 weeks of age. With IOHC, minor trauma may lead to condylar fracture and acute onset of lameness. Although the disease occurs early in life, the age at presentation is commonly between 3 and 8 years. This condition occurs mainly in spaniels, for which an autosomal recessive mode of inheritance has been suggested. It is also described for rottweilers, English pointers, wachtelhunds, German wirehaired pointers, Bernese mountain dogs, Newfoundlands, giant schnauzers, Polish lowland sheepdogs, and Labrador retrievers.

Radiographs should be obtained in 15° cranio medial to 15° caudolateral views. Both legs should be examined. The affected leg usually shows a fracture of the humeral condyle (FIGURES 9). If radiographs are not sufficient, computed tomography or arthroscopy may help diagnose a bilateral condition (FIGURE 10). Lag screw fixation for the fractured leg or prophylactic transcondylar fixation for the contralateral leg is the best method of treatment; however, if radiographs are not sufficient, computed tomography or arthroscopy may help diagnose a bilateral condition (FIGURE 10).

Osteochondrosis, incomplete ossification of the humeral condyle, retained cartilaginous core, and ununited anconeal process should be on the differential diagnosis for young animals with orthopedic problems affecting the growth plate.
nonunion is common with this disease.\textsuperscript{60,64} It has been suggested that dense cancellous bone, fibrous tissue, or undue motion may prevent appropriate or complete healing.\textsuperscript{60}

**Ocular-Skeletal Dysplasia**

There are several reports of ocular-skeletal dysplasia in dogs.\textsuperscript{65–67} This condition occurs mainly in Labrador retrievers, but it has also been described for Samoyeds and German shepherds. Barnett and colleagues\textsuperscript{68} reported the mode of inheritance as an autosomal recessive defect. Affected dogs present with a typical “downhill conformation” (front limbs shorter than hindlimbs; Figure 11) and bony abnormalities, such as bone shortening in the forelegs with malformation of the humeral condyles, fracture of the lateral portion of the humeral condyle, asynchronous growth of the radius and ulna, varus deformity and secondary degenerative joint disease of the elbows, ununited and hypoplastic anconeal and/or coronoid process, and carpus valgus (Figure 12). Hip dysplasia and retarded tibial growth may also occur.\textsuperscript{65–67} The degree of lameness varies with severity. Cortical thickness and density of the bones can appear to be reduced, and the epiphyses and cuboid bones are larger and misshapen compared with those of normal littermates.

The ocular component of this disease may present as night blindness. Ocular pathology includes cataracts, retinal dysplasia, and retinal detachment. The degree of vision impairment depends on severity of the lesions.\textsuperscript{65–67} Carrig and colleagues\textsuperscript{66} established a breeding colony of Labrador retrievers to further investigate the mode of inheritance of this disorder. The heterozygotes were found to have a clinically normal skeleton with mild ocular abnormalities, while homozygotes showed clinical signs of both ocular and skeletal dysplasia. It was concluded that abnormalities resulted from a single gene with recessive effects on the skeleton but with incomplete dominant effects on the eyes.\textsuperscript{66}

Depending on the degree of orthopedic and/or ophthalmologic disease, surgical treatment may be beneficial. The prognosis for complete restoration of normal orthopedic function is guarded to poor, and affected dogs should not be bred.\textsuperscript{67}

**Chondrodysplasia**

Chondrodysplasia, also commonly called dwarfism, has been described in Great Danes, Scottish deerhounds, Alaskan malamutes, Norwegian elkhounds, and miniature poodles.\textsuperscript{5} Other breeds may be affected. In contrast to ocular-skeletal
dysplasia, in which limb shortening is mild, chondrodysplasia is characterized by severely shortened limbs, normal body length, and normally sized skull, leaving the impression of a disproportionate dwarf5 (FIGURE 13). The disease is genetically transmitted as a simple autosomal recessive trait. In Alaskan malamutes, it is combined with a permanent macrocytic, hypochromic anemia.6,7 Affected animals are usually not lame.

Chondrodysplasia appears radiographically similar to rickets and can easily be misinterpreted. Radiographic changes include flaring of the distal metaphyseal borders of the radii and ulnae. Prominent curvature of the front limbs and carpus valgus are present. For Great Danes, flaring of all metaphyses, including a “trumpet-like” flaring of the distal tibia, has been described.6 Ossification of vertebral end plates and/or centers of ossification is delayed. Epiphyseal plates show delayed closure with shortened, disorganized columns of chondrocyte proliferation, swollen chondrocytes, and diminished endochondral ossification. Affected chondrocytes reveal a markedly irregular dilatation of cisternae of the rough endoplasmic reticulum.5 There may be a generalized defect involving all hyaline cartilage throughout the body. If no severe abnormalities occur, the quality of life of affected dogs may be good. However, if animals show signs of osteopenia, kyphosis, joint laxity, reduced diameter of the tracheal lumen, or angular limb deformities, the prognosis is guarded.7

Epiphyseal Dysplasia

Epiphyseal dysplasia is a hereditary condition characterized by delayed and irregular ossification of the cartilage of the epiphysis.

**QuickNotes**

Typical radiographic findings for epiphyseal dysplasia include shortening of long bones and widening of the metaphysis, lack of radiopacity, and ossification of cartilage. Affected animals may present with painless swelling, typically on the medial aspect of the joint, limited range of motion, and recurrent locking of the joints. Radiographic findings include shortening of long bones and widening of the metaphysis, lack of radiopacity, and ossification of cartilage (ellipse). Secondary joint degeneration can be expected at a later stage.
The Epiphyseal Plate: Influences and Disorders

Alterations of bone and cartilage with hyperplastic chondrocytes and fibrous tissue interrupted by phases of normal endochondral ossification can also be seen. This condition may lead to pain and interference with function or to angular limb deformity. Corrective surgery may be required to restore function.

Premature Closure of the Distal Ulnar Physis in Skye Terriers

Thirty years ago, Lau described premature closure of the distal ulnar physis in Skye terriers as a hereditary disease. Twenty-three dogs, all the offspring of two females and four males, presented with forelimb lameness between 3 and 5 months of age. Radiographic and physical examination findings included carpus valgus, lateral subluxation of the radial head, circumduction of the elbows, and decreased range of motion of the elbow joint. Lau provided evidence of a recessively inherited trait as the etiology for this disease. A similar syndrome, although not well described in genetic terms, is seen in Welsh corgis, basset hounds, and other chondrodystrophic breeds.

Multiple Cartilaginous Exostoses

MCE are “mini growth plates” found in random areas of metaphyseal bone. They are identified in cats and dogs of any age as bony proliferations on the body and spinous processes of the vertebrae; the processes of the scapula, sternum, ribs, and ischii; and the petrous portion of the temporal bones. The disease is commonly an incidental finding because animals do not usually show clinical signs. This condition was formerly called osteochondromatosis. Currently, a solitary lesion is described as osteochondroma, whereas multiple lesions are referred to as MCE. These malformations are a result of disturbed endochondral ossification at the periphery of the growth plate with abnormal, “benign” development of cartilage and fibrous connective tissue. MCE seem to be associated with FeLV infection in cats.

MCE are reported to occur predominantly in Siamese cats, in which the temporal bone, vertebral bodies, and spinous processes of the scapula, vertebrae, sternum, ribs, and ischium can be affected. The disease is a heritable entity in dogs and has been identified in the vertebrae, ribs, and long bones. Interestingly, lesions are not observed to affect long bone growth. Dogs and cats may have no clinical signs, unless the exostoses cause dysfunction of a joint or vital structure such as the trachea or spinal cord. Affected animals may present with myelopathy, commonly at younger than 1 year. Depending on the severity of the lesion, progressive neurologic signs may be seen. Histologically, lesions are consistent with a site of endochondral ossification. Clinical presentation and radiographic and histologic findings help to differentiate MCE from other benign polyostotic exostoses such as tumoral calcinosis or canine disseminated idiopathic skeletal hyperostosis. While the disease is often aggressive and carries a poor prognosis in cats, dogs usually have a good prognosis because the process ceases at maturity. However, in rare cases, transformation into malignant neoplasia several years after initial diagnosis has been reported.

Mucopolysaccharidosis

MPS is a rare storage disease in which different lysosomal enzyme defects result in the inability to degrade glycosaminoglycans. Endochondral ossification is disturbed, resulting in skeletal abnormalities.

Six subtypes of MPS (I, II, IIIA, IIIB, VI, VII) have been described in dogs and cats. Mixed-breed dogs, German shepherds, Plott hounds, rottweilers, Labrador retrievers, wire-
haired dachshunds, New Zealand huntaways, miniature poodles, Chesapeake Bay retrievers, and miniature schnauzers have been reported to be affected by different types of MPS. The only MPS disorder with mostly neurologic signs is MPS IIIA, reported in wirehaired dachshunds and New Zealand huntaways. MPS I, MPS VI, and MPS VII have also been observed in cats. MPS VI occurs in Siamese cats and follows an autosomal recessive inheritance. Animals present with dwarfism, a disproportionately small and broad maxilla, small ears, large paws, hip dysplasia, crepitus and hypermotility in multiple joints, and fusion of vertebral bodies with subsequent neurologic deficiencies (Figures 16 and 17). Affected cats excrete high amounts of dermatan sulfate in their urine. Dermatan sulfate glycosaminoglycan (formerly called mucopolysaccharide) accumulates abnormally in several of the different subtypes of MPS disorders and is found mostly in skin tissue but also in blood vessels, heart valves, tendons, and the lungs. Breed-specific DNA tests have been established for the diagnosis of affected animals and for carrier detection. Treatment and prognosis depend on the type and severity of MPS. MPS III is usually incurable, and most animals are euthanized before 5 years of age.

**QuickNotes**

Mucopolysaccharidosis is associated with dwarfism, small ears, large paws, pathologic changes in multiple joints, and neurologic deficiencies.
Conclusion
A thorough understanding of the anatomy and physiology of the growth plate is necessary to understand the effect of nutritional imbalances, hormonal influences, and hereditary disorders on developing long bones. Early correction of each specific condition is warranted to avert permanent damage. Dogs and cats that are affected with a familial or potentially heritable problem should not be considered for breeding purposes.

Acknowledgments
To Dr. Cheri Johnson, DVM, MS, DACVIM, for editorial work, and Sandra Schallerger, Dr.med.vet., for technical support.

References
1. What is the recommended percentage of calcium on a dry matter basis in food for large-breed puppies?
   a. 0.3% to 1.5%  d. 7.4% to 8.1%
   b. 0.8% to 1.4%  e. 25% to 30%
   c. 3.2% to 3.8%

2. What could be the effect of feeding ad libitum diets with high amounts of protein, calcium, phosphorus, and vitamin D?
   a. retarded cartilage maturation and osteochondrosis
   b. ocular-skeletal dysplasia
   c. rickets
   d. chondromalacia
   e. kidney failure

3. Prepubertal gonadectomy has been associated with which of the following?
   a. diabetes mellitus
   b. MPS
   c. hypothyroidism
   d. SCFE
   e. hair loss

4. Epiphyseal dysplasia is characterized by which of the following?
   a. a double physeal line
   b. lack of opacity and ossification of cartilage
   c. a circular defect in the articular cartilage
   d. flaring of the distal metaphyseal borders of the radius and ulna
   e. endosteal proliferation

5. Ocular-skeletal dysplasia occurs most commonly in
   a. basset hounds.
   b. German shepherds.
   c. Welsh corgis.
   d. rottweilers.
   e. Labrador retrievers.

6. ______ is a finding in dogs with chondrodysplasia.
   a. Osteopenia and angular limb deformity
   b. Kyphosis
   c. Joint laxity
   d. Reduced diameter of tracheal lumen
   e. all of the above

7. Which statement regarding hypertrophic osteodystrophy is false?
   a. It is a self-limiting disease.
   b. The hallmark is a double physeal line.
   c. Swollen, extremely painful distal metaphyses are common.
   d. It is common in chondrodystrophic breeds.
   e. The etiology is unknown.

8. ______ has/have been implicated in the etiology of osteochondrosis.
   a. Nutritional factors
   b. Hereditary factors
   c. Rapid growth
   d. Vascular impairment
   e. all of the above

9. ______ has/have been associated with MCE in cats.
   a. FeLV
   b. Feline infectious viremia
   c. A single dominant autosomal gene
   d. Multiple genes
   e. A single gene with recessive effects on the skeleton

10. Which is not a clinical sign of MPS?
    a. large ears
    b. dwarfism
    c. disproportionate and broad maxilla
    d. hip dysplasia
    e. fusion of vertebral bodies with subsequent neurologic signs