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Nutritional Risks to Large-Breed Dogs: From Weaning to the Geriatric Years

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L arge-breed puppies can be defined generally as puppies whose mature body weight exceeds 50 lb. A list of diseases associated with large- and giant-breed dogs includes those related to growth and development; however, risks inherent with breed and size persist throughout life and require specific health management. Nutrition may modify expression of genetic predisposition, influence severity of acute disease, and directly affect development of chronic disease.

The domestic dog has the greatest size diversity across mammalian species. Mature adult dogs can weigh from 2 to 200 lb. To achieve that disparity, large- and giant-breed puppies undergo an extremely rapid rate of growth. Giant-breed puppies born weighing 1 lb can easily gain 150 lb within the first 18 months of life, with the most rapid growth rate occurring between 3 and 6 months of age. With such exaggerated rates of growth, large- and giant-breed puppies are sensitive to nutrient and caloric deficiencies or excesses.

Simple overfeeding at any time of life can result in obesity, a disease that is associated with an increased risk of several inflammatory diseases (see the article by Laflamme elsewhere in this issue on understanding and managing obesity in dogs and cats). Dilated cardiomyopathy (DCM) occurs more frequently in large- and giant-breed dogs. Although a clear nutritional deficiency has not been found, low blood taurine and carnitine concentrations have been associated with some cases of DCM (see the article by Sanderson elsewhere in this issue on taurine and carnitine in canine cardiomyopathy). The risk of bloat or gastric dilatation-volvulus (GDV) is included in the list of health concerns of at least 30 purebred large- and giant-breed dogs (see the American Kennel Club web site [1]). The purpose of this article is to review nutritional factors associated with increased risk for developmental orthopedic disease (DOD) and GDV and to provide practical feeding recommendations for prevention.

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WHAT NUTRITIONAL FACTORS ARE ASSOCIATED WITH DISEASE RISK IN LARGE- AND GIANT-BREED PUPPIES?

Few reports of general undernutrition are reported by veterinarians today. Current cases of malnutrition are more frequently associated with overnutrition, which may involve specific nutrients or an excess of food intake in general. Nutrient imbalances are also responsible for development of disease. All growing puppies can be harmed by diet imbalances and excessive food intake, but the term *developmental orthopedic disease* is used to describe those seen predominantly in large-breed dogs. This term refers to a group of diseases that occur during growth and development, including hypertrophic osteodystrophy (HOD), osteochondrosis (OC), osteochondritis dissecans (OCD), retained cartilaginous core, panosteitis, hip dysplasia (HD), and canine elbow dysplasia (CED) [2]. Briefly, the causes of OC and OCD are multifactorial, and identified factors include excess calcium intake, genetics, overnutrition, trauma, and ischemia [3]. HD, another multifactorial developmental problem, is associated with hip joint laxity, leading to incongruent and inconsistent articulation of the femoral head with the acetabulum. HD is associated with excess dietary energy and calcium [4–6]. CED is a group of elbow joint developmental disorders that includes OCD of the elbow joint, ununited anconeal process, and fractured medial coronoid process [7–10]. Breed predilections suggest a genetic component, but trauma, nutritional imbalances, and OC have been associated with this group of developmental disorders. HOD is considered an idiopathic disease that affects puppies between 2 and 8 months of age. Large- and giant-breed puppies are most frequently affected, and male dogs may be more predisposed, as are particular breeds [11–13]. Potential causes include distemper virus infection, overnutrition, genetics, and immune-mediated response to vaccination [14]. HOD has been reported in Great Dane dogs fed excess levels of calcium and phosphorus (Fig. 1) [15].

DCM affects large- and giant-breed dogs. Recent research has suggested that dietary deficiencies of taurine or carnitine, poorly available amino acid precursors in lamb meal, or ingredients like rice bran may pose an increased risk of

Fig. 1. Hypertrophic osteodystrophy in a growing Great Dane puppy fed higher than recommended levels of calcium, diagnosed by radiography.

DCM in certain large and giant breeds. Dietary management of this disease is sometimes possible with supplementation of carnitine and taurine (see the article by Sanderson elsewhere in this issue for a complete discussion). Finally, a risk for obesity is present in puppies of several large breeds from weaning onward, that increases after spaying and neutering, and continues throughout the life of the dog (see the article by LaFlamme elsewhere in this issue).

HOW DOES NUTRITION INFLUENCE RISK FACTORS FOR DEVELOPMENTAL ORTHOPEDIC DISEASE AND OBESITY IN LARGE- AND GIANT-BREED PUPPIES?

Metabolic differences in large-breed dogs exist, such that rapid growth and feeding recommendations have now changed to address nutritional risks associated with feeding large-breed puppies. Fifteen years ago, commercial diets formulated for canine growth were marketed to all puppies with no distinction or formulation differences based on the puppy's anticipated mature size. These diets had evolved to nutrient-dense high-energy foods designed to encourage maximal growth, which is a risk factor for growth disorders. A variety of nutritional risk factors have been associated with DOD. Strong evidence supports the role of excess energy, calcium, phosphorus, and vitamin D in the development of DOD and subsequent osteoarthritis. Several diseases, including osteoarthritis, are associated with excess energy intake, which has been shown to decrease the life expectancy of dogs by 1.8 years compared with dogs provided restricted energy intake [16].

ENERGY

Excess energy intake can be provided to dogs by feeding high-fat nutrient-dense diets or an excess of food. When fed free choice, Great Dane puppies aged 0 to 6 months had an increase in the incidence of DOD compared with littermates that were food restricted to 70% to 80% of the amount consumed by puppies fed free choice [17]. A lifelong study of Labrador Retrievers involved restricting energy in one group of dogs, whereas the second group was fed fee choice for life. Osteoarthritis occurred less frequently and occurred later in life in dogs fed a diet restricted in energy compared with dogs that were fed free choice [18].

Accordingly, large-breed puppy foods contain an energy density of approximately 3.5 to 4.0 kcal/g, which is lower than the 4.0 to 4.5 kcal/g of regular puppy foods. This reduced-energy level results in reduced fat deposition and a lower caloric intake in puppies fed free choice [19]. The dietary fat component of large-breed puppy foods has also been reduced to approximately 12% fat (compared with >20% in other foods) on a dry matter basis (DMB) in the presence of normal fiber content. Dietary fat increases caloric density, thereby increasing caloric intake. Studies in other species have shown that high-fat diets affect levels of insulin-like growth factor-1 (IGF-1), whereas diets high in saturated fats increased bone formation in chicks [20].

The recommendation for lower energy intake and requirements in large-breed puppies is supported by results of a recent study showing that

large-breed puppies have greater nutrient digestibility at 11, 21, 35, and 60 weeks compared with smaller breed puppies [21].

PROTEIN

Currently, no evidence exists to suggest that high-protein intake contributes to the development of orthopedic disease in growing large-breed puppies. Previous studies suggesting a risk for high protein and DOD were confounded by higher energy intake in high-protein foods. In general, large-breed puppy diets are formulated to contain approximately 30% protein (DMB) similar to other puppy foods.

CALCIUM

The mechanism of dietary calcium absorption in puppies undergoes maturation after weaning. Several studies have shown that passive absorption of calcium from the intestine is directly proportional to dietary calcium intake in puppies from weaning ($\sim$6 weeks) until 6 months of age [22–25]. Passive absorption can represent up to 70% of total calcium uptake [26]. Absorption of calcium by active transport is also functional in weaned puppies but undergoes a maturation process, with active absorption decreasing with increasing age in young puppies fed an excess of calcium [27,28]. Active absorption is regulated by vitamin D_3, parathyroid hormone (PTH), growth hormone, and calcitonin [26]. Regardless of the calcium-to-phosphorus ratio, excess dietary calcium intake in large-breed puppies has been shown to increase expression of all individual DODs listed previously in this article (Fig. 2).

After puppies reach 6 months of age, calcium hormonal regulation and subsequent intestinal absorption mechanisms begin to mature and account for nearly 90% of calcium uptake [27]. Although puppies are able to "manage" dietary excesses of calcium after 6 months of age, this maturation process occurs

Fig. 2. Four-month-old Great Dane puppy with DOD fed higher than recommended levels of calcium.

after the rapid growth phase (3–5 months) and time of highest risk for DOD. Excess dietary calcium is stored in skeletal bone of growing puppies. In overfed puppies, normal remodeling begins to reverse the excess calcium deposition at approximately 6 months of age, and the bone mineral content of overfed puppies becomes statistically identical to that of puppies fed normally from weaning by 1 year of age [15]. DOD sequelae develop before puppies are 1 year old, and diseases may be self-limiting, may result in permanent skeletal changes, or may require surgical intervention to correct. The clinical diagnosis of HD or CED may come after 1 year of age, but the joint abnormalities begin at an extremely young age. Great Dane puppies undergoing rapid growth have higher serum levels of growth hormone and IGF-1 than smaller breed puppies when fed normally, yet neither Great Danes nor Miniature Poodles showed differences in calcium handling when fed different levels of calcium [27,29].

PHOSPHORUS

This essential mineral has been not been researched as frequently as calcium in the growth of large-breed puppies. Intestinal absorption of phosphorus, in the presence of normal calcium intake, is well regulated in young puppies [28]. Digestibility or absorption of phosphorus was negatively correlated with levels of dietary calcium when puppies were overfed calcium [28,30]. Dietary deficiencies of phosphorus are not reported frequently in growing puppies, but when they are created under experimental conditions, they consist of poor weight gain and reduced growth rate [18]. The popular home-cooked and raw food diets, most of which are not balanced, usually contain sufficient or high quantities of phosphorus from the meat protein in the diet but have a low calcium content. This imbalance results in an increase in PTH levels (nutritional secondary hyperparathyroidism), but the increase is largely attributable to deficiencies in calcium content of the diet and not to excesses of phosphorus content (see section on calcium/phosphorous balance).

CALCIUM/PHOSPHORUS BALANCE

The optimum calcium/phosphorus ratio for dog foods is generally considered to be between 1.2:1 and 1.4:1 as reported by the National Research Council on Nutrient Requirements of Dogs in 2006. The American Association of Feed Control Officials (AAFCO) lists the acceptable range of ratios in commercial feeds to be between 1:1 and 2:1 [31]. These ratios can become skewed by supplementation of diets with calcium carbonate, multivitamin and/or mineral products, or top-dressing diets with various meats, for example. Weaned giant-breed puppies fed high calcium without an appropriate increase in phosphorous for 4 months developed hypercalcemia, hypophosphatemia, and DOD [29]. After normalization of the diet for a period of 2.5 months, many clinical signs of DOD resolved but OC lesions remained. Another group of giant-breed puppies was fed the same high-calcium diet for a 4-month period but in a proper ratio to phosphorus after weaning. These puppies became slightly hypophosphatemic, but their growth was retarded and DOD occurred. When

the diets of these puppies were normalized for 2.5 months, the developmental disturbances improved but were not resolved. These puppies experienced the effects of high calcium and subsequent increased secretion of PTH that increased intestinal absorption of calcium through the action of vitamin D_3. The resulting asynchronous growth that occurs when mineral accretion and mineral resorption is imbalanced results in DODs, such as HD and CED, and abnormal maturation of subchondral bone resulting in OC and OCD.

VITAMIN D

Vitamin D_3, in its active forms of 1, 25-dihydroxycholecalciferol and 24,25-dihydroxycholecalciferol, regulates intestinal absorption of calcium and phosphorus and renal excretion or resorption of minerals. Hormonal regulation of vitamin D is achieved by PTH, growth hormone, and calcitonin. Dietary calcium and phosphorus intake affects serum levels of metabolites of vitamin D_3, serum levels of calcitonin, and PTH [27,32].

Vitamin D_3 is required in canine diets, because dogs do not have adequate levels of the intermediate compound 7-dehydrocholesterol in the skin that can be converted by ultraviolet radiation to previtamin D_3 [33,34]. Previtamin D_3 is converted to 25-hydroxycholecalciferol in the liver in response to regulation by negative feedback from serum levels of 25-hydroxycholecalciferol and vitamin D_3 intake [35]. The final conversion to the biologically active forms of vitamin D_3 (1, 25-dihydroxycholecalciferol and 24, 25-dihydroxycholecalciferol) occurs in the kidney. The conversion to the 1, 25 or the 24, 25 product in the kidney is controlled by plasma levels of growth hormone, IGF-1, calcitonin, PTH, and inorganic phosphate [36–39].

1, 25-Dihydroxycholecaliferol is considered to be the biologically active form, and 24, 25-dihydroxycholecalciferol is less active; its synthesis is regulated by negative feedback from 1,25-dihydroxycholecalciferol and serum calcium levels. The action of the 1, 25 product is highest in the kidney, intestine, and skeleton, and the activity of the 24, 25 product is highest in the skeleton [40–42]. Vitamin D compounds are influential in regulation of calcium homeostasis by up- and downregulating dietary calcium absorption in the intestine and by regulating resorption of calcium in the kidney. During skeletal development, mineralization is dependent on calcium and phosphorus dietary intake and the proper regulation of absorption, excretion, and resorption of vitamin D metabolites.

Levels of the 24, 25 metabolite are significantly higher in small-breed dogs, and that difference is believed to be the result of growth hormone activity and IGF-1 in the giant-breed puppy [32]. Children with growth hormone deficiencies who are treated with growth hormone experience increases in levels of 1, 25-dihydroxycholecalciferol and decreases in levels of 24, 25-dihydroxycholecalciferol [43]. The role of 24, 25-dihydroxycholecalciferol is seen at the skeletal level, and it is active in bone formation but not in bone resorption, whereas the 1, 25-dihydroxycholecalciferol metabolite is involved in osteoclast generation and related bone resorption as well as in absorption of calcium [44,45]. In giant-breed puppies, levels of 24, 25-dihydroxycholecalciferol were

significantly lower, suggesting that high demand for synthesis of 1,25-deoxy-cholecalciferol may be at the expense of synthesis of 24,25-dehydroxycholecal-ciferol [27].

In a study using Miniature Poodles, supraphysiologic doses of growth hormone were injected to compare the metabolic effects with an untreated group. Supplementation with growth hormone rapidly increased serum levels of growth hormone, resulting in increased plasma levels of IGF-1, increases in serum levels of 1,25-dihydroxycholecalciferal, lower serum levels of 24, 25-dihydroxychole-calciferol, minimal effect on calcium and phosphate absorption in the intestine, elevated glomerular filtration rate, and no significant change in inorganic phosphate urine excretion [45]. At the skeletal level, growth hormone supplementation resulted in increased bone formation, with no bone resorption effects. Further investigation is needed into the role of vitamin metabolites in giant-breed puppies to determine how dietary levels of vitamin D_3 may affect the development of DOD by altering bone synthesis and resorption in rapidly growing large-breed puppies [45]. Commercial dog foods contain ample levels of vitamin D_3 based on the current knowledge of dietary requirements, but hormonal regulation of metabolites of vitamin D_3 can affect the skeletal development of the growing dog with adequate levels of dietary vitamin D [32,46]. Current feeding recommendations are likely to undergo further adjustment as more is understood about vitamin D metabolism in large- and giant-breed puppies.

WHAT FEEDING METHOD AND STRATEGIES SHOULD BE USED FOR LARGE-BREED PUPPIES?

Large-breed puppy foods are designed to restrict energy intake and provide more defined calcium and phosphorus levels and ratios than original puppy foods (Table 1). Clients should be urged to avoid any dietary supplementation or top dressing to diets of large-breed puppies, because commercial diets for large-breed puppies contain proper levels and balance of nutrients. Supplementation can unbalance the diet and contribute to the development of DOD by

Table 1
General nutrient contents of large-breed puppy diets compared with nonspecific puppy diets

Nutrient	Puppy diets (% DMB)	Large-breed puppy diets (% DMB)
Protein	29-36	29-34
Fat	20-23	11-16
Fiber	1.6-4.4	2.4-5.6
Calcium	1.3-1.4	0.8-1.4
Phosphorus	1.2	0.7-1.2
Ca:P ratio	1.1:1	1.1:1 to 1.3:1
Energy density[a]	3.8-4.5	3.4-4.1

Abbreviations: Ca:P ratio, calcium-to-phosphorous ratio; DMB, dry matter basis.
   [a]Energy density is provided as kilocalories per grams of diet as fed.

increasing intake of calcium or phosphorus or changing the desired 1.2:1 calcium-to-phosphorus ratio.

Regardless of careful selection of appropriate puppy foods, maintenance of the proper body weight or body condition score is critical for healthy growth. Puppies should be monitored frequently (perhaps weekly during the rapid growth phase), and managed carefully by increasing or decreasing food to maintain a trim body condition. Several of the major pet food companies have produced body condition score programs to aid in determining an ideal body condition of 3/5 measured on a five-point scale or 5/9 on a nine-point scale. A copy of scoring charts should be provided to puppy owners so that they have a reference for weekly or bimonthly weight monitoring.

Quantities of food offered should be consistently measured, divided, and offered at multiple feeding times. Free-choice feeding frequently leads to overeating and obesity as well as ingestion of higher levels of calcium and phosphorus. Timed feedings can also result in overeating, and caloric intake cannot be controlled with this feeding method. Multiple feedings may slow the rate of eating and decrease the volume of food present in the stomach. Large meal volume and infrequent feeding have been identified as risk factors for the development of GDV, which is discussed elsewhere in this article.

Meal feeding a measured amount of large-breed puppy food divided into several daily feedings is recommended. Amounts to be fed can be estimated from the formulas in Box 1, with alterations in multipliers based on the age of the puppy, where resting energy requirements are computed by the equation RER (kcal/d) = 30 (body weight$_{kg}$) + 70, where RER is resting energy expenditure. Using the age of the puppy to determine the correct multiplier results in a calculation of maintenance energy requirements.

This calculation provides a guideline to be used when estimating the proper intake for a puppy. Each puppy should be objectively compared with a body condition chart at least twice per month to avoid excess weight gain, particularly during the important rapid growth period. A healthy puppy that is maintained at ideal body weight, is being fed an appropriate large-breed puppy formula, and without additional supplementation can be assumed to be growing at a rate normal for that dog. Once the large-breed puppy approaches 12 months of age, it is recommended to change to an adult maintenance food, but feeding adult foods to large-breed puppies before 1 year of age is not recommended because the calcium-to-energy ratio is generally lower in adult foods compared with large-breed puppy food. Feeding an adult food can

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Box 1: Energy requirements for growth in puppies

Weaning to 4 months: 3 × RER

4 months to 1 year: 2 × RER

1 year and older: 1.6 × RER (adult requirements)

actually result in greater intake of calcium than feeding puppy foods. Because the puppy must consume a larger portion of adult food to meet energy needs for growth, total calcium intake may actually be higher than with a properly formulated large-breed puppy formula.

It is also recommended that caloric intake be carefully monitored and possibly decreased once the puppy is spayed or neutered. At this time, there are contradictory reports as to the need for adjusting intake; however, it is apparent that energy needs decrease and obesity risk increases after spaying or neutering. If clients are appropriately educated about body condition scoring and have access to these tools, the incidence of obesity in all puppies can be reduced.

SUMMARY

To prevent skeletal growth disturbances and obesity in large-breed dogs, a preventive or proactive approach to managed growth of large-breed puppies is recommended. Many large-breed puppy rations are available, but not all are comparable, and adult maintenance foods are not suitable substitutes for large-breed puppy foods. Energy density, calcium, and phosphorus levels should be lower than those of standard puppy feeds. Energy density of a large-breed puppy food should be 3.5 to 4.0 kcal/g, with a fat content less than 15% (DMB). The calcium content of foods should be approximately 1%, with phosphorus levels at approximately 0.8% (DMB), with an ideal calcium-to-phosphorus ratio of 1.2:1. Vitamin D levels are usually adequate in commercial pet foods but may require supplementation when balancing home-cooked recipes. Second, feeding large-breed puppies should be accomplished by meal feeding rather than free-choice or timed feedings. Body condition should be assessed weekly or biweekly, because changes can occur quickly and adjustments to intake should be made promptly to maintain an ideal body condition. If home-cooked diets are used, recipes should be balanced by a nutritionist to ensure proper quantities of key nutrients, such as protein, fat, calcium, phosphorus, and vitamin D. The effects of skeletal disturbances and obesity can result in deformities in bones and joints that are irreversible. Any disease that affects mobility and quality of life for these large dogs can result in early death or euthanasia.

NUTRITIONAL RISKS FOR LARGE- AND GIANT- BREED DOGS: GASTRIC DILATATION AND VOLVULUS

Why Is Gastric Dilatation and Volvulus an Important Concern in Large- and Giant- Breed Dogs?

GDV is a rapidly developing syndrome that carries a mortality rate of 24%, despite surgical intervention [47]. Mortality rates have declined as medical management has improved (mortality rates of 15% to greater than 60% have been reported) [48,49]. The risk of development of GDV during a lifetime is 24% in giant-breed dogs and 21% in large-breed dogs [47]. A specific breed predilection seems to exist, as does a familial predisposition for incidence, potentially through selective breeding for physical characteristics, such as depth and width of thorax [50–52]. Great Danes and Bloodhounds are most

frequently represented in reports, although more than 30 breeds are considered to be susceptible [53]. GDV is a leading cause of death in giant-breed dogs [50].

The etiology of GDV is still uncertain. It is multifactorial, and the pathophysiology is complex. Many inciting factors have been identified, such as gastric motility dysfunction, aerophagia, gastric content fermentation, gastric inflow and outflow hindrance, and rotation of the stomach. Many risk factors have been suggested. At this time, careful management of these risk factors and prophylactic surgery may be the best tools for reducing the incidence of GDV in dogs.

What Happens During Gastric Dilatation and Volvulus and How Is It a Significant Threat to Life?

The progression of this syndrome occurs quickly and can be fatal in just a few hours. During this progression, gases rapidly accumulate in a stomach, where intake and outflow are obstructed. The stomach may or may not rotate on the long axis of the esophagus, and bloat may or may not precede gastric dilatation [54–57].

What Risk Factors Have Been Identified for the Development of Gastric Dilatation and Volvulus in Dogs?

Dietary risk factors

The extrusion method of dry pet food manufacturing was introduced in 1957, and an "epidemic" of GDV was reported in the early 1960s through 1995. Naturally, an association was made between dry commercial foods and the increased incidence of this disease [58–62]. The same commercial dry dog food implication was made in 1994 with a report of a greater than doubling incidence of bloat and GDV cases in 12 veterinary hospitals between 1980 and 1989. Diet was considered to be the primary factor, and extruded foods containing cereal or soy (which were considered to be poorly digestible when compared with animal source proteins, and gas producing) were assumed to contribute to the development of GDV [63–65]. Subsequent research that measured the gastric emptying time of dogs eating canned foods or dry cereal foods, with and without added water, did not support the association between extruded dry foods and an increased risk for the development of GDV [66]. A recent study analyzing dry food ingredients did not show that animal protein sources were associated with lower risk than plant protein sources, but did find that dry diets with oil or fat among the first four ingredients were associated with a 2.4 times increase in the risk of developing GDV and that moistening dry foods before feeding also increased the risk [67,68]. Large food particle size (>30 mm) was found to decrease the risk of development of GDV [69]. Irish setters, one of the breeds with the highest susceptibility, were three times more likely to develop GDV if fed a single food type, such as dry or canned foods [59]. These data were confirmed when the addition of table scraps or canned food to a commercial dry diet reduced the risk of development of GDV by 59% and 28% in large- and giant-breed dogs, respectively [63].

Specific recommendations have been made for feeding management of susceptible animals. Feeding just one meal per day and rapid ingestion of meals

(37.8% of cases) are identified as risk factors for GDV, as is feeding a heavier meal (physical weight), which promotes gastric distention [63,70]. Once-daily feeding is associated with stretching or laxity of the hepatogastric ligaments, and rapid eating is associated with aerophagia, which is also associated with the development of GDV [59].

Results of prospective studies have shown that earlier recommendations made from retrospective study results may have been incorrect. Current research suggests that feeding from an elevated food bowl increases the risk by 51.9% despite earlier studies suggesting that a raised food bowl reduced the risk. Restricting water before and after meals was also associated with a higher risk of development of GDV, contradicting early recommendations for water restriction near meal times [67]. Exercise restriction before and after meals has also been removed from the list of potential risk factors through the results of prospective studies.

Nondietary risk factors

In addition to dietary factors, nondietary risk factors have been identified for GDV. Male dogs of less than ideal weight and with an apprehensive temperament were associated with an increased risk of GDV [50,63]. A recent stressful event (8–36 hours previously) seemed to increase the risk for development of GDV, whereas a cheerful disposition reduced the risk of development [63]. Increasing age and being of such breeds as the Great Dane, Weimaraner, St. Bernard, Gordon Setter, and Irish Setter were associated with the development of GDV as well as having a first-degree relative (sibling or offspring) that was affected by GDV [51,70].

Weather conditions and a first-quarter calendar or seasonal association with GDV were reported in a group of military working dogs [71]. A second study was unable to support the association between ambient temperature, humidity, season, or atmospheric pressure and GDV incidence [53]. Morphometric measurements of lower thoracic width, greater abdominal width, and a lower abdominal depth-to-width ratio were associated with a reduced risk of GDV in giant-breed dogs but not in large-breed dogs [67]. Giant-breed dogs but not large-breed dogs are more susceptible with a thin or lean body condition, postprandial abdominal distention, and a history of other medical problems. A thin body weight and a major health problem in the first year of life were factors for an increased risk of GDV [72]. A potential association between GDV and inflammatory bowel disease was reported in a study in which intestinal biopsies were collected at the time of surgery to correct GDV. Sixty-one percent of these biopsies were found to contain an identifiable inflammatory process, and 86% of the dogs in this study had prior histories of gastrointestinal disturbances [73].

How Can We Prevent Gastric Dilatation and Volvulus in Large- and Giant-Breed Dogs?

Two feeding practices that have repeatedly been identified in reported studies to reduce the risk of GDV occurrence are multiple daily feedings to reduce

food volume and slowing the rate of eating. Separation of dogs in multidog households may be required to slow the rate of meal consumption. In addition, more recent feeding recommendations include feeding from floor level (as opposed to the use of raised feeders) and management of gastrointestinal health to reduce inflammatory conditions within the gastrointestinal tract, such as management of food intolerances and other potentiators of inflammatory conditions, as well as avoiding the use of dry commercial diets with high-fat content. The addition of canned foods or meats to the diet may also reduce the incidence of GDV. Larger kibble size (>30 mm) may help to reduce the risk of GDV.

Dogs that have had GDV or have siblings, sires, or dams that have had the disease should not be bred. Selection of stable temperaments in breeding stock can also reduce the risk for the development of GDV, whereas selective breeding for particular physical traits, such as a deep chest and tight abdominal tucks, can increase the risk for development of GDV.

SUMMARY

Large- and giant-breed dogs present veterinarians and pet owners with distinct challenges throughout their lives. Among the most important are proper nutrition to ensure moderate growth rates to reduce developmental orthopedic disorders and promote proper cardiac development, maintenance of an ideal body condition to prevent diseases associated with obesity, and proper feeding techniques to reduce the risk of GDV.

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