Chapter 33

Developmental Orthopedic Disease of Dogs

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"The beginning is the most important part of the work."
Plato

The Dance of the Solids

The Polymers, those giant Molecules
Like Starch and Polyoxymethylene,
Flesh out, as protein serfs and plastic fools,
This Kingdom with Life's Stuff. Our time has seen
The synthesis of Polyisoprene
And many cross-linked Helixes unknown
To Robert Hooke; but each primordial Bean
Knew Cellulose by heart. Nature alone
Of Collagen and Apatite compounded Bone.
John Updike

CLINICAL IMPORTANCE

The prevalence of musculoskeletal disorders for all dogs at multicenter referral practices has been reported to be approximately one in four, with 70% of these disorders involving the appendicular skeleton (Johnson et al, 1994; LaFond et al, 2002). Furthermore, the prevalence of musculoskeletal problems in dogs less than one year old in all breeds is about 22%, with 20% possibly having a nutrition-related etiology.[a] Developmental orthopedic disease (DOD) includes a diverse group of musculoskeletal disorders that occur in growing animals (most commonly fast-growing, large- and giant-breed dogs whose adult weight will exceed 25 kg) and that are sometimes related to nutrition. Canine hip dysplasia and osteochondrosis make up the overwhelming majority of the musculoskeletal problems with a possible nutrition-related etiology.[a]

Canine Hip Dysplasia

Canine hip dysplasia is the most frequently encountered orthopedic disease in veterinary medicine with heritability and a potential nutrition-related etiology (Johnson et al, 1994). Canine hip dysplasia is an abnormal development, or growth, of the hip joint (**Figure 33-1**) manifested by varying degrees of laxity of surrounding soft tissues, instability of the joint and malformation of the femoral head and acetabulum with osteoarthrosis (Brinker et al, 1990). The number of cases of canine hip dysplasia is estimated to be in the millions worldwide (Corley and Hogan, 1985).

Osteochondrosis

Osteochondrosis is widespread among people and young, rapidly growing, domesticated species. Generally, osteochondrosis is a disruption in endochondral ossification that results in a focal lesion (Brinker et al, 1990a). Osteochondrosis occurs in the physis and/or epiphysis of growth cartilage, and may be considered a generalized or systemic disease. Clinical signs of osteochondrosis are related to the severity and location of disease.

When osteochondrosis affects physeal cartilage, it may cause growth abnormalities in long bones such as angular limb deformities. Osteochondrosis of articular epiphyseal cartilage

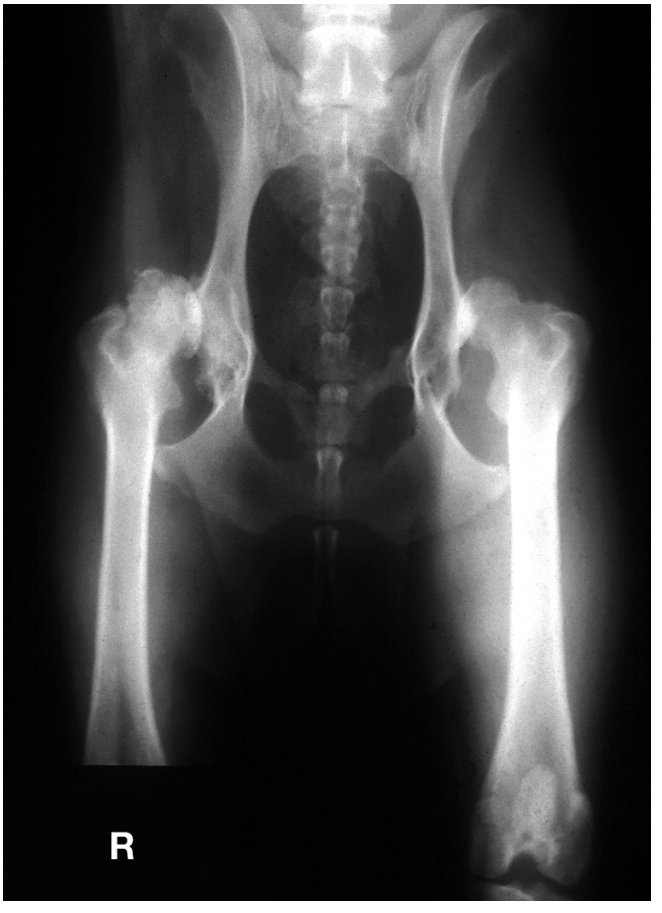

Figure 33-1. Progression of joint disease in a dog with rear-limb lameness due to severe bilateral hip dysplasia. This ventrodorsal radiograph shows degenerative joint disease in both coxofemoral joints. The right hip has advanced osteophyte formation on the femoral neck. The right acetabular cup and femoral head have remodeled to form a pseudoarthrosis. The left femoral neck also has osteophyte formation and the hip is luxated craniodorsally.

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commonly occurs in the shoulder (proximal humerus, **Figure 33-2**), stifle (distal femur), hock (talus) and elbow (distal humerus). Acute inflammatory joint disease (or degenerative joint disease) may ensue subsequent to development of osteochondrosis when the cartilage surface is disrupted and subchondral bone is exposed to synovial fluid. Inflammatory mediators and cartilage fragments are released into the joint (osteochondritis dissecans), which perpetuates the cycle of degenerative joint disease (Hill et al, 1984) (Chapter 34). Other disease processes such as spondylolisthesis, intra-articular fracture, complete or partial epiphysiolysis and deformed joint surfaces have been associated with osteochondrosis but their etiology is still undetermined.

Elbow Dysplasia

Elbow dysplasia describes the four main developmental dysplastic conditions that are frequently diagnosed in the elbow joint: 1) osteochondrosis of the medial condyle, 2) fragmented medial coronoid process, 3) ununited anconeal process and 4) elbow incongruency, due to a relatively short ulna. These conditions result in severe lameness although the clinical manifestation varies with the breed, the underlying diagnosis, the typical exercise pattern and the amount of osteoarthritis.

Breed specific distribution of the elbow dysplasia types has been recognized and elbow dysplasia has a complex hereditary background. Each of the conditions included may occur independently in the canine population, although they can occur in the same elbow in different combinations.

The frequency and severity of elbow dysplasia are also subject to environmental factors such as nutrition, body weight and exercise. The etiology is similar to what has been described for osteochondrosis-related conditions. It seems that fragmented medial coronoid process is not influenced by nutrition as much as the other three conditions.

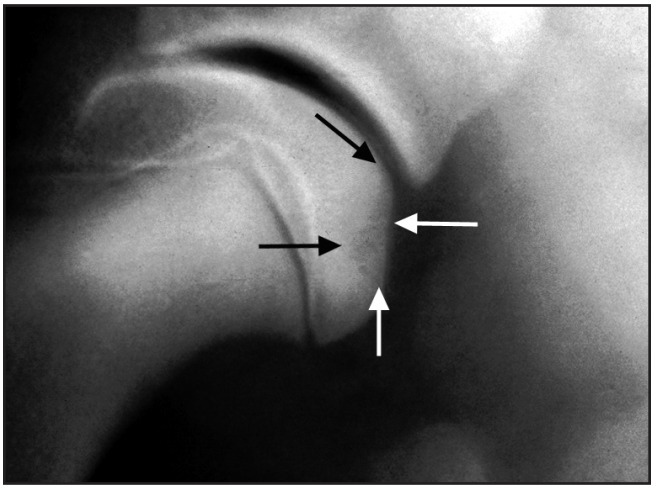

Figure 33-2A. Radiograph of the proximal humerus of a nine-month-old male Labrador retriever examined for forelimb lameness. The radiolucent area (arrows) indicates disrupted endochondral ossification and subchondral bone necrosis associated with osteochondrosis.

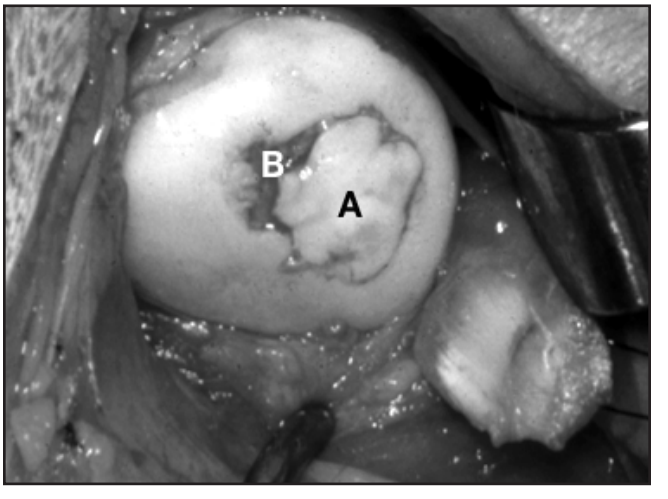

Figure 33-2B. Intraoperative view of an osteochondritis dissecans lesion in the articular epiphyseal cartilage of the proximal cartilage of the same dog. Note the cartilage flap (A) and exposed subchondral bone (B) where a portion of the cartilage flap is missing.

Growth Velocity

Puppy growth can be measured in height/length and body weight. Typically, when discussing growth in people, we assume an increase in height. Rapid growth in children means they reach an above average height at a certain age. Growth curves have been established per population and gender. However, in dogs, growth is usually measured in terms of body weight. When considering rapid growth, the risk is that real growth (increased height/length) becomes confused with the development of relative overweight during growth, resulting from excess food intake. This can become an issue when discussing risk factors for developmental disease conditions of the skeleton. The statement in the literature that overnutrition results in rapid growth is typically supported by body weight data and not by height/length measures. Because osteochondrosis conditions may be related to disturbed height/length growth and not just excessive weight, this discrepancy between human and canine growth references needs to be taken into account when interpreting growth. Some breed standards for popular dogs (including Labrador retrievers) tend to reflect overweight. This has to be taken into account when calculating energy requirements during growth relative to adult body weight.

Box 33-1. Use of DEXA to Assess the Skeleton.

The ability to make repeated, accurate assessments of body composition is crucial to the investigation of many key nutritional issues of cats and dogs. In a research setting, dual energy x-ray absorptiometry (DEXA) allows the body to be viewed as three compartments: bone mineral, fat tissue and lean tissue. The ability to evaluate changes in these three compartments independently greatly benefits the study of growth, obesity and geriatrics.

DEXA uses x-rays of two different energy levels (70 and 140 kVp) to distinguish the nature and amount of each tissue in the part of the body being scanned. The x-ray source below the table and the detector above the table move in concert to measure the amount of radiation passing through the subject. Because x-rays of different energy levels are impeded differently by bone mineral, fat and lean tissue, it is possible to calculate the quantity of each tissue in each area scanned.

The accuracy of DEXA in companion animals is supported by the good correlation between values obtained from DEXA and chemical analysis.

The Bibliography for **Box 33-1** can be found at www.markmorris.org.

PATIENT ASSESSMENT

History and Physical Examination

Breed and familial history are important predisposing factors for DOD. For mixed-breed dogs, it is useful to know the breed of the stud and bitch as well as historical wellness of offspring. If possible, it may be helpful to gather information pertaining to skeletal abnormalities of previous litters from the same bitch and stud. If it is anticipated that the puppy will have an adult weight of more than 25 kg, it should probably be considered at risk.

Food intake and history should be evaluated as described in Chapter 1. Any treats and supplements fed to the patient should be scrutinized closely, paying special attention to the calcium and energy intake. It is critical to calculate, or closely estimate, metabolizable energy (ME) and calcium and phosphorus intake to provide good advice for feeding growing large- and giant-breed dogs.

Puppies should be weighed during the initial visit and all subsequent visits to help monitor their growth rate. A body condition score (BCS) should be determined and recorded at each visit (Chapter 1). Attention to abnormal changes in weight or BCS will help in assessing and managing growing dogs. In some cases, graphs of body weight and BCS may prove useful in recognizing variances from desired goals. BCS does not always reliably indicate overfeeding, because many young dogs react to higher energy intake with an accelerated growth rate.

Before a physical examination is conducted, historical information should be gathered about the degree, if any, of perceived lameness, the affected limb(s), duration of lameness and any peculiarities regarding the lameness. Following historical evaluation, the patient should be observed at rest for any gross conformational abnormalities. Next, the patient should be observed in motion to ascertain the degree of lameness and location of involvement (Brinker et al, 1990b).

If a locomotor defect is confirmed, the etiology should be determined. To determine the cause, the examination should include: 1) palpation of limbs for asymmetry, swelling, heat and sensitivity, 2) deep palpation of long bones, 3) flexion/extension of joints to determine range of motion, crepitation, instability and sensitivity and 4) neurologic evaluation. Even after a thorough physical examination, the exact cause of the lameness may remain undetermined.

Radiography

Radiographs should be taken to further define the clinical diagnosis. Radiographic identification of lesions aids in confirming the disease. However, inability to identify lesions by survey radiography does not always negate the presence of disease (Henry, 1992) (**Box 33-1**).

Laboratory Information

Diagnostic tests to detect other diseases that may result in skeletal abnormalities should be considered when appropriate. Confounding diseases such as osteomyelitis, septic emboli and mycotic infection should be considered. DOD is usually typified by a lack of abnormal laboratory findings (Hazewinkel, 1994; Nap and Hazewinkel, 1994).

Uncomplicated cases of DOD rarely have altered complete blood counts. Severe elevations or decreases in white blood cell counts usually indicate other disease processes. If anemia is present, classifying the type may give insight to other causes for select skeletal disorders (e.g., copper deficiency) (Zentek et al,

Table 33-1. Parathyroid hormone (PTH), ionized calcium and 1,25-dihydroxyvitamin D_3 concentrations in different physiologic/disease states.*

States	PTH	Ionized calcium	1,25-dihydroxyvitamin D_3
Apocrine gland tumors of the anal sacs	Low	High	Low
Chronic renal failure	High	Low/normal	Normal/low
High calcium intake	Low	High	Normal/low
Hypervitaminosis D	Low	High	Normal/high
Hypoparathyroidism	Low	Low	Low
Lymphosarcoma	Low	High	Low
Primary hyperparathyroidism	High	High	Normal/high

*Adapted from Feldman EC, Nelson RW, eds. Canine and Feline Endocrinology and Reproduction, 2nd ed. Philadelphia, PA: WB Saunders Co, 1996; 455-493. Hazewinkel HAW. In: Bojrab MJ, ed. Disease Mechanisms in Small Animal Surgery, 2nd ed. Philadelphia, PA: Lea & Febiger, 1993; 1119-1128. Chastain CB, Ganjam VK, eds. Clinical Endocrinology of Companion Animals. Philadelphia, PA: Lea & Febiger, 1986; 192-217.

1991; NRC, 2006).

Occasionally, serum concentrations of calcium and phosphorus may be elevated or decreased during the genesis of DOD. However, absence of calcium or phosphorus perturbations does not rule out a diagnosis of DOD. Conversely, many other disease processes may result in altered calcium or phosphorus homeostasis, which indicates abnormal values are not pathognomonic for a diagnosis of DOD (Nap and Hazewinkel, 1994).

Increased bone remodeling may result in increased serum alkaline phosphatase activity. This parameter is already high in young, growing animals and may not be a very sensitive indicator of ongoing metabolic bone disease. Other enzyme activities in serum are not very useful for diagnosis of DOD. Biochemical markers of human bone metabolism such as type I collagen propeptides, tartrate-resistant acid phosphatase and osteocalcin are useful in research studies; however, the significance for veterinary diagnostics remains to be proven (Robey and Termine, 1990).

Serum and urinary assays of bone markers are of interest as noninvasive alternatives to bone biopsy. Assays that were developed for people have been shown to cross-react in dogs. Serum bone-specific alkaline phosphatase, urinary deoxypyridinoline and N-terminal telopeptide of collagen were measured in dogs with commercial enzyme immunoassays designed for people. Serum osteocalcin and carboxy-terminal cross-linked telopeptide of type I collagen were measured with commercial radioimmunoassays. Significant diurnal rhythms were identified for osteocalcin, bone-specific alkaline phosphatase, carboxy-terminal cross-linked telopeptide of type I collagen and urinary deoxypyridinoline. No clear rhythm was evident for N-terminal telopeptide of collagen. Due to the variability in marker excretion in individual animals, the most appropriate use for these assays is as a screening tool for cohort studies, rather than as a diagnostic or prognostic tool in individual animals (Ladlow et al, 2002). Breed effects cannot be excluded; however, for serum alkaline phosphatase and carboxyl-terminal cross-linked telopeptide of type I collagen, the concentrations were comparable in giant and toy breeds and in beagles (Breur et al, 2004).

Urinalysis results are usually within normal limits for animals with DOD. Advanced techniques, including measurement of calcium and phosphorus partial clearance ratios, may add insight about calcium and phosphorus nutrition, but repeated measurements may be needed for accurate interpretation. Evaluation of other mineral partial clearances may give some insight into dietary excesses or deficiencies. Analysis of urine for markers of bone turnover such as hydroxylysine glycosides, free pyridinolines or pyridinoline cross-links of collagen may prove useful in the future (Eyre, 1996).

Measuring and Interpreting Specific Laboratory Tests

PARATHYROID HORMONE

Interpretation of serum parathyroid hormone (PTH) concentrations from other species has proven that evaluation must be made in conjunction with presenting signs and other biochemical tests such as concentrations of ionized calcium and 1,25-dihydroxyvitamin D_3 (**Table 33-1**). PTH values may be increased, decreased or normal in DOD depending on the etiology. Increased PTH concentrations may be observed in association with renal disease, vitamin D deficiency and states in which insufficient calcium is present in foods. Decreased PTH concentrations may be observed when excess calcium or vitamin D is present in foods, and in other metabolic diseases.

PTH concentration is most accurately measured by a "two-site" immunoassay. This assay eliminates interference by mid-region or terminal fragments that are abundant in animal serum. Single time-point evaluations of PTH may not prove useful in determining the etiology of DOD. Repeated evaluations may yield more useable information, but are probably not cost effective.

CALCITONIN

Calcitonin, a peptide hormone, is released primarily from C-cells of the thyroid gland in response to sudden increased concentrations of ionized calcium in serum. Calcitonin may also be released in response to other stimuli such as gastrin secretion stimulated by food intake (Azria, 1989). Calcitonin is measured by radioimmunoassay (Hazewinkel et al, 1985, 1999). The test is not commercially available and rational interpretation requires multiple sample evaluations. If calcitonin levels are evaluated to investigate the etiology of DOD, results should be compared to normal values for the laboratory and interpreted in conjunction with results of other tests (e.g., PTH, ionized calcium and vitamin D_3 analyses).

VITAMIN D

Vitamin D_3 may be required in foods for dogs because endogenous synthesis may be limited (Hazewinkel et al, 1987; How et al, 1994). Because commercial foods contain added vitamin D_3, and in light of potentially limited endogenous synthesis, measurement of vitamin D_3 in serum may reflect dietary changes rather than specific disease states. 25-hydroxyvitamin D_3 is produced in the liver from vitamin D_3 and is a good indicator of general vitamin D_3 deficiency (Hazewinkel and Tryfonidou, 2002) or excess (Tryfonidou et al, 2003a). Another useful indicator of vitamin D_3 status is measurement of the most biologically active metabolite of vitamin D_3, 1,25-dihydroxyvitamin D_3, which is produced in the kidneys via the 1-α-hydroxylase enzyme. The concentration of 1,25-dihydroxyvitamin D_3 in serum is not a good indicator of vitamin D_3 toxicity (Tryfonidou et al, 2003a); however, it is a more sensitive indicator of deficiency than serum concentrations of 25-hydroxyvitamin D_3.

All metabolites of vitamin D_3 in serum may be measured by high-pressure liquid chromatography. Concentrations should be compared with reference values from laboratories performing the analysis, preferably derived from healthy dogs fed similar foods (Tenenhouse, 1990). A multitude of factors affect production of 1,25-dihydroxyvitamin D_3 including breed differences (Hazewinkel and Tryfonidou, 2002; Tryfonidou et al, 2003a) and laboratory results should be interpreted in conjunction with other physical and biochemical findings (**Table 33-2**). Generally, high concentrations of 1,25-dihydroxyvitamin D_3 indicate low availability of calcium to animals, normal concentrations indicate adequate calcium availability and low concentrations may indicate vitamin D_3 deficiency.

The amount of growth hormone and IGF-1 (insulin-like growth factor-1) may also directly influence vitamin D metabolism. In puppies, these hormones are inherently associated with growth rate and breed, with large-breed puppies having higher levels of these hormones than small breeds. Therefore, both dietary content and breed may influence metabolism of vitamin D and resultant bone development (Tryfonidou et al, 2003b).

Table 33-2. Factors affecting activity of 25-hydroxyvitamin D_3 renal 1-α-hydroxylase.*

Factors	Changes
Acidosis	Decrease
Alkalosis	Increase
Decreased ionized calcium	Increase
Decreased parathyroid hormone	Decrease
Increased 1,25-dihydroxyvitamin D_3	Decrease
Increased calcitonin	Increase/decrease/no effect
Increased growth hormone	Increased vitamin D intake
Increased ionized calcium	Decrease
Increased parathyroid hormone	Increase
Increased phosphate (serum)	Decrease
Increasing age	Decrease
Insulin	Increase
Insulin-like growth factor-1	Increase
Pregnancy	Increase
Prolactin	Increase/no effect
Sex steroids	Increase

*Adapted from Tenenhouse HS. In: Simmons DJ, ed. Nutrition and Bone Development. New York, NY: Oxford University Press, 1990; 164-201. Hazewinkel HAW, Tryfonidou MA. Vitamin D3 metabolism in dogs. Molecular and Cellular Endocrinology 002; 197: 23-33.

Table 33-3. Composition of bone.

Bone is composed of a mineral phase, a non-mineral (organic) phase and a cellular phase

Mineral phase
99% of body calcium
85% of body phosphorus
40-60% of body sodium and magnesium
Ca-P ratio 1.67:1 on a molar basis. Ratio is 2.15:1 on a weight basis (hydroxyapatite crystals = $[Ca_{10}(PO_4)_6(OH)_2]$)

Organic phase
Type I collagen (90% of bone protein)
Noncollagenous protein (cell attachment proteins, proteoglycans, gamma carboxylated gla proteins, growth-related proteins)

Cellular phase
Osteoclasts
Osteoblasts
Osteocytes

CALCIUM

Bone contains 99% of the calcium in the body with the majority in the form of hydroxyapatite crystals (**Table 33-3** and **Box 33-2**). Bone functions physiologically as a structural material and an ion reservoir. When bone acts as an ion reservoir, it is in equilibrium with ionized calcium in serum and under tight homeostatic control.

Calcium homeostasis is maintained by the sum of physiochemical and calciotropic hormonal processes. Calcium in blood is in equilibrium between the ionized state (45 to 50%), a protein-bound state (40 to 45%) and a complexed or chelated state (5 to 10%). Generally, the concentration of ionized calcium is approximately 45 to 50% of the total concentration of calcium in serum over a wide range of total calcium concentrations. The concentration of ionized calcium is the most important determinant of calciotropic homeostatic regulation initiated by the parathyroid gland and the C-cells of the thyroid gland. Sudden increases in ionized calcium concentrations stimulate release of calcitonin from the thyroid gland, whereas decreases in concentrations of ionized calcium stimulate release of PTH from the parathyroid gland. The total concentration of calcium in serum is affected by the interplay of the homeostatic mechanisms involving influx (gastrointestinal [GI] absorption and bone resorption), efflux (GI and renal loss) and skeletal mineralization of the less labile bone pool as outlined below.

When concentrations of ionized calcium are below the normal range:

1. PTH secretion is stimulated, which in turn stimulates conversion of 25-hydroxyvitamin D_3 to the biologically more potent 1,25-dihydroxyvitamin D_3 in the kidneys.

Table 33-4. Summary of reports delineating risk factors for developmental orthopedic disease in dogs.

Diseases	Key points	Interpretation	Risk factors	References*
Hip dysplasia	Rapid weight gain German shepherd dogs First 60 days of age	Rapid weight gain increased risk of hip dysplasia. Dysplastic parents increased risk	Breed Rapid weight gain	Riser et al, 1964
Hip dysplasia	Pups born by cesarean section-hand reared Vaginally born pups, pair-fed bitch milk at 70% free choice	Free-choice feeding resulted in increased weight gain and increased occurrence of hip dysplasia	Free-choice feeding Rapid weight gain	Lust et al, 1973
Hip dysplasia	Labrador retrievers Rapid growth rate	Early fusion of triradiate growth plate in acetabulum	Breed Rapid weight gain	Lust et al, 1985
Hip dysplasia	Weight gain >breed standards	Increased occurrence of hip dysplasia	Rapid weight gain	Kässtrom, 1975
Hip dysplasia	Restricted feeding Labrador retrievers	Restricted feeding decreased occurrence of hip dysplasia	Breed Rapid weight gain	Kealy et al, 1992
Osteochondrosis	Epidemiologic study	Labrador retrievers, Great Danes, Newfoundlands, rottweilers at greatest risk All large breeds at increased risk	Breed Gender Anatomic location	Slater et al, 1991; Slater et al, 1992
Osteochondrosis	Epidemiologic study	Males at higher risk of OCD in shoulder	Gender Calcium content Well water	Dobenencker et al, 1997; Slater et al, 1991; Slater et al, 1992
Osteochondrosis	Great Danes Rapid growth Overnutrition	Rapid growth increased occurrence of developmental orthopedic disease	Breed Rapid growth	Daemmrich, 1991; Hedhammar et al, 1974; Meyer and Zentek, 1992
Osteochondrosis	Great Danes Excess calcium	Excessive calcium intake increased occurrence of developmental orthopedic disease	Breed Excessive calcium	Hazewinkel et al, 1985
Osteochondrosis	Great Danes Excess calcium and phosphorus intake	Excessive mineral intake at young age leads to hypercalcitoninism	Breed Excess minerals	Schoenmakers et al, 2000
Osteochondrosis	Great Danes Excess vitamin D intake	Imbalance of vitamin D metabolites at chondrocytes	Breed Excess vitamin D	Tryfonidou et al, 2003
Developmental orthopedic disease	Large breeds Rapid growth	Excessive energy intake increased occurrence of developmental orthopedic disease	Breed Rapid growth High energy density food	Richardson and Toll, 1997

*Adapted from Hedhammar A, Wu F, Krook L, et al. Overnutrition and skeletal disease. An experimental study in growing Great Dane dogs. Cornell Veterinarian 1974; 64 (Suppl. 5): 1-160. Meyer H, Zentek J. Über den Einfluß einer unterschiedlichen Energieversorgung wachsender Doggen auf Körpermasse und Skelettentwicklung. Journal of Veterinary Medicine A 1992; 39: 130-141. Lust G, Geary JC, Sheffy BE. Development of hip dysplasia in dogs. American Journal of Veterinary Research 1973; 34: 87-91. Lust G, Rendano VT, Summers BA. Canine hip dysplasia: Concepts and diagnosis. Journal of the American Veterinary Medical Association 1985; 187: 638-640. Kasström J. Nutrition, weight gain and development of hip dysplasia. Acta Radiologica Suppl. (Stockholm) 1975; 344: 135-179. Kealy RD, Olsson SE, Monti KL, et al. Effects of limited food consumption on the incidence of hip dysplasia in growing dogs. Journal of the American Veterinary Medical Association 1992; 210: 857-863. Daemmrich K. Relationship between nutrition and bone growth in large and giant dogs. Journal of Nutrition 1991; 121: S114-S121. Dobenencker B, Kienzle E, Matis U. Mal- and overnutrition in puppies with and without clinical disorders of skeletal development (abstract). In: Proceedings. European Society of Veterinary and Comparative Nutrition, Munich, Germany, 1997: 25. Riser WH, Cohen D, Linquist S, et al. Influence of early rapid growth and weight gain on hip dysplasia in the German Shepherd dog. Journal of the American Veterinary Medical Association 1964; 145: 661-668. Richardson DC, Toll PW. Relationship of nutrition to developmental skeletal disease in young dogs. Veterinary Clinical Nutrition 1997; 4: 6-13. Hazewinkel HAW, Goedgebuure SA, Poulos PW, et al. Influences of chronic calcium excess on the skeletal development of growing Great Danes. Journal of the American Animal Hospital Association 1985; 21: 377-391. Schoenmakers I, Hazewinkel HAWH, Voorhout G, et al. Effects of diets with different calcium and phosphorus contents on skeletal development and blood chemistry of growing Great Danes. Veterinary Record 2000; 147: 652-660. Slater MR, Scarlett JM, Donoghue S, et al. Diet and exercise as potential risk factors for osteochondritis dissecans in dogs. American Journal of Veterinary Research 1992; 53: 2119-2124. Slater MR, Scarlett JM, Kaderly RE, et al. Breed, gender, and age risk factors for canine osteochondritis dissecans. Journal of Veterinary Comparative Orthopedics and Traumatology 1991; 4: 100-106. Tryfonidou MA, Stevenhagen JJ, Buurman CJ, et al. Dietary 135-fold cholecalciferol supplementation severely disturbs the endochondral ossification in growing dogs. Domestic Animal Endocrinology 2003; 24(4): 265-285.

2. 1,25-dihydroxyvitamin D_3 stimulates calcium uptake in the gut via receptor-mediated mechanisms.
3. 1,25-dihydroxyvitamin D_3, in conjunction with PTH, stimulates bone resorption and calcium reabsorption in the kidneys.
4. PTH induces phosphaturia.

When concentrations of ionized calcium are above the normal range:

1. Calcitonin secretion is stimulated, PTH secretion is suppressed and 1,25-dihydroxyvitamin D_3 production is not stimulated. Instead, the kidneys produce 24,25-dihydroxyvitamin D_3, which is generally considered biologically inactive.
2. Gut absorption and bone resorption of calcium are not stimulated.
3. Calcitonin decreases osteoclastic activity.
4. Renal calcium excretion is increased.

The equilibrium between the protein-bound and ionized fraction of calcium is affected by a variety of physiologic conditions. Alterations of serum proteins usually do not affect the equilibrium of bound to ionized calcium, but the total calcium may be increased or decreased. Alterations in albumin or total serum protein concentrations should be corrected before calcium values are evaluated (Feldman and Nelson, 1996).

Albumin correction:

Corrected total calcium (mg/dl) = Total calcium (mg/dl) – albumin (g/dl) + 3.5

Total serum protein correction:

Corrected total calcium (mg/dl) – (0.4 x total protein [g/dl]) + 3.3

The percent of total calcium bound to protein may be roughly estimated using differential binding affinities of albumin and globulin (Arnaud and Kolb, 1991).

% protein-bound calcium = 8 x albumin (g/dl) + 2 x globulin (g/dl) + 3

Acidosis shifts the equilibrium toward ionized calcium and is not accounted for by the above equation. Other physiologic perturbations, such as alkalosis, chloride ion concentration and phosphate ion concentration may also affect the equilibrium and are not accounted for by the above equation. Accurate determination of ionized calcium is best performed using ion selective electrodes.

Risk Factors

The genesis of DOD is presumed to be a multifactorial process. The most critical period for development of DOD is during the growth phase, before physeal closure (**Figure 33-3**). Specific factors that are currently thought to increase the risk of DOD in young dogs include: 1) belonging to a large or giant breed (genetics) (>25 kg adult weight), 2) free-choice feeding (management), particularly of high-energy foods (nutrition) and 3) excessive intake of calcium and vitamin D from food, treats and supplements (nutrition) (**Table 33-4**) (Hazewinkel et al, 1985; Daemmrich, 1991; Dobenencker et al, 1997; Hedhammar et al, 1974; Kasström, 1975; Kealy et al, 1992; Lust et al, 1973, 1985; Meyer and Zentek, 1992; Riser et al, 1964; Slater et al, 1991, 1992; Richardson and Toll, 1997; Tryfonidou et al, 2003a).

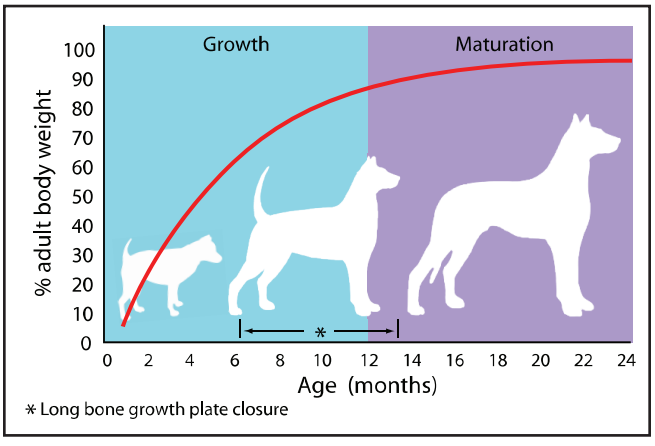

Figure 33-3. Growth phase vs. long bone physeal closure in dogs. Note that weight gain still occurs under the maturation phase although growth plate closure is complete. This is attributable to bone remodeling and especially to the acquisition of adult body mass, with the possible consequence of overweight/obesity.

Box 33-2. Calcium Deposition in Bone.

The actual physical mechanism of calcium deposition in bone is controversial. Evidence suggests the following:

- Calcium and phosphorus exist in metastable equilibrium in solution.
- A nucleation molecule initiates precipitation of solid calcium in collagen.
- Calcium is deposited initially as poorly crystalline type B (carbonate) apatite.
- Initial crystals have brushite properties but as they mature they become more hydroxyapatite in nature.
- Initial nucleation sites are within collagen fibrils.
- Nucleation sites are independent of each other (multicentric).
- Nucleation initiating molecules may include phosphoproteins, proteolipids and complex acidic phospholipids.
- Proteoglycans may inhibit or promote calcification centers.

The Bibliography for **Box 33-2** can be found at www.markmorris.org.

Etiopathogenesis

A variety of mechanisms are plausible in considering the pathogenesis of DOD. No one specific etiology is considered ultimately responsible for all observed clinical manifestations of DOD. Historically, feeding dogs imbalanced foods, especially those deficient in calcium, phosphorus or vitamin D_3, was the main risk factor predisposing them to skeletal diseases such as secondary hyperparathyroidism with subsequent development of osteodystrophia fibrosa (Daemmrich, 1991). Dietary deficiencies are rare in young, growing dogs fed commercial growth foods because most foods are formulated to meet or exceed allowances for specific nutrients (Kallfelz and

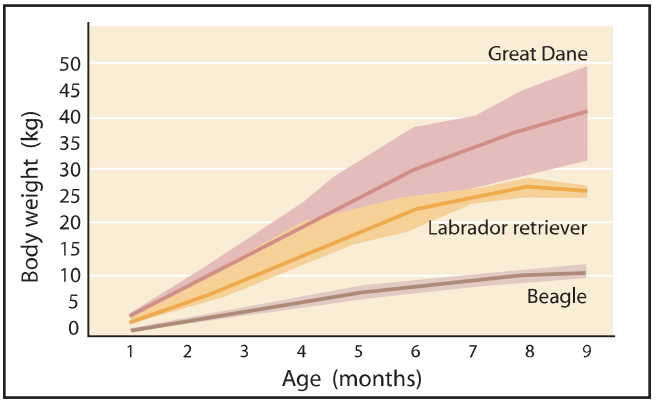

Figure 33-4. Growth curves (weight vs. age) for Great Dane, Labrador retriever and beagle dogs. Note that rapid growth occurs during the first few months in all breeds, but is prolonged in giant-breed dogs such as Great Danes.

Dzanis, 1989). Two popular theories for the pathogenesis of some types of DOD are discussed in the following sections. Specific nutrients are addressed in the Key Nutritional Factors section.

Theory 1: Energy/Growth/Biomechanical Stress

The musculoskeletal system changes constantly throughout life with the most rapid changes occurring during the first few months (**Figure 33-4**) (Hedhammar et al, 1974; Lust et al, 1973; Alexander et al, 1988; Allard et al, 1988; Booles et al, 1991; Booles et al, 1994; Meyer and Zentek, 1989; Romsos et al, 1976; Sheng and Huggins, 1971; Chakraborty et al, 1983; Lavelle, 1989; Rainbird and Kienzle, 1990). The skeletal system apparently is most susceptible to physical, nutritional and metabolic insults during the first 12 months of life because of heightened metabolic activity. Large- and giant-breed dogs are most susceptible to DOD, presumably because of their genetic propensity for rapid growth (Daemmrich, 1991; Meyer and Zentek, 1991).

Present knowledge about energy intake effects on bone growth gives rise to an hypothesis for the etiopathogenesis of growth disorders associated with overfeeding of energy to young, large- and giant-breed dogs. High energy intake directly affects growth velocity via nutrient supply and indirectly through changes in concentrations of growth hormone, IGF-1, triiodothyronine (T_3), thyroxine (T_4) and insulin (Blum et al, 1992; Danforth and Burger, 1989; Eigenmann et al, 1985; Nap, 1993). Dysregulation of these endocrine factors, whether attributable to nutrition, feeding management or genetics, during this critical period of skeletal growth may be responsible for producing an environment in which DOD develops.

Growth hormone and IGF-1 stimulate chondrocyte proliferation and differentiation (Daughaday et al, 1972; Froesch et al, 1985; Glade, 1984; Harris and Heaney, 1969; Hochberg et al, 1989; Isaksson et al, 1987; Eigenmann, 1986). Growth hormone release in non-canids is influenced primarily by energy intake but may also be affected by food protein content, specific amino acids or peptides, exercise and environmental factors (Nap and Hazewinkel, 1994; Blum et al, 1992; Glade, 1984; Eigenmann, 1986). IGF-1 is released systemically primarily from the liver but also locally from chondrocytes in response to growth hormone stimulus. Little is known about dietary influences on growth hormone secretion in dogs; however, young Labrador retrievers had a temporal decrease in concentrations of growth hormone from weaning to 14 weeks of age, followed by an increase in the prepubertal period (Chakraborty et al, 1983). IGF-1 was found in significantly higher concentrations in growing dogs fed free choice compared with animals on restricted feed allowance (Blum et al, 1992), whereas dietary protein intake only weakly influenced IGF-1 levels (Nap et al, 1993).

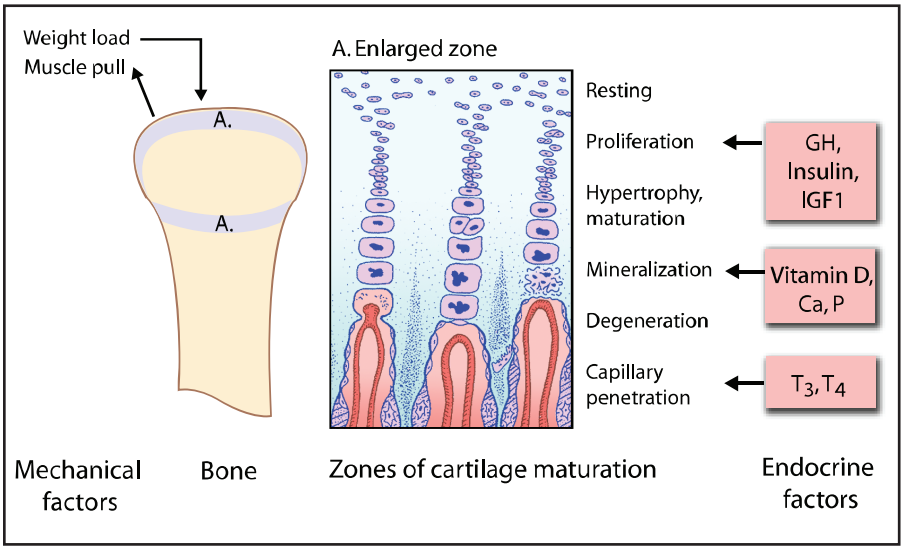

Figure 33-5. Biomechanical and endocrinologic influences on the growing skeleton are depicted. Biomechanically, excessive static (weight load) and dynamic (muscle pull) forces can damage immature skeletons. Note the various zones of cartilage maturation (resting zone, proliferation, hypertrophy and maturation, mineralization, degeneration and capillary penetration) where hormonal influences are thought to occur.

Free-choice feeding of dogs that results in excess energy intake is also accompanied by higher circulating concentrations of T_3 and T_4 compared with levels in food-restricted controls, reflecting a general stimulation of metabolic processes (Blum et al, 1992). Thyroid hormones are not only general stimuli for metabolic processes, including increasing the rate of bone formation and resorption, but are also important for capillary penetration of degenerating cartilage cells and the final stage of endochondral bone formation (Glade, 1984). In conjunction with the food-hormone relationships summarized here, additional endocrine or autocrine factors are involved in cartilage and bone metabolism (Glade, 1984); unfortunately, relevant data for growing dogs are unavailable. The result of these hormon-

al influences is enhanced mitotic activity of proliferative cartilage cells, which may enlarge the width of the inherently mechanically unstable zone of chondrocyte growth.

Histologic examinations have revealed articular cartilage is less well supported by solid bone plates in rapidly growing dogs, compared with smaller breeds or to littermates fed restricted amounts after weaning (Daemmrich, 1991). The epiphyseal spongiosa of giant-breed dogs is inherently less dense and therefore assumed to be weaker than the spongiosa in small breeds, a tendency that may be exaggerated by overnutrition. Free-choice feeding may lead to a mismatch between bone growth and body growth, resulting in a lower ratio of long bone diaphyseal shaft cross-sectional area to body weight and also a less dense epiphyseal spongiosa.

The biomechanical stress induced by rapid weight gain during growth as discussed above has been cited as an etiology for DOD. It is unknown whether small focal cartilaginous lesions occur first and are then exacerbated by biomechanical stress (Daemmrich, 1991; Carlson et al, 1991), or if biomechanical stress first induces cartilaginous lesions (Hazewinkel et al, 1985; Hedhammar et al, 1974). In either case, increased static forces (weight load) and dynamic forces (muscle pull) may damage immature skeletons, especially in large- and giant-breed dogs. These dysregulations of nutrient supply, bone formation and endocrine regulation may interfere with skeletal maturation, thus increasing the risk for DOD in young animals (**Figure 33-5**).

Theory 2: Excess Calcium and Hypercalcitoninism

A contrasting theory to the preceding theory about high energy intake and rapid growth rate stems from the observation that the rate of DOD is increased in dogs with high calcium intakes (Dobenencker et al, 1997; Slater et al, 1992; Schoenmakers et al, 1997; Voorhout and Hazewinkel, 1987). Young Great Dane puppies fed a food high in energy and minerals free choice (Hedhammar et al, 1974), or high in calcium alone (Hazewinkel et al, 1985), developed osteochondrosis lesions with overt clinical signs of disease (**Figure 33-6**). These lesions appeared at both weight-bearing sites and sites where weight bearing was of no influence, such as the growth plates of ribs.

Feeding high-calcium foods to growing small-breed dogs results in histologic lesions but no clinical manifestations of DOD (Nap et al, 1993a). Large-breed dogs raised on food with a high calcium content or high calcium and phosphorus content had disturbed endochondral ossification (Nunez et al, 1974; Goedegebuure and Hazewinkel, 1986), retained cartilaginous cores in the distal radius and ulna (Schoenmakers et al, 1997; Voorhout and Hazewinkel, 1987) and delayed skeletal

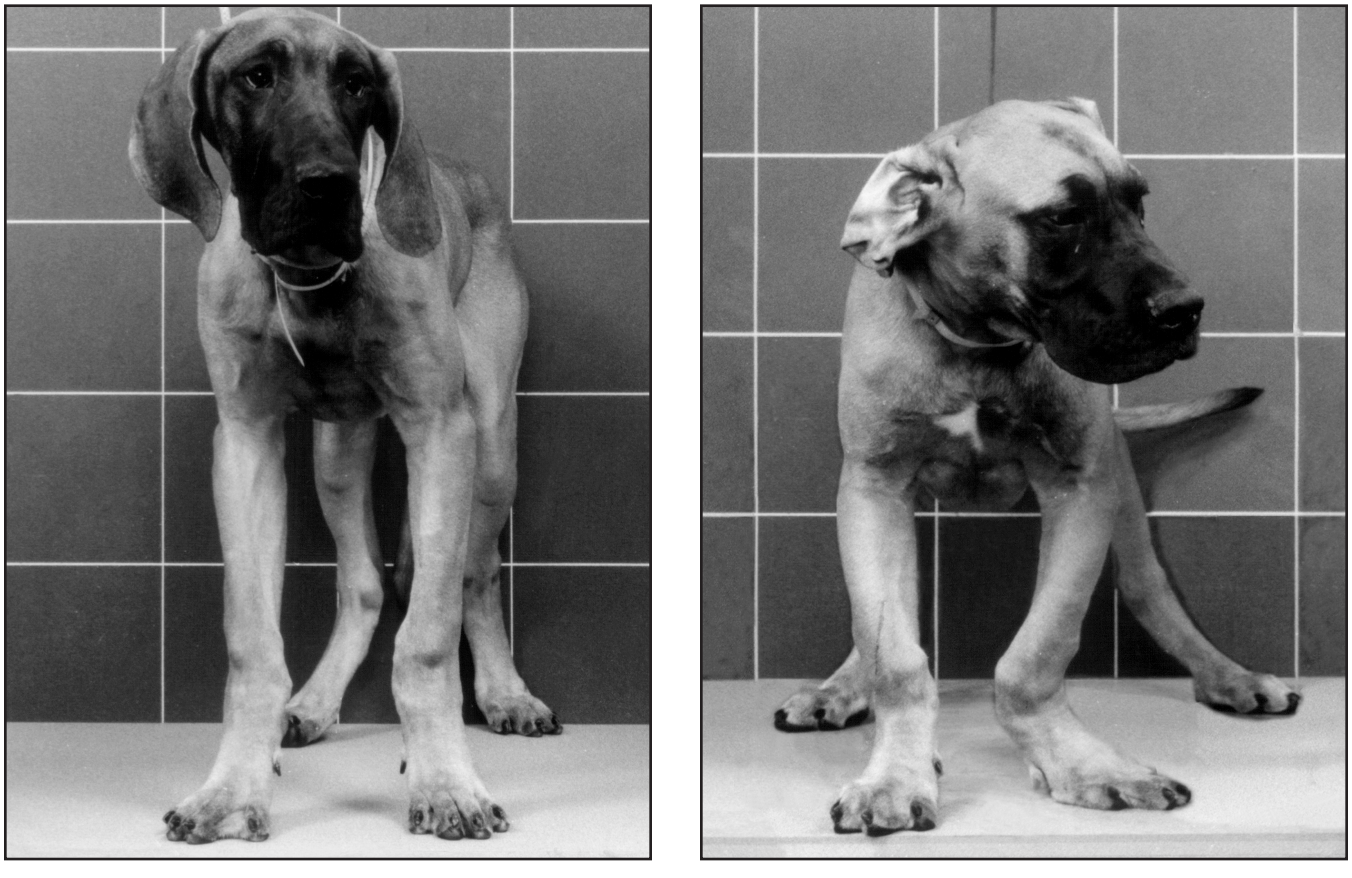

Figure 33-6. Littermate Great Dane puppies fed two different levels of dietary calcium. The puppy on the left was fed a growth food containing 1.1% dry matter calcium. The puppy on the right was fed a similar growth food containing 3.3% dry matter calcium. Note the poor growth and angular limb deformities in the puppy consuming excess calcium.

Table 33-5. Key nutritional factors for foods for growth (postweaning) of large- and giant-breed puppies.*

Factors	Dietary recommendations
Energy density	Energy density = 3.2 to 4.1 kcal/g; recommend the lower end of range if clients use free-choice feeding**
Fat	8.5 to 17%
Docosahexaneoic acid***	≥0.02%
Calcium	0.8 to 1.2% calcium
Phosphorus	Phosphorus amount is based on calcium amount to maintain recommended Ca-P ratio (below)
Ca-P ratio	1.1:1 to 2:1 (the lower end of range is preferred)
Supplements	None recommended if a commercial food is fed

Key: Ca = calcium, P = phosphorus.
*Dry matter basis.
**To convert kcal to kJ, multiply kcal by 4.184. Free-choice feeding is not recommended. Energy intake can be better controlled through food-limited feeding.
***For improved learning.

maturation and growth of bone length (Voorhout and Hazewinkel, 1987). Calcium intake, therefore, seems to be a significant determining factor in DOD. This may occur either directly by calcium competing with other minerals or indirectly by stimulating hormonal effects (PTH or calcitonin) or acid-base balance (**Box 33-3**). Accordingly, hypercalcitoninism may be a contributing factor to DOD in dogs (Hazewinkel et al, 1985; Hedhammar et al, 1974). Dogs ingesting excessive amounts of calcium for a prolonged period exhibited hyperplastic C-cells in their thyroid glands (Goedegebuure and Hazewinkel, 1986; Martin and Moseley, 1990). Great Dane puppies, with access to food with increased calcium content from three to six weeks (i.e., partial weaning), had significantly higher calcitonin release after challenge with calcium infusion, compared with the response of littermates that had access to food containing 1% calcium (Schoenmakers et al, 2000). These same dogs had clinical and radiographic evidence of DOD when compared with controls (Hazewinkel et al, 1985; Goedegebuure and Hazewinkel, 1986; Martin and Moseley, 1990).

Calcitonin is released into blood, where it has a half-life of a few minutes, and reduces concentrations of calcium and phosphorus (Hazewinkel, 1994; Martin and Moseley, 1990). Extrapolation of calcitonin action in other species indicates that increased osteoblastic activity and decreased osteoclastic activity are responsible for shifts in plasma concentrations of calcium and phosphorus, which in turn may affect production of 1,25-dihydroxyvitamin D_3 (**Table 33-2**) (Weisbrode and Capen, 1990). It has been proposed that the physiologic action of calcitonin on bone turnover (decreased skeletal remodeling) and endochondral ossification are inciting causes of DOD in dogs. Commercial foods with increased levels of calcium, calcium and phosphorus or vitamin D are associated with severe disturbances in endochondral ossification, with subsequent osteochondrosis and radius curvus syndrome (Schoenmakers et al, 2000; Tryfonidou et al, 2003).

Key Nutritional Factors

Nutrients must be provided in appropriate amounts and balances for optimal bone development. Excesses of calcium and energy, together with rapid growth, appear to predispose dogs to certain musculoskeletal disorders such as osteochondrosis and hip dysplasia (Hedhammar et al, 1974; Meyer and Zentek, 1991). However, severe excesses, deficits and imbalances of any nutrient may affect bone development. The recommended levels of key nutritional factors are summarized in **Table 33-5**.

Energy and Fat

Energy intake is a major determinant of growth rate. The detrimental influence of excess energy intake on skeletal development during growth has been demonstrated in dogs (Hedhammar et al, 1974; Kealy et al, 1992; Daemmrich et al, 1992; Zentek et al, 1995) and other animals (e.g., chickens, turkeys, pigs) (Carlson et al, 1988; Hester et al, 1990; Nakano and Aherne, 1994; Oviedo-Rondon et al, 2006). Associated lesions appear in physeal and/or articular epiphyseal cartilages as disturbances of endochondral ossification (Daemmrich, 1991). The best method for avoiding excess energy intake is to limit it quantitatively by means of food-limited (food-restricted) feeding.

The risk of DOD appears to be increased in large- and giant-breed puppies fed highly palatable, energy-dense foods, free choice. This is sometimes true even if foods are well balanced (Lavelle, 1989; Daemmrich, 1991; Kealey et al, 1992; Meyer and Zentek, 1992; Hoefling, 1989; Meyer, 1990; Richardson, 1992). However, when large-breed puppies were fed a very low energy density food (3.16 kcal [13.22 kJ]/g ME, 8.0% fat dry matter [DM] basis free choice vs. a food of higher energy density and increased fat (3.98 kcal [16.65 kJ]/g ME, 23.9% DM fat), the puppies eating the low energy density food had less body fat but not slower growth (no difference between groups in radius/ulnar lengths) (Richardson et al, 2000). It should be noted that none of the puppies in either group developed signs of DOD. The results of this report suggest that if free-choice feeding is used, it should only be done in combination with a low energy density food to decrease the risk for DOD and obesity. However, generally, free-choice feeding is risky and is not recommended for large- and giant-breed puppies until they have attained adulthood. Furthermore, commercial foods for large- and giant-breed puppies typically have energy densities of approximately 4 kcal (16.7 kJ) ME/g (DM) and should be food-limited fed.

Dietary fat is an important contributor to the energy density of a food. Dietary fat yields 8.5 kcal ME/g, whereas dietary digestible carbohydrate and protein each yield 3.5 kcal ME/g. Thus, as the fat content of a food is increased, the energy density is also increased (unless sufficient fiber is substituted for either carbohydrate or protein). Furthermore, when the energy density of a food is increased, concentrations of other essential nutrients need to be increased accordingly so that requirements for these nutrients are met at a lower food intake. The minimum recommended allowance for dietary fat in foods for growing puppies is 8.5% (DM) (NRC, 2006). Upper limits for

dietary fat in foods intended for large- and giant-breed puppies have not been established but a dietary fat level of 17% is acceptable as long as the puppies are fed properly (food-limited feeding). The associated energy density range would be between 3.2 and 4.1 kcal ME/g (DM). Besides being an important energy source, dietary fat is necessary for the absorption of fat-soluble vitamins. Dietary fat is also important from the standpoint of its constituent fatty acids and their effects on bone metabolism.

Some dietary fatty acids may play a role in preventing DOD. Metabolism of lipid in bone is thought to be under the same regulatory controls as in other tissues (Gilder and Boskey, 1990). Lipid content of mineralized tissues ranges from 1.7% of dry weight for cartilage to 0.2% for bone and dentin. Although specific studies on long bone growth have not been performed, interesting results have been obtained in studies of dentin formation. Essential fatty acid deficiency leads to abnormal calculus deposition, loosened teeth and poor gingival color in rats (Prout and Tring, 1971). Other lipids may play equally important roles in several metabolic aspects of tissue calcification:

1. Phospholipids form matrix vesicles that may be important in new calcification sites.
2. Calcium-acidic phospholipid phosphate complexes may signal nucleation and apatite formation under appropriate conditions.
3. Proteolipids may help initiate apatite formation and calcification.
4. Prostaglandins may influence calcium resorption similar to PTH, and affect collagen synthesis.
5. Inositol phospholipids may mediate calcium transport in and out of cell organelles via second messenger systems.
6. Glycolipids are important constituents of most cell membranes and are found in high concentrations in epiphyseal cartilage. Their specific function is not understood.
7. Phosphatidylserine may act as an ionophore to mediate calcium translocation.

Although no specific studies have been performed in growing dogs to assess the effect of omega-3 (n-3) or omega-6 (n-6) fatty acids on musculoskeletal growth, studies in other species may prove important. Rats fed foods high in lard (animal fat) compared with those fed foods high in linolenic acid (vegetable fat) had increased weight gain and depressed T_3 concentrations (Takeuchi et al, 1995). Chicks fed four different lipid sources had the highest bone formation rate when fed butter and corn oil as the dietary fat (Watkins et al, 1997). Dietary lipids modulate bone prostaglandin E and IGF-1 production, and bone formation rate in chicks. Changing dietary omega-6 and omega-3 fatty acid concentrations alter eicosanoid production in dogs and help manage osteoarthritis. Omega-3 fatty acids are used to help manage osteoarthritis (Hansen et al, 1990). In a double-blind efficacy study with 36 osteoarthritic dogs, increased omega-3 fatty acid intake increased plasma concentrations of LTB_5 (a less inflammatory leukotriene), although these findings did not coincide with improved ground reaction forces (locomotion) (Hazewinkel et al, 1998) (Chapter 34).

It is difficult to determine the appropriate daily energy requirement (DER) for growing dogs because few well-controlled studies have been conducted. Energy intake reaches a maximum, as related to body weight, in the second to fourth month of life (<45% adult body weight) (**Figure 33-7**). The data used to develop **Figure 33-7** were free-choice energy intakes from several breeds. These average intakes may be used as a crude guideline for determining energy requirements in growing puppies of different breeds.

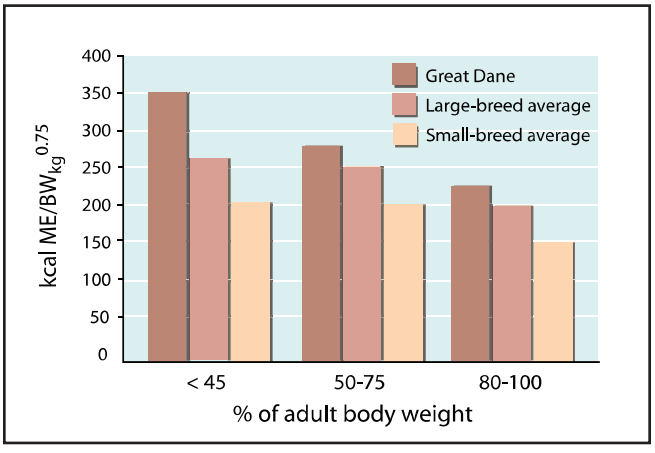

Figure 33-7. Average free-choice energy intake of Great Dane, large-breed and small-breed dogs in kcal metabolizable energy (ME)/$BW_{kg}^{0.75}$ as a percent of adult body weight. Note that energy intake is highest at <45% of adult body weight, which, on an age basis, is between the second and fourth month of life. Great Dane puppies appear to have higher energy requirements for growth than other large-breed dogs. (Adapted from Hedhammar A, Wu F, Krook L, et al. Overnutrition and skeletal disease. An experimental study in growing Great Dane dogs. Cornell Veterinarian 1974; 64 (Suppl. 5): 1-160. Meyer H, Zentek J. Über den Einfluß einer unterschiedlichen Energieversorgung wachsender Doggen auf Körpermasse und Skelettentwicklung. Journal of Veterinary Medicine A 1992; 39: 130-141. Meyer H. In: Ernährung des Hundes, 2nd ed. Stuttgart, Germany: Auflage. Eugen Ulmer, 1990. Rainbird A, Kienzle E. Untersuchungen zum Energiebedarf des Hundes in Abhängigkeit von Rassezugehörigkeit und Alter. Kleintierpraxis 1990; 35: 149-158. Zentek J, Meyer H, Daemmrich K. Untersuchungen einer Unterschiedlichen Energieversorgung auf die Wachstumsintensität und Skelettentwicklung bei Wachsenden Doggen. 3. Mitteilung: Klinisches Bild und chemische Skelettuntersuchungen. Journal of Veterinary Medicine A 1995; 42: 69-80.)

Breed differences in DER during growth may occur, but it is difficult to give specific recommendations because of the lack of quality data. Based on a small number of observations in dogs over eight months of age, the average ME intake for Great Dane puppies ranged from 311 kcal (1,300 kJ)/$(BW_{kg})^{0.75}$ at weaning to 263 kcal (1,100 kJ)/$(BW_{kg})^{0.75}$ at six months of age. These values are higher than for other large breeds and are consistent with reports of higher energy requirements for Great Dane puppies (Rainbird and Kienzle, 1990; Meyer, 1990; Zentek and Meyer, 1992; Zentek et al, 1995). Marked restriction of ME intake (191 kcal/$(BW_{kg})^{0.75}$ [800 kJ/$(BW_{kg})^{0.75}$]) for Great Dane puppies may lead to unacceptable body composition (Zentek and Meyer, 1992).

Unrelated to DOD, but considered important for all grow-

Box 33-3. Dietary Cation-Anion Balance.

Alteration of dietary cation-anion balance (DCAB) has been reported to influence skeletal development in several species. The DCAB of a food can be described, most simply, by the equation ([Na] + [K] – [Cl] mEq)/100 g dry matter (DM). As the DCAB increases, the net physiologic effect is alkalinization. Conversely, as it decreases an acidification effect is observed and calcium excretion in urine is increased. Acidification will be buffered by carbonate liberated from bone by increased osteoclasia, thus increasing osteoporosis in adult and bone remodeling in young animals. The mechanism for these effects on skeletal development is unclear. In addition, regulation of body acid-base balance, calcium homeostasis and osmolality of the synovial fluid compartment may be influenced.

The role of electrolyte balance in canine nutrition appears to be most relevant to preventing canine hip dysplasia. Investigators have associated the DCAB with the radiographic changes of subluxation in the coxofemoral joints in several canine breeds. A food with a DCAB ([Na] + [K] – [Cl]) <23 mEq/100 g DM fed to large-breed puppies was associated with less severe femoral head subluxation, on average, when the puppies reached six months of age. The slowed progression of subluxation was also observed in dogs fed a food with a reduced DCAB from 33 to 45 weeks of age. Hip joint laxity was determined using Norberg hip scores computed from radiographs. Significant correlation between radiographic findings (e.g., Norberg hip scores) and progression of canine hip dysplasia, either radiographic or clinical, was not proved. The authors proposed the balance of anions and cations in the food (specifically Na, K, Cl) influenced the electrolytes and osmolality in joint fluid. The joint fluid of dysplastic dogs has higher osmolality and is increased in volume when compared with that of disease-free hips from dogs of the same breed. The changes in osmolality and fluid volume could be a result rather than a cause of canine hip dysplasia. These studies suggest an association between DCAB and joint laxity without proving a mechanism of action. Most commercial growth foods encompass a very small range of DCAB and probably do not vary greatly in risk.

In commercial dog foods, the relation between cations and anions is 22 to 46 mEq/100 g DM. The balance of only the electrolytes Na, K, Cl, calculated as equivalents, will be between 15 to 42 mEq/100 g DM. Feeding foods with a dietary anion gap of 8 mEq/100 g DM lowered the severity of subluxation of the femoral head in growing dogs of different breeds. Increasing this relation to 41 mEq/100 g food was accompanied by a higher degree of subluxation as determined by the Norberg angle on radiography. Because the knowledge and experimental databases are very small in dogs, mineral salts should not be added in large amounts to nutritionally balanced foods. Problems may arise not only from the addition of anions, but also from increasing the amounts of cations. Further research is needed in this field but it seems prudent to avoid excessive acidifying foods or acidifying agents in growing puppies.

The Bibliography for **Box 33-3** can be found at www.markmorris.org.

ing puppies, are the effects of specific fatty acids on trainability and the development of special senses. Studies indicate that docosahexaneoic acid (DHA) is essential for normal neural, retinal and auditory development in puppies (Pawlosky et al, 1997). Similar results have been found in other species (Pawlosky et al, 1997; Birch et al, 2002; Diau et al, 2003; Hoffman et al, 2003). The inclusion of fish oil as a source of DHA in puppy foods improved trainability (Kelley et al, 2004). The conversion of short-chain polyunsaturated fatty acids to DHA is an inefficient process in puppies (Bauer et al, 2005). Thus, the essentiality of adding a source of DHA should be considered for this growth phase. The minimum recommended allowance for DHA plus eicosapentaenoic acid (EPA) is 0.05% (DM) with EPA not exceeding 60% of the total (NRC, 2006). Thus, DHA needs to be at least 40% of the total DHA plus EPA, or 0.02% (DM).

Calcium, Phosphorus and the Ca-P Ratio

The amount of true calcium absorption in dogs ranges from 25 to 90% depending on the amount of intake and the age of the animal (Nap and Hazewinkel, 1994; Hazewinkel et al, 1991). Calcium is absorbed via three mechanisms: 1) active absorption, 2) facilitated absorption and 3) passive diffusion. Passive diffusion is especially important in young animals. Active absorption is most important in the proximal GI tract. Passive diffusion and facilitated absorption, however, are important in the distal GI tract, primarily because of prolonged transit time and increased calcium concentration through that section. Vitamin D_3 metabolites, especially 1,25-dihydroxyvitamin D_3, are the most important hormonal regulators of GI calcium absorption (Birge and Avioli, 1990). Analysis of 90 dogs revealed that active calcium absorption decreases with increasing age, whereas passive absorption remains constant during the period of rapid growth. When calcium intake is high, active absorption becomes negligible and passive absorption accounts for up to 53% of total absorption of the amount eaten. There is no difference between breeds (Tryfonidou et al, 2002). Previous studies showed serious consequences for skeletal development in large-breed dogs (Hazewinkel et al, 1985) but not for small breeds (Nap et al, 1991). PTH, vitamin D_3 and dietary cation-anion balance modulate renal handling of calcium (**Box 33-3**), whereas calcitonin does not play a significant role in this aspect in dogs.

In the face of adequate levels of calcium in the food, the absolute level of calcium, rather than an imbalance in the calcium-phosphorus ratio, influences skeletal development (Hazewinkel et al, 1985; Hazewinkel et al, 1991) (**Boxes 33-4** and **33-5**). In one study, the prevalence of DOD was significantly increased in young, giant-breed dogs fed a food containing excess DM calcium (3.3%) with either normal DM phosphorus (0.9%) or high DM phosphorus (3%, to maintain a normal calcium-phosphorus ratio) (Hazewinkel et al, 1991). These puppies apparently were unable to protect themselves against the negative effects of long-term calcium excess (Hazewinkel et al, 1985; Tryfonidou et al, 2002). Furthermore, long-term calcium intake increases the frequency and severity of osteochondrosis (Nap and Hazewinkel, 1994). The minimum calcium

requirement for growth in puppies of both large and small breeds is 0.8% DM and the recommended allowance for calcium in foods for puppies is 1.2% DM. These recommendations are for foods with an energy density of 4 kcal ME/g (DM) (NRC, 2006). There should be no supplementation of such foods with additional calcium.

Excessive as well as inadequate phosphorus intake may affect calcium homeostasis and thus bone development. Chronic, inadequate phosphorus intake, to a lesser degree than calcium depletion, may stimulate 1,25-dihydroxyvitamin D_3 synthesis (**Table 33-2**), which stimulates calcium and phosphorus resorption from bone and absorption in the gut (Tanaka and DeLuca, 1977). Mobilization of calcium and phosphorus decreases PTH secretion, increases the renal threshold for phosphorus and eliminates excess calcium in the urine. The result is an increase in serum phosphorus concentration while maintaining serum calcium levels (Broadus, 1996).

Conversely, excessive phosphorus intake with inadequate calcium intake may result in nutritional secondary hyperparathyroidism. The excess phosphorus in food reduces the ionized calcium concentration in serum via mass action equilibrium, thus resulting in hypersecretion of PTH. The end result is a decreased renal threshold for phosphorus and excessive osteoclasia and pathologic fractures of growing bone.

The phosphorus level recommended must be considered in conjunction with calcium recommendations. The calcium-phosphorus ratio should be maintained at 1.1:1 to 2:1; however, the lower end of the range is preferred (NRC, 2006). The absolute amount of calcium in the food is more important than the calcium-phosphorus ratio in young growing dogs (Schoenmakers et al, 1997; Hazewinkel et al, 1991). Great Dane puppies raised on food with a calcium to phosphorus ratio of 1.1:1 but with an excessive absolute amount of calcium (3.3% DM calcium:3.0% DM phosphorus) developed more severe signs of DOD than did control dogs (1.1% DM calcium:1.0% DM phosphorus) or dogs raised on low-calcium food (0.55% DM calcium:0.9% DM phosphorus). The last group (e.g., those fed the lowest calcium level) developed pathologic fractures due to hyperparathyroidism as described above. When calcium intake is set at 0.8 to 1.2% DM of the food, as recommended previously for large breeds at risk for DOD, the calcium-phosphorus ratio should be kept within physiologic limits (1.1:1 to 2:1).

Other Nutritional Factors

Other nutritional factors may be conditionally important in some animals. For example, animals fed improperly formulated homemade foods may receive insufficient calcium and excessive phosphorus. Animals fed such foods may develop nutritional secondary hyperparathyroidism as described above. Other nutritional factors are described below.

Digestibility

Digestibility is a nutritional factor that becomes important in certain physiologic states such as growth. Apparent digestibility is the difference between the amount of food ingested and that excreted in feces. During the growth period, the ability to ingest and absorb adequate amounts of various nutrients depends on food intake capacity and the quality of ingredients. It is especially important to consider quality of ingredients when trying to limit energy intake for at-risk dogs. The goal of energy restriction is not to provide low-quality foods that are poorly digestible, but to provide high-quality foods in a low energy density package that will promote appropriate growth. It is important to assess digestibility and recommend foods with at least average or above average digestibility for growth. Typically, foods that are highly digestible are also higher in energy density.

Box 33-4. Dangers of Feeding Puppies Adult Maintenance Foods to Decrease Energy Intake.

Often puppies are switched from growth to adult maintenance-type foods under the pretense it will help avoid calcium excess and skeletal disease. However, because some maintenance foods have much lower energy density than most growth foods, the puppy must consume more dry matter (DM) volume to meet its energy requirement. If the DM calcium levels are similar between the two foods, the puppy may actually consume more calcium when fed the maintenance food.

This point is exemplified in the case of switching a 15-week-old, 15-kg, male rottweiler puppy from a growth food containing, on an as fed basis, 4.0 kcal (16.74 kJ)/g metabolizable energy and 1.35% calcium (1.5% DM) to a maintenance food containing the same amount of calcium but at a lower energy density (3.2 kcal [13.4 kJ]/g). The puppy would require approximately 1,600 kcal/day (6.69 MJ). To meet this energy need, the puppy would consume approximately 400 g of the growth food (containing 5.4 g of calcium) vs. 500 g of the maintenance food (containing approximately 6.7 g of calcium).

Box 33-5. Dangers of Feeding Calcium Supplements to Dogs.

Feeding dogs treats that contain calcium or providing calcium supplements further increases daily calcium intake. Two level teaspoons of a typical calcium supplement (calcium carbonate) added to the growth food of a 15-week-old, 15-kg rottweiler puppy more than doubles its daily calcium intake. This calcium intake is well beyond levels shown to increase the risk for developmental orthopedic disease. A review article best summed up the need for calcium supplements: "Because virtually all dog foods contain more calcium than is needed to meet the requirement, the use of a calcium supplement certainly is unnecessary. Now that the deleterious effects of excess dietary calcium have been delineated, we can say that the feeding of calcium supplements not is unnecessary, but, in fact, contraindicated!"

The Bibliography for **Box 33-5** can be found at www.markmorris.org.

Copper

Copper plays an important role in the metabolism of collagen and elastin. The copper-dependent lysyl oxidase is specific for connective tissue and functions biologically to catalyze the oxidative deamination of the ε-amino groups of lysine and hydroxylysine to form allysyl or hydroxyallysyl residues (Harris et al, 1980; Siegel, 1979). This step forms intermolecular cross links between collagen fibrils, and is therefore essential for stabilization of connective tissues (Eyre et al, 1984).

In several animal species and in people, copper deficiency induces severe skeletal disease (Danks, 1980). Dietary copper levels less than 1 mg/kg DM were related to severe growth deformities, fractures, wide "knotty" epiphyses and especially severe hyperextension of the limb axis in growing dogs (Baxter and Van Wyk, 1953). In young beagles, clinical signs of copper deficiency were less severe than those previously reported; however, hyperextension of the forelegs was a characteristic feature (Zentek et al, 1991). Feeding a low-copper food (1.2 mg/kg DM) vs. a normal copper food (14.1 mg/kg DM) resulted in depletion of plasma (1.4 vs. 9.7 µmol/l) and liver copper stores (19 vs. 246 mg copper/kg DM). Secondary copper deficiency resulted in osteoporotic lesions in growing Great Dane puppies, which could be attributed to impaired osteoblastic function (Read et al, 1989). These dogs were fed an experimental food containing high concentrations of molybdate, which strongly impaired copper absorption and induced secondary copper deficiency.

The overall prevalence of primary copper deficiency (i.e., a dietary deficiency) should not be overestimated. Most common ingredients are rich in copper; however, some homemade, unsupplemented foods (made of rice, dairy products, fat, starch) may contain low or suboptimal copper concentrations. Under certain circumstances, these foods may contribute to the development of skeletal disease, even if copper levels are higher than in deficient experimental foods. A suboptimal copper supply could evoke negative effects especially if combined with high growth intensity or other dietary imbalances (e.g., calcium, zinc or carbohydrates). The possibility that large dogs are more susceptible to a low dietary copper intake cannot be excluded. Impaired copper absorption may also occur with high dietary calcium or zinc levels; the latter induces copper binding metallothionein in the gut mucosa (Brewer et al, 1992). High amounts of poorly digestible carbohydrates or foods that are rich in certain types of dietary fiber may also reduce copper absorption (Zentek, 1995).

The recommendation for copper in canine growth foods is 11 mg/kg (DM) (NRC, 2006). Most commercial canine growth foods deliver copper in the range from 11 mg/kg to 20 mg/kg (DM) and, therefore, meet this recommendation.

Zinc

Zinc is an essential trace element that is widely distributed in the body. It serves as an important coenzyme in numerous biochemical processes. The zinc concentration in newborn puppies is about 22 mg/kg body weight and concentrations increase to 120 mg/kg in tissues formed during the growth phase (GfE, 1989). Inadequate zinc supply, especially in growing animals, causes severe clinical signs within days, including growth depression, skin defects, impaired immune function and growth disorders of the skeleton. These disorders may be linked to the role of zinc as a cofactor in enzymes that are important for connective tissue metabolism. A low activity of alkaline phosphatase (<300 IU/l) is a good indicator of a low zinc status (i.e., deficient zinc intake) in growing animals and young dogs (Kirchgessner, 1987).[b] There are no reports that excessive zinc intake is detrimental to skeletal development in dogs; however, excess zinc is presumed to be toxic at higher levels, as observed in other species.

The essentiality of zinc for skeletal development is unequivocal; reports are available for many species describing severe growth disorders induced by zinc deficiency (Hambidge et al, 1986). Zinc deficiency in dogs is of practical importance mainly with regard to skin diseases (NRC, 2006) (Chapter 32). Skeletal abnormalities have been described in Alaskan malamutes with an inborn error in zinc metabolism (Smart and Fletch, 1971; Brown et al, 1978) and skeletal malformation in bull terriers with lethal acrodermatitis enteropathica, a genetically determined defect of zinc metabolism (Jezyk et al, 1986). Experimental zinc deficiency in beagles leads to a significant decrease of zinc concentrations in the skeleton especially in metaphyseal bone, which represents newly formed tissue. It is unknown to what extent marginal zinc intake, due to either subnormal dietary zinc concentrations or high concentrations of interacting substances (e.g., phytic acid, calcium, copper, low digestible carbohydrates) (Zentek, 1995), contributes to DOD. Foods for growing dogs should contain enough zinc to compensate for negative interactions with other dietary ingredients, especially if the originally balanced food is "improved" by dog owners who add large amounts of calcium carbonate or other calcium salts.

Canine growth foods should contain 100 mg/kg DM zinc (NRC, 2006). Most commercial canine growth foods contain higher levels of zinc to ensure this recommendation is met.

Iodine

Iodine is essential for function of the thyroid glands (Belshaw et al, 1975). The amino acid tyrosine is iodinated and, in subsequent metabolic steps, T_4 and the biologically more active form T_3 are formed. Both hormones, but particularly T_3, influence normal maturation of growing cartilage, penetration of capillaries and mineralization of newly formed bone. Thyroid hormones stimulate formation and resorption of bone, which results in remodeling of the skeleton (High et al, 1981). Boxers with congenital hypothyroidism were found to have shortened limb bones and severe disturbances of the ossification and mineralization process, problems that were alleviated by L-thyroxine supplementation (Saunders and Jezyk, 1991).

Low dietary iodine induces dysfunction of the thyroid glands. Goiter (enlarged thyroid glands) develops with extreme deficiency. In some regions of the world, goiter still occurs in dogs because they are fed unbalanced, homemade rations (Kienzle and Hall, 1994). Stunted limb development, hyperpla-

sia of the thyroid glands and myxedema with no loss of hair typically occur in young puppies born to bitches that were iodine deficient during pregnancy. Most commercial foods meet the recommended iodine level of 0.88 mg/kg (DM) (NRC, 2006).

Manganese

Manganese acts as a coenzyme in glycosyl transferases in the metabolism of the ground substance in cartilage. In different species, experimental dietary deficiency leads to disproportionate shortened and thickened long bones, defective skull development and otoliths in the inner ear (Hurley and Keen, 1986). Currently, no reports describing manganese deficiency in dogs exist. Less than 5% of dietary manganese is absorbed in the canine intestinal tract and the process seems to be strictly regulated (Zentek, 1995). The dietary requirement of manganese for dogs appears to be lower than that of most other species (1.4 mg/1,000 kcal [0.33 mg/MJ] ME) (Meyer, 1990). Most commercial foods meet or exceed the recommended allowance of 5.6 mg/kg (DM) (NRC, 2006).

Protein

Protein is required for a variety of structural and functional molecules to achieve proper growth. The minimum adequate level of dietary protein depends on digestibility, amino acid composition, proper ratios among the essential amino acids, energy density of the food and amino acid availability from protein sources. The dietary protein requirements of healthy growing dogs decrease as they approach adulthood (Richardson and Toll, 1997).

Protein excess has not been shown to negatively affect health or skeletal development during growth of Great Dane puppies when compared with isoenergetically fed controls (Nap et al, 1993b). Protein deficiency may affect the general health of developing puppies, decrease plasma growth hormone levels and reduce skeletal growth (NRC, 2006; Gessert and Phillips, 1956). In Great Dane puppies, a DM protein level of 14.6% with 13% of the dietary energy derived from protein resulted in significant decreases in body weight and plasma albumin and urea concentrations with no increased frequency of osteochondrosis (Nap et al, 1993b, 1991). A growth food with average energy density should contain 22 to 32% DM protein of high biologic value (Dzanis, 1995). The recommended allowance for dietary protein (of high biologic value) for growth of puppies after weaning is at least 17.5% (DM) (NRC, 2006).

Vitamins

VITAMIN D

Metabolites of vitamin D_3 act in concert with other hormones to regulate calcium metabolism and therefore skeletal development in dogs. Vitamin D_3 metabolites aid in calcium and phosphorus absorption from the gut and influence bone cell activity (Hazewinkel, 1993). The vitamin D requirement of dogs may be met from food sources from plants (vitamin D_2) or animals (vitamin D_3) (How et al, 1994).

Clinical cases of vitamin D_3 deficiency (rickets) are extremely rare in dogs fed commercial foods (Kallfelz and Dzanis, 1989). Measuring circulating levels of vitamin D_3 metabolites can help make a diagnosis of vitamin D_3 deficiency. Increased growth plate width and thin bone cortices are not associated with low-calcium, high-phosphorus foods, but are strong indicators of rickets (Hazewinkel, 1993).

Vitamin D excess (i.e., 135x the recommended amount of 550 IU/kg food [DM]), in growing Great Dane puppies, caused no increase in calcium or phosphorus plasma concentrations and no increase in intestinal calcium absorption; however, severe disturbances occurred in endochondral ossification, resulting in osteochondrosis and radius curvus syndrome (Tryfonidou et al, 2003). Vitamin D intoxication can cause hypercalcemia, hyperphosphatemia, anorexia, polydipsia, polyuria, vomiting, muscle weakness, generalized soft tissue mineralization and lameness. In growing dogs, excessive supplementation with vitamin D can markedly disturb normal skeletal development because of increased calcium and phosphorus absorption (Richardson and Toll, 1997; Hazewinkel, 1993). The minimum recommended allowance is 13.8 μg/kg food (DM) or 550 IU/kg of food (DM) (NRC, 2006). The safe upper limit is 3,200 IU/kg (DM) (NRC, 2006). In a study published in 1989, commercial pet foods were shown to contain from two to 10 times the recommended amount (Kallfelz and Dzanis). Therefore, it is best to recommend against providing supplements that contain vitamin D to growing dogs fed commercial foods.

VITAMIN A

Vitamin A is an essential factor in bone metabolism, especially osteoclastic activity (Hayes, 1971). Deficiency or excess may lead to severe metabolic bone disease in growing dogs (NRC, 2006). Concentrations of vitamin A in canine serum range from 1,800 to 18,000 IU/l (Keane et al, 1947); however, with higher intakes, most of the retinol is bound to esters, making dogs relatively insensitive to higher intakes.

Hypervitaminosis A may result in anorexia, decreased weight gain, hyperesthesia, narrowing of long bone epiphyseal cartilage, ankylosis, new bone formation without osteolysis and thin bone cortices (Hazewinkel, 1994). High doses of vitamin A given to pregnant bitches may result in cleft palates in puppies (Wiersig and Swenson, 1967). Adult beagles fed at maintenance levels for 26 weeks demonstrated a very high tolerance to 200,000 IU of vitamin A/kg body weight with no detrimental effects on selected parameters (Goldy et al, 1996).

Hypovitaminosis A results in a variety of clinical signs including anorexia, weight loss, ataxia, xerophthalmia, metaplasia of bronchiolar epithelium, conjunctivitis and increased susceptibility to infection. In addition, faulty bone remodeling may constrict nerves passing through bone foramina resulting in neural degeneration.

The recommended concentration of vitamin A (all *trans*-retinol) in dog foods is 1,515 μg/kg DM or 5,050 IU/kg DM (NRC, 2006). Most commercial dog foods are supplemented well above the minimum requirement for vitamin A. The safe upper limit of vitamin A is 15,000 μg/kg DM (NRC, 2006). In

Table 33-6. Recommended levels of key nutrients for dogs at risk for developmental orthopedic disease compared to levels in selected dry commercial foods marketed for large- and giant-breed puppies.*

	Energy density (kcal/cup)**	Energy density (kcal/g) DM	Fat (%)	DHA (%)	Ca (%)	Ca-P ratio
Recommended levels	-	**3.2-4.1**	**8.5-17**	**≥0.02**	**0.8-1.2**	**1.1:1–2:1*****
Hill's Science Diet Puppy Lamb Meal & Rice Recipe Large Breed	357	3.9	18.0	0.220	1.17	1.1:1
Hill's Science Diet Puppy Large Breed	357	3.9	16.8	0.223	1.20	1.4:1
Iams Eukanuba Large Breed Puppy Formula	362	4.4	17.2	na	0.88	1.2:1
Iams Smart Puppy Large Breed	368	4.5	16.1	na	1.0	1.3:1
Nutro Natural Choice Large Breed Puppy	346	3.9	15.4	0.011	1.32	1.1:1
Purina ONE Large Breed Puppy Formula	404	4.1	16.7	na	1.44	1.1:1
Purina Pro Plan Large Breed Puppy Formula	377	4.0	16.4	na	1.37	1.1:1
Royal Canin Maxi Large Breed Puppy 32	365	4.3	15.6	na	1.12	1.2:1

Key: DM = dry matter, DHA = docosahexaneoic acid, na = not available from manufacturer.
*Nutrients are expressed on dry matter basis except for energy density, which is expressed on an as fed basis.
**Energy density values are as fed and are useful for determining the amount to feed; cup = 8-oz. measuring cup. To convert to kJ, multiply kcal by 4.184.
***The lower end of the range is preferred.

Box 33-6. Treatment of Dogs Affected with Developmental Orthopedic Disease.

1. If possible, determine if a nutritional imbalance is causing the skeletal disease observed. The feeding history, clinical signs, radiographic changes and laboratory values may be helpful.
2. To correct either deficiencies or excesses, recommend the pet owner feed a nutritionally adequate growth food designed for large- and giant-breed puppies (**Table 33-6**).
3. If a well-balanced growth food is being fed and skeletal diseases occur, reduce food intake up to 25%.
4. Do not give vitamin or mineral supplements to dogs eating commercial foods, particularly calcium, phosphorus, vitamin D and vitamin A. If a nutritionally adequate commercial growth food is being fed, supplementation is contraindicated.
5. Provide appropriate treatment for specific problems, such as pathologic fractures. Remember, dietary recommendations are inferred from limited group/breed observations and applied to individual animals. All feeding programs need to be tailored to individual animal and client situations. Initial dietary recommendations are a generalized starting point for veterinary/client interactions. Monitoring the body condition score, which necessitates veterinarian-client interaction at regular intervals, assesses dietary adequacy.

the rare case of suspected vitamin A toxicosis, foods low in vitamin A should be fed until signs diminish (Donoghue et al, 1987).

VITAMIN C

L-ascorbic acid (vitamin C) is integral to hydroxylation of proline and lysine during biosynthesis of collagen, of which type I collagen is the most widely distributed in connective tissue (primarily in bone and ligaments). Foods devoid of vitamin C and fed to puppies for 147 to 154 days neither affected growth nor caused skeletal lesions (Dzanis, 1995). Dogs supplemented with vitamin C had transiently elevated plasma vitamin C concentrations; however, long-term supplementation did not increase concentrations much above normal. Excess vitamin C supplementation is generally considered to have little or no effect on the skeleton but may enhance calcium absorption in some cases, thus increasing the risk for DOD (Teare et al, 1979). The relationship between vitamin C and DOD in dogs is unproved; therefore, supplementation is not recommended (Richardson, 1995).

FEEDING PLAN

Assess and Select the Food

To help prevent DOD in large- and giant-breed puppies (>25 kg adult weight), it is best to feed a commercial food specific for their unique nutrient requirements. The recommended intake of most nutrients in fast-growing, large- and giant-breed puppies is similar to that of other breeds (Chapter 17). However, recommendations are more stringent for dietary fat, energy, calcium and the calcium-phosphorus ratio (**Table 33-5**) in dogs at risk for DOD. Several commercial foods are available that have been formulated for fast-growing, large- and giant-breed puppies and their key nutritional factor profiles are compared to the key nutritional factor targets developed above (**Table 33-6**). A food should be selected that is most similar to the key nutritional factor benchmarks.

Foods for growth should have passed an Association of American Feed Control Officials or similar feeding trial specific for that lifestage, or at the very least have a formula that is approved by such an agency (AAFCO, 2007). For feeding trials to be meaningful, they should be conducted in large-breed puppies. However, even feeding trials do not ensure adequacy or safety for every breed. Generally, dry foods are more economical than moist foods. Considering most DOD occurs in

large- and giant-breed puppies, the usual type of food selected is a dry formulation. However, moist foods may be fed as long as special attention is paid to key nutritional factors and successful feeding tests.

Owners should not add vitamin or mineral supplements to balanced foods, particularly calcium, phosphorus, vitamin D and vitamin A. If a nutritionally adequate growth food is being fed, supplementation is contraindicated.

Box 33-6 describes treatment plans often prescribed for dogs with DOD.

Assess and Select the Feeding Method

Both the key nutritional factor profile of a food and how it is fed (feeding method) are risk factors for DOD. Assessment of the feeding method requires owner knowledge of current feeding practices, which includes the amount being fed. If owners do not know how much food their puppies are consuming, they should measure how much is being ingested for several days under the current feeding regimen. This information will help when making recommendations for future feeding plans.

Ideally, the food selected for feeding large- and giant-breed puppies should also be fed during the weaning process (Chapters 16 and 17), either throughout weaning or for the last week or so. The advantage of the former is that a food change is unnecessary during or at weaning. The advantage of the latter is that a food change is unnecessary at the stressful time of weaning. Both ensure that appropriate food is being fed during a period of rapid growth.

There are three basic feeding methods: 1) free choice (ad libitum), 2) time limited or 3) food limited. In any feeding regimen an initial estimate of the amount to be fed is required.

Food-Restricted Meal Feeding

The method of choice for feeding puppies at risk for DOD is limiting food intake to maintain optimal growth rate and body condition. Food-limited feeding requires feeding a measured amount of food based on the puppy's DER divided into two or three meals per day.

There are several ways to determine a puppy's initial DER. However, all methods are estimates and even if a puppy's DER is determined accurately, it must be revised continually as the puppy grows.

One way to determine a DER starting point is based on a puppy's age. From weaning to four months of age, consider a puppy's DER to be three times its resting energy requirement (RER), followed by two times the RER until about one year of age (**Table 33-7**). RER can be calculated using either of the following equations: RER (kcal/day) = $70(BW_{kg})^{0.75}$ or RER (kcal/day) = $30(BW_{kg}) + 70$ or it can be obtained from Table 5-2.

Another method for determining a starting point for a puppy's DER is based on its relative body weight. At 15% of adult body weight, energy intake should be 2.5 x the maintenance energy requirement (MER) (MER = 130 x RER), at 30% it should be 2.1 x MER, at 60% 1.6 x MER and at 80% 1.3 x MER (**Table 33-8**) (NRC, 2006).

Table 33-7. Worksheet for determining the daily amount to feed for large- and giant-breed puppies.

I. **Weigh** (determine body weight in kg)
II. Estimate the initial amount to **feed** (starting point)
  A. Determine RER (use tabulated values or energy formulas) Table 5-2 or RER formulas:
    Linear formula: RER (kcal/day*) = $30(BW_{kg}) + 70$
    Exponential formula: RER (kcal/day) = $70(BW_{kg})^{0.75}$
  B. Determine DER
    DER = RER x 3.0 (2 to 4 months of age)
    = RER x 2.0 (4 to 12 months of age)
    = RER x 1.2 to 1.4 (inactive/obese prone adult)
    = RER x 1.6 (neutered adult)
    = RER x 1.8 (intact adult)
  C. Convert DER to 8-oz. measuring cups, cans or grams (divide DER by energy density of food as fed)
III. Reassess (**evaluate**) every two weeks
  A. Weigh
  B. BCS
  C. Clinical judgment
IV. **Adjust** amount to feed (as needed)
  A. If BCS >3/5, decrease amount fed by 10%
  B. If BCS <2/5, increase amount fed by 10%

Key: DER = daily energy requirement, RER = resting energy requirement, BCS = body condition score, BW = body weight.
*To convert kcal to kJ, multiply kcal by 4.184.

Table 33-8. A method for estimating daily energy requirement (DER) for growth of puppies after weaning.*

Puppy weight as a percent of anticipated adult body weight	MER factor**
15	2.5
30	2.1
43	1.9
60	1.6
71	1.4
80	1.3
100	1.0

Example calculation:
A puppy that has a current weight of 5.25 kg with an expected adult body weight of 35 kg would be at 15% of its anticipated adult weight. The corresponding MER factor from above would be 2.5.
The puppy's MER = $130(5.25_{kg})^{0.75}$ = 130(3.47) = 450 kcal/day. Multiply MER by the MER factor to obtain the puppy's estimated DER for this stage of growth = 450 kcal/day x 2.5 = 1,127 kcal/day. Extrapolate MER factors for % anticipated adult weights not shown in table.
To convert kcal to kJ, multiply kcal by 4.184.
*Adapted from NRC, 2006.
**MER = maintenance energy requirement; MER (kcal/day) = $130(BW_{kg})^{0.75}$

After the DER is determined, it is converted to a daily amount of food to feed by dividing the DER by the energy density of the food on an as-fed basis (i.e., kcal/cup, kcal/can or kcal/g). The as-fed energy density of the food under consideration can be obtained from **Table 33-6**, the product label or the manufacturer (toll-free customer service telephone number on label, other published information or website content). **Table 33-9** provides an example calculation.

Great Dane puppies are the exception to the previous recom-

Table 33-9. Example calculation for converting estimated daily energy requirement (DER) to a daily amount of food to feed.

To determine the daily amount to feed, divide the estimated DER by the as-fed energy density of the food. For example, if a puppy's estimated DER is 1,127 kcal/day and the food selected provides 375 kcal/8-oz. measuring cup, feed three cups/day (1,127 kcal ÷ 375 kcal/cup = three cups). This amount would be divided into two or three meals per day. Regardless of how the DER is estimated, or if the manufacturer's recommendations are used as a starting point, adjust the amount to feed (as needed) every two weeks based on the puppy's body condition score (BCS). If the BCS is >3/5, decrease the amount fed by 10%; if the BCS is <2/5, increase the amount fed by 10%.
To convert kcal to kJ, multiply kcal by 4.184.

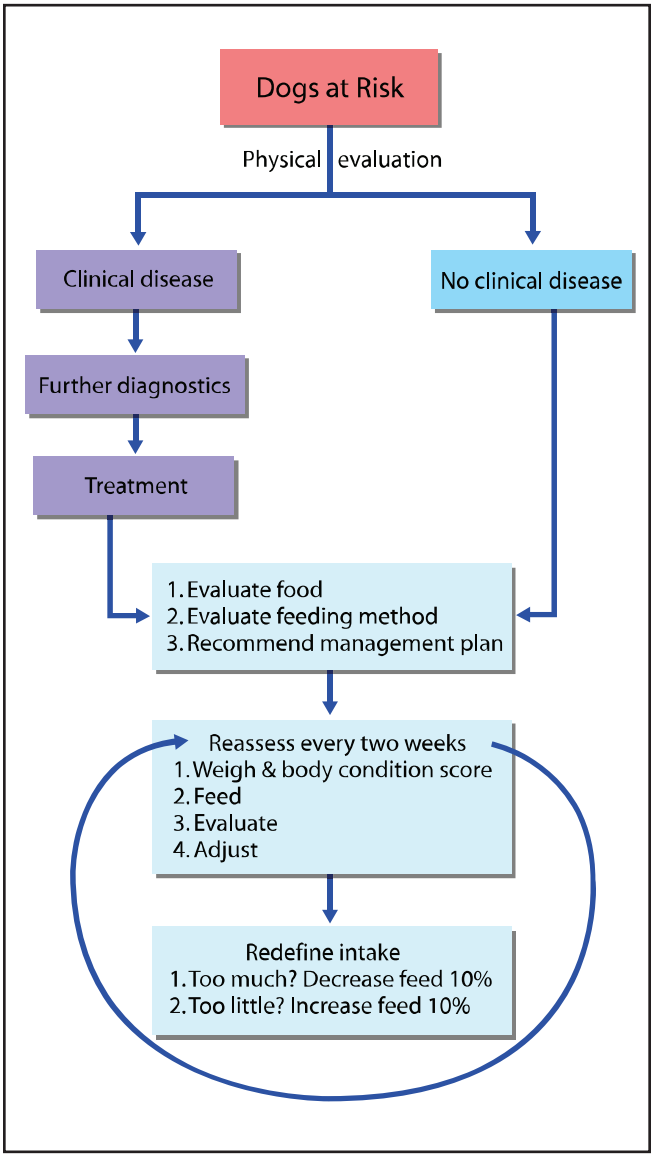

Figure 33-8. Flowchart for assessing dogs at risk for developmental orthopedic disease.

mendations because they may require 20% more energy than other large/giant breeds. Marked restriction (191 kcal [800 kJ]/$BW_{kg}^{0.75}$) of ME intake for Great Dane puppies may lead to unacceptable body condition (Zentek and Meyer, 1992).

Regardless of which method is used to determine an initial amount to feed, it is critical that subsequent regular (every two weeks) body condition assessment is done to ensure the amount being fed is appropriate (**Table 33-7** and **Figure 33-8**). Large- and giant-breed puppies should maintain a body condition score of 2/5 to 3/5.

Most large- and giant-breed puppies will increase body weight and muscle mass after 12 months of age, but the growth rate is reduced and most, if not all, growth plates are closed. At 12 months, these puppies can be fed as adults (1.6 x RER for neutered dogs and 1.8 x RER for intact dogs). Slower growth during the first year does not deleteriously affect final adult body size (**Figure 33-9**).

Free-Choice Feeding

Free-choice feeding is relatively effortless and may reduce abnormal behavior such as barking at feeding time. Additionally, frequent trips to the food bowl may help reduce boredom and timid or unthrifty puppies experience less competition when eating. Coprophagy may be decreased and frequent small meals may result in a more constant blood level of nutrients and hormones.

Disadvantages of free-choice feeding include food wastage, only dry or semi-moist forms of pet food can be fed and competition or boredom may stimulate overeating. The most serious disadvantage is increased risk of DOD because of potential overconsumption by large- and giant-breed puppies (Hedhammar et al, 1974; Kealy et al, 1992; Lavelle, 1989; Meyer and Zentek, 1991). If free-choice feeding is used, it is especially important to recommend a food with an energy density less than 3.8 kcal/g (15.9 kJ) (<12% DM fat) to decrease the risk of excess energy intake.

However, free-choice feeding is not recommended for large- and giant-breed puppies until they have reached skeletal maturity (about 12 months of age or at least 80 to 90% of adult weight).

Time-Restricted Meal Feeding

Time-limited feeding is a method in which dogs are allowed access to food for a defined period, usually 10 to 15 minutes, once or twice daily (three times per day for the first month after weaning, then twice per day). In some cases, the feeding periods may need to be even shorter.

Some investigators have proposed that puppies fed in this way consume less food because they have a smaller stomach volume than that of adults. The energy requirement of young animals may be two to three times that of adult dogs of the same weight, but the stomach volume may be smaller on a per body weight basis.

Investigators who advocate this feeding method suggest that puppies have slightly reduced growth rates, but achieve similar adult size and lean body mass when compared with puppies fed

free choice (Alexander and Wood, 1987). Other studies have shown that feeding 15 minutes twice daily does not reduce food intake between free-choice and time-restricted groups (Toll et al, 1993). Again, it is important in this type of feeding program to recommend foods with a lower energy density (<12% DM fat) to decrease the risk of overconsumption.

Time-limited feeding may also help in disciplining and housetraining young puppies. The owner interacts with the puppy during this time and is able to observe its general condition and behavior, which may lead to earlier detection of health problems. A routine of feeding a puppy and then taking it outdoors can assist housetraining by taking advantage of the gastrocolic reflex. Advocates of this feeding method suggest that when some puppies fed in this manner reach adulthood they may voluntarily limit their feeding to once or twice a day and thus avoid overeating.

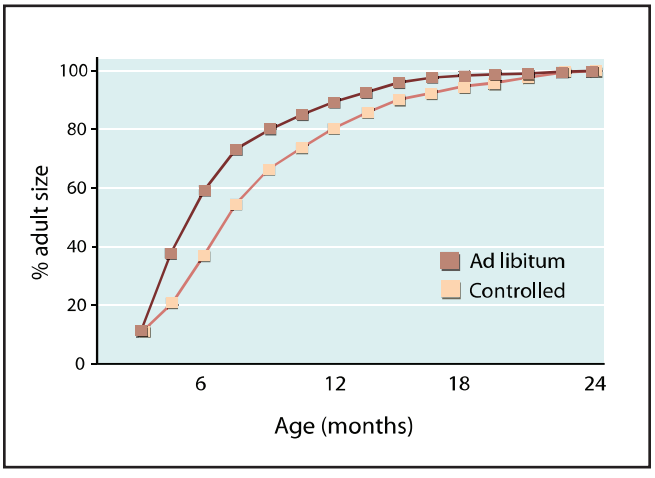

Figure 33-9. Comparison of growth curves of large-breed dogs fed free choice vs. those in a food-restricted feeding regimen.

REASSESSMENT

Regular clinical evaluation of growing puppies and adjustments in the food offered are crucial. Rapidly growing, large- and giant-breed dogs have a very steep growth curve and their intake requirements can change dramatically over short periods. These puppies should be weighed, their body condition evaluated and their daily feeding amount adjusted at least once every two weeks (**Figure 33-8** and **Table 33-7**). Large- and giant-breed puppies should be fed to maintain a BCS between 2/5 to 3/5.The veterinary health care team can perform this evaluation in the hospital and owners can be taught to perform this evaluation at home.

Skeletal disease can be influenced during growth by feeding technique and nutrient profile. However, nutritional management alone will not completely prevent DOD because there is a hereditary component (i.e., canine hip dysplasia and osteochondrosis can develop in genetically affected animals fed balanced foods). Additionally, the occurrence and clinical signs of DOD can be aggravated when forced exercise or environment are not adapted to the vulnerability of the young skeleton. Dietary deficiencies are of minimal concern in this age of commercial foods specifically prepared for young, growing dogs; the major potential for harm results from excess consumption of energy and calcium.

A balanced food fed in appropriate quantities will help optimize skeletal development and decrease the risk of DOD. After DOD has manifested, nutritional management becomes a minor component of treatment unless obesity is a contributing factor.

ENDNOTES

a. Breuer GJ. Purdue University, West Lafayette, IN, USA. Unpublished data. 1997.
b. Zentek J. Unpublished data, 1996.

REFERENCES

The references for **Chapter 33** can be found at www.markmorris.org.

CASE 33-1

Lameness in a Labrador Retriever

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Patient Assessment

A four-year-old female Labrador retriever was examined for difficulty in rising (standing up) and walking, especially the first few minutes of a walk. These problems were worse after the patient had been out for a long walk or played with other dogs, as often happened on weekends. The current exercise program included three 15- to 30-minute walks per day, free exercise in the yard between walks and two 60-minute walks in the woods on weekends.

Physical examination was unremarkable except for moderate obesity (body weight 45 kg, body condition score [BCS] 4/5) and abnormalities identified in the musculoskeletal system. The range of motion of both hip joints was diminished, crepitation was palpated and the dog reacted painfully when its hips were extended. Neither hind limb could be abducted normally in the sagittal plane when the dog was in dorsal recumbency. A thorough examination of limbs and lumbosacral area revealed no other abnormalities.

Radiographs of the coxofemoral joints confirmed a diagnosis of severe osteoarthritis due to hip dysplasia (**Figure 1**).

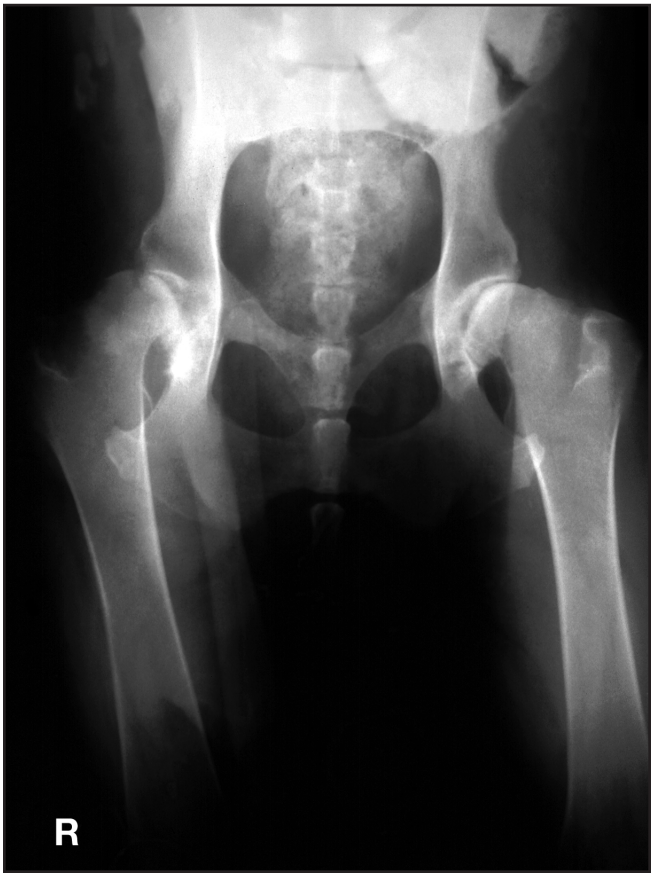

Figure 1. Ventrodorsal radiograph of a four-year-old Labrador retriever with bilateral hip dysplasia. Degenerative joint disease is evident in both coxofemoral joints. Note that the femoral heads have remodeling changes, the acetabuli are shallow and subchondral sclerosis and osteophyte formation are present in the femoral and acetabular components of the joint.

Assess the Food and Feeding Method

The dog was fed four cups (1,500 kcal [6.28 MJ]) of a commercial dry dog food and table foods. The dry food was fed once daily. The table foods were leftovers from the childrens' food; the amount varied daily. The owners indicated the dog gained most of its weight after an ovariohysterectomy two years earlier and during the summer holidays when the dog spent a month with the owners' parents who lived in an apartment for retired people. The food intake during that month was unknown.

Questions

1. What feeding plan should be implemented to improve the condition of this patient?
2. What non-nutritional management practices can be used to reduce the biomechanical stress on the hip joints of this patient?

Answers and Discussion

1. The biomechanical stress induced by rapid weight gain during growth has been cited as a popular etiology for canine developmental orthopedic disease (DOD). It is unknown, and somewhat contested, whether skeletal lesions occur first and are then exacerbated by biomechanical stress, or if biomechanical stress first induces cartilaginous lesions. In either case, increased static forces (excessive weight load) or dynamic forces (excessive muscle pull) may damage immature skeletons.

   In older overweight dogs with established osteoarthritis, biomechanical stress can be diminished with weight reduction. Weight reduction should be continued until very little subcutaneous fat is evident (BCS 2/5).

   Dietary fatty acid changes may provide antiinflammatory benefits that result in clinical improvement in some dogs with osteoarthritis. Changing the food or adding a supplement can manipulate fatty acid levels in the diet.
2. Biomechanical stress on the hip joints can also be reduced through alterations in the exercise protocol for the dog. Exercise is an important component of weight-loss and weight-control programs but must be carefully managed in patients with arthritis. Swimming is an excellent form of exercise that builds cardiovascular endurance and hastens weight loss without overloading the joints. Short, frequent walks on a leash should also be encouraged to prevent overloading of the joints, rather than long walks or unsupervised exercise. Nonsteroidal antiinflammatory drugs can be prescribed as needed for joint pain and lameness.

Progress Notes

A weight-reduction program was outlined for the owners. All table scraps were eliminated and the owners chose to feed a reduced quantity of the dog's current food (two cups [750 kcal, 3.14 MJ] per day divided into two equal feedings). The owners were advised to take the dog for daily swims or as often as possible. In addition, the owners walked the dog on a leash several times daily for approximately 20 minutes or for shorter periods when they recognized the dog was having difficulty rising. The target weight loss was 1% of body weight per week. The owners were instructed to return to the clinic every two weeks for body weight recordings and reinforcement of the program.

The target weight of 35 kg was reached in four months with this controlled exercise and feeding plan. The owners reported the dog could more easily accompany them on long walks. Few signs of lameness were present after the dog reached target weight.

CASE 33-2

Feeding a Golden Retriever Puppy

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Patient Assessment

A 10-week-old male golden retriever puppy was presented for examination and routine health maintenance procedures. The owner had purchased the puppy from a breeder in a neighboring state. The dog was to be used as a family pet and for occasional hunting. The puppy was housed indoors and in an outdoor fenced enclosure.

Physical examination revealed a normal 6.7-kg puppy with a body condition score (BCS) of 3/5. Results of a fecal flotation test were negative. The puppy was vaccinated with an appropriate product and heartworm preventive medication was dispensed. Routine grooming procedures and socialization were discussed with the owner.

Assess the Food and Feeding Method

The breeder had given the owner a bag of a commercial dry specialty brand food (NutroMax Puppy[a]) specifically formulated for growing dogs. The owner brought the bag of food with him to the veterinary clinic. The guaranteed analysis was: crude protein, 28% minimum; crude fat, 17% minimum; crude fiber, 4% maximum; moisture, 10% maximum and ash, 8% maximum (8.9% on a dry matter [DM] basis). The breeder had instructed the owner to offer as much of this food as the dog would eat each day.

The owner was also given a dietary supplement, which was to be added to the food each day. The supplement powder was to be sprinkled over the food (1.5 scoops/day) or moistened to make a broth. The supplement's guaranteed analysis was: crude protein, not less than 42%; crude fat, not less than 19%; crude fiber, not more than 1% and moisture, not more than 4%.

Questions

1. What key nutritional factors are important to consider for this puppy?
2. What additional information is important to obtain about the food and supplement that have been recommended for this puppy?
3. Outline an appropriate feeding (food and feeding method) and monitoring plan for this patient.

Answers and Discussion

1. The key nutritional factors for growing, large- and giant-breed puppies at risk for developmental orthopedic disease (DOD) include energy, fat and calcium. Excessive intake of energy (fat is the primary contributor to energy intake) during growth directly affects growth velocity, contributes to rapid weight gain and may contribute to endocrine dysregulation. Abnormalities of nutrient supply, bone formation and endocrine regulation may interfere with skeletal maturation, thus increasing the risk for DOD in young animals.

   Dogs that ingest excessive amounts of calcium for prolonged periods may develop hypercalcitoninism. The physiologic action of calcitonin on bone turnover (decreased skeletal remodeling) has been proposed as an inciting cause of DOD in dogs.

   Adequate dietary protein is necessary for growth; however, excessive protein intake is not considered a risk factor for canine DOD.
2. The food should be assessed for energy density and specific levels of fat, calcium, phosphorus and protein. These nutrient levels should then be compared with those levels known to be optimal for growth and development of large- and giant-breed puppies. Most of this information is not found on the guaranteed analysis of the package label. The information should be obtained by contacting the manufacturer, reading manufacturers' technical information or consulting other published information or website content. A food for growing dogs should also have passed an Association of American Feed Control Officials (AAFCO) or similar feeding trial. Similar information should be obtained for the supplement.
3. The feeding and monitoring plan should include these steps:
   - Weigh the patient.
   - Estimate the caloric requirement (daily energy requirement [DER] = 3 x resting energy requirement [RER]).
   - Choose a food with metabolizable energy of 3.2 to 4.1 kcal/g (13.4 to 17.15 kJ/g), not more than 17% DM fat, 0.8 to 1.2% DM calcium and 22 to 32% DM protein.
   - Advise the owner to feed the calculated amount of food (energy basis) divided into two to three feedings per day.
   - Reassess the patient every two weeks by weighing it and evaluating its body condition (dogs should have a BCS of 2/5 to 3/5).
   - Adjust the amount of food offered if the BCS is greater than 3/5 or less than 2/5.

Progress Notes

The manufacturers of the food and supplement were contacted. The food had the following nutrient profile (DM): fat = 20.3%, calcium = 1.67%, energy density = 4.4 kcal/g (18.4 kJ). All other nutrient levels exceeded minimum recommendations established by AAFCO for growing dogs. The supplement had the following nutrient profile (DM): fat = 19%, calcium = 3.3%. The supplement also contained essential amino acids, essential fatty acids, vitamins and other minerals.

The combination of the food, supplement and free-choice feeding method probably provided excessive amounts of energy and calcium for optimal growth. The food was changed to another commercial dry specialty brand food (Science Diet Puppy Large Breed[b]), which is specifically formulated to reduce nutritional risk factors for DOD in dogs. This food contains 1% DM calcium, 14.8% DM fat and has an energy density of 3.6 kcal/g (15.0 kJ/g). The DER was estimated (3 x RER = 800 kcal/day [3.35 MJ]) and the owner was asked to discontinue free-choice feeding and begin meal feeding (DER divided into two or three meals per day). The owner was shown how to assign a BCS, asked to record the weight and BCS of his puppy every two weeks and to adjust the amount of food to maintain a BCS of 2/5 to 3/5. The weight and BCS were also recorded in the medical record at 10, 20 and 30 weeks of age when the puppy returned for further vaccinations and other health maintenance procedures. The supplement was discontinued. The owner was also encouraged to maintain a regular exercise and obedience program with the puppy.

Table 1 shows growth data for the puppy during the next 12 months. When the dog was 12 months old, its food was changed to a commercial dry specialty brand food for adult dogs (Science Diet Adult Large Breed[b]). At two years of age, there was no radiographic evidence of hip dysplasia and no clinical problems associated with the musculoskeletal system.

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Endnotes

a. Nutro Products Inc., City of Industry, CA, USA.
b. Hill's Pet Nutrition, Inc., Topeka, KS, USA.

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Table 1. Body weights and body condition scores (BCS) for a golden retriever puppy at 10-week intervals.

Age (weeks)	Weight (kg)	BCS (1-5)
10	6.7	3
20	16.3	3
30	24.0	3
40	28.2	3
50	28.8	3

CASE 33-3

Forelimb Lameness in a Great Dane Puppy

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Patient Assessment

An eight-month-old, male Great Dane puppy was examined for a stiff gait at the outset of walking and right forelimb lameness after taking a long walk. The dog was otherwise healthy but slightly overweight (body weight 48 kg, body condition score [BCS] 4/5). The owner reported that the dog was one of the largest of the litter and that it grew very fast from four to six months of age.

Physical examination was normal except for the musculoskeletal system. Palpation of the scapular region revealed bilateral muscle atrophy that was more pronounced on the right side. Passive movement of all the digits, carpi and elbow joints allowed full range of motion with no pain. Deep palpation of the radius, ulna and humerus did not elicit pain. Movement of both shoulder joints

caused slight crepitation and elicited a painful response, especially with hyperflexion of the joint. Radiographs of the shoulder joints were obtained (**Figure 1**).

Assess the Food and Feeding Method

The puppy was initially fed a commercial dry food formulated for growth (protein = 29%, fat = 18%, calcium = 1.6%, phosphorus = 1.2%, all values listed on a dry matter basis) free choice. Because the puppy was "such a good eater," the owner supplemented the food occasionally with meat and table foods. When the puppy was 14 weeks old, the owner switched foods because the puppy developed abnormal locomotion, which members of the owner's dog club attributed to excessive protein intake. The new food was a commercial dry product formulated for adult maintenance (protein = 20%, fat = 13.3%, calcium = 1.7%, all values listed on a dry matter basis). The new food was offered free choice because of the puppy's "good appetite."

Questions

1. What is the tentative diagnosis and how does this condition cause the clinical signs in this dog?
2. How should this patient be managed?
3. Outline an appropriate feeding plan for this dog.

Answers and Discussion

1. Great Danes and other large- and giant-breed dogs are prone to osteochondrosis in the shoulder joints, especially when excessive energy and calcium are consumed during the period of rapid growth (two to six months of age). Osteochondrosis is a disturbance in endochondral ossification that can result in localized separation of articular cartilage and subchondral bone, and may lead to splitting of cartilage fragments into the joint, i.e., osteochondritis dissecans. Osteochondrosis is not painful; osteochondritis dissecans causes osteoarthritis and inflammation of subchondral bone, which is painful. A diagnosis of osteochondritis dissecans is very likely in this case based on the clinical (painful shoulder joints) and radiologic findings (indentation in the caudal humeral head).

   Because flexion and extension of both shoulder joints is painful with osteochondritis dissecans, dogs will shift their body weight to the rear limbs, resulting in abnormal locomotion. Dogs with osteochondritis dissecans of the shoulder joints also appear stiff because of the limited range of joint motion and have variable degrees of lameness.
2. Surgical treatment is indicated for most cases of shoulder joint osteochondritis dissecans when lameness is present and persisting, and when manipulation of the joint is painful. The loose cartilage flap is removed and the flap bed curetted until the subchondral bone bleeds. Granulation tissue and, ultimately, fibrocartilage fill the curetted defect in the articular surface. The joint is thoroughly irrigated and any floating "joint mice" or bony ossicles attached to the joint capsule are removed. Recovery is predictable with appropriate surgical treatment. Osteochondrosis and osteochondritis dissecans of the shoulder joint will cause secondary osteoarthritis, possibly causing clinical problems in later years, although osteoarthritis of the shoulder joint is usually well tolerated by dogs.
3. Altering the feeding plan may have no beneficial effects on osteochondrosis or osteochondritis dissecans at this stage of the disease process. Excess energy and calcium intake should be avoided during the rapid growth phase between two and six months of age. This puppy is slightly overweight; therefore, feeding to maintain a BCS of 2/5 can reduce biomechanical stress on the shoul-

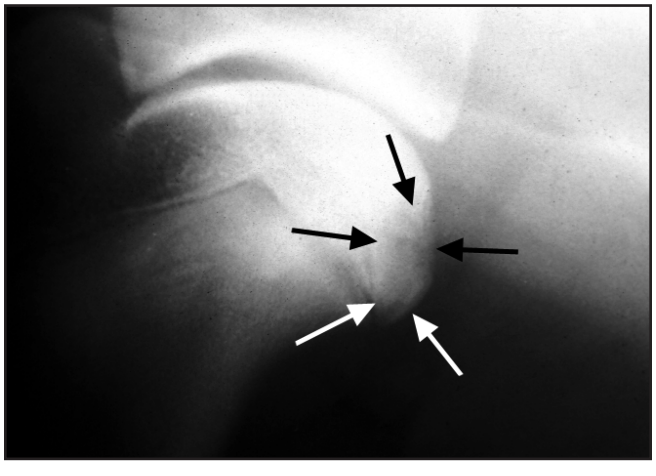

Figure 1. Radiograph of the proximal humerus of an eight-month-old male Great Dane puppy examined for forelimb lameness. The radiolucent area (arrows) is associated with disrupted endochondral ossification (osteochondrosis).

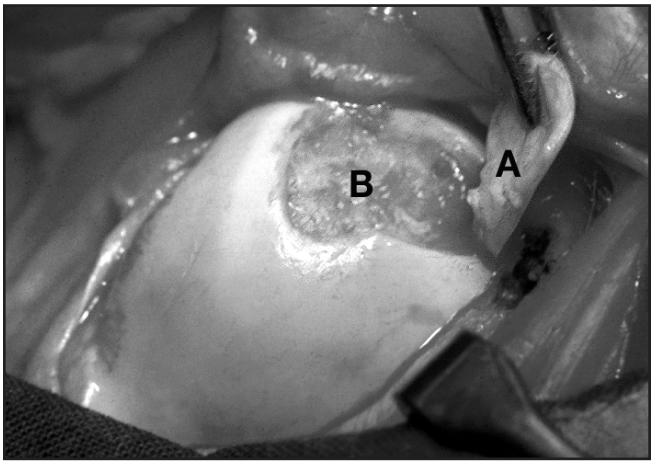

Figure 2. Intraoperative view of an osteochondritis dissecans lesion in the articular epiphyseal cartilage of the proximal cartilage of the same dog. Note the cartilage flap (A) and exposed subchondral bone (B) where a portion of the cartilage flap is missing.

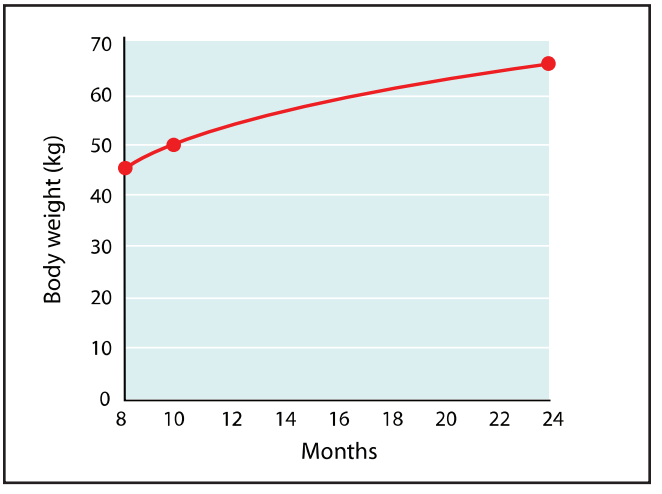

Figure 3. Growth curve recommended for an overweight eight-month-old Great Dane puppy with osteochondrosis.

der joints. Appropriate body condition is especially important considering 60 to 65% of the body weight is normally carried by the forelimbs of walking dogs. Daily energy requirement (DER) should be estimated for the ideal weight at the current age. The owner should discontinue free-choice feeding the puppy and begin meal feeding (DER divided into two or three meals per day). The owner should be shown how to assess body condition, and should record the weight and BCS of his puppy every two weeks. He should adjust the amount of food as necessary to obtain a BCS of 2/5.

Decreasing energy intake by decreasing food intake (meal feeding) and switching to a food with a lower fat level should effectively slow this puppy's growth rate. Adequate dietary protein is necessary for growth; however, excessive protein intake is not considered a risk factor for canine DOD.

Progress Notes

An arthrotomy was performed on the right shoulder and confirmed a diagnosis of osteochondritis dissecans (**Figure 2**). The cartilage flap was removed and the lesion curetted. Examination six weeks later revealed normal locomotion. A decision was made not to perform surgery on the left shoulder.

Because the puppy weighed more than the upper limit for its age, a feeding plan was implemented to slow growth for the next few months. The dog's mature body weight was estimated to be 65 kg, based on knowledge of adult body weights of its parents. The puppy should attain this target body weight at 18 to 24 months of age and have a BCS of not more than 3/5. The owner was given a recommended growth curve (**Figure 3**), taught body condition scoring techniques and given a new feeding plan.

The food was changed to a commercial dry food with 4.14 kcal (17.29 kJ)/g, 25% protein, 12% fat and 1.1% calcium, all values reported on a dry matter basis. No other foods or supplements were fed. The initial DER was estimated to be 1.6 x resting energy requirement at the current body weight or 2,484 kcal (10.37 MJ). It was emphasized to the owner that this was only a starting point and that he would need to monitor body weight and condition carefully, and compare body weights with values on the recommended growth curve. Food intake should be increased 10 to 15% if poor body condition occurred.

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CASE 33-4

Front-Leg Lameness in an Eight-Month-Old Rottweiler

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Patient Assessment

An eight-month-old rottweiler was presented for repetitive front-leg lameness. Signs first appeared when the dog was six months old. According to the owner, although the lameness had no effect on the patient's temperament and playfulness, the puppy had lameness especially after vigorous exercise with other dogs. There were no complaints about the patient's general health; however, the lameness episodes had become more frequent, and no differences in locomotion were noticed when the dog was exercised on different surfaces. The dog had no history of trauma.

When examined, the dog had no signs of impaired health. However, the patient bore more weight on its left than its right front leg. The dog's right elbow joint bulged slightly more at the site of the anconeal muscle than could be palpated on the ipsilateral side. No temperature differences or pain were detected on palpation. More distally, no differences were noticed between the right and left front legs. No abnormalities were found in either hind leg. When examined in lateral recumbency, the dog did not exhibit pain upon passive movement of the shoulder joint, but did on hyperextension of the right elbow joint especially when the antebrachium was concomitantly supinated; no crepitation was evident during the whole range of motion of the elbow joint. Thorough inspection, palpation and passive movement of all joints of the left front leg and both hind legs did not elicit abnormalities.

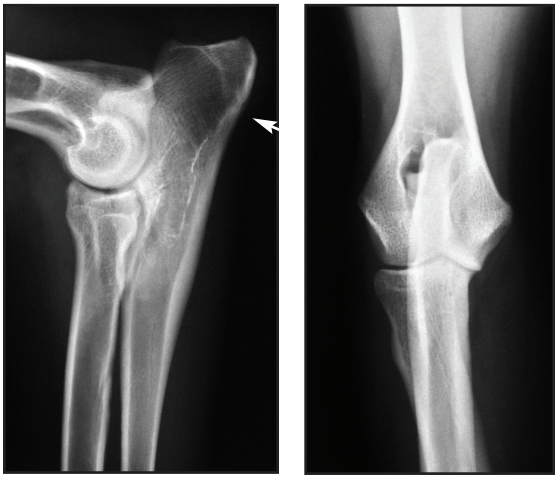

Figure 1. Mediolateral extended view, with sclerosis (arrow) at the ulnar trochlea. No other signs of osteoarthritis are present. The anterioposterior view reveals no abnormalities.[b]

Mediolateral flexed (ML_{flexed}) and anterior-posterior (AP) radiographic views of the elbow joint were taken. The ML_{flexed} view revealed subtle sclerosis of the ulna in the region of the caudal end of the semilunar notch, but neither elbow radiograph showed signs of osteoarthrosis (OA) (**Figure 1**). An ununited anconeal process or indentation of the contours of the medial humeral condyle was excluded radiographically. Additional radiographs were taken of the elbow joint, including a mediolateral view with the elbow joint naturally extended, and a mediolateral oblique view. The normal contour of the coronoid process was visible and an incongruity of the joint was excluded as a diagnostic possibility; also the $ML_{extended}$ view did not reveal any irregularity at the medial humeral condyle or the margin of the medial condyle or ulna.

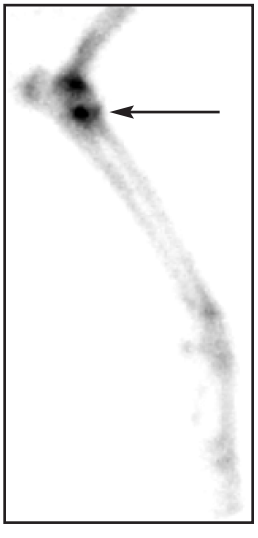

Figure 2. A botscintigram reveals increased calcium turnover irrespectible of its cause. A hot spot (black arrow) appears just distal to the overlapping humeral condyles.[b]

In conclusion, the dog was lame on the right front leg, showed slight bulging over the right anconeal muscle and was painful when the elbow was hyperextended. Radiographs revealed subtle sclerosis of the ulna in the area of the coronoid process. A large percentage of rottweilers have OA of the elbow joint due to a fragmented coronoid process. Epidemiologic studies showed an incidence of more than 50% of elbow OA due to fragmented coronoid process in rottweilers in Scandinavia. The fragmentation occurs at four to six months of age and may cause irritation of the joint with signs of OA (pain, joint effusion, osteophyte formation), although not all dogs are affected to the same degree. Early diagnosis and removal of the fragmented coronoid process will relieve pain and possibly slow, but not prevent, the OA process. Late removal may cause severe cartilage damage, especially at the opposite (humeral) side. It is not advocated to perform invasive diagnostic procedures (i.e., arthrotomy or arthroscopy) when other options exist to further investigate the elbow joint for fragmented coronoid process. Noninvasive approaches include: 1) conservative therapy for an additional period (i.e., four to six weeks) with repeated clinical and radiologic investigation to reveal other abnormalities (e.g., panosteitis, osteochondritis of the shoulder joint), 2) computed tomography scanning to visualize fragmented coronoid process, which is located between the radius and the medial aspect of the ulna and 3) bone scintigraphy to visualize remodeling processes, which are increased in growth, infection, fracture and tumor formation. Bone scintigraphy makes visible different locations that could be overlooked clinically or radiographically.

The owner agreed to a bone scan and a computed tomography scan if the bone scan was positive. Scintigraphy revealed hot spots at the coronoid process (**Figure 2**). Computed tomography scanning of the elbow joint revealed fragmentation of the medial coronoid process (**Figure 3**). The fragmented coronoid process was surgically removed and the dog was put on a diet for young, large-breed puppies to optimize skeletal development during the remaining part of its growth phase (**Case 33-2**).

Assess the Food and Feeding Method

The patient was meal fed a food with reduced amounts of calcium and energy (Science Diet Puppy Large Breed[a]).

Questions

1. How is fragmented coronoid process diagnosed?
2. What is the heritability of fragmented coronoid process, and which environmental factors play a role?
3. Outline an appropriate feeding plan for this dog.

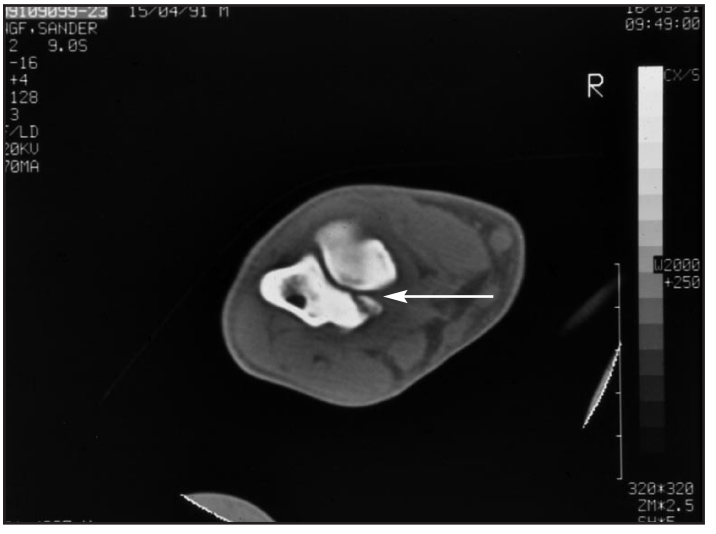

Figure 3. A fragmented coronoid process is evident (white arrow) on a computed tomography scan as cause of the lameness.[b]

Answers and Discussion

1. A good clinical examination will disclose abnormalities in the elbow joint including: more or less joint effusion due to over- or under-production of synovial fluid. In more advanced cases, osteophytes at the joint margins cause slight crepitation during extension, flexion with and without pronation and supination and pain upon hyperflexion and hyperextension, often more prevalent with concomitant pronation. Because 70% of fragmented coronoid process cases can be bilateral it is important to investigate the ipsilateral elbow for a prognosis and therapeutic plan. Hyperextension can be painful in dogs with panosteitis, which occurs in the same breeds and age category as those with fragmented coronoid process; therefore, it is important to rule out deep bone pain here and in other extremities. Also, hip dysplasia and osteochondritis dissecans in the shoulder, stifle and hock joints are seen in rottweilers of this age and warrant extra consideration.

   When no diagnosis can be made, despite serious complaints, clinical and further diagnostic procedures can be postponed for a reasonable period allowing bony changes to develop without much irreversible damage. It is at the discretion of the veterinarian to decide if, during this period, analgesics with provocative activity or rest with confinement should be advised.

   Noninvasive procedures include radiology, computed tomography scanning, bone scanning, sonography and magnetic resonance imaging. The last two are especially informative for soft tissue disorders, whereas the two scanning methods are available in some specialty practices. Radiology, in many cases, helps differentiate the different entities grouped together as elbow dysplasia (i.e., incongruity [visible at $ML_{extended}$ views], ununited anconeal process [visible at the ML_{flexed} view] and osteochondritis dissecans [visible at the AP and/or APMO view]). Damage to growing cartilage, due to a fragmented coronoid process, may cause an indentation at the medial humeral condyle near the location of the osteochondritis dissecans lesion and can, in some instances, be the only indication of a fragmented coronoid process.
2. Because it is difficult to evaluate entire litters and parent animals (i.e., complete clinical and radiologic investigations) information about elbow dysplasia is often incomplete; information about littermates is incomplete, the entities are grouped together or both parents aren't thoroughly investigated. From what is known, the h^2 is between 0.28 and 0.40, indicating that between 28 and 40% of the phenotype is influenced by the genotype and that the remaining depends on environmental influences. In addition to the thought that environment may play a major role in the occurrence of the disease, it has been demonstrated that not all animals with a positive genotype may express the entity. Excessive food intake, excessive mineral intake and excessive body weight have all been mentioned as factors that can play a role in the expression of disturbances in skeletal development. Altered growth in length and thus elbow incongruity and local disturbed endochondral ossification (due to genetic diseases, natural influence, under loading during early development or overloading when vulnerable) are possible causes.

   For screening the population and understanding the etiology, further molecular and population genetic investigations are necessary.
3. Although it is questionable whether dietary changes at this stage of life will prevent other expressions of disturbed endochondral ossification (including osteochondritis dissecans in the shoulder, stifle and hock joints), it is very important to avoid elbow joint stress after surgery. It is clear that an overweight condition coincides with the development of osteoarthritis, possibly in joints without a primary cause. A caloric intake adapted to the changing activity of the patient (decreased activity before surgery and during the recovering period) to prevent excess weight gain is of extreme importance. A balanced, high quality food especially designed for fast growing, large- and giant-breed dogs, characterized by a relatively low calcium content, should be fed until 18 months of age. After 12 to 14 months, dogs will not grow in height, but body conformation will change due to muscle development; a high quality food will support this conformational growth. Frequent palpation of the ribcage and inspection of abdominal lines should help the owner raise a healthy dog. Eventually this patient can be fed a balanced, high-quality diet containing chondroprotective agents (chondroitin sulfate and glucosamine glycans) with increased levels of omega-3 fatty acids to provide constituents that may support cartilage repair and thus help prevent osteoarthritis development.

Progress Notes

After surgery, the patient's leg was bandaged for three days. Exercise was restricted for three weeks after which the dog was exercised on a leash for three more weeks. The owner started a swimming program to help the dog gain musculature without overloading the leg. No lameness was present when sutures were removed 10 days after surgery; the dog was completely recovered after six

weeks. Two years later, the dog was castrated and developed a tendency to become overweight. When the dog was 10% overweight, it developed clinical signs of osteoarthritis (i.e., lameness after intense exercise). A weight-reduction program was implemented and nonsteroidal antiinflammatory drugs were prescribed. The owner was advised to feed the dog a "joint-care" diet characterized by omega-3 fatty acids, chondroprotective nutraceuticals (chondroitin sulfate and glucosamine) and L-carnitine to assist in maintaining a healthy weight.

Endnotes

a. Hill's Pet Nutrition, Inc., Topeka, KS, USA.

b. ©Radiographs, bone scintigraphy and computed tomography are kindly provided by the section of diagnostic imaging of the Department of Clinical Sciences of Companion Animals, Utrecht University, The Netherlands.

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